Efficacy of Coronary Artery Bypass Grafting in Patients With a Dilated Left Ventricle due to Myocardial Infarction

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Myocardial infarction (MI) induces ventricular enlargement as an initial compensation to maintain stroke volume after the loss of contractile tissue (ie ventricular remodeling) accompanied by a persistently depressed ejection fraction (EF). This ventricular dilatation has been associated with a reduction in survival rate.

Left ventricular function has usually been described in terms of the EF, but it is not clear whether the EF is the most meaningful index of this function in the postinfarction situation. White et al reported that end-systolic volume (ESV) was the primary predictor of survival after MI, and that patients with a dilated left ventricle (LV) after MI had a poorer prognosis than those with a non-dilated LV, even if the EF values were the same. Moreover, they reported that coronary artery bypass grafting (CABG) showed no predictive value. We have also reported that the most significant factor influencing long-term prognosis after CABG was left ventricular ESV. These results may indicate a limitation to the efficacy of CABG, including exercise tolerance, in patients with a dilated LV due to MI.

In order to clarify the efficacy of CABG and the causes of the poor prognosis in those patients with a dilated LV due to MI, who had obtained complete revascularization, we evaluated pre- and postoperative left ventricular function and examined the responses to exercise stress in the chronic stage after CABG.

Methods

Study Population

Among 70 patients who underwent CABG only at Sakurabashi Watanabe Hospital from 1991 to 1994, 20 patients had an enlarged LV with an ESV index (ESVI) over 60 ml/m² (ie twice the normal value) due to prior MI. We studied 16 patients (2 females, 14 males; age, 60±8 years, range, 48–72) who had obtained complete revasc-
The subjects consisted of 2 patients with 2-vessel disease and 14 patients with 3-vessel disease. Four patients had MI on 1 of 3 major vessels (left circumflex artery, left anterior descending artery and right coronary artery), 10 patients had MI on 2 vessels, and 2 patients had MI on 3. The indication for bypass grafting in all patients was postinfarction angina. Informed consent was obtained by all patients, including for the postoperative exercise study.

The preexisting MI was defined by history, electrocardiographic findings, echocardiography, and coronary arteriography. All MI areas had wall motion abnormalities (hypokinesis or akinesis) as observed by left ventriculography and 2-dimensional echocardiography. In those areas with wall motion abnormalities, there were good collateral vessels or spontaneous recanalized vessels. Those areas also had myocardial viability, which was assessed by 201Tl myocardial scintigraphy and myocardial contrast echocardiography. In one patient with a preoperative ESVI of 228 ml/m² myocardial viability was also assessed by dobutamine stress ventriculography and echocardiography. We did not include patients with dyskinetic areas in the study.

Surgical Technique

Coronary artery bypass grafting was performed under cardiopulmonary bypass. For myocardial preservation, systemic moderate hypothermia (25–28°C), topical iced saline (4°C) and cold crystalloid cardioplegic solution (4°C) were used. Revascularization with autogenous saphenous vein grafts and internal thoracic arteries was performed by continuous suture technique under cardiac arrest by the same surgeon. Mean aortic cross-clamp time was 86±22 min (excluding the patient noted earlier with the preoperative ESVI of 228 ml/m² with ventricular fibrillation), and mean ECC time was 141±32 min. The average number of coronary artery bypass grafts placed per patient was 2.6±0.7 because 10 of the 16 patients had had percutaneous transluminal coronary angioplasty preoperatively.

Cardiac Catheterization Procedures

Cardiac catheterization and coronary arteriography were performed via a femoral vein and artery just before and 7.1±2.5 months (range, 3–12) after CABG. All medication was withheld for at least 12–18 h before the catheterization. Pressure measurement through a fluid-filled catheter connected to a Statham P23Db or P23ID strain gauge (Spectramed Inc. Cardiovascular Products Division, Oxnard, CA, USA), cardiac output determination by the indicator-dilution method and blood gas sampling from the aorta and pulmonary artery were performed just before left ventriculography.

