

Anesthesia Case of the Month

Sponsored by the American College of Veterinary Anesthesiologists. Comments and contributions should be directed to Dr. Jeff C. H. Ko, Department of Veterinary Clinical Sciences, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK 74078; telephone: 405/744-8584; e-mail: kjeff@okstate.edu

History

A 7-year-old 60-kg (132-lb) female pot-bellied pig was admitted to the Boren Veterinary Medical Teaching Hospital at Oklahoma State University. The pig was lethargic and had a history of vomiting (regurgitation), especially following eating. Physical examination revealed a heart rate of 100 beats/min, respiratory rate of 40 breaths/min, and pink mucous membranes. A CBC and serum biochemical analysis were performed. Serum hepatic enzyme activities were high, and albumin concentration was high, but all other values were within reference range limits.

The pig was scheduled for abdominal radiography, ultrasound-guided liver biopsy, and endoscopic examination of the upper portion of the gastrointestinal tract, and while anesthetized, the owner wanted the hooves trimmed. Anesthesia was induced with a mixture of tiletamine hydrochloride/zolazepam hydrochloride^a (4.4 mg/kg [2 mg/lb] of body weight), ketamine hydrochloride^b (2.2 mg/kg [1 mg/lb]), and xylazine hydrochloride^c (2.2 mg/kg) in a single IM injection into the cervical epaxial muscles.¹ The pig assumed lateral recumbency in 8 minutes, and endotracheal intubation was achieved 10 minutes after administration of the anesthetic agents. Anesthesia was maintained with isoflurane^d in oxygen with the vaporizer initially set at 2% and oxygen flow set at 2 L/min.

Following induction of anesthesia, the pig was placed in lateral recumbency, and electrocardiographic leads were attached for monitoring the ECG.^e An oscillometric blood pressure monitor and blood pressure cuff^f were used to monitor the pig's blood pressure, with the cuff placed on the forelimb. A small animal pulse oximetry probe was placed on the tongue and connected to a pulse oximeter^g to monitor saturation of hemoglobin with oxygen (SpO₂). The initial blood pressure readings were 130, 110, and 72 mm Hg for systolic, mean, and diastolic blood pressure, respectively. The pig's heart rate (and pulse rate) obtained from a pulse oximeter was 75 beats/min, and respiratory rate was 12 breaths/min. The ECG was normal in appearance. However, the SpO₂ was 70%. Upon examination of the oral mucous membranes and tongue, cyanosis was observed, confirming the pulse oximeter's results

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and further indicating that hypoxemia existed. A quick examination of the anesthetic machine, including the oxygen flowmeter and breathing circuit, revealed no apparent malfunctions. A lateral thoracic radiograph was obtained (Fig 1).

Question

What is the cause of this low saturation of hemoglobin with oxygen and cyanosis in this pig?

Answer

Inadvertent endobronchial intubation resulted in hypoxemia.

Discussion

Pulse oximeters are commonly used during anesthesia to measure oxygen saturation of hemoglobin. Pulse oximeter readings should normally be > 95%. An SpO₂ of 70% indicates serious hypoxemia.² Potential causes of hypoxemia during anesthesia include inspiration of low concentrations of oxygen, hypoventilation, alveolar-capillary diffusion impairment, airway obstruction, hemoglobin abnormalities, anatomic shunting, and ventilation-perfusion mismatching.³ In addition, erroneous readings from the pulse oximeter are possible.

In this pig, the anesthesiologist quickly attempted to rule in or out a number of potential causes of hypoxemia. Hypoxemia secondary to low inspired oxygen concentrations can result from a number of anesthetic equipment-related malfunctions, including the following: a lack of oxygen supply from the oxygen source

