ELICITATION OF THE ACOUSTIC CHANGE COMPLEX (ACC) TO LONG-DURATION SPEECH STIMULI IN FOUR-MONTH-OLD INFANTS

By

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Abstract

The acoustic change complex (ACC) is a cortical auditory-evoked potential (CAEP) that comprises overlapping slow cortical responses (P1-N1-P2) and occurs in response to changes during an ongoing stimulus (Martin, Tremblay, & Korczak, 2008). Research findings suggest that the ACC indicates discrimination at the level of the auditory cortex and provides insight into the brain’s capacity to process acoustic features of speech (Kaukoranta, et al., 1987; Ostroff, et al., 1998). Only one study to date has attempted to record ACCs to speech stimuli in young infants (Small & Werker, 2012). Small and Werker (2012) tested a group of English-learning four-month-old infants with speech contrasts generated from a synthetic place-of-articulation continuum: a native dental-dental contrast /dada/, dental-labial contrast /daba/, and a non-native Hindi dental-retroflex contrast /daDa/. Slow cortical responses resembling adult P1-N1-P2 complex were recorded for all conditions with significantly prolonged latencies compared with adults. Robust ACCs were elicited in most infants to /daba/ with distinct P1, N1 and P2 components, but fewer infants had ACC components in response to /dada/ and /daDa/. The author suggested that the absence of ACCs in /dada/ and /daDa/ conditions might be due to short stimulus length.

The purpose of the present study was to investigate the effect of long-duration speech stimuli for eliciting ACCs in four-month-old infants. By increasing the stimulus length from 564 to 816 ms, ACCs were reliably elicited for all stimulus conditions (/dada/, /daba/ and /daDa/) in infants with more distinct cortical components and better morphology compared with previous findings (Small & Werker, 2012). The amplitude of P1 elicited to the acoustic change in /daba/ and /daDa/ was significantly larger compared
with that of P1 for /dada/, indicating that the brain discriminated between the speech tokens. In conclusion, our results support the findings by Small and Werker (2012) showing that adult-like slow cortical responses can be recorded in young infants. Our results also suggest that ACCs can be reliably elicited in four-month-old infants given optimized stimulus parameters (e.g. longer ISIs and stimulus duration) and provide further evidence for the use of ACCs as an index of discrimination ability.
Preface

All of the work presented in this master thesis was conducted in the Pediatric Audiology Lab at the University of British Columbia, Point Grey campus. All studies and associated methods were approved by the Behavioural Research Ethics Board of the University of British Columbia (certificate #H07-01218).

This Master’s thesis is based on previous work by S. Small and J. F. Werker (2012). None of the text of the thesis is taken directly from previously published articles. The stimuli were created by P. Kandhadai and J. F. Werker at the Infant Studies Centre at the University of British Columbia. The infant participants were recruited through a subject database of the Infant Studies Centre. I performed data collection and analyses, as well as manuscript composition under the supervision of my supervisor, S. Small.
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Dedication

To my mother and husband:

I couldn’t have done it without you.
1 Introduction

One of the early steps for infants to learn a language is to recognize the phonetic distinctions and sound patterns of their native language. Although studies have revealed many developmental changes in language skills in the first year of life, behavioural methods provide rather limited information about perceptual capacities in infants and it is still unclear whether infant neural responses would reflect similar age-related changes in the brain mechanism that underlies speech perception. Therefore, electrophysiological measures such as cortical event-related potentials (ERPs) offer a useful complement to behavioural measures. ERP methodology is increasingly used to investigate central sensory processing in infants because it is non-invasive and does not involve active participation by the infant in some cases. Speech-evoked auditory ERPs are measured brain responses time-locked to the eliciting speech stimulus and are commonly used for examining speech detection and discrimination tasks, thus providing information about the underlying neural processing of the frequency, amplitude, and timing cues within the speech signal (Tremblay, Billings, & Rohila, 2004). Although considerable research has been conducted to further our understanding of the neural mechanism underlying language development in infants and young children, there remain several areas in the development of cortical auditory evoked potentials (CAEP) that need to be investigated, and the behavioural and perceptual correlates of infant ERPs are yet to be established.

Cortical auditory ERPs are usually classified as either exogenous (or obligatory) or endogenous (or cognitive) (Näätänen, 1992). Exogenous ERPs are obligatorily elicited by the occurrence of a stimulus and highly dependent upon the stimulus features. Since obligatory ERPs are less affected by non-auditory factors, such as attention and
memory, they can be recorded reliably in infants and young children (Wunderlich & Cone-Wesson, 2006). Endogenous ERPs, on the other hand, are not determined by physical stimulus characteristics, but rather induced due to some level of cognitive processing (Purdy et al., 2001).

1.1 Slow cortical responses (P1-N1-P2 complex)

The slow cortical response (P1–N1–P2), which was first reported by Davis in 1939, have been extensively used to investigate the neural mechanism underlying sound processing in the human auditory system. These CAEPs are generated at the level of the primary auditory cortex and auditory association areas of the temporal lobe (Čeponienė et al., 1998; Ponton et al., 2002) and reported to reflect the neural encoding of sound signals (Hillyard & Picton, 1978). The slow cortical response is obligatorily elicited by the brief onset of an auditory stimulus and is comprised of several temporally overlapping waves or components that represent activity from various cortical sources. The acoustic parameters of the stimulus and the integrity of the primary auditory pathway can greatly affect the presence, latency and amplitude of the P1-N1-P2 complex (Wunderlich & Cone-Wesson, 2006). Slow cortical responses can be elicited by different stimuli such as tone bursts, clicks and words, and they can be reliably recorded in awake adults as well as young infants and children; however, the latency, amplitude and scalp distribution of slow cortical responses undergo maturational changes through infancy and late adolescence before they develop into the adult form (Ponton et al., 2002; Wunderlich & Cone-Wesson, 2006).

Slow cortical responses recorded in infants differ substantially from those
recorded in adults. A major feature of the maturation of the slow cortical response is the change in morphology and the increasing complexity of the waveforms. Infant waveforms tend to be biphasic, and gradually develop into more complex multiphasic (P1-N1-P2-N2) responses with age. In contrast to adult waveforms, which are dominated by N1, P1 is the most prominent peak for children’s waveforms. This positive peak typically occurs at 100–300 ms post-stimulus onset, which is later than the adult’s P1 latency (Wunderlich & Cone-Wesson, 2006; Sharma & Dorman, 2006)). Some studies have shown that newborn and very young infants tend to have a broad positive peak P2 with a latency of 200–250 ms and a broad negative trough N2 at around 300–550 ms, while N1 and P1 are less frequently recorded and have smaller amplitudes (Little et al., 1999; Molfese, 2000). As infants become older, the peak component becomes more clearly defined. Studies examining slow cortical responses in older children have shown that P1 and N1 components, which are often missing in the recording of very young infants, become more frequently evoked with larger amplitudes than the later components, P2 and N2. This decline in the amplitude and peak latency of N2 leads to general maturation of morphology at 12 years of age, while other components of the slow cortical response continue to change beyond this age (Ponton et al., 2000; Sharma & Dorman, 2006; Wunderlich & Cone-Wesson, 2006).

The maturational change in slow cortical waveforms are reported to be consistent with the general development of the central nervous system. In particular, the decrease observed in the latency of all P1-N1-P2 components has been found to concur with developmental changes in myelination and synaptogenesis throughout the first twenty years of life (Sharma et al., 1997). Furthermore, research has suggested that P1 and N1
may have different underlying neural generators because the amplitude and dominancy of those two peaks show different patterns in their maturational changes. N1 is reported to indicate activity in the primary auditory cortex, while P1 demonstrates activity from thalamocortical sites (Sharma et al., 1997; Wunderlich et al., 2006). Therefore, slow cortical responses are believed to indicate the arrival of a sound stimulus in the auditory cortex and provide an index for encoding of acoustic events (Stapells, 2002; Picton et al., 2000).

P1 and N1 components are reported to primarily reflect sensory encoding of auditory stimulus properties compared with more endogenous components such as N2 and P3, which are more likely to be affected by attention and cognition. P1 in children has been established as an indicator, or biomarker, for assessing the maturation of the auditory cortex (Sharma et al. 2005). The P1 response is a robust positive response occurring at a latency of 100–300 ms in young children, and it is generated by auditory thalamic and cortical sources (Dorman et al., 2007). The P1 peak latency, which is regarded as an index of cortical auditory maturation, changes with age. In newborn infants with normal hearing, the average latency of the P1 waveform is about 300 ms. During the first two to three years of life, however, a rapid decrease occurs in P1 latency. Three-year-olds have a P1 latency of about 125 ms, and in children aged 5–6 years the peak latency is around 85–95 ms. Studies have demonstrated that middle-aged adults have an even shorter P1 latency of approximately 40–60 ms. The age-related changes of P1 latency thus can be used to assess the maturation of the central auditory pathway in children with hearing loss and to track their development over time (Sharma et al., 1997; Ponton et al., 2000; Sharma et al., 2005). There is also a decline in the amplitude of P1,
which is possibly due to the emergence of N1 instead of maturation of P1 generators (Čeponienė et al., 2002; Wunderlich et al, 2006).

Although different components of the slow cortical response have been extensively studied in adults, there are widely varying results regarding the first appearance and development of the component in young children. Some studies have reported that N1 first appears about 5–6 years of age with peak latency approximately 100–150 ms. N1 peak latency generally decreases while the amplitude increases with age (Bruneau et al., 1997; Cunningham et al., 2000; Ponton et al., 2000;). Other studies have suggested that the N1 component is absent in young children and there is little or no age related change in latency and amplitude (Čeponienė et al., 1998; Ohlrich et al., 1978; Sharma et al., 1997; Tonnquist-Uhlen, 1996; Tonnquist-Uhlen et al., 2003). These inconsistent findings may be in part due to the sensitivity of N1 amplitude to inter-stimulus intervals (ISIs) used in different studies, indicating the highly refractory nature of the underlying neural generators of the response in children (Paetau et al., 1995; Rojas et al., 1998).

