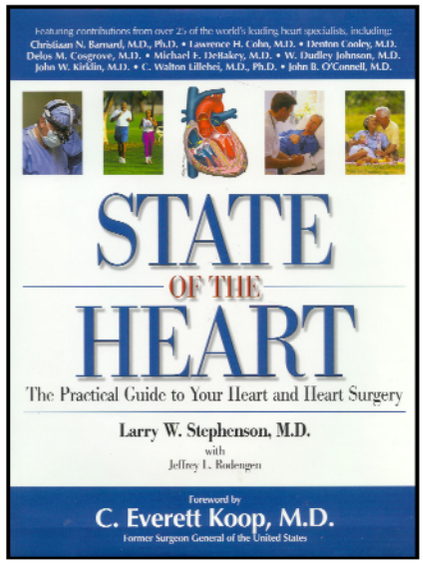

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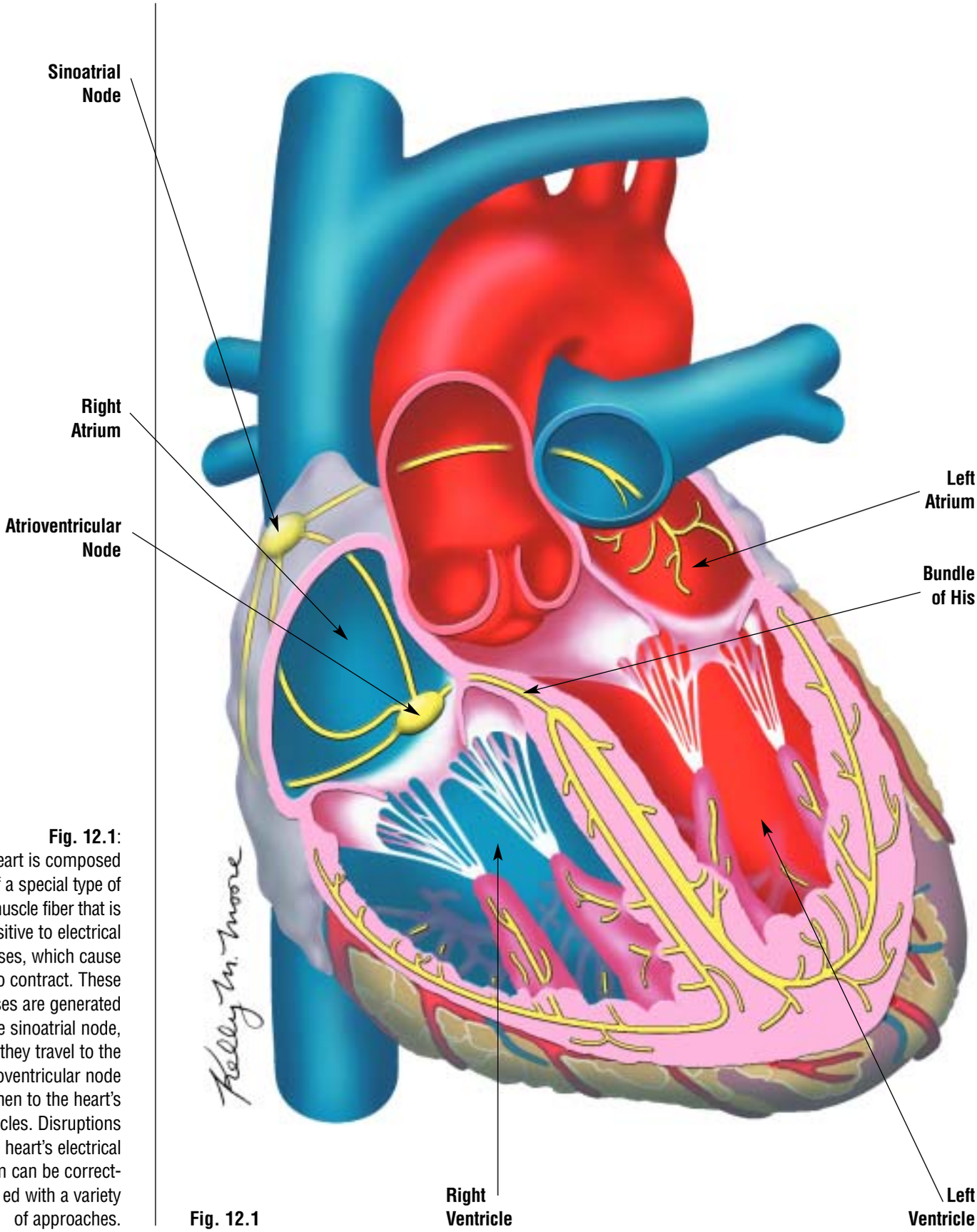


Fig. 12.1: The heart is composed of a special type of muscle fiber that is sensitive to electrical impulses, which cause it to contract. These impulses are generated in the sinoatrial node, then they travel to the atrioventricular node and then to the heart's ventricles. Disruptions in the heart's electrical system can be corrected with a variety of approaches.

Fig. 12.1

CHAPTER TWELVE

ARRHYTHMIAS, PACEMAKERS, AND DEFIBRILLATORS

EVERYBODY HAS AN IRREGULAR heartbeat now and then, and not all types of irregular heartbeats, or **arrhythmias,** are necessarily bad. They can range in severity from simply a nuisance to life threatening, especially those that originate from the heart ventricles.

In most cases, an arrhythmia is caused by a faulty electrical conduction system, or pacemaker cells, in the heart muscle. Abnormal heart rhythms may interfere with the heart's pumping function and therefore may cause fatigue, lightheadedness, a sensation of uneasiness, or sometimes even passing out, particularly if the heart rate is very slow or very fast. Some patients can develop angina-type chest pain from a decreased flow of oxygenated blood to the coronary arteries or from very rapid heart rates.

The normal heart rhythm is called **sinus rhythm**. If there is a problem with the heart rhythm, patients may be referred to a cardiologist who specializes in heart rhythms called a cardiac electrophysiologist. Patients may undergo studies of their heart rhythm called EPS (electrophysiology studies). In this case, wires are guided into the heart through blood vessels in the groin or arm to study its electrical system.

