CHAPTER 1

Basic electrophysiology

For our discussion, electrophysiology is a general term used to describe the “electrical” characteristics of the heart. The fundamental diagnostic tools used by electrophysiologists are small catheters that are placed in the heart most commonly via the vascular system. The catheters are essentially plastic-coated wires with metal electrodes at the tip that have two functions: measurement of the electrical activity of the heart and provision of a conductive pathway to allow stimulation of the heart. Often, multiple catheters are placed in different parts of the heart, which when evaluated together can provide clues into possible mechanisms for bradycardia or tachycardia in an individual patient. Bradycardia is a far less common reason for performing an electrophysiology study and is often evaluated in the context of a patient with syncope (Chapter 26).

Most commonly, electrophysiology studies are often performed in patients with known tachycardia or patients who are at risk for tachycardia. Practical use and performance of the electrophysiology study in the evaluation of tachycardias will be the focus of all but one of the subsequent cases. Before we go through the cases, it is instructive to review the basic methods for classifying tachycardias. Clinically, tachycardias are usually classified by QRS complex width, because a narrow QRS complex suggests that the ventricles are being activated normally, while a wide QRS complex is a sign of abnormal activation of the ventricles. Although supraventricular tachycardias can be associated with profound symptoms such as shortness of breath or chest pain, they are generally not life-threatening. An electrical impulse (generally an early beat) conducts “down” only one pathway and is able to enter the other pathway in a retrograde direction. The most well-studied example of this phenomenon are tachycardias that use an accessory pathway (Figure 1.1). In this case, the accessory pathway and the atrioventricular (AV) node provide an anatomically separate connections between the atria and the ventricles. The accessory pathway and the AV node/His bundle axis are anatomically discrete, and the atria and the ventricles are electrically separated by the mitral and tricuspid annuli. Reentry can also occur in the setting of a scar that has a slowly conducting pathway through it. In this case, the slowly conducting pathway and normal tissue represent the two pathways that form the substrate for development of reentry. Traditionally, scar is described in ventricular tissue because of a myocardial infarction but could also be present in atrial tissue in the setting of atrial fibrosis from natural processes or prior ablations. It is not surprising that reentry generally is more common around “holes”
Figure 1.1 For most types of reentry, two separate pathways have different electrophysiologic properties, and with a carefully timed impulse, depolarization can occur in one pathway and “turn around” to depolarize the parallel pathway. The most well-described and the best clinical example of this is a patient with an accessory pathway (a). Normally, the AV node forms the only electrical connection between the atria and the ventricles, but in patients with an accessory pathway, the second electrical connection between the atria and ventricles can allow a reentrant circuit to develop. Another common scenario for reentrant arrhythmias is ventricular tachycardia in the setting of a prior myocardial infarction (b). In this case, a “patchy” myocardial scar forms an alternate pathway along with normal myocardium to activate one side of a scar to the other side of the scar.

in the heart, because these “holes” increase the likelihood that two separate pathways of electrical activation exist. For example, in the most common form of atrial flutter, the reentrant circuit uses a critical isthmus formed by the inferior vena cava and the tricuspid valve. The inferior vena cava and the tricuspid valve act as natural barriers that allow perpetuation of a reentrant circuit. In this case, the inferior cavitricuspid isthmus serves as one pathway, and the superior portion of the right atrium serves as the second pathway (Figure 1.2).

The second type of tachycardia is a focal source of rapid activation. This tachycardia mechanism is easier to conceptualize and can be thought of as outward spreading of electrical activation similar to a series of waves from repetitive drips into a pool of still water. Often, rapid activation is due to abnormal automaticity from a nest of cells, but in some cases, very small reentrant circuits can appear as focal activation. This type of reentry is often called microreentry to differentiate it from larger circuits of reentry that are called

Figure 1.2 Initiation of typical atrial flutter. (a): In sinus rhythm (*), atrial depolarization proceeds down the lateral wall of the right atrium (RA) and superiorly toward the septum. (b): With a premature atrial contraction from the left atrium (*), inferior atrial depolarization blocks in the cavitricuspid isthmus (CTI), but the wave of depolarization travels superiorly to activate the superior and lateral portions of the right atrium and enters the CTI from the other direction. (c): Slow conduction through the CTI initiates atrial flutter.
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Macroreentry. The actual reentrant circuit cannot be visualized unless specialized techniques are used, and often, the reentrant mechanism must be inferred by the behavior of the tachycardia. For example, termination of a tachycardia with a premature extrastimulus suggests the possibility of reentry. While reentrant circuits can be extinguished by a single early depolarization causing refractoriness, a tachycardia due to automaticity generally does not terminate, instead the usual response is transient suppression of the tachycardia.

