

Influence of hypothermia and acidosis upon some indices of blood coagulation in three schemes of anaesthesia in dogs

Galina Petkova Simeonova*, Dinco Nedev Dinev, and Irina Ivanova Todorova

Department of Veterinary Surgery, Faculty of Veterinary Medicine, Trakia University, Stara Zagora, Bulgaria

SIMEONOVA, G. P., D. N. DINEV, I. I. TODOROVA: Influence of hypothermia and acidosis upon some indices of blood coagulation in three schemes of anaesthesia in dogs. Vet. arhiv 75, 233-242, 2005.

ABSTRACT

The aim of the present study was to investigate the influence of hypothermia and acidosis on blood coagulation in different anaesthesia protocols. Our experiment was performed with 28 dogs, divided into four groups: three experimental submitted to inhalation, balanced, and epidural anaesthesia and one control. In all animals blood pH, core body temperature and some principal parameters of blood coagulation (platelet count, activated partial prothrombine time-APPT, prothrombine time-PT and plasma fibrinogen concentrations) were investigated. The dynamics included five periods; prior to anaesthesia (0th minute), at the time of pre-medication (30th minute), during deep anaesthesia (120th minute), after recovery (about 140th minute), and on the next day (24th hour). The results indicated that the most significant were changes in balanced anaesthesia. APPT was shortened after recovery from balanced anaesthesia (14.1 ± 0.9 seconds, $P < 0.05$) and on the next day (14.5 ± 0.7 , $P < 0.05$) compared to the initial value (16.1 ± 0.5). The most pronounced acidosis in this group was recorded during deep anaesthesia (7.126 ± 0.041 , $P < 0.001$) and after recovery (7.241 ± 0.028 , $P < 0.05$) by comparison with the baseline (7.312 ± 0.008). Parameters of blood coagulation in inhalation anaesthesia group were unchanged. Statistically significant alterations in blood pH were observed only during the deep anaesthesia stage (7.199 ± 0.049 , $P < 0.01$) compared to the beginning (7.316 ± 0.006). Epidural anaesthesia did not result in blood pH and coagulation changes. In this group an increase in fibrinogen concentrations at 24th hour (3.7 ± 0.2 , $P < 0.05$) were found, compared to the baseline (3.1 ± 0.2), which was probably due to the intervention. In the three groups the core body temperature was decreased at the 120th minute and 140th minute. In conclusion, balanced anaesthesia activated blood coagulation at the 140th minute and 24th hour, which was manifested by a shortening in APPT at these periods. Hypothermia and acidosis accompanying balanced and inhalation anaesthesia groups, as well as hypothermia in epidural anaesthesia had no influence upon blood coagulation parameters.

Key words: anaesthesia, blood coagulation, hypothermia, acidosis, dog

* Contact address:

Dr. Galina Petkova Simeonova, Department of Veterinary Surgery, Faculty of Veterinary Medicine, Trakia University, Stara Zagora - 6000, Bulgaria, Phone: +359 42 2801 2697, E-mail: gpsimeonova@uni-sz.bg; galinavet@abv.bg

Introduction

There are many studies in emergency and critical care medicine which claim that the reason for death in severely ill patients (shock, trauma, endotoxaemia, etc.) is the deadly triad – hypothermia, acidosis, coagulopathy (MIKHAIL, 1999; EDDY et al., 2000; LAPOINTE and VON RUEDEN, 2002). These cases are challenging for anaesthesiologists in that these symptoms often develop in anaesthetized animals and therefore such anaesthesia may be noxious to the patients.

