Symposium: Pitfalls in Diagnosing Coronary Artery Disease

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Dr. Louis N. Katz, Moderator: The diagnosis of coronary artery disease is a momentous one. Many conditions imitate coronary artery disease. This diagnosis should not be an end product of hasty judgment. To overlook coronary artery disease is bad enough, but in many ways the infliction of this diagnosis on a patient without the disease may be even worse.

My distinguished colleagues will now present their views and afterwards, time permitting, we will have some discussion. The first of the panelists, Dr. Herrman L. Blumgart of Boston, will discuss the pitfalls in the clinical diagnosis of coronary artery disease.

Dr. Blumgart: Dr. Katz, ladies and gentlemen, and my companions in the pit this morning: On a Sunday morning, a discussion of Pitfalls in Diagnosing Coronary Artery Disease might well cite the Book of Jeremiah, Chapter XVII, Verse IX, "The heart is deceitful above all things; who can know it?"

The heightened interest in heart disease during the past decade has led clinicians at times to attribute certain symptoms such as cyanosis, dyspnea, palpitation, chest pain, and edema of the legs to heart disease when, in fact, they are due to extracardiac disease, often of remedial nature. It is therefore worthwhile to review some of the more common errors encountered in clinical practice and to emphasize certain clues that may be helpful in preventing mistakes that lead to a false diagnosis of heart disease.

An instance of how prevalent this is may be exemplified by an interesting report, which some of you probably recall, of Goldwater, Bronstein, and Kesky of the Work Classification Group in New York. (J.A.M.A. 148: 89, 1952.) They re-examined 631 cardiac cripples, who were crippled because of the diagnosis of heart disease, and tried to ascertain the degree of effort that these individuals could undertake without harm. Among the 631 patients examined, they found that 175, or 28 per cent, had no heart disease but had been diagnosed as such and consequently were crippled not by the heart disease but by the diagnosis of heart disease. Most of the instances of erroneous diagnoses were based on faulty interpretation of palpitation, dyspnea, and edema of the legs; in some instances the patients were merely told, "There is something wrong with your heart." The diagnosis of clinically significant coronary heart disease is based on the evidence of congestive heart failure, angina pectoris, myocardial infarction or, in some instances, the occurrence of arrhythmias in the absence of other etiology. The etiologies of angina pectoris, myocardial infarction, and congestive failure are numerous and include rheumatic fever, viral and bacterial myocarditis, the group diseases, amyloid, sarcoidosis, subendocardial fibroelastosis, hemachromatosis, and tumors, both primary and metastatic, as well as, of course, congenital lesions that may announce themselves symptomatically only in adult life.

At this time, however, I should like to emphasize the noncardiac conditions that may mimic the manifestations of coronary artery diseases. The condition most frequently confused with congestive failure is pulmonary emphysema. The presence of orthopnea, palpitation, cyanosis, increasing limitation of work, reduced vital capacity, rales in the chest, and a ptotic liver or low diaphragm...
with palpable, but nontender edge, frequently lead to an erroneous diagnosis of congestive heart failure, especially when coupled with faint heart sounds because of the overlying emphysematous lungs. Similarly, in the instance of the Pickwickian syndrome, namely, reversible cardiopulmonary changes because of extreme obesity with somnolence, stupor, cyanosis, periodic breathing, and polycythemia, loss of weight is usually sufficient to reverse the syndrome and to eradicate all the manifestations of congestive heart failure. In another condition you well know, with increased venous pressure, ascites, large liver, marked dyspnea, the presence of a paradoxical pulse, a normal-sized and quiet heart, no evidence of intrinsic heart disease, and no marked electrocardiographic changes suggesting coronary artery disease, the diagnosis of chronic constrictive pericarditis may be made.

