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When Heartbeats Go Haywire:

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by Margie Patlak

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Skating champion Sergei Grinkov, who died during skating practice in 1995, is shown in an earlier photo with his wife and skating partner, Ekaterina Gordeeva, and their daughter, Daria. He died within minutes of suffering from the most severe form of ventricular arrhythmia.

Two-time Olympic gold-medalist skater Sergei Grinkov shocked the world in fall 1995, when he collapsed on the ice rink and died hours later at the young age of 28. A blood clot that blocked blood flow to his heart triggered it to quiver instead of beat. Medical technicians were unable to restore a normal heart rhythm, and by the time he reached a hospital, Grinkov's heart had stopped beating altogether.

Grinkov suffered what is known as ventricular fibrillation. This inefficient and deadly quivering of the heart is one of several types of irregular heartbeats, or arrhythmias, that afflict the lower chambers, called the ventricles, of the heart. Not all arrhythmias are ventricular; some, for example, arise from the upper chambers of the heart.

Ventricular arrhythmias often occur in people with various forms of heart disease and, according to the American Heart Association, cause most cases of sudden cardiac death. But new drugs and devices show promise in curbing the number of deaths from this condition. These new treatments, approved by the Food and Drug Administration, include a longer-acting form of an anti-arrhythmia drug, a wide availability of portable and implantable electrical devices that can spark a return to normal heart rhythm, and techniques for destroying heart tissue that triggers ventricular arrhythmias.

Heart of the Matter

Not much bigger than a fist, the human heart beats 100,000 times each day, sending about 2,000 gallons of blood coursing through vessels, which, laid end-to-end, would be long enough to circle the earth more than twice.

To carry out the vital task of pumping blood, the electrical timing of millions of heart cells must be exquisitely coordinated. Their timing sparks the heart to pump in a rhythmic, efficient fashion. When that coordination is disrupted, life-threatening ventricular arrhythmias result.

Each heartbeat normally starts in the upper right chamber of the heart, or right atrium. Here, a specialized bunch of cells called the sinus node, or pacemaker, sends an electrical signal. The signal spreads throughout the right and left atria and then travels along specific pathways to the lower chambers or ventricles. As the signal travels, the heart muscle contracts. First the atria (the upper right and left chambers) contract, pumping blood into the ventricles. A fraction of a second later, the ventricles contract in a squeezing motion, sending blood throughout the body. Each contraction is a heartbeat.

Ventricular arrhythmias occur when a group of heart cells in the ventricles triggers contractions out of sync with the normal rhythm established by the sinus node. A number of factors can prompt a ventricular arrhythmia, including stress, exercise, caffeine, tobacco, alcohol, amphetamines, tricyclic antidepressant drugs, and cough and cold medicines containing pseudoephedrine, as well as several drugs (such as diuretics and digitalis) used to treat various heart conditions.

Many types of heart disease also are associated with ventricular arrhythmias. Atherosclerosis, the buildup of plaque on artery walls, can reduce blood flow to heart tissue. That, in turn, can impede the transmission of electrical signals governing heart contractions. This can prompt groups of ventricle cells to generate their own "back-up" rhythm. In the

extreme case of a heart attack, blood flow to specific parts of the heart muscle is completely blocked, and that heart tissue dies. If the affected area includes cells in the electrical pathways of the heart, arrhythmias ensue.

People with enlarged hearts or faulty heart valves also are prone to experiencing ventricular arrhythmias. Ventricular arrhythmias also commonly occur after heart attacks, heart infections, or heart surgery, or when the body is under severe physical stress from, for example, lack of oxygen, very low blood pressure, or major blood loss. They also are triggered by heart failure, surgery, and other conditions that cause abnormal blood and tissue concentrations of potassium, magnesium, sodium, or calcium. These minerals play key roles in triggering and conducting electrical impulses in the heart.

Harmless or Deadly Beats

Ventricular arrhythmias can be either deadly or innocuous, depending on their type and persistence and whether the person's heart function is already compromised. The most common type of ventricular arrhythmia in both healthy and diseased individuals is the ventricular premature beat. The incidence of this condition increases with age.

