

Anesthesia Case of the Month

History

An 8-year-old 22-kg (48.4-lb) spayed female Curly-Coated Retriever was examined because of severe obstipation. Medical treatment, consisting of dioctyl sodium sulfosuccinate enemas every 4 hours, oral administration of lactulose (5 mL) every 8 hours, and IV fluid therapy with lactated Ringer's solution (100 mL/h) containing additional potassium chloride (16 mEq/L), was ineffective in relieving the obstipation, and general anesthesia was necessary to manually extract the feces.

Prior to anesthesia, food, but not water, was withheld for 12 hours. Preoperative rectal temperature, pulse rate, and respiratory rate were 38.3°C (100.9°F), 120 beats/min, and 48 breaths/min, respectively. Heart and lung sounds were normal. Pulses were strong, capillary refill time was 1.5 seconds, and mucous membranes were pink. An indwelling IV catheter had been placed in the left cephalic vein the previous day for administration of lactated Ringer's solution.

The dog was premedicated with diazepam (0.2 mg/kg [0.09 mg/lb], IV) and butorphanol (0.2 mg/kg, IV), and anesthesia was induced with propofol (3.5 mg/kg [1.59 mg/lb], IV). The dog was orotracheally intubated without difficulty with a 10-mm silicone endotracheal tube. Anesthesia was maintained with isoflurane in oxygen delivered with a coaxial small animal breathing circuit.^a The vaporizer was initially set to deliver a 2% concentration of isoflurane; the initial oxygen flow rate was 1 L/min. The anesthetic machine had been pressure checked prior to use, and no leaks had been identified. However, several layers of elastic adhesive bandage tape^b were found to have previously been wrapped around the breathing hose just distal to the connection between the hose and the anesthetic machine.

A pulse oximeter^c was applied to the tongue to measure oxygen saturation (SpO₂). Respiratory rate was assessed visually, and heart rate was assessed by manual palpation of the lingual or dorsal pedal artery. The SpO₂ remained between 96% and 97%, with a spontaneous respiratory rate of 20 to 30 breaths/min. Respiratory effort initially appeared normal, and the rebreathing bag moved in synchrony with each breath. The heart rate was 123 to 130 beats/min, mucous membranes were pink, capillary refill time was 1 second, and pulse quality was strong.

Depth of anesthesia was consistently assessed as light given the presence of a brisk palpebral reflex, the high respiratory rate, and the appearance of leg movements during placement of an enema tube and fecal extraction. Although the vaporizer setting was increased to 2.5% and then to 3%, 3 additional doses of

propofol (20 to 30 mg each, IV) were required to maintain anesthesia.

Ten minutes after anesthetic induction, the dog appeared to breathe with an exaggerated inspiratory effort and substantial abdominal component, prompting an assessment of the patient and anesthetic circuit. When the dog was momentarily disconnected from the anesthetic circuit, it appeared to breathe normally, without any abdominal component during inspiration. However, when the dog was reconnected to the anesthetic circuit, the exaggerated inspiratory effort again became evident.

Question

What problem would cause exaggerated inspiratory effort during inhalant anesthesia in a dog?

Answer

The coaxial breathing circuit appeared straight, and there were no discernible kinks in the hose. The pop-off valve was open. The oxygen flow rate (1 L/min) was adequate to keep the 2-L rebreathing bag three-fourths full, allowing sufficient volume of gas for a full tidal vol-



Figure 1—Photograph of a small animal anesthetic machine that uses a coaxial breathing circuit; a single hose connects the breathing circuit to the anesthetic machine. The rebreathing bag, carbon dioxide absorber, and 1-way valves are similar to those used in anesthetic machines with standard circle breathing circuits.

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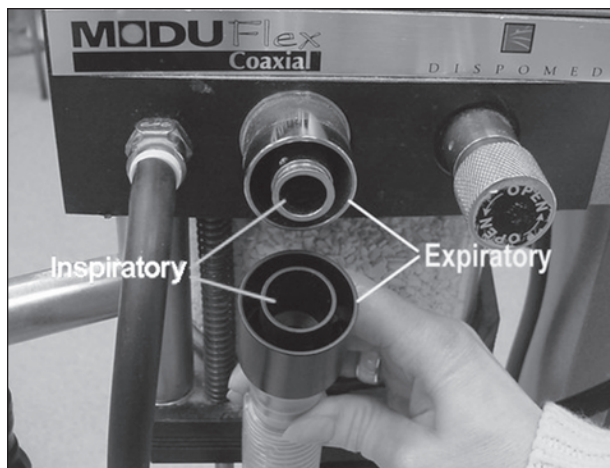


Figure 2—Photograph of a coaxial breathing circuit indicating the inspiratory and expiratory sites.

