In cooperation with

ACVAA
FOUNDED IN 1975

American College of Veterinary Anesthesia and Analgesia

Anesthesia Case of the Month

History

A 5-month-old 6-kg (13-lb) sexually intact male Chow Chow was anesthetized to undergo phacoemulsification for treatment of congenital cataracts. At the time of initial examination, the patient appeared healthy. The patient's medical record included an episode of pneumonia at 2 months of age that had apparently resolved with antimicrobial treatment.

Prior to sedation and general anesthesia, a complete physical examination was performed. The patient was tachypneic (40 breaths/min), although results of thoracic auscultation were unremarkable; a slightly high rectal temperature (38.8°C [101.8°F]) was attributed to the patient's young age and to the stress of being in the hospital. A CBC revealed high RBC count (8.75 X 10^6 cells/µL; reference range, 5.5 X 10^6 to 8.5 X 10^6 cells/µL), hemoglobin concentration (19.2 g/dL; reference range, 12 to 18 g/dL), Hct (58%; reference range, 37% to 55%), and mean corpuscular volume (79 fL; reference range, 60 to 77 fL). All other values were within reference limits. Serum biochemical analysis revealed a slightly high alkaline phosphatase activity (149 U/L; reference range,10.6 to 101 U/L), which was regarded as normal given the dog's age.

An IV catheter was placed in a cephalic vein, and the patient was premedicated with buprenorphine (0.02 mg/kg [0.009 mg/lb], IV). Once in the preoperative area, 100% oxygen was administered at a rate of 5 L/min via face mask for 5 minutes; propofol (2 mg/kg [0.9 mg/lb], IV) and diazepam (0.5 mg/kg [0.23 mg/lb], IV) were then administered to induce general anesthesia. The patient was orotracheally intubated with a reinforced cuffed tube (internal diameter, 6 mm) without a Murphy eye, and 2% isoflurane in oxygen was administered at a rate of 2 L/min via a nonrebreathing coaxial system. After the patient had been in the preoperative room for 30 minutes and had been clipped and prepared for surgery, it was moved to the operating room, connected to a modified Mapleson D breathing system (Bain system) with an oxygen flow rate of 2 L/min, and positioned in dorsal recumbency. The patient was attached to a multiparameter monitor,[a] and an ECG, oxygen saturation determined by means of pulse oximetry (Spo_2), end-tidal partial pressure of CO_2 ($Petco_2$), blood pressure (determined indirectly be means of oscillometry), and rectal temperature were monitored.

After surgical preparation was completed, the ophthalmologist positioned the patient with the head and neck flexed. Immediately after this, the capnogram ($Petco_2$ curve) disappeared from the monitor screen while the patient was breathing spontaneously. After it was ensured that the anesthetic breathing system was not disconnected, manual ventilation was started. However, the patient's thorax did not expand, and the capnogram did not reappear on the screen. The mouth and endotracheal tube were examined, and the tube was found to be kinked immediately rostral to the epiglottis, obstructing the tube. The patient was disconnected from the breathing system and repositioned in sternal recumbency. To resolve the obstruction and because of the possibility that the reinforced tube had been defective, the reinforced endotracheal tube was replaced with a nonreinforced cuffed tube (internal diameter, 6.5 mm) with a Murphy eye. The duration of obstruction of the endotracheal tube was estimated to be approximately 60 seconds. Several minutes later, the patient's breathing pattern had stabilized, and monitored parameters were within reference limits (heart rate, 120 beats/min; Spo_2, 98%; $Petco_2$, 43 mm Hg; and mean arterial pressure, 68 mm Hg). Therefore, despite this incident, the decision was made to proceed with surgery. The patient was again positioned in dorsal recumbency with the neck flexed, with no apparent complications.

A single dose of atracurium (0.1 mg/kg [0.045 mg/lb], IV) was administered, and mechanical ventilation[b] was initiated in pressure-controlled mode (peak inspiratory pressure, 11 cm H_2O; respiratory rate, 12 breaths/min; inspiratory-to-expiratory time ratio, 1:3). Anesthesia lasted for 180 minutes, with no additional complications. Two hours after anesthetic induction, the patient sustained a slight decrease in mean arterial blood pressure (55 mm Hg) for 10 minutes; this decrease was corrected with a bolus (10 mL/kg [4.5 mg/lb], IV over 20 minutes) of hydroxyethyl starch. All other parameters were within reference limits for the duration of general anesthesia.

