In this section, you will learn about the various disorders of impulse formation and conduction that can cause tachycardia, often called tachyarrhythmias. Before diving into details regarding the various tachyarrhythmias, you need to learn a few basic concepts that will help you to understand and to organize your thinking.

**Concept: Supraventricular Tachycardia vs. Ventricular Tachycardia**

One classification scheme for tachycardias is to group them as supraventricular or ventricular based on the site of the abnormal impulse formation. Tachycardias that result from abnormal impulse formation in the SA node, atria, or AV node are called supraventricular tachycardias (SVT). Tachycardias that are caused by abnormal impulse formation in the ventricles are called ventricular tachycardias (VT).

**Concept: Regular Tachycardia vs. Irregular Tachycardia**

The terms “regular” and “irregular” are often used to describe and classify tachycardias. The terms simply refer to the regularity of the timing of the QRS complexes, or, in other words, whether or not the QRS complexes are evenly spaced on the ECG.

**Concept: Automatic Tachycardias vs. Reentrant Tachycardias**

Normally, cardiac impulses originate in the cells of the SA node due to the characteristic feature of the action potential (spontaneous phase 4 depolarization) that allows those cells to depolarize themselves in a rhythmic fashion. This feature is referred to as automaticity. You have learned previously that other cells in the heart (e.g. AV Node) also have automaticity, but that the natural rate of phase 4 depolarization is slower. Thus these other cells only assume pacemaker control of the heart rate when there is failure of the SA Node, a process known as escape. However, various pathologic conditions may cause myocardial cells that do not normally have automaticity to develop spontaneous phase 4 depolarization. If the rate of spontaneous depolarization is faster than the SA node, a tachyarrhythmia may result. Tachycardias that result from enhanced automaticity in some part of the heart are called Automatic tachycardias.
Reentry is another general mechanism for tachyarrhythmia. Reentry may occur when an electrical impulse encounters separate and electrophysiologically distinct pathways that are anatomically connected at both ends as shown below. In this example (Figure 1), a wave of depolarization (impulse for short), shown as the heavy black arrow in the left panel, depolarizes from top to bottom via separate but anatomically connected pathways. In this example, the alpha pathway is characterized by fast conduction, but slow recovery (longer refractory period). The beta pathway is the opposite, slow conduction but fast recovery. In the normal circumstance (left panel) the impulse travels from the proximal common pathway to the distal common pathway via both pathways, albeit at slightly different speeds. The middle panel shows what happens when the next impulse arrives early (for example, due to a premature atrial contraction, PAC). Conduction in the alpha pathway is blocked because the tissue is slower to recover and is still refractory while conduction does occur in the beta pathway because of its faster recovery. The right panel shows what happens when the impulse traverses the beta pathway and reaches the distal common pathway. Due to the extra time it takes the impulse to propagate the slower beta pathway, the alpha pathway tissue is no longer refractory. Therefore, in addition to exciting the distal tissue, the impulse travels retrograde up the alpha pathway (bottom dotted arrow). When it reaches the proximal common pathway, it may send a retrograde impulse to the proximal tissue (heavy white arrow) and also reenter the beta pathway (top dotted arrow). Since the overall distances involved are quite small, this “circus movement” may spin off rapid depolarizations in both directions, resulting in tachycardia.

**Figure 1** A schematic representation of reentry.
Concept: Narrow QRS Tachycardia vs. Wide QRS Tachycardia

Another classification scheme for tachycardias is based on whether the QRS complex duration is normal (< 0.10 sec) or wide. To understand this, consider a tachycardia that originates from abnormal impulse formation in the atria (SVT). If conduction of the impulse to the ventricles is normal (i.e. no bundle branch block), the QRS duration will be normal (narrow). However, if the impulse is conducted abnormally to the ventricles (for example, if bundle branch block were present), the QRS duration will be prolonged (wide). Thus, supraventricular tachycardias (SVTs) can be classified as either Narrow QRS or Wide QRS, depending on whether impulse conduction is normal or abnormal. Now consider a tachycardia caused by abnormal impulse formation in the ventricles (VT). Clearly, impulse conduction will not occur in the usual fashion (via the His-Purkinje system) and thus it will take longer than normal to completely depolarize the ventricles. This will result in a prolonged (wide) QRS. Thus all ventricular tachycardias (VT) are also Wide QRS Tachycardias, while VT may be either narrow or wide. In clinical practice, when we see a Wide QRS Tachycardia (WCT), it is not always easy to tell whether it is SVT or VT, so the term Wide QRS Tachycardia (also called Wide Complex Tachycardia) is most appropriately used to reflect that uncertainty.

