Postmyocardial Infarction Syndrome

By Nelson J. Weiser, M.D., Milton Kantor, M.D., and Hollis K. Russell, M.D.

A new syndrome that is likely to follow myocardial infarction has recently been described. The cardinal manifestations of this syndrome may easily be confused with other, more common sequels of myocardial infarction, including cardiac failure, further infarction of the myocardium, and pulmonary embolism. The result of such confusion may be inappropriate therapy. This paper reviews the recent literature and our own experience with this syndrome and discusses the therapeutic implications.

In April 1956 Dressler first called attention to a syndrome that he had observed in patients following myocardial infarction and that he described under the rather nonspecific title of The Post Myocardial Infarction Syndrome. Subsequently, he and his co-workers reported additional cases, pointing out the importance of recognition of this syndrome and theorizing as to its possible pathogenesis. By January 1959, Dressler had personally observed 53 cases and estimated the incidence of this disorder at approximately 3 per cent of patients with acute myocardial infarction.

The postmyocardial infarction syndrome is characterized by prolonged or recurrent fever, chest pain, and clinical and laboratory evidence of pericarditis, pleurisy, and pneumonitis. These abnormalities may occur alone or in combination, and all show a marked tendency to recurrence.

The fever may merge with the initial temperature elevation of the myocardial infarct, in which case the febrile period was unusually prolonged for infarction alone, or it may appear as a secondary rise from 1 to 8 weeks after the infarction. The temperature usually ranged between 101 and 102 F., persisted for approximately 1 week, and might be followed by a low-grade elevation to about 100 F. until the next bout of higher temperature occurred.

The chest pain usually appeared at the time the temperature became elevated, though in some instances it did not develop until the second or third febrile episode. Characteristically, the pain was pleuropericardial in type, but at times it closely resembled angina. In exceptional cases, pain was absent throughout the entire course of the illness.

Fibrinous pericarditis during the early course of acute myocardial infarction is fairly common, appearing on the second to fourth day when the evanescent pericardial rub denoting its presence is most frequently heard. Pericarditis in patients with the post-infarction syndrome differed in several important respects from that usually associated with infarction. A pericardial rub, which was found in 80 per cent of the cases, developed 3 to 24 days after the infarction; instead of being evanescent, it remained audible 7 to 10 days, occasionally recurred several weeks later. Moreover, although fibrinous pericarditis has not been uncommon with acute myocardial infarction, pericardial effusion has been so rare that it has become the subject of isolated case reports. In the cases studied by Dressler, almost half of the patients had pericardial effusion of sufficient degree to be diagnosable on serial x-ray studies. In several instances the pericardium was aspirated; the fluid was at times straw colored, and at other times was hemorrhagic even in the absence of anticoagulant therapy.

When pleural effusion appears during the course of myocardial infarction, it is usually attributed to either congestive heart failure
or pulmonary infarction. In the cases described by Dressler, pleural involvement was very common, but in only 1 instance was cardiac failure coexistent, and none showed signs of pulmonary infarction. The effusion was minimal in some; in others it was a paramount change necessitating repeated paracentesis for relief. Like the pericardial effusion, the fluid obtained was at times straw colored, at other times intensely hemorrhagic.

Pneumonitis developed in approximately 25 per cent of the cases observed. In addition to cough, rales, and x-ray evidence of infiltration in one or both lungs, hemorrhagic expectoration also occurred. Hemorrhagic pneumonia after myocardial infarction has been observed in postmortem studies. In none of the cases reported by Dressler were the pulmonary changes associated with thrombophlebitis, pulmonary embolism, or congestive heart failure.

Laboratory studies showed only a leukocytosis with a shift to the left and an increased sedimentation rate. In protracted cases, a moderate anemia and hypoproteinemia occasionally developed. The electrocardiogram, in addition to the signs of recent myocardial infarction, showed changes suggestive of pericarditis in about half of the cases. Such studies as cultures of blood and of the pericardial and pleural fluids, search for acid fast bacilli, tests for lupus erythematosus cells, cold agglutinins, heterophile antibodies, agglutinins for typhoid and brucella, and tuberculin tests were invariably negative.

