The aim of the study was to assess the value of capnometry during anaesthesia with spontaneous breathing and controlled ventilation in dogs free of pulmonary disease. Ten beagle dogs were included in the study. End-tidal carbon dioxide (ETCO2), minute respiratory volume (ME), heart rate and arterial blood gases were measured. The correlation between ETCO2 and PaCO2 was positive and statistically significant in both types of general anaesthesia. ME was negatively correlated with ETCO2 and PaCO2, although this was statistically significant only during controlled ventilation. The PaCO2 – ETCO2 gradient increased significantly comparing to the awake state during the experiment with controlled ventilation indicating haemodynamic depression as a consequence of deepening of anaesthesia. The results of the study demonstrated that capnometry can noninvasively provide valuable information about changes in minute respiratory volume and arterial blood gases during general anaesthesia with spontaneous breathing and controlled ventilation in dogs free of pulmonary disease. Comparable literature data imply that capnometry is equally useful in other animal species.

Key words: anaesthesia, capnometry, blood gases, dogs

INTRODUCTION

Carbon dioxide (CO2) is an end product of aerobic metabolism in mitochondria. The partial pressure of CO2 (pCO2) is greatest within them. A row of pressure gradients enable transport from the place of formation through the cytoplasm and extracellular fluid into the blood. As a result of the higher pCO2 tension in the blood entering the lung capillaries, the pCO2 in the alveoli enables diffusion into the alveolar mixture of gases and exhalation from the organism is possible. Non-physiological values of pCO2 cause important systemic patho-physiological effects (Nunn, 1987a).

Carbon dioxide is transported through the blood in three forms: dissolved as bicarbonate ions (HCO3−) and bound to amino groups and blood proteins (West, 1999). Cell membranes in the organism especially the brain barrier allow fast diffusion of CO2 and are relatively impermeable for hydrogen ions. The intra-
cellular concentration of hydrogen ions (H\(^+\)) does not change with changes in extracellular pH but is changed by dissolved CO\(_2\), which hydrates, ionizes and produces hydrogen ions after transfer into the cell. Because of this unique characteristic, CO\(_2\) is the only substance normally present in the organism, which is capable of changing the intracellular pH in this way. This feature of CO\(_2\) is responsible for the changes in the acid-base balance, vascular resistance and permeability (Nunn, 1987a).

The partial pressure of CO\(_2\) depends on the balance between formation and excretion. It is smaller in tissues with low metabolic activity and relatively high blood flow, like skin, and larger in tissues with high metabolic activity in relation to their blood flow, like the myocardium. Mixed venous blood, which can be sampled via catheterisation of the pulmonary artery, represents a uniform value of pCO\(_2\) within the entire organism. CO\(_2\) passes into the alveolar air through the lung capillaries, which causes the pCO\(_2\) to increase during exhalation. During inhalation the inhaled air dilutes the alveolar air and pCO\(_2\) decreases by about 0.4 kPa. The blood that exits on the artery side in the lung capillaries has a pCO\(_2\) almost the same as the pCO\(_2\) in the alveolar air and therefore changes over time similarly to pCO\(_2\) in the alveolar air (Nunn, 1987b). There are pulmonary regional differences in pCO\(_2\) because of ventilation/perfusion mismatch (V/Q) (Nunn, 1987b). Mixed arterial blood coming from the lungs represents a uniform value of paCO\(_2\) with cyclic changes due to breathing.

Alveolar ventilation VA, the product of respiratory frequency (f) and the difference between tidal volume (VT) and physiologic dead space (VD) is the major factor influencing pCO\(_2\) in the alveolar air (Nunn, 1987c; Keele et al., 1983).

In normal physiological conditions excretion of CO\(_2\) is equal to production. Different abnormal conditions may change this ratio. During acute hypoventilation the greater part of CO\(_2\) accumulates in the body, so excretion temporarily decreases until the alveolar concentration of CO\(_2\) increases. On the contrary, during acute hyperventilation the excretion of CO\(_2\) temporarily increases (Nunn, 1987d). A sudden decrease of cardiac index also induces a decrease in CO\(_2\) excretion until the concentration of CO\(_2\) in the mixed venous blood increases (Nunn, 1987e).

