The effects of antithrombotic drugs in patients with left ventricular thrombi: assessment with indium-111 platelet imaging and two-dimensional echocardiography

JOHN R. STRATTON, M.D., AND JAMES L. RITCHIE, M.D.

ABSTRACT Patients with left ventricular thrombi not caused by recent myocardial infarction were prospectively studied by indium-111 platelet imaging and two-dimensional echocardiography to determine the reproducibility of these techniques and the short-term effects of sulfinpyrazone (200 mg four times daily), aspirin (325 mg three times daily) plus dipyridamole (75 mg three times daily), and full-dose warfarin. At baseline, all patients underwent indium-111 platelet imaging and echocardiography, and the results were positive for thrombus. In six patients on no antithrombotic drug therapy, repeat platelet scans and echocardiographic studies at 6.0 ± 3.3 weeks remained positive and were unchanged. In seven patients studied on sulfinpyrazone, three platelet scans became negative, two became equivocal, and two were unchanged; the presence and size of thrombus was constant by echocardiography in all seven patients. Of the six patients studied on aspirin plus dipyridamole, one platelet scan became negative, those of three became equivocal, and two were unchanged; all echocardiographic findings remained positive, but one patient had decreased thrombus size. Among four warfarin-treated patients, three had resolution of platelet deposition and one was unchanged; by echocardiography, thrombus resolved in one patient, was decreased in size in one, and was unchanged in two. We conclude that, in the absence of antithrombotic drug therapy, platelet imaging and echocardiographic findings are stable in patients with left ventricular thrombi not caused by recent myocardial infarction. Sulfinpyrazone, aspirin plus dipyridamole, and warfarin all interrupt platelet deposition in some patients with chronic left ventricular thrombi. The hematologic activity of ventricular thrombus, i.e., the extent of ongoing platelet incorporation, may be inhibited by short-term drug therapy without significant changes in echocardiographically measured thrombus size.


PATIENTS with left ventricular mural thrombi are at risk for systemic embolism. However, appropriate identification of and therapy for such patients remain uncertain, largely because reliable methods of thrombus detection during life have not been previously available. Two recently developed noninvasive techniques, indium-111 platelet imaging and two-dimensional echocardiography, can now be used for the diagnosis of thrombi and for the assessment of antithrombotic drug therapy. These two methods of thrombus detection offer distinctly different pathophysiologic information. Platelet imaging, which detects ongoing platelet deposition on the thrombus surface, can estimate left ventricular thrombus activity; echocardiography, which produces anatomic images of thrombi, can estimate thrombus size. Thrombus resolution after anticoagulation with warfarin has been shown after acute myocardial infarction. However, the effects of warfarin, which inhibits fibrin deposition, have not been studied in patients with left ventricular thrombi present for months to years after myocardial infarction. The frequent detection of platelet deposition by indium-111 platelet imaging in such patients implies that ventricular thrombus formation is also partially dependent on platelet mechanisms. The effects of platelet inhibitors on left ventricular thrombi have not been prospectively evaluated. Unlike warfarin, these drugs are quite safe and are easily administered.
The purposes of this study were to define the reproducibility of results from platelet imaging and echocardiography for the detection of left ventricular thrombi in patients without recent myocardial infarction and to prospectively determine the effects of short-term antithrombotic drug therapy by these noninvasive techniques. Our results suggest that platelet inhibitors or warfarin inhibit left ventricular thrombus activity in some patients and that platelet imaging is more likely than echocardiography to detect short-term drug effects.

