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Changes in tracheal, oesophageal and rectal temperature over 60 minutes anaesthesia in non-heated dogs, spontaneously breathing or mechanically ventilated

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Abstract: The objective of this prospective randomised clinical study was to determine the differences in the tracheal, oesophageal and rectal temperature in spontaneously breathing or mechanically ventilated dogs. A total of thirty dogs were allocated to the SPO-group breathing spontaneously (n = 15) or the MEC-group ventilated mechanically (n = 15). Anaesthesia was established using a medetomidine-butorphanol-propofol-isoflurane combination. The tracheal (T-Tra), oesophageal (T-Oes), rectal (T-Rec), inspired gas (T-Gas), room (T-Room) temperatures, respiratory frequency (f_R), heart rate (HR), mean arterial pressure (MAP) and end-tidal carbon dioxide concentration ($ETCO_2$) were measured after connecting to a re-breathing system (baseline) and subsequently in 10-minute intervals for 60 minutes. The data were analysed using ANOVA and Steel-Dwass tests ($P < 0.05$). In the SPO-group, the T-Tra, was significantly lower at T30, T40, T50, T60, the T-Oes and T-Rec at T40, T50, T60, compared to the baseline. In the MEC-group, the T-Tra and T-Oes was significantly lower at T30, T40, T50, T60, the T-Rec at T40, T50, T60, compared to the baseline. In the SPO-group, the f_R was significantly lower for all the times and the $ETCO_2$ higher at T10, T20, T30, T40, T50 compared to the MEC-group. No other differences were detected. During anaesthesia, there is a comparable decrease in body temperatures, regardless of whether the dogs are breathing spontaneously or ventilated mechanically.

Keywords: anaesthesia; artificial ventilation; hypothermia; medetomidine

Hypothermia is the most common peri-anaesthetic complication. It occurs in almost 83% of dogs undergoing general anaesthesia (Redondo et al. 2012). In human medicine, hypothermia relates to a higher risk of a postoperative infection, higher blood losses during surgery, a higher rate of cardiovascular complications and a prolonged recovery time (Brandt et al. 2010). The canine physiological temperature range is 37.8–39.2 °C (Armstrong et al. 2005). Hypothermia occurs when the heat loss exceeds the heat production. Primary hypothermia is caused during normal thermal production with exposure to cold, secondary hypothermia can be induced by anaesthetics, systemic diseases, or injuries (Stepaniuk and Brock 2008). Therefore, secondary hypothermia can be found in a relatively warm environment. Hypothermia is caused due to radiation, convection, conduction and evaporation. The

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induction of general anaesthesia causes the redistribution of heat from the core to the periphery. Body heat is lost with the delivery of fresh anaesthetic gases, mainly due to convection and evaporation (Stepaniuk and Brock 2008; Grimm 2015). Yuksek et al. (2019) found that lung protective ventilation in humans using positive end expiratory pressure has no advantage in preserving a patient's temperature compared to conventional ventilation. It has been described that the use of heat and moisture exchangers alone cannot prevent development of hypothermia (Hofmeister et al. 2011). However, currently, the influence of mechanical ventilation on the body temperature in dogs is not well described.

Oesophageal and rectal temperatures are considered as the standard for the determination of the body core temperature (Schauvlijege 2016). The trachea is also described as a reliable site for the body core temperature measurement (Hayes et al. 1996).

The aim of this prospective study was to investigate the 60-minute effects of mechanical ventilation on selected body temperatures in healthy, non-heated, adult dogs. It was hypothesised that the mechanical ventilation would cause a more significant decrease in the body temperature compared to spontaneous breathing due to the increased respiratory activity.

MATERIAL AND METHODS

None of the procedures exceeded the commonly used clinical measurements and were performed in accordance with the current law for animal protection and the Ethics committee of the University of Veterinary Sciences Brno. All the animal owners provided consent to have their dog participate in the study. The study was designed as a prospective randomised clinical study.

Animals

A total of thirty client-owned adult dogs scheduled for periodontal treatment were enrolled in this study. All the dogs were clinically healthy, a classified American Society of Anesthesiologists (ASA) physical status I or II, aged 1–10 years and weight 10–30 kg. The dogs were fasted for 12 h before the general anaesthesia with free access to water, and spontaneously defecated before the anaesthesia premedication.

