Clinical Reports

Hypokalaemia and respiratory acidosis following partial obstruction of the airway

Peter B. Kane MD, Jorge Torretti MD

An 87-year-old female, with a history of hypertension controlled with hydrochlorothiazide, was scheduled for excision of a cystic mass of the left lobe of the thyroid. In the course of the anaesthetic, she developed partial airway obstruction that resulted in respiratory acidosis (PaCO_2 108 mmHg, pH 7.06), developed premature ventricular contractions and experienced a reduction in plasma potassium concentration from 3.9 to 2.9 mmole·L⁻¹. We interpret this hypokalaemia as a consequence of the epinephrine discharge due to hypercapnia. The case is reported to emphasize the importance of minimizing the sympathetic response to induction of anaesthesia, intubation and surgery in patients with marginal potassium stores.

Hypocarbica secondary to hyperventilation during anaesthesia has been reported to induce hypokalaemia.¹ We recently observed a decrease in serum potassium during the course of a general anaesthetic complicated by partial airway obstruction and respiratory acidosis. This observation, as well as other reports,^{2,3} serves as a reminder of the lability of serum potassium in situations where changes in hydrogen ion concentration and catecholamine output may occur concurrently.

Case report

An 87-year-old female was admitted for excision of a cystic mass of the left lobe of the thyroid. The patient gave

an indefinite history of a myocardial infarction six years previously, a cerebrovascular accident without residua and of hospitalization six weeks prior to this admission for congestive heart failure "thought to be secondary to hypertension." On physical examination, the patient was alert but somewhat confused; had a blood pressure of 150/80 mmHg, and a pulse rate of 72. She was edentulous, with a moderately enlarged thyroid gland. Medications included sublingual nitroglycerine prn, hydrochlorothiazide 50 mg po daily and supplemental potassium chloride. X-ray examination of the chest revealed "old calcified granulomas in the left lower lobe and scarring in the right upper lobe"; the ECG demonstrated first degree A-V block. Laboratory values were within normal limits including a serum potassium of 3.9 mmole·L⁻¹ the evening before surgery.

Upon arrival in the operating room, the patient's blood pressure was 160/100 mmHg, pulse 65. Anaesthesia was induced at 8:05 a.m. with 300 mg of thiopentone administered in incremental doses followed by N₂O:O₂ (50:50) and one to three per cent enflurane inspired. Lidocaine 100 mg was administered intravenously to blunt the pressor response to intubation. Following induction, the blood pressure fell to 120/80 mmHg. The rate of five per cent dextrose/lactated Ringer's solution (D5/LR) intravenous fluid infusion was increased and after ascertaining that it was possible to control the patient's ventilation, vecuronium 0.1 mg·kg⁻¹ was administered to facilitate tracheal intubation. During four unsuccessful attempts at intubation alternating with ventilation by mask, the patient's blood pressure rose to 190/100 mmHg. Considering the inability to intubate and the lability of the blood pressure, aspiration and needle biopsy of the thyroid were performed while anaesthesia was maintained with controlled ventilation by mask. At the end of the procedure, 40 minutes after induction of anaesthesia, the patient had received 1000 ml D5/LR, had a single twitch response to a train-of-four stimulus and remained apnoeic. To reverse neuromuscular blockade, neostigmine 3 mg and atropine 1.2 mg were given IV. The situation was

Key words

IONS: potassium, hypokalaemia; RESPIRATORY SYSTEM: hypercapnia, airway obstruction; SYMPATHETIC NERVOUS SYSTEM: epinephrine.

From the Departments of Anesthesiology and Pharmacology, University Hospital, S.U.N.Y. Health Science Center at Syracuse, Syracuse, New York.

Address correspondence to: Dr. Peter B. Kane, Department of Anesthesiology, S.U.N.Y. Health Science Center at Syracuse, 750 East Adams Street, Syracuse, New York 13210, U.S.A.

