Modulation of the 40-Hz Auditory Steady State Response by Attention during Acoustic Training

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Abstract. We investigated whether attention modulates the 40-Hz auditory steady-state response (SSR) which localizes to cortical sources in the region of primary auditory cortex (A1). Subjects in Group E performed a tone discrimination task requiring sustained attention. This task was preceded and followed by a passive stage in which identical stimuli were presented while subjects viewed a silent video. Control groups received three successive passive stages only (with or without the video). SSR amplitude increased from the passive baseline in Group E and with respect to both control groups when the discrimination task was performed, and reverted to baseline afterwards. The SSR source waveform was enhanced in right hemisphere, implying modulation of A1 by attention in this hemisphere. SSR phase and 3d source parameters did not shift when the discrimination task was introduced, implying similar SSR A1 generators in the passive and task conditions. Enhancement of N1, P2, and N2 during the discrimination task pointed to concurrent modulation of secondary auditory cortex by attention.

Keywords: Attention, 40-Hz auditory steady-state response, Primary and secondary auditory cortex

1. Introduction

Neural activity in the human auditory cortex is modulated by cholinergic projections from the basal forebrain (BF) that appear to perform the functions of an attention system [1]. Stimulus-driven (bottom-up) and task-driven (top-down) inputs appear to converge through this structure which gates neural plasticity by making cortical neurons more sensitive to their afferent inputs [2]. Transient N1 and P2 auditory evoked potentials (AEPs) and magnetic fields (AEFs) which localize to secondary auditory cortex (A2) are sensitive to attention and are modified by experience [see ref #3 for an overview], consistent with BF projections to A2 and a linkage between attention and plasticity.

Less clear is the role of human primary auditory cortex (A1) in this system. Here we used the 40-Hz auditory steady-state response (SSR) which localizes to the region of the

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auditory core [3] to investigate attentional modulation of A1. Although competitive interactions may constrain remodeling of the SSR representation in this region, modulation could be expected given the established density of cholinergic projections from the BF to the auditory core [4]. To increase the likelihood of an effect, we used a procedure that required sustained attention throughout a 1-second 40-Hz amplitude modulated (AM) stimulus. SSR changes were assessed during and after task performance to distinguish attention from short-term neuroplastic effects.

2. Materials and methods

Sixty-three students at McMaster University aged 17-44 years (mean 20.6 years, 6 left-handed, 18 male) with measured normal hearing at 2 kHz provided written consent. Auditory stimuli were 2 kHz pure tones (duration 952 ms, 60 dB SPL) AM at 40.9626 Hz (modulation depth 100%, Tucker-Davis RP2) and delivered binaurally via ER-2 ear inserts. Stimuli were presented in S1/S2 pairs (S1 offset to S2 onset 500 ms). In each pair either S1 or S2 (p=0.5) contained a single 40-Hz target pulse of variable amplitude that occurred randomly in time commencing 400 ms after stimulus onset. During training subjects in Group E (n=31) indicated by a button press which tone contained the target (2IFC procedure, feedback for correctness provided). In Group E this task was introduced after (and followed by) a passive stage during which identical auditory stimuli were presented while subjects watched a silent video. Subjects in Groups C1 (n=17) and C2 (n=15) received three passive stages only. For C2 the video was switched off in stage 2. All stages (three in each group) were 15 minutes in duration.

EEG (128-channels, Biosemi Active2, sampled at 2048 Hz, low pass -3 dB at 417 Hz) was recorded using CP1 as reference and CP2 as ground (10-10 system). EEG responses to S1 stimuli were re-referenced off-line to the average reference including 200 ms pre/post baselines. These epochs were filtered 0.2-20 Hz to extract transient responses and 40-42 Hz for the SSR. Symmetrical regional sources determined for each subject in Group E (BESA v5) were converted to single dipoles (orientation allowed to vary).

3. Results

Psychophysical functions generated for subjects in Group E during training spanned the threshold of target amplitude detection, demonstrating that subjects attended to the stimuli and performed the task during the training stage.

Fig. 1 shows the time domain average transient waveforms at electrode Fz for each group across the three stages of the experiment. N1 amplitude decreased across stages 1-3 in the control groups, and between stage 1 and stage 3 in Group E (both effects $p < 0.05$). In Group E transient responses (N1, P2, N2) and the auditory sustained response (SR) were enhanced during the training stage compared to the passive stages (all effects $p < 0.01$). Control groups C1 and C2 did not differ for any AEP, confirming that enhancement during training in Group E was due to the task and not to the absence of the video.