The reason that tachycardia mechanism is so important is that it will dictate the therapeutic plan, particularly if ablation will be an option. In patients with reentry, effective ablation targets a critical pathway, isthmus, or “channel” that is essential for maintaining the tachycardia (often called “substrate”). Successful ablation is characterized by producing conduction block that can be thought of as a “dam” or a “wall” that prevents electrical current from passing. In patients with a local source of tachycardia successful ablation targets the specific “source” of the arrhythmia.

Anatomic tachycardia classification

In addition to mechanism, it is also important from an electrophysiologic standpoint to consider tachycardia using an anatomic classification. Obviously, considering the anatomic location of the tachycardia is important to effectively treat the arrhythmia with ablation. A comprehensive review of different types of tachycardia is discussed in Understanding Intracardiac EGMs and ECGs (Wiley Blackwell 2010) and is beyond the scope of this case-based book. However, a cursory review at this point is reasonable. In general terms, there are only four basic anatomic locations for tachycardia (Figure 1.3).

Firstly, a tachycardia can arise solely within the atrium. This could be due to a stable reentrant circuit within the atrium, continuous irregular activation of the atrium (perhaps due to unstable reentrant circuits), or one or more abnormal foci of automaticity. The way to consider this is that in atrial flutter or atrial fibrillation, there is activation of the atrium somewhere all the time. In contrast, in focal or multifocal atrial tachycardias, a site(s) leads to depolarization of the atria, and the atria are quiescent until the next depolarization. Perhaps, another analogy distinguishing between reentry and automaticity is useful. Automaticity or repetitive focal firing is similar to a blinking light(s): when the light goes “off” there is no light. Reentry can be compared to the spinning earth, where as the earth spins, different areas of the earth are in the dark and “lights are turned on.” In the latter case, there is always somewhere where the lights are on (the Pacific Ocean, with very few lights from ships and islands could be thought of as a region of “slow conduction”). Regardless of whether automaticity or reentry is the cause of the arrhythmia, the entire abnormal focus or circuit is located within atrial tissue. To further confuse things, all of these arrhythmias are often collectively called atrial tachycardias. The second “anatomic” type of tachycardia is an abnormal focus or small reentrant circuit localized within the AV junction. Although the most common cause of tachycardia arising from the junction is due to small reentrant circuits within the AV node and perinodal atrial tissues, rapid automatic rhythms can be observed from the AV junction in certain situations. The most commonly encountered automatic junctional tachycardia is called nonparoxysmal junctional tachycardia (NPJT) and can be seen after cardiac surgery, particularly surgeries that involve the aortic or mitral valve. An example of NPJT is shown in Figure 1.4. Notice the AV dissociation and underlying sinus bradycardia. In NPJT, AV conduction is intact, and properly timed P wave will conduct to the ventricles and result in a shorter R-R interval or a subtle change in the QRS morphology as shown in this example. In this case, a “blinking light” in the AV junction is competing with a slower “blinking light” in the sinus node. The third anatomic classification of tachycardia arises solely from ventricular tissue and includes ventricular tachycardia and ventricular fibrillation (again differentiated by whether there is relatively repetitive depolarization of ventricular tissue vs irregular chaotic depolarization of ventricular tissue). The fourth anatomical classification is a tachycardia that uses an accessory pathway. Tachycardias using accessory pathways will be the subject of several of the cases in this book, and the reader should refer back to Figure 1.3 as required. Identification of the tachycardia mechanism (reentry vs automaticity) and the critical anatomic sites necessary for initiating or for sustaining tachycardia is the principal goal of electrophysiology studies and will be the principal subject for all of the cases covered throughout this book.
Figure 1.3 Anatomic classification of tachycardias (adapted with permission from Kusumoto F, Cardiovascular Pathophysiology, Harris Barton Press 1999).

