been implicated in the past, but symptoms appeared approximately four weeks after the last dose of gentamicin. It is possible that one or more of the previously administered cymycines may have been responsible, but this has not been reported before, and the last course of treatment was eight weeks before the onset of her neurological symptoms. Furthermore, the possibility is that our patient developed OM as a direct manifestation of Hodgkin's disease, but at the time of development of OM the extent of her disease had considerably reduced following high dose chemotherapy. Furthermore, she had had clinical and radiological evidence of regression of her disease. Normal CT and MRI scans of her brain excludes a diagnosis of cerebral Hodgkin's disease. A metabolic abnormality resulting from destruction of Hodgkin's disease tissue is also unlikely, although it has been previously postulated that production of neutotoxic amines and/or peptides might be responsible. This report, however, failed to show any correlation between the presence or severity of OM and the routinely available biochemistry or serum catecholamine levels.\footnote{1} The discovery of antineuronal antibodies in patients with other paraneoplastic syndromes has suggested to the diagnosis that these diseases may have an autoimmune aetiology. Support for an autoimmune basis to paraneoplastic OM has come from reports of a specific antineuronal autoantibody (anti-Ri) in the serum of patients with opsoclonus and breast carcinoma.\footnote{2} The presence of the Ri antigen in tumour tissue of these patients suggests that it is the body's immune response to a tumour antigen that elicits the antibody response.\footnote{3} There was no evidence of an autoimmune serological data to support such a response in our patient, it is possible that breakdown of lymphomatous tissue resulting from effective chemotherapy led to the production of antineuronal antibodies and the subsequent development of OM.

Treatment for opsoclonus from whatever cause remains uncertain. While some authors recommend ACTH or steroids,\footnote{4} this is by no means proven benefit. At very least, it is imperative to treat the underlying condition.

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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 halluci-
nosis, formed auditory perceptions that occurred 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 lesions of the thalamus, 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 stenocerebrovascular disease. 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 exter-

nal 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 tine. The music was slowly, warm, slow, 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, dis-
turbing her sleep, and severely interfering with her daily activities. Amitriptyline parti-
cially 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.1 Our patient illustrates the fact that dominant hemispheric stroke can also result in musical hallucinations.

As with many 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 com-
mon in elderly, hearing impaired, female patients.1 It is possible that in our patient, musical hal-
 lucinations represent a “deselection” phenomenon, reminiscent of visual halluci-
nations in the blind, thalamic pains or phantom limb. It appears that both central and end organ pathology contribute to the appearance of musical hallucinations.1 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”,2 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 mag-
netic stimulation could suppress muscle contraction. We have choosen that inhibition of voluntary contraction can be elicited by transcranial magnetic stimulation and have presented evidence3 that the mechanism is likely to be supraspinal, presumably involving inhibitory interneurons 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 excita-
tion under the same conditions. A recent study has shown that the discharge of motor neurons in the first dorsal interosseus muscle of the hand of a patient with multi-
ple sclerosis could be suppressed by trans-
cranial magnetic stimulation of the motor cortex, but this was not observed in normal subjects. In our previous studies1 we aver-
aged the rectified surface electromyogram (EMG) to reveal inhibition of voluntary con-
traction in a number of different arm and hand muscles. We have now re-investi-
gated 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 indi-
vidual motor units in normal humans.

The subject was a right handed male (age 49 years) with no history of neurological ill-
ness. Local ethical clearance 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. The subjects' EMG, surface electrodes were placed over the belly of the muscle and at an indif-
ferent point over the proximal interpha-
langeal joint of the first digit. A concentric needle electrode was inserted percutaneous-
ously 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 anterior to and adjacent to the stimuli preferentially excited muscles on the right side.

The threshold transcranial magnetic stimulation required to produce an initial excitation 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 stim-
ulation pulses.77% of output. The percentage 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 par B of the figure is constructed, gauged from the sur-
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Very late onset X-linked recessive bulbospinal neuronopathy: mild clinical features and a mild increase in the size of tandem CAG repeat in androgen receptor gene.

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