The influence of thoracic epidural anesthesia on liver hemodynamics in patients under general anesthesia

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Key words: *liver; thoracic epidural anesthesia; indocyanine green; plasma disappearance rate; regional perfusion.*

Summary. *Background and objective. Hepatic hypoperfusion is regarded as an important factor in the pathophysiology of perioperative liver injury. Although thoracic epidural anesthesia is a widely used technique, limited data are available about the effects on hepatic blood flow with blockade restricted to thoracic segments in humans. The main objective of the present study was to investigate the effects of thoracic epidural anesthesia on hepatic blood flow under general anesthesia in humans.*

Material and methods. In 40 patients under general anesthesia, we assessed hepatic blood flow using plasma disappearance rate of indocyanine green (PDR_{ICG}) as a simple noninvasive method before and after induction of thoracic epidural anesthesia. The epidural catheter was inserted at the $Th_{7/8}$ or $Th_{8/9}$, and 1% lidocaine at a mean (range) dose of 8 (6–10) mL was injected. Ephedrine bolus was given to patients who demonstrated a decrease in mean arterial blood pressure below 60 mm Hg after induction of thoracic epidural anesthesia (TEA-E group). Other patients did not receive any catecholamines during the study period (TEA group). Ten patients who did not undergo TEA served as controls (control group).

Results. In 7 patients, administration of ephedrine was necessary to avoid a decrease in mean arterial blood pressure below 60 mm Hg. Thus, the TEA-E group consisted of 7 patients and TEA group of 33. In the TEA group, thoracic epidural anesthesia was associated with a mean 2.3% min^{-1} decrease in PDR_{ICG} ($P<0.05$). In the TEA-E group, all seven patients showed a 2.2% min^{-1} decrease in PDR_{ICG} ($P<0.05$). Patients in the control group showed a mean 1.1% min^{-1} increase in PDR_{ICG} ($P<0.05$). In contrast to hepatic blood flow, cardiac output was not affected by thoracic epidural anesthesia.

Conclusions. In humans, thoracic epidural anesthesia is associated with a decrease in hepatic blood flow. Thoracic epidural anesthesia combined with ephedrine bolus was found to result in further decrease in hepatic blood flow.

Introduction

Patients undergoing major abdominal surgery are at risk of developing postoperative complications including multiple organ failure. Hepatic hypoperfusion is an important factor in the pathophysiology of perioperative liver injury (1). Furthermore, it has been hypothesized that hypoperfusion of the liver may initiate or contribute to the development of a systemic inflammatory response syndrome (2).

For decades, there have been discussions about whether or not thoracic epidural anesthesia (TEA) is able to reduce perioperative risk. Although thoracic epidural anesthesia and analgesia is widely used technique, the effects on hepatic blood flow with blockade restricted to thoracic segments have not been investigated extensively. TEA may induce different physiological reactions with conflicting effects on hepatic blood flow. For example, blockade of thoracic efferent sympathetic fibers causes regional arteriolar dilation and thus increases regional blood flow. On the other hand, hypotension as a result of a decreased systemic vascular resistance may reduce hepatic blood flow. Lumbar epidural anesthesia has been shown to induce a decrease in hepatic blood flow in animals (3, 4) and in humans (5, 6). In contrast to these studies, Vagts et al. (7), using a pig model, did not observe alterations in total hepatic blood flow after induction of thoracic epidural anesthesia despite a significant decrease in systemic arterial blood pressure. Thus, we hypothesized that thoracic epidural anesthesia maintains hepatic blood flow also in humans.

The main objective of the present study was to investigate the effects of TEA on hepatic blood flow

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under general anesthesia in humans. Plasma disappearance rate of indocyanine green (PDR_{ICG}) has been evaluated as a marker of hepatic perfusion and hepatocellular function, and its prognostic value in the critically ill patient has been demonstrated (8–9).

