The electrocardiogram remains a crucial tool in the identification and management of acute myocardial infarction. A detailed analysis of patterns of ST-segment elevation may influence decisions regarding the use of reperfusion therapy. The early and accurate identification of the infarct-related artery on the electrocardiogram can help predict the amount of myocardium at risk and guide decisions regarding the urgency of revascularization. Electrocardiographic signs of reperfusion represent an important marker of microvascular blood flow and consequent prognosis. The electrocardiogram is also crucial for identifying new conduction abnormalities and arrhythmias that influence both short- and long-term outcome. In this review, we discuss approaches to the interpretation of the electrocardiogram in the clinical management of patients during the first 24 hours after a myocardial infarction.

The specificity of the electrocardiogram in acute myocardial infarction is limited by large individual variations in coronary anatomy as well as by the presence of preexisting coronary artery disease, particularly in patients with a previous myocardial infarction, collateral circulation, or previous coronary-artery bypass surgery. The electrocardiogram is also limited by its inadequate representation of the posterior, lateral, and apical walls of the left ventricle. Despite these limitations, the electrocardiogram can help in identifying proximal occlusion of the coronary arteries, which results in the most extensive and most severe myocardial infarctions.

Identification of the Infarct-related Artery

The electrocardiogram may help identify the culprit vessel. For example, in inferior myocardial infarction, the electrocardiogram may reveal greater ST-segment elevation in lead III than in lead II and ST-segment depression of more than 1 mm in leads I and aVL, suggesting involvement of the right coronary artery. Conversely, ST-segment depression in leads V1 and V2 with ST-segment elevation in the inferior leads also suggests involvement of the left circumflex vessel, but this pattern may also be seen in infarction caused by occlusion of a dominant right coronary artery. In either circumstance, ST-segment depression in leads V1 and V2 suggests con-
ST-segment elevation in III > ST elevation in II
and
ST-segment depression in I, aVL, or both (>1 mm)

Yes

Right coronary artery
Sensitivity 90%
Specificity 71%
Positive predictive value 94%
Negative predictive value 70%

No

ST-segment elevation in I, aVL, V₅, and V₆
and
ST-segment depression in V₁, V₂, and V₃

Left circumflex coronary artery
Sensitivity 83%
Specificity 96%
Positive predictive value 91%
Negative predictive value 93%

In addition, ST-segment elevation in V₁, V₃, or both

Proximal right coronary artery
Sensitivity 79%
Specificity 100%
Positive predictive value 100%
Negative predictive value 88%

ST-segment elevation in I, aVL, V₅, and V₆
and
ST-segment depression in V₁, V₂, and V₃

In addition, ST-segment elevation in V₁, V₃, or both

Proximal right coronary artery
Sensitivity 79%
Specificity 100%
Positive predictive value 100%
Negative predictive value 88%

The management of acute myocardial infarction is targeted toward restoration of blood flow in the infarct-related artery. There is increasing evidence that the presence of normal epicardial blood flow does not always correlate with microvascular perfusion.

By Matthew G. Williams, Ann C. Kehoe, and Joseph P. Ornato
of the myocardial tissue. The absence of tissue perfusion is the most potent predictor of impaired ventricular function and the risk of death after myocardial infarction. Conversely, resolution of ST-segment elevation is believed to be an excellent marker of tissue perfusion, and the degree of resolution has proved to be a powerful indicator of short-term (30-day) and long-term (1-year) prognosis. Assessment of ST-segment resolution is also useful for guiding reperfusion therapy: the absence of ST-segment resolution during the first 90 minutes after the administration of fibrinolytic medications should prompt consideration of rescue angioplasty. A reduction in ST-segment elevation by more than 70 percent in the leads with maximal elevation is associated with the most favorable outcomes. In the future, therapies that promote microvascular blood flow after restoration of blood flow in the infarct-related artery may become available. The simplicity of assessing ST-segment resolution will probably make this step an important component of the decision to administer such therapies.

