Understanding the 12-lead ECG part II

Learn to recognize bundle-branch blocks, myocardial infarction, and common dysrhythmias.

BY GUY GOLDICH, RN, CCRN, MSN

LAST MONTH, I described the components of the 12-lead electrocardiogram (ECG) and how to recognize a normal ECG. In this article, I'll explain some advanced techniques that you can use to interpret common ECG abnormalities: bundle-branch blocks, myocardial infarction (MI), and common dysrhythmias.

Bundle-branch blocks: Obstruction in the conduction

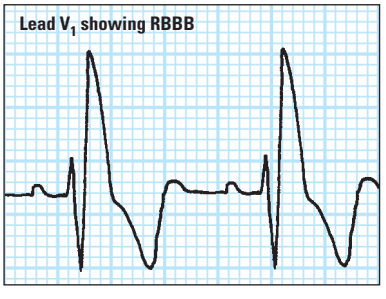
Probably the most common ECG abnormality you'll encounter is a bundle-branch block, which appears on the ECG as a wider-than-normal QRS complex (more than 0.12 second in duration). As you know, the cardiac impulse, originating in the sinoatrial (SA) node, normally travels through the bundle of His into the right and left bundle branches in the septum. The two bundle branches terminate in the Purkinje fibers. When the impulse reaches them, ventricular depolarization begins.

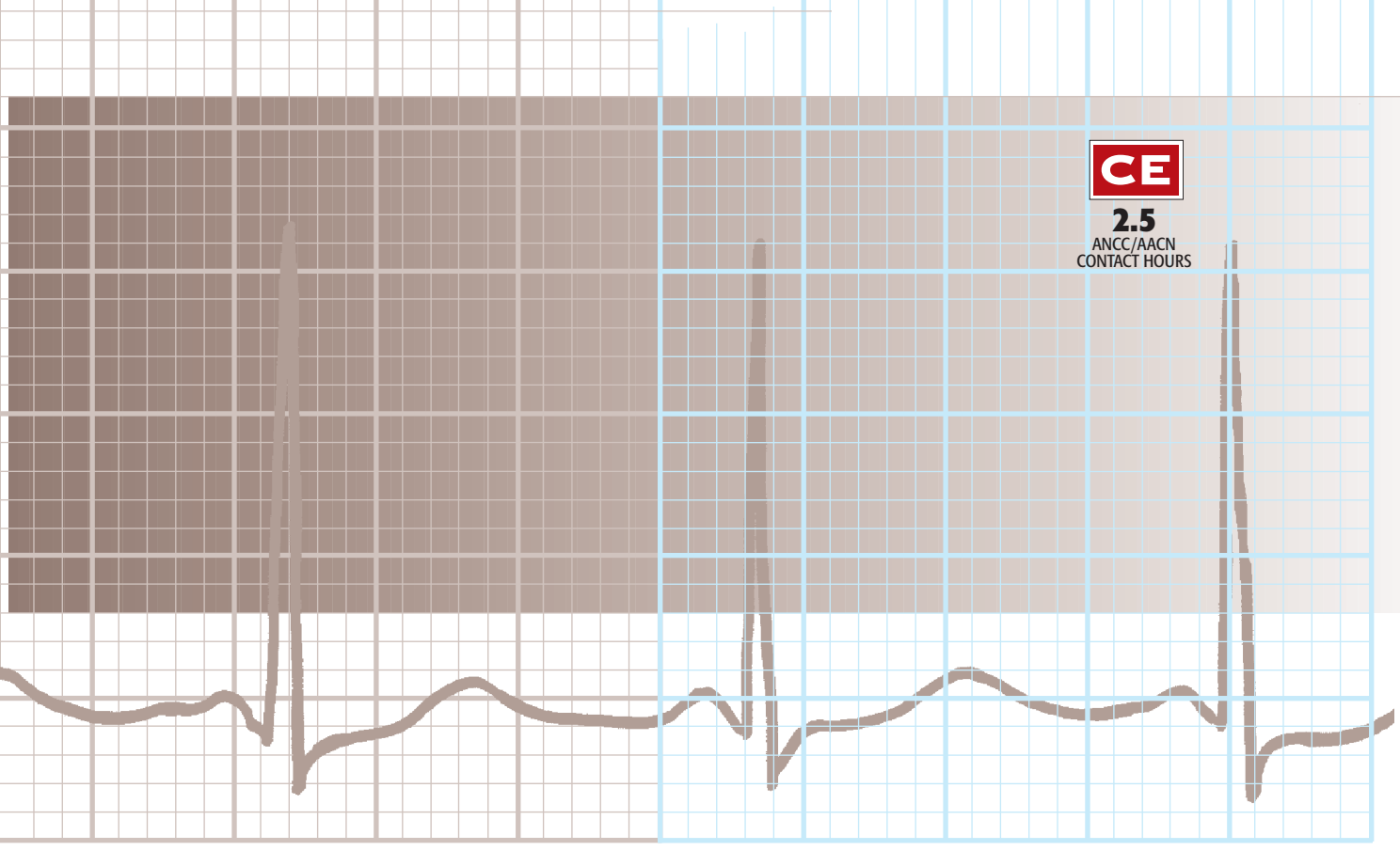
Normally the impulse is delivered to myocardial cells on both sides of the heart simultaneously, so depolarization begins at the same time on both sides of the heart. The result is a very fast, synchronous contraction of the ventricles. On ECG, the normal QRS complex duration from two intact bundle branches is 0.12 second or less (three or fewer small squares of the ECG paper).