Of great importance, and I am emphasizing purposely the remedial forms of heart disease that may mimic congestive heart failure due to coronary artery disease, we should include the hyperkinetic states such as masked hyperthyroidism, anemia, A-V fistula and, in some instances, Paget’s disease. I make it a rule for myself, and I am sure it is a good one, never to neglect in taking the history and doing the physical examination to say to myself, “Are you sure you are not overlooking hyperthyroidism or myxedema?” It is only in this way that a clue of somewhat bright eyes or slight tremor gives us the diagnosis of the underlying disease responsible for the fatigue, palpitation, dyspnea, the lowered vital capacity, and atrial fibrillation. Not infrequently one is somewhat disturbed to find that a diagnosis has been made of congestive heart failure due to coronary artery disease because of edema of the legs in the absence of other evidence. Obviously, in some instances, the standing position reveals varicose veins. One also must remember that in older people with lax tissues, there is edema on standing, particularly in hot weather. In this day of therapeutic enthusiasm, one must remember that guanethidine, hydralazine, or any of the rauwolfia preparations may lead to slight or even marked edema of the legs, which is reversible on stopping the medication. Similarly, hormones such as the corticosteroids, the estrogens, progesterone, and testosterone may lead to edema of the legs and, if not recognized as such, may lead to the erroneous suspicion of underlying heart failure. Patients with low serum proteins, particularly on ingestion of much salt and fluid, may have considerable edema of the legs.

Now let us take a brief glance at the false diagnosis of acute myocardial infarction and of angina pectoris. Anterior chest pain or pain elsewhere in the thorax or even upper abdomen is frequently misleading. In general, one must remember that approximately one third of angina pectoris patients have no abnormality on physical examination or electrocardiogram and a normal blood pressure. A painstaking history is all important. Contrariwise, even advanced coronary artery disease may not give rise to clinical manifestations. Before discussing other diagnoses I shall mention a remark of Dr. Paul Zoll, who sees many patients with Stokes-Adams disease and heart block. He tells me that he is disturbed that Stokes-Adams attacks are often the sole basis of a diagnosis of acute myocardial infarction, whereas many of these patients have no evidences of acute myocardial infarction, but are nevertheless confined to bed, given anticoagulants, and greatly restricted as though they had had an acute myocardial infarction.

Diagnoses always to be considered in dealing with a person who is suspected of acute myocardial infarction are pulmonary embolism, dissecting aneurysm, idiopathic pericarditis, particularly in these days of viral infections, and acute spontaneous interstitial mediastinal emphysema. Common to both idiopathic pericarditis and acute myocardial infarction is the presence of pain, fever, leukocytosis, and electrocardiographic changes. The difference lies in the association with respiratory disease, and particularly the fact that the fever and the leukocytosis in benign
pericarditis occur with the inception of the attack, whereas in acute myocardial infarction leukocytosis and fever occur usually after some 24 hours. And, as Dr. Kossmann may tell us later, the electrocardiographic changes are very different. Similarly, we must exclude pulmonary embolism and that very benign but rare condition of Hamman’s syndrome, namely, acute spontaneous interstitial mediastinal emphysema. A person may be condemned to a life of prohibition from all types of activity because of a mistaken diagnosis of acute myocardial infarction in the absence of any cardiovascular disease. I need not speak in detail to a sophisticated audience such as this in regard to the differential diagnosis between upper abdominal conditions such as peptic or esophageal ulcer, diseases of the spine and spinal cord with pain on motion, and gallbladder disease, nor of the xiphoid syndrome or Tietze’s syndrome.

I would say in conclusion that the fascination, the challenge, and the despair experienced by every clinician are due in part to the limitless possibilities of error. For every correct diagnosis in a patient, there are dozens of possible mistaken ones. The list of conditions that I have merely mentioned is far from complete. The pains of hiatus hernia, of esophageal ulcers, of herpes zoster in the first 24 to 48 hours before the eruption occurs are a few additional possibilities. Errors will be minimized, however, if one continues to consider the numerous possibilities in differential diagnosis, realizing that the correct diagnosis in obscure cases is usually made mainly by thinking of the diagnosis.

Finally, may I emphasize as did Dr. Ern- stene in his paper, “Explaining to the Patient, a Therapeutic Tool and Professional Obligation,” the opportunity we have to allay apprehension, fear, and invalidism by carefully explaining to the patient and perhaps to his family the nature of his illness and reassuring them regarding the rationale of the therapeutic program.

