MECHANICAL BEHAVIOUR OF VENTRICULAR ANEURYSMS

I. MIRSKY
Department of Medicine,
Harvard Medical School
and Peter Bent Brigham Hospital,
Boston, Massachusetts, U.S.A.

P. L. MCGILL and R. F. JANZ
Data Systems Analysis Section,
The Aerospace Corporation,
Los Angeles, California, U.S.A.

A mathematical model describes the mechanical behaviour of ventricular aneurysms assuming a spherical geometry for the left ventricle. Employing pressure-volume data obtained from normal dog hearts 1–2 hours after infarction, conditions are obtained on infarct thickness and angle of damage for ventricular rupture to occur. The results indicate that rupture is more likely to occur in the early period following infarction and that the dominant factor is the per cent thickness of the infarct.

Introduction. Lancisius (1740) first described a left ventricular aneurysm as being a generalized dilatation of the heart chamber and Cruveilhier (1816) attributed it to myocardial fibrosis. However, it was not until Tennant and Wiggers (1935) demonstrated the presence of systolic paradoxical motion in acutely ischemic myocardium that the physiological implications of this ventricular injury became apparent. This systolic paradoxical expansion was shown by Murray (1947) to be associated with a diminished cardiac output and falling blood pressure. Since this time, a number of animal and human studies have been conducted in relation to the performance of infarcted myocardium, among these being those by Tyson et al. (1962), Austen et al. (1962), Forward et al. (1966), Heimbecker et al. (1967), Klein et al. (1967), Gorlin et al. (1967), Hood et al. (1970), Kumar et al. (1970), Feild et al. (1972), Diamond and Forrester (1972), Forrester et al. (1972), Parmley et al. (1973), Swan et al.
In association with these studies, there has been a concentrated effort towards the development of methods for estimating infarct size so as to enable appropriate selection of candidates to be made for coronary bypass surgery.

It is well recognized that aneurysm formation in the walls of the left ventricle is a sequel of myocardial infarction. Gorlin et al. (1967) studied ventricular aneurysms in a number of patients with coronary artery disease and noted that in 16 of the patients there was a lack of wall motion in the region of the aneurysm and in 8 patients, local paradoxical expansion occurred. Contrary to expectation, aneurysms were composed solely of thin-walled fibroses in only seven patients and seven additional patients had mixed “full thickness” muscle and fibrosis. The majority of the aneurysms were located at the apex or on the anterior surface and varied in size from 10 to 50 per cent of estimated left ventricular surface area. Parmley et al. (1973) quantitated the length–tension relations of surgically resected human ventricular aneurysms and also noted that aneurysms were of the fibrous, fibrous–muscular and muscular type. Their analysis indicated that fibrous aneurysms produce their primary mechanical disadvantage by a loss of contractile tissue with minimal systolic expansion whereas acute muscular aneurysms produce mechanical disadvantage by both loss of contractile tissue and significant paradoxical systolic expansion.

Lewis et al. (1969) have found that rupture of aneurysms occur during the first week of infarction and that these ruptured infarcts are often of the transmural type. These results are supported by the recent studies of Penther (1976) who in addition states that cardiac rupture is a far more common cause of death than has previously been assumed. It is therefore important that we gain a better understanding of the mechanism by which the myocardial infarction relates to the genesis of the aneurysm and also its mechanical behaviour.

Apart from the study by Vayo (1966) who developed a model relating the amount of muscle shortening to the amount of “inactive” muscle in the aneurysmal area, the only significant mathematical model of ventricular aneurysms was developed by Lowe and Love (1948) and forms the basis for the present study. The present analyses may have clinical applications and differs from that of Lowe and Love (1948) in several respects.

(a) The diastolic pressure–volume (P–V) relations obtained from infarcted dog hearts will be employed rather than pressure and geometry data at an isolated time in systole.

(b) Stress–strain relations for the normal muscle are assumed to be exponential in form and are consistent with the elastic properties of the particular ventricular muscle in question. This contrasts with the linear length–tension relation employed by Lowe and Love and obtained on the basis of experimental data with frog cardiac muscle.