The degree of newly emerging mitral regurgitation during off-pump coronary artery bypass is predicted by preoperative left ventricular function

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Abstract
Purpose. During off-pump coronary artery bypass (OPCAB), the displacement of the heart causes mitral regurgitation. We hypothesized that patients with impaired left ventricle (LV) function would be more prone to develop mitral regurgitation, due to further LV end-diastolic pressure elevation and mitral annulus distortion. Therefore, in this study, we examined the relationship between LV function and the severity of mitral regurgitation.

Methods. We studied 41 patients undergoing elective OPCAB. LV function was evaluated by LV ejection fraction (LVEF), serum brain natriuretic peptide (BNP) levels, the Tei index (myocardial performance index) and mitral infl ow propagation velocity (Vp).

Results. Among all of the anastomoses performed mitral regurgitation was most severe during anastomosis of the left circumflex artery (LCX) territory (P < 0.001). Twenty-five patients (61%) had no to mild mitral regurgitation during anastomosis of the LCX territory (M-MR group) and 16 patients (39%) had moderate to severe mitral regurgitation during anastomosis of the LCX territory (S-MR group). There were significant differences between these groups in preoperative serum BNP levels (median, 26 pg·ml⁻¹ interquartile range [IQR, 14 to 75 pg·ml⁻¹] vs median, 173 pg·ml⁻¹ [IQR, 91 to 296 pg·ml⁻¹]; P < 0.001), Tei index values (median, 0.35; [IQR, 0.27 to 0.41] vs median, 0.53 [IQR, 0.47 to 0.57]; P < 0.001), and Vp (median, 63 cm·s⁻¹; [IQR, 57 to 72 cm·s⁻¹] vs median, 47 cm·s⁻¹; [IQR, 40 to 57 cm·c⁻¹]; P = 0.008), while there was no significant difference in LVEF between the patients in the M-MR group and those in the S-MR group.

Conclusion. Preoperative LV dysfunction is a predictor of severe mitral regurgitation during OPCAB. When poor LV function is suggested, it is necessary to be prepared for further hemodynamic deterioration caused by mitral regurgitation.

Key words Left ventricular function · Mitral regurgitation · OPCAB

Introduction
The main goals of the intraoperative management of off-pump coronary artery bypass (OPCAB) are to facilitate coronary anastomosis, to avoid myocardial ischemia, and to maintain sufficient cardiac output for the systemic circulation. However, it is often challenging to stabilize the hemodynamics during the displacement of the heart that is done in order to expose the site of anastomosis during OPCAB. Myocardial ischemia, reduced preload, cardiac dysfunction caused by the compression exerted by a stabilizer, and mitral regurgitation have all been implicated in the hemodynamic derangement that may occur.

Mitrail regurgitation is often associated with ischemic heart disease, with dilatation of the left ventricle (LV) leading to displacement of the papillary muscles, annular dilatation, and tethering of the mitral leaflet [1]. Mitrail regurgitation worsens when the heart is displaced during OPCAB [2–4].

In addition, mitral regurgitation emerges in some patients whose preoperative cardiac evaluation shows no mitral regurgitation. It is accepted that moderated to severe mitral regurgitation reduces forward cardiac output, increase pulmonary artery pressure (PAP), and complicates the control of hemodynamics, particularly in patients with LV dysfunction.

A recent report has described the causes of mitral regurgitation during OPCAB, including mechanical distortion of the LV owing to LV displacement and the use of a stabilizer [4]. We hypothesized that patients with impaired LV function and LV dilatation would be more prone to develop moderate to severe mitral regurgitation because of further LV end-diastolic pressure elevation and mitral annulus distortion during displacement of the heart. Consequently, in this study, we examined the relationship between LV function and the severity of newly emerging mitral regurgitation and its effect upon circulatory status.
Patients and methods

Our study involved 41 consecutive patients without preoperative mitral regurgitation undergoing elective OPCAB at our institution between November 1, 2005, and August 31, 2006. Our study complied with institutional review board (IRB) approval, and informed consent was obtained from all patients. The following patients were excluded from our study: those with significant valvular heart disease, those with atrial fibrillation, those with acute myocardial infarction (less than 1 week old), and those with chronic renal failure on hemodialysis.

Preoperatively, serum brain natriuretic peptide (BNP) levels were measured. The LV ejection fraction (LVEF) was determined by using a transthoracic echocardiogram (TTE). LVEF was calculated by a modified Simpson method.

