Mechanical and Myocardial Factors in Chronic Constrictive Pericarditis

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The hemodynamic findings in five patients are presented in order to demonstrate the variability in the clinical and physiologic patterns that may be encountered in restrictive disease of the pericardium. The mechanical effects of pericardial encasement previously described were confirmed. Furthermore, it was found that myocardial insufficiency and hypervolemia were not only important causative agents in the circulatory dysfunction but were in some measure reversible, thus permitting resolution of the congestive state and disappearance of the "typical" hemodynamic findings of constrictive pericarditis.

The hemodynamic abnormalities of constrictive pericarditis were first described in 1946 by Bloomfield and associates who stressed "the virtually normal (right) ventricular systolic pressure, a low ventricular pulse pressure, a marked elevation of the mean (right) auricular and ventricular diastolic, prominence of the early diastolic dip in the auricle and ventricle and a marked fall in auricular pressure during ventricular ejection, which, with the diastolic dip, give to the (auricular) tracing a distinct 'W' form." The presence of this diastolic dip was subsequently confirmed by Hansen and co-authors who also described it in the left ventricular curve obtained during surgery and noted its disappearance after pericardectomy. In enlarging on the circulatory abnormalities of constrictive pericarditis, Burwell confirmed the pressure pattern previously described and showed further that there was a small pulse pressure in the pulmonary artery due to a large increase in diastolic and little or no rise in systolic pressure. He focused attention on left ventricular involvement by the pericardial encasement to account for this finding and consequently urged more complete stripping of the left heart surgically. In order to explain the hemodynamic findings more fully Burwell concluded that the stiffened pericardium imposed its own distensibility characteristic equally on the two sides of the heart. The detailed effects of experimentally produced constriction were explored by Isaacs, Carter and Haller who noted, as was implied by Burwell, that the volume-elasticity curves of the two ventricles become identical after experimental production of generalized constriction and that constriction of the ventricular chambers, rather than caval or auricular cavities, is the primary anatomic lesion.

In consequence of these publications, a more or less uniform picture has been accepted as characteristic of constrictive pericarditis, in which the mechanical effects of an inelastic pericardium have been stressed as of primary importance in the production of the clinical and hemodynamic findings.

As a result of studying a group of patients with pericardial disease, using the cardiac catheterization technic, it has become apparent to us that there may be considerable variability in the clinical and physiologic patterns encountered and that in addition to the mechanical factor, there may be an important element of myocardial insufficiency contributing to the circulatory disturbances. A series of five pa-
tients will be presented to elucidate these points. The physiologic observations and their significance will be described and discussed separately in each of the five cases.

Methods

The methods of study, using the cardiac catheterization technic, have been described in detail elsewhere. All the cases were studied under basal conditions at rest, three of them after digitalization, and two of them during a steady state of exercise.

Results and Discussion

First, the importance of the mechanical factors and their effect upon hemodynamics will be analyzed in a patient studied preoperatively and after pericardectomy.

Case 1. This 36 year old Puerto Rican male (no. 596, J. M.) had a four year history of right upper quadrant pain, dyspnea, orthopnea and ankle edema, and on physical examination had a liver extending to the iliac crest and evidence of a left hydrothorax. Cardiac examination revealed a sinus rhythm, no murmurs, thrills or diminution in heart sounds. The etiology of his pericarditis was not known. The preoperative and postoperative x-ray films are seen in figure 1.

The hemodynamic data, (table 1, fig. 2) are similar to those reported as typical of constrictive pericarditis. Preoperatively, the resting cardiac output and stroke volume were not greatly reduced from normal levels. There was pulmonary hypertension present which was almost entirely a diastolic hypertension, the pulse pressure remaining small. This accompanied a threefold increase in the right ventricular diastolic pressure and the usual early diastolic dip was present on the right ventricular pressure curve. The blood volume was considerably increased.

At operation a 5 mm. thick pericardium was found uniformly involving auricles, ventricles and the venae cavae. An extensive decortication of all the chambers was carried out.

