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Auditory cortical development charted by transient and 40-Hz steady-state responses in typical children and in Down syndrome

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Abstract. We are charting neocortical development in typical children and in children with Down syndrome (DS) using evoked transient and 40-Hz steady-state fields (SSFs) which localize to the region of A2 and A1 respectively, in the adult brain. Transient responses contrasted between cohorts aged 5–8 and 11–18 years showed declining P1m and increasing N1m amplitude in typical children (RMS N1m/P1m ratio increasing from 0.97 to 5.91 between the age groups), and decreasing P1m and N1m latency. Neither component changed appreciably with age in DS, although P1m latency decreased. SSF amplitude increased by a factor of 2.3 with age in control children and phase coherence by 2.0, pointing to a developmental effect putatively in A1. Neither SSF amplitude nor phase coherence showed a strong developmental trend in DS. Hearing thresholds did not differ between DS and typical children, implicating central rather than peripheral mechanisms underlying group differences.

Keywords: Auditory cortex development; Evoked transient and 40-Hz steady-state magnetic fields; Down syndrome; Primary and secondary auditory cortex

1. Introduction

In the typical brain neural connectivity in the deep layers (V, VI) of the human auditory cortex is well formed by age 5 but arborizes in the superficial neocortical layers (II, III) between

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age 5 and mid-adolescence in the primary (core region, A1) as well as secondary (belt and parabelt, A2) auditory cortex [1]. Animal research [2] and studies of children receiving music lessons [3] suggest that differentiation of neural connectivity is enabled and sculpted by acoustic inputs experienced early in life. This developmental process is expressed in auditory evoked fields (AEFs) which are generated by current sinks and sources in the superficial neocortical laminae [3,4]. We are charting auditory brain development in typical children and in children with Down syndrome (DS) with the eventual goal of establishing whether acoustic experience modifies development by rules for DS that are different from the typical case. We report initial results contrasting developmental baselines between two age cohorts of children with DS and age-matched controls. Development of A1 and A2 was distinguished by recording transient and 40-Hz "steady-state" AEFs that localize to these regions respectively in the adult brain [5].

2. Methods

We report data on the first 13 subjects (4 DS and 9 controls, aged 5 to 18 years, one male per group) in a planned cohort of 30 of each type. Brain responses (dc to 600 Hz, sampled at 2400 Hz) were recorded by a 151 sensor MEG (VSM) at the Down Syndrome Research Foundation in Vancouver, Canada. Transient P1m/N1m/P2m AEFs were evoked by piano and violin tones (C4 and A4) and by the consonant–vowel/ba/while subjects watched a silent video. Stimuli (400 ms duration, 2 s SOA) were given in a mixed order in three blocks, yielding 120 AEFs for each of the three stimulus types (piano, violin, consonant–vowel) overall. Head position was measured before and after each block. Following this series, 40-Hz amplitude modulated tones (carrier frequency 500 + 1000 + 4000 Hz) were presented to evoke the 40-Hz steady-state field (SSF). 20 trains each of 10-s duration were presented with a 500-ms pause between trains. All sounds were presented binaurally over ear inserts at 60 dB SPL. At the beginning and end of the session N1m was evoked by 100 1-kHz pure tones presented at 2 Hz to confirm sound delivery. Hearing thresholds and linguistic skills were measured for each subject (the latter not reported here).

Root mean square (RMS) averages (151 sensors) were used to identify P1m, N1m, and P2m transient responses after filtering (1–12 Hz) and artifact rejection (±2500 fT). The SSF was extracted by bandpass filtering (35–45 Hz). Sine–cosine (polar) plots were determined for each subject and aligned to a common phase on the basis of the first principal component using BESA 2000. Polar plots were then averaged across subjects to evaluate changes in SSF amplitude and phase coherence between the age groups.

