the "Severely Subnormal", deprived of adequate facilities for investigation, treatment and research. This retrograde separation of a section of the retarded population from the mainstream of optimistic, forward-looking activity of the large, comprehensive Mental Deficiency Hospitals is a direct result of assuming that there is some fundamental difference in therapeutic opportunity between the two grades of patient. The danger, which the authors point out, that a patient might be denied appropriate treatment and training because he had been misclassified and sent to the wrong hospital is best prevented by having comprehensive hospitals with no dichotomy. Even maximal discrimination between categories will always result in some error—with personal tragedy for the unfortunate individual. The traditional unified hospital service under one clinical team denies facilities to none, and produces the greatest ease of transfer and flexibility in the training programme. The Ministry itself is confused, for the arguments it gives in favour of District General Hospitals are the exact opposite of those advanced for the fragmentation of Mental Deficiency Hospitals.

In short, Heber's ceiling for intellectual deficit at −1 S.D. is more realistic than that of Castell and Mittler; legal terminology should not be used for clinical practice or planning clinical services; nomenclature should be precisely used after definition for a specific objective if the inherent technical difficulties in Mental Deficiency are not to be compounded and confounded by semantic promiscuity.

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SLEEP PATTERNS AND REACTIVE AND ENDOGENOUS DEPRESSIONS

Dear Sir,

In their interesting paper (*Journal*, June 1965, pp. 497–501) Costello and Selby criticize the findings of Kiloh and Garside (1) on the grounds that they "may simply reflect the knowledge [i.e. of clinical tradition] and need to arrive at a diagnosis of the clinicians producing the case histories", but do not say how their own "independent interviewer" approached the problem of differential diagnosis.

If their interviewer employed a relatively simple, single criterion, such as the presence or absence of an environmental precipitant, then it is not surprising that Kiloh and Garside's findings were not fully borne out. For these authors did not use any single criterion, but diagnosed their cases on the basis of the feature-pattern as a whole (a common procedure in psychiatry). Their subsequent statistical analysis showed that the clinical differentiation of the two syndromes arrived at by this means was not arbitrary or intuitive, but in fact corresponded with the mathematical composition of the matrix of intercorrelated items. "Precipitation" was only one item among many, and its correlation with diagnosis fell well short of unity (0·654).

If, on the other hand, Costello and Selby's interviewer himself took account of a number of features, then we need to know about *his* attitude to traditional views, and in particular, what importance, if any, he attached to the sleep pattern? Also, to what extent may he have been influenced by knowledge of the investigation being carried out on his patients? All these factors could have affected the final groupings. Indeed, if Costello and Selby are right and clinicians' observations are too fallible to lead to reliable diagnoses, then it seems doubtful if their own study justifies any conclusions about the sleep patterns in so-called reactive and endogenous depression.

Actually, one of the purposes of Kiloh and Garside's study was to put diagnosis in depression on a surer footing by studying the frequency and inter-relationships of individual symptoms. As they point out, the clinical diagnosis, although made in every case, was doubtful in 51 out of 143, presumably because the feature-patterns were not sufficiently clear-cut for a confident clinical judgment; it does not seem, therefore, that much "reinforcing desired responses" from the patient actually took place. Nevertheless, all cases were included, and their analysis showed that the data must be due to two separate factors, interpreted as a general illness factor and a bipolar factor corresponding to neurotic versus endogenous depression. Costello and Selby, it may be noted, omitted 32 of their 73 cases for reasons that are not stated.

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Reference

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Dear Sir,

I wish to make a number of points in relation to the letters of Drs. Kay (above) and Garside (*Journal*, August 1965, p. 773):

(1) Both Dr. Kay and Dr. Garside wish to know how the diagnosis of reactive or endogenous depression was arrived at in our study (Costello and Selby, 1965), and rightly so. It was an unfortunate omission. The diagnosis was arrived at by the independent interviewer—a psychiatrist—on the basis of the feature pattern as a whole. This is, of course, as Kay notes, " . . . a common procedure in psychiatry". Our cases would therefore appear to be comparable to those of Kiloh and Garside (1963).

