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# Evaluation of Acute Ischemic Mitral Regurgitation Following Cardiopulmonary Bypass Assessed by Biplane Transesophageal Echocardiography

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## ABSTRACT

The aim of this study was to evaluate pathogenesis and outcome of acute ischemic mitral regurgitation (MR) in patients undergoing coronary artery bypass grafting (CABG) using biplane transesophageal echocardiography (TEE).

Biplane TEE was continuously monitored in a total of 96 patients who were scheduled for elective CABG surgery. Of 96 patients, 10 with no MR at stages 1 (after anesthetic induction but before skin incision) and 2 (after cardiopulmonary bypass [CPB] and decannulation) were excluded. In the remaining 86 patients with MR between stages 1 and 2, 45 (group A) had an increase in MR, and 41 (group B) had a decrease in MR. An increase in MR at stage 2 in group A was associated with a significant increase in annular diameter ($p < 0.05$), and pulmonary capillary wedge pressure ($p < 0.01$) compared with stage 1. A significant decrease in the left ventricular end-diastolic area ($p < 0.01$), end-systolic area ($p < 0.05$) and the mean wall motion abnormality score (WMA score) ($p < 0.001$) was observed at stage 2 compared with stage 1 in group B. In 16 of 17 patients (94%) with an increased WMA score in group A, a regional wall motion abnormality (RWMA) was detected in the right coronary artery (RCA) and/or left circumflex coronary artery (LCX) areas. In 7 patients in group A, MR increased continuously until stage 3 (after sternal closure) despite treatment. In 2 of these 7 patients, pulmonary venous systolic flow (PVSF) decreased during stage 2 and persisted to stage 3. The post operative course of these 2 patients was complicated with atrial fibrillation (AF).

The increase in annular diameter and worsening in RWMA in RCA and/or LCX areas are associated with acute ischemic MR following CPB. The majority of acute ischemic MR cases were resolved by pharmacological intervention. Post operative AF was noted in 2 patients with acute ischemic MR associated with persistently decreased PVSF following CPB despite treatment.

***Key words***: *Acute ischemic mitral regurgitation, Coronary artery bypass grafting, Biplane transesophageal echocardiography*

Transesophageal echocardiography (TEE) provides high-resolution images of the mitral apparatus and regurgitant flow[18]. Trivial to mild degrees of mitral regurgitation (MR) are often seen in patients during coronary artery bypass grafting (CABG). Previous clinical studies have focused primarily on chronic ischemic mitral insufficiency and have demonstrated papillary muscle dysfunction[2], incomplete mitral leaflet closure[5], mitral annular dilatation[10], and accompanying ventricular dyskinesia or dilatation[5] as pathogenic mechanisms. Little information is available regarding the echocardiographic changes associated with acute ischemic MR, defined as MR which developed following cardiopulmonary bypass (CPB). Thus, this study was designed to evaluate the morphological factors and outcome of acute ischemic MR in CABG patients by biplane TEE.

## MATERIALS AND METHODS

### Patient population

Biplane TEE was performed as a routine monitor in 124 consecutive patients undergoing CABG. Twenty eight patients, i.e., 4 with atrial fibrillation (AF), 12 with mitral valve surgery, 5 with intra-aortic balloon pump (IABP) preoperatively, and 7 with inadequate resolution of TEE

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views were excluded from this study.

The pathomorphology of the leaflets or chordae and the etiology of the valve disease were easily assessed with TEE[18] prior to CPB and thus only patients who had normal leaflets and chordae were included in this study.

### Anesthesia and surgery

Anesthesia was induced and maintained intravenously with Sufentanil (20 to 40 mg/kg) and Diazepam (0.3 to 0.5 mg/kg) supplemented with muscle relaxants (Vecuronium and/or Pancuronium). CPB was maintained with hollow-fiber membrane oxygenators, i.e., Maxima$^R$ – (Medtronic, Sunnheim, California, USA), Plexus$^R$ – (Shiley, Arvin, California, USA), $SMO_2{}^R$ – (Sarns, Michigan, USA), HF-5700$^R$ – (Bard, Perksburg, Massachusetts, USA). Either antegrade delivery or combined antegrade-retrograde delivery of cardioplegia was used for myocardial protection. The internal thoracic artery and/or saphenous veins were routinely used as conduits for CABG. During the operation, the surgeons and anesthesiologists were aware of the real-time transesophageal echocardiographic findings such as ischemic change, ventricular size and other pathoanatomical changes, and specific therapeutic treatment was performed accordingly.

### Transesophageal echocardiographic studies

After anesthesic induction and intubation, a 5-MHz phased-array biplane transesophageal echocardiographic probe (PEF-510B, Toshiba, Japan) was inserted into the esophagus, and the probe was connected to an ultrasonograph (SSH-140A, Toshiba, Japan). All echocardiograms were stored on S-VHS video tapes.

