An Electrocardiographic, Anatomic, and Metabolic Study of Zonal Myocardial Ischemia in Coronary Heart Disease

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SUMMARY
An integrated approach, utilizing cine coronary angiography, the standard 12-lead and post-exercise electrocardiograms, and regional myocardial lactate metabolism, is presented for detection of regional myocardial ischemia in patients with coronary heart disease. The normal electrocardiogram was of no predictive value and was present with extensive coronary disease and myocardial production of lactate. The abnormal electrocardiogram gave an accurate indication of a portion, but not all zones of ischemia. Multiple electrocardiographic abnormalities were invariably associated with severe coronary artery disease, although in most patients many more coronary lesions were present than electrocardiographic abnormalities. The regional lactate pattern was very helpful in localizing myocardial ischemia and significant coronary artery lesions. Regional lactate abnormalities may have a great practical value in the selection of patients for myocardial revascularization surgery and in their postoperative evaluation.

Additional Indexing Words:
Coronary arteriography
Exercise tests
Lactate production of myocardium
Surgery for coronary artery disease

With the recent success in appropriately transplanted internal mammary artery, assessment of zonal myocardial ischemia has become of prime importance in patients with angina pectoris and other findings of coronary heart disease. Such an evaluation is necessitated by the regional nature of ischemia resulting from coronary artery disease: the sites of coronary atherosclerosis are variable in number and degree; myocardial pathology is non-uniform, patchy, and localized. Electrocardiographic diagnosis of infarction or ischemia is possible, primarily because of this zonal nature of the disorder. The demonstration of localized metabolic abnormalities associated with ischemia in the myocardium would be helpful in the functional analysis of an individual with coronary heart disease.

Certain basic principles are established: Lactate production in the heart is evidence for excessive glycolysis and in coronary heart disease is most often due to ischemia, and as such can be used as a sensor of this process. Frequently a stress to cardiac energy metabolism, such as exercise or catecholamine infusion, is necessary to induce myocardial production of lactate in the presence of coronary lesions. Samples of coronary sinus blood can be obtained from the mouth of the anterior interventricular vein and from the region of efflux of the marginal and posterior interventricular veins, but not from the anterior...
cardiac (right ventricular) veins. Blood can be sampled from multiple coronary venous sites to examine venous effluent as more streams of flow are progressively added from anterior to posterior ventricular venous entries. Thus, with left anterior descending artery disease, one might expect a different coronary venous lactate pattern from that seen with an isolated right coronary artery lesion. This present study was carried out to seek the interrelationships between arterial lesions determined by coronary cineangiography, sites of abnormality in the electrocardiograms obtained during rest and after exercise, and regional myocardial lactate abnormalities identified by multiple-site coronary venous sampling.

**Methods**

From a much larger series of patients with cineangiographically documented coronary artery

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**Figure 1**

Schematic representation of right heart catheter through coronary sinus to mouth of anterior interventricular vein. Venous effluent sampled at this site represents the anterior and septal zones of the left ventricle. By withdrawal of the catheter, multiple coronary venous sites can be sampled to examine as more streams of flow are progressively added from anterior to posterior ventricular venous entries.

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**Figure 2**

Schematic representation of myocardial metabolism. Under steady state conditions, the heart is completely aerobic, utilizing free fatty acids, glucose, pyruvate, and lactate. The Krebs cycle is functioning within the mitochondria. The heart extracts lactate at rest and in an unchanged or greater amount during stress. Under ischemic conditions glycolysis occurs in the sarcoplasm and proceeds in excess of mitochondrial oxidation. This results in decreased extraction (<10%) or in gross production of lactate by the myocardium. DPNH = reduced dihydrophopyridine nucleotide.
Myocardial lactate patterns in the control group. Samples were taken at rest and from multiple sites along the coronary sinus during catecholamine stress. Note the fairly uniform pattern of extraction in all instances. Brackets indicate standard deviations.

