comparison of the timing of melatonin secretion. Finally, the depressed patients, but presumably not the control subjects, were given benzodiazepines. Kabuto *et al* (1986) recently reported suppression of the nocturnal melatonin surge by benzodiazepines in normal subjects. Although this observation must be taken cautiously, because the reported plasma melatonin concentrations are much higher than in most laboratories (Arendt, 1985), the potential effect of benzodiazepines on melatonin secretion should not be ignored. One might even speculate that these drugs could have a paradoxical effect, increasing melatonin concentrations in depression and thus explaining Thompson's results.

In conclusion, the relationship between melatonin secretion and depression remains undetermined.

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SIR: Dr Cavallo's letter raises three interesting questions about our study. Firstly, the relationship between a family history of depression and abnormalities in melatonin secretion is speculative. We tested the more cautious hypothesis that low melatonin secretion would be a state marker of depression. Since we were able to find no evidence in support of this, it would be illogical to search for an association with family history (i.e. genetic marker status).

Secondly, melatonin is not a sleep-related hormone. The sleep-wake cycle should not be equated with the light-dark cycle, since the two cycles may clearly dissociate from each other. Since we controlled for month of testing, the light-dark cycle was not a confounding factor.

Thirdly, the question of benzodiazepine use appears to strengthen the findings rather than diminish them. It is speculation indeed to suggest a paradoxical effect of benzodiazepines on melatonin in depression, one for which we know of no evidence. We agree that the relationship between melatonin secretion (volume, timing, and suppression by light) and depression remains of great interest, and there are still a number of important hypotheses to be tested. However, we also believe that it is important in such studies to control for all the relevant variables as we have endeavoured to do. We will be lead to doubt our study's findings if mistakes in design can be levelled at it rather than speculation.

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Self-Esteem: A Psychiatric View

SIR: Robson (*Journal*, July 1988, **153**, 6–15) mentions some of the views on self-esteem taken by cognitive therapists. While referring to the work of some rational emotive therapy (RET) therapists, he fails to mention the current position of self-esteem in RET theory, namely that self-evaluation, whether positive or negative, accurate or distorted, is a source of emotional disturbance. Ellis has delineated this position on many occasions (e.g. Ellis, 1972; Ellis *et al*, 1975).

Briefly, RET theory regards self-esteem as the individuals' rating of self as being either good or bad, based on the presence or absence of certain traits, behaviours or attributes. It regards such evaluation as irrational and self-defeating on the basis that human beings are simply too complex to be accurately rated. Ellis recommends that people rate only their abilities to perform specific tasks and give up rating themselves completely. He advocates that people accept themselves, *a priori*, as fallible human beings who like all human beings do some things particularly well, some things poorly, and many things adequately.

The confusion in the literature concerning the definition, measurement, and relationship to mental illness of self-esteem, to which Dr Robson draws our attention, would tend to support Ellis' contention that humans are too complex to logically rate their own worth.

I would wish those still seeking a scientific measurement of how people perform this illogical evaluation good luck in their task.

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https://doi.org/10.1192/S0007125000223957 Published online by Cambridge University Press

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Depersonalisation and Self-Perception

SIR: I found Mellor's discussion (*Journal*, July 1988, **153, Suppl. 2,** 15–19) of the phenomenological philosophy of depersonalisation both interesting and disturbing. Having pointed out how some great men, such as Schilder and Schneider, may fail to agree on certain fundamental aspects of interpretation, he goes on to offer a "tentative proposal that two forms of depersonalisation exist", a malignant, dysphoric variety, and a benign, possibly protective type. Surely this proposal fails to take the reactions of the subject into account? I have seen patients who have been given morphine react differently, with euphoria or with dysphoria, depending on a number of circumstances, not the least of which was the presence or absence of pain respectively. This reaction is also modified by the duration of exposure to opiates (O'Shea & Falvey, 1988).

If a subject experiences dizziness as a result of a brain tumour or because of beta-blocking drugs, is the experience qualitatively different of necessity? Again, surely, one factor determining those other-than-lost emotional reactions (Schneider, 1959) is the patient's interpretation of the significance of the phenomenon? My own viewpoint, be the setting delusional or otherwise, is that of Mayer-Gross (1935) and some more recent observers (for example Cohen, 1988): i.e. that the experience is probably a physiological event, complicated by factors such as genetics, personality, biochemistry, structural change, environmental circumstances, and so on. Philosophy and psychoanalysis fail to provide uniform explanations simply because of this complexity.

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Lithium-Induced Carpal Tunnel Syndrome

SIR: With reference to the article by Deahl (*Journal*, August 1988, **153,** 250–251), we should like to point out that this is not the first report of lithium-induced carpal tunnel syndrome, as stated. We reported just such a case in these very correspondence columns two years ago (*Journal*, September 1986, **149,** 386–387).

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The Truth About ECT

SIR: At the end of their interesting review on the use of electricity in the treatment of mental illness, Drs Beveridge & Renvoize (*Journal*, August 1988, **153,** 157–162) bring in the practice of ECT. Strictly this is irrelevant to their subject and simply serves yet again to perpetuate misunderstanding of the nature of ECT. ECT is in no sense electrical *treatment* or electrotherapy, but only the use of an electrical stimulus instead of a pharmacological one (subcutaneous insulin, intravenous metrazol, or inhaled flurothyl) to set off an epileptiform disturbance in the brain; it is this disturbance which is therapeutic. We do not talk of the motor car as an electro-automobile because it has spark plugs, or of electro-central heating because an electric pump shifts the hot water. We do not pit the mysterious force of electricity against (mysterious) mental illnesses, as a hostile lay public may believe, nor (with muscle relaxants) should there be any convulsion (unpleasant word). So electroconvulsive therapy as a name has all the wrong associations and helps to perpetuate the bad image of the treatment. A more accurate name would be relaxant ictal therapy (RIT), which would be better for public relations. As for shock treatment, it does not mean, as some suppose, electric shock treatment like the painful tingling from a shocking coil or worse from the mains, nor surgical shock, nor emotional or physical shock (as given to the mentally ill in the past with the whip, the ducking stool, or the release of snakes in the dark). The word shock was introduced by Sakel to express the fairly fast action and non-specific nature of the effects of insulin therapy, and got carried over into pharmacological treatment.

Many authors writing briefly about the origins of ECT link it as Drs Beveridge and Renvoize do with the history of electrobiology, or with old practices of fright and torture. But the roots of its discovery are

https://doi.org/10.1192/S0007125000223957 Published online by Cambridge University Press