investigation, I strongly suspect that an underlying stressor was present. The recurrence of symptoms upon the school re-opening, suggests that the precipitating stressor was related to the school. African outbreaks typically involve missionary schools (Ebrahim, 1968) or some conflict between students and administrators (Dhadphale & Shaikh, 1983) which may not be readily apparent to outside investigators. They are typified by children dominated by autocratic elders and having little means of redress, with conflict arising from exposure to foreign ideas which challenge traditional beliefs, fostering escape through conversion (Ebrahim, 1968).

There are many questions requiring clarification through interviews with a representative sample of those affected, and not just 12 pupils. It is clear from our sample that 'mass motor hysteria' subsides only after school administrators reduce or eliminate the anxiety-generating precipitant which typically involves strict academic or religious discipline. Hence, it is imperative for investigators to provide some ethnographic description of the participants. It is not enough to state that symptoms were attributed by parents to illness or evil spirits, as this is not a case of mass hysteria by proxy (*vide* Philen *et al*, 1989). Of key import is the folk belief of those affected, as conversion symptoms are a symbolic representation of an unresolvable conflict.

Two episodes bearing a remarkable similarity to that in Ali-Gombe *et al*'s report (laughing in conjunction with abnormal movements) have been recorded. One affected six schoolgirls aged 11–14 in France over 18 days (Armainguad, 1879), while the second was a three-day epidemic in Zambia among 125 students aged 16–17 (Dhadphale & Shaikh, 1982). Both were triggered by rigid educational policies and involved identifiable index cases.

Ali-Gombe, A., Guthrie, E. & McDermott, N. (1996) Mass hysteria: one syndrome or two? *British Journal of Psychiatry*, **168**, 633–635.

Armainguad, M. (1879) Recherches cliniques sur l'hystérie; relation d'une petite épidemié d'hystérie observee a Bordeaux. *Mémoire et Bulletin de la Société de Médecine et Chirurgie de Bordeaux*, 551–579.

Bartholomew, R. E. & Sirois, F. (1996) Epidemic hysteria in schools: an international and historical overview. *Educational Studies*, **22**, 285–311.

Dhadphale, M., & Shaikh, S. P. (1983) Epidemic hysteria in a Zambian school: "the mysterious madness of Mwinilunga". *British Journal of Psychiatry*, **142**, 85–88.

Ebrahim, G. J. (1968) Mass hysteria in school children, notes on three outbreaks in East Africa. *Clinical Pediatrics*, **7**, 437–438.

Philen, R., Kilbourn, E. & McKinley, T. (1989) Mass sociogenic illness by proxy: parentally reported in an elementary school. *Lancet*, *ii*, 1372–1376.

Wessely, S. (1987) Mass hysteria: two syndromes? *Psychological Medicine*, **17**, 109–120.

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Recovered memories

Sir: If Merskey (1996) is saying simply that great care must be exercised in evaluating memories of early childhood events recovered in therapy, then we are clearly in agreement (Brewin, 1996). However, it is not possible on the basis of personal opinions, position statements, court judgements, insurance company policies or allegations about the political bias of other investigators, to address the scientific issue of whether memories of events may be forgotten for long periods of time and then remembered with essential accuracy. Now that researchers are turning their attention to finding evidence for genuine recovered memories, new and more convincing data are being reported. For example, four additional case studies with high-quality corroboration have been presented by Schooler *et al* (1997, in press). Another recent study conducted by Andrews *et al* (details available from author) involved in-depth interviews with 108 chartered British psychologists concerning patients they had seen with recovered memories of trauma. Between them, the psychologists described 690 cases, and provided detail in 236 cases. Of the 236 patients, 97 (41%) had obtained some corroborative evidence for their memories; 33 had obtained corroborative evidence from more than one source. In 11 cases, the psychologist had seen this evidence at first-hand. Similar rates of corroboration have also been reported by Feldman-Summers & Pope (1994).

