Comparison of the effects of sevoflurane and isoflurane on myocardial protection in coronary bypass surgery

Koroner baypas cerrahisinde miyokardiyal korumasında sevofluran ve isofluranın etkilerinin karşılaştırılması

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ABSTRACT

Objective: The aim of this prospective randomized study was to compare the myocardial protective effects of sevoflurane and isoflurane during coronary bypass surgery.

Methods: After induction of general anesthesia with etomidate 0.3 mg/kg, a bolus dose of pancuronium 0.1 mg/kg and remifentanil 1 mcg/kg was administered. For the maintenance of anesthesia, patients received either sevoflurane (n=20) at 2-4% or isoflurane (n=20) at 1-2%. Arterial blood samples were obtained as follows: before induction of anesthesia, after aortic unclamping, at postoperative period. Troponin-T, creatine kinase (CK), and creatine kinase-MB (CKMB) values were measured in all obtained samples. Statistical analysis was performed using two-way ANOVA analysis and Mann-Whitney test.

Results: Heart rate was significantly higher in the sevoflurane group during the aortic side-clamp period, at the 10th minute and 20th minute after cardiopulmonary bypass (CPB) ending. The CK-MB values at 24th postoperative hour in the sevoflurane group were found to be significantly lower from the isoflurane group. The troponin-T values following the removal of the cross-clamp (1.015 (0.935-1.850) ng/ml vs 1.469 (1.290-1.645) ng/ml, p<0.001) and those at the 24th postoperative hour (5.345±0.654 ng/ml vs 8.715±1.020 ng/ml, p<0.001) were significantly lower in the sevoflurane group when compared to those in the isoflurane group.

Conclusion: Sevoflurane provides a better myocardial protection than isoflurane, as may be inferred by the lower levels of the myocardial injury markers troponin-T and CK-MB observed with sevoflurane. *(Anadolu Kardiyol Derg 2011; 11: 257-62)*

Key words: Sevoflurane, isoflurane, myocardial protection, coroner bypass surgery

ÖZET

Amaç: Bu prospektif randomize çalışmanın amacı sevofluran ve isofluranın koroner baypas cerrahisi sırasında miyokart üzerindeki koruyucu etkilerini karşılaştırmaktır.

Yöntemler: Etomidat (0.3 mg /kg) ile genel anestezi indüksiyonunu takiben 0.1 mg/kg pankuronyum ve 1 mikrogr/kg remifentanil bolus olarak verildi. Anestezi idamesi Grup 1'deki hastalarda (n=20) %2-4 değerinde sevofluran, Grup 2'deki hastalarda (n=20) %1-2 değerinde isofluranla sağlandı. Anestezi indüksiyonundan önce, aortik klemp kaldırıldıktan sonra ve postoperatif periyotta arteriyel kan örnekleri alındı. Bu örneklerde Troponin T, kreatinin kinaz ve kreatinin kinaz MB değerleri ölçüldü. İstatistiksel analizler -iki-yönlü ANOVA ve Mann-Whitney testleri ile yapıldı.

Bulgular: Kalp hızı sevofluran grubunda pompa periyodunda, pompadan çıkış sonrası 10. ve 20. dakikalarda anlamlı yüksek bulunmuştur. CKMB düzeyi postoperatif 24. saatte sevofluran grubunda isofluran grubuna göre anlamlı düşüktür. Troponin T kros- klemp kaldırıldıktan sonra (1.015 (0.935-1.850) ng/ml, 1.469 (1.290-1.645) ng/ml, p<0.001) ve postoperatif 24.saatte (5.345±0.654 ng/ml, 8.715±1.020 ng/ml, p<0.001) sevofluran grubunda isofluran grubu ile karşılaştırıldığında anlamlı düşüktür.