Methods

Subjects. Sixteen patients with left ventricular thrombi were prospectively studied to determine the reproducibility of results from platelet imaging and echocardiographic examination and the short-term effects of sulfinpyrazone, aspirin plus dipyridamole, or full-dose warfarin on left ventricular thrombi. Patients were identified by review of all two-dimensional echocardiograms performed in our laboratory. Patients with a two-dimensional echocardiographic study that was definitely positive for left ventricular thrombus were asked to undergo indium-111 platelet imaging. Patients were excluded when they had a recent (<4 months) myocardial infarction, were receiving anticoagulants or platelet inhibitors, or had a technically inadequate echocardiogram. No patients were excluded because of technically inadequate platelet images; two patients with positive echocardiographic findings were excluded because of negative baseline results of platelet imaging. In our experience and that of Ezekowitz et al., platelet imaging has been 100% specific for the detection of thrombi; i.e., there have been no studies in which the results were false positive. Similarly, the specificity of an echocardiographic study that is definitely positive for the detection of thrombus has been high (95%) in our laboratory. All patients were men with a mean age of 58 years (range 27 to 80). Fifteen of the 16 patients had definite or probable evidence of prior myocardial infarction. Fourteen patients had definite transmural infarctions by electrocardiographic Q wave criteria (anterior in 10, anterior plus inferior in three, and lateral in one), and one patient had a probable infarction evidenced by left bundle branch block and an anterior-apical aneurysm. At the time of initial study, the mean time after infarction was 20 ± 15 (SD) months (range 4 to 60). The one patient without evidence of prior infarction had normal coronary arteries, a normal ejection fraction, normal cardiac volumes, and no regional wall motion abnormalities, but had left ventricular thrombus documented by biopsy at cardiac surgery (patient 14).

These 16 patients participated in four types of studies. The first study comprised six patients who underwent repeat platelet imaging and echocardiography and who were not taking any antithrombotic drugs (warfarin, heparin, aspirin, dipyridamole, sulfinpyrazone, indomethacin, or other nonsteroidal antiinflammatory drugs); this was done to determine the reproducibility of results from platelet imaging and echocardiographic examination. The mean interval between studies was 6.0 ± 3.3 weeks (range 3 to 12). Four of these patients (Nos. 1, 8, 9, and 10) subsequently underwent drug studies, and two (patients 15 and 16) did not. To assess the effects of short-term antithrombotic drug therapy, patients were prospectively studied by platelet imaging and echocardiography at baseline and while receiving sulfinpyrazone (200 mg four times daily, seven patients), aspirin (325 mg three times daily) plus dipyridamole (75 mg three times daily, six patients), or full-dose warfarin (prothrombin time <20%, four patients). The four patients who received warfarin were started on therapy because of the clinical indications of suspected emboli to the central nervous system (patients 11 and 12), pulmonary emboli (patient 13), or large thrombus size with evidence of outflow tract obstruction (patient 14). Antithrombotic drugs were administered for 10 to 30 days before and throughout the 5 days of repeat platelet imaging. Repeat studies of patients on drugs were performed a mean of 3.8 ± 1.8 weeks (range 2 to 8) after the initial baseline tests. The baseline study preceded the drug study for all patients. Sulfinpyrazone was chosen for study since previous studies suggested a platelet inhibitor effect during short-term therapy in patients with thrombi in abdominal aortic aneurysms. None of the seven patients on sulfinpyrazone had undergone prior drug studies; one had undergone a reproducibility study (patient 1). All six patients studied on aspirin plus dipyridamole had previously undergone reproducibility studies (patients 8, 9, and 10) or had received sulfinpyrazone (patients 2, 4, and 5), which was discontinued a minimum of 2 weeks before the aspirin plus dipyridamole study; in these six patients, the initial baseline study was used for comparison with both subsequent studies. No patient in the aspirin-dipyridamole or sulfinpyrazone groups received any antithrombotic drugs during their baseline evaluation, and all patients were asked not to take aspirin or compounds containing aspirin during the study; additionally, patients who occasionally took aspirin were given a supply of acetaminophen. One patient in the warfarin group (patient 12) received aspirin (650 mg daily) and dipyridamole (100 mg daily) during the baseline study only; these drugs were stopped after a transient episode of left hemiparesis that was thought to be embolic, at which time heparin and subsequently warfarin were begun. One subject in the warfarin group received dipyridamole (25 mg three times daily) in addition to warfarin during his drug study (patient 11). Two patients in the warfarin group had serial studies performed at 2 and 5 weeks (patient 12) and at 4 and 6 weeks (patient 14) after beginning warfarin. Thus, overall, 16 patients had a total of 41 studies (16 baseline, six reproducibility, and 19 drug studies). This study was approved by the University of Washington Human Subjects Review Committee, and all subjects gave informed consent.

