Right Auricular and Ventricular Pressure Patterns in Constrictive Pericarditis

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The characteristic intracardiac pressure patterns of four patients with constrictive pericarditis are described. The significance of a high ratio between right ventricular end-diastolic and systolic pressure is demonstrated. Postoperative changes are described in one patient and the mechanism of the production of the pressure patterns is discussed.

Pressure patterns from the right auricle and ventricle in constrictive pericarditis were first described in 1946 by Bloomfield and associates1 and recently by Hansen and associates2 and McKusick.3 The right auricular pressure curve consists of (a) an M or W shaped pattern with two upward and two downward deflections, (b) moderately or markedly elevated mean pressure and (c) failure of the downward deflections to reach the baseline. The right ventricular pressure curve consists of (a) a slightly elevated systolic pressure and (b) a rapid diastolic dip followed by a high diastolic plateau. These pressure patterns were considered diagnostic of constrictive pericarditis and disappeared in one case following successful pericardiectomy.2 In the series of cases of constrictive pericarditis reported by McKusick,3 no postoperative pressure tracings are available in patients who had obtained significant clinical improvement. In 1948 Wood and associates4 thought that the pressure curves obtained from the right ventricle in one of their patients with constrictive pericarditis might be due to artifact but Hansen and associates and McKusick contended that the pressure patterns are characteristic of this disorder.

Clinical differentiation between constrictive pericarditis and other simulating conditions may be difficult. A correct diagnosis is of paramount importance, since constrictive pericarditis can be treated surgically; therefore, demonstration of characteristic pressure patterns by cardiac catheterization in constrictive pericarditis may prove an important diagnostic adjunct.

It is the purpose of this report (a) to describe and emphasize the various features of the pressure patterns in four patients with constrictive pericarditis and (b) to show the difference between preoperative and postoperative pressure curves in one patient. The significance of the ratio between right ventricular end-diastolic and systolic pressures is emphasized.

Method and Material

Cardiac catheterizations were performed as usual. The pressure tracings were recorded using a Statham strain gage. A Sanborn strain gage amplifier in a multichannel direct writing oscillograph* was used in most instances. The records also included a simultaneous registration of the pneumogram and electrocardiogram.

All pressures were recorded in millimeters of mercury above atmospheric pressure. The reference point for each patient was 6.5 cm. below the angle of Louis. The ventricular pressure tracings were measured for systolic and end-diastolic values in each heart beat during two respiratory cycles and the results averaged. The right ventricular and auricular mean pressures were determined by planimetric integration. The upper limits of the various pressures in normal subjects are as follows: right ventricular systolic pressure, 30 mm. Hg; right

* Sanborn Poly-Viso Cardilette.
ventricular end-diastolic pressure, 5 mm. Hg; and mean right auricular pressure, 5 mm. Hg. Four patients with chronic constrictive pericarditis were studied. Pericardiectomy was performed on two patients and postoperative pressure curves were obtained in one of these.

**Results**

**Case Report**

**Case 1.** C. T. This 20 year old man was admitted to the hospital in January, 1948 complaining of aching in the anterior chest, swelling of the abdomen, and ankle edema offour months duration. He had suffered a crush injury of the chest 10 months previously. The jugular veins were slightly distended. The heart was normal in rhythm and no murmurs were audible. The blood pressure was 100/72. The liver edge was palpable 4 cm. below the costal margin and there were signs of free fluid in the peritoneal cavity. Fluoroscopy of the chest showed slight enlargement of both left and right ventricles. The pulsations of the heart were diminished. Venous pressure was 330 mm. of saline. Electrocardiogram showed sinus tachycardia with low voltage and inverted T waves in leads I, II, III, and CF4. Circulation time from arm to lung (ether) was 40 seconds and from arm to mouth (Macasol) was 46 seconds.

The right auricular pressure curve obtained by cardiac catheterization showed an M or W shaped pattern with a mean value of 16 mm. Hg without respiratory variation (fig. 1). Unfortunately, the tip of the catheter could not be introduced into the right ventricle.

A clinical diagnosis of constrictive pericarditis was made and the patient was operated on in June, 1948. The pericardium was pale, thickened and fibrous, and visible pulsations were minimal. The pericardium and epicardium were removed from the anterior surface of both ventricles, laterally to the phrenic nerve and medially well around the right ventricle. The apex was completely freed. As the thickened cardiac envelope was relieved the heart action became more forceful. Since the operation the patient has improved and has returned to a full work program. The venous pressure fell to 130 mm. of saline. In March, 1952 he was well and cardiac catheterization was attempted twice unsuccessfully.

