Hemodynamic Effects of Catecholamine Stimulation in Constrictive Pericarditis

By Fred K. Nakhjavan, M.D., and Harry Goldberg, M.D.

SUMMARY

Cardiac hemodynamic studies during control resting state and isoproterenol infusion were performed on five patients with constrictive pericarditis. During isoproterenol infusion, stroke volume increased by 60%, while there was no significant change in pulmonary wedge and right atrial mean pressures. The relatively fixed filling pressure is the indirect evidence of fixed diastolic size of the heart. However, the fixed cardiac size in diastole per se does not necessarily indicate hindrance to filling of the heart unless the limits of diastolic size of the heart are reached. It is concluded that during the supine resting state, when the influence of catecholamines is minimal, systolic emptying of ventricles is hindered. The systolic hindrance is, however, overcome by strong positive inotropic stimulation.

Additional Indexing Words:
Systolic hindrance  Diastolic hindrance  Isoproterenol

It is generally agreed that in constrictive pericarditis there is “hindrance of ventricular filling.” Whether or not there is also systolic impediment to ventricular emptying is debatable. Sawyer and associates in 1952, in their report on constrictive pericarditis, raised the possibility “that the thick pericardial scar itself may interfere with ventricular contraction.” Lindell and associates in 1963 reported an increase in the stroke volume in patients with constrictive pericarditis during intravenous histamine infusion. They suggested that “the fact that ventricular output may be increased by pharmacological agents indicates the importance of factors that hinder ventricular emptying.” Zimmerman also has stated that in constrictive pericarditis there is impairment of ventricular ejection.

The hemodynamic characteristics of constrictive pericarditis are well described, but the effects of positive inotropic stimuli on such hearts have not been thoroughly investigated. To study the emptying ability of the heart in constrictive pericarditis, a strong inotropic agent, such as isoproterenol, seems to be ideal, especially since the diastolic size of the heart is fixed. It is the purpose of this communication to report the hemodynamic influence of isoproterenol infusion on five patients with constrictive pericarditis who were subjected to cardiac catheterization studies.

Methods

Five patients formed the material of this study. All the patients had had symptoms referable to constrictive pericarditis for several years. Complete history and physical examination, chest roentgenogram, a 12-lead electrocardiogram, carotid tracing, and phonocardiogram were obtained on all. Diagnosis of constrictive pericarditis was made on all the patients and confirmed by cardiac catheterization. Three patients were operated on subsequently (cases 1, 2, and 3). Patient 4 has not consented to operation. Patient 5 has a severe coagulation defect and protein-losing gastroenteropathy and is being prepared for operation. All the patients except one (case 1) had calcification of the pericardium. Right heart catheterization was performed in the postabsorptive state after premedication with 50 mg each of pentobarbital sodium and diphenhydramine hydrochloride. Cardiac output was determined by direct Fick...
principle. Pressures were measured by Statham transducers, model 23 Db. Mean pressures were measured by electrical integration. The zero reference level was set at 5 cm below the sternum. Cardiac output and pressures were measured during control state and were repeated during an intravenous infusion of isoproterenol (1 μg/ml). The rate of isoproterenol infusion was titrated until a satisfactory chronotropic response was steadily maintained. All the recordings were made on an Electronics for Medicine photographic recorder, model DR-8.

Results

The cardiac catheterization data are demonstrated in table 1.

Cardiac Index

Isoproterenol infusion caused an increase in cardiac output in all the patients. The mean increase was 2.36 L/min/m² or 102%. Systemic arteriovenous oxygen difference diminished in four patients and did not change in one (case 4). In this patient the increase in cardiac output was mainly due to an increase in oxygen uptake.

Stroke Index

Of particular interest was the increase in stroke index which occurred in every patient during isoproterenol infusion. The mean increase was 15 ml/beat/m² or 60%.

Pulmonary Arterial and Wedge Pressures

During the control state, pulmonary wedge pressure was elevated in each case and revealed the characteristic “W” or “M” configuration. After isoproterenol infusion, there was no significant change in wedge pressure. The change in pulmonary arterial pressure was also insignificant.

Right Ventricular Pressure

During control state, right ventricular end-diastolic pressure was elevated in all the patients and the tracing revealed a typical “dip and plateau” configuration. During isoproterenol infusion, right ventricular pressure was obtained in three patients (cases 3, 4, and 5) and did not reveal a significant change.

Right Atrial Pressure

The tracing revealed a typical M or W configuration. The right atrial mean pressure in the control state was elevated in all the patients. In the four patients, whose right atrial pressure was recorded during isoproterenol infusion, no significant change occurred.

