麻酔器の酸素濃度を調節できます

ヒトの気管内麻酔では酸素と窒素を混合して使用します。

それは、純酸素麻酔による無気肺を防ぐ目的です。

【以前笑気ガスを多用していた時代は、笑気ガス（N_2O）に含まれる窒素で代替していましたが、笑気ガスを使わなくなった近年、人医では酸素以外に窒素ガスを用いてブレンドしています】

ところが多くの動物病院では窒素ガスの準備が困難で、純酸素（ボンベ）での麻酔が一般的でした。
これでは短時間の手術では支障はない様ですが、手術時間が長くなると問題が起きることが指摘されています。

そこで当社では、獣医医療の現場に即した対応として、酸素濃度を 25%～90%範囲で調節可能の酸素濃縮器を世界に先駆けて開発しました。

ご参考にして下さい（本論文は日本大学生物資源科学部獣医学教授　山谷吉樹先生より頂戴いたしました）

Computed tomographic analysis of the effects of two inspired oxygen concentrations on pulmonary aeration in anesthetized and mechanically ventilated dogs

Francesco Staffieri, DMV, PhD; Delia Franchini, DMV, PhD; Giuseppina L. Carella, DMV; Manuela G. Montanaro, DMV; Valerio Valentini, DMV; Bernd Driessen, DVM, PhD; Salvatore Grasso, MD; Antonio Crovace, DMV

Objective—To compare the effect of 2 concentrations of oxygen in inspired gas (fraction of inspired oxygen [FIO_2] 1.0 or 0.4) on pulmonary aeration and gas exchange in dogs during inhalation anesthesia.

Animals—20 healthy dogs.

Procedures—Following administration of acepromazine and morphine, anesthesia was induced in each dog with thiopental and maintained with isoflurane in 100% oxygen (100% group; n = 10) or a mixture of 40% oxygen and air (40% group; 10). Dogs were placed in dorsal recumbency and were mechanically ventilated. After surgery, spiral computed tomography (CT) of the thorax was performed and Pao_2, $Paco_2$, and the alveolar-arterial oxygen tension difference ($P_{(A-a)}O_2$) were assessed. The lung CT images were analyzed, and the extent of hyperinflated (–1,000 to –901 Hounsfield units [HUs]), normally aerated (–900 to –501 HUs), poorly aerated (–500 to –101 HUs), or nonaerated (–100 to +100 HUs) areas was determined.

Results—Compared with the 100% oxygen group, the normally aerated lung area was significantly greater and the poorly aerated and nonaerated areas were significantly smaller in the 40% oxygen group. The time to CT (duration of surgery) was similar in both groups. Although $Paco_2$ was similar in both groups, Pao_2 and $P_{(A-a)}O_2$ were significantly higher in the 100% oxygen group. In both groups, pulmonary atelectasis developed preferentially in caudal lung fields.

Conclusion and Clinical Relevance—In isoflurane-anesthetized dogs, mechanical ventilation with 40% oxygen appeared to maintain significantly better lung aeration and gas exchange than ventilation with 100% oxygen. (*Am J Vet Res* 2007;68:925–931)

Pulmonary atelectasis is a condition in which there is an absence of gas from portions of the lungs because of failure of the alveoli to open or impairment of gas absorption from alveoli.[1] It is known that in 90% of humans with normal lung function before anesthesia, pulmonary atelectasis develops in the most dependent part of the lungs during general anesthesia, and it is considered the major cause of impairment of gas exchange and lung compliance.[1,2] The principal pathophysiologic mechanism that may contribute to the development of atelectasis during anesthesia is a cascade of events, beginning with compression of lung tissue followed by airway closure and absorption of alveolar gas.[1,3]

Abbreviations

FIO_2	Fraction of inspired oxygen
CT	Computed tomography
HU	Hounsfield unit
PEEP	Positive end-expiratory pressure
P_{AW}	Peak airway pressure
$ETCO_2$	End-tidal partial pressure of CO_2
ROI	Region of interest
$P_{(A-a)}O_2$	Alveolar-arterial oxygen tension difference
FRC	Functional residual capacity
V_A/Q	Ventilation-to-perfusion

