

Doppler Left Ventricular Flow Pattern Versus Conventional Predictors of Left Ventricular Thrombus After Acute Myocardial Infarction

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Objectives. The value of Doppler-derived left ventricular spatial flow patterns in predicting left ventricular thrombus formation after myocardial infarction was compared with that of conventional clinical and echocardiographic variables.

Background. Assessment of left ventricular thrombosis risk after myocardial infarction is important because of potential embolic sequelae that are reduced by oral anticoagulant agents.

Methods. Clinical, two-dimensional and Doppler echocardiographic data were prospectively obtained in 104 patients with acute myocardial infarction within 48 h of admission. Ventricular flow was assessed by Doppler echocardiography and considered normal when brisk ventricular inflow with simultaneous onset at the mitral valve and apical levels was present, together with alternating directions of apical flow throughout the cardiac cycle. In addition to normal flow, two abnormal flow patterns were

recognized: apical rotating flow and vortex ring formation. Oral anticoagulant agents were prescribed only to patients with abnormal flow at admission. The incidence of left ventricular thrombosis was assessed by echocardiography during 9 months of follow-up.

Results. Abnormal flow pattern had a positive predictive value of 63% and a negative predictive value of 99%. On stepwise logistic regression analysis, only abnormal flow pattern had an independent relation to left ventricular thrombus (odds ratio 92).

Conclusions. Left ventricular flow pattern derived by Doppler echocardiography soon after admission is superior to conventional clinical and two-dimensional echocardiographic assessment in estimating the risk of left ventricular thrombosis after myocardial infarction.

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Abnormal spatial patterns of mitral inflow develop in impaired left ventricles, which may be characterized by Doppler echocardiography (1-9). Abnormal Doppler flow is related to the risk of ventricular thrombosis, as was shown in clinical studies (5,6,10). Risk assessment of left ventricular thrombus by Doppler echocardiography is important for optimal management of oral anticoagulant agents, which decrease embolic sequelae but also introduce the risk of bleeding (11).

To determine the additive value of Doppler flow examination, its accuracy for predicting ventricular thrombus after myocardial infarction was compared in the present study with that of clinical and two-dimensional echocardiographic variables conventionally used to estimate risk of thrombus.

Methods

Patient selection. Between September 1991 and February 1993, 315 consecutive patients with acute myocardial infarction were screened for study eligibility by review of coronary care

admission logs and patient charts. The diagnosis was based on a combination of prolonged chest pain, evolutionary electrocardiographic (ECG) changes (QRS complex or ST-T wave, or both) and an increase in cardiac enzymes to more than twice normal (4 U/liter for creatine kinase [CK]-MB isoenzyme). Exclusion criteria were prospectively defined: Patients with indications or contraindications for anticoagulation (including admission left ventricular thrombus) were not eligible (n = 74), nor were patients with aortic regurgitation (n = 34) or obstructive mitral valve pathology (n = 2) because these may influence left ventricular flow. Major illness or other factors potentially interfering with follow-up (n = 31) also were exclusion criteria. An explicit age limit was not used. Patients with technically poor echocardiograms were also excluded (n = 31). Eight patients died before admission echocardiography, and in 10 patients, echocardiography was delayed because infarction was not initially recognized. For other reasons (logistics, refused consent, participation in other trials), an additional 21 patients were excluded.

Follow-up. Patients attended follow-up at 1.5, 3, 6 and 9 months after discharge. During analysis, patients terminating follow-up prematurely but having developed thrombus beforehand were considered to have complete follow-up (n = 6). Follow-up was ended because of death (n = 4), admission to another hospital with recurrent myocardial infarction (n = 3) or loss to follow-up (n = 5) or in patients with normal left ventricular flow who were prescribed oral anticoagulant agents