Other electrocardiographic markers of reperfusion include T-wave inversion within four hours after myocardial infarction. T-wave inversion that occurs during the first few hours of reperfusion therapy is a highly specific sign of reperfusion. T-wave inversion that develops more than four hours after the start of reperfusion therapy is consistent with the normal electrocardiographic evolution of myocardial infarction and does not indicate that reperfusion has occurred. An accelerated idioventricular rhythm (defined as a heart rate of 60 to 120 beats per minute initiated by a late, coupled, ventricular premature depolarization) is a highly specific marker of reperfusion. This rhythm is benign and should not be suppressed with medication. Isolated ventricular premature depolarizations may also be seen with reperfusion. Polymorphic ventricular tachycardia and ventricular fibrillation may be seen with reperfusion but are rare and should raise the suspicion of ongoing arterial occlusion.

Conduction abnormalities, including bundle-branch block or varying forms of heart block during acute myocardial infarction, may be associated with a poor prognosis. The incidence of conduction abnormalities associated with acute myocardial infarction has diminished in the era of early revascularization therapy, but the mortality and morbidity associated with these abnormalities remain unchanged. The presence and clinical significance of different types of bradyarrhythmias and conduction disease depend on the location of the infarct and the mass of the involved myocardium.

An understanding of the bradyarrhythmias and conduction abnormalities is crucial in the management of acute myocardial infarction.
conduction disease that may be associated with acute myocardial infarction requires a review of the anatomy and blood supply of the conduction system. The sinus node is supplied by the right coronary artery in 60 percent of people and by the left circumflex artery in 40 percent. The atrioventricular node is supplied by the right coronary artery in 90 percent of people and by the left circumflex artery in 10 percent. The bundle of His is supplied by the atrioventricular nodal branch of the right coronary artery, with a small contribution from the septal perforators of the left anterior descending artery. The bundle of His divides into the right and left bundle branches in the interventricular septum. The right bundle branch receives most of its blood from septal perforators of the left anterior descending artery. There may also be collateral blood supply from the right coronary artery or left circumflex artery. The proximal left bundle branch divides into the left anterior fascicle and the left posterior fascicle. The left anterior fascicle is supplied by septal perforators of the left anterior descending artery and is particularly susceptible to ischemia or infarction. The proximal portion of the left posterior fascicle is supplied by the atrioventricular nodal artery (i.e., the right coronary artery) and by septal perforators of the left anterior descending artery. The distal portion of the posterior fascicle has a dual blood supply from the anterior and posterior septal perforating arteries.

**Inferior Myocardial Infarction**

Conduction abnormalities in association with inferior myocardial infarction can occur immediately or hours or days after infarction. Sinus bradycardia or varying degrees of atrioventricular block (including complete heart block) can occur within the first two hours after an acute inferior myocardial infarction as a result of heightened vagal tone. Such conditions often resolve within 24 hours, and they are very responsive to atropine. Later in the course of inferior myocardial infarction, progressive conduction delay and block may occur. Their spontaneous resolution is regressive, as third-degree atrioventricular block becomes second-degree and then first-degree block and finally resolves, with normal conduction ensuing. This phase of atrioventricular conduction problems appears to be related to edema and local accumulation of adenosine. It is less responsive to atropine than the acute phase and may respond to aminophylline. The atrioventricular node is the site of conduc-

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**Figure 3. Electrocardiogram Showing Inferior Myocardial Infarction Associated with Complete Heart Block with a Narrow Escape Rhythm.**

There is ST-segment elevation in lead III that is greater than the ST-segment elevation in lead II, marked ST-segment depression in leads I and aVL, and ST-segment elevation in lead V₁ — all consistent with the occurrence of proximal occlusion of the right coronary artery in association with right ventricular infarction. If the patient is in hemodynamically stable condition, a temporary pacemaker is not required.
tion disturbances in inferior myocardial infarction; therefore, complete atrioventricular block is generally associated with a narrow complex escape rhythm of between 40 and 60 beats per minute (Fig. 3). It is usually asymptomatic but may be associated with hemodynamic instability due to loss of atrioventricular synchrony. It is generally transient and resolves within five to seven days but may persist for up to two weeks. A ventricular escape rhythm with a widened QRS complex may signify the presence of block below the atrioventricular node and impaired collateral circulation to an occluded left anterior descending artery.

