Delayed Peak of the Posterior Wall

A New Echocardiographic Index of Posterior Wall Aneurysm*

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Delayed posterior wall (PW) peak in the detection of PW asynergy was studied by M-mode echocardiography in 53 patients with angiographically proven PW aneurysm, hypokinesis and normal PW. The timing of the PW peak was evaluated by the intervals between (1) the aortic valve closure (Ac) and the PW peak, and (2) the R wave of the electrocardiogram and the PW peak (R-peak). The PW excursion predicted only 39 percent with aneurysm and none with hypokinesis. In contrast, 13 of 18 patients with aneurysm and 4 of 13 patients with hypokinesis demonstrated significantly delayed PW peaks occurring between 0.05 and 0.1 sec following Ac (normal 0 to 0.05 sec). A new index, a ratio of R-peak to ejection time (R-peak/ET) of 1.35 or greater was found to be highly indicative of PW aneurysm in 16 of 18 patients.

A lthough the overall predictive ability of clinical M-mode echocardiography in coronary artery disease appears to be satisfactory, detection of posteroinferior wall abnormality is rather discouraging. In the study of acute myocardial infarction by Corya et al, only 57 percent of the patients with electrographic (ECG) evidence of inferior wall infarction showed a decreased posterior wall excursion (PWE). More recently, a correlative study with angina pectoris also found less predictability (49 percent) of posterior wall echogram in the presence of angiographic evidence of posterior wall asynergy, as compared to 79 percent associated with anterior wall abnormality.

In contrast, consistent changes in the contour of posterior wall motion have been well documented in animals with experimental posterior wall infarction. One of these changes was an abnormal anterior recoil motion during isometric relaxation of the left ventricle (LV). Thus far, no clinical studies have been performed in the presence of posterior wall asynergy to evaluate this motion which could result in a delayed peak of the posterior wall, as well as a normal PWE. The purpose of this study was to determine (1) if posterior wall asynergy could be predicted by echocardiographic evidence of delayed peak of the posterior wall, and (2) if this delayed peak might be a more sensitive index than PWE in detecting posterior wall asynergy.

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METHODS

Left ventricular cineangiograms of 31 consecutive patients with posterior and/or inferior wall infarction documented by evolutionary electrocardiographic change and elevation of enzymes were evaluated for abnormalities of posterior wall contraction. Patients with either left bundle branch block or WPW syndrome on ECG were excluded from this study. The patients with a history of myocardial infarction occurring within three months prior to cardiac catheterization were not included. None of the patients had congestive heart failure at the time of the study.

Prior to coronary angiography, LV cineangiography was performed in the 30 degrees right anterior oblique projection on 35 mm film taken at 30 frames/second using a nine inch C.E. Fluricon 300 image intensification system. The LV was opacified with 45 ml of Meglumine diatrizoate (Renografin 76) injected at 15 ml/second through a No. 7 or No. 8 French angiography catheter. Patterns of abnormal contraction of the LV posterior wall were defined by the criteria previously described: “hypokinesis”-diminished regional systolic shortening in which there was less inward excursion of the posterior-inferior segment than the remaining unaffected areas; “akinesis”-absent systolic movement of the segment; “dyskinesis”-paradoxic outward systolic expansion. Dyskinesis and akinesis were considered to represent aneurysm of the corresponding ventricular segment. According to this classification, 18 of 31 patients were found to have inferoposterior wall aneurysm (group 1). An aneurysmal bulging with sharply defined margins of the inferoposterior wall was shown in 5 of these 18 patients. Thirteen of 31 patients had hypokinesis (group 2). Another 22 patients who underwent catheterization for evaluation of chest pain were also consecutively selected from the catheterization laboratory on the basis of the presence of normal coronary arteries and angiography-proven normal posterior wall (group 3). These patients consisted of 39 men and 14 women with an age range of 17 to 66 years (44±2.0, mean ± SEM).

All 53 patients had an M-mode echocardiogram recorded within 24-48 hours prior to cardiac catheterization. Echocardiographic studies were carried out with a Smith-Kline Ekoline 20A ultrasonoscope using a 2.25 MHz transducer 0.5
inch in diameter focused at 7.5 cm. The echocardiograms were recorded on a multichannel oscilloscope recorder (Honeywell 3820 or Irex 241). The patients were studied in a semi-recumbent or left lateral position. The transducer was positioned in the fourth or fifth intercostal space just to the left of the sternum. An echocardiographic M-mode scan from the aortic root to the apex of the LV was performed by the technique described by Feigenbaum to verify that the transducer was in the optimal position.