SSR amplitude measured at its amplitude maximum (Fz electrode) increased when subjects attended to the task stimuli during training (Fig 2A, stage TR, group x stage interaction $p = 0.025$). Increases from the passive baseline of stage 1 were observed for

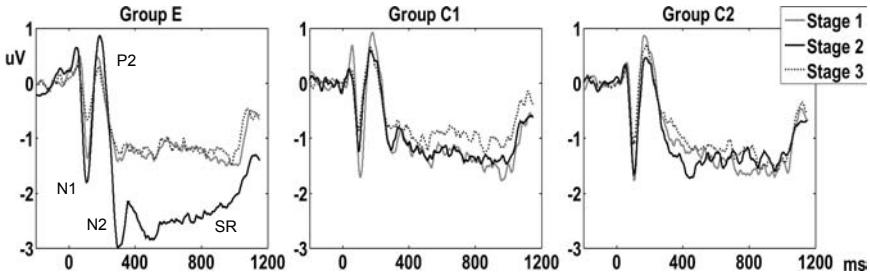

Fig. 1. Transient responses in each stage of the experiment (Stage 1 light solid, Stage 2 dark solid, and Stage 3 dark dotted). Group E (left) shows enhanced amplitude for P1, N1, P2, N2, and SR during training (Stage 2). Control groups C1 and C2 (right) show a decrease in N1 amplitude across the stages of the experiment.

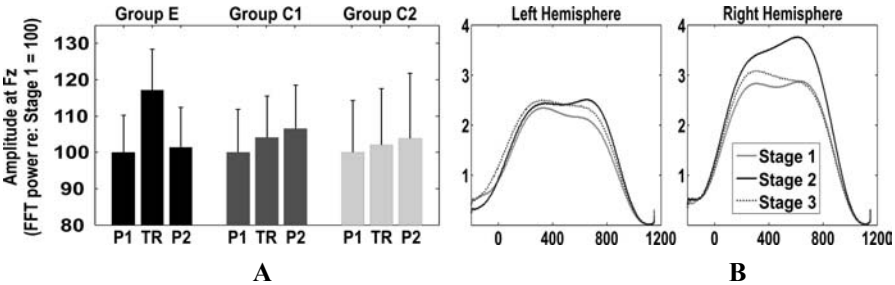

Fig. 2. Steady-state response. **A.** SSR 40-Hz amplitude for each group and stage of the experiment (P1=Stage 1, TR=Stage 2, P2=Stage 3). Amplitude is measured as 40 Hz spectral power at electrode Fz, normalised to the first stage in each group (bars are 1 standard error). **B.** Source waveform for each hemisphere across the three stages for Group E. Stage 1=light solid, Stage 2=dark solid, and Stage 3=light dashed.

27 of 31subjects in Group E ($p < 0.0001$) compared to 18 of 32 subjects ($p = 0.597$) in the two control groups which did not differ from one another in any stage of the experiment. SSR source waveforms analyzed by Hilbert transform showed enhancement by attention in the right hemisphere only, throughout the stimulus (Fig. 2B, $p = 0.013$). Attention had no detectable effect on SSR phase, 3d dipole location, or dipole orientation (passive/task comparisons not significant).

3. Discussion

In stage 2 subjects in Group E were required to detect a single 40-Hz AM pulse of enhanced amplitude that occurred at a random time point commencing 400 ms after the onset of a 1-s, 2 kHz 40-Hz AM sound. Under this condition SSR amplitude increased compared to a passive baseline where the same stimuli were presented while subjects watched a silent video. Two control groups receiving the same stimuli under passive conditions found no effect of the presence or absence of the video in stage 2, confirming that the auditory task requirement was responsible for SSR enhancement in Group E.

Previous studies of attentional modulation of the SSR have provided mixed results. An early report did not find enhancement of SSR amplitude [5]; however the stimulus was presented at 85 dB SPL which is near the limit of the 40-Hz SSR amplitude-intensity function [6]. More recently, Ross et al. [7] found SSR amplitude enhancement when

subjects were required to detect a brief perturbation of the AM envelope presented 100 ms after stimulus onset. Enhancement was restricted to the time interval ~200-500 ms which is likely when the discrimination was made. In contrast, we found enhancement extending over the wider interval of 200-850 ms, which included the time interval when target sounds could have occurred. These results suggest that SSR modulation is sensitive to the timing requirements of the task. Modulation may also be sensitive to the stimulus feature to be detected. Our enhancement where stimulus intensity was detected occurred in the right hemisphere, compared to enhancement in the left hemisphere reported by Ross et al. [6] where a temporal feature of the stimulus was discriminated.

Because the cortical sources of the SSR localise to the region of A1 [see ref #3 for a review], our findings imply that neurons in this area are attention sensitive, in accordance with cholinergic projections to this region [4]. Attention did not affect SSR phase or the 3d location or orientation of current dipoles fitted to SSR field patterns, which would have been predicted had additional sources from A2 contributed [8]. Transient responses which localize to A2 were strongly modulated, however, consistent with earlier findings. Because all responses reverted to baseline values in the second passive session, enhancement by the task in Group E appeared to be a consequence of attention and not short term plastic changes induced by stimulus processing.

Acknowledgements

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