If someone suffers from an arrhythmia, they may need treatment with medication or even an implantable electrical device such as a **pacemaker** or **defibrillator**. These devices monitor the heart's rhythm and intervene in case of certain types of irregularity. There are many companies worldwide that produce heart pacemakers, and each company makes several different types that approach different heart rhythm problems with various design strategies.

Pacemakers

The first successful electrical pacing probably took place in Australia in the 1920s when two doctors supposedly revived a stillborn baby. By the 1950s, doctors were able to control the heart rate in dogs by using external pacemakers.

The real advent in pacing as we know it today, however, is credited to Dr. Paul Zoll from Harvard Medical School. In 1952, he used an external pacemaker on two patients suffering from recurring, prolonged episodes of ventricular standstill (heart ventricles not contracting, therefore not pumping blood). The first patient was a seventy-year-old man with complete

Arrhythmia:
Any abnormal heart rhythm. Also called dysrhythmia.

Sinus Rhythm:
The normal rhythm of the heart that is stimulated by the sinoatrial node.

Pacemaker:
A small, battery-powered device implanted in the chest wall to send electrical impulses to the heart, causing it to contract in a rhythmic fashion. Mechanical pacemakers are used when the body's natural pacemaker is not functioning properly.

Defibrillator:
A device used to electrically shock the heart into a more normal rhythm.

Sinoatrial Node: Also referred to as the sinus node and S-A node. This is the true pacemaker of the heart, located at the junction of the right atrium and superior vena cava. These cells rhythmically discharge electrical impulses that cause the heart to contract. This impulse also travels to the A-V node, and then to the ventricles, causing them to contract.

Atrioventricular Node: Also called the A-V node. A specialized nerve-type tissue located in the wall of the right ventricle. It receives electrical impulses from the sinoatrial node, then relays the impulse to the ventricles, which causes them to contract.

heart block, meaning the atrial heartbeats could not get through to pace the ventricles. The patient had been revived with thirty-four separate intracardiac injections (sticking a needle connected to a syringe through the chest straight into the heart!) of adrenalin given during four hours. Zoll applied electric shocks, two milliseconds long, through the chest wall at frequencies of twenty-five to sixty shocks per minute and increased the intensity of the shocks until ventricular responses were observed. After twenty-five minutes, however, the response became weaker, and the patient died. Many subsequent patients, however, survived.

Shortly afterward, Dr. C. Walton Lillehei and associates reported on a series of patients whose hearts needed pacing after open heart operations at the University of Minnesota. The major difference between Zoll's pacing and Lillehei's was that Zoll used large external electrodes placed on the patient's chest wall, whereas Lillehei attached electrodes directly to the heart during the operation and connected these electrodes to an external power source. In this way, he could pace the heart with much less current, and it was not painful to the patient, in contrast with Zoll's shocks permeating the skin and chest wall. It was also a more efficient way to stimulate the heart. The survival rate of Lillehei's patients with surgically induced heart block, a complication of heart surgery, was substantially improved with the pacemaker.

Only a few years later, Rune Elmquist and Dr. Ake Senning in Sweden developed a prototype of the first totally implantable pacemaker. It had a battery that was small enough for a pocket under the skin and electrodes that were connected directly to the heart. The first unit was implanted in a patient in 1958.

Drs. William Chardack, Andrew Cage, and Wilson Greatbatch from the University of Buffalo School of Medicine in Buffalo, New York, are perhaps better known for their development of a totally implantable pacemaker. In 1961, they reported on a series of fifteen patients in whom they placed the totally implantable pacemakers they had developed.

In these early days, implantable pacemakers were not synchronized with the heart rhythm. They delivered an electrical impulse independent of the underlying cardiac rhythm. During the past forty years, however, enormous progress has been made in cardiac pacing. The number of individuals with artificial pacemakers is unknown. However, estimates indicate that about five hundred thousand Americans are living with a pacemaker, and that each year another one hundred thousand or more patients require permanent pacemakers in the United States alone.

Heart Pacemakers

The most common need for heart pacemakers today occurs when there is an inappropriate slowing of the **sinoatrial node**, or S-A node, which is located in the right atrium and is responsible for generating a normal heart rhythm. Electrical impulses travel from the S-A node through the atrium and arrive at the **atrioventricular node**, or A-V node, which is located at about the junction of the right atrium and right ventricle. From there, the impulse travels through another electrical system that activates the right and left ventricles and stimulates them to contract (Fig. 12.1).

If the A-V node is diseased, the electrical impulse cannot get through to the ventricles. In this case, the A-V node is often able to generate its own electrical impulse, but this impulse tends to occur at a slower rate than that of the S-A node, and the rhythm is not in synchrony with the atrial rhythm. Thus, the atria may contract at one rate, and the ventricles may contract at a slower rate. In fact, the ventricles may not contract at all.

THE FIRST PRACTICAL PACEMAKER

C. WALTON LILLEHEI'S FIRST battery-powered pacemakers were built by Earl Bakken, an engineer and medical equipment repairman at the University of Minnesota.

Bakken gained interest in electricity and medicine from the Mary Shelley novel *Frankenstein; or, The Modern Prometheus.* In the book, Dr. Frankenstein rejuvenates an inert body with electricity. As a boy, Bakken imagined using the same treatment on sick people.

> *"Later, while in graduate school at the University of Minnesota, I started wandering over to the hospital. I got acquainted with people in the labs and the EKG department, and was asked to help repair some equipment. That's when the idea came to set up Medtronic in 1949 as an electronic repair service in the field of medicine."*

Shortly afterward, he met Lillehei, who was working on cross circulation for cardiopulmonary bypass, and sold the surgical team some equipment.

"They wanted lots of monitoring for both the parent and the child, which I was glad to sell them," Bakken said. "Then they wanted to be sure it was running right, so they wanted me in surgery with them. Lillehei used to say I was the only engineer whom he could get to come into surgery; the other engineers wouldn't do it. So I got to know all the residents and interns who later went on to become the heads of surgery all across the country, all around the world."

