marked reduction of muscle permeability to potassium.[1] Effects of lithium on potassium metabolism have been the topic of in vivo and in vitro studies. Results are contradictory, depending on the study design and the patient's psychiatric state. No noticeable and consistent systematic effect of lithium on body potassium has been reported. Nevertheless, lithium could enhance Na-K pump activity, similar to potassium.[2]

Lithium therapy has already been proposed in various forms of familial periodic paralysis with varying results.[3,4] To our knowledge, there is only one other report concerning lithium therapy in a patient with FHPP.[5] In this case, carbonate lithium was administered to reach serum lithium levels up to 1·0 mmol/l. No benefit was observed, notably on attack frequency which remained about one per week. Biochemical homogeneity of FHPP may be questioned on the basis of such discrepant results. Some forms could be lithium sensitive and others, lithium resistant. Further studies are clearly needed to elucidate this problem. Lithium, as an oral potassium add-on therapy, is worth trying in FHPP cases resistant to standard therapies. It is safe and can be beneficial on rate of attack.

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Hyperphagia in dementia: fluvoxamine takes the biscuit

Marked overeating has been described in a number of conditions which involve brain damage.[1,2] Such overeating can cause management difficulties, but there have not been any reports of effective drug treatment for this problem. We describe the case of a man with probable Pick's disease whose marked hyperphagia appears to have been reduced by fluvoxamine.

A 69 year old man presented with a four year history of personality change and difficulty in planning tasks. All his personal interactions became bland and his persistent mood was one of fatuous bonhomie. In addition he became incapable of carrying on his work as a builder. At this stage he scored 29/30 on the Mini Mental State Examination,[3] but repeated examination over the next three years showed clear and increasing impairment in sequencing, categorising and problem-solving tasks. A diagnosis of Pick's disease was made on the basis of the history, neurological examination, the neuropsychological tests and SPECT imaging. His mother had died aged 54 years apparently confused and unable to walk. No further details of her clinical state are known.

Two years ago he began to eat large amounts, taking food inappropriately from his son's plate, from supermarket shelves, and continually searching the house for more food. He was admitted to a residential home but his persistent attempts to obtain food led to admission to a psychogeriatric ward. On the ward he ate all food put in front of him, he took food from other patients and he raided the larder.

To see how much he would eat if given a limitless supply we observed the patient in a standard setting. On a table there were five plates containing a variety of biscuits (40 biscuits in all), a large pot of tea and four magazines. The observations were made from 9–10 am after an overnight fast. Mr C was invited to help himself to whatever he wanted. He was observed through a window from the adjacent room. The stock of biscuits was replenished if required. Observations were made approximately weekly.

On the hypothesis that the marked hyperphagia might be due to reduction in effective 5–HT function he was treated with fluvoxamine (a selective 5-HT uptake blocker) 100 mg/day for four weeks. The medication was tailed off and observations continued for a further 11 weeks.

Three baseline observations were made in the standard setting before starting fluvoxamine. These showed that he ate at a constant rate throughout the hour consuming a total of 60 or 61 biscuits (about 3500 kilocalories) on each occasion.

Within one week of starting the fluvoxamine the nursing staff reported a clear improvement in his behaviour. This improvement was confirmed by the standardised observations. Eleven days after starting fluvoxamine he ate 19 biscuits in the first 30 minutes and then looked through one of the magazines for the remainder of the hour. During this treatment phase we carried out five observations. The median number of biscuits eaten per hour was 21 (range: 15–40). The patient did not experience nausea whilst taking fluvoxamine.

On stopping the fluvoxamine there was considerable fluctuation in the number of biscuits eaten, but he did not return to the behaviour observed before treatment. Ward staff reported that, after stopping fluvoxamine, his behaviour worsened, but that it was considerably less of a problem than it had been before treatment. We carried out a further 12 observations during this period. The median number of biscuits eaten per hour was 18 (range: 7–47).

Animal studies have implicated the 5-HT system as crucial in the satiety mechanism.[4,5] Fluvoxamine is a selective 5-HT uptake blocker. However, its effectiveness in this case does not prove that the primary defect lies in the 5-HT system. Indeed, it implies that there is sufficient intrinsic 5-HT on which the uptake blocker can work. On discontinuing the fluvoxamine the patient's behaviour did not return to the pre-treatment levels. One possible explanation is that the apparent effect of fluvoxamine was purely coincidental. However, the marked change in long-standing behaviour on starting treatment would argue against this. A second explanation is that whilst he was on treatment there was sufficient progression of the disease to cause a change in his eating behaviour. A third explanation is that the fluvoxamine caused long-lasting effects on brain function. Whatever the mechanism, fluvoxamine appears to have had an effect on his hyperphagia which was measurable and clinically important.

