

Cardiomyopathic syndrome due to coronary artery disease¹

I: Relation to angiographic extent of coronary disease and to remote myocardial infarction

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The relation between the extent of angiographic coronary artery disease and the presence of chronic heart failure in patients with coronary artery disease has not hitherto been clearly elucidated. In the present study clinical, ventriculographic, and coronary arteriographic features are compared in 84 patients with coronary artery disease. The extent of coronary disease is measured by the angiographic jeopardy score, which expresses how many of six coronary arterial segments are jeopardised by significant (>70% estimated luminal area reduction) proximal stenoses. Each jeopardised segment is counted as 2 points.

Thirty of the 84 patients had clinical evidence of chronic heart failure, all of whom were classified as having a cardiomyopathic syndrome caused by coronary artery disease since all had significant reduction of the left ventricular ejection fraction (<0.48) because of multiple and widespread left ventricular wall motion abnormalities. There were no patients in this study with chronic heart failure from other causes (e.g. isolated ventricular aneurysm).

The mean jeopardy score in the patients with cardiomyopathy was much higher than in the patients without cardiomyopathy (10.7 ± 0.4 vs 5.3 ± 0.6 , $P < 0.01$), and all patients with cardiomyopathy had a jeopardy score ≥ 8 . Distal coronary artery disease was unrelated to the presence or absence of cardiomyopathy (0.5 ± 0.2 vs 0.4 ± 0.2 diseased distal vessels per patient). Cardiomyopathy was strongly associated with the occurrence of multiple myocardial infarcts (1.9 ± 0.3 myocardial infarcts per patient) in the group with cardiomyopathy and 0.9 ± 0.2 myocardial infarcts per patient in the group without it; 19 of 30 (64%) patients with cardiomyopathy had ≥ 2 myocardial infarcts, whereas 11 of 54 (20%) without had ≥ 2 myocardial infarcts ($P < 0.001$).

The findings of this study, that cardiomyopathy due to coronary artery disease is specifically related to the extent of proximal coronary artery disease and to the occurrence of multiple myocardial infarcts, constitute reasons for distinguishing this cardiomyopathic syndrome from the several other causes of chronic heart failure in coronary artery disease.

The angiographic scoring system used here, which more precisely relates the extent of coronary disease to the extent of left ventricle involved, was a stronger predictor of the presence or absence of the cardiomyopathic syndrome than the 1, 2, 3-vessel disease notation.

It is well known that patients with coronary artery disease may develop chronic heart failure. However, the relation between the extent of left ventricular dysfunction in such patients and the corresponding

extent of coronary artery disease as shown by coronary angiography has not been clearly elucidated. Reported studies have come to differing conclusions. Proudfit and co-workers (1968) found that, if patients with extracardiac factors (severe anaemia, etc.) or isolated aneurysms were excluded, chronic heart failure in coronary artery disease was

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always associated with severe 2- or 3-vessel disease. Other investigators, however, have emphasised that there is little relation between clinical heart failure, or between the haemodynamic and ventriculographic correlates of heart failure, and the extent of coronary artery disease (Herman *et al.*, 1967; Baxley *et al.*, 1971; Cohn *et al.*, 1974b).

The present study compares clinical, ventriculographic, and arteriographic features in 84 patients with coronary artery disease. In particular, the purpose of this study is to analyse the extent of angiographic coronary disease in a subgroup of patients with coronary artery disease and chronic heart failure—those with multiple and widespread left ventricular wall motion abnormalities present in enough degree to reduce significantly the left ventricular ejection fraction at rest. We refer to this condition as 'cardiomyopathy due to coronary artery disease' in order to distinguish it from other causes of left heart failure in coronary artery disease and because it resembles idiopathic cardiomyopathy on ventriculography. We will also examine the relation of this cardiomyopathic syndrome to previous myocardial infarction.

Methods

PATIENT POPULATION

The study population consists of 84 patients shown by coronary angiography to have significant coronary artery disease affecting at least one major coronary artery. Since these patients were also selected for purposes of another study (Dash *et al.*, 1977), involving a comparison of coronary disease in patients with and without diabetes mellitus, one half had evidence of diabetes mellitus.

The clinical indications for angiographic study in these patients were representative of the clinical indications for angiography generally applicable in our institution. Seventy-four patients had angina of effort. Sixty-two patients had had one or more hospital admissions for unstable angina (prolonged chest pain without evidence of myocardial infarction, or a recent change in the frequency of angina). Nine patients were studied because of symptoms of heart failure and did not have disabling angina.