During the operations, Lillehei was using big, externally powered pacemakers. Although these helped some children who developed heart block, they had the major drawback of needing to be plugged into a wall socket. When the patient needed to be moved, Lillehei's team ran extension cords down the hospital corridors to keep the pacemakers working — even going so far as dropping cords down elevator shafts.

During a 1957 blackout, the pacemakers stopped working in several patients and, according to Bakken's recollection, one child died. Lillehei turned to Bakken and asked him to develop a back-up system. Bakken recently told how this request sparked development of the modern pacemaker:

> *"I envisioned that because the pacemaker was a large AC unit sitting on a cart, we could put an automobile battery on the lower shelf.... If the power failed, the battery would keep it going for several hours. Automobile batteries were six-volt back then. An invertor could change that to a fifteen-volt pulse.*
>
> *"I went back to my garage to think about building that. Then it dawned on me. Why go through all that big cart full of apparatus and end up with a little fifteen-volt pulse? In fact, further work showed we didn't need more than five volts. So that's when I built the first battery-operated, wearable, transistorized pacemaker."*

The pacemaker electrodes were attached to the heart, and the wires ran through the skin to the relatively small battery-powered unit. Bakken's pacemaker provided the foundation for modern pacemakers and also spurred the development of Medtronic into a one of the world's largest medical device companies.

The first wearable, transistorized, battery-powered pacemaker, above, was developed in 1957 by Earl Bakken, middle. The Chardack-Greatbatch pacemaker, below, is a later-generation implantable device.

Earl Bakken

Obviously, if this happens, it is a very serious situation.

The Single-Chamber Pacemaker

The simplest type of pacemaker is called a VVI and is designed to pace the ventricle (Fig. 12.2). It senses the ventricle's electrical activity and is inhibited from pacing if the heart rate is faster than a preset rate. The first "V" in VVI means the heart's ventricle is paced, the second "V" means the heart's electrical activity in the ventricle is sensed by the pacemaker, and the "I" refers to the mechanism by which the pacemaker is turned on and off.

The most common way we connect pacemaker leads is to thread them through a vein in the shoulder area to the right atrium and into the right ventricle, then connect them to what's called a pulse generator (the brains and battery of the system) located in a pocket under the skin.

These pacemakers' electrical impulses can be set so that if the ventricle is naturally contracting at an acceptable rate (usually around sixty or seventy beats per minute, or bpm), the pacemaker will simply monitor the heart rhythm. However, if the patient's heart rate drops below a preset number of beats per minute, the pacemaker takes over. For example, the pacemaker could be programmed so that if the ventricular rate is seventy bpm or faster, the pacemaker remains in "standby" mode. If the rate drops below seventy bpm, the pacemaker kicks in and delivers electrical impulses at the preset rate. This rate is whatever the physician decides to set and varies from patient to patient. The pacemaker does

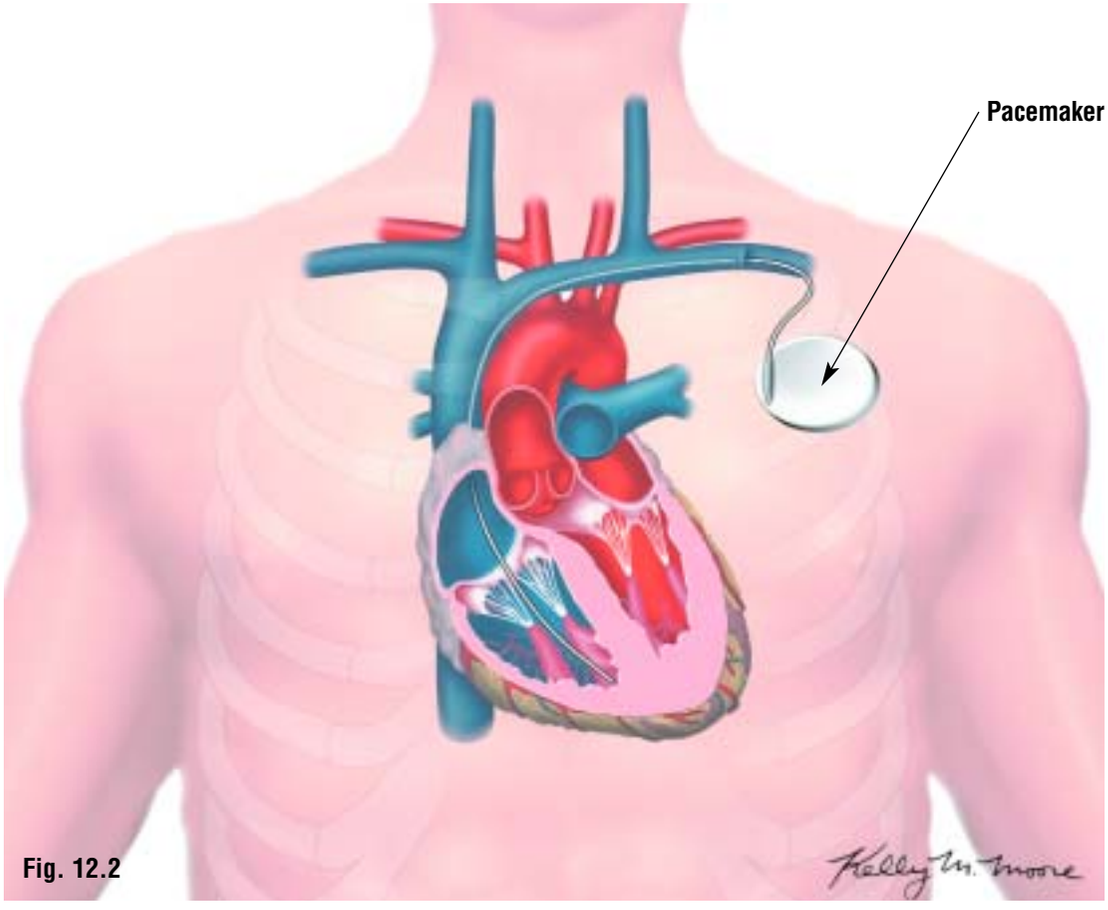


Fig. 12.2

Fig. 12.2: A VVI pacemaker, or single-chamber pacemaker, is designed to pace the ventricle. It is set to send an electrical impulse only when the ventricular contractions slow to an unacceptable rate.

