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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.

FRANK RAWLINSON

*St. Ann's Hospital,
Haven Road,
Canford Cliffs,
Poole, Dorset*

JOHN M. EAGLES

*Clinical Research Centre,
Royal Cornhill Hospital,
Aberdeen AB9 2ZH*

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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-

borough Rehabilitation Unit, Oxford. This is comparable with the figure of 7% reported by Kaminer for his out-patients in Israel and by Pakes for his in the United States.

G. P. PULLEN
N. R. BEST
J. MAGUIRE

*Littlemore Hospital,
Littlemore, Oxford OX4 4XN*

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ROCK AND ROLL DELUSIONS

DEAR SIR,

Further to the account by Glass and Campbell of two female patients with delusions involving rock performers (*Journal*, July 1984, **145**, 95–96). I report four patients who have had delusions and/or hallucinations concerning pop stars.

Case 1: A 22 year old single man with schizophrenia believed falsely that he was famous as a celebrity and that he had met Davie Bowie and John Lennon. He was upset by Lennon's death in December 1980 but continued to hear voices of famous people including Lennon talking to him. He died by committing suicide in 1983.

Case 2: This 25 year old single man whose mother suffered from schizophrenia presented with the belief that Hank Marvin owed him a large sum of money. This idea took seed some years earlier when he had been at a Hank Marvin concert and the star had given him a special look. He refused treatment after experiencing side effects from a depot neuroleptic. When last seen he intended to go to the next local Hank Marvin concert and he expected that Hank would make a sign to invite him for a drink.

Case 3: This 30 year old single man had a history of delinquency, multiple drug abuse and overdose in his teens. Schizophrenia was diagnosed when he was 21 and he has required continuous in-patient treatment since the age of 22. He has experienced voices coming from the television which say that he is David Cassidy's double. At times he has been convinced that he is David Cassidy, Elvis Presley or David Essex. He believed that John Lennon was trying to get him out of hospital and blamed hospital staff for killing Lennon and Presley.

Case 4: A 30 year old married girl has suffered from schizophrenia for five years. There is a past history of alcohol and multiple drug abuse. In 1980 she burnt her wrist with a

cigarette after hearing Linda McCartney's voice telling her to do it. She also heard Paul McCartney's voice telling her to wait for him. Recently she relapsed nine months after stopping treatment and she has heard several voices including those of Boy George and Sting commenting on her actions.

All these patients have had severe illnesses characterised by many other delusions and hallucinations in addition to their experiences concerning pop stars.

I thank Dr. John Le Gassicke for permission to report case 3.

A. J. ROBINSON

*St. George's Hospital,
Morpeth, Northumberland NE61 2NU*

DEAR SIR,

The review of *Sexual Strands: Understanding and Treating Sexual Anomalies in Men*, by F. E. Kenyon, is misleading (*Journal*, May 1984, **144**, 563). The book discusses theories and their supporting empirical evidence on the causes and treatments for a wide range of sexually anomalous behaviours.

Dr. Kenyon's review suggests that he has not read the book carefully for its intended purpose or its content. I object to his statement that the book is "within the limitations of a committed behaviourist's standpoint", when most major psychological theories and treatments, from castration and Provera to psychoanalysis, have been discussed and evaluated.

He further states that my "ultra critical approach leaves little scope for treatment so that what is eventually advocated appears rather impersonal and mechanistic." This statement epitomises the inaccuracy of the review. My attempt has been to humanise our clinical practice. As I said in *Sexual Strands*: "One factor has aroused my anger and perplexed me. It is the selection of aversion therapy as a first and most frequent choice in treating sexual anomalies . . . aversion methods have no greater effect in changing behaviour than the less noxious positive behaviour therapies or psychotherapies. Can we continue to permit patients to suffer unnecessarily? . . . *A range of behavioural and psychotherapy methods is available and should be tried out.* Any growth experience or change can be painful, but if the patient feels he is the master of his destiny, it is a mutual concern of therapist and client to change and the patient has dignity and control." (p. 498).

R. LANGEVIN

*Clarke Institute of Psychiatry,
250 College Street, Toronto M5T 1R8*

CORRECTION

The heading to the letter from Drs Addonizio and Susman (*Journal*, November 1984, **145**, 556–557) should read NEUROLEPTIC MALIGNANT SYNDROME AND HEAT STROKE.