Different anaesthetics and schemes influence blood coagulation in different ways. Surgery also changes blood coagulation parameters. According to DINEV and HUBENOV (2002) and DINEV and ANDONOVA (2003) a synergism is presented between effects of both anaesthesia and surgery on the coagulation system. The authors reveal activation of this adaptive system, demonstrated by shortening of activated partial prothrombime time (APPT), prothrombime time (PT), and an increase in thromboxane-B2 concentrations. This was more pronounced in halothane anaesthesia and surgery than in anaesthesia without surgery. It is known that certain anaesthetics, including halothane, suppress platelet aggregation in vitro (DALSGARD-NIELSON et al., 1981) although others can induce haemorrhagic complications in animals suffering from primary disturbances in platelet aggregation (BARR et al., 1992). Results reported by GO-SHINE HUANG et al. (2002) are contradictory to the foregoing. They consider that neither general anaesthesia using isoflurane nor epidural with bupivacaine applied in arthroscopic surgery have any effect upon blood coagulation. The explanation is probably that unlike halothane, isoflurane does not influence platelet aggregation in vitro (DOGAN et al., 1999). Another study demonstrates that more invasive operation was necessary to result in stress-related release of catecholamines, which facilitate platelet aggregation (VAN VLIET et al., 1985). Stress and fever may direct blood coagulation in another way by increasing release of tissue plasminogen activator by endothelial cells (TROY, 1988). Plasmin acts only on fibrin clot and does not prevent circulation. PAROLARI et al. (1999) investigated in vitro and ex vivo the effects of different anaesthetics (droperidol, fentanyl, succinylcholine, pancuronium, thiopental and diazepam) on platelet function. They found that only thiopental suppresses platelet aggregation. Local anaesthetics used in spinal anaesthesia suppress haemocoagulation and platelet aggregation as well, but only in plasma concentrations much higher than clinically accepted (BORG and MODIG, 1985; TOBIAS et al., 1996). Other surveys prove that epidural anaesthesia used in patients with atherosclerotic changes in cardiac valves decreases blood hypercoagulation (TUMAN et al., 1991). The effects of epidural in comparison with balanced anaesthesia upon blood coagulation in conditions of surgery with high risk of thromboembolism were established. MODIG et al. (1981, 1983) ascertained the advantages of epidural anaesthesia. It was their opinion that it was due to more intensive circulation of legs, more effective fibrinolysis, and stabilizing effect of local anaesthetics on platelets, neutrophiles and endothelial cells.

It is known that most anaesthetics cause hypothermia and acidosis, but the question is how they affect haemostasis and whether anaesthetics are able to induce the deadly triad.

The aim of the present study was to evaluate the influence of core body temperature and blood pH on some principal parameters of haemocoagulation in halothane, balanced, and epidural anaesthesia in dogs.

Materials and methods

The experiment was performed on 28 mongrel (mixed breed) dogs equalized according to weight (17.4 ± 2.7 kg) and age (3-4 years). Animals were divided into four groups: three experimental groups were submitted to inhalation, balanced and epidural anaesthesia, and one control.

Premedication was the same in all three experimental groups and consisted of an application of atropine sulphate (Sopharma - Bulgaria; $0.02 \text{ mg.kg}^{-1} \text{ s/c}$) and acepromazine maleate (Combistress[®], Kela - Belgium, $0.1 \text{ mg.kg}^{-1} \text{ i/m}$) after 10 minutes.

Induction in the inhalation and balanced anaesthesia groups was achieved using thiopental sodium (Biochemie GmbH - Austria, $10 \text{ mg.kg}^{-1} \text{ i/v}$) injected as 2.5% solution 20 minutes after acepromazine.

Group I. Inhalation anaesthesia ($n = 8$) - after endotracheal intubation anaesthesia was maintained with 2.5-3% halothane (Narcotan[®], Leciva - Czech Republic) and oxygen flow of $2.5\text{-}3 \text{ L.minutes}^{-1}$. Fluotec Mark III halothane vaporiser and semi-closed re-breathing circuit were used.

