William Einthoven, who used a string galvanogram for his recordings, first invented the ECG in 1901. This time-honored and relatively simple tool remains, even today, a mainstay in clinical cardiac diagnosis and therapy. In the perioperative setting, the ECG serves two main functions: diagnosis and monitoring. In the preoperative period, standard 12-lead ECG is predominantly used for risk assessment. It provides information on the patient’s baseline (chronic) cardiac status with regard to myocardial ischemia and conduction or rhythm abnormalities as part of the entire preoperative clinical assessment.

Occasionally, the preoperative ECG may reveal acute or new abnormalities, especially with urgent or emergent operations, and in comparing current with previous ECGs. During and after surgery, the ECG can detect changes in rate and rhythm or myocardial ischemia. With many patients coming to surgery with pacemakers or implantable cardiac defibrillators in place, the ECG monitor enables the anesthesiologist to follow the proper function of these devices during the surgical procedure. (Perioperative management of these devices is described in Chapter 48.) During cardiac surgery, both myocardial...
ischemia and arrhythmias are common, especially after separating from the cardiopulmonary bypass. The proper diagnosis and management of intraoperative ischemia and arrhythmias is especially part of the expertise of the cardiovascular anesthesiologist (see Chapters 67 and 68). A postoperative 12-lead ECG is often obtained in patients who are at high risk for developing new ischemic or rhythm changes. In all these scenarios, a good understanding of the principles of the ECG and its abnormalities is vital for the anesthesiologist. This chapter covers most aspects of ECG interpretation with a special emphasis on issues relevant to anesthesia and perioperative care.

**ELECTROCARDIOGRAPHIC LEAD SYSTEMS**

### STANDARD RECORDING ELECTRODES AND LEADS

The small currents produced by the electrical activity of the cardiac muscle spread an electrical field throughout the body, which behaves as a volume conductor, allowing it to be recorded at various sites on the surface of the body as ECG signals. Electrodes (leads) placed at specific locations record the electrical potentials reaching the skin, and the output of these leads is amplified, filtered, and displayed. Two types of ECG leads are used: bipolar and unipolar. A bipolar lead consists of two electrodes placed at two different sites to measure the difference in potentials between these two electrodes. Unipolar leads measure the absolute electrical potential at one site in relation to a reference, or remote site, at which the potential is deemed to be zero. The standard clinical ECG includes recordings from 12 leads. These 12 leads include three bipolar leads (I, II, and III), six unipolar precordial leads (V₁ through V₆), and three modified unipolar limb leads (augmented limb leads aV₁, aV₂, and aV₃) (Table 47-1).

**TABLE 47-1 LOCATION OF ELECTRODES AND LEAD CONNECTIONS FOR THE STANDARD 12-LEAD ELECTROCARDIOGRAM AND ADDITIONAL LEADS**

<table>
<thead>
<tr>
<th>Lead Type</th>
<th>Positive Input</th>
<th>Negative (Reference) Input</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bipolar Limb Leads</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lead I</td>
<td>Left arm</td>
<td>Right arm</td>
</tr>
<tr>
<td>Lead II</td>
<td>Left leg</td>
<td>Right arm</td>
</tr>
<tr>
<td>Lead III</td>
<td>Left leg</td>
<td>Left arm</td>
</tr>
<tr>
<td>Augmented Unipolar Limb Leads</td>
<td></td>
<td></td>
</tr>
<tr>
<td>aV₁</td>
<td>Right arm</td>
<td>Left arm plus left leg</td>
</tr>
<tr>
<td>aV₂</td>
<td>Left arm</td>
<td>Right arm plus left leg</td>
</tr>
<tr>
<td>aV₃</td>
<td>Left leg</td>
<td>Left arm plus left arm</td>
</tr>
<tr>
<td>Precordial Leads</td>
<td></td>
<td></td>
</tr>
<tr>
<td>V₁</td>
<td>Right sternal margin, 4th intercostal space</td>
<td>Wilson central terminal</td>
</tr>
<tr>
<td>V₂</td>
<td>Left sternal margin, 4th intercostal space</td>
<td>Wilson central terminal</td>
</tr>
<tr>
<td>V₃</td>
<td>Midway between V₂ and V₄</td>
<td>Wilson central terminal</td>
</tr>
<tr>
<td>V₄</td>
<td>Left midclavicular line, 5th intercostal space</td>
<td>Wilson central terminal</td>
</tr>
<tr>
<td>V₅</td>
<td>Left anterior axillary line</td>
<td>Wilson central terminal</td>
</tr>
<tr>
<td>V₆</td>
<td>Left midaxillary line</td>
<td>Wilson central terminal</td>
</tr>
</tbody>
</table>


### STANDARD ELECTROCARDIOGRAPHIC RECORDINGS

The ECG is normally recorded on special paper consisting of grids of horizontal and vertical lines. The distances between the vertical lines represent time intervals, whereas the distances between the horizontal lines represent voltages. The lines are 1 mm apart, with every fifth line intensified. The speed of the paper is standardized to 25 mm/sec. On the horizontal axis, 1 mm represents 0.04 second, and 0.5 cm represents 0.20 second. On the vertical axis, 10 mm represents 1 millivolt (mV). On every recording, a 1-cm (1-mV) calibration mark should indicate that the ECG is appropriately calibrated.

### NORMAL ELECTRICAL ACTIVITY

**P WAVE**

Under normal circumstances, the sinoatrial (SA) node has the most rapid spontaneous depolarization rate and is therefore the dominant cardiac pacemaker. From the SA node, the impulse normally spreads to the atrioventricular (AV) node, through one left-sided and two right-sided pathways. The P wave is the result of normal depolarization of the atria, and its identification is important for the determination of normal sinus rhythm. The anatomically anterior right atrium is activated earlier, and only later does the signal shift posteriorly as activation proceeds over the left atrium. Therefore, the P wave in the right precordial leads (V₁ and, occasionally, V₃) is commonly biphasic, a positive deflection followed by a negative one, whereas in the lateral leads, the P wave is upright and reflects a right-to-left spread of the activation front.

**PR INTERVAL**

The PR interval is the temporal bridge between atrial and ventricular activation, during which the AV node, the bundle of His, the bundle branches, and the intraventricular conduction systems are activated (Fig. 47-1). Most of the conduction delay during this segment is due to slow conduction within the AV node. The normal PR interval measures 120 to 200 milliseconds (msec) in duration.

### MYOCARDIAL ACTIVATION—THE QRS COMPLEX

The QRS complex is the manifestation of left and right ventricular muscle depolarization. Its pattern lead represents the sum of all electrical forces emanating from the wavefront propagation of ventricular electrical excitation aimed at the direction of that lead. Ventricular excitation spreads within several milliseconds from the bundle branches to the His-Purkinje fibers, which are broadly dispersed throughout the entire endocardial surfaces of both ventricles. Excitation of the Purkinje-ventricular
muscle junctions in the endocardium then proceeds by conduction from muscle cell to muscle cell to activate the entire ventricle thickness toward the epicardium. The normal pattern of activation of the ventricles starts in the interventricular septum with a vector oriented from left to right in the frontal plane and anteriorly in the horizontal plane (as determined by the anatomic position of the septum in the chest). This produces an initial small positive R wave in the right-sided leads (aVR and V1) and small negative waves (septal Q waves—less than 30 to 40 msec) in the left-sided leads (I, aVL, V5, and V6). Subsequent QRS complex elements reflect the activation of the free walls of the left and right ventricles. However, because the right ventricular muscle mass is considerably smaller than the left ventricle, in reality, the QRS complex generally represents left ventricular (LV) activity with its main vector proceeding from right to left in the axial plane and anterior, followed by posterior vectors in the horizontal plane (R wave followed by a small S wave in the left-sided leads). The main vector of the QRS complex in the frontal plane serves to calculate the electrical axis of the heart, which is normally between −30 and +90 degrees. An axis more negative than −30 degrees is called left axis deviation, and an axis higher than +90 degrees is termed right axis deviation. The duration of the normal QRS complex is less than 120 msec (Fig. 47-2).

ST SEGMENT AND T WAVE

Repolarization of the ventricles generates the ST segment and T wave. Repolarization, similar to activation, occurs in a characteristic geometric pattern. Because the endocardial action potential lasts longer than that of its overlying epicardium, repolarization of the epicardium often starts earlier than in the endocardium. However, under normal conditions, regional differences in electrical recovery properties of the ventricles are present, and the transmural gradients predominantly determine ST-segment patterns.

Normally, concordance exists between the orientation of the QRS complex and the T wave, with both deflecting in the same direction. The junction of the QRS complex and the ST segment is called the J junction. The QT interval—the duration from the Q wave to the end of the T wave—is highly heart rate (HR)–dependent, and formulas have been developed to calculate the corrected QT interval (QTc), which, if prolonged, may be associated with serious ventricular arrhythmias. The T wave is sometimes followed by a small U wave, which may be associated with hypokalemia or hypomagnesemia electrolyte disturbances.

THE ABNORMAL ELECTROCARDIOGRAM

ATRIAL ABNORMALITY

The initiation of atrial electrical activation from a site other than the SA node occurs in one of two ways: (1) an escape rhythm if the normal SA nodal pacemaker slows or fails, or (2) an accelerated atrial ectopic rhythm if the automaticity of the ectopic site is associated with a rate higher than the SA. An abnormal morphologic appearance of the P wave (different from the native P) often expresses ectopic atrial activation. Most commonly, negative P waves are observed in the leads where the
P wave is normally upright (leads I, II, aV₅, and V₆ through V₆), with or without a shortening of the PR interval. The exact site of an ectopic atrial pacemaker is usually of little clinical significance except that left-sided atrial ectopic rhythm is more often associated with LV or left heart valvular abnormalities, whereas right-sided ectopic rhythm is more common in patients with chronic obstructive lung disease or other causes of right heart dysfunction.

VENTRICULAR HYPERTROPHY AND ENLARGEMENT

Left ventricular hypertrophy (LVH) or enlargement produces changes throughout the QRST complex. The most characteristic is increased voltage of the QRS complex: tall R waves in left-sided leads (I, aV₅, V₅, and V₆) and deep S waves in right-sided leads (V₁ and V₂). ST-segment and T-wave amplitudes can be normal or increased. ST-segment depression with downsloping from a depressed J point and inverted asymmetric T waves are common in long-standing and severe LVH. Similarly, prolonged QRS duration beyond 110 msec reflects the longer duration of activation of the thickened ventricular wall (Fig. 47-3). Diagnostic ECG criteria for LVH have been developed, although their importance in the era of echocardiography has diminished. For example, Sokolow and Lyon found the following voltage criteria to correlate best with postmortem pathologic findings of LVH: S₅₁ + (R₅₅ / R₅₂) >3.5 mV and/or R₅₁ >1.1 mV.

Right ventricular hypertrophy (RVH) is signified on the ECG by abnormally tall R waves in the rightward-directed leads (aV₆, V₁, and V₂), reversal of normal R-wave progression in the precordial leads, deep S waves with abnormally small R waves in left-sided leads (I, aV₆, V₅, and V₆) and obvious right axis deviation (>110 degrees) (Fig. 47-4). Chronic obstructive pulmonary disease can lead to RVH, changes in the position of the heart in the chest, and hyperinflation of the lungs. Acute right ventricular pressure overload, such as that caused by pulmonary embolism, can produce a characteristic ECG pattern: a QR or qR pattern in the right-sided leads, an S₁Q₃T₃ pattern, and an acute incomplete or complete right bundle branch block (RBBB). However, even the classic S₁Q₃T₃ pattern occurs in only approximately 10% of patients with acute pulmonary embolism.

MYOCARDIAL ISCHEMIA

The ST-T segment, representing myocardial repolarization, is the ECG component most sensitive to acute myocardial ischemia. ST elevation, with or without tall positive (hyperacute) T waves, indicates transmural ischemia and is most often the result of acute coronary artery occlusion either by coronary thrombosis or vasospasm (Prinzmetal-variant angina). Reciprocal ST-segment depression may appear in the contralateral leads. Ischemia confined to the subendocardial area is usually denoted by ST-segment depression. Subendocardial, ST-depression-type ischemia typically occurs during episodes of symptomatic or asymptomatic (silent) stable angina pectoris, which is characteristic of ischemia occurring during exercise, tachycardia, or pharmacologic stress test in patients with significant but stable coronary artery disease (CAD) (Fig. 47-5).

