J Anesth (1996) 10:296–299

Journal of
Anesthesia

Anesthetic management for surgical repair of postinfarction ventricular septal defect

MASAYASU ARAI, SEIJI KATO, and FUMIO GOTO

Department of Anesthesiology, Kitasato University School of Medicine, 1-15-1 Kitasato, Sagamihara 228, Japan

Key words: Postinfarction ventricular septal defect, Fentanyl anesthesia, Heart failure

Introduction

Postinfarction ventricular septal defect (PIVSD) in acute myocardial infarction is a complication that induces cardiogenic shock and biventricular failure [1–3]. Its incidence is low [1]. In patients receiving conservative therapy alone, the mortality rate was 100% within 2 months. Surgical repair of the rupture is thus considered to be the only effective treatment [3–5]. There have been several case reports on the anesthetic management used for individual PIVSD patients; however, there is no report comparing the hemodynamics of more than one patient treated with similar anesthetic techniques in the same hospital. The main object of the present report is to clarify the patient's preoperative status, the effects of the anesthetics, and the combined effect of several vasodilators on systemic circulation and biventricular function in PIVSD patients.

Patients and methods

Nine patients who underwent surgical repair of PIVSD following acute myocardial infarction (1987–1993) were examined retrospectively. All of the patients were anesthetized with fentanyl and benzodiazepine. The site of infarction, coronary segment of stenosis, surgical method, outcome, ASA anesthesia risk, preoperative treatment, and complications were examined. The duration to surgery after the onset of PIVSD, preoperative change of right heart pressure, shunt ratio, and use of inotropic drugs were studied. Intraoperative circulatory management and the total infused dose of fentanyl were also examined. Mean arterial pressure (MAP), heart rate (HR), central venous pressure (CVP), mean pulmonary artery pressure (MPAP), pulmonary artery occlusion pressure (PAOP), and temperature difference between esophagus and toe (ΔT) were measured six times: at S1, baseline (before induction of anesthesia); S2, following induction of anesthesia; S3, after sternotomy; S4, immediately before the start of cardiopulmonary bypass (CPB); S5, 1 h after aortic cross clamp; and S6, at the end of surgery.

Statistical comparisons were made by the nonparametric method using the Wilcoxon matched signed rank test for paired data. A P value of <0.05 was considered statistically significant and values are presented as means ± SD.

Results

The patients' ages ranged from 55 to 81 years (Table 1). The most common infarction site was the anterior wall (anteroseptal wall and/or extensive anterior wall). Inferior wall infarction was observed in 3 patients, and 2 of these 3 died (nos. 8 and 9). Both patients had total obstruction of the right coronary artery (segments 1 and 2). Three vessel diseases were observed in 5 patients (nos. 1, 4, 5, 8, and 9), and total obstruction of segment 6 or 7 was observed in 4 patients (nos. 1, 2, 5, and 6). Patch closure was performed in 8 patients. Aneurysmectomy was performed in 3 patients, and coronary arterial bypass graft surgery combined with patch closure was performed in 2. There were 7 patients who survived over 1 month. The patients' anesthesia risks were estimated to be ASA IIIE or IVE. In 8 patients, hyperglycemia was observed and was managed by infu-

Address correspondence to: M. Arai
Received for publication on October 24, 1995; accepted on June 7, 1996

Table 1. Site of infarction, surgical method, preoperative right heart condition, and outcome

Case	Age	Sex	AMI~PIVSD	PIVSD ~OPE	Site of infarction	Segment and degree of stenosis		Surgical method	ASA	CVP1 (mmHg)	CVP2 (mmHg)	MPAP1 (mmHg)	MPAP2 (mmHg)	Shunt ratio 1 %	Shunt ratio 2 %	Outcome
						LCA	RCA									
1	55	M	1	2	Ext.ant	seg 7; 100%, seg 11; 50%	seg 3; 50%	PC, A	4E	7	6	12	22	50	72	Survived
2	74	M	1	1	Ext.ant	seg 7; 100%, seg 11; 75%	(–)	DS, CABG	4E	8	11	23	20	77	77	Survived
3	76	M	6	5	Ant, Sept	seg 7; 99%	(–)	PC, A	4E	1	0	13	13	*	*	Survived
4	71	F	7	16	Ext. ant	seg 6; 90%, seg 7, 8; 90%	seg 4; 95%	PC, A	4E	4	9	16	24	80	71	Survived
5	68	F	2	7	Ant, Sept	seg 7; 100%, seg 9; 99%	seg 4; 75%	PC	3E	9	4	22	15	47	42	Survived
6	67	M	3	12	Ext. ant	seg 6; 100%	(–)	PC	3E	9	13	28	26	66	72	Survived
7	77	F	3	7	Inf, Ant	*	*	PC	4E	6	10	25	18	84	60	Survived
8	76	F	3	11	Inf, Ant	seg 7; 99%, seg 11; 75%	seg 2; 100%	PC, CABG	4E	13	11	18	18	50	57	Died
9	81	M	4	1	Inf, Post	seg 5; 90%, seg 6, 7; 90%	seg 1; 100%	PC	4E	14	12	19	21	83	77	Died