TABLE Serum electrolytes and arterial blood gases

Time	Na (mmole·L ⁻¹)	K (mmole·L ⁻¹)	PaO ₂ (mmHg)	PaCO ₂ (mmHg)	pH	BE
Preceding evening	144	3.9	—	—	—	—
9:20 a.m.	144	2.9	385 (FiO ₂ 1.0)	84	7.14	-3.0
10:15 a.m.	144	2.9	259 (FiO ₂ 1.0)	108	7.06	-3.0
11:15 a.m.	142	3.29	465 (FiO ₂ 1.0)	38	7.36	-3.8
4:10 p.m.	144	3.31	82 (FiO ₂ 0.4)	42	7.38	-0.2

now complicated because attempts to assist ventilation were opposed by splinting of the abdominal musculature. Over the next 30 minutes, blood pressure rose to 250/120 and premature ventricular contractions developed. A radial artery catheter was inserted and arterial blood gases and serum electrolytes measured (Table). Blood pressure was controlled with an infusion of sodium nitroprusside. Additional neostigmine and atropine was administered. A successful attempt at intubation was made, this time with a 7.5 mm ID anode tube. PaCO₂ was allowed to return to normal over the following hour (Table) during which lidocaine and potassium were given to treat persistent ventricular ectopy. Six hours after surgery, the patient had been extubated and was discharged from the recovery room. SNP and lidocaine infusions had been discontinued. The blood pressure was stable at 120/80 mmHg with a pulse rate of 60. Arterial blood gases immediately prior to discharge from the recovery room were acceptable, considering the patient's age (Table). Subsequent recovery was uneventful and the patient was discharged from the hospital the following day.

Discussion

The management of the airway of this patient encountered unanticipated difficulties that resulted in hypoventilation and respiratory acidosis, with a concomitant drop in serum potassium of 1 mmole·L⁻¹ below the value obtained the preceding evening. Low serum potassium is usually considered a factor increasing the risk of intra-operative dysrhythmias. When observed preoperatively, it is frequently a reason for delay of elective surgery. The purpose of this report is not to present the anaesthetic management of the patient, but rather to discuss the possible cause of the recorded potassium shift.

Airway obstruction with hypoventilation is frequently associated with cardiac dysrhythmias, which are commonly attributed to sympathetic discharge and increase in circulating catecholamines which accompany hypercarbia.⁴ In the present case, blood was drawn for blood gas

analysis and determination of serum sodium and potassium concentrations. Although we did not draw blood for catecholamine levels, we believe that a rising level of catecholamines stimulated an acute intracellular shift of potassium resulting in hypokalaemia despite a simultaneous decrease in serum pH, due to CO₂ retention.

Normally, extracellular fluid contains two per cent of total body potassium, although its actual level will fluctuate with potassium balance as well as shifts between intracellular and extracellular spaces. Potassium balance is determined by transfer across the gastrointestinal epithelium and the renal tubules. The former is normally affected by ingestion and by hormonal modification of potassium secretion by the colon. Gastrointestinal losses of potassium increase substantially with diarrhoea or the chronic abuse of laxatives. Urinary losses of potassium increase in patients taking thiazides or loop diuretics and in patients with hyperadrenalism. Potassium retention results from oligo-anuria and the use of potassium sparing diuretics. Finally, potassium may be removed from the body by haemo or peritoneal dialysis.

A shift of potassium to the extracellular space occurs during acidosis. Causes of acute shifts of potassium from the extracellular to the intracellular space are (a) glucose transfer across cell membranes, usually decreased during anaesthesia and surgery, (b) alkalosis, the opposite change occurred in this patient, (c) beta₂ receptor mediated catecholamine action. There is mounting evidence that beta₂ receptor mediated adrenergic agonists promote intracellular displacement of serum potassium.^{2,3} Anaesthetists have recognized hypokalaemia as a complication of terbutaline administration⁵ and more recently called attention to the dangers of potassium administration to patients treated with propranolol^{6,7} and treated hyperkalaemia with epinephrine infusion.⁸ Epinephrine infusion lowered serum potassium from 3.8 mmole·L⁻¹ to 3.1 mmole·L⁻¹ in normal volunteers. Serum potassium fell to even lower levels, from 3.4 mmole·L⁻¹ to 2.7 mmole·L⁻¹ in volunteers previously given diuretics.⁹ The plasma

levels of epinephrine measured during these experiments were similar to those reported during hypercarbia.⁴