The MR jet area was measured with a 45° angle sector of imaging, at 15 frame/sec scanning rate, and a 4.0K-MHz pulse-repetition frequency. The gain was adjusted step-by-step to exclude the low velocity monochromatic swirling from outside of the area of mosaic turbulence, which is created by MR. Color Doppler flow imaging settings were maintained constantly throughout the observation period. During each examination, the probe was moved up and down to obtain the entire maximal regurgitant jet area. Also color flow mapping of the entire left atrium was performed. MR jet area was evaluated by off-line analysis using a cine-freeze frame image. Multiple tomographic cuts of each four- and two-chamber view were obtained by using internal calculation software (Fig. 1), and the greater of these two maximal regurgitant jet areas was chosen for data analysis[28]. MR jet direction was also assessed by both four-and two-chamber views. The left upper pulmonary venous flow was recorded using pulsed-wave Doppler for off-line analysis[13].

An accurate image of mitral annular diameter was obtained from the junction of the anterior mitral leaflet and aortic root to the junction of the posterior mitral leaflet and atrioventricular

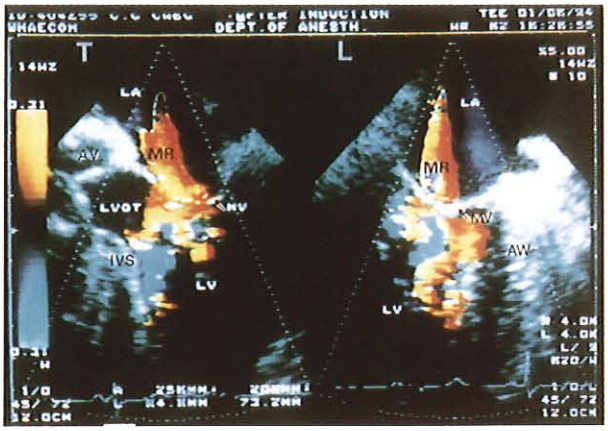


**Fig. 1.** Areas of mitral regurgitation were measured in a transverse scan shown on the left side and a longitudinal scan shown on the right side. The greater of these two maximal regurgitant jet areas was used for the analysis.
T, Transverse scan; L, longitudinal scan; MR, mitral regurgitation; LA, left atrium; AV, aortic valve; LVOT, left ventricular outflow tract; IVS, interventricular septum; LV, left ventricle; MV, mitral valve; AW, anterior wall of left ventricle.

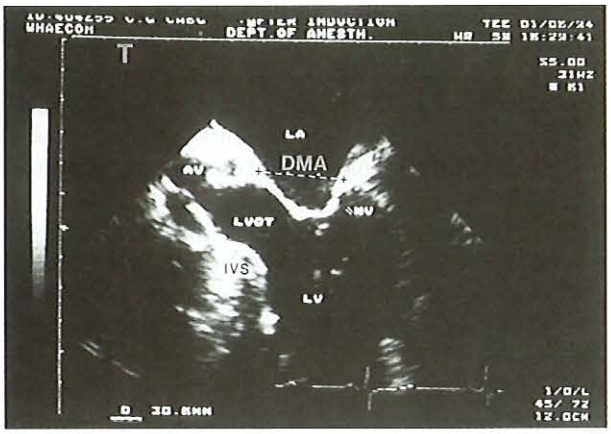


**Fig. 2.** The diameter of the mitral annulus was measured by off-line analysis using a cine-freeze frame image.
It was defined as the end-systolic distance from the junction of the anterior mitral leaflet and aortic root to the junction of the posterior mitral leaflet and atrioventricular groove in the four-chamber view.
T, Transverse scan; DMA, diameter of the mitral annulus; LA, left atrium; AV, aortic valve; LVOT, left ventricular outflow tract; LV, left ventricle; IVS, interventricular septum; MV, mitral valve.

groove in the transverse scan at the end-systole[16] by off-line analysis using a cine-freeze frame image (Fig. 2).

To measure the left ventricular end-diastolic area (EDA) and systolic-area (ESA), a cross-sectional view of the left ventricle (LV) at mid-papillary muscle level was obtained using the ECG synchronization mode. The percentage fractional area change (%FAC) was calculated with the following equation.

$$\%FAC = [(EDA - ESA)/EDA] \times 100$$

Measurements were made at three consecutive regular heart beats and the average was used as data at each stage.

## Analysis of regional wall motion

Assessment of regional wall motion abnormality (RWMA) was performed according to the nomenclature recommended by the American Society of Echocardiography[8]. The short-axis view (SAV) at mid-papillary muscle level was divided into 8 segments, and the long-axis view (LAV) into 3 portions (apical, mid, basal) and divided into 2 segments (anterior, inferior) (Fig. 3). The mid-portion of LAV (2 segments) was excluded from the analysis because it overlaps with the SAV. A LAV was always orthogonal to the corresponding transverse scan (T-scan) and thus an accurate SAV was confirmed by switching to a LAV for probe position. The wall motion of each of the twelve segments was graded in a semiquantitative fashion. Additionally, the estimate of endocardial excursion and myocardial thickening was made visually in slow motion video recordings. The scoring system was derived as follows: 0 = normal (non-reduced radial shortening [RS] and wall thickening [WT]), 1 = mild hypokinesis (slightly reduced RS and WT), 2 = severe hypokinesis (markedly reduced RS and WT), 3 = akinesis (absent RS and WT), and 4 = dyskinesia (outward systolic radial lengthening and wall thinning)[11]. The wall motion abnormality score (WMA score) was defined as the sum of the grading of all 12 LV segments. The wall motion in each segment soon after tracheal intubation was used as the baseline value for each segment. When wall motion of any segment changed by two or more grades from a baseline for at least 1 minute, it was judged to be a newly developed RWMA, and by one grade, it was judged to be a worsened RWMA[11,20].

