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.

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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

former and some possible effect of anti-cholinergic anti-depressants in the latter. They alluded to one study reporting Parkinsonian symptoms among the early features of presenile dementia. This is hard to reconcile with their rarity in Alzheimer's dementia generally but might be explained as an *early* phase of denervation supersensitivity in the neostriatum. Obviously much careful, objective collaborative research by psychiatrists and neurochemists is necessary. Findings were unanimous, that receptor affinity for acetylcholine remains intact. The fault is in the presynaptic neurone. Also, remarkably, acetylcholine alone among the brain neurotransmitters is affected. There is some debate whether the cholinergic neurones actually 'fall out' or simply don't function. Urgent questions the clinician will want answered are whether the chemical failure can be reversed chemically, and why cholinergic neurones specifically are at risk (thoughts of anti-choline acetyltransferase antibodies obviously occur). For some the urgency of the answers is greater than for others: for a rapidly ageing population it is an area of work of the highest priority.

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