Henderson (1965), but there is a lack of positive proof of its occurrence in psychiatric patients (at the present time). Response of the mental illness to drug therapy before treatment of the deficiency state is not a strong argument, as it is probable that the effects of antidepressants, etc. are non-specific and may not be correcting the basic biochemical abnormality. Relapse while on vitamin B_{12} therapy is a more powerful argument, but again there are possible explanations. Environmental factors may be relevant, but it is more probable that either (1) insufficient vitamin B_{12} is being administered (it does seem that larger quantities of vitamin B_{12} are required to induce a remission in the mental state than are needed for the haematological disorder), or (2) the patients are also suffering from folate deficiency. From the limited experience so far gained in epileptic patients, mental illness responds more satisfactorily to a combination of folic acid and vitamin B_{12} than to folic acid alone.

Until we have a clearer understanding of the metabolic interrelationships of folic acid and vitamin B_{12} both within and outside the nervous system and their relative roles in the production of psychiatric illness, the possibility that some patients need to be treated with both vitamins should be kept in mind.

3. Finally, the problem of satisfactory screening procedure for vitamin B_{12} deficiency. This has recently been discussed in the correspondence columns of the *Lancet* following the publication of their experience with the antigastric-antibody test by Henderson, Strachan, Beck, Dawson and Daniel (1966). Dr. Shulman concludes from his findings that careful examination of a peripheral blood film is adequate. One hopes that too much weight will not be attached to this conclusion, as half of his patients with vitamin B_{12} deficiency were already anaemic. One suspects that if his patients with pernicious anaemia at least had had vitamin B_{12} levels assayed years earlier, they might not have reached such a state of advanced dementia as to render them unresponsive to treatment. Certainly epileptic patients can suffer from folate deficiency for many years without any detectable trace of this being found in the peripheral blood film (Reynolds *et al.*, 1966a).

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WHAT THEY REALLY SAID

Dear Sir,

Perhaps the least creditable aspect of the careers of those two men of genius Freud and Jung was their bitter schism; and one of its most invidious accompaniments was the subsequent vendetta between their disciples. Outliving Freud by over thirty years, Jung seemed inevitably to have the last public word on most points of contention; but even so, neither he nor Freud ever claimed to have subjected the other to a personal analysis, nor to have received one at the other's hands.

This has not prevented Dr. E. A. Bennet, author of *What Jung Really Said* (1), from devoting almost two pages of his 185-page treatise to challenging three and a half lines from my own 264-page predecessor, *What Freud Really Said* (2), and thereby misleading your reviewer (*Journal*, April, 1967, p. 453) into believing that "Freud and Jung analysed each other".

What is the substance of Dr. Bennet's uncharacteristic lapse into tendentious factionalism? Simply that for seven weeks on a trans-Atlantic trip in 1909, Freud and Jung swapped dreams and speculated on their interpretation. From the single example given, Jung at least would seem to have preferred his own version to Freud's invited observations; nor did either of them continue the experiment after the trip was over.

In the perspective of their two prodigious lives this might seem a fairly trivial incident; and that is how I treated it, regarding Dr. Bennet's subsequently disproportionate emphasis with objective tolerance in my own review of his book. But to persist in restrained silence while Dr. Flanagan swallows bait, hook, line and sinker would possibly be to compound an initially avoidable confusion.

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ANAEMIA AND RETICULUM CELL HYPERPLASIA IN SCHIZOPHRENIA

DEAR SIR,

In answer to Dr. Baur's letter in the *Journal* for May, 1967 (referring to my article in the December, 1966 issue), I agree with your correspondent concerning the blood dyscrasia which can be caused by modern tranquillizers, especially by the phenothiazine derivatives, as I have investigated several of these myself. I did consider this possibility in each of the cases described, but came to the conclusion that no particular type of drug could be incriminated in my series. In addition I discovered that very many of the control cases were on similar drugs over long periods, and therefore I accepted "the other mental cases" as sufficient control in this respect.

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https://doi.org/10.1192/bjp.113.499.683 Published online by Cambridge University Press