Myocardial Infarction Patterns in Young Subjects with Normal Coronary Arteriograms

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Over the years the alterations in the degree and sequence of myocardial depolarization and repolarization, which are produced by infarction, have been clearly documented. Similar patterns also may develop during the active stages of pericarditis and myocarditis, in unusual forms of heart disease such as accompany muscular dystrophy, sarcoidosis and amyloidosis, and in certain metabolic and neurogenic disorders. It generally is not difficult to recognize causative factors when these electrical changes are correlated with other clinical manifestations. Diagnostic dilemmas do arise, however, when the latter are obscure or absent.

This report, animated by the discovery of the electrocardiographic patterns of infarction in six white males 16 to 24 years of age who had no stigmata whatever of coronary artery disease, is an appurtenant example of the confusion that arises when an electrocardiographic indictment is not supported by other manifestations and of the difficulties encountered in resolving such a problem. The total experience, however, is recorded for additional reasons. The patients comprising this report are singularly bound to each other by youth, comparable historical backgrounds, an auscultatory abnormality, electrocardiographic deviations, and the capacity for vigorous activity. Hence, they seem to represent an obscure congenital defect that has not been previously recorded. Secondly, this appears to be one of the earliest attempts to resolve the paradox incidental to the discovery of the electrocardiographic patterns of myocardial infarction in patients who are otherwise well by means of a maturing investigative procedure, namely, coronary arteriography.

The format of the presentation includes a summary of the pertinent clinical features, the results of the special investigative studies, and a review of the factors possibly responsible for the finding described.

Clinical Features

The medical records of the six patients included in this report did not disclose early suspicions of a congenital cardiac lesion or past affliction with diphtheria, overt rheumatic activity, unusually persistent or recurrent infections, or hypersensitivity states in any of the group. In one individual rheumatic activity was suspected at age 6, but not confirmed after a careful clinical and laboratory survey. Another of the patients suffered from bronchopneumonia at age 10.

When observed, all of the subjects were and remained asymptomatic within the normal requirements of their types of life. Three of the patients were students, two were commercially employed and one was a professional athlete of considerable renown.

The physical examinations revealed that all of the patients had normal body structure with appropriate height-weight ratios. Chest deformities were not evident. One abnormality, a murmur, was common to the entire group. This was first discovered in each subject during childhood or adolescence and, focusing attention upon the possibility of a cardiac lesion, immediately or subsequently initiated the electrocardiographic examination. Each of the murmurs was systolic, ejection in type, grade I to II in intensity and was heard best to the left of the sternum at the third interspace (fig. 1). In three of the patients the murmur was of equal intensity at the apex.

The cardiac sizes and contours were uni-

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formally within normal limits in the postero-

anterior, oblique, and lateral roentgen views. The pulmonary vascular markings likewise

were not unusual.

In three patients the electrocardiographic anomalies involved the depolarization waves and thus were suggestive of combined myocardial necrosis, injury, and ischemia involving the anterior or posterior surfaces of the left ventricle (figs. 2 and 3).

The electrical deviations in the remainder of the subjects were limited to the repolarization waves of the anterior or posterior surfaces of the left ventricle (figs. 4 and 5).

The routine laboratory evaluation inclusive of urinalysis, complete blood cell count, sedimentation rate, blood sugar, blood urea nitrogen, cholesterol, and serology was uniformly within normal limits.

**Special Investigative Procedures**

High and lateral electrocardiographic leads, taken in each of the patients, failed to clarify the basic abnormalities previously described.

Supported by the knowledge of the asymptomatic state of these individuals, Master two-

step exercise tolerance tests were performed in one subject with depolarization and in two

with repolarization wave abnormalities. These were not altered.

In each of the patients with repolarization deviations, the parenteral administration of

atropine sulfate, epinephrine, and potassium chloride failed to change the basic pattern.

The vectorcardiograms were abnormal in each of the patients, but failed to reveal any abnormalities of the ventricular conduction system. The variations in the initial forces and the inscription of the T-wave loops in specific tracings were indistinguishable from those encountered in coronary artery insufficiency.

In the five patients in whom a right heart catheterization was performed in order to eliminate the possibility of a congenital defect, there was no evidence of a left-to-right shunt or of a gradient across the tricuspid or pulmonary valves. All of the right ventricular end-diastolic pressures were within normal limits.

Without exception, visualization of the coronary arteries failed to reveal ocluding or obstructive lesions of the major or peripheral vessels (figs. 6 and 7).

