Constrictive Pericarditis: Serial Hemodynamic Studies with an Explanation for Reversible Congestive Phenomena

By Timothy R. Murphy, M.D., Julius M. Meyer, M.D., and Jules Chase, M.D.

A new explanation for certain instances of acute reversible congestive phenomena in constrictive pericarditis is offered on the basis of 6 patients studied clinically and by right heart catheterization. In 2 cases intermittent tamponade in the presence of a rigid pericardium accounted for the reversibility of the congestive state, as contrasted with 4 cases presenting the chronic progressive course of classical constrictive pericarditis. The diagnosis in the 6 cases was proved by surgery or autopsy.

The clinical picture of constrictive pericarditis with pericardial thickening and calcification together with the relentless and progressive course of the disease is the subject of many excellent papers. However, only one single report has been found on the reversible aspects of the congestive phenomena in constrictive pericarditis.

This report presents 2 cases of constrictive pericarditis exhibiting reversible congestive findings secondary to superimposed cardiac tamponade. Serial hemodynamic studies in 4 other cases of classical constrictive pericarditis are discussed.

Material and Methods

Right heart catheterization was performed on 6 patients under basal conditions with no premedication. Repeated catheterizations were carried out in 4 of the patients over 1- to 4-year periods. In 2 patients postoperative measurements could not be obtained.

Pressure measurements were obtained in the right atrium, right ventricle, pulmonary artery, and pulmonary wedge position during rest and exercise. Cardiac output in the resting state was determined by the direct Fick method, usually in duplicate. All duplicate determinations agreed within 10 per cent. The average is used in table 1.

The exercise consisted of simple leg raising at a rate of 30 per minute for 7 to 8 minutes with measurements of cardiac output during the last 3 minutes of this period.

Expired air was collected in the Collins gasometer of the Tissot type. Blood gases were measured according to the method of Van Slyke and Neill and expired air was analyzed by the Schoenheimer micrometer apparatus.

Case Reports

Cases with Reversible Congestive Phenomena

Case 1, R.B. This 50-year-old white man was hospitalized for a herniorrhaphy in August 1951. A chest film revealed extensive calcification over the left ventricle. The patient had no cardiovascular complaints until September 4, 1951, when frequent cough and breathlessness on effort appeared. On October 8 the rapid onset of severe dyspnea, chest oppression, and facial edema necessitated hospitalization.

Three episodes of pneumonia had occurred during childhood and early adult life.

On physical examination there was mild facial edema, cyanosis of the lips and distended neck veins; the blood pressure was 120/80. The cardiac rate was regular at 90. The heart tones were muffled and there were no murmurs. Bilateral basal rales and a left pleural friction rub were present. The latter disappeared the following day. The liver edge was felt 5 cm. below the costal margin. There was no peripheral edema. The electrocardiogram demonstrated a sinus rhythm, low QRS voltage, and T-wave inversion in the standard leads. A chest roentgenogram revealed pericardial calcification, left atrial enlargement, pleural thickening at both bases, and a normal transverse diameter unchanged from August 1951. Digitalization, low-salt diet, and mercurial diuretics were followed by a 10-pound weight loss and marked clinical improvement. The venous pressure on the third hospital day was 220 mm. of water. A pericardial friction rub persisting for 24 hours was heard on the ninth hospital day.

On October 25, 1951, digoxin was stopped. There was no weight gain, venous pressures were unchanged, liver enlargement persisted, and 1 episode of transient atrial fibrillation occurred. The patient at this time was asymptomatic except for...
CONSTRUCTIVE PERICARDITIS

breathlessness on effort. The results of cardiac catheterization on November 11, 1951, are recorded in table 1. He was redigitalized preoperatively without change in weight or symptoms.

