Behavior of Right and Left Ventricles During Episodes of Variant Angina in Relation to the Site of Coronary Vasospasm

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The effects of single-vessel coronary occlusion on simultaneously evaluated right (RV) and left ventricular (LV) performance were assessed and compared with LV perfusion patterns in 25 patients with variant angina. Coronary spasm involved the right coronary artery in 15 patients (group 1) and the left anterior descending coronary artery in 10 patients (group 2). Biventricular function was assessed by radionuclide angiography under basal conditions, during spontaneous or ergonovine-induced ischemia, and after resolution of the ischemic attack. Myocardial perfusion was assessed by thallium 201 scintigraphy in 21 patients of this series during superimposable ischemic episodes. In group 1, ischemia caused RV (14 of 15 patients) and LV (13 of 15 patients) regional dysfunction with significant reduction in RV and LV ejection fractions. The interventricular septum was involved in six of 15 patients, causing a more pronounced LV impairment. In group 2, all patients showed septal dyssynergies associated with a reduction of LV ejection fraction; absent or trivial RV involvement was observed. In both groups, LV perfusion defects were present in all patients with LV wall motion abnormalities during ischemia, matching the site of regional dysynergies. Thus, in a group of patients with variant angina and single-vessel disease, transient occlusion of the right coronary artery directly caused RV and LV impairment; in these patients, the extent of LV but not RV dysfunction appeared related to the presence of septal ischemia. Vasospasm of the left anterior descending coronary artery consistently caused LV dysfunction not associated with secondary effects on RV systolic function. (Circulation 1990;81:567–577)

Coronary vasospasm is a well-recognized cause of chest pain during rest. Coronary angiography, perfusion, and hemodynamic studies have characterized the circulatory response to acute, transient coronary occlusion; correlation between these findings and the electrocardiographic abnormalities observed in angina at rest demonstrated the existence of a continuous spectrum of vasospastic myocardial ischemia.1,2 Echocardiography and radionuclide ventriculography have also documented the time course and the extent of left ventricular (LV) impairment, according to the duration and coronary location of vasospasm.3,4 More recently, the systemic approach to this syndrome has shown a prevalent, marked dysfunction of the right ventricle during episodes of variant angina caused by right coronary artery (RCA) vasoconstriction.5 Thus, variant angina may also cause a wide spectrum of transient LV and right ventricular (RV) impairment: in this setting, the prevalence of the ischemic involvement of the right ventricle and the functional interplay between the two ventricles are still to be elucidated. This appears particularly appealing because variant angina is a clinical model of acute coronary occlusion that may mimic the very early phase of myocardial infarction. Some autopsy6,7 and clinical reports8–14 have well described the pattern of LV and RV involvement in patients with inferior myocardial infarction. However, all these studies have been performed with some obligatory delay after the acute coronary event and, therefore, deal with the late effects of necrosis. Moreover, variable extent of coronary artery disease, presence of previous myocardial infarction, and possible LV and RV stunning are other elements that
may prevent the extension of these results to acute ischemia.

To date, no previous investigation has assessed in humans the effect of acute single-vessel coronary artery occlusion on both the LV and the RV studied simultaneously. To this purpose, patients with variant angina were evaluated by radionuclide angiography and thallium 201 scintigraphy during transient ischemia caused by vasospasm of the RCA or of the left anterior descending coronary artery (LAD). The prevalence of LV and RV dysfunction and the systolic interplay between the two ventricles were analyzed from the radionuclide data in relation to the involved vessel. 201TI scintigraphy provided the evidence of primary LV ischemia and the information on the septal involvement.