The off-line qualitative analysis of RWMA was performed by two independent experienced echocardiographers (TN and KF) viewing the videotaped data after surgery. All TEE data were re-examined six months later by one observer (TN).

All echocardiographic data were recorded at the following two stages: Stage 1: after anesthetic induction but before skin incision; Stage 2: after CPB and decannulation (post protamine administration), and under stable hemodynamic conditions. The stage 1 data were defined as baselines. Moreover, degrees of MR, MR jet direction and the pattern of pulmonary venous systolic flow (PVSF) were followed up until sternal closure (stage 3).

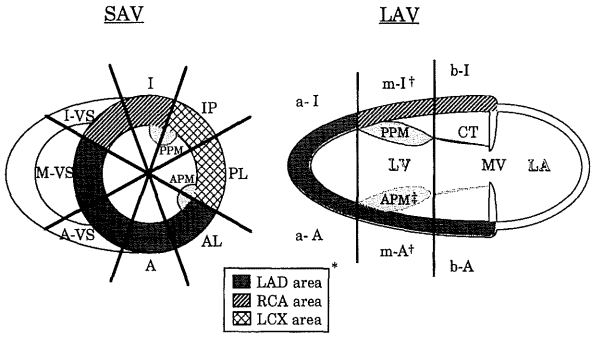


**Fig. 3.** Schematic diagrams of transesophageal short-axis view (SAV) at mid-papillary muscle level and the long-axis view (LAV) were evaluated semiquantitatively and assigned a wall motion abnormality score.
A, Anterior left ventricle; AL, anterolateral left ventricle; PL, posterolateral left ventricle; IP, inferoposterior left ventricle; I, inferior left ventricle; A-VS, anterior ventricular septum; M-VS, middle ventricular septum; I-VS, inferior ventricular septum; a-A, apical anterior left ventricle; a-I, apical inferior left ventricle; m-A, mid anterior left ventricle; m-I, mid inferior left ventricle; b-A, basal anterior left ventricle; b-I, basal inferior left ventricle; LV, left ventricle; LA, left atrium; MV, mitral valve; CT, chordae tendineae; APM, anterior papillary muscle; PPM, posterior papillary muscle.
*LAD area (M-VS, A-VS, A, AL in SAV and b-A, m-A, a-A, a-I in LAV) is perfused by left anterior descending coronary artery. LCX area (PL, IP in SAV) is perfused by left circumflex coronary artery. RCA area (I, I-VS in SAV and m-I, b-I in LAV) is perfused by right coronary artery.
†The mid-portion of LAV (m-A, m-I) was excluded from the analysis.
‡APM is not always visualized in LAV.

## Hemodynamics

Hemodynamic measurements, including heart rate (HR), systolic arterial pressure (SAP), mean arterial pressure (MAP), pulmonary capillary wedge pressure (PCWP) and cardiac output (CO) with thermodilution technique, were obtained simultaneously at each transesophageal echocardiography study point.

## Other parameters

The use of pharmacological support (inotropic and/or vasodilator agents) and IABP was evaluated.

### Statistics

Data of each group were presented as the mean ± standard deviation. The significance of inter-group comparison was tested by one-way ANOVA with a multiple comparison test. The significance of inter-stage comparison was performed with a two-tailed Student's paired t test or Wilcoxon signed-rank test. Frequency data were assessed using Chi-Square analysis and Yates continuity correlation. Significance was assumed at $p < 0.05$, but described in the highest value.

The accuracy of qualitative analysis of RWMA was defined as the percentage of segments with identical scores, and its concordance as the percentage of segments within one grade of variation in the score[11].

**Table 1.** Patient Characteristics

| | Group A | Group B |
|---|---|---|
| No. of patients | 45 | 41 |
| Age (years)† | 63.6 ± 11.4 | 66.5 ± 12.4 |
| Sex (male/female) | 28/17 | 33/8 |
| NYHA classification† | 3.0 ± 0.7 | 3.3 ± 0.8 |
| No. of patients with previous disease | | |
| Myocardial infarction | 26 | 33* |
| Location | | |
| LAD area | 10 | 12 |
| LCX area | 2 | 1 |
| RCA area | 11 | 17 |
| LAD+RCA areas | 3 | 3 |
| Unstable angina pectoris | 24 | 27 |
| Hypertension | 28 | 23 |
| Diabetes mellitus | 16 | 12 |
| CABG | 2 | 1 |
| No. of diseased arteries †, ‡ | 2.49 ± 0.59 | 2.66 ± 0.62 |
| LMT | 7 | 9 |
| LAD | 43 | 40 |
| LCX | 37 | 32 |
| RCA | 34 | 32 |
| No. of bypass grafts† | 3.22 ± 0.95 | 3.02 ± 0.88 |

Abbreviations: NYHA, New York Heart Association; LAD, left anterior descending coronary artery; LCX, left circumflex coronary artery; RCA, right coronary artery; LMT, left main coronary trunk; CABG, coronary artery bypass grafting.

* $p < 0.05$ compared with group A.

