原 著 (Original)

Evaluation of Mitral Regurgitation During Coronary Artery Bypass Surgery by Transesophageal Color Doppler Imaging

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Summary

We studied 1) incidence and degree of mitral regurgitation (MR) during coronary artery bypass surgery and 2) relationship between change in degree of MR and ventricular function, i. e., left ventricular end-diastolic and end-systolic area, area ejection fraction, diameter of mitral annulus, and regional wall motion abnormality (RWMA). Transesophageal color Doppler imaging detected MR in all 20 patients after induction of anesthesia; 15 patients showed trivial MR, and only 5 had mild MR. However, 8 patients showed trivial, 11 mild, and 1 monderate MR following cardiopulmonary bypass (CPB). MR increased following CPB in 9 of 20 patients (45%). These 9 patients showed 1) an enlarged left ventricle and low area ejection fraction even before CPB compared with others without the increase in MR, 2) mitral annular dilatation in the post-CPB, and 3) more frequent and severe RWMA in both pre- and post-CPB.

Introduction

Transesophageal color Doppler imaging (CDI) facilitates detection of mitral regurgitation (MR) because the transducer is close to the mitral valve. It has been reported that many patients with coronary artery disease have MR, and ventricular dilatation and papillary muscle dysfunction are most likely the cause of MR in patients with myocardial infarction.[1] We have often observed MR during coronary artery bypass surgery (CABS). However, these functional changes of MR during CABS have not yet been reported in detail. In this study, we examined 1) incidence and severity of MR and 2) relationship between grade of MR and left venticular function, i. e., end-diastolic and end-systolic area of left ventricle, area ejection fraction, diameter of mitral annulus, and regional wall motion abnormality of left ventricle.

Methods

This study was approved by the institutional research committee, and informed consent was obtained from each patients. Twenty patients undergoing elective CABS were studied. There were 15 men and 5 women with a mean age of 62.5 years (range from 49 to 74). Patient who had valvular disease or dysrhythmia were excluded from the study. Fourteen patients had a history of myocardial infarction; 5 had inferior

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wall infarction, 8 had anteroseptal infarction and 1 had both.

Transesophageal CDI examination was performed with Toshiba SSH-140A system (Japan). Following tracheal intubation, a 5 MHz phased-array transesophageal echocardiographic probe (PEF-511SA, Toshiba) was inserted and connected to the echocardiographic system. Measurements of MR jet area by CDI were performed at the following four stages: stage 1—after induction of anesthesia; stage 2—in the pre-cardiopulmonary bypass (CPB) period before cannulation; stage 3—in the post-CPB period after decannulation; and stage 4—at the end of the operation following closure of the chest wall. Echocardiographic data were recorded on VHS video tapes. Measurements of MR jet area, left ventricular end-diastolic area, end-systolic area, area ejection fraction, diameter of mitral annulus, and regional wall motion abnormality were performed. Mitral regurgitant jet area was measured with a 45° color sector, at 10 frames/sec scanning rate, and a 4 kHz pulse-repetition frequency. Care was taken to use an optimal gain setting, which is defined as maximal gain level without introduction of signals outside of flow area or onto tissues. To obtain the largest jet area, a complete scan of the left atrium was performed by tilting, withdrawing and advancing the tip of the probe. The outline of MR signals was traced with a track ball (Fig. 1). Three measurements of regurgitant jet area were averaged. Grade of MR was divided into four grades according to MR jet area. MR jet area of less than 1.5 cm^2, between 1.5 and 4.0 cm^2, between 4.0 and 7.0 cm^2, and more than 7.0 cm^2 correspond to trivial, mild, moderate, and severe regurgitation, respectively.[2]

Left ventricular end-diastolic area and end-systolic area at the mid-papillary muscle level were obtained using ECG synchronization mode. Area ejection fraction was calculated from these areas (Fig 2). The diameter of the mitral annulus was also measured using ECG synchronization mode (Fig. 3). The diameter of the mitral annulus was defined as the end-systolic distance from the junction of the anterior mitral leaflet and aortic root to the junction of the posteiror mitral leaflet and the atrioventricular groove.[3] The position of the probe was adjusted to obtain the largest