Type of lactate abnormality seen in the group with coronary heart disease. The patients were classified as having anterior, lateral, posterior, or a combination of these abnormalities based on the lactate pattern found in the venous effluent from multiple site coronary venous sampling. LAD = left anterior descending artery; LCf = left circumflex artery; RCA = right coronary artery.
ZONAL MYOCARDIAL ISCHEMIA

position (fig. 1), multiple sites along the coronary sinus were sampled, and thus, venous drainage representing selected areas of the left ventricle was obtained. For example, with the catheter in the most distal portion of the great cardiac vein, sampling would represent the anterior and septal areas of the left ventricle; with the catheter tip in the proximal part of the coronary sinus, sampling would represent blood from the distal areas plus selectively the posterior-inferior drainage of the left ventricle. Although no definite proof exists at present, it is presumed that this would be the normal pattern of venous drainage and is consistent with previous anatomic and experimental descriptions. In all instances, the pathways of venous drainage were carefully followed during selective arteriography or venography to aid in corroboration, and a diagram was drawn to relate catheter position to venous pattern, particularly entry of tributary veins. The lactate sample was labeled as anterior, lateral, or posterior from study of the catheter position in relation to the venogram. Samples of arterial and coronary venous blood for lactate and pyruvate analysis were obtained during the resting state. During catecholamine stress, intravenous infusion of isoproterenol (1-5 µg/min), paired arterial and coronary venous lactate samples were obtained from two or three sites along the coronary sinus to elicit metabolic abnormalities unapparent at rest and to assess possible zonal aberrations as described above. Lactate and pyruvate were determined enzymatically in duplicate with an error between samples of less than 5%. Analysis of lactate data in each state and location was made by determining the percentage extraction or production across the myocardium: \( A-V/A \times 100 = \% \), where \( A = \) arterial and \( V = \) venous concentration of lactate in millimolars (mM). Under steady state conditions (fig. 2), the heart extracts lactate at rest (> 10%), and in an unchanged or greater percentage during stress.\(^4\) Markedly decreased extraction (<10%) or gross production of lactate was considered abnormal and to be consistent with ischemia.

Neither the calculation of myocardial excess lactate nor comparison of arterial and venous lactate/pyruvate ratios is presented. This is due to the problem inherent in accurate measurement of small arteriovenous pyruvate differences. Pyruvate is present in low concentration (about 0.1 mM) and tends to decarboxylate spontaneously.

| Figure 5 |

Interrelationship of coronary artery lesions and zones of ischemia as indicated by lactate metabolism and electrocardiographic abnormalities in 30 cases. Comparisons were made in terms of no agreement or agreement between the above-named parameters.

| Figure 6 |

Comparison of coronary artery lesions and electrocardiogram. Specific vessel lesions are compared with corresponding electrocardiographic abnormality (crosshatched area in bar = cases with electrocardiographic abnormality concordant with the known vessel lesion), and the specific electrocardiographic abnormality is compared with the anticipated vessel lesion (crosshatched area in bar = vessel lesion supplying an area predicted to be abnormal by the electrocardiogram). Note the almost complete agreement between an abnormal electrocardiographic finding and an anticipated vessel lesion. In this and subsequent illustrations LAD = left anterior descending artery; LCF = left circumflex artery; RCA = right coronary artery; Ao = aorta; PA = pulmonary artery; ASMI or ST-T = anteroseptal myocardial infarct or ST-T wave abnormalities; Ant-Lat MI or ST-T wave abnormalities = anterolateral myocardial infarct or ST-T wave abnormalities; Inf-Post MI or ST-T = inferoposterior myocardial infarct or ST-T wave abnormalities.
Results

Control Group

Lactate patterns in the control group of patients at rest and from multiple sites along the coronary sinus during catecholamine stress are shown in figure 3. A fairly uniform pattern of extraction in the normal range was seen in all samples.

Coronary Group

The types of lactate abnormality seen in this study are depicted in figure 4. The patients were arbitrarily grouped as having anterior, lateral, posterior, or a combination of abnormalities based on lactate pattern found in the venous effluent as illustrated. The interrelationships of the pattern of coronary artery lesion, electrocardiographic findings, and the pattern of regional lactate abnormalities are illustrated in figures 5 to 10.

Coronary Artery Lesions as Base Line

Electrocardiogram

More coronary artery lesions were found than electrocardiographic abnormalities (figs. 5 and 6). Less discrepancy was noted, however, the greater the number of vessels involved. In general, when an electrocardiographic abnormality was present, its location corresponded to the site of a coronary lesion.

