Aortic root and left atrial wall motion

An echocardiographic study

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The echocardiographically recorded movement of the aortic root was studied by analysing the relation between posterior aortic wall motion and other intracardiac events. The systolic anterior movement of the aortic root continued beyond aortic valve closure and in cases with mitral regurgitation began significantly earlier than in normal subjects. The diastolic rapid posterior movement began after mitral valve opening but did not occur in patients with mitral stenosis. The total amplitude of aortic root motion was increased in patients with mitral regurgitation, diminished in cases of mitral stenosis, and was normal with aortic regurgitation. In patients with atrioventricular block an abrupt posterior movement followed the P wave of the electrocardiogram irrespective of its timing in diastole. These observations correlate with the expected changes in left atrial volume during the cardiac cycle both in the normal subjects and patients with heart disease.

The results support the hypothesis that phasic changes in left atrial dimension are largely responsible for the echocardiographically observed movement of the aortic root and indicate a potential role for echocardiography in the analysis of left atrial events.

Echocardiographic examination of the aortic root and aortic valve has proved to be valuable in the assessment of patients with a variety of disorders including aortic valve disease (Gramiak and Shah, 1970; Feizi *et al.*, 1974), dissecting aneurysm of the ascending aorta (Nanda *et al.*, 1973), and supra-valvar aortic stenosis (Bolen *et al.*, 1975). At the same time it is possible to make an estimate of left atrial dimension (Hirata *et al.*, 1969; tenCate *et al.*, 1974) which is helpful in cases of mitral stenosis and other conditions in which left atrial dilatation is a feature. Recently attention has been directed to observation of the movement of the aortic root and in particular the amplitude of this movement. It has been shown that this may vary considerably between patients (Gramiak and Shah, 1968), and a relation between the amplitude and stroke volume has been reported (Pratt *et al.*, 1976). This observation was based on the assumption that the motion of the aortic root is the result of the ejection phase of left ventricular contraction. However, combined angiocardiographic and echocardiographic studies have suggested that changes in left atrial volume may be the major determinant of aortic root motion (Strunk *et al.*, 1976a). The relation of posterior aortic wall movement to other cardiac events has not been established.

As a result of observations made on echocardiograms obtained by the routine diagnostic service at the London Hospital we also gained the impression that the motion of the aortic root is largely the result of movement of the left atrial wall and phasic changes in left atrial dimension. This implies that aortic root motion is not an index of left ventricular function but might provide useful information about left atrial events in patients with heart disease. A prospective study using echocardiography and phonocardiography was, therefore, undertaken to establish the determinants of aortic root motion throughout the cardiac cycle.

Subjects and methods

Studies were performed on 12 normal subjects and 30 patients with heart disease. The latter included 10 patients with isolated mitral stenosis, 11 with pure mitral regurgitation, 7 with free aortic regurgitation, and 2 with atrioventricular block. All the patients had characteristic clinical, electrocardiographic, radiographic, and echocardiographic findings associated with their cardiac lesion.

Echocardiograms were obtained using a Smith Kline Ekoline 20 ultra-sound recorder with a 2.25 MHz focussed transducer and a repetition rate of 1000 a second. The output of the Ekoline 20 was interfaced with a Cambridge multichannel

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photographic recorder allowing simultaneous recording of the echogram, electrocardiogram (lead II) and phonocardiogram at a paper speed of 100 mm a second. Phonocardiograms were obtained using a piezoelectric crystal microphone placed either at the cardiac apex or at the left sternal edge in the second interspace.

The mitral valve echogram was recorded in the conventional way, with care taken to ensure that both cusps were clearly seen during opening of the valve. Similarly for the aortic valve at least two cusps were visualised at the time of separation. The aortic root and left atrium were then examined ensuring clear delineation of the posterior aortic wall and posterior left atrial wall throughout the cardiac cycle. To help standardise the transducer position, elements of the aortic valve cusps were always included within the aortic lumen when recording the aortic root and left atrial echogram.

