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Use of High-Thoracic Epidural Analgesia in Pulmonary Endarterectomy: A Randomized Feasibility Study

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ABSTRACT

Background: The suitability of combined high-thoracic epidural anesthesia for pulmonary endarterectomy was studied.

Methods: A prospective randomized clinical study was conducted in a university medical center from November 2005 to December 2006. The primary endpoint of this study was to evaluate perioperative hemodynamic data; secondary endpoints were to evaluate the duration of artificial ventilation, length of stay in the intensive care unit, and the impact on postoperative morbidity and mortality.

Results: The 16 patients in the study group received high-thoracic epidural anesthesia plus general anesthesia; the 16 control patients received total intravenous anesthesia alone. Hemodynamic parameters and drug use, as well as the time to extubation, rate of complications, postoperative pain, the length of intensive care unit stay, and mortality, were recorded. The 2 groups were comparable with respect to hemodynamic stability during induction of anesthesia. The study group patients had significantly lower sufentanil consumption (mean ± SD, 2.1 ± 0.7 μg/kg versus 9.1 ± 3.1 μg/kg; $P < .001$), a shorter period of artificial ventilation (34 ± 35 hours versus 52 ± 49 hours; $P = .0318$), and lower postoperative pain scores at 3 hours (0.10 ± 0.26 versus 0.93 ± 1.38; $P = .015$), 12 hours (0.14 ± 0.53 versus 0.93 ± 0.79; $P = .002$), and 24 hours (0.35 ± 0.49 versus 1.33 ± 1.04; $P = .007$).

Conclusions: This study has shown that combined epidural and general anesthesia is a suitable anesthetic option in patients who are selected for pulmonary endarterectomy. It provides hemodynamic stability and reduces the duration of tracheal intubation postoperatively and improves postoperative pain relief, although this option has not been shown to decrease either the length of the intensive care unit stay or mortality.

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INTRODUCTION

Chronic thromboembolic pulmonary hypertension (CTEPH) is an underdiagnosed disease that occurs following pulmonary embolus. The exact incidence of CTEPH is not known; however, studies have shown it to be more frequent than previously estimated—up to 3.8% in the 2 years following the first embolism [Pengo 2004].

Fibrotic remodeling of thrombotic masses in addition to secondary peripheral vasculopathy and stenosis results in blockage of pulmonary artery branches. Pulmonary arterial pressure increases along with pulmonary vascular resistance (PVR), and right heart failure gradually develops [Jamieson 2003]. Conservative therapy is often characterized by a poor prognosis, and only 10% of patients with a mean pulmonary artery pressure (MPAP) >50 mm Hg have a 5-year life expectancy [Riedel 1982].

Pulmonary endarterectomy (PEA) is a potentially curative therapy for CTEPH for a certain group of patients. The surgical procedure involves removal of obstructive material from both branches of the pulmonary artery together with the attached vascular layer. The in-hospital mortality rate ranges from 5% to 24% [Fedullo 2001], and the best results are achieved at centers with the greatest experience and effective interdisciplinary cooperation.

Anesthesia management usually includes a balanced technique using opioids, benzodiazepines, and volatile anesthetics, or a regimen of total intravenous anesthesia (TIVA) with continual or intermittent administration of opioids, propofol, and muscle relaxants [Manecke 2006; Roscoe 2008]. General anesthesia (GA) combined with high-thoracic epidural anesthesia (HTEA) is a safe and established method in cardiac anesthesia that may offer such benefits as earlier extubation, improved postoperative analgesia and pulmonary function, earlier ambulation, and improved myocardial protection and myocardial function among patients with decreased ventricular function [Liem 1992; Swenson 1994; Liu 1995; Desborough 1996; Fillinger 2002; Kiliçkan 2005].

