Anesthesia Case of the Month

History

A 3.5-year-old 4.6-kg (10.1-lb) castrated male Scottish Fold cat was examined by the Small Animal Emergency Service at the University of Florida Veterinary Medical Center because of dyspnea. Thoracic radiography and thoracocentesis had been performed by the referring veterinarian, and a tentative diagnosis of pyothorax had been made.

On initial examination at the Veterinary Medical Center, the cat was tachypneic, with a respiratory rate of 120 breaths/min and open-mouth breathing. The cat was anesthetized with isoflurane, and a thoracotomy tube was inserted in the left side of the thorax. Radiography was used to confirm correct placement of the thoracotomy tube.

Hematologic and serum biochemical testing was performed; abnormalities included neutrophilia (14,900 neutrophils/µL; reference range, 2,500 to 12,500 neutrophils/µL) with a left shift (1,456 band neutrophils/µL) and 2+ toxic changes; anemia (Hct, 25%; reference range, 30% to 47%); high fibrinogen (600 mg/dL; reference range, 250 to 300 mg/dL) and carbon dioxide (28 mEq/L; reference range, 13 to 21 mEq/L) concentrations; high aspartate aminotransferase activity (84 U/L; reference range, 2.0 to 36 U/L); low hemoglobin (8.55 g/dL; reference range, 9.0 to 15 g/dL); albumin (1.6 g/dL; reference range, 2.0 to 3.0 g/dL); calcium (7.0 mg/dL; reference range, 8.6 to 11.1 mg/dL); creatinine (0.7 mg/dL; reference range, 1.0 to 2.4 mg/dL), and sodium (143 mEq/L; reference range, 135 to 162 mEq/L) concentrations; and low anion gap (2 mEq/L; reference range, 13 to 35 mEq/L).

The following morning, the cat was eating and grooming itself while in the oxygen cage. The respiratory rate had decreased to 54 breaths/min, but the cat remained dyspneic, and SpO2 was 88%. A total of 45 mL of brownish, viscous, opaque, malodorous fluid was obtained from the thoracotomy tube overnight. Cytologic examination of this fluid revealed a mixed population of bacteria, including extracellular organisms, and bacterial spores. Medical management of the cat during the subsequent 4 days included IV administration of crystalloid fluids (2.3 to 3 mL/kg/h [1.1 to 2.3 mL/lb/h]), ampicillin (22 mg/kg, IV, q 6 h), and trimethoprim-sulfamethoxazole (15 mg/kg, IV, q 12 h); administration of metronidazole (10 mg/kg [4.5 mg/lb], PO, q 8 h); administration of buprenorphine (0.02 mg/kg, SC, q 8 h); continuous pleural drainage; and monitoring of rectal temperature, heart rate, respiratory rate and effort, SpO2, Hct, and serum total protein concentration.

On thoracic radiographs obtained on the third day of hospitalization, a focal, severe, unstructured, interstitial to alveolar pattern involving the right cranial and middle lung lobes and a moderate, diffuse, unstructured, interstitial pattern involving the entire left lung field could be seen. In addition, there was a 3-cm-diameter, circular, soft tissue opacity superimposed on the left caudal lung lobe. Cytologic examination of an ultrasound-guided biopsy specimen of the mass in the left caudal lung lobe revealed a large number of highly degenerate neutrophils and a mixed population of bacteria, including cocci, rods, filamentous bacteria, and, possibly, bacterial spores. Bacterial culture of previously collected thoracic fluid yielded growth of Porphyromonas gingivalis, Bacteroides capillosus, and Clostridium difficile. A diagnosis of pulmonary abscess formation was made on the basis of the radiographic and cytologic findings. Although medical treatment had stabilized the cat's condition, surgical removal of the abscess and affected lung lobes was considered the best option for full recovery.