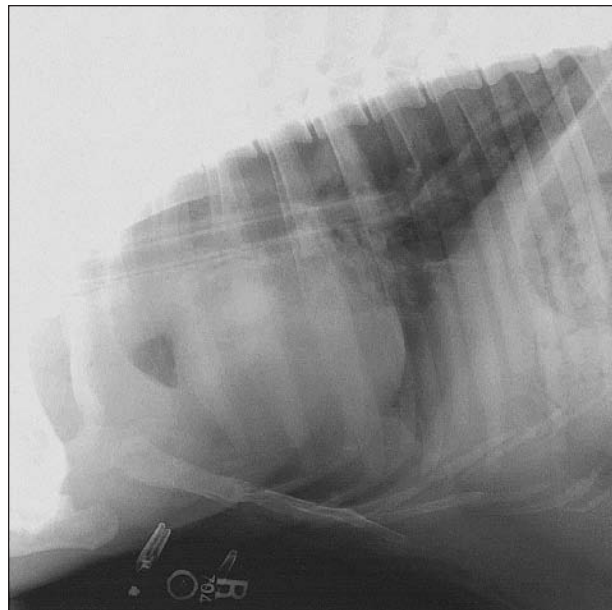


Figure 1—Right lateral thoracic radiographic view of a 7-year-old female pot-bellied pig under general anesthesia.

(depletion of an oxygen cylinder or the central oxygen supply from a pipeline system), improper connection to an oxygen source, failure to turn on the oxygen flowmeter or inadvertently turning off the oxygen flowmeter; delivery of a hypoxic gas mixture (excessive nitrous oxide), and failure to properly connect the breathing circuit to the anesthetic machine or patient. Rapid assessment of the anesthetic machine and breathing circuit did not reveal any of these problems. Although inspired oxygen concentration was not measured, and the hypoxemia developed early in anesthesia while the oxygen concentration was still increasing within the anesthetic circuit, it was unlikely that inspiration of a low concentration of oxygen was a problem. The inspired oxygen concentration was at least that of room air and almost certainly was at some value greater than that of room air and increasing as time progressed.

Hypoventilation is common during anesthesia as a result of the respiratory depressant effects of anesthetic agents and, if severe, potentially can cause hypoxemia. Although the anesthetic drugs used in this pig are known to cause hypoventilation and exacerbate hypoxemia, visual evaluation of the rebreathing bag suggested that the tidal volume was adequate; respiratory rate was 12 breaths/min. We recognized that visual evaluation of the rebreathing bag is only a crude measurement of the adequacy of ventilation. A capnograph would have been helpful in more accurately assessing the adequacy of ventilation by measuring end-tidal CO₂ concentration, but the equipment was not available at the time. Likewise, an arterial blood gas measurement would have provided valuable information, but it was not attempted because of the difficulty in obtaining an arterial blood sample from pigs and the time involved in obtaining results. Regardless, hypoxemia secondary to hypoventilation usually responds rapidly to increasing the inspired oxygen concentration with assisted or controlled ventilation.³ Because manually assisting ventilation did not improve saturation, it was unlikely that hypoventilation was the cause of the hypoxemia.

Hypoxemia resulting from diffusion impairment (eg, interstitial pneumonia, pulmonary edema) was unlikely, because there was no history of pulmonary disease prior to anesthesia and no evidence of such disease processes on physical examination prior to anesthesia or abnormal lung sounds on thoracic auscultation during anesthesia. In addition, hypoxemia secondary to diffusion impairment usually resolves with increased inspired oxygen concentration.³

Endotracheal tube obstruction was ruled out by visual confirmation of fogging of the endotracheal tube, movement of the rebreathing bag, and movement of the 1-way valves in the breathing circuit with each breath. Furthermore, the pig did not have exaggerated respiratory efforts. Hemoglobin abnormalities can potentially produce hypoxemia as the result of impairment of the affinity of abnormal hemoglobin for oxygen, but these are extremely rare, and there was no evidence of hemoglobin problems observed in the physical examination, patient history, or preanesthetic laboratory evaluations.

Anatomic shunting or ventilation-perfusion mismatching may result in substantial hypoxemia that does not respond rapidly to an increase in inspired oxygen concentration.³ Examples of anatomic shunts include congenital cardiac defects such as atrial or ventricular septal defects or a patent ductus arteriosus. Thoracic auscultation of the pig did not reveal any heart murmur at the time of hypoxemia. This, together with the clinically normal findings on the preanesthetic physical examination and the pig's history, made preexisting cardiac shunts unlikely.