Dr. Katz: In this modern era, the clinician feels more secure if his clinical diagnoses are supported by laboratory tests. To talk about the pitfalls in that sphere, we have Dr. George E. Burch of New Orleans.

Dr. Burch: One of the important pitfalls of laboratory studies in patients with coronary disease is, I believe, the overreliance on laboratory data. There is a great tendency today to believe that a satisfactory work-up of patients is not possible unless complicated computers, data from charts, or studies from the laboratory are available. There is no one disease that requires a more meticulous history and physical examination and careful discussion with the patient, as Dr. Blumgart has said, than ischemic heart disease or coronary heart disease. One must remember, as Dr. Katz suggested, that the laboratory data obtained on our patients are provided by non-professional people working as technicians in laboratories—unfortunately, some of them with no more than a high school education. A good example of this is the reports on prothrombin time. When the physician increases the dose or decides not to vary anticoagulant therapy, it is done entirely on the basis of the “number” furnished from the laboratory. Thus, the physician is in the position of allowing a high school graduate to guide the treatment of the patient. We depend excessively on laboratories without first and periodically making an effort to evaluate carefully the quality of the laboratory. There are some laboratories that provide accurate data.

One should first be a doctor. If the laboratory data do not conform to the clinical data, the problem should be investigated further by checking for either clinical errors or for errors in the laboratory data. This should certainly be done before a definite decision in diagnosis or an important therapeutic decision is made. We should also recognize, as Dr. Blumgart has said, that many patients have coronary atherosclerotic disease with no symptoms whatever; the laboratory data can be perfectly normal yet the patient, especially if in the older age group, will have diffuse arteriosclerosis with impending ischemic heart disease. The heart disease is often so subtle.
that it is difficult to make a definite diagnosis of ischemic heart disease. It is certainly much more important to try to prevent infarction than to diagnose it after it develops.

We should remember that those patients with changes in the white-cell count do not always have only one disease, especially older patients. They often have complicating diseases that alter some of the laboratory manifestations, particularly the white blood-cell count in patients with congestive heart failure. This is true for patients with associated infections such as urinary tract infections. In fact, coronary episodes are frequently precipitated by diseases such as emotional disturbances, infections, and the like. We therefore must differentiate in the clinical and laboratory data just which changes originate from the coronary disease and which originate from the associated diseases.

In regard to the circulating enzymes measured in the blood, we realize that a great deal of benefit in diagnosis is derived from these determinations. However, there is still much to be learned about them. For example, among the LDH enzymes are several components that are difficult to measure. Some of these components are more likely to be found in association with coronary disease and infarction than are others. Until we know more about measuring these accurately, one must be careful about interpretations. We know that these enzymes increase in concentration in association with diseases outside of the heart. The physician must integrate these laboratory data with all of his clinical data in order to appreciate their importance.

Serum cholesterol, uric acid, and the hematocrit level are important data to obtain but a good doctor finds that in association with a careful history and physical examination, a minimal amount of laboratory data such as blood count, urinalysis, stool examination, fluoroscopic and x-ray studies, as well as electrocardiogram are most important. Other laboratory studies are obtained as indicated. The physician usually obtains the correct diagnosis in most instances without fancy laboratory data. As Dr. Blumgart said, the physician must always also consider things such as hernia, peptic ulcer, and gallstones when a patient complains of chest pain.

Coronary angiography is still in the purely research stage; very important research is being done and it certainly should be continued. From the point of view of a doctor practicing medicine, however, it should not yet be used in the management of his patients. Such studies should be relegated to special cardiology centers which have highly trained people and good equipment. Cardiac catheterization is also highly technical and difficult and better left to these special centers. These procedures as well as others are of no clinical value in the routine care of a patient in private practice.

I can assure you that nothing makes a doctor more humble than the autopsy table. I can also assure you that the circulating enzymes, circulation time, and the like, actually assist very little in the management of patients. If one could follow through to the autopsy table routinely for all patients and not just selected ones, one could learn that these special studies really have little to offer for the average patient. The same applies, I might add, to electrocardiograms and many other laboratory procedures.