The cat was assigned an American Society of Anesthesiologists status of IV on the basis of disease severity and concurrent anemia. The cat was premedicated with fentanyl (5 µg/kg [2.3 µg/lb], IV), and 100% oxygen was provided via a face mask. Ten minutes after the fentanyl was given, anesthesia was induced with ketamine (0.25 mg/kg [0.1 mg/lb], IV), and diazepam (0.25 mg/kg [0.1 mg/lb], IV). A 4-mm, cuffed endotracheal tube with a Murphy eye was inserted in the trachea with the aid of direct laryngoscopy and the tip advanced to the level of the thoracic inlet. Isoflurane (initial vaporizer setting, 1.5%) in oxygen (initial flow...
rate, 1.5 L/min) delivered via a nonrebreathing anesthetic circuit was used to maintain anesthesia during the surgical preparation and instrumentation period. The cat was mechanically ventilated with a volume- and time-limited ventilator to maintain PetCO2, measured by means of mainstream capnography, between 30 and 45 mm Hg at a respiratory rate of 12 breaths/min and peak inspiratory pressure < 10 mm Hg. The capnography wave pattern appeared normal during this period, and SpO2 was > 95%. Heart rate, arterial blood pressure (measured indirectly), rectal temperature, respiratory rate, PetCO2, and SpO2 were monitored continuously and recorded every 5 minutes.

The cat was placed in right lateral recumbency with a rolled towel placed under the middle portion of the thorax to facilitate left lateral thoracotomy. A CRI of fentanyl was initiated at a rate of 1 µg/kg/min (0.45 µg/lb/min), IV, and the isoflurane vaporizer setting was reduced to 1%. Hetastarch was administered IV at a rate of 5 mL/kg/h throughout the procedure.

Fifteen minutes after the start of surgery (10 minutes after the thorax was opened surgically and 45 minutes after the start of anesthesia), the mechanical ventilator alarm sounded, indicating a pressure abnormality. The capnography wave pattern was not reflecting every ventilator-delivered breath, and the SpO2 had decreased to 82%. Mechanical ventilation was discontinued, and manual controlled ventilation was initiated. No obvious reasons for the problems with mechanical ventilation and capnography were found. However, it was noticed during manual ventilation that a peak inspiratory pressure of 12 to 15 mm Hg was required for left lung lobe inflation. The SpO2 increased to 93% with manual ventilation, but within 5 minutes after manual ventilation was initiated, it was again noticed that the capnograph did not display a waveform for every breath delivered. To obtain a waveform on the capnograph, a peak inspiratory pressure of 20 to 30 mm Hg was required, and the expiratory upstroke of the capnograph waveform had a distinct slope. The surgeons reported that even during these high-pressure breaths, left lung lobe inflation was minimal.

During the subsequent 5 minutes, manual ventilation of the lungs became impossible, although ECG and Doppler ultrasonic flow detection monitoring suggested that the cardiac rhythm was normal. Despite the use of peak inspiratory pressures as high as 35 mm Hg, the lungs could not be inflated, and no capnograph waveform was obtained. The SpO2 decreased to 77%, and the cat was visibly cyanotic.

Questions

Why was the capnograph not showing a waveform for every breath delivered? Why were such high peak inspiratory pressures required to obtain a capnograph waveform? What could be done to restore ventilatory function to this patient?

Answer

The endotracheal tube had become obstructed by the contents of the lung abscess. As the left caudal lung lobe was reflected cranially during surgical dissection, the contents of the abscess were forced into the trachea and endotracheal tube. Initially, the obstruction was neither complete nor continuous, which was reflected by the intermittent loss of the capnograph waveform. When no waveform was produced as breath delivery was attempted, the endotracheal tube was completely obstructed in a ball-valve fashion. On subsequent breaths, the obstruction may have moved out of the tube and into the trachea, which had a wider lumen, allowing a breath to be delivered to the lungs and the resulting exhaled CO2 to be detected by the capnograph. The partial obstruction of the endotracheal tube progressed to complete obstruction, preventing ventilation.