DENTON COOLEY: BUILDING HIS OWN DEFIBRILLATOR

WHEN DENTON COOLEY WAS A surgical resident at Johns Hopkins Hospital in the 1940s, it was not uncommon for seriously ill infants and children with congenital heart defects to develop ventricular fibrillation, a fatal rhythm of the heart's ventricles, during surgery. The usual treatment was to give various medications in hope that the heart rhythm would change back to normal. Sometimes it did not work, and the patient died.

Cooley recently remembered that during the surgery to close a patent ductus arteriosus, or abnormal communication between the aorta and pulmonary artery, the child's heart went into ventricular fibrillation and could not be returned to normal rhythm. Cooley was extremely discouraged by this. He went to see Dr. William Kouwenhouven, who was chairman of the department of electrical engineering and who had been working with cardiac resuscitation for electrocution victims. They spoke about building a defibrillator for the operating room.

Subsequently, based on an article by Dr. Donald Hooker and Kouwenhouven and another by Dr. Claude Beck, Cooley built his own defibrillator with the help of the hospital's machine shop. Unlike most other defibrillators that were used by placing the electrodes on the patient's chest, Cooley's defibrillator allowed the electrodes to be placed directly on the patient's heart in the operating room. It cost about $90 of Cooley's own money to build.

"The defibrillator was used at Johns Hopkins in the operating room for about ten years until commercial devices were available," Cooley said. "My salary then, as a fourth-year surgical resident, was $25 a month."

not actually wait a full minute and count seventy beats before activation. Rather, it measures the time interval between heart electrical impulses so if the next heart impulse is slow to arrive, the pacemaker has already taken over.

Usually patients do not know when their heart is being paced and when it is not. They can sometimes tell when the pacemaker switches on or off, however, just like some people can feel when their heart skips a beat or the heart rhythm changes.

Sometimes the leads or wires can also be attached directly to the surface of the heart, as opposed to threaded inside the ventricles. This requires a surgical incision, usually in the chest or upper abdomen, so the leads can be fastened to the surface of the heart. This might be done in a patient needing a pacemaker whose chest is already open during a heart operation. Leads might be connected directly to the surface of the heart if the patient has an infection in the bloodstream or an artificial heart valve replacing the tricuspid valve.

More sophisticated ventricular pacemakers have a feature that can sense your physical activity level and actually increase the heart rate so more oxygenated blood is pumped to the body tis-

sues. This is helpful in active patients. These devices usually sense changes in body heat or increases in skeletal muscle activity.

Dual-Chamber Pacemakers

The next most common type of pacemaker stimulates and monitors both the ventricles and the atria (Fig. 12.3). This more sophisticated pacemaker is called a DDD pacemaker (each D stands for dual: it can pace both the atria and the ventricles; it can sense the electrical activity in both the atria and the ventricles; and there are dual methods to make sure it turns on or off at the correct time). In addition to the lead connected to the ventricle, it also requires an electrode connected to the atrium. This additional electrode is also introduced through a vein and threaded into the right atrium. The other end of the lead is connected to the pacemaker.

A DDD pacemaker has certain advantages. It will be able to sense your S-A node rhythm and pace the ventricles in harmony with the atria. If you're resting, the S-A node rate may only be sixty bpm, but, if you're active, it may be one hundred. The pacemaker will sense the appropriate S-A node rate and deliver a corresponding impulse to the ventricle. If the S-A node is pacing the atria at an undesirably slow rate, it can also take over and pace the atria. This type of pacemaker allows the ventricles and atria to coordinate their activity.

