Value of reference tracings in diagnosis and assessment of constrictive epi- and pericarditis

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Reference tracings are of great value in the diagnosis and assessment of constrictive pericarditis. The Q-h interval in the jugular venous pulse tracing is strongly correlated with the mean right atrial pressure (r = 0.91). The left ventricular ejection time, the Q-A2 interval, and the Q-h interval are independent during atrial fibrillation from the preceding diastolic filling interval. This differentiates constrictive pericarditis from valvular heart disease. Cases with haemodynamically significant constrictive epicarditis are characterized by a rapid evolution, absence of pericardial calcification and absence of an early diastolic filling sound, a dominant wave in the jugular venous pulse tracing, and a high early diastolic ventricular pressure. The haemodynamic behaviour is similar to that found in cases with myocardial fibrosis.

As a disease of the heart, constrictive pericarditis possesses several unique features. In most cases these are due to the presence of a nearly normal contractility of the myocardium in the presence of a highly raised filling pressure.

In the more severe cases, however, contractility of the myocardium is also impaired. Moreover, though the clinical signs of pericarditis constrictiva are well known (Kussmaul, 1873; Pick, 1896; White, 1944; Wood, 1956), its differential diagnosis from other fibrotic myocardial disease is often difficult (Gunnar et al., 1955; Nye, Lovejoy, and Yu, 1957; Soulé, Chiche, and Acar, 1958; Acar and Godeau, 1960; Shillingford and Somers, 1961; Somers et al., 1968). In constrictive pericarditis the epicardium can also be thickened and this complicates surgical treatment of the disease. The purpose of our study has been to evaluate the severity of constrictive pericarditis by means of indirect reference tracings. The possibility of diagnosing an eventual participation of the epicardium in the disease and the differential diagnosis with other forms of heart disease have also been examined.

Subjects and methods
During the period from 1960 to 1968, 26 cases of constrictive pericarditis have been studied. There were 18 men and 8 women. The mean age of the group studied was 27 years (range 16–68). In 16 cases the pericardium was calcified and in 10 cases no calcification was present. Of the 10 cases without calcification, 7 had a subacute and 2 had a chronic evolution towards constriction, while in 1 case the beginning of the disease could not be established with certainty. In the patients with a subacute evolution, the time interval between the beginning of the pericardial disease and the finding of constrictive pericarditis was less than 1 year. In 25 cases a complete clinical and haemodynamic evaluation has been performed, including a right heart catheterization in 24 cases and a right and left heart catheterization in 1 case. A level 5 cm below the angle of Louis has been considered as the zero level for the pressure measurements. Twenty cases have undergone operation, including all cases with a non-calcified constrictive pericarditis. In 7 cases during operation constrictive epicarditis was found to coexist with the pericardial disease. This epicardial thickening could be partially removed during operation in 5 cases, and was verified at necropsy in 2 cases. In all 7 cases the constrictive epicarditis was thought to be the major factor in the disease. Sinus rhythm was present in 21 cases, and 3 were seen during atrial fibrillation and 2 during fibrillo flutter.

Post-operative observation included a right heart catheterization in 4 patients. Phonomechanocardiographic reference tracings were recorded before operation in 25 cases, and in several cases after operation. This included a jugular venous pulse tracing, an indirect carotid artery tracing, and a phonocardiogram. The left ventricular ejection time was measured from the indirect carotid artery tracing. In some cases it was also possible to obtain an apex cardiogram. The reference tracings were obtained by means of a piezoelectric transducer with a time constant...
of 1·2 sec. or with a strain gauge transducer EMT 32 Elema with a DC output. It has been shown previously that a time constant of 1·2 sec. causes only an insignificant time shift when compared to a DC recording (Kesteloot, Willems, and van Vollenhoven, 1969).

The method of recording has been described elsewhere (Kesteloot, 1965). For the reference tracings the standard nomenclature was used (Hartman, 1960). The upstroke of the y trough has accordingly been referred to as the h wave. This corresponds to the z wave in the terminology used by Wood (1956).

