insomnia that is proportional to the degree of brain serotonin depletion (Petitjean *et al*, 1978), which can be reversed by serotonin precursors such as tryptophan. There is also evidence from animal and human studies that serotonin is involved in the control of slow-wave sleep and that serotonin receptor subtypes may mediate different aspects of sleep and wakefulness (Sharpley & Idzikowski, 1991). However, there appear to have been no specific studies on the use of SSRIs for the treatment of sleep disorders, although they do normalise the REM advance seen in depression. This case illustrates an interesting paradox in that, at least in volunteer studies, the SSRIs tend to decrease sleep continuity (Nicholson & Pascoe, 1986), whereas in this case the patient's sleep was improved.

Clonazepam was substituted for her diazepam because it is recognised that there is often an increase in subjective anxiety and sleep disturbance on first commencing treatment with SSRIs. Clinically, we have found that by using a small dose of clonazepam these effects are minimised, and withdrawal, once the patient has become established on the SSRI, has not proved to be a problem to date. The possibility that paroxetine alone would have been effective might be worth evaluating in the future.

In summary, we report the use of domiciliary sleep recording in diagnosing a case of marked night terrors with sleep-walking. This patient was treated very successfully with a combination of a benzodiazepine and an SSRI and is currently in a period of prolonged remission on the SSRI alone.

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A. R. Lillywhite, MRCPsych, *Senior Registrar*; S. J. Wilson, *Research Assistant*; *D. J. Nutt, DM, MRCP, MRCPsych, *Honorary Consultant Psychiatrist, Psychopharmacology Unit, School of Medical Sciences, University of Bristol, University Walk, Bristol BS18 1TD*

*Correspondence

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Sleep Deprivation as a Diagnostic Instrument

C. J. WILLIAMS, J. D. I. YEOMANS and A. K. COUGHLAN

A 59-year-old man with chronic treatment-resistant depression developed severe cognitive impairment. The severity of depression and the mode of presentation led to difficulty in diagnosis between a dementing disorder and a depressive illness. The diagnostic conundrum was resolved by the use of sleep deprivation (SD), which demonstrated clear subjective and objective improvements in his condition. These changes were quantified by a range of psychometric tests, which showed that initial deficits in cognitive performance improved temporarily after SD, and these improvements were maintained after effective treatment of the depression with electroconvulsive therapy.

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Difficulties can arise in distinguishing between dementia and depression in patients with cognitive

decline. In 1971, Pflug & Tolle reported that depressed patients deprived of sleep for one night enjoyed a short-lived remission of depressive symptoms. Wu & Bunney (1990) reviewed 61 papers describing over 1700 depressed subjects, and found that sleep deprivation (SD) had an antidepressant effect in 59% of patients.

Letemandia *et al* (1986) suggested that SD can distinguish between dementia and depression. They found that SD caused a brief reversal of mood and an increase in intellectual function that can be measured by psychometric testing. This can indicate the degree of recovery possible, thus setting a goal for subsequent treatment.

Case report

C, a 54-year-old man, had become increasingly depressed for 2 months with anhedonia, anergia, anorexia, sleep disturbance, and early-morning wakening. There was no previous history of mental or physical illness or alcohol or drug abuse. He had separated from his wife 2 years before and she described a short period of overactive behaviour 1 year prior to this admission. His brother had a history of depression.

He was treated with clomipramine 75 mg/day for 10 days, and this was then stopped because of a pyrexia of uncertain origin. Neuroleptic malignant syndrome was excluded. His mental state deteriorated despite concurrent treatment with tricyclic antidepressants, neuroleptics, and electroconvulsive therapy (ECT). He became increasingly confused and developed paranoid delusions.

After nine ECT treatments and the introduction of lithium and monoamine oxidase inhibitors, he remained confused, incontinent of urine, and was unable to dress or eat without assistance. His behaviour became more bizarre, with episodes of singing and shouting in a high-pitched voice. He complained of being 'mixed up' and 'puzzled'. By this stage he had received treatment with imipramine at doses of up to 300 mg/day, with doses of 225 mg/day for a period of more than 5 weeks. This was augmented with 800 mg lithium carbonate at night (serum lithium concentration, 0.54 mmol/l). Phenelzine was commenced in place of imipramine at doses of up to 90 mg/day, and he received more than 60 mg/day for 5 weeks. The lithium was increased to 1200 mg to give a serum concentration of 1.05 mmol/l. In addition, he received 100 mg chlorpromazine twice daily.

Electroencephalogram (EEG) recordings on three occasions revealed only a small increase in slow-wave activity compatible with medication effects. Two computerised tomograms 5 months apart during his admission showed no evidence of cerebral degeneration or focal lesions. Haematological, biochemical, microbiological, and endocrine investigations were all normal. Neurological examination revealed no significant abnormality. After 9 months in hospital he had lost over 25 kg in weight. The differential diagnosis at this point was depression or, in view of the confusion, an organic dementia of unknown aetiology.

