

Left temporal impairment of auditory information processing in prematurely born 9-year-old children: An electrophysiological study

Marie Gomot ^{a,*}, Nicole Bruneau ^a, Jean-Paul Laurent ^b, Catherine Barthélémy ^a, Elie Saliba ^c

^a INSERM U619, Université François-Rabelais, CHRU de Tours,

Service Universitaire d'Explorations Fonctionnelles et Neurophysiologie en Pédiopsychiatrie, Tours, France

^b Equipe de Recherche en Psychologie Clinique, Université de Paris 8, Saint-Denis, France

^c INSERM U619, CHRU de Tours, Service de Médecine Néonatale, Tours, France

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Abstract

Children born preterm more than average display cognitive difficulties that are significant enough to prevent normal schooling. The aim of our study was to provide better understanding of the long-term neuropathological processes associated with preterm injury, through the hypothesis that mild cognitive disorders might be related to slight deficits in primary functions such as attention and perception. Assessment of auditory pre-attentive processes was performed by recording the obligatory sensory response (N250) and the change-detection response (Mismatch Negativity, MMN). Topographic study of these responses was performed in fifteen 9-year-old children born preterm (27–33 weeks gestational age) matched to fifteen control children born at term. The auditory stimulus sequence consisted of 1000 Hz standard and 1100 Hz deviant tones (15%) delivered binaurally with an interstimulus interval of 700 ms. The results showed that MMN was similar in both groups. Analysis of the responses to standard repetitive tones demonstrated significantly smaller N250 wave amplitude in children born preterm. Scalp current density maps showed that this reduction in amplitude was associated with lower activity of both frontal and left supratemporal generators. Although the functional significance of the N250 wave in children remains to be clarified, our results indicate a disorder of auditory processes related to prematurity that might have consequences on the development of higher-level processes.

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1. Introduction

This study focuses on the long-term neuropathological processes associated with preterm birth. Several studies have shown that children born preterm tend to do less well academically, although they perform in the normal range on full-scale IQ tests (Saigal et al., 2000; Ornstein et al., 1991). Clinical (Hille et al., 1994) and electrophysiological studies (Lavoie et al., 1997; Dupin et al., 2000) have reported attention-related deficits in these children, even in those without apparent neurological impairment. Auditory discrimination abnormalities

(Davis et al. 2001; Therien et al., 2004) and language and learning difficulties (Jennische and Sedin, 2001; Grunau et al., 2002) are also frequently observed in prematurely born children. Our hypothesis was that the cognitive problems frequently displayed by children born preterm might be related to slight disorders of primary functions such as auditory attention and perception. Auditory evoked potentials (AEP) might provide a useful tool to detect subtle neurodevelopmental abnormalities that could underlie such discrimination and learning difficulties.

The auditory electrophysiological response evoked in children by any sound occurring in the surrounding environment is characterized by a large negative wave which culminates at fronto-central sites around 250 ms and called the N250 wave (Bruneau and Gomot, 1998; Gomot et al., 2000; Ponton et al., 2000; Ceponiene et al., 2002). This response remains consistent until adolescence and it has been found to predict reading disabilities at

* Corresponding author. INSERM U619, Centre de Pédiopsychiatrie, CHU Bretonneau - 2 bd Tonnellé, 37044 Tours Cedex, France. Tel.: +33 2 47 47 86 64; fax: +33 2 47 47 38 46.

E-mail address: m.gomot@chu-tours.fr (M. Gomot).

3, 5 and 8 years of age (Molfese, 2000). Despite being characteristic of childhood, the N250 wave is understudied in clinical paediatric populations and to date only one study has been performed in children born preterm. Fellman et al. (2004) reported that N250 amplitude recorded during the first year of life was lower in prematurely born children compared to full-term born controls.

