Echocardiographic Features of Constrictive Pericarditis

A. Gene Voelkel, M.D., Daniel A. Pietro, M.D., Edward D. Folland, M.D., Michael L. Fisher, M.D., and Alfred F. Parisi, M.D.

SUMMARY The most characteristic echocardiographic features of 12 patients with constrictive pericarditis were compared with the findings in 10 normal volunteers. Left ventricular posterior wall (LVPW) "flatness" was quantified by measuring the diastolic change in distance from the crystal artifact to the LVPW endocardium. In 11 of 12 patients the net diastolic LVPW endocardial movement was < 1 mm. In 10 normal volunteers LVPW endocardium moved posteriorly in diastole from 1.5 to 4 mm (mean 2.2 ± 0.8).

Abnormal septal motion was present in five of 12 patients with constriction. Pericardial thickness measured using standard damping techniques for both constriction and normal populations did not distinguish the two groups. The "flattening" of the left ventricular endocardium as quantified above verifies earlier qualitative observations and was the most consistent finding in this series of patients with constrictive pericarditis.

THE RECOGNITION OF constrictive pericarditis is clinically important, but often difficult. Radiographic evidence may provide a clue, but is not diagnostic and may be absent. Noninvasive tests are of limited value in identifying the impaired ventricular filling which characterizes this disorder. While echocardiography is very useful in identifying pericardial fluid, specific diagnostic criteria for identifying constriction are not widely accepted.

Feigenbaum1 and others2,3 reported a "flat" motion of the posterior left ventricular wall during diastole corresponding to the abrupt transition of rapid ventricular filling to diastasis in patients with constrictive pericarditis.2,3 To date, this observation has not been quantified and applied to a series of patients with constrictive pericarditis. In this paper we report the echocardiographic features of 12 patients with pericardial constriction. We describe a method of quantifying the relative "flatness" of the posterior left ventricular wall endocardium and document the validity of this early observation.

Methods

The records of all patients from our institutions with a diagnosis of constrictive pericarditis who had an echocardiogram were reviewed. The diagnosis was established in 11 of the 12 patients by right heart catheterization. Surgical or autopsy findings were confirmatory in eight patients. Surgery was offered to two additional patients, but refused. In the remaining two patients, we felt that the clinical syndrome could be controlled with medical therapy.

Hemodynamic data used to establish the diagnosis accorded with the early observations by Sawyer et al.4 and the later criteria described by Shabetai.5 All patients manifested the early diastolic dip and late high plateau in the right ventricular pressure recording and equilibrium of pulmonary arterial diastolic pressure, mean pulmonary arterial wedge pressure, mean right atrial pressure, right ventricular diastolic pressure, and, if available, the left ventricular diastolic pressure (table 1). Right-sided pressures were recorded on pullback and simultaneous right and left ventricular pressures were recorded in the seven patients who underwent left heart catheterization. The 12th patient, FM, refused catheterization. He had dyspnea on exertion and the associated signs of jugular venous distension, ascites, liver engorgement, dependent edema, and a pericardial knock on auscultation. His chest x-ray had an "eggshell" calcification pattern around the heart.

Echocardiographic strip chart recordings were made on either a Kent-Cambridge multichannel...
TABLE 1. Clinical, Hemodynamic and Echocardiographic Features of Study Population

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<th>Pt</th>
<th>Age</th>
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* Positive TBC skin test.

Abbreviations: Cau = caucasian; Bl = black; Id = idiopathic; RA = right atrial mean pressure; RVEDP = right ventricular end-diastolic pressure; PAD = pulmonary artery diastolic pressure; PCW = pulmonary capillary wedge mean pressure; HR = heart rate; NSR = normal sinus rhythm; NA = not available; Junct. = junctional rhythm; AF = atrial fibrillation; NL = normal; HYPO = hypokinetic. See text for definitions of Δb–c(A–RVE), Δb–d(A–LVE), Δb–e(A–LVE).

Recorder interfaced with a Smith Kline Ekoline 20A Echograph or an Irex Cardiac Ultrasound Module 150–149 interfaced with the Irex Continutrace 101 Recorder. An Aerotech 1.6 or 2.25 MHz uncollimated ultrasound transducer was used with the patient either flat or in the left lateral decubitus position. The mitral valve and left ventricle were examined using standard techniques. All observations of the movement of the interventricular septum were made at or just below the tips of the mitral leaflets.

