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Correspondence

Letters for publication in the Correspondence columns should be addressed to:
The Editor, British Journal of Psychiatry, 17 Belgrave Square, London SW1X 8PG

NEW LONG-STAY PATIENTS IN A HOSPITAL FOR MENTAL HANDICAP

DEAR SIR,

Dr Spencer has reported (*Journal*, January 1977, **130**, p 104) that a proportion of clients transferred to Meanwood Park Hospital remained there for a long time.

He asserts that the only or most appropriate intervention that could have been performed for these clients was to transfer them to Meanwood Park Hospital. However, he cannot be said to have justified this assertion, because he has not provided any description of what each client in Meanwood Park Hospital does throughout each day or any description of the unique components of Meanwood Park Hospital necessary to attain this level and range of activity.

He further asserts that *only* those facilities that are labelled similarly to Meanwood Park (i.e. 'Hospital'), and staffed by persons labelled similarly to the staff of Meanwood Park (i.e. 'nurse', 'doctor', 'auxiliary', etc) are appropriate places of residence for any other similar clients. Again, he has presented no evidence to support this assertion.

He gives no information for any client, on current problems, targets, interventions implemented or progress made, but suggests, wrongly, that the only conceivable target for clients in hospital is transfer out of hospital.

It is obvious that many possible service options differing markedly from Meanwood Park Hospital could achieve the minimal criteria described by Dr Spencer—namely, the maintenance of clients without specification of problems, targets and interventions or the documentation of progress. We believe, however, that it is important to investigate what arrangements of resources (service options) can be shown to achieve the criteria we proposed in this *Journal* (September 1976, **129**, pp 287–8).

There may be many alternative service options, but they can only be tested, and a rational choice made between them, if service personnel document precisely what they are doing (interventions), to what end (targets) and why (problems). Both they and other observers will then be able to judge whether or not services are attaining 'progressive and enlightened aims', and also what skills and performances are required if they are to do so.

The new and exciting advances being made in this field necessitate, as a priority, the setting of precise performance standards. We have made the case for this in detail in our evidence to the Jay Committee (Kushlich *et al*, 1976).

ALBERT KUSHLICK
DAVID FELCE
JOHN PALMER
JOHN SMITH

Health Care Evaluation Research Team,
Dawn House, Sleepers Hill,
Winchester SO22 4NG

Reference

KUSHLICH, A., FELCE, D., PALMER, J. & SMITH, J. (1976) Evidence to the Committee of Inquiry into Mental Handicap Nursing and Care from the Health Care Evaluation Research Team, Winchester. Duplicated.

DO ALCOHOLICS NEED VITAMINS?

DEAR SIR,

There have been numerous investigations (1, 2, 3) into the folate and B^{12} status of patients with the diagnosis of alcoholism. Serum folate is generally held to be a good index of dietary deficiency. The body stores of folate are small, and severe dietary deficiency usually manifests itself within about six weeks in low serum levels. Because of this, most physicians will treat alcoholics with high doses of vitamins with the idea of replenishing the patient's nutritional status as soon as possible (4).

I should like to report the folate and B^{12} status of 66 alcoholic patients admitted to a Regional Alcoholic Unit (Bexley Hospital, Old Bexley, Kent). Serum was collected, usually on the first day of admission, before routine vitamins supplements were administered. Serum folate concentration and vitamin B^{12} concentration were assayed microbiologically, using Lactobacillus casei and Lactobacillus leichmanni, respectively (5).

Different opinions exist regarding the normal folate levels in the general population. In this communication I follow the classification used by Reynolds and

his colleagues (6). This is to enable a comparison with another common group of patients admitted to a psychiatric hospital, i.e. those suffering from depressive illness.

The results are shown in the Tables I and II,

TABLE I

		Serum folate (ng/ml)		
Patient group	n	0–2.5	2.6–5.0	5.1+
Alcoholic	61	3	25	33
Depressive	91	22	41	28

$\chi^2 = 13.3$ (df 2) $P < 0.005$

TABLE II

		Serum B^{12} (pg/ml)	
Patient group	n	0–200	201+
Alcoholic	66	2	64
Depressive	84	13	71

$\chi^2 = 5.1$ $P < 0.025$

together with those reported by Reynolds *et al.* It will be seen that alcoholic patients have significantly less folate deficiency than depressive patients, a group not usually treated with vitamins. A nutritional history was taken on admission, but this showed no difference between those patients with a poor nutritional history (mean folate 5·3 ng/ml; SD 2·3; n = 48) and those with a good nutritional history (mean folate 4·6 ng/ml; SD 2·6; n = 14). There was also no correlation between age and either folate or B^{12}.

The results indicate that alcoholics are not specifically deficient in folate and B^{12}, and the question arises whether the bulk of alcoholics derive any substantial benefit from the massive doses of vitamins they often receive. Reynolds *et al* have shown that low serum folate rises towards normal during the patients' stay in hospital, when they receive normal hospital diet. Only controlled trials can decide whether alcoholics, depressives, and indeed other psychiatric groups, benefit from the vitamin supplements that are so commonly given at considerable expense.

I am grateful to Dr K. Shaw for allowing me to study his patients, and to Dr Cuddigan, Brook Hospital, for the serum folate and B^{12} estimations.

GINETTE THEANO

Warlingham Park Hospital,
Warlingham, Surrey

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TRANSMITTERS IN DEMENTIA

DEAR SIR,

The recent findings by separate workers of a specific cholinergic defect in the elderly and demented brain (p 318 this *Journal*) has considerable clinical implications. Firstly it offers some understanding of otherwise puzzling clinical findings. My own recent survey (Silverman) of elderly agitated demented patients treated at home shows the special value of haloperidol in controlling difficult behaviour. Major advantages are the negligible effects on cardiovascular and respiratory function in high dosage (e.g. 60–100 mg per day). What was *unexpected* was the low incidence and severity of Parkinsonian side-effects. Dr Davies has shown choline acetyltransferase significantly reduced in the neostriatum of the dement. This system is usually under tonic dopaminergic inhibitory control; extrapyramidal side-effects of dopaminergic blockers such as haloperidol would therefore be *expected* to be less if the cholinergic system is already impaired. Dr Davies found much more profound loss of choline acetyltransferase in the hippocampus and mamillary bodies, more pertinent to intellectual incapacity and linking with the greater sensitivity of older subjects to confusion with anticholinergic drugs. Thus confusional adverse effects with imipramine are far commoner in the elderly (Schmied, 1962). It is fortunate that Parkinsonism is rare in dementia with adequate haloperidol control; added anticholinergic anti-Parkinsonian drugs would only compromise the intellect further.

Drs Perry suggested increased choline acetyltransferase in treated schizophrenic brain against possibly lowered activity in the treated depressive. This might just be due to neuroleptic dopaminergic blockade disinhibiting cholinergic systems in the