active drugs made any significant difference on the results in our trials. Thus, there was no significant difference in results between the groups of patients who received phenobarbitone first or nitrazepam first. As to the possibility of rebound effects interfering with sleep on placebo nights, re-examination of our data does not lend support to the assumption that such an effect should be more marked after phenobarbitone than after nitrazepam.

Dr. Oswald comments at some length on the presumed non-validity of the comparison between active drugs and placebo. Although he makes the erroneous assumption that the night nurse, who made the objective rating, also gave the sleeping pills and knew the different appearance of active tablets and placebo tablets, we agree that the comparison between placebo and active drugs was not quite valid, and we believe we made this point quite clear in our paper. We had no intention of making a valid comparison between active drugs and placebo; our primary aim was to compare phenobarbitone and nitrazepam. We consider it amply proven by numerous previous trials that both drugs are better than placebo. As already stated, our main reason for using placebo was to have a wash-out period in between the drugs; the first placebo night also served as a crude control of the patient's sleeping pattern.

Dr. Oswald objects to our use of the term 'quality' of sleep. However, we think it appears from the context in our paper that this term was only used as a reference to those aspects of sleep, rather than the mere length of the sleeping period, which may have some influence on the patient's own judgement of whether he has slept well or badly. We think common clinical experience indicates that such a term, even if not precisely defined, is warranted.

Our main conclusions were, firstly, that the active drugs did not differ significantly in their over-all effect on sleep, and secondly, that there was a highly significant tendency for patients complaining of early insomnia because of disturbing thoughts to report better sleep after phenobarbitone than after nitrazepam. We do not see that Dr. Oswald's comments make these conclusions invalid.

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'PARASUICIDE'

DEAR SIR,

So far the score on 'parasuicide' as an alternative term to 'attempted suicide' (proposed by us in your columns, June 1969) is two-nil against us. Dr. Merskey (October 1969, p. 1227) and Professor Stengel (February 1970, pp. 237–8) both vote for continuing the status quo. Both seem to us to have missed our main contention, which is that 'attempted suicide' continues to cause untold mischief and confusion, especially among general practitioners and other non-psychiatrists. Not unreasonably these colleagues assume that the term means what it appears to mean, namely an attempt at self-destruction. Consequently we still hear of patients not receiving psychiatric assessment because they were 'only making a gesture' or the like. The importance of this issue is not that our terminological sensibilities are outraged, but that patients suffer. The matter is a serious one, and we submit that some alternative to existing nomenclature *must* be found. Our critics contribute nothing to this task.

An awareness of the urgency of the problem led Professor Kessel to propose 'deliberate self-poisoning or self-injury'. While we endorse some of the reasoning behind his suggestion, the choice of term seems to us unfortunate for the reasons set out in our previous letter. In particular, dropping all reference to suicide seems regrettable; hence our own proposal. It is ironic that we are then attacked for being insufficiently alert to the overlap between suicide and parasuicide, even though we are proposing the former as the model which the latter simulates. It becomes bewildering when we are also accused of failing to recognize the complexity of motivations underlying suicidal behaviour, of denying the element of gamble and of relying exclusively on the patient's stated intention. Nothing we have written justifies such comments.

It is difficult to comment briefly on the numerous other issues raised by Professor Stengel. Of course we accept that the usual legalistic concept of suicide is too narrow, and that self-inflicted deaths often bear elements of a desire to survive. Among parasuicides, however, the ambivalence is not, in our experience, usually related to living or dying so much as to a mixture of other seemingly incompatible motivations such as hostility against, and appeal to, a spouse. Often the patient has little wish, overt or covert, to die, but may want others to consider him as one who has been driven to desperate measures. We note Professor Stengel's *new* definition of suicidal behaviour, but doubt if it can be used operationally, for all his and Dr. Merskey's concern for the difficulties of the epidemiologist.

Although words and ideas are mutually dependent, our primary aim is to advocate not so much a new concept as a change in terminology. We are proposing that those patients currently labelled as 'attempted suicides' should receive a less confusing designation. Conundrums on points of theory, however important and intriguing, can afford to wait; clinical needs cannot.

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EFFECTS OF LITHIUM ON PLASMA MAGNESIUM

DEAR SIR,

The recent work of Frizel *et al.* (*Journal*, December 1969, p. 1375) on plasma levels of magnesium and calcium in depression contains some very interesting results which are worthy of some further consideration. Calculation from the published data of the 'non-ionized' fraction of the metals indicates that there is a trend for the mean 'non-ionized' calcium to be higher in the depressed state than in the normal, recovered or lithium treated states. There is also a converse trend for the 'non-ionized' magnesium to be lower in depression than in normal or recovered states. However, after lithium treatment the 'non-ionized' magnesium is decreased by about 50 per cent. This suggests that lithium medication and spontaneous recovery result in opposite changes in 'non-ionized' plasma magnesium.

Some current theories on the mode of action of lithium assume an interaction with sodium. From a chemical point of view, lithium can also be considered to be very closely related to magnesium and calcium, owing to the so called 'Diagonal Relationship'. Lithium is in the typically anomalous position of a first row element in the periodic table. In this position group relationships are weaker, and diagonal affinities with the next group are relatively stronger. This effect is shown in both chemical and physical properties. From the chemical standpoint it could therefore be postulated that the normothymotic effect of lithium (Schou, 1968) is due to an interference with the metabolism or binding of magnesium. The logical corollary is that defects in magnesium metabolism could be involved in the pathophysiology of the manic-depressive syndrome. The central role of magnesium in energy-producing enzyme reactions, such as ATP-ATPase interactions, could indicate that this defect might be of a fundamental nature leading to the changes in other parameters already clearly documented.

From the view point of these hypotheses it is interesting to deduce the possible reason for the reduction in the 'non-ionized' fraction of plasma magnesium after lithium treatment. Since magnesium is involved in the structural stabilization of proteins, (Wacker, 1969), it is possible that lithium, with its similar crystal ionic radius and polarizing power, could insinuate itself into the same or similar sites in the structure. In this case, the non-ionized magnesium fraction will decrease and the plasma ionized magnesium fraction will increase due to released Mg^{2+} ions The free excess ions would be removed by the homeostatic mechanisms controlling plasma ionized magnesium, and would either be excreted or transferred to intracellular or bone compartments.

In this laboratory we are undertaking experiments to determine the effects of lithium on the distribution and metabolic balance of other ions. Preliminary results indicate that during lithium treatment of rats the daily urinary excretion of magnesium rises, but returns to about normal levels on cessation of lithium administration. This finding has been confirmed by Gotfredsen *et al.* (1969).

If in fact lithium can be shown to compete with magnesium at sites in some phase of plasma or elsewhere, a useful pharmacological model would be available for the evaluation of the role of magnesium in psychophysiology.

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ECT

DEAR SIR,

Literature on ECT is becoming an ever increasing dreary repetition of past work, or 'abusing the plaintiff's attorney' with great statistical expertise.