MYOCARDIAL INFARCTION

With prolonged ischemia, the risk of developing myocardial necrosis or myocardial infarction (MI) is present. The MI signature on an ECG is decreased R-wave...
amplitude and pathologic Q waves, which may develop as a result of a loss of electromotive forces in the area of infarction. Transmural infarctions are more likely to culminate in abnormal Q waves, whereas subendocardial (nontransmural) infarctions are less likely to produce Q waves. However, pathologic studies have shown a wide overlap between the two entities and their ECG expression; therefore, Q-wave or non–Q-wave infarction is not synonymous with transmural or nontransmural infarction. Pathologic Q waves usually develop days after the onset of acute MI; once they develop, they seldom disappear and serve as an indicator for the location of the infarction. Persistent T-wave inversions may also be the only sign of chronic ischemia and a recent or an old MI. Pathologic Q waves, with ST-segment elevation that persists weeks or longer after an MI, strongly correlate with a severe myocardial mechanical dysfunction, akinesis, or ventricular aneurysm.

The ECG leads with ST-T changes or Q waves may help define the location and the coronary artery responsible for the ischemia or infarction. For example, precordial leads V₁ to V₃ correspond to the anteroseptal or apical walls of the left ventricle; leads V₄ to V₆ to the apical or lateral LV walls (Fig. 47-6); leads II, III, and aVF to the inferior LV wall (Fig. 47-7); and the right-sided leads to the right ventricle. Posterior wall infarction induces ST-segment elevation or Q waves in leads placed over the left side and back (V₇ to V₉), and reciprocal ST-segment depression or tall R waves in leads V₁ to V₃ may develop.

**ELECTROLYTE ABNORMALITIES**

The ECG is affected not only by structural or functional myocardial abnormalities but also by numerous metabolic and electrolyte aberrations.
**Calcium.** Hypercalcemia shortens and hypocalcemia prolongs the phase 2 of the action potential duration, thus leading to an abbreviation or a prolongation of the QT interval, respectively. Severe hypercalcemia (e.g., total serum Ca²⁺ >15 mg/dL) causes a decrease in T-wave amplitude or T-wave inversion, and hypercalcemia may produce a high-takeoff ST segment in leads V₁ and V₂, simulating acute ischemia.

**Potassium.** Hyperkalemia leads to a distinctive sequence of ECG changes, starting with a narrowing and peaking of the T wave and a shortening of QT interval. Progressive hyperkalemia causes QRS widening, low P-wave amplitude, and PR interval prolongation with the possibility of second- to third-degree AV block (Fig. 47-8). Severe hyperkalemia leads to a sine-wave ventricular flutter and eventual asystole. Hypokalemia, in contrast, may cause ST-segment depression, flattened T waves, and prominent U waves, which may sometimes exceed the amplitude of T waves. Hypokalemia prolongs repolarization and leads to long QT(U) syndrome, predisposing to a torsades de pointes–type ventricular fibrillation.

**Magnesium.** Mild-to-moderate hypermagnesemia or hypomagnesemia is not associated with specific ECG changes. Yet, severe hypermagnesemia can cause AV and intraventricular conduction disturbances, including complete heart block and cardiac arrest (Mg²⁺ >15 mEq/L). Hypomagnesemia is often associated with hypocalcemia or hypokalemia and may predispose to long QT(U) syndrome and torsades de pointes.

**ELECTROCARDIOGRAPHIC MONITORING SYSTEMS**

**NOISE FILTERING**

**Low-Frequency Filtering**

All ECG monitors use filters to narrow the signal bandwidth in an attempt to reduce environmental artifacts and to improve signal quality. Low-frequency noise, such as that produced by respiration or any other patient motion, causes the tracing to wander above and below the
According to current AHA recommendations, baseline frequencies tend to vary among manufacturers. One manufacturer allows a choice of three different filtering modes: (1) a diagnostic mode with a bandwidth of 0.05 to 130 Hz for adults and 0.5 to 130 Hz for neonates, (2) a monitoring mode with a bandwidth of 0.5 to 40 Hz for adults and 0.5 to 60 Hz for neonates, and (3) a filter mode with a bandwidth of 0.5 to 20 Hz. Slogoff and associates evaluated the importance of bandwidth selection on the detection of perioperative myocardial ischemia and showed that the ST-segment positions with all systems using the lower filter limit (0.05 Hz) recommended by the AHA were similar.²

**Figure 47-8.** Electrocardiographic signs of hyperkalemia. The earliest change with hyperkalemia is peaking (“tenting”) of the T waves. With progressive increases in the serum potassium concentration, the QRS complexes widen, the P waves decrease in amplitude and may disappear, and finally a sine-wave pattern leads to asystole unless emergency therapy is administered. (From Goldberger AL: Clinical electrocardiography: a simplified approach, ed 7. St. Louis, 2006, Mosby.)

**High-Frequency Filtering**

High-frequency filters are needed to reduce distortions from muscle fasciculations, tremors, and electromagnetic interference from other electrical equipment. Older monitors used a 40-Hz filter to reduce electrical current interactions. However, the higher the frequencies contained in the filtered signal, the more accurate the measurement of rapid upstroke velocity, peak amplitude, and waves of short duration will be. A high-frequency cutoff of 100 Hz was considered adequate by the AHA in 1975 to maintain diagnostic accuracy during visual inspection of an ECG, although it has long been recognized that higher frequency components of the QRS complex may have clinical significance in patients with various forms of heart disease. According to current AHA recommendations, to measure routine duration and amplitudes accurately in adults, adolescents, and children, an upper frequency cutoff of at least 150 Hz is required, and an upper frequency cutoff of 250 Hz is more appropriate for infants.

Most modern ECG monitors allow the operator a choice among several bandwidths. The actual filter frequencies tend to vary among manufacturers. One manufacturer allows a choice of three different filtering modes: (1) a diagnostic mode with a bandwidth of 0.05 to 130 Hz for adults and 0.5 to 130 Hz for neonates, (2) a monitoring mode with a bandwidth of 0.5 to 40 Hz for adults and 0.5 to 60 Hz for neonates, and (3) a filter mode with a bandwidth of 0.5 to 20 Hz. Slogoff and associates evaluated the importance of bandwidth selection on the detection of perioperative myocardial ischemia and showed that the ST-segment positions with all systems using the lower filter limit (0.05 Hz) recommended by the AHA were similar.²

**THREE-ELECTRODE ELECTROCARDIOGRAPHIC MONITORING**

In contrast to the standard 12-lead ECG, in which the four limb electrodes are placed on the wrists and ankles, electrode placement for continuous cardiac monitoring is on the torso to reduce artifacts from limb movement, as well as to avoid tethering the patient. Therefore, the right arm (RA) and left arm (LA) electrodes are placed in the infraclavicular fossae close to the right and left shoulders, respectively, and the left leg (LL) electrode is placed below the rib cage on the left side of the abdomen. The ground or reference electrode (RL), if present, can be placed anywhere, but it is usually placed on the right side of the abdomen.

The three-electrode system is the simplest and most common mode of ECG monitoring in surgical units and intensive care units (ICUs). It allows monitoring three bipolar leads by recording the potential differences between each of three pairs of electrodes: lead I (positive electrode, LA; negative electrode, RA), lead II (positive electrode, LL; negative electrode, RA), and lead III (positive electrode, LL; negative electrode, LA), or other

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Figure 47-8: Electrocardiographic signs of hyperkalemia. The earliest change is peaking (“tenting”) of the T waves. With progressive increases in the serum potassium concentration, the QRS complexes widen, the P waves decrease in amplitude and may disappear, and finally a sine-wave pattern leads to asystole unless emergency therapy is administered. (From Goldberger AL: Clinical electrocardiography: a simplified approach, ed 7. St. Louis, 2006, Mosby.)
modified chest leads. Three-electrode monitoring is usually adequate for tracking HR, detecting R waves for synchronized direct-current shock in cardioversion, and detecting ventricular fibrillation. However, it is inadequate for diagnosing more complex arrhythmias for which a true V lead is necessary, such as to distinguish between right versus left bundle branch block (LBBB) or ventricular tachycardia (VT) and supraventricular tachycardia (SVT) with aberrant ventricular conduction. The three-electrode system is also inadequate for ST-segment monitoring because it does not provide multilead monitoring or precordial leads, which are often most sensitive for detecting ischemia. Modified chest leads such as CS (the RA electrode is placed under the right clavicle, and the LA electrode is placed in the V position) or CB (the RA electrode is placed over the center of the right scapula, and the LA electrode is placed in the V position) may be suitable for the detection of anterior wall myocardial ischemia; however, these modified leads are currently not recommended for monitoring myocardial ischemia.

**FIVE-ELECTRODE ELECTROCARDIOGRAPHIC MONITORING**

In the five-electrode monitoring system, the four limb electrodes, LA, RA, LL, and RL placed at their corresponding monitoring locations allow any of the six limb leads (I, II, III, aVR, aVL, and aVF) to be obtained, and a fifth chest electrode can be placed in any of the standard V locations (Fig. 47-9). V is the preferred lead for special arrhythmia monitoring, whereas the other precordial leads, especially V to V are the preferred leads for ischemia monitoring. The five-electrode monitoring system is currently the standard for monitoring patients with suspected perioperative myocardial ischemia. The differences in sensitivity and specificity among the different ECG leads in detecting myocardial ischemia are discussed later in this chapter.

**TEN-ELECTRODE, TWELVE-LEAD ELECTROCARDIOGRAPHIC MONITORING**

In 1966, Mason and Likar introduced a variation on positioning the standard limb electrodes of the 12-lead ECG during exercise stress testing to minimize artifacts in the limb leads caused by movement. In this design, the RA and LA electrodes are attached to the right and left infraclavicular fossae and the LL electrode is attached to the left iliac fossa. The RL electrode can be positioned anywhere but is usually placed on the right iliac fossa for symmetry. Although the limb lead QRS complexes are slightly different in amplitude and axis and the precordial leads may also vary slightly from the standard 12-lead ECG recording, studies have shown that the ST-segment measurements during exercise stress testing are only incidentally affected when the Mason-Likar 12-lead ECG system is used, as compared with the standard 12-lead ECG. A major advantage of cardiac monitors using the Mason-Likar 12-lead system is that ST-segment monitoring software has been developed to analyze all 12 leads and to sound an alarm for ST-segment changes, whether or not multiple leads are being displayed on the bedside or central monitor (Fig. 47-10). Therefore, if lead II is being displayed but the patient has a transient ischemic event involving lead V, then an ST-segment alarm is triggered.

**HOLTER MONITORING**

Holter monitoring, originally a cardiologist's tool, has been used by a number of anesthesiologists to document the perioperative incidence of arrhythmias or ischemia. In Holter monitoring, ECG information from two or three bipolar leads is recorded by a miniature recorder. Up to...
48 hours of ECG signals can be collected. Subsequently, the data are processed using a playback system, and the ECG signals are analyzed. On most modern systems, the playback unit includes a dedicated computer for rapid analysis and automatic recognition of arrhythmias and ischemia.

A significant early obstacle to the widespread use of conventional Holter monitoring in the perioperative period was its delayed, retrospective analysis and interpretation. This limitation was, to some extent, overcome by real-time Holter monitors, which record specific ECG segments for later playback and also analyze the rhythm and ST segment in real time to alert the user to an acute event.6,7 However, despite significant technical progress, Holter monitoring devices continue to be limited primarily to clinical investigations.

**ARRHYTHMIA INTERPRETATION AND MANAGEMENT**

Little doubt remains that during prolonged visual ECG monitoring, certain arrhythmias may go undetected. It has been demonstrated that coronary care unit nurses failed to detect serious ventricular arrhythmias in 84% of their patients.8 Subsequently, computerized monitors have been designed for the automatic detection of arrhythmias in an attempt to increase the detection rate of abnormal, potentially dangerous rhythms. These monitors use proprietary sophisticated algorithms such as pattern recognition, measurements of QRS width, onset, offset, amplitude, and area calculations to classify complexes into morphologic families.9,10 Most monitors are capable of detecting potentially fatal arrhythmias such as severe bradycardia or asystole and dangerous tachycardia such as VT or fibrillation. Yet, no study to date has evaluated the accuracy, sensitivity, and specificity of these automated, real-time arrhythmia detection monitors. However, the detection of clinically important dysrhythmias in the cardiac ICU has been shown to improve significantly if a nurse watches the monitor and pays attention to the alarms.11 Thus today’s computerized monitoring systems still have not achieved the level of accuracy sufficient to eliminate the need for human surveillance; the alarms must be recognized and the ECG interpreted and acted upon by a knowledgeable person in a timely fashion (see also Chapters 67 and 68).