Lactate Pattern

There were more coronary artery lesions than anticipated lactate abnormalities (figs. 5 and 6).
and 7). However, no discordant results were noted in that a zone of lactate abnormality did not appear in the absence of a corresponding coronary artery lesion in this series.

Zonal Lactate as Base Line

Coronary Lesions

The sites of lactate abnormality increased as the number of arterial lesions increased (fig. 8).

Electrocardiogram

More lactate abnormalities were found than electrocardiographic abnormalities (figs. 8 and 9). However, the more sites of lactate abnormality, the closer was the agreement with electrocardiographic findings. Posterior lactate abnormalities corresponded to fewer electrocardiographic abnormalities than other zones did (fig. 9).

Zonal ECG Abnormalities as Base Line (Figs. 6, 9, and 10)

Coronary Artery Lesions

Seven of 50 patients showed normal resting and post-exercise electrocardiograms in spite of significant coronary artery lesions (fig. 10). Those patients with abnormal exercise trac- ings showed complete agreement with expected sites of coronary artery disease, although more coronary artery lesions were found than electrocardiographic abnormalities. Single or multiple electrocardiographic zones of abnormality determined during rest agreed with an anticipated coronary vessel lesion in all patients (fig. 10).

Lactate Pattern

Three of the seven patients with normal resting and post-exercise electrocardiograms showed regional lactate abnormality (fig. 10). Most of the individuals with abnormal exercise electrocardiograms showed a corresponding abnormal regional lactate pattern. On the other hand, when the resting tracing contained a single regional electrocardiographic abnormality, such as an old myocardial infarction, fully one third of such patients did not have corresponding lactate abnormalities (fig. 10). Those patients with multiple abnormal electrocardiographic zones, however, displayed, as anticipated, multiple abnormal lactate zones as well.

Nine individuals in this series with coronary atherosclerosis and angina pectoris showed a completely normal pattern of lactate metabolism with catecholamine stress: (1) Two

![Figure 10](image1.png)

**Figure 10**

Interrelationship of electrocardiographic findings as a base line and coronary artery lesions and lactate metabolic findings. See text.

![Figure 11](image2.png)

**Figure 11**

In spite of normal electrocardiograms made during rest and after exercise, significant coronary artery disease was present with evidence for diffuse ischemia from multiple site lactate studies. Key as in figure 6. LCA = left coronary artery.
of these nine patients had ventricular aneurysm and congestive failure. (2) One patient had disease confined solely to the right coronary artery. (3) Four patients had mild coronary disease with either 50% stenosis or symmetrical narrowing of a single vessel. (4) Two patients had severe involvement of all three coronary vessels.

**Illustrative Cases**

*Patient J.V. (Fig. 11)*

A 59-year-old clergyman with severe angina pectoris had normal resting and post-exercise electrocardiograms. Coronary angiograms demonstrated an irregular right coronary artery (RCA), stenosis of the main left coronary artery (LCA), with narrowing and irregularity of the left anterior descending (LAD), and left circumflex (LCf) arteries. Studies of lactate metabolism disclosed abnormalities in blood draining from the anterolateral and posterior surfaces of the left ventricle. Thus, in spite of normal resting and post-exercise electrocardiograms, significant coronary artery disease was present with evidence of diffuse ischemia derived from lactate studies at multiple sites.

*Figure 12*

The post-exercise electrocardiographic tracing was abnormal and gave a clue to the underlying pathological lesion, namely, isolated occlusion of the LAD. The functional significance of this isolated lesion was defined by the isolated anterior lactate abnormality. Key as in figure 6.
The resting electrocardiogram clinically defined abnormal inferior and lateral zones of the myocardium where blood supply was definitely limited by the occluded RCA and LC; 26% lactate production by the posterior zone was consistent with posterior ischemia. DMI = diaphragmatic myocardial infarct. Key as in figure 6.

Patient W.H. (Fig. 12)

A 43-year-old physician with incapacitating angina pectoris had a normal resting electrocardiogram but definite abnormalities appeared after exercise. Angiography demonstrated a normal right coronary artery and normal left circumflex vessel. Total occlusion of the left anterior descending artery was noted, with bridge collateral vessels and distal filling of the anterior descending artery beyond the site of the lesion. Lactate studies showed abnormal production in blood drained from the anterior myocardium, but extraction in the other areas sampled. Here, the post-exercise electrocardiogram gave a clue to the underlying pathological lesion which was an isolated one, and in turn, the functional significance of this isolated lesion was defined by the lactate production. Also, lactate production was no longer detected as the catheter was withdrawn to receive a coronary venous sample with mixed normal and ischemic streams of venous blood.

