Acute Nonspecific Pericarditis
Clinical, Laboratory, and Follow-up Considerations

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Acute nonspecific pericarditis has been recognized with increasing frequency in recent years although there is reason to believe that it was considered distinct from rheumatic pericarditis almost 100 years ago. The authors have been able to secure follow-up information on 50 cases seen between 1930 and 1949. A few have shown persistent electrocardiographic changes, and calcification of the pericardium occurred in 1 case. In general the prognosis appears excellent and we have not observed constricitive pericarditis as a sequela.

In recent years the syndrome of acute nonspecific pericarditis has stimulated the interest of clinicians in increasing degree. The frequency of this condition and its potential relationship to chronic constrictive pericarditis prompted us to review the experience with this form of pericarditis at the Massachusetts General Hospital. The study consists of two parts: a review of the clinical and laboratory aspects of this syndrome and a follow-up study of these patients. Fifty cases were reviewed whose acute illness occurred in the two decades from 1930 to 1949, and long-term observations were obtained on those patients whose illness antedated this study by more than two years.

Historical Background

Ninety-six years ago there appeared in the Boston Medical and Surgical Journal a case record reported by Hodges entitled "Idiopathic Pericarditis," from which he drew the following conclusion: "Acute pericarditis is likely to occur from exposure to cold or when no exciting cause can be detected...." The report described a 40 year old female patient at the Massachusetts General Hospital on the service of Dr. M. S. Perry, with complaints of left anterior chest pain, chills, fever, weakness, nausea, and palpitation. Physical findings were diagnostic of acute pericarditis with effusion, and the judicious application of several blisters to the thorax promptly resulted in a cure. The author noted the rarity of cases without associated rheumatism and declared that "the rapidity of recovery, the simple treatment, and the facility with which the effusion was dispersed are points to be specially remarked."

The association of this form of pericardial reaction with an antecedent upper respiratory infection has been stressed. Credit for the initial case description demonstrating such a relationship is generally given to Comer, but the conclusions of Hodges cited above and published in 1854 indicate that this relationship must have been appreciated many years ago. In 1906 Morison reported the case of a child with acute follicular tonsillitis in which acute pericarditis of benign nature was a sig-
nificant finding.\textsuperscript{3} In 1942 Barnes and Burchell reviewed the status of this syndrome.\textsuperscript{4} Since then numerous case reports have appeared and the syndrome described under multiple designations including acute benign pericarditis, serofibrinous pericarditis of undetermined cause, pericarditis simulating coronary occlusion, cryptic pericarditis, and idiopathic pericarditis.\textsuperscript{5–25}

Method and Clinical Description

The cases described in this review were assembled from the records of all cases of acute pericarditis occurring at the Massachusetts General Hospital and on the private services of Dr. P. D. White, Dr. H. B. Sprague, and Dr. E. F. Bland. All cases in which a specific etiology was ascertainable were excluded. Pericardial inflammation accompanying uremia, myocardial infarction, rheumatic carditis, purulent chest disease, tuberculosis, trauma, myocarditis, and endocrine imbalance was not included. Of the remaining cases, 50 fulfilled the requirements of acute nonspecific pericarditis and were submitted to clinical review and follow-up examination.

Review of these records indicates that acute nonspecific pericarditis has been more common during the recent years of the series. These data are summarized in table 1. Reports have indicated that this syndrome is most frequent in young adults. That this is not strictly the case is shown in table 2. It may be seen that a fair percentage of patients had acute pericarditis during the "coronary" period of life, and hence the patient's age cannot be depended upon in the differential diagnosis from myocardial infarction. The usual predomiance of pericarditis in the male is found in this group of cases.

