Anesthesia for patients with mitral valve disease secondary to rheumatic and coronary artery disease[1]

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Hemodynamic changes during anesthesia were studied in 53 patients undergoing mitral valve surgery. The patients were divided according to type of lesion (stenosis or insufficiency), etiology (rheumatic or ischemic), and anesthetic technique (halothane or fentanyl). One group was also treated with a vasodilator (sodium nitroprusside). Following intubation and except for minor variations, all patients showed a decrease in cardiac index (CI), an increase in systemic vascular resistance (SVR), and no change in central venous pressure (CVP) or pulmonary capillary wedge pressure (PCWP). These changes persisted following sternotomy and occurred irrespective of type of lesion, etiology, or anesthetic technique. Sodium nitroprusside resulted in an increase in CI and a decrease in SVR. Fentanyl anesthesia was characterized by a stable mean arterial pressure (MAP), whereas with halothane, MAP was labile.

Index terms: Anesthesia · Coronary disease · Fentanyl · Halothane · Mitral valve · Nitroprusside · Rheumatic heart disease

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Mitral valve disease carries special anesthetic risk. In the early experience of cardiac surgery, mitral valve disease was of rheumatic origin, and most patients had a prolonged course complicated by multiple drug therapy and a high incidence of congestive heart failure, electrolyte imbalance, low cardiac index (CI), and high systemic vascular resistance (SVR). However, in a large percentage of patients now undergoing surgery for mitral valve replacement, the disease is of ischemic origin. We undertook this study to

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evaluate the anesthetic management of patients with mitral valve disease in relation to (1) type of lesion and predisposing disease, (2) anesthetic used (fentanyl or halothane), and (3) hemodynamic response to vasodilators in the early stages of surgery.

Methods

In a prospective study, 51 patients undergoing surgery for mitral valve replacement were divided into two main groups. Group I (39 patients) was studied to evaluate the effects of the two anesthetic agents and to detect any difference in the hemodynamic response related to type of valvular lesion or predisposing disease. At random, half of each group received halothane and half fentanyl (*Table 1*). Group II (12 patients) included those with various mitral valve lesions who were anesthetized with fentanyl and given a peripheral vasodilator, sodium nitroprusside (SNP) (*Table 4*).

All patients were premedicated with morphine sulfate, 0.05–0.15 mg/kg, and scopolamine, 0.4 mg intramuscularly, 30–45 minutes before transfer to the operating room. Lines were inserted to monitor systolic, mean, and diastolic pressures (SAP, MAP, and DAP), central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), heart rate (HR), and cardiac output (CO). The latter was calculated as the average of three outputs measured by thermodilution. The following values were derived: cardiac index (CI), systemic vascular resistance (SVR), and pulmonary vascular resistance (PVR). These measurements were determined (1) before induction (control), (2) three to five minutes postintubation (PI), and (3) five minutes postmedian sternotomy (PS). The data were analyzed by Student's t-test for paired and unpaired measurements.

In Group I-A (halothane), anesthesia was induced with 100–200 mg thiopental, intravenously (IV) until loss of the eyelash reflex. The patients were then manually ventilated via face mask with a mixture of 50% O_2 and N_2O with increasing concentrations of halothane, 0.5% to 3%. The concentration of halothane was adjusted so that the diastolic arterial pressure remained above 60 mm Hg. Concurrently, 0.1–0.15 mg/kg pancuronium bromide was injected IV to facilitate intubation.

Group I-B (fentanyl) received 2 mg pancuronium bromide IV followed by 80–100 μg/kg of fentanyl infused over two to three minutes; concurrently, pancuronium bromide, 0.15 mg/kg, was administered. The patients were ventilated by 100% oxygen. Endotracheal intubation was performed three to four minutes after administration of the total dose of fentanyl and pancuronium bromide.