A bundle-branch block occurs when one of the two bundle branches can't conduct the cardiac impulse to the myocardial cells. The most common cause of chronic bundle-branch block is ischemic heart disease. When an artery supplying the bundle branch narrows, the flow of oxygenated blood is reduced and the bundle branch can't conduct impulses normally.

A common cause of acute bundle-branch block is acute MI. If the MI involves the ventricular septum, one of the bundle branches may become infarcted, leading to a loss of conduction. Although uncommon, physical injury of a bundle branch during an invasive procedure such as cardiac catheterization or heart surgery also may produce a bundle-branch block.[1]

• In a **right bundle-branch block (RBBB),** impulse conduction to the right ventricle is blocked. The cardiac impulse is conducted only to the left side of the heart where left ventricular depolarization





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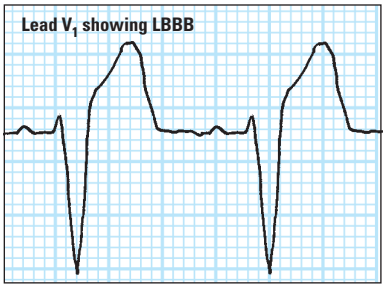
begins. The right side of the heart depolarizes only in response to the cell-to-cell wave of depolarization that travels from the left side of the heart. This cell-to-cell depolarization is much slower than the normal synchronous depolarization; that's why the QRS complex is significantly wider than normal.

Examine lead V_1 to identify an RBBB. In lead V_1, the normal QRS complex consists of a small R wave, then a large S wave. As you recall, lead V_1 looks at the right side of the heart. A small vector originating in the septum toward V_1 creates a small upward R wave, then the predominant mean QRS vector creates the large S wave as the mean QRS vector flows away from lead V_1.

In RBBB, the path of the mean QRS vector is changed due to left-to-right slow conduction; lead V_1 now records a delayed R wave approaching it, resulting in a positive R wave. So the key identifier of RBBB in lead V_1 is a QRS complex wider than 0.12 second with a delayed (longer than 0.07 second) positive main R wave. Some RBBBs may display a triphasic waveform ("rabbit ears") consisting of a small r wave, downward S wave, and a second, larger R wave.[2]

• In a **left bundle-branch block (LBBB),** electrical impulses don't reach the left side of the heart normally, so once again, synchronous depolarization of the ventricles doesn't occur. Depolarization begins in the right side of the heart and travels in a right-to-left direction via slow cell-to-cell depolarization. Lead V_1 records the mean QRS vector directed away from its positive lead, resulting in a wide downward complex. Because the mean vector takes a relatively longer time to cross to the left side of the heart, the QRS complex is wider than 0.12 second. The key to recognizing an LBBB is a wide, downward S wave or rS wave in leads V_1 and V_2.

