ECG Discrimination Between Right and Left Circumflex Coronary Arterial Occlusion in Patients With Acute Inferior Myocardial Infarction*

Value of Old Criteria and Use of Lead aVR

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Study objectives: Prior studies have proposed several ECG criteria for identifying the culprit artery in patients with acute inferior myocardial infarction (MI). We applied each criterion to our patients to assess its utility. In doing so, we discovered a previously unreported, but highly useful, criterion utilizing lead aVR.

Study design: Retrospective review.

Patients: Thirty consecutive patients with symptoms of acute MI, ST-segment elevation in the inferior ECG leads, an appropriate rise and fall of creatine kinase and troponin I levels, and coronary arteriography within 7 days of the onset of symptoms.

Measurements: The ECG recorded within 24 h of the onset of symptoms that had the most prominent ST-segment changes was analyzed. In the 12 standard leads and in lead V4R, ST-segment elevation or depression was measured 0.06 s after the J point.

Results: Four previously described criteria were useful in identifying the right coronary artery (RCA) or the left circumflex coronary artery (LCX) as the culprit: ST-segment elevation in lead I, ST-segment more or less elevated in lead II than in lead III, ST-segment elevation ≥ 0.5 mm in lead V4R, and various combinations of ST-segment elevation or depression in leads V1 and V2. A new criterion was found to be at least as useful as any previously described: the presence and amount of ST-segment depression in lead aVR.

Conclusions: At least five different ST-segment criteria help to identify the RCA or the LCX as the culprit artery in patients with acute inferior MI. One of these, the amount of ST-segment depression in lead aVR, has not been reported previously and needs validation in a larger study.

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Key words: coronary angiography; coronary occlusion; coronary thrombosis; ECG; myocardial infarction, acute, inferior

Abbreviations: LAD = left anterior descending coronary artery; LCX = left circumflex coronary artery; MI = myocardial infarction; RCA = right coronary artery

With anterior myocardial infarction (MI), the occlusion is nearly always in the left anterior descending coronary artery (LAD). With inferior MI, however, either the right coronary artery (RCA) or the left circumflex coronary artery (LCX) may contain the culprit lesion, and mortality and morbidity in part are determined by the location of the occlusion.1,2 For example, in patients with inferior MI who have right ventricular infarction, the culprit artery virtually always is the RCA.3 Such patients, including those in whom ECG evidence of right ventricular MI is masked,4 are at increased risk for death,5,6 shock,4–6 and arrhythmias,5–7 including atrioventricular block. Thus, identifying the culprit artery in acute inferior MI helps define those in whom aggressive reperfusion strategies are likely to yield the most benefit.

Coronary arteriography is the best means of determining the culprit artery in acute inferior MI. When both the RCA and LCX are severely diseased, however, deciding which is the culprit can be difficult, and we have seen several examples of angioplasty being performed on a chronic lesion while the acute occlusion was ignored. In such circumstances,
having an independent predictor of the culprit artery, such as the ECG, can be very helpful. Several studies\textsuperscript{2,3,8–15} have proposed ECG criteria to aid in identifying the culprit artery in acute inferior MI. The purpose of this study has been to apply these various criteria to the ECGs of our patients with acute inferior MI to determine which of them have utility. In addition, we describe a previously unreported ECG criterion that we have found useful in identifying the artery containing the culprit lesion.

**Materials and Methods**

Thirty consecutive patients at the Medical Center of Louisiana who had symptoms compatible with acute MI, ST-segment elevation in the inferior ECG leads, an appropriate rise and fall of creatine kinase and troponin I levels, and coronary arteriography within 7 days of the onset of symptoms were included in the study. Exclusion criteria were bundle-branch block, prior Q-wave MI, prior coronary artery bypass graft operation, and inability to identify the culprit lesion (in two patients, one of whom had normal coronary arteriographic findings). A lesion was considered to be the culprit when it occluded or severely narrowed the artery and was ulcerated and/or contained thrombus. In each of the 30 patients included in the study, the authors and the arteriographers who performed the study agreed independently on the culprit artery and lesion within that artery. For each of the 30 patients, the ECG that was recorded within 24 h of the onset of symptoms and had the most prominent ST-segment changes was chosen for analysis. In each of the 12 standard leads, and in V\textsubscript{4}R when it was recorded, ST-segment elevation or depression was measured 0.06 s after the J point with the aid of a handheld magnifying lens. Measurements were made to the nearest 0.25 mm (0.025 millivolts). The TP segment of the ECG was used as the isoelectric line unless tachycardia caused fusion of the T and P waves, in which case the PR segment was used. Previously proposed criteria for identifying the culprit were then evaluated using our patients' data, and a new criterion, the amount of ST-segment depression in lead aVR, was tested.