Figure 2. The tachycardias
Approach to Diagnosing Tachycardias

Diagnosing tachycardias can be challenging, but it is one of the most important aspects of reading ECGs. Shortly, you will learn an easy step-wise approach that will make the task much easier. One general tip in diagnosing tachycardias is to look at the rhythm strip at the bottom of the ECG. You may need to look at the other leads as well, but start with the rhythm strip. On the wards, it is often possible to set the ECG machine to record extra long rhythm strips if needed or to print long rhythm strips when the patient is hooked to a bedside monitor. A general point of clarification is that when we talk about tachycardias, we mean that the atria, or the ventricles, or both, are going fast. This will become clear shortly.

Tachycardia diagnosis involves a short series of questions.

♥ **Question 1**: Is the QRS narrow or wide?

If the QRS is **wide** (Wide Complex tachycardia, WCT), you have learned that the tachycardia could be a ventricular tachycardia (VT) or a supraventricular tachycardia (SVT) with aberrant conduction. We will discuss WCT at the end of this chapter.

If the QRS is **narrow**, you can be sure that the tachycardia is supraventricular (SVT). However, there are many different types of SVT, so you must ask the next question.

♥ **Question 2**: Is the rhythm regular or irregular?

By this question is meant whether the QRS complexes are evenly spaced (regular) or are the R-R intervals variable (irregular). The answer to this question allows us to separate the SVTs into a couple of groups as shown in the table below. The various SVTs are then classified by whether they are caused by increased automaticity (*) or by reentry ®. To narrow the diagnosis down further, you need to know some details about each of the tachycardias.

<table>
<thead>
<tr>
<th>Regular SVT</th>
<th>Irregular SVT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus Tachyrdia (A)</td>
<td>Sinus Tachycardia with PACs (A)</td>
</tr>
<tr>
<td>(Ectopic) Atrial Tachycardia (EAT) (A)</td>
<td>Multifocal Atrial Tachycardia (A)</td>
</tr>
<tr>
<td>PSVT</td>
<td>Atrial Fibrillation (R)</td>
</tr>
<tr>
<td>- AV Nodal Reentrant Tachycardia (R)</td>
<td></td>
</tr>
<tr>
<td>- AV Reentrant Tachycardia (R)</td>
<td></td>
</tr>
<tr>
<td>Atrial Flutter (R)</td>
<td>Atrial Flutter with variable block (R)</td>
</tr>
</tbody>
</table>

Table 1. The Supraventricular Tachycardias

(A) Tachycardia due to increased automaticity
(R) Tachycardia due to reentry mechanism
Premature Atrial Complex (PAC)

Premature atrial complexes (PACs) are due to enhanced automaticity in one or more foci in the atrium. PACs are recognized as P waves which are “early” or premature relative to the normal P-P interval. If they originate near the SA node they may have a nearly normal appearance, but may have a different appearance than the usual sinus P wave if they originate at a site away from the SA node. When they originate low in the atrium, the wave of atrial depolarization is directed superiorly and the P wave may be inverted in lead II. The PAC is usually conducted to the ventricles but if it is too early, and the AV Node is still refractory, it may be “blocked” and no QRS will be seen following the PAC. PACs are commonly found in healthy subjects and usually do not require specific treatment, but may be increased by fatigue, stress, alcohol, smoking and structural heart disease. Examples of PACs are shown below.

Figure 3. Premature atrial complex (PAC). Shown are examples of PACs. In the top panel the sixth and QRS complex follows a PAC. Note how the morphology of the PAC differs from the normal sinus P wave and how the PR interval is slightly shorter. Why? In the bottom panel, the third and sixth QRS complexes follow PACs.
**Sinus Tachycardia**

Sinus tachycardia is due to an increased rate of discharge from the SA node. It is generally due to increased sympathetic and/or decreased parasympathetic (vagal) tone. It is a normal physiological response to stress and thus may occur in response to a wide variety of stimuli, including exercise, anxiety, fever, hypotension, hypoxemia, anemia and medications. Since Sinus Tachycardia is an automatic tachycardia, it is not treated with electrical cardioversion. Rather, any treatment is usually directed at the underlying cause.

