Left ventricular function in presence of small pericardial effusion

Echocardiographic study

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SUMMARY Using echocardiography, various indices of left ventricular function were studied in a group of 11 patients with small posterior pericardial effusion. The only index of left ventricular function that was abnormal in this group was maximum diastolic endocardial velocity (DEV), a measure of left ventricular relaxation. When evaluated as a group, DEV was significantly decreased in the presence of effusion as compared with the control group. This change in DEV in the presence of small effusion, without any other associated changes in left ventricular function, suggests that the mechanism of impaired left ventricular relaxation is a change in compliance of the pericardium. In large effusions, left ventricular relaxation abnormalities are associated with changes in left ventricular dimensions, and impaired relaxation may also be the result of shift in intracardiac structures.

Reflected ultrasound has become the primary diagnostic tool for pericardial effusion since the pioneering work of Edler¹ and Feigenbaum.² Many studies have evaluated its sensitivity and specificity in the diagnosis of pericardial effusion.²⁻⁴ Others have used echocardiograms to quantify the size of effusions.⁵ Recent reports have used various echocardiographic measurements of the mitral valve and left ventricle to explain some of the physical signs associated with pericardial effusion and cardiac tamponade.⁶⁻⁸

There have been few studies of left ventricular function in pericardial effusion. The reason may be that with large pericardial effusion, the whole heart oscillates within the pericardial sac, resulting in an exaggerated motion of the posterior wall. Conventional measurements of left ventricular function are, therefore, not applicable.⁹⁻¹¹ Because of this, we decided to study various indices of left ventricular function in a group of patients with small-to-moderate posterior pericardial effusion with no anterior effusion. The purpose of this study was to determine which of these indices is impaired in a small pericardial effusion and to consider the mechanism of this impairment.

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Methods

Echocardiograms of patients with posterior pericardial effusions were selected from the files of the paediatric heart station of the Johns Hopkins Hospital. Patients with an anterior component to the effusion were excluded in order to minimise swinging of the heart. Each patient also had a control echocardiogram taken when no effusion was present. Thus, the indices measured in the presence of an effusion for each patient could be compared with his own control.

Eleven patients satisfied these criteria and ranged in age from 4 to 17 years, with a mean of 10 years. The causes of pericardial effusion were varied and included uraemia, neoplasm, collagen vascular disease, and post-pericardiotomy syndrome. Echocardiography was performed on a Smith Kline Ekosector I interfaced with a Honeywell Stripchart Visicorder. The ultrasound beam had a frequency of 3.5 or 2.25 MHz, with the former non-focused and the latter focused at 7.5 cm.

The indices measured on each echocardiogram were internal left ventricular systolic and diastolic diameter, maximum diastolic endocardial velocity (DEV), maximum systolic endocardial velocity

(SEVM), anterior mitral leaflet excursion, and EF slope of anterior leaflet of mitral valve. Maximum diastolic endocardial velocity and systolic endocardial velocity were measured by drawing a tangent along the maximum systolic and diastolic motion of the posterior wall of the left ventricle. An example of such a measurement is shown in Fig. 1. Late diastolic endocardial excursion (LDEE) was determined as in Voelkel *et al.*¹² Measurements were made from the crystal artefact to the endocardium at the end of the rapid diastolic relaxation phase and at the beginning of systolic isotonic contraction in the next cardiac cycle; the difference between these two is the LDEE. Indices were measured by two independent observers; there was excellent concordance.

Results

The mean size of the posterior pericardial effusion for the selected patients was 1.5 cm, with a range of 0.8 to 4.1 cm. A sample for one patient is illustrated in Fig. 2. All measurements are reported as mean $\pm$ standard error. The results of the various indices are given in the Table.

(1) MAXIMUM DIASTOLIC ENDOCARDIAL VELOCITY (DEVm)

The DEVm for all 11 patients was significantly less in the presence of posterior pericardial effusion than their respective control values ($p < 0.001$). When taken as a group, the average DEVm with effusion (102 ± 11 mm/s) was less than the average

control value (145 ± 12 mm/s) ($p < 0.02$) (Fig. 3).

(2) MAXIMUM SYSTOLIC ENDOCARDIAL VELOCITY (SEVM)

There was no significant correlation between the presence of pericardial effusion and the SEVM ($p > 0.5$). The SEVM with effusion was 61 ± 4 mm/s, and without effusion it was 60 ± 4 mm/s (Fig. 4).

(3) LATE DIASTOLIC ENDOCARDIAL EXCURSION

There was no correlation between the LDEE and the presence (1.8 ± 0.5 mm) or absence of effusion (1.9 ± 0.4 mm) ($p > 0.5$).