Material and methods

Participants. The protocol of this prospective study was reviewed and approved by the Kaunas Regional Ethics Committee for Biomedical Research. This study was designed with the aim to enroll 50 patients consecutively: 40 patients who underwent TEA and 10 patients who did not undergo TEA (control group). The control group consisted of patients with contraindications for TEA, in whom the insertion of a epidural catheter was not possible. We aimed at inclusion of 50 ASA physical status II or III patients with a mean age of 64.94 years (range, 33 to 80 years), who underwent elective upper abdominal interventions for carcinoma of the stomach, papilla of Vater, and pancreas. The informed consent was obtained from all the patients.

The abdominal interventions were carried out under combined general-epidural anesthesia. Patients with a history of cardiac or vascular disease, including hypertension, were excluded from the study. Hypertension was defined as a mean diastolic blood pressure of more than 100 mm Hg or a systolic blood pressure of more than 160 mm Hg or both at three measurements. Patients taking antihypertensive drugs, including diuretics, as well as antiarrhythmics or α_1-sympathicolytic medications for benign prostate hypertrophy were excluded. Furthermore, patients who had a history of known hypersensitivity to amide local anesthetics; severe respiratory, renal, or hepatic disease; diabetes mellitus; or neurological, psychiatric, or seizure disorders were excluded. In addition, patients with positive results of Allen's test (no collateral flow in the ulnar artery) at both upper extremities were excluded.

Preparation. Patients fasted from midnight. They received 7.5 mg of midazolam orally approximately 45–60 min before the procedure. After the patient was connected to standard equipment to monitor the blood pressure noninvasively and for continuous registration of heart rate and rhythm, an intravenous cannula was inserted into a large vein in the forearm or the antecubital fossa for fluid or drug administration.

Subsequently, after local infiltration of the skin with 1.0% lidocaine, the epidural space was accessed under sterile conditions with a 16-gauge Tuohy needle at the $Th_{7/8}$ or $Th_{8/9}$ interspace with a patient in the sitting position. Using a median or paramedian approach, the epidural space was identified using the loss of resistance to air technique. An epidural catheter was inserted 5 cm into the epidural space. After excluding an intravascular or arachnoid positioning of the catheter by aspiration, a bacterial filter was attached, and the catheter was capped sterilely. Thereafter, the patient was placed again in a supine horizontal position. Standardized general anesthesia was induced with 3–5 mg/kg of sodium thiopental and 2.5 μg/kg of fentanyl. Rocuronium (0.5 mg/kg) was administered to facilitate tracheal intubation. Anesthesia was maintained with sevoflurane and fentanyl as required. Ventilation was mechanically controlled to maintain end-tidal carbon dioxide tension near 35 mm Hg. After the induction of anesthesia, a 20-gauge arterial cannula was inserted into a radial artery, preferentially in the nondominant arm, for invasive blood pressure monitoring. A central venous line was placed via the right internal jugular vein for central venous pressure monitoring. Patients were actively warmed with forced air on the lower body to maintain intraoperative normothermia (Bair Hugger, Augustine Medical, Eden Prairie, MN). Liver hemodynamics was measured noninvasively using LiMON monitor and fingertip sensor (PULSION Medical Systems AG, Munich, Germany), which allows for noninvasive measurement of the blood concentration-time curve of indocyanine green (ICG). ICG was used as an indicator.

Experimental procedure. The experiment consisted of four parts. The first part of the experiment was aimed at obtaining baseline values. Before baseline measurements, all patients received at least 500 mL of crystalloids. Depending on the mean arterial pressure (MAP), further fluids were administered with the aim to maintain MAP above 60 mm Hg. After complete preparation for the experiment, a 10-min stabilization period was observed.