Group II: Anesthesia was induced by fentanyl as described for Group I-B. Following induction of anesthesia and intubation, one or more of the following values were considered as an indication for the use of SNP: CI < 2.0 L/min/m^2, MAP > 90 torr, and PCWP > 20 torr. Sodium nitroprusside (0.3–1.5 μg/kg/min) was administered by continuous drip adjusted according to response.

A drop in MAP < 70 mm Hg, accompanied by a drop in PCWP > 5 mm Hg, was considered an indication for transfusion. Ringer's lactate solution was used.

Results (*Fig. 1*)

Rheumatic origin

Patients with mitral valve lesions secondary to rheumatic disease were significantly younger (56.4 ± 2.8 years) than those with lesions secondary to ischemia (68.2 ± 1.1 years). Hemodynamic responses to induction of anesthesia were as follows:

Mitral stenosis (14 patients) (*Table 1*): Fentanyl induction (7 patients) resulted in a significant increase in HR and SVR and a decrease in CI. Following sternotomy, SVR increased and CI decreased further.

Halothane induction (7 patients) resulted in an increase in SVR and a decrease in CI.

Mitral regurgitation (12 patients) (*Table 2*): Fentanyl induction (6 patients) significantly increased the HR. Following sternotomy, CI decreased ($P < 0.05$) and SVR increased ($P < 0.05$).

Halothane induction (6 patients) decreased MAP ($P < 0.05$). Following sternotomy, the CI decreased ($P < 0.05$).

The trend of increasing SVR in the fentanyl group following intubation differed significantly from that in the halothane subgroup (slight drop).

Ischemic origin (13 patients) (*Table 3*): All patients had mitral insufficiency.

Fentanyl induction increased the HR ($P < 0.001$). Following sternotomy, tachycardia persisted, CI decreased ($P < 0.01$), and SVR increased ($P < 0.05$).

Halothane induction increased HR ($P < 0.001$)

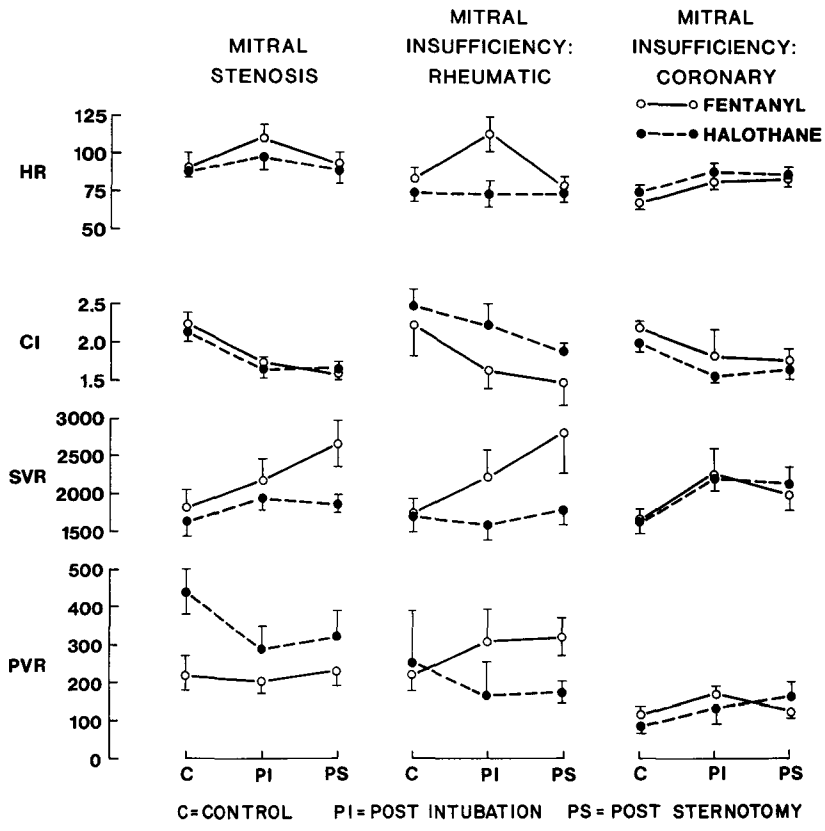

Fig. 1. Hemodynamic changes during mitral valve surgery (mean ± SEM).

and SVR ($P < 0.05$) and decreased CI ($P < 0.01$). These changes persisted following sternotomy.

