

## CASE REPORTS

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## Early Hemodynamic Changes following Emergency Mitral Valve Replacement for Traumatic Mitral Insufficiency following Balloon Mitral Valvotomy: Report of Six Cases

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PERCUTANEOUS transvenous balloon mitral valvotomy (BMV) is widely performed for relief of symptoms in patients with pure mitral stenosis.<sup>1</sup> Massive mitral insufficiency may occur as a complication of this technique, necessitating urgent surgical intervention.<sup>2</sup> The anatomic lesions<sup>3</sup> and mechanism of regurgitation<sup>2</sup> in such patients have been reported. However, to the best of our knowledge, reports of the hemodynamic data of these patients undergoing emergency mitral valve replacement are not available.

### Case Series

Between September 1995 and October 1996, 841 patients underwent BMV at our institution. Acute severe mitral regurgitation (MR) developed in nine patients (1.01%) and they underwent emergency mitral valve replacement. Hemodynamic data were obtained in six of these patients (1 male, 5 females; age,  $26.8 \pm 6.4$  yr [mean  $\pm$  SD]; weight,  $36.5 \pm 3$  kg). The pre-BMV mean mitral valve area by echocardiography was  $0.77 \pm 0.13$  cm<sup>2</sup>, with a right ventricular systolic pressure of  $69 \pm 24$  mm Hg. Three patients had mild MR (jet area  $< 4$  cm<sup>2</sup>). The pre-BMV left atrial (LA) pressure and mean pulmonary artery pressure (MPAP) measured in the catheterization laboratory were  $27 \pm 8$  and  $49 \pm 14$  mmHg, respectively. The BMV was performed using the Inoue technique.<sup>4</sup>

The post-BMV diagnosis of MR was based on the appearance of large 'v' waves and a rise in mean LA pressure after balloon inflation. Left

atrial pressure increased from  $27 \pm 8$  to  $34 \pm 7$  mmHg; the LA 'v' wave increased from  $36 \pm 10$  to  $57 \pm 7$  mmHg ( $P < 0.001$ ); and the MPAP increased from  $49 \pm 14$  to  $63 \pm 19$  mmHg. A pansystolic murmur, hypotension (systolic blood pressure  $< 70$  mmHg), and pulmonary edema were present in all patients. Hypotension was treated using dopamine; pulmonary edema was treated using furosemide; and two patients also received nitroprusside. Patients arrived in the operating room sitting and receiving oxygen by face mask. The mean duration between BMV and surgery was  $7 \pm 6.6$  h (3-20 h).

In the operating room, in addition to standard monitors, an indwelling femoral catheter (placed at the time of original BMV) was used for arterial pressure measurements and a 14-gauge venous cannula was inserted during local anesthesia for volume infusion. General anesthesia was administered using 20-30 mg morphine, 50-100 mg thiopentone, and 2.5-5 mg diazepam. Muscle relaxation was achieved with pancuronium bromide, and the trachea was intubated. A thermodilution pulmonary artery catheter with a rapid-response thermistor (Bio-sensor International Pte Ltd, Singapore) was inserted. Baseline hemodynamic measurements were obtained 20-30 min after intubation and were repeated 20-30 min after discontinuation of cardiopulmonary bypass (CPB), 4 h after transfer to the intensive care unit, and 30 min after tracheal extubation.

A standard bypass technique with membrane oxygenator and Ringier's lactate prime was used. Myocardial protection was achieved with cold crystalloid cardioplegia and topical cooling. The patients underwent CPB for  $127 \pm 50$  min and aortic cross-clamp for  $86 \pm 30$  min.

Nitroglycerin infusion ( $0.5-1 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ ) was started electively in all patients before discontinuing the CPB and was continued in the postoperative period. In addition, all the patients except one needed infusions of moderately high doses of epinephrine ( $0.1$  to  $0.2 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ ) and dopamine ( $5-15 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ ) for weaning from CPB. The patients were electively ventilated for a variable period until the extubation criteria were met.