An upper respiratory infection or mild, nonspecific period of general malaise commonly precedes the onset of this form of pericarditis. In a recent series Levy and Patterson report 23 of 27 cases ushered in with upper respiratory infections.\textsuperscript{22}

<table>
<thead>
<tr>
<th>Description</th>
<th>Cases</th>
<th>Interval period in days</th>
</tr>
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<tbody>
<tr>
<td>&quot;Grippe&quot;</td>
<td>7</td>
<td>1, 7, 8, 10, 14, 14, 30</td>
</tr>
<tr>
<td>&quot;Cold&quot;</td>
<td>7</td>
<td>2, 3, 5, 7, 14, 21, 60</td>
</tr>
<tr>
<td>&quot;Pleurisy&quot;</td>
<td>2</td>
<td>3, 30</td>
</tr>
<tr>
<td>&quot;Upper respiratory infection&quot;</td>
<td>9</td>
<td>5, 8, 10, 10, 11, 14, 14, 40</td>
</tr>
<tr>
<td>&quot;Acute bronchitis&quot;</td>
<td>2</td>
<td>14, 30</td>
</tr>
<tr>
<td>&quot;Cold and pneumonia&quot;</td>
<td>2</td>
<td>4, 40</td>
</tr>
</tbody>
</table>

Of the 50 cases summarized in this report 29 had an antecedent respiratory illness and 21 failed to give such history. These data are summarized in table 3. It will be noted that in over one-half there was an interval of 7 to 14 days of partial or complete freedom from symptoms prior to the onset of the complicating pericarditis. In only one instance was a patient so ill that hospitalization during the interim was necessary. Among the patients in whom an episode of previous illness was not elicited, it was not infrequent for the pericarditis to occur following unusual physical or emotional exertion, or exposure to cold.

Five patients gave a history of known contact with active tuberculosis, and the number of cases with a personal or family history of allergy was high.

The onset of pericarditis was described as occurring abruptly in 25 instances and less acutely in 25. By far the most common present-
ing complaint was pain with 46 of the 50 cases (92 per cent) listing this as the initial symptom.

The pain was generalized over the entire anterior chest in 17 cases, restricted to the mid-chest and substernal area in 18, limited to the left shoulder in 6, to the left anterior chest in 3 and the right anterior chest in 2, and over the abdomen in 1. The pain was intensified by respiration in 35 patients (76 per cent), by change in position in 5, and by cough in 2. Pain was aggravated by the prone position in 7 cases. The patients described the pain as severe in 19 cases, moderate in 24, and minimal in 3. In 34 instances it was intermittent, but was continuous in 14.

There was no consistent description of the discomfort. A galaxy of terms was used by the patients to describe their distress and the most commonly used terms included: sharp, dull, aching, pressing, gripping, constricting, and tightness. The variable nature of the pain and the difficulty of differentiating it from the pain of other cardiac and pulmonary diseases is readily apparent.

Radiation of the pain was common, and the locations are listed in table 4. Dyspnea was the initial symptom in 2 instances and fever in 2.

Collateral or subsequent symptoms appearing during the acute illness are listed in table 5. Malaise, fever, cough, and dyspnea were noted to be the most common accompanying complaints. Signs of circulatory collapse were infrequent.

The physical finding of greatest significance in the diagnosis of this illness is a pericardial friction rub. Its presence within a few hours of the onset of chest pain is a valuable aid in the differentiation of this syndrome from acute myocardial infarction, where such a sign seldom appears within the first 36 hours. In some instances a pleuropericardial element is present. In 37 patients of this group a friction rub was heard. This figure is lower than might be anticipated in cases selected purely from a hospital population. Several of the patients in this series were seen in consultation after the period of probability of a friction rub had diminished. All degrees of loudness were described, and in 30 cases the rub was heard on the initial visit of the physician. The mean duration of the rub was 9 days, with an extreme of intermittent appearance for 60 days in 1 patient. A loud to-and-fro rub was fairly consistently associated with a more prolonged course than a poorly discernible friction sound.

Enlargement of the cardiac shadow was found in 25 instances. This was attributed to pericardial fluid in 3 cases and to cardiac dilata-

### Table 4.—Radiation of Pain

<table>
<thead>
<tr>
<th>Location</th>
<th>Frequency</th>
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<tbody>
<tr>
<td>Left shoulder</td>
<td>19</td>
</tr>
<tr>
<td>Right shoulder</td>
<td>9</td>
</tr>
<tr>
<td>Neck</td>
<td>7</td>
</tr>
<tr>
<td>Back</td>
<td>6</td>
</tr>
<tr>
<td>Costal margins</td>
<td>4</td>
</tr>
<tr>
<td>Left arm</td>
<td>4</td>
</tr>
<tr>
<td>Epigastrium</td>
<td>3</td>
</tr>
<tr>
<td>Back of neck</td>
<td>2</td>
</tr>
<tr>
<td>Jaw</td>
<td>1</td>
</tr>
<tr>
<td>Elbows</td>
<td>1</td>
</tr>
<tr>
<td>Right chest</td>
<td>1</td>
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</table>

