The treatment aspects, which include inadequate and inappropriate treatment, do not appear to be given any weighting over and above the other factors. Yet treatment is the only aspect that can be decisively influenced by the clinician. He cannot alter the symptom profile, the past history nor the pre-morbid personality. But he certainly can treat adequately or inadequately and Dr Scott herself gives many references reporting examples of substandard treatment, subtherapeutic doses of antidepressants and even no treatment at all. Her observation that, "it is noteworthy that the introduction of a wide variety of new treatments has not significantly altered the prevalence of the disorder" may be true, but introducing new treatments does not ensure that they are used effectively.

Therefore, a span of two years before depression can be considered as chronic seems entirely arbitrary and without clinical meaning unless it is linked to the use of some defined and potent forms of therapy. The opinion that, "with regard to depression being treatment-resistant, the definition of the term is problematical as there is little agreement on the ... treatments that should be used" simply evades a fundamentally important issue. For example, we now regard it as essential for a combination of tryptophan, clomipramine, and lithium (Hale *et al*, 1987) to be tried unsuccessfully before a patient with an affective disorder is considered for psychosurgery.

A textbook of psychiatry (Gelder *et al*, 1983) observes of psychosurgery that "the operation should never be carried out until the effects of at least a year of vigorous in-patient treatment has been observed". I would agree if this meant that one year of intractable illness is usually the minimum in practice before psychosurgery should be seriously considered. But it could mean that, regardless of the clinical circumstances, the patient must wait for at least a year. Similarly, another textbook (Kendell & Zeally, 1988) takes this unit to task for stating that we would, if it were clinically indicated, assess a patient for psychosurgery less than one year after the onset of the illness and this, the authors observe, seems unjustified. Treatment-resistant depressions of that duration are not uncommon and spontaneous remission does occur". This has the rigidity that I am concerned about. The authors are likely to find it more comfortable to wait a full year than some severely depressed patients would. Surely the patient can be allowed to decide when distressing symptoms are quite intolerable, especially when the doctor has been unable to prove relief. Linking treatments to arbitrary periods of time is an autocratic and not particularly logical way of managing psychiatric illnesses.

For psychosurgery to be appropriate, the essential needs are for an appropriate and truly intractable illness (intractability will take time to be sure of, it is true) and a really desperate and incapacitated patient. But the time factor alone is not at all the issue. I believe that the shortest illness from onset to surgery that we have treated was about 10 months. During this period, earlier responses to antidepressant medication and ECT were soon lost. Suicidal drive and weight loss due to depressive anorexia became major and increasing problems. Why should we have waited a further therapeutically sterile two months, even although remission *might* have occurred?

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References

GELDER, M., GATH, D. & MAYOU, R. (1983) *Oxford Textbook of Psychiatry*. Oxford: Oxford University Press.

HALE, A. S., PROCTER, A. & BRIDGES, P. K. (1987) Clomipramine, tryptophan and lithium in combination for resistant depression: seven case studies. *British Journal of Psychiatry*, **151**, 213–217.

KENDELL, R. E. & ZEALLEY, A. K. (1988) *Companion to Psychiatric Studies* (4th edn). Edinburgh: Churchill Livingstone.

CPK levels and neuroleptic malignant syndrome

SIR: I read with great interest the two cases presented by Goldwasser *et al* (*Journal*, January 1989, **154**, 102–104). Their correlation of creatinine phosphokinase (CPK) profiles with clinical state and drug treatment is convincing – perhaps too convincing. Their findings certainly support the idea that elevated CPK values are a major sign of NMS and should be considered as 'classic'. However, moderate increase of MM isoenzyme fractions of this enzyme is also found in muscle injury, physical exertion, muscle cramping, intramuscular injections, and acute psychotic episodes (Zilva & Pannall, 1981).

The cases presented by the authors are young black males with features of rigidity, acute psychosis, and severe aggression, requiring forceful restraint and intramuscular injections. In spite of their impressive demonstration of "CPK steadily falling toward normal – even with the use of intramuscular medications and restraints" (case 1), is it not necessary to correct for these and other factors that contribute to rising CPK levels? Or are they not significant here? What are the levels and mode of degradation of CPK in black, aggressive psychotic patients needing restraint, but without NMS?

It seems difficult to accept the elevation of CPK as a 'classic sign' of NMS when so many other variables

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

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Reference

ZILVA, J. F. & PANNALL, P. R. (1981) *Clinical Chemistry in Diagnosis and Treatment*. London: Lloyd-Luke.

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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References

BEARY, M. D., CHRISTOFIDES, J., GHODSE, A. H. *et al* (1987) The benzodiazepines as substances of abuse. *The Practitioner*, **231**, 19–20.

NOYES, R., GARVEY, M. J., COOK, B. L. *et al* (1988) Benzodiazepine withdrawal: a review of the evidence. *Journal of Clinical Psychiatry*, **49**, 382–389.

PERERA, K. M. H., TULLEY, M. & JENNER, F. A. (1987) The use of benzodiazepines among drug addicts. *British Journal of Addiction*, **82**, 511–515.

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