Restoration of Normal Intracardiac Pressures after Extensive Pericardiectomy for Constrictive Pericarditis

By D. P. Fitzpatrick, M.D., E. M. Wyso, M.D., L. H. Bosher, M.D., and D. W. Richardson, M.D.

CHRONIC constrictive pericarditis produces a characteristic clinical picture that results from a characteristic circulatory alteration, namely, restriction of ventricular filling. Pericardial constriction is accompanied by typical abnormalities of right atrial and right ventricular pressure-pulse contours, and although the majority of patients are improved clinically after pericardiectomy, some authors have indicated that the abnormal pressures persist postoperatively. The observation of normal right ventricular and atrial pressure tracings in four patients following pericardiectomy has led us to review cases subjected to operation in two general hospitals. This paper presents the clinical features and preoperative and postoperative catheterization findings in patients with chronic constrictive pericarditis who have undergone pericardial resection since December 1955, under the supervision of one surgeon (LHB).

Material and Methods

Nine male and two female patients, ranging in age from 4 to 58 years, were studied. The two women and four of the males, including the 4-year-old, were Negroes.

Tuberculous etiology was proved in three patients by culture of acid-fast bacilli from the pericardium or pericardial fluid, and was suspected in a fourth patient on the basis of histologic evidence, namely, caseous necrosis and Langhans' giant cells. One patient, L. H., without any history of chest pain or trauma, showed extravasated blood and hemosiderin-filled macrophages between layers of fibrous tissue in the pericardium and was thought to have an organized hemopericardium. Etiology was unknown in the remaining patients. Dense pericardial fibrosis with minimal inflammation was found in each, and extensive calcification accompanied the fibrosis in four. Identifying clinical data on the 11 subjects are presented in table 1.

Six of the 11 patients were studied with preoperative and postoperative cardiac catheterization; two underwent cardiac catheterization postoperatively only, and three preoperatively only. Catheterization of the right side of the heart was carried out by usual methods. Pressures were recorded with Statham P23A strain gages and an Electronics for Medicine cathode-ray oscillographic recorder. Cardiac output was measured by the direct Fick or the dye-dilution methods, or both, described in detail previously.

Results

Catheterization Findings

Data presented in table 2 demonstrate that normal right heart, pulmonary artery, and "pulmonary capillary" pressures occurred eventually in seven of the eight patients studied postoperatively, whereas all patients catheterized prior to operation had shown elevated right atrial pressures with an "M" or "W" shaped contour, as well as elevated right ventricular end-diastolic pressures with a typical early diastolic "dip." Subject E. M. showed abnormal catheterization findings 2 weeks after surgery, at which time the pressures and contours showed little change from preoperative values. Because of death due to unrelated causes (apparently acute alcoholism) 6 months later, this patient was not catheterized again, but autopsy demonstrated an unrestricted myocardium. Another subject, D. B., had abnormally high ventricular diastolic and atrial pressures with contours typical of constriction at catheterization 2 months postoperatively, but quite normal findings 5 months later. A third patient, J. M., showed borderline normal pressures 7 months postoperatively and perfectly normal values 6 years later. One patient, L. M.,
had normal pressures and contours 6 weeks after operation.

Clinical Results

Table 3 summarizes pertinent preoperative clinical findings in these 11 patients. Dyspnea and elevation of venous pressure were noted in all subjects, and electrocardiograms were also always abnormal. In retrospect, the electrocardiogram was usually typical, nine patients showing low voltage and low or slightly inverted T waves, whereas the other two had T-wave changes only. Most of the patients, however, were known to have abnormal cardiograms for weeks before the diagnosis of pericardial constriction was made, the changes being so commonly observed in various types of heart disease as to be of little diagnostic value.

Hepatomegaly, neck vein distention in the sitting position, narrow pulse pressure, and prolonged circulation time occurred frequently, and edema, orthopnea, ascites, and decreased cardiac pulsation at fluoroscopy occurred in about two thirds of the patients. Atrial fibrillation and heart murmurs were uncommon.

All 11 patients underwent pericardiectomy. Two died in the postoperative period and will be discussed subsequently.

