In our hospital sterilized anesthetic masks come in disposable, clear plastic bags. If the bag is not removed prior to circuit assembly, attaching the curved mask/endotracheal tube connector to the mask through the plastic cuts the plastic bag off flush with the joint, creating a loose plastic disc within the circuit. Pressurizing the circuit during the preanesthetic check probably forced the plastic disc into the expiratory limb of the circuit. During induction the plastic disc may have migrated down the expiratory limb, producing obstruction only when it met the volume monitor sensor.

Preanesthetic circuit integrity is not assured unless both inspiratory and expiratory flow have been checked. As recommended by Grundy *et al.*[1] and Dorsch and Dorsch,[8] someone (presumably the anesthetist) should breathe through the system *via* the patient port. Application of negative pressure at the patient port will reveal obstruction in the inspiratory limb; application of positive pressure will reveal obstruction in the expiratory limb. In this case, such a maneuver might not have detected the foreign body, as it did not appear to obstruct the circuit until it reached the volume monitor sensor. Thus, any apparatus in the expiratory limb has the potential to create obstruction. Cases of expiratory obstruction and pneumothorax have been reported associated with bacterial filters[1,7] and foreign bodies[4] in the expiratory limb, respectively. The volume monitor sensor present on the Ohmeda Modulus® II anesthetic machine, although providing a valuable measure of ventilation, does pose a risk for expiratory obstruction if a foreign body should enter the circuit.

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Treatment of Hyperkalemia with Epinephrine

To the Editor:—Several authors have outlined how beta-2 adrenergic agonists lower serum potassium concentrations.[1–4,*] Epinephrine has been used to reduce serum potassium of hemodialysis patients.[5] I write to report my experience using epinephrine infusions to treat hyperkalemia.

My first experience involved a patient with 90% body burns. The patient had developed anuria, septic shock, respiratory failure, and hyperkalemia resistant to all therapy. The serum potassium concentration increased to 8.5 mEq/l, and cardiac arrest occurred. Calcium chloride reestablished a junctional tachycardia, but hyperkalemia persisted. Norepinephrine was discontinued, and an epinephrine infusion was begun (20 μg/min). Serum potassium was 8.3 mEq/l immediately prior to epinephrine. Ten minutes later, the serum potassium was 6.7 mEq/l, and a sinus rhythm appeared.

My second case involved an anuric, hemodialysis patient who underwent coronary artery bypass. At the end of cardiopulmonary bypass, the patient's serum potassium was 6.7 mEq/l, unchanged from 20 min prior. The electrocardiogram revealed prolonged P–R interval, biphasic QRS, and tall, peaked T waves. An infusion of epinephrine (2 μg/min) was begun. In 5 min, the serum potassium dropped to 4.9 mEq/l. The electrocardiogram was within normal limits, and bypass was discontinued without difficulty.

* Peters KR, Hurlbert BJ, Edelman JD: Potassium supplementation in beta2-agonist induced hypokalemia (abstract). ANESTHESIOLOGY 57:A388, 1982.

The last case involved a patient who had undergone aortic valve replacement. In the first postoperative hours, serum potassium gradually rose from 5.8 to 7.0 mEq/l. Norepinephrine was discontinued, and epinephrine was begun (13 μg/min). Thirty minutes later, a repeat potassium was 6.4 mEq/l. Other therapies (furosemide, bicarbonate) had little additional effect; potassium was 6.2 mEq/l 1 h later.

I believe these cases indicate that beta-2 adrenergic agonists may offer the most rapid treatment of hyperkalemia. In my cases, I chose epinephrine as the beta adrenergic agonist because of the hemodynamics of the situations. Although there is concern that epinephrine will exacerbate hyperkalemia, there was no indication that this occurred. The last case demonstrates that epinephrine therapy is not always very effective. Other beta-2 adrenergic agonists such as ritodrine may have equal or superior activity. Curiously, isoproterenol does not appear to affect serum potassium.[1] More clinical investigation is required to identify the best agents, optimal doses, and appropriate durations for beta-2 adrenergic therapy of hyperkalemia.

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