

syndrome undergoes accelerated aging (Kedziora *et al*, 1981).

R. D. EASTHAM

Pathology Department,
Frenchay Hospital,
Bristol BS16 1LE

J. JANCAR

Stoke Park Hospital,
Stapleton,
Bristol BS16 1QU

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PSYCHIATRIC MORBIDITY AND CIRCADIAN RHYTHMS

DEAR SIR,

In commenting on the paper by Jauher and Weller (*Journal*, March 1982, **140**, 231–5), Dr Gunnar Götestam (*Journal*, September 1982, 317–18) has confused certain matters. One of the confusions arises from the ambiguity of the references to what is 'advanced' when we refer to alterations in the circadian rhythm. He writes, 'Flying east also means a phase advance in the sleep-wake cycle', and cites Wehr *et al* (1979) in support of the observation that a flight eastwards tends to produce elevation of mood. What Wehr *et al* have written is, 'Sleep in depressed patients resembles sleep in normal subjects whose circadian rhythms of temperature and rapid-eye-movement sleep are phase-advanced (shifted earlier) relative to their sleep schedules' (*Ibid*, p. 710). Thus if the phase of the sleep-wake cycle is *advanced* the phase of the other circadian systems is *delayed*. But in writing of flights westwards Götestam states, 'Flying west prolongs the 24 hour day and delays the circadian cycle'. This is incorrect: it does not—it *advances* it (shifts it earlier) relative to the sleep-wake cycle, hence the depressive effect.

Neither is Götestam correct when he states that, 'It is now well known that sleep deprivation elevates mood (Pflug, 1978). A deprivation of a night's sleep may result in a slight increase in mood, as is often experienced by doctors after an entire night on duty'. But the study of Pflug to which he refers was concerned with endogenously depressed patients: in a previous study Pflug and Töller (1971) showed that normal subjects suffer some dysphoria after the loss of a night's sleep in contrast to the relative euphoria of depressed patients. There is little doubt about the

validity of this finding for it has been confirmed independently by Cutler and Cohen (1970) and Gerner *et al* (1979). It seems hardly likely that Götestam's medical colleagues were endogenously depressed! Now that the therapeutic possibilities of using sleep deprivation are being further explored (see Lovett Doust and Christie, 1980) it is of great importance to get our basic facts right.

Finally, I must point out that Götestam is hardly correct in stating that, 'Tricyclic antidepressants (TCA) are not so far known to affect the circadian rhythm . . .' He cites Wehr *et al* (1979) but the latter state quite unequivocally that tricyclics do have this effect—'. . . tricyclic antidepressants and estrogen, all of which have profound effects on depressive illness, also alter the basic timekeeping function of the circadian clock'. (*Ibid*, p. 712).

H. B. GIBSON

The Hatfield Polytechnic,
PO Box 109,
Hatfield,
Herts AL10 9AB

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MONTHLY VARIATION OF SUICIDAL AND ACCIDENTAL POISONING DEATHS

DEAR SIR,

Barracrough and White (1978a, 1978b) reported that the monthly distributions of accidental and undetermined deaths due to poisons were significantly

different from the monthly distribution of suicidal deaths due to poisons. They reported that the undetermined and accidental deaths due to poisons showed no monthly variation, but that suicidal deaths due to poisons peaked in May.

The present study sought to replicate this finding in the USA and to examine differences for different poisons. Deaths due to poisons in the USA are classified as due to solids and liquids and as due to gases and vapors. Undetermined deaths due to poisons are not classified in the USA data.

Data for the year of 1970 were obtained from the *Vital Statistics of the United States, 1970*. The mean number of deaths per day for each month from each of the officially defined causes is shown in the Table below.

The seasonal variation in suicide differed for poisoning by solids and liquids and for poisoning by gas ($\chi^2 = 33.75$, $df = 11$, $P < 0.001$). Suicidal deaths due to solids and liquids peaked in May and September, whereas suicidal deaths due to gas peaked in February and October.

The seasonal variation in accidental deaths due to drugs/medicaments/other solids/liquids also differed significantly from that due to gases and vapors ($\chi^2 = 430.59$, $df = 11$, $P < 0.001$). Accidental deaths due to solids/liquids had a trough in February to March, whereas accidental deaths due to gases and vapors had a trough in June to September.

The seasonal variation in poisoning deaths as a whole by accident and by suicide also differed significantly ($\chi^2 = 101.23$, $df = 11$, $P < 0.001$). Suicidal

deaths peaked in March to May and September. Accidental deaths peaked in November to January.

These results support those of Barraclough and White, namely that accidental deaths due to poisons have a different seasonal variation than suicidal deaths due to poisons. The present study found, in addition, different seasonal variations for different types of poison. The present study supports, therefore, the conclusion of Barraclough and White that accidental deaths due to poison are not concealed suicides.

DAVID LESTER

*Richard Stockton State College,
Pomona,
New Jersey, USA*

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The Claybury Selection Battery Manual

DEAR SIR,

We wish to comment on the review of the above by Paul Kline (*Journal*, January 1983, **142**, 108). His comments are quite misleading. He states that “little evidence for validity is presented and group scores are based on very small numbers”. However, of the 21 groups involved in this aspect of validation some are over 80 and the average size is 35. Other validation studies reported in manual include large samples of

TABLE

Monthly variation of suicidal and accidental deaths in the USA in 1970 for different poisons. Number of deaths per day

	Suicides by poisoning		Accidental deaths by poisoning		
	Solids/liquids	Gas	Drugs/medicaments	Other solids/liquids	Gases/vapors
January	10.1	6.6	7.0	3.3	10.1
February	10.4	8.1	5.2	2.7	6.5
March	10.7	7.8	5.1	2.6	6.7
April	11.5	7.8	6.4	2.9	3.7
May	12.0	7.1	6.9	3.5	3.2
June	10.7	6.5	7.3	3.4	1.8
July	10.5	5.8	7.0	3.6	1.9
August	11.5	5.7	8.1	2.9	1.6
September	12.9	6.1	7.6	3.1	1.4
October	11.2	8.1	7.1	3.2	4.4
November	11.4	7.1	7.4	3.1	5.9
December	10.0	6.9	7.3	4.3	6.2