We therefore designed a study to determine the suitability of HTEA for PEA by focusing on its potential advantages

compared with standard GA. These advantages include hemodynamic stability, duration of artificial ventilation (AV), pain management, and length of stay in the intensive care unit (ICU). We chose a randomized design to compare HTEA and GA using TIVA to eliminate potential bias caused by surgical conduct and disease severity.

MATERIALS AND METHODS

We prospectively studied 32 patients who gave informed consent and underwent isolated elective PEA between November 2005 and December 2006. The study protocol was approved by the local review committee. The patients were randomized to a standard anesthetic technique (GA with TIVA) or to HTEA by means of sealed envelopes. One surgeon performed the procedure on all of the patients, and anesthesia was provided by 1 of 3 fully trained anesthetists.

Warfarin treatment was stopped 5 days before surgery, and the prothrombin time was allowed to normalize. We administered 1 mg/kg enoxaparin (Clexane; Sanofi-Aventis, Paris, France) once daily in the morning. The last subcutaneous dose of low molecular weight heparin was administered 24 hours before the planned induction of anesthesia.

Study Group: HTEA plus GA

All patients were premedicated with 0.1 mg/kg of diazepam (Zentiva, Hlohovec, Slovak Republic) administered orally 1 hour before transfer to the operating room. On patient arrival, oxygen was administered by face mask, and peripheral venous and arterial cannulas were placed. The patient was placed in a sitting position, and an epidural puncture was performed with a Tuohy needle (18 gauge; B. Braun Melsungen, Melsungen, Germany) at least 60 to 90 minutes before planned systemic heparinization. The hanging-drop technique and a median approach at T2 to T5 were used. A polyurethane epidural catheter (EC) was inserted and fixed after negative aspiration of blood and cerebrospinal fluid was demonstrated. We then inserted a triple-lumen central venous cannula, a Swan-Ganz catheter, and a single-lumen jugular bulb catheter under local anesthetic blockade.

Approximately 20 to 25 minutes before the induction of anesthesia, a bolus dose of 7 mL of 0.25% bupivacain (Marcaine; AstraZeneca, Lund, Sweden) and 10 μg sufentanil (Sufenta; Janssen Pharmaceutica, Beerse, Belgium) was administered via the EC, followed by a continuous infusion of 0.25% bupivacain at 7 mL/h and sufentanil at 7 μg/h. Both bupivacaine and sufentanil are approved for epidural administration by the local State Bureau for Drug Control. After confirmation of an adequate epidural effect by measuring the loss of temperature discrimination, GA was induced with 10 μg sufentanil, 3 to 5 mg of midazolam (Dormicum; Roche, Prague, Czech Republic), 1 mg/kg propofol (Diprivan; AstraZeneca, London, UK), and 0.15 mg/kg cisatracurium (Nimbex; GlaxoSmithKline, Verona, Italy) administered intravenously. Following endotracheal intubation, the lungs were ventilated in pressure control mode with an inspired oxygen fraction (FiO_2) of 0.6 to 1.0 to achieve normocapnea (end-tidal CO_2 pressure, 30-35 mm Hg) and a pulse oximetry saturation (SpO_2) of 90% to 95%. Small boluses of ephedrine (Ephedrin; Biotika, SlovenskáĽupča, Slovak Republic) or norepinephrine (Noradrenalin; Zentiva) were used whenever significant hypotension (mean arterial pressure <60 mm Hg) or bradycardia (heart rate <55 beats/min) with hypotension occurred following induction of anesthesia. Rectal and nasopharyngeal thermometers, a urinary catheter equipped with a thermometric sensor, and a femoral artery cannula were inserted, and a BIS sensor (Aspect Medical Systems, Norwood, MA, USA) was attached. GA was maintained with a continuous infusion of propofol guided by a target BIS value of 40 to 50. Incremental doses of cisatracurium were administered only if interference with the ventilator was noted. Aprotinin (Gordox; Gedeon Richter, Budapest, Hungary) was administered intravenously (2,000,000 IU prior to induction of anesthesia plus 2,000,000 IU in the cardiopulmonary bypass [CPB] prime plus 50,000 IU continuously per hour throughout the entire procedure). A bolus of 3 mg/kg of heparin (Heparin Sandoz; Biochemie, Vienna, Austria) was administered to achieve an activated clotting time >480 seconds, which was maintained by additional bolus doses of heparin if necessary. The patients were cooled to <20°C immediately after institution of CPB. Deep hypothermic circulatory arrest (DHCA) was instituted once the following 3 conditions had been reached: a bladder temperature between 16°C and 18°C, a BIS value of 0, and a saturation of the jugular bulb blood of >95%. Immediately before DHCA, 500 mg thiopental (Thiopental; Valeant Pharmaceuticals International, Prague, Czech Republic), 30 mg/kg methylprednisolone (Solu-Medrol; Pfizer, Brussels, Belgium), and 15 mg/kg phenytoin (Epanutin; Pfizer, New York, NY, USA) were administered. Head cooling with ice bags was also part of the brain-protection protocol. The surgeon performed the endarterectomy of the right and left branches of the pulmonary artery during 2 periods of DHCA, with reperfusion carried out between these 2 periods. After the pulmonary artery was closed, CPB was recommenced along with controlled rewarming. After normothermia was reached, right atrioventricular epicardial pacing at 100 beats/min was achieved, and the patient was slowly weaned from CPB. Failure to achieve at least a 25% reduction in the MPAP led to the use of 20 μg of nebulized iloprost (Ventavis; Bayer Schering Pharma, Berlin, Germany). If the mean arterial pressure was <70 mm Hg, vasopressor support in the form of norepinephrine was administered. If the patient's hemodynamic indices continued to be unfavorable (increasing central venous, pulmonary, and left atrial pressures), dobutamine (Dobutrex; Eli Lilly, Indianapolis, IN, USA) infusion was added.

