1975) and therefore the reference range used should be stated. But more importantly, it is now established that a low serum folate is not diagnostic of "folate deficiency". It has been stressed (Chanarin, 1983) that a low serum folate may better be interpreted as negative folate balance, possibly dietary in origin, but for the diagnosis of folate deficiency red blood cell folate level is required. It is therefore more appropriate to perform red blood cell folate assay in the investigation of demented patients.

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Vitamin Supplement to Alcoholic Beverages

DEAR SIR,

Dr Weller (*Journal*, March 1984, **144**, 329) is correct in asserting that prompt use of thiamine can reverse or prevent some of the more serious side-effects of alcohol abuse. Contrary to his assertion however, thiamine deficiency in alcohol abuse arises from inadequate dietary intake of vitamin B1, impaired absorption of the vitamin across the intestine and reduced hepatic activation of vitamin B1 to its metabolically active form Thiamine Pyrophosphate (T.P.P.) (Thompson *et al*, 1980). It follows then that, "Wernicke's encephalopathy following glucose infusion and upon refeeding prisoners of war or patients following a starvation diet", is due to the sudden flooding of the carbohydrate metabolic pathways and consequent increased utilisation of T.P.P. co-factor leading to acute deficiency of the co-factor in an already nutritionally compromised individual.

Therefore, addition of vitamin B1 to alcoholic beverages would not necessarily increase the available pool of biologically active T.P.P. I am concerned that drinkers may be falsely assured that added vitamin B1 counteracts any possible sequelae of their alcohol abuse. It has been shown (Brin, 1964) that as little as 200 days of a diet containing 200 micrograms of Thiamine, approximately one tenth of the normal daily requirement, can cause Wernicke's encephalopathy.

I submit that more concern for the nutritional state of alcohol abusers and less assurances to them that additives make drinking safer would be more appropriate.

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Hazards of Hard Contact Lenses in Psychotic Patients

DEAR SIR,

Psychotic individuals are more likely than normals to ignore the rules for contact lens wearers (Scheie & Albert, 1977; Cooper & Constable, 1977). Our two case histories illustrate this point.

Case i: A 37 year old male paranoid schizophrenic was admitted to our hospital with loosening of associations, delusions and gaze avoidance. For several weeks he had lived in complete darkness, using mirrors in the windows to reflect the sun's light outwards. He had been wearing contact lenses continuously for three weeks. Examination revealed hard contact lenses, severe bilateral conjunctivitis, and abrasions of the left cornea. These slowly recovered with treatment: chloramphenicol drops and an eye patch.

Case ii: A 30 year old male was admitted with schizophrenic mutism and extreme gaze avoidance. The latter clinical feature was attributed to his disturbed mental state. The nursing staff discovered an empty contact lenses carrying case among his possessions. A second physical examination revealed hard contact lenses and bilateral severe conjunctivitis. The latter cleared with sulphacetamide eye drops. A collateral history revealed that the lenses had been worn day and night for one week before admission.

Caution should be exercised when prescribing contact lenses to patients with severe psychiatric illness. Their presence should be considered in the evaluation of gaze avoidance amongst psychotics, especially in schizophrenia.

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