

RESEARCH PAPER

## Thermographic imaging of superficial temperature in dogs sedated with medetomidine and butorphanol with and without MK-467 (L-659'066)

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### Abstract

**Objective** To record, with a thermal camera, peripheral temperature changes during different sedation protocols and to relate the results to changes in the rectal temperature.

**Study design** Randomized crossover part-blinded experimental study.

**Animals** Eight healthy purpose-bred neutered Beagles (two females and six males) weight  $14.5 \pm 1.6$  kg (mean  $\pm$  SD) and aged 3–4 years.

**Methods** Each dog was sedated four times. Treatments were medetomidine  $20 \mu\text{g kg}^{-1}$  and butorphanol  $0.1 \text{ mg kg}^{-1}$  (MB) with or without MK-467  $500 \mu\text{g kg}^{-1}$  (MK). Both drug combinations were administered IV and IM as separate treatments. A thermal camera (T425, FLIR) with a resolution of 320 by 240 was used for imaging.

The dogs were placed in lateral recumbency on an insulated mattress. Digital (DFT) and metatarsal footpad temperatures (MFT) were measured with thermography. Thermograms and rectal temperature (RT) were taken before and at 3, 10, 20, 30, 45 and 60 minutes after treatment.

**Results** At 60 minutes after drug administration, MFT was higher ( $p < 0.001$ ) after MB+MK

( $34.5 \pm 1.1$  IV,  $34.8 \pm 0.5$  IM) than MB ( $31.1 \pm 2.9$  IV,  $30.5 \pm 3.6$  IM), DFT was higher ( $p < 0.001$ ) after MB+MK ( $33.6 \pm 1.4$  IV,  $34.0 \pm 0.6$  IM) than MB ( $26.7 \pm 1.4$  IV,  $26.7 \pm 2.5$  IM), and RT was lower ( $p < 0.001$ ) after MB+MK ( $36.7 \pm 0.8$  IV,  $36.9 \pm 0.3$  IM) than MB ( $37.5 \pm 0.3$  IV,  $37.4 \pm 0.4$  IM), with both routes. The change from baseline was greater with MB+MK than MB in all variables.

**Conclusions** Superficial temperature changes can be seen and detected with thermography. MK-467 used with MB resulted in increased superficial temperatures and a decline in rectal temperature compared to MB alone.

**Clinical relevance** The sedation protocol may influence core temperature loss, and may also have an effect on thermographic images.

**Keywords** butorphanol, dog, medetomidine, MK-467, sedation, thermography.

### Introduction

Medetomidine is an alpha-2-adrenergic agonist, which is used widely in small animal practice. In addition to its beneficial sedative and analgesic effects on the central nervous system, medetomidine also causes some unwanted cardiovascular side

effects, such as vasoconstriction and bradycardia. The medetomidine-induced vasoconstriction is mediated via post-synaptic alpha-2-adrenoceptors located in vascular smooth muscle, and it results in an increased systemic vascular resistance and cardiac afterload (Pypendop & Verstegen 1998). Central alpha-2-adrenoceptors mediate cardiovascular depression, such as a decrease in blood pressure, heart rate and cardiac output, by means of central sympathetic inhibition and vagal activation (Kobinger 1983).

In dogs, butorphanol is used frequently in combination with medetomidine in order to improve the sedative and analgesic (Ko et al. 2000; Kuo & Keegan 2004).

The peripherally acting alpha-2-adrenergic antagonist MK-467 (also termed L-659'066) selectively blocks the alpha-2-adrenoceptors outside the blood-brain barrier (Clineschmidt et al. 1988). It has been shown to attenuate the peripherally mediated cardiovascular effects of medetomidine (Enouri et al. 2008) and dexmedetomidine (Pagel et al. 1998; Honkavaara et al. 2011) in dogs, and also in sheep (Bryant et al. 1998; Raekallio et al. 2010). However, the effects of MK-467 on dexmedetomidine-induced sedation in dogs are minimal (Honkavaara et al. 2008; Restitutti et al. 2011).

Hypothermia is a well known problem in sedated and anaesthetized animals. It can occur in animals sedated with medetomidine (Vainio 1989) when no active attempts have been made to maintain the body temperature. However, it has been suggested that the peripheral vasoconstriction and the central redistribution of blood induced by alpha-2-agonists may enhance the maintenance of body temperature by reducing cutaneous heat loss (Sinclair 2003). Therefore, preventing the medetomidine-induced peripheral vasoconstriction by MK-467, although reducing peripheral resistance, may further impair thermoregulation.

Thermography is a non-invasive and safe method of detecting and visualizing changes in superficial temperature in animals (Schweinitz von 1999; Turner 2001; Kízková & Kunc 2007; Levet et al. 2009). The temperature of an area of the body is a product of cell metabolism and local blood flow (Head & Dyson 2001). If the superficial blood flow is enhanced, the skin temperature increases due to warmer blood coming to the surface from larger vessels. Whilst the human hand and fingers are sensitive enough to detect only a  $\geq 2$  °C difference in temperature on the animal's skin (Holmes et al.

2003), modern infrared cameras are 10 times more sensitive. Thus thermography will detect changes in peripheral blood flow from the resulting changes in heat loss (Varjú et al. 2004; Vianna & Carriave 2005; Stewart et al. 2007).

The aim of this study was to record superficial and rectal (for core) temperature changes in dogs during sedation with medetomidine and butorphanol with and without MK-467. The superficial temperature was recorded with a thermal camera as an indicator of potential peripheral heat loss in dogs.

## Materials and methods

### Animals

Eight healthy purpose-bred neutered Beagle dogs (two females and six males) were used for this study. The dogs weighed (mean  $\pm$  SD)  $14.5 \pm 1.6$  kg, and were aged between 3 and 4 years old. They were considered healthy based on a thorough clinical examination, complete blood count and serum chemistry.

The study protocol was approved by the National Animal Experimentation Board of Finland (ESAVI-2010-07734/Ym-23) and the Ethical Committee of the Viikki Campus at the University of Helsinki.

### Study design

All dogs received four different treatments in a randomized crossover design, with a minimum of 14 days' washout period between treatments. The drugs used in the treatments were medetomidine (Dorbene 1 mg mL<sup>-1</sup>, laboratories Syva S.A., León, Spain), butorphanol (Butordol 10 mg mL<sup>-1</sup>, Intervet International B.V., Netherlands) and MK-467 (Merck, Sharpe & Dohme, PA, USA). 10 mg of MK-467 was solubilised into 1 mL of saline.

The treatments consisted of the following combinations: medetomidine 20 µg kg<sup>-1</sup> and butorphanol 0.1 mg kg<sup>-1</sup> (MB) with or without MK-467 500 µg kg<sup>-1</sup> (MK). Both combinations (MB and MB+MK) were given IV and IM as separate treatments. All drugs were mixed in the same syringe prior to administration. In treatments without MK, saline was added to the syringe in order to keep the administered volume constant. A catheter was inserted in the cephalic vein (for each dog) to enable medications. After catheterization, the dogs were allowed to rest in the room for a minimum of 1 hour.

The dogs were then placed in lateral recumbency on a mattress made of insulating material, without

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