There were 72 men and 12 women. The mean age of the 84 patients was 54 years (range 33 years to 68 years). Twenty-one patients were known to have a history of persistent or labile hypertension (systolic blood pressure ≥ 150 mmHg, diastolic blood pressure ≥ 95 mmHg, or both).

EVALUATION OF CLINICAL DATA

The medical record and electrocardiograms of each patient were carefully reviewed. Signs and symptoms

of heart failure, as well as histories of digitalis and diuretic administration, were noted (see below).

Myocardial infarcts were defined as transmural or non-transmural. The former were diagnosed only in the presence of electrocardiographic Q waves of at least 0.04 s in duration; or, if Q waves were equivocal, akinesis or dyskinesis of the appropriate segment of the left ventriculogram and a typical history. Non-transmural infarcts were diagnosed when typical serum enzyme changes were unaccompanied by the evolution of Q waves.

Electrocardiographic left atrial enlargement was defined as terminal P wave negativity in lead V1 exceeding 0.03 mm/s.

RADIOLOGICAL ASSESSMENT

Coronary arteriograms, left ventriculograms, and plain chest films were viewed by one of us (R.E.D.) without knowledge of the clinical status of the patient.

All patients had chest x-ray films which were assessed for size of cardiac chambers and signs of pulmonary venous hypertension.

Selective coronary angiography was performed by either the Sones or Judkins techniques. Single-plane left ventriculograms were performed in the right anterior oblique projection. Three patients had biplane ventriculography. Three patients did not have ventriculograms (none of these patients had clinical manifestations suggesting left heart failure). Left ventricular volumes were measured by the modified area-length method of Sandler and Dodge (1968), using outlines of estimated end diastolic and end systolic cine frames.

The ejection fraction (left ventricular stroke volume divided by left ventricular end diastolic volume) was calculated for each ventriculogram. Large ventricles often were not all encompassed on a single cine frame. In these cases, ventricular outlines were drawn by using a composite of end systolic and end diastolic frames from several cardiac cycles. This method is probably less precise than when the ventricle can be outlined on single frames and for this reason absolute volumes are not reported. Five of the ventriculograms in which several cardiac cycles were used to complete end diastolic and end systolic outlines were selected at random and shown in blinded fashion to the same observer (R.E.D.) more than 18 months after his initial assessment. The difference in calculated ejection fraction between these two sets of observations averaged 0.03 (range 0.01 to 0.06). The same ventriculograms were also shown in blinded fashion to an independent observer. The difference between ejection fractions calculated from this set of

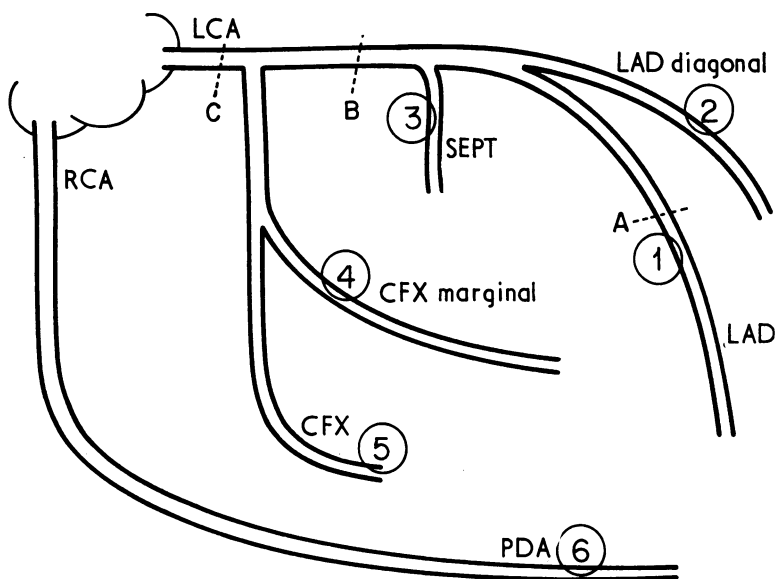


Fig. 1 The jeopardy score system as a semiquantitative measure of extent of left ventricular involvement by coronary artery disease. The coronary circulation is considered as six arterial segments as numbered in the figure. Each segment involved, directly or indirectly (by a lesion proximal to its origin), counts two points. For example, lesions located at points A, B, or C in the figure would give jeopardy scores of 2, 6, and 10, respectively. Also see text. RCA, right coronary artery; LCA, left coronary artery; LAD diagonal, major diagonal branch of left anterior descending artery; SEPT, major septal perforating artery; LAD, left anterior descending artery beyond the major diagonal and septal branches; CFX marginal, major marginal branch of the left

circumflex artery; CFX, left circumflex artery beyond the major marginal branch; PDA, posterior descending coronary artery.

