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DEFIBRILLATORS, ELECTRICAL

Defibrillators are devices that are used to apply a strong electrical shock to the heart. The shock changes ventricular fibrillation to an organized ventricular rhythm or changes a very rapid and ineffective cardiac rhythm to a slower, more effective rhythm. In order to understand how defibrillators work, it will be necessary to briefly review the four cardiac rhythm disorders which are treated by defibrillators. These include ventricular fibrillation, ventricular tachycardia, atrial fibrillation, and atrial flutter. The common factor in all these rhythm disorders is that they are tachyarrhythmias; that is, they are very rapid heart rhythms. Each of the four rhythms and a normal rhythm is shown in Fig. 1. Figure 1(a) shows a regular heart rhythm, known as a sinus rhythm because the pacemaker is located in the sinoatrial node. The rhythm is regular, and the rate is within the range for effective pumping of blood by the heart. Figure 1(b) shows the most serious of tachyarrhythmias, ventricular fibrillation. Because of the asynchrony of electrical activity, the contraction of the many ventricular fibers is asynchronous, and virtually no blood is pumped.

Figure 1(c–e) shows atrial fibrillation, atrial flutter, and ventricular tachycardia, respectively. The usual reason that these rhythms require a defibrillator shock is that the ventricular rate is too rapid to allow the heart chambers to fill with blood between contractions. With inadequate filling, the cardiac output and blood pressure become dangerously low. For more details regarding criteria for diagnosis of these rhythms, the reader is referred to standard cardiology texts.

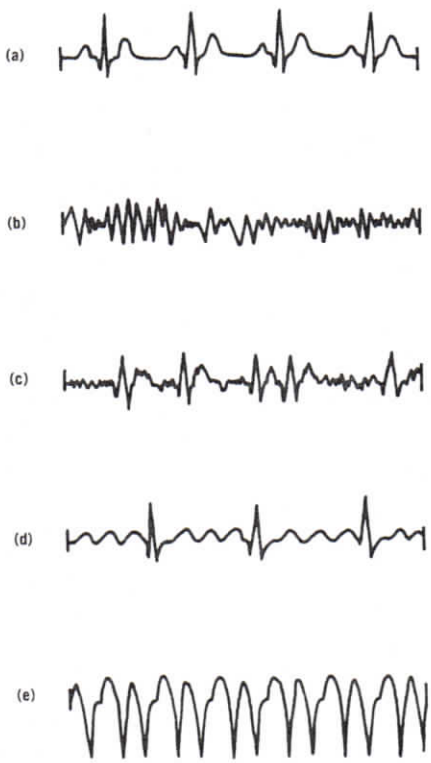

Figure 1. (a) A normal electrocardiogram. (b)–(e) Typical tachyarrhythmias which may require treatment with a defibrillator (see text).

MECHANISM OF FIBRILLATION

It is generally thought that these rhythm disorders are the consequence of one of two mechanisms; the first is circus motion, and the second is presence of multiple pacemaker sites in the heart. The concept of circus motion can be illustrated as shown in Fig. 2. In Fig. 2(a), a ring of tissue is stimulated at the point indicated by the arrow, and excitation travels around the ring in both directions until the two excitatory wave fronts encounter each other at the bottom of the ring. Since depolarized tissue cannot be excited, the stimulus wave front of activation is extinguished. This is the usual and normal sequence for heart tissue. In Fig. 2(b and c), the effect of a wave front being propagated in only one direction, due to altered metabolism, and of temporary refractoriness of the excitable tissue in the other direction is shown. In the latter case, the stimulus may travel around the ring in perpetuity [Fig. 2(c)], sending off "daughter" pacemaker signals to the rest of the heart each time the circle is traversed. This mechanism is often proposed to explain atrial flutter, which is a very rapid rate of the atria of the heart, and ventricular tachycardia, which is the rapid, but still organized, arrhythmia of the ventricles.

The second mechanism, which is presence of multiple pacemaker sites, proposes simply that there is more than one pace-