At surgery on November 20, a thick calcified pericardium was found. On incision of the pericardium a milky fluid under pressure escaped. The blood pressure immediately rose 20 mm. Hg. The pericardium was adherent to the epicardium only at the apex. The epicardium had a dull gray appearance and calcium plaques covered one third of the anterior surface and one half of the posterior aspect. The presence of tamponade, secondary to the accumulation of pericardial fluid, as a cause of the acute congestive phenomena was not appreciated prior to surgery.

Postoperatively the patient did very well, until on the tenth postoperative day a wound infection accompanied by sternal separation occurred. Repeated surgical attempts to immobilize the sternum were unsuccessful. He died 20 days postoperatively. At necropsy the valves were normal and the foramen ovale was closed. Neither the etiology nor pathogenesis of the pericardial inflammation and pre-existing constriction could be ascertained. Cultures of the pericardial fluid were negative. Microscopic examination of the pericardium demonstrated calcification, fibrous thickening, and perivascular lymphocytic infiltration.

Comment. Cardiac catheterization (table 1, R.B.) revealed a cardiac index of 2.36 L. per min. per M.$^2$ and moderate elevation of the pulmonary wedge, right ventricular end-diastolic, and right atrial mean pressures. The pulmonary “wedge” pressure had the configuration usually associated with mitral stenosis, though the mitral valve was normal at autopsy. In this instance, the configuration was probably secondary to constriction of the left ventricle with the atrium contracting against an elevated left ventricular end-diastolic pressure.$^2$

The presence of calcification confirmed the impression that the pericardial involvement was of some duration. The left atrial enlargement would suggest that a minor degree of constriction about the left ventricle had been present for some time, despite the absence of symptoms. The immediate increase in the blood pressure on incising the pericardium establishes tamponade as the cause of the sudden failure.

This case represented mild chronic pericardial constriction with sudden congestive symptoms occurring only after the development of an acute pericardial effusion where the pericardium was thick and could not expand.

Case 2, G.E. This 28-year-old white man had pneumonia at ages 3, 18, and 28. The second episode was apparently of viral etiology with a stormy course and electrocardiographic evidence of toxic myocarditis or pericarditis. The third episode of pneumonia occurred on June 28, 1953 (fig. 1). Sudden dyspnea, chest oppression, atrial flutter, and syncope led to hospitalization on July 8, 1953.

Physical examination revealed cyanosis, neck vein distention, blood pressure of 110/70, a grade I apical systolic murmur, and hepatosplenomegaly. Sinus rhythm was restored with digitalis and symptoms abated, but hepatosplenomegaly and the murmur persisted. The electrocardiogram showed low QRS voltage and isoelectric T waves in the standard leads. The patient was discharged on the tenth day. Right heart catheterization was performed on November 17, 1953 (table 1, G.E.), 1 month after cessation of digitalis.

Breathlessness on effort and palpitation with occasional syncope required rehospitalization in May 1954. There had been no change in the physical findings since his discharge in July 1953. Cardiac fluoroscopy and venous pressure were normal. The electrocardiogram showed normal QRS voltage, a P-R interval of 0.24 second with wide, notched P waves, and isoelectric T waves in the standard leads. One episode of atrial flutter occurred while in the hospital with conversion to sinus rhythm by digitalis. The skin tests for tuberculosis were positive. The patient was discharged on August 24, 1954, with a diagnosis of "myocarditis of unknown etiology."

He was readmitted in January 1956 with exertional dyspnea, syncope, and facial edema of 2 days' duration. The physical examination was unchanged with the exception of progressive hepatosplenomegaly. Cardiac fluoroscopy now demonstrated pericardial calcification, predominantly over the posterior and diaphragmatic aspect of the left ventricle. There were frequent episodes of supraventricular arrhythmias.

The venous pressure varied between 210 and 224 mm. of water. An injection of a mercurial diuretic resulted in a 10-pound weight loss and right heart catheterization 2 days later (table 1, G.E.) demonstrated a resting right atrial pressure of 6 mm. Hg. Digitalization with intravenous digoxin resulted in no significant change in intracardiac pressures or cardiac output over a 3-hour
period. After a regime without medication or salt restriction, the venous pressure was 192 mm. of water, responding to mercurial diuresis with a fall to normal.