In normal healthy organisms in an awake state the air at the end of exhalation consists almost exclusively of alveolar air. When certain parts of the lungs are ventilated but not perfused for different reasons, they do not contribute CO\(_2\) from the alveolar space to ETCO\(_2\). There fore, ETCO\(_2\) from this part of the lungs has a lower pCO\(_2\) than that from perfused parts of the lungs. Despite this, the arterial PCO\(_2\) has nearly the mean value of PCO\(_2\) of perfused alveoli (Nunn, 1987f). Different investigations in patients free of lung disease during general anaesthesia have shown that paCO\(_2\) values exceed ETCO\(_2\) values by approximately 0.4 – 0.7 kPa (5 mmHg). For practical reasons arterial and alveolar pCO\(_2\) are equated despite small differences due to shunts and ventilation/perfusion mismatch (Nunn, 1987f).

The CO\(_2\) reserves and the bicarbonate ions in the body are approximately 100 times larger than the amount of oxygen. In cases of ventilatory – metabolic activity disproportion the paCO\(_2\) changes very slowly. CO\(_2\) reserves are ranked into fast, mid-fast and slow. Fast reserves are present in circulating blood, brain, kidneys and other well perfused tissues. The mid-fast reserves are in rested muscles
and other tissues with a moderate degree of perfusion. The slow ones include bones, fat and tissues that can accumulate CO₂. The rate of pCO₂ increase is actually much slower than the rate of pCO₂ decrease, which is favourable for patients in respiratory distress.

Lung diseases can adversely affect the accuracy of capnometry because of the changing arterial – ETCO₂ gradient (Nunn, 1987f). Therefore, indirectly monitoring the inspired and expired air may check lung ventilation, cardiac output, distribution of blood flow and metabolic activity of the organism (Gilbert et al., 1992; Isserles et al., 1991; Dubin et al., 2000).

Capnometry is a method of measuring and a numerical presentation of the concentration or partial pressure of carbon dioxide during inspiration and expiration, while capnography is a continuous graphical presentation of changes in CO₂ during the breathing cycle (Pierce, 1984). Capnometry is a widely used noninvasive substitute for PaCO₂ in anaesthesia, anaesthetic recovery and intensive care, although it is not a replacement for arterial blood gas analysis, but rather serves as an adjunctive monitoring tool (Cheng et al., 1999; Proulx, 1999). Besides dogs and cats, the most common patients in veterinary medicine, capnometry is also useful during anaesthesia of different animal species even during short-term injectable anaesthesia (Raußer et al., 2002). During anaesthesia the method depends on connections between arterial CO₂ (PaCO₂), alveolar CO₂ (PACO₂) and CO₂ at the end of expiration (ETCO₂). ETCO₂ enables the clinical assessment of the arterial CO₂ (PaCO₂) in cases where ventilation and perfusion in the lungs are in accordance, if CO₂ uninterruptedly passes through the alveolar capillary membrane and there are no mistakes in sampling and measuring. After completing the listed criteria ETCO₂ can reflect the changes in PaCO₂, despite the fact that all the alveoli during breathing do not empty simultaneously. In an ideal mathematical model the difference between PaCO₂ and PACO₂, is equal to 0 (Schieber et al., 1985). We may assume that under the above conditions ETCO₂ = PACO₂ = paCO₂. If the gradient between PaCO₂ and ETCO₂ is small and constant, capnometry enables a non-invasive, continuous and real time assessment of breathing (Schieber et al., 1985; Badgwell et al., 1987). The typical gradient between paCO₂ and ETCO₂ during general anaesthesia is 5 – 10 mmHg (0.7 – 1.2 Kpa). Ventilation/perfusion mismatch (V/Q) and disturbances in sampling unfortunately increase the gradient between paCO₂ and ETCO₂. The bigger the gradient, the more difficult is assessment of paCO₂, especially if the causes are of a dynamic nature.

