An important factor to consider when using findings on electrocardiograms for clinical decision making is that the waveforms are influenced by normal physiological and technical factors as well as by pathophysiological factors. Traditionally, the focus of bedside monitoring is detection of arrhythmia. However, continuous ST-segment monitoring for the detection of myocardial ischemia is now readily available. Many factors affect electrocardiographic waveforms and may interfere with diagnosis of myocardial ischemia based on electrocardiographic findings. Accordingly, a principal leadership role for clinical nurse specialists and nurse practitioners is to become knowledgeable about interpretation of 12-lead electrocardiograms and to share this knowledge with staff nurses who care for patients with acute coronary syndromes. The factors that alter electrocardiographic findings are reviewed, and the alterations that interfere with electrocardiogram-based diagnosis of myocardial ischemia are discussed. (American Journal of Critical Care. 2003;12:9-18)

FACTORS TO CONSIDER WHEN ANALYZING 12-LEAD ELECTROCARDIOGRAMS FOR EVIDENCE OF ACUTE MYOCARDIAL ISCHEMIA

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Since the advent of bedside electrocardiographic (ECG) monitoring in the early 1960s, nurses in intensive care units, emergency departments, operating rooms, and telemetry units have been responsible for interpreting ECGs. In the 1970s and 1980s, the goals of ECG monitoring and interpretation were to detect alterations in heart rate and rhythm. Disorders of heart rate and rhythm alter the time intervals on the horizontal axis of ECG tracings and usually can be diagnosed by analyzing the findings on a single lead from a bedside monitor. Moreover, because determinations of changes in amplitude, which are measured on the vertical axis on ECG tracings, are not required for such diagnoses, standard calibration is unnecessary. For these reasons, learning to interpret standard 12-lead ECGs has not been a high priority or expectation of critical care nurses.

In the 1990s, multiple factors led to a shift in emphasis from solely monitoring cardiac rate and rhythm to monitoring to detect both arrhythmia and
ischemia. First, new antithrombotic and antiplatelet agents were introduced that abolish acute myocardial ischemia in patients with acute coronary syndromes (unstable angina and acute myocardial infarction). Research with continuous ST-segment monitoring indicated that most ischemic events are asymptomatic. Therefore, monitoring ECGs for evidence of ischemia is more sensitive than using patients’ signs and symptoms to assess the effectiveness of these new agents. Second, new percutaneous catheter interventions (eg, stenting) have become the standard of care for early reperfusion therapy in hospitals with cardiac catheterization laboratories. As a result, ECG monitoring for ischemia has been valuable in diagnosing the complication of abrupt coronary artery reocclusion after percutaneous catheter interventions. Third, multiple studies have indicated that patients who have ischemic events in the hospital have worse short- and long-term outcomes than do patients who do not experience ischemia. Therefore, ECG monitoring for myocardial ischemia is useful in detecting a high-risk group of patients who may warrant more aggressive management and follow-up.

Because ischemia can occur in any of many discrete zones of the myocardium, multiple ECG leads (ideally, 12 leads) are required for accurate detection of the ischemia. In addition, because acute ischemia is diagnosed on the basis of changes in ST-segment amplitude, standard calibration is necessary. Thus, it behooves critical care nurses, especially those who provide care to patients with acute coronary syndromes, to become knowledgeable in interpreting standard 12-lead ECGs.

An important factor to consider when using ECG findings for clinical decision making is that ECG waveforms are influenced by normal physiological and technical factors as well as by pathophysiological factors. Thus, in this article, we review factors that alter ECG findings and specify which of these alterations interfere with ECG-based diagnosis of myocardial ischemia.

**Physiological Effects on ECG Waveforms**

Physiological factors that affect ECG waveforms include sex, age, ethnicity, height, weight, torso morphology, body mass index, and pregnancy (Figure 1). Such characteristics account for the differences among individuals and produce interindividual variability in ECGs. However, this type of variability is not a problem in continuous monitoring because clinicians do not compare ECGs between patients; rather, they compare sequential ECGs in individual patients. For example, if a patient has an increased anteroposterior chest diameter (“barrel” chest) due to emphysema, which produces ECG waveforms with amplitudes lower than normal, it is reasonable to assume that every ECG recorded in this patient will have the same alteration in amplitudes.

