

## Hemodynamic effects of epidural lidocaine vs lidocaine-adrenaline in dogs

**Dražen Vnuk<sup>1\*</sup>, Višnja Neseć-Adam<sup>2</sup>, Marko Pećin<sup>1</sup>, Andrija Musulin<sup>1</sup>,  
Nikša Lemo<sup>3</sup>, Nataša Brajenović<sup>4</sup>, Irena Brčić Karaconji<sup>4</sup>, Berislav  
Radišić<sup>1</sup>, Ozren Smolec<sup>1</sup>, and Marija Lipar<sup>1</sup>**

<sup>1</sup>*Clinic for Surgery, Orthopaedics and Ophthalmology, Faculty of Veterinary Medicine, University of Zagreb, Zagreb, Croatia*

<sup>2</sup>*Clinic for Anaesthesiology, Resuscitation and Intensive care, Sveti Duh General Hospital, Zagreb, Croatia*

<sup>3</sup>*Clinic of Internal Medicine; Faculty of Veterinary Medicine, University of Zagreb, Zagreb, Croatia*

<sup>4</sup>*Analytical Toxicology and Mineral Metabolism Unit, Institute for Medical Research and Occupational Health, Zagreb, Croatia*

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### ABSTRACT

Adrenaline is often added to local anaesthetic solutions to minimize and slow the systemic absorption of local anaesthetics, and thus reduce the possibility of adverse effects of these drugs and to prolong duration of action. The authors compared the effects of epidural anaesthesia with lidocaine and lidocaine-adrenaline mixture on hemodynamic changes and lidocaine serum concentrations in dogs. A total of 10 dogs of both sexes were included in study. The animals were randomly divided into one of the two groups: the lidocaine group (Group L, n5) receiving 2% lidocaine 3.3 mg/kg and the second group (Group LA, n = 5) receiving 2% lidocaine solution containing adrenaline in the same dose and volume. Lidocaine serum concentrations and hemodynamic changes associated with epidural block were recorded at 5, 15, 30, 45 and 60 minutes after local anaesthetic administration. The associated changes in respiratory rates (RR), partial pressure of carbon dioxide (PaCO<sub>2</sub>), pH and body temperature were also noted. Statistical analysis was performed for both inter-group and in-group comparisons of parameters. Heart rate and cardiac output increased significantly in Group LA compared to baseline values. Arterial and pulmonary artery pressure decreased significantly in Group L with significant differences between the groups for the changes in these parameters. The mean serum concentrations of lidocaine were lower in Group LA compared to Group L at all times after administration. Addition of adrenaline to epidural lidocaine is associated with positive effects of adrenaline in preventing hypotension. However, because

\*Corresponding author:

Dražen Vnuk, DVM, PhD, Clinic for Surgery, Orthopaedics and Ophthalmology, Faculty of Veterinary Medicine, University of Zagreb, Heinzelova 55, Zagreb, Croatia, Phone: +385 1 2390 388; E-mail: [dvnuke@vef.hr](mailto:dvnuke@vef.hr)

of the potential hazards of increase in heart rate and cardiac output especially in elderly and critical ill patients, the routinely use of epidurally adrenaline should be reconsidered in patients with high hemodynamic risk.

**Key words:** lidocaine, lidocaine-adrenaline combination, epidural anaesthesia

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### **Introduction**

Due to its simplicity, safety and efficacy, epidural anaesthesia is one of the most commonly used central neuraxial block techniques for control of intraoperative and postoperative pain. Diagnostic, obstetrical and minor surgical procedures in the rear limbs, perineum, pelvis and tail are the main indications for the use of epidural anaesthesia. It provides excellent analgesia, and improves postoperative outcome (BRODNER et al., 1999). Furthermore, epidural anaesthesia is increasingly applied during high-risk surgery (VAISANEN et al., 1998; PIPER et al., 2000) as well as in critically ill animal (JONES, 2001). Many beneficial aspects of epidural anaesthesia have been reported, including better suppression of surgical stress and better postoperative pain control (PIPER et al., 2000).

