The Role of Collateral Circulation in the Various Coronary Syndromes

Valentin Fuster, M.D., Robert L. Frye, M.D., Margaret A. Kennedy, B.S., Daniel C. Connolly, M.D., and Harold T. Mankin, M.D.

SUMMARY Coronary collaterals were evaluated at arteriography within 1 year of the onset of symptoms of coronary disease in 73 patients with transmural myocardial infarction (TMI), 63 patients with subendocardial myocardial infarction (SMI), and 164 patients with angina pectoris (AP) alone. An occluded artery was present in 79% of patients with TMI, 70% of patients with SMI and elevated serum enzymes, and 42% of patients with AP. Collateral vessels supplied the occluded artery in 91% of patients with AP, 93% of patients with SMI — all had postinfarction AP — and 78% of patients with TMI and postinfarction AP, but in only 35% of patients with TMI and no postinfarction AP (p<0.01). With the treadmill stress test in patients with AP alone and coronary lesions in the anterior wall coronary distribution, 97% had ischemia in leads V<sub>5</sub> to V<sub>6</sub>. Only 25% of patients with coronary lesions in the inferior wall coronary distribution had ischemia in leads III and aV<sub>R</sub> (positive group) and 75% did not (false negative group). The affected artery was occluded and supplied by collaterals in only 9% of patients of the positive group, but in 57% of the false negative group (p<0.01).

These data and additional findings in the infarction syndromes suggest that in TMI and SMI the presence of collaterals maintains a peri-infarction ischemic zone with subsequent AP. In patients with AP alone, the treadmill stress test is highly predictive of coronary disease in the anterior but not in the inferior wall coronary distribution, in part because of the influence of collaterals.

THE CLINICAL ROLE of angiographically demonstrable collateral circulation in patients with coronary artery disease has been a subject of controversy for many years. There are studies suggesting that coronary collaterals are of no significant importance; however, recent experimental and clinical studies support the importance of coronary collaterals in preserving myocardial function. One reason for this controversy is that in most studies, collateral circulation has not been analyzed independently in the three well-defined coronary syndromes: transmural myocardial infarction, subendocardial myocardial infarction and angina pectoris.

Accordingly, our purpose has been: 1) to document the clinical-arteriographic correlations of the three specific coronary syndromes at an early stage — within 1 year of the initial manifestation of coronary artery disease; 2) to determine whether regional collateral circulation, by supplying the obstructed coronary artery, might play a role in determining the clinical syndromes that follow a transmural or subendocardial myocardial infarction; and 3) to determine whether regional collateral circulation might play a role in determining the presence and location of electrocardiographic ischemia during the submaximal treadmill stress test in patients with angina pectoris alone.

Patients and Methods

Clinical Groups

The study group comprised 300 patients (265 men and 35 women) who were seen consecutively at the Mayo Clinic in a 5-year period and who had had an arteriographic study of satisfactory quality within 1 year (mean 23 weeks) of the onset of typical symptoms of coronary artery disease. This strict inclusion criterion requiring a short interval between the onset of symptoms and the arteriographic study explains the limited number of patients entered in the study. Patients who had congenital or rheumatic heart disease were not included. Five patients with classic angina pectoris and one patient with transmural myocardial infarction who had normal coronary arteriograms were also excluded from the study. All patients' histories were taken by at least three physicians, and the clinical records were carefully examined in retrospect by one of us who had no knowledge of the arteriographic findings. Additional information, when needed, was obtained from letters and telephone calls to the referring physicians. The patients were divided into three clinical groups, as follows.

Transmural Myocardial Infarction

The 73 patients in this group had clinical histories suggestive of acute myocardial infarction, definite QRS changes in the ECG (fulfilling the criteria of the Coronary Drug Project Research Group) and diagnostic increases in the serum enzymes, creatine phosphokinase and glutamic-oxaloacetic transaminase. Only patients with strictly anterior (43 patients) or inferior (30 patients) myocardial infarction were included in the study. Patients with suspected posterobasal or high lateral wall infarction were excluded because of the uncertainty of the electrocardiographic diagnosis of these conditions. The ECGs in all patients in the study were reviewed in retrospect by one of us who had no knowledge of the arteriographic findings. About two-thirds of the patients experienced angina pectoris soon after the infarction, which was
the indication for coronary arteriography; most of the remaining patients without subsequent angina pectoris had angiography because of persistent left ventricular failure.

