Transient musical hallucinosis

Paquier et al reported a patient who, following subarachnoid haemorrhage, developed musical hallucinosis. Based on a literature review, they suggested that musical hallucinosis, formed auditory perceptions that occur in the absence of external acoustic stimulus while the patient is aware of their non-real nature, may result from lesions of either side of the brain, and not necessarily from the auditory hemisphere, as previously proposed.1 A patient recently seen by us reinforces the authors’ conclusion. A 75 year old right handed woman had been suffering from severe hearing loss due to a presbycusis for about 30 years. Her past history revealed no insulin dependent diabetes mellitus, ischaemic heart disease, peripheral vascular disease and paroxysmal atrial fibrillation. In September 1992, her stroke suddenly developed right hemiparesis and dysphasia which recovered within a few weeks. Her CT scan revealed a left thalamic infarction, mild cortical atrophy and ventricular dilatation. A few days after the event, she started hearing a melody, which seemed in the first days to originate externally and was heard bilaterally. The melody she heard was extremely loud, leading her to ask surrounding people to turn off the radio, which she believed to be the source of the tune. The melody was suddenly, when it stopped, slow, clear and reminiscent of popular songs that she had heard in her youth, but were still unknown to her. She was able to sing this melody. Shortly after the onset of this phenomenon, she gained full insight into the problem and realised that this incessant tune originated in her own mind. The volume was variable and sometimes the melody was enjoyable; this volume was mostly high, especially during the night, disturbing her sleep, and severely interfering with her daily activities. Amitriptyline partially helped her sleep. The intensity of the music diminished during the following weeks, but the same melody persisted.

Musical hallucinations after stroke are reported rarely. Only three cases, all with right hemispheric pathology, were quoted in a recent review.2 Our patient illustrates the fact that dominant hemispheric stroke can also result in musical hallucinations.

As with several other reported cases, including that of Paquier et al, our patient had suffered from hearing loss for many years. Berrios in a review, pointed out that musical hallucinations are far more common in elderly, hearing impaired, female patients.3 It is possible that the musical hallucinations in our patient represent a “deafferentation” phenomenon, reminiscent of visual hallucinations in the blind, thalamic pains or phantom limb. It appears that both central and end organ pathology contribute to the appearance of musical hallucinations.4 The prolonged lack of normal input to cortical areas involved in hearing, due to peripheral disease, might cause a specific vulnerability which results in the generation of this abnormal sensation following a central insult. Appropriately, Wengel et al entitled their manuscript “musical hallucinations, the sounds of silence?”, as they occur when the mind is chronically deprived from music and sound.5

Inhibition of motor unit discharge in humans evoked by transcranial stimulation

The inhibition was revealed in our studies during a period of voluntary contraction by stimulating the motor cortex at a strength lower than that required to produce excitation under the same conditions. A recent study has reported that the discharge of motor neurons in the first dorsal interosseus muscle of the hand of a patient with multiple sclerosis could be suppressed by transcranial magnetic stimulation of the motor cortex, but this was not observed in normal subjects.6 In our previous studies7 8 we averaged the rectified surface electromyogram (EMG) to reveal inhibition of voluntary contraction in a number of different arm and hand muscles. We have now re-investigated one of our subjects to examine the effect of transcranial magnetic stimulation on the probability of discharge of single motor units in the first dorsal interosseus muscle. We can confirm that transcranial magnetic stimulation at a strength which causes a reduction in gross surface EMG, and is sub-threshold for excitation, does lower the probability of discharge of individual motor units in normal humans.

The subject was a right handed male (age 49 years) with no history of neurological illness. Local ethical approval was obtained and the subject gave his informed consent to the procedures. Two forms of electromyographic recordings were made from the first dorsal interosseus muscle of the right hand. EMG, surface electrodes were placed over the belly of the muscle and at an indifferent point over the proximal interphalangeal joint of the first digit. A concentric needle electromyogram was inserted percutaneously into the first dorsal interosseus muscle to record the discharges of single motor unit. The subject was required to make a weak voluntary contraction of the muscle. Auditory feedback of the signal was provided to enable the subject to recruit and maintain the discharges of a motor unit that could be reliably identified and selected for peri-stimulus time histogram analysis. Transcranial magnetic stimulation was delivered from a Novametrix 200 stimulator using a 9 cm round coil centered over the vertex. The initial direction of current flow in the coil was antiperiodic and adjacent somatosensory stimuli preferentially excited muscles on the right side.

The threshold transcranial magnetic stimulation required to produce an initial excitatory response, gauged from the surface EMG recording, was 40% of maximum output. The response had a latency of 23 ms and was followed 5–6 ms later by a period of suppressed EMG lasting 30 ms and culminating in a late period of increased EMG activity. Part A of the figure shows the average of the full-wave rectified surface EMG response to 50 magnetic stimuli at 77% of output. Recovery of stimulation no initial excitation occurs but suppression of EMG is evident with a latency of 29 ms and duration 26 ms. The peri-stimulus time histogram in part B of the figure is constructed, gauged from the surface EMG recording of a single motor unit (average frequency 9-1 impulses/s) during 100 magnetic stimuli at 37% of maximal output. A profound depression of firing occurred during the period of 26 ms and with a latency of 29 ms. Within the period of suppressed firing the unit discharges on only 8 occasions during the 100 trials. The number of discharges per the period of the peri-stimulus time histogram in the absence of stimulation was


MRS in a case of rigid Huntington's disease

In a recent paper Savioardi et al report MRS findings of 18 patients with Huntington's disease (HD), seven of whom had the rigid variant. They found increased signal intensities of the caudate and putamen in all the rigid patients in intermediate weighted images and in all but one in T2 weighted images. All but one hypokinetic patient had no signal changes on MRI. Sethi et al also report putaminal hyperintensities in single cases of rigid HD. In contrast, Rutledge et al reported hyperintensities of the caudate and putamen in four patients. Since authors either did not report on intensity changes or explicitly did not find any. We report a case of rigid HD with a pattern of MRS findings, which has not been described previously.

At the age of 18 our patient developed clumsiness of her hands. There was a rapid progression with development of gait disturbances, dysarthria, loss of spontaneous movements, urinary dysfunction and hyper-salivation. When referred to our hospital at the age of 26 she had orofacial apraxia, aphonia, spastic gait, severe bradykinesia with bamboo pole-like posture, rigidity of all extremities, dystonia of the upper extremities with hyperextension of the fingers, abducted arms and flexed elbows. Tendon reflexes were exaggerated with bilateral Babinski signs. Blood smears were negative for acanthocytes. Four other members of the family were also affected by either choreatic or rigid movement disorders. Pathological, investigations were not carried out on any of these cases. Mode of inheritance seemed to be autosomal dominant. Our diagnosis was rigid HD.

MRI (1.5 Tesla) in our patient showed bilateral caudate atrophy (figure). Additionally, on T2-weighted images (SE 2:190) there was mild hypointensity of the putamen and the globus pallidus, which did not involve the caudate. The same constellation was found in one of the 33 patients, who was hyperkinetic and the only clinically affected family member that we were able to study. Our findings do not agree with Savioardi's study in three ways: 1) instead of hyperintensities we found hypointensity in the basal ganglia; 2) in our case the puta-
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P H Ellaway, N J Davey and D W Maskill

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