'PLAYING AND REALITY' BY D. W. WINNICOTT

DEAR SIR,

I would ask for a modest space in your correspondence columns in order to set right the record concerning the circumstances in which Donald Winnicott's posthumous work, *Playing and Reality*, came to be compiled and published, since the facts are quite other than the suppositions made by your reviewer, Frank J. Menolascino, in your issue of January 1972 (p. 106).

It is obviously legitimate for a reviewer to criticize structure, content, and style in a work under scrutiny. The reviewer's reputation alone is at risk if what he writes is inaccurate or irrelevant. When, however, the reviewer speculates about the procedures followed by an author and his publisher in order to bring a book to publication, he should take care to ascertain from a reliable source the facts of the case.

Playing and Reality is a volume to which Dr. Winnicott gave much thought during the last few years of his life. With Mr. Masud Khan and myself he determined most carefully what material should go into this book and what into a companion volume entitled *Maturational Processes and the Facilitating Environment* now published in the International Library of Psychoanalysis. Dr. Winnicott lived to correct the proofs of both books, and he himself provided the beautiful drawing that was used for the book-jacket. Furthermore, this is not Dr. Winnicott's 'final' publication. Material exists, and was discussed in great detail by Dr Winnicott, for two further volumes, which will be prepared for publication by Clare Winnicott, his widow, and Masud Khan. The rich store of his writings is not yet exhausted, though I hope that your reviewer's fantasies may be stilled or diverted by the facts I have given.

JOHN HARVARD WATTS,
Managing Director.

Tavistock Publications Ltd.,
11 New Fetter Lane,
London, EC4P 4EE.

DEAR SIR,

May I refer to the review of *Playing and Reality* which appeared in your issue of January 1972?

I am barely concerned with your critic's views of the nature of the book, for Dr. Winnicott's work will long outlast Mr. Menolascino's opinion thereof. I must, however, take objection to his statement concerning the compilation of *Playing and Reality*. I had the privilege of knowing Dr. Winnicott for a number of years and I clearly recall discussing with him in September 1970, various suggestions for a title for his book, the proofs of which he had already corrected.

I believe it fair, Sir, that your readers be made aware of the injustice and incorrectness of your reviewer's allegation on the mode of compilation of *Playing and Reality*.

HARRY KARNAC.

H. Karnac (Books) Ltd.,
56/58 Gloucester Road,
London, S.W.7.

A MODEL FOR MANIC-DEPRESSIVE PSYCHOSIS

DEAR SIR,

Court (1972) suggests a continuum model for manic-depressive psychosis with mania at the top end of the scale as the most severe form of this disease. The observations make for interesting reading but the evidence in itself is flimsy. He mentions the triangular model of Whybrow and Mendels (1969) put forward to explain the 'paradoxes' of this disease, forgetting that Baillarger, who originally described the disease in 1853, termed it 'folie circulaire'. Court puts forward nine lines of argument for his model which I should like to answer.

(1) I do not agree that a transition from depression to mania without a period of normality excludes a bipolar illness.

(2) The addition of stress to a depressed patient rarely results in mania but usually in a deepening of the depression and/or increase in agitation.

(3) The same forms of treatment do not generally prove effective in both mania and depression. It is true that tranquillizers damp down activity in both forms, but I have yet to see the effectiveness of antidepressant drugs in mania. Knowing but little about the 'blanket' effect of ECT, it is very difficult to discuss objectively its effect in manic-depressive psychosis.

(4) The occurrence of depression before, during and after manic states could support Court's model although depression after mania is rare, but in my opinion also supports a continuum model with, equal weight given to depression and mania.

(5) The existence of 'mixed states' does not disturb the bipolar model according to Kleist (1942) and Neele (1949). Leonhard (1959) explains this phenomenon by subdividing the illness into unipolar states of depression and mania, and manic-depressive psychosis.

(6) The biochemical and psychophysiological findings in manic-depressive psychosis are still in an early stage of evolution, and support for almost any model can be found. Court himself states that Whybrow and Mendels (1969) conclude that catecholamine secretion 'may reflect a general response to stress'.