Effect of Vasodilator Therapy (sodium nitroprusside) (14 patients) (*Table 4*; *Fig. 2*): Sodium nitroprusside was indicated in 12 of 14 patients (3 with mitral stenosis and 9 with mitral insufficiency). Five patients had a low CI, 3 a low CI and a high PCWP, 2 a low CI and a high MAP, and 2 an elevated MAP only. In mitral insufficiency, SNP resulted in an increase in CI ($P < 0.02$), a decrease in MAP ($P < 0.02$), and a decrease in SVR ($P < 0.01$). Mitral stenosis followed the same trend, but there were insufficient cases for statistical analysis.

Table 1. Hemodynamic changes during fentanyl and halothane anesthesia in mitral stenosis

	Control	Postintubation	Poststernotomy
HR (BPM)			
Fentanyl	92.3 ± 8.6	112.0 ± 8.7*	93.7 ± 8.5
Halothane	90.4 ± 4.7	98.4 ± 8.8	88.9 ± 7.7
MAP (torr)			
Fentanyl	90.0 ± 6.3	88.1 ± 6.4	97.0 ± 6.8
Halothane	81.1 ± 4.5	88.0 ± 3.3	75.0 ± 3.1‡
PCWP (torr)			
Fentanyl	20.9 ± 1.9	23.7 ± 1.9	21.3 ± 1.4
Halothane	26.9 ± 2.5	31.1 ± 3.6	25.9 ± 2.6
CI (L/min/m^2)			
Fentanyl	2.25 ± 0.14	1.75 ± 0.04*	1.59 ± 0.07†
Halothane	2.14 ± 0.12	1.66 ± 0.13†	1.64 ± 0.1*
SVR (d·sec·cm^{-5})			
Fentanyl	1825 ± 229	2183 ± 267*	2666 ± 314*
Halothane	1627 ± 201	1953 ± 178*	1876 ± 117‡
PVR (d·sec·cm^{-5})			
Fentanyl	227 ± 49	204 ± 29	234 ± 48
Halothane	441 ± 59	291 ± 54	323 ± 68

* Statistically different from control $P < 0.05$.
† Statistically different from control $P < 0.01$.
‡ Statistical difference between halothane and fentanyl $P < 0.05$.

Table 2. Hemodynamic changes during fentanyl and halothane anesthesia in mitral insufficiency (rheumatic)

	Control	Postintubation	Poststernotomy
HR (BPM)			
Fentanyl	84.6 ± 8.1	115.0 ± 12.0*	81.0 ± 5.7
Halothane	76.0 ± 7.4	75.5 ± 8.5†	76.5 ± 8.0
MAP (torr)			
Fentanyl	91.8 ± 4.2	87.4 ± 2.1	89.4 ± 5.9
Halothane	98.0 ± 7.1	83.2 ± 4.0*	81.5 ± 2.6*
PCWP (torr)			
Fentanyl	30.4 ± 4.6	32.2 ± 1.5	29.8 ± 3.8
Halothane	23.2 ± 5.3	17.7 ± 4.1	18.7 ± 4.6
CI (L/min/m^2)			
Fentanyl	2.23 ± 0.41	1.65 ± 0.25	1.47 ± 0.28*
Halothane	2.50 ± 0.21	2.26 ± 0.28	1.90 ± 0.10*
SVR (d·sec·cm^{-5})			
Fentanyl	1745 ± 186	2254 ± 333	2832 ± 549*
Halothane	1707 ± 205	1598 ± 197	1790 ± 189
PVR (d·sec·m^{-5})			
Fentanyl	225 ± 43	312 ± 84	323 ± 49
Halothane	255 ± 135	166 ± 81	178 ± 25†

* Statistically different from control $P < 0.05$.
† Statistical difference between halothane and fentanyl $P < 0.05$.

