Increased embolic risk in patients with left ventricular thrombi

JOHN R. STRATTON, M.D., AND ARTHUR D. RESNICK, M.D.

ABSTRACT Although left ventricular thrombi are associated with an increased embolic risk in the first few weeks after acute myocardial infarction, the long-term risk remains undefined. To ascertain the incidence of strictly defined systemic emboli, we followed 85 patients with echocardiographically documented left ventricular thrombi. At the time of the entry echocardiogram, most patients (n = 57) had remote myocardial infarction, while 19 had recent (<1 month) infarction, and nine had idiopathic cardiomyopathy. Because of the difficulty in classifying events as embolic in patients with advanced atherosclerosis, a matched control group of 91 patients without thrombi was also studied. The thrombus and control groups were similar with regard to recent myocardial infarction, remote infarction, anterior infarction, ejection fraction, atrial fibrillation, echocardiographic referral for source of emboli, and warfarin therapy. During a mean follow-up of 22 months after echocardiography, embolic events occurred in 13% (11 of 85) of patients with thrombi compared with 2% (two of 91) of control patients (p < .01). The actuarial probability of being embolus free at 2 years after echocardiography was 86% in patients with thrombi compared with 97% in control patients (p < .01). All embolic events occurred greater than 1 month after myocardial infarction (range 1 to 96 months). The only clinical or echocardiographic features predictive of embolization were protrusion and mobility of the thrombus (both p < .02). We conclude that the incidence of embolic events is definitely increased in patients with left ventricular thrombi compared with control subjects during long-term follow-up. The increased embolic risk is not restricted to the immediate postinfarction period, documenting that chronic thrombi continue to embolize. Whether long-term anticoagulation can reduce the embolic rate with an acceptable risk of bleeding remains to be determined.


LEFT VENTRICULAR THROMBI occurring in the setting of acute myocardial infarction have been associated with an increased embolic risk; in patients with acute infarction the risk of embolization probably decreases over time, with both older1–7 and more recent studies8–12 noting that approximately two-thirds of systemic emboli have occurred within the first month after an acute myocardial infarction. However, many left ventricular thrombi remain present for indefinite periods after acute infarction, with or without acute anticoagulant therapy, and become chronic thrombi. Very little is known regarding the long-term embolic risk of left ventricular thrombi. Three recent largely retrospective studies have yielded conflicting results.12–14

There have been no large prospective studies to date of patients with left ventricular thrombi.

The purpose of this study was to define prospectively the incidence of systemic embolization in a patient population with predominantly chronic left ventricular thrombi, and to determine whether clinical or echocardiographic features are strong predictors of embolization. Due to the difficulty in classifying events as embolic in patients who frequently have coexisting cerebral and peripheral vascular disease, we took two precautions. First, we used rigidly defined criteria for the diagnosis of emboli, requiring surgical or autopsy proof for peripheral emboli and for cerebral events and requiring that the criteria of Hart et al.15 be met. Second, a control group of patients without thrombi was studied to determine the rate of embolic-type events in patients similar to the study group except for the absence of left ventricular thrombus.

Methods

Patient selection. All patients with left ventricular thrombus diagnosed by two-dimensional echocardiography between Oc-
PATHOPHYSIOLOGY AND NATURAL HISTORY—MYOCARDIAL INFARCTION

tober 1979 and June 1984 were entered. During this period, 2996 two-dimensional studies were performed. Control subjects were selected if they had one or more of the following clinical or echocardiographic features: echocardiographic referral to rule out left ventricular thrombus, history or echocardiographic evidence of ventricular aneurysm, or anterior myocardial infarction and moderately or severely reduced left ventricular function. Patients were excluded if they had clinically identified mitral valve disease or a prosthetic valve because of the known increase in embolic rate in these groups. One hundred eighty-eight patients with thrombus and control patients were identified.