Dr. Katz: In selecting Dr. Burch and Dr. Kossmann, I deliberately picked men who are primarily clinicians because I think this is the facet we should stress. Dr. Charles E. Kossmann of New York, will tell you some of the pitfalls in relation to electrocardiography.

Dr. Kossmann: In considering the electrocardiographic pitfalls that the physician may encounter in the diagnosis of coronary artery disease, I must emphasize at the beginning that abnormalities in the electrical behavior of the heart that may occur do not depend on the extent or nature of this arterial disease. Chronic obstructive disease of the coronary arteries may be very far advanced with little change, anatomic or functional, in the myocardium. Conversely, infarction may occur
without any disease of the coronary arteries to account for it. The point to be made is that, in the last analysis, it is the resulting anatomic and functional impairment of the myocardium that determines the form and rhythm of the electrocardiograms obtained, and this impairment need not bear an exact relation to the extent of the disease in the supplying arteries.

Knowledge of this basic truth is necessary for the understanding of some of the four categories of electrocardiographic pitfalls to be discussed.

**Pitfall Category No. 1: Normal Electrocardiogram in Coronary Arterial Disease without Myocardial Infarction.** Every physician knows that coronary disease can exist with the electrocardiogram being normal at rest or even after exercise. Yet it is surprising how often the coronary bill of health is declared clean because of these negative findings.

An example (fig. 1) of a patient with stenotic arteries without myocardial infarction was a 45-year-old white man with mild angina on effort of 2 months' duration. The electrocardiogram at rest was not beyond normal limits. In contrast (fig. 2) exercise produced rather characteristic changes in form, particularly of the final ventricular deflections. This man's electrocardiogram remained normal for 7 years after the onset of the anginal syndrome. To conclude that there was no significant coronary arterial disease simply because the functional elements of the heart, the myocardial cells, were not lost in sufficient number to cause distinctive alterations in the ventricular deflections at rest possibly contributed to his premature demise. The patient died a few hours after an emergency but routine appendectomy under inhalation anesthesia.

A subdivision of this category of pitfall includes the numerous sources of error that can creep into the performance and interpretation of the electrocardiographic exercise or other stress test. They are of two principal types: those resulting from faulty technic, and those resulting from faulty interpretation. Time does not permit any significant treatment of this large and important subject. However, the principal technical pitfalls usually lie in the inadequacy of the exercise or stress used, the use of leads not best suited for recording the effects of the induced relative ischemia, or the making of records long after the effects of the stress have worn off. The recent use of continuous recording during exercise promises to overcome the last of these* but has brought with it additional problems in interpretation in the differentiation of the normal from the abnormal response.

Pitfall Category No. 2: Normal Electrocardiogram in Coronary Arterial Disease with Myocardial Infarction. This category is believed to be rare despite claims to the contrary. It must be distinguished from a large group in which the electrocardiogram, though not normal, is abnormal in a way that is not characteristic of localized lesions. It is estimated that as high as 30 per cent of patients with infarcts recognized at necropsy may have demonstrated only nonspecific electrocardiographic abnormalities during life.

Any statement* that ventricular infarction, particularly of certain regions such as the left lateral wall or the subendocardium, can occur without electrocardiographic abnormalities, cannot be made with any confidence unless continuous recordings or observations on an oscilloscope are made for the 24 or more hours immediately following the clinical episode, and only if a sufficient number of leads have been made in seriatim thereafter.

A pitfall of a related but slightly different kind can be illustrated by an example of what may be called "impending infarction." The patient had a clinical infarction in 1945 and the bipolar leads at the time (fig. 3, top, dated August 28, 1945) were fairly characteristic. Ten years later he came to his physician complaining of pain regarded as being of coronary origin. An electrocardiogram (fig. 3, bottom, dated November 15, 1955) was made when the patient had no pain. No chest leads were made in the haste to get him to the hospital. Two days later the record was normal (fig. 4). Because minor twinges of pain continued, he was kept at rest, and 3 weeks later (fig. 5) evidence of necrosis finally appeared after a slightly longer and more intense bout of pain.