The aim of this study was to assess paCO₂ by ETCO₂ indirectly, to prove or disprove the reliability and clinical applicability of capnometry in distinct phases of general anaesthesia with spontaneous breathing and controlled ventilation along with establishing a correlation between ETCO₂ and CO₂ in all these phases.

MATERIAL AND METHODS

Ten beagles dogs of both sexes, weighing on average 14 kg and 1.5 years of age, were included in the study. They originated from the same kennel and were dewormed and vaccinated. They where clinically healthy and fed a commercial
diet for dogs. Tidal volume, respiratory frequency and minute volume were measured and arterial blood gas analyses done and compared to the results of capnometry. Measurements were first taken during general anaesthesia with spontaneous breathing and a month later with controlled ventilation. Each animal served as its own control.

The experiment was divided into the following phases:

- F1 Before premedication
- F2 After premedication
- F3 Directly after intubation
- F4 After one minute of general anaesthesia
- F5 After five minutes of general anaesthesia
- F6 After ten minutes of general anaesthesia
- F7 After twenty minutes of general anaesthesia
- F8 After forty minutes of general anaesthesia
- F9 After ten minutes of recovery
- F10 After fifteen minutes of recovery

In the phases preceding intubation, tidal volume, respiratory frequency and capnometric sampling were performed with a face mask connected to the Ohmeda Volume Monitor 5410 set-up to the paediatric programme and the capnometer (Ohmeda – Oxicap 4700). The measurements were taken for 1 minute and the mean value of all was recorded. Minute respiratory volume was calculated from f and Vt. ETCO2 of all the expirations in one minute were measured and the mean value recorded. A sample of 1.0 ml of arterial blood from the left femoral artery was taken by percutaneous puncture with a purpose made syringe and put into an ice-cold bath to prevent changes in blood gas content and pH in vitro until analysis (Madiero et al., 1980). Samples of arterial blood were taken during all phases of the experiment.

Premedication drugs, 0.5 mg/kg b.w. of acepromazine maleate (Prom Ace, Forth Doge Animal Health) and 0.2 mg/kg b.w. of butorphanol tartrate (Torbugesic, Forth Doge Animal Health) were then injected intravenously. Measurements were taken again after 10 minutes. The area of the right femoral artery was aseptically prepared and infiltrated with 2 ml of 1% lidocaine HCl (Xylocaine, Astra Zeneca), the artery was surgically prepared and an arterial cannula inserted to enable arterial blood sampling throughout the experiment.

Dogs were induced with a bolus injection of 6 mg/kg b.w. of thiopental sodium (Nesdonal, Specia, Paris). General anaesthesia was maintained with circular, partially rebreathing anaesthetic equipment (Ohmeda – VMS) and a pneumatic ventilator (Ohmeda – Fluidor MK2). Respiratory frequency was set at 17/min and an average respiratory volume of 16.5 ml/kg. Halothane (Fluothane, Zeneca, UK) at 2 vol. % in O2 and N2O (1:2) was used for maintaining anaesthesia. The flow of O2 was 30 ml/kg b.w. with an adequate flow of N2O. During anaesthesia with controlled ventilation we adapted the flow of gases to the needs of the ventilator.
(Ohmeda – Fluidor MK2) with a ratio of O₂ : N₂O = 1 : 2 (FiO₂ = 0.33). Dogs were neuromuscularly blocked with 0.4 mg/kg of atracurium besylate.

The time of the beginning of recovery was taken at the turning off of the knob of the vaporiser, which cut off the flow of N₂O. The animals remained intubated and inhaled pure O₂ until the next measurement in 15th minute of recovery when arterial blood was taken for analysis and the arterial cannula was removed. After the measurements were taken reflexes started to occur and the dogs were extubated.