observations compared with the initial observations averaged 0.02 (range 0.00 to 0.06).

For purposes of this study the coronary circulation was divided into 6 arterial segments. The arteriograms were assigned an arteriographic score, which we have called the 'jeopardy score', designed to express the number of segments jeopardised by significant proximal stenoses. The term 'proximal' is defined as that part of the coronary artery large enough to be bypassed by a venous bypass graft. By 'distal' is meant smaller than a size potentially able to accept connection of a venous bypass graft (estimated diameter less than 1 to 1.5 mm). An artery was considered significantly stenosed if more than 70 per cent of the estimated luminal area was obstructed. In the jeopardy score system, stenoses were not differentiated from complete occlusions (though complete occlusions were enumerated separately). Fig. 1 illustrates the 6 arterial segments and shows the principle of the jeopardy score. For example, if there had been a normal coronary arteriogram in the series, it would have been assigned a jeopardy score of zero. A patient with a lesion involving only 1 of the 6 segments was arbitrarily assigned 2 jeopardy score points. If the lesion were in a parent vessel proximal to more than 1 of the 6 designated segments, the arteriogram was assigned 2 points for each segment indirectly jeopardised. Patients with disease affecting all of the segments, either

directly or indirectly, had a jeopardy score of 12 points. In patients with left coronary arterial dominance, the 2 points assigned to the posterior descending artery were considered in the circumflex system, rather than in the right coronary arterial system.

Distal disease was considered separately according to the total number of segments significantly narrowed in their distal extent. Vessels counted as being distal included arterial branches other than the 6 segments designated in the jeopardy score system as well as the distal third (approximately) of these 6 arteries.

Collateral circulation was noted, where collaterals were counted in terms of the number of sources filling other segments in retrograde fashion. For example, if the left anterior descending artery beyond a proximal occlusion filled both from branches of the circumflex artery as well as from the right coronary artery, the arteriogram was credited as showing 2 collateral sources.

DEFINITION OF CARDIOMYOPATHIC SYNDROME DUE TO CORONARY ARTERY DISEASE

We have intended this term to refer to patients with coronary disease who have heart failure caused by multiple and widespread abnormalities of left ventricular wall motion severe enough to have resulted in a significant reduction of resting left ventricular ejection fraction. All patients classified

Table 1 *Criteria for heart failure (30 patients)*

Clinical features	No. of patients
Symptoms of dyspnoea	24
Gallop sounds (S4 and/or S3)	26
Electrocardiographic left atrial enlargement	20
Chronic digitalis and diuretic requirements	25
Raised left ventricular end diastolic pressure at time of catheterisation*	17
Left ventricular enlargement (chest film)	20

*Post-A wave end diastolic pressure > 15 mmHg.

as having it had a left ventricular ejection fraction ≤ 0.48 . Heart failure was defined as being present when a patient had radiological evidence of left heart failure, had had a raised mean jugular venous pressure, or had 2 of the 6 clinical features listed in Table 1. Other causes of heart failure due to coronary artery disease (see Discussion) were specifically excluded before a diagnosis of cardiomyopathy due to coronary artery disease was made.

STATISTICAL ANALYSIS

All statistical analyses were done by either the Student's *t* test or by χ^2 analysis with Yates' correction.