† Data are expressed as the mean ± standard deviation.

‡ LMT ≥ 50% stenosis and LAD, LCX, and RCA ≥ 70% stenosis were present.

## RESULTS

Of 96 patients, 10 with no MR at stages 1 and 2 were excluded. The remaining 86 patients with MR between stages 1 and 2 were retrospectively divided into two groups: group A was composed of the patients who had an increase in MR jet area (n = 45), group B was composed of the patients who had a decrease in MR jet area (n = 41) at stage 2 compared with stage 1.

Patients' clinical and demographic characteristics are shown in Table 1. The incidence of patients with prior myocardial infarction was significantly higher ($p < 0.05$) in group B, compared with that in group A. The change of MR had no relation to the preoperative infarct location. Therefore, acute ischemic MR was not correlated with extent or region of prior myocardial infarction. There was no difference in other variables between these two groups. The preoperative ejection fraction was not available in all patients.

### Hemodynamic data

Hemodynamic data are shown in Table 2. Only group A was associated with a significant increase in PCWP at stage 2 compared with stage 1. Concerning the other hemodynamic data, HR and CO were significantly increased between stages 1 and 2 in the two groups. SAP and MAP were significantly decreased between stages 1 and 2 in the two groups.

**Table 2.** Heodynamic Data

| | Stage | Group A | Group B |
|---|---|---|---|
| HR (beat/min) | 1 | 65.9 ± 12.7 | 64.9 ± 14.8 |
| | 2 | 91.6 ± 20.6*** | 85.5 ± 11.5*** |
| SAP (mm Hg) | 1 | 121.6 ± 18.3 | 127.3 ± 22.5 |
| | 2 | 97.3 ± 15.9*** | 103.1 ± 17.0*** |
| MAP (mm Hg) | 1 | 83.8 ± 12.8 | 87.0 ± 14.6 |
| | 2 | 69.9 ± 10.6*** | 72.0 ± 10.8*** |
| PCWP (mm Hg) | 1 | 14.2 ± 5.2 | 15.1 ± 5.9 |
| | 2 | 16.5 ± 5.1*** | 14.8 ± 5.1 |
| CO (l/min) | 1 | 4.9 ± 1.7 | 4.5 ± 1.2 |
| | 2 | 6.3 ± 2.3** | 5.9 ± 1/6*** |
| SV (ml/beat) | 1 | 77.2 ± 29.1 | 72.1 ± 21.6 |
| | 2 | 69.2 ± 27.9 | 70.2 ± 20.9 |

Abbreviations: HR, Heart rate; SAP, systolic arterial pressure; MAP, mean arterial pressure; PCWP, pulmonary capillary wedge pressure; CO, cardiac output; SV, stroke volume.

Data are expressed as the mean ± standard deviation.

** $p < 0.01$ in the inter-stage comparison versus stage 1.

*** $p < 0.001$ in the inter-stage comparison versus stage 1.

**Table 3.** Transesophageal Echocardiography Data

| | Stage | Group A | Group B |
|---|---|---|---|
| EDA ($cm^2$) | 1 | 13.7 ± 5.6 | 15.4 ± 5.6 |
| | 2 | 13.9 ± 5.1 | 13.9 ± 5.0** |
| ESA ($cm^2$) | 1 | 7.8 ± 6.7 | 7.6 ± 4.3 |
| | 2 | 7.6 ± 6.2 | 6.8 ± 4.0* |
| %FAC (%) | 1 | 52.1 ± 18.8 | 52.4 ± 14.9 |
| | 2 | 51.6 ± 21.8 | 54.1 ± 16.7 |
| DMA (mm) | 1 | 32.7 ± 3.8 | 32.9 ± 3.3 |
| | 2 | 33.5 ± 3.8* | 32.3 ± 2.9 |
| MR area ($cm^2$) | 1 | 1.43 ± 1.50 | 1.87 ± 1.18 |
| | 2 | 2.72 ± 1.72*** | 0.93 ± 0.88***, † |

Abbreviations: EDA, End-diastolic area; ESA, end–systolic area; % FAC, percent fractional area change; DMA, diameter of the mitral annulus; MR area, mitral regurgitant area.

Data are expressed as the mean ± standard deviation.

* $p < 0.05$ in the inter-stage comparison versus stage 1.

** $p < 0.01$ in the inter-stage comparison versus stage 1.

*** $p < 0.001$ in the inter-stage comparison versus stage 1.

† $p < 0.001$ in the inter-group comparison versus group A.

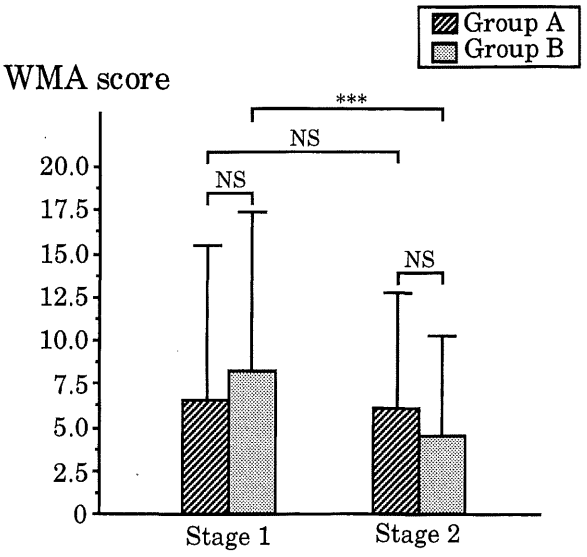


**Fig. 4.** Changes in the mean wall motion abnormality (WMA) score between stages 1 and 2.
The mean WMA score in group B decreased significantly, from 8.3 ± 9.1 to 4.6 ± 5.8. Data are expressed as the mean ± standard deviation.
***$p < 0.001$ in the inter-stage comparison. NS, Not significant.

