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Probing dexfenfluramine

SIR: The recent article by O'Keane *et al* (*Journal*, May 1992, **160,** 643–646) on the use of dexfenfluramine was most interesting. However, one wonders how predictable that response to dexfenfluramine is, in terms of its neuroendocrine action? Rowland & Carlton (1986) have indicated that the plasma half-life of this compound is around 24 hours, with a half-life of about four hours in brain tissue, and one wonders how the neuroendocrine parameters might vary within this time frame?

Our own experiences with this compound have led us to believe that the drug is quite capricious in terms of its effects. Three patients taking dexfenfluramine have recently come to our attention, and their responses have been vastly different. The first, a middle-aged woman, slept solidly for 72 hours after a single dose of dexfenfluramine. She subsequently stopped taking the drug. The second, a young woman, became acutely suicidal following a three-week course of dexfenfluramine, and the third, a man in his 40s, presented with agitation and dysphoria following a four-week course of the drug. Both of these patients were euthymic following discontinuation of the compound.

The manufacturers of dexfenfluramine assure us that the preparation is safe and effective, but the recent work by Ricuarte *et al* (1991) would suggest that the potential for neurotoxicity with dexfenfluramine is greater than previously believed. Our clinical experiences have made us wary of this drug, as we have found it to be quite unpredictable in terms of its affects.

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Carbamazepine and episodic dyscontrol

SIR: Lewin & Sumners describe remission of aggression with carbamazepine, in a chronically irritable, brain-damaged man (*Journal*, August 1992, **161,** 261–262), and argue that the lesion may be frontal or temporal. However, they make a diagnosis of episodic dyscontrol (ED), while admitting that their patient falls short of DSM–III–R Intermittent Explosive Disorder, which excludes cases with between-episode aggressiveness.

Although the term episodic dyscontrol is often used loosely, an intermittent or 'out of character' quality of the aggression is central to the syndrome, and patients often express extreme remorse (Malatsky, 1973). This is reminiscent of the co-occurrence of aggression and 'hypermoralism' in temporal lobe epilepsy (TLE; Waxman & Geschwind, 1975). Indeed, TLE-like symptoms are frequent in ED (e.g. Maletsky, 1973). I have recently treated three young male out-patients, who presented with many years of worsening outbursts of violence on minimal provocation, with amnesia for, but not denial of, the assaults. All reported olfactory hallucinations of burning, and two, *déjà vu* phenomena. Two had psychotic features. All had long periods of placidity between episodes. Only one had electroencephalogram (EEG) abnormalities. Carbamazepine produced complete remission in all three, followed by relapse after non-compliance.

The anticonvulsant phenytoin was effective in 19 of 22 cases of ED (Maletsky, 1973), so it is no surprise that carbamazepine, with its specific effect on limbic structures, should be effective in ED, particularly in the presence of TLE-like symptoms.

Episodic dyscontrol is best understood as paroxysmal violence, due to epilepsy-like dysfunction of limbic structures in the temporal lobe, which responds well to anticonvulsants.

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