

has spent a major part of his life in hospital, so the psychological closeness of relationship may not be the same as the biological closeness may suggest. Furthermore, underdeveloped cognitive abilities may cause delayed realisation of loss. The mentally handicapped do react adversely to stressful changes, e.g. change of wards or staff. This report of psychiatric illness following a major life event highlights the significance of bereavement and grief work with the mentally handicapped.

IQBAL SINGH  
SAYED HASAN JAWED  
SARAH WILSON

*Leavesden Hospital  
College Road, Abbots Langley  
Watford, Herts WD5 0NU*

#### References

- AMBELAS, A. (1979) Psychologically stressful events in the precipitation of manic episodes. *British Journal of Psychiatry*, **135**, 15–21.
- (1987) Life events and mania: a special relationship? *British Journal of Psychiatry*, **150**, 235–240.
- McLOUGHLIN, I. J. & BHATE, M. S. (1987) A case of affective psychosis following bereavement in a mentally handicapped woman. *British Journal of Psychiatry*, **151**, 552–554.
- ROSEMAN, S. J. & TAYLOR, H. (1986) Mania following bereavement: a case report. *British Journal of Psychiatry*, **148**, 468–470.

#### Psychiatric Munchausen's Syndrome: A College Register?

**SIR:** We wish to report an unusual psychiatric variant of Munchausen's syndrome and to propose the formation of a College Munchausen's register.

*Case report:* A 23-year-old woman arrived by taxi at the A & E department of a provincial teaching hospital. She was mute, but wrote that she was being pursued by aliens, whom she could see and hear, and who were telling her to kill herself.

It was decided to admit her informally to a psychiatric ward. During the admission procedure she claimed to be of no fixed abode, writing the abbreviation NFA in a defiant fashion. She gave no addresses of family and friends, but provided a list of contacts with people and establishments, for example a battered wives refuge, all of which proved to be fictitious. Immediately after admission she bought chocolates for the ward staff. She also bought soft toys and children's books from the hospital shop and sat playing with them. She then demanded in writing that the police be called because she had been raped prior to admission.

After six days of silence, and following a visit from the police, she began to talk, denying that rape had occurred. She said that she had been in many hospitals, travelling throughout the country using a railcard. She explained that she lived with a much older man who had befriended her on one of her train journeys. He was contacted and finally

arrived to collect her. He confirmed her story, saying that she had been living with him for a year and that she frequently disappeared from his home leaving him to wait for a telephone call from yet another hospital. He listed 25 hospitals at which she had presented. She had been admitted to both medical and psychiatric wards and had at times been detained under section 2 of the Mental Health Act.

The patient then claimed to have been sexually abused in childhood by her father and to have been abandoned by her parents. Her real wish was "to have parents like everyone else". She also claimed to have trained as a psychiatric nurse but to have failed her examinations. Before her history and mental state could be explored further, the patient and her cohobitee packed her belongings and left to catch the train to their home.

We report this case for two reasons. Firstly, we are unaware of any other descriptions of elective mutism as a central feature of the psychiatric variant of Munchausen's syndrome. We think that this case will be of general interest to those of our colleagues who have not seen our patient in person. Secondly, we wish to propose that, since psychiatric Munchausen's syndrome may be increasing in frequency (Jones & Sternberg, 1985), there is a strong indication for establishing a central register of such cases. The Royal College of Psychiatrists is uniquely placed to administer this and could record patient descriptions, aliases, and previous patterns of presentation. In the case described above we suspected a factitious disorder from early in the patient's admission. If a register had been functioning at the College, then instead of many telephone calls, a single one may have sufficed.

A. MARKANTONAKIS  
A. S. LEE

*University Hospital  
Queen's Medical Centre  
Nottingham NG7 2UH*

#### Reference

- JONES, M. E. & STERNBERG, M. P. (1985) Munchausen's syndrome. *British Journal of Psychiatry*, **147**, 729–730.

#### Migraine, Insomnia and Reactive Depression Due to Brain Serotonin Deficiency?

**SIR:** Within a little over a one-year period, ten female patients (age range 20–40 years) were seen in our outpatient department, complaining of severe migraine, troublesome insomnia, and depressed mood. In six of these subjects, the first symptom to occur was severe migraine, with a frequency of two-three times per week; this was followed several weeks later by troublesome insomnia, and later on by a feeling of

hopelessness and helplessness, accompanied by frequent crying spells. In the other four patients, unbearable insomnia was followed after a few months by frequent severe cephalgia, and later by feelings of hopelessness, worthlessness, and crying spells. At times most of them, due to their migraine, were forced to lie in bed in a dark silent room, as every little noise increased their discomfort. None of them had in the past suffered from depression or anxiety, and they had always been capable of carrying out their usual duties. Prior to their first visit to our department, they had been treated with analgesic or hypnotic drugs or both, with little or no relief.

