Coronary Hemodynamic Findings During Spontaneous Angina in Patients with Variant Angina

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SUMMARY To define more completely regional coronary hemodynamic changes that occur during spontaneous angina pectoris in patients with variant angina, we measured coronary sinus and great cardiac vein blood flow (CSF and GCVF) and aortic and left ventricular pressures before and during spontaneous angina in six patients with variant angina. During spontaneous angina, ECGs in four patients showed evidence for transient anterior regional ischemia (ST-T-wave changes in I, aVL, II, III, aVF) and in two patients showed evidence for transient inferior regional ischemia (ST-T-wave changes II, III, aVF). During spontaneous angina, CSF decreased in five of six patients (27 ± 10 ml/min, p < 0.05), compared with measurements made during a pain-free interval. In all four patients with anterior ischemia, GCVF decreased 34 ± 13 ml/min (p < 0.005). In the two patients with inferior ischemia, GCVF was unchanged, but the difference between CSF and GCVF, an index of inferior regional blood flow, decreased 36 ± 20 ml/min during ischemia. Heart rate was not significantly different during angina, and mean aortic pressure decreased in three patients, increased in two and was unchanged in the other. Left ventricular end-diastolic pressure increased 10 ± 2 mm Hg during spontaneous angina (p < 0.01).

These data provide direct evidence that blood flow to the ischemic region during spontaneous angina is decreased in patients with variant angina. These results support the concept that a functionally important decrease in regional myocardial oxygen delivery occurs in certain patients with variant angina coincident with angina and ST-segment and T-wave changes.

A TRANSIENT DECREASE in myocardial blood flow is the hypothesis proposed to explain attacks of angina pectoris and ST-segment shifts in patients with variant angina without detectable increases in myocardial oxygen demands, i.e., heart rate or blood pressure. Angiography has supported this concept by showing coronary artery vasospasm during angina in such patients. However, demonstration of coronary vasospasm during angina does not prove that blood flow is actually reduced in any ventricular region. Indirectly, attempts have been made to assess ventricular blood flow by the use of several techniques. Maseri and co-workers found a relative decrease in regional left ventricular uptake of thallium-201 during both spontaneous and ergonovine-induced angina in patients with variant angina. In another study this group observed decreased great cardiac vein oxygen saturation during episodes of anterior left ventricular region ischemia but not inferior left ventricular regional ischemia. Ricci et al. described a patient with variant angina in whom total coronary sinus blood flow (CSF) declined during spontaneous angina. We have shown a decrease in regional left ventricular blood flow in patients with variant angina during ergonovine-induced angina. To our knowledge, regional coronary hemodynamic changes occurring during spontaneous angina in patients with variant angina have not been reported. In this study we evaluated and quantitated total and regional left ventricular blood flow responses during spontaneous angina in patients with variant angina.

Patients and Methods

Selection of Patients

We studied six men (average age 53 years; range 44–62 years) undergoing cardiac catheterization to further define the basis for chest pain. All of these patients had recurrent episodes of angina, often at rest and usually cyclical, associated at times with transient ST-segment elevation. Electrocardiographic leads I, aVL, and V₅₋₆, reflecting the anterior left ventricular region, showed changes in patients 1–4 (tables 1 and 2). Electrocardiographic leads 2, 3 and aVF, reflecting the inferior left ventricular region, showed changes in patients 5 and 6 (table 1). These changes, which accompanied angina pectoris, were used to define which left ventricular region was considered ischemic. Patient 6 was receiving intraaortic balloon pump support, initiated during catheterization to control angina refractory to other medical therapy.

These patients were selected from a larger group with variant angina evaluated in our catheterization laboratories over the past 4 years. During these evaluations, we made detailed observations when angina occurred spontaneously. The six patients reported here represent those in whom coronary flow measurements could be accomplished during spontaneously occurring pain.
TABLE 1. Observations Before and During Spontaneous Angina Pectoris