Sonuç: Miyokardiyal hasarlanma işaretleyicileri olan troponin T ve CKMB düzeylerinin daha düşük düzeylerde saptanması ile sevofluranın isofluranа göre miyokardiyal korumayı daha iyi sağlamakta olduğu kanısındayız. *(Anadolu Kardiyol Derg 2011; 11: 257-62)*

Anahtar kelimeler: Sevofluran, isofluran, miyokardiyal koruma, koroner baypas cerrahisi

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Introduction

It is well known that coronary artery bypass graft (CABG) surgery contributes to the myocardial dysfunction, and is associated with high morbidity and mortality. The effectiveness of myocardial preservation will decrease consequences of ischemia/reperfusion injury and duration of in-hospital stay (1). Many methods have been tried as means for myocardial protection. Systemic hypothermia (with its influence on collateral flow to the heart), topical hypothermia and above all, some combination of the available cardioplegic techniques are used for this purpose (2). Experimental data indicate that volatile general anesthetics have protective effects against ischemia-reperfusion injury that is independent of their hemodynamic effects (3). Volatile anesthetics have been shown to have a preconditioning-like effect, resulting in protection against myocardial infarction and irreversible myocardial dysfunction. Volatile anesthetics have also been shown to provide protection against reperfusion injury when administered after myocardial ischemia (4, 5). However, the studies on the effects of sevoflurane and isoflurane on myocardial protection reported conflicting results.

The aim of this study is, to compare the myocardial protective effects (by means of investigated enzyme profile) of sevoflurane and isoflurane administered to patients selected for CABG surgery during the whole time span of the surgery, including the hypothermic cardiac arrest period when sevoflurane and isoflurane are administered by the cardiopulmonary bypass (CPB) machine.

Methods

Patients

In this prospective randomized study, following approval by the hospital's Ethic Committee (protocol-2005/416), our study was conducted on 40 American Society of Anesthesiologists (ASA) Class I-II patients, aged 48-69 years, scheduled for elective CABG surgery. The patients were randomly allocated into two groups of 20 patients each.

Patients with a history of myocardial infarction within the past 6 months, those with ejection fraction less than 40%, those who needed valvular or aortic surgery in addition to the planned CABG surgery, unstable angina patients, emergency cases, patients with high cardiac enzymes 24 hours prior to surgery, hemodynamically unstable patients (requiring inotropic agents support or intraaortic balloon pump preoperatively), patients with any severe systemic disease, diabetes mellitus, and patients on immune-suppressive therapy were excluded from the study. None of the patients included received theophylline.

Perioperative and operative period

All patients were premedicated with morphine 0.1 mg/kg intramuscular (im) 30 minutes before the surgery. Radial arterial catheter placed under local anesthesia prior to induction. After induction of general anesthesia with etomidate 0.3 mg/kg intravenously (iv), a bolus dose of pancuronium 0.1 mg/kg and remifentanil 1 mcg/kg was administered. For the maintenance of anesthesia, group 1 (n=20) patients received volatile anesthesia with sevoflurane at 2-4% and group 2 (n=20) patients with isoflurane at 1-2%. Following intubation, the patients in both groups were started on a remifentanil infusion at a rate of 0.1-0.4 µg/kg/min iv.

Cardiopulmonary bypass was performed with using a membrane oxygenator, hemodilution and moderate systemic hypothermia (28-32°C). Multidose cold crystalloid cardioplegia with potassium (20 mEq/l) and topical saline ice slush were used for myocardial protection during bypass. Patients had median sternotomy with harvesting of radial artery, saphenous veins and internal thoracic arteries as conduits. Distal anastomoses were performed during continuous aortic cross clamping, followed by proximal vein grafting during partial aortic occlusion. A hematocrit value of 25-30%, a MAP value of 50-80 mmHg, and CPB flow was maintained between 2-2.5 L/m^2.

Before starting to extracorporeal circulation pancuronium 0.05 mg/kg, remifentanil 0.05 µg/kg and pentothal 3 mg/kg were injected into the cardiotomy reservoir in both groups. During extracorporeal circulation, group 1 patients continued to receive sevoflurane in concentrations of 0.5-2%, while group 2 patients continued to receive isoflurane 0.5-2%.

Heart rate (HR), systolic arterial pressure (SAP), mean arterial pressure (MAP) and diastolic arterial pressure (DAP) readings were recorded just prior to induction (t1), post induction (t2), post intubation (t3), post skin incision (t4), post sternotomy (t5), after the removal of the cross-clamp (t6), at the 10th minute (t7) and at the 20th minute following completion of the extracorporeal circulation (t8), at the 1st hour (t9), at the 6th hour (t10), at the 12th hour (t11), and at the 24th hour (t12) after admission to the intensive care unit.