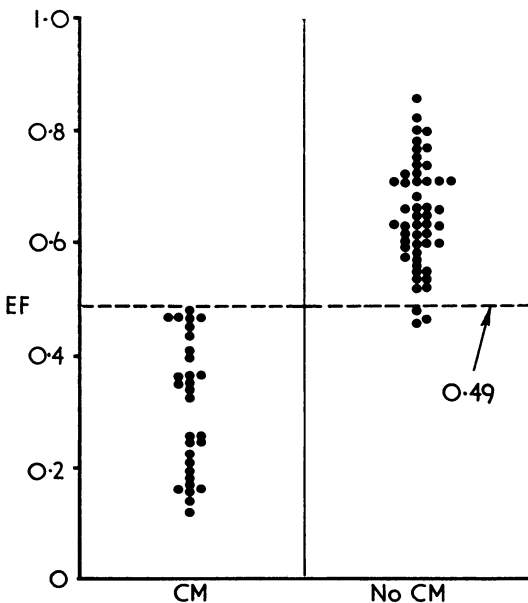


Fig. 2 The study population is shown according to ejection fraction (EF) and presence or absence of cardiomyopathy (CM).

Results

Of the 84 patients, 30 were classified as having the cardiomyopathic syndrome. The mean ejection fraction was 0.31 in this group and 0.63 in the group without cardiomyopathy. The highest ejection fraction encountered in a patient we believed to have clinical features related to left ventricular function impairment was 0.48. The total group of 84 patients, classified as having or not having cardiomyopathy, with 0.49 used as the 'upper cutoff' ejection fraction in the definition of cardiomyopathy, is shown in Fig. 2. Only 3 patients not classified as having cardiomyopathy had an ejection fraction below 0.48, and the lowest ejection fraction of these was 0.44; none of these patients had any of the clinical features shown in Table 1. Thus, there was very little overlap in ejection fraction between the group with cardiomyopathy compared with the group without it. This was so even though most of the patients had ventriculograms performed in only a single plane. There is, on average, a variation of 0.08 in ejection fractions calculated from single plane, compared with biplane, ventriculograms in such patients (Cohn *et al.*, 1974a).

The cardiomyopathy group included all patients in the study who at any time had had documented evidence of heart failure, as well as all patients having 2 or more of the 6 clinical stigmata of chronic heart failure shown in Table 1. There were 4 patients with isolated ventricular aneurysms, but none of these was large enough to cause the clinical features of heart failure or significant reduction in ejection fraction. There were no patients with heart failure judged to be due to relatively isolated mitral regurgitation or who had episodic pulmonary oedema thought to be secondary to recurrent, reversible ischaemia. There were no patients with rupture of the ventricular septum. One-third (10/30) of the patients with cardiomyopathy did not have increased heart size on plain chest films. This finding is in agreement with the recent study of Yatteau *et al.* (1974).

There was a much higher arteriographic jeopardy score in patients with cardiomyopathy (10.7 ± 0.4)¹ compared with those without it (5.3 ± 0.6) ($P < 0.01$) (Fig. 3). Every patient with cardiomyopathy had a jeopardy score of 8 or greater. Also, on average there were a larger number of completely occluded proximal vessels in patients with cardiomyopathy (1.4 ± 0.2), compared with those without it (0.8 ± 0.1) ($P < 0.002$). It is of interest that each patient with cardiomyopathy who had a jeopardy score of 8 had at least one completely occluded proximal vessel, whereas 5 patients with this syndrome and

¹Throughout the text, \pm refers to standard error of the mean.

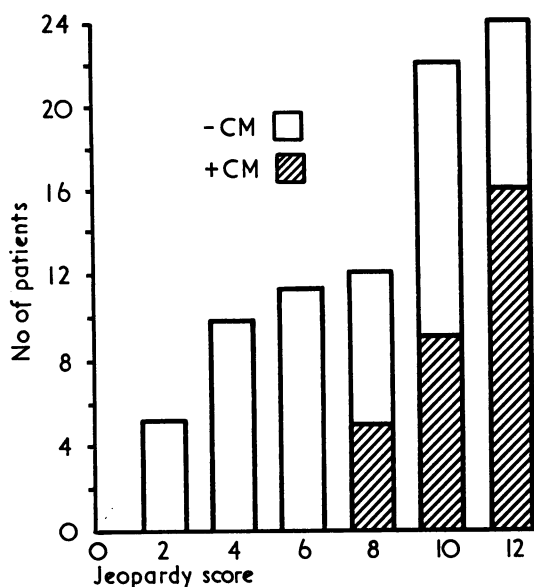


Fig. 3 The study population shown as numbers of patients with a given jeopardy score. It can be seen that all patients with cardiomyopathy (CM) had a jeopardy score ≥ 8 .

a jeopardy score of 10 or 12 did not have any completely occluded vessels. Neither the presence of collateral vessels (0.8 ± 0.2 vs 0.6 ± 0.2 collateral sources in patients with and without cardiomyopathy), nor the extent of distal disease (0.5 ± 0.2 vs 0.4 ± 0.2 diseased distal vessels in patients with and without cardiomyopathy) was related to the occurrence of cardiomyopathy.

