Effects of high thoracic epidural anesthesia on mixed venous oxygen saturation in coronary artery bypass grafting surgery

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Background: To investigate possible effects of high thoracic epidural anesthesia (HTEA) on mixed venous oxygen saturation (SvO_2) in coronary artery bypass grafting surgery (CABGS).

Material/Methods: Sixty-four patients scheduled for CABGS were randomly assigned to either test (HTEA) or control group. Standard balanced general anesthesia was applied in both groups. Mean arterial blood pressure (MAP), heart rate (HR), oxygen saturation (SpO_2), central venous pressure (CVP), cardiac output (CO), cardiac index (CI), systemic vascular resistance (SVR), pulmonary vascular resistance (PVR), mean pulmonary arterial pressure (PAP), pulmonary capillary wedge pressure (PCWP), pulmonary compliance (C), bispectral index (BIS), body temperature, SvO_2, hematocrit values were recorded before induction. Postoperative hemodynamic changes, inotropic agent, need for vasodilatation, transfusion and additional analgesics, recovery score, extubation time, visual analogue scale (VAS) values, duration of stay in intensive care unit (ICU) and hospital were recorded.

Results: Study groups were similar in SpO_2, CVP, PCWP, PAP, C, body temperature, BIS values, development of intraoperative bradycardia. In HTEA group, intraoperative MAP, SVR, PVR, need for transfusion were lower, whereas CO, CI, SvO_2, hematocrit values were higher (p<0.05). Postoperative MAP, HR, hypertension development, need for vasodilatator, transfusion, analgesics, extubation time, recovery data, duration of stay in ICU, hospital were lower in HTEA group (p<0.05). VAS score decreased in 30 minutes and 12 hours following extubation in HTEA and control group, respectively.

Conclusions: HTEA may improve balance between oxygen presentation and usage by suppressing neuroendocrin stress response; provide efficient postoperative analgesia, more stabile hemodynamic, respiratory conditions, lower duration of stay in ICU, hospital.

Key words: coronary arteries bypass grafting surgery • mixed venous oxygen saturation • thoracic epidural anesthesia

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Background

It is quite well known that increased metabolism during and after coronary artery bypass grafting surgery (CABGS), impaired immune response and hemodynamic, respiratory and thromboembolic complications are mostly due to the unsuppressed neuroendocrin stress response [1–6]. Sympathetic activity leading to hypertension, tachycardia and increased metabolism impairs platelet activation and peripheral blood flow on the one hand and impairs peripheral oxygen saturation on the other which leads to paradox ischemia despite the high FiO_2 level. Therefore, it is critical to determine the balance between oxygen delivery (DO_2) and consumption (VO_2) in CABS [4,7].

Mixed venous oxygen saturation is an important parameter in determining general tissue oxygen balance. It is closely related with the arterial oxygen content and cardiac output. Mixed venous oxygen saturation (SvO_2) has been suggested to be more sensitive than the hemodynamic parameters to indicate early findings of sympathetic activity [8].

Respiratory complications are mainly due to patient's inability to use the protective mechanisms such as cough, deep breath and sigh which arise from not only the pump lung but also insufficient pain control [9]. Improvement in postoperative diaphragm and respiratory functions has been reported with high thoracic epidural anesthesia (HTEA) [10,11]. Compliance (C) is a widely-used parameter in anesthetized or mechanically ventilated patients in intensive care unit (ICU) to evaluate respiratory functions [12]. It has been reported that HTEA suppresses mediator release, significantly decreases need for analgesics,

minimizes respiratory complications by perfect postoperative pain control eventually enabling early extubation and discharge from intensive care unit [1,13–15].

Possible effects of HTEA in patients with coronary artery diseases have not been clarified, yet. Therefore, the present study was planned to investigate likely influences of HTEA on SvO_2, hemodynamic and respiratory parameters in CABGS.