PIVSD, postinfarction ventricular septal defect; Ext. ant, extensive anterior wall; Ant, anterior wall; Sept, septal wall; Inf, inferior wall; Post, posterior wall; CVP, central venous pressure; MPAP, mean pulmonary artery pressure; LCA, left coronary artery; RCA right coronary artery; CABG, coronary artery bypass graft surgery; PC, patch closure; A, aneurysmectomy; DS, direct suture; CVP1/MPAP1/Shunt ratio 1, CVP, MPAP, or shunt ratio at the onset of PIVSD; CVP2/MPAP2/Shunt ratio2, CVP, MPAP, or shunt ratio on the day before surgery; AMI-PIVSD, duration after onset of acute myocardial infarction to onset of PIVSD (days); PIVSD-OPE, duration after onset of PIVSD to surgery (days). With the exception of case 7, the segment and degree of stenosis were assessed by preoperative coronary angiography; *, no available data.

sion of insulin. In 7 patients, renal dysfunction and hypoproteinemia were observed. Renal dysfunction was mainly managed by infusion of a diuretic agent; however, urine flow under 20 $ml \cdot h^{-1}$ was observed in most of these 7 patients just before surgery. Anemia was observed in 4 patients.

The duration from onset of PIVSD to surgery varied from 1 day to 16 days (Table 1). There were 6 patients who had emergency surgery (within 7 days from the onset of PIVSD). The main causes of emergency surgery were (a) increased doses of inotropics due to unstable hemodynamics and (b) a decrease in urine flow. Preoperative changes in CVP, MPAP, and shunt ratio resulting from conservative therapy are shown in Table 1. Patients no. 8 and 9, who had inferior wall infarction and total obstruction of the right coronary artery, had a high CVP from the onset of PIVSD. In most patients, there appeared to be no change in CVP, MPAP, and shunt ratio before and after conservative therapy. The preoperative mean intraventricular shunt ratio was 64.2 ± 11.1%.

Before surgery, most patients needed support with moderate to high doses of inotropics (Table 2). Isosorbide dinitrate was infused as a coronary vasodilator and was combined with intraaortic balloon pumping to increase systemic diastolic pressure and decrease afterload in all patients. This management was continued throughout the surgery. After the induction of anesthesia, some patients needed norepinephrine to maintain hemodynamics. Phentolamine 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ was infused for afterload reduction. The total of infused doses of fentanyl during surgery was 32.6 ± 20.5 $\mu g \cdot kg^{-1}$.

Hemodynamic data are shown in Fig. 1. MAP and HR gradually decreased; however, the change was not significant. CVP progressively increased following the induction of anesthesia and there was a significant increase before CPB (S4; 13.1 ± 2.3 mmHg) compared with the baseline (6.7 ± 4.3 mmHg) ($P < 0.05$). CVP at the end of surgery (13.6 ± 2.1 mmHg) was also significantly higher than the baseline value ($P < 0.05$). MPAP and PAOP did not change significantly at any time.

ΔT (the temperature difference between the esophagus and the toe) decreased significantly just before CPB (S4; 6.2 ± 3.4°C) and after CPB (S6; 2.7 ± 2.4°C) compared with the postinduction period (S2; 7.5 ± 3.3°C) ($P < 0.05$).

Discussion

Postinfarction ventricular septal defect is a rare complication with an incidence rate of 2% [1], and its prognosis is poor. Sanders et al. [2] reported that over 50% of the patients died within the 1 week, 13% survived the 2nd month, and 7.5% survived 1 year. Cummings et al.

Table 2. Preoperative and intraoperative circulatory management and anesthesia

	Infusion rate	Preoperative	Before CPB	After CPB
Dopamine	<9	5	4	4
	≧10	4	5	5
Dobutamine	<9	2	1	1
	≧10	3	4	1
Noradrenaline	<0.9	0	2	5
	≧1.0	2	4	4
ISDN or TNG	0.2–0.5	9	9	9
Phentolamine	5	0	9	9
IABP (1:1)	/	9	9	9

Values represent no. of patients.
Preoperative, time just before surgery; Before CPB, from induction of anesthesia to cardiopulmonary bypass; After CPB, after weaning from CPB to end of surgery; Infusion rate, $\mu g \cdot kg^{-1} \cdot min^{-1}$; ISDN, isosorbide dinitrate; TNG, trinitroglycerine; IABP, intraaortic balloon pumping; 1:1, IABP counterpulsation on each heartbeat.

