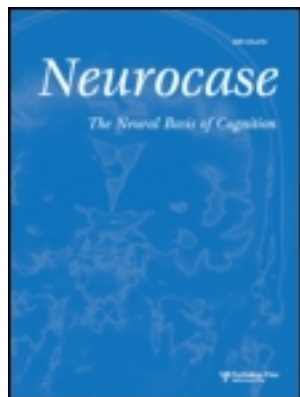


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Neurofunctional assessment in a stroke patient with musical hallucinations

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We reported a case of an elderly female patient affected by musical hallucinations (MHs) as the unique symptom of a right temporal ischemic stroke. A functional magnetic resonance imaging examination was performed in the patient and in five age- and sex-matched normal controls (NC) to detect the complex neural substrate subserving MHs in such a context. Although an activation pattern involving the primary auditory cortex and the temporal associative areas bilaterally was found in the patient and NC, a significant increased activation mostly located in right temporal cortex (in the ischemic area), was observed in the patient. Further functional neuroimaging studies should be performed to detect the complex neural pathways underlying MHs and to find out differences between these hallucinations and real music perception.

Keywords: Functional magnetic resonance imaging; Ischemic stroke; Musical hallucinations.

Musical hallucinations (MHs) are a disorder of complex sound processing. Subjects perceive complex sound in the form of music in the absence of an acoustic stimulus. Thus, the phenomenon might be regarded as an example of mental imagery, defined as 'mental acts in which we seem to re-enact the experience of perceiving an object when the object is no longer available' (Halpern & Zatorre, 1999). Mental imagery is, however, usually less vivid than actual perception, and is never attributed to an external process. MHs may be experienced under a variety of conditions, including diseases of the ear, neurological and psychiatric disorders, toxic states, and as side-effects of antidepressant treatment (Keshavan, David, Steingard, & Lishman, 1992).

MHs are a disorder of the processing of the high-level pattern in sound. Music comprises discrete sounds that are characterized by fundamental

properties such as pitch and onset time. These sounds are built into a high-level pattern at the temporal level of hundreds of milliseconds. MHs require a mental representation of such high-level pattern which might be based on the same neural mechanism of that active during normal musical perception. Nevertheless, distinct mechanisms for musical perception and imagery are also possible.

Previous studies have suggested that the processing of discrete sounds depends on a different neural substrate from that involved in the processing of sound high-level patterns. Several studies have shown that the processing of pitch is related to the function of the human primary auditory cortex in Heschl's gyrus (Griffiths, Buchel, Frackowiak, & Patterson, 1998; Pantev, Hoke, Lutkenhoner, & Lehnerts, 1989; Zatorre, 1988), whilst the onset time of auditory transients is accurately represented in

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the primary auditory cortex of animals (Phillips & Hall, 1990).

Berrios (1990) has stated that MHs are far more common in females and that age, deafness, and brain disease affecting the non-dominant hemisphere play an important role in the development.

Structural neuroimaging, using computerized tomography (CT) or magnetic resonance imaging (MRI) is often used clinically to rule out a focal lesion such as a tumor or a stroke. Braun, Dumont, Duval, Hamel-Hébert, and Godbout (2003) reviewed a large number of case studies of post-lesion hallucinations in the visual, auditory, and somatic modalities. The authors report that the lesion is almost always located in the brain pathway of the sensory modality of the hallucination and suggest that compensatory over-activation of the nearby brain tissue as causative in hallucinosis.

The advent of neuroimaging techniques in the last two decades has allowed us to at least begin to formulate answers to this question. Techniques, such as functional magnetic resonance imaging (fMRI) have been widely used. Studies have also been directed at investigating the structural correlates of hallucinations, i.e., whether volume reductions of certain brain areas are associated with hallucinations.

By using fMRI, we studied an elderly female patient with MHs as the unique symptom of a right temporal brain infarction.

CASE REPORT

An 82-year-old, non-musician, elderly female patient was referred to our Centre for auditory hallucinations.