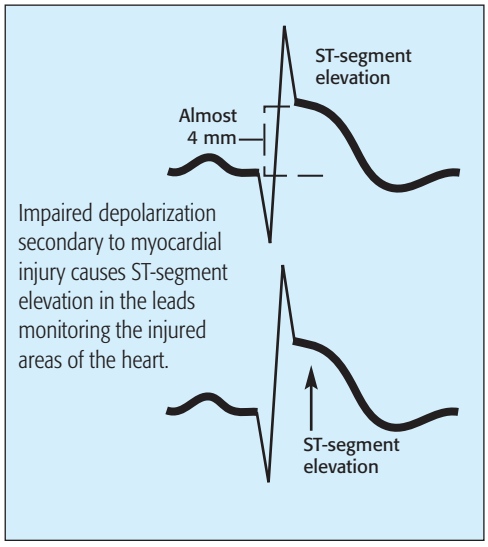


Recognizing an MI

One of the most critical functions of the 12-lead ECG is to determine whether a patient is experiencing an acute MI. A series of predictable ECG changes that occur during an MI help you identify it quickly and initiate appropriate treatment.

Among one of the earliest changes in the ECG tracing is an elevation of the ST segment, indi-

Understanding ST-segment elevation

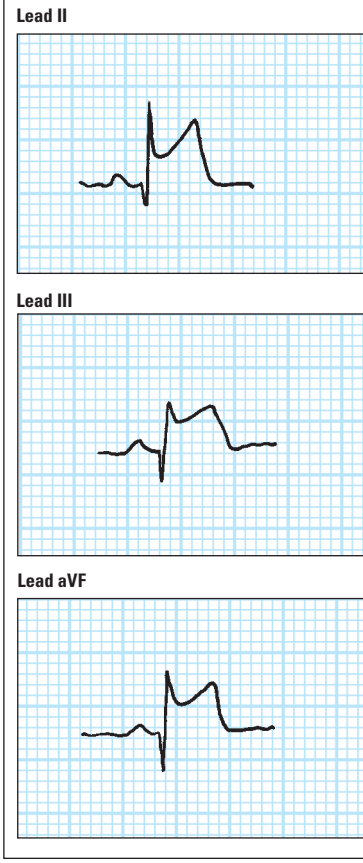


cating reversible myocardial injury (see *Understanding ST-segment elevation*). In a normal ECG, the ST segment is level with the tracing's baseline. When myocardial cells sustain injury from MI, depolarization is impaired, resulting in ST-segment elevation in the leads monitoring the affected areas of the heart. An ST-segment-elevation MI (STEMI), the most serious type of MI, is associated with more complications and a higher risk of death.[3]

The leads with ST-segment elevations identify the area of myocardial injury, so you can determine the region of the heart affected by knowing which area is monitored by which ECG lead. Let's look at some examples.

• Because leads II, III, and aVF all monitor the inferior (or bottom) wall of the heart from slightly different directions, they're usually

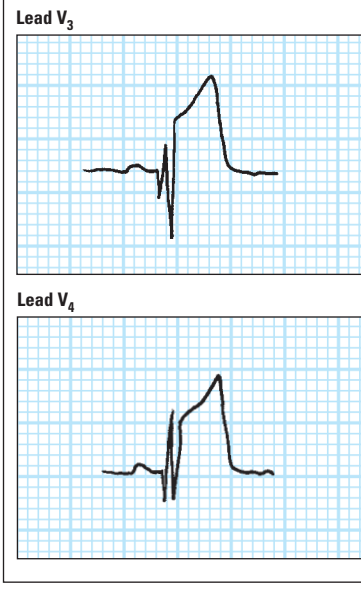
Inferior-wall STEMI



described as the inferior leads. This area of the heart is perfused by the right coronary artery. A patient with a STEMI involving the inferior wall of the heart will have elevated ST segments in leads II, III, and aVF (see *Inferior-wall STEMI*).

• Another common infarct lead pattern occurs when an MI involves the intraventricular septum, which is perfused by the left anterior descending (LAD) coronary artery. In a *septal MI*, the leads monitoring the septum's electrical activity will display elevated ST segments. Precordial (or chest) leads V_1 and V_2, which are

Anterior-wall STEMI

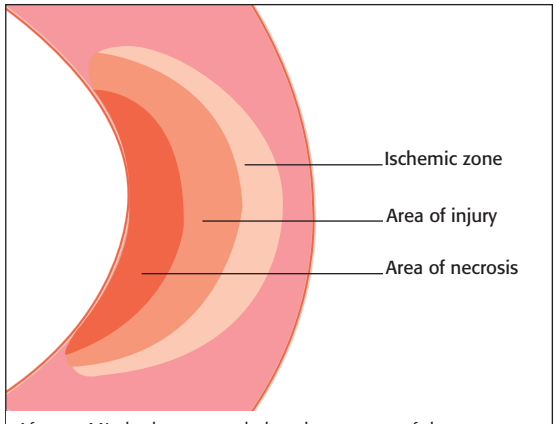


located on the anterior chest wall directly over the septum, most accurately monitor the septum's electrical activity. (These leads also are known as the septal leads.) The patient experiencing a septal MI will have ST-segment elevations in leads V_1 and V_2.