Subendocardial Myocardial Infarction

The 63 patients in this group had typical histories of acute myocardial infarction and ST-T changes only in the ECG (fulfilling the criteria of the Coronary Drug Project Research Group\textsuperscript{17}) which persisted for more than 48 hours. Only patients with strictly anterior (33 patients) or inferior (30 patients) subendocardial infarction were included in the study. These patients were divided into two subgroups: one group (32%) had a diagnostic increase in creatine phosphokinase and glutamic-oxaloacetic transaminase at the time of the infarction and the other group (68%) did not. Some of the latter patients might be classified by the criteria of other authors as having an intermediate coronary syndrome.\textsuperscript{19} Most patients (87%) had angina pectoris soon after the subendocardial infarction and before the arteriographic study.

Angina Pectoris Alone

This group comprised 164 patients in whom substernal or precordial pain or discomfort was typically precipitated by walking (and relieved within 15 minutes by rest), stress or excitement. None of the patients in this group had evidence of transmural or subendocardial myocardial infarction.

Treadmill Stress Test

Sixty-three patients with angina pectoris alone had a treadmill stress test near the time of angiography. All of these patients were chosen for our analysis because of their normal resting ECGs, which permitted an accurate evaluation of the electrocardiographic changes during the test. The other patients with angina pectoris or myocardial infarction who had a treadmill stress test all had abnormal resting ECGs, and were therefore excluded from the analysis. The test was performed initially for diagnostic purposes and was reviewed later by one of us. If there was a disagreement between the two readings, the final interpretation was done by another author. The test consisted of having the patient walk at a 10% grade starting at 1.5 mph and increasing the speed by 0.5 mph every minute. After 5 minutes, the patient continued walking at 3.5 mph but the grade was then increased by 4% every minute until the patient had reached 85% of the predicted maximum heart rate. At this point, the exercise load was maintained constant and the patient was exercised for 3 more minutes. The appearance of chest pain associated with positive electrocardiographic changes of ischemia was indication for discontinuing the test.

The test was considered positive for ischemia when one of the following criteria had been met: 1) ST-T segment depression > 1.0 mm below the resting level — with the ST segment extending horizontally or sloping downward — which persisted for at least 0.08 second; 2) ST-segment-elevation > 1.0 mm above the resting level. We noted whether the ST changes occurred in the inferior leads, in the anterior leads, or in both. Continuous recording of the 12-lead ECG was monitored with an oscilloscope throughout the exercise and for at least 9 minutes after exercise. Electrocardiographic strips at a paper speed of 25 mm/sec were taken every minute. All patients who had a positive exercise test for ischemia experienced typical angina during the exercise. This is not surprising, since we studied patients with typical angina and normal resting ECGs, which makes the appearance and interpretation of symptoms and ischemic electrocardiographic changes very accurate.

Coronary Arteriography and Left Ventricular Angiography

Selective coronary cineangiography and left ventriculography were performed in all patients by the Sones and Shirey\textsuperscript{20} technique. The angiograms were viewed initially for diagnostic purposes and reviewed later by one of us who had no knowledge of the patients' clinical or hemodynamic data. The study sequence at catheterization was as follows: 1) left ventricular pressure determination and uniplane left ventriculography; 2) administration of sublingual isosorbide dinitrate (Isordial) 5 mg; and 3) selective coronary cineangiography.

Left ventriculograms were made in 45° right anterior oblique projections. Meglumine diatrizoate (Renografin-76), 0.7 ml/kg, was injected over 3–4 seconds during deep inspiration. The left ventricular anterior surface (which included the anterobasal, anterolateral, and apical segments) and the inferior surface (which included the diaphragmatic and posterobasal segments) were both analyzed for the distinct types of abnormal contraction as described by Herman et al.\textsuperscript{21}

