Veterinary Anaesthesia and Analgesia
Formerly the Journal of Veterinary Anaesthesia

Veterinary Anaesthesia and Analgesia, 2011, **38,** 467–474 doi:10.1111/j.1467-2995.2011.00644.x

RESEARCH PAPER

Hemodynamic effects of butorphanol in desflurane-anesthetized dogs

Paulo Sérgio P dos Santos*, Newton Nunes†, Almir P de Souza†, Márlis L de Rezende†, Celina TD Nishimori†, Danielli P de Paula† & Patrícia C Ferro Lopes†

*Department of Clinical Surgery and Animal Reproduction, College of Agricultural and Veterinary Sciences, Universidade Estadual Paulista, Araçatuba, SP, Brazil

†Department of Veterinary Medicine and Surgery, College of Agricultural and Veterinary Sciences, Universidade Estadual Paulista, Jaboticabal, SP, Brazil

Correspondence: Paulo Sérgio P dos Santos, Departamento de Clínica, Cirurgia e Reprodução Animal, Faculdade de Medicina Veterinária, Campus Araçatuba, Universidade Estadual Paulista, Rua Clóvis Pestana, 793, Bairro Dona Amélia, CEP 16050-680, Araçatuba, SP, Brasil. E-mail: patto@fmva.unesp.br

Abstract

Objective To evaluate the effects of butorphanol on cardiopulmonary parameters in dogs anesthetized with desflurane and breathing spontaneously.

Study design Prospective, randomized experimental trial.

Animals Twenty dogs weighing 12 ± 3 kg.

Methods Animals were distributed into two groups: a control group (CG) and butorphanol group (BG). Propofol was used for induction and anesthesia was maintained with desflurane (10%). Forty minutes after induction, the dogs in the CG received sodium chloride 0.9% (0.05 mL kg^{-1} IM), and dogs in the BG received butorphanol (0.4 mg kg^{-1} IM). The first measurements of body temperature (BT), heart rate (HR), arterial pressures (AP), cardiac output (CO), cardiac index (CI), central venous pressure (CVP), stroke volume index (SVI), pulmonary arterial occlusion pressure (PAOP), mean pulmonary arterial pressure (mPAP), left ventricular stroke work (LVSW), systemic (SVR) and pulmonary (PVR) vascular resistances, respiratory rate (f_R), and arterial oxygen (PaO_2) and carbon dioxide ($PaCO_2$) partial pressures were taken immediately before the administration of butorphanol or sodium chloride solution (T0) and then at 15-minute intervals (T15–T75).

Results In the BG, HR, AP, mPAP and SVR decreased significantly from T15 to T75 compared to baseline. f_R was lower at T30 than at T0 in the BG. AP and f_R were significantly lower than in the CG from T15 to T75. PVR was lower in the BG than in the CG at T30, while $PaCO_2$ was higher compared with T0 from T30 to T75 in the BG and significantly higher than in the CG at T30 to T75.

Conclusions and clinical relevance At the studied dose, butorphanol caused hypotension and decreased ventilation during desflurane anesthesia in dogs. The hypotension (from 86 ± 10 to 64 ± 10 mmHg) is clinically relevant, despite the maintenance of cardiac index.

Keywords anesthetic agent, monitoring, opioid, thermodilution.

Introduction

Many published studies have described using the combination of inhalational general anesthetics with opioids to promote balanced anesthesia, which is frequently used in routine procedures to provide analgesia and decrease anesthetic requirements and

subsequent cardiovascular depression (Ko et al. 2000; Nunes et al. 2001).

Butorphanol is a synthetic kappa opioid agonist and mu opioid antagonist and has been used extensively in a wide variety of veterinary species (Lamont & Mathews 2007). The use of this drug alone promotes minimal changes in cardiopulmonary function and the respiratory depression caused by this opioid is less than that induced by morphine, a pure mu agonist opioid (Trim 1983).

Minimal changes were observed in pH, bicarbonate concentration, $PaCO_2$ and PaO_2 in dogs treated with acepromazine and oxymorphone or butorphanol (Cornick & Hartsfield 1992) and even epidural administration of this opioid, at a dose of 0.25 mg kg^{-1}, induces minimal cardiorespiratory depression and does not have adverse neurologic effects (Troncy et al. 1996).

