Left Ventricular Function
Following Coronary Bypass Surgery

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SUMMARY The effect of coronary artery bypass grafts on left ventricular performance was evaluated by analyzing preoperative (preop) and postoperative (postop) biplane left ventriculograms of 37 patients who were restudied at a mean of 13 months after operation. Eighty-two percent of the grafts were patent. Segmental wall shortening and segmental shortening velocities (mean \( V_{c,t} \)) in the anterior, inferior and lateral regions of the left ventricle were compared in four groups: 1) regions with patent grafts and normal preop shortening, 2) regions with patent grafts and preop asynergy, 3) regions with occluded grafts and 4) ungrafted regions. In the 38 regions with preop normal shortening and patent grafts, shortening and shortening velocities were unchanged postop. In the 13 regions with preop asynergy and patent grafts, \( \% \) shortening increased \( 22 \pm 8 \) (\( P < 0.01 \)) and mean \( V_{c,t} \) increased \( 0.82 \pm 34 \) lengths/sec (\( P < 0.025 \)). In the 13 regions with occluded grafts, \( \% \) shortening decreased \( 6 \pm 3 \) (\( P < 0.05 \)) and mean \( V_{c,t} \) decreased \( 0.24 \pm 0.11 \) lengths/sec (\( P < 0.025 \)). In the 28 regions which were ungrafted, there was no change in shortening, while mean \( V_{c,t} \) decreased \( 0.56 \pm 0.22 \) lengths/sec (\( P < 0.025 \)). The effect of bypass grafts on global ventricular performance as measured by the ejection fraction (EF) was examined in patients with all patent grafts and normal preop wall motion, patients with all patent grafts and preop asynergy, patients with one or more occluded grafts and all patients combined. In the 11 patients with all patent grafts and normal preop wall motion, the EF was unchanged \( (0.74 \pm 0.03 \) preop and \( 0.71 \pm 0.02 \) postop; \( P = NS \)), while the EF increased in the 11 patients with all patent grafts and preop asynergy \( (0.53 \pm 0.02 \) preop and \( 0.65 \pm 0.05 \) postop; \( P < 0.05 \)). In the 11 patients with one or more occluded grafts, the EF decreased \( (0.67 \pm 0.04 \) preop and \( 0.57 \pm 0.03 \) postop; \( P < 0.05 \). The mean EF did not change in the entire group \( (0.65 \pm 0.02 \) preop and \( 0.64 \pm 0.02 \) postop; \( P = NS \)). We conclude that patent coronary artery bypass grafts are associated with maintenance of myocardial function in patients with normal preop ventricular function. In patients with depressed ventricular performance, patent grafts result in improvement of regional and global function while occluded grafts result in depression of regional and global performance.

THE RELIEF OF ANGINA PECTORIS after coronary artery bypass surgery is now well-documented. 1-3 Although symptomatic improvement is a therapeutic goal, assessment of patient symptoms remains subjective, and there is poor correlation of symptoms with objective parameters of myocardial blood flow and ventricular performance in patients with coronary artery disease. The question of whether surgery ultimately improves patient longevity is currently under study. It is known that the state of left ventricular function is a potent predictor of patient survival for patients with coronary artery disease. 4, 5 Therefore, the effects of coronary bypass surgery upon left ventricular function may have important implications for patient longevity.

This study tested the hypothesis that patent grafts improve regional and global left ventricular function in patients who have depressed left ventricular function preoperatively. This was accomplished by a biplane, multiple frame, computer-based analysis of left ventriculograms both before and after operation. This analysis provided a quantitative profile of ventricular volumes, wall shortening and shortening velocities of the three regions supplied by each major coronary vessel.

Methods

Patient Population

Two hundred nineteen consecutive patients with angina and coronary artery disease who underwent cardiac catheterization and aortocoronary bypass grafting at Temple University Hospital were selected for study.

