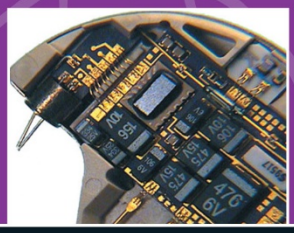

THE NUTS AND BOLTS OF

Cardiac Pacing

SECOND EDITION

Tom Kenny

WILEY-BLACKWELL

The Nuts and Bolts of Cardiac Pacing

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2ND EDITION

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Introduction

This book was first published in 2005, but the idea for this book dates back at least 10 years earlier. I was a former clinician who had just taken a job with a pacemaker manufacturer so I could educate clinicians about pacemakers. I realized pretty quickly that there was no book for the kind of classes I was starting to teach.

That is not to say that there are not fine books on cardiac pacing. There are many excellent books available, but they tend to be written by pacing gurus for other pacing gurus.

I did not start out wanting to write books – in fact, I tried pretty hard over the years to avoid it – but it seemed to me that this was the book that so many clinicians needed. It was also the one book I needed for my courses that simply could not be found on the market.

Back when I was in the clinic, you learned about pacing only if you absolutely had to know it and you could find somebody to help mentor you. In my background, I learned from Dr Orlando Maytin, Dr Michael Chizner, Barbara Perra, Kathy King, and Eliot Ostrow. These people took plenty of time to educate me in the fine points of cardiac pacing. I owe them a great debt.

In today's hectic clinical environment, many clinicians tasked with managing device patients may not enjoy the luxury of having qualified, willing, and generous mentors to teach them. Most device manufacturers offer excellent training programs to those clinicians who can carve some precious time out of their already jam-packed schedules to participate.

In short, it is more likely than ever that today's clinical personnel have to know pacing and it is less likely than ever that they will find mentors or the time.

That is why I wrote this book. It was intended to be a book on pacing for clinicians who were educated in clinical practice but not necessarily knowledgeable about pacemakers. The book was such a tremendous success that I was grateful for the opportunity this year to go back and update it.

A lot has changed in pacing in even the few years since this book was first published. The DAVID trial, for one thing, has changed the way a lot of people think about pacing. New device features and software have been added.

Yet the basics of pacing are still the same.

If you are a clinician who sees pacemaker patients and feels overwhelmed by pacing technology or if you are a busy clinician who just needs to know more about the pacemaker patients all around you (they're everywhere!), this book was written for you. I hope that pacing experts and novices alike can derive benefit from this book, but my heart has always been with the rookie.

If you are new to the world of pacing, welcome aboard! These tiny but powerful medical devices have literally given millions of people around the world a new lease of life. As they grow more technologically advanced, they also become easier to use – providing you know the basics. This book will introduce you to what pacemakers can do and how they work. There is a lot to learn, so be patient, but it is not really very difficult when you approach it systematically.

As always, I welcome your comments and ideas on this book.

Tom Kenny
Austin, January 2008

1 CHAPTER 1
The healthy heart

The human heart – about the size of a clenched fist – is the center of a complex system designed to help the body nourish its organs with life-giving oxygen and to remove waste products in the form of carbon dioxide from the body. Simple animals, such as insects, have an open circulatory system, in which the heart pumps blood through the body cavity, washing the organs directly. More complex animals, including all vertebrates, have a closed circulatory system, which requires the heart to pump blood throughout a network of vessels. No system of vessels is as complex as that of a human being, and it is so closely related to the heart's function that we frequently talk of the "cardiovascular" or CV system rather than the heart in isolation.

In the human CV system, blood stays in the vessels while oxygen and carbon dioxide are exchanged by diffusion through the vessel walls. So intricate is the human circulatory system that there are actually two complementary networks: a pulmonary circulatory system, designed to get deoxygenated blood to the lungs so it can be "revitalized" with oxygen, and a systemic circulatory system which pumps oxygen-rich blood throughout the body to nourish muscles, tissues, and organs.