Group II. Balanced anaesthesia ($n = 7$) - for maintenance pancuronium bromide (Pavulon[®], Troyapharm, Bulgaria, $0.06 \text{ mg.kg}^{-1} \text{ i/v}$) was used by repeating half of the initial dose after every single spontaneous respiratory movement; fentanyl citrate (Stobium[®], The Chemical Pharmaceutical Research Institute, Sofia, Bulgaria, $0.01 \text{ mg.kg}^{-1} \text{ i/v}$) every 30 minutes; halothane (0.5%) and controlled ventilation with mean respiratory volume 340 ml, respiratory rate 12 minutes^{-1} , oxygen flow $2.5\text{-}3 \text{ l.minute}^{-1}$. At the end of anaesthesia, nivalin hydrochloride (Nivalin[®], Sopharma, Bulgaria, 10 mg i/v) was administered after four spontaneous respiratory movements to reverse the neuromuscular blockade.

Group III. Epidural anaesthesia ($n = 7$) - Lidocaine solution (Sopharma, Bulgaria, 2%, 0.3 ml.kg^{-1}) was administered into the epidural space between L7 and S1 using a 22-gauge 6.35 cm Tuohy needle.

A deep plane (III/3) in the two groups of general anaesthesia was maintained by controlling the following reflexes (central position of eye globe, median dilated pupils, lack of corneal, palpebral, patellar, anal, swallowing reflexes). The end of anaesthesia (recovery) was determined as the moment when the dog took a sternal recumbent position.

In all three groups 0.9% physiological solution (5 ml.kg⁻¹.hour⁻¹ i/v) was administered to prevent the hypotension accompanying all the used anaesthetic methods. Anaesthesia was maintained up to 120 minutes in the experimental groups.

Group IV. (Control, n = 6). Only blood samples were collected without any anaesthetic protocol to estimate the effect of blood loss upon the haemocoagulation.

Blood samples were collected from all 28 animals as follows: prior to anaesthesia (baseline), at the time of premedication (minute 30), during the deep stage of anaesthesia (minute 120 after the beginning), after recovery from anaesthesia (about minute 140 after the beginning) and on the next day (hour 24).

The following parameters common in all four groups were estimated: core body temperature (°C); pH - blood gas analyzer ABL Radiometer; haemocoagulation system - activated partial prothrombine time (APPT), prothrombine time (PT) in seconds - Amelung KC 1A Coagulation monitor, Germany, adjusted according to core temperature, plasma fibrinogen concentration - in gram per litre (g/L), Human Diagnostica, Germany, platelet count - in giga per litre (G/l), electronic method Serano plus 150, Germany

Data are presented as mean ± SEM. Two-way analysis of variance (ANOVA) was used to detect statistically significant differences. The effects of two factors were studied: time and anaesthetic protocol. Differences at P<0.05 level were considered as significant.

Results

The core body temperature was changed in the three scheme of anaesthesia. The most pronounced decrease was found at minute 120 (36.6 ± 0.2 , P<0.001) and after recovery (36.3 ± 0.5 , P<0.001) in the halothane anaesthesia group (Table 1) compared to the initial values (39.5 ± 0.2). Similar alterations were found in the balanced anaesthesia group (Table 2) and epidural anaesthesia (Table 3). The temperature in balanced anaesthesia was 37.0 ± 0.5 , P<0.05 at minute 120 and 36.5 ± 0.8 , P<0.01 at minute 140 in comparison with the beginning (39.1 ± 0.4). Its values in epidural group were 37.6 ± 0.5 , P<0.05 at minute 120 and 37.2 ± 0.4 , P<0.01 at minute 140 in relation to the baseline (39.0 ± 0.4).

The most significant acidosis was found in the deep stage of balanced anaesthesia (7.126 ± 0.041 , P<0.001) compared to the initial period (7.312 ± 0.008). In the next period acidosis was less pronounced (7.241 ± 0.028 , P<0.05) in comparison with the beginning. In the halothane anaesthesia group pH was statistically decreased only at minute 120 (7.199 ± 0.049 , P<0.01) in relation to the baseline (7.316 ± 0.006). Epidural anaesthesia did not affect blood pH.

