CHAPTER 1
Basic principles of pacing

Malcolm Kirk

The aim of this chapter is to give sufficient background and information about cardiac pacemakers to allow interpretation of electrocardiograms (ECGs) and telemetry strips of normal pacemaker behavior. For more in-depth information, such as would be necessary for programming pacemakers, a standard pacing text should be consulted. Several of these are listed in the bibliography. Most italicized terms are defined in the glossary at the end of the chapter.

Anatomy

The pertinent anatomy for cardiac pacing includes the sinoatrial (SA) node, the atrioventricular (AV) node, and the His-Purkinje system (Figure 1.1).

The SA node is located at the superior aspect of the *crista terminalis* (not pictured), near the junction with the superior vena cava. It is normally the dominant pacemaker in the heart, because its rate of depolarization exceeds that of other areas that normally possess properties of automaticity, such as the more inferior areas of the *crista terminalis* and the His-Purkinje system. The SA node can, in turn, be suppressed by an even faster rhythm, such as an atrial tachycardia, or pacing by an implanted pacemaker.

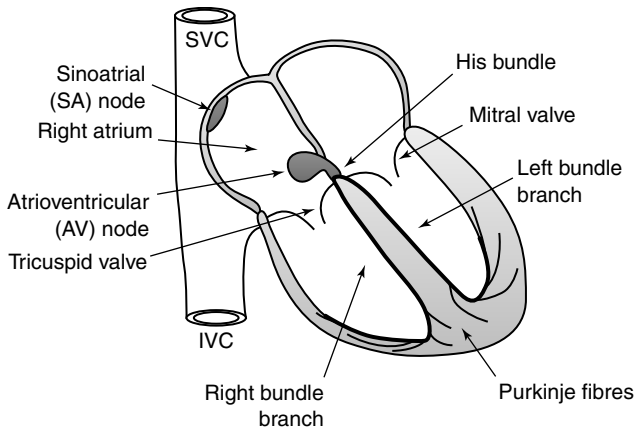

Figure 1.1 Schematic of conduction system anatomy

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The AV node is normally the only electrical connection between the atria and the ventricles. Electrical activation proceeds from the right atrium, through the AV node to the His-Purkinje system, and then to the ventricles.

The His-Purkinje system comprises myocardial cells that are specialized for rapid conduction. Its anatomic components are (in order of activation) the His bundle, the bundle branches (right and left) and the Purkinje fibers. The His-Purkinje system delivers the electrical impulse rapidly from the AV node to widely dispersed areas of the left and right ventricular endocardium, making activation nearly simultaneous throughout the ventricles. This rapid conduction, and simultaneous activation of the right and left ventricles, results in the narrow QRS complex seen on a normal ECG. If an impulse is transmitted throughout the ventricle without using the His-Purkinje system, it takes longer for the ventricles to be activated, and hence the QRS complex is wider. An example would be a premature ventricular contraction (PVC). Another would be ventricular pacing, because the pacemaker lead is usually not positioned so as to activate initially the His-Purkinje system. (Furthermore, in this case the ventricles are activated sequentially rather than simultaneously.)

The components of the surface ECG reflect the cardiac chambers and conducting system (Figure 1.2). The activation of the atria creates the *P wave* on the surface ECG. Electrical conduction through the AV node to the His-Purkinje system is relatively slow, so there is normally a 120–200 millisecond (ms) delay between the start of atrial activation and the start of ventricular activation. The delay between the onset of the surface P wave and the onset of the QRS complex is due mostly to conduction through the AV node, with some contribution from intraatrial conduction, and His-Purkinje system conduction. The activation of the ventricles creates the QRS complex.

As noted above, the AV node and His-Purkinje system are normally the only electrical connection between the atria and the ventricles. Failure of electrical conduction through the AV node and/or His-Purkinje system results

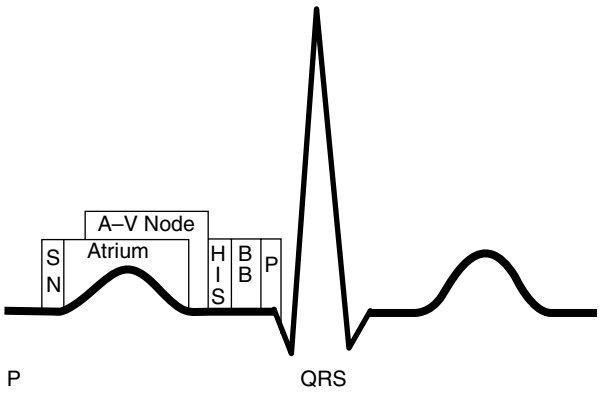

Figure 1.2 The conducting system is reflected in the normal QRS complex. SN = sinus node, His = His bundle, BB = bundle branches, P = Purkinje fibers.

in *heart block*, also known as AV block, and is one of the indications for pacemaker implantation (see Chapter 3). When assessing a pacemaker patient, it is important to consider whether or not the patient has intact *intrinsic conduction* through the AV node and His-Purkinje system. If electrical conduction from the atrium to the ventricles is not present (AV block), then the patient is likely to be dependent on ventricular pacing to maintain an adequate heart rate.

Physiology

Cardiac muscle cells, like other excitable cells, have a resting electrical gradient across the cell membrane. In the quiescent state (during diastole), the inside of the cell (cytoplasm) is electrically negative relative to the outside of the cell. That is to say, the cell membrane separates positive (outside) and negative (inside) charges. Thus the cell membrane is *polarized*. It becomes *depolarized* when an electrical current causes opening of sodium (Na^+) and calcium (Ca^{2+}) channels in the cell membrane, allowing these positive ions to rush into the cell. This flow of positive ions into the cell has two important consequences: propagation of electrical activity (the action potential) and contraction of the cell.

Propagation of the action potential

Propagation is the spread of depolarization in a wave across the heart. Because the flow of positive ions into the cell is itself a small current, it causes opening of Na^+ and Ca^{2+} channels in adjacent cells. The opening of these channels, in turn, creates a current that causes opening of channels in cells beyond, and so forth, so long as adjacent cells are *excitable*, and not *refractory* (see below).

Refractoriness

Refractoriness is a normal property of cardiac tissue. After depolarization, cells need a certain amount of time to recover before they can be stimulated again. In the most general sense, *refractoriness* is the opposite of *excitability*. After a cardiac muscle cell has been *depolarized* (also called *excited*), it cannot be depolarized again until the membrane has become polarized again (or repolarized). The time between an electrical stimulus that excites a certain part of the heart, and the latest repeat stimulus that cannot excite the same tissue is known as the *refractory period*. A stimulus that fails to excite the heart because it occurs too soon after the previous stimulus or depolarization is said to find the tissue *refractory*.

The amount of time required for recovery of excitability (i.e. the refractory period) depends on the type of cardiac tissue (atrium, ventricle, AV node, conducting system), and may be influenced by medications or by rate of stimulation. The refractory period of the AV node is important in pacing and atrial arrhythmias. It will determine how frequently atrial impulses can be transmitted to the ventricle. For example, in atrial fibrillation, the atrial rate can

exceed 400 beats per minute (bpm), but not every impulse is transmitted to the ventricle. Most of the atrial beats will be blocked at the AV node, because they reached the AV node at a time when it is refractory and cannot conduct. The response of the AV node to rapid stimulation rates differs from other cardiac tissue, in that the refractory period of the AV node generally increases with increased rates of stimulation, whereas the refractory period of the atria and ventricles decreases with increased rates of stimulation. The AV node thus limits the maximum rate at which the ventricle can follow a rapid atrial rhythm.

Parts of a pacemaker system

A pacemaker consists of a *pulse generator* (Figure 1.3) and pacing *leads*.

The pulse generator contains the battery of the pacemaker, as well as the circuits that deliver the pacing stimuli. The lead input and circuitry in a pacemaker pulse generator dedicated to a particular chamber of the heart is known as a *channel*. For example, the ventricular channel transmits the ventricular pacing impulse to the ventricular lead.

Pacemaker leads are electrical conductors (wires), covered with insulation. They transmit the electrical impulses from the pulse generator to the heart, and from the heart to the pulse generator.

