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Correspondence

Psychiatrists on Popper

Sir: When a philosopher's work becomes as well known as that of Sir Karl Popper, it must be expected that it would be mis-quoted and mis-interpreted on occasions. Popper appears, however, to suffer more than most, often at the hands of psychiatrists. There have been two recent examples of this.

Taylor (Journal, July 1986, **149**, 37–41) in his fascinating article on 'Hysteria, Play-acting and Courage', uses Popper's concepts of worlds I, II and III in support of his argument that hysteria is not a mental state but a product of human culture more akin to play-acting. In Popper's terms (Popper & Eccles, 1981) this is therefore a world III phenomenon: world III being the universe of human artifacts such as novels, plays, and gardens. World III develops when consciousness or the human mind (world II) acts upon the material world (world I) to alter it in a meaningful way. Diagnosis, according to Taylor, is a world I phenomenon. This, I think, can be contested. Diagnosis is a concept and hence belongs either to world II or III. For example, in a disease (diagnosis) carcinoma of the lung, the world I phenomenon which relates to this diagnosis is the cancerous tissue itself, which, in itself, is not the diagnosis. That requires a mind (world I) before it can come into existence as a world III phenomenon. Hence, since all diagnoses/diseases are world III phenomena, the use of Popper's concepts in no way advances Taylor's argument that hysteria is different from other diagnoses, an argument which may nevertheless be correct.

The second example by Mathers (*Bulletin*, May 1986, 103–104) criticizes Popper's theory of science as it applies to psychiatry and, in particular, psychoanalysis. Popper's epistemology is a demonstration of the logical asymmetry between verification and falsification in the proof of theories. Mathers disagrees, preferring to believe in the possibility that falsifiability is not a prerequisite for testability and that theories can be *proven* to be true as well as to be false. In that case any theory which has been as decisively proven to be true as many have been proven false must be produced in evidence for this startling assertion. Of course many theories are held to be true by the scientific community because their falsifiability has been used to test them so frequently that a sort of Popperian truce has been called for the time being – but always with the possibility that an experiment at sometime in the future might disprove or modify the theory.

Mathers makes a further assumption which is attributed by implication to Popper, that non-science (psycho-analysis) is the equivalent of non-sense. Popper has pointed out that this is not the case. Indeed it would be difficult to devise an experiment to falsify Popper's own epistemological theory, but I doubt if he would accept that it is nonsense. Hence, Popper would not necessarily be led into asserting that analysis is non-sense only that it is non-science. Dr Mathers has therefore inadvertently performed for analysis more of a disservice than did Popper.

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Tardive Dyskinesia and Parkinsonism

Sir: We read with interest the letter by Drs Oyebode & McClelland (*Journal*, July 1986, **149**, 122–123). Their results coincide with our clinical observations that coexistence of drug-induced parkinsonism (PS) and tardive dyskinesia (TD) in the same subjects is not exceptional, and that the intensity of the two disorders is not correlated. Nevertheless, we do not think that 'an independence of their relative severities calls into question the currently held view that PS results from a blockade and TD a hypersensitivity of dopamine receptors'. Nor do we consider that 'the two conditions are either mediated through different dopamine systems or through independent but related neurotransmitter systems'.

Easier explanations are available. Some nigrostriatal dopaminergic regions could be hypofunctioning as a result of the neuroleptic blockade of these neurons, while other regions could have become 'hypersensitivised' after a prolonged blockage at the same time. Differing levels of receptor sensitivity in the corpus striatum have been proposed by Carlsson (1970). Moreover, observations of parkinsonian tremor shifting into a dyskinetic movement would support the hypothesis that dopaminergic hyperfunction, clinically TD, would develop after

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hypofunction produced by dopaminergic blockers, clinically PS (Goetz *et al*, 1982). A decrease of striatal dopaminergic neurotransmission by neuroleptics increases sensitivity for the posterior appearance of TD determining the later localization since it affects the same substrate and implies a topographical correlation of both disorders (Garcia Ribera *et al*, 1985). Crane (1972) showed that 'tardive dyskinesia is more likely to develop in patients with pseudo-Parkinsonian symptoms than in patients not exhibiting such manifestations'.

In similar cases, such as L-dopa induced dyskinesias in patients suffering from idiopathic Parkinson's disease, the severity of both disorders is frequently not related without calling into question the coexistence of dopaminergic hyperfunction and hypofunction. In spite of the apparently opposite theoretical support, coexistence of both disorders could be the expression of two different moments in the same physiopathological process. Prospective studies are needed to clarify how and where both drug-induced disorders appear, disappear or shift.

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Psychotherapy and Placebo

Sir: Professor Eysenck (*Journal*, May 1986, **148**, 610) says that I failed completely to understand his point. I feel that, on the contrary, it is he who has not understood mine.

I share Professor Eysenck's scepticism as to the 'specificity' of the various types of analytic psychotherapy. I have long thought that any positive or negative effects of the various types of psychotherapy have more to do with the 'non-specific' personal relationship between therapist and patient than with 'specific' factors inherent in the particular therapeutic technique employed — Professor Frank's 'shared therapeutic functions' in fact. That such factors are, to some extent, also operative in behavioural as well as analytic psychotherapy was suggested by the study of Sloane *et al* to which I referred. However, if it is the case, as Professor Eysenck states, that comparative studies show a clear superiority for behaviour therapy over placebos in general and the other psychotherapies in particular, then this is, of course, an important finding, which I never questioned.

The actual words of Professor Eysenck to which I referred and to which I objected were — 'Do we have the right to... get the State to pay us for treatment that is no better than a placebo?' My objections to this were threefold. Firstly, I wished to draw attention to the confusion that can occur in psychotherapy research because both the 'specific' treatment in question (psychotherapy) and the 'non-specific' placebo with which it is compared are both presumed to be psychological in their mode of action. Attention to the conceptual confusion surrounding the terms 'specific' and 'non-specific' in placebo theory has recently been drawn by Professor Grunbaum. His article supports my contention that the allotment of the various therapeutic factors to the categories 'specific' and 'non-specific' (or in his preferred terminology 'characteristic' and 'incidental') is purely arbitrary and relative to the theoretical standpoint of the investigator concerned. Unless we are clear about this Eysenck's statement that 'psychotherapy is no better than a placebo' could be reduced to the vacuous proposition 'psychotherapy is no better than psychotherapy.' I objected secondly because a great deal of psychotherapy is in fact done with little extra cost to the State, and thirdly because the phrase 'no better than a placebo' appears derogatory — especially insofar as people tend to associate placebos with inert pills. In his letter he says that 'placebo treatment is as successful as psychoanalysis and psychotherapy in general...' This at least implies acceptance of the fact that general psychotherapy does have some degree of success even if this is not quantitatively greater than that of placebos in general. Furthermore, the fact that the effects of psychotherapy may not be *quantitatively* greater than those of placebos in general does not, of course, imply that they are *qualitatively* identical. Few, for example, would exchange a therapeutic relationship (whether in a personal or a professional setting) for an inert pill even though both could be regarded as placebos insofar as they are 'non-specific' and psychological in their effects.

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