At the heart of this elaborate circulatory system is, literally, the heart. At its most basic, the healthy human heart is an efficient and effective pump, beating about 70 times a minute without stopping over the course of a human lifespan. The circulatory system is designed to get oxygen-rich blood where it is needed when it is needed, so the heart regulates its own activities, beating more rapidly during times of increased oxygen consumption and more slowly during periods of decreased demand, such as rest and sleep.

The heart is a muscle with four hollow chambers: two upper and two lower (Fig. 1.1). On top are the atria (singular: atrium), thin-walled, small chambers that take their name from our architectural word "atrium." They are the ante-chambers or front lobby of the heart. The two lower chambers are large, thick-walled, heavily muscled chambers called ventricles. The ventricles are responsible for most of the pumping action of the heart.

While physicians can talk about the heart in terms of atria and ventricles, or upper and lower chambers, it is also possible to talk about the heart in terms of right side and left side. The right side of the

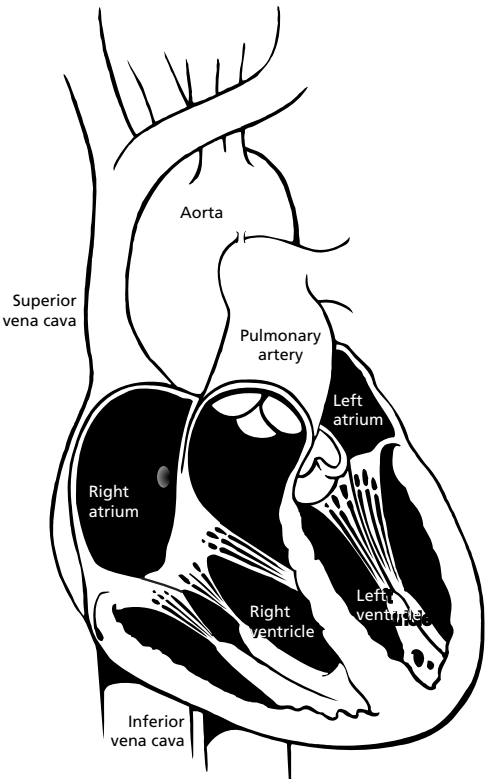

Fig. 1.1 The four chambers of the heart with the largest vessels in the body: the superior and inferior vena cava (which feed deoxygenated blood to the heart) and the aorta (which carries oxygen-rich blood from the heart to the rest of the body).

heart consists of the right atrium and the right ventricle, which are connected to each other through the tricuspid valve. When deoxygenated blood flows back to the heart to become reoxygenated, it first enters the right side of the heart. This deoxygenated blood arrives at the right side of the heart through the body's largest veins: the superior vena cava and the inferior vena cava. (In this case, "superior" and "inferior" refer to physical locations of "above" and "below" the heart.) The right side of the heart pumps this oxygen-depleted blood back out through the pulmonary artery to the lungs, where it picks up much-needed oxygen. Once the blood has received oxygen, the venous system routes the blood back into the heart, this time to the left side.

Loaded with oxygen, the blood reenters the heart through the pulmonary veins into the left atrium and the left ventricle, connected by the mitral valve. The left side of the heart pumps the blood back out to the rest of the body (the systemic circulatory system) through the aorta and the arteries that branch off the aorta.

Cardiac pacing and defibrillation leads for conventional pacemaker and implantable cardioverter defibrillator (ICD) systems are implanted in the right side of the heart. A transvenous lead – i.e. a wire that goes through a patient's vein – can "go with the flow" of blood into the right atrium, through the tricuspid valve, and into the right ventricle. Conventional pacemakers and ICDs have found that pacing the right side of the heart is sufficient to cause a contraction of the entire heart. More recent cardiac resynchronization therapy (CRT) devices require a pacing lead to be implanted in both the right and the left sides of the heart. This poses some technical challenges as a transvenous lead cannot readily travel to this area without going through the heart and then *against* the heart's natural powerful pumping action. CRT therapy and lead placement falls outside the scope of this book, but it is mentioned to give the reader a more complete view of the therapies available.

