SPECIAL COMMENTARY

Left Ventricular Aneurysm, Intraaneurysmal Thrombus and Systemic Embolus in Coronary Heart Disease*

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Although left ventricular aneurysm has been reported in association with noncoronary types of cardiac disease, such as hypertrophic cardiomyopathy,1 congenital deficiencies of myocardium,2 sarcoidosis,3 and postoperatively after mitral valve replacement,4 most occur as a consequence of severe coronary narrowing with myocardial infarction. Most reports on surgical and/or necropsy patients with coronary-induced left ventricular myocardial infarcts and aneurysms have demonstrated a high frequency of thrombi within the aneurysms. Intraaneurysmal thrombi were present in 155 (49 percent) of 314 necropsy patients (from five different studies)5-8 and in 411 (47 percent) of 867 surgical patients who underwent left ventricular aneurysmectomy (from ten different studies).9-19 In contrast to the relative uniform reporting of a high frequency of thrombi within left ventricular aneurysms, the reported frequency of systemic emboli in patients with left ventricular aneurysms has varied considerably. Systemic emboli were described in 100 (32 percent) of the 314 patients with left ventricular aneurysms documented at necropsy,4,6 but in only 59 (5 percent) of 1,180 reported patients in whom aneurysms were diagnosed by angiography and/or operation.11-14,18-29 Simpson and associates,30 examined the frequency of intraaneurysmal thrombi and systemic emboli in patients who underwent left ventricular aneurysmectomy. Mural thrombi were found at operation in 38 (66 percent) of their 58 patients and only two (3 percent) of the 58 had clinical events compatible with systemic emboli. These authors concluded that despite the frequent occurrence of intraaneurysmal thrombi, clinically apparent systemic emboli were rare.

Before accepting the conclusion that systemic emboli are rare in patients with left ventricular aneurysms, it might be useful to examine the definitions used for "left ventricular aneurysm" and "systemic embolus" in several reported studies to determine the uniformity of criteria for diagnosis. Obviously, if definitions of the two items to be analyzed vary from study to study, the results or conclusions may do likewise. Left ventricular aneurysm was defined by Simpson and associates30 as "... an abnormally thinned, scarred, or bulging segment of left ventricular free wall noted at the time of surgery;" by Cheng30 as "... a local area of total lack of motion (akinesia) or of paradoxical expansile motion (dyskinesis) during systole of the ventricular wall which may vary in thickness from a paper-thin scar to full-thickness muscle;" by Hines and associates30 as "... a localized area of paradoxically contracting or akinetic ventricle..." by Letac and colleagues30 as "... a sac protruding from the remaining left ventricular contour during both systole and diastole [at angiography]..." by Grondin and associates27 as "... an obvious diastolic bulge with a systolic paradoxical motion [at fluoroscopy or angiography]..." by Favaloro and colleagues19 as "... a full-thickness scar tissue replacement of a large segment of the left ventricular wall, usually containing a thrombus and attached to the pericardial sac by adhesions...[with] a clear-cut demarcation from the rest of the left ventricle... The absence of a frank bulging mass does not exclude a surgical diagnosis of ventricular aneurysm;" by Loop and associates15 as "... thinned-out transmural scars that have completely lost their trabecular pattern. Although the aneurysm did not always bulge outward, the scar was localized and clearly delineated from surrounding ventricular muscle;" by Phares and colleagues3 as "... defects in the ventricular wall which demonstrate a definite bulge in the external contour of the heart, together with a thinning of the affected region;" by Schlichter and associates6 as "... a

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506 CABIN, ROBERTS

CHEST, 77: 5, MAY, 1980
localized outpouching of the cavity of a cardiac chamber, with or without outward bulging of the external surface.” It is apparent that there is no uniformly accepted definition of left ventricular aneurysm. Consequently, although all the above-cited authors reported findings on patients with “left ventricular aneurysm,” their criteria for including and excluding patients varied considerably and, therefore, their findings could also vary considerably.