Indium-111 platelet imaging. Autologous platelet labeling was performed with previously described techniques. The initial and subsequent centrifugations have been modified since the original description (currently 350 g × 15 min and 1300 g × 15 min, respectively). The mean injected dose for all 41 studies was 330 ± 192 µCi (± SD) of indium-111. There were no significant differences (by paired t testing) in injected dose between the initial and subsequent platelet-imaging studies (reproducibility studies 332 ± 62 vs 376 ± 19 µCi; sulfinpyrazone studies 355 ± 66 vs 336 ± 129 µCi; aspirin plus dipyridamole studies 371 ± 66 vs 380 ± 157 µCi; warfarin studies 309 ± 96 vs 288 ± 148 µCi). The mean total dose per patient was 896 ± 244 µCi of indium-111. On the basis of radiation dosimetry estimates from four studies, each 1000 µCi of indium-111 results in a total dose to the body of approximately 0.3 to 0.9 rad, a dose to the liver of 0.6 to 2.5 rad, a dose to the spleen of 25 to 34 rad, and a dose to the male gonads of 0.1 to 0.5 rad.

Anterior, 45 degree left anterior oblique, and left lateral images were obtained for 300,000 counts/view at 48, 72, and 96 hr after labeled-platelet injection with a Sigma 410 Ohio Nuclear Gamma Scintillation Camera with a medium-energy parallel-hole collimator. Both gamma photon peaks of indium-111 (173 and 247 keV) were counted and recorded on Polaroid trilens film. Unprocessed platelet images were interpreted by two blinded observers who were unaware of the study sequence. A study result was defined as positive for thrombus when a discrete area of intracardiac activity clearly greater than the background blood pool was present in at least two of the three views. Results were defined as equivocal when the area of presumed
abnormal platelet deposition was seen in one or more views and was only slightly greater than the adjacent intracardiac blood pool. A result was considered negative when no views had a localized area of increased uptake. There was one disagreement that was resolved by consensus. Drug inhibition of platelet deposition was defined as conversion of a positive result to negative or equivocal. Intraobserver reproducibility of this method was assessed by blinded repetitive analysis of 25 studies by one observer 8 months later. On initial interpretation, 11 results were positive, three were equivocal, and 11 were negative. The repeat analysis was the same in 22 of 25 (88%) studies. In three studies, the second analysis was different (equivocal to negative in one, negative to equivocal in one, and positive to equivocal in one). Interobserver reproducibility was tested by having a second investigator analyze the results of the same 25 studies. The two observers were in agreement in all cases. Thus, the methods of visual analysis were considered reproducible.

**Two-dimensional echocardiography.** Two-dimensional echocardiograms were obtained during each platelet study with a phased-array wide-angle sector scanner (Toshiba Medical Systems; Tokyo), and the results were blindly interpreted as positive, negative, or equivocal for left ventricular thrombus by previously defined criteria. We estimated maximal left ventricular thrombus thickness in millimeters in an apical view by measuring perpendicular to the underlying myocardium from the presumed endocardial echo to the thrombus-blood interface, using a calibration scale displayed with each image. Although the thrombus and underlying myocardium often have similar echo densities, an interface was often detectable by the use of multiple gain and reject settings; when this interface could not be detected, the measurement was made to the epicardial-pericardial interface. Similar views were measured on each serial study.

