Clinical Reports
BURNELL R. BROWN, JR., M.D., PH.D., Editor

Anesthesiology
49:208–210, 1978

Prolonged Asystole after Succinylcholine Administration

CHARLES H. MCLESKEY, LCDR MC USNR,* DONALD S. MCLEOD, M.D.,† TERRANCE L. HOUGH, LT MC USNR,‡ JAMES M. STALLWORTH, LCDR MC USNR* 

A single administration of succinylcholine to a child may result in profound bradycardia and junctional rhythm,1,2 while similar arrhythmias have also been seen in both children and adults following repeat doses of succinylcholine.3,4 After single administrations of succinylcholine to normal adult patients, bradycardia has occasionally been observed. We present the cases of two adult patients in whom prolonged cardiac asystole developed following single doses of succinylcholine.

REPORT OF TWO CASES

Patient 1. A 48-year-old white man who had a one-year history of bilateral leg claudication was admitted to the Naval Regional Medical Center, Oakland, for aorto-iliaic endarterectomy and lumbar sympathectomy. Systems review was non-contributory except for a ten-year history of mild hypertension treated with hydrochlorothiazide, 50 mg, b.i.d., and a 45-pack-year history of cigarette smoking. There was no history of cardiac arrhythmia or syncope. Five years prior to admission, the patient had received general anesthesia for spinal fusion without complication. On physical examination, the patient appeared healthy and was ambulatory. Weight was 75 kg, sitting blood pressure 140/80 torr, and pulse regular at 76/min. Cardiopulmonary systems were normal with the exception of decreased pedal pulses bilaterally. Hematocrit was 48 per cent, potassium 3.7 mEq/l. Chest x-ray and EKG were within normal limits.

Patient 2. A 50-year-old white woman who had end-stage renal disease was admitted to University of California at San Francisco, Moffitt Hospital, for parathyroidectomy. Past history included an uncomplicated homologous renal transplant with general anesthesia 18 months prior to admission. There was no history of cardiac arrhythmia or syncope. Medications included azathioprine, 100 mg, and prednisone, 15 mg, daily, and furosemide, 80 mg, q.d. On physical examination the patient appeared cushingoid, with weight 80 kg, supine blood pressure 110/80 torr, and a regular pulse at 80/min. Cardiopulmonary systems were normal. Hematocrit was 28 per cent; potassium 3.4 mEq/l.

An hour before operation, the patient received morphine sulfate, 15 mg, and scopolamine, 0.4 mg, im. On his arrival in the operating room, intravenous and arterial catheters were inserted. Other monitors included precordial stethoscope, blood pressure cuff, and EKG, which showed normal sinus rhythm at 80/min.

Induction of anesthesia was begun with morphine, 30 mg, iv, given in four divided doses over 10 min, with supplemental O2 by mask. After administration of d-tubocurarine, 3 mg, iv, and a 50-mg iv test dose of thiopental, blood pressure was stable at 150/80 torr, with a heart rate of 75/min. Induction of anesthesia was completed with thiopental, 200 mg, iv. Blood pressure and pulse remained unchanged after approximately a minute of hyperventilation with 100 per cent O2. Succinylcholine, 100 mg, iv, was administered to facilitate tracheal intubation. Approximately 45 sec later, prior to laryngoscopy, asystole suddenly occurred, with flat EKG and arterial pressure traces. Normal sinus rhythm at 80/min abruptly returned 20 seconds later without intervention. Subsequently, atropine, 0.4 mg, iv, increased the heart rate to 120/min. The trachea was intubated, and the operation was cancelled; the patient emerged from anesthesia and the trachea was extubated 10 minutes later. There were no sequelae. Arterial blood gases and electrolytes were normal in the recovery room.

Received from the Naval Regional Medical Center, Oakland, California, and Moffitt Hospital, University of California, San Francisco, California. Accepted for publication January 21, 1978.

