NARROW-QRS TACHYCARDIAS

Supraventricular arrhythmias (SVA) begin above the bifurcation of the bundle of His. This means that SVA include rhythms that begin in the SA node, the atrial tissue, or the AV junction.

SINUS TACHYCARDIA

Normal heart rates vary with age. In adults, the rate associated with sinus tachycardia is usually between 101 and 180 bpm (Table 3-10, Figure 3-28). Because an infant or child's heart rate can transiently increase during episodes of crying or pain, or in the presence of a fever, the term tachycardia is used to describe a significant and persistent increase in heart rate. In infants, a tachycardia is a heart rate of more than 200 bpm. In a child older than 5 years of age, a tachycardia is a heart rate of more than 160 bpm.

What Causes It?

Sinus tachycardia is a normal response to the body's demand for increased oxygen because of many conditions (Box 3-1). The patient is often aware of an increase in heart rate. Some patients complain of palpitations, a racing heart, or "pounding" in their chest. Sinus tachycardia is seen in some patients with acute MI, especially those with an anterior infarction.

TABLE 3-10 Characteristics of Sinus Tachycardia

| Rate       | 101-180 bpm |
| Rhythm     | Regular     |
| P waves    | Uniform in appearance, positive (upright) in lead II, one precedes each QRS complex; at very fast rates it may be difficult to distinguish a P wave from a T wave |
| PR interval| 0.12-0.20 sec and constant from beat to beat |
| QRS duration| 0.10 sec or less unless an intraventricular conduction delay exists |

BOX 3-1 Causes of Sinus Tachycardia

- Exercise
- Fever
- Pain
- Fear and anxiety
- Hypoxia
- Congestive heart failure
- Acute MI
- Infection
- Sympathetic stimulation
- Shock
- Dehydration, hypovolemia
- Pulmonary embolism
- Hyperthyroidism
- Medications such as epinephrine, atropine, dopamine, and dobutamine
- Caffeine-containing beverages
- Nicotine
- Drugs such as cocaine, amphetamines, "ecstasy," and cannabis
What Do I Do About It?

In a patient with coronary artery disease, sinus tachycardia can cause problems. The heart's demand for oxygen increases as the heart rate increases. As the heart rate increases, there is less time for the ventricles to fill and less blood for the ventricles to pump out with each contraction. This can lead to decreased cardiac output. Because the coronary arteries fill when the ventricles are at rest, rapid heart rates decrease the time for coronary artery filling. This decreases the heart's blood supply. Chest discomfort can result if the supply of blood and oxygen to the heart is inadequate. Sinus tachycardia in a patient who is having an acute MI may be an early warning signal for heart failure, cardiogenic shock, and more serious dysrhythmias.

Treatment for sinus tachycardia is directed at correcting the underlying cause (i.e., fluid replacement, relief of pain, removal of offending medications or substances, and reducing fever and/or anxiety). Sinus tachycardia in a patient experiencing an acute MI may be treated with beta-blockers. Beta-blockers such as atenolol (Tenormin) or metoprolol (Lopressor) are given to slow the heart rate and decrease myocardial oxygen demand, provided there are no signs of heart failure or other contraindications to beta-blocker therapy.

*Never* shock a sinus tachycardia; treat the reason for the tachycardia.
Impulse begins in SA

Sinus rhythm continues at 60 to 100 bpm.

Sinus bradycardia continues at less than 60 bpm.

Sinus tachycardia continues faster than 100 bpm.

Figure 3-28 • A, Sinus rhythm, sinus bradycardia, and sinus tachycardia. B, Sinus tachycardia.
The term SVT includes three main types of fast rhythms, which are shown in Figure 3-29.

- **Atrial tachycardia (AT).** In AT, an irritable site in the atria fires automatically at a rapid rate.
- **Atrioventricular nodal reentrant tachycardia (AVNRT).** In AVNRT, fast and slow pathways in the AV node form an electrical circuit or loop. The impulse spins around the AV nodal (junctional) area.
- **Atrioventricular reentrant tachycardia (AVRT).** In AVRT, the impulse begins above the ventricles but travels via a pathway other than the AV node and bundle of His.

A *nonsustained* rhythm lasts from three beats up to 30 seconds. A *sustained* rhythm lasts more than 30 seconds.

**KEEPING IT SIMPLE**

Some SVTs need the AV node to sustain the rhythm and some don’t. For example, AVNRT and AVRT require the AV node as part of the reentry circuit to continue the tachycardia. Other SVTs use the AV node only to conduct the rhythm to the ventricles. For example, atrial tachycardia, atrial flutter, and atrial fibrillation arise from a site (or sites) within the atria. They do not need the AV node to sustain the rhythm.

![Diagram of normal sinus rhythm, atrial tachycardia, AV nodal reentrant tachycardia, and AV reentrant tachycardia](image)

**Figure 3-29** - Types of supraventricular tachycardias. A, Normal sinus rhythm is presented here as a reference. B, Atrial tachycardia. C, AV nodal reentrant tachycardia. D, AV reentrant tachycardia.
ATRIAL TACHYCARDIA

AT is usually the result of altered automaticity or triggered activity. It consists of a series of rapid beats from an irritable site in the atria. This rapid atrial rate overrides the SA node and becomes the pacemaker. Conduction of the atrial impulse to the ventricles is often 1:1. This means that every atrial impulse is conducted to the ventricles (Figure 3-30).