**DIAGNOSIS OF ARRHYTHMIAS**

Arrhythmias are common during and after surgery and have numerous causes. Postoperative dysrhythmias are most likely to occur in patients with structural heart disease. The initiating factor for an arrhythmia after surgery is usually a transient insult such as hypoxemia, cardiac ischemia, catecholamine excess, or electrolyte abnormality.12 Using perioperative Holter recordings, Mahla and colleagues13 evaluated how anesthesia and surgery affect the course of ventricular dysrhythmias (premature ventricular beats and repetitive forms of ventricular beats—couplets and nonsustained VT) preoperatively noted in patients with structural heart disease undergoing...
noncardiac surgery. They concluded that the frequency of ventricular dysrhythmias was not associated with an adverse cardiac outcome. The incidence of perioperative dysrhythmia in patients with an adverse outcome (8%) did not differ from those with a good outcome. Patients undergoing cardiac surgery have a higher incidence of cardiac dysrhythmias. The incidence of new onset atrial fibrillation alone after cardiac surgery approaches 33% and is associated with a worse outcome. Several major factors contribute to the development of perioperative arrhythmias:

1. **General anesthetics.** Volatile anesthetics, such as halothane or enflurane, produce arrhythmias, probably by a reentrant mechanism. Halothane also sensitizes the myocardium to endogenous and exogenous catecholamines. Drugs that block the reuptake of norepinephrine, such as cocaine and ketamine, can facilitate the development of epinephrine-induced arrhythmias (see Chapter 28). In contrast, volatile anesthetics may have an antiarrhythmic effect in response to acute coronary occlusion and reperfusion, at least in a canine model. Sevoflurane may cause severe bradycardia and nodal rhythm when used in high concentrations during induction in infants, and desflurane may prolong QTc within the first minute of anesthesia in patients with a normal heart.

2. **Local anesthetics.** Regional anesthesia by central neuraxial blockade, the goal of spinal or epidural anesthesia, may be associated with a profound, albeit transient, pharmacologic sympathectomy (also see Chapters 36 and 56). This phenomenon may cause parasympathetic nervous system dominance, leading to mild to very severe bradycardhythmias. This result is especially true when the blockade extends to very high thoracic levels. An inadvertent intravascular injection of a large dose of local anesthetic agent may lead to asystole and cardiac arrest that are difficult to treat. One proposed treatment is the administration of 20% intralipid.

3. **Abnormal arterial blood gases or electrolyte levels.** Excessive hyperventilation, especially in the presence of low serum potassium levels, may precipitate severe cardiac arrhythmias. Alterations of blood gases or electrolytes may lead to arrhythmias by producing reentrant mechanisms or by altering phase 4 depolarization of conduction fibers. Electrolyte disturbances associated with cardiopulmonary bypass can also lead to intraoperative arrhythmias (see Chapters 59 and 67).

4. **Endotracheal intubation.** This maneuver may be the most common cause of arrhythmias during surgery and is often associated with hemodynamic disturbances by eliciting autonomic reflexes (see also Chapter 55).

5. **Autonomic reflexes.** Vagal stimulation may produce sinus bradycardia and may allow ventricular escape mechanisms to occur. Vagal stimulation may also produce AV block or even asystole. These reflexes may be related to traction on the peritoneum or to direct pressure on the vagus nerve during carotid surgery (see Chapter 69). During jugular vein cannulation, stimulation of the carotid sinus by palpation of the neck can lead to bradyarrhythmias. Specific reflexes, such as the oculocardiac reflex, can also produce severe bradycardia or asystole.

6. **Central nervous system stimulation and dysfunction of the autonomic nervous system.** Many ECG abnormalities can occur in patients with intracranial disease, especially subarachnoid hemorrhage. These abnormalities are most commonly ST-T wave changes and may easily mimic myocardial ischemia and MI (see Chapter 70). The mechanism of these arrhythmias appears to be related to changes in autonomic nervous system tone.

7. **Preexisting cardiac disease.** Preexisting cardiac disease is probably the most common background for arrhythmias during anesthesia and surgery. Patients with a preexisting tendency for atrial or ventricular arrhythmias are more likely to exhibit them during or after surgery in response to the perioperative stresses or secondary to acute withdrawal of oral antiarrhythmic medications, most commonly β-adrenergic blockers.

8. **Central venous cannulation.** The insertion of catheters or wires into the central circulation often leads to arrhythmias (see Chapter 45).

9. **Surgical manipulation of the cardiac structures.** Arrhythmias are often observed during the insertion of atrial sutures or the placement of venous cannulae for cardiopulmonary bypass during cardiac surgery (see Chapters 67, 68, and 94). These arrhythmias are usually self-limiting and cease at the end of manipulation.

10. **Location of surgery.** Dental surgery is often associated with arrhythmias because profound stimulation of sympathetic and parasympathetic nervous systems often occurs. Junctional rhythms are often observed and may be caused by stimulation of the autonomic nervous system by the fifth cranial nerve. The oculocardiac reflex leads to severe bradycardia in response to traction of the rectus muscles of the orbit. This reflex, which is mediated by the trigeminal nerve as the afferent limb and the vagus nerve as the efferent limb, is especially sensitive in neonates and children and common during strabismus operations.

After an arrhythmia is recognized, determining whether it produces a hemodynamic disturbance, what type of treatment is required, and how urgently therapy should be instituted are important. Treatment should be promptly initiated if the arrhythmia results in significant hemodynamic impairment. Prompt treatment should also be instituted if the arrhythmia is a precursor for a more severe arrhythmia (e.g., frequent multifocal ventricular premature beats [VPBs] with R-on-T phenomenon can lead to ventricular fibrillation). In addition, arrhythmias that may be detrimental on the background of a patient’s underlying cardiac disease (e.g., any tachycardia in a patient with mitral valve stenosis, aortic valve stenosis, or ischemic heart disease) need immediate attention. Arrhythmias can be classified by HR or by their anatomic origin in the heart. Using HR criteria, arrhythmias can be broken down into three categories: bradyarrhythmias (HR <60 bpm), tachyarrhythmias (HR >100 bpm), and conduction blocks (at any HR). The anatomic origin of an arrhythmia can be ventricular, supraventricular,
junctional, or elsewhere. Using the following checklist can simplify the diagnosis and treatment of arrhythmias when looking at an ECG display:

1. What is the HR?
2. Is the rhythm regular?
3. Is one P wave present for each QRS complex?
4. Is the QRS complex normal?
5. Is the rhythm dangerous?
6. Does the rhythm require treatment?

The following text analyzes some common intraoperative arrhythmias.

**SINUS BRADYCARDIA**

Sinus bradycardia is defined when the pacemaker site is in the sinus node, but the rate is slower than normal. Etiologic factors include drug effects, acute inferior MI, hypoxia, vagal stimulation, and high sympathetic blockade. Sinus bradycardia accounts for approximately 11% of intraoperative arrhythmias. The characteristics of sinus bradycardia are as follows:

1. HR: Is slower than 60 bpm. In patients on chronic β-adrenergic blocker therapy, sinus bradycardia is defined as a HR slower than 50 bpm.
2. Rhythm: Is regular, except for occasional escape beats from other pacemaker sites.
3. P/QRS: The ratio of the P waves to the QRS complexes is 1:1.
4. QRS complex: Has a normal morphologic appearance.
5. Significance: HRs slower than 40 bpm are poorly tolerated even in healthy patients and should be evaluated on the basis of their effect on cardiac output. Treatment is recommended if hypotension, ventricular arrhythmias, or signs of poor peripheral perfusion are observed. Sinus bradycardia may be part of the sick sinus syndrome, in which sinus node dysfunction can precipitate bradycardias, heart block, tachyarrhythmias, or alternating bradyarrhythmias and tachyarrhythmias.
6. Treatment: Is not usually necessary. When treatment is deemed necessary, the following progression may be considered: (1) atropine, 0.5 to 1.0 mg by intravenous (IV) bolus, repeated every 3 to 5 minutes, up to 0.04 mg/kg or approximately a 3.0-mg total dose for the average 75-kg male patient; (2) ephedrine, 5 to 25 mg by IV bolus; (3) dopamine or dobutamine (if blood pressure is adequate), 5 to 20 μg/kg/min by IV infusion; (4) epinephrine, 2 to 10 μg/min IV infusion; and (5) isoproterenol, 2 to 10 μg/min by IV infusion. Temporary transcutaneous or transvenous pacing may be necessary for severe, drug-refractory sinus bradycardia. Immediate institution of transcutaneous pacing is especially important in symptomatic patients.

**SINUS TACHYCARDIA**

Sinus tachycardia is defined when the pacemaker site is in the sinus node, and the rate is faster than normal. Sinus tachycardia is the most common arrhythmia in the perioperative period. It occurs with such frequency that it is not included in most arrhythmia incidence studies. Common causes include pain, inadequate anesthesia, hypovolemia, fever, hypoxia, hypercarbia, heart failure, and drug effects. The characteristics of sinus tachycardia are as follows:

1. HR: Is faster than 100 bpm in the adult patient and may be as high as 170 bpm. Patients with significant CAD may not tolerate HRs as low as 70 to 80 bpm and may develop subendocardial ischemia. Similarly, patients with severe mitral or aortic stenosis may be sensitive to even moderate increases in the HR.
2. Rhythm: Is regular.
3. P/QRS: The ratio of the P waves to the QRS complexes is 1:1.
4. QRS complex: Is normal, but ST-segment depression with severe increases in HR and resulting myocardial ischemia may be associated.
5. Significance: Prolonged tachycardias in patients with underlying heart disease can precipitate MI and congestive heart failure because of the increased myocardial work required and the decreased supply of myocardial oxygen by means of decreased diastolic coronary perfusion time. A major diagnostic problem is encountered when the HR is 150 bpm, because this rate is common for sinus tachycardia, paroxysmal atrial tachycardia, or atrial flutter with a 2:1 block. These three arrhythmias can sometimes be separated by the use of carotid sinus massage, IV administration of edrophonium, or adenosine phosphate.
6. Treatment: The underlying disorder should be treated. Hypovolemia and light anesthesia are the most common causes. In patients with ischemic heart disease who develop tachycardia, β-adrenergic blockers should be judiciously used to prevent myocardial ischemia, regardless of whether ST-segment changes occur. Hypovolemia or other causes should also be addressed in these patients.

**SINUS ARRHYTHMIA**

In sinus arrhythmia, the impulses arise from the SA node and a variable HR characterizes the rhythm. The PR interval is normal, as is the QRS complex. Most commonly, but not invariably, the rate increases with inspiration and decreases with expiration. This arrhythmia occurs more often in children than in adults. The characteristics of sinus arrhythmia are as follows:

1. HR: Is 60 to 100 bpm.
2. Rhythm: Is irregular.
3. P/QRS: The ratio of the P waves to the QRS complexes is 1:1.
4. QRS complex: Has normal morphologic appearance.
5. Significance: Has little clinical significance.

**ATRIAL PREMATURE BEATS**

An ectopic pacemaker site in the left or the right atrium initiates the atrial premature beat (APB). The shape of the P wave is different from the usual SA node P wave and may be inverted. The PR interval may be shorter or longer than normal, depending on the site of the ectopic
focus and on the refractoriness of the AV nodal pathway. The APB spreads through the AV node and ventricular conduction system and, in retrograde fashion, reaches the SA node, resetting the sinus pacemaker. The interval from the APB to the next sinus beat is therefore a normal sinus cycle (i.e., no compensatory pause). The absence of a compensatory pause is an important distinguishing feature between APBs and VPBs. Occasionally, APBs may find part of the ventricular conduction system refractory. In these instances, they travel down an aberrant pathway and create an abnormal QRS complex. They are then called APBs with aberrant ventricular conduction and can easily be confused with VPBs. Because the recovery period of the right ventricular conduction system outlasts that of the left, the most common form of aberration appears as an RBBB. Helpful points in separating APBs with aberrant ventricular conduction from VPBs include (1) the presence of a preceding P wave, usually abnormally shaped; (2) an RBBB configuration of the QRS complex; (3) the presence of an rsR' ventricular complex in V1; and (4) the finding that the initial vector forces are identical to the preceding beat but are usually the opposite with a VPB. The characteristics of APBs are as follows:

1. HR: Is variable, depending on the frequency of the APBs.
2. Rhythm: Is irregular.
3. P/QRS: The ratio of P waves to QRS complexes is usually 1:1. The P waves have various shapes and may even be lost in the QRS or T waves. Occasionally, the P wave is so early as to find the ventricle refractory, and a nonconducted beat occurs.
4. QRS complex: Is usually normal unless ventricular aberration occurs.
5. Significance: In one study, APBs represented 10% of all intraoperative arrhythmias. They have little clinical significance, but frequent APBs may lead to other, more serious supraventricular arrhythmias or may be a sign of digitalis intoxication.
6. Treatment: Is rarely necessary.

PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA

A rapid regular rhythm, usually with a narrow QRS complex and lacking the normal SA node P wave, characterizes paroxysmal SVT (PSVT). The inclusion of tachycardias involving the AV node (Fig. 47-11) allows a useful classification of tachycardias as caused by reentry in the AV node, by apparent or concealed accessory AV pathways or, less often, by the SA node. Ectopic atrial or ectopic nodal tachycardias are also among the less frequent SVTs. Inappropriate or persistent sinus tachycardia is another variant. PSVT rhythms are usually abrupt in onset and termination. PSVT is easily distinguished from rapid atrial fibrillation, which is an irregular rhythm, or from rapid atrial flutter, which has flutter waves. The characteristics of PSVT are as follows:

1. HR: Is between 130 and 270 bpm.
2. Rhythm: Is usually regular unless the impulse originates from multiple atrial foci.
3. P/QRS: Has a 1:1 relationship, although the P wave may often be hidden in the QRS complex or T wave.
4. QRS complex: Is generally normal, but ST-T changes indicative of ischemia may be observed. Aberration of ventricular conduction may occur, complicating the differential diagnosis with VT. SVT may also be confused with sinus tachycardia, atrial flutter, and atrial fibrillation. In differentiating these rhythms, carotid sinus massage or edrophonium (5 to 10 mg IV) was traditionally used. More recently, adenosine (6 to 12 mg by IV bolus) has been used to slow the rate by transiently enhancing the normal degree of AV block or terminate the arrhythmia. Esophageal electrocardiographic leads may also be helpful to better define atrial activity.
5. Significance: PSVT can be observed in 5% of normal young adults and in patients with Wolff-Parkinson-White syndrome or other preexcitation syndromes. During anesthesia, PSVT accounts for up to 2.5% of all arrhythmias, and the arrhythmia has been associated with intrinsic heart disease, systemic illness, thyrotoxicosis, digitalis toxicity, pulmonary embolism, and pregnancy. When a patient is under anesthesia, PSVT can be precipitated by changes in the tone of the autonomic nervous system, by drug effects, or by intravascular volume shifts and can produce severe hemodynamic deterioration. Sometimes the PSVT may be associated with AV block because of the fast atrial rate and slow AV conduction. PSVT with 2:1 block represents digitalis intoxication in many patients.
6. Treatment: Often, this arrhythmia must be treated because of its rapid rate and associated poor hemodynamic function. One or several of the following treatments can be undertaken:
   a. Vagal maneuvers such as carotid sinus massage should only be applied to one side.
   b. Adenosine, which is the drug of choice, is given by 6-mg rapid (2 seconds) IV bolus, preferably through an antecubital or central vein. If no response is elicited, the second and third doses of 12 to 18 mg of adenosine may be administered by rapid IV bolus.
   c. Verapamil (2.5 to 10 mg given IV) successfully terminates AV nodal reentry in approximately 90% of patients. This was the first drug of choice but is now the second choice.
   d. Amiodarone (150-mg infusion over 10 minutes for the loading dose) is a recent addition.
   e. Esmolol (1 mg/kg by bolus and 50 to 200 mg/kg/min by infusion) has been shown to be effective.
   f. Edrophonium or neostigmine by IV bolus can be administered.
   g. Phentylephrine (100 μg by IV bolus) is administered if the patient is hypotensive.
   h. IV digitalization is performed with one of the short-acting digitalis preparations: ouabain (0.25 to 0.5 mg administered IV) or digoxin (0.5 to 1.0 mg administered IV, slow loading).
   i. Rapid overdrive pacing may be performed in an effort to capture the ectopic focus.
   j. Synchronized cardioversion may be performed with incremental doses of energy of 100, 200, 300, and 360 J, preferably after light sedative premedication. Electrode catheter ablation using radiofrequency energy has evolved as the definitive, long-term treatment for most persistent AV reentrant or focal atrial SVTs.
Atrial flutter most commonly represents a macro-reentrant arrhythmia that circulates in a specific manner in the right atrium (i.e., counterclockwise rotation as viewed in the angiographic left anterior oblique view). Because atrial flutter is associated with very fast HRs, it is usually accompanied by AV block. Classic sawtooth flutter waves (F waves) are usually present (Fig. 47-12). The characteristics of atrial flutter are as follows:

1. HR: The atrial HR is 250 to 350 bpm with a ventricular rate of approximately 150 bpm (2:1 or 3:1 AV conduction block).
2. Rhythm: The atrial rhythm is regular. The ventricular rhythm may be regular if a fixed AV block is present or irregular if a variable block exists.
3. P/QRS: Usually, a 2:1 block exists with an atrial rate of 300 bpm and a ventricular rate of 150 bpm, but the block may vary between 2:1 and 8:1. F waves are best observed in leads V1 and II and in the esophageal lead.
4. QRS complex: Is normal. T waves are lost in the F waves.
5. Significance: Atrial flutter usually indicates the presence of severe heart disease. It is recognized with increased incidence in patients with CAD, mitral valve disease, pulmonary embolism, hyperthyroidism, cardiac trauma, cancers of the heart, and myocarditis.
6. Treatment: Pharmacologic or synchronized direct current (DC) cardioversion, when indicated, should be performed only after careful consideration or evaluation of a possible thromboembolic event.

The initial treatment should be to gain control of the ventricular response rate with agents that slow AV node conduction:

a. β-Adrenergic blockers such as IV esmolol (1 mg/kg by IV bolus) or propranolol
b. Calcium channel blockers such as verapamil (5 to 10 mg given IV) or diltiazem

β-Adrenergic blockers and calcium channel blockers are also effective as pharmacoprophylaxis.
for the prevention of postoperative atrial tachyarhythmias after thoracic and cardiac surgery.\textsuperscript{40}

If the ventricular response is excessively rapid or secondary hemodynamic instability is present, or both, then the following guidelines are used:

a. Synchronized DC cardioversion is indicated, starting with a relatively high energy of 100 J and gradually increasing to 360 J.

b. The class III antiarrhythmic agent ibutilide (Corvert, 1 mg in 10 mL saline, or 5\% dextrose in water (D\textsubscript{5}W), infused IV slowly over 10 minutes) has been documented to convert atrial flutter to sinus rhythm in most patients with relatively new-onset atrial flutter\textsuperscript{41} and may be repeated once. Although it is highly effective, life-threatening torsade de pointes (discussed later in this chapter) may occur hours after ibutilide administration, making 4- to 8-hour monitoring after treatment highly desirable.

c. Procainamide (5 to 10 mg/kg for the IV loading dose, infused no faster than 0.5 mg/kg/min) may rarely be used in an attempt to restore sinus rhythm after the ventricular response has been adequately controlled.\textsuperscript{42}

ATRIAL FIBRILLATION

Atrial fibrillation is an excessively rapid and irregular atrial focus with no P waves appearing on the ECG; instead, a fine fibrillatory activity is seen (F waves, see Fig. 47-12) and is the most irregular rhythm; it is called \textit{irregularly irregular} and may be associated with a pulse deficit. The characteristics are as follows:

1. HR: The atrial rate is 350 to 500 bpm, and the ventricular rate is 60 to 170 bpm.
2. Rhythm: Is irregularly irregular.
3. P/QRS: The P wave is absent and replaced by F waves or no obvious atrial activity.
4. QRS complex: Is normal.
5. Significance: The causes of atrial fibrillation are similar to those of atrial flutter. This rhythm is often associated with significant cardiac disease; however, idiopathic lone paroxysmal atrial fibrillation has become increasingly recognized. The clinical significance and treatment of atrial fibrillation are also similar to those of atrial flutter, except for two important considerations. The loss of an atrial \textit{kick} from inefficient contraction of the atria may reduce ventricular filling and may significantly compromise cardiac output. After 24 hours, atrial fibrillation may be associated with the development of atrial thrombi, with resultant pulmonary and systemic embolization.

Atrial fibrillation is the most common postoperative arrhythmia with significant consequences on patient health. Postoperative atrial fibrillation complicates up to 8\% of all noncardiac surgeries, between 3\% and 30\% of noncardiac thoracic surgeries, and between 16\% and 46\% of cardiac surgeries. It has been associated with increased morbidity; mortality; and longer, more costly hospital stays. Several epidemiologic and intraoperative factors, as well as the presence of pre-existing cardiovascular and pulmonary disorders, may affect the risk of atrial fibrillation after cardiac and noncardiac surgeries. It is typically a transient, reversible phenomenon that may develop in patients who possess an electrophysiologic substrate for the arrhythmia that is present before or as a result of surgery. The efficacy of β-adrenergic blockers in postoperative atrial fibrillation prevention is well documented. Perioperative amiodarone, sotalol, nondihydropyridine calcium channel blockers, and magnesium sulfate can reduce the occurrence of atrial fibrillation.\textsuperscript{43}

6. Treatment:

a. Acute atrial fibrillation: The treatment of acute atrial fibrillation is similar to that for atrial flutter. More attention should be focused on ventricular response, especially with the administration of IV diltiazem or esmolol. Ibutilide may restore sinus
rhythm, but it is less effective than in the treatment of flutter. Synchronized DC cardioversion should be used in patients with pronounced hemodynamic instability. However, if fibrillation is present for longer than 48 hours, then attempts to restore sinus rhythm may be associated with a heightened risk of thromboembolism. In this setting, for a patient with normal coagulation function, adequate anticoagulation for 3 to 4 weeks should be considered before attempting to restore sinus rhythm. New developments in electrophysiologic techniques for ventricular defibrillation with biphasic current shocks have shown superiority to the conventional monophasic current techniques (discussed later in this chapter). However, data regarding biphasic shocks for conversion of atrial fibrillation are still emerging. A randomized, double-blind, multicenter trial by the BiCard Investigators demonstrated that for the cardioversion of atrial fibrillation, a biphasic shock waveform has greater efficacy, requires fewer shocks and lower delivered energy, and results in less dermal injury than a monophasic shock waveform. These results mirror those observed in the ventricular fibrillation trials.

b. Long-term therapy: Long-term therapy of atrial fibrillation varies and depends on factors such as whether the arrhythmia is constant or paroxysmal, the nature of the underlying heart disease, and the state of ventricular function or hemodynamic stability or reserve. In the older individual or in the setting of specific risk factors (e.g., hypertension, diabetes mellitus, severe LV systolic dysfunction), anticoagulation with warfarin should be strongly considered. When control of ventricular response is difficult with standard drugs (e.g., β-adrenergic blockers, calcium channel blockers, digitalis), electrode catheter ablation of the AV junction and permanent pacemaker insertion are sometimes used. Another treatment option for chronic or recurrent atrial fibrillation is the Maze procedure, often performed during cardiac surgery with cardiopulmonary bypass. During this procedure, a number of incisions or scars are made by means of radiofrequency, freezing, or microwaves on the left and right atrium, resulting in a disruption of the abnormal electrical pathways from the sinus node to the AV node. The scar tissue also prevents erratic electrical signals from recurring. In the absence of CAD or significant LV systolic dysfunction, class Ic antiarrhythmic agents (e.g., flecaïnine, propafenone) have become the agents of choice. The use of class Ia drugs (e.g., quinidine, procainamide, disopyramide) has sharply diminished because of concerns about their significant proarrhythmic function and their systemic and organ side effects. The use of antiarrhythmic drugs that block repolarizing potassium currents (e.g., sotalol, amiodarone) has gained popularity for the suppression of atrial fibrillation in individuals with significant structural heart disease. Sotalol, however, has a much lower efficacy of converting atrial fibrillation than the class Ic agents. New class III agents show good efficacy in converting atrial fibrillation. Ibutilide converts rapidly, can be effective in up to 50% of patients, and is more effective than sotalol or procainamide. Nevertheless, all these drugs also exhibit significant proarrhythmic properties.