A premature beat occurs when there is an extra contraction of the ventricles midway between two normal contractions or shortly after a normal contraction. In the latter case, they can delay the next heartbeat prompted by the natural pacemaker.

Ventricular premature beats often do not prompt symptoms, but they may be perceived as skipped beats or fluttering or thumping in the chest known as heart palpitations, and they may cause dizziness or weakness. Probably everyone develops ventricular premature beats at one time or another, according to the American Heart Association. This type of arrhythmia is commonly encountered in cardiac monitoring, even in healthy individuals.

Ventricular premature beats are not by themselves harmful, but they can be a precursor to two more serious types of ventricular arrhythmias: ventricular tachycardia and ventricular fibrillation.

Ventricular tachycardia is rapid heartbeat that arises from the lower chambers of the heart and is usually much faster than the normal heart rate of 60 to 100 times per minute. Ventricular tachycardia is considered "nonsustained" if it lasts only seconds or "sustained" if it lasts for more than 30 seconds. Like ventricular premature beats, ventricular tachycardias commonly occur in healthy people, particularly those who are frightened or excited.

Ventricular tachycardias prevent ventricles from properly filling with blood. This reduces pumping efficiency, which can be made worse if there are underlying heart muscle abnormalities.

Nonsustained ventricular tachycardias may cause no noticeable symptoms, or they may be felt as palpitations. When sustained, however, tachycardias often cause palpitations, as well as weakness, dizziness, chest pain, and breathing difficulties. Particularly rapid or long-lasting ventricular tachycardias or sustained tachycardias in people whose heart function is already compromised by disease can cause loss of consciousness or lead to fatal cardiac arrest.

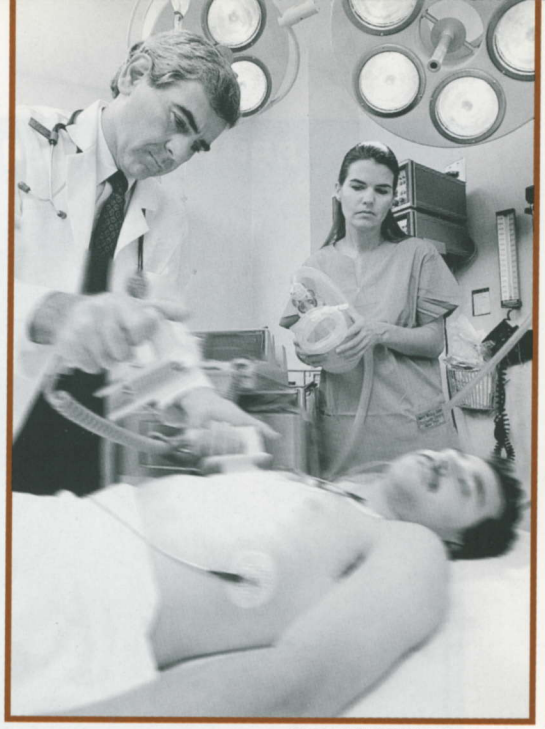
A ventricular tachycardia can degenerate into ventricular fibrillation, which is an extremely rapid, chaotic rhythm that starts in the ventricles and causes the heart to quiver. Such quivering prevents the heart from pumping blood to the rest of the body. The onset of a ventricular fibrillation is dramatic: People suddenly lose consciousness and collapse in a shock-like state. Their pulse, heartbeat and blood pressure cannot be detected, and death occurs in minutes without effective treatment. A common cause of ventricular fibrillation is a heart attack.

Emergency Care

Patients with ventricular fibrillation must be treated immediately with one or more electric shocks to the heart, which are transmitted externally with defibrillator paddles placed on the chest. Severe ventricular tachycardias also must often be treated with defibrillators.

Defibrillators tend to synchronize the heart's electrical system. "By giving a shock you start things from scratch again and organize a disorganized rhythm," said Andrew Epstein, M.D., of the University of Alabama at Birmingham.