Of the nine surviving patients, seven had complete pericardial decortication. In one (J. M.) of the two remaining patients, both of whom had evidence of active tuberculosis in the pericardium, the scar posterior to the left ventricle was somewhat immature and the plane for decortication poorly defined. In addition, a few bands of scar were left in the right atroventricular groove, along the course of the descending coronary, and over the apex of the left ventricle. Although progress was satisfactory and his clinical condition appeared good, intracardiac pressures determined 7 months postoperatively were not completely normal. At the time of final catheterization 6 years postoperatively, intracardiac pressures could be considered normal. The second patient (M. W.) with incomplete decortication was in a subaute

Table 1

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Race</th>
<th>Duration of illness preop.</th>
<th>Clinical result</th>
<th>Cause of death</th>
<th>Tuberculin skin test</th>
<th>Pericardial pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. J.M.</td>
<td>58</td>
<td>M</td>
<td>W</td>
<td>6 months</td>
<td>Cured</td>
<td>.</td>
<td>Positive</td>
<td>Granulomata, AFB stained</td>
</tr>
<tr>
<td>2. D.B.</td>
<td>59</td>
<td>M</td>
<td>N</td>
<td>1 year</td>
<td>Cured</td>
<td>.</td>
<td>Positive</td>
<td>Caseation</td>
</tr>
<tr>
<td>3. L.M.</td>
<td>39</td>
<td>M</td>
<td>W</td>
<td>4 months</td>
<td>Cured</td>
<td>.</td>
<td>Positive</td>
<td>Fibrosis, calcification</td>
</tr>
<tr>
<td>4. L.S.</td>
<td>63</td>
<td>M</td>
<td>N</td>
<td>3 years</td>
<td>Cured</td>
<td>.</td>
<td>Not done</td>
<td>Fibrosis, calcification</td>
</tr>
<tr>
<td>5. L.J.</td>
<td>36</td>
<td>F</td>
<td>N</td>
<td>5 plus yrs.</td>
<td>Cured</td>
<td>.</td>
<td>Neg.1:1000</td>
<td>Fibrosis, no inflammation</td>
</tr>
<tr>
<td>6. E.M.</td>
<td>44</td>
<td>M</td>
<td>W</td>
<td>1 year</td>
<td>Cured</td>
<td>Alcoholism</td>
<td>Positive</td>
<td>Caseation and AFB stained</td>
</tr>
<tr>
<td>7. H.J.</td>
<td>4</td>
<td>M</td>
<td>N</td>
<td>1 year</td>
<td>Cured</td>
<td>.</td>
<td>Positive</td>
<td>Dense fibrosis, few lymphs</td>
</tr>
<tr>
<td>8. M.W.</td>
<td>29</td>
<td>F</td>
<td>N</td>
<td>10 months</td>
<td>Cured</td>
<td>.</td>
<td>Positive</td>
<td>Caseation, AFB stained</td>
</tr>
<tr>
<td>9. J.H.</td>
<td>59</td>
<td>M</td>
<td>W</td>
<td>3 years</td>
<td>Died</td>
<td>Cardiopulmonary insufficiency</td>
<td>Positive</td>
<td>Calcified heavily</td>
</tr>
<tr>
<td>10. L.H.</td>
<td>41</td>
<td>M</td>
<td>W</td>
<td>5 months</td>
<td>Cured</td>
<td>.</td>
<td>Positive</td>
<td>Organized hemopericardium</td>
</tr>
<tr>
<td>11. J.C.</td>
<td>33</td>
<td>M</td>
<td>N</td>
<td>7 years</td>
<td>Died</td>
<td>Staphylococcal infection</td>
<td>Not done</td>
<td>Dense fibrous tissue with calcification</td>
</tr>
</tbody>
</table>