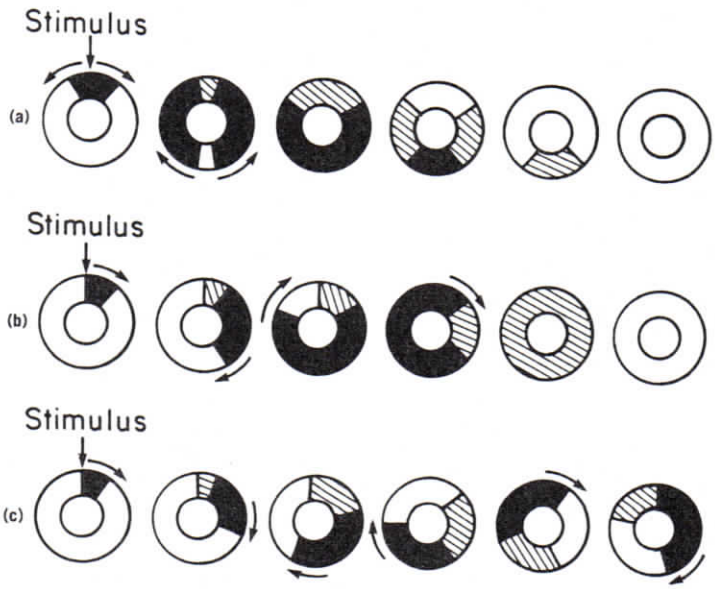

Figure 2. Circus motion in a ring of tissue. (a) The normal sequence of events is as follows: The impulse travels in two pathways, and the two impulses cancel each other when they meet. (b and c) If, for some reason, the impulse travels only one way, it may catch its tail (b) or continue indefinitely (c), depending on the rate of conduction of the excitatory impulse and on the rate of recovery after the response.

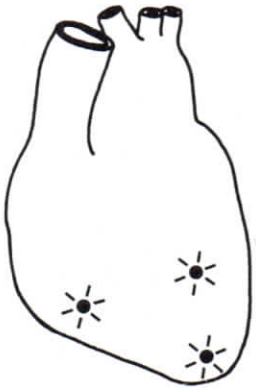

Figure 3. Diagram of three pacemakers located in the ventricles that may produce a tachyarrhythmia.

maker located in the myocardial cells of a heart chamber, as shown in Fig. 3, and that these pacemakers are depolarizing rapidly and independently to produce a chaotic and/or rapid rhythm disorder, recognizable in the electrocardiogram (ECG) as shown in Fig. 1(b). This is frequently proposed as the mechanism underlying ventricular and/or atrial fibrillation. In fact, considering the three-dimensional aspects of a mass of myocardial tissue in the heart, it is easy to understand how multiple pacemaker sites could initiate a tachyarrhythmia that would then deteriorate into circus motion.

Anything that alters conduction velocity or refractoriness of cardiac tissue, that alters the excitability or automaticity of pacemaker cells, that creates changes in membrane potentials of adjacent cardiac cells, or that converts latent pacemaker tissue to active pacemaker tissue can lead to these rhythm disorders. In practice, the most common cause of tachyarrhythmias is ischemia of heart tissue. Less frequent causes are other forms of heart disease, electric shock applied to the heart, a number of drugs (including many drugs used to treat heart disease), electrolyte disorders of the body fluids, metabolic acidosis and alkalosis, drowning, and hypothermia. Sometimes the cause is unknown.

DETECTION OF FIBRILLATION

The vast majority of the cardiac tachyarrhythmias, shown in Fig. 1(b–e), is detected by visual inspection of electrocardiographic tracings. This may be on either a monitor screen or on an ECG paper-strip recording. Occasionally, presence of a tachyarrhythmia is assumed under conditions in which a pulseless, apneic patient is encountered in the field and no electrocardiographic recording capabilities are available. If a defibrillation shock is applied under these conditions, it is referred to as "blind" defibrillation.

Automated electrocardiographic detection devices, which use signal processing and decision algorithms for detection of tachyarrhythmias, including fibrillation, tachycardia, and flutter are also available. These devices are used in implantable defibrillators, which will be described subsequently in this article, and in defibrillators, which are designed for automatic or semiautomatic use by emergency personnel. The automated detection schemes are usually based on counting the rate of QRS complexes per minute and/or looking for changes in a "normal" envelope for the ECG waveforms. Also, some of the detection algorithms include analysis for impedance changes due to respiratory or cardiac activity, and in the event that electrographic criteria are met for tachyarrhythmia, the device will not diagnose fibrillation, if the patient has mechanical evidence for cardiac or respiratory activity.