A close examination of the features of each record was made as follows (fig. 1):

1) Diastolic points b, c, and d were identified on the left ventricular posterior wall endocardium (LVE). Point b coincided with the most abrupt change in LVE motion during early diastole. Point c was defined on the LVE immediately before the a wave for patients in sinus rhythm with discernible a waves. Point d occurred at the maximum depth of the a wave. For those patients in atrial fibrillation or without significant a waves, point c was defined as the LVE point at the onset of the electrocardiographic QRS complex. Analogous points were likewise identified on the right ventricular anterior wall endocardium (RVE).

![Figure 1](http://circ.ahajournals.org/)

**Figure 1.** Schematic echocardiograms demonstrating points used on left ventricular posterior endocardial surface for measurement. RV = right ventricle; LVE = left ventricle; IVS = interventricular septum; LVPW = left ventricular posterior wall; RVE = right ventricular endocardium; LVE = left ventricular endocardium. See text for definitions of b, c, d, (A–LVE) and (A–RVE).
To quantify the diastolic “flatness” of both LVE and RVE, measurements were made from the crystal artifact to the endocardia from three to five consecutive cycles at the predetermined points b, c and d. In cases where respiration appeared to affect recordings, the patient was asked to halt breathing in mid-expiration. In this manner, the changes in distance from the crystal artifact (A) to LVE or RVE during intervals b-c and b-d were measured to the nearest millimeter and averaged. A record was considered to be “positive for flatness” if the average change in diastolic distances (A-LVE) and (A-RVE) remained ≤ 1 mm from points b to c (∆b-c) or b to d (∆b-d).

2) Pericardial thickness was quantified by measuring the width of the pericardial reflection at a damping level just sufficient to obliterate all remaining cardiac structures.

3) Assessment was also made of the systolic anterior and posterior movement of the interventricular septum as described in other recent reports. Normal septal motion consisted of > 3 mm posterior systolic movement; hypokinetic motion consisted of 0-3 mm posterior systolic movement and paradoxical motion was judged to be present when the septum moved anteriorly in systole. The above measurements were also made from records of 10 normal volunteers, eight men and two women, ages 25–42 years (mean 30 years).

4) Three patients who had pericardectomy consented to right heart catheterization during the third postoperative week to assess the degree of relief from constriction. Echocardiograms were also performed to study the relative changes in motion.

**Results**

The study population is described in table 1. Ages ranged from 47–66 (mean 56 years); all were men. Etiologies were carcinoma of the lung in one patient, a late sequel to hemopericardium following previous cardiac surgery in another and idiopathic in the remaining 10. Of these 10 patients, three had positive intermediate strength PPD skin tests but none had had clinical tuberculosis. Eight of the 12 patients displayed calcified areas of pericardium when examined by fluoroscopy. Hemodynamic data were consistent with constriction in the 11 patients catheterized. Seven patients were in sinus rhythm, four in atrial fibrillation, and one had a junctional mechanism. Interventricular septal motion was normal in six, hypokinetic in one and paradoxical in five patients. Measurements ∆b-c(A-LVE), ∆b-d(A-LVE) and ∆b-c(A-RVE), shown in table 1, correspond to the points described schematically in figure 1.

Figure 2 illustrates a record from each study group. The record to the right is from a patient who had constriction documented by right heart catheterization and postmortem examination. This record contrasts to the normal study at the left. Both records are taken at the level of the chordae tendineae and illustrate the damping procedure to determine pericardial thickness.

The thickness of the posterior pericardium was measured below the tips of the mitral leaflets with the gain settings adjusted to just define the point of interventricular septum and LVPW disappearance. The range of thickness of the constriction population was 5–13 mm (mean 8.6 mm). Similar techniques yielded a range of 4–11 mm (mean 7.9 mm) for pericardial thickness in a normal population.