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Table 2

Constrictive Pericarditis—Cardiac Catheterization Data

<table>
<thead>
<tr>
<th>Patients</th>
<th>Right atrial Contour</th>
<th>Pressures—mm. Hg Right ventricular Systole Early diastolic</th>
<th>Pulmonary artery Syst.</th>
<th>P.A. Wedge Mean</th>
<th>Cardiac output L./Min</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.M. Preop. 18 W</td>
<td>40 10 17</td>
<td>40 18 . . .</td>
<td>. . .</td>
<td>. . .</td>
<td>. . .</td>
</tr>
<tr>
<td>7 mos. Postop. 7 W</td>
<td>30 2 7</td>
<td>30 11 . . .</td>
<td>11 . . .</td>
<td>. . .</td>
<td>. . .</td>
</tr>
<tr>
<td>6 yrs. Postop. 3 Normal</td>
<td>26 0 2</td>
<td>26 8 . . .</td>
<td>8 . . .</td>
<td>. . .</td>
<td>5.4</td>
</tr>
<tr>
<td>D.B. Preop. 17 W</td>
<td>35 10 20</td>
<td>35 17 . . .</td>
<td>17 . . .</td>
<td>. . .</td>
<td>4</td>
</tr>
<tr>
<td>2 mos. Postop. 9 W</td>
<td>28 4 10</td>
<td>34 17 . . .</td>
<td>17 . . .</td>
<td>. . .</td>
<td>5.7</td>
</tr>
<tr>
<td>7 mos. Postop. 4 Normal</td>
<td>26 2 4</td>
<td>26 12 . . .</td>
<td>12 . . .</td>
<td>. . .</td>
<td>6.1</td>
</tr>
<tr>
<td>L.M. Preop. 18 W</td>
<td>34 7 24</td>
<td>27 17 . . .</td>
<td>17 . . .</td>
<td>. . .</td>
<td>5.5</td>
</tr>
<tr>
<td>1½ mos. Postop. 6 Normal</td>
<td>36 4 6</td>
<td>28 16 . . .</td>
<td>16 . . .</td>
<td>. . .</td>
<td>5.5</td>
</tr>
<tr>
<td>L.S. Preop. 20 W</td>
<td>. . .</td>
<td>48 25 . . .</td>
<td>25 . . .</td>
<td>. . .</td>
<td>. . .</td>
</tr>
<tr>
<td>36 mos. Postop. 7 Normal</td>
<td>35 0 7</td>
<td>. . .</td>
<td>7 . . .</td>
<td>. . .</td>
<td>. . .</td>
</tr>
<tr>
<td>L.J. Preop. 11 W</td>
<td>38 0 15</td>
<td>36 15 14* .</td>
<td>14* . . .</td>
<td>. . .</td>
<td>. . .</td>
</tr>
<tr>
<td>48 mos. Postop. 4 Normal</td>
<td>29 0 1</td>
<td>20 8 . . .</td>
<td>8 . . .</td>
<td>. . .</td>
<td>4.7</td>
</tr>
<tr>
<td>E.M. Preop. 17 W</td>
<td>36 8 20</td>
<td>30 19 . . .</td>
<td>19 . . .</td>
<td>. . .</td>
<td>. . .</td>
</tr>
<tr>
<td>2 wks. Postop. 12 W</td>
<td>35 5 17</td>
<td>40 18 . . .</td>
<td>18 . . .</td>
<td>. . .</td>
<td>5.0</td>
</tr>
<tr>
<td>H.J. 12 mos. Postop. 2 Normal</td>
<td>33 2 0</td>
<td>28 10 . . .</td>
<td>10 . . .</td>
<td>. . .</td>
<td>4.8</td>
</tr>
<tr>
<td>M.W. 7 yrs. Postop. 2 Normal</td>
<td>29 2 2</td>
<td>24 10 . . .</td>
<td>10 . . .</td>
<td>. . .</td>
<td>5</td>
</tr>
<tr>
<td>J.H. Preop. 13 W</td>
<td>45 5 20</td>
<td>45 18 . . .</td>
<td>18 . . .</td>
<td>. . .</td>
<td>. . .</td>
</tr>
<tr>
<td>L.H. Preop. 20 M</td>
<td>49 4 20</td>
<td>40 18 . . .</td>
<td>18 . . .</td>
<td>. . .</td>
<td>20</td>
</tr>
<tr>
<td>J.C. Preop. 15 W</td>
<td>31 10 16</td>
<td>30 12 . . .</td>
<td>12 . . .</td>
<td>. . .</td>
<td>. . .</td>
</tr>
</tbody>
</table>

*Left atrial.

stage of the disease with a considerable amount of tuberculous granulation tissue posterior to the left ventricle. The decortication was considered satisfactory except at this site. For the first week the patient improved, but after that she exhibited almost complete symptomatic recurrence. No fluid could be aspirated from the anterior mediastinum. Antituberculous chemotherapy was prescribed for 6 months postoperatively. She continued to accumulate ascites and pleural fluid and exhibited dyspnea and hepatomegaly. Then she began to show slight improvement. Between the sixth and the eleventh months, during which time she was not seen, she made remarkably rapid improvement and appeared almost normal on subsequent examination. Cardiac catheterization 7 years postoperatively showed completely normal intracardiac pressures. These two patients were the first in the series, both had positive evidence of tuberculosis in the pericardium and, inexplicably, neither received digitalis in the postoperative period.