• Directly to the left of the septal area of the heart is the large frontal or *anterior* wall of the heart, which is also perfused by the LAD coronary artery. As the most muscular and powerful pumping wall of the heart, the anterior wall is responsible for a large proportion of cardiac output. Anatomically, leads V_3 and V_4 are located directly above the anterior wall of the heart and monitor its electrical activity. An anterior-wall STEMI will cause the ST segments in these leads to be elevated (see *Anterior-wall STEMI*).

• The lateral wall of the heart, perfused by the left circumflex artery,

Tissue damage after MI



After an MI, the heart muscle has three zones of damage. Necrotic tissue dies from lack of blood flow. Injured cells may recover and ischemic cells can be saved if the area is reperfused promptly.

is located to the left of the anterior wall and follows the curve of the left lateral chest wall. Relatively muscular, it also contributes significantly to the heart's pumping ability. The ECG lead pattern that monitors the lateral wall's electrical activity is more complex because the lateral wall is monitored by a combination of precordial (chest) leads and frontal (limb) leads.

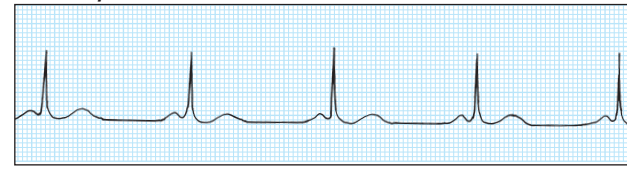
Chest leads V_5 and V_6 are located on the left lateral chest wall and monitor electrical activity by looking down at the lateral heart wall. Leads I and aVL also monitor the lateral wall's electrical activity. A patient with a lateral-wall STEMI will have ST-segment elevations in leads I, aVL, V_5, and V_6.

Although patients can have MIs affecting a single heart wall, such as a discrete septal MI or anterior-wall MI, the area of infarction may involve more than one area of the heart. In such a case, ST-segment elevations appear in the leads monitoring all of the involved areas. For example, if the infarction extends into both the septum and the anterior wall, the ST-segment elevations would appear in leads V_1, V_2, V_3, and V_4. The areas involved in the MI are reflected by the descriptive name; in this case, an *anterioseptal* MI. For information on how MI affects heart muscle, see *Tissue damage after MI*.

Identifying common dysrhythmias

Now let's examine some common ECG rhythm abnormalities you may encounter in your practice, keeping in mind that you always treat the patient, not the rhythm. When you find an abnormal rhythm or a rhythm change, assess your patient and document level of consciousness, vital signs, chest pain, shortness of breath, and other signs and symptoms associated with the dysrhythmia. By using your assessment skills, nursing judgment, and knowledge of ECGs, you can determine the level of urgency of the situation.

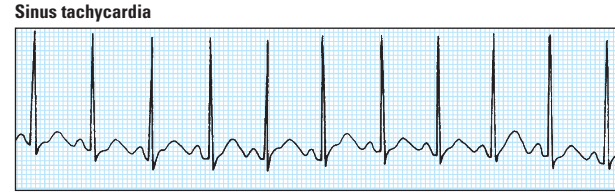
Sinus bradycardia



• **Sinus bradycardia** is a sinus rhythm slower than the lower normal sinus rate of 60 beats/minute. The P waves, QRS complexes, and T waves are all normal. Sinus bradycardia is commonly caused by ischemic heart disease that causes the SA node to malfunction. Sinus bradycardia can also be caused by acute MI and some types of medications, such as beta-blockers. Well-conditioned athletes may have normal resting heart rates slower than 60 beats/minute. Assess your patient for hemodynamic stability if he has a new or profound sinus bradycardia. Contact the health care provider if your patient is symptomatic. Signs and symptoms that may accompany sinus bradycardia include hypotension, lethargy, fatigue, chest pain, and difficulty breathing. Be prepared to transfer your patient to the intensive care unit (ICU) for a temporary pacemaker.