At the completion of surgery, mechanical ventilation was discontinued. The patient immediately began breathing spontaneously, and the $Petco_2$ was within reference limits (38 mm Hg) despite the fact that the patient was tachypneic (68 breaths/min). Oxygen supplementation was continued via endotracheal tube while the patient recovered from anesthesia because of possible respiratory depression

This report was submitted by Adrià Aguilar, DVM; Xavier Moll, DVM, PhD; Félix García, DVM, PhD; Isabel Vigueras, DVM; and Anna Andaluz, DVM, PhD; from the Small Animal Medicine and Surgery Department, College of Veterinary Medicine, Autonomous University of Barcelona, Bellaterra 08193, Barcelona, Spain.

Address correspondence to Dr. Aguilar (adria.aguilar@uab.cat).

induced by the anesthetic drugs that had been administered. However, when the palpebral reflex returned, supplemental oxygen administration was discontinued. Pulse oximetry was continued to ensure that the Spo_2 was > 95%. Once the dog's swallowing reflex returned, 12 minutes after the end of anesthesia, the endotracheal tube was removed. The patient was then sedated with diazepam (0.5 mg/kg, IV) to avoid postanesthetic excitement. Five minutes later, the patient was moved to the intensive care unit for postoperative care. At that time, Spo_2 was > 95%,

Thirty minutes after being transferred to the intensive care unit, the patient became dyspneic, with marked signs of respiratory distress and slightly cyanotic mucous membranes. Pulse oximetry revealed an Spo_2 of 80%. Butorphanol (0.2 mg/kg [0.09 mg/lb], IV) was administered for sedation, and supplemental oxygen (5 L/min) was administered via a nasal cannula to produce an expected inspired oxygen concentration (Fio_2) of 30% to 40%. Thoracic auscultation revealed pulmonary crackles in all lung fields. Because the patient's oxygen saturation did not improve over the next few minutes, the patient was transported to the radiology suite; supplemental oxygen administration was maintained throughout this period. Two-view thoracic radiography revealed pulmonary edema. Furosemide (2 mg/kg, IV) was immediately administered. At this time, heart rate was 160 beats/min, mean arterial pressure (determined noninvasively by means of oscillometry) was 78 mm Hg, and Spo_2 was 82%.

Ten minutes after thoracic radiography was performed, respiratory arrest occurred. The patient was intubated, and manual ventilation was initiated. In about 3 minutes, spontaneous ventilation resumed. Five minutes later, the patient underwent cardiorespiratory arrest, and CPR was initiated. External chest compressions at a rate of 80 to 100 compressions/min were initiated with manual ventilation at a rate of 10 to 12 breaths/min. Atropine (0.02 mg/kg IV) and epinephrine (0.01 mg/kg [0.005 mg/lb], IV) were administered twice 2 minutes apart; however, the dog did not respond. Epinephrine (0.1 mg/kg, IV) was administered again, and constant rate infusions of dopamine (5 µg/kg/min [2.27 µg/lb/min], IV) and norepinephrine (0.25 µg/kg/min [0.11 µg/lb/min], IV) were initiated. After 20 minutes of CPR, ventilation (respiratory rate, 25 breaths/min; Spo_2, 75%) and heart beat (heart rate, 170 beats/min; mean arterial pressure, 55 mm Hg) were recovered; however, the patient did not regain consciousness. Ten minutes later, cardiorespiratory arrest reoccurred, and CPR was reinitiated. After 30 minutes of CPR, vital signs of the patient did not improve, and CPR was discontinued.

A necropsy was performed to determine the cause of death. On gross inspection, the lungs were firm and dark red. The trachea contained a small amount of pink froth. Histopathologic findings consisted of severe alveolar hemorrhage, hyaline membranes over the alveolar septum, and congestion of the perivascular space and pulmonary vessels. These lesions were indicative of severe pulmonary edema with lung injury, probably owing to high negative pressures related to an airway obstruction.

Question

What are the possible causes of endotracheal tube obstruction? How could this complication have been managed in this patient?

