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David A. Fishbain
South Shore Hospital and Medical Center
600 Alton Road
Miami Beach
Florida 33139

Tardive dyskinesia, depression and ECT

Sir: It has been variously reported that ECT may precipitate (Flaherty *et al*, 1984) or ameliorate (Price & Levin, 1978) tardive dyskinesia in depressives. Furthermore, Keshavan & Goswamy (1983) have observed that tardive dyskinesia in bipolar affective illness is worse during the euthymic phase than during the depressive phase. The opposite has also been reported (Cutter *et al*, 1981).

Case report. An 83-year-old female patient had a six-year history of tardive dyskinesia induced by prochlorperazine maleate prescribed for dizziness at that time. She was admitted with a major depression with psychotic features (DSM–III), (Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960) = 24) and at that time scored 22 on the AIMS scale for tardive dyskinesia. HRSD and AIMS ratings were taken one day before and one day after each ECT treatment and one week after the final ECT (8th unilateral nondominant ECT). AIMS and HRSD scores before and after each of the 3rd to 8th ECT showed a mean fall of 1.4 points on each scale. Overall falls from before ECT to one week after treatment were 24 to 13 for the HRSD, and 22 to 16 for the AIMS.

This patient's tardive dyskinesia was clearly worse during the depressive phase of the illness and improved (but was still present) at recovery. Each treatment with ECT was associated with improvement in mood and tardive dyskinesia and this occurred in parallel. This case study supports the observation that tardive dyskinesia is worse in the depressive phase than in the euthymic phase and that ECT does not worsen the condition. The improvement with ECT may be due to the improvement in the depressive illness but an additional direct effect on tardive dyskinesia may also occur.

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J. A. O. Besson
A. N. Palin
Department of Mental Health
University Medical Buildings
Foresterhill
Aberdeen

A case of depression responding to spontaneous epilepsy but not ECT

Sir: We wish to report the case of a severely depressed man with a long history of manic–depressive psychosis whose depression improved following spontaneous grand-mal epilepsy, but not following repeated electroconvulsive therapy (ECT).

Case report. The patient was a 67-year-old author, with a 35-year history of manic–depressive psychosis. He had been an in-patient with a resistant psychotic depression for two years before his current episode, but had eventually responded to a combination of amitriptyline, phenelzine, and lithium. He had failed to respond to a course of 10 × bilateral ECT given twice weekly. Six months later the patient had a brief manic illness, and then started becoming increasingly depressed again. It was decided to start him on carbamazepine, initially 100 mg t.d.s. and then 200 mg t.d.s. Three weeks after starting carbamazepine his condition deteriorated further, and he had a depressive psychosis with profound psychomotor retardation and nihilistic delusions. He stopped eating and taking medication. ECT was instituted, but the first ECT failed to produce any improvement. Some three nights later the patient had a total of five spontaneous grand-mal seizures lasting 1–2 minutes before responding to intravenous valium. Despite full investigation, no cause for these was found and they were ascribed to acute carbamazepine withdrawal, which was not restarted.

Some 36 hours following the spontaneous epileptic seizures the patient's mood was noted to be radically different. He was no longer depressed, was laughing and talking appropriately, and was eating and drinking normally. Over the following 10 days his condition deteriorated and it was decided to reinstitute ECT. Over the next six weeks the patient had a further 11 ECTs, which all produced bilateral seizures lasting at least 25 seconds, but he did not improve. The patient's depression again proved resistant, only improving some eight months later on high doses of tricyclic antidepressants and monoamine oxidase inhibitors.

As far as we are aware, this is the first reported case in which spontaneous grand-mal seizures have improved the clinical state of a patient with depression, while ECT has had no effect. This differential response may possibly have been due to either the differences in time course between the spontaneous and induced seizures, or because the patient received

submaximal ECT, perhaps due to residual effects of his carbamazepine. However, when multiple ECT has been given therapeutically in one session, it has not led to improved clinical efficacy. In a study of 38 patients receiving multiple ECT, only one patient improved after the first session (Abrams & Fink, 1972). It is also unlikely that the patient's ECT was submaximal since all 11 ECTs produced bilateral seizures lasting from 25 seconds to 100 seconds. It is also unlikely that carbamazepine would have affected seizure generation up to eight weeks after the last dose. We are thus uncertain as to the mechanism underlying this patient's differential response to spontaneous seizures and ECT, and would be very interested to hear of any other similar cases.