Stimulus variables can greatly affect the characteristic of the slow cortical responses. The amplitude, latency and scalp distribution can be determined by different parameters such as stimulation rate, stimulus duration, rise time and intensity (for a full review see Näätänen & Picton, 1987; Roberts et al., 2000). Although many studies have examined the effects of these variables in adult auditory system, only a few studies have investigated their effects in the young infant brain. The ISI can greatly affect the morphology and amplitude of the slow cortical response in both adults and children, but the effects are more distinct in children than in adults (Picton et al., 1978; Wunderlich &
Cone-Wesson, 2006). In adults, N1 is the most prominent negative peak regardless of ISI; however, in older children, waveforms become more complex with increased ISI (Čeponienė et al., 1998). While the typical biphasic response of P1 at 100 ms and a broad N2 at 250 ms have been recorded for a short ISI (350 ms), two additional negative peaks are reported to be present at 160 ms (N1) and 460 ms when ISI is increased. N1 has been shown to be absent for a short ISI due to the prominence of P1 and N2 components, but as ISI is increased to 700 ms, N1 has become the predominant negative peak in the waveform (Čeponienė et al., 2002).

Neural refractoriness is the amount of time it takes for a neural population to recover after generating a response to a stimulus before getting ready for a second stimulus (Wilson & Groves, 1981; Munemori et al., 1984). The central auditory system in young infants is immature due to incomplete myelination and synaptogenesis, resulting in longer neuronal refractory periods and lower cortical excitability for CAEP components (Čeponienė et al., 1998; Gilley et al., 2005). Because myelination and synaptic refinement underlie the developmental changes in the latency and synchrony of the neural signal, evoked potentials will have shorter latencies, larger amplitudes, and more defined waveform morphology with maturation (Sharma et al., 2005). Gilley et al. (2005) have investigated developmental changes in refractoriness of the cortical auditory evoked potential by examining the morphological change in P1-N1-P2 waveform as a function of varying stimulation rates. In a paradigm indirectly assessing refractory properties of the underlying neuronal generators, a train of brief vowels [uh] was presented sequentially with decreasing ISIs of 2000, 1000, 560, and 360 ms to children aged 3–12 years and young adults. Latencies and amplitudes of the P1, N1, and P2 components at the Cz
electrode were examined as a function of stimulus rate and age. Their results showed that
the N1-P2 component was elicited only at a longer ISI at younger ages, and the waveform
became more clearly apparent at successively faster stimulation rates with increased age,
indicating significant changes in the morphology of the slow cortical response as a
function of age and stimulation rate.

1.2 Mismatch negativity (MMN)

Mismatch negativity (MMN) is an automatic neural response elicited by an
infrequent “deviant” event when it is presented with a sequence of “standard” events,
which was first described by Näätänen et al. (1978). The MMN is the difference
waveform obtained by subtracting the ERPs to the standard event from the ERPs to the
deviant event. In other words, the MMN indicates discrimination between the neural
memory trace formed by a repetitive standard stimulus and a new stimulus (Näätänen,
2007). When elicited by sudden changes in stimulation, the MMN peaks at about
100–250 ms after the onset of stimulus presentation and demonstrates a temporal-frontal
topography (Sams et al., 1985). It is maximally recorded from electrodes in the vicinity of
Fz. The MMN originates from the primary and secondary auditory cortices, and may
have an additional generator in the frontal cortex (Näätänen, 1990; Opitz, et al., 2002).
While the generators in the auditory cortex are reported to index brain activity related to
detection of change, the generator in the frontal cortex is suggested to underlie switching
or orienting of attention, which is provoked by the change detection mechanism (Giard, et
al., 1990; Rinne, et al., 2000). Due to the underlying process of automatic auditory
change detection, the MMN has been associated with pre-attentive cognitive operations
in audition, indicating “primitive intelligence” in the auditory cortex (Näätänen et al., 2001).

Similar to other ERPs, the MMN undergoes maturational changes during the first few years of life due to the major structural and functional development of the brain (e.g., myelination, synaptogenesis, and axonal growth). The MMN can be recorded in early infancy (Cheour et al., 1997a; Kurtzberg et al., 1995), and it has even been observed in pre-term infants (Cheour-Luhtanen, 1996) and fetuses (Huotilainen et al., 2003). MMN responses are different between adults and infants. While the latency of MMN does not differ significantly between adults and school-age children (Kraus et al., 1992), it tends to be slightly longer in infants and young children (Kurtzberg et al., 1995, Cheour-Luhtanen et al., 1996, Cheour et al., 1997a). Although studies indicate that MMN latency is developmentally stable, MMN amplitude has been reported to be more susceptible to developmental changes (Csépe 1995; Kraus et al., 1992). MMN peak magnitudes increase rapidly in infants and young children so that the overall magnitude of the MMN (peak to offset) tends to be larger in children than in adults (Aaltonen et al., 1987; Cheour et al., 1997a). In addition to the traditional negative MMN response peaking between 100–300 ms (Cheour et al., 1997; 1998a,b; Kushnerenko et al., 2001), researchers have also recorded a positive discriminative response peak emerging between 300–400 ms in young infants (Dehaene-Lambertz & Dehaene, 1994; Friederici et al., 2002). Therefore, both negative and positive responses have been observed in infants. Vaughan and Kurtzberg (1991) suggest that the negative MMN is only observed in pre-terms and neonates, and it grows into a positive peak with age. However, other studies indicate that
negative discriminative responses have been observed more frequently as infants become older (Cheour et al., 1998b; Trainor et al., 2001).

The scalp distribution of the MMN is also different in infants and adults. It has been reported that the MMN is fronto-centrally dominant and larger over the right hemisphere in adults when speech stimuli are used (Alho, 1995; Paavilainen et al., 1991). In contrast, infants demonstrate a prominent MMN response not only over frontal and central areas but also over parietal areas (Cheour-Luhtanen et al., 1996; Leppänen & Lyytinen, 1997). Other studies have reported that the MMN scalp distribution is more central in infants than in adults (Kraus et al., 1999). Therefore, a broader MMN scalp distribution is observed in infants and young children, which may be due to differences in the conductivity of the skull (Cheour, 2007). Another explanation for the difference in the scalp distribution between the two age groups is that the connections between the different parts of the cortex are less specific and more redundant in infants than in adults (Neville, 1995). Therefore, infant MMN may reflect the activity of much larger parts of the brain than the adult MMN.

There are several advantages to using the MMN to investigate speech perception. First, the MMN can be used to index pre-attentive discrimination because the participant is not required to pay attention to the sounds during recording (Paavilainen, et al., 1987). Therefore, the participant may not find it difficult to engage in the task. Secondly, research has shown that the MMN can be elicited to a variety of acoustic features of speech, including changes in frequency (Sams, et al., 1985), intensity (Näätänen, et al., 1987), timing (Näätänen, et al., 1989), location (Paavilainen, et al., 1989), and the pattern of stimulation (Alain, et al., 1998). Lastly, the MMN has demonstrated reasonable
agreement with behavioural discrimination performance (Sams, et al., 1985; Martin & Boothroyd, 2000).

Although the auditory mismatch paradigm has been commonly used with adults to assess their discrimination ability, there are potential limitations that hinder its use with young children and infants and more research needs to be done before it can be used clinically. One limitation is that MMN cannot be obtained and interpreted reliably in different participants due to individual variations in MMN amplitude and latency (Cheour et al., 2000). Also, studies have shown that MMN cannot be obtained in some healthy infants. Kurtzberg et al. (1995) and Cheour et al. (1998a) have reported that the MMN is absent in approximately 20–30% of the neonates. Leppänen et al. (1997) have also failed to record the MMN in about 50% of the infant participants. These results may be explained in part by different parameters such as ISIs used in those studies (Cheour et al., 2000). Another explanation for the absent MMN in some infants is that the MMN has a poor signal to noise ratio due to small amplitudes obtained after subtraction (Lang, et al., 1995; Stapells, 2002). In addition, some studies have reported that the MMN cannot be reliably elicited in all individual participants, especially children and infants who have shorter attention spans (Kurtzberg, et al., 1995; Escera & Grau, 1996; Pekkonen, et al., 1995), in part because the MMN requires sustained periods of ERP recording to obtain better morphology (Lang, et al., 1995) and may introduce concerns about refractoriness and habituation (McGee, et al., 2001).

Despite of the limitation, the MMN has proved useful as a clinical tool for investigating certain diseases such as schizophrenia and dyslexia in adults and older children (Stephan et al., 2006; Näätänen, 2003; Korpilahti & Lang, 1994; Kraus et al.,
For example, Kraus et al. (1996) has reported a reduced MMN in children with learning disabilities suggesting that the deficit occurs at a processing stage in the auditory pathway prior to conscious perception. To date, the majority of pediatric MMN studies have been conducted in older children and very few studies have investigated the MMN in clinical infant populations due to its limitations.

1.3 **Acoustic change complex (ACC)**

The P1–N1–P2 complex can also be recorded in response to change(s) in an ongoing stimulus, such as intensity and frequency modulations or in response to multiple time-varying acoustic changes within a speech sound (Martin & Boothroyd 1999). The resultant waveform pattern, which contains multiple, often overlapping P1-N1-P2 responses that occur to the onset and offset of a stimulus, as well as the change(s) within a stimulus, are termed the acoustic change complex (ACC) (Martin et al., 2008). Research findings suggest that the ACC indicates discrimination at the level of the auditory cortex and provides insight into the brain’s capacity to process the acoustic features of speech (Kaukoranta, et al., 1987; Ostroff, et al., 1998). The ACC has been recorded in response to intensity, frequency, and phase modulations in sustained tones (Jerger & Jerger, 1970; Näätänen & Picton, 1987; Yingling & Nethercut, 1983; Dimitrijevic et al. 2008). In addition, both speech and non-speech stimuli have been used to evoke ACCs (Kaukoranta et al., 1987; Ostroff et al., 1998; Tremblay et al., 2006). The ACC has also been recorded in response to changes of periodicity (Martin & Boothroyd, 1999), amplitude and spectrum (Martin & Boothroyd, 2000). Ostroff (1999) has demonstrated that the ACC shows good agreement with behavioural tests of frequency
discrimination as small as 38 Hz in the second formant of synthetic, three-formant vowels, which is considered a threshold for defining vowel contrast. The ACC has also been elicited to simple syllables at the transition from consonantal segment to vocalic segment (Hari, 1991; Kaukoranta, Hari, & Lounasmaa, 1987; Ostroff, Martin, & Boothroyd, 1998).

