Brit. J. Psychiat. (1969), 115, 371–5

Correspondence

MANIC-DEPRESSIVE PSYCHOSIS

DEAR SIR,

I found fascinating and provocative reading in J. H. Court's article (*Journal*, December 1968, p.1523), which proffered a daring new 'conceptual model' for manic-depressive psychosis. Although I must thank the author for a stimulating account, its provocativeness makes me seek your permission to suggest a few points of possible criticism.

1. It is stated 'if mania and depression are opposites, then a move from one to the other would demand a period of normality during the transition'. Now, firstly, talking in terms of a *model* rather than an hypothesis (and this is a distinction most central throughout this problem) the *overt* mood of depression may indeed appear the same as that of an extrapolated lower extreme on a continuum of mood seen in 'normality', and the *overt* mood of mania vice versa. However, the depressive state and the manic one may be metastable alternative states to normality with *their* own limited continua, and a change from the metastable depressive to the metastable manic state would then not necessarily demand such an intervening state of euthymia.

2. The difficulty of the 'mixed state' would seem to be more embarrassing to the author's model than to that of the classical form. If the new model is to be useful as a *model* rather than an *hypothesis* it should epitomize the *common*, not the *un*common observation, rather than strive to be true in fine. Since the mixed state (I do not include the agitated depression) is generally admitted a moderate rarity, a model which makes especial provision for it seems unsuitable in general. Further, in the old model a mixed state could adequately be explained in terms of lag phenomena acting on clusters of features which migrate as a group along a continuum but are distinct and can move at different rates.

3. Quoting again: 'But when a person who is already depressed suffers further trauma . . . it is easier to envisage a deepening of depression to such a degree that an alternative reaction is necessary if life is to be preserved, so the manic defence is invoked.' Here two objections are immediately apparent. Firstly, it is an unfortunate reality that nature has not been as wise or as humane as the author, suicidal acts being considerably more frequent than manias. Secondly, to here introduce the teleological and 'analytic' concept of 'defence' into this setting is to mix models disastrously and do damage to both; they are immiscible.

4. Later, it is stated that 'the frequency of administration of ECT usually offers some clue to the extremity of the psychotic state'. This just won't do either conceptually or as fact; indeed mild neurotic depressions are often notoriously resistant to the effects of ECT.

5. On the subject of the effects of drugs, firstly it is claimed that lithium is effective in depressions, but this, I think it fair to say, is still quite disputable. Moreover, regarding patients who are classified as manic-depressives but 'with a history of depression only', such patients simply cannot be called manic-depressive in any meaningful way (*vide infra*). As an argument, the author points to possible beneficial effects of imipramine in *both* depression and mania as paradoxical in terms of the classical model. This is only so if one ignores the pharmacology of the drug and disregards the fact that many complex molecules exert quite different biological actions at different positions on the molecule. Imipramine is after all a very close relation of chlorpromazine and was discovered in the search for a new *tranquillizer*. It is hardly surprising if it has some 'atavistic' anti-manic properties demonstrable.

6. The author offers the measurement of reaction time as support for the fitness of his model. Now reaction time has very little to do with the common assessment of affective illness, and hence once again support for the *model* is being confused with an attempt to test something else, a particular *hypothesis*. However, excepting that point for the moment, there are further objections here. Firstly, reaction time is merely one psychomotor measure, and it is at least of interest that a number of workers have shown that other tests show no objective retardation in depression, e.g. in the digit-symbol test (Beck, Feshbach and Legg, 1962). Secondly, to try to make out a case for the 'hypothesis that elated patients are more severely ill than depressed patients . . .' is not only unnecessary in terms even of the *author's* model (it is never stated that the ordinate of his sine curve measures 'severity', whatever that means in this con-

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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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