Malnutrition and Mental Disease

SIR,—In a recent letter (January 20, p. 191) Dr. Gerald Milner has described an impressive change for the better in the mental state of a chronic paranoid schizophrenic given massive doses of ascorbic acid. Dr. Milner draws attention to the work of Lucksch,[1] who reported similar results, and goes on to suggest that vitamin C deficiency may contribute to mental illness in the community and in chronic mental hospital patients. I would like to suggest that there may be avitaminosis C or hypovitaminosis C states in schizophrenics for reasons other than poor dietary habits, though these may well be contributing factors.

One of the most controversial biochemical findings in schizophrenia is the possibility of chronic hypercupraemia. Many workers have reported[2-9] high blood copper and raised ceruloplasmin in schizophrenics, but the findings have not been universally accepted. However, Abood[10] has shown that blood copper levels fluctuate rapidly in emotional states, and it is likely that, particularly during phases of excitement, schizophrenics show a tendency towards hypercupraemia. Now copper is a potent catalyst for the oxidation of ascorbate to dehydroascorbate. Even if this catalysis is confined to non-ceruloplasmin copper, an excessive breakdown of ascorbate by schizophrenics is to be predicted. In as yet unpublished work, studies in this laboratory have provided evidence of significantly greater 24-hour urinary excretion of dehydroascorbate and diketogulonate in a group of chronic schizophrenics than in a group of hospitalized epileptics receiving the same diet, or in a normal group. I have suggested elsewhere[11] that chronic hypovitaminosis C can lead to an imbalance in aromatic transformations due to a partial blockage of the liver phenylalanine oxidase system at the *p*-hydroxyphenylpyruvate oxidase level. Such an imbalance could lead to excess tissue adrenaline, and possibly to psychotomimetic adrenaline metabolites.

A clinical study into the effects of a daily 1 g. ascorbic acid on chronic schizophrenics is at present being undertaken in co-operation with Dr. B. D. Hart at the Porirua Mental Hospital.—I am, etc.,

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Aversion Therapy for Homosexuality

SIR,—Dr. Basil James (March 17, p. 768) is to be congratulated on confirming the pioneer work of Srnec and Freund[1] by his successful cure of a case of homosexuality by aversion therapy. There is no doubt that this may prove a valuable addition to our armamentarium in dealing with this difficult disease.

Some psychiatrists continued to teach that homosexuality was incurable in spite of the fact that there were many recorded cures. This idea persisted into the Wolfenden Report. However, we were not asked at the inquiry if we could provide "any references in the medical literature to a complete change of this kind," otherwise one would have suggested successful cases by London,[2] Naftaly,[3] Lilienstein,[4] Laforgue,[5] Stekel,[6] Gordon,[7] Serog,[8] Frey,[9] Bircher,[10] Sumbaev,[11] Allen,[12 13] and others. Nevertheless, those who have treated cases will agree that if a patient is willing to face the discomfort of aversion therapy it may be much briefer, although not all successful psychotherapy need be prolonged.

The fact that homosexuality can be cured, whether by aversion therapy or not, suggests that the work of those who have discovered genetic bases for it is mistaken. Similarly the absurd suggestion in the Wolfenden Report that homosexuality is not a disease is refuted by successful cures.

Aversion therapy is so unpleasant that many people will still prefer psychotherapy and only if this fails seek the more drastic cure. However, since it is believed that more than 5% of the population suffer from some form of homosexuality the important thing is effective treatment rather than futile imprisonment which leaves the sufferer on discharge in the same condition psychologically as when he went in.—I am, etc.,

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Diagnosis of Vertigo

SIR,—I was interested to read your annotation (December 23, p. 1696) in which you referred to the various publications on epidemic vertigo (vestibular neuronitis). In May and June, 1946, while serving with the armed Forces in Malaya, 25 cases of this condition came under my care and were admitted to the combined military hospital at Jahore Bharu (South Malaya).

Most of the cases were admitted with a history of sudden onset of vertigo and vomiting which persisted for several days. Nystagmus of a coarse type was noticed in all these patients. No other neurological abnormality was noticed. Laboratory investigations proved essentially negative, but in two cases there was increase in cells in the C.S.F. and in one additional case both cells and protein were increased. The course was invariably benign. A detailed report of the epidemic was published in 1951.[1] I have since come across sporadic cases of this syndrome from time to time in