Special Article

Thoughts About the Abnormalities in the Electrocardiogram of Patients with Acute Myocardial Infarction with Emphasis on a More Accurate Method of Interpreting S-T Segment Displacement: Part II

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Problems Related to the Correlation of Data

We learn by correlating the data collected by the use of one technique, with the data collected by the use of other techniques. Although much has been written about the subject, there is still an inadequate amount of information derived from correlating the abnormalities noted in the electrocardiogram with the abnormalities found in the autopsies of patients who died of myocardial infarction or with the findings from coronary arteriography.

Dr. Michael Gravanis, an expert cardiac pathologist, points out:

*“There are three different types of myocyte necrosis. They are: coagulation; contraction band necrosis, which is secondary to reperfusion; and apoptosis.”

The correlation of abnormal Q-waves in the electrocardiogram with the location of the necrotic areas in the ventricular myocardium has been done many times. Unfortunately, the anatomic position and orientation of a heart that is held in the pathologist’s hand may or may

not precisely reproduce the actual position and orientation of the heart as it was in the living person. Accordingly, the exact location of the dead zone predicted by the presence of abnormal Q-waves recorded from a living person may not be exactly where the pathologist states it is. In addition, the determination of the degree of occlusion of a coronary artery by the pathologist may not be exactly the same as the degree of occlusion that is determined by coronary arteriography. One of the reasons for this is that coronary artery spasm is not evident at autopsy.

Gravanis states:

*Pathologists observe a change in the subendocardial area in the vicinity of an infarct, or around any ischemic injury, a change that is called myocytolysis. These changes are characterized by clear vacuolated cytoplasm caused by the loss of cytoplasmic contractile elements and the accumulation of neutral fat, which indicates a reduction in mitochondrial oxidation of fatty acids. It is not clear whether these changes may lead to cellular death although no polys are seen in the vicinity of these areas. Whether these abnormal myocytes, that reflect hypoperfusion, participate in any excitation-contraction mechanism or if they produce changes in the electrocardiogram is not known.

A pathologist cannot identify the electrically abnormal myocytes that produce the injury current that creates the S-T segment displacement in the electrocardiogram, or the electrically abnormal myocytes whose repolarization is delayed, thereby producing abnormal T-waves. Stated another way, the pathologist cannot examine the heart and predict the presence of S-T segment displacement due to injured myocytes, or predict the presence of a T-wave abnormality due to a delay in repolarization. This being true, it is not possible to correlate the S-T and T-wave abnormalities with findings at autopsy.

The S-T and T-wave abnormalities seen in the electrocardiogram of patients with myocardial infarction can

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be correlated with the finding at coronary arteriography, but this approach is far from perfect because the electrocardiographic abnormalities may be recorded at one point in time and the coronary arteriogram may not be done until later. Then too, spontaneous lysis of coronary thrombi probably occurs more frequently than we have previously believed. This makes the correlations of data difficult.

Finally, isoemboli are probably more common than is generally appreciated. Their presence or absence cannot be determined by coronary arteriography and the pathologist cannot accurately determine the presence of small emboli that are located in the subendocardial region of the ventricles.

These limitations are but of a few of the obstacles that prevent the gathering of accurate data that can be used to correlate the findings in the electrocardiogram with the findings obtained by autopsy and coronary arteriography. Therefore, as is often the case in our efforts to explain the electrocardiogram, many of the comments made in this essay are based on deductive reasoning along with the inadequate data derived from autopsies and coronary arteriograms.

Why is Thrombolytic Therapy Beneficial for Patients with a Myocardial Infarct who Show Epicardial Injury in the Electrocardiogram, but is Not Beneficial for Patients who do not Show Epicardial Injury?

When it is realized that an interpreter of electrocardiograms is trying to identify predominant epicardial injury and predominant subendocardial injury by studying the displacement of the S-T segment in order to determine how to treat the patient, the following question must be asked. Why should it matter—why is thrombolytic therapy beneficial to patients with S-T segment elevated myocardial infarction (STEMI) myocardial infarct but is not beneficial when there is S-T segment depression or only T-wave abnormalities?