The patients were continuously monitored with regard to inadequate anesthesia depth. The following findings were assumed to indicate in adequate anesthesia: a HR of more than 90 beats/minute lasting for more than 1 minute prior to the CPB, a preoperative baseline SAP value above 150 mmHg prior to CPB, or a SAP of more than 140 mmHg lasting for more than 1 minute prior to or following the CPB, a MAP above 80 mmHg during CPB, observation of somatic (movements, eye opening, deglutition) and autonomic (sweating, tear production) responses. Whenever in adequate anesthesia depth was observed, the sevoflurane or isoflurane concentrations were increased by 0.5%. Sevoflurane did not exceed 4% and isoflurane did not exceed 2.3%.

Aortic cross-clamp duration and total CPB duration for all cases were recorded. Previous doses of sevoflurane or isoflurane and remifentanil were restored upon completion of extracorporeal circulation. The anesthetic agents were discontinued upon completion of surgery and patients were administered morphine 0.015 mg/kg iv before they being to the intensive care unit.

Hemodynamically stable, normothermic (body temperature above 36°C) patients without arrhythmias, whose chest tube drainage volume was less than 100 ml/hr, urinary output was greater than 0.5 ml/kg/hr, PaO_2 greater than 90 mmHg with an FiO_2 less than 50%, respiratory rate - 10-30/min, pH more than 7.5, and PCO_2 less than 55 were extubated.

Laboratory analyses

Arterial blood samples were obtained as follows: baseline sample (before induction of anesthesia), after aortic unclamping, at the 2nd postoperative hour and at the 24th postoperative hour. Troponin-T, creatine kinase (CK) and CK-MB values were measured in all obtained samples. Samples were centrifuged and frozen at -80°C to be analyzed at laboratory. Photometric determination of the activity of CK (the reference range: 22-240 IU/L), and CK-MB (the reference range:0-25 IU/L) in blood samples that was measured with standard measured by autoanalyser (Cobas, Roche, USA). Troponin-T was measured according to manufacturer recommendation by standard immunoassay techniques (Dimension, Newark, USA).

Statistical analysis

Data were processed using the SPSS for Windows version 13.0 program (Chicago, IL, USA). Continuous variables are presented as mean±standard deviation and median (minimum-maximum) values. Intergroup and intra-group comparisons were accomplished using t-test, Chi-sguare test, Mann-Whitney test and two-way analysis of variance. A sample size of 20 patients per group was needed for comparison between two groups (α=0.05, two samples t-test and power of the study of 85%). The statistically significant value was assumed to be p<0.05.

Results

No statistically significant difference was found between Group 1 and Group 2 with respect to demographic characteristics, perioperative medications, number of grafts, CPB pump duration and cross-clamping duration (p>0.05; Table 1).

The heart rate data of the cases are shown in Figure 1. Intergroup comparison shows that HR at the 6th postoperative hour is statistically higher in the sevoflurane group (p<0.05). The data showed no statistically significant difference in the HR between the two groups in the rest of the time intervals (p>0.05).

The HR prior to induction in the sevoflurane group was found to be statistically significantly higher when compared to HR during the partial pump period, at the 10th minute and 20th minute following completion of the extracorporeal circulation (p<0.05).

In the isoflurane group, the comparison of the heart rates prior to and following induction yielded a statistically significant reduction in HR following induction (p<0.05). The HR values following intubation, skin incision, and sternotomy were found to be significantly higher than those following induction (p<0.05).

Table 1. Demographic data, number of coronary grafts, pump and cross-clamping durations

Variables	Sevoflurane (n=20)	Isoflurane (n=20)	p*
Age, years	59.0±1.5	57.0±1.5	NS
Weight, kg	69.0±2.6	72.0±1.8	NS
Gender, M/F, n	13/7	15/5	NS
Perioperative medication			
Beta-blockers	19	20	NS
Calcium channel blockers	18	17	NS
Nitrates	10	10	NS
Number of coronary grafts, n	2.5±0.1	2.5±0.1	NS
EF, %	55.0±1.6	54.0±1.5	NS
Pump duration, min	78.0±2.9	74.0±1.9	NS
Cross-clamping duration, min	51.0±2.8	54.0±1.9	NS

Data are expressed as mean±SD values and proportions
unpaired t-test and Chi-square test
EF - ejection fraction, F - female, M - male, NS-non-significant (p>0.05)