Auditory pre-attentional change-detection processes are commonly studied through the AEP response called *Mismatch Negativity* (MMN). This response is evoked by any deviant stimuli occurring in a sequence of repeated sounds, and it is thought to reflect the automatic detection of a difference between the active sensory memory trace of the recent repetitive event (the standard) and an incoming deviant stimulus (Näätänen, 1992; Näätänen et al., 2005). MMN can be recorded early in development, and mechanisms underlying this response are assumed to be similar across the lifespan (Cheour-Luhtanen et al., 1995; Gomot et al., 2000).

A few studies have been conducted on the auditory change-detection mechanism underlying MMN in children born preterm, and no group differences in MMN characteristics have been demonstrated from birth to 3 months between children born preterm and those born at term (Alho et al., 1990a,b; Cheour-Luhtanen et al., 1996; Cheour et al., 1998; Leppanen et al., 2004). Dupin et al. (2000) also reported that MMN was normal in preterm children aged 5 years at the time of recording. However, another AEP study performed in prematurely born children during their first year of life indicated that MMN was absent in these children (Fellman et al., 2004). This discrepancy in results might be due to the wide variability of responses in young children.

Here we studied both MMN and the sensory response (N250) in prematurely born 9-year-old children and in controls using scalp potential (SP) and scalp current density (SCD) topographic methods in order to dissociate the brain regions involved and to evidence the dynamics of their activation.

2. Material and methods

2.1. Participants

Fifteen children born preterm (PRE) and fifteen normally developing children born at term (control group: CTRL) participated in the experiment. Children were aged 9 years (years; months (SD), CTRL: 9; 3 (6), 9 males; PRE: 9; 6 (7), 7 males). Children born preterm had a mean gestational age of 31 (1.3) weeks and a mean birth weight of 1525 (408) g. Control children born at term (more than 37 weeks gestation) were matched for chronological age, sex and mother's educational level with the children born preterm. They had a normal developmental history with normal language and motor development and no medical antecedent.

The children in the preterm group were born between August 1993 and October 1994 at the University Hospital of Tours, France. They were recruited from the follow-up program of our neonatal department. Psychological, paediatric, ophthalmologic and audiometric assessments were performed by systematic examinations at ages 1, 2, 4, 5, and 9 years. They all had IQs in

the normal range as assessed by the K-ABC (Mental Processing Composite score: 104.7 (9.9)) (Kaufman and Kaufman, 1983).

Reading and writing skills were assessed in both the preterm and the control groups using the ECHAS-C (Simonart, 1998) and found to be in the normal range (PRE: 4.5 (1.4); CTRL: 4.6 (1.6)). Behavior was evaluated according to the revised Conner's Parent Rating Scale (Conners et al., 1998) to exclude children with major attention-deficit disorders, hyperactivity or impulsivity. All the participating children were performing satisfactorily in school (according to results at the systematic 3rd year school assessment). None had failed or required extra scholar support.

All subjects were right-handed and had normal hearing as assessed by brainstem auditory evoked responses (BAER) recorded at 2 years of age, and by a subjective audiometric task performed prior to studying the late auditory evoked potentials. Children were not included if they had a history of substantial neurological disorders or seizures, or had an abnormal electroencephalogram (EEG) with either slow waves or epileptiform discharges. The Ethics Committee (CCPPRB) of the University Hospital of Tours approved the protocol. Signed informed consent was obtained from parents, and assent was given by the children.

2.2. Stimuli and procedure

Auditory stimulus sequences consisted of 1000 Hz standard tones and 1100 Hz deviant tones (probability of occurrence: $p=0.15$) delivered in random order, with the constraint that each deviant tone was preceded by at least 3 standard tones. All tones had an intensity of 70 dB SPL and duration of 50 ms (5 ms rise/fall). A block of 1000 stimuli was presented binaurally through headphones with a constant (onset to onset) interstimulus interval of 700 ms. All subjects watched a silent movie on a TV screen during the recording session that lasted 15 min. Subjects were instructed to pay attention to the video story in order to tell the story to the experimenter at the end of the session.