The endotracheal tube was replaced under direct laryngoscopy with a long (150-mm) 4-mm internal diameter, cuffed endotracheal tube with a Murphy eye that was advanced to approximately the level of the carina with the open bevel of the tube directed ventrally, toward the right lung. Manual, controlled ventilation was successfully restored, the surgeons noticed inflation of the cranial portions of the left lung during each breath, and the SpO2 increased to 96%. Following restoration of ventilation, the endotracheal tube was suctioned, and thick, brownish, mucoid material was aspirated. The surgeons aspirated similar material from the left mainstem bronchus prior to ligature during resection of the entire left lung. The lumen of the endotracheal tube that had been removed was completely obstructed with inspissated, brown, mucoid material.

The remainder of the surgery was uncomplicated. The endotracheal tube was suctioned again after transfer to the recovery area while the cat was in left lateral recumbency, but minimal mucoid secretions were obtained. Following return of spontaneous breathing, the cat was placed in an oxygen cage. The endotracheal tube was removed when swallowing was noted, and head movements were obvious and vigorous. A bandage placed to protect a thoracotomy tube hindered auscultation of lung sounds, but respiratory rate and effort were normal, and SpO2 was 96% 10 minutes after tracheal extubation.

Discussion

Rapid and accurate identification of the source of an anesthetic problem is essential to correction of the underlying problem and a positive outcome. In the case described in the present report, systematic efforts to rule out all possible causes of the capnograph abnormalities and high peak inspiratory pressures were required to identify the true causative factor. The first indicator that there was a problem with a component of the anesthetic circuit was the high-pressure alarm sounding on the mechanical ventilator during breath delivery. This alarm had been set to sound if airway pressure was > 20 mm Hg. The list of rule-outs for high peak inspiratory pressure includes problems with the breathing circuit or endotracheal tube and decreases in pulmonary compliance.1

Malfunctions of the breathing circuit, such as disconnections and obstructions, are most likely at the start of an anesthetic procedure or at times when the circuit is disrupted or altered as part of the anesthetic procedure. In this case, the first indicator of a ventila-
A cause-and-effect relationship between the left lung and the ventilation problem was identified 45 minutes after the start of anesthesia at a time when no alterations in the circuit had taken place. Malfunction of a 1-way valve, which can occur when rebreathing systems are used, was not possible because a nonrebreathing circuit was used. Obstruction of the endotracheal tube was considered soon after problems arose. However, no secretions were evident in the oral cavity at the time of the ventilation attempts. The tip of the endotracheal tube was inadequate as well, as evidenced by the decrease in SpO2. Atelectasis decreases lung compliance and increases the inspiratory pressure required to inflate collapsed alveoli. However, if the ventilation problem had been a result of lung atelectasis alone, the initial improvement associated with manual ventilation would have persisted as the atelectatic lung was re-expanded.

Possible causes for the high peak inspiratory pressure and the capnograph waveform abnormalities were reexamined as it became obvious that manual ventilation was failing to correct the problem. Causes of an intermittent capnograph waveform during ventilation include partial disconnection of the nonrebreathing circuit, pulmonary embolism, and partial obstruction of the endotracheal tube. The high peak inspiratory pressure evident during every ventilation attempt and careful checking of the ventilation circuit ruled out circuit disconnections. A pulmonary embolus would cause complete, or near complete, obstruction of pulmonary arterial blood flow, thus preventing alveolar perfusion and gas exchange. As PETCO2 subsequently decreased, the capnograph waveform would be expected to diminish, but this should have been reflected in every breath, not just some breaths. In addition, the cat’s cardiovascular status would have reflected global pulmonary arterial obstruction with profound changes in cardiac output and systemic perfusion, which were not observed. The possibility that the endotracheal tube was being intermittently obstructed by material not visible in the rostral end of the tube became the most likely cause at this time.