Material and Methods

This prospective, randomized and controlled study was conducted with ethical principles, including the World Medical Association's Declaration of Helsinki, as revised in 2008. The study was approved by the Ethics Committee of the Medical School, Pamukkale University. Written informed consent was received from each individual before their entry to the study. A total of 64 patients were included in the study between July 2010 and January 2011 (Figure 1).

All patients were scheduled for CABS in elective conditions with ASA II-III. There were 46 male and 18 female patients with an age range of 40–79 years. Exclusion criteria were; hypersensitivity towards any of the chemicals to be used, contraindication of epidural anesthesia (dermal infections, nervous system diseases, severe hypovolemia, high intracranial pressure, severe aorta stenosis, severe mitral stenosis etc.), patients with a history of vertebral surgery, cervical or thoracic vertebra arthritis, morbid obesity (BMI >35), coagulopathy, <40% ejection fraction (EF) and preoperative inotropic agent usage.
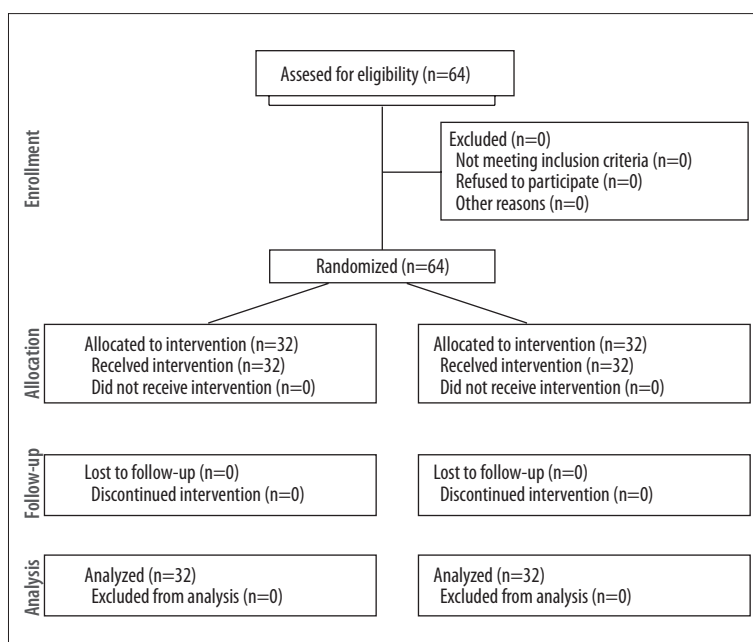
Figure 1. Consort diagram.

Patients were randomly assigned to the study groups by the closed envelope method. Pain during the postoperative follow-up period was assessed on a 10 cm visual analogue scale (VAS) anchored by verbal descriptors, "no pain" and "worst imaginable pain". Forms were given to all patients on the day of operation and they were asked to thick the value of pain at the end of each day.

Following venous access in the operation room, DII-V5 ECG, SpO2, nasopharyngeal body temperature, BIS and invasive arterial blood pressure were monitored with Datex Ohmeda Cardiocap 5. Invasive arterial blood pressure monitorization was done after radial artery cannulation with standard 20 gauge arterial cannula (BD Arterial Cannula, BD Critical Care Systems Pte Ltd., 198 Yishun Ave 7, Singapore). After local anesthesia under aseptic conditions with 2% lidocaine (Jetmonal 2% ampoule, Adeka Drug Ltd., Samsun, Turkey), a pulmonary arterial catheter (7.5 F REF: 774HF75 Edwards Lifesciences, USA) was placed through the right internal jugular vein with a single lumen sheat (8.5 F REF: I351BF85H Edwards Lifesciences LLC, Irvine, CA, USA). First measurements were performed after calibration of the instrument.

