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PREMENSTRUAL SYNDROME

DEAR SIR,

Anthony Clare's (*Journal*, January 1981, **138**, 82–83) reservations about sine wave fitting to daily recorded symptom scores to assess and diagnose 'premenstrual' tension, and his preference for a polynomial fit and F test, set us to work on that comparison. We concluded that Gödel's premenstrual hypothesis might have been as follows. To use statistics in this field you require a null hypothesis, which is a formal definition of the mathematical way the symptoms must be temporally related. But, whatever axioms you choose, there will be examples which will fit your formal definitions but not fit your clinical meaning, and vice versa.

A polynomial or harmonic analysis of enough terms will completely represent any time series, but how many terms have any clinical meaning? A sine wave is a crude representation of data from a menstrual cycle, as is a straight line of much other clinical data. However, the sine wave implies a more appropriate approach to a periodic phenomenon than a polynomial function, and the equation's constants have approximately meaningful clinical significance. The struggle for a more completely objective analysis may be commendable yet questionable.

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INTERMITTENT PIMOZIDE IN CHRONIC SCHIZOPHRENIA

DEAR SIR,

I read Dr McCreadie and his colleagues' recent paper (*Journal*, December, 1980, **137**, 510–517) with great interest. Although it is cost saving, intermittent antipsychotic medication ('drug holiday') may be hazardous. Dr McCreadie rightly pointed out that tardive dyskinesia can be precipitated by "drug holiday". There may also be relapse of schizophrenia, which is sometimes called dopaminergic supersensitivity psychosis (Chouinard and Jones, 1980), as well as physical complications (Kitamura, 1976) as a rebound phenomenon.

If, on the other hand, four-day-a-week medication is recommended *because* of pimozide's half life as long as 50 hours, then why not prescribe pimozide once every other day?

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MONOSYMPTOMATIC DELUSION TREATED WITH BEHAVIOURAL PSYCHOTHERAPY

DEAR SIR,

May I comment on the paper by Beary and Cobb (*Journal*, January 1981, **138**, 64–66).

A patient may present with the idea that he smells (when this is not objectively the case) in a variety of different psychiatric syndromes. The symptom can occur in certain sensitive personality developments as an over-valued idea which may dominate the patient's whole psychic life. It is also sometimes seen in depressive illness as a delusion-like idea secondary to the morbid affect, in attenuated schizophrenic illness (or monosymptomatic hypochondriacal psychosis) as a delusional belief, and rarely in organic psychosyndromes. We are only given cursory clinical details of one of the three patients mentioned in the above paper, so that it is difficult to be satisfied regarding the underlying diagnosis in all three. The psychopathology of delusion is all important here. One's confidence is not helped by their woolly comment "avoidance behaviour may reinforce delusional thinking, as often happens in obsessive compulsive and phobic neurosis". Delusions do not occur in obsessional neurosis. I am not arguing that their patients were not deluded, simply that the reader needs more information to be satisfied.

My main contention, however, is with the conclusion "mono-symptomatic delusion is now a treatable condition". They report that two of their three patients improved regarding everyday behaviour, but in both the "delusion" persisted albeit with "reduced

intensity". The third patient was not helped by the treatment. The length of the follow up period was only five months. These results surely do not warrant such a dogmatic and optimistic conclusion. This needs to be said as we are being told on the one hand that pimozide is a therapeutically effective drug for such patients, and now that pimozide plus behaviour therapy or behaviour therapy alone can work the miracle. A larger number of patients, stringently defined inclusion criteria, controlled studies and a much longer follow up period are required before any such claim can be made.

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DIAGNOSTIC USE OF SLEEP DEPRIVATION

DEAR SIR,

It is generally agreed that the improvement in mood that follows Sleep Deprivation (SD) in depressed patients is usually short-lived (Post *et al*, 1976). For this reason, the therapeutic use of SD has been questioned (Knowles *et al*, in press).

There is, however, one use of SD which has received no mention in the literature, that is, as an aid to diagnosis. In our experience, SD can be decisive in clarifying the sometimes very difficult differential diagnosis between depression and dementia. We have studied a series of such cases referred, because the diagnosis was obscure, to the Treatment Evaluation Unit, Kingston Psychiatric Hospital. In some cases, one 40-hour period of SD resulted in a complete reversal of mood and a dramatic return to normal intellectual function. The duration of this reversal, though it may be brief, is usually long enough to allow psychometric testing to be done to determine whether there is intellectual deterioration. When faced with such a diagnostic question, it is also clinically useful to have a measure of the degree of recovery that is attainable, thus setting a goal for subsequent treatment. Knowing that the syndrome can be reversed has allowed us to undertake treatment with a more precise indication and with greater confidence in the results than would have been possible otherwise.

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ADOPTION RESEARCH IN SCHIZOPHRENIA

DEAR SIR,

I was involved recently in the adoption of the baby of a sixteen-year-old schizophrenic, whose own mother had also suffered from schizophrenia. The social workers explained that the adopting parents were entitled to the background information about the baby's origins, and I agreed to meet them. Questions were asked about the heritability of schizophrenia, and even what signs to look out for in adolescence in the unfortunate event that the daughter should develop the illness. I realized that the child entered its family trailing a background of schizophrenia, and would be watched closely all its life to see if the hereditary taint would show itself in abnormality.

What is the bearing of this on the adoption research from Oregon and Denmark (see Gottesman, 1978), which I had thought represented cast-iron evidence for schizophrenia being to a substantial extent truly inherited by genetic mechanisms? If there was some transmission of background information to the adopting parents, as can occur in this country, did it invalidate the aim of the research to separate genetic and environmental influences on the children studied?

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EARLY AGE OF ONSET OF PSYCHIATRIC DISORDER AND THE PROPORTION OF ILL RELATIVES

DEAR SIR,

Family studies of psychiatric disorders have consistently revealed higher rates of morbidity in the relatives of patients with an earlier onset of disorder. From a review of 18 studies, we recently noted that this relationship appeared to be nonspecific and held for affective disorders in general, bipolar and unipolar subtypes, alcoholism, and possibly schizophrenia. In an almost repetitive fashion, several authors concluded that a stronger or independent genetic component was involved in the etiology of early onset disorders,