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CONTROLLED TRIALS OF IMIPRAMINE

Dear Sir,

Drs Rogers and Clay (*Journal*, December 1975, **127**, 599) suggest that further drug-placebo trials in endogenous depression are not justified as imipramine is of indisputable benefit in such patients who have not become institutionalized. The data presented are open to other interpretations, and the effectiveness of imipramine for the treatment of depression has still not been established beyond doubt. A suitably designed trial comparing antidepressants with placebo might still help to clarify the problem. Many psychiatrists would expect most patients (certainly over 50 per cent) with endogenous depression to get better in due course without treatment because of the natural history of the disorder. There is no indication of the length of time for which any of the patients were treated. We think the distinction between endogenous and other depressions is not so readily made as implied in the table. The great variation in percentage improvement rates in both imipramine and placebo groups of endogenous depressions needs some explanation. The criteria for rating improvement are not mentioned, and the sample sizes vary from 6 in one trial to 140 in another.

The trials analysed by Rogers and Clay form only a small proportion of the published trials on antidepressants. The method of statistical analysis does not allow for all trials to be tabulated. There are many trials in which placebo has achieved a better result than an antidepressant, and these have not been included. Also, only two trials carried out since 1966 are mentioned and it is in the first years of a drug's commercial life that favourable reports tend to be published. Some of these points concerning antidepressants have been made previously by Leyburn (1967) and by Porter (1971). It would be unfortunate if the results of this particular statistical review were accepted uncritically as evidence that imipramine is in fact so therapeutically effective.

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A MARRIAGE THAT OUGHT TO ENDURE

Dear Sir,

In his pamphlet *The Future of Psychiatry* Professor Eysenck advocates an amicable divorce between the disciplines of Psychiatry and Clinical Psychology. It is our opinion that this would not be in the best interest of the psychoneurotic patient. By arguing that psychoneurosis is behaviour largely determined by conditioning, deconditioning or failure to learn or condition, Eysenck (p 6) is stretching his stimulus-response theories just as far as he claims many psychiatrists are stretching the disease model. Recent work (Beech, H. R. and Perigault, J., 1974; Crowe, M. J. *et al*, 1972) suggests that both acquisition and extinction of morbid fears and obsessions constitute a very complex process—'it is obvious that multiple conditions are involved which interact with one another, so a satisfactory model cannot be simple', comments Marks (1975). 'Unusual states of arousal' and 'right cognitive set' are postulated, but elude precise qualitative definition. Thus the door once again opens to concepts such as idiosyncratic meaning and conflict. Many behaviour therapists, contrary to Eysenck's view, emphasize the role of cognitive factors in the cause and treatment of psychoneurosis. At this point there is a great deal of overlap between behaviour therapy and psychotherapy.

A significant proportion of our patients resist exploration so that basic drives remain unrevealed: the obstacles of denial, dissociation, projection and displacement of feeling can be formidable, and it is the psychotherapist's often slow and arduous task to evaluate and disentangle them. Such obstacles do not necessarily constitute complexes in the classical sense (Eysenck, p 17), but may represent interpersonal emotions or phobias hidden from conscious awareness and therefore not accessible to treatment until the patient can be brought into direct contact with the phobic object or situation: but if the latter remains unrevealed, unrecognized or unknown, what precisely do we help the patient to confront?

The danger of neglecting covert factors is not so much symptom replacement as resistance to treatment or only very partial improvement. In a series of agoraphobic patients, psychological gain appeared to have prevented success with deconditioning therapies in 56 per cent of cases (Shafar, 1975); psychodynamic gains operated, but many were relinquished with

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the aid of psychotherapy, leading to subsequent success with behaviour therapy. Because of the lack of theoretic application to many of a patient's problems, the need is for a combined approach to ascertain the relevant factors involved and to specify goals for treatment. The present trend is for the two disciplines to move closer together. Many psychotherapists are acquiring skills in behaviour therapy; many clinical psychologists are recognizing the role of covert factors and are inclining towards psychotherapy. Far from the psychiatrist interfering in treatment in which the psychologist is expert (Eysenck, p 18), there is little reason why a flexible collaboration cannot be created.

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THE CONCEPT OF DISEASE

DEAR SIR,

Professor Kendell (*Journal*, October 1975, **127**, 305–15) has argued the most interesting thesis that disease should be defined as that which decreases fertility and increases mortality, but excludes 'purely cultural factors determining who lives and dies'. Since man is biologically a cultural animal—his culture being a major determinant in individual and species survival—this is a curious position. Kendell is forced to the arbitrary exclusion of cultural factors because he has confused two questions. These are the scientist's question and the practitioner's question.

The scientist, the passive outsider, may ask, 'What factors reduce fertility and increase mortality?' His answer will clearly encompass cultural factors, including the efforts of doctors. The practitioner, the active insider, may ask, 'Whom should doctors treat?' The answer will depend upon doctors' competence and optimism and their given role in a community. The group treated will continually change as doctors' competence and the community change. Their role is subject to continual negotiation, as is the role of, say, psychologists, social workers and so on. The answer to the second question is specific to time, place and culture.

The answers to the two questions will *not* be the same. We may use the term 'illness' in one or other answer, or neither, just as we wish, but we may not, as Kendell does, confound the two and use a partial answer to the scientist's question to try to answer the practitioner's question. Logically it is wrong, practically it could be disastrous.

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DEAR SIR,

Professor Kendell's address (*Journal*, October 1975, **127**, 305) encourages us to rethink our concepts of disease. Briefly, he finds it difficult to define disease and advocates in its place the concept of 'biological defect'. While appreciative of his thoughtful contribution, I am more in agreement with the customary definitions of disease and the morbid process than with his position; the customary definitions are rarely challenged by their critics, they are simply ignored.

Disease stands for 'absence of ease' (Oxford English Dictionary)—the patient's subjective awareness that there is something wrong, covered by the clinician with the term 'symptom'. The lack of ease, or symptom, is the discerned result of the underlying morbid process. The patient is usually, but not always, aware of his disease; discernment is increased by screening devices. The symptom must not be confused with the underlying morbid process.

The morbid process of disease is well defined in most adequate medical dictionaries (e.g. Butterworths). It results essentially from one or more noxious agents acting on a structure, setting up dysfunction in it, and releasing coping devices to restrict and repair the damage, which, if they fail, cannot be prevented. The power of the coping devices varies with individuals and populations. The noxious agent can be psychic or somatic; the structure can be the psyche or the soma; the morbid process can be psychic or somatic. Indeed psychic trauma can lead to somatic

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