For comparative purposes a certain number of cases of rheumatic valvular heart disease were also studied with the same methods as the cases of constrictive pericarditis.

Results

Jugular venous pulse tracing

Morphology

In the cases with sinus rhythm three types of jugular venous pulse tracing were encountered. Type A had a normal morphology, with the a wave as the dominant wave and an x trough or systolic descent larger than the y trough or diastolic descent. This type was found in cases with a normal or only slightly raised mean right atrial pressure. Type B was the form of jugular venous pulse tracing most commonly encountered in constrictive pericarditis. It had either a W or an M form, but the y trough was always deeper than the x trough. Type C was very similar to type A but the a wave tended to be higher and the y trough to be nearly absent. This type was found in the presence of a very high mean right atrial pressure (Fig. 1). The haemodynamic findings are summarized in Fig. 2. Type C was only found in cases where an important constrictive epicarditis was also present, and the disease was the most severe in this group. The pressure difference between the summit of the v wave in the right atrium

FIG. 1a The three types of jugular venous pulse tracing (see text). A great similarity exists between the morphology of type A and C.

FIG. 1b Another example of type B. The nadir of the y trough coincides with the early diastolic sound.

FIG. 2 Haemodynamic findings in the three subgroups, divided according to the morphology of the jugular venous pulse tracing and the height of the mean right atrial pressure. CO = cardiac output (in litres per minute). \( \bar{m}_{RAP} \) = mean right atrial pressure. \( \bar{m}_{EDPRV} \) = mean early diastolic pressure in the right ventricle. \( \bar{m}_{PvRA} \) = mean pressure of the summit of the v wave in the right atrium. \( \bar{m}_{dpRV} \) = mean end diastolic pressure in the right ventricle. \( \bar{m}_{PAP} \) = mean pressure in the pulmonary artery. \( \bar{m}_{PCP} \) = mean pulmonary capillary pressure. The pressures are given in mm. Hg.
and the nadir of the early diastolic pressure in the right ventricle was measured in all cases. This mean pressure difference was only about 3 mm Hg in cases of type A and C, and 13 mm Hg in cases of type B.

**Haemodynamic correlations**

The relation that exists between different time intervals measured between electrocardiographic, phonocardiographic, and mechanocardiographic events derived from the jugular venous pulse tracing and the mean right atrial pressure was examined. Only the cases with a mean right atrial pressure of 4 mm Hg or more were used for the calculations, as in cases with a lower pressure the constractive pericarditis could not be considered as clinically important. The best correlation was found between the mean right atrial pressure and the Q-h interval, i.e. the interval between the beginning of the depolarization measured from the electrocardiogram and the h wave measured on the jugular venous pulse tracing (Fig. 3). The h wave signals the end of the ventricular filling during diastole. As an h wave cannot always be identified on the venous pulse tracing in cases with a rapid heart rhythm, other intervals were also measured. The second best correlation was found between the mean right atrial pressure and the nadir of the y trough of the jugular venous pulse tracing (Fig. 4). The regression equations relating mean right atrial pressure to the different measured time intervals are given in Table 1.

We also examined the relation between the mean right atrial pressure and the time interval between the beginning of the pulmonary component of the second sound (P2) and the summit of the v wave of the jugular venous pulse tracing. A shortening of this interval has been previously described in constractive pericarditis (Kesteloot, 1963). In 11 out of 12 cases with an interval P2-summit v less than or equal to 0.03 sec., the mean right atrial pressure was higher than 12 mm Hg, and the reverse was true in 6 out of 7 cases with a P2-summit v interval >0.03 sec. ($\chi^2 = 11.9, p < 0.001$).

A remarkable constancy was found in the Q-h wave interval during atrial fibrillation, making it independent from variations in the duration of the preceding diastole (Fig. 5). The same conclusion can be drawn from the multiple regression equation between the mean right atrial pressure and the Q-h interval and heart rate. Only the regression coefficient of the Q-h interval is statistically significant (Table 2).