### Transesophageal echocardiography data

The transesophageal echocardiography data are shown in Table 3. Between stages 1 and 2, EDA and ESA were significantly decreased in group B, but not in group A. The diameter of the mitral annulus (DMA) was significantly increased at stage 2 compared with stage 1 in group A, but there was no change in group B. MR area was significantly increased at stage 2 compared with stage 1 in group A and it was significantly decreased at stage 2 compared with stage 1 in group B. There were no significant differences between the two groups in other echocardiographic variables.

### Change in Wall Motion Abnormality Score

Fig. 4 shows changes in the mean WMA score, and Table 4 shows the relationship between changes in WMA score and changes in MR jet area between stages 1 and 2. At stage 1, the mean WMA score for group A was not significantly different from that for group B (6.6 ± 8.9 vs 8.3 ± 9.1; mean ± standard deviation; p = NS [Not significant]). In group A, there was no significant change in the mean WMA score between stages 1 and 2 (6.6 ± 8.9 vs 6.1 ± 6.6; p = NS). The WMA score increased between stages 1 and 2 more prominently in group A compared with group B (38% vs 2%). In group B, the WMA score decreased in 71% of patients and the mean WMA score was significantly lower at stage 2 compared with stage 1 (8.3 ± 9.1 vs 4.6 ± 5.8; $p < 0.001$).

**Table 4.** Relationship between Changes in Wall Motion Abnormality (WMA) Score and Changes in Mitral Regurgitant Jet Area*

| | Changes in WMA score | | |
|---|---|---|---|
| | ↑ | = | ↓ |
| Group A (n = 45) | 17 (38%) | 11 (24%) | 17 (38%) |
| Group B (n = 41) | 1 ( 2%) | 11 (27%) | 29 (71%) |

*The difference was statistically significant ($p < 0.001$).

In group A, 17 patients had an increased WMA score (10 patients [59%] with newly developed RWMA, 7 patients [41%] with worsened RWMA), and the remaining 28 patients had a decreased or unchanged WMA score. On the other hand, in group B, only one patient with worsened RWMA had an increased WMA score, and the remaining 40 patients had a decreased or unchanged WMA score.

Table 5 shows the site of RWMA in 17 patients with an increased WMA score in group A. In 16 of these 17 patients (94%), RWMA was detected in the right coronary artery (RCA) and/or left circumflex coronary artery (LCX) areas. In 7 of those 17 patients (41%), RWMA was detected in the basal inferior segment by LAV. Newly developed RWMA was detected in 10 of those 17 patients; in 6 patients by SAV, and in 4 patients by both views. Fig. 5 shows the incidence of RWMA in each segment in those 17 patients. The

**Table 5.** Site of the Regional Wall Motion Abnormality (RWMA) in 17 Patients with Increased WMA Score in Group A

| | Site of RWMA | | | | |
|---|---|---|---|---|---|
| | SAV | | | LAV | |
| Patient no. | LAD† area | LCS† area | RCA† area | LAD† area | RCA† area |
| 1 | | | I-VS | | |
| 2 | | IP | I, I-VS | a-I | b-I |
| 3 | M-VS | IP | I, I-VS | a-I | b-I |
| 4 | A-VS | IP* | I, I-VS | a-I | |
| 5 | | | I* | | b-I* |
| 6 | | IP* | I | | |
| 7 | M-VS | | I, I-VS | | |
| 8 | AL* | IP*, PL* | | a-I*, a-A* | |
| 9 | | | I* | | |
| 10 | | | I* | | b-I |
| 11 | A | | | | |
| 12 | | IP | I*, I-VS | a-I*, a-A* | b-I* |
| 13 | AL | IP*, PL* | I* | a-I | |
| 14 | | | I* | | b-I* |
| 15 | | | I* | | b-I |
| 16 | | | I-VS | | |
| 17 | | | I-VS | | |

Abbreviations: SAV, Short-axis view; LAV, long-axis view; LAD, left anterior descending coronary artery; LCX, left circumflex coronary artery; RCA, right coronary artery; a-A, apical anterior left ventricle; a-I, apical interior left ventricle; b-I, basal inferior left ventricle; A, anterior left ventricle; AL, anterolateral left ventricle; I, inferior left ventricle, IP, inferoposterior left ventricle; PL, posterolateral left ventricle; A-VS, anterior ventricular septum; M-VS, middle ventricular septum; I-VS, inferior ventricular septum.

* Newly developed RWMA.