[3] reported that the hospital mortality of patients who did not receive surgical therapy was 100%. Fatalities are characterized by inferior wall infarction and decreased right heart function, and the survival rate of surgical treatment was 58% [3]. In our patients, inferior wall infarction was observed in only 2 out of 9 patients, which resulted in the high survival rate in our study. It has been reported that delayed surgical correction is recommended to reduce surgical risks [6]. However, the appropriate time for surgical intervention is still controversial and in another report, early repair of the septal rupture is recommended to minimize multiple organ failure occurring during conservative drug therapy [7]. In our hospital, the basic plan for surgical management of PIVSD was to delay correction (3 weeks after onset of PIVSD) as much as possible except in patients who had impaired hemodynamics, progression of renal failure, or respiratory failure. However, no patient received 3 weeks of conservative therapy, for the reasons mentioned above (case nos. 3, 4, 5, 6, 7, and 8). In these types of patients, when delayed correction is selected, there are anesthesia risks not only with respect to the heart but also involving many multiple organ systems.

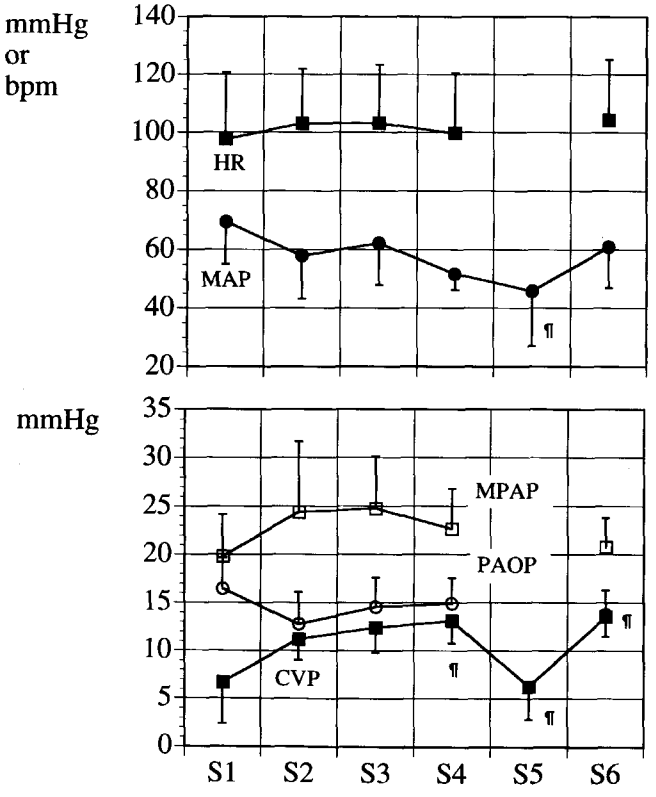

Fig. 1. Hemodynamic changes in heart rate (*HR*), mean arterial pressure (*MAP*), mean pulmonary arterial pressure (*MPAP*), pulmonary artery occlusion pressure (*PAOP*), and central venous pressure (*CVP*). ¶$P < 0.05$ *vs* S1

As shown in Table 1, CVP, MPAP, and shunt ratio did not change significantly from the onset of PIVSD to surgery in all patients. There were 2 patients with inferior wall infarction or total obstruction of the right coronary artery who had a high CVP value from the onset of PIVSD, suggesting impaired right heart function; however, the as with the other patients, the values did not improve during the preoperative period. Intraaortic balloon pumping (IABP) is widely recommended for medical management of PIVSD [8]. In our patients, IABP was combined just after the onset of PIVSD with other conservative therapy including infusion of inotropics. However, the data suggested that the condition of the right heart did not significantly improve with conservative therapy before anesthesia. The benefit of conservative therapy and delayed surgical correction for PIVSD should be further investigated.

Since the report of Stanley and Webster [9] on high-dose fentanyl anesthesia, this method has been widely used for poor-risk cardiac surgery; however, there have

been few reports on anesthesia using fentanyl in PIVSD surgery [4,5]. In our patients, HR, MAP, and MPAP did not change significantly after the induction of anesthesia (S2) and sternotomy (S3) using lower doses of fentanyl than those reported by Stanley and Webster. This data suggested that cardiac failure induced poor responses to surgical stress, regardless of the depth of anesthesia [10]. The total dose of fentanyl varied in each patient according to preoperative cardiac function; however, the dose of fentanyl we used before CPB was 19.5 ± 13.1, and the total dose was 32.6 ± 20.5 $\mu g \cdot kg^{-1}$, respectively, which was appropriate for minimum suppression of hemodynamics in PIVSD patients.