As in a recent survey of British False Memory Society members (Andrews, 1997; Gudjonsson, 1997), Andrews *et al* (details available from author) found that only a small minority of memories concerned events that had supposedly begun and ended before the age of three years. About one-third of memories involved non-sexual traumas such as physical abuse, traumatic medical procedures, or witnessing the death or injury of a close other. About one-third of memories were recovered prior to any therapy. These observations are only some among many that are inconsistent with Merskey's view that genuine recovered memories of trauma are either impossible or vanishingly rare. The evidence at present is supportive both of the possibility of genuine recovered memories and of the possibility that inappropriate therapeutic procedures can lead to the production of false memories. Far more research is needed before either of these positions may be rejected with confidence.

Andrews, B. (1997) Can a survey of British False Memory Society members reliably inform the recovered memory debate? *Applied Cognitive Psychology*, **11**, 19–23.

Brewin, C. R. (1996) Scientific status of recovered memories. *British Journal of Psychiatry*, **169**, 131–134.

Feldman-Summers, S. & Pope, K. S. (1994) The experience of 'forgetting' childhood abuse: a national survey of psychologists. *Journal of Consulting and Clinical Psychology*, **62**, 636–639.

Gudjonsson, G. H. (1997) Accusations by adults of childhood sexual abuse: A survey of the members of the British False Memory Society. *Applied Cognitive Psychology*, **11**, 3–18.

Merskey, H. (1996) Recovered memories (letter). *British Journal of Psychiatry*, **169**, 665–666.

Schooler, J.W., Bendiksen, M. & Ambadar, Z. (1997) Chapter in *False and Recovered Memories* (ed. M. Conway). Oxford: Oxford University Press, in press.

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Cognitive impairment associated with lamotrigine

Sir: Lamotrigine is well established as adjunctive anticonvulsant medication in people with epilepsy. It is of particular value in individuals who have seizures secondary to brain damage (Buchanan, 1995). We report the case of a 69-year-old female patient with a 10-year history of epilepsy and alcohol-induced dementia, whose epilepsy had been well controlled for 2 years with valproate 1000 mg b.d. and lamotrigine 100 mg b.d. She had had no seizures for three months. She had been abstinent from alcohol for 10 years. She was admitted for assessment because of a gradual deterioration in her cognitive state and functional level over a six-month period. She was alert, without psychotic features. She was very disoriented and unable to cooperate with most of the Mini-Mental State Examination (MMSE; Folstein *et al*, 1975), speaking in her native tongue despite usually having a good command of English.

Apart from an unsteady gait, physical examination was unremarkable as were routine laboratory investigations. Serum valproate was 95 mg/l (normal range

< 100 mg/l) and lamotrigine 13.6 mg/l (normal range < 4 mg/l).

After discontinuation of lamotrigine she became more communicative with clear improvement in cognition (MMSE 16/30) and a steady gait. Following two seizures she was recommenced on lamotrigine at 25 mg b.d. Repeat serum level was 3.3 mg/l. At six-month follow-up she remained seizure-free with a stable mental state.

This is a case of lamotrigine toxicity in a woman with known dementia presenting with gradual cognitive deterioration. Her increasing cognitive impairment could easily have been mistaken for the progression of her underlying dementing illness.

Valproate is known to inhibit the hepatic enzymes, notably causing a marked increase in the serum level of lamotrigine (*British National Formulary*, 1996, no. 32). In this case, however, the patient had been stabilised on this combination for the past two years without a change in dose. The mechanism of the raised serum lamotrigine levels remains unclear. When assessing a patient with a deterioration in cognitive function it is important to investigate for potentially treatable causes. Lamotrigine toxicity should be considered in all patients taking lamotrigine, in particular if taken in combination with valproate.

Buchanan, N. (1995) Lamotrigine: clinical experience in 93 patients with epilepsy. *Acta Neurologica Scandinavica*, **92**, 28–32.

Folstein, M. F., Folstein, S. E. & McHugh, P. R. (1975) Mini-Mental State: A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, **12**, 189–198.

