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Musicogenic epilepsy \^{1,2} is a rare medical condition generally classified as a specific stimulus-triggered (reflex) epilepsy. It is characterized by a long latency between stimulus exposure and seizure induction, frequently in the range of minutes. Musicogenic seizures involve temporal lobe structures \^{1,2} and are most frequently complex partial.

The uniqueness and specificity of the musical triggers include a wide range: the sound of particular church bells, the melody of the Marseillaise, the metallic character of a singer’s voice, or the sound of a street vendor’s flute, only at sunset. \^{1,2} In some cases the trigger for seizures was the actual performance of a specific musical piece on a given instrument. Emotional cofactors may contribute to the development of a brain state close to a threshold from which seizure activity may be initiated. \^{2}

Few studies have investigated the effects of epi-

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**References**

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**fMRI of triggerable aurae in musicogenic epilepsy**

I.Á. Mórocz, MD; A. Karni, MD, PhD; S. Haut, MD; G. Lantos, MD; and G. Liu, PhD

**Abstract**—The authors studied a patient with musicogenic epilepsy triggered by one specific musical piece using 3D PRESTO fMRI. During epileptic aurae initiated by the stimulus, signal increases were found in the left anterior temporal lobe, correlating with ictal EEG and SPECT showing a left anterior temporal focus, and the right gyrus rectus. Because fMRI indicated a cascade of recruitment of the ventral frontal lobes by epileptogenic music, left anterior temporal lobe activity could be secondary to a right gyrus rectus focus, possibly triggered by emotional processing of music.

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leptonic activity on the fMRI signal in patients with spontaneous epileptic discharges. Here we report an fMRI study in an individual with medically well-controlled musicogenic seizures where blood oxygenation level dependent (BOLD) signal changes were induced by epileptic auras upon exposure to specific musicogenic music.

**Materials and methods.** Patient A 48-year-old right-handed woman had a history of music-induced “strange feelings” since age 41. Beginning at age 42, she had music-induced complex partial seizures. The musical triggers were one song performed by Whitney Houston and one by Boyz II Men. She underwent continuous EEG monitoring during which four episodes of musicogenic complex partial seizures (triggered by music by Boyz II Men) with a left anterior temporal focus were captured (figure 1). An ictal SPECT scan showed left temporal hyperperfusion; the interictal SPECT revealed mild hypoperfusion in the same area. Results on MRI studies and neurologic examination were normal. Medication included phenobarbital and Tegretol (Novartis, East Hanover, NJ).

**Music tasks.** The tune “I believe in you and me” by Whitney Houston was selected as the trigger condition causing strong aurae feelings: pressure in the abdominal and then pectoral area, a “rushing” sensation, palpitations, and heart racing. A similar sounding song (“Somebody bigger than you and I,” from the same album) served as control condition. Both tunes were played each session (twice in the fifth session). Average response time for the aura onset in 5 of 10 fMRI sessions (twice in the fifth session). Average response time for the first button press after initiation of the epileptogenic music was 23.6 seconds (SD1 = 5.6 seconds, n = 5). No abnormal movements or adverse reactions were observed. Pulse rate increased from about 92 beats per minute (bpm) to 106 to 110 bpm toward the end of the control music conditions with maximum of 110 to 116 bpm reached by the end of the seizure triggering music conditions. Figure 2 shows the SPM99 comparison of the two music conditions (epileptogenic vs control music): signal increases were found in both aura sessions and nonaura sessions in the bilateral frontal poles, right anterior cingulate, and the right gyrus rectus (rGR), whereas signal decreases occurred bilaterally in the caudal GR and adjacent structures in the orbital and subcallosal cingular gyri (not shown). Differential activation for the aura sessions (during epileptogenic music) was evident in rGR and lATL. The raw fMRI signal time course (figure 2, lower panels) shows signal increases for the epileptogenic music in rGR during the first four aura sessions.

**Results.** The patient reported an aura onset in 5 of 10 fMRI sessions (twice in the fifth session). Average response time for the first button press after initiation of the epileptogenic music was 23.6 seconds (SD1 = 5.6 seconds, n = 5). No abnormal movements or adverse reactions were observed. Pulse rate increased from about 92 beats per minute (bpm) to 106 to 110 bpm toward the end of the control music conditions with maximum of 110 to 116 bpm reached by the end of the seizure triggering music conditions. Figure 2 shows the SPM99 comparison of the two music conditions (epileptogenic vs control music): signal increases were found in both aura sessions and nonaura sessions in the bilateral frontal poles, right anterior cingulate, and the right gyrus rectus (rGR), whereas signal decreases occurred bilaterally in the caudal GR and adjacent structures in the orbital and subcallosal cingular gyri (not shown). Differential activation for the aura sessions (during epileptogenic music) was evident in rGR and lATL. The raw fMRI signal time course (figure 2, lower panels) shows signal increases for the epileptogenic music in rGR during the first four aura sessions.