Pacemaker leads are usually inserted into the subclavian vein or its tributaries, and positioned on the inner surface (endocardium) of the heart. They are attached to the endocardium by a small screw mechanism, or are held in place by tines. If a screw (also known as a helix) is used to fix the lead to the heart, the lead is called an active fixation lead (Figure 1.4). A passive fixation

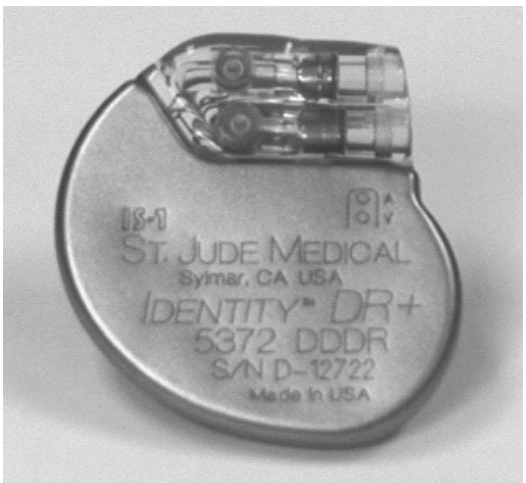

Figure 1.3 Pacemaker pulse generator. Dimensions: 4.4 cm × 5.2 cm × 0.6 cm. Courtesy of St. Jude Medical.

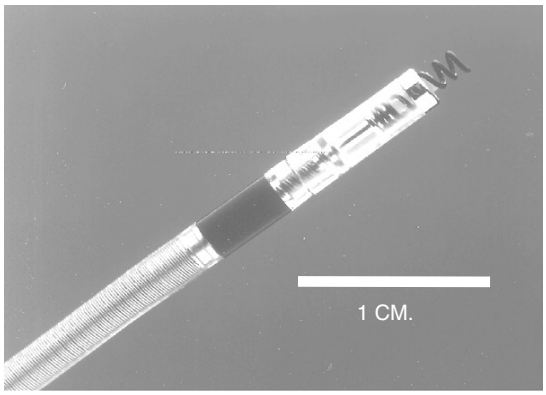

Figure 1.4 Active fixation lead. Courtesy of St. Jude Medical.

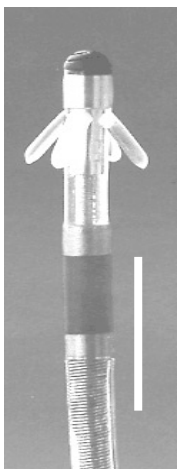

Figure 1.5 Passive fixation lead. The white bar is 1 Cm. Courtesy of St. Jude Medical.

lead has tines (Figure 1.5), which are designed to engage the trabeculae on the inner surface of the heart.

Pacemaker leads may also be placed on the outside of the heart (epicardium) during a surgical procedure. These leads are either sewn onto the heart, or fixed in place with a small screw-in mechanism.

The pacing stimulus

Pacemakers function by delivering a small electrical current to myocardial cells. The electrical activation spreads from cell to cell, throughout the heart. As each cell is electrically activated, it contracts.

The pacemaker delivers the electrical current between two points, called electrodes. These two points may be either two electrodes on the pacemaker lead, or one electrode on the pacemaker lead, and the metal covering of the

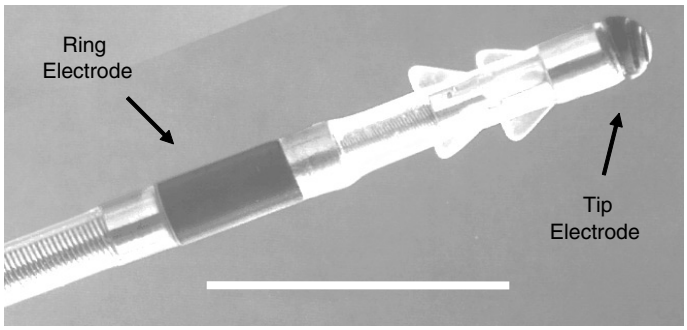

Figure 1.6 The two electrodes of a bipolar pacing lead. The white bar is 1 cm. Courtesy of St. Jude Medical.

pacemaker pulse generator. Electrical current is caused by the flow of electrons. This must occur in a circuit (i.e. a closed loop). A source of current, such as the battery of a pacemaker, will have a negative end (from which electrons are emitted) and a positive end (to which electrons are attracted). For reasons beyond the scope of this chapter, pacing is more efficient when the tip electrode of the pacing lead is the negative pole. The positive pole can either be a metallic ring, about a centimeter back from the tip of the lead (Figure 1.6), or can be the body of the pacemaker pulse generator itself.

Bipolar and unipolar pacing – ECG

Bipolar and *unipolar pacing* have different appearances on surface ECGs. If the current flows between the two electrodes on the pacemaker lead (the tip and the ring), this is referred to as bipolar pacing. If the current flows between the tip of the lead and the pacemaker generator, this is referred to as unipolar pacing. In unipolar pacing, the current travels through a large area of the body between the tip of the lead and the pulse generator. Unipolar pacing, therefore, creates a large *stimulus artifact* on the surface ECG. It may stimulate electrically excitable tissue, other than the heart, which lies in the path of the current. An example of such a tissue would be the pectoralis muscle over which the pacemaker generator is placed. The bipolar pacing stimulus may be very difficult to see on the surface ECG, because in bipolar pacing the distance between the two poles that deliver current (i.e. the tip and the ring of the pacemaker lead) is very small (about a centimeter), as illustrated in Figure 1.6. It will also be noted that leads that are used for bipolar pacing must have two insulated wires within its outer insulation: one wire for the negative pole (the tip), and one for the positive pole (the ring).

Pacing by defibrillators

The implantable cardioverter-defibrillator (usually called an ICD or defibrillator) was developed to detect life-threatening ventricular tachyarrhythmias,

Figure 1.7 Defibrillator pulse generator. Dimensions: 6.7 cm × 5.0 cm × 1.4 cm. Courtesy of St. Jude Medical.

and terminate them by delivering a high-energy defibrillating shock to the heart. The pulse generator is somewhat larger than that of a pacemaker (Figure 1.7). All modern defibrillators also function as pacemakers. This is not commonly understood. A patient will often be told that he or she has "a pacemaker and a defibrillator" when the patient, in fact, has only a single device – a defibrillator. Modern defibrillators include nearly as many pacing features as modern pacemakers; anything in this chapter about pacemakers also applies to the pacing function of defibrillators, unless otherwise stated.

Single and dual chamber pacing

The word *chamber* in dual or single chamber pacing refers to a chamber of the heart in which a lead is placed. Each lead is connected to a *channel* of the pacemaker (see section on "Parts of a pacemaker system"). The channel is the part of the pacemaker circuitry and memory assigned to that particular lead (and therefore, the corresponding cardiac chamber).

A single chamber pacemaker usually has a pacing lead in either the right atrium, or the right ventricle. These would be called, respectively, an atrial single chamber pacemaker and a ventricular single chamber pacemaker. (They are also sometimes called an AAI pacemaker and a VVI pacemaker, respectively, for reasons that will become apparent in the section on "Pacing modes.")

A standard dual chamber pacemaker has a lead in the right atrium and a lead in the right ventricle. (A dual chamber defibrillator has a pacing lead in the right atrium and a pacing/defibrillation lead in the right ventricle. The latter delivers the defibrillation shock, as well as pacing.) Biventricular pacing is a newly developed type of pacing incorporating a third lead, which is positioned to activate the posterolateral wall of the left ventricle. This results in

"ventricular resynchronization," which can improve ventricular hemodynamics, and relieve heart failure symptoms in certain patients with heart failure and conduction abnormalities. (This is detailed in Chapter 7.)

The interaction between the atrial and ventricular chambers in dual chamber pacing can be somewhat complex. Failure to understand this interaction is the source of many questions about pacemaker behavior. The elements needed to understand dual chamber pacing are covered below.

Conceptual building blocks of pacemaker function

Pacing

Pacing refers to the regular output of electrical current, for the purpose of depolarizing the cardiac tissue in the immediate vicinity of the lead, with resulting propagation of a wave of depolarization throughout that chamber. A pacemaker will pace at a certain frequency, or rate, for example, 60 bpm. This rate is *programmable*. That is, it can be changed by using the manufacturer's *programmer*.