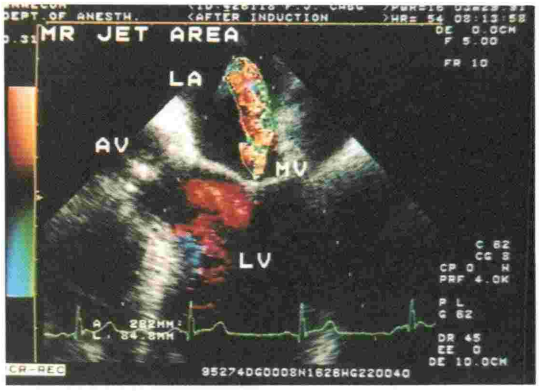

Fig. 1 Transesophageal color Doppler imaging of mitral regurgitation. Mitral regurgitant jet area was traced with a track ball and calculated. LA: left atrium. MV: mitral valve. LV: left ventricle. AV: aortic valve.

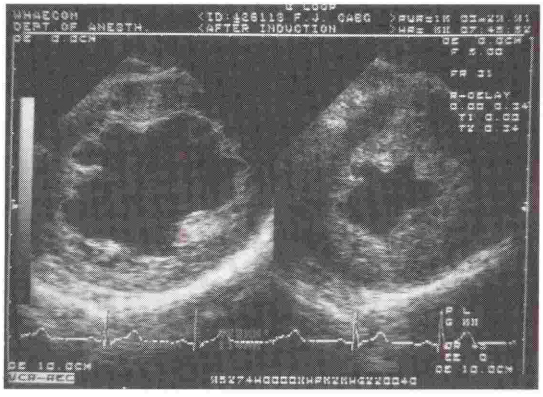

Fig. 2 Transgastric short-axis views of the left ventricle at mid-papillary muscle level. End-diastolic (left) and end-systolic view (right) were obtained with ECG trigger. Endocardial border was traced with a track ball. End-diastolic and end-systolic area were calculated with an on-line computer.

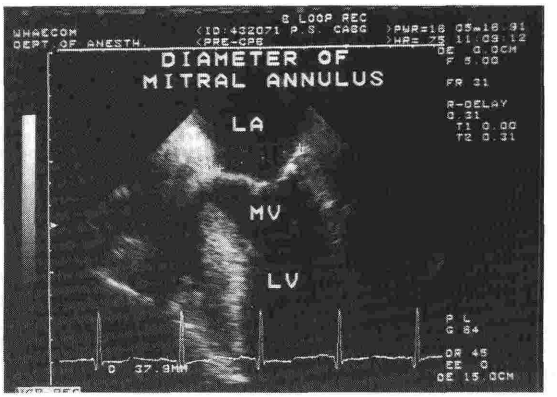

Fig. 3 Transesophageal long-axis view of mitral valve showing measurement of the diameter of the mitral annulus. This panel shows an end-systolic view obtained with ECG trigger. The diameter of the mitral annulus is the distance between (+) the junction of anterior leaflet and aortic root and (+) the junction of the posterior leaflet and atrioventricular groove. LA: left atrium. MV: mitral valve. LV: left ventricle.

diameter of the annulus. The short-axis view at the mid-papillary muscle level was divided into 8 segments using nomenclature recommended by the American Society of Echocardiography.[4] The wall motion of each of the 8 segments was graded as follows: normal, mild hypokinesis, severe hypokinesis, akinesis, and dyskinesis. Newly developed regional wall motion abnormality was defined as any of those segments worsened by two or more grades.

Hemodynamic variables, heart rate (HR), systolic arterial pressure (SAP), mean arterial pressure (MAP), pulmonary capillary wedge pressure (WP), cardiac output (CO), stroke volume (SV), and systemic vascular resistance (SVR) were evaluated. Those data were obtained simultaneously with the echocardigraphic measurements.

Statistical analysis was performed with student t-test, chi-square test, and Fisher's exact test. $P<0.05$ was considered to be significant.