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From the Dipartimento delle Emergenze e dei Trapianti d'Organo, Sezione di Chirurgia Veterinaria, Università degli Studi di Bari, 70010 Valenzano, Bari, Italy (Staffieri, Franchini, Carella, Montanaro, Valentini, Grasso, Crovace); and the Section of Critical Care/Anesthesia, Department of Clinical Studies, New Bolton Center, School of Veterinary Medicine, University of Pennsylvania, Kennett Square, PA 19348 (Staffieri, Driessen).
Supported by intramural funding of the Dipartimento delle Emergenze e dei Trapianti d'Organo, Sezione di Chirurgia Veterinaria, University of Bari, Bari, Italy.
Address correspondence to Dr. Staffieri.

In humans, administration of a high inspired oxygen fraction of 80% to 100% (ie, FIO_2 0.8 to 1.0) during anesthesia is associated with development of more extensive atelectasis in the dependent lung areas, compared with that which develops during administration of a lower FIO_2 (0.3 to 0.4).[1] Results of several clinical and experimental studies have confirmed this factor as a determinant for atelectasis formation in each phase of anesthesia: induction (preoxygenation),[4,5] maintenance,[6] and prior to extubation.[7] Thus, use of low FIO_2 (0.3 to 0.4) for the maintenance of anesthesia is considered an appropriate technique to reduce atelectasis

formation in humans who do not have preexisting lung disease.[1]

Computed tomography represents the gold standard method for the study of lung aeration and particularly for detection of atelectasis. On the basis of differences in radiographic densities recorded in each individual CT image (expressed in HUs), it is possible to distinguish between hyperinflated (–1,000 to –901 HUs), normally aerated (–900 to –501 HUs), poorly aerated (–500 to –101 HUs), and nonaerated (–100 to 100 HUs) areas of lungs.[8,9]

The use of high FIO_2 is currently standard practice in veterinary anesthesia, but results of systematic analyses that support this practice are lacking to our knowledge. In fact, we are not aware of any studies to investigate how differences in FIO_2 affect lung aeration and, consequently, pulmonary gas exchange in dogs during inhalation anesthesia. The purpose of the study reported here was to compare the effect of 2 FIO_2 conditions (1.0 and 0.4) on pulmonary aeration and gas exchange in isoflurane-anesthetized dogs positioned in dorsal recumbency for abdominal surgery. We hypothesized that administration of high FIO_2 will lead to a greater impairment of lung aeration and gas exchange than administration of lower FIO_2 in dogs.

Materials and Methods

The study was conducted in compliance with the Italian Animal Welfare Act and statutes of the University of Bari relating to the use of client-owned animals in clinical investigations.

Animals—Twenty adult healthy client-owned female mixed-breed dogs scheduled for elective ovariohysterectomy were enrolled in the study after written owner consent had been obtained. An equal number of dogs was randomly assigned to each of 2 groups (designated as the 40% and 100% groups on the basis of the administered FIO_2). Preoperative screening included a CBC, serum biochemical analyses, and thoracic radiography (right lateral view). Dogs with abnormal clinicopathologic findings or physical examination evidence of pulmonary disease were excluded from the study.