Three months later the liver edge lay at the costal margin, and the patient was free of symptoms. Measurements of cardiocirculatory function at this time were within normal limits and the ventricular diastolic dip was absent.

The hemodynamic alterations seen in this patient certainly appear to have been due to the presence of a poorly distensible pericardium. Obviously the thickened pericardium, which enclosed the entire heart, altered the distensibility of the auricles and ventricles and modified the conditions under which a normal venous return was accommodated. As a result of the alteration in distensibility of the right ventricle and auricle the diastolic pressure rose
in these chambers. Similarly diastolic pulmonary hypertension was the reflection of the reduced distensibility of the left heart. Whether the effects of the increased systemic venous pressure upon renal function eventually played a part in the development of hydropsedema can only be surmised.

A close inspection of the pressure abnormalities reveals several points of interest concerning this modification of distensibility. In this patient with pericarditis, preoperatively, the diastolic pressure in the right ventricle was 15 mm. Hg and the diastolic pressure in the pulmonary artery was 21 mm. Hg. Obviously the mean pressure in the left auricle and the end diastolic pressure in the left ventricle were at most 21 mm. Hg, and very likely less. Therefore, this rise was of the same magnitude as that of the end diastolic pressure in the right ventricle. It is known that the distensibility of the normal right and left auricles and ventricles is different, the right being more distensible than the left, which implies that the same increment of volume would tend to produce less pressure response in the right than in the left chamber. Hence it would seem that the most plausible explanation of an absolute rise of similar magnitude in diastolic, and hence venous pressure, in each side of the circulation is that the distensibility characteristic of the pericardium has been substituted for that of the individual chambers.² ⁴

It is important to point out that an identical increment in venous pressure on both sides of the circulation has entirely different effects upon the dynamics of the systemic and pulmonary capillaries. Normally the systemic capillary hydrostatic pressure is close to the oncotic pressure, therefore small increases in systemic venous and hence capillary pressure result in a considerable increase in transudation of fluid from the systemic capillaries. Since the lung hydrostatic capillary pressure is normally around 5 mm. Hg, obviously much lower than systemic capillary pressure, there is a greater pressure differential or margin of safety, between oncotic and hydrostatic pressure in the pulmonary capillary system than in the systemic capillaries. Thus a rise in pulmonary venous pressure, even though it adds to the lung capillary hydrostatic pressure an increment of similar magnitude to the rise of systemic venous pressure, may not cause hydrostatic to exceed oncotic pressure and hence transudation into alveoli would not occur. This explains why with clinical evidence of considerable systemic congestion there is seldom, if ever, frank pulmonary edema in constrictive pericarditis.

Furthermore, it is noteworthy that the pulse pressure in the pulmonary artery is small, in contradistinction to the large pulse pressure seen in left ventricular failure. In the latter condition, heterodynamism of the two ventricles, the right being spared and capable of delivering more blood to the lungs than the left can eject into the systemic circulation, permits trapping of blood in the pulmonary bed with a rise in pulmonary artery blood volume and pulse pressure. The small pulse pressure in constrictive pericarditis indicates that heterodynamism of the ventricles probably does not exist.³

It is a matter of speculation whether the presence of a stiff pericardium actually influences the contractile mechanism of the encased myocardial fibers, in particular their tension and elongation. Although this might well be expected, it is quite interesting that in this case, as well as in some others with this same type of lesion, stroke volume is normal or only slightly reduced although the ventricular filling pressure is elevated considerably above normal.

