Use of Transesophageal Atrial Pacing during Electroconvulsive Therapy

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ELECTROCONVULSIVE therapy (ECT) is associated with transient autonomic hyperactivity, which may be associated with cardiac dysrhythmias.1 Dysrhythmias are often benign and self-limiting but may be life-threatening or fatal. They most commonly are seen in patients with organic heart disease and those receiving digitalis, antidysrhythmic, or diuretic medication but can occur in the otherwise healthy individual. They are not limited to the first or second treatments but may occur throughout the course of therapy. Patients tend to have the same dysrhythmia after each treatment.1

Various pharmacologic methods have been advocated to prevent these dysrhythmias, but none is entirely successful, free from unwanted effects, or easy to titrate to the individual patient. This case report describes the use of a nonpharmacologic modality, transesophageal atrial pacing (TAP), in a patient who, despite the use of atropine or glycopyrrolate, suffered asystole on two occasions during a course of ECT.

Case Report

A 39-year-old, 80-kg man presented for a course of ECT after an acute exacerbation of schizophrenia disorder. Chronic essential hypertension was controlled with 10 mg oral nifedipine and 10 mg lisinopril. Medical history was negative for coronary artery disease, dysrhythmia, or syncope. He had not taken tricyclic antidepressants or monoamine oxidase inhibitors within the previous 3 months. Physical examination was unremarkable, and resting electrocardiogram (ECG) results, chest x-ray results, and electrolyte limits were normal; in particular, serum potassium was 4.1 mmol/L.

For all treatments, general anesthesia was induced with 1 mg/kg methohexital, 1 mg/kg succinylcholine, and 0.2 mg glycopyrrolate. Monitoring consisted of two ECGs (single channel built into the ECT machine and five-lead on the anesthesia machine), pulse oximetry, noninvasive blood pressure, and electroencephalogram (EEG). In addition, the patient’s left foot was isolated by tourniquet before administering the succinylcholine so that motor seizures could be observed. Ventilation with 100% O2 was assisted or controlled via a face mask during apnea, seizure, and the postictal period.

The first treatment was comprised of a current of 0.8 A at 90 Hz applied unilaterally for 2 s. This produced a convulsive seizure and a sinus tachycardia of 150 beats/min. At the end of ECT seizure activity, the heart rate began to slow and, despite 1.2 mg atropine, degenerated to 16 s of asystole (fig 1). This was confirmed by reference to another EEG lead and by the absence of a signal on pulse oximetry. Isolated sinus beats subsequently appeared, followed by a sinus tachycardia. Recovery from the anesthetic was thereafter uneventful.

The patient was evaluated by the cardiology department. Serial ECGs and rhythm strips showed sinus rhythm of 70–90 beats/min and no ST-segment changes. Cardiac enzyme limits were normal. He completed a Bruce protocol stress test to stage 5 with no symptoms or ECG evidence of ischemia, and there were no dysrhythmias during the recovery period. After psychiatric reevaluation and discussion with the patient and his family, it was decided to proceed with further ECT treatment, because other therapies had failed in the past. External pacing equipment was readily available.

For the second treatment, an additional 0.2 mg glycopyrrolate was given at induction, and the heart rate responded by increasing from 70 to 110 beats/min. The unipolar ECG stimulus of 0.8 A for 1 s generated a seizure and a cardiac response of an AV junctional tachycardia of 140 beats/min with a blood pressure of 150/100 mmHg. When EEG activity diminished, a sinus bradycardia occurred, culminating in 8 s of asystole; confirmed on another EEG lead and by loss of pulse pressure. Atropine (0.8 mg) was administered, but probably before this had acted, sinus complexes returned, and the heart rate progressively increased to 160 beats/min.

For the third treatment, it was decided to try TAP. After induction and muscle paralysis, an 18-G esophageal stethoscope incorporating pacing electrodes (TAPSCOPE, Arzco Medical Systems, Tampa, FL) was inserted into the esophagus to a depth of 35 cm from the infrahyoid oropharynx and connected to a pulse generator (model 2A, Arzco Medical Electronics). A grounding electrode was applied to the patient. Pacing was initiated using square wave pulses of 10 ms at a current of 20 mA and rate of 100 beats/min; 10 beats/min greater than the intrinsic rate. Confirmation of capture was made by reference to surface ECG leads II and V5 and to the pulse oximeter. We did
not attempt to determine the pacing threshold. No atropine or glycopyrrolate was given. Seizure was provoked with a 1 s shock of 0.8 A. A tachycardia of 150 beats/min ensued, comprising both paced and intrinsic complexes. The highest blood pressure, recorded 1 min after shock, was 150/100 mmHg. At the conclusion of seizure activity, the heart rate remained greater than 100 beats/min (fig. 1). Two minutes later, when the pacemaker was turned off, the patient's heart was in sinus rhythm.

For the fourth and all subsequent treatments to the completion of the course, the patient was pretreated with 0.2 mg glycopyrrolate. After induction, the TAP was inserted and capture confirmed by pacing at 10–20 beats/min greater than the intrinsic heart rate at a current of 20 mA. The pulse generator was turned off before the electric shock. All subsequent treatments were uneventful, except treatments 5 and 8, when 1–2 s of asystole immediately followed the shock, but the TAP was not activated because sinus rhythm promptly returned. Tachycardia of 120–140 beats/min occurred in all treatments and lasted for the duration of the seizure. The heart rate slowed at the end of the seizure but always remained greater than 100 beats/min. The TAP was not activated, and recovery was uneventful.