The degree of flatness was quantified for both the left ventricular endocardium and the right ventricular endocardium. Figure 3 shows the best separation between the two study populations was provided by ∆b-c(A-LVE), with 11 of 12 patients with constrictive pericarditis being “positive for flatness” in evaluation of the left ventricular endocardium. The mean ± SEM of the two populations for these values were as...
follows: constriction 0.2 ± 0.2 mm; normal 2.2 ± 0.8 mm (P < 0.02). The separation of the two study populations by Δb-c(A-RVE) was less dramatic. The mean of the Δb-c(A-RVE) for constriction was 0.95 ± 0.7 mm and that for normals was 1.55 ± 0.9 mm (P = NS). Due to technical problems in recording anterior structures, only six of 12 patients had acceptable right ventricular landmarks for measurement purposes; four of these six had excursions ≤ 1.0 mm. Eight of 10 records from the normal population had acceptable right ventricular targets suitable for measurement and two of these eight had excursions ≤ 1.0 mm.

The three patients who had consented to repeat right heart catheterization following pericardectomy all had improved right-sided pressure pulses. The right atrial mean pressures fell dramatically (fig. 4) and none had evidence for equalization of diastolic pressures. The echocardiograms also showed continued diastolic expansion after the initial rapid filling phase and none were "positive for flatness" postoperatively. This data is demonstrated graphically by the solid lines in figure 4. These same three patients clearly had paradoxical interventricular septal motion on their postoperative echocardiograms, while none had abnormal septal motion before operation.

Discussion

A spectrum of qualitative, nonspecific characteristics associated with the diagnosis of constrictive pericarditis have been identified by various investigators. The relative thickness of the pericardium has been described as being increased in this disorder. However, pericardial thickness is also a function of gain setting and has been shown to correlate poorly with the degree of thickening found at surgery. In our study pericardial thickness was not useful in separating patients from normals.

Gibson et al. noted that abnormal interventricular septal motion was the principle echocardiographic feature in eight of 10 patients with constriction. All observations of septal motion were made at or below the level of the chordae tendineae. Furthermore, in Gibson’s series one case that was normal preoperatively became abnormal after pericardectomy. However, abnormal septal motion has been noted following cardiopulmonary bypass alone. Although abnormal interventricular septal motion was noted in five of our patients, it is too nonspecific to be of diagnostic value since it is a common finding in patients with ischemic heart disease, conduction defects or right ventricular volume overload. There was no direct evidence of right ventricular overload or bundle branch block in our patients, but one patient with a junctional rhythm did have paradox septal motion. In this instance ischemic heart disease was not completely ruled out; etiology of the abnormal septal motion in the other four patients remains obscure.

Qualitative descriptions of posterior wall flatness have been reported by Feigenbaum. Although Gibson et al. considered the flatness as primarily an epicardial and pericardial phenomenon, endocardial flattening was also seen. They attributed this to decreased ventricular compliance with little ventricular filling occurring after the rapid early diastolic phase. Horowitz
had noted this abrupt change to an almost horizontal line in diastole to be a consistent finding in their patients with constrictive pericarditis. We have extended these observations by quantifying left ventricular posterior wall endocardial flattening as ≤1 mm change in distance from crystal artifact to endocardium during diastolic filling. This finding was present in 11 of 12 patients with constriction but in none of 10 normals. Flattening of the right ventricular endocardium also appears more frequently in our constriction population compared with normals, but this difference was not significant in a study of this size.

There appears to be a complex of echocardiographic findings in patients with constrictive pericarditis. Left ventricular endocardial flatness was the most consistent feature of constriction in this series of patients compared with normals. Three patients that were studied postoperatively by right heart catheterization had a definite decrease in filling pressures. These same patients no longer displayed left ventricular wall endocardial flatness on echocardiography. While this criterion of flatness of posterior wall motion has not been studied in other disease states with impaired left ventricular filling such as congestive cardiomyopathy or mitral stenosis, the diagnosis of these conditions is usually readily apparent from other features of their echocardiograms. The differential diagnosis between restrictive myocardial disease and constrictive pericarditis is often difficult even with cardiac catheterization. While a pattern of diastolic posterior wall flatness might be anticipated with infiltrative processes leading to myocardial restriction, this was not a prominent feature in two series reported.\textsuperscript{10, 11}

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References

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