Coronary cineangiography was obtained by the selective injection of 1–3 ml of Renografin-76 at both coronary orifices. Right and left oblique and left lateral projections were studied, and the degree of obstruction in a vessel was derived from estimates of the width of the respective vessel in its normal portion compared with that of its most diseased portion, the latter being expressed as a percentage of the former. Obstruction of at least 50% of one or more of the four major arteries — right coronary, left anterior descending, left circumflex, and left main coronary artery — or of their main branches was considered. For the purpose of the study, two degrees of obstruction were defined; 1) 50% to subtotal and 2) occlusion. The designation of coronary disease in the inferior wall coronary distribution was made when obstruction occurred in a dominant right coronary, in a dominant left circumflex artery, or in either one if it was a balanced circulation; designation of coronary disease in the anterior wall coronary distribution was made when obstruction occurred in the left anterior descending coronary, a nondominant left circumflex, or the left main coronary artery. Collaterals were defined as
the distinct arteriographic visualization of accessory vessels connecting either two separate arteries — intercoronary collaterals — or a segment of a coronary artery proximal to an obstruction — intracoronary or bridging collaterals. In our patients, 91% of the coronary arteries supplied by collaterals were totally occluded, and the rest (9%) were more than 90% stenotic. For this reason, in our study we paid the greatest attention to the presence or absence of collaterals in patients with coronary occlusion.

Results

Transmural Myocardial Infarction

In this group, more than three-fourths (79%) had coronary artery occlusion (54% had one occlusion, 25% had two occlusions). The remaining patients (21%) had stenotic lesions without occlusions (fig. 1). In 78% of the patients with coronary occlusion in whom angina pectoris was subsequently present, the occluded vessel had apparent collateral supply (fig. 2).

In one-third of the patients (35%) without subsequent angina pectoris, however, the occluded vessel had an apparent collateral supply; the remaining two-thirds (65%) did not (p < 0.01). Moreover, all patients without subsequent angina pectoris and no collaterals had very large infarcts, as judged by the presence of ventricular aneurysm or a large akinetic zone, a high left ventricular end-diastolic pressure, and clinical evidence of left ventricular failure. There was no statistically significant difference in the number of vessels diseased (table 1) and the degree of vessel obstruction (table 2) between the groups with or without subsequent angina pectoris. Thus, collateral supply of the occluded coronary artery was a significant finding in patients with transmural myocardial infarction and postinfarction angina.

Subendocardial Myocardial Infarction

In this group, nearly three-fourths (70%) of the patients who had a diagnostic increase in serum enzyme levels had a coronary occlusion (60% had one occlusion, 10% had two occlusions); of the patients who did not have an increase in enzyme levels, more than one-third (37%) had a coronary occlusion (35% had one occlusion, 2% had two occlusions) (fig. 1). All patients with coronary occlusion experienced subsequent angina pectoris and the occluded vessel had apparent collateral supply in 93%. Thus, collateral supply of the occluded coronary artery was a significant finding in patients with subendocardial myocardial infarction and postinfarction angina.

Angina Pectoris Alone

In this group, more than one-third (42%) had coronary occlusion (28% had one occlusion, 14% had two

FIGURE 1. Arteriographically demonstrable vessels obstructed and occluded in relation to the various coronary syndromes. Results are expressed as percentage of patients with each clinical syndrome. AP = angina pectoris; SMI = subendocardial myocardial infarction; TMI = transmural myocardial infarction.

FIGURE 2. Transmural myocardial infarction. Correlation between absence or presence of collaterals supplying the occluded artery in patients who did not have angina pectoris after the infarction (left) and in patients who did have subsequent angina (right).
occlusions) (fig. 1). In 91% of all patients with coronary occlusion and angina pectoris alone, the occluded vessel had apparent collateral supply.

On the treadmill stress test, 97% of the patients with angina pectoris and coronary disease in the anterior wall coronary distribution had electrocardiographic changes of ischemia in the anterior leads V₅, V₆, or V₄ (fig. 3); these results were not statistically related to the presence or absence of occlusions and collateral supply of the diseased coronary artery (table 3). About 25% of the patients with coronary disease in the inferior wall coronary distribution had electrocardiographic changes of ischemia in the inferior leads III and aVF. Thus, the electrocardiographic detection of ischemia was frequent in anterior wall coronary disease, but not in obstructive lesions involving the inferior wall coronary distribution.

On analysis of collateral circulation, 91% of the patients with coronary lesions in the inferior wall coronary distribution who had no electrocardiographic changes of ischemia in the inferior leads (negative group), the artery was occluded and had collateral supply (fig. 4). Moreover, from the remaining patients (43%), most (36%) had no more than mild-to-moderate disease of the right coronary system (<75% obstruction), which is not usually associated with electrocardiographic changes during exercise. The relationship between collateral supply and electrocardiographic changes of ischemia in the inferior leads was very significant (p < 0.01).

