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Research Article

Acid-Base Status and Blood Gas Analysis in Three Different Anaesthesia Schemes in Dogs

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Abstract: Changes in the parameters of acid-base status and blood gas analysis in 3 main methods of anaesthesia in dogs were investigated. The experiment was carried out in 22 male and female dogs, matched for breed, age and body weight and divided into 3 groups.

Animals from the first group (n = 8) were subjected to standard inhalation anaesthesia using halothane. Those from the second group (n = 7) received balanced anaesthesia using pancuronium, and those from the third group (n = 7) received epidural lumbosacral anaesthesia using lidocaine. Arterialised capillary blood samples were obtained from all animals in the following periods: immediately before anaesthesia (0 min), during premedication (30 min), during deep anaesthesia (120 min), after recovery (about 140 min) and the next day (24 h). The acid-base and blood gas parameters (pH, $PaCO_2$, PaO_2, HCO_3, TCO_2, ABE, SBE, SBC, SAT, and O_2CT) were determined.

The results showed that during the deep stages of halothane and balanced anaesthesia primary noncompensated respiratory acidosis and overoxygenation developed. These changes were only eliminated during the recovery period. The epidural anaesthesia was not accompanied by any changes in blood gas and acid-base status.

Key Words: Respiratory acidosis, halothane, pancuronium, lidocaine

Introduction

Different anaesthetics, their combinations, type of surgery, animal species, body position etc. alter blood gas and acid-base compositions (1-6).

As a rule, anaesthesia is accompanied by hypotension and hypothermia, which adversely affect oxygen and fluid-salt metabolism. The anaesthetics themselves can cause hypoxia and acidosis, which influence recovery from anaesthesia and can have fatal results in critically ill patients with diminished opportunities for compensation (7).

The aim of the present study was to trace the changes in acid-base and blood gas parameters during the progress of 3 different methods of anaesthesia in dogs.

Materials and Methods

The investigation was carried out in 22 male and female mongrel dogs, aged 3-4 years, with a mean body weight of 17.4 ± 2.7 kg, divided into 3 groups.

The dogs from the first group (n = 8) were subjected to standard halothane anaesthesia. Premedication was performed with atropine sulphate (Sopharma, Bulgaria; 0.02 mg/kg SC) and acepromazine hydrochloride (Combistress", Kela, Belgium, 0.1 mg/kg IM) after 10 min. Thiopental natricum (Biochemie GmbH, Austria, 10 mg/kg IV) was given 20 min later to induce anaesthesia. After endotracheal intubation this was maintained with halothane (Narcotan", Leciva, Czech Republic), inhalation of 2.5-3% and an oxygen flow of 2.5-3 l/min. A Fluotec Mark III halothane vaporiser and semi-closed rebreathing circuit were used.

The dogs in the second group (n = 7) received balanced anaesthesia using the same premedication and induction techniques, but the maintenance of anaesthesia was performed with pancuronium bromide (Pavulon®, Troyapharm, Bulgaria, 0.06 mg/kg IV) by repeating the half of the initial dose after every single spontaneous respiratory movement; with fentanyl citrate (Stobium®, The Chemical Pharm. & Research Institute, Bulgaria, 0.01 mg/kg IV) every 30 min, halothane (0.5%) and controlled ventilation with a mean respiratory volume of 340 ml, a respiratory rate of 12/min, and an oxygen flow of 2.5-3 l/min. At the end of anaesthesia nivalin P (Sopharma, Bulgaria, 10 mg IV) was administered after 4

spontaneous respiratory movements to reverse the neuromuscular blockade.

The anaesthesia in these 2 groups was maintained in the third degree of the third stage by tracing the following reflexes: central position of the eye globe, moderately dilated pupils, and the absence of palpebral, corneal, patellar, anal or pharyngeal reflexes. The end of anaesthesia was assumed to be when the dog adopted a sternal recumbancy position.

The dogs in the third group (n = 7) received lumbosacral epidural anaesthesia after the same premedication. In order to achieve motor and sensory block up to L1 lidocaine solution (Sopharma, Bulgaria, 2%, 0.3 ml/kg) was administered into the epidural space between L7 and S1 using a 22-gauge 6.35 cm Tuochy needle.

