Effect of one-lung ventilation on oxygen delivery in anesthetized dogs with an open thoracic cavity

Simon T. Kudnig, BVSc, MVS; Eric Monnet, DVM, PhD; Miriam Riquelme, DVM; James S. Gaynor, DVM, MS; Denise Corliss; M. D. Salman, BVMS, PhD

Objective—To evaluate effects of one-lung ventilation on oxygen delivery in anesthetized dogs with an open thoracic cavity.

Animals—8 clinically normal adult Walker Hound dogs.

Procedure—Each dog was anesthetized and subjected to one-lung ventilation during a period when it had an open thoracic cavity. A Swan-Ganz catheter was used to measure hemodynamic variables and obtain mixed-venous blood samples. A catheter was inserted in the dorsal pedal artery to measure arterial pressure and obtain arterial blood samples. Oxygen delivery index was calculated and used to assess effects of one-lung ventilation on cardiopulmonary function. Effects on hemodynamic and pulmonary variables were analyzed.

Results—One-lung ventilation caused significant decreases in PaO₂, arterial oxygen saturation (Sao₂), mixed-venous oxygen saturation, and arterial oxygen content (CaO₂). One-lung ventilation caused significant increases in PaCO₂, physiologic dead space, and alveolar-arterial oxygen difference. Changes in Sao₂, CaO₂, and PaCO₂, although significantly different, were not considered to be of clinical importance. One-lung ventilation induced a significant increase in pulmonary arterial wedge pressure, mean pulmonary artery pressure, and shunt fraction. One-lung ventilation did not have a significant effect on cardiac index, systemic vascular resistance index, pulmonary vascular resistance index, and oxygen delivery index.

Conclusions and Clinical Relevance—One-lung ventilation affected gas exchange and hemodynamic function, although oxygen delivery in clinically normal dogs was not affected during a period with an open thoracic cavity. One-lung ventilation can be used safely in healthy dogs with an open thoracic cavity during surgery. (Am J Vet Res 2003;64:443–448)

Thoracoscopy is a minimally invasive surgical technique that can be used to explore the thoracic cavity, cranial mediastinum, thoracic wall, and thoracic lymph nodes. Operative procedures performed on animals by use of a thoracoscope include partial pedicadectomy, pleural biopsy, lymph node biopsy, lung lobectomy, lung biopsy, correction of persistent right aortic arch, and thoracic duct ligation. Thoracoscopy is replacing surgical thoracotomy as the technique of choice for many surgical procedures, because it provides better visibility of thoracic structures and can decrease the incidence of morbidity associated with thoracotomy.

Thoracoscopy can be performed with bilateral hemithorax ventilation during sustained pneumothorax without substantial effects on cardiopulmonary variables in clinically normal dogs. The sophistication of thoracoscopic procedures is increasing; therefore, the use of one-lung ventilation is desirable to improve visibility and surgical access to intrathoracic structures. Furthermore, one-lung ventilation in humans is superior in terms of surgical visibility and hemodynamic stability, compared to insufflation with carbon dioxide.

One-lung ventilation allows the collapse of lung lobes on the side of the thoracoscopic surgical approach to facilitate observation of intrathoracic structures and to achieve lung immobility. One-lung ventilation can be performed with selective intubation of a mainstem bronchus or by use of a bronchial blocker, and it is the standard technique used for thoracoscopy procedures in humans. The feasibility of one-lung ventilation during thoracoscopy in dogs has been documented. It has been argued, however, that given the risks associated with one-lung ventilation (e.g., hypoxemia, bronchial rupture, and malpositioning of bronchial blockers), bilateral hemithorax ventilation is preferable during diagnostic thoracoscopy in dogs. Because of the induction of a pulmonary shunt, it is expected that there will be a reduction of oxygen tension with one-lung ventilation, even in situations involving a closed thoracic cavity. Opening of the thoracic cavity during thoracotomy or thoracoscopy induces a reduction in arterial oxygen tension attributable to atelectasis in the dependent lung. Therefore, the combination of one-lung ventilation and an open thoracic cavity may be extremely detrimental to the pulmonary function status of patients. Optimization of oxygen delivery (DO₂) is the goal of anesthetists during surgical intervention. Oxygen delivery is the product of arterial oxygen content (CaO₂) and cardiac output (CO). Therefore, instead of only measuring PaO₂ and CaO₂, it would be more relevant to measure DO₂ to assess the effect of one-lung ventilation in situations involving an open thoracic cavity.

The purpose of the study reported here was to evaluate the net effect of one-lung ventilation on DO₂ to tissues during a period with an open thoracic cavity.
Our hypothesis was that one-lung ventilation would not have detrimental effects on DO₂ to peripheral tissues in anesthetized clinically normal dogs with an open thoracic cavity.