Eight patients were catheterized within six months after operation because of recurrent chest pain. All patients were asked to return for repeat catheterizations approximately one year after operation. Thirty-three additional patients agreed to return, while the remaining patients either declined a repeat catheterization or died. Informed consent was obtained from all patients. Of these 41 patients, three had ventricular aneurysmectomies and one had a mitral valve replacement. The remaining 37 patients constituted the study group of nine women and 28 men. Their ages ranged from 31-72 years, with a mean of 53 \pm 1.5 years. All patients had angina before operation. Eighty-four vessels in these 37 patients contained significant stenoses and 77 of these vessels received a bypass graft. Ten patients received one graft; 15 patients received two grafts; 11 patients

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received three grafts; and one patient received four grafts. Following operation, 27 of the 37 patients were 
angina-free, while 10 patients continued to have chest 
pain.

Data Acquisition

All patients underwent cardiac catheterization in 
the fasting state while mildly sedated with diazepam. 
In patients receiving propranolol therapy, the drug 
was discontinued at least 48 hours before the 
catheterization. The preoperative study was per-
formed within one month of operation and the post-
operative study was performed until 24 months 
(13 ± 1 month) after operation. The preoperative study 
was performed using the transbrachial technique, 
and the postoperative study was performed using 
the transfemoral technique. Aortic and left ventric-
ular pressures were measured using Statham P-23 Db 
transducers with pressures referenced to atmospheric 
pressure 5 cm below the sternal angle. The left ventric-
ular end-diastolic pressure was measured following 
the a wave and averaged over one respiratory cycle. 
The heart rate was measured during ventriculog-
raphy.

Biplane left ventriculography was performed using 
a biplane Siemens's 10-6 in image intensifier system 
and recorded on 35 mm film at 60 frames/sec. A 
power injection into the left ventricle using 30-50 ml 
of Renografin 76 was made through a multihole 
catheter placed retrogradely across the aortic valve. 
The simultaneous electrocardiogram was recorded 
with a cinetrace in the upper left hand corner of the 
film. Twenty-seven patients had biplane 30° right 
anteior oblique ventriculography, and 10 patients had 
monoplane 30° right anterior oblique ventriculo-
graphy. In all patients, left ventriculography preceded coronary angiography. Calibration of the 
ventricular image size was made using either a 10 cm 
grid or a 6.6 cm ball filmed at the level of the left ven-
tricle with the same tube to image intensifier distance 
used during ventriculography. Selective coronary and 
graft cineangiograms were recorded in multiple pro-
jections.

Data Analysis

Frame-by-frame biplane analysis was performed 
using a digital plotting table connected on line to a 
PDP-9 digital computer. The images were displayed 
side by side on the plotting table by two overhead pro-
jectors which allowed the investigator to scan the 
biplane ventricular images (fig. 1). The apex and mid-
point of the aortic valve of each projection were iden-
tified and a long axis determined. As the outlines were 
scanned, position coordinates of the endocardial sur-
face were transferred to the computer and stored on 
digital magnetic tape for analysis. During the scan-
nling of one biplane image, the long axis was divided 
by the computer into 50 equal segments, and 50 
diameters perpendicular to the long axis were con-
structed. The distance from the long axis to the endo-
cardial surface delineated 50 hemisegments on each 
side of the long axis. X-Y coordinates identifying 
these points, the calibration data, and the electro-
cardiogram were stored on digital tape. One complete 
cardiac cycle was analyzed for each ventriculogram. 
All premature contractions and immediately 
successive beats were excluded from analysis.

Left ventricular volumes were calculated using 
Simpson's rule applied to the 50 equidistant diameters 
constructed perpendicular to the long axis as described 
above. Based on the analysis of seven human left ven-
tricular casts, the volumes were corrected according 
to the formula \( V_a = V_s (1.028) - 7.937 \), where \( V_a \) = ac-
tual volume and \( V_s \) = calculated volume. In the single 
plane studies, the 50 diameters from the right anterior 
oblique images were used.

