Nonconvulsive Status Epilepticus as a Cause for Delayed Emergence after Electroconvulsive Therapy

Bruce A. Crider, M.D., Sherri Hansen-Grant, M.D.

PSYCHOMOTOR and cognitive dysfunction are inherent, relatively short-lived, and immediate postanesthetic sequelae to any general anesthetic regardless of the anesthetic agents selected. Postanesthetic central nervous system dysfunction only becomes a concern when emergence becomes delayed or abnormal. We report the case of an elderly man who was suffering from a major depressive episode, recurrent and severe, who underwent electroconvulsive therapy and did not regain consciousness for 2–3 days after the procedure. This patient’s delay in awakening ultimately was found to be due to nonconvulsive status epilepticus.

Case Report

A 70-yr-old alert and oriented man was referred for electroconvulsive therapy after unsuccessful treatment of major depression despite several adequate trials of medication. This patient had a history of cardiomyopathy after myocardial infarction, congestive heart failure, and atrial fibrillation for which a pacemaker was placed 10 months before admission. His medical history also included mild hypertension and mild chronic obstructive pulmonary disease. The patient had no history of any neuropsychiatric illnesses. For these conditions, he was receiving Coumadin (prothrombin time 21 s) and theophylline (plasma concentration 12 μg/ml). The most recent pharmacologic regimen for the treatment of his depressive disorder included lithium carbonate, amoxapine thiothixene, and lorazepam. These medications were discontinued within the week before his scheduled electroconvulsive treatment. The patient was judged to be medically stable before his hospital admission.

Before his initial electroconvulsive treatment, serum electrolytes were within normal limits. General anesthesia was induced with 80 mg intravenous methohexital, and the trachea was intubated after receiving 100 mg succinylcholine intravenously. It was believed that the patient had inadequate muscle relaxation, and an additional 30 mg succinylcholine was administered. The patient received a right unilateral electroconvulsive therapy (ECT) at approximately 55 J. The motor seizure lasted 90 s and, by electroencephalograph, the seizure activity was interpreted to last 109 s. Given his history of hypertension, an esmolol infusion was started before treatment and continued throughout the treatment. It was noted during treatment that the patient had an incomplete neuromuscular block, and there was some generalized motor seizure activity noted in the uncuffed, nonisolated lower extremity. (Note: During electroconvulsive therapy, it is standard practice to isolate the circulation of a lower extremity from the general circulation by placing a tourniquet or blood pressure cuff around that extremity. This prevents the isolated extremity from the pharmacologic effects of drugs administered elsewhere in the general circulation.)

The patient did not recover spontaneously from ECT. Immediately after the stimulus, the patient was hemodynamically stable with a peak blood pressure of 177/121 mmHg with a pulse of 90 beats/min. He was noted to have Cheyne-Stokes respirations, and assisted ventilation was continued. Reintubation eventually was required for the purposes of continued mechanical ventilatory support. Although the electroencephalogram (EEG) indicated cessation of seizure activity at 109 s, continued seizure activity was suspected. The patient was not manifesting this with any noticeable gross motor movements. The pupils were reactive but asymmetrical. Lorazepam (2 mg) and phenytoin (1 mg) were administered. Twenty minutes later, the patient became hypotensive, and an infusion of dopamine was initiated. The neurology service was consulted, and it was initially suspected that the patient had suffered a pontine or a mid-brain hemorrhage. An emergency computed tomography scan of the head was obtained, which did not show any evidence of hemorrhage. The patient was transferred to the intensive care unit. In the intensive care unit, a follow-up EEG demonstrated nonconvulsive status epilepticus, and additional phenytoin was administered. An EEG done 24 h later showed a resolution of the ictal activity but documented periodic lateralizing epileptiform discharges on the right side. A follow-up computed tomography scan of the head 2 days after the ECT showed no evidence of a hemorrhage or new infarct, and an EEG at that time showed only generalized slowing with no focal ictal activity. The patient slowly began to awaken and become more alert over the next several days and eventually was discharged to home. At discharge, he had some mild confusion and generalized weakness but was deemed neurologically stable and not clinically depressed.
Discussion

Failure to regain consciousness after anesthesia can be the result of many different etiologies. Examples include the ongoing effects of anesthetic agents, residual muscle relaxant activity, and new neurologic impairment discovered postoperatively. Although this patient received lorazepam in preparation for long-term mechanical ventilation, and this, indeed, may have contributed to a prolonged somnolence after his ECT, it is the persistence of prolonged seizure activity in this patient after ECT that is most remarkable. This ongoing nonconvulsive seizure activity was thought to be the major contributor to this patient not awakening after treatment.

