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Steroid-induced catatonia

SIR: The case report "A steroid stupor in a surgical ward" (Doherty *et al*, *Journal*, January 1991, **158**, 125–127) describes the development of secondary catatonia and stupor in a 17-year-old man. The patient had symptoms of bowel disease, became increasingly distressed at his failure to improve, and developed a catatonia syndrome upon the administration of steroids. He was treated with haloperidol and chlorpromazine for four weeks and then received ECT after which "he made an excellent response to a course of five treatments."

Anti-psychotic drugs were apparently given in the belief that the catatonia was a manifestation of a psychosis for which these drugs are deemed effective. But ECT has been known as an effective treatment for catatonia since the first experiments of Meduna with convulsive therapy in 1934, and those of Cerletti & Bini in their introduction of electroconvulsive therapy in 1938. There has not been any compelling evidence since then that any other treatment is as effective as ECT, although transient improvement has been reported after intravenous amobarbitone or thiopentone. Some authors have recently suggested that catatonia may be responsive to multiple doses of lorazepam, but such observations need verification and comparison with ECT before being accepted.

Present classification of catatonia as a subtype of schizophrenia, as in DSM–III, leads to the mind-set exhibited in this case: catatonia is a manifestation of schizophrenia and must be responsive to anti-psychotic drugs. But such a connection is fallacious. Catatonia is often described in patients with mania, depression, infections, endocrinopathy, and as in this case, secondary to drug toxicity. We have recently argued that catatonia should be considered a separate class in DSM–IV, distinct from schizophrenia (Fink & Taylor, 1991). Such a reclassification would have encouraged the physicians treating this patient to introduce ECT at the earliest opportunity, allowing the patient to recover sufficiently for any specific treatments for his bowel disease without the four week delay.

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Three thousand days of pregnancy

SIR: Recently, DePauw (*Journal*, December 1990, **157**, 924–928) reported an interesting case of monosymptomatic hypochondriacal psychosis (MHP) in which the somatic delusion was that of pain and pseudocyesis. This case, as reported, presented a major treatment problem as the patient's pain and delusional belief appeared to be refractory to a large number of psychotropic agents and ECT. In addition, although pimozide appeared to be effective, the patient was inconsistent in her response to this drug and finally required (40 mg), almost four times the dose of pimozide usually necessary to obtain symptomatic relief in MHP.

These observations prompt me to raise two questions about this case:

(a) As Dr DePauw points out, pimozide is an opiate antagonist. At the dose of pimozide utilised, could the patient's improvement be simply attributed to pimozide's effect on pain perception?

(b) Recent reports (Ross *et al*, 1987; Hollander *et al*, 1989; Fishbain & Goldberg, 1991) indicate that some forms of monosymptomatic hypochondriasis (MH) and MHP could selectively respond to serotonin reuptake blockers such as clomipramine and fluoxetine. Because of the success of the serotonin reuptake blockers where other agents have failed, one author (Ross *et al*, 1987) has suggested a controlled double-blind study of pimozide v. clomipramine for the treatment of MH–MHP. It is to be noted that no serotonin reuptake blockers were used in the treatment of this patient. One wonders if these would have had some measure of success?

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