The increase in CVP suggested progression of right heart failure due to an intracardiac shunt, while MPAP showed a slight increase that was not statistically significant. The high CVP value after CPB suggested that right ventricular dysfunction was still present just after the surgical repair of PIVSD. The importance of right ventricular function in the prognosis of PIVSD [1,3] is already widely known. In our patients, the two with inferior wall infarction, both of whom died, had had a high CVP value from the onset of PIVSD, suggesting impairmed right heart function. Tecklenberg et al. reported on the beneficial effects of sodium nitroprusside for forward cardiac output, left-to-right shunt, systemic and pulmonary vascular resistance, and ventricular filling pressure in PIVSD patients [11]. We did not use sodium nitroprusside because it is not commercially available in Japan. Therefore, phentolamine was infused to reduce the afterload. According to Gould et al., phentolamine increases cardiac output, and decreases systemic vascular resistance, left ventricular filling pressure, and CVP in acute myocardial infarction [12]. From this report, we expected phentolamine to increase forward cardiac output and to decrease vascular resistance and ventricular filling pressure, as well as improve right ventricular function in PIVSD patients. We could not determine the exact direct beneficial effects of phentolamine on hemodynamics due to the shunt; however, the significant improvement of ΔT after CPB suggests that forward cardiac output increased due to disappearance of the intracardiac shunt, which was partially supported by vasodilators. Many factors can affect the change in ΔT, which is an indirect measurement of circulation; therefore, the ΔT must be carefully interpreted.

In conclusion, patients with emergency and/or delayed correction for PIVSD have many severe anesthetic risks to multiple organ systems. Right ventricular dysfunction persisted just after surgical repair of the defect. Careful titration of fentanyl has little effect on HR or MAP in these patients; however, CVP progressively increased. The use of vasodilators to protect the right heart and maintain forward cardiac output in patients with PIVSD may be recommended; however, it should be used carefully because exact estimation of the effects was difficult due to the intracardiac shunt.

References

1. Moore CA, Nygaard TW, Kaiser DL, Cooper AA, Gibson RS (1986) Postinfarction ventricular septal rupture: the importance of location of infarction and right ventricular function in determining survival. Circulation 74(1):45–55
2. Sanders RJ, Kern WH, Blount SG (1956) Perforation of the interventricular septum complicating myocardial infarction. A report of eight cases, one with cardiac catheterization. Am Heart J 51:736–748
3. Cummings RG, Califf R, Jones RN, Reimer KA, Kong YH (1989) Correlates of survival in patients with postinfarction ventricular septal defect. Ann Thorac Surg 47:824–830
4. Ikeda T, Kubota K, Aizawa K, Isshiki A, Shimizu T, Ishikawa M, Kudo T (1985) Emergency anesthesia of a patient with acute myocardial infarction complicated by perforation of the ventricular septum (in Japanese with English abstract). Masui (Jpn J Anesthesiol) 34:236–240
5. Sugiuchi N, Nagashima N, Seki I, Takahashi K (1988) Anesthetic management of two patients with ventricular septal perforation following acute myocardial infarction (in Japanese with English abstract). Masui (Jpn J Anesthesiol) 37:740–744
6. Campion BC, Harrison CE, Giuliani ER, Schattenberg TT, Ellis FH (1969) Ventricular septal defect after myocardial infarction. Ann Intern Med 70:251–261
7. Gaudiani VA, Miller DC, Stinson EB (1981) Postinfarction ventricular septal defect: An argument for early operation. Surgery 89:48
8. Gold HK, Leinbach RC, Sanders CA, Buckley MJ, Mundth ED, Austen WG (1973) Intraaortic balloon pumping for ventricular septal defect or mitral regurgitation complicating acute myocardial infarction. Circulation 47:1191–1196
9. Stanley TH, Webster LR (1978) Anesthtic Requirements and cardiovascular effects of fentanyl-oxygen and fentanyl-diazepam-oxygen anesthesia in man. Anesth Analg 57:411–416
10. Wynands JE, Wong P, Whalley DG, Sprigge JS, Townsend GE, Patel YC (1983) Oxygen-fentanyl anesthesia in patients with poor left ventricular function: hemodynamics and plasma fentanyl concentrations. Anesth Analg 62:476–82
11. Tecklenberg PL, Fitzgerald J, Allaire BI, Alderman EL, Harrison DC (1976) Afterload reduction in the management of postinfarction ventricular septal defect. Am J Cardiol 38:956–958
12. Gould L, Reddy CVR, Kalanithi P, Espina L, Gomprecht RF (1974) Use of phentolamine in acute myocardial infarction. Am Heart J 88:144–148