Methods

Study Population

From July 1982 to July 1987, 138 patients with the diagnosis of variant angina were admitted to our coronary care unit. Among them, 25 subjects (23 men, two women; mean age, 54 years) granted informed consent to enter the study protocol after fulfilling the following admission criteria: 1) angiographically documented normal coronary arteries or single-vessel disease involving the RCA or the LAD; 2) no clinical and electrocardiographic (ECG) evidence of previous myocardial infarction; 3) spontaneous or ergonovine-induced ischemic episodes promptly reversed by nitrate administration; 4) ECG during ischemia characterized by ST segment elevation (more than 0.1 mV) in the inferior leads in patients with normal coronary arteries or RCA disease (15 patients, group 1) or in the anterior leads in patients with normal coronary arteries or LAD disease (10 patients, group 2). Patients with critical stenosis in nonischemia-related vessels or with poor exercise tolerance according to history or during the effort test were excluded. All patients were submitted to routine cardiologic examination: maximal exercise stress test showed diagnostic ST segment depression in three patients. Ergonovine intravenous infusion was used to diagnose variant angina in patients with normal coronary arteries or with single-vessel coronary artery stenosis and a negative exercise test and was repeated to assess functional and flow patterns during ischemia. In such a population, with chest pain at rest, this is an accepted diagnostic test and has proven to be a safe procedure.15 Patients were continuously monitored in the coronary care unit by one-lead ECG while daily basal 12-lead ECG and daily blood samples for creatine kinase-MB determination were obtained, ruling out the occurrence of acute myocardial infarction.

Long-acting nitrates were discontinued 12 hours before each study, and sublingual nitroglycerin was allowed if anginal attacks occurred. No other drug was used during the study period.

Angiographic Study

Before admission to the study, all patients underwent selective coronary angiography in multiple views of both right and left coronary artery systems and biplane left ventriculography by the Judkins technique. Coronary artery injection of contrast medium during spontaneous or ergonovine-induced ischemia was performed in 11 patients. Angiograms were interpreted by an experienced cardiologist not aware of the clinical data and of the noninvasive results. Coronary artery stenosis or spasm was judged significant when the vessel’s lumen was restricted by more than 50%.

Scintigraphic Studies

Blood pool images were obtained by in vivo labeling of red blood cells by 0.03 mg/kg stannous agent (stannous chloride, Amersham, UK) followed 30 minutes later by 0.95 GBq (25 mCi) of technetium 99m pertechnetate. The scintigraphic data were collected with a standard large-field gamma-camera (Selo KR7, Turin, Italy) interfaced with a dedicated computer (Medusa 12B, Sepa, Turin, Italy) or with a small-field mobile camera (Apex 410M, Elscint, Haifa, Israel) both equipped with a high-resolution, parallel hole collimator. The study protocol included acquisitions in the anterior projection during control conditions and in the left anterior oblique “best septal” projection during control conditions, during spontaneous (three patients) or ergonovine-induced (0.05–0.2 mg i.v., 22 patients) ischemia and in the recovery phase, 2–4 minutes after isosorbide dinitrate (2–4 mg i.v.) administration or after spontaneous resolution. Acquisitions during ischemia lasted from 150 to 240 seconds, depending on the individual tolerance of ischemia, the occurrence of ventricular ectopic beats, or conduction disturbances. The gated acquisitions were stored in 16 (Medusa system) or 24 (Apex system) frames into a 64x64 matrix. A minimum of 100,000 counts/frame were always collected. Regional wall motion from radionuclide angiography was visually assessed by two independent observers with the help of parametric images (phase, amplitude, and stroke) for five LV regions (proximal and distal septal walls, inferoapical wall, and proximal and distal posterolateral walls) and four RV regions (proximal and distal anterolateral walls and inferior and apical walls). Normal wall motion, hypokinesia, and akinesia were scored 0, 1, and 2, respectively. Summed scores from the two observers provided final regional scores. The scores of all segments were then summed to obtain a wall motion abnormality index (WMI) for each ventricle in different conditions. The two observers agreed in the detection or the exclusion of new regional dysynergies during ischemia for the LV and the RV in 47 of 50 cases (94%). In the remaining three cases, the observers finally agreed upon the presence of regional ventricular ischemia. The regional involvement of each ventricle during ischemia was defined by a WMI increment (ischemic minus basal value) ≥2. LV and RV ejection fraction.
Figure 1. Angiograms of coronary anatomy during vasospasms (lower panels) and during basal conditions (upper panels) in three representative patients. Left panels: Incomplete vasospasm (middle third) in a nearly normal right coronary artery (patient 2). Middle panels: Complete vasospasm (middle third) superimposed on a 50% stenosis in the right coronary artery (patient 4). Right panels: Complete vasospasm (proximal third) superimposed on a 90% stenosis in the left anterior descending artery (patient 18).
(EF) values were obtained according to methods described previously.\textsuperscript{16,17} Computation of RV EF from radionuclide angiography data, critically influenced by different methodologic approaches, was validated in our laboratory using, as reference, the RV EF measurement from the first pass.\textsuperscript{18} The interobserver variability of RV and LV EF measurements at control and during ischemia showed a standard error of estimate lower than 5% ($r=0.97$). A fall in RV or LV EF greater than 5% during ischemia was considered pathologic. Myocardial perfusion was assessed during episodes of resting angina and at redistribution by $^{201}$TI scintigraphy. The tracer (1.5–2 mCi i.v.) was injected in 21 patients during episodes of spontaneous (four patients) or ergonovine-induced (17 patients) ischemia with electrocardiographic changes superimposable on those observed during the radionuclide angiography study. Scintigrams were obtained in left anterior oblique 40° and 70° and anterior projections starting within 8 minutes after $^{201}$TI injection and 3 hours later at control; 300,000 counts/frame were always collected for each view. In four patients, perfusion was not evaluated because the tracer was not available during the study period. Both the original and background-subtracted\textsuperscript{19} scintigrams were qualitatively assessed by two independent observers unaware of the clinical data; in case of discordance, the agreement was reached by a third observer.

