Videometric Analysis of Regional Left Ventricular Function before and after Aortocoronary Artery Bypass Surgery

CORRELATION OF PEAK RATE OF MYOCARDIAL WALL THICKENING WITH LATE POSTOPERATIVE GRAFT FLOWS

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ABSTRACT The peak rate of systolic wall thickening ($\bar{p}dT_w/dt$) in regions of the left ventricle was determined by biplane roentgen videometry in 60 patients before and a median of 14 mo after aortocoronary bypass graft surgery. The left ventricular ejection fraction, stroke volume, and end-diastolic volume and pressure did not change significantly after surgery in the presence of patent or occluded grafts ($P > 0.05$).

Statistically significant increases occurred in the peak rate of systolic wall thickening of regions supplied by patent bypass grafts, and significant decreases occurred in regions with occluded grafts ($P < 0.01$). Of 42 preoperatively hypokinetic regions ($\bar{p}dT_w/dt > 0$ < 5.0 cm/s) supplied by a patent graft, 30 improved by an average of 2.6 cm/s after operation; 18 returned to normal. Failure of 24 hypokinetic regions to improve to normal was associated with myocardial infarction in 11 or with late postoperative graft blood flows of <60 ml/min, measured by videodensitometry, in 10. All seven preoperatively akinetic ($\bar{p}dT_w/dt = 0$) or dyskinetic ($\bar{p}dT_w/dt < 0$) regions did not improve after the operation despite the fact that, in five of the seven, coronary bypass flows were over 60 ml/min.

All eight preoperatively hypokinetic regions supplied by coronary artery graft flows of ≤40 ml/min failed to improve to normal after operation. All nine preoperatively hypokinetic regions supplied by coronary artery graft flows of over 60 ml/min improved to normal after surgery. Late postoperative coronary

artery bypass graft flows, the functional status of the myocardium, the status and distribution of the native coronary circulation, and decreased regional function elsewhere in the ventricle must all be considered when regional left ventricular function is interpreted.

INTRODUCTION

Aortocoronary artery bypass graft surgery affords relief or improvement of angina pectoris in the majority of patients (1–5). Evidence for improvement of myocardial function has been contradictory (1, 6–14), especially in late postoperative studies. Decreased regional left ventricular (LV)[1] function in segments supplied by patent grafts has been reported (1). However, demonstration of graft patency alone does not necessarily indicate sufficient blood flow to relieve myocardial ischemia. To date, flow in individual aortocoronary artery bypass grafts and its relationship to regional function have been measured only at the time of surgery with an electromagnetic flow meter (15). In that study (15), mean flow through the graft correlated with the effect of its temporary occlusion on the percentage decrease in myocardial contractile force, measured by a strain-gauge arch. Recently developed, quantitative, computer-based videoangiographic methods of analysis allow postoperation measurement of blood flow through individual grafts (16–18) and 60 fields determination of LV volumes and

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[1] *Abbreviations used in this paper:* EJF, ejection fraction; LV, left ventricular or ventricle; $\bar{p}dT_w/dt$, peak rate of systolic wall thickening.

regional myocardial wall dynamics (19, 20). Not sur-prisingly, aortocoronary artery bypass graft flow measured during operation by electromagnetic flow-meter has correlated poorly with videodensitometric flows measured at cardiac catheterization and angiog-raphy soon after the operation (21). It is therefore essential to assess both regional LV function and graft flows at the same postoperative study. The present study was designed to evaluate LV function before and after aortocoronary artery bypass graft surgery and, in particular, the relationship between blood flow in a graft and the peak rate of systolic wall thickening in the region supplied by the graft.

METHODS

600 patients underwent aortocoronary artery bypass graft surgery at the Mayo Clinic between April 1972 and December 1974 for angina pectoris due to coronary artery disease. 60 patients (56 males), ranging in age from 34 to 67 yr (median, 54 yr), were selected for study. Preoperative angina pectoris was considered to be stable in 58 patients. Before operation, all patients were in functional class III or IV (New York Heart Association classification) on the basis of angina pectoris. Transmural myocardial infarction by definite electro-cardiographic and vectorcardiographic criteria (22) was present before operation in 21 patients; 2 additional patients were classified as having a subendocardial infarction before surgery (chest pain, persistent ST-T wave changes for 48 h, elevated enzymes). The heart size on chest roentgenograms was normal except for two patients who had mild cardiomegaly (cardiothoracic ratio 0.55).

Selection of these 60 patients was based on the following criteria: (a) There was no evidence of valvular heart disease. (b) There was no clinical evidence of heart failure. (c) Aorto-coronary artery bypass grafting was the only surgical pro-cedure; for example, patients with resection of any part of the left ventricle were excluded. (d) Left ventriculograms before and after surgery were technically suitable for videometric analysis (19, 20). (e) No medication known to influence LV function was being taken. (f) In the last year of the study, patients in whom the preoperative left ventriculogram demonstrated a regional abnormality were selected for restudy. (g) Informed consent was required. Patients were given the opportunity to refuse the study after full explanation of the risks and objectives.

The patients were restudied 3–36 mo (median 14 mo) after aortocoronary artery bypass surgery. 15 of the 60 patients returned because of chest pain. Repeat angiography was considered clinically indicated. The remaining 45 patients had been recalled without regard to symptoms as potential candidates for restudy because of satisfactory analysis of the preoperative left ventriculogram. Of these 45 patients, 17 had angina and 24 had an abnormal treadmill electrocardiogram at the time of postoperative evaluation. The study population is biased with patients having less favorable results because many of the patients who were asymptomatic and had negative treadmill electrocardiograms refused repeat angiog-raphy when recalled.