**Materials and Methods**

**Animals**—Eight healthy sexually intact purpose-bred male and female Walker Hound dogs that weighed between 25.6 and 29.2 kg were used in the study. All dogs were clinically normal, as determined on the basis of results of physical examination, a CBC, and serum biochemical analyses. This study was approved by the Animal Care and Use Committee at Colorado State University.

**Study design**—Each dog served as its own control animal. Cardiopulmonary variables were recorded during two-lung ventilation with an open thoracic cavity and one-lung ventilation with an open thoracic cavity. A period of 15 minutes was provided after the onset of each experimental condition to allow equilibration of cardiopulmonary variables, and data were then collected. At the completion of the experiment, a 20-F thoracostomy tube was inserted, and sutures were placed in the subcutaneous tissue and skin to close the transthoracic sites. A continuous-rate infusion of fentanyl (2 to 4 µg/kg/h, IV) was administered, and a transdermal fentanyl patch (7.5 mg) was placed to alleviate pain during recovery from anesthesia. The dogs were allowed to recover from anesthesia in a critical care unit that provided standard care similar to that for client-owned animals.

**Anesthesia**—An 18-gauge over-the-needle catheter was inserted in a cephalic vein, and a bolus of lactated Ringer's solution (10 mL/kg) was administered IV. Anesthesia was induced by administration of propofol (3.0 mg/kg, IV) followed by diazepam (0.3 mg/kg, IV). After standard endotracheal intubation, anesthesia was maintained by administration of isoflurane in oxygen (end-tidal concentration of isoflurane, 1.85 to 1.95% [approx. 1.5 times minimum alveolar concentration]) delivered through a precision out-of-circuit vaporizer in a semiclosed circle rebreathing circuit. An agent analyzer was used to measure end-tidal isoflurane concentration. A side-stream capnograph was connected to the endotracheal tube to measure end-tidal partial pressure of carbon dioxide (PETCO₂). Lactated Ringer's solution and dextran 70 were each administered at a rate of 5 mL/kg/h during anesthesia. An esophageal temperature probe was advanced to the level of the heart base to measure core body temperature. To provide additional muscle relaxation, each patient was administered atracurium (0.2 mg/kg, IV, as an initial bolus, followed by 0.1 mg/kg/IV, until the desired effect was achieved). Muscle relaxation was assessed by use of a nerve stimulator applied over the peroneal nerve and observation of the response to a train-of-four electrical impulse. Mechanical ventilation was accomplished by use of a volume ventilator to achieve a P ETCO₂ of 35 to 45 mm Hg throughout the experiment. Respiratory rate (7 to 16 breaths/min) and tidal volume (V̇ₜ; 14 to 15 mL/kg) were maintained constant during the experiment. A respirometer was used to measure respiratory V̇ₜ. A warm water blanket was applied to the endotracheal tube to control body temperature. A 20-gauge over-the-needle catheter was placed in the dorsal pedal artery and connected to a fluid-filled transducer. The pressure transducer was calibrated to 0 at the level of the right atrium. Systolic arterial pressure (SAP), diastolic arterial pressure (DAP), and mean arterial pressure (MAP) were recorded continuously on a pressure monitor. Results of ECG and pulse oximetry were also recorded on the monitor.

Dogs were positioned in right lateral recumbency. An 8-F introducer was placed into the left jugular vein by use of the Seldinger technique. A 7.5-F Swan-Ganz catheter was passed through the jugular vein introducer and advanced to the level of the pulmonary artery. Catheter position was verified by observation of characteristic pressure waveforms on a pressure monitor. The proximal part of the Swan-Ganz catheter was connected to a fluid-filled pressure transducer. The pressure transducer was calibrated to 0 at the level of the right atrium. Systolic pulmonary arterial pressure (SPAP), diastolic pulmonary arterial pressure (DPAP), and mean pulmonary arterial pressure (MPAP) were recorded continuously on the pressure monitor.

**Creation of an open thoracic cavity**—The left hemithorax was clipped and surgically prepped. An open thoracic cavity was achieved by use of a left-sided lateral thoracoscopic approach and insertion of 12-mm cannulas at the fourth, sixth, and tenth intercostal spaces. A 5-mm 30° telescope was inserted at the sixth intercostal space.

**Creation of one-lung ventilation**—One-lung ventilation was achieved by placement of an endobronchial blocker into the left mainstem bronchus. A multiple-port airway adapter was attached to the endotracheal tube, and the endobronchial blocker was passed through the adapter and into the lumen of the endotracheal tube. The endobronchial blocker was then advanced to the level of the left mainstem bronchus by use of direct observation via a 5.3-mm flexible fiberoptic bronchoscope. Under bronchoscopic observation, the silicone balloon of the endobronchial blocker was inflated with air until the left mainstem bronchus was occluded. The left hemithorax was periodically auscultated to verify a lack of respiratory sounds, and one-lung atelectasis was confirmed by thoracoscopic evaluation.