During both scintigraphic studies, nine-lead ECG (standards, V$_2$, V$_4$, and V$_6$) were obtained every minute, and arterial blood pressure (cuff manometer) was measured every 2 minutes. A systematic evaluation of right precordial leads was not performed in this study.

Statistical Analysis

Scintigraphic data in different groups of patients are presented as the mean±SD. The analysis of variance for multiple comparison (Newman-Keuls procedure) was performed to identify differences among rate-pressure products or radionuclide angiography data obtained in different conditions. The same analysis allowed comparisons within each patient group and among different groups. A probability value less than 0.05 was considered significant.

Results

Clinical and Angiographic Findings

No patient experienced severe arrhythmias or clinical manifestations of heart failure during either spontaneous or ergonovine-induced ischemia. The ischemic episodes lasted no more than 5 minutes and were promptly interrupted by intravenous isosorbide dinitrate administration in 20 patients. In the remaining five patients, earlier spontaneous resolution occurred. The ergonovine test, when applied, always reproduced the spontaneous attack according to the electrocardiographic and hemodynamic responses. Rate-pressure products obtained during the acquisitions in ischemic conditions ($10.352±3.074$ during radionuclide angioscopy and $10.864±2.477$ during $^{201}$TI scintigraphy) were not significantly different from those calculated in basal conditions ($7.852±1.321$) and significantly lower than those reached during the exercise stress test ($24.743±6.530$) ($p<0.05$). The angiographic study showed normal or minimally abnormal coronary arteries in 14 patients, RCA disease in five (involving the middle third of the vessel in four and the distal third in one), and LAD disease in six (always involving the proximal third). The posterior descending coronary artery originated from the RCA in all patients. No detectable collateral circulation from the LAD to the RCA system could be demonstrated in group 1, whereas RCA to LAD collateral circulation was present in three patients of group 2. During the angiographic procedure, RCA spasm was documented in six patients (in five with normal coronary arteries and in one with RCA stenosis) and LAD spasm in five patients (in one with normal coronary arteries and in four with LAD stenosis). Spasms were located in the proximal third (one patient), in the middle third (three patients) or in the distal third (two patients) of the RCA, and in the proximal third of the LAD (five patients). Angiograms in Figure 1 show coronary vasospasms of different severity and location. Among the 25 patients, contrast ventriculography showed localized basal LV hypokinesia in 11 patients. The clinical and angiographic data, including the location of stenosis or spasm along the artery, are listed in Table 1.