Seven men, 36–64 yr of age (median 53 yr), underwent coronary arteriography and left ventriculography for definition of the location and severity of coronary disease without intervening surgery at intervals of 10–29 mo (median 15 mo). These patients provide a control group with a comparison of the same measurements of global and regional left ventricular function in sequential studies. All had stable angina pectoris

(New York Heart Association class II or III) at the time of both studies. Five patients had had transmural myocardial infarc-tion before the initial study; none had a myocardial infarction between the first and second studies.

Cardiac catheterization was performed in the fasting patient after premedication with 100 mg of pentobarbital given intramuscularly. No patient received nitrates within the 8 h preceding the study. The study sequence at the time of catheterization was as follows: (a) left ventricular pressure determination, (b) biplane left ventriculography, (c) graft transit times, (d) spot films for graft dimensions, (e) sublingual isosorbide dinitrate (Isordil, Ives Laboratories, Inc., New York) (5 mg), and (f) selective graft and coronary cineangiog-raphy. Simultaneous biplane left ventricular videoangiograms were made in orthogonal projections (45° right anterior oblique and 45° left anterior oblique projections). Meglumine diatrizoate (Renografin-76, 0.7 ml/kg, E. R. Squibb & Sons, Princeton, N.J.) was injected over 3–4 s during deep inspira-tion. The preoperative ventriculogram was always reviewed before the postoperative study to place the patient in the same position for both studies. Ventriculography was never performed during or immediately after an episode of angina. In two patients, the left ventricle was opacified by injection of contrast medium into the main pulmonary artery.

Videodensitometric measurements of the mean transit times in the vein grafts were obtained by the selective injection of 1–3 ml of Renografin at the orifice of each graft. The resulting fluoroscopic image was recorded on videotape. Graft transit times were determined 5–10 min after ventriculography by measuring changes in roentgen density caused by the contrast medium indicator (17, 18) and recording computer-generated indicator-dilution curves at the proximal and distal ends of the graft. Nonspecific changes in roentgen density caused by cardiogenic motion of the catheter or vessel and changes in cardiac size and position were compensated for by background subtraction of the cyclic roentgen density changes of the last two or three cardiac cycles recorded before injection of contrast medium. The mean transit time of the bolus of con-trast medium along the length of the graft was determined as the difference between the mean transit times measured at the proximal and distal sampling sites. The volume of the graft between the two sampling sites was determined from biplane orthogonal roentgenograms of the grafts obtained after selective injections of 5–10 ml of Renografin-76 into the graft orifices (17). Graft blood flow is equal to graft volume divided by mean transit time of the bolus along the graft (17, 18). Unsatisfactory measurements were due primarily to poor patient positioning and inadequate breath holding or tech-nically unsatisfactory spot film exposure for determination of graft dimensions.

Graft flows determined from two consecutive Renografin injections were performed 5–10 min apart at the same patient study and were very similar: an increase in half and a decrease in half of the second flow measurements (16 grafts), a median change of 0.5 ml/min, and a change >11 ml/min in only 2 of 16 grafts. This is in agreement with studies in man (23), wherein the hyperemic response to 3 ml of Renografin lasted 1 min, and previous studies in dogs, wherein the hyperemic response to larger-per-kilogram doses of contrast medium lasted 1–4 min (24).

Ventricular volumes (end-diastolic and end-systolic volume indexes), together with the calculated stroke volume and ejection fraction (EJF), were determined from the biplane ventriculograms by roentgen videometry. The operator-inter-active video system interfaced with a CDC 3500 computer (Control Data Corp., Minneapolis) has been previously described (19). The volume-computing program was based on Simpson's rule (25). Only regular beats were analyzed. In eight patients, biplane determinations of LV volume were not

TABLE I
Grouping and Location of Segments Analyzed (Grafted and Ungrafted) in Paired
(Preoperative and Postoperative) Left Ventriculograms

	Segments										
	A	2A	3A	L	2L	A,L	2A,L	A,2L	I,L,2A	I,A,2L	I,A,L
No. of patients (57 total)	16	5	1	5	2	16	6	3	1	1	1
No. of paired segments (108 total)	16	10	3	5	4	32	18	9	4	4	3

Regional left ventricular function was analyzed in as many segments as technically possible in each patient. In 57 patients an average of 1.9 paired segments per patient were analyzable; in 3 patients poor epicardial definition prevented analysis. A, anterior segment (high, mid, or low); L, posterolateral segment (high, mid, or low); I, inferior segment. Number preceding letter designates number of segments in each location.

possible either before or after operation because of ectopic beats or X-ray exposure unsuitable for analysis.

The same videometric, computer-interfaced system was used to determine and display 60 fields measurements of wall thickness throughout several cardiac cycles and to calculate and display the peak rate of systolic wall thickening as previously described (20). Although the thickness of the wall was computed as the shortest distance between the endocardium and the epicardium, even with accurate definition of these borders, the shortest distance is not necessarily the true wall thickness of the segment (26). Nonetheless, for convenience, the measured value will be referred to as "wall thickness."

Up to three segments in each of the anterior and posterolateral walls are analyzable, depending on the magnification and orientation of the cardiac silhouette and the definition of the epicardium (Table I). Because of previously noted (20) difficulties in obtaining optimal orientation of inferoseptal segments and because of the superimposed diaphragm, only three patients had a single inferoseptal segment that was analyzable before and after surgery.

Segments were classified quantitatively as "normal" when the peak rate of wall thickening ($\bar{p}dT_w/dt$) was greater than or equal to 5.0 cm/s, "hypokinetic" when the $\bar{p}dT_w/dt$ was less than 5.0 cm/s, "akinetic" when the $\bar{p}dT_w/dt$ was equal to 0, and "dyskinetic" when the $\bar{p}dT_w/dt$ was less than 0 because of systolic wall thinning (20).

Heart rate was measured from the simultaneous electrocardiogram recorded while the ventriculogram was being videotaped. Aortic diameters were measured at end-systole on all ventriculograms with the same videometric system to verify that appropriate magnification factors had been used for studies both before and after operation.