The case illustrates the electrocardiographic capriciousness that accrues from a slowly progressive coronary stenosis and myocardial necrosis occurring over a period of a little more than a month. It illustrates the pitfall


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Electrocardiograms of patient R.C., made 2 days after the records shown in the lower part of figure 3. There are no deviations from normal. (From Kossmann, C. E.: J. Chron. Dis. 4: 434, 1956.)

of drawing a conclusion of any kind from a single record made in the course of a decidedly unsteady state.

Pitfall Category No. 3: Electrocardiographic Simulation of Coronary Artery Disease by Noncoronary Myocardial Disease. The number of examples that fall into this category are legion. They include the electrocardiogram in an infinite number of diseases of the myocardium (inflammatory, parasitic, neoplastic, congenital, metabolic, unclassified) as well as the electrocardiogram made abnormal by drugs, physical agents, emotional states, body contours, and unknown factors. It is probable that the penchant of the average physician to think immediately in terms of coronary artery disease whenever the electrocardiogram shows some abnormality in form is the basis for this common pitfall.

Whenever there are abnormalities in configuration characteristic of infarction in all three major divisions of the ventricular electrocardiogram—QRS deflections, S-T segment, and T wave—the diagnosis of infarction can be made with a great degree of confidence. Only rarely will the combination indicate some other lesion such as a tumor. But if there are abnormalities only in two of these divisions, and particularly if both involve only the final ventricular deflections, the diagnosis of infarction or of lesser degrees of coronary arterial disease must be made with extreme caution. Only a few examples to illustrate this well-known pitfall will be shown.

The first of these patients was a man ad-
mitted to the hospital in congestive heart failure. The teleroentgenogram disclosed a large heart with a shadow in the right lung (fig. 6). The electrocardiogram disclosed abnormalities (fig. 7) in both the initial and final ventricular deflections. The clinical diagnosis was infarction of the myocardium with infarction of the right lung; the diagnosis at necropsy was primary carcinomata of the lung with neoplastic infiltration of the myocardium.

The second patient was a man who showed an enlarged heart with congestion of the lungs (fig. 8), and abnormalities of the final ventricular deflections (fig. 9) on admission to the hospital. The diagnosis, on the basis of these findings, was myocardial infarction with congestive heart failure. The subsequent course and the subsidence of the roentgenographic (fig. 10) and electrocardiographic abnormalities left no doubt that all of the findings were on the basis of acute diffuse glomerulonephritis.

The third patient (fig. 11) illustrates the electrocardiographic effects of diphtheritic myocarditis that could be confused with those produced by certain types of ischemic necrosis.

Pitfall Category No. 4: Abnormal Electrocardiogram with a Normal Heart. This, in a sense, is the most serious pitfall, for it may result in a diagnosis of a disease that is not present, and in the usual chain of undesirable iatrogenic complications.

All physicians have seen examples: minor degrees of prolongation of the P-R or QRS intervals; abnormalities of the T wave in the asthenic or emotional individual; certain types of anomalous atrioventricular excitation; disturbances of rhythm, which may occur and recur in a normal lifetime, often with the post-tachycardia syndrome.

I will show only one example. This 57-year-old man (fig. 12) was admitted for pain in the chest following an automobile accident. Although the steering wheel had struck him in the chest, it was not considered that this had been severe enough to cause contusion of his heart. The Q wave and elevated S-T segment in lead V2 and the abnormalities in higher midprecordial leads resulted in a clinical diagnosis of myocardial infarction. When the record remained stationary, suspicion of error became high. With investigation it was learned that an electrocardiogram had been made 5 years before. Acquisition and inspection of this one revealed that it was identical with the one shown. Several examples of this type of "normal" have been published.

In summary, then, four major categories and several subcategories of electrocardiographic pitfalls in the diagnosis of coronary disease have been enumerated.

These pitfalls result from a single basic error—the faulty practice of making an anatomic diagnosis from an electrocardiographic record. To avoid the error the physician must keep in mind that when he says that an ele-

Figure 6
Patient P.L., Negro man, age 40. Teleroentgenogram of chest with irregular pulmonary shadow and pleural adhesions on the right. The heart is enlarged. The marker, A, was put on the chest for another reason.
trocardiogram indicates a certain myocardial disease, he has skipped a correlative step, and is not taking adequate cognizance of the fact that the electrical phenomena on which he is basing his diagnosis may be caused by a great many variables other than anatomic deviation of the heart from normal.