In all groups 0.9% physiological solution (5 ml/kg per h IV) was administered to prevent the hypotension accompanying all anaesthetic methods. Anaesthesia was maintained up to 120 min in the 3 groups.

Arterialised capillary blood samples were collected anaerobically in heparinised tubes by inserting a pin through the nose pads of all animals as follows: prior to anaesthesia (baseline), at the time of premedication (30 min), during the deep stage of anaesthesia (120 min), at the end of anaesthesia (about 140 min) and the next day (24 h). The assay was carried out immediately after blood samples were obtained. A blood gas analyzer, an AVL radiometer, was used to determine the following parameters:

- pH
- partial pressure of carbon dioxide – $PaCO_2$, mmHg
- partial pressure of oxygen – PaO_2, mmHg
- actual bicarbonate contents – HCO_3, mmol/l
- total carbon dioxide – TCO_2, mmol/l
- actual base excess – ABE, mmol/l
- standard base excess – SBE, mmol/l
- standard bicarbonate contents – SBC, mmol/l
- oxygen saturation of haemoglobin – SAT, %
- total oxygen contents – O_2CT, vol%

Two-way ANOVA was used to detect statistically significant ($P < 0.05$) differences of mean values from the baseline and between groups.

Results

Alterations in parameters of acid-base status in dogs subjected to halothane anaesthesia revealed the development of primary respiratory acidosis without metabolitic compensation, which was best manifested at 120 min (Table 1). The statistically significant changes in acid-base parameters during this period demonstrate this. The arterial pH decreased (7.199 ± 0.046, $P < 0.01$) in comparison with the baseline (7.316 ± 0.006). At the same time, the respiratory parameter $PaCO_2$ increased (58.03 ± 9.46, $P < 0.01$) compared to the initial period (33.98 ± 1.15), as did TCO_2 (from initial levels of 18.83 ± 1.24 it rose to 22.83 ± 1.41, $P < 0.05$), whereas the metabolitic components HCO_3, SBC, and ABE did not change, except for SBE (from the baseline level of -8.93 ± 0.38 it fell to -6.15 ± 0.65, $P < 0.05$). All the investigated parameters were normal in the following periods.

The dynamics of acid-base changes in the balanced group were similar to those in the halothane group (Table 2). During deep anaesthesia pH decreased to 7.126 ± 0.041 from the baseline level of 7.312 ± 0.008, $P < 0.001$; $PaCO_2$ rose to 54.64 ± 7.76 in comparison with the initial levels of 33.93 ± 1.28, $P < 0.05$; ABE and SBE changed in a negative direction - from baseline levels of -7.94 ± 1.13 and -8.20 ± 1.05 to -11.47 ± 1.53, $P < 0.05$ and -11.13 ± 1.64, $P < 0.05$, respectively; SBC fell to 15.13 ± 1.22 compared to the initial period 17.80 ± 0.59, $P < 0.05$; and HCO_3 and TCO_2 remained unchanged. In the next periods (140 min and 24 h) all parameters returned to the baseline levels with the exception of pH, which was lower (7.241 ± 0.028, $P < 0.05$) during the recovery period compared to the initial period.

In the epidural group, no statistically significant alterations in acid-base status were observed during the investigated periods (Table 3).

The previously mentioned differences in acid-base metabolism in the 3 experimental groups were also manifested by comparative assessments between them (Table 4).

The changes in blood gas parameters (PaO_2, SAT, O_2CT) were again observed during the deep stage of halothane anaesthesia (Table 1). An elevation was observed in PaO_2 (141.73 ± 10.98 compared with initial levels of 99.58 ± 6.36, $P < 0.05$) as well as an increase

Table 1. Alterations in parameters of acid-base status and blood gas exchange in dogs subjected to halothane anaesthesia (n = 8).