<table>
<thead>
<tr>
<th>Key Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Narrow, regular SVT</td>
</tr>
<tr>
<td>• Automatic mechanism</td>
</tr>
<tr>
<td>• Upright P wave Lead II</td>
</tr>
<tr>
<td>• P before each QRS</td>
</tr>
<tr>
<td>• QRS for every P wave</td>
</tr>
<tr>
<td>• Rate &gt; 100 bpm</td>
</tr>
<tr>
<td>• Max ~ (220-age)</td>
</tr>
</tbody>
</table>

**Figure 4.** Sinus Tachycardia
(Ectopic) Atrial Tachycardia

Atrial Tachycardia is often called Ectopic Atrial Tachycardia (EAT). It is due to increased automaticity from a single ectopic atrial focus. Short paroxysms of EAT are common in normal subjects but in some patients recurrent or sustained EAT may cause symptoms and require treatment. Since EAT is an automatic tachycardia, it is not treated with electrical cardioversion. Antiarrhythmic drugs may sometimes be effective. Since the focus is not from the Sinus Node, the P waves are usually inverted in Lead II.

Key Features

- Narrow, regular SVT
- Automatic mechanism
- Negative P wave Lead II
- P before each QRS
- Rate ~ 100 - 240
- With very fast rates, 2:1 AV block may be present

Figure 5. Ectopic Atrial Tachycardia
Paroxysmal Supraventricular Tachycardia (PSVT)
- AV Nodal Reentrant Tachycardia (AVNRT)

Paroxysmal Supraventricular Tachycardia (PSVT) is a general term applied to several related reentrant tachycardias that are characterized by sudden onset and termination. AV Nodal Reentrant Tachycardia (AVNRT) and Atrioventricular Reentrant Tachycardia (AVRT) are the most common forms, accounting for ~ 90% of “PSVT”. In practice, it is sometimes difficult or impossible to determine the exact type of PSVT from the 12-lead ECG. However, you should be aware of the theoretical differences between AVNRT and AVRT and the general differences between PSVTs and other narrow complex tachycardias. Both types of PSVT involve the AV node and thus are sensitive to vagal maneuvers such as carotid sinus massage (CSM) and medications that act on the AV Node (e.g. Beta blockers, calcium channel blockers, digoxin, adenosine). Both may also be terminated using DC electrical cardioversion or radiofrequency catheter ablation.

**AV Nodal Reentrant Tachycardia (AVNRT)**

![Diagram of AV Nodal Reentrant Tachycardia](image)

**Key Features**

- Narrow, regular SVT
- Reentry mechanism
- P waves often “buried” in the QRS or barely visible after QRS
- Rate ~ 150 - 250
- Terminate with vagal maneuvers (e.g. CSM)

---

**Figure 6.** The top panel shows AV Nodal Reentrant Tachycardia (AVNRT). Note the small inverted P waves appearing at the end of the QRS. The bottom panel is the same patient after carotid sinus massage. Note that the P wave now appears upright and before the QRS, and there is no longer an inverted P wave at the end of the QRS.
Paroxysmal Supraventricular Tachycardia (PSVT)
- AV Reentrant Tachycardia (AVRT)

Atrioventricular Reentrant Tachycardia (AVRT) differs from AVNRT in that one limb of the reentrant loop is the AV node and the other is an accessory pathway. An accessory pathway, sometimes called a “bypass tract” is an abnormal band of myocardial tissue that connects atrial and ventricular tissue and permits electrical conduction. During normal sinus rhythm, the ventricle is partially activated via the bypass tract. This is often called preexitation. Since conduction in the bypass tract is faster than in the AV node, the PR interval is short, there is slurring of the initial portion of the QRS (called a delta wave) and the QRS is slightly widened. This constellation of findings is called Wolff-Parkinson-White (WPW). Patients with WPW are predisposed to SVT. During tachycardia, activation usually is retrograde up the bypass tract and antegrade in the AV Node, and thus no delta wave is seen.