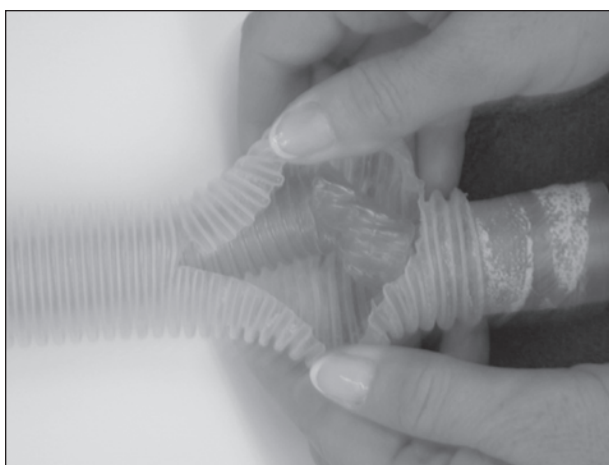


Figure 3—Photograph of a coaxial breathing circuit used in a dog that developed signs of inspiratory obstruction during general anesthesia. The outer tube of the coaxial breathing circuit has been cut to reveal a kink in the inner inspiratory tube.

ume breath in this dog. The attachment of the coaxial system to the endotracheal tube was secure. The endotracheal tube was of an appropriate length and not kinked. The dog's neck was in a level plane, without severe extension or flexion.

Closer inspection of the anesthetic circuit revealed a severe twist in the inner tube of the coaxial breathing circuit, resulting in obstruction of the inspiratory limb of the circuit. The twist was not readily visible during inspection of the anesthetic circuit because it was a coaxial system and because tape covered a portion of the circuit. After the twist was corrected, the procedure was completed with a total anesthesia time of 30 minutes. The dog recovered without complications. The SpO_2 was normal throughout the anesthetic period.

Discussion

Coaxial anesthetic breathing circuits use a tube-within-a-tube arrangement of the inspiratory and expiratory limbs of the circuit. Fresh gas flows into the circuit via the common gas outlet and arrives at the rebreathing bag, where it mixes with exhaled gases that have passed through the carbon dioxide absorbent.

During inhalation, gas flows through the small inner tube, opening the inspiratory valve and closing the expiratory valve. During exhalation, gas flows through the larger outer tube, closing the inspiratory valve and opening the expiratory valve. In this way, the warmer expired gases can partially preheat the cooler inhaled gases^d (Figures 1 and 2).

When the coaxial breathing circuit used to anesthetize the dog described in the present report was disconnected from the anesthetic machine and visually inspected, an obstruction consisting of a large kink involving the inner inspiratory tube of the circuit was visible (Figure 3). This obstruction had not been seen during the initial inspection of the anesthetic circuit because of tape covering the end of the tube. When the tape was removed, a small hole in the outer tube of the coaxial breathing circuit was identified, and the tape had apparently been applied in an attempt to seal this leak. The proximal end of the inner inspiratory tube was still appropriately attached to the end of the circuit. We speculate that when this breathing circuit had been used sometime prior to this anesthetic episode, a leak had been detected and someone thought that the leak was attributable to a loose connection between the breathing circuit and the anesthetic machine. Therefore, the outer expiratory tube was repeatedly twisted on the machine in an attempt to seal the leak, with the result that the inner inspiratory tube was repeatedly twisted, causing the kink. Apparently, when it was determined that the leak was from a hole in the outer expiratory tube, rather than a loose connection, tape was applied to cover the hole. The kink in the inner inspiratory tube could be removed by twisting the tube in the opposite direction; however, the inner tube was permanently deformed at the kink site and did not completely return to its normal diameter. This obstruction of the inner inspiratory tube decreased the rate at which gas moved through the inspiratory limb of the breathing circuit.

The case described in the present report illustrates the need to pay close attention to the physical condition of patients undergoing general anesthesia and to not rely solely on monitoring equipment. The dog's exaggerated inspiratory efforts prompted examination of the breathing circuit, leading to the identification of the problem. In contrast, SpO_2 values were normal, presumably because there was sufficient oxygen flow to keep the dog oxygenated. Pulse oximetry would not be expected to detect partial inspiratory obstruction in an animal receiving 100% oxygen because the oximeter only measures the percentage of hemoglobin saturated with oxygen and does not assess ventilation. Measurements of carbon dioxide partial pressure are necessary to determine whether ventilation is adequate.