Coronary cinearteriograms before and after operation were compared in multiple views by at least two observers (Drs. Frye, Rutherford, and Davis) to determine whether there were any changes in the native coronary circulation. The reported percent stenosis of a coronary artery is the visually estimated maximum narrowing in arterial diameter.

The operative techniques utilized in all patients were mild to moderate hypothermia (15–35°C); venting via the left atrium (27 patients), pulmonary veins (10 patients), or left ventricular apex (23 patients); and blood flow on cardiopulmonary bypass of 2.0–2.4 liters/min per m². Ventricular fibrillation was not established deliberately. The aorta was clamped for 10–20 min/graft. A reversed segment of autogenous saphenous vein was used for all grafts. In the entire study population, 21 patients had single grafts, 27 had double grafts, 10 had triple grafts, and 2 had four grafts.

The patients were divided into two groups according to the

patency of the aortocoronary artery bypass grafts for comparison of indices of global LV function before and after operation.

Validation of methods. The reliability, accuracy, and reproducibility of the videometric techniques (27) and computer analysis of wall thickness determination by the same observer (20) have been reported. The reproducibility of $\bar{p}dT_w/dt$ by the same observer was within 0.6 cm/s in 13 segments supplied by normal or diseased coronary arteries at two different studies (a median of 14 mo apart) in six unoperated patients with no significant change in the coronary angiogram (Fig. 1). The comparison of the EJF in six unoperated patients with coronary artery disease at two different studies (a median of 14 mo apart) was within a range of ±11% (EJF units).

An excellent correlation (r = 0.95) has been demonstrated between graft flow values less than 150 ml/min determined by simultaneous videodensitometric and electromagnetic flowmeter measurements (17, 18).

RESULTS

Clinical. The left anterior descending artery was bypassed in 53 patients (16 grafts were occluded); the left circumflex artery was bypassed in 24 patients (7
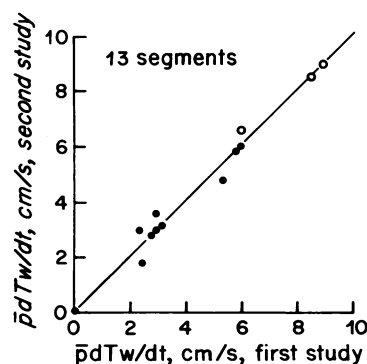

FIGURE 1 Reproducibility of peak rate of systolic wall thickening in two studies (median, 14 mo apart) of six unoperated control patients with no significant change in coronary angiogram was within 0.6 cm/s in 13 segments. ○, segments supplied by normal or <50% stenosed coronary arteries; ●, segments supplied by ≥50% stenosed coronary arteries.
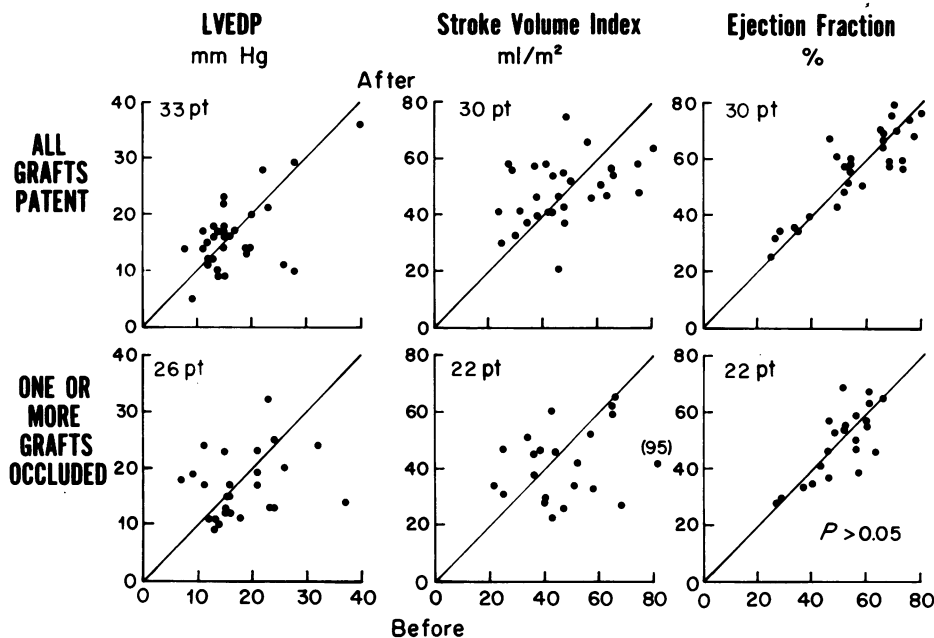
FIGURE 2 Indices of global LV functional status (left ventricular end-diastolic pressure [LVEDP], stroke volume index, and ejection fraction) before operation (top) and late after operation (bottom) are shown for a group of patients with all grafts patent and a group with one or more grafts occluded. No statistically significant changes ($P > 0.05$) occurred after operation compared with before operation. Note, however, that only about 30% of patients had abnormal values before operation. Pt, number of patients.
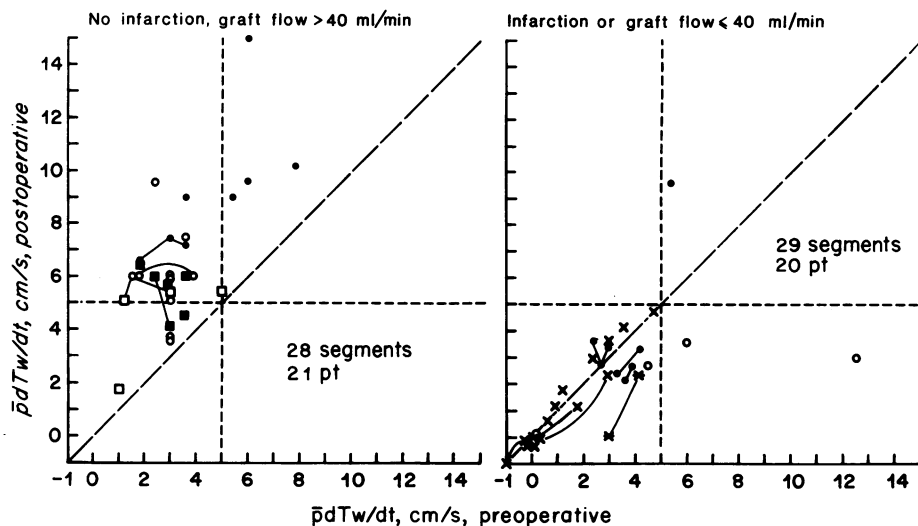

