Modulation of P2 auditory-evoked responses by the spectral complexity of musical sounds

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We investigated whether N1 and P2 auditory-evoked responses are modulated by the spectral complexity of musical sounds in pianists and non-musicians. Study participants were presented with three variants of a C4 piano tone equated for temporal envelope but differing in the number of harmonics contained in the stimulus. A fourth tone was a pure tone matched to the fundamental frequency of the piano tones. A simultaneous electroencephalographic/magnetoencephalographic recording was made. P2 amplitude was larger in musicians and increased with spectral complexity preferentially in this group, but N1 did not. The results suggest that P2 reflects the specific features of acoustic stimuli experienced during musical practice and point to functional differences in P2 and N1 that relate to their underlying mechanisms. *NeuroReport* 16:1781–1785 © 2005 Lippincott Williams & Wilkins.

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Introduction

The auditory P2 is a dipolar vertex-positive auditory-evoked potential generated by cortical sources lateral to Heschl's gyrus in the region of the secondary auditory cortex (A2) [1–4]. A distinctive property of P2 is that its amplitude is enhanced in non-musicians by training at acoustic discrimination with complex tones [5–7] and with pure tones amplitude-modulated at 40 Hz [4] in agreement with animal data showing a high degree of plasticity in A2 neurons [8]. P2 is also enhanced in musicians when evoked by tones of musical timbre [9,10], as predicted by the sensitivity of P2 to neuroplastic remodeling and the prior experience of musicians with musical sounds during practice and performance [9].

The present study investigated the mechanisms underlying enhancement of the P2 in musicians. Animal studies indicate that cortical modeling of auditory representations is gated by forebrain mechanisms that release neuromodulators known to make neurons more sensitive to their afferent inputs [11–14]. In the presence of neuromodulation, the cortex appears to encode its experience via Hebbian mechanisms operating within a competitive network [15]. If P2 enhancement in musicians depends on this process, we would expect A2 neurons underlying the P2 to encode the specific spectral and temporal features of acoustic stimuli experienced during musical practice. Furthermore, P2 enhancement should depend on the processed features being present in the input.

In order to test these predictions, we recorded P2 responses while musicians with prior training on the piano and non-musician controls listened to piano tones that were manipulated to differ with respect to the number of upper harmonics that were preserved in the stimulus. The temporal envelope of the piano tones was held constant. This procedure allowed us to determine whether P2 enhancement is modulated preferentially in musicians by upper harmonics of natural piano tones they would have experienced during musical practice. A further goal was to contrast the effect of spectral complexity on P2 and N1 auditory-evoked potentials. These auditory-evoked potentials differ in their latencies and source localizations [1–4] and express different functional properties. When measured by electroencephalography, P2 is enhanced by acoustic training in non-musicians [4–7] and is larger in musicians than in non-musicians [9,10] whereas N1 is not. In the present study, simultaneous electroencephalographic (EEG) and magnetoencephalographic (MEG) recordings were undertaken.

Materials and methods

Study participants

Eight musicians (aged 27.9±7.25 years) and eight non-musician controls (aged 28.9±4.34 years) participated in the study, which was conducted in the MEG laboratory at the Rotman Research Institute of Baycrest Centre (affiliated with

the University of Toronto). Four of the musicians and one control were women; all study participants were right handed. Musicians were recruited by posters placed in the Faculty of Music at the University of Toronto. All musicians reported that they played the piano. Six musicians identified the piano as their principal instrument; one musician reported the flute and another musician percussion as their principal instrument and piano as a secondary instrument. Musicians reported practicing their principal instrument an average of 9.25 ± 5.1 h a week; six musicians reported commencing practice at or before the age of 6 years (mean years of practice 14.9 ± 6.8). Non-musician participants reported that they had no formal musical training and had never played a musical instrument. Normal auditory thresholds [less than 20 dB hearing level (HL)] were confirmed for each participant by audiometry to 8 kHz. Participants gave written, informed consent in accordance with procedures approved by the Research Ethics Committee of Baycrest Centre for Geriatric Care.