Myocardial cells

The heart pumps blood through rhythmic contractions or beats, also known as "depolarizations." Depolarization explains what happens to the heart at the cellular level, which is the best way to understand how it beats. Unlike other muscles, which respond to the control of the brain, the heart regulates its own actions without specific input from the brain. To accomplish this, it relies on some of the body's most complex cellular constructions and interactions.

The healthy human heart has two main types of cells: myocardial cells (heart muscle cells) and conduction system cells (electrical cells). Myocardial cells are the ones that make the heart beat.

All heart cells are cylindrical and branch at their ends into one or more limbs. These cardiac cells are held together with intercalated disks sandwiched between them to form a network (Fig. 1.2). Think of myocardial cells as a dense forest of trunks and limbs and branches with intercalated disks forming connections. These intercalated disks help conduct electricity from cell to cell by relaying the impulse.

While myocardial cells do not conduct electricity as rapidly as the electrical cells of the heart, they do have the property of contractility, an ability to shorten and then return to their original length. Contractility allows myocardial cells to stretch and snap back into place. In this way, the myocardium

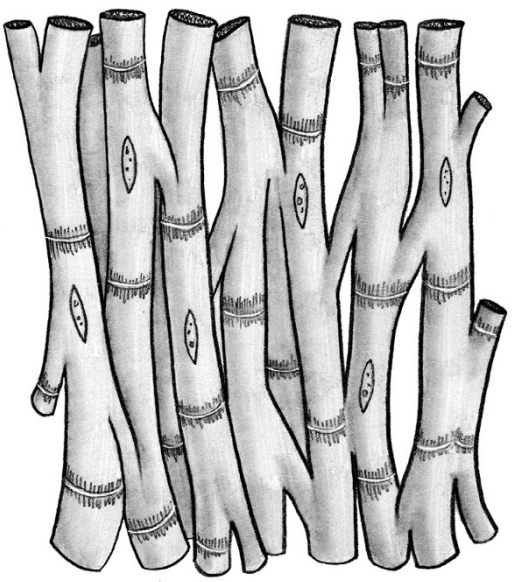

Fig. 1.2 Myocardial cells are specialized cylindrical cells that relax and contract, changing the shape of the heart. Intercalated disks are membranes that include gap junctions for conducting electricity rapidly from one cell to another.

Table 1.1 Common cardiac drugs

	Increases myocardial contractility	*Decreases myocardial contractility*
Sympathomimetics (digitalis, bretylium)	X	
Beta blockers		X
Quinidine		X
Procainamide		X
Excessive potassium		X
Hypovolemia	X	
Anemia	X	
Hypocalcemia		X
Hypothyroidism		X
Emotion	X	
Increased venous return to the heart	X	
Shock		X
Fever	X	
Exercise	X	
Emotion	X	

or heart muscle is able to expand to take in blood and then to contract powerfully to pump the blood back out.

Myocardial contractility responds to a variety of influences. Physical stimuli (including exercise, emotion, fever) and some drugs (sympathomimetics such as digitalis) can increase myocardial contractility, forcing the heart to beat more vigorously. Likewise, other stimuli (shock, hypothyroidism, and others) and some drugs (beta blockers, quinidine, procainamide, and excess potassium) can decrease myocardial contractility (Table 1.1).

The heartbeat

An electrical impulse traveling through the heart causes the cardiac cells to depolarize and contract. The human heartbeat is not one single contraction, but is a precisely timed sequence of four specific events (Fig. 1.3).

Starting with the heart at rest, blood flows naturally into the heart. The valves are open and the heart gets a considerable amount of blood into it through a descriptively named process known as the passive filling of the ventricles. The atria are relaxed in a state known as atrial diastole. When an electrical impulse fires in the heart, the heart beat begins its four-part cycle.

