Third Ventricle Cyst: A Difficult Diagnosis

SIR: Upadhyaya & Sud (*Journal*, April 1988, **152,** 567–569) reported difficulties in diagnosing third ventricle cysts. We describe another patient whose psychological and physical symptoms were hard to tie together.

Case report: Mrs A. was a successful professional Jewish woman in her 40s with an underachieving unhappy husband and four children. Ten years ago the family tried family therapy and EST, prompted by the second child's behavioural problems. Eighteen months ago Mrs A. became exhausted and forgetful, and moved to a less demanding job. As she was post-menopausal, with hot flushes and sweating, her GP referred her to the menopause clinic where she received a hormonal implant, with little effect. Through a medical friend, she sought a physician's opinion privately. She now also complained of waking early, a weight gain of 12 kg, breathlessness on effort, and "dragging feet towards the end of the day". Physical examination, as well as metabolic and endocrine blood tests and respiratory function tests, were normal. The physician diagnosed primary depression and referred her non-urgently to psychiatrist I, who was sent her notes.

Meanwhile Mr A. took a large overdose. The medical friend involved psychiatrist II, who admitted Mr A. under psychiatrist III. Mrs A. felt desperate and suicidal and was referred by psychiatrist II to psychiatrist IV for emergency psychiatric assessment. Psychiatrist IV arranged admission under psychiatrist V on our psychosomatic ward.

On admission Mrs A. seemed to give a full history but forgot to mention her shuffling and recent headaches. This information could only later be obtained, as psychiatrist I still had the medical notes and Mr A. was too distressed to be interviewed. Mrs A. was tearful and depressed. Bedside cognitive testing was normal. There were no abnormal physical signs.

Psychiatrist III's team felt that family treatment was urgently needed and proposed a total system review including all those involved with the couple. At the time a major disaster was draining resources away from our ward. We did not know Mrs A. well enough to be confident about starting the system review, and decided to assess her further in her own right, taking stock of information already gathered. We wanted to wait for any psychological benefits of her recent high-dose hormone implant. We hoped that her complaint of poor memory would resolve with lifting depression and postponed psychometric testing.

Away from her family Mrs A.'s mood improved, but there were still sufficient depressive features to justify imipramine treatment. Over the next month her mood improved; instead, she emphasised her memory problems and her shuffling which she had first noticed on a visit to Jerusalem 15 months before. Bedside cognitive testing remained normal, but Mrs A. repeatedly forgot appointments and names. Full psychometric assessment revealed an IQ of 106 and marked discrepancy between verbal and performance scales. On repeat physical examination Mrs A. was ataxic and had early bilateral papilloedema with a flame haemorrhage. A CT scan demonstrated a third ventricle cyst. Rapid deterioration followed, so dexamethasone was started before the cyst was excised. She made a good post-operative recovery and 3 months later her performance on psychometric testing showed above-average intelligence (IQ = 131) and short-term memory.

Different doctors had interpreted Mrs A.'s story as a clear-cut case of a depressed menopausal woman in family crisis. Organic illness had been excluded. The many contaminating factors from Mrs A. and her family and also within our own system impeded our diagnostic process considerably. We too initially failed to recognise the symptoms of slowly developing hydrocephalus, despite all early pointers. Only once we had assimilated all the information on Mrs A. and observed her further deterioration did we pick up the extremely rare third ventricle cyst.

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Naloxone treatment for opiate withdrawal syndrome

SIR: In an open-trial study with five patients, Vlissides *et al* (*Journal*, April 1988, **152,** 565–567) could not reproduce Hendrie's (1985) experimental findings, that the abstinence syndrome in opiate addiction is attenuated by naloxone. All patients experienced severe withdrawal syndromes within minutes after intravenous administration of an unspecified dose of naloxone.

