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RELATION BETWEEN END-TIDAL AND ARTERIAL CARBON DIOXIDE PARTIAL PRESSURE DURING GENERAL ANAESTHESIA WITH SPONTANEOUS BREATHING AND CONTROLLED VENTILATION IN DOGS – AN EXPERIMENTAL STUDY

KRULJC P*, NEMEC ALENKA*, VINTAR NELI** and BUTINAR J*

*University of Ljubljana, Veterinary Faculty, Ljubljana, Slovenia , ** Clinical Department for Anesthesiology and Intensive Care, University Clinical Centre, Ljubljana, Slovenia

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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 ($ETCO_2$), minute respiratory volume (ME), heart rate and arterial blood gases were measured. The correlation between $ETCO_2$ and $PaCO_2$ was positive and statistically significant in both types of general anaesthesia. ME was negatively correlated with $ETCO_2$ and $PaCO_2$, although this was statistically significant only during controlled ventilation. The $PaCO_2 - ETCO_2$ 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 (CO_2) is an end product of aerobic metabolism in mitochondria. The partial pressure of CO_2 (pCO_2) 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 pCO_2 tension in the blood entering the lung capillaries, the pCO_2 in the alveoli enables diffusion into the alveolar mixture of gases and exhalation from the organism is possible. Non-physiological values of pCO_2 cause important systemic patho-physiological effects (Nunn, 1987a).

Carbon dioxide is transported through the blood in three forms: dissolved as bicarbonate ions (HCO_3^-) and bound to amino groups and blood proteins (West, 1999). Cell membranes in the organism especially the brain barrier allow fast diffusion of CO_2 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 V_A, the product of respiratory frequency (f) and the difference between tidal volume (V_T) and physiologic dead space ($V_{D)}$ 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 CO_2. The rate of pCO_2 increase is actually much slower than the rate of pCO_2 decrease, which is favourable for patients in respiratory distress.

Lung diseases can adversely affect the accuracy of capnometry because of the changing arterial – $ETCO_2$ gradient (Nunn**,** 1987f). Therefore, indirectly monitoring the inspired and expired air may chech 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 CO_2 during the breathing cycle (Pierce, 1984). Capnometry is a widely used noninvasive substitute for $PaCO_2$ 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 (Raušer *et al.*, 2002). During anaesthesia the method depends on connections between arterial CO_2 ($PaCO_2$), alveolar CO_2 ($PACO_2$) and CO_2 at the end of expiration ($ETCO_2$). $ETCO_2$ enables the clinical assessment of the arterial CO_2 ($PaCO_2$) in cases where ventilation and perfusion in the lungs are in accordance, if CO_2 uninterruptedly passes through the alveolar capillary membrane and there are no mistakes in sampling and measuring. After completing the listed criteria $ETCO_2$ can reflect the changes in $PaCO_2$, despite the fact that all the alveoli during breathing do not empty simultaneously. In an ideal mathematical model the difference between $PaCO_2$ and $PACO_2$, is equal to 0 (Schieber *et al.*, 1985). We may assume that under the above conditions $ETCO_2 \cong PACO_2 \cong paCO_2$. If the gradient between $PaCO_2$ and $ETCO_2$ 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 $paCO_2$ and $ETCO_2$ 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 $paCO_2$ and $ETCO_2$. The bigger the gradient, the more difficult is assessment of $paCO_2$, especially if the causes are of a dynamic nature.

The aim of this study was to assess $paCO_2$ by $ETCO_2$ 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 $ETCO_2$ and CO_2 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. $ETCO_2$ 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 O_2 and N_2O (1:2) was used for maintaining anaesthesia. The flow of O_2 was 30 ml/kg b.w. with an adequate flow of N_2O. During anaesthesia with controlled ventilation we adapted the flow of gases to the needs of the ventilator

(Ohmeda – Fluidor MK2) with a ratio of $O_2 : N_2O = 1 : 2$ ($FiO_2 = 0.33$). 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 N_2O. The animals remained intubated and inhaled pure O_2 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 ($ETCO_2$, $PaCO_2$, pH, HCO_3^-, difference between $PaCO_2$ and $ETCO_2$) 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. $ETCO_2$, $PaCO_2$ and heart rate during anaesthesia with spontaneous breathing

The values of $ETCO_2$ (figure 1) increased after 1 minute of general anaesthesia with spontaneous breathing and remained increased until the end of the ex-