<table>
<thead>
<tr>
<th>Pt</th>
<th>HR (beats/min)</th>
<th>Pressure (mm Hg)</th>
<th>Blood flow (ml/min)</th>
<th>Coronary resistance (mm Hg/ml/min)</th>
<th>Documented coronary vasospasm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ST</td>
<td>Aortic</td>
<td>LVEDP</td>
<td>CSF</td>
<td>GCVF</td>
</tr>
<tr>
<td>1</td>
<td>0</td>
<td>I, V5, V6*</td>
<td>70</td>
<td>66</td>
<td>130/75</td>
</tr>
<tr>
<td>2</td>
<td>0</td>
<td>I, V2</td>
<td>65</td>
<td>64</td>
<td>130/85</td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>V2</td>
<td>60</td>
<td>72</td>
<td>150/100</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>aV2, V4*</td>
<td>55</td>
<td>60</td>
<td>138/66</td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>II, III, aV4</td>
<td>65</td>
<td>80</td>
<td>165/95</td>
</tr>
<tr>
<td>6</td>
<td>0</td>
<td>II, III, aV4</td>
<td>48</td>
<td>47</td>
<td>90/125/50</td>
</tr>
<tr>
<td></td>
<td></td>
<td>aV4</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Peaked T waves but no ST shift.
†Intraventricular balloon pump assist.
Abbreviations: C = control; SA = spontaneous angina; LVEDP = left ventricular end-diastolic pressure; HR = heart rate; CSF = coronary sinus flow; GCVF = great cardiac vein flow; CRt, CRANT, CRINF = total, anterior and inferior region coronary resistance; LAD = left anterior descending; TOT = total occlusion; DIFF = diffuse; CAD = coronary artery disease.

TABLE 2. Observations Before and During Ergonovine-induced Angina Pectoris

<table>
<thead>
<tr>
<th>Pt</th>
<th>HR (beats/min)</th>
<th>Pressure (mm Hg)</th>
<th>Blood flow (ml/min)</th>
<th>Coronary resistance (mm Hg/ml/min)</th>
<th>Ergonovine dose (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ST</td>
<td>Aortic</td>
<td>LVEDP</td>
<td>CSF</td>
<td>GCVF</td>
</tr>
<tr>
<td>1</td>
<td>0</td>
<td>I, V5, V6</td>
<td>73</td>
<td>79</td>
<td>140/85</td>
</tr>
<tr>
<td>2</td>
<td>0</td>
<td>I, V2</td>
<td>72</td>
<td>75</td>
<td>140/80</td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>V2</td>
<td>60</td>
<td>70</td>
<td>150/90</td>
</tr>
</tbody>
</table>

Abbreviations: B = before ergonovine; A = after i.v. ergonovine. Other abbreviations as in table 1.
Catheterization Procedure

Each patient gave informed consent. Nitroglycerin was omitted for at least 8 hours before study. Catheterization was performed with the patients in a postabsorptive state without premedication. A #8F Sones catheter was positioned from the right brachial artery into the aorta to measure pressure with a Statham P23Db transducer. Mean pressure was obtained by electronic filtration. The catheter was advanced to the left ventricle to record left ventricular end-diastolic pressure at high amplification. The same catheter was used for coronary angiography.

A multithermistor catheter was advanced from a right antecubital vein and positioned in the coronary sinus. Our use of this specially designed catheter to measure regional left ventricular blood flow has been described in detail. Briefly, the distal external thermistor was advanced to the great cardiac vein. The proximal external thermistor was positioned in the sinus between the ostium and the termination of the middle cardiac veins. The venous phase of a coronary angiogram was recorded on a videodisc recorder (General Electric). This image was used to aid in positioning the coronary venous catheter. Location of the catheter was frequently checked by referring to this recording. Selected ECG leads, chosen because ST-segment shifts had been noted during spontaneous angina, were recorded and continuously monitored. All other standard limb leads and V₄ were also recorded during the measurement periods.

Control Period

After catheters were in place, a control set of coronary angiograms was filmed. Ten minutes later, aortic and left ventricular pressures and CSF and great cardiac vein flow (GCVF) were measured and recorded during a hemodynamically stable interval. This was defined as a symptom-free interval that lasted at least 1 minute and had not been preceded by angina or spontaneous ECG changes for at least 30 minutes. During this interval, left ventricular systolic and end-diastolic pressures varied ±5% or less. Selected ECG leads were also recorded during this period.

Spontaneous Angina

After control recordings, either left ventricular or aortic pressure and ECG leads were continuously monitored. When the patient reported angina, both aortic and left ventricular pressures and CSF and GCVF were measured and recorded over a 1-minute interval. Selected ECG leads were also recorded during angina. Nitroglycerin (one to three tablets, 0.4 mg each) was then administered until angina was relieved and ST shifts returned to control values. In patient 4, intravenous (200 µg, bolus) and intracoronary (50–100 µg, bolus) nitroglycerin were administered instead of sublingual nitroglycerin to relieve angina.

