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Mismatch negativity to speech stimuli in 8-month-old infants and adults

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Abstract

The mismatch negativity (MMN) was measured in 15 normal awake 8-month-old infants and 10 adults to the speech consonants /da/ and /ta/. ERPs were analyzed at 11 electrodes (Fz, Cz, Pz, C3, C4, T3, T4, T5, T6, P3, P4). Four-hundred trials were presented: the /da/ standards with 80% probability and the /ta/ deviants with 20% probability. The ISI was 600 ms.

An MMN was observed for both adults and infants but with different scalp distributions. A clear infant MMN was observed only at C3 and T3 electrodes, whereas the adult MMN was present at Fz, Cz, C3, C4 and Pz. A repeated-measures ANOVA on the normalized summed area between 200 and 250 ms revealed an age (adult vs. infant) × electrode interaction. Paired *t*-tests indicated that adults and infants showed significant differences at the C3, Cz, T3, Pz and T6 electrodes. The adult MMN was largest at Cz and C3 whereas the infant MMN was largest at T3. These data are discussed in terms of possible maturational changes in the MMN.

1. Introduction

The acquisition of language occurs rapidly over the first few years of life and there has been considerable behavioral research on early language development. Although this research has documented the ability of young infants to discriminate fine differences between speech sounds (Jusczyk, 1992; Best, 1995; Werker and Desjardins, 1995), less is known about the development of the brain mechanisms that underlie the acquisition of language. More recently, this issue has been examined by the measurement of event-related potentials (ERPs). ERPs are a powerful tool in the study of developmental neurophysiology, as not only does the measurement of ERPs provide precise temporal information, but it also can give a spatial description allowing localization of the relevant processes in the brain.

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These characteristics are advantageous in tracking the development of various sensory and cognitive processes (e.g. Mills et al., 1991; Taylor, 1993, 1995).

One ERP in particular, is of interest as it reflects the ability of the brain to discriminate small differences in stimuli; it is called the mismatch negativity (MMN). The MMN reflects the operation of a cerebral processor that automatically detects the presence of deviant stimuli within a train of homogenous stimuli; this cerebral processor is a finely-tuned discriminator for incoming auditory information. Although considerable research on the MMN has been conducted with adults, there have been only a few studies measuring the MMN in infants and children. As of yet, there have been no studies that have directly compared the MMN of adults to those of infants, when evoked by the same stimuli in the same experimental situation.

The current article examines MMN in normal 8-month-old infants, as compared to adults, when presented with speech sounds, where the consonant /ta/ is to be discriminated from the consonant /da/. Physically, these consonants differ only in voice onset time (VOT: the time at which the vocal chords begin to vibrate, measured from the beginning of the sound). The literature suggests that the MMN in neonates resembles that in adults but this is based on data collected at only a small number of electrode sites. We hoped to extend these data by measuring the MMN for these stimuli at a number of electrode locations in awake 8-month-old infants and adults.

1.1. Mismatch negativity (MMN) in adults

The MMN is well established and understood in adults (for a review, see Näätänen and Alho, 1995). It is elicited by deviant stimuli that are presented within a series of homogenous, or standard, stimuli and can reflect a number of changes in acoustic parameters, such as frequency (Sams et al., 1985), intensity (Näätänen and Picton, 1987), location (Paavilainen et al., 1989) and duration (Kaukoranta et al., 1989). As well, the MMN can reflect very fine discriminations as it is measurable with near threshold stimulus differences (Sams et al., 1985).

The generators of the MMN are already well known and involve sources in both supratemporal auditory cortices (Scherg et al., 1989) and frontal cortex (Giard et al., 1990). The MMN is generated automatically without the influence of attention and does not require active participation on the part of the subject (Näätänen, 1990). The MMN can be recorded while subjects are engaged in other tasks (with adults, they are often reading while being tested) and unlike many other ERP paradigms, no motor response is required. Also, the MMN is best recorded when the deviant stimuli are embedded in a rapidly presented stimulus train (Näätänen and Alho, 1995). Thus, the MMN possesses several characteristics that make it readily amenable to studies involving infants: it can be evoked without a behavioral response, it does not require conscious attention and stimuli can be presented rapidly so that many trials can be collected over a short period of time.

