Neuroplastic Adaptations of the Auditory System in Musicians and Nonmusicians

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1. INTRODUCTION

The human ear has been adapted by evolutionary processes to respond to sound frequencies that are present in the environment and convey information relevant to survival and reproductive fitness. However, the specific features of most sounds that we hear on a second-by-second basis (for example, the harmonic structure, loudness, and temporal shape of a particular voice, language, or musical note) and the meaning attached to these sounds are unique for each individual and cannot be anticipated by a genetic code. The evolutionary response to this limitation on natural selection has been the development of mechanisms that represent the detailed features of sensory input (sensory maps) and update those representations on a millisecond time scale (neural plasticity). We describe two experiments which used auditory evoked potentials (AEPs) to study these processes in the human brain.

2. EXPERIMENT 1

Alteration of the tuning properties of auditory neurons by aversive conditioning in the adult guinea pig has been documented in primary (A1) and secondary (A2) auditory cortex as well as in the medial, dorsal, and ventral divisions of the auditory thalamus (Edeline 1999). When brain

regions are contrasted with the same training procedure, tone-evoked plasticity is expressed more commonly by neurons in A1 (95%) than by neurons in A2 (62%; Diamond and Weinberger, 1984). Neural plasticity of the magnitude seen in these and other animal studies suggests that cortical reorganization induced by behavioral training in humans should be expressed in AEPs which reflect the activity of populations of neurons in the brain. We therefore trained 8 nonmusician subjects to detect small increments in pitch from a carrier frequency of 2.0 kHz using 40-Hz amplitude modulated tones (Bosnyak *et al.*, 2004). This stimulus procedure allowed us to separate transient components of the AEP which have been localized to spatially differentiable generators distributed in the belt and parabelt regions of A2 from the 40-Hz "steady-state" response (SSR) whose sources localize to Heschl's gyrus in A1 (Schneider *et al.*, 2002). In this way the effects of training on distributed auditory cortical representations could be examined.

Subjects were trained for 15 daily sessions to discriminate between an S1 stimulus of 2.0 kHz and an S2 stimulus of either the same or a slightly higher frequency (each stimulus 1s duration separated by 0.5 sec). On each of 480 trials in a training session subjects stated whether the S1 and S2 were the same or different and were informed of the correctness of their decision. Two "test" sessions were also administered, one before the training series and the second after its completion, which evaluated discrimination ability without feedback for the trained stimulus set (2.0 kHz S1) and for two untrained control sets 200 Hz lower (1.8 kHz S1) or 200 Hz higher (2.2 kHz S1) than the trained stimuli. All stimuli were 40-Hz amplitude-modulated pure tones. The EEG was measured continuously (64 channels, sampled dc to 100 Hz @ 500 Hz using a Cz reference, re-referenced offline to an average reference) on the two test sessions and on the 3rd and 13th sessions of discrimination training. Subjects gained familiarity with 40 Hz AM tones in a preliminary session administered before the first test session in which auditory thresholds and initial discrimination ability at 2.0 kHz were assessed without feedback using staircase procedures.

Behavioral performance (hit rate corrected for false alarms) is summarized in Figure 1a (upper panel) where it can be seen that discrimination on the trained stimulus set improved rapidly in the early sessions and more gradually thereafter. Improvement was confirmed by comparing the before/after test sessions ($p<0.001$) and sessions 3 and 13 of training ($p<0.05$). Subjects also improved on the untrained stimulus sets ($p<.02$ in each case, lower panel, Figure 1a) although to a lesser degree than on the trained stimuli. These results were corroborated by d′ values and psychophysical functions plotted for each subject and stimulus set.

We analyzed the EEG for the 2.0 kHz S1 which the subjects experienced most frequently over training and was not interrupted by behavioral responses. Two transient AEPs were augmented by discrimination training