Answer

Endotracheal intubation is routinely used in veterinary anesthesia to maintain the patient's airway.[1,2] However, use of an endotracheal tube does not always ensure a patent airway, because the endotracheal tube can become obstructed. Acute endotracheal tube obstruction is a potentially life-threatening situation, and rapid recognition of this condition and appropriate intervention are essential.[3,4] Endotracheal tube obstructions can be divided into intraluminal and extraluminal causes. Intraluminal causes include obstruction of the endotracheal tube with mucus, blood, or gastric contents as a result of pneumonia, hemorrhage, or aspiration. Airway secretions can also obstruct the tube once positive pressure ventilation starts, with potentially life-threatening consequences.[5] Intraluminal obstruction of the endotracheal tube with foreign bodies such as teeth, dental implants, granulation tissue,[5] or even beans are rare events[6] encountered more frequently in human patients. Use of endotracheal tubes with small internal diameters may cause an increase in airway resistance, causing increased respiratory effort resembling that associated with partial airway obstruction.

A number of extraluminal causes can result in endotracheal tube obstruction. Kinking of the endotracheal tube is a relatively common cause of extraluminal obstruction of the endotracheal tube[2] and is usually related to patient positioning.[7] With many ophthalmic surgeries and other procedures involving cervical manipulation,[2,7] it is necessary to flex the patient's neck, which can cause the endotracheal tube to kink, occluding the lumen and preventing airflow. Compression of the tip of the endotracheal tube against the tracheal wall may also cause extraluminal obstruction, as described in 1 report.[3] Herniation of the cuff is a rare complication that should also be considered as a possible cause of extraluminal airway obstruction, and overinflation of the cuff can also obstruct the lumen of the endotracheal tube. In a previous report[4] describing patients who were intubated with a reinforced, resterilized endotracheal tube and received oxygen with or without nitrous oxide, bubbles appeared in the walls of the endotracheal tube and displaced the metal reinforcement, which occluded the lumen and obstructed the patients' airway. In our opinion, this problem was likely related to resterilization of the tubes rather than to the gases used. Finally, an endotracheal tube obstruction can

also result from defective manufacturing of the endotracheal tube.[8] These types of obstructions can be classified as intraluminal or extraluminal, depending on the manufacturing defect.

Appropriate and timely management of endotracheal tube obstruction is critical to avoid complications and prevent death. Clinical signs may be absent or nonspecific until substantial or complete obstruction of the lumen occurs.[5] Therefore, inspection of the endotracheal tube prior to anesthetic induction and patient intubation is critical.[8] Preemptive monitoring and periodic inspection of respiratory parameters such as ventilator pressure, capnography, and reservoir bag movement may assist clinicians in detecting obstructions early, allowing them to take prompt corrective actions.[5,8] Once an obstruction occurs, the patient's peak inspiratory pressure will increase,[3,4,6,8] and manual or mechanical ventilation may become difficult or impossible.[5] End-tidal partial pressures of CO_2 and Spo_2 may initially be unchanged if a partial obstruction occurs.[4]

In general, when there is suspicion of an endotracheal tube obstruction, management depends on the extent of the obstruction. When there is a partial obstruction, deflation of the endotracheal tube cuff and repositioning of the tube may be attempted. If available, suction of the tube lumen and fiber-optic examination of the tube might be helpful.[4,6] If the partial obstruction cannot be resolved or complete endotracheal tube obstruction is present, removal of the obstructed endotracheal tube and intubation with a new tube should be performed as rapidly as possible to reduce morbidity and mortality rates.[5]

Discussion

The patient described in the present report experienced a rare complication that can occur following any anesthetic procedure during which endotracheal tube obstruction occurs. The endotracheal tube in this dog became occluded during an ophthalmic surgery, resulting, we believe, in negative pressure pulmonary edema (NPPE) during the early postoperative period and a fatal outcome. To our knowledge, the earliest description of an upper airway obstruction leading to pulmonary edema in dogs breathing spontaneously against an inspiratory load was published in 1927.[9] In human patients, the first description of postobstructive pulmonary edema appeared in a 1973 case report[10] of a pediatric patient with shock.