A discussion of this question is aided by appreciating the anatomic differences between the endocardium, epicardium, and ventricular myocardium as well as certain physiologic differences between the left and right ventricles during mechanical systole and diastole.

Anatomic Considerations

The endocardium of both ventricles consists of a single layer of endothelial cells beneath which there is loose connective tissue and a few smooth muscle cells. In cases of ventricular dilatation these cells may proliferate and lay down collagen resulting in endocardial thickening. The myocardium beneath the endocardium is called the subendocardium. The subendocardium is divided into

two parts. The electrically silent area, which seems to be spared by infarction and is “invisible” to the electrocardiogram, and a slightly deeper area that makes up the subendocardium which, when severely damaged, creates abnormal Q-waves in the electrocardiogram.²⁶

The epicardium is produced by the visceral portion of the pericardium. It is easy to visualize this anatomic point by assuming that the pericardium is like a partially blown-up balloon into which the heart is thrust. Accordingly, the epicardium is in anatomic terms the visceral pericardium and the parietal pericardium surrounds the visceral pericardium and the space between the two normally contains about 15–50 mL of fluid.

The myocytes just beneath the epicardium respond to damage by the creation of S-T segment elevation in the electrocardiogram. This is known with certainty because an electrocardiogram recorded from the needle used to remove pericardial fluid will reveal instant S-T segment elevation the moment the needle touches the epicardium. Accordingly, it is not necessary to use the word subepicardium although the dead zone of infarction rarely reaches the epicardium. On the other hand, an electrocardiogram may reveal little change when a cardiac catheter touches the endocardium of the left ventricle.²⁷ This explains why we do not use the word subepicardium but do use the word subendocardium.

Physiological Considerations

Physiologically, it is important to remember that the perfusion of the left ventricular myocardium with blood occurs almost exclusively during mechanical diastole. This is because the systolic pressure within the left ventricle is equal to the systolic pressure in the aorta and coronary arteries and no pressure gradient is produced during systole. During diastole the pressure within the left ventricle is very low while the diastole pressure in the aorta and coronary artery is far greater—usually 60–80 mm. This produces a pressure gradient that permits the perfusion of blood in the left ventricular myocardium during diastole.

The arterial pressure in the aorta and coronary arteries during systole and diastole is normally greater than the systolic and diastolic pressure within the right ventricle. Accordingly, in the right ventricle the myocardial perfusion of blood takes place during systole and diastole. However, perfusion of the right ventricle will be diminished wherever there is severe systolic hypertension within the right ventricle.

With the foregoing as background information, one is compelled to ask the question—Why is the subendocardium of the left ventricle so susceptible to hypoxia? Stated another way the question becomes—why is the dead zone of the usual myocardial infarction larger in a segment of the subendocardium than it is in the epicardium. One must postulate that myocardial perfusion of

FIG. 1 This conceptual illustration is designed to show the electrocardiographic abnormalities produced by the usual myocardial infarction.

The mean spatial *Q-wave* vector is directed away from the *dead* portion of the myocardium. Note that the dead area does not extend to the endocardium or to the epicardium. The dead area is larger in the subendocardium than it is near the epicardium.

The mean spatial *S-T* vector is directed toward the area of predominant *epicardial injury*.

The mean spatial *T* vector is directed away from the area of predominant *epicardial ischemia*.

Each of the three vectors influences all 12 of the routine electrocardiographic leads, and any other lead that might be used, in a very predictable way. Note how the deflections would appear in leads I, *AV_F*, and *V₃*.