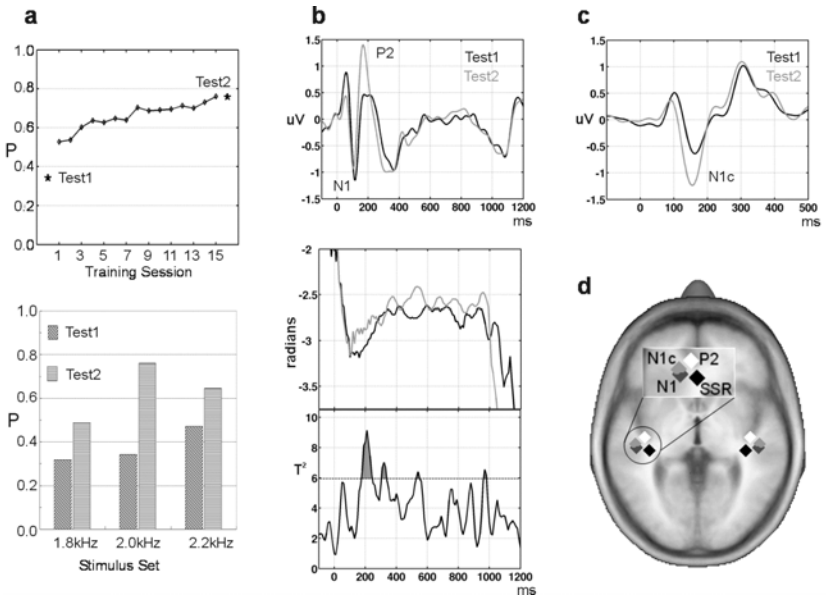

Figure 1. a. Behavioral performance [P=P(H)-P(FA)/1-p(FA)] on trained stimulus set (2.0 kHz S1, upper) and before/after differences on trained and control sets (lower). b. Transient AEP evoked by the trained 2.0 kHz S1 in before/after test sessions (upper), phase of the SSR in the two test sessions (middle), and bivariate T^2 evaluating before/after SSR differences (lower; horizontal line $p<0.01$, Monte Carlo determination), at electrode Fz. c. N1c before and after training (T_8 electrode, right hemisphere). d. Source localizations.

when before/after test sessions were compared, the P2 (latency 170 ms, Figure 1b, upper panel, $p<0.001$) and, in the right hemisphere only, the N1c (latency 155 ms, Figure 1c, $p = 0.007$). P2 and N1c responses evoked by untrained S1 control stimuli were also larger after training than before, but before/after differences did not reach significance. The amplitude of the N1 (Figure 1b, upper panel) did not change with training although N1 latency decreased by 9 ms ($p<.001$).

The SSR was analyzed by sliding a bivariate T^2 statistic sensitive to phase and amplitude in a window 100 ms wide across the EEG in 10 ms time steps, correcting for phase shifts induced by moving the window at each step. T^2 statistics comparing before/after SSR differences revealed a training-induced modification of the SSR commencing near the P2 (Figure 1b, lower panel) which was caused by a shortening of SSR phase (Figure 1b, middle panel) with no consistent effect on SSR amplitude (amplitude not shown;

animated phase and amplitude dynamics can be viewed at www.psychology.mcmaster.ca/hnplab). The phase effect was more pronounced for the trained 2.0 kHz S1 than for the untrained control S1 stimuli ($p<0.05$), although some generalization to the untrained 2.2 kHz S1 was observed (these results not shown). No 40-Hz activity corresponding to the phase effect was detected in a control experiment (n=10 subjects) in which P2 transient responses were evoked by unmodulated 2.0 kHz tones. This finding indicates that modification of SSR phase by discrimination training was not caused by a 40 Hz component of the P2 transient response but was a separate brain event.

Regional sources were modeled for each AEP (six determinations for each AEP, based on the three stimulus sets before and after training) and are averaged in the axial plane in Figure 1d. Sources for the SSR localized medially with respect to those of the N1, N1c, and P2 ($p<0.03$ or better) and posterior to the P2 ($p<0.0002$). These results are consistent with studies which have localized SSR generators by source modeling (Schneider *et al.*, 2002) and by intracortical measurements (Godey *et al.*, 2001; Liégeois-Chauvel *et al.*, 1993) to Heschl's gyrus. Differentiation of SSR sources from those of the N1, N1c, and P2 is also in agreement with previous findings reviewed by Shahin *et al.* (2003) which have localized N1, N1c, and P2 sources to the region of A2 including P2 sites anterior to the auditory core. P2 sources may reflect activation centered in anterior auditory belt regions which receive reciprocal connections from one another and from parabelt zones that project reciprocally to prefrontal cortex (Kass and Hackett, 1998). The N1 and N1c sources shown in Figure 1d localized laterally to those of the P2 ($p<0.0005$) and may reflect activation of posterior and lateral parabelt regions which have dense connections with caudal and rostral parts of the superior temporal gyrus. A note of caution is that source analysis differentiates only centers of activation and cannot resolve overlapping generators of similar orientation or determine their spatial extent.

Enhancement of the N1c and P2 in our data suggests that the number of A2 neurons depolarizing synchronously was increased by training on the discrimination task. This finding is consistent with animal data indicating that plasticity is a general property of A2 neurons. The expression of the N1c in the right hemisphere in our subjects is consistent with evidence for specialization of auditory neurons in this hemisphere for processing of spectral information (Zatorre and Belin, 2001). However, contrary to animal studies (Edeline, 1999; Recanzone *et al.*, 1993), our SSR results do not point to an expansion of the tonotopic representation for the trained frequencies in A1. Rather, the temporal properties of the response were modified such that SSR phase appeared to plateau more quickly after training than before training began. It is possible that competitive interactions induced by processing of multiple S2 frequencies during discrimination may have

preserved a segregated representation in A1, such that only temporal properties of the representation were affected (Kilgard *et al.*, 2001). Temporal effects obtained for the SSR and for the N1 and N1c invite the hypothesis that acoustic properties of the S1 stimulus may have been represented more rapidly after training compared to before training.