2.3. EEG recording and data analysis

EEG was recorded from 28 Ag/AgCl electrodes referenced to the nose (the ground electrode was placed on Fpz location). Seventeen of the electrodes were placed according to the international 10–20 system (Fz, Cz, Pz, F3, F4, C3, C4, P3, P4, O1, O2, F7, F8, T3, T4, T5, T6). The remaining positions were midway between two positions of the 10–20 system i.e. FC1 (between Cz and F3), CP1 (between Cz and P3), FT3 (between T3 and F3), TP3 (between T3 and P3), and their homologous locations on the right hemiscalp; electrodes were also placed at FFz (between Fz and Fpz) and M1 and M2 (left and right mastoid sites). The impedance value of each electrode was less than 10 k Ω . Horizontal and vertical electro-oculograms (EOG) were recorded differentially from two electrodes located on the outer canthi of the right and left eye (horizontal bipolar) and two electrodes above and below the right eye (vertical bipolar).

The EEG and EOG were amplified with an analog bandpass filter (0.5–70 Hz; slope 6 dB/octave) and digitized at a sampling rate of 256 Hz. Epochs with either movements or eye blinks exceeding $\pm 100 \mu\text{V}$ were discarded. Automatic correction of eye

movements was then applied using a spatial filter transform developed by Neuroscan, specifying signal of no interest to be removed (ocular artefacts) and brain signal of interest to be retained (a reference sample of EEG). EEG epochs were averaged separately for the standard and deviant tones over a 700 ms-analysis period, including a 100 ms prestimulus baseline, and were digitally filtered (0–30 Hz). The ERPs to deviant tones included at least 120 responses for each subject. MMN was measured in the

difference waveforms obtained by subtracting the responses to the standard tones from responses to the deviant stimuli.

The N250 wave is the most prominent negative deflection occurring over fronto-central sites in response to repeating tones (standard) in children. Peak amplitude and latency of this response were measured in each subject in a 150–250 ms latency range. MMN peak amplitude and latency were measured in each subject by locating the most negative

