

Automatic Brain Responses to Pitch Changes in Congenital Amusia

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Congenital amusia is a lifelong disorder affecting the processing of pitch. This pitch deficit can be traced down to abnormal brain responses elicited by pitch changes smaller than a semitone in conditions requiring attention. Here, we use the mismatch negativity (MMN) to investigate pre-attentive pitch change detection in 10 amusics and eight matched controls. Results indicate similar MMN in amusics and controls, even for an eighth of a tone change, revealing that the amusic brain can process small pitch changes at a pre-attentive level. Thus, the pitch deficit in congenital amusia may be related to a problem of perceptual awareness.

Key words: congenital amusia; auditory ERP; mismatch negativity; pitch change detection

Introduction

Congenital amusia is a lifelong disorder that is characterized by difficulties in acquiring basic musical skills, such as music perception and recognition despite normal hearing and intelligence.¹ Behavioral studies have shown that the deficit underlying congenital amusia is critically dependent upon fine-grained pitch discrimination.^{2,3} Indeed, when asked to detect a deviant tone that varies in pitch within a sequence of successive identical tones, amusics have problems detecting pitch changes that are smaller than a semitone.²

In a recent electrophysiology study, Peretz *et al.*⁴ found that the fine-grained deficits in pitch processing could be related to abnormal electrical brain responses. They measured the electroencephalogram (EEG) and used the event-related potential (ERP) method to examine brain responses in amusic individuals while they were monitoring short sequences of tones for the presence of a pitch change. They found

that the amusic brain did not respond to pitch deviances smaller than one semitone, whereas the normal brain did so reliably. In contrast, the amusic brain “over-reacted” to large pitch changes, by eliciting an N2 wave (which was not present in normals) and a P3 wave that was almost twice as large as that observed in controls’ brains. These results suggest that the pitch deficit underlying congenital amusia might depend on limited attentional resources.

Here, we tested this attentional hypothesis by assessing the presence of a mismatch negativity (MMN) wave in response to small pitch deviations in amusia. The MMN wave is an ERP component known to reflect pre-attentive change detection.⁵ According to a recent study by Braun *et al.*,⁶ the MMN should be altered in tune-deafness. However, the tune-deaf population tested by Braun *et al.*⁶ was selected on different criteria than the ones used here. Therefore, it was important to assess the MMN in amusia with the same population and the same experimental context as used previously,⁴ under controlled attentional demands. In the present study, we used the MMN to investigate automatic pitch change detection in an acoustical context in individuals suffering from congenital amusia.

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Method

Ten amusic adults (five men; mean age, 64.7 years; mean education, 17.5 years) and eight matched controls (five men; mean age, 65.4 years; mean education, 16.4 years) with no musical impairment were selected for this study. To be considered amusic, subjects had to obtain an average score two standard deviations below the average of the controls on the Montreal Battery of Evaluation of Amusia.⁷

The auditory stimuli were tones synthesized in a piano timbre with a duration of 100 ms (rise and fall of 10 ms). They were presented at a fixed inter-stimulus interval of 400 ms. The experimental stimulation consisted of a frequent standard tone played at a pitch level of C6 (1047 Hz). The rare deviant tones were lower or higher in pitch than the standard tone, with pitch differences of 25 cents (1032 or 1062 Hz) or 200 cents (933 or 1175 Hz; 100 cents corresponds to one semitone). The sequence of tones contained 2700 standard tones (probability of occurrence = 0.9) and 150 tones for each pitch deviance (probability of occurrence = 0.05) for a total of 3000 sounds. The presentation of the sounds was pseudo-randomized so that every deviant tone was preceded by at least four standard tones.

The subjects were seated in an electrically shielded and sound-attenuated chamber approximately one meter away from an 18-inch computer screen. They were instructed to ignore the auditory stimulation and to watch a self-selected movie presented in silence with subtitles. The auditory stimulation was presented binaurally through headphones and the intensity level was adjusted to each individual's hearing level for an average intensity level of presentation of 70 dB SPL throughout.

The EEG was recorded (bandpass, 0.05–70 Hz; sampling rate, 256 Hz; impedance < 5 K Ω) via a Neuroscan amplifier (Neuroscan SynAmps²; Compumedics, El Paso, TX) from 66 electrodes at the standard 10–10 scalp sites referenced online to the tip of the nose. Bipolar electrode pairs monitored hori-

zontal and vertical electro-oculograms (EOGs). Offline, the EEG data were corrected for eye movement,⁸ and the signal was filtered (bandpass, 0.05–30 Hz, 24 dB/octave). An artifact rejection was conducted on all EEG channels except for the EOG ($\pm 100 \mu\text{V}$). The data were then divided into epochs of 600 ms including a 100 ms pre-stimulus interval for baseline correction and re-referenced to the averaged mastoids. Separate averages were computed for the standard tones and for both pitch deviances (25 cents and 200 cents). MMN waveforms were obtained by subtracting the waveform for the standard tone from the waveform of each pitch deviance. The MMN amplitudes were quantified by computing the mean amplitude over a 40-ms window centered at the grand average peak latency detected within a time window of 100 to 250 ms, and the latency was measured as the time point of peak amplitude. The results are reported at the electrode Fz, where the MMN amplitude was the largest.

