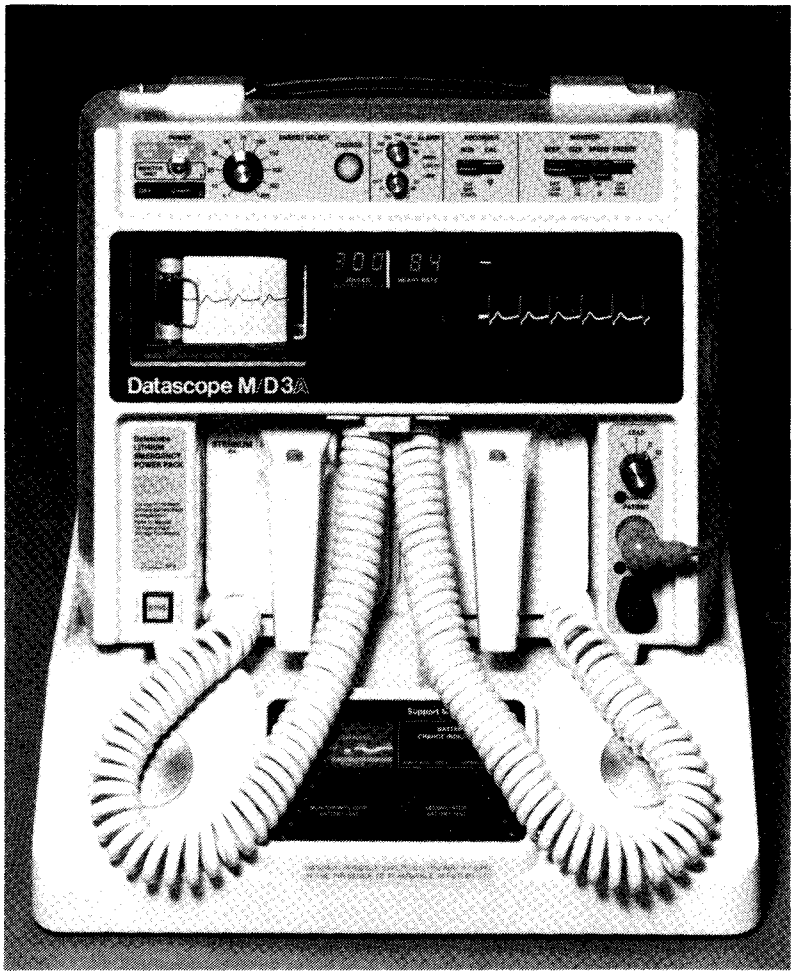
Medical Uses of Electric Shock



Electrical stimulation of the body can be dangerous, but there are some instances where it can save a life.

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ACCIDENTAL CONTACT WITH HIGH VOLTAGES or currents can often result in injury or even death. However, there are many instances when deliberate exposure to some type of electrical stimulation is an effective part of treatment for an illness or condition. In this article we are going to look at some of those instances.

Electrical stimulation of the heart

In a healthy heart, muscle contractions are initiated and coordinated by a natural "pacemaker" and "conduction" system that is made up of special muscle cells. While many cells in the heart will electrically discharge, causing a contraction to occur, the pacemaker area of the heart contains cells that spontaneously discharge the fastest. After the pacemaker cells discharge, electrical signals travel throughout the atria (the top half of the heart). The atria are electrically insulated from the ventricles (the bottom half of the heart). The signal from the atria is conducted to the ventricles by a specialized collection of muscle fibers (the AV node and associated fibers) that cause a delay of about a tenth of a second. Thus, after the atria contract to push blood into the ventricles, there is a delay of about a tenth of a second before the ventricles receive the signal to contract. It is the ventricles that produce the real pumping action, giving a pulse that can be felt at the wrist and elsewhere.

When that normal pattern is disrupted, the heart is said to be in fibrillation. When fibrillation is brought on, due to extra pacemaker signals being generated or to an irritation of the heart, the beating of the heart muscle becomes uncoordinated. During fibrillation the exposed heart can be seen to writhe like a bag of worms, rather than contracting in a useful manner. If the ventricles beat normally but there is atrial fibrillation, about 20% of the efficiency of the heart is lost because blood does not enter the ventricles as well as it should. In addition, the fibrillation signals passing through the AV node usually stimulate the ventricles to contract at a rate of about 170 beats per minute, a condition not well tolerated by most individuals. While many people have atrial fibrillation which cannot be stopped, the rate of the beating of the ventricles can be controlled by medication that slows conduction of impulses through the AV node and decreases the ventricular contraction rate (the effective heart rate).

