ECT-Induced Postictal Delirium and Electrode Placement

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The authors report eight instances of ECT-induced postictal (emergence) delirium that occurred after bilateral ECT, right unilateral ECT, or left unilateral ECT. They conclude that postictal delirium is a random phenomenon unrelated to lateralized hemispheric mechanisms.

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ECT-induced postictal (emergence) delirium, an acute confusional state that occurs as the patient awakens from ECT, is characterized by agitation, restlessness, clouded sensorium, disorientation, and failure to respond to commands. When postictal delirium is severe or persistent, it requires termination with intravenous diazepam.

Sackeim et al. (1) reported two patients who manifested postictal delirium after bilateral ECT and right unilateral ECT but not after left unilateral ECT. These authors hypothesized that ECT-induced postictal delirium reflected disruption of right-sided cerebral systems consequent to heightened neurometabolic activity in the electrically stimulated hemisphere. Daniel (2) claimed nonspecificity for the syndrome when he reported the contradictory case of a patient who developed a postictal delirium after bilateral ECT but not after right unilateral ECT. Recently, we (3) reported the case of a fully dextral patient who developed delirium after left unilateral ECT; we concluded that it was premature to attribute postictal delirium solely to right hemisphere mechanisms. Since then, we have systematically monitored the postictal course of all patients participating in a continuing study of ECT, and we now report seven additional patients who exhibited postictal delirium.

METHOD

All patients were fully dextral men who received a research diagnosis of melancholia consistent with *DSM-III* criteria and were randomly assigned to receive either bilateral ECT, left unilateral ECT, or right unilateral ECT for the first six treatments. Informed consent was obtained from each patient. When clinically indicated, additional ECT treatments were given after the sixth, and electrode placements were changed for the seventh and eighth ECT treatments according to a standard protocol. Treatments after the seventh were administered with bilateral ECT. After patients were premedicated with glycopyrrolate, methohexital, and succinylcholine, seizures were induced with a Thymatron brief-pulse instrument set to deliver a fixed stimulus of 378 millicoulombs.

CASE REPORTS

In each case, the delirious state began with motor restlessness during the patient's emergence from anesthesia and was initially characterized by automatic behaviors (e.g., picking at the sheets) that inexorably progressed to frank agitation that was unresponsive to reassurance or commands and necessitated manual restraint. The sensorium was clouded, speech was characterized by perseveration and non sequiturs, and comprehension was markedly impaired. In each case, we attempted to treat the delirious state with intravenous diazepam; however, in three instances, the patients' agitation and aimless thrashing about dislodged the intravenous line and attempts to reestablish it were unsuccessful. These patients were manually restrained until their delirium subsided, usually over a period of 15–20 minutes.

Delirium After Bilateral ECT

Case 1. A 48-year-old man awakened from his first ECT treatment in a pleasant, jovial mood and was immediately able to converse without difficulty. Emerging from his second treatment, however, he became restless and uncooperative and pulled out his intravenous line. Two attempted intravenous injections of diazepam each infiltrated, and six staff members had to restrain him throughout a 30-minute postictal delirium. Intravenous diazepam was administered after each subsequent treatment, and no further delirium occurred.

Case 2. A 34-year-old man recovered uneventfully after his first ECT treatment but developed delirium after his second. Intravenous diazepam rapidly aborted the delirium and was

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used successfully for this purpose after each subsequent seizure.

Delirium After Right Unilateral ECT

Case 3. After his first ECT treatment, a 34-year-old man developed delirium, which responded promptly to diazepam; subsequent treatments were successfully managed with prophylactic postictal diazepam.

Case 4. A 56-year-old man exhibited the same pattern of response as the patient described in case 3.

Case 5. A 62-year-old man became restless after his first ECT treatment but responded to reassurance and minimal physical restraint. After his second treatment, however, he became increasingly agitated and unresponsive to verbal commands, thrashed about aimlessly, and dislodged his intravenous line. Thereafter, he was given intravenous diazepam after each treatment, and no further delirium occurred. For the seventh treatment, the unilateral electrode placement was switched to the left hemisphere. Diazepam was withheld on this occasion, and he again became delirious. He was totally unresponsive to commands and required physical restraint until diazepam could be given, which rapidly terminated the delirium.

Delirium After Left Unilateral ECT

Case 6. After his first ECT treatment, a 60-year-old man developed delirium, which responded to diazepam.

Case 7. A 30-year-old man initially received right unilateral ECT but was inadvertently switched to bilateral ECT on the third treatment; his recovery from each of the first three treatments was unremarkable. According to the protocol for patients whose electrode placements were switched, he next received left unilateral ECT; on emerging from anesthesia, he became delirious, pulled out his intravenous line, and had to be restrained. Before another line could be restarted, the delirium began to subside and diazepam was withheld. The rest of his recovery was unremarkable. After the fourth treatment, he refused further ECT.

DISCUSSION

The hypothesis that ECT-induced postictal delirium is due to disruption of mechanisms lateralized to the right hemisphere (1) is not supported by our data. Including the patient from our previous report (3), we have now observed nine instances of ECT-induced postictal delirium in eight patients, distributed among all three conditions of electrode placement: bilateral ECT (N=2), right unilateral ECT (N=3), and left unilateral ECT (N=4).

Once a patient develops postictal delirium, it is our usual procedure to administer diazepam after each subsequent treatment. Thus, in most cases, it could not be determined whether patients who became delirious with one particular electrode placement would also have developed delirium after receiving treatment with a different electrode placement.

In a recent study that used a low-dose titration procedure to determine the seizure threshold, Sackeim et al. (4) calculated the mean seizure threshold in a mixed sample of male and female patients to be 154.31 millicoulombs (range=36–459 millicoulombs). Therefore, the substantial, suprathreshold amount of charge used in our study (378 millicoulombs) might have obscured lateralized postictal effects that would have been observed at a lower dose. The experience of our patient 7 militates against this possibility, as he developed delirium after left unilateral ECT but not after right unilateral ECT or bilateral ECT, all given with the same 378-millicoulomb stimulus. Moreover, the second patient of Sackeim et al. (1) did not receive titrated low-dose stimulation but, nonetheless, exhibited a markedly different emotional response to left unilateral ECT than to right unilateral ECT.

We agree with Daniel (2) that the most parsimonious interpretation of the data thus far on postictal delirium is that it is a randomly occurring phenomenon unrelated to lateralized hemispheric mechanisms.

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