After transfer to the postoperative ICU, all patients were sedated overnight with a propofol infusion titrated to light sedation (level from –3 to –2 according to the Richmond Agitation Sedation Scale), which allowed both toleration of AV and neurologic monitoring. Epidural infusion of 0.1% bupivacain and 1 μg/mL sufentanil at a rate of 3 to 8 mL/h continued along with intravenous administration of 1 g of paracetamol (Perfalgan; Bristol-Myers Squibb, Rueil-Malmaison, France) 4 times daily. The tracheal tube was removed according to standard extubation criteria (full consciousness, SpO_2, >90%;

$PaCO_2$, <40 mm Hg; FiO_2, 0.4; spontaneous breathing with peak end-expiratory pressure [PEEP], <5 cm H_2O). Intravenous administration of heparin was started after the chest tube output dropped below 20 mL/h, and the dose of heparin was adjusted according to a target activated partial thromboplastin time (APTT) of 45 seconds to 55 seconds. Heparin administration was stopped on the second day after extubation, and the APTT was checked after 4 hours. Then, either the EC was withdrawn if the APTT level was within the laboratory reference range or EC withdrawal was delayed for a few more hours until APTT normalization. Heparin administration was then immediately restarted. Warfarin therapy was started after chest tube removal on postoperative day 4 or 5.

Control Group: TIVA

Premedication and cannulation were identical to those of the study group. Anesthesia was induced via intravenous administration of 0.5 μg/kg sufentanil, 3 to 5 mg midazolam, 1 mg/kg propofol, and 0.15 mg/kg cisatracurium and was maintained by continuous infusion of propofol and sufentanil with a BIS value maintained at 40 to 50. Incremental doses of cisatracurium were administered if interference with the ventilator was noted. Ephedrine or norepinephrine was used whenever needed following induction of anesthesia. Ventilation, brain protection, aprotinin and heparin administration, conduct of DHCA, weaning from extracorporeal circulation, inotropic or vasopressor support, and iloprost administration were all identical to the study group. Sufentanil administration was by intravenous infusion at a dosage of 0.1 to 0.2 μg/kg per hour and was titrated according to the patient's report of pain to the nurse after waking. Paracetamol was also administered (1 g intravenously 4 times a day). The sedation strategy and extubation criteria were identical to the study group.