**Sex**

Women generally have smaller thoracic cavities, hearts, and coronary vessel diameters than do men. Women also typically have shorter PR intervals, shorter QRS durations, and longer QT intervals on resting 12-lead ECGs. In addition, women have smaller amplitudes of R, S, and T waves across the precordium. Influences due to sex tend to be important in young adulthood but diminish with age, so the differences are not clinically significant in older women. Indications of ischemia on an ECG are often ambiguous in women. Possible reasons for the ambiguity include (1) less cardiac muscle mass than in men; (2) presence of endogenous estrogens that cause a digitalis-like effect, producing depression of the ST segment; (3) changes in repolarization associated with mitral valve prolapse, which is more common in women than in men; (4) lower hematocrits and fewer total circulating red cells than in men; and (5) attenuation of the ECG signal by breast tissue.

Reported differences between men and women in ST-segment deviation during myocardial ischemia vary, depending on the study. Dellborg et al reported that men in the Thrombolysis Early in Acute Heart Attack Trial who had acute myocardial infarction had significantly more ST-segment elevation than did women in the trial. Thus, the pattern of high-amplitude ST-segment elevation in patients with acute anterior myocardial infarction (“tombstone” pattern) rarely occurs in women. However, Pelter et al found no sex-related differences between men and women in ST-segment deviation during inflation of coronary angioplasty balloons. Thus, it remains unclear whether sex-related differences exist in myocardial ischemia.

**Age**

Both amplitudes and intervals of ECG waveforms change as patients become older. In general, the QRS voltage tends to increase up to the age of 30 years and decreases thereafter. In the longitudinal Framingham study, ECGs recorded 40 years after the baseline measure in the same person showed a decrease in the QRS amplitude. In addition, whereas the QRS duration decreases, the PR interval increases slightly with age. However, changes in the PR interval and QRS duration most likely do not interfere with accurately detecting ECG evidence of myocardial ischemia.

Olbrich and Woodford concluded that ECG findings in elderly patients are influenced by the position of the heart, that is, the rotation and position of the
heart in relation to the chest wall. With advancing years, the position of the heart changes for a number of reasons. First, the shape of the thorax is altered because of the shrinkage of intervertebral disks that may reduce a person’s height by 6 to 8 cm. Second, the diaphragm takes a lower position in the thorax, so the space where the heart lies actually increases with age. Third, lung tissue becomes less elastic and atrophies in senile atrophic emphysema, so the heart has a greater degree of freedom in its movement; greater movement can cause changes in the ST segment during changes in body position that may trigger false ST-segment alarms. Fourth, increased tortuosity and elongation of the aorta with aging may affect the position of the heart. Because of these age-related changes, the heart may rotate differently because of its altered position in the thoracic space in older patients. Such rotation will change a person’s frontal plane QRS axis.

Ethnicity

Nearly all ECG standards were developed by using North American white populations, and the applicability of the standards to other ethnic groups has been questioned. Early studies indicated significant differences in baseline ECGs among various ethnic groups. For example, unlike their white counterparts, healthy adults of African descent often have an early repolarization pattern of diffuse ST-segment elevation beginning at the J point (where the QRS complex ends and the ST segment begins). Japanese men and women have greater precordial voltages than do their white counterparts, a difference that must be considered when diagnosing ventricular hypertrophy. In contrast, young Chinese men and women have significantly lower precordial voltages than do white men and women.

Although ethnic differences have been reported, ECG computer interpretation algorithms do not make distinctions among ethnic groups. However, because clinicians do not compare ECGs between patients, ethnic variation mimicking ischemia is limited.

Body Physique

Four anthropological measurements of body physique influence ECG waveforms: height, weight, torso
morphology, and body mass index. Compared with height and weight, body mass index may be a better measure of the influence of obesity on ECG findings.  

Obesity is thought to cause an upward shift of the diaphragm, resulting in a more horizontal position of the heart, a change that influences the QRS axis. In addition, subcutaneous fat of the torso generally dampens ECG amplitude, partly because of the higher resistance of fat tissue to the electrical impulse and partly because of the greater distance from the heart to the electrode. 