Family history was unremarkable. The patient was affected by severe arterial hypertension treated with sartanic, diuretic, and beta-blocker therapy. Two months before our evaluation, she presented with a transient moderate hemiparesis associated to hemi-hypoesthesia, but she refused admission to hospital as her symptoms recovered approximately 2 hours later. The day after, she started complaining of MHs that increased in intensity and frequency in the following weeks. When the patient was admitted to our hospital, she experienced complex MHs in the form of popular Italian songs (i.e., *Vola Colomba*, *Che Sarà*, *Sei Bellissima*, *Papaveri e Papere*): these were songs that the patient listened to when she was young and they represented her musical background.

She had insight of these hallucinations and did not present any psychiatric diseases or hearing defects. Neurological examination was unremarkable. Mini Mental State Examination (score = 28/30), Activities of daily living (score = 6/6), Instrumental Activities of daily living (score = 7/8), Hamilton Rate scale for Depression (score = 7) and Neuropsychiatric Inventory (9/120) were within the normal range.

She underwent a brain MRI that showed an ischemic lesion in the right temporal area (see Figure 1) as well as a mild leukoaraiosis. Cardiac evaluation, electroencephalography (EEG), color doppler ultrasound of the neck vessels, coagulation and autoimmunity markers were within normal limits. Interestingly, during one of her MHs, patient underwent a prolonged EEG recording that did not show any epileptic abnormalities, so to exclude a possible epileptics status. Thus, we decided to perform a neurofunctional examination to investigate the neuronal pathways involved in her MHs.

The patient and five age- and sex-matched normal controls (NC) underwent an fMRI examination on system operating at 1.5 T (Sonata Siemens, Erlangen, Germany). Blood oxygen-dependent contrast echo planar imaging scans were performed (TR = 3000 ms; TE = 40 ms, 25-cm field of view; 64 × 64 matrix for 10 × 7-mm slices). We digitally recorded and adapted a music segment

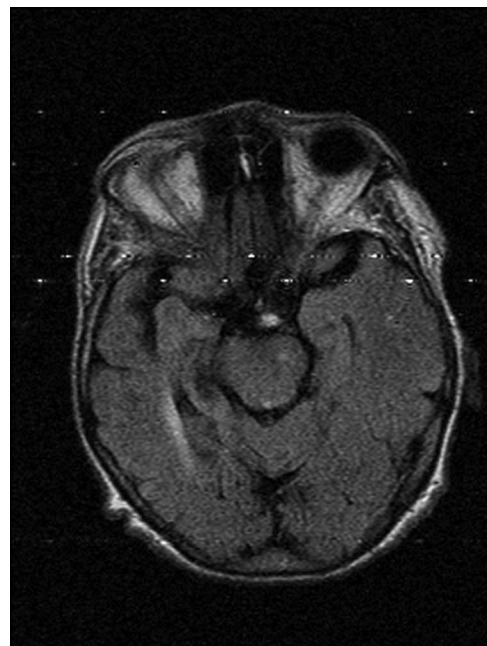


Figure 1. T2-weighted image showed the right temporal ischemic stroke.

lasting 5 minutes, containing the aforementioned songs. We performed an fMRI examination using a ‘cognitive task’ when the patient was without hallucinations, presuming to measure cognitive processes underlying the disposition to hallucinate: we used sensory stimuli in the same modality as the hallucinations in patient with hallucinations (an excerpt of the whole popular Italian songs, as monophonic instrumental voice, which were an exact replica of a recorded version). The subjects experienced different songs on different BOLD examinations.

The task used an event-related design in which the auditory stimuli were administered by a MRI-compatible noise attenuated headphones. Event-related design can detect transient variations in hemodynamic response, allow for analysis of individual response to trials, reduce subject’s expectation effects, and is less sensitive to head motion. The interstimulus-interval (ISI) of an event-related design is a very important parameter. We used a rapid event-related design referring to an ISI that is shorter than the duration of the hemodynamic response function (HRF). In fact, compared to the slow design, a rapid event-related design allows for an ISI that is similar to those used in the classical neuropsychological experiments – e.g., event-related potentials (ERPs) – and also increases the statistical power by allowing for more stimuli per time.

