Your correspondents warn that 'great danger indeed exists in combining the tricyclic drugs and MAOI's in large dosage', and then go on to report in support of this statement a case where a patient has received not a *large* dose but a *gross overdose* of one of the drugs.

Their patient received up to 180 mg. of phenelzine daily for ten days; a large enough dose to account for all of the adverse effects observed even if the patient were not receiving any other drug. Of course the addition of 75 mg. of amitriptyline daily no doubt ensured the certainty of disaster.

180 mg. of phenelzine daily is more than twice the maximum recommended dose and four times the usual starting dose even when used alone. It is twelve times the reasonable starting dose when used in combination with amitriptyline (1) (2).

This case adds no new information to our knowledge of the dangers of any of the drugs involved, but it does underline the importance of ensuring that there is careful medical management and control when powerful and potentially lethal drugs of any nature are prescribed.

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REFERENCES

1. WINSTON, F. (1971). 'Combined antidepressant therapy.' *Brit. J. Psychiat.*, **118**, 301.
2. SCHUCKIT, M. *et al.* (1971). 'Tricyclic antidepressants, and monoamine oxidase inhibitors.' *Arch. gen. Psych.*, **24**, 6.

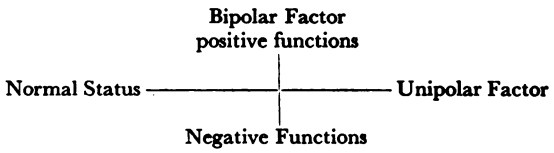
## THE CONTINUUM MODEL OF MANIC-DEPRESSIVE PSYCHOSIS

DEAR SIR,

J. H. Court (*Journal*, February 1972, p. 133–41) has sought to reaffirm that a continuum model explains manic-depressive psychosis better than the bipolar Kraepelinian model. He draws attention in the continuum to a number of unipolar variables, namely, progressive failure of the sodium pump, increasing reaction time and insomnia; one might add increasing distractibility and psychoticism. The correlation of these variables with mood is a valuable concept. However, it is inherently unlikely that so complex an illness could be modelled, as he suggests, on a single dimension. (Following his argument *ad absurdum*, minimal hypomania is predicted to alternate with maximal depression.)

The unipolar variables from which he constructs the continuum are, with the exception of mood, primarily physiological, relating to underlying biochemistry and brain function. Conversely the bipolar model is a clinical or behavioural model. These may not be mutually exclusive, but rather complementary.

Bipolar changes from one clinical state to the other are the characteristic of this illness. They are obvious in mood, activity and speech, but also apparent in changes in superego pressures, in the tendency to take percepts from internal or external cues and the alterations in the balance of intra/extrapunitive hostility. At the risk of creating a fourth 'model', I would suggest that these two sets of functions, the unipolar and bipolar, seem likely to subserve different physiological (and/or psychic) phenomena. They might usefully be investigated against a combined axis as under:




It is likely that a complete statement to account for the wealth of clinical presentations would involve many more variables than these two groups.

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## BRAIN HYPOXIA

DEAR SIR,

I am writing to you, as one of the Assessors for your *Journal*, to protest about the review of *Brain Hypoxia*, edited by Brierley and Meldrum, in the February 1972 issue (p. 239).

The review is not only unfair to contributors, editors and publishers of the symposium, but is singularly unhelpful to your readers who may want to know whether to get the book from a library or even whether to buy it. Your reviewer's criticism that in this volume '. . . results can be made public for a second (or even a third) time . . .' is unjustified. The book, in fact, contains an unusual amount of new information; for example in the section on the physiological and neuropathological effects of hypoglycaemia in adult and new-born animals, in the section on the physiology of induced seizues, or in that on continuous monitoring of intracranial pressure during and after exposure to hypoxia. And I for one shall certainly refer students to this book, if they want to find out how these problems and many others are being tackled experimentally and clinically.

As far as I know there has been no Symposium on cerebral hypoxia and related subjects for ten years. These well edited and illustrated Proceedings would