Stimuli

The stimuli consisted of four tones each of 500 s duration: (1) a C4 piano tone consisting of the fundamental (262 Hz) and eight harmonics (called piano); (2) a tone with a temporal envelope matched to the piano tone consisting of the C4 fundamental and the first two harmonics (called piano2); (3) a tone matched in envelope to the piano tone but consisting only of the C4 fundamental (called piano0); and (4) a pure tone at the C4 fundamental (called pure). In order to prepare the piano stimuli, a Fourier transform was applied to a natural piano sound followed by bandpass filtering in the Fourier domain. For each harmonic f_i (i=0,…,8) of the fundamental f_0, a Hanning window was applied ranging between $0.5f_i$ and $1.5f_i$, with a maximum of 1.0 at f_i. The partials of the sound data extracted by the window contained the harmonic component and side lobes reflecting the corresponding temporal structure. For the piano0 tone, the spectral segment under window i=0 was used; for the piano2 tone, the windows 0, 1, and 2 were used; for the piano tone, the windows were 0–8. Inverse Fourier transforms reconstructed the time series. Figure 1 shows the waveforms and spectra for all tones.

Procedure

MEG was recorded using a 151-channel whole-head MEG system (VSM MedTech, Coquitlam, British Columbia, Canada). The EEG was recorded using the EEG amplifier integrated with the MEG system (32 Ag/AgCl electrodes placed in an Easy Cap, 10–20 array). The reference electrode was Cz and a single ground electrode was placed at the collarbone. Skin impedances were reduced to less than 10 kΩ at all electrode sites. MEG and EEG were simultaneously sampled at 312.5 s^{-1} after 100-Hz lowpass filtering. The experimental sessions were conducted in an acoustically silent and electrically and magnetically shielded room.

Tones were presented through plastic tubes at 60 dB above thresholds measured individually for each participant, tone, and ear prior to the session [60 dB sensation level (SL)]. Participants watched a silent movie during the session and were videotaped and monitored for head movements. Eight blocks each containing 60 stimuli of the same type were arranged and presented in the following order: pure tone (first and last), piano2 (second and seventh), piano (third and sixth) and piano0 (fourth and fifth). Tones were presented using a variable interstimulus interval ranging from 3 to 4 s offset to onset.

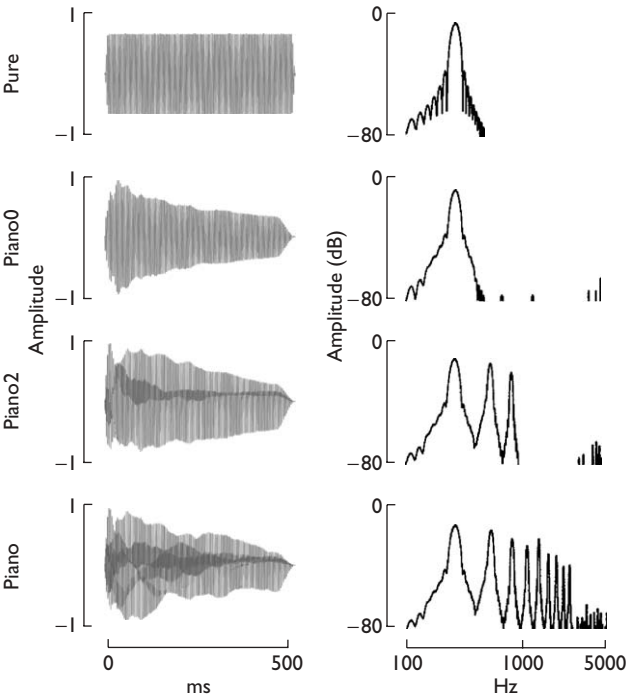

Fig. 1 Time domain waveforms (left) and spectra (right) for the C4 stimuli. From bottom to top: piano tone (fundamental plus the first eight harmonics), piano2 tone (fundamental plus the first two harmonics contained in a piano envelope), piano0 tone (fundamental contained in a piano envelope), and a pure tone with only the fundamental.