In clinical studies reporting patients with left ventricular aneurysms, criteria for diagnosis of systemic (called by some “peripheral”) emboli rarely have been defined. Whether the diagnosis of embolism was based entirely on appropriate clinical symptoms and signs or whether confirmation was obtained by angiogram or operation or both has rarely been stated. Reports of necropsy patients with left ventricular aneurysms and systemic emboli usually do not state whether diagnosis was based on the above clinical criteria or on the finding of infarcts in one or more organs at necropsy. Systemic emboli, obviously, might be detected at necropsy, but not during the patient’s life and vice versa. Thus, the variability in reported frequency of systemic emboli in patients with left ventricular aneurysms might in part be the result of differing criteria for diagnosis.

Because findings depend to a large extent on the definition of “aneurysm” employed, we have attempted to define left ventricular aneurysm in a way applicable to angiographic, surgical or necropsy studies. We have found it useful to define left ventricular aneurysm as a localized cavity (other than when filled with thrombus) protrusion of left ventricular free wall. Use of the words “anatomic” and “functional” (Fig 1) also is helpful. An anatomic aneurysm is a localized protrusion of left ventricular free wall in both ventricular systole and diastole,

LEFT VENTRICULAR ANEURYSM IN CORONARY HEART DISEASE

I ANATOMIC

A. True

Mouth of aneurysm

Thrombus

B. False

Parietal pericardium

Figure 1. Diagrams of hearts in systole and diastole with true and false anatomic, and functional left ventricular aneurysms and healed myocardial infarction. A diagram of a normal heart in systole and diastole is shown for comparison. The true anatomic left ventricular aneurysm protrudes during both systole and diastole, has a mouth that is as wide or wider than the maximal diameter of the aneurysm, has a wall that was formerly the wall of the left ventricle, and is composed of fibrous tissue with or without residual myocardial fibers. A true aneurysm may or may not contain thrombus and almost never ruptures once the wall is healed. The false anatomic left ventricular aneurysm protrudes during both systole and diastole, has a mouth that is considerably smaller than the maximal diameter of the aneurysm and represents a myocardial rupture site, has a wall that is made up of parietal pericardium, virtually always contains thrombus, and often ruptures. The functional left ventricular aneurysm protrudes during ventricular systole, but not during diastole and consists of fibrous tissue with or without myocardial fibers.

CHEST, 77: 5, MAY, 1980  ANEURYSM, THROMBUS AND EMBOLUS IN CORONARY HEART DISEASE  507
whereas a functional aneurysm is a protrusion only during ventricular systole. At angiography or at operation, the anatomic aneurysm appears as a localized protrusion of left ventricular free wall during diastole, and during systole the aneurysmal wall may not move at all (akinetie) or may protrude even more than in diastole (dyskinetic or paradoxical movement). A functional aneurysm at angiography or at operation protrudes only during ventricular systole; its wall, therefore, is dyskinetic since it moves outward when the remaining wall moves inward. At necropsy, only an anatomic aneurysm appears aneurysmal. The distinction between anatomic and functional aneurysm is useful because the wall of a functional aneurysm may consist of ischemic (potentially reversible) or necrotic myocardium or mainly fibrous tissue, whereas the wall of an anatomic aneurysm consists of either necrotic or fibrotic tissue or both and neither is reversible. An anatomic left ventricular aneurysm may be either “true” or “false” (Fig 1). A true anatomic aneurysm has a mouth that is as wide or wider than the maximal diameter of the aneurysm; its wall was formerly the wall of the left ventricle and consists of necrotic myocardium (acute myocardial infarction) or fibrous tissue (healed myocardial infarction) or both. A false anatomic aneurysm, has a mouth that is considerably smaller than the maximal diameter of the aneurysm and it was the rupture site at the time of the acute myocardial infarction; its wall is composed of parietal pericardium and never contains residual myocardial fibers.