The diagram illustrates how S-T segment elevation is created.

blood is less in the subendocardial area than it is in the epicardium. The question then becomes—what causes the decrease in perfusion of the subendocardial region of the left ventricle during diastole? The answer to the question is not definitely known, but there are several possible causes that may act individually or in concert. They are:

- The vulnerability of the subendocardium of the left ventricle to hypoxia may be caused by the higher resistance to arterial blood flow in the smaller, longer, arterioles in that region of the heart.
- The “work” produced by the subendocardial myocytes of the left ventricle is greater than the myocytes located in the epicardium because of the unique anatomic configuration of the ventricles. This leads to an increase in ischemia of the subendocardial region compared to that in the epicardium.
- James, using an excellent injection technique to study human coronary arteries and their branches, provided a plausible answer to the question in his contribution to the first edition of “The Heart”, which was published in 1966. James wrote:²⁸

* Although there are anastomoses in the region of the endocardium of both ventricles, they are not as numerous or large as those in the epicardium. Epicardial anastomoses on the left ventricle connect all three major trunks. These epicardial anastomoses over the surfaces of both ventricles appear from an anatomic standpoint to be among the most important routes for collateral circulation . . .

Collateral coronary vessels are used mainly when the need arises. The need occurs when there is myocardial hypoxia due to occlusive disease of the epicardial coronary arteries. Accordingly, one can logically conclude that the subendocardium of the left ventricle is more likely to be deprived of collateral circulation than is the epicardium when there is occlusive epicardial coronary artery disease. This may explain why the dead zone is commonly larger in a segment of the subendocardial area than it is in the epicardial area in most patients with myocardial infarction.

* Personal communication with Dr. Thomas James on October 2, 2006 confirmed that his current view remains the same as stated in 1966.

FIG. 2 This conceptual illustration is designed to show the vector arrangement of electrocardiographic abnormalities produced by subendocardial injury and subendocardial infarction.

The mean spatial *S-T* vector is directed away from *subendocardial injury*. The area of injury may be segmental or more generalized. It may occur transiently due to spontaneous angina pectoris or during an exercise stress test, or it may be persistent, indicating subendocardial infarction. When it is localized it may morph into the abnormality shown in Figure 1. When there is a large, generalized subendocardial infarction it may create a critical state (see text).

The abnormally directed *S-T* vector influences all 12 of the routine electrocardiographic leads, and any other lead that may be used. Note how the *S-T* segment deflection would appear in leads I, *AV_F* and *V₅*.

This diagram illustrates how *S-T* segment depression is created.

- When there is triple vessel coronary artery obstruction, or when the smaller branches of the coronary arteries are filled with isoemboli from a proximal coronary artery thrombus, the electrocardiogram may show a persistent *S-T* segment displacement—the mean *S-T* vector points away from a rather large area of generalized subendocardial injury. Its *persistence*, compared to the *transient* finding in patients with a positive exercise stress test or spontaneous angina pectoris, indicates *generalized subendocardial infarction*. This serious condition is uncommon but is likely to occur when there is left ventricular dilation and hypertrophy and an elevated diastolic pressure in the left ventricle plus triple vessel coronary disease. In such cases a large subendocardial area of the left ventricle may become necrotic. One can justly label the infarct as *circumferential necrosis*.
- Thoughts about the hemodynamic control of myocardial perfusion help to partially understand the production of generalized subendocardial infarctions. Suppose the systolic pressure during myocardial

contraction in an epicardial coronary artery is 120 mmHg and the systolic pressure within the left ventricle is 120 mmHg. In such a case there would be little myocardial perfusion of blood during systole to the left ventricular myocardium. This normally causes no ischemia of the left ventricular myocardium. Now suppose there is a 60% diameter obstruction in the coronary artery. The diastolic pressure distal to an obstruction in a coronary artery during mechanical diastole may be 60 mmHg and the left ventricular diastolic pressure is 5 mmHg. The difference in perfusion pressure is 55 mmHg so perfusion of the myocardium is adequate. Suppose the diastolic pressure below the obstructing lesion is 30 mmHg and the pressure within the left ventricle is 5 mmHg. The myocardial perfusion pressure is 25 mmHg. Myocardial perfusion would be decreased in subendocardial areas. Now suppose there was a dilation of the left ventricle and eccentric hypertrophy of the left ventricle and a diastolic pressure below the coronary obstruction is 30 mmHg and the diastolic pressure within the left

ventricle of 20 mmHg. In such a case the perfusion pressure of the subendocardial of the left ventricle would be 10 mmHg. This could cause severe generalized subendocardial injury and infarction whenever this occurred in several branches of the coronary arteries simultaneously.