Study protocol

A randomising software (www.randomizer.org) was used to allocate the dogs to one of two groups – the spontaneously breathing group (SPO-group, n = 15) or mechanically ventilated group (MEC-group, n = 15).

In all the dogs, an intravenous cannula was placed into the cephalic vein, then premedicated with medetomidine (Domitor; Orion Pharma, Espoo, Finland) 0.01 mg/kg and butorphanol (Butomidor; Richter Pharma AG, Wels, Austria) 0.2 mg/kg intravenously. The anaesthesia was induced with propofol (Propofol 1% MCT/LCT; Fresenius Kabi, Bad Homburg, Germany) 1–2 mg/kg and maintained with isoflurane (Aerrane; Baxter SA, Lessines, Belgium) in an oxygen-air mixture. A circle rebreathing system was used. The fresh gas flow was set to 50 ml/kg/min, the inspired oxygen fraction was 0.6 and the inspired isoflurane was 1.5%. All the dogs were placed on a dental surgery table in dorsal recumbency using a non-heated V-shape foam positioner. The dogs of the SPO-group were breathing spontaneously, the dogs of the MEC-group were mechanically ventilated during the entire observed period. A volume-controlled time cycling mode (Veterinary Anaesthesia Ventilator Model 3000; Matrx, New York, USA) with a baseline ventilator setting consisting of a peak inspiratory pressure (PIP) set at 12 cm H_2O, a positive end-expiratory pressure (PEEP) at 2 cm H_2O, an inspiratory to expiratory time ratio of 1 : 2 and an inspiratory pause of 33% of the inspiratory time was used. The ventilation frequency was set based on the end-tidal carbon dioxide ($ETCO_2$) level, which was maintained between 35–40 mm Hg. All the dogs received 5 ml/kg/h of a non-heated lactated Ringer solution (Hartmann; B. Braun, Melsungen, Germany).

Measurements

In all the dogs, the tracheal, oesophageal, rectal, room and inspired gas temperatures, respiratory frequency, heart rate, mean arterial pressure and end-tidal carbon dioxide concentration were measured immediately after connection to the breathing system (baseline) and subsequently in 10-minute intervals for 60 minutes (T10–T60).

The tracheal temperature (T-Tra) was measured with a temperature probe placed through the bron-

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choscopy-piece connected between a breathing Y-piece and the dogs' endotracheal tube. The tip of the tracheal temperature probe was inserted to the IV–V intercostal space level. The oesophageal temperature (T-Oes) was measured using a transoesophageal electrocardiograph (ECG) probe with a thermometer. The tip of the oesophageal temperature probe was inserted to the IV–V intercostal space level. The rectal temperature (T-Rec) probe was inserted into the rectum to a depth of 10 cm. The inspired gas temperature (T-Gas) was measured by a temperature probe placed inside the hose between the inspiration valve and inspiration branch of the animal's breathing circuit. The room temperature (T-Room) was measured by a digital thermometer (SWS 1500 B; Sencor, Říčany, Czech Republic) placed at a 50-centimetre distance from the dog's body.

The respiratory frequency (f_R) was monitored using a capnography curve contour analysis. The heart rate (HR) was measured by a 3-lead ECG that was part of the transoesophageal ECG probe. The mean arterial pressure (MAP) was measured using a cuff applied to the left front limb. The cuff's width was 40% of the circumference of the limb. The sensor for the end-tidal carbon dioxide concentration ($ETCO_2$) measurement was attached to the end of the endotracheal tube, the $ETCO_2$ was measured using a main-stream technique. The temperatures, f_R, HR, MAP and $ETCO_2$ were measured using vital-sign monitors (Cardel 9500 HD; Midmark, Versailles, USA).

Statistical analysis

All the data were analysed using KyPlot v2.0 beta 15 (Koichi Yoshioka©). Anderson–Darling and Bartlett's tests were used to confirm the normal distribution of the data and the homogeneity of variance, respectively. The data measured at T10–T60 were compared to the baseline using an analysis of variance (ANOVA) for the repeated measures with a Bonferroni correction. All the variables were compared between groups at each specific time point using the Steel-Dwass test ($P < 0.05$).