As already mentioned, the course of the postmyocardial syndrome is characterized by its appearance within the first few weeks after the acute infarction, at which time it may be misinterpreted, and by the tendency to recurrences. A single episode of pleuropericarditis might last for 6 weeks, and there have been as many as 6 recurrences in some cases. The number of recurrences determines the duration of the illness, which may vary from a week to 6 months. In a few instances, relapses appeared as late as 2 years after the onset of illness but these late recurrences were seen only in patients treated with steroids for prolonged periods. The severity of the illness varied markedly. In some, the pericardial effusion was massive enough to endanger life by tamponade; in others, the syndrome had no effect on the patients’ well being. In general, however, the prognosis is good if anticoagulant therapy is not administered because of an erroneous diagnosis. In 1 case at least, such therapy produced fatal cardiac tamponade.

Since the illness is usually self-limited, symptomatic treatment with salicylates, analgesics, or narcotics suffices for the majority of cases. Corticosteroids have been found to be almost specific, causing fever and chest pain to disappear within 24 hours. However, rebounds have been observed frequently after withdrawal, even as late as 2 years after the onset of treatment. Therefore, these drugs have been reserved for those patients with excruciating pain or with an unduly prolonged illness.

The cause of this syndrome remains unknown. Attempts to implicate an infectious process, bacterial or viral, have been unsuccessful. Antibiotics have had no effect on the clinical course. Anticoagulant drugs were suspected but cases have been found in which no such treatment was administered and the serous effusions have at times been straw colored.

The clinical features of the postmyocardial infarction syndrome resemble those also seen in the postcommissurotomy syndrome. It has been suggested by some that the latter actually represents a hypersensitivity reaction to antigens produced by the traumatized heart, and the same type of mechanism may underly the postinfarction syndrome. Both clinically and experimentally, allergic pneumonitis and serositis have been observed and reported by Rich and Gregory, Ellis and McKinlay, and Harkavy. It is, therefore, postulated by Dressler that the postmyocardial infarction syndrome may represent a
hypersensitivity reaction to autoantigens that result from necrosis of the myocardium. At times, the resultant inflammatory reaction may be hemorrhagic in character.

As is true with any diagnosis, that of the postmyocardial infarction syndrome can be made only if the condition is borne in mind. In the past, this complication was not recognized because its manifestations were erroneously attributed to other causes. When the fever and pericardial rub appeared early and were prolonged, it was considered that extensive infarction had taken place. Late appearance of fever, chest pain, and pericarditis was thought to represent additional myocardial infarction at that time. Pleuropericardial pain or pleural effusion, especially if associated with hemoptysis or hemorrhagic pleural fluid, was usually attributed to pulmonary infarction. Increase in the size of the heart shadow was explained as due to cardiac enlargement unless serial x-rays, which are usually not taken on patients with recent myocardial infarction, showed it to be due to pericardial effusion.

Recognition of this syndrome should not be difficult when it presents itself with the full-blown clinical picture. In its less classical form, when only the incomplete syndrome appears, it should be suspected, especially if there are recurrences. The diagnosis becomes difficult when fever is the only manifestation or when pneumonitis or pleural effusion is the dominant feature. It may readily be missed if the manifestations of the myocardial infarction are so mild and atypical as to remain unrecognized, and therefore not obviously related to an attack of pleuropericarditis and fever, which appears several weeks later.

Case Reports

Case 1. The patient, a 43-year-old white man with angina pectoris for 1 month was admitted to the hospital on November 3, 1958, complaining of pain in the chest of 2 hours' duration. There were associated sweating, weakness, nausea and apprehension. His blood pressure was 150/100. The heart was not enlarged on percussion, the heart tones were quite distant, and there was a grade-I blowing systolic murmur at the base. The pedal pulses were present. The lungs were clear to percussion and auscultation and the remainder of the physical examination was normal.

The electrocardiograms showed the usual progressive changes of a posterolateral myocardial infarction (fig. 1). The chest x-ray on admission was normal. The serum transaminase was 148.