Table 3. Hemodynamic changes during fentanyl and halothane anesthesia in mitral insufficiency (coronary)

	Control	Postintubation	Poststernotomy
HR (BPM)			
Fentanyl	69.7 ± 4.2	84.1 ± 5.6‡	85.3 ± 3.8‡
Halothane	77.2 ± 4.0	91.7 ± 3.6‡	87.7 ± 5.3*
MAP (torr)			
Fentanyl	93.6 ± 4.4	94.9 ± 3.3	93.0 ± 4.0
Halothane	87.8 ± 6.7	91.0 ± 5.5	93.0 ± 5.3
PCWP (torr)			
Fentanyl	18.7 ± 2.9	25.8 ± 4.8	22.3 ± 2.6
Halothane	20.5 ± 2.7	22.8 ± 2.9	24.8 ± 2.9
CI (L/min/m^2)			
Fentanyl	2.25 ± 0.07	1.87 ± 0.35	1.80 ± 0.14†
Halothane	2.03 ± 0.12	1.60 ± 0.09†	1.67 ± 0.12†
SVR (d·sec·cm^{-5})			
Fentanyl	1686 ± 137	2299 ± 324	2027 ± 229*
Halothane	1678 ± 173	2254 ± 183*	2166 ± 216*
PVR (d·sec·cm^{-5})			
Fentanyl	124 ± 18	173 ± 22	131 ± 18
Halothane	92 ± 23	139 ± 45	171 ± 41*

* Statistically different from control $P < 0.05$.
† Statistically different from control $P < 0.01$.
‡ Statistically different from control $P < 0.001$.