Cardiomyopathy was strongly associated with the occurrence of multiple myocardial infarcts. When transmural and non-transmural myocardial infarcts were combined, there were an average of 1.9 ± 0.3 myocardial infarcts per patient in the group with cardiomyopathy and 0.9 ± 0.2 myocardial infarcts per patient in the group without it. Nineteen of 30 (64%) patients with cardiomyo-

pathy had ≥ 2 myocardial infarcts, whereas only 11 of 54 (20%) without it had ≥ 2 myocardial infarcts ($P < 0.001$). This finding is even more striking when transmural myocardial infarcts are considered separately; 14 of 30 (47%) patients with cardiomyopathy and only 4 of 54 (9%) without it had ≥ 2 transmural myocardial infarcts ($P < 0.001$).

Discussion

It is important when examining the correlation between clinical heart failure and angiographic data that heart failure be defined as precisely as possible. Yet in retrospective studies of the present type, just as in the practice of clinical cardiology, radiological evidence of left heart failure or clinical evidence of right heart failure is often not available in patients who are none the less appropriately considered as having had heart failure in the past. This is so because chest x-rays were not always done when the patient was symptomatic, because the patient improved after the institution of therapy, or because left atrial hypertension reached symptomatic levels only with effort and was not present in enough degree at rest to be evident on chest films. To counter this problem we have taken a probability approach similar to that which is taken in clinical practice. Not only were patients counted as having heart failure who had had documented radiological evidence, but also those who had a constellation of other features of varying specificity (Table 1). Of the 6 clinical features shown in Table 1, 2 were required for the diagnosis of heart failure because these features considered in isolation lack specificity. The specificity of each of these 6 features considered individually for designating a diagnosis of heart failure in the present study is shown in Table 2. Not surprisingly, raised left ventricular end diastolic pressure had the highest specificity, but the lowest sensitivity. The most sensitive feature, but by far the least specific, was the presence of gallop sounds. This relative lack of specificity was accounted for entirely by fourth heart sounds; every patient with a third heart sound was counted as having heart failure. The individual specificity of the other features ranged from 0.69 to 0.83; these values seemed acceptably high and consistent with everyday clinical experience.

All patients with evidence of heart failure in the present series of 84 patients with coronary artery disease had diffuse abnormalities of left ventricular wall motion severe enough to reduce significantly the left ventricular ejection fraction at rest, a syndrome we refer to as 'the cardiomyopathic syndrome due to coronary artery disease'. We have used this designation in part because it bears a

Table 2 Specificity of criteria for heart failure

Feature	No. of patients with feature who were counted as having heart failure	
	Total No. of patients with feature	
Dyspnoea	24/31	0.77
Gallop sounds	26/60	0.44
Electrocardiographic left atrial enlargement	20/26	0.77
Digitalis and diuretic	25/36	0.69
End diastolic pressure	17/19	0.90
Left ventricular enlargement	20/24	0.83

ventriculographic resemblance to primary myocardial disease. More importantly, we wish to distinguish it from other causes of chronic heart failure in coronary artery disease: isolated ventricular aneurysm, chronic mitral regurgitation (those cases where the left ventricular ejection fraction is relatively preserved), rupture of the interventricular septum, and cases where episodic pulmonary oedema is secondary to reversible ischaemia (Brody and Criley, 1970). Distinguishing these syndromes is important not only because they may differ in their relation to the extent of coronary disease, but also because they may differ in their clinical and therapeutic implications. That the cardiomyopathic syndrome was the only cause of heart failure present in this series may have been partly fortuitous, but it also reflects the fact that it is the most common of these syndromes.

Our results show that the occurrence of cardiomyopathy is clearly related to the angiographic extent of coronary artery disease. Not only was there a highly significant difference in mean jeopardy score between patients with and without cardiomyopathy, there was also a sharp cutoff in jeopardy score (8) below which it did not occur (Fig. 3).