• **Sinus tachycardia** is a sinus rhythm that's faster than the

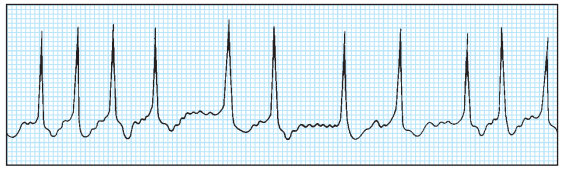
Sinus tachycardia



upper normal sinus rate of 100 beats/minute. Sinus tachycardia can produce heart rates of 100 to 150 beats/minute. At faster rates, the heart's myocardial oxygen demand increases, and a patient with preexisting heart disease may experience chest pain or other cardiac symptoms. Sinus tachycardia usually is related to a physiologic cause, such as fever, infection, pain, physical exertion, anxiety, hypoxia, or shock. If you can identify and treat the cause, the heart rate will usually decrease. To manage sinus tachycardia with an unknown cause, the health care provider may order a beta-blocker such as metoprolol or atenolol.[4]

• **Atrial fibrillation (AF),** one of the most common dysrhythmias you'll see in practice, has two predominant characteristics: an irregularly irregular heart rhythm and no meaningful P waves. Normally, after passive ventricular filling, the atria contract regularly and eject their load of blood into the ventricles (atrial kick). In AF, atrial kick is lost. Instead of contracting normally, the atria quiver due to random and chaotic depolarization of atrial cells. The random atrial depolarization is also responsible for the irregular ventricular rate, which can vary from 40 to 180 beats/minute. Atrial fibrillation has many causes, including atrial enlargement from chronic obstructive pulmonary disease or other lung disease, thyroid disease, ischemic heart disease, acute MI, stress or fatigue, and excessive use of caffeine, alcohol, or cigarettes.

Atrial fibrillation



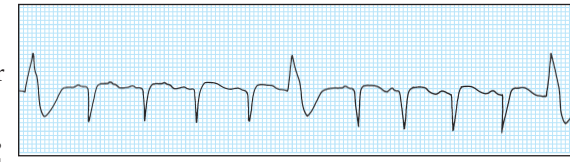
You may first encounter AF during a routine vital signs check. If your patient has a new irregular heart rate, or has an abnormally fast or slow heart rate, obtain an order for a 12-lead ECG. Look for an irregularly irregular rhythm and f waves, the two hallmarks of AF. Perform a thorough physical assessment because patients can rapidly become hemodynamically unstable or develop worsening heart failure. If your patient has unstable or symptomatic AF, administer supplemental oxygen and establish or maintain intravenous (I.V.) access before transferring him to the ICU or telemetry unit for treatment with I.V. diltiazem or a beta-blocker.

If the patient is stable, the health care provider may order various oral medications to control or convert AF, such as digoxin, diltiazem, amiodarone, or metoprolol.

All patients with AF lasting more than 48 hours are at high risk for thrombus formation because of irregular blood flow in the atria. If released into the circulation, these thrombi can cause arterial obstruction resulting in life-threatening complications such as stroke. As ordered, administer I.V. heparin and start the patient on oral warfarin to prevent thrombus formation.[5]

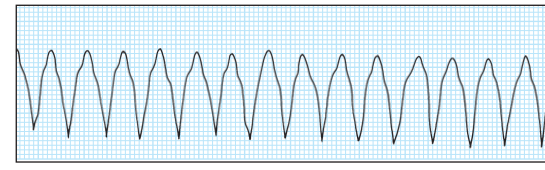
• **Premature ventricular contractions (PVCs)** are characterized by a wide, abnormal QRS complex because conduction is through the ventricular tissue and not the His-Purkinje system. Look for a QRS greater than 0.12 second that appears large, abnormal, and premature (occurring *before* the next sinus beat). Caused by irritable ventricular tissue that depolarizes early and unpredictably, PVCs can be triggered by heart failure, electrolyte imbalances, stimulants such as caffeine, hypoxia, acute MI, mitral valve prolapse, thyroid disease, and injury or infarct of the myocardial tissue. Rare or isolated PVCs seldom require aggressive treatment. However, if you notice that the frequency of PVCs is increasing, or if you see new groups or "runs" of PVCs, contact the health care provider for further evaluation.