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My Music—a case of musical reminiscence diagnosed courtesy of the BBC

Musical reminiscence is a disorder characterised by formed auditory hallucinations of a musical nature. This case is unusual in that the patient made the diagnosis and was subjected to NMR and SPECT studies.

On Christmas Eve 1985, an active 73 year old widow retired to bed in a particularly distressed state. She had just learned that her son and daughter-in-law were about to separate. On Christmas Day she was surprised to find her "elderly" neighbours playing Christmas tunes loudly on what she presumed to be a new music centre.

She was reluctant to complain at first as she felt her old neighbours "had so few pleasures left to them". After a few days the continuous and repetitive tunes became so irksome that she asked her home-help to make discrete enquiries. She was dismayed to learn that her neighbours had not bought a new music centre. Sometime later her son visited and she described to him the sounds she was hearing. He realised that there was no basis for his mother's complaints and initiated a series of medical referrals through the family doctor. The ENT surgeons prescribed a tinnitus

mask but the sounds persisted. Neurological and neurosurgical opinion was sought. Thioridazine and temazepam were prescribed. The benzodiazepine helped to modify the sound slightly and lessen its intensity. However, the musical sounds continued to plague her waking life and the "hallucinations" triggered a psychiatric referral.

At the first psychiatric interview, the patient referred to the book "The Man who mistook his wife for a hat" by Oliver Sacks.[1] She mentioned that her son had heard a description of this book on the radio including several examples of patients described therein. One of these was the case of a medical oddity which struck him as having similar features to those of his mother. They acquired the book and the patient recognised that her problem was similar to the "musical reminiscence" (see[1], chapter 15).

In the five years since the onset of her symptoms, the musical sounds heard by the patient have not ceased during her waking hours apart from a brief period of a few hours several months after the onset. At the time of her psychiatric referral, the sounds were phrases from folk songs and non verbal sounds, for example "Highland Laddie ta ra ra, Bonnie Laddie". Subsequently the words became less distinct and more of a tuneful humming. Much to her annoyance, the sounds she experienced were often slightly out of tune. Shortly after the start of her illness, she gained full insight into her problem and from that point recognised that the tunes were from within her mind rather than originating in external space. She was not found to have any features of psychiatric illness. During her psychiatric contact she hinted at suicide if no resolution of her problem could be found but at no time did she appear to be suffering from a depressive illness.

Pharmacological treatments with haloperidol, thioridazine, benzodiazepines, carbamazepine and sodium valproate were tried but without any success.

A psychometric assessment concluded that she was above average intelligence and that there were "no pointers to suggest the right hemisphere dysfunction which has been inferred in some published cases of this disorder". In 1988 a further EEG showed intermittent irregular sharp and slow wave complexes in anterior-mid temporal regions, maximal on the right side though frequently bilateral.

An MRI scan in October 1988, showed multiple small areas of abnormal signal scattered throughout both hemispheres. A SPECT scan in February 1989 was normal.

The unusual symptom of formed auditory hallucinations of a musical nature are rare in the medical literature, with only three dozen cases fully reported[2] and none giving details of NMR or SPECT studies. There was a longstanding notion that hallucinations are to be equated with schizophrenia but this is clearly unfounded and hallucinations are never pathognomonic of any given disorder.[3]

In most of the reported cases, there are certain common features. These are amplified in a definitive review.[2] The patients are generally elderly and often have a history of progressive deafness over the previous decade. The deafness may be a contributory factor as external noise would be diminished allowing for an emphasis on the internally generated hallucinatory sounds. As in the case of our patient, Hammeke *et al*[4] described how the auditory hallucinations of their two patients were affected by ambient noise levels and the content and speed of the hallucinations were influenced by attentional and intentional factors. They postulated that a combination of peripheral and associated central "disinhibition" may be responsible for the occurrence of such hallucinations.