1.2. Mismatch negativity in infants and children

Although it has been suggested that the MMN in infants and children resembles that of adults, these data were collected from a small set of electrodes. As well, no studies have directly compared adult and infant MMNs evoked by the same stimuli and test situation. Most of the work examining the MMN in children has been done by two research groups. One group (e.g. Alho et al., 1990) has looked primarily at infants and has been attempting to determine how early in life the MMN can be evoked. Another group (e.g. Kraus et al., 1993), has compared the MMN in school-aged children to the MMN in adults, to investigate the neurophysiological bases of speech discrimination.

One of the first studies that demonstrated a negative component in infants resembling the MMN in adults, presented sinusoidal tone bursts at a rate of one tone/610 ms (Alho et al., 1990). They identified the MMN as the difference between the waveforms evoked by the standard 1000 Hz tones, which occurred with 90%

probability and the deviant 1200 Hz tones. Data were recorded from the midline scalp sites, Fz, Cz and Pz. The subjects were eight 1–4-day-old full-term infants who were in quiet sleep during the ERP recording. These researchers reported that a negative component similar to the adult MMN was elicited in these neonates with the waveform largest at Fz, smaller at Cz and essentially non-existent at Pz. The authors drew several conclusions from these results. They commented on the fundamental nature of the brain mechanism involved in the generation of the MMN since the MMN was manifested at such an early stage of life. As well, they discussed the practical applications of this finding: the MMN could be used as a method of diagnosing early brain dysfunction, since the absence of a MMN in infancy would indicate that the cerebral processing of auditory sensory input was not occurring correctly.

A later study found that a MMN could also be elicited in infants by speech stimuli (Cheour-Luhtanen et al., 1995). Similar to the earlier report (Alho et al., 1990), this study examined MMN in healthy, sleeping babies ($n = 12$) who were 1–5 days old. The stimuli presented were Finnish vowels, the standard (/y/), presented 80% of the time while the deviant consisted of two other vowels, each presented 10% of the time. One of these deviants was clearly in a different vowel category (/i/) than the standard, while the other was a boundary vowel (/y//i/), which was not as readily discriminable from the standard (/y/). The MMN was measured at six lateral electrodes: F3, F4, C3, C4, P3 and P4, which were chosen to maximize the possibility of observing hemispheric differences. A negativity was seen which peaked between 200 and 250 ms in the difference waveform, obtained by subtracting the ERP to the standard stimulus from the ERP to the deviant stimulus. This negativity, presumably the MMN, was largest at the frontal and central scalp sites, but the only significant differences from the standard (/y/) were for the clearly distinct deviant (/i/) at the F3 and F4 electrodes. There were no hemispheric differences. These researchers reported that their data showed MMNs similar in morphology and distribution to the MMNs observed in adults using similar stimuli, although the MMNs obtained in the infants were smaller than those in the adult study. However, since adults and infants were not tested under identical circumstances, a direct comparison could not be made. Nevertheless, the finding that a reliable MMN was not recorded to the more difficult discrimination (/y//i/) compared to the MMN evoked to the clearly discriminant vowel (/i/), suggested that the MMN might be less sensitive to vowel changes in newborns than in adults. Cheour-Luhtanen et al. (1995) alternatively suggested that this may be due to the sleeping state of the newborns since the MMN amplitude has been seen to attenuate in sleeping adults (Nielsen-Bohlman et al., 1991). The midline chain was not measured in this study, so the data cannot be compared directly to the first investigation which used simpler, tonal stimuli (Alho et al., 1990). Such a comparison could have determined whether the difficulty of the discrimination affected the amplitude of the MMN as is seen in adults (Sams et al., 1985), or whether it was a result of the infants being tested when asleep.