The atria contract (atrial systole) while the ventricles remain relaxed (ventricular diastole). Since the ventricles are already passively filled with blood, this atrial contraction forces even more blood into the ventricles. Known as the atrial contribution to ventricle filling (or "atrial kick") this atrial contraction ensures that the ventricles are filled to the point where they have to stretch to accommodate all of the blood within them. The valves joining atrial to ventricular chambers close, so the ventricles now contain a great deal of blood that cannot backflow into the atria.

There is a brief period of rest – measured in ms (thousandths of a second).

The ventricular cells depolarize forcing a contraction of the powerful ventricular muscles (ventricular systole). This forces blood out over the pulmonary artery (and into the lungs) on the right side or into the aorta (and into the systemic circulatory system) on the left side. This beat is the most powerful action of the heart and it forms the largest complex on an electrocardiogram (ECG).

After contraction, the ventricular muscles repolarize or resume their resting state. The heart resumes the cycle with the passive filling of the ventricles.

Seen on an ECG, a healthy heartbeat shows three distinct wave patterns plus some flat areas of rest (Fig. 1.4). The cycle begins with an atrial beat, shown by the small P wave on the ECG. The flat line between the P wave and the next complex indicates the short rest phase. The large complex, called the

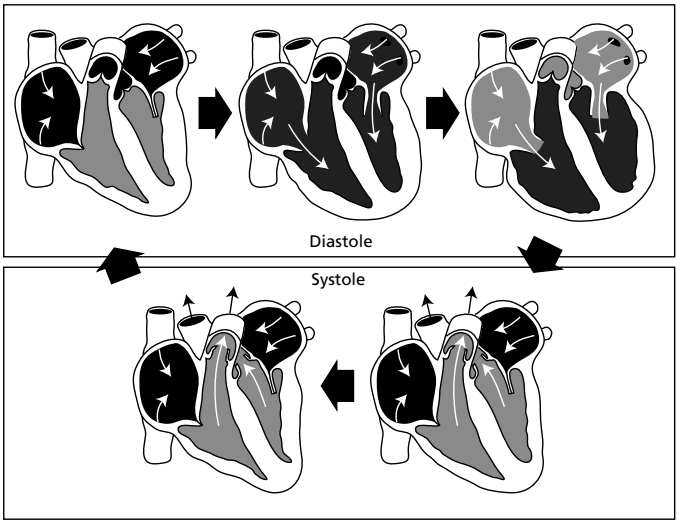

Fig. 1.3 The heartbeat is a sequence of events that begins with ventricular diastole when the ventricles relax, begin to fill, and complete filling. The next phase is ventricular systole when the ventricles contract and empty. Atrial systole helps completely fill the ventricles, and the valves work to ensure that blood moves efficiently through the heart.

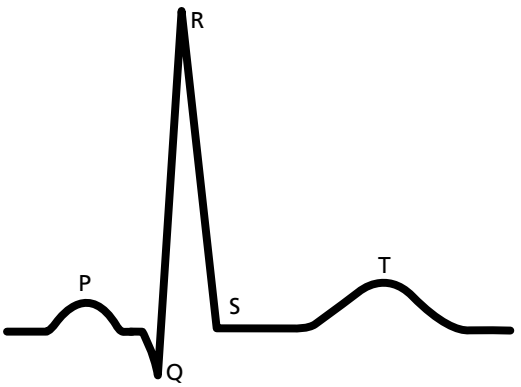

Fig. 1.4 The main waveforms on a surface ECG correspond to the various parts of the heartbeat. The P wave indicates atrial depolarization. This is followed after a short delay by the large QRS complex, which represents ventricular depolarization. A short pause follows, then the ventricles repolarize. This is shown on an ECG by the T wave. The ventricular contraction is the "biggest" event in the cardiac cycle in terms of creating electrical energy, so it appears as the largest portion of the ECG.