A lead 2 ECG, carotid pulse (CP), and phonocardiogram (PCG) were simultaneously recorded on the echocardiogram (Fig 1). Echoes from the interventricular septum and LV posterior wall endocardium were recorded at the level of chordae tendineae or just below the tip of the mitral leaflets at a paper speed of 50 mm/second. The posterior wall excursion is the amplitude of posterior wall motion as measured by the vertical distance between the most posterior position of the posterior wall endocardium in early systole and the peak.

Special attention was paid to the timing of the posterior wall peak and the following measurements were performed: (1) the interval between the aortic valve closure and the posterior wall peak (AC-peak), and (2) the interval between the R wave of ECG and the posterior wall peak (the R-peak interval). In order to evaluate the effect of LV ejection time (ET) on posterior wall motion, this R-peak interval was plotted against the ET in patients with normal posterior wall and posterior wall aneurysm. Delay of the posterior wall peak was then expressed by the ratio of the R-peak interval to the ET (R-peak/ET). The ET was measured on a simultaneously recorded carotid pulse (from rapid upstroke to the incisura). Mean heart rate and ET were not different between the three groups. The timing of aortic valve closure was determined by an aortic component of the second heart sound on a simultaneously recorded PCG.

The echocardiographic interpretations were performed without knowing the result of catheterization and any information on patients to eliminate observer bias. Student's t-test for paired data was utilized for statistical analysis. All values are given as mean ± standard error of mean.

**RESULTS**

**Echocardiographic Data**

Of the 22 patients with normal posterior wall (group 3), 11 patients demonstrated the posterior wall peak at the time of aortic valve closure (AC-peak = 0). In all 22, the peak was reached within 0.05 sec after aortic valve closure (0.013 ± 0.003 sec). In contrast, the group I patients with posterior wall aneurysm revealed a markedly delayed posterior wall peak with a range between 0 and 0.1 sec (0.064 ± 0.007 sec) after aortic valve closure (P < 0.001). Of the 13 patients with posterior wall hypokinesis (group 2) the posterior wall peak occurred 0.05 sec...
after aortic valve closure in four individuals (0.045 ± 0.007 sec).

Figure 2 shows the echocardiogram of a patient with posterior wall dyskinesia and ECG evidence of posteroinferior wall infarction. The CP and PCG were recorded simultaneously with the echocardiogram. The two vertical dotted lines represent the timing of R wave of ECG and aortic second sound, respectively. The posterior wall peak (vertical arrow) is markedly delayed (0.08 sec following Ac) and the R-peak/ET ratio is 1.51. It is noted that the PWE is within normal range (1.2 cm).

The R-peak intervals were plotted against the ET in groups 1 (aneurysm) and 3 (normal) (Fig 3). In both groups, the R-peak interval appeared to be prolonged as the ET increased (correlation coefficient [r] = 0.58 in group 1 and 0.76 in group 3). Although a significant overlap of the R-peak interval between groups was noted, (below the dotted line), all patients but one in group 1 with aneurysm had a longer R-peak interval than in group 3 patients for a given ET (above the solid diagonal line). As a result, these two groups could be clearly separated by an R-peak/ET ratio (Fig 4). The R-peak/ET ratio in normal subjects (group 3) measured from 1.05 to 1.34 (1.22 ± 0.02). Of the 18 patients with posterior wall aneurysm, 16 patients (92 percent) demonstrated an abnormally increased ratio (1.36 to 1.68) and the mean ratio (1.47 ± 0.03) differed significantly from those in normal subjects (P < 0.001). Of the 13 patients with hypokinesis, 4 patients had abnormal ratios. The Ac-peak interval and R-peak/ET ratio did not differ significantly between the patients in group 1 with and without bulging with sharply defined margins.

In contrast, normal PWE was observed in 11 (61 percent) of the 18 patients with an aneurysm although the mean PWE was significantly lower than the normals (0.88 ± 0.06 cm vs 1.16 ± 0.04 cm, P < 0.01). None of the patients with hypokinesis showed a decreased PWE.