† RWMA in LAD area (M-VS, A-VS, A, AL in SAV and a-A, a-I in LAV) is perfused by left anterior descending coronary artery. RWMA in LCX area (PL, IP in SAV) is perfused by left circumflex coronary artery. RWMA in RCA area (I, I-VS in SAV and b-I in LAV) is perfused by right coronary artery.

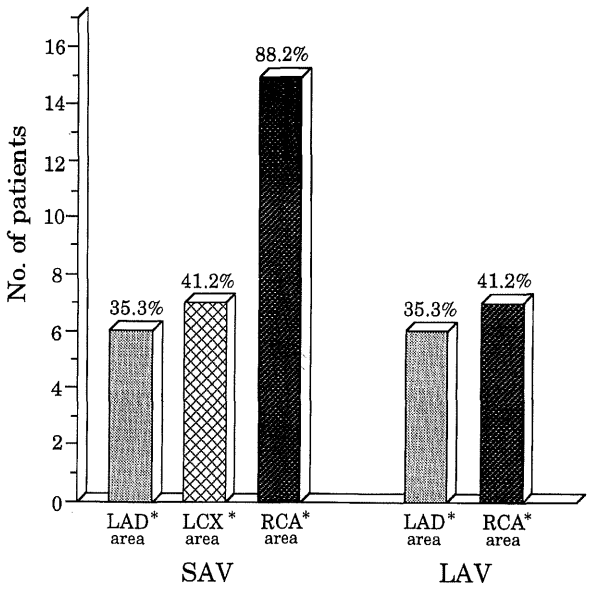


**Fig. 5.** Incidence of the regional wall motion abnormality (RWMA) in each segment in 17 patients with an increased WMA score in group A. The highest percentage of patients showed RWMA in the RCA area of SAV.
SAV, Short-axis view; LAV, long-axis view.
*LAD area supplied by left anterior descending coronary artery (middle and anterior ventricular septum, anterior and anterolateral left ventricle in SAV; apical anterior and apical inferior left ventricle in LAV). LCX area supplied by left circumflex coronary artery (posterolateral and inferoposterior left ventricle in SAV). RCA area supplied by right coronary artery (inferior left ventricle and inferior ventricular septum in SAV; basal inferior left ventricle in LAV).

percentages of patients with RWMA in the left anterior descending coronary artery (LAD), LCX, and RCA areas by SAV were 35.3%, 41.2%, and 88.2%, respectively. Further, the percentages of patients with RWMA in the LAD and RCA areas by LAV were 35.3% and 41.2%, respectively.

The accuracy of qualitative analysis by visual inspection was randomly selected in 480 segments of the SAV and 240 segments of the LAV. The intraobserver accuracy for RWMA was 93.3% in the SAV, and 87.9% in the LAV, while its concordance was 99.6% in the SAV, and 99.2% in the LAV with the observation 6 months after initial analysis. The interobserver accuracy was 86.7% in the SAV, and 80.4% in the LAV, while its concordance was 98.3% in the SAV, and 95.8% in the LAV.

### Change in MR area and PVSF

Fig. 6 shows changes in the MR area among stages 1, 2 and 3. In 1 patient in group A and 2 in group B, the MR area was not measurable at stage 3 because of the limitation in measurement time. In 37 patients in group A, the increased MR following CPB returned to the initial level by the end of surgery. In the remaining 7 patients in group A, MR increased continuously to the end of surgery despite treatment. The degree of MR at stage 3 in these 7 patients was significantly larger than the degree of MR in the remaining 37 patients in group A ($3.06 \pm 1.85$ cm$^2$ vs $1.05 \pm 1.02$ cm$^2$; $p < 0.001$). Moreover, in 2 of these 7 patients, PVSF decreased (peak systolic flow velocity < peak diastolic flow velocity) at stage 2