Negative pressure pulmonary edema can be divided into 2 types on the basis of the timing of the obstruction that causes it. Type I NPPE appears after an acute obstruction of the airway (eg, angioedema, foreign body, endotracheal tube obstruction, or laryngospasm), resulting in a forceful inspiratory effort against a closed airway. Type II NPPE occurs following surgical resolution of a chronic airway obstruction such as a laryngeal mass.[10,11] In human patients, the incidence of NPPE has been reported to be between 0.05% and 0.1% for all anesthetic procedures.[11] According to 1 study, type I NPPE develops following 9.6% to 12% of all instances of acute upper airway obstructions; in contrast, the incidence of type II NPPE is reportedly 44%.[12]

Two different mechanisms may explain the pathogenesis of NPPE during upper airway obstruction. Young healthy subjects can generate high levels of negative inspiratory pressure (up to –140 cm H_2O).[10,13] This high negative pressure is transmitted to the intrapleural spaces, increasing venous return to the right side of the heart and pulmonary venous pressures. At the same time, perivascular interstitial hydrostatic pressure is decreased, favoring the movement of fluid from the pulmonary capillaries into the interstitium and alveolar spaces.[10,14,15] With the latter mechanism, the mechanical stress associated with respiration against an obstructed upper airway can rupture alveolar epithelial and pulmonary microvascular membranes, resulting in increased pulmonary capillary permeability and protein-rich pulmonary edema.[13] Sympathetic stimulation also creates a hyperadrenergic state that causes pulmonary hypertension and peripheral vasoconstriction. This condition and the concomitant increase in venous return may further exacerbate the pulmonary edema. Additionally, the resulting hypoxemia decreases myocardial contractility and increases pulmonary arterial resistance, thus increasing pulmonary capillary pressure and worsening the edema.[13,15]

Negative pressure pulmonary edema has been reported in dogs,[9,16,17] cats,[17] and horses.[15,18] Most cases of NPPE in human patients are uncomplicated and resolve within 24 to 72 hours with prompt treatment.[11,14] In the veterinary literature, there are only a few reports with variable outcomes. Five of 17 dogs and cats[16,17] and 3 of 5 horses[15,18,19] had a fatal outcome. However, recently, diagnosis and treatment of NPPE have improved, enhancing the likelihood of a successful patient outcome. Negative pressure pulmonary edema usually occurs rapidly following the removal of an obstruction. However, delayed onset has been reported.[11,20] Tachypnea, tachycardia, hypoxemia, hypercapnia, and the presence of frothy fluid in the airways or endotracheal tube are clinical signs of pulmonary edema and can be an indicator of NPPE[14]; thoracic radiography can be used to confirm the diagnosis. Radiographic changes include a diffuse interstitial or mixed interstitial and alveolar infiltrate.[15] Ultrasonography is also used in human patients for the diagnosis of NPPE, with a reported sensitivity of 94.6% and specificity of 96.1%.[9]

The treatment of NPPE generally consists of maintaining patency of the patient's airway and providing supplemental oxygen with positive end-expiratory pressure or noninvasive positive pressure ventilation while monitoring the patient's vital signs and arterial blood gases.[11,13,14,18] Approximately 50% of human patients reportedly require continuous positive airway pressure, and treatment of NPPE in humans is generally successful.[18] When oxygen supplementation is

required for a long period, clinicians should remember that exposure to 100% oxygen can have cytotoxic effects owing to the generation of free radicals; these effects can lead to pulmonary vasodilatation and atelectasis. In cases where supplemental oxygen is needed for more than 12 hours, the inspired oxygen fraction should be reduced to < 60% to avoid adverse effects.

In the dog of the present report, some improvements in the postoperative management may have prevented the fatal outcome. Monitoring (including pulse oximetry, blood pressure measurement, and ECG) immediately after cessation of general anesthesia and while in the intensive care unit as well as earlier thoracic radiography might have resulted in earlier recognition of the patient's worsening status and might have allowed earlier identification of the initial phase of pulmonary edema, thus allowing more rapid intervention. When a patient develops signs of pulmonary edema, oxygen supplementation nasally or via a face mask should be initiated immediately. Pulse oximetry is an important tool for noninvasive evaluation of ventilatory status. In patients that have received opioids, naloxone should be administered if Spo_2 is < 90% to reverse respiratory depression. Naloxone was not administered in our patient because we wanted to maintain sedation to avoid excitement and further distress. Nonetheless, some degree of respiratory depression may have been present, which might have been deleterious.