Anesthesia was induced as follows; preoxygenation with 100% oxygen for 1 min, 3–5 mg/kg thiopentale (Pental Sodium 1 gr flacon, İ.E. ULAGAY, Istanbul, Turkey) and 0.6 mg/kg rocuronium bromure (Esmeron 50 mg/5 ml flacon, N.V, Organon, Oss, Holland) intravenous bolus. Maintenance of anesthesia was provided with sevoflurane (Sevorane 250 ml solution, Abbott Laboratories Ltd., Istanbul, Turkey) in the mixture of 50% O_2 and 50% dry air to preserve BIS in the range of 40–50 and 0.5 µg/kg/min remifentanyl hydroclorur (Ultiva 2 mg flacon, GlaxoSmithKline Ltd., Istanbul, Turkey) infusion and with 0.15 mg/kg rocuronium bromur injection when required.

The patients were given lateral decubitis position in the HTEA group following anesthesia induction, intubation and achievement of hemodynamic stability. Under sterile conditions, epidural catheter (Braun Perifix 20 G) was placed at C6-C7 level at least 1 hour prior to heparin injection. Catheter was moved caudally 3–4 cm and fixed to the skin approximately at the level of T2-T4. After this induction, 0.075 mg/kg levobupivacaine hydroclorur (Chirocaine 5 mg/ml Abbott Lab., Istanbul, Turkey) + 2 µg/kg fentanyl (Fentanyl citrate, 50 µg/ml, Abbott Lab., Istanbul, Turkey) in total 10 ml bolus was given and 0.0375 mg/kg/h levobupivacaine + 0.5 µg/kg/h fentanyl epidural infusion was started with patient-controlled analgesia instrument (Abbott Pain Management Provider, Abbott Laboratoires, North Chicago IL, USA).

Blood pressure values as well as CVP, CO, CI, PVR, SVR, PCWP, PAP, SpO_2, body temperature and BIS values were recorded by CO monitor (Vigilance CO Monitor, REF: VGS2V, Edwards Lifesciences LLC, Irvine, CA, USA) in all patients before and after induction (before and after opening up the sternum) and

after CBP (before and after the closure of sternum). SvO_2 values were recorded before induction and also 5, 10, 15, 20, 40, 60-min after induction and 5, 10, 15, 20, 40, 60-min after CPB. Pulmonary compliance was determined in the study groups; after induction before opening the sternum, after ending CBP and closing the sternum, and during the postoperative follow-up (30-min, 1-hour, 2-hour, 4-hour and before extubation). Intraoperative hypotension or hypertension, development of bradycardia and disrhytmia, need for vasopressor or vasodilatator and transfusion were also recorded.

All patients were taken to the coronary surgery ICU in the postoperative period, monitored and mechanically ventilated. ECG, HR, SpO_2, body temperature, MAP, CVP and pulmonary compliance were determined by Avea mechanical ventilator (Viasys Respiratory Care Inc. 1100 Bird Centre Drive, Palm Springs, CA, USA) on 30-min, 1-hour, 2-hour, 4-hour and before extubation. Epidural infusion was continued during the patients' stay in ICU.

Intramuscular diclofenac sodium (Dikloron 75 mg 10 amp, Mefar Drug Ltd., Istanbul, Turkey) was injected as the analgesic agent in the control group. Analgesia was evaluated by asking patients to record VAS on 30-min, 1-hour, 2, 4, 8, 12, and 24 hours following extubation.

In case of hypotension (OAB <50 mmHg) development in ICU, crystalloid, colloid liquid, and efedryn (Efedrin 50 mg/ml amp, Osel Drug Ltd., Istanbul, Turkey) was given respectively. It was planned to administer 2.5–10 mg intravenous bolus and adrenaline (Adrenalin Biofarma 1 mg/ml ampul, Biofarma Drug Ltd., Istanbul, Turkey) with 0.05–1 mg intravenous bolus. In case of bradycardia, atropine (Atropin Sulphate 1 mg/ml, Biofarma Drug Ltd., Istanbul, Turkey) was administered with a dosage of 0.01–0.02 mg/kg. Blood transfusion was provided to preserve hematocrit value above 25%. If MAP exceeds 100 mmHg, nitroglycerin infusion (Perlinganit 10 ml amp, Adeka Drug Ltd., Samsun, Turkey) with a dosage of 0.5–10 µg/kg/min or beta blocker esmolol infusion (Brevibloc Premiks 10 mg/ml, 250 ml Inf. Solution, Eczacıbaşı-Baxter Ltd., Istanbul, Turkey) was used. Patients were extubated when they completely recover and regain muscular power (if the Aldrete's recovery score is 9, $PaCO_2$ <45 mmHg, PaO_2 >100 mmHg FiO_2 0.4 and pH is between 7.35–7.45 together with stabile hemodynamic and metabolic parameters) and VAS was recorded after extubation. Need for additional analgesics, recovery score, duration of stay in ICU and hospital were all recorded in the two study groups.

