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American College of Veterinary Anesthesia and Analgesia

# Anesthesia Case of the Month

## History

A 7-year-old sexually intact female 7.4-kg (16.3-lb) Jack Russell Terrier with a history of exercise intolerance, coughing, and weight loss was referred to the Companion Animal Clinic of the Faculty of Veterinary Medicine, Ghent University. The dog had been hit by a car 2 years previously. Thoracic radiographs were taken immediately after the accident at a local veterinary clinic, and a diagnosis of pleural effusion was made. The dog was subsequently hospitalized for several days. After discharge, the dog never completely regained its former activity level. Over the months preceding referral, the dog gradually became exercise intolerant, lost weight, and started to cough. Food and water intake were normal, and neither vomiting nor diarrhea was reported. The dog was currently not receiving any medications.

On physical examination, the dog was bright, alert, and responsive, with a body condition score of 2 of 5. On thoracic auscultation, muffling over the entire right and left ventral aspects of the thorax was noticed; however, in the left dorsal thoracic quarter, lung and heart sounds could be evaluated. Respiratory rate was 64 breaths/min, and heart rate was 108 beats/min. No murmurs or arrhythmias were detected. Metatarsal pulses were strong, symmetric, and regular. Mucous membranes were pink, and capillary refill time was normal. Rectal temperature was 39.1°C (102.4°F). On thoracic radiographs (laterolateral and dorsoventral views), a diaphragmatic hernia was visible, with presence of the stomach, small intestines, and liver in the thoracic cavity. Results of a preanesthetic CBC, serum biochemical analysis, and measurement of electrolyte concentrations were within reference limits. A 22-gauge catheter was placed aseptically in the right cephalic vein. The dog was hospitalized overnight for observation, and surgery (herniorraphy and elective ovariectomy) was scheduled for the next morning.

## Case Management

Results of preanesthetic clinical examination on the morning of surgery were unchanged from the day of admission, and the dog was classified as American Society of Anesthesiologists class III (ie, severe systemic disease). The dog was preoxygenated (2 L/min via a face mask) for 5 minutes while propped up on a tilted table. Premedication consisted of fentanyl (5 µg/kg [2.27 µg/lb], IV) and midazolam (0.25 mg/kg [0.11 mg/lb], IV). After sedation, the dog became sternally recumbent but remained responsive. General anesthesia was induced (0 minutes) with alfaxalone (2.03 mg/kg [0.92 mg/lb], IV) administered to effect. The dog was intubated with a cuffed 6-mm (internal diameter) endotracheal tube, which was connected to a Bain breathing system. Initial flow of oxygen was 2 L/min. Isoflurane administration commenced 5 minutes later (5 minutes). Lactated Ringer's solution was administered (10 mL/kg/h [4.5 mL/lb/h], IV) throughout anesthesia. After induction of anesthesia, the dog was positioned in right lateral recumbency and prepared for surgery. A 26-gauge catheter was placed in the right metatarsal artery for direct monitoring of blood pressure and arterial blood gas measurement (pH, $Paco_2$, $Pao_2$, and derived parameters). Cefazolin (20 mg/kg [9.1 mg/lb], IV, q 2 h during surgery) and dexamethasone (0.5 mg/kg [0.23 mg/lb], IV) were administered after endotracheal intubation.

The dog was moved to the operating room (8 minutes), positioned in dorsal recumbency, and connected to an anesthetic machine with a circle breathing system, integrated ventilator, and patient monitoring system[a] for monitoring of inspiratory and expiratory gas composition, capnography, tidal volume, airway pressures, and respiratory rate as well as pulse oximetry for measuring peripheral hemoglobin oxygen saturation. A separate monitor[b] was used for ECG and direct blood pressure measurement. Initial gas flow was 0.9 L of oxygen/min, mixed with 0.8 L of medical air/min. This was a prophylactic measure used in our hospital in most patients to reduce the likelihood of absorption atelectasis, the process whereby small airways may collapse if proximally obstructed. This may be exacerbated by nitrogen washout, such as that which occurs when patients are exposed to high inspired oxygen concentration. Decreasing the fraction of inspired oxygen may slow or minimize this process.[1] Fraction of inspired oxygen varied between 50% and 65%. Initial vaporizer setting was 1.5% isoflurane. Median expired isoflurane concentration was 1.2% (range, 0.9% to 1.4%) during the surgery. A constant rate infusion of fentanyl (5 µg/kg/h) was administered IV. Intermittent positive pressure ventilation (volume controlled but with pressure limitation set at 13 cm $H_2O$) was started. Normal minute volume for a dog this size was estimated at approximately 1,480 mL/min.[2,3] Because the dog was expected to have a lower minute volume owing to chronic pulmonary compression, the initial ventilator settings were more conservative (respiratory rate, 25 breaths/min; tidal volume, 40 mL [minute volume, 1,000 mL]; inspiratory-to-expiratory ratio, 1:2.5). Twenty minutes prior to celiotomy, positive end expiratory pressure was instituted at 3 cm $H_2O$. Tidal and thus minute volume were slowly increased over time. A tidal volume of 75 mL was reached

