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Failure to Convulse with Electroconvulsive Therapy

A Case Report

A case is reported of a severely depressed patient in whom treatment with electroconvulsive therapy was abandoned because of repeated failure to induce a convulsion. Reasons for the failure to convulse and alternative treatment approaches are discussed.

There is now substantial evidence that electroconvulsive therapy (ECT) is an effective treatment for severe depression (Johnstone *et al*, 1980; Brandon *et al*, 1984; Gregory *et al*, 1985). This treatment produces a generalised convulsion by passage of electric current through the brain. The convulsion manifests as a brief initial stimulus-related tonic and subsequent longer clonic phase. It is the latter that is associated with therapeutic efficacy (Ottoson, 1962). Failure to recognise non-occurrence of the clonic phase is common (Pippard & Ellam, 1981). Careful monitoring can avoid this pitfall (Fink & Johnson, 1982). We describe a case in which no clonic phase was observed during 15 treatment applications despite clinical and 'cuff' monitoring (Addersley & Hamilton, 1953).

Case history

A 32-year-old right-handed woman of mixed African and Indian extraction, and with a two-year history of recurrent depressive disorder, was admitted to hospital during her third episode. This was characterised by unremitting low mood, early morning wakening, reduced appetite, weight loss of 5 kg and a suicide attempt. Treatment with amitriptyline and chlorpromazine for four weeks effected no improvement. ECT was then prescribed and administered in eight sessions over four weeks with a total of 15 applications (see Table I).

Convulsions were monitored for the first eight applications by clinical observation only. This was supplemented for the remaining seven applications by the 'cuff' method: a sphygmomanometer cuff was placed around the right upper arm and was inflated to above systolic pressure prior to the injection of succinylcholine, thus allowing observation of unmodified clonic contractions. On each occasion that current was applied a brief tonic phase was observed, but this was never followed by a clonic phase indicative of continuing seizure activity.

Scrupulous attention was paid to electrode application: the hair was parted and the skin was cleaned with Savlon. Lint-covered steel electrodes were soaked for 30 seconds in Ectronolyte and applied with firm pressure. For unilateral applications the electrode positions were temporoparietal

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TABLE I
ECT stimulus characteristics, anaesthetic dose and additional measures in 15 unsuccessful applications[1]

Treatment session	*Electrode position*	*Stimulus*[2]	*Anaesthetic*		*Pretreatment*	
			Methohexitone: mg/kg	*Succinylcholine: mg/kg*	*Oxygen*	*Chlorpromazine*
1	Unilateral	E2, E2	1.4	0.8	–	–
2	Unilateral	E2, E2	0.9	0.7	–	–
3	Bilateral	E1, E1	1.1	0.8	–	–
4	Bilateral	E1, E1	0.9	0.7	–	–
5	Bilateral	E1, E1	1.1	0.5	+	+
6	Bilateral	E1, E1	0.5	0.7	+	+
7	Bilateral	T	0.5	0.4	+	+
8	Bilateral	T, T	0.9	0.7	+	+

1. Amitriptyline and chlorpromazine were taken as regular medications throughout this period.
2. E1 = Ectron Duopulse, setting 1 (modified sine wave); E2 = Ectron Duopulse, setting 2 (modified sine wave); T = Theratronics Transpsycon, setting 2 PH (biphasic pulses) at 50 J.

as described by Lancaster *et al* (1958). For bilateral applications the electrodes were positioned bitemporally. The machines used were the Ectron Duopulse Mark IV and the Theratronics Transpsycon ZUSS/3. The Ectron is a transformer model; settings 2 and 1 were selected to produce a modified sine wave of nominal 25 joules and 36 joules respectively. The Theratronics is a capacitor-based machine producing 5 millisecond pulses and was used in a biphasic mode at a nominal 50 joules setting. Anaesthesia was achieved with methohexitone and succinylcholine only (see Table I for doses). Medication during treatment comprised amitriptyline (150 mg *nocte*) and chlorpromazine (50 mg *t.d.s.*). No other sedative drugs were prescribed. A skull X-ray revealed no abnormally thick bone. For treatments 9–15 inclusive the following steps were taken, each with the aim of lowering seizure threshold: (a) reduction in methohexitone dose; (b) preoxygenation over 20 seconds; (c) an additional dose of chlorpromazine (100 mg intramuscularly) one hour before treatment.