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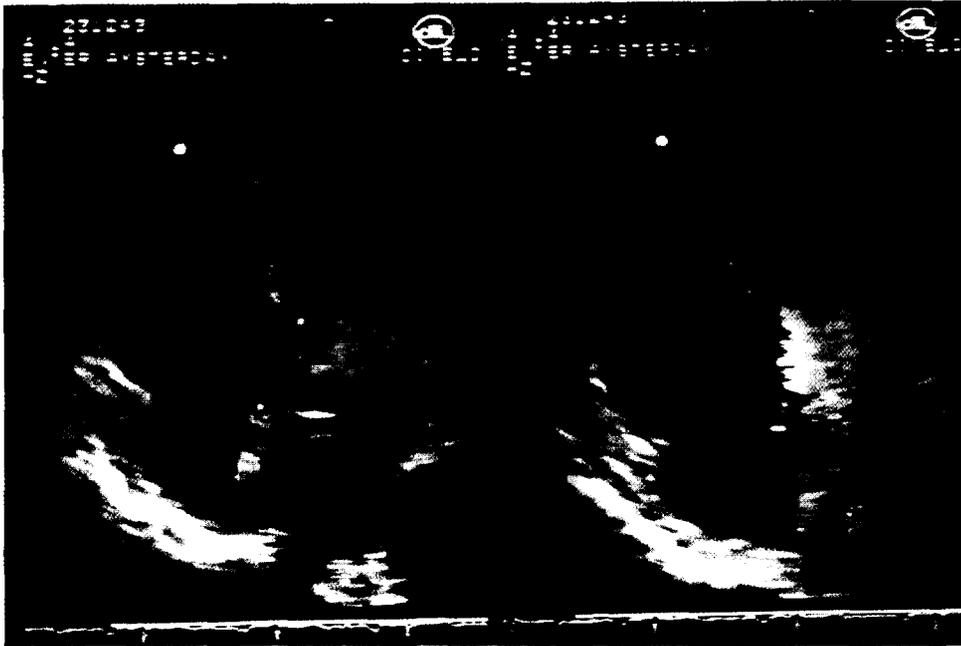


Figure 1. Normal left ventricular flow dynamics on color flow imaging. Blood motion toward the **top** of the image is encoded in **red** and that toward the **bottom** of the image in **blue**. **Left panel**, In early diastole, the entire left ventricular cavity is encoded in **red**. **Right panel**, During systole, the left ventricle is completely encoded in **blue**. Simultaneous onset of inflow throughout the left ventricle and discontinuous apical flow directions are the hallmarks of normal flow.

(e.g., after coronary artery bypass surgery, occurrence of atrial fibrillation) ($n = 7$).

All patients gave informed consent, and the study was approved by the hospital medical ethics committee.

Clinical data. Maximal serum CK-MB levels, measured every 4 h, were used as an index of infarct size. Electrocardiographically, infarctions were categorized as anterior, inferoposterior or indeterminate (e.g., in left bundle branch block) (12). Infarctions were classified as first or recurrent according to the patient's history supplemented with clinical records. Heart failure during coronary care stay was classified according to clinical criteria (13).

Echocardiography. An Ultramark 9 ultrasound system (Advanced Technology Laboratories) with 2.25- and 3.5-MHz phased-array transducers was used, and examinations were recorded on videotape. The first echocardiogram was obtained within 48 h after admission to determine flow pattern. Follow-up echocardiograms to determine thrombosis were obtained at discharge from the coronary care unit and at follow-up visits up to 9 months after admission.

Two-dimensional echocardiography. Thrombus was defined as a mass distinct from the endocardium, and with an echo density different from that of myocardium, visualized in at least two orthogonal planes in an area of myocardial asynergy (14). To discriminate from artifact, multiple gain settings were used together with a shallow depth of field. At admission, left ventricular volumes were measured by the apical biplane Simpson's rule using the ultrasound system's standard calculation software. The left ventricular wall motion score index was obtained in a 16-segment model according to established methods (15). A score of 1 to 4 was assigned to normokinetic, hypokinetic, akinetic and dyskinetic segments, respectively. To determine the influence of apical asynergy, the score of the

four apical segments was summed and divided by 4, thus yielding the apical wall motion score index.

The mitral annulus was defined as the hinge point of the mitral leaflets and measured in triplicate in the apical four- and two-chamber views at maximal diastolic diameter just before onset of atrial contraction (16). The annular area was calculated assuming an ellipsoid annular shape.

Flow pattern examination. Left ventricular flow was investigated by pulsed wave Doppler, two-dimensional color flow mapping and color M-mode echocardiography. Low wall filter settings (100 to 200 Hz) enabled detection of low flow velocities (± 3 to 6 cm/s).

Definitions of normal and abnormal flow patterns were based on our previous experience (3-5) and are consistent with experimental and clinical echocardiographic studies (1,2,6-9,17). **Normal flow** was defined as simultaneous onset of inflow throughout the left ventricle together with discontinuous apical flow. On two-dimensional color-flow recordings, inflow was considered normal when the left ventricle was completely encoded in red in early diastole (Fig. 1, left panel). On color M-mode echocardiography, a nearly vertical flank of early diastolic inflow indicated normal onset of inflow.

