authors who have reviewed the evidence and come to favourable conclusions about the effects of psychotherapy are unacceptable, pointing out the many errors of omission and commission characteristic of this type of work. Bloch & Lambert are certainly right in saying that "psychotherapy requires an answering commitment to its intelligent and rigorous study, as well as the exploration of new paradigms for research." This aim is hardly furthered by optimistic and unwarranted conclusions about the effectiveness of psychotherapy as it is practiced at the moment, or the failure to look at the facts as they really are. The question I raised 30 years ago (Eysenck, 1952) concerning the effectiveness of psychotherapy cannot be so easily swept under the carpet. The negative conclusion I came to then is still not contradicted by any facts I know of. Only behaviour therapy has succeeded in clearly beating bogey, but traditional psychotherapy has still failed to demonstrate its superiority to placebo treatment, and psychoanalysis in particular has been shown to have frequently very detrimental effects on the mental health of patients (Strupp *et al*, 1977). There clearly is very little ground for optimism in all this!

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References

EYSENCK, H. J. (1983) Special review of M. L. Smith, G. V. Glass and T. I. Miller: The benefits of psychotherapy. *Behaviour Research and Therapy*, **21**, 315–320.

RACHMAN, S. J. & WILSON, G. T. (1980) *The Effects of Psychological Therapy*. London: Pergamon Press.

SMITH, M. L., GLASS, G. V. & MILLER, T. I. (1980) *The Benefits of Psychotherapy*. Baltimore: John Hopkins University Press.

STRUPP, H. H., HADLEY, S. W. & GOMEZ-SCHWAARTZ, G. (1977) *Psychotherapy for Better or Worse: The Problem of Negative Effects*. New York: Jason Aronson.

British Journal of Psychiatry (1985), **146,** 557–562

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CIRCADIAN RHYTHMS

DEAR SIR,

Roy-Byrne *et al's* paper on 'Approaches to the evaluation and treatment of rapid cycling affective illness' (*Journal,* November 1984, **145,** 543–550) was of great interest in the way it was able to draw together the research findings and weld them into an approach to clinical evaluation of the individual patient. Theoretically this is a very important group of patients and so it was with some concern that I noted an apparent error in the theoretical section of the paper. Tricyclic antidepressant drugs are stated to be similar to oestrogen in their ability to "accelerate the frequency of free running circadian rhythms" (Wirz-Justice *et al,* 1980). As can be seen from the reference (which is reproduced below) the paper which was quoted, in accordance with most of the other literature, (Thompson, 1984) actually shows a slowing of circadian rhythms under free running conditions. This error rather detracts from the theory, which is suggested later in the paper, that rapid cycling illness is due to an unusually short intrinsic period, or the induction of a short period by tricyclic antidepressants.

In the same issue Drs Nair and Hariharasubramanian (*Journal,* November 1984, **145,** 557) criticise my recent review of circadian rhythms for leaving out studies of the melatonin rhythm in depression. In this context it is necessary to distinguish clearly between a reduction of melatonin secretion (Beck-Friis *et al,* 1984; Claustrat *et al,* 1984) which does not necessarily have any implications for an underlying circadian abnormality, and a phase shift in secretion which does. Dr Nair refers to a study which shows the onset of the melatonin rhythm to be delayed in depressed patients (Nair *et al,* 1984). I look forward to reading the definitive report of this work (at present the reference is to an abstract) and at this time would only comment that the result is contrary to other published data which purport to find a phase advance of melatonin (Lewy, 1983). Our own work (Thompson *et al,* 1983, 1985) has found no phase shift in depressed patients, either before

treatment, compared with normal subjects, or during treatment with antidepressants.

In the light of these findings perhaps the decision to leave out a detailed discussion of the melatonin literature from the review of circadian rhythms is more comprehensible.

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References

BECK-FRIIS, J., VON ROSEN, D., KJELLMAN, B. F., LJUNGGREN, J. G. & WETTERBERG, L. (1984) Melatonin in relation to body measure, sex, age, season and the use of drugs in patients with major affective disorders and healthy subjects. *Psychoneuroendocrinology*, **9**, 261–277.