Anesthetic procedure and monitoring—Each dog was premedicated with acepromazine[a] (30 µg/kg) and morphine sulphate[b] (0.3 mg/kg) administered IM. Once an adequate level of sedation was achieved, a cephalic vein was catheterized (20-gauge catheter) by use of aseptic techniques, and lactated Ringer's solution was administered (5 mL/kg/h). Thoracic radiography was performed with the dog in right lateral recumbency to exclude major lung disease. Approximately 30 minutes after premedication, anesthesia was induced via IV administration of 10 mg of thiopental[c]/kg. The dog was restrained in sternal recumbency, and endotracheal intubation was performed; the endotracheal tube was connected to a rebreathing circuit with soda lime as a CO_2 absorber. Subsequently, the dog received isoflurane[d] in 100% oxygen (100% group; n = 10) or a gas mixture of 40% oxygen and air (40% group; 10). Five minutes after connection to the breathing circuit, the dog was positioned in dorsal recumbency and was mechanically ventilated by use of a respirator[e] operated in a volume-controlled mode with tidal volumes of 15 mL/kg, an inspiratory-to-expiratory ratio of 1:2, an inspiratory hold of 25% of the inspiration time, zero PEEP, and a P_{AW} limit of 20 cm H_2O. Respiratory rate was adjusted to maintain an $ETCO_2$ of 35 to 45 mm Hg. A continuous lead II ECG; heart rate; systolic, diastolic, and mean arterial pressures (determined at the left dorsal metatarsal artery by use of a noninvasive oscillometric technique); oxygen saturation as measured by pulse oximetry; FIO_2; end-tidal isoflurane concentration; $ETCO_2$; P_{AW}; plateau airway pressure; tidal volume; and minute ventilation were continuously monitored throughout anesthesia. The multigas analyzer unit[f] was calibrated prior to each experiment by use of gas standards. At the end of the surgical procedure, CT of the thorax was performed and an arterial blood sample was withdrawn from the right femoral artery. The dog was kept in dorsal recumbency throughout the procedure until the end of the CT procedure. The interval between placement into dorsal recumbency and commencement of the CT procedure (ie, the time to scan) was recorded.

CT and analysis of lung densities—Lung aeration and distribution of atelectasis were analyzed by means of a spiral CT scanner.[g] At the end of surgery, each dog was maintained in dorsal recumbency and transported to the nearby CT scanner, whereupon the endotracheal tube was reconnected to the anesthesia machine and mechanical ventilation with the same FIO_2 administered during surgery (0.4 or 1.0) was recommenced. The dog was positioned in the scanner in dorsal recumbency, and a dorsal plane scout image that extended over the thorax was obtained. Spiral CT of both lungs was then performed during end-expiration apnea. All images were obtained at a setting of 120 kVp and 160 mA by use of a lung algorithm; matrix size was 512 × 512, field of view was 35, and pitch was 1.5. Images of 10-mm slice thickness were reconstructed.

All CT images were analyzed for lung abnormalities; if pathologic changes were detected, the dog was excluded from the study. An operator (VV) who was unaware of the FIO_2 administered analyzed the CT images by means of a computer program.[h] Both right and left lungs were chosen as ROIs for analysis by manually drawing the outer boundary along the inner aspect of the ribs and the inner boundary along the mediastinal organs.[9] The ROIs were drawn by use of a bone window for the outer boundary along the inner aspect of the ribs (window width, 2,000; window level, 200) and a lung window for the inner boundary along the mediastinal organs (window width, 1,600; window level, –600; **Figure 1**). The total area (mm^2) of right and left lungs was calculated by including pixels with density values of –1,000 to +100 HUs.[9] The computer software plotted the distribution of radiographic attenuations (HUs) among the selected ROIs. In accordance with previous human studies,[8,9] we identified the following regions or compartments within the lungs: hyperinflated (ie, composed of pixels with CT numbers of –1,000 to –901 HUs), normally aerated (ie, composed of pixels with CT numbers of –900 to –501 HUs), poorly aerated (ie, composed of pixels with CT numbers of –500 to –101 HUs), and nonaerated (ie, composed of pixels with CT numbers of −100 to +100 HUs and indicating complete atelectasis). The area (mm^2) of each compartment in each CT image was calculated. For each dog, the data acquired in each CT image were then added together to yield the total area that each compartment occupied within both lungs. Numeric surface area values of each compartment were expressed as a percentage of total lung surface area. In addition, all slices performed in each dog were subdivided equally into apical (cranial), median, and caudal fields, and the percentage of total atelectasis in each field of both the right and left lungs was calculated in both study groups (100% and 40% groups). Moreover, in each group, the percentage of total atelectasis in each field (apical, median, and caudal) of the right and left lung was separately calculated.