The second part of the experiment was aimed at measuring hepatic blood flow before epidural anesthesia. Patients received a rapid intravenously ($\leqslant$2 s) injected bolus administration of ICG (0.25 mg/kg body weight), followed by measurement of plasma disappearance (PDR) and retention 15 (R15) rate of ICG using the pulse densitometer. Simultaneously, heart rate, invasive arterial blood pressure, central venous pressure (CVP), cardiac output (CO), and systemic vascular resistance index (SVRI) were continuously recorded.

The third part of the experiment was aimed at assessing the effects of epidural anesthesia on liver hemodynamics. Using the epidural catheter that had been installed before the experiment, 1% lidocaine was epidurally administered at a rate of 1 mL $\cdot$ s^{-1}. Epidural bolus dose of lidocaine was calculated according to the patient's age: 20–40 years, 1.5 mL/segment; 40–60 years, 1.3 mL/segment; 60–80 years, 1.0 mL/segment; and more than 80 years,

0.7 mL/segment (10). We aimed to achieve epidural block from Th_5 to Th_{12}. Fluid administration was continued with approximately 5 $mL \cdot kg^{-1} \cdot h^{-1}$. Hypotension (decrease in SBP >30% of the pre-experimental value or SBP <90 mm Hg) was treated by administering the rapid intravenous infusion of a colloid solution (6% hydroxyethyl starch; Voluven, Fresenius Kabi, Bad Homburg, Germany). After 30 min, the second measurement of PDR_{ICG} and R15 was carried out as described above. A PDR_{ICG} of 18–25% min^{-1} is considered to be normal. Like all dynamic liver tests, PDR_{ICG} is dependent on sinusoidal perfusion as well as hepatocellular function. While higher values can be observed in hyperdynamic states, lower values are due to acute or chronic hepatocellular dysfunction or due to macrocirculatory or microcirculatory perfusion disorders leading to reduced overall sinusoidal perfusion (11). Central hemodynamic parameters were recorded simultaneously. If MAP decreased below 60 mm Hg within a period of 15 min after injection of lidocaine, 5-mg ephedrine was administered intravenously. Waiting periods of 15 min after a bolus administration of ephedrine were observed before ICG was administered to measure liver hemodynamics. Patients who received ephedrine were separately analyzed as the TEA-E subgroup (TEA-E group).

The fourth part of the experiment was aimed at assessing central hemodynamic parameters (CVP, CO, and SVRI) 1 hour after the epidural bolus.

All measurements were performed before surgery. The function of the epidural catheter was checked by pinprick evaluation of sensory blockade on the first postoperative day.

Hemodynamic assessment. The arterial blood pressure and CVP were monitored throughout the study period using a pressure transducer (MEMSCAP AS, Skoppum, Norway). Further hemodynamic values (CO and SVRI) were assessed using an impedance cardiography monitor (BioZ CardioDynamics International Corp., San Diego, CA, USA).

Statistical analysis. Measured parameters were analyzed using the SPSS for Windows 13.1 (SPSS Inc., Chicago, Illinois, USA). An a priori power analysis was conducted indicating that, given the scatter of the data, a sample size of 40 would be sufficient to detect a 4% min^{-1} difference in PDR_{ICG} that is a 20% improvement or deterioration of a normal value of about 20% min^{-1} with a power of 0.9 and an α error of 5%. All data were expressed as means±SD. When normal distribution of the variables could not be assumed, we compared significance level of Student's and Wilcoxon's tests. Because significance level of these tests was equal, we presented our results like parametric variables. Intragroup changes in hemodynamic variables were analyzed using the Wilcoxon's signed rank test (P_1 value). The statistical significance level was set at 0.05.