At that time the importance of myocardial failure was insufficiently appreciated.

Two other patients, completely decorticated, exhibited somewhat slow clinical recoveries. Caseous material, which showed Langhan's giant cells on microscopic examination, was present in the pericardium in one, (D. B.). From the other (E. M.) a positive culture had been obtained 9 months previously on pericardial tap. The ultimate result was good in one and probably also in the other, but he died of acute alcoholism 6 months postoperatively before final physiologic evaluation. Postmortem examination showed a satisfactory result without evidence of constriction. Thus all of the surviving cases made excellent clinical recoveries, verified in seven by cardiac catheterization. There has been no evidence of recurrence in any. In the four cases exhibiting a slow postoperative recovery, the demonstration of active tuberculous inflammation at or near the time of surgery suggests the role of active myocarditis. None of these four had
## Table 3

Summary of Pertinent Clinical Findings in Eleven Cases

<table>
<thead>
<tr>
<th>Finding</th>
<th>No. of patients exhibiting finding</th>
<th>Total patients in whom observation was recorded</th>
<th>Cooley et al., 72 Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Dyspnea</td>
<td>11</td>
<td>11</td>
<td>60</td>
</tr>
<tr>
<td>2. Orthopnea and paroxysmal nocturnal dyspnea</td>
<td>11</td>
<td>11</td>
<td>..</td>
</tr>
<tr>
<td>3. Ascites</td>
<td>11</td>
<td>11</td>
<td>67</td>
</tr>
<tr>
<td>4. Ascites</td>
<td>11</td>
<td>11</td>
<td>55</td>
</tr>
<tr>
<td>5. Ascites</td>
<td>11</td>
<td>11</td>
<td>45</td>
</tr>
<tr>
<td>6. Peripheral edema</td>
<td>11</td>
<td>11</td>
<td>36</td>
</tr>
<tr>
<td>7. Pleural effusion</td>
<td>11</td>
<td>11</td>
<td>30</td>
</tr>
<tr>
<td>8. Diastolic third heart sound</td>
<td>11</td>
<td>11</td>
<td>20</td>
</tr>
<tr>
<td>9. Pulse pressure less than 20 mm. Hg</td>
<td>11</td>
<td>11</td>
<td>10</td>
</tr>
<tr>
<td>10. Murmurs</td>
<td>11</td>
<td>11</td>
<td>8</td>
</tr>
<tr>
<td>11. Atrial fibrillation</td>
<td>11</td>
<td>11</td>
<td>6</td>
</tr>
<tr>
<td>12. Patients presenting primarily with</td>
<td>11</td>
<td>11</td>
<td>11</td>
</tr>
<tr>
<td>right-sided heart failure</td>
<td>11</td>
<td>11</td>
<td>4</td>
</tr>
<tr>
<td>13. Patients presenting with right and</td>
<td>11</td>
<td>11</td>
<td>2</td>
</tr>
<tr>
<td>left sided heart failure</td>
<td>11</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>14. Circ. time more than 20 seconds</td>
<td>11</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>15. Venous pressure more than 200 mm. H₂O</td>
<td>11</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>16. ECG-low QRS and/or T-wave changes</td>
<td>11</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>17. Diminished cardiac pulsations on fluoroscopy</td>
<td>11</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>18. Pericardial calcification by x-ray</td>
<td>11</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>19. Tuberculous etiology by histology and cultures</td>
<td>11</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>20. Tuberculin skin test positive</td>
<td>11</td>
<td>11</td>
<td>1</td>
</tr>
</tbody>
</table>

Symptoms longer than 1 year. Acute tuberculous pericarditis could be accurately dated within 1 year before surgery in two of them. The other five surviving patients showed complete remission of symptomatology in the first 2 weeks postoperatively.

Of the two deaths, one patient (J. H.) aged 59, died on the ninth day postoperatively. At operation, the lungs were partially constricted with a thin fibrous peel. An attempt was made to decorticate the left lower lobe, but this was abandoned owing to the difficulties encountered and the consequent air leak. This patient probably died of myocardial insufficiency with pulmonary insufficiency as a contributing factor. He was fully digitalized. In retrospect, a less traumatic thoracotomy approach might have been helpful as well as some ventilatory support. The need for a modified thoracotomy approach was also seen in case 11 (J. C.), a patient with disease of 8 years’ standing who had annually refused surgery until his condition deteriorated badly. Extreme difficulty was encountered in obtaining a satisfactory exposure even with the bilateral, transpleural, transverse sternotomy approach because of a firm pleural symphysis. After elevating the upper thoracic segment sufficiently, considerable blood loss had resulted and a number of air leaks had been produced. Decortication of the pericardium was tedious, the pleural problem extensive. He ultimately died 4 weeks postoperatively from the effects of staphylococcus wound infection, empyema, sternal osteomyelitis, and toxic nephritis.

