It is true that in the practice of mental handicap, as in all other branches of medicine, we rely heavily on the help of associated professions and disciplines but this in no way invalidates the primacy of medicine in the provision of treatment and care, whether for neuro-surgery, paediatrics, obstetrics or mental handicap.

The director of research of an M.R.C. Unit, working under the auspices of a University Faculty of Medicine, surely cannot seriously query the advantages of academic departments and professional chairs which would stimulate more clinical research and improve clinical training!

It is a surprising fact that in this country, although there is a comprehensive network of chairs in general psychiatry, as well as chairs in forensic psychiatry and child psychiatry, and two chairs in psychology of mental handicap, there should not be a chair devoted to mental handicap as such.

One can be forgiven for assuming (particularly after reading Sir George Godber's paper) that this lack is part of a deliberate attempt to minimize the involvement of medicine in the care of the mentally handicapped and to hinder the improvement both of the quantity and the quality of consultants in the specialty. Against this background of official neglect and disparagement, which has Dr. Kushlick's blessing, it is not for my recommendation and those of the Mental Deficiency Section of the College 'to be judged against the current policy', but rather for the current official policy to be judged against the dismal record of the deterioration of the services of the provision of care. Thus:

1. The service previously integrated under medical guidance has been dismembered into separate medical, social and educational services.

2. The present services have attained under medical guidance impressive achievements in the provision of multi-disciplinary treatment and care, both in the hospital and in the community, and any shortcomings in it can be directly attributed to lack of money, facilities and official discouragement. The process of replacement of the existing method of care by any other, even if it were in the long run equally satisfactory, is bound to be very costly and would produce further deterioration in the quality of care during the interim period through lack of availability of alternative personnel.

3. The services, such as they are, have ground to a stop. The hospitals are being run down without prior building up of community services to take their place, if only because the cost of their provision is very much higher than was predicted. It is now more difficult to return a rehabilitated patient into the community than it has ever been before. At the same time, the hospitals with their reduced beddage are incapable of admitting desperately urgent cases, subjecting patients and their families to intolerable stress.

4. The academic status of the specialty is disparaged to the point when a man like Professor Berg (whom Dr. Kushlick quotes in his letter) has been forced to emigrate to Canada to obtain both research facilities and academic status which he could not get in this country. Consequently morale in the profession is low and recruitment is becoming more difficult. The nurses, equally discouraged by the reorganization and by Briggs, see their career prospects dwindle.

When Dr. Kushlick and Mr. Blunden say that 'Government targets for the implementation of the White Paper policies are very low and recent cuts have impeded progress further' they do not appear to realize that these events are built-in consequences of a policy which is not only ill-considered and ill-designed but also one that has not been tried for feasibility, particularly under present economic conditions.

I agree that 'unclear criticism in the absence of clear alternative proposals' are to be deprecated but I submit that it is for the Department of Health and Social Security and Dr. Kushlick to defend if they can the alternatives which events have already shown to be unworkable.

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INTERACTION BETWEEN DEPRESSED PATIENTS AND THEIR SPOUSES

DEAR SIR,

Mary Hinchliffe *et al.* (*Brit. J. Psychiat.* (February 1975), **126**, 164–72) present a fascinating analysis of the interpersonal behaviour of patients with depression. However, despite their conclusion, the evidence does not support their hypothesis that depressive behaviour is maintained by the behaviour of others. To do this, it would be necessary to show that a patient's communication with a stranger showed a communication pattern which was closer to the communication pattern with his spouse on recovery.

Including all the data where communication with the stranger was recorded, one finds seven conditions in which there appeared some improvement (overall expressiveness for male and female, negative expressiveness for male and female, objective focused movements for slow speaking, and body focused movements for slow and fast speaking), and five conditions where the contrary occurs (positive

expressiveness for male and female, congruence for slow and fast speakers, and objective focused movements for fast speakers). Furthermore, the interview with the stranger was apparently later than the interview with the spouse and some degree of recovery would have been expected.

If anything, these figures show that depressive behaviour is *not* consistently reduced by changing the social environment.