RESULTS

Thirty adult dogs, fifteen males and fifteen females, body weight of 21.5 ± 6.3 kg (mean ± standard deviation), aged 5.6 ± 3.4 years were included. No significant differences were noted between the groups with respect to the sex, body weight, age or measured parameters at the baseline.

In the SPO-group, the T-Tra was significantly lower compared to the baseline after 40 min (at T40 $P = 0.001$, at T50 $P < 0.001$, at T60 $P < 0.001$), the T-Oes was significantly lower compared to the baseline after 30 min (at T30 $P = 0.018$, at T40 $P < 0.001$, at T50 $P < 0.001$, at T60 $P < 0.001$), the T-Rec was significantly lower compared to the baseline after 40 min (at T40 $P < 0.001$, at T50 $P < 0.001$, at T60 $P < 0.001$; Table 1). There were no significant differences in the T-Gas and T-Room compared to the baseline.

Table 1. Changes in the tracheal (T-Tra), oesophageal (T-Oes), rectal (T-Rec), inspired gas (T-Gas) and room (T-Room) temperatures (mean ± standard deviation) in dogs breathing spontaneously (SPO-group) or mechanically ventilated (MEC-group)

Group	Parameter	Temperature						
		baseline	T10	T20	T30	T40	T50	T60
SPO-group	T-Tra	37.5 ± 0.5	37.4 ± 0.5	37.3 ± 0.6	37.0 ± 0.5	36.6 ± 0.6*	36.5 ± 0.6*	36.3 ± 0.5*
	T-Oes	38.7 ± 0.5	38.8 ± 0.5	38.5 ± 0.4	38.1 ± 0.5*	37.8 ± 0.5*	37.5 ± 0.6*	37.3 ± 0.6*
	T-Rec	38.5 ± 0.5	38.4 ± 0.4	38.3 ± 0.5	38.0 ± 0.4	37.7 ± 0.6*	37.5 ± 0.6*	37.3 ± 0.6*
	T-Gas	22.4 ± 1.4	22.5 ± 1.3	22.7 ± 1.5	22.8 ± 1.5	22.9 ± 1.5	23.1 ± 1.6	23.1 ± 1.5
	T-Room	21.2 ± 0.5	21.2 ± 0.6	21.1 ± 0.4	21.2 ± 0.5	21.3 ± 0.5	21.2 ± 0.5	21.3 ± 0.6
MEC-group	T-Tra	37.7 ± 0.5	37.6 ± 0.5	37.4 ± 0.6	37.1 ± 0.6*	37.1 ± 0.7*	36.8 ± 0.6*	36.7 ± 0.6*
	T-Oes	38.8 ± 0.4	38.8 ± 0.5	38.5 ± 0.5	38.2 ± 0.5*	38.0 ± 0.5*	37.7 ± 0.6*	37.6 ± 0.6*
	T-Rec	38.6 ± 0.5	38.6 ± 0.5	38.4 ± 0.5	38.2 ± 0.6	38.0 ± 0.5*	37.8 ± 0.6*	37.6 ± 0.6*
	T-Gas	22.5 ± 1.6	22.6 ± 1.6	22.7 ± 1.6	22.8 ± 1.6	23.0 ± 1.6	23.1 ± 1.7	23.2 ± 1.7
	T-Room	21.1 ± 0.5	21.3 ± 0.4	21.3 ± 0.5	21.2 ± 0.4	21.2 ± 0.5	21.3 ± 0.5	21.1 ± 0.4

*Significantly lower compared to the baseline

https://doi.org/10.17221/229/2020-VETMED

In the MEC-group, the T-Tra was significantly lower compared to the baseline after 30 min (at T30 $P = 0.044$, at T40 $P = 0.014$, at T50 $P < 0.001$, at T60 $P < 0.001$), the T-Oes temperature was significantly lower compared to the baseline after 30 min (at T30 $P = 0.018$, at T40 $P < 0.001$, at T50 $P < 0.001$, at T60 $P < 0.001$), the T-Rec was significantly lower compared to the baseline after 40 min (at T40 $P = 0.017$, at T50 $P < 0.001$, at T60 $P < 0.001$; Table 1). There were no significant differences in the T-Gas and T-Room compared to the baseline.

