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Seizures Terminable and Interminable with ECT

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The authors discuss the incidence, detection, management, and significance of prolonged seizures with ECT and present a case example. They suggest that the incidence of this phenomenon may be underreported and that its occurrence may be linked to hyperoxygenation, the use of multiple-monitored ECT, and preexisting states of cerebral hyperexcitability. Since prolonged seizures may result in adverse metabolic changes, prompt detection and intervention are essential.

ECT is acknowledged as an effective treatment in psychiatry, with its therapeutic potency derived from the induction of a series of generalized seizures in susceptible individuals. One of the advantages of electricity over pharmacologic agents as a mode of inducing seizures has been that the ictal duration with ECT is relatively brief, usually less than a minute. At the same time, however, the use of modern refinements in ECT technique such as muscular relaxation, oxygenation, and, most recently, multiple-monitored ECT (in which multiple seizures, monitored electroencephalographically, are produced during a single period of anesthesia) appears to have increased the incidence of prolonged seizures, although unilateral ECT, at least with sinewave stimuli, may act to decrease seizure duration. As will be discussed later, the actual incidence of prolonged seizures may be somewhat greater than would be expected, in part due to the relatively low use of EEG monitoring. Despite the fact that prolonged seizures represent a potential risk for the development of neurologic sequelae and are not associated with increased therapeutic benefits, this phenomenon has not been adequately dealt with in the psychiatric literature, and many practitioners are not aware of its significance, detection, and management.

Accordingly, using a recent clinical case example,

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we will attempt to outline some of the problems presented by a prolonged seizure with ECT. In particular, this case demonstrates that a seizure of only 6-10 minutes' duration can be associated with both metabolic insufficiency and delayed return to baseline neurologic function, even in the presence of apparently adequate oxygenation. We will also discuss the possible role of an underlying state of cerebral hyperexcitability.

CASE REPORT

The patient was a 26-year-old man with a diagnosis of schizoaffective schizophrenia, manifested by auditory hallucinations, delusions of thought control, depressed affect, and suicidal ideation. He had a history of polydrug abuse that included apparent daily intake of marijuana and intermittent use of alcoholic beverages, diazepam, oral amphetamines, cocaine, and hallucinogens. When he was admitted, his prescribed medication consisted of 1000 mg of chlorpromazine daily. There was no known personal or family history of epilepsy or other neurologic conditions and no family history of psychiatric illness.

After 15 days of imipramine, 200 mg, in addition to chlorpromazine, the level of the patient's suicidal ideation had markedly increased, and accordingly a laboratory evaluation for ECT was initiated, revealing only a borderline abnormal EEG, with brief bursts of bilateral sharply hypersynchronous 7-8 Hz theta rhythms, which were not considered significant.

As soon as the decision to use ECT had been made and informed consent for ECT obtained, imipramine and chlorpromazine therapy were discontinued. After 5 days the patient received his first electroconvulsive treatment, which was modified by atropine (.6 mg subcutaneously, 30 minutes before the treatment), methohexital (60 mg i.v.), and oxygen (100% by Ambubag, via oral airway, beginning at the onset of anesthesia and continuing until resumption of spontaneous respirations, except for a period of a few seconds at the time of stimulation). The electrical stimulation was provided by an 800-mA bipolar pulse device (MECTA Corp.) with the following settings: 60 pulse pairs per second, .75-second pulse-width, and .75-second stimulus duration. The stimulus electrodes were in the standard bifrontotemporal location. A single channel of bifrontal EEG was monitored.

The initial stimulus (intensity of 6 joules) did not produce a seizure either behaviorally or electroencephalographically and after approximately 15 seconds a second stimulus was applied, with the duration increased to 1.25 seconds. The stimulus (11 joules) produced a modified tonic-clonic convulsion, estimated by several observers to have lasted 10

than 60 seconds. Electroencephalographically, however, the seizure continued long past the termination of the clonic movements. After several minutes clonic movements involving the rostral half of the body appeared; they lasted approximately 45 seconds and were presumably concurrent with the metabolism of the succinylcholine, since no further relaxant or anesthetic was given. At 5½ minutes, the EEG seizure activity continued to be present and diazepam, 5 mg i.v., was given. Forty seconds later, with seizure activity still present in the EEG, the paper in the chart recorder ran out. Several minutes later, while the paper was being changed, movements of the patient's face and upper extremities again occurred, but on a more disorganized basis.

Because of concern that the motor activity might represent recurrent seizure activity, a second dose of diazepam, 5 mg i.v., was given. By this time, the chart recorder was again operating, but EEG activity was obscured by muscle and movement artifact for the next 2 minutes, following which a clear period of postictal suppression was present. Because the irregular movements persisted slightly into the period of postictal suppression, it is likely that they may have been part of a postictal delirium, rather than status epilepticus, and that the actual length of the seizure had been 6-10 minutes. The EEG was continuously monitored for the next 15 minutes and revealed an unusually prolonged period of postictal suppression, with activity consisting only of low amplitude delta rhythms.