(4) LEFT VENTRICULAR DIMENSIONS

There was no significant correlation between the presence of small posterior pericardial effusion and left ventricular systolic or diastolic diameters ($p > 0.5$). Only two of the 11 patients had significant decreases in both dimensions. Of these two, one had the largest effusion in this study (4.1 cm posterior). When this patient's effusion resolved, his systolic diameter increased from 4.2 to 5.4 cm and the diastolic diameter increased from 5.3 to 6.2 cm. The other patient had the second largest effusion (2.4 cm posterior) and had an increase in systolic and diastolic diameters when the effusion resolved. Systolic diameters increased from 2.1 to 3.5 cm and diastolic 3.4 to 3.8 cm, respectively.

(5) MITRAL VALVE INDICES

There was no significant difference in the EF slope

Fig. 1 Normal echocardiogram. LVS, left ventricular systolic internal diameter; LVD, left ventricular diastolic internal diameter; Peri, pericardium; Endo, endocardium; DEVm, maximum diastolic endocardial velocity; SEVM, maximum systolic endocardial velocity; IVS, interventricular septum.

of the anterior leaflet of the mitral valve in the presence of effusion (133 ± 12 mm/s) compared with controls (135 ± 14 mm/s) ($p > 0.5$). Similarly, no significant difference in mitral valve excursion was observed (21 ± 1 mm both with and without effusion ($p > 0.5$)).

Discussion

The effective ventricular filling pressure is the difference between the intraventricular and extraventricular pressures. Since the heart is invested in the pericardium, pericardial pressure is a major

Fig. 2 Echocardiogram for a patient with and without posterior pericardial effusion. IVS, interventricular septum; Endo, endocardium; Epi, epicardium; Peri, pericardium.

determinant of extraventricular pressure.

The pericardial pressure is a function of intra-pericardial volume and the rate at which this volume changes. If the change is acute, pericardial pressure-volume characteristics are non-linear, with the slope increasing as volume increases.¹³ This, by definition, indicates that the pericardial compliance decreases with increasing volume and pressure. Because of the intimate association between the heart and the pericardium, a decrease in pericardial compliance will decrease the effective ventricular compliance.

Under normal circumstances, the pericardium imposes a slight restrictive force on ventricular filling. In the dog heart-lung preparation, when the pericardium is removed, this extraventricular restrictive force is removed, allowing venous pressure to drop without compromising ventricular filling or cardiac output.¹⁴ In addition, hypervolaemic pericardiectomised dogs have a dilated left ventricle.¹⁵

In the dog, if pericardial pressure is increased by injecting saline or air into the pericardium, the effective ventricular filling pressure is decreased and cardiac output decreases.¹³ If the pericardial pressure is increased enough to cause cardiac tamponade, the effective filling pressure is decreased to the point where there is a loss of early rapid left ventricular diastolic filling and severe restriction of cardiac output.

When cardiac tamponade occurs in humans, there are certain characteristic echocardiographic changes. Cyclic respiratory variations in mitral valve EF slope^{6, 8, 16} and septal motion⁷ have been suggested as explanations of pulsus paradoxus caused by decreased left ventricular filling. Large pericardial effusions that have not yet progressed to tamponade do not have these features,^{8, 16} though swing of the heart⁹⁻¹¹ and false positive appearances of mitral valve prolapse¹⁶⁻¹⁸ have been noted by many observers.

In this study, we examined patients with effusions too small to cause cardiac tamponade and found a significant decrease in the maximum diastolic endocardial velocity when compared with controls. Because we restricted our patient population to those with no anterior component to their effusion, cardiac swing is minimised. This implies that the decreased DEVM is not merely a reflection of motion of the heart as a whole, but instead represents a slowed ventricular relaxation in early diastole. Swing of the heart observed in large effusions tends to accentuate indices of posterior wall motion. This was observed by Vignola *et al.*,¹⁶ who measured an increase in the diastolic epicardial velocity in the presence of moderate to large

