Arrhythmogenic effect of hypercapnia in ducks anesthetized with halothane

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Objective—To determine effects of hypercapnia on arrhythmias in ducks anesthetized with halothane.

Animals—12 ducks, 6 to 8 months old, weighing 1.1 to 1.6 kg.

Procedures—Each duck was anesthetized with a 1.5% mixture of halothane in oxygen, and anesthetic depth was stabilized during a 20-minute period. We added CO₂ to the inspired oxygen to produce CO₂ partial pressures of 40, 60, and 80 mm Hg in the inspired gas mixture. The CO₂ partial pressure was increased in a stepwise manner. When arrhythmias were not evident during inhalation of the gas mixture at a specific CO₂ partial pressure, the CO₂ partial pressure was maintained for 10 minutes before a sample was collected for blood gas analysis. When arrhythmias were detected, a sample for blood gas analysis was collected after the CO₂ partial pressure was maintained for at least 2 minutes, and CO₂ inhalation then was terminated.

Results—During the stabilization period, PaCO₂ (mean ± SD) was 33 ± 5 mm Hg, and arrhythmias were not detected. In 6 ducks, arrhythmias such as unifocal and multifocal premature ventricular contractions developed during inhalation of CO₂. Mean PaCO₂ at which arrhythmias developed was 67 ± 12 mm Hg. In 5 of 6 ducks with arrhythmias, the arrhythmias disappeared after CO₂ inhalation was terminated.

Conclusion and Clinical Relevance—Analysis of data from this study indicated that hypercapnia can lead to arrhythmias in ducks during halothane-induced anesthesia. Thus, ventilatory support to maintain normocapnia is important for managing ducks anesthetized with halothane. (Am J Vet Res 2001;62:127–129)

In mammals, halothane sensitizes the myocardium to the arrhythmogenic action of catecholamines. Cardiac arrhythmias such as premature ventricular contraction (PVC), ventricular tachycardia, and ventricular fibrillation sometimes are associated with halothane-induced anesthesia in birds. On the other hand, studies have documented that halothane increases PaCO₂ in a dose-dependent manner in birds during spontaneous ventilation. In mammals, hypercapnia induces stimulation of the sympathetic nervous system and increases plasma concentrations of catecholamines. In addition, hypercapnia can affect cardiovascular performance.

Materials and Methods

The study was conducted in accordance with guidelines established by an institutional animal care and use committee. Twelve healthy male and female domestic Mallard ducks (Anas boschas), 6 to 8 months old and weighing between 1.1 and 1.6 kg, were included in the study. A nonrebreathing anesthetic circuit was used. Anesthesia was induced, using a mask, with a 4% mixture of halothane in oxygen (3 L/min). After induction, each duck was positioned in left-lateral recumbency, and tracheal intubation was accomplished using an endotracheal tube with a low-pressure cuff. The cuff was gently inflated as required, and controlled ventilation with a pressure-limited ventilator was started. The inspiratory-to-expiratory ratio (I:E), respiratory rate, and maximal airway pressure (measured at the ventilator) were initially set at 1:5, 10 breaths/min, and 5 to 7 cm H₂O, respectively. Ventilatory settings were adjusted to maintain end-tidal CO₂ partial pressure between 30 and 40 mm Hg. Flow rate for inspired gas was maintained at 2 L/min. An ECG (lead II) was monitored continuously throughout the anesthetic period. An arterial catheter in each duck was cannulated via an incision to enable continuous measurement of arterial pressure and collection of arterial blood samples. Blood gas analysis was performed immediately after blood samples were collected, and results were compensated for body temperature. Body temperature was monitored via an esophageal thermometer and maintained at 39.5 to 41.5°C, using an electrical heating pad or lamp. In some ducks, PVC were observed after induction of anesthesia or during surgery. When PVC were detected, the concentration of halothane was decreased to a concentration at which the arrhythmia disappeared. After cannulation, the vaporizer setting for the concentration of halothane was adjusted to 1.5%, and each anesthetized duck was stabilized for 20 minutes. Following basal blood gas analysis, a 10% CO₂-90% O₂ gas mixture was added to the inspired oxygen to produce CO₂ partial pressures of 40, 60, and 80 mm Hg. The CO₂ partial pressure in the inspired gas mixture, which was monitored with a multiple-gas monitor, was increased in a stepwise manner. Flow rate of the inspired gas during CO₂ inhalation was maintained at 2 L/min by reducing the oxygen flow rate. When arrhythmias were not evident during inhalation at a specific CO₂ partial pressure, the CO₂ partial pressure was maintained for 10 minutes before a sample was collected for blood gas analysis. Inhalation of CO₂ was terminated after data were collected at a CO₂ partial pressure of 80 mm Hg. When arrhythmias were detected, a sample was collected for blood gas analysis after the CO₂ partial pressure was main-
arrhythmias were not detected in 6 ducks. The mean PaCO2 developed in the 6 ducks was 67 ± 12 mm Hg.