In these 11 patients electrocardiograms became normal in five, and showed increased QRS voltage in five of the remaining six, all of whom continued to show abnormal T waves. The survivors have continued to be well clinically, and have had normal venous pressures for periods from 3 to 7 years after operation.
Typical right ventricular and right atrial pressure patterns in constrictive pericarditis. Simultaneous atrial and ventricular pressures, recorded at two different paper speeds, in patient E.M. Note coincidence of the “y” in the atrial tracing, with early ventricular diastole. End-diastolic ventricular pressure is about half the systolic value.

Discussion

Postoperative Catheterization Findings

The classic study by Sawyer, Burwell, Dexter, Eppinger, Goodale, Gorlin, Harken and Haynes, demonstrating the abnormal physiology presented by patients with constrictive pericarditis, showed that elevation in right ventricular end-diastolic pressure persisted for months after pericardiectomy, despite marked clinical improvement in six individuals. In our series of eight patients who underwent cardiac catheterization after removal of constricting pericardia, normal right heart pressures were found in seven and the eighth (E. M.) was catheterized only 2 weeks after operation. Two patients (L. M. and J. M.), whose pressures were abnormal or borderline soon after operation, showed normal values subsequently. The patients of Sawyer et al. still showed abnormal pressure contours up to 144 weeks after operation, so that progressive improvement as time passes does not seem an adequate explanation for the normal postoperative findings in our patients, especially since two of our patients presented normal findings 6 and 30 weeks after surgery. The most likely explanation for persistence of abnormal right heart pressures after operation in the series of Sawyer et al. was presented in their paper, and lies in the completeness of the pericardial removal: “In none of these patients was decortication of the ventricles complete. . . .” A more complete pericardiectomy has been carried out on the patients in our series. Extensive pericardiectomy has been emphasized also by Malm, who demonstrated return to normal of right ventricular diastolic pressures in five patients catheterized after very extensive removal of pericardium.

Myocardial atrophy and fibrosis were suggested by Sawyer et al. as a possible explanation for persistence of abnormal hemodynamics after pericardiectomy. Dines et al. have demonstrated definite atrophy of myo-
cardial fibers throughout the heart in 11 cases of constrictive pericarditis studied at autopsy. The high percentage of patients in our series who eventually developed perfectly normal ventricular and atrial pressures suggests that irreversible myocardial abnormality must not be a common occurrence, and that inadequate pericardiectomy is a more likely explanation for persistently abnormal hemodynamics. Abnormal pressures early in the postoperative course in three patients, with subsequent development of normal contours, in two, suggests that myocardial atrophy may be an important factor in the immediate postoperative period.

Pathologic Physiology in Constrictive Pericarditis

The physiologic abnormality that forms the basis of the circulatory derangement in chronic constrictive pericarditis is restriction of ventricular filling by the dense, rigid pericardium surrounding the heart. Consequently, less blood is able to enter the ventricles, and stroke output into the pulmonary artery and aorta decreases. The ventricles are apparently able to eject most of their blood with each systole, since early diastolic pressure is normal. Because the ventricles are restricted by the dense scar tissue, ventricular dilation is restricted and a relatively small volume of blood entering the ventricles during diastole results in rapid elevation of the end-diastolic ventricular pressure to a value about half the systolic pressure. The combination of normal early diastolic pressure and elevated late diastolic pressure in the ventricles is responsible for the "early diastolic dip" described as characteristic of constrictive pericarditis.