There is more than one type of AT. Multifocal AT is discussed later in this chapter with irregular tachycardias.

KEEPING IT SIMPLE

AT is often confused with sinus tachycardia. Although atrial looks similar to sinus tachycardia, atrial P waves differ in shape from sinus P waves.

- AT that begins in a small area (focus) within the heart is called focal atrial tachycardia. There are several types of focal atrial tachycardia. Focal AT may be due to an automatic, triggered, or reentrant mechanism. A patient with focal AT often presents with paroxysmal AT. The atrial rate is usually between 100 and 250 bpm and rarely 300 bpm.
- Automatic AT (also called ectopic AT) is another type of AT in which a small cluster of cells with altered automaticity fire. The impulse is spread from the cluster of cells to the surrounding atrium and then to the ventricles via the AV node. This type of AT often has a “warm up” period. This means there is a progressive shortening of the P-P interval for the first few beats of the arrhythmia. Automatic AT gradually slows down as it ends. This has been called a “cool down” period. The atrial rate is usually between 100 and 250 bpm (Table 3-11). P waves look different from sinus P waves but are still related to the QRS complex. Vagal maneuvers do not usually stop the tachycardia, but they may slow the ventricular rate.

What Causes It?

AT can occur in persons with normal hearts or in patients with organic heart disease. Atrial tachycardia associated with automaticity or triggered activity is often related to an acute event including:
- Stimulant use (such as caffeine, albuterol, theophylline, cocaine)
- Infection
- Electrolyte imbalance
- Acute illness with excessive catecholamine release
- MI

<table>
<thead>
<tr>
<th>TABLE 3-11</th>
<th>Characteristics of Atrial Tachycardia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate</td>
<td>150-250 bpm</td>
</tr>
<tr>
<td>Rhythm</td>
<td>Regular</td>
</tr>
<tr>
<td>P waves</td>
<td>One positive P wave precedes each QRS complex in lead II but the P waves differ in shape from sinus P waves. With rapid rates, it is difficult to distinguish P waves from T waves.</td>
</tr>
<tr>
<td>PR interval</td>
<td>May be shorter or longer than normal and may be difficult to measure because P waves may be hidden in T waves</td>
</tr>
<tr>
<td>QRS duration</td>
<td>0.10 sec or less unless an intraventricular conduction delay exists</td>
</tr>
</tbody>
</table>
What Do I Do About It?

If episodes of AT are short, the patient may be asymptomatic. If AT is sustained and the patient is symptomatic because of the rapid rate, treatment usually includes oxygen, IV access, and vagal maneuvers. Although AT will rarely stop with vagal maneuvers, they are used to try to stop the rhythm or slow conduction through the AV node. If this fails, antiarrhythmic medications should be tried. Adenosine is the drug of choice, except for patients with severe asthma. A significant percentage of ATs will terminate with administration of adenosine. If needed, Ca\(^2+\) channel blockers or beta-blockers may be used to slow the ventricular rate (if no contraindications exist). Synchronized cardioversion seldom stops automatic ATs but may be successful for ATs due to reentry or triggered automaticity. Synchronized cardioversion should be considered for patients with drug-resistant arrhythmia. Synchronized cardioversion is discussed in Chapter 4.

The signs and symptoms experienced by a patient with a tachycardia depend on the following:

- Ventricular rate
- How long the tachycardia lasts
- General health, presence of underlying heart disease

The faster the heart rate, the more likely the patient is to have signs and symptoms due to the rapid rate.

---

All the atrial impulses are conducted through the AV node. This results in a P wave preceding each QRS complex.

Although the P waves appear upright, they tend to look different from those seen when the impulse is initiated from the SA node.

An ectopic site in the atria fires at a rate of 150 to 250 times/minute.

Because conducted impulses travel through the ventricles in the usual manner, the QRS complexes appear normal.

**Figure 3-30** Atrial tachycardia.
ATRIOVENTRICULAR NODAL REENTRANT TACHYCARDIA

AVNRT is the most common type of SVT. It is caused by reentry in the area of the AV node. In the normal AV node, there is only one pathway through which an electrical impulse is conducted from the SA node to the ventricles. Patients with AVNRT have two conduction pathways within the AV node that conduct impulses at different speeds and recover at different rates. The fast pathway conducts impulses rapidly but has a long refractory period (slow recovery time). The slow pathway conducts impulses slowly but has a short refractory period (fast recovery time) (Figure 3-31). Under the right conditions, the fast and slow pathways can form an electrical circuit or loop. As one side of the loop is recovering, the other is firing.

AVNRT is usually caused by a premature atrial complex (PAC) that is spread by the electrical circuit. This allows the impulse to spin around in a circle indefinitely, reentering the normal electrical pathway with each pass around the circuit. The result is a very rapid and regular rhythm that ranges from 150 to 250 bpm (Table 3-12, Figure 3-32).

What Causes It?