Defibrillators may become more readily available as a result of FDA's approval in September 1996 of a smaller portable version that may be particularly beneficial to police officers, firefighters, flight attendants, and others who may be the first to respond to cardiac emergencies and can now equip their vehicles with the compact units.



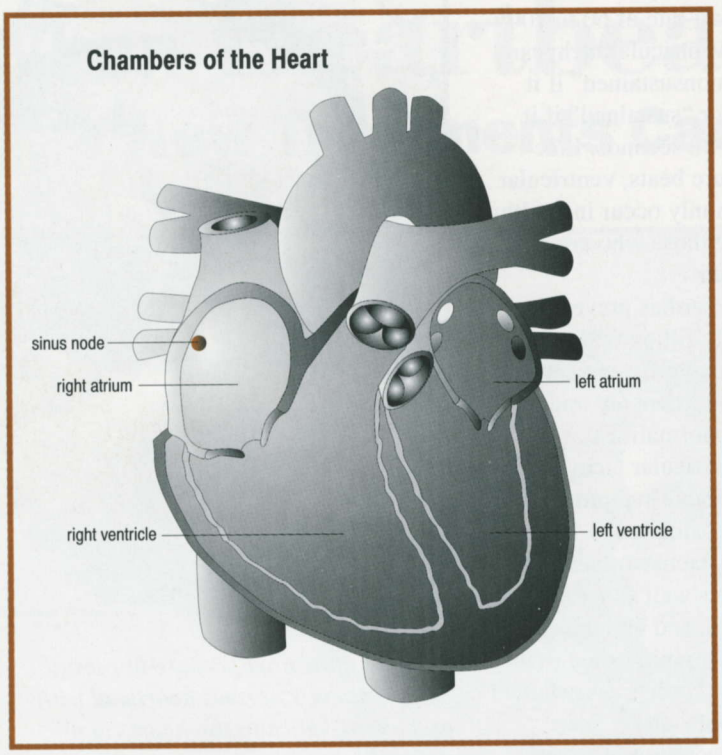
Medical personnel prepare to reestablish a patient's normal heartbeat with an electric shock to the heart via a defibrillator.

Once the heartbeat has been restored, patients usually are given lidocaine hydrochloride (Xylocaine) or bretylium tosylate (Bretylol) intravenously to stabilize their heart rhythm.

The Telltale Heart

The safety and effectiveness of long-term arrhythmia treatment depend on accurate diagnosis. Sometimes ventricular arrhythmias can be detected by listening to the heart with a stethoscope. The diagnostic tool of choice, though, is the electrocardiogram (ECG or EKG), which shows the relative timing of atrial and ventricular electrical events. ECG's generate telltale spikes, whose characteristic rhythm and shape identify the specific type of arrhythmia.

To make an ECG, a technician attaches several electrodes to the chest and sometimes the limbs. The electrodes detect electrical activity that is recorded on a moving strip of paper or projected on a computer-like screen. The procedure is



harmless and painless.

Because of the fleeting nature of many arrhythmias, they may not occur while the ECG is running, and so they may go undetected on the ECG. In these cases, doctors may ask patients to wear a small portable ECG recorder, called a Holter monitor, for 24 hours. This device, about the size of a tape recorder, records continuous electrocardiographic signals or selectively records arrhythmias causing symptoms. Holter monitoring requires patients to wear electrodes continuously on their chests during the 24 hours.

ECG's of arrhythmias with symptoms that occur less frequently than daily can be transmitted via the telephone to a doctor's office or a hospital with a handheld device called an event monitor. This monitor converts ECG signals into tones that travel over a telephone line and are then converted to paper tracings. If a telephone is not available at the time the arrhythmia occurs, the ECG signals can be recorded and stored in the device's memory for transmission later. For such transtelephonic monitoring, patients place electrodes on their chests only when they are experiencing symptoms.

