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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 inflow 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}] versus 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] versus 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}] versus 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

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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.

Mitral 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]. Mitral 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 $mg \cdot kg^{-1}$ and fentanyl 5–8 $\mu g \cdot kg^{-1}$, and intubation was facilitated by vecuronium 0.15 $mg \cdot kg^{-1}$ 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 $\mu g \cdot kg^{-1}$. Patients received up to 2000 ml of crystalloid solution to maintain the cardiac index (CI) at more than 2.0 $l \cdot min^{-1} \cdot m^{-2}$ and urinary output at more than 1 $ml \cdot kg^{-1} \cdot h^{-1}$ 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 $\mu g \cdot kg^{-1} \cdot min^{-1}$.

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 atrioventricular-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 Trendlenburg 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 2 $l \cdot min^{-1} \cdot m^{-2}$ and MAP was less than 60 mmHg, we administered a bolus injection of phenylephrine (in 0.1 mg increments) or a continuous infusion of norephinephrine, at a rate of 0.02 to 0.1 $\mu g \cdot kg^{-1} \cdot min^{-1}$. If the CI was less than 2.0 $l \cdot min^{-1} \cdot m^{-2}$ and MAP was greater than 60 mmHg, an additional indusion of 500 ml of crystalloid was administered. If the CI was less than 2.0 $l \cdot min^{-1} \cdot m^{-2}$ and the heart rate was less than 45 bpm, we administered a continuous infusion of dopamine at a rate of 3 to 10 $\mu g \cdot kg^{-1} \cdot min^{-1}$. 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 $\mu g \cdot kg^{-1} \cdot min^{-1}$. When hypertension was associated with an increase heart rate of greater than 80 bpm, we administered a bolus injection of esmolol (0.5 $mg \cdot kg^{-1}$).

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

early filling from the mitral leaflet tips to a position 4 cm into the LV. The Nyquist limit was set to 70% of the peak early mitral inflow velocity.

The myocardial performance index (Tei index) was measured by TEE, using the following published method [6]. The mitral inflow velocity curve was recorded from the midesophageal four-chamber view with a pulsed-wave Doppler sample volume positioned at the tips of the mitral leaflets during diastole. The LV outflow velocity curve was recorded from the transgastric long-axis view. The interval (a) from cessation to the onset of mitral inflow is equal to the sum of the isovolumetric contraction time (IVCT; ms), isovolumetric relaxation time (IVRT; ms), and ejection time (ET; ms). ET (b) was measured from the left ventricular outflow velocity curve. The Tei index was calculated as follows:

Tei index = IVCT + IVRT / ET = a − b / b

All Tee examinations were performed and concurrently interpreted by cardiac anesthesiologists experienced in perioperative echocardiography, with The Japanese Board of Perioperative Transesophageal Echocardiography (JB-POT) certification. The anesthesiologists were blinded to the patients' clinical data.

Baseline Tee and hemodynamic data were measured after the optimization of circulating blood volume and before median sternotomy. During each anastomosis, TEE data and hemodynamic indices were measured, between the time of application of the coronary stabilizer and coronary occlusions after hemodynamics were stabilized. All TEE and hemodynamic data were obtained during apnea.

Values for continuous variables are expressed as means (± SD) or medians (interquartile ranges [IQRs]) depending on the distribution of the variables, and dichotomous variables are presented as numbers and percentages. One-way repeated-measures analysis of variance (ANOVA) was performed for comparisons of hemodynamic data. When significant differences ($P < 0.05$) were found, post-hoc comparisons were made using Bonferroni corrections. Friedman's test was performed for intraposition comparison of the MRJA. Two-way repeated measures ANOVA was performed for intragroup comparison of mean pulmonary artery pressure. Receiver operating characteristic (ROC) curves were generated to determine the accuracy of the indices to predict moderate or sever mitral regurgitation. Optimal cutoff values of each index were defined with the highest sum of sensitivity and specificity. The area under the ROC curve (AUC) was used as an index of global performance, with an AUC of 0.5 indicating no discrimination ability. AUC values were compared using a previously reported method [7].

Because the distribution of the BNP level was positively skewed, a natural log transformation was used. A P value of less than 0.05 was considered statistically significant. The statistical analysis was performed using SPSS for Windows version14 (SPSS, Chicago, IL, USA).