The patient was treated on the basis of a diagnosis of myocardial infarction with bed rest, morphine, and anticoagulants. He ran a somewhat atypical course. His temperature remained elevated for a total of 16 days and reached a maximum of 102 F. In addition, there was a loud pericardial friction rub first heard on the fifth hospital day and lasting until the sixteenth day of hospitalization. A bedside x-ray of the chest on the tenth hospital day showed considerable enlargement of the cardiac shadow as compared with the film taken on admission (fig. 2). It also showed evidence of fluid in the oblique fissure on the right. This enlargement persisted on serial x-rays until the thirtieth hospital day. There were no physical signs to suggest that the cardiac enlargement was on the basis of congestive failure, and congestive failure would also not have accounted for the prolonged fever or the prolonged pericardial friction rub. There was no evidence of tamponade at any time and the patient had no unusual symptoms after the fourth hospital day. Because of the evidence of more than the usual postinfarction pericarditis, anticoagulants were discontinued in order to avoid the risk of a hemopericardium. The heart size returned to normal, the friction rub disappeared, and the temperature became normal. The subsequent hospital course was entirely uneventful.

Case 2. This 52-year-old man was admitted to another hospital on June 12, 1956, with symptoms and electrocardiographic evidence of an acute anterior wall myocardial infarction. His recovery was uneventful and the expected serial electrocardiographic changes occurred. He was readmitted 2 months later with a 3-week history of frequently recurring pain in the epigastrium radiating to the chest and both shoulders. The pain was worse on recumbency. On admission, a pericardial friction rub was heard and was continuously audible for 6 days. The pain lasted for 4 weeks. The temperature during this period was normal except for the first 2 days of hospitalization when the patient had an irregular temperature elevation to a maximum of 102 F. The sedimentation rate was elevated as was the white count. The chest x-ray on

*We wish to acknowledge the courtesy shown by the Wilkes-Barre General Hospital in permitting us to review the hospital records of this patient.
the seventh hospital day showed a small left pleural effusion, which was gone by the fourteenth hospital day. The heart size remained normal on both films. On admission, an electrocardiogram showed that the previously inverted T waves in the anterior chest leads had become upright. Serial tracings showed a return to the inverted T waves in all precordial leads followed by a return of the T waves to normal in V1, 2, and 3. There was no ST-segment elevation at any time and no Q waves.

Two years later, he was again admitted to another hospital with a 5-day history of substernal pain radiating to the right arm and neck. This pain was not affected by nitroglycerin. A pericardial friction rub was heard from the eighth to the fifteenth day of this hospitalization. The temperature was essentially normal throughout this hospitalization and the serum transaminase was normal on 2 occasions. The chest x-ray was also normal. The electrocardiograms remained stable and showed essentially the residuals of a healed anterior wall myocardial infarction.

The patient was first admitted to the Wilkes-Barre Veterans Administration Hospital on November 22, 1958, complaining of episodes of rapid heart action and substernal pain. He stated that some episodes had been terminated by his private physician by means of pressure on the eyeballs but no electrocardiograms had been obtained at the time of these episodes. On admission, a sinus tachycardia had developed at a rate of 132, the blood pressure was normal, auscultation of the heart was negative, and the remainder of the physical examination was normal. There was no physical or laboratory evidence of thyrotoxicosis. The admission electrocardiogram was compatible with

Fig. 1. Case 1. Electrocardiograms depicting the acute and healed stages of posterolateral wall infarction.
a healed anteroseptal myocardial infarction and serial electrocardiograms showed no significant changes. No episodes occurred during the patient’s hospital stay to suggest paroxysmal tachycardia and it was concluded that the sinus tachycardia present on admission was on a functional basis. The patient responded to reserpine therapy, with symptomatic improvement and with a fall in the cardiac rate to normal.

