# MYOCARDIAL REVASCULARISATION IN CORONARY ARTERY DISEASE

## THE SPECTRUM OF ABNORMAL LEFT VENTRICULAR FUNCTION IN ISCHEMIC HEART DISEASE AND THE INFLUENCE OF SUCCESSFUL SAPHENOUS VEIN GRAFTING

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Coronary artery disease frequently results in regional abnormalities in ventricular contraction. With the development of saphenous vein by-pass grafting a practical method to revascularize the myocardium is now available. Clinical results in most cases are good in the control of anginal pain. It is not yet known, how frequently successful saphenous vein by-pass grafts will improve left ventricular function in patients with chronic ischemic heart disease. This study reports the spectrum of left ventricular functional abnormalities present in 66 patients with ischemic heart disease who were studied by selective coronary arteriography and left ventricular biplane angiocardiography. Twenty one additional patients have been studied in a similar manner before and following saphenous vein aorta to coronary artery by-pass grafting. The changes in left ventricular function and regional contraction were analysized in an attempt to show the effect of successful revascularization on left ventricular performance.

### METHODS

All patients were evaluated by left heart catheterization by the retrograde femoral approach. The left ventricle was entered and pressure, and cardiac output by the Fick method was measured. Following these measurements left ventricular angiocardiography was performed. In most instances filming was carried out with a biplane film changer at 6 or 12 exposures per second. In five of the operative cases right anterior oblique single plane cine angiography was employed.

Left ventricular volume was calculated during one or more cardiac cycles and left ventricular time—volume curves were constructed. End diastolic volume, end systolic volume, stroke volume and ejection fraction $\frac{SV}{EDV} = EF$ were determined from these curves.[1] Left ventricular pressure-volume diagrams were constructed and stroke work was determined by integrating the area enclosed by the pressure volume loop. Peak systolic and end diastolic stress were determined by the thin wall model of Sandler and Dodge.[2]

The pattern of left ventricular contraction was determined in two planes by the method of Herman *et al.*[3] Left ventricular contraction patterns were divided into five groups as suggested by Hamilton.[4]

Type I —Normal symmetrical contraction.

Type II —Borderline abnormality in contraction comprising less than 25% of the left ventricular surface.

Type III —Localized akinesis or hypokinesis envolving 25% to 75% of the left ventricular surface.

Type IV —Localized dyskinesis greater than 25% of the left ventricular surface (true ventricular aneurysm).

Type V —Diffuse hypokinesis or akinesis greater than 75% of the ventricular surface.

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**Results—non-operative group**

Of sixty-six patients studied in the non-operative group contraction patterns were as follows:

Type I — 16 patients
Type II — 17 "
Type III — 14 "
Type IV — 11 "
Type V — 8 "

The type of contraction abnormality was not related to age, duration of symptoms, presence of angina pectoris or hypertension. The percentage of patients with congestive heart failure showed a striking increase with progressive contraction abnormalities. Mitral regurgitation was seen primarily in patients with diffuse akinesia or hypokinesia (type V).

The ejection fraction was normal (above 50%) in nearly all patients with type I and II contraction patterns (32/33). Eleven of 14 patients with type III and all with type IV and V contraction patterns had abnormally low ejection fractions.

An elevated LV end diastolic pressure was found in 41% of patients (27/66) and increased in frequency with the severity of contraction abnormality occurring in 12% in type I, 35% of type II, 21% of type III; 82% of type IV and 88% of type V. 68% of patients who had definitely abnormal contraction had elevated filling pressure.

Left ventricular stroke work decreased as contraction abnormalities increased in severity whereas left ventricular peak systolic and end diastolic stress showed considerable overlap among the various contraction groups.

**Clinical Relationships**

Angina pectoris was present in 86% of these patients. When angina occurred in the absence of a history of myocardial infarction, heart failure, or evidence of mitral regurgitation left ventricular volume, mass, ejection fraction, contraction pattern, wall stresses and stroke work were nearly always normal.

Myocardial infarction had occurred in 67% of these patients. All patients with type IV and V contraction abnormalities and 92% with type III abnormalities had experienced one or more myocardial infarctions. 18% with normal contraction, and 52% with borderline contraction abnormalities had a history of myocardial infarction. Patients without myocardial infarction had normal left ventricular contraction except for a few with minor increases in end diastolic and end systolic volume.