STATISTICAL ANALYSES

Data were analysed using the SPSS 10.0 statistical programme for Windows (SPSS - statistical package for social sciences). Changes in parameters measured or calculated (ETCO₂, PaCO₂, pH, HCO₃⁻, difference between PaCO₂ and ETCO₂) were assessed by repeated measures ANOVA (Petrie et al., 1999). Pearson correlation coefficients were calculated by use of Pearson corr. Unless otherwise stated, a value of p<0.05 was considered significant.

RESULTS

1. ETCO₂, PaCO₂ and heart rate during anaesthesia with spontaneous breathing

The values of ETCO₂ (figure 1) increased after 1 minute of general anaesthesia with spontaneous breathing and remained increased until the end of the exp-

Figure 1. ME, ETCO₂ and PaCO₂ in spontaneous breathing
F1 - before premedication; F2 - after premedication; F3 - directly after intubation; F4 - after one minute of general anaesthesia; F5 - after five minutes of general anaesthesia; F6 - after ten minutes of general anaesthesia; F7 - after twenty minutes of general anaesthesia; F8 - after forty minutes of general anaesthesia; F9 - after ten minutes of recovery; F10 - after fifteen minutes of recovery.
experiment with mild decreases during recovery (the ninth and tenth phases). A statistically significant increase in ECTO₂ in comparison to the first phase, when the dogs were awake, occurred from the fourth to ninth phase and was above the reference value (Jacobs et al., 1995; Ilkiw et al., 1991).

The pressure gradient between PaCO₂ and ETCO₂ did not statistically significantly change compared to the gradient in the first phase of the experiment.

The correlation coefficient (r) between ETCO₂ and PaCO₂ was 0.59 (P=0.0001).

Heart rate was also measured during anaesthesia with spontaneous breathing, but the data are not shown as values remained within the normal range throughout the experiment.

1.1 Comparison of ETCO₂, PaCO₂ and minute respiratory volume (ME)

The correlation coefficient (r) between ETCO₂ and ME was -0.17 (P=0.108) and between PaCO₂ and ME -0.01 (P=0.487).

1.2 Values of the arterial HCO₃⁻ and pH and their comparison to ETCO₂ values

Arterial HCO₃⁻ (figure 2) increased significantly in the second phase compared to the first phase and remained increased until the end of the experiment. In spite of the statistically significant increase, the values of HCO₃⁻ did not rise above the reference range throughout the experiment (Jacobs et al., 1995; Ilkiw et al., 1991).

![Figure 2: Arterial HCO₃⁻ during spontaneous breathing](image-url)

F1 - before premedication; F2 - after premedication; F3 - directly after intubation; F4 - after one minute of general anaesthesia; F5 - after five minutes of general anaesthesia; F6 - after ten minutes of general anaesthesia; F7 - after twenty minutes of general anaesthesia; F8 - after forty minutes of general anaesthesia; F9 - after ten minutes of recovery; F10 - after fifteen minutes of recovery.

*statistically significant difference (p<0.05) compared to F1
Arterial pH (figure 3) decreased in the second phase. Compared to the first phase when the pH was within the reference range a statistically significant decrease in pH value below the reference range (Jacobs et al., 1995; Ilkiw et al., 1991) occurred in the fourth phase and it remained low to the end of the experiment.

The correlation coefficient ($r$) between $\text{HCO}_3^-$ and pH was 0.21 ($P=0.072$) in all the phases. The correlation coefficient ($r$) between $\text{ETCO}_2$ and $\text{HCO}_3^-$ was $-0.06$ ($P=0.49$) and between $\text{ETCO}_2$ and pH $-0.62$ ($P=0.0001$).