Data analysis

Continuous EEG and MEG files for each participant were loaded into BESA 2000 (MEGIS Software, Gräfelfing, Germany), digitally filtered between 0.1 and 20 Hz (zero phase shift), and divided into epochs of 600 ms duration according to tone type including a 100-ms prestimulus interval. Trials contaminated by shifts in any channel greater than $\pm 200\,\mu$V in EEG and/or ± 2000 fT in MEG were rejected. Accepted trials (mean 86%, range 75–98%) were averaged according to stimulus type (pure, piano0, piano2, and piano).

Two methods of signal processing were used. In one, the root mean square (RMS) across all channels was calculated from averaged electrical and magnetic data for each participant and tone type. N1/N1m amplitude and latency were determined for each participant and stimulus at the RMS maximum during the 90–140 ms interval after stimulus onset. P2/P2m amplitude and latency were determined at the RMS maximum during the 160–260 ms interval after stimulus onset. Results were also analyzed in source space [16]. Using BESA 2000, two symmetrical regional sources (one in each hemisphere) were fitted separately to the N1, N1m, P2, and P2m peaks of the average waveforms of each participant (collapsed across tones), utilizing a time window of ± 5 ms around the peak global field power. Regional sources were then converted to single equivalent current dipoles and applied as spatial filters to give a source

waveform for each participant, stimulus, hemisphere, and response component. Response amplitudes were determined using the same latency windows as in the RMS data.

Statistical analyses

Sound thresholds measured in the MEG sound delivery system were assessed by repeated-measures ANOVAs including the variables group (musician/control), tone (pure, piano0, piano2, and piano), and ear, using the general linear model module of Statistica version 6.0 (Statsoft Inc., Tulsa, Oklahoma, USA). N1, N1m, P2, and P2m amplitudes and latencies were evaluated by ANOVAs conducted separately for each response component using the variables group and tone in the RMS analysis, and group, tone, and hemisphere in the source space analysis. Post-hoc comparisons were made with the least significant difference test. All tests were two-tailed (α=0.05) and corrected for sphericity violations (Greenhouse–Geisser) where appropriate. Effects of sex were tested by regrouping the data according to this variable but were not significant for any measure and are not considered further. As the RMS data and source waveforms yielded nearly identical statistical results, we present the RMS data herein, citing source space results only when differences occurred or when effects of hemisphere were considered.

Results

Sound thresholds measured in the MEG sound delivery system were lower for the spectrally complex piano and piano2 tones (30.0 and 32.3 dB, respectively) than for the pure and piano0 tones (39.8 and 40.6 dB, respectively, $P<0.015$) and for the left ear (30.9 dB) than for the right ear (40.0 dB, $P<0.0001$). As the stimuli were equated for sensation level, sound pressure levels (threshold + 60 dB) were lower for the more complex piano and piano2 tones and for the left ear (effects of ear reflecting characteristics of the sound delivery system). No group differences or interactions of group with tone were found in the threshold data (F's<1.0).

RMS waveforms are shown in Fig. 2a (upper panel) for musicians and non-musicians separately, averaged over the four tones. N1 and P2 peaks (EEG, left panel) and N1m and P2m peaks (MEG, right panel) are identified in each waveform. Scalp topographies evoked by the piano tone are shown (averaged over groups) in the lower panel of Fig. 2a. P2 and P2m amplitudes were larger in musicians than in non-musicians [P2: F(1,14)=14.16, P=0.002; P2m: F(1,14)=5.85, P=0.03]. The N1 auditory-evoked potential tended to be larger in musicians, but group differences in N1 and N1m amplitude were not significant.