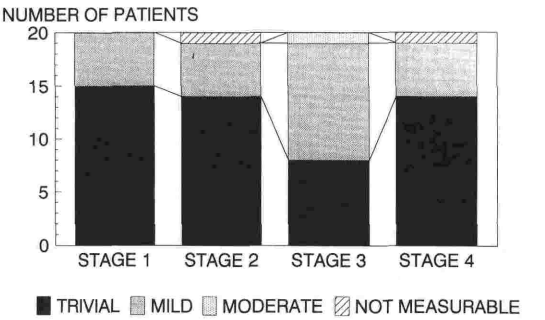

Fig. 4 Grade of mitral regurgitation during coronary artery bypass surgery at four stages.

Results

Transesophageal CDI detected MR in all 20 patients. Mitral regurgitant jet areas were 0.92 ± 0.67 cm^2, 1.18 ± 0.73 cm^2, 2.15 ± 1.47 cm^2, and 0.90 ± 0.69 cm^2 at stages 1, 2, 3 and 4, respectively. At stage 1, 15 patients (75%) showed a trivial grade of MR, and only 5 patients (25%) had mild MR. However, at stage 3, 8 patients (40%) showed trivial MR, 11 patients (55%) had mild MR and 1 patient (5%) had moderate MR. At stage 4, 14 patients (70%) showed trivial MR, 5 patients (25%) had mild MR, and 1 was not able to measure (Fig. 4).

Patients were divided into two groups: Group A was defined as patients that had a grade of mitral regurgitation worsened by one or more grades from stage 1 to stage 3, and group B consisted of those that had no increase in grade of mitral regurgitation. In group A (9 patients), end-diastolic area and end-systolic area were significantly larger and area ejection fraction was significantly ($p<0.01$) lower at both stages than those of group B (11 patients). Also in group A, the diameter of mitral annulus was significantly ($p<0.01$) larger at stage 3 than at stage 1. However, there was no significant change of annular diameter in group B (Table 1).

Fig. 5 showed the location and degree of regional wall motion abnormality (RWMA) at

stage 1 and 3 in all patients. All patients in group A and 5 of 11 in group B had RWMA (Table 2). This difference was statistically significant ($p<0.05$). Seven of 9 patients in group A had severe hypokinesis or akinesis at stage 3. However, no patients in group B had severe hypokinesis or akinesis at mid-papillary muscle level at both stages. Severity of RWMA was significantly ($p<0.05$) higher in group A than group B. There was no difference in location of RWMA between groups. One patient in group A had newly developed RWMA at mid-papillary muscle level at stage 3. Another patient in group B also had newly developed RWMA, but the RWMA was located only in the apex of the left ventricle.

There were no differences in hemodynamic variable between group A and group B at stages 1 and 3 (Table 3). Inotropic agents were administrated in 4 of 9 patients in group A (norepinephrine in 3, dobutamine and epinephrine in 1), and 3 of 11 patients in group B (norepinephrine in 1, dobutamine in 2). There was no difference in incidence of the use of inotropic agents between groups.

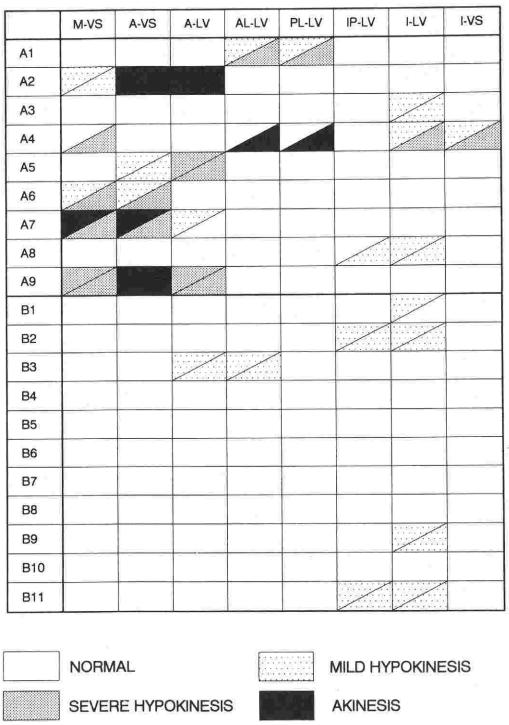

Fig. 5 The location and grade of regional wall motion abnormality at stage 1 and stage 3 in all patients. Upper left half of box: stage 1; and lower right half of box: stage 3. A: group A, B: group B

Table 2 Relationship between mitral regurgitation and regional wall motion abnormality.

	regional wall motion abnormality	
	+	−
Group A: MR ↑	9	0
Group B: MR →	5	6

Discussion

In our hospital, transesophageal echocardiography is used as an essential monitor to assess both global and regional left ventricular function during CABS. We often observed MR during CABS. MR is found in 35%-43% of

Table 1 Comparison of echocardiographic measurements between patients with increased grade of MR (group A) and those without change in grade of MR (group B) at stages 1 and 3.