2. $\text{ETCO}_2$, $\text{PaCO}_2$ and heart rate during anaesthesia with controlled ventilation

The values of $\text{ETCO}_2$ (figure 4) in different phases changed significantly compared to the first phase. $\text{ETCO}_2$ increased significantly in the second and third phases and then decreased from the fourth to the tenth phase. The values of $\text{PaCO}_2$ (figure 4) also increased significantly in the second and third phases and then decreased in the sixth, seventh, and eighth phase. They were above the reference range (Jacobs et al., 1995; Ilkiw et al., 1991) in the third phase and below it in the fifth phase and remained decreased until the end of the experiment.
The pressure gradient of PaCO₂ and ETCO₂ significantly increased during controlled ventilation from the fourth phase until the end of the experiment (table 1). Not only was there a statistically significant increase in the gradient compared to the value in the first phase, the gradient value from the fourth phase on had also increased, which is evident in table 1.

Table 1. Pressure gradient between PaCO₂ and ETCO₂ during controlled ventilation

<table>
<thead>
<tr>
<th>Phases of experiment</th>
<th>Mean value of the gradient between PaCO₂ and ETCO₂ (kPa)</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>F1</td>
<td>-0.478</td>
<td>0.492</td>
</tr>
<tr>
<td>F2</td>
<td>-0.196</td>
<td>0.397</td>
</tr>
<tr>
<td>F3</td>
<td>0.092</td>
<td>0.475</td>
</tr>
<tr>
<td>F4</td>
<td>0.820*</td>
<td>0.511</td>
</tr>
<tr>
<td>F5</td>
<td>0.790*</td>
<td>0.322</td>
</tr>
<tr>
<td>F6</td>
<td>0.988*</td>
<td>0.351</td>
</tr>
<tr>
<td>F7</td>
<td>1.086*</td>
<td>0.476</td>
</tr>
<tr>
<td>F8</td>
<td>1.092*</td>
<td>0.324</td>
</tr>
<tr>
<td>F9</td>
<td>1.122*</td>
<td>0.201</td>
</tr>
<tr>
<td>F10</td>
<td>0.918*</td>
<td>0.461</td>
</tr>
</tbody>
</table>

* statistically significant difference in the gradient compared to F1

Figure 4. ME, ETCO₂ and PaCO₂ during controlled ventilation

F1 - before premedication; F2 - after premedication; F3 - directly after intubation; F4 - after one minute of general anaesthesia; F5 - after five minutes of general anaesthesia; F6 - after ten minutes of general anaesthesia; F7 - after twenty minutes of general anaesthesia; F8 - after forty minutes of general anaesthesia; F9 - after ten minutes of recovery; F10 - after fifteen minutes of recovery.

*statistically significant difference (P<0.05) in the gradient between PaCO₂ and ETCO₂ compared to F1
The correlation coefficient (r) between the values of ETCO$_2$ in PaCO$_2$ was 0.91 (P=0.0001) during all the phases.

Heart rate measurements during anaesthesia with controlled ventilation showed statistically significant changes (table 2) with bradycardia in the last two phases.

Table 2. Heart rate during anaesthesia with controlled ventilation

<table>
<thead>
<tr>
<th>Phases of experiment</th>
<th>Mean value (beats/min)</th>
<th>Standard deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>F1</td>
<td>106.2</td>
<td>16.5</td>
</tr>
<tr>
<td>F2</td>
<td>80.0*</td>
<td>21.4</td>
</tr>
<tr>
<td>F3</td>
<td>97.3</td>
<td>13.8</td>
</tr>
<tr>
<td>F4</td>
<td>88.7*</td>
<td>19.6</td>
</tr>
<tr>
<td>F5</td>
<td>88.1*</td>
<td>17.1</td>
</tr>
<tr>
<td>F6</td>
<td>93.0</td>
<td>12.0</td>
</tr>
<tr>
<td>F7</td>
<td>93.5</td>
<td>9.8</td>
</tr>
<tr>
<td>F8</td>
<td>90.3*</td>
<td>11.1</td>
</tr>
<tr>
<td>F9</td>
<td>59.5*</td>
<td>12.4</td>
</tr>
<tr>
<td>F10</td>
<td>58.7*</td>
<td>16.5</td>
</tr>
</tbody>
</table>

* statistically significant difference in the gradient compared to F1

2.1 Comparison of ETCO$_2$, PaCO$_2$ values and the minute respiratory volume (ME)

The correlation coefficient (r) between ETCO$_2$ and ME was –0.77 (P=0.001), and between PaCO$_2$ and ME it was –0.61 (P=0.0001).