Arterial blood gas analysis should be performed to assess ventilatory function and tissue perfusion in any patient with signs of increased ventilatory effort. Evaluation of $Paco_2$ and Pao_2 as well as lactate concentration and base excess is helpful in directing treatment. If $Paco_2$ is high (> 50 mm Hg) and Pao_2 is low (< 60 mm Hg), indicating decreased tissue oxygenation, mechanical ventilation with positive end expiratory pressure will help to improve oxygenation. In the patient of this report, an Spo_2 of 80% was observed prior to butorphanol administration. Oxygen supplementation was initiated without an improvement in Spo_2. At that moment, we should have immediately intubated the patient and initiated mechanical ventilation before moving the patient to the radiology suite, rather than waiting until respiratory arrest occurred. After radiographs were obtained, we administered furosemide and maintained nasal oxygen supplementation. Nevertheless, the oxygen saturation did not improve. Intubation and mechanical ventilation with 100% oxygen in addition to furosemide administration might have improved the patient's Spo_2. In retrospect, arterial blood gas analysis and earlier initiation of manual or mechanical ventilation might have helped to improve the patient's oxygen saturation, avoiding the rapid worsening of ventilatory status.

Monitoring base excess and plasma lactate concentration is helpful in assessing tissue oxygenation. These measurements, in combination with arterial blood pressure and heart rate, can indicate the necessity for cardiovascular support. If the patient is hemodynamically stable, administration of diuretics in addition to mechanical ventilation and oxygen supplementation may be appropriate.[17] However, the use of diuretics remains controversial.[13,21,22] Koh et al[21] reported the treatment of NPPE in 5 human patients without use of diuretics, with great improvement reported for 3 of the 5 patients, 2 of whom were discharged from the hospital without symptoms in < 2 days and the other discharged without symptoms in 7 days. Unfortunately, one of the patients died because airway obstruction occurred at home, and although pulmonary edema resolved, the patient died 3 days after the incident. Additionally, Maxwell and Mihm[22] reported that diuresis might not be helpful in the case of a sudden shift of fluid into the pulmonary interstitium as in patients with NPPE. Administering diuretics in these cases can increase intravascular volume depletion, worsening hypotension and leading to life-threatening hypovolemic shock, especially in patients that cannot compensate for this fluid loss.[13,22] Despite this potential complication, diuretics are often administered to patients with NPPE in an effort to decrease intra-alveolar fluid and improve ventilatory status. We suggest that when the choice is made to administer a diuretic in patients with suspected or confirmed NPPE, hemodynamic status should be evaluated, including checking for signs of dehydration or hypovolemia prior to diuretic administration.[11] In hypovolemic and dehydrated patients, diuretics are contraindicated until intravascular volume is normalized. After administration of diuretics, patients should be monitored in case volume resuscitation is required. In the patient of the present report, we administered furosemide because no signs of dehydration or hypovolemia were noted. Nonetheless, we suggest that further investigation is warranted to clarify the role of diuretics in the treatment of patients with NPPE.

In the patient of the present report, endotracheal tube occlusion most likely caused the NPPE and death. Nonetheless, other contributing causes of pulmonary edema cannot be excluded. Postoperative pulmonary edema may be caused by an anaphylactic or anaphylactoid reaction to drugs administered during anesthesia, although other signs such as hypotension, bronchospasm, and hives would be expected.[14,23] Volume or pressure acute lung injury can cause or contribute to postoperative pulmonary edema in manually or mechanically ventilated patients. The patient of the present report was mechanically ventilated during the entire surgical procedure, and although airway pressure was monitored, this may have contributed to the development of postoperative NPPE. Fluid overload as a result of fluid maldistribution can also cause pulmonary edema in patients with hyponatremia or receiving hypotonic fluids[14] and in patients with cardiac disease. In our patient, fluid overload was not considered because the dog did not present any of these situations. Other causes of postoperative pulmonary edema include neurogenic and cardiogen-

ic causes[14]; these were not considered in our patient, because no history of neurologic or cardiac disease was evident.

In the patient described in the present report, several factors potentially contributing to the fatal outcome could be identified. The Chow Chow breed can be considered brachycephalic. During preoperative examination, no signs of brachycephalic syndrome were noted, and even during the postoperative period, when the dog was sedated, no signs of partial obstruction of the airway, as caused by stenotic nares or an elongated soft palate, were evident. Nevertheless, we cannot discount that brachycephaly may have exacerbated the patient's condition. In addition, neuromuscular blocking agents were administered to facilitate the surgery. Although a single dose of atracurium was administered and the surgical procedure was long (180 minutes), residual curarization might have contributed to postoperative hypoventilation and hypoxia, worsening the NPPE.

