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DIETARY VITAMIN B₁₂ DEFICIENCY

DEAR SIR,

Psychiatrists tend to screen elderly patients with cognitive impairment for vitamin B₁₂ deficiency, despite the finding of Elwood *et al* (1971), in a community sample of over 500 elderly patients, that there was no association between vitamin B₁₂ deficiency and impairment on tests of memory and learning. As Shulman (1967a) had pointed out, both dementia and vitamin B₁₂ deficiency are common in the elderly, and finding the two conditions associated may imply nothing about the aetiology of the dementia.

We examined 50 demented patients, aged between 67 and 89 (mean age 80.8 years) who were admitted to the Royal Cornhill Hospital, Aberdeen. As part of an assessment procedure, serum vitamin B₁₂ and folate levels were measured in each patient. Thirty-eight of the patients were diagnosed clinically as suffering from senile dementia of the Alzheimer type and 12 as suffering from multi-infarct (arteriosclerotic) dementia). These two groups did not differ significantly with respect to serum vitamin B₁₂ or folate levels. The patients were also divided into those who had lived alone and those who had been accompanied prior to admission. Of those 21 patients living alone 8 (38 per cent) had low serum vitamin B₁₂ levels (below 200 pg/ml) and 10 (48 per cent) had low serum folate levels (below 2 ng/ml). The corresponding figures for the 29 accompanied patients were 3 (10 per cent) and 10 (35 per cent) respectively. While the higher proportion of patients who lived alone having low folate levels did not reach statistical significance, the higher proportion of vitamin B₁₂ deficient patients living alone was statistically significant (Chi-squared = 5.47, df 1, $P < 0.02$).

Elsborg *et al* (1976) have described dietary deficiency of vitamin B₁₂ in the elderly, although this aetiology is usually held to be rare (Magnus *et al*, 1982). The increased incidence of vitamin B₁₂ deficiency in the socially isolated patients in our sample strongly suggests a dietary aetiology. Furthermore, in 7 of the 11 deficient patients, it was not coupled with, and thus not secondary to, a primary folate deficiency.

The findings also suggest that vitamin B₁₂ deficiency is much more likely to be a result, rather than a cause, of dementing illnesses. This is supported by the findings that while mild dysmnesic syndromes associated with vitamin B₁₂ deficiency are potentially reversible with vitamin replacement therapy (Shul-

man, 1976b), the same does not apply to established dementias (Shulman, 1967a).

We would suggest that clinicians continue to screen demented elderly patients for vitamin B₁₂ deficiency. It should be recognised however that this screening is being done not to detect a cause, but more a possible result of dementing illness and its concomitant dietary deficiencies.

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ANTICHOLINERGIC ABUSE

DEAR SIR,

In their study of benzhexol abuse, Crawshaw & Mullen, (*Journal*, September 1984, **145**, 300–303) have made a valuable contribution towards the recognition of this important problem. They draw attention to the fact that their series of cases, like that of Jakubczyk *et al*, included only young men and women. We feel, however, that this is a reflection of the nature of their sample and not an inherent feature of anticholinergic abuse. Our recently published series includes patients ranging from 22–56 years old (Pullen, Best & Maguire, 1984). It is important, therefore, to be alert to the possibilities of anticholinergic abuse whatever the age of the patient. Our series also confirmed that all current anticholinergic drugs, not just benzhexol, have a potential for abuse.

It may also be of interest that we have now calculated a 10% incidence of history of anticholinergic abuse for the current out-patients of the Marl-