Parameter	Baseline	30 min	120 min	140 min	24 hour
pH	7.316 ± 0.006	7.350 ± 0.017	7.199 ± 0.046**	7.297 ± 0.018	7.334 ± 0.006
$PaCO_2$, mmHg	33.98 ± 1.15	33.08 ± 0.9	58.03 ± 9.46**	39.63 ± 2.05	36.03 ± 1.63
PaO_2, mmHg	99.58 ± 6.36	95.23 ± 3.06	141.73 ± 10.98*	99.80 ± 8.53	106.80 ± 8.52
HCO_3, mmol/l	17.78 ± 1.15	17.78 ± 1.30	21.05 ± 1.10	18.83 ± 0.31	18.83 ± 0.84
TCO_2, mmol/l	18.83 ± 1.24	18.73 ± 1.36	22.83 ± 1.41*	20.03 ± 0.33	19.90 ± 0.88
ABE, mmol/l	-6.76 ± 0.55	-6.08 ± 0.61	-7.30 ± 0.38	-6.73 ± 0.38	-5.03 ± 0.63
SBE mmol/l	-8.93 ± 0.38	-7.30 ± 1.01	-6.15 ± 0.65**	-7.70 ± 0.58	-7.80 ± 0.51
SBC, mmol/l	18.68 ± 1.02	19.38 ± 0.29	19.08 ± 0.18	19.20 ± 0.30	20.03 ± 0.54
SAT, %	95.03 ± 0.97	95.60 ± 0.75	97.83 ± 0.59*	96.75 ± 0.48	95.63 ± 1.42
O_2CT, vol%	14.00 ± 2.21	13.95 ± 1.91	15.53 ± 1.19	16.68 ± 0.78	18.28 ± 2.25

* P < 0.05; ** P < 0.01 compared to baseline

Table 2. Alterations in parameters of acid-base status and blood gas exchange in dogs subjected to balanced anaesthesia (n = 7).

Parameter	Baseline	30 min	120 min	140 min	24 hour
pH	7.312 ± 0.008	7.332 ± 0.009	7.126 ± 0.041***	7.241 ± 0.028*	7.314 ± 0.010
$PaCO_2$, mmHg	33.93 ± 1.28	33.96 ± 1,11	54.64 ± 7.76*	40.49 ± 3.01	33.61 ± 0.91
PaO_2, mmHg	89.83 ± 8.68	91.19 ± 1.64	446.70 ± 52.94***	203.84 ± 85.75	80.91 ± 2.92
HCO_3, mmol/l	16.46 ± 0.92	17.27 ± 0.63	17.07 ± 1.62	16.71 ± 0.45	16.40 ± 0.55
TCO_2, mmol/l	17.37 ± 0.94	18.23 ± 0.66	18.44 ± 1.77	18.00 ± 0.50	17.36 ± 0.58
ABE,mmol/l	-7.94 ± 1.13	-7.03 ± 0.74	-11.47 ± 1.53*	-9.77 ± 0.68	-8.20 ± 0.58
SBEmmol/l	-8.20 ± 1.05	-7.20 ± 0.72	-11.13 ± 1.64*	-9.46 ± 0.60	-8.33 ± 0.59
SBC,mmol/l	17.80 ± 0.59	18.56 ± 0.59	15.13 ± 1.22*	16.51 ± 0.51	17.66 ± 0.44
SAT,%	91.76 ± 1.96	94.60 ± 0.34	99.49 ± 0.27**	93.00 ± 2.50	91.36 ± 1.29
O_2CT,vol%	13.49 ± 2.43	12.10 ± 0.83	11.90 ± 2.08	13.69 ± 1.23	13.67 ± 1.49

* P < 0.05; ** P < 0.01 compared to baseline

Table 3. Alterations in parameters of acid-base status and blood gas exchange in dogs subjected to epidural anaesthesia (n = 7).