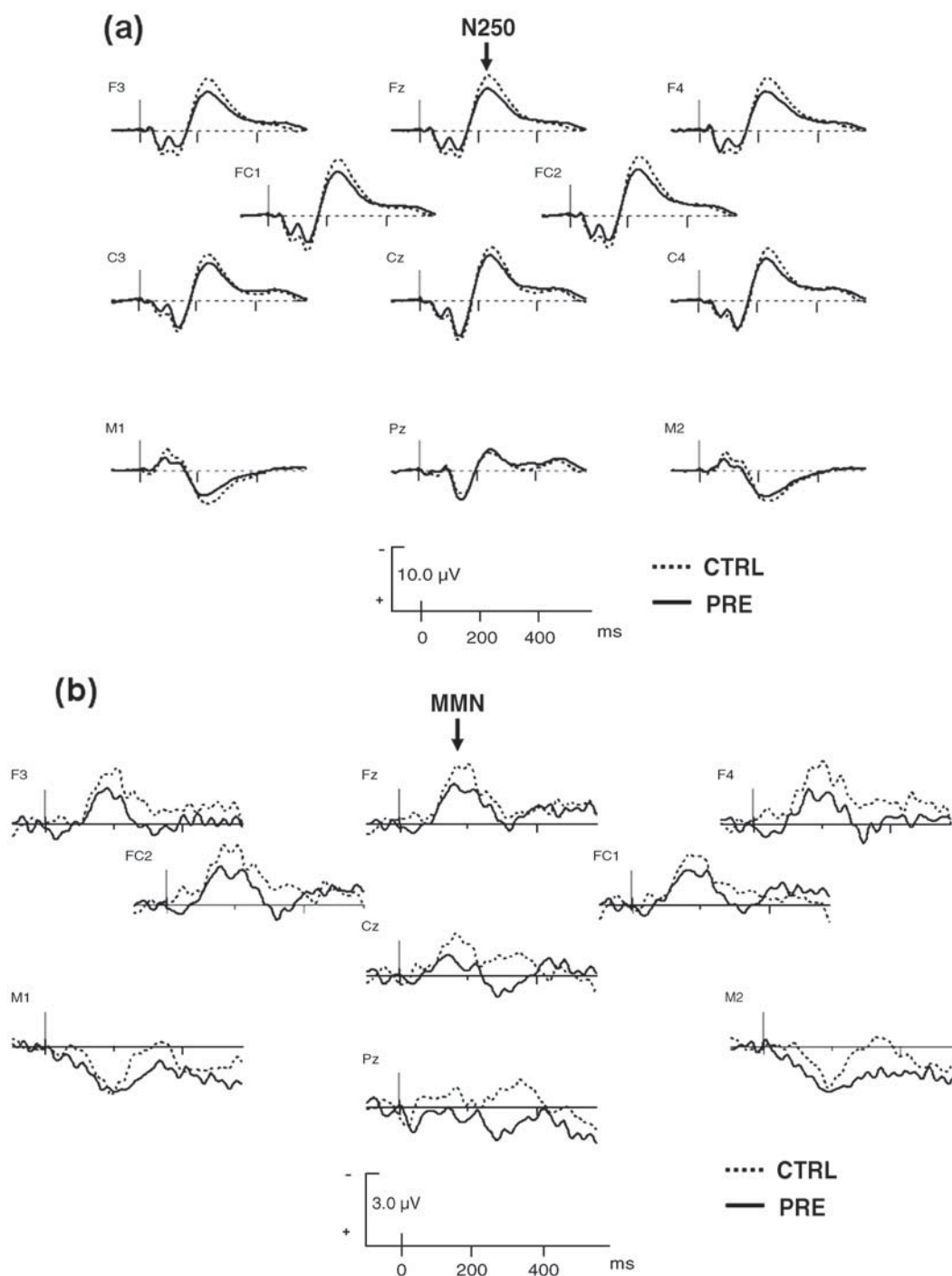


Fig. 1. Grand average response to standard tones in each group recorded over fronto-central electrodes and M1 and M2 (a) and grand average difference waveform in each group (b).

deflection within a ± 30 ms latency window around the peak of the grand average waveform of each group (i.e. 180 ms).

SCD amplitudes of MMN and N250 were estimated for each subject as the mean value over a ± 30 ms time window around the peak latency in the grand average SCD waveform for each group.

Scalp potential topographic maps were generated using a two-dimensional spherical spline interpolation (Perrin et al., 1989), and a radial projection from Cz (top views), which respects the length of the meridian arcs. SCD maps were estimated by computing the second derivative of the interpolated potential distribution ($m=4$, no regularization applied) (Perrin et al., 1987, 1989).

Amplitudes and latencies were analyzed using repeated measures analysis of variance (ANOVA) with Group (CTRL, PRE) as the between-subjects factor and Electrode as the within-subjects factor.

3. Results

3.1. Analysis of potentials

The grand average waveforms in response to standard and deviant stimuli for each group at selected electrodes are illustrated in Fig. 1a. The obligatory responses in children consist of fronto-central negativity peaking at around 240 ms and called the N250 wave. This response was clearly identified at fronto-central electrodes in all children and was significantly smaller in amplitude in children born preterm than in controls ($F(1,28)=6.37$; $p=0.017$; response to standard at Fz: mean (SD) μV , CTRL: -8.9 (2.0), PRE: -6.9 (2.3)). No latency difference was observed according to group (mean (SD) ms, CTRL, PRE: 239 (17) ms and 234 (22) ms, respectively). The same tendency of a smaller amplitude in children born preterm than in controls was observed on the positive deflection that preceded the N250 (i.e. the P150) but the difference did not reach significance

($F(1,28)=1.8$; ns; response to standard at Fz: mean (SD) μV , CTRL: 3.5 (2.1), PRE: 2.5 (1.8)).