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105 minutes after initiating controlled ventilation (peak airway pressure then reached 13 cm $H_2O$).

Arterial blood pressure was mostly stable (mean arterial blood pressure [MAP], 65 to 80 mm Hg). At 24, 59, and 74 minutes after induction, MAP increased (suspected to result from increased nociception), and ketamine (0.5 mg/kg, IV) was injected as treatment, after which blood pressure stabilized again (ie, returned to previous values). One crystalloid fluid bolus (15 mL/kg [6.82 mL/lb], IV, over 15 minutes) was given at 30 minutes to maintain MAP > 60 mm Hg. Peripheral hemoglobin oxygen saturation remained > 93% throughout anesthesia. This was confirmed by repeated arterial blood gas sampling (arterial oxygen saturation, 95% to 100%; $Pa_{O_2}$, 94 to 280 mm Hg). Although moderate hypercapnia appeared to be present (end-tidal $P_{CO_2}$ [$P_{ETCO_2}$] range, 47 to 62 mm Hg), $Pa_{CO_2}$ initially indicated severe alveolar hypoventilation (83 mm Hg at 22 minutes). This was corrected with the gradual increase in minute volume to $Pa_{CO_2}$ of 61 mm Hg at the end of anesthesia with a closed thorax and spontaneous ventilation. This resulted in a correction of the pH (from 7.15 at 22 minutes to 7.22 at 149 minutes).

A ventral midline incision was made, and the diaphragmatic defect was identified, extending from the left costochondral arch to well over the level of the vertebral column. The displaced abdominal organs were repositioned. The left liver lobes had formed several intrathoracic adhesions. Careful adhesiolysis was performed. The borders of the diaphragmatic defect were fibrotic, and the edges could not be mobilized to allow primary closure. A synthetic mesh was sutured over the defect. A 20-gauge IV catheter was inserted via the abdomen through an intact part of the diaphragm on the right side of the thorax for pneumothorax evacuation. A free edge omental flap was positioned over the mesh and sutured in place.

At 143 minutes, a sudden decrease in $P_{ETCO_2}$ was noted, along with a lowering of delivered tidal volume to 30 mL/breath (previously stable at 75 mL/breath) and the alarm of pressure limitation (still set at 13 cm $H_2O$) sounded. The dog became bradycardic (56 beats/min), and arterial blood pressure decreased (MAP, 48 mm Hg). The surgeon was alerted, and communication between the thorax and the atmosphere was restored by lifting the omental flap. The catheter in the mesh became dislodged and was removed from the patient. Cardiovascular and ventilatory parameters quickly stabilized. An 8F thoracostomy tube was inserted in the left hemithorax and left open to the atmosphere. A second attempt was made to close the omental flap over the mesh. Air was removed manually from the thorax as the omental flap was sutured to the mesh through the thoracostomy tube with a 3-way stopcock and syringe, so that the drain was intermittently closed. Again, $P_{ETCO_2}$ decreased and recordings stopped suddenly. This was accompanied by severe bradycardia (lowest value, 35 beats/min), hypotension (MAP, 30 mm Hg), and a flattened arterial waveform trace. Apart from the bradycardia, no abnormalities were observed on the ECG trace. The surgeon was alerted, air was removed from the thorax via the thoracostomy tube at a faster rate, and respiratory rate decreased to 9 breaths/min. A total of 500 mL of air was withdrawn from the thorax. A colloid solution (hydroxyethyl starch) was administered (5 mL/kg, IV, over 15 minutes) to alleviate the hypotension. Heart rate and blood pressure returned to normal values within 15 minutes; $P_{ETCO_2}$ slowly increased to 60 mm Hg. After stabilization, ventilation was gradually restored to preincident values (tidal volume, 75 mL; respiratory rate, 25 breaths/min).