**Electrophysiology basics**

From a simplistic standpoint, an electrophysiology study involves measuring the baseline electrical characteristics of the heart, “stressing” the heart by pacing, and then evaluating whatever arrhythmias are induced by the “stress.” Once catheters are in position, all electrophysiology studies begin by measuring baseline parameters. Even in an electrophysiology study,
Figure 1.4 Patient with an automatic tachycardia from the AV node region observed in the first few hours after aortic valve surgery. This arrhythmia, called nonparoxysmal junctional tachycardia (NPJT), usually resolves after 1 or 2 days. Intermittent intrinsic AV conduction can sometimes be observed when a properly timed P wave results in an early QRS complex or a subtle change in the QRS morphology (*).

standard electrocardiogram (ECG) measurements such as the PR interval, QRS width, and QT interval are important to document. It is important to reemphasize that intracardiac recordings are simply electrodes within the heart and simply provide more accurate and complete evaluation of timing and direction of depolarization for both atrial and ventricular tissues.

For example, we infer that the sinus node is driving the atrium by the shape of the P wave from the surface ECG. If the P wave is negative in lead AVR and positive in lead II, we know that the atria are being depolarized from right to left, and “High-Low.” However, by placing electrodes in different regions of the atria, we can confirm this by direct recording of electrical activity. For most electrophysiology studies, catheters will be placed in the high right atrium (HRA), straddling the tricuspid valve, in the right ventricle, and in the coronary sinus (Figure 1.5 and Table A2 in Appendix). These initial catheter positions are chosen because they all can be placed via venous puncture (generally the two femoral veins, although the internal jugular and subclavian veins can also be used), and together they can evaluate electrical activity in the critical regions of the heart. The HRA is near the location of the sinus node; the right septal region straddles the tricuspid valve and allows identification of septal atrial activation. His bundle depolarization, and depolarization of the septal portion of the right ventricle, and the right ventricle records depolarization of the right ventricular apex. The coronary sinus travels in or near the left AV groove separating the left atrium and the left ventricle and is accessible from the right atrium via the coronary sinus os located in the inferior portion of the right atrial septum near the tricuspid valve, an additional catheter is often placed here when evaluation of left atrial and left ventricle depolarization would be helpful.

Baseline electrograms from a patient undergoing an electrophysiology study for supraventricular tachycardia are shown in Figure 1.6. The general strategy for evaluating electrograms is shown in Figure 1.7. Although laboratories vary, all will generally place several ECG recordings at the top of the page. In our laboratory, we usually use three leads – I, II, and V1. This set provides a lateral lead (lead I), an inferior lead (lead II), and a
Figure 1.5 Basic catheter positions for electrophysiology study in the right anterior oblique (RAO) and the left anterior oblique (LAO) projections. Quadripolar catheters are located in the right atrium (RA), His bundle region (His) straddling the tricuspid valve, and the right ventricle (RV). A decapolar catheter is placed in the coronary sinus (CS). For orientation, the approximate locations of the mitral valve (MV), tricuspid valve (TV), inferior vena cava (IVC), and superior vena cava (SVC) are shown (reprinted with permission from Kusumoto FM, Understanding EGMs and ECGs, Wiley 2010).

Figure 1.6 Flow sheet for the evaluation of electrograms during a typical electrophysiology study.

Assess the ECG
What leads? What is the basic rhythm?

Initial EGM evaluation
What EGMs have been provided? What is the orientation?

Is anything being done?
Atrial Pacing Ventricular Pacing

EGM evaluation
Atrioventricular relationship Pattern of atrial depolarization Pattern of ventricular depolarization

precordial lead (lead V1). We use lead II for the inferior lead, as the P waves are generally most easily seen in this lead particularly during sinus rhythm, and lead V1 as our precordial lead, as the QRS morphology in the setting of bundle branch block has been very well described. The ECG leads are placed “on top” because they serve as basic landmarks and should always be the first signals to be assessed when evaluating intracardiac electrograms.

