

the allegation. On hospital admission, the patient was hostile and suspicious towards all staff. When asked to give consent for her daughter to be examined by the police surgeon, she refused and, as consent could not be given by anyone else, no physical evidence for or against abuse was obtained.

On recovery, Mrs X stated that the allegations of sexual abuse had been untrue and that she had told her daughter to say that she had been abused. Her daughter again confirmed her mother's story. However, local social workers were now concerned not with whether the child had been sexually abused but with whether she could have suffered emotionally by being party to false accusations. As a result, an initial interim care order was extended and further care proceedings are currently planned.

There are three points to note from this case. Firstly, although Remington & Rosenblat believe it is rare for false accusations of sexual abuse to arise solely from psychosis, it may become more common as sexual abuse joins other preoccupations of our society in the content of delusional psychosis. Secondly, the outcome of such accusations can be far-reaching and serious, in part because of the sensitivity of social workers in this field. If such cases are increasingly common, it is therefore vital that psychiatrists and social workers are able to develop a joint approach to the management of women whose allegations are features of their illness. Thirdly, the case illustrates how a child can be persuaded to confirm a parent's delusions, although whether this is in itself damaging to the child remains a subject for debate.

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Fitness to plead

SIR: The law on fitness to plead is generally held to be highly unsatisfactory (Bluglass & Bowden, 1990). If a jury (and it has to be a jury) finds that a person is unfit to instruct solicitors, challenge jurors and comprehend the nature of his acts or the court proceedings, then the law admits no alternative to an indefinite hospital order with release only by the Home Secretary. Few hospitals will accept patients under such strict circumstances and, to obviate this, many defendants are persuaded to plead guilty to small charges when, in fact, much mental illness fully meets the criteria for unfitness.

It is good to know that British Justice can have a human face – and in a Crown Court which has a famous, if not notorious reputation for a rigorously punitive attitude.

Case report. Mr X, a man in his 30s, was arrested as a result of erratic behaviour culminating in a car chase during which several police vehicles were damaged and officers had to be treated in hospital. He faced eleven charges of actual bodily harm, theft, reckless driving, and property damage, but was (somewhat surprisingly) allowed bail. When a psychiatrist called in by his solicitors saw him, he was immediately admitted to hospital under Section 2 of the Mental Health Act. There had been problems in his life during the year before this episode, but he had always been sane and law-abiding. For several months, however, he had become increasingly unpredictable with unrealistic plans of the most grandiose kind. Although unemployed, he claimed to have made £6 million profit, for which reason Social Security was understandably unwilling to allow his family to claim benefit. His recovery in hospital was swift but he lapsed into an anxiety state with panic and some secondary depression as a presumed result of trying to come to terms with the consequences of what he had done. All memory of the four months of his developing illness seemed to have been obliterated, including a whole archive of manic writings and the circumstances of two broken ankles and the plastering thereof.

Three psychiatrists, including one for the prosecution service, concurred with the diagnosis of hypomania or mania and accepted the amnesia as genuine.

The prosecution decided to offer no evidence, and the judge, after consulting with both counsel in Chambers and reading the reports, decided not to put the issue to a jury. Describing in open court "an extremely serious series of events under ordinary circumstances calling for extreme punishments", he agreed that offering no evidence was the right decision by the Crown. He called attention to the special circumstances of the case and the background of the defendant. Severe warnings were given if Mr X defaulted from treatment and relapsed, and the Crown Psychiatrist's report was to be sent to the Driver & Vehicle Licensing Centre in Swansea. Those advising Mr X were both astonished and happy: the reaction of the police is not known.

BLUGLASS, R. & BOWDEN, P. (1990) *Principles and Practice of Forensic Psychiatry*. pp. 172–177. Edinburgh: Churchill Livingstone.

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Obsessive slowness revisited

SIR: Ratnasuriya *et al* (*Journal*, August 1991, 159, 273–274) discuss preponderance of males in cases of obsessive slowness, and of Parkinsonism, the latter being associated with pathology in the basal ganglia

and substantia nigra. The neurobiology has in fact been demonstrated in the case of a 17-year-old boy manifesting reduction of obsessive slowness in response to fluoxetine, with return to normality of decreased tracer deposition on brain scanning in the basal ganglia and adjacent temporal lobe of the right hemisphere, in which the metabolic rate is lower in males (Friedman, 1990). This neurobiology is also demonstrated by Parkinson's disease of the right hemisphere manifested by elongated pauses (Friedman, 1987). Neural circuits concerned with acting on decisions and completing the ensuing behavioural sequences may be more sensitive to damage in males. This is suggested both by frontal damage, which may cause an inability to reactivate previously generated intentions, after a delay, when they are not directly signalled by the stimulus situation (Shallice & Burgess, 1991), and by the fact that delay-dependent speeding of reaction time, reflecting motor readiness, is abolished by depletion of dopamine (Brown & Robbins, 1991) subserving the mesocorticolimbic system (Csernansky *et al*, 1991).