Evaluation of gas exchange—During CT imaging, temperature-corrected Pa_{O_2} and Pa_{CO_2} were determined. The $P_{(A-a)}O_2$ was calculated in each dog by use of a formula as follows:

$$P_{(A-a)}O_2 = (PB - PH_2O) \times FI_{O_2} - Pa_{CO_2}/R - Pa_{O_2}$$

where PB is the barometric pressure at sea level (760 mm Hg; Bari is located at sea level); PH_2O is the water vapor pressure at 37°C (47 mm Hg); and R is the respiratory exchange ratio, which is assumed to be 0.9 in dogs.[10]

Statistical analysis—Data are reported as mean ± SD. Demographic data, hemodynamic and respiratory variables measured during anesthesia, total lung surface area analyzed, pulmonary aeration compartments, Pa_{CO_2}, and $P_{(A-a)}O_2$ for the 2 study groups were compared. In addition, the relative distributions (%) of atelectasis in the apical (cranial), median, and caudal lung fields were compared between the 2 study groups, between lung fields in each group, and between the right and left lung in each group. Statistical analysis included a 1-way ANOVA, and a value of $P < 0.05$ was considered significant.

Results

There were no significant differences between the 40% and 100% groups with respect to age (3.2 ± 1.2 years and 3.1 ± 1.4 years, respectively); weight (20.6 ± 7 kg and 20.3 ± 7.3 kg, respectively); time to scan (62.9 ± 8.2 minutes and 62.2 ± 8.1 minutes, respectively); or mean heart rate (103 ± 10 minutes^{-1} and 104 ± 12 minutes^{-1}, respectively), mean arterial pressure (73.0 + 6.4 mm Hg and 72.3 ± 6.9 mm Hg, respectively), respiratory rate (9 ± 1 minutes^{-1} and 9 ± 1 minutes^{-1}, respectively), P_{AW} (14.3 ± 2.7 cm H_2O and 14.5 ± 2.6 cm H_2O, respectively), plateau airway pressure (13.8 ± 2.6 cm H_2O and 13.6 ± 2.6 cm H_2O, respectively), $ETCO_2$ (37.6 ± 2.7 mm Hg and 36.8 ± 2.2 mm Hg, respectively), oxygen saturation as measured by pulse oximetry (99.1 ± 1.1% and 99.3 ± 0.7%, respectively), and end-tidal isoflurane concentration (1.5 ± 0.1% and 1.4 ± 0.5%, respectively) measured during anesthesia.

In all dogs, the CT procedure was completed during end-expiration apnea, thereby taking advantage of the apnea in the immediate period following the sudden discontinuation of mechanical ventilation. The CT procedure required < 1 minute for completion in all dogs, and immediately after the end of the procedure, mechanical ventilation was resumed. Lung aeration and gas exchange data were collected (**Table 1**); the mean lung area (mm^2) distribution of radiographic attenuations (HUs) within the selected ROIs in both groups was assessed (**Figure 2**). The total analyzed lung surface area was similar in both groups. In dogs ventilated at an FI_{O_2} of 0.4, the percentage of normally aerated lung area

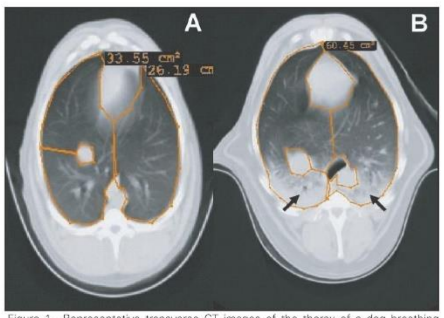

Figure 1—Representative transverse CT images of the thorax of a dog breathing 40% oxygen (A) and thorax of a dog breathing 100% oxygen (B) during isoflurane anesthesia. The boundaries of the ROIs are drawn manually (orange line) on each image. Atelectatic (nonaerated) areas (arrows) in the dependent lung fields are present in the dog breathing 100% oxygen. The numbers indicate the surface area of the ROIs.