Documentation of Coronary Artery Vasospasm

To avoid coronary hemodynamic changes related to angiography, none of these patients had coronary angiography performed with the hemodynamic recordings made during spontaneous angina. After these measurements, patient 4 had repeat angiography (before nitroglycerin administration); at another time, patients 1–3 received ergonovine maleate (0.05–0.20 mg intravenously in divided doses) and had repeat coronary angiography during ergonovine-induced angina. These angiographic observations were used in addition to the ECG changes that accompanied angina to define which left ventricular region was considered ischemic. Angiographic and coronary hemodynamic data obtained before and during ergonovine-induced angina in these three patients have been described in detail (table 2).

Calculations

Blood flows from the coronary sinus and great cardiac vein were calculated as described elsewhere. Blood flow was calculated three or four times over consecutive 10–15-second intervals. Values obtained during a 1-minute interval were averaged and reported as blood flows for respective periods. CSF was used as an index of total left ventricular flow. GCVF was taken as an index of flow from the anterior left ventricular region supplied predominantly by the anterior descending branch of the left coronary artery. Blood flow from the other regions was assumed to represent the difference between the anterior regional contribution (GCVF) and total left ventricular flow (CSF). Blood flow from the other regions should be predominantly supplied by the circumflex and right coronary arteries and have a variable contribution from the anterior descending and diagonal coronary arteries. For simplicity, this region was arbitrarily referred to as the inferior region.

During each period, phasic and mean aortic pressure and left ventricular end-diastolic pressure were determined by averaging measurements from at least 10 consecutive beats. Indexes of anterior regional, inferior regional and total coronary resistance were derived from the ratio of simultaneously recorded mean aortic pressure and respective flow. For example, anterior regional resistance (in mm Hg/ml/min) was taken as the quotient of mean aortic pressure and GCVF. Heart rate was calculated from the ECG. The magnitude of ST-segment shift was calculated by measuring the difference between the position of the ST segment (0.08 second after the onset of QRS complex) during the control period and during spontaneous angina. New ST depression or elevation ≥ 1 mm was considered a significant change. The magnitude of dynamic narrowing in diameter of the coronary artery was estimated as described elsewhere.

Briefly, dynamic coronary diameter reduction > 50% (focal or diffuse), which was reversed after nitroglycerin administration and which accompanied symptoms and signs suggesting transient ischemia, was considered indicative of coronary vasospasm. "Fixed" atherosclerotic coronary narrowing was considered present when the diameter of a narrowing noted on the control angiogram was unchanged (±20%) after nitro-
glycerin. Fixed coronary narrowings > 50% were considered indicative of significant atherosclerotic coronary artery disease.

Statistical Analysis

The mean ± SEM were determined during control and spontaneous angina periods. The paired t test was used to compare data from these periods for each patient. A p value < 0.05 was considered statistically significant.

Results

Individual clinical, ECG, angiographic, aortic and left ventricular end-diastolic pressures, and regional coronary hemodynamic data obtained before and during spontaneous angina are summarized in table 1. The episode of spontaneous angina was temporally close to the end of the control period, 10 minutes average (range 3–20 minutes).

Patients 2–4 and 6 had significant coronary artery disease in at least one major artery. No collaterals were observed during pain-free periods in these patients and were observed only in patient 4 during either spontaneous or ergonovine-induced angina. Patients 1 and 5 had normal coronary arteriograms. Patient 5 also had a right coronary artery arising from the left sinus of Valsalva and terminating in the posterior interventricular groove. Clinical, ECG, heart rate, aortic and left ventricular end-diastolic pressure, and regional coronary hemodynamic responses before and during spontaneous angina appeared similar in patients with and without coronary artery disease.

Angina and ECG Changes

Each patient had a spontaneous episode of angina accompanied by transient ST-segment or T-wave changes during cardiac catheterization: ST-segment elevation in patients 2, 3 and 5, ST-segment depression and T-wave inversion in patient 6, and T-wave peaking without significant ST-segment elevation or depression in patients 1 and 4 (figs. 1 and 2). These ST- and T-wave changes noted during spontaneous angina at catheterization involved the same ECG leads previously noted in all patients. Each patient reported that these spontaneously occurring episodes of angina were mild to moderate in intensity compared with the episodes of angina they usually experienced.