Research findings have suggested that the ACC may have the potential to be used as a clinical tool for assessing speech perception capacity. First, the ACC has been recorded in response to speech stimuli such as consonant-vowel syllables, in which the acoustic change included frequency, amplitude, and periodicity cues similar to those found in normal conversational speech (Kaukoranta, Hari, & Lounasmaa, 1987; Ostroff, Martin, & Boothroyd, 1998). A study by Ostroff et al. (1998) has investigated cortical potentials in response to naturally produced speech syllable /sei/ and a typical N1-P2 complex has been recorded to the acoustic change from /s/ to /ei/. The finding suggests that the ACC reflects changes of cortical activation caused by amplitude or spectral change at the transition from consonant to vowel and it may have the potential to demonstrate discrimination capacity. The change from aperiodic to periodic stimulation has also produced changes in cortical activation that contribute to the observed response.

Secondly, research has shown that the ACC indicates intensity discrimination (~3 dB) (Martin & Boothroyd, 2000) and frequency discrimination (~10 Hz) (Martin, 2007; Martin et al., 2010), which are consistent with the behavioural findings. Thirdly, Tremblay et al. (2003) have reported excellent test-retest reliability of the ACC elicited to natural speech stimulus within individual adult participants. Good efficacy of the ACC has also been found in individuals with sensorineural hearing loss (Tremblay et al, 2006)
and in those with cochlear implants (Friesen & Tremblay, 2006). Furthermore, when compared to other ERPs, such as the MMN, the ACC elicits responses with larger amplitudes and better signal-to-noise ratios, thus requiring less time and fewer stimulus presentations for recording (Martin & Boothroyd, 1999). These advantages can be very important for testing infants and other populations that are difficult to test. As a result, the ACC has the potential to be a viable tool for determining neural encoding abilities.

Despite the potential advantages of the ACC as an index of discrimination capacity, only one study to date has attempted to record the ACC to speech stimuli in very young infants (Small & Werker, 2012). In their study examining the ACC in infant speech perception, Small and Werker presented four-month-old infants, who were learning English as a first language, with speech contrasts generated from a synthetic place-of-articulation continuum: a native dental-dental contrast /dada/, a dental-labial contrast /daba/, and a non-native Hindi dental-retroflex contrast /daDa/. These stimuli adhered to the description reported in the behavioural study by Werker and Lalonde (1988). In their study, Werker and Lalonde investigated cross-language speech perception using a conditioned head-turn paradigm and they reported that infants whose native language was English successfully discriminated the native /daba/ and non-native Hindi /daDa/ contrast when they were under six months of age. In contrast, infants older than 10 months of age, like adult participants, were no longer able to discriminate the non-native Hindi speech contrast. These findings supported the hypothesis, which was first suggested by Eimas (1995), that infants are born with a biological predisposition allowing them to discriminate the universal set of phonetic contrasts; however, a decline or reorganization in this universal phonetic sensitivity takes place by the end of the first year.
as a function of linguistic experience of the ambient language. In order to investigate the
behavioural and ERP correlates of infant speech perception, Small and Werker tested
four-month-old English-learning infants with the same speech contrasts from the previous
study (Werker & Lalonde, 1988). Consistent with the behavioural data, Small and Werker
(2012) reported that robust ACCs were elicited in most infant participants to /daba/ with
P1, N1 and P2 components, but fewer infants had the different components of the ACC
present in their responses to /dada/ and /daDa/. The morphology of the ACCs elicited to
/dada/ and /daDa/ was also more variable compared to /daba/. The ACC was recorded
infrequently to English /dada/ presumably because no distinct acoustic change between
the two speech tokens was detected. This study did not replicate the behavioural findings
for non-native stimuli (i.e., elicitation of the ACC to non-native stimuli /daDa/ in the
four-month-old infants) and the authors suggested that one of the possible causes for the
inconsistency might be the relatively short stimulus length, which might not have been
long enough to accommodate the neural refractory periods in the immature brain.
Another possible reason for the absence of a clear ACC for /daDa/ was that the linguistic
experience could have already had an impact on the discrimination of the phonemes, even
though the perceptual reorganization from language-universal to language-specific
phonetic categories has not taken place behaviorally. An interesting finding of the study
was that an adult-like P1-N1-P2 complex was recorded in response to the onset of the
stimulus, while the latencies of the components of the slow cortical response were
significantly prolonged compared with adults, indicating immaturity of the infant brain.
Given that the vast majority of studies in the literature have reported a biphasic waveform
with a large positive and negative peak recorded in young infants, Small and Werker
(2012) attributed this result to an appropriate combination of stimulus parameters (e.g. long ISIs).

The speech stimulus used in the study by Small and Werker (2012) consisted of two consonant-vowel syllables (CVCV), which was different from the typical CV (e.g. /da/) or VV (e.g. /ui/) stimulus used for ACC research. In this case, although the major acoustic change was between two CV syllables, the brief transition from the consonant to the vowel within the CV token could have also evoked cortical responses resulting in overlapping cortical waveforms thus affecting the overall morphology of the ACC. Furthermore, the acoustic change of interest to the second CV syllable occurred right after the initial onset P1-N1-P2 complex. Although the stimulus duration was adequate for eliciting the ACCs to the native speech contrast /daba/, the neural population in the infant brain may need longer time to recover from the initial firing in a more challenging test condition (i.e., the non-native /daDa/ condition) because the linguistic experience with their ambient language might have already played a role on the development of speech perception; thus, the four-month-old infant brain might find it more difficult to discriminate the non-native /daDa/, even though they were able to discriminate the contrast behaviorally. Therefore, longer stimulus duration could potentially be used for eliciting ACCs to allow a longer refractory period for young infants.

The purpose of the present study was to investigate the effect of long stimulus duration on the ACC elicited to speech contrasts in four-month-old infants who are learning English as a first language based on the previous study by Small and Werker (2012). By increasing the stimulus duration from 564 ms to 816 ms and allowing more time for neural refractoriness, ACCs were expected to be recorded in response to the
changes in both native and non-native speech contrast condition with better morphology. The information obtained from this study will further our knowledge of the optimal parameters for eliciting the ACC and provide evidence for its application in the pediatric population.
2 Methods

2.1 Participants

Two groups of participants were included in this study. The first group included 24 four-month-old infants with normal hearing (mean age: 4 month 11 days; range: 4 month 0 day to 5 months 15 days). These infants were recruited through a subject database of the Infant Studies Centre at the University of British Columbia. All infants were learning English as a first language and had no exposure to the Hindi language. Infant participants were screened for hearing using transient-evoked otoacoustic emission (TEOAEs) with the Madsen AccuScreen Pro (GN Otometrics). Infants who did not pass the hearing screening or had a history of hearing loss were excluded from the study (N=1). Infant participants were also excluded if data collection was not completed due to crying, excessive movement, or technical problems (N=9). The second group consisted of a total of ten English-speaking adults and they were recruited from the University of British Columbia. All adult participants had normal hearing with thresholds lower or equal to 25 dB HL for 500, 1000, 2000, and 4000 Hz. They served as a control group and ranged in age from 24 to 38 years with a mean age of 29 years.

2.2 Stimuli

The stimuli were created from three speech tokens: English bilabial /ba/, dental /da/, and Hindi retroflex /Da/, which were selected from a synthetic voiced place-of-articulation continuum that was originally constructed to examine the perception of retroflex and dental-stop consonants in infants (Werker & Lalonde, 1988). The three speech tokens were paired to form speech contrasts containing acoustic changes from /da/
to /ba/ and from /da/ to /Da/ (i.e., /daba/ and /daDa/) with no gap in between. Both speech contrasts started with the same token /da/ (denoted as S1), which was followed by one of the other two tokens /ba/ or /Da/ (denoted as S2). A third paring, /dada/, was also created to serve as a control condition where there was no acoustic change between S1 and S2.

Werker and Lalonde (1988) created five formant stimuli and constructed a synthesized 16-step continuum by varying the starting frequency of F2 and F3 (second and third formants). The three speech tokens /ba/, /da/ and /Da/ selected in the present study represented equal step intervals across articulation locations and they were equivalent to the 3rd, 8th, and 13th tokens among the 16 steps, respectively. The fundamental frequency was 100 Hz for the first 100 ms then rose to 120 Hz. F1 rose from 250 to 500 Hz over a period of 50 ms while F4 and F5 remained constant at 3500 and 4000 Hz, respectively. The steady-state frequency was 1090 Hz for F2 and 2440 Hz for F3, and the transitions for both F2 and F3 lasted 50 ms. The starting frequency of F2 varied for /ba/, /da/ and /Da/, and they were 1000, 1250 and 1500 Hz, respectively. The starting frequency for F3 were 2384, 2528 and 2627 Hz for /ba/, /da/, and /Da/. Small and Werker (2012) used these tokens to elicit slow cortical responses in adult participants and they reported no significant difference in the amplitudes and latencies of the P1, N1, P2, and N2 components except for N1 which had a larger amplitude to /da/ and /ba/ compared with /Da/.