FIGURE 3 Peak rate of systolic wall thickening in segments supplied by patent grafts, a comparison of $\bar{p}dT_w/dt$ before operation and a median of 14 mo after operation. Left panel: segments supplied by graft flows of >40 ml/min (circles) and segments to which graft flows were not technically feasible (squares). Right panel: segments in regions of infarction (before or during operation) or segments without infarction supplied by grafts with blood flows of ≤40 ml/min. Note that the coordinates start at (−1, −1) and that in the right panel there are six akinetic segments and one dyskinetic segment, none of which changed after surgery. Changes observed after operation were statistically significant ($P < 0.01$). ○, □, segments associated with no decrease in regional function elsewhere in LV. ●, ■, segments associated with decreased regional function elsewhere in LV (graft occlusion or decreased postoperative regional wall dynamics). ×, preoperative infarction. *, Intraoperative infarction. Lines connect segments from same patient at same studies. Pt, number of patients.

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grafts were occluded); the right coronary artery was bypassed in 31 patients (11 grafts were occluded); and the diagonal branch was bypassed in 5 patients (2 grafts were occluded). This high graft-occlusion rate may reflect a bias, in the greater likelihood of patients to consent to restudy if they had postoperative angina or an abnormal treadmill exercise electrocardiogram.

At the time of the postoperative study, 33 patients were free of angina, 22 had class II (New York Heart Association) angina, and 5 had class III angina.

Three patients had an early postoperative (within 30 days) transmural myocardial infarction by definite electrocardiographic and vectorcardiographic criteria (22); two infarcts were in the region of an occluded graft, and one was in the region of a patent graft. Two patients had a late postoperative (>30 days) myocardial infarction in the region of an occluded graft.

Patent graft group. In 33 patients, all 54 grafts were patent. Only six patients in this group were completely revascularized; that is, they had no other coronary arteries or major branches with ≥50% stenosis that were not grafted. Five patients had coronary arterial narrowing of ≥50% which was not bypassed in a coronary branch artery (diagonal branch, an obtuse marginal branch, or a nondominant left circumflex atrioventricular groove branch). The remaining 22 patients in this group had at least one major coronary artery with ≥50% stenosis that was not graftable. 12 of the 33 patients had angina at the time of the post-operative study.

Occluded graft group. In these 27 patients, 36 of 59 grafts were occluded; 9 patients had all grafts occluded. 15 of the 27 patients had angina at the time of the postoperative study.

Total ventricular functional status. Compared with measurements before operation, the groups with patent and occluded grafts showed no statistically significant ($P > 0.05$) changes after operation in resting left ventricular end-diastolic pressure, stroke volume, EJF, or end-diastolic or end-systolic volume index (Fig. 2). No statistically significant ($P > 0.05$) post-operative changes in these same parameters occurred in patients with abnormal preoperative values, with all grafts occluded, or with an occluded graft of the left anterior descending coronary artery.

Regional ventricular functional status. In contrast to the lack of statistically significant changes in global LV functional status, significant ($P < 0.01$) increases occurred in $\bar{p}dT_w/dt$ of segments supplied by patent grafts (Fig. 3) and significant ($P < 0.01$) decreases occurred in segments with occluded grafts (Fig. 4).

Segments supplied by patent grafts (57 segments). Preoperatively, 42 were hypokinetic segments (Fig. 3, $\bar{p}dT_w/dt > 0 < 5$ cm/s on the abscissa), 7 were akinetic or dyskinetic, and 8 were normal. Of the 42 preoperatively hypokinetic segments, 30 improved subsequent
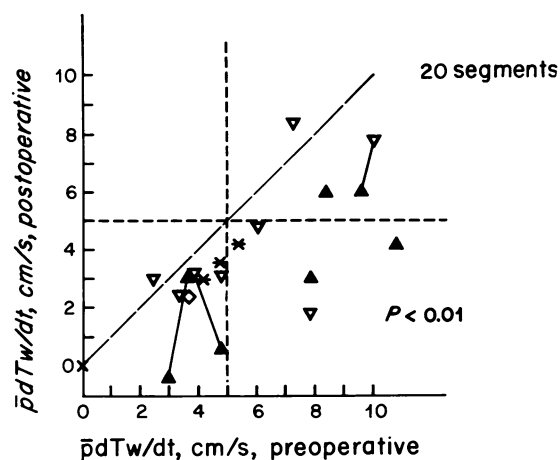

FIGURE 4 Peak rate of systolic wall thickening before and after operation in 20 segments (from 17 patients) supplied by occluded grafts; a statistically significant decrease in $\bar{p}dT_w/dt$ occurred after operation ($P < 0.01$). ▽, no change in native coronary circulation supplying this segment. ▲, total occlusion of part or all of native coronary artery supplying this segment. ◊, anterior wall segment with native left anterior descending coronary artery occluded, diagonal branch unchanged. ×, preoperative infarction. *, postoperative infarction. Lines connect segments from same patient at same studies.

to operation (average increase 2.6 cm/s); 18 of the 30 improved to normal (>5 cm/s on the ordinate). Failure of the remaining 24 hypokinetic segments to improve to normal after operation was associated with infarction before or after surgery (11 segments) or a graft blood flow of <60 ml/min (10 segments) in 21 of 24 segments; in the 3 other segments, associated graft blood flow measurements were not technically satisfactory.