Systemic and Left Ventricular Hemodynamic Findings

Heart rate during both control and spontaneous angina periods was similar (61 ± 3 vs 65 ± 5 beats/min; NS), as was mean aortic pressure (100 ± 6 vs 96 ± 6 mm Hg; NS). However, left ventricular end-diastolic pressure increased during spontaneous angina in each of five patients in whom this measurement was made, from 16 ± 0.5 to 27 ± 2.0 mm Hg (p < 0.01).

Regional Coronary Hemodynamic Findings

CSF decreased during spontaneous angina in five of the six patients, from 141 ± 11 to 114 ± 16 ml/min (p < 0.05). This decrease in total left ventricular blood flow was primarily a result of decreased flow from the region that ECG changes indicated was ischemic. Anterior regional flow during spontaneous angina decreased in patients 1–4, who had transient ischemia involving the anterior region, from 96 ± 13 to 62 ± 11 ml/min (p < 0.05). In contrast, in patients 5 and 6, who had transient ischemia involving the inferior region, anterior regional flow during spontaneous angina was similar to control period measurements (61 ± 8 vs 59 ± 7 ml/min). Inferior regional blood flow during spontaneous angina decreased in patients 5 and 6, with transient ischemia involving the inferior region, from 67 ± 18 to 31 ± 2 ml/min. In contrast, in

![Figure 1](http://circ.ahajournals.org/)

**FIGURE 1.** *Patient 1. ECG, aortic and left ventricular pressures and regional coronary venous flows during control period and spontaneous angina pectoris (AP). Coronary sinus flow (CSF) and great cardiac vein flow (GCVF) decreased, while aortic and left ventricular pressures increased slightly. Electrocardiographic leads I, V2, and V5 did not show significant ST elevation during this angina episode, but did demonstrate peaking of the T waves compared with the control ECG.*
patients 1–4, inferior regional flow during spontaneous angina was similar to control (53 ± 8 vs 65 ± 19 ml/min; NS). Overall, during spontaneous angina, blood flow decreased in all six ECG-defined ischemic regions, from 86 ± 11 to 51 ± 9 ml/min (p < 0.01). In contrast, blood flow in the nonischemic regions did not change significantly during spontaneous angina (56 ± 6 vs 63 ± 12 ml/min; NS).

Total coronary resistance increased during spontaneous angina in five of the six patients, from 0.74 ± 0.10 to 0.90 ± 0.09 mm Hg/ml/min (p < 0.05). The major portion of this increased total resistance was attributed to an increase in the region that ECG changes indicated was ischemic. Resistance increased during spontaneous angina in ischemic regions in all six patients, from 1.33 ± 0.27 to 2.08 ± 0.25 mm Hg/ml/min (p < 0.01). In contrast, mean coronary resistance in nonischemic regions was similar during control and spontaneous angina periods (1.90 ± 0.23 vs 1.89 ± 0.46 mm Hg/ml/min; NS).

**Discussion**

These data suggest that left ventricular blood flow in ischemic regions decreased and ischemic region coronary resistance increased during spontaneous angina.
in certain patients with variant angina. This decrease in ischemic regional flow and increased ischemic region coronary resistance occurred concurrently with angina, ST-T-wave changes and increased left ventricular end-diastolic pressure. Coronary artery vasospasm was documented in four of these patients. Coronary artery perfusion pressure, dependent on the difference between aortic and left ventricular diastolic pressures, often declined in these patients. This decline probably contributed to the decrease in the ischemic regional blood flow and to the decrease in one of the two instances in the nonischemic region.

These coronary hemodynamic observations supported the concept of measuring total left ventricular and regional, rather than only total, values in patients in whom functional or anatomic differences exist between the coronary arteries supplying the anterior and inferior left ventricular regions. In these six patients, five of six total left ventricular blood flow measurements decreased during spontaneous angina, while all six measurements of ischemic region blood flow decreased. Nonischemic region blood flow measurements during spontaneous angina changed variably: Two decreased, two increased and two did not change. Consideration of total and regional blood flow measurements supplied information not available with only total measurements. Similar considerations should be important when evaluating other groups of patients. In particular, measurement of total and regional coronary hemodynamic responses to various stimuli in patients with coronary artery disease may add important observations when compared with measurements of only total responses.

We have described coronary and left ventricular hemodynamic responses during ergonovine-induced angina in variant angina patients. We found evidence that left coronary artery vasospasm significantly reduced CSF and GCVF. In contrast, in patients with right coronary artery vasospasm, GCVF was unaltered, whereas CSF decreased in one of three patients with right coronary vasospasm. We interpreted these results to support the concept that ergonovine significantly reduced delivery of oxygen to a left ventricular region coincident with angina and ST-segment shifts in patients with variant angina.

