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Correspondence

Correspondents should note that space is limited and shorter letters have a greater chance of publication. The Editors reserve the right to cut letters and also to eliminate multitudinous references. Please try to be concise, strictly relevant and interesting to the reader.

SOMNAMBULISM, BED-TIME MEDICATION AND OVER-EATING

DEAR SIR,

Several cases of somnambulism induced by bedtime medication have been reported in the literature in the last few years.

Huapaya (1979) reported seven cases of somnambulism induced by combinations of bedtime medication, including hypnotics, neuroleptics, antidepressants, minor tranquillizers, stimulants and anti-histamines. Sometimes the patients also drank alcohol.

Luchins *et al* (1978) reported a case of filicide performed during somnambulism induced by a combination of bedtime psychotropic medications.

Case Report:—Mrs B. a 35-year-old woman, divorced mother of two daughters, had a 10 year history of a schizo-affective illness.

During these ten years she was treated with many combinations of psychotropic drugs. While the patient was a teenager and before she took any medication she had experienced two episodes of somnambulism.

In January 1979 the patient weighed 91 kg and she put herself on a diet of 1200 calories per day. Her severe anxiety, depression and insomnia increased as she attempted to lose weight. She had been on 400 mg of chlorpromazine and 100 mg of amitriptyline per day, the whole dose to be taken at bedtime. As the patient continued to complain of insomnia methyprylon 300 mg was added at bedtime, shortly after the patient went on the 1200 calorie diet. She stayed on this regimen for two weeks. During this time she experienced 5 episodes of somnambulism. In her sleepwalking episodes she laid the table in the kitchen and ate all the food available in the refrigerator. The patient had total amnesia about the sleepwalking episodes. She became aware of these episodes when her daughter woke her at 1 a.m. while the patient was sitting at the kitchen table and eating. This happened during the fifth and last episode.

When the patient reported the episodes of sleepwalking the methyprylon was discontinued and replaced by chloral hydrate. There was no recurrence of sleepwalking during a follow-up of over one year, although she continued to take chlorpromazine and amitriptyline.

Scrutiny of the histories in the published cases of psychotropically induced somnambulism reveals several factors in common.

(1) Most of the patients who experienced somnambulism had a history of sleeptalking or sleepwalking, in the past, while taking no medication.

(2) In these cases, when taking medication at bedtime, it seems that only a specific drug, or combination of drugs, different for each individual, induced sleepwalking. When this combination was altered, the sleepwalking ceased.

If a history of sleepwalking or sleeptalking is obtained, the physician should refrain, whenever possible, from prescribing a combination of psychotropic drugs as bedtime medication in order to avoid the potential hazards of somnambulism.

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ANTICIPATORY GRIEF

DEAR SIR,

Parkes (*Journal*, February 1981, **138**, 183) acknowledges our caveats concerning the research on anticipatory grief, but fails to heed them in his letter. He asserts that among studies of young spouses (i.e. under 45 years of age) the findings are unanimous: forewarning of beareavement leads to good outcome. But as we observed in our article, the studies cited may not have successfully operationalized and measured 'forewarning' and 'outcome'. This casts some doubt on the internal validity of those several studies and may make any 'clear evidence' of a relationship illusory. The difference between us here, we believe, is that we choose to approach these seemingly consistent findings with greater scepticism.

Parkes discounts the single study (Maddison and Walker, 1967) which challenges his conclusion, on the

grounds that it did not include young and middle-aged widows as we reported. But Maddison himself later described the subjects as 'young and middle-aged' (Maddison, 1968). Perhaps the confusion lies in the fact that it was the deceased *husbands* who were reported as being older (i.e. over 45).

Parkes cites Lundin's Swedish study as added evidence for his position even though that study included persons up to 65 years of age. But Lundin's study is especially vulnerable to the criticisms we have raised. We seriously question, for example, whether social insurance claims are a valid measure of morbidity.

Lundin reported that relatives of 32 sudden-death patients showed a marked increase in morbidity following the death while relatives of 55 controls who suffered a more prolonged death did not. However, Lundin also reported that the prolonged-loss group showed higher-than-normal morbidity both *before* and *after* the loss. This raises the question: Does the fact that their condition did not worsen after the death necessarily indicate 'good outcome?' We think not.

We recognize that a sudden and untimely death can be a catastrophic event for a survivor and may lead to pathogenic grief. But so may a prolonged and stress-filled anticipated loss, even for a young survivor. As we observed, the important point for the clinician is that 'outcome' is not determined simply by the presence of 'forewarning', but rather by the manner in which the 'forewarning' is experienced.

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GUIDED MOURNING FOR MORBID GRIEF

DEAR SIR,

In the article 'Guided Mourning for Morbid Grief' (*Journal*, March 1981, **138**, 185–93) the concept of phobic avoidance concerning the loss of a loved person does not fit comfortably in the process of mourning. An apt comparison would be with a phantom limb: the victim may try helplessly to avoid the non-existent limb and a lay therapist may be tempted to treat with desensitization.

In the present study the time factor and general supportive measures may conceivably have played a crucial rôle in the healing process: therefore, guided mourning may not, in fact, have had any therapeutic effect, while the directive avoidance therapy given to the control group may well have had a positive anti-therapeutic effect. It is indeed likely that neither therapists nor patients in the control group believed in the efficacy of the treatment. Hence the modest significant difference between the two groups in favour of guided mourning.

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SEX DIFFERENCE IN SEASONAL VARIATIONS IN SUICIDE RATE

DEAR SIR,

I think I might be able to throw some light on the finding of Meares *et al* (*Journal*, April 1981, **138**, 321–5) that whereas for men there is a single peak in suicide rate in April/May, for women there are two peaks, one in March/April and one in October/November.

I am at the moment publishing a hypothesis (Skutsch, 1981) about the causes of manic/depression which I believe to be due to a disorder of dopamine metabolism such that dopamine is too low in manics and too high in depressives.

As a corollary I have, in the same paper, suggested that other types of depression might well prove to be due to high levels of dopamine. Thus Euvraard *et al* (1980) have shown (in women) that oestrogen stimulates prolactin release by inhibiting the output of dopamine. (Dopamine, of course, exerts an inhibitory control over prolactin release). Valsik (1965) has demonstrated in a large group of European girls that menarche occurs least often in March and in October (the effect being more marked in March). Menarche is caused by rising oestrogen levels so one can probably deduce that oestrogen levels are unusually low (and therefore that dopamine levels are exceptionally high) in March and in October. So far as I know nobody has demonstrated that there *is* a circannual rhythm of basal oestrogen levels in adult women, but Reinberg *et al* (1978) *have* shown (in a group of young European men) that there is a circannual rhythm of testosterone in which there is a trough in May and a peak in November. As oestrogen in men is derived from peripheral conversion of testosterone one can probably safely assume that oestrogen is also at its lowest