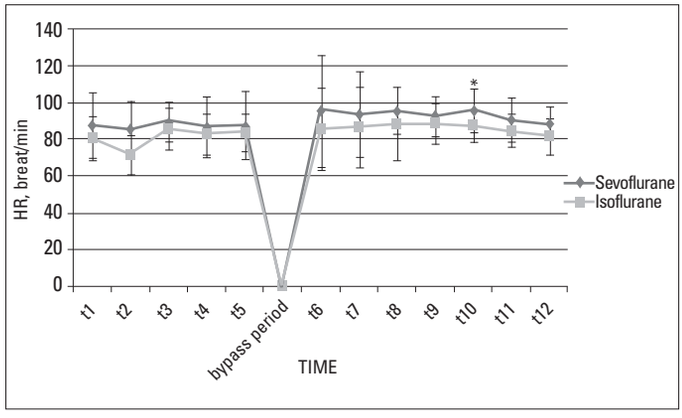

Figure 1. Heart rate values of cases. Intergroup comparison shows that HR at the 6th postoperative hour is statistically higher in the sevoflurane group (*p=0.025, (ANOVA for repeated measurements)
Prior to induction (t1), post induction (t2), post intubation (t3), postskin incision (t4), poststernotomy (t5), after the removal of the cross-clamp (t6), at the 10th minute (t7) and at the 20th minute following completion of the extracorporeal circulation (t8), at the 1st hour (t9), at the 6th hour (t10), at the 12th hour (t11), and at the 24th hour (t12) after admission to the intensive care unit

No statistically significant difference was observed with regard to SBP, DBP and MBP values between the groups (p>0.05).

The troponin-T data are shown in Table 2. The troponin-T values following the removal of the cross-clamp and those at the 24th postoperative hour were significantly lower in the sevoflurane group when compared to those in the isoflurane group (p<0.05).

Within the sevoflurane group, troponin-T values at the 2nd and 24th postoperative hours were significantly higher when compared to the values prior to induction (p<0.05). No statistically significant difference was observed upon comparison of troponin-T values at 2nd and 24th postoperative hours. However, a decrease in the 24th postoperative hour troponin-T value was observed with relation to its 2nd postoperative hour value.

Table 2. Troponin-T, creatine kinase and creatine kinase MB values of the cases

Variables	Group 1 (n=20)		Group 2 (n=20)		p*
	Mean±SD	Median (min-max)	Mean±SD	Median (min-max)	
Troponin T, ng/ml					
1	0.50±0.45	0.49 (0.445-0.54)	0.476±0.22	0.474 (0.46-0.49)	0.409
2	1.015±0.75	1.10 (0.935-1.185)	1.469±0.150	1.397 (1.29-1.645)	<0.001
3	6.112±0.524	6.1 (5-7)	4.11±0.53	4.1 (3.2-5.1)	<0.001
4	5.340±0.654	5.20(4.37-5.96)	8.71±1.03	8.8 (7.2-10.8)	<0.001
Creatine kinase, IU/L					
1	89.25±3.44	99 (87-96)	97.15±9.99	98 (79-106)	0.002
2	216.10±6.22	217 (205-227)	143.2±11.64	144 (127-171)	<0.001
3	449.1±3.1	448 (446-452)	252.5±4.8	254.5 (247.5-262.0)	<0.001
4	768.05±5.56	769 (760-780)	465.85±8.17	466.5 (450.0-486.0)	<0.001
Creatine kinase MB, IU/L					
1	26.75±2.41	26.8 (23.0-31)	27.85±3.39	26.5 (23.0-33.0)	0.244
2	48.15±2.28	48.2 (45.0-53.0)	52.35±2.70	53 (48-57)	<0.001
3	113.35±3.12	113 (109-118)	87.30±3.57	87 (80-95)	<0.001
4	104.75±3.46	103.8 (99.0-110.0)	155.60±4.88	156 (146-163)	<0.001

Data are expressed as mean±SD values and median(minimum-maximum) values

*Unpaired t-test and Mann-Whitney U test

1-before induction of anesthesia, 2-after aortic clamping, 3-at the 2nd postoperative hour, 4-at the 24th postoperative hour

Within the isoflurane group, troponin-T values at the 2nd and 24th postoperative hours were significantly higher when compared to the values prior to induction (p<0.05). However, there was a statistically significant difference in the troponin-T values at the 24th and 2nd postoperative hours (p<0.05).

The CK data pertaining to the study cases are shown in Table 2. The CK values obtained following after the removal of the cross-clamping, at the 2nd and 24th postoperative hours were found to be significantly higher in the sevoflurane group than in the isoflurane group (p<0.05).