Electrograms are usually grouped by catheter and by personal preference. In Figure 1.6, atrial signals are placed above ventricular signals, right-sided structures above left-sided structures, and with distal electrodes “on top” and proximal electrodes “below.” This setup is personal preference and dates back to my initial training at the University of California, San Francisco. Preferences for electrogram display will vary from operator to operator usually depending on training (you can often guess where an electrophysiologist trained by their electrogram display and their approach to electrophysiology studies). How many electrograms displayed for a particular catheter will be dependent on the number of electrodes on a specific catheter. Traditionally, quadripolar catheters with four electrodes have been used, and recordings between the distal electrode pairs and the proximal pairs are depolarized. If a catheter has more electrodes, then more electrogram pairs can be displayed. All of this emphasizes that once the ECG is evaluated, the next step in evaluating electrograms is to first look at the anatomic location and
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Figure 1.7 Electrograms recorded from catheter positions in Figure 1.5. The AV relationship is 1:1 with atrial activation (both P waves and atrial EGMs) preceding or "driving" ventricular activation (both QRS complexes and ventricular EGMs). Atrial activation is seen first in the high right atrium (HRA), followed by the His bundle, and last in the coronary sinus (vertical arrows). A His bundle deflection (H) and a right bundle (RB) potential can be seen during the isoelectric period of the PR interval. AV conduction is usually divided into two parts: the AH interval (first septal atrial activation to the His deflection) that represents conduction through the AV node and the HV interval (His deflection to first ventricular depolarization) that represents His bundle, bundle branch, and Purkinje fiber depolarization. Ventricular (V) activation is seen first in catheters located in the septum (His and RV) and later in the coronary sinus.

In our patient, the basic cycle length is 775 ms, and the PR interval is normal (160 ms), with the QRS interval being normal as well (106 ms). The electrograms from four catheters are provided, from superior to inferior: the HRA, the His bundle, the coronary sinus, and the right ventricle. The electrodes are recorded as discrete pairs except for the His bundle where overlapping signals from the four electrodes are recorded – distal: first and second; mid: second and third; proximal: third and fourth. There is a 1:1 relationship between atrial and ventricular electrograms (as would be predicted by the normal PR interval on the surface ECG). Closer inspection of the atrial electrograms shows that atrial depolarization occurs from right to left and superior to inferior (first in the HRA, then the His, and last atrial signals in the left atrium). After atrial depolarization, the sharp deflection of the His bundle can be seen followed by a sharp deflection from the right bundle.

Ventricular activation is first identified in the septum (the His and RV catheters) and later in the left ventricle (low amplitude potentials recorded in the coronary sinus catheter). As mentioned previously, the coronary sinus straddles the left atrium and left ventricle, so electrograms from both structures are recorded, but generally the atrial signal is of higher amplitude and higher frequency ("sharp and spikey").

For AV conduction via the AV node and His Purkinje system, the AH interval (measured from the atrial electrogram to the His bundle electrogram) represents conduction time through the AV node proper, and the HV interval (measured from the His bundle to first sign of ventricular depolarization) represents depolarization of the His bundle, the right and left bundles, and the Purkinje fibers. This is the reason why the right bundle potential is located within the HV interval. As the AV node conduction is slow and has innervation from the autonomic nervous system, the normal range for the AH interval is quite wide from 50 to 140 ms.
The HV interval measures conduction via the His Purkinje system, and because these structures have rapid conduction properties and do not have significant innervation by the autonomic system, the normal range is shorter with less variation (35–55 ms).

Baseline atrial pacing

Atrial pacing is performed for the evaluation of any changes in the QRS complex with higher atrial rates and of AV conduction and for the induction of tachycardia. There are two forms of atrial pacing: (i) atrial overdrive pacing where the atria are paced from a single location at progressively faster rates and (ii) delivery of premature extrastimuli, where after consistent capture of the atria occurs (usually 8 beats), one, two, or three premature atrial stimuli are introduced.

Atrial overdrive pacing is shown in Figures 1.8 and 1.9. Normally, as the atria are paced at progressively faster and faster rates, delay begins to develop in the AV node (think of the AV node as a “regulator” that limits ventricular depolarization; this characteristic is important for “protecting” the ventricle from rapid stimulation in the setting of rapid atrial activity, e.g., in atrial fibrillation, ventricular depolarization, although fast, is often limited to 120–180 beats per minute depending on the patient’s age). The cycle length in which 1:1 AV conduction stops (in other words there are more “A’s” than “V’s”) is called the AV blocked cycle length (AVBCL). The AVBCL is a measure for AV conduction, with a shorter AVBCL associated with more “slick” AV node conduction.

