no time did diazepam produce statistically significant decreases in the ventilatory response to $CO_2$. As stated in "Materials and Methods," we used a form of multivariate analysis for repeated measures to show no change in the slope of $\dot{V}_E/PET_{CO_2}$ after diazepam. That test statistic was F 4, 20 = 1.6013, $P = 0.2129$. As the joint hypothesis was not rejected, Bonferroni adjusted paired $t$ tests were not used. The $P$ value 0.0508 represents a simple $t$ test without Bonferroni adjustment. Admittedly, this is not pointed out and has perhaps led Dr. Gross to misinterpret our findings. Therefore, contrary to what Dr. Gross suggests, the $P$ value for ventilatory depression at 5 min should, if anything, be larger, not smaller. Therefore, we believe our conclusions and title should remain the same to emphasize the significant variability of the respiratory response to diazepam.

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# Partial Expiratory Limb Obstruction by a Foreign Body Abutting upon an Ohio® 5400 Volume Monitor Sensor

*To the Editor:*—Obstruction of the expiratory limb of an anesthetic circuit is a rare but potentially lethal occurrence. Such obstructions are associated with incomplete pre-use circuit checking,[1] inappropriate circuit assembly,[1,2] the presence of a foreign body,[1,3–6] or anesthetic apparatus placed in the expiratory limb.[1,7] We wish to describe an episode of expiratory limb obstruction by an accidentally created foreign body abutting upon an Ohio® 5400 Volume Monitor Sensor.

An otherwise healthy patient was to undergo mediastinal node biopsy under general anesthesia. Prior to induction, an Ohmeda Modulus® II anesthetic circuit and ventilator, assembled by a previous operator, were tested for leaks and alarm function *via* thumb occlusion of the patient connector with the mask attached. Both inspiratory and expiratory valves had appropriate movement with positive pressure, and both nitrous oxide and oxygen flow meters demonstrated flow. After induction adequate positive pressure ventilation *via* mask was obtained with no inspiratory or expiratory obstruction noted. Following tracheal intubation, breath sounds were equal bilaterally with manual ventilation. Subsequently, the ventilator was engaged, whereupon 10–15 $cmH_2O$ of positive pressure was noted in the circuit during the expiratory phase. The expiratory limb was immediately disconnected from the Ohio® 5400 Volume Monitor Sensor. A disc of clear plastic was seen partially obstructing the expiratory limb, abutting upon the patient side of the volume monitor sensor (fig. 1). It was removed, and the circuit was reassembled, with no further expiratory obstruction being present.


FIG. 1. Plastic disc abutting upon volume monitor sensor in expiratory limb.

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.