Nonspecific Pericarditis and Myocardial Infarction
Their Differential Diagnosis

By JOHNSON McGuire, M.D., ROBERT A. HELM, M.D., ARNOLD IGGLAUER, M.D., RALPH C. SCOTT, M.D., J. HAROLD KOTTE, M.D., AND BENJAMIN FELSON, M.D.

DR. JOHNSON McGuire: Within the past few years we, as well as others, have encountered a number of instances in which nonspecific pericarditis has been erroneously diagnosed as myocardial infarction.

The case reports and discussion that follow in this clinical conference serve to illustrate certain of the problems encountered in this important differentiation.

CASE 1

A physician, aged 37, who had hitherto enjoyed excellent health, suddenly developed severe precordial pain that radiated to the left shoulder and along the inner aspect of his left arm to the fingers. Shortly after the onset of pain, the physician listened to his own chest with a stethoscope and heard a pericardial friction rub.

He was admitted to the hospital with the diagnosis of coronary thrombosis and myocardial infarction.

Physical examination showed an acutely ill man with severe chest pain. The blood pressure was 120/80 mm. Hg, pulse 68, and respiratory rate 24. Physical examination suggested slight cardiac enlargement. No murmurs or gallop rhythm were heard. There was a loud pericardial friction rub. The physical examination was otherwise normal.

An electrocardiogram showed elevation of the S-T segments in leads I and II, interpreted as anterior myocardial infarction complicated by pericarditis. Initial x-ray examination of the heart showed slight enlargement thought by the roentgenologist to be due to cardiac hypertrophy rather than pericardial effusion. The white blood count was 15,500 with 75 per cent polymorphonuclear cells; sedimentation rate 60/mm./hr. (Wintrobe technic).

Anticoagulant therapy was instituted with dicumarol. The course in the hospital was characterized by recurring episodes of severe chest pain modified somewhat by changes in position of the body and by intermittent fever.

Dr. McGuire: Because of the appearance of the friction rub at the onset of the chest pain, the marked modification of pain with change of position, and the character of the serial electrocardiograms, the diagnosis of myocardial infarction was changed to that of acute nonspecific pericarditis and the subsequent course made this diagnosis quite clearly the correct one. It is probable that many patients with nonspecific pericarditis have been incorrectly and permanently labeled as having had myocardial infarction. From the point of view of peace of mind of the patient as well as insurability, such a mistake is unfortunate. Also, anticoagulant therapy in acute benign pericarditis is contraindicated as in 1 case, reported by McCord and Taguchi, acute fatal hemorrhagic tamponade was described.

Physician: How quickly following myocardial infarction or the onset of acute nonspecific pericarditis do pericardial friction rubs develop?

Dr. McGuire: A pericardial friction rub most
commonly appears on the second or third day following the infarction and usually disappears in a day or 2. In the case under discussion, the audible pericardial friction rub heard immediately after the onset of pain made the diagnosis of myocardial infarction questionable. The friction rub associated with pericarditis often appears within a few hours of the onset of pain, although cases have been observed in which the rub appears considerably later.

Physician: Is variation in the intensity of the pain with change of position commonly present in patients with myocardial infarction with complicating pericarditis?

Dr. McGuire: In our experience, modification of chest pain with change of position in myocardial infarction, with or without pericarditis, is very rare. However, in patients with acute nonspecific pericarditis intensification or relief of pain with change of body position is common. Rotation of the trunk, change from recumbency to the sitting position, or deep breathing may profoundly alter the severity of the pain. Severe pain is rarely present in the pericarditis due to tuberculosis or rheumatic fever. A possible explanation, as suggested by Burchell,² is that a portion of the inner aspect of the parietal pericardium, like the epicardium, is insensitive to pain while the exterior surface of the parietal pericardium, like the pleura, when inflamed, causes severe pain.

Physician: Is the radiation of pain helpful in the differential diagnosis?