2.2 The values of the arterial HCO$_3^-$ and pH and comparison to values of ETCO$_2$

Arterial HCO$_3^-$ (figure 5) increased significantly in the second, third and fourth phases compared to the first phase. Despite the increase, HCO$_3^-$ remained within the reference range (Jacobs et al., 1995; Ilkiw et al., 1991). HCO$_3^-$ then decreased below the reference range from the seventh to tenth phase.

Arterial pH (figure 6) changed similarly to HCO$_3^-$, although in the opposite direction (figure 5). The decrease was statistically significant in the second and third phases (below the reference range in the third phase). There was a statistically significant increase above the reference range between the fifth and eighth phase (Jacobs et al., 1995; Ilkiw et al., 1991).
The correlation coefficient \( r \) between \( \text{HCO}_3^- \) and pH was –0.53 (\( P=0.0001 \)). Between ETCO\(_2\) and pH it was –0.85 (\( P=0.0001 \)) and between ETCO\(_2\) and \( \text{HCO}_3^- \) 0.68 (\( P=0.0001 \)).
DISCUSSION

It is known that end-tidal carbon dioxide (ETCO₂) measurements have the potential to reflect PaCO₂ and to serve as a noninvasive measure of alveolar ventilation, metabolic responses and haemodynamic changes (Szaflarski et al., 1991). Capnometry became the golden standard in human and veterinary anesthesia and is probably the most important parameter obtained noninvasively during human and veterinary general anesthesia and in human intensive care units. Limited data are available from studies where correlations were calculated between PaCO₂ and end-tidal carbon dioxide in healthy, anaesthetised, spontaneously breathing and ventilated dogs, although several studies have been performed in human patients (Hendricks, 1995). The aim of this study was to obtain our own data on the above mentioned relations. The experiment was carried out in ten healthy anaesthetised spontaneously breathing and ventilated dogs. Although different parameters of arterial blood gas analysis have been measured and correlated to end-tidal measurements, we focused on the correlations between ETCO₂, minute respiratory volume and PaCO₂. The accuracy of noninvasive assessment of PaCO₂ with ETCO₂ is expressed in the PaCO₂ – ETCO₂ gradient, which should theoretically be small and constant (Schieber et al., 1985; Badgwell et al., 1987). It was calculated and presented in ventilated dogs due to the statistically significant changes of gradient.

The correlation of the results for PaCO₂ and ETCO₂ during spontaneous breathing showed a typical gradient between PaCO₂ and ETCO₂ with values of PaCO₂ greater than ETCO₂ except in two phases, although without statistical significance, which is in accordance with results from the literature for healthy patients during general anaesthesia. The correlation was statistically significant ($r=0.59$, $P=0.0001$), which additionally confirms the accuracy of assessment of PaCO₂ by ETCO₂. The correlation between minute ventilation and PaCO₂ and ETCO₂ tended to be negative with no statistical significance and a slight delay between minute ventilation and PaCO₂ and ETCO₂ values, although the close time agreement between PaCO₂ and ETCO₂ again demonstrates the value of capnometry during general anaesthesia in patients without primary cardiorespiratory dysfunction (Prause et al., 1997). Arterial HCO₃⁻ and pH changed together with the decrease in minute ventilation during anaesthesia with spontaneous breathing. There was not statistically significant correlation between ETCO₂ and HCO₃⁻ but a strongly negative significant correlation with pH.