### Table 5.—Collateral Symptoms

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Frequency</th>
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<tbody>
<tr>
<td>Malaise</td>
<td>49</td>
</tr>
<tr>
<td>Fever</td>
<td>49</td>
</tr>
<tr>
<td>Cough</td>
<td>49</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>49</td>
</tr>
<tr>
<td>Nausea</td>
<td>49</td>
</tr>
<tr>
<td>Anorexia</td>
<td>49</td>
</tr>
<tr>
<td>Chill</td>
<td>49</td>
</tr>
<tr>
<td>Orthopnea</td>
<td>49</td>
</tr>
<tr>
<td>Palpitation</td>
<td>49</td>
</tr>
<tr>
<td>Night sweats</td>
<td>49</td>
</tr>
<tr>
<td>Numbness of arms</td>
<td>49</td>
</tr>
<tr>
<td>Collapse</td>
<td>49</td>
</tr>
<tr>
<td>Dizziness</td>
<td>49</td>
</tr>
<tr>
<td>Headache</td>
<td>49</td>
</tr>
<tr>
<td>Cyanosis</td>
<td>49</td>
</tr>
<tr>
<td>Insomnia</td>
<td>49</td>
</tr>
</tbody>
</table>

The assumption that the cardiac enlargement was due to dilatation rather than fluid was based upon the lack of suppression of cardiac sounds, absence of signs of cardiac compression, and persistence of normal voltage in the electrocardiogram. It has been stressed that a dilated heart may mimic pericardial effusion so closely that the roentgenologist is unable to establish the differential diagnosis. Without angiocardiographic studies or diagnostic pericardial aspirations, only a presumptive conclusion is possible.

Pleural fluid was also noted in 14 instances and the chest x-ray examination indicated bilateral effusion in 6, isolated left-sided fluid in 7, and isolated right-sided effusion in 1 instance.

Albuminuria was observed in 2 cases. Tuberculin skin tests were performed on 12 patients. The result was negative in 8 instances and revealed a minimal response in the remaining 4 cases. Where sedimentation rates were obtained, all but 3 were elevated, and this test proved an accurate index of improvement in
the clinical status of the patient. A moderate normocytic anemia was observed in 8 of 37 patients in which hematologic studies were performed.

Blood, urine, throat, and nasal cultures were obtained in several instances but no consistent findings were observed. The duration of the acute illness was very variable with extremes of 2 to 70 days. The mean duration of illness was 13 days. Treatment was supportive and no specific effect was noted when penicillin, sulphonamides, or salicylates were used.

The electrocardiogram is a valuable diagnostic aid in many cases of acute pericarditis, and this has been the subject of a number of excellent reviews.29–34 Changes in the T wave following trauma to the pericardium were initially observed by Barnes and Mann in 1932, and the initial concept that such changes are due to subepicardial myocardial damage stems from a report by Fowler, Rathe, and Smith, published the next year.35 Following this work pericarditis was the subject of numerous electrocardiographic observations.

The electrocardiographic pattern depends upon several factors, including the phase of the pericarditis, degree of subepicardial damage, amount of pericardial effusion, and adequacy of electrocardiographic exploration. Unquestionable cases have been reported without electrocardiographic changes, and in such instances it must be assumed that the integrity of the myocardium has been so minimally disturbed that RS-T changes did not occur.

RS-T segment elevation may be quite evanescent and was not observed after the twelfth day. Nay reports an interesting differential point from myocardial infarction, observing that in pericarditis the T-wave negativity seldom appears until the RS-T segment has returned to the isoelectric line.34 Late electrocardiographic changes will be considered in a subsequent part of this paper.