The investigated parameters of blood coagulation were almost invariably in the three anaesthesia protocols during all periods, with exception of APPT which was shortened after recovery from balanced anaesthesia (14.1 ± 0.9 , P<0.05) and on the next day (14.5

Table 1. Core body temperature, blood pH, and blood coagulation parameters in dogs (n = 8) submitted to halothane anaesthesia

Parameter	0 minute	30 minute	120 minute	140 minute	24 hour
Core body temperature (°C)	39.5 ± 0.2	38.8 ± 0.3	36.6 ± 0.2***	36.3 ± 0.5***	39.3 ± 0.2
pH	7.316 ± 0.006	7.350 ± 0.017	7.199 ± 0.046**	7.297 ± 0.018	7.334 ± 0.006
Platelet count (G/L)	124.0 ± 37.2	102.8 ± 31.8	82.0 ± 23.8	99.4 ± 28.1	117.0 ± 35.7
Prothrombine time (PT) (sec)	13.4 ± 0.6	13.8 0.6	14.2 ± 0.6	13.6 ± 0.5	13.2 ± 0.6
Activated partial prothrombine time (APPT) (sec)	16.4 ± 0.7	16.1 ± 0.7	16.5 ± 0.7	14.8 ± 0.6	16.7 ± 0.4
Fibrinogen concentration (g/L)	3.4 ± 0.2	3.2 ± 0.2	3.0 ± 0.2	3.0 ± 0.2	3.6 ± 0.2

*P<0.05; ** P<0.01; ***P<0.001 compared to initial period (0 minute)

Table 2. Core body temperature, blood pH, and blood coagulation parameters in dogs (n = 7) submitted to balanced anaesthesia

Parameter	0 minute	30 minute	120 minute	140 minute	24 hour
Core body temperature (°C)	39.1 ± 0.4	38.6 ± 0.4	37.0 ± 0.5*	36.5 ± 0.8**	38.4 ± 0.8
pH	7.312 ± 0.008	7.332 ± 0.009	7.126 ± 0.041***	7.241 ± 0.028*	7.314 ± 0.010
Platelet count (G/L)	238.4 ± 51.3	176.6 ± 48.5	158.7 ± 43.4	175.3 ± 39.2	207.9 ± 43.9
Prothrombine time (PT) (sec)	14.0 ± 0.6	13.8 ± 1.0	14.6 ± 1.2	13.6 ± 0.5	12.6 ± 0.5
Activated partial prothrombine time (APPT) (sec)	16.6 ± 0.5	14.9 ± 0.5	15.0 ± 0.6	14.1 ± 0.9*	14.5 ± 0.7*
Fibrinogen concentration (g/L)	3.8 ± 0.2	3.6 ± 0.2	3.5 ± 0.2	3.4 ± 0.2	4.0 ± 0.3

*P<0.05; ** P<0.01; ***P<0.001 compared to initial period (0 minute)

Table 3. Core body temperature, blood pH, and blood coagulation parameters in dogs (n = 7) submitted to epidural anaesthesia

Parameter	0 minute	30 minute	120 minute	140 minute	24 hour
Core body temperature (°C)	39.0 ± 0.4	39.2 ± 0.4	37.6 ± 0.5*	37.2 ± 0.4**	38.9 ± 0.1
pH	7.309 ± 0.017	7.316 ± 0.016	7.302 ± 0.015	7.325 ± 0.015	7.349 ± 0.06
Platelet count (G/L)	189.3 ± 27.8	164.6 ± 26.2	147.3 ± 21.0	154.1 ± 23.9	176.6 ± 27.9
Prothrombine time (PT) (sec)	13.6 ± 0.9	13.9 ± 0.8	12.8 ± 1.0	13.0 ± 1.0	13.0 ± 0.3
Activated partial prothrombine time (APPT) (sec)	17.9 ± 0.7	16.7 ± 0.8	16.5 ± 1.3	15.2 ± 0.9	16.5 ± 1.1
Fibrinogen concentration (g/L)	3.1 ± 0.2	3.0 ± 0.2	2.9 ± 0.2	3.0 ± 0.2	3.7 ± 0.2*

*P<0.05; ** P<0.01; ***P<0.001 compared to initial period (0 minute)