Dr. McGuire: The pain of both myocardial infarction and pericarditis may radiate to the left arm and hand, but the pain of pericarditis rarely extends to the right arm. Back, shoulder, and lower neck pain occur in both diseases. Pain in the jaw or teeth with pericarditis has been reported, to our knowledge, in only 1 case.³

Physician: Are antibiotics of value in the treatment of acute nonspecific pericarditis?

Dr. Arnold Iglauer: Despite a few case reports of apparent benefit from broad spectrum antibiotics and streptomycin, the improvement of these patients may well have been coincidental. In our experience, antibiotics have apparently been of no benefit. Steroid therapy in a few reported instances in which it has been used has produced symptomatic improvement.

Physician: In what way does x-ray examination help to differentiate pericarditis from myocardial infarction?

Dr. Benjamin Felson: Radiologically speaking, this distinction may be difficult or impossible to make, as in both conditions the chest film may appear entirely normal.

Unfortunately, most cases of myocardial infarction and benign pericarditis do not produce typical x-ray changes in heart configuration. However, even in these instances roentgen study may still prove helpful. Thus, localized pulmonary infiltrate is sometimes associated with benign pericarditis, whereas widespread pulmonary congestion and edema may occur from cardiac failure following myocardial infarction. The cardiopericardial silhouette may rapidly enlarge soon after the onset of either condition, but it occurs more frequently and to a greater degree in benign pericarditis. Whether dilatation of the heart or small effusion is responsible for the rapid fluctuations in size of the cardiac silhouette is still a controversial question. Fluoroscopically, recent infarction at or near the cardiac apex quite often results in a localized segment of impaired, absent, or even paradoxical pulsation, whereas impairment of pulsation resulting from benign pericarditis with effusion is more generalized and, if anything, more evident along the right border.

If benign pericarditis results in an extensive effusion, evidence of a large pear-shaped contour, rapid change in size of the cardiovascular silhouette, impaired pulsation, and the occurrence of noncongested lung fields all offer confirmation of the diagnosis. Contrary to the statements in many textbooks, changes in the contour of the pericardial silhouette with change from the upright to the Trendelenberg position has not proved of differential diagnostic value, since the flabby dilated heart also apparently enlarges at the base in the reclining position.

Physician: Does angiocardiography help in the differential diagnosis between dilatation and effusion?

Dr. Felson: Yes. When the dye-filled cham-
Numbers of the heart are seen well within the shadow of the cardiac silhouette, one can be certain that pericardial effusion is present rather than cardiac dilatation.

Dr. McGuire: The second case, which was initially incorrectly diagnosed as myocardial infarction, emphasizes additional features of differential diagnostic value.

Case 2

A. McC., a 55-year-old truck driver, who had had pleuritic left chest and left shoulder pain for 10 days, was admitted to the hospital because of an increase in the intensity of the pain accompanied by an episode of syncope. Positive physical findings included a temperature of 100.4 F., blood pressure of 90/64 mm. Hg, pulse of 72, with frequent premature contractions, pallor, profuse perspiration, slightly distended neck veins, distant heart sounds, and moist rales at the left lung base. The admission diagnosis was myocardial infarction. The blood pressure rose to 112/90 without specific therapy. A transient pericardial friction rub was heard on the fourth hospital day.

Laboratory findings included a polymorphonuclear leukocytosis of 17,500 and a sedimentation rate of 32 mm. per hour. Chest x-ray on the second hospital day showed the transverse diameter of the cardiac silhouette to be 16 cm.; of the chest, 28 cm. There was slight haziness in the left lower lung field and, in addition, a small left pleural effusion. On the twelfth hospital day the cardiopericardial shadow had diminished to 14.5 cm. and the lung fields were clear. Cardiac pulsations were normal on fluoroscopy.

Electrocardiograms 8 and 4 days before hospitalization showed only minimal S-T elevation in the standard leads and several precordial leads. During the first 4 days after admission there was slight S-T elevation in the standard leads with rather marked S-T elevation in V1 and V5 and progressive flattening and inversion of the T waves in the standard leads and in V4, V5, and V6. An electrocardiogram on the fifteenth hospital day showed inverted or diphasic (+ −) T waves in leads I, II, aV6, V5, V6. All tracings demonstrated frequent premature ventricular contractions.