Within the sevoflurane group, CK values following cross-clamp removal, at the 2nd and 24th postoperative hours were significantly higher when compared to control values (p<0.05).

Within the isoflurane group, CK values prior to induction and following cross-clamp removal were found to be significantly lower when compared to the values at the 2nd and 24th postoperative hours (p<0.05).

The CK-MB readings of the cases are shown in Table 2. The CK-MB values at 24th postoperative hour in the sevoflurane group were found to be significantly lower when compared to the 24th postoperative hour CK-MB values of the isoflurane group (p<0.05). The CK-MB values at 2nd postoperative hour in the isoflurane group were found to be higher when compared to the 2nd postoperative hour CK-MB values of the sevoflurane group, but the increase was not statistically significant (p>0.05).

The CK-MB values in the sevoflurane group prior to induction were higher than the values following cross-clamp removal, but the difference was not statistically significant (p>0.05). The CK-MB values obtained at the 2nd and 24th postoperative hours were significantly higher than the CK-MB values obtained prior to induction and following cross-clamp removal (p<0.05).

Within the isoflurane group, the CK-MB values obtained at the 2nd and 24th postoperative hours were significantly higher when compared to the values prior to induction (p<0.05). The second postoperative hour CK-MB values were higher than the values following cross-clamp removal, but the difference was not found to be statistically significant (p>0.05). The CK-MB values obtained at the 24th hour postoperatively were significantly higher than the values following cross-clamp removal (p<0.05).

Discussion

The results of our study showed that troponin -T and CK-MB values after aortic clamping and at the 24th postoperative hour were significantly lower in the sevoflurane group when compared to those in the isoflurane group.

Transient myocardial dysfunction following CPB is a well described condition. In addition to a sufficient revascularization, elective myocardial protection is needed for the ventricular function to be maintained. There are various factors affecting postoperative myocardial function and extent of myocardial injury (6, 7). Despite the great number of studies conducted, the mechanism of action of volatile anesthetic agents with regard to myocardial injury is not entirely elucidated. However, multiple

mechanisms have been reported to participate in anesthetic myocardial protection (3, 8-10). Volatile anesthetic agents, when administered prior to and following ischemia, have been reported to improve cardiac function and decrease the frequency of revascularization rhythm disturbances (11, 12). This feature has been linked to anesthetic preconditioning effect; however, volatile anesthetics have been reported to provide protection even when administered during the revascularization period only (4).

De Hert et al. (4) administered 4 different anesthesia protocols to the 200 patients scheduled to be subjected to CPB. The first group received intravenous propofol anesthesia, the second group was given sevoflurane until CBP, in the third group sevoflurane was initiated after the coronary anastomoses were completed, and the forth group was kept on sevoflurane until the surgery was completed. The results showed that the cardiac protective effect in the group on continuous sevoflurane administration during the surgery was clinically significant. This group had lower troponin-I levels and better cardiac function when compared with the group on intravenous propofol anesthesia. In the groups where sevoflurane was administered only prior to CBP and only after completion of the coronary anastomosis an earlier recovery of the postoperative stroke volume was noticed, but postoperative Troponin-I levels failed to demonstrate a significant difference when compared to the intravenous anesthesia group (4). Ebert et al. (13) compared sevoflurane and isoflurane in CPB cases basing on the HR increase produced by the surgery, and found the HR increase with sevoflurane to be less than that with isoflurane. It was observed that the HR of patients with coronary artery disease patients undergoing cardiac or noncardiac surgery was more stable with sevoflurane. Similarly, we found no significant difference between the two groups of our study with respect to increase in HR as a response to surgical stimuli. In the sevoflurane group, we did not found any statistically significant difference in the HR prior to induction when compared to the readings following induction, following skin incision, following sternotomy and during follow-up in the intensive care unit. On the other hand, the HR during the aortic side-clamp period, at the 10th and 20th minutes following pump detachment were higher when compared to the readings prior to induction. During the hypothermia period, a reduction in the metabolism and clearance rates of anesthetic agents is expected secondary to hemodilution and nonpulsatile perfusion (14). Immediately following the CPB ending, the heart rate of the surgically injured stunned myocardium increases secondary to increased clearance and increased metabolic rate. This explains the increase in the HR upon completion of the bypass surgery.

