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Do Heat and Moisture Exchangers in the anaesthesia breathing circuit preserve body temperature in dogs undergoing anaesthesia for MRI?

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Abstract:

**Objective:** To investigate whether use of a Heat and Moisture Exchanger (HME) preserve body temperature in dogs weighing < 10kg anaesthetised for MRI.

Study design: prospective, randomised, clinical trial.

**Animals:** Thirty one client owned dogs (17 females, 14 males), 8 months-11 years old, weighing between 2.5 kg and 10 kg.

**Methods:** Dogs were randomly assigned to a treatment group (HME (n= 16) or no HME (n=15)). Dogs were pseudorandomized according to the premedication they received, either dexmedetomidine or no dexmedetomidine. Induction agents were not standardised. General anaesthesia was maintained with isoflurane vaporised in 100% oxygen delivered using a T-piece and a fresh gas flow of 600 ml kg<sup>-1</sup> minute<sup>-1</sup>. Rectal temperature was measured before premedication (T1), after induction (T2), before moving to the MRI unit (T3) and at the end of the MRI scan (T4). Ambient temperatures were measured in the induction room, outside and inside the MRI unit. Data were analysed using a General Linear Model (GLM) with T4, as the outcome variable. Linear correlations were performed between T1, T2, T3 and T4 and variables that predicted T4 were investigated.

**Results:** Gender, age or body mass were not significantly different between groups. There were no significant differences in rectal temperature between groups at any time point (HME<sub>end MRI</sub>=  $36.26 \pm 1.05$  °C, No HME<sub>end MRI</sub>=  $36.24 \pm 1.41$  °C) but dogs receiving dexmedetomidine (DEX<sub>end MRI</sub> =  $36.6 \pm 0.7$  °C) had a higher rectal temperature compared to dogs that did not receive dexmedetomidine (No DEX<sub>end MRI</sub> =  $35.9 \pm 1.6$  °C) for premedication. Rectal temperature varied directly with ambient temperature in MRI scanning room and inversely with anaesthetic duration.

**Conclusions and Clinical Relevance:** Using a HME did not alter body temperature in dogs weighing less than 10 kg undergoing an MRI but including dexmedetomidine in the premedication regimen can preserve the body temperature during anaesthesia.

**Keywords:** MRI, Heat and Moisture Exchanger, hypothermia, dexmedetomidine, dogs

## **Introduction:**

Post-anaesthetic hypothermia is very common in small animals (Redondo et al. 2012a; Redondo et al. 2012b). Recent retrospective studies estimate the frequency of hypothermia to be 97.4 % and 83.6 %, respectively, for cats and dogs when hypothermia is defined as  $T^{\circ} <$ 38.5 °C (Redondo et al. 2012a; Redondo et al. 2012b). However normothermia for dogs and cats has also been previously described as 37.8°C-39.2 °C (Armstrong et al. 2005) so this prevalence of hypothermia might be an overestimation. However there appears to be no overall agreement in the literature about the definition of hypothermia in small animals.

Hypothermia has a myriad of effects on body systems. For example it can affect the cardiovascular system to cause bradycardia, arrhythmias and hypotension; the respiratory system to cause hypoventilation, apnoea and hypoxaemia; the metabolic and endocrine systems to decrease metabolic rate, and reduce the efficacy of the coagulation cascade, and the immune system to increase the risk of wound infection (Armstrong et al. 2005). Many studies in small animals (Cabell et al. 1997; Machon et al. 1999; Kibanda & Gurney 2012; Clark-Price et al. 2013) have shown that active warming techniques such as heat mats and forced warm-air blankets are effective at preventing or treating perioperative hypothermia. However, preservation of body temperature during anaesthesia in patients undergoing MRI is problematic. MRI scanners use magnetic fields and radio waves to form images of the body. Therefore all electrical devices used in MRI scanners have to be non-magnetic and electrically non-conductive. To the authors' knowledge, none of the currently available active warming devices are MRI compatible. Maintenance of normothermia is one of the key goals of supportive practices during anaesthesia. The only option to support body temperature when animals are undergoing an MRI scan is to use passive warming devices that are compatible with MRI technology. Another feature of MRI rooms is their low temperature, which is mandatory to keep the magnet cool.