The patient then underwent bilateral ovariectomy, and the abdomen was closed without further complications. After evacuation of all possible air and fluid from the thorax via the thoracostomy tube, bupivacaine (1 mg/kg [0.45 mg/lb]) was injected into the drain with the patient in sternal recumbency. Isoflurane administration and controlled ventilation were discontinued (total duration of anesthesia, 195 minutes) while a bandage was applied. A fentanyl patch (25 µg/h) was placed on the right caudal aspect of the thorax under the bandage, but IV fentanyl administration was continued at a rate of 3 µg/kg/h (1.36 µg/lb/h). Spontaneous ventilation returned within 1 minute. The dog maintained adequate oxygen saturation (assessed by pulse oximetry) readily when breathing room air.

Apart from hypothermia (33.6°C [92.48°F]), recovery was without complications. Normothermia was quickly reached; no shivering was noted, and supplemental oxygen was not administered. Fentanyl administration was maintained at 3 µg/kg/h, IV, for the first 18 hours after surgery, lowered to 2 µg/kg/h (0.91 µg/lb/h) for another 4 hours, then discontinued. The dog was hospitalized in the intensive care unit and observed for changes in respiratory pattern, dyspnea, or cyanosis. Oxygen saturation was measured every hour via pulse oximetry,[c] and the dog was assessed for signs of pain every 2 hours. Respiratory rate varied slightly (median, 24 breaths/min; range, 20 to 40 breaths/min). No abnormal breath sounds were detected on auscultation at any time. The thoracostomy tube was removed 48 hours after surgery, when air was no longer present in the aspirate. The dog was discharged 4 days after surgery. The dog received omeprazole (10 mg, PO) for 3 weeks owing to an incident of vomiting and appearance of melena immediately after surgery. At a postoperative follow-up visit 3 weeks after the surgery, except for the presence of the surgical site, findings on physical examination were normal, body weight had increased to 8.25 kg (18.15 lb), and the surgical incision was clean and healing well. Exercise intolerance was no longer present as reported by the owner.

## Question

What are the factors that should be considered in the anesthetic management of patients with chronic diaphragmatic hernias, and what is the most likely cause of the severe hemodynamic and ventilatory compromise that occurred twice during surgery in this patient?

## Answer

Adequate preparations must be made before anesthetizing a patient with a diaphragmatic herniation because these patients can decompensate suddenly after anesthetic induction. Occasionally, emergency surgical

incision and abdominal organ retraction may even be required because of severe ventilatory compromise, particularly when patients are positioned in dorsal recumbency. To minimize the risk of complications due to insufficient preparation, it is essential to ensure that sufficient trained personnel are present, specific responses to emergency scenarios are discussed ahead of time, and each team member is assigned a defined task in advance of the procedure.

Whenever possible, patients should receive supplemental oxygen before anesthetic induction (eg via a face mask or nasal cannula) and additional stress should be avoided.[2] Rapid airway control via immediate intubation is essential,[2] necessitating a quick IV anesthetic induction technique. Preparations for providing mechanical ventilation need to be in place before inducing anesthesia, given that it may be needed immediately. During the surgery, mechanical ventilation is mandatory.[2] If the patient has chronic diaphragmatic herniation, ventilation should be conservative (high respiratory rate with low tidal volume and peak airway pressure) because the lungs have become atelectatic and cannot support quick reinflation without sustaining damage. The aim is not to reinflate the lungs but merely to maintain oxygenation and normocapnia.[2] This will hopefully prevent ventilator-induced lung injury[4] and reexpansion pulmonary edema.[2]

Positioning the animal with the head and thorax up (reverse Trendelenburg position) by tilting a working table may minimize pressure on the lungs from herniated abdominal organs. Patient preparation including clipping needs to be performed quickly, with the patient in lateral recumbency. Dorsal recumbency should be avoided until positioning for surgery.