DESIGN OF CONVENTIONAL DEFIBRILLATORS

The earliest defibrillations were done in animals in 1899 by Prevost and Batelli, two Swiss scientists. They used capacitor discharge or alternating current waveforms. Later, in the United States, Kouwenhoven and colleagues studied effectiveness of other electrical waveforms. Beck et al. and Zoll et al., in the United States, had pioneered emergency human defibrillation with alternating current. However, the most widely used defibrillator in clinical medicine today is the damped sine wave defibrillator, which consists of a capacitor and inductor circuit for discharge of shock across the resistance of the patient's chest or heart. This basic circuit is shown in Fig. 4. This waveform was described first by Gurvich and Yuniev and was later popularized by Lown et al. and Edmark et al., who used the device clinically. The damped sine wave defibrillator replaced the simpler alternating current or pure capacitor discharge defibrillators because it produced fewer postshock arrhythmias and less tissue damage than the other types of defibrillators. Because the inductor adds some resistance to the discharge circuit, this type of defibrillator is not as efficient as a pure capacitor discharge in delivering all of the stored energy. However, delivered energy can be determined rather easily by knowing the characteristics of the particular defibrillator involved. The exact waveform produced by a damped sine wave defibrillator depends on the inductance and capacitance within this circuit as well as the resistance of the circuit and the resistance of the patient. Application of Kirchhoff's voltage law to the circuit shown in Fig. 4 gives

$$L\frac{di}{dt} + (R_i + R)i + \frac{1}{C}\int i\,dt = 0$$

where L is the inductance, i is the instantaneous current, t is the time, R_i is the internal resistance, R is the subject resistance, and C is the capacitance.

From this, the second-order differential equation, which describes operation of a damped sine wave defibrillator, is

$$L\,d^2\frac{i}{dt^2} + (R_i + R)\frac{di}{dt} + \frac{1}{C}i = 0$$

Depending on the particular values included for capacitance, inductance, and resistance, the waveform may be un-

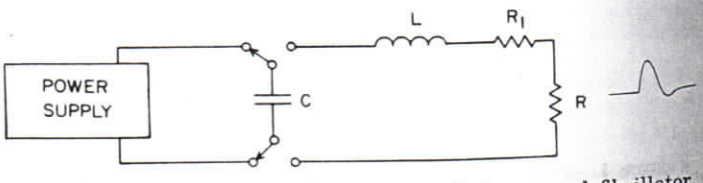

Figure 4. Basic circuit diagram of a damped sine wave defibrillator.

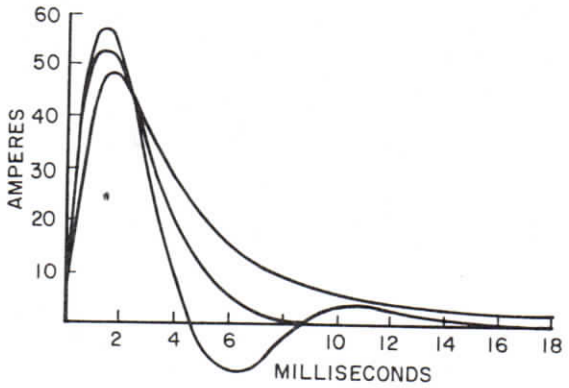

Figure 5. Waveforms from a damped sine wave defibrillator, showing underdamped (highest peak and negative current flow), critically damped (intermediate peak), and overdamped (lowest peak) conditions.

derdamped and therefore have a negative component or ringing of the circuit, overdamped, in which case the pulse decays very slowly, or critically damped, which is the condition in which capacitance, inductance, and resistance are matched to exactly produce no voltage reversal. Figure 5 shows these three conditions.

Actual calculation of stored energy can be achieved from the equation

$$W_s = \tfrac{1}{2}CE^2$$

where W_s is the stored energy in joules, C is the capacitance in farads, and E is the voltage on the capacitor. Delivered energy (W_d) is then calculated as

$$W_d = W_s \times \frac{R}{(R + R_i)}$$

By convention, most defibrillator manufacturers report delivered energy as the energy which would be delivered into a 50-Ω resistive load. This simulates the patient's resistance.

A few commercially available defibrillators produce trapezoidal or exponential decay waveforms by discharging a capacitor and then by stopping the flow of current by short-circuiting the capacitor at a time that is relatively short with respect to the time constant of the circuit (see Fig. 6). Current is generally truncated at a preselected duration between 3 and 30 ms, during which time the waveform will have decayed to some percentage of its initial value. There are two variations for selecting energy from trapezoidal wave defibrillators. One design has fixed pulse duration with variable voltage on the capacitor; the other design uses a fixed voltage on the capacitor with variable pulse duration. Either of these designs provides for a control to select the energy desired by the operator of the defibrillator. Both damped sine wave and trapezoidal waveform defibrillators are effective, and to date, there is no convincing evidence that one is superior to the other.

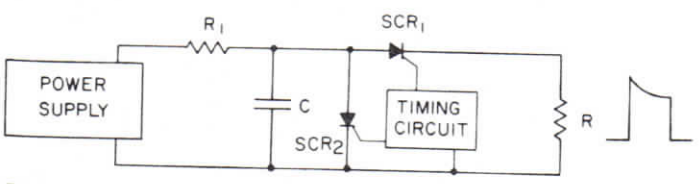

Figure 6. Basic circuit diagram of a truncated exponential decay defibrillator (sometimes called trapezoidal).