Atrioventricular Reentrant Tachycardia (AVRT)

![Diagram of AVRT](image)

**Key Features**
- Narrow, regular SVT
- Reentry mechanism
- Retrograde P waves often follow the QRS
- Rate ~ 150 - 250
- Terminate with vagal maneuvers (e.g. CSM)

*Figure 7.* An example of AVRT in a patient with Wolff-Parkinson-White (WPW). In the top panel the patient is in normal sinus rhythm. The PR interval is at the lower limit of normal and there is slurring of the upstroke of the QRS (delta wave) indicating “pre-exitation” of the ventricle via the bypass tract. In the bottom panel, the patient has AVRT and there is a more narrow QRS with no delta wave since the ventricle is activated via the normal conduction pathway.
Atrial Flutter

Atrial Flutter is usually due to a single large reentrant loop involving a relatively large portion of the right atrium. As a result of involvement of a large portion of the atria, prominent “flutter” waves are characteristically seen. In the inferior leads (II, III, aVF), this gives the ECG often has a “sawtooth” appearance. Atrial flutter is usually seen in patients with structural heart disease. While the atrial rate is usually high (220-300), there is usually some degree of block in the AV node (e.g. 2:1, 3:1, 4:1), and thus the ventricular rate is less. CSM and medications that act on the AV Node usually increase the degree of block (and thus reduce the ventricular rate), but do not terminate the arrhythmia. Atrial Flutter can be terminated with DC cardioversion and can be cured with radiofrequency catheter ablation.

Figure 8. Shown is an example of atrial flutter. Notice that the classic “sawtooth” flutter waves are seen better in some leads (usually best in II, III or aVF) and not as well in other leads. In this patient, the atrial rate is approximately 260 while the ventricular rate is approximately 65, indicating that every 4th flutter beat is conducted to the ventricle. You would read this as “Atrial Flutter with 4:1 block”.

Key Features

- Narrow, regular SVT
- Reentry mechanism
- “Sawtooth” flutter waves
- 2:1, 3:1, 4:1 AV block
- Atrial Rate 220-300
- 2:1, 3:1, 4:1 AV block
- CSM increases AV Block
Sinus Tachycardia with Premature Atrial Complexes

Sinus tachycardia alone is considered a “regular” tachycardia. However, when premature atrial complexes (PAC) are also present, the rhythm may be “irregular”. PACs are due to increased automaticity from a site other than the SA node and therefore the PAC P waves may look different than the sinus P waves. PACs are generally harmless and not treated, but occasionally may cause palpitations or may initiate reentrant SVTs.

Figure 9. Sinus tachycardia with PACs. The 2nd, 6th, 9th and 12th QRS complexes are preceded by PACs. The “early” P waves are best seen in the V1 rhythm strip. In the Lead II and V5 rhythm strips, the PACs merge with the preceding T wave and more difficult to distinguish.

Key Features

- Narrow, irregular SVT
- Automatic mechanism
- Most beats have upright P wave Lead II
- One dominant pacemaker
- P before each QRS
- Rate > 100 bpm
- Max ~ (220-age)
Multifocal Atrial Tachycardia (MAT)

Multifocal Atrial Tachycardia is due to increased automaticity from 3 or more atrial foci. It is most often seen in the setting of severe pulmonary disease and hypoxemia and is not responsive to DC cardioversion. Treatment is directed at the underlying medical conditions. It is characterized on the ECG by 3 or more P wave morphologies and variable PR, PP and RR intervals. The rate is usually 100-150 but can be as high as 250. When the HR is less than 100, it is called Wandering Atrial Pacemaker (WAP).

**Key Features**

- Narrow, irregular SVT
- Automatic mechanism
- 3 or more P wave morphologies
- Variable PR, PP, RR intervals
- Rate ~ 100 – 150 (rarely to 250)
- When HR < 100, called WAP
- Strong association with severe pulmonary disease

**Figure 10.** Continuous V1 rhythm strip showing Multifocal Atrial Tachycardia (MAT). Note that there are at least 3 different P wave morphologies, varying P-P, PR and R-R intervals.
Atrial Flutter with Variable Block

Atrial Flutter may be associated with regular conduction through the AV node or at times the conduction may vary at irregular intervals. In the latter case, the conduction pattern is called “variable block”. The presence of variable block does not have any specific clinical implication, except that the rhythm is “irregular” and therefore may be confused with one of the irregular SVTs.

