Echocardiographic Features of the Interventricular Septum in Chronic Constrictive Pericarditis

J. Candell-Riera, M.D., H. Garcia del Castillo, M.D., G. Permuyer-Miralda, M.D., and J. Soler-Soler, M.D.

SUMMARY Echocardiographic characteristics of the interventricular septum (IVS) have been studied in eight patients with chronic constrictive pericarditis (CP). Values of septal thickening (ST) were clearly below normal in all cases. Interventricular septal systolic motion (IVSSM) was normal in four cases, hypokinetic in three and paradoxical in one. In seven out of the eight patients, an early interventricular septal diastolic motion (IVSDM) consisting of a sudden anterior displacement followed by a brisk posterior rebound was recorded. The beginning of this anomalous movement was coincident with the pericardial knock in the phonocardiogram and its peak was coincident with the simultaneously recorded deep "y" trough in the jugular pulse tracing. The tendency toward normality of IVSSM observed after pericardiectomy in six out of seven patients suggests that this peculiar interventricular septal systolic motion may be a frequent and probably specific echocardiographic finding in constrictive pericarditis.

Even though the value of echocardiography in the diagnosis of pericardial effusion is well defined, its usefulness in chronic constrictive pericarditis (CP) remains to be established. Several echocardiographic parameters — pericardial thickness,

1, 2, 3 posterior left ventricular wall diastolic motion,

4, 5 early pulmonary valve opening

— have been studied in CP, but no specific sign has been reported. Recently, Poff and colleagues

6 and Gibson and colleagues

7 have pointed out that abnormal systolic interventricular septal motion is the most constant echocardiographic finding in CP; however, this feature has been noted in many other conditions.

The object of the present paper is to analyze the echocardiographic features of the interventricular septum (IVS) in our cases, and to characterize a previously undescribed feature and its possible diagnostic value in CP.

Materials and Methods

Eight patients with chronic CP seen consecutively in our department between March 1976 and February 1977 were studied. Their ages ranged between 14 and 69. The diagnosis (table 1) of CP was established by clinical criteria in all cases; in seven patients it was confirmed at operation and in five by catheterization criteria; five patients had pericardial calcification seen at X-ray; six showed an early diastolic pericardial knock on the phonocardiogram; the apexcardiogram of three showed a "dip plateau" morphology. In every patient, except case 4, a deep "y" trough followed by steep ascent toward an "h" wave was recorded in the indirect jugular pulse tracing. No patient had heart disease other than CP except case 6 who had rheumatic mitral stenosis. All patients had heart rates between 60 and 100/min except case 2 who showed bradycardia of 40 beats/min. Five patients had sinus rhythm; two had atrial fibrillation; and one had atrial flutter with 4:1 A-V block.

Echocardiograms were obtained with a Unirad 100 echocardiographer interfaced to a Honeywell 1858 strip chart recorder on photosensitive paper. A nonfocused 2.25 MHz transducer was placed perpendicular to chest wall on the third or fourth intercostal space along the left sternal border. The free edge of the mitral valve was taken as a reference point: from there the transducer was directed leftward and downward until a recording of the septum and the posterior left ventricular wall was obtained. In this position the following parameters were studied:

1. Septal Thickening (ST). The interventricular septal thickness was measured at end diastole (ED) and end systole (ES). Although maximum anterior movement of posterior left ventricular wall occurs after the end of the second heart sound and probably represents early diastole, ES was arbitrarily considered to coincide with this maximum anterior motion. The ST was calculated by the formula: \( ES - ED \)

Normal values were considered to be \( \geq 0.25 \) mm.

2. Interventricular Septal Systolic Motion (IVSSM). The displacement of the posterior septal wall was measured. It was considered normal when posterior motion was greater than 3 mm. If this degree of motion was not reached, the IVSSM was interpreted as hypokinetic. When the septum moved anteriorly during systole, the motion was considered paradoxical.

