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A hemodynamic evaluation of propofol/fentanyl compared with isoflurane/fentanyl anesthesia in coronary artery bypass grafting

Takeshi Kubota[1], Akimasa Miyata[2], Asahei Maeda[2], Kazuyoshi Hirota[1], Hironori Ishihara[1], and Akitomo Matsuki[1]

[1] Department of Anesthesiology, University of Hirosaki School of Medicine, Hirosaki 036-8216, Japan
[2] Department of Anesthesia, Hachinohe City Hospital, Hachinohe 031-0011, Japan

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Introduction

Anesthetic technique for coronary artery bypass grafting (CABG) requires minimal hemodynamic changes and adequate suppression of reflex responses to surgical stimuli to maintain the balance between myocardial oxygen demand and supply. Skin incision and sternotomy are particularly potent surgical stimuli in the prebypass period, and inadequate depth of anesthesia may cause myocardial ischemia and dysrhythmias accompanied by increases in plasma catecholamine levels [1].

Although high-dose fentanyl anesthesia produces hemodynamic stability, intraoperative awareness and postoperative prolonged respiratory depression sometimes occur [2–4]. Therefore, to solve these problems, balanced anesthetic techniques, such as the combination of moderate doses of fentanyl with halogenated agents [5], benzodiazepines [6], or propofol [7,8], have been common for cardiac surgery. Although comparison of the effects of propofol-fentanyl and isoflurane-fentanyl anesthesia on hemodynamics in patients undergoing CABG has been reported in several institutes in Europe [9,10], hemodynamic data showed little difference between the two anesthetic techniques.

Because propofol became available in Japan recently (in 1995), there are few domestic reports, although racial differences between Caucasians and Asias in responses to several agents have been reported [11]. In this study, we compared the hemodynamic effect of propofol-fentanyl anesthesia and isoflurane-fentanyl anesthesia in the prebypass period in Japanese patients undergoing CABG.

Address correspondence to: T. Kubota
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Methods

Twenty-four patients aged 54 to 76 years scheduled to undergo CABG were studied. The ejection fraction in all patients was greater than 0.45. The patients had been given β-blockers, calcium-channel antagonists, or nitrates until the day before the operation (Table 1), and were premedicated with 10 mg of oral diazepam and 75 mg of roxatidine 90 min before the induction of anesthesia and 5–10 mg of rectal morphine 30 min before induction. Continuous monitoring of the electrocardiogram (leads II and V_5) was commenced on arrival in the operating room. Radial arterial and peripheral venous lines were also established using local anesthesia.

Anesthesia was induced with 15 $\mu g \cdot kg^{-1}$ of fentanyl and 1–4 mg of midazolam. After loss of consciousness, 0.1 $mg \cdot kg^{-1}$ of vecuronium was administered to provide adequate neuromuscular blockade for tracheal intubation. The lungs were ventilated mechanically with an air–oxygen mixture (F_IO_2 = 0.4) to maintain normocapnea. A pulmonary artery catheter (Swan-Ganz catheter 7.5F, Baxter Healthcare, Irvine, CA, USA) was cannulated into the right internal jugular vein after the induction of anesthesia. Patients were randomly allocated to two groups: the propofol adminstration group (n = 12, group P) and the isoflurane administration group (n = 12, group I). In group P, after cannulation with the Swan-Ganz catheter, propofol infusion started at the rate of 6 $mg \cdot kg^{-1} \cdot h^{-1}$ through a central venous line, and its rate was reduced to 3 $mg \cdot kg^{-1} \cdot h^{-1}$ after sternotomy. In group I, isoflurane inhalation started at 0.5% inspiratory concentration until sternotomy was performed, and then its concentration was maintained

between 0.5% and 1.0%. The total doses of fentanyl were 20 μg·kg^{-1} and 25 μg·kg^{-1} up to skin incision and sternotomy, respectively. Vecuronium was administered intermittently as needed.

Heart rate (HR), systemic arterial pressure (AP), pulmonary artery pressure (PAP), central venous pressure (CVP), pulmonary artery wedge pressure (PCWP), and cardiac output (CO) were measured as hemodynamic variables just before propofol or isoflurane administration (pre-AD), 3 min after skin incision (post-SK), and 3 min after sternotomy (post-ST). Cardiac index (CI), left and right ventricular stroke work index (LVSWI and RVSWI, respectively), and systemic and pulmonary vascular resistance index (SVRI and PVRI, respectively) were also calculated.

Statistical analysis was performed by the unpaired *t*-test for between-group comparisons and a repeated-measures ANOVA followed by the Scheffe F test for intergroup comparisons. All data are expressed as means ± SD. A P value less than 0.05 was considered significant.

Results

The demographic data for the patients in the two groups are shown in Table 1. There were no significant differences between the groups.

The doses of midazolam at induction were 2.4 ± 1.6 mg and 2.2 ± 0.6 mg in groups P and I, respectively (difference not significant). None of the patients had intraoperative awareness, as confirmed by postoperative interviews. No myocardial ischemia or life-threatening arrhythmia was observed on the ECG.