Regional Wall Motion and Perfusion During Ischemia

Radionuclide angioscopy showed localized basal dyssynergies (hypokinesia) in the LV of seven patients and in the RV of eight patients. In group 1, during ischemia, LV and RV regional impairment (WMI increment ≥2) was demonstrated in 13 of 15 (87%) and in 14 of 15 (93%) patients, respectively, with a prevalent location of the dyssynergies in the inferoapical wall of the LV and the anterolateral and the inferoapical walls of the RV (Figure 2); the mean WMI increment during ischemia was comparable for the LV ($3.5±3$) and the RV ($4.5±2.1$). In group 2, LV and RV regional dysfunction occurred in 10 of 10 (100%) and two of 10 (20%) patients, respectively, with the systematic involvement of the interventricular septal wall (Figure 2); the mean WMI increment during ischemia was marked for the LV ($8±2.7$) and negligible for the RV ($0.8±1.7$). From the comparison of the two groups, as also shown in Figure 2, LV WMI increment during ischemia was significantly higher in group 2, whereas RV WMI increment was significantly higher in group 1.

LV perfusion was assessed in 13 of 15 patients of group 1 and in eight of 10 patients of group 2. Basal perfusion defects were detected in only three patients, all in group 1. During ischemia, new LV defects were documented in all patients of group 2, whereas no defects were detected in two patients of group 1 (patients 1 and 2). The concordance between radionuclide angioscopy and $^{201}$TI scintigraphy in
detecting or excluding LV wall motion abnormalities and perfusion defects during ischemia was 100%. A good agreement was also demonstrated in the location of ischemia to the septal wall (95%) and the inferoapical wall (86%) but not to the posterolateral wall (57%). The use of multiple projections in ²⁰¹Tl studies, which was not possible in the radionuclide angiography studies because of the short duration of ischemia, permitted the differentiation of inferior (five patients) from apical (five patients) perfusion defects or the determination of the simultaneous involvement of inferior and apical walls (six patients) in the two groups of patients and the detection of anterior wall defects (four patients of group 2). In no patient of both groups could RV perfusion be reliably evaluated from ²⁰¹Tl scintigrams.

**Quantitative Evaluation of Biventricular Function**

In group 1, 12 of 15 (80%) and 14 of 15 (93%) patients showed a pathologic decrease in LV and RV EF during ischemia, respectively; mean LV and RV EF significantly fell from 62±7% to 51±6% (p<0.05) and from 48±9% to 34±7% (p<0.01), respectively. In group 2, all patients showed a pathologic decrease in LV EF, whereas only one patient showed an abnormal change in RV EF during ischemia; mean LV EF significantly fell from 62±7% to 45±8% (p<0.01), whereas mean RV EF did not change significantly from 51±8% to 51±7%. The correlation between regional dyssynergies and global dysfunction during ischemia was examined. In group 1, a WMI increase was associated with an EF decrease in 10 of 15 patients (67%) for the LV and in 13 of 15 patients (87%) for the RV. In group 2, an LV WMI increase was associated with an LV EF decrease in all patients (100%), whereas RV EF increased or did not change in seven of eight patients (87%) without RV dyssynergies and in the two patients with RV WMI increment.

In both groups of patients, the early recovery phase was characterized by a full restoration of regional function (in all patients but one, patient 19, who showed a residual LV hypokinesia) and reversion of mean LV and RV EF to basal values (62±8% and
45±9%, respectively, in group 1) (60±11% and 52±14%, respectively, in group 2). The changes in mean EF values in the different conditions are represented in Figure 3.

**Septal Ischemia and Biventricular Function**

In six patients of group 1 (patients 10–15, Table 2) LV regional dysfunction during ischemia involved the interventricular septum and the inferoapical wall (Figure 4). Septal ischemia was also confirmed by the presence of septal perfusion defects in four of five patients studied by 201T1 scintigraphy. Results in patients with septal impairment, separately analyzed, showed a fall in LV EF and an increase in LV WMI that were significantly more pronounced than those documented in patients without septal involvement (patients 1–9, Table 2). By contrast, the fall in RV EF and the RV WMI increment were similar in both subgroups.