Heat and Moisture Exchangers (HME) are disposable devices placed between the patient and the breathing system. The exchanging medium is composed of foam, paper, or a substance that acts as a condensation and absorption surface. This medium is enclosed in plastic housing. There is also commonly a port built into the plastic housing to allow a gas sampling line for a respiratory gas monitor to be attached. The aim of the device is to conserve exhaled water and heat and return it to the patient in the inspired gas (Dorsch & Dorsch 2014). There are no clinical data in the literature regarding the efficacy of HMEs in preservation of body temperature when used alone (i.e. without external warming devices such as heat mats) in small animals < 10 kg.

Perioperative hypothermia develops in 3 phases (Armstrong et al. 2005). The first phase, which happens during the first hour of anaesthesia, is a rapid decrease in body temperature due to redistribution of heat from the core to the peripheral compartment. This redistribution is promoted by two mechanisms: the reduction in threshold temperature required to cause reflex vasoconstriction and by the direct vasodilation induced by anaesthetic agents. The second phase is characterised by a slow decrease in body temperature due to an imbalance between heat loss (evaporation, conduction, convection and radiation) and heat production. During the third phase, body temperature stabilises because of equal heat loss and heat production. As Sinclair (2003) suggested, it is believed that anaesthetic drugs with vasoconstrictor properties might decrease the heat loss that happens during the first phase. Vainionpää et al. (2013) confirmed this hypothesis in an experimental setting in medetomidine sedated dogs.

The primary aim of this study was to investigate the efficacy of HMEs to maintain normothermia in dogs < 10 kg bodymass undergoing anaesthesia for MRI. We hypothesised that dogs receiving an HME would maintain a higher rectal body temperature than dogs that did not receive an HME in the anaesthetic breathing system.

A secondary aim was to test the hypothesis that anaesthetised dogs receiving dexmedetomidine for premedication would have a smaller decrease in rectal temperature than dogs that did not receive dexmedetomidine as part of their premedication protocol.

# Materials and methods

# Dogs

A prospective, pseudorandomised, clinical trial on 31 client-owned dogs weighing less than 10 kg and requiring general anaesthesia (GA) for an MRI scan was conducted. The American Society of Anesthesiologists (ASA) physical status grade was also recorded but was not part of the inclusion criteria. This study was approved by the University of \*\*\*\* ethical committee (VIN/13/028). Study-specific owner consent was not considered necessary by the institution's ethics committee because anaesthetists were free to choose between routinely used sedative and anaesthetic medications and the consent to participate in the study was covered by the clinic's general client consent form. The inclusion criteria for the study were that animals weighed between 2.5 kg and 10 kg, had intravenous access established with an intravenous catheter before premedication, and tolerated rectal measurement of body temperature. Dogs with hypothermia (< 37.8°C) or hyperthermia (> 39.2°C) before premedication were excluded from the study.

# Procedures:

Due to the clinical nature of the study, it was problematic to standardise the anaesthesia protocol for all dogs that entered the study, therefore the anaesthesia protocol was matched between cases receiving an HME (figure 1) (HME group) and the control group (no HME). Hence true randomisation of cases recruited to the study was not possible, but pseudorandomisation ensured that there was the same number of cases receiving

dexmedetomidine for premedication in the group with the HME and the group without the HME. Thus two randomisation charts were used, one for dogs premedicated with dexmedetomidine and the other for dogs not premedicated with dexmedetomidine. Following a complete physical examination by a clinical anaesthetist, dogs were randomly allocated to group (HME or No HME) according to the premedication used (dexmedetomidine or not). Dose and choice of the premedication and induction agent were at the discretion of the clinician in charge of the case. All dogs received an opioid, which was most commonly butorphanol. Dexmedetomidine dose, in those patients that received it, ranged between 1 and 10 µg kg<sup>-1</sup>. Anaesthesia was induced in all dogs with either propofol or alfaxalone administered to effect to allow orotracheal intubation. Anaesthesia was maintained with isoflurane (IsoFlo; Abbott, UK) in 100 % oxygen, and the isoflurane concentration was adjusted to achieve an adequate depth. All dogs were connected to a T-piece non-rebreathing system with a fresh gas flow of 600 ml kg<sup>-1</sup> min<sup>-1</sup>. All patients received 5 ml kg<sup>-1</sup> hour<sup>-1</sup> Hartmann's solution (Vetivex 11, Dechra Veterinary Products Ltd, Shrewsbury, UK). In case of hypotension (defined as mean arterial pressure (MAP) < 60 mmHg) or evidence of hypovolaemia (heart rate (HR) > 150 beats per minute), a fluid bolus was administered and the volume recorded.