**Collection of data and samples**—Blood samples were collected from the dorsal pedal artery via the inserted catheter, and a blood gas analyzer was used to measure selected variables in the arterial samples. Hemodynamic data were collected via the catheter in the dorsal pedal artery and the Swan-Ganz catheter in the pulmonary artery. The Swan-Ganz catheter was also used to measure mixed-venous variables. Cardiac output was measured by use of a thermodilution technique that involved injection of 10 mL of iced saline (0.9% NaCl) solution through a closed injection system into the right atrium. A CO computer was used to calculate CO. Mean of 3 consecutive CO measurements was used for each data point. Hemoglobin (Hb) content was measured by use of a spectrophotometric hematologic system.

**Calculation of variables**—Several variables were calculated from cardiopulmonary measurements. Cardiac index (CI) was calculated as follows: CI = CO/body surface area. Alveolar oxygen tension (PAO₂) was calculated as follows: PAO₂ = (F I O₂ × [P A – P H₂O]) − (1.2 × P ACO₂), where F I O₂ is the fractional concentration of inspired oxygen, P A is the barometric pressure, and P H₂O is vapor pressure of water (47 mm Hg at 37°C). Arterial oxygen content (CAO₂) was calculated as follows: CAO₂ = (1.36 × Hb concentration × S aO₂) + (0.003 × P aCO₂), where S aO₂ is the arterial oxygen saturation. Mixed-venous oxygen content (CVO₂) was calculated as follows: CVO₂ = (1.36 × Hb concentration × S vO₂) + (0.003 × P vCO₂), where S vO₂ is the mixed-venous oxygen saturation, and P vO₂ is the mixed-venous oxygen content. Pulmonary end-capillary oxygen content (C eO₂) was calculated as follows: C eO₂ = (1.36 × Hb concentration × 100/100) + (0.003 × P aO₂). Systemic vascular resistance index (SVRI) was calculated by use of right atrial pressure (RAP) as follows: SVRI = (MAP – RAP)/CI. Pulmonary vascular resistance index (PVRI) was calculated by use of pulmonary arterial wedge pressure (PAWP) as follows: PVRI = (MPAP – PAWP)/CI.

Unauthenticated | Downloaded 01/12/24 04:06 AM UTC
Oxygen extraction ratio (O₂ ER) was calculated as follows: O₂ ER = (\(\frac{[CaO₂ − CvO₂]}{[CaO₂ − CvO₂]}\)) × 100. Oxygen delivery index (DO₂I) was calculated as follows: DO₂I = CaO₂ × CI × 10.

Shunt fraction (Qs/Qt) was calculated as follows: Qs/Qt = \(\frac{\{[CcO₂ − CaO₂]/[CcO₂ − CvO₂]\}\} × 100\). Physiologic dead space (\(Vd/Vt\)) was calculated as follows: \(Vd/Vt = \frac{[Paco₂ − PetCO₂]}{Paco₂}\). Alveolar-arterial oxygen difference (Pao₂ – Pao₂) was calculated as Pao₂ – Paco₂.

**Discussion**

One-lung ventilation with an open thoracic cavity did not produce DO₂I in clinically normal dogs. The net result of the hemodynamic and respiratory effects of any treatment can be assessed by evaluation of DO₂I. One-lung ventilation induced a decrease in Paco₂ and Saco₂, which resulted in a reduction of CaO₂. However, CI did not change between two-lung and one-lung ventilation, resulting in a negligible effect of one-lung ventilation on DO₂I. A limited number of studies have assessed the effects of one-lung ventilation on hemodynamic and pulmonary variables.

Atelectasis of the nonventilated lung that developed after occlusion of a mainstem bronchus increased the Qs/Qt and resulted in a decrease in Paco₂. One-lung ventilation can cause hypoxemia and increases in Qs/Qt. The Qs/Qt of 30% determined in the study reported here is similar to values for Qs/Qt reported in other studies. The decrease in Paco₂ was not significantly affected by treatment. One-lung ventilation did not have a significant effect on CI, SVRI, SAP, DAP, and MAP. The DO₂I was not significantly affected by treatment.
attributable to the pulmonary shunt resulted in an augmentation of $\text{PAO}_2 - \text{PAO}_2$, a reduction in $\text{SaO}_2$, and a reduction in $\text{CaO}_2$. However, it is important to mention that although the changes in $\text{SaO}_2$ and $\text{CaO}_2$ were significantly different from the initial values, they were only 1 and 3% different from the initial value, respectively, which is of little biological or clinical importance.