Ventricular fibrillation, if not treated within minutes, is fatal. About 350,000 Americans die each year from disruptions in cardiac rhythm, usually ventricular fibrillation. Most of those electrical disturbances take place during or within a few days of a myocardial infarction (heart attack). Many of the people who die do so needlessly. Though the heart muscle has been damaged by a heart attack, if the electrical disturbance is corrected, the person might live for many more years.

One way to stop fibrillation of the atria and ventricles is to apply an electrical shock to the chest. That shock is administered via large paddles (as you've doubtless seen on TV or at the movies) using a machine called a defibrillator. When that electrical shock is applied, all muscle cells in the heart are depolarized, as if they had just discharged. The first cells to recover are those in the pacemaker area of the heart; they send signals to the conduction system, which causes organized contraction of the heart muscles.

Figure 1 shows an electrocardiagram of a patient in ventricular fibrillation. The region labeled A shows irregular, nonrepeating voltage fluctuations. A defibrillating shock of about 1,000 volts is applied at B. That causes the electrocardiogram amplifier to overload. For a period the electrocardiogram shows a signal with an elevated baseline, then the amplifier saturates again (C). After a few seconds the heart begins to beat slowly (D), though not in a normal manner.

A relatively small number of people have recurrent episodes of cardiac rhythm disturbances (arrhythmias) that are not as-

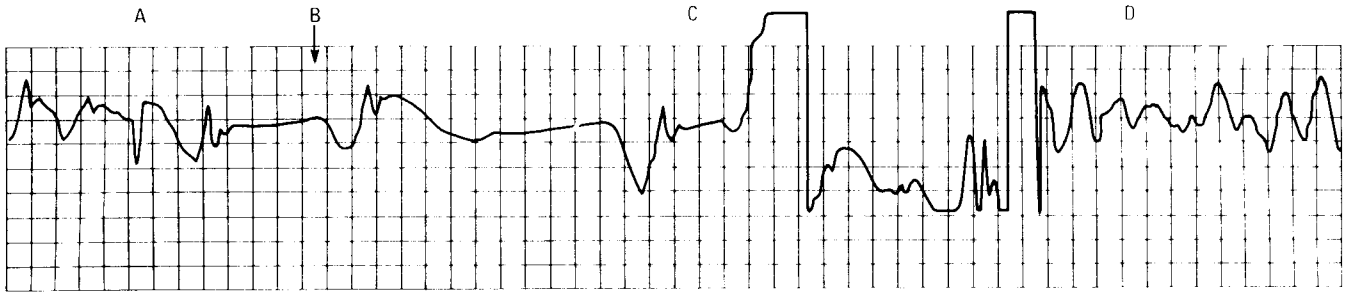

FIG. 1—ELECTROCARDIOGRAM of a patient with ventricular fibrillation. At point *b* defibrillation (the applying of a voltage) is performed. A heartbeat, although abnormal, slowly begins to appear after several seconds (*d*).

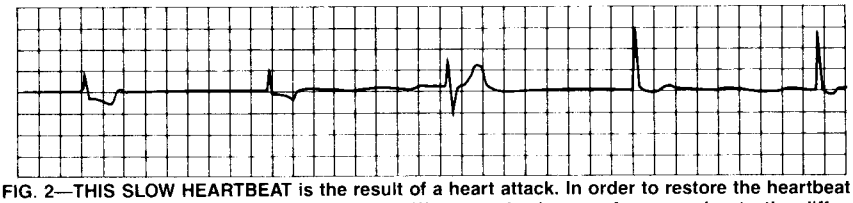

FIG. 2—THIS SLOW HEARTBEAT is the result of a heart attack. In order to restore the heartbeat to normal, a pacemaker had to be implanted. Differences in the waveform are due to the different locations of electrodes used to take the reading.

sociated with heart attacks. Many of those disturbances are corrected by electrical shock, or defibrillation. A few dozen people with recurrent potentially-lethal arrhythmias have been treated with surgically implanted defibrillators. When a fatal rhythm is recognized by such a unit, a shock of 30 joules is applied to the heart. That is less than the 300 joules usually applied when the defibrillation shock is administered externally. But then again, the power requirements are less because the shock is applied directly to the heart rather than to the chest wall.