Discontinuity of left ventricular apical flow was present on two-dimensional color flow when the apex was completely encoded in blue during systole without persistence of apical red (Fig. 1, right panel), when color M-mode echocardiography showed alternating apical red and blue, and pulsed Doppler showed alternating direction of apical flow during the cardiac cycle. For pulsed Doppler and color M-mode echocardiography, sample locations near the ventricular septum and near the lateral free wall were used.

Two distinct abnormal flow patterns can be recognized (3-5): apical rotating flow and vortex ring formation (Fig. 2).

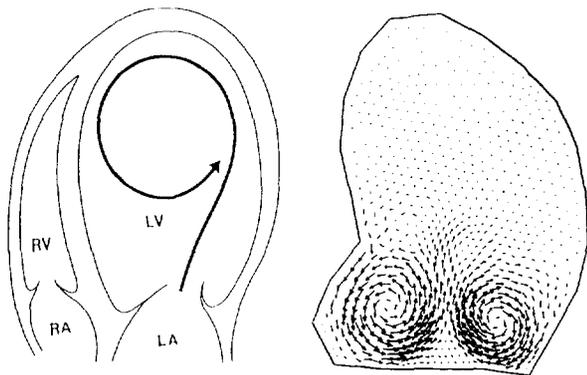


Figure 2. Schematic representation of abnormal flow patterns. **Left,** Apical rotating flow. Continuous counterclockwise whirling of blood in the left ventricular (LV) apex. **Right,** Vortex ring formation. Ringlike structure of blood moves into the left ventricular cavity. Ring consists of blood spinning toward the left ventricular apex in the center and blood spinning in the direction of the mitral valve at the periphery. LA (RA) = left (right) atrium; RV = right ventricle.

Apical rotating flow was characterized by holosystolic persistence of apical red near the lateral wall on color flow recordings (Fig. 3, inset). It was indicated on color M-mode echocardiography by a continuous apical red band with the M-mode cursor near the lateral wall (Fig. 3, left panel) and a corresponding blue continuous band with the cursor near the septum (Fig. 3, right panel). On pulsed Doppler, apical rotating flow was evident by a continuously positive flow signal near the lateral wall and a continuously negative signal near the septum.

Vortex ring formation was considered present when a complex consisting of central red flanked on both sides by blue was seen to move in diastole from the mitral valve to the apex on color flow recordings (Fig. 4), confirmed by upwardly sloping blue bands during diastole on color M-mode recordings with the cursor positioned through the blue areas.

Patients were classified into groups with normal and abnormal flow without consideration of the specific abnormal flow pattern involved. The intraobserver and interobserver reproducibility of flow pattern determination in our institution are 88%.

The mitral velocity profile was obtained with the pulsed Doppler sample volume at the mitral annulus. Because mitral annular diastolic area was similar in the patients with and without thrombus during follow-up (6.42 ± 1.36 vs. 6.65 ± 1.12 cm², $p = 0.5$), the mitral time-velocity integral was used as an index of mitral stroke volume.

Anticoagulation and thrombolysis. All patients received subcutaneous heparin (5,000 U twice daily) to prevent venous thromboembolism. Patients were treated with anticoagulant agents (acenocoumarol or phenprocoumon) immediately after detection of abnormal flow because we previously found them at risk for left ventricular thrombus, and oral anticoagulation has been advised in patients at such risk (5,18,19). The international normalized ratio was maintained between 2.5 and

4.0 and monitored by a specialized outpatient thrombosis service. Patients with normal flow received platelet inhibitors at the discretion of their cardiologist. Pharmacologic thrombolysis followed by intravenous heparin (48 h) was administered according to standard criteria at the discretion of the responsible clinicians.

Statistical analysis. All data were obtained prospectively. Continuous variables are presented as mean value \pm SD and were tested for significance by a two-sided Student *t* test. Dichotomous variables were analyzed in two-by-two tables and tested for statistical significance by the Fisher exact test. Significance was assumed for $p < 0.05$. Analysis was by intention to treat.

For comparison of predictive accuracy of Doppler flow data, clinical and echocardiographic variables used to predict the risk of mural thrombosis were identified by review of published data (20-28). Sensitivity and specificity were calculated at several values of the variable investigated, and the optimal cutoff, yielding the highest combined sensitivity and specificity, was determined in receiver operating characteristics curves. Predictive values were subsequently calculated at this cutoff.