Hemodynamic data (systemic mean arterial pressure, MPAP, PVR, systemic vascular resistance, heart rate, and cardiac index) were recorded before the start of the epidural infusion in the study group and just prior to induction of anesthesia in the control group (T0), immediately following sternotomy (T1), after patient arrival in the ICU (T2), and at 24 hours after surgery (T3). A trained nurse assessed patient postoperative pain by means of a standard visual analogue scale at 6, 12, and 24 hours following extubation and with the patient at rest. Other data recorded included the highest norepinephrine dose, the total propofol and sufentanil dose, the ephedrine and norepinephrine doses used during hypotension or bradycardia following induction, the need for inotropic support or iloprost, the duration of AV, the length of the ICU stay, and the occurrence of postoperative complications (pulmonary, neurologic, and renal). Pulmonary complications were specified as radiographically proven reperfusion edema necessitating prolonged AV (>48 hours). Neurologic complications were specified either as mild when agitation, delirium, and disorientation were manifested or as severe when ictus, transient ischemic attack, coma, or other signs of cerebral vascular accident occurred. Renal complications were defined as a worsening of renal function necessitating renal-replacement therapy.

Table 1. Preoperative Data*

Parameter	HTEA + GA (n = 16)	TIVA (n = 16)
Age, y	60 ± 11.1	46.6 ± 13.8
M/F sex, n	13/3	8/8
BMI, kg/m^2	25.5 ± 4.1	26.5 ± 3.4
NYHA class (1-4)	3.2 ± 0.5	3.2 ± 0.4
6MWT, m	323 ± 92	328 ± 98
Tricuspid regurgitation score (1-4)	3.0 ± 0.7	3.1 ± 0.7

*Data are presented as the mean ± SD where indicated. HTEA + GA indicates high-thoracic epidural anesthesia and general anesthesia; TIVA, total intravenous anesthesia; BMI, body mass index; NYHA class, dyspnea classification according to the New York Heart Association; 6MWT, 6-minute walking test.

Data were statistically analyzed with Statistica 6.0 (StatSoft, Tulsa, OK, USA). We used the Fisher exact test in 4-field tables to compare the distributions of discrete variables. We analyzed the distributions of continuous variables among the groups with the Kolmogorov-Smirnov test and verified the results with other tests (Mann-Whitney test and the 2-tailed test). Repeated-measures analysis of variance was used to compare the differences in hemodynamic variables between the groups. A difference was considered statistically significant for P values <.05. Data are presented as the mean ± SD for continuous variables and as percentages or numbers for discrete variables.

RESULTS

We enrolled 32 patients, 16 in each group. Demographic data are presented in Table 1. There were no significant demographic differences between the 2 groups.

Data for operative variables are presented in Table 2. There were no differences between the 2 groups in the lowest temperature achieved, the length of DHCA, extracorporeal circulation, cross-clamp time, and doses of vasopressors needed during postinduction hypotension or bradycardia.

Table 2. Perioperative Data*

Parameter	HTEA + GA (n = 16)	TIVA (n = 16)
ECC, min	315 ± 44	331 ± 34
CCT, min	124 ± 26	127 ± 22
DHCA, min	39 ± 12	43 ± 12
Minimum temperature, °C	15.6 ± 0.9	15.2 ± 0.7
Ephedrine, mg	11 ± 11.4	8.7 ± 11.5
Norepinephrine, μg	71.3 ± 57.4	76.9 ± 65.3

*Data are presented as the mean ± SD. HTEA + GA indicates high-thoracic epidural anesthesia and general anesthesia; TIVA, total intravenous anesthesia; ECC, extracorporeal circulation; CCT, cross-clamp time; DHCA, deep hypothermic circulatory arrest; minimum temperature, lowest core temperature recorded during surgery; ephedrine, cumulative dose used during hypotension with bradycardia following induction; norepinephrine, cumulative dose used during hypotension following induction.