When variables which describe left ventricular systolic function such as ejection fraction, stroke work and contractile pattern were compared with parameters of left ventricular diastolic function including end diastolic pressure, volume and stress, a curvilinear relationship was apparent. This curve resembles a Starling left ventricular function curve made up of single observations from a population of ventricles variously affected by coronary artery disease. Fig. 1 presents the relationship between end diastolic volume and the ejection fraction in these patients. The curvilinear relationship is evident. The circled points indicate those patients with a history of congestive heart failure. All patients who had heart failure had an ejection fraction below 40%. The comparison of systolic and diastolic events and their relationship to myocardial infarction, compensated and decompensated congestive heart failure,

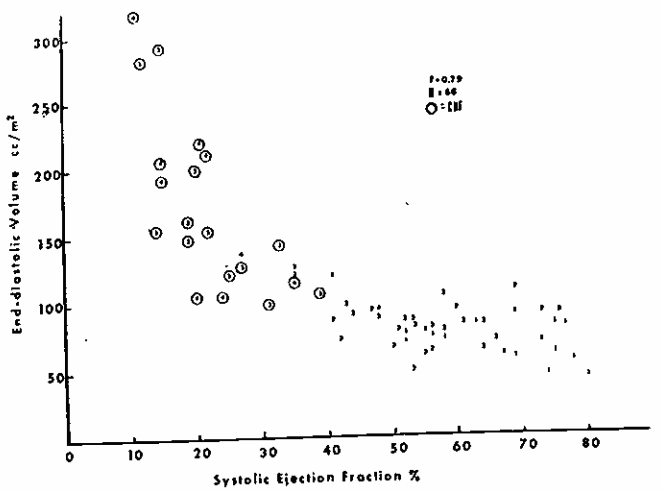


Fig. 1. The relationship between systolic ejection fraction, end diastolic volume and type of contraction as indicated by the numbers representing each patient. The circled points are patients who have congestive heart failure (C.H.F.). Note all patients with heart failure have consistent elevation in end diastolic volume and ejection fraction below 40%.

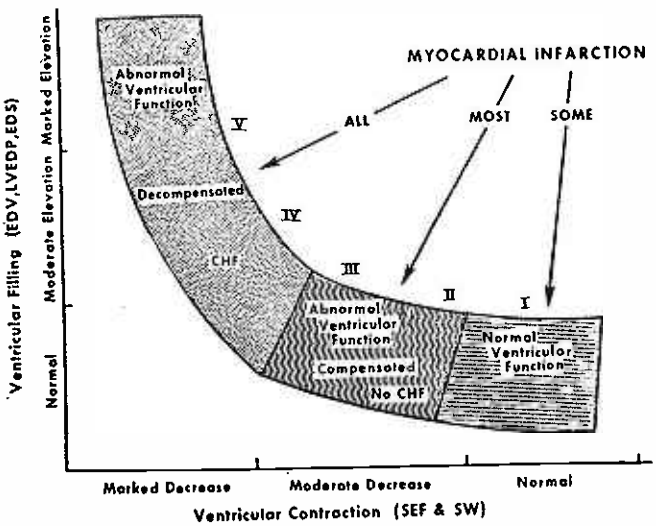


Fig. 2. This chart represents an idealized summary of the relationship between systolic and diastolic events in patients with ischemic heart disease. Ventricular contractile function on the x axis is represented by ejection fraction or stroke work (SEF, SW) and diastolic function on the y axis by end diastolic volume, pressure and stress (EDV, LVEDP and EDS). Areas of the curve are identified as to ventricular function, incidence of myocardial infarction and type of ventricular contraction pattern.

and normal and abnormal left ventricular contractile pattern has been summarized in Fig. 2.

### Results of Saphenous Vein Aorta to Coronary Grafting

Of 45 patients studied an average of 4·5 months following vein graft by-pass surgery, 21 patients had left ventricular angiocardiography before and after surgery which was adequate to analysis left ventricular volume during at least one cardiac cycle. Sixteen of these patients were studied by biplane angiocardiography at 12 exposures per second and 5 were studied by single plane cine angiography in the RAO projection.

## RESULTS

All grafts were patents in 17 of these patients, 1 of 2 grafts were patent in 3 patients and a single graft was closed in one patient. One patient suffered a q wave myocardial infarction during the operative procedure.

All 17 patients with completely patent grafts were improved with 5 becoming asymptomatic and 12 having mild symptoms. Only one had residual angina with the symptoms in the others being mild dyspnoea on exertion or fatigue.