To overcome the pitfalls, therefore, a diagnosis of coronary arterial disease should not be made or excluded on the basis of the electrocardiographic findings alone.

Dr. Katz: The final speaker is Dr. Earl N. Silber of Chicago, who will undertake the task of showing the pitfalls in attempting to integrate clinical, laboratory, and electrocardiographic findings.

Dr. Silber: My purpose here will be to highlight some of the things that may be helpful in integrating the clinical data and avoiding pitfalls. For this purpose, coronary artery disease may be divided into four clinical phases: (1) asymptomatic coronary artery disease, (2) angina pectoris, (3) pre-infarction syndrome, (4) actual myocardial infarction. This classification is not meant to imply that the natural history of coronary artery disease necessarily evolves in this order.

We shall first consider asymptomatic heart disease, coronary in origin. Asymptomatic patients are of three kinds. First are those who have no symptoms and who have a normal resting electrocardiogram and no other laboratory tests that suggest coronary artery disease. The second group of asymptomatic patients have had a recognized myocardial infarct and are subsequently asymptomatic. The third group consists of patients who apparently have had silent myocardial infarction and present only electrocardiographic evidence of this past event. How do we recognize the first type of patient with asymptomatic coronary artery disease? Firstly, one should survey patients with respect to those features that appear to increase coronary risk. The Framingham Study and other epidemiologic investigations have clearly delineated that such factors as obesity in excess of 20 per cent of one's weight at the age of 20, a cholesterol level in excess of 285 mg. per cent, or the presence of hypertension, diabetes, or

Figure 7
Serial electrocardiograms of same patient shown in figure 6. The bipolar (I, II, III), augmented unipolar (aVp, aVp, aVp) and precordial leads (V1, V2, V3 at half normal) were recorded on January 6, 9, 13, and 18, 1948 (first four horizontal lines). On January 26 (last line) leads V2, V3, and V4 were recorded in addition to the others. There are abnormalities of the initial and final ventricular deflections that were erroneously ascribed to infarction of the heart, and actually were the result of malignant infiltration of the myocardium by a primary carcinoma of the lung.
myxedema increase the risk. Secondly, in this assessment, one should explore the family history. Is there a history of hypercholesteremia, of premature arcus senilis, of xanthomatosis, or overt heart attacks in relatives? These, too, will suggest that the patient may represent an increased risk of developing clinical coronary artery disease. The next step in such patients is properly to obtain the exercise electrocardiogram. What are the circumstances under which, despite the best technics and the most skillful measurement of the deflections, one may still end with the false positives? It is known that digitalis will give false-positive exercise tolerance tests. This drug should be withdrawn for at least a week. In the presence of combined hypertrophy or left or right ventricular hypertrophy in the electrocardiogram, false-positive tests may occur. Patients who hyperventilate may produce records that simulate ischemia. The presence of hypertension may give false-positive exercise tolerance tests. Finally, women in their forties, with or without hypertension, or elderly patients without significant coronary artery disease may have false-positive tests, apparently as an index of their lack of training for this type of physical exertion. Whether or not this constitutes coronary insufficiency is a question. However, if these pitfalls are avoided and the test is positive in an individual of this coronary-prone group, I think that one can have some reasonable certainty that this patient should be subjected to prophylactic measures.

What if under these circumstances the test is negative; what does that prove? It proves not that the patient is free from coronary artery disease, but that, from a functional point of view, he either has segmental disease or that he has sufficient collateral circulation, despite coronary artery disease, to perform
Figure 10

Teleroentgenogram of same patient shown in figures 8 and 9 made on April 15, 1955. The heart has decreased in size, and the evidence of congestion of the lungs has subsided. A trace of pleural fluid persists.

Figure 11

Electrocardiogram in diphtheritic myocarditis recorded Feb. 9, 1959. The patient, a 38-year-old narcotics addict, was admitted with diphtheritic pharyngitis on Dec. 28, 1958. The electrocardiogram then was normal. Note that the lower row of simultaneous precordial leads was recorded with the left-sided leads above the right-sided leads.

