

Propranolol prior to ECT Associated with Asystole

To the Editor:—A recent Clinical Report by Hood and Mecca¹ recommends the use of intravenous propranolol to attenuate the hypertensive response to ECT. We would like to report a case illustrating the occurrence of asystole associated with the use of iv propranolol in combination with ECT.

REPORT OF A CASE

A 68-year-old woman with her sixth episode of severe depression was scheduled for ECT. Electroconvulsive therapy was recommended because the current episode did not improve with an outpatient trial of pharmacotherapy. The patient's medical history included a myocardial infarction 5 yrs prior to admission and insulin-dependent diabetes mellitus. Admission laboratory tests were all within normal limits except for a fasting blood sugar of 337 mg/dl and the ECG, which showed an old anteroseptal myocardial infarction. Physical ex-

amination was unremarkable. The morning of the first scheduled treatment, the patient exhibited anxiety prior to treatment, with a pulse rate of 100/min and blood pressure of 180/120 mmHg. Before induction of anesthesia, propranolol, 1 mg iv, was administered, and the standard administration of atropine 0.4 mg iv was omitted. Thiopental 1.9 mg/kg, and succinylcholine, 0.5 mg/kg, were used to induce general anesthesia and muscle relaxation. Oxygen (100%) was administered via mask by positive pressure from the onset of induction. The treatment was monitored continuously by single-channel ECG and EEG. A bidirectional brief pulse electrical stimulus was delivered bilaterally without eliciting a seizure. The stimulation, however, was followed by progressive slowing of sinus rhythm for a period of 5 s, ultimately resulting in asystole. Cardiopulmonary resuscitation was instituted, following which regular sinus rhythm resumed after a total of 15 s of asystole. The patient recovered uneventfully from the anesthesia. Cardiologic evaluation at follow-up reported no sequelae from the event. ECT treatment was interrupted in favor of further pharmacotherapy and psychotherapy. After 4 months of treatment without

any symptomatic improvement, the patient was referred back to ECT. She was premedicated with atropine, 0.5 mg im and 0.4 mg iv, and then was given 0.9 mg/kg methohexital and 0.5 mg/kg succinylcholine. Electric stimulation of sufficient intensity to elicit a generalized seizure was used during the treatment. The patient tolerated a course of 13 treatments without cardiac complications and with good remission of symptoms.

Cardiac arrest is a well-documented, although rare, complication of ECT. In the present case, the tendency to bradycardia and asystole was probably heightened by the use of beta blockade. The subconvulsive electrical stimulation also could have contributed. Experimental data support the notion that an adrenergic mechanism is involved in the phenomenon of vagal escape² and that, in the presence of sympathetic blockers, a shock-induced activation of the autonomic nervous system can lead to a parasympathetic mediated cardiac arrest. This does not normally occur with ECT because the seizure elicits a marked peripheral sympathetic response that results in a rise in heart rate. With a subconvulsive shock, the central parasympathetic mechanism was unopposed and resulted in a slowing of the heart rate; a phenomenon exacerbated by beta blockade. It seems probable that propranolol was involved, at least in part, in the pathogenesis of the asys-

tole. Therefore, we feel that one should exercise caution when using the combination of propranolol and ECT.

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