is far from simple as Garside appears to suggest. It is a problem that cannot be discussed here. It has been discussed at length in the papers by Binder (1963), Edwards (1965), Grant (1962), and Wilson and Miller (1964).

Until further data are available one must make a decision as to whether or not sleep patterns are to be considered a valid differentiating feature. In cases such as this it is probably advisable to accept the null hypothesis, albeit tentatively. It may be mentioned here that Type I errors—rejection of a true null hypothesis—are probably more serious than Type II errors, and, as Edwards (1965) has pointed out, the problem with classical significance tests is that they " . . . are violently biased against the null hypothesis".

Let us suppose we were to continue to use reports of sleep patterns as diagnostic indicators. Of course, no clinician would depend solely on one such feature. But since sleep pattern data have probably equal weight to other data in deciding between the two types of depression we are justified in examining them alone. Taking the data from our study concerning reports of initial insomnia at home we find that 53 per cent. of the cases would be diagnosed correctly. The data on early morning awakening at home would result in 41 per cent. correct diagnosis. Now such data are not too meaningful without base rate data, which are not available for the area from which our sample of patients is drawn. Kiloh and Garside (1963) have presented data indicating that in a survey of 2,104 depressives in the North-East of England, 63 per cent. were diagnosed endogenous depressives and 37 per cent. reactive depressives. If the base rates are similar for Saskatchewan, then it can be seen that one would make more correct diagnoses by calling all of the patients endogenous depressives.

Dr. Garside has examined in detail the data on the reports of patients concerning their sleep the first night in hospital. Comparing the reactive and endogenous depressives, this results in a between-groups difference of 21 per cent. for initial insomnia and 17 per cent. for early morning awakening. This may, particularly with a standard error of 16 per cent., make some people a little wary of accepting the null hypothesis. But if we look at the data concerning sleep at home we find a difference of 1 per cent. for initial insomnia and 3 per cent. for early morning awakening. These figures are not at all impressive, and though we may note in Garside's vein that with an error of 16 per cent. the true difference may be considerably larger than 1 per cent. or 3 per cent. *it may also be considerably less*—a true difference that is quite contrary to clinical prediction!

(7) Whatever may be the case regarding the *reports* of patients, objective data on sleep patterns reviewed by us and our own data on nurses' observations strongly suggest that there is no actual difference between the two groups of depressives in their sleep patterns. Those who would suggest that the two groups of depressives do differ in their reports about their sleep patterns must demonstrate that this is so on the basis of objective, uncontaminated data.

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ANTI-BARBITURATE EFFECTS OF BEMEGRIDE

Dear Sir,

May I criticize the recent paper by Orwin, Sim and Waterhouse (June 1965, pp. 531–533)?

Using EEG studies as a criterion of sedation, the authors found no significant difference between intravenous amylobarbitone sodium alone and intravenous amylobarbitone sodium combined with 10 per cent. bemegride *at therapeutic doses*. Using slurring of speech as a criterion of sedation, the authors noted a statistically significant difference between the sedative effect of intravenous amylobarbitone sodium alone and "bemegrated" amylobarbitone sodium. However, they considered the difference of no clinical importance. Although the authors do not mention as much, these results confirm the sedative effect of the combination in therapeutic doses.

Orwin and his colleagues have extrapolated from data obtained at therapeutic levels to draw conclusions at toxic levels. Ignoring the work of Trautner, Murray and Noack (1957), Orwin *et al.* have drawn conclusions based on the assumption that the dose response curves of (i) amylobarbitone and (ii) the combination run parallel throughout their range. This may not be so, and there is some evidence to the contrary.

Trautner *et al.* (1957) used oral doses of barbiturate containing 12 to 23 per cent. bemegride, and found that up to a barbiturate intake of 750 mg. there was no effect on onset, depth or duration of sleep. The mixture acted exactly as the same amount of the barbiturate alone. At a barbiturate intake of between 1 and 1·5 grammes, however, the duration and depth of sleep were greatly reduced as compared with the effect of the same amount of barbiturate. At a barbiturate intake of between 1·5 and 3 grammes subjects either slept or were merely somnolent for a few hours.

Orwin *et al.* also fail to mention that there have been no fatalities reported with the combination tablets containing amylobarbitone 100 mg. and bemegride 10 mg. in each (Mylomide) since their introduction.

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REFERENCE

TRAUTNER, E. M., MURRAY, T., and NOACK, C. H. (1957). *Brit. med. J.*, *ii*, 1514-1518.

DEAR SIR,

We were familiar with the paper by Trautner *et al.* (1957) which is quoted by Dr. Neville, but did not see fit to take it into account in our study. Superficially, this work does suggest that bemegride is relatively more effective in larger doses, but Dr. Neville does not point out that the authors were studying the value of bemegrated barbiturate as a hypnotic in disturbed and chronic schizophrenic patients. The assessment of sedative effect was a subjective one in the control group, while in the patient group the criteria were clinical, namely, the dose required to render a disturbed patient tranquil and asleep. Our study used the objective evidence of the inflection point in the EEG graph, which in turn was matched with slurring when the patient was used as his own control.

The schizophrenic patients who comprised the major study had a 2–5 years' history and had been subjected to a variety of treatments, "several" having had continuous narcosis with barbiturates. None had responded to the ordinary doses of sedation, having had continuous narcosis with barbiturates. and the authors were searching for a method whereby they could prescribe even larger doses without the risk of severe poisoning.

Our criticisms of this work are:

(1) Many of the patients had had barbiturates over the years and in heavy doses with probable development of tolerance. This could have rendered them relatively more sensitive to the bemegride.

(2) The response of chronic and disturbed schizophrenic patients to barbiturates is notoriously difficult to assess. For example, a catatonic patient may respond to 0·5 grammes of intravenous barbiturate with remission of the psychotic features but little drowsiness. The authors did not demonstrate whether the sleep their patients enjoyed was due to the sedative effect of the barbiturate or to the amelioration of the psychotic process.

(3) Subjective tests and clinical observations are not as reliable as EEG studies. We tried to assess the duration of sedation on the patient's return to the ward, but had to abandon it because of the many variables involved, one being observer error.

(4) The authors used oral preparations, while we used the intravenous route. The latter does ensure that the drugs are in the blood stream, which we felt was important in a scientific study. It also eliminates the possibility of uneven absorption and produces a speed of reaction which is more readily observed, and the blood concentration of barbiturate approaches that of the toxic doses used by Trautner *et al.* (1957).

(5) The patient's mental state can influence the amount of barbiturate required to produce sedation, and with disturbed schizophrenics a constant baseline would be very difficult to obtain.

While Trautner *et al.* (1957) indicate that their selected patients were less drowsy with bemegride, we frankly do not know what conclusions can be drawn from this work. The absence of a report of suicide with bemegrated barbiturate is of interest, but must be correlated with the population at risk, which is probably small, and with the type of patient who has the drug prescribed, who may be addicted. The suggestion that the dose response curve does not run parallel throughout the range is an interesting one which still awaits proof.

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THE COUVADE SYNDROME

DEAR SIR,

Couvade, though recognized by the psychiatrist, is less well known to the general practitioner, who

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