The auditory stimuli were the same for all subjects. All subjects underwent an acoustic-evoked potential, before the fMRI examination and all were similarly familiar with the selected music.

At the end of fMRI acquisition, high-resolution whole brain structural scan was acquired using a T1-weighted sequence (TR = 20 ms, TE = 3 ms, flip angle = 20°, 25 contiguous slices of 5 mm thickness acquired parallel to the AC-PC plane). Significantly activated voxels within each area were calculated. The data were processed and analyzed using advanced image analysis tools from the Oxford Centre for Functional Magnetic Resonance Imaging of the Brain (FMRIB) (Smith et al., 2004). Software Library (available at <http://www.fmrib.ox.ac.uk/fs>). Before statistical analysis, motion correction using MCFLIRT, spatial smoothing (Gaussian kernel of 5 mm full width at half maximum), intensity normalization, and non-linear high pass temporal filtering were applied. Statistical analysis was done using the general linear model with FMRIB’s improved linear model (FILM) to generate activation maps expressing relative signal change in

active versus rest blocks. Cluster detection was undertaken on voxels at a threshold of $Z > 2.3$. A corrected probability threshold of $p = .001$ was then applied to determine significant clusters. Using FLIRT, we registered the unthresholded statistical images from the first level analysis to the high resolution T1 weighted scan and transformed them into a standard brain space based on the MNI 152 template.

Taking the results from the first level analysis, we tested for anatomical correspondences between mean activations for data from both the patient and controls. To do this, we used a fixed effects analysis, which accounts for the small number of subjects in the groups, to contrast activation patterns between the patient and controls.

Both in the patient and controls, a significant activation of primary (Heschl’s Gyrus; left coordinates $-43, -28, 8$; right coordinates $47, -17, 7$, for the patient. Left coordinates $-44, -30, 11$; right coordinates $49, -20, 9$ for the controls) and associative (planum temporal) auditory cortex left coordinates $-50, -30, 10$; right coordinates $51, -19, 9$, for the patient. Left coordinates $-42, -27, 14$; right coordinates $44, -26, 12$ for the controls of both hemispheres was found.

However, there was more significant associative cortices activation after auditory stimuli in the patient compared to the controls (see Figure 2). Extent of activation was measured by number of voxels initially thresholded at $Z = 2.3$ with cluster probability at $p < .001$. For determination of the voxels of maximum signal change, the group mean parameter estimates were masked by the threshold, fixed effect Z -statistic images (at a corrected cluster significance threshold of $p < .001$) and the position of the local maxima in each independent activation cluster was defined.

An informed consent was signed by all participants. The study was approved by the Local Ethics Committee.

Interestingly, olanzapine up to 10 mg/day was then introduced with a complete resolution of MHs after approximately 2 weeks.

DISCUSSION

MHs are defined as complex auditory hallucinations. They usually consist of melodies that are repetitions of well-known music from former times, in particular religious, folk, and