Fig. 2a (top) presents the scalp potential distribution of N250 at the mean peak latency for each group. The maps display a large negativity over fronto-central areas in both groups, associated with bilateral positivity at temporo-mastoid sites. The positive potential was of smaller amplitude on the left mastoid site in PRE than in CTRL ($F(1,28)=6.92$; $p=0.013$; response to standard at M1: mean (SD) μV , CTRL: 5.5 (1.7), PRE: 4.2 (1.0)).

Fig. 1b shows the grand mean difference waveform (deviant-standard) for each group at selected electrodes. MMN was evident at fronto-central sites around 170–200 ms after stimulus onset in both groups, and its positive counterpart was recorded at mastoid electrodes (M1 and M2). There were no significant group differences in MMN latency or amplitude at either fronto-central or mastoid sites, although the response measured at Fz tended to be smaller in PRE than in CTRL (mean (SD) μV , CTRL: -3.5 (2.8); PRE: -2.9 (1.5), $F(1,28)=0.56$, $p=0.4$, ns). Curves also showed larger amplitude of the positive deflection following the MMN (i.e. possibly a P3a) in children born preterm than in controls, but the group difference was not confirmed by statistical comparison ($F(1,28)=1.12$; ns; mean amplitude at Cz in a 250–350 ms latency window: mean (SD) μV , CTRL: -0.62 (3.1), PRE: 0.38 (1.8)).

The scalp potential topography of the MMN (Fig. 2b, top) was identical in both groups and characterized by fronto-central negativity (although less spread in PRE) associated with bilateral positive temporo-mastoid fields, indicating involvement of generators located in the supratemporal cortex.

3.2. Scalp current density analysis

For both groups, the SCD maps of MMN and N250 at their respective peak latency presented a current sink–source pattern