Statistical analysis

The primary outcome measure was mixed venous oxygen saturation. An a priori power analysis based on the previously published data suggested that a minimum of 32 patients

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Table 1. Demographic and operative data of study groups (mean ± standard deviation).

	HTEA	Control
Age (years)	62.8±10.5	61.7±8.8
Weight (kg)	75.6±12.1	75.6±9.2
Height (cm)	167.5±8	168.5±6.8
BMI	26.9±3.9	26.7±3.4
BSA (m²)	1.85±0.2	1.99±0.1
NYHA II/III	8/24	7/25
CPB Time (min)	137.7±54.1	134.9±44.2
EF (%)	49.4±11	51.3±11
Operation Time (min)	328.2±12.7	325.8±12.3
Number of Anastomoses	3.3±0.7	3.6±0.7
ASA II/III	8/24	7/25
Female/Male	8/24	10/22

There were no significant differences between the study groups in demographic or operative data (p>0.05).

in each group would be required to detect a 15% increase in mixed venous oxygen saturation, with a power of 90% at the 0.05 level of significance.

A statistical software program (SPSS 16.0 for Windows, SPSS Inc., Chicago, IL, USA) was used for statistical analysis. Qualitative parameters were compared between the study groups by Chi-Square and Fisher Exact tests. Numerical variables were compared by independent samples t test. Intragroup comparisons were made by repeated measures ANOVA test and Bonferroni Post-hoc test. All tests were performed at α=0.05 significance level.

Results

The study groups were similar in terms of demographic variables (Table 1). Data about MAP, HR, CO, CI, SVR, PVR and C are presented in Table 2. Before induction, MAP was similar in the study groups, but it was significantly lower in the HTEA group before and after the opening of sternum, before the closure of sternum (p<0.05). There was no significant difference between the groups after the closure of sternum and the data was within normal ranges. There are significant differences between the recordings before and after induction (p<0.05) (Table 2). Intraoperative HR and SpO$_2$ values were similar in the study groups. CO, CI and SVR were similar in the study groups before induction, but after induction CO and CI

were significantly higher and SVR was significantly lower in the HTEA group (p<0.05) (Table 2).

Pulmonary compliance values were higher in the HTEA group after the closure of sternum, but there was no significant difference between the study groups. Intragroup comparisons in the control group indicated that the recordings decreased significantly after the closure of sternum compared to those before induction (p<0.05) (Table 2). PVR and SVR were similar in the study groups before induction, but they were lower in the HTEA group after induction (p<0.05) (Table 2). CVP, PCWP, PAP were all within normal ranges and similar in the study groups at all recording time points (Table 2).

There was no significant difference in SvO$_2$ between values before and after induction, but it started to increase in the HTEA group at 10 min and stayed high until 60 min after CPB (p<0.05) (Table 3).

Hematocrit values before induction were 35.2±4.6% and 33.5±5.3% in the HTEA and control groups, respectively. At 5 min after CPB it was 31.4±4.6% and 28.2±4.1% in the HTEA and control groups, respectively (p<0.05). At 60-min after CPB it was 32±4.5% and 27.8±4.2% in the HTEA and control groups, respectively (p<0.05). Hematocrit values decreased significantly in the control group compared to the HTEA group (p<0.05).

