

text) but unreasonable on the basis of such reaction time measurements. Thirdly, in the author's series of reaction times are included depressives who '... had never had a manic episode'. This simply confounds the experiment at its roots and nullifies any conclusions. Such depressives may well not be true cyclothymes, and there is now increasing evidence that manic-depressive psychosis is genetically distinct from other forms of depression, and hence may well require a *model* of its own. (Winokur and Clayton, 1967; Slater, 1953).

7. It is my experience, shared with others, that certain drugs which are very effective against mania can induce a return to normal mood without intervening depression. However, if the drug, and I am thinking particularly of haloperidol, is continued overlong, a deep depression sometimes ensues, especially in known cyclothymes. It is significant that adding further haloperidol at this stage never leads to the 'lower' state of normality, but only to further deepening of the depression. Here the suitability of the author's model seems strained to its limit.

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REFERENCES

- BECK, A. T., FESHBACH, S., and LEGG, D. (1962). *J. Consult. Psychol.*, 26, 263-8.
SLATER, E. T. O. (1953). *Spec. Rep. Ser. Med. Res. Council. (Lond.)* No. 278.
WINOKUR, G., and CLAYTON, P. (1967). *Recent Adv. biol. Psychiat.*, 9, 35.

DEAR SIR,

I am delighted that my paper has provoked thought as intended, and welcome the opportunity to respond to Dr. Silverman's reflections.

1. If I understand the point, I think I agree. The postulate 'if mania and depression are opposites' is of course the traditional one I reject, so I would happily grant that an intervening state of euthymia need not occur. However, the graphical representations of Klein and Nunn (1945) and Jenner *et al.*, (1967) undoubtedly do imply this, as do most verbal accounts of the transition from the one state to the other. I fear that Silverman has inadvertently put up a straw man and then knocked it down.

2. A model must do justice to observations, and in principle even a single observation might justify one discarding a model. I would therefore disagree that the model must only account for common observations.

Further, I would want documentary evidence that the 'mixed state is generally admitted a moderate rarity'. On the contrary, I would assert that features of depression are commonly apparent in hypomanic patients. As long as assessments are made only on the basis of clinical presupposition, depressive features are readily overlooked, but when measuring devices assessing both aspects are used evidence of both is often apparent. My own current research, using the Foulds Symptom-Sign Inventory (with both depressive and manic scales), has confirmed for me the importance of recognizing the co-existence of manic and depressive symptoms.

3. To object that suicide is more common than mania ignores the concept of an hereditary predisposition to manic-depressive psychosis. This objection might be relevant if it could be shown that suicide *among manic-depressives* is more common than is hypomania, but even then it would not destroy the model. The phrase 'if life is to be preserved' represents suicide or the manic defence as alternative forms of adjustment whose relative frequency has nothing to do with the model. It seems quite proper to relate this view to what many already endorse, viz. the analytic concept of defence, not in order to incorporate this into the model but as a means of communication with colleagues, since at least on this point one finds common ground.

4. I can only disagree completely on this point. My psychiatric colleagues undoubtedly judge the spacing of ECT in relation to urgency, when this form of treatment is indicated. I am prepared to believe that mild neurotic depressions are resistant to ECT, never having worked with colleagues who considered it was indicated in this condition, but the point is irrelevant in a discussion of psychosis.

5. Since lithium has been proclaimed as specifically a treatment for mania, it takes a brave man to use it in depression. Not surprisingly the evidence for its value in depression has been marginal or anecdotal. My hope was that a revised model might encourage others to explore the possibilities more extensively. It appears that Silverman did not examine the most recent psychiatric literature before writing, since there appeared, shortly before publication of my own paper, further support from Fieve, Platman and Plutchik (1968), who found a mild anti-depressant effect for lithium in a double-blind trial, and from Dyson and Mendelson (1968), who incidentally considered it appropriate to include patients who had suffered only from recurrent depression. They even see their results as strengthening the case for retaining the category of recurrent cyclical depression within the framework of manic-depressive psychosis. Far be

it for me to reject the traditional classification without sufficient evidence. My use of depressives who had not experienced a manic phase *may* not be defensible according to some evidence, but equally it may be entirely defensible, and Dyson and Mendelson's paper I take as strengthening my position. My understanding of imipramine is that in spite of its pharmacological similarity to chlorpromazine it is *not* noted for its tranquillizing effects. Even if Dr. Silverman finds its anti-manic properties unremarkable, those writers quoted in my paper certainly do betray a note of surprise at its effectiveness.