Abdominal distension caused by pregnancy shifts the heart horizontally. In a well-designed study in which subjects served as their own controls, variations of the mean electrical axes of P, QRS, and T waves were evaluated in 52 healthy women during pregnancy and after delivery. The results indicated a leftward deviation in the axes during pregnancy that resolved 5 days after delivery. Because the effects of body physique do not have a sudden onset, during continuous ECG monitoring, they most likely do not mimic changes associated with myocardial ischemia.

Summary of Physiological Influences

Most physiological influences do not pose a significant problem in ECG-based diagnosis of acute myocardial ischemia. These influences produce long-term ECG changes that most likely would not be mistaken for the transient changes associated with ischemia, which typically occur during a period of a few minutes to several hours. Probably, the physiological factor with the greatest potential for misdiagnosis is the pattern of early repolarization of ST-segment elevation normally observed in some patients. This change may be erroneously interpreted as evidence of acute myocardial ischemia (Figure 2). When such an ECG pattern occurs in a patient who comes to the emergency department because of chest pain, a prior ECG should be sought for comparison. If the same pattern of ST-segment elevation is evident on prior ECGs, the changes most likely are due to early repolarization rather than to acute ischemia.

Pathophysiological Effects on ECG Waveforms

The pathophysiological influences on ECG waveforms are numerous and have been well studied. Among them are pacemakers, left bundle branch block (LBBB), coronary artery disease, electrolyte imbalances, ventricular hypertrophy, acute pericarditis, pneumothorax, subarachnoid hemorrhage, and hypothermia.

Pacing

Although atrial pacing does not alter the QRS complex or the ST segment, ventricular pacing produces wide, bizarre QRS complexes with secondary changes in the ST-T segment. Kozlowski et al reported that the expected morphology in patients with ventricular pacers is one of discordance between the QRS complex and the ST-T segment. Thus, diagnosing transient ischemia is difficult in patients with ventricular paced rhythms because the ST-segment morphology is changed by the altered intraventricular conduction. However, in certain instances, ST-segment deviation during acute myocardial ischemia is great enough to alter the normal baseline ventricular pacing pattern to the point that detection of ischemia is possible.

Left Bundle Branch Block

Diagnosis of myocardial infarction in patients with LBBB can be difficult and may be impossible. Consequently, patients with LBBB and suspected acute myocardial infarction may receive suboptimal treatment. Recently, to test ECG criteria for the diagnosis of acute myocardial infarction in the presence of LBBB, researchers analyzed ECGs of patients enrolled in the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries trial. ECGs of patients with LBBB and acute myocardial infarction were blindly compared with the ECGs of control patients who had chronic coronary artery disease and LBBB. The researchers developed and validated clinical criteria based on changes in the ST segment that may help detect patients with acute myocardial infarction, who can then receive appropriate treatment. The 3 ECG criteria with independent value in the diagnosis of acute infarction in these patients were an ST-segment elevation of 1 mm or more that was concordant with (in the same direction as) the QRS complex; ST-segment depression of 1 mm or more in lead V1, V5, or V6; and ST-segment elevation of 5 mm or more that was discordant with (in the opposite direction from) the QRS complex. However, the specificity of the proposed criteria has recently been disputed.

Coronary Artery Disease

Coronary artery disease may affect the amplitude of the ST segment by causing either transient ischemia or injury. These changes can range from subtle deviations in the ST segment of 100 µV (1 mm on standardized ECG tracings) in a single lead to massive deviations as great as 1 mV (10 mm on standardized ECG tracings) in multiple leads. ECG evidence of myocardial ischemia is generally defined as a deviation of 1 to 2 mm in the ST segment (elevation or depression) that persists at least 1 minute. In addition, acute myocardial ischemia may also cause changes in the T wave, including T-wave inver-
sions 1 mm or greater; deeply inverted T waves, especially in the precordial leads; and tall, hyperacute T waves. Normal T waves should be 5 mm or less in the limb leads and 10 mm or less in the precordial leads.29

Electrolyte Abnormalities

Electrolyte imbalances are common in hospitalized patients because of renal failure, starvation, malabsorption syndromes, ketoacidosis, severe vomiting, diarrhea, burns, and so forth. Electrolyte influences on the ECG can be classified into 3 categories: effects on depolarization (P wave and QRS complex), effects on repolarization (ST segment and T wave), and effects on impulse formation and conduction (sinus, atrioventricular, and intraventricular blocks).