As a result of elevation of ventricular end-diastolic pressure, atrial pressure increases as do the peripheral venous and capillary pressures. If the left ventricle is involved by the constriction, as it frequently is, the result is a similar increase in pressures in the left ventricle, left atrium, and the pulmonary veins and capillaries. Hansen has stated that similar pressures and pressure contours are found in the left atrium and left ventricle during left heart catheterization of patients with constrictive pericarditis, and a "W-shaped" left atrial pressure of 14 mm Hg was found in the single left heart catheterization done in our series. Elevated pulmonary capillary "wedge" pressures have frequently been found in right heart catheterization studies of constrictive pericarditis, reflecting the high left heart and pulmonary vein pressures.7,8

Figure 1 shows the simultaneously recorded atrial and ventricular pressures in patient E. M., and demonstrates both the early diastolic "dip" in ventricular pressure and the "M" shaped atrial pressure contour characteristically seen.9

The "M" shaped atrial contours and "early diastolic dip" with elevated late ventricular diastolic pressures are always present in constrictive pericarditis, but are also observed in a variety of diseases affecting the heart which have in common restriction of ventricular filling, such as pericardial effusion,10 subendocardial fibroelastosis,11 amyloidosis,12 hemachromatosis,13 and myocardial fibrosis.14 There are some helpful features in distinguishing pressure contours found in myocardial fibrosis from constrictive pericarditis. In myocardial fibrosis the mean right atrial pressure is usually 15 mm Hg or less, whereas in constrictive pericarditis this is more likely to be over 15 mm Hg.14 Also, in myocardial fibrosis the diastolic pressure is usually less than one third the systolic pressure of the right ventricle, while in constrictive pericarditis end-diastolic pressure is frequently more than one third the right ventricular systolic pressure.7

Surgical Considerations

A successful clinical result following decortication of the constricted heart depends upon the adequacy of the pericardial resection and upon the degree of myocardial inflammation, atrophy, and fibrosis.

In all of the 11 cases reviewed above, a complete decortication has been attempted. By this definition the peel is excised from both ventricles, including the posterior sur-

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face of the left ventricle and the great vessels at the base of the heart; the atrio-
ventricular grooves are liberated and any scar encasing the cavae is removed. If easily
accomplished, the right atrium is decorticated down to the level of the right phrenic nerve.
Often the peel over the superior vena cava is thin and nonconstricting, and decortica-
tion is not required at this site. Clearly the liberation of both ventricles and the corre-
sponding atrioventricular grooves constitutes the most important step of the surgical pro-
cedure in the majority of cases. However, in one patient we observed a significant
drop in the venous pressure (superior caval system) that coincided with the decortica-
tion of the superior vena cava. This may have occurred in a second patient with a
similar scar at this site, but the pressure drop was not convincingly demonstrated
manometrically. In a third patient, the heart dilated as the inferior vena cava was
decorticated. In the future we plan to monitor continuously both the inferior and the
superior vena caval pressures as the heart is decorticated in stepwise fashion beginning
with the left ventricle.

Since Dr. Holman’s paper\textsuperscript{15} in 1949, in which he urged that decortication for con-
strictive pericarditis include the cavae, a number of authors have reemphasized the
primary importance of the ventricular con-
striction. There can be no argument regarding this. The best experimental investigation
bearing on this subject was conducted by Isaacs, Carter, and Haller.\textsuperscript{16} By production
of pericardial constriction in dogs, they con-
clusively demonstrated the separate impor-
tance of right and left ventricular constric-
tion, but were unable to show any role for
right atrial constriction. Their conclusions do
not necessarily infer a negligible role for
caval constriction, although this has been
implied by other authors in reference to
their work. Experimentally, Holman showed in
the dog that gross physiologic alterations
due to inferior caval obstruction occurred
after the diameter of the inferior cavae had
been reduced 50 per cent. When only one cava
was constricted, the development of venous
collateral subsequently ameliorated these
changes. The absence of a demonstrable pres-
sure gradient between cava and atrium during
preoperative catheterization has been fre-
quently cited as evidence against the physio-
logic importance of caval constriction. This
reasoning seems to us invalid. Uniform con-
striction of the right heart and cavae would
not be expected to produce an easily de-
tectable pressure gradient until after the
ventricles had been decorticated, and even
then myocardial insufficiency could lead to
erroneous interpretation. Nevertheless, pres-
sure determinations in both inferior and
superior cava during decortication could
yield interesting information. Marshall and
Pantridge\textsuperscript{17} have claimed to show a distinct
drop in the inferior caval pressure at the
time of decortication of this vessel in four
of five cases. However, convincing evidence
against the importance of caval involvement
is that presented by Johansson.\textsuperscript{18} He cited
25 cases of visualization of the cavae pre-
operatively with angiocardiography without
evidence of stenosis. In most cases the cavae
were actually dilated, as was also the right
atrium.