The hemodynamic measurements at various time intervals are shown in table 1. The baseline measurements (postinduction) suggested that the patients were maintaining adequate systemic circulation, with a cardiac index of  $3 \pm 0.5 \text{ l} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$ , a mean arterial pressure of  $65 \pm 11$  mmHg, a central venous pressure of  $9 \pm 4$  mmHg, and a heart rate of  $107 \pm 17$  beats/min. However, they had severe pulmonary arterial hypertension (PAH).

Surgery resulted in a marked reduction in pulmonary capillary wedge pressure (PCWP) and a modest decrease in MPAP and right ventricular systolic pressure. However, statistical significance was achieved only on two occasions (one each for MPAP and PCWP). In addition, an improvement in mean arterial pressure, heart rate, and left ventricular stroke work was also observed. However, as expected,

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Table 1. Hemodynamic Measurements Observed at Various Stages

	Pre BMV	Post BMV	Post Induction	Off CPB	4 h ICU	Post Extubation
CI ( $L \cdot min^{-1} \cdot m^2$ )	—	—	$3. \pm 0.5$	$2.5 \pm 0.7$	$2.6 \pm 0.3$	$2.9 \pm 1.2$
MAP (mmHg)	—	—	$65 \pm 11$	$64 \pm 8$	$86 \pm 13^\dagger$	$72 \pm 8$
CVP (mmHg)	—	—	$9 \pm 4$	$12 \pm 6$	$10 \pm 4$	$8 \pm 3$
MPAP (mmHg)	$49 \pm 14$	$63 \pm 19$	$49 \pm 16$	$32 \pm 5^*$	$43 \pm 7$	$38 \pm 7$
LA (mmHg)	$27 \pm 8$	$34 \pm 7$	—	—	—	—
LA 'v' (mmHg)	$36 \pm 10$	$57 \pm 7$	—	—	—	—
PCWP (mmHg)	—	—	$28 \pm 10$	$15 \pm 6$	$20 \pm 7$	$14 \pm 8^*$
HR (beats/min)	—	—	$107 \pm 17$	$114 \pm 13$	$103 \pm 12$	$85 \pm 9^*$
SV (ml/beat)	—	—	$35 \pm 5$	$28 \pm 8^*$	$32 \pm 5$	$40 \pm 16$
LVSW ( $g \cdot ml/beat$ )	—	—	$17 \pm 6$	$19 \pm 9$	$29 \pm 10$	$33 \pm 11^\dagger$
RVSW ( $g \cdot ml/beat$ )	—	—	$19 \pm 9$	$7 \pm 4^\dagger$	$14 \pm 2$	$18 \pm 13$
PVR ( $dyne \cdot s \cdot cm^{-5}$ )	—	—	$480 \pm 314$	$489 \pm 242$	$586 \pm 157$	$590 \pm 391$
SVR ( $dyne \cdot s \cdot cm^{-5}$ )	—	—	$1,224 \pm 362$	$1,338 \pm 264$	$1,890 \pm 501^*$	$1,583 \pm 543$
RVSP (mmHg)	—	—	$82 \pm 37$	$56 \pm 13$	$58 \pm 10$	$58 \pm 22$
RVEDP (mmHg)	—	—	$8 \pm 4$	$12 \pm 6$	$11 \pm 3$	$9 \pm 5$

Post Induction = 30 min after intubation; Off CPB = 30 min after termination of bypass; 4 h ICU = 4 h after the patient is transferred to ICU; Post Extubation = 30 min after extubation; CI = cardiac index; MAP = mean arterial pressure; CVP = central venous pressure; MPAP = mean pulmonary artery pressure; LA = mean left atrial pressure; LA 'v' = left atrial v wave; PCWP = pulmonary capillary wedge pressure; HR = heart rate; SV = stroke volume; LVSW = left ventricular stroke work; RVSW = right ventricular stroke work; PVR = pulmonary vascular resistance; SVR = systemic vascular resistance; RVSP = right ventricular systolic pressure; RVEDP = right ventricular end diastolic pressure.