Most patients (90%) with a coronary occlusion in the inferior wall coronary distribution had collateral supply (table 3) and, as mentioned, most of them were free of inferior wall ischemia during exercise. In contrast, only about half of the patients (53%) with a coronary occlusion in the anterior wall coronary distribution had collateral supply (table 3) and, moreover, none of these were free of anterior wall ischemia during exercise. Thus, the occluded vessels in the inferior wall coronary distribution appear to be frequently supplied by collaterals, which also seem to be more effective than the occluded vessels in the anterior wall coronary distribution.

![Figure 3. Treadmill exercise test in patients with angina pectoris. Correlation between the location of coronary lesions in the anterior wall coronary (AWC) distribution and the presence or absence of electrocardiographic changes due to ischemia in leads V₄ to V₆ (right). Correlation between the location of coronary lesions in the inferior wall coronary (IWC) distribution and the presence or absence of electrocardiographic changes due to ischemia in leads III and aVF (left).](image-url)
Table 3. Coronary Occlusion and Collateral Vessels in Patients with Angina Pectoris Who Had a Treadmill Stress Test

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<th>No. of patients with coronary occlusion</th>
<th>Collateral supply</th>
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<td></td>
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<td>Yes (%)</td>
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<tr>
<td>Anterior wall coronary lesion (60 pts)</td>
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<td>10 (53)</td>
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<tr>
<td>Inferior wall coronary lesion (39 pts)</td>
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<td>15 (90)</td>
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*All had anterior wall ischemia during exercise. †Fifteen were free of inferior wall ischemia during exercise.

Discussion

The present study was stimulated by the striking controversy in the literature regarding the role of collateral circulation in patients with coronary artery disease. In reviewing the literature and our case material, it became apparent that one reason for this controversy was that in most studies, collateral circulation was not analyzed in well-defined coronary syndromes.

The present large series of patients differs from other series in that we selected only patients who had had an arteriographic study at an early stage — within 1 year — of the onset of typical symptoms of coronary artery disease. This criterion allowed us to determine the initial manifestations of the disease accurately and to classify the three different coronary syndromes — transmural myocardial infarction, subendocardial myocardial infarction, and angina pectoris — clinically and angiographically. Thus, we were able to analyze the relationship between regional collateral circulation, defined at arteriography, and some of the clinical and electrocardiographic features of the three syndromes.

In the assessment of collateral vessels, three problems have to be discussed. First, it is important to consider the possible limitations in trying to establish a correlation between the extent of collateral vessels visualized angiographically and the amount of blood flow conveyed by these vessels. Nevertheless, recent clinical evidence indicates that there is a good correlation between preoperatively quantified angiographic appearance and subsequent intraoperatively measured collateral flow in patients undergoing coronary artery bypass surgery. Such correlation was maximal when collaterals and the distal coronary segment were clearly visualized, as was required in our study. Moreover, in our analysis we considered not only the intercoronary collaterals but also the intracoronary or bridging collaterals, which appear to be functionally important.

Second, we must consider the effect of nitroglycerin, which was given routinely in all patients before angiography. Since nitroglycerin can reduce resistance to collateral flow, its role in the visualization of collaterals may be significant. However, the responsiveness to nitroglycerin, as evaluated by percent reduction in coronary collateral resistance, has not been correlated with the angiographic appearance of collaterals. Moreover, any effects on collateral visualization would apply to all patients studied.

Third, patient selection must be considered because cardiac catheterization was indicated in all of our patients for some specific reason. Thus, we do not yet have information on collateral circulation in patients with subendocardial myocardial infarctions who are free of postinfarction angina. However, this is not only because these patients were not catheterized but, most important, because they are unusual. Thus, in our recent prospective study, only one-fourth of patients (24%) with subendocardial infarction were free of angina after the infarct. In transmural myocardial infarction, our two groups — patients with and without postinfarction angina — were large enough for data analysis.