Table 1

<table>
<thead>
<tr>
<th>Case</th>
<th>State</th>
<th>Age (yr) &amp; sex</th>
<th>BSA (m²)</th>
<th>HR &amp; rhythm</th>
<th>Oxygen consumption (cm³/min/m²)</th>
<th>CI (L/min/m²)</th>
<th>SI (ml/beat/m²)</th>
<th>A-V O₂ diff. (vol. %)</th>
<th>Art. O₂ sat. (%)</th>
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<tbody>
<tr>
<td>1</td>
<td>C</td>
<td>43 M</td>
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<td>22</td>
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<td>I</td>
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<td></td>
<td>135 NSR</td>
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<td></td>
<td>100 AF</td>
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</tbody>
</table>

Change

Mean

35

33

2.36

15

-1.6

1.0

% 44

28

102

60

-33

0.8

Abbreviations: AF = atrial fibrillation; BA = brachial artery; BSA = body surface area; C = control; CI = cardiac index; D = diastolic; I = isoproterenol; M = mean; NSR = normal sinus rhythm; PA = pulmonary artery; PAR = pulmonary arteriolar resistance; RA = right atrium; RV = right ventricle; S = systolic; SI = stroke index; TPVR = total pulmonary vascular resistance; TSVR = total systemic vascular resistance.
CONSTRUCTIVE PERICARDITIS

<table>
<thead>
<tr>
<th>Pressures (mm Hg)</th>
<th>Vascular resistance (dyne sec cm⁻²)</th>
</tr>
</thead>
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<td>Wedge (M)</td>
<td>PA (M)</td>
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<tr>
<td>-2.3</td>
<td>4.8</td>
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</table>

Brachial Arterial Pressure

Isoproterenol caused a moderate rise in brachial arterial systolic, diastolic, and mean pressures in cases 1, 4, and 5.

Vascular Resistances

During isoproterenol infusion there was diminution in the total pulmonary vascular resistance in all the patients with a mean reduction of 144 dynes sec cm⁻⁵ (−43%). Pulmonary arteriolar resistance decreased in three patients (cases 1, 2, and 3) and showed no significant change in cases 4 and 5. Total systemic vascular resistance decreased in all the patients during isoproterenol infusion with a mean reduction of 564 dynes sec cm⁻⁵ (−41%).

Discussion

The two interesting findings in the present study are: (1) the relatively fixed end-diastolic pressure, and (2) the increase in stroke volume during isoproterenol infusion.

In constrictive pericarditis, the diastolic size of the heart is essentially fixed because of restriction by a tense fibrous or calcified pericardium. The fixed diastolic size of the heart deprives the heart of volume changes and hence the Frank-Starling mechanism. In the present study, the end-diastolic volume of the heart was not measured; however, we have assumed that the changes in end-diastolic pressure "in a low compliant chamber" are indicative of changes in volume. Is the above assumption correct? Isaacs and associates⁷ have demonstrated that in constrictive pericarditis, because of decreased compliance, the pressure-volume curve of the heart is essentially steep. It is a fact that in the steep portion of any pressure-volume curve, small changes in volume are reflected fairly accurately in changes in pressure. Hence, in the present study the relatively fixed pulmonary wedge pressure and right atrial pressure during control state and isoproterenol infusion are indicative of a fixed diastolic volume of the heart.

The relatively fixed end-diastolic pressure of the ventricles in constrictive pericarditis is in marked contrast to the effect of isoproterenol in normal and diseased hearts where end-diastolic pressure and volume diminish.⁸,⁹ Gould and associates⁹ have demonstrated that in nonobstructive primary myocardial disease, isoproterenol increased the stroke index (+32%) and decreased the left ventricular end-diastolic pressure (−41%). In the present study, on the other hand, the filling pressure of the heart was not significantly altered after isoproterenol infusion, while stroke index increased 60%. The increase in stroke volume

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in the present study is similar to the finding of Lindell and associates during intravenous histamine infusion and is due to better emptying of the heart.

In constrictive pericarditis, after a more complete systolic emptying, the heart "must" return to its fixed diastolic size. In this regard, there is no hindrance to filling until the limits of diastolic size of the heart are reached.

In this study, cardiac output and stroke volume at rest and in supine position were diminished or were at lower limits of normal in all the patients. The most likely explanation is that at rest in the supine position where the influence of catecholamine stimulation is minimal, and the "systolic emptying of the heart is hindered." However, during inotropic stimulation, the systolic hindrance is overcome by a more forceful contraction and hence by better emptying of the heart and an increase in the stroke volume. This effect of catecholamine stimulation may explain the observed increase in the stroke volume during supine exercise in patients with constrictive pericarditis.  

Two extremes of the disease must be considered in this pathophysiologic picture. First is when the constriction is so mild that the diastolic size of the heart may change to some extent. The other situation is when the myocardium is so severely atrophied or so extensively involved in the fibrocalcific process of the pericardium that it is not capable of responding to inotropic stimuli. The cases reported in the present communication do not seem to belong to either of the above two categories.

References
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