Parameter	Baseline	30 min	120 min	140 min	24 hour
pH	7.309 ± 0.017	7.316 ± 0.016	7.302 ± 0.015	7.325 ± 0.015	7.349 ± 0.016
$PaCO_2$, mmHg	31.94 ± 0.83	30.61 ± 1.08	32.10 ± 0.96	31.04 ± 0.94	29.50 ± 1.11
PaO_2, mmHg	99.46 ± 3.11	105.46 ± 3.28	94.77 ± 5.12	106.23 ± 6.74	101.30 ± 4.64
HCO_3, mmol/l	15.31 ± 0.61	14.86 ± 0.62	15.41 ± 0.75	15.86 ± 0.75	15.69 ± 0.57
TCO_2, mmol/l	16.03 ± 0.50	15.71 ± 0.63	16.37 ± 0.77	16.81 ± 0.77	16.51 ± 0.59
ABE, mmol/l	-8.96 ± 0.83	-9.31 ± 0.84	-9.54 ± 0.90	-8.77 ± 0.90	-7.93 ± 0.69
SBE mmol/l	-9.37 ± 0.77	-9.71 ± 0.78	-9.64 ± 0.86	-8.91 ± 0.87	-8.53 ± 0.72
SBC, mmol/l	17.24 ± 0.59	16.90 ± 0.65	16.63 ± 0.70	17.26 ± 0.69	17.96 ± 0.52
SAT,%	94.49 ± 0.72	95.69 ± 0.44	94.77 ± 1.01	96.46 ± 0.46*	95.74 ± 0.60
O_2CT, vol%	18.19 ± 1.76	14.33 ± 1.45	12.94 ± 1.09*	13.74 ± 1.41*	16.51 ± 1.74

* P < 0.05; ** P < 0.01 compared to baseline

in saturation (97.83 ± 0.59 versus the baseline 95.03 ± 0.97, $P < 0.05$). Oxygenation was better in balanced anaesthesia (Table 2) during the same period than it was in halothane anaesthesia. PaO_2 increased from an initial 89.83 ± 8.68 to 446.70 ± 52.94, $P < 0.001$ and SAT rose from 91.76 ± 1.96 to 99.49 ± 0.27, $P < 0.01$. O_2CT was unchanged.

Epidural anaesthesia was accompanied by a decrease in O_2CT at 120 min (12.94 ± 1.09, $P < 0.05$ in comparison with the initial values of 18.19 ± 1.76) and during the recovery period (13.74 ± 1.41, $P < 0.05$ versus baseline). SAT was elevated (96.46 ± 0.46, $P < 0.05$) at 140 min compared to the initial levels (94.49 ± 0.72).

A comparative estimation of blood gas metabolism between the groups showed statistically significant changes only in PaO_2 and SAT (Table 4).

Discussion

Most studies on changes in blood gases and acid-base status during general halothane anaesthesia indicate that respiratory or metabolitic acidosis develops, but it is disputable whether acidosis is compensated or not (2,4,5,8-10). Some researchers report the presence of hypoxia whereas others do not (3).

Our results indicated that halothane anaesthesia in dogs was accompanied by primary respiratory acidosis without metabolitic compensation (ABE and HCO_3 were not increased) and without concomitant hypoxia. The explanation for these changes can probably be associated with the direct suppressive effect of halothane on the respiratory centre of the brain. That results in a decrease in the excitation threshold to CO_2, a delay in respiratory rate, and an increased buffer blood capacity to CO_2; in addition, the lungs do not expel CO_2 adequately. Stuth et al. (11) investigated the reason for the halothane depressing effect on respiratory neurones by consecutively blocking off N-methil-D-aspartate (a positive mediator) receptors and gamma-aminobutyrate (negative mediator) receptors. According to their results, the suppressing effect of halothane was due to a decrease in synaptic excitation and not to an increase in inhibitory mechanisms. On the other hand, in conditions of hypercarbia the haemoreceptors in the glomus caroticus and arcus aortae play a significant role in breathing regulation. Their main irritant is a decrease in PaO_2, and hypoxaemia thus appears as the main stimulant of the respiratory centre. The results of the present study did not show any evidence of hypoxia, meaning that the second regulatory mechanism for the amplification of ventilation was suppressed. In this case oxygen therapy may be disastrous. Other investigators claim that halothane selectively disturbs the response of haemoreceptors in the glomus caroticus to hypoxia (12). Therefore, even if halothane does not cause hypoxia this anaesthetic increases it when hypoxia is already present.