**Similar effects of music conditions in the frontal cortex were also found in the ICA analysis. Figure 3 depicts the spatial extent and time course of the ICA component best corresponding to the stimulus paradigm (frequency spectrum) in all 10 sessions. A comparison of the signal amplitudes in the two music conditions—control and epileptogenic—during the aura vs nonaura sessions was significant (interaction between music × aura, gen-

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**Figure 1. EEG tracing acquired while the patient was exposed to the epileptogenic music. Left temporal seizure activity: a period of 9-rhythmic waves lasting 6 seconds was followed by discharges of lower voltage and higher frequency, which persisted through the end of the record. Electrode positions are indicated. Each gridline represents 1 second.**

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**Figure 2.** Location of significant activity—red line indicates group level activity (P < 0.001 uncorrected). Graphs at right show time course of the significant source at each voxel in that area. **A** topography of significant activity in the left hemisphere during aura vs nonaura sessions is shown. Significant activity is seen bilaterally in the posterior cingulate and the precuneus. Significant activity is seen bilaterally in the caudal middle frontal gyrus (IFG), and the right orbital frontal cortex (OFC) showing a trend toward higher activity in aura vs nonaura sessions. **B** topography of significant activity in the right hemisphere during aura vs nonaura sessions is shown. Significant activity is seen bilaterally in the inferior parietal lobule (IPL) and the lateral occipital cortex (LOC) showing a trend toward higher activity in aura vs nonaura sessions.
Figure 2. fMRI activity maps for the epileptogenic music effect and for the aura effect superimposed on anatomic MRI slices (slice numbers and positions relative to anterior commissure/posterior commissure plane indicated). Red and yellow represent voxels with significant fMRI signal increase for the seizure music conditions: red in the nonaura sessions, yellow in the aura sessions. Orange voxels represent overlapping signal increases in both session types. Voxels in green in the rGR and the left anterior temporal lobe (laTL) demonstrate significantly higher fMRI signals during the epileptogenic music in the aura as compared to the nonaura sessions. Lower panels show the averaged fMRI signal time course: top, the 10 green voxels in the rGR; middle, the orange, yellow, and green voxels in the rGR; bottom, green and yellow laTL voxels. Blue curve = control music conditions; pink curve = seizure music conditions. Vertical yellow marks indicate aura onset report times.
eral linear model–analysis of variance for repeated measures, $F[1,7] = 20.97, p < 0.0025$).

Two mathematical control experiments tested for movement-related artifacts in areas prone to susceptibility by movement artifacts. The inclusion of movement parameter estimates as covariates in the SPM99 design matrix and the application of the Unwarp technique (http://www.fil.ion.ucl.ac.uk/spm/toolbox/unwarp.html) made no substantial difference in the analysis results.

**Discussion.** Repeated exposure to the unique seizure-triggering music resulted in two distinct patterns of consistent BOLD signal changes: one related to the actual triggering of musicogenic auras, the other related to exposure to the specific epileptogenic music. The fMRI data and the ictal EEG and SPECT measurements indicated the latTL as a locus for seizure-related activity. However, the PRESTO fMRI measurements not only revealed additional foci in the ventral frontal lobes but also indicated that the rGR activation, occurring at an earlier phase of exposure to epileptogenic music, may have initiated the seizure cascade. This is supported by the finding that the latTL (not known to play any role in music processing) was not activated by seizure-music exposure per se, as the fronto-orbital lobes were. The fronto-orbital structures are believed to be key structures in processing emotional aspects of music.

Fronto-orbital activation was found in a PET study in which the effect of increasingly pleasant music was investigated. Lesion studies also support that emotional processing of music depends on the fronto-orbital brain. The patient reported here expressed no interest in music in general, has never played a musical instrument, and had no particular memories or feelings related to the triggering pieces.
of music. However, her pulse measurements indicated that she was having an autonomic response even before being exposed to the epileptogenic music.

Because our experimental fMRI design was inherently sensitive to the effect of prolonged listening to music (control followed by epileptogenic music), the observed activity changes in the fronto-orbital lobes may have reflected emotional arousal and memory related to the music rather than seizure activity per se. Our findings suggest that during the patient's aura, the main differential evoked activity was localized in the rGR. Nevertheless, the control music may have contributed to the enhancement of the patient's susceptibility to the ensuing seizure-inducing music, possibly in the form of progressive cortical recruitment. Indeed, the large negative activations that surrounded the rGR in the epileptogenic music conditions may indicate uncompensated hypermetabolism or vascular dysregulation.

Even within the relatively short time frame of the fMRI study, the ability of the same stimulus to evoke an epileptic aura varied. The imminent exposure to the feared stimulus and the foreign atmosphere of an fMRI experiment may have contributed to the failure to induce epileptic auras in the first three exposures, whereas the results for the final two sessions are suggestive of habituation to the repeated stimulus presentation. A similar habituation was found for the signal in the rGR during the aura sessions although a clear-cut correlation with the button press latencies was not evident.

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References

Trends in dementia mortality from two National Mortality Followback Surveys

Daniel J. Foley, MS; Dwight B. Brock, PhD; and Douglas J. Lanska, MD, MS, MSPH

Abstract—The National Center for Health Statistics conducted National Mortality Followback Surveys (NMFS) in 1986 and 1993. The next-of-kin's report of a physician’s diagnosis of AD before death and a listing of AD or other dementia as the underlying cause increased significantly among women but remained stable among men. Currently, AD is among the top 10 leading causes of death in elderly white men and women in the United States.

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As the US population ages, mortality from dementia is likely to increase concomitantly due to the well-known association between incidence of dementia-related diseases and aging. Planning for future health care services, especially long-term care, is served by knowledge of trends in dementia mortality. The National Center for Health Statistics (NCHS) provided data for an initial examination of national estimates of dementia mortality in the 1986 National Mortality Followback Survey (NMFS). Although death certificate data are known to seriously underestimate the prevalence of dementia, the NMFS also provided rates based on next-of-kin interviews addressing the decedent’s history of physician diagnoses and disabilities before death.

In 1993, the NCHS conducted a second NMFS.
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