The DDD pacemakers can be equipped with an additional feature that senses

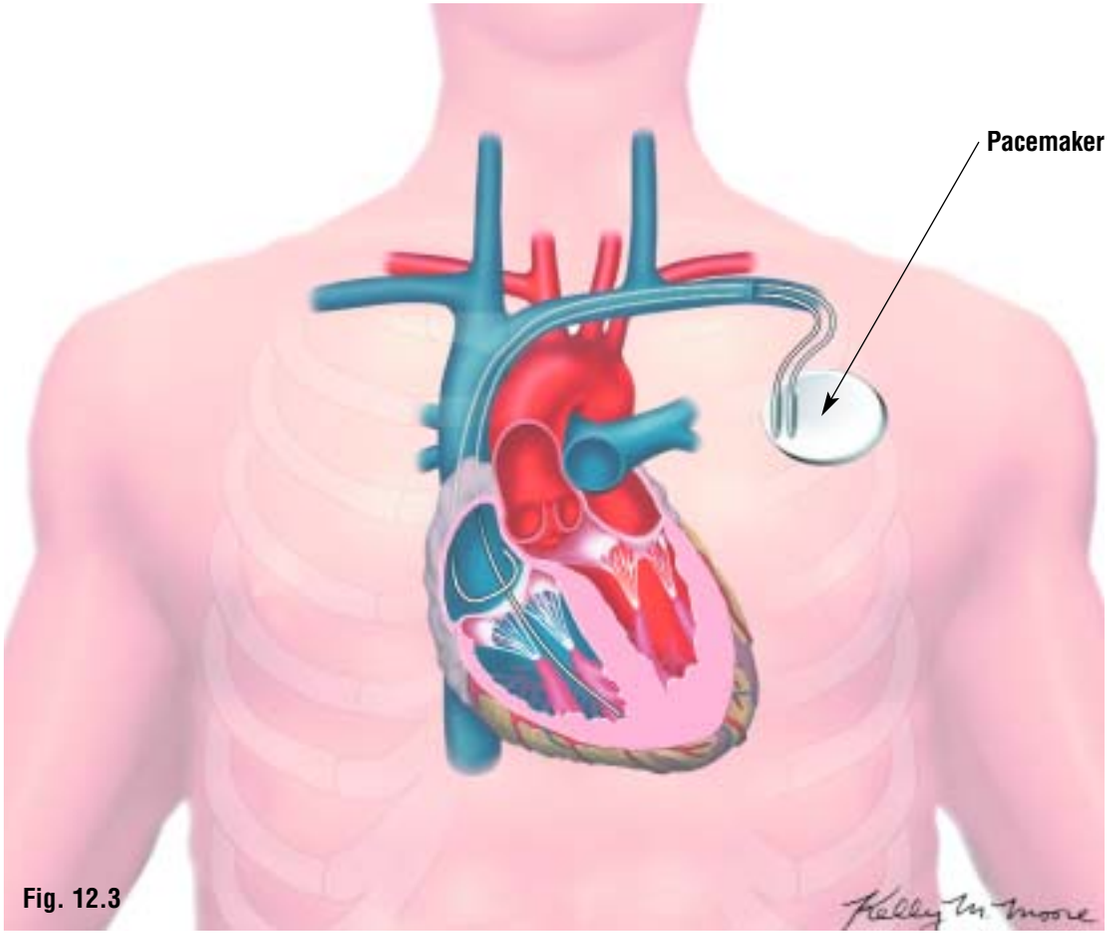


Fig. 12.3: Dual-chamber pacemakers can monitor and pace both the ventricles and the atria.

AKE SENNING AND THE FIRST IMPLANTABLE PACEMAKER

SWEDISH DOCTOR AKE SENNING became a doctor by default. Although he had wanted to be an engineer, his mother urged him towards medicine. That summer in the 1930s when he was signing up for school, he went on a motorcycle holiday and missed the deadline to apply for technical school. With that option closed, Senning pursued medicine. In 1949, he was invited to join the thoracic clinic of the renowned surgeon Dr. Clarence Crafoord, where he was assigned to develop a heart-lung machine.

There, another young surgeon, who also would distinguish himself in cardiac surgery, Dr. Viking Bjork, was also working on a heart-lung machine of his own design. At one point, Senning remembered in a recent interview, he even visited Dr. John Kirklin's lab at the Mayo Clinic to see the first-generation heart-lung machine and, through an accident, had blood sprayed all over a new suit (the suit was saved).

Senning gained experience with external pacemakers during his heart-lung machine experiments when, in some cases, he had to stimulate the heart with electricity to regain a beat.

"In 1955, a friend of mine came from the United States," Senning said. "He had a small pacemaker. When I saw this, I thought, 'We can make it smaller.'" But Senning was concerned about infection because the wires had to run through the skin to the external pacemaker. Senning started with external pacemakers, but many became infected. The next step was to work towards an implantable pacemaker.

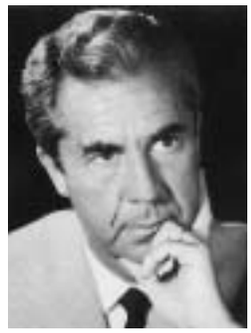
By 1957, Senning and his colleagues had developed what they hoped would become a workable internal pacemaker. But there was skepticism: "The cardiologist said there was no indication; a patient with A-V block can live at least two years. And then you had the priest, who said if you have a heart that God stopped, you shouldn't try to start it!"

Then an agitated woman came to him and pleaded with him to implant a pacemaker in her husband. He had been hospitalized for months and was having twenty to thirty cardiac arrests every day. Senning told her their pacemaker was not ready for human implantation. She replied, "So make one!"

Senning referred her to the engineer Rune Elmquist, who was working on the experimental pacemaker. That day, the woman drove back and forth several times between Elmquist's office and Senning's.

Shortly thereafter, on October 8, 1958, Senning received his pacemaker and placed it in his patient. It functioned for eight hours, then stopped. Senning replaced it with another, which failed several days later. Eventually, the patient survived more than twenty-five years and during that period had several more pacemaker implants.

One of Senning's other major contributions to cardiac surgery was the Senning operation, which he reported on in 1959. This was the first corrective operation for a congenital heart defect called transposition of the great arteries, which until then was usually fatal within the first year of life.



Dr. Ake Senning placed the first totally implantable pacemaker, which he developed with a Swedish engineer.

an increase in physical activity. If the S-A node does not increase its rate with increased physical activity like walking or running, the pacemaker can be programmed to increase the heart rate so more blood is pumped.

There are numerous variations on the VVI and DDD types of pacemakers, but these are the basic principles on which these pacemakers work.

Tachycardia: An abnormally fast heart rate, usually more than one-hundred beats per minute.

Pacemakers for Atrial Tachyarrhythmias

Some pacemakers are designed for patients who have episodes of abnormally fast heartbeats that are generated in the atria. If these fast beats in the S-A node or other areas in the atria reach the range of one-hundred sixty to more than two hundred beats per minute and are transmitted to the ventricles, causing them to contract at an abnormally fast rate, the heart becomes inefficient, and you may become lightheaded or even pass out from less oxygenated blood getting to the brain.

A kind of pacemaker called an anti-tachyarrhythmia pacemaker can sense these rhythms, take them over, and pace the atria into a more normal rhythm. Tachy means fast; arrhythmia means abnormal heart rhythm.

Surgery for Pacemakers

Pacemakers are usually implanted by either heart surgeons or cardiologists. The surgery is done in the operating room, cardiac catheterization laboratory, or electrophysiology laboratory. The pacemakers are usually implanted after local anesthesia is induced with the patient awake. The skin is washed and painted with soap; then sterile drapes are placed over the patient and around the area where the inci-

During an electrophysiology study, physicians are able to locate the exact source of abnormal heart rhythms. This procedure is performed with catheters that are guided into the heart through the groin or arm.

sion will be made. A local anesthetic, usually lidocaine or xylocaine, is injected under the skin in the shoulder area just below the collar bone. Next, an incision is made through the skin about two to two-and-a-half inches long and down to the muscle layer. The tissue is freed up just above the muscle layer to make a pocket for the pulse generator. The size of pacemakers is variable, but they can be as small as a quarter or a silver dollar and a bit thicker. Next, using one of several techniques, a vein in that area is located, and one or two leads are threaded through it into the heart.

The surgery to install pacemakers can take anywhere from a half hour up to more than two hours depending on how difficult it is to get the leads into the best spot in the ventricle and atrium (for the DDD type) for the best pacing and sensing thresholds. Every patient's anatomy is a little different, and the exact best spot to place the lead in the atrium or ventricle can vary from patient to patient, which can account in part for the relative difficulty of insertion in some patients.