Fever and pain gradually subsided. The patient was discharged after 3 weeks of hospitalization and has since remained well, having returned to his strenuous occupation. The discharge diagnosis was acute nonspecific pericarditis. An electrocardiogram approximately 2 months after the onset of the illness was within normal limits except for a flat T1.

Dr. J. Harold Kotte: This case brings to mind 2 other features of acute pericarditis, namely the common association of pleural or pulmonary lesions and the relative infrequency of shocklike symptoms. Pneumonitis or pleurisy may precede the development of pericarditis, and pain from the latter may merge with that of pleuritis and obscure the diagnosis unless variation in the pain pattern is considered carefully.

Fall in blood pressure with attendant weakness, sweating, and impairment in level of consciousness may occur briefly at the onset of pericarditis but rarely reaches the magnitude observed in myocardial infarction. Manifestations resembling shock are probably due to vasodepressor reflexes originating from severe pain, as was apparent in this case from the development of fainting and absence of concomitant tachycardia. Rarely cardiac tamponade may be sufficiently severe to produce the manifestations of shock. There has been 1 report of a probable case of nonspecific pericarditis with death from peripheral circulatory failure shortly after the onset of the illness. In the majority of patients with this condition, the manifestations of tamponade are not sufficiently marked to require pericardiocentesis.

Physician: What are the characteristic electrocardiographic findings in acute nonspecific pericarditis in contrast to the alterations that occur with myocardial infarction?

Dr. Robert A. Helm: In the acute stage of pericarditis the S-T segments are typically elevated in all 3 standard leads and in most or all of the precordial leads. Lead aVR, as conventionally recorded, displays S-T depression. In myocardial infarction the S-T displacement frequently takes opposite directions in leads I and III, and the S-T segment elevation may be limited to a few precordial leads with isoelectric or depressed S-T segments in the remainder of these leads.

The elevated S-T segment in pericarditis usually takes a straight course or has a curvature that is concave upward. This has been emphasized as a distinguishing characteristic in contrast to the frequently upward convexity of the elevated S-T segment of myocardial infarction. In our experience, the S-T contour
in both conditions is sufficiently variable that this differential point has only limited diagnostic value. The S-T segment displacement in pericarditis may not be marked and is sometimes within the limits of what is considered to be normal. In such instances, observation of serial tracings for the evolutionary changes characteristic of pericarditis is most helpful. The S-T segments usually return to the baseline within or at the end of the first week. Although rarely the T waves remain upright, characteristically they flatten and begin to invert. The inversion is usually symmetrical. In this intermediate stage the flattened T waves may be notched. The T waves may remain inverted or flattened for weeks or months and frequently show variations in their depth and contour from day to day despite absence of other evidence of active disease. It has been stated that, in contrast to infarction, T-wave inversions do not occur in pericarditis until the S-T segments return to the isoelectric level. This is not a universal finding, however, and the cove-plane S-T and T contour classically associated with infarction may be seen in pericarditis.

Pericarditis does not distort the QRS complex except that pericardial effusion may diminish the voltage; slight positional variation of the QRS axis may also occur. Pathologic Q waves characteristic of infarction are never seen. However, the electrocardiographic diagnosis of pericarditis presents a problem when the S-T and T-wave changes that it produces must be differentiated from those caused by myocardial injury and ischemia or by infarction occurring without alteration of the QRS complex. Thus, Pruitt and his associates found massive T-wave inversion in precordial leads (without QRS alteration) to be associated frequently with subendocardial infarction or with coronary insufficiency; such changes may be indistinguishable from those sometimes seen in the subacute or chronic stage of pericarditis. If, in such a circumstance, a friction rub is present, the diagnosis of pericarditis is much more likely. Myocardial infarction produces a friction rub only when it is transmural or when it involves at least the epicardium. In such instances the absence of significant alteration in the QRS complex, indicative of infarction, is probably rare in view of the experimental work carried out in Prinzmetal's laboratory. Likewise, when acute myocardial infarction is complicated by pericarditis leading to electrocardiographic changes from the latter, QRS alteration resulting from the infarction is usually present.

