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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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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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

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