The original speech tokens from Werker and Lalonde (1988) were saved as .wav files and edited in the sound program in Neurobiomedics STIM2 software. For the purpose of the current study, the vowel portion of the original tokens was lengthened
using Praat 5.3.23 software so that the duration of the token was increased from 282 to 410 ms. Beyond 410 ms, the speech token became distorted and sounded unnatural. By increasing the S1S2 stimulus duration from 564 ms to 816 ms and allowing more time for the longer neural refractory period in infants, ACCs were expected to be recorded in the infants. Each stimulus was made using the Sound program in Neurobiomedics STIM2 by concatenating two speech tokens, S1 and S2, with no silent period in between. The longer S1S2 stimuli were presented with the same 2200 ms inter-stimulus interval (ISI) as in the previous study (Small & Werker, 2012); however, the onset-to-onset duration was necessarily increased. Cortical ERPs were recorded to the three S1S2 stimuli (i.e., /dada/, /daba/, and /daDa/). These stimuli were played at 86 dB peak SPL continuously in blocks of at least 250 trials. Due to the excessive movement and vocalization of the infant participants, more trials were recorded at the discretion of the examiner to ensure that a minimum of 130 accepted epochs would be included in the final waveform.

The stimuli were generated by STIM2 and then delivered to Tucker Davis Technologies PA5 and SM5 modules. The overall gain of the stimulus was reduced by 13 dB before routing it to the HB7 headphone driver. A loudspeaker was used to deliver the stimulus in the sound booth. It was centered and placed one meter in front of the infant participant who was held by a parent seated in a comfortable chair, both facing toward the loudspeaker. A Larson Davis System 824 and Larsen Davis Model 2559 0.5 were used to calibrate the speech stimuli in the sound field with a random-incidence microphone placed at the approximate position of the infant’s head. The stimulus level was calibrated in dB peak SPL.
2.3 Recordings

A four-channel electrode montage was used to record the ERPs in all participants. Individual gold-plated cup electrodes filled with electrode paste were placed at Cz, C3, M1, M2, and FPZ (International 10–20 system) and secured with tape. The electrode site M2 was chosen as the reference and the electrode located on the forehead served as ground. Eye-blink activity was monitored using bipolar electrodes pasted above and below the centre of the left eye. The Compumedics Neuroscan Synamps2 and SCAN 4.3 software were used to record the electroencephalograph (EEG). All inter-electrode impedances were measured and kept below 5 kOhms with the SCAN 4.3 impedance routine.

During data acquisition, the EEG channels were filtered using a 30 Hz low-pass filter and a 1.0 Hz high-pass filter. The continuous EEG was amplified with a gain of 500 and converted using an analog-to-digital rate of 1000 Hz. The recording window consisted of a 100 ms pre-stimulus period and a 1400 ms post-stimulus time. After acquisition, single trails were baseline corrected across the entire sweep duration and an ocular artifact reduction was applied using an average of three epochs, which contained ocular movement greater than 250 ms epoched over -100 to 300 ms. Single trials were rejected manually by visual inspection when electrophysiologic activity exceeded 75 µV in amplitude over a range of -100 to approximately 800 ms. As a result, each average ERP waveform was optimized by subtracting the rejected epochs from the total number of epochs recorded and the numbers varied with each infant.

To optimize the number of accepted epochs to be included in the final waveform, a minimum of 250 epochs was recorded for each condition and the rejected epochs were
excluded from final averaging. The total number of recorded epochs varied with each infant from 260 to 483 across stimulus conditions because it was difficult to estimate how much the movement and vocalization shown by the participant would affect the data collection during online recording (Tables 1 and 2). A minimum of 130 accepted epochs was required for each stimulus condition to be included in final data analysis to allow for a minimum of two replications for each condition. The minimum number of accepted epochs was determined by examining the data from a pilot study with four-month-old infants, which revealed that fewer than 130 accepted epochs in total resulted in poorer morphology and replicability of averaged waveforms. A split-epoch method was used to generate two replications (i.e., odd- versus even-numbered epochs) for each condition tested. The actual number of accepted epochs ranged from 135 to 277 across stimulus conditions and participants (Tables 1 and 2).
Table 1: Summary of the mean, standard deviation and range for the total number of epochs recorded and the final number of accepted epochs in the waveform elicited in 10 adults (N=10 per stimulus condition) after baseline adjustments, ocular corrections and manual artifact rejection were conducted for each stimulus condition.

<table>
<thead>
<tr>
<th>Stimulus condition</th>
<th>/dada/</th>
<th>/daba/</th>
<th>/daDa/</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total epochs recorded</td>
<td>Mean</td>
<td>333</td>
<td>322</td>
</tr>
<tr>
<td></td>
<td>1 SD</td>
<td>25</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>303-371</td>
<td>301-360</td>
</tr>
<tr>
<td>Final epochs accepted</td>
<td>Mean</td>
<td>310</td>
<td>305</td>
</tr>
<tr>
<td></td>
<td>1 SD</td>
<td>28</td>
<td>18</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>267-365</td>
<td>275-329</td>
</tr>
<tr>
<td>Artifact rejection rate</td>
<td>7%</td>
<td>5%</td>
<td>8%</td>
</tr>
</tbody>
</table>

Table 2: Summary of the mean, standard deviation and range for the total number of epochs recorded and the final number of accepted epochs in the waveform elicited in 24 infants (N=8 per stimulus condition) after baseline adjustments, ocular corrections and manual artifact rejection were conducted for each stimulus condition.

<table>
<thead>
<tr>
<th>Stimulus condition</th>
<th>/dada/</th>
<th>/daba/</th>
<th>/daDa/</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total epochs recorded</td>
<td>Mean</td>
<td>378</td>
<td>369</td>
</tr>
<tr>
<td></td>
<td>1 SD</td>
<td>68</td>
<td>65</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>260-451</td>
<td>293-457</td>
</tr>
<tr>
<td>Final epochs accepted</td>
<td>Mean</td>
<td>217</td>
<td>198</td>
</tr>
<tr>
<td></td>
<td>1 SD</td>
<td>56</td>
<td>55</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>135-277</td>
<td>137-262</td>
</tr>
<tr>
<td>Artifact rejection rate</td>
<td>43%</td>
<td>46%</td>
<td>48%</td>
</tr>
</tbody>
</table>
2.4 Procedure

All tests were carried out in a double-walled sound-attenuated booth in the Pediatric Audiology Lab at the University of British Columbia. Adult participants were seated comfortably in an armed reclining chair and watched a movie with subtitles with no sound throughout testing. They were instructed to ignore the stimuli presented to them and to remain as quiet and still as possible. The infant participant was held by a parent who sat in a comfortable chair facing a loud speaker. An age-appropriate movie was played silently on a flat-screen monitor placed directly behind the loud speaker. An assistant also stayed in the booth to engage the infant’s attention in order to minimize the head movement and reduce myogenic noise in the EEG.

Adults were required to complete all three stimulus conditions (i.e. /dada/, /daba/, and /daDa/) to be included in the study, while infants were required to complete at least one condition. The order of stimulus conditions was randomized for each participant. Only five infants completed more than one stimulus condition and none of them completed all three conditions. An experimenter observed the EEG during data acquisition to monitor the infant’s state, muscle movement, and electrical artifact. Testing was stopped if the infant started to cry or vocalize continuously during the recording. Hearing screening was conducted in both ears at the end of the test session. The duration of the recording was approximately 1.5 hours for adults and 10 to 40 minutes for infants. After explaining the study to the adult participants and the parents of infant participants, written consent was obtained. An honorarium was given to the adult participants. A small honorarium and a little gift were given to the parents and their infants at the end of the session.
2.5 *Data Analysis*

The EEG epochs recorded from a condition were divided into two separate groups (i.e. odd and even) and averaged into two ERP waveforms. By superimposing the two average waveforms, the components of the slow cortical response could be compared and the degree to which they differ was presumed to be a reflection of the amount of residual noise. If the average waveforms show a strong similarity in morphology, it means that the signal-to-noise ratio is reasonable in the given data. On the other hand, if the split average waveforms do not show strong correspondence, there may be excessive noise in the waveforms and the data should be excluded from the analysis. Figure 1 shows the example of split-epoch average waveforms for each stimulus condition.
Figure 1. Split-epoch waveforms elicited to /dada/, /daba/, or /daDa/ in one infant participant for each stimulus condition.
The discrete latency and amplitude of slow cortical components were analyzed only from the recordings of C3 channel for each participant and the morphology of ERP waveforms to /dada/, /daba/, and /daDa/ were compared qualitatively. ERP peaks evoked to the S1 and S2 portion of the stimulus were identified by means of visual inspection based on the response latency window for the cortical component. Response latency windows were determined by measuring latency values for cortical components to S1 and S2 from the onset of S1 and the onset of S2, respectively. The largest peaks within each of the latency windows were manually selected for each stimulus condition and the mean amplitude and latency of that peak were recorded. Baseline-to-peak amplitudes were measured for S1 responses and the individual latencies were measured at the peak amplitude. For S2, the baseline was not always at 0 µV and, in these cases, the baseline was moved to the point where the first cortical component (i.e., P1 or N1 if P1 absent) elicited to S2 began to undergo deflection. All baseline-to-peak amplitudes of the subsequent cortical components were measured with reference to that point. In order to compare the overall amplitude of the S2 relative to S1, individual peak-to-peak amplitudes were also measured. The percentage of response components present for each stimulus condition was calculated. Grand mean ERP waveforms to each of the stimulus conditions were compared in terms of morphology, mean amplitudes, and latencies.