In a single patient, a left ventriculogram was performed in order to ascertain whether incompetency of the mitral valve was responsible for the murmur described. The result of this study was normal.

In each patient, lupus erythematosus preparations, antistreptolysin determinations, and the cardiac enzyme concentrations were negative or within normal limits.

**Discussion**

In patients in whom abnormal repolarization was an exclusive finding, the deviations appeared as symmetrically inverted T waves in the anterolateral or posterolateral leads. Since these were not initiated by digitalis medication and were unaltered by position,
respiration, exercise, or the parenteral administration of drugs influencing the function of the sympathetic or parasympathetic nervous system, they could not have developed as a consequence of extracardiac physiologic or pharmacologic stimuli. Furthermore, they did not resemble the changes recorded in the so-called "juvenile" pattern. The latter usually consist of T-wave inversions alone or in association with striking elevations of the RST segments in the leads from the right side of the precordium. Even on those infrequent occasions when they are recorded in the mid and left precordial leads, they tend to disappear with exercise.

The very configuration of the depolarization waves, which appeared in the three remaining patients, precluded the possibility that they
were normal variants. Furthermore, extracardiac physiologic and pharmacologic stimuli are not known to produce this type of abnormality.

In considering the possibility that the QRS variations developed as a result of the Wolff-Parkinson-White (pre-excitation) syndrome, it is pertinent to note that none of the patients had an inherent tendency to episodes of paroxysmal heterogenic rhythms. Furthermore, short P-R intervals of 0.12 second or less and prolongations of the QRS complexes to 0.11 second or more were not encountered. In addition, vector analysis did not disclose delays.
in ventricular conduction. Finally, delta waves were not discernible. These facts together with the additional observation that normalization of the QRS complexes did not follow exercise or the parenteral administration of atropine sulfate strongly support the contention that the pre-excitation syndrome was not responsible for the recorded abnormalities of depolarization.

The etiologic role of abnormalities in the distribution or patency of the coronary circulation was appraised by arteriography. The reliability of this procedure in disclosing obstructive lesions is currently undergoing critical evaluation. The method employed in these examinations, namely, aortic root flushing, has already been correlated with the clinical, electrocardiographic, and pathologic findings in patients with overt myocardial infarction. These initial observations imply that the arterial changes responsible for infarction are visualized consistently. Selective coronary arteriography may be even more satisfactory for this purpose. Admittedly, however, with either technic coronary arteriograms cannot be relied on completely to rule out pathology of the coronary vessels. Lesions discretely positioned in the peripheral arteries and those

Figure 6
Coronary arteriogram obtained in patient A.S., age 21, indicating normal visualization of coronary arterial system.

Figure 7
Coronary arteriogram obtained in patient D.F., age 19, indicating normal visualization of coronary arterial system.

that produce minimal symmetrical narrowing certainly may escape detection. Yet it is unlikely that modest pathology of this type ac-
counts for the significant electrocardiographic abnormalities that were documented in these patients. In terms of present knowledge, therefore, it appears reasonable to conclude that gross obstructive coronary arterial lesions did not exist.

The possibility remains that noncoronary myocardial disease was present in each of these patients. This conclusion is not supported by past or present evidence of infections, collagen disease, amyloidosis, hemochromatosis, muscular dystrophy, or metabolic abnormalities. It is a simple matter to reason that the electrocardiographic changes stem from a healed myocarditis. If true, additional examples should be forthcoming in this age of viral infections. Confirmation of this diagnosis and that of fibroelastosis or endomyocardial fibrosis actually is warranted without tissue studies.

**Summary**

For the present, the primary obligations of this communication are to acknowledge that the electrocardiographic patterns of myocardial ischemia, injury, and necrosis may appear in asymptomatic, young white males who have low-intensity systolic murmurs; to record that any implication that these electrical deviations result from obstructive coronary disease cannot be substantiated by coronary arteriography; and to suggest that individuals possessing these diagnostic features may represent a distant, recognizable clinical entity the etiology of which remains obscure.

**References**


Observers, then, must be photographers of phenomena; their observations must accurately represent nature. We must observe without any preconceived idea; the observer's mind must be passive, that is, must hold its peace; it listens to nature and writes at nature's dictation.

But when a fact is once noted and a phenomenon well observed, reasoning intervenes, and the experimenter steps forward to interpret the phenomenon.—Claude Bernard. *An Introduction to the Study of Experimental Medicine*. New York, The Macmillan Company, 1927, p. 22.
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