The effect of oxygen concentration on arterial blood partial pressure of oxygen in dogs under general anesthesia

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Abstract

Oxygen is used for medical treatment and general anesthesia. However, high concentrations of oxygen can have toxic effects on cells. In veterinary medicine, 100% oxygen is usually used during general anesthesia and it can be toxic to animals. However, there is little concern about its harmful effects in humans. The objective of this study was to demonstrate that using a high concentration of oxygen increases the partial pressure of oxygen in arterial blood (PaO₂) more so than a lower concentration, by comparing PaO₂ at three different oxygen concentrations (100%, 60%, and 40%) in six dogs under general anesthesia for 3 hours. The mean PaO₂ and standard error values at the 100%, 60%, and 40% oxygen concentrations were 535.8 ± 24.01, 374 ± 17.19, and 239 ± 8.78 mmHg, respectively (p<0.05). These results show that 100% and 60% oxygen concentrations could increase oxidative stress. Further studies are needed to examine the oxygen concentration that causes toxicity.

Key words: arterial blood gas, dog, general anesthesia, oxidative stress, oxygen concentration, partial pressure of oxygen in arterial blood
Introduction

Oxygen is used in many medical fields, such as to restore tissue oxygen tension during cyanosis, shock, or cardiac/respiratory arrest. In addition, oxygen is used to carry inhaled anesthesia gas in the anesthesia machine. However, inhalation of high concentrations of oxygen or pure oxygen for long durations can cause oxygen toxicity (Patel 2003, Mach et al. 2011). Oxygen toxicity occurs in cells exposed to a sufficiently enough concentration of oxygen for a sufficient duration, where the cells eventually die (Mach et al. 2011).

Oxygen is usually used at a 100% concentration in general anesthesia machines, and no concerns about oxygen toxicity have been reported. Nevertheless, newer machines adjust the concentration of oxygen, so a lower concentration of oxygen can be used, thus decreasing the likelihood of toxicity. The purpose of this study was to examine whether use of different concentrations of oxygen during anesthesia affects the partial pressure of oxygen in arterial blood (PaO$_2$) during general anesthesia.

Materials and Methods

In this study, we used six healthy beagle dogs (four males and two females; mean weight, 16.18 kg). All dogs were treated in accordance with guidelines approved by the Animal Use Committee of Iwate University. Complete blood counts and blood chemistry values were checked the day before anesthesia. All dogs were premedicated with 0.04 mg/kg atropine subcutaneously, 0.3 mg/kg midazolam intravenously (IV) and 0.2 mg/kg butorphanol IV. Then, 7 mg/kg propofol IV was used to induce anesthesia, and the trachea was intubated. Arterial blood gases were checked by collecting 0.5 ml blood from the femoral artery. Arterial blood gases were measured before and 3 hours after sevoflurane general anesthesia. All dogs were anesthetized for 3 hours using one of three anesthesia protocols: i. sevoflurane with 100% oxygen; ii. sevoflurane with 60% oxygen; or iii. sevoflurane with 40% oxygen; the protocols were applied randomly to each dog with an at-least 1-week interval between them. The maintenance dosage of sevoflurane was 2-3% in oxygen/normal air at 3 L/min. The PaO$_2$ values were compared before and after general anesthesia, and between the three anesthesia protocols, using the paired t-test and analysis of variance (run in R software, ver. 3.4.1; R Foundation for Computing, Vienna, Austria), respectively.

Results and Discussion

All dogs were healthy. The complete blood count, blood chemistry, and vital signs values were all within the normal ranges. Before anesthesia, the mean PaO$_2$ ± standard error of the mean (SEM) was 59.65 ± 1.99
mmHg. After anesthesia, the mean $\text{PaO}_2$ of the 100%, 60%, and 40% oxygen groups was $535.8 \pm 24.01$, $374 \pm 17.19$, and $239 \pm 8.78$ mmHg, respectively. The $\text{PaO}_2$ values before and after anesthesia were significantly different ($p<0.01$). In addition, the $\text{PaO}_2$ value after anesthesia was significantly different among the three groups ($p<0.01$).

In this study, the mean $\text{PaO}_2$ before anesthesia was lower than the normal range of $\text{PaO}_2$ (80-100 mm Hg). It has been reported that a propofol infusion can decrease $\text{PaO}_2$, transiently, both in humans (65.1 mm Hg) and dogs (57.75-92.25 mmHg) (Vainio 1991, Yamakage et al. 1999). The other reason for the lower $\text{PaO}_2$ before anesthesia was that the dogs were intubated with an endotracheal tube during the propofol infusion, but were not given any oxygen until after the arterial blood was collected.

A previous study showed that oxidative stress increases with increasing $\text{PaO}_2$ and that oxygen toxicity initially occurs at partial pressures of 340-1,215 mm Hg in the lung (Smith 1899). In this present study, the mean $\text{PaO}_2$ values after anesthesia in the 100%, 60%, and 40% oxygen groups were $535.8$, $374$, and $239$ mmHg, respectively (Fig. 1), suggesting that the 100% and 60% oxygen concentrations may have increased oxidative stress. However, in this study, the vital signs (heart rate, respiratory rate, $\text{SpO}_2$, and blood pressure) were stable during all anesthesia protocols, even at 100% oxygen concentration. The 40% oxygen concentration may have been safer than the others. One previous study showed that oxygen concentrations > 40% should be avoided to prevent retrolental fibroplasia in neonates (Patel 2003).

Reactive oxygen species are created under hyperoxic conditions, which may destroy cells and tissues (Patel 2003). To prevent hyperoxia or oxygen toxicity, $\text{PaO}_2$ should be less than 80-160 mmHg (Patel 2003, Mach et al. 2011). However, oxygen toxicity during general anesthesia in animals should be studied further. Finally, the percentage oxygen concentration should be considered to avoid oxygen toxicity. In conclusion, our results suggest that a high concentration of oxygen increases the $\text{PaO}_2$ and risk of hyperoxia.

References


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