The above evidence supports other behavioral studies that indicate that infants have the ability to differentiate speech sounds (e.g. Eimas et al., 1971; Jusczyk, 1992; Best, 1995; Werker and Desjardins, 1995). In light of the complexity of speech sounds and considering the subtle differences that differentiate words acoustically, these findings raise the question of which cerebral mechanisms are involved in producing this discriminative ability in neonates. Cheour-Luhtanen et al. (1996) provided evidence of the human brain's very early ability to discriminate complex sounds and demonstrated the presence of a cerebral processor underlying this discrimination in preterm babies (25–34 weeks gestational age, $n = 11$). The stimulus parameters were identical to those of their earlier experiment with the full-term infants. Data were collected from the six electrodes (F3, F4, C3, C4, P3 and P4) as previously, as well as two temporal sites (T3 and T4). The negativity observed resembled the MMN response recorded in their other studies with adults and with full-term neonates, both in morphology and scalp topography. The MMN was largest frontally and predominant in the right hemisphere. As well, the

researchers indicated that the MMN appeared to be longer in duration in infants than in adults.

Kraus et al. (1992) examined developmental changes in the MMN between children (7–11 years, $n = 10$) and young adults (17–29 years, $n = 10$) when presented with speech stimuli (/da/ and /ga/ consonants); the deviant (/ga/) stimuli occurred in 15% of trials. The MMN was recorded only from the Fz electrode referenced to the ipsilateral earlobe. An MMN was seen in both adults and children as a negative difference wave at approximately 235 ms after stimulus onset. These researchers found that the MMN latency and amplitude were not significantly different between adults and children at Fz and suggested maturation of this component was complete by school-age.

This same group (Kraus et al., 1993) repeated the above study with a deviant stimulus that was more closely related to the standard, to determine the reliability of the MMN for studies in children with auditory dysfunction. Both standard and deviant stimuli were just perceptibly different variants of the phoneme /da/. Again, they only measured the MMN at the Fz electrode. They found that there were no significant differences between the latency, duration and onset-to-peak amplitude of the MMN obtained in adults vs. that obtained in children. However, MMN areas were statistically larger for the children than the adults. The authors interpreted these data to indicate that children can process just perceptibly different variants of the same phoneme as effectively as adults and that the MMN may be more sensitive than behavioral measures. Clearly, as subtle acoustic differences in speech stimuli were reflected in children's MMNs but not in infants' MMNs (Cheour-Luhtanen et al., 1995), some maturational changes are occurring either in the learning of language stimuli and/or the MMN itself.

To further investigate possible developmental changes, this article directly compares the MMN in infants and adults to speech sound differentiation. Eleven electrode sites were used in order to better examine whether the distribution of the MMN differs between awake infants and adults.

2. Methods

2.1. Subjects

Full-term infants were recruited from the well baby maternity wards of St. Joseph's Hospital and McMaster University Medical Center in Hamilton and through contacts with friends and colleagues. Fifteen babies (11 females, four males; mean age = 8.4 months; range: 8.0–9.2 months) and 10 adults (six females, four males; mean age = 32 years; range: 26–44 years) were presented with the speech consonants. Although infant ERPs can be readily recorded, this presupposes an attentive, 'cooperative' baby. A total of 28 infants were recruited, but due to either excessive movement causing high rates of EMG artefact rejection (a minimum of 15 artefact-free trials were required) or the refusal of the infant to wear the electrode cap, only 15 infants had sufficient, usable ERP data.

2.2. ERP data collection

In the 8-month-old infants, ERPs were recorded from 12 active electrodes in a fitted ElectroCap (according to the 10–20 International System) (Fz, F7, Fp1, C3, C4, T3, T4, Pz, P3, P4, T5, T6). Adults ERPs were recorded with 26 active electrodes but only the above 12 electrodes were analyzed. For both babies and adults, all electrodes were referenced to Cz for data collection. Electrode impedances were below 5 kΩ. ERPs were recorded with a bandpass of 0.1–30 Hz on a Neuroscan 3.1 system. Data were collected continuously and epoched into 1-s sweeps, which included a 50-ms prestimulus baseline. All analyses were done off-line. Trials were baseline corrected and automatic artefact rejection was set at $\pm$ 150 μV using Fp1 and F7, then an averaged reference (Picton et al., 1995) was recalculated with the remaining electrodes. The trials were sorted according to trial type and averaged separately.