The main electrolyte abnormalities that alter ECG findings are hypokalemia, hyperkalemia, hypocalcemia, and hypercalcemia (see Table). Either abnormally low or abnormally high serum levels of potassium or calcium may produce marked ECG abnormalities.30 Hypokalemia (potassium levels <2.7 mmol/L) causes prolongation of the action potential, which may produce ST-segment depression of about 0.5 mm or cause T-wave inversion. Hyperkalemia (potassium levels 5.5-6.5 mmol/L) causes shortening of the action potential, which produces tent-shaped, peaked T waves. ST-segment depression may also occur because of shortening of the plateau phase of the action potential. Hypercalcemia (calcium levels <3.1 mmol/L [<6.2 mEq/L]) shortens the plateau phase of the action potential, causing the QT interval to shorten to the degree that the ST segment may disappear altogether. Conversely, hypocalcemia (calcium levels <1.8 mmol/L [<3.6 mEq/L]) increases the length of the plateau phase of the action potential, a change that lengthens the ST segment and QT interval.

Ventricular Hypertrophy

Another pathophysiological influence on ECG waveforms is ventricular hypertrophy, which is due to chronic pressure overload that enlarges myocardial muscle fibers (eg, hypertension or aortic stenosis). This increase in ventricular mass is often evident on ECGs as

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**Table 1** Electrocardiographic changes related to electrolyte imbalances

<table>
<thead>
<tr>
<th>Electrolyte imbalance</th>
<th>Electrocardiographic effects</th>
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<td>Hypokalemia</td>
<td>ST-segment sagging and low-amplitude T wave</td>
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<td></td>
<td>Prominent U wave</td>
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<tr>
<td>Hyperkalemia</td>
<td>Early: tentlike, peaked T waves</td>
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<td></td>
<td>Late: absence of P waves; wide QRS complexes</td>
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<tr>
<td>Hypocalcemia</td>
<td>Long ST segment, normal T wave</td>
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<td>Short or normal QT interval</td>
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greater than normal QRS amplitudes. Patients with left ventricular hypertrophy may also have a “strain pattern”: ST-segment depression in leads I, aVL, V₅, and V₆.

**Drugs**

Many drugs influence ECG waveforms. One is worth mentioning here because its effects may be misdiagnosed as myocardial ischemia. Digitalis alters repolarization of the myocardium, resulting in a negative scooped or “bowl-like” ST segment and/or a flattened T wave. In addition, the J point may be depressed.

**Acute Pericarditis**

Inflammation of the pericardial sac, which is directly adjacent to the epicardial layer of the myocardium, causes global ST-segment elevation. In contrast, changes in the ST segment associated with myocardial injury are limited to a few ECG leads that lie directly over the myocardium supplied by the occluded coronary artery. Clinicians should suspect pericarditis when ST-segment elevation occurs in both “anterior” leads (ie, precordial leads) and “inferior” leads (ie, leads II, III, and aVF), because epicardial injury most likely would not involve 2 coronary artery domains at the same time.

**Pneumothorax**

ECG manifestations of left-sided tension pneumothorax include a rightward shift of the frontal plane QRS axis, reduced precordial R-wave amplitude, QRS alternans (alternating amplitude), and precordial T-wave inversion. In general, these changes do not mimic those associated with myocardial ischemia; however, 2 case studies reported ECG changes consisting of precordial ST-segment elevation suggestive of acute myocardial infarction. In both cases, immediately after placement of a chest tube, the ECG findings returned to nearly normal and serial measurements of cardiac biomarkers indicated no evidence of acute myocardial infarction. Strizik and Forman suggested that the ECG changes may be related to the hypotensive state induced by the tension pneumothorax, with resulting decreased coronary artery blood flow and myocardial ischemia that resolves without infarction. Kamimura et al theorized that the ECG changes suggest the importance of air insulating the chest wall, rather than cardiac rotation, dilatation, or displacement, as a mechanism of the ECG change related to pneumothorax.

**Subarachnoid Hemorrhage**

ECG abnormalities often occur in patients with subarachnoid hemorrhage. These changes primarily affect repolarization, which involves the ST segment, the T wave, the U wave, and the QT interval (Figure 3). In addition to lengthened QT intervals, patients with subarachnoid hemorrhage also have ECG changes (eg, ST-segment deviation) that mimic those associated with myocardial ischemia. Neurologically induced ECG changes are not well understood and merit further research.