Clinical signs of pulmonary edema may not appear until several hours after the problem initially develops.[11,21] However, development of pulmonary edema immediately following extubation has been previously reported.[14] In our patient, both options should be considered. Pulmonary edema may have developed during the postoperative period in the intensive care unit, with a delayed onset. However, it should be considered that respiratory distress might have been overlooked during the anesthetic period because the patient was mechanically ventilated or because of sedation during the early postoperative period. Once the patient's sedation wore off, the respiratory distress was evident, and the patient's condition deteriorated. In most cases of NPPE involving human patients, recovery occurs within 12 to 72 hours with appropriate treatment.[11,18] The patient of the present report died approximately 5 hours after endotracheal tube occlusion occurred. At necropsy, there was only a small amount of pink froth in the trachea, in contrast with the findings of other studies.[16,18] However, there was congestion in the pulmonary lobes similar to that described by Tute et al.[18] Histopathologically, the pulmonary lesions documented in our patient resembled those reported by Tute et al[18] and included multifocal intra-alveolar hemorrhage and hyaline membrane formation in the alveoli.

Selection of an appropriate endotracheal tube for each patient and surgical procedure is critical. Endotracheal tubes can be manufactured from polyvinyl chloride, silicone, or red rubber.[1,2,24,25] Red rubber tubes are opaque and are more prone to kinking, but they are easy to insert and can be reused.[3,23,24] Polyvinyl chloride and silicone tubes are similar and are less prone to kinking because they mold to shape when warmed by the patient's body temperature.[25] The main problems with silicone tubes is that they are expensive and may require a stylet to insert.[24,25] Reinforced or armored tubes are the most rigid because they have an internal metallic spiral with a thicker wall. These tubes are not prone to kinking, and they are well suited to certain surgical procedures such as those during which the patient must be positioned with the head and neck flexed.[1] A potential disadvantage of these tubes is that if an animal bites the tube, it can be permanently obstructed.[24,25] Furthermore, these tubes are manufactured with a smaller internal diameter than standard tubes because of the thickness of the wall.[24] Endotracheal tubes may also have a sealing cuff that prevents aspiration of regurgitated or foreign material and avoids the dilution of inspired gases with room air.[24] High-volume, low-pressure cuffs require high volumes to be inflated, but the pressure is distributed across a large tracheal surface,[24,25] avoiding tracheal ischemia and necrosis. Low-volume, high-pressure endotracheal tube cuffs provide better sealing of the trachea but increase the risk of tracheal damage.[24,25] Additionally, endotracheal tubes may have a Murphy eye, which is an oval hole in the tip that allows gas flow even if the tip hole is occluded.[1,24,25]

For the patient of the present report, we had selected a reinforced tube to avoid tube kinking because the patient needed to be positioned with the neck in hyperflexion. Despite the use of this tube, signs of occlusion occurred, most likely because of kinking. The kinking may have been attributable to the dog's biting the tube or a manufacturing error, but no obvious defects were seen during inspection of the tube. Therefore, we cannot discount a mucus plug, cuff herniation, or compression of the tube tip against the tracheal wall as the cause of the obstruction. The endotracheal tube might have been moved down the trachea during flexion of the neck, as described by Quandt et al,[7] pushing the tube against the carina and obstructing the tip of the tube. Consequently, it is important to measure the length of the endotracheal tube to be used and to adjust it to the patient. Nonetheless, when dealing with reinforced tubes, this may become a problem. Reinforced tubes cannot be cut; thus, part of the tube can protrude outside the mouth. If the length is not measured before intubation, the tip of the tube can travel deeper into the carina.

Negative pressure pulmonary edema is a life-threatening complication that can occur after upper airway obstruction. Progress has been made in understanding the pathophysiology and management of this pathology. Rapid recognition of the problem is essential to achieve a favorable outcome. In addition, the status of the patient and the surgical procedure to be performed should be taken into account when an endotracheal tube is selected. In the authors' opinion, polyvinyl chloride or clear silicone tubes with a Murphy eye should be used if no contraindication exists, as they will mold to the patient's position and the 2 orifices will prevent obstruction of the tube. For procedures during which kinking of the tube can occur, reinforced tubes should be used to avoid tube occlusion. Finally, appropriate selection of the endotracheal tube

and inspection of the tube prior to anesthetic induction as well as careful positioning and monitoring of the patient are necessary for timely intervention and avoidance of possible anesthetic complications.

Acknowledgments

No funding was necessary for this study.

Footnotes

a. VetCare Multiparametric monitor, B. Braun, Barcelona, Spain.
b. MAV3, EKU Electronik GmbH, Leiningen, Germany.