Intraoperative complications such as hypotension (13–22%), disrythmia (12.5–16%), need for inotropic agent after CPB (15.6–22%) were all lower in the HTEA group, but there was no significant difference between the groups. There was significantly less need in the HTEA group than the control group for transfusion after CPB (56–84%) before the patient is transferred to ICU (p<0.01). Mean arterial blood pressure and heart rate were higher in the control group after the patients' transfer to ICU (p<0.05). Intergroup and intragroup comparisons revealed similar and within normal range recordings of postoperative SpO2, CVP and compliance (p<0.05) (Table 4). Development of postoperative hypertension was significantly higher in the control group (p<0.001). There were no significant differences in the frequencies of postoperative disryhtmia, bradycardia, hypotension need for inotropic agents. Vasodilatator agents and additional analgesics were more frequently needed in the control group during the ICU stay (p<0.001). Transfusion requirement was significantly less in the HTEA group (p<0.05), as less than 30% of patients in this group required transfusion. Recovery scores were less in the HTEA group (p<0.001). Extubation time, stay in ICU and stay in hospital were all shorter in the HTEA group (p<0.001) (Table 5).

Intergroup comparisons with regard to VAS indicated that the scores at 30 min, 1, 2, 4 and 8 hours were all less in the HTEA

Table 2. Intraoperative hemodynamic and respiratory parameters (mean ±SD).

		Before induction	Before opening of sternum	After opening of sternum	Before closure of sternum	After closure of sternum	p*
MAP (mmHg)	HTEA	84.6±12.3	71.8±10.4	67.9±9.5	68.1±6.9	72.6±6.3	**<0.05**
	Control	86.8±11.7	80.2±10.1	75.8±7.7	74.2±8.6	73.3±8.9	**<0.05**
	p**	>0.05	**<0.05**	**<0.05**	**<0.05**	>0.05	
HR (beat/min)	HTEA	79.6±8.3	72.6±6.7	70.9±6.6	73.5±6.6	73.3±6.6	**<0.05**
	Control	77±8.1	72.8±7.8	69.4±4.7	72.3±6.5	72.6±7.7	**<0.05**
	p**	>0.05	>0.05	>0.05	>0.05	>0.05	
SpO2 (%)	HTEA	97.8±1.3	98.8±0.9	98.9±0.9	99±0.9	98.8±0.9	>0.05
	Control	97.4±2.2	98.9±1.4	99±1.5	98.5±2.2	98.3±1.1	>0.05
	p**	>0.05	>0.05	>0.05	>0.05	>0.05	
CO (l/min)	HTEA	6±0.6	6.2±0.7	6.3±0.7	6.1±0.7	6±0.7	>0.05
	Control	5.9±0.7	5.2±0.6	5.3±0.9	5.1±0.8	5±0.7	**<0.05**
	p**	>0.05	**<0.05**	**<0.05**	**<0.05**	**<0.05**	
CI (l/min/m^2)	HTEA	3.2±0.4	3.3±0.4	3.4±0.5	3.3±0.5	3.2±0.5	>0.05
	Control	3.1±0.5	2.8±0.4	2.8±0.5	2.7±0.4	2.6±0.4	**<0.05**
	p**	>0.05	**<0.05**	**<0.05**	**<0.05**	**<0.05**	
SVR (din.sec/cm^5)	HTEA	1052±214	817±200	736±199	767±191	871±186	**<0.05**
	Control	1092±222	1086±228	981±258	1019±239	1063±299	**<0.05**
	p**	>0.05	**<0.05**	**<0.05**	**<0.05**	**<0.05**	
PVR (din.sec/cm^5)	HTEA	89.2±23	69.5±21	67.8±26	68.5±24	67.1±23	**<0.05**
	Control	92.1±26	91.5±26	95.5±27	91.6±20	90.9±24	>0.05
	p**	>0.05	**<0.05**	**<0.05**	**<0.05**	**<0.05**	
C (ml/cmH$_2$O)	HTEA		52±8.8	52.3±8.9	52.5±9.7	51.8±8.9	>0.05
	Control		52.9±8.9	51.8±9	50.6±10	46.6±9.1	**<0.05**
	p**		>0.05	>0.05	>0.05	>0.05	
CVP (cmH$_2$O)	HTEA	10.4±3.4	10.0±3.3	11.2±3.6	10.5±3.2	9.2±3.4	**<0.05**
	Control	11.3±4.2	11.2±3.3	12.7±4.1	10.9±3.8	9.6±4.1	**<0.05**
	p**	>0.05	>0.05	>0.05	>0.05	>0.05	
PCWP (mmHg)	HTEA	15.6±4.7	16.4±4.8	15.9±3.7	13.9±3.6	13.7±3.5	**<0.05**
	Control	15.6±3.6	15.1±3.6	14.6±3.2	13.2±2.3	12.4±2.6	**<0.05**
	p**	>0.05	>0.05	>0.05	>0.05	>0.05	
PAP (mmHg)	HTEA	22±4.9	21.6±4.5	21±4.2	19±4	18.5±3.4	**<0.05**
	Control	22.7±4.5	21.2±4	21±3.9	18±2.5	18±2.6	**<0.05**
	p**	>0.05	>0.05	>0.05	>0.05	>0.05	