QRS complex, is the ECG depiction of the ventricular contraction. As the ventricles are massively large compared to the atria, the ventricular complex dominates the ECG in terms of size. There are three strokes to the ventricular complex, known as the Q, R, and S. Taken together, they describe the ventricular contraction. Another short expanse of flat line shows a rest period. The last wave in the complex is a T wave, which is the electrical depiction of the ventricles repolarizing or resuming their old form.

The pump

The healthy heart beats in a four-part cycle consisting of systole (contraction) and diastole (rest) of upper and lower chambers. When the cycles are precisely timed, the heart is able to pump very effectively. The passive filling of the ventricles combined with the "atrial kick" assure that the maximum amount of blood is brought into the ventricles to be pumped back out. The ventricles – forced to stretch to accommodate the large quantity of blood – contract even more strongly because of this stretch (Starling's law of contractility states that the heart muscle is like a rubber band; the more it is stretched, the more force with which it will snap back). In good working order, the valves in the heart (tricuspid, mitral, pulmonary, and aortic) open clearly and close securely, thus allowing and stopping the flow of blood at the right moments.

The healthy heart relies on a system of vessels to transport blood in and out of the heart. In addition, a separate network of very fine vessels delivers oxygenated blood to the heart muscle itself: the coronary arteries are the heart's own system for nourishment. When these small vessels get clogged or damaged in coronary artery disease (CAD), the heart muscle may be deprived of the oxygen it needs to work properly.

The healthy heart is able to keep a large amount of blood in constant circulation in the body. When the

body consumes more oxygen, the heart increases its pumping action to keep pace, usually by beating faster. In its perfect state, the heart does a remarkable job of keeping the body fueled with oxygen and exchanging waste products. Of course, many things can occur in such a complex system to impair its ability to perform. Some of the main malfunctions of the cardiovascular system – the heart and its vessels – are listed below.

- **Coronary artery disease** in which the network of small arteries that help feed the heart muscle itself become occluded, typically through hyperlipidemia (cholesterol and plaque deposits). This can limit the heart's ability to perform. In extreme cases, blood flow is blocked causing a heart attack and ischemia to portions of the heart muscle itself. A coronary artery bypass graft (CABG) procedure is a typical intervention to treat blocked coronary arteries.
- **Heart failure** refers to the gradual decline in the ability of the heart muscle to pump efficiently. Although there are many manifestations of heart failure, all involve a deterioration of the pumping capacity of the heart. In some cases, the heart muscle gets flabby and enlarged; this is known as dilated cardiomyopathy. In other cases, such as hypertrophic cardiomyopathy, the ventricular wall thickens to the point that not only can it not contract properly, it cannot hold an adequate quantity of blood for a heartbeat. Heart failure is typically treated with drugs, but biventricular device therapy (sometimes called cardiac resynchronization therapy or CRT) holds enormous promise, at least for certain types of patients.
- **Conduction disorders** occur when the electrical system that governs the heart does not work correctly. In such cases, the heart muscle may still be strong, but the electrical signals do not allow the heart to function properly. Conduction disorders, also known as rhythm disorders, are the subject of the next chapter – and the main heart condition treated by pacemakers.

Further reading

Huszar RJ. *Basic Dysrhythmias: Interpretation and Management*. St Louis, MO: C. V. Mosby, 1988.

The nuts and bolts of the healthy heart

- The human heart is a four-chambered pump that circulates blood through a complex network of vessels.
- The heart can be talked about in terms of upper chambers (atria) and lower chambers (ventricles) or right side (right atrium and right ventricle) and left side (left atrium and left ventricle). The right side pumps oxygen-depleted blood through the pulmonary artery over the lungs, while the left side receives the oxygenated blood and pumps it out through the aorta and into the rest of the body.
- It is much easier to implant a pacing lead in the right side of the heart (which is needed for conventional pacemakers) than the left side of the heart (which is required for "biventricular pacing").
- When the heart "beats," it contracts owing to changes at the cellular level called "depolarizations."
- The heart has two types of cells: myocardial cells (which can depolarize) and conduction system cells (which conduct electricity).
- Myocardial contractility (how the heart muscle contracts) responds to many influences, including exercise, drugs, and fever.
- A single heartbeat breaks down into four phases: (a) atrial systole, when the atria contract but the ventricles remain relaxed; (b) rest; (c) ventricular systole, when the blood is pumped out over the body as the atria relax; (d) rest.
- An ECG is a visual depiction of the heartbeat taken from electricity on the surface of the skin. The P wave is the atrial activity. It is followed by the large QRS complex, which represents ventricular depolarization. The T wave after the QRS represents ventricular repolarization or the resumption of the resting state.
- Systole is the contraction phase, and diastole is the resting phase. Thus systolic blood pressure is