In a second analysis, logistic regression analysis was used to identify variables with an independent association with left ventricular thrombosis after myocardial infarction. First, the relation of pathogenetically important variables was determined using univariate analysis. Subsequently, the variables with $p < 0.1$ in this univariate analysis were entered into a stepwise logistic regression model. For this analysis, standard statistical software was used (BMDP-LR release 7.0, BMDP Statistical Software Inc.). These data are reported as odds ratios and their 95% confidence intervals.

Results

At admission, 35 patients had abnormal flow, 22 of whom developed thrombus. Apical rotating flow was found in 32 (19 developed thrombus), vortex ring formation in 1 (who developed thrombus) and both flow patterns in 2 (both developed thrombus). Sixty-nine patients had normal flow, one of whom had a subsequent thrombus. The predictive value of Doppler flow data is shown in Table 1. Results of univariate analysis of risk factors for thrombosis are shown in Table 2. Patients with thrombosis during follow-up more frequently had an abnormal flow pattern at admission. Associations of thrombosis were also found with ECG anterior infarct localization and enzymatic infarct size, together with greater left ventricular dilation. At admission, both global left ventricular function—as reflected by wall motion score index, left ventricular ejection fraction and mitral time-velocity integral—as well as regional apical function were significantly more impaired in patients with thrombus during follow-up. Recurrent infarction was more frequent in patients with thrombus, but this was of marginal statistical significance only.

On stepwise logistic regression analysis, the flow pattern shortly after admission was the only independent predictor of

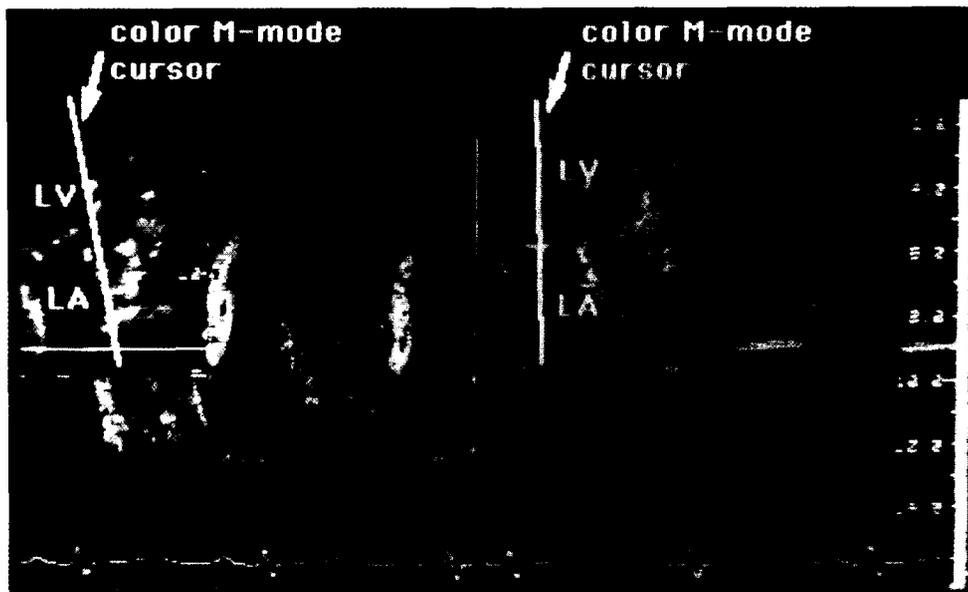


Figure 3. Color M-mode recordings of apical rotating flow. **Left panel,** With M-mode cursor along the lateral wall (**inset**), continuous **red band** is recorded. **Right panel,** With the cursor near the ventricular septum (**inset**), a corresponding continuous **blue band** is evident. Abbreviations as in Figure 2.

thrombus during follow-up, with an odds ratio of 92 (95% confidence interval 11 to 782).

Flow pattern during follow-up. Sixty-two (90%) of 69 patients had normal flow throughout the study. In two patients follow-up was unavailable, and five (7%) developed abnormal flow in association with significant worsening of left ventricular wall motion score index ($+0.38 \pm 0.17$, $p = 0.001$) despite thrombolysis in four.

Only one patient with normal flow at admission subsequently developed thrombus. After thrombolysis for a large anterior infarction, flow was normal on admission but became abnormal before detection of a small mural thrombus 6 weeks

after discharge. This was associated with progressive left ventricular dilation and decreasing ejection fraction. During follow-up without oral anticoagulant agents, no evidence of peripheral embolism was found in this patient.