The following time intervals and amplitudes were measured:

- Q—MC (ms)—onset of Q wave to mitral valve closure;
- Q—AVO (ms)—onset of Q wave to aortic cusp separation;
- Q—peak AVO (ms)—Q wave to maximum separation of aortic cusps;
- A₂—MVO (ms)—aortic component of second heart sound (or aortic cusp apposition) to mitral cusp separation;
- A₂—peak MVO (ms)—aortic valve closure to maximum separation of mitral valve cusps;
- Q—start ARM (ms)—Q wave to start of anterior motion of aortic root;
- A₂—peak ARM (ms) valve closure to most anterior position of aortic root;
- Amp ARM (mm)—amplitude of aortic root motion;
- LA max (mm)—maximum left atrial dimension.

All measurements given were calculated as the average of at least five cardiac cycles. Time intervals were measured to the nearest 5 ms. The differences in the values found for these variables in the patient groups when compared to the normal subjects were analysed using Student's unpaired t test.

Results

(a) NORMAL SUBJECTS

The typical pattern of aortic root motion in the normal subjects is shown in Fig. 1. During ventricular systole there is an anterior movement followed in the early part of diastole by a posterior motion. In later diastole little further movement is seen until after the P wave of the electrocardiogram though in some subjects a definite slow anterior deflection is seen. Following the P wave a rapid

posterior movement occurs, which usually consists of two distinct components with a brief anterior movement separating the two. The details of the timing of the cardiac events are shown in the Table. In all normal subjects the anterior systolic motion of the aortic root did not begin until after the onset of aortic valve opening occurring with a mean delay of 121 ms after Q. In 3 cases it was delayed by up to 14 ms after peak opening of the aortic valve; in one subject its onset was synchronous with peak aortic valve opening and in the remainder it preceded peak aortic opening. In all subjects the anterior motion continued beyond aortic valve closure and the onset of mitral valve opening but was always completed before peak opening of the mitral valve had occurred. In some cases a transient posterior movement occurred synchronous with aortic valve closure. The first component of the presystolic posterior movement always followed the P wave of the electrocardiogram and was completed before mitral valve closure. The second component invariably occurred after mitral valve closure mainly occupying the period of isovolumic ventricular contraction. In between these two presystolic posterior movements a small anterior displacement is seen (Fig. 1).

The mean amplitude of posterior aortic wall motion for the normal group was 11.6 mm (SEM = 0.4). The mean left atrial dimension at its maximum point was 31.3 mm (SEM = 1.2).

(b) MITRAL STENOSIS

In all patients with mitral stenosis a distinct deviation from the normal pattern of aortic root motion was seen (Fig. 2). During ventricular systole the aortic root moved anteriorly with a temporal relation to aortic valve opening similar to that in the normal subjects. However, mitral valve opening occurred earlier though this difference was not statistically significant. The peak of the anterior movement of the aortic root also occurred earlier but this too was not statistically significant. As in the normal group this followed mitral valve opening in each patient. During the early part of diastole the rapid posterior movement seen in the normal subjects was replaced by a slower motion which continued throughout the length of diastole, thus replacing the plateau in late diastole found normally. Since all the patients in this group had atrial fibrillation the posterior motion associated with atrial systole in the normal subjects was not seen. Closure of the mitral valve was significantly delayed in the patients with mitral stenosis. The relation of aortic root movement to other cardiac events is shown in the Table.

The mean amplitude of posterior aortic wall

motion (Fig. 3) was 7.8 mm (SEM=0.5) which was significantly less than for the normal subjects ($P < 0.001$). Maximum left atrial diameter was 47.7 mm (SEM=2.0) which was significantly greater than the normal group (Fig. 4).

(c) MITRAL REGURGITATION

The pattern of movement of the posterior aortic wall in these patients was similar to that seen in the normal subjects (Table). However the onset of forward movement of the aortic root occurred 96 ms after Q and this was significantly earlier ($P < 0.001$) than in the normal group. Similarly, the interval from aortic valve closure to the maximum anterior position of the aortic root was also significantly shorter at 71 ms. The amplitude of posterior

aortic wall motion was significantly greater than for the normal group ($P < 0.005$) and also greater than for the other patients studied (Fig. 3). The left atrial diameter (52 mm) was also significantly greater than for the normal group (Fig. 4).