References

1. Hartsfield SM. Airway management and ventilation. In: Tranquilli WJ, Thurmon JC, Grimm KA, eds. *Lumb & Jones' veterinary anesthesia and analgesia*. Ames, Iowa: Blackwell Publishing, 2007;495–532.
2. Campoy L, Hughes JML, McAllister H, et al. Kinking of endotracheal tubes during maximal flexion of the atlanto-occipital joint in dogs. *J Small Anim Pract* 2003;44:3–7.
3. Parray T, Martin T, Shah S, et al. Maneuvers to relieve the endotracheal tube obstruction caused by the bevel orifice abutting the trachea in the prone position. *WebMedCentral Anaesth* 2010;1:9.
4. Mercanoglu E, Topuz D, Kaya N. The dissection of reinforced endotracheal tube internal wall causing intraoperative airway obstruction under general anesthesia: case report. *Rev Bras Anestesiol* 2013;63:372–374.
5. Mort T. Acute airway obstruction following intubation: a clinical dilemma. Available at: www.anesthesiologynews.com/download/Obstruction_ANGAM12_WM.pdf. Accessed Dec 7, 2016.
6. Shahid M, Khan E. Difficult to ventilate. Unusual cause of endotracheal tube obstruction. *J Anesth Clin Res* 2011;2:131.
7. Quandt JE, Robinson EP, Walter PA, et al. Endotracheal tube displacement during cervical manipulation in the dog. *Vet Surg* 1993;22:235–239.
8. Hajimohammadi F, Taheri A, Eghtesadi-Araghi P. Obstruction of endotracheal tube; a manufacturing error. *Middle East J Anaesthesiol* 2009;20:303–305.
9. Moore RL, Binger CAL. The response to respiratory resistance: a comparison of the effects produced by partial obstruction in the inspiratory and expiratory phases of respiration. *J Exp Med* 1927;45:1065–1080.
10. Galster KT, Mills LD, Silva FR. Postobstructive pulmonary edema in the setting of aspiration and air travel. *J Emerg Med* 2014;47:e143–e146.
11. Bhaskar B, Fraser JF. Negative pressure pulmonary edema revisited: pathophysiology and review of management. *Saudi J Anaesth* 2011;5:308–313.
12. Lathan SR, Silverman ME, Thomas BL, et al. Postoperative pulmonary edema. *South Med J* 1999;92:313–315.
13. Lemyze M, Mallat J. Understanding negative pressure pulmonary edema. *Intensive Care Med* 2014;40:1140–1143.
14. Krodel DJ, Bittner EA, Abdulnour R, et al. Case scenario: acute postoperative negative pressure pulmonary edema. *Anesthesiology* 2010;113:200–207.
15. Kollias-Baker CA, Pipers FS, Heard D, et al. Pulmonary edema associated with transient airway obstruction in three horses. *J Am Vet Med Assoc* 1993;202:1116–1118.
16. Kerr LY. Pulmonary edema secondary to upper airway obstruction in the dog: a review of nine cases. *J Am Anim Hosp Assoc* 1989;25:207–212.
17. 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.
18. 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;25:519–523.
19. Ball MA, Trim CM. Post anaesthetic pulmonary oedema in two horses. *Equine Vet Educ* 1996;8:13–16.
20. Glasser SA, Siler JN. Delayed onset of laryngospasm-induced pulmonary edema in an adult outpatient. *Anesthesiology* 1985;62:370–371.
21. Koh MS, Ling Hsu AA, Eng P. Negative pressure pulmonary oedema in the medical intensive care unit. *Intensive Care Med* 2003;29:1601–1604.
22. Maxwell BG, Mihm FG. Questioning diuretic use in acute negative-pressure pulmonary edema. *Anesthesiology* 2011;114:461.
23. Boutureira J, Trim CM, Cornell KK. Acute pulmonary edema after diazepam-ketamine in a dog. *Vet Anaesth Analg* 2007;34:371–376.
24. Apparatus for administration of anaesthetics. In: Clarke KW, Trim CM, Hall LW, eds. *Veterinary anesthesia*. 11th ed. London: Saunders Elsevier, 2014;209–242.
25. Alibhal H. Breathing systems and ancillary equipment. In: Seymour C, Duke-Novakovski T, eds. *BSAVA manual of small animal anaesthesia and analgesia*. Gloucester, England: British Small Animal Veterinary Association, 2007;30–47.