	Stage	E-D (cm^2)	E-S (cm^2)	aEF (%)	DMA (cm)
Group A	1	15.6±4.0	9.7±4.3	40.4±12.1	3.3±0.3
(n=9)	3	16.1±2.9	10.0±3.2	39.1±10.2	3.6±0.3***
Group B	1	12.0±2.4*	4.2±1.4**	64.8±11.2**	3.3±0.4
(n=11)	3	10.9±2.2**	4.0±1.5**	63.4±10.4**	3.2±0.4

E-D: left ventricular end-diastolic area. E-S: left ventricular end-systolic area. aEF: area ejection fraction. DMA: diameter of mitral annulus.

*: $p<0.05$, **: $p<0.01$, Group A vs B, ***: $p<0.01$, Stage 1 vs 3

Table 3 Hemodynamic variables in patients with increased grade of MR (Group A) and those without change in grade of MR (Group B).

	Stage	HR (beat/min)	SAP (mmHg)	MAP (mmHg)	WP (mmHg)
Group A	1	67.0± 9.2	113±17	79±13	8.2±4.2
(n=9)	3	96.0±11.8**	101±11*	71± 7*	10.6±2.6
Group B	1	61.3±10.5	118± 7	82± 8	7.6±3.7
(n=11)	3	86.2±11.8**	99±12**	70±10**	9.9±3.0

	Stage	CO (*l*/min)	SV (m*l*/beat)	SVR (dynes·sec/cm^5)
Group A	1	3.8±0.6	57.8±15.9	1534±165
(n=7)	3	5.0±1.3*	52.8±16.9	1039±236**
Group B	1	3.7±0.7	61.4± 9.7	1703±331
(n=11)	3	5.4±1.1**	63.4±15.5	978±327**

*, **: $p<0.05$, $p<0.01$, stage 1 vs stage 3

HR: heart rate. SAP: systolic arterial pressure. MAP: mean arterial pressure. WP: pulmonary capillary wedge pressure. CO: cardiac output. SV: stroke volume. SVR: systemic vascular resistance.

healthy subjects and 84% of patients with myocardial infarction because CDI is very sensitive in detecting MR.[1,5,6] When MR jet is small (<1.0 cm^2) in area, short (<30 mm) in length, and narrow (<10 mm) in width, MR is considered to be physiological (trivial). In this study, MR was found in all 20 CABS patients and was trivial in 15 od 20 (75%) patients after induction of anesthesia. In 9 of 20 (45%) patients, however, the grade of MR increased by one grade to mild or moderate following CPB. Worsening of MR was trasient, and MR improved at the end of operation as same level as after induction of anesthesia.

Patients were divided into two groups to examine the cause of worsening MR: 9 patients had worsened MR, and 11 patients had no changes in MR. There were no differences in hemodynamic variables between two groups. Our study showed that mitral annular dilatation was a major cause of worsening MR in the post-CPB period, although left ventricle tended to be dilated but these change were not statistically significant. Furthermore, those patients whose MR worsened following CPB were significant for enlarged left ventricle and low area EF even before CPB compared with others without increased MR. They also had RWMA in both the pre- and post-CPB. There were no increase of MR in patients without RWMA. New development of RWMA was a less causative factor, beacuse only one of patients in group A had newly developed RWMA at the mid-papillary muscle level in the post-CPB period. Furthermore, one patient in group B had newly developed RWMA at the apex of left ventricle.