The sudden onset of congestive phenomena, the fluctuation of venous pressures and symptoms, together with minimal variation in heart size over a 3-year period, suggested the diagnosis of constrictive pericarditis with superimposed tamponade. This diagnosis was confirmed at surgery on April 17, 1956, when a dissection of the thickened pericardium over the right ventricle revealed a space 10 by 10 cm. containing fluid under pressure. The fluid had a calcium value of 1484 mg./100 ml. and the cultures were negative. The postoperative course was uneventful, the symptoms and physical findings returning to normal. All medications were discontinued in July and right heart catheterization was repeated in October, 1956 (table 1, G.E.).

Comment. The right heart catheterization in 1953 revealed a low cardiac index of 1.65 L. per min. per M.² and a right ventricular end-diastolic pressure of 12 mm. Hg. In 1956 the cardiac index had risen to 2.33 L. per min. per M.² and the right ventricular end-diastolic pressure had fallen to 9 mm. Hg, with marked increases on exercise. The configuration of the resting pressure curves in 1953 and 1956 were only suggestive of constrictive pericarditis. The exercise curves in 1956 showed the "typical constrictive pattern." The studies 6 months after operation revealed a relatively unchanged cardiac index from that immediately preoperatively, but resting and exercise pressures were normal.

The pneumonia at age 18 was quite severe and accompanied by electrocardiographic changes compatible with pericardial injury. The third episode of pneumonia in 1953 was associated with a definite increase in heart size (fig. 1), low voltage on the electrocardiogram, and marked symptoms, and presumably marked the onset of the pericardial effusion limited by a thickened pericardium. Roentgenograms showed fluctuation in heart size between 1953 and 1956, being largest at the time of pneumonia in 1953 and smallest just prior to surgery in 1956.

After the pneumonia in 1953 the patient was in ill health with episodes of atrial ar-

rhythmia, progressive hepatosplenomegaly, and fluctuating venous and right heart pressures. These alterations in clinical status, venous and right heart pressures, and cardiac output indicate a reversible element. Myocardial insufficiency and changes in blood volume have been suggested as causes for this reversibility. A more plausible explanation in this case is the fluctuation in the volume of fluid within a rigid pericardial sac.
Postoperative right heart catheterization on October 18, 1956, 6 months after surgery, showed the intracardiac pressures to be within normal limits at rest and on exercise. The cardiac index had risen slightly.

Cases with Classical Features

Case 3, G.U. A 49-year-old white man was admitted in November 1941, with impending delirium tremens. The cardiovascular examination was negative except for distention of the neck veins. The electrocardiogram showed low voltage of the QRS-T complexes. No cardiac diagnosis was made and the patient was discharged after treatment for portal cirrhosis.

Subsequently, there were repeated hospital admissions with hepatomegaly appearing in 1942 followed 2 years later by ascites and exertional dyspnea.

In September 1951 the diagnosis of constrictive pericarditis was made and right heart catheterization was performed (table 1, G.U.). The patient refused surgery, and with salt restriction and mercurial diuretics was only moderately limited in his activity.

Pericardial calcification was first detected in 1953, at which time cardiac catheterization was repeated. The clinical status was unchanged.

In August 1954, an episode of atrial flutter responded to digitalis. The ascites, neck vein distention, peripheral edema, and exertional dyspnea became progressively more severe. Atrial fibrillation appeared in July 1955. Cardiac catheterization was repeated.

A few months later death occurred due to spontaneous thrombosis of the abdominal aorta. Autopsy revealed a constricting, heavily calcified pericardium with complete obliteration of the pericardial space. Portal cirrhosis was also present.

Comment. The major hemodynamic changes between the catheterizations of 1951 and 1953 included a definite fall in the cardiac index, unchanged right atrial pressures, and slight elevation of the right ventricular end-diastolic and pulmonary artery mean pressures (table 1).