The management of conduction abnormalities associated with myocardial infarction depends on the associated symptoms. As noted above, bradyarrhythmias during the first few hours after an acute inferior myocardial infarction are responsive to atropine; conduction disease that begins or persists after the first 24 hours of myocardial infarction is not responsive to atropine. If the patient has hemodynamic instability, worsening ischemia, or ventricular arrhythmias, a temporary pacemaker should be used. Placement of temporary pacing wires in the right atrium (or coronary sinus) and right ventricle allows restoration of atrioventricular synchrony and improves hemodynamic function. In most instances, conduction abnormalities associated with acute inferior myocardial infarction resolve within two weeks, and permanent pacing is not required (Table 1).

**Anterior Myocardial Infarction**

As opposed to inferior myocardial infarction, conduction disease associated with anterior myocardial infarction is not related to heightened vagal tone but instead to necrosis of the intramyocardial conduction system. This condition occurs almost exclusively in the presence of proximal occlusion of the left anterior descending artery and septal necrosis. PR prolongation in acute anterior myocardial infarction is rarely due to atrioventricular nodal ischemia, since in most people the atrioventricular node is

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**Table 1. Guidelines of the American College of Cardiology and the American Heart Association for Temporary or Permanent Implantation of Pacemakers in Patients with Acute Myocardial Infarction.**

<table>
<thead>
<tr>
<th>Class†</th>
<th>Indications for Temporary Pacing</th>
<th>Indications for Permanent Pacing</th>
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<tbody>
<tr>
<td>I</td>
<td>Asystole&lt;br&gt;Symptomatic bradycardia (including sinus bradycardia or Mobitz type I block with hypotension) &lt;br&gt;Bilateral BBB (alternating BBB or right BBB alternating with LAFB or LPFB) &lt;br&gt;Bifascicular block that is new or of indeterminate age (right BBB with LAFB or LPFB or left BBB) with a prolonged PR interval &lt;br&gt;Mobitz type II second-degree AV block</td>
<td>Persistent second-degree AV block in the His–Purkinje system, with bilateral BBB or third-degree AV block within or below the His–Purkinje system after myocardial infarction &lt;br&gt;Transient advanced (second- or third-degree) infranodal AV block and associated BBB† &lt;br&gt;Persistent and symptomatic second- or third-degree AV block</td>
</tr>
<tr>
<td>IIa</td>
<td>Right BBB and LAFB or LPFB that is new or of indeterminate age &lt;br&gt;Right BBB with a prolonged PR interval &lt;br&gt;Left BBB that is new or of indeterminate age &lt;br&gt;Recurring sinus pauses not responsive to atropine</td>
<td>None</td>
</tr>
<tr>
<td>IIb</td>
<td>Bifascicular block of indeterminate age &lt;br&gt;Isolated right BBB that is new or of indeterminate age</td>
<td>Persistent second- or third-degree AV block at the level of the AV node</td>
</tr>
<tr>
<td>III</td>
<td>Prolonged PR interval &lt;br&gt;Type 1 second-degree AV block with normal hemodynamics &lt;br&gt;Accelerated idioventricular rhythm &lt;br&gt;BBB or fascicular block known to exist before acute myocardial infarction</td>
<td>Transient AV conduction disturbances in the absence of intraventricular conduction defects &lt;br&gt;Transient, isolated AV block in the presence of isolated LAFB &lt;br&gt;Acquired LAFB in the absence of AV block &lt;br&gt;Persistent first-degree AV block in the presence of BBB that is old or of indeterminate age</td>
</tr>
</tbody>
</table>

*The information is adapted from Ryan et al. and Gregoratos et al. AV denotes atrioventricular, BBB bundle-branch block, LAFB left anterior fascicular block, and LPFB left posterior fascicular block.
†Class designations refer to the level of evidence supporting the effectiveness of the procedure or treatment, where class I indicates that the evidence is very strong and class III that it is absent or that the procedure is not useful and may be harmful.
‡An electrophysiological study may be useful to determine the site of the block.
supplied by the right coronary artery. More commonly, necrosis of the septum is associated with slight PR prolongation (usually <0.25 second) due to involvement of the conducting system below the atrioventricular node. In this situation, PR prolongation is often associated with a wide QRS complex (>0.12 second) with a right bundle-branch block pattern.