AVNRT can occur at any age. Whether a person is born with a tendency to have AVNRT or whether it develops later in life for an unknown reason has not been clearly determined. AVNRT is common in young, healthy persons with no structural heart disease. It occurs more often in women than in men. AVNRT also occurs in persons with chronic obstructive pulmonary disease (COPD), coronary artery disease, valvular heart disease, heart failure, and digitalis toxicity. AVNRT can cause angina or MI in patients with coronary artery disease. Possible triggers of AVNRT include the following:

- Hypoxia
- Caffeine
- Stress
- Smoking
- Overexertion
- Sleep deprivation
- Anxiety
- Medications

<table>
<thead>
<tr>
<th>TABLE 3-12</th>
<th>Characteristics of AVNRT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate</td>
<td>150-250 bpm; typically 170-250 bpm.</td>
</tr>
<tr>
<td>Rhythm</td>
<td>Ventricular rhythm is usually very regular.</td>
</tr>
<tr>
<td>P waves</td>
<td>P waves are often hidden in the QRS complex. If the ventricles are stimulated first and then the atria, a negative (inverted) P wave will appear after the QRS in leads II, III, and aVF. When the atria are depolarized after the ventricles, the P wave typically distorts the end of the QRS complex.</td>
</tr>
<tr>
<td>PR interval</td>
<td>P waves are not seen before the QRS complex, therefore the PR interval is not measurable.</td>
</tr>
<tr>
<td>QRS duration</td>
<td>0.10 sec or less unless an intraventricular conduction delay exists</td>
</tr>
</tbody>
</table>

Figure 3-31 • Schematic for SVT due to AV nodal reentry. NSR, Normal sinus rhythm.
What Do I Do About It?

Treatment depends on the severity of the patient’s signs and symptoms. Signs and symptoms that may be associated with rapid ventricular rates include:

- Palpitations (common)
- Nausea
- Lightheadedness
- Nervousness, anxiety
- Neck vein pulsations
- Chest pain or pressure
- Syncope or near-syncope
- Signs of shock
- Neck vein pulsations
- Congestive heart failure
- Dyspnea
- Weakness

If the patient is stable but symptomatic (and symptoms are due to the rapid heart rate), treatment usually includes oxygen, IV access, and vagal maneuvers. If vagal maneuvers do not slow the rate or cause conversion of the tachycardia to a sinus rhythm, the first drug given is usually adenosine. If the patient is unstable, treatment usually includes oxygen, IV access, and sedation (if the patient is awake and time permits), followed by synchronized cardioversion.

A regular, narrow-QRS tachycardia that starts or ends suddenly is called paroxysmal supraventricular tachycardia (PSVT) (Figure 3-33). (PSVT is discussed here because most SVTs are due to AVNRT.) P waves are seldom seen because they are hidden in T waves of preceding beats. The QRS is narrow unless there is a problem with conduction of the impulse through the ventricles, as in a BBB.

ST-segment changes (usually depression) are common in patients with SVTs. In most patients, these ST-segment changes are thought to be the result of repolarization changes. However, in elderly patients and those with a high likelihood of ischemic heart disease, ST-segment changes may represent ECG changes consistent with an acute coronary syndrome. The patient should be watched closely. Appropriate laboratory tests and a 12-lead ECG should be obtained to rule out infarction as needed.
ATRIOVENTRICULAR REENTRANT TACHYCARDIA

AVRT is the second most common type of SVT. Remember that the AV node is normally the only electrical connection between the atria and ventricles. Preexcitation is a term used to describe rhythms that originate from above the ventricles but in which the impulse travels via a pathway other than the AV node and bundle of His. Thus, the supraventricular impulse excites the ventricles earlier than would be expected if the impulse traveled by way of the normal conduction system. Patients with preexcitation syndromes are prone to AVRT.

What Causes It?

During fetal development, strands of myocardial tissue form connections between the atria and ventricles, outside the normal conduction system. These strands normally become non-functional shortly after birth; however, in patients with preexcitation syndrome, these connections persist as congenital malformations of working myocardial tissue. Because these connections bypass part or all of the normal conduction system, they are called accessory pathways. The term bypass tract is used when one end of an accessory pathway is attached to normal conductive tissue. This pathway may connect the right atrial and ventricular walls, the left atrial and ventricular walls, or the atrial and ventricular septa on either the right or the left side.

There are three major forms of preexcitation syndrome, each differentiated by their accessory pathways or bypass tracts (Figure 3-34).

- In Wolff-Parkinson-White (WPW) syndrome, the accessory pathway is called the Kent bundle. This bundle connects the atria directly to the ventricles, completely bypassing the normal conduction system. WPW is the most common preexcitation syndrome. It is more common in men than women. Between 60% and 70% of people with WPW have no associated heart disease. WPW is one of the most common causes of tachyarrhythmias in infants and children. Although the accessory pathway in WPW is believed to be congenital in origin, symptoms associated with preexcitation often do not appear until young adulthood (Table 3-13).

![Figure 3-34](image)

**Figure 3-34** – The three major forms of preexcitation. Location of the accessory pathways and corresponding ECG characteristics.

<table>
<thead>
<tr>
<th>TABLE 3-13</th>
<th>Characteristics of Wolff-Parkinson-White Syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate</td>
<td>Usually 60-100 bpm, if the underlying rhythm is sinus in origin</td>
</tr>
<tr>
<td>Rhythm</td>
<td>Regular, unless associated with atrial fibrillation</td>
</tr>
<tr>
<td>P waves</td>
<td>Upright in lead II unless WPW is associated with atrial fibrillation</td>
</tr>
<tr>
<td>PR interval</td>
<td>If P waves are observed, &lt;0.12 sec because the impulse travels very quickly across the accessory pathway, bypassing the normal delay in the AV node</td>
</tr>
<tr>
<td>QRS duration</td>
<td>Usually &gt;0.12 sec. Slurred upstroke of the QRS complex (delta wave) may be seen in one or more leads.</td>
</tr>
</tbody>
</table>
In Lown-Ganong-Levine (LGL) syndrome, the accessory pathway is called the James bundle. This bundle connects the atria directly to the lower portion of the AV node, thus partially bypassing the AV node. In LGL syndrome, one end of the James bundle is attached to normal conductive tissue. This congenital pathway may be called a bypass tract.