Table 4. Core body temperature, blood pH, and blood coagulation parameters in dogs (n = 6) not submitted to any anaesthesia – control group

Parameter	0 minute	30 minute	120 minute	140 minute	24 hour
Core body temperature (°C)	39.2 ± 0.3	39.5 ± 0.4	39.1 ± 0.3	39.2 ± 0.3	38.7 ± 0.2
pH	7.303± 0.018	7.322 ± 0.023	7.300 ± 0.017	7.332 ± 0.031	7.320 ± 0.020
Platelet count (G/L)	164.3 ± 40.0	157.7 ± 40.0	141.7 ± 36.0	132.7 ± 36.0	134.0 ± 33.4
Prothrombine time (PT) (sec)	13.7 ± 1.3	12.1 ± 0.6	15.0 ± 2.4	10.9 ± 0.8	16.7 ± 1.8
Activated partial prothrombine time (APPT) (sec)	17.2 ± 0.5	16.7 ± 0.5	15.0 ± 1.1	16.8 ± 1.9	18.0 ± 1.4
Fibrinogen concentration (g/L)	3.8 ± 0.4	3.7 ± 0.4	3.6 ± 0.3	3.5 ± 0.4	3.4 ± 0.2

*P<0.05; ** P<0.01; ***P<0.001 compared to initial period (0 minute)

± 0.7 , $P < 0.05$) compared to the beginning (16.1 ± 0.5). In epidural anaesthesia increased fibrinogen concentrations were found at hour 24 (3.7 ± 0.2 , $P < 0.05$) in comparison with the initial values (3.1 ± 0.2).

Blood loss (caused by taking blood samples) did not influence core body temperature, arterial pH, or investigated parameters of blood coagulation (Table 4).

Discussion

Blood coagulation can be activated or suppressed in different ways. The reasons for the development of coagulopathies are more numerous: metabolic, cardiovascular disorders, endotoxaemia, viraemia, massive blood transfusions and blood losses, immune complexes composition, drugs, cytokines, ions, ect. (VAN DER POLL et al., 1994; TODOROKI et al., 2000; LAPOINTE and VON RUEDEN, 2002).

One of the accompanying manifestations of anaesthesia is hypotension. It appears to be a potential protector of coagulation system and preserves from consumptive coagulopathy, as it decreases platelet aggregation without any influence on other coagulation factors (FELFERING-BOEHM et al., 2001). At the same time, the normothensive anaesthesia decreases platelet count, fibrinogen and antithrombin concentrations, shortens PT, but elongates APPT and TT.

All volatile and injectable anaesthetics, opioids, as well as spinal-used local anaesthetics increase the limits of temperature regulation from 0.2 to 4 °C by vasodilatation and vasoconstriction. The hypothermia accompanying all anaesthesia types markedly suppresses metabolism, phagocytosis, and blood coagulation (DE WAELE and VERMASSEN, 2002; SHIMOKAWA et al., 2003). This statement cannot be confirmed by our study because we found hypothermia in all three anaesthesia protocols which did not correlate with changes in blood coagulation. According to other investigators, platelet function is suppressed and enzyme reactions are delayed only when temperature falls below 34 °C, a critical point for the alterations referred to (WATTS et al., 1998). Temperature values below 35 °C elongate the time of thrombgeneration, which has the same effect as heavy deficiency of coagulation factors (GENTILELLO and PIERSON, 2001). In our investigation, temperature did not drop below this critical point.