Table 1
Results of neuropsychological assessment

	Before sleep deprivation	After sleep deprivation	After ECT	Comments and comparative data
Estimated premorbid WAIS-R full-scale IQ	97	97	101	Derived from reading ability on the National Adult Reading Test
WAIS-R age-scaled subtest scores				Mean (s.d.) of age-scaled scores = 10 (3) for general population
Digit span	9	13	10	
Arithmetic	6	8, 8	10	
Picture completion	6, 8	10	12	
Picture arrangement	6, 7	9	10	
Digit symbol	7, 3	9, 10	12	
Memory				
Cued recall list learning (five trials: max. = 60)	18	48	46	Age group mean (s.d.) = 53.9 (4.3)
Recognition memory test (max. = 50, chance = 18–32)				
Words	Unable	43	-	Age group mean (s.d.) = 45.3 (3.4)
Faces	22	46	-	Age group mean (s.d.) = 44.3 (3.5)
Frontal lobe				
Word fluency				
Letters C,F,L (60" each)	41	68	48	25th %ile = 31, 75th %ile = 44, 96th %ile = 53
Four-legged animals (90")	2	16, 20	14	38 controls, mean (s.d.) = 19.4 (5.6)
Countries (90")	1	15	-	Informal test, no control data

Occasionally tests were repeated within an assessment: where this occurred, both scores are given. A more detailed account of the neuropsychological testing and the tests used can be obtained from the authors on request.

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Sleep deprivation was instituted as a diagnostic aid in addition to his existing drug treatment. The patient was kept awake from 10 a.m. to 10 a.m. the following day on four occasions at approximately weekly intervals. There was a response to three of these trials. On the most successful trial he 'woke up' suddenly from his confusion at 2 a.m., fully orientated and alert. He gave a clear account of his mental state and expressed depressive and paranoid delusions. He was able to discuss current events in a clear and coherent way. He relapsed into his usual confused and indecisive state by the early evening.

Neuropsychological assessment

Neuropsychological assessment was carried out before SD, on the days after SD, and following a subsequent course of ECT. The results are summarised in Table 1.

Performance on several subtests of the Wechsler Adult Intelligence Scale-Revised (WAIS-R; Wechsler, 1981) improved following SD. Performance on tests of verbal learning (cued recall list learning) and recognition memory for words and for faces, which beforehand were extremely poor, improved substantially following SD.

He showed marked improvement in word fluency tasks, reflecting frontal lobe function, following SD. On one task he became hyperfluent after SD, performing well above the 95th centile for this test. Following ECT these improvements were maintained.

It is improbable that the various improvements noted are merely attributable to practice effects. His condition fluctuated considerably between the assessments, and his mental state appeared to exert a much greater influence than previous exposure to the tests.

On the basis of these results, ECT was restarted and continued in spite of an initial poor response. Antidepressant medication was continued throughout the period of ECT. After 18 treatments he became slightly hypomanic for a few days before his mood settled. His appetite returned and he regained his previous weight. He returned home and remained well at the 6-month follow-up, taking lithium medication.

Discussion

This case illustrates the usefulness of SD in confirming the diagnosis of an underlying mood disorder in a patient with a depressive pseudodementia. The striking clinical response to SD, coupled with objective evidence that cognitive function had improved, encouraged determined treatment of his depression. Sleep abnormalities and the effects of endogenous circadian rhythms are possible factors in the aetiology of depression. Antidepressant agents and ECT can reverse these sleep disturbances (Healy & Waterhouse, 1991). Sleep deprivation may act on the abnormal sleep rhythms found in depression.

Sleep deprivation combined with a clinical assessment and psychometric testing is an aid to diagnosis in depressive pseudodementia. With adequate nursing provision, SD may be employed simply and at no extra cost in many hospital settings. A clinical psychologist can quantify the cognitive response to SD. As with electroencephalogram results in epilepsy, a negative result is of little diagnostic significance, whereas a positive improvement, such as described above, is an immediate indicator of the underlying depressive component. In this case three out of four tests were positive to differing degrees, suggesting that a single negative SD response may be an inadequate test and that repeated sleep deprivations should be performed. Mood-related side-effects such as hypomania (Wehr, 1991; Wright, 1993) also may be diagnostic and are manageable in hospital should they occur.

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*C. J. Williams, BSc, MBChB, MRCPsych, *Lecturer/Honorary Senior Registrar in Psychiatry, St James's University Hospital, Beckett Street, Leeds LS9 7TF*; J. D. I. Yeomans, BSc, MBChB, MRCPsych, MMedSc, *Senior Registrar and Honorary Clinical Tutor in Psychiatry, Department of Psychiatry, St James's University Hospital, Leeds LS9 7TF*; A. K. Coughlan, PhD, Dip Clin Psychol, *Consultant Clinical Psychologist, Department of Clinical Psychology, St James's University Hospital, Beckett Street, Leeds LS9 7TF*

*Correspondence

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