The left ventriculogram was obtained with 35 mm cineangiograms (30° right anterior oblique projection and 60° left anterior oblique projection), after which coronary arteriography was done. Coronary stenosis and graft patency were assessed by 2 independent reviewers. When disagreement arose, a decision was reached by consensus and joint review of films. Coronary stenosis was considered physiologically significant if there was more than 50% reduction in luminal diameter.

Exercise studies were only performed postoperatively, using a supine bicycle ergometer at a load of 50 J for about 6 min. After the patients had achieved a steady-state level of exercise for 4 min, the measurement of pressure and cardiac output was performed and blood gas sampling was performed from the pulmonary artery and aorta to examine oxygen saturation. No patient developed angina, significant ST–T change or ventricular arrhythmia.

Measurements and Calculations

Left ventricular volumes were computed by the area–length method and a regression equation. Extrasystolic and postextrasystolic beats were excluded, with the earliest satisfactory beat being selected for analysis. Volumes were indexed for body surface area. All patients had sinus rhythm.

End-diastolic volume (EDV) was defined as the largest volume and ESV as the smallest volume. The EF was determined as (EDV–ESV)/EDV. Minute work (MW, kg·M/min) was determined as 0.0136·mean ejection pressure of LV (mmHg)·stroke volume·heart rate (HR).
Assessment of Left Ventricular Wall Motion

Left ventricular wall motion was quantitatively estimated using 2-dimensional echocardiography. The left ventricular wall was divided into 17 segments, and each segment was graded on a four-point scale: akinesis = 3; severe hypokinesis = 2; hypokinesis = 1; normal = 0. The wall motion score (WMS) was estimated by summing the number of the score of asynergic segments. This procedure was performed at the same time as cardiac catheterization.

Statistics

Values of continuous variables were expressed as mean ± standard deviation. Comparisons of preoperative and postoperative variables in the same patient were made by the nonparametric Wilcoxon method. Comparison between the variables at rest and during exercise in the same patient was also made by the nonparametric Wilcoxon method.

Results

Patients’ profiles are presented in Table 1. After CABG, postinfarction angina, which had been the indication for surgery, had vanished in all patients, with no significant ST–T change detected on the ECG even during exercise.

Fig 1 demonstrates the relationship between preoperative ESVI and the pre- and postoperative EF. We divided our patients into 2 groups according to those results.

Patients With a Preoperative ESVI >100 ml/m²

In these 5 patients the EF did not change from the preoperative value, both being under 30% (before to after: 26±4 to 26±4%). Neither the ESVI (148±50 to 133±39 ml/m²) nor the end-diastolic volume index (EDVI) (198±62 to 180±37 ml/m²) changed (Fig 2).

During exercise after CABG, in spite of the increase in HR (at rest, 81±20 beats/min; during exercise, 111±21 beats/min, p<0.005) and the increase in LV end-diastolic pressure (EDP) (at rest, 22±9 mmHg; during exercise, 35±13 mmHg, p<0.02), there was no increase in either cardiac index (CI: at rest, 2.4±0.3 L/min·m²; during exercise, 2.6±0.4 L/min·m²) or MW (at rest, 4.1±0.4 kg·M/min; during exercise, 4.1±0.4 kg·M/min) (Fig 3). There was no change in WMS even after surgery (33±2 vs 33±3) (Fig 6).

Patients With a Preoperative ESVI <100 ml/m²

In 5 of the 11 patients, the increment of the EF was more than 10% and the EF exceeded 45% (ie improved subgroup) after CABG. In the other 6 patients, the postoperative EF remained under 40% (nonimproved subgroup) (Fig 1).

The Improved Subgroup: EF improved after CABG (before to after; 36±4 to 60±6%, p<0.005). The ESVI reduced after CABG (82±14 to 48±7 ml/m², p<0.02), but the EDVI did not change (130±16 to 120±13 ml/m²) (Fig 4). HR (at rest, 66±12 beats/min; during exercise, 85±8 beats/min, p<0.001), LVEDP (at rest, 14±7 mmHg; during exercise, 22±2 mmHg, p<0.05), CI (at rest, 3.0±0.2 L/min·m²; during exercise, 4.0±1.0 L/min·m², p<0.02), and MW (at rest, 4.8±0.4 kg·M/min; during exercise, 8.9±2.9 kg·M/min, p<0.005) increased during exercise (Fig 5).

