LONG SURVIVAL WITH A CARDIAC ANEURYSM

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Although rarely reported, cardiac aneurysms are not purely pathological curiosities that are discovered by chance at autopsy. They can sometimes be recognized during life by correlation of clinical and radiographic findings. Thus, a history of anginal pain, followed by pericarditis and recovery, should suggest the possible development of a parietal aneurysm of the heart, if the patient survives long enough. Further, in spite of the usually accepted view, it is not true to assert that the prognosis of such chronic aneurysms is necessarily bad. Thus, I have been able to follow the development of such a condition for some 13 years in a man who is still free from symptoms.

There are not many reports of long survival after the development of a cardiac aneurysm, but Laubry et al. (1930) mention two patients who lived 10 and 12 years respectively, while Clerc and Deschamps (1931) observed survival for 13 years.

My case was that of an agricultural engineer, aged 45, whose illness started with a series of mild anginal attacks, with considerable intervals between them, over a period of about 5 years. On October 25, 1935, after a more severe physical effort than usual, he developed extremely severe precordial pain, which could not be alleviated completely by any drugs. All the clinical features of coronary occlusion were present—fall of blood pressure, thready pulse, tachycardia, cyanosis, dyspnea, sweating, anxiety, etc.

Two days later, the temperature rose to 103, accompanied by rigors and signs of left-sided pleurisy. Then, on the fourth day, pericarditis developed, dry at first, but with some effusion later.

All the symptoms diminished in intensity from the twelfth day onwards, the temperature returning to normal, perhaps as the result of giving salicylates. The patient left the hospital 48 days after the original attack.

The radiographic changes are of particular importance, because they have been followed over a period of 13 years.

On the tenth day after the coronary occlusion, the heart was flask-shaped, and no pulsations could be detected, i.e., the appearances were typical of pericardial effusion (Fig. 1).

On the forty-second day of the stay in hospital, the cardiac shadow itself was within normal limits, but there was a small triangular prominence on the left border of the left ventricle, which, at that time, was wrongly interpreted as being due to a pericardial adhesion (Fig. 2). This is now known to have been the early stage of a parietal aneurysm.

Twenty months later, the aneurysm was obvious, the sac being relatively large (Fig. 3). This appearance was detected fortuitously when the chest was being examined because of a suspicion of pulmonary tuberculosis; but it was not until September 18, 1940, five years after the original attack, that the real nature of the condition was recognized.

The teleradiograms taken at this time show clearly that the projection is continuous with the left ventricle; and, when screened in various positions, it was seen to dilate during systole of the left ventricle—the condition known as "paradoxical diastole." These appearances, considered in conjunction with the history, demonstrated clearly that the condition was a parietal aneurysm of the heart, not, as had tentatively been supposed, a tumour or a cyst, because these do not pulsate although these may show transmitted pulsation.

Thus, for a period of five years after the attack of coronary occlusion, the diagnosis was uncertain, and had varied from tumour, myocardial cyst, and even a ordinary pericardial adhesion. The present case bears out the old aphorism that, in order to diagnose a disease, it is necessary first to think of it. If we had not considered the possibility of a parietal

* Translator’s note. Codounis makes no reference to the possibility that there may have been a pericardial adhesion, which played a part in causing the subsequent aneurysm.
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Fig. 1.—31/10/35. Pericardial effusion after cardiac infarction.

Fig. 2.—3/12/35 (42 days after cardiac infarction). A small projection is seen on the left lateral wall of the left ventricle.

Fig. 3.—3/8/37. A large aneurysmal sac is visible.

Fig. 4.—18/7/40. The sac is still present, but shows a rather diffuse edge.
aneurysm, we should not have attempted to demonstrate it by looking for paradoxical diastole: a further proof, if one be needed, of the value of screening in cardiology.

Fig. 4, which was taken seven years after the attack of pericarditis, shows slight decrease in the size of the sac, which still shows the phenomenon of paradoxical diastole, although its density has changed a little, now being different from that of the ventricle. Oddly enough, paradoxical diastole was even more striking than at earlier examinations.

Towards the end of 1945, fifteen years after the beginning of the illness, the patient, who felt perfectly well, showed no clinical signs of heart disease except occasional slight anginal pain on exertion or after repeated bending. From this time onwards, there has been little change in the radiographic appearances, except that the sac shows areas of increased density, which are, presumably, due to calcification: a feature which was more easily recognized on screening than in films. In spite of this, pulsation of the aneurysm was still distinctly visible.

In our view orthodiagraphy is of less value than telediography and kymography, mainly because of the subjective element in the former, which leads to considerable differences between the orthodiagrams made by different radiologists, even on the same day.

It seems clear that the radiographic history of the cardiac aneurysm recorded here has all the features that would be expected from our knowledge of myocardial infarction. And the fact that the aneurysmal sac plays only a passive part during ventricular systole is consistent with the replacement of muscle fibres by scar-tissue.

Differential diagnosis is not difficult in cases in which the phenomenon of paradoxical dilatation of the aneurysmal sac can be detected; but, if the contents become thrombosed or the wall becomes calcified, this may disappear. Then, the possibility of such conditions as hydatid cyst, neoplasm, pericardial diverticulum, etc., will need to be considered.

Again, large aneurysms of the heart may cause such great alteration in the cardiac outline as to cause possible confusion with pericardial effusion, but the invisibility of the heart-beat in that condition should prevent mistakes.

Finally, coronary aneurysms present an identical radiographic appearance, but usually occur on the right, whereas parietal aneurysms are almost invariably left-sided. Then also, coronary aneurysms are due to syphilis and develop insidiously, without any history of the type found in coronary thrombosis.

**SUMMARY**

A case illustrating the development of an aneurysm of the heart-wall during a period of 13 years is recorded in detail; and special attention is drawn to the occurrence of paradoxical diastole as an important diagnostic feature.

**REFERENCES**