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SERUM CREATINE KINASE IN ACUTE PSYCHOSIS

DEAR SIR,

In a recent report in the *Journal* (1974, **125**, 280), Harding reported that 5 out of 34 acutely psychotic patients had increased serum creatine phosphokinase (CPK) activity (>100 IU/L). They compared this result to the study of Smith *et al.* (1970), in which, according to Harding, 25 normal subjects were found to have serum CPK levels greater than 300 IU/L. Since this latter figure is far in excess of any previous report of serum CPK activity in the normal population (Rosalki, 1967; Meltzer, Elkun and Moline, 1969), I checked the report of Smith *et al.* (1970) and found that serum CPK levels greater than 300 IU/L were found in only two of 296 subjects and none had serum CPK levels between 200–300 IU/L.

It is unfortunate that Harding did not report his data in acutely psychotic patients in relation to the time of onset of their psychotic symptoms. He states only that the onset was less than one month before admission. We have found that the incidence of increased serum CPK levels at admission in those psychotic patients with onset of gross psychotic symptoms less than seven days before admission (54/98) is significantly greater than those with onset greater than seven days (35/105; Chi-square, Yates' correction $= 8{\cdot}892$, $p < 0{\cdot}005$). The incidence of CPK increase in psychotic patients with symptoms greater than two weeks in our series is about 15 per cent, which is similar to the find of Harding (1974). The mean duration of the serum CPK increase in psychotic patients in our series is $3{\cdot}8 \pm$S.D. $3{\cdot}1$ days. Thus, duration of illness is a key factor in studying serum CPK levels in psychotic patients, just as it is in patients with myocardial infarction and cerebrovascular accidents, head injuries or infections (Roe *et al.*, 1972; Dubo *et al.*, 1967).

Harding believes that I have not sufficiently attended to the effects of activity on serum CPK activity in my previous studies and cites the finding of Griffith *et al.* (1966) that an 87 km. walk raised serum CPK activity markedly. Such massive activity is not characteristic of psychotic patients in my experience. Harding himself states that some psychotic patients with decreased motor activity had increased serum CPK levels. We have reported the same (Meltzer, 1969). We have specifically studied the effects of exhaustive isotonic exercise (Meltzer and Moline, 1969) and isometric exercise (Goode, D. and Meltzer, H. Y., in preparation) and found relatively small increases in serum CPK activity compared to those at the time of an acute psychotic period.

The increases in serum CPK levels in acute psychotic patients are comparable to those in patients with a variety of acute brain diseases in duration, magnitude, source, and percentage of patients with increases (Dubo *et al.*, 1967; Wolintz *et al.*, 1969). In these latter patients, there is no possibility that increased motor activity is the cause of the increases in serum CP levels. We have proposed that a similar mechanism underlies the serum CPK increase in patients with acute psychoses and patients with known acute brain diseases (Meltzer, 1969). Our current studies suggest that the psychiatric patients with increased serum CPK levels have more florid psychotic symptoms, require higher doses of medication and longer stay in hospital than those without increased serum CPK levels.

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REFERENCES

DUBO, H., PARK, D. C., PENNINGTON, R. J. T., KALBAG, R. M. & WALTON, J. N. (1967) Serum creatine kinase in cases of stroke, head injury and meningitis. *Lancet, ii*, 743–8.

GRIFFITHS, P. D. (1966) Serum levels of A.T.P.: Creatine phosphotransferase (creatine kinase), the normal range and effect of muscular activity. *Clinica Chimica Acta*, **13**, 413–20.

HARDING, T. (1974) Serum creatine kinase in acute psychosis. *British Journal of Psychiatry*, **125**, 280–5.

MELTZER, H. Y. (1969) Muscle enzyme release in the acute psychoses. *Archives of General Psychiatry*, **21**, 102–12.

MELTZER, H., ELKUN, L. & MOLINE, R. (1969) Serum enzyme changes in newly-admitted psychiatric patients. *Archives of General Psychiatry*, **21**, 731–8.

MELTZER, H. Y. & MOLINE, R. (1970) Plasma enzymatic activity after exercise: Study of psychiatric patients and their relatives. *Archives of General Psychiatry*, **22**, 390–3.