Two-dimensional echocardiography. Two-dimensional echocardiography was performed with a wide-angle, phased-array scanner, as previously described. During the study period, echocardiographic diagnostic criteria for left ventricular thrombi evolved as a result of studies in our laboratory and others. To ensure that the same criteria were applied to all subjects, at the conclusion of the study all echocardiograms from the 188 patients were reinterpreted by two observers who were blinded to all clinical data. Left ventricular thrombus was defined as a discrete mass of echoes in the left ventricle that was distinct from the endocardium and seen in both diastole and systole, located in an area of asynergy, and identified in at least two views. Care was taken to exclude false tendons and trabeculae. Both the sensitivity (95%) and specificity (86%) of the echocardiographic diagnosis of left ventricular thrombus in our laboratory are relatively high, as previously described. At the time of the blinded review several other echocardiographic features were also evaluated, including the presence or absence of a left ventricular aneurysm by the criteria of Baur et al., by which an aneurysm is defined as a localized area of akinesis or dyskinesis that deforms the ventricular chamber during both diastole and systole. The presence or absence of mitral annular calcification, which has been reported to be associated with strokes, was noted. In addition, in studies in which a definite left ventricular thrombus was present, we evaluated the characteristics of the thrombus, including intracavitary motion, shape (protruding vs flat), echo density, heterogeneity, and central lucency as defined by Haugland et al. Intracavitary motion was considered present if a portion of the thrombus that protruded moved independently of the underlying myocardium. Thrombi that projected into the ventricular cavity were classified as protruding and thrombi that did not were classified as flat. An estimate of thrombus size, a one-dimensional measurement of maximal thrombus thickness was made perpendicular to the myocardium from the epicardial-pericardial interface to the innermost border of the thrombus-blood interface, as previously described.

On the basis of the blinded review, 12 studies were classified as technically inadequate; the remaining 176 studies were graded as positive (n = 75), equivocally positive (n = 10), or negative (n = 91) for left ventricular thrombus. The 12 patients with technically inadequate studies were excluded from further analysis, leaving 176 patients in the study. The 12 patients with equivocally positive studies were grouped with the 75 patients with positive studies for all further analyses. The results of the study were not different if the patients with the equivocal studies were instead classified with the negative group.

Patient follow-up, data collection, and embolic criteria. Follow-up was by chart review and/or phone call at 6 to 9 month intervals. At the conclusion of the study, an attempt was made to contact all patients by phone and all charts were reviewed. The criteria for diagnosis of a peripheral embolus was either surgical removal or documentation at autopsy. The classification of cerebral ischemic events as embolic or nonembolic is imperfect. Hart et al. have recently developed an algorithm that was used in this study. Events were classified as embolic if the following criteria were met: (1) absence of ipsilateral carotid bruit or transient ischemic attack, (2) if chronic hypertension was present, nonlacunar infarction by x-ray computed tomography, (3) presence of one or more of the following: (a) abrupt onset, (b) absence of stenoses by angiography or noninvasive studies, (c) history of other prior embolic events, (d) age less than 50 without diabetes or hypertension, (e) autopsy evidence of embolic stroke, or (f) bilateral, nonlacunar infarctions by x-ray computed tomography.

An event was classified as nonembolic if one or more of the following were present: (1) ipsilateral bruit, stenosis, transient ischemic attack, or prior carotid endarterectomy, (2) hypertension with clinical and x-ray computed tomographic evidence of lacunar infarction, or (3) nonsudden onset while awake. An event was classified as indeterminate if the onset occurred while the patient was asleep and there was no progression after waking, there was no information available about the onset, or there was absence of sufficient information to allow classification as an embolic or nonembolic event.