Results

Forty-one patients were evaluated for analysis (Table 1). All patients underwent left internal thoracic artery (LITA) to LAD anastomoses. Anastomoses of the LCX and RCA territories were performed in 41 and 24 patients, respectively. Hemodynamic variables during anastomoses showed no significant difference in CI values but there were significant differences in of Sv_{O_2}, MAP, and MRJA between position (Table 2). During anastomoses of all territories, mean pulmonary arterial pressure (MPAP) increase significantly compared to the baseline. MRJA during anastomosis of the LCX territory (MRJA-LCX) was significantly greater than that during anastomosis of the other two territories ($P < 0.001$). Therefore, we examined the relationships between the degree of mitral regurgitation during

Table 1. Patients' characteristics

Variable	
No. of patients	41
Age (years)	67 ± 9
Weight (kg)	62 ± 11
Male	31 (76)
Lesion	2.8 ± 0.4
Hypertension	27 (66)
Diabetes	24 (59)
β-Antagonist	11 (27)
Ca^{2+} antagonist	18 (44)
ACE inhibitor	7 (17)
Number of grafts	
LAD territory ($n = 41$)	
LAD	41
DG	26
LCX territory ($n = 41$)	
OM	28
PL	25
RCA territory ($n = 24$)	
PD	30
AV	11
RVB	7
Total	168
History of MI	19 (46)
LVEF	0.58 ± 0.14
BNP pg·ml^{-1}, median (range)	75.8 (7.3 – 1045)

Data values are shown as means ± SD numbers (percentages) unless otherwise indicated

ACE inhibitor, angiotensin-converting enzyme inhibitor; LAD, left anterior descendeing artery; DG, diagonal artery; OM, obtuse marginal artery; PL, posterolateral artery; PD, posterior descending artery; AV, AV-node branch; RVB, right ventricular branch; MI, myocardial infarction; LVEF, left ventricular ejection fraction; BNP, serum brain natriuretic peptide level

Table 2. Hemodynamic changes during anastomosis

	Baseline	LAD ($n = 41$)	LCX ($n = 39$)	RCA ($n = 24$)	Post
HR (beats·min^{-1})	60 ± 11	68 ± 11*	67 ± 10*	68 ± 10*	75 ± 9*
MAP (mmHg)	72 ± 11	70 ± 10	67 ± 7*	69 ± 10	70 ± 11
MPAP (mmHg)	16 ± 4	22 ± 6*	21 ± 5*	21 ± 5*	24 ± 6
Sv_{O_2} (%)	76 ± 7	73 ± 7*	66 ± 9*,**	69 ± 8*	75 ± 6
CI (l·min^{-1}·mm^2)	2.2 ± 0.4	2.4 ± 0.6	2.2 ± 0.5	2.5 ± 0.5	2.6 ± 0.6*
MRJA (cm^2), median (range)		0.5 (0 – 4.01)	2.03 (0 – 5.8)***	0.81 (0 – 4.41)	

* $P < 0.05$ vs baseline; ** $P < 0.05$, intraposition comparison; *** $P < 0.01$, intraposition comparison
Values are shown as means ± SD unless otherwise indicated
HR, heart rate; MAP, mean arterial pressure; MPAP, mean pulmonary arterial pressure; Sv_{O_2}, mixed venous oxygen saturation; MRJA, mitral regurgitation jet area; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery

Table 3. ROC analysis for indices of LV function

	AUC	95% CI	Optimal cutoff value	Sensitivity	Specificity
BNP	0.92*	0.84–1.00	73.9	0.94	0.75
Tei index	0.92*	0.82–1.00	0.47	0.8	0.95
Vp	0.79	0.62–0.96	55.5	0.84	0.75
EF	0.63	NS			

* $P < 0.05$ vs Vp
ROC, Receiver operator characteristic; AUC, area under the curve; CI, confidence interval; BNP, brain natriuretic peptide; Vp, propagation velocity; EF, ejection fraction; NS, not significant

anastomosis of the LCX territory (MR-LCX) and the indices of LV function.