6. It is absurd to object that because one paper reporting the use of the digit-symbol test does not reflect depressive retardation neither will a measure of reaction time. Several different factors have been isolated among psychomotor measures (Seashore, 1951). The digit-symbol test involves a continuous response, while simple RT involves 'speed of initiating a single response'. The differential properties of psychomotor tests in relation to clinical status have been reported elsewhere (Court and Cameron, 1963). That reaction time provides a suitable measure for judging severity of illness may be concluded from my own work (Court, 1964), and it would be difficult to be more positive than King (1968) in a recent review—'a synthesis of the empirical findings reported by many different investigators, working with quite varied hypotheses, populations and procedures, makes clear the following themes: psychomotor retardation is found at all three levels of function (sub-factors) tested among the psychoses; most noticeably among the schizophrenias, but present in depression and mania as well. The degree of slowing follows closely clinical estimates made of the severity of disorder. . . .'

7. I respect Dr. Silverman's observations relating to the appearance of depression following the administration of haloperidol. While the point may prove an embarrassment to the model, I would prefer to await scientific documentation of this before rejecting the view that spontaneous fluctuations of the illness account for his observations. Hitherto it has appeared that the neuroleptics do not induce depression.

8. Finally, Sir, since my proposal is by no means conclusively established, may I allude to one final piece of evidence which has come to my notice since preparing the paper for publication. It follows that, with the model proposed, any improvements in the treatment of manic-depressive depression should lead to a corresponding decline in the incidence of mania. Older clinicians speak of such a decline, and this has been documented by Lachman and Abrams

(1963) who show a dramatic decline in admissions of manic patients to Bellevue Hospital during the years 1948–62. They did not find any single causal factor to explain this, but it could be argued that a high percentage of patients who would previously have developed manic episodes were effectively treated while depressed and never developed the more extreme reaction. In other words, the decline of mania arises from the improved treatment of recurrent depression.

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REFERENCES

- COURT, J. H. (1964). 'A longitudinal study of psychomotor functioning in acute psychiatric patients.' *Brit. J. med. Psychol.* **37**, 167–73.
- and CAMERON, I., (1963). 'Psychomotor assessment of the effects of haloperidol.' *Percept. Mot. Skills*, **17**, 168–70.
- DYSON, W. L., and MENDELSON, M. (1968). 'Recurrent depressions and the lithium ion.' *Amer. J. Psychiat.*, **125**, 4, 136–40.
- FIEVE, R. R., PLATMAN, S. R., and PLUTCHIK, R. R. (1968). 'The use of lithium in affective disorders: I. Acute endogenous depression.' *Amer. J. Psychiat.*, **125**, 4, 79–83.
- JENNER, F. A., GJESSING, L. R., COX, J. R. *et al.* (1967). 'A manic-depressive psychotic with a persistent forty-eight hour cycle.' *Brit. J. Psychiat.*, **113**, 895–910.
- KING, H. E. (1968). 'Psychomotility: a dimension of behaviour disorder.' Paper read at American Psychopathological Association. To be published.
- KLEIN, R., and NUNN, R. F. (1945). 'Clinical and biochemical analysis of a case of manic-depressive psychosis showing regular weekly cycles.' *J. ment. Sci.*, **91**, 79–88.
- LACHMAN, J. H., and ABRAMS, A. L. (1963). 'The decline and fall of manic-depressive psychosis, manic type.' *Amer. J. Psychiat.*, **120**, 276–7.
- SEASHORE, R. H. (1951). 'Work and motor performance.' In: *Handbook of Experimental Psychology*, (Ed. S. S. Stevens). New York: J. Wiley and Sons.

CHROMOSOME ABNORMALITIES IN PSYCHIATRIC PATIENTS

DEAR SIR,

Anders *et al.* (*Journal*, September 1968, p. 1167) found a significantly higher frequency of hypermodal cells and acentric chromosome fragments in psychiatric patients, compared with a control group. The authors mention that these abnormalities might be causally related to the mental illness of these patients.