Sensing

The heart's intrinsic electrical activity (i.e. the P wave or QRS complex) transmits a small electrical current (a few millivolts), through the pacemaker leads, to the pulse generator. This current can be registered or *sensed* by the pacemaker circuitry. Pacemaker sensing describes the response of a pacemaker to intrinsic heartbeats. The P waves, or atrial activity, are transmitted through the atrial lead (if present) to the atrial channel of the pacemaker, and sensed as atrial activity. Ventricular activity (the QRS complex) is transmitted through the ventricular lead (if present) to the ventricular channel of the pacemaker, and this is sensed as ventricular activity.

For electrical activity to be transmitted from the heart to the pacemaker, a closed electrical circuit must be present, just the same as for an electrical impulse to be transmitted from the pacemaker to the heart. Thus, just as with pacing, sensing can be unipolar or bipolar. *Bipolar sensing* detects the intrinsic electrical activity occurring between the tip electrode and the ring electrode of the lead. *Unipolar sensing* detects electrical activity occurring between the tip of the lead, and the metal shell of the pulse generator. Because this is a much larger area, other electrical signals, such as might be generated by the muscles of the diaphragm or sources outside the body, are more likely to be detected (and therefore incorrectly interpreted by the pacemaker as heart beats).

It is important to note that the only way the pacemaker can determine which chamber a signal originates from is by which lead transmits the signal to the pacemaker. For example, the pacemaker will interpret any electrical signal transmitted through the atrial lead to the atrial channel as a P wave, even if the signal is in fact a QRS complex large enough in amplitude to be sensed by the atrial channel.

Figure 1.8 A single P–QRS complex is seen in two leads in a patient with a pacemaker. The vertical black line marks the instant when the pacemaker *senses* the QRS complex. Note that sensing by the pacemaker occurs well after the onset of the surface QRS. Up until this point, the pacemaker is not "aware" that a QRS complex is in progress. This illustrates how a normally functioning pacemaker can deliver a pacing spike early in the QRS complex, before it senses the presence of that complex.

Note also that the time at which the pacemaker senses the atrial or ventricular signal is not necessarily the beginning of the P wave or QRS. The pacemaker cannot sense activity in a chamber until the electrical activity actually reaches the pacemaker lead. Figure 1.8 illustrates that sensing in the ventricle occurs after the onset of the QRS complex.

Inhibition of output

A pacemaker can be programmed to inhibit pacing if it senses intrinsic activity, or it can be programmed to ignore intrinsic activity and deliver a pacing stimulus anyway. If a pacemaker is set so that it can be inhibited by *intrinsic beats*, then the pacemaker will not deliver a stimulus if it senses an intrinsic beat at the proper time. For example, if a pacemaker is set to pace in this way at 60 bpm, it will deliver a pacing stimulus only if an intrinsic beat does not occur within 1 second of the last sensed or *paced beat*.

Triggered pacing

Pacemakers can be programmed to deliver a pacing stimulus whenever intrinsic activity is sensed. This type of pacing is most often used in dual chamber pacemakers. Dual chamber pacemakers can be programmed to sense activity in one chamber (usually the atrium) and deliver a pacing stimulus in the other chamber (usually the ventricle) after a certain time delay. This is known as triggered pacing. When referring to the appearance of this type of pacing on telemetry or ECGs, it is commonly said that the ventricle is *tracking* the atrium, because if the atrial rate becomes faster, the ventricular pacing rate will follow faster, in a 1 : 1 relationship. Thus the exact rate of ventricular pacing will not be determined by any *setting* on the pacemaker, but by the

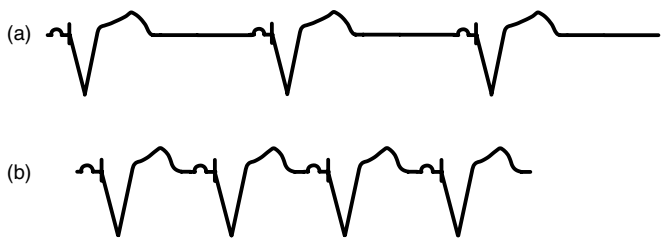

Figure 1.9 Triggered pacing. (a) and (b) both show atrial sensing, and ventricular pacing, in a tracking mode. The pacemaker settings are the same in both panels. The difference is that in (b) the intrinsic atrial rate is faster.

patient's own atrial rate (Figure 1.9). One would want to limit, of course, the maximum rate at which the pacemaker will track the atrial rhythm with ventricular pacing. This is discussed more extensively in the section on "Basic pacemaker programming – timing cycles."

Pacing modes

A standard pacemaker code has been developed jointly by the North American Society for Pacing and Electrophysiology, and the British Pacing and Electrophysiology Group (the NASPE/BPEG Generic Code, known as the NBG Code). The pacing function provided by a pacemaker (or defibrillator) is usually given by a series of three or four letters. (There is a fifth position in the code, which is not commonly used.) Each position denotes a different aspect of pacemaker function. The identity of the letter in that position specifies the function. A given combination of three or four letters is called a *mode*. A more in-depth discussion of these modes, and in the clinical scenarios for which each is used, appears in Chapter 3.

The positions and letters are as follows:

- Position 1: chamber being paced

  V = ventricle
  A = atrium
  D = atrium and ventricle (dual)
  O = no pacing.

- Position 2: chamber being sensed

  V = ventricle
  A = atrium
  D = atrium and ventricle (dual)
  O = no sensing.

- Position 3: pacing response to a *sensed beat*

  I = inhibited
  T = triggered

D = inhibited or triggered (dual) depending on the chamber
O = neither inhibited or triggered.

- Position 4: rate response or absence thereof (see section on "Basic pacemaker programming – sensing, pacing , and refractory periods")

R = rate responsive
O = absence of rate response. (But this is usually just omitted.)

Here are some examples:

- The VVI mode paces and senses only in the ventricle.

Position 1 – V indicates that it will pace only in the ventricle.
Position 2 – V indicates that it senses intrinsic heartbeats only in the ventricle.
Position 3 – I indicates that the response to a sensed heartbeat is inhibition of ventricular pacing.
Position 4 – blank, indicating that it is not rate responsive.

- The VAT mode would pace as follows:

Position 1 – V indicates that it will pace only in the ventricle.
Position 2 – A indicates that it will sense only in the atrium.
Position 3 – T indicates that the pacemaker will deliver a ventricular stimulus every time it senses an atrial beat.
Position 4 – blank, indicating that it is not rate responsive.

- DDD is a common mode. It operates as follows:

Position 1 – D indicates that the pacemaker will pace in both the atrium and ventricle.
Position 2 – D indicates that it will sense in both the atrium and the ventricle.
Position 3 – D indicates that it will respond to a sensed beat in either chamber with inhibition of pacing output in that chamber, but it will also deliver a pacing stimulus in the ventricle after an atrial beat is sensed, unless there is inhibition by an intrinsic ventricular beat (i.e. triggered response).
Position 4 – blank, indicating that it is not rate responsive.

Therefore, depending on the patient's intrinsic heart rate, and the programmed settings, a pacemaker in DDD mode might pace both the atrium and the ventricle, pace neither (sense in both), pace the atrium with intrinsic conduction to the ventricle (ventricular pacing output inhibited by sensed *R waves*) or track intrinsic P waves with ventricular pacing. These are illustrated in Figure 1.10.

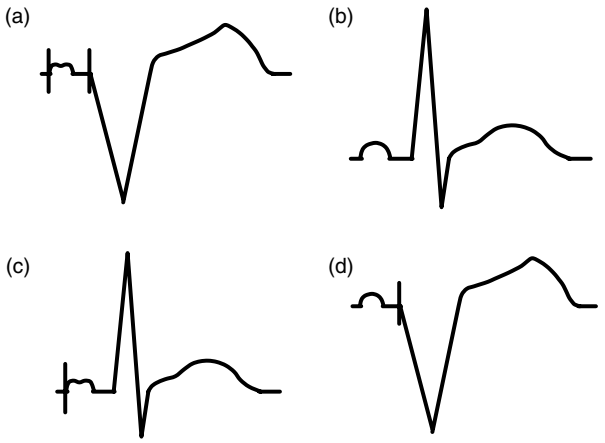

Figure 1.10 Types of pacing and sensing possible in DDD mode. (a) Pacing both the atrium and the ventricle; (b) pacing neither (sensing in both); (c) pacing the atrium with intrinsic conduction to the ventricle (ventricular pacing output inhibited by sensed R waves); (d) tracking intrinsic P waves with ventricular pacing. Note that which of these four electrograms is seen depends on the relationship of the intrinsic atrial rate and PR interval to the pacemaker lower rate and AV delay settings.

