Pressure Curves from the Right Auricle and the Right Ventricle in Chronic Constrictive Pericarditis

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Characteristic patterns of the pressure curves from the right auricle and the right ventricle have been found in chronic constrictive pericarditis by various investigators. It has been uncertain, however, whether or not the findings were pathognomonic, and whether they were reliable pictures of the pressure conditions or artefacts. Consequently the views upon the underlying mechanism have been different too. It is concluded from the studies here presented that the pressure curves are pathognomonic when in their most pronounced form, and that they are not artefacts and thus furnish a proper basis for inferences as to the hemodynamics in chronic constrictive pericarditis.

PRESSURE recording from the accessible sections of the heart and great vessels by means of the catheterization technic has proved an important aid in the diagnosis of different types of congenital heart disease. Generally speaking the same is not the case in acquired heart diseases. However, as a deviation from that rule, chronic constrictive pericarditis seems to give rise to a very characteristic pattern of the pressure curves from the right auricle and the right ventricle.

Earlier Publications

Pressure curves from a case of constrictive pericarditis from Cournand’s laboratory were published by Bloomfield and associates in 1946. The peculiarities of the tracings as compared with normal findings were emphasized. Wood and co-workers at the Mayo Foundation have also published pressure recordings from a patient suffering from constrictive pericarditis. The unusual tracings from the ventricle were, however, explained as artefacts resulting from too low a natural frequency of the pressure recorder. Recently, Eliasch, Lagerlöf and Werko have demonstrated the characteristic pattern of the pressure curves in 3 cases of adhesive pericarditis.

In an earlier publication we presented briefly our experiences with constrictive pericarditis and mentioned the probable diagnostic value of the pressure recording from the right auricle and the right ventricle in such cases. Since then we have had an opportunity to make some additional observations which may serve to elucidate the functional basis of the characteristic findings.

Methods and Material

The pressure recordings were the essential part of the studies. They were made by means of an electric condenser manometer constructed by one of us.

Its dynamic properties when connected to the heart catheter were known and adapted so that artefacts of consequence for the present studies could be excluded. The midaxillary line was chosen as zero line. The electrocardiogram was recorded simultaneously. In some cases the phonocardiogram was recorded at the same time as the pressure measurement. The time marking interval was 0.1 second, subdivided in some of the tracings into 0.02 second intervals. Ordinary x-ray examination and x-ray kymograms of the heart were made in all cases.

Our material comprises 6 clinically classic cases of chronic constrictive pericarditis (P. M. J. f. 155/47; M. C. f. 951/48; G. L. K. f. Vp. 714/48; C. A. S. m. 411/49; S. G. m. 27-10-10/49; N. C. L. m. 1118/49). One of them (N. C. L. m. 1118/49) was operated upon and examined postoperatively also. A seventh patient (L. S. P. f. 1112/49) had presented a typical syndrome before operation which was performed prior to the present studies. Only postoperative studies were made in this patient. In one additional case (P. S. m. 860/48) the diagnosis was suspected but could not be made with certainty on the basis of the results obtained by means of
ordinary methods of examination. To these cases is added for comparison one case (E. A. m. 547/49) of effusive pericarditis.

**Results**

The pressure recordings from the 6 typical cases showed quite similar patterns and can be satisfactorily represented by the curves shown in figure 1. They were obtained from a 45 year old woman (G. L. K. f. Vp. 714/48) who had had symptoms for at least five years. There were engorged veins, enlarged liver, moderate ascites, fairly marked edema of the legs and exertional dyspnea. At the left sternal border a pronounced pericardial click was audible. X-ray examination showed the heart to be only slightly enlarged (ratio 16:30), and to a wide extent covered by a solidly calcified pericardium. Nonetheless the part of the border of the heart represented by the left ventricle showed almost normal movements throughout the heart cycle. The movements of the right auricular and right ventricular boundaries, however, were much restricted.

The auricular pressure curve is characterized by a higher maximum pressure than the normal one and by equal heights of the two plateau-like maxima. The minimum pressure (corresponding to the minimum in the ventricle) does not reach the zero level as is the case normally. The mean pressure is, therefore, relatively more elevated than is the maximum pressure. The electrocardiogram shows that auricular fibrillation is present so that the form of the curve must largely depend on other factors than the contractions of the auricle.

The ventricular pressure curve shows a maximum pressure which is slightly elevated but not definitely outside the normal range. The descending part of the curve runs a normal course except that the pressure does not fully reach the zero line even though it comes more closely to it than does the auricular minimum pressure. Having reached its minimum, however, the pattern differs very markedly from normal findings, as the pressure rises steeply to a level roughly midway between maximum and minimum pressure where it forms a plateau which is not changed until the onset of systole. In the presented case there are very pronounced pressure oscillations located in the first part of the diastolic plateau. Their frequency is not high enough for them to be audible. The most conspicuous finding, the diastolic dip, may be found in other cases, even in normal ones, but is never of a relative magnitude comparable with that found in constrictive pericarditis, as far as could be concluded from the present material.*

That the diastolic dip is not a consequence of an elevated venous and auricular pressure is well known from measurements in other types of congestive heart failure. This is illustrated in figure 2 (J. P. O. m. 928/49). The auricular pressure is very elevated, yet the

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*Recently we have observed a case in which a fairly marked diastolic dip was present, though the minimum pressure was far from reaching the zero line. On autopsy a chronic myocarditis with an unusually widespread and high degree of fibrosis was found.*
tracing shows no M-form. The ventricular diastolic dip is only vaguely indicated. The diastolic pressure is much higher than normal.