\*  $P < 0.05$ .

†  $P < 0.01$ .

pulmonary vascular resistance did not decrease in these patients with chronic PAH.

The patients were ventilated for a mean duration of  $21.4 \pm 11.6$  h (range, 10–38 h). The postextubation arterial oxygen tension ( $Pa_{O_2}$ ) was  $100 \pm 51$  mmHg (range, 58–185 mmHg) and arterial carbon dioxide tension ( $Pa_{CO_2}$ ) was  $39 \pm 8$  mmHg.

The surgical findings revealed that all patients had a thickened mitral valve with severe subvalvular fusion. One patient also had evidence of endocarditis. The anterior mitral leaflet was perforated in one patient and torn in the other five. The tear extended up to the annulus in four patients, and in one patient it was paracommissural, extending to the base of the annulus. In addition, all patients had septal perforations less than 5 mm in diameter.

One patient died of intractable cardiac failure within 4 h of surgery, and her hemodynamic data in the intensive care unit could not be measured. This patient had undergone closed mitral valvotomy 8 yr previously and underwent BMV approximately 8 h previously. Her congestive failure was well controlled, and she was transferred to the operating room with dopamine infusion with a systolic blood pressure of 80–90 mmHg. She was separated from CPB with high-inotropic support with a systolic blood pressure of 70–80 mmHg, a PCWP of 16–18 mmHg and a central venous pressure of 20–24 mmHg. Her pressures continued to remain low in the intensive care unit, and she died of cardiac failure.

## Discussion

Percutaneous transvenous BMV, first described by Inoue *et al.*<sup>4</sup> in 1984, is widely performed and is considered to be the first choice of the nonpharmacologic

treatments for isolated mitral stenosis.<sup>2</sup> Reports of large series of more than 600 patients undergoing percutaneous transvenous BMV with satisfactory results are available.<sup>5,6</sup> However, one of the most serious complications of the procedure is the new occurrence of or significant increase in MR, necessitating urgent surgical correction.<sup>2,3,5</sup>

The patients in the current study had mitral stenosis of rheumatic origin and presented at a fairly late stage of the disease. Successful BMV has been reported to lead to significant decreases in LA and pulmonary artery (PA) pressures after BMV in patients with preprocedure mild-to-moderate PAH<sup>7–9</sup> and in those with severe PAH.<sup>5,6,10</sup> The occurrence of acute severe MR in our patients with severe PAH led to hemodynamic compromise. This was controlled considerably by pharmacologic intervention during the period before surgery. The MPAP and PCWP returned to values that were seen at the pre-BMV stage. However, there was little hemodynamic improvement after mitral valve replacement. Pulmonary vascular resistance and right ventricular end-diastolic pressure increased and cardiac index decreased after surgery, although, statistical significance was not achieved. This is in contrast to the hemodynamic improvement that is known to occur after successful BMV<sup>5,10</sup> and elective mitral valve surgery<sup>11</sup> in these patients.



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The early use of Nitroglycerin has been recommended in patients with elevated PA pressures.<sup>12</sup> All our patients received Nitroglycerin infusion. The other potentially promising agents, such as prostaglandin E1 and nitric oxide, may be useful but are not easily available in India.

In conclusion, the hemodynamic parameters that indicate severity of PAH (MPAP, PCWP, right ventricular systolic pressure) showed some improvement, but pulmonary vascular resistance did not decrease after mitral valve replacement in patients with acute MR during BMV. This might constitute a high-risk factor in this group of patients. Perhaps a longer duration of elective ventilation (36–48 h), along with the use of pulmonary vasodilators, may be beneficial in these patients. Monitoring the PA pressures may be helpful in guiding the therapy. Even mild hypercapnia has been shown to be detrimental to right ventricular function in patients of mitral stenosis.<sup>13</sup> Therefore, careful monitoring of  $\text{Pa}_{\text{CO}_2}$  levels during elective ventilation and spontaneous ventilation after extubation also are recommended.

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