In fibrillation there are too many signals or depolarizations occurring. With a slow or absent heart rate, there are too few depolarizations. Figure 2 shows an electrocardiogram from a patient who has suffered an acute heart attack. The beats are coming much too slowly, about one every 2 seconds. That gives a pulse rate of about 30 beats per minute. It was decided that a pacemaker was needed in that case because drugs would not increase the heart rate, and the patient was in shock. A temporary pacemaker, actually a specially built pulse generator in a small box, was connected to the heart by a catheter with wires built into it. The pacemaker catheter was inserted into a large vein under the clavicle (collar bone) and pushed forward until it reached the heart. (See Figs. 3 and 4.)

Usually when a pacemaker catheter is inserted, the positioning of the catheter is obtained using "fluoroscopy." That technique uses a continuous X-ray source and an image intensifier system that permits the physician to see a real-time image of the catheter working its way to the heart. Because veins branch and turn it is useful to be able to see where the catheter is going and to twist or redirect it when needed.

Because the condition of the patient whose electrocardiogram is shown in Fig. 2 was so acutely poor, there was not time to set up the equipment or wait for the technicians who run it to come to the hospital. Thus the catheter was inserted "blindly." That is, the catheter was inserted in the vein via a large needle and pushed forward until small spikes, caused by the signal from the pacemaker, were seen in the patient's electrocardiogram. The presence of those spikes meant that the catheter was near the chest electrodes used to monitor the electrocardiogram. The catheter was further advanced until the small spikes were followed by heartbeats, as seen in Fig 5. That was electrical confirmation of the fact that the pacemaker had "captured" (was stimulating) the heart. Later, routine X-rays showed that the pacemaker was in a good location. Because of the type of heart attack, that pacemaker was only needed for a few days. In other cases a permanent pacemaker must be surgically implanted in the patient and connected to the heart.

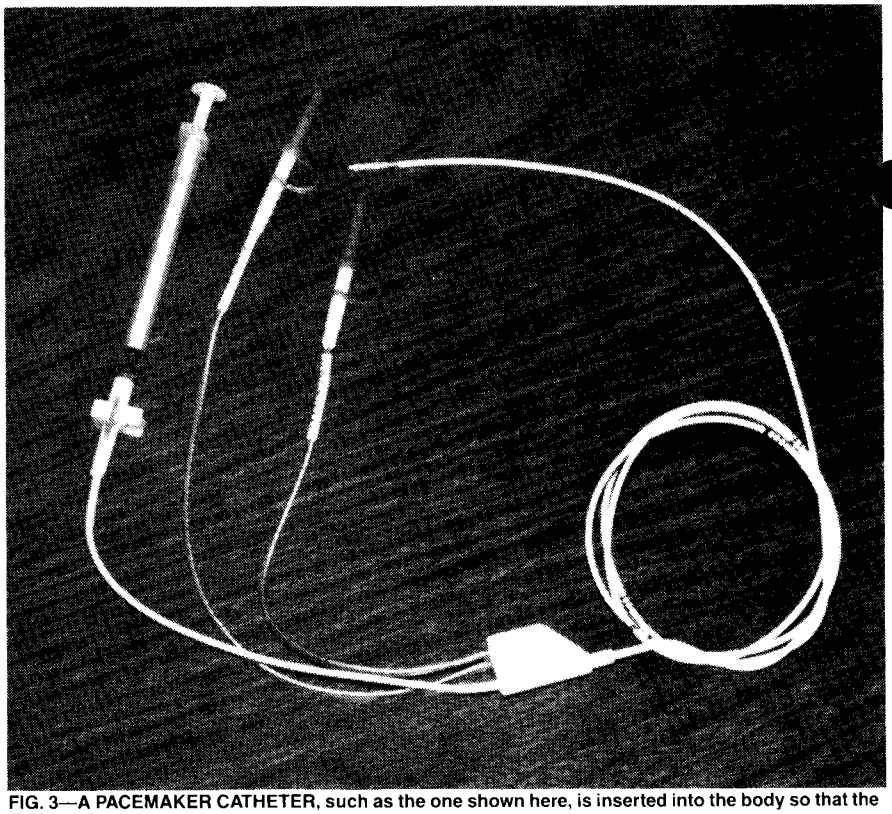

FIG. 3—A PACEMAKER CATHETER, such as the one shown here, is inserted into the body so that the electrodes at its end contact the heart muscle.