(d) AORTIC REGURGITATION

In patients with aortic regurgitation forward movement of the aortic root began 122 ms after Q (Table) which was similar to the finding in the normal group but the maximum anterior position was reached significantly later (98 ms after aortic valve closure) than in the normal subjects. Opening of the mitral valve was also delayed significantly in these patients, occurring 84 ms after aortic valve closure.

The amplitude of posterior aortic wall move-

Table Time relation of intracardiac events derived from echocardiograms and phonocardiograms

	Q-MC	Q-AVO	Q-start ARM	Q-pkAVO	A ₁ -MVO	A ₁ -pkARM	A ₁ -pkMVO
Normal	58 ± 2	96 ± 3	121 ± 3	126 ± 4	61 ± 4	85 ± 2	115 ± 5
Mitral stenosis	76 ± 4 P < 0.005	103 ± 4	125 ± 6	127 ± 5	55 ± 4	74 ± 8	97 ± 6
Mitral regurgitation	63 ± 5	96 ± 4	96 ± 2 P < 0.001	120 ± 5	62 ± 4	71 ± 7 P < 0.005	117 ± 6
Aortic regurgitation	53 ± 7	87 ± 6	122 ± 4	115 ± 8	84 ± 10 P < 0.01	98 ± 9 P < 0.05	148 ± 13 P < 0.01

AVO, aortic valve opening; pkAVO, peak separation of aortic cusps; MVO, mitral valve opening; ARM, aortic root movement. Time intervals are measured in ms and are given as the mean and standard error of the mean.

Fig. 1 Normal left atrial and aortic root echocardiogram. The anterior systolic motion continues beyond aortic valve closure and is followed by a rapid posterior movement in early diastole. A further posterior movement follows the P wave.

ment was 11.6 mm (SEM=1.1) and the left atrial diameter was 33.7 mm (SEM=1.8) both of which did not differ significantly from the normal values (Fig. 3 and 4).

(e) ATRIOVENTRICULAR BLOCK

Two patients with complete atrioventricular block were investigated to examine the effect of atrial systole on the movement of the posterior aortic wall. Detailed measurements were not made in these cases. The pattern of aortic root movement seen is shown in Fig. 5. Irrespective of its timing in diastole the P wave of the electrocardiogram was followed by a posterior movement of the aortic wall which then maintained its new position with only minor variation for the rest of diastole.

Discussion

Angiocardiology and left atrial echocardiography show that considerable changes in left atrial anteroposterior dimension occur throughout the cardiac cycle (Strunk *et al.*, 1976a). Since the posterior wall of the left atrium is fixed in position by its attachment to the pulmonary veins, virtually all of this dimension change is the result of movement of the anterior wall of the atrium which is attached to the aortic root and posterior aortic wall. This motion

of the last may, therefore, be the result of phasic changes in left atrial volume or alternatively may be the result of an isovolumic change in left atrial shape. In the latter case a reduction in left atrial

Fig. 3 Amplitude of aortic root motion in the four groups studied. Bars indicate standard error of the mean.

Fig. 2 Left atrial and aortic root echocardiogram in mitral stenosis. The amplitude of root movement is reduced and the rapid early diastolic posterior movement is replaced by a slower motion continuing throughout diastole.

Fig. 4 Left atrial diameter in the four groups studied. Bars indicate standard error of the mean.

anteroposterior dimension must be accompanied by an increase in dimension in some other plane. While it is accepted that the diameter of the aortic root changes only slightly through the cardiac cycle except in patients with free aortic regurgitation (Arcilla *et al.*, 1961) it has nevertheless been suggested that aortic root motion is the result of left ventricular systole and especially the ejection phase (Pratt *et al.*, 1976). This concept has been extended by a report apparently indicating that the amplitude of aortic posterior wall motion is a useful indicator of left ventricular performance and may even be used to quantify the stroke volume (Pratt *et al.*, 1976). If the concept is correct it would suggest that the left atrial dimension changes in a passive manner as a result of atrial isovolumic shape alterations related to displacement of other cardiac structures, in particular the aorta; further, these shape changes would be determined by the function of the left ventricle. The results of the studies reported here, however, cast serious doubts on the validity of this concept.