Another unnamed preexcitation syndrome involves the Mahaim fibers. These fibers do not bypass the AV node but originate below the AV node and insert into the ventricular wall, bypassing part or all of the ventricular conduction system. Delta waves are produced with accessory pathways that insert directly into ventricular muscle. A delta wave is the initial slurred deflection at the beginning of the QRS complex. It results from initial activation of the QRS by conduction over the accessory pathway (Figures 3-35, 3-36).

**KEEPING IT SIMPLE**

Recognizing WPW
- Short PR interval
- Delta wave
- Widening of the QRS

**Figure 3-35** • Lead V3, Typical WPW pattern showing the short PR interval, delta wave, wide QRS complex, and secondary ST and T-wave changes.

**Figure 3-36** • Sinus rhythm with a ventricular rate of about 70 bpm. Patient with known WPW; note delta waves. WPW confirmed by 12-lead ECG.
What Do I Do About It?

Persons with WPW are predisposed to tachydysrhythmia (most commonly atrial fibrillation, atrial flutter, or PSVT). This is because the accessory pathway bypasses the protective blocking mechanism provided by the AV node and provides a mechanism for reentry. Some people with WPW never have symptoms. Common signs and symptoms associated with WPW and a rapid ventricular rate include palpitations, lightheadedness, shortness of breath, anxiety, weakness, dizziness, chest discomfort, and signs of shock.

If the patient is symptomatic because of the rapid ventricular rate, treatment will depend on how unstable the patient is, the width of the QRS complex (wide or narrow), and the regularity of the ventricular rhythm. Consultation with a cardiologist is recommended. A stable but symptomatic patient with narrow QRS AVRT is usually treated with O₂, an IV, and attempts to slow or convert the rhythm with vagal maneuvers. If vagal maneuvers fail, IV medications such as amiodarone may be used. Don’t give drugs that slow or block conduction through the AV node, such as adenosine, digoxin, diltiazem, or verapamil. They may speed up conduction through the accessory pathway.

Junctional Tachycardia

Junctional tachycardia is an ectopic rhythm that begins in the pacemaker cells found in the bundle of His. When three or more sequential premature junctional complexes (PJC) occur at a rate of more than 100 bpm, a junctional tachycardia exists. Nonparoxysmal (gradual onset) junctional tachycardia usually starts as an accelerated junctional rhythm, but the heart rate gradually increases to more than 100 bpm. The usual ventricular rate for nonparoxysmal junctional tachycardia is 101 to 140 bpm (Table 3-14). Paroxysmal junctional tachycardia starts and ends suddenly and is often precipitated by a PJC. The ventricular rate for paroxysmal junctional tachycardia is generally faster, 140 bpm or more.

If the AV junction paces the heart, the electrical impulse must travel in a backward (retrograde) direction to activate the atria. If a P wave is seen, it will be upside down in leads II, III, and aVF because the impulse is traveling away from the positive electrode (Figure 3-37).

<table>
<thead>
<tr>
<th>TABLE 3-14</th>
<th>Characteristics of Junctional Tachycardia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate</td>
<td>101-180 bpm.</td>
</tr>
<tr>
<td>Rhythm</td>
<td>Very regular.</td>
</tr>
<tr>
<td>P waves</td>
<td>May occur before, during, or after the QRS. If visible, the P wave is inverted in leads II, III, and aVF</td>
</tr>
<tr>
<td>PR interval</td>
<td>If a P wave occurs before the QRS, the PR interval will usually be ≥0.12 sec. If no P wave occurs before the QRS, there will be no PR interval.</td>
</tr>
<tr>
<td>QRS duration</td>
<td>Usually 0.10 sec or less unless an intraventricular conduction delay exists.</td>
</tr>
</tbody>
</table>

Figure 3-37 • Junctional tachycardia.

What Causes It?

Junctional tachycardia may occur because of an acute coronary syndrome, heart failure, theophylline administration, or digitalis toxicity (common cause).
What Do I Do About It?

With sustained ventricular rates of 150 bpm or more, the patient may complain of a “racing heart” and severe anxiety. Because of the fast ventricular rate, the ventricles may be unable to fill completely, resulting in decreased cardiac output. Junctional tachycardia associated with an acute coronary syndrome may

- Increase myocardial ischemia
- Increase the frequency and severity of chest pain
- Extend the size of an MI
- Cause heart failure, hypotension, or cardiogenic shock
- Predispose the patient to ventricular dysrhythmias

Treatment depends on the severity of the patient’s signs and symptoms. If the patient tolerates the rhythm, observation is often all that is needed. If the patient is symptomatic as a result of the rapid rate, initial treatment should include oxygen and IV access. Because it is often difficult to distinguish junctional tachycardia from other narrow-QRS tachycardias, vagal maneuvers and, if necessary, IV adenosine may be used to help determine the origin of the rhythm (Table 3-15). A beta-blocker or Ca²⁺ channel blocker may be ordered (if no contraindications exist). If the rhythm is the result of digitalis toxicity, the drug should be withheld. If the rhythm is the result of theophylline administration, the infusion should be slowed or stopped.