Although it appears that during systole the ventricular musculature more or less successfully contracts against the forces of the inelastic pericardium, at the end of systole these forces become manifest suddenly, and the ventricle relaxes precipitously instead of in an isometric fashion. With this rapid change in the volume of the ventricular cavity there is a rapid fall in pressure which produces the postystolic and early diastolic dip in the ventricular and auricular tracings. However, an alternative mechanism could be offered to explain this dip, namely, that the stiffened pericardium may, at the time of isometric relaxation, set into play a low frequency vibration producing an overshooting of the pressure curve. In any event, the dip appears to be a consistent finding
**Table 1.—Physiologic Measurements in Five Patients with Pericardial Disease**

<table>
<thead>
<tr>
<th>Case</th>
<th>BSA</th>
<th>Cardiac Index $(L/min/\text{M}^2 \text{BSA})$</th>
<th>Stroke Volume $(cc.)$</th>
<th>Heart Rate $(beats/\text{min.})$</th>
<th>Oxygen Consumption $(cc./\text{min.}/\text{M}^2 \text{BSA})$</th>
<th>R.Q.</th>
<th>AV diff $(\text{vol.} %)$</th>
<th>Arterial Blood Oxygen $\text{cont.} (\text{vol.} %)$</th>
<th>Systemic Artery $(s/d, \text{m})$</th>
<th>Pulmonary Artery $(s/d, \text{m})$</th>
<th>Right Ventricle $(d)$</th>
<th>TBV $(cc./\text{M}^2 \text{BSA})$</th>
<th>PV $(cc./\text{M}^2 \text{BSA})$</th>
<th>H'ert $(%)$</th>
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<td>2.60</td>
<td>56</td>
<td>86</td>
<td>119</td>
<td>0.82</td>
<td>4.6</td>
<td>16.6</td>
<td>93</td>
<td>118/77</td>
<td>96</td>
<td>30/21</td>
<td>23</td>
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</tr>
<tr>
<td>J. M.</td>
<td>(b) 1.85</td>
<td>3.27</td>
<td>78</td>
<td>78</td>
<td>125</td>
<td>0.72</td>
<td>3.8</td>
<td>20.8</td>
<td>96</td>
<td>110/72</td>
<td>90</td>
<td>18/8</td>
<td>11</td>
<td>5</td>
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<tr>
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<td>79</td>
<td>80</td>
<td>134</td>
<td>0.76</td>
<td>4.2</td>
<td>15.8</td>
<td>95</td>
<td>100/65</td>
<td>81</td>
<td>35</td>
<td>19</td>
<td>25</td>
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<td>2.76</td>
<td>68</td>
<td>80</td>
<td>133</td>
<td>0.77</td>
<td>4.8</td>
<td>16.0</td>
<td>95</td>
<td>108/67</td>
<td>80</td>
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<td>19</td>
<td>25</td>
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<tr>
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<td>78</td>
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<td>4.6</td>
<td>20.2</td>
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<td>115/70</td>
<td>86</td>
<td>15/5</td>
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<td>124/78</td>
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<td>33/19</td>
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<td>18</td>
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<tr>
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<td>2.88</td>
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<td>96</td>
<td>138</td>
<td>0.80</td>
<td>4.8</td>
<td>17.3</td>
<td>91</td>
<td>97/66</td>
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<td>22/8</td>
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<td>5</td>
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<tr>
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<td>45</td>
<td>81</td>
<td>118</td>
<td>0.81</td>
<td>5.3</td>
<td>18.9</td>
<td>97</td>
<td>93/60</td>
<td>70</td>
<td>18/8</td>
<td>12</td>
<td>6</td>
<td>2598</td>
</tr>
<tr>
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<td>84</td>
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<td>0.81</td>
<td>6.9</td>
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<td>82</td>
<td>18/9</td>
<td>14</td>
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</table>

(a) At rest, preoperatively; (b) at rest, 3 months postpericardectomy; (c) control at rest, 1951; (c') 46 minutes after intravenous injection of 1.25 mg. Digoxin, 1951; (d) control at rest, 1953; (d') after 8 minutes of exercise, 1953; (e) at rest; (f) control at rest; (f') 64 minutes after intravenous injection of 1.0 mg. Digoxin; (g) at rest, 1951; (h) at rest, 4 weeks later, 1951; (i) control at rest 24 months later, 1953; (i') after 10 minutes of exercise, 1953.