Twenty-five patients (61%) had no to mild mitral regurgitation (M-MR group) and 16 patients (39%) had moderate to severe mitral regurgitation (S-MR group). The indices of LV function in the two groups are shown in Fig 1. There were significant differences in preoperative serum BNP levels (median, 26 pg·ml^{-1} [IQR, 14 to 75 pg·ml^{-1}] versus median, 173 pg·ml^{-1} [IQR, 91 to 296 pg·ml^{-1}]; $P < 0.001$; (Fig. 1A), Tei index values (median, 0.35 [IQR, 0.27 to 0.41] versus median, 0.53 [IQR, 0.47 to 0.57]; $P < 0.001$; (Fig. 1B), and Vp (median, 63 cm·s^{-1} [IQR, 57 to 72 cm·s^{-1}] versus median, 47 cm·s^{-1} [IQR, 40 to 57 cm·s^{-1}]; $P = 0.008$; (Fig. 1C) between the patients in the M-MR group and those in the S-MR group. There was no significant difference in LVEF between the two groups, but there was a tendency for the patients in the S-MR group to have a lower LVEF (M-MR group; median, 0.65 [IQR, 0.52 to 0.73] versus: S-MR group: median, 0.50 [IQR, 0.42 to 0.65]; $P = 0.08$; (Fig. 1D).

ROC analysis was applied to evaluate the predictive power of each index of LV function (Table 3). ROC analysis showed that the Tei index, serum BNP level, and Vp were significant predictors of moderate or sever mitral regurgitation. There were significant differences between the AUCs of the serum BNP level and Vp ($P < 0.05$), and between the AUCs of the Tei index and Vp ($P < 0.05$).

The correlation between the Tei index and serum BNP level was examined. The Tei index and the log transformed serum BNP level were significantly correlated (Fig. 2; $R^2 = 0.47$; $P < 0.001$).

Figure 3 show comparisons of MPAP in the M-MR and S-MR patient groups at baseline and at positioning for anasomosis of the LCX territory. There was a significant difference in MPAP between the two groups during displacement of the heart for anastomnosis of the LCX territory ($P = 0.044$).

Discussion

Our results support the hypothesis that the severity of mitral regurgitation during anastomosis of the LCX territory is predicted by the degree of LV dysfunction. Moreover, in the patients with moderate to severe mitral regurgitation, MPAP was found to be significantly greater than that in the patients with no to mild mitral regurgitation during anastomosis of the LCX territory.

LV function can be divided into systolic and diastolic functions. It is not clear whether systolic or diastolic dysfunction is more closely related to the appearance and the degree of mitral regurgitation that occurs during anastomosis of the LCX territory. We evaluated LV function by four indices (LVEF, Vp, the Tei index, and serum BNP level). We evaluated global cardiac pump function by LVEF, as a load-dependent index; LV dia-