Once the wire leads are connected to the pulse generator, the pulse generator is placed under the skin and a fat layer in the shoulder area, and the wound is closed. The pulse generator has a battery and a small, sophisticated computer. Most pacemakers (computer plus battery) are small enough so they are not noticeable under the skin.

The settings of the pacemaker are adjusted through the skin so that no needles or objects need to break the skin barrier. The device used to program your pacemaker is somewhat like your remote control television programmer. The batteries in the pulse generator usually need to be changed about every seven to ten years. When the pulse generators are changed, generally the leads are left in place, but tests are done to check them.

My patients usually go home later that day or the next morning with a prescription for some minor pain medicine, which is usually needed for only a few days. Pacemakers require routine follow-up to ensure proper function and to assess battery longevity.

MANAGING ARRHYTHMIAS

By

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THE HEART'S PUMPING action, which creates the pulse, is generated by a burst of electrical energy that activates specialized cells in the heart muscle. Normally, this electrical activity originates in a structure called the sinus node (or sinoatrial or S-A node), which is located in the right atrium. From there, it spreads across the atria, causing them to contract, or beat.

After stimulating the atria, the impulse travels along a bridge of special conducting tissue called the A-V node to the ventricles. Similarly to the atria, ventricles pump in response to the electricity, thus sending blood out to the organs.

Any abnormalities in this electrical circuit affect the heartbeat. These may give rise to abnormal heart rhythms, called arrhythmias. The heart may beat too slowly (bradycardia), or it may beat to quickly (tachycardia). In some instances, slow heartbeats are normal, such as during sleep or in well-trained athletes. Likewise, fast heartbeats can be normal in some circumstances, such as with exercise, excitement, or high fevers.

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Randy A. Lieberman

In other cases, however, the electrical impulse itself is slowed or blocked and leads to an abnormally slow heartbeat, which may result in heart block. These slow heartbeats may require a pacemaker to correct them. The A-V node is the most common place for the electrical impulse to be blocked.

Tachycardias, or rapid heartbeats, may originate from either the atria or the ventricles and can be treated with various techniques.

Finally, arrhythmias can occur both in people whose hearts are otherwise normal and in those whose hearts have structural abnormalities.

Treatment Approaches: Medication and Electrophysiology

Antiarrhythmic medications are designed to suppress or prevent irregular heartbeats. They

can be used to slow a fast heartbeat but do not necessarily eliminate it. Although medications can be very effective, they may have side effects as well as a problem known as proarrhythmia. This means that antiarrhythmic medications may actually worsen heart rhythms. Patients with the sickest and weakest hearts are most prone to this complication, and a cardiac electrophysiologist (a cardiologist who specializes in heart rhythms) should decide whether the potential benefits of drug treatment outweigh the risks.

Many tachycardias, or fast heartbeats, can be cured by electrophysiology procedures. These are performed by cardiac electrophysiologists, generally in an outpatient setting. A very small area inside the heart, about the size of a pen tip, is cauterized, or burned. This is done to eliminate the focus that may be triggering the abnormal rhythm, or to break a circuit that allows a tachyarrhythmia to start or to maintain itself.

This procedure is done with catheters that are threaded through a blood vessel in the groin or arm, and patients are generally able to go home the same day or early the next day. Increasingly sophisticated computers, catheters, and methods of viewing the heart are already beginning to improve an already impressive cure rate and are increasing efficiency and safety.

Implantable Defibrillators

Implantable cardioverter defibrillators (ICDs) are another tool that focuses on electricity in the heart. These are implanted into a patient's body and automatically detect life-threatening arrhythmias (Fig. 12.4). When one occurs, the device shocks the patient back to a more normal heart rhythm, much like a built-in EMS squad!

These novel implantable devices were invented by Dr. Michel Mirowski in 1969 and first used in patients at Johns Hopkins Hospital in 1980. Cardiac patients owe much to his vision, ingenuity, dogged perseverance, brilliance, and caring. Deeply saddened by the sudden death of his friend and mentor, Mirowski felt there had to be a better and faster way to resuscitate patients who suffered a sudden cardiac death, as can occur, for example, during a heart attack.

Fig. 12.4: The implantable defibrillator device acts like an EMS squad. It senses the heart rhythm, and if the heart develops a harmful rhythm, it delivers a mild shock to the ventricles and returns the heartrate to a normal level.

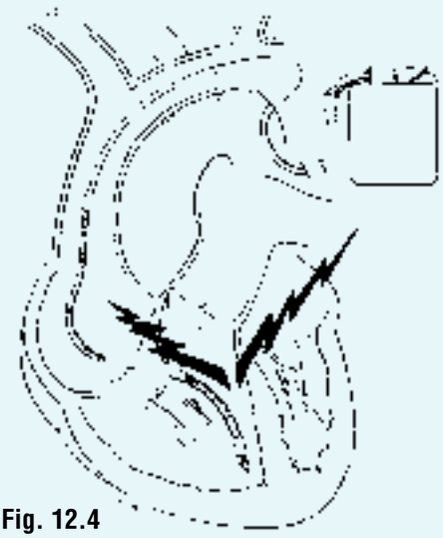


Fig. 12.4

Since the early prototypes, amazing technological advances have been made. Today, ICDs are only slightly larger than pacemakers and can be implanted exactly like them. Programming is performed with a wand that "talks" via telemetric transmission to the pulse generator (computer and battery). The implantation can be completed in about an hour or less, and patients can often go home soon after surgery.

Tachyarrhythmias

Atrial Fibrillation

There are several types of atrial arrhythmias. The most common is called atrial fibrillation, in which the atria beat extremely rapidly (at more than six hundred beats per minute) and somewhat chaotically. This may cause a fast and irregular overall heart beat. Atrial fibrillation occurs more often in the elderly and in patients with hypertension, diabetes, or enlarged atria. It may also be caused by special situations such as an overactive thyroid, serious infection, or open heart surgery.

This condition is not always accompanied by visible symptoms. Only some may feel irregularity in their pulse. Whether there are symptoms or not, the biggest concern is the risk of stroke. For this reason, such patients generally receive anticoagulants, such as coumadin, to minimize stroke risk. Aspirin is the second-best treatment.

