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RE: False Memory Syndrome – Balancing the evidence for and against

Sir – Following publication of this paper, I have been asked whether I would care to comment further in the light of the recent Brandon Report, which has been published since my paper was accepted. (Brandon *et al* 1998)

Most of the conclusions and recommendations of the Brandon Report will be endorsed by the psychiatric profession. Both the report and my paper emphasise that we have a responsibility not to cause harm to patients or their families.

The Report makes the following statements:

"There can be no justification for the use of memory recovery techniques which involve significant departure from normal interview or psychotherapy techniques".

It adds, "Great caution is needed if memory is reported after years of apparent amnesia. There is considerable evidence that such memories cannot be relied upon". It goes on to make extremely helpful recommendations regarding interview procedures.

Both the Brandon Report and my own paper emphasise that if accounts of sexual abuse in childhood are to be believed, then there must be corroborative evidence. It is deplorable that so many families have been harmed by the 'discovery' of previous sexual abuse, when no such abuse has occurred.

The difference in view point between the Brandon Report and my paper is this. The Report states, "given the prevalence of childhood sexual abuse, even if only a small proportion are repressed and only some of them are subsequently recovered, there should be a significant number of corroborated cases. In fact there is none".

I quoted one or two cases at length in my paper. This was to emphasise that even if there are only one or two well documented cases, this would prove that sexual abuse may indeed occur, be forgotten, and subsequently be recalled. It is my opinion that this happens although probably rarely.

Extreme caution needs to be exercised before any accounts of sexual abuse which are 'remembered during therapy' are believed, and definite corroborative evidence should always be obtained where possible. It will be interesting to see if further well documented cases come to light.

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False Memory Syndrome

Sir – Dr Critchlow is to be congratulated on his effort to balance the evidence in this difficult area (IJPM June 1998). It is an issue that will continue to be debated for many years. The most important fact for psychiatrists to grasp is that false memories of childhood abuse can be induced and often the results cause great suffering in families and in the 'victim'. There are some techniques which are particularly likely to result in false memories, most of them discredited techniques and these will be avoided by sensible doctors.[1] What is not always recognised is that in any therapeutic relationship the therapist may inadvertently communicate his/her beliefs and expectations to the subject. Public and professional education can bring about dramatic changes. The so called epidemic of recovered or false memories seems to have come to an end in the USA though whether this is due to the successful litigation against therapists or widespread professional and media attention remains uncertain.[2] We have a clear duty to avoid the creation of false memories but we must also treat with respect and concern anyone who presents with a story of childhood abuse. Where these are memories recovered after a long period of amnesia then an element of scepticism is justified but psychotherapy rather than confrontation is then called for.

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Fatal pharmacokinetic interaction involving amitriptyline combined with valproate and clozapine

Sir – The purpose of this case history is to draw attention to the possible fatal consequences of combining amitriptyline with valproate and clozapine.

A physically healthy young adult male was diagnosed as suffering from schizophrenia in 1993. This was complicated at times by a significantly depressed affect which required antidepressant medication.

Following failure to respond to standard antipsychotic medication he was commenced on clozapine, with some clinical improvement. However he experienced epileptiform-like phenomena which were confirmed by an EEG. Consequently the dose of clozapine was reduced to 200mg bd and prophylactically he was commenced on valproate 500mg bd, which appeared to successfully alleviate this side-effect. During this time he continued to receive

amitriptyline 150mg for depression. Serum valproate levels were within the normal range. He remained stable on these three drugs for approximately 18 months. Unexpectedly and tragically he experienced a grand mal seizure from which he appeared to recover, but subsequently was found dead a number of hours later. A post-mortem showed no physical cause of death. Toxicology however revealed an amitriptyline blood concentration of 1.5µg/ml. Although interpretation of the post mortem blood amitriptyline concentrations is complex[1] cardiotoxicity can result with tricyclic antidepressant blood concentration in excess of 1.0µg/ml.[2] Such toxicity can begin insidiously, continue silently and become only clinically apparent in a sudden life threatening manner.[2] A careful review of the patient's mental state and behaviour around the time of his death shows no indication of suicidal risk nor that he consumed all overdose of amitriptyline.

This raises the possibility of a pharmacokinetic interaction resulting in a cardiotoxic level of amitriptyline. Valproate significantly effects the pharmacokinetic disposition of amitriptyline, with a 30% increase in amitriptyline area wider the curve, which may result from the combined effect of decreased first-pass metabolism and inhibition of systemic metabolism.[3] There is no information on a possible pharmacokinetic interaction between amitriptyline and clozapine. The possibility is suggested by the reported case[4] of a significant increase (from 93ng/ml to 185ng/ml) in nortriptyline blood concentration when clozapine was prescribed for a patient already taking nortriptyline 100mg/day. It was thought to have been a consequence of competitive inhibitions of CYP2D6 which is responsible for nortriptyline metabolism. This isoenzyme, along with others, is also involved in amitriptyline metabolism.[5]

In the case reported here the patient was prescribed two drugs (amitriptyline and clozapine) with the potential to cause epileptiform activity. When this occurred his dose of clozapine was reduced and commenced on Valproate, which is the anti-epileptic of choice as carbamazepine carries a haematological risk. For 18 months he remained well until he had a grand mal seizure. His death shortly afterwards may have been mediated by cardiac arrhythmia caused by cardiotoxic levels of amitriptyline and in turn by all insidious pharmacokinetic interaction.

Caution is therefore required when a tricyclic such as amitriptyline is simultaneously prescribed with valproate or clozapine, either singly or in combination. If such is ever clinically necessarily, then measuring plasma tricyclic concentrations is indicated.

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