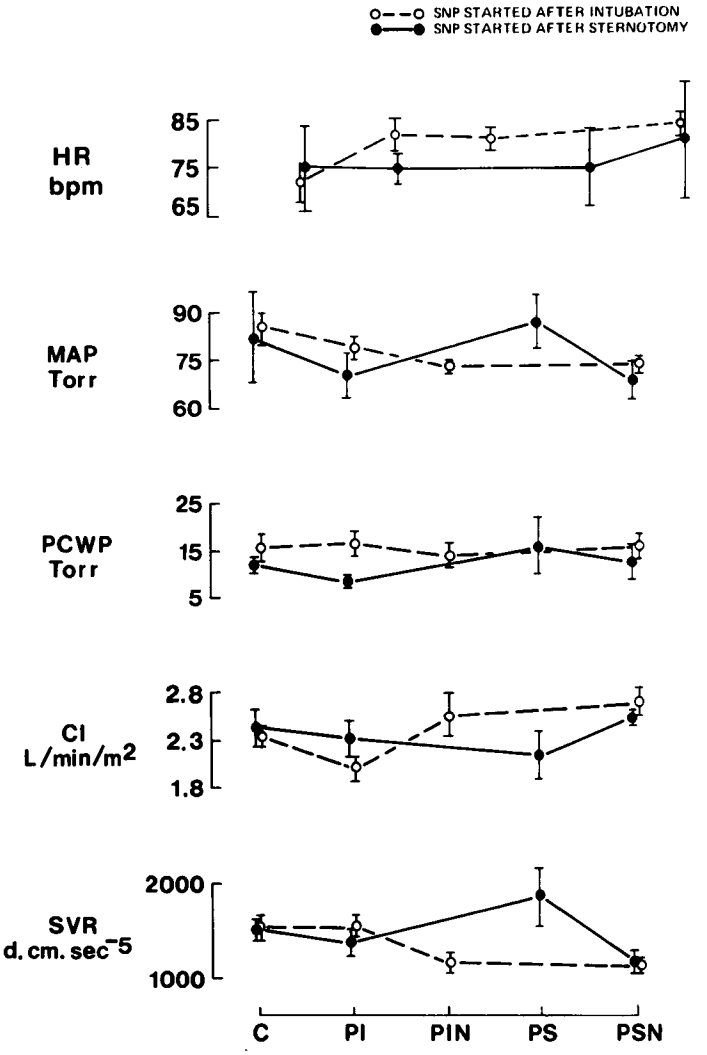

Fig. 2. Group II. Effect of vasodilator therapy (sodium nitroprusside) in 14 patients. *C* = control; *PI* = postintubation; *PIN* = postintubation plus *SNP*; *PS* = poststernotomy; *PSN* = poststernotomy plus *SNP*; *SNP* = sodium nitroprusside.

Discussion

Most previous studies neither differentiate aortic from mitral valve lesions, nor refer to predisposing disease or type of lesion.[1-4] All patients in our series had mitral valve disease and were classified according to predisposing disease, type of lesion, and anesthetic agent used.

This study reconfirmed that patients with mitral valve disease are at added anesthetic risk: Their control values showed a low baseline CI (2.23 ± 0.07 L/min) and high PCWP (23.5 ± 1.4), and mean arterial pressure (MAP) was maintained at the expense of increased sympathetic tone and higher systemic vascular resistance (SVR), (1748 ± 81 dyne · cm · sec^{-5}). Mitral valve lesions, stenosis, or regurgitation and the predisposing disease (rheumatic or ischemic) did not result in a significant difference in measured hemodynamic values. One exception was lower pulmonary vascular resistance (PVR) in patients with mitral insufficiency secondary to ischemic disease. This can be attributed to the shorter duration of the predisposing disease.[5]

The anesthetic management of patients with

Table 4. Effect of vasodilator therapy

	Mitral stenosis (3 patients)		Mitral insufficiency (9 patients)	
	Before SNP	After SNP	Before SNP	After SNP
HR (BPM)	93.0 + 3.5	76.0 ± 0.6	76.2 ± 2.7	81.4 ± 4.2
MAP (torr)	78.7 ± 2.3	73.3 ± 0.3	81.7 ± 4.3	71.2 ± 2.9*
PCWP (torr)	20.3 ± 1.3	16.0 ± 1.0	15.1 ± 2.8	12.6 ± 2.8
CI (L/min/m^2)	1.92 ± 0.20	2.32 ± 0.18	2.10 ± 0.14	2.64 ± 0.21*
SVR (d·sec·cm^{-5})	1661 ± 249	1302 ± 134	1627 ± 146	1130 ± 98†
PVR (d·sec·cm^{-5})	151 ± 43	126 ± 4	184 ± 49	155 ± 44

SNP = sodium nitroprusside.
Significant difference between pre-SNP and post-SNP:
* $P < 0.02$.
† $P < 0.01$.

valve disease has been investigated for more than a decade. In 1969 Lowenstein et al[6] maintained cardiovascular stability in patients with different valve diseases during induction with morphine (1–3 mg/kg). Stoelting et al described induction with (*a*) halothane and nitrous oxide[2] with no significant hemodynamic change, and (*b*) with morphine (1 mg/kg) and reported decreased MAP and SVR and improved CI and stroke volume (SV).[3] They also reported that the use of fentanyl anesthesia (10 μg/kg) for patients with heart valve disease decreased the MAP and SVR and increased CI and SV.[1] Stanley and Webster[4] also used fentanyl induction (50 μg/kg) in 23 patients with unclassified mitral valve disease and reported a significant drop in MAP and HR and increased SV.

Our study did not reveal a significant difference in the response of patients with mitral valve lesions secondary to either coronary or rheumatic disease. Also, there was no significant difference in the hemodynamic response of patients with mitral insufficiency from those with mitral stenosis. Indeed, the initial hemodynamic status of the patient (low CI and elevated SVR) tended to move further in the same direction following induction in all groups, though to a variable extent.