In patients with an accessory pathway, delay in the AV node due to atrial pacing can lead to more of the ventricles being depolarized by the accessory pathway. Delay in the AV node is characterized by prolongation of the AH interval, and more ventricular depolarization due to the accessory pathway leads to a more abnormal-looking QRS complex. We will discuss these changes extensively in the cases that involve patients with accessory pathways.

For premature atrial extrastimuli, after a drive of 8–10 stimuli at a constant drive cycle length, a premature atrial stimulus is delivered. This premature beat is programmed to earlier and earlier values, and the response of the AV node and His bundle are assessed. The usual response is shown in Figures 1.10 and 1.11. In Figure 1.10, after pacing
Figure 1.9 More rapid atrial pacing leads to AV Wenckebach behavior. Pacing associated with gradual prolongation of the AH interval is observed until a dropped “H” and QRS complex. This response is characteristic of block within the AV node.

Figure 1.10 After a basic cycle length of 600 ms, a premature atrial stimulus at a coupling interval of 300 ms, the AH interval prolongs to 200 ms because of delayed conduction in the AV node.
from the HRA at a basic cycle length of 600 ms, a premature extrastimulus at a coupling interval of 300 ms is delivered. In response to this premature atrial stimulus, there is prolongation of the AH interval due to delay within the AV node. In Figure 1.11, when the coupling interval is shortened to 250 ms, an atrial electrogram is observed, but there is no subsequent His bundle recording and QRS because of block in the AV node. The AV node effective refractory period (AVNERP) would be defined as 250 ms (usually measured as the interval between atrial signals in the His catheter).

Sometimes, atrial pacing will induce an arrhythmia of interest. Initiation of different arrhythmias with atrial pacing will be described in detail in later chapters.

**Baseline ventricular pacing**

Just as in atrial pacing, ventricular pacing is performed by pacing at shorter and shorter cycle lengths and assessing retrograde VA conduction (ventricular overdrive pacing) or by delivering earlier premature ventricular beats (premature ventricular stimulation). In Figure 1.12, baseline ventricular pacing at a cycle length of 500 ms is not associated with VA conduction. The atria continue to be depolarized by the sinus node with first atrial activation recorded in the HRA. Figure 1.13 shows the response to ventricular pacing in the setting of sympathetic activation with isoproterenol. Isoproterenol is a synthetic amine related to epinephrine but exclusively acts on beta-receptors causing both an increase in heart rate (β1 receptors) and hypotension (β2 receptors). It is generally started as an intravenous infusion at 0.5–1 mcg/min and titrated up until the desired clinical effect is achieved (generally initiation of sustained tachycardia – usually 2–5 mcg/min). Isoproterenol is useful in the electrophysiology laboratory because of its rapid onset of action and short half-life (<1.5 min). In this case, low-dose isoproterenol infusion improves VA conduction, and now stable VA conduction is observed. VA conduction can be identified by a change in the pattern of atrial depolarization and an increase in the atrial rate to the paced ventricular rate (450 ms). The typical response to ventricular premature stimulation is shown in Figure 1.14. In this case, after pacing at a basic cycle length of 400 ms (notice the 1:1 VA conduction since the patient is on isoproterenol), a premature ventricular stimulus at a coupling interval of 200 ms is associated with a delay in VA conduction. Prolongation of VA conduction can be due to delay in intraventricular conduction, the bundle branches, the
Figure 1.12  Baseline ventricular pacing is not associated with VA conduction. Notice the pattern of atrial depolarization remains “high-low” with first atrial depolarization noted in the HRA.