Results

Fifty patients were included in this study. No patient was excluded because of epidural catheter dislocation, because on the first postoperative day, a correct catheter position in the epidural space could be confirmed in all patients. After induction of TEA, seven patients demonstrated a decrease in MAP below 60 mm Hg, so ephedrine bolus was administered (TEA-E group). The remaining 43 patients did not

Table 1. Patients' characteristics

Characteristic	Group		
	TEA (n=33)	TEA-E (n=7)	Control (n=10)
Age, mean (SD), years	65.3 (12.7)	64.1 (13.0)	64.4 (8.1)
Gender, M/F, %	57.6/42.4	42.9/57.1	50.0/50.0
Height, mean (SD), cm	170.8 (4.0)	170 (4.5)	170.5 (4.4)
Weight, mean (SD), kg	74.8 (9.2)	75.6 (13.6)	78.0 (9.4)
Body mass index, mean (SD), kg/m^2	25.6 (2.4)	26.0 (3.7)	26.7 (2.2)
ASA class, II/III, %	81.8/18.2	85.7/14.3	70.0/30.0
Surgical diagnosis, %			
Cancer of papilla of Vater	15.2	0	10.0
Pancreatic cancer	18.2	14.3	0
Chronic pancreatitis	9.1	0	20.0
Stomach cancer	57.6	85.7	70.0

ASA, American Society of Anesthesiologists; TEA, thoracic epidural anesthesia; TEA-E, thoracic epidural anesthesia-ephedrine.

receive any catecholamines during the study period. There were no significant differences comparing the groups except gender differences (Table 1).

The volume of lidocaine was 8 (6–10) mL both in the TEA and TEA-E groups. There were no significant differences in end-tidal sevoflurane and CO_2 concentrations and amounts of administered fluids between the two groups. PDR_{ICG} showed a significant (P=0.03) decrease after induction of TEA, but not below the normal value (18% min^{-1}). This difference was similar in the TEA and TEA-E groups (Fig.). In the control group, PDR_{ICG} showed an increase after 30 min following hemodynamic stabilization. TEA was associated with a significant decrease in HR and MAP. TEA was not associated with a significant change in CO and SVRI in the TEA or TEA-E groups (Table 2).

Discussion

In the present study, we have investigated the influence of TEA on PDR_{ICG} as a surrogate of liver blood flow in patients under general anesthesia. We used PDR_{ICG} as a simple noninvasive method to assess hepatic perfusion. ICG binds avidly to plasma proteins, so that its distribution volume equals the blood plasma volume. ICG is exclusively eliminated from blood by the liver with a half-life of 150–180 s and without enterohepatic circulation (12). The elimination of ICG strongly correlates with hepatic blood flow, but is not similar to hepatic flow as the extraction ratio may differ among patients and clinical conditions (13). As a matter of principle, both liver blood flow and hepatocellular function affect dynamic liver tests such as PDR_{ICG}; however, rapid changes that was within 30 min as in our study can be explained almost exclusively by changes in sinusoidal blood flow (11).

Hepatic blood flow in surgical patients can be altered by a variety of factors, including arterial blood pressure, posture changes, carbon dioxide levels, intravascular volume shifts, positive pressure ventilation, and volatile anesthetics (1). In the present study, measurements were performed under general anesthesia and mechanical ventilation; hence, hepatic blood flow was presumably already decreased during baseline measurements. Many of the factors affecting hepatic blood flow, such as carbon dioxide levels, end-tidal sevoflurane concentration, and mode of ventilation remained constant during this investigation. All measurements were performed before surgery to exclude the influence of intraoperative surgical manipulations. Fluid administration was continued during the study period, so there was no decrease in intravascular volume. Measurements were started when patients were hemodynamically stable according to blood pressure and heart rate. In contrast to our hypothesis, we found a consistent