All seven preoperative akinetic or dyskinetic regions did not change after operation (Fig. 3, right), despite graft blood flows of >60 ml/min to five of the seven regions where flow measurements were technically satisfactory. Six of these seven regions had documented preoperative transmural myocardial infarction.

Of eight preoperatively normal segments, six remained normal after surgery; and two were hypokinetic at the late postoperative study in association with graft flow of <40 ml/min (see *Regional ventricular functional status versus graft flow*).

Segments supplied by occluded grafts (20 segments). Inadequate blood supply results in decreased $\bar{p}dT_w/dt$, as documented by the analysis of segments supplied by occluded grafts (Fig. 4); 17 of these 20 segments showed a decrease. The largest decreases in segmental function occurred when both the native coronary circulation and the graft were occluded (Fig. 4, solid triangles). Smaller decreases occurred in segments in which the grafts were occluded but the native circulation was unchanged (Fig. 4, open triangles). In the
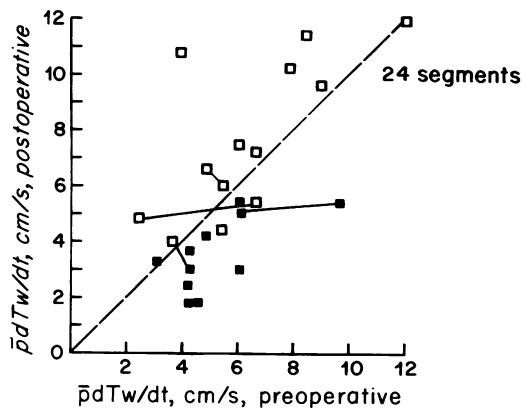
FIGURE 5 Peak rate of systolic wall thickening before and after operation in 24 segments (20 patients) supplied by ungrafted coronary arteries. Statistically significant increases occurred in segments supplied by normal or <50% stenosed ungrafted coronary arteries (□) and significant decreases occurred in segments supplied by ungrafted arteries with ≧50% stenosis (■), ($P < 0.01$). Lines connect segments from same patient at same studies.

one region supplied by new collateral circulation, $\bar{p}dT_w/dt$ increased 1.2 cm/s.

Segments supplied by ungrafted arteries (25 segments). In 13 segments supplied by ungrafted arteries with <50% stenosis, $\bar{p}dT_w/dt$ increased an average of 1.3 cm/s after surgery. In 12 segments supplied by ungrafted arteries with ≧50% stenosis, $\bar{p}dT_w/dt$ decreased an average of 1.6 cm/s after surgery (Fig. 5).

Segments supplied by ungrafted arteries with <50% stenosis appeared capable of a compensatory increase in regional wall dynamics subsequent to operation if there was deterioration of circulation or wall motion elsewhere in the LV. However, ungrafted coronary arteries with ≧50% stenosis appeared to preclude a compensatory increase in wall dynamics of the region they supplied (Fig. 6).

Regional ventricular functional status versus graft flow. Independent measurement of $\bar{p}dT_w/dt$ and graft flows were performed by different observers. Regional wall dynamics before and after operation were plotted for segments supplied by three levels of graft flow: ≦40 ml/min, 41–60 ml/min, and >60 ml/min (Fig. 7). Two of three preoperatively normal segments with graft flows of <40 ml/min were hypokinetic at the late postoperative study without evidence of myocardial infarction. These two segments had normal $\bar{p}dT_w/dt$ at catheterization study 7 and 8 days after surgery when graft flows were 120 and 73 ml/min, respectively; however, they were hypokinetic 16 and 11 mo later when flows were 39 and 32 ml/min (33% and 44% of the early postoperative flows), respectively (Fig. 7). All eight preoperatively hypokinetic segments failed to improve to normal after operation with graft flows of ≦40 ml/min. With graft flows of 41–60 ml/min, three of five

hypokinetic segments improved to normal after operation (Fig. 7). With graft flows of >60 ml/min, all nine hypokinetic segments improved to normal after operation and increases in $\bar{p}dT_w/dt$ were the largest (Fig. 7).

There was no significant ($P > 0.05$) change in arterial pressure between the two studies. The heart rate was slightly increased (4 beats/min average) at the time of postoperative study ($P < 0.01$) and was comparable in the patent and occluded graft groups.

DISCUSSION

The present study demonstrates an improvement in hypokinetic segments of the left ventricle in patients after aortocoronary artery bypass graft surgery for relief of angina pectoris. The amount of graft flow and the functional status of the regional myocardium are important modifiers of the response observed. The observed improvement in regional myocardial function was unaccompanied by significant changes in global LV function.

Regional function of the left ventricle late after aortocoronary artery bypass graft surgery has been reported as unchanged or deteriorated (1, 2, 14) or improved (28, 29). The reasons for these reported differences are not entirely clear but may relate in part to the following factors: (a) different methods of regional LV function measurement, (b) lack of graft blood flow measurement at the time of postoperative study, (c) imprecise correlation of coronary artery distribution with specific myocardial segments and incomplete revascularization of major arterial branches, (d) functional status of the myocardium before and after operation, and (e) variable surgical technique and experience.
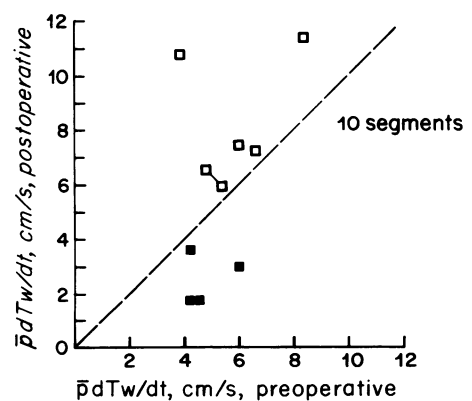