In group 2, all patients showed septal ischemia: the decrease in LV EF and the increase in LV WMI were similar to those documented in group 1 patients with septal impairment and significantly more pronounced than those in group 1 patients without septal involvement. RV EF and RV WMI, as described above, showed negligible changes during ischemia. Individ-

**FIGURE 3.** Plot of changes of mean left (LV EF) and right (RV EF) ventricular ejection fractions in the two groups (G.I and G.II) in basal conditions (B), during ischemia (I), and in the recovery phase (R). Inferior ischemia (solid circles) induced biventricular dysfunction, whereas anterior ischemia (solid squares) did not impair the systolic function of the right ventricle. *p<0.05; **p<0.01; n.s., not significant.

**FIGURE 2.** Bar graphs of frequencies of ischemic wall motion abnormalities are shown for the interventricular septum (SEPTUM), the inferoapical (INF-API) wall, and posterolateral (POST-LAT) wall of the left ventricle and for the anterolateral (ANT-LAT) wall and inferoapical (INF-API) wall of the right ventricle. Also, the increment of wall motion abnormality index (WMI) during ischemia is shown for the left and right ventricles. A significantly different extension of left and right ventricular involvement is observed in the two groups. *p<0.01.
ual EF values and WMI increments from basal to ischemic conditions are represented in Table 2.

Discussion

This is the first study in humans that describes the acute effect of single-vessel coronary artery occlusion on LV and RV regional and global function and on LV myocardial perfusion. The prevalence of LV and RV dysfunction and the systolic interplay between the two ventricles were evaluated and correlated with the coronary and myocardial site of absolute flow reduction. The close correlation between the site of ST segment elevation and the angiographic site of spasm\(^2\) ensured the identification of the ischemia-related vessel also in those patients without angiographic documentation of vasospasm.

We found that transient occlusion of a dominant RCA almost systematically caused RV regional impairment (93% incidence) that was associated, in most patients, with LV regional dysfunction (87% incidence). The extent of RV global impairment, as assessed by RV EF decrease during ischemia (93% of patients), was similar in all patients, whereas the extent of LV global impairment appeared related to the presence of septal ischemia (40% of patients). Conversely, transient occlusion of the LAD systematically induced obvious LV regional wall motion abnormalities and depression of LV EF; the RV impairment was absent or trivial in this group of patients with LAD spasm.

In our study, LV ischemic dyssynergies were always associated with LV perfusion defects that matched the site of wall motion abnormalities and the location of ST segment elevation. Similar evidence could not be obtained for the RV because of inadequate RV visualization. However, the presence of regional reversible wall motion abnormalities, associated with a reversible decrease in RV EF, during RCA but not during LAD spasm, provided indirect evidence for primary ischemia of the RV due to RCA transient occlusion.

No evidence for secondary LV or RV systolic impairment, due to mechanisms of "ventricular interference,"\(^{20,21}\) could be derived from EF measurements. In the patients with RCA spasm, the amount of LV EF decrease was not dependent on RV impairment but on the extension of LV ischemia. Conversely, either in patients with RCA or in patients with LAD spasm, the decrease in RV EF was not related to the LV and septal impairment but only to the presence of RV ischemia. In our study, no measurements of hemodynamic variables were
TABLE 2. Individual Values of Ejection Fraction and Wall Motion Abnormality Index for Each Ventricle