(2) The independent interviewer considered sleep patterns to constitute an important differentiating feature for the two groups of depressives. However, the question concerning this problem which was put by Dr. Kay does not appear to be a very important one, since even were the independent interviewer to have excluded data on sleep patterns in arriving at his diagnostic decisions, sleep patterns, if important, should still have been revealed as such. As it was, no effort was made to exclude consideration of sleep patterns in the diagnostic decisions so that the dice was loaded in favour of positive results. There is of course no direct evidence concerning the extent to which the interviewer was influenced by knowledge that a research project was in operation. On the other hand, he was not told of the purpose or details of the project until after its completion. Dr. Kay appears to be searching for contamination in our data. If there is any contamination it is probably slight, whereas in the data of Kiloh and Garside (1963) it is probably considerable.

(3) Dr. Kay suggests that if " . . . clinicians' observations are too fallible to lead to reliable diagnoses, then it seems doubtful if . . . Costello and Selby's . . . study justifies any conclusions about the sleep patterns in so-called reactive and endogenous depressions". But we did not intend to provide sleep pattern data on reliable and valid categories of depression. Our intent was to compare sleep pattern data obtained from case histories (Kiloh (1963) has confirmed that this was the procedure in his study with Garside) with sleep pattern data obtained in standardized interviews. The former method of collecting data used in Kiloh and Garside's study results in substantiation of clinical prediction, whereas the latter method used in our study does not result in such substantiation. It should be emphasized here that we were not questioning the validity of the distinction between the two types of depressives, but rather the validity of sleep patterns as a differentiating feature between the two types.

(4) Dr. Kay's statement that " . . . the clinical differentiation of the two syndromes . . . was not arbitrary or intuitive, but in fact corresponded with the mathematical composition of the matrix of intercorrelated items", is quite true, but then we did not suggest that the clinical differentiation was arbitrary or intuitive. On the contrary, we suggested that it was done in relation to the established criteria of clinical tradition. Thus clinical tradition shaped the collecting of the case history data and the diagnosis. It would follow that statistical analysis of the case history data would substantiate the clinical differentiation. This is the old problem of getting out of factor analysis what you put into it. All this does not, of course, rule out the possibility that analysis of *uncontaminated* data would confirm the clinical differentiation.

(5) Since, as Dr. Kay points out, 51 of Kiloh and Garside's cases were considered doubtful in terms of diagnosis, the biased questioning and reinforcing of desired responses which we suggest may occur in the clinical interview, clearly does not result in clear-cut case histories and diagnoses in all cases (some patients may resist the interviewer!). But this does not rule out the possibility that it is this that is happening in the remainder of the cases or that the decisions, doubtful though they may be, for the 51 cases are due to a certain degree to such interviewer behaviour. With regard to the omission of 32 cases in our study, these were patients who, though interviewed for sleep data by Selby, were not subsequently diagnosed as depressives by the independent interviewer.

(6) Dr. Garside's main criticism is a more problematical one. He suggests that Dr. Selby and I have committed what is known as a Type II error—the failure to reject the null hypothesis when it is false. He talks of this error initially in relation to the data from the nurses' observations, but only goes into detail about the error in relation to the interview data, presumably since these data are more vulnerable—the differences being in the direction predicted by clinical tradition. It will be remembered that the differences in the case of the nurses' observations were not only non-significant but in a direction *contrary* to that predicted by clinical tradition.

Dr. Garside rebukes us because we "use non-significant results in sleep patterns to confirm the null hypothesis that 'reactive and endogenous depressions do not differ in sleep pattern' ". We did not talk about confirming the null hypothesis. Dr. Garside, of course, leaves the word "confirm" outside the quotation marks. Then again the supposed quotation does not appear in our paper! It is true that the data from the standardized interview are consistent with the null hypothesis. It is also true that in one sense the data are consistent with the hypothesis that sleep patterns do differ between the two depressive groups. Non-significant findings present us with a problem that

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.