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Evidence of the inaccuracy of standard echocardiographic and angiographic criteria used for the recognition of true and "false" left ventricular inferior aneurysms

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SUMMARY Diagnosis of so-called false aneurysms of the left ventricle after infarction is judged to be important because the risk of rupture is high and resection of aneurysms with a narrow orifice is usually successful. Aneurysms with larger communication orifices are less likely to rupture. Echocardiographic and angiographic criteria have been devised to classify left ventricular aneurysms into two distinct types. In four cases of inferior aneurysms the echocardiographic and angiographic criteria were typical of a "false aneurysm" but the defects were diagnosed as true aneurysms after intraoperative and histological examination. These aneurysms were characterised by their site in the inferior wall and by late diagnosis and treatment, which may have influenced their occurrence and determined the development of their characteristic shape.

These findings suggest that the classic echocardiographic and angiographic diagnostic criteria for "false" aneurysms may have to be abandoned.

Left ventricular aneurysms with narrow communication orifices are more likely to rupture than aneurysms with wider necks. In 1975 Roelandt *et al* established echocardiographic criteria to differentiate between so-called false and true aneurysms.[1] Angiographic[2] and anatomical criteria[3] have also been developed. The four patients we report had an inferior left ventricular aneurysm that developed after myocardial infarction; their echocardiographic and angiographic features were in every respect those that are regarded as being characteristic of a "false" aneurysm. At operation and histological examination, however, these so-called false aneurysms were found to be "true" aneurysms.

These findings suggest that the classic diagnostic criteria for "false" aneurysms may have to be abandoned, at least for inferior aneurysms.

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Case report

All four patients had an acute myocardial infarction as the first sign of coronary artery disease. Diagnosis of myocardial infarction was delayed (12 hours to 2 months), mean 18 days after the onset of chest pain. This implied that there had been a long delay before bed rest and treatment were started (more than three days in three patients). The electrocardiogram indicated an inferior or inferolateral transmural infarction in all instances. All the patients had symptoms (congestive heart failure (two patients), angina pectoris (one patient), or palpitation related to ventricular extrasystoles (one patient)). Echocardiographic examination (Roche-Kontron RT 400 or Diasonics CV 4000) showed an inferior or inferolateral aneurysm in every patient between the mitral annulus and one of the papillary muscles, that was typical of a false aneurysm (fig). The following features were present: (*a*) evidence of a narrow orifice between the left ventricular chamber and the "aneurysm"; (*b*) at the end of systole the internal diameter of the orifice (O max) was much smaller than the maximal internal diameter (D max) of the aneurysmal sac and a ratio O max/D max was much

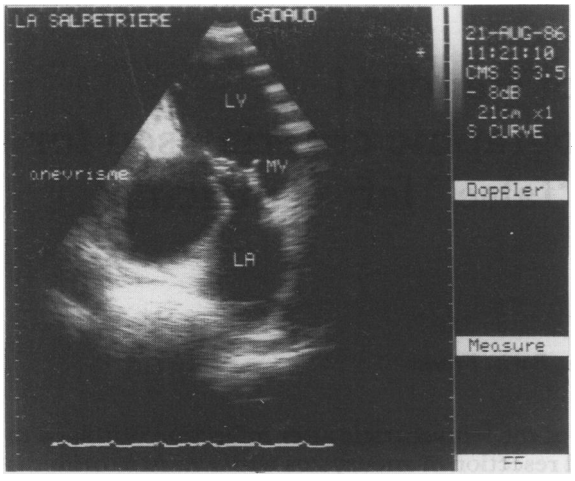

Figure *An apical two chamber echocardiogram showing an aneurysm of the inferior left ventricle. LV, left ventricle; LA, left atrium; MV, mitral valve.*

less than one; (*c*) the "aneurysm" was saccular and there was an acute angle between the aneurysmal wall and the ventricular wall.[4-6] The expansion of the aneurysmal formation in systole is not usually regarded as being specific.[4-6] The best echographic views for imaging the aneurysmal sac were the apical two chamber and subxiphoid four chamber. Left ventricular function was depressed in all the patients, with a fractional shortening of less than 26% (20–26%). The end diastolic diameter was also abnormal (60–70 mm). Angiographic data were consistent with the echocardiographic data and also suggested evidence of a false aneurysm[4 7] (table). The table also shows other angiographic findings. The four patients were operated on because of their symptoms and because they were thought to have a false aneurysm.

Table *Haemodynamic and angiographic data*

Data	*Patient* 1	2	3	4
Site	Inf	Inf	Inf lat	Inf
EF	0·39	0·25	0·37	0·28
LVEDP (mm Hg)	16	11	10	19
EDV (ml/m²)	143	148	91	283
ESV (ml/m²)	87	110	57	204
An EDV (ml/m²)	139	8	31	104
An ESV (ml/m²)	151	14	41	70
O max/D max	0·38	0·60	0·67	0·49
Coronary arteries (% stenosis)	Cx 100%	RCA 100% LAD 75%	Cx 100% RCA 90%	Cx 100%

EF, ejection fraction; LVEDP, left ventricular end diastolic pressure; EDV, end diastolic volume; ESV, end systolic volume; An EDV, aneurysm end diastolic volume; An ESV, aneurysm end systolic volume; Cx, circumflex artery; RCA, right coronary artery; LAD, left anterior descending artery.

But the intraoperative and histological findings were typical of a true aneurysm according to several criteria. At operation a true aneurysm has a circumscribed area of scar tissue which is thin and often adherent to the pericardium. In this area elements of the original wall can be identified and the area may or may not bulge paradoxically in systole.[4-6 9] Histologically a so-called true aneurysm is characterised by the presence of myocardial cells and coronary vessels in the aneurysmal wall.[3]

The aneurysms were resected in all four patients; one patient also had coronary artery bypass grafting. After at least one year of follow up all the patients were in New York Heart Association functional class I or II without angina.

Discussion

The diagnostic criteria for true and false aneurysms have been developed and are now routinely used.[1 4-6] These criteria were derived from small series of patients.[1 4-6] None of these series had more than five patients with a false aneurysm. Diagnoses were usually based solely on echocardiography and angiography because few patients had a surgical and histological examination. In our four patients the echocardiographic and angiographic findings were typical of a "false" aneurysm but true aneurysms were found at operation. Neither the width of the communication orifice, the ratio O max/D max, nor the angle between aneurysm wall and ventricular wall could reliably distinguish between the two types. It is possible that these criteria cannot be applied to lesions at inferior or inferolateral sites; these have not been extensively studied because they are so rare. Two features can be recognised as typical of inferior true aneurysms. Firstly, they occurred after late diagnosis therefore delaying initiation of bed rest and treatment. To our knowledge this potentially predisposing factor has not been identified previously. Secondly, an inferior infarction is located between two resistant structures—the mitral annulus and the papillary muscle—and if the wall stress is large enough an aneurysm may develop but its orifice will necessarily be narrow.

In conclusion, the present study clearly shows that the present criteria that are used to differentiate between so-called true and false aneurysms should be reconsidered, particularly in the case of inferior or inferolateral aneurysms.

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