3. EXPERIMENT 2

The results of Experiment 1 showing that P2 and N1c responses are neuroplastic implies that these responses should be augmented when evoked by musical tones in highly skilled musicians who have processed musical stimuli extensively in the context of musical practice. Experiment 2 (Shahin *et al.*, 2003) evaluated this prediction.

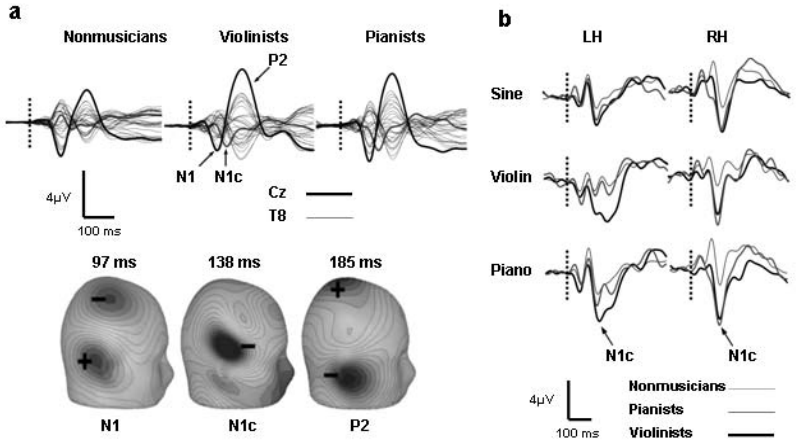

Figure 2. *a.* Upper panel shows 32 channel EEG response observed in nonmusician, violinist, and pianist groups averaged over sine, violin, and piano tones. N1 and P2 are identified in electrode C_z and N1c in T_8. Lower panel shows latency and scalp current density for N1, N1c, and P2. *b*. N1c evoked by each tone in the left (T_7) and right (T_8) hemispheres.

We studied 11 highly skilled violinists (age 24.3 ± 2.2 years) who were members of Canada's National Academy Orchestra and 9 skilled pianists (aged 23± 2.5 years) who had at least Grade 10 certification from Canada's Royal Conservatory of Music. Nonmusician controls (n=14) were age matched university students who did not play a musical instrument and reported listening passively to music for less than 1 hour/day. Subjects were presented with violin and piano tones (A3 and C3, American notation) and pure tones of the same fundamental frequency. Each tone (500 ms duration) was presented 120 times in a randomized order (free field, ISI 2s) while

subjects read a newspaper. The EEG was recorded from 32 channels (10-20 system, DC to 100 Hz) using methods described previously.

As predicted, we found an enhanced P2 in our two musician groups compared to nonmusician controls (Figure 2a, main effect of group $p<0.0001$, C_z electrode). A main effect of stimulus was also found ($p<0.0001$, not shown) which was attributable to a larger P2 occurring to piano and violin tones than to sine tones, but the interaction of stimulus with group was not significant. In addition an enhanced N1c was detected in our two musician groups compared to controls (group main effect $p<0.025$, electrodes T_7 and T_8) and is shown for each stimulus, group, and hemisphere in Figure 2b. Compared to control subjects both musician groups showed larger N1c responses to all stimuli in the right hemisphere ($p<0.04$ or better), particularly for violin and piano tones; group comparisons were not significant for any stimulus in the left hemisphere. No effects of group on N1 amplitude or latency were found. However, N1c latency was shorter for violin and piano tones compared to sine tones in the right hemisphere (tone by hemisphere interaction $p<0.025$). Regional sources fitted to the P2 localized medially to those of the N1 ($p<0.02$) and N1c ($p<0.0001$) in the region of auditory cortex, as was found in Experiment 1.

4. GENERAL DISCUSSION

In Experiment 2 the predicted enhancements of P2 and N1c in musicians were obtained for musical tones as well as for sine tones which have the quality of pitch. At present it is not clear whether musical skill is associated with augmented brain responses for sounds in general or only for sounds processed during musical practice (Pantev *et al.*, 2001; Shahin *et al.*, 2003). In our study enhancement of the P2 and N1c in musicians was not specific to the instrument of practice, perhaps because several violinists reported piano as a secondary instrument. Laboratory training results which show P2 and N1c responses to be neuroplastic (Experiment 1; cf. Tremblay *et al.* 2001 and Atienza *et al.* 2002 for the P2) indicate that intrinsic genetic and/or prenatal factors need not be invoked to explain augmentation of these responses in musicians, although such factors could play a contributing role.