**Abbreviations:** BSA = body surface area; AV diff = arteriovenous oxygen difference; TBV = total blood volume; PV = plasma volume; H'ert = hematocrit.
in chronic constrictive pericarditis, and, in the successfully operated case, disappears after pericardectomy, as it did in this patient.

The second case demonstrates the surprising fact that the classical hemodynamic abnormalities associated with constrictive pericarditis may exist in the absence of any clinical expression of circulatory congestion.

Case 2. This 47 year old white male (No. 638, J. R.) gave a story of pulmonary and laryngeal tuberculosis associated with mild dyspnea dating back 15 years. In addition there was a well documented story of tuberculous pericarditis with effusion in 1946 for which he received streptomycin. The x-ray film of this date is seen in figure 3. Subsequent films (fig. 3) have shown a reduction in heart size which has been stationary from 1948 to 1953. In 1951 he was admitted to Bellevue Hospital because of a small hemoptysis, but without other complaints.

Examination at this time revealed a sinus mechanism, no murmurs or diminution of heart sounds, and no evidence of an enlarged liver or edema. The first hemodynamic study in 1951 showed a normal cardiac output and a slight increase in blood volume (table 1, fig. 4). Mild pulmonary hypertension, a considerable elevation of the right ventricular diastolic pressure and the characteristic early right ventricular diastolic dip were all present. In the course of this study, the patient was given intravenous Digoxin and the only change observed was a fall in cardiac output of 14 per cent. This type of response to intravenous Digoxin has been noted in subjects in whom there was no evidence of congestive heart failure. The patient was discharged and continued his usual activities as an itinerant salesman. Clinically he remained free of pulmonary or peripheral congestion and received no further medications.

In 1953, two years after his first study, he was re-evaluated. The only significant changes in the hemodynamic findings (table 1, fig. 4) were a slight fall in cardiac output which still lay within normal limits and a slight reduction in blood volume. On mild exercise, which in a normal subject would cause a rise in blood flow of at least 600 cc. for every 100 cc. increase in oxygen consumption, there was only an increase of 330 cc. With this small increase in cardiac output, pulmonary artery and right ventricular hypertension became aggravated. Since myocardial failure in the usual sense appears to have been eliminated as a factor by the Digoxin study, it would appear that the abnormal findings on exercise were caused by the restrictive effects of the pericardium upon filling and contraction of the heart.

Hence at rest and during exercise there is evidence that the diseased pericardium adversely affected the circulation despite which the clinical manifestations of this lesion were absent. Why then did not this patient show the classic clinical signs, namely edema, a large liver and ascites? Was the degree of cardiac restriction due to stiffening of the pericardium not great enough to limit cardiac output nor to elevate venous pressure to a critical level above which edema is observed, or is it that myocardial function, although altered by this
abnormal pericardium, is still able to perform well under this duress? It is possible that this patient with evidence of hematogenous tuberculosis may have had some adrenal insufficiency and alteration in salt and water metabolism sufficient to prevent edema formation. Be that as it may, at the time of the first study it was decided not to subject the patient to pericardectomy in the absence of the clinical criteria. After a two-year follow-up during which he led a fairly active life it would seem that the lack of change in the clinical as well as the physiologic status still justifies surgical abstinence.

That stiffening of the pericardium is not always productive of hemodynamic or clinical abnormalities is clearly shown in the next patient, where the presence of a calcified pericardium was not attended by any alteration in the dynamics of the circulation.

Case 3. (No. 661, G. M.) This 59 year old white male had an extensively calcified pericardium of unknown cause (fig. 5); nevertheless, there were no objective findings of either pulmonary or peripheral congestion. The tracing from the right ventricle of this patient (fig. 6) did not show the early diastolic dip. Furthermore there were normal pressures in both the greater and lesser circuits and a normal blood flow and blood volume (table 1). Certainly this calcified pericardium was not interfering with the patient's circulation at rest.