The circulatory changes between 1953 and 1955 consisted of an unchanged cardiac index, despite the presence of atrial fibrillation, and a distinct rise in the right atrial and pulmonary wedge pressures.

The 35 per cent reduction in cardiac output between 1951 and 1953 was unaccompanied by discernible clinical deterioration. Progression of symptoms did not become manifest until 1 year later. Then clinical deterioration was relentless but the cardiac index showed no further fall.

Case 4, R.Ru. A 29-year-old white man was admitted June 21, 1953, with mild epigastric distress and occasional bouts of palpitation. The physical examination was negative. The diagnosis of duodenal ulcer was established and pericardial calcification was demonstrated by x-ray, the latter having been present since 1943. The electrocardiogram showed marked right axis deviation, vertical electric axis, and a left heart strain pattern.

Between 1953 and 1956 the only cardiac complaint was vague exertional fatigue. However, during this period, the patient was studied annually by right heart catheterization, which revealed progressive increases in right atrial, right ventricular end-diastolic, and pulmonary wedge pressures with no change in the cardiac output.

In view of the pressure increases, pericardectomy was performed in January 1956. A thick calcified pericardium was found with complete obliteration of the pericardial space. The patient returned to his road construction work in May 1956 and noted a distinct improvement in his work tolerance. In July 1956 right heart catheterization showed return of all pressures to normal at rest and during exercise, without change in the cardiac output.

Comment. Over the period of observation, the pressures increased without comparable progression of symptoms. This emphasizes that in chronic constrictive pericarditis appreciable pressure increases may occur with clinical manifestation so insidious that their significance is apparent only postoperatively.

Case 5, R.Ru. A 26-year-old white male musician was seen in October 1954, with progressive ankle edema, exertional breathlessness, and paroxysmal atrial flutter of 5 years' duration, considered to be secondary to rheumatic heart disease with mitral stenosis until pericardial calcification was detected in 1954. There were moderate cardiac enlargement, a grade II apical systolic murmur, neck vein distention, and hepatosplenomegaly.

Chest roentgenogram revealed extensive pericardial calcification with moderate enlargement of the left atrium and pulmonary artery, and minimal enlargement of the left ventricle. The electrocardiogram showed wide notched P waves, frequent atrial premature beats, and a left heart strain pattern.
TABLE 1.—Hemodynamic Data in Six Patients with Constrictive Pericarditis