Comment. This patient had a proved myocardial infarction 30 months prior to our first examination. From the records it appeared that 2 episodes of pericarditis had occurred 2 months and 2 years after the myocardial infarction. These were characterized by prolonged pericardial friction rubs without electrocardiographic evidence of fresh myocardial infarction. Fever and pleural effusion occurred in 1 episode. The transaminase was normal at the time of the second episode. On each occasion, the electrocardiogram was compatible with a healed myocardial infarction without evidence of further destruction of the heart muscle.

Case 3. This 50-year-old white man was admitted to the hospital on October 18, 1958, complaining of pain in the chest and hemoptysis, both of 12 hours' duration. Between 1955 and the summer of 1958 he had had 3 previous hospitalizations, each time for a fresh myocardial infarction. There was no previous history of hemoptysis or of chronic chest disease of any kind, no history of symptoms to suggest phlebitis; and no history of cardiac arrhythmia.

On admission, the blood pressure was 120/90. The heart sounds were distant and there were a few scattered rhonchi in the lungs. The electrocardiogram on admission showed, in addition to evidences of old infarction, generalized S-T depressions, suggesting coronary insufficiency. Subsequent tracings showed return to the pattern of healed infarction. A serum transaminase was normal.

The patient received oxygen, narcotics, and anticoagulant therapy with heparin and coumadin; adequate, but not excessive, anticoagulant levels were achieved. Nevertheless, the patient continued to cough up 2 to 3 ounces of grossly bloody sputum daily. Physical examination disclosed persistent rales at both bases, and digitalis was administered because it was difficult to differentiate left-sided decompensation from what was presumed to be multiple pulmonary emboli. Serial bedside chest films during this period disclosed gradually progressive, disseminated infiltrations in all lung fields (fig. 3). The final chest x-ray, obtained about 18 hours before the patient died, suggested pulmonary edema, but the patient's respiration was relatively easy at this time. During the third hospital day a distinct pericardial friction rub was heard, but none was heard subsequently. Moderate temperature elevation to 102 F. was present throughout the hospital course. On the seventh hospital day dyspnea rapidly increased and the patient expired within 2 hours, despite the usual therapy for left ventricular failure.

It was our clinical impression that the patient had had multiple pulmonary emboli from the right ventricle subsequent to previous infarction involving the septum, and that the coronary insufficiency or myocardial infarction had resulted from the secondary hypotension.

At postmortem examination the heart weighed 450 Gm. The pericardial sac contained 40 ml. of cloudy fluid and there were some fibrinous adhesions between the visceral and parietal pericardium, both anteriorly and posteriorly; these were
broken without difficulty. The right coronary artery was occluded, 2.5 cm. from its origin by atherosclerotic plaques with hemorrhage beneath the intima and a thrombus in the lumen. A similar lesion occluded the left circumflex coronary artery 2 cm. from its origin. The valves on the left side of the heart showed moderate atherosclerotic thickening while those on the right side of the heart appeared normal. The left ventricular wall in its greatest thickness measured 1 cm. and the right ventricular wall measured 0.4 cm. The cardiac muscle showed aneurysmal dilatation in the region of the apex near the interventricular septum. In this area, the ventricular wall was thin, measuring only 3 mm., and intensely fibrotic. The cardiac muscle in other areas revealed a coarse pattern of fibrosis with no gross areas of acute necrosis of the cardiac muscle. No intramural thrombi were present.

The left lung weighed 775 Gm. and on palpation revealed patchy areas of consolidation involving most of the upper lobe and about half of the lower lobe. On section, the consolidated areas had a reddish brown appearance but were not granular and were thought to be resolving infarcts although the vessels of these areas failed to reveal evidence of thrombosis. The uninvolved portions of the lung showed some congestion and edema. The right lung weighed 1,000 Gm. and on palpation revealed nodular areas of consolidation involving primarily the upper and middle lobes and to a lesser degree the lower lobe. On section, the consolidated areas were rather ill defined and had a reddish brown color but were not granular. The pleural surface involved in the consolidated areas was not elevated or intensely congested. The bronchi of both lungs contained some blood-tinged sputum. The blood vessels appeared normal and showed no evidence of thrombosis.