The observation that a calcified pericardium need not embarrass the circulation has been known clinically for some time and suggests that it is the degree of cardiac restriction which may follow pericardial disease rather than stiffening alone, that plays an essential role in the development of the clinical or hemodynamic picture.

In sharp contrast to the cases previously described, the physiologic studies made in the

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Fig. 3. Posteranterior x-ray films of patient No. 638, J. R. For discussion see text.

Fig. 4. Graphic representation of the hemodynamic findings in patient No. 638, J. R. The patient received 1.25 mg. Digoxin intravenously at the time of the first study and 23 months later, at the time of the second study, the response to exercise was determined. The value 330 which appears in the exercise column refers to the blood flow increase in cubic centimeters per 100 cc. oxygen consumption increase. For discussion see text.
next patient emphasize the fact that failure of the myocardium may contribute in considerable measure to the congestive state in constrictive pericarditis.

Case 4. This 28 year old white male (No. 619, G. R.) had only a two-month history of edema and dyspnea. At the time of examination he was found to have auricular flutter with a high degree of A-V block and a slow ventricular rate. No murmurs were heard, nor were the heart sounds faint. A diastolic gallop was audible at the apex. Calcium was present in the pericardium as seen in the x-ray films (fig. 7). The liver edge was palpated four finger breadths below the costal margin. No definite etiology of the pericarditis was established.

In contrast to the first case the cardiac output was considerably reduced. However, many
of the other hemodynamic features were characteristic of constrictive pericarditis since the pulmonary artery pulse pressure was small with a high diastolic and only a slight elevation of systolic pressure. The right ventricular diastolic pressure was elevated (table 1), and the early diastolic dip was apparent. It should be noted that there was a considerable degree of hypervolemia. Immediately following the administration of intravenous Digoxin the cardiac output rose, and even before there was a slowing of the ventricular rate, there was a significant fall in the pulmonary artery and right ventricular pressures (fig. 8). This response is characteristic of a failing myocardium in which improved ventricular emptying follows the administration of the glycoside and, therefore, this acute Digoxin study demonstrates the existence of myocardial failure in this individual with constrictive pericarditis. The fact that the pulmonary artery and right ventricular diastolic pressures were still elevated at the conclusion of the study (80 minutes after the administration of Digoxin) might be ascribed to the persistent mechanical interference of the pericardium. However this explanation cannot be offered without reservation because experience with other patients in congestive failure and
marked hypervolemia, but without pericardial disease, has shown that the complex readjustments set into play by the glycoside may not restore the intracardiac pressures to a normal state for a considerable period of time.5, 12

In summary this case serves to emphasize the important role that myocardial failure may play in this disease. Since further definitive studies are not available either after prolonged medical regime or after pericardectomy, the role played by the stiffened pericardium in this man was not further elucidated.

In the last patient of this group, serial measurements over a two-year period revealed that considerable improvement. An intensive search at this time for the cause of his pericarditis was unrewarding but later on (in 1952) he developed a minimal lesion of pulmonary tuberculosis. He returned to work as a laborer, and discontinued all medications. For two years he remained asymptomatic. In 1946 he returned with the same complaints and findings although he was not as ill as previously nor was there thought to be a significant amount of pericardial fluid. Again a medical regimen abolished evidence of congestion. X-ray films at the time of his first and second admissions are in the first two frames in figure 9, and in the last frame is a follow-up film taken in 1947. He remained well, at work, and without medication until 1951 when he reappeared dyspneic and edematous. Examination of the heart showed no murmurs or diminution in heart sounds. For the first time x-ray films revealed evidence of pericardial calcification as indicated by the arrow in figure 10.

It was at this time that he was first studied (table 1 and fig. 11). The cardiac output was at the upper limits of normal. All the other hemodynamic findings noted in the first case were present, namely a high diastolic and small pulse pressure in the pulmonary artery, a high diastolic pressure in the right ventricle and the characteristic dip in the ventricular curve (fig. 6). The blood volume was also markedly elevated.