(7) The severity of impairment of psychomotor performance in mania is a reflection of the severity of the condition and thus is hardly surprising. This severity supports no theory other than that most psychiatrists would rather manage a depressed patient than a manic patient. Similarly one cannot assume that leukaemia is a more severe form of hypochromic anaemia, just because it is a more severe blood disorder.

(8) The relative frequency of mania and depression is a reflection of the expression of manic-depressive psychosis. A bipolar model does not demand equal distribution.

(9) The relative infrequency of pure manic states is one again a reflection of the expression of the disease.

Court's model is an oversimplified explanation. It makes for many unanswered questions. Is a mild hypomanic state more severe than a depressive stupor? Why don't most manic states respond to antidepressant drugs?, etc. As shown in the above answers, I am not in agreement with Court's predictions which he states are necessary for a bipolar model. However, I also feel that the bipolar model does not explain all the paradoxes that occur in manic-depressive psychosis. I myself would suggest that a bi-axial bipolar model could better explain these 'paradoxes'. In this model it is suggested that there is a primary disturbance of mood along a depressive-euphoric axis and a primary disturbance of motility along a retardation-hypermotility axis. Disturbances could occur along either axis, in different directions at the same time. Thus we see manic stupor, agitated depression, hypomania etc. Support for this idea is indirectly given by Mayer-Gross *et al.* (1969) who consider involutional depression as 'depressive affect and manic hypermotility'. The bi-axial bipolar model does not explain all the paradoxes of manic-depressive psychosis, but is a model which I feel is worth further consideration.

M. H. Abenson.

Director of Psychiatry,
Kaplan Hospital,
Rehovot, Israel.

References

Court, J. H. (1972). *Brit. J. Psychiat.*, **120**, 133–41.

Kleist, K. (1942). *Nervenarzt*, **16**, 1.

Leonhard, K. (1959). In: *Aufteilung der Endogenen Psychosen.* Berlin.

Mayer-Gross, W., Slater, E., and Roth, M. (1969). *Clinical Psychiatry.* London.

Neele, E. (1949). Die phasischen Psychosen nach ihrem Erscheinungs und Erbbild.

Whybrow, P. C., and Mendels, J. (1969). *Amer. J. Psychiat.*, **125**: ii, 1491–1500.

DO MENTAL EVENTS EXIST?

Dear Sir,

May I add a further response to the article by J. J. Ray (*Journal*, February 1972, pp. 129–32), who is to be congratulated on the ingenuity of his imagination but must be criticized for his conclusions. Watson was justifiably discredited for his denial of the existence of mental events. He was, for example, unable to account for his consciousness of the non-existence of consciousness. Ray also denies the existence of mental events, but for different reasons.

His physicalistic thesis would seem to be that because every so-called mental event may have a physio-chemical counterpart it follows that mental events are identical or, as Ray puts it, 'completely interchangeable' with their physical correlates. But if they were identical the connection would not need to be established by an experiment, it would be established by logic and nothing more (1). If Ray insists that mental events are to be translated into the class of physical statements he leaves us without any way of communicating in ordinary everyday language about meanings, values, purposes and the like. In doing so he fails to accept phenomena as they are but rather dictates what they shall be. His use of the meaning of words becomes arbitrary and monopolistic.

When, for example, I say to a friend about someone else 'he came to know something', I am not ordinarily saying, as Ray states, that the other person 'had an orienting and perceptual response to a particular event that caused structural alterations in the brain'. This is not to deny that his statement can express one meaning of the phrase, but it is difficult to see why we are not allowed to have other meanings.

In contradistinction, Ray writes of his man wired up to an oscilloscope looking at a series of objects shaded blue, and noting as he looks at his oscilloscope the one brain event going on which always coincides with his seeing blue, and which never occurs without his seeing blue. He considers that all people except some philosophers would agree that this man is right and his statement accurate when he says, 'Now I know what the perception of blue is made up of.' It could be contended, however, that a more accurate statement would be if the man said, 'Now I can see and to some extent know what goes on electro-physically in my brain when I perceive blue.' To claim what Ray says is right is to limit the use of the word 'know' to nothing but representations of physical events in the brain. His thesis also, of course, reduces the personal category of the 'I' who does the seeing and knowing to a similar representation.

It might help if he did some revision on N. Hartmann's hierarchical model of the structure of know-

https://doi.org/10.1192/S0007125000001781 Published online by Cambridge University Press