Rectal temperature was recorded at a number of time points before and during anaesthesia (figure 2): before premedication (T1), immediately after induction of anaesthesia (T2), before the dog entered the bore of the MRI magnet (T3), and just after leaving the bore of the MRI magnet (T4) using a single, rapid response rectal thermometer (WelchAllyn SureTemp plus; WelshAllyn, Republic of Ireland).

The ambient temperature in the anaesthesia induction area, ambient outdoor environmental temperature (animals had to be transported outdoors to enter the MRI unit) and the

temperature in the MRI unit (both before and after the MRI scan) were recorded. Figure 2 summarises each important time point of the temperature recording.

All animals were wrapped in a single, standardised fleece blanket (Pet blanket fleece; DWDUK) following premedication until the end of the MRI scan. Length of anaesthesia (between induction and end of MRI) was recorded. The study ended at the end of the MRI scan.

#### Statistical analysis:

A sample size calculation indicated that a total of 64 animals (32 per arm) were required to have an 80 % power at  $\alpha = 0.05$  to detect a difference of 1 °C in rectal temperature, the minimum temperature difference considered to be clinically relevant. Unfortunately, midway through the study MRI unit was changed preventing collection of this number of cases. However data collected at this time point were analysed. Confidence intervals indicated that the current sample size was sufficient to show a difference in rectal temperature of less than 1 °C between groups.

A General Linear Model (GLM) analysis, which combined a variance analysis and a covariance analysis adjusting for continuous variables, was carried out. The normality of the residual error and the homogeneity of the variance were checked to ensure that the modelling approach was appropriate. The use of HME and dexmedetomidine were entered into the model as categorical variables, each with two levels. The initial temperature of the dog (T1), the outdoor temperature, the duration of the MRI, the mean MRI unit temperature and the body weight were tested as covariates within the model. All analyses were performed using SPSS for Windows (IBM SPSS statistic version 21).

A Mann-Whitney U test was used to check randomisation of the baseline variables (ASA grade, gender, the region of the MRI scan, induction agent used and the body condition score

(BCS)) and a t-test to check the continuous baseline variables (body mass and age). *P*-values  $\leq 0.05$  were considered statistically significant.

#### Results:

A total of 31 dogs, weighing (mean  $\pm$  SD) 6.2  $\pm$  2.2 kg were recruited to this study. The ages of dogs were relatively uniformly distributed from 8 to 132 months with a median age of 60 months. Respectively, 16 and 15 dogs were included in the HME and No HME groups. Eight dogs in each group (HME and No HME) received dexmedetomidine as part of their premedication protocol. Each group comprised a wide distribution of breeds that precluded inclusion of breed in the statistical analysis. The breeds represented within each group are shown in Table 1. No significant differences were found between the age, body mass, BCS, ASA status and MRI scan region when dogs in the HME and No HME groups were compared. Dogs that received dexmedetomidine had significantly lower ASA status (i.e. were more likely to be systemically healthy) than dogs that were premedicated with other sedative agents (p = 0.02).

The patients' characteristics in each group are summarized in Table 1.

Table 2 presents the results of the GLM used to analyse factors affecting rectal temperature at the end of anaesthesia in dogs receiving an HME or no HME throughout anaesthesia. It shows that the use of an HME did not affect the rectal temperature at the end of the MRI scan (T4). Dogs that were not premedicated with dexmedetomidine had a significantly greater decrease in rectal temperature between T1 and T4. Body mass and MRI ambient temperature had a significant positive correlation with rectal temperature. Increasing the body mass by 1 kg, or the temperature in the MRI by 1°C, was associated with an increase in the rectal temperature at the end of the MRI by 0.3 °C.