Results

As can be seen in Figure 1, both pitch deviances elicited an MMN in the amusic group as well as in the control group. A mixed ANOVA with group (amusic or control) as a between-subjects factor and pitch distance (25 or 200 cents) as a within-subjects factor conducted on the measures of MMN amplitude yielded a main effect of pitch distance, revealing that the MMN was smaller in the 25-cents condition than in the 200-cents condition, $F(1, 16) = 77.84$, $P < 0.001$. No significant effect of group $F(1, 16) = 1.53$, n.s., nor any significant interaction between group and pitch deviance was found, $F(1, 16) = 0.54$, n.s., revealing that the mean amplitude of the MMN for the amusic group did not differ significantly from that of the control group. Although the difference in MMN amplitude between the amusics and the controls was not significant, we note that there was numerical difference, visible in the waveforms shown in Figure 1, in which

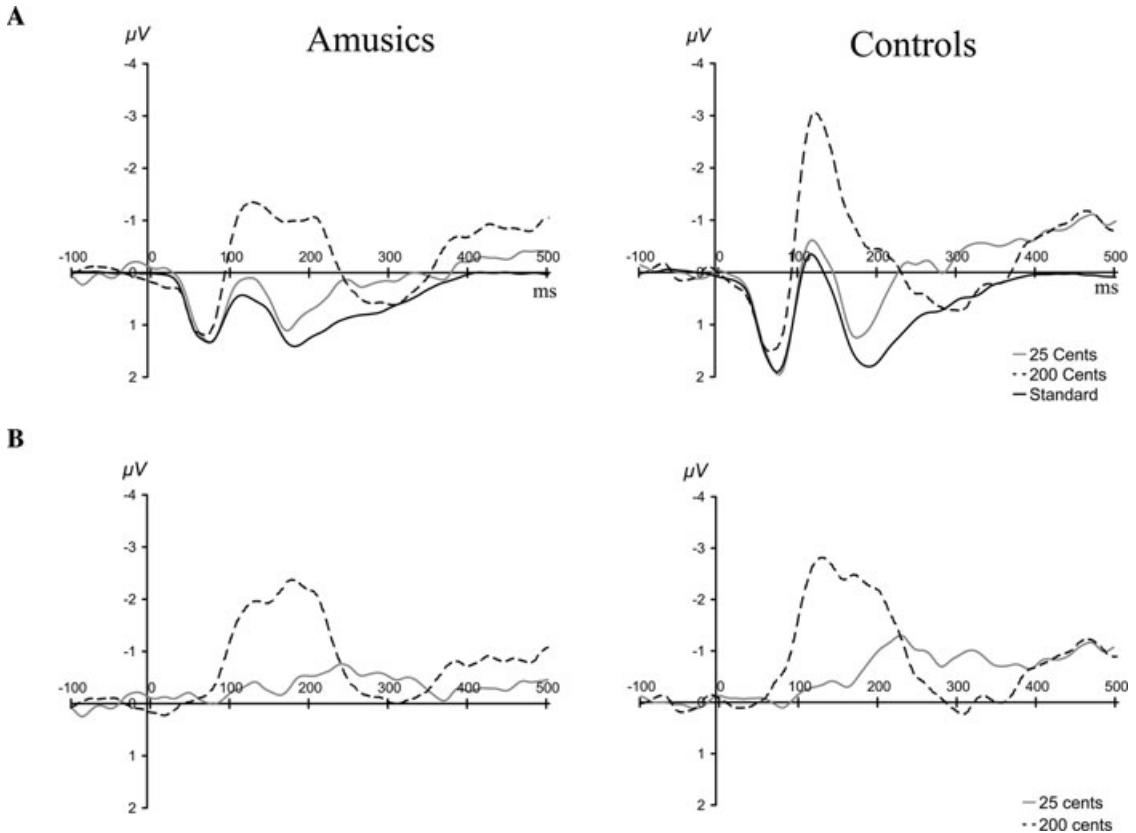


Figure 1. (A) Grand average ERP waves obtained for the standard sound and the 25- and 200-cents pitch differences for the amusics and the control subjects at Fz. (B) MMN difference waves (deviant minus standard) for the 25-cents and 200-cents pitch differences for the amusics and the control subjects at Fz.

the MMN amplitude was lower in the amusic group compared to the controls. Importantly, the MMN elicited by the 25-cents deviation was significantly different from base line, with $t = -3.23$, $P < 0.05$ in amusics and $t = -4.17$, $P < 0.05$ in controls, showing that an MMN was elicited by this small pitch difference even for the amusics.

A similar mixed ANOVA was also conducted on the measures of MMN latency, which yielded a main effect of pitch distance, $F(1, 16) = 31.72$, $P < 0.001$, reflecting that MMN latency was delayed for the 25-cents condition as compared to the 200-cents condition. There was no main effect of group (amusics versus controls), $F(1, 16) = 0.44$, n.s., nor any significant interaction, $F(1, 16) = 4.66$, n.s.

Conclusion

The major finding of this study is that amusic individuals display normal pitch change detection in an acoustical context, as indexed by similar MMN ERP components elicited by pitch changes corresponding to an eighth of a tone in the amusic and the control brain. The MMN amplitude and latency changes typically associated with the physical characteristics of pitch differences (25 versus 200 cents) were preserved in both groups, revealing intact automatic change detection for both pitch differences. It appears that the amusic brain can accurately process small pitch changes at an early pre-attentive level of processing.

In line with Peretz *et al.*'s latest results,⁹ the present study adds to our comprehension of

the origin of the fine-grained pitch deficit underlying congenital amusia by demonstrating that the amusic brain can perceive small pitch changes without awareness. It suggests that congenital amusia may originate from a deficit in conscious perception of pitch changes.

Conflicts of Interest

The authors declare no conflicts of interest.

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