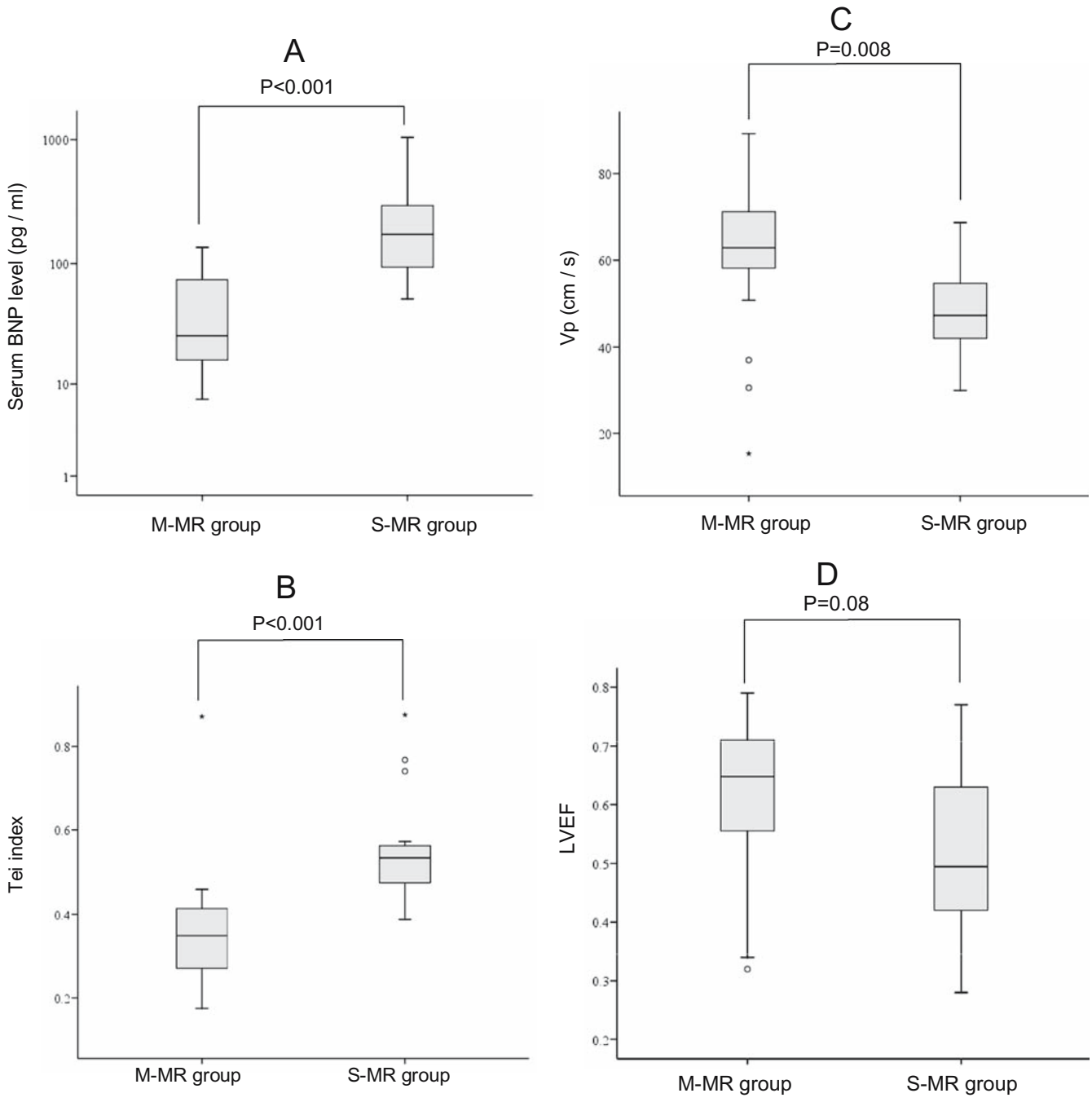

Fig. 1. A–D. Comparison of hemodynamic indices in patients with no to mild mitral regurgitation (*M-MR group*) and those with moderate to severe mitral regurgitation (*S-MR group*). **A** Serum brain natriuretic peptide (*BNP*) level; **B** Tei index; **C** mitral inflow propagation velocity (*Vp*); **D** left ventricular ejection fraction (*LVEF*)

stolic function by Vp; and combined systolic and diastolic function by the Tei index [8,9]. BNP is released from the heart in response to wall stress [10]. Although the quantitative relationship between serum BNP level and LV systolic and diastolic function remains controversial [11,12], increased serum BNP level is considered to be a useful marker for identifying patients with heart failure caused by either systolic or diastolic dysfunction. The severity of mitral regurgitation was well correlated with the increased serum BNP level and a greater Tei index. This suggests that it is crucial to evaluate both systolic and diastolic function and to identify patients with heart failure in order to predict the severity of mitral regurgitation during OPCAB.

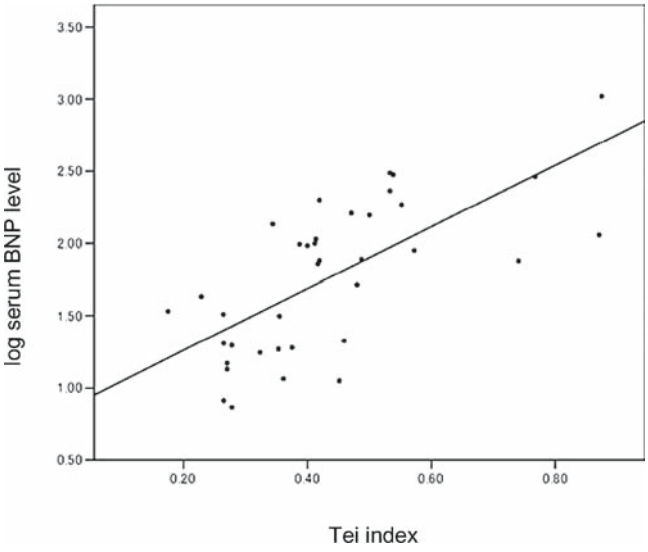

Fig. 2. Correlation between the Tei index and the log transformed serum brain natriuretic peptide level (*log serum BNP level*). Y = –0.833x + 2.135; $R^2 = 0.471$; $P < 0.01$