Regional ventricular analysis was performed on the 
anteior and inferior surfaces of the right anterior 
oblique projection and the lateral surface of the left 
anteior oblique projection, corresponding to the dis-
bution of the three major coronary arteries. The 
anteior wall was considered to correspond to the left 
anteior descending artery distribution; the inferior 
region to the right coronary artery distribution, in a 
right dominant system; and the lateral region to the 
circumflex marginal artery distribution. In the case of 
a left dominant system, the inferior region was con-
sidered to be in the distribution of the circumflex cor-
onary artery. For the regional wall motion analysis, 
the superior 9 and inferior 8 of the original 50 
hemisegments were deleted. The remaining 33 
hemisegments were consolidated into 11 by success-
vously averaging every three of the hemisegments (fig. 
1). The 11 hemisegments identifying each region were 
averaged to give a mean hemisegment length. The computer was programmed to calculate the hemiseg-
ment length for each frame analyzed, to find end 
diastolic hemisegment (longest) length and the end 
systolic hemisegment (shortest) length. Using the
lengths obtained, the percent shortening was calculated from the formula:

\[
\text{% shortening} = \frac{\text{end diastolic length} - \text{end systolic length} \times 100}{\text{end diastolic length}}
\]

Mean shortening velocity (mean \(V_{ef}\)) was calculated by subtracting end systolic length from end diastolic length and dividing by time. An average of the values of the middle 33 hemisegments along one region was calculated and represented the value for a region.

Selective coronary and graft cineangiography were evaluated and the severity of a stenosis was graded by the estimated smallest diameter from multiple projections. The native circulation was evaluated for changes in the original lesions and the appearance of new stenoses. Grafts were evaluated for patency, narrowing and the presence of disease distal to the insertion of the graft. Preoperative and postoperative regional wall motion, as measured by percent shortening and by mean wall shortening velocity, were compared in four groups: 1) 38 regions with patent grafts and normal preoperative regional wall motion as judged qualitatively from the ventriculogram, 2) 13 regions with patent grafts and preoperative asynergy as judged qualitatively from the ventriculogram, 3) 13 regions with occluded grafts and 4) 28 regions that received no grafts. The preoperative and postoperative left ventricular end-diastolic pressure, mean aortic pressure, heart rate, left ventricular end-diastolic volume index and ejection fraction were compared in four groups: 1) all 37 patients combined, 2) 11 patients with preoperative normal ventricular function and patent grafts, 3) 12 patients with preoperative asynergy and patent grafts and 4) 11 patients with one or more occluded grafts.

The paired t test with n-1 degrees of freedom was used for statistical analyses. Group data are expressed as mean ± SEM.

**Results**

A summary of individual patient data is provided in table 1.

**Electrocardiographic Changes and Graft Patency**

Seven patients had preoperative resting electrocardiograms diagnostic of old myocardial infarctions. One of the 37 patients (2.7%) developed new Q waves diagnostic of myocardial infarction in the postoperative period.

Sixty-two of 77 grafts (82%) were patent at the time of the postoperative study. Seven of the 62 patent grafts had an area of 50% or greater narrowing. Since the efficacy of the stenotic grafts was uncertain, the regions supplied by these grafts were not included in the analysis of wall motion or ventricular volumes. Twenty-seven out of 30 (90%) of the left anterior descending grafts, 23 out of 30 (77%) of the right coronary grafts, and 13 out of 17 (77%) of the circumflex grafts were patent.

**Hemodynamic Findings (table 2)**

In the 22 patients with all grafts patent and the 11 patients with one or more occluded grafts, there was no change in either the heart rate or the mean aortic pressure from the preoperative to the postoperative study.

In the 22 patients with patent grafts, the left ventricular end-diastolic pressure increased from 8 ± 1 mm Hg preoperatively to 11 ± 1 postoperatively (\(P < 0.05\)). In the 11 patients with one or more occluded grafts, the left ventricular end diastolic pressure increased from 8 ± 1 mm Hg to 11 ± 1 mm Hg (\(P < 0.05\)). These changes, while statistically significant, were small, and the left ventricular end-diastolic pressure remained in the normal range postoperatively in both groups.

**Regional Wall Motion (figs. 2 and 3)**

In the 38 regions with no preoperative asynergy and patent grafts, there was no change in percent shortening, averaging 41 ± 4 preoperatively and 43 ± 3 postoperatively (\(P = NS, fig. 2\)); and there was no change in mean \(V_{ef}\), averaging 1.35 ± 0.16 lengths/sec and 1.40 ± 0.11 lengths/sec postoperatively (\(P > 0.10\), fig. 3).

