consultant psychiatrists serving Waterford over seven years), the problem is minimal. If that is so, it is important to appreciate the fuller picture:

- very few adults, after making 'recovered memory' accusations, are referred to a psychiatrist by their therapist. Psychiatrists, as a group, are therefore a poor litmus for false memories;
- from our initial analysis, only 13% of 'recovered memory' accusations arise while the patient is actually undergoing psychiatric treatment (as opposed to other forms of 'therapy');
- accusations which are not retracted may nevertheless be false accusations. However sincerely believed at the time, retracting the allegation, when it is realised that it is false, is a difficult, guilt-laden process for the accuser;
- there have been no "massive repression/recovered memory" accusations against parents which have subsequently been verified by independent sources. With the vast number of claimed cases around the world this lack of evidence might well render them liable to the same scepticism with which allegations of multi-generational satanic ritual abuse are now being met.[4]

Unfortunately this article does little to advance our understanding of why adults 'recover' false memories of childhood abuse.

Until the mental health professions realise the dangers involved in validating patients' uncorroborated, long-delayed abuse 'memories' and stop using coercive, directive therapy and interviewing techniques, all based on pseudo-science, to uncover what *they* presume to be buried sexual trauma – as Freud did 100 years ago[6] – wrongful accusations will continue to divert our attention from the genuine cases which do deserve our understanding, respect and sympathy.

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Allegations of child sexual abuse: delayed reporting and false memory

Authors' reply:

Sir – In reply to the general criticisms of the US and British False Memory Foundations, we would wish to remind readers of both letters and our paper[1] that we are aware of the limitations of the case series *(see Subjects and methods)* and that psychiatrists may be reviewing a biased sample. Our central point is that in the variety of experiences of delayed reporting of child sexual abuse and/or allegations subsequently withdrawn, the new 'syndrome' of false memory represents only a proportion of those patients, and that many other possibilities, some with psychiatric diagnoses, exist *(see Discussion)*. In her observation that psychiatrists have learned of the false memory syndrome (FMS) societies through the lay press, Freyd makes our point for us: the FMS debate has taken place largely in this arena, and we argue for discussion and standardisation within the mental health professions *(see Conclusion)*.

Our monograph does not advocate or deny the validity of false memory – it seeks to clarify this difficult clinical setting. Nor did we speculate on the theoretical basis of repression; our cases, having accepted the above limitations, presented a wide range of reasons (not causes) other than FMS, and raised the possibility of true FMS in only two cases *(cases C and K)*.

With regard to some of the specific points raised by Scotford, we agree with the four points and appreciate that neither FMS society is condemning the totality of psychotherapeutic practice. His letter does, however, juxtapose the "consultant psychiatrist in a leading teaching hospital" with the "correspondence course-trained hypnotherapist in the back room".

Certainly no psychiatrist can defend the so-called recovered memory therapy, and we share the concerns about one best-selling self-help book.[2]

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Dissociative psychosis: an atypical presentation and response to cognitive analytic therapy

Sir – Having trained and worked in psychiatry on both sides of the Atlantic, I am puzzled by the apparent North American-European discrepancy in diagnostic and therapeutic practice with respect to dissociative disorder. I am pleased to see that the *Irish Journal of Psychological Medicine* is attempting to bridge the gap by publishing Drs Graham and Thavasothy's article.[1]

However, I am mystified by their statement that "there is very little literature on the treatment of such disorders". There have been many advances in the field since the work of Freud and Janet referred to by the authors. There is now a wealth of literature on the treatment of dissociative disorders.[2-4] The International Society for the Study of Dissociation has held conferences and workshops for over 10 years and publishes a journal, *Dissociation*, devoted entirely to the subject.

The case subject of Dr Graham and Dr Thavasothy's article would not be considered greatly unusual or atypical on this side of the Atlantic. I commend the authors for pursuing

a creative treatment approach in an environment where dissociative disorder diagnoses are apparently infrequent.

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Dementia-like state in a patient treated with vigabatrin

Sir – Vigabatrin is a novel anti-convulsant drug, and structural analogue of the neurotransmitter GABA. It was first administered to man in 1979, and controlled studies proved its efficacy in refractory complex partial seizures.[1,2] A non-progressive and reversible micro-vacuolation or intramyelinic oedema localised to certain white matter tracts of the CNS has been shown in rats and dogs following long term treatment.[3] However, neurophysiological[4] and neuropathological[3] studies confirmed its safety in human beings. Most common side effects reported[5] are irritation, aggression and memory problems. We report on a case presenting with a picture similar to dementia in a patient treated with vigabatrin.

Case report

A 62 year old lady was admitted for assessment, and treatment of worsening epileptic attacks, who had epilepsy for 33 years following a left hemiparetic stroke. Her seizures were complex partial with secondary generalisation, poorly controlled on carbamazepine 600mg daily and sodium valproate 2,000mg daily. She had been living with and was cared for by her husband. She had a previous right mastectomy for carcinoma of the breast 16 years earlier, but was otherwise well. All routine biochemical and haematological investigations were normal. Sodium valproate was reduced to 1,000mg daily, and vigabatrin was started at 500mg daily then increased to 1,500mg after one week.

Two weeks later she became seizure free but was observed to be tearful and depressed believing that her husband had killed himself. She was disoriented for time and place, verbally aggressive and incontinent of urine. A CT scan showed generalised atrophy, but no focal lesion. She was referred for psychiatric opinion after two months for assessment of dementia. She was observed to be visually hallucinating and scored five on the mini mental state. Her mood was fatuous, but she denied any mood congruent or incongruent delusion. She had right/left disorientation, constructional apraxia, tactile agnosia and nominal dysphasia. Her EEG showed generalised slowing in the theta range, but no sharp waves. Serial mini-mental test scores remained at five.

Vigabatrin was reduced gradually at a rate of 500mg weekly and was accompanied by a corresponding improvement in her physical and mental state. Two weeks later there was no evidence of cognitive impairment, and her seizures recurred with a reduced frequency and severity.

Discussion

Confusion has been reported in 3.4% of patients receiving vigabatrin in controlled studies.[5] Our patient had all the signs suggestive of dementia that were clearly associated with vigabatrin treatment and remitted on its discontinuation. The most likely explanation is a prolonged acute organic brain syndrome (acute confusional state with visual hallucinations) that has been mistaken for a state of dementia. Depressive pseudodementia is unlikely considering the severity of the cognitive impairment. There is currently no published evidence that vigabatrin has any deleterious effects on indices of cognitive function.[5] However, schizophrenia-like state, depression and aggressive behaviour were reported in 4%-8% of patients receiving the drug especially in patients with a past psychiatric history.[5]

The possible pathophysiology of this adverse effect is complex and the presence of brain damage is a major contributory factor. Cortical GABA concentrations are reduced in Alzheimer's disease[6] and hence the mechanism of action of vigabatrin in epilepsy associated with increased cortical GABA is unlikely to explain the above adverse effect. It is very unlikely that forced normalisation[7] is the likely cause since the improvement in cognitive functions preceded the recurrence of seizures. However, it can only be ruled out with sequential EEG recordings before, during, and after the abnormal behavioural state. A reversible dementia like state needs to be included among possible side effects of vigabatrin especially in patients with brain damage.

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