In addition to data regarding systemic embolization, data were collected regarding warfarin therapy and bleeding complications, survival status, the ejection fraction by radionuclide angiography (available in 125 patients), the type (anterior, inferior, nontransmural) and time of all prior myocardial infarctions, and occurrence of myocardial infarction during follow-up. Decisions regarding anticoagulation therapy were made by the patients’ primary physicians. Left atrial dimension (available in 119 patients), left ventricular end-diastolic (n = 109) and end-systolic dimensions (n = 106), and shortening fraction (n = 106) were obtained from M mode echocardiographic reports. All available electrocardiograms (n = 168 patients) were blindly interpreted by one of the investigators for evidence of transmural myocardial infarction (anterior or inferior) and evidence of chronic or paroxysmal atrial fibrillation. The interval from the last myocardial infarction of any type (transmural or nontransmural) to echocardiography and to an embolic event was noted. A patient was defined as having had a myocardial infarction if there was a definite Q wave infarction present on the electrocardiogram, if there was documentation of typical electrocardiographic and enzyme changes, or in the case of the 14 patients with left bundle branch block, if there was a definite clinical history and enzyme changes documented on the chart. Patients were divided into three groups depending on whether they had a recent myocardial infarction (<1 month before echocardiography), a remote infarct (≥1 month before echocardiography), or idiopathic cardiomyopathy.

Statistical analysis. Continuous variables were compared by an unpaired t test and discrete variables were compared by the chi-square test or Fisher's exact test. Life table analysis was used to construct curves for the proportion of patients who were free of emboli, and the generalized Wilcoxon statistic was used to compare the curves of the thrombus and control groups. Data are expressed as the mean ± SD.

Results

Patient characteristics. The clinical features of the thrombus and control groups at study entry are listed in table 1. The groups were well matched. Most patients in both the thrombus (67%) and control (70%) groups had remote myocardial infarctions at study entry (i.e., the time of echocardiography). Only small numbers in both groups had a recent myocardial infarction (22% of patients with thrombus and 21% of controls)
at the time of echocardiography. In approximately two-thirds of patients in both groups the location of the infarction was anterior. Among those with prior anterior myocardial infarction, the interval from the anterior myocardial infarction to study entry averaged 33 ± 46 months (range 0 to 192) in the patients with thrombus and 40 ± 55 months (range 0 to 243) in the control patients (p = NS). Similar proportions of the thrombus and control groups had been referred to the echocardiography laboratory to rule out a source of embolus and similar proportions had chronic or paroxysmal atrial fibrillation. Global ventricular function, as assessed by the radionuclide ventriculographic ejection fraction in 125 patients, was reduced to a similar extent in both groups. Echocardiographic features are summarized in table 2. Patients with thrombus had a higher prevalence of left ventricular aneurysm than did the control subjects, but other features, including atrial and ventricular dimensions and the prevalence of mitral annular calcification, were similar.

Duration of follow-up after echocardiography was similar in the two groups (table 3). Death during follow-up was frequent in both the thrombus (28%) and control (24%) groups (p = NS). Myocardial infarction (transmural or nontransmural) during follow-up was uncommon in both groups. The proportion of patients receiving warfarin anticoagulation during follow-up but before an embolic event was not significantly different in the thrombus group (27%) and the control group (19%). The mean duration of anticoagulation was 12.9 months in the thrombus group and 10.1 months in the control group (p = NS). Similar proportions of both groups received platelet-inhibitory therapy at some point during follow-up (aspirin, dipryidamole, sulfinpyrazone, indomethacin, or other nonsteroidal anti-inflammatory drugs).

**Embolic events.** Before echocardiography, 19% (16 of 85) of patients with thrombi had embolic events compared with 5% (five of 91) of control patients (p < .01) (table 4). Of the patients with embolic events before echocardiography, only one patient with a thrombus and only one control patient had additional embolic events during follow-up.