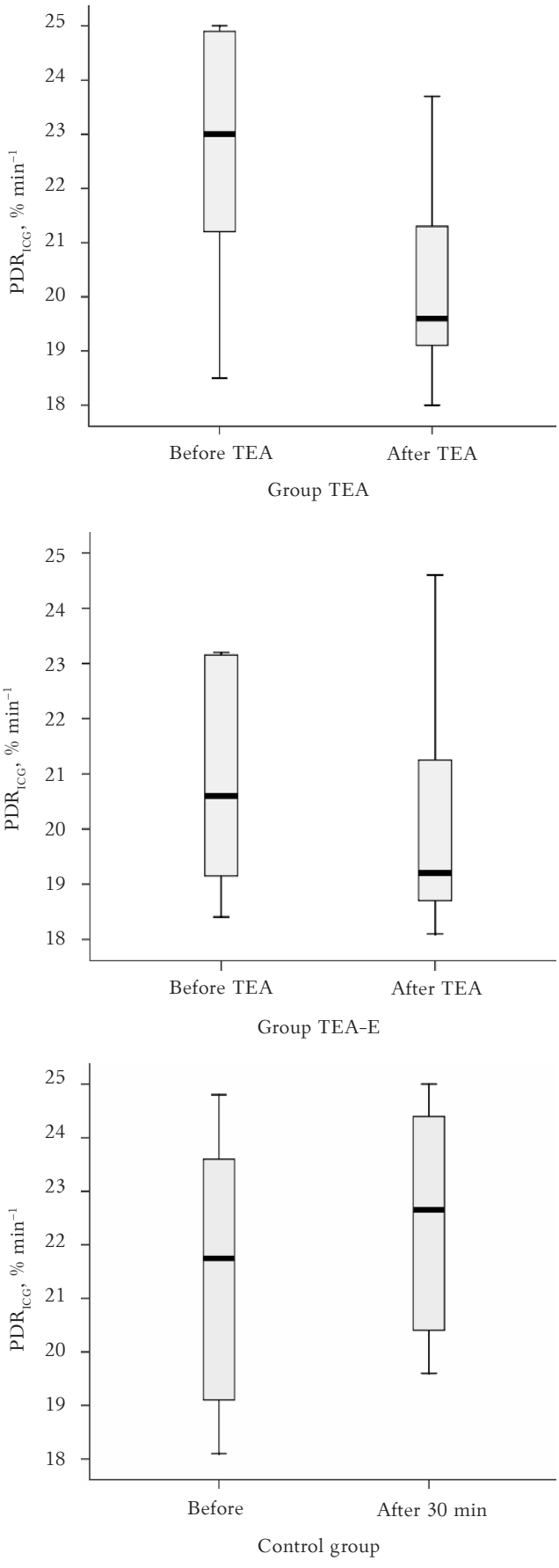

Fig. Effects of thoracic epidural anesthesia on hepatic blood flow

Table 2. Effects of thoracic epidural anesthesia on global hemodynamic variables

Clinical parameter	Baseline	30 min after induction of TEA	*P* value
Heart rate, beats/min			
TEA group	68.0 (5.4)	66.2 (5.3)	<0.04
Control group	67.0 (7.0)	69.2 (5.2)	0.02
Systolic arterial pressure, mm Hg			
TEA group	115.9 (12.7)	107.6 (8.9)	<0.001
Control group	111.3 (7.4)	118.9 (5.70)	0.01
Diastolic arterial pressure, mm Hg			
TEA group	66.8 (10.1)	62.1 (8.8)	0.001
Control group	61.2 (8.4)	61.9 (8.4)	NS
Mean arterial pressure, mm Hg			
TEA group	83.4 (11.2)	76.7 (8.9)	<0.001
Control group	79.5 (6.6)	83.0 (6.6)	0.04
Central venous pressure, mm Hg			
TEA group	6.6 (2.5)	7.1 (2.0)	0.03
Control group	4.4 (1.8)	3.6 (1.0)	NS
Cardiac output, L/min			
TEA group	4.8 (0.5)	4.9 (0.5)	NS
Control group	5.2 (0.5)	5.2 (0.5)	NS
SVRI, dyne · s · cm^{-5} · m^2			
TEA group	2055.9 (309.2)	1757.7 (311.9)	<0.001
Control group	2062 (117.1)	2072.4 (113.0)	0.004

Data are given as mean (SD). TEA, thoracic epidural anesthesia; SVRI, systemic vascular resistance index.

decrease in PDR_{ICG} as a marker of regional hepatic perfusion after induction of TEA. The combination of TEA with ephedrine (TEA-E group) did not improve liver blood flow. However, cardiac output was not significantly affected by TEA.