Table 1—Pulmonary aeration and blood-gas exchange variables (mean ± SD) in isoflurane-anesthetized and dorsally recumbent dogs undergoing mechanical ventilation with gas mixtures containing either 40% or 100% inspired oxygen (40% and 100% groups, respectively).

Variable	40% group (n = 10)	100% group (n = 10)
Total lung surface area* (cm^2)	8,966 ± 4,756	10,517 ± 1,801
Aeration status (%)†		
Hyperinflated	2.5 ± 2.8	1.2 ± 0.8
Normally aerated	77.1 ± 5.0	58.9 ± 8‡
Poorly aerated	17.5 ± 6.4	26.7 ± 5.3‡
Nonaerated	2.5 ± 0.9	12.8 ± 3.7‡
$P_{(A-a)}O_2$ (mm Hg)	35.6 ± 11.7	176.7 ± 49.2‡
Pa_{O_2} (mm Hg)	211.4 ± 11.9	499.4 ± 49.0‡
Pa_{CO_2} (mm Hg)	38 ± 4	37 ± 3

*Total lung surface area derived via computer-assisted analysis of radiographic attenuation (HUs) in CT images. †Percentage of total lung surface area that was classified as hyperinflated (−1,000 to −901 HUs), normally aerated (−900 to −501 HUs), poorly aerated (−500 to −101 HUs), and nonaerated (−100 to +100 HUs). ‡Value significantly ($P < 0.05$) different from 40% group value.

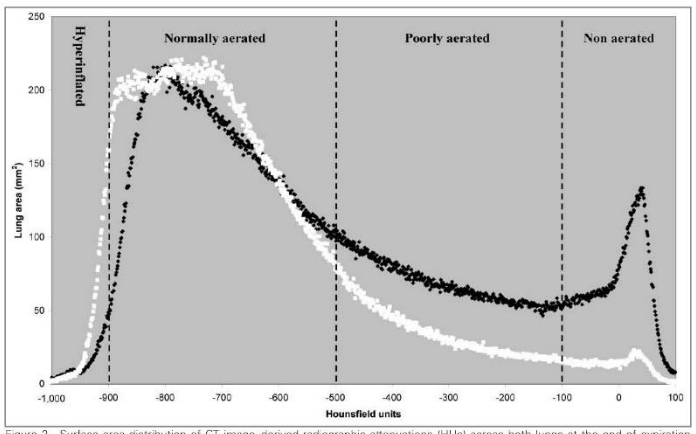

Figure 2—Surface area distribution of CT image–derived radiographic attenuations (HUs) across both lungs at the end of expiration in isoflurane-anesthetized and dorsally recumbent dogs undergoing mechanical ventilation with gas mixtures containing either 40% (white circles; n = 10) or 100% inspired oxygen (black circles; 10). Vertical dashed lines delineate hyperinflated, normally aerated, poorly aerated, and nonaerated lung compartments defined by computer iteration of HUs.

Table 2—Regional distribution (%) of atelectasis within the lungs of isoflurane-anesthetized and dorsally recumbent dogs undergoing mechanical ventilation with gas mixtures containing either 40% or 100% inspired oxygen.

Lung field	40% group (n = 10)	100% group (n = 10)
Apical	3.7 ± 2.3*	2.4 ± 1.1*
Median	46.6 ± 16.6	29.6 ± 6.0†
Caudal	50.0 ± 16.0	67.9 ± 6.7*†

*Within a group, value was significantly ($P < 0.05$) different from the median field value. †Within a field, value was significantly ($P < 0.05$) different from that in the 40% group.

was significantly greater and the percentages of poorly aerated and nonaerated compartments were significantly smaller than those values in dogs ventilated at an FI_{O_2} of 1.0. Mean Pa_{CO_2} was similar in both groups, whereas Pa_{O_2} and $P_{(A-a)}O_2$ were significantly higher in the 100% group, compared with findings in the 40% group. The percentage of atelectasis in the apical (cranial) lung field was similar in both groups but significantly lower than the percentage of atelectasis in the median and caudal lung fields (**Table 2**). In the 40% group, atelectasis was almost equally distributed across the median and caudal lung fields. In the 100% group, a significantly greater degree of atelectasis was detected in the caudal lung field, compared with the median lung field. Moreover, the percentages of atelectatic surface area in the median and caudal lung fields were significantly higher in the 100% group, compared with values in the 40% group. In each study group, there was no difference in formation or distribution of atelectasis in affected lung fields between the right and left lungs.