VOO is the simplest pacing mode (Figure 1.11).

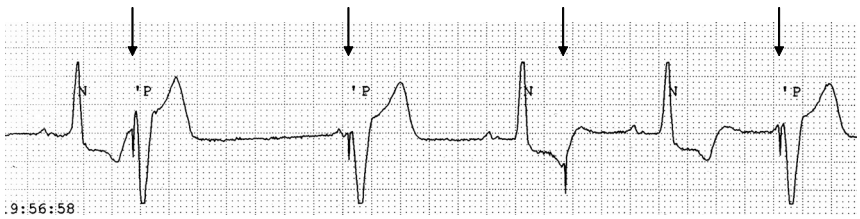

Figure 1.11 VOO pacing. The pacemaker is set in VOO mode. This is known as an *asynchronous* mode because the pacing stimuli are unrelated to the intrinsic rhythm. The arrows indicate the pacing stimuli, which are delivered at a rate of 40 bpm, and fall in random relationship to the intrinsic rhythm. Note that the first pacing stimulus falls just after an intrinsic QRS. It is not inhibited by the preceding QRS, which is not sensed because there is no sensing in this mode (the second position in the code is "O"). The third pacing stimulus falls when the ventricle is *refractory*, and so it fails to *capture* (i.e. it does not result in a QRS complex). The "N" and "P" are drawn in by the telemetry system when it designates beats as normal and paced, respectively.

Position 1 – V indicates that the pacemaker paces only in the ventricle.
Position 2 – O indicates that the pacemaker does not sense intrinsic activity; therefore, the third letter must also be O because it cannot have any response to intrinsic activity.
Position 3 – O indicates it is neither triggered nor inhibited by sensed activity.
Position 4 – blank, indicating that it is not rate responsive.

Basic pacemaker programming – sensing, pacing, and refractory periods

Sensing and pacing

The amount of electrical current delivered by a pacemaker is adjustable. The minimum amount of energy required to pace the heart can be found by decreasing the output energy until the pacing stimulus no longer causes depolarization of the atria or ventricles, seen as a P wave or QRS complex following the pacing stimulus artifact. The minimum energy needed to pace is called the pacing threshold. The output is set sufficiently above this threshold (often twice the threshold) to provide a safety margin for pacing *capture*, without excessive battery drain.

Sensing is conceptually more difficult. In order not to pace the heart inappropriately, the pacemaker must be able to sense intrinsic local depolarization. The *sensitivity* of a pacemaker channel is the threshold that the intrinsic electrical activity transmitted from that chamber must meet to be registered by the pacemaker. For example, if ventricular sensing is set to 2.5 mV, then the amplitude of the intrinsic QRS complex, as transmitted by the ventricular pacing lead to the pacemaker, must be at least 2.5 mV for the pacemaker to register that an intrinsic QRS complex has occurred. This is illustrated in Figure 1.12. Increasing the sensitivity value raises the threshold that the intrinsic activity must meet to be registered (i.e. it makes the pacemaker less sensitive). Proper adjustment of this level for an individual patient allows sensing of intrinsic activity without *oversensing* of extraneous activity, such as T waves, muscle potentials from the diaphragm, or sensing of R waves on the atrial lead.

Rate response

Currently available pacemakers (and defibrillators) can be programmed to vary the pacing rate in response to the patient's level of activity. Various

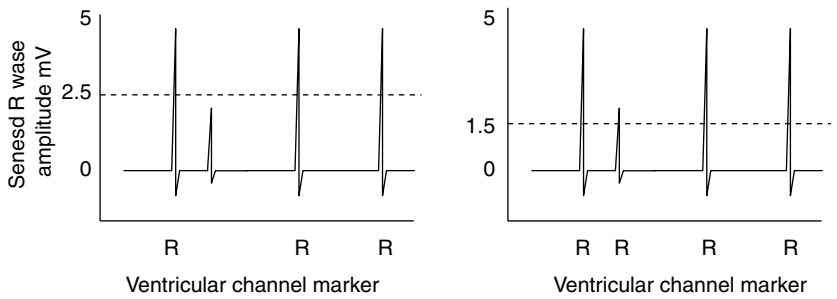

Figure 1.12 The second intrinsic complex (a PVC) does not create a large enough signal to be sensed in the ventricular channel with the sensing set at 2.5 mV (dotted line). Therefore, the pacemaker does not recognize the presence of the second intrinsic R wave. (The marker channel at the bottom indicates which intrinsic R waves are sensed by the pacemaker.) If the sensitivity (i.e. the threshold for sensing) had been lowered to 1.5 mV, the second beat would have been sensed.

types of sensors have been developed to allow the pacemaker to sense the patient's level of activity. The parameters sensed include patient movement, respiration, and changes in the T wave. Activation of the sensor by changes in these factors, which are not related to exercise, might be a cause of more rapid than expected pacing at rest. For example, hyperventilation or an activity that causes motion of the pacemaker pulse generator (e.g. physical therapy of the chest or shoulder) may increase the activity of pacemakers driven by minute ventilation and accelerometer sensors, respectively.

Refractory period

Pacemakers have refractory periods on each channel, which are generally programmable. During the refractory periods, sensed events are ignored. The most clinically important refractory period occurs in dual chamber pacing. It is the *postventricular atrial refractory period*, or *PVARP*. This is the period after a ventricular sensed or paced event, when the atrial channel is refractory. The effect is that in a dual chamber tracking mode, a P wave that falls shortly after the QRS complex (either a paced or intrinsic QRS) will not be *tracked*. That is, it will not be followed by a ventricular paced event. An important function of PVARP is to prevent pacemaker-mediated tachycardia. Pacemaker-mediated tachycardia (often abbreviated: "PMT") begins when a ventricular sensed or

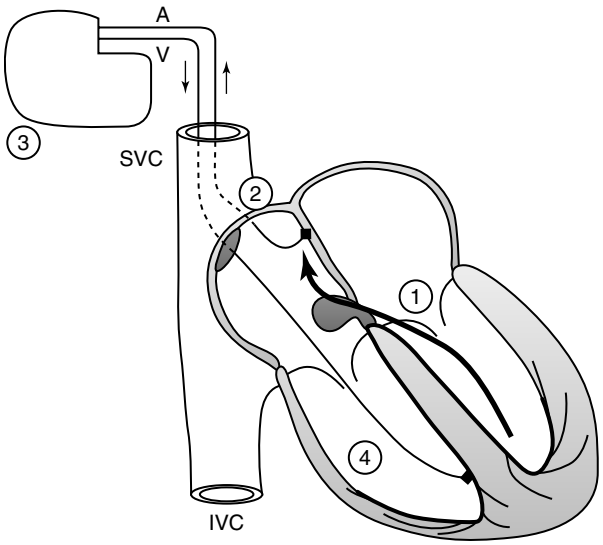

Figure 1.13 Pacemaker-mediated tachycardia. Step 1: conduction retrogradely from the ventricles to the right atrium through the AV node. Step 2: sensing of the resulting retrograde P wave by the atrial channel of the pacemaker. Step 3: triggered ventricular pacing tracking the sensed atrial event. (Note that the pacemaker will deliver ventricular pacing no faster than the upper tracking rate.) The paced ventricular beat is again conducted retrogradely up the His-Purkinje system, beginning step 1 again.

paced beat is transmitted *retrogradely* (from ventricle to atrium) through the His-Purkinje system and AV node, resulting in atrial activation. Without a pacemaker, the resulting P wave would not be conducted back to the ventricle, because the AV node and His-Purkinje system are *refractory*, having just been activated retrogradely. However, if the patient has a dual chamber pacemaker set in the tracking mode, then the pacemaker can sense the resulting P wave, and "track" it by delivering a ventricular impulse after an appropriate delay. Thus an endless loop is created. The electrical activity is transmitted from the ventricle to the atrium through the His-Purkinje system and AV node, and from the atrium to the ventricle through the pacemaker (Figure 1.13). The rate of PMT will be limited by the upper rate limit of the pacemaker (see section on "Basic pacemaker programming – timing cycles"), because the pacemaker will not track atrial beats faster than this rate. The presence of a PVARP helps prevent PMT. If atrial beats that have been transmitted retrogradely from the ventricle fall within the PVARP, then the pacemaker will not track them and they will not be transmitted back to the ventricle via ventricular pacing.