Our experiences with other forms of pericarditis are limited to one case of tuberculous, exudative pericarditis. The ventricular curve (shown in figure 3) differs from both the normal pressure curve and that found in chronic constrictive pericarditis. Most conspicuous seems to be the marked diastolic oscillations in conjunction with a steadily rising pressure throughout diastole.

The Effect of Operation on the Pressure Curve in the Right Auricle and Ventricle

The direct proof of the causal relation between the pericardial constriction and the pressure curves presented here as being characteristic is given in the case that was examined before and after pericardiectomy. The patient was a 21 year old man (N. C. L. m. 1118/49) with a typical syndrome. There was no systolic retraction of the ictus which was palpable in the fourth intercostal space. A marked pericardial click was heard at the left sternal bor-

Fig. 2. Upper tracing: auricular pressure curve. Lower tracing: ventricular pressure curve. The electrocardiogram is the lower curve in both tracings. In the lower tracing phonocardiogram and femoral arterial pulse curve are also shown. From a patient (J. P. O. m. 928/49) suffering from cardiac failure with elevated venous pressure but no pericardial constriction. It is noted that the auricular pressure and the diastolic ventricular pressure are at a high level. No “constriction pattern” is visible. ECG shows auricular fibrillation.
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operation, recovered completely clinically and returned to normal life, showing no restricted physical activity whatsoever.

Figure 4 shows the same type of auricular and ventricular pressure curve as in the first case of constrictive pericarditis (fig. 1). The tracings from the control examination one year after the operation (fig. 5) show that the dip has completely disappeared and that the pressure curves are practically normal. Concur-

mm. Only the sixth rib was resected. The patient, who was in poor condition before the

Fig. 3. Ventricular curve from a 25 year old male patient (E.A. m. 547/49) with a tuberculous effusive pericarditis. ECG at bottom of the tracing. Note the very marked oscillations in diastole. No diastolic plateau.

Fig. 4. Upper tracing: auricular curve. Lower tracing: ventricular curve. Only a portion of the electrocardiogram is shown in the upper tracing. From a 21 year old male (N.C.L. m. 1118/49) with typical chronic constrictive pericarditis. The pressure record was obtained before operation and shows the typical "constriction pattern."

rently with the change of the pressure curve the kymographic picture had shifted to a picture which could not be distinguished from a normal one. As was the case before the operation fluoroscopy showed little or no movements of the heart on changing the position of the body.

Fig. 5. Upper tracing: auricular curve. Lower tracing: ventricular curve. From the same patient illustrated in figure 4 but recorded 12 months after successful pericardectomy. The typical "constriction pattern" has disappeared.

In the case that was only examined postoperatively the tracings show normal or almost normal configuration. The patient was a 25 year old female (L. S. P. f. 1112/49) on whom
pericardiectomy was performed 11 years earlier. Clinical and roentgenologic signs almost completely disappeared soon after the operation. She was admitted to the hospital because of ascites and pleural effusion. She was febrile with widely fluctuating temperature. Her symptoms had reappeared rather acutely. The pressure curve from the right ventricle is shown in figure 6. Although not entirely normal it does not display the characteristicsof a constriction curve. It is notable that the minimumpressure reaches the zero line. The auricular

maximum pressure was less than 10 mm. Hg. Thus the result of pressure recording did not support the diagnosis of renewed constriction of the heart as the reason for her effusions. Rather, together with the fever and the fact that she recovered on treatment with dihydrostreptomycin, it pointed towards a flare-up of her (tuberculous?) polyserositis.

**Discussion**

The consistent finding of characteristic right auricular and ventricular pressure curves in patients presenting the classic syndrome of chronic constrictive pericarditis suggests on a purely empiric basis that pressure measurements in uncertain cases may be of some diagnostic help. This has already been referred to above in a “negative” case, and is further illustrated by the curves in figure 7 from a case (P. S. m. 860/48) in which clinical and roent-

![Image](http://circ.ahajournals.org/)
served, however, prior to any attempt to correlate the peculiar pattern with the hemodynamics in chronic constrictive pericarditis.