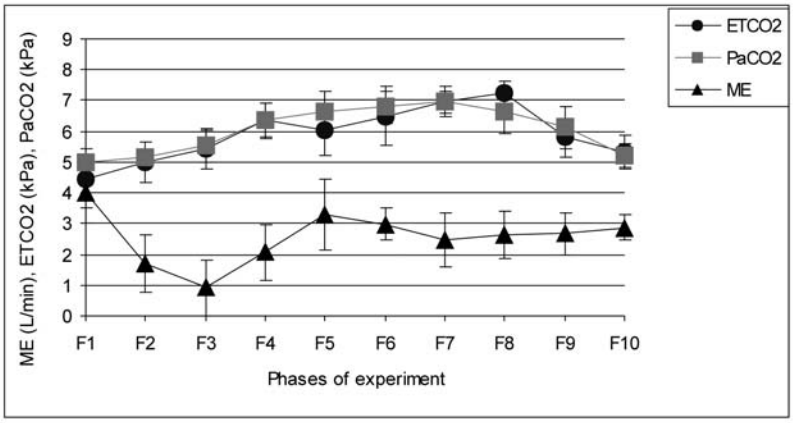

Figure 1. ME, $ETCO_2$ and $PaCO_2$ in spontaneous breathing

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.

periment with mild decreases during recovery (the ninth and tenth phases). A statistically significant increase in $ECTO_2$ 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 $PaCO_2$ and $ETCO_2$ did not statistically significantly change compared to the gradient in the first phase of the experiment.

The correlation coefficient (r) between $ETCO_2$ and $PaCO_2$ 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 $ETCO_2$, $PaCO_2$ and minute respiratory volume (ME)

The correlation coefficient (r) between $ETCO_2$ and ME was -0.17 (P=0.108) and between $PaCO_2$ and ME -0.01 (P=0.487).

1.2 Values of the arterial HCO_3 and pH and their comparison to $ETCO_2$ values

Arterial HCO_3^- (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 HCO_3^- did not rise above the reference range throughout the experiment (Jacobs *et al.,* 1995; Ilkiw *et al.*, 1991).

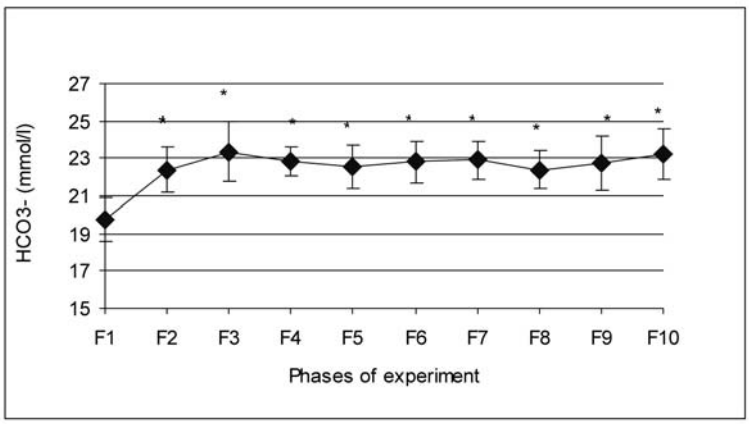

Figure 2: Arterial HCO_3^- during spontaneous breathing

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.
*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.

Figure 3. Arterial pH during 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.
*statistically significant difference ($p<0.05$) compared to F1

The correlation coefficient (r) between HCO_3^- and pH was 0.21 ($P=0.072$) in all the phases. The correlation coefficient (r) between $ETCO_2$ and HCO_3^- was –0.06 ($P=0.49$) and between $ETCO_2$ and pH –0.62 ($P=0.0001$).

2. $ETCO_2$, $PaCO_2$ and heart rate during anaesthesia with controlled ventilation

The values of $ETCO_2$ (figure 4) in different phases changed significantly compared to the first phase. $ETCO_2$ increased significantly in the second and third phases and then decreased from the fourth to the tenth phase. The values of $PaCO_2$ (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.

Figure 4. ME, $ETCO_2$ and $PaCO_2$ 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 $PaCO_2$ and $ETCO_2$ compared to F1

The pressure gradient of $PaCO_2$ and $ETCO_2$ (figure 4, table 1) significantly increased during controlled ventilation from the fourth phase until the end 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.

Table1. Pressure gradient between $PaCO_2$ in $ETCO_2$ during controlled ventilation

Phases of experiment	Mean value of the gradient between $PaCO_2$ and $ETCO_2$ (kPa)	Standard Deviation
F1	-0.478	0.492
F2	-0.196	0.397
F3	0.092	0.475
F4	0.820*	0.511
F5	0.790*	0.322
F6	0.988*	0.351
F7	1.086*	0.476
F8	1.092*	0.324
F9	1.122*	0.201
F10	0.918*	0.461

* statistically significant difference in the gradient 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

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

* 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).