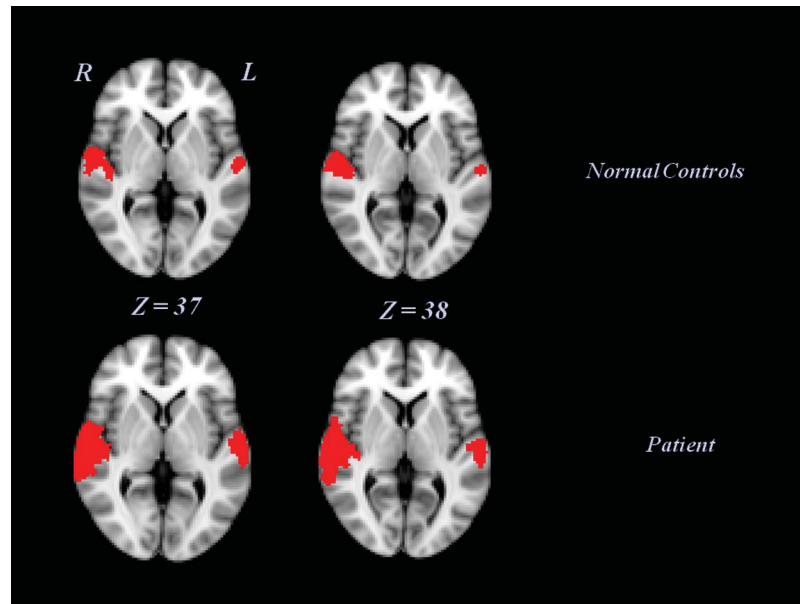


Figure 2. Mean random effect statistical activation maps for patient and controls, demonstrating regions of significant activation during auditory stimuli ($Z > 2.3$, $p = .001$, corrected). Activation is averaged over four runs for each subject and superimposed on a standard space image. [To view this figure in color, please visit the online version of this Journal.]

pop songs heard during childhood or adolescence. MHs are very unusual phenomena with a reported prevalence of 0.16% among a general hospital population of patients with psychiatric disorders, while in a sample of elderly subjects with audiological complaints; their prevalence was 2.5% (Cole, Dowson, Dendukuri, & Belzile, 2002). Nevertheless, the prevalence of MHs in other groups of patients or in the general population is still unknown. These phenomena seem to be more common in females, who constitute 70–80% of clinically significant sufferers, and in those aged over 60 years. MHs have been reported in people with hearing deficits, psychiatric and neurological diseases, and who also have adverse side-effects from different drugs such as opioids and tricyclics (Davies & Quinn, 2005; Griffiths, 2000; Terao, 1995). Focal brain lesions are an unusual cause of MHs and a few cases of stroke-related MHs have been described so far, especially after cortical strokes (Lampl, Lorberboym, Gilad, Boaz, & Sadeh, 2005; Warren & Schott, 2006). In general, MHs have been associated with right auditory cortex in musically untrained individuals, and with the left auditory cortex in the musicians.

In our patient, a plausible explanation of MHs was the right temporal ischemic stroke, since she did not present any neuropsychiatric or hearing diseases, and she was not taking any drugs potentially

inducing auditory hallucinations. Moreover, epileptic seizures were ruled out by recording a normal EEG during MHs.

There is little consensus on the optimal treatment for MHs. Different studies have reported variable relief with antipsychotics, antiepileptic drugs, benzodiazepines, and anticholinesterases, with patients exhibiting variable idiosyncratic response, especially in those without any evident cause (Cope & Baguley, 2009; Evers & Ellger, 2004). In our patient MHs disappeared after the administration of olanzapine; thus, a possible involvement of the dopaminergic system in hallucination production may be taken into account.

The role of central lesions in producing MHs has been the subject of controversy. Although gross structural lesions in the brainstem or either hemisphere can be associated with musical hallucinosis (Inzelberg, Vishnievskaya, & Korczyn, 1993; Murata, Naritomi, & Sawada, 1994; Paquier et al., 1992; Tanabe, Sawada, Asai, Okuda, & Shiraishi, 1986) such lesions are not seen in most patients with this condition.

Central vascular lesions might be the additional factor that distinguishes subjects with musical hallucinosis from subjects with the same degree of deafness without hallucinosis. Several mechanisms may be considered. Indeed, it is possible that vascular disease could lead to a degree of

disconnection between the primary auditory and association cortices. A second possibility is that vascular lesions within the network for the perception and imagery of segmented sound may alter the threshold for spontaneous activity within this network. We think that this mechanism could involve a network including deep brain structures, which are more likely to be affected by small vessel disease than the superior temporal cortex.