The PVARP also allows the pacemaker to respond to fast atrial rates in a physiologic fashion (see section on Upper rate behavior). The PVARP thus partially mimics the normal physiology of the intrinsic conduction system (see discussion of AV node refractory period in the section on "Physiology").

Basic pacemaker programming – timing cycles

The lower rate limit

This is the simplest aspect of pacemaker timing. In AOO or VOO (*asynchronous*) mode, this is the rate at which the pacemaker will pace. In AAI, or VVI mode, this is the rate at which the pacemaker will pace, unless it is inhibited by intrinsic beats occurring at a faster rate. (Two reasons that the heart rate may fall below the lower rate limit of the pacemaker are hysteresis and sleep function, both described below in the FAQ section.) If the pacer is in a rate-responsive (or rate-modulated) mode, then the actual lower rate limit will vary, depending on the activity of the rate-response sensor, but will not be less than the programmed lower rate limit.

AV delay

The AV delay is the time interval between an atrial paced or sensed event, and the delivery of a ventricular pacing stimulus. Because it involves events in two chambers, it is a programmable parameter in dual chamber pacemakers, but is not found in single chamber pacemakers. It is analogous to the intrinsic PR interval, in that it allows time for atrial contraction, and active ventricular filling, before ventricular contraction occurs. There are two types of AV delay: *sensed AV delay* and *paced AV delay*. The terms "sensed" and "paced" refer to events in the atrium.

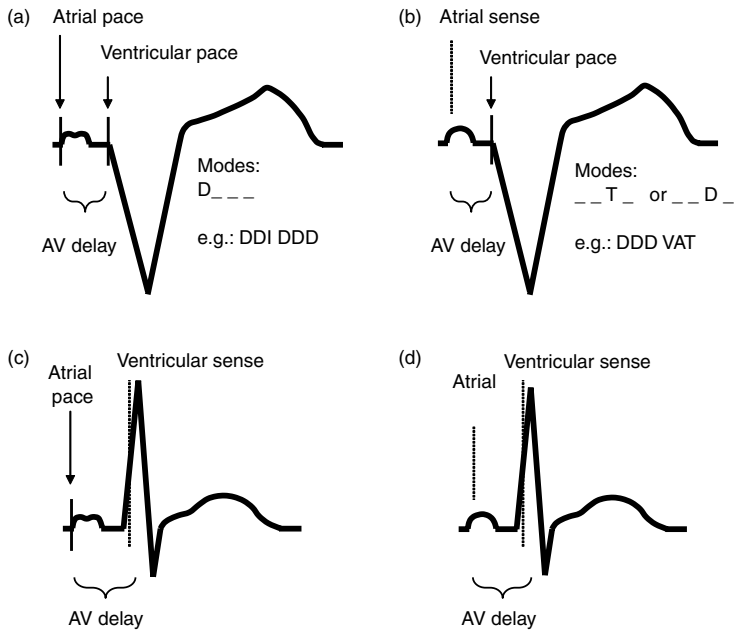

Figure 1.14 AV delay. (a) and (c) Paced AV delay in a dual chamber pacing mode. (b) and (d) Sensed AV delay in a triggered mode. The AV delay is a programmed parameter. (a) and (b) show a programmed AV delay that is shorter than intrinsic conduction. The pacer delivers a pacing stimulus at the end of the AV delay. In (c) and (d) a longer AV delay is programmed. An intrinsic QRS is sensed before the end of the AV delay, and ventricular pacing is inhibited. Note that bottom panels illustrate modes with ventricular sensing.

Paced AV delay

This is the delay (Figure 1.14a) between the delivery of the atrial pacing stimulus, and the delivery of the ventricular pacing stimulus. It therefore occurs in modes in which both chambers are paced. Examples would be DDD mode, DDI mode, and DOO mode.

Sensed AV delay

This is the time interval between a sensed atrial event, and the delivery of a paced ventricular stimulus. The sensed AV delay (Figure 1.14b) will therefore only be applicable to a pacemaker that is programmed in a mode including sensing in the atrium, pacing in the ventricle, and a triggered response. Examples of such settings might be DDD or VAT.

AV delay and intrinsic AV conduction

If intrinsic conduction is more rapid than the duration of the programmed AV delay (Figure 1.14c and d), the intrinsic QRS will inhibit ventricular pacing, so long as the pacing mode includes ventricular sensing.

Upper rate limit

There are two types of upper rate limit: upper sensor rate, and upper tracking rate. To have an upper sensor rate, the rate-response sensor must be programmed "on" (see section on "Basic pacemaker programming – sensing, pacing, and refractory periods"). In other words, the mode (see section on "Pacing modes") must be rate responsive (e.g. VVIR or DDDR). To have an upper tracking rate, the device must be in a triggered or tracking mode (see sections on "Conceptual building blocks of pacemaker function and Pacing modes").

Upper sensor rate limit

A single or dual chamber pacemaker in a rate-response mode (see section on "Basic pacemaker programming – sensing, pacing, and refractory periods") will have an upper sensor rate. This is the maximum rate at which the sensor will drive the pacemaker. For example, if the sensor upper rate is set at 120 bpm, then the pacemaker will pace at 120 bpm when the sensor is maximally activated (unless the patient's heart rate is even higher and inhibits pacing).

Upper tracking rate

In a dual chamber pacemaker programmed to a *triggered mode* (e.g. DDD or VAT), the upper tracking rate is the maximum atrial rate at which a pacemaker will deliver a ventricular pacing stimulus following each sensed atrial beat (i.e. in a 1 : 1 ratio). For example, if the upper tracking rate is set at 120 bpm, and the atrial rate is 130 bpm, the pacemaker will not deliver a ventricular pacing stimulus after each P wave. If it did, then it would be pacing the ventricle also at 130 bpm, and this would be said to "violate" the upper rate limit. If programmed correctly, the pacemaker rate will plateau at about 120 bpm. If the atrial rate is 130 bpm, and the ventricular rate is 120 bpm, this will have an appearance somewhat like Wenckebach conduction (see Figure 1.15). This, therefore, is sometimes referred to as pseudo-Wenckebach or pacemaker-Wenckebach behavior. The programming issues surrounding upper rate behavior are discussed below.

Upper rate behavior

Upper rate behavior describes the response of a dual chamber pacemaker, programmed in a tracking mode, to increasing atrial rates. The upper rate behaviors described below will be most apparent in patients who are dependent on the pacemaker for AV conduction. In patients with intact AV conduction, *competition* from intrinsic conduction (see section on "Competition and fusion") may partially mask these behaviors. An example of a situation in which the upper rate response of a pacemaker would be important would be a patient with complete heart block, who has a dual chamber pacemaker programmed in DDD mode, and is exercising. Upper rate behavior combines the concepts of AV delay, upper tracking rate (discussed in the section on

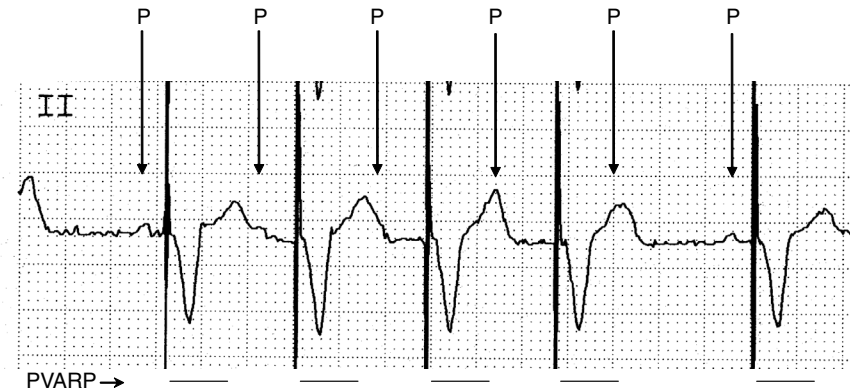

Figure 1.15 Upper rate behavior of a properly programmed pacemaker. The sinus rate is 120 bpm, and the pacemaker upper tracking rate is 110 bpm. The arrows indicate P waves. The heavy vertical black lines are ventricular pacing stimuli. The horizontal black bars below the tracing indicate the duration of the PVARP after each pacing stimulus. The pacemaker delays the ventricular stimulus (increases the AV delay) progressively after each P wave, so that the upper rate limit is not violated. When the penultimate P wave falls into the PVARP, it is not tracked and the cycle begins again.