During a mean follow-up of 22 months after echocardiography, 13% (11 of 85) of patients with thrombus had embolic events compared with 2% (two of 91) of control patients (p < .01). By life table methods, at

---

**Table 1**

<table>
<thead>
<tr>
<th>Patient characteristics at entry</th>
<th>Patients with thrombus</th>
<th>Control patients</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>60 ± 10</td>
<td>63 ± 12</td>
<td>NS</td>
</tr>
<tr>
<td>Idiopathic cardiomyop. (%)</td>
<td>11</td>
<td>9</td>
<td>NS</td>
</tr>
<tr>
<td>Recent (&lt;1 month) MI (%)</td>
<td>22</td>
<td>21</td>
<td>NS</td>
</tr>
<tr>
<td>Remote MI (%)</td>
<td>67</td>
<td>70</td>
<td>NS</td>
</tr>
<tr>
<td>Anterior MI (%)</td>
<td>74</td>
<td>66</td>
<td>NS</td>
</tr>
<tr>
<td>Interval from last MI to study entry (months)</td>
<td>32 ± 51</td>
<td>48 ± 71</td>
<td>NS</td>
</tr>
<tr>
<td>Echocardiographic referral for source of embolus (%)</td>
<td>20</td>
<td>24</td>
<td>NS</td>
</tr>
<tr>
<td>Atrial fibrillation (chronic or paroxysmal) (%)</td>
<td>13</td>
<td>15</td>
<td>NS</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>0.30 ± 0.14</td>
<td>0.32 ± 0.13</td>
<td>NS</td>
</tr>
</tbody>
</table>

**Table 2**

<table>
<thead>
<tr>
<th>Echocardiographic features in thrombus and control groups</th>
<th>Patients with thrombus</th>
<th>Control patients</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricular aneurysm (%)</td>
<td>75</td>
<td>46</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Apical akinesia or dyskinesia (%)</td>
<td>93</td>
<td>84</td>
<td>NS</td>
</tr>
<tr>
<td>Mitral annular calcification (%)</td>
<td>17</td>
<td>26</td>
<td>NS</td>
</tr>
<tr>
<td>Left atrial size (cm) (n = 119)</td>
<td>4.4 ± 0.9</td>
<td>4.6 ± 0.9</td>
<td>NS</td>
</tr>
<tr>
<td>Left ventricular end-diastolic size (cm) (n = 109)</td>
<td>6.3 ± 0.8</td>
<td>6.3 ± 0.9</td>
<td>NS</td>
</tr>
<tr>
<td>Shortening fraction (%) (n = 105)</td>
<td>21 ± 9</td>
<td>21 ± 10</td>
<td>NS</td>
</tr>
</tbody>
</table>

**Table 3**

<table>
<thead>
<tr>
<th>Follow-up data: duration, death, myocardial infarction, and antithrombotic therapy</th>
<th>Patients with thrombus</th>
<th>Control patients</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Follow-up duration (months)</td>
<td>22 ± 17</td>
<td>22 ± 15</td>
<td>NS</td>
</tr>
<tr>
<td>Death during follow-up (%)</td>
<td>10</td>
<td>9</td>
<td>NS</td>
</tr>
<tr>
<td>MI during follow-up (%)</td>
<td>5</td>
<td>7</td>
<td>.05</td>
</tr>
<tr>
<td>Warfarin during follow-up before embolism (%)</td>
<td>4.0</td>
<td>3.7</td>
<td>NS</td>
</tr>
<tr>
<td>Platelet-inhibitor drugs during follow-up (%)</td>
<td>4.0</td>
<td>3.7</td>
<td>NS</td>
</tr>
</tbody>
</table>

**Table 4**

<table>
<thead>
<tr>
<th>Embolic events before diagnosis of thrombus and during follow-up</th>
<th>Patients with thrombus</th>
<th>Control patients</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with emboli before echo exam (%)</td>
<td>19 (16/85)</td>
<td>5 (5/91)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Patients with emboli during follow-up (%)</td>
<td>13 (11/85)</td>
<td>2 (2/91)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Patients with emboli either before echo exam or during follow-up (%)</td>
<td>31 (26/85)</td>
<td>7 (6/91)</td>
<td>&lt;.0001</td>
</tr>
</tbody>
</table>
2 years of follow-up 97% of control subjects were embolus free compared with 86% of patients with thrombi (p < .01) (figure 1). At 64 months, 97% of control subjects were free of emboli compared with 76% of patients with thrombi.