Vagal Maneuvers

Vagal maneuvers are methods used to stimulate baroreceptors located in the internal carotid arteries and the aortic arch. Stimulation of these receptors results in reflex stimulation of the vagus nerve and release of acetylcholine. Acetylcholine slows conduction through the AV node, resulting in slowing of the heart rate. Although there is some overlap of the right and left vagus nerves, it is thought that the right vagus nerve has more fibers to the SA node and atrial muscle and the left vagus more fibers to the AV node and some ventricular muscle.

Examples of vagal maneuvers include the following:

- Coughing.
- Squatting.
- Breath-holding.
- Carotid sinus pressure. This procedure is performed with the patient’s neck extended. Firm pressure is applied just underneath the angle of the jaw for up to 5 seconds. Carotid pressure should be avoided in older patients and in patients with carotid artery bruits. Simultaneous, bilateral carotid pressure should never be performed.
- Application of a cold stimulus to the face (such as a washcloth soaked in iced water, cold pack, or crushed ice mixed with water in a plastic bag or glove) for up to 10 seconds. This technique is often effective in infants and young children. When using this method, do not obstruct the patient’s mouth or nose or apply pressure to the eyes.
- Valsalva’s maneuver. Ask the patient to blow through an occluded straw or take a deep breath and bear down as if having a bowel movement for up to 10 seconds. This strains the abdominal muscles and increases intrathoracic pressure.
- Gagging. Use a tongue depressor or culturette swab to briefly touch the back of the throat. When using vagal maneuvers, keep the following points in mind:
  - Make sure oxygen, suction, a defibrillator, and emergency medications are available before attempting the procedure.
  - A 12-lead ECG recording is desirable when a vagal maneuver is performed.
  - Continuous monitoring of the patient’s ECG is essential. Note the onset and end of the vagal maneuver on the ECG rhythm strip.
  - In general, a vagal maneuver should not be continued for more than 10 seconds.
  - Application of external ocular pressure may be dangerous and should not be used because of the risk of retinal detachment.
  - Carotid massage is less effective in children than in adults and is not recommended.
WIDE-QRS TACHYCARDIAS

The width of a QRS complex is most accurately determined when it is viewed and measured in more than one lead. The measurement should be taken from the QRS complex with the longest duration and clearest onset and end.

INTRAVENTRICULAR CONDUCTION DEFECTS

A delay or block can occur in any part of the intraventricular conduction system. If a delay or block occurs in one of the bundle branches, the ventricles will not depolarize at the same time. The impulse travels first down the unblocked branch and stimulates that ventricle. Because of the block, the impulse must then travel from cell to cell through the myocardium (rather than through the normal conduction pathway) to stimulate the other ventricle. This means of conduction is slower than normal, and the QRS complex appears widened on the ECG. The ventricle with the blocked bundle branch is the last to be depolarized.

A QRS measuring 0.10 to 0.12 second is called an incomplete right or left BBB. A QRS measuring more than 0.12 second is called a complete right or left BBB. If the QRS is wide but there is no BBB pattern, the term “wide QRS” or “intraventricular conduction delay” is used to describe the QRS.

ECG criteria for identification of a right or left BBB are

- A QRS duration of more than 0.12 second (if a complete BBB).
- QRS complexes produced by supraventricular activity (i.e., the QRS complex is not a paced beat nor did it originate in the ventricles).
- To determine right versus left BBB
  - Look at lead V1 or MCL1.
  - Move from the J-point back into the QRS complex and determine if the terminal portion (last 0.04 second) of the QRS complex is a positive (upright) or negative (downward) deflection (Figures 3-38, 3-39).
  - If the two criteria for BBB are met and the terminal portion of the QRS is positive, a right BBB is most likely present. If the terminal portion of the QRS is negative, a left BBB is most likely present.

In left bundle branch block (LBBB), activation of the septum is altered and the right ventricle depolarizes before the left. Thus, abnormal Q waves originating from the left ventricle may be obscured. Further, ST-segment and T-wave changes are often present with LBBB, making the diagnosis of acute MI even more difficult.1

<table>
<thead>
<tr>
<th>Dysrhythmia</th>
<th>Effects of Vagal Maneuvers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus tachycardia</td>
<td>Gradual slowing and return to previous rate upon cessation of maneuver</td>
</tr>
<tr>
<td>AVNRT</td>
<td>Abrupt cessation of the tachycardia or no effect</td>
</tr>
<tr>
<td>AVRT</td>
<td>Abrupt cessation of the tachycardia or no effect</td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>Ventricular rate unchanged or temporarily slowed; flutter waves may be revealed during maneuver</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>Ventricular rate unchanged or temporarily slowed</td>
</tr>
<tr>
<td>Ventricular tachycardia</td>
<td>No effect</td>
</tr>
</tbody>
</table>
ACCELERATED IDIOVENTRICULAR RHYTHM

An accelerated idioventricular rhythm (AIVR) exists when three or more ventricular escape beats occur in a row at a rate of 41 to 100 bpm (Figure 3-40, Table 3-16). Although this heart rate is not considered a tachycardia, AIVR is discussed here because some cardiologists consider the upper end of the rate range to be about 120 bpm.