* Indicates statistical analyses of serial evaluation in each group; ** indicates statistical analyses of two groups.

group than the control group (p<0.001). The VAS in the control group approached to that of the HTEA group only at hour-12.

Discussion

Surgery stress, insufficient pain control, hypercoagulability, release of proinflammatory cytokines, and activation of neuroendocrin system are among the major factors determining postoperative morbidity [16,17]. HTEA improves the balance between oxygen presentation and usage in ischemic myocardium via selective blockage of cardiac sympathetic innervation (T1-T5) [11,17]. Thoracic epidural anesthesia decreases myocardial infarcts by 40% and protects lung functions during the postoperative 24 hours [18,19].

The risk for spinal hematoma due to the heparinisation, haemodilution and coagulation factors limit the usage of HTEA in open cardiac surgeries [20,21]. Goldstein et al. [13] gave heparin at least 1 h after placement of epidural catheter and reported no neurological complication in patients with normal coagulation test results who are not under anticoagulation therapy. Accordingly, systemic anticoagulation was applied at least 1 h after placement of epidural catheter in the present study and our findings are similar as no complication was detected.

Table 3. Intraoperative SVO$_2$ values in the study groups (mean ± SD).

SVO$_2$ (%)	HTEA	Control	P*
Before anesthesia induction	69±6.8	67.6±6.9	>0.05
After anesthesia induction	68.4±7.1	65.7±7.2	>0.05
After epidural Bolus, 5.min	70.6±7.8	68±8.5	>0.05
After epidural Bolus, 10.min	71.3±6.4	63.2±6.6	**<0.05**
After epidural Bolus, 15.min	75.9±8.9	64.4±5.9	**<0.05**
After epidural Bolus, 20.min	74.6±7.2	62.6±9.7	**<0.05**
After epidural Bolus, 40.min	77.5±9.7	69.4±7.9	**<0.05**
After epidural Bolus, 60.min	81.7±10.4	65.2±10.1	**<0.05**
After CPB, 5 min	82.4±9.2	68.8±8.6	**<0.05**
After CPB, 10.min	79±9.7	65.2±8.1	**<0.05**
After CPB, 15.min	84±11	66.6±7.7	**<0.05**
After CPB, 20.min	86.4±10.8	67±9.4	**<0.05**
After CPB, 40.min	84.9±12.1	67.7±8.8	**<0.05**
After CPB, 60.min	86.1±9.7	67.9±10.1	**<0.05**
P**	**<0.05**	**<0.05**	

* Indicates statistical analyses of serial evaluation in each group; ** indicates statistical analyses of two groups.

Table 4. Postoperative MAP, HR, SpO2, CVP and C values (mean±SD).