There are several types of electronic pacemakers. An atrial pacemaker can be used if the atria are not contracting often enough, but the natural conduction between the atria and ventricals is normal. A ventricular pacemaker can be used to stimulate just the ventricles. The atria

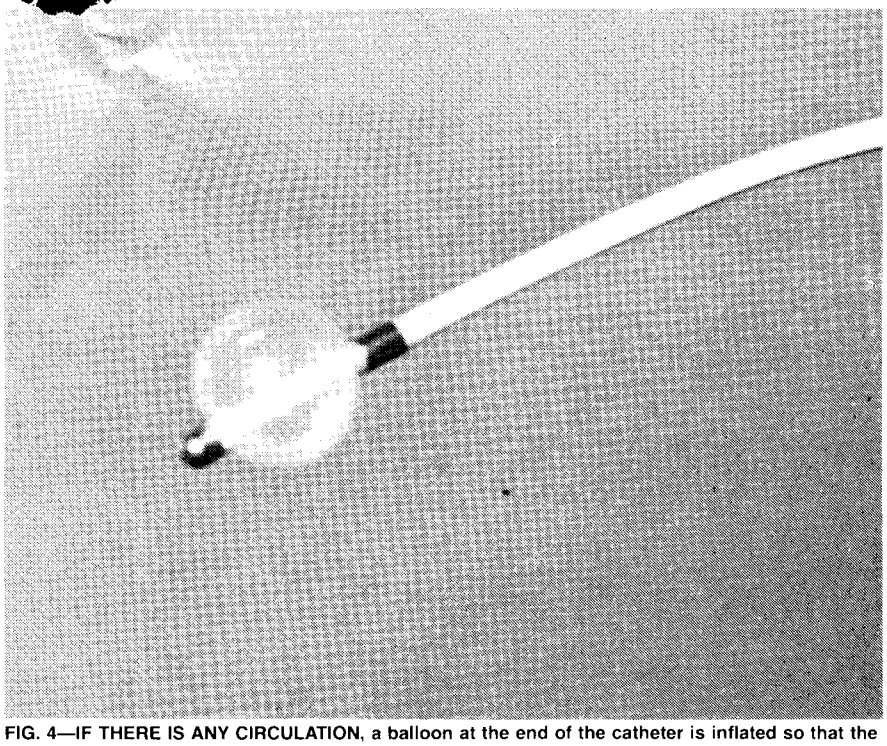

FIG. 4—IF THERE IS ANY CIRCULATION, a balloon at the end of the catheter is inflated so that the catheter tip will be carried to the heart by blood flow.

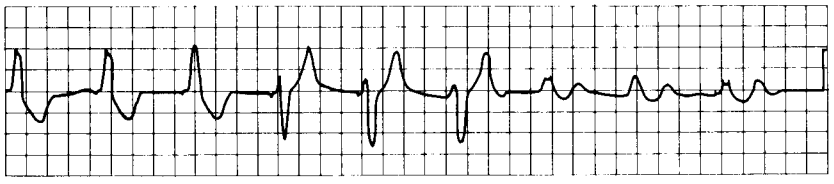

FIG. 5—SMALL SPIKES, which are the output signals from the pacemaker, are followed by heartbeats. This shows that the pacemaker has "captured" the heart.

may be stimulated by backward conduction through the AV node, but the resulting atrial contraction would be at the wrong time to do any good. A sequential pacemaker has two sets of wires: One stimulates or senses the signal from the atria; the second stimulates the ventricles after a delay that simulates the delay normally caused by the AV node. With a sequential pacemaker, the 20% boost in cardiac output provided by the atria is retained.

Different pacemakers operate in different manners. Some work only when needed; those are called *demand pacemakers*. The same wires that stimulate the heart are used to detect heart signals that are spontaneously present. Thus, if the heart is beating on its own, the pacemaker does not fire. If the heart rate gets below the minimum a patient could tolerate, the pacemaker fires.

Some pacemakers fire regardless of spontaneous heart activity. Those are called *asynchronous pacemakers*.

Instead of turning off when a heartbeat is detected, some pacemakers fire within milliseconds of each detected heartbeat (with a maximum rate of firing). In that way externally induced electrical signals cannot trick the pacemaker into shutting off. Units that operate in that manner are called *stimulated pacemakers*.