Previous studies[1,7] have been shown that there are two major causative factors of MR accompanying myocardial infarction; (1) papillary muscle dysfunction and/or dysfunction of the immediately adjacent left ventricular myocardium. (2) annular and/or ventricular dilatation. These reports studied patients with old myocardial infarction. These results may not be implicated in an acute change during operation. Recently, Kaul S et al.[8] studied the mechanism of ischemic MR using an acute canine model in which the circulation to the papillary muscles was isolated from that of the rest of the LV. They concluded that papillary muscle dysfunction and/or dysfunction of immediately adjacent

LV myocardium does not result in MR and that MR occurs only when global LV function is affected during ischemia. They also reported that ischemic MR is related to the end-systolic mitral annular dimension (r=0.91), the LV end-diastolic size (r=0.78) and the end-diastolic LA size (r=0.74). We and others[3,8,9] showed that enlargement of the mitral annulus was most closely associated with the development of MR, although some reports[1,10] showed that mitral annular dilatation was not a major determinant for MR.

RWMA was frequently detected after induction of anesthesia in the group with increased MR. Myocardial preservation may be insufficient during CPB in patients with severe stenosis, total occlusion, or multiple lesion. Insufficient myocardial preservation is supposed to result in the decreased global left ventricular function following CPB in these patients.

Study limitation: 1) We used CDI in the assessment of MR because good correlation has been demonstrated with the most widely accepted angiographic grading. Current color Doppler grading of MR is based on spacial criteria, using single still frame to measure maximal two-dimensional area of the regurgitant jet, or a ratio of jet area to the area of the left atrium.[11,12] Color Doppler jet area continues to be used as a semiquantitative indicator of severity of MR until quantitative method can be proven to be clinically applicable.[13,14] We evaluated MR jet area with monoplane transducer based on Yoshida's biplane scoring system.[2] Measurements with biplane transducer is more associated with angiographic grading than that with monoplane probe, although we tried to obtain the largest jet area by manipulating the probe. 2) One observer measured all echocardiographic data in this study. Echocardiographic data of 6 patients were re-examined 6 months later to evaluate intraobserver variability. Intraobserver correlation was excellent for the MR jet area (r=0.93). The intraobserver correlation for the left ventricular end-diastolic area, end-systolic area, area ejection fraction, and diameter of mitral annulus were r=0.97, 0.99, 0.98 and 0.95, respectively. Interobserver variability was not evaluated in this study, but Smith et al.[15] reported that interobserver correlation for MR jet area was excellent as well as intraobserver corelation.

The major causative factor for ischemic MR following CPB in patients undergoing CABS was not papillary muscle dysfunction, but the decreased global left ventricular function and mitral annular dilatation. Increased MR following CPB returned to the initial level by the end of surgery in most of cases. Those patients should be first treated with vasodilators and/or inotropic agents[16] to decrease preload and/or increased contractility when increased MR is detected with transesophaeal CDI. Surgical intervention for the mitral valve needs careful consideration. Transesophageal echocardiography was able to offer many information on severity and cause of MR during CABS.

In conclusion, the grade of MR increased following CPB in 9 of 20 patients. These 9 patients differed from the patients without increased MR in the post-CPB period in the following aspects: 1) an enlarged left ventricle and low area ejection fraction even before CPB, 2) mitral annular dilatation in the post-CPB period, and 3) more frequent and severe RWMA in both the pre- and post-CPB period.

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経食道心エコー・カラードプラ法による冠動脈再建術中の僧帽弁逆流の評価

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著者らは，(1)冠動脈再建術中の僧帽弁逆流(MR)の頻度と重症度，(2)MRの重症度の変化と左室機能，すなわち，左室拡張末期面積，収縮末期面積，面積駆出率，僧帽弁輪径，そして局所壁異常運動との関連について研究を行なった．経食道心エコー・カラードプラ法により20例すべてに麻酔導入後MRを検出した．15例はtrivial（生理的）MR，そして5例のみmild（軽度）MRであった．しかしながら，体外循環後では8例のみがtrivialであり，11例がmild MR，1例がmoderate（中程度）MRであった．MR体外循環20例中9例（45%）で増加した．これら9例の患者では，(1)MRが増悪しなかった症例と比較して，左室の拡張，面積駆出率の低値を認め，(2)体外循環後の僧帽弁輪の拡大，(3)体外循環前後とも，局所壁運動異常を認める頻度が高くかつ重篤な局所壁運動異常の存在が指摘された．