3. Interventricular Septal Diastolic Motion (IVSDM). The septal movement characteristics were analyzed during diastole. Diastole was considered from ES onward.

Phonocardiograms and jugular venous pulse tracings were recorded simultaneously with echocardiograms.

In every patient undergoing surgery (except case 7) echocardiograms were recorded 1-12 months after pericardiectomy. The parameters measured before operation were evaluated again.

Results

The values of the various echocardiographic parameters are shown in table 2.

Septal Thickening (ST)

The thickness of IVS at ED varied between 6 and 9 mm (mean 7.7 mm) and at ES between 7 and 11 mm (mean 9
mm). The ST showed values ranging from 0.11 to 0.22 (mean 0.15).

The thickness of IVS at ED after pericardiectomy ranged from 9 to 10 mm (mean 9.4 mm) and end-systolic thickness from 11 to 16 mm (mean 13.6 mm). The ST after pericardiectomy showed values between 0.22 and 0.55 (mean 0.43).

**Interventricular Septal Systolic Motion (IVSSM)**

Movement (IVSSM) was normal in four cases (cases 2, 4, 6 and 8), hypokinetic in three cases (cases 1, 3 and 7) and paradoxical in case 5 (fig. 1A).

After pericardiectomy, IVSSM was normal in six out of the seven patients (cases 1, 2, 3, 4, 6 and 8); it remained paradoxical in case 5, two months after operation (fig. 1B).

**Interventricular Septal Diastolic Motion (IVSDM)**

As shown in figure 1, IVSDM was abnormal in all patients except case 4. In these seven patients an abnormal early diastolic movement of IVS was recorded. This movement consisted of a brisk displacement of the septum, initially anterior and then posterior. It started 0.04-0.12 sec after end systole, and its duration ranged from 0.08 to 0.20 sec (mean 0.12 sec). Its maximal span (measured in the posterior aspect of IVS from the starting point of the movement to its apex) ranged from 3 to 8 mm (mean 4.7 mm). Its beginning was coincident with the pericardial knock (fig. 2) and its peak was coincident with the “y” trough of the jugular venous pulse (fig. 3). Only one of the eight patients had a normal motion of IVS in systole as well as in diastole.

**Table 1. Diagnostic Criteria**

<table>
<thead>
<tr>
<th>Case/Age/Sex</th>
<th>Surg</th>
<th>Cath</th>
<th>Cale</th>
<th>PH</th>
<th>ACG</th>
<th>JVP</th>
<th>ECG</th>
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<tr>
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<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>SR</td>
</tr>
<tr>
<td>2/56/M</td>
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<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>AF</td>
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<tr>
<td>3/32/F</td>
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<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>SR</td>
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<tr>
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<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>SR</td>
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<tr>
<td>5/14/M</td>
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<td></td>
<td>—</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>SR</td>
</tr>
<tr>
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<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>FL</td>
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<tr>
<td>8/69/F</td>
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<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>SR</td>
</tr>
</tbody>
</table>

X indicates presence.

Abbreviations: Surg = surgery; Cath = catheterization; Cale = calcineum; ACG = apexcardiogram; JVP = jugular venous pulse; SR = sinus rhythm; AF = atrial fibrillation; FL = atrial flutter.

