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Does auditory discrimination training modify representations in both primary and secondary auditory cortex?

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Abstract. Several components of evoked auditory potentials and magnetic fields have been reported to be enhanced by musical training and by laboratory discrimination training. The P2 component, believed to originate in secondary auditory cortex (A2), is highly plastic, and enhanced both in trained musicians and by a variety of acoustic discrimination tasks. The 40-Hz auditory steady-state response (SSR) is believed to be generated in primary auditory cortex (A1). This response has been reported to be larger for trained musicians; however, in previous research SSR amplitude was not enhanced by laboratory training at pitch discrimination in non-musician adults. To explore whether competitive interactions among frequency representations in A1 may constrain remodeling, we designed a training procedure that provided subjects with experience of a single carrier frequency for 10 training sessions, alternating training and passive control blocks. P2 amplitude increased across sessions ($p<0.01$) on passive as well as training blocks. SSR amplitude was larger in training compared to passive blocks ($p<0.01$) and increased across sessions in both blocks ($p<0.01$), giving the first evidence of acoustic remodeling in A1.

Keywords: Evoked potentials; Auditory cortical plasticity; 40 Hz auditory steady-state response

1. Introduction

Because the majority of neurons in the primary (core, A1) and secondary (belt, parabelt, A2) auditory cortex are neuroplastic (79% A1, 95% A2 [1]), training effects should be expressed in AEPs and AEFs. A growing literature confirms this expression in transient responses (particularly the P2) which localize to A2 outside of the auditory core [2–5]. On the other hand, amplitude

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enhancement of the 40-Hz auditory steady-state response (SSR) localizing to the region of A1 [6,7] has not been successfully demonstrated by training procedures. Bosnyak et al. [2] found only a brief modulation of SSR phase at ~200 ms when the subjects were trained to discriminate changes in the carrier frequency of a steady-state stimulus (a spectral discrimination task).

We hypothesize that competitive interactions within the SSR frequency map may have constrained expansion of the cortical representation of the trained frequencies in the Bosnyak et al. study [2]. Here we describe the first of a series of experiments asking whether we can demonstrate amplitude enhancement of the 40-Hz SSR by acoustic training. The stimulus procedure adopted for the experiments requires that subjects distribute their attention for the duration of the steady-state stimulus (attention gating changes in A1 via the basal forebrain [8,9]) and exposes them to only a single carrier frequency thus reducing competitive interactions in the frequency map. The experiments are guided by the proposed principle that under the conditions of attention the auditory cortex encodes its acoustic experience.

2. Methods

Nine experimental subjects and five controls aged 20–28 years (mean 23 years, 7 male, students at McMaster University) provided written consent. They were paid for participation and some received partial course credit. All were tested for normal hearing.

The stimulus was a 2 kHz tone 1 s in duration, amplitude modulated (AM) at 40.96 Hz. In two thirds of the stimuli, a single pulse in the 40-Hz train was amplitude-enhanced in one of three positions (randomly) in the second half of the stimulus (target). All stimuli were presented binaurally at 60dB SPL via Etymotic ER2 ear inserts.

Each experimental session consisted of 10 training and 10 passive blocks alternating, each about 2.5 min in duration. During the training blocks (each containing 27 stimuli, 18 with targets) subjects were instructed to indicate with a button press after each trial whether

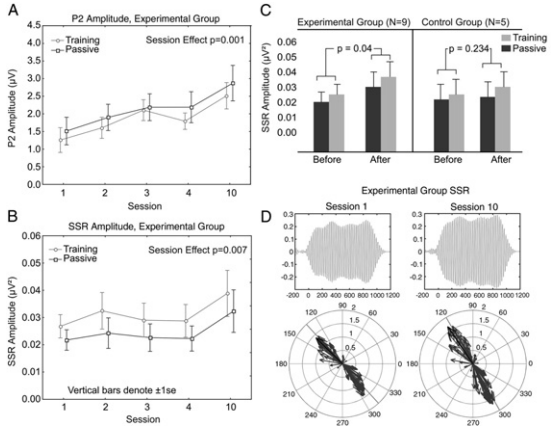

Fig. 1. A. P2 amplitude over sessions in the Experimental group for training and passive bocks. B. SSR amplitude over sessions in the Experimental group for training and passive blocks. C. Comparison of SSR amplitude in the Experimental and Control groups before and after training. D. Time domain SSR (upper) and polar plots (lower, dark FCz) before and after training in the Experimental Group, for the training blocks.

they detected a target (continuous performance task). Feedback was given following the response. The SOA was ~3 s. The passive block consisted of an identical set of trials but subjects were instructed to stop responding and ignore the stimulus.