Because this pig was obese, substantial ventilation-perfusion mismatch was a distinct possibility. However, our clinical experience has been that positive pressure ventilation often improves ventilation and oxygenation in obese patients. However, another potential cause of substantial ventilation-perfusion mismatching is endobronchial intubation. With endobronchial intubation, only 1 lung is ventilated—the lung in which the endotracheal tube is located. The other lung is not ventilated but is perfused. Therefore, the unventilated lung does not participate in gas exchange, and the blood that flows through that lung remains hypoxic and hypercarbic. Reflex hypoxic pulmonary vasoconstriction, which diverts blood flow away from poorly ventilated sections of lung, may be inhibited by some anesthetics, including isoflurane, resulting in greater hypoxemia.^{4,5} Endobronchial intubation was suspected in this pig. Before intubation, it was recognized that the endotracheal tube was potentially too long for this pig. However, during endotracheal intubation the tube length was overlooked. As abdominal radiographs were obtained at the time hypoxemia was detected, a lateral thoracic radiograph was also obtained.

Radiography (Fig 1) clearly revealed endobronchial intubation in this pig. When the endotracheal tube was moved cranially by approximately 10 cm, the pulse oximeter reading immediately increased from 70 to 99%. To confirm that endobronchial intubation was actually the cause of the hypoxemia, the endotracheal tube was reinserted to its original position, and the SpO₂ decreased to 75%. After correcting the endotracheal tube placement again, anesthesia proceeded smoothly without further incident. Because movement of the endotracheal tube corrected the hypoxia, further radiographic evaluation of the thorax was not completed. A dorsoventral thoracic radiograph would have been useful in determining the extent of pulmonary atelectasis and the exact location of the endotracheal tube in the right or left main stem bronchus.

Findings in this pig demonstrate the potential usefulness of pulse oximeters in the diagnosis of endobronchial intubation. The increase in SpO₂ values with movement of the endotracheal tube cranially should be viewed as an indication that endobronchial intubation was the likely cause of the hypoxemia. In this pig, thoracic radiographs and the knowledge that the endotracheal tube was potentially too long confirmed this conclusion. However, it has been demonstrated in experimental 1-lung intubation in dogs that SpO₂ may not change substantially if the endobronchial intubation

occurs after a high P_{aO_2} has been established.⁶ Pulse oximetry may have been particularly useful to us in this pig, because the endobronchial intubation occurred immediately following anesthetic induction. Falsely low SpO_2 values are caused by several factors, including motion of the lingual probe or excessive ambient light, and these should be ruled out at the same time other causes of hypoxemia are investigated.⁷ Poor perfusion of the tongue or vasoconstriction as the result of anesthetic agents or hypothermia can also cause erroneous SpO_2 values.⁷

A capnograph or similar CO_2 monitor can warn of accidental endobronchial intubation if an acute increase or decrease in end-tidal CO_2 is observed.⁷ If an animal is being ventilated with a pressure-cycled ventilator, endobronchial intubation will result in a reduced tidal volume, hypoventilation, and a subsequent increase in end-tidal CO_2 . With a volume-cycled ventilator, endobronchial intubation will cause a sudden doubling of the alveolar ventilation in the ventilated lung and at least an initial sudden decrease in the end-tidal CO_2 .⁸ Regardless, changes in oxygenation as detected by pulse oximetry will usually be more pronounced than changes in capnometry.^{7,9} Definitive diagnosis of endobronchial intubation can only be made with radiography or bronchoscopy (direct visualization). Normally, the distal tip of the endotracheal tube should rest near the thoracic inlet, ideally, between the first and second intercostal spaces (Fig 2). This is particularly important in swine, because a right