In the 13 regions with preoperative asynergy and patent grafts, there was an increase in percent shortening from 12 ± 8 preoperatively to 36 ± 8 postoperatively (\(P < 0.01\), fig. 2), and there was an increase in mean \(V_{ef}\) from 0.46 ± 0.2 lengths/sec preoperatively to 1.29 ± 0.37 lengths/sec postoperatively (\(P < 0.025\), fig. 3).

In the 13 regions with occluded grafts, there was a decrease in percent shortening from 43 ± 4 preoperatively to 37 ± 5 postoperatively (\(P < 0.05\), fig. 2), and a decrease in mean \(V_{ef}\) from 1.43 ± 0.14 lengths/sec preoperatively to 1.19 ± 0.14 lengths/sec postoperatively (\(P < 0.025\), fig. 3).

In the 28 regions where no grafts were placed, percent shortening was unchanged, measuring 43 ± 3% preoperatively and 40 ± 4% postoperatively (\(P = NS\), fig. 2). Mean \(V_{ef}\) decreased from 1.90 ± 0.22 lengths/sec preoperatively to 1.33 ± 0.13 lengths/sec postoperatively (\(P < 0.025\), fig. 3).

**Left Ventricular Volumes**

In all 37 patients, the left ventricular end-diastolic volume index remained unchanged, averaging 82 ± 4 ml/m² preoperatively and 80 ± 3 ml/m² postoperatively (\(P = NS\)). Similarly, in the subgroups of 11 patients with patent grafts and preoperative normal wall motion, 11 patients with patent grafts and preoperative asynergy, and 11 patients with one or more occluded grafts, there was no significant difference between the preoperative and postoperative end-diastolic volume.
TABLE 1.  Hemodynamic, Volumetric, Regional and Graft Data for 37 Patients

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<tr>
<th>Patients with no Preoperative Asynergy and Patent Bypass Grafts</th>
<th>age (Yrs)</th>
<th>2nd Study</th>
<th>LVEDP</th>
<th>Mean aortic pressure</th>
<th>Heart rate</th>
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Patients with Preoperative Asynergy and Patent Bypass Grafts

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Patients with Graft Stenosis or Distal Vessel Disease

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<th>2nd Study</th>
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<th>Mean aortic pressure</th>
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</table>

* = region with preoperative asynergy; (+) = patent graft; (-) = occluded graft; (0) = ungrafted; (O) = patent graft with graft stenosis or distal disease. Mean aortic pressure measured in mm Hg; heart rate measured in beats/min; shortening expressed as a percent of end diastolic heme segment length; mean V6 measured in ml/sec.

Abbreviations: LVEDP = left ventricular end diastolic pressure measured in mm Hg; LVEDVI = left ventricular end diastolic volume index measured in ml/m²; LAD = left anterior descending coronary artery; RCA = right coronary artery; Mrg = circumflex marginal coronary artery.

Left Ventricular Ejection Fraction (fig. 4)

In the entire group of 37 patients, there was no change in the ejection fraction, averaging 0.65 ± 0.02 preoperatively and 0.64 ± 0.02 postoperatively. In the 11 patients with patent grafts and normal preoperative ventricular function, there was no change in the ejection fraction, averaging 0.74 ± 0.03 preoperatively and 0.71 ± 0.02 postoperatively. In the 11 patients with patent grafts and preoperative asynergy, the ejection fraction increased from 0.53 ± 0.02 preoperatively to 0.65 ± 0.05 postoperatively.

TABLE 2. Hemodynamic Parameters

<table>
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<th>Mean aortic pressure (mm Hg)</th>
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<td>Postop</td>
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<tr>
<td>(22 patients)</td>
<td>98 ± 3</td>
<td>96 ± 3</td>
<td>77 ± 3</td>
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<td>One or more grafts occluded</td>
<td>100.1 ± 3</td>
<td>94.6 ± 3.4</td>
<td>75 ± 5</td>
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</table>

* = P < 0.05.