JUNCTIONAL RHYTHMS

The AV node itself shows no intrinsic phase 4 depolarization. Cells in the node cannot act as pacemakers. Ectopic activity, however, may be initiated from sites just above and below the AV node. Considering these arrhythmias as AV junctional in nature makes sense. The resultant P wave is abnormal and, depending on the position of the ectopic pacemaker, may be close to, buried in, or after the QRS complex. Depending on the rate of fire of the ectopic pacemaker, the resultant rhythm is nodal premature, nodal quadrigeminy, trigeminy, or bigeminy, nodal rhythm, or nodal tachycardia. The characteristics of junctional rhythms are as follows:

1. HR: Is variable, between 40 and 180 bpm (i.e., nodal bradycardia to junctional tachycardia).
2. Rhythm: Is regular.
3. P/QRS: The ratio is 1:1 but, three varieties have been identified:
   a. High-nodal rhythm: The impulse reaches the atrium before the ventricle; the P wave therefore precedes the QRS but has a shortened PR interval (0.1 second).
   b. Mid-nodal rhythm: The impulse reaches the atrium and the ventricle at the same time. The P wave is lost in the QRS complex.
   c. Low-nodal rhythm: The impulse reaches the ventricle first and then the atrium; therefore, the P wave follows the QRS complex.
4. QRS complex: Is normal, unless altered by the P wave.
5. Significance: Junctional rhythms are common in patients under anesthesia (approximately 20%) especially with volatile anesthetic agents. The junctional rhythms frequently decrease blood pressure and cardiac output by approximately 15%, but they can decrease it up to 30% in patients with heart disease.
6. Treatment: Usually, no treatment is required, and the rhythm spontaneously reverts. If hypotension and poor perfusion are associated with the rhythm, then treatment is indicated. Atropine, ephedrine, or isoproterenol can increase the activity of the SA node and take over as the pacemaker. Dual-chamber electrical pacing at a rate faster than a slow nodal rhythm is another option.

VENTRICULAR PREMATURE BEATS

VPBs result from ectopic pacemaker activity arising below the AV junction. The VPB originates in and spreads through the myocardium or ventricular conducting system, resulting in a wide (>0.12 second) and bizarre QRS complex. The ST segment usually slopes in the direction opposite to that of the main deflection of the QRS complex. No P wave is associated with a VPB, but retrograde depolarization of the atria or blocked sinus beats may obscure the diagnosis.
The most important entity in the differential diagnosis is APB with aberrant ventricular conduction. The distinction should be made whenever possible.

Although an APB normally reaches the SA node and resets the sinus rhythm, such an occurrence is rare when the ectopic pacemaker is in the ventricle. A VPB often blocks the next depolarization from the SA node, but the following sinus beat occurs on time. The result is a compensatory pause, consisting of the interval from the VPB to the expected normal QRS, which is blocked at the AV node, plus a normal sinus interval.

VPBs are common during anesthesia, accounting for 15% of observed arrhythmias. They are significantly more common in anesthetized patients with preexisting cardiac disease. Other than heart disease, known etiologic factors include electrolyte and blood gas abnormalities, drug interactions, brainstem stimulation, and trauma to the heart. The characteristics of VPBs are as follows:

1. HR: Depends on the underlying sinus rate and frequency of the VPBs.
2. Rhythm: Is irregular.
3. P/QRS: No P wave is associated with VPBs.
4. QRS complex: Is wide and bizarre, with a width of more than 0.12 second. If it is of an RBBB nature, prominent R forces are present in V1. If it is a LBBB in appearance, then notching of the S wave and less acute downslopes are common.
5. Significance: The new onset of VPBs must be considered a potentially serious event because, in certain clinical situations, the arrhythmia may progress to VT or ventricular fibrillation. These situations include coronary artery insufficiency, MI, digitalis toxicity with hypokalemia, and hypoxemia. VPBs are more likely to lead to fibrillation if they are multiple, multifocal, or bigeminal; occur near the vulnerable period of the preceding ventricular repolarization (i.e., R-on-T phenomenon); or appear in short-long-short coupling sequences.
6. Treatment: In most patients, VPBs (occurring as single, bigeminy, or trigeminy but excluding nonsustained VT) do not need to be treated, particularly if the patient does not have an acute coronary syndrome (ACS); but the presence of symptoms attributable to VPBs usually dictates the treatment. The first step in treatment is to correct any underlying abnormalities such as decreased serum potassium or low arterial oxygen tension. If the arrhythmia is of hemodynamic significance or if it is believed to be a harbinger of worse arrhythmias, then lidocaine is usually the treatment of choice, with an initial bolus dose of 1.5 mg/kg. Recurrent VPBs can be treated with lidocaine infusion of 1 to 4 mg/min; additional therapy can be supplied with esmolol, propranolol, procainamide, quinidine, disopyramide, atropine, verapamil, or overdrive pacing.

VENTRICULAR TACHYCARDIA

The presence of three or more sequential VPBs defines VT (Fig. 47-13). Diagnostic criteria include the presence of fusion beats, capture beats, and AV dissociation. The specific morphologic appearance of the QRS complex may also be helpful in distinguishing VT from other arrhythmias. VT is classified by its duration and morphologic appearance. In duration, nonsustained VT lasts three beats and up to 30 seconds, and sustained VT lasts 30 seconds or longer. With monomorphic form, all complexes have the same pattern, and with polymorphic form, complexes constantly change patterns. Polymorphic VT with a long QTc is also called torsade de pointes. The characteristics of VT are as follows:

1. HR: Is 100 to 200 bpm.
2. Rhythm: Is usually regular but may be irregular if the VT is paroxysmal.
3. P/QRS: No fixed relationship exists because the VT is a paroxysmal.
4. QRS complex: Is wide, more than 0.12 second in width, with similar morphologic criteria in lead V1 as for VPB.
6. Treatment: If the patient is hemodynamically stable, then amiodarone, administered in one or more doses of 150 mg given IV in 100 mL saline or D5W over 10 minutes, followed by an IV infusion of 1 mg/min for 6 hours and 0.5 mg/min thereafter, is the recommended current treatment (maximum IV dose 2.2 g/24 hr). Although amiodarone produces less hypotension, compared with bretylium, hypotension and bradycardia are its main side effects. Amiodarone’s pharmacologic effects persist for longer than 45 days. Lidocaine and procainamide have been used in the past with varying degrees of success to treat VT. Synchronized cardioversion is the indicated nonpharmacologic intervention in any wide complex tachycardia, whether monomorphic VT or a wide-complex SVT. Polymorphic VT with a normal QT interval is treated with amiodarone and cardioversion. Metabolic abnormalities and drug toxicity must be considered and treated. Polymorphic VT with prolonged QT interval is a more serious rhythm disturbance, and the recommended current treatment is IV infusion of 1 g of magnesium over 2 to 3 minutes. Precipitating metabolic or toxic causes should be treated. Overdrive pacing may also be helpful in this setting.

VENTRICULAR FIBRILLATION

Ventricular fibrillation is an irregular rhythm that results from a rapid discharge of impulses from one or more ventricular foci or from multiple wandering reentrant circuits in the ventricles (Fig. 47-14). The ventricular contractions are erratic and are represented on the ECG by bizarre patterns of various sizes and configurations. P waves are not seen. Important causes of the arrhythmia include myocardial ischemia, hypoxia, hypothermia, electric shock, electrolyte imbalance, and drug effects. The characteristics of ventricular fibrillation are as follows:

1. HR: Is rapid and grossly disorganized.
2. Rhythm: Is totally irregular.
3. P/QRS: No relationship is seen.
4. QRS complex: Is not present.
5. Significance: No effective cardiac output exists, and artificial means, such as external cardiac massage, must be used to sustain life.

6. Treatment: Cardiopulmonary resuscitation must be immediately initiated, and then defibrillation must be performed as rapidly as possible. Asynchronous external defibrillation should be performed with a DC defibrillator, using incremental energies in the range of 200 to 360 J. The introduction of the biphasic (and rectilinear) transthoracic shocks has reduced the energy levels required and increased the efficacy of ventricular defibrillation. In a prospective, randomized, multicenter trial conducted by the ZOLL Investigators, 51,52 120 J of biphasic current was superior to 200 J of monophasic current, especially in patients with increased chest wall impedance.

Early administration of 1 g of magnesium sulfate may facilitate defibrillation. In some instances, epinephrine has been used to coarsen the fibrillation just before and to facilitate defibrillation. Vasopressin has

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**Figure 47-13.** Short bursts of ventricular tachycardia. *(From Goldberger AL: Clinical electrocardiography: a simplified approach, ed 7. St. Louis, 2006, Mosby.)*

**Figure 47-14.** Ventricular fibrillation (VF) in the form of coarse and fine waves. *(From Goldberger AL: Clinical electrocardiography: a simplified approach, ed 7. St. Louis, 2006, Mosby.)*
been added as a drug for the treatment of ventricular fibrillation. The vasopressin dose is a single 40-unit IV bolus. If elected, subsequent administration of epi- nephrine should not be sooner than 5 minutes after vasopressin. Supportive pharmacologic therapy may include lidocaine, amiodarone, bretylium, procainamide, phenytoin, or esmolol.

Torsade de pointes, which may mimic ventricular fibrillation, is a life-threatening arrhythmia that occurs in the presence of disturbed repolarization; hence, its association with prolonged QT interval. Discontinuation of drugs that predispose to QT-interval prolongation and correction of electrolyte abnormalities are essential in the treatment of torsades de pointes. Acute therapy may include defibrillation, 1 to 2 g of IV magnesium sulfate, IV amiodarone, IV isoproterenol, and overdrive pacing.

CONDUCTION DEFECTS

Conduction defects are most often chronic, are present on the patient’s preoperative ECG, and represent the patient’s underlying disease state of the myocardium or conduction system. However, conduction defects may be observed for the first time during surgery and anesthesia. They can occur as a result of simple manipulation, such as the passage of a pulmonary artery catheter through the right ventricle, but they can also be a manifestation of myocardial ischemia. Because high-grade conduction defects (i.e., second- and third-degree AV blocks) often have deleterious effects on hemodynamic performance, their intraoperative recognition is important.

Three types of conduction system blocks are possible: SA block, AV heart block, and intraventricular conduction block. The bundle of His ECG used by cardiologists has greatly improved the understanding of conduction through the heart. In SA block, the block occurs at the sinus node. Because atrial excitation is not initiated, P waves are not found on the ECG. The next beat can be a normal sinus beat, a nodal escape beat, or a ventricular escape beat.

The second type of heart block is an AV heart block, or AV block, which may be incomplete or complete. First- and second-degree AV blocks are usually considered incomplete, whereas a third-degree AV block is considered to be complete heart block. First-degree AV block is often found in healthy hearts, in addition to CAD or the administration of digitalis. It is characterized by a PR interval longer than 0.21 second. All atrial impulses progress through the AV node to the Purkinje system. This form of heart block ordinarily requires no treatment. Second-degree AV block is associated with the conduction of some of the atrial impulses to the AV node and into the Purkinje system. It is further subdivided into two specific types. Mobitz type I block, or Wenckebach block, is characterized by progressive lengthening of the PR interval until an impulse is not conducted and the beat is dropped (Fig. 47-15). This form of block is relatively benign and often reversible, and it does not require a pacemaker. It may be caused by digitalis toxicity or MI and is usually transient. Mobitz type I block reflects disease of the AV node.

The other form of second-degree heart block is Mobitz type II block, which may reflect disease of the bundle of His and Purkinje tissues, especially when the QRS complex is broad. In this type, the less common and more serious form of second-degree heart block, dropped beats occur without any progressive lengthening of the PR interval (Fig. 47-16). This type of block has a serious prognosis because it frequently progresses to complete heart block and may require pacemaker insertion before major surgical procedures.

Third-degree AV block, also called complete heart block, occurs when all electrical activity from the atria fails to progress into the Purkinje system. The atrial and ventricular contractions have no relationship with each other,
although each chamber regularly contracts. The ventricular rate is approximately 40 bpm. The QRS complex may be normal if the pacemaker site is in the AV node, but it is usually widened to longer than 0.12 second when the pacemaker site is located in the ventricle (Fig. 47-17). The HR is usually too slow to maintain adequate cardiac output, and syncope or Adams-Stokes syndrome may occur, as well as heart failure. These patients usually require insertion of a transvenous endocardial or epicardial pacemaker to increase their HR and cardiac output.