**Figure 11.** Atrial flutter with variable block. The flutter waves are regular at a rate of approximately 300 beats per minute while the QRS complexes occur at irregular intervals due to varying degrees of block at the AV node. In this example, the classic “sawtooth” pattern is again best seen in Lead II and are easiest to see when the QRS complexes are more widely spaced near the end of the tracing.
Atrial Fibrillation (AF)

Multiple reentry circuits within the atria cause Atrial Fibrillation (AF). The AV node is thus bombarded by irregular impulses of which only a portion can be conducted. Thus the resulting ventricular rate (often called ventricular “response” is irregularly irregular (no pattern). AF may be paroxysmal or permanent and is most commonly associated with hypertension and/or heart failure, but can be seen in otherwise normal individuals. A very fast ventricular response may cause hemodynamic deterioration in patients with heart failure. The absence of atrial contraction predisposes to the blood stasis and intraatrial thrombus that may lead to systemic emboli including stroke. The rhythm may be terminated using DC cardioversion. Medications that slow AV node conduction are used to control the ventricular rate. Most patients with AF require treatment with warfarin or aspirin to reduce the likelihood of stroke.

Figure 12. Atrial Fibrillation. Note the chaotic baseline and the “irregularly” irregular rhythm.
Summary of Approach to Narrow Complex tachycardia

Regular Rhythm

And the P waves are

<table>
<thead>
<tr>
<th>Normal</th>
<th>Inverted in Lead II</th>
<th>Sawtooth</th>
<th>Absent/retrograde</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus</td>
<td>(Ectopic) atrial</td>
<td>Atrial</td>
<td>“PSVT”</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>Tachycardia</td>
<td>Flutter</td>
<td>AVNRT or AVRT</td>
</tr>
</tbody>
</table>

Irregular Rhythm

And the P waves are

<table>
<thead>
<tr>
<th>Mostly Normal</th>
<th>3 or more types</th>
<th>Sawtooth</th>
<th>Absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 3 types</td>
<td></td>
<td></td>
<td>Chaotic baseline</td>
</tr>
<tr>
<td>Sinus Tachycardia</td>
<td>Multifocal atrial Tachycardia (MAT)</td>
<td>Atrial Flutter</td>
<td>Atrial Fibrillation</td>
</tr>
</tbody>
</table>
We have now completed our discussion of the “Narrow-Complex” tachycardias and are ready to discuss the “Wide-Complex” Tachycardias (WCT).

There are two general mechanisms for WCT: Ventricular Tachycardia (VT) and Supraventricular Tachycardia (SVT) with aberrant conduction. Sometimes it is easy to tell the difference between VT and SVT with aberrancy, especially when the ventricular rate is relatively slow and it is easy to see atrial activity. However, there are times when it is very difficult to distinguish between these two possibilities and experienced ECG readers rely on a number of ECG criteria to help make the correct diagnosis. The ECG criteria that help to diagnose WCT in difficult situations are listed in an appendix at the end of this chapter. You are not expected to know these criteria for HuBio 540. They are included for reference purposes during your clinical years. One general principal that will serve you well in caring for patients with WCT is that when the patient has known coronary artery disease or heart failure, assume that a WCT is ventricular tachycardia until proven otherwise.

Figure 13. The Tachycardias
Supraventricular Tachycardia with Aberrant Conduction

A wide QRS may accompany any of the SVTs discussed above. A simple example is a patient with SVT and Right Bundle Branch Block. Sometimes the patient may have abnormal conduction at baseline, but sometimes it is the presence of tachycardia that exposes conduction abnormalities. When this happens you may hear the term *Rate-Related Bundle Branch Block*. This simply refers to the fact that the conduction system is able to conduct normally at relatively low heart rates but that when the heart rate is relatively fast, a bundle-branch block occurs. Following is an interesting example of SVT with aberrancy.

Figure 14. Examples of Supraventricular Tachycardia (SVT) with aberrant conduction. In the top panel is shown the tracing from a patient who presented to the emergency room with a very fast wide-complex tachycardia. The patient was given a beta blocker and a few minutes later the middle tracing was obtained showing a now narrow QRS. With further treatment, the rate slowed even more allowing flutter waves to be seen (best in lead II). In the top tracing, the patient had atrial flutter with 1:1 conduction to the ventricles and the depolarization pattern is aberrant. In the middle tracing the block is now 2:1 and the slower ventricular rate allows a normal ventricular activation pattern.
Premature Ventricular Complexes (PVC)