Despite the large number of patients studied, it could not be definitely concluded that induction with one anesthetic agent per se without the use of adjuvant agents was better for a specific pathology or that it would produce the desired hemodynamic status. However, certain trends were significant.

Halothane/N_2O induction frequently caused an undesirable drop in arterial pressure (diastolic below 60 mm Hg), necessitating a decrease in concentration or even discontinuation of the drug. The drop in MAP was more severe in patients with mitral stenosis. It also resulted in a decrease in the CI in all groups. In patients with mitral insufficiency secondary to rheumatic disease, halothane caused a less significant drop in CI than did fentanyl. This can be explained by the vasodilatory effect of halothane which facilitated left ventricular outflow and decreased the regurgitation fraction.[7,8] That was not the case in patients with mitral insufficiency secondary to coronary artery disease; the decrease in CI produced by halothane equaled that of fentanyl.

Fentanyl induction provided a stable MAP in all groups and no significant change in PCWP. The lack of change in MAP ensured adequate perfusion pressure and provided an acceptable pressure baseline for initiation of vasodilator therapy when required. However, the undesirable increase in HR can decrease the filling time in patients with mitral stenosis and increase the $M\bar{v}O_2$, which may be critical in patients with coronary artery disease. However, several studies indicate that the use of pancuronium bromide may have caused such an increase in HR.[9,10] The new muscle relaxants, such as Organon C45 (ethylestrenol), coupled with fentanyl induction, will provide better anesthetic regimens.

The control hemodynamic values in Stage I of this study reconfirmed the previous findings that these patients have elevated SVR and PCWP and decreased CI. These factors changed further in the same direction following induction and sternotomy. We agree with other authors[11–14] that the use of peripheral vasodilators can be a valuable adjuvant in the management of these patients. According to our criteria, sodium nitroprusside was used successfully in 12 of 14 patients, 9 with mitral regurgitation and 3 with mitral stenosis. It increased the CI by 18% to 27%, decreased the SVR by 25% to 37% and PCWP by 17% to 21% as compared to control values. Peripheral vasodilators decrease left ventricular size and regurgitation fraction in acute as well as chronic mitral insufficiency[8,15] in contrast to vasopressors, such as angiotensin, which increase SVR, MAP, and regurgitation fraction, and result in further reduction of the CI.

In moderate ventricular dysfunction, peripheral vasodilators lower filling pressure, increase CI, and decrease the MVO_2.[7] In comparison, inotropics increase CI by enhancing myocardial contractility and increasing MVO_2. However, a combination of inotropics and vasodilators may be indicated in severe ventricular dysfunction.

Sodium nitroprusside improved the CI in 2 of 3 patients with mitral stenosis but was ineffective in the third. However, nitroglycerin, a potent venodilator, would have been a better choice: It decreases venous return, lowers PAP, PVR, and PCWP, and decreases right ventricular afterload. As an arteriodilator, it decreases left ventricular afterload and can increase the CI.

Despite the clear advantages of peripheral vasodilators in the anesthetic management of patients with mitral valve disease, it has to be stressed that they cannot be used routinely or at a specific time during the procedure. Indeed, 2 patients did not require such treatment. One patient with mitral insufficiency (rheumatic) maintained adequate hemodynamic values throughout the prebypass period; the other (with

mitral stenosis) had relatively low preinduction MAP (60 mm Hg) and CI (1.85), became more hypotensive following induction, and did not respond to SNP and fluid administration.

Conclusion

This study did not reveal a specific anesthetic agent or a classic anesthetic management for patients with mitral valve disease. Indeed, it served to stress the importance of individual response and adjustment of hemodynamic factors. However, fentanyl seems to be preferable to halothane as it maintains the MAP and gives a greater sense of security to the anesthesiologist. However, it is important to emphasize that pharmacologic manipulations with agents such as beta blockers or peripheral vasodilators are frequently required to stabilize the hemodynamics.

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