**LONG-TERM OBSERVATIONS**

In spite of the extensive literature devoted to the clinical findings in acute nonspecific pericarditis few reports have included data describing possible late effects of this condition. Burchell indicates that he is following a number of cases and Levy recently reported 17 patients who had been observed two years or more after the initial episode.37, 32 To our knowledge no instance of chronic constrictive pericarditis has occurred in the follow-up observations of the reported cases.

Forty-seven instances of acute pericarditis had occurred more than two years prior to follow-up and their subsequent course was reviewed. Since 2 patients had been hospitalized on two occasions with similar findings the actual number of patients was two less than the number of episodes of pericarditis, making a total of 45 patients and 47 instances of pericarditis concerned in the follow-up.

**Table 6.—Duration of Follow-up**

<table>
<thead>
<tr>
<th>Years</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–4</td>
<td>12</td>
</tr>
<tr>
<td>5–9</td>
<td>19</td>
</tr>
<tr>
<td>10–14</td>
<td>6</td>
</tr>
<tr>
<td>15–18</td>
<td>6</td>
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**Table 7.—Present Ages**

<p>| | | | | |</p>
<table>
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<tr>
<th></th>
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<th></th>
<th></th>
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<tbody>
<tr>
<td>0–10</td>
<td>0</td>
<td>0.0</td>
<td>0.0</td>
<td>13</td>
</tr>
<tr>
<td>11–20</td>
<td>3</td>
<td>0.0</td>
<td>0.0</td>
<td>5</td>
</tr>
<tr>
<td>21–30</td>
<td>7</td>
<td>0.0</td>
<td>0.0</td>
<td>4</td>
</tr>
<tr>
<td>31–40</td>
<td>8</td>
<td>0.0</td>
<td>0.0</td>
<td>1</td>
</tr>
</tbody>
</table>

Information was available on all but 4 of these individuals. Twenty-nine patients were seen and examined in this hospital and 9 were examined for us by competent internists elsewhere. Partial information was available in 2 instances, and 1 patient had expired. The duration of the follow-up and present ages of the patients are summarized in tables 6 and 7. All living patients were able to carry on normal activity and many were engaged in strenuous vocations.

Because of the frequency of recurrences in this syndrome the term "acute relapsing pericarditis" has been used.7 Two of our patients gave a history compatible with previous acute pericarditis when seen, and 4 more suffered recurrences (14.6 per cent). In addition there were 3 other patients in which a recurrence was suspected, but evidence was not conclusive.

Four recurrences each were noted by 2 patients. In general recurrences were of less intensity and of shorter duration than the initial illness.
pain, fever, and malaise were the common symptoms and were quickly recognized by the patient as being similar in nature to his first episode.

A further group noted transient bouts of sharp, stabbing left chest or shoulder pain unattended by fever, malaise or abnormal physical findings. Ten patients (24.4 per cent) were so afflicted and in 5 it was a distressing symptom. This pain frequently was related to deep breathing but was not associated with exercise and was unaccompanied by dyspnea. Characteristically the pain appeared at intervals and the patient would be symptom free for progressively greater periods as time passed. One patient described continuous dull left anterior chest discomfort for several years after his acute illness.

Three patients complained of diminished strength following the illness, and 5 patients dated the onset of nervousness to this event. Two patients were moderately incapacitated by a cardiac neurosis. Two individuals noted exercises dyspnea and 3 complained of a minimal ankle edema in the evenings.

During the interim between the initial illness and this examination 5 patients had had pneumonia. Five considered themselves highly susceptible to virus infections and 6 had recently been in contact with a person with pulmonary tuberculosis. Sixteen patients (39 per cent) gave a personal history of allergy and 6 others (14.6 per cent) had a high familial incidence of disease commonly attributed to an allergic background.

In general it may be stated that the majority of patients were in excellent health. General physical examination indicated severe rheumatoid arthritis in 1 case, and signs compatible with a mild virus pneumonitis in another. Seven patients were moderately obese.

Three patients had developed hypertension and moderate cardiomegaly was observed in 4 instances. In 3 of the latter cases no adequate explanation was ascertainable. Six patients (14.6 per cent) had functional systolic murmurs, and 2 patients had murmurs of grade II intensity at the apex which were unexplained and probably without significance. Bilateral basal pulmonary rales were observed in 1 instance to be described more fully subsequently. The patient who expired died of chronic hypertensive cardiovascular disease which had been present for years prior to the episode of acute pericarditis.