Discussion

The key finding of the present study in dogs undergoing inhalation anesthesia was that ventilation with 40% of inspired oxygen maintained significantly better lung aeration and gas exchange than ventilation with 100% oxygen. Compared with findings in dogs inhaling 40% oxygen, inhalation of 100% oxygen was associated with significant increases in nonaerated (increase of 10.3%) and poorly aerated (increase of 9.2%) lung areas and a reduction (decrease of 18.2%) in normally aerated lung areas. These changes negatively affected gas exchange, and $P_{(A-a)}O_2$ was 176.7 ± 49.2 mm Hg in the 100% group and 35.6 ± 11.7 mm Hg in the 40% group.

In 1963, Bendixen et al[11] detected a progressive decrease in lung compliance and gas exchange in anesthetized humans and animals during inspiration of oxygen-enriched gas mixtures. Following the advent of CT, Brismar et al[12] determined that dense areas could be detected in dependent regions of both lungs of humans within 5 minutes of induction of anesthesia. Results of a morphologic study[13] of similar dense areas in animals supported the diagnosis of atelectasis.

The technique used to characterize the aeration pattern of lungs on the basis of HUs (defining hyperinflated, normally aerated, poorly aerated, and nonaerated areas) was originally applied in humans with acute respiratory distress syndrome[14] but has since been ap-

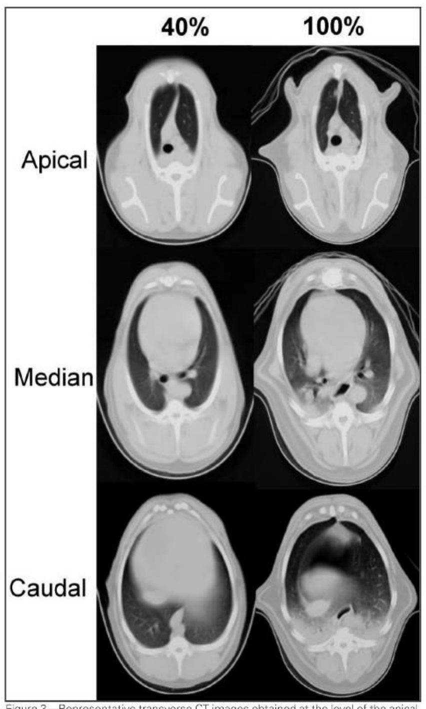

Figure 3—Representative transverse CT images obtained at the level of the apical, median, and caudal lung fields of the thorax of a dog breathing 40% oxygen and a dog breathing 100% oxygen during isoflurane anesthesia. The dog's sternum is at the top of each image.

plied in several clinical and experimental studies[15,16] in animals. To our knowledge, this is the first study to apply this classification in a clinical context in dogs; nevertheless, this classification has already been applied in previous experimental studies[15,16] in dogs. In 1 study,[16] the mean HU values representative of normally aerated lung areas in healthy dogs ventilated with 100% oxygen were −790 to −870 HUs.

Anesthesia-induced atelectasis is a well-known confounding factor that may influence the interpretation of diagnostic lung CT images.[16] For this reason, in dogs undergoing thoracic CT for evaluation of the lungs, the scanning procedure is usually performed during inspiratory breath holding at 15 to 20 cm H_2O, so that the alveoli that were collapsed at the end of expiration are reopened.[16] In nonparalyzed dogs, a brief period of hyperventilation before the inspiratory breath hold allows CT to be performed while the dogs are apneic. Unlike a clinical situation, the specific aim of our study was to quantify the degree of anesthesia-induced atelectasis; therefore, in accordance with another study,[17] we performed the CT procedure at the end of expiration. Moreover, because CT was performed during the period of apnea that follows the sudden discontinuation of mechanical ventilation in dogs undergoing anesthesia with isoflurane, hyperventilation was avoided, which could have induced resolution of part of the anesthesia-induced atelectasis. In the present study, spiral CT of the lungs was conducted and the procedures each required < 1 minute for completion; thus, the CT examination was successfully completed during apnea in each dog.