2.3. Auditory ERP task and stimuli

Four-hundred stimuli were presented in each

series. An 'oddball' paradigm was used in which 80% of the stimuli consisted of a repeated 'standard' stimulus. The remaining 20% of the repetitions consisted of a 'deviant' stimulus. The order of presentation was random with the constraint that at least two standard stimuli separated successive deviant stimuli. Generally, 400 trials gave an adequate number of target trials for averaging in a reasonably cooperative baby. The interstimulus interval (ISI) was 600 ms.

The stimuli were recorded tokens of /da/ (standard) and /ta/ (target), spoken by a fluent native speaker of English. The tokens were matched for intensity (72 dB SPL) and duration (212 ms). The VOT for /da/ was 7 ms and the VOT for /ta/ was 49 ms. The auditory stimuli were generated by a Pro Audio Spectrum 16 sound card in a Comptech 486 computer.

2.4. Procedure

Infants were held on the parent's lap facing the sound-presentation speaker. Two methods were used in attempts to minimize infant movements while the ERPs were recorded. A screen-saver routine (Disney After Dark®) was running on a Macintosh screen placed in front of the infant (with no audio); the speaker was behind and extending above the computer screen. If the infant was uninterested in the computer screen, one of the research assistants attempted to hold the baby's attention with hand puppets held in front of the screen. All infants were awake throughout the testing session. The parent and infant sat in a sound-attenuated booth at a distance of 90 cm from the speaker. The adult subjects were tested under comparable conditions; they sat comfortably in a chair facing the speaker at a distance of 90 cm and watched the same screen-saver routine. No instructions were given to adults. The entire procedure took approximately 25 min.

2.5. Data analyses

For each subject, the MMN was obtained by subtracting the averaged waveforms for the standards from the averaged waveforms for the deviants. The resultant waveform contains the MMN. Using the adult grand averages as a template, latency windows were determined and the summed area between the windows, 150–200 and 200–250ms, were calculated for each subject. Due to the greater variability and the overall higher amplitude of the infants' ERPs, the data were normalized prior to analyses. Two-way repeated-measures ANOVAs for age (adult vs. infants) × electrode (Fz, Cz, C3, C4, T3, T4, Pz, P3, P4, T5, T6) were performed on the normalized summed area measurements for each of the two latency windows.

3. Results

The average number of artefact-free deviants (/ta/) collected was 35 for babies and 46 for adults; for standards (/da/), it was 191 and 255 for babies and adults, respectively. Fig. 1a and Fig. 2a show the grand-averaged standard and deviant waveforms for the adults and infants, respectively.

Fig. 1b shows the grand-averaged difference waveforms for adults. The MMN was clearly observable as a negative peak with a fronto-central distribution (Fz, C3, Cz and C4), although it was also present at Pz. It had an onset of 200 ms and a duration of approximately 100 ms. The MMN at C3 appeared larger in area than the MMN at C4 and this was confirmed by a paired t-test at these two electrodes in the 200–250 ms latency window ($t(9) = -4.18$, $P = 0.002$).

Fig. 2b shows the grand-averaged difference waveforms for infants. An MMN-like waveform was observable at C3 and T3. An MMN was not evident at Fz or Cz and parietally there was no negative wave at all, only a large positive wave at Pz. As well, a large late negative wave was observed at T6. The MMN had an onset of approximately 200 ms and a duration of 100 ms at C3 and 150 ms at T3. Although there was a negative peak at C4 in the infants, it was from 100–200 ms (i.e. with an offset at the same latency as the onset of the MMN in adults), we felt that it was too early to be considered an MMN. In the 200–250 ms window the ERP was positive in the infants at C4, yet no significant difference was found between C3 and C4.