Residual abnormalities in the electrocardiogram following acute nonspecific pericarditis have not been reported. It has been assumed in the past that the record always returned to a normal pattern.

In 6 instances among our cases significant abnormalities were observed which were unexplained by other cardiac or systemic disease. Four patients showed a low voltage or isoelectric T\(_1\) associated with an inverted T in aV\(_L\), and reversal of the T\(_1\)/T\(_3\) ratio (fig. 1). In one of these cases T waves were inverted in leads I, II, III, aV\(_F\), V\(_3\), V\(_4\), V\(_5\) and V\(_6\) (fig. 2). Two additional cases revealed low voltage complexes in leads I and aV\(_L\). One of these latter cases was associated with a diphasic T\(_2\), low voltage T wave in V\(_5\), and isoelectric T in V\(_6\) (fig. 3). Serial records are available on this patient and indicate the apparent permanency of the pattern.

Minor deviations from the normal were observed in several other cases. Four patients had a left axis deviation and 1 a right axis deviation. In 2 the A-V conduction was prolonged to 0.22 and 0.26 second; however, these were elderly patients and this may have represented “normal” variant. A poor axis shift with change from the left to the right lateral recumbent position was observed in 3 instances. Standard two step exercise tests were performed on a majority of cases and no significant deviations from the resting record were observed.

Residual roentgenologic pulmonary or cardiac changes have not been reported in cases of acute nonspecific pericarditis. The patients observed in this hospital during follow-up examinations were fluorosceloped and posteroanterior, posteroanterior grid, and lateral chest films were obtained. Wing indicated special spot films and oblique views were taken. The films were interpreted by the Department of Radiology. In the patients studied elsewhere a report was obtained.

Two were in the military service and chest x-rays were assumed to be within normal limits.
FIG. 1. Two examples of residual low voltage of T₁ and inverted T in aVL. In the lower tracing (T. T.) the semivertical electrical position of the heart may be partially responsible for these changes.

FIG. 2. Residual electrocardiographic abnormality in case showing pericardial calcification on x-ray.

X-ray follow-up examination was made on 37 of the 41 patients. In this group were 3 patients with apparent left ventricular enlargement, and 6 more cases in which the left ventricle was noted to be prominent without cardiac enlargement. In 1 case to be described in detail subsequently there was calcification of the pericardium. Three cases gave evidence
of old pleurisy and in another x-ray findings coincided with the clinical impression of pneumonia.

The following case report is of interest:

A.M., female, age 54. Four years ago this patient developed an upper respiratory infection which was abating when she experienced the gradual onset of generalized chest pain of moderate severity, radiating to both shoulders. It was intermittent and mark-

edly aggravated by deep inspiration. She noted some relief in a semirecumbent position. Night sweats were a prominent feature of the illness. There was no dyspnea and although she did not record her temperature, she did not feel feverish. She had no cough and her appetite was fairly well maintained during the illness. Except for occasional rest periods she carried on her housework for several days before consulting her local physician who examined her and referred her to one of us (E. F. B.).

There was no past history of disease of the cardiac or pulmonary systems, and nothing to suggest a previous episode of myocardial infarction or angina pectoris. Her health had always been vigorous, and except for an abdominal operation 11 years previously she had been well.

Physical examination revealed her to be somewhat obese and in no distress. Inconstant pulmonary rales were noted in the lung bases but other physical findings were within normal limits. The blood pressure was 140/85 and no friction rub was elicited. Fluoroscopy revealed slight fullness of the left ventricle and minimal haziness in the left lung base, compatible with a resolving pleuritis. No calcification of the pericardium was observed. The electrocardiogram was abnormal and is reproduced in figure 2. A diagnosis of nonspecific pericarditis was made.

![Serial records showing residual electrocardiographic changes.](http://circ.ahajournals.org/)

In the interval period of four years this patient had been well and active. She managed a household and carried on a full and normal life. Her only complaint was intermittent sharp chest pain located over the cardiac apex and unrelated to exertion or emotion. Her weight was maintained and she had no edema, exertional dyspnea, cough, or hepatic tenderness. She stated that she sleeps on two pillows but attributes this to habit.