Seizures in the perioperative period have been linked to hypoxia, metabolic disorders, such as hypocalcemia or hypoglycemia, fever, or occult central nervous system disease. Both volatile and intravenous anesthetics have been associated with perioperative seizure activity. Examples of some of the medications include enflurane and ketamine. Some anesthetic medications, such as etomidate and methohexital, may produce seizure-like motor activity without being associated with clinical EEG evidence of seizure activity. Propofol, like ketamine, can increase seizure frequency in patients with a previous seizure disorder but does not produce EEG seizure activity in nonepileptic patients. It is unclear at this time whether the EEG activity produced by these various medications are of clinical importance and therefore necessitates therapy.

Prolonged seizures after ECT are rare but have been reported. Risk factors may be due to hyperoxegenation, lowered seizure threshold associated with medications, or preexisting epileptiform activity. Neuroleptic agents and most antidepressants also can decrease seizure threshold. Rapid withdrawal from benzodiazepines have the ability to decrease seizure threshold. Status epilepticus also have been more common with multiple monitored technique, in which several seizures are induced during one period of anesthesia. However, they have occurred with bilateral and unilateral single treatments. They also have been associated with concurrent lithium treatment. However, this patient's lithium was discontinued well in advance of treatment. Finally, this patient was receiving theophylline at the time of ECT, which can decrease seizure threshold, possibly causing prolonged seizures. Although one could speculate that this patient's treatment of depression may have benefited from the prolonged seizure activity, obviously, this is not recommended therapy. It should be noted that this patient has been subsequently readmitted for repeat ECT.

Appropriate preventative measures and quick treatment are essential to achieving a good outcome in such cases. These measures might consist of addressing potential risk factors and identifying high-risk individuals. Multichannel EEG recording also might be helpful in identifying subclinical status epilepticus. If a prolonged seizure occurs, benzodiazepines, such as intravenous diazepam or midazolam, might be used. Also, phenytoin and carbamazepine may be given for seizures that do not terminate with benzodiazepines or in high-risk individuals. Electroconvulsive therapy has been successfully repeated, as it was in this patient, without further complications in individuals with histories of prolonged seizures after ECT. Certainly, these patients should be approached with the recognition of their potential for prolonged seizure activity. Subclinical status epilepticus should be considered in patients who do not appear to regain consciousness quickly after ECT.

References

Large Doses of Topical Lidocaine during Microvascular Surgery Are Not Associated with Toxic Blood Concentrations

Robert E. Johnstone, M.D.,* Mark K. Wax, M.D.,† David J. Bishop, M.D.,‡ J. Brett Chafin, M.D.§

Surgeons performing microvascular anastomoses often apply lidocaine topically in doses exceeding published safe limits without apparent systemic toxicity. Standard pharmacology and anesthesia references list maximal doses of lidocaine as 750 mg or less.1-3 Reconstructive surgeons, however, may apply several thousand milligrams of lidocaine, at concentrations of 4-20%, directly on blood vessels being anastomosed, to prevent vasospasm during finger reattachment or tissue transfer surgery.4,5 To acquaint anesthesiologists with our resolution of this dose dilemma, we report four consecutive patients requiring free-flap reconstruction, during which topical lidocaine doses of 1,340–1,900 mg were used. After institutional review board approval and with the consent of each patient, we measured systemic blood concentrations of lidocaine in these patients before and every 15 min during and for 1 h after topical application.

Case Reports

Case 1

A 54-yr-old, 72-kg man with squamous cell carcinoma of the hypopharynx underwent total laryngopharyngectomy with bilateral modified radical neck dissections. He had a history of alcoholic cirrhosis, took amitriptyline, and was rated an ASA physical status 3. His tumor was treated preoperatively with radiation therapy. Anesthesia was induced intravenously with thiopental and 70 mg lidocaine and maintained with nitrous oxide, isoflurane, and fentanyl. The mucosal defect was reconstructed with a free fasciocutaneous radial forearm flap. Arterial inflow to the graft was from the facial artery; outflow was into the superior thyroid vein. Vascular anastomoses were secured with 9-0 nylon sutures. The surgeons applied 1,900 mg 4% lidocaine, using a 10-ml syringe with an 18-G intravenous catheter, onto the operative blood vessels during the 3 h required for anastomoses. Fifteen blood samples, collected from a radial artery catheter, throughout this period, all had less than 0.5 µg/ml lidocaine. Postoperatively the patient required surgical exploration for flap problems but was ultimately discharged in satisfactory condition. No evidence of systemic lidocaine toxicity was detected.

Anesthesiology, V 82, No 2, Feb 1995