ECT was abandoned after 15 unsuccessful applications. The patient's clinical condition remained unchanged.

Discussion

It might be argued that cerebral seizure activity did in fact occur but was inaccessible to our monitoring techniques. We think this is unlikely. While it has been shown that clinical observation underestimates occurrence of seizures when compared with the electroencephalogram (EEG) (Christensen & Koldbaek, 1982), there is evidence that the cuff method which we used is of comparable sensitivity to the EEG (Fink & Johnson, 1982). In our case the factors resulting in non-occurrence of a convulsion may be divided into two categories: first, the electrical stimulus used and, secondly, the patient being treated.

For a convulsion to occur, an adequate electrical stimulus is required: this is determined by electrode application, placement and the characteristics of the machine. Poor contact between electrodes and skin, resulting in inadequate stimulus conduction, is a common reason for 'missed' seizures (Fraser, 1982). We believe that careful attention to recommended technique (Fraser, 1982) makes this unlikely in our case. Unilateral electrode placement is associated with more missed seizures than bilateral (d'Elia & Raotma, 1975; Gregory *et al*, 1985; Pettinati & Nilsen, 1985). One reason is the relatively small separation of the electrodes. The likelihood of a seizure is increased by greater separation of unilateral electrodes than in the standard Lancaster position (Weiner, 1980). Faced with failure to convulse, we could have tried a more widely separated unilateral electrode placement (d'Elia, 1970), but we chose to move directly to bilateral treatment after four failed unilateral applications. The specific characteristics of the electrical stimulus depend on the machine, its functioning, type, and setting. The machines we used were regularly serviced and effective in producing convulsions in patients treated concurrently. The Ectron Duopulse IV, on the settings used, produced a modified sine wave. This was the machine and setting used in three recent trials of ECT (Johnstone *et al*, 1980; Brandon *et al*, 1984; Gregory *et al*, 1985) with no reported failure to convulse. As it has been suggested that pulsed current is more effective in producing seizures (Weaver *et al*, 1974) the Theratronics machine, delivering high energy pulses, was substituted, but to no avail. We therefore think it likely that factors within the patient were responsible for the failure of treatment to induce a convulsion.

There is much individual variation in total head impedance and this will influence the actual amount of current delivered to the brain (Gordon, 1982). The proportion of this current passing into the brain will depend on the electrical properties of cranial tissues,

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especially bone (Gordon, 1981). Abnormal thickness and calcification of the skull in our patient was excluded by a normal skull X-ray. Further, the repeated occurrence of a brief tonic seizure indicates the passage of current through skull into brain. It is also possible that our patient had an abnormally high seizure threshold. Amitriptyline and chlorpromazine are known to reduce seizure threshold and may be expected to enhance seizure production (Tyrer, 1982). The only drug administered to our patient reported to raise seizure threshold was the anaesthetic agent methohexitone (Fink, 1979). Reduction of the dose in later treatments did not result in a convulsion.

We must conclude that our patient's failure to convulse resulted from a high seizure threshold, and that this was likely to be a constitutional feature rather than an effect of medication.

Failure to convulse may be an occasional and unrecognised cause of non-response to ECT. In order for this problem to be detected, adequate monitoring of seizure activity is required. If no convulsion is observed, attention should be directed first to the production of an adequate stimulus, and secondly to electrode position and contact (Fraser, 1982). Recent surveys have suggested that failings in basic techniques are common (Pippard & Ellam, 1981; Latey & Fahy, 1985).

If, as in our case, the patient still does not convulse, the medication should be reviewed; benzodiazepines (Stromgren *et al*, 1980) and barbiturate anaesthetic agents (Fink, 1979) both elevate seizure threshold. A skull radiograph should be done to exclude abnormal bone thickness. Finally, measures aimed at artificially lowering the seizure threshold may be considered. We administered chlorpromazine and oxygenation from treatment 9 onwards; these measures could have been introduced earlier. Other workers have used pentylene-tetrazol (Fink, 1979) or caffeine (Shapira *et al*, 1985).

As an alternative, a non-convulsive treatment may be considered. In our case the addition of lithium carbonate to the existing drug regimen (de Montigny *et al*, 1983; Pai *et al*, 1986) was followed by a rapid and complete recovery.

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