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RESEARCH PAPER

Cardiovascular, respiratory, electrolyte and acid–base balance during continuous dexmedetomidine infusion in anesthetized dogs

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Abstract

Objective To evaluate the cardiovascular, respiratory, electrolyte and acid-base effects of a continuous infusion of dexmedetomidine during propofol-isoflurane anesthesia following premedication with dexmedetomidine.

Study design Prospective experimental study.

Animals Five adult male Walker Hound dogs 1–2 years of age averaging 25.4 ± 3.6 kg.

Methods Dogs were sedated with dexmedetomidine 10 μg kg^{-1} IM, 78 ± 2.3 minutes (mean ± SD) before general anesthesia. Anesthesia was induced with propofol (2.5 ± 0.5 mg kg^{-1}) IV and maintained with 1.5% isoflurane. Thirty minutes later dexmedetomidine 0.5 μg kg^{-1} IV was administered over 5 minutes followed by an infusion of 0.5 μg kg^{-1} $hour^{-1}$. Cardiac output (CO), heart rate (HR), ECG, direct blood pressure, body temperature, respiratory parameters, acid-base and arterial blood gases and electrolytes were measured 30 and 60 minutes after the infusion started. Data were analyzed via multiple linear regression modeling of individual variables over time, compared to anesthetized baseline values. Data are presented as mean ± SD.

Results No statistical difference from baseline for any parameter was measured at any time point. Baseline CO, HR and mean arterial blood pressure (MAP) before infusion were 3.11 ± 0.9 L $minute^{-1}$, 78 ± 18 beats $minute^{-1}$ and 96 ± 10 mmHg, respectively. During infusion CO, HR and MAP were 3.20 ± 0.83 L $minute^{-1}$, 78 ± 14 beats $minute^{-1}$ and 89 ± 16 mmHg, respectively. No differences were found in respiratory rates, PaO_2, $PaCO_2$, pH, base excess, bicarbonate, sodium, potassium, chloride, calcium or lactate measurements before or during infusion.

Conclusions and clinical relevance Dexmedetomidine infusion using a loading dose of 0.5 μg kg^{-1} IV followed by a constant rate infusion of 0.5 μg kg^{-1} $hour^{-1}$ does not cause any significant changes beyond those associated with an IM premedication dose of 10 μg kg^{-1}, in propofol-isoflurane anesthetized dogs. IM dexmedetomidine given 108 ± 2 minutes before onset of infusion showed typical significant effects on cardiovascular parameters.

Keywords cardiac output, cardiovascular, dexmedetomidine, infusion, LiDCO, lithium dilution.

Introduction

The alpha-2 agonist dexmedetomidine has been recommended to provide reliable sedation, analgesia and chemical restraint in dogs (Bloor et al. 1992; Kuusela et al. 2001; Alvaides et al. 2008; Congdon

et al. 2011). It has also been shown to reduce the requirement of isoflurane when administered as a bolus (Weitz et al. 1991) and also when used as a constant rate infusion in dogs (Pascoe et al. 2006; Uilenreef et al. 2008). The cardiovascular, respiratory and acid–base changes from bolus administration of alpha-2 agonists have been well characterized in dogs in other studies (Bloor et al. 1992; Lemke et al. 1993; Alvaides et al. 2008; Congdon et al. 2011). Concern for the decrease in heart rate, cardiac output and subsequent potential for decreases in oxygen delivery associated with dexmedetomidine may lead to hesitation over the use of constant rate infusions despite its' analgesic effects and reduction in requirements for inhaled anesthetics.

Low dose dexmedetomidine infusions in the range of 0.1–3 μg kg^{-1} $hour^{-1}$ have been previously investigated and reported on in the literature (Pascoe 2005; Braz et al. 2008; Lin et al. 2008; Uilenreef et al. 2008) and have generally found that even low dose infusions have the typical cardiovascular responses seen at higher doses, while there is a trend toward increasing severity and duration of side effects as dose increases. Medetomidine infusions from 0.2–3 μg kg^{-1} $hour^{-1}$ have also been evaluated and have shown similar results (Grimm et al. 2005; Gomez-Villamandos et al. 2008; Carter et al. 2010; Kaartinen et al. 2010) with respect to increasing severity and duration of side effects with increasing dose. Many of these studies included an intravenous bolus or loading dose of dexmedetomidine or medetomidine before beginning infusion(s), in the range of 0.2–3 μg kg^{-1}. Only one study used a dose of 3 μg kg^{-1} administered intravenously (Uilenreef et al. 2008). Despite this wealth of information about infusions of these agents at low doses with or without small loading doses, to the author's knowledge no studies have evaluated these cardiovascular parameters of a microdose dexmedetomidine bolus and subsequent infusion in dogs after a larger intramuscular dose of dexmedetomidine at or near 10 μg kg^{-1}. The purpose of the current study then, was to characterize cardiovascular, respiratory, acid base, hemoglobin and electrolyte changes during a 1 hour infusion of dexmedetomidine at 0.5 μg kg^{-1} $hour^{-1}$ during isoflurane anesthesia following premedication with dexmedetomidine.

Methods

The design and methods for the current study were approved by the University's Institutional Animal Care and Use Committee. Five 1 year old male intact Walker Hound dogs weighing 25.4 ± 3.6 kg were used in this study. The dogs were found to be healthy on physical examination, were interacted with daily, fed twice daily and water was continuously available during non-testing periods. Food was withheld the morning of experiments. The dogs were restrained in lateral recumbency and instrumented with a 20-gauge (Becton Dickinson, Insyte, 48 mm, UT, USA) in the dorsal pedal artery, and two 18-gauge venous catheters in the cephalic and lateral saphenous veins. Lidocaine 0.2 mL was infused subcutaneously over the dorsal pedal artery 5 minutes prior to catheter placement to prevent pain from arterial catheter introduction. Catheters were flushed with 2 mL heparinized saline after placement.

After instrumentation, the dogs were sedated with dexmedetomidine 10 μg kg^{-1} IM 78 ± 2.3 minutes before induction of general anesthesia for an unrelated study. Cardiovascular, respiratory and blood gas data were collected before IM dexmedetomidine for this unrelated study in the exact manner as done for the current study, as described below. At the completion of this study anesthesia was induced with propofol 2 mg kg^{-1} IV over 5–10 seconds to allow intubation. If orotracheal intubation was not possible, additional propofol boluses of 1 mg kg^{-1} boluses were given every 15–25 seconds until intubation was possible. Study subjects were intubated and maintained with 1.5% isoflurane in oxygen. Dogs were placed in lateral recumbency after induction and allowed to breathe spontaneously for the duration of the study. Body temperature was maintained between 37.2 and 38 °C by placing the dogs on an electric warming blanket (HotDog, Augustine Biomedical, MN, USA) and covering with blankets if necessary. Lactated Ringer's solution was delivered at 3–5 mL kg^{-1} $hour^{-1}$ during general anesthesia via the cephalic catheter. Thirty minutes were allotted after induction for stabilization of depth of anesthesia before control measurements and onset of infusion. After baseline measurements, dexmedetomidine 0.5 μg kg^{-1} IV was administered with a precision syringe pump via the cephalic catheter as a loading dose over 5 minutes followed by a constant rate infusion of dexmedetomidine at 0.5 μg kg^{-1} $hour^{-1}$ for 60 minutes. The dexmedetomidine bolus was started at 108 ± 2 minutes after IM dexmedetomidine premedication.

Data for the current study were measured before the dexmedetomidine bolus ('baseline') and then at 5, 30 and 60 minutes during the infusion. Data

