ECG Diagnosis of Acute Coronary Syndrome

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This article is part of a series on the electrocardiogram (ECG) diagnosis of acute coronary syndrome and myocardial infarction (MI) mimics. This article covers the typical ECG diagnosis and the less well-known ECG indications of myocardial ischemia, injury, and infarction. Infarction mimics involving Q waves were presented in Vol 18, No. 4 (Oct–Dec 2007); infarction mimics that involve the ST segment or T wave will be presented in a subsequent issue.

The Normal ECG

A detailed description of a normal ECG and of each of the 12 leads can be found in any book on electrocardiography. For the purposes of this issue, normal is defined as no significant Q waves, ST segment at the isoelectric line, and normal T waves in all 12 leads. Small Q waves (ie, <0.04 second wide and <25% the height of the QRS complex) can be normal in all leads except V1, V2, and V3; and lead AVR is often a QS complex normally. T waves can be slightly inverted in the right precordial leads (V1 and V2), and should be inverted in AVR in normal ECGs. Figure 1 shows which leads of the 12-lead ECG face the different surfaces of the heart and where those leads are located on a standard 12-lead ECG. Myocardial ischemia, injury, and infarction are diagnosed on the ECG by the presence of ECG changes in leads facing the involved area of the myocardium.

Acute Coronary Syndrome

The term acute coronary syndrome (ACS) refers to the pathophysiologic continuum that begins with plaque rupture in a coronary artery and ultimately results
in total occlusion of the artery by thrombus unless the process is arrested. Three distinct phases of this continuum are unstable angina (UA), non-ST elevation MI (NSTEMI), and ST elevation MI (STEMI).

Patients presenting with chest pain and ST elevation on the ECG are classified as STEMI. Patients presenting with chest pain, no ST elevation on the ECG, and normal cardiac biomarkers are classified as UA. Patients with elevated biomarkers are classified as NSTEMI. Patients in whom cardiac biomarkers are elevated, regardless of the presence or absence of ST elevation, are ultimately diagnosed with either Q-wave MI or non–Q-wave MI based on the presence or absence of Q waves on the ECG.

**ECG Signs of Myocardial Ischemia**

Ischemia occurs when there is a mismatch between myocardial O₂ supply and demand and can occur with either decreased O₂ supply or increased O₂ demands. Ischemia is a reversible process if blood flow is restored to the myocardium before permanent cell damage occurs. Patients presenting early in the ACS process often have ECGs that show changes in the ST segment or T wave that are consistent with myocardial ischemia, because ischemia precedes myocardial cell injury, and injury precedes myocardial cell death (necrosis or infarction).

The most familiar ECG patterns of ischemia are horizontal or downsloping ST segment depression of 1 mm or more and T-wave inversion. These signs of ischemia can be isolated to ECG leads overlying the involved myocardium and thus suggest localized ischemia, or they can be present in many ECG leads, suggesting more widespread ischemia. There is a correlation between the number of ECG leads that show ST deviation and the extent and severity of coronary artery disease. If ST segment depression occurs in 8 or more leads along with ST elevation in AVR and V1, there is a high risk of either left main coronary artery disease or severe triple vessel disease. Figure 2 is a 12-lead ECG of a patient who presented with chest pain unrelieved by his usual dose of nitroglycerin in the emergency department (ED). His cardiac biomarkers (creatine kinase-MB [CK-MB] and troponin I) were normal, but his ECG showed ST depression in leads I, II, III, AVF, and V₃–V₆, and ST elevation in AVR and V₁. This pattern placed him at high risk for either left main or triple vessel disease and indicates the need for antiplatelet and anti-ischemic therapy and a trip to the catheter laboratory to evaluate coronary anatomy and possible intervention. Because his ECG does not show ST elevation and his biomarkers are normal, his diagnosis is UA.

Figure 3 shows widespread T-wave inversion, which is another familiar sign of ischemia. There is T-wave inversion in the inferior leads (II, III, AVF) and in all of the precordial leads (V₁–V₆). If the cardiac biomarkers are normal, the diagnosis is UA; if they are elevated, the diagnosis is NSTEMI.