To assess the intraobserver reproducibility of this measurement, 21 echocardiograms were blindly reanalyzed by one observer 4 to 14 days apart. For intraobserver reproducibility, the correlation coefficient between the first analysis (mean thickness = 2.1 ± 0.8 cm) and the second analysis (2.2 ± 0.7 cm) was \( r = .93 \) (SEE = .27, \( p = .000 \)). Interobserver reproducibility was similarly assessed by having a second observer independently and blindly analyze the same 21 echocardiograms. The correlation between findings of the second observer (2.2 ± 0.7 cm) and the initial analysis by the first observer was \( r = .95 \) (SEE = .23, \( p = .000 \)). To assess the reproducibility of this measurement of thrombus thickness over time, 20 other patients with left ventricular thrombi, no myocardial infarction within 3 months, and not undergoing heparin or warfarin therapy were restudied at a mean interval of 1.8 ± 2.7 months. The interval between studies was less than 1 month for 14 patients, 1 to 2 months for three patients, and 4.8, and 10 months for the remaining three patients. Study results were interpreted by one observer without knowledge of any clinical characteristics or the study sequence. In 18 patients, thrombus thickness neither increased nor decreased greater than 5 mm on serial study (mean thickness 1.8 ± 0.8 vs 1.7 ± 0.7 cm, \( p = NS \) by paired ttesting). In one patient, thrombus thickness increased 0.6 cm in a study performed 4 months later, and in one patient studied 2 weeks apart, the result of the second echocardiographic examination was interpreted as being only equivocal for thrombus. Thus, the measurement of thrombus thickness over time in patients without recent myocardial infarction who did not receive heparin or warfarin was reproducible over the period of study. To account for the intraobserver and temporal variation in the measurement of thrombus thickness, we defined a reduction in thrombus size as either complete thrombus resolution or a >5 mm reduction in thrombus thickness. This cut off appeared reasonable, since in the intraobserver reproducibility study, only one of the 21 studies showed more than a 5 mm difference on repeat analysis. Additionally, in the temporal reproducibility study, only one of 20 thrombi decreased in size by more than 5 mm during follow-up.

**Results**

For all six patients who underwent baseline and repeat studies on no antithrombotic drugs, results remained positive by platelet imaging and there was no change in thrombus size by echocardiography (figure 1). Of the seven patients who had serial studies before and during sulfinpyrazone therapy, none showed any change in thrombus size by echocardiography (table 1). Of these five patients, however, had inhibition of platelet deposition as shown by platelet imaging; in three, the result of the platelet scan became negative (figure 2), and in two the result of the scan became equivocal. In two patients, deposition was unchanged during sulfinpyrazone therapy.

The results of aspirin plus dipyridamole therapy are listed in table 2. Four patients had inhibition of platelet deposition while receiving aspirin plus dipyridamole; in one the result of the platelet scan became negative, and in three the result of the imaging became equiv-
TABLE 1

Results of sulfinpyrazone studies

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Platelet imaging</th>
<th>Echocardiography</th>
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<tr>
<td></td>
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</tr>
<tr>
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<td>+</td>
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</tr>
<tr>
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<td>7</td>
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+ = positive study result for thrombus; – = negative result for thrombus; EQ = equivocal result for thrombus; NC = no change in thrombus size.

Two patients had no change in platelet deposition. By echocardiography, thrombus size was unchanged in five patients but decreased modestly (6 mm) in one patient (patient 2) in whom the results of the platelet scan had become negative.

Three of four patients studied during warfarin therapy had complete inhibition of platelet deposition and in the fourth (patient 14) deposition was unchanged (table 3, figure 3). By echocardiography, thrombus was unchanged in two patients (13 and 14) and was resolved in one patient whose platelet scan became negative (patient 11). In the fourth patient (number 12) platelet imaging was negative at 2 and 5 weeks after beginning warfarin; the echocardiogram was unchanged at 2 weeks but showed a definite reduction in thrombus size at 5 weeks, suggesting that study over the longer term may be necessary to demonstrate drug effects with echocardiography. The patient with persistently positive platelet scans had no change in thrombus size by echocardiography (patient 14). During all studies in patients receiving warfarin, the prothrombin time was in the therapeutic range (<20%).

Discussion

Platelet imaging and echocardiography offer distinctly different pathophysiologic information. Platelet imaging, which detects thrombi with ongoing platelet accumulation, can define the hematologic activity of a thrombus. Echocardiography, by contrast, produces anatomic images of the heart and can assess thrombus size but not thrombus activity. As shown in this study,
TABLE 2
Results of aspirin plus dipyridamole studies

<table>
<thead>
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<th>Echocardiography</th>
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<tbody>
<tr>
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<tr>
<td>10</td>
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</tr>
</tbody>
</table>

ASA + D = aspirin (325 mg tid) plus dipyridamole (75 mg tid); + = positive study result for thrombus; − = negative result for thrombus; EQ = equivocal result for thrombus; NC = no change in thrombus size.