The blood coagulation system is also influenced by acid-base status and gas exchange. Most anaesthetics are cardiovascular and respiratory depressants, so when hypoxia develops the APPT shortens (O'BRODOICH et al., 1984). Other investigators reveal that non-compensated respiratory acidosis in horses submitted to halothane anaesthesia and abdominal surgery contributes to increased inclination for hypercoagulation, manifested by shortening of APPT (DINEV and HUBENOV, 2002). In our study, the shortening of APPT in balanced anaesthesia did not coincide by time with acidosis development. In the

halothane anaesthesia group acidosis was well manifested but without any evidence of coagulation disorders. The increased fibrinogen concentration after epidural anaesthesia can be explained by the influence of intervention upon release of acute phase proteins, such as fibrinogen. Therefore, the increase fibrinogen concentration in epidural group was not provoked by changes in acid-base status because of the lack of concomitant acidosis. According to the results of ILERI et al. (1999), diabetic ketoacidosis results in platelet and endothelial activity, increased coagulation and decreased fibrinolytic activity. HARKE and RAHMAN (1980) reveal that shock-induced acidosis lasting more than 150 minutes results in significant elongation of APPT and suppressed activity of factor V.

In conclusion, balanced anaesthesia caused activation of blood coagulation after recovery and at hour 24 of anaesthesia, which was manifested by shortening of APPT at these periods, whereas such changes were not found in halothane and epidural groups. Hypothermia and acidosis accompanying balanced and halothane anaesthesia, as well as hypothermia in epidural anaesthesia, were not factors which change blood coagulation parameters.

References

- BARR, S., J. LUDDERS, A. LOONELY, R. GLEED, H. ERB (1992): Platelet aggregation in dog after sedation with acepromazine and atropine and during subsequent general anaesthesia and surgery. *Am. J. Vet. Res.* 53, 2067-2070.
- BORG, T., J. MODIG (1985): Potential anti-thrombotic effects of local anaesthetics due to their inhibition of platelet aggregation. *Acta Anesth. Scand.* 29, 739-742.
- DALSGARD-NIELSON, J., A. RISBO, P. SIMILKJAER (1981): Impaired platelet aggregation and increased bleeding time during general anaesthesia with halotane. *Br. J. Anaesth.* 53, 1039-1042.
- DE WAELE, J. J., F. E. VERMASSEN (2002): Coagulopathy, hypothermia and acidosis in trauma patients: the rationale for damage control surgery. *Acta Chir. Belg.* 102, 313-316.
- DINEV, D., M. ANDONOVA (2003): The effect of general anaesthesia and abdominal surgery upon plasma thromboxane B2 concentrations in horses. *Vet. Anaesth. Analg.* 30, 1-4.
- DINEV, D., H. HUBENOV (2002): Acid-base, blood gas and haemocoagulation changes in halothane-anaesthetized horses. Submitted to abdominal surgery. *Bul. J. Vet. Med.* 5, 241-251.
- DOGAN, I. V., E. OVALI, Z. ETI, A. YAYCI, F.Y. GORUS (1999): The in vitro effects of isoflurane, sevoflurane and propofol on platelet aggregation. *Anesth. Analg.* 88, 432-436.
- EDDY, V. A., J. A. Jr. MORRIS, D. C. CULLINANE (2000): Hypothermia, coagulopathy and acidosis. *Surg. Clin. North Am.* 80, 845-854.
- FELFERNIG-BOEHM, D., A. SALAT, C. KINSTNER, T. FLECK, M. FELFERNIG, O. KIMBERGER, A. ANDEL, M. R. MUELLER (2001): Influence of hypotensive and normotensive anaesthesia on platelet aggregability and hemostatic markers in orthognathic surgery. *Thromb. Res.* 103, 185-192.

G. P. Simeonova et al.: Influence of hypothermia and acidosis upon some indices of blood coagulation in three schemes of anaesthesia in dogs