Abbreviation: LVEDP = left ventricular end diastolic pressure.
(P < 0.05). In the 11 patients with one or more occluded grafts, the ejection fraction decreased from 0.67 ± 0.04 preoperatively to 0.57 ± 0.03 postoperatively (P < 0.025).

### Discussion

There are four problems in the evaluation of the effects of coronary bypass surgery on left ventricular performance. First, factors that may effect changes in left ventricular function other than the bypass grafts themselves should be accounted for. These factors include unrelated changes in preload, afterload and contractility. For example, changes in autonomic tone between the preoperative and postoperative study are known to alter ventricular function. Although precise manipulation of these variables is not possible in human studies, no change in left ventricular end-diastolic volume (preload), systemic blood pressure (afterload) or heart rate (autonomic tone) was found between the preoperative and postoperative studies.

Second, the method of assessing left ventricular performance may alter the conclusions. Qualitative estimates of ventriculographic contraction patterns alone are too imprecise to accurately determine changes between a preoperative and postoperative study. Even quantitative measurements have limitations. For example, measurements of ventricular volumes and the ejection fraction are useful but do not adequately describe the regional changes in wall motion which occur in patients with coronary artery disease. Therefore, an analysis of regional wall motion utilizing quantitative determinations of shortening is essential. Quantitative measurements are particularly important in comparative studies where the changes between studies may be small. This investigation does provide a comprehensive quantitative analysis of regional wall shortening and velocity of shortening in patients before and after coronary artery bypass surgery.
Third, single plane, right anterior oblique ventriculographic studies do not assess all regions of the ventricle, particularly the lateral wall which is supplied by the circumflex coronary artery. Biplane ventriculographic studies in patients with coronary artery disease provides a more comprehensive assessment of ventricular function than can be derived from single plane studies alone. The biplane analyses performed in 27 of 37 patients in this study gives a complete profile of ventricular function in a majority of the patients.

Fourth, the left ventricular contractile state may vary with the timing of the postoperative ventriculographic study. It has been suggested that the increased stress and sympathetic tone in the early postoperative period may result in increased left ventricular contractility and function. This study has circumvented this potential effect by performing the postoperative study an average of 13 months following operation.

The data presented relies on the accuracy of the method for assessing regional function. It has been suggested that the hemisegment length analysis not only measures shortening in the region of interest but may be influenced significantly by shortening of surrounding myocardium. Although this may occur, the averaging of several hemisegment lengths from a single region as performed in this investigation minimizes the influence of any discordant shortening changes on the borders of the region. Hugenholtz et al. have suggested that the angiographic methods for quantification of regional wall motion are only marginally useful when applied to patients with coronary artery disease since it is difficult to obtain truly comparable films and the data is internally inconsistent. This has not been a problem in our study, since we have made special efforts to maintain uniform film quality, taken special care in performing the left ventriculogram so that adequately opacified and arrhythmia-free beats were obtained, and the exact degree of rotation was duplicated on each study. The inconsistency of the data was eliminated by the same computer-based mathematical techniques on all ventriculograms. We have measured both the interobserver and intra-observer variation and found that the data is reproducible within the limits necessary to assess regional wall motion.
The results of this study demonstrate that patent bypass grafts are associated with increased shortening and shortening velocities in segments that are asynergic preoperatively, whereas there is no change in segments which contract normally preoperatively. Improved regional myocardial function following successful bypass surgery has been described by some investigators, while others have found little or no change in regional function. Our data illustrate that regional function cannot be expected to improve unless preoperative function is depressed. This concept is perhaps intuitive, but frequently overlooked in the analysis of data by previous investigators. It is tempting to attribute the maintenance of normal ventricular function in regions which were normal preoperatively to the preserving effects of the bypass grafts. However, this phenomenon cannot be established unless the natural history of the segment supplied by a stenotic coronary artery is known and can be compared to the natural history of the bypassed segment. In the present study, the depression of ventricular function seen with occluded bypass grafts occurred whether or not there was progression of the disease in the native vessels. This suggests that occlusion of a bypass graft even without changes in the native circulation is not a benign phenomenon. The regions which were not bypassed showed no significant change in the magnitude of shortening, although shortening rates were reduced. These data do suggest a subtle depression of contractility in ungrafted segments, although the reasons for this are unclear from this study.