"Basic pacemaker programming – timing cycles"), and PVARP (see section on "Basic pacemaker programming – sensing, pacing, and refractory periods"). If the atrial rate becomes fast enough, the pacemaker will stop tracking every atrial beat. Depending on the atrial rate, and pacemaker programming, the ECG may have the appearance of Wenckebach AV block (sometimes called pacemaker Wenckebach or pseudo-Wenckebach) or sudden 2 : 1 AV block. Under some circumstances, the pacemaker may stop tracking all atrial beats (mode switch).

Wenckebach upper rate response

When the atrial rate just exceeds the upper tracking rate, the pacemaker prolongs the AV delay progressively so that the ventricular pacing stimuli are not delivered at a rate faster than the upper rate limit (Figure 1.15). Therefore, as the AV delay gets progressively longer, the P wave gets closer to the preceding paced ventricular beat, until eventually the P wave is within the PVARP of the preceding beat, and that P wave is not tracked. This is the way that a properly programmed dual chamber pacemaker will behave when the atrial rate increases to, and is then above, the programmed upper tracking rate.

2 : 1 upper rate response

Sometimes, a pacemaker is improperly programmed such that the P wave encroaches on the preceding PVARP at an atrial rate at or below the upper rate limit of the pacemaker. In this case, the AV delay will not prolong. Instead, the pacemaker will suddenly track 2 : 1. That is, it will deliver one

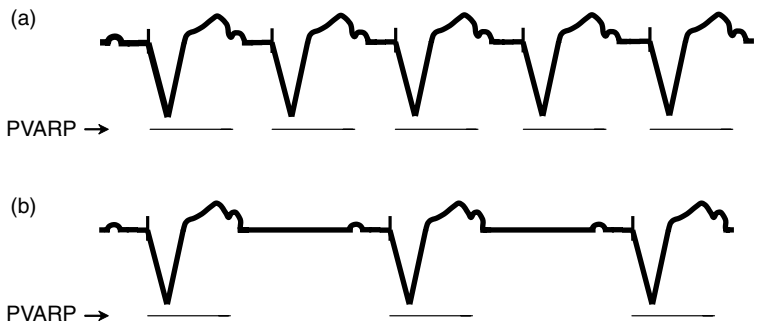

Figure 1.16 Upper rate behavior of an improperly programmed pacemaker. This patient does not have intrinsic AV conduction. The sinus rate is similar to Figure 1.15. The horizontal black bars below the tracings indicate the duration of the PVARP. Note that both the programmed AV delay, and the PVARP are longer than in Figure 1.15. In (a), the sinus rate is just slow enough so that the P wave does not fall into the PVARP of the preceding paced ventricular beat. In (b), the sinus rate has increased slightly, such that every second P wave falls into the PVARP of the preceding paced beat. P waves falling in the PVARP are not tracked, and so ventricular pacing does not occur after these P waves. The result is a sudden change from 1 : 1 to 2 : 1 AV conduction. (If the programmed lower pacing rate is greater than the 2 : 1 block rate, then ventricular pacing will not fall below this rate.) To allow 1 : 1 tracking at the sinus rate shown in the figure, the pacemaker must be reprogrammed to a shorter PVARP or shorter sensed AV delay, or both.

ventricular pacing impulse for every two atrial impulses (Figure 1.16). For example, a pacemaker programmed in this way might suddenly change ventricular pacing rate from 120 bpm (1 : 1 AV conduction) to 61 bpm (2 : 1 AV conduction) as the patient's sinus rate increased from 120 to 122 bpm. A sudden decrease in heart rate like this during exercise would normally cause symptoms.

Proper programming of upper rate limit, sensed AV delay, and PVARP will ensure a smooth transition from 1 : 1 tracking to Wenckebach, and then to 2 : 1 tracking as the heart rate increases. The details of how to program a pacemaker for correct upper rate behavior are beyond the scope of this chapter, but can be found in the references listed in the bibliography.

Mode switching

Mode switching is, as the term suggests, an automatic change from a triggered mode (e.g. DDD) to a nontriggered mode (e.g. DDI). This feature was developed to deal with the problem of the response of a pacemaker programmed in a triggered mode to atrial tachyarrhythmias, such as atrial fibrillation. Such rapid atrial arrhythmias would otherwise cause sustained high ventricular rates in triggered modes. Ventricular tracking of a rapid atrial rate is physiological during exercise, but if the rapid atrial rate is caused by an arrhythmia, ventricular tracking at the upper rate limit is not desirable.

When a pacemaker with mode switch capability, programmed to the DDD mode, senses a very rapid atrial rate, it automatically switches to a nontracking mode. (The patient may still have rapid intrinsic conduction, and a rapid ventricular response, but it will not be due to the pacemaker.) In practice, the pacemaker uses an atrial rate threshold to distinguish sinus tachycardia from atrial arrhythmias such as atrial flutter, which can have an atrial rate of 300 bpm, or atrial fibrillation, which is even faster. When the atrial rate falls below the rate programmed for mode switch, then the pacemaker changes back to a tracking mode.

Competition and fusion

Competition describes the relationship between intrinsic beats and paced beats in a chamber, when both are present. If the surface QRS morphology is a blend of the intrinsic QRS and the paced QRS, it is said to be *fused.* There can be competition between an intrinsic pacemaker (such as the sinus node) and an implanted pacemaker (Figure 1.17). There can also be competition between intrinsic AV conduction and dual chamber pacing of the ventricle (Figure 1.18). If intrinsic AV conduction is intact, then ventricular pacing that follows a sensed or paced atrial beat may *compete* with intrinsic QRS complexes resulting from intact AV conduction. In other words, there will be a race to capture the ventricle. If the electrical activity resulting from the paced beat spreads throughout the heart before intrinsic conduction progresses through the His-Purkinje system, then the QRS complex is broad, and is said to be *fully paced*.

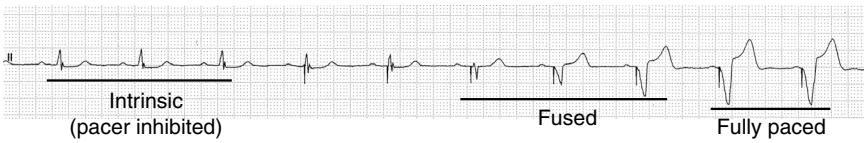

Figure 1.17 VVI pacing competing with sinus rhythm. This illustrates fusion, as well the concepts of inhibition and competition. The pacemaker is programmed in VVI mode at a rate of 60 bpm. The patient's sinus rate is initially 61 bpm at the beginning of the strip, but then slows slightly to about 58 bpm by the end of the strip. Thus the sinus node *competes* with the pacemaker for control of the heart rhythm. At the beginning of the strip, the pacemaker is *inhibited* by sinus rhythm (no pacemaker stimuli are seen), and so the *intrinsic* QRS morphology is seen. At the end of the strip, the ventricle is fully paced, and a paced QRS morphology is seen. (Note that the P wave is not affected, as this pacemaker is not programmed to pace or sense in the atrium.) In the middle of the strip, the QRS morphology is a combination of the intrinsic QRS seen on the left of the strip, and the paced QRS seen on the right of the strip. It is therefore said to be *fused*. (The fourth and fifth complexes on the strip show a pacer stimulus artifact in front of a fully intrinsic QRS. This is called pseudofusion.)

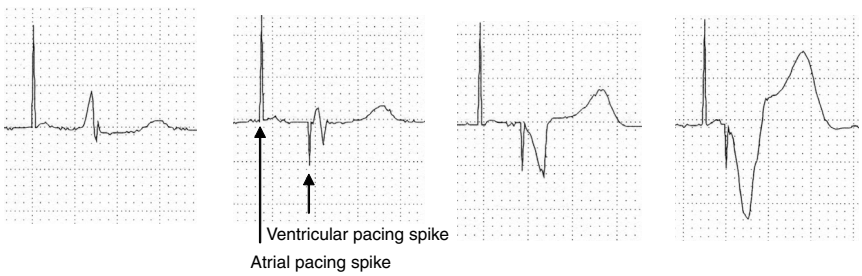

Figure 1.18 Competition between DDD pacing and intrinsic AV conduction. The large amplitude signal before each P wave is the atrial pacing stimulus artifact. The pacemaker is set in DDD mode. The AV delay is programmed to progressively shorter values in each panel from left to right. [(a) 300 ms, (b) 220 ms, (c) 190 ms, and (d) 100 ms.] (a) shows intrinsic AV conduction following a paced atrial complex. Ventricular pacing is inhibited by the intrinsic QRS complex, which therefore must have been sensed by the ventricular channel of the pacemaker prior to the end of the programmed AV delay. (b) and (c) show fusion of ventricular pacing and intrinsic conduction. (d) shows a fully paced QRS complex.