Table 3. Hemodynamic Data and Time Course*

	Technique	T0 (Baseline)	T1 (After Sternotomy)	T2 (After Surgery)	T3 (POD1)
CI, L/min per m^2	HTEA + GA	1.9 ± 0.3	1.6 ± 0.5	2.8 ± 0.8†	2.9 ± 0.6†
	TIVA	1.8 ± 0.4	1.4 ± 0.4§	2.9 ± 0.6†	3.0 ± 0.5†
PVR, dyn·s/cm^5	HTEA + GA	942 ± 329	829 ± 356	157 ± 162‡	129 ± 89‡
	TIVA	994 ± 286	820 ± 228	155 ± 87‡	129 ± 75‡
SVR, dyn·s/cm^5	HTEA + GA	2552 ± 500	2043 ± 770‡	953 ± 239‡	1029 ± 334‡
	TIVA	2595 ± 777	2484 ± 1065	1050 ± 435‡	1027 ± 240‡
MPAP, mm Hg	HTEA + GA	52 ± 8	49 ± 10	29 ± 7‡	24 ± 7‡
	TIVA	55 ± 6	52 ± 13	31 ± 7‡	24 ± 5‡
MAP, mm Hg	HTEA + GA	75 ± 10	62 ± 4‡	78 ± 20	83 ± 17
	TIVA	74 ± 9	66 ± 8‖	75 ± 10	79 ± 10
HR, beats/min	HTEA + GA	79 ± 9	67 ± 7‡	102 ± 6†	100 ± 6†
	TIVA	73 ± 9	73 ± 10	104 ± 9†	102 ± 9†

*Data are expressed as the mean ± SD. POD1 indicates postoperative day 1; CI, cardiac index; HTEA + GA, high-thoracic epidural anesthesia and general anesthesia; TIVA, total intravenous anesthesia; PVR, pulmonary vascular resistance; SVR, systemic vascular resistance; MPAP, mean pulmonary artery pressure; MAP, mean arterial pressure; HR, heart rate.

†$P < .001$, increase versus T0.

‡$P < .001$, decrease versus T0.

§$P = .0016$, decrease versus T0.

‖$P = .0051$, decrease versus T0.

Table 3 and Figure 1 compare the hemodynamic data for the 2 groups. There were several notable differences. Compared with the baseline (T0), the cardiac index decreased in the control group following the induction of GA (T1), whereas systemic vascular resistance and heart rate decreased in the study group at T1. At the remaining time points (T2 and T3), both groups showed decreases in PVR, systemic vascular resistance, and MPAP and showed significant increases in cardiac index and heart rate compared with the baseline (T0).

Postoperative data are presented in Table 4. The study group had significantly shorter times to tracheal extubation (34 ± 35 hours versus 52 ± 49 hours) and lower sufentanil consumption (2.1 ± 0.7 μg/kg versus 9.1 ± 3.1 μg/kg). Moreover, the study group had lower postoperative pain scores following extubation at each time point than the control group (3 hours, 0.10 ± 0.26 versus 0.93 ± 1.38; 12 hours, 0.14 ± 0.53 versus 0.93 ± 0.79; and 24 hours, 0.35 ± 0.49 versus 1.33 ± 1.04).

One patient in each group died, and the 2 groups did not differ with respect to complication rates or the lenght of ICU stay. EC insertion was successful in all patients in the study group, and there were no neurologic complications related to the use of epidural anesthesia. No severe neurologic complications occurred in either group.