We recently examined the hearts of 28 necropsy patients with true, anatomic left ventricular aneurysms occurring at sites of healed myocardial infarcts. In agreement with most previous studies, we found that intraaneurysmal thrombi were frequent (11 [39 percent] of 28 patients), and histories of clinical signs and symptoms compatible with systemic emboli, infrequent (one [4 percent] patient).

Why are clinical events compatible with systemic emboli infrequent in patients with healed left ventricular infarcts, aneurysms and intraaneurysmal thrombi? For a left ventricular mural thrombus to embolize, a large portion of its surface must be unattached to the underlying wall and exposed to the blood flowing in the ventricular cavity. In patients with idiopathic dilated cardiomyopathy, portions of left ventricular mural thrombi frequently embolize because they overly contracting myocardium, have a relatively small area of attachment to left ventricular wall, and protrude into the left ventricular cavity (Fig 2, 3).1 In patients with left ventricular aneurysms, in contrast, intraaneurysmal thrombi infrequently embolize apparently because they are located in a portion of left ventricle (the

![Diagram of hearts with idiopathic dilated cardiomyopathy, healed left ventricular myocardial infarct without aneurysm, and healed left ventricular myocardial infarct with aneurysm. The left ventricle in each contains thrombus. A diagram of a normal heart is shown for comparison. The thrombi in the left ventricular cavities of the hearts with idiopathic dilated cardiomyopathy and healed myocardial infarct without aneurysm are more likely to embolize than is an intraaneurysmal thrombus.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21139/ on 04/06/2017)
aneurysm, which does not propel its contents in the direction of the outflow tract, the aneurysmal wall underlying the thrombus is virtually devoid of contractile fibers, more of the surface of the thrombus is attached than unattached, and the intraaneurysmal thrombus does not protrude into the left ventricular cavity (Fig 2 and 3).

**Should patients with left ventricular aneurysms receive anticoagulants?** Simpson and associates found anticoagulants to have no effect on the frequency of intraaneurysmal thrombi or systemic emboli, leading them to suggest that long-term use of anticoagulants is unnecessary in patients with left ventricular aneurysms and "remote" myocardial infarctions. Of their 17 patients receiving anticoagulants, nine had mural thrombi and one of the 17 patients had a clinical event compatible with a systemic embolus; of the 41 patients not receiving anticoagulants, 29 (71 percent) had mural thrombi and 1 (2 percent) of the 41 had a clinical embolic event. The low frequency of clinically apparent systemic emboli in our own 28 patients and in those reported by others supports that conclusion. In contrast, patients with chronic congestive cardiac failure after healing of acute myocardial infarction but without aneurysm ("ischemic cardiomyopathy" or "coronary dilated cardiomyopathy") commonly have left ventricular thrombi which contact blood on three sides, making dislodgment and embolism a high possibility (Fig 2 and 3). Distinguishing patients with ischemic cardiomyopathy, however, from those with true left ventricular aneurysm after healing of acute myocardial infarction is extremely difficult in the absence of left ventricular angiography. Only two of our 18 patients with left ventricular aneurysm and without left ventricular angiography had the diagnosis of aneurysm made clinically. Thus, in the absence of angiography but in the presence of severe chronic congestive heart failure after healing of acute myocardial infarction, anticoagulants may be warranted in most patients.

In conclusion, a precise definition of "aneurysm" is useful in analyzing patients with "left ventricular aneurysm." Clinical events compatible with systemic emboli are rare despite the frequent presence of intraaneurysmal thrombi in patients with healed myocardial infarcts and anatomic left ventricular aneurysms; the infrequency of systemic emboli probably results from the intraaneurysmal location of the thrombus and the relatively small portion of thrombus exposed to the left ventricular cavity; although it may be reasonable to withhold anticoagulants from patients with left ventricular aneurysm, the difficulty in diagnosing aneurysm in the absence of angiography leads us to suggest their use in most patients with severe, chronic congestive heart failure after healing of acute myocardial infarction.

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