**Results**

In 25 of 30 patients, the culprit lesion was in the RCA while in 5 patients it was the LCX (Table 1). In four of five patients with ST-segment depression $\geq 1.0$ mm in lead aVR, the culprit lesion was in the LCX. Conversely, the culprit lesion was in the RCA in 24 of the 25 patients with ST-segment elevation (2 RCA), an isoelectric ST segment (18 RCA), or ST-segment depression $< 1.0$ mm (4 RCA, 1 LCX) in lead aVR.

ST-segment elevation in lead III exceeded that in lead II in 26 patients, and 23 of them had an RCA culprit lesion. In three patients, the LCX contained the culprit. Of the three patients in whom ST-segment elevation in lead II exceeded that in lead III, the culprit lesion was in the LCX in two patients and in the RCA in one patient. The RCA contained the culprit lesion in the one patient in whom ST-segment elevation in lead II equaled that in lead III.

Right-sided precordial leads were obtained in 18 patients. In each of the 12 with $\geq 0.5$ mm ST-segment elevation in V\textsubscript{4}R, the RCA contained the culprit lesion. In two of the four patients with $< 0.5$ mm ST-segment elevation or an isoelectric ST-segment in V\textsubscript{4}R, the culprit lesion was in the RCA, and in two patients it was in the LCX. Each of the two patients with ST-segment depression in V\textsubscript{4}R had the culprit lesion in the LCX.

When the ST-segment was depressed both in lead V\textsubscript{1} and V\textsubscript{2}, the culprit lesion was in the LCX in five patients and in the RCA in three patients. With any other pattern in leads V\textsubscript{1} and V\textsubscript{2}, the culprit was in the RCA (Table 1). ST-segment elevation in lead I was found in only three patients, each of whom had the culprit lesion in the LCX. Conversely, of 27 patients with an isoelectric (3 RCA) or depressed (22 RCA, 2 LCX) ST-segment in lead I, 25 patients had the culprit lesion in the RCA.

ECG findings with features typical of RCA and LCX occlusions are illustrated in Figures 1, 2, respectively. The sensitivities, specificities, and predictive values of the various ST-segment changes for culprit lesions in the RCA and LCX are shown in Table 2.

**Discussion**

Among our 30 patients with acute inferior MI, the culprit lesion was in the RCA in 25 patients and in the LCX in 5 patients, a ratio of 5:1. Ten other studies of patients with acute inferior MI have found...
RCA to LCX ratios ranging from 2.2:1 to 7.0:1, and averaging 3.9:1.2,3,8–15 Thus, the RCA is much more likely than the LCX to contain the culprit lesion in patients with acute inferior MI. Rarely, acute inferior MI may result from occlusion of the recurrent LAD branch,2,3 which is the terminal portion of a “wrap-around” LAD, but this was not the case in any of our patients. Occasional studies also have included a few patients with normal coronary arteriographic findings2 and others in whom the culprit lesion could not be identified.3 We excluded two such patients from our study.

In addition to statistical probability, several ECG criteria help identify the RCA or the LCX as the artery containing the culprit lesion (Tables 1, 2). Each of these criteria is based on one of two anatomic facts. First, the myocardial distribution of the RCA is slightly rightward in the frontal plane, and consequently the current of injury resulting from its occlusion will be reflected more in lead III than lead II. Conversely the distribution of the LCX is slightly leftward in the frontal plane, and the current of injury from its closure will be seen more in lead II than lead III. Similarly the current of injury with RCA occlusion is more or less perpendicular to the axis of lead aVR, whereas the current of injury resulting from occlusion of the LCX has a mean vector that forms an obtuse angle with the axis of aVR. Therefore, significant ST-segment depression in aVR is more likely to occur with LCX occlusion. An injury vector leftward enough to cause ST-segment elevation in lead I is common with LCX occlusion, but rare with RCA occlusion.9

Second, the RCA provides almost all of the blood supply to the right ventricle, which is anterior as well as rightward. When the RCA is occluded proximal to one or more of its major right ventricular branches, ST-segment elevation is likely to be seen in lead V4R.16 Similarly the ST segment in lead V1 (V2R) may be elevated even when the more leftward precordial leads show ST-segment depression due to the posterior injury that so frequently accompanies acute inferior MI, and Mak and colleagues17 have drawn attention to this infrequent but highly specific finding for right ventricular infarction due to RCA occlusion. Evidence of acute right ventricular infarction is important, not only because it identifies the RCA as harboring the culprit lesion, but especially because it predicts a greatly increased morbidity and mortality.4–7 Consequently, right precordial leads, or at least a V4R lead, should be recorded in all patients with acute inferior MI. ST-segment depression in V1 and V2 indicates posterior injury and is typical of LCX occlusion. All 5 of our patients with LCX
occlusion had ST-segment depression in both V1 and V2, but only 3 of our 25 patients with RCA occlusion had this pattern. Any other ST-segment pattern in leads V1, V2 identified the RCA as containing the culprit lesion in each of 22 patients.

