

Correspondence

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Hazards of Edrophonium

To the Editor:—Youngberg¹ emphasized a serious and potentially fatal complication during the use of edrophonium chloride, 10 mg, for the conversion of paroxysmal atrial tachycardia. His caution concerning dosage was extended to the seemingly fit, elderly patient not taking any medication. We have recently had occasion to verify the concern expressed by Youngberg with an observation of an example of its use that caused a severe dysrhythmia.

A 62-year-old, previously healthy woman came to the Emergency Department complaining of pain in the right arm. In the past, she had had several episodes of short-lived palpitations, the nature of which had never been documented, the last episode being a year previously. She was not taking any medication. Physical examination was unremarkable, apart from a rapid heart rate and a blood pressure of 80/50 torr. A 12-lead electrocardiogram demonstrated supraventricular tachycardia at a rate of 170 beats/min, with no ischemic change. During monitoring of the electrocardiogram, edrophonium, 5 mg, was administered intravenously over 1 min. Within 2 min the supraventricular tachycardia was converted transiently to sinus rhythm at a rate of approximately 60 beats/min, followed by a 30-sec interval of atrial bradycardia at approximately 50 beats/min with no ventricular activity. Consciousness was lost within

15–20 sec. Sinus rhythm at a rate of approximately 60 beats/min was restored immediately by a precordial thump. The patient immediately regained consciousness, and full recovery occurred. We believe that when edrophonium is used to convert supraventricular tachycardias, the dosage for every patient should be 2 mg initially, as suggested by Youngberg. Furthermore, when ventricular asystole occurs as a result of the acetylcholine response, a precordial thump should be performed immediately.

S. LUDWIG, M.D.
Medical Resident
Health Sciences Centre
G. BRISTOW, M.D.
Associate Professor
Department of Anesthesia and
Director, Emergency Medical Service Unit
Health Sciences Centre
University of Manitoba
Winnipeg, Manitoba

REFERENCE

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Succinylcholine and Serum Calcium

To the Editor:—Bourke and Rosenberg¹ reported changes in plasma calcium during the administration of succinylcholine with or without pretreatment with a nonpolarizing muscle relaxant. Several months ago we undertook a similar study, which we have not submitted for publication. Our study differed from that of Bourke and Rosenberg in the following manner. We studied 18 patients. Arterial blood samples were drawn after premedication with atropine, 0.5 mg, intravenously and induction of anesthesia with thiopental (control); immediately after cessation of muscle fasciculations from succinylcholine 1 mg/kg

intravenously (sample 1); and one minute after cessation of fasciculations (sample 2). Ionized plasma Ca^{++} was measured from plasma separated anaerobically (Orion Biomedical® model SS 20). Simultaneous determinations of plasma pH (Radiometer® BMS 3 + PHM 73) were made.

When the slight respiratory acidosis (7.38) observed is corrected to pH 7.40 using Schaer and Bachmann's regression equation,² our results are: control, 1.96 ± 0.06 mEq/l; sample 1, 1.99 ± 0.06 mEq/l; sample 2, 1.99 ± 0.06 mEq/l. Statistical calculations using analysis of variance showed no significant dif-