Nevertheless, although this deafferentation phenomenon may occur in some individuals, our fMRI study seems to support the hypothesis of a 'compensatory overactivation' of the lesion's nearby brain tissue as causative in MHs. Indeed, Braun et al. (2003) suggested that the lesioned area may contain a predominance of inhibitory over excitatory neurons for a specific sensory modality.

fMRI studies showed that hallucinations in a given modality involve areas that normally process sensory information in that modality (Allen, Larøi, McGuire, & Aleman, 2008; Kasai, Asada, Yumoto, Takeya, & Matsuda, 1999).

fMRI cognitive studies are based upon paradigms intended to uncover specific cognitive processing abnormalities or biases associated with the predisposition to hallucinate.

The simplest studies just present sensory stimuli in the same modality as the hallucinations in patients with hallucinations. The rationale behind these cognitive 'interference' studies is that lower activation in perceptual areas in response to external stimulation during periods of hallucination as compared to periods without hallucination would suggest that hallucinations and the processing of external auditory stimuli compete for common neurophysiological mechanisms. Studies by David et al. (1996) and Woodruff et al. (1997) provide evidence that sensory areas may be 'occupied' during the experience of hallucination. These investigators found that the auditory association cortex (superior temporal gyrus) is less responsive to external auditory stimulation during hallucinations compared to when they are absent. They interpret this as indicative of physiological competition for a common neural substrate by the hallucinations. This finding was confirmed in a recent study involving 15 patients with schizophrenia with daily auditory hallucinations for at least 3 months prior to the study (Plaze et al., 2006).

Thus, studies comparing brain activity during hallucination vs. non-hallucination periods identified wide-spread brain activation related to auditory verbal hallucinations (AVHs) including the

inferior frontal gyri (IFG), Broca's area, primary auditory cortex (Heschl's gyrus) and the temporal planum, particularly the right temporal cortex (Dierks et al., 1999; Lennox, Park, Jones, Morris, & Park, 1999; Shergill et al., 2001; Sommer et al., 2008).

Dierks et al. (1999) showed that the area in the transverse temporal gyrus (GTT) is activated during AVHs includes the transverse gyrus of Heschl in the dominant hemisphere and matches the location of primary auditory cortex. The activation was similar to the present study. In particular the activation of the GTT during hallucinations was found predominantly in the left hemisphere for the two right-handed patients and in the right hemisphere for the left-handed patient studied by the authors: this result is suggestive of a particular role for the language-dominant hemisphere in the generation of auditory hallucinations, even if it is limited to a small sample size of subjects, such as the present study.

Shergill et al. (2001) reported both middle and superior temporal gyri showing activation during auditory hallucinations. Similarly to our findings, although only limited conclusions can be drawn from single subject design, it is clearly advantageous to be able to map different modality hallucinations in the same subject, avoiding many of the issues in between group comparisons.

Although an activation pattern involving bilaterally the primary auditory cortex and the temporal association areas was observed in the 6 subjects, our patient (who did not present deafness problems) showed an increased activation of the temporal planum, mostly of the right temporal cortex, where the lesion existed. Therefore, the aforementioned 'overactivation' hypothesis of the lesion's nearby acoustic associative areas may also be assumed for our patient. In particular, the observed activation of primary and associative auditory cortex of both hemispheres is in line with earlier findings showing structural changes in the connections of the temporal lobe in subjects experiencing AVH (Hubl et al., 2004).

If the baseline neuronal activity of the auditory cortex is higher during hallucinations than in the resting state, the increase of neuronal activity attributable to external stimuli would be expected to be smaller, which would be reflected in an attenuated BOLD response to external acoustic stimulation.

We hypothesized that the cortical activations during the auditory tasks are very similar to those that

occur during an auditory hallucination, for two reasons: (1) the musical tasks are the same that caused the auditory hallucinations and (2) there is a difference in the activation between the patient and the normal control.

Moreover, our findings seem to demonstrate how brain activation within a large-scale neuronal network involved in the reality distortion symptoms (delusions and hallucinations) is associated to the subjective perception of the symptom. Indeed, it is likely that the primary auditory cortex is related to a specific form of hallucination, whereas other circuitries could be involved in other reality distortions. Further functional neuroimaging studies should be fostered to detect the complex neural pathways underlying MHs and to find out differences between these hallucinations and real music perception.

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