Of the embolic events occurring in patients with left ventricular thrombi during follow-up, two were peripheral emboli and nine were central nervous system emboli (one to the eye, one transient ischemic attack, and seven strokes). Both emboli in control patients were strokes. Of the 11 patients with thrombi who had embolic events during follow-up, repeat echocardiography at the time of the event was performed in seven and continued to demonstrate thrombus, and an additional patient had thrombus proven at autopsy. In the two control subjects with emboli during follow-up, repeat echocardiography continued to show the absence of left ventricular thrombus.

All emboli in the thrombus group occurred in patients with documented prior myocardial infarction; none occurred in the small group of patients (n = 9) with idiopathic cardiomyopathy. After the entry echocardiogram that documented thrombus, 26% (five of 19) of the patients with recent (<1 month) myocardial infarction subsequently had emboli compared with 11% (six of 57) of the patients with a remote myocardial infarction. Although this grouping of patients suggests that emboli were more common in patients with recent infarction, all 11 embolic events occurred 1 month or more after myocardial infarction (mean 31 ± 32 months, range 1 to 96 months). In fact, only two of the 11 embolic events occurred within the first 6 months after a myocardial infarction (at 1 and 4 months), and the remaining embolic events occurred at 7, 9, 12, 18, 34, 51, 72, and 96 months after infarction. (One patient had a remote infarction at an uncertain date that was at least 6 months before his embolic event.) Thus, in our study population most embolic events occurred at times remote from the last myocardial infarction.

Warfarin anticoagulation. Among the 85 patients with left ventricular thrombi, 23 were treated with warfarin before the time of an embolic event or to the end of follow-up and 62 were not. Among the warfarin-treated patients, 9% (two of 23) had emboli during follow-up compared with 14% (nine of 62) of nontreated patients (p = NS). However, both patients who had embolic events while being treated with warfarin had received anticoagulant therapy for less than 5 days and did not have therapeutic prothrombin times. If these
two patients were grouped with those not receiving effective anticoagulation, then 0% (0 of 23) of effectively anticoagulated patients had emboli compared with 18% (11 of 62) of patients with intracardiac thrombi not receiving therapeutic anticoagulation (p = .04).

Among both the thrombus and control groups, a total of 45 patients received anticoagulants during follow-up either before or after an embolic event; 20% (nine of 45) had significant complications associated with warfarin therapy (central nervous system bleed in one, gastrointestinal bleed in three, hematuria in one, pulmonary hemorrhage in two, severe nosebleeds in one, and purple toe syndrome in one).

**Comparison of patients with thrombus and without emboli during follow-up.** To determine whether clinical or echocardiographic features were predictive of embolization, we compared the 11 patients with thrombus and systemic emboli during follow-up with the 74 patients without emboli (table 5). None of the clinical features examined were more common in patients with thrombus and embolization than in those without it. Among echocardiographic features, left ventricular aneurysm was equally prevalent. The measurement of thrombus thickness, which included the underlying myocardium, was not significantly different between groups. Thrombus protrusion occurred in 90% of patients with emboli and 49% of patients without emboli (p < .02). Thrombus mobility was significantly more common in patients who had emboli (70%) than in those who did not (20%, p < .01).

**Discussion**

The current study documented that left ventricular thrombi were associated with an increased risk of systemic embolization during a mean follow-up of 22 months. The risk of embolization within 2 years after the echocardiographic detection of thrombus was 14% by life table methods. This study differs from prior studies of the embolic potential of left ventricular thrombi in several respects. First, we used recently developed strict criteria for the diagnosis of cerebral embolism. Second, patients were studied prospectively from the time of echocardiography, while most prior studies reported a mixture of embolic events that occurred both before and after the entry echocardiogram or at an unspecified time. Third, the current study used a control group of patients who were similar to the study group with the exception of the absence of left ventricular thrombus and a lower prevalence of left ventricular aneurysm. The control group was important because of the frequent difficulty in diagnosing a central nervous system event as embolic in a patient population that commonly has associated atherosclerotic disease in the cerebral vasculature. This difficulty is underscored by the findings in the control group, in which five subjects were diagnosed as having emboli before the entry echocardiogram and two had embolic events during follow-up. Despite the occurrence of events that were classified as embolic in the control group, patients with left ventricular thrombi had a clearly increased embolic risk compared with the control subjects without left ventricular thrombi. Lastly, most patients in our study had remote myocardial infarction, with the last infarct being a mean of 32 months previously, whereas most prior studies have examined patients with predominantly acute myocardial infarction and left ventricular thrombi.