Isolated PVCs



• **Ventricular tachycardia (VT)** is a very rapid (100 to 250 beats/minute) series of wide-complex ventricular depolarizations. In this dysrhythmia, abnormal ven-

Ventricular tachycardia



tricular tissue rapidly depolarizes, taking rhythm control away from the sinus node. Along with the rapid rate, VT is characterized by wide, bizarre QRS complexes usually followed by large T waves in the opposite direction of the major QRS deflection.

If your patient is unconscious, apneic, and pulseless, call a code and start cardiopulmonary resuscitation. If your patient has a pulse and is awake, treat this situation as a medical emergency. Call for the physician stat (and the rapid response team, if your facility has one), bring a crash cart with a monitor/defibrillator to the bedside, and prepare to transfer the patient to the ICU.

Summing up

Like any new skill, interpreting 12-lead ECGs takes practice and commitment. Make a habit of reviewing your patients' ECGs routinely. Seek guidance from colleagues experienced in ECG interpretation, such as senior staff nurses, clinical nurse specialists, and clinical nurse-educators. Many physicians will also be happy to review an ECG with you if they know you're interested in learning to spot problems early.

With practice and experience, ECG interpretation will become a valuable nursing tool, helping you to recognize problems promptly and provide even better patient care.

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Understanding the 12-lead ECG, part II

GENERAL PURPOSE To provide nurses with techniques to analyze common ECG abnormalities. **LEARNING OBJECTIVES** After reading the preceding article and taking this test, you should be able to: **1.** Differentiate between LBBB and RBBB. **2.** Determine the relationship between leads with ECG changes and areas of myocardial damage. **3.** Identify common dysrhythmias.

1. A bundle-branch block occurs when one of two bundle branches can't conduct cardiac impulses to the
a. atria.
b. bundle of His.
c. myocardial cells.
d. atrioventricular node.

2. The *most common* cause of a chronic bundle-branch block is
a. ischemic heart disease.
b. an acute MI.
c. an acute coronary syndrome.
d. trauma to one of the bundle branches.

3. In addition to a QRS complex wider than 0.12 second, which ECG abnormality is a key indicator of an RBBB?
a. a negative R wave in lead V_1
b. a positive R wave in lead V_3
c. a negative R wave in lead V_3
d. a positive main R wave in lead V_1

4. Which is one of the earliest changes indicative of reversible myocardial injury?
a. QT prolongation
b. Q-wave deepening
c. ST-segment elevation
d. PR interval shortening

5. Which leads view the inferior wall of the heart?
a. V_1 through V_6
b. I, II, and III
c. I and aVL
d. II, III, and aVF

6. Elevated ST segments in V_1 and V_2 indicate which type of MI?
a. anterior wall
b. septal wall
c. lateral wall
d. inferior wall

7. Which heart wall is perfused by the circumflex branch of the left coronary artery?
a. lateral
b. inferior
c. anterior
d. septal

8. A patient with ST-segment elevation in leads I, aVL, V_5, and V_6 may have
a. an anterior-wall MI.
b. an inferior-wall MI.
c. a lateral-wall MI.
d. a posterior-wall MI.

9. An anterioseptal MI would have ST elevation in leads
a. V_1 and V_2 only.
b. V_3 and V_4 only.
c. V_5 and V_6 only.
d. V_1 through V_4.

10. Which *isn't* a possible cause of sinus bradycardia?
a. atropine
b. beta-blockers
c. acute MI
d. a well-conditioned heart

11. Sinus tachycardia usually is related to
a. medications.
b. an MI.
c. a physiologic cause.
d. ischemic heart disease.

12. One hallmark of AF is
a. a regularly irregular rhythm.
b. a prolonged QRS complex.
c. an irregularly irregular rhythm.
d. a prolonged PR interval.

13. Atrial contraction just before ventricular contraction is called
a. atrial kick.
b. repolarization.
c. synchrony.
d. f waves.

14. Which medication may be used to treat AF?
a. lidocaine
b. atropine
c. captopril
d. diltiazem

15. Premature ventricular contractions are characterized by wide, abnormal
a. QRS complexes.
b. U waves.
c. P waves.
d. T waves.

16. If your patient is in pulseless VT, which action would you take first?
a. Call the rapid response team.
b. Obtain a stat 12-lead ECG.
c. Call a code.
d. Ensure adequate vascular access.

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