The incorporation of new platelets on the active surface of a thrombus could be decreased without a significant change in thrombus size as shown by echocardiography. Presumably, any changes in thrombus size were beneath the resolution of the echocardiographic measurement. Echocardiography may not then be a reliable indicator of short-term drug effect. This conclusion is also supported by the findings for patient 12, in whom platelet deposition but not thrombus size was decreased by 2 weeks of warfarin, while both deposition and size were decreased with therapy over the longer term (5 weeks). This study demonstrated that for short-term drug interventions, platelet imaging was much more likely than echocardiography to detect a drug effect.

The effects of platelet inhibitors in patients with left

FIGURE 3. At baseline, a hematologically active left ventricular thrombus was present as detected by indium-111 platelet imaging (anterior view, 48 hr image) and by two-dimensional echocardiography (apical two-chamber view). During full-dose warfarin therapy, platelet deposition ceased (48 hr image), and the thrombus resolved as determined by echocardiography (patient 11).

TABLE 3
Results of warfarin studies

<table>
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<th>Echocardiography</th>
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<tr>
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<td>14</td>
<td>+</td>
<td>+, +</td>
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+ = positive study result for thrombus; − = negative result for thrombus; NC = no change in thrombus size.

*Patient 12 had received aspirin (650 mg) plus dipyridamole (100 mg) during baseline study only and then underwent two studies at 2 weeks and 5 weeks after beginning warfarin.

*Patient 14 underwent two studies on warfarin at 4 weeks and 6 weeks. During the second, he was also receiving dipyridamole, 75 mg tid.
ventricular thrombi have not been previously tested in a prospective study. The current study demonstrates that such drugs diminish the hematologic activity of some left ventricular thrombi. Presumably, inhibition of activity as assessed by platelet imaging or complete anatomic resolution as assessed by echocardiography may be associated with a decreased embolic risk. The noninvasive diagnostic techniques of platelet imaging and echocardiography may be useful for testing this hypothesis. The current results suggest that antithrombotic therapy may be possible with less bleeding risk than is associated with traditional anticoagulants in patients with left ventricular thrombi. Both our repeat platelet imaging and echocardiographic studies in six patients on no antithrombotic drugs, as well as the additional serial echocardiographic studies of 20 other patients not receiving anticoagulants 3 or more months after myocardial infarction, document the stability of ventricular thrombi in patients who have not had a recent infarction. Thus, we believe that the observed drug effects are valid and do not represent spontaneous resolution or conversion to hematologic inactivity.

Whether our findings in patients without recent myocardial infarction would apply to patients with acute myocardial infarction and associated thrombus formation is speculative. We did not study patients with recent myocardial infarction, since left ventricular thrombi may recede spontaneously in this setting and since the embolic risk is highest then. The susceptibility to antithrombotic medications may differ between freshly formed and chronic thrombi. In this regard, a preliminary echocardiographic study of patients less than 5 weeks after infarction showed thrombus reduction or resolution in four of 10 patients treated with sulfinpyrazone (400 mg twice a day).

We chose sulfinpyrazone as the primary study drug since it appeared to inhibit platelet deposition in a similar model of arterial thrombosis in two of four patients with thrombi in abdominal aortic aneurysms. Additionally, in randomized studies, sulfinpyrazone reduced the incidence of arteriovenous shunt thrombosis and decreased the incidence of systemic embolism in patients with mitral stenosis, presumably by decreasing left atrial thrombus formation. In contrast, we have not found sulfinpyrazone effective in decreasing platelet deposition on Dacron arterial grafts in man, possibly because Dacron surfaces are a much more potent stimulus to platelet deposition. In the current study, sulfinpyrazone inhibited deposition in five of seven subjects with chronic thrombi during short-term therapy (three results became negative and two became equivocal) without affecting thrombus size.