**Table 2. Echocardiographic Features of IVS**

<table>
<thead>
<tr>
<th>Case</th>
<th>EDT</th>
<th>EST</th>
<th>ST</th>
<th>IVSSM</th>
<th>ES-Notch</th>
<th>Noteh</th>
<th>Maximal ant span</th>
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<td>H</td>
<td>0.08</td>
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<td>10</td>
<td>0.11</td>
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<tr>
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<td>8</td>
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<tr>
<td>5</td>
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<td>11</td>
<td>0.22</td>
<td>P</td>
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<td>0.12</td>
<td>5</td>
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<tr>
<td>7</td>
<td>7</td>
<td>8</td>
<td>0.14</td>
<td>H</td>
<td>0.04</td>
<td>0.12</td>
<td>5</td>
</tr>
<tr>
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<td>9</td>
<td>11</td>
<td>0.22</td>
<td>N</td>
<td>0.08</td>
<td>0.18</td>
<td>4</td>
</tr>
<tr>
<td>Min</td>
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<td>7</td>
<td>0.11</td>
<td></td>
<td>0.04</td>
<td>0.08</td>
<td>3</td>
</tr>
<tr>
<td>Mean</td>
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<td>9</td>
<td>0.15</td>
<td></td>
<td>0.09</td>
<td>0.12</td>
<td>4.7</td>
</tr>
<tr>
<td>Max</td>
<td>9</td>
<td>11</td>
<td>0.22</td>
<td></td>
<td>0.12</td>
<td>0.20</td>
<td>8</td>
</tr>
</tbody>
</table>

Abbreviations: EDT = end-diastolic thickness; EST = end-systolic thickness; ST = septal thickening; IVSSM = interventricular septal systolic motion; IVSDM = interventricular septal diastolic motion; ES-Notch interval = interval between end systole and beginning of notch; H = hypokinetic; N = normal; P = paradoxial.

**Figure 1.** Diagrammatic illustration of interventricular septum (IVS) and posterior left ventricular wall (PLVW) motion in the eight studied patients. A) preoperative echocardiograms. B) postoperative echocardiograms.
The usefulness of such findings has been observed in patients with chronic constrictive pericarditis. So far, however, none of them has been described which may be considered specific for the disease. The main usefulness of such a sign would be in ruling out restrictive cardiomyopathy, whose findings may be similar to those observed in CP.

The most outstanding echocardiographic sign in our study was the recording of a brisk early diastolic movement of the IVS, consisting of a rapid anterior displacement followed by a rebound toward the posterior left ventricular wall (figs. 1, 4A, 5A). The beginning of this movement was coincident with the pericardial knock (fig. 2) and its apex with the "y" trough of jugular venous pulse (fig. 3). This movement took place immediately after the early diastolic dip in all cases where the latter could be recorded. Early diastolic dip, which may be seen in normal subjects and in mitral stenosis, must not be confused with the abnormal movement described herein, which has an exactly opposite motion sequence.

So far as we know, there is no previous mention of this finding in CP or any other disease. It was registered in a paper by Pool and colleagues 8 in their cases of CP (figs. 1, 2, and 4 of their paper) although they fail to mention its presence. Gibson and colleagues 1 in figure 2 of their paper and Feigenbaum 1 (fig. 11–24 of his book) mention the finding but do not comment upon its value.
In CP the compliance of the ventricles and pericardium as a whole is reduced. Ventricular filling is impaired and ejection volume is low. In early diastole, and during a short period of time, ventricular filling is rapid; however, the limit of ventricular compliance is soon reached and the ventricular pressure curve shows an early diastolic dip followed by a plateau of a greater height than one third of systolic pressure. In the jugular venous pulse recording the "y" trough is also steep, as the counterpart of early diastolic "dip" of ventricular pressure. It is during this phase of diastole when the abnormal septal movement is recorded. We assume that it is related to the above mentioned abnormal ventricular dynamics. To establish its mechanism it would be necessary to record the echocardiogram simultaneously with pressure curves of both ventricles. The disappearance of the abnormal motion after a satisfactory pericardiectomy suggests that it is related to the sudden limitation to ventricular filling exerted by the stiff pericardium.

In our series, instances of normal, hypokinetic, and paradoxical IVSSM were recorded. These results are coincident with those of Gibson and colleagues and differ from the findings of Pool and colleagues, who found paradoxical IVSSM in all of their five patients. However, the above authors agree that systolic motion characteristics lack specificity.

Septal thickening has not been well documented in CP except by Gibson and colleagues who found values clearly below normal as in the present series. On the contrary, Popp considers that this parameter is within normal range in CP. In other conditions, such as cardiomyopathies and coronary artery disease, low ST values have been reported.