**Intraventricular Blocks**

Under normal conditions, activation of the left ventricle spreads simultaneously through both left anterior and left posterior fascicles. Blockage or even a modest delay in conduction in one of these fascicles results in a sequential rather than the simultaneous activation of the corresponding sites, producing characteristic abnormal ECG patterns (Fig. 47-18).

**Left Anterior Fascicular Block.** The left bundle branch of the bundle of His itself bifurcates distally into two fascicles, the anterior and the posterior. A left anterior fascicular block (LAFB) is relatively common as a result of the delicate structure of the left fascicle. It causes delayed activation of the anterosuperior LV wall and is characterized by pronounced (−45° to −90°) left axis deviation. LAFB may occur in persons without overt cardiac disease but is mainly found in patients with a wide range of myocardial and conduction system diseases, such as CAD or LVH. It has minimal and no independent prognostic significance.

**Left Posterior Fascicular Block.** A left posterior fascicular block (LPFB) is considerably less common than the

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**Third-degree (complete) AV block**

![Complete heart block with underlying sinus rhythm.](image)

**Figure 47-17.** Complete heart block with underlying sinus rhythm is characterized by independent atrial (P) and ventricular (QRS complex) activity. The atrial rate is almost always faster than the ventricular rate. The PR intervals are completely variable. (From Goldberger AL: Clinical electrocardiography: a simplified approach, ed 7. St. Louis, 2006, Mosby.)

![Diagrammatic representation of fascicular blocks in the left ventricle. Interruption of the left anterior fascicle (LAF) results in an initial inferior (1) followed by a dominant superior (2) direction of activation (negative in II and positive in I and aV L). Interruption of the left posterior fascicle (LPF) results in an initial inferior (1) followed by a dominant inferior (2) direction of activation (negative in I and positive in II and III). AVN, Atrioventricular node; HB, His bundle; LB, left bundle; RB, right bundle. (Courtesy of Fisch C from Zipes DP, Libby P, Bonow R, Braunwald E: Braunwald’s heart disease: a textbook of cardiovascular medicine, ed 7. Philadelphia, 2005, Saunders.)**

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anterior fascicular block because of the thicker structure of the right fascicle. It is also at a less vulnerable location near the LV inflow tract. LPFB results in delayed activation of the inferoposterior aspect of the left ventricle, and the ECG features of LPFB are pronounced right axis deviation (>120 degrees). LPFB can occur in patients with almost any cardiac disease but is unusual in otherwise healthy people. As in the case of LAFB, the overall QRS duration remains normal (<120 msec).

**Left Bundle Branch Block.** LBBB is caused by a serious delay or block of the main left bundle branch or in both of its two fascicles. This block results in a prolonged QRS duration, abnormal QRS complex, and ST-T wave abnormalities. A basic requirement is QRS duration ≥120 msec (Fig. 47-19). A broad, sometimes notched R wave is evident in the left-sided leads (I, aV1, V5, and V6) with deep S waves in the right precordial leads and absent septal Q waves. The QRS axis is highly variable. It can be normal or deviated to the left or even to the right. The ST-T waves are usually discordant with the direction of the QRS complex. LBBB is an ominous prognostic sign and has associated survival rates as low as 50%, probably reflecting the severity of the underlying cardiac disease. The QRS duration inversely correlates with LV ejection fraction and is one
A conduction delay anywhere in the right-sided intraventricular conduction system causes an RBBB. The high prevalence of RBBB corresponds to the relative fragility of the right bundle branch, as suggested by the development of RBBB after minor trauma produced by right ventricular catheterization. The ECG manifestation of RBBB is prominent, and notched R waves with rsr’, rsR’, or rSR’ on the right-sided leads and wide S waves on left-sided leads, along with QRS prolongation (≥120 msec), are evident. If the QRS duration is not prolonged, then this block is termed incomplete RBBB. As with LBBB, the ST-T waves are discordant with the QRS complex (Fig. 47-20).

RBBB is common in the general healthy population without clinical evidence of structural heart disease and has no prognostic significance in this group. However, in patients with organic heart disease, the new onset of RBBB predicts a higher rate of CAD, congestive heart failure, and mortality.

Trifascicular blocks usually consist of one of the foregoing bilateral bundle branch blocks (i.e., RBBB plus a LAFB or LPFB) in addition to a prolonged PR interval. Bundle of His ECGs are necessary to determine whether the AV conduction disturbance is localized in the AV node or whether it is distal, possibly representing an incomplete fascicular block in the last remaining fascicle.

### REAL-TIME MYOCARDIAL ISCHEMIA MONITORING

Real-time ST-segment analysis first appeared in cardiac monitoring in the mid-1980s, and currently this feature is the standard in most ECG monitors. On some monitors, the ST-segment analysis is set up to turn on automatically when a five-electrode system is used. Unfortunately, however, anesthesiologists mainly use the five-electrode monitoring system with automatic ST-segment analysis in the surgical unit during surgery, but ST-segment monitoring is widely underused postoperatively in the postanesthesia care unit or ICU. A recent study has shown that even among coronary care units, less than 50% routinely use ST-segment monitoring for the detection of myocardial ischemia in patients admitted with ACSs. Chief among the reasons for the underuse of ST-segment analysis are the frequent number of false alarms, the lack of education on how to use the technology, and the lack of knowledge of what to do in response to ST-segment alarms. In addition, no evidence exists as to whether the addition of computerized ST-segment ischemia monitoring improves patient outcomes in patients with ACSs or in patients after surgery.

Technically, the algorithm for ST-segment analysis is relatively simple. The computer determines in each ECG lead the voltage of the ST segment at 60 or 80 msec after the J point (termed as J+60 or J+80 msec) and compares it with the isoelectric point normally measured during the PR interval. One millimeter of ST-segment deviation is equivalent to a 100-mV difference. The changes in ST-segment level over time in each lead are displayed as ST-segment trends. Consequently, to overcome problems of noise and artifacts, most monitors use filters to exclude erroneous or abnormal ECG beats. Averaging processes are then used to calculate the mean ST-segment level over a period of 8 to 15 beats. Current monitors are trained to detect the J point automatically and calculate the J+60 msec ST-segment level. However, they also allow the operator to adjust manually the J-point and the ST-segment measurement point in patients with abnormal ECG patterns.

One major advantage of continuous ST-segment monitoring, compared with serial 12-lead ECGs, is that if the electrodes stay in place and do not vary as standard 12-lead ECGs may do between different measuring sessions. However, for improved diagnostic accuracy of ST-segment monitoring, the following points should be recognized:

1. Changes in body position may cause ST-segment changes and lead to false ST-segment alarms. However, changes in QRS complex almost always accompany these positional ST-segment changes and therefore can be easily distinguished from true ST-segment deviations. In addition, returning the patient to the supine position may help distinguish true from positional ST-segment deviation. Changes in position of the heart in

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**Figure 47-20.** QRS changes in left bundle branch block (LBBB) are compared with the right bundle branch block (RBBB). (From Zipes DP, Libby P, Bonow R, Braunwald E: Braunwald’s heart disease: a textbook of cardiovascular medicine, ed 7. Philadelphia, 2005, Saunders; and from Goldberger AL: Clinical electrocardiography: a simplified approach, ed 7. St. Louis, 2006, Mosby.)
the mediastinum have also shown to affect the ST segment. Mark and associates\textsuperscript{56} observed that placement of a sternal retractor during cardiac surgery was associated with a reduction in V\textsubscript{5} R-wave amplitude. Simultaneously, V\textsubscript{5} S-wave amplitude and absolute ST-segment deviation were reduced. These investigators concluded that inclusion of an R-wave gain factor might improve perioperative ECG ischemia monitoring.

2. Many patients with CAD do not have perfectly isoelectric ST segments. Early repolarization (a normal variant), intraventricular conduction delays, ventricular hypertrophy, digitalis, and nonspecific ST-T wave abnormalities may cause baseline ST-segment abnormalities. Therefore, tailoring the ST-segment alarm parameters to the patient’s baseline ST-segment level is important. Many current cardiac monitors with ST-segment monitoring software allow clinicians to set alarm parameters manually. If alarm parameters are set 1 to 2 mm around the isoelectric (0 mV) line rather than at the patient’s baseline ST-segment level, then false alarms will frequently occur. Alarm parameters should be set 1 mm above and below the baseline ST-segment level in patients at high risk for ischemia. It should also be remembered that underlying ECG abnormalities might hinder ST-segment analysis in approximately 10% of the patients. These abnormalities include hypokalemia, digitalis, LBBB, Wolff-Parkinson-White syndrome, LVH with strain, and acute pericarditis. In these patients, other modalities for diagnosis of myocardial ischemia, such as transesophageal echocardiography, may be considered.

3. Most cardiac monitors with ST-segment monitoring software provide displays of ST-segment trends in a single lead or summated leads. Although such graphic trends are convenient for the quick identification of potential ischemic events, printing the ECG tracing in question to confirm that the ST-segment changes are the result of ischemia rather than of a transient arrhythmia (e.g., accelerated ventricular rhythm, new bundle branch block) is also of paramount importance.

## ELECTROCARDIOGRAPHIC CRITERIA FOR ACUTE MYOCARDIAL ISCHEMIA

### ST-DEPRESSION–TYPE ISCHEMIA

The ECG criteria most accepted for detecting myocardial ischemia on continuous ECG monitoring are those established and validated during exercise stress testing.\textsuperscript{57} During stress testing and with acute subendocardial ischemia, the electrical forces responsible for the ST segment are deviated toward the inner layer of the heart, causing ST-segment depression (Fig. 47-21). With acute transmural (epicardial) ischemia, the electrical forces in the ischemic area are deviated toward the outer layer of the heart, causing ST-segment elevation in the overlying leads (see Fig. 47-5). As the HR increases, J-point or junctional up-sloping depression normally occurs. In patients with myocardial ischemia, however, the ST segment typically becomes horizontal (flattens) as the severity of ischemia increases. With progressive exercise or tachycardia during ischemia, the ST-segment depression may worsen; the ST segment may become down-sloping, even causing T-wave inversion in more than one ECG lead; and the patient may develop angina (Fig. 47-22). In approximately 10% of patients, especially those who are asymptomatic, ischemia may appear only in the recovery phase after exercise.

The criteria established for stress-induced ischemia are 1 mm (0.1 mV) or more of horizontal or down-sloping ST-segment depression measured 60 to 80 msec after the J point in at least three consecutive beats with a stable baseline. As previously mentioned, the ST-segment depression may be accompanied by T-wave flattening or inversion. Junctional or J-point depression is normal during exercise. In addition, a rapidly up-sloping ST segment (>1 mV/sec) that is also depressed less than 0.15 mV (1.5 mm) is considered normal. In patients with slowly up-sloping ST-segment depression in which the ST segment is depressed 0.15 mV (1.5 mm) or more at J+80 msec, the slowly up-sloping ST-segment depression may signify CAD.

Some patients have preexisting ST-segment abnormalities from a previous MI, bundle branch blocks, or LVH that make ST-segment interpretation more difficult. In these patients, additional horizontal or down-sloping ST-segment depression of 1 mm or greater from baseline is required to be considered as myocardial ischemia. Causes of ST-segment changes other than myocardial ischemia include drugs (most notably digitalis), temperature changes, hyperventilation, and position changes. The distribution of ST-depression–type ischemia correlates poorly with the specific location of CAD.

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**Figure 47-21.** A demonstration of how the ST-segment deviation is measured at 80 msec after the J point (J+80 msec) is illustrated.

**Figure 47-22.** Horizontal ST depression (A) and down-sloping (B) ST depression are indicative of myocardial ischemia. Up-sloping ST depression (C) may be a normal finding.
ST-SEGMENT ELEVATION–TYPE ISCHEMIA

ST-segment elevation in a non–Q-wave lead is extremely uncommon during stress testing, occurring in approximately 1% of patients with CAD, and suggests transmural myocardial ischemia caused by coronary vasospasm or a high-grade coronary narrowing. The ECG site of ST-segment elevation is relatively specific for the myocardial territory involved, in contrast to ST-depression–type ischemia. ST-segment elevation occurring during exercise testing in ECG leads with pathologic Q-waves does not indicate myocardial ischemia, but it does correlate with reduced LV function and a worse prognosis.