Table 4. Comparative characteristics of alterations in blood gas and acid-bas parameters between groups.

Parameter	Baseline	30 min	120 min	140 min	24 hour
pH			♦•••		
$PaCO_2$, mmHg			♦♦••	♦♦••	♦♦♦•
PaO_2, mmHg		♦••	♥♥♥•••		♥♥•
HCO_3, mmol/l		♦♦•	♦♦♥	♦♦♦♥♥	♦♦♦♥♥
TCO_2, mmol/l		♦♦•	♦♦♦♥	♦♦♦♥	♦♦♦♥♥
ABE, mmol/l		♦	♥♥	♦♥♥	♦♦♥♥♥
SBE mmol/l		♦•	♦♥♥		
SBC, mmol/l		♦	♦♥♥	♦♥♥	♦♥
SAT, %			♦♦•••		♥♥••
O_2CT, vol%					

♦ $P < 0.05$; ♦♦ $P < 0.01$; ♦♦♦ $P < 0.001$; between halothane and epidural anaesthesia
♥ $P < 0.05$; ♥♥ $P < 0.01$; ♥♥♥ $P < 0.001$; between halothane and balanced anaesthesia
• $P < 0.05$; •• $P < 0.01$; ••• $P < 0.001$; between balanced and epidural anaesthesia

It is known that nondepolarising miorelaxants reduce oxygen consumption in the body. It was later determined that in hypoxic conditions they change cardiovascular function as an increase in pulmovascular pressure, increase the blood flow to the brain and the heart and thus maintain a normal oxygen supply to the vital organs (13). The hypoxia itself decreases, total tissue oxygen consumption by 40% independent of whether an animal is anaesthetised or not. Therefore, the influence of miorelaxants is upon tissue oxygen supply, which follows the changes in tissue blood flow. The defensive mechanisms against hypoxia result in redistribution of the blood to the brain and the heart and are activated by miorelaxants. Probably because of this property, balanced anaesthesia was not accompanied by hypoxia in our study although hypercarbia was manifested with reference to the blocked respiratory muscles.

Pancuronium, used in our scheme of balanced anaesthesia, favours the oxygen supply to tissues, as shown by the high levels of PaO_2 and low levels of $PaCO_2$ in studies by Runkle and Bancalari (1). Du et al. (14) maintain that pancuronium does not change pulmonary arterial pressure in healthy lungs, but that when the lungs are damaged and hypoxaemia is present this neuromuscular blocking agent results in increased pulmonary arterial pressure, increased cardiac output and pulmonary vascular resistance in a sympathetic manner. Pancuronium contributes to the redistribution of the blood and relaxing of renal and coronary arteries not only by sympathetic action but also by mediators – the releasing of prostacyclin by subendothelial tissue (15).

With regard to epidural anaesthesia, this does not influence the acid-base balance and blood gases (6). However, it is unacceptable to discuss the effects on respiratory and cardiovascular functions separately. The application of local anaesthetics into the epidural space induces a sympathetic blockade because of the presence of many sympathetic nerve fibres. The hypotensia expected as a result of that was not clearly demonstrated, which could be explained by a contrary compensatory response of the body. The studies by Peters et al. (16-17) tend in this direction. They determined that epidural anaesthesia did not completely abolish a normal cardiovascular response to hypoxaemia. They also specified that this was not due to a release of noradrenaline, adrenaline and rennin, but to enhanced secretion of vasopresine. At the same time, epidural anaesthesia preserves the respiratory response to hypoxaemia. Other studies confirm this and reveal that during epidural anaesthesia only vasopresine is specifically activated for the maintenance of adequate blood pressure whereas endothelin remains unchanged (18).

In conclusion:

1. Halothane and balanced anaesthesia of the same duration in dogs resulted in similar acid-base and blood gas changes,
2. During the deep stages, halothane and balanced anaesthesia were accompanied by primary respiratory acidosis without metabolitic compensation and with signs of overoxygenation, probably because of the applied respiratory regimen. These changes returned to the baseline only during the recovery period,
3. Epidural anaesthesia was not accompanied by any alterations in blood gases and acid-base status.

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