AIVR is usually considered a benign escape rhythm that appears when the sinus rate slows and disappears when the sinus rate speeds up. Episodes of AIVR usually last a few seconds to a minute. Because AIVR usually begins and ends gradually, it is also called nonparoxysmal VT.
AIVR occurs most often in the setting of acute MI, most often during the first 12 hours. It is particularly common after successful reperfusion therapy. AIVR has been observed in patients with the following:

- Digitalis toxicity
- Cocaine toxicity
- Subarachnoid hemorrhage
- Acute myocarditis
- Hypertensive heart disease
- Dilated cardiomyopathy

AIVR generally requires no treatment because the rhythm is protective and often transient, spontaneously resolving on its own. However, possible dizziness, lightheadedness, or other signs of hemodynamic compromise may occur because of the loss of atrial kick.

### VENTRICULAR TACHYCARDIA

VT exists when three or more PVCs occur in immediate succession at a rate greater than 100 bpm. VT may occur as a short run lasting less than 30 seconds (nonsustained) (Figure 3-41), but it more commonly persists for more than 30 seconds (sustained). VT may occur with or without pulses, and the patient may be stable or unstable with this rhythm.

VT, like PVCs, may originate from an ectopic focus in either ventricle. When the QRS complexes of VT are of the same shape and amplitude, the rhythm is called **monomorphic VT** (Figure 3-42, Table 3-17). When the QRS complexes of VT vary in shape and amplitude from beat to beat, the rhythm is called polymorphic VT. In polymorphic VT, the QRS complexes appear to twist from upright to negative or negative to upright and back. Polymorphic VT is discussed later in this chapter, with irregular tachycardias.

Monomorphic VT with a ventricular rate greater than 200 bpm is called ventricular flutter by some cardiologists.

<table>
<thead>
<tr>
<th>TABLE 3-16</th>
<th>Characteristics of Accelerated Idioventricular Rhythm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate</td>
<td>41-100 bpm (41-120 bpm per some cardiologists)</td>
</tr>
<tr>
<td>Rhythm</td>
<td>Essentially regular</td>
</tr>
<tr>
<td>P waves</td>
<td>Usually absent or, with retrograde conduction to the atria, may appear after the QRS (usually upright in the ST-segment or T wave)</td>
</tr>
<tr>
<td>PR interval</td>
<td>None</td>
</tr>
<tr>
<td>QRS duration</td>
<td>&gt;0.12 sec; T wave usually in opposite direction of the QRS complex</td>
</tr>
</tbody>
</table>

**Figure 3-41** - Nonsustained VT.
What Causes It?
Sustained monomorphic VT is often associated with underlying heart disease, particularly myocardial ischemia. It rarely occurs in patients without underlying heart disease. Common causes of VT include the following:
- Acute coronary syndromes
- Cardiomyopathy
- Tricyclic antidepressant overdose
- Digitalis toxicity
- Valvular heart disease
- Cocaine abuse
- Mitral valve prolapse
- Acid-base imbalance
- Trauma (such as myocardial contusion, invasive cardiac procedures)
- Electrolyte imbalance (such as hypokalemia, hyperkalemia, hypomagnesemia)

What Do I Do About It?
Signs and symptoms associated with VT vary. VT may occur with or without pulses. The patient who has sustained monomorphic VT may be stable for long periods of time. However, when the ventricular rate is very fast, or when myocardial ischemia is present, monomorphic VT can degenerate to polymorphic VT or ventricular fibrillation (VF). Syncope or near-syncope may occur because of an abrupt onset of VT. The patient’s only warning symptom may be a brief period of lightheadedness.

Treatment is based on the patient’s signs and symptoms and the type of VT. If the rhythm is monomorphic VT (and the patient’s symptoms are due to the tachycardia):
- CPR and defibrillation are used to treat the pulseless patient in VT.
- Stable but symptomatic patients are treated with oxygen, IV access, and ventricular anti-arrhythmics (such as amiodarone) to suppress the rhythm.
- Unstable patients (usually a sustained heart rate of 150 bpm or more) are treated with oxygen, IV access, and sedation (if awake and time permits) followed by synchronized cardioversion.
- In all cases, an aggressive search must be made for the cause of the VT.
Sustained VT does not always produce signs of hemodynamic instability.

SVT with an intraventricular conduction delay may be difficult to distinguish from VT. Keep in mind that VT is considered a potentially life-threatening dysrhythmia. If you are unsure whether a regular, wide-QRS tachycardia is VT or SVT with an intraventricular conduction delay, treat the rhythm as VT until proven otherwise. Obtaining a 12-lead ECG may help differentiate VT from SVT, but do not delay treatment if the patient is symptomatic.

IRREGULAR TACHYCARDIAS

MULTIFOCAL ATRIAL TACHYCARDIA

Wandering atrial pacemaker is rhythm in which the size, shape, and direction of the P waves vary, sometimes from beat to beat. The difference in the look of the P waves is a result of the gradual shifting of the dominant pacemaker between the SA node, the atria, and/or the AV junction (Figure 3-43). When a wandering atrial pacemaker is associated with a ventricular rate greater than 100 bpm, the rhythm is called multifocal atrial tachycardia (MAT) (Figure 3-44, Table 3-18). MAT is also called chaotic atrial tachycardia.