Premature contractions may occur as in this second case but serious arrhythmias and various degrees of A-V block are exceedingly rare in nonspecific pericarditis and are much more common in myocardial infarction.

The effect of exercise on the electrocardiogram during pericarditis has received relatively little attention. S-T segment elevation, occurring as a normal variant, disappears with exercise; in acute pericarditis the segment shift is said to persist but a sufficient number of patients with this condition have not been subjected to exercise to indicate that this is a universal finding. Transient inversion or deepening of already inverted T waves have been reported to occur with the Master 2-step test in patients who were convalescing from pericarditis. We have observed such a "positive" exercise test in 2 such cases. On the other hand, patients who have recovered completely from pericarditis show no higher incidence of abnormalities after exercise than would be expected in the general population. Thus, Carmichael and his associates observed no significant deviations from the resting record in patients who had had nonspecific pericarditis 2 or more years previously.

Physician: Would you discuss the electrophysiology of the electrocardiographic changes found in pericarditis?

Dr. Helm: There are 2 important pathologic characteristics of pericarditis that explain the electrocardiographic findings. Firstly, only the superficial epicardial layer of the myocardium is involved and, secondly, the process is usually diffuse rather than localized. The absence of involvement of a significant portion of the myocardium accounts for the absence of alteration of the contour of the QRS complex. This same pathologic characteristic explains a
finding that is emphasized in Lepeschkin's text but that apparently has not received general attention. In a lead demonstrating an Rs complex, elevation of the S-T segment resulting from pericarditis will not obliterate the small s wave but will displace it above the baseline so that it forms a well-defined notch on the lowest portion of the descending limb of the R wave. This is well illustrated in a number of tracings of pericarditis published in the literature and has been seen in our own material. It may be explained by the hypothesis that in pericarditis the S-T segment elevation does not begin until the activation front has reached the epicardial surface. In myocardial infarction the S-T segment elevation commences earlier in the QRS complex, since the area of injury extends into the subendocardial region. A small s wave is therefore obliterated by the S-T segment elevation accompanying infarction. Perhaps differences in the contours of the S-T segments in the 2 conditions may be similarly explained by differences in the time of onset of the S-T segment shift with respect to the last portion of the QRS complex inscribed from areas of myocardium not involved by the pathologic process.

There are 2 explanations for the development of S-T segment shift in pericarditis:

1. A difference in potential develops between the inflamed, partially depolarized epicardium and the adjacent resting or polarized myocardium resulting in displacement of the T-P segment in either an upward or downward direction, depending on the direction of the lead axis as described below, during the diastolic phase of the cardiac cycle. Although no longer isoelectric, the T-P segment continues to be taken as the baseline of the tracing, since its actual shift could not be apparent unless the injury were actually to occur during the inscription of the record. The S-T segment, which, according to this explanation, remains isoelectric, is then inscribed at a different level than that of the T-P "baseline."

2. The process of electric activation may fail to penetrate or may only partially penetrate the injured area. This results in a failure of the S-T segment to return to the isoelectric level during the systolic phase of the cardiac cycle, since a potential difference exists between the polarized or only partially depolarized epicardium and the adjacent, completely depolarized myocardium. According to this explanation, the T-P segment remains isoelectric.

Hellerstein and Katz recorded continuous tracings during the experimental production of injury to various portions of the heart. They showed that both explanations are applicable in that systolic displacement of the S-T segment and diastolic displacement of the T-P segment occurred simultaneously, each to a

![Fig. 1. The hexaxial reference frame has been superimposed upon a vertically placed heart in A, and upon a horizontally positioned heart in B. The small arrows emanating from the epicardium represent "injury vectors" resulting from diffuse pericarditis.](image-url)
variable degree. Since these respective systolic and diastolic potential differences have opposite polarities, they summate to increase the divergence between the 2 segments. In electrocardiographic terminology this divergence is referred to as S-T segment displacement.