Presumably, the TEA-related decrease in systemic arterial blood pressure is the main reason for the observed decrease in hepatic blood flow. The negative effects of the decrease in systemic arterial blood pressure prevail over the anticipated positive effects of local vasodilation. One reason for the arterial blood pressure effect of hepatic blood flow regulation might be that the hepatic artery exhibits almost no autoregulatory capacity, as a nearly linear correlation between systemic arterial blood pressure and hepatic arterial blood flow has been found (14, 15).

In previous studies, there had been conflicting results on the influence of EA on parameters of splanchnic perfusion, most of them using mucosal pH and arterial-mucosal p_{CO2} difference as surrogate parameters. This might be due to different therapy regimens, patient population, or insertion sites of epidural catheters or simply point to the limitations of gastrointestinal tonometry (16, 17). The effects of TEA on hepatic blood flow have been investigated in only one animal study and two studies in humans. In contrast to the present study, Vagts et al. (7), using a pig model, observed an unaltered total hepatic blood flow after induction of TEA (Th_5–Th_{12}) despite a 30% decrease in MAP. The reasons for the discrepancy between the data found in pigs and our findings in humans may be species-related and could be due to different physiological reactions on TEA-induced sympathicolysis. Kortgen et al. (11) found that liver perfusion was increased with thoracic but not lumbar EA after major abdominal surgery in most patients. The study by Meierhenrich et al. (18) found a significant decrease in hepatic venous blood flow after induction of TEA using multiplane transesophageal echocardiography. The combination of TEA with continuous infusion of norepinephrine seems to induce a further decrease in hepatic blood flow.

Three human studies have been performed to evaluate the effects of high lumbar epidural anesthesia (LEA) on hepatic blood flow. Kennedy et al. (5) measured hepatic venous flow in conscious volunteers by the ICG clearance technique using a hepatic venous catheter and, after induction of high LEA, found a 25% decrease in hepatic blood flow despite a constant cardiac output. Tanaka et al. (6) also performed high LEA. They estimated hepatic blood flow by pulse densitometric determination of the PDR_{ICG} and reported a 35% decrease in hepatic blood flow. Simon et al. (19) measured liver blood

flow using pulse dye densitometry (PDD), which allows for noninvasive measurement of the blood concentration of ICG. They found a decrease in liver blood flow after LEA. We performed TEA and assessed the changes in hepatic blood flow by the use of noninvasive ICG clearance test. Nevertheless, we found almost the same reduction in hepatic blood flow. Thus, the present data do not support the hypothesis that TEA is superior to LEA with respect to the effects on hepatic perfusion.

All 7 patients (TEA-E group) treated with ephedrine to compensate for the TEA-induced decrease in arterial blood pressure showed any improvement in PDR_{ICG} and did not differ from the main TEA group. Neither human nor experimental studies have been performed to investigate the effects of ephedrine in combination with TEA on hepatic blood flow. Experimental studies assessed the sole effect of norepinephrine on hepatic blood flow and consistently found a decrease in flow to be between 20% and 45% (20–22). The present data do not suggest that TEA attenuates ephedrine-induced vasoconstriction in the splanchnic bed.

We cannot draw any conclusion as to what extent the observed decrease in hepatic blood flow was associated with alterations in hepatic oxygen balance and metabolic activity. However, the decrease might be relevant in specific patients' groups, such as those with pre-existing liver disease or those undergoing hepatic surgery. Outcome studies should be performed to evaluate TEA with respect to possible perioperative hepatic damage, especially in these patients' groups. In addition, the intraoperative use of ephedrine in combination with TEA should be investigated in outcome studies and compared with over vasopressor therapies.