The test without clinical or electrocardiographic evidence of ischemia.

As to the pitfalls of angina pectoris, the other panelists have noted that pain that comes on with exertion and is relieved by rest need not be coronary artery disease. Pain that is relieved by nitroglycerin need not be coronary artery disease and, conversely, many patients with bona fide angina pectoris are not benefited by nitroglycerin. It is evident then that what I have said about the role of the exercise electrocardiogram in asymptomatic disease would be useful in these circumstances.

In regard to the preinfarction syndrome, it is important, as Dr. Blungart has pointed out, to recognize that the preinfarction syndrome, or acute coronary failure, or imminent myocardial infarction, as it has been called, represents a definite anatomic, as well as physiologic change, in the status of angina pectoris. What pitfall is there to avoid here? If the electrocardiogram is normal or unchanged, it would be a grave mistake to assume that nothing has changed in the patient. This group of patients requires being treated as if they had suffered actual myocardial infarction because it is recognized that the incidence of such an event within hours, days, or weeks after the onset of this syndrome is very high; on the other hand, in those who are put to rest in bed and placed on anticoagulants, a significantly smaller number of these patients go on to clinical myocardial infarction.

Finally, in regard to overt myocardial infarction, I think it is not an exaggeration to state that it is here that the determination of
Electrocardiograms of a 57-year-old white man who had recently been struck in the chest by the steering wheel in an auto accident. Leads taken at the horizontal level of the third intercostal space at the sternal edge (3V₁ to 3V₆) show to better advantage the elevation of the S-T junction and segment seen only in lead V₂ of the conventional precordial electrocardiograms. A control electrocardiogram 4 years earlier showed an identical form of QRS and T in the same leads. (From Kossmann, C. E.: Advances in Electrocardiography, New York, Grune & Stratton, 1958, Chap. 8.)
serum has its enzymes greatest usefulness. It had been hoped that vectorecardiography might be helpful in patients with hypertrophy of the ventricles, bundle-branch block, or Wolff-Parkinson-White syndrome, but that has not been our experience; the vectorecardiogram has proved of value primarily in those instances of healed myocardial infarction, where one can rely much more on the abnormality of the initial deflection of the vector loop than on the Q wave in the traditional scalar electrocardiogram. While the vectorecardiogram will disclose ischemic injury by failure of the terminal portion of the loop to return to the isoelectric line, there is no advantage of this over the S-T deviation in the scalar electrocardiogram.

We have not reached the point in diagnosing coronary artery disease where laboratory tests will replace judgment and broad knowledge of the pitfalls that one may encounter in using these tests.

**Dr. Katz:** Now I would like to call on each of the panelists for any additional remarks they care to make; Dr. Burch?

**Dr. Burch:** I might add something concerning the vectorecardiogram. I absolutely agree that the vectorecardiogram has yet to be established to be of value in the routine care of patients. I do believe, however, that we do encounter patients who have infarcts involving parts of the heart that are depolarized late in the electric cycle so that they would not reflect changes in the early parts of the QRS complex. In order to detect these electric alterations one must use technics of recording vectorecardiograms, such as have been proposed by Dr. Langer in Philadelphia, or the vectorecardiogram, which spreads out the complexes in great detail. Thus, alterations in depolarization that occur in the mid-part or in the late part of the electric cycle can be noted. I am not suggesting that everyone should have a vectorecardiograph; that is not necessary. I do believe that the vectorecardiogram can help in localizing infarcts that are not readily detectable by conventional methods of electrocardiography.