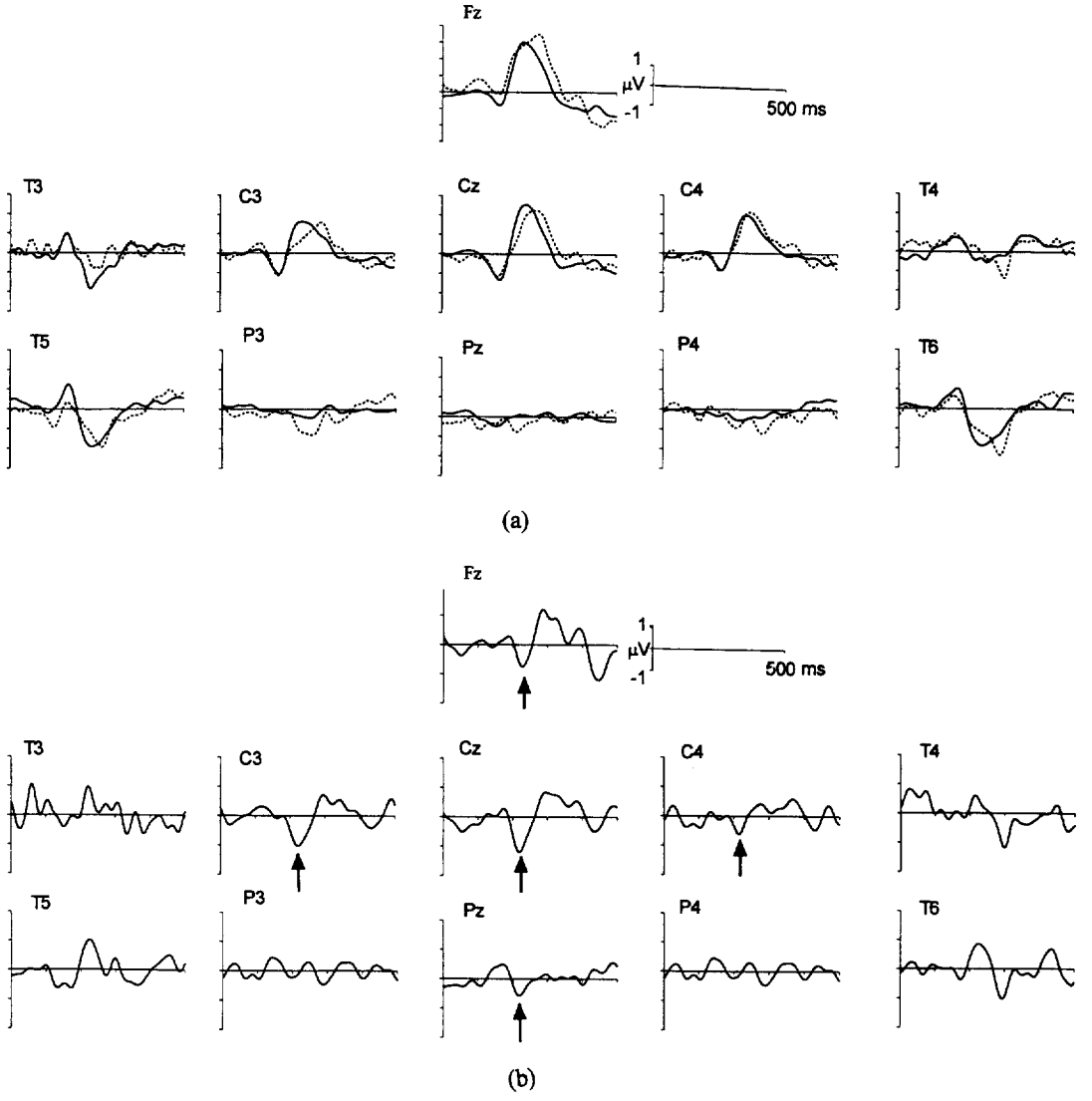

Fig. 1. (a) Grand-averaged adult waveforms to /da/ standards (solid line) overplotted with /ta/ deviants (dotted line). (b) Grand-averaged difference waveforms for adults. The MMN is noted by the arrows.