Conclusions

In humans, thoracic epidural anesthesia is associated with a decrease in hepatic blood flow. Thoracic epidural anesthesia combined with ephedrine bolus was found to result in further decrease in hepatic blood flow.

Epidurinės anestezijos krūtinės lygyje įtaka kepenų kraujotakai bendrosios anestezijos metu

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Raktažodžiai: kepenys, epidurinė anestezija krūtinės lygyje, indociano žaliasis, išnykimo iš kraujo plazmos greitis, regioninė kraujotaka.

Santrauka. *Tyrimo tikslas.* Kepenų hipoperfuzija turi didelę įtaką perioperacinio kepenų pažeidimo fiziologijai. Klinikinėje praktikoje epidurinė anestezija krūtinės lygyje naudojama plačiai, tačiau duomenų apie epidurinės krūtininių segmentų blokados poveikį žmonių kepenų kraujotakai yra labai nedaug. Pagrindinis šio tyrimo tikslas – nustatyti epidurinės anestezijos krūtinės lygyje įtaką žmonių kepenų kraujotakai bendrosios anestezijos metu.

Tyrimo medžiaga ir metodai. 40 pacientų bendrosios anestezijos metu tyrėme kepenų kraujotaką indociano žaliojo išnykimo iš kraujo plazmos (PDR_{ICG}) metodu prieš ir po epidurinės anestezijos sukėlimo krūtinės lygyje. Epidurinis kateteris buvo įkištas $Th_{7/8}$ arba $Th_{8/9}$ lygyje, į kurį buvo švirkščiama lidokaino 1 proc. tirpalo 8 (6–10) ml. Pacientams, kuriems po epidurinės anestezijos sukėlimo vidurinis arterinis kraujo spaudimas sumažėjo mažiau kaip 60 mmHg (TEA-E grupė) buvo skiriama į veną efedrino bolusas. Kitiems pacientams tyrimo metu katecholaminų neskirta (TEA grupė). Kontrolinę grupę (KG) sudarė 10 pacientų.

Rezultatai. Septyniems pacientams buvo būtina sušvirkšti efedrino siekiant išvengti vidurinio arterinio kraujo spaudimo kritimo žemiau nei 60 mmHg. Taigi, TEA-E grupę sudarė septyni, o TEA – 33 pacientai. TEA grupės pacientams epidurinė anestezija buvo susijusi su vidutiniškai 2,3 proc. $min.^{-1}$ ($p<0,05$) PDR_{ICG} sumažėjimu. Visiems septyniems TEA-E grupės pacientams PDR_{ICG} sumažėjo 2,2 proc. $min.^{-1}$ ($p<0,05$). Kontrolinės grupės pacientams PDR_{ICG} padidėjo 1,1 proc. $min.^{-1}$ ($p<0,05$). Priešingai – kepenų kraujotakai epidurinė anestezija krūtinės lygyje širdies minutinio tūrio pokyčių nesukėlė.

Išvados. Žmonėms epidurinė anestezija krūtinės lygyje yra susijusi su kepenų kraujotakos sumažėjimu. Epidurinė anestezija krūtinės lygyje derinyje su efedrinu taip pat mažina kepenų kraujotaką.