There was no significant difference between the groups in the T-Tra, T-Oes, T-Rec, T-Gas, T-Room, HR and MAP. The f_R was significantly lower in the SPO-group compared to the MEC-group at all the times (at T10 $P < 0.001$, at T20 $P < 0.001$, at T30 $P < 0.001$, at T40 $P < 0.001$, at T50 $P < 0.001$, at T60 $P < 0.001$). The $ETCO_2$ was significantly higher in the SPO-group compared to the MEC-group at T10 ($P = 0.042$), at T20 ($P < 0.001$), at T30 ($P < 0.001$), at T40 ($P = 0.001$) and at T50 ($P = 0.037$).

DISCUSSION

In the present study, we compared the effect of spontaneous breathing and mechanical ventilation on the body temperature measured at three different anatomical sites for 60 minutes of anaesthesia.

The measurement of the tracheal temperature, described by Hayes et al. (1996), was obtained by an endotracheal tube with a temperature sensor mounted on the endotracheal tube shaft underneath the balloon. Hayes et al. (1996) found that the temperature measurement from the tracheal mucosa is a reliable site for measuring the body core temperature. We used a thermometer probe placed inside the tracheal lumen, introduced through the endotracheal tube. We observed differences between the T-Tra and T-Oes or T-Rec, which corresponded to the results of the study of Hayes et al. (1996). In the MEC-group, the decrease in the T-Tra was significantly lower ten minutes earlier than in the SPO-group. This can be due to the better alveolar ventilation and faster temperature decrease in the mechanically ventilated patient. However, the difference between the groups at the end of the measurement was not statistically significant. The oesophagus close to the heart base, or the rectum are places frequently used for the temperature measurement during general anaesthesia in small animals (Hayes et al. 1996).

Breathing dry and cold anaesthetic gases leads to heat losses from the pulmonary airways (Solway 1990). This is mainly due to convection (Stepaniuk and Brock 2008). In mechanical ventilation, there is usually a higher tidal volume, higher inspiratory pressure and higher respiratory frequency compared to the spontaneous breathing. Therefore, the level of the convection heat losses should be higher. However, we did not detect a significant difference in the temperature between the spontaneous breathing and mechanical ventilation during the sixty minutes of anaesthesia. This can be caused by the small volumes of fresh gases which can be warmed by recirculating gases.

The difference in the f_R and $ETCO_2$ can be explained by the improved ventilation in the lungs due to a higher respiratory rate and tidal volume during the mechanical ventilation.

The medetomidine used for premedication is an alpha-2 adrenergic agonist, which causes peripheral vasoconstriction and the central redistribution of the blood (Murrell and Hellebrekers 2005). These changes may enhance the maintenance of the body temperature by reducing the cutaneous heat loss (Sinclair 2003). Therefore, the temperature decrease should be slower when compared to other, e.g., vasodilatation drugs.

Our study has several limitations, it is focused on middle-sized dogs; further studies should focus on small breeds. We only used the combination of medetomidine and butorphanol as the premedication, comparison of alternative combinations could be the aim of measurements in further studies. We did not measure the minute volume because we did not have the proper equipment, the volumes displayed on the ventilator only provide inaccurate values. If the minute volume in our dogs differed significantly, it could affect the changes in the measured temperatures. However, in our study, healthy animals of the same weight category were included in both groups, in which we assumed a comparable tidal minute volume while maintaining the same ventilation parameters. In addition, the decrease in the observed temperatures in the spontaneously breathing and mechanically ventilated dogs was comparable. We, therefore, assume that the minute volume did not affect our results. We also did not assess the changes in the body temperatures for

https://doi.org/10.17221/229/2020-VETMED

a longer period than 60 min, this could be an objective for future research.

Hypothermia is a common peri-anaesthetic complication. Future studies could focus on the comparison of other ventilation methods, with or without active or passive heating of the patient. Also, comparison of different anaesthetic protocols should give more information about the development of the hypothermia.

In conclusion, during medetomidine-butorphanol-propofol-isoflurane anaesthesia in dogs, there is a comparable decrease in the body temperature from 30 to 60 min, irrespective of whether the dogs are breathing spontaneously or ventilated mechanically.

Conflict of interest

The authors declare no conflict of interest.

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