It would have been interesting to measure end-tidal or blood carbon dioxide partial pressure to determine the adequacy of ventilation in the dog described in the present report. An end-tidal carbon dioxide monitor with a capnograph may have allowed for early detection of the partial obstruction via an abnormal waveform. Carbon dioxide partial pressure may have been increased because of inadequate ventilation, as has been found^{1,2} in human patients in whom kinks

have blocked the inner tube of a coaxial breathing circuit and obstructed inspiration.

Breathing against an obstructed airway can lead to rapid development of pulmonary edema because of the increased negative pressure required for inspiration.³ This has been reported³⁻⁶ in dogs, cats, and horses and in awake animals and animals recovering from general anesthesia. In these reports,^{3,6} the obstructions were a result of upper airway abnormalities, such as laryngeal paralysis, laryngeal masses, and acute upper airway obstruction. The pulmonary edema can be profuse and, if severe, can be hemorrhagic. Immediate treatment is required, consisting of relief of the obstruction, oxygen therapy, and IV administration of a loop diuretic such as furosemide. Consistent features of these cases are the exaggerated inspiratory effort and the lack of appropriate air movement. Mucous membrane cyanosis may be observed, and profuse, frothy, blood-tinged fluid may be seen in the airway or nasotracheal tube.³⁻⁶ The dog described in the present report did not develop signs of pulmonary edema, and abnormal fluid was not seen in the endotracheal tube at the time of extubation. In addition, the dog had normal respiratory activity when extubated. Results of thoracic auscultation were normal, and crackles or increased bronchovesicular sounds suggestive of pulmonary edema were not detected. The dog recovered from anesthesia without complications and was discharged from the hospital the following day. It is likely that this dog did not develop pulmonary edema because the inspiratory effort, although increased, was not sufficient to generate the substantial negative pressure required for formation of pulmonary edema.

Other important factors in the development of pulmonary edema associated with high negative pressures during inspiration include desaturation and subsequent hypoxemia.³⁻⁶ This dog experienced an obstruction while

being supplemented with oxygen, and desaturation did not occur, as indicated by the SpO₂ values.

The dog described in the present report experienced acute inspiratory obstruction, a potentially life-threatening complication. Fortunately, the obstruction was only partial and was rapidly detected because of physical monitoring of the patient's ventilatory effort. The equipment malfunction causing the obstruction was compounded by inappropriate use of tape to repair a leak in the system. Use of tape, particularly porous tape, not only is rarely effective in repairing leaks but, as in this case, may obstruct the view of underlying problems. When a leak in a breathing circuit occurs, the circuit should be replaced promptly.

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- a. Dispomed, Joliette, QC, Canada.
 - b. Elastikon, Johnson & Johnson Medical Inc, Arlington, Tex.
 - c. Nellcor, Puritan Bennett, Pleasanton, Calif.
 - d. Day TK. Effects of the Universal F breathing circuit on inspired gas and rectal temperatures compared to the standard circle in dogs (abstr), in *Proceedings. Annu Meet Am Coll Vet Anesth* 1995;16.

References

1. Marshall FPF. Kinked inner tube of coaxial breathing system. *Br J Anaesth* 1993;71:171.
2. Dorsch JA, Dorsch SE. Hazards of the anesthesia machines and breathing systems. In: Dorsch JA, Dorsch SE, eds. *Understanding anesthesia equipment*. 3rd ed. Baltimore: The Williams & Wilkins Co, 1994;325-361.
3. Kerr LY. Pulmonary edema secondary to upper airway obstruction in the dog: a review of nine cases. *J Am Anim Hosp Assoc* 1989;24:207-212.
4. Drobatz KJ, Saunders HM, Pugh CR, et al. Noncardiogenic pulmonary edema in dogs and cats: 26 cases (1987-1993). *J Am Vet Med Assoc* 1995;206:1732-1736.
5. Tute AS, Wilkins PA, Gleed RD, et al. Negative pressure pulmonary edema as a post-anesthetic complication associated with upper airway obstruction in a horse. *Vet Surg* 1996;24:519-523.
6. Ball MA, Trim CM. Post anaesthetic pulmonary oedema in two horses. *Equine Vet Educ* 1996;8:13-16.