On histologic examination, an epicarditis was seen corresponding with the fibrinous adhesions noted grossly. Sections of the coronary artery showed an organizing thrombus with some fresh thrombus grafted upon the partially organized lesion. The adjacent myocardium revealed areas of necrosis infiltrated by neutrophilic leukocytes accompanied by an intense fibrinoblastic reaction. In addition, there were focal areas of subendocardial necrosis accompanied by acute inflammation.

The lungs showed fibrin plugs within the alveoli with areas of moderate infiltration by neutrophilic leukocytes. In many areas there was fibrinoid swelling and necrosis of the alveolar walls, and a hyaline membrane lined many of the alveoli. Fibrinoid changes were seen in the walls of some of the smaller arteries. There was proliferation of some of the septal cells forming a low cuboidal-cell lining with many of the alveoli containing a mononuclear exudate as well as many pigment-laden phagocytes. Many of the alveoli contained organizing fibrinous plugs containing desquamated alveolar lining cells. In many areas, the alveolar walls were thickened and infiltrated by mononuclear cells and occasionally by neutrophilic leukocytes. Occasional focal collections of lymphoid cells were noted (fig. 4).
POSTMYOCARDIAL INFARCTION SYNDROME

Fig. 4. Case 3. Microscopic picture of the lungs showing (1) hyaline membrane; (2) desquamation of lining cells and accumulation of mononuclear exudate; (4) fibrinoid necrosis of the alveolar walls.

At the time of autopsy, it was thought that the pulmonary changes might represent organizing infarcts. Histologic study, however, failed to confirm this impression and no occlusion of the pulmonary vessels could be found. The microscopic picture resembled that seen in atypical or viral pneumonia, and was similar to the changes described by Geever et al. as the atypical inflammatory reaction appearing in the lungs following acute myocardial infarction.

Comment. This patient had had at least 3 myocardial infarctions subsequent to 1955. His present episode began 1 week prior to death and was characterized by substernal pain, a pericardial friction rub, an electrocardiographic pattern of old myocardial infarction with superimposed coronary insufficiency, and profuse persistent hemoptysis. Physical and radiologic evidence suggested progressive, massive consolidation of both lungs; it was probably interstitial in its early stages, as suggested by the disproportion between radiologic consolidation and physical findings. Contrary to the expected behavior of pulmonary infarction, this lesion manifested itself by early hemoptysis and by progression in the face of anticoagulant therapy. The resemblance of this patient's x-rays to those of Dressler's patient with hemorrhagic bronchial pneumonia is striking.

Postmortem examination confirmed the absence of pulmonary emboli or of a source for pulmonary emboli. There were no postmortem findings in the pericardium that could be correlated with the pericardial friction rub that was heard early in the patient's course.

Case 4. This 64-year-old patient was first admitted to this hospital in 1954 with an anterior wall myocardial infarction. In late 1955 he was at another hospital with chest pain but no hemoptysis and was treated for pneumonia at both lung bases. The electrocardiograms at that time showed the residue of his myocardial infarction. In the spring of 1957, he was hospitalized with substernal
pain and hemoptysis. The hemoptysis began 48 hours after the onset of substernal pain. At that time, he had the progressive electrocardiographic changes of a fresh myocardial infarction as well as infiltration at the base of the right lung. The patient recovered without anticoagulant therapy, which was withheld because of a duodenal ulcer that had recently bled. The clinical diagnosis was myocardial infarction followed by pulmonary infarction. Six months later, he was readmitted with substernal pain and left pectoral pain, which was worse on inspiration. Hemoptysis, which had not been noted since his last admission, developed very shortly after the chest pain and totaled about 2 ounces in 3 days. On admission, the heart sounds were of poor quality and the lungs were clear on auscultation. There was no evidence of thrombophlebitis at any time. Serial electrocardiograms showed a third myocardial infarction but the chest x-ray revealed no pulmonary infiltration to correspond to the hemoptysis. The patient received anticoagulant therapy on this occasion and hemoptysis continued for a period of 1 week. The patient was readmitted to the hospital on December 15, 1958, with ulcer symptoms. There had been no clinical or electrocardiographic evidence of further myocardial infarction in the 14-month interval and no further hemoptysis.

