Editorial

Ischaemic ventricular aneurysms: true or false?

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Left ventricular aneurysm is a common complication of acute myocardial infarction. The reported incidence ranges from 4%, if the definition is restricted to a localised protrusion of the external aspect of the left ventricle, to 40%, if localised akinesia or dyskinesia during systole is the criterion.[1] It has become a common clinical practice to classify ventricular aneurysms with an external bulge as true or false.[2][3] The wall of the true aneurysm is derived from the myocardium itself whereas the wall of the false aneurysm (pseudoaneurysm) is derived from the pericardium (figure). The pseudoaneurysm is thought to be the result of a "near miss" myocardial rupture during an acute infarct with the formation of a subpericardial haematoma.[4-6] An acute pericarditis develops and fibrous thickening of the pericardium encompasses the haematoma, ultimately forming the free wall of a sac that connects by a narrow aperture with the left ventricular cavity. The designation of true or false applies solely to the postulated origin of the wall of the sac; both are aneurysms as defined as an external bulge filled with pulsating blood.

Although pseudoaneurysms were first described at necropsy, this approach cannot either establish the frequency of pseudoaneurysms in living patients or define the risk of rupture. Basic principles suggest that the risk that a haematoma retained only by the visceral pericardium will rupture fatally within a day or two must be very high; long term survival with the formation of a pseudoaneurysm would accordingly be rare.

Two series composed of nine pathologically proven cases of pseudoaneurysm, in which echocardiography had been carried out in life, laid down criteria for the diagnosis.[2][7] The diagnostic echocardiographic features were that the maximal internal diameter of the neck (O max) was less than the

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maximal parallel internal diameter of the sac (D max) and that the ratio O max/D max never exceeded 0·5. The ratio for true aneurysms was > 1.

A paper in this issue of the *British Heart Journal*[8] challenges the view that pseudoaneurysms can be diagnosed in life with such confidence. Four lesions that conformed to the echocardiographic criteria of "false aneurysms" (pseudoaneurysms) were found to be "true" aneurysms when their wall was subsequently examined histologically. The histological criteria used to establish the myocardial origin of the aneurysm wall were the presence of residual islands of myocytes and coronary arteries draping the external surface of the sac. Both criteria are established as the morphological features that distinguish true aneurysms from pseudoaneurysms, in which there is no residual myocardium or coronary arteries in the wall.[2][6][7]

For true ventricular aneurysms therefore there is a spectrum from those with a wide neck, at one extreme, to those with a narrow neck relative to the size of the aneurysm sac at the other extreme (figure). It is aneurysms with narrow necks that cannot be distinguished from pseudoaneurysms at echocardiography. Aneurysms with narrow necks and large external sacs that extend behind the intact portion of the ventricular wall produce a typical parrot beak outline on angiography and can be identified as pseudoaneurysms with more certainty. Pseudoaneurysms with a similar shape occur as congenital diverticula or after trauma.[9][10]

The spectrum of shapes in true ventricular aneurysms may result from the different mechanisms by which they are formed. All ischaemic ventricular aneurysms arise from transmural infarction[11]; aneurysms can arise through the stretching, sliding, or tearing of muscle bundles in the necrotic myocardium of the acute infarct. The acute infarct rapidly stretches to form an aneurysm, and later fibrous replacement merely stabilises the existing shape. An alternative mechanism is that the aneurysm does not
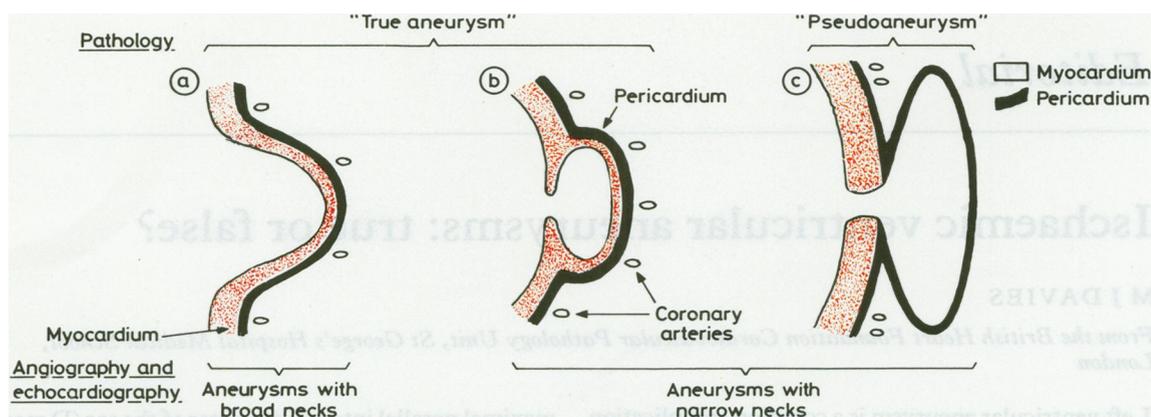
Figure   *Aneurysms with narrow necks on echocardiography (b, c) may either have their wall derived from the myocardium or the pericardium. So clinical use of the terms "true" and "false" is confusing and irrelevant.*

begin to form until the necrotic infarct has at least, in part, been replaced by fibrous tissue, and aneurysm growth is the result of the weakness of recently formed collagen under high stress; if an aneurysm has not formed by three months the reparative fibrosis is sufficiently established to prevent any dilatation of the healed infarct.[3]