The most frequent disadvantages of epidural administration of local anaesthetics are motor impairment, sympathetic block, hypotension and local anaesthetic toxicity (ROELANTS et al., 2005). In an attempt to diminish these undesirable side-effects the adjuvant drugs have been routinely mixed with local anaesthetic solution. FÖRSTER and ROSENBERG (2003) described clinically useful adjuvants in epidural anaesthesia. Vasoconstrictor drugs, in particular adrenaline, are commonly used as adjuvants in solutions of local anaesthetics to slow the systemic absorption, leading to increased neuronal uptake of local anaesthetics. The blood concentration of local anaesthetic agents following regional anaesthesia is determined by the absorption, tissue redistribution, metabolism and excretion characteristics (TUCKER, 1986).

The aim of this study was to observe the impact of epidurally administered lidocaine and lidocaine combined with adrenaline on hemodynamic variables and lidocaine serum concentrations in dogs.

### **Materials and methods**

A total of 10 dogs of both sexes, mean age  $6.3 \pm 3.5$  years and mean weight  $34.7 \pm 10.7$  kg were included in this study. The dogs were hospitalized at the Surgery Clinic for lower abdominal or extremity surgery. Diagnosis and type of surgery are listed in Table 1. The study protocol was approved by the institutional ethics committee. Informed consent was obtained from all dog owners. They were informed that the blood sample would be taken painlessly by intravenous cannula under general anaesthesia and that invasive hemodynamic monitoring would be performed.

The health status of each dog was confirmed by physical examination, complete blood count and serum biochemical analyses just before the trials. Only ASA I-II patients were included in the study.

The animals were premedicated with 5 µg/kg i.m. of medetomidine (Domitor<sup>®</sup>, Pfizer, Finland) and 0.4 mg/kg i.m. of methadone (Heptanon<sup>®</sup>, Pliva, Croatia). An intravenous catheter (20 or 22 G) was inserted into both cephalic veins for continuous infusion and administration of additional drugs and for blood sampling. General anaesthesia was induced by combination of midazolam 0.25 mg/kg i.v., ketamine 2 mg/kg i.v. (Narketan<sup>®</sup>, Vetoquinol, Switzerland) and propofol 2-4 mg/kg i.v. (Propofol Abbott<sup>®</sup>, Abbott Laboratories, Spain). Additional doses of ketamine and propofol were added intermittently. Saline solution was administered through catheters at a rate of 10 mL/kg/h (Braun Vista Basic infusion pump) before and during the procedures. Cephazoline 20 mg/kg (Kefzol<sup>®</sup>, Eli Lilly, Canada) was administered intravenously for preoperative antibiotic prophylaxis.

Following this, the epidural insertion of needle was performed with the dogs lying in the left lateral position with the hind limbs flexed. The lumbosacral region (L7-S1) was shaved and surgically prepared with povidone iodide. An epidural needle was introduced through the gap between the L7-S1 interspaces. The epidural space was identified by the loss of resistance technique, using normal saline, and confirmed by the spontaneous inflow of a drop of saline placed in the needle hub. A 5 mL syringe was attached to the needle and aspiration was applied to confirm correct needle placement and to detect accidental intravascular or subarachnoid needle placement. Local anaesthetic or the combination of local anaesthetic and adrenaline was administered via this catheter afterwards. The effect of epidural administration was controlled by observing the dilation of the external anal sphincter, followed by relaxation of the tail and pelvic limbs. If these signs were not observed, animals were excluded from the study.

The animals were randomly divided into one of two groups. The first group received 2% lidocaine 3.3 mg/kg, 1 mL/6 kg (5 mL; i.e. 100 mg of lidocaine, Lidokain<sup>®</sup>, Belupo, Croatia) (Group L, n = 5) and the second group received 2% lidocaine solution containing adrenaline in the same dose and volume (2 mL; i.e. 40 mg of lidocaine plus 0.025 mg adrenaline, Lidokain-Adrenalin<sup>®</sup>, Belupo, Croatia) (Group LA, n = 5). Epidural anaesthesia was performed 10-15 minutes before skin incision.