Frequently asked questions: interpretation of pacing behavior on telemetry and ECGs (questions that arise from normal pacemaker behavior)

Arrhythmia services frequently field questions regarding pacing behavior seen on telemetry monitors and on ECGs. As a general rule, if the pacemaker or defibrillator was implanted more than a few weeks ago, and is followed on a regular basis, malfunction is quite rare. The vast majority of questions we receive are the result of normal pacemaker behavior. The following section includes frequently asked questions grouped by the type of pacemaker behavior, and a description of normal pacemaker behavior that may account for them.

"I don't see pacemaker spikes"

1 The pacemaker may not be pacing: this is usually due to inhibition by the patient's intrinsic heart rate, which may be faster than the lower rate limit of the pacemaker.

2 The pacemaker may be pacing, but the stimulus artifacts (spikes) are not visible: unipolar pacing stimulus artifacts are large and nearly always visible. Bipolar pacing stimulus artifacts, however, may be too small in amplitude to see. In addition, certain monitors do not have sufficiently rapid frequency response to detect pacemaker stimuli in their usual recording mode, and must specifically be set to detect pacing.

"The pacer is pacing too slowly"

1 Inaccurate heart rate reading from the cardiac monitor: a cardiac monitor's numerical display of heart rate may be inaccurate. The correct way

to determine heart rate is to print an ECG strip and measure the heart rate manually.

2 Hysteresis: hysteresis is the term for a pacemaker function that allows heart rates to go below the lower rate limit of the pacemaker. If the pacemaker is set to a lower rate limit of 60 bpm, with a hysteresis of 50 bpm, then the pacemaker will allow the intrinsic heart rate to drop until it reaches 50. At this point, the pacemaker will begin pacing at the lower rate limit of 60. It will continue to pace at that rate until the patient's intrinsic heart rate becomes faster than the pacemaker's lower rate limit. Once the intrinsic heart rate takes over from the pacer, then the intrinsic heart rate may again drop from above 60 bpm down to 50 bpm before pacing again occurs.

3 Sleep function: some pacemakers can be set for a reduced pacing rate during the time period when the patient would be expected to be asleep, so that the patient is not bothered by pacing that is more rapid than is physiologically necessary during sleep.

"The heart rate is going too fast"

1 Intrinsic rhythm: it is important to remember that pacemakers treat only slow heart rhythms. Pacemakers (with rare exceptions) cannot slow a rapid rhythm. (Defibrillators may treat certain types of rapid heart rhythms; this will be presented in a later chapter.) To distinguish between pacing and intrinsic rhythm, look for pacing stimulus artifacts before the atrial or QRS complexes. If these are not visible, and if the QRS complexes are narrow, then the rhythm is probably intrinsic. A rapid and markedly irregular rhythm usually suggests atrial fibrillation.

2 Tracking of atrial rapid rhythm: a pacemaker set to DDD, or another triggered mode, will track a rapid atrial rhythm. Each atrial beat will be followed by a ventricular paced beat (unless inhibited by more rapid intrinsic conduction) up to the *upper tracking rate*. The upper tracking rate can be programmed: 120 bpm is a common default setting, but it can range up to 180 bpm. The pacemaker will not pace the ventricle any faster than the upper rate limit is set (see discussion of upper rate limits above). Ventricular tracking of sinus or atrial tachycardia is a common source of confusion. In order assess this behavior, look carefully at the 12-lead ECG to see if there are intrinsic P waves before every paced ventricular beat. If so, the rapid ventricular pacing is due to tracking of a rapid atrial rate (see Figure 1.9 above). The treatment for this (if needed) is directed toward the cause of the rapid intrinsic atrial rate.

3 Rate response: as noted above, current pacemakers and defibrillators can be set to be rate responsive. Increased minute ventilation or motion of the device can sometimes inappropriately activate the sensors. Examples might be hyperventilation, physical therapy, manipulation of the shoulder area. To make this diagnosis, examine a 12-lead ECG. If the pacemaker is in DDD mode, there will be dual chamber (AV sequential) pacing. If there is atrial pacing before every ventricular paced beat, then this diagnosis is in play.

If the atrial activity is intrinsic, then it is simply ventricular tracking of a rapid atrial rhythm (see item 2 above). If the pacemaker is a single chamber ventricular pacer, then atrial activity will not bear a constant relationship to the paced ventricular activity. If the device is a single chamber atrial pacer, then of course, only the atrium will be paced, with intrinsic conduction to the ventricle.

4 Rate smoothing algorithm: some pacemakers and defibrillators can be set to smooth out heart rate variations. This feature prevents abrupt slowing of the heart rate, which may be responsible for certain arrhythmias. Premature beats, such as a run of premature atrial complexes (PACs) or PVCs, may be followed by pacing above the lower rate limit. The pacing rate will then gradually slow down, unless there are more premature beats.

5 PMT: this is discussed in the section on "Basic pacemaker programming – sensing, pacing, and refractory periods" under the heading "Refractory period," and in Figure 1.13.

"Pacemaker spikes are where they shouldn't be"

1 Monitor artifact: monitors that do not have sufficiently fast frequency response to actually record pacemaker stimuli may attempt to mark pacemaker depolarization by using a vertical black line (i.e. these monitors create a stimulus artifact). If the marking is incorrect, then these lines may appear in places where pacemaker depolarization is not actually present.

2 Ventricular sensing which occurs late in the QRS: as noted above (see section on "Conceptual building blocks of pacemaker function"), it is not the onset of the QRS complex that inhibits the ventricular channel output, but the actual arrival of the sensed R wave signal at the ventricular channel of the pacemaker via the ventricular lead. This is likely to happen after the onset of the QRS complex, and the pacemaker can deliver a ventricular pacing stimulus before it is "aware" that there is a QRS complex in progress.

3 Crosschannel blanking and safety pacing: this is a common source of pacing stimulus artifacts occurring in the ST segment (Figure 1.19).

"What is it?"

If a ventricular sensed beat occurs at the same time as an atrial paced beat, the pacemaker will deliver a ventricular pacing stimulus. This feature is usually activated when a PVC occurs at exactly the time that the atrial pacing stimulus is delivered, so that the pacemaker senses a beat in the ventricular chamber at the same time that it is delivering an atrial pacing stimulus. The pacemaker will then deliver a ventricular stimulus, even though it just sensed a ventricular event, resulting in a pacemaker stimulus later in the QRS complex or in the ST segment of the PVC (Figure 1.19). This is frequently mistaken for pacemaker malfunction. The way to determine if normal pacemaker behavior is occurring, is to march out the atrial stimulus artifacts using calipers. If there is an early ventricular depolarization (a PVC), which falls at the same time as the atrial stimulus, and there is a ventricular stimulus in the ST segment, or T wave, this is normal pacemaker behavior.