Recently, the N19-P30 source waveform underlying the 40-Hz SSR has been reported to be augmented by 102% in professional musicians compared to nonmusicians when extracted by deconvolution from SSRs near 39 Hz (Schneider *et al.*, 2002). The SSR source waveform also correlated highly ($r = .87$) with the volume of gray matter in the anteromedial portion of Heschl's gyrus well as with musical aptitude ($r = .71$). Our results taken with those of Schneider *et al.* (2002) therefore suggest a dissociation of transient P2 and

N1c AEPs from the SSR, with the neuroplastic P2 and N1c expressing as amplitude enhancements in training studies and in musicians but SSR amplitude enhancement in musicians only where it could be an anatomical marker for musical skill. These findings call for study of the principles and mechanisms that govern cortical reorganization induced by experience over the life span and point to the tractability of their investigation in humans. Training procedures other than the one we studied in Experiment 1 may modify SSR amplitude and its anatomical substrate depending on the type of training that is given, its duration, and when it is delivered in the course of brain development.

ACKNOWLEDGEMENTS

This research was supported by grants from NSERC (operating) and CIHR (Operating and NET) of Canada.

REFERENCES

Atienza, M., Cantero, J. L., and Dominguez-Marin, E., 2002, The time course of neural changes underlying auditory perceptual learning. *Learn. Mem.* **9**: 138-150.

Bosnyak, D. J., Eaton, R. A., and Roberts, L. E., 2004, Distributed auditory cortical representations are modified when nonmusicians are trained at pitch discrimination with 40-Hz amplitude modulated tones. *Cereb. Cortex*, in press.

Diamond, D. M., and Weinberger, N. M., 1984, Physiological plasticity of single neurons in auditory cortex of the cat during acquisition of the pupillary conditioned response: II. Secondary field (AII). *Behav. Neurosci.* **98**: 189-210.

Godey, B., Schwartz, D., de Graaf, J. B., Chauvel, P., and Liégeois-Chauvel, C., 2001, Neuromagnetic source localization of auditory evoked fields and intracerebral evoked potentials: A comparison of data in the same patients. *Clin. Neurophysiol.* **112**: 1850-1859.

Edeline, J., 1999, Learning-induced physiological plasticity in the thalamo-cortical sensory systems: A critical evaluation of receptive field plasticity, map changes and their potential mechanisms. *Prog. Neurobiol.* **57**: 165-224.

Kaas, J. H., and Hackett, T. A., 1998, Subdivisions of auditory cortex and levels of processing in primates. *Audiol. Neurootol.* **3**: 73-85.

Kilgard, M. P., Pandya, P. K., Vazquez, J., Gehi, A., Schreiner, C. E., and Merzenich, M. M., 2001, Sensory input directs spatial and temporal plasticity in primary auditory cortex. *J. Neurophysiol*. **86**: 326-338.

Liégeois-Chauvel, C., Musolino, A., Badier, J. M., Marquis, P., and Chauvel, P., 1993, Evoked potentials recorded from the auditory cortex in man: evaluation and topography of the middle latency components. *Electroenceph. Clin. Neurophysiol.* **92**: 204-214.

Pantev, C., Roberts, L. E., Schulz, M., Engelien, A., and Ross, B., 2001, Timbre-specific enhancement of auditory cortical representations in musicians. *NeuroReport* **12**: 169-174.

Recanzone, G. H., Schreiner, C. E., and Merzenich, M. M., 1993, Plasticity in the frequency representation of primary auditory cortex following discrimination training in adult owl monkeys. *J. Neurosci.* **13**: 87-103.

Schneider, P., Scherg, M., Dosch, H. G., Specht, H. J., Gutschalk, A., and Rupp, A., 2002, Morphology of Heschl's gyrus reflects enhanced activation in the auditory cortex of musicians. *Nat. Neurosci.* **5**: 688-694.

Shahin, A., Bosnyak, D. J., Trainor, D. J., and Roberts, L. E., 2003, Enhancement of Neuroplastic P2 and N1c Auditory Evoked Potentials in Musicians. *J. Neurosci.* **23**: 5545-5552.

Tremblay, K., Kraus, N., McGee, T., Ponton, C., and Otis, B., 2001, Central auditory plasticity: Changes in the N1-P2 complex after speech-sound training. *Ear Hear.* **22**: 79-90.

Zatorre, R. J., and Berlin, P., 2001, Spectral and temporal processing in human auditory cortex. *Cerebral Cortex* **11**: 946-953.