Our data suggest that coronary hemodynamic changes during spontaneously occurring angina are similar to those during ergonovine-induced angina in some patients. Patients 1–3 had coronary hemodynamic measurements during both spontaneous (table 1) and ergonovine-induced angina (table 2). During both episodes of angina, ECG changes were noted in the same leads and ischemic region coronary flow decreased and resistance increased. These findings agreed with our observations and those of others that spontaneous and ergonovine-induced angina were often remarkably similar. However, a decrease in aortic pressure seemed to occur more often during spontaneous than during ergonovine-induced angina.

The character of the ECG responses warrants additional comment, and it would be interesting to compare episodes of angina associated with ST-segment elevation with episodes without ST elevation. Three of the six spontaneous angina episodes observed during this study were not accompanied by ST-segment elevation. It is unclear whether angina episodes not associated with ST elevation represent a lesser degree of myocardial ischemia than do episodes with ST elevation, as suggested by some investigators. Patient 1 (fig. 1) had peaked T waves and small changes in left ventricular end-diastolic pressure and regional coronary flow during spontaneous angina. ST elevation and large end-diastolic pressure and flow changes occurred during ergonovine-induced angina, supporting this concept. However, patient 4 (fig. 2) also showed only peak T waves during spontaneous angina. But large changes in end-diastolic pressure and anterior regional blood flow occurred and were associated with nonvisualization of the anterior descending branch. This patient's data indicate that a considerable amount of myocardium (i.e., the entire anterior wall) may be ischemic without ST elevation. These data support the statement of Maseri and co-workers that "the relation between regional myocardial ischemia and ST-segment changes is indeed a complex one, and that some traditional concepts must be revised in the light of direct experimental evidence."

These data support the findings in a patient described by Ricci et al. In that patient, total CSF was greater after, compared with during, an episode of spontaneous angina. To our knowledge, no other direct measurements of total or regional left ventricular blood flow have been reported in patients with variant angina during spontaneous angina. Prior investigation has used thallium-201 uptake as an indirect estimate of left ventricular perfusion during spontaneous angina. These studies showed decreased uptake in regions, defined from the ECG, compared with control or redistribution studies. Other studies by Chierchia et al. used a drop in regional oxygen saturation to suggest that regional flow decreased during spontaneous angina.

This study has certain limitations. First is the method of measuring coronary blood flow. We used the multithermistor thermodilution technique in more than 150 patients. In patients with constant (± 5%) systemic blood pressure and heart rate, we have found that CSF and GCVF measurements, repeated at intervals of 2–5 minutes, usually vary < 10%. Thus, the flow responses observed should reflect actual changes in regional left ventricular blood flow rather than variation in the accuracy of the method of measurement.

Second, changes in CSF in all patients, and particularly in patients 5 and 6, whose ECG changes suggested transient inferior regional ischemia, must be interpreted cautiously. The pattern of coronary venous drainage of the right coronary artery or inferior wall is important. These veins often enter the coronary sinus at or near its ostium or drain separately into the right atrium. Individual patient variability relative to the degree of venous effluent from the right coronary artery, which was sensed by coronary sinus sampling, may explain the larger decrease in inferior region flow...
during spontaneous angina observed in patient 6 (ST depression) than in patient 5 (ST elevation). CSF, measured near the coronary sinus ostium, may also be distorted by right atrial admixture, and in this regard proper patient selection is important.24

Third, data are lacking concerning myocardial oxygen uptake and lactate use during these spontaneous angina episodes. Abnormalities in these areas, particularly lactate extraction, would have indicated the presence of transient myocardial ischemia when only small changes in regional blood flow occurred without ST elevation. However, blood sampling for oxygen and lactate through the small (0.8-mm injectate) lumen of the regional thermodilution catheter requires about 1–2 minutes. Furthermore, sampling cannot be done while coronary flow is being measured, because the indicator is continuously infused through this lumen. In our experience spontaneous angina episodes are often brief, lasting 2 minutes or less. Therefore, the sampling would be likely to occur over a period of changing or resolving ischemia or even reactive hyperemia. Thus, we chose to limit this study to measurements that could be made continuously and rapidly. Recently, however, continuous on-line coronary venous oxygen saturation has been monitored with a highly accurate fiberoptic catheter system.10 Application of this technique in future studies of spontaneous angina should provide some important metabolic data.