<table>
<thead>
<tr>
<th>Case and age (yr.)</th>
<th>Date of catheterization</th>
<th>State</th>
<th>Body surface area</th>
<th>Cardiac index (L./min./M.² BSA)</th>
<th>Oxygen consumption (ml./min./M.² BSA)</th>
<th>Respiratory quotient</th>
<th>A-V O₂ diff.(vol. %)</th>
<th>Right atrial mean</th>
<th>Right ventricle (s/d)</th>
<th>Pulmonary artery (s/d,m)</th>
<th>Pulmonary wedge mean</th>
<th>Systemic artery pressure</th>
<th>Arterial blood oxygen</th>
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<td>R</td>
<td>1.9</td>
<td>2.36</td>
<td>148</td>
<td>.71</td>
<td>6.3</td>
<td>15</td>
<td>35/17</td>
<td>35/21/24</td>
<td>16</td>
<td>120/85</td>
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</tr>
<tr>
<td>G.E., 30</td>
<td>11/17/33</td>
<td>R</td>
<td>2.00</td>
<td>1.65</td>
<td>122</td>
<td>—</td>
<td>7.3</td>
<td>10</td>
<td>25/12</td>
<td>25/10,—</td>
<td>—</td>
<td>110/70</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>2/23/56†</td>
<td>R</td>
<td>—</td>
<td>—</td>
<td>—</td>
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<td>6</td>
<td>20/9</td>
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<td>19.3</td>
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<tr>
<td></td>
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<td>E</td>
<td>2.02</td>
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<td>.80</td>
<td>5.2</td>
<td>3</td>
<td>20/2</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>7</td>
<td>30/4</td>
<td>28/10,17</td>
<td>—</td>
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<tr>
<td>G.U.* 59</td>
<td>12/8/51</td>
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<td>2.61</td>
<td>119</td>
<td>.82</td>
<td>4.5</td>
<td>12</td>
<td>40/14</td>
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<td>—</td>
<td>124/70</td>
<td>18.4</td>
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<tr>
<td></td>
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<td>1.85</td>
<td>103</td>
<td>.85</td>
<td>5.7</td>
<td>12</td>
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<td>18.7</td>
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<tr>
<td></td>
<td>7/5/55†</td>
<td>R</td>
<td>1.86</td>
<td>1.75</td>
<td>116</td>
<td>.86</td>
<td>6.5</td>
<td>19</td>
<td>40/20</td>
<td>40/20,25</td>
<td>22</td>
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<tr>
<td>R. Ro., 29</td>
<td>8/25/53†</td>
<td>R</td>
<td>1.73</td>
<td>3.63</td>
<td>128</td>
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<td>4.0</td>
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<td>128/88</td>
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<td></td>
<td>E</td>
<td>—</td>
<td>4.25</td>
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<td>15</td>
<td>31/17</td>
<td>31/15,28</td>
<td>11</td>
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<tr>
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<td>10</td>
<td>30/15</td>
<td>30/10,18</td>
<td>—</td>
<td>140/96</td>
<td>17.3</td>
</tr>
<tr>
<td>R. Ru.* 26</td>
<td>11/17/54</td>
<td>R</td>
<td>1.82</td>
<td>2.73</td>
<td>188</td>
<td>.86</td>
<td>6.9</td>
<td>26</td>
<td>45/25</td>
<td>40/30,35</td>
<td>28</td>
<td>140/80</td>
<td>21.5</td>
</tr>
<tr>
<td>H.T., 27</td>
<td>3/24/53</td>
<td>R</td>
<td>1.79</td>
<td>2.27</td>
<td>169</td>
<td>.86</td>
<td>7.5</td>
<td>13</td>
<td>36/18</td>
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<td>17</td>
<td>40/21</td>
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<td>27/12,22</td>
<td>—</td>
<td>—</td>
<td>18.9</td>
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* Postoperative catheterization not done.
† Done in duplicate, see text; R—Rest; E—Exercise.

Cardiac catheterization (table 1, R.Ru.) revealed a normal cardiac index, marked elevation of right atrial, right ventricular end-diastolic, and pulmonary wedge pressures.

Pericardiectomy was performed in November 1954. A thick pericardium with obliteration of the pericardial space was found. Postoperative recovery was excellent.

Comment. Despite a 6-year period of severe symptoms and marked pressure elevation, this patient's cardiac output was within the normal range.

Case 6, H.T. A 27-year-old white man was admitted in November 1952 with melena and epigastric distress. The diagnosis of duodenal ulcer was established and pericardial calcification was demonstrated by x-ray, the latter having been present since 1948.

Cardiovascular complaints were elicited only on specific questioning and consisted of vague breathlessness on effort since September 1951.

Physical examination revealed dilated neck veins and moderate hepatomegaly. Cardiac examination was normal. The electrocardiogram showed nonspecific T-wave changes.
CONSTRUCTIVE PERICARDITIS

Right heart catheterization (table 1, H.T.) revealed a low cardiac index and moderate elevation of right atrial, right ventricular end-diastolic, and pulmonary artery pressures.

Pericardectomy was performed in April 1953. A thick calcified pericardium with obliteration of the pericardial space was found. Postoperative recovery was excellent. Right heart catheterization 19 months postoperatively revealed a normal cardiac index and normal pressures at rest with moderate elevation of the right atrial pressure during exercise.

