

Correspondence

MANAGEMENT OF SUICIDE RISK

DEAR SIR,

May I add my voice to that of Dr Goldney (*Journal*, September 1980, 137, 303) in urging that we psychiatrists should not abrogate our clinical responsibility for the assessment and management of parasuicide patients. The current fashion of transferring more and more clinical tasks to social workers, community nurses and even, in the case of parasuicide patients, to overworked physicians is to be deplored.

At present, there is conflicting evidence concerning the efficacy of psychiatric intervention; consequently, it would be foolish and possibly dangerous to ignore those investigations in which psychiatric intervention has been associated with a significant reduction in subsequent self-poisoning behaviour (Greer and Bagley, 1971; Kennedy, 1972; Montgomery *et al*, 1979). More research is needed. What is also needed is a willingness on the part of psychiatrists to continue to accept responsibility for diagnosis and management of parasuicide patients.

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References

- GREER, S. & BAGLEY, C. (1971) Effect of psychiatric intervention on attempted suicide: a controlled study. *British Medical Journal*, *i*, 310–12.
- KENNEDY, P. (1972) Efficacy of a Regional Poisoning Treatment Centre in preventing further suicidal behaviour. *British Medical Journal*, *iv*, 255–7.
- MONTGOMERY, S. A., MONTGOMERY, D. B., RANI, S. J., ROY, D. H., SHAW, P. G. & MCCAULEY, R. (1979) Maintenance therapy in repeat suicidal behaviour: a placebo controlled trial. *Proceedings 10th International Congress for Suicide Prevention and Crisis Intervention*, 227–9, Ottawa, Canada.

DEAR SIR,

Dr R. D. Goldney (*Journal*, September 1980, 137, 303) is concerned at my view that it is rightly so that the psychiatrist cannot be regarded as the most expert in management of the suicidal. I am happy to explain my position further.

I accept that the psychiatrist must take ultimate clinical responsibility for any such clinical problems

under his care. He is the most expert at correlating all aspects of the situation and in deciding on executive action: only he has the necessary breadth of knowledge through his training concerning physical, psychological, social, and behavioural problems, and of course adequate assessment must be based on a synthesis of all these.

Nevertheless, this does not mean that he has an exclusive expertise in clinical management of the suicidal, to which members of other disciplines may contribute their own distinctive skills. A nurse may be best at assessing general behaviour in a ward situation, and a general practitioner or social worker may be more insightful into relationship and social problems. With regard to psychopathology, I have known some highly skilled Samaritan volunteers who seemed to be as expert as anyone else (perhaps more so) at making contact with the suicidal and helping them in their despair. The crucial point is that clinical management must concern the patient as a whole, and this total synthesis is the essence of the psychiatrist's ultimate clinical responsibility. He may exercise this either by direct contact with his patients or in providing consultative advice for other workers.

My original comment was designed to open up rather than close debate on the precise nature and extent of the psychiatrist's expertise and his relationship with others, whether professionals or not, who also provide mental health care. I believe that the psychiatrist's role will ultimately be strengthened if he is concerned with shared rather than exclusive expertise. Management of the suicidal is, of course, a paradigm of a situation in which such debate is crucial.

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THE ISOENZYMES OF CREATINE PHOSPHOKINASE IN ACUTE PSYCHOTIC STATES

DEAR SIR,

Numerous investigations have found elevated creatine phosphokinase (CPK) activity in the serum of

patients with acute psychotic states. These studies have been extensively reviewed by Meltzer (1976). The source of this increase is thought to be skeletal muscle since it is the skeletal muscle isoenzyme that is found in these patients. However, recently some uncertainty has arisen owing to the finding that the brain-type isoenzyme of CPK is highly unstable (Nealon and Henderson, 1975; Cho and Meltzer, 1979).