Many anaesthetists consider normalcy of serum potassium as a prerequisite for anaesthesia for elective surgery. Recently, doubts shed on such dictum¹⁰ have met editorial resistance.¹¹ We wish to call attention to the events observed in this patient in as much as they illustrate the fact that in the planning of an anaesthetic for patients with marginally low potassium stores, proper steps to minimize the sympathetic response to induction, intubation and surgery may be far more important than the actual serum potassium level measured during preoperative laboratory testing.

References

- 1 Edwards R, Winnie AP, Ramamurthy S. Acute hypocapneic hypokalemia: an iatrogenic complication. *Anesth Analg* 1977; 56: 786-92.
- 2 Brown MJ, Brown DC, Murphy MB. Hypokalemia from beta₂-receptor stimulation by circulating epinephrine. *N Engl J Med* 1983; 309: 1414-9.
- 3 Williams ME, Gervino EV, Rosa RM et al. Catecholamine modulation of rapid potassium shifts during exercise. *N Engl J Med* 1985; 312: 823-7.
- 4 Price HL, Lurie AA, Jones RE, Price ML, Linde HW. Cyclopropane anesthesia. II. Epinephrine and norepinephrine in initiation of ventricular arrhythmias by carbon dioxide inhalation. *Anesthesiology* 1958; 19: 619-30.
- 5 Moravec MA, Hurlbert BJ. Hypokalemia associated with terbutaline administration in obstetrical patients. *Anesth Analg* 1980; 59: 917-20.
- 6 Hickey DR, Harrison L, Ramsey JG, Wynands JE. Hyperkalemic cardiac arrest following intravenous hydralazine and propranolol therapy post-embolectomy. *Can Anaesth Soc J* 1986; 33: 379-81.
- 7 Torretti J, Gerson JJ, Oates RP, Lange JS. B-adrenoceptor blockade and tolerance to potassium. *Anesthesiology* 1986; 64: 846-78.
- 8 Vitez TS. Treatment of hyperkalemia with epinephrine. *Anesthesiology* 1986; 65: 350-1.
- 9 Struthers AD, Whitesmith R, Reid JL. Prior thiazide diuretic treatment increases adrenaline-induced hypokalaemia. *Lancet* 1983; 1: 1358-61.
- 10 Vitez TS, Soper LE, Wong KC, Soper P. Chronic hypokalemia and intraoperative dysrhythmias. *Anesthesiology* 1985; 63: 130-3.
- 11 McGovern B. Hypokalemia and cardiac arrhythmias. *Anesthesiology* 1985; 63: 127-9.

Résumé

Une femme de 87 ans, avec une anamnèse d'hypertension contrôlée à l'hydrochlorothiazide, a été cédulée pour l'excision d'une masse kystique du lobe gauche de la thyroïde. Au cours de l'anesthésie, il s'est formé une obstruction partielle des voies respiratoires résultant en une acidose respiratoire (pH 7.06, PaCO₂ 108 mmHg), il est apparu des extrasystoles ventriculaires et la patiente a présenté une réduction de la kaliémie de 3.9 à 2.9 mmol·L⁻¹. Nous interprétons cette hypokaliémie comme étant une conséquence de la libération d'épinéphrine attribuable à l'hypercapnie. Nous présentons ce cas afin de souligner l'importance de minimiser la réponse sympathique à l'induction de l'anesthésie, à l'intubation, et à la chirurgie chez les patients ayant des réserves marginales de potassium.