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Correspondence

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THYROID FUNCTION SCREENING IN PSYCHIATRIC IN-PATIENTS

DEAR SIR,

In relation to the paper by Drs Carney, MacLeod and Sheffield (*Journal*, February 1981, **138**, 154–56) we would like to make the following comments:

1. The authors diagnosed abnormal thyroid function if one of 3 criteria was reached: (a) successively abnormal FTI's during the same admission: (b) single FTI exceeding 1.55 or below 0.45; or, (c) a well-founded clinical diagnosis of thyrotoxicosis or myxedema. The first two criteria, if accepted as sufficient, may lead according to our findings, to false positive diagnoses of thyroid dysfunction.

We have reported that among 480 newly-admitted psychiatric patients, 85 had abnormal FTI's (called in our report Estimated Free Thyroxine or EFT). Fifty per cent (27 elevated and 16 decreased) returned to normal within 4–14 days of admission. These 'transient' abnormal values were as low as 0.5 and as high as 3.6 (normal range is 1.0–2.1). This indicates that even a severely abnormal index may reflect only a transient effect of psychiatric illness and/or hospital admission. Distinguishing between such 'transient' and 'persistent' abnormalities is useful since it is only the latter type of dysfunction that has any clinical significance (Cohen and Swigar, 1979).

2. It would be interesting to know whether, in patients whose thyroid dysfunction was diagnosed before onset of their mental disorder, the abnormal FTI reflected non-compliance with thyroid medication prescribed. In our group, this was the cause of the abnormal index in 9 patients.

3. Finally, we wonder whether alcohol abuse, common among psychiatric patients with various diagnoses, and especially among those with affective illness (Kolakowska and Swigar, 1977; Stokes, 1974) could have played a role in low FTI's in some patients. In our sample transient decreases in thyroid function were highly associated with alcohol or sedative use and abuse.

MARY E. SWIGAR
KENNETH COHEN

School of Medicine, Yale University,
New Haven, Connecticut, USA

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PROPRANOLOL IN SCHIZOPHRENIA

DEAR SIR,

It will be a pity if the dubious conclusion of Peet *et al* (*Journal*, August 1981, **139**, 105–11) is allowed to confound the important issue of the place of propranolol in treating schizophrenia.

On the measures actually presented (BPRS schizophrenia scale, NOSIE total assets, relapse rate) the authors found no significant difference between placebo and propranolol, and conclude "We have demonstrated that propranolol has no important advantages over placebo". On the same measures, over the same period, they also found no significant difference between placebo and chlorpromazine—yet conclude that "our patients as a group were chlorpromazine-resistant"! This is special pleading—why not conclude that chlorpromazine has no advantage over placebo?

Perhaps the most practical measure in the study was the numbers of patients withdrawn from it by their physicians due to relapse. The relapse rates were chlorpromazine 25 per cent, propranolol 21 per cent, placebo 39 per cent. These figures suggest that it is not propranolol which is ineffective, but short-duration trials in chronic conditions.

A. C. CARR

Institute of Psychiatry,
De Crespigny Park,
Denmark Hill,
London SE5 8AF