FIGURE 6 Peak rate of systolic wall thickening before and after operation in 10 segments (9 patients) supplied by ungrafted coronary arteries in patients with occluded or stenosed grafts in other regions of LV ($P < 0.01$). □, segments supplied by normal or <50% stenosed ungrafted coronary arteries. ■, segments supplied by ungrafted coronary arteries with ≧50% stenosis. Line connects segments from same patient at same studies.
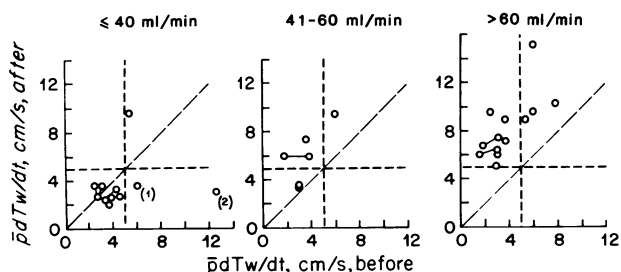
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FIGURE 7 Dependence of peak rate of systolic wall thickening on late postoperative graft flow. p̄dT_w/dt before and after operation are shown for three levels of graft flow: ≦40 ml/min, 41–60 ml/min, and >60 ml/min. Two segments, (1) and (2), with normal rates of wall thickening before surgery and 7–8 days after operation, when graft flows were 120 and 73 mg/min, respectively, were noted to be hypokinetic 16 and 11 mo later when reduced flows of 39 and 32 ml/min, respectively, were noted. Lines connect segments from same patient at same studies.

Regional wall dynamics are most often determined by assessing endocardial motion from superimposition of end-diastolic and end-systolic LV cineangiographic silhouettes and measuring changes in minor axes of the ventricle. This method is sensitive to changes in position of the heart during the cardiac cycle. The assessment of wall motion has been demonstrated to differ significantly according to the method of superimposing the silhouettes (30). The relative sensitivity of these methods in sequential studies and whether these methods contributed significantly to the differences noted in sequential studies in the same patient are not known. We have applied computer-assisted roentgen videometry to the analysis of biplane left ventriculograms to quantitate regional wall thickening during systole. With this method, which is less sensitive to changes in cardiac position and adjacent myocardial motion, it was found that the peak rate of systolic wall thickening best separated apparently normal and abnormal segments of the left ventricle (20).

All angiographic measurements of regional LV function, including wall thickness, are limited in accuracy by the inherently ambiguous relationship between a three-dimensional structure and its roentgen projection image (26). The accuracy of the relationship between projected and true wall thickness is greatest when the wall is thin, has a smooth surface, and has a radius of curvature that is large in comparison with the wall thickness. In addition, the trabeculae carneae and papillary muscles may squeeze together during systolic contraction so that the contrast medium is extruded from between these structures, apparently thickening the wall. Both of these mechanisms cause the wall thickness measurement to be most accurate in the diastolic, poorly contracting, and distended ventricular wall. The wall thickness tends to be increasingly overestimated with the vigor of contraction.

Therefore, it is most likely that the large increases in computed wall thickness noted in some normal regions represent an overestimate. This effect is least likely in the hypokinetic regions. Fortuitously, the rate of wall thickening in the hypokinetic regions, where an increased rate of thickening is of particular interest, is most likely to be the most accurate measurement of true wall thickening. High rates of computed wall thickening, although likely to be an overestimate of the true rate, occur in vigorously contracting and normally functioning myocardium.

The use of dynamic measurements of wall thickness as an index of regional myocardial ischemia is supported by De Jong and Goldstein's study of open-chest pigs (31). A negative correlation was demonstrated between coronary venous inosine concentration (a metabolic index of myocardial ischemia) and myocardial systolic wall thickening during experimentally induced myocardial ischemia.

Factors other than myocardial ischemia that might influence the rate of systolic wall thickening have been considered in the present study. Arterial pressure did not significantly change at studies before and after surgery. Even though heart rate was slightly increased in both the patent and occluded graft groups, the postoperative changes in rates of wall thickening correlated with graft patency (Fig. 8). On the basis of our preliminary unpublished studies in dogs, the slight heart rate increase after surgery appears insufficient to account for the differences in rates of wall thickening. The consistent p̄dT_w/dt noted in our control patients (Fig. 1) makes it unlikely that the differences noted in the patients after operation result from other factors not considered in this study.
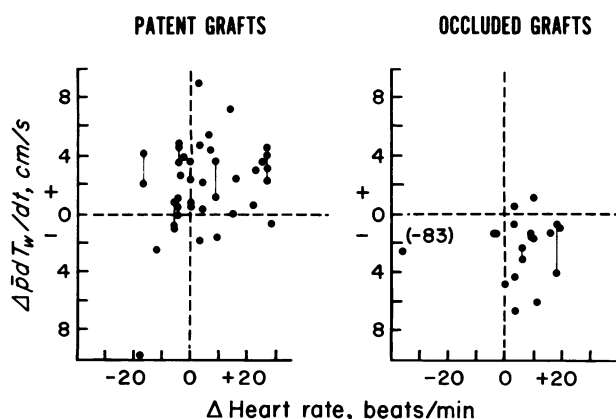

FIGURE 8 Changes in peak rate of systolic wall thickening (postoperative minus preoperative) vs. change in heart rate (postoperative minus preoperative) in segments without infarction supplied by patent grafts (40 segments, 28 patients) (left panel) and segments without preoperative infarction supplied by occluded grafts (19 segments, 16 patients) (right panel). Lines connect segments from same patient at same studies.