Fluid-filled catheters were placed in the right metatarsal artery for systolic, diastolic and mean arterial pressure (SAP, DAP and MAP) monitoring via a pressure transducer. A 5.7 and 7.5 Fr balloon-tipped Swan-Ganz thermodilution catheter was placed into the pulmonary artery via the right external jugular vein, and its correct location was confirmed by detection of the typical pressure wave of this artery displayed on a monitor (Ultraview 1050, Spacelabs, USA). The catheter was connected to a pressure transducer to allow monitoring of pulmonary artery systolic, diastolic and mean pressure (PASP, PADP and PAMP). All transducers were zeroed to the midchest of the animal. Calibrations were checked before each set of measurements. Cardiac output (CO) was determined in

triplicate by injecting 5 mL of cold, heparinised saline solution (4 °C). Heart rate was measured by a electrocardiogram, which was also continuously monitored. Respiratory rate was measured by a capnograph, and body temperature oesophageally.

Before epidural administration, blood samples were drawn from the femoral artery for gas analysis using a blood gas analyser (ABL5, Radiometer, Copenhagen, Denmark) and a venous blood sample was taken using a cephalic intravenous catheter from the contralateral limb for serum lidocaine concentration measurement. After baseline measurements were taken, the hemodynamic data (heart rate, arterial blood pressures, pulmonary artery pressure, CO) respiratory rate and body temperature were then recorded at 5, 15 30, 45 and 60 minutes after epidural administration of the study drug.

Serum lidocaine concentrations were measured by gas chromatography-mass spectrometry. Prior to analysis, solid-phase extraction was performed using Bond Elut Certify columns (Varian, Harbor City, CA, USA) to achieve high purity of serum. The analysis was performed using a Varian 3400 CX GC with Saturn ion trap mass spectrometer. The chromatographic column was DB-5MS (5% phenyl-95% dimethylpolysiloxane, 30 m, 0.25 mm i.d., 0.25 µm film thickness; J&W Agilent Technologies; Santa Clara, SAD). The following ions were used for monitoring:  $m/z$  86,  $m/z$  234 and  $m/z$  120. The ion underlined was used for quantitation.

This method for lidocaine assay is accurate, highly specific and precise. The limit of detection was 5 ng/mL. The inter- and intra-assay coefficient of variation was <10%.

All quantitative data with normal distribution between and within the groups were analyzed statistically using the parametric Student's t-test for independent and dependent samples. The quantitative data with abnormal distribution were tested using the nonparametric Mann-Whitney U-test for inter-group comparison and Wilcoxon test for in-group comparison. Statistical significance was accepted at the level of  $P \leq 0.05$ .

## Results

Patient characteristics and surgical procedures performed were similar in the two study groups with the exception of the male:female ratio. It is noteworthy that in both groups the patients were predominantly male (Table 1). The respiratory effects and body temperature were not statistically significant between study groups (Table 2).

Haemodynamic data are shown in Table 3. Although baseline values were not significantly different between the groups, at 15, 30, 45 and 60 min in Group LA heart rates were significantly higher than those of Group L. When in-group analysis was performed, it was observed that heart rates values increased at 5, 15, 30, 45 and 60 min in Group LA compared to the baseline without a significant difference from baseline measurements in Group L.

Table 1. Patient characteristics

Breed	Sex	Age (y)	Body mass (kg)	Diagnosis	Surgery
Lidocaine group					
Labrador retriever	M	2	39	Scrotal oedema	Scrotal ablation
Mixed	M	3	31	Rupture of cruciate ligament	Lateral suture
Mixed	M	3	20	Hip joint luxation	Single-suture technique for a dislocated hip
German shepherd dog	F	13	34	Pyometra	Ovariohysterectomy
German boxer	M	5	37	Femoral fracture	Bone plate fixation
Lidocaine plus adrenaline groups					
Bull mastiff	M	6	51	Inguinal neoplasia	Surgical excision of neoplasia
German shorthaired pointer	M	8	32	Penile neoplasia	Partial penile amputation
German shepherd dog	M	9	33	Colonic neoplasia	Biopsy of colonic neoplasia
Newfoundlander	M	10	51	Perianal adenoma	Surgical excision of neoplasia
Mixed	F	4	19	Tibial fracture	Bone plate fixation

After epidural administration of lidocaine alone, SAP, DAP and MAP decreased slightly with a significant effect only on DAP and MAP. Diastolic pressure showed a significant reduction observed up to 15 min. Parallel to that, mean arterial pressure was also found to be lower at 15, 30, 45 and 60 min of observation compared to the baseline. In Group LA, SAP, DAP and MAP did not decrease at any point. There were no significant differences between the groups for SAP, DAP and MAP values at 5 and 15 min after the administration of epidural anaesthesia. The change reached significant value at 30 min, then increased to a non-significant level at 45 and 60 min during the procedures.