The Nonimproved Subgroup: EF did not change before or after surgery, both being under 40% (before to after: 31±7 to 35±3%). Neither the ESVI (78±8 to 74±12 ml/m²) nor the EDVI (115±10 to 115±20 ml/m²) changed (Fig 4). HR (at rest, 88±13 beats/min; during exercise, 108±11 beats/min, p<0.005), LVEDP (at rest, 20±6 mmHg; during exercise, 29±7 mmHg, p<0.01), CI (at rest, 2.5±0.6 L/min·m²; during exercise, 3.3±0.5 L/min·m², p<0.05), and MW (at rest, 4.6±1.0 kg·M/min; during exercise, 6.5±1.5 kg·M/min, p<0.05) increased during exercise.

However, HR both at rest and during exercise in the nonimproved subgroup was greater than that in the improved subgroup (p<0.02, p<0.01, respectively) (Fig 5).
Moreover, the change ratio of MW during exercise/at rest in the nonimproved subgroup was smaller than that in the improved subgroup (1.8±0.5, 1.3±0.1, respectively, p<0.05).

Left Ventricular Wall Motion in the Nonimproved Subgroup and the Improved Subgroup: There were no differences in WMS between both subgroups before CABG (29±6 and 27±3, NS). These values in the nonimproved subgroup did not change even after CABG, while those in the improved subgroup were greater than those in the improved subgroup. LVESVI, left ventricular end-systolic volume index.

NYHA Functional Class

After CABG, one patient with a preoperative ESVI of 165 ml/m² died due to pneumonia. The NYHA functional class remained 2 in all the patients with a preoperative ESVI greater than 100 ml/m² and in 5 of the 6 patients in the nonimproved subgroup with a preoperative ESVI<100 ml/m².

Discussion

Dilatation of the LV after MI consists of expansion in the infarcted region and a secondary volume-overload hypertrophy in the noninfarcted region. The extent of ventricular dilatation after MI is related to the magnitude of the initial damage to the myocardium. Although an increase in cavity size tends to restore stroke volume, despite a persistently depressed EF, left ventricular remodeling following MI can profoundly affect the function of the ventricle and, thereby, the patient’s prognosis.

Recently we have been performing CABG more often for such patients. In the present study, the postinfarction angina, for which we operated, vanished in all patients after CABG, with no significant ST–T change detected on the ECG even during exercise. However, the efficacy of CABG on left ventricular function was limited, especially in those patients with a preoperative ESVI >100 ml/m². Also in those patients neither the ESV, EDV nor the EF changed. Exercise tolerance was poor. Furthermore, left ventricular wall motion did not improve, even after CABG, suggesting that the extent of nonviable myocardium (ie, scar) was large, and had contributed to the severely depressed preoperative left ventricular function. Therefore, we think that an ESVI >100 ml/m² is a useful predictor of poor prognosis.

On the other hand, the patients with a preoperative ESVI ranging from 60 to 100 ml/m² were thought to be on the borderline. We obtained 2 different results in these patients. In the improved subgroup, reversible ischemia and/or myocardial hibernation as well as nonviable myocardium were important causative factors in the severely depressed preoperative LV function. However, in the nonimproved subgroup, there were no changes in ESV, EDV or EF, with left ventricular wall motion not improving even after CABG. These results suggested that the extent of nonviable myocardium in this subgroup was smaller than in the group with an ESVI greater than 100 ml/m², but larger than that in the improved subgroup. Accordingly, the exercise tolerance was poorer than that in the improved subgroup and the postoperative NYHA functional class remained as 2.