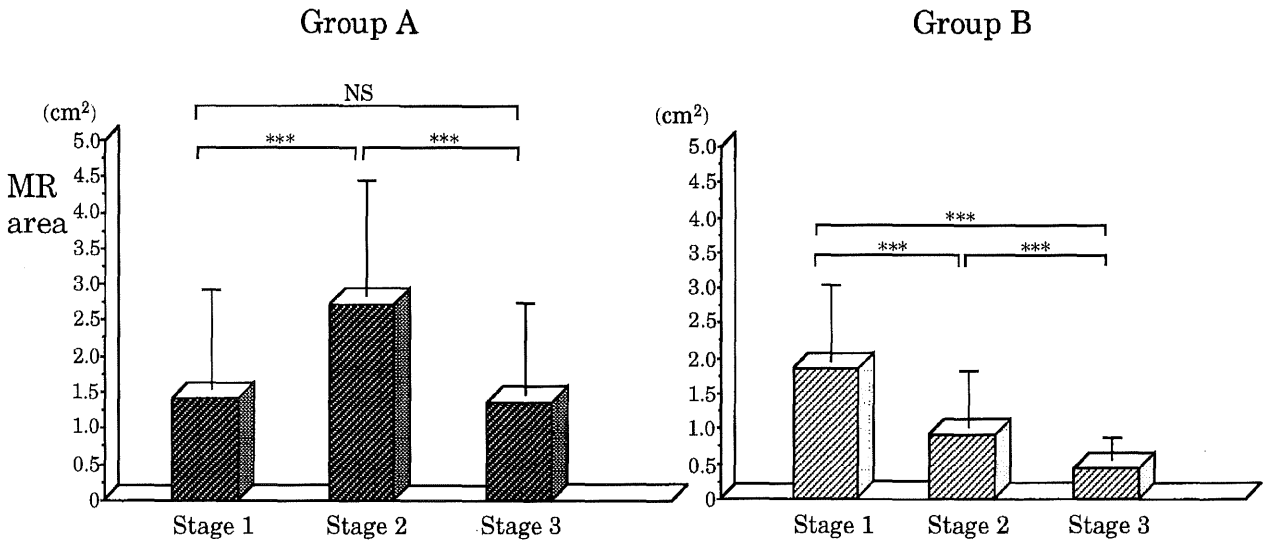


**Fig. 6.** Changes in mitral regurgitant area (MR area) among stages 1, 2, and 3.
In 1 patient in group A and 2 in group B, the MR area was not measurable at stage 3. In group A, increased MR following CPB returned to the initial level by the end of surgery. On the other hand, in group B, MR decreased by degrees at each stage.
***$p < 0.001$ in the inter-stage comparison. NS, Not significant.

and persisted to stage 3. Of these 2 patients, one had a newly developed RWMA following CPB, which was persistent to the end of surgery, and perioperative myocardial infarction (PMI) was noted in the inferior wall. This patient had intermittent AF in the postoperative course but was discharged on postoperative day (POD) #7. Another patient had continued AF despite direct-current (DC) cardioversion but was discharged on POD #15.

In the remaining 5 patients with decreased PVSF at stage 2, decreased PVSF was normalized by the end of surgery and their postoperative course was uneventful.

In group B, the degree of MR decreased progressively at each stage, and the postoperative course of all patients was uneventful.

### MR Jet Direction

MR jet direction changed in only 6 patients between stages 1 and 2; in 4 patients in group A, from central to eccentric, and in 2 patients in group B, from eccentric to central. In all other patients, MR jet direction did not change to the end of surgery, even in 7 patients with persistently increased MR.

### Treatment

Table 6 shows the requirement for inotropic support (dobutamine, dopamine, amrinone, epinephrine, norepinephrine), vasodilator agents (nitroglycerin, nitroprusside) and IABP. In group A, 33 of 44 patients (1 patient excluded from analysis because MR area was not measureable at stage 3) were treated with either inotropic and/or vasodilator agents, and in 26 of those 33 patients (79%), MR decreased by the end of surgery. However, in the remaining 7 patients (21%), MR increased continuously to the end of surgery despite treatment. In the remaining 11 patients in group A, MR decreased by the end of surgery without therapy.

In group B, 24 of 39 patients (2 patients excluded from analysis for the same reason as in group A) were treated with either inotropic and/or vasodilator agents, and in 22 of those 24 patients (92%), MR decreased continuously to the end of surgery with either an increased, or decreased dose. In the remaining 15 patients in group B, MR continuously decreased to the end of surgery without therapy.

**Table 6.** Requirement for Inotropic Support, Vasodilator Agents and Intra-aortic Balloon Pump (LABP)*

| | inotropic support | | | vasodilator agents | | | IABP |
|---|---|---|---|---|---|---|---|
| Stage | 1 | 2 | 3 | 1 | 2 | 3 | 2–3 |
| Group A (n = 45) | 0 | 17 | 16 | 19 | 29 | 32 | 3 |
| Group B (n = 41) | 1 | 9 | 13 | 14 | 21 | 22 | 0 |
| p Value | NS | NS | NS | NS | NS | NS | NS |

*1 patient in group A and 2 in group B were excluded at stage 3. NS, Not significant.

**Clinical outcomes**

Although their MR decreased by the end of surgery, three patients in group A died with sustained ventricular tachycardia, acute respiratory arrest due to left vocal cord paralysis, and cardiogenic shock, respectively.

## DISCUSSION

Using color Doppler imaging, authors have reported that trivial MR was found in 38–45% of healthy subjects[27] and in 53% of patients with previous myocardial infarction[10]. The presence of significant residual ischemic MR following CPB is an important prognosticator for patients undergoing CABG[21].

The precise etiology of chronic ischemic MR appears to be multifactorial and difficult to define clearly. Previous clinical studies have proposed that ischemic MR occurs secondary to papillary muscle dysfunction[2], incomplete mitral leaflet closure[5], mitral annular dilatation[10], and accompanying ventricular dyskinesia or dilatation[5]. These clinical studies have focused on chronic MR associated with ischemia due to coronary artery disease. In canine studies, Llaneras and coworkers[15] concluded that neither posterior papillary muscle nor left ventricular dilatation alone produces ischemic MR after moderate inferior wall infarction, but that the combination is necessary. The prognostic significance of acute transient ischemic MR in patients undergoing CABG has not previously been assessed. Moreover, the potential benefits of appropriate therapy for mitral insufficiency that worsens intraoperatively have not been firmly established. Thus we evaluated morphological factors and perioperative treatment strategies which influence the outcome of acute ischemic MR.