**Case 2.** M. F. This 61 year old woman was first seen in the Medical Out-Patient clinic in December, 1948 with complaints of exertional dyspnea and swelling of the legs and abdomen. She had had a history of rheumatic fever in childhood. The pertinent findings were: elevated venous pressure, moist rales over both lung bases, apical systolic and diastolic murmurs, enlarged liver and spleen, and ascites and edema. The working diagnosis was rheumatic heart disease with mitral stenosis and insufficiency associated with congestive heart failure. During the following three years she was treated with bed rest, digitalis preparations, mercurial diuretics, low-salt diet, and, most recently, ion exchange resins. In spite of this regimen her edema was still marked. In November, 1951 the patient was restudied. Physical examination revealed distended jugular veins, massive edema of the legs, and cardiac enlargement both to the left and right. She had auricular fibrillation without a pulse deficit. There was a loud systolic murmur over the precordium, loudest at the apex. A diastolic click was suspected by two observers. The pulmonic second sound was accentuated. Moist rales were present at both lung bases, and there was moderate ascites. Roentgenograms and fluoroscopy of the chest showed enlargement of the right auricle but not of the left, and extensive calcification of the pericardium. Cardiac catheterization revealed a right ventricular pressure pattern characteristic of constrictive pericarditis with a rapid diastolic dip followed by a high diastolic plateau (fig 2). The systolic and end-diastolic pressures were 52 and 18 mm. Hg respectively. The right auricular pressure showed two upward and two downward deflections with a mean pressure of 21 mm. Hg. The downward deflections did not touch the baseline and the second downward deflection coincided with the ventricular diastolic dip. An intrauricular septal defect was also demonstrated.

**Case 3.** H. A. This 54 year old man was admitted in August, 1951 with symptoms of malaise, fatigue, anorexia, weight loss, exertional dyspnea and cough for more than two weeks. He had always enjoyed good health until the present illness. The patient was febrile and toxic with distended jugular and thoracic veins. Expiratory rales were heard at both lung bases posteriorly. The heart sounds were distant and there was an apical systolic murmur as well as
a precordial friction rub. The blood pressure was 92/70. The liver was 3 cm. below the costal margin by percussion. Roentgenograms and fluoroscopy of the chest revealed an enlarged cardiac shadow with diminished heart pulsations. Electrocardiograms showed auricular fibrillation on admission which spontaneously converted to sinus tachycardia on the following day. In addition, the voltage was low and the T waves in leads over the left precordium were flat. A tentative diagnosis of pericarditis, probably of tuberculosis etiology, was made. While in the hospital the patient developed bilateral hydrothorax requiring numerous thoracenteses on the right. The administration of Aureomycin, streptomycin and para-aminosalicylic acid failed to alter the course of his illness.

The venous pressure continued to increase and the patient died three months after admission. Post-mortem examination revealed chronic constrictive pericarditis associated with acute tuberculous pericarditis and hydropericardium.

Case 4. R. W. This 58 year old man was admitted in October, 1951 with a history of weakness and fatigue for one year and ankle swelling for about five weeks prior to admission. In the past five years the patient had had periodic swelling of the ankles without chest pain, cough, dyspnea, or orthopnea.

There was pronounced edema of the legs and distended jugular veins. Examination of the lungs showed some dullness over the left base posteriorly, with diminished breath sounds. The heart was slightly enlarged to percussion. The lateral border was just outside the midclavicular line in the fifth intercostal space. The rhythm was regular and the sounds were of good quality. No murmurs were heard. The abdomen was distended with signs of free fluid. The liver was palpable about 4 cm. below the costal margin.

An electrocardiogram showed low voltage and inverted T waves in the left precordial leads compatible with chronic pericarditis. Because of the presence of a QS wave in leads V1-V4 the possibility of an old anteroseptal infarct was suspected. Venous pressure was 320 mm. of saline. Fluoroscopy of the chest showed some enlargement of the cardiac shadow, the configuration of which did not change significantly with changing position. However, there was diminished amplitude of pulsation. A moderate amount of fluid blunted the left costophrenic angle.