Results

Hemodynamic data and results of blood gas analyses were summarized (Table 1). During the 20-minute stabilization period, PaCO2 was 33 ± 5 mm Hg, and arrhythmias were not detected (Fig 1). In 5 ducks, arrhythmias such as unifocal and multifocal PVC developed during inhalation of CO2 at 40 mm Hg in the inspired gas mixture. In 1 duck, arrhythmias developed during inhalation of CO2 at 60 mm Hg in the inspired gas mixture. Mean PaCO2 at which arrhythmias developed in the 6 ducks was 67 ± 12 mm Hg. Heart rate and arterial blood pressure could not be evaluated, because arrhythmias caused values for those variables to be irregular. In 5 of the 6 ducks with arrhythmias, the arrhythmias disappeared after CO2 inhalation was terminated. In these ducks, once arrhythmias developed, they lasted until CO2 inhalation was terminated. In 1 duck with an arrhythmia, the arrhythmia did not disappear even when CO2 inhalation was discontinued.

In the remaining 6 ducks, CO2 administration did not induce development of arrhythmias. Mean arterial pressure after termination of CO2 inhalation was significantly lower than that recorded before CO2 inhalation. In 1 duck, a sample could not be collected for blood gas analysis after termination of CO2 inhalation, because the catheter became disconnected after measurement of the heart rate and arterial blood pressure that time point but before the blood sample could be collected.

Discussion

In the study reported here, ventricular arrhythmias developed during inhalation of CO2 in 6 of 12 ducks anesthetized with a 1.5% mixture of halothane. Most arrhythmias were easily detected by their bizarre appearances. The rhythm and arterial blood pressure were irregular when the arrhythmias developed. Goelz et al.1 reported the development of arrhythmias in 4 of 8 spontaneously breathing Pekin ducks anesthetized with a mixture of 2.0 to 2.5% halothane. Ludders et al. also reported the development of arrhythmias in 5 of 9 spontaneously breathing Pekin ducks during halothane-induced anesthesia at a halothane concentration as low as 1.15%. The number of ducks developing arrhythmias in our study is similar to that reported in those studies.

In mammals, halothane sensitizes the myocardium to catecholamine-induced arrhythmias.1,2 This, in combination with endogenous catecholamines that are released when ducks are restrained during induction with halothane, is considered to be a cause of arrhythmias.3 In the study reported here, arrhythmias also were observed in some ducks after induction of anesthesia or during the surgical period. Restraint during induction or surgical stimulation may have facilitated development of arrhythmias in those ducks. However, when the concentration of halothane was maintained at 1.5% and PaCO2 was maintained at 33 ± 5 mm Hg, arrhythmias did not develop in any ducks during the

![Figure 1](image-url)
20-minute stabilization period. This PaCO₂ value is considered to be almost normocapnic, because PaCO₂ of conscious ducks is reportedly 28 to 38 mm Hg.10,15

Arrhythmias developed in 6 ducks during inhalation of CO₂. In humans, hypercapnia can cause arrhythmias during halothane-induced anesthesia.3,4,16 Thus, halothane per

onetheless, we believed that the inspired halothane concentration remained relatively constant even when CO₂ was administered. In birds, the use of halothane at concentrations ranging from 1 to 2% is recommended by some authors.14,15 A mean Pa

CO₂ was reported in spontaneously breathing Pekin ducks anesthetized with halothane at a concentration of 1.04 to 1.58%.4 This indicates that the PaCO₂ we detected could be typical during halothane-induced anesthesia in spontaneously breathing ducks.

Although hypercapnia has been reported during anesthesia induced by use of halothane and isoflurane,5,6 arrhythmias develop more commonly during halothane-induced anesthesia.3,4,16 This indicates that the PaCO₂ during halothane-induced anesthesia.

In the study reported here, anesthesia was maintained with a 1.5% mixture of halothane in oxygen. Inspired and end-tidal halothane concentrations were not measured. However, because the flow rate for inspired gas was maintained at a constant value throughout the study, we believed that the inspired halothane concentration remained relatively constant even when CO₂ was administered. In birds, the use of halothane at concentrations ranging from 1 to 2% is recommended by some authors.14,15 A mean PaCO₂ ranging from 57.0 to 113.3 mm Hg was reported in spontaneously breathing Pekin ducks anesthetized with halothane at a concentration of 1.04 to 1.58%.4 This indicates that the PaCO₂ we detected could be typical during halothane-induced anesthesia in spontaneously breathing ducks.

Although hypercapnia has been reported during anesthesia induced by use of halothane and isoflurane,5,6 arrhythmias develop more commonly during halothane-induced anesthesia.3,4,16 Thus, halothane per se also would play an important role in development of arrhythmias. In an ostrich anesthetized with isoflurane, PVC were observed, and it was speculated that hypercapnia had developed.17 However, the association between the development of arrhythmia and hypercapnia was unclear in that ostrich. Because use of isoflurane is recommended in avian species, determination of the effects of hypercapnia during isoflurane-induced anesthesia would be useful.

Increases in the inspired concentration of CO₂ to ducks anesthetized with halothane resulted in arrhythmias in half of the birds. Thus, ventilatory support to maintain normocapnia is important for managing ducks anesthetized with halothane.

References