Second-degree atrioventricular block with anterior myocardial infarction is usually Mobitz type II block secondary to block in the His–Purkinje system. Complete heart block results from extensive necrosis of the ventricular septum. It usually occurs abruptly during the first 24 hours after myocardial infarction and is almost always preceded by the development of right bundle-branch block with right- or left-axis deviation (Fig. 4). In right bundle-branch block associated with anterior myocardial infarction, a Q wave precedes the R wave in lead V1 (QR in lead V1).

Both the left anterior fascicle and the right bundle branch are supplied by the septal branches of the proximal left anterior descending artery. Anteroseptal infarctions may be associated with the development of new right bundle-branch block, with left anterior (or, less commonly, left posterior) fascicular block. The development of bifascicular block with anteroseptal infarction is associated with an as much as a 30 percent excess risk of complete heart block. The addition of PR prolongation to bifascicular block further increases this risk of complete heart block. The mortality associated with complete heart block in anterior myocardial infarction, with or without preceding right bundle-branch block and left fascicular block, may be as high as 80 percent. This mortality rate is largely related to progressive pump failure as a result of extensive myocardial necrosis. A temporary pacemaker should be placed in patients with anterior infarction and new right bundle-branch block (QR in lead V1) with left anterior or left posterior fascicular block if there is associated PR prolongation (Table 1). Another indication for temporary pacing during anterior myocardial infarction is alternating right and left bundle-branch block.

Tachyarrhythmias

Tachyarrhythmias that occur during acute myocardial infarction may result from reperfusion, altered autonomic tone, or hemodynamic instability. Sinus tachycardia generally results from heightened adrenergic tone and is usually a manifestation of he-
modynamic failure. Atrial fibrillation may also occur in acute infarction and may result from increased vagal tone, increased left atrial pressure, atrial infarction, or pericarditis. Atrial fibrillation is associated with a worsened prognosis, regardless of the site of infarction.\textsuperscript{28} Ventricular premature depolarizations are common during acute myocardial infarction but do not predict the subsequent development of sustained ventricular arrhythmias and should not be suppressed.\textsuperscript{29} Sustained monomorphic ventricular tachycardia (with a heart rate above 150 beats per minute) is not generally associated with acute myocardial infarction unless there is a preexisting scar in the region that has become ischemic or a very large myocardial infarction.\textsuperscript{30}

Initially, ventricular fibrillation may be seen as an acute manifestation of ischemia; later (at two or three weeks), it may be seen as a consequence of progressive pump dysfunction. The presence of anterior myocardial infarction with right bundle-branch block and an ejection fraction of 35 percent or less is associated with recurrent ventricular tachycardia or fibrillation in the second and third weeks after myocardial infarction. In most instances, ventricular fibrillation in acute myocardial infarction is associated with lack of reperfusion of the infarct-related artery and should prompt evaluation for cardiac catheterization.\textsuperscript{31}

**CONCLUSIONS**

Important information to guide management and determine prognosis can be derived from the electrocardiogram in patients with acute myocardial infarction. Electrocardiographic markers of proximal coronary-artery occlusion identify relatively large myocardial infarctions that benefit most from early and complete revascularization strategies, such as primary angioplasty. The degree of ST-segment resolution is a simple and powerful predictor of ventricular function and prognosis after myocardial infarction. Finally, the recognition of abnormalities of conduction that result from different types of myocardial infarction is essential to the proper management of these conditions.

We are indebted to William C. Quist, M.D., for his review of the anatomy of the cardiac conduction system.

**REFERENCES**


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