The statistical modelling process also produced a predictive mathematical equation which may be used to construct an estimated mean of the rectal temperature at the end of the MRI for any combination of treatment or study variables (within the ranges measured within the study). For example, if a dog was not premedicated with dexmedetomidine and did not receive an HME, rectal temperature at the end of the MRI scan would be equal to the intercept added to the product of B and each variable no HME, no dexmedetomidine, duration of MRI scan in minutes, ambient temperature (both MRI unit and outdoor), body temperature, body mass. B is the estimation of the effect of the parameter. For this dog the equation would be (table 2):

$$T_{end} = 25.576 - 0.395 - 0.716 + (T_{before} \times 0.137) + (T_{MRI} \times 0.286) - (duration MRI \times 0.024) - (T_{outdoor} \times 0.073).$$

There were no significant differences in ambient temperatures recorded in the induction room, outdoors, and in the MRI unit at the start and the end of the MRI scan, between each group.

Figure 3 presents the mean ( $\pm$ SD) rectal temperature before premedication, after induction of anaesthesia, before entering the MRI unit and at the end of the MRI scan for each group. The lowest and the highest rectal temperatures recorded at the end of the MRI scan were 32.4° C and 37.7° C, respectively. Rectal temperature before premedication was significantly higher in dogs receiving dexmedetomidine for premedication. All dogs showed a significant decrease in rectal temperature when the rectal temperature before premedication and at the end of MRI were compared (p <0.001). Most of the dogs in each group, irrespective of premedication choice, had a rectal temperature at the end of the MRI scan below 37° C.

### **Discussion:**

Perioperative hypothermia is a common complication in small animal anaesthesia (Redondo et al. 2012a; Redondo et al. 2012b). Efficient warming devices all need an electricity source and are therefore not compatible with an MRI scanner. Therefore the first aim of our study was to evaluate a passive warming device compatible with MRI technology, the HME, in dogs weighing less than 10 kg anaesthetised for an MRI scan. Sinclair (2003) suggested that use of an alpha2-agonist for premedication could decrease heat loss by redistribution. This hypothesis was confirmed by Vainionpää et al. (2013) in experimental beagle dogs sedated with medetomidine. Therefore our secondary aim was to confirm this hypothesis in a clinical setting in anaesthetised dogs.

All dogs recruited to the study were scheduled to undergo anaesthesia for an MRI scan of the brain or spinal cord. As such, although all dogs weighed less than 10 kg, a wide variety of breeds and ages of dogs of both gender were recruited. Our population was therefore heterogeneous, which made it a good representative of the wider clinical situation. All dogs had a statistically significant reduction in rectal temperature at the end of the MRI scan when compared with rectal temperature before premedication. The use of an HME did not significantly alter rectal temperature at the end of anaesthesia. However, dogs that received dexmedetomidine for premedication had a higher rectal temperature at the end of anaesthesia than dogs that did not receive dexmedetomidine, independent of use of an HME or not.

To the authors' knowledge, only one study regarding the use of HMEs in dogs has been published in the veterinary literature (Hofmeister et al. 2011). This study also found that the inclusion of an HME in the breathing system did not facilitate maintenance of rectal temperature. However, Hofmeister et al. (2011), studied a patient population (dogs weighing at least 15 kg undergoing single-limb orthopaedic procedures) different to the present study. Additional heating methods were also used to warm the dogs. In the human field, HMEs are widely used to maintain the humidity of the airway and to decrease the risk of pneumonia

during long term mechanical ventilation in the ICU or during a prolonged general anaesthesia. Only a few studies have investigated the effect of an HME on body temperature of human patients. Haslam and Nielsen (1986) studied the effect of an HME on the body temperature of men undergoing surgery lasting more than 2 hours. They found that patients receiving an HME maintained a higher body temperature. This finding was not affected by the fresh gas flow used (high or low). On the other hand, a study by Goldberg et al. (1988) in which patients were anaesthetised for more than 30 minutes but less than 90 minutes did not find any beneficial effect of HME on body temperature. HMEs are designed to reduce the heat loss by evaporation. Heat loss during general anaesthesia is divided in three phases (Armstrong et al. 2005). The first one occurs within the first hour of anaesthesia and is caused by the redistribution of heat from the core to the peripheral compartment. The second phase, which happens within the next two hours is due to heat loss from radiation, conduction, convection and evaporation, which exceeds the metabolic heat production. Therefore, the use of an HME to decrease heat loss by evaporation might be more evident during this phase. This could explain the different findings of the Goldberg and Haslam studies. The dogs in our study, were anaesthetised for up to 70 minutes, which corresponds to the end of the first phase or the start of the second phase. It is possible therefore that the effect of an HME to decrease heat loss by evaporation would be more apparent in patients anaesthetised for at least 2 hours. Also, many factors affect the efficacy of the HME; whether they are hydrophobic or hygroscopic (Dorsch & Dorsch 2014), the initial humidity of the gas, the fresh gas flow and the existence of a leak in the breathing system (Gedeon et al. 1987). In the human literature it is well described that hygroscopic HMEs have better heat conserving and exchanging properties than the hydrophobic HMEs used in our study (Eckerborn & Lindholm 1990; Sottiaux et al. 1993; Martin et al. 1994). Also the higher the fresh gas flow, the less efficient the HME is in conserving heat and moisture (Eckerborn & Lindholm 1990).