The two-way repeated-measures ANOVA for age × electrode did not show any effects for the 150–200 ms latency window; however, for the 200–250 ms latency window, there was a main effect for age ($F_{1,21} = 5.07$, $P = 0.035$) and an age × electrode interaction ($F_{10,210} = 4.54$, $P = 0.001$). To examine the age × electrode interaction in the 200–250 ms latency window, electrode-by-electrode comparisons between the adult and infant data were performed. Significant differences were observed between the adults and babies at the C3 ($t(22) = -4.29$, $P = 0.001$), Cz ($t(23) = -3.98$, $P = 0.001$), T3 ($t(23) = 2.47$, $P = 0.021$), Pz ($t(19) = -2.55$, $P = 0.02$) and T6 ($t(16) = 2.78$, $P = 0.013$) electrodes. The differences between adults and infants at the Pz and T6 are due to the large positivity at the infant Pz electrode and a large negative peak at the infant T6

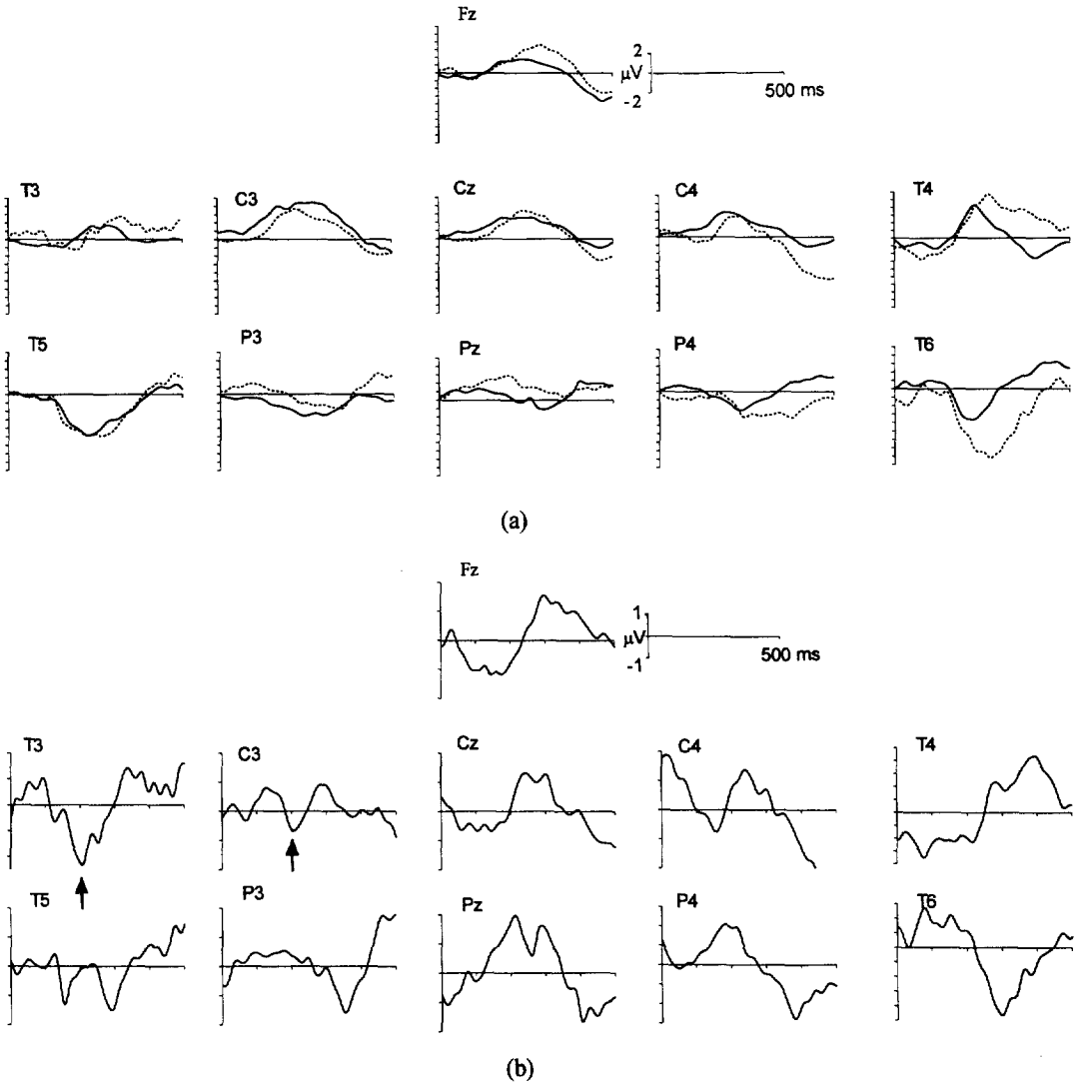

Fig. 2. (a) Grand-averaged infant waveforms to /da/ standards (solid line) overplotted with /ta/ deviants (dotted line). (b) Grand-averaged difference waveforms for infants. The MMN is noted by the arrows. The negativity observed at C4 is in the 100–200 ms latency range and is too early to be considered an MMN.

electrode; these do not appear to be related to the MMN.