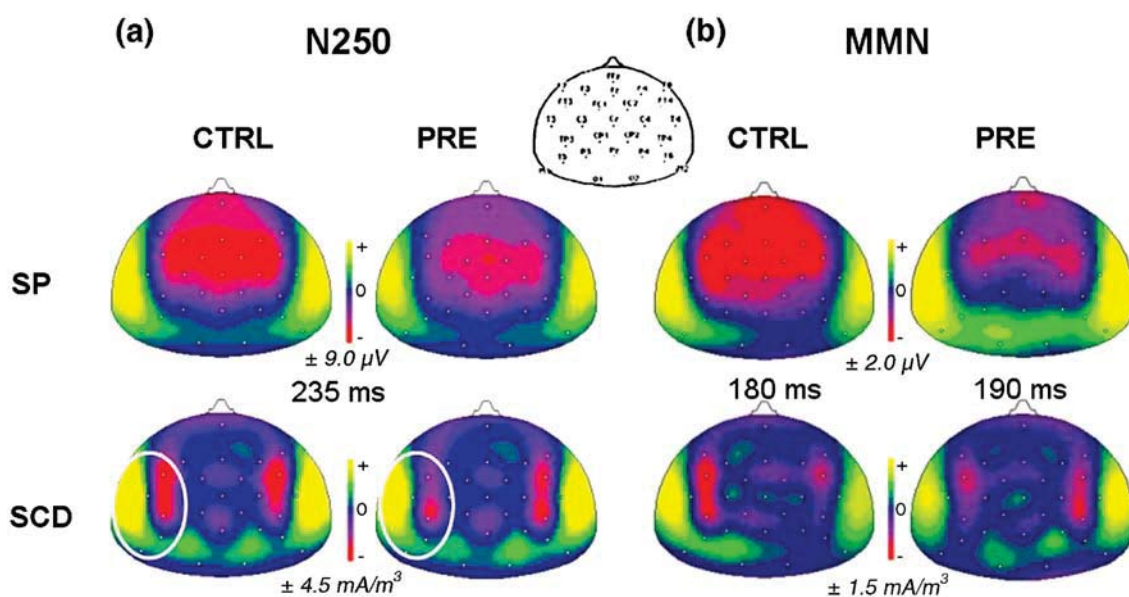


Fig. 2. N250 (a) and MMN (b) scalp potentials (top) and SCD (bottom) maps calculated at the mean latency peak of the responses for each group (positivity shown in yellow and negativity in red).

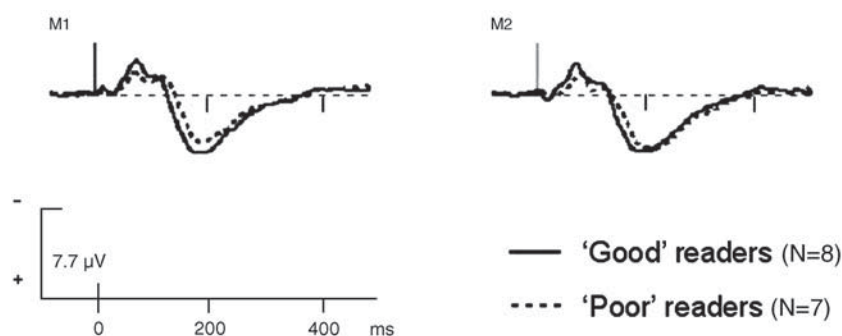


Fig. 3. Grand average response to standard tones recorded over mastoid electrodes in the 'poor' readers and 'good' readers subgroups (distinguished in the children born preterm group on the basis of reading level).

at inferotemporal areas, inverted in polarity over the approximate location of the Sylvian fissure (Fig. 2a, bottom). In addition, SCD maps showed frontally distributed current sinks of weak amplitude spreading over FC1 and FC2 sites in both groups.

SCD analysis of N250 indicated that both the frontal and the temporal sink current differed in amplitude between the two groups, with significantly lower values in the PRE group. The frontal sink was measured at FC1 and FC2 ($F(1,28)=7.87$; $p=0.03$; mean at FC1FC2 mA/m³ (SD), CTRL: -0.9 (0.4), PRE: -0.6 (0.2)), and the temporal sink at FT3 and FT4 ($F(1,28)=7.03$; $p=0.01$). This group effect was on the left temporal sink as indicated by planned comparisons which showed a significantly greater group difference on the left ($p=0.03$; FT3 mean (SD)mA/m³, CTRL: -4.0 (1.6), PRE: -2.0 (1.8)) than on the right hemisphere (ns, FT4 CTRL: -4.1 (1.8), PRE: -3.3 (1.9)).

There were no significant differences between the two groups of children for temporal sink/source or frontal negative currents of MMN (Fig. 2b, bottom).

3.3. Clinical subgroups

Two subgroups were then distinguished in the PRE group on the basis of reading level as assessed by the ECHAS-C. Reading scores were 2.9 (1.2) and 5.3 (1.3) ($F(1,13)=13.38$; $p=0.003$) for 'poor readers' (POOR) and 'good readers' (GOOD), respectively. The two subgroups were otherwise equivalent in terms of socioeconomic group, chronological age, gestational age, birth weight, sex ratio and IQ. Electro-behavioral relationships showed that N250 amplitude measured at M1 (positive field) tended to be smaller in the POOR than in the GOOD subgroup (Fig. 3), although the result did not reach significance ($F(1,13)=4.33$; $p=0.057$, mean (SD) μ V of the positive potential measured at M1 (GOOD: 4.6 (0.7); POOR: 3.7 (1.0)).

4. Discussion

The electrophysiological pattern in response to standard tones reported here showed significantly reduced auditory responses in children born preterm and provided evidence of abnormal functioning of the left temporal cortex and of the frontal region associated with prematurity.

