are present and the fluctuation of enzyme in psychotic patients without NMS is not investigated.

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Benzodiazepine addiction in heroin addicts

SIR: The recent article by Gossop *et al* (*Journal*, March 1989, **154**, 360–363) on opiate withdrawal symptoms was broadly in keeping with previous work showing that 80% of heroin addicts could be withdrawn as in-patients using methadone, and also that withdrawal symptoms have a large psychological weighting and are therefore more severe around the time of completion of the programme. The 10-day withdrawal regime was, however, associated with a higher drop-out rate soon after, and as they point out, this is likely to be due to variables other than opiate withdrawal.

There has been little work on why 20% of addicts fail to finish their detoxification, and it would prove methodologically difficult to study this adequately. One possible reason that might account for a proportion of the 20% would be concurrent benzodiazepine addiction, and this might also explain some of the cases of higher relapse after the 10-day detoxification.

A study of 298 addicts attending the drug dependence unit at St George's Hospital, London, found benzodiazepines in the urine of nearly 60% (Beary *et al*, 1987), and in a study of 79 addicts in Sheffield, 90% said that they used benzodiazepines, with regular use in about 50% (Perera *et al*, 1987). Rebound anxiety can occur if benzodiazepines have been used for three weeks, and about half of patients who have taken them for three years experience a specific withdrawal syndrome (Noyes *et al*, 1988). If we hypothesise that 30% of benzodiazepine users experience rebound anxiety or a withdrawal state on abrupt discontinuation, and that 50% of addicts take benzodiazepines, then we could expect 15% of heroin addicts to experience some form of withdrawal reaction if detoxified using methadone alone.

The symptoms of rebound anxiety and some of the symptoms of benzodiazepine withdrawal would lead to a score on the Opiate Withdrawal Scale, but the perceptual abnormalities seen in benzodiazepine withdrawal would not. The symptoms, however, would not necessarily respond to methadone. With short-acting benzodiazepines, a withdrawal reaction may be seen within 24 hours. For the more commonly used benzodiazepines, however, they are more likely to present at about 5 days and peak at about 10 days. The combination of this and finishing the methadone withdrawal regime, which is a psychologically difficult time for the addicts, may partly explain the higher drop-out rate in the 10-day withdrawal group than in the 21-day programme in the study of Dr Gossop *et al*.

I feel that this is an aspect of opiate withdrawal that warrants further attention if we are to successfully treat heroin addicts.

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Post-traumatic stress

SIR: McFarland (*Journal*, February 1989, **154**, 221–228) comments that past history of treatment for psychological disorder was a better predictor of post-traumatic morbidity than the degree of the exposure to the disaster or the losses sustained. He suggests that his results raise doubts about the postulated central aetiological role a traumatic event plays in the onset of morbidity.

In my study of burn victims who had been admitted for seven days or more to a burns unit (White, 1981), over a third of 86 patients who were followed-up one year after their accident had marked psychological sequelae. Only three of those patients had a past psychiatric history of a neurotic illness (requiring admission to hospital or two or more psychiatric consultations). The main predictors for a poor psychological outcome after a year were the severity of the injury and the length of stay in hospital. The presence of anxiety, depression, or a personality problem at the time of the initial interview (within seven days of the accident) was also related to an increased incidence of psychological sequelae. Other factors found to be important were age, social class, and whether patients lived on their own or had large

families. There was also no clear relationship between the area of injury and the frequency or severity of psychological after-effects.

Thus, although I would accept that premorbid vulnerability may indeed be an important predictor of psychological outcome following a frightening event, the overwhelming majority of victims in my study who developed post-traumatic stress did not have a past psychiatric history.

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Listeria and psychiatric syndromes

SIR: In view of the recent publicity given to the almost forgotten listeria monocytogenes, it might interest psychiatrists to know that outwith the British Isles this bacterium has been incriminated as the causative agent in a wide variety of psychiatric syndromes. Although abortion most readily springs to mind in association with listeria, studies have shown that the syndrome most commonly occurring in man is the meningeal encephalitic form (Seeliger, 1958; Colmant, 1961). As would be expected, psychiatric syndromes are described in cases where the condition has become subacute and chronic.

In the German literature, Lang (1955*a*) described five children who had raised listeria titres in the course of recurrent bouts of disturbed behaviour with screaming and temper tantrums, all of which disappeared after adequate treatment with antibiotics. Lang (1955*b*) also claimed to have found a high incidence of raised listeria titres among a group of 300 children from the Bonn area suffering from different types of brain damage. Seeliger (1958) described a patient who in the course of a recognised listeriosis infection developed a manic illness which lapsed into a chronic depressive state and eventually led to suicide. In the Russian literature, Timofeyeva *et al* (1953) described three cases presenting with predominantly schizophrenic symptoms. Recurrent bouts of temperature together with a raised listeria titre strongly suggested that listeria was responsible for the psychiatric symptoms.

So far listeria seems to have made little "headway" in Britain, and hopefully this will continue to be the case. However, Colmant (1961) warned that although listeriosis is an illness resembling tuberculosis and syphilis in its complexity, its diagnosis depends on the familiarity of the doctor with the illness. Listeria fell from grace and general interest after it was found not to be the cause of infectious mononucleosis in man, following its original discovery in 1924 in Cambridge by Murray *et al* as a monocytosis producing bacteria in laboratory rabbits. However, given its known predeliction for soft cheeses, perhaps it now merits shortlisting as a possible causative agent in psychotic gourmets.

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The Melbourne Maudsleys

SIR: Readers of Henry Maudsley's autobiography in the *Journal* (December 1988, **153**, 736–740) may be interested in some further family history. Maudsley paid a visit to Australia in 1903, apparently to see "the best of cricket in its best home". I wonder, however, whether the visit offered an opportunity to meet his nephew, also Henry Maudsley (1859–1944) and his great-nephew, Henry Fitzgerald Maudsley (1891–1962). Both Australian members of the Maudsley family were prominent physicians, with the younger Henry being a key figure in the development of Australian psychiatry.

Henry Maudsley (1859–1944), like his uncle, was born in Yorkshire, near Settle. He obtained his MB BS at the University of London in 1881. Again like his uncle, some 30 years previously, he was a medical officer at University College Hospital. He migrated to Australia and settled in Melbourne in 1888. In 1903 he joined the staff of the Melbourne Hospital and was Lecturer in Medicine in the University of Melbourne. He became a leading consultant physician and, unlike his uncle, was knighted (Obituary, 1944).

A short biography of his son, Henry ('Hal') Fitzgerald Maudsley, has recently been published