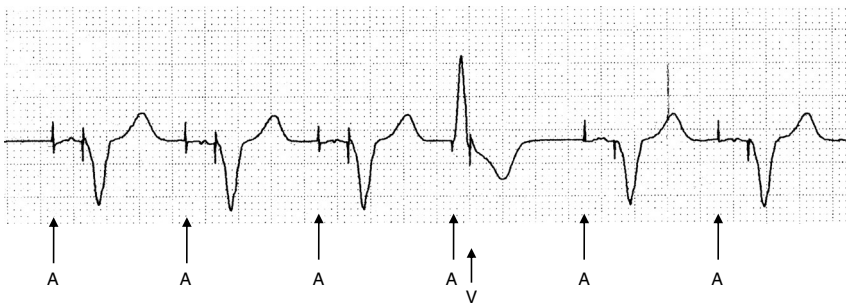

Figure 1.19 Crosschannel blanking/safety pacing behavior. Atrial pacing stimulus artifacts are labeled "A". A PVC happens to occur simultaneously with the fourth atrial pacing stimulus. The pacemaker thus has sensed an event on the ventricular channel, occurring nearly simultaneously with the pacing output on the atrial channel. The concern is that the sensed event on the ventricular channel might actually be the atrial pacing stimulus, rather than an intrinsic QRS. (The pacemaker cannot tell the difference; they both would be simply an electrical signal.) To make sure that a ventricular beat occurs, the pacemaker delivers a ventricular pacing stimulus V. If there is already an intrinsic QRS complex, the pacing will do no harm, but if the ventricular channel is instead sensing the atrial pacing stimulus, a ventricular paced beat may prevent asystole. The difference between this AV delay and the normal paced AV delay depends on the manufacturer. To recognize this, march out the atrial stimulus artifacts. If one coincides with a PVC, and is followed by another stimulus artifact, consider this type of behavior.

"Why do pacemakers have this feature?"

An understanding of the reason for the feature may aid in recognizing it. In dual chamber pacemakers, there is the concern that atrial pacing might be sensed by the ventricular lead. This might occur if the atrial pacing output is large in amplitude, is close to the ventricle, or if the atrial pacing lead dislodges and falls into the ventricle, and paces without capturing the ventricle. If the ventricular channel is inhibited by the atrial pacing stimulus which the pacemaker interprets as a QRS complex, then asystole may result if there is no intrinsic ventricular rhythm.

Differential diagnosis: failure of the pacemaker to sense in the ventricle, or oversensing in the atrium followed by triggered ventricular activity, can be a cause of similar behavior. These subjects are beyond the scope of this chapter. If there is any question about the behavior, the strip should be reviewed with a pacemaker specialist.

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Glossary

Asynchronous: Pacing that is not inhibited by intrinsic beats (see Figure 1.11).

Bipolar pacing: Pacing stimulus delivery between the tip and the ring electrodes of a bipolar pacing lead.

Bipolar sensing: Sensing of intrinsic electrical activity between the tip and the ring electrodes of a bipolar pacing lead.

Capture: The actual excitation or depolarization of cardiac tissue by a pacing stimulus. A pacing stimulus might be too weak to actually depolarize surrounding cardiac tissue, or the tissue might be refractory, in which case the pacing stimulus is said to *fail to capture*.

Channel: The lead input and circuitry on a pacemaker dedicated to a particular chamber of the heart. For example, in a dual chamber pacemaker, the ventricular channel receives sensed beats from the ventricular lead, and transmits the ventricular pacing impulse to the ventricular lead.

Compete: What two independent sources of depolarization may do (see Figures 1.17 and 1.18).

Depolarized: The initial state of activation of cardiac tissue.

Excitable: Cardiac tissue is said to be *excitable* when it can be activated by a pacing stimulus or an intrinsic wave of depolarization. The opposite of this is *refractory*.

Fully paced: A beat or series of beats that result (at least as far as is visible on the surface ECG) only from pacing, in contrast to *fused* beats or *intrinsically conducted* beats.

Fused: The QRS appearance of beats that result from a combination of activation via a pacing stimulus, and intrinsic activation, occurring simultaneously.

Heart block: The absence of intrinsic conduction between the atria and the ventricles.

*Intrinsic beat (*also *intrinsic depolarization, intrinsic complex)*: A P wave or QRS complex arising from the heart's own electrical activity, in contrast to a *paced beat*.

Intrinsic conduction: Conduction of an atrial impulse (sensed or paced) via the His-Purkinje system, causing an intrinsic ventricular QRS complex. Intrinsic conduction may be absent, in which case there is heart block, or masked by ventricular paced beats occurring before the intrinsic impulse has had a chance to make its way through the His-Purkinje system to activate the ventricle.

Lead: The insulated wire that runs from the pacemaker or defibrillator pulse generator into the heart.

Mode: A description of the general way a pacemaker is programmed to pace. This is described by a code of letters, which specifies which chambers are paced, and in what ways. See Chapter 3 for a more complete description.

Oversensing: Sensing on a channel of a pacemaker of something other than what that channel is supposed to sense. For example, if the ventricular channel of a pacemaker sensed the T wave, an atrial pacing stimulus, or electrical noise from a lead fracture (and therefore the pacemaker interpreted one of these signals as QRS complexes) the ventricular channel would be *oversensing*.

P wave: Atrial activity, on the surface ECG, or as sensed by the pacemaker or defibrillator.

Paced AV delay: The delay between the delivery of the atrial pacing stimulus and the delivery of the ventricular pacing stimulus.

*Paced beat (*also *paced complex)*: A P wave or QRS complex initiated by pacing, in contrast to an *intrinsic beat*.

Postventricular atrial refractory period (PVARP): The period after a ventricular sensed or paced event, when the atrial channel is refractory.

Polarized: The state of the cardiac cell membrane prior to activation (*depolarization*). This refers to the separation of positive and negative charges on the outside and inside of the cell, respectively.

Programmable: A pacemaker parameter that can be altered by using the manufacturer's programmer is said to be programmable.

Programmer: A portable device that allows communication with the pacemaker or defibrillator via an electromagnetic telemetry link. This link allows downloading of stored information from the pacemaker or defibrillator, and programming of various parameters. Programmers are specific to the pacemaker brand.

Pulse generator: The main body of the pacemaker or defibrillator that houses the battery and circuits.

R wave: The intrinsic QRS complex, as sensed by the pacemaker or defibrillator.

Refractory: Cardiac tissue is said to be *refractory* when it cannot be activated by a pacing stimulus or an intrinsic wave of depolarization. The opposite of this is *excitable*.

Refractory period: When the term is applied to cardiac tissue, this is the period of time after a depolarization during which the tissue cannot be electrically excited. The exact definition is the time between an electrical stimulus that excites a certain part of the heart, and the latest subsequent stimulus that cannot excite the same tissue. When the term is applied to a pacemaker, it refers to the period after a sensed or paced beat during which the pacemaker will ignore another sensed event. This is a programmable parameter.

Retrograde: Electrical activity moving in the direction opposite to normal (which is anterograde).

Sensed AV delay: The time interval between a sensed atrial event, and the delivery of a paced ventricular stimulus.

Sensed beat: An intrinsic R wave or P wave that has been registered by the pacemaker or defibrillator. Similar to *intrinsic beat*, but with the added requirement that the pacemaker has actually sensed (or registered) the beat.

Sensing: The function of detecting the intrinsic electrical activity of the heart.

Sensitivity: A description of the level of the sensing function. The setting of this function gives the minimum amplitude of intrinsic electrical activity that the pacemaker will register. For example, if the sensitivity in the ventricular chamber of the pacemaker is set to 5 mV, the intrinsic R wave must transmit an electrical signal at least 5 mV in amplitude for the pacemaker to register that an intrinsic beat has occurred.

Settings: The way a pacemaker is programmed.

Stimulus: The electrical output delivered by a pacemaker, through a pacemaker lead to the heart, in order to pace the heart.

Stimulus artifact: The mark created by a pacemaker stimulus on the surface ECG or telemetry monitor.

Tracking: A normal behavior of dual chamber pacing in a triggered mode. The pacemaker senses activity in one chamber (usually the atrium) and delivers a pacing *stimulus* in the other chamber (usually the ventricle) after a certain time delay (the AV delay).

Triggered mode: A pacing mode in which a sensed beat triggers a paced beat. This is most commonly used in a dual chamber pacemaker, so that a sensed atrial beat triggers a paced ventricular beat, after an adjustable delay. In this setting, the ventricle is said to be *tracking* the atrium.

Unipolar pacing: Pacing stimulus delivery between the electrode of pacing lead and the shell of the pulse generator.

Unipolar sensing: Sensing of intrinsic electrical activity between the electrode of pacing lead and the shell of the pulse generator.

*Wenckebach upper rate response (*also known as *pseudo-Wenckebach,* and *pacemaker Wenckebach)*: A progressive increase in the interval between an intrinsic atrial beat and the ventricular pacing stimulus, culminating in an atrial beat that is not followed by ventricular pacing, after which the cycle begins again (Figure 1.15). This occurs in dual chamber pacemakers, programmed in a tracking mode, at atrial rates just above the upper tracking rate limit (see section on "Upper rate behavior").