		Min-30	Hour-1	Hour-2	Hour-4	Pre-extubation	p*
MAP (mmHg)	HTEA	71.9±13.4	72.1±14	75±12	75.8±11.4	73.1±14.5	**<0.05**
	Control	74.5±12.7	84.8±13.4	83.2±12.5	86.2±12	86.3±13	**<0.05**
	p**	>0.05	**<0.05**	**<0.05**	**<0.05**	**<0.01**	
HR (beat/min)	HTEA	77±10.6	76.8±9.3	77.6±8.6	78.1±9.4	78±11.4	>0.05
	Control	83.5±9.2	84.3±11	85.4±10.5	83±11.3	83.7±10.6	>0.05
	p**	**<0.05**	**<0.05**	**<0.05**	**<0.05**	**<0.05**	
SpO2 (%)	HTEA	98.7±0.8	98.5±0.6	98.7±0.5	98.6±0.6	98.9±0.6	>0.05
	Control	98.6±1	98.7±0.9	98.7±0.7	98.5±0.8	98±0.8	>0.05
	p**	>0.05	>0.05	>0.05	>0.05	>0.05	
CVP (mmHg)	HTEA	8.9±2.2	9±2.3	8.6±2.1	9±1.8	9.2±1.7	>0.05
	Control	9.4±2.7	9.1±2.3	9±2	8.7±2	8.9±1.4	>0.05
	p**	>0.05	>0.05	>0.05	>0.05	>0.05	
C (ml/cmH$_2$O)	HTEA	51.5±6.9	52.3±6.3	52.4±6.3	52.2±6	53.6±6.5	>0.05
	Control	49.2±9.6	49±7.1	49.9±7.2	50.7±6.9	51±7.3	>0.05
	p**	>0.05	>0.05	>0.05	>0.05	>0.05	

* Indicates statistical analyses of serial evaluation in each group; ** indicates statistical analyses of two groups.

In patients undergoing CABGS, the transient sympathetic blockage provided by HTEA has been reported to decrease perioperative myocardial ischemia, regulate coronary perfusion, and significantly improve functions of left ventricle [14,22–24]. Mendez-Tellez [25] stated that intrathoracic blood volume, PVR and hypoxic pulmonary vasoconstrictor response may be induced by the change in pulmonary vascular tonus due to the sympathetic disinnervation, and HTEA may reduce PVR through the vasodilatation in pulmonary blood vessels. Accordingly, we found similar intraoperative SpO$_2$, CVP, PCWP, PAP and C in the study groups, whereas in the HTEA group MAP, HR, SVR and PVR measurements and intraoperative hypotension, disrhythmia development, need for

Table 5. Postoperative complications in the study groups (N and%) and (Mean ±SD).

	Control		HTEA	
	N	**%**	**N**	**%**
Hypotension	4	13	5	16
Hypertension*	15	47	2	6.2
Bradycardia	1	3.1	2	6.2
Dysrhythmia	3	9.3	3	9.3
Need for inotrop	7	22	6	19
Need for vasodilatators*	15	47	2	6.2
Need for additional analgesics*	22	69	1	3.1
Transfusion 0**	11	34	22	69
Transfusion 1**	15	47	6	19
Transfusion 2**	4	13	2	6.2
	Mean ±SD		**Mean ±SD**	
Aldrete's Recovery Score 9 (h)*	4.7±1.9		2.9±1.8	
Extubation time (h)*	6.8±2		4.1±1.7	
Duration of stay in ICU (days*)	3.6±1.8		1.5±1.6	
Hospitalization period (days)*	8.8±1.7		4.9±1.5	

* $p < 0.001$; ** $p < 0.05$.

inotropic and transfusion were all less and CO, CI, SvO_2 and Htc measurements were higher than the control group.

Kessler et al. [14] have placed the thoracic epidural catheter at T1-T2 or T2-T3 level, while Fillinger et al. [24] placed it between the T1 and T10 levels in a cranial direction. In the present study, we placed the thoracic epidural catheter at the C6-C7 level in a caudal direction. We think this application provided stabile MAP and HR in the HTEA group through the direct blockage of cardiac sympathetic nerves. The level of CO changes inversely with the change in systemic vascular resistance when the arterial pressure is stabile. Therefore, the increase observed in the CO and CI levels in the present HTEA group may be explained by the decrease in SVR.