The present study suggests that the particular IVSDM described herein is a fairly consistent and possibly specific echocardiographic finding in CP. It is worthwhile to emphasize that this sign was absent in our two cases of restrictive cardiomyopathy (surgery and necropsy proven) and in the cases of the latter condition reported by other authors. If the specificity of our findings is further documented, it may prove to be a very useful sign in the usually equivocal diagnosis between restrictive cardiomyopathy and noncalcific chronic constrictive pericarditis.

References
Short-Term Intramuscular Therapy with Procaine Penicillin Plus Streptomycin for Infective Endocarditis due to Viridans Streptococci

WALTER R. WILSON, M.D., JOSEPH E. GERACI, M.D., CONRAD J. WILKOWSKE, M.D., AND JOHN A. WASHINGTON II, M.D.

SUMMARY Thirty-three patients with viridans streptococcal infective endocarditis were treated for two weeks with intramuscular procaine penicillin, 1.2 million units every 6 hours, plus streptomycin, 500 mg intramuscularly every 12 hours. Nine patients (27%) had infections with relatively penicillin-resistant microorganisms (MIC > 0.1 µg/ml or MBC ≥ 3.12 µg/ml). Follow-up ranged from 2 months to 3.5 years. There were no relapses. Mild vestibular toxicity developed in one patient. One patient died two months after completion of antimicrobial therapy from sudden onset of severe congestive heart failure. Seven patients required cardiac valve replacement after completion of antimicrobial therapy. None died. We believe that this therapeutic regimen is effective antimicrobial therapy for infective endocarditis caused by viridans streptococci, irrespective of in vitro microbiologic data.

STREPTOCOCCI OF THE VIRIDANS GROUP are reported to be the most common bacterial etiologic agents of infective endocarditis, constituting about 30 to 40% of cases.1,2 Penicillin alone or in combination with streptomycin is the preferred antimicrobial therapy for these patients.3-11 The use of short-term combined penicillin-streptomycin therapy for infective endocarditis caused by penicillin-sensitive streptococci was first suggested more than 25 years ago,5-7,12 but it has not gained wide acceptance.

In a prospective study, we treated 33 patients who had infective endocarditis caused by viridans streptococci for two weeks with intramuscular procaine penicillin plus streptomycin. Our experience with these patients is the subject of this report.

Material and Methods

Only patients with infective endocarditis caused by streptococci of the viridans group were enrolled in our prospective study. Our criteria for the diagnosis of infective endocarditis were (1) at least two positive blood cultures with viridans streptococci on at least two separate days and (2) at least two of the following clinical signs — fever, development of a new regurgitant murmur, newly developed splenomegaly, or peripheral embolic phenomenon.

Congestive heart failure was categorized according to criteria defined by the New York Heart Association.13 Antimicrobial susceptibility tests were done by methods described elsewhere.14 Minimum inhibitory concentrations (MIC) and minimum bactericidal concentrations (MBC) were determined by serial dilution in broth in concentrations ranging from 100 to 0.09 µg/ml, and the results were expressed as the lowest concentration of antibiotic that killed at least 99.9% of the initial inoculum. Serum bactericidal tests (SBT) were determined during the third day of treatment on a serum sample taken one hour after intramuscular injection of procaine penicillin and streptomycin; the results were expressed as the greatest dilution of serum that killed at least 99.9% of the inoculum.

Relative penicillin resistance of viridans streptococci was defined as MIC > 0.1 µg/ml or MBC ≥ 3.12 µg/ml, as measured by broth dilution. Antimicrobial therapy in each patient was procaine penicillin G, 1.2 million units every 6 hours, and streptomycin, 500 mg every 12 hours, given intramuscularly for 14 days. The 12-hourly injections of procaine penicillin and streptomycin were administered simultaneously.
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J Candell-Riera, H García del Castillo, G Permanyer-Miralda and J Soler-Soler

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