**DISCUSSION**

The contraction pattern of the posterior wall on echocardiogram was described in detail by Krauz and Kennedy in 1970. The posterior wall peak, which occurs at the end of systolic ejection, is followed by a slow posterior movement during the isometric relaxation period. Subsequently, McDonald et al performed a detailed analysis of motion of both the interventricular septum and posterior wall. These investigators indicated anterior shift of the entire LV during late systole in the thorax and demonstrated that the peak of a normal posterior wall echo does not necessarily occur simultaneously with the end of systolic ejection. Another group of investigators described that the Ac-posterior wall peak interval ranged from 0 to 0.08 sec (0.024 ± 0.005 sec). Similarly, in our preliminary study of
45 normal subjects, the Ac-peak interval ranged from 0 to 0.05 sec with a mean value of 0.022 ± 0.003 sec (unpublished data). Results of the present study were in agreement with these previous observations and indicated that the posterior wall peak occurred within 0.05 sec after aortic valve closure in patients with angiographically proven normal posterior wall motion. This delayed peak in normal subjects could be attributed to the anterior shift of the entire LV during late systole as postulated by McDonald et al.11

The experimental study of posterior wall infarction by Kerber et al12 would provide strong supportive evidence for the abnormally delayed peak of the posterior wall observed herein in the patients with aneurysm. Of note in their study was that during the period of isometric relaxation at the time of a rapid fall of ventricular pressure, the stretched and bulging infarcted myocardium recoils from its abnormally posterior location; the rapid motion back to its original position results in the rapid anterior displacement. Although it should be mentioned that their study was performed by the production of an acute infarction in the animals and their observations should be of limited value, it is possible that the same mechanism observed in the animal studies could account for the delayed posterior wall peak observed in this study. This assumption is also supported by the angiographic study of regional contraction patterns in man.13,14 Sniderman et al13 demonstrated that the ischemic posterior wall segment revealed a marked anterior displacement during late systole, motion that was more prominent than in the normal posterior wall. Furthermore, according to the frame-by-frame analysis of LV angiograms by Gibson et al,14 the ischemic segment revealed abnormal inward movement during the isometric relaxation period, while symmetrical outward motion occurred during this period in normal subjects. Conceivably, it was suggested that this abnormality could not reflect the normal motion of the heart as a whole, but specifically the local event in the ischemic segment. Thus, the delayed posterior wall peak as expressed by the increase in the Ac-peak interval and R-peak/ET ratio on echocardiogram seems to be closely related to the abnormal motion pattern of the posterior wall during the isometric relaxation period described in both animal and human studies.

It is of interest that one of the group 2 (hypokinesis) patients with the abnormal R-peak/ET ratio was found to have a severely scarred posterior wall with complete akinesis at the time of coronary bypass surgery. One patient who was classified in the group of posterior wall aneurysm by angiography, but with a normal R-peak/ET ratio (1.24) was found to have an enormous antero-apical-inferior LV aneurysm, but with well preserved posterior wall contractility at the time of aneurysmectomy. Thus, echocardiographic evidence of delayed posterior wall peak, indicated by the increased R-peak/ET ratio, appears to be a sensitive index in the detection of posterior wall aneurysm and may possibly be associated with the existence of noncontracting scar tissue that moves passively throughout the cardiac cycle.

From the present study, the specificity of these indices remains to be demonstrated. A variety of heart disorders, including ventricular excitation abnormalities,12 prolapsed mitral valve,15,16 or cardiomyopathy17 which may affect the segmental contraction pattern of the posterior wall, could also show the delayed posterior wall peak. Therefore, echocardiographic parameters provided herein (Ac-peak or R-peak/ET) may not be entirely specific for posterior wall aneurysm and further study will be required. However, these indices will be quite useful clinically to predict posterior wall aneurysm in patients with known posterior and/or inferior wall infarction.

Another important aspect in this study was the limited value of the PWE in assessing posterior wall abnormality in the presence of the delayed posterior wall peak, since abnormal anterior displacement of the posterior wall during the isometric relaxation period would be superimposed upon the inherent posterior wall motion during the ejection period. This could account for the normal PWE in some patients with posterior wall aneurysm or hypokinesis and with the delayed posterior wall peak.

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