At the very least, it is important to control the heart rate in atrial fibrillation. Physicians may opt to try to restore a normal rhythm. As with other forms of

abnormal heartbeat, this can often be done with antiarrhythmic medications.

The physician may try to restore the normal heart rhythm with electricity in a process called electrical cardioversion. Patients are sedated, and a shock is delivered through special pads placed on the patient's chest and/or back. This does not damage the heart and is not painful; it is often very helpful in treating atrial fibrillation.

Much progress has been made in understanding atrial fibrillation. It appears that a certain group of patients may have a trigger spot that sets off their atrial fibrillation. These may be cured with a catheter-based technique that inactivates the abnormal area.

Implantable defibrillators are also used to shock patients out of atrial fibrillation. Patients with such devices may choose when they want the device to terminate their arrhythmias (when they have bothersome symptoms), or they may elect to have the device automatically terminate the arrhythmias. Finally, permanent pacing from two different sites in the atrium at the same time may help prevent or decrease the number of atrial fibrillation recurrences.

Atrial flutter is a different kind of atrial arrhythmia. It consists of a rapid and regular beating of the atria. Although electrical cardioversion can be very effective, atrial flutter can also be terminated by rapid pacing inside the heart. An exciting advance has been ablation, or the destruction of the electrical pathway with a catheter, which provides a cure in more than 90 percent of patients. Even when a total cure is not achieved, the patients may have far fewer recurrences of the flutter, particularly if they are also using anti-arrhythmic medication

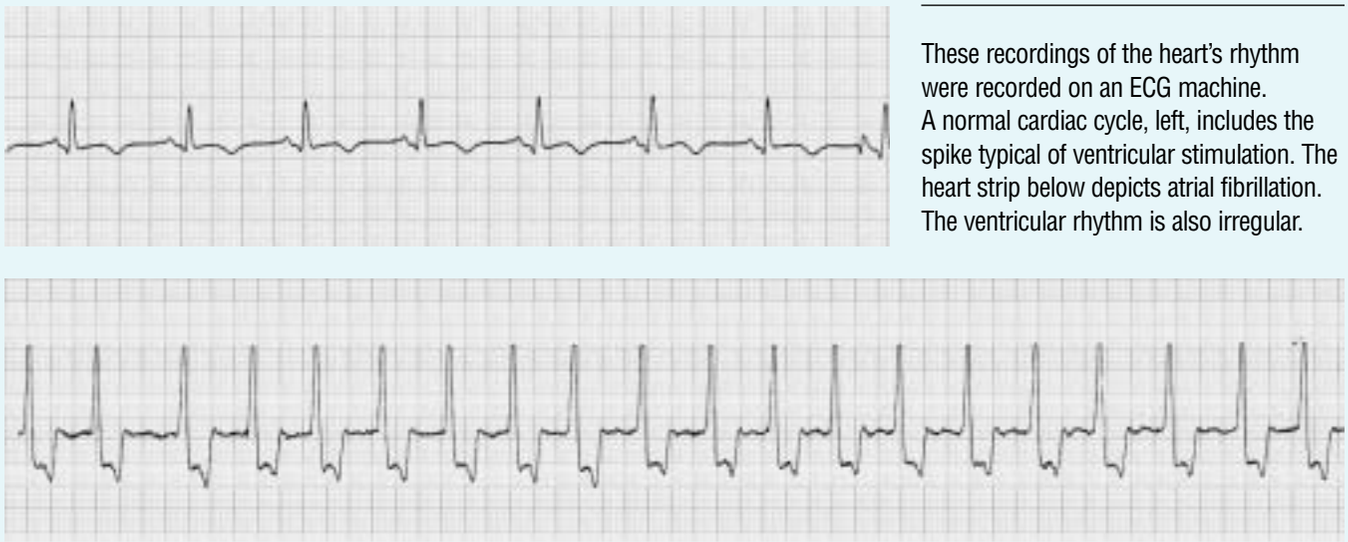
Atrial Tachycardia

Atrial tachycardia is a condition in which a rapid rhythm originates in one or both of the atria. Wolff-Parkinson-White (WPW) syndrome, named after the men who first described it, is a pattern of this condition that can be seen on the ECG. Not every person born with WPW has an arrhythmia problem. However, the potential exists for an abnormal rhythm to occur. Ablation has an extremely high chance of curing the condition and is the treatment of choice in most of these patients.

Finally, patients may have isolated "skipped" heartbeats (one or two at a time) relating to electrical impulses coming from different areas of the heart without a sustained arrhythmia. Frequently, no therapy is required. On other occasions, the patient may wish to discuss with his or her physician what the approach should be. These are rarely, if ever, life threatening or problematic.

Ventricular Arrhythmias

Ventricular arrhythmias, which involve the main pumping chambers of the heart, tend to be more serious than atrial arrhythmias. The three main ventricular arrhythmias include premature ventricular complexes (PVCs),



These recordings of the heart's rhythm were recorded on an ECG machine. A normal cardiac cycle, left, includes the spike typical of ventricular stimulation. The heart strip below depicts atrial fibrillation. The ventricular rhythm is also irregular.

ventricular tachycardia, and ventricular fibrillation.

Premature ventricular complexes refer to beats originating from the ventricles. They may or may not indicate a serious cardiac problem, and each patient should have his or her situation individually assessed. Some people are completely unaware of their PVCs; others are very symptomatic. The approach may vary from no treatment to anti-arrhythmia drugs to ablation when appropriate.

Ventricular tachycardia is an abnormal rhythm originating in the ventricles, which often beat faster than one hundred beats per minute. It can occur in structurally normal or abnormal hearts. The rate, duration (number of beats in a row), ECG appearance, and symptoms can also vary, both within a given person and among people. As with atrial arrhythmia, some people appear to be born with the predisposition for ventricular tachyarrhythmias. Others develop this predisposition.

Management of ventricular tachyarrhythmia can be very complex, and many clinical factors need to be considered. In some cases, anti-arrhythmia medication provides good control. In other cases, ablation is the best approach. Currently, implantable cardioverter defibrillators can provide life-saving treatment for patients with potentially or actually life-threatening ventricular tachyarrhythmia.