Following this study he was kept on bedrest, digitalized, and given diuretics. Four weeks later, following marked clinical improvement and alleviation of all signs and symptoms of congestion, he was restudied. At this time the cardiac output was lower, the pulmonary artery pressures were normal, and the right ventricular diastolic pressure almost normal. There had been also a large reduction in blood volume. The right ventricular and auricular curves, however, still showed the dip. There was a

![Fig. 9. Posteroanterior x-ray films of patient No. 647, A. G. For discussion see text.](image-url)
Fig. 10. Posteroanterior and left anterior oblique x-ray films of patient No. 647, A. G. The upper two x-ray films were taken at the time of the first study and the lower two at the time of the second. For discussion see text.
striking diminution in heart size as can be seen in figure 10. The patient was discharged and remained on digitalis. Six months later he developed a minimal lesion of pulmonary tuberculosis for which he received six months of bedrest and streptomycin. Another period free of symptoms, this time without medication, ensued. However, in December 1952 dyspnea and weight gain prompted redigitalization and resulted in relief of dyspnea. Five months later he suffered from severe paroxysmal nocturnal dyspnea, had a protodiastolic gallop and pulmonary rhonchi. He was therefore hospitalized and mercurial diuretics were added to the digitalis therapy. After one month he was restudied and at this time no gallop, rhonchi or other signs of congestion were present. The heart size and degree of calcification were unchanged and the x-ray films resembled those made June 11, 1951.

This third hemodynamic evaluation demonstrated pulmonary artery and right ventricular pressures which were the same as on the second study, that is, within normal limits save for a slight elevation in the diastolic pressure of the right ventricle. The blood volume was the lowest recorded on this patient. However, cardiac output was now definitely lower than normal at rest and its increase during leg exercise was limited to 466 cc. per 100 cc. of increase in oxygen consumption. During this exercise, with a level of cardiac output which was less than that of a normal subject at rest, the pressure in the pulmonary artery remained within normal limits although the diastolic pressure in the right ventricle was definitely abnormal. The exercise response of this patient (table 1 and fig. 11) should be contrasted with that of the second case (no. 638) in whom restriction was felt to be responsible for the pulmonary hypertension at rest and its aggravation on exercise. The dip still persisted in the ventricular and auricular tracings.

In the light of these follow-up studies one cannot escape the conclusion that there was a reversible phenomenon which was largely responsible for the original congestive state of this patient, particularly since his improvement has been more or less maintained on a medical regimen for a period of two years. Since both the clinical and hemodynamic phenomena were reversible, it is difficult to accept the mechanical interference of pericardial constriction as the sole agent in the production of this picture. Actually the degree of restriction was probably less than in other cases, and a reduction in the capacity of the pericardial sac was only a hindrance to the circulation when other factors, namely myocardial insufficiency and hypervolemia were present.

The following observations may be cited to support the interpretation that a relatively small degree of pericardial restriction existed. First, one notes the absence of pulmonary hypertension even on exertion once congestion had been relieved, in sharp contrast to the second case (No. 638), whose restriction was severe enough to produce pulmonary hypertension at rest and aggravate it on exercise, even though he also was not in a congestive state. Second, diminution in the size of the cardiac silhouette between the first two studies indicates that the volume of the heart was not determined solely by the fixed pericardium and that the pericardial wall was able to yield when the length of the myocardial fibers was reduced. In spite of the small degree of pericardial restriction one cannot escape the conclusion that when the patient was in the congestive state this degree of restriction did influence the hemodynamic picture since the pressure pattern encountered at the time of the first study was indistinguishable from that observed in the first case (No. 596) before pericardectomy.