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<td>Mean±SD</td>
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<td>54±5</td>
<td>51±11†</td>
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| Group 2  |                |                  |                |                  |
| 10*      | 63             | 41               | 46             | 19               | 6               |
| 11*      | 68             | 56               | 50             | 29               | 4               |
| 12*      | 60             | 48               | 47             | 26               | 7               |
| 13*      | 59             | 43               | 47             | 36               | 5               |
| 14*      | 75             | 50               | 38             | 29               | 6               |
| 15*      | 51             | 45               | 42             | 35               | 4               |
| Mean±SD  | 63±8†          | 47±5             | 45±4†          | 29±6             | 5.3±1.2         |

| Group 2  |                |                  |                |                  |
| 16       | 71             | 52               | 38             | 39               | 0               |
| 17       | 61             | 40               | 49             | 48               | 0               |
| 18       | 56             | 37               | 55             | 51               | 0               |
| 19       | 55             | 39               | 48             | 45               | 0               |
| 20       | 64             | 39               | 55             | 52               | 0               |
| 21       | 60             | 54               | 53             | 56               | 0               |
| 22       | 57             | 48               | 57             | 64               | 4               |
| 23       | 54             | 38               | 44             | 46               | 0               |
| 24       | 71             | 60               | 44             | 48               | 4               |
| 25       | 75             | 45               | 64             | 58               | 0               |
| Mean±SD  | 62±7†          | 45±8             | 8±2.7          | 51±8 NS          | 51±7            | 0.8±1.7         |

EF, ejection fraction; WMI Increment, increment of wall motion abnormality index from basal to ischemic conditions; NS, not significant.  
*Patients of group 1 with documented septal ischemia.  
†p<0.01. Comparison of means for LV WMI increments: patients 1–9 vs. 10–15 and patients 1–9, vs. 16–25 (p<0.01); patients 10–15 vs. 16–25 (NS). Comparison of means for RV WMI increments: patients 1–9 vs. 16–25 (p<0.05); patients 10–15 vs. 16–25 (p<0.01); patients 1–9 vs. 10–15 (NS).

obtained to assess the hemodynamic severity of LV dysfunction; however, preliminary findings from the hemodynamic monitoring performed in a similar population confirm the biventricular impairment during RCA but not during LAD spasm and exclude a concomitant negative effect of LV dysfunction on RV performance during LAD transient occlusion.22

Right Coronary Artery Spasm and Right Ventricular Myocardial Infarction

Autopsy studies6,7,23 in coronary care unit populations have shown that RV infarction is relatively common (66% maximal incidence) and almost exclusively associated with posterior and septal LV necrosis. However, the diagnosis of acute RV infarction by means of hemodynamic, echocardiographic, or scintigraphic criteria8–14,24 is less frequent, possibly because of the different sensitivity of clinical techniques.24,25

In the present study, we observed in patients with RCA spasm an almost constant abnormal response of the RV during ischemia (93% of the patients). One explanation for these apparently contrasting findings may be that our clinical model allowed us to study the effects of coronary occlusion immediately after its occurrence. By contrast, patients with myocardial infarction were studied with some obligatory delay after the onset of the coronary event. The effects of transient myocardial ischemia may not be the same as those occurring during prolonged periods of coronary
occlusion. The changes in the topography of infarcted zones can be different from that of the ischemic zones and may thus influence the functional response differently. Furthermore, there is a well-documented tendency of RV function to improve in the phase immediately after inferior myocardial infarction that is possibly related to recovery of a “stunned” RV. It is conceivable that, as in our population, RV dysfunction frequently occurs in the very early phase of inferior myocardial infarction before the patient is referred to medical attention or submitted to instrumental evaluation. A second explanation may be the better sensitivity of radionuclide angiography in detecting transient changes of regional and global RV function in the patient who serves as his own control.

A second question arising from our data is related to the role of LV involvement in inferior infarction. Recently, some investigators stressed the negative prognostic value of LV impairment in this setting, which included those patients with early hemodynamic evidence of prevalent RV failure. Our results in transient ischemia support the relevance of ischemic LV impairment due to RCA occlusion. During RCA spasm, with RV dysfunction, 40% of patients showed interventricular septal ischemia and marked, “primary” LV impairment. In these patients, the LV dysfunction was quantitatively similar to that caused by LAD spasm. This condition may be even more severe during inferior infarction in patients with multivessel disease, which could limit the compensatory response from areas not involved in the necrosis; moreover, a low RV output may further affect perfusion of the left coronary artery system and precipitate cardiogenic shock.