Figure 2b depicts N1, N1m, P2, and P2m amplitude for each tone separately in the two groups. N1 and N1m did not differ among the stimuli in the musician or control groups (all F's<1.0). For P2, however, a main effect of tone [F(3,42)=35.80, $P<0.00001$], as well as an interaction of tone and group [F(3,42)=6.74, $P<0.0009$], was found, indicating that the effect of spectral complexity on P2 was larger in the musician group than in non-musician controls (Fig. 2b, lower left panel). P2 amplitude was larger for the piano tone than for all remaining tones in the musician group, whereas in non-musicians P2 evoked by the piano tone was larger than P2 evoked by the pure and piano0 tones but not the piano2 tone ($P<0.02$). The interaction of group with tone remained significant (P=0.0073) when P2 amplitude for the three piano tones was referenced to P2 amplitude for the pure tone, removing the group effect. A main effect of tone was also found for P2m amplitude [F(3,42)=6.26, $P<0.002$], which reflected larger P2m for the piano and piano2 tones than for the pure tone, and for the piano tone than for the piano0 tone ($P<0.02$; Fig. 2 bottom right panel). The interaction of group and tone did not reach significance for P2m in the RMS analysis, although it was significant for the P2m source waveform [F(3,42)=4.11, P=0.016] where P2m was enhanced in musicians when evoked by the piano0 and piano2 tones ($P<0.005$).

No effects of group or tone were found for P2 or P2m latency (P2 overall mean 195.8 ms). A main effect of tone was found for N1 [F(3,42)=8.33, $P<0.0002$] and N1m latency [F(3,42)=14.6, P=0.0002], however. N1 latency (overall mean 109.3 ms) was on average shorter by 3.8 ms

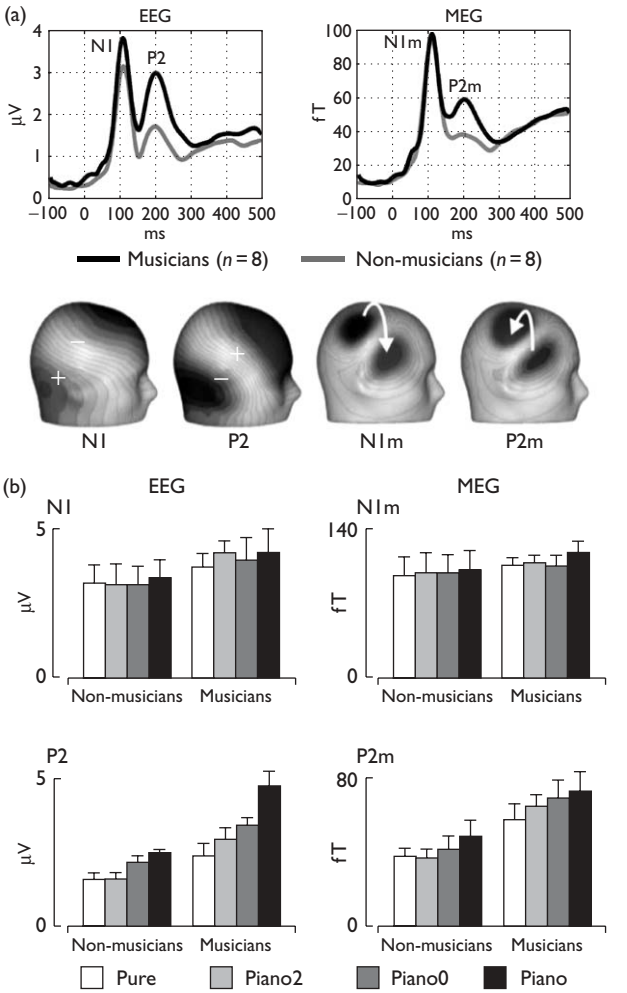

Fig. 2 (a) Root mean square (RMS) waveforms for musicians (black line) and non-musicians (gray line) showing NI and P2 peaks (left, EEG) and NIm and P2m peaks (right, MEG) averaged over all four tones. Tone onset was at 0 ms. Voltage and magnetic flux topographic maps for NI, P2, NIm, and P2m responses evoked by the piano tone are shown below the RMS plots. (b) The amplitude of each brain response as determined from RMS waveforms is shown for the tones separately in the musician and non-musicians groups. The bars denote one standard error. EEG, electroencephalogram; MEG, magnetoencephalogram.