3. Results

3.1. Transient responses

In previous EEG studies of typical children [3,4], P1 and N1 latency has been found to shorten with development while N1 amplitude enlarges. Both effects were evident in the RMS averaged transient AEFs for control subjects of this study (Fig. 1, evoked by the 1 kHz tones). The ratio N1m/P1m amplitude increased from 0.97 to 5.91 between the younger and older groups, respectively, while response latency decreased by 25.0 ms for P1m ($p < 0.01$) and 21.6 ms for N1m ($p < 0.05$). P1m latency decreased with age in DS as

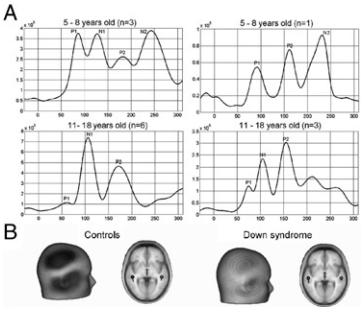

Fig. 1. (A) RMS averages showing P1m, N1m, and P2m peaks are contrasted between children 5–8 years old and 11–17 years old (Controls on left, DS on right, scaling varies). (B) N1m field pattern evoked by pure tones in the older cohorts. Cortical sources localized to the region of auditory cortex (regional sources, BESA 2000).

well, although not to the same extent (16.6 ms). However, we could not identify an N1m in our young subject with DS. In the older groups N1m amplitude was reduced by 67% in DS compared to controls. P2m amplitude increased and latency decreased with age in DS and controls, while N2m amplitude decreased in both groups. These effects are congruent with earlier normative data [3,4].

3.2. Steady-state field

Recent studies have found that the amplitude of the steady-state response (SSR) recorded electrically increases by a factor of ~2.4 between the ages of 5 and 16 years [6,7] in typical children, and phase coherence (expressed as reduced vector dispersion in polar plots) by a factor of ~2.1 over this age range [7]. Both of these effects were observed in the magnetically recorded SSF of control children. SSF amplitude increased by 2.3 with age (Fig. 2, scaling ×3 for older controls) and SSF coherence by 2.0, converging on a distinct dipolar pattern in the older group. These effects were less evident in children with DS. SSF amplitude did not increase with age in DS, and phase remained notably dispersed in older subjects. The difference in phase dispersion seen between older children with and without DS was not attributable to a larger sample size for the older typical cohort, since it was unchanged for subsets of equal size.

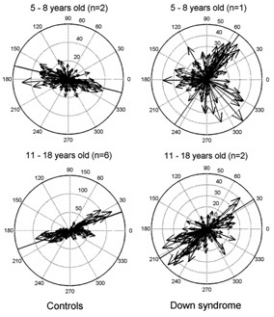

Fig. 2. Polar plots contrasting 40-Hz SSFs between age cohorts of typically developing children (left panels) and children with DS (right panels). The horizontal line is the first principal component (scaling increased ×3 for 11–18 year old controls).

3.3. Hearing function

Hearing thresholds measured at 500, 1000, and 4000 Hz in the MEG sound delivery system did not differ significantly between children with DS and controls (mean over frequencies and ears 27.1 dB and 19.3 dB respectively, $p=0.17$).

4. Discussion

Changes in response latency with age were observed in typical children and in DS. While increased myelination between age 5 and mid-adolescence may contribute [1], the magnitude of the change (>20 ms in controls) suggests that additional developmental processes are important. Competition among synaptic inputs favoring fast synapses can generate latency shifts of this order [8]. If this or another sculpting process is at work, our latency data suggest it is expressed in typical children and in children with DS although N1m amplitude does not grow with age at the same rate in DS.

The dipolar SSF field pattern that emerged in older typical children suggests that the steady-state stimulus was well described by a discrete cortical source putatively centered in A1. This sharpening of the cortical representation was not as evident for older children with DS where multiple cortical sources appeared to be activated at different phases. Asynchronous output from A1 to A2 might be a factor impeding the development of N1m in DS. Language development which is also delayed in DS could similarly be affected, to the extent that a precise representation of acoustic inputs is essential. Our results should be interpreted with caution pending further study, given the small number of subjects with DS available for this report (see Figs. 1 and 2).

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