Patient J. K. (Fig. 13)

The resting electrocardiogram of a 54-year-old laborer with almost continual angina pectoris showed evidence of an old diaphragmatic myocardial infarction. Coronary angiography demonstrated total occlusion of both the right coronary artery and left circumflex vessel with narrowing and multiple sites of stenosis in the left anterior descending artery. Lactate studies showed extraction in the anterior zone and abnormal production in the posterior zone of the left ventricle. Here, the electrocardiogram defined clinically an abnormal inferior region of the myocardium. Its blood supply was definitely limited by occlusion of both the right coronary artery and left circumflex vessel, and this region was clearly ischemic by lactate studies.

Patient D. C. (Fig. 14)

The patient was a 59-year-old construction worker with persistent angina pectoris and a past history of several myocardial infarctions...
and congestive heart failure. The resting electrocardiogram demonstrated an old diaphragmatic myocardial infarction. Coronary arteriography showed total occlusion of both the right coronary and left anterior descending arteries, and a normal left circumflex artery. There was gross lactate production in the venous drainage from anterior and septal regions of the myocardium, and normal extraction in blood sampled from the posterior myocardium. Here, the abnormal anterior lactate production was consistent with ischemia beyond the site of occlusion in the left anterior descending artery. The normal lactate extraction from the posterior and inferior portions of the myocardium corresponded to the normal left circumflex vessel but did not substantiate the electrocardiographic evidence of old diaphragmatic infarction which may have been the result of the old right coronary occlusion. This may now be the site of metabolically inactive scar, or represent an ischemic area not draining to the coronary sinus.

Figure 15

Two separate regions of ischemia are inferred from the anterior and posterior production of lactate corresponding to the anatomic pattern of significant coronary disease of the LAD, RCA, and atrioventricular branch of the LCf with remarkable preservation of the two major marginal branches to the lateral wall. Note the pattern of lactate extraction from the lateral zone which suggests dilution of the anteriorly produced lactate with normal venous blood from the marginal left ventricular veins. The lactate production from the posterior zone indicates the addition of abnormal venous effluent from the inferoposterior venous sites. Key as in figure 6.
PATIENT E. P. (FIG. 15)

A 56-year-old man had been severely limited by angina pectoris. Resting electrocardiogram showed nonspecific ST-T wave changes in leads I, aV \textsubscript{L}, V \textsubscript{5} and V \textsubscript{6} which were consistent with ischemia. Coronary angiography demonstrated two areas of 90\% stenosis in the right coronary artery, total occlusion of the left anterior descending artery with distal filling of this vessel by right-to-left and left-to-left collaterals. The main left circumflex artery and its marginal branches appeared normal, but significant intraluminal disease was seen in its atroventricular branch. Lactate studies showed 103\% production anteriorly, 6\% extraction laterally, and 8\% production posteriorly; these findings are consistent with the finding of ischemic zones in both the anterior and posterior regions of the left ventricular myocardium. These biochemically determined ischemic zones corresponded well with the anatomic pattern of disease.

**Discussion**

In the evaluation of patients with suspected coronary artery disease, selective cine coronary angiography can give an accurate demonstration of the morbid anatomy of the arterial vessels.\textsuperscript{10} With a knowledge of the type, amount, and degree of the coronary lesion, one can try to estimate the presence and quantitative aspects of potential underlying myocardial ischemia. Such estimates, however, are only speculative, and the need exists for methods to evaluate myocardial ischemia much more directly. There are three potential avenues of approach to measurement of myocardial ischemia: (1) biochemical effects—assessment of myocardial metabolic abnormalities associated with underlying ischemia, that is, myocardial lactate metabolism; (2) electrophysiological effects—the use of the resting and post-stress electrocardiogram to evaluate the effect of ischemia on myocardial electrophysiology; and (3) mechanical effects—the measurement of myocardial performance by hemodynamic, angiographic, and external recording techniques to demonstrate aberrations in left ventricular contraction induced by ischemia.