**Lead II (continuous)**

**Figure 3-43**  •  Wandering atrial pacemaker. Continuous strip (lead II).

**Figure 3-44**  •  MAT.

**TABLE 3-18**  Characteristics of Multifocal Atrial Tachycardia

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rate</strong></td>
<td>Ventricular rate is &gt;100 bpm.</td>
</tr>
<tr>
<td><strong>Rhythm</strong></td>
<td>May be irregular as the pacemaker site shifts from the SA node to ectopic atrial locations and the AV junction.</td>
</tr>
<tr>
<td><strong>P waves</strong></td>
<td>Size, shape, and direction may change from beat to beat; at least three different P-wave configurations (seen in the same lead) are required for a diagnosis of wandering atrial pacemaker or multifocal atrial tachycardia.</td>
</tr>
<tr>
<td><strong>PR interval</strong></td>
<td>Variable.</td>
</tr>
<tr>
<td><strong>QRS duration</strong></td>
<td>Usually 0.10 sec or less unless an intraventricular conduction delay exists.</td>
</tr>
</tbody>
</table>
What Causes It?
In MAT, multiple ectopic sites stimulate the atria. MAT is most often seen in:
- Severe COPD
- Hypoxia
- Acute coronary syndromes
- Digoxin toxicity
- Rheumatic heart disease
- Theophylline toxicity
- Electrolyte imbalances

What Do I Do About It?
Treatment of MAT is directed at the underlying cause. If the patient is stable and symptomatic but you are uncertain if the rhythm is MAT, you can try a vagal maneuver. If vagal maneuvers are ineffective, you can try adenosine IV. Remember that MAT is the result of random and chaotic firing of multiple sites in the atria. MAT does not involve reentry through the AV node. Therefore, it is unlikely that vagal maneuvers or giving adenosine will terminate the rhythm. However, they may momentarily slow the rate enough so that you can look at the P waves and determine the specific type of tachycardia. By determining the type of tachycardia, treatment specific to that rhythm can be given.

If you know the rhythm is MAT and the patient is symptomatic, treatment may include medications such as Ca²⁺ channel blockers. Beta-blockers are usually contraindicated because of the presence of severe underlying pulmonary disease.

ATRIAL FLUTTER
Atrial flutter is an ectopic atrial rhythm in which an irritable site fires regularly at a very rapid rate. Because of this extremely rapid stimulation, waveforms are produced that resemble the teeth of a saw, or a picket fence, called “flutter” waves (Figure 3-45). Flutter waves are best observed in leads II, III, aVF, and V1. If each impulse were sent to the ventricles, the ventricular rate would equal 300 bpm. The healthy AV node protects the ventricles from these extremely fast atrial rates.

Atrial flutter with an atrial rate of 300 bpm and a ventricular rate of 150 bpm = 2:1 conduction; 100 bpm = 3:1 conduction; 75 bpm = 4:1 conduction; 50 bpm = 6:1 conduction, and so on. Although conduction ratios in atrial flutter are often even (2:1, 4:1, 6:1), variable conduction can also occur, producing an irregular ventricular rhythm (Table 3-19).

<table>
<thead>
<tr>
<th>TABLE 3-19</th>
<th>Characteristics of Atrial Flutter</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate</td>
<td>In type I atrial flutter (also called typical rapid atrial flutter), the atrial rate ranges from 250-350 bpm. In type II atrial flutter (also called atypical or very rapid atrial flutter), the atrial rate ranges from 350-450 bpm.</td>
</tr>
<tr>
<td>Rhythm</td>
<td>Atrial regular, ventricular regular or irregular depending on AV conduction/blockade.</td>
</tr>
<tr>
<td>P waves</td>
<td>No identifiable P waves; saw-toothed ‘flutter’ waves are present.</td>
</tr>
<tr>
<td>PR interval</td>
<td>Not measurable.</td>
</tr>
<tr>
<td>QRS duration</td>
<td>Usually 0.10 sec or less but may be widened if flutter waves are buried in the QRS complex or if an intraventricular conduction delay exists.</td>
</tr>
</tbody>
</table>
What Causes It?

Atrial flutter is usually caused by a reentry circuit in which an impulse circles around a large area of tissue, such as the entire right atrium. It is usually a paroxysmal rhythm that is precipitated by a premature atrial complex. It may last for seconds to hours and occasionally 24 hours or more. Chronic atrial flutter is unusual. This is because the rhythm usually converts to sinus rhythm or atrial fibrillation, either on its own or with treatment. Conditions associated with atrial flutter are shown in Box 3-2.

What Do I Do About It?

Treatment decisions are based on the ventricular rate, the duration of the rhythm, the patient’s general health, and how he or she is tolerating the rhythm. When atrial flutter is present with 2:1 conduction, it may be difficult to tell the difference between atrial flutter and sinus tachycardia.
cardia, atrial tachycardia, AVNRT, AVRT, or PSVT. Vagal maneuvers may help identify the rhythm by temporarily slowing AV conduction and revealing the underlying flutter waves (Figure 3-46). If atrial flutter is associated with a rapid ventricular rate and the patient is stable but symptomatic, treatment may be directed toward controlling the ventricular rate or converting the rhythm to a sinus rhythm. In the prehospital and emergency department setting, treatment is usually aimed at controlling the ventricular rate. Consider synchronized cardioversion if a patient is in atrial flutter with a rapid ventricular rate and has serious signs and symptoms (such as low blood pressure, signs of shock, or heart failure).

