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.,

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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 pericardectomy. 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 atrioventricular 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 subacute case of tuberculosis, and the postoperative course was bad.

**Table 3**

<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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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

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**Table 2**

Constrictive Pericarditis—Cardiac Catheterization Data

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

*Left atrial.
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.
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.

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.

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, subendocardial fibroelastosis, amyloidosis, hemachromatosis, and myocardial fibrosis. 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. 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.

**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-
face of the left ventricle and the great vessels at the base of the heart; the atrioventricular 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 noneconstricting, and decortication is not required at this site. Clearly the liberation of both ventricles and the corresponding atrioventricular grooves constitutes the most important step of the surgical procedure in the majority of cases. However, in one patient we observed a significant drop in the venous pressure (superior cava system) that coincided with the decortication 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 in 1949, in which he urged that decortication for constrictive pericarditis include the cavae, a number of authors have reemphasized the primary importance of the ventricular constriction. There can be no argument regarding this. The best experimental investigation bearing on this subject was conducted by Isaacs, Carter, and Haller. By production of pericardial constriction in dogs, they conclusively demonstrated the separate importance of right and left ventricular constriction, 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 cava had been reduced 50 per cent. When only one cava was constricted, the development of venous collaterals subsequently ameliorated these changes. The absence of a demonstrable pressure gradient between cava and atrium during preoperative catheterization has been frequently cited as evidence against the physiologic importance of caval constriction. This reasoning seems to us invalid. Uniform constriction of the right heart and cavae would not be expected to produce an easily detectable pressure gradient until after the ventricles had been decorticated, and even then myocardial insufficiency could lead to erroneous interpretation. Nevertheless, pressure determinations in both inferior and superior cava during decortication could yield interesting information. Marshall and Pantridge 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. He cited 25 cases of visualization of the cavae preoperatively with angiocardioography 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 decortication (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 decortication if a longer period of recovery is accepted. Scientific appraisal has been difficult owing to uncertainty regarding the state of the myocardial function in an individual case at the time of decortication. Dines et al. have emphasized the frequent occurrence of atrophy 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-
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tually compensation 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 in most instances 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 as 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 pericardiectomy between 1955 and 1960.

References

10. Yu, P. N. G., Lovejoy, F. W., Jr., Joos, H. A., Nye, R. E., Jr., and Mahoney, E. B.: Right auricular and ventricular pressure patterns in

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 dementalize 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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