The mean AP (MAP) and HR just before skin incision did not differ between groups P and I: MAP was 70 ± 19 and 72 ± 12 mmHg and HR was 56 ± 8 and 58 ± 8 · min^{-1}, respectively. The hemodynamic data are summarized in Table 2. MAP significantly increased post-ST as compared with pre-AD ($P < 0.05$), although there was no significant difference between groups P and I. In

Table 1. Demographic data

Variable	Group P	Group I
No. (M/F)	12 (8/4)	12 (7/5)
Age (yr)	64 ± 9	66 ± 6
Height (cm)	159 ± 8	154 ± 8
Weight (kg)	62 ± 12	57 ± 9
No. of grafts	2.7 ± 0.8	2.6 ± 0.8
Ejection fraction (%)	62 ± 8	59 ± 10
Preoperative medications (no. of patients)		
Nitrates	11	12
Calcium channel blockers	9	9
β-Blockers	3	4

All data are expressed as means ± SD.

Table 2. Changes in hemodynamic variables

Variable	Group	Pre-AD	Post-SK	Post-ST
HR	P	57 ± 6	62 ± 12	62 ± 10
(beats·min^{-1})	I	60 ± 12	63 ± 11	66 ± 9
MAP	P	81 ± 11	88 ± 17	93 ± 20*
(mmHg)	I	80 ± 11	86 ± 13	90 ± 11*
CVP	P	5 ± 2	6 ± 2	6 ± 4
(mmHg)	I	5 ± 4	6 ± 3	6 ± 3
MPAP	P	14 ± 4	15 ± 4	16 ± 6
(mmHg)	I	14 ± 5	15 ± 3	16 ± 4
PCWP	P	10 ± 3	11 ± 3	11 ± 6
(mmHg)	I	9 ± 4	10 ± 3	10 ± 3
CI	P	2.6 ± 0.7	2.5 ± 0.6	2.6 ± 0.5
(l·min^{-1}·m^{-2})	I	2.6 ± 0.6	3.1 ± 0.6*#	3.0 ± 0.7*
SVRI	P	2479 ± 727	2652 ± 921	2854 ± 874
(dynes·s·cm^{-5}·m^{-2})	I	2394 ± 628	2149 ± 467	2337 ± 633
PVRI	P	149 ± 74	142 ± 52	166 ± 99
(dynes·s·cm^{-5}·m^{-2})	I	166 ± 90	138 ± 55	174 ± 102
LVSWI	P	45 ± 12	44 ± 12	47 ± 12
(g·m·beat^{-1}·m^{-2})	I	42 ± 13	55 ± 14*	55 ± 21*
RVSWI	P	5.9 ± 2.4	4.9 ± 2.4	6.1 ± 2.2
(g·m·beat^{-1}·m^{-2})	I	5.4 ± 2.9	7.1 ± 2.5#	7.2 ± 3.6

All data are expressed as means ± SD. Pre-AD: just before propofol or isoflurane administration; Post-SK: 3 min after skin incision; Post-ST: 3 min after sternotomy; HR: heart rate; MAP: mean arterial pressure; CVP: central venous pressure; MPAP: mean pulmonary arterial pressure; PCWP: pulmonary capillary wedge pressure; CI: cardiac index; SVRI: systemic vascular resistance index; PVRI: pulmonary vascular resistance index; LVSWI: left ventricular stroke work index; RVSWI: right ventricular stroke work index.
* $P < 0.05$ vs. pre-AD. # $P < 0.05$ vs. group P.