Pulmonary atelectasis during anesthesia may be caused by compression of lung tissue, airway closure, and absorption of alveolar gas.[1,3] Airway closure and absorption of alveolar gas (absorption atelectasis) are the 2 mechanisms that are influenced primarily by differences in FI_{O_2}.[1,18] In humans, induction of anesthesia causes a reduction of the FRC of the lungs, compared with FRC in the awake state.[3,19] In contrast, the lung closing capacity is not influenced to the same extent by anesthesia.[19] Hence, there is a change in the relationship between FRC and closing capacity during anesthesia, with the closing capacity exceeding FRC in the distal part of the bronchial tree.[18,19] Because of this alteration, there is complete or partial closure of the distal portions of the airways and lung units with pockets of trapped gas or alveoli with a low V_A/Q ratio (ie, poorly aerated areas) are formed.[1,18] Mixed-venous blood continues to perfuse areas in the lung affected by airway closure, which causes progressive gas uptake by continuing blood flow and results in alveolar collapse and atelectasis. The rate of absorption of gas from an unventilated lung area increases with an increase of FI_{O_2}.[18,20,21] In poorly aerated areas (ie, areas characterized by partial airway closure), the inspired V_A/Q ratio of a lung unit decreases. Eventually, a point is reached where the rate at which inspired gas enters the alveolus is equal to the amount of gas taken up from the alveolus into blood. This point is known as the critical V_A/Q ratio.[9,22] If the inspired V_A/Q ratio is lower than the critical V_A/Q ratio, the lung unit will collapse. This is more likely to occur when FI_{O_2} is high and thus gas uptake is large.[9,22]

To explain the results of pulmonary aeration assessments in the present study, we must assume that in the 100% group, the high FI_{O_2} induced more rapid collapse of lung units affected by airway closure (contributing to an increase in atelectasis), collapse of more lung units with low V_A/Q ratio (contributing to an increase in atelectasis), and formation of more lung units with low V_A/Q ratio (contributing to an increase of poorly aerated areas) than developed in dogs ventilated with an FI_{O_2} of 0.4. In a previous study in dogs, Lundquist et al[23] identified a lack of pulmonary densities in dependent lung regions of dogs that were ventilated with room air during barbiturate anesthesia. The results of that study are in agreement with our findings and would suggest that further reduction of FI_{O_2} to 0.21 may be associated with almost complete absence of nonaerated areas in lungs of anesthetized dogs.

As in humans, atelectatic areas were found predominantly in the caudal dependent lung field[1,3] (ie, cranial to the diaphragm) in the dogs of the present study. The diaphragm separates the intrathoracic cavity from the abdominal cavity and allows different pressures to exist in the thorax and abdomen. After induction of anesthesia, the diaphragm relaxes and moves cranially; therefore, it is less effective in maintaining different pressures in the 2 body cavities. More specifically, the pleural pressure increases much more in the dependent portion of the thorax, compressing adjacent lung tissue.[3] In dogs ventilated at an FI_{O_2} of 0.4, the small degree of atelectasis (2.5 ± 0.9%) was almost equally distributed between median and caudal lung fields, whereas in dogs ventilated at an FI_{O_2} of 1.0, a significantly greater amount of atelectasis was present in the caudal lung field, compared with that in the median and cranial lung fields.