- GENTILELLO, L. M., D. J. PIERSON (2001): Trauma critical care. *Am. J. Respir. Crit. Care Med.* 163, 604-607.
- GO-SHINE HUANG, JEN-HUEI CHANG, MEEI-SHYUAN LEE, CHIA-CHUN WU, SHU-PI LIN, SHINN-LONG LIN, CHIH-SHUNG WONG (2002): The effect of anesthetic techniques on hemostatic function in arthroscopic surgery: Evaluation by Thrombelastography. *Acta Anaesthesiol. Sin.* 40, 121-126.
- HARKE, H., S. RAHMAN (1980): Haemostatic disorders in massive transfusion. *Bibl. Haematol.* 46, 179-188.
- ILERI, N. S., Y. BUYUKASIK, S. KARAAHMETOGLU, D. OZATLI, N. SAYINALP, O. I. OZCEBE, S. KIRAZLI, O. MUFTUOGLU, S. V. DUNDAR (1999): Evaluation of the haemostatic system during ketoacidotic deterioration of diabetes mellitus. *Haemostasis* 29, 318-325.
- LAPOINTE, L. A., K. T. VON RUEDEN (2002): Coagulopathies in trauma patients. *A. A. C. N. Clin. Issues* 13, 192-203.
- MIKHAIL, J. (1999): The trauma triad of death: hypothermia, acidosis and coagulopathy. *A. A. C. N. Clin. Issues-American Association of Critical-care Nurses clinical issues* 10, 85-94.
- MODIG, J., A. HJELMSTEDT, B. SAHLSTEDT, E. MARIPUU (1981): Comparative influences of epidural and general anaesthesia on deep venous thrombosis and pulmonary embolism after total hip replacement. *Acta Chir. Scand.* 147, 125-130.
- MODIG, J., T. BORG, G. KARLSTROM, E. MARIPUU, B. SAHLSTEDT (1983): Thromboembolism after total hip replacement: role of epidural and general anesthesia. *Anesth. Analg.* 62, 174-180.
- O'BRODOICH, H. M., M. ANDREW, G. W. GRAY, C. COATES (1984): Hypoxia alters blood coagulation during acute decompensation in humans. *J. Appl. Physiol.* 56, 666-670.
- PAROLARI, A., D. GUARNIERI, F. ALAMANNI, T. TOSCANO, V. TANTALO, T. GHERLI, S. COLLI, F. FOIENI, V. FRANZE, M. STANGHELLINI, G. A. GIANOTTI, P. BIGIGLIOLI, E. TREMOLI (1999): Platelet function and anesthetics in cardiac surgery: an in vitro and ex vivo study. *Anesth. Analg.* 89, 26-31.
- SHIMOKAWA, M., K. KITITAGUCHI, M. KAWAGUCI, T. SAKAMOTO, M. KAKIMOTO, H. FURUYA (2003): The influence of induced hypothermia for hemostatic function on temperature-adjusted measurements in rabbits. *Anesth. Analg.* 96, 1209-1213.
- TOBIAS, M. D., M. A. PILLA, C. ROGERS, D. R. JOBES (1996): Lidocaine inhibits blood coagulation: Implications for epidural blood patch. *Anesth. Analg.* 82, 766-769.
- TODOROKI, H., S. NAKAMURA, A. HIGURE, K. OKAMOTO, S. TAKEDA, N. NAGATA, H. ITOH, K. OHSATO (2000): Neutrophils express tissue factor in a monkey model of sepsis. *Surgery* 127, 209-216.
- TROY, G. C. (1988): An overview of hemostasis. *Vet. Clin. North Am. Small Anim. Prac.* 18, 5-20.
- TUMAN, K. J., R. J. McCARTHY, R. J. MARCH, G. A. DeLARIA, R. V. PATEL, A. D. IVANKVICH (1991): Effects of epidural anesthesia and analgesia on coagulation and outcome after major vascular surgery. *Anesth. Analg.* 73, 696-704.

G. P. Simeonova et al.: Influence of hypothermia and acidosis upon some indices of blood coagulation in three schemes of anaesthesia in dogs