BROWN, V. J. & ROBBINS, T. W. (1991) Simple and choice reaction time performance following unilateral striatal dopamine depletion in the rat: impaired motor readiness but preserved response preparation. *Brain*, **114**, 513–525.

CERNANSKY, J. G., MURPHY, G. M. & FAUSTMAN, W. O. (1991) Limbic/mesolimbic connections and the pathogenesis of schizophrenia. *Biological Psychiatry*, **30**, 383–400.

FRIEDMAN, E. H. (1987) More on Hitler and Parkinson's disease. *New England Journal of Medicine*, **316**, 114.

— (1990) Dopamine and obsessive symptom expression. *Neuropsychiatry, Neuropsychology and Behavioral Neurology*, **3**, 313.

SHALLICE, T. & BURGESS, P. W. (1991) Deficits in strategy application following frontal lobe damage in man. *Brain*, **114**, 727–741.

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Musical hallucinations triggered by clomipramine?

SIR: We would like to report a case of musical hallucinations similar to other cases recently reported in this journal (Fenton & McRae, 1989; Berrios, 1990; McLoughlin, 1990), but associated with the use of clomipramine.

Case report. A 67-year-old widowed female was referred for consultation about her anxiety and depressive symptoms which had gradually become incapacitating over the previous five years. Her medical history included asthma since childhood, deteriorating eyesight due to uveitis, cataract and trachoma, and a decreasing ability to discriminate

between both sounds and words, notably over the previous three years. ENT consultation and audiometric tests showed a slight deafness at high frequencies, worse on the left. She reported childhood experiences of sudden inversion of visual images, as if she were looking in a mirror. A neurological evaluation including CT scan, clinical EEG, and routine laboratory tests was normal. A diagnosis of panic/agoraphobia with secondary depressive symptoms was made, and she was prescribed clomipramine in increasing dosage up to 50 mg/day. Within two weeks her panic attacks had ceased. During week three the dosage was raised to 75 mg/day because of residual depressive symptoms. She felt sedated and drowsy for two days, and then noticed that she was hearing music: the national anthem, hallelujahs, and other choral pieces. The sounds were so clear and loud that she thought they were coming from the street. She soon realised that the music was coming from her own ears. In the beginning the experience was pleasant and sometimes she could select the music at will. It was always familiar songs she herself had sung in a chorus years before. Sometimes music heard during the day would return at bedtime. Within days, the experience became distressing. The music became louder and out of control, speeding up or scratching like a damaged record. The dosage of her clomipramine was reduced to 35 mg/day, and this led to a reduction in volume. She remained for a year on this dosage. She continued to work and, living alone, was competent in her activities. She had no panic, phobic avoidance or depression. The music remained, low in volume, and pleasant. A reduction in her clomipramine to 10 mg/day led to a relapse of panic and breathlessness. Even at this low dosage she continued to hear the music but only in quiet places. The dosage was first raised to 30 mg and then kept at 10 mg/day. She has continued to have tolerable musical hallucinations.

Musical hallucinations (MH) are a rare phenomenon, the actual incidence of which is unknown. They can be associated with a variety of clinical situations such as diseases of the ear, brain tumours, depression, alcohol and drugs. Typically, MH have been associated with marked hearing loss, advanced age, and female sex. The hallucinations are usually vivid, pleasant, and often consist of familiar hymns and songs. The mechanism involved in the production of these hallucinations is unclear. MH may result from a combination of peripheral ("sensory-deprivation") and central nervous system dysfunction. It is conceivable that its production involves memory retrieval (Keshavan *et al*, 1988; Fenton & McRae, 1989; Berrios, 1990).

The temporal association with use of clomipramine reported in this case, and its apparent dose relationship suggest a direct pharmacological effect, perhaps on the right temporal lobe (Berrios, 1990), although the brain area of origin of MH is still controversial (Keshavan *et al*, 1988). Anticonvulsants may attenuate MH in some, but not all, patients (Keshavan *et al*, 1988; Berrios, 1990). This patient's increasing