As our patients were under 10 kg, we used a non-rebreathing breathing system necessitating a high fresh gas flow.

An important finding in this study was that premedication with dexmedetomidine prevented, to some extent, a fall in rectal temperature during MRI. This has not been previously shown in a clinical setting in anaesthetised animals and may be another clinical benefit of the use of dexmedetomidine in very small patients which are known to be more prone to hypothermia because of their large surface area to mass ratio. The use of alpha-2-adrenoceptor agonist drugs is well described to cause a decrease in body temperature in human beings (Doufas et al. 2003) as well as in small animals (Ponder & Clark 1980; Livingston et al. 1984; Ansah et al. 1998; Vainionpää et al. 2013). It is believed that this decrease in body temperature is due to the depression of the central nervous system (CNS) associated with a decreased in muscular activity (MacDonald et al. 1988; Virtanen 1988). However, it was also suggested that the peripheral vasoconstriction produced by the administration of medetomidine might decrease the heat loss by redistribution (Sinclair 2003). This has been recently confirmed by Vainionpää et al. (2013) in experimental beagle dogs receiving a combination of medetomidine and butorphanol or medetomidine, butorphanol and MK-467. The latter group showed a lower rectal temperature. As MK-467 is a peripherally acting alpha-2-adrenergic antagonist, it was believed that this difference in body temperature might be because of a higher peripheral heat loss. Although dogs in our study treated with dexmedetomidine also had a higher baseline temperature before the administration of dexmedetomidine, the statistical model did not show any influence of the baseline temperature on the body temperature at the end of the MRI scan. It is unknown why the baseline temperature was higher in the dogs receiving dexmedetomidine, but it could be hypothesized that in some cases anaesthetists may have chosen dexmedetomidine due to time constraints or patient temperament. For example, where a dog is presented the day prior to carrying out an MRI

scan, it may be less likely to receive dexmedetomidine than a dog who is scheduled within a shorter period of time. Dogs undergoing MRI scanning on the same day as presentation may be held in the anaesthesia induction area pending pre-medication rather than be admitted to the frequently cooler ward.

Dogs that received dexmedetomidine had a lower ASA grade probably because of the cardiovascular side effects of this class of drug (Murrell & Hellebrekers 2005). In a retrospective study (Redondo et al. 2012b), ASA III-IV dogs had a lower rectal temperature at the end of anaesthesia than ASA I dogs. They speculated that animals with a higher ASA grade are less likely to have an intact thermoregulatory mechanisms. This could be another confounding factor in the study.

The analysis used in the present study also highlighted the effect of the duration of the MRI scan and the ambient temperature in the MRI unit on rectal temperature at the end of the MRI scan. The longer the MRI scan or the lower the ambient temperature, the greater the fall in rectal temperature. This has also been show previously in dogs (Redondo et al. 2012b) and cats (Redondo et al. 2012a). Interestingly, our model failed to detect an effect of rectal temperature before premedication on subsequent rectal temperature.