CLAUSTRAT, B., CHAZOT, G., BRUN, J., JORDAN, D. & SASSOLAS, G. (1984) A chronobiological study of melatonin and cortisol secretion in depressed subjects: plasma melatonin, a biochemical marker in major depression. *Biological Psychiatry*, **19**, 1215–1228.

LEWY, A. L. (1983) Mammalian melatonin production: *The Pineal Gland* (ed. R. Relkin). Elsevier: Amsterdam.

NAIR, N. P. V., HARIHARASUBRAMANIAN, N. & PILAPIL, C. (1984) Circadian rhythm of plasma melatonin and cortisol in endogenous depression. *Journal of Steroid Biochemistry*, **20**, 1460 (abstract).

THOMPSON, C., MEZEY, G., CORN, T., FRANEY, C., ENGLISH, J., ARENDT, J. & CHECKLEY, S. A. (1985) The effect of desipramine upon melatonin and cortisol secretion in depressed and normal subjects. *British Journal of Psychiatry*. (In press).

—— (1984) Circadian rhythms and psychiatry. *British Journal of Psychiatry*, **145**, 204–206.

—— CHECKLEY, S. A., CORN, T., FRANEY, C. & ARENDT, J. (1983) Down regulation at pineal B adrenoceptors in depressed patients treated with desipramine? *Lancet*, *i*, 1101.

WIRZ, JUSTICE A., WEHR, T. A., GOODWIN, F. K., KAFKA, M. S., NABER, D., MARANGOS, P. J. & CAMPBELL, I. C. (1980) Antidepressant drugs slow circadian rhythms in behaviour and brain neurotransmitter receptors. *Psychopharmacology Bulletin*, **16**, 45–52.

Dr Roy-Byrne and colleagues reply

We want to thank Dr Thompson for pointing out our error. He is correct, of course, in affirming that tricyclic antidepressants slow the frequency of free-running circadian rhythms. Therefore, the effect of oestradiol on circadian rhythms is *opposite* to that of tricyclics. We hope that this theoretical error has not detracted from our attempt to provide a practical overview of the clinical management of the rapid cycling patient.

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TREATMENT OF RAPID CYCLING AFFECTIVE ILLNESS

DEAR SIR,

In their paper Roy-Byrne *et al* (*Journal*, November 1984, **145**, 543–550) indicated a need for further systematic study to assess the definitive role of neuroleptics in treating this condition. The depot preparation of haloperidol decanoate became available in the U.K. on a research basis in the autumn of 1981 and my clinical experience to date includes the treatment of fifteen patients with a diagnosis of manic depressive psychosis, four of whom form a distinct subgroup of rapid cycling affective illness. They have a combined history of eleven hospital admissions for hypomania in the previous two years while on lithium (serum levels 0.50 to 1.0 mmol/l) and oral neuroleptics. These frequent admissions resulted in a total of twenty three months in-patient treatment (almost six months per patient) before starting haloperidol decanoate in a dose range between 100 and 400 mg monthly. There have been no re-admissions for hypomanic relapses, all four patients having received this treatment for over 36 months, and two short lasting depressive episodes in one patient were successfully treated as an outpatient by adding a tricyclic antidepressant. The result of this open study certainly supports the authors' proposition that prolonged neuroleptic treatment, possibly in combination with lithium, may reduce the severity and frequency of manic episodes.

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IS THERE REALLY A SCHIZOPHRENIA? THE LONG-TERM COURSE OF PSYCHOTIC PHENOMENA

DEAR SIR,

As my article (*Journal*, December 1984, **145**, 636–640) could not be published in full, some additional points, included in the original manuscript, may be helpful for understanding my thesis. According to systems theory, identical states may be reached by way of very different combinations of influencing factors (the principle of equifinality). On the other hand, identical states can evolve, under varying circumstances, in very different directions. Both phenomena are currently observed in the long-term course of psychotic states diagnosed as schizophrenia. With other arguments presented in the paper, this speaks against the classical concept of a clearly delimitable disease entity with constant causes, psychopathological picture, and course. A more flexible view, based on the vulnerability- and