**Left ventricular ejection time** The regression line relating left ventricular ejection time to heart rate in cases of constractive pericarditis was about 22 msec. lower than the

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**FIG. 3** Relation between Q-h interval measured on the jugular venous pulse tracing and the mean right atrial pressure.

**FIG. 4** Relation between Q-y interval measured on the jugular venous pulse tracing and the mean right atrial pressure.

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**TABLE 1** Regression equation between the mean right atrial pressure in mm. Hg (Y) and the different time intervals in sec. (X), measured on the jugular venous pulse tracing

<table>
<thead>
<tr>
<th>Y</th>
<th>X</th>
<th>Regression equation</th>
<th>r</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>mRA pressure</td>
<td>Q-h</td>
<td>$Y = 45.90 - 48.75X$</td>
<td>-0.91</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Q-nadir y</td>
<td>$Y = 58.79 - 86.18X$</td>
<td>-0.88</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>summit v-w wave</td>
<td>$Y = 29.62 - 60.35X$</td>
<td>-0.84</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>P2-nadir y</td>
<td>$Y = 29.06 - 112.62X$</td>
<td>-0.83</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>summit v-nadir y</td>
<td>$Y = 30.00 - 124.14X$</td>
<td>-0.64</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>
regression line obtained in normal subjects (Fig. 6). In patients successfully operated upon, the left ventricular ejection time returns to normal. The regression line obtained in normal subjects has been published previously (Willems and Kesteloot, 1967). The left ventricular ejection time also is largely independent of the length of the preceding RR interval in subjects with constrictive pericarditis during atrial fibrillation (Fig. 7). This contrasts with the behaviour of the left ventricular ejection time during atrial fibrillation in cases with mitral disease and predominant mitral stenosis (Fig. 8). In 8 such cases the following linear regression equation was calculated between the left ventricular ejection time in msec. (Y) and the natural logarithm of the preceding RR interval in sec./100 (X): 

\[ Y = -157.68 + 93.96 \ln X \]  

\[ r = 0.845 \]  

\[ n = 65 \]

**Q-A2 interval** The behaviour of the Q-A2 interval was very similar to that of the left ventricular ejection time interval. In patients with constrictive pericarditis the value of the Q-A2 interval related to heart rate was usually smaller than in normal subjects, especially at higher heart rates. It also tended to return to normal values in patients who had been successfully operated upon (Fig. 9). The regression line obtained in normal subjects is mentioned in Fig. 9 and has been published elsewhere (Kesteloot, Willems, and Joossens, 1968). In patients with atrial fibrillation the Q-A2 interval showed little variation with heart rate (Fig. 10), whereas this was not the

**FIG. 5** The Q-h distance is nearly independent of the duration of the preceding RR interval during atrial fibrillation.

**FIG. 6** Relation between left ventricular ejection time and heart rate in patients with constrictive pericarditis. The ejection time returns to normal in the patients who have been successfully operated on.

**TABLE 2** Multiple regression line and multiple and partial correlation coefficients between the mean right atrial pressure in mm. Hg, the Q-h distance in sec., and the RR interval in sec.

<table>
<thead>
<tr>
<th></th>
<th>Regression Line</th>
<th>Correlation Coefficient</th>
<th>Partial Coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>PRA_m</td>
<td>( R^{2} = 0.915 ) (p &lt; 0.001)</td>
<td>( R = 0.915 ) (p &lt; 0.001)</td>
<td>( R = 0.915 ) (p &lt; 0.001)</td>
</tr>
<tr>
<td>RR</td>
<td>( R^{2} = 0.777 )</td>
<td>( R = 0.777 )</td>
<td>( R = 0.777 )</td>
</tr>
<tr>
<td>q-h</td>
<td>( R^{2} = 0.005 )</td>
<td>( R = 0.005 )</td>
<td>( R = 0.005 )</td>
</tr>
</tbody>
</table>

**FIG. 7** Relation between left ventricular ejection time (LVET) and RR interval during atrial fibrillation in constrictive pericarditis. The left ventricular ejection time remains constant or shows only little variation over a wide range of preceding RR intervals. Each point represents the mean of 5 measurements during closely related RR intervals. The different symbols used in Fig. 7, 8, 10, and 11 refer to different patients.
case in patients with mitral disease and predominant mitral stenosis (Fig. 11). In 7 patients with mitral disease and atrial fibrillation the following relation was found between the Q-A2 interval in msec. (Y) and the natural logarithm of the preceding RR interval in csec. (X): 

\[ Y = -26.93 + 84.605 \ln X \] 

\( r = 0.857, n = 85 \).