There were no significant differences in PASP and PADP between the groups at baseline and 5 min after administration of epidural anaesthesia, but the change reached significant value at 15 and 30 min, then increased to non-significant level at 45 and 60 min. When groups were evaluated individually, PASP and PAMP decreased significantly at 15, 30 and 45 min during epidural anaesthesia in Group L compared to the baseline value, whereas in Group LA there were no significant differences in PASP, PADP and PAMP at any point of observation compared to the baseline.

Table 2. Comparison of body temperature, respiratory rate, pH and PaCO<sub>2</sub> between groups at different time intervals

Variables	Baseline	Time after administration of epidural anaesthesia, min				
		5	15	30	45	60
L group						
Body temperature (°C)	38.2 ± 0.3	38.3 ± 0.3	38.2 ± 0.3	38.4 ± 0.3	38.4 ± 0.3	38.4 ± 0.3
Respiratory rate (breaths/min)	16 ± 2	15 ± 3	15 ± 4	14 ± 2	15 ± 3	13 ± 3
pH	7.40 ± 0.08	7.41 ± 0.12	7.40 ± 0.12	7.42 ± 0.15	7.42 ± 0.19	7.42 ± 0.18
PaCO <sub>2</sub> (mmHg)	38.0 ± 1.3	37.6 ± 4.2	39.4 ± 3.7	35.6 ± 4.1	38.6 ± 4.8	36.4 ± 3.5
LA group						
Body temperature (°C)	38.7 ± 0.4	38.6 ± 0.3	38.6 ± 0.4	38.7 ± 0.4	38.8 ± 0.3	38.8 ± 0.3
Respiratory rate (breaths/min)	15 ± 3	16 ± 1	15 ± 2	15 ± 3	14 ± 2	14 ± 2
pH	7.42 ± 0.13	7.41 ± 0.18	7.41 ± 0.22	7.42 ± 0.25	7.43 ± 0.21	7.40 ± 0.18
PaCO <sub>2</sub> (mmHg)	36.4 ± 0.7	36.0 ± 3.5	35.0 ± 5.1	35.8 ± 4.5	34.8 ± 4.3	33.6 ± 4.2

Data are presented as mean ± SD of five dogs per group. There were no significant differences between groups

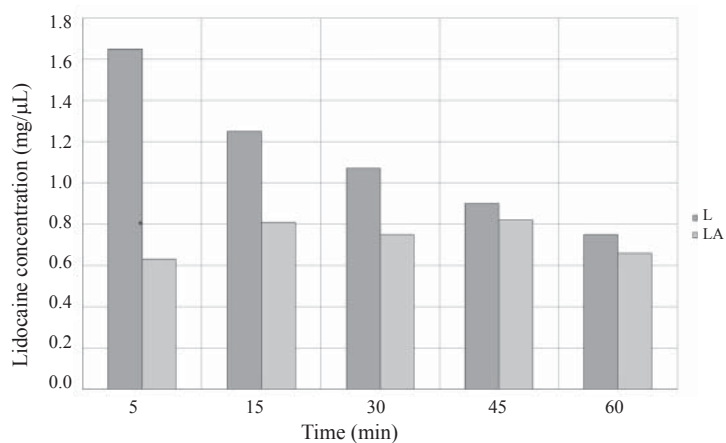


Fig. 1. Lidocaine serum concentrations in L and LA groups at different time intervals. In group L, lidocaine was administered alone. In group LA, lidocaine was administered in combination with adrenaline. Values are showed as mean ± SD.