**Hypothermia**

ECG changes also develop at lower than normal body temperatures (<36.6°C). All ECG intervals may lengthen (PR, QRS, and QT) along with a progressive slowing of the sinus rate. The appearance of an enhanced J wave, also called the Osborne wave or camel-hump sign, is the most striking and classic ECG finding in patients with hypothermia. The camel-hump sign may also occur in patients with cerebral injuries and can mimic the ST-segment elevation associated with Prinzmetal angina (coronary vasospasm). Low temperatures during angiography and surgical interventions may also produce ST-segment abnormalities.

**Summary of Pathophysiological Effects**

In summary, ECG changes due to pathophysiological influences that are most likely to be misdiagnosed as evidence of acute myocardial ischemia include (1) ST-segment depression due to a left ventricular hypertrophy strain pattern, (2) ST-segment depression due to digitalis therapy, and (3) ST-segment elevation due to acute pericarditis. The first 2 conditions produce ST changes that persist for years; the third condition produces ST changes that usually persist for days. Thus, all 3 conditions can be distinguished from acute ischemia, which typically lasts minutes to hours. Although ventricular arrhythmias, ventricular pacing rhythms, and bundle branch blocks alter the ST segment dramatically, they can be distinguished from acute ischemia on the basis of a widened QRS complex.

**Technical Effects on ECG Waveforms**

**Noisy Signal**

Nonphysiological artifact potentials can distort ECG findings. An artificial signal is anything on an ECG that is not caused by the electrical currents generated by the heart. Artifacts include 60-cycle electrical interference, movement by the patient causing “baseline” wander, muscle tremor, and loss of electrode contact. Surrounding electrical interferences common in hospital environments can compromise signal quality. For example, equipment plugged into an outlet but turned off still generates an electrical field called low-frequency interference. Low-frequency noise cannot be filtered without compromising the ECG complex because of its similarity to the signal frequency of the
Inconsistent Electrode Positioning

The most important issue regarding lead placement for ST-segment monitoring is keeping electrodes in the same location over time. Because diagnostic 12-lead ECG criteria are often lead specific, serial changes in the ST segment cannot be interpreted accurately if electrode placement is inconsistent over time. The recommended standard for ensuring consistent electrode placement is marking the location of the electrodes with indelible ink so that any electrodes removed for any reason can be replaced in their original location. If electrode locations must be changed because of skin breakdown, wounds, defibrillator pads, and so forth, a notation should be made on the 12-lead ECG so that the resultant changes in the ST segment are not mistaken for evidence of acute myocardial ischemia.

Body Position

The effect of changes in body position is a particularly difficult problem for ST-segment monitoring for the following reasons: Similar to ischemia, a change in body position can cause transient changes in the ST segment. The effect of changes due to ischemia, changes in body position are not associated with a wide, bizarre QRS complex, which would normally indicate ventricular arrhythmia, pacing, or bundle branch block. A patient’s position during continuous ECG monitoring cannot be determined for a particular tracing unless a nurse happens to be in the patient’s room when the ECG tracing in question is recorded. In the recently published guideline

Figure 3 A 12-lead ECG of a 42-year-old woman with subarachnoid hemorrhage. The changes in the T wave mimic those associated with acute myocardial ischemia: T-wave inversions of 1 mm or greater and deeply inverted T waves, especially in the precordial leads.
Conclusions

A review of physiological, pathophysiological, and technical factors that influence ECG waveforms indicates that most factors do not interfere with an accurate diagnosis of myocardial ischemia based on ECG findings. The factors that pose the most risk of misdiagnosis include early repolarization, a left ventricular hypertrophy strain pattern, acute pericarditis, digitalis therapy, inconsistent electrode placement, a noisy signal from inadequate skin preparation, and changes in body position. Often, clinicians can distinguish these factors from ischemic events by noting that the changes persist for a longer period than do the changes in the ST segment (minutes or hours) that occur in patients with acute coronary syndromes.

An important leadership role for advanced practice nurses (clinical nurse specialists and nurse practitioners) is to become knowledgeable in 12-lead ECG interpretation and to share this knowledge with staff nurses who provide care for patients with acute coronary syndromes.

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