Searle et al. (15) used isoflurane and sevoflurane as a volatile anesthetic agents in patients scheduled for elective CPB surgery. No difference was noticed in the cardiovascular effects of either agent. However, they had not investigated the myocardial protection effects of these agents. There was a similar decrease in SBP, HR and cardiac index in both groups following anesthesia induction. Hemodynamic data remained stable following endotracheal intubation, skin incision and sternotomy. The authors' comment on their study results was that both agents seemed to be useful in providing for the hemodynamic control in low-risk CPB surgical patients. The incidence of intraoperative myocardial ischemia and postoperative morbidity and mortality incidences were found to be similar in both groups.

Bennett et al. (16) in their study which included 60 patients, maintained anesthesia with either sevoflurane or isoflurane at the beginning of surgery and evaluated myocardial injury using transesophageal echocardiography (TEE) and electrocardiography (ECG). Though recovery properties were found to be similar with equal doses of sevoflurane or isoflurane, sevoflurane was stated to be more useful.

Malagon et al. (17) compared three different anesthesia techniques with regard to their effects on myocardial protection in 90 pediatric patients scheduled for CPB surgery. Patients received midazolam, propofol and sevoflurane, and troponin-T values within the first 24 hours following surgery were compared. In all three groups troponin-T levels were found to be significantly high. As a result, based on troponin-T values, the myocardial protection in all of the three groups was found to be equal. They found that contrary to adult coronary bypass surgery, sevoflurane did not reduce troponin-T levels significantly, when compared to propofol.

Hemmerling et al. (18) compared the cardioprotective effects of sevoflurane and isoflurane in off-pump cardiac bypass surgery. Unlike our study, it has been shown that sevoflurane and isoflurane provide the same ischemic cardioprotective effects.

Cardiac troponin-T is a protein found in the heart muscle and is specific for myocardial tissue. This protein is quite sensitive for myocardial necrosis (19). It is not encountered in the blood of healthy subjects. Jaffe et al. (20) defined cardiac troponin as more useful and more sensitive markers of myocardial injury when compared to CK-MB.

Troponin-T level was one of the parameters we used to evaluate the extent of myocardial injury in our study. We found that its values were higher following cross-clamp removal (median value sevoflurane: 1.01, isoflurane: 1.46), at the 2nd (sevoflurane: 6.11, isoflurane: 4.11) and 24th postoperative hours (sevoflurane: 5.34, isoflurane: 8.71), when compared to values prior to induction (sevoflurane: 0.5, isoflurane: 0.48). However, the increase following the 2nd postoperative hour was observed to be less in the isoflurane group as compared to the sevoflurane group. We also observed that creatine kinase values, one of our other parameters, were higher when compared to its pre-induction levels. The increase in CK in the sevoflurane group was more than that observed in the isoflurane group, as indicated by the values following cross-clamp removal (median value sevoflurane: 216, isoflurane: 138) at the 2nd (sevoflurane: 449 isoflurane: 449 isoflurane: 252) and 24th postoperative hours (sevoflurane: 768, isoflurane: 465). We observed that CK-MB values, too, increased in both of the groups as compared to pre-induction values. Based on measurements of 24th (median value

sevoflurane: 104, isoflurane: 155) postoperative hour levels, we found CK-MB values to be significantly lower in the sevoflurane group than in the isoflurane group. None of the cases in our study suffered perioperative myocardial infarction, and all patients completed the study. Postoperative complications were found to be similar for both groups of the study.

Study limitations

There are limitations to our study. Firstly, histopathological species may support the blood data. Secondly, long-term follow-up of patients for cardiac function may have been included in our study.

Conclusion

Based on our results, we conclude that by providing sufficient anesthesia level and hemodynamic stability, both of the inhalational anesthetics, sevoflurane and isoflurane. In our opinion, sevoflurane provides a better myocardial protection than isoflurane, as may be inferred by the lower levels of the myocardial injury markers troponin-T and CK-MB observed with sevoflurane. Further studies are necessary to address the potential influence of choices of anesthetic regimens on long- term cardiac function after coronary surgery.

Conflict of interest: None declared.