For the adult group, two-way repeated-measures analyses of variance were carried out to compare peak amplitudes and latencies of the P1, N1, and P2 components elicited to /dada/, /daba/, and /daDa/. N1–P2 peak-to-peak amplitudes of S1 and S2 stimuli for the /dada/, /daba/, and /daDa/ stimulus conditions were also compared for the adults. For the infant group, peak amplitudes and latencies of the P1 and N1 components evoked to
/dada/, /daba/, and /DaDa/ were compared using one-way repeated-measures analyses of variance. Newman-Keuls post hoc comparisons were performed for significant main effects. Results for all analyses were considered statistically significant if p < 0.05.
3 Results

3.1 Adults

Because the overall morphology of waveforms evoked to each stimulus condition was very similar across individual adult participants, only grand mean waveforms obtained to /dada/, /daba/ and /dDa/ at C3 are displayed, as shown in Figure 2. A classical pattern with robust P1, N1 and P2 components elicited to the onset of S1 was observed in all three conditions. A similar pattern with smaller amplitude was also recorded for S2, and presumably in response to the acoustic change. As shown in Table 3, clear P1 and N1 component were recorded in 90% of the adult participants, while P2 and N2 were present in all adults for S1 stimuli. Table 4 indicates the mean latencies elicited to S1 stimuli across stimulus conditions, which are 66, 110, 183 and 293 ms for P1, N1, P2 and N2, respectively. The mean baseline-to-peak amplitudes for the P1, N1, P2 and N2 component elicited to S1 stimuli across stimulus conditions were 1.92, -2.79, 5.34 and -3.67 μV, respectively.
Figure 2. Grand mean waveform elicited to /dada/, /daba/, and /daDa/ at C3 for 10 adult participants. The onset of the S1 and S2 portion of the S1S2 stimuli are shown at the bottom of the graph. The P1, N1, P2, and N2 components of the obligatory cortical response elicited to S1 are indicated on the graph.
<table>
<thead>
<tr>
<th>Token</th>
<th>Component</th>
<th>/dada/</th>
<th>/daba/</th>
<th>/daDa/</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Infant</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>S1</td>
<td>P1</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>N1</td>
<td>88</td>
<td>100</td>
<td>100</td>
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<tr>
<td></td>
<td>P2</td>
<td>100</td>
<td>88</td>
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<td></td>
<td>N2</td>
<td>88</td>
<td>63</td>
<td>75</td>
</tr>
<tr>
<td>S2</td>
<td>P1</td>
<td>100</td>
<td>88</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>N1</td>
<td>75</td>
<td>75</td>
<td>88</td>
</tr>
<tr>
<td></td>
<td>P2</td>
<td>75</td>
<td>50</td>
<td>38</td>
</tr>
<tr>
<td></td>
<td>N2</td>
<td>25</td>
<td>38</td>
<td>13</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>BN</td>
<td>38</td>
<td>13</td>
<td>25</td>
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<tr>
<td><strong>Adult</strong></td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>S1</td>
<td>P1</td>
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<td>90</td>
</tr>
<tr>
<td></td>
<td>P2</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>N2</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>S2</td>
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<td>80</td>
</tr>
<tr>
<td></td>
<td>N2</td>
<td>40</td>
<td>50</td>
<td>30</td>
</tr>
</tbody>
</table>

Table 3: Percentage of responses present for each stimulus condition for infant (N=8) and adult (N=10) participants. The terms “BP” and ”BN” denoted “broad positive” and “broad negative” peaks, respectively.
The overall morphology of the waveforms recorded in response to the acoustic change from S1 to S2 (i.e., the ACC) was similar when compared across stimulus condition /dada/, /daba/ and /daDa/ (Figure 2). P1 component was present in all adult participants, while N1 was recorded in the majority of cases for /dada/, /daba/ and /daDa/. P2 was present in 80% of adults for /daba/ and /daDa/, but only half of the participants showed a clear P2 component for S2 /dada/. On the other hand, N2 was absent in more than half of the adults. The mean latencies for S2 stimuli were on average 89, 168 and 254 ms for P1, N1 and P2, which were 23, 58, and 71 ms longer in comparison to the components evoked to S1. The mean amplitudes for the different components elicited to S2 were smaller when compared to the P1-N1-P2 complex elicited to S1. There were no obvious differences in S2 amplitudes for individual peaks across stimulus conditions.

Figure 3 compares the peak-to-peak amplitudes of N1-P2 complex between S1 and S2. It was found that the amplitude elicited to /daba/ was 11–12% larger compared to /dada/ and /daDa/, but the amplitude of acoustic change from syllable /da/ to /ba/ was not significantly larger than the change from /da/ to /da/ and /da/ to /Da/ [F(2,8) = 0.402, p = 0.682]. However, the amplitude of N1-P2 elicited to S1 were statistically larger than those elicited to S2 [F(1,4) = 8.251, p = 0.045].
Table 4: Adults: mean (1SD) baseline-to-peak amplitude and peak latency measurements for individual components of the waveform elicited to the S1 and S2 portion of the /dada/, /daba/ and /daDa/ stimulus conditions are shown. The latencies were measured from the onset of the S1 stimulus and from the onset of the change in the stimulus at S2. Mean (1SD) N1-P2 amplitudes are also indicated. Mean values that represent measurements from fewer than five responses are denoted with an asterix (*).

<table>
<thead>
<tr>
<th>Peak</th>
<th>/dada/</th>
<th>/daba/</th>
<th>/daDa/</th>
<th>/dada/</th>
<th>/daba/</th>
<th>/daDa/</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean amplitude in µV (1SD)</td>
<td>Mean peak latency in ms (1SD)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>S1</td>
<td>P1</td>
<td>1.86</td>
<td>2.08</td>
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<td>68</td>
<td>64</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1.01)</td>
<td>(1.54)</td>
<td>(0.83)</td>
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<td>(15)</td>
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<tr>
<td></td>
<td>N1</td>
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<td>-2.30</td>
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<td></td>
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<td>(1.29)</td>
<td>(1.30)</td>
<td>(2.50)</td>
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<td>(15)</td>
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<td>(2.70)</td>
<td>(2.58)</td>
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<td>(1.08)</td>
<td>(1.73)</td>
<td>(1.73)</td>
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<td>(30)</td>
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<tr>
<td></td>
<td>N1-P2</td>
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<td>7.93</td>
<td>8.62</td>
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<td></td>
<td>(3.42)</td>
<td>(3.20)</td>
<td>(4.10)</td>
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</tr>
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<td>S2</td>
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<td>(0.97)</td>
<td>(43)</td>
<td>(32)</td>
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<tr>
<td></td>
<td>N1</td>
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<td>-2.19</td>
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<td></td>
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<td>(1.17)</td>
<td>(0.53)</td>
<td>(63)</td>
<td>(23)</td>
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<tr>
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<td>P2</td>
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<td>(1.05)</td>
<td>(1.29)</td>
<td>(1.23)</td>
<td>(18)</td>
<td>(17)</td>
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<tr>
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<td>-0.71*</td>
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<tr>
<td></td>
<td></td>
<td>(0.58)</td>
<td>(0.97)</td>
<td>(0.16)</td>
<td>(38)</td>
<td>(21)</td>
</tr>
<tr>
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<td>N1-P2</td>
<td>4.08</td>
<td>4.68</td>
<td>3.44</td>
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</tr>
<tr>
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<td></td>
<td>(1.63)</td>
<td>(1.84)</td>
<td>(0.79)</td>
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</tr>
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</table>
Figure 3. The peak-to-peak amplitudes of the N1-P2 components of the grand mean ACC in adults expressed as a percent of the response to S1 /da/ for /dada/, /daba/, and /daDa/ stimulus conditions (N = 10).
3.2 Infants

The overall morphology of the waveform elicited to each of the stimulus conditions was similar across the infant participants, as shown in Figure 4. Cortical responses from each of the 24 infants are shown in Figure 5. Similar to adults, cortical responses from most participants showed a classic P1-N1-P2 complex in response to the onset of the S1 stimuli. A robust P1 was present in all cases and only 12% of the infants either failed to show a clear N1 in response to /dada/ or a clear P2 to /daba/. As indicated in Table 3, 63–88% of the infants also demonstrated a N2 component. The overall morphology of the grand mean responses to S1 stimuli was similar to that of the adult, but the peak latencies of the components were 26–222 ms later compared to adults. Table 5 summarizes the baseline-to-peak amplitude and peak latency measurements for individual components of the waveform elicited to the S1 and S2 portion of the /dada/, /daba/ and /daDa/. The mean latencies were calculated across the three stimulus conditions for P1, N1, P2 and N2 and, on average, they were 132, 226, 320 and 415 ms, respectively. The mean amplitudes for the components of the cortical response elicited to S1 were larger in comparison to adults and ranged from -9.30 to +7.46 µV (Table 5).
Figure 4. Grand mean waveform elicited to /dada/, /daba/, and /daDa/ at C3 for a total of 24 English-learning 4-month-old infant participants with normal hearing. The onset of the S1 and S2 portion of the S1S2 stimuli are shown at the bottom of the graph. The P1, N1, P2, and N2 components of the obligatory cortical response elicited to S1 are indicated on the graph.
Figure 5. Individual waveform elicited to /dada/, /daba/, and /daDa/ at C3 for the 24 infants described in Figure 4.
Table 5: Infants: mean (1SD) baseline-to-peak amplitude and peak latency measurements for individual components of the waveform elicited to the S1 and S2 portion of the /dada/, /daba/ and /daDa/ stimulus conditions are shown. The latencies were measured from the onset of the S1 stimulus and from the onset of the change in the stimulus at S2. Mean (1SD) N1-P2 amplitudes are also indicated. Mean values that represent measurements from fewer than five responses are denoted with an asterix (*). The dashed line indicates that no responses were detected. The terms “BP” and “BN” denoted “broad positive” and “broad negative” peaks, respectively.