This study has dealt with two of these areas: the rest and post-exercise electrocardiograms and regional lactate abnormalities.

**Regional Lactate Abnormalities as an Indication of Ischemia**

Under normal steady state conditions, the heart extracts lactate at rest or in an unchanged or greater percentage during catecholamine stress (fig. 3). Abnormalities in myocardial lactate metabolism are the result of glycolysis proceeding in excess of oxidation. The commonest clinical cause of this is low tissue oxygen concentration due to myocardial ischemia.

Although many early workers associated increased production of lactic acid by the heart with an oxygen deficit, Clark and associates\textsuperscript{11} first demonstrated that in the well-oxygenated heart, no lactic acid production occurred, whereas a partial or complete oxygen deficit resulted in such production. Brief ligation of a coronary artery has been shown to induce lactate production.\textsuperscript{12} It has also been shown that not only total occlusion but decreased left ventricular coronary flow (25\% of control) will result in lactate production when coronary venous Po\textsubscript{2} is less than 10 mm Hg.\textsuperscript{13} More recently, Scheuer and Brachfeld\textsuperscript{14} have shown in a similar experimental design that lactate production preceded both electrocardiographic current of injury and associated left ventricular failure.

Objection can be raised concerning use of isoproterenol to induce myocardial stress because isoproterenol per se may promote glycolysis and lactate production.\textsuperscript{15} It has been our empirical observation, however, that subjects with normal coronary arteries do not show lactate production or even decreased extraction in the dose range of 1 to 5 \textmu g/min of isoproterenol. The percent extraction of lactate (<10\%) does not truly equate with glycolysis in excess of oxidation. However, our control observations suggest that the myocardium extracts uniform amounts of lactate. Therefore, it is valid to assume that some
Regional production of lactate has occurred when there is a marked variation or a decrease in extraction, or both. The control group with documented normal coronary arteries depicted in figure 3 illustrates this normal pattern of extraction. Other possible theoretical objections to the interpretation of lactate as being an indicator of ischemia have been discussed elsewhere and found to be not applicable.

Practical Considerations of This Study

Coronary Artery Lesions

Many more coronary lesions were noted than electrocardiographic abnormalities. In general, sites of significant disease were associated with electrocardiographic or regional lactate evidence of ischemia, or with both. With an increasing number and severity of coronary lesions, subjects were more prone to widespread ischemia. When a coronary artery lesion was not associated with an electrocardiographic or regional lactate abnormality, opinion was often tempered regarding its current clinical significance.

Electrocardiogram

The abnormal electrocardiogram was always associated with an anticipated underlying coronary artery lesion and thus gave an accurate indication of a portion but not all of the underlying coronary pathology. Multiple electrocardiographic abnormalities were almost invariably associated with severe multiple vessel disease. The normal electrocardiogram was of no predictive value. Fourteen percent of subjects studied had normal rest or post-exercise electrocardiograms in spite of significant coronary artery disease.

The electrocardiographic and vectorcardiographic patterns of subendocardial ischemia and injury have been equated with those found in coronary insufficiency and angina pectoris. In this situation the S-T vector is directed away from the local zone of ischemia in the left ventricle while the T-wave vector points toward the ischemic zone. Thus, localized depressed S-T segments and upright T waves are seen. As the ischemic process extends through the entire thickness of the myocardium, the mean spatial T-wave vector swings away from the local zone of ischemia and inverted T waves are seen. Such abnormal electrocardiographic zones have been broadly localized to portions of the left ventricle, that is, anteroseptal, anterior, and inferior. That specific ST-T abnormalities do indicate local coronary pathology is controversial. The demonstration of abnormal lactate metabolism and a significant coronary artery lesion associated with such a localized abnormal zone would present strong evidence that a specific arterial lesion causing local ischemia was the basis for the zonal electrocardiographic abnormality. Of the 10 individuals in the study with localizing rest electrocardiographic findings suggesting ischemia but without infarction, seven had a corresponding lactate abnormality and all had an associated coronary arterial lesion. In the seven subjects with such ischemic changes in the electrocardiograms only after exercise, six had corresponding local lactate abnormalities and all seven had a local arterial lesion.