**Dr. Katz:** Dr. Kossmann, any comments?

**Dr. Kossmann:** Dr. Katz, I did not mean to belittle the electrocardiogram. I, in contrast to Dr. Silber, think it is still the most valuable single laboratory aid available in the diagnosis of coronary disease. One reason for this opinion is that my experience with the enzymes has been a little bit disappointing. I presume that part of the problem is that we probably don't determine them often enough. Acute infarction is a changing situation and unless one is lucky enough to catch the enzymes at the right time, one may miss them. I am always amused a little bit by our efforts to refine our laboratory procedures to a very sharp edge which might increase our diagnostic capability a few percentage points. Actually, when you sit down to think about it, and I hope Dr. Blumgart will agree to this, the easiest way to make a diagnosis of coronary artery disease is to ask the patient. I would say that upwards of 90 per cent will tell you that they have coronary disease by the symptom complex that they present, and it seems a little bit odd that we should try to make this diagnosis with refinements when we can do it so easily by asking the patient what his symptoms are. Now, I do not mean to discourage any efforts to improve our capability at the laboratory level, but at the same time this is a fairly easy clinical diagnosis to make in most cases by asking the patient, "What are you complaining of?" And he will tell you.

**Dr. Katz:** Dr. Blumgart, Dr. Kossmann said you would agree with him; do you?

**Dr. Blumgart:** I should like to make one comment on an important group of cases in which one cannot be certain initially whether or not they have symptomatic coronary artery disease. Actually, it doesn't make a great deal of difference over the next months because in any event you are going to instruct this man of 55, 60, or 65 in regard to a more hygienic form of life. You will tell him to avoid playing squash, handball; you will give him the same advice that is just good common sense, whether you feel he has very mild an-
gina precipitated on extreme exertion or whether you think he has no angina pectoris but ought to take sensible precautions that any man of his age should take. It is in this group that one can produce crippling fear by indulging in the refinements of technics; that, moreover, may give you false information.

Dr. Katz: Dr. Silber, any comments?

Dr. Silber: I would like to point out with respect to Dr. Kossmann's comment that it was not my intent to convey the impression that the electrocardiogram has been supplanted by enzyme studies over the whole spectrum of coronary artery disease. That certainly is not the case. When it comes to the matter of which test is more likely to stand one in good stead in any given case of acute myocardial infarction, there, again, one cannot be dogmatic. Very often when the electrocardiogram is equivocal, the enzymes are also equivocal. But, as Dr. Burch has indicated, efforts are going on to refine and extend the usefulness of the enzymes tests, particularly to find tissue-specific enzymes. In other words, it is already recognized that LDH is not just one such enzyme, but a spectrum of enzymes in which LD5 is apparently almost entirely cardiac in origin and the others are, from our point of view, "nonspecific" coming from other tissues. This whole area requires further exploration and it would be personally precarious for me, coming as I do from Dr. Katz's department—as well as scientifically unsound—to deny the enormous clinical usefulness of the electrocardiogram in the whole spectrum of coronary disease.

Dr. Katz: Any other comments from anyone else on the panel? If not, I will take a few minutes to close.

There are many things that we could have covered if we had had more time. I think, however, it is quite clear from what we have said that the most important error or pitfall is that of omission. If the clinician will think of all diagnostic possibilities, there will be fewer pitfalls. I think, secondly, from what we have heard, that one cannot ignore positive findings. In other words, a positive finding cannot be dismissed lightly; it must be considered and evaluated with respect to the whole spectrum of evidence. A negative finding does not deny a positive one; it may modify the interpretation. Sometimes a negative observation must be dismissed when it cannot be reconciled with the positive ones. One cannot, however, dismiss a positive finding without good cause.

I think it is quite clear that we must avoid the pitfall of too much reliance on so-called ancillary findings, whether they be in the electrocardiogram or in laboratory tests. It seems to me that the diagnosis of coronary heart disease is to be made by a clinician ultimately and by no one else. I would hope you will go away with the idea that you as clinicians are primarily responsible for your case, and with responsibility comes respect for misjudgments.

Finally, it seems to me that with the increased popularity of epidemiologic studies, one must be careful that the criteria used in such studies on large masses of people to define the coronary-prone (high risk) patient, should not be applied without proper modification to single individual patients.

I hope that in another 5 or 10 years a panel similar to this one will be held and that at that time many gaps which we have left unfilled can be more completely covered.

Finally, I want to thank the panelists for their cooperation and for their splendid performance under difficulties. We of the panel wish to thank the audience for being so cooperative, patient, and kind to us. Thank you very much.
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