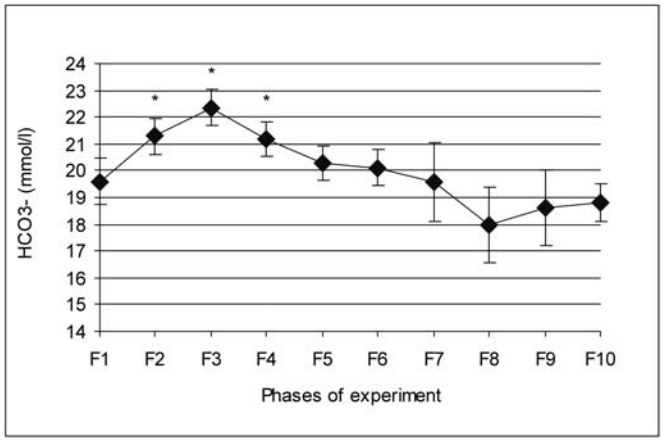

Figure 5. Arterial HCO_3^- during controlled ventilation

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 ($p<0.05$) compared to F1

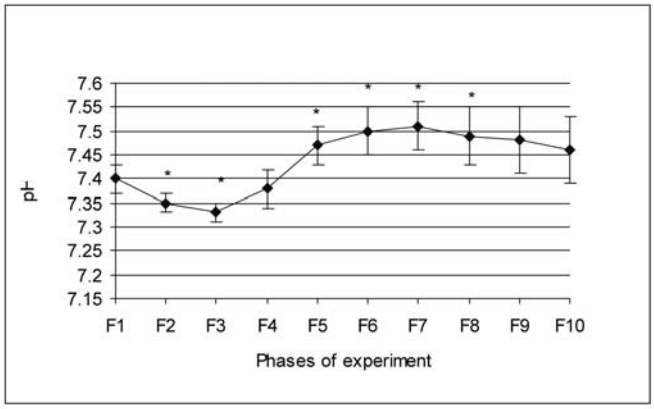

Figure 6. Arterial pH during controlled ventilation

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 ($p<0,05$) compared to F1

The correlation coefficient (r) between 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 HCO_3^- 0.68 ($P=0.0001$).

DISCUSSION

It is known that end-tidal carbon dioxide ($ETCO_2$) measurements have the potential to reflect $PaCO_2$ 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 anaesthesia and in human intensive care units. Limited data are available from studies where correlations were calculated between $PaCO_2$ 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 $ETCO_2$, minute respiratory volume and $PaCO_2$. The accuracy of noninvasive assessment of $PaCO_2$ with $ETCO_2$ is expressed in the $PaCO_2 - ETCO_2$ 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 $PaCO_2$ and $ETCO_2$ during spontaneous breathing showed a typical gradient between $PaCO_2$ and $ETCO_2$ with values of $PaCO_2$ greater than $ETCO_2$ 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 $PaCO_2$ by $ETCO_2$. The correlation between minute ventilation and $PaCO_2$ and $ETCO_2$ tended to be negative with no statistical significance and a slight delay between minute ventilation and $PaCO_2$ and $ETCO_2$ values, although the close time agreement between $PaCO_2$ and $ETCO_2$ again demonstrates the value of capnometry during general anaesthesia in patients without primary cardiorespiratory dysfunction (Prause *et al.,* 1997). Arterial HCO_3^- and pH changed together with the decrease in minute ventilation during anaesthesia with spontaneous breathing. There was not statistically significant correlation between $ETCO_2$ and HCO_3^- bbut a strongly negative significant correlation with pH.

The changes of $PaCO_2$ and $ETCO_2$ 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 $PaCO_2$ and $ETCO_2$ 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 $PaCO_2$ and $ETCO_2$ (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 $ETCO_2$ 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 $PaCO_2$ and $ETCO_2$ 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 CO_2 excretion via the lungs (Weil *et al.,* 1985; Weil *et al.,* 1986). We may also assume that at time of lowest $ETCO_2$ values, the experimental animals developed mixed venous hypercarbia and normal – to slight arterial hypocarbia with an increased $PaCO_2 - ETCO_2$ gradient due reduced excretion of CO_2 (Weil *et al.,* 1985; Weil *et al.,* 1986).

Arterial HCO_3^- and pH measurements also reflected hyperventilation and haemodynamic depression. The correlation between $ETCO_2$ and pH was negative ($r=-0,85$, $P=0.0001$) and between $ETCO_2$ and HCO_3 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.

Address for correspondence:
Dr. Peter Kruljc
University of Ljubljana,
Veterinary faculty,
Gerbičeva 60,
1000 Ljubljana, Slovenia
e-mail: peter.kruljc@vf.uni-lj.si

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PARCIJALNI PRITISCI U TOKU OPŠTE ANESTEZIJE PASA PRI SPONTANOM DISANJU I KONTROLISANOJ VENTILACIJI - EKSPERIMENTALNA STUDIJA

KRULJC P, NEMEC ALENKA, VINTAR NELI i BUTINAR J

SADRŽAJ

Cilj ove studije je bio da se oceni značaj kapnometrije u anesteziji sa spontanim disanjem i kontrolisanom 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), frekvenca 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 kontrolisanom 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 kontrolisanom ventilacijom kod pasa bez oboljenja pluća. Podaci iz literature ukazuju da je kapnometrija jednako upotrebljiva i kod drugih životinjskih vrsta.