Against the form of the pressure curves being due to artefacts are the following:

(1) The frequency response in the manometer-catheter system used in the present studies was suitable. Poor frequency response would not account for an elective occurrence of the curve pattern in constrictive pericarditis. (2) Bending and twisting of the catheter as the basis for such artefacts is not tenable for the same reasons as given in (2). (4) The fact that in constrictive pericarditis typical recordings are obtained on electrokymography and on ballistocardiography very strongly suggests that there is also a characteristic pressure curve. (5) The typical curve has been recorded directly by puncture of the right ventricle in one case during operation (see later comments).

We feel justified, therefore, in explaining the changes in the pressure curve as being due to essential factors governing the function of the constricted heart.

In accordance with the generally accepted concept expressed by Chevers in 1842 (quoted by White), we consider the hampered diastolic dilatation to be the main factor in the abnormal hemodynamics. This view is supported by the comparable effect of the normal pericardium as shown in heart-lung preparations and in animal experiments as well as by clinical experiments with patients suffering from chronic constrictive pericarditis.

The systolic contraction of the heart is in our opinion relatively unimpaired. That means that the (right) ventricle is emptied as completely as in normal cases or at least almost so, so that the static pressure at the end of systole drops to almost normal values. Hereby is created a steep pressure gradient between the auricle and the ventricle. As a result blood rushes from the auricle into the ventricle which rapidly reaches its maximum distention, which is limited by the constricted pericardium. This explains the sudden rise in pressure. At the time the pressure has reached the diastolic level again the diastolic filling has been completed. The ventricular systolic pressure seems, in the presence of irregular heart action, to be much less dependent upon the duration of the preceding diastole than usual, provided diastole lasts longer than the diastolic dip. As mentioned in the description of figure 1, the diastolic dip is followed by smaller oscillations. Although most conspicuous at the beginning of the diastolic level, they are detectable in the upstroke of the dip (fig. 4). They are presumably a result of the water-hammer-like action of the blood against the rigid wall of the heart. Their low frequency causes them to be inaudible, but it seems reasonable to believe that the protodiastolic gallop (pericardial click) which so often is heard in cases of chronic constrictive pericarditis is caused by the same phenomenon.

The pressure curve from the auricle reflects the events in the ventricle. The minimum pressure lags 0.01 to 0.02 second behind that of the ventricle.

In our explanation of the pressure curves in constrictive pericarditis we have not found it necessary to assume that an elastic recoil of the thickened pericardium and/or the thoracic wall plays any important part. Physically it is also difficult to imagine such an effect on the basis of surgical and pathologic observations. Moreover, recent studies in a case of constrictive pericarditis during surgical operation have shown that the early diastolic dip may be present under circumstances where no elastic effect of the pericardium and the thoracic wall is possible. The pressure curves that are shown in figure 8 were recorded by puncture of the heart wall with a 25 gauge needle directly connected with the manometer. This system has a natural frequency of about 90 cycles per second and is properly dampened.

Due to the difficulties of localizing the right ventricle before stripping the pericardium, only pressure curves from the left ventricle were obtained at this stage of the operation. After an adequate pericardietomy had been performed pressure curves were recorded from both ventricles. The right ventricular curve showed the typical constriction pattern al-

* We are indebted to Dr. Alfred Blalock, Department of Surgery, The Johns Hopkins Hospital, for permission to include this case in our material.
though the diastolic dip was a little broader than that found preoperatively on heart catheterization.

At first glance this result appears somewhat confusing; however, the persistence of the dip was in conformity with the direct observation that a sharp diastolic arrest was still present, resulting from paper-thin inelastic strands left on the surface of the heart. Postoperative studies by means of heart catheterization will show whether or not the ventricular pressure curve will revert to normal as in other cases. A gradual stretching of the remaining strands is not at all improbable. The fact that in some cases clinical recovery requires a certain time to be completed may suggest such a mechanism.

The interpretation of the pressure curves given above suggests that more than qualitative diagnostic information might be obtained from the appearance of a certain curve. A well pronounced diastolic dip almost reaching the zero line should indicate a remarkable degree of constriction in conjunction with a well functioning myocardium. In such cases a good result of pericardectomy should be expected, all other conditions being equal. If the diastolic dip is relatively less prominent and the pressure rise begins farther from the zero line we have reasons to assume that a certain degree of "myocardial insufficiency" has entered the picture. Such curves are predominantly found in cases of chronic constrictive pericarditis with large hearts.

**Summary**

1. The existence of characteristic patterns of the right auricular and the right ventricular

![Fig. 8. Pressure curves from left ventricle, right ventricle and pulmonary artery obtained by puncture (25 gage needle) during thoracotomy. Zero line = midaxillary line. Upper curves: left ventricle before and after stripping the heart. The systolic pressure is slightly higher after the pericardectomy. Lower left curve: right ventricle after removal of pericardium. The early diastolic dip is still present. (See text.) Lower right curve: pulmonary artery. The later part of diastole is on the same level, and reveals the same pattern, as the pressure curve of the right ventricle in the corresponding part of the heart cycle. An early diastolic dip is absent in the pulmonary curve in this particular case.](image-url)
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