In dogs, butorphanol combined with inhalational anesthetics decreases heart rate (HR), arterial pressure (AP) (Greene et al. 1990; Quandt et al. 1994) and cardiac output (CO) (Tyner et al. 1989), but does not decrease pulmonary arterial pressure (PAP) and pulmonary arterial occlusion pressure (PAOP) (Tyner et al. 1989; Greene et al. 1990).

The dose of butorphanol used in dogs is 0.1–0.8 mg kg^{-1} via intravenous (IV), intramuscular (IM) or subcutaneous (SC) routes (Hosgood 1990; Lamont & Mathews 2007). The onset time of this drug is about 16 minutes when administered intramuscularly (IM) (Kojima et al. 1999) and its analgesic duration ranges from 1 to 3 hours (Hosgood 1990). In dogs anesthetized with isoflurane (MAC 1.28 ± 0.14%), administration of butorphanol (0.4 mg kg^{-1}) significantly reduced the MAC of this inhalant anesthetic (1.03 ± 0.22%) (Ko et al. 2000). However, in dogs anesthetized with halothane or enflurane the anesthetic sparing effect of this opioid agonist-antagonist was minimal (Murphy & Hug 1982, Quandt et al. 1994).

Desflurane is a volatile anesthetic agent introduced into clinical practice in 1992 (Weiskopf 1995). The low blood/gas solubility coefficient of this drug (0.42) allows rapid increases or decreases in alveolar concentration, providing fast induction and recovery (Eger 1992). In humans, desflurane induces dose dependent decreases in CO, AP and systemic vascular resistance (SVR) (Patel & Goa 1995; Pagel et al. 1998), while in dogs it promotes a decrease in AP and increase in HR (Clarke et al. 1996). PAP and PAOP increase in humans when a high concentration of desflurane is used (Weiskopf et al. 1991). In spontaneously breathing animals, desflurane causes dose dependent respiratory depression (Eger 1992; Clarke et al. 1996).

The aim of this study was to evaluate the effects of butorphanol on some cardiopulmonary parameters in dogs anesthetized with desflurane and allowed to breathe spontaneously.

Material and methods

This study was approved by the Institutional Ethics and Animal Welfare Committee (protocol nº 003724-05). After the experiment, the animals were offered for adoption.

Twenty adult mongrel dogs, eleven males and nine females, weighing 12 ± 3 kg, were enrolled in the study. All animals were determined to be healthy based on clinical and laboratory evaluation. Complete blood count, serum biochemistry, urinalysis, electrocardiography and thoracic radiographs were performed and were normal compared with reference intervals described in dogs by Feldman et al. (2000), Kaneko et al. (1997), Osborne & Stevens (1981) and Tilley & Burtinick (1999), respectively.

The animals were provided with water and regular dog food and kept in individual cages at the Veterinary Hospital. Food, but not water, was withheld for 12 hours prior to the experiment.

The dogs were randomly assigned to one of two groups: CG (control group) and BG (butorphanol group). Anesthesia was induced with intravenous (IV) propofol (Diprivan; Zeneca Farmacêutica do Brasil Ltda., Brazil) to effect to allow tracheal intubation. After intubation, desflurane (Suprane; Zeneca Farmacêutica do Brasil Ltda) was administered at a concentration of 10% or 1.4 MAC (MAC = 7.2%; Doorley et al.1988) in oxygen at a flow rate of 30 mL kg^{-1} $minute^{-1}$, using a re-breathing anesthetic circuit with partial rebreathing of gases (Mod. Excel 210SE; Datex Ohmeda, WI, USA) and equipped with a calibrated desflurane vaporizer (Mod. TEC 6; Datex Ohmeda).

The dogs were positioned in dorsal recumbency on a heating pad (T/pump Localized Heat Therapy System – Model Tpp522; Gaymar Industries, Inc., NY, USA) and a teflon catheter (Angiocath 20G; Becton Dickinson Indústria Cirúrgica Ltda, Brazil) was surgically placed in the left femoral artery to