**Figure 4.** Treadmill exercise test in patients with angina pectoris and coronary lesions in the inferior wall coronary distribution. Correlation between electrocardiographic ischemia in leads III and aVF and the presence or absence of collaterals supplying a coronary obstruction (left). Correlation between the lack of electrocardiographic changes caused by ischemia in leads III and aVF and the presence or absence of collaterals supplying a coronary obstruction (right).
analysis. However, the group without postinfarction angina had mostly large infarcts with signs of left ventricular failure and was, therefore, a selective group. Thus, we do not yet have information on collateral circulation in patients with transmural myocardial infarction who have neither postinfarction angina nor left ventricular failure because these patients were not catheterized.

Coronary Collaterals and Transmural Myocardial Infarction

The frequency of total occlusion of at least one major artery was much higher in patients with transmural myocardial infarction than in patients with angina pectoris. This finding, together with the observation that arterial occlusions in patients with transmural myocardial infarction were proximal to the location of the infarct as defined by ventriculography or electrocardiography, supports the thesis that a major cause of myocardial infarction is coronary occlusion.

A striking finding, seen only in the group of patients who experienced angina after the infarction, was the high frequency of collateral vessels supplying the occluded artery proximal to the infarct. A reasonable explanation might be that the number of vessels diseased and the degree of vessel obstruction were more severe in the group with postinfarction angina than in the group without postinfarction angina. However, there was no statistical difference in the number of diseased vessels or in the degree of obstruction between both groups of patients. Another definitive explanation for our findings might be that when collateral vessels are present, they supply the peri-infarction region, preventing extension of necrosis but maintaining an ischemic zone manifested by postinfarction angina. Thus, as suggested by other clinical investigators,5, 9, 10, 12, 17, 20, 29, 30, 31, 32, 33 collaterals prevent the extension of necrosis. This explanation is also supported by our finding that all of our patients without postinfarction angina and no collateral supply had large infarcts. Moreover, as demonstrated experimentally,31, 33 collaterals appear to be insufficient to prevent a peri-infarction ischemic zone, the presence of which is only apparent during exercise. Accordingly, the presence of collaterals may explain the syndrome of postinfarction exertional angina as seen in our patients. In further support of this explanation, there is now clinical and arteriographic evidence that the fate of the peri-infarction zone, also referred to as "twilight zone"32 or "zone of marginal perfusion,"34 depends mainly on collateral circulation.35

Coronary Collaterals and Subendocardial Myocardial Infarction

Little information is available on the arteriographic, electrocardiographic and ventriculographic correlations in patients with subendocardial myocardial infarction. In our patients who had subendocardial myocardial infarction without serum enzyme elevation, the frequency of total occlusion of at least one major artery was very low. It is reasonable to assume that the myocardial ischemic event occurred in these patients as a result of transient occlusion or impairment in blood flow that resulted only in a limited insult, which was insufficient to elevate the level of enzymes. Because of this limitation of the myocardial insult and also because the coronary anatomy was comparable to patients with angina pectoris alone, some authors might categorize these patients as having an intermediate coronary syndrome. However, we preferred to use the anatomical term subendocardial myocardial infarction, for the persistent electrocardiographic changes and the abnormal regional contractility at ventriculography (Fuster V, Frye RL: unpublished data) suggested to us that a small subendocardial infarction had occurred. By contrast, the incidence of arterial occlusion was very high in patients with subendocardial myocardial infarction and significant necrosis, as manifested by an elevation in the level of serum enzymes. This finding, together with the observation that the arterial occlusion was proximal to the location of the infarct as defined by ventriculography or electrocardiography, supports the thesis that coronary occlusion is a major cause of the subendocardial infarction in these patients.

It is known experimentally36, 37 that when an artery undergoing progressive obstruction suddenly becomes occluded, the subendocardium is the zone of the myocardium that is most vulnerable to the ischemia. It has been recognized, also experimentally,33, 6-38 that the basic mechanism includes the development of adequate collateral circulation to the subepicardial layers which, at the time of occlusion, is sufficient to protect this region from necrosis. These experimental data are now supported by our findings in man. In almost all patients with coronary occlusion and subendocardial infarction, the occluded vessel proximal to the infarcted zone had a collateral supply that presumably prevented the infarction from becoming transmural. This preventive effect does not contradict the high incidence of collaterals found in transmural myocardial infarction with postinfarction angina, since in these patients the infarct was also clinically and angiographically small (Fuster V, Frye RL: unpublished data).