In clinical studies, Fehrenbacher and coworkers[4] reported that stress-induced MR was associated with mitral leaflet displacement secondary to ventricular dyskinesia rather than to mitral annular dilatation. Pinson and colleagues[19] reported that the degree of ischemic MR was correlated with the wall motion score in the diaphragmatic segment, not other segments. In our study, the increase in mitral annular diameter and the worsening in RWMA in RCA and/or LCX areas were considered to be the main mechanism involved in acute ischemic MR following CPB. Such RWMA could affect the posterior papillary muscle so that it fails to move toward the annulus or that it moves away from the annulus during systole. Posteromedial papillary muscle involvement has two major causes as etiologic factors for ischemic MR. First, anatomically, the posteromedial papillary muscle is more vulnerable to ischemia, infarction, and rupture than the anterolateral papillary muscle because of its single blood supply from either RCA or LCX. The anterolateral papillary muscle is supplied dually from both LAD and LCX[23,25]. Second, the inferoseptal and inferior myocardial segments supplied by RCA are not well protected regardless of the cardioplegia delivery method used. Regional function abnormalities in this area were common following CPB[17].

Intraoperative TEE is a sensitive and useful means of assessing RWMA indicative of myocardial ischemia in the specific territory of each coronary artery at SAV together with the 4 segments of LAV. Reports[6,24] have shown that transmural myocardial infarction occurred most commonly in the basal inferior and apical anterior segments, which were supplied from RCA and LAD, respectively. In our study, RWMA in the basal inferior segment was often associated with an increase of acute ischemic MR compared with RWMA in other parts of LAV. The evaluation in LAV was slightly inferior as compared with that of SAV due to a difference in the degree of endocardial excursion in the basal and apical portions, being greater at the apex.

Ventricular size (EDA and ESA) and %FAC had little influence in acute ischemic MR. However, mitral annular dilatation caused acute ischemic MR following CPB. It appears that an acute change in the global left ventricle during CABG alone does not produce sufficient mitral annnular dilatation to cause MR. Therefore, the pathophysiology of progressive acute ischemic MR must be due to misalignment of the chordae and papillary muscle by the annular dilatation and remodeling.

TEE provides an excellent image of the pulmonary veins. Klein and coworkers[12] reported that changes in pulmonary venous flow patterns (ranging from normal to blunted or reversal of systolic flow) may be potential physiological predictors of the severity of MR. In our study, post operative AF was noted in 2 patients with acute ischemic MR associated with persistently decreased PVSF following CPB. The finding of persistently reduced PVSF may be important in assessing the physiological significance of residual MR. Acute ischemic MR was also significantly related with an increase in PCWP. However, it must be acknowledged that PCWP does not faithfully capture the dynamics of pulmonary venous systolic filling fraction[13].

It is clearly recognized that eccentric MR jets directed at the left atrial wall suffer a loss of energy and hence may appear smaller than the comparable central regurgitant volume[3]. In our study, MR jet direction had little influence in determining the amount of MR.

Ischemic MR remains a relatively high risk prognostic factor[21], but it is difficult to state what magnitude of MR change will be required to be considered significant. The majority of acute ischemic MR was treated successfully following

CPB by pharmacological intervention to decrease preload and/or increase LV contractility. However, when acute ischemic MR is associated with changes in PVSF following CPB, additional drug treatment or surgical therapy may be required to prevent postoperative complications such as further hemodynamic compromise, prolonged hospitalization and embolic stroke, associated with AF[26].

The present study has some limitations. First, SAP in the post bypass period was found to be significantly below the prebypass value in both groups. This could lead to inappropriate underestimation of the severity of MR[9]. However, it is important to realize that the relationship between MR jet size and SAP is not reciprocal[14]. Moreover, Fehrenbacher and coworkers[4] reported that transient MR did not correlate significantly with an increase in SAP. Many other physiological variables such as atrial pressure, size and compliance, pulmonary venous compliance, regurgitant orifice size and geometry, and papillary muscle function may influence regurgitant size. Additionally, a significant increase in HR could alter the assessment of the severity of MR. Second, current clinical color Doppler grading of MR is based on spatial criteria, using single still frames to measure the maximal two-dimensional area occupied by the regurgitant jet, or a ratio of jet area to the cross-sectional area of the receiving atrium[1,7]. MR measured with transesophageal color Doppler imaging has several potential limitations for quantitative analysis because it only assesses flow velocity, and not necessarily flow volume. However, we used this method because an excellent correlation with angiography has been demonstrated when the maximum MR area obtained from both transverse and longitudinal scans by biplane transesophageal color Doppler imaging was used[6]. In the near future, with the advent of multiplane TEE, clinical diagnostic capabilities will be expanded[22]. Third, measurements on left atrial size and compliance, and afterload (systemic vascular resistance) are not described in our study, because these data were not available in all patients. Finally, the patients with clinically significant MR due to the abnormality of the leaflets or chordae and/or valve disease who needed an additional procedure were excluded from this study. Therefore, the conclusions are limited to patients with normal leaflets and chordae.

In conclusion, (1) the increase in annular diameter and worsening in RWMA in the RCA and/or LCX areas were considered to be the main mechanisms involved in acute ischemic MR following CPB. (2) The majority of cases of acute ischemic MR following CPB were resolved by pharmacological intervention by the end of surgery. (3) Post operative AF was noted in 2 patients with acute ischemic MR associated with decreased PVSF, which was persistent to the end of surgery despite treatment.

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