Experimental subjects (Group E, $n=9$) received 4 training sessions at weekly intervals (spaced practice) followed by 6 more at daily intervals (massed practice) which accommodated their academic schedule (10 sessions overall). EEG was recorded in sessions 1–4 and 10. Control subjects (Group C, $n=5$) received two training sessions aligned in time to sessions 1 and 10 of Group E.

128 channel EEG (Biosemi ActiveTwo) was sampled at 512 Hz (low pass-3dB at 100 Hz, reference at Cp1 and ground at Cp2 10-10). EEG responses were epoched into 1 s segments with 200 ms pre/post baselines. After selecting approximately 90% artifact-free segments, data were averaged and re-referenced to an average reference. Transient responses were extracted after filtering 1–20 Hz. For SSR responses the averages were filtered 40–42 Hz. All waveforms and results reported in this preliminary analysis were calculated on electrode FCz where the SSR typically reaches its amplitude maximum.

3. Results

3.1. Behavioural results

All subjects showed behavioural improvement across sessions ($p<0.004$). Mean Threshold (50% hit rate) decreased gradually from 32% enhancement on Day 1 to 20% enhancement on Day 10.

3.2. Transient responses

P2 amplitude increased across sessions (Fig. 1A), reaching a final amplitude ~50% greater than at outset ($p<0.001$). Although the effect of attention (training versus passive blocks) was not significant for P2, its enhancement by training was more pronounced under passive conditions, probably because N2 was enhanced when subjects attended to the stimuli during training. The Control group showed no significant effect of session on P2 amplitude (results not shown).

3.3. Steady-state response

Experimental and control subjects differed in the amount of training delivered over 6 weeks (10 versus 2 sessions, respectively), but their initial and concluding evaluations were performed at the same time. For the Experimental group, the effect of session was significant ($p=0.007$), with the SSR generally increasing across sessions (Fig. 1B). Before/after differences were found for experimental subjects (Fig. 1C, $p<0.04$) but not controls ($p=0.23$), indicating that the amount of training and not the passage of time determined the outcome. SSR amplitude was enhanced in the training as well as passive blocks, which differed from one another in the Experimental ($p=0.0001$) and Control ($p=0.033$) groups pointing to SSR enhancement by attention. Enhancement of SSR amplitude in the Experimental group was evident throughout the trained stimulus (upper panel, training blocks) and in polar plots depicting all channels (lower panel, Fig. 1D) where dipolar patterns suggested a single cortical source contributing to the SSR, putatively in A1.

4. Discussion

Behavioral improvements in discrimination ability were accompanied by P2 enhancement, corroborating previous findings [2–4] and giving a picture of cortical reorganization expressed in the region of A2. SSR amplitude increased after ten sessions in the experimental group but not controls, and was modulated by attention in both groups. The enhancement of SSR amplitude by training seen here contrasts with effects on phase only reported by Bosnyak et al. [2] where discrimination of carrier frequency was trained. The different outcomes may be a consequence of our having trained amplitude detection using only a single carrier frequency. This procedure was developed to reduce competitive interactions in the region of A1 where surround inhibition is strong and the cortical sources of the SSR typically localize [6,7].

There are qualifications, however. SSR amplitude did not track a steadily increasing course as P2 did. After an initial increase in the second training session, SSR amplitude did not grow further until massed training between sessions 5–10. At present we do not know whether session spacing or number was the relevant variable. Here we used a minimal control condition. Additional control groups will be required to assess whether SSR enhancement fails when competing frequencies are added to the training procedure. It may be noteworthy that P2 was modulated by training and not by attention, whereas the SSR was modulated by both. This suggests that the rules that describe plasticity and its expression in the SSR may be different from those describing transient responses. The differences may reflect properties intrinsic to different regions of the human auditory system.

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