There are a number of limitations to our study which could have confounded the results. The duration of the MRI scan was not controlled between dogs, therefore it was included in the statistical model as a factor that may affect rectal temperature. The ambient environmental temperature and the temperature in the MRI scanner was also not controlled between animals and this would have increased the variability of data in this clinical study. Although the effect of dexmedetomidine was investigated in the statistical model other premedication or induction drugs were not standardised between animals. It is possible that some drugs cause more depression of the CNS control of body temperature than others. For example some dogs

received acepromazine which causes vasodilation and could potentially worsen the heat loss. However, because every dog in the study received isoflurane, which also causes vasodilation, we hypothesized that the administration of acepromazine to some dogs would not affect our results. Similarly, opioids are also implicated in decrease in body temperature (Clark 1979). However, because all dogs received opioids as part of their premedication protocol, it is unlikely to have confounded the results of this study. On the other hand, this heterogeneity is a strength of the study as it is a good representation of the clinical situation.

In conclusion, in the present study, the use of an HME did not preserve the rectal temperature in anaesthetised dogs weighing less than 10 kg undergoing MRI. Also, premedication with dexmedetomidine was associated with a better maintenance of the rectal temperature compared with dogs that received other drugs for premedication. This may be considered when making decisions about which premedication agent to use in patients of low body mass.

### References

- Ansah OB, Raekallio M, Vainio O (1998) Comparison of three doses of dexmedetomidine with medetomidine in cats following intramuscular administration. J Vet Pharmacol Ther 21, 380-387.
- Armstrong SR, Roberts BK, Aronsohn M (2005) Perioperative hypothermia. J Vet Emerg Crit Car 15, 32-37.
- Cabell LW, Perkowski SZ, Gregor T et al. (1997) The effects of active peripheral skin warming on perioperative hypothermia in dogs. Vet Surg 26, 79-85.
- Clark-Price SC, Dossin O, Jones KR et al. (2013) Comparison of three different methods to prevent heat loss in healthy dogs undergoing 90 minutes of general anesthesia. Vet Anaesth Analg 40, 280-284.
- Clark WG (1979) Influence of opioids on central thermoregulatory mechanisms. Pharmacol Biochem Behav 10, 609-613.
- Dorsch JA, Dorsch SE (2014) Understanding Anesthesia equipment. (5th Edition edn), Wolters Kluwer, New Delhi (India), 298-300.
- Doufas AG, Lin CM, Suleman MI et al. (2003) Dexmedetomidine and meperidine additively reduce the shivering threshold in humans. Stroke 34, 1218-1223.
- Eckerbom B, Lindholm CE (1990) Heat and moisture exchangers and the body temperature: a peroperative study. Acta Anaesthesiol Scand 34, 538-542.
- Gedeon A, Mebius C, Palmer K (1987) Neonatal hygroscopic condenser humidifier. Crit Care Med 15, 51-54.
- Goldberg ME, Jan R, Gregg CE et al. (1988) The heat and moisture exchanger does not preserve body temperature or reduce recovery time in outpatients undergoing surgery and anesthesia. Anesthesiology 68, 122-123.

- Haslam KR, Nielsen CH (1986) Do passive heat and moisture exchangers keep the patient warm? Anesthesiology 64, 379-381.
- Hofmeister EH, Brainard BM, Braun C et al. (2011) Effect of a heat and moisture exchanger on heat loss in isoflurane-anesthetized dogs undergoing single-limb orthopedic procedures. J Am Vet Med Assoc 239, 1561-1565.
- Kibanda JO, Gurney M (2012) Comparison of two methods for the management of intraoperative hypothermia in dogs. Vet Rec 170, 392.
- Livingston A, Low J, Morris B (1984) Effects of clonidine and xylazine on body temperature in the rat. Br J Pharmacol 81, 189-193.
- MacDonald E, Scheinin H, Scheinin M (1988) Behavioural and neurochemical effects of medetomidine, a novel veterinary sedative. Eur J Pharmacol 158, 119-127.
- Machon RG, Raffe MR, Robinson EP (1999) Warming with a forced air warming blanket minimizes anesthetic-induced hypothermia in cats. Vet Surg 28, 301-310.
- Martin C, Papazian L, Perrin G et al. (1994) Preservation of humidity and heat of respiratory gases in patients with a minute ventilation greater than 10 L/min. Crit Care Med 22, 1871-1876.
- Murrell JC, Hellebrekers LJ (2005) Medetomidine and dexmedetomidine: a review of cardiovascular effects and antinociceptive properties in the dog. Vet Anaesth Analg 32, 117-127.
- Ponder SW, Clark WG (1980) Prolonged depression of thermoregulation after xylazine administration to cats. J Vet PharmacolTher 3, 203-207.
- Redondo JI, Suesta P, Gil L et al. (2012a) Retrospective study of the prevalence of postanaesthetic hypothermia in cats. Vet Rec 170, 206.
- Redondo JI, Suesta P, Serra I et al. (2012b) Retrospective study of the prevalence of postanaesthetic hypothermia in dogs. Vet Rec 171, 374.
- Sinclair MD (2003) A review of the physiological effects of alpha2-agonists related to the clinical use of medetomidine in small animal practice. Can Vet J 44, 885-897.
- Sottiaux T, Mignolet G, Damas P et al. (1993) Comparative evaluation of three heat and moisture exchangers during short-term postoperative mechanical ventilation. Chest 104, 220-224.
- Vainionpää M, Salla K, Restitutti F et al. (2013) Thermographic imaging of superficial temperature in dogs sedated with medetomidine and butorphanol with and without MK-467 (L-659'066). Vet Anaesth Analg 40, 142-148.
- Virtanen R (1988) Pharmacological profiles of medetomidine and its antagonist, atipamezole. Acta vet Scand Suppl 85, 29-37.