**More Subtle Signs of Ischemia**

In addition to the well-known ST depression and T-wave inversion, other ECG changes can also indicate coronary insufficiency and be an early indicator of ischemia. These include a horizontal ST segment that forms a sharp angle with the ascending limb of the T wave and U wave inversion following an upright T wave. Table 1 illustrates several patterns of myocardial ischemia. ST segment and T wave changes are often nonspecific and can occur in a variety of conditions, but they should be considered as possible indicators of ischemia in patients presenting with chest pain. Figure 4 shows an ECG with U wave inversion in V₄–V₆ in a patient presenting with chest pain but no other obvious ECG changes.
### Table 1: Patterns of Ischemia

<table>
<thead>
<tr>
<th>ST segment depression</th>
<th>Horizontal ST depression</th>
<th>Downsloping ST depression</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><img src="example.png" alt="Heart ECG" /></td>
<td><img src="example.png" alt="Heart ECG" /></td>
</tr>
</tbody>
</table>

| T-wave inversion      | ![Heart ECG](example.png) | ![Heart ECG](example.png) |
|                       | ![Heart ECG](example.png) | ![Heart ECG](example.png) |

<table>
<thead>
<tr>
<th>Horizontal ST with ST–T angulation</th>
<th><img src="example.png" alt="Heart ECG" /></th>
<th><img src="example.png" alt="Heart ECG" /></th>
</tr>
</thead>
</table>

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<tr>
<th>Tall, wide based T waves</th>
<th><img src="example.png" alt="Heart ECG" /></th>
<th><img src="example.png" alt="Heart ECG" /></th>
</tr>
</thead>
</table>

| U-wave inversion                   | ![Heart ECG](example.png) | ![Heart ECG](example.png) |
ECG Signs of Myocardial Infarction

ECG changes of infarction include ST elevation (indicating injury), Q waves (indicating necrosis), and T-wave inversion (indicating ischemia and evolution of the infarction). These changes are called the indicative changes of infarction and occur in leads facing the damaged tissue. Reciprocal changes are the mirror image of the indicative changes and are often seen in leads recording from the opposite area of the heart. Reciprocal changes include taller-than-normal R waves (mirror image of Q waves), ST depression (mirror image of ST elevation), and tall T waves (mirror image of T-wave inversion). Table 2 shows the leads in which indicative and reciprocal changes are recorded with different types of MI.

The earliest ECG changes that occur with acute coronary artery occlusion are tall, peaked, and often wide-based T waves.6,8

Table 2: Leads Showing ECG Changes of MI

<table>
<thead>
<tr>
<th>MI Location</th>
<th>Indicative Changes</th>
<th>Reciprocal Changes*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inferior</td>
<td>II, III, aVF</td>
<td>I, aVL, V1 to V6, sometimes V4–V6</td>
</tr>
<tr>
<td>Anterior</td>
<td>V3, V4</td>
<td>II, III aVF</td>
</tr>
<tr>
<td>Septal</td>
<td>V1, V2</td>
<td>II, III, aVF, possibly I, aVL</td>
</tr>
<tr>
<td>Lateral</td>
<td>I, aVL, V5, V6</td>
<td>II, III, aVF</td>
</tr>
<tr>
<td>Anterolateral</td>
<td>I, aVL, V1–V6</td>
<td>II, III, aVF</td>
</tr>
<tr>
<td>Anteroskeletal</td>
<td>V1–V4</td>
<td>II, III, aVF</td>
</tr>
<tr>
<td>Posterior</td>
<td>None on standard 12 lead ECG. Posterior leads V7, V8, V9</td>
<td>V1, V2; sometimes V1–V4</td>
</tr>
<tr>
<td>Right ventricle</td>
<td>Occurs with inferior MI, so II, III, aVF and V1. Right side leads V4R–V6R</td>
<td>V2–V6</td>
</tr>
<tr>
<td>Non-Q-wave MI</td>
<td>Reduced R-wave height, ST depression, T-wave inversion in leads over damaged area</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: ECG, electrocardiogram; MI, myocardial infarction.

*Reciprocal changes are not always seen, but when they occur they are seen in these leads.
This sign is rarely seen in the clinical setting because patients are usually not having a 12 lead ECG recorded at the moment their artery occludes. The first ECG change seen clinically is usually ST segment elevation, which indicates myocardial injury in tissue underlying the electrodes. The American College of Cardiology/American Heart Association Task Force on Practice Guidelines for management of STEMI define significant ST elevation as 1 mm or more in 2 contiguous leads (eg, 2 neighboring leads facing the same area of the heart). Figure 5 is a 12-lead ECG of STEMI in the inferior wall (ST elevation is present in leads II, III, and AVF); reciprocal ST depression is present in most other leads. The presence of large R waves and ST depression in V1–V3 indicates that the posterior wall is also involved in the infarct. Figure 6 is a 12-lead ECG of STEMI in the anterior wall (ST elevation is present in leads V1–V3); there are no reciprocal changes in other leads.

Q waves are the indicative change of infarction (necrosis). Many leads can record normal Q waves, which are less than 0.03 seconds in width and usually small (no more than 25% the height of the following R wave). Significant Q waves are more than 0.03 seconds wide and often 25% or more the height of the following R wave. Other conditions can also cause abnormal Q waves on the ECG, but in a patient with chest pain and ST elevation, Q waves are considered diagnostic of infarction. Q waves usually appear within 8 to 12 hours of ST elevation if the artery is not reperfused; however, some patients do not develop Q
waves until days after the MI. If an artery is reperfused early, Q waves may disappear in subsequent ECGs. In Figure 5, there are significantly wide Q waves in leads III and AVF, indicating inferior wall infarction.