We have measured the CPK isoenzyme patterns of three groups of psychiatric patients using a technique thought to preserve brain-type CPK isoenzyme activity (Cho and Meltzer, 1979). The blood samples were promptly brought to 4°C and were transported to the laboratory in ice. Most assays were performed immediately, but if this was impossible samples were deep frozen for a few days at most. Total CPK activity was determined by the method of Rosalki (1967) and CPK isoenzymes were separated and measured by a sensitive fluorescent technique based on the method of Somer and Konttinen (1972).

The subjects were 30 female inpatients. All had been admitted to hospital within the previous two days. Using Spitzer's research diagnostic criteria (Spitzer *et al.*, 1975) 10 patients had definite schizophrenia, 10 had definite manic disorder and the remaining 10 had various neurotic disorders. All the subjects were between 20 and 55 years old. Those who had received a recent muscle injury or intramuscular injection were excluded as were any who had been forcibly restrained. None of the patients had thyroid, muscle or cardiovascular disease and none abused alcohol. Patients taking drugs known to affect CPK activity were also excluded.

We found that in all three groups of patients the CPK isoenzyme pattern was the same as in normal healthy individuals with no brain-type CPK isoenzyme being present. It therefore seems probable that in patients with acute psychotic states serum CPK activity does indeed originate from skeletal muscle.

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References

- CHO, H. WON. & MELTZER, H. Y. (1979) Factors affecting stability of isoenzymes of creatine phosphokinase. *American Journal of Clinical Pathology*, **71**, 75–82.
- MELTZER, H. Y. (1976) Neuromuscular dysfunction in schizophrenia. *Schizophrenia Bulletin*, **2**, 106–35.
- NEALON, D. A. & HENDERSON, A. R. (1975) Lability of human creatine kinase isoenzymes at 37°C: A complication of electrophoretic separation. *Journal of Clinical Pathology*, **28**, 834–6.
- ROSALKI, S. B. (1967) An improved procedure of serum creatine phosphokinase determination. *Journal of Laboratory and Clinical Medicine*, **69**, 696–705.
- SOMER, H. & KONTTINEN, A. (1972) Determination of serum creatine kinase isoenzymes by fluorescence technique. *Clinica Chimica Acta*, **40**, 133–8.
- SPITZER, R. L., ENDICOTT, J. & ROBINS, E. (1975) Research diagnostic criteria for a selected group of functional disorders. *Instrument Number 58*. 2nd ed. New York State Psychiatric Institute.

PREMENSTRUAL SYNDROME

DEAR SIR,

I find Dr Katharina Dalton's objections (*Journal*, August 1980, **137**, 199) to Dr Gwyneth Sampson's double-blind trial of progesterone in the premenstrual syndrome (*Journal*, September 1979, **135**, 209–15) very difficult to understand. The Moos Menstrual Distress Questionnaire, as its title and Dr Dalton suggest, does indeed measure affective and somatic discomfort and behavioural change during the menstruum, but it was also expressly designed to measure, retrospectively, such changes in other phases of the cycle as well (Moos, 1969). Its ambition in this respect renders it somewhat unwieldy to use (Clare, 1977; Rouse, 1978) and many prefer to use Form T, which is composed of the same 47 items but which allows the subject to rate herself daily throughout a cycle. Dr Sampson appears to have used the daily self-rating form in her study. In the circumstances, Dr Dalton's objection that the MDQ 'only measures menstrual distress' is utterly mistaken and can have no bearing on Dr Sampson's results.

Dr Dalton queries the definition of the premenstrual syndrome used by Dr Sampson. In doing so, she provides her own, whereby *only* women who have symptoms premenstrually and *at no other time* in the cycle qualify as sufferers. The problem with this definition is that it presupposes that symptoms such as anxiety, depression, irritability, headache, backache and tension are relatively uncommon. The fact is, however, that many of the symptoms which make up the premenstrual syndrome are not uncommon and occur intermittently in women of childbearing years (Banks and Beresford, 1979; Ingham and Miller, 1979). In such cases, only the evidence of a premen-