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A more significant potential influence on the measured postoperative rate of wall thickening is the effect of changed dynamics elsewhere in the ventricle as a result of infarction, graft occlusion, or progression of coronary artery disease. Clinical studies in our laboratory have documented unusually high rates of thickening in normal segments, associated with abnormalities of regional function elsewhere in the same ventricle, suggesting a compensatory response (20). Studies of experimental canine myocardial infarction have also demonstrated a compensatory increase in regional myocardial function in the uninvolved myocardium (32, 33). Thus, improved regional blood supply may not be the only reason for the higher rates of regional wall thickening in the patent graft group. For these reasons, the segments supplied by a patent graft were further classified into segments with or without reduction in circulation or wall motion elsewhere in the ventricle (Fig. 3).

Our studies indicate that graft flows at the time of late postoperative study may be sufficiently diminished to preclude normal regional function, even though the graft is patent. By measuring the blood flow in individual grafts supplying the segment of myocardium under study, a positive correlation was noted between the amount of flow provided by the graft and the postoperative increase in the peak rate of systolic thickening in regions with preoperative hypokinesis (Fig. 7). It is apparent that conclusions on effects of aortocoronary artery bypass graft surgery on regional left ventricular function should ideally include quantitation of flow to the region under study.

Revascularization of a region with an ungrafted >50% stenosis of a branch of the major grafted artery also may preclude improvement in postoperative regional LV function. For example, a patent graft to a left anterior descending coronary artery with residual disease in a large ungrafted diagonal branch may account for lack of improvement in regional wall thickening noted in five of our patients. Also related is the lack of absolute precision in locating the specific region supplied by a single coronary branch artery by subjectively relating a small number of X-ray projections (26).

The status of the myocardium in the region of the left ventricle supplied by the graft must also be considered in assessing results of surgery on regional function. Half of grafted LV regions with new postoperative electrocardiographic evidence of transmural infarction are supplied by patent grafts (34). It is possible that perioperative damage to the myocardium may explain the lack of improvement or deterioration of segments supplied by patent grafts. An example is one of our patients who sustained a perioperative anterior transmural myocardial infarction. 28 mo after operation, a patent left anterior descending coronary artery graft with 82 ml/min blood flow was found in association with deterioration in anterior regional wall dynamics (Fig. 3). Increasing experience with the operation appears to reduce the frequency of postoperative myocardial infarction, and reports on regional myocardial function based on early surgical experience may thus bias postoperative results (34).

Regional wall motion abnormalities with normal indices of global LV function may occur in coronary artery disease. It appears that normal regions compensate for regions of decreased contractility to maintain overall performance of the LV. However, improved regional function after operation did not always result in measurable improvement in global LV function, at least with the indices employed. This may be related to the small size of the regions studied in relation to the rest of the LV. Most indices of global LV function in our patients were normal before surgery and would not be expected to improve; however, even patients with abnormal indices of global LV function did not improve significantly after surgery. This may be related in part to the large number of patients with preoperative infarction and to incomplete revascularization in these subsets.

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REFERENCES

1. Shepherd, R. L., S. B. Itscoitz, D. L. Glancy, E. B. Stinson, R. L. Reis, G. N. Olinger, C. E. Clark, and S. E. Epstein. 1974. Deterioration of myocardial function following aorto-coronary bypass operation. *Circulation.* **49:** 467–475.
2. Griffith, L. S. C., S. C. Achuff, C. R. Conti, J. O. Humphries, R. K. Brawley, V. L. Gott, and R. S. Ross. 1973. Changes in intrinsic coronary circulation and segmental ventricular motion after saphenous-vein coronary bypass graft surgery. *N. Engl. J. Med.* **288:** 589–595.
3. Spencer, F. C., G. E. Green, D. A. Tice, and E. Glassman. 1971. Bypass grafting for occlusive disease of the coronary arteries: a report of experience with 195 patients. *Ann. Surg.* **173:** 1029–1044.

4. Lapin, E. S., J. A. Murray, R. A. Bruce, and L. Winterscheid. 1973. Changes in maximal exercise performance in the evaluation of saphenous vein bypass surgery. *Circulation.* 47: 1164–1173.

5. Bartel, A. G., V. S. Behar, R. H. Peter, E. S. Orgain, and Y. Kong. 1973. Exercise stress testing in evaluation of aortocoronary bypass surgery: report of 123 patients. *Circulation.* 48: 141–148.

6. Rutherford, B. D., G. T. Gau, G. K. Danielson, J. R. Pluth, G. D. Davis, R. B. Wallace, and R. L. Frye. 1972. Left ventricular haemodynamics before and soon after saphenous vein bypass graft operation for angina pectoris. *Br. Heart J.* 34: 1156–1162.

7. Danielson, G. K., G. T. Gau, and G. D. Davis. 1973. Early results of vein bypass grafts for coronary artery disease. *Mayo Clin. Proc.* 48: 487–491.

8. Rees, G., J. D. Bristow, E. L. Kremkau, G. S. Green, R. H. Herr, H. E. Griswold, and A. Starr. 1971. Influence of aortocoronary bypass surgery on left ventricular performance. *N. Engl. J. Med.* 284: 1116–1120.

9. Chatterjee, K., H. J. C. Swan, W. W. Parmley, H. Sustaita, H. S. Marcus, and J. Matloff. 1973. Influence of direct myocardial revascularization on left ventricular asynergy and function in patients with coronary heart disease: with and without previous myocardial infarction. *Circulation.* 47: 276–286.

10. Deal, P., L. P. Elliott, T. D. Bartley, M. W. Wheat, Jr., and H. W. Ramsey. 1973. Quantitative left ventriculography in the immediate postoperative period after aorto-coronary bypass. *J. Thorac. Cardiovasc. Surg.* 66: 1–11.

11. Arbogast, R., A. Solignac, and M. G. Bourassa. 1973. Influence of aortocoronary saphenous vein bypass surgery on left ventricular volumes and ejection fraction. Comparison before and one year after surgery in 51 patients. *Am. J. Med.* 54: 290–296.

12. Bourassa, M. G. 1973. Left ventricular performance following direct myocardial revascularization. *Circulation.* 48: 915–916.

