either of which creates a condition similar to thoracic-outlet syndrome. The costoclavicular compression and partial obstruction of the flow in the subclavian artery cause damping of the distal pulse and pressure readings.[3-6]

In this case, during bypass, partial subclavian-artery compression similar to thoracic-outlet syndrome occurred when the sternal retractor was opened fully, and was unrecognized until the patient was taken off bypass. During the last 50 minutes of bypass, the patient received a total of 5 mg phenylephrine hydrochloride because of low mean arterial pressure.

In the presence of hypotension secondary to false arterial pressure readings, administration of vasoconstrictor substances could lead to fatal complications, such as cerebral hemorrhage or an increase in cardiac afterload that in turn will lead to increased O_2 consumption, myocardial ischemia, myocardial infarction, and cardiac decompensation.[7,8] In addition, severe vasoconstriction can lead to impaired tissue perfusion, cellular hypoxia, and acidosis, which might also adversely affect myocardial contractility.

We strongly suggest that during cardiopulmonary bypass, pressures observed through radial-artery cannulation be periodically checked against readings from a second site of measurement to ensure accuracy.

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Anesthetic Management of Hypokalemic Periodic Paralysis

Janet Naidl Siler, M.D., and William J. Discavage, C.R.N.A.

Familial periodic paralysis, associated with hyper-, hypo-, or normokalemia, is a rare disease of undetermined etiology characterized by intermittent attacks of skeletal-muscle weakness and flaccid paralysis, which usually spare the bulbar musculature. Attacks may be precipitated by high-carbohydrate meals, emotional excitement, severe muscular exertion, infectious disease, cold, menstruation, or accidental or surgical trauma. Cardiac arrhythmias may occur with the paralysis. Abnormalities of adrenal and thyroid function may be present. Children are susceptible to more severe and frequent attacks. The disease is observed predominantly in males; all races are affected; transmission is thought to be by an autosomal dominant trait, but sporadic cases have been reported.[1]

A patient with hypokalemic periodic paralysis underwent three surgical procedures with three different anesthetic techniques. Postoperatively, he experienced one episode of paralysis, for which intubation of the trachea and respiratory support were necessary.

Report of a Case

A 44-year-old man who had hypokalemic periodic paralysis controlled with acetazolamide and potassium chloride medications needed an emergency appendectomy. General anesthesia was induced with sodium thiamylal, 500 mg, and maintained with halothane, nitrous oxide, oxygen and a slow infusion of succinylcholine, 100 mg. The operation ended with the patient awake and breathing

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Address reprint requests to Dr. Siler.

spontaneously. Upon return to his room he complained of bilateral numbness and weakness of his toes, which progressed over 6–8 hours to complete muscle paralysis that necessitated intubation of the trachea. Serum potassium was 2.0 mEq/l. Potassium supplementation corrected this to 4.0 mEq/l, but mechanical ventilation was necessary for approximately 36 hours. Following extubation the postoperative course was uneventful.

Six months later the patient was hospitalized for repair of an incisional hernia. Anesthesia consisted of paravertebral blocks with 60 ml of 1 per cent lidocaine with epinephrine, 5 μg/ml. The patient's course was uncomplicated.

Two years later, the patient was admitted for a partial gastrectomy and vagotomy for duodenal ulcer. On admission, serum sodium, potassium, chloride, and bicarbonate were 145, 4.7, 104, and 25.5 mEq/l, respectively. Premedication was with pentobarbital, 100 mg, im, 2 hours prior to operation, and meperidine, 75 mg, and atropine, 0.4 mg, im, 1 hour before operation.

With the patient on his side, a spinal catheter was placed at the L3–4 interspace. When he was supine, tetracaine, 4 mg/ml, was injected through the catheter. T12 analgesic level was obtained. The tongue, pharynx, and trachea were topically anesthetized and the trachea intubated. Promptly, the patient clamped his teeth upon the endotracheal tube and thiopental, 250 mg, iv, was rapidly administered, followed by succinylcholine, 60 mg, iv. Generalized muscle fasciculations ensued, with spontaneous respirations returning within 5 minutes. General anesthesia was maintained with enflurane, nitrous oxide, and oxygen. Three more injections of tetracaine were given to maintain muscle relaxation during the 3½-hour operation. Intravenous fluids were 1,000 ml 5 per cent dextrose in water with 40 mEq KCl and 1,000 ml Ringer's lactate solution with 40 mEq KCl.

In the recovery room, serum sodium, potassium, chloride and bicarbonate were 139, 5.0, 111, and 17.7 mEq/l, respectively. Spinal anesthesia regressed and the patient was free from muscle weakness.

Eighteen hours after operation the patient experienced numbness and weakness in his feet and legs. Serum potassium was 2.6 mEq/l. Potassium chloride, 60 mEq, was added to his intravenous infusion, resulting in recovery of his muscle strength. The remaining postoperative course was uncomplicated.

Discussion

Anesthetic considerations for patients with familial periodic paralysis have been reviewed by Miller and Katz.[2] Paralysis has been reported to occur in such patients upon awakening from thiopental or general anesthesia.[1,3] To our knowledge, anesthesia of patients with normo- or hypokalemic periodic paralysis has not been reported.

In the case of our patient, who had hypokalemic periodic paralysis, anesthetic considerations for his third operation included adequate preoperative preparation, maintenance of normal serum potassium levels, choice of intravenous fluids, and selection of muscle relaxants.

Preoperatively the patient received balanced meals without an excess of carbohydrate. At the preoperative visit the patient voiced his anxieties. Appropriate reassurance was attempted and events anticipated for the following day were explained. Sedation was ordered to avoid mental stress. In retrospect, this preparation was inadequate for the awake intubation. Fortunately, the patient was amnesic for this event postoperatively.

Intraoperative intravenous fluids were chosen to avoid a large carbohydrate load, a large salt load, and hypokalemia. The EKG monitor was observed for changes in T-wave configuration. None occurred. Without knowledge of the action of muscle relaxants in this disease, the decision was made to avoid them. However, during the induction of general anesthesia succinylcholine was needed and was clinically well tolerated by the patient.

The postoperative paralysis that had occurred after the patient was first anesthetized appeared related to his low serum potassium level. Despite correction of this hypokalemia he had needed respiratory support for approximately 36 hours.

In conclusion, anesthesia for patients with hypokalemic familial periodic paralysis requires close attention to minimizing stress and anxiety, maintaining normal serum potassium levels, and avoiding a carbohydrate load. The use of muscle relaxants is preferably avoided; however, in this case the use of a short-acting depolarizing muscle relaxant appeared satisfactory.

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