The occurrence of S-T elevation in a particular lead depends upon the axis of the lead, the position of the heart, and the extent of the pericarditis. In the case of the extremity leads these relationships are illustrated in figure 1, where it is assumed that the inflammatory process involves the epicardial surface diffusely. The leads, represented as vectors, of the hexaxial reference frame in which lead aVR is considered to have a polarity opposite to that with which it is usually recorded in A and on a more horizontally positioned heart in B. The differences in potential between the inflamed epicardium and adjacent myocardium, accounting for the different levels at which the S-T and T-P segments are inscribed, are represented by "injury vectors" radiating out from the periphery of the epicardial surface. Coincidence of direction of lead axis and an injury vector component contributes to the insinuation of S-T elevation, whereas divergence of direction contributes to S-T depression. The sum total of the components of all of the injury vectors projected on the lead axis determines the direction and the degree of S-T segment shifted recorded in that particular lead. Therefore, regardless of heart position, leads II and -aVR usually demonstrate substantial degrees of S-T elevation. In the vertical heart position lead aVR records the components of a larger number of injury vectors producing S-T elevation than does lead I, whereas, in the horizontal heart position, these relationships are reversed. In the vertically placed heart, lead III demonstrates S-T elevation whereas, in the horizontal heart, figure 1B illustrates the reason for the frequency of an isoelectric or perhaps slightly depressed S-T segment in this lead. Similarly, in the case of lead aVL, an isoelectric or slightly depressed S-T segment may occur in the vertical heart position, whereas an elevated S-T segment is to be expected in a more horizontally placed heart. It should be realized that the manner in which pericarditis alters S-T segments in various leads has been greatly simplified in figure 1, but this scheme may be contrasted with the effect of localized epicardial and subepicardial injury resulting from narrowing or occlusion of a branch of a coronary artery. If, in the latter condition, a lead axis passes through or adjacent to the injured area with such a polarity that the injury vectors project sufficient components upon the lead to cause the insinuation of S-T depression, these components are not canceled or exceeded by those of oppositely directed injury vectors arising from the epicardial region of the opposite side of the heart. Thus, in myocardial infarction, leads I and aVL tend to demonstrate segment shift that is displayed in the opposite direction in leads III and aVR, regardless of heart position. Since apical infarction is relatively rare, leads II and -aVR, whose axes point in a general direction from base to apex, usually show less marked segment shift than the other 4 extremity leads, whereas in diffuse pericarditis, II and -aVR are the leads whose S-T segments are most frequently and markedly elevated.

The wide distribution of S-T segment elevation in precordial leads commonly seen with pericarditis in contrast to the localized distribution of S-T elevation or concomitant occurrence of S-T elevation and depression with infarction, may be explained by applying the principles already enumerated for the limb leads although one must be more cognizant of the importance of proximity effects.

In most cases, S-T segment deviation results from direct injury to the epicardial region by the agent responsible for the pericarditis. It has been shown experimentally, however, that S-T elevation can be produced by injection of innocuous substances, such as warm saline and serum, into the pericardial sac, the apparent mechanism being compression and resultant ischemia of the epicardial muscle layer.

A normally upright T wave represents the
spread of the repolarization process in the general direction from epicardium to endocardium. Since inverted T waves commonly develop with pericarditis in those leads in which elevated S-T segments had previously occurred, it may be postulated that the duration of the repolarization in the previously injured epicardial muscle layer is sufficiently lengthened to bring about a net reversal of the direction of this process. This explanation is in line with the finding that the Q-T interval may be increased at this time.

Physician: Is there any characteristic ballistocardiographic pattern in pericarditis?

Dr. Kotte: There is no characteristic pattern. We have observed normal ballistocardiograms during the course of acute nonspecific pericarditis when the electrocardiogram showed typical alterations. However, nonspecific abnormalities may occur. The ballistocardiogram has not been studied sufficiently in nonspecific pericarditis to furnish adequate data concerning the incidence of such abnormalities or to determine whether they correlate with clinical evidence of cardiac tamponade or myocardial dilatation. At the present time the possible potential usefulness of this instrument in the differentiation of acute pericarditis from myocardial infarction is purely speculative.