The type of electrocardiographic abnormality and the associated lactate pattern were of interest and have several implications: Of those individuals with a single resting locus of infarction or ischemia, two thirds showed associated regional lactate abnormalities suggesting a current ischemic process or residual ischemic myocardium in a region of old scar. In the remaining one third, the single electrocardiographic zone of abnormality was associated with a normal lactate pattern and may represent old fibrous scar which is metabolically inactive or the development of adequate collaterals to the adjoining area. Multiple electrocardiographic abnormalities corresponded well with associated multiple lactate abnormalities, suggesting severe underlying disease with multiple sites of ischemia.

Regional Lactate Pattern

The use of the regional lactate pattern to indicate zones of myocardial ischemia usually permitted the prediction of sites of coronary
vessel lesions and adequacy of existing collaterals. Two important practical applications of regional lactate abnormalities were in the selection of candidates for myocardial revascularization surgery and in the postoperative evaluation of such patients for signs of objective improvement in myocardial perfusion.

**Regional Lactate Studies and Revascularization Surgery**

Collateral connections between surgically implanted vessels and the somatic coronary circulation are thought to depend on the presence of local ischemia for their development. The ability to identify lactate production from selected venous drainage sites of the left ventricle testifies to the location of such local ischemic zones. Experience now permits selective implantation based on recognition of a zone of ischemia as in the following examples:

1. Patient W.H., whose case was reported herein, presents as an ideal candidate for anterior implantation with an isolated single lesion of the left anterior descending ramus, and clear-cut anterior lactate production.

2. A patient with a 90% stenosis of the right coronary artery and 50% stenosis of the left anterior descending artery showed only posterior lactate production—this finding confirmed the right coronary artery as the major site of trouble. This patient was rejected for implantation, and a right coronary patch graft was recommended.

3. No decision about the site for revascularization could be made from arteriography in a patient with severe three-vessel disease. Based on isolated posterior lactate production, an implant was placed under a branch of the left circumflex artery on the inferior wall of the left ventricle.

4. In one patient with three-vessel disease (fig. 15), two isolated areas of lactate production (anterior and posterior) were demonstrated. Because of the lactate findings an internal mammary pedicle implant was placed anteriorly, and an intercostal branch of this artery was brought forward around the apex to the inferior zone.

In the postoperative evaluation of patients after myocardial revascularization, regional lactate sampling has given objective evidence for improvement in the revascularized zone. Six of nine patients evaluated 1 year after surgery showed reversal of preoperative lactate production to postoperative lactate extraction in the venous effluent from the revascularized region, whereas the lactate production in other untreated regions remained unchanged or worsened. Three patients with unsuccessful implants showed persistent lactate production, confirming the constancy of the myocardial metabolic abnormality.

**Practical Problems of This Study**

In order to identify and quantify coronary arterial lesions accurately, high-quality coronary angiograms are absolutely essential. This has been stressed by a correlative study between pre-mortem angiograms and postmortem injection with pathological studies.

Certain zones of ischemia may not be detected by the methods presented, that is, ischemic myocardium associated with occlusion of the right coronary artery when the venous effluent may drain via the middle or anterior cardiac (right ventricular) veins and hence may be undetected even in the most proximal coronary sinus. This may have occurred in one patient with isolated right coronary disease in our series. The converse of this occurred in one patient seen recently and not reported in this series, in whom lactate production was detected posteriorly in the presence of a solitary left anterior descending arterial lesion. Effluent of contrast material injected selectively into the left coronary artery appeared in the posterior interventricular vein.

If the area of myocardial ischemia is small or the degree of applied stress is too minimal for a limited amount of coronary artery disease, lactate may not be produced in an amount sufficient to be sampled or may be diluted by normal blood from adjacent areas. Such dilution was frequently seen in this study: for example, in patient W.H. with anterior ischemia, lactate was detected only at the anterior sampling site. In a second

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sample taken only 1 to 2 cm back in the coronary sinus, production reverted to extraction. Normal venous blood diluted the abnormal. This may have been the explanation for normal lactate metabolism in four patients each with single vessel stenosis.

No explanation was evident for two of the nine patients with normal lactate metabolism.

In certain instances of left ventricular failure due to ventricular aneurysm, catecholamine infusion may actually improve myocardial function and thus not provide stress to induce ischemia. This occurred in two patients with severe coronary heart disease and failure who did not show lactate production.

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