The changes in regional ventricular function seen in patients with patent and occluded bypass grafts was of sufficient magnitude to result in corresponding changes in global function as measured by the ejection fraction. However, no change in the end-diastolic volume of patients with either patent or occluded grafts was seen. Rees, Hammermeister and Shepherd also found no change in the end-diastolic volume postoperatively. However, Chatterjee et al. found reductions in the end-diastolic volume postoperatively in patients who had depressed ejection fractions preoperatively. Reduced postoperative end-diastolic volumes were also found by Kennedy et al. in patients who were considered completely revascularized. Arbegast et al. found increases in the end-diastolic volume in patients with occluded grafts. Our data suggest that the end-diastolic volume is not closely related to graft patency or to the effects of coronary bypass surgery on left ventricular contractile performance. This is an expected finding, since increases of left ventricular end-diastolic volume are not consistently found, even in patients with advanced coronary atherosclerosis, unless ventricular function is severely depressed.

This study demonstrates a close relationship between changes in left ventricular performance and the patency of coronary artery bypass grafts. Patent coronary artery bypass grafts are associated with improved myocardial performance in regions that are asynergic preoperatively, while patent coronary artery bypass grafts are associated with no change in myocardial function of segments which are preoperatively normal. Conversely, occlusion of bypass grafts results in depression of myocardial performance.

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The Björk-Shiley Aortic Prosthesis: Flow Characteristics, Thrombus Formation and Tissue Overgrowth

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SUMMARY. Thrombus formation and tissue overgrowth were observed in nine Björk-Shiley aortic prostheses recovered six months or longer after implantation. These pathologic findings may be attributed to the flow characteristics of the prosthesis. The open disc of the valve separates the flow into two unequal regions. Varying degrees of thrombus formation were observed in the minor outflow region, including the depression in the aortic face of the disc and the metal strut bridging this area. Tissue overgrowth was noted along the perimeter of the prosthesis adjacent to the minor outflow region. That overgrowth further reduced the available cross section for flow in this already constrained area. In vitro velocity measurements with a laser-Doppler anemometer identified a zone of stagnation about 20 mm wide near the aortic face of the disc. The average velocities in the major and minor outflow regions were around 100 and 25 cm/sec, respectively, and the corresponding peak-shear stresses were approximately 700 and 150 dynes/cm². There is reason, then, to attribute the thrombus formation and tissue overgrowth to the stagnation zone and the low shear in the minor outflow region.

THE BJÖRK-SHILEY TILTING AORTIC PROSTHESIS has been in clinical use since January 1969.1 Its major advantages are 1) low pressure drop,2 2) low level of hemolysis,3, 4 3) low profile,2, 5 and 4) structural durability. The majority of patients achieve significant functional improvement after replacement of their aortic valves with this prosthesis.

As shown in figure 1, the valve creates two regions of unequal flow. In the past few years several reports6-15 have described massive thrombosis with the use of this valve, especially in patients who were not receiving anticoagulation therapy. A ring-shaped radiopaque marker has recently been incorporated into the disc to assist in diagnosing this problem.11 In this present work thrombus formation was also observed along with tissue overgrowth around the perimeter of the minor outflow region, which further reduced the flow in this area.

The present article reports two studies: 1) clinical pathology findings of recovered Björk-Shiley aortic prostheses and 2) in vitro measurements of velocities in the near vicinity of a normally functioning and a partially occluded (fig. 2) Björk-Shiley valve. Correlation of these studies is unique and very useful in understanding why and where thrombus formation and tissue overgrowth occur.

Methods

Clinical Pathology Findings

Seventeen Björk-Shiley aortic prostheses were examined, 16 at autopsy and one after surgical removal. In eight patients the implant duration was less than one month. The other nine patients had un-
Left ventricular function following coronary bypass surgery.
N M Wolf, T H Kreulen, A A Bove, M T McDonough, K M Kessler, M Strong, G LeMole and J F Spann

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