Analysis of other varying hemodynamic features points out the important part played by the state of the myocardium and by hypervolemia in the progress of this man's disease.
It should be noted that the cardiac output was highest in the congestive state and that as the cardiac output decreased the blood volume also fell. It is tempting therefore to relate these two functions and raise the question as to whether the hyervolemia of itself influenced the level of blood flow, since it has been found in some other forms of congestive heart failure that hyervaolemia is a primary factor in increasing blood flow and in bringing about failure of the myocardium.19 At the time of the third study the level of cardiac output was so markedly reduced at rest and rose so little on exercise that one must assume that the integrity of the myocardium itself was compromised.

It is impossible to reconstruct with any degree of certainty the sequence of events which transpired throughout this patient's illness. One could however postulate, for example, that a mild degree of restriction remained in this man after the initial phase of pericardial inflammation subsided. This restriction was adversely influential only during bouts of severe exertion associated with the patient's resumption of his normal activity as a manual laborer, when it produced a rise in ventricular diastolic pressure together with an inadequate response in blood flow. These in turn stimulated progressive hyervolemia and thereby an increasing venous return to the point where the diastolic residual volume of the heart was large enough and dilatation of myocardial fibers great enough to be influenced by and assume the distensibility characteristics of the restricted pericardium at rest. At this stage all the findings resembled the so-called typical restrictive phase of the disease.

It may well be that myocardial insufficiency, apart from the mechanical factors, supervened at some point and further contributed to the congestive state. The nature of this myocardial insufficiency is unknown but may be related to the original inflammatory process, perhaps by contiguity, or may result from the stress imposed by the stiffened pericardium on the cardiac muscle.

In summary, one can only say that increased cardiac residual volume associated with generalized hyervolemia, some restriction of the pericardium and the effects of abnormal myocardial function are probably all involved in producing the physiologic patterns described. Perhaps no one factor alone was abnormal enough to destroy circulatory compensation, but the interplay of all three combined to generate the congestive state.

Summary and Conclusions

1. Five patients studied by means of the cardiac catheterization technique are presented in order to analyze the factors contributing to circulatory dysfunction in restrictive disease of the pericardium.

2. It is noteworthy that the clinical as well as the hemodynamic picture was not the same in any of the five subjects. In one, the mechanical factor proved to be the dominant agent. In a second, the classic hemodynamic abnormalities of chronic constrictive pericarditis were found to exist in the absence of any clinical expression of circulatory congestion. A third patient demonstrated that a calcified pericardium need not be attended by any circulatory disturbance. Myocardial failure was shown to contribute to the congestive state in a fourth patient. The reversibility of the typical clinical hemodynamic pattern of congestion on medical management alone in a fifth subject posed the question as to what was the primary cause of his abnormalities.

3. Previous hemodynamic analyses have presented a more or less uniform picture of constrictive pericarditis laying the basic disturbance to the mechanical effects of the encasement. In this report analysis of the hemodynamic pattern which in four of the five patients with pericarditis initially was quite similar to that previously described, has revealed however that all the disturbances could not be ascribed solely to mechanical restriction of the heart. Indeed it was found that myocardial insufficiency and hyervolemia were not only important causative agents in the circulatory dysfunction but by proving to be in some measure reversible, permitted resolution of the congestive state and disappearance of the "typical" hemodynamic findings of constrictive pericarditis.

4. It is obvious from these findings that considerable study should be given to the developmental changes which occur as this disease...
progresses and to the relative importance and interplay of such factors as the variation in the restrictive action of the pericardium, myocardial function and hypervolemia.

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SUMARIO ESPAÑOL

Se presentan los hallazgos hemodinámicos en 5 pacientes para poder demostrar la variabilidad en los patrones clínicos y fisiológicos que puedan encontrarse en enfermedad constrictiva del pericardio. Los efectos mecánicos de constrictión por el pericardio previamente descritos se confirman. Además, se encontró que insuficiencia del miocardio e hipervolemia no solamente fueron causas importantes en la disfunción circulatoria pero también probaron ser en alguna medida reversibles, así permitiendo una resolución del estado congestivo y desaparición de los hallazgos hemodinámicos típicos de la pericarditis constrictiva.

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