The differential diagnosis between constrictive pericarditis and coronary artery disease with chronic
Congestive failure was not clear cut in this case. Cardiac catheterization showed a right ventricle pressure pattern with a characteristic diastolic dip followed by a high diastolic plateau. The right ventricular systolic pressure was 36 mm Hg and the end-diastolic pressure 21 mm Hg. Because of the typical pressure patterns a diagnosis of constrictive pericarditis was made and the patient was prepared for pericardectomy with mercurial diuretics and thoracentesis.

At the time of pericardectomy there was marked thickening of the pericardium through which the pulsations of the heart were barely transmitted. This was particularly true over the region of the left ventricle. The pericardium was incised and about 300 cc. of fluid under pressure was obtained after which the excursion of the ventricle improved. The pericardium over the right ventricle was then incised and no free fluid was found. The pericardium was dissected free from the right side of the heart up to the auriculovenous septum and towards the right side until both the inferior and superior venae cavae could adequately be visualized. There was no evidence of constriction around the venae cavae. Decortication around the left ventricle was more difficult. In some areas there was marked calcification and much of the thickened epicardium had to be removed by sharp dissection. The left ventricle was decorticated laterally to the phrenic nerve and superiorly to the auricle. The apex was freed from the diaphragm. The pulsation of the heart then increased markedly and the apparent increased filling was indicated by the bulging of the heart during the diastole. Pathologic examination of the pericardium showed fibrosis and chronic inflammation. The day after operation the venous pressure had dropped to 130 mm. of saline and on the tenth postoperative day it was 100 mm. of saline.

Four months later the patient returned for follow-up studies. Physical examination revealed no abnormalities of the heart, lungs, or abdomen. A 12-lead electrocardiogram was not different from that taken preoperatively. A chest film showed a definite decrease in the size of the cardiac silhouette and the lung fields were clear.

Another cardiac catheterization was performed and the pressure tracings from the right heart showed distinct changes in comparison with the curves obtained before operation (fig. 4). In the right ventricle there was no longer a distinct diastolic dip followed by a high diastolic plateau. Instead, the pressure at the end of systole reached the baseline and gradually rose during diastole to a level of about 8 mm. Hg. The right ventricular systolic pressure was 39 mm. Hg. The right auricular pressure curve still retained the M shaped pattern but the mean pressure was reduced to 5 mm. Hg. In addition, the downward deflections touched the baseline and definite respiratory variations were observed.

The ratio between the right ventricular end-diastolic and systolic pressures in patients with constrictive pericarditis has not been previously described. The right ventricular systolic pressure may markedly increase in certain cardiopulmonary diseases, such as pulmonary stenosis, severe mitral stenosis, and chronic pulmonary diseases. However, the right ventricular end-diastolic pressure usually remains within normal limits unless right ventricular failure is also present. In the latter condition, both right ventricular systolic and end-diastolic pressures may be abnormally high, but in our experience the ratio between the end-diastolic and systolic pressures is always less than one-third.

The significance of this ratio in constrictive pericarditis is shown in figure 5. The end-diastolic to systolic pressure ratio was always less than one-third in 132 patients with various cardiopulmonary diseases other than constrictive pericarditis studied in this laboratory. However, the ratio was more than one third in 15 patients with constrictive pericarditis.
where measurements of the pressure were made or could be obtained (including 12 patients reported by other investigators). In six patients following pericardiectomy (including five patients reported in the literature and one of our own cases) the ratio is below one-third with the exception of one patient in whom the cardiac catheterization was performed only 12 days after pericardiectomy.

**Discussion**

The preoperative pressure patterns of the right auricular and ventricle presented in this report agree with those described by other investigators.\(^1\) Hansen and associates\(^2\) noted disappearance of the characteristic pattern of the pressure curve in patients following successful pericardiectomy. Similar change was also observed in one of our cases (case 4).