T-WAVE CHANGES

Body position, respiration, hyperventilation, and drug therapy, as well as myocardial ischemia or necrosis, largely influence T-wave morphologic appearance. Therefore, T-wave changes not accompanied by significant ST-segment displacement rarely signify myocardial ischemia. Pseudonormalization of T waves (i.e., inverted at rest, becoming upright during exercise) may, in rare instances, be a marker for myocardial ischemia.

In a meta-analysis that included 147 reports of patients who underwent both angiography and exercise testing, the sensitivity and specificity of the exercise ECG stress test for the detection of CAD were 68% and 77%, respectively.58

ELECTROCARDIOGRAPHIC LEAD SENSITIVITY FOR DETECTING PERIOPERATIVE MYOCARDIAL ISCHEMIA

During the perioperative period, ECG monitoring most commonly identifies stress-induced, ST-depression–type ischemia. Such ECG changes do not provide information about the location of the ischemic myocardial area. In contrast, ST-segment elevation indicating transmural ischemia, observed particularly during cardiac surgery, provides useful information about the myocardial segment and coronary perfusion territory responsible for the ischemic episode. Because the majority of modern patient-monitoring systems do not simultaneously monitor all 12 ECG leads, selecting which chest leads to monitor is of great importance, particularly in noncardiac surgery. During exercise stress testing, investigators have identified leads V4 and V5 as the most sensitive leads to detect exercise-induced ischemia (90% to 100% sensitivity).

London and colleagues60 studied high-risk patients undergoing noncardiac surgery and showed that the greatest sensitivity for ischemia was obtained with lead V5 (75%), followed by lead V4 (61%). Combining leads V4 and V5 increased the sensitivity to 90%, whereas with the standard lead II and V5 combination, the sensitivity was only 80%. They also suggested that if three leads (II, V4, and V5) could be simultaneously examined, the sensitivity would increase to 98%. More recently, Landesberg and associates61 monitored continuous 12-lead ST-segment changes greater than 0.2 mV from baseline in a single lead or more than 0.1 mV in two contiguous leads at J+60 msec, lasting longer than 10 minutes in patients undergoing major vascular surgery. Troponins were used as markers for MI. They showed that, in fact, leads V3 and V4 were more sensitive than V5 in detecting perioperative ischemia (87%, 79%, and 66%, respectively). Among patients who experienced an MI, V4 was most sensitive for ischemia (83.3%), followed by V3 and V5 (75% each). Combined monitoring of two of these leads (V3 and V5) increased the sensitivity to approximately 97%.64 The baseline, preanesthesia ST segment was above the isoelectric point in V1 through V3 and below the isoelectric point in V5 through V6. Lead V4 was closest to the isoelectric level on the baseline ECG, rendering it most suitable for detecting ischemia. Lead V4 also detected ischemia earlier and showed a greater ST-segment deviation. Martinez and colleagues,62 who also used the same method to monitor postoperative ischemia in patients in the ICU after major vascular surgery, corroborated these main findings.

For patients with AVSs (i.e., those with atherosclerotic plaque disruption), monitoring limb lead III and leads V3 and V5 as the most sensitive combination for ischemia detection is recommended.63 Monitoring a right-sided precordial lead (V4R) may be of benefit in patients with occlusive disease of the right coronary artery,64 as might inspection of posterior leads (V7 to V9) in suspected posterior ischemia.

Monitoring for intraoperative myocardial ischemia is commonly believed to be unnecessary in neonates. Whereas ECG lead systems for adults are concerned with the detection of ischemia and arrhythmias, neonatal ECG monitoring has focused on arrhythmia recognition alone. Results of some studies, however, suggest that the neonatal heart is more susceptible to ischemia than the adult heart.65 These studies demonstrate the importance of calibrated ECG monitoring in neonates with congenital heart disease (see Chapter 94).

PERIOPERATIVE MYOCARDIAL ISCHEMIA AND INFARCTION

Perioperative myocardial ischemia monitoring in patients undergoing cardiac or noncardiac surgery started more than 20 years ago. Tinker66 noted in 1980 the importance of intraoperative ST-segment monitoring via lead V5 and determined that large fluctuations in systolic blood pressure, HR, or hypothermia with postoperative shivering are associated with ischemia. Coriat and colleagues67 (1982) were pioneers in using Holter monitoring in patients undergoing major vascular surgery, showing that intraoperative ischemia on Holter monitoring was strongly associated with the severity of preoperative angina pectoris. In 1985, Slogoff and Keats68 first demonstrated an association between intraoperative ischemia and postoperative MI. They showed that MI during coronary artery bypass graft (CABG) surgery is strongly associated with ischemia on Holter monitoring detected either before the induction of anesthesia or before the onset of cardiopulmonary bypass. During 1989, studies using perioperative Holter monitoring in patients undergoing major vascular surgery showed that early postoperative tachycardia-induced silent myocardial ischemia was frequent after surgery and associated
with postoperative clinical ischemic events. In 1990, Mangano and associates demonstrated that postoperative Holter-detected ischemia was most common (41%) and most predictive of adverse cardiac events, with a ninefold increase in the odds of postoperative cardiac morbidity and mortality, more than intraoperative or preoperative ischemia.

Between 1990 and 2003 a number of studies with perioperative ischemia monitoring were published. Altogether, these studies included more than 2400 patients. Most studies included patients who underwent major vascular surgery, and most of the studies used Holter monitoring for perioperative ischemia detection (except the studies by Landesberg and colleagues who used continuous on-line 12-lead ECG monitoring). The data show that perioperative ischemia was common in these high-risk patients with an incidence between 24% and 63% and was almost exclusively ST-depression–type ischemia (97% to 100%). The average postoperative MI rate in these studies was 3.9% (range: 0.6% to 15%), the majority of which were non–Q-wave infarctions (66% to 100%). The incidence of all ischemic postoperative cardiac events was 7.3% (range: 3% to 37%), and the average mortality was 1.04% (range: 0% to 2.8%). An important observation was that postoperative ischemia duration was strongly associated with postoperative ischemic cardiac events. Patients with prolonged and often hours of postoperative ST-depression–type ischemia were likely to experience postoperative MI and serum biomarker elevation, whereas short episodes of ischemia (<30 minutes) were unlikely to culminate in MI. Perioperative ischemia was associated not only with early postoperative morbidity and mortality but also with long-term (5 years) morbidity and mortality.

**EXAMPLES OF PERIOPERATIVE ISCHEMIA AND INFARCTION**

**Example 1. Postoperative mortality related to HR-induced prolonged postoperative ST-depression–type ischemia.** Frank and colleagues published one of the first and most detailed cases of postoperative cardiac mortality related to HR-induced prolonged postoperative ST-depression–type ischemia detected on Holter monitoring. A patient who underwent lower-extremity arterial bypass surgery was observed with Holter monitoring, before, during, and after the procedure. His records showed that he had HR-related silent myocardial ischemia preoperatively that subsided intraoperatively, immediately after anesthesia was instituted, only to recur immediately as the patient woke up from the surgery. Figure 47-23 shows the ischemia progressively worsening after surgery until the patient suddenly died 10 hours after the surgery. Interestingly, the ST-depression–type ischemia of this patient occurred at a relatively low HR of 80 to 85 bpm and in the absence of significant blood pressure alterations. Two snapshots of ECG disclosure are presented at approximately 12 o’clock midnight: one at a HR of approximately 90 bpm, which showed deep ST-segment depressions and the other a short time later with a temporary slowing of the HR to approximately 60 bpm, which showed almost complete but transient resolution of the ST-segment depression. Although cardiac markers were not measured and no data were provided on myocardial function by ECG, it is suggested that the patient died as a result of prolonged, silent, HR-related, ST-depression–type ischemia, which was at least temporarily reversible by slowing his HR.

**Example 2. Prolonged myocardial ischemia and infarction immediately after carotid endarterectomy surgery.** A 70-year-old patient with a history of CAD and who was treated 7 years earlier with CABG now has reduced LV function, insulin-dependent diabetes mellitus, and peripheral vascular disease. On arrival to the surgical unit, the patient was connected to a continuous, 12-lead ECG monitor with on-line ST-segment analysis. During surgery, no ischemia was observed (Fig. 47-24). However, immediately after surgery and upon emergence from anesthesia, a significant ST-depression–type ischemia occurred in association with a moderate increase in HR to a maximum of 102 bpm, in accordance with vague pain in his jaw.
Ischemic ST-segment changes lasted 193 minutes, after which the ECG completely reverted to its baseline. Nevertheless, an increase in troponin I to 10.2 ng/mL was measured 6 hours after surgery and up to 32.1 ng/mL on the morning after surgery (see Fig. 47-24; the figure insert depicts all 12-lead ECG complexes during peak ischemia). As demonstrated, significant ST-segment depression occurred in all the chest leads and was deepest in lead V3 (~3.7 mm at peak ischemia). It is interesting to note the significant abnormalities—LVH, RBBB, left anterior hemiblock, and baseline ST-T changes—of this patient’s baseline ECG. Despite these abnormalities, the continuous ST-trend monitoring in 12 leads combined with the significant troponin I elevation leave little doubt that the patient had prolonged (>3 hours) ischemia, which culminated in troponin elevation and, hence, MI.

**Example 3. Prolonged postoperative ischemia and infarction in a patient with chronic total occlusion of one coronary artery.** A 65-year-old patient was admitted for surgery because of an asymptomatic abdominal aortic aneurysm (7 cm). His medical history was only significant for hypertension, mild obesity (85 kg), and smoking until 12 years before admission. He had no clinical history of ischemic heart disease, but, because of a shortness of breath when climbing stairs, a preoperative thallium scan was performed (Fig. 47-25) that showed a moderate-to-severe, large reversible defect in the entire LV anterior wall.

On echocardiography, the size and function of both ventricles were normal with moderate mitral regurgitation. In light of the thallium scanning findings, the patient underwent preoperative coronary angiography (Fig. 47-26), which showed total ostial occlusion of the left anterior descending artery with retrograde filling through a large collateral from the right coronary artery.

Based on these findings, a decision was made not to perform preoperative coronary revascularization; instead, the patient was given bisoprolol (β1-selective blocker) 50 mg daily for a month before he returned for surgery (aortobifemoral bypass).

As in the previous example, this patient was monitored during and after surgery by continuous 12-lead ECG with on-line ST-segment monitoring. Intraoperatively, the HR was 50 to 70 bpm, and no significant ST-segment changes were observed. However, immediately at the end of surgery and extubation, a significant ST-depression–type ischemia developed in association with an increase

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**Figure 47-24.** Lead V3 ST-segment and heart rate trends in a patient with prolonged (193 minutes) postoperative ST-segment depression and troponin elevation. The 12-lead ECG complexes at peak ischemia are superimposed on their baseline patterns (insert).
in HR to a maximum of 98 bpm (Fig. 47-27) and a blood pressure of 155/86 mm Hg. IV esmolol and labetalol were immediately administered, and the ischemia subsided within 15 minutes. The patient was transferred extubated and comfortable to the ICU under continuous infusion of esmolol. In the ICU, the doses of esmolol had to be gradually escalated because of a persistent increase in HR. Postoperative pain was managed by the epidural catheter. Nevertheless, 5 hours after surgery, severe ST-depression–type ischemia recurred in the entire anterior wall. This time, the ischemia lasted more than 5 hours and responded poorly to β-adrenergic blocking drugs (see Fig. 47-27 ). The patient became delirious and developed shortness of breath. Treatment included IV diuretics and a slow infusion of packed red blood cells. Twenty-four hours after surgery, the ischemic ECG changes regressed almost completely to normal, yet serum troponin T levels increased to 0.64 ng/mL and remained elevated for another 5 days.

This unique case demonstrates that postoperative MI may occur as a result of prolonged stress-induced ST-depression–type ischemia in the presence of stable CAD with chronic total coronary artery occlusion of even one coronary artery and without coronary plaque rupture.

Figure 47-25. Preoperative dipyridamole-thallium single photon–emission computed tomographic scan of a patient scheduled for abdominal aortic surgery showing a moderate-to-severe reversible defect in the entire anterior wall.
Figure 47-26. A coronary angiogram shows chronic total ostial occlusion of the left anterior descending (LAD) artery with excellent filling of the distal LAD by a collateral from the right coronary artery. A possible mild stenosis is also noted in the first marginal coronary artery.