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Paroxetine discontinuation syndrome in association with sertindole therapy

Sir: Sertindole is a novel atypical antipsychotic of great promise. We report a case in which we believe that sertindole therapy significantly worsened the symptoms of paroxetine discontinuation.

D. is a physically healthy 30-year-old man who had a diagnosis of paranoid psychosis and unipolar depression. Following review, his depot neuroleptic medication was stopped and he was commenced on sertindole, the dose titrated as recommended to 20 mg daily, and then paroxetine was stopped. He continued to complain of fatigue, poor motivation and somnolence. Severe withdrawal problems were not anticipated as he had previously stopped paroxetine completely with no ill effects. Within five days he became sleepless, sweaty, agitated and nauseous. There was no evidence of recurrent depressive or psychotic symptoms, and a diagnosis of 'serotoninergic syndrome' secondary to paroxetine withdrawal was made. Within two weeks he had returned to his pre-withdrawal state.

The severity of his reaction prompted us to investigate the interaction between paroxetine and sertindole further. Paroxetine increases serotonin availability by action on the neuronal membrane. It has been reported to down-regulate 5-HT_2 receptors on chronic admission. Sertindole antagonises 5-HT_2 receptors, an effect enhanced by paroxetine-induced inhibition of sertindole metabolism. On stopping paroxetine, the cessation of these additive effects would leave insufficient serotonin to produce adequate stimulation for the subsensitive receptors. Neither we nor the manufacturers of sertindole (Lundbeck Ltd) have located previous reports of this phenomenon.

We note the possible increased risk of a serotoninergic withdrawal syndrome with paroxetine in patients being treated with sertindole, and recommend that paroxetine either be withdrawn before the commencement of sertindole, or with caution thereafter. The new atypical antipsychotic olanzapine is metabolised by the cytochrome p450 pathway and may also interact with paroxetine in this way.

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Schizophrenia in Trinidad

Sir: I was pleased to read the study by Bhugra *et al* (1996) about the incidence of schizophrenia in Trinidad as few data have been published about the epidemiology of psychiatric illness in the Caribbean. Comment was made on the lower rate compared with the African–Caribbean population in London.

However, Trinidad is different to the other Caribbean islands in that the population is only 43% African–Caribbean, with 40% of Indian descent, 14% mixed, 3% Chinese, 1% White and 1% other (Central Intelligence Agency, 1995). I note the "total population of the relevant age-groups in the two catchment areas was 214 048", and wonder how many of those were not African–Caribbean. The capital, Port of Spain (one of the catchment areas), is certainly not populated solely by African–Caribbeans. Similarly, being non-African–Caribbean was not listed as an exclusion criterion, and the 56 patients reported were not explicitly identified as such.

From the information provided by the authors, the rate reported appears to be the incidence of schizophrenia in a multi-racial population, and comparison with African–Caribbean rates in the UK should perhaps be made with this caveat. Data from other Caribbean islands with more homogeneous populations may be more robust in such comparisons.

Bhugra, D., Hilwig, M., Hossein, B., *et al* (1996) First-contact incidence rates of schizophrenia in Trinidad and one-year follow-up. *British Journal of Psychiatry*, **169**, 587–592.

Central Intelligence Agency (1995) *The World Factbook 1995*. Washington, DC: CIA.

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Authors' reply: We read Dr Ratan's comments on our paper with interest. We acknowledge the diversity of the population within the descriptive term Afro-Caribbean. However, as the term African–Caribbean is used in the UK to include people who originate from a number of Caribbean territories, in the same way that White is used to include English, Irish, Welsh, Scottish and other groups, we decided to conform to the Office of Population Censuses and Surveys categories of ascription of ethnicity. We acknowledge that there are problems with such an approach, but until a different, more satisfactory classificatory system is devised this is what we have to work with. We recognise that the population in Trinidad is ethnically heterogeneous as is the White population in the UK. Because of the conventions we adopted we did not ascertain Trinidadian patients' self-ascribed ethnicity at the point of contact, hence we are unable to present incidence rates in various ethnic groups in Trinidad.

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