During this postictal period, approximately 15-20 minutes after seizure onset, while the patient was still unconscious on 100% oxygen but breathing spontaneously (the respiratory rate was not recorded), arterial blood gases were measured; they revealed a mild acidosis (pH=7.26), with hypercapnia (pCO₂=51), but no anoxia (pO₂=85, O₂ saturation=95%). Measurement of arterial blood gases 30 minutes later showed a resolution of the above changes (pH=7.50, pCO₂=35). It should be noted that no cyanosis was present at any time. Values for serum electrolytes, calcium, magnesium, and glucose, measured during the acute postictal period, were all normal. During this time, examination revealed hypertonia and extensor plantar reflexes. When the patient regained consciousness 25-30 minutes after seizure onset, he was markedly confused.

Within a few hours, a computerized tomographic brain scan was obtained, both with and without radionuclide enhancement; it showed bilateral enlargement of the cerebral ventricles, without any evidence of cortical atrophy. Approximately 8 hours postictally, a lumbar puncture was carried out, with completely normal findings. An EEG done one day postictally showed an increase in the amplitude, duration, and incidence of the bilaterally synchronous paroxysmal bursts noted on the pre-ECT record; a second EEG 1 day later revealed a partial resolution of these electrographic changes. Neurological examination 1 day postictally continued to show evidence of symmetrical hypertonia and hyperreflexia with bilateral ankle clonus. The patient was also noted to be profoundly amnesic for events happening even hours after the ECT.

Behaviorally, the patient's affective disturbance began to clear somewhat over the next few days, and his psychotic agitation appeared to diminish in intensity. At the same time, however, he became uncooperative and refused further medical evaluation. He was discharged against medical advice 7 days after ECT.

DISCUSSION

A review of the literature has revealed only one previous report of a seizure lasting longer than 5 minutes when ECT was given at standard intervals of 3 times weekly (1). In that series the use of continued oxygenation before and throughout the seizure was felt to have significantly increased seizure duration. However, in the more recent technique of multiple-monitored ECT, in which two or more EEG-monitored seizures are evoked during a single period of anesthesia, prolonged seizures occur on a much more frequent basis (2-4), lasting as long as an hour (5). The only previous report of neurologic sequelae associated with prolonged seizure activity occurred with a multiple-monitored ECT session of four seizures, the last of which continued for 25 minutes and was followed by a 28-minute period of status epilepticus (6).

The fact that prolonged seizures have been reported only in the presence of EEG monitoring raises the question of whether this phenomenon actually occurs on a more frequent basis. As noted in the present case, seizure activity can continue long past the end of the convulsive motor activity. Without EEG monitoring, an undetected prolonged seizure might appear to be a prolonged postictal period of apnea or coma and would probably be ascribed to such factors as succinylcholine deficiency, idiosyncratic reactions to the anesthetic agent, or an unusually persistent and severe postictal state (7). At the same time, however, even with EEG monitoring, prolonged seizures appear to be uncommon except with multiple-monitored ECT. A recently studied series of 40 patients given ECT 3 times weekly had no seizures longer than 2 minutes, even with continuous oxygenation during the seizure (8).

Clinical and experimental data suggest that in the adequately oxygenated and relaxed patient, prolonged seizures can be tolerated without the development of ischemic changes (9). However, the case we have described indicates that ventilation with an oral airway may be insufficient to compensate for the increased metabolic demands of a prolonged seizure. This suggests that, under such circumstances, the patient be intubated and the seizure terminated with intravenous diazepam to minimize the risk of related morbidity. Bridenbaugh and associates (5) have suggested lowering ventilation rate and intensity in order to allow the seizure to be terminated by the resulting hypoxemia and hypercapnia; however, if ineffective and continued for a long time, such a procedure could actually add to the morbidity. Oxygenation only before and after the seizure or the use of room air represent potential means of minimizing the role of oxygen in prolonging the seizure, but they are not optimal from a metabolic standpoint (10).

The etiology of the prolonged seizure in the case we have described remains uncertain. The presence of preexisting paroxysmal EEG findings might suggest a

latent state of CNS hyperexcitability (11). In this regard, Bagchi and associates (12) have reported that nonepileptics with isolated paroxysmal EEG abnormalities occasionally showed increased paroxysmal activity after ECT. Furthermore, Hill and Parr (13) have reported seizures during insulin coma treatment or after catatonic episodes in nonepileptic schizophrenic patients possessing such paroxysmal abnormalities. The possibility that other exogenous factors, such as withdrawal of sedative hypnotic drugs, may have contributed to the prolongation of our patient's seizure is somewhat doubtful in view of the 20-day hospitalization preceding the ECT and the absence of clinical or EEG signs of withdrawal. However, the role of chlorpromazine and imipramine cannot be ruled out, even though they had been discontinued 5 days previously, since these agents, which increase cerebral excitability, have rather long half-lives.

The finding, in our patient, of cerebral ventricular dilation is consistent with reports (14) that some chronic schizophrenic patients may tend to have enlarged cerebral ventricles. Since the morphology and distribution of our patient's EEG abnormality could suggest a deep subcortical disturbance, a possible relationship between ventricular and EEG abnormalities may be worthy of future investigation. Studies have repeatedly shown a slightly higher incidence of various types of EEG abnormality in schizophrenic populations than in normal populations (15), although the consistency of specific findings has been poor. In most studies of abnormalities in ventricular size, correlation with EEG data has received little intensive investigation, although Haug (16) has reported that patients with definite ventricular dilation showed EEG abnormalities (which were not described) almost twice as frequently as did those without ventricular dilation.

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