<table>
<thead>
<tr>
<th>Peak</th>
<th>/dada/</th>
<th>/daba/</th>
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<th>/dada/</th>
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<th>/daDa/</th>
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<tr>
<td></td>
<td>Mean amplitude in µV (1SD)</td>
<td>Mean peak latency in ms (1SD)</td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>P1</td>
<td>N1</td>
<td>P2</td>
<td>N2</td>
<td>BP</td>
<td>BN</td>
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<tr>
<td>S1</td>
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<td>124 (15)</td>
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<td>N1</td>
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<td>-9.13 (4.36)</td>
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<td>225 (16)</td>
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<tr>
<td>P2</td>
<td>7.46 (3.46)</td>
<td>5.61 (2.67)</td>
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<td>319 (29)</td>
<td>328 (55)</td>
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<td>S2</td>
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<td>9.62 (1.63)</td>
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<td>134 (36)</td>
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<td>13.69 (8.27)</td>
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Table 5: Infants: mean (1SD) baseline-to-peak amplitude and peak latency measurements for individual components of the waveform elicited to the S1 and S2 portion of the /dada/, /daba/ and /daDa/ stimulus conditions are shown. The latencies were measured from the onset of the S1 stimulus and from the onset of the change in the stimulus at S2. Mean (1SD) N1-P2 amplitudes are also indicated. Mean values that represent measurements from fewer than five responses are denoted with an asterix (*). The dashed line indicates that no responses were detected. The terms “BP” and “BN” denoted “broad positive” and “broad negative” peaks, respectively.
The morphology of the grand mean waveform elicited to the acoustic change from S1 /da/ to S2 /da/, /ba/, or /Da/ resembled the morphology of the P1, N1, and P2 components of S1 responses (Figure 4); however, great variation was observed when the participants’ waveforms were examined individually (Figure 5). All infant participants except one showed a robust P1 component and the mean peak latencies were 136, 134 and 142 ms for /dada/, /daba/, and /daDa/, respectively (Table 5). In contrast, the N1 component elicited to S2 was more variable. A clear N1 component was recorded in the majority of the participants (75−88%), but the grand mean waveform obscured some of the individual differences, thus having later peak latencies and broader negative troughs for /daba/ and /daDa/ in comparison to /dada/. The mean peak latencies of N1 elicited to S2 were 259, 285 and 296 ms for /dada/, /daba/, and /daDa/, and the response occurred 34, 55 and 74 ms later compared with the S1 peaks. For /dada/, the N1 component elicited to S2 /da/ was absent in two out of eight infant participants and a broad negative trough was present instead. The other six participants who had a sharp negative peak that resembled an N1 in this condition could be separated into two groups based on their latencies: two of them had N1 occurring at approximately 207 ms and four participants had an N1 latency of approximately 285 ms, which was 78 ms later. In /daba/ condition, two out of eight participants did not have a sharp negative peak in response to S2 /ba/. Among the rest of the participants, five infants had an N1 component at approximately 716 ms, while one infant had an N1 occurring at 127 ms earlier at 589 ms. For /daDa/, one infant participant did not have a discernible S2 N1 peak. Among those who had a clear N1 component in this condition, one participant showed an earlier response at 251 ms and six participants showed a later N1 response at approximately 303 ms. Therefore,
the latency of the N1 response was more variable than that of P1 across the three stimulus conditions. A second positive peak that resembled P2 elicited to S2 was also present in some infant participants. For /dada/, five out of eight infants had a P2 component at approximately 472 ms and one infant had an earlier P2 at 324 ms. For /daba/, only half of the participants had a second positive peak and the latencies varied largely across the infants (Table 3). For /daDa/, only three out of eight infants had a second positive peak at approximately 421 ms. Because the second positive peak to S2 did not occur consistently for /daba/ and /daDa/, only /dada/ had a discernible S2 P2 peak.

A two-way ANOVA was performed to compare the mean amplitudes of P1 and N1 elicited to S1 and S2 across the three stimulus conditions. There was a significant main effect of stimulus condition for P1 amplitudes \[F(2,20) = 4.741, p = 0.021\]. As shown in Figure 6, the amplitude of P1 for /dada/ was significantly smaller than P1 amplitude for /daba/ and /daDa/. The effect of S1 versus S2 stimulus presentation was marginally significant \[F(1,20) = 4.309, p = 0.051\], and was explained by a larger mean P1 amplitude for S2 condition compared to S1 condition. A significant interaction between S1 versus S2 stimulus presentation and stimulus condition was also revealed for P1 amplitudes \[F(2,20) = 5.350, p = 0.014\]. Figure 6 shows that P1 amplitude elicited to S1 was larger than P1 elicited to S2 for /dada/, while the opposite pattern was found for /daba/ and /daDa/. Post hoc comparisons using the Newman-Keuls test showed that the amplitudes of S2 P1 were significantly larger for /daDa/ versus /dada/ \((p=.003)\) and marginally significantly larger for /daba/ versus /dada/ \((p =.055)\).

In contrast to P1, N1 had the largest mean amplitudes elicited to both S1 and S2 across the stimulus conditions (Table 5), indicating its prominence in the morphology of
the slow cortical responses recorded in the infants. Although the amplitude of S2 N1 was larger for /daba/ than for /dada/ and /daDa/ by visual inspection, two-way ANOVA tests did not reveal any significant effect for N1 amplitudes.

Table 6 shows the results for one-way ANOVAs performed to compare the mean amplitudes and latencies for P1 and N1 elicited to S1 and S2 for the three stimulus conditions in separate analyses. There were no significant differences across stimulus conditions for either amplitudes or latencies except for the amplitudes of S2 P1, which was explained by larger S2 responses for /daba/ and /daDa/ as mentioned earlier.
Figure 6. Infant: Baseline-to-peak amplitude of P1 component elicited to the S1 and S2 portion of the /dada/, /daba/ and /daDa/ stimulus conditions are shown.
Figure 7. Infant: Baseline-to-peak amplitude of N1 component elicited to the S1 and S2 portion of the /dada/, /daba/ and /daDa/ stimulus conditions are shown.
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<td>.369</td>
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*significant (p < 0.05)

Table 6: Comparisons of amplitude and latencies for the P1, N1 and P2 components of the slow cortical response to S1 and S2 elicited by /dada/, /daba/ and /daDa/ in infants using one-way analyses of variance.
4 Discussion

4.1 Slow Cortical Response to S1

The slow cortical response is an obligatory response that reflects the neural encoding of an auditory signal. In the present study, cortical responses elicited to S1 in adults were consistent with typical characteristics of the slow cortical response reported in the literature in terms of morphology, amplitude, and latency. A robust P1-N1-P2 sequence of waveforms were identified in most of the cases starting about 50 ms after stimulus onset and continuing to around 300 ms, and the overall pattern of the slow cortical response was similar across the three stimulus conditions. P1 occurred approximately 66 ms after the onset of S1 with the smallest amplitude among the three components. The most prominent peaks were N1, which appeared approximately 110 ms after stimulus onset, and P2, a second positive peak occurred at around 183 ms. These findings are in agreement with a great deal of the previous work in adult slow cortical response (Geissler, et al., 1958; Ponton, et al., 2002; Wunderlich & Cone-Wesson, 2006; Näätänen & Picton, 1987; Näätänen, 2000).

Like adults, the slow cortical response to S1 was present in most infant participants and the morphology, amplitude, and latency were similar for all three stimulus conditions. A robust P1 was recorded in all cases and most infants also had a clear N1 and P2 component. In fact, the infant response resembled the P1-N1-P2 complex recorded in the adult participants in terms of the morphology of the waveforms and the relative prominence of the peaks. However, the latency of the component elicited to S1 tended to be significantly prolonged compared with adults, suggesting the immaturity of the cortical response and the underlying auditory central nervous system. This finding
was inconsistent with what was reported in the literature where young infants tended to have a biphasic waveform with a large positive and negative peak (Little et al., 1999; Molfese, 2000; Wunderlich & Cone-Wesson, 2006) but accorded with the observation by Small and Werker (2012). Although many studies have investigated the maturation of cortical components in infants and children, different paradigms and parameters have resulted in conflicting results thus requiring more systematic examination (Čeponienė, Rinne, & Näätänen, 2002; Eggermont & Ponton, 2003; Ponton et al., 1996b, 2000, 2002; Sharma et al., 1997; Surwillo, 1981). For example, it is suggested that the P1-N1-P2 complex is the typical slow cortical response in adults, while these responses in children are dominated by P1 and N2 peaks. Some studies indicate that P1 is the most predominant peak in early childhood (1–4 years) (Kushnerenko et al., 2002), and the N2 becomes increasingly robust (3–6 years) and dominates the cortical response until adolescence. Some researchers claim that N1 can be recorded in addition to the N2 peak from about three years of age with slow stimulation rate (Paetau et al., 1995; Sharma et al., 1997), while other suggest that N1 can only be reliably evoked when children reach 9–13 years (Ponton et al., 2000; Sharma et al., 1997). It is likely that variations in the stimulation rate underpin some of the differences in the research findings. ISIs can have profound effects on the morphology, amplitude and scalp distribution of the cortical response. Increasing the ISI up to at least 10 s results in larger amplitudes of N1 and P2 (and their magnetic counterparts) (Czigler et al., 1992; (Picton, Woods, & Proulx, 1978). When the ISI is decreased to less than 300 ms, the amplitude of N1 is usually diminished and may not be readily detected in some cases (Näätänen & Picton, 1987). The ISI interacts with most other stimulus variables such as amplitude and latency to affect the
parameters of cortical components (Picton et al., 1978). Some researchers suggest that only with longer ISIs could an adult-like N1 component be recorded in children (Čeponienė, Cheour, & Näätänen, 1998). An early study reported that a systematic decrease in the latency of the N1 component occurred with an increase in ISIs (from 250 to 1000 ms) for children aged 9−13 years, but not for adults. Researchers have suggested that for N1 to be evoked in children, auditory stimuli need to be presented with ISIs of 1 s or longer (Čeponienė, Cheour, & Näätänen, 1998; Paetau et al., 1995). This is most likely due to the longer N1 recovery cycle in children and its overlap by robust P1 and N2 peaks since the refractory properties of underlying neural components involved in N1 response may not have been fully developed in children. Wunderlich et al. (2006) examined the maturation of cortical auditory evoked potentials using the speech stimulus “bad”, which was 200 ms in duration with a median ISI of 3100 ms (range = 3100−6050). The stimulus duration and ISIs were longer than those used in other studies because the stimulus parameters were optimized to elicit P1 and N1 component with enhanced morphology. With longer stimulus duration and ISIs, they were able to elicit N1 in infants, but it was much smaller compared with P2 and N2 and did not resemble slow cortical responses in adults. The ISI used in the present study (2200 ms) was shorter than that of Wunderlich et al. (2006), but adult-like P1-N1-P2 responses were recorded with delayed latencies (i.e., 66 to 137 ms later in latency compared with the adult group). The morphology of the infant cortical response in the present study replicated that of Small and Werker (2012), suggesting that adult-like slow cortical responses can be elicited at four months of age given optimized stimulus parameters; however, the cortical components recorded in the
infants might not reflect neural activity from the same cortical sources that generated the cortical components in adults.