The amount of oxygen presentation to tissues may be indirectly evaluated by the changes in DO_2 and VO_2. Mixed venous oxygen saturation is a marker of the balance between whole body DO_2 and VO_2. SvO_2 shows a positive correlation with CO, Htc and SaO_2 together with a negative correlation with VO_2. Therefore, continuous monitorization of SvO_2 enables simultaneous monitorization of these four parameters. Normally, peripheral VO_2 is independent from DO_2. When CO and DO_2

decrease, peripheral O_2 extraction increases to keep VO_2 stabile and as a result SvO_2 decreases. Sepsis on the other hand, decreases peripheral O_2 usage and thereby increases SvO_2 [12]. Lorentzen et al. [8] reported that CI, Htc, SpO_2, aortic valve surgery, vasodilatation treatment and VO_2 are the factors affecting SvO_2; they result in low SpO_2, Htc or CI, increased peripheral VO_2 during recovery as well as affecting veins. The present findings revealed increased intraoperative SvO_2 values in the HTEA group compared to the control group after the epidural injection. HTEA decreased usage of oxygen significantly leading to an increase in SvO_2 measurements. These findings may be explained by the suppressive effect of HTEA on sympathetic neuroendocrin stress response. Moreover, hypotension development was significantly less in the HTEA group during the intraoperative period, together with significantly less disrhythmia, less need for inotropic agent and less need for transfusion during the period after the CPB before the patient's transfer to ICU. These findings are in agreement with the previous reports and suggest that more stabile hemodynamic conditions can be provided by HTEA [1,5].

Kessler et al. [14] detected higher MAP measurements in the postoperative ICU in the general anesthesia group. In the present study, we found higher postoperative MAP values in the control group. Less hypertension seen in the HTEA group during the postoperative ICU period may be explained by the efficient pain control and sympatholitic affects of HTEA.

Early extubation has potential benefits such as decrease in intubation-related complications, fast normalization of cilia functions, early recovery of coughing reflex, less pulmonary atelectasis development, significant improvement in intrapulmonary shunt fraction and elimination of disadvantages like deterioration of venous return, decrease in cardiac flow [26]. Numerous studies have previously reported that HTEA decreases the duration of intubation [20,21,27–29]. Similarly, the present study revealed shorter intubation time in the HTEA group. Furthermore, pulmonary compliance measurement which is considered as a marker of postoperative lung functions was similar in the study groups. This finding may be due to the similarities in the duration of cardiopulmonary bypass and surgery.

Requirement for additional analgesics or methods as well as nausea and vomiting were also significantly less in the HTEA group. The segment where epidural catheter was placed and the type and volume of the local anesthetic agent used may explain these findings [14,20,21,23]. The VAS was also less in the HTEA group, whereas the control group revealed smaller VAS value only 12-hour after surgery. The requirement for additional postoperative analgesics was significantly more in the control group. The fast and efficient analgesia provided in the HTEA group explains this difference between the study groups.

Despite that HTEA has been reported to have no superiority in terms of stay in ICU [14], there are studies revealing less stay in ICU and hospital in the HTEA group than the systemic opioid group [15,27]. In the present study, mean duration of stay in ICU as well as hospital was significantly shorter in the HTEA group. These findings suggest that the quality of analgesia provided by HTEA enables early recovery of pulmonary functions without suppressing protective reflexes such as coughing and deep breath. Eventually, hospitalization becomes shorter by early mobilization of patients.

Conclusions

As a conclusion, HTEA may be suggested to improve the balance between oxygen presentation and usage by suppressing neuroendocrin stress response, provide efficient postoperative analgesia, more stabile hemodynamic and respiratory conditions, and finally lower the duration of stay in ICU and in hospital after early extubation.

Conflicts of interest

The authors declare that they have no conflicts of interest.

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