Twenty-four (69%) of 35 patients had abnormal flow throughout the study. In three patients follow-up was unavailable, and in eight (23%) the flow pattern normalized. All eight had undergone thrombolysis, and a trend toward improvement of the wall motion score index was found (-0.47 ± 0.59 , $p = 0.06$).

Embolitic events. Possible embolic events were reported by two patients. The first patient had an anterior infarction with

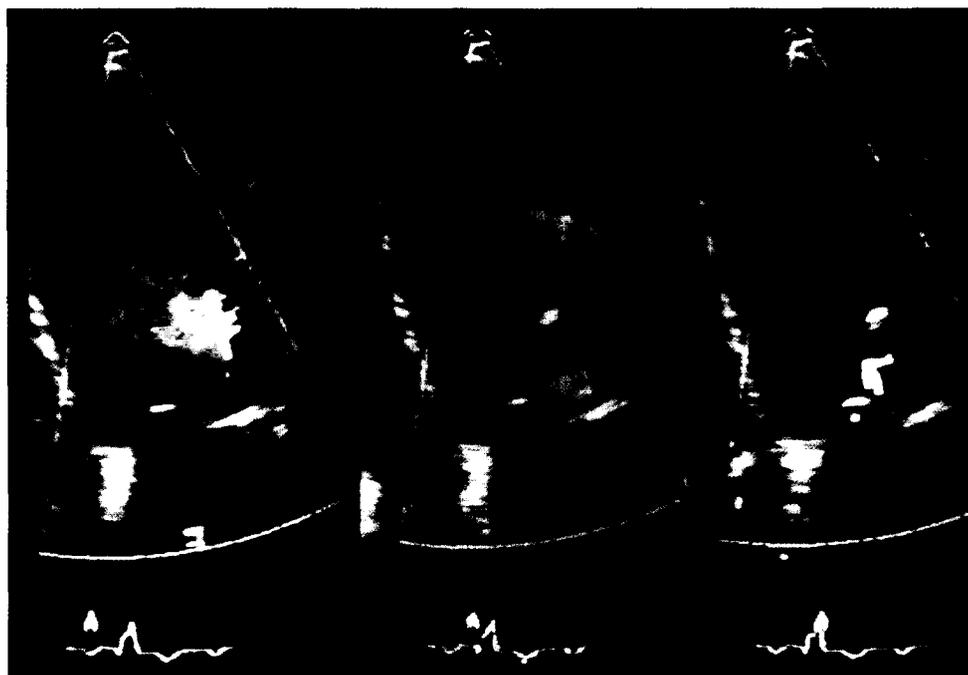


Figure 4. Color flow images of vortex ring formation. These three sequential frames show motion of vortex ring, consisting of central **red** flanked on both sides by **blue**, from the mitral valve toward the apex.

Table 1. Value of Left Ventricular Spatial Flow Pattern for Prediction of Left Ventricular Thrombosis After Myocardial Infarction Compared With Conventional Criteria

	Sens	Spec	PPV	NPV	RR (95% CI)	p Value
Abnormal flow	96	84	63	99	43 (6-309)	<0.001
Anterior infarction	70	64	36	88	2.9 (1.3-6.5)	<0.01
CK-MB >120 U/liter	52	77	38	86	2.7 (1.3-5.6)	<0.05
Killip class >1	48	84	46	85	4 (1.5-5.9)	<0.01
Anterior infarction and CK-MB >120	43	89	85	53	3.4 (1.8-6.6)	<0.005
Anterior infarction and Killip class >1	26	93	50	82	2.7 (1.3-5.5)	<0.05
WMSI ≥ 2	87	81	57	96	12.8 (4.1-40)	<0.001
Anterior infarction and WMSI ≥ 2	57	88	57	88	4.6 (2.3-9.1)	<0.001
≥ 2 apical akinetic or dyskinetic segments	91	71	48	97	13.8 (3.4-55.9)	<0.001
Anterior infarction and ≥ 2 apical akinetic or dyskinetic segments	48	90	58	86	4.1 (2.1-7.9)	<0.001

CI = confidence interval; CK = creatine kinase; NPV (PPV) = negative (positive) predictive value; RR = relative risk; Sens = sensitivity; Spec = specificity; WMSI = wall motion score index.

abnormal flow after admission. Despite treatment with anticoagulant agents, thrombus was documented. Nine months after the index admission, he was admitted to another hospital with a reversible focal neurologic deficit. The second patient had an anterior infarction with normal admission flow that converted to abnormal flow during follow-up. There was no echocardiographic evidence of thrombus, and he was followed up without anticoagulant agents. After undergoing emergent cholecystectomy at another hospital 6 months after the index infarction, sudden peripheral vascular compromise necessitated femoral embolectomy. No data were available regarding recurrence of myocardial infarction or presence of thrombus in the perioperative phase of cholecystectomy.