Amitriptyline (25 mg, t.i.d.) and 5-hydroxytryptophan (100 mg, t.i.d.) was prescribed to all of these patients. The latter was preferred to L-tryptophan, as it crosses the brain/blood barrier more easily (Wurtman & Fernstrom, 1976). Amitriptyline was chosen for its known mild sedative effect. Each patient was checked on a weekly basis for the following three months, and in eight cases a mild improvement of symptoms was reported after four weeks of treatment. This became more marked at the end of the eighth week. Both migraine (or cephalgia) and insomnia had progressively decreased in frequency and intensity without the use of any analgesia or hypnotic drugs, and consequently, the patients' mood had also improved so that they could more easily carry out their usual duties.

At the end of the 12th week of treatment these eight patients claimed to be symptom-free. In the other two patients some amelioration of symptoms occurred, but they still needed to use analgesics or hypnotics from time to time, although less frequently than before.

In those cases where a complete recovery took place, the drug regimen was gradually reduced and then totally discontinued after six months of therapy.

They were followed-up for a while at random, and no relapses were found. The other two cases followed the same treatment for over one year, but no further improvement was reported.

This drug regimen was implemented on the assumption that migraine and insomnia – and the subsequent depressed mood – could be due to a brain serotonin deficiency. The role played by serotonin in the genesis of migraine was first shown by Sicuteri *et al* (1961). Both Jouvét (1969) and Jouvét & Pujorl (1974) demonstrated the influence of serotonin on the sleep cycle, whereas Van Praag (1981) showed the usefulness of 5-hydroxytryptophan for the treatment of a depressed mood.

In all the cases reported, the symptom complex began either as a severe migraine or as troublesome insomnia. The depressed mood which occurred some time later was reactive to the discomfort provoked by the other two aforementioned symptoms, which are very likely due to a brain serotonin deficiency.

LUIGI BUCCI

*Ospedale S. Maria della Pietà  
Piazza S. Maria della Pietà 5  
00135 Rome, Italy*

#### References

- JOUVET, M. (1969) Biogenic amines and state of sleep. *Science*, **163**, 32–34.
- JOUVET, M. & PUJORL, J. P. (1974) Effects of central alterations of serotonergic neurons upon the sleep-waking cycles. In *Advances in Biochemical Psychopharmacology, Vol. 2* (eds E. Costa, G. L. Gessa and M. Sandler). New York: Raven Press.
- SICUTERI, F., TESTI, A. & ANSELMINI, B. (1961) Biochemical investigation in headaches: increased in hydroxy-indol-acetic-acid excretion during migraine attacks. *International Archives of Allergy*, **19**, 55–61.
- VAN PRAAG, H. M. (1981) Management of depression with serotonin precursors. *Biological Psychiatry*, **16**, 291–310.
- WURTMAN, R. J. & FERNSTROM, J. D. (1976) Commentary: control of brain neurotransmitter synthesis by precursors. Availability and nutrition states. *Biochemical Pharmacology*, **25**, 1691–1696.

## A HUNDRED YEARS AGO

### Tea Drinking and Nervous Disorders

Tea has a powerful action on the nervous system of some individuals. Dr Bullard of Boston believes that it may produce a chronic poisoning of the nervous centres, shown in increased excitability, due partly to direct action of the alkaloid on the nervous matter, and also indirectly by the production of gastric derangement. Taken, therefore, too frequently, even in moderate doses, it places the nervous system in a condition of greater vulnerability to slight external influences, and favours the development of functional neuroses, or helps to render them permanent. Whilst there is no evidence to show that tea causes organic changes in the nervous tissues, yet, if such

exist, tea may readily aggravate some of the symptoms. Tea may act as an important factor in the causation of neuralgia, hysteria, and allied affections. When taken constantly in large doses, dyspepsia usually supervenes before irreparable harm is done to the nervous system. In hemiplegia, and possibly some other functional neuroses, there is probably a craving for some stimulant, and tea is better than other equally accessible articles, and so it happens that many sufferers from megrim are great tea-drinkers.

#### Reference

- The Lancet*, 14 January 1988, 86.