Dr. McGuire: We believe our third case is of interest since it may represent an example of the syndrome recently described by Dressler.10

Case 3

On January 16, 1954, a 51-year-old white man was admitted to the hospital because of the onset of severe precordial pain with radiation into the left arm accompanied by profuse diaphoresis. Physical examination revealed a pulse rate of 120 and slight cyanosis of the lips. There was no cardiac enlargement, and no murmurs or friction rubs were heard. An electrocardiogram revealed an extensive acute anteroseptal myocardial infarct.

The initial pain was controlled with morphine. After the third day he was free of pain. Anticoagulant therapy was started.

During the last week of January the patient complained of aching in the left shoulder region. The temperature rose to 100.8 F., and slight tachycardia developed. A pericardial friction rub was heard over the lower sternum. The aching pain in the lower left chest was accentuated by deep breathing. The white count rose to 19,000 with 76 per cent neutrophils. The prothrombin time had fallen to 6 per cent of normal, and dicumarol was temporarily discontinued. A portable x-ray of the chest showed slight left pleural effusion and marked cardiac enlargement. Physical examination suggested pericardial effusion. By the end of the first week in February, the patient felt well again. Chest x-ray showed the heart size to be normal. The pleural effusion had disappeared. Convalescence was uneventful and by March 1 he felt entirely normal. Electrocardiograms showed a stabilized pattern of anteroseptal myocardial infarction. He was discharged and while at home felt well until the twelfth of March when he developed aching pain in the left shoulder and arm. The pain occasionally radiated into the left part of the neck. The substernal sharper pain was definitely aggravated by movement and deep inspiration and temporarily relieved by sitting up. The temperature was 100.3 F. Physical examination revealed a to-and-fro friction rub over the precordium and a loud pleural friction rub over the left lower anterior chest.

He was readmitted to the hospital. An electrocardiogram showed slight elevation of S-T segments of V1 and V2 in addition to the typical changes of anteroseptal myocardial infarction. Chest x-rays demonstrated pleural effusion at the left base without cardiac enlargement. The prothrombin time was 21 per cent. Because of the possibility of bleeding into the pericardial cavity and pleural space, dicumarol was discontinued. By March 18 the patient had greatly improved and the pericardial friction rub had disappeared. The sedimentation rate was 48 mm., white blood count 9,000, with 60 per cent neutrophils. Electrocardiograms showed no evidence of a new infarct. He was discharged April 1 and has continued to be asymptomatic.

Dr. Ralph Scott: This patient had 2 distinct bouts of pericarditis and pleurisy during the second and eighth week of his convalescence from an acute myocardial infarction. Complications such as these are commonly attributed either to an extension of the area of myocardial infarction or to pulmonary infarction. However, there was no evidence of either, in that the electrocardiogram remained stable, the effect of change in position and respiration on the pain was not characteristic of myocardial ischemia, and there was no hemoptysis nor any evidence of thrombosis of the leg veins. With the first episode the rapid change in the size of the cardiac silhouette in the absence of clinical and x-ray evidence of pulmonary congestion strongly suggested peri-
cardial effusion rather than cardiac dilatation. Massive effusion with pericarditis resulting from epicardial infarction is uncommon unless, with anticoagulant therapy, bleeding from the infarcted area occurs. The possibility of hemopericardium and hemothorax complicating the excessively prolonged prothrombin time was considered, but it seemed unlikely that bleeding would occur into 2 separate serous cavities in the absence of both microscopic hematuria and hemorrhagic manifestations in skin and mucous membranes. The clinical manifestations, particularly their recurrent nature, were most reminiscent of acute nonspecific pericarditis, and this diagnosis was strongly entertained. Dressler has since described a postmyocardial infarction syndrome characterized by recurrent febrile manifestations of pericarditis, pleurisy, and pulmonary inflammation, mimicking those of nonspecific pericarditis, and we feel that our case falls into this category. Of Dressler’s 10 reported cases, 8 were observed during a period of 13 months in a single hospital, this incidence being greater than that of uncomplicated nonspecific pericarditis in the same hospital over the same period. This fact led Dressler to conclude that the chance coincidence of myocardial infarction and nonspecific pericarditis is unlikely. With the increased recognition of the postinfarction syndrome, which will undoubtedly occur as the result of Dressler’s excellent contribution, a larger statistical study of its incidence in relation to that of spontaneously occurring nonspecific pericarditis would be of interest.