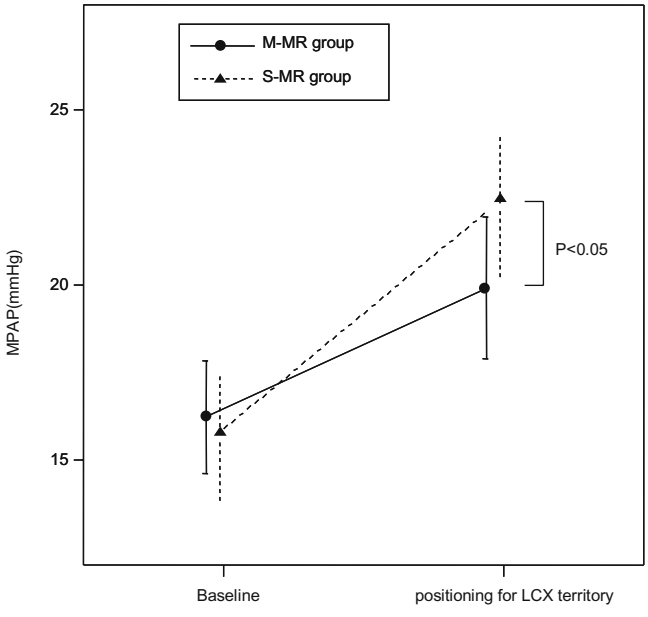

Fig. 3. Comparison of mean pulmonary artery pressure (*MPAP*) in the patient group with no to mild mitral regurgitation (*M-MP group*) and the group with moderate to severe mitral regurgitation (*S-MR group*), at baseline and at positioning for left circumflex artery (*LCX*) territory anastomosis. *$P < 0.05$, intragroup comparison by two-way repeated-measures analysis of variance (ANOVA)

We hypothesized that increased LV end-diastolic pressure may lead to newly emerging mitral regurgitation by distortion of the mitral annuli. The LV end-diastolic dimension (LVDd) should correlate with the LV end-diastolic pressure, although their relationship is nonlinear. We found no significant difference in LVDd between the S-MR and M-MR groups (data not shown). This can be explained by several factors, including the volume status of the patient, the compliance of the LV being changed by regional myocardial scarring due to an old myocardial infarction, and generalized myocardial hypertrophy. Because these factors were not controlled in this study, the data could not be interpreted in relation the end-diastolic pressure. Therefore, we note that our data showed no clear evidence that the severe mitral regurgitation was attributable to elevated end-diastolic pressure.

George et al. [4] reported that, during displacement of the heart for anastomosis, the mitral annulus became distorted, causing mitral regurgitation, leading to the elevation of left atrial pressure. These authors showed that the greatest distortion of the mitral annulus occurred during positioning for anastomosis of the LCX territory. This finding is compatible with our results demonstrating that newly emerging mitral regurgitation during anastomosis was most severe during the displacement of the heart for anastomosis of the LCX territory.

This report by George et al. [4] and our results lead to the following conjecture about the mechanism that causes more severe mitral regurgitation in patients with LV dysfunction. If systolic function is impaired and the heart is dilated due to an old myocardial infarction, the heart needs to be displaced more extensively. In patients with a hypertrophic heart with diastolic dysfunction, the heart also needs to be extensively displaced by exerting a strong force. In addition, a hypertrophic heart is more susceptible to ischemia caused by the reduced coronary blood flow resulting from displacement of the heart, and ischemia could exacerbate mitral regurgitation. Therefore, we speculate that this more extensive displacement of the heart caused greater distortion of the mitral annulus associated with more severe mitral regurgitation, and it is possible that ischemia exacerbated the mitral regurgitation.