Table Echocardiographic data

Case no.	Size of post. effusion (cm)	Diameter		SEVM (mm/s)	DEVm (mm/s)	Mitral excursion (cm)	LDEE (mm)	EF slope (mm/s)
		LV systolic (cm)	LV diastolic (cm)					
1	1.8	3.0	4.3	69	92	2.2	3.0	186
	0	3.0	4.3	77	175	2.2	3.0	238
2	1.1	2.6	4.0	74	82	—	1.5	—
	0	2.1	3.4	68	160	—	1.0	—
3	1.1	2.1	3.5	47	46	1.8	—	84
	0	2.5	3.3	42	84	1.7	3.0	87
4	1.2	3.0	5.8	79	155	3.1	3.5	156
	0	3.2	4.9	73	168	2.3	3.5	124
5	1.3	3.8	6.0	80	117	2.0	0.0	188
	0	3.8	6.2	77	135	2.1	0.5	175
6	0.8	1.9	2.7	46	84	1.5	2.5	125
	0	2.0	2.8	45	119	1.5	2.5	104
7	2.4	2.1	3.4	65	157	1.5	1.0	123
	0	2.5	3.8	65	202	1.9	1.0	121
8	1.0	2.0	3.0	56	95	1.9	1.5	109
	0	2.3	4.0	53	138	2.1	2.5	126
9	4.1	4.2	5.3	54	107	2.3	0.0	156
	0	5.4	6.2	40	133	2.2	0.0	156
10	0.8	3.7	5.0	50	55	2.2	1.5	87
	0	3.3	4.5	52	89	2.0	2.5	109
11	0.9	3.1	5.0	43	134	2.5	4.0	113
	0	2.6	4.5	65	196	2.6	2.5	106

SEVM, maximum systolic endocardial velocity; DEVm, maximum diastolic endocardial velocity; LDEE, late diastolic endocardial excursion.

effusions. We measured endocardial rather than epicardial velocity, and excluded patients with cardiac swing, so our results cannot be compared with those of Vignola *et al.*¹⁶

DEVm has been used as a measure of ventricular relaxation.^{19, 20} The decrease in DEVm in the

presence of pericardial effusion measured in this study suggests that the diastolic relaxation of the left ventricle is impaired. This impairment is probably the result of an increase in pericardial pressure and a decrease in pericardial compliance. We cannot exclude primary myocardial disease as the cause of the decrease in DEVm. This seems less likely because the echocardiographic variables traditionally employed to evaluate left ventricular function are not impaired. In addition, the increase in DEVm after resolution of the effusion argues against myocardial dysfunction. Our data corroborate the early dog studies of Isaacs *et al.*,¹³ in which increased pericardial fluid decreased pericardial compliance and increased pericardial pressure, resulting in impaired ventricular relaxation. Since the primary functional defect appears to involve diastolic relaxation, it is not surprising that we found no change in the maximum systolic endocardial velocity.

The decrease in DEVm in the presence of effusion implies that the rate of early diastolic relaxation is impaired. However, a measure of the absolute amount of relaxation, the late diastolic endocardial excursion, is not affected by small effusions. If one thinks of the heart as being inside a balloon, the compliance of the balloon, representing the pericardium, will decrease as fluid is added. Though this will slow the rate of ventricular filling, the final ventricular volume and the LDEE will be the same. Our finding that left ventricular dimensions are not affected by small effusions supports

Fig. 3 Maximum diastolic endocardial velocity (DEVm) for patients with and without posterior pericardial effusions. ●, data points; ○, mean $\pm$ SE.

this. Voelkel *et al.*¹² demonstrated a decrease in LDEE in patients with constrictive pericarditis. Here, the pericardium acts as a fixed barrier to late diastolic filling, as if the heart were in a rigid box. The primary defect is impaired posterior wall excursion rather than the rate of early diastolic relaxation.

A decreased EF slope of the anterior leaflet of the mitral valve has been used as a sign of decreased ventricular compliance.²¹ We found no significant decrease in the mitral EF slope or in the mitral excursion in the presence of effusion. However, as noted above, only effusions large enough to cause tamponade cause cyclic changes in mitral valve behaviour related to respiration. It appears, therefore, that DEVM is a more sensitive measure of impaired ventricular relaxation secondary to pericardial effusion.

Left ventricular systolic and diastolic internal diameters have been shown to decrease with increased pericardial pressure.²² However, in the presence of the small effusions observed in this study, we found no significant decrease. This apparent discrepancy is probably quantitative rather than qualitative since the pericardial pressures generated by the small effusions in our patients were probably not high enough to affect ventricular dimensions.

Both large and small pericardial effusions result in impaired left ventricular relaxation, but the mechanism appears to be different. In large effusions there are significant changes in systolic and diastolic dimensions of the left ventricle suggesting that the mechanism of impaired left ventricular relaxation

may be the result of shift in intracardiac structures such as the interventricular septum. In small effusions, as is shown by the present study, DEVM, a measure of left ventricular relaxation, is significantly decreased without any associated changes in other left ventricular function indices. This strongly suggests that in small pericardial effusions, impaired left ventricular relaxation is secondary to decreased pericardial compliance and increased pericardial pressure.

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Fig. 4 Maximum systolic endocardial velocity (SEVM) for patients with and without posterior pericardial effusions. ●, data points.

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