**Apex cardiogram** The apex cardiogram is usually negative in cases with constrictive pericarditis (Hartman, 1964) and this was also true in our cases. The end of the rapid protodiastolic upstroke of the apex cardiogram coincides with the loud third heart sound (T3) and marks the end of the rapid protodiastolic filling of the heart. In cases with atrial fibrillation, the Q-T3 interval was independent of the preceding RR interval (Fig. 12).

**Findings in constrictive peri-epicarditis**

In 7 of the 26 patients a haemodynamically important constrictive epicarditis coexisted with the constrictive pericarditis. As mentioned before, in 4 cases the jugular venous pulse tracing was of type C. In 3 cases, 1 of which was in atrial fibrillation, it was of type B. In all 7 cases the evolution to constriction had occurred in less than 1 year after the acute episode during which a pericardial effusion was often present. In none of the 7 cases was the pericardium calcified. In the cases with a calcified constrictive pericarditis, a protodiastolic dip was always present in the pressure curve of the right ventricle, and the early diastolic pressure in the right ventricle at the nadir of the dip was always less than 10 mm. Hg. In cases with coexisting constrictive epicarditis, however, the dip was either absent or the early diastolic pressure was more than 10 mm. Hg (p < 0.001).

**Discussion**

A dominant y trough in the jugular venous pulse tracing, known as the diastolic collapse of Friedreich (1855), is the most common finding in constrictive pericarditis, and this was confirmed in 10 out of 20 cases in sinus rhythm. During atrial fibrillation a dominant y trough is an obligatory finding. As could be expected, a nearly normal jugular venous pulse tracing was found in cases with only a small degree of constriction.

An interesting finding in our series was the existence of a dominant a wave with an x trough deeper than the y trough (type C) in very severe cases of constrictive pericarditis (Fig. 1). In these cases a third heart sound was constantly absent. As the third heart sound in constrictive pericarditis is normally thought to signal the end of the rapid filling of the ventricle by putting the pericardium under tension, the absence of such a sound would point to the absence of a rapid filling in protodiastole. This is confirmed by the absence of a deep y trough or the presence of a less steep y descent in the jugular venous pressure trace in cases with an absent third heart sound, as mentioned by Mounsey (1955). The presence of a dominant a wave can be explained by a low compliance of the right ventricle in diastole, and the same factor
could also explain the absence of a deep y trough (Kesteloot, 1963).

Constrictive epicarditis has been suggested as a possible explanation for this abnormal behaviour of the jugular venous pulse tracing in certain cases of constrictive pericarditis (Kesteloot, 1963), and could be the factor causing the reduced compliance of the right ventricle. The thickening of the epicardium would not permit a rapid dilatation of the myocardium in the early diastole. This was confirmed by the finding of either an absent dip-plateau wave form in the pressure tracing of the right ventricle or by finding a high early diastolic pressure in the right ventricle. The existence in these cases of only a small pressure difference between the summit of the v wave in the right atrium and the nadir of the early diastolic right ventricular pressure is also compatible with this explanation. Moreover, in all the cases where a jugular venous pulse tracing of type C was present, a haemodynamically important constrictive epicarditis was found during operation to coexist with the pericardial thickening. Similar cases where the x trough was deeper than the y trough have also been described by Gibson (1959) and Wood (1961). They point out that the x trough is very sharp in these cases, a fact that we are able to confirm. It is not easy, however, to formulate this sharpness in a quantitative way. Furthermore, it is not known whether constrictive epicarditis was also present in their cases. A dominant a wave with an x trough deeper than the y trough was also present in a case of constrictive peri-epicarditis published by Gibbons, Goldbloom, and Dobell (1965).