Follow-up physical examination four years later revealed her to be moderately obese, with a blood pressure of 140/80 and pulse of 68. The left cardiac border coincided with the left midclavicular line. No thrill was detected. The second aortic exceeded the second pulmonic sound. In recumbency at the pulmonary area an inconstant grade I systolic murmur
was detected. At both pulmonary bases moist rales were heard in all phases of respiration and were not cleared by coughing. The liver and spleen were not palpable. The neck veins were normal, and there was no ankle or sacral edema.

Fluoroscopy and x-ray films revealed cardiac enlargement chiefly in the region of the left ventricle with a cardiothoracic ratio of 15.5/29 cm. Calcification was noted surrounding the apex of the heart in the region of the anterior-inferior aspect of the left ventricle. It was the impression of the fluoroscopist that this region pulsated with diminished amplitude (fig. 4).

**Fig. 4**: Posteroanterior chest film demonstrates a thick ring of calcium encasing the anterior-inferior aspect of the left ventricle.

It is most unfortunate that this patient was not observed earlier in the initial phase of her illness. Her lack of early electrocardiograms, x-ray studies, and postponement of medical assistance until the acute phase had subsided forces us to accept with considerable reservation a conclusion that only acute nonspecific pericarditis could be the cause of the residual changes observed at the present time. It is entirely possible that a minor amount of pericardial calcification could have been missed fluoroscopically at the first visit.

At the present time the only indication of pericardial disease is found in the x-ray examination. Physical findings have not appeared and she must be considered to have healed pericardial disease which is not producing symptoms.

**Etiologic Considerations**

Descriptions of the syndrome of acute nonspecific pericarditis appearing subsequent to the paper by Barnes and Burchell have added little to the clinical pattern, and in nearly all instances their contentions have been verified. The problem of etiology, however, has remained unsolved. The prevailing hypotheses include: a, virus infection of the pericardium; b, tuberculous infection of the pericardium; c, pericardial response to unknown toxins and d, hypersensitive response of the pericardium. Proponents of the concept of a virus infection point to the history of epidemics of pericarditis, the relationship to virus infection of the upper respiratory passages, absence of bacterial flora in cultures of pericardial fluid, benign course, and, more recently, evidence supporting response to aureomycin.23 As yet unproved, this theory lacks the support of a controlled experiment with antiviral agents. An adequate explanation of the usual lag between infection of the respiratory system and pericarditis has not been forthcoming; and the characteristic leukocytosis has no counterpart in known viral pulmonary infections.

Because of the known frequency of tuberculous infection of the pericardium, suspected relationship with chronic constrictive pericarditis, and reports of pericarditis during the appearance of the Ghon complex, tuberculosis has been suspected as the cause for acute nonspecific pericarditis. Pericarditis, particularly when accompanied by effusion, has been deemed similar to idiopathic pleurisy with effusion in the young adult. For years such pleural effusions have been considered tuberculous but proof has not always been forthcoming.

It is certainly true that the syndrome of acute nonspecific pericarditis bears little similarity to the description we accept as representing primary tuberculous pericarditis. Clinically primary tuberculous pericarditis was described originally by Riesman in 1901 and numerous reports substantiating his conclusions have appeared in subsequent years.27-39
With few exceptions authors agree that this disease is notable for the infrequency of complaints referable directly to the chest. Chest pain is an uncommon symptom and is a particularly uncommon presenting complaint, appearing in but 1 of 13 cases reviewed by Clarke. Stepman and Owyang noted chest pain in 35 per cent of their cases, but this symptom was overshadowed by fever (89 per cent), shortness of breath (76 per cent), edema (68 per cent), cough (51 per cent), and weakness (43 per cent). The physical findings, laboratory results, and prolonged, unfavorable course serve further to distinguish this disease from acute nonspecific pericarditis. Pericardial effusion occurs in the majority of instances and distended neck veins, pleural effusion, palpable liver, and cardiac arrhythmias are common. The leukocyte count is seldom elevated. Unless we radically revise our concepts of primary tuberculous pericarditis, there seems little chance for confusion.