We have treated 12 opiate addicts (2–10 year's addiction history, satisfying DSM–III–R criteria) for detoxification by an acute intravenous administration of a single dose of naloxone (10 mg) during a short (30–60 min) general anesthesia followed by continuous administration of 0.8 mg/h naloxone for a further 24 hours. After awaking from anesthesia, all patients showed no or only mild withdrawal signs, although naloxone treatment was continued. Six of these patients were then treated further with single doses (0.4 mg naloxone) every two hours for an additional 24 hours. In contrast, the other patients were treated with 0.4 mg/h naloxone intravenously as long as their urine was found opiate positive (approximately 72 hours). Opiate excretion in urine was monitored in all patients by EMIT-dau test. The urines of the patients were found to be opiate-positive for approximately 72–96 hours after the last opiate intake. In the first group, mild withdrawal signs (goose flesh, stomach pain, insomnia) were seen only after naloxone treatment had been discontinued, and could be still observed as long as the urine

of these patients was opiate positive. In the second group of patients, withdrawal symptoms could be observed neither during the continuous naloxone treatment nor after cessation of this drug therapy as soon as opiate excretion in urine was no longer measurable.

Our observations, which we will report in detail, seem to confirm the experimental data of Hendrie (1985), that withdrawal symptoms will be attenuated by high doses of naloxone. A short anaesthesia was administrated in these patients, because during the flooding phase of naloxone in the body, acute withdrawal symptoms had to be expected acutely after administration of a high dose of naloxone (Jasinsky *et al*, 1967). This seems to be why the naloxone treatment was acutely discontinued and not followed up in the study of Dr Vlissides *et al*. According to our hypothesis, the paradoxical attenuation of withdrawal symptoms may be observed only when the naloxone in the body reaches a certain level. Furthermore, from our study it can be concluded that naloxone treatment should be continued for as long as morphine alkaloids are present in the body.

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Temporal Lobe Epilepsy

SIR: We read with interest the article by Herzberg & Fenwick (*Journal*, July 1988, **153**, 50–55), and we would like to add our experience regarding this important topic. From a prospective sample of 65 temporal lobe epileptics (TLEs), derived from a variety of sources including a neurological clinic and private practice, 15 (23%) manifested aggression, according to the relevant outlines of Bear & Fedio (1977), as estimated by two psychiatrists by means of psychiatric interview and additional information from the patient's relatives.

The aggressive behaviour, as estimated by psychiatic interview as well as from the index of aggressiveness of the MMPI, was associated with early onset of seizures (and especially with onset before the age of 5). No relationship was found between aggression and EEG features or frequency of seizures. However, we did not find any relationship of the aggressive behaviour with the male sex, nor with the current age of the patients.

We cannot agree with the statement of Drs Herzberg & Fenwick that the existence of temporal lobe personality is dubious. We found that from the 65 TLEs, 16 (24.6%) who received a DSM–III diagnosis of organic brain syndrome (organic personality syndrome and/or organic delusional syndrome or organic affective syndrome) were also differentiated significantly from the rest of the TLEs and a control group of 36 non-TLEs in 9 out of 18 of Bear & Fedio's personality traits (Garyfallos *et al*, 1988). Those traits were religiosity, sexual alteration, aggression, viscosity, paranoia, circumstantiality, humourlessness, hypergraphia, and hypermoralism. We believe that a minority of TLEs manifest some personality traits. Not every patient who has traits has all of them, and perhaps the traits are not exactly the 18 reported by Bear & Fedio (1977). Furthermore, more recently Bear (1986) wrote that "not every patient with temporal lobe epilepsy develops interictal behaviour changes or changes to the same degree".

Concerning the aetiology of aggression in TLE, we agree that this cannot be attributed to the epilepsy itself, i.e. to the actual occurrence of seizures. However, we speculate that both the epileptic seizures and the aggression (but also other psychopathological and behavioural manifestations) are the result of the same cause, namely the limbic epileptic focus, or more correctly "the intermittent limbic spike focus" as Geschwind (1983) suggested. This does not necessarily mean that there is a structural brain damage detectable in the CT scan. The phenomenon of kindling, which was proposed as an experimental model of epilepsy, gives a possible explanation. Its action is not limited to the development of epileptic seizures, but also leads to interictal aggressive behaviour (Goddard, 1980) as well as to other emotional and behavioural alterations. It is possible that for the development of aggression through the kindling procedure, some indispensable conditions are needed. One of these can be its influence from very early age. According to our findings, the aggressive subjects were significantly more likely to experience onset of seizures before the age of 5. Furthermore, Geschwind (1983) reports that the influence of kindling is different in intrauterine life, in childhood