Continued p.6

Continued.

the blood pressure that occurs when the heart is pumping. Diastolic blood pressure is the blood pressure that occurs when the heart is at rest.

- Coronary artery disease (CAD) occurs when the network of tiny vessels that feed the heart muscle become occluded and limit the heart's ability to perform.
- Heart failure is the gradual decline in the ability of the heart muscle to pump efficiently. Heart failure may manifest itself as a flabby, enlarged heart (dilated cardiomyopathy) or as the abnormal thickening of the ventricular wall (hypertrophic cardiomyopathy).
- Conduction disorders occur when the heart's electrical system does not work properly. Pacemakers address conduction disorders of the heart.

2 CHAPTER 2
The conduction system

Although we can think of the heart as a pump and the cardiovascular system as a "plumbing system," the heart is also regulated by an elaborate electrical network known as the "conduction system." The heart has unique electrical properties that make it different from any other muscle in the human body. To understand the electrical system of the heart, it is necessary to get down to the cellular level.

All cardiac cells have the ability to conduct electrical impulses. In terms of structure, cardiac cells are cylindrical and branch into two or more limbs at either end. Cardiac cells connect with other cardiac cells at the end of these branches through a type of cellular membrane called an "intercalated disk" (Fig. 2.1). These intercalated disks – found nowhere else in the body – sandwich themselves between the cylindrical cardiac cells. With its profusion of branches and sandwiched disks, cardiac cells form an almost tree-like network.

Electricity can travel through any part of the body, but nowhere else in the body is the pathway for electrical energy as efficient and specific as in the heart. When an electrical pulse enters the cardiac system, it travels rapidly from cell to cell by jumping through the intercalated disks. These intercalated disks facilitate and speed the flow of electrical energy so that an electrical impulse that enters the heart moves swiftly through cardiac tissue. Clinicians sometimes call these intercalated disks "gap junctions" because they join (junction) the spaces (gaps) between cardiac cells in such a way that allows electricity to flow smoothly and very rapidly.

The heart contains two types of cells: myocardial cells (muscle cells responsible for contracting and relaxing to make the heart pump) and electrical cells. Both conduct electricity efficiently, but the electrical cells of the heart have far more intercalated disks and can conduct electricity up to six times faster than myocardial cells. These electrical cells form the pathways for electricity through the heart. In the healthy heart, they allow for the proper timing of all phases of the human heartbeat.

The electrical pathway

These electrical cells form an electrical pathway through the heart which can be considered the heart's conduction system. It begins with a small collection of highly specialized cells known as the sinoatrial (SA) node, located on the high right atrium. The SA node contains a special type of cardiac electrical cell with the property of automaticity. This means these cells have the ability to spontaneously generate electricity.