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PETER SILVERSTONE
MRC and University Department of Pharmacology
Radcliffe Infirmary
Oxford OX2 6HE

THOMAS FAHY
Bethlem and Maudsley Hospital
Denmark Hill
London SE5

Nifedipine-induced depression

SIR: Eccleston & Cole (1990) recently reported a case of treatment-resistant depression associated with nifedipine. They also cited Hullett *et al* (1988) who described four cases of depression associated with nifedipine, including one case of treatment resistance. A computer-based literature search failed to produce any further similar case reports.

The following is a description of a case of major depression with melancholia and mood-congruent psychotic features, further characterised by a positive dexamethasone suppression test, associated with nifedipine:

Case report. Mr A., a 66-year-old property developer, presented with a two-month history of severe agitated depression associated with delusions of poverty. Symptoms included total anhedonia, loss of energy and interest, hopelessness, guilt, suicidal ideas and nihilism for the future. He was unable to concentrate or make decisions and had completely lost confidence. He was restless and agitated, had a high level of anxiety and had experienced several panic attacks. He could not stop worrying about his financial state, believed that he was bankrupt and 'ruined' and could not be reassured by evidence to the contrary. Associated symptoms included middle and terminal insomnia, anorexia with weight loss of 7 kg and marked diurnal mood variation.

He had no past personal or family history of psychiatric illness. Medically, Mr A. had never had any serious illnesses. Mild hypertension had been discovered three months before the onset of his depression, initially treated with alphamethyl dopa, but this was changed to nifedipine 20 mg b.d. after two months. He felt quite well when nifedipine was introduced. About two to three weeks after nifedipine was commenced, the symptoms of depression appeared. Other psychosocial stresses were the death of his mother three months earlier, and some genuine but not excessive economic losses.

Mr A.'s usual alcohol intake was 40–50 g per day and he was taking no other medication. Premorbidly, he was described by his wife as an active, energetic and interested man who had been successful in business, was sociable and well-liked.

On admission, the Hamilton Rating Scale Depression (HRSD) score (21 item) was 39. Physical examination was normal and his blood pressure was 140/80. The nifedipine was ceased and he was commenced on dothiepin (50 mg increasing gradually over five days to 150 mg nocte) and haloperidol (5 mg twice daily). Biochemistry screen, full blood count, thyroid function tests, B12 and folate levels were all normal. The dexamethasone suppression test showed non-suppression at 17 hours following dexamethasone (1 mg orally) (baseline cortisol 344 nmol/l, 9-hour level 74 nmol/l, 17-hour level 189 nmol/l. Dexamethasone levels at nine hours and 17 hours were 5.2 and 2.3 nmol/l respectively).

Mr A. showed significant improvement within 48 hours of ceasing nifedipine and at one week his HRSD score had fallen to 10. The haloperidol was rapidly reduced. At discharge after two weeks he was virtually asymptomatic, with only some preoccupation with financial matters persisting. At follow-up two weeks later he was completely recovered, with a HRSD score of zero.

The major features which implicate nifedipine in the aetiology of Mr A.'s depression are temporal. The symptoms began within one month of commencing nifedipine and improved dramatically within days of its cessation. The negative past history and family history also weigh against a non-organic aetiology. However, significant psychosocial stresses were present, i.e. a recent bereavement and concurrent financial difficulties, which are also of aetiological significance. In addition, an antidepressant and an antipsychotic were commenced on the same day as the nifedipine was ceased and even though it would be most unusual for such a severe depression with psychotic features to respond to a low dose of tricyclic within 48 hours, such a response cannot be discounted.

The most striking similarity between this case and those reported by Eccleston & Cole (1990), and Hullett *et al* (1988) is the rapidity with which recovery