Factors causing deterioration in hemodynamics during OPCAB include myocardial ischemia, cardiac chamber compression, reduced ventricular filling, and reduced diastolic function [2,13]. In patients with impaired LV function, it is difficult to optimize the hemodynamic status because multiple factors tend to be involved in the deterioration of hemodynamics. If mitral regurgitation emerges newly, it causes further hemodynamic derangement because mitral regurgitation reduces forward flow and elevates pulmonary artery pressure. Moderate or severe mitral regurgitation can lead to significant hemodynamic consequences, especially when the cardiac output is reduced by the displacement of the heart for anastomosis. Therefore, it is crucial to evaluate LV function and hemodynamics in a

highly precise manner during OPCAB. Consequently, intraoperative TEE is useful for both diagnosis and for the optimization of hemodynamics.

At our institution, revascularization most often begins with the LCX territory, according to the cardiac surgeon's preference. Consequently, there is a possibility that the most prominent hemodynamic deterioration and severe mitral regurgitation occurring during anastomosis of the LCX territory was partially attributable to ischemia.

In patients undergoing OPCAB, LV systolic function is often impaired due to myocardial infarction. In addition, LV diastolic function is also frequently impaired by aging, myocyte hypertrophy, and post-infarct scarring in patients with ischemic heart disease [14]. Therefore, we need to evaluate LV systolic and diastolic function separately. Among the methods used for echocardiographic LV diastolic measurements, the ratio of the early filling peak velocity (E) and the atrial peak velocity (A) (E/A ratio) of transmitral inflow is a popular index of LV diastolic function. However there is a problem of pseudonormalization and the dependence of this ratio on preload. The mitral inflow Vp is an index of the quantitative analysis of LV diastolic function. Vp is simple to evaluate and is a reliable method [15]. The advantage of Vp is that it is independent of both heart rate and preload [16,17]. Both the Tei index and serum BNP level are easy to measure, and they are useful for evaluating combined LV systolic and diastolic function. Our present data showed a significant correlation between serum BNP level and the Tei index, which is consistent with previous reports [18,19]. Our results also confirm that we need to evaluate both systolic and diastolic function preoperatively to predict hemodynamic changes during OPCAB. Our results show that if serum BNP level and TTE data are not measured preoperatively, then intraoperative TEE can provide good indices to assess LV systolic and diastolic function.

When mitral regurgitation is detected and hemodynamic optimization is necessary, positive inotropic drugs and vasodilators should be considered, according to the mechanisms of mitral regurgitation; if it is mainly due to systolic dysfunction, then, theoretically, it may be better treated with positive inotropes such as catecholamines. If it is mainly due to diastolic dysfunction, then, theoretically, it may be better treated with agents that cause lusitropic effects, such as phosphodiesterase (PDE) III inhibitors. Omae et al. [20] reported that milrinone was effective in patients with coexisting mitral regurgitation. If both systolic and diastolic dysfunction are related to mitral regurgitation, then both catecholamines and PDE III inhibitors may be administered. Preload reduction by either nitrates or PDE III inhibitors may also be useful to decrease mitral orifice dilatation.

Study limitations

The mitral regurgitant jet area is a semiquantitative index of the severity of mitral regurgitation. As the first limitation of the study, although quantitative methods such as the regurgitant stroke volume or the regurgitant orifice area are ideal, they are both technically demanding during surgery [21]. A second limitation is that LVEF was measured by TTE between 1 week and 3 weeks before the day of operation. Serum BNP level was measured between 2 days and 4 days before the day of operation. TEE data were obtained intraoperatively from patients under gerenal anesthesia. It is possible that these differences in measurement time may have contributed to the better fit of the BNP level and TEE data than the LVEF data. Additionally, some of the patients had regional wall-motion abnormalities, which limited the accuracy of LVEF measured by echocardiography, even though it was measured by a modified Simpson method.

In conclusion, preoperative LV dysfunction is a strong predictor of moderate to severe mitral regurgitation during OPCAB. Both systolic and diastolic function may be useful for predicting the occurrence of mitral regurgitation and for selecting the proper agents for treatment. Intraoperative TEE is very useful for this purpose. When poor LV function is indicated by preoperative examination and intraoperative TEE, it is necessary to be prepared for further hemodynamic deterioration caused by mitral regurgitation.

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