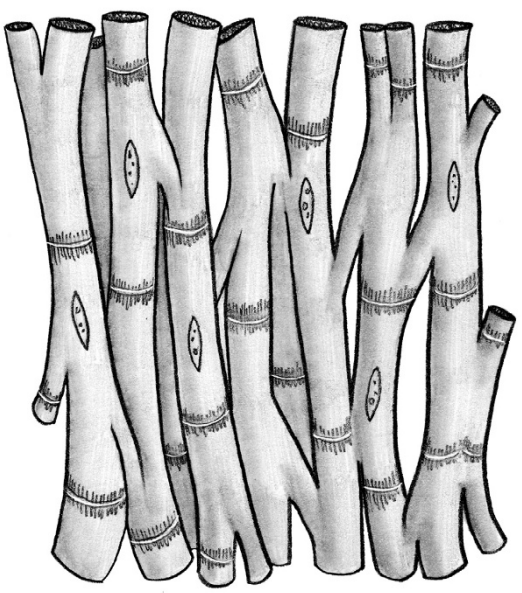

Fig. 2.1 Cardiac cells consist of myocardial cells and specialized electrical cells. Intercalated disks act as "gap junctions" to facilitate and speed up the flow of electricity. In fact, electricity can travel through a gap junction six times faster than it can travel through the myocardial cells.

When working properly, the SA node fires precisely timed electrical output pulses that flow through the conduction system and keep the heart beating properly. The SA node does not require intervention by the brain to know when and how to fire; it happens automatically. For this reason, the cells of the SA node are also called "pacemaker cells" and make up the heart's natural pacemaker. An implantable pacemaker is used when some part of the heart's conduction system fails and an external pacemaker is needed to help the heart beat at the right pace.

The electrical pulse that causes a heartbeat is issued from the SA node. It then travels along a special pathway through the atria down to the atrioventricular (AV) node. The AV node is another group of highly specialized cardiac electrical cells. Located on the right side of the interatrial septum near the opening of the coronary sinus, the AV node acts like a relay station. The electrical energy flows to the AV node where it is delayed for a short time and then allowed to travel down into the ventricles. This AV nodal timing delay is measured in split seconds (ms or thousandths of a second), but this fraction of a second allows the atria to contract and relax prior to the ventricular contraction.

From the AV node, the electrical energy then flows downward through the ventricles along the bundle of His, the right and left bundle branches, and the Purkinje network. These various components are sometimes grouped together and called the His–Purkinje system. The bundle of His is uppermost, and it links the AV node with the right and left branches. The right and left branches – as the name implies – carry the electrical energy to the right and left ventricles. The right and left branches run down the middle of the heart, along the right and left sides of the ventricular septum. As they travel down into the ventricles, the branches get smaller and smaller and form an increasing number of limbs until they become the very fine network of Purkinje fibers that conduct electricity throughout all parts of the myocardium (Fig. 2.2).

As electricity travels through the heart, it causes the myocardial cells (which conduct electricity, but

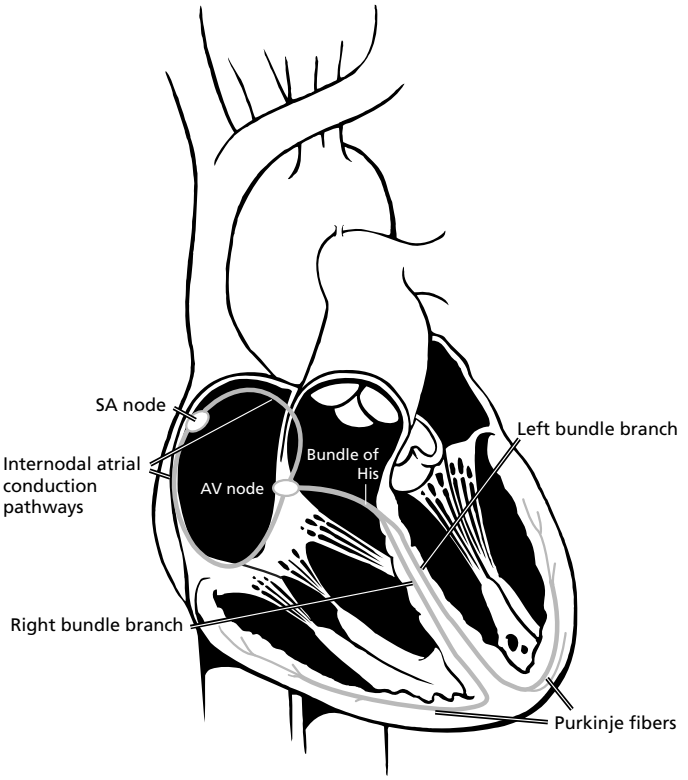

Fig. 2.2 The electrical pathway of the heart starts at the SA node, travels out and across the atria and collects at the AV node. From there, the electricity flows over the bundle of His, down the interventricular septum (through the right and left bundle branches) to the ventricular apex through the very fine network of Purkinje fibers, where the electrical impulse dissipates.