The same conclusion would have been reached had the 1, 2, 3-vessel disease notation been used to score the coronary arteriograms: of 15 patients with single-vessel disease, none had cardiomyopathy; of 19 patients with 2-vessel disease, 3 had it; and, of 50 patients with 3-vessel disease, 27 had it. Further analysis of these data, however, shows the jeopardy score system to be more useful than the 1, 2, 3-vessel disease notation for predicting the presence or absence of cardiomyopathy. The predictive advantage of the jeopardy score is seen mainly in the mid-range of the two systems; a jeopardy score of less than 8 predicted more patients not at risk for cardiomyopathy (26 patients) than did 1-vessel disease (15 patients), and a jeopardy score of 8 or greater was more specific for predicting the presence of cardiomyopathy (30 of 58 had it) than was 2- and 3-vessel disease (30 of 69 had it). It is to be noted that the degree of stenosis is evaluated in all-or-none fashion, i.e. judged to be haemodynamically significant or not, in both systems. In the interest of simplicity this seems justifiable since there appears from recent studies to be an element of interobserver variability in judging degree of stenosis (Zir *et al.*, 1976). The superiority of the jeopardy score is presumably because it more precisely relates the extent of coronary disease to the extent of left ventricle involved, which is accomplished by assigning values to lesions according to their location relative to major branch points of the involved artery.

Saltups *et al.* (1971) have used a similar system in evaluating the electrocardiographic and haemodynamic consequences of exercise in patients with coronary artery disease.

The finding that the cardiomyopathic syndrome is specifically related to the extent of coronary disease supports the finding of Proudfit *et al.* (1968) that chronic heart failure in coronary artery disease is always associated with severe 2- or 3-vessel disease if patients with ventricular aneurysms, severe mitral regurgitation, or extracardiac causes of heart failure are excluded. The relation of clinical heart failure or its haemodynamic correlates to the angiographic extent of coronary disease has not been apparent in studies which have failed to distinguish the cardiomyopathic syndrome from other causes of chronic heart failure in coronary artery disease (Herman *et al.*, 1967; Baxley *et al.*, 1971; Cohn *et al.*, 1974b). This is explained by the fact that other heart failure syndromes are related in a less specific way to the coronary arteriogram than is cardiomyopathy due to coronary artery disease. For example, patients with ventricular aneurysms, even when the aneurysm is large, commonly have jeopardy scores of 6 and below (Lee *et al.*, 1976). Similarly, some of the patients with mitral regurgitation and heart failure in the study of Baxley *et al.* (1971) had single-vessel coronary artery disease.

Of 58 patients in the present study with jeopardy scores of 8 or more, 30 had cardiomyopathy and 28 did not. Thus, extensive coronary artery disease is necessary but not always sufficient for the development of cardiomyopathy. We do not know why some patients with high jeopardy scores develop multiple myocardial infarctions and other patients with high jeopardy scores do not. Despite more than three decades of study, beginning with the pioneering contributions of Blumgart *et al.* (1940), it is not known why the same degree of arterial obstruction results in myocardial infarction in one patient and not in another. Nevertheless, our data strongly suggest that extensive coronary disease places patients at risk for the development of cardiomyopathy by virtue of being a prerequisite to multiple myocardial infarcts. There were on average twice as many myocardial infarcts in patients with, compared with those without, cardiomyopathy. This finding is in accord with the recent study of Yatteau *et al.* (1974) and is consistent with the observation (Hamilton *et al.*, 1972; Amsterdam, 1973; Hamby *et al.*, 1974) that significant depression of the resting left ventricular ejection fraction in coronary disease virtually always implies previous infarction. It is for this reason that we prefer the designation 'cardiomyopathic syndrome due to coronary artery disease'

to the designation 'ischaemic cardiomyopathy' (Burch *et al.*, 1970), which carries the connotation, inherent in the word 'ischaemia', of reversibility. It might be argued that patients with this cardiomyopathic syndrome do not invariably have infarcts which can be historically or electrocardiographically documented (Raftery *et al.*, 1969), and, in fact, this was true of 1 of the 30 patients with cardiomyopathy in the present study. It is probable, however, that all patients with cardiomyopathy have infarcts, albeit non-transmural and clinically silent in a few. The high correlation of the cardiomyopathic syndrome with multiple infarcts explains why several studies have found that such patients, at least those with the most severely impaired left ventricular function (ejection fraction less than 0.25), do not appear to benefit from revascularisation procedures (Yatteau *et al.*, 1974; Kouchoukos *et al.*, 1972).

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