There is experimental evidence that stretching of necrotic myocardial tissue before the start of fibrous replacement is the predominant mechanism of aneurysm formation.[12] In human beings acute expansion is a feature of anterior infarction and is associated with a high early mortality from rupture. Once the aneurysm is stabilised by replacement of the necrotic tissue by collagen the risk of rupture becomes slight but mortality from heart failure remains high.[3] Infarct expansion is rare in the acute phase of posterior/inferior infarction yet posterior aneurysms with narrow necks are twice as common on the posterior wall as on the anterior wall, which suggests that the process leading to the formation of posterior aneurysms is different. Small endocardial tears often develop over acute infarcts; such tears may allow the formation of an intramural haematoma and may provide the basis for the development of a narrow necked aneurysm with walls derived from the subpericardial myocardium. The tendency for narrow necked aneurysms to be more common on the posterior/inferior wall may also reflect variation in myocardial thickness or support by surrounding structures such as the base of the papillary muscles.

Because echocardiography cannot distinguish the exact origin of the wall in aneurysms with a narrow neck, the terms "true" and "false" have no clinical relevance. Such a distinction can only be made by pathological examination. The risk of late rupture is said to be higher for aneurysms with narrow necks than for those with broader necks.[4 6 13 14] If this is true, it probably reflects the thickness of the aneurysm wall rather than its histological origin (that is myocardium or pericardium). Claims that aneurysms with narrow necks are much more susceptible to late rupture are, however, largely based on necropsy reports of a few fatal cases and not on a valid estimate of the number of living subjects at risk with unruptured aneurysms of this type.

References

1 Tibbutt DA. True left ventricular aneurysm. *Br Med J* 1984;289:450–1.
2 Gatewood RP, Nanda NC. Differentiation of left ventricular pseudoaneurysm from true aneurysm with two dimensional echocardiography. *Am J Cardiol* 1980;46:869–78.
3 Visser CA, Kan G, Meltzer RS, Koolen JJ, Dunning AJ. Incidence, timing and prognostic value of left ventricular aneurysm formation after myocardial infarction: a prospective, serial echocardiographic study of 158 patients. *Am J Cardiol* 1986;57:729–32.
4 Roberts WC, Morrow AG. Pseudoaneurysm of the left ventricle. An unusual sequel of myocardial infarction and rupture of the heart. *Am J Med* 1967;43:639–44.
5 Cone RB, Hawley RL. Psuedoaneurysm of the heart following infarction. *Arch Pathol* 1964;77:166–71.
6 Vlodaver Z, Coe JI, Edwards JE. True and false left ventricular aneurysms. Propensity for the latter to rupture. *Circulation* 1975;51:567–72.
7 Catherwood E, Mintz GS, Kotler MN, Parry WR,

Segal BL. Two-dimensional echocardiographic recognition of left ventricular pseudoaneurysm. *Circulation* 1980;**62**:294–303.

8 Lascault G, Reeves F, Drobinski G. Evidence of the inaccuracy of standard echocardiographic and angiographic criteria used for the recognition of true and "false" left ventricular inferior aneurysms. *Br Heart J* 1988;**60**:125–7.

9 Higgins CB, Lipton MJ, Johnson AD, Peterson KL, Vieweg VR. False aneurysms of the left ventricle. *Radiology* 1978;**127**:21–7.

10 Mills PG, Rose JD, Brodie BR, Delany DJ, Craige E. Echophonocardiographic diagnosis of left ventricular

pseudoaneurysm. *Chest* 1977;**72**:365–7.

11 Hochman JS, Bulkley BH. Pathogenesis of left ventricular aneurysms: an experimental study in the rat model. *Am J Cardiol* 1982;**50**:83–8.

12 Weisman HF, Healy B. Myocardial infarct expansion, infarct extension, and reinfarction: pathophysiologic concepts. *Prog Cardiovasc Dis* 1987;**30**:73–110.

13 Winzelberg GG, Strauss HW, Bingham JB, McKusick KA. Scintigraphic evaluation of left ventricular aneurysm. *Am J Cardiol* 1980;**46**:1138–43.

14 Van Tassel RA, Edwards JE. Analysis of forty cases including nine examples of left ventricular false aneurysm. *Chest* 1972;**61**:104–15.