The presence of tension pneumothorax was suspected to be the cause of the severe hemodynamic and ventilatory compromise that occurred twice during surgery in this patient, considering the nature of the surgery and the clinical signs observed on the monitors (ie, sudden decrease in and disappearance of $P_{ETCO_2}$, smaller delivered tidal volume, bradycardia, hypotension, and flattening of the arterial waveform). The first incident was resolved by restoring communication between the thorax and the atmosphere and the second by removing air quickly from the thoracic cavity through the thoracic drain.

## Discussion

Animals with chronic diaphragmatic hernias can be initially examined for clinical signs related to the herniation or for completely different problems and have a subclinical herniation.[5] The fact that these patients can decompensate acutely when anesthetized highlights the importance of a thorough preanesthetic examination.[6,7] In 1 study,[8] overall perioperative survival rates for diaphragmatic herniation surgery in dogs and cats were 89.1%. In chronic cases, perioperative mortality rate in dogs has been described as 19% to 21%.[5,8] These data indicate that treatment of chronic diaphragmatic herniation is associated with some risk and both anesthesia and surgery must be approached carefully. Postoperative complications can be expected in as many as 50% of all diaphragmatic hernia patients.[9] In a study[5] of chronic diaphragmatic herniation in dogs and cats, pneumothorax was the most common postoperative complication, occurring in 10% of patients. Postoperative tension pneumothorax has been reported in 1 dog,[5] whereas in human patients, it has been described as an intraoperative complication during laparoscopic diaphragmatic herniorraphy.[10] To our knowledge, this is the first time tension pneumothorax has been described as an intraoperative complication during diaphragmatic herniorraphy in a dog.

Tension pneumothorax is defined as a pleural injury communicating with the atmosphere via a 1-way valve that opens on inspiration and closes on expiration, which will lead to an expanding pneumothorax. Tension can be said to exist when notable respiratory (in awake patients) or cardiovascular (more common in mechanically ventilated patients) compromise occurs. This pathological change can have its origin in the parietal, mediastinal, or visceral pleura (by perforation of the lung parenchyma).[11] Regardless of origin, treatment consists of immediate decompression, usually to atmospheric pressure by restoring communication between the intrapleural space and the atmosphere but, ideally, by reestablishing negative pressure into the intrapleural space.

We hypothesize that the first tension pneumothorax occurring in this dog found its origin in air aspiration via the over-the-needle catheter present in the diaphragm. When the omental flap was placed over the graft, this closed the thorax, while the bellow-like motions of the ventilated lung changed the pressure gradient in the pleural space. The catheter then let air into the pleural space during the expiratory phase, but because of its small diameter, the release of air during the shorter inspiratory phase was more difficult. During the fast intervention by the surgical team (the temporary lifting of the omental flap so that the mesh graft was once again exposed), the catheter became dislodged. The second tension pneumothorax occurred in the presence of an intermittently closed chest tube while air was being evacuated from the pleural space. It seems probable that air evacuation did not occur fast enough. The situation was corrected by speeding up the process of air evacuation and by adapting the ventilation for a short while. Fortunately, the hazardous situation was quickly recognized and adequately managed in both instances. The dog recovered well. Excessive intrapleural pressure seems the most likely cause; however, the cardiovascular collapse could have been caused by excessive airway pressure, which could potentially be caused by several mechanisms.

Positive pressure ventilation has been known to cause ventilator-induced injury by way of barotrauma (alveolar overexpansion resulting from high lung volume with or without high pressure), atelectrauma (alveolar shear-stress injury that occurs with repetitive alveolar recruitment and derecruitment), or biotrauma (alveolar injury secondary to inflammation by cytokines released in response to mechanical injury to the alveolus).[4] Patients with chronic diaphragmatic herniation are potentially more vulnerable than healthy patients and are also known to be susceptible to reexpansion lung injury.[2] This is a complex type of pulmonary edema caused by increased vascular permeability, thought