Premature Ventricular Complexes (PVCs) are caused by increased automaticity of an ectopic focus within the ventricle. Recognized as “early” wide QRS complexes that are not associated with a preceding P wave, PVCs are wide since ventricular activation does not occur via the specialized conduction system. Usually, though not always there is a *compensatory pause* following PVCs, which means that the PP interval that contains the PVC is twice the normal sinus PP interval. PVCs are common in patients with no heart disease, do not impose increased risk and generally require no treatment. In patients with heart disease, PVCs alone are not harmful, but very frequent PVCs may indicate an increased risk of more serious ventricular arrhythmias such as Ventricular Tachycardia (discussed below). Two or three successive PVCs are called a ventricular *Couplet* and *Triplet* respectively. When every other beat, or every third beat is a PVC, the rhythm is called ventricular *Ventricular Bigeminy* or *Ventricular Trigeminy* respectively.

**Figure 15.** Examples of Premature Ventricular Complexes (PVC). In the top tracing every other beat (starting with the first) is a PVC. Note the absence of a P wave and the widened QRS. This rhythm, in which every second beat is a PVC is called ventricular bigeminy. In the bottom tracing, there are two PVCs in a row. This is referred to as a ventricular couplet.

<table>
<thead>
<tr>
<th>Key Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Wide “early” QRS complex</td>
</tr>
<tr>
<td>• No associated P wave</td>
</tr>
<tr>
<td>• Compensatory pause</td>
</tr>
<tr>
<td>• 2 in a row = couplet</td>
</tr>
<tr>
<td>• 3 in a row = triplet</td>
</tr>
<tr>
<td>• Every other = bigeminy</td>
</tr>
<tr>
<td>• Every third = trigeminy</td>
</tr>
</tbody>
</table>
Ventricular Tachycardia (VT)

Ventricular tachycardia (VT) is defined as 3 or more PVCs in succession and typically is due to a reentry mechanism within the ventricle. Arbitrarily, when the rhythm lasts for less than 30 sec. it is called Non-sustained VT and if greater than 30 sec it is called Sustained VT. When all the QRS complexes look the same and the rate is regular, the VT is called Monomorphic VT and when the QRS complexes look different and the rate varies, the VT is called Polymorphic VT. VT is usually seen in patients with underlying structural heart disease but may also occasionally occur as a result of electrolyte abnormalities, certain medications or QT prolongation. Patients with VT may or may not experience symptoms such as palpitations or syncope (loss of consciousness). VT may also degenerate into Ventricular Fibrillation (VF, see below). VT is a shockable rhythm and this is the preferred therapy in the acute setting. Patients with symptomatic or repeated episodes of sustained VT are usually treated with antiarrhythmic medications (e.g. amiodarone) and/or with an implantable cardioverter defibrillator (ICD).

**Figure 16. Ventricular Tachycardia**

**Key Features**

- Wide QRS complex
- Reentry mechanism
- Regular or irregular rate
- Typically 140-200 bpm

- Sustained = > 30 sec
- Non-sustained = < 30 sec

- Monomorphic vs. Polymorphic
- Mimicked by SVT with aberrancy
Ventricular Fibrillation (VF)

Ventricular Fibrillation (VF) is a life-threatening arrhythmia that is the leading cause of sudden cardiac death (SCD). The ECG is characterized by chaotic and irregular deflections of variable size and morphology. There is an absence of distinct P waves, QRS complexes and T waves. The only effective treatment is DC electrical cardioversion that is very effective when performed within the first minute. When cardioversion is delayed for 4-5 minutes, it is effective in only about 25% of patients. Recognition of the time-dependency of effective cardioversion has lead to the development of the Automatic External Defibrillator (AED) as well as the Implantable Cardioverter Defibrillator (ICD). The AED is a small portable defibrillator that automatically gives verbal instructions to the user when the case is opened. Once electrode pads are placed on the victim’s chest, the rhythm is analyzed and a shock is delivered automatically when appropriate. Increasingly, AEDs are being deployed in public places such as airlines and casinos. ICDs are small pacemaker-like devices that are implanted subcutaneously and with wires inserted into the subendocardium of the right ventricle.

### Ventricular Fibrillation (VF)

**Key Features**

- Very rapid chaotic rhythm
- Leading cause of SCD
- No discernable P, QRS, T
- Lethal without DC cardioversion
- May be mimicked by artifact – Check the patient!