This study showed that pre-attentional change-detection processes were normal in prematurely born children compared to controls, as assessed by MMN. This confirms previous findings of normal MMN in response to frequency deviance in younger children born preterm (Alho et al., 1990a,b; Dupin et al., 2000). However in response to speech deviance MMN has been shown to be reduced in prematurely born 6-year-old children, being correlated with their difficulties in naming (Jansson-Verkasalo et al., 2004). Although MMN in response to tones was not significantly affected in our study (possibly due to a large variability of the responses in children), it tended to be smaller in amplitude in the group of children born preterm. Between group differences in automatic change-detection processing might therefore be more marked for complex signal changes than for simple tone changes.

We showed reduced amplitude of the N250 wave in children born preterm when measured at fronto-central sites. Potential and SCD maps analysis showed that the left positive potential field and the left current sink, which both reflect the activity of generators in the left supratemporal gyrus, were smaller in amplitude. The electrophysiological pattern reported here in response to repeating tones might reflect lower activity and/or different orientation of the N250 left temporal generator in prematurely born children. This functional result corroborates findings from a recent structural study showing lower left temporal grey matter volume associated with left temporal gyrification in 8-year-old preterm children. Increased left temporal gyrification was also negatively correlated with reading recognition scores (Kesler et al., 2006).

Such reduction in N250 amplitude has previously been evidenced in infants with plagiocephaly, another high-risk group for developmental difficulties, and interpreted as reflecting an auditory processing dysfunction, as a possible result of the delayed or disturbed brain maturation (Balan et al., 2002).

Although the functional significance of the N250 wave in children remains to be clarified, some studies have indicated that it could reflect high cognitive processes. N250 is sensitive to the speech structure of the stimulation and thus might be dependent on auditory processing of complex signals (Vidal et al., 2005). The N250 wave in response to tones has previously been found to be smaller in amplitude in 7–13-year-old children with developmental dysphasia (Korpilahti and Lang, 1994) and in

reading impaired children (Neville et al., 1993). Thus the reduction in N250 wave amplitude we found in the left hemisphere may be an indicator of minor cognitive difficulties in prematurely born children, particularly of language processing impairment. This is partly supported by results from combining electro-clinical data that showed a tendency towards lower left mastoid positivity (associated with N250) in children who displayed poorer reading skills. Further investigation in this direction in a larger sample might be interesting.

SCD analysis allowed dissociation between temporal and frontal components of the N250 wave. The results showed that N250 frontal generator activity was also reduced in prematurely born children, possibly reflecting immature frontal activity. Although the children were selected according to their 'normal academic levels', they all were described as particularly 'dreamy', 'distractible' and 'inattentive' by their parents and teachers. The reduced frontal activity we found could thus be related to difficulties in auditory attention in prematurely born children. Understanding the underlying processes by which the ex-preterm child fails to achieve has proved difficult. As attention deficit is a candidate unifying mechanisms, the role of attention in the groups' difference found can not be ruled out. Further studies controlling for attention modulation of these responses in children born preterm are needed to strengthen this assumption.

Studies of magnetic resonance images in relation to outcome have not shown good correlation with brain injuries or regional measures of size (Cooke and Abernethy, 1999). However there is evidence that the preterm brain at term is less complex in terms of its cortical development and this is reflected in measures of regional brain volumes in later childhood (Peterson et al., 2003). The pervasive insult provided by preterm birth and subsequent extra uterine development clearly has major effects on brain organization and development, which await elucidation.

To conclude, even if children born preterm do not display hearing or cognitive impairment (possibly due to compensatory processes), the slight deficit evidenced in auditory information processing might have consequences on the development of higher-level processes, especially on language acquisition. These particular features of auditory processing should therefore be taken into account when considering the long-term outcome of premature children and educational support.

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