Implantable Cardiac Pacemakers and Defibrillators:
All You Wanted to Know

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Introduction

Advances in pacing and defibrillator technology in recent years, supported by findings of a large number of well-designed, randomized clinical trials have resulted in the increasing application of this technology for the treatment of a variety of cardiac disorders. The result has been a huge increase in the numbers of devices implanted for tachyarrhythmias and bradyarrhythmias, as well as heart failure therapy. As newer implantable devices have acquired increasing functionality, interpretation of their operation has become progressively complex.

This book is intended as an introduction for all medical and allied professionals including cardiovascular trainees, generalists, nonpacing specialists, and associated medical personnel, who want to understand and embrace this expanding field.

Our aim with this book is to remove some of the mystique surrounding pacemakers and defibrillators, taking the reader from basic concepts to complex functions of these devices, covering the indications for their use and common problems encountered with this technology, in a logical, evidence-based manner. We have also taken a "how-to-do-it" approach for certain areas of implantable device function. The inclusion of these sections will be of interest for trainees and those who do not normally deal with the technical aspect of the discipline. This type of information is often difficult to acquire, and normally learned at the bedside, or in the clinical electrophysiology laboratory.

To many, implantable pacemakers and defibrillators appear as unfathomable black boxes. These devices are in fact pieces of hardware that function with intrinsic logic. It is an introduction to these concepts that we seek to convey with this book. We have brought together here a group of authors who have gone to extraordinary lengths in order to demystify the function of pacemakers, implantable defibrillators, and cardiac resynchronization devices with the hope of improving the readers' understanding of these arrhythmia devices and their functions.

It should be recognized by all that this field is moving rapidly, as a result of both technological advances as well as clinical data derived from clinical trials. Nevertheless, we anticipate that the fundamental principles underlying pacemaker and defibrillator function outlined herein will remain valid for the foreseeable future.

Anthony W.C. Chow and Alfred E. Buxton

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.

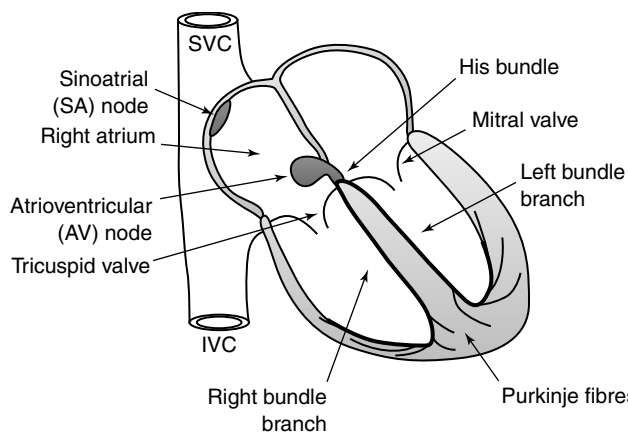

Figure 1.1 Schematic of conduction system anatomy

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

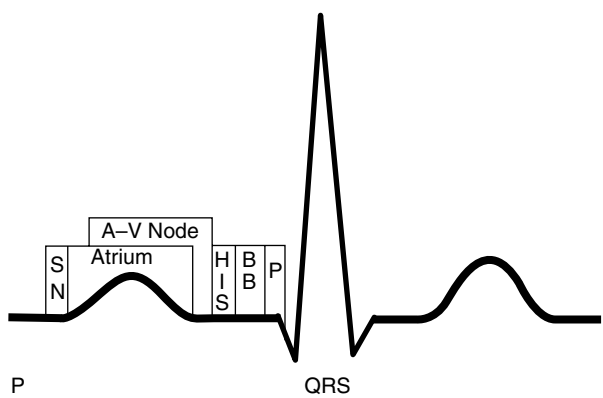

Figure 1.2 The conducting system is reflected in the normal QRS complex. SN = sinus node, His = His bundle, BB = bundle branches, P = Purkinje fibers.

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

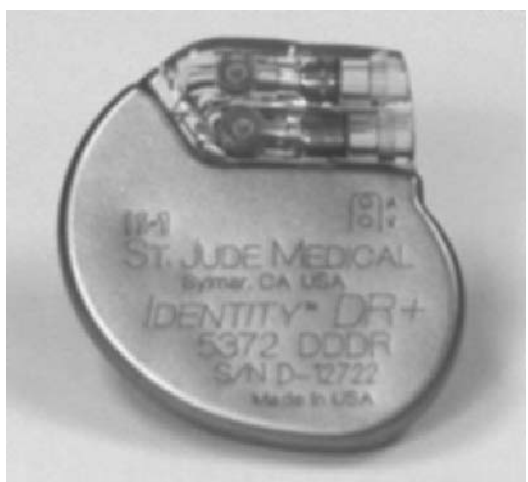

Figure 1.3 Pacemaker pulse generator. Dimensions: 4.4 cm × 5.2 cm × 0.6 cm. Courtesy of St. Jude Medical.

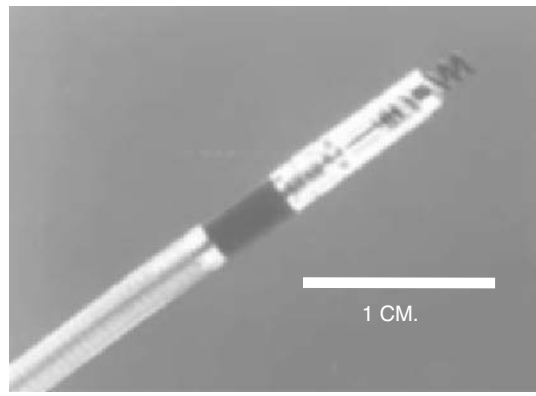

Figure 1.4 Active fixation lead. Courtesy of St. Jude Medical.

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

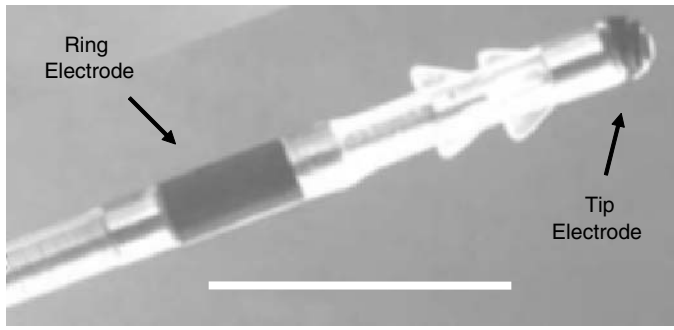

Figure 1.6 The two electrodes of a bipolar pacing lead. The white bar is 1 cm. Courtesy of St. Jude Medical.

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.