not as quickly as the electrical cells) to contract. Since the heart does not contract as one unit, but rather relies on an atrial contraction, a rest, and a subsequent ventricular contraction and rest, the electricity has to flow in such a way that it causes the contractions to occur at the correct times.

Therefore, the electricity travels rapidly from the SA node down through the atria (this takes about 0.003 s) but then can navigate its way through the AV node only relatively slowly (0.06–0.12 s). This delay gives the atria time to contract and relax before the ventricles contract. Once the electricity reaches the bundle of His at the top of the His–Purkinje system that feeds the ventricles, the electricity travels more rapidly again (0.03–0.05 s). By the time the electricity reaches the end of the Purkinje fibers throughout the ventricular myocardium, the electrical energy has dissipated.

Cellular depolarization and repolarization

All cardiac cells are covered with a semi-permeable membrane that allows certain charged particles (ions) to flow in and out of them. The electricity generated by the SA node and traveling through the healthy heart is mainly the result of positively charged sodium and potassium ions that flow through the semi-permeable membrane of the cardiac cell and change its electrical balance.

The concentration of ions in the cardiac cells gives it an electrical potential (sometimes called "membrane potential") which can be measured in voltage (millivolts mV, thousandths of a volt).

In a resting state, a cardiac cell contains a concentration of negative ions within the cell with a large concentration of positive ions surrounding the cell on the outside. The negatives on the inside (anions) and the positives on the outside (cations) line up almost as opposites, and it is from this that the cellular state gets its name as "polarized." There are two poles: the negatives inside the cell and the positives outside the cell. In this polarized state, the cardiac cell still has a measurable electrical potential, known as "resting membrane potential." Resting membrane potential is higher in myocardial cells and lower in the highly specialized cells of the SA node and AV node.

When an electrical impulse reaches a cardiac cell, it causes that cardiac cell to become permeable to positively charged sodium ions. Positive ions start to flow into the cell, shifting the balance inside the cell from negative to less negative. This decreases the cell's resting membrane potential. When the cell's resting membrane potential falls below a certain level, fast sodium channels open. Just like they sound, fast sodium channels are pores in the cell membrane that allow a quick inflow of positively charged sodium ions. The result is that the inside of the cell quickly becomes positively charged while the cells clustered around the outside are now more negative than positive. Basically, the cell's polarized state is now reversed – or depolarized. The cell's inside is positive and the exterior is mostly negative.

Depolarization causes the myocardial cells to contract, and when they do, positively charged sodium ions start to escape from the interior of the cardiac cell. This outflow of positive ions returns the cardiac cell to its polarized state: negative on the inside with mostly positive ions on the outside. This is known as repolarization or the relaxing of the heart muscle cells as they resume their old shape.

Repolarization is actually a much more complex cellular process than described, involving sodium, calcium and potassium ions. For the purposes of understanding cardiac conduction, these details are not as important as understanding that depolarization and repolarization are cellular processes involving the flow of ions across a cardiac cell membrane.

Fast sodium channels are present in most cardiac electrical cells; these allow the cells to depolarize quickly. The highly specialized cells of the SA node and the AV node do not have fast sodium channels, which would allow them to conduct electricity too quickly. Instead, they have slow calcium-sodium channels. These channels also permit positive ions to enter the cell membrane, but at a much slower rate than the fast sodium channels. The result is that the SA node and the AV node depolarize at significantly slower rates than the rest of the cells in the conduction system.

The action potential

The best way to illustrate the process of depolarization and repolarization of a cardiac cell is through