**Left ventricular thrombi and emboli in acute infarction.** In patients with acute myocardial infarction, although early anticoagulation with heparin or warfarin has been observed to reduce development of left ventricular thrombus or lead to resolution of thrombus,1, 23-26 early anticoagulation does not totally prevent the formation of thrombus and does not uniformly lead to its resolution.27-30 Thus, left ventricular thrombi can develop and persist in some patients in spite of anticoagulation. The effects of anticoagulants on the risk of embolization continues to be debated. Although recent small studies have suggested a low embolic risk in patients without anticoagulant therapy27, 29, 31 another study documented that 27% of untreated patients with left
ventricular thrombi due to acute anterior myocardial infarction suffered emboli in the hospital.30 Recent nonrandomized studies have suggested a reduction in embolic events by early anticoagulation,48-50 and four older trials of anticoagulation in acute infarction including a total of 4362 patients documented that short-term anticoagulation reduced all systemic emboli from a range of 3% to 6%, to 1% to 3%, and stroke from a range of 3% to 4%, to 1% to 2%.1,23,32,33 On the basis of the available data, a recent NHLBI National Conference on Antithrombotic Therapy “strongly” recommended that all patients with acute anterior myocardial infarction be treated with heparin followed by warfarin for 1 to 3 months to reduce systemic emboli.2

Chronic left ventricular thrombi. Much less is known regarding the long-term fate or optimal therapy of left ventricular thrombi that are present months to years after myocardial infarction. Although approximately 20% of thrombi resolve spontaneously after acute infarction without therapy,31 and others are prevented or resolve with heparin or warfarin therapy,26,28 a significant population of patients exists in which chronic thrombi develop. The risk of embolization after acute infarction appears to decrease over time, with approximately two-thirds of emboli occurring within the first month after infarction.1-12 Although the risk of embolism appears highest early after an infarction, two recent large but predominantly retrospective studies noted that between 13% and 20% of patients with chronic thrombi had emboli either before or after echocardiographic documentation of a thrombus, suggesting an ongoing embolic risk due to chronic thrombi.12-14 However, in the one study in which a control group was examined, the embolic risk was not significantly greater in patients with left ventricular thrombi than in control subjects.13 Due to the small numbers of patients who were entered into our study early after myocardial infarction, the current report does not add to the available data from patients with acute infarction and recently formed thrombi. However, our prospective study does confirm that chronic left ventricular thrombi are associated with an ongoing embolic risk, albeit at a lesser rate than that associated with acute infarction. In fact, in our patient population only two of the 11 embolic events occurred within the first 6 months after a myocardial infarction. The increased embolic risk is concordant with indium-111 platelet imaging studies in which most thrombi, regardless of age, have been observed to have externally detectable ongoing platelet accumulation, indicating continued surface activity.21,34-36

The proper therapy, if any, for patients with chronic left ventricular thrombi is unknown. The National Conference on Antithrombotic Therapy offered no recommendation for the management of such patients.2 A recent review suggested that anticoagulant therapy may not be needed due to the low risk of emboli.37 In the current study, there was no suggestion that platelet-inhibitory therapy reduced emboli. Although there was a trend toward fewer embolic events in warfarin-treated patients, the results are inconclusive due to the lack of randomization. The risks of long-term full-dose warfarin therapy were substantial, with 20% of 45 patients on warfarin suffering a significant complication during a mean follow-up of 15 months in the current study. This is similar to the risk noted in other studies of patients with ischemic heart disease in which 17% of 1890 patients had bleeding complications in seven randomized studies.28 Two potentially lower risk alternatives to full-dose warfarin therapy exist: less intense warfarin therapy39 and platelet-inhibitory agents.21,28 However, neither of these approaches has proven clinical efficacy in reducing the embolic rate. Due to the relatively low rate of clinically apparent embolization from chronic thrombi, a large study would be needed to address these questions.

