Acute pericarditis is an anatomic diagnosis with a greatly varied etiologic background. Generally the entity is classified upon an etiologic basis, an important approach for treatment and prognosis. Studies by many investigators have shown a two-fold difference between necropsy and clinical incidence of acute pericarditis. Obviously the classically described symptoms and signs for recognition of acute pericarditis are either not met or not elicited in a number of cases even if one allows for terminal episodes.

Some of the difficulties in diagnosis are brought out when one reclassifies patients, not into etiologic categories, but into two large groups based on the bedside problem in the patient:

1. Those in whom the pericarditis represents the predominating clinical picture, and
2. Those in whom pericarditis is a part of a general picture with predominating symptoms elsewhere.

Indeed in group 1, patients may have pericarditis secondary to some general process but if so, the symptomatic picture is outstandingly pericarditic.

The objective of the present report is to emphasize the importance of acute pericarditis as a diagnostic tool in the recognition and treatment of disease other than pericarditis itself. However to evaluate its recognition for this purpose the reliability of the diagnostic signs of acute pericarditis itself must be established. This has been done in a previous report. Acute nonspecific (idiopathic) pericarditis was chosen for this purpose because there is generally little else in the patient to draw attention away from this single process. It occurs in young adults in whom other unrelated changes, for example in the electrocardiogram, are less likely to interfere with interpretation; its onset is often clearly signalled, especially by sudden pain, giving us a definitive point of reference in the evolution of the disease and the friction rub is generally well-developed. Analysis of the clinical records in patients in this group indicated clearly that the development and course of the diagnostic findings are sufficiently variable to account for many errors in diagnosis.

Table 1 shows, in 38 instances of our group observed by the same personnel, the frequency with which the various diagnostic findings were elicited from the time of admission through the active course of the disease. Note that the incidence of findings diagnostic of pericarditis is far from the level at which any one alone could be depended upon uniformly. There was a lapse of 24 hours to five days from the onset of symptoms until observations were started. To be sure the figures
would be different if all patients were followed from the beginning of symptomatology, a variable accountable for part of the results shown in this table. Specific cases point this out clearly. For example, one patient, awakened at night with severe chest pain representing the clinical onset of her disease, was seen promptly within an hour by one of the staff men. He heard a friction rub and recommended admission to the hospital. The next morning the friction rub could not be heard and electrocardiograms for the next two days were normal, to be followed on the third day by recurrence of the friction and typical electrocardiograms. In another such instance electrocardiograms never became positive and friction did not occur. Variability in the timing of observations and rapid changes account for the low incidence of some of the findings in Table 1 and stress the importance of sequential examination.

It may be seen in Table 1 that diagnostic electrocardiographic changes were observed only two-thirds of the time. Often there were nonspecific abnormalities. The electrocardiographic findings are reminiscent of those of Bellet and McMillan in 1938. In 57 instances of acute pericarditis of different types they found electrocardiographic changes in 80 percent but characteristic ST segment changes in only 37 percent. If sequential examination is started after electrocardiograms have reached a positive but nondiagnostic pattern, and in the absence of friction, diagnosis may have to be made upon the general course without elicitation of pathognomonic signs and be helped in support by nonpathognomonic roentgenographic signs such as transient changes in heart size. Rapid increase in heart size with reversion to normal is such evidence, and Table 1 shows that cardiac enlargement occurred about as frequently as friction. Without friction this enlargement, especially when transient, is important in diagnosis. Like the electrocardiographic findings the roentgenogram of the heart is frequently abnormal but not diagnostic. In addition small effusions, under 300 ml, may not distort the cardiac contour at all. Large effusions may produce the pearshaped shadows frequently described and be accompanied by changing contour with changing posture. However, a large flabby heart may produce similar changes. In such doubtful instances correlation of the findings with other data which alone are not diagnostic may lead to proper final diagnosis.

The findings utilized in the diagnosis of acute pericarditis, therefore, are sufficiently irregular to pose problems in recognition even under most ideal clinical circumstances. The variable appearance and disappearance of friction and the inconstancy of and rapid evolution of the electrocardiographic and roentgenographic patterns make serial determinations important. Nonspecific abnormalities in the electrocardiogram or chest roentgenogram, when friction is not elicited, magnify the importance of the chronologic development of diagnostic data, and are often the most important leads in diagnosis.