to be caused by chronically collapsed lungs developing changes in the capillary wall. The abnormal microvasculature is likely to become damaged during rapid pulmonary reinflation.[12] Decreased surfactant activity decreases perivascular pressure, thereby increasing vessel injury, and reperfusion occurs in the damaged blood vessels as oxygen-derived free radicals are produced and leukocytes are sequestered in the region of the injured blood vessels, also increasing capillary permeability.[12] Reexpansion pulmonary edema has been described in humans, cats, and a dog.[12–14] In this case, a conservative ventilation strategy was applied during the intervention. The methods we used (positive end-expiratory pressure, low tidal volume, and low peak airway pressure) have been advocated as lung-protective strategies during mechanical ventilation in human patients.[4] Additionally, the dog received dexamethasone in an effort to increase membrane stabilization as protection against reexpansion lung injury.[15] Sudden surges in delivered volume and flow are both unlikely to have occurred and to have been responsible for the hemodynamic collapse, given that pressure in the breathing system is measured and controlled by the anesthesia machine[a] that was used. When the set maximum pressure is reached, an alarm will sound, and inspiration is adjusted to guarantee airway pressure remains constant to the end of inspiration and the delivered tidal volume is reduced. If peak airway pressure was to exceed the set maximum pressure by 5 cm $H_2O$, inspiration would immediately be terminated, expiration would start, and gas would be released.[16] The anesthesia machine function was tested during the automated self-test performed before use, and no malfunction occurred. In conclusion, the volume-controlled but pressure-limited ventilation would prevent airway pressure from exceeding set limits, which, if carefully selected, makes ventilator-induced lung injury very unlikely.

Although surgical dissection of pulmonary adhesions (often present in chronic diaphragmatic herniation) could lacerate the lungs, also leading to a visceral pleura leak,[5] this seems very unlikely in this case, considering that only minimal adhesions were present. Regardless of potential origin, a visceral leak is unlikely to have occurred in this case, as the intrapleural air disappeared quickly after surgery. If there had been a persistent leak of substantial size in the lungs or bronchi, pneumothorax would have recurred repeatedly and would have persisted for a longer time.

However, excessive airway pressure could also originate from anesthetic equipment problems,[3] and pressure-limited ventilation would not protect against this. Failure to open the automatic pressure limitation valve after testing the system for leaks can be excluded, given that the automatic pressure limitation valve in the anesthetic machine[a] used is isolated from the breathing system during controlled ventilation and therefore could not have caused problems.[16,17] Obstruction of the breathing system (by water, foreign bodies, or kinking) can also increase airway pressure.[3] The presence of water seems unlikely, considering that all breathing systems are habitually dried overnight and the patient was the first animal to undergo surgery that day. Foreign bodies were not observed during visual inspection of the breathing system prior to use. The system was capped after the leak test and subsequently was attached to and was at no time detached from the patient, excluding the entry of foreign bodies into the breathing system. Kinking seems unlikely, considering that the breathing system was not overly long and is made of kink-resistant silicone.

An obstruction in the scavenging system, such as crushing or kinking of the transfer tube, can also cause an excessive buildup of pressure in the breathing system.[18] This causes air to flow into the lungs but prevents expiration. The set maximum pressure alarm of the anesthetic machine would give early warning of such an incident, but the problem could not be solved by expelling more gas out of the scavenging system and airway pressure could still increase, unless (as in this case) the transfer tubing used has an opening at the connection point to the anesthetic machine, which would allow gas to escape even in the presence of an occlusion.

In conclusion, considering the incidents took place at moments when the thoracic cavity was suddenly closed off from the atmosphere, this strongly supports the presence of increased intrapleural pressure rather than excessive airway pressure.

Last, a defective monitoring system could also explain the observed problems. However, 2 monitoring machines for blood pressure and capnography were used, and simultaneous failure would be highly unlikely. Moreover, removing air from the thoracic cavity resulted in a normalization of the cardiorespiratory parameters, confirming functionality of the monitors.

After reviewing all possible causes of the complications seen in this patient, a tension pneumothorax of parietal origin remains the most probable diagnosis. Early recognition by means of continuous monitoring of cardiorespiratory parameters, good communication between surgeon and anesthesiologist, and fast intervention consisting of rapid decompression before physiologic decompensation were imperative for patient survival.

a. Cicero EM with Integrated PM8020 Data Manager, Dräger, Lübeck, Germany.
b. Cardiocap II, Datex-Ohmeda Instrumentarium Corp, Helsinki, Finland.
c. PM-60Vet, Shenzhen Mindray Bio-Medical Electronics Co Ltd, Huntingdon, Cambridgeshire, England.

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