correlates with motor activity and behavioural activation, and has been implicated in parasomnias such as REM sleep without atonia (Jacobs & Azmitia, 1992).

It must, moreover, be emphasised that this second sleep record is not "normalised" as suggested by the authors. There is considerably less REM sleep and more awakenings out of REM sleep. These findings are in accordance with literature describing increased alertness and decreased REM sleep following 5-HT reuptake inhibitors (Gaillard, 1990).

Considering these misperceptions we do not feel that this case report offers valid arguments for treating sleep terrors and/or somnambulism with SSRIs.

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B. VAN SWEDEN
M. G. VAN ERP
F. MESOTTEN

Medisch Centrum St.-Jozef
Bilzen, Belgium

J. PEUSKENS

University Psychiatric Centre
St.-Jozef, Kortenberg
Belgium

AUTHORS' REPLY: The main comment of Van Sweden *et al* seems to be that because night terrors occur from slow wave sleep (SWS), which was minimal in our patient, the diagnosis of night terrors was wrong. The main thrust of our original discussion addressed this point and we would still contend that a major reason for the paucity of SWS was probably that each episode of SWS was rapidly terminated by a terror. We examined the nature of the SWS prior to each terror and found no evidence of hypersynchronous delta bursts; the delta activity fulfilled Rechtschaffen and Kales criteria for SWS. The next comment, that the increase in SWS on treatment did not worsen the terrors, misses the point. We used the SSRI treatment not to increase slow wave sleep directly but to reduce the anxiety that gave rise to the interruption of sleep by terrors, thus permitting more continuous SWS episodes. It seems that, in humans, SSRIs in general have no effect on SWS (Kupfer *et al*, 1991; van Bemmel *et al*, 1993) which is interesting given the established finding that directly-acting serotonin agonists such as mCPP reduce this stage (Lawlor *et al*, 1991; Katsuda *et al*, 1993).

We never claimed that the second sleep recording was normalised, although the patient thought her sleep was. The second hypnogram shows the classic picture of REM delay caused by SSRIs. Naturally we would not, on the basis of a single case, claim to have found the ideal treatment for night terrors. However, we feel that in similar cases, where night terrors are similarly profoundly problematic, SSRIs might be tried.

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S.W. WILSON
A.R. LILLYWHITE
D. J. NUTT

Psychopharmacology Unit
University of Bristol
Bristol BS8 1TD

Selective serotonin reuptake inhibitors and mania

SIR: Peet (*BJP*, April 1994, **164**, 549–550) recommended that patients at risk for mania be treated with selective serotonin reuptake inhibitors (SSRIs) rather than tricyclic antidepressants (TCAs).

This report does not include information about concurrent antimanic drug use. TCAs have been available longer than SSRIs, with much greater concern about TCA-induced mania or rapid-cycling. Therefore bipolar patients recently treated with newer antidepressants may be more likely to receive prophylactic antimanic treatment. This would bias the rate of treatment-emergent mania.

Peet also does not distinguish between bipolar subtypes. Antidepressant-induced mania may occur less often in bipolar II than in bipolar I disorder

(Kupfer *et al*, 1988). Moreover, bipolar I patients might be more likely to receive concurrent antimanic treatment because of a greater concern about treatment-emergent mania. The proportion of bipolar subtypes could bias the rate of treatment-emergent mania.

Finally, the rate of mania increases from the placebo to the TCA to the SSRI unipolar groups. The rates are significantly greater for drug groups compared with placebo, but the significance between the antidepressant groups is not reported. This trend hints at a possible heightened sensitivity to SSRIs. Fluoxetine may be more effective than imipramine in bipolar depression (Benfield *et al*, 1986; Cohn *et al*, 1989), further suggesting that bipolar patients are more sensitive to SSRIs and have a greater risk for treatment-emergent mania.

The problems associated with TCAs have been recognised only after many decades of clinical use. SSRIs are believed to be a 'safer' class of antidepressants, but there has not been sufficient clinical experience to support this notion. Claims about the superior safety of the SSRIs must therefore be carefully and critically evaluated. Hypomanic symptoms are notoriously under-recognised, but are often a prodrome to bipolar disorder (Howland & Thase, 1993). Because patients are more likely to seek treatment for depression, a careful assessment of risk factors for bipolar disorder is imperative before starting an antidepressant. The SSRIs now enjoy widespread use, especially by non-psychiatric physicians, and many depressed patients seek treatment in non-psychiatric settings (Howland, 1993). Hence, many depressed patients may not have been adequately evaluated and may be at risk for antidepressant-induced mania.

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ROBERT H. HOWLAND

Department of Psychiatry
University of Pittsburgh School of Medicine
3811 O'Hara Street
Pittsburgh, PA 15213

Substance abuse in schizophrenics

SIR: Smith & Hucker (*BJP*, July 1994, **165**, 13–21) reviewed the literature on substance abuse in schizophrenics, focusing on the link between substance abuse and violence, and showed substance abuse as a possible "marker of either some underlying propensity or of an increased likelihood of aggression under certain conditions". Having addressed the problem of delinquency and violent behaviour in schizophrenics recently (Soyka, 1993), their conclusions require discussion.

High rates of substance abuse and dependence have been reported in North American and in some European samples. For example, in two large German samples of schizophrenic in-patients, lifetime prevalence estimates for substance abuse were found to be 21.8% and 42.9%, with 3-month prevalence rates being 21.3% and 29.0% (Soyka *et al*, 1993). Whether these high rates reflect a real increase of substance abuse in schizophrenia or not is a matter of controversy. Cuffel (1992) pointed out that recent studies show higher prevalence estimates for substance abuse than older studies, but a variety of reasons may account for the observed differences (Alterman, 1992).

Dual diagnosis schizophrenics are not only at increased risk for violence and aggression but delinquency in general. In the two German samples mentioned above, 25% of all schizophrenics had been convicted before, mostly for offences against property (19.5%) and traffic offences (4.3%), while violent behaviour was comparatively rare (1.8%). Patients with substance abuse had been convicted more often than other schizophrenics (40.1% v. 13.7%, $P<0.01$).

Variables that may acount for the apparently higher risk for violent behaviour in dual diagnosis schizophrenics include the psychopathological and social factors reviewed by Hucker & Smith. Since causes of violence in schizophrenics are multifactorial, in most cases it may be more appropriate to speak of "risk factors" instead of "markers", with substance abuse being one among many. The role of intoxication in the development of violence seems to be overestimated. Boeker & Haefner (1973) found that only 10.4% of violent schizophrenics studied were intoxicated at the time of their delinquent action. Further research is needed on the relationship between substance abuse and schizophrenia, especially on the aetiology and treatment of substance abuse.

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