- Transient subendocardial ischemia is commonly observed in patients who have a positive exercise stress test and in patients with secondary unstable angina pectoris. The latter—secondary unstable angina pectoris may occur in patients with coronary disease who develop anemia, hypertension, tachy arrhythmia, thyrotoxicosis, or hypoxemia from lung disease, etc. Note that exercise, tachycardia, hypotension, and thyrotoxicosis place an increased *demand* on the myocardium while anemia and hypoxia prevent an *adequate amount* of oxygen from reaching the myocytes. In general, one can conclude that epicardial injury suggests that there has been a decrease in the *supply* of oxygen due to a change in the internal diameter of a coronary artery by thrombosis, spasm, or both.

Why is thrombolytic therapy of little value in patients with generalized subendocardial injury? One can speculate that the injured myocytes that are located in the large area of subendocardium that are more severely damaged than the myocytes that are located in an area of epicardial injury. This could be true because of the reasons just stated. Accordingly, the large and severely injured myocytes located in the subendocardial area tend to morph more toward the dead area than they do toward the area of repolarization delay.

Some Additional Questions

How does one square up the response discussed above with the very different electrocardiographic abnormalities seen in patients with an acute cerebral catastrophic or the tako tsubo syndrome? Subarachnoid hemorrhage, cerebral hemorrhage, and head trauma may stimulate an instantaneous outpouring of catecholamines that produce intense constriction of the epicardial coronary arteries. In addition the sympathetic nerves to the heart of such patients may damage the myocytes by delivering catecholamines directly to them. This causes severe generalized epicardial ischemia that is commonly accompanied by generalized predominant epicardial injury. The extremely large mean spatial T-vector is usually directed away from the centroid of the left ventricle and the mean S-T segment vector is directed toward the centroid of the left ventricle. Similar electrocardiographic abnormality may occur in grieving patients with the broken heart syndrome.¹² These abnormalities may be associated with pulmonary edema and global hypokinesia.

The question is—why do patients with a positive exercise stress test or spontaneous angina pectoris exhibit

FIG. 3 This conceptual illustration is designed to show the approximate direction of the mean spatial S-T segment vector that is produced by ten different myocardial infarcts. Note how the direction of the spatially oriented mean S-T segment vector identifies epicardial and subendocardial injury, and determines the leads in which the S-T segment is elevated or depressed.

The names of the infarcts are listed below. (i) septal, (ii) anteroseptal, (iii) anterolateral, (iv) posterolateral, (v) apical, (vi) inferior, (vii) infero-posterior, (viii) right ventricular, (ix) true posterior (the vector is directed straight back), and (x). subendocardial.

When the location of the ventricles and coronary arteries are visualized, an interpreter can identify the area of the heart muscle that is infarcted, the culprit artery causing it, and the location of the obstruction in the artery most of the time. This is not always possible because some areas of the heart are nourished by more than one coronary artery.

generalized subendocardial injury in the electrocardiogram whereas patients with an acute cerebral catastrophe or the broken heart syndrome develop severe generalized epicardial injury and ischemia? Perhaps the epicardial coronary arteries in patients with acute cerebral catastrophes and the tako tsubo syndrome have such intense constriction of several epicardial coronary arteries that it temporarily shuts off all of the coronary blood flow, including the anastomotic vessels themselves that lie on the epicardial surface of the heart.

Summary

Displacement of the S-T segment in the electrocardiogram has become one of medicines most important abnormalities because physicians use the abnormality to determine whether or not they use thrombolytic therapy in the treatment of acute myocardial infarction.

FIG. 4 This electrocardiogram was recorded from a 66-year-old woman who experienced the characteristic pain of an acute myocardial infarction and developed enzymatic evidence of considerable cardiac muscle destruction.