Echocardiographic and clinical predictors of embolization. Thrombus protrusion was more frequent in patients who had emboli during follow-up than in those who did not; among patients with protruding thrombi, 22% suffered an embolus in follow-up compared with 3% in other patients with thrombi. This finding has been previously reported,11-13 but the incidence of embolization in patients with this characteristic has been higher, at 56%,41-43 and 100%,11 in studies in which most of the embolic events occurred before echocardiography. Similarly, thrombus mobility was more common among patients with an embolus than in those without; the incidence of embolization during follow-up was 35% in patients with a mobile thrombus, compared with 5% in those without. In prior studies, mobility was not associated with embolization in one study,14 but was highly associated in three others, with 83%,13,60%,12 and 100%,11 of patients with mobile thrombi reported as having embolic events. The lower incidence of embolization in our patients with either protrusion or mobility of thrombus may be due to the fact that we considered only embolic events that occurred after the echocardiogram was obtained and that these features may be less predictive in patients with remote infarction and presumably older thrombi than in patients with acute infarction. Nevertheless, our study confirms that mobility of thrombus and to a lesser extent thrombus protrusion are risk factors.
Prior studies of patients with left ventricular aneurysms documented by contrast angiography have suggested a low embolic risk (1% to 5%), despite the fact that about one-half of such patients have associated left ventricular thrombi. However, all three studies were retrospective. Our results do not suggest that the formation of aneurysm is associated with a low embolic risk in patients with documented thrombi, since embolism occurred in 13% of patients with thrombi and aneurysm, compared with 14% of patients with thrombi and no aneurysm.

Spirito et al. recently suggested that the formation of left ventricular thrombus after acute myocardial infarction was predictive of a high mortality (12 of 24 patients), especially if formation occurred within 2 days of infarction (10 of 11 patients). Our results confirm that patients with left ventricular thrombi have a high mortality (28% during a 22 month follow-up). However, mortality in patients with thrombi was not increased compared with that in the control group (24%). The high mortality rate probably relates to the moderately severe reduction in left ventricular function present in both groups.

Study limitations. The ability to diagnose a cerebral ischemic event as embolic, even with strict criteria, has clear limitations. In the current study, patients with indeterminate events by the criteria of Hart et al. were not considered as having emboli. It is possible that some of these indeterminate events, which occurred during follow-up in four of the patients with thrombus and in none of the control patients, were in fact embolic. However, the inclusion of these events as embolic would only have strengthened our main conclusion regarding an increased embolic risk in patients with left ventricular thrombi. Many patients were lost to follow-up within the first year, many due to death. Our study population was nonhomogeneous and contained only small numbers of patients with idiopathic cardiomyopathy or with a recent infarction at study entry and thus this report does not add to the available data in such patients. Anticoagulation was not controlled as a part of this study. Therefore, although the data convincingly demonstrate an increased risk of embolization in our population, we cannot determine whether anticoagulation reduced the risk. Additionally, we cannot determine whether spontaneous or anticoagulant-induced resolution of thrombus leads to a reduction in embolic risk.

Clinical implications. The current study documents that patients with left ventricular thrombi, largely due to remote myocardial infarction, have an increased embolic risk. The increased embolic risk is not restrict-
Increased embolic risk in patients with left ventricular thrombi.
J R Stratton and A D Resnick

_Circulation._ 1987;75:1004-1011
doi: 10.1161/01.CIR.75.5.1004

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1987 American Heart Association, Inc. All rights reserved.
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:
http://circ.ahajournals.org/content/75/5/1004