In the first instance no increase in the amplitude of aortic posterior wall motion could be shown in patients with free aortic regurgitation, despite the large stroke volume usually present in these cases (Miller *et al.*, 1965). Secondly, in patients with gross mitral regurgitation the amplitude of movement was increased though forward flow into the aorta would not be expected to be raised (Miller *et al.*, 1965). Thirdly, atrial systole distinctly alters the

position of the aortic root causing a reduction in the left atrial dimension.

Further evidence suggesting that atrial volume is the major determinant of aortic root movement is obtained from the analysis of the temporal relations of this motion to other intracardiac events. The anterior movement which occurs during ventricular systole always continues beyond aortic valve closure even in patients with aortic regurgitation and cannot, therefore, be the result of ventricular contraction. It is always completed after the mitral valve has started to open but before peak separation of the cusps has occurred, this relation being maintained in the patients with aortic regurgitation in whom mitral valve opening is delayed. It is, therefore, compatible with an increase in left atrial dimension during systole reflecting the enlarging volume of that chamber when the mitral valve is closed. After mitral cusp separation begins left ventricular filling occurs, the left atrial dimension decreases and the aortic root moves rapidly posteriorly. In patients with mitral stenosis the rapid ventricular filling phase is lost and this is reflected by a more gradual posterior motion of the aortic root and reduction in left atrial dimension. This can be related to the severity of the mitral stenosis (Strunk *et al.*, 1976b). Furthermore, this movement continues throughout the length of diastole consistent with the prolongation of left ventricular filling and altered pattern of mitral valve flow (Kalmanson *et al.*, 1976).

Fig. 5 Left atrial and aortic root echocardiogram in atrioventricular block. The P wave is followed by a posterior movement of the aortic root irrespective of its timing in diastole.

In sinus rhythm the posterior movement of the aortic root during atrial systole ends with mitral valve closure but during isovolumic ventricular systole a further small posterior motion occurs. This clearly cannot relate to ventricular ejection but may be the result of a change in left atrial shape as the mitral valve moves in a posterior and superior direction away from the apex of the left ventricle (Strunk *et al.*, 1976a). This small change in left atrial dimension during isovolumic ventricular contraction is, therefore, the only event that cannot be explained on the basis of atrial volume but rather as a consequence of a change in left atrial shape.

These results indicate that movement of the posterior aortic wall is primarily determined by left atrial events rather than the effects of left ventricular systole. This finding has immediate practical significance since it implies that observation of posterior aortic wall (or more correctly left atrial wall) motion cannot be used to assess the performance of the left ventricle. In particular the apparent relation between stroke volume and amplitude of aortic root movement does not apply in the presence of mitral or aortic regurgitation. However, left atrial echocardiography can provide information related to left atrial volume and specifically left atrial anteroposterior dimension.

In mitral stenosis the left atrial dimension changes slowly during diastole. The change in dimension is also considerably smaller than normal as a result of reduced posterior aortic wall motion. This may reflect a diminished left atrial blood flow but could also result from reduced left atrial compliance. In addition the left atrial volume is increased so that small changes in dimension may be associated with normal changes in volume. In patients with mitral regurgitation even larger left atrial dimensions are found but the amplitude of left atrial wall movement, and therefore the amplitude of variation in dimension, were also increased. This results from the large phasic changes in left atrial volume expected in this condition.

Although the mitral valve echogram can be used as an index of the severity of mitral stenosis (Gustafson, 1967), mitral regurgitation has proved more difficult to evaluate. Further analysis of left

atrial echograms based on measurements of phasic left atrial dimensions may yield additional information of value in this area. In particular the functional data can be expected to serve as an adjunct to the predominantly anatomical information available from the mitral echogram.

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