Address reprint requests to Dr. Mcleskey at his new address: Assistant Professor, Department of Anesthesiology, Bowman Gray School of Medicine, Winston-Salem, North Carolina 27104.

The opinions expressed in this paper are those of the authors and should not be considered to represent those of the Department of the Navy or the Department of Defense.

208
mg, iv, cardiac rhythm rapidly reverted to normal sinus rhythm at 80/min. Laryngoscopy was difficult because of poor relaxation. Additional succinylcholine, 40 mg, iv, permitted easy laryngoscopy and tracheal intubation, with no further change in cardiac rate or rhythm. The subsequent intraoperative and postoperative courses were uneventful.

Discussion

A single dose of succinylcholine may produce bradycardia in 16–75 per cent of healthy children.2 Transient asystole is reported to have occurred in one child.2 When succinylcholine is administered in repeat doses, the incidence of bradycardia may be as great as 100 per cent.3 Two episodes of transient cardiac asystole under these conditions have been reported.3,5

In adults, the incidence of bradycardia following repeat doses of succinylcholine is reported to be as high as 95 per cent.4 However, following a single administration, tachycardia is usually observed, with bradycardia occurring only infrequently.6 Although 15 cases of asystole after repeat doses of succinylcholine have been reported,2,3,5,7–9 none has been reported after single administrations of succinylcholine to healthy adult patients. The two cases described in this article represent the first such report.

Schoenstatt and Whitker have suggested that bradycardia following repeat administrations of succinylcholine is the result of sensitization of the heart by a metabolite of the previously administered succinylcholine, i.e., choline.10 The minimum period between doses of succinylcholine thought to be necessary for bradycardia to occur on repeat administration (2–3 min)10,11 may be explained by the period required for hydrolysis of succinylcholine by plasma pseudocholinesterase to choline and succinylmonocholine.

Bradycardia or other arrhythmia following an initial dose of succinylcholine is more difficult to explain. Galindo11 and Goat12 and their co-workers believe that succinylcholine has a biphasic action similar to that of acetylcholine; accordingly, low doses directly stimulate cardiac cholinergic receptors, resulting in bradycardia, and higher doses stimulate cardiac sympathetic ganglia, indirectly resulting in tachycardia. Others propose that succinylcholine may directly stimulate the carotid baroreceptors, producing reflex bradycardia.13 Still others suggest that succinylcholine may compete with acetylcholine for available hydrolytic sites of true cholinesterase, so that acetylcholine may accumulate and result in bradycardia.14

Two procedures have been proposed to lessen the likelihood of bradycardia and asystole due to succinylcholine. First, a nondepolarizing muscle relaxant, when administered approximately 3 min prior to succinylcholine, will reduce the incidence of succinylcholine-induced bradycardia.6,13,15 Mathias and Evans-Prosser13 determined that d-tubocurarine or pancuronium, in a minimum dose of 70 μg/kg or 30 μg/kg, respectively, prevents the bradycardia associated with repeat succinylcholine administration, while similar protection has been found with the use of gallamine, 0.3 mg/kg.13 Second, administration of anticholinergics, either im with the premedication2,13 or iv, 5 minutes prior to succinylcholine,13,5 has been reported to reduce the incidence of succinylcholine-induced bradycardia. Recently, controlled studies have demonstrated that atropine, in dosages of 6–10 μg/kg, im, does not protect against the bradycardia resulting from initial or repeat doses of succinylcholine.10,16,17 Similarly, little protection was obtained with atropine administered iv 5 minutes prior to induction of anesthesia, until doses as large as 15 μg/kg were employed.17 However, these doses frequently resulted in unwanted tachycardia and supraventricular arrhythmias.