Another diagnostic option for infrequent arrhythmias is to provoke them purposely through exercise or with electrical devices. For example, a patient whose arrhythmias are thought to be prompted by exercise may undergo a treadmill workout while his or her heart activity is being monitored by an ECG device.

An arrhythmia also may be induced with electrophysiologic testing. In this procedure, electrodes are attached to small tubes known as electrode catheters, which are threaded through arm or leg veins until they reach the heart. There, they are placed at strategic positions in the ventricles, atria or both.

These electrodes record electrical signals and allow doctors to "map" the spread of electrical impulses during each heartbeat. The electrodes also can electrically stimulate the heart at programmed rates to trigger latent ventricular tachycardias. These arrhythmias are then stopped by electrical stimuli transmitted via the electrode catheters. An externally applied shock may be required if the patient loses consciousness during the tachycardia.

Being able to "turn on" tachycardias during electrophysiologic testing allows doctors to test antiarrhythmic drugs quickly for effectiveness. It also can indicate the electrically blocked areas of the heart responsible for triggering a patient's arrhythmia. If these areas are limited in size and number, destruction of them is a treatment option.

Cardiologists usually reserve electrophysiologic testing for patients whose arrhythmias do not occur during ECG monitoring or are not controlled by their current medication. Electrophysiologic testing is considered safe, although rare complications, such as bleeding, infection, perforation of the heart, and fatal arrhythmias, can occur.

Preventive Treatment

Before starting any preventive drug treatment regimen, doctors first try to rule out reversible causes of ventricular arrhythmias: for example, caffeine, alcohol and tobacco consumption, and certain over-the-counter and prescribed medicines.

Also, because treatments pose substantial risks relative to the risk of the arrhythmias themselves, doctors tend not to treat ventricular arrhythmias unless they are tied to significant symptoms or are life-threatening. For this reason, FDA has not approved any treatments for premature ventricular beats.

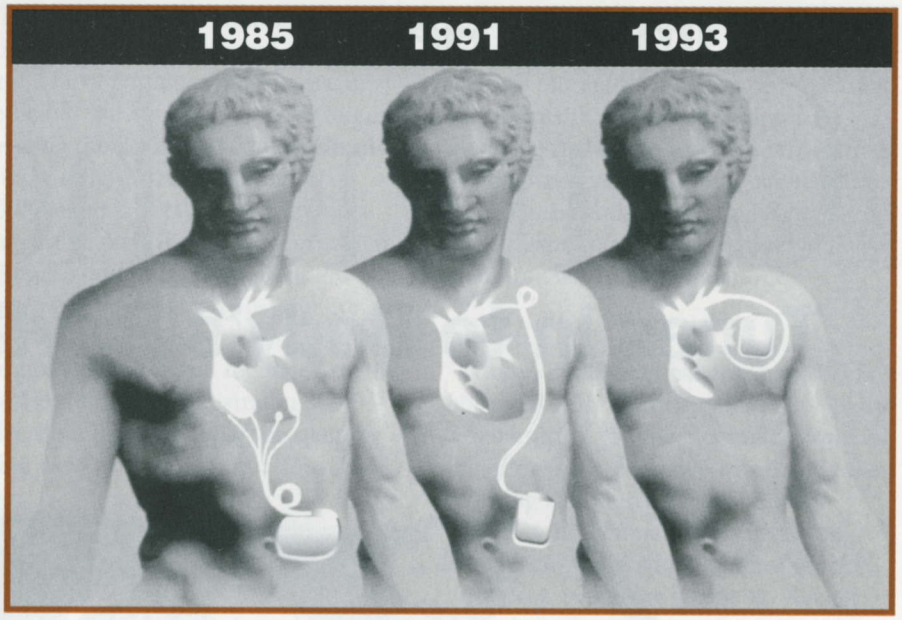
However, there are several drugs approved for preventing ventricular tachycardia. The main types are beta blockers and sodium or potassium channel blockers. Most drugs to prevent ventricular tachycardias are taken orally up to four times daily and often must be taken for life.