Unlike earlier studies,12,15 we did not find the ratio of ST-segment depression in lead V2 or V3 to ST-segment elevation in lead aVF or III to be helpful in identifying the artery with the culprit lesion. We did find, however, that quantifying ST-segment depression in lead aVR in our 30 patients distinguished a culprit LCX (≥ 1 mm) from a culprit RCA (< 1 mm or no depression) as well or better than other criteria. Because our study is small and because the amount of ST-segment depression in aVR has not previously been used to identify the culprit artery in patients with acute inferior MI, the true usefulness of these new criteria need to be evaluated in a larger study. One disadvantage of the aVR criteria is that ST-segment changes in that lead are rarely large, and distinguishing 1-mm depression from lesser amounts may be difficult.

Table 2—Utility of ECG Signs in Identifying the Culprit Artery*

<table>
<thead>
<tr>
<th>ST-Segment Finding</th>
<th>Sensitivity, %</th>
<th>Specificity, %</th>
<th>Positive Predictive Value, %</th>
<th>Negative Predictive Value, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>aVR ↑, ↔, ↓ &lt; 1 mm</td>
<td>96</td>
<td>80</td>
<td>96</td>
<td>80</td>
</tr>
<tr>
<td>I ↔, ↓</td>
<td>100</td>
<td>60</td>
<td>93</td>
<td>100</td>
</tr>
<tr>
<td>↑ III ≥ ↑ II</td>
<td>96</td>
<td>40</td>
<td>89</td>
<td>67</td>
</tr>
<tr>
<td>↑ V4R ≥ 0.5 mm</td>
<td>86</td>
<td>100</td>
<td>100</td>
<td>67</td>
</tr>
<tr>
<td>Not ↓ V1, not ↓ V2</td>
<td>88</td>
<td>100</td>
<td>100</td>
<td>63</td>
</tr>
<tr>
<td>LCX</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>↓ aVR ≥ 1 mm</td>
<td>80</td>
<td>96</td>
<td>80</td>
<td>96</td>
</tr>
<tr>
<td>↑ I</td>
<td>60</td>
<td>100</td>
<td>100</td>
<td>93</td>
</tr>
<tr>
<td>↑ III &gt; ↑ III</td>
<td>40</td>
<td>96</td>
<td>67</td>
<td>89</td>
</tr>
<tr>
<td>V4R ↓, ↔, ↑ &lt; 0.5 mm</td>
<td>100</td>
<td>86</td>
<td>67</td>
<td>100</td>
</tr>
<tr>
<td>↓ V1, ↓ V2</td>
<td>100</td>
<td>88</td>
<td>63</td>
<td>100</td>
</tr>
</tbody>
</table>

*↑ = elevated; ↓ = depressed; ↔ = isoelectric.
Lead aVR usually has been ignored in ECG diagnosis, but the Duke group and other eminent electrocardiographers have made a rational case for the increased use of the reciprocal of lead aVR (− aVR) in diagnosis.18 Goldberger19 long ago pointed out the utility of an initial r or R wave in lead aVR in distinguishing infarct-related from positional Q waves in the inferior leads, and Menown and Adgey20 found ST elevation in lead − aVR predictive of higher creatine kinase levels both in patients with inferior and in those with lateral acute MI. Wellens and Conover21 and Viik et al.22 have found that ST elevation in lead aVR or depression in − aVR when combined with ST-segment depression in other leads indicates more severe subendocardial ischemia. Engelen et al.23 reported ST-segment elevation in lead aVR when combined with ST-segment depression in other leads indicates more severe subendocardial ischemia.

Because the right arm is opposite the vector of the subepicardial ventricular current of injury in acute, diffuse pericarditis, ST-segment depression in lead aVR not only is the rule, but is one of the ECG criteria of acute, diffuse pericarditis. All of the ECGs used in this study were recorded within 24 h of the onset of symptoms, a time too early for the changes of infarct-related regional pericarditis.26 Because the ST-T changes in this entity usually mimic the ST-T changes seen with the acute infarct several days earlier, it is possible that such changes would be different in RCA and LCX occlusions, but we have no data to support or deny that hypothesis.

In conclusion, our study confirmed the utility of four previously described parameters for identifying the RCA or the LCX as containing the culprit lesion in patients with acute inferior MI. The calculated sensitivities, specificities, and predictive values were similar to those previously reported.8,9,11,13,14 In addition, we found a previously unreported parameter, the amount of ST-segment depression in lead aVR, also to be an accurate predictor, an observation that needs validation in a larger group of patients.

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