It has been stated by surgeons who have
employed both a radical and a limited de-
cortication (of the ventricles only) that a
return to a more normal state occurs more
rapidly following a radical decortication, but
that a satisfactory clinical result may be
achieved following a less complete decortica-
tion if a longer period of recovery is accepted.
Scientific appraisal has been difficult owing
to uncertainty regarding the state of the myo-
cardial function in an individual case at
the time of decortication. Dines et al.\textsuperscript{8} have
emphasized the frequent occurrence of atro-
phy of muscle fiber. We have reasoned that
if the ventricles and atrioventricular grooves
are incompletely decorticated, a longer period
of time and more complete recovery from
myocardial atrophy will be necessary for
adequate ventricular function, but that even-
Intracardiac pressures by the partially liberated myocardium may be sufficient. On the other hand, with completely decorticated ventricles, good hemodynamic function is achieved during an earlier state of myocardial recovery.

In order to achieve complete decortication, we have usually employed the transpleural, transsternotomy incision, usually through the fourth interspace on the right and the fifth interspace on the left. The midline sternal splitting incision advocated by Holman does not provide satisfactory exposure for decortication of the posterior surface of the left ventricle, particularly when the heart is large. Holman recognized this, but apparently did not think that decortication was essential. Other recommended left thoracotomy approaches do not provide satisfactory exposure of the right atrioventricular groove or the cavae. In two patients, we used a slightly modified and somewhat less traumatic approach, which seems equally satisfactory as a transsternal, transpleural thoracotomy and allows a complete decortication of both the right and left sides of the heart. A midline vertical sternal splitting incision is employed with a T extension into the fourth or fifth interspace. Appropriate spreaders are needed for adequate retraction of the thoracic flaps. The midline skin incision is arched slightly to the left. The left pleural space is always drained and if the right pleural space is entered, this is intubated as well. A heavy no. 20 wire mattress suture is used to reapproximate the sternal segments at the T. With this approach, problems arising from extensive pleural symphysis are minimized. We do not believe that this is an appropriate time for indulging in a very difficult pulmonary decortication for a peel that has often resembled a fibrous pleuritis. Further evaluation of this approach is needed.

Summary

In each of seven patients studied by cardiac catheterization more than 2 weeks after extensive pericardietomy, performed because of constrictive pericarditis, normal right heart pressure-pulse contours were demonstrated.

The extensiveness of pericardial removal seems the most likely explanation for the normal catheterization findings, which contrast with demonstration by other workers of persistence for periods up to 2 years of the 'W'-shaped atrial pressures and high end-diastolic ventricular pressures characteristic of pericardial constriction.

Clinical findings, abnormal right heart pressures, and surgical considerations are presented for a total of 11 patients with constrictive pericarditis who were subjected to pericardietomy between 1955 and 1960.

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The Early History of Instrumental Precision in Medicine

Thinking over the number of instruments of precision, a single case may require, it is clearly to be seen that no matter how expert we may be, the diagnostic study of an obscure case must today exact an amount of time far beyond that which Sydenham may have found need to employ. A postmortem section used to take us an hour or two, and now, alas! it goes on for weeks in some shape until the last staining is complete, the last section studied, the last analysis made.

These increasing demands upon us are due to the use of instruments of precision, or to accurately precise methods. As in factories more and more exact machines have trained to like exactness a generation of workmen, so with us, the use of instruments of precision, rendering the comparison of individual labor possible, has tended to lift the general level of acuteness of observation. The instrument trains the man; it exacts accuracy and teaches care; it creates a wholesome appetite for precision which, at last, becomes habitual. The microscope, the balance, the thermometer, the chronograph have given birth to new standards in observation, by which we live, scarce conscious of the change a generation has brought about. Certain interesting intellectual results have everywhere followed the generalisation of precision by the use of instruments, like the world-wide lesson in punctuality taught by the railway and made possible by the watch. . . . For unless men keep ahead of their instrumental aids, these, to coin a word, will merely demonetize them, and but measurably lift the mass without in proportion advantaging the masters of our art, who were so easily masters in days when the erudite touch was more uniquely advantageous than it is today.—S. WEIR MITCHELL, M.D., Transactions of the Congress of American Physicians and Surgeons, Second Triennial Session held at Washington, D.C., 1891. New Haven, The Congress, 1892, p. 164.
Restoration of Normal Intracardiac Pressures after Extensive Pericardiectomy for Constrictive Pericarditis

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