Comment. Between 1954 and 1957, this patient had 3 myocardial infarctions. One year after the first infarction, he was treated for “pneumonia” without hemoptysis and without further evidence of myocardial infarction. The second and third episodes of myocardial infarction were accompanied by rather profuse and prolonged hemoptysis which, on the last occasion, began only a few hours after the onset of the symptoms of myocardial infarction. During the 5-year period of this patient’s heart disease, hemoptysis had occurred only on these 2 occasions. It was the clinical impression, at the time of these 2 episodes, that this patient had had myocardial infarction and pulmonary infarction occurring almost simultaneously. It is our impression now, that this patient had myocardial infarction complicated by a hemorrhagic bronchopneumonia and that pulmonary infarction did not occur. The therapeutic implications, particularly with regard to anticoagulant therapy, are apparent.

DISCUSSION

Four patients have been presented with past histories of myocardial infarction who then developed unusual manifestations during the postinfarction period. These manifestations occurred as early as 5 days after the infarction and as late as 2 years after the infarction. They included prolonged or recurrent pericarditis in 2 cases and hemorrhagic pneumonia in 2 cases.

Therefore, there were 2 of Dressler’s manifestations noted in our 4 patients. None of our patients had pleural effusion. Three of the 4 patients were febrile beyond the period of acute myocardial infarction and 1 had chest pain which could not be accounted for by infarction (case 2).

In treating myocardial infarction, it is standard practice to be aware of certain complications. Some of these (brief pericarditis without effusion, cardiac failure, and particularly pulmonary embolism) may mimic the manifestations described by Dressler. Failure to recognize this syndrome resulted in misdiagnosis in 2 of our 4 patients. A third patient was properly diagnosed as suffering from prolonged pericarditis with effusion (case 1), and a fourth suffered the major manifestations of his illness at another hospital.

Our errors and the resulting therapeutic implications may be summarized as follows:

Two of our patients developed hemoptysis and pulmonary infiltrations within a few hours after the substernal pain that signaled the onset of the infarction. One of these patients behaved in this fashion on at least 2 and perhaps on 3 occasions (case 4). In both patients we made a diagnosis of almost simultaneous pulmonary and myocardial infarction, attributing the pulmonary infarction to the previous history of arteriosclerotic heart disease and the myocardial infarction to the fall in blood pressure consequent to the pulmonary infarction. The explanation is labored enough, although supported by previous clinical experience; however, it was made even less tenable in 1 patient (case 3) by the clinical and radiologic evidence of progressive hemorrhagic consolidation of both lungs in the face of well-controlled anticoagulant therapy. It is fair to say that the unresponsiveness to anticoagulants was sufficient to call our attention to the true
state of affairs in this patient and that in the other patient the coincidence of pulmonary and myocardial infarction might reasonably have been accepted once, but not twice. The therapeutic implications with regard to anticoagulant therapy are apparent. In addition, Dressler claimed success in treating this syndrome with steroids, which might have been tried in the patient who died of the progressive syndrome.

Two of our patients had pericarditis. The use of anticoagulants in the presence of severe and prolonged pericarditis is probably contraindicated, and it is particularly necessary to be prepared to tap the pericardium should severe tamponade result.

Pleural effusion is less a threat to the patient than are the other 2 complications, and therefore demands less therapy. It is necessary to be aware that the complication exists in order to avoid an erroneous diagnosis of cardiac failure or of pulmonary infarction. Once more the therapeutic implications are apparent.

We have nothing to add to Dressler’s discussion of the possible etiology of these complications, or to his comments concerning the possible relation to similar manifestations following mitral commissurotomy, which also follow damage to heart muscle. If Dressler’s concept of an allergic response to necrotic heart muscle is correct, then it is possible that the syndrome may occur in response to the gradual and progressive necrosis that occurs in some arteriosclerotic hearts without major infarction. This observation may or may not be related to the unexplained pleural effusions that are sometimes found in elderly and arteriosclerotic individuals who have never suffered acute myocardial infarctions. This is admittedly speculation and will require the observation of many such patients with this possibility in mind.