The Cz, C3 and T3 electrodes for the infants vs. the adults are overplotted in Fig. 3. Fig. 3a demonstrates the presence of an MMN at Cz in adults but clearly shows that there was not a comparable MMN in infants. Fig. 3b shows that the MMN was present in both groups at the C3 electrode, although the amplitude was smaller and the duration was shorter in infants. Fig. 3c shows that an MMN-like waveform is clearly observable in the babies at T3, but there was no negativity at that latency in adults.

4. Discussion

The findings of the present study confirm and extend the results of previous developmental

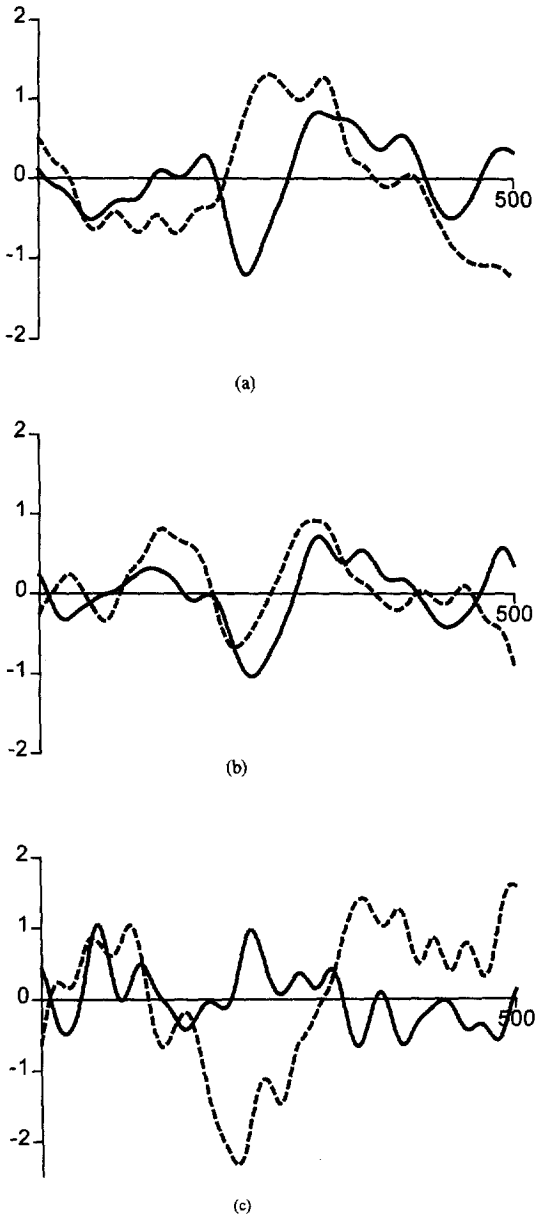

Fig. 3. (a) The difference waveform for adults (solid line) vs. infants (dotted line) at the Cz electrode. (b) The difference waveform for adults (solid line) vs. infants (dotted line) at the C3 electrode. (c) The difference waveform for adults (solid line) vs. infants (dotted line) at the T3 electrode.

studies on the MMN. Our results are somewhat consistent with other studies using speech consonant stimuli in infants (Cheour-Luhtanen et al., 1995, 1996), children (Kraus et al., 1992, 1993) and adults (Maiste et al., 1995). All of these studies found the MMN to be present to speech stimuli, seen as a negative waveform that peaked at approximately 200–250-ms. We also found that the MMNs in both the adult and 8-month-old infant data peaked in the 200–250 ms latency range, and in adults, were largest in the fronto–central regions. However, three new findings emerged from the present study.