Table 3. Comparison of hemodynamic changes between groups at different time intervals

Variables	Baseline	Time after administration of epidural anaesthesia, min				
		5	15	30	45	60
Lidocaine group						
HR beats/min	86.2 ± 9.5	94.8 ± 9.7	88.6 ± 8.4	82.4 ± 5.6	78.2 ± 6.3	76.8 ± 4.2
SAP (mm Hg)	164.6 ± 20.0	166.8 ± 13.0	166.8 ± 9.3	143.6 ± 9.4	151.0 ± 10.4	153.8 ± 11.0
DAP (mm Hg)	93.6 ± 10.1	93.0 ± 7.5	84.4 ± 10.8 <sup>a</sup>	75.2 ± 9.7 <sup>a</sup>	80.6 ± 9.1 <sup>a</sup>	82.4 ± 8.0 <sup>a</sup>
MAP (mmHg)	118.6 ± 8.7	113.4 ± 9.3	113.6 ± 11.4	98.6 ± 9.0 <sup>a</sup>	93.4 ± 6.9 <sup>a</sup>	106.0 ± 5.1 <sup>a</sup>
PASP (mm Hg)	26.6 ± 3.6	27.8 ± 2.6	19.4 ± 2.3 <sup>a</sup>	19.2 ± 2.7 <sup>a</sup>	20.8 ± 3.27 <sup>a</sup>	22.0 ± 4.3
PADP (mmHg)	11.2 ± 3.7	12.2 ± 3.2	9.8 ± 2.4	7.4 ± 2.8 <sup>a</sup>	10.8 ± 1.9	11.4 ± 1.5
PAMP (mm Hg)	17.6 ± 2.4	19.4 ± 1.6	14.2 ± 1.4 <sup>a</sup>	10.8 ± 1.3 <sup>b</sup>	13.6 ± 1.9	15.6 ± 1.8
Cardiac output (l/min)	2.0 ± 0.4	2.1 ± 0.4	2.2 ± 0.3	1.8 ± 0.2	1.8 ± 0.3	2.0 ± 0.2
Lidocaine + adrenaline						
HR beats/min	90.6 ± 9.8	104.4 ± 8.6 <sup>a</sup>	104.4 ± 12.6 <sup>a,c</sup>	114.8 ± 13.0 <sup>ad</sup>	106.2 ± 8.2 <sup>bd</sup>	102.4 ± 8.5 <sup>ad</sup>
SAP (mm Hg)	169.6 ± 20.3	160.2 ± 9.3	161.4 ± 9.7	161.6 ± 8.9 <sup>c</sup>	163.8 ± 11.0	160.6 ± 11.6
DAP (mm Hg)	88.4 ± 10.3	91.0 ± 7.4	90.2 ± 6.4	100.6 ± 7.5 <sup>d</sup>	83.4 ± 10.9	82.6 ± 10.2
MAP (mmHg)	112.4 ± 7.5	113.6 ± 8.6	115.6 ± 6.6	119.4 ± 7.0 <sup>c</sup>	101.4 ± 10.8	106.4 ± 9.4
PASP (mm Hg)	27.8 ± 2.9	29.6 ± 3.2	30.6 ± 3.0 <sup>d</sup>	26.2 ± 3.1 <sup>c</sup>	24.2 ± 3.8	27.8 ± 4.4
PADP (mmHg)	13.6 ± 3.1	14.8 ± 3.4	16.6 ± 1.1 <sup>d</sup>	13.4 ± 1.3 <sup>c</sup>	12.4 ± 1.1	13.4 ± 1.3
PAMP (mm Hg)	19.6 ± 0.8	20.8 ± 1.3	20.4 ± 1.5 <sup>d</sup>	18.2 ± 1.9 <sup>d</sup>	16.8 ± 2.4 <sup>c</sup>	18.8 ± 1.9 <sup>c</sup>
Cardiac output (l/min)	2.1 ± 0.3	2.3 ± 0.4 <sup>a</sup>	2.5 ± 0.3 <sup>a</sup>	2.5 ± 0.4 <sup>ac</sup>	2.1 ± 0.3	2.3 ± 0.4