The aspirin plus dipyridamole studies are less conclusive because of the smaller number of observations and because only one patient had complete resolution of platelet deposition on aspirin plus dipyridamole. The conversion of a platelet imaging result from positive to equivocal, such as occurred in three of the aspirin-dipyridamole studies, may not have the same implication as conversion from positive to negative. Clearly, aspirin plus dipyridamole does not prevent platelet deposition in all patients with arterial thrombi as demonstrated in this study and as suggested in an earlier study of patients with thrombi in abdominal aortic aneurysms, in which we noted no effect in seven patients. Ezekowitz et al. also reported five patients with left ventricular thrombi who had positive scans while receiving variable doses of aspirin (300 to 2400 mg/day), suggesting that aspirin alone does not completely interrupt platelet accumulation.

Although controlled studies are lacking, three small series have suggested that full-dose heparin or warfarin anticoagulation may lead to resolution of left ventricular thrombi as assessed by echocardiography in the setting of acute myocardial infarction. Additionally, in the two randomized trials of full-dose anticoagulation in patients with acute myocardial infarction for whom data at autopsy was reported, left ventricular thrombi occurred in 49% of the 53 control patients but in only 19% of the 49 treated patients, suggesting that inhibition of fibrin deposition reduced thrombus formation or led to thrombus resolution. The effects of warfarin in patients with remote infarction and presumably chronic thrombi have not previously been reported. Our findings of complete inhibition of platelet accumulation in three patients receiving warfarin suggests that anticoagulant therapy may also have an effect months to years after myocardial infarction. The one patient who did not respond to warfarin (patient 14) did not have evidence of coronary artery disease or abnormal wall motion and was thus not typical of most patients. He had a large ventricular thrombus occupying approximately 40% of his ventricular cavity as determined by echocardiography. At open heart surgery, performed because of the presumptive diagnosis of cardiac tumor, only thrombus was noted in multiple histologic sections; endocardial biopsy specimens were normal. The cause of this patient’s thrombus is obscure. Overall, our results support the concept that formation of left ventricular thrombus is partially fibrin-dependent, since warfarin acts predominantly by inhibiting fibrin polymerization. Additionally, the finding of platelet deposition in left ventricular thrombi documents a substantial platelet dependence.
as well. The relative importance of these two thrombogenetic mechanisms in the formation of left ventricular thrombi has not been determined.

**Study limitations.** This prospective study was designed to test the feasibility of detecting drug-induced changes in platelet deposition or thrombus size in patients with chronic left ventricular thrombi. It was neither randomized nor placebo-controlled. At present, both platelet imaging and two-dimensional echocardiography offer largely qualitative information. The magnitude of change in either thrombus activity or size necessary to be externally detected by these noninvasive techniques has not been defined. New developments in platelet imaging, such as single photon emission computed tomography, and in echocardiography, such as three-dimensional image reconstruction, may allow a more precise quantitative evaluation of both thrombus activity and size. Another limitation concerns the sensitivity and reproducibility of indium-111 platelet imaging for detection of left ventricular thrombus. In the only study assessing sensitivity, platelet imaging detected 10 of 14 (71%) patients with proven thrombi. Since the sensitivity of platelet imaging is not 100%, a high degree of reproducibility is essential if platelet imaging is to be used to evaluate the effects of therapy. Although our results of platelet imaging were reproducible in six patients restudied on no antithrombotic medications, additional reproducibility studies are desirable. We have previously documented that findings from platelet imaging in patients with Dacron arterial grafts are reproducible. This study was not designed to assess a clinical end point, such as reduction in embolic events. Hence, no conclusions can be reached as to the potential clinical significance of our findings in an individual patient, since it remains to be proven whether platelet deposition or its inhibition predicts subsequent embolization. Additionally, longer-term studies that use platelet inhibitors in a larger number of patients are required to determine if such drugs alone can lead to thrombus resolution as assessed by echocardiography.

**Implications.** Both platelet imaging and echocardiography can measure antithrombotic drug effects in patients with left ventricular thrombi. During short-term therapy with platelet inhibitors, platelet deposition can be inhibited without an identifiable change in thrombus size. Determination of the clinical and long-term importance of these discordant observations awaits further study. However, these findings suggest that the earliest evidence of drug effect may be demonstrated by platelet imaging and that echocardiography may not be a reliable indicator of short-term drug activity. These findings were obtained in patients with remote myocardial infarction whose thrombi were chronic. More rapid changes in thrombus mass might be found in more recently formed thrombi, such as those associated with acute myocardial infarction.

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