Figure 1.

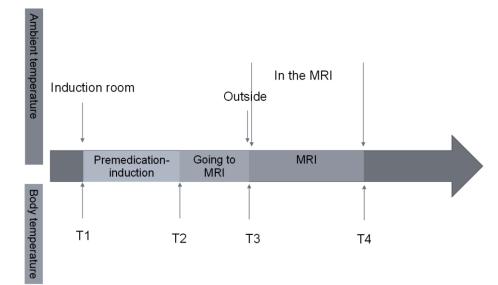


Figure 2 : Mean ( $\pm$  SE) ambient temperature in degrees Celsius at different time points for dogs receiving no HME and no dexmedetomidine, no HME and dexmedetomidine, HME and no dexmedetomidine; HME and dexmedetomidine:

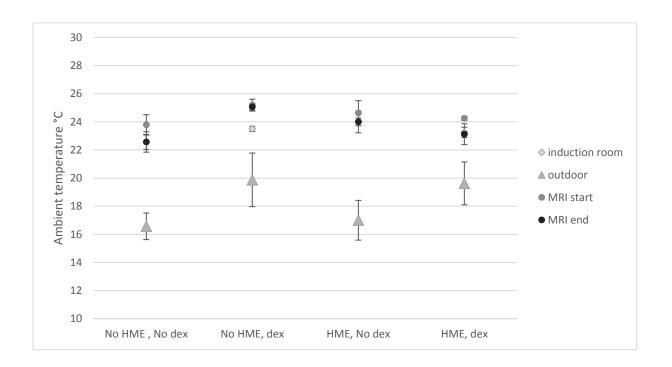


Figure 3: Mean ( $\pm$  SE) ambient temperature in degrees Celsius at different time points for dogs receiving no HME and no dexmedetomidine, no HME and dexmedetomidine, HME and no dexmedetomidine.

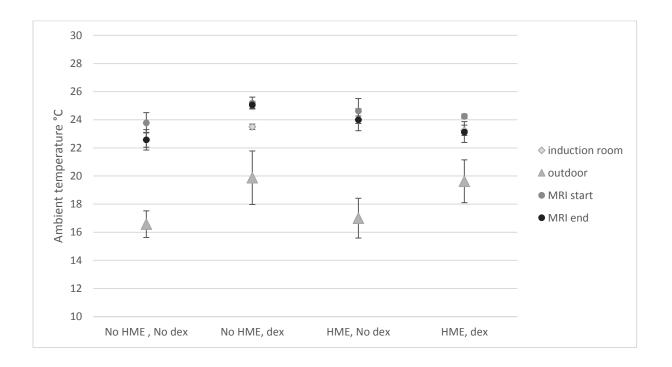


Table 1: Patient characteristics (age (median [range]); body mass (mean  $\pm$  SD); BCS (median [range]); reproductive status; breed; ASA grade), anaesthetic protocol, and reason for and the duration of the MRI scan in minutes (mean  $\pm$  SD):