### 4.2 Acoustic change complex (ACC) to S2

The acoustic change complex (ACC) is the obligatory cortical response elicited to a change in an ongoing sound (Martin & Boothroyd, 1999). The aim of the study was to investigate the effect of long stimulus duration on ACCs elicited to speech contrasts in four-month-old infants based on a previous study by Small and Werker (2012). In the present study, the acoustic change from S1 to S2 (i.e., from /da/ to /da/, /ba/, and /Da/) was reliably elicited for /dada/, /daba/, and /daDa/ in adults and occurred at a mean latency of 499 ms. The P1 component was present in all adult participants, while N1 was recorded in the majority of cases. The amplitude of P2 was larger than that of other ACC components. Although the differences in ACC magnitude across stimulus conditions were not statistically significant, the grand mean ACC tended to be larger for /daDa/.

Small and Werker (2012) found that the magnitude of the ACC was larger for /daba/. In both studies, the amplitude of the ACC for the control condition, /dada/, was smaller than that of other two experimental conditions for adults. ACCs were also successfully elicited to the onset of S2 token for all three stimulus conditions in the infant group. Robust P1 and N1 components were present in the majority of the participants, while fewer infants had a clear P2. For /dada/ condition, most of the infant participants had the different components of the ACC, which were similar to the P1-N1-P2 complex recorded at the onset of S1 token. Fewer infants had all three components of the ACC in response to /daba/ and /daDa/, and the morphology of the ACC elicited to these stimuli was more
variable compared with ACCs to /dada/; in these cases, a broad negative peak or positive peak occurred at approximately 729 to 1015 ms instead of distinct N1 and P2 peaks appeared earlier in the waveform between 589 and 920 ms. The amplitude of P1 elicited to the second token of the control condition /dada/ was significantly smaller compared with that of P1 to the experimental condition /daba/ and /daDa/, indicating that the brain discriminated between different S2 tokens.

As mentioned earlier, this study was a follow-up to the previous work by Small and Werker (2012). Our findings confirm that an ACC to a change within a speech stimulus can be successfully recorded in four-month-old infants. Our findings also suggest that by extending the stimulus length, allowing longer time to accommodate the longer neuronal refractory period for infants, better-defined components of the ACC can be recorded and the overall morphology of the grand mean waveforms are improved. Small and Werker (2012) reported that only the infant ACC elicited to /daba/ consisted of P1, N1, and P2 components in their study, while the cortical response to the S2 of /dada/ and /daDa/ were comprised primarily of broad positive and negative peaks. In contrast, with longer stimulus duration, clear P1 and N1 components of the ACC were elicited to all three stimulus conditions for infants in the present study. Research has shown that age-related changes in myelination, synaptic refinement and cortical fiber density underlie the maturation in latency, amplitude and refractoriness of the cortical component (Huttenlocher & Dabholkar, 1997; Moore & Guan, 2001). The formation of myelin along the axon increases the conduction velocity of a signal in transmission, and consequently affects the timing of subsequent signal propagation (Salamy, 1978). Because the latency and synchrony of the neuronal signal are affected by myelination, the evoked potential
will have shorter latency, increased amplitude, and more defined waveform morphology with maturation (Musiek et al., 1988). Incomplete myelination and synaptogenesis will lead to longer neuronal refractory periods and lower cortical excitability in the immature central auditory system (Surwillo, 1981). Therefore, our results suggest that long-duration stimuli are needed for revealing robust cortical responses in infants and young children.

Our findings support that the infant’s brain can detect a change in the stimulus from /da/ to /da/, /ba/, and /Da/. Moreover, the larger P1 amplitudes recorded for /daba/ and /daDa/ may suggest that the brain has noticed that the acoustic change from /da/ to /da/ was smaller than the change from /da/ to /ba/ and from /da/ to /Da/. In our hypothesis, we had predicated that both /daba/ and /daDa/ would have larger magnitude of ACCs with distinct components compared with /dada/ because behavioral studies had shown that English-learning infants under six months of age were able to discriminate the non-native /daDa/ contrast (Werker et al. 1981; Werker & Tees 1984; Werker & Lalonde 1988). Our findings revealed that the P1 amplitude elicited to S2 of the experimental condition /daba/ and /daDa/ was indeed significantly larger than that of the control condition /dada/, which supported our hypothesis. This result is consistent with other research findings, which have shown that speech tokens can evoke distinct neural response patterns - for example, synthesized voiced tokens have been reported to evoke responses that are larger in amplitude when compared with responses evoked by voiceless stimuli (Steinschneider et al., 1999; Tremblay et al., 2003). Interestingly, our adult group did not show the same significant difference in the magnitude of ACC components for different conditions found in infants. A possible explanation for this may be the setting of the stimulus parameters. First, the speech stimulus used in the present
study consisted of two consonant-vowel structures (CVCV), which was different from the typical CV (e.g. /da/) or VV (e.g. /ui/) stimulus used for this type of research. Although we only focused on the acoustic change between two CV syllables, the brief transition from consonant to vowel within a CV token could have also evoked cortical responses resulting in overlapping cortical waveforms thus affecting the overall morphology of the ACC. The stimulus parameters of the present study were optimized to accommodate the longer refractory period of young infants; thus, the long ISI (2200 ms) and stimulus duration (816 ms) might be merely enough for evoking the dominant ACC response to the acoustic change between the CV syllables, while cortical responses evoked by the brief change from the consonant to the vowel within a CV syllable might have been overridden or cancelled out by larger responses. On the other hand, because the ISI and stimulus duration were too long for adult participants, the resultant cortical responses evoked to the complex speech stimuli might overlap or cancel out each other, resulting in less reliable waveforms and smaller magnitudes.

In the present study, we also hypothesized that the ACC elicited to /daba/ would be similar to the cortical response to /daDa/ since /ba/ and /Da/ represent CVs that are separated from /da/ on a continuum of formant frequencies by an equal number of steps. Our data, however, showed that although the magnitude of ACC for /daba/ was not significantly different from that of ACC for /daDa/, fewer participants had the cortical components indicating the onset of S2 in /daDa/ when compared with /daba/. A possible explanation for this might be the participants’ unfamiliarity with the Hindi /Da/ since they had more exposure to /ba/ in their ambient language. Therefore, auditory experience might have played a role on discrimination of the phonemes. In fact, some behavioural
studies suggest that although young infants can detect both native and non-native acoustic-phonetic features of speech, they have a preference for their native language. The distinction between discrimination versus preference indicates the influence of language experience on determining an infant’s perceptual sensitivity for a particular phonetic feature (Yeung, Chen, & Werker, 2013; Narayan et al., 2010).

There are some limitations to the present study. Due to the short attention span of infants and limited test time, each infant participant was only assigned to one stimulus condition, which might have introduced individual variance into the data. It is possible that the variability in ACC morphology for /daba/ and /daDa/ could have resulted, in part, from the variability between the participants in those two conditions because the participants were not equivalent with respect to individual difference variables; that is, the infants in /daba/ condition might have shorter refractory periods than the infants in /daDa/ condition and thus have better morphology and more robust components of their ACCs. As a result, individual cortical responses could not be compared across stimulus conditions and individual difference factors could not be teased out.

Future studies should aim to replicate the findings of the present study by using a different set of stimuli. Numerous studies on early speech perception have shown that infants categorically discriminate speech sounds suggesting that infants are born with a perceptual system that is tuned to detect acoustic-phonetic properties important for identifying phonemes in languages (Eimas et al., 1971; Werker & Tees, 2005). Speech sounds /ba/, /da/, and /ga/ have often been used to investigate the phonetic feature of place of articulation for consonants (Liberman et al., 1957; Eimas et al., 1971). Acoustically, these three consonants are distinguished by the duration and direction of the
second formant (F2) transition to the following vowel, with /ba/ having the shortest, /da/ having the medium-length, and /ga/ having the longest transition. Therefore, by using the starting frequency of the F2 transition to create a continuum of synthesized speech sounds varying in the place of articulation from /ba/ to /da/ to /ga/ with equal steps, the acoustic change within /baba/, /bada/, and /baga/ can be examined. We hypothesize that the magnitude of ACC for /baga/ will be larger than that of ACC for /bada/ and /baba/ because the acoustic change from /ba/ to /ga/ is much larger than the change from /ba/ to /da/ and from /ba/ to /ba/. The ACC components for /baba/, on the other hand, should have the smallest amplitude because this condition has the least acoustic change from S1 to S2. Therefore, by creating three levels of F2 transition with equal steps, the magnitude of cortical components elicited to an acoustic change within a speech contrast can be examined in a more systematic way. In addition, since /ba/, /da/, and /ga/ are all native and familiar to English-learning infants, the morphology of ACC elicited to /bada/ and /baga/ is expected to be similar; thus, the auditory experience issue which might have affected the results of the present study may be avoided. In addition to speech sounds, non-speech stimuli such as pure tones can also be used for eliciting ACCs. Speech stimuli represent highly complex time-varying signals, which evoke multiple overlapping neural response patterns. In contrast, non-speech sounds allow the researcher to control certain aspects of the stimulus in a more rigorous manner and vary one feature of the stimulus at a time. Thus, the cortical responses elicited by non-speech stimuli may be more reliable compared with those evoked by speech sounds. Tonal stimuli such as 250, 1000, and 4000 Hz can be used to create three stimulus conditions – 250 Hz tone alone, change from 250 to 1000 Hz tone, and change from 250 to 4000 Hz tone. Research has shown
that cortical potentials to frequency change differ between low and high frequencies (Alain, Woods, & Covarrubias, 1997; Dimitrijevic, Michalewski, Zeng, Pratt, & Starr, 2008). Alain et al. (1997) found that N1 amplitude increased with stimulus duration and was greater for low than high frequencies suggesting a frequency dependence on the temporal window of integration. The 250 Hz tone alone can be served as the control condition for baseline comparison with the other two conditions. Since the acoustic change from 250 to 1000 Hz within a continuous tone is smaller than the change from 250 to 4000 Hz, the magnitude of ACC components evoked in infants is expected to be largest for the acoustic change from 250 to 4000 Hz and the amplitude for the control condition (i.e. 250 Hz alone) to be the smallest, while the ACC magnitude is expected to be of medium size for the 250 to 1000 Hz condition. As a result, the relationship between the amplitude of cortical components and the magnitude of acoustic change within a stimulus can be established.
5 Conclusion