Figure 47-27. The perioperative ST-segment and heart rate trends of the patient are demonstrated on the left-sided panels. All 12-lead electrocardiographic complexes during peak ischemia, superimposed over the corresponding baseline (preoperative) complexes, are demonstrated on the right-sided panels. The ST-segment trends show two episodes of ST depression–type ischemia: a short episode of 15 minutes immediately at the end of surgery, followed by a 5-hour period of no ischemia and then another long episode of ST depression lasting longer than 5 hours. Ischemia is evident in all anterior leads but is maximal in V3 and V4 leads.
PATHOPHYSIOLOGIC MECHANISMS OF PERIOPERATIVE MYOCARDIAL INFARCTION

Two distinct mechanisms may lead to perioperative MI (PMI): (1) ACS and (2) prolonged, myocardial oxygen supply-demand imbalance in the presence of significant yet stable CAD. These mechanisms have been recently named type 1 and type 2 MIs by the collaborative Task Force for Universal Definition of MI. A distinction between them is pivotal to therapeutic considerations (Fig. 47-28).

Acute Coronary Syndrome (Type 1 Myocardial Infarction)

The pathophysiologic processes of ACS have been extensively reviewed. Briefly, ACS occurs when an unstable or vulnerable plaque, typically one with a large lipid core, thin fibrous cap, and activated leukocytes, undergoes spontaneous rupture or fissuring with subsequent exposure of thrombogenic material to the bloodstream, leading to acute coronary thrombosis, ischemia, and infarction. Clearly, plaque inflammation plays a pivotal role in spontaneous ACS. In addition, however, external stimuli, such as those operating in the postsurgical setting, are believed to contribute to vulnerable plaque destabilization.

- Physiologic and emotional stresses are long known to be associated with acute MI, raising the hypothesis that sympathetic nervous system–induced hemodynamic, coronary vasoconstrictive, and prothrombotic forces promote plaque disruption and coronary thrombosis. Theoretically, the perioperative period is the perfect playground for such events. Plasma catecholamine and cortisol levels increase early after surgery and may remain high for days. Stress hormones increase with pain, greater surgical trauma, anemia, and hypothermia. Plasma catecholamine levels have been shown to correlate with postoperative cardiac troponin elevations and with thrombotic graft occlusion after vascular surgery. However, an increased rate of plaque rupture and coronary thrombosis in the immediate postoperative period has not been confirmed when these postoperative stresses are at their peak.
- Tachycardia and hypertension, common in the perioperative period, may exert excess shear forces on unstable coronary plaques Severe, stable CAD

Unstable coronary plaques

- Sympathetic hyperactivity (increased plasma catecholamines)
- Hemodynamic instability (tachycardia/hypertension)
- Coronary vasoconstriction

Plaque rupture or Plaque erosion

- ↑ Coagulability/↓ Fibrinolysis
- Recent PCI with stent/premature cessation of dual antiplatelet therapy

Acute coronary thrombosis

Severe, stable CAD

- ↑ Myocardial O₂ demand
- Heart rate/arrhythmia
- Postoperative pain
- Withdrawal of β-blockers
- Hypovolemia
- Cardiac decompensation
- Systemic vasodilatation
- ↑ Blood pressure
- Hypervolemia/↑ LVEDP
- Hemodynamic instability
- Tachycardia/hypertension

Subendocardial O₂ supply

- ↑ Myocardial wall stress
- Hypotension
- Pulmonary congestion/atelectasis
- Hypoxemia

ACS - Type-I MI

Prolonged ST-depression ischemia >> Type-II MI

Figure 47-28. Two distinct mechanisms may lead to postoperative myocardial infarction: (1) type I—spontaneous plaque rupture or erosion and acute coronary thrombosis (acute coronary syndrome [ACS]); and type II—prolonged stress-induced myocardial oxygen supply-demand imbalance in the presence of severe yet stable coronary artery disease (CAD) (non-ACS). LVEDP, Left ventricular end-diastolic pressure; MI, myocardial infarction; PCI, percutaneous coronary intervention. (Redrawn from Landesberg G, Beattie WS, Mosseri M, et al: Perioperative myocardial infarction, Circulation 119:2936-2944, 2009.)
vulnerable plaques with thin fibrous caps and high circumferential tensile stress or cause endothelial stripping or erosion of plaques with severe coronary stenosis, associated with high blood velocities. 99,100

- Increased perioperative procoagulant agents (e.g., fibrinogen, factor-VIII coagulant, von Willebrand factor, α1-antitrypsin), platelet reactivity, 101 decreased endogenous anticoagulant medications (e.g., protein-C and antithrombin-III, α2-macroglobulin), 102 and decreased fibrinolysis 103 have been reported. Postoperative hypercoagulability, however, is principally known for its venous thrombotic complications precipitated by stasis and immobilization. Reports on the association of hypercoagulability and/or decreased fibrinolysis with postoperative myocardial ischemia 104,105 or infarction 106 are rare. An association between impaired fibrinolysis and postoperative lower extremity arterial bypass thrombosis has been reported. 107 Attempts to mimic postoperative stress and hypercoagulability in healthy volunteers caused elevated tissue-plasminogen activator and protein-C activity, consistent with the inhibition of coagulation and augmented fibrinolysis and failed to cause an increase in coagulability. 108

Myocardial Oxygen Supply-Demand Imbalance (Non–Acute Coronary Syndrome, Type 2 Myocardial Infarction)

Data obtained from studies using perioperative monitoring for myocardial ischemia in patients undergoing major, mostly vascular, surgery suggest an alternative, probably significantly more common pathophysiologic condition for PMI. Perioperative Holter monitoring for up to a week after surgery in patients at high risk for cardiac events consistently showed that silent, HR-related postoperative ischemia is common (up to 50%) postoperatively and associated with both in-hospital and long-term morbidity and mortality. 90,91 Postoperative cardiac complications, including sudden death, occur almost exclusively after prolonged (>30 minutes, 84,88 >2 hours, 81,82 or >5 hours72,87), silent ST-depression ischemia. These data have been corroborated by continuous perioperative 12-lead ECG monitoring with on-line ST-segment analysis after major vascular surgery and serial perioperative cardiac troponin measurements. 89,90 Troponin was elevated within hours after the onset of prolonged transient, ST-depression ischemia occurring early after the surgery, and the magnitude of troponin elevation correlated with the duration of ST-depression ischemia. 89 These findings led to the notion that prolonged, stress-induced ST-depression ischemia is a major cause of postoperative troponin elevation and myocardial infarction.

Importantly, however, sequential postoperative troponin measurements demonstrated that low-level, yet prognostically significant troponin elevations occur in patients at high risk who are undergoing major surgery with little or even no evidence of ischemia on ECGs. 90,109,110 Low-level troponin elevation (troponin T >0.03 ng/mL) occurred in as many as 24% of all patients early after major vascular surgery. Only 32% of patients had ECG evidence of ischemia. In contrast, among patients (8.7%) with conventional troponin elevations (>0.1 ng/mL), 88% had ischemia on continuous 12-lead ECG monitoring. 90 Higher troponin values were associated with longer ST-depression-type ischemia and with higher incidence of cardiac symptoms (e.g., pulmonary congestion, chest pain). Hence, prolonged postoperative ischemia, myocardial injury, and type 2 MI probably constitute a spectrum of clinical events ranging from silent, minor, myocardial injury with low-level troponin elevation and low frequency of ischemia on ECG to prolonged, overt ischemia in multiple ECG leads, associated with significant troponin elevation and PMI. Troponin elevation has an immense prognostic importance because troponin elevation measured in the first 3 postoperative days incrementally predicts both early (30 days) 111 and long-term (5 years) 90 postoperative mortality.

As opposed to coronary plaque rupture (type 1 MI) that typically occurs in relatively young and mildly occluding coronary plaques, prolonged stress-induced ST-depression–type ischemia and infarction (type 2 MI) occurs in patients with long-standing, high-grade, often multivessel yet stable CAD 93 (Fig. 47-29).

Multiple triggers may cause myocardial oxygen supply-demand imbalance in the postoperative period (see Fig. 47-28):

- Tachycardia is by far the main cause of postoperative ischemia and type 2 MI. 89,111 An increase in HR of 80 to 90 bpm in patients with significant CAD whose resting HR is 50 to 60 bpm may cause prolonged postoperative ischemia and PMI, demonstrating the low threshold for ischemia in patients after surgery.

Figure 47-29. A schematic representation depicts the probability of type-I and type-II myocardial infarctions as a function of the severity of the coronary artery disease. Coronary plaque rupture mainly occurs in relatively nonoccluding coronary plaques, whereas prolonged myocardial oxygen supply-demand imbalance mainly occurs with severe, often multivessel coronary artery disease. MI, Myocardial infarction.  (Adapted from Landesberg G: The pathophysiology of perioperative myocardial infarction: facts and perspectives, J Cardiothorac Vasc Anesth 17:90-100, 2003.)
• Postoperative tachycardia is frequently associated with either hypotension, secondary to hypovolemia, bleeding, or systemic vasodilatation, or with hypertension induced by stress hormones and vasoconstriction, aggravating tachycardia-induced ischemia. The product of blood pressure and HR correlates with myocardial oxygen demand, whereas the quotient of arterial pressure divided by HR is critical for coronary perfusion, particularly in collateral-dependent myocardium.\textsuperscript{112} 
• Fluid overload in patients with reduced ventricular function may aggravate myocardial wall stress and subendocardial ischemia. 
• Perioperative anemia,\textsuperscript{113} hypoxemia, and hypercarbia are other common precipitants of myocardial ischemia in patients with significant CAD. 
• Stress- and ischemia-induced coronary vasoconstriction\textsuperscript{114,115} may further limit blood flow to areas with marginal coronary circulation.

**TREATMENT AND PREVENTION OF PERIOPERATIVE ISCHEMIA AND INFARCTION**

Because the majority of postoperative ischemic events and MIs are silent, the key to their prevention is a high index of suspicion and the early detection and treatment of any sign or symptom related to the heart such as chest pain, shortness of breath, hypoxemia, and ECG changes. Figure 47-30 proposes an algorithm for the prevention and treatment of perioperative ischemia and infarction. Unfortunately, however, no treatment strategy has thus far been tested by a large enough clinical trial.

The role of prophylactic perioperative \( \beta \) blockade remains highly controversial.\textsuperscript{117,118} Nevertheless, the importance of preventing and treating even modest postoperative increases in the HR cannot be overemphasized. All causes of tachycardia, hypertension, hypotension, anemia, and pain should be aggressively treated. Treatment of tachycardia associated with hypotension is particularly challenging and requires an understanding of the patient’s baseline and postoperative myocardial, valvular, and coronary physiologic condition. Frequently, vasopressors to maintain blood pressure and \( \beta \)-adrenergic blockers to slow HR while managing blood volume, postoperative pain, and respiratory function are necessary. Emergent coronary intervention, anticoagulant agents, or glycoprotein Iib/IIIa antagonists are rarely indicated in the immediate postoperative course and are hazardous because of the risk of bleeding, unless ST-segment elevation or intractable cardiogenic shock ensues.\textsuperscript{119}

Anemia (hematocrit level <39%) independently predicts 30-day mortality, and its correction improved survival in one cohort study of patients who underwent noncardiac surgeries.\textsuperscript{120} Blood transfusion improved survival in patients with CAD who were critically ill and hemoglobin less than 10% but not in patients without ischemia.\textsuperscript{121} Other studies reported increased mortality and nosocomial infections with blood transfusion for hematocrit levels less than 30%.

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**Figure 47-30.** Algorithm for the prevention and treatment of perioperative ischemia and infarction. (Redrawn from Landesberg G, Beattie WS, Musseri M, et al: Perioperative myocardial infarction, Circulation 119:2936-2944, 2009.)
than 25% in stable patients with ACS,\textsuperscript{122} in patients after cardiac surgery,\textsuperscript{123} and in patients in the ICU.\textsuperscript{124} Therefore, hematocrit levels between 25% and 33% is a gray zone in which transfusion must be individualized. Patients who are hemodynamically unstable postoperatively with ischemia may benefit from transfusion. Tight perioperative hemodynamic monitoring, including echocardiography, arterial line, central venous, or possibly pulmonary arterial pressure measurement, is often necessary to determine volume status and to avoid congestive failure.

Complete references available online at expertconsult.com

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REFERENCES


References


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