We believe that the recording of the right auricular and ventricular pressure curves may be a very useful diagnostic aid in constrictive pericarditis. We agree with Hansen and coworkers\(^2\) and McKusick\(^2\) that the pressure patterns are characteristic of this condition. However, the right ventricular pressure pattern is not pathognomonic for constrictive pericarditis since similar patterns have been observed in patients with right ventricular failure and myocardial fibrosis.\(^5\)\(^,\)\(^6\)

The mechanism of the characteristic right auricular and ventricular pressure patterns in constrictive pericarditis has been discussed by Hansen and associates and McKusick.\(^5\)\(^,\)\(^6\) These are caused mainly by impaired diastolic filling of the right ventricle. The right ventricle is almost completely empty immediately following the systolic ejection. The resulting drop in pressure constitutes the “diastolic dip” which is actually more apparent than real. In the absence of a diastolic plateau, this “dip” is a normal phenomenon, and touches or falls below the baseline. Both limitation of ventricular distension by the constricted pericardium and the high right auricular pressure cause the right ventricle to fill to its maximum capacity. Therefore, the “diastolic dip” usually does not touch the baseline but suddenly rises to a high diastolic plateau until it reaches the end-diastolic pressure.

The end-diastolic pressure in the right ventricle is exceedingly high, so that the mean right auricular pressure has to be high in order to maintain a positive gradient between these two chambers. As the right ventricle fills up rapidly the drop in the auricular pressure is relatively insignificant and of short duration. This explains why the upward deflections of the right auricular pressure are high and the downward deflections fail to reach the baseline. The second downward deflection of the right auricular pressure almost coincides with the ventricular diastolic dip in time, shape, and amplitude. Therefore, the change of the pressure pattern in the right ventricle is primary, and that in the right auricle is secondary.

The ratio between the right ventricle end-diastolic and systolic pressures may be useful in differentiating constrictive pericarditis from other conditions which give similar right auricular and ventricular pressure patterns. It is our experience that this ratio is not above one third in conditions other than constrictive pericarditis. Elevation of the end-diastolic pressure is associated with a corresponding rise of the systolic pressure in other cardiopulmonary diseases. On the other hand the right ventricular systolic pressure is only slightly increased in a typical case of constrictive pericarditis and the end-diastolic pressure is markedly elevated; furthermore, the ratio

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**Fig. 5.** This graph shows the ratio between right ventricular systolic and end-diastolic pressures in 132 patients with various cardiopulmonary diseases other than constrictive pericarditis, in 15 patients with constrictive pericarditis, and in six patients with constrictive pericarditis following pericardiectomy.
between the end-diastolic and systolic pressures is always more than one third.

Striking changes occur in the right ventricular and auricular pressure patterns of patients with constrictive pericarditis after successful pericardiectomy. The "diastolic dip" in the right ventricular pressure curve becomes less prominent, largely due to more adequate filling of the right ventricle. The diastolic plateau almost disappears and the end-diastolic pressure is much lower. The right auricular pressure curve may retain an M shaped pattern but the mean pressure returns to normal value. The downward deflections touch the baseline and respiratory variation appears in the pressure curves. It is reasonable to assume that if the pericardiectomy is not satisfactory, the preoperative pressure pattern may be retained. Therefore, the changes in the right auricular and ventricular pressure patterns may be useful in evaluating the results of pericardiectomy.

**Summary**

1. The right auricular and ventricular pressure patterns in four patients with constrictive pericarditis are described.

2. The right auricular pressure is markedly elevated and shows an M or W shaped pattern with two upward and two downward deflections. The downward deflections do not touch the baseline and the pressure curve shows no respiratory variation.

3. The right ventricular pressure curve consists of (a) a slightly elevated systolic pressure, (b) a rapid "diastolic dip" followed by a high diastolic plateau and end-diastolic pressure and (c) an end-diastolic to systolic pressure ratio of more than one-third. The significance of this ratio is emphasized. This ratio may help to distinguish constrictive pericarditis from simulating conditions where a high end-diastolic pressure may be recorded.

4. The pressure patterns of one patient show distinct changes following successful pericardiectomy.

5. The mechanism of the production of the pressure patterns is discussed.

**Acknowledgments**

The authors wish to express their thanks and appreciation for the secretarial aid of Mrs. Julia Gooding and the technical assistance of Miss Carol Gouverneur and Mr. S. John Vernarelli.

**Sumario Español**

Las presiones intracardíacas de cuatro pacientes con pericarditis restrictiva son descritas. El significado de la proporción alta entre la presión intraventricular derecha al final de diástole y la presión sistólica es demostrado. Cambios postoperatorios se describen en un paciente y el mecanismo de la producción de estas presiones se discute.

**References**


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Circulation. 1953;7:102-107
doi: 10.1161/01.CIR.7.1.102

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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the World Wide Web at:
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