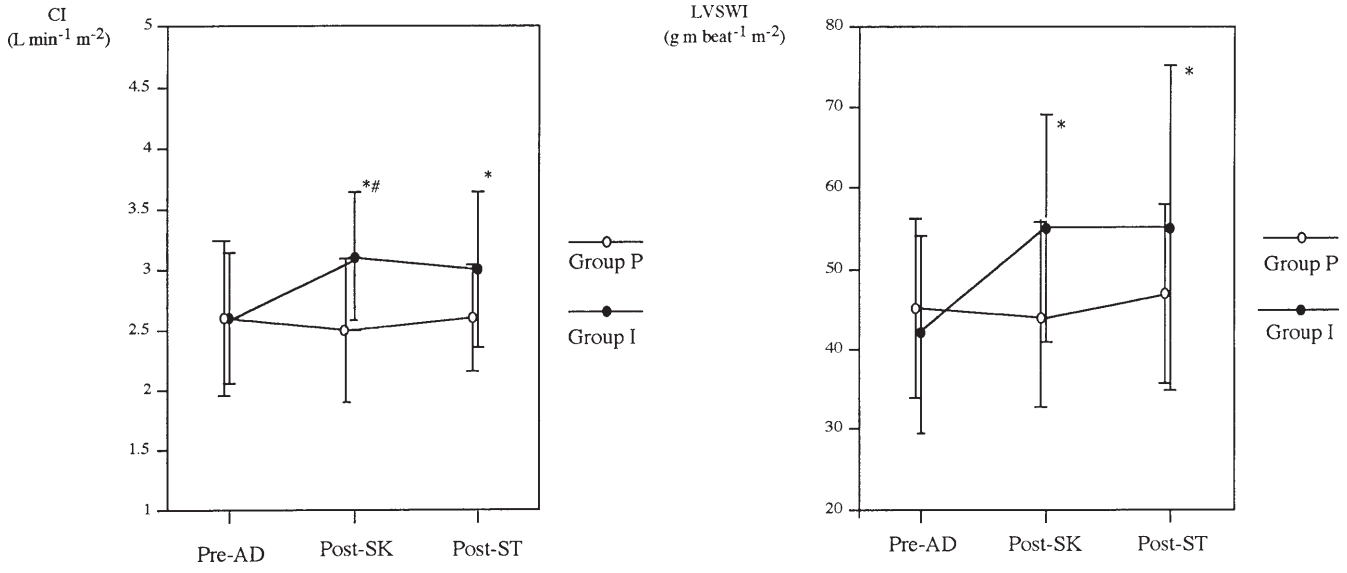

Fig. 1. In group I, Cardiac index (*CI*) and left ventricular stroke work index (*LVSWI*) significantly increased at post-SK and post-ST as compared with pre-AD, while those in group P did not. Moreover, CI at post-SK in group I was significantly higher than in group P. Pre-AD: just before propofol or isoflurane administration; post-SK: 3 min after skin incision; post-ST: 3 min after sternotomy, * $P < 0.05$ vs. pre-AD. # $P < 0.05$ vs. group P

group I, CI and LVSWI significantly increased post-SK and post-ST as compared with pre-AD ($P < 0.05$), whereas CI in group P did not (Fig. 1). Moreover, CI and RVSWI post-SK in group I were significantly higher than in group P ($P < 0.05$). Other hemodynamic variables did not change significantly in either group.

Discussion

The present study showed that although changes in MAP, MPAP, and HR were similar during propofol-fentanyl anesthesia and isoflurane-fentanyl anesthesia, CI and LVSWI were not changed by surgical stimuli during propofol-fentanyl anesthesia, whereas they significantly increased during isoflurane-fentanyl anesthesia, even though the same dose of fentanyl was used in both anesthesias. Although it is difficult to compare the anesthetic potency of inhaled and intravenous anesthetics, we considered that the two anesthetic methods may have similar anesthetic potency, because MAP and HR just before skin incision in both groups showed no significant difference. Moreover, in the previous study, we found that the plasma concentrations of the stress hormones such as norepinephrine and cortisol did not increase in the prebypass period under isoflurane-fentanyl anesthesia (isoflurane, 0.25%–1.0%; fentanyl, 30 μg·kg^{-1}), similar to the present anesthetic method [12]. Therefore, the depth of anesthesia in group I may be adequate against surgical stimuli. The absence of changes in CI and LVSWI suggests that propofol-fentanyl anesthesia may not change cardiac pump function during surgical stimuli. Therefore, it may prevent myocardial ischemia from increasing sympathetic tone caused by surgical stimuli. Phillips et al. compared the hemodynamics during anesthesia with propofol (4 mg·kg^{-1}·h^{-1}) and fentanyl (32.5 μg·kg^{-1} until sternotomy) with the hemodynamics is during anesthesia with isoflurane (1%) and fentanyl (32.5 μg·kg^{-1} until sternotomy) in patients undergoing CABG[9]. However, they found that LVSWI was significantly decreased (around 20%) by surgical stimuli in both anesthesia groups. Sorbara et al. also reported that both propofol (3 mg·kg^{-1}·h^{-1}) and isoflurane (0.6%) combined with fentanyl (30 μg·kg^{-1}) produced a slight depression of myocardial contractility (15.3% and 11.1%, respectively), as assessed by the end-systolic pressure-diameter relationship[10]. In both studies the doses of fentanyl used were higher than in our study, which might affect the data.

Propofol has been reported to have a negative inotropic property caused by calcium-channel blockade and reduction in sympathetic tone [13]. However, it may be antagonized by sympathetic responses to surgical stimuli. Therefore, appropriate control of the propofol infusion rate may provide adequate anesthesia and hemodynamics. On the contrary, a previous report demonstrated that CI may be relatively well maintained during isoflurane anesthesia [14]. In addition, isoflurane has been reported to produce vasodilatation [15].

Therefore, isoflurane might attenuate vasoconstriction but not the increase in cardiac contractility caused by sympathetic responses to surgical stimuli in the present study.

In conclusion, propofol-fentanyl anesthesia for CABG patients may be superior to isoflurane-fentanyl anesthesia in maintaining the myocardial oxygen balance because of the lack of increase in LVSWI after surgical stimuli.

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