Beta blockers, such as propranolol hydrochloride (Inderal and others), stem the automatic stimulation of heart contractions by the nervous system. Sodium and potassium channel blockers hamper transmission of electrical impulses in heart cells. Some sodium channel blockers are quinidine (Quinidex Extentabs, Quinaglute and others) and procainamide hydrochloride (Procan, Pronestyl and others). FDA approved in February 1996 a long-acting form of procainamide, Procanbid, which is taken only twice a day, compared with other procainamides, which must be taken four times daily.

Potassium channel blockers, such as amiodarone hydrochloride (Cordarone) and sotalol (Betapace), also are used to prevent ventricular tachycardias.

Doctors monitor the effectiveness of antiarrhythmia drug therapy with an ECG, or with electrophysiologic testing. Monitoring is essential not only to ensure effectiveness but safety, as well, because many of these drugs can make arrhythmias worse. Other side effects of antiarrhythmia drugs that can limit their use are low blood pressure, lung damage, nausea, and dizziness.

According to Wilber Aronow, M.D., a cardiologist with Mount Sinai School of Medicine in New York City, studies show that people treated with certain



Implantable defibrillators keep getting smaller. The top left photo shows, counterclockwise from top, the devices' downsizing, so that today, some are only about the size of a 2 1/2-inch square block. Their smaller size now makes it possible for doctors to implant them in the chest rather than the abdomen. (Photos courtesy of Medtronic Inc.)

beta blockers following a heart attack have a significantly reduced risk of sudden cardiac death. But many large-scale studies of several different types of sodium channel blockers, as well as studies of certain potassium channel blockers, have shown that treatment with these drugs following heart attacks does not improve survival odds, or reduce them.

Ventricular arrhythmias are common within a month of a heart attack and are associated with an increased risk of sudden cardiac death.

An Internal Jolt

Another treatment option for people at risk for life-threatening arrhythmias is an implanted cardioverter defibrillator. FDA approved the first implantable defibrillators more than 10 years ago.

Today's device typically consists of a generator slightly smaller than the size of a wallet attached to electrode catheters. The generator is surgically placed under or over chest or abdominal muscles. The catheters are threaded through veins to their permanent positions in the heart. Complications of implanting defibrillators are rare but serious and include bleeding, infections, and perforation of the heart.

Implanted defibrillators monitor the heart rhythm and automatically treat, with electrical stimuli or shocks, rhythms recognized as abnormal. Newer devices also can record and store data of the electrical activity of the heart that doctors can later download and evaluate for arrhythmias. The data also can be used to perform electrophysiologic testing.

Implanted defibrillators can often stem ventricular arrhythmias with low-energy shocks. Sometimes, however, high-energy shocks are needed. These shocks, though short-lasting, can be painful—somewhat akin to a kick in the chest.

The generators in implanted defibrillators usually last three to five years and can be replaced with a surgical procedure that usually requires only local anesthesia. The electrode leads tend to last longer, although they can develop cracks or component failures that require their replacement.

A recent study of heart attack survivors by Arthur Moss and colleagues from the University of Rochester (N.Y.) Medical Center found implantable defibrillators cut survivors' risk of death in half.

A National Heart, Lung, and Blood Institute study under way is assessing whether implanted defibrillators or drug therapy is more effective in extending the lives of patients with ventricular arrhythmias.

Opening the Chest

Open-heart surgery to remove heart tissue causing or contributing to arrhythmias may be warranted for patients whose ventricular arrhythmias cannot be controlled by drugs. But, this is feasible only for patients whose arrhythmias can be attributed to heart sites that are limited in size and number. Most patients who undergo this procedure survive.

To avoid the risks and painful recovery of this procedure, a number of clinical investigators have used radiofrequency energy, delivered via catheters threaded through veins to the heart, to destroy heart tissue at the root of ventricular arrhythmias. FDA has not yet fully evaluated the safety and effectiveness of this experimental procedure.

But the availability of other treatment options means that many patients with ventricular arrhythmias can be treated effectively.

That wasn't true a decade ago, cardiologist Epstein points out. "We're a lot further along." ■

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