Thus, it appears that pretreatment with a nondepolarizing muscle relaxant prior to succinylcholine offers a greater likelihood for protection against bradycardia and less arrhythmogenicity than does pretreatment with an anticholinergic. It is somewhat discouraging that the two patients reported here had cardiac asystole despite the use of iv administration of d-tubocurarine (3 mg) prior to succinylcholine. However, in both cases the pretreatment dose of d-tubocurarine was less than that recommended by Mathias and Evans-Prosser15 (70 μg/kg), and in the second case, 8 minutes (rather than the suggested 3 minutes)8 followed before succinylcholine administration.

This paper reports cardiac asystole following single administrations of succinylcholine in two adult patients without predisposing disease. Mechanisms of the arrhythmia and methods of preventing its future occurrence are discussed.

References

A Complication of Percutaneous Cholangiography Resulting in Hypoxia and Death of an Anesthetized Patient

LYNDA S. KOEHLER, M.D.,* PEGGY S. CHRISTIDES, C.R.N.A.,† THOMAS A. ADAMEC, M.D.‡

It is common for a patient who has obstructive jaundice to receive a diagnostic percutaneous cholangiogram. This procedure may be performed preliminary to surgical intervention, or may lead to surgical correction of bile leakage. The following case history illustrates a complication of percutaneous cholangiography during general anesthesia that has not, to our knowledge, been reported before.

REPORT OF A CASE

A 33-year-old Caucasian woman had been in good health until three months prior to admission after failure to recover in 2½ months from what had originally been diagnosed as hepatitis. Radiographic and ultrasonic investigation showed dilatation of the biliary tree, common bile duct and gallbladder, and a mass lesion in the region of the head of the pancreas. During percutaneous cholangiography extravasation of contrast material was noticed and the procedure was abandoned.

Three hours later the patient arrived in the operating room for exploratory celiotomy. She was deeply jaundiced and in distress from abdominal pain. The blood pressure was stable at 120/80; heart rate 130-140 beats/min; hematocrit 41 per cent; serum electrolytes within normal limits; urinary output normal.

General anesthesia was planned. Anesthesia was induced slowly with a total of 200 mg thiopental iv, with only a slight decrease in blood pressure. Succinylcholine, 80 mg, was given iv and the trachea intubated with a cuffed orotracheal tube without difficulty. Anesthesia was maintained initially with 60 per cent nitrous oxide; pancuronium bromide, 5 mg, was injected iv when muscle activity returned. Initially, enflurane 0.5 per cent, was administered, but blood pressure decreased to 90 systolic, and enflurane was discontinued. Arterial blood-gas values in blood drawn shortly after induction of anesthesia (FiO2 = 0.4) were pH 7.35, PaO2 66 torr, and PaCO2 32 torr. After the blood-gas values were known, FiO2 was increased to 0.65, the position of the endotracheal tube was rechecked, and the presence of bilateral breath sounds reconfirmed. A second arterial blood sample showed pH 7.30, PaO2 85 torr, PaCO2 59 torr, and hematocrit 39 per cent. By this time the surgeons had opened the abdomen, biopsied a nodule found in the liver, and sent the nodule for frozen-section examination. Despite vigorous fluid administration the blood pressure continued to decrease to a systolic pressure of 80 torr. Nitrous oxide was discontinued and ventilation with 100 per cent oxygen began with a tidal volume of 600 ml at 12/min. A third arterial blood sample had a pH of 7.55, PaO2 67 torr, and PaCO2 33 torr. Approximately 5 min later blood pressure was unobtainable and cardiac arrest occurred. A vigorous resuscitation effort, including open-chest heart massage, was begun. The total elapsed time from induction was about 40 min. The patient could not be resuscitated. She was pronounced dead about 45 min after resuscitation efforts commenced. At this point, the report of the frozen section of tumor found in the liver was returned. It suggested carcinoid tumor. Despite the fact that the patient had no sign or symptom suggestive of "carcinoid syndrome," it was felt that cardiac arrest might have resulted from massive release of serotonin secondary to manipulation of an unsuspected carcinoid tumor. However, this did not account for the anecedent hypoxia, which certainly contributed to the failure to respond to resuscitative