Figure 1.13  Continuation of Figure 1.12. Infusion of isoproterenol and sympathetic activation facilitates development of VA conduction. Notice that in response to the pacing stimulus, the pattern of atrial depolarization switches from “high-low” to “low-high” due to retrograde VA conduction via the AV node. The atrial rate now increases to the ventricular paced rate. Notice the subtle changes in the timing of atrial depolarization as activation progressively switches from being “sinus node driven” (S) to “AV node driven” (R). Although one could quibble about whether atrial activation recorded in the His bundle region (S*) for the first paced beat is actually from retrograde activation, it is likely from the sinus node, as the coronary sinus depolarization remains relatively late. Similarly, one could argue whether coronary sinus depolarization from the third paced beat is actually due to depolarization from the sinus node, but it is likely due to retrograde depolarization because of the shortened interval between atrial depolarization recorded in the His bundle and the proximal coronary sinus. It is instructive to note that as a separate but important point, the pattern of coronary sinus depolarization (from proximal to distal) is the same in this case regardless of whether atrial depolarization is “sinus node driven” or “AV node driven.”
His bundle, or in the AV node. Although a retrograde His bundle can often be difficult to identify, the absence of an identifiable His bundle suggests that most of the VA conduction delay is due to delay in the AV node. Just as in atrial pacing, ventricular pacing may lead to induction of arrhythmias.

**Electrograms during tachycardia**

The most common reason for patients to be referred for electrophysiologic testing is for the evaluation (and possibly definitive treatment) of tachycardia. Once the baseline electrophysiologic characteristics for a patient have been determined, pacing is generally performed in hopes of inducing the patient’s clinical tachycardia. If the clinical tachycardia can be induced, the tachycardia can be studied and the critical anatomic components can be defined with a sense for the critical tissues involved.

When tachycardia is induced, the electrophysiology study can help define the relationship between atrial and ventricular electrograms. For example, if there are more atrial signals than ventricular signals, then an atrial tachycardia driving ventricular activation is the most likely possibility. Conversely, if there are more ventricular signals than atrial signals, ventricular tachycardia becomes the likely diagnosis. In fact, the greatest benefit of intracardiac electrograms is definitive information on the timing of atrial depolarization. Remember that the most difficult thing about analyzing the ECG of a patient in tachycardia is identifying the P waves that are often obscured by the QRS complexes or the T waves.

In patients with supraventricular tachycardia, ventricular activation is normal, so generally evaluation of a supraventricular tachycardia initially focuses on the timing and pattern of atrial activation (Figures 1.15–1.17 and Table 1.1). Generally, supraventricular tachycardias are classified first by the relative number of atrial and ventricular electrograms. If there are more atrial electrograms than ventricular electrograms, it is likely that atrial activation is “driving” ventricular activation and increases the likelihood that an atrial tachycardia is present. If there are more ventricular electrograms than atrial electrograms during a supraventricular tachycardia, both atrial tachycardia and accessory pathway-mediated tachycardia are very unlikely and essentially rules in a tachycardia focus from the junctional region. When a 1:1 relationship between atrial electrograms and ventricular electrograms is present, any of the three anatomic types of tachycardia may be present, and the clinician must focus on the timing...
between atrial activation and ventricular activation and the pattern of atrial activation. Firstly, the timing of atrial activation and ventricular activation is assessed: is atrial activation present in the first half of the interval between QRS complexes ("short RP" tachycardia) or in the second half of the interval between QRS complexes ("long-RP" tachycardia)? The likely causes of tachycardia vary by the relationship between ventricular and atrial activation, although this can be difficult (Figure 1.16). In addition to the timing of atrial activation, the direction of atrial activation is evaluated (Figure 1.17). The direction of atrial activation can be evaluated in the plane of the tricuspid and mitral valves (left to right, right to left, or from the center outward) and the perpendicular plane – anterior to posterior or posterior to anterior. In supraventricular tachycardia, it is important to decide whether retrograde activation is present – in other words, is it likely that ventricular depolarization “drives” atrial depolarization? If atrial activation is high-low, then the ventricles are generally not responsible for atrial depolarization. When atrial depolarization appears to be coming from the mitral or tricuspid annulus, atrial depolarization is further classified as “concentric” or beginning from the septum and extending to the left and right (and thus the AV node could be responsible for retrograde activation), or “eccentric,” where initial atrial activation is identified more laterally away from the septum in either the left or the right atria.