The presence of a dominant a wave in the jugular venous pulse or right atrial pressure tracing and of a high early diastolic pressure in the right ventricle is found in different forms of myocardial fibrosis. These features have been described as the best means to differentiate between this disease and constrictive pericarditis (Wood, 1961; Storstein, 1966; Somers et al., 1968). Higher early diastolic pressures in constrictive pericarditis than in myocardial fibrosis have, however, also been described (Nye et al., 1957). The present study shows that these signs are unreliable for the differential diagnosis between these different conditions when constrictive epicarditis is also present. This can be explained by the similar behaviour of the myocardium during diastole in myocardial fibrosis and constrictive epicarditis.

The Q-h interval in the jugular venous pulse tracing is shortened in constrictive pericarditis, due to the abrupt early end of ven-

![Fig. 10 Relation between Q-A2 and RR interval during atrial fibrillation in constrictive pericarditis. The Q-A2 interval remains constant over a wide range of preceding RR intervals. Each point represents the mean of 3 measurements during closely related RR intervals.](image)

![Fig. 11 Relation between Q-A2 and RR interval during atrial fibrillation in mitral disease. In cases with mitral disease and predominant mitral stenosis the Q-A2 interval varies directly with the length of the preceding RR interval.](image)
The correlation between the mean right atrial pressure and the Q-h wave is not due to the heart rate alone, as multiple regression analysis has shown (Table 2).

Moreover, the Q-h interval, the Q-A2 interval, and the left ventricular ejection time are remarkably independent of heart rate and remain nearly constant during atrial fibrillation in each individual case of constrictive pericarditis (Fig. 5, 7, 10). This could be expected because the filling of the heart is only possible to a constant end-diastolic volume, independent from the duration of diastole. The high venous pressure, moreover, permits a complete filling of the constricted ventricle even during a short diastole. This behaviour differentiates constrictive pericarditis from heart insufficiency due to valvular heart disease (Fig. 8, 11). In these cases a steep decrease in the mentioned time intervals occurs during a short diastolic interval. A further study of this problem in other cases of heart disease and especially of myocardial fibrosis will be of interest. Preliminary results show that in cases of congestive cardiomyopathy, the q-A2 interval and the left ventricular ejection time during atrial fibrillation vary with the length of the preceding RR interval in a similar manner as the cases of valvular heart disease studied above. Both the left ventricular ejection time and the Q-A2 interval are shortened in cases of constrictive pericarditis. This can be explained by a smaller stroke volume in this disease. The values return to normal in cases which have been successfully operated (Fig. 6, 9). The Q-h interval also returns to normal values in these subjects.

A negative apex cardiogram was found whenever it could be recorded, and this has also been found by Hartman (1964). In our opinion this is due to the negative pressure in the pericardial sac during systole, which causes the atmospheric pressure to push the thoracic wall inward at the place of the apex beat. During early diastole a pronounced rapid filling wave occurs, the end of which coincides with the third heart sound. The interval Q-end of the early diastolic ascent is also independent of the preceding diastolic interval (Fig. 12).

In 1951, Gonin, Froment, and Gravier described a peculiar form of pericarditis with a rapid evolution toward constriction and with haemodynamically important epicardial involvement. This has been confirmed by others (Soulé et al., 1958; Gibbons et al., 1965), and by the present study. In all our cases of coexisting constrictive epicarditis the interval between the acute episode and the diagnosis of constriction was less than one year.

From the study of our material it appears that the presence of a constrictive epicarditis is strongly suggested by: (1) a rapid evolution toward constriction of the disease in the absence of pericardial calcification; (2) a dominant a wave and a deep x trough in the jugular venous pressure trace; (3) an absent third heart sound; and (4) a high early diastolic right ventricular pressure.

A clinical differentiation between the two forms of constrictive heart disease with and without constrictive epicarditis thus becomes possible. This differentiation is important because the mortality at operation was much higher and the operation more difficult in the cases with constrictive epicarditis.

References


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