Triazolam (0.125 or 0.25 mg) was given orally 2 h prior to the induction of anesthesia. In the operating room, a large-bore intravenous catheter and a radial arterial catheter were inserted percutaneously, under local anesthesia. Anesthesia was induced by the administration of midazolam 0.1 mg kg\(^{-1}\) and fentanyl 5–8 µg kg\(^{-1}\), and intubation was facilitated by vecuronium 0.15 mg kg\(^{-1}\) given intravenously. After intubation, the patients were mechanically ventilated to maintain normocapnea. A pulmonary artery catheter (Baxter Healthcare, Irvine, CA, USA) was inserted through the internal jugular vein to measure mixed venous oxygen saturation (Svo\(_2\)), continuous cardiac output, PAP, and central venous pressure (CVP) in all patients. A transesophageal echocardiography (TEE) probe was also inserted to measure a variety of parameters (described in detail below). Anesthesia was maintained with sevoflurane, oxygen, and air. Fentanyl was added as required, to a total dose of about 15 µg kg\(^{-1}\). Patients received up to 2000 ml of crystalloid solution to maintain the cardiac index (CI) at more than 2.01 min\(^{-1}\) m\(^{-2}\) and urinary output at more than 1 ml kg\(^{-1}\) h\(^{-1}\) prior to the commencement of cardiac displacement. When mean arterial pressure (MAP) remained below 60 mmHg after optimization of the circulating blood volume, we administered a bolus injection of phenylephrine in 0.1-mg increments, or a continuous infusion of norepinephrine at the rate of 0.02 to 0.1 µg kg\(^{-1}\) min\(^{-1}\).

Median sternotomy was performed in all patients. Bilateral internal thoracic were harvested. The right gastroepiploic artery, radial arteries, of saphenous veins were also harvested if necessary. After heparinization to achieve an activated clotting time greater than 300 s, three pericardial stitches were placed to facilitate exposure of the posterior or lateral site of the anastomoses. In all patients, surgery was performed using either the Acrobat stabilizer system (Guidant, Santa Clara, CA, USA) or Octopus 2 myocardial stabilization devices (Medtronic, Minneapolis, MN, USA). The anastomoses were performed in sequence, starting with left circumflex artery (LCX) territory (obtuse marginal branches [OM] and posterolateral [PL] branches of the LCX), then the left anterior descending artery (LAD) territory (LAD and diagonal branches [DG]), and lastly the right coronary artery (RCA) territory (posterior descending artery [PDA] and atroventricular-node branches [AV]). During anastomoses of the LCX territory, the heart was tilted and fixed in a vertical and rightward-rotated position with the pericardial stitches and stabilizer devices. Patients were held in the Trendelenburg position with their right side down, at an angle of approximately 20°. For anastomoses of the RCA territory, the heart was fixed in a vertical position while the patients were held in the Trendelenburg position. For the LAD territory, the heart was lifted up with pericardial stitches and stabilized with the stabilizer devices while the patients were held in a horizontal position. During the anastomosis, an intraluminal shunt was inserted. In all patients, the LAD was bypassed with the left internal thoracic artery.

When hemodynamic variables deteriorated despite optimization of the circulating volume, the following hemodynamic treatments were applied. If the CI was greater than 201 min\(^{-1}\) m\(^{-2}\) and MAP was less than 60 mmHg, we administered a bolus injection of phenylephrine (in 0.1 mg increments) or a continuous infusion of norepinephrine, at a rate of 0.02 to 0.1 µg kg\(^{-1}\) min\(^{-1}\). If the CI was less than 2.01 min\(^{-1}\) m\(^{-2}\) and MAP was greater than 60 mmHg, an additional induction of 500 ml of crystalloid was administered. If the CI was less than 2.01 min\(^{-1}\) m\(^{-2}\) and the heart rate was less than 45 bpm, we administered a continuous infusion of dopamine at a rate of 3 to 10 µg kg\(^{-1}\) min\(^{-1}\). If hypertension (systolic blood pressure greater than 140 mmHg) occurred despite adequate depth of anesthesia, we infused nitroglycerin at a rate of 0.1 to 1 µg kg\(^{-1}\) min\(^{-1}\). When hypertension was associated with an increase heart rate of greater than 80 bpm, we administered a bolus injection of esmolol (0.5 mg kg\(^{-1}\)).

Intraoperative TEE data were acquired using a Philips Sonos 7500 (Philips, Best, The Netherlands) ultrasound system equipped with a multiplane transesophageal probe. The severity of mitral regurgitation was evaluated by analysis of the color Doppler mitral regurgitant jet area (MRJA; cm\(^2\)). The grade of mitral regurgitation, determined by using a previously published classification scheme was noted as: trivial to mild mitral regurgitation (MRJA < 3 cm\(^2\)), on moderate to severe mitral regurgitation (MRJA > 3 cm\(^2\)) [5].

Mitral inflow propagation velocity (Vp) was obtained by TEE from a midesophageal four-chamber view by measuring the slope of the first aliasing velocity during