Discussion

The cardiac rhythm changes caused by ECT are thought to be due to autonomic stimulation and increase in the levels of circulating catecholamines. Initially, on application of the current, there is an immediate parasympathetic surge that may produce bradycardia or asystole. This post-stimulus pause is usually brief and is superseded in the clonic phase by tachycardia secondary to a sympathetic surge and release of catecholamines. Hypertension and dysrhythmias occurring in this period can be severe and potentially catastrophic in patients with ischemic heart disease. At the end of the clonic phase, a second sequence of parasympathetic and sympathetic activity occurs that is usually less rapid and less severe.

Atropine commonly is administered before ECT to protect against excess vagal stimulation of the heart. However, its routine use has been questioned with regard to efficacy and exacerbation of tachycardia. Glycopyrrolate has been recommended as an alternative for its milder chronotropic effect.

Prolonged asystole during ECT is uncommon. Our literature search has shown that all the documented cases of asystole were in the early parasympathetic
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phase after shock and lasted from 15 to 30 s. These patients generally were elderly with cardiovascular disease and were medicated with β-blockers and/or calcium channel antagonists. β-Blockers administered just before ECT to attenuate the sympathetic response were implicated in the mechanism of cardiac arrest when given in the absence of anticholinergic pretreatment.11

Our case is unique in that, on both occasions, the prolonged asystole did not immediately follow the shock; rather it occurred late, as an abrupt slowing of the tachycardia that coincided with termination of seizure activity on the EEG. The mechanism is unclear. We speculate that our patient suffered an unusually severe second parasymptathetic response in the recovery phase as described by Selvin 1 or may have had a hyperactive (cardioinhibitory) carotid sinus reflex. The effects (cardiac slowing, vasodilation) of the carotid sinus reflex have been shown in laboratory animals to begin at a pressure of about 60 mmHg and progress steadily as the pressure is raised to about 200 mmHg, when it is maximally active.12 In essential hypertension, carotid reflexes might be hyperactive.12 Our patient had well controlled chronic hypertension with no history of syncope; his peak blood pressure during treatment was 150 mmHg.

Because of the history of hypertension, efforts were made to exclude significant coronary artery disease. Nevertheless, without angiographic studies, the possibility of cardiac ischemia occurring during the tachycardia and producing conduction disturbances cannot be ruled out. However, we think this is unlikely because tachycardia and hypertension presented in all the treatments—albeit to a lesser degree when anticholinergics were omitted—yet asystole only occurred in the first two treatments.

Acute hyperkalemia and calcium channel blockers commonly are listed as causes of asystole. Increased serum potassium concentration within the first minute has been found to occur after methohexital-succinylcholine before ECT but not to dangerous levels.13 Our patient had normal electrolyte concentrations pre-treatment, and there were no ECG changes to suggest hyperkalemia. Serum potassium was not measured immediatley after asystole, because this possible cause was not entertained.

Succinylcholine has a vagotonic effect that has been proposed to enhance bradycardia during ECT. Although a possible contributing factor, we do not think that succinylcholine was solely responsible for the asystole in our patient because the dose of 1 mg/kg is not large, and the bradycardia occurred more than 1 min after fasciculation.

Electrode placement influences parasympathetic outflow.7 Given the same stimulation parameters, bilateral electrode placement is associated with greater current passage through the body than unilateral placement. Greater current was shown to increase cholinergically mediated asystole in dogs.7 Our patient, however, had unilateral ECT and moderate currents.

Asystole after ECT has been related to a failure to produce a convulsion: The vagal phase remains unopposed.19 In our case, there was EEG and clinical confirmation of seizure, and a definite sympathetic response was apparent.

The use of an external ventricular cardiac pacemaker for the treatment of asystole after ECT has been described.14 In commenting on a case report, Myles suggested the use of transesophageal pacing before ECT in a patient with bundle branch block and previous asystole, but we have no knowledge of this having been tried.

In our patient, TAP was chosen because of the history of prolonged asystole despite administration of vagolytic drugs. Another concern was to avoid the severe increase in the rate-pressure product that might occur in this hypertensive patient with larger doses of atropine.

TAP has been shown to be an effective treatment for drug-resistant bradycardia and bradycardia occurring intraoperatively.16 Compared to other temporary pacing modalities, TAP preserves atrial transport function and is relatively noninvasive.16 The commercially available model is incorporated into an esophageal stethoscope and is easily placed under general anesthesia. In the management of intraoperative bradycardia, Smith et al.17 found TAP to be more reliable than either atropine or glycopyrrolate, to have a faster onset of action, and to allow more precise control of the heart rate.

Although there have been no reports of esophageal damage, the manufacturers recommend grounding the patient if electrosurgery is used, and we took this precaution during ECT. The optimum site for pacing with
the TAPSCOPE probe in anesthetized patients is not precise and is around 30–35 cm from the teeth. We found that capture occurred at 20 mA, the first current tried. No attempt was made to determine the stimulation threshold because we anticipated a short procedure.

In summary, we report the use of TAP in a patient at risk of asystole during ECT. We propose a role for TAP in ECT for patients at increased risk of or having documented bradysrhythms and for those in whom atropine-enhanced increase of the rate pressure product is best avoided.

References


Arzco Medical Electronics: Personal communication. 1995.