Atrial flutter or atrial fibrillation that has a ventricular rate of more than 100 bpm is described as “uncontrolled.” The ventricular rate is considered “rapid” when it is 150 bpm or more. New-onset atrial flutter or atrial fibrillation (AFib) is often associated with a rapid ventricular rate. Atrial flutter or AFib with a rapid ventricular response is commonly called “flutter with RVR” or “AFib with RVR.”

Atrial flutter or AFib that has a ventricular rate of less than 100 bpm is described as “controlled.” A controlled ventricular rate may be the result of a healthy AV node protecting the ventricles from very fast atrial impulses or drugs used to control (block) conduction through the AV node, decreasing the number of impulses reaching the ventricles.

**Figure 3-46** Atrial flutter. A, This rhythm strip shows a narrow-QRS tachycardia with a ventricular rate just under 150 bpm. B, The same rhythm shown in A with arrows added indicating possible atrial activity. C, When carotid sinus massage is performed, the rate of conduction through the AV node slows, revealing atrial flutter. CSM, Carotid sinus massage.
ATRIAL FIBRILLATION

AFib occurs because of altered automaticity in one or several rapidly firing sites in the atria or reentry involving one or more circuits in the atria (Figure 3-47). These rapid impulses cause the muscles of the atria to quiver (fibrillate). This results in ineffectual atrial contraction, decreased stroke volume, a subsequent decrease in cardiac output, and loss of atrial kick (Table 3-20).

What Causes It?

Atrial fibrillation can occur in patients with or without detectable heart disease or related symptoms. Conditions associated with AFib are shown in Box 3-3.
What Do I Do About It?

Treatment decisions are based on the ventricular rate, the duration of the rhythm, the patient’s general health, and how he or she is tolerating the rhythm. If AFib is associated with a rapid ventricular rate and the patient is stable but symptomatic, treatment may be directed toward controlling the ventricular rate or converting the rhythm to a sinus rhythm (Figure 3-48). In the prehospital and emergency department settings, treatment is usually aimed at controlling the ventricular rate. Consider synchronized cardioversion if a patient is in AFib with a rapid ventricular rate and has serious signs and symptoms (such as low blood pressure, signs of shock, or heart failure).

Patients who experience AFib are at increased risk of having a stroke. Because the atria do not contract effectively and expel all of the blood within them, blood may pool within them and form clots. A stroke can result if a clot moves from the atria and lodges in an artery in the brain.

**Figure 3-48** - Atrial fibrillation with a rapid ventricular response.
POLYMORPHIC VENTRICULAR TACHYCARDIA

In polymorphic VT, the QRS complexes appear to twist from upright to negative or negative to upright and back (Figure 3-49). Polymorphic VT that occurs in the presence of a long QT interval is called torsades de pointes. Polymorphic VT that occurs in the presence of a normal QT interval is simply referred to as polymorphic VT or polymorphic VT resembling torsades de pointes (Table 3-21).

What Causes It?

Polymorphic VT may be precipitated by slow heart rates or associated with medications or electrolyte disturbances that prolong the QT interval. A prolonged QT interval may be congenital or acquired.

What Do I Do About It?

Symptoms are usually related to the decreased cardiac output that occurs because of the fast ventricular rate. Signs of shock are often present. The patient may experience a syncopal episode or seizures. The rhythm may occasionally terminate spontaneously and recur after several seconds or minutes, or it may deteriorate to VF.

If the rhythm is polymorphic VT, it is important to determine if the patient's QT interval just before the tachycardia is normal or prolonged. If the QT interval is normal and the patient is symptomatic due to the tachycardia, treat ischemia if present, correct electrolyte abnormalities, and proceed with electrical therapy or antiarrhythmic medications if necessary. If the QT interval is prolonged and the patient is symptomatic due to the tachycardia, discontinue any medications the patient may be taking that prolong the QT interval, correct electrolyte abnormalities, and proceed with electrical therapy or antiarrhythmic medications if necessary. If the rhythm is sustained polymorphic VT and the patient is unstable or has no pulse, defibrillate.

**TABLE 3-21** Characteristics of Polymorphic Ventricular Tachycardia

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate</td>
<td>150-300 bpm, typically 200-250 bpm</td>
</tr>
<tr>
<td>Rhythm</td>
<td>May be regular or irregular</td>
</tr>
<tr>
<td>P waves</td>
<td>None</td>
</tr>
<tr>
<td>PR interval</td>
<td>None</td>
</tr>
<tr>
<td>QRS duration</td>
<td>&gt;0.12 sec; gradual alteration in amplitude and direction of the QRS complexes; a typical cycle consists of 5-20 QRS complexes</td>
</tr>
</tbody>
</table>

*Figure 3-49* • When the QRS complexes of VT vary in shape and amplitude, the rhythm is termed polymorphic VT.
**Quick Review 3-13**

1. What dysrhythmia is characterized by waveforms resembling teeth of a saw or picket fence before each QRS?

2. What is the significance of the relative refractory period?

3. How do you determine if the ventricular rhythm on a rhythm strip is regular or irregular?

4. Describe the appearance of atrial activity in atrial fibrillation on the ECG.

5. Name a consequence of decreased ventricular filling time.

6. What is the most common preexcitation syndrome?