Data are presented as mean ± SD of five dogs per group. <sup>a</sup> P<0.05, <sup>b</sup> P<0.001 Differences from baseline; <sup>c</sup> P<0.05, <sup>d</sup> P<0.001 Differences between groups. SAP- Systolic arterial pressure; DAP- Diastolic arterial pressure; MAP- Mean arterial pressure; PASP- Pulmonary artery systolic pressure; PADP- Pulmonary artery diastolic pressure; PAMP- Pulmonary artery mean pressure

Baseline CO values were not significantly different between the groups. There was also no statistically significant difference between the groups at 5 and 15 min after epidural anaesthesia. CO values at 30 min after epidural anaesthesia in Group LA were significantly higher than those of Group L. In-group analysis revealed that CO did not change in Group L, but increased significantly at the 5, 15 and 30 min after anaesthesia when compared to baseline values in Group LA.

The mean serum concentrations of lidocaine were lower in Group LA compared to Group L at all times after administration. However statistically significantly different lidocaine concentrations between groups were observed only at 5 min after administration ( $P=0.028$ ). Thereafter, there was no significant difference in lidocaine concentrations between groups (Fig. 1). In Group L peak serum concentration of lidocaine ( $C_{max}$ ) ( $1.65 \pm 0.9 \mu\text{g/mL}$ ) was achieved 5 min after lidocaine administration whereas in group LA after 15 min ( $0.81 \pm 0.5 \mu\text{g/mL}$ ).

### Discussion

To determine any advantages or disadvantages of lidocaine alone versus its combination with adrenaline with respect to hemodynamic change and peak serum concentration of lidocaine, two groups of dogs allocated to receive either lidocaine alone or a lidocaine-adrenaline combination were evaluated in the present study. Lidocaine has been widely used in all species because of its excellent diffusing and penetrating properties, as well as rapid onset of surgical analgesia. However, its action is too short lived to be used for major procedures (JONES, 2001; ADETUNJI et al., 2002) and is not without side effects. Hypotension and neurotoxicity are some limiting factors with this anaesthetic. The toxic dose of lidocaine in dogs is  $>10 \text{ mg/kg}$  and is determined when a muscle tremor is seen (LEMO et al., 2007). To overcome absorption of lidocaine from the epidural space and possible side effects, multiple drugs from different pharmacological classes have been used in combination (SOLOMON and GEBHART, 1994). There are several reports from different studies about the use of adrenaline along with other anaesthetics for epidural analgesia in different species. Although there are several assumptions on the mechanism of action of adrenaline, its effect on epidural administered drugs remains unclear. In our study, adrenaline was used in a dose of  $12.5 \mu\text{g/mL}$ , but other studies used adrenaline in lower doses ( $5 \mu\text{g/kg}$ ) (MAZOIT et al., 1996). We used a commercially prepared lidocaine-adrenaline combination registered for epidural anaesthesia in human medicine. Theoretically, it is unknown what would happen if we used a lower dose of adrenaline. Also, in our study, the quality and duration of analgesia, and the quantity of general anaesthetics administered was not observed and compared between the two groups. Also, no difference was observed between the total dose used of intermittently used ketamine and propofol.



Adrenaline added to the epidural solution may produce cardiovascular effects either directly through systemic absorption, or indirectly by altering the character of the epidural block, or by altering the rate of absorption of the local anaesthetic. It is well known that adrenaline stimulates both alpha and beta adrenergic receptors in a dose-dependent manner. Stimulation of alpha receptors produces vasoconstriction, resulting in an increase in total peripheral resistance (TPR), and stimulation of beta receptors produces an increase in stroke volume, CO and HR. The net effect of adrenaline on blood pressure depends upon which of the two receptor systems predominates. Adrenaline injected into the epidural space will be absorbed very slowly because of its local vasoconstrictor effect. The maintenance of increased drug concentrations in the epidural space could account for higher uptake into neural tissue, decreasing the serum concentrations, which are directly responsible for systemic toxicity. In addition, the blood levels of adrenaline achieved will produce a predominant  $\beta$ -adrenergic effect with reduction in TPR, increase in CO, and unchanged MAP. Morphine is often used in combination with local anaesthetics to prolong analgesia by a different mechanism than adrenaline, but morphine can cause numerous side effects (pruritus, vomiting, sedation, respiratory depression, urinary retention). Also, by usage of bupivacaine it is possible to prolong duration of epidural anaesthesia (4-6 h), but bupivacaine will also prolong motoric block (JONES, 2001).