- Take a class in Basic and Advanced Life Support and Learn to use an AED!

---

**Figure 17.** Ventricular Fibrillation (VF)
Bullet Point Review

Tachycardia Classification Schemes

• Location of origin: Supraventricular (SVT) vs. Ventricular (VT)
• Mechanism: Automatic (*) vs. Reentry ®
• Width of QRS: Wide-Complex vs. Narrow-Complex

Clinical Approach to Diagnosis

♥ Question 1: Is the QRS wide or Narrow?
• Narrow QRS = SVT
• Wide QRS = VT or SVT with aberrant conduction

Clinical Approach to Narrow QRS Tachycardia (SVT) diagnosis

♥ Question 2: Is the rhythm regular or irregular?

Regular SVT

• Sinus Tachycardia * Upright P waves in Lead II
• (Ectopic) Atrial Tachycardia* Inverted P wave in Lead II
• Paroxysmal Supraventricular Tachycardia
  • AVNRT® P waves often “buried” in the QRS – response to CSM
  • AVRT® Retrograde P waves follow the QRS – response to CSM
• Atrial Flutter® Look for “sawtooth” flutter waves in the inferior leads

Irregular SVT

• Sinus Tachycardia with PACs* One dominant P wave morphology
• Multifocal Atrial Tachycardia (MAT) * 3 or more P wave morphologies
• Atrial Flutter with variable block®
• Atrial Fibrillation® No P waves, chaotic baseline oscillations

Clinical Approach to Wide QRS Tachycardia (SVT or VT) diagnosis

• It is often difficult to distinguish SVT with aberrancy from VT
• For more information on the differentiation of VT vs. SVT, see appendix B
• Patients with CAD or Heart Failure: Think VT first.
• VF needs to be shocked quickly
Appendix B. Differentiation of VT from SVT with Aberrant Conduction

A tachycardia with a QRS duration of 0.12 sec or longer may represent Ventricular Tachycardia (VT) or Supraventricular Tachycardia (SVT) with aberrant conduction. The latter may occur when SVT occurs in the setting of a new or previous bundle branch block or, less commonly, when the SVT is propagated to the ventricles via an accessory pathway. Clinically, it may be difficult or impossible to distinguish between VT and SVT with aberrancy with absolute certainty using the 12-lead ECG alone. However, several features of the ECG may provide clues in difficult cases. These are outlined in the table below. Remember that when the patient has known CAD or heart failure, you should think VT first. The following material is not required reading for HuBio 540.

<table>
<thead>
<tr>
<th>ECG finding</th>
<th>Favors VT</th>
<th>Favors SVT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tachycardia initiated by PAC</td>
<td></td>
<td>*</td>
</tr>
<tr>
<td>QRS morphology similar to PVC</td>
<td>*</td>
<td></td>
</tr>
<tr>
<td>QRS morphology similar to preexisting RBBB or LBBB</td>
<td></td>
<td>*</td>
</tr>
<tr>
<td>AV dissociation (1) present</td>
<td>*</td>
<td></td>
</tr>
<tr>
<td>Precordial concordance (2) present</td>
<td>*</td>
<td></td>
</tr>
<tr>
<td>QRS axis left or right superior</td>
<td>*</td>
<td></td>
</tr>
<tr>
<td>QRS duration &lt; 0.14 sec</td>
<td></td>
<td>*</td>
</tr>
<tr>
<td>Response to vagal maneuvers</td>
<td></td>
<td>*</td>
</tr>
<tr>
<td>RSR’ pattern in lead V1</td>
<td>R taller than R’</td>
<td>R’ taller than R</td>
</tr>
</tbody>
</table>

(1) AV dissociation refers to when there are P waves and QRS complexes on the tracing but there is no temporal association between them. The finding of AV dissociation implies the existence of a ventricular focus that is generating QRS complexes independently from any concurrent atrial activity. When P waves are not clearly identified on the tracing, AV dissociation may be inferred from the finding of a capture beat (a narrow QRS complex standing alone in a run of wide complexes) or a fusion beat (a QRS which standing alone in a run of wide complexes which does not look like the other wide beats, but also does not resemble the normal sinus beat.

(2) Precordial concordance refers to the finding that all QRS complexes in Leads V1-V6 are either positive or negative

Back to Student Page