- VAN DER POLL, T., M. LEVI, C. E. HACK, H. TEN VATE, S. J. VAN DEVENTER, A. J. EERENBERG, E. R. GROOT, J. JANSEN, H. GALLATI, H. R. BULLER, J. W. TEN CATE, L. A. AARDEN (1994): Elimination of interleukin 6 attenuate coagulation activation in experimental endotoxemia in chimpanzees. *J. Exp. Med.* 179, 1253-125.
- VAN VLIET, K. J., G. L. SMIT, J. J. PIETERSE, H. J. SCHOONBEE, J. H. VAN VUREN (1985): A thrombelastographic study of the effect of stress on the blood coagulation in *Cyprinus carpio* (Cyprinidae) and *Oreochromis mossambicus* (Cichlidae). *Com. Biochem. Physiol.* 82, 23-27.
- WATTS, D. D., A. TRASK, K. SOEKEN, P. PERDUE, S. DOLS, C. KAUFMANN (1998): Hypothermic coagulopathy in trauma: effect of varying levels of hypothermia on enzyme speed, platelet function, and fibrinolytic activity. *J. Trauma* 44, 846-854.

Received: 21 November 2003

Accepted: 5 May 2005

SIMEONOVA, G. P., D. N. DINEV, I. I. TODOROVA: Utjecaj hipotermije i acidoze na neke pokazatelje zgrušavanja krvi u tijeku tri postupka anestezije u pasa. *Vet. arhiv* 75, 233-242, 2005.

SAŽETAK

Cilj rada bio je istražiti utjecaj hipotermije i acidoze na zgrušavanje krvi pri različitim postupcima anestezije. U pokus je uzeto 28 pasa podijeljenih u četiri skupine. Jedna od pokusnih skupina bila je izložena inhalacijskoj, druga uravnoteženoj, a treća epiduralnoj anesteziji, dok je četvrta bila kontrolna skupina. U svih životinja izmjeren je pH krvi, tjelesna temperatura i neki glavni pokazatelji zgrušavanja krvi (broj trombocita, aktivacijsko parcijalno protrombinsko vrijeme - APPV, protrombinsko vrijeme - PV i koncentracija fibrinogena u plazmi). Kretanje pretraživanih pokazatelja promatrano je u vremenu prije anestezije (0. minuta), u trenutku premedikacije (30. minuta), u tijeku duboke anestezije (120. minuta), nakon oporavka (oko 140. minute) i sljedećeg dana (nakon 24 sata). Najznačajnije promjene ustanovljene su u tijeku uravnotežene anestezije. Pri toj anesteziji APPV je u usporedbi s početnim vrijednostima ($16,1 \pm 0,5$) bio skraćen nakon oporavka ($14,1 \pm 0,9$ sekundi; $P < 0,05$) i sljedećeg dana ($14,5 \pm 0,7$; $P < 0,05$). U usporedbi s početnom vrijednošću ($7,312 \pm 0,008$), najizraženija acidoza u toj skupini bila je utvrđena u tijeku duboke anestezije ($7,126 \pm 0,041$; $P < 0,001$) i nakon oporavka ($7,241 \pm 0,028$; $P < 0,05$). Pokazatelji zgrušavanja krvi u skupini pasa izloženih inhalacijskoj anesteziji nisu bili promijenjeni. Statistički značajne promjene u pH krvi utvrđene su samo u tijeku duboke anestezije ($7,199 \pm 0,049$; $P < 0,01$) u usporedbi s početnim vrijednostima ($7,316 \pm 0,006$). Epiduralna anestezija nije uzrokovala promjene u pH i zgrušavanju krvi. U skupini pasa s tom anestezijom ustanovljeno je povećanje koncentracije fibrinogena nakon 24 sata ($3,7 \pm 0,2$; $P < 0,05$) u usporedbi s početnim vrijednostima ($3,1 \pm 0,2$), što se pripisuje samom zahvatu. Tjelesna temperatura bila je smanjena u 120. i 140. minuti u životinja pokusnih skupina. Zaključno se može reći da je uravnotežena anestezija potaknula zgrušavanje krvi u 140. minuti i nakon 24 sata, što se očitovalo skraćenjem APPV. Hipotermija i acidoza u skupinama s uravnoteženom i inhalacijskom anestezijom, kao i hipotermija u skupini s epiduralnom anestezijom, nisu utjecale na pokazatelje zgrušavanja krvi.

Ključne riječi: anestezija, zgrušavanje krvi, hipotermija, acidoza, pas