Comment. This case illustrates that significant cardiac pressure elevations and reduced cardiac output may occur before the patient is aware of a limitation of activity.

DISCUSSION

The congestive state in constritive pericarditis is usually considered secondary to mechanical interference with ventricular diastolic filling resulting in reduction in stroke volume and cardiac output. Postmortem findings have also implicated myocardial atrophy and hepatic and pulmonary phlebosclerosis occurring after prolonged pericardial constriction. These mechanisms explain the slowly progressive course of constritive pericarditis, but do not explain the sudden reversibility of episodes of congestion occasionally observed. Harvey and co-workers implicated myocardial insufficiency and hypervolemia as causes of this reversibility.

An additional mechanism is illustrated by the findings at surgery in the cases R.B. and G.E. In these cases the formation of relatively small amounts of fluid within a rigid pericardial space with marked reduction in the diastolic volume would account for the rapid onset of congestive symptoms. With resorption of the fluid the patient may become asymptomatic. This fluid might be the result of an exacerbation of the original infection or a fresh pericardial insult. The presence of loculated fluid, limited by a rigid pericardium, has been described at surgery in other cases of constritive pericarditis. However, the role of this fluid in production or the reversibility of the congestive state was not commented upon.

The remaining 4 cases had complete obliteration of the pericardial space, and presented the classical features of constritive pericarditis. From our serial studies and reported hemodynamic studies of others, a pattern of the course of constritive pericarditis may be constructed: 1. An initial phase of pericardial insult as a result of infection or trauma, may be followed in months or years by pericardial constriction (second phase). 2. Early in the second phase, the constriction is of such mild degree that intracardiac pressures are normal at rest, and elevated only with exercise (case 4, R.Ro.). As constriction progresses, intracardiac pressures rise until they are abnormal at rest. Symptoms may be absent or minimal although pressures are distinctly elevated, as demonstrated by venous pressures or cardiac catheterization (case 4, R.Ro., and case 6, H.T.). Similar cases have been described in other studies. The third phase is one of obvious symptoms and usually occurs relatively late in the course of the disease. The pressures are regularly elevated but other factors must be active in the production of the congestive state, since similar pressures may exist in different patients with one relatively asymptomatic and the other moderately incapacitated. (case 6, H.T., case 3, G.U.) The duration of the cardiac constriction, as well as the severity, appears to play a role in the production of symptoms.

SUMMARY

Hemodynamic studies were correlated with the clinical course of constritive pericarditis in 6 patients. In 2 patients, with reversible congestive phenomena fluid accumulation and resorption in a rigid pericardial space is offered as an explanation for this reversibility.

The contrasting progressive course of classic, chronic, constritive pericarditis is demonstrated in 4 patients.

The discrepancy between the symptoms and the height of intracardiac pressures is discussed.
ACKNOWLEDGMENT

The authors wish to acknowledge gratefully the assistance given them in the course of this investigation by Drs. Howard Correll and Ross C. Kory.

SUMMARIO IN INTERLINGUA

Studios hemodynamic esseva correlationate con le curso clinic de pericarditis constrictive in un serie de 6 patientes. In 2, le reversibilitate del phenomenos congestive es explicate per le resorption del fluido in un rigide spatio pericardial. Le 4 altere patientes monstava per contrasto le curso progressive de classic constrictive pericarditis chronic.

Le discrepantia inter symptomas e nivello del pressiones intracardiac es discutite.

REFERENCES


Cheyne-Stokes Respiration. A form of respiratory distress, peculiar to this affection, consisting of a period of apparently perfect apnoea, succeeded by feeble and short inspirations, which gradually increase in strength and depth until the respiratory act is carried to the highest pitch of which it seems capable, when the respirations, pursuing a descending scale, regularly diminish until the commencement of another apnoeal period. During the height of the paroxysm the vesicular murmur becomes intensely puerile.—William Stokes. The Diseases of the Heart and the Aorta. Dublin. 1854.
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