Table 2. Univariate Analysis of Variables Important in Left Ventricular Thrombus After Myocardial Infarction

	LVT Present (n = 23)	LVT Absent (n = 81)	p Value
Abnormal flow	22 (96%)	13 (16%)	<0.001
LV EDV (ml)	107 \pm 25 (61-158)	88 \pm 26 (38-177)	<0.005
LV ESV (ml)	66 \pm 21 (34-103)	43 \pm 19 (12-100)	<0.001
LV EF (%)	39 \pm 10 (13-59)	53 \pm 12 (28-75)	<0.001
WMSI	2.15 \pm 0.23 (1.72-2.67)	1.57 \pm 0.33 (1-2.33)	<0.001
Apical WMSI	3.07 \pm 0.5 (1.75-4)	1.73 \pm 0.78 (1-3.75)	<0.001
TVI (cm)	9 \pm 2 (5-13)	11 \pm 3 (5-20)	<0.005
CK-MB (U/liter)	166 \pm 100 (31-430)	88 \pm 65 (11-374)	<0.001
Anterior infarction	17 (74%)	39 (48%)	<0.05
Q wave infarction	18 (78%)	48 (59%)	0.2
Recurrence	9 (39%)	15 (19%)	0.08
Thrombolysis	16 (70%)	50 (62%)	0.7

Data presented are mean value \pm SD (range) or number (%) of patients. EDV (ESV) = end-diastolic (end-systolic) volume; EF = ejection fraction; LV = left ventricular; LVT = left ventricular thrombus; TVI = time-velocity integral of mitral inflow; other abbreviations as in Table 1.

Discussion

The main finding of this study is that abnormal left ventricular flow at admission is the only independent correlate of left ventricular thrombosis after myocardial infarction. Although significant differences were present on univariate analysis, no independent associations were found for any of the other risk factors examined.

The predictive accuracy of clinical or echocardiographic variables used to predict left ventricular thrombus was lower than the accuracy of Doppler flow data, which thus appears to provide better information regarding thrombosis risk. This method also has other practical advantages: It is a single examination (as opposed to the combinations of clinical and echocardiographic variables that previously have been proposed) and can be performed soon after admission, allowing early stratification of left ventricular thrombosis risk. This is important because the risk of cardiogenic embolism is highest in the early period of myocardial infarction.

Pathogenesis of thrombus after myocardial infarction.

Our observations, together with the finding that abnormal flow preceded thrombus formation, support the causal role of abnormal flow in ventricular thrombosis after myocardial infarction. Presumably, changes in left ventricular contractility, volume and shape after myocardial infarction induce abnormal flow patterns, causing stasis of blood in the apex and resulting in thrombus formation.

Influence of thrombolytic therapy. On univariate analysis no influence of thrombolysis on the frequency of thrombosis was found (Table 2). However, all patients whose flow converted from abnormal to normal had undergone thrombolysis, with a trend to improvement in global left ventricular function. Conversion of normal flow to abnormal was associated with deterioration of left ventricular function. Of note, the majority

of these patients had also undergone thrombolysis. No coronary angiographic data were available, but it is tempting to speculate that these differences resulted from successful and failed reperfusion, respectively.

Study limitations. All patients with abnormal flow were assigned to receive oral anticoagulant agents for reasons stated previously. This may have decreased the incidence of thrombosis in abnormal flow. However, any increase in incidence of left ventricular thrombosis in patients with abnormal flow by withholding oral anticoagulant agents would only have improved the predictive value of abnormal flow.

Conclusions. Abnormal spatial distribution of left ventricular flow was the only independent correlate of ventricular thrombus after myocardial infarction in our study. Its predictive accuracy was superior to that of clinical and two-dimensional echocardiographic variables. This confirms the pathogenetic significance of flow disturbances in thrombus formation. Furthermore, these findings substantiate the clinical relevance of Doppler flow determination after myocardial infarction, with important consequences for management of oral anticoagulant agents. The management implications of changes in flow pattern during follow-up remain to be defined.

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