External pacemakers have controls that allow the physician to program the stimulus current and heart rate. It is also possible to choose whether or not the pacemaker should be inhibited by spontaneous heart activity. With surgically implanted pacemakers that is not possible. To overcome that problem, remotely programmable pacemakers have been developed. The pacemaker contains a radio receiver that detects coded signals sent by a hand-held transmitter that looks something like a calculator or small computer. With such a unit, it is possible to control a wide variety of pacemaker parameters.

If a permanently implanted pacemaker is not functioning, troubleshooting can be difficult. However, there are some tests that can be done to help determine the cause of the problem.

Almost all demand pacemakers contain a magnetic reed switch. Placing a strong permanent magnet near the pacemaker will activate the switch and convert the pacemaker to an asynchronous mode of operation. As long as the magnet is held in place, the pacemaker will fire at a constant rate regardless of the person's spontaneous heart rate. If a magnet is applied and no pacemaker spikes can be seen on the electrocardiogram, either the pulse generator or the electrode wire is defective.

If a pacemaker fails within weeks of being implanted, the problem is usually with the electrode wire. The wire may have dislodged, or the electrical resistance between the electrode and the heart tissue may have increased.

A failure after years of operation is most often due to weak batteries; placing a magnet over the pacemaker may give a relatively slow heartrate when batteries are weak.

When pacemaker spikes are seen on the electrocardiogram, but there is no capture of the heart, the problem might be with the patient or with electrode placement. A chest X-ray may show a break in the electrode wire. The X-ray can also indicate if the electrode has moved or perforated the wall of the heart. Listening to heart sounds may reveal a "pericardial rub" that indicates irritation of the lining of the outside of the heart; the patient may feel pain due to the electrode. If the electrode is in the proper location, loss of capture may be due to chemical imbalances in the patient's blood. Low levels of potassium or calcium can increase the amount of energy needed for electrical capture of the heart.

Further troubleshooting can be done if the pacemaker is a programmable type. Some such pacemakers allow measurements of lead impedance, sensitivity, and output energy.

Stimulating the inner ear

Severe hearing loss can be partially corrected by a new kind of hearing aid, manufactured by 3M (St. Paul, MN), that electrically stimulates the inner ear. Unlike conventional hearing aids that amplify and filter sound, those new hearing aids perform a spectrum analysis and use the resulting signals to stimulate certain portions of the auditory nerve.

The human ear is shown in Fig. 6. Normally sound travels through the air to the eardrum. The eardrum vibrates and those vibrations are transferred to three small bones. Those bones in turn cause vibration of fluid in a snail-shaped structure called the cochlea. Sound causes a membrane, called the basilar membrane, in the cochlea to vibrate at various places. Which portion of the membrane vibrates depends on the frequency of the sound present. Specialized receptor cells along the membrane transmit signals to fibers of the auditory nerve. Thus the ear acts as a real time "spectrum analyzer" and transmits many parallel signals to the brain.

The portion of the basilar membrane that vibrates determines the pitch that is perceived. To a lesser extent the frequen-