DISCUSSION

HTEA accompanying GA is an accepted method in cardiac anesthesia. This study has shown that it is also feasible in PEA, a particularly specialized procedure with marked hemodynamic changes and prolonged DHCA. There may indeed be some benefit in terms of weaning from mechanical ventilation and pain therapy, but this study has not demonstrated any significant difference in the length of ICU or hospital stay, or, indeed, mortality. The small sizes of the groups may have contributed to this result, and further research in this area is required.

Even though epidural anesthesia has been used successfully in patients with primary pulmonary hypertension [Weiss 2000], some effects of central neuroaxial blockade are still considered risky in patients with pulmonary hypertension and right ventricle dysfunction in which hypotension may lead to critical coronary hypoperfusion. The onset of central neuroaxial blockade may lead to a reduction in the venous return, arterial pressure, and heart rate because of sympathetic blockade. Bradycardia was indeed a feature in the study group. The cardiac index was maintained, however, and hypotension was not a feature. Hemodynamic changes following anesthesia induction, which were similar in both groups, may be due to the institution of positive-pressure ventilation and the state of anesthesia itself rather than to the chosen mode of anesthesia delivery. In addition, PEEP has been shown to worsen the values of circulatory variables in patients with right ventricle dysfunction [Martin 1987]. Episodes of bradycardia or hypotension necessitating pharmacologic intervention are common following induction of anesthesia in patients with severe right heart failure.

The significant improvements in hemodynamic parameters following surgery in both groups of patients were an expected effect of the operation. Following successful PEA, the patient experiences substantial hemodynamic improvement, with the right ventricle working against significantly lowered resistance [Thistlethwaite 2006].

Tracheal extubation was achieved earlier in the study group. The known effects of HTEA on postoperative pulmonary function include improvements in diaphragmatic contractility [Pansard 1993] and vital capacity [Hendolin 1987]. In some studies, these apparently unique effects of