Physiologic dead space increased after the induction of one-lung ventilation in the study. The entire $V_T$ is directed into 1 lung, which results in a high ventilation-perfusion ratio ($V/Q$) in the ventilated lung. The alveoli that are still ventilated receive ventilation in excess of the amount typically required. This excess ventilation has been defined as wasted ventilation, and it results in an increase in $V_{Q}/V_{T}$. Overventilated lung units are inefficient at eliminating $CO_2$; therefore, PaCO$_2$ increases. The variation in PaCO$_2$ seen after the onset of one-lung ventilation is not as substantial as the change in PaO$_2$, because the physiologic behavior of the 2 gases differs. The CO$_2$ dissociation curve is almost straight within the physiologic range, with the result that an increase in ventilation will increase the CO$_2$ output of lung units with high and low $V/Q$ values. However, the flat portion at the top of the oxygen dissociation curve means that only alveoli with moderately low $V/Q$ values will benefit from increased ventilation, whereas alveoli with high $V/Q$ values are not able to substantially increase the oxygen concentration of the blood leaving the alveoli. Therefore, the net effect during one-lung ventilation is that the ventilated lung with a high $V/Q$ cannot compensate for the nonventilated lung with a low $V/Q$, resulting in a substantial decrease in PaO$_2$ and more modest hypercapnia. The significant increase in PaCO$_2$ that was detected in the study reported here has little clinical importance. In another study, investigators observed a decrease in $V_{Q}/V_{T}$ after the onset of one-lung ventilation in patients with pulmonary disease. One possible explanation for the observed improvement in $V_{Q}/V_{T}$ in other studies is that cessation of ventilation to poorly perfused diseased portions of the lungs, as opposed to healthy, well-perfused portions of the lungs, can resolve a high $V/Q$ in these areas. Johnson et al documented a significant increase in $V_{Q}/V_{T}$ immediately after the onset of one-lung ventilation in dogs anesthetized with a combination of halothane-pentobarbital, but the effect was only transient. The authors of that study assumed that hypoxic pulmonary vasoconstriction rapidly developed in the blocked lung lobe, thereby increasing the blood flow to the ventilated lung lobe and improving the $V/Q$ mismatch. In the study reported here, we used isoflurane, which can inhibit hypoxic pulmonary vasoconstriction in a dose-dependent manner. Furthermore, isoflurane inhibits hypoxic pulmonary vasoconstriction to a greater degree than do injectable anesthetics.

One-lung ventilation can affect pulmonary vascular resistance. One-lung ventilation can trigger hypoxic pulmonary vasoconstriction in the nonventilated lung. Pulmonary vascular resistance is a function of CO, MPAP, and PAWP. In the study reported here, MPAP and PAWP were significantly increased; however, CI was unchanged. The net effect was that the value for PVRI did not change when calculated in accordance with the standard equation derived from Ohm's law. Transmural pressure at the capillaries, recruitment of capillaries with increases in CO, and hypoxic vasoconstriction are important factors that will affect pulmonary vascular resistance. Transmural pressure is affected by one-lung ventilation, because the entire $V_T$ is redistributed to only 1 lung. Because CI did not change after establishment of one-lung ventilation, increases in MPAP and PAWP were likely to be the result of an increase in peak airway pressure. Therefore, it is recommended during anesthesia in humans to decrease the $V_T$ when one-lung ventilation is established to prevent an increase in airway pressures and concomitant increases in pulmonary vascular resistance.

Hypoxic vasoconstriction is a defense mechanism that redistributes blood flow away from nonventilated alveoli to ventilated alveoli. Hypoxic pulmonary vasoconstriction can be inhibited by a number of factors, including low or high pulmonary vascular pressure, low or high $\text{PaO}_2$, hypocapnia, vasodilating agents, and anesthetic agents. It was beyond the scope of the study reported here to determine the physiologic mechanisms of pulmonary circulation during one-lung ventilation in dogs. Distribution of colored microspheres injected in the pulmonary artery would be required to determine the distribution of blood flow in the pulmonary parenchyma after initiation of one-lung ventilation.

Two limitations were apparent for the study reported here. First, the number of dogs evaluated limited our confidence in the nonsignificant differences between treatments. A power of 80% provides confidence in rejection of the null hypothesis. In our study, the power value for CI and DO$_2$I was < 80%, which places a limitation on our conclusions. The second limitation was that the dogs evaluated were healthy dogs free of cardiopulmonary disease. Despite the observed lack of a detrimental effect of one-lung ventilation on DO$_2$I in the study, close monitoring of cardiopulmonary variables in patients with cardiopulmonary disease is mandatory. In patients with compromised gas exchange, there is an increased risk associated with one-lung ventilation, and means of improving gas exchange, such as positive end-expiratory pressure or continuous positive-airway pressure, should be considered.

In the study reported here, one-lung ventilation had detrimental effects on gas exchange and hemodynamic function, although DO$_2$I in clinically normal dogs was not affected. Therefore, we concluded that one-lung ventilation can be used safely in healthy dogs with an open thoracic cavity.
References