The prognosis is largely unknown. In many cases the symptoms last until death. On the whole, patients are able to adjust to the symptoms and find their own individual methods of shutting out the noise, for example, turning up the radio, trying to ignore it mentally or occupying themselves with a range of activities. Benzodiazepines seem to be useful in alleviating the insomnia. Our patient (and many of the reported cases) gained immense relief when they were assured there was no evidence of psychiatric illness and they were not "going mad" as they had feared.

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Pituitary hormones in relation to magnetic stimulation of the brain

There has been considerable interest in the technique of stimulating the central motor pathways with a brief high intensity magnetic field.[1] This form of transcranial stimulation causes less discomfort than the electrical technique described previously, making it feasible to study function in central motor pathways in children. Although no adverse effects have been reported in adults,[2] it is essential to collect further information about possible effects on cerebral function before this technique can be used routinely on children. A previous study of transcranial electrical stimulation in adults did not show any EEG changes or rise in prolactin, but the numbers of stimuli were limited by the discomfort of stimulation.[3] The reduction in discomfort related to magnetic stimulation might well lead to large numbers of stimuli being used. It was therefore decided to carry out a study in normal adults to measure any possible effects of transcranial magnetic stimulation (at clinically relevant levels of testing) on plasma concentrations of pituitary hormones and EEG.

Transcranial magnetic stimulation was carried out on 17 healthy adult volunteers aged 21 to 66 years (6 males and 11 females). The subjects were not receiving any medication, and informed consent was obtained in all cases. The study was approved by the Ethical Committee.

The subjects were fasted overnight and an indwelling venous catheter was inserted at least 15 minutes before stimulation. Two blood samples were taken before stimulation (−15 minutes and time zero). Seven further samples were taken at 10, 20, 30, 45, 60, 75 and 90 minutes after the beginning of magnetic stimulation. In 11 subjects (all females aged 21 to 58 years) a control investigation using a dummy coil was carried out on a different day, the order of testing being randomised. The dummy coil was held over the subject's head while the active coil was discharged, with its characteristic "crack", away from and out of sight of the subject. Blood sampling was repeated as described above, taking care to carry out measurements in each individual at the same time of day to minimise the effects of diurnal variation.

Growth hormone (GH), prolactin (PL), and cortisol were measured in all samples. Luteinising hormone (LH), follicle stimulating hormone (FSH) and thyroid stimulating hormone (TSH) were measured in the basal sample and at 30, 60 and 90 minutes. Standard radioimmunoassay procedures were used for all hormone assays. Coefficient of variation for the assays was between 3–5% for all hormone studies.

EEGs were taken in six subjects, using an identical recording protocol to that described in the previous study of electrical stimulation.[3] The EEG recordings were repeated one to three weeks after stimulation. The EEGs of four subjects who had also taken part in the earlier study were compared with their previous recordings.

A Digitimer D190 magnetic stimulator was used with a 6-turn coil, 10 cm in diameter, positioned with its centre over the vertex. The intensity of stimulation was adjusted until an obvious jerk of the arms was seen. The number of stimuli varied between 10 and 50 (most subjects receiving 30) with an interstimulus interval of around five seconds. No attenuation was made to determine precise thresholds for cortical excitation.

The responses of GH, PL and cortisol in relation to magnetic stimulation and the control period are shown in the figure. There were increments in GH in five out of 17 subjects following stimulation and in three out of 11 subjects during the control period. These subjects were all females aged between 26 and 58 years. In all except one, the GH concentration had begun to rise before the onset of magnetic stimulation. In this subject, GH started to rise 30 minutes after stimulation and reached a peak at 60 minutes. In two subjects who showed GH changes there were similar patterns of change in PL and cortisol. In all male subjects the GH levels remained $< 0{\cdot}5$ mU/l throughout the test and control periods.

Cortisol and PL responses in all subjects were very similar on both days. No significant changes in LH, FSH or TSH concentrations were seen in any subject.

All EEG records taken before stimulation were within normal limits. No change in EEG activities was seen immediately after stimulation or in recordings one to three weeks later. There were no differences between EEGs taken during the present and previous study[3] in the four subjects participating in both procedures.

Changes have been reported in several pituitary hormones in relation to electroconvulsive therapy and seizures.[4,5] It has been suggested that increments in PL, GH, TSH and gonadotrophins are specific, and PL in particular has been widely used in the differential diagnosis of seizures. In this study,