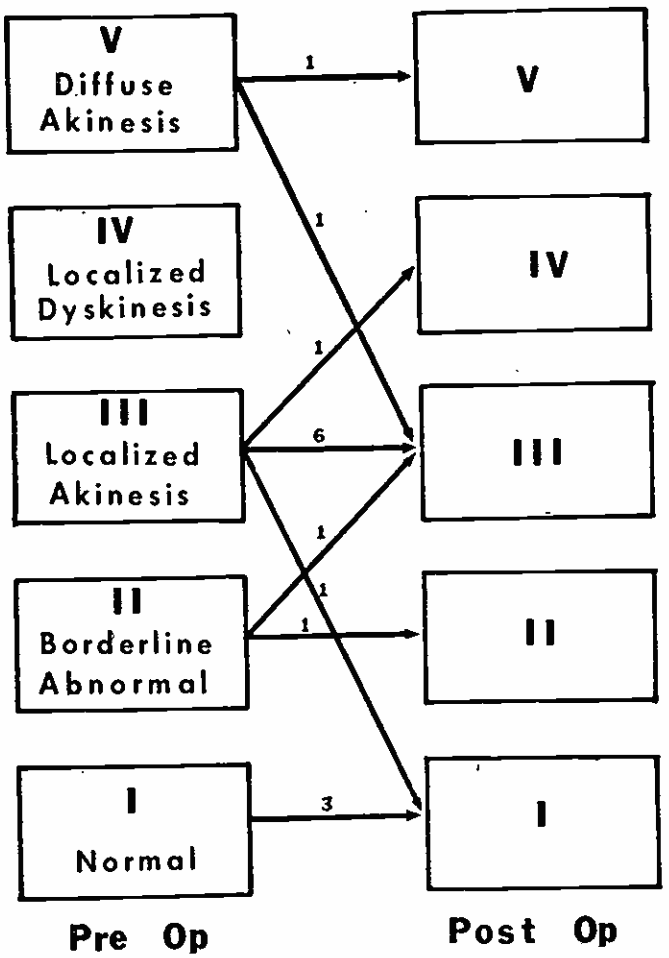


Fig. 3. Contraction patterns seen in 15 cases before and following by-pass graft surgery. Note only two patients improved and two deteriorated. Eleven patients showed no change in contraction pattern following surgery.

Thirteen patients with patent grafts were tested to maximal exertion on a treadmill before and after surgery. Five patients showed improved exercise tolerance, seven showed no significant change and one deteriorated. Failure to improve by objective testing in 8 patients is in contrast to improved functional class and loss of angina pectoris in all but one patient.

Ten of thirteen patients tested had positive ischemic ST response on the exercise electrocardiogram prior to surgery. Following revascularization 8 of 10 patients lost their ischemic response and two remained positive.

All 21 patients were included in the volume analysis. Left ventricular end diastolic volume average 86·9 ml./M$^2$ $\pm$ 23·5 ml. before surgery and 86·0 ml./M$^2$ $\pm$ 28·4 ml. following surgery. There was no significant difference for the group as a whole, for the 17 patients with all patent grafts or for the six patients with abnormally elevated end diastolic volume preoperatively. End systolic volume average 43·1 ml./M$^2$ $\pm$ 20·9 ml. before surgery and did not change following surgery.

The ejection fraction or percent of left ventricular emptying per beat averaged 50·9% $\pm$ 10·6 before surgery and was 49·0% $\pm$ 2·4 following surgery. There was no significant change in the 17 patients with patent grafts or in those 17 patients who had reduced ejection fractions prior to surgery. The ejection fraction increased more than 7% in 5 patients and decreased more than 7% in 7 patients following surgery. Three of four patients with close grafts had a reduction in ejection fraction following surgery. In only one case was a reduced ejection fraction following surgery due to a recognized operative myocardial infarction. Contraction plots were analysized in 16 patients before and after surgery, and are presented in Fig. 3. These revealed no consistent change although there were alterations in contractile pattern following surgery in four patients two of whom improved and two deteriorated.

## CONCLUSIONS

Studies of left ventricular function and contractile pattern in patients with ischemic heart disease reveals a high proportion of patients with local contractile abnormalities. Angina pectoris without a history of heart failure, myocardial infarction or mitral regurgitation is usually associated with normal left ventricular contractile pattern and function as studied and rest in the catheterization laboratory.

Patients with prior myocardial infarction nearly always have localized contraction abnormalities and those with heart failure or mitral regurgitation have severe abnormalities in left ventricular contractile pattern and function. A comparison of systolic and diastolic parameters of ventricular function yields a curvilinear relationship indicating a ventricular function curve made of single observations from a population of diseased ventricles.

Successful saphenous vein aorta to coronary by-pass in a small series of patients has resulted in uniform subjective improvement, lost of angina pectoris in 16 of 17 and improved objectively measured exercise performance in 5 of 13 patients tested. Ischemic changes present on the exercise ECG in 10 patients became normal in 8 patients following surgery. End diastolic volume, end systolic volume, ejection fraction and contraction pattern showed no consistent change following surgical treatment.

We conclude that saphenous vein aorta to coronary by-pass grafting is effective treatment for angina pectoris. When applied to patients with chronic ischemic heart disease by-pass grafting does not appear to improve left ventricular function as evaluated by quantitative angiocardiographic techniques.

## REFERENCES

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