In our study, the epidural administration of lidocaine alone caused a significant decrease in diastolic and mean artery pressure and also pulmonary artery pressure (PASP, PADP and PAMP) without change in HR. However, the epidural administration of lidocaine and adrenaline significantly affected HR, leaving arterial and pulmonary artery pressure unchanged. Also, our results show a significant increase in CO in animals who received a combination of lidocaine and adrenaline, probably due to an increase in HR. These data confirm the results of an earlier study by BONICA et al. (1971). In their study a greater increase in HR and CO was demonstrated after administration of lidocaine with adrenaline with the maximum effects noted at 15 min after epidural injection, but, in contrast to our results, these changes were accompanied by a significant decrease in SVR and MAP.

Although in our study the epidural block was accompanied with alterations in hemodynamics in both groups, the addition of adrenaline effectively reduced lidocaine induced hypotension. However, it is important to emphasize that at the same time HR and CO increased. These changes increased the work and oxygen demands on the heart and could be potentially harmful, particularly in patients with cardiac disease.

Furthermore, in our study epidurally administered adrenaline altered the absorption of lidocaine by delaying the peak plasma concentration and by reducing the magnitude of this peak, although statistically significantly different lidocaine concentrations were only observed between groups at 5 min after administration. The delay in the time to peak lidocaine concentration in Group LA can be explained by the local vasoconstrictor

action of epidural adrenaline and decreased lidocaine absorption into the systemic circulation. Our results are in agreement with previous studies looking at the effect of adding adrenaline to a local anaesthetic solution (MATHER et al., 1976; MAZOIT et al., 1996; SAKURA et al., 1999; SINNOTT et al., 2003).

Since the haemodynamic effects of systemic local anaesthetic are drug- and dose-dependent (SALEVSKY et al., 1990), we believe that the lower plasma lidocaine concentration observed in Group LA was one of the reasons contributing to maintenance of normal blood pressure.

In conclusion, it has been demonstrated that the addition of adrenaline to epidural lidocaine when compared with lidocaine alone is associated with the positive effect of adrenaline in prevention of hypotension. Differences between groups with respect to quality or duration of analgesia were not observed. However, because of the potential hazards of an increase in heart rate and CO, especially in elderly and critical ill patients, the routine use of epidural adrenaline should be reconsidered in patients with high hemodynamic risk.

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**SAŽETAK**

Adrenalin se često dodaje lokalnom anestetiku da smanji i uspori apsorpciju lokalnoga anestetika u krvotok, te na taj način smanji mogućnost nastanka neželjenih nuzućinaka. U ovom radu uspoređuju se učinci epiduralne anestezije lidokainom te lidokainom-adrenalinom na hemodinamske pokazatelje i koncentraciju lidokaina u serumu pasa. U istraživanje je bilo uključeno 10 pasa oba spola, koji su bili podijeljeni u dvije skupine. Psi skupine L (n=5) primili su 2%-tni lidokain u dozi 3,3 mg/kg, a psi iz skupine LA (n=5) primili su 2%-tni lidokain uz dodatak adrenalina u istoj dozi i količini. Koncentracija lidokaina u serumu te hemodinamski pokazatelji praćeni su 5, 15, 30, 45 i 60 minuta nakon epiduralne anestezije. Učinjena je statistička analiza podataka. Frekvencija bila i minutni volumen povećali su se značajno u skupini LA u odnosu na početne vrijednosti. Arterijski krvni tlak te tlak u a. pulmonalis smanjili su se u skupini L, te je u tim pokazateljima uočljiva i razlika između dviju skupina. Koncentracija lidokaina u serumu psa bila je niža u skupini LA u usporedbi sa skupinom L u svim mjerenjima. Dodavanje adrenalina lidokainu pri epiduralnoj anesteziji smanjilo je mogućnost nastanka hipotenzije. Međutim, zbog rizika povećanja frekvencije bila i minutnog volumena, preporučuje se oprez pri davanju adrenalina u starijih i kritično bolesnih pacijenata.

**Ključne riječi:** lidokain, lidokain-adrenalin, epiduralna anestezija

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