**Inferior Variant Angina and Stable Angina**

In the last decade, several studies have demonstrated the presence of an abnormal response of RV EF to exercise in patients with RCA disease. Whether the RV function is primarily affected by local ischemia or related to LV dysfunction is still debated. Berger et al reported that the major determinant of an abnormal exercise RV response is represented by a concomitant impairment of LV function; other studies suggest that the RV systolic performance during exercise is influenced by the impairment of LV blood supply and by exercise-induced changes in RV afterload.

The prevalence of RV “primary” ischemia and the systolic interplay between the two ventricles could be inferred in our study from the comparison between RCA and LAD spasm. We documented 1) a very high prevalence of RV ischemic impairment in patients with inferior variant angina and 2) an inconsistent RV systolic dysfunction secondary to LV ischemia in patients with anterior variant angina.

Vasospasm causes absolute blood flow reduction, whereas demand-induced ischemia is responsible for an inadequate increment of blood flow during stress.

These pathophysiologic differences may explain the lower threshold in the detection of ischemia in angina at rest that may, in turn, “amplify” the signal of RV dysfunction, which is not easily detectable in the classic form of angina. Accordingly, a higher incidence of severe RV dysfunction in our patients with RCA spasm was observed compared with that in patients with RCA disease and stable effort ischemia.

**Limitations**

In this study, the short duration of the explored event (few minutes) imposed the use of a single projection during the blood pool gating acquisitions. The incomplete exploration of RV and LV walls would have caused underestimation of wall motion abnormalities and may explain some discrepancies between the change of EF values and WMI from basal to ischemic conditions. As a matter of fact, patients 1 and 2 showed a decrease in LV EF, and patient 8 showed a decrease in RV EF, but these three did not show an increase in WMI during ischemia. Conversely, localized dyssynergy, probably unable to induce global LV and RV dysfunction, was not detected by EF measurements (patients 4 and S and patients 22 and 24, respectively). The use of multiple views, not feasible in the present study because of the shortness of ischemia, would prevent possible discrepancies and provide a proper separation of different walls. This limitation, however, has been settled in all studies dealing with radionuclide angiography during transient ischemia by the compromise of using the single left anterior oblique 40° projection.

Some anatomic factors that could have influenced the biventricular response to ischemia were not fully investigated in the present study. The location along the artery, as well as the degree of the RCA vasospasm, may determine the presence and extent of RV involvement. This information is not available in all patients of this series with inferior ischemia; however, in all six patients with angiographically documented RCA spasm, similar RV dysfunction was documented without regard to the degree and location of coronary vasoconstriction.

As demonstrated during coronary vasospasm or by coronary angioplasty, the status of collaterals from the contralateral vessel during coronary occlusion may be different from that detected in basal conditions and could influence the functional response to ischemia in patients with normal coronary arteries or single-vessel disease. Basal collateral circulation was detected in only three of 25 patients, all in group 2. Unfortunately, we were not able to inject contrast medium into contralateral vessels during ischemia because of the need to first visualize the vasospastic artery and then to rapidly interrupt the episode. However, the occurrence of similar RV dysfunction in almost all patients with RCA spasm probably rules out any determining influence of transient collateral filling on RV performance during ischemia.
In conclusion, in our study population, transient RCA occlusion almost systematically induced RV dysfunction; an important LV dysfunction was also observed in 40% of these patients. Conversely, transient LAD occlusion did not affect RV systolic function. Thus, in patients referred to intensive coronary care unit because of ST segment elevation in the inferior leads, an early evaluation of biventricular function is recommended.

Acknowledgment
We are indebted to Mr. Antonio Fontana for invaluable technical assistance in the acquisition of scintigraphic data.

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KEY WORDS * myocardial function * coronary vasospasm * radioisotopes * ventricular function
Behavior of right and left ventricles during episodes of variant angina in relation to the site of coronary vasoospasm.
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Circulation. 1990;81:567-577
doi: 10.1161/01.CIR.81.2.567

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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