The changes of PaCO₂ and ETCO₂ in ventilated dogs showed a similar pattern to that during anaesthesia with spontaneous breathing in the first phases. They reflected the respiratory depressive effect of anaesthetic drugs at premedication and intubation. However in the following phases when the patients were ventilated with a constant minute respiratory volume the changes of PaCO₂ and ETCO₂ showed an opposite pattern. The correlation between those two parameters was statistically significant ($r = 0.91; P = 0.0001$), which is in agreement with some recently published data (Cheng et al., 1999). The constant minute ventilation ($f = 17$/min and TD = mean 16.5 ml/kg) with unchanged vol.% of halothane (2 vol.%) caused hyperventilation and haemodynamic depression with moderate to
severe bradycardia, and most probably an accompanying decrease in cardiac output reflected by a decrease in PaCO₂ and ETCO₂ (Grudler et al., 1984; Trevino et al., 1985; Weil et al., 1985; Isserles et al., 1991; Dubin et al., 2000). In normal clinical settings such a decrease in ETCO₂ would be followed by resetting of minute ventilation through changes in respiratory frequency or tidal volume to assure normocapny and/or readjusting the vol.% of halothane to decrease the depth of anaesthesia. At the time the experiment was carried out the Swan – Ganz method of pulmonary artery catheterisation with cardiac output measurements and mixed venous blood sampling was unavailable and only arterial blood was analyzed for blood gases. Namely, the gradient between PaCO₂ and ETCO₂ was increased significantly from phase four to the end of the experiment, to be the greatest in phase 9. Only mixed venous blood gas analysis could explain this phenomenon. We may assume that, due to depression of haemodynamics induced by hyperventilation with an unchanged vol.% of halothane, the pulmonary circulation was also impaired with a decrease in CO₂ excretion via the lungs (Weil et al., 1985; Weil et al., 1986). We may also assume that at time of lowest ETCO₂ values, the experimental animals developed mixed venous hypercarbia and normal – to slight arterial hypocarbia with an increased PaCO₂ – ETCO₂ gradient due reduced excretion of CO₂ (Weil et al., 1985; Weil et al., 1986).

Arterial HCO₃⁻ and pH measurements also reflected hyperventilation and haemodynamic depression. The correlation between ETCO₂ and pH was negative \( r=-0.85, \ P=0.0001 \) and between ETCO₂ and HCO₃ positive \( r=0.68 \ P=0.0001 \).

The results of this study confirm that capnometry can noninvasively provide invaluable information about changes in minute respiratory volume and arterial blood gases during general anaesthesia with spontaneous breathing and controlled ventilation in dogs without pulmonary disease. Comparable literature data imply that capnometry is equally useful in other animal species.

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REFERENCES


Cilj ove studije je bio da se oceni značaj kapnometrije u anesteziji sa spontanim disanjem i kontrolišanoj ventilacijom kod pasa bez bolesti pluća. U studiju je bilo uključeno deset pasa rase beagle. Mereni su ugljen dioksid na kraju ekspirijuma (ETCO$_2$), minutni disajni volumen (ME), frekvencu rada srca i parcijalni pritisci gasova u arterijskoj krvi. Korelacije između ETCO$_2$ i PaCO$_2$ bile su u oba tipa opšte anestezije pozitivne i statistički signifikantne. ME je bio u negativnoj korelaciji sa ETCO$_2$ i PaCO$_2$, ali su razlike bile statistički signifikantne samo kod kontrolisane ventilacije. PaCO$_2$ – ETCO$_2$ gradijent se statistički signifikantno povećavao u poređenju sa budnim stanjem u vreme studije sa kontrolisanoj ventilacijom pokazujući hemodinamičku depresiju kao učinak produbljenja anestezije. Rezultati studije potvrđuju, da kapnometrija na neinvazivan način omogućava dobijanje značajnih informacija o promenama u minutnom respiratornom volumenu i parcijalnim pritiscima gasova u arterijskoj krvi u toku opšte anestezije sa spontanim disanjem i kontrolisanoj ventilacijom kod pasa bez oboljenja pluća. Podaci iz literature ukazuju da je kapnometrija jednakо upotrebljiva i kod drugih životnijskih vrsta.