First, the data clearly demonstrated that although the MMN was present for these stimuli at C3 and T3 for infants, it was not evident at the midline chain or over the right hemisphere. The morphology of the infant waveform at Cz was quite different from the clear-cut adult MMN observed at this electrode (Fig. 3a). Conversely, Fig. 3b indicates that the adult and infant MMN at C3 are very similar in morphology, but the infant MMN is smaller. The smaller MMN amplitude in infants is consistent with other data (Cheour-Luhtanen et al., 1995), but the distributional data are not. The significant differences between groups at Cz and C3 suggest that the generators of the MMN undergo marked maturational changes, as is seen in other sensory and cognitive ERPs (e.g. Taylor, 1993, 1995). This is in contrast to the extant developmental MMN literature, which suggests that the MMN is very similar in infants and adults; however, in infant studies, the midline chain was not recorded and in children's studies, only Fz was recorded.

Second, the significant asymmetry between C3 and C4 in adults suggests that there is a left hemisphere bias when processing speech sounds. The absence of a significant asymmetry between C3 and C4 in infants would appear to suggest that infants have not yet developed this specialization; however, examination of Fig. 2b shows that the MMN is present at C3 but is not evident at C4 in the appropriate latency window. The C3 vs. C4 comparison was not significant in the infants and we postulate that this may be due to both the large variability found in infant data and the small amplitude of the C3 MMN. If, in fact, the infant MMN is present at C3 and absent at C4, this would suggest a differential development of the generators underlying the processing of speech

sounds. It may be that there is a left hemisphere specialization that predisposes the generator in the left hemisphere to develop first and to specialize to speech and this is indicated by the presence of an MMN at C3 in infants as well as a larger MMN at C3 in adults.

Finally, consistent with the C3 vs. C4 data, the findings at the T3 electrodes were particularly intriguing. In the infants there was a large MMN at T3, whereas in the adults there was a positive component at that latency (see Fig. 3c). A recent report by Bruneau et al. (1997) showed that children displayed maximal amplitude auditory ERPs at the temporal sites and poor responses at the midline, whereas adults displayed fronto–central midline maxima. This is consistent with the present data which showed that in adults (Fig. 1b) the MMN is largest at the vertex and decreases in amplitude as one moves laterally. Infants, on the other hand (see Fig. 2b), displayed maximal MMN amplitude at the left temporal site and this decreased as one moved medially. One would speculate that the temporal generator in the left hemisphere is in a more vertical, or even radial, orientation in the infants and hence, the MMN is maximal at the T3 and C3 electrodes. With utilization of other types of stimuli that are non-speech, we could determine if the generator in the right temporal area was more in evidence. In adults, the bilateral dipoles are medial in orientation (Scherg et al., 1989) such that the maximal responses are midline.

There may be several reasons for the marked differences in the distribution between our data and that of Cheour-Luhtanen et al. (1995, 1996). They found the MMN in term neonates larger frontally, but reasonably symmetrical (although Fig. 3 shows a greater difference at F3 than F4, this was not significant), whereas in preterm neonates the MMN was larger over the right frontal–central leads. Perhaps there is a shift very early in life from right through symmetrical to left sided asymmetries in the MMN to speech stimuli, but that cannot be determined from comparisons of current data across the different studies and labs. A more likely explanation for our findings of asymmetry in the 8-month-old infants may be due to our use of an averaged reference. Picton et al. (1995) has shown that with auditory stimuli the average reference accentuates lateral asymmetries, compared to ear or mastoid references. A study that includes recordings from 32 electrodes using averaged and ear references in infants across these age ranges could resolve this question, but would be difficult to implement.

In summary, the findings from this study suggest that the neural systems utilized in language processing do show developmental changes. Not only does there seem to be a shifting in orientation of the generators underlying the processing of speech sounds, but there may also be a differential maturational timeline for the two hemispheres. This is particularly interesting since it is contrary to the current understanding of the developmental course of the MMN. Clearly, there is a need for future studies utilizing a greater number of electrodes in more closely spaced age groups with the presentation of both speech and non-speech stimuli so that these maturational effects can be tracked.

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