We need find the difference between the nonimproved and improved subgroups before CABG in those patients with a preoperative ESVI ranging between 60 and 100 ml/m². Retrospective assessment by means of the WMS showed that the extent of MI in the nonimproved subgroup was larger than that in the improved subgroup.
The most important clinical event was the revascularization of the infarcted segments restoring myocardial viability. If impaired left ventricular function may be attributed to actively ischemic or hibernating myocardium in the noninfarcted segments, augmentation of coronary blood flow to viable but ischemic or underperfused myocardium would be expected to improve left ventricular function. It has been well established that exercise-induced LV dysfunction, reflecting reversible ischemia, usually improves after successful CABG.12–14 Moreover, the asynergic regions represent viable myocardium that is functionally impaired by recent intense ischemia (stunned myocardium), ongoing subclinical ischemia or myocardial hibernation.15,16 If such dysfunction involves a large portion of the LV, then resting global LV function may be depressed but should improve after revascularization17,18 as in our improved subgroup. In addition, Dilsizian et al reported that LV global and regional function at rest improved after successful CABG, even in patients with a normal global LV ejection fraction and no visually detectable wall motion abnormality before surgery.19 We evaluated myocardial viability using 201Tl scintigraphy and myocardial contrast echocardiography in all the patients, and dobutamine stress echocardiography in the patient with highly dilated LV, and performed revascularization. However, the infarcted segments consisted of various ratios of viable versus infarcted myocardium. We could not assess whether postoperative cardiac function improved or not using the preceding methods. This led us to consider that the preoperative LVESVI is the best index of the improvement of LV function after CABG. We further consider that the preoperative ESVI indicates the extent of reserved viable myocardium.

In previous studies, it has been unclear whether resting global LV function after CABG improves or not in patients with severely depressed ventricular function.7–10 Milano et al reported that survivors with severely depressed ventricular function2 showed significant improvement in angina symptoms, which would probably reduce future hospitalization and improve the quality of life for these patients.11 Although the LV function was not stated, survivors also showed significant improvement in the class of congestive heart failure.

As regards the value of an ESVI of 100 ml/m², Yamaguchi et al also reported the same predictive value.20 However, their data might lead to an underestimation of functional recovery because their postoperative study was performed on an average of only 28 days after CABG. The postoperative EF in 5 of their 10 patients with an ESVI >100 ml/m² was lower than the preoperative EF, which might suggest not enough recovery from recent intense ischemia (stunned myocardium), due to difficult myocardial recovery. Further, they found that the ESVI of 100 ml/m² was not predictive of functional recovery in patients with severely depressed ventricular function.20

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dial protection for those patients, or myocardial hibernation. Our studies were performed 7.2±2.3 months postoperatively to avoid such an underestimation of functional recovery. White et al have suggested that ESV is the primary predictor of survival after MI and is superior to EF when the EF is low or when ESV is high. The value of ESV is 100 ml/m². We believe that this value includes not only the patients with an ESVI >100 ml/m² but also the patients in the nonimproved subgroup in our study with an ESVI between 60 and 100 ml/m².

**Limitations**

The duration and intensity of the exercise must be tailored to fit the needs of the individual patient. The duration of exercise in the present study was determined by Lorell and Grossman’s method. Oxygen saturation of the pulmonary artery (SvO₂) was significantly reduced during exercise in all the patients (at rest, 67.1±4.0% (range, 57.7–74.0%); during exercise, 36.5±6.4% (range, 47.6%), p<0.001). We think that appropriate intensity levels were obtained.

Moreover, we withheld all medication for at least 12–18 h before catheterization. Accordingly, the effect of beta-blockers and calcium antagonists, which may reduce ventricular function, was avoided. Digoxin, which might have played a role in modifying LV performance, was used in 11 patients (63%), including 4 patients (80%) in the group with a preoperative ESVI >100 ml/m² and 4 patients (67%) in the nonimproved subgroup with a preoperative ESVI <100 ml/m². Their LV function remained low in spite of the use of digoxin.

The clinical implications should be considered. Because the extent of ventricular dilatation after MI is related to the magnitude of initial damage to the myocardium, our results suggest that the initial therapy for acute MI is very important.

**Conclusions**

Our data suggested that CABG in patients with a dilated LV was effective against anginal symptoms, but was restricted to left ventricular function. It may be possible to estimate postoperative left ventricular function, including exercise tolerance, from the preoperative ESVI.

**References**