for the piano and piano2 tones than for the piano0 and pure tones ($P<0.03$), with no significant differences occurring between the piano0 and pure tones. No group differences in N1 or N1m latency or interactions of group with tone were observed. The dipole moment for N1 was larger in the right than in the left hemisphere ($P=0.039$) and that for P2m in the left than in the right hemisphere ($P=0.017$), but the variable hemisphere did not interact with group or tone for any response component in the source analysis.

Discussion

Our findings agree with recent studies showing that P2 amplitude evoked by piano tones is enhanced in musicians compared with that in non-musicians [9,10]. In addition, we found that P2 amplitude was enhanced by the presence of natural harmonics of piano tones in both groups. Significant group by tone interactions in the EEG data indicated that this effect was larger in musicians who would have experienced the upper harmonics of natural piano sounds while practicing their piano skills. Group by tone interactions were less strongly expressed in the MEG data, pointing to radial or deep sources contributing to this effect in the EEG findings. Sound intensity does not appear to have been a factor in the results, because the stimuli were presented at equivalent sensation levels and there were no group differences or interactions of group with tone in sound thresholds that were measured for each participant, stimulus, and ear. As the temporal envelope was equated for the piano tones, P2 differences among the tones appear to have been a consequence of our manipulation of spectral content.

In contrast to P2, N1 amplitude measured electrically or magnetically was not influenced by the spectral complexity of the stimuli. This suggests that N1, which is known to be sensitive to onset parameters [17], was influenced more by synchronous activity induced by the temporal envelope of the stimuli than by spectral content. Simulations of auditory nerve activity carried out by Seither-Preisler *et al.* [18] suggest that evidence for an effect of spectral bandwidth on the N1m reported by these investigators and by other studies [19,20] could, in principle, be accounted for by this factor. The shorter N1 latencies that we observed for piano and piano2 sounds than we did for piano0 and pure tones may be a consequence of enhanced P2 responses for these tones, which may have partially overlapped the polarity-opposed N1 and shifted its peak to an earlier point in time. We cannot rule out the possibility that the same process may have restricted an effect of spectral complexity on N1.

Differential effects of spectral complexity on N1 and P2 may also relate to differences in how these responses are organized in the auditory cortex and in the network architectures leading to their generation. Cortical generators modeled for P2 are centered in the A2 region [1–4], where neurons are known to be highly plastic [8]. Patterns of connectivity and/or diminished surround inhibition in these regions may allow P2 cortical representations to expand with acoustic training as specific spectral features are coded. N1 events may reflect activity in cortical regions and/or in neocortical laminae where surround inhibition may be stronger in the adult brain. If so, this factor would be expected to normalize population activity underlying N1 when spectral content is manipulated, constraining the effect of spectral bandwidth and the effect of acoustic training on the response. N1 and P2 appear to be generated by current sinks occurring in different neocortical laminae [21] where laminar differences in inhibition and plasticity have been described for other sensory systems [22]. Constraints on N1 may be less prominent in the maturing brain where neural networks of the superficial neocortical laminae undergo major development between the ages of 5 and 12 years [23] and where evidence of N1 and P2 plasticity has been reported [24,25].

Conclusion

P2 amplitude evoked by piano tones is enhanced preferentially in musicians by the presence of natural upper harmonics that the musicians would have heard during the practice of their piano skills. The results are consistent with acoustic training studies showing P2 to be a highly plastic brain event and with the principle that the auditory cortex encodes the specific features of acoustic stimuli experienced during practice. The effect of spectral complexity on P2 is larger than on N1 when temporal envelope is held constant, pointing to different mechanisms underlying these responses.

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