The tachycardia can be carefully evaluated to determine whether changes in the atrial cycle lengths precede and predict those in the AV node or His bundle cycle lengths (making atrial tachycardia more likely), or whether AV node conduction is essential for continuation of tachycardia (AV node dependent), which would "rule out" atrial tachycardia.
Pacing during tachycardia

Once a tachycardia is induced, after initial inspection of the electrograms (and confirmation of hemodynamic stability!), the electrophysiologist can study the response of the tachycardia to different stimuli.

For example, as we have covered, in a patient with supraventricular tachycardia, there are only three basic anatomic possibilities: atrial tachycardia, junctional tachycardia, and tachycardias that use an accessory pathway. Once a supraventricular tachycardia is induced and the patient is stable, the best course of action is delivery of ventricular stimuli (either single, double, or as a pacing train). Regardless of how ventricular stimuli are delivered, the stimuli are most likely to interact with a tachycardia using an accessory pathway, less likely with a tachycardia within the junction, and least likely with an atrial tachycardia (Figure 1.18).
Figure 1.17 Determining the direction of atrial activation during supraventricular tachycardia. The focus of investigation should be whether “High to Low” or “Low to High” and whether “Left to Right,” “Right to Left,” or “Septal Outward.”

Understanding this graded response rate can help differentiate between these tachycardias. Although the response of the tachycardia to atrial pacing can also be evaluated, it is generally less helpful because it will interact “early” with both atrial tachycardias and accessory pathway-mediated tachycardias. Table 1.1 shows the general characteristics of the three different anatomic types of supraventricular tachycardia.

Table 1.1 Findings that are generally associated with the most common forms of specific supraventricular tachycardia types.

<table>
<thead>
<tr>
<th>Tachycardia type</th>
<th>Characteristics</th>
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| Atrial tachycardia | • “Long-RP” tachycardias (VA interval > 1/2 RR)  
                               • Continuation of the tachycardia despite AV block (AV node independent)  
                               • Variable VA intervals  
                               • Requires very early V’s to interact with the tachycardia (sometimes not at all) |
| AVNRT | • Very “short-RP” tachycardia (VA interval < 65 ms)  
                               • Usually AV node dependent  
                               • Usually VA intervals  
                               • Early V’s can interact with the tachycardia |
| AVRT | • “Short-RP” tachycardia (VA interval > 65 ms, but < 1/2 RR)  
                               • AV node dependent  
                               • Fixed VA intervals  
                               • Late V’s can interact with the circuit |

AVNRT, AV node reentrant tachycardia; AVRT, AV reentrant tachycardia; RR, Interval between QRS complexes; VA, Ventriculoatrial; V, Ventricular stimulation.
Figure 1.19  A premature ventricular stimulus delivered during supraventricular tachycardia initiates a wide complex tachycardia at a more rapid rate than the underlying tachycardia (which is unaffected by the PVC). Ventricular tachycardia can be identified immediately, because there are more QRS complexes than atrial activity during the stable wide QRS tachycardia (the last four QRS complexes).

Likewise, in patients with wide complex tachycardia, it becomes very important to distinguish between ventricular tachycardia and supraventricular tachycardia with aberrant conduction. In this case, pacing from the atrium is extremely helpful, because it will interact with the different types of supraventricular tachycardia before interacting with ventricular tachycardia. Obviously, if a wide complex tachycardia is induced, it is even more important to confirm that the patient is stable from a clinical standpoint before embarking on the electrophysiologic analysis of the arrhythmia.

Figure 1.19 emphasizes the importance of continuous evaluation of the patient during electrophysiology studies. In this case, the patient has supraventricular tachycardia, and timed premature ventricular stimuli are being delivered. Although generally safe in patients with structurally normal hearts, a premature ventricular stimulus delivered on the T wave results in the initiation of ventricular tachycardia.

KEY POINTS

- Anatomically, there are only four types of tachycardia.
- In an electrophysiology study, baseline conduction intervals are measured and pacing protocols are performed to evaluate the electrical characteristics of different cardiac tissues and to induce tachycardias.
- If a tachycardia is induced/observed during the evaluation, the response to pacing during tachycardia can help define the mechanism of the tachycardia.
- Generally, in patients with supraventricular tachycardia, response to ventricular pacing is most useful for determining the type of tachycardia present.