Transient hallucinations. Paquier et al reported a patient who, following subarachnoid haemorrhage, developed musical hallucinations. Based on a literature review, they suggested that musical hallucinations, 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 hemisphere, as previously proposed. 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 stenosis of the external auditory meatus. Her past history revealed no insulin dependent diabetes mellitus, ischaemic heart disease, peripheral vascular disease and paroxysmal atrial fibrillation. In September 1992, she 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 music began suddenly, was very 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; the volume was mostly high, especially during the night, disturbing her sleep, and severely interfering with her daily activities. Amitryptiline 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. 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. It is possible that musical hallucinations result from a “defaerentation” 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. 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.

Inhibition of motor unit discharge in humans evoked by transcranial stimulation

Transcranial magnetic stimulation of the motor cortex can elicit contraction of contralateral muscles but, until recently, there had been no reports that transcranial magnetic stimulation could suppress muscle contraction. We have shown that inhibition of voluntary contraction can be elicited by transcranial magnetic stimulation and have presented evidence that the mechanism is likely to be superspinal, presumably involving inhibition of motor neurons with consequent disfacilitation of motor neurons. 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 disfacilitation of motor neurons in the first dorsal interosseous 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. In our previous studies, 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 interosseous 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. A local ethical committee had been consulted and the subject gave his informed consent to the procedure. Two forms of electromyographic recordings were made from the first dorsal interosseus muscle: the first was the gross 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 electrode was inserted percutaneously into the first dorsal interosseous 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 such that the magnetic field was directed towards the scalp and induced weakly exciting magnetic field. The magnetic field originated from an external source, and was stronger when the coil was oriented such that the magnetic field induced by the coil was directed away from the scalp. The magnetic field induced by the coil was directed towards the scalp and induced weakly exciting magnetic field. The magnetic field originated from an external source, and was stronger when the coil was oriented such that the magnetic field induced by the coil was directed away from the scalp.

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 5–8 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 maximum output of stimulation. The period of suppression was followed by a period of increased EMG activity. Part B of the figure shows the difference of the preceding period of suppression and the subsequent period of increased EMG activity. The difference is most pronounced at 26 ms and 29 ms.

The threshold of the peri-stimulus time histogram in the absence of stimulation was