A response to unknown toxins by the pericardium has been suggested. To date no definitive evidence has appeared which would support this concept, except for the known frequency of pericarditis in terminal uremia.

A hypersensitive response by the pericardium has been postulated to explain many forms of pericarditis. Reports have appeared of pericarditis clinically indistinguishable from the acute nonspecific form occurring during serum sickness. In isolated case reports eosinophilia has been noted and fluid with high eosinophil counts has been recovered by pericardial and pleural aspiration. In 1 such case prompt recovery attended administration of an antihistamine compound. Partially elucidated by such an hypothesis would be the acute pericarditis which appears as an incidental finding in a diversity of afflictions. There is no question but that the pericardium may act as a 'shock' organ in a form resembling acute nonspecific pericarditis, although there is no definitive evidence indicating that this is a frequent event.

**Differential Diagnosis**

The differential diagnosis of acute nonspecific pericarditis includes other clinical entities causing chest pain and a friction rub. Of predominant importance is the recognition of this syndrome from acute myocardial infarction. Although shock, circulatory collapse, and fear of impending death are infrequent in acute pericarditis, the clinical resume reveals that they may occur. The early appearance of leukocytosis, fever, and a pericardial friction rub aid in recognition of pericarditis. Pain with respiration is uncommon in acute myocardial infarction. Early and adequate electrocardiography also aids in differentiating the two.

Acute rheumatic pericarditis may antedate other clinical signs of acute rheumatic fever. Endocarditis is ultimately manifest in most instances and the diagnosis is clarified.

Other causes of acute chest discomfort, such as dissecting aneurysm, angina pectoris, diaphragmatic hernia, spontaneous pneumothorax, mediastinal emphysema, acute pleurisy, and so forth, seldom cause difficulty after preliminary physical and laboratory examination.

**RELATIONSHIP TO CHRONIC CONSTRICTIVE PERICARDITIS**

In reviewing a series of patients with chronic constrictive pericarditis a history of acute pericarditis, acute pleurisy, or severe respiratory infection is not infrequent. Paul, Castleman, and White obtained a history of pericarditis in 4 of 53 cases, and 2 further patients had been afflicted with pleurisy. Blalock and Burwell note that a certain number may have antecedent respiratory infection. In Kelly's classic review of polyserositis with chronic pericardial changes 5 cases of 39 gave a history suggestive of previous acute pericarditis, (cases of Mott, Feierabend, 1887; Feierabend, 1866; Rumpf, 1895; and Heidemann, 1897). Nicholls reported 13 cases, and in 2 acute pericarditis had occurred. Isolated case reports have recorded similar findings.

Realization of this fact has led clinicians to have some reservation in stating an entirely optimistic prognosis for patients with acute nonspecific pericarditis.

Analysis of these case reports indicates that in the majority of instances signs of cardiac compression appeared soon after the initial
pericarditis. In 3 of the 4 cases reported by Paul and associates, the onset was a matter of months. Mott's 2 cases noted symptoms seven months after the acute episode, and the cases reported by Rumpf and Heidemann had only a short interval of normal health. In this group, only 1 case reported by Paul and co-workers and that reported by Feierabend enjoyed good health for an appreciable period. Although such acute pericarditis may be indistinguishable from acute nonspecific pericarditis, further analysis of cases appearing in the literature indicates that it is rare for this to be true. In the usual instance the initial complaints are more commonly dyspnea and weakness, and chest pain is not stressed. Such cases fit the picture of primary tuberculous pericarditis more closely in some respects than the nonspecific form.

Among the 41 patients observed for a period greater than two years there was only one in whom a suggestion of chronic changes appeared to be legitimate.

**Summary**

1. Fifty cases of acute nonspecific pericarditis have been reviewed with special reference to clinical, laboratory, and follow-up considerations.

2. The prevailing theories of causation of this syndrome have been summarized and discussed.

3. Long-term follow-up examinations on this group indicate an excellent prognosis, although residual electrocardiographic abnormalities are not infrequent.

4. Pericardial calcification is present in only 1 patient and fluoroscopy reveals a diminution in the amplitude of cardiac contractions, but there is no evidence of impaired function.

**REFERENCES**

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