The above findings compound the problem in the use of acute pericarditis as a diagnostic tool for the recognition and treatment of disease other than pericarditis itself (group 2 above). In this group pericarditis is not the predominating clinical picture but occurs as a part of a general process with striking symptoms elsewhere. Here too the findings of pericarditis are likely to develop sometime after the evolution of the primary clinical picture and, if the signs are not outstanding, they may be missed without repeated, pointed examination carried out to elicit them. One must keep in mind from the clinical picture present, the possibility of pericarditis as a part of the total clinical pattern. In a group of 240 cases of acute pericarditis of varying etiology we found this to be true not only in myocardial infarction, where its development may be a critical factor in diagnosis, but in confusing instances of disseminated lupus, rheumatic fever, and in neoplastic disease of the lungs. Interestingly enough, in rheumatic fever, where necropsy studies show practically all patients have pericarditis, clinical reports show an incidence under 50 percent, even in severe attacks.4,5 Repeated search for pericarditis in patients not clearly diagnosed as rheumatic fever may be very rewarding.

These examples, taken from the various etiologic types seen in the group analyzed, indicate that in a large percentage of patients suspicion must be drawn to pericarditis because of findings other than the usual reliable signs and the latter sought out or the patient followed with sequential examina-

### Table 1—Acute Nonspecific Pericarditis: Incidence of Positive Diagnostic Findings.

<table>
<thead>
<tr>
<th></th>
<th>Total patients</th>
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<tbody>
<tr>
<td>Friction rub</td>
<td>26 (88.4%)</td>
</tr>
<tr>
<td>ECG abnormal</td>
<td>35 (92.1%)</td>
</tr>
<tr>
<td>Diagnostic 25</td>
<td>35 (85.8%)</td>
</tr>
<tr>
<td>Nonspecific 10</td>
<td>23 (60.5%)</td>
</tr>
<tr>
<td>Roentgenographic change/</td>
<td>23 (60.5%)</td>
</tr>
<tr>
<td>cardiac shadow</td>
<td></td>
</tr>
<tr>
<td>Paracentesis</td>
<td>9 (23.7%)</td>
</tr>
</tbody>
</table>
tion to establish the involvement of the pericardium. In suspected disseminated lupus this approach may be the only way in which pericarditis is found and which, after establishment, is in turn helpful in confirming the original suspected diagnosis. General symptoms and localized manifestations elsewhere may mask or suppress findings of pericarditis or by their prominence draw attention to other expressions of the disease. In tuberculous and pyogenic pericarditis we frequently found this to be true.

Clearly our series points out the diversity of the approach to acute pericarditis. So it is that although the diagnosis of acute pericarditis depends upon pathognomonic signs, including demonstration of the friction rub, typical electrocardiographic evidences, roentgenographic signs, and the demonstration of pericardial fluid, the diagnosis is easily missed unless these findings are deliberately sought. The seeking of these findings will depend upon the suspicion of pericarditis. This is based upon:

1. The presence of disease known to cause pericarditis frequently. This includes not only such disturbances as rheumatic fever, pneumonia with complications, general septic pictures, chronic nephritis, myocardial infarction, but disseminated lupus, trauma and others. The presence of any one of these disturbances would bring to mind the possibility of pericarditis and the deliberate search for it.

2. The presence of thoracic symptoms which are not adequately explained. At times the acute onset of a clinical picture suggesting pneumonia, pulmonary infarction, or other disturbances with anterior chest pain, but not clearly definitive, should bring to mind the possibility of pericarditis.

3. The well-known diminution in the intensity of heart tones under observation. Although not necessary for the diagnosis of pericarditis with effusion, this change frequently occurs, and its development under observation should make one suspect this possibility.

4. The development under observation of apparent cardiomegaly, which if unexplained, should lead to a similar suspicion.

5. The signs of acute cardiac tamponade which should, of course, lead one to suspect pericarditis with effusion.

Under these circumstances persistent pointed attempts to demonstrate pathognomonic findings of pericarditis are important.

In summary, when the pericarditis does not represent the predominating clinical picture and occurs as a part of a general process with predominating symptoms elsewhere the diagnosis is frequently missed unless repeated pointed examination is carried out for signs of pericarditis. Often suspicion must be drawn to pericarditis because of findings other than the usual reliable signs and the latter then sought out or the patient followed with sequential examinations.

Great variability in etiologic factors and subsequent symptomatology make diagnostic thought often a problem primarily of etiologic rather than anatomic consideration.

Differential diagnosis springing from pathognomonic signs frequently is not the clinical problem.

References


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