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Acute Myocardial Infarction and Left Bundle-Branch Block — Can We Lift the Veil?

When a patient presents to the emergency room with an acute episode of cardiac ischemia, the challenges for the physician are to determine whether the patient is having a myocardial infarction or has unstable angina and to determine the amount of myocardium at risk and the responsible coronary artery. That information will influence the decision to restore blood flow to the jeopardized area with thrombolytic agents or balloon angioplasty. In the acute phase of cardiac ischemia, the electrocardiogram is still the most important source of information to address these questions. Both the QRS complex and changes in the ST-T segment help locate the jeopardized area and indicate the severity of damage to that portion of the myocardium.

When considering the value and limitations of the electrocardiogram in making the diagnosis in patients with acute cardiac ischemia, two points are worthy of note. First, the spread of ventricular activation during sinus rhythm normally proceeds by way of an intact bundle-branch system; when that system is not intact, the diagnosis of acute ischemia may be more difficult. Second, locating ischemic or infarcted areas is easy when they are in parts of the ventricle that under normal circumstances are activated early in the QRS complex but is more difficult when the affected areas are in parts of the ventricle that are activated late in the QRS complex. Typically, ischemia or infarction of an area that is normally activated early results in an initially negative QRS complex and a Q wave in the electrocardiographic leads reflecting that area. Damage to myocardium in the anterior and inferior areas of the left ventricle is thus more easily identified than is damage in the posterobasal area.

When the sequence of ventricular activation is altered by bundle-branch block, ventricular pacing, or ventricular preexcitation, there will be a change in the timing of activation in the areas that are normally activated earliest. The diagnosis of ischemia or infarction is relatively easy in the presence of right bundle-branch block. The left ventricle accounts for the largest mass of myocardium, and sites of early activation of the left ventricle are not altered by right bundle-branch block. However, the situation is different with left bundle-branch block, which results in left ventricular activation by radial spread from the point of termination of the right bundle branch. Areas of the left ventricle that are normally activated early are activated much later in the QRS complex, and it is difficult to recognize ischemia or infarction in those areas.

This problem has puzzled physicians for more than 50 years.[1] One reason there is so much interest in the diagnosis of acute myocardial infarction in the presence of left bundle-branch block is that a patient with both these problems has a much worse prognosis than a patient with infarction and normal ventricular conduction.[2] As was shown 20 years ago,[3] this is true both for patients who already have left bundle-branch block before infarction (usually such patients have hypertensive heart disease) and for those in whom left bundle-branch block develops as a result of acute anteroseptal myocardial infarction. It is still the case in the modern era of thrombolytic therapy.[4]

Fifteen years ago, Wackers et al.[5] reviewed the various electrocardiographic criteria that were reported to be of value in making the diagnosis of acute myocardial infarction in the presence of complete left bundle-branch block. That was followed by a critical analysis of the criteria involving localization of infarcts with thallium-201 scintigraphy. Wackers et al. found that there was considerable interobserver variability in the interpretation of the electrocardiogram. They further concluded that the most valuable electrocardiographic criteria for the diagnosis of myocardial infarction were serial electrocardiographic changes (67 percent sensitivity), ST-segment elevation (54 percent sensitivity), abnormal Q waves (31 percent sensitivity), the sign of Cabrera (notching of 0.05 second in the ascending limb of the S wave in leads V_3 and V_4; 27 percent sensitivity), and initial positivity in lead V_1 and a Q wave in lead V_6 (20 percent sensitivity but 100 percent specificity for anteroseptal infarction). Finally, they found that a single electrocardiogram obtained during left bundle-branch block was of only limited value for the diagnosis of myocardial infarction.

Now, 15 years later, how has our understanding of this common clinical problem changed? In this issue of the *Journal*, Sgarbossa et al.[6] report on 131 patients with left bundle-branch block who were among the 26,003 patients enrolled in the GUSTO-1 (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) trial, in which two thrombolytic regimens were studied in patients with acute myocardial infarction. It is important to note that the electrocardiographic evidence of acute myocardial infarction in these patients was confirmed by enzyme measurements and therefore may not be representative of electrocardiographic findings in an earlier stage of severe cardiac ischemia. Three electrocardiographic criteria were found to have independent value in the diagnosis of acute infarction in the presence of left bundle-branch block: ST-segment elevation equal to or greater than 1 mm in the presence of a positive QRS complex; ST-segment depression equal to or greater than 1 mm in lead V_1, V_2, or V_3; and ST-segment elevation equal to or greater

than 5 mm in the presence of a negative QRS complex.

This is useful information, but several questions remain. First, is there a correlation between the particular electrocardiographic findings and the location and size of the ischemic or infarcted area? Second, what happens to the electrocardiographic changes when reperfusion occurs, as with thrombolytic therapy or angioplasty? Third, are there differences between the electrocardiographic changes in patients with severe ischemia but no infarction and the changes in patients with infarction? Serial electrocardiograms, enzyme measurements, documentation of the site and size of the infarcted area, and information on the patency of the culprit coronary artery are required to answer these questions.

It is understandable that these questions cannot be answered with the information available from the GUSTO-1 study. From a practical point of view, the evidence favors an aggressive approach to the treatment of the patient admitted with chest pain suggestive of acute cardiac ischemia and left bundle-branch block. Unless there are contraindications, thrombolytic therapy should be given. In the event that chest pain does not respond to thrombolytic therapy and the patient's hemodynamic status does not improve, an echocardiogram should be obtained to evaluate left ventricular function and wall motion as an aid in deciding about invasive procedures. Sgarbossa et al.[6] have attempted to lift the veil of left bundle-branch block in patients with acute myocardial infarction. Their work is a valuable contribution, but we still have more to learn about the value of the electrocardiogram in the diagnosis of acute myocardial infarction with left bundle-branch block.

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Electrocardiographic Diagnosis of Acute Myocardial Infarction in the Presence of Left Bundle-Branch Block
Ackermann R. J., Vogel R. L., Levenson J., Byrne J., Liron M., Sgarbossa E. B., Pinski S. L., Wagner G. S.
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N Engl J Med 1996; 335:131-133, Jul 11, 1996. **Correspondence**

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