Ventricular fibrillation is an extremely rapid, often chaotic rhythm of the ventricles. It is a very serious and often fatal condition because the heart cannot effectively pump blood to the body. Unless rapidly corrected by an electrical shock (such as from an EMS team or an external or internal defibrillator), death will follow. For most patients who have been successfully resuscitated from ventricular fibrillation, the treatment of choice is implantation of an ICD.

General Observations

When dealing with the vast and complicated field of arrhythmia, each person's situation should be evaluated on a case by case basis. However, some general observations do apply.

Symptoms: Some people with arrhythmias do not feel them; others do. The same person may notice some arrhythmias and not others; symptoms may vary and can include palpitations (feeling certain heart beats), skipped beats or fluttering sensations, fatigue, dizziness, lightheadedness, or even loss of consciousness. The factors accounting for this variability are complex and not always understood. The presence or absence of other cardiac medical problems may play a role in some cases. An abnormal ECG or irregular or unusually fast pulse may be the first clue of an arrhythmia. Some people have symptoms but when monitored have no arrhythmias.

Triggers: Many people do not notice a pattern or trigger to their arrhythmias. For some people, drinking excess caffeine or alcohol may be a trigger, in addition to eating certain foods.

Cause: Some people are born with very subtle electrical abnormalities in their hearts, which may not be noticed by using methods such as the ECG, echocardiography, or other tests. Many such people go through life without ever having an arrhythmia. Still others develop a problem later in life. It is not well understood why this occurs.

An electrical problem can develop as a result of an abnormality or damage (such as after a heart attack, after heart valves are damaged from rheumatic fever, or after heart muscle is damaged from another cause — high blood pressure, a viral infection, etc.).

Natural history: In some people, the frequency of an arrhythmia may change spontaneously with age; more often than not, there is no clear pattern or trend in the frequency of arrhythmia recurrences. In some cases, an arrhythmia such as atrial fibrillation will recur and persist. Many factors, including certain medications, may affect what would have been the natural history of the arrhythmia.

The Future

Advances in understanding of arrhythmias make this an exciting time in arrhythmia management. Many lives have already been saved and improved. For example, studies are presently evaluating the use of specialized pacing techniques (e.g., pacing of both ventricles at the same time) to help patients with congestive heart failure. Heading into the next millennium, we anticipate even more exciting developments.

SURGERY FOR THE IRREGULAR HEARTBEAT

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IRREGULARITIES OF THE heartbeat (cardiac arrhythmias) are the most common maladies affecting the heart. Because the heart does not pump blood as efficiently when it has an irregular heartbeat, patients with cardiac arrhythmias usually complain of tiredness and shortness of breath, especially with physical exertion. More serious cardiac arrhythmias may result in heart failure, strokes, and death. Fortunately, many cardiac arrhythmias can be successfully treated with medicines or catheters. However, when arrhythmias are not responsive to drug therapy and cannot be treated with catheters, heart surgery may be required if the symptoms are particularly severe or life threatening.

One of the unique problems facing surgeons who operate on the heart for cardiac arrhythmias is that the arrhythmia cannot be seen. In the past, it was necessary to place tiny electrodes on the heart to record abnormalities in the heart's electrical

activity. Once the abnormalities were identified and located, the physician could apply the specific surgical procedure required to cure them.

The first such operation was performed for a simple arrhythmia in 1968. Although the surgical treatment of cardiac arrhythmias flourished in the 1970s and 1980s, catheter techniques were developed in 1990 that could cure virtually all arrhythmias that were not responsive to drug therapy. The one exception, unfortunately, is the most common of all cardiac arrhythmias, atrial fibrillation.

Atrial fibrillation is a type of cardiac arrhythmia in which the electrical activity in both atria becomes chaotic, causing them to quiver (or fibrillate) rather than beat in a regular fashion. More importantly, it also results in severely irregular beating in the lower two pumping chambers, the left ventricle and the right ventricle. Because the left ventricle is the main pumping chamber of the heart, symptoms invariably develop when it pumps less efficiently than normal.

Many times, atrial fibrillation can be successfully treated with drugs and/or an electrical shock to the heart called cardioversion. Unfortunately, the drugs and cardioversion don't always work. This is particularly unfortunate because blood clots can form inside the left atrium as a result of atrial fibrillation and subsequently break off and pass through the bloodstream to the brain, where they cause a stroke. Recent studies have shown that

about 2.2 million people in the United States suffer from atrial fibrillation and that seventy-five thousand strokes occur each year from this common cardiac arrhythmia.

After years of laboratory research, my research team, first working at Duke University, then at Washington University in St. Louis, developed a heart operation to cure atrial fibrillation. The procedure is referred to as the "maze procedure" and involves making several incisions in the right and left atria. The heart-lung machine is used during the surgery.

In 1987, we first used the maze procedure to treat a patient. During the past eleven years, this technique has proven to be essentially 100 percent successful. In addition, the maze procedure has been shown to eliminate the risk of stroke from atrial fibrillation, primarily because patients no longer have atrial fibrillation after the surgery.

Over the last two years, we have also developed a new minimally invasive surgical approach for the maze procedure that has resulted in much less pain and debilitation for patients. The recuperation time has been markedly reduced as well.

The maze procedure has now been applied in virtually all of the major national and international medical centers. Its use is particularly common in Japan, where several thousand patients have undergone the maze procedure for treatment of atrial fibrillation. When performed as originally described, the excellent results are reproducible by most cardiac surgeons. In our own institution, we are now performing nearly one hundred maze procedures per year. The average length of the operation is three hours, and the usual hospital stay is about one week. Most patients return to full-time activity, short of heavy physical exertion, about one month after surgery.

Although there is no catheter-based approach available that uses the maze procedure, numerous cardiologists around the world are working daily on this challenge. They are developing a technique for performing the maze procedure or some related procedure without surgery, even minimally invasive surgery. Thus far, these approaches have been highly experimental and largely unsuccessful or dangerous. Nevertheless, it is almost certain that in the near future some type of nonsurgical approach will be perfected that will be capable of curing this last, most common, and potentially dangerous cardiac arrhythmia.