One additional point regarding the difference between CSF and GCVF as an index of inferior region flow should be considered. Great cardiac vein sampling has shown to be an index of anterior regional blood flow12, 14 and coronary sinus sampling an index of total left ventricular blood flow.15–18 However, the difference between these two blood flow values was not likely to precisely reflect only inferior region flow in most patients. First, the variable venous drainage of the right coronary will affect its contribution to this index of inferior region flow. Second, although the venous effluent of the anterior descending largely drains into the great cardiac vein, a portion may drain to the midcoronary sinus and be reflected in the inferior region flow index.14 Thus, the spacing of the two thermostors on the coronary thermodilution catheter was important, and this potential source of error will vary from patient to patient. This depended on the relationship in each patient between coronary venous drainage and coronary venous catheter thermostor placement.

We consider this difference in GCVF and CSF as a reasonable index of flows from the inferior and other regions. Therefore, in most patients a change in anterior regional flow will cause a primary change and a directionally similar change in GCVF and may also cause an independent and directionally similar change in CSF. In contrast, a change in inferior regional flow should not primarily produce a change in GCVF but should cause a directionally similar change in CSF.

These data provide direct evidence that in these patients with spontaneous angina, left ventricular blood flow decreases in ischemic regions. These results support the concept that a significant reduction in regional delivery of oxygen occurs in patients with variant angina coincident with spontaneous angina and ST-T changes.

Acknowledgment
We thank Dr. R. Carter for aid in statistical evaluation of the data, R.T. Solloway for technical assistance, and Alice Cullu for editorial assistance.

References
Patterns of Disturbed Myocardial Perfusion in Patients with Coronary Artery Disease

Regional Myocardial Perfusion in Angina Pectoris


SUMMARY Fifty patients who presented with angina pectoris were studied to examine the disturbances of regional myocardial perfusion during stress. Each patient underwent 16-point precordial mapping of the ECG during an exercise test, and coronary and left ventricular angiography. Regional myocardial perfusion was assessed using an atrial pacing test and a short-lived radionuclide, krypton-81m. Eleven patients had negative exercise tests and uniform increases in myocardial activity of krypton-81m of 98 ± 18.0% during pacing. Ten patients performed 30,000-43,000 J in positive exercise tests. These patients showed abnormal coronary anatomy and increases in myocardial activity of krypton-81m to remote and jeopardized myocardium at the onset of pacing. However, further pacing produced a decrease in activity in the affected segment of 68.0 ± 9.0% accompanied by ST-segment depression and angina. Twelve patients achieved 26,000-32,000 J in positive exercise tests and had significant coronary artery disease. Atrial pacing produced increased activity of krypton-81m to remote myocardium. The jeopardized segment at first showed no change and then a decrease in regional activity of krypton-81m (89.0 ± 17%) accompanied by ST-segment depression and chest pain. Seventeen patients achieved only 7000–22,000 J in positive exercise tests. These patients showed abnormal coronary anatomy and developed decreases in regional activity of krypton-81m to the affected segment of myocardium starting at the onset of atrial pacing and decreasing by 88 ± 7.0% below control. We conclude that different patterns of disturbed myocardial distribution of krypton-81m are present during stress-induced ischemia in patients with coronary artery disease. There was a close temporal relationship between these disturbances and ST-segment depression.

CLINICAL RESEARCH in patients with ischemic heart disease should take into account the coronary anatomy, hematologic and hemodynamic factors, as well as coronary vasomotor tone. A detailed understanding of the disturbances of regional perfusion is difficult because of methodologic problems.1-4 Krypton-81m, with a half-life of 13 seconds, has been introduced and used in patients to assess changes in myocardial perfusion.6-7

In this study we assessed disturbances of regional myocardial perfusion in patients with angina pectoris and coronary artery disease. Changes in the regional myocardial distribution of the short-lived krypton-81m has been measured during atrial pacing and then related to the patient's symptoms, ECG during exercise and the findings at coronary arteriography.

Methods

Fifty patients (42 male and eight females, ages 38–64 years, mean 52 years) were admitted to Hammersmith Hospital with a history of angina pectoris.8 The 50 patients in this study all underwent the same sequence of investigations. The patients were admitted into the study as outpatients, and were admitted to the wards within 1–3 weeks. Exercise electrocardiography was performed within 1–3 days of admission. Left ventricular and coronary arteriography and krypton-81m scintigraphy of myocardial perfusion...
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