**Summary**

Four patients have been described who had in common a history of myocardial infarction followed, after a varying interval, by unusual manifestations comprising prolonged or delayed pericarditis with or without effusion or hemorrhagic pneumonia without pulmonary infarction. The significance of these events is discussed, particularly with regard to potential misinterpretation as fresh myocardial infarction, pulmonary embolism, and cardiac failure. The therapeutic pitfalls involved in these errors in diagnosis are also discussed. Similar case material previously reported by Dressler is summarized with a discussion of the etiology, as yet unknown, of this syndrome. It is suggested that Dressler’s original concept may require expansion to include patients with myocardial fibrosis on an arteriosclerotic basis but without acute infarction. In view of the ease with which we collected 4 cases of this syndrome after we became aware of it, we believe that these manifestations may not be uncommon.

**Summario in Interlingua**

Es describite 4 patientes qui habeva in commun un historia de infarcimento myocardial, seuque—a varie intervallos—per un serie de manifestationes unusual consistente de prolongate o retardate pericarditis con o sin effusion o pneumonia hemorrhagica sin infarcimento pulmonar. Le significacion de iste evenimentos es discutite, particularmente con respecto al risco de lor interpretation erronee como recurrentia de infarcimento myocardial, como embolismo pulmonar, e como disfallimento cardiac. Le periculo therapeutic inherente in tal errores diagnostic es etiam discutite. Simile observationes, previemente reportate per Dressler, es summarisate. Le question del non ancora cognoseite etiologia de iste syndrome es discutite. Es presentate le suggestion que le conception original de Dressler debe esser expandite de maniera que illo pote includer patientes con fibrosis myocardial a base arteriosclerotic e sin infarcimento. Viste le facto que nos habeva pauc difficultate a trovar 4 casos de iste syndrome post que nos habeva devenite conscie de su existentia, nos crede que le supra-describite manifestationes non es incommun.
REFERENCES


SCIENCE AND LIFE

J. Arthur Thomson
British: professor of natural history, editor and author; 1861-1933

The old discouragement expressed in the saying that increase of knowledge is increase of sorrow has been replaced by a more robust confidence in what science may achieve in the control of life. The modern outlook is expressed in Herbert Spencer's pithy sentence: "Science is for life, not life for science," or in Comte's well-known saying: "Knowledge is foresight, and foresight is power."

Bacon had the idea clearly in mind when he wrote in The Advancement of Learning: "This is that which will indeed dignify and exalt knowledge if contemplation and action be more nearly and straitly conjoined and united together than they have been." And the passage ends by declaring that what is sought in science should be "a rich storehouse for the glory of the Creator and the relief of man's estate." But what is distinctively modern is the ideal of bringing the light of science to bear on man's problems all along the line, on health of mind as well as of body, on education as well as on agriculture, on ethical development as well as on the more economical exploitation and usage of natural resources, on eugenics as well as on eutopias. Just as many ills that the flesh is heir to are met no longer with folded hands but by confident therapeutics, so over a wide range there is a promising application of all kinds of science to the amelioration of the conditions of human life. Great stores of wealth are awaiting the scientific "Open Sesame"; a great heightening of the standard of health will be attainable in a few generations if men of good-will take science as their torch. But wealth and health are the pre-conditions of true progress, which means a fuller embodiment of the true, the beautiful, and the good in lives which are increasingly a satisfaction in themselves.—The Outline of Science, Vol. 4, p. 1165. From Great Companions. Readings on the Meaning and Conduct of Life from Ancient and Modern Sources. Vol. I, Boston, The Beacon Press, 1952.
Postmyocardial Infarction Syndrome
NELSON J. WEISER, MILTON KANTOR and HOLLIS K. RUSSELL

*Circulation*. 1959;20:371-380
doi: 10.1161/01.CIR.20.3.371

*Circulation* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1959 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/20/3/371

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Circulation* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to *Circulation* is online at:
http://circ.ahajournals.org/subscriptions/