confusion by eliciting a patchy memory, depersonalization, time disorder, disorder of visual perception, return to previous haunts and disturbance of sleep, and evidence of changed personality by what might be termed extreme 'out-of-character' acts. He suggests that these phenomena were caused by alcohol (in the earlier two necrophiliac episodes) and a combination of alcohol and clonidine (on the night of the stabbing). The contribution of clonidine may arguably be discounted. Even if all six tablets of 0.025 mg were ingested the dose is not of much magnitude. Hypertensive patients on clonidine may take up to ten times that amount per day and three times as much in a single dose. There is no known evidence that in dosage of 0.15 mg there is any significant interaction with alcohol.

However, on each of the three critical dates there is evidence of heavy alcoholic indulgence. Perhaps the Jury inclined to the view that the defendant's aberrations of behaviour were simply the result of drunkenness. That is understandable but there is an alternative formulation.

The clinical findings listed are all consistent with a diagnosis of temporal-lobe epilepsy, including the defendant's remembered feelings of fear. Ingestion of alcohol features high on the list of known precipitants and indeed stress is widely thought to play a part. The defendant's landlady appears to have been the last witness to have seen him and that a few hours before the crime. He is said to have 'had a glazed look, stared silently and did not accept a cup of tea offered'. She may well have been describing the state of altered consciousness occurring in an individual who is experiencing an epileptic fugue, rather than someone who is simply drunk. Was this approach considered and was the EEG investigated? A defence based on epileptic automatism and a plea of diminished responsibility might have enjoyed a more favourable outcome.

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Dear Sir,

In my experience a plea of diminished responsibility has always involved the defendant knowing that he was killing someone but there were reasons which substantially impaired his mental responsibility for his act. The defendant had no real idea that he was killing someone even though he knew that he was stabbing at something in the bed; hence a plea of insanity was more appropriate. Epilepsy and particularly temporal lobe epilepsy was carefully considered and excluded from diagnosis by each of the four psychiatrists who examined the defendant. The EEG was normal during routine examination but was not tested with the defendant under the influence of severe alcoholic intoxication. I would have very much liked the opportunity to have carried out this test.

I would not accept the behaviour as being simply due to alcoholic drunkenness but would accept that the defendant was one of those unusual persons in whom alcohol not only induces 'simple drunkenness' but in addition a most complex change of personality: 'Jekyll and Hyde' effect.

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SPECIFICITY OF SLEEP DEPRIVATION IN DEPRESSION

Dear Sir,

There is substantial evidence that the majority of depressed patients deprived of sleep for up to 36 hours on one or more occasions show some degree of clinical improvement during the course of this regimen. In some instances, a single sleep deprivation treatment is said to be followed by sustained remission of depressive symptoms (e.g. Pflug, 1976).

Reviewing the then available studies, Roy and Bhanji (1976) cautioned that, as in the case of insulin coma therapy, the essential elements of the treatment could prove non-specific. Given that improvement sometimes occurs when depressives are treated solely by a placebo (e.g. Medical Research Council, Clinical Psychiatry Committee, 1965), this possibility cannot be ruled out. However, in experimental studies of a single case—a 73-year-old depressed lady —we have found that improvement in clinical state, as judged by 'blind' ratings on a 100 mm bipolar visual analogue scale (mania-depression), is contingent on the passage of an apparently critical period of wakefulness. This relationship has held under four conditions, and has done so on replication of each one (see Figure). (The four conditions were: the patient awoke as usual at about 6 a.m. and remained awake for the next 36 hours (○); 21 hours of wakefulness following nearly 3 hours of sleep taken between 2 and 5 a.m. (△); 21 hours of wakefulness, beginning at approximately 1 a.m., following an awakening 3 hours after sleep onset (□); and 21 hours of wakefulness following nearly 3 hours of sleep taken between 2 p.m. and 5 p.m., i.e. acute reversal of the sleep-wakefulness cycle (●). In all instances, the duration of sleep was ascertained by EEG recording, and the duration of wakefulness timed from the point at which the recording had been terminated). As the Figure shows, under these conditions, a

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relatively abrupt transition in her clinical state occurred reliably after some nineteen to twenty hours of sustained wakefulness. In our view, non-specific factors are unlikely to exert so predictable an influence.

In addition, in the experiments from which these data are taken, we have commonly contrasted two conditions in counterbalanced order (e.g., A B B A). In one, the patient was woken after three hours of sleep and either kept awake (A), or allowed to return to sleep and to awaken at her normal hour (B). At 9 p.m., by which time she had been awake for different periods of time under the two conditions (approximately twenty and fifteen hours for conditions A and B respectively), improvement was noted only under the former condition. It is difficult to attribute the difference in outcome to non-specific factors since they are likely to have been common to both conditions. Rather, it appears that the difference is due to the fact that only under condition A was she awake long enough for recovery to occur.

To date we have observed only one major departure from the relationship depicted in the Figure (below). When reversal of the sleep-wakefulness cycle was maintained beyond 24 hours, the patient both improved more rapidly than predicted, and relapsed shortly afterwards. However, while unexpected, this sequence of events proved replicable. Thus, in sum, our experience suggests that, while not wholly predictable as yet, changes in this patient's clinical state are specifically related to the imposed modifications of the sleep-wakefulness cycle.

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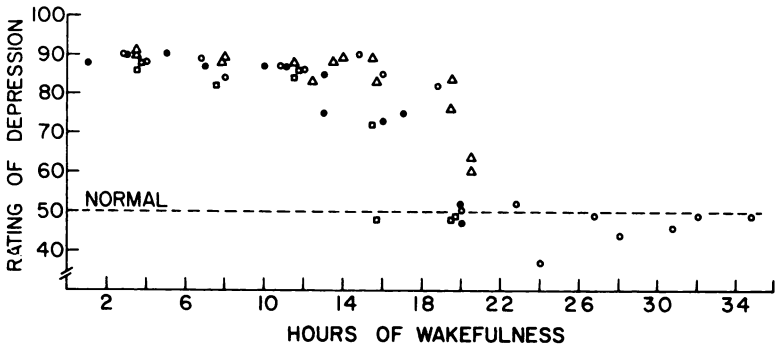
Figure for letter from J. B. Knowles and S. Southmayd (see below)



References

MEDICAL RESEARCH COUNCIL, CLINICAL PSYCHIATRY COMMITTEE (1965) Clinical trial of the treatment of depressive illness. *British Medical Journal*, *i*, 881–6.

PFLUG, B. (1976) The effect of sleep deprivation on depressed patients. *Acta Psychiatrica Scandinavica*, **53**, 148–58.

ROY, A. & BHANJI, S. (1976) Sleep deprivation treatment in depression: a review. *Postgraduate Medical Journal*, **52**, 50–5.

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