References

1. Gelman S. General anesthesia and hepatic circulation. Can J Physiol Pharmacol 1987;65:1762-79.
2. Maynard ND, Bihari DJ, Dalton RN, Beale R, Smithies MN, Mason RC. Liver function and splanchnic ischemia in critically ill patients. Chest 1997;111:180-73.
3. Greitz T, Andreen M, Irestedt L. Haemodynamics and oxygen consumption in the dog during high epidural block with special reference to the splanchnic region. Acta Anaesthesiol Scand 1983;27:211-7.
4. Sivarajan M, Amory DW, Lindbloom LE. Systemic and regional blood flow during epidural anesthesia without epinephrine in the rhesus monkey. Anesthesiology 1976;45:300-10.
5. Kennedy WF, Everett GB, Cobb LA, Allen GD. Simultaneous systemic and hepatic hemodynamic measurements during high peridural anesthesia in normal man. Anesth Analg 1971;50:1069-78.
6. Tanaka N, Nagata N, Hamakawa T, Takasaki M. The effect of dopamine on hepatic blood flow in patients undergoing epidural anesthesia. Anesth Analg 1997;85:286-90.
7. Vagts DA, Iber T, Puccini M, Szabo B, Haberstroh J, Villinger F, et al. The effects of thoracic epidural anesthesia on hepatic perfusion and oxygenation in healthy pigs during general anesthesia and surgical stress. Anesth Analg 2003;97:1824-32.
8. Sakka SG, Reinhart K, Meier-Hellmann A. Prognostic value of the indocyanine green plasma disappearance rate in the critically ill patients. Chest 2002;122:1715-20.
9. Kimura S, Yoshioka T, Shibuya M, Sakano T, Tanaka R, Matsuyama S. Indocyanine green elimination rate detects hepatocellular dysfunction early in septic shock and correlates with survival. Crit Care Med 2001;29:1159-63.
10. Marchertienė I. Regioninė anestezija. (Regional anesthesia.) Kaunas: KMU Spaudos ir leidybos centras; 2001. p. 121.
11. Kortgen A, Silomon M, Pape-Becker C, Buchinger H, Grundmann U, Bauer M. Thoracic but not lumbar epidural anaesthesia increases liver blood flow after major abdominal surgery. Eur J Anaesthesiol 2009;26:111-6.
12. Henschen S, Busse MW, Zisowsky S, Panning B. Determination of plasma volume and total blood volume using indocyanine green: a short review. J Med 1993;24:10-27.
13. Sakka SG. Assessing liver function. Curr Opin Crit Care 2007;13:207-14.
14. Ayuse T, Brienza N, O'Donnell CP, Robotham JL. Pressure-flow analysis of portal vein and hepatic artery interaction on porcine liver. Am J Physiol 1994;267:H1233-42.
15. Ezzat WR, Lautt WW. Hepatic arterial pressure-flow autoregulation is adenosine mediated. Am J Physiol 1987;252: H836-45.
16. Bams JL, Mariani MA, Groeneveld ABJ. Predicting outcome after cardiac surgery: comparison of global haemodynamic and tonometric variables. Br J Anaesth 1999;82:33-7.
17. Hamilton MA, Mythen MG. Gastric tonometry: where do we stand? Curr Opin Crit Care 2001;7:122-7.
18. Meierhenrich R, Wagner F, Schütz W, Rockemann M, Steffen P, Senftleben U, et al. The effects of thoracic epidural anesthesia on hepatic blood flow in patients under general anesthesia. Anesth Analg 2009;108:1331-7.
19. Simon MJG, Reekers M, Veering BT, Boer F, Burm AGL, Kleef JW, et al. Cardiovascular parameters and liver blood flow after infusion of a colloid solution and epidural administration of ropivacaine 0.75%: the influence of age and level of analgesia. Eur J Anaesthesiol 2009;26:166-74.
20. Turk LN, Shoemaker WC. Hepatic vascular response to norepinephrine. Am J Physiol 1962;202:1175-8.
21. Priebe HJ, Nöldge GFE, Armbruster K, Geiger K. Differential effects of dobutamine, dopamine, and noradrenaline on splanchnic haemodynamics and oxygenation in the pig. Acta Anaesthesiol Scand 1995;39:1088-96.
22. Macas A, Kriščiukaitis A, Buivydaitė K, Bakšytė G, Žaliūnas A. Hemodinamikos tyrimai ūminio miokardo infarkto pasekmėms prognozuoti. (Hemodynamic studies for prediction of acute myocardial infarction outcomes.) Medicina (Kaunas) 2008;44(8):640-9.

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