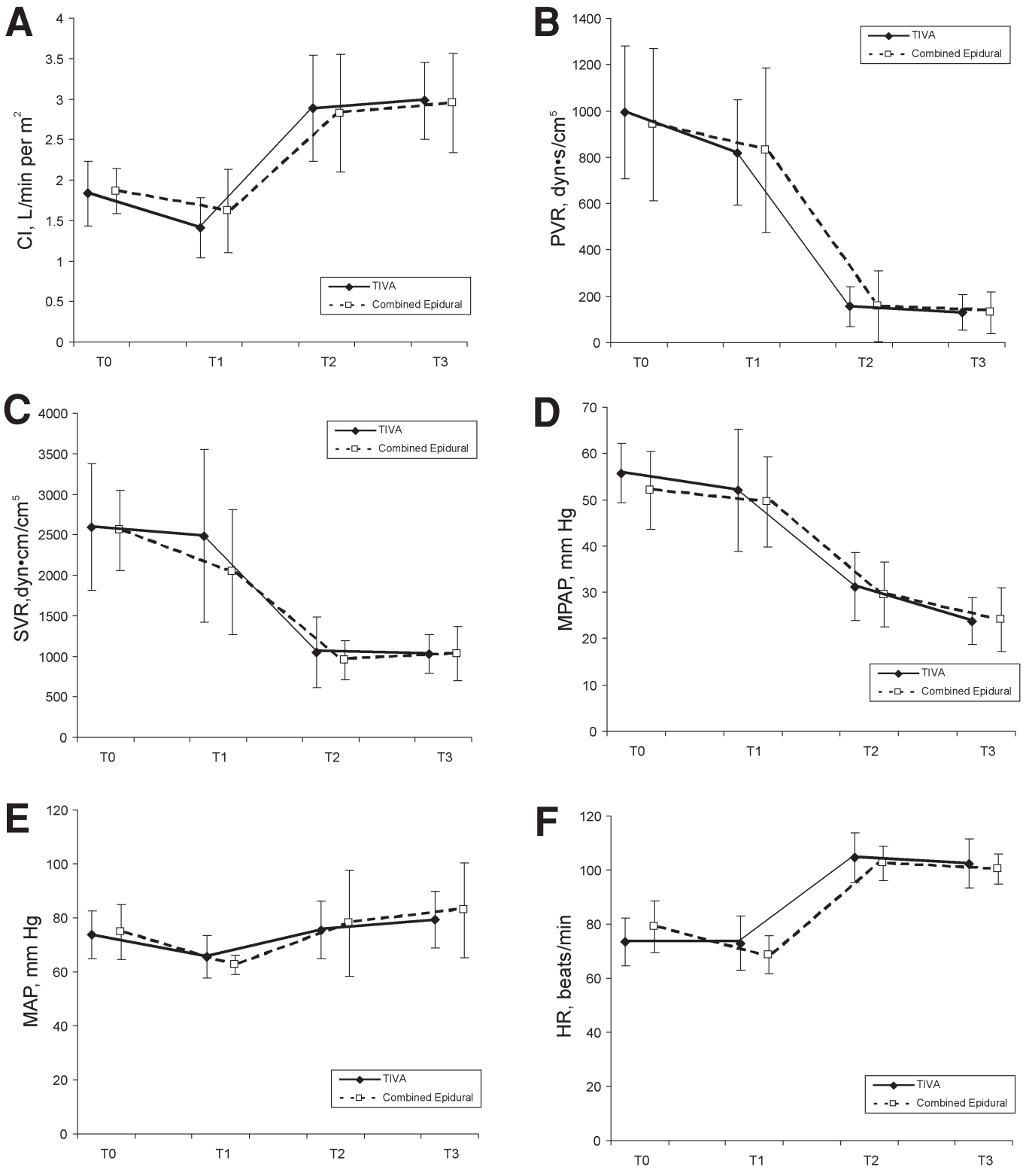

Figure 1. The development of the hemodynamic variables. T0, before the start of the epidural infusion in the study group and just prior to induction of anesthesia in the control group; T1, immediately following sternotomy; T2, after patient arrival in the intensive care unit; T3, 24 hours after surgery. A, Cardiac index (CI). B, Pulmonary vascular resistance (PVR). C, Systemic vascular resistance (SVR). D, Mean pulmonary artery pressure (MPAP); E, Mean arterial pressure (MAP); F, Heart rate (HR). TIVA indicates total intravenous anesthesia.

Table 4. Postoperative Data*

Parameter	HTEA + GA (n = 16)	TIVA (n = 16)
Norepinephrine, µg/kg per min	0.22 ± 0.13	0.55 ± 0.68
Propofol, mg/kg	22 ± 5	23.4 ± 7
Sufentanil, µg/kg	2.1 ± 0.7†	9.1 ± 3.1
AV, h	34 ± 35‡	52 ± 49
ICU stay, days	8.1 ± 1.9	7.2 ± 2.3
Pain score, 3 h (VAS)	0.10 ± 0.26§	0.93 ± 1.38
Pain score, 12 h (VAS)	0.14 ± 0.53‖	0.93 ± 0.79
Pain score, 24 h (VAS)	0.35 ± 0.49¶	1.33 ± 1.04
Mortality, n/%	1/6.25	1/6.25
Complications of ventilation, n/%	3/18.75	3/18.75
Renal complications, n/%	1/6.25	1/6.25
Neurologic complications, n/%	0/0	1/6.25

*Data are presented as the mean ± SD as indicated. HTEA + GA, high-thoracic epidural anesthesia and general anesthesia; TIVA, total intravenous anesthesia; norepinephrine, highest norepinephrine dose; propofol, perioperative propofol dose; sufentanil, perioperative sufentanil dose; AV, artificial ventilation; ICU stay, lenght of stay in the ICU; VAS, visual analogue scale; pain score, pain assessment at indicated times following extubation.

†$P < .001$, versus TIVA.

‡$P = .0318$, versus TIVA.

§$P = .015$, versus TIVA.