The mean spatial QRS vector is directed at -20° in the frontal plane and about 75° posteriorly. The initial 0.04 s vector is directed to the left and slightly anterior. The mean spatial T-vector is directed at about $+80^\circ$ in the frontal plane and about 60° anteriorly. The mean spatial S-T segment vector is directed at $+150^\circ$ in the frontal plane and about 75° posteriorly. This indicates infero-posterior predominant epicardial injury.

The direction of the mean spatial S-T segment vector is not directed as it usually is when it is caused by subendocardial injury. The mean spatial S-T segment vector due to subendocardial injury is usually directed between -90° and $\pm 180^\circ$ in the frontal plane and is usually about parallel with the frontal plane or directed slightly anterior. Here, in this tracing, it is directed $+150^\circ$ in the frontal plane and 75° posteriorly. An electrocardiogram recorded from the appropriate place on the back would show S-T segment elevation. The direction of the mean spatial S-T segment vector indicates epicardial injury in the infero-posterior portion of the left ventricle.

It is likely that some interpreters would classify the tracing as showing S-T segment depression and think no more. Interpreters who understand that they are searching for predominant epicardial injury and can plot the direction of the mean spatial S-T segment vector can arrive at the correct diagnosis.

Coronary arteriography revealed 100% occlusion of the mid right coronary artery, 90% occlusion of the proximal portion of the circumflex coronary artery, and 70% occlusion of the distal portion of the left anterior descending coronary artery.

The P-Q segment displacement suggests the likelihood of an atrial infarction. The patient had an extension of the infarction, developed A-V block and died. As reported by Jim *et al.* atrial infarction in the setting of inferior-posterior infarction predicts a high mortality from arrhythmias and rupture.²⁸

The mechanisms responsible for the abnormal Q-waves, S-T segment displacement, and T-waves produced by an acute myocardial infarction have been reviewed.

The commonly used description of STEMI and "non S-T elevated"(NSTEMI) acute myocardial infarctions without understanding the mechanism responsible for their development leads to errors.

Accordingly, the purpose of this communication is to discuss the mechanism for S-T segment displacement and to propose a more accurate way of describing the abnormalities (Figs. 1–3).

Wagner and his 12 nationally known co-authors have also addressed this problem. Although they express their views in a different manner, their discussion of the "pitfalls" in the current system of describing S-T segment displacement indicates their concern.²⁹

The interpreter is trying to identify the location of the injured myocytes, which are either located predominantly in the epicardium or subendocardium. The Grant

methods of identifying predominate epicardial injury and subendocardial injury is proposed as a more accurate and more scientific method of making the measurement than is the memorization of leads that show S-T segment displacement. His method of analysis is discussed briefly, but the fine details of Grant's method are found in his many publications.

An electrocardiogram is shown to illustrate how an error can be made (Fig. 4). The pattern reader might simply interpret the tracing as showing S-T segment depression and would not use thrombolytic therapy. On the other hand, an interpreter who uses vector concepts and basic principles would diagnose infero-posterior epicardial injury and would use thrombolytic therapy if there were no contraindication to its use.

Because we have barely scratched the surface of inquiry, some of the problems associated with the correlation of data are discussed.

The following question is discussed—why is thrombolytic therapy beneficial for patients with acute myocardial infarction who exhibit epicardial injury in the electrocardiogram, but is not beneficial for patients who exhibit generalized subendocardial injury in the electrocardiogram? Several mechanisms probably conspire to account for the difference in therapeutic benefit. First of all, there are fewer anastomotic vessels in the subendocardial area. Patients with generalized subendocardial injury usually have severe triple vessel disease, so the injury is severe. Emboli from a proximally located thrombus may obstruct the smaller penetration branches of the coronary arteries. The patient who has left ventricular dilation and hypertrophy, an elevated left ventricular diastolic pressure, and severe triple vessel coronary artery disease is particularly vulnerable to the development of a large generalized subendocardial infarction. *It seems that generalized subendocardial injury is so severe that thrombolytic therapy does not reverse the process.* All of the injured myocytes join the dead cells despite what is done. It is possible that, when the conditions are right that even smaller areas of the subendocardial injury might behave in a similar manner.

Finally, a few additional problems are discussed and tentative solutions are offered.

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