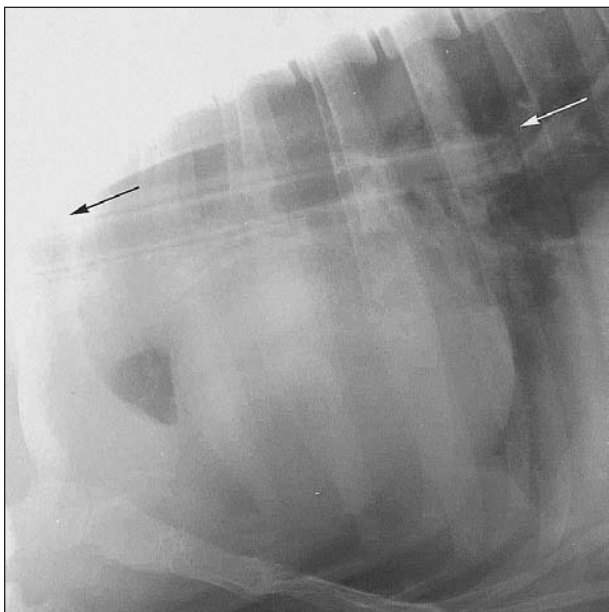


Figure 2—Right lateral thoracic view of a 7-year-old female pot-bellied pig under general anesthesia. Notice the distal end of the endotracheal tube in a caudal main stem bronchus (white arrow). Usually, the distal end of the endotracheal tube should be at the level of the first intercostal space (black arrow).

apical lobe bronchus originates from the trachea at the level of the third rib and ventilates the right apical lobe of the right lung. The trachea then divides into right and left principal bronchi at the level of the fifth intercostal space.¹⁰ In the pig of our report, the tip of the endotracheal tube clearly passed both of these landmarks and rested between the fifth and sixth intercostal spaces.

The situation described here is unique because of the peculiar conformation of pot-bellied pigs. They have short necks with relatively short tracheas compared with other commonly encountered species, which increases the risk of endobronchial intubation. Before intubation, it was recognized that the endotracheal tube was potentially too long for this pig. However, during endotracheal intubation the length of the tube was overlooked until low SpO_2 values from the pulse oximeter alerted the anesthesiologist to the problem. Pulse oximeters are useful warning tools for inadvertent endobronchial intubation.

^aTelazol, Fort Dodge Animal Health, Fort Dodge, Iowa.

^bKetaset, Fort Dodge Animal Health, Fort Dodge, Iowa.

^cSedazine, Fort Dodge Animal Health, Fort Dodge, Iowa.

^dIsoflo, Abbott Laboratories, North Chicago, Ill.

^ePassport LT, Datascope Corp, Paramus, NJ.

^fDinamap model 8300, Critikon Inc, Tampa, Fla.

^gNellcor N-20PA, Nellcor Inc, Pleasanton, Calif.

References

1. Ko JC, Williams BL, Smith VL, et al. Comparison of Telazol, Telazol-ketamine, Telazol-xylazine, and Telazol-ketamine-xylazine as chemical restraint and anesthetic induction combination in swine. *Lab Anim Sci* 1993;43:476-480.
2. Haskins SC. Monitoring the anesthetized patient. In: Thurmon JC, Tranquilli WJ, Benson GJ, eds. *Lumb and Jones' veterinary anesthesia*. 3rd ed. Baltimore: The Williams & Wilkins Co, 1996;409-424.
3. West JB. Gas exchange. *Pulmonary pathophysiology—the essentials*. 4th ed. Baltimore: The Williams & Wilkins Co, 1992;18-40.
4. Karzai W, Haberstroh J, Priebe HJ. The effects of increasing concentrations of desflurane on systemic oxygenation during one-lung ventilation in pigs. *Anesth Analg* 1999;89:215-217.
5. Lennon PF, Murray PA. Attenuated hypoxic pulmonary vasoconstriction during isoflurane anesthesia is abolished by cyclooxygenase inhibition in chronically instrumented dogs. *Anesthesiology* 1996;84:404-414.
6. Cantwell SL, Duke T, Walsh PJ, et al. One-lung versus two-lung ventilation in the closed-chest anesthetized dog: a comparison of cardiopulmonary parameters. *Vet Surg* 2000;29:365-373.
7. Dorsch JA, Dorsch SE. *Understanding anesthesia equipment—construction, care and complications*. 3rd ed. Baltimore: The Williams & Wilkins Co, 1994.
8. Gandhi SK, Munshi CA, Coon R, et al. Capnography for detection of endobronchial migration of an endotracheal tube. *J Clin Monit* 1991;7:35-38.
9. McCoy EP, Russell WJ, Webb RK. Accidental bronchial intubation. An analysis of AIMS incident reports from 1988 to 1994 inclusive. *Anaesthesia* 1997;52:24-31.
10. Getty R. *Sisson and Grossman's the anatomy of the domestic animals*. 5th ed. Philadelphia: WB Saunders Co, 1975.