‖$P = .002$, versus TIVA.

¶$P = .007$, versus TIVA.

HTEA have translated into clinical benefits, including decreased pulmonary complications [Ballantyne 1998] and decreased duration of AV [Liem 1992; Swenson 1994]. Moreover, patients with limited right ventricular function may benefit more from shortening of the duration of intermittent positive-pressure ventilation, which decreases right and left ventricular preload, increases right ventricular afterload, and decreases cardiac output. Earlier tracheal extubation is associated with improved patient comfort, while decreasing sedation requirements and reducing the risk of postoperative infection. Pain scores were low in both groups but were especially low in the HTEA group. Thus, our data confirm the positive effect of epidural analgesia on postoperative pain control reported by other investigators [Priestley 2002].

It is necessary to point out that important major factors of the postoperative course, including a shortened time in the ICU, a shorter hospital stay, and early mortality, have not been demonstrated in this study or indeed in previous studies, perhaps because of the large number of confounding factors. We presume that the outcome following PEA depends on many variables and that anesthesia is only one of them.

In the present study, the 2 groups did not differ with respect to propofol consumption. Some investigators have used propofol in anesthesia for patients with primary pulmonary hypertension [Martens 2001]. Others, however, do not recommend propofol for induction of anesthesia in patients undergoing PEA because of a fear of its negative inotropic effect [Manecke 2006]. This study has shown that propofol may be administered to patients undergoing PEA without producing major hemodynamic deterioration. Moreover, Boyd and coworkers [1994] proved its benefit on right ventricular function, and Choi et al [2007] showed recently that propofol can even provide protection against reperfusion injury after prolonged cold ischemia. This effect might be helpful in PEA patients.

This study was not powered for safety or complications. The risk of development of an epidural hematoma and neurologic complications related to insertion of an EC is always a concern, and this risk may be exaggerated in cardiac surgery in which full heparinization is required [Ruppen 2006]. HTEA should be performed only by experienced clinicians. Among the more than 2600 patients in our institution who had thoracic epidural anesthesia in cardiac surgery over the last 10 years, there has been a zero incidence of severe complications associated with the epidural technique [Rubes 2005]. There also have been no instances of neurologic complications in the present study, but the limited number of patients does not allow a definitive conclusion. According to the second ASRA Consensus Conference on Neuraxial Anesthesia and Anticoagulation [Horlocker 2003], the following precautions are recommended for patients who require surgery on CPB: (1) Neuraxial blocks should be avoided in patients with known coagulopathy; (2) surgery should be delayed 24 hours in the event of a traumatic tap; (3) the time from instrumentation to systemic heparinization should exceed 60 minutes; (4) the heparin effect and its reversal should be tightly controlled (i.e., use of the smallest amount of heparin for the shortest duration compatible with the therapeutic objectives); and (5) ECs should be removed when normal coagulation is restored, with patients monitored postoperatively for signs and symptoms of hematoma formation. In addition, needle placement should occur at least 24 hours following the last heparin administration. In our protocol, we have followed all of these precautions.

We conclude that HTEA plus GA is a possible anesthetic option in patients with chronic right heart failure secondary to CTEPH who undergo PEA. Hemodynamic stability is noted, along with a significant shortening of AV and an improvement in postoperative pain management, although this study has not shown this factor to decrease the length of stay in the ICU or improve the outcome.

The small sample size mentioned above is one limitation of the present study; however, to prove safety of epidural anesthesia in this type of surgery, its effect on mortality, or its effect on reducing of the risk of postoperative infection would require enrollment of a very large number of patients. We therefore performed a feasibility study of HTEA in this group of patients.

Another limitation may be a lack of blinding, although it is technically very difficult to blind these 2 groups, one of which has an EC connected with a line and infusion pump. Nevertheless, decisions on the exact time of extubation and the transfer of patients from the ICU were made by colleagues who were not involved in the study and were not familiar with the grouping of the study.

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