References

1. De Hert SG, Cromheecke S, ten Broecke PW, Mertens E, De Blier IG, Stockman BA, et al. Effects of propofol, desflurane and sevoflurane on recovery of myocardial function after coronary surgery in elderly high-risk patients. Anesthesiology 2003; 99: 314-23.
2. Nyhan D, Johns RA. Anesthesia for cardiac surgery procedures: In: Miller's Anesthesia. 6th ed. Miller RD, editor. Philadelphia: Churchill Livingstone; 2005. p. 1941-2004.
3. De Hert SG, Turani F, Mathur S, Stowe DF. Cardioprotection with volatile anesthetics: mechanisms and clinical implications. Anesth Analg 2005;100:1584-93.
4. De Hert SG, Van der Linden PJ, Cromheecke S, Meeus R, Neils A, Van Reeth V, et al. Cardioprotective properties of sevoflurane in patients undergoing coronary surgery with cardiopulmonary bypass are related to the modalities of its administration. Anesthesiology 2004;101:299-310.
5. Landoni G, Bigmani E, Oliviero F, Zangrillo A. Halogenated anaesthetics and cardiac protection in cardiac and non-cardiac anaesthesia. Ann Card Anaesth 2009;12:4-9.
6. Tanoka K, Ludwig LM, Krolikowski JG, Alcindor D, Pratt PF, Kersten JR, et al. Isoflurane produces delayed preconditioning against myocardial ischemia and reperfusion injury: role of cyclooxygenase-2. Anesthesiology 2004;100:525-31.
7. Paparella D, Cappabianca G, Visicchio G, Galeone A, Marzovilla A, Galio N, et al. Cardiac Troponin I release after coronary artery bypass grafting operation: effects on operative and midterm survival. Ann Thorac Surg 2005;80:1758-64.
8. Tanaka K, Ludwig LM, Kersten JR, Pagel PS, Warltier DC. Mechanisms of cardioprotection by volatile anesthetics. Anesthesiology 2004;100:707-21.
9. Kersten JR, Schmeling TJ, Pagel PS, Gross GJ, Warltier DC. Isoflurane mimics ischemic preconditioning via activation of K (ATP) channels: reduction of myocardial infarct size with an acute memory phase. Anesthesiology 1997;87:361-70.
10. Cason BA, Gamperl AK, Slocum RE, Hickey RF. Anesthetic-induced preconditioning: previous administration of isoflurane decreases myocardial infarct size in rabbits. Anesthesiology 1997;87:1182-90.
11. Preckel B, Schlack W, Comfère T, Obal D, Barthel H, Thämer V. Effects of enflurane, isoflurane, sevoflurane and desflurane on reperfusion injury after regional myocardial ischemia in the rabbit heart in vivo. B J Anaesth 1998;81:905-12.
12. Schlack W, Preckel B, Stunneck D, Thämer V. Effects of halothane, enflurane, isoflurane, sevoflurane and desflurane on myocardial reperfusion injury in the isolated rat heart. B J Anaesth 1998;81:913-9.
13. Ebert TJ, Harkin CP, Muzi M. Cardiovascular responses to sevoflurane: a review. Anesth Analg 1995;81:S11-22.
14. Kayhan Z. Klinik Anestezi Kardiyovasküler cerrahide anestezi. 2nd ed. Ankara; Logos Yayıncılık; 1996.
15. Searle NR, Martineau RJ, Conzen P, al-Hasani A, Mark L, Ebert T, et al. Comparison of sevoflurane/fentanyl and isoflurane/fentanyl during coronary artery bypass surgery. Sevoflurane venture group. Can J Anaesth 1996;43:890-9.
16. Bennett SR, Griffin SC. Sevoflurane versus isoflurane in patients undergoing coronary artery bypass grafting: a hemodynamic and recovery study. J Cardiothorac Vasc Anesth 1999;13:666-72.
17. Malagon I, Hogenbirk K, van Pelt J, Hazekamp MG, Bovill JG. Effect of three different anaesthetic agents on the postoperative production of cardiac troponin T in pediatric cardiac surgery: B J Anaesth 2005;94:805-9.
18. Hemmerling T, Olivier JF, Le N, Prieto I, Bracco D. Myocardial protection by isoflurane vs. sevoflurane in ultra-fast-track anaesthesia for off-pump aortocoronary bypass grafting. Eur J Anaesthesiol 2008;25:230-6.
19. Katus HA, Looser S, Hallermayer K, Remppis A, Scheffold T, Borgya A, et al. Development and in vitro characterization of a new immunoassay of cardiac troponin T. Clin Chem 1992;38:386-93.
20. Jaffe AS, Ravkilde J, Roberts R, Naslund U, Apple FS, Galvani M, et al. It's time for a change to a troponin standard. Circulation 2000;102:1216-20.