Along with the near-complete absence of capnograph waveforms, it was noticed that when a waveform did appear after a successful breath, this waveform was abnormally shaped. Changes in the slopes of the expiratory (phase II) and inspiratory (phase IV) portions of the waveform and rounding of the plateau (phase III) portion of the waveform became more pronounced as the ventilation problem worsened. These changes reflected bidirectional partial obstruction of airflow through the endotracheal tube. In addition, the PETCO2 was low (< 20 mm Hg), suggesting reduced alveolar ventilation. The accuracy of capnography readings during thoracotomy, particularly in smaller patients, has been questioned, and the use of capnometry without display of breath waveforms is of limited value. However, in a recent report, capnography was shown to be critical in the identification of an equipment malfunction during inhalant anesthesia in a cat. In the case described in the present report, ventilation was controlled to maintain PETCO2 within a certain range, but trends in PETCO2 and, more importantly, changes in waveform patterns were used to guide ventilation decisions, rather than exact values. The decrease in PETCO2 as ventilation failed was interpreted as reduced alveolar ventilation secondary to endotracheal tube obstruction, reflected by abnormal capnograph waveforms. Capnography may be the most accurate and immediate monitor for changes in airway patency in patients ventilated with 100% O2.

There are numerous reports of endotracheal obstruction in the human literature, including 1-way valve–type obstruction, obstruction by mucoid material, and delayed obstruction. In addition, airway obstruction has been used as an experimental noxious stimulus for determination of the minimum alveolar concentrations of halothane, isoflurane, and sevoflurane in cats, although the utility of these determinations for clinical applications is questionable. Endotracheal tube obstructions have been detected by the presence of high peak inspiratory pressures and abnormal capnograph waveforms. Most previous reports, however, describe obstructive complications occurring at the time of orotracheal or nasotracheal intubation. In these cases, the obstruction was often confirmed by means of fiberoptic evaluation of the endotracheal tube lumen. Correction of the obstruction, even in a pediatric case, involved suctioning of the endotracheal tube in situ, rather than removal of the endotracheal tube. In the case described in the present report, the obstruction had an insidious onset, and the diameter of the endotracheal tube (4.0-mm internal diameter) meant that endoscopic examination and in situ suctioning were nearly impossible. Replacement of the endotracheal tube provided an immediate solution to the obstruction as well as confirmation of the problem. At the time the endotracheal tube was replaced, the source of the material obstructing the original tube was identified as the contents of the left caudal lung lobe abscess, which had been ruptured during surgical manipulation. To avoid obstruction of the replacement tube, an attempt was made to direct the tip of the replacement tube ventrally into the right mainstem bronchus. Although location of the tube was not confirmed, no further obstruction occurred. Partial ventilation of the left lung after endotracheal tube replacement was noticed by the surgeons and may have occurred via the Murphy eye, which was directed toward the left lung. Use of endobronchial intubation in a cat to facilitate repair of a tracheal laceration secondary to prior endotracheal intubation has recently been described. The initially successful endobronchial intubation was achieved via endoscopy and...
direct examination, although the size of the endotracheal tube precluded proper replacement when the tube became displaced during patient transport.\textsuperscript{13}

A criticism of the anesthetic management of the case described in the present report may be that recognition of the endotracheal tube obstruction was delayed despite careful monitoring. However, a review\textsuperscript{15} of the human literature concerning adverse anesthetic incidents attributable to endotracheal tube problems suggests that obstructions are not uncommon and that capnography is the best method for detection of endotracheal tube obstruction. Attempts to improve detection of endotracheal tube obstruction by computer modeling or computer-assisted analysis of existing monitoring under research conditions have been reported\textsuperscript{15,16} suggesting detection and correction of problems are considered inadequate in human anesthetic practice. As capnographs become more readily available and affordable, they will hopefully be incorporated into routine anesthetic monitoring for all veterinary patients. However, a monitor cannot diagnose the cause and location of an anesthetic problem, as is apparent from the case described in the present report. A rapid, but systematic, approach to troubleshooting the problem must be used, paying careful attention to both the patient and the monitors, to achieve a successful outcome.

\textsuperscript{a} Hallowell veterinary anesthesia ventilator model 2000, Hallowell EMC, Pittsfield, Mass.
\textsuperscript{b} Datascope Passport, Datascope Corp, Parasmus, NJ.

References