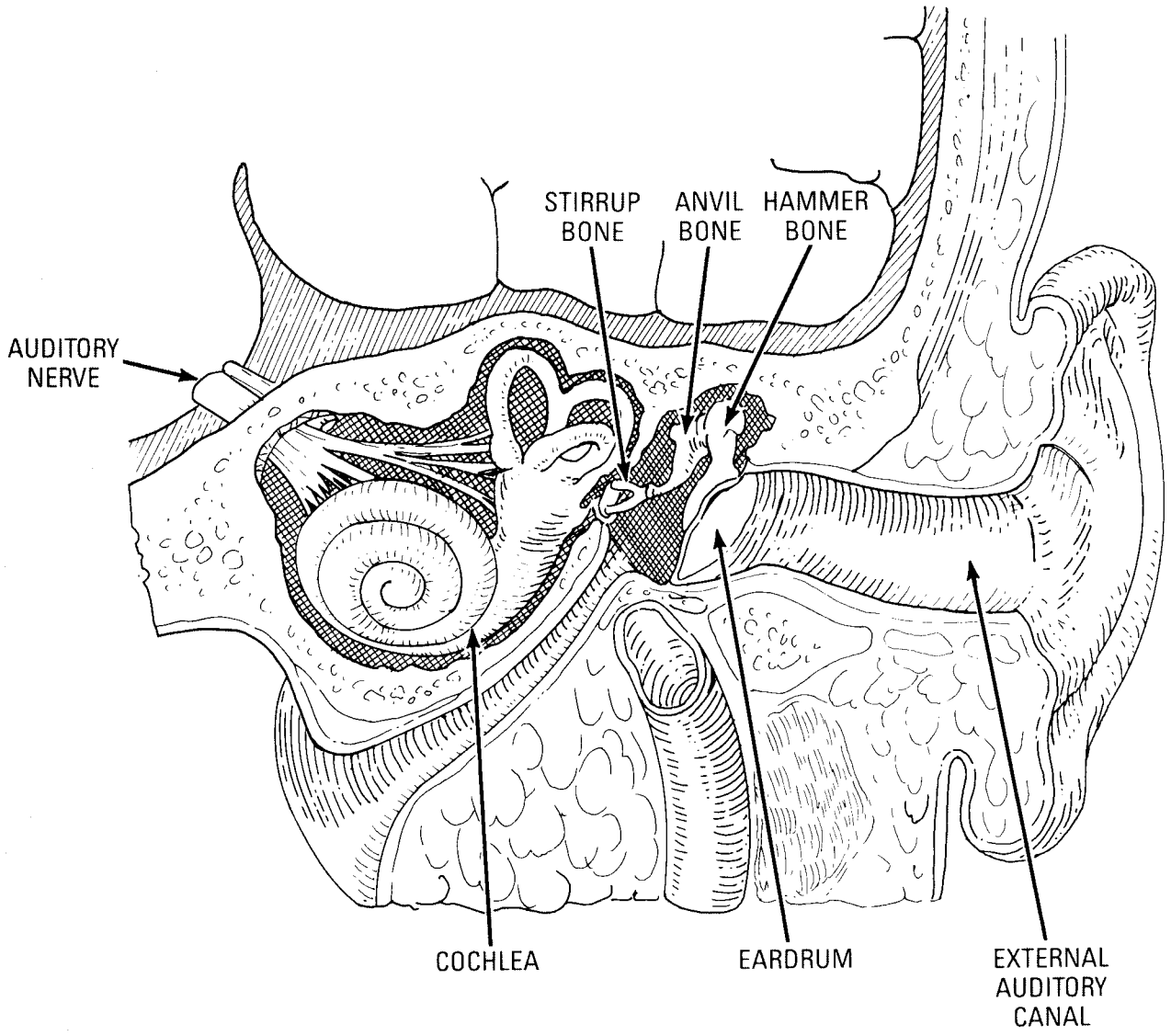

FIG. 6—THE MAJOR STRUCTURES of the human ear are shown here.

cy of stimulation of the basilar membrane as well as the location of the stimulation influences the perception of pitch; that is more important at lower frequencies.

Surgery and conventional hearing aids can correct many hearing problems that are caused by damage to the structures of the outer ear (ear canal) and the middle ear (the eardrum and three bones). But if the cochlea (inner ear) were damaged, there was little that could be done until recently. Clinical trials of cochlear implants began in 1972. Since then hundreds of people have had electrode arrays inserted into their inner ears.

The earliest cochlear implant, or prosthesis, used a single electrode. Such a device allows the user to recognize differences in a sound's intensity and duration, but allowed only low frequency sounds to be recognized. Single electrode devices allow users to recognize some environmental sounds and make lipreading easier.

Multiple channel cochlear implants contain spectrum analyzers and may have 16 electrodes. The stimulating voltage on each electrode is determined by the amplitude of sound in a certain frequency range. Information about the timing and amplitude of signals in various frequency ranges is preserved. Some users of multiple channel cochlear implants are able to understand speech in a limited manner. One study reported 70% recognition of vowels and 30% recognition of consonants without lipreading. Persons with cochlear implants can hear with sensitivity approaching normal. Although the sound is distorted, doorbells, car horns, and other sounds can be heard almost as well as by a person with normal hearing.

A typical cochlear prosthesis consists of two electronic assemblies—one outside of the head, and one that is surgically implanted. The external electronics package detects sound, performs spectrum analysis, and transmits signals to the implanted electronics.

The surgically implanted internal electronics package detects the transmitted signals and converts them so that they can be perceived by the auditory nerve. Some systems use a rigid electrode that is inserted directly into the auditory nerve. More commonly, however, a flexible electrode array is inserted into the cochlea and made to lie along the basilar membrane, which is then made to vibrate by the electrodes in response to the transmitted signal. That vibration is detected by the auditory nerve as normal. Which frequencies cause which electrodes to vibrate can be varied to adjust for differences in perception from patient to patient. **R-E**