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# Successful Treatment of Episodic Dyscontrol with Carbamazepine

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**Following a road-traffic accident, an 18-year-old man developed episodic dyscontrol which brought him into conflict with the law. Two years after the accident, treatment with carbamazepine was initiated and further aggressive outbursts subsided. The efficacy and mode of action of carbamazepine is discussed and the literature reviewed.**
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Bach-Y-Rita *et al* (1971) studied 130 patients with episodic dyscontrol (ED), and found that a large proportion of these patients had histories of birth injury, mental retardation, coma-inducing illness, head injury, episodes of unconsciousness, and seizure-like episodes. Investigations revealed a high incidence of abnormality on electroencephalography (EEG) and psychometric testing. Most patients with ED may not be diagnosed as having intermittent explosive disorder according to DSM–III–R as they do not meet the third criterion: absence of generalised impulsiveness or aggression between episodes.

The relationship between aggressive behaviour and neurological illness is controversial (Shaffer *et al*, 1980; Stevens & Herman, 1981). Elliott (1982) investigated 286 patients with episodic dyscontrol and found that 94% had evidence of brain dysfunction: primarily minimal brain dysfunction, complex partial seizures, or a history of significant brain trauma. Mark & Ervin (1970) summarised several studies which showed high but variable rates of electroencephalographic abnormalities in people prone to violence.

We report a young man who suffered a brain injury in a road-traffic accident at the age of 18. Following the head trauma he appeared to undergo a personality change, developing irritability and violent outbursts which brought him into conflict, for the first time, with the law.

## Case report

The patient was run over by a car while riding a motorcycle at the age of 18. In the casualty department he complained of diplopia, altered smell and taste sensations, and back pain. Examination showed left facial weakness, left-sided deafness and a watery discharge from the left ear. His level of consciousness was reduced. A subsequent computerised tomography (CT) scan revealed generalised brain oedema, blood in the ventricular system and some intracranial air. The patient was discharged after several weeks of observation.

He was seen by a neurologist six months later who noted that his mood fluctuated. Four months later, the same neurologist noted irritability and violent outbursts which were not within the patient's control.

Fourteen months after the incident, the patient was assessed by a neuropsychologist who noted marked cognitive deficits and a tendency to perserverate. The patient had difficulty in abstract reasoning and there was impairment of verbal and visual memory. The neuropsychologist concluded that there was evidence of bilateral frontal and temporal lobe lesions.

At this time the patient came into conflict with the law, and was charged with actual and grievous bodily harm. He was subsequently assessed by a psychiatrist who noted that the patient had violent outbursts which he could not control. In addition, there were signs of depression: tearfulness, anxiety, poor self-image, and suicidal ideation.

Two years after the accident the patient was assessed at the North West Thames Regional Health Authority Brain Injury Rehabilitation Unit (BIRU). On examination he was

found to be irritable and intolerant of other people. His family reported that this contrasted markedly with his previous personality, which was an easy-going and pleasant one. The diagnosis of ED was made because of the presenting features of uncontrolled disproportionate episodic violence. For the first few weeks, treatment was conducted under out-patient supervision and carbamazepine was prescribed, building up to a dose of 300 mg b.d. At the first follow-up appointment after commencing treatment, the patient appeared depressed and tearful but less irritable. His family reported that he was much calmer and no further outbursts had occurred. As the dose of carbamazepine was increased, he became less depressed. The patient was subsequently admitted to make a more comprehensive assessment and to provide rehabilitation in the form of assertiveness and social-skills training. While on BIRU the patient showed no aggressive behaviour. He was always polite and friendly towards staff and other patients, even when occasionally provoked, but repeatedly expressed his boredom.

He soon felt that he did not need any further in-patient treatment or medication, but after some persuasion compromised on a lower dose. Medication was reduced slowly to carbamazepine 200 mg nocte and the patient was discharged on this dose. There have been no violent outbursts during 12-month follow-up.

An EEG performed during the admission showed a mild excess of temporal theta activity for his age. There were no spikes, sharp waves or complexes.

## Discussion

Carbamazepine has been widely used in the treatment of aggressive behaviour. Neppe (1982) found carbamazepine effective in controlling aggressive behaviour in psychotic patients with non-specific temporal lobe EEG abnormalities. Luchins (1983) noted a significant decrease in the number of aggressive episodes in violent psychiatric patients without any EEG abnormalities.

Gardner & Cowdry (1986) found carbamazepine to be significantly superior to propranolol in decreasing the severity of loss of behavioural control in patients with borderline personality disorder. They suggested that carbamazepine may have a specific anti-aggressive effect due to anti-kindling activity in the limbic system. Foster *et al* (1989) conducted a double-blind pilot study and found that patients with frontal lobe damage showed improvements on cognitive, affective, and behavioural measures with carbamazepine treatment. It may be concluded that carbamazepine is useful in the treatment of aggression caused by either frontal lobe or limbic system dysfunction (Tunks & Dermer, 1977).

Our patient presented with episodic dyscontrol but like many brain-injured patients did not meet the third DSM-III-R criterion for intermittent explosive disorder, as he was described as generally irritable. There was evidence to suggest that the aggressive outbursts were based on the brain injury: a repeat CT scan was not performed but psychological testing demonstrated bilateral frontal and temporal dysfunction and the EEG confirmed mild abnormalities of the temporal lobes. It remains unclear whether the patient's aggressive behaviour was caused by a lesion in his limbic system or in his frontal lobe.

In this case, treatment with carbamazepine resulted in a diminution of aggressive behaviour before admission, after which the other components of rehabilitation were initiated. Indeed, without drug therapy, admission would have been most difficult because of the patient's aggressive attitude towards the Unit's most disabled patients. In such circumstances, a trial of carbamazepine seems well justified.

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