Variable	No HME (n = 15)		HME (n = 16)		
	No dex $(n = 7)$	Dex (n = 8)	No dex ( n = 8)	Dex ( n = 8 )	
Age (months)	53 [24-132]	66.5 [27-90]	73.5 [22-132]	53 [8-120]	
Bodymass (kg)	5 ± 3	7 ± 1	$6 \pm 2$	$5 \pm 2$	
BCS [1-9]	5 [4-6]	5 [3-7]	5 [3-6]	5 [2-7]	
Gender					
male	2	3	3	5	
female	5	4	5	3	
Breed					
Yorkshire terrier	1	0	1	1	
Chihuahua	2	0	0	1	
Jack Russell terrier	1	2	1	1	
Border terrier	2	0	1	0	
Shi tzu	1	2	1	0	
Cavalier King Charle	S				
spaniel	0	1	2	1	
Patterdale terrier	0	1	0	0	
Maltese terrier	0	1	0	2	
Cavapoo	0	1	0	0	
Dachshund	0	0	2	1	
Cocker spaniel	0	0	0	1	
ASA grade					
I	0	1	0	0	

II	5	7	6	8	
III	2	0	2	0	
IV	0	0	0	0	
V	0	0	0	0	
Premedication					
Acepromazine					
(0.02-0.03 mg kg <sup>-1</sup> )					
(ACP injection ;Novartis,					
UK)	4	0	2	0	
Butorphanol					
(0.2-0.4 mg kg <sup>-1</sup> )					
(Alvegesic vet; Dechra,					
UK)	2	6	6	5	
Methadone					
(0.2-0.4 mg kg <sup>-1</sup> )					
Comfortan; Dechra, UK)	5	2	2	2	
Buprenorphine (0.02mg					
kg <sup>-1</sup> )					
(Buprecare; Animalcare,					
UK)	0	0	0	1	
Midazolam					
(0.3-0.4 mg kg <sup>-1</sup> )					
(Hypnovel; Roche, UK)	0	0	3	0	
Dexmedetomidine					
(2-5.5 µg kg <sup>-1</sup> )					
(Dexdomitor 0.1; Orion					
Pharma, UK)	0	8	0	8	
Induction					
Propofol	5	5	5	6	

(1.5-5.5 mg kg <sup>-1</sup> )				
(PropofloPlus; Abbott,				
UK)				
Alfaxalone				
(0.9-3 mg kg <sup>-1</sup> )				
(Alfaxan; Jurox, UK)	2	3	3	2
MRI region				
Spine	4	5	4	4
Brain	3	3	4	4
Duration (min)	$52 \pm 30$	$65 \pm 14$	$63 \pm 28$	$70 \pm 34$

SD = standard deviation; BCS = Body condition score; ASA grade = American Society of Anesthesiologist

physical status classification

Table 2: The parameter estimates from the fitted general linear model are shown together with their significance and a 95 % confidence interval: use of HME, or not, and use of Dexmedetomidine or not, were entered as categorical variables, each with two levels. The MRI temperature, body mass, the initial rectal temperature of the dog (T1), the outdoor temperature, and the duration of the MRI were entered as covariates within the model. The parameter estimates can be used to construct an equation which summarises the mean (predicted) temperature for different combination/levels of the predictive variables (an example is given within the results section).

Parameter	В	Std.Error	t	p-value	95 % Confidence Interval	
					Lower	Upper
					bound	bound
Intercept	25.576	13.743				
No HME	- 0.395	0.287	- 1.380	0.181	- 0.988	0.197
No	- 0.716	0.342	- 2.093	0.048	-1.424	- 0.008
dexmedetomidine						
MRI temperature	0.286	0.079	3.633	0.001	0.123	0.448
Outdoor	- 0.073	0.037	- 1.951	0.063	- 0.150	0.004
temperature						
MRI duration	-0.024	0.006	- 4.219	< 0.001	- 0.035	- 0.012
Body mass	0.314	0.073	4.294	< 0.001	0.163	0.466
Temperature	0.137	0.353	0.388	0.702	- 0.594	0.868
before						
premedication						

B = estimate of the effect size, thus as "No HME" is -0.395 this means that "No HME" is 0.194°C less than HME. When the variable is continuous, for example, MRI temperature, then B is the size of the effect for a one unit change. So for MRI temperature where B = 0.286 this means that for every 1°C increase in MRI temperature there is a 0.286 °C increase in rectal temperature at the end of the MRI scan.