As all of these patients had postinfarction angina, it is reasonable to think, as demonstrated experimentally31, 32 and postulated earlier, that these collaterals maintain a viable but ischemic peri-infarction zone manifested clinically as postinfarction angina. Our paradoxical findings suggesting that collateral circulation limits extension of necrosis at the expense of maintaining a "zone of marginal perfusion" are interpreted by us and by others as the most likely explanation not only for the high frequency of postinfarction angina, but also for the ventricular dysrhythmias so commonly found in these infarctions of limited size.28, 40

Coronary Collaterals and Angina Pectoris

More than one-third of our patients with angina pectoris had a coronary occlusion without clinical or electrocardiographic evidence of myocardial infarc-
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We postulate that these occlusions resulted from slowly progressive lesions which permitted the development of collateral vessels and thus prevented infarction. This concept is supported by our findings, for in almost all patients with angina and a coronary occlusion, the occluded vessel was supplied by collaterals. Similar results have been reported in a more limited number of patients, and this concept has also been suggested experimentally. 31, 32, 41-48

Our study demonstrated, as did the studies of others, 44-46 that most patients with coronary disease in the anterior wall coronary distribution had electrocardiographic changes of ischemia in the anterolateral leads during the treadmill test, but only one-fourth of the patients with coronary disease in the inferior wall coronary distribution had changes of ischemia in the inferior leads.

Because of the discrepancy between coronary disease in the inferior wall coronary distribution and the location of ischemia during exercise, we investigated whether regional collateral circulation might explain some of these findings. As expected, in most patients with coronary lesions in the inferior wall coronary distribution who had electrocardiographic changes of ischemia in the inferior leads, the diseased artery had no collateral supply. However, in most patients with coronary lesions in the inferior wall coronary distribution and without electrocardiographic changes of ischemia in the inferior leads, the affected artery had either a coronary occlusion with collateral supply or no more than mild-to-moderate disease. Therefore, it is reasonable to think that collateral supply of occluded arteries or the absence of severe lesions explained the lack of inferior wall myocardial ischemia during exercise in patients with coronary disease in the inferior wall coronary distribution.

Our clinical findings demonstrate that the occluded vessels in the inferior wall coronary distribution are more frequently (and probably more effectively) supplied by collaterals than the occluded vessels in the anterior wall coronary distribution; this has also been demonstrated in pigs, which have a coronary system very similar to that in man. 47 The effectiveness of collateral supply to vessels of the inferior wall coronary distribution appears to support the clinical impression that patients with isolated right coronary disease have infrequent clinical manifestations unless they experience sudden occlusion with myocardial infarction.

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The Quality of Resonance of the First Heart Sound After Myocardial Infarction: Clinical Significance

WILLIAM F. RENNER, M.D. AND GERARD W. RENNER, A.B.

SUMMARY  Frequency analyses of the first heart sound (S₁) were performed in 80 normal subjects and 80 postinfarction patients. A readily recognizable frequency pattern characterized by a quality of resonance ≥2, as measured by the Q factor at 3 db down, was noted in 78 of the 80 apparently normal subjects. An aberrant pattern with a Q < 2, often accompanied by a lowering of the frequency content, was found in 78 of 80 postinfarction patients. We propose that the quality of resonance of S₁ is a measure of the degree to which the structural homogeneity of the left ventricle as a compliant contractile unit has been preserved after myocardial infarction.

IN 1961, RUSHMER suggested that the first heart sound (S₁) results from the vibrations of the entire blood-filled heart as a dynamically coupled system.¹ In the intervening years, this “cardiohemic” concept has won increasing acceptance.² In 1970, Adolph et al. tested Rushmer’s hypothesis by performing frequency analyses of S₁ during isovolumic contraction.³ They found a consistent and reproducible pattern in 74 normal subjects and an aberrant pattern characterized by a greater voltage output at 30 Hz than at 40 Hz in 21 of 24 patients with acute myocardial infarction. A similar pattern was found in patients with healed infarcts, cardiomyopathy, and in highly trained athletes. Normal patterns were found in patients with rheumatic valvular disease, including four patients with Starr-Edwards prosthetic valves. They reasoned that the variations between normal and abnormal patterns could be explained in terms of myocardial...
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