The purpose of the present study was to examine the effect of long stimulus duration on ACCs elicited to speech contrasts in four-month-old infants. Our results support the findings by Small and Werker (2012) showing that adult-like slow cortical responses can be elicited in four-month-old infants given optimized stimulus parameters (e.g. longer ISIs and stimulus duration). However, it is important to note that although the morphology of the infant cortical response resembled that of P1-N1-P2 complex elicited to the onset of a stimulus in adults, the cortical components recorded in the infants may not reflect neural activity from the same cortical sources in adults due to the immaturity of the auditory central nervous system of infants. The most important finding of the present study is that an ACC to a change within a speech stimulus can be successfully recorded in young infants. By extending the stimulus length and allowing longer time to accommodate the longer neuronal refractory period for infants, better-defined components of the ACC can be elicited and the overall morphology of the grand mean waveforms is enhanced. Our ACC results also suggest that distinct neural response patterns may be elicited to acoustic changes with different magnitude: in the present study, ACC components had larger amplitudes in response to a larger acoustic change within a stimulus. To confirm that the ACC is sensitive to subtle acoustic changes in speech, more research needs to be undertaken to compare different neural detection patterns underlying speech perception. As a technique in development, the ACC may hold promise for providing insight into the infant brain’s capacity to discriminate the acoustic features of speech. Because the ACC can be recorded reliably in individual
infants, it also has the potential to measure speech perception ability in infants and young children in a clinical setting.
References


Miyazaki, T., Thompson, J., Fujioka, T., & Ross, B. (2013). Sound envelope encoding in the auditory cortex revealed by neuromagnetic responses in the theta to gamma frequency bands. *Brain Research,*


Appendices

Appendix A – Adult individual data

Baseline-to-peak peak latency (Latency) and amplitude (Amp) measurements for individual components of the waveform elicited to the S1 and S2 portion for each individual subject.

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Appendix B – Infant individual data

Baseline-to-peak peak latency (Latency) and amplitude (Amp) measurements for individual components of the waveform elicited to the S1 and S2 portion for each individual subject.

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CONSENT FORM
Adult ERP

Project Title: “Speech Perception and Early Auditory Experience”

Principal Investigator: Dr. Susan A. Small, Professor, School of Audiology & Speech Sciences, University of British Columbia, Phone: 604-822-5696
Co-Investigator: Dr. Janet F. Werker, Professor, Department of Psychology, University of British Columbia, and Canada Research Chair, Phone: 604-822-6408

Purpose: We are interested in understanding how the brain functions and how it develops in infants with normal and impaired hearing. We are assessing adults with normal and impaired hearing who have ‘mature’ speech perception skills so that we can investigate new techniques for assessing discrimination of speech sounds in infants.

Study Procedures:
If you agree to participate in this study, the following will require about 1.5 hours of your time:

1) You will be asked to either wear individual electrodes or a cap with holes in it with wires coming out the top. The holes will be filled with electrode gel, a gooey substance that might feel cool on your head. There are no known risks and you will not experience any pain or discomfort. There may be a bit of gooey gel left in your hair when the cap is taken off, but it will wash out when you shampoo your hair. You can stop at any time without penalty.
2) You will sit in the testing room that you have been shown and listen to words, sounds, or both words and sounds. You may also be asked to watch a movie with captioning while listening to words or sounds.
3) Brain waves will be recorded from your scalp while you listen to words or sounds. The brain waves will show how your brain works when you hear and think about these sounds. Sometimes you will be asked to push a button when you hear certain sounds.
4) A hearing screening test may be conducted using a behavioural hearing test. This test involves raising your hand or pushing a button when you hear soft sounds.

Confidentiality:
Your identity will be coded using a code known only to the researchers and that all information that is collected about you will remain confidential. Only group results or coded individual results will be given in any reports about the study. Only coded results (no personal information) will be kept in computer files on a password protected hard disk.

Thank you: To show our appreciation, you will receive a $15 honorarium.
Contact:
If you have any questions or desire further information with respect to this study, you may contact Dr. Susan Small at 604-822-5696 or Dr. Janet Werker at 604-822-6408.

If you have any concerns about your treatment or rights as a research subject, you may contact the Research Subject Information Line in the UBC Office of Research Services at 604-822-8598 or if long distance e-mail to RSIL@ors.ubc.ca.

Consent:
My signature below confirms that I DO consent participating in this study as has been described above.

I understand that my participation in this study is entirely voluntary and that I may refuse to participate or withdraw from the study at any time without consequence. I have received a copy of this consent form for my own records.

______________________________________________    _______________________
Name of participant (print)                           Date

______________________________________________
Signature of participant
Appendix D – Infant consent form

The University of British Columbia
School of Audiology and Speech Sciences
Faculty of Medicine
2177 Friedman
Vancouver, B.C. Canada V6T 1Z3
Phone: (604) 822-5591, Fax: (604) 822-6569

CONSENT FORM
Infant ERP

Project Title: “Speech Perception and Early Auditory Experience”

Principal Investigator: Dr. Susan A. Small, Professor, School of Audiology & Speech Sciences, University of British Columbia, Phone: 604-822-5696
Co-Investigator: Dr. Janet F. Werker, Professor, Department of Psychology, University of British Columbia, and Canada Research Chair, Phone: 604-822-6408

Purpose: We are interested in understanding how the brain functions and how it develops in infants with and without hearing loss.

Study Procedures:
The study will take between 1.0-1.5 hours.

(i) A hearing screening test may be conducted using otoacoustic emissions. This test involves placing a soft rubber-tipped probe in the outer ear canal and presenting soft sounds. The test takes a few minutes if the infant is sitting quietly or asleep.

(ii) An appropriately fitted elastic cap with small metal disks (electrodes) sewn into it will be placed on your infant’s head and will be removed at the end of the test session. Electrodes may also be positioned behind each ear, beside each eye, and beneath one eye on the cheek. The skin will be cleansed before the electrodes are applied and after they are removed. A small amount of electrode gel may be applied to the scalp. Under rare circumstances, infants with very sensitive skin may react to the application of electrode gel or medical tape. A small red mark may be apparent at one or more of the electrode locations. It has been our experience that this reaction is very rare (i.e., 1 in 200 infants) and goes away quickly.

(iii) Your infant will sit on your lap in a dimly lit room and will hear different types of auditory and/or visual stimuli (e.g., speech sounds, images). A research assistant will entertain your infant by putting on a puppet show, or a movie (without sound) will be shown. This will help your baby sit relatively quietly while listening or watching the stimuli. You may be asked to complete one or more questionnaires which may take up to an hour to complete.

(iv) Brain waves will be recorded from your infant’s scalp while s/he listens to sounds and/or looks at images on a screen. The brain waves will show how your infant’s brain works when s/he thinks about the stimuli that are presented.
(v) Prior to the study, you may be mailed a vocabulary questionnaire to fill out, which may take up to an hour to complete. You may be asked to bring this with you to the study. If you have already filled out vocabulary questionnaires, you will be asked to consent to providing access to these questionnaires for the purpose of using the results in this study. You will also be asked a set of questions regarding your infant’s language exposure.

(vi) If your infant begins to cry or fuss excessively, we will likely stop the study as we do not wish to upset your infant.

Confidentiality:
Your infant’s identity will be coded using a code known only to the researchers and that all information that is collected about your infant will remain confidential. Only group results or coded individual results will be given in any reports about the study. Only coded results (no personal information) will be kept in computer files on a password protected hard disk.

Thank you: To show our appreciation, you will receive a t-shirt for your infant to take home. As well, you will be reimbursed for your parking expenses while visiting our facility.

Contact:
If you have any questions or desire further information with respect to this study, you may contact Dr. Susan Small at 604-822-5696 or Dr. Janet Werker at 604-822-6408.

If you have any concerns about your treatment or rights as a research subject, you may contact the Office of Research Services at the University of British Columbia at 604-822-8598.

Consent:
My signature below confirms that I DO consent to having my child participate in this study as has been described above.

I understand that my participation in this study is entirely voluntary and that I may refuse to participate or withdraw from the study at any time without consequence.

________________________  ____________________  __________________
Parent/Guardian Name (print)   Date

Name of Child (print)   Age

Parent/Guardian Signature

☐ I filled out vocabulary questionnaires about my child prior to this study. I consent to providing access to these questionnaires for the purpose of using the results in this study. A check mark in the box confirms my consent.