**Other ECG Signs of Injury**

In addition to ST elevation, other ECG changes can indicate acute injury, including an ST segment that pulls up to the peak of the T wave with no obvious J point visible; tall, peaked T waves; and symmetrical T-wave inversion. Table 3 shows acute injury patterns. Some ECG changes can indicate either ischemia or injury (eg, T-wave inversion), and most of the ECG changes that occur in ischemia and injury can also be seen in other conditions (eg, electrolyte imbalances, other cardiac disease processes). The ECG is not specific in diagnosing anything other than the rhythm, but it remains a useful diagnostic tool, especially in the patient presenting with ACS.

**Case Study**

A 61-year-old woman with mild epigastric pain and nonradiating chest pressure, which she described as being 2 on a 10-point scale (0, no pain; 10, worst possible pain), presented to the emergency department (ED). She had a history of hypertension and high cholesterol and was a smoker. Her medications included atenolol (Tenormin), omeprazole (Prilosec), premarin, and atorvastatin (Lipitor). Her blood pressure was 134/68 mm Hg, heart rate 56 beats per minute, and respiratory rate 16/min. Figure 7 is her admission ECG. Cardiac biomarkers (CK-MB and troponin I) were both normal, and a sestamibi scan was negative.

Do you see anything suspicious for myocardial ischemia, injury, or infarction on her ECG?

She was sent home, where she continued to have pain for the next few hours. She returned to the ED when her pain worsened. On readmission, her pain was 8 on a scale of 1–10 and radiated to her jaw and left shoulder. She was
nauseated and lightheaded, and her blood pressure was 98/60 mm Hg. Figure 8 is her ECG on readmission to the ED. Her CK-MB was 6 U/L (normal 0–5 U/L), and her troponin I was 1 ng/ml (normal < 0.7 ng/ml).

Do you see anything suspicious for myocardial ischemia, injury, or infarction on her ECG now? Which artery is the culprit? What is her rhythm?

Her cardiologist was notified and she was sent immediately to the cardiac catheter laboratory, where she was found to have a totally occluded right coronary artery (RCA). She was started on heparin and Integrilin and received two stents to her RCA. Four hours later, her CK-MB was 661 U/L and her troponin I was 13.2 ng/ml. Figure 9 is her ECG taken the morning after her stent insertion.
What signs of infarction are present on her postprocedure ECG?
Her postprocedure recovery was uneventful and she was discharged 2 days later with clopidogrel (Plavix), atorvastatin, metoprolol, and premarin prescribed.

Discussion
The admission ECG (Figure 7) was interpreted as normal. There are no abnormal Q waves of infarction and there is no ST segment elevation indicating injury. There is a horizontal ST segment with a fairly sharp angle between the ST segment and the ascending limb of the T wave in leads I, II, and V3–V5. There is suspicion of an inverted U wave in V5 and V6.

On return to the ED, her ECG (Figure 8) showed ST elevation in leads II, III, and AVF with reciprocal ST depression in all other leads. Based on the presence of ST elevation, her diagnosis was STEMI of the inferior wall. She had not yet developed Q waves in the inferior leads. Her rhythm was type I second-degree AV block (Wenckebach), which is not unusual with inferior infarction because the RCA that supplies the inferior wall also supplies the AV node in most people.

The postprocedure ECG shows resolution of the ST segment elevation, Q waves, and T-wave inversions in leads II, III, and AVF, all signs of an evolving MI. The Q waves indicate that some myocardial necrosis did occur although the artery was opened with stents. The ST-T angle is more gradual and normal looking, and the hint of U-wave inversion is gone. There is a large upright T wave in V2 with a fairly large R wave, which could indicate involvement of the posterior wall in the infarction.

This is a good example of the value of a chest pain monitoring unit. If the patient had been observed for several hours instead of being sent home, changes of ischemia might have been detected before injury could occur and treatment might have been initiated before myocardial damage could result.

References

In Memoriam
The nursing and medical professions have lost one of our greatest educators. Dr Henry J. L. Marriott died on August 31, 2007, at the age of 90. Barney, as he was known by friends and colleagues, spent a lifetime teaching electrocardiography to nurses and physicians. His book Practical Electrocardiography, now in its 10th edition, has been one of the best resources for 12-lead electrocardiogram (ECG) and cardiac arrhythmia interpretation for decades. He had a way of presenting complex information in a humorous manner that was easy to understand, and his passion for teaching made learning from him an enjoyable experience. He had an immense respect for nurses and believed that the bedside nurse needed to know at least as much about arrhythmias and ECG interpretation as physicians in order to provide the best possible patient care. Barney, you will be greatly missed.