Physician: What is known concerning the cause of acute nonspecific pericarditis, of the pericarditis of the postcommissurotomy syndrome, and of the postinfarction pericarditis described by Dressler?

Dr. McGuire: Both Burchell and Wolff believe that there is no single cause for acute nonspecific pericarditis but that a number of different agents are responsible for this peculiar disorder.

The notorious frequency of relapse in nonspecific pericarditis, the fever, leukocytosis, and increased sedimentation rate strongly suggest an infectious etiology. However, the agent or agents causing acute nonspecific pericarditis have not been identified. The frequency of upper respiratory infections preceding the onset of this form of pericarditis is reminiscent of respiratory infections preceding the onset of acute glomerulonephritis and of rheumatic fever. However, no consistent elevation of the antistreptolysin or antihyaluronidase titers has been noted in patients with acute nonspecific pericarditis, nor have throat cultures in the few reported cases shown any marked predominance of streptococci. The few cultures reported from the pericardial fluid have been sterile. Sera from 5 children with acute nonspecific pericarditis have been negative for cold antibodies and for antibodies to the viruses of influenza A and B, lymphogranuloma venereum, psittacosis, Q fever, and to several strains of Coxsackie virus. Pericarditis mimicking acute nonspecific pericarditis has occurred during the course of infectious mononucleosis.

The clinical pictures of pericarditis in the postcommissurotomy syndrome and in acute nonspecific pericarditis have much in common although the arthralgias of the postcommissurotomy syndrome are not present in acute nonspecific pericarditis. Whether the pericarditis following mitral valve surgery is due to activation of latent rheumatic fever or to an unknown etiologic agent is not known.

The peculiar pericarditis following myocardial infarction described by Dressler closely resembles acute nonspecific pericarditis and may in fact be caused by the same agent or agents responsible for acute nonspecific pericarditis. It is interesting to speculate whether trauma by the surgeon or by a myocardial infarction may play a similar role in the causation of pericarditis.

Dr. McGuire: The primary purpose of this conference has been to call attention to the relative frequency with which acute nonspecific pericarditis is erroneously diagnosed as acute myocardial infarction. This has unfortunate implications, both from a therapeutic standpoint, since anticoagulants are contraindicated in pericarditis, and also from a
prognostic standpoint because the diagnosis of infarction has important economic and social ramifications. The first 2 cases represent instances of such mistaken diagnoses that could have been avoided on the initial examinations by careful evaluation of certain differential diagnostic points. It is possible that, in the future, serial estimations of the serum transaminase reaction\(^\text{13}\) may prove to be a distinct laboratory aid in the differentiation of these 2 conditions. The third case was presented to illustrate a new syndrome recently described by Dressler in which an apparently benign form of pleuropericarditis complicates myocardial infarction.

REFERENCES


The author discussed the subject of cardiac arrest and reported 3 illustrative cases, 1 being a successful resuscitation after 50 min. of asystole.

It was pointed out that cardiac arrest is a much more frequent occurrence than is generally realized. The data collected from the literature also indicate that the actual incidence of this condition is increasing. Most cardiac arrests are preventable if attention is paid to the preoperative preparation of the patient. During the operation, the most important prophylactic measure is anesthetic management, particularly the maintenance of adequate oxygenation.

Treatment resolves itself into the main purposes of early oxygenation of tissues, especially the brain, and production of blood flow by cardiac massage until normal heart beat is restored. As adjunct therapy, intracardiac epinephrine, barium chloride, and whole blood, in the presence of hemorrhage, are worthy of trial. For ventricular fibrillation, cardiac massage, procaine amide and electric shock to produce defibrillation, are indicated.

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