13. Corday, E. 1972. Myocardial revascularization. Need for hard facts. *JAMA (J. Am. Med. Assoc.)* 219: 507–508.

14. Hammermeister, K. E., J. W. Kennedy, G. W. Hamilton, D. K. Stewart, K. L. Gould, K. Lipscomb, and J. A. Murray. 1974. Aortocoronary saphenous-vein bypass. Failure of successful grafting to improve resting left ventricular function in chronic angina. *N. Engl. J. Med.* 290: 186–192.

15. Moran, S. V., R. C. Tarazi, J. U. Urzua, R. G. Favaloro, and D. B. Effler. 1973. Effects of aorto-coronary bypass on myocardial contractility. *J. Thorac. Cardiovasc. Surg.* 65: 335–342.

16. Cody, M. C., H. C. Smith, R. L. Frye, G. K. Danielson, J. R. Pluth, and R. B. Wallace. 1974. Long-term follow-up of flow and dimension changes in saphenous vein bypass grafts to the coronary arteries. *Circulation.* 50 (Suppl. 3): III-132. (Abstr.)

17. Smith, H. C., R. L. Frye, D. E. Donald, G. D. Davis, J. R. Pluth, R. E. Sturm, and E. H. Wood. 1971. Roentgen videodensitometric measure of coronary blood flow. Determination from simultaneous indicator-dilution curves at selected sites in the coronary circulation and in coronary artery-saphenous vein grafts. *Mayo Clin. Proc.* 46: 800–806.

18. Smith, H. C., R. E. Sturm, and E. H. Wood. 1973. Videodensitometric system for measurement of vessel blood flow, particularly in the coronary arteries, in man. *Am. J. Cardiol.* 32: 144–150.

19. Ritman, E. L., R. E. Sturm, and E. H. Wood. 1973. Biplane roentgen videometric system for dynamic (60/sec) studies of the shape and size of circulatory structures, particularly the left ventricle. *Am. J. Cardiol.* 32: 180–187.

20. Dumesnil, J. G., E. L. Ritman, R. L. Frye, G. T. Gau, B. D. Rutherford, and G. D. Davis. 1974. Quantitative determination of regional left ventricular wall dynamics by roentgen videometry. *Circulation.* 50: 700–708.

21. Smith, H. C., R. L. Frye, G. D. Davis, G. K. Danielson, J. R. Pluth, R. B. Wallace, R. E. Sturm, and E. H. Wood. 1971. Measurement of flow in saphenous vein-coronary artery grafts by roentgen videodensitometry. *Circulation.* 44 (Suppl. 2): II-107. (Abstr.).

22. McConahay, D. R., B. D. McCallister, F. J. Hallermann, and R. E. Smith. 1970. Comparative quantitative analysis of the electrocardiogram and the vectorcardiogram: correlations with the coronary arteriogram. *Circulation.* 42: 245–259.

23. Bassan, M., W. Ganz, H. S. Marcus, and H. J. C. Swan. 1975. The effect of intracoronary injection of contrast medium upon coronary blood flow. *Circulation.* 51: 442–445.

24. Gould, K. L., K. Lipscomb, and G. W. Hamilton. 1974. Physiologic basis for assessing critical coronary stenosis. Instantaneous flow response and regional distribution during coronary hyperemia as measures of coronary flow reserve. *Am. J. Cardiol.* 33: 87–94.

25. Chapman, C. B., O. Baker, J. Reynolds, and F. J. Bonte. 1958. Use of biplane cinefluorography for measurement of ventricular volume. *Circulation.* 18: 1105–1117.

26. Wood, E. H. 1976. Cardiovascular and pulmonary dynamics by quantitative imaging. *Circ. Res.* 38: 131–139.

27. Ritman, E. L., R. E. Sturm, and E. H. Wood. 1970. Comparison of volume of canine left ventricular casts and angiograms using biplane and monoplane roentgen videometry. *Physiologist.* 13: 294. (Abstr.).

28. Bourassa, M. G., J. Lésperance, L. Campeau, and J. Saltiel. 1972. Fate of left ventricular contraction following aortocoronary venous grafts. Early and late postoperative modifications. *Circulation.* 46: 724–730.

29. Jacob, K., M. Chabot, J. Saltiel, and L. Campeau. 1973. Improvement of left ventricular asynergy following aortocoronary bypass surgery related to preoperative electrocardiogram and vectorcardiogram. *Am. Heart J.* 86: 438–443.

30. Chaitman, B. R., J. D. Bristow, and S. H. Rahimtoola. 1973. Left ventricular wall motion assessed by using fixed external reference systems. *Circulation.* 48: 1043–1054.

31. De Jong, J. W., and S. Goldstein. 1974. Changes in coronary venous inosine concentration and myocardial wall thickening during regional ischemia in the pig. *Circ. Res.* 35: 111–116.

32. Theroux, P., S. Sasayama, V. Massullo, S. W. Kemper, C. Bloor, D. Franklin, and J. Ross, Jr. 1975. Regional myocardial function early and during the healing of acute myocardial infarction. *Am. J. Cardiol.* 35: 173 (Abstr.).

33. Hood, W. B., Jr., R. Kumar, J. C. Norman, and W. H. Abelmann. 1970. Experimental canine myocardial infarction: serial changes in ventricular function related to mechanical properties of normal and infarcted myocardium. *In* VI World Congress of Cardiology. British Medical Association, London. 167.

34. Assad-Morell, J. L., R. L. Frye, D. C. Connolly, G. T. Gau, J. R. Pluth, D. A. Barnhorst, R. B. Wallace, G. D. Davis, L. R. Elveback, and G. K. Danielson. 1975. Relation of intraoperative or early postoperative transmural myocardial infarction to patency of aortocoronary bypass grafts and to diseased ungrafted coronary arteries. *Am. J. Cardiol.* 35: 767–773.