Formation of atelectatic units and units with a low V_A/Q ratio is responsible for impairment of gas exchange. In collapsed lung areas that are still perfused, a complete shunt situation develops with lack of any gas exchange. Perfusion of regions with low V_A/Q ratio will also impede oxygenation of blood to an extent that is directly related to the change of the V_A/Q ratio.[18] Thus, compared with the 40% group, the significantly greater amount of nonaerated and poorly aerated lung areas and the smaller amount of normally aerated lung area in the 100% group can explain the significant increase in $P_{(A-a)}O_2$ in this group. The significantly lower Pa_{O_2} in dogs ventilated with 40% inspired oxygen (211.4 ± 11.9 mm Hg), compared with dogs ventilated with 100% oxygen (499.4 ± 49 mm Hg), was attributed to the lower FI_{O_2} administered in the former group. However, the Pa_{O_2} achieved in the 40% group can still be considered a safe level of arterial oxygenation, especially if it is associated with a low $P_{(A-a)}O_2$ gradient.

Atelectasis also plays an important role in the postoperative period. In humans, the formation of pulmonary atelectasis during anesthesia is an important factor for the onset of postoperative hypoxemia because atelectasis resolves only within 24 hours after surgery.[1,3,9] Results of recent studies[24-26] have indicated that hypoxemia during the postoperative period could be an important complication in dogs that have undergone abdominal surgery during anesthesia with volatile agents delivered in 100% oxygen, even in dogs without preexisting lung disease. Although more studies are needed to better define the time necessary to resolve anesthesia-induced atelectasis and the impact of atelectasis formation on gas exchange in the postoperative period in dogs, one may assume that there is a correlation between development of anesthesia-related pulmonary atelectasis and hypoxemic events following anesthesia. In addition, atelectasis may contribute to the development of pneumonia after surgery, secondary to bacterial entrapment in alveoli.[1,9]

The use of low FI_{O_2} is considered a preventative measure to reduce formation of absorption atelectasis. In humans, PEEP and recruitment maneuvers can also be applied for intraoperative treatment of anesthesia-induced atelectasis.[1,3] The application of increasing levels of PEEP can be useful for the re-expansion of collapsed alveoli. Some patients require high levels of PEEP to re-expand atelectatic lung areas, potentially causing pronounced impairment of important hemodynamic and respiratory functions that can limit its application.[3,9] Application of low levels of PEEP from the beginning of anesthesia could be a better strategy to prevent atelectasis formation. The recruitment maneuver is a technique that has been used in humans to re-expand collapsed alveoli via pulmonary hyperinflation. It involves administration of breaths of sufficient tidal volume to cause airway pressures to increase to 30 to 40 cm H_2O.[3] Various protocols of recruitment maneuver for use in humans have been reported, also in combination with PEEP.[3] The high airway and intrathoracic pressures that are achieved during the recruitment maneuver limit its application to relatively short episodes (10 to 15 seconds' duration) to avoid severe impairment of hemodynamic and pulmonary functions. After a recruitment maneuver, reduction of FI_{O_2} allows the re-expanded alveoli to remain open for a longer period (40 minutes), compared with maintenance of high FI_{O_2}, which favors the recollapse of previously expanded alveoli within 5 minutes.[6] Future studies are required to evaluate the efficacy of the recruitment maneuver and PEEP techniques of ventilation in minimizing the extent of anesthesia-induced atelectasis in anesthetized dogs. Regardless, in the present study of healthy dogs positioned in dorsal recumbency and undergoing inhalation anesthesia with mechanical ventilation, an FI_{O_2} of 0.4 provided significantly greater lung aeration and gas exchange than that achieved with an FI_{O_2} of 1.0.

a. Prequillant 1%, Fatro SpA, Bologna, Italy.
b. Morfina Cloridrato Molteni 1%, Molteni SpA, Firenze, Italy.
c. Pentotal Sodium, Gellini SpA, Aprilia, Italy.
d. Isoba, Shering-Plough SpA, Milano, Italy.
e. Ohmeda 7850 ventilator, Datex Ohmeda, Helsinki, Finland.
f. Ohmeda Modulus CD, Datex Ohmeda, Helsinki, Finland.
g. GE ProSpeed SX, General Electric Co, Milwaukee, Wis.
h. DicomWorks, version 1.3.5; 2000.2002, inviweb, Philippe *PEUCH – Loic BOUSSEL*. Available at: dicom.online.fr/fr/download.htm. Accessed Mar 12, 2006.

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