THE STRENGTH-DURATION CURVE FOR DEFIBRILLATION

Defibrillation is believed to occur when adequate numbers of myocardial cells are depolarized to extinguish the circus motion wave fronts through the myocardium. This occurs when cells are depolarized such that there is no large group of contiguous cells in the repolarized state and therefore are capable of being excited.

In practice, as the duration of current flow is decreased, the amount of current required to defibrillate is increased. The general shape of a strength–duration curve for defibrillation current is hyperbolic as shown in Fig. 7. Starting at very short durations of less than 1 ms, current requirement decreases as duration increases. This relationship is analogous to the stimulus–response characteristics of single irritable cells. Figure 7 also shows the strength–duration curve for energy. Since current is virtually the same for longer durations, the energy required to defibrillate will increase for very long durations. However, energy also increases at very short durations because current requirements are increasing. Hence, there is a minimum energy for defibrillation. In fact, this minimum energy occurs for clinically available waveforms at durations in the range of 3–10 ms. Finally, Fig. 7 shows the relationship of charge to duration. Charge is virtually linear with duration for the pulse durations which effectively defibrillate, greater charge being delivered to the heart as duration increases. It should be pointed out that these relationships have been verified only for durations of current between approximately 0.5 and 30 ms. Furthermore, the relationships in Fig. 7 do not apply to all waveforms, particularly those which have a prolonged descending ramp. It is thought that this descending ramp or "tail" rapidly refibrillates the heart, after defibrillation has been achieved.

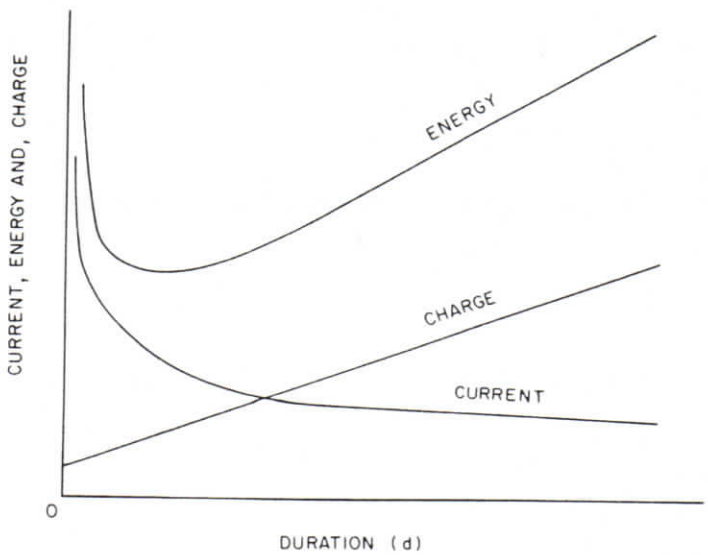

Figure 7. Strength–duration curve for defibrillation, showing current, energy, and charge relationships with pulse duration.

The strength–duration curve is important in the design of defibrillators because it shows how to achieve equal effectiveness in defibrillation with a medium current, short duration pulse, or a low-current, long duration pulse. High-current, very short duration pulses are undesirable, as they are known to produce postdefibrillation rhythm disorders to a greater degree than the other waveforms.

Another important variable related to the dose–response curve is the size of the subject for transchest defibrillation or the size of the heart for direct defibrillation. It has been clearly shown that large animals require more energy to defibrillate than small ones. It is also widely appreciated that pediatric patients require less energy than adult patients. The hearts of large animals require more energy than the hearts of small animals, but it is more difficult to demonstrate this phenomenon in humans because defibrillation is usually attempted in patients who have heart disease, metabolic disorders, or other problems which complicate the analysis of the heart weight to the defibrillation shock–strength relationship. Factors that affect defibrillation shock-strength requirements include various disease states, cardioactive drugs, electrolyte concentrations in the body, and possibly hypoxia of the myocardium.

Obviously, proper technique must be used to assure that the applied current passes through the myocardium, otherwise excessively great shock strengths will be required to achieve defibrillation. Practically speaking, this means that proper electrode use is important. Another important variable is the particular arrhythmia which is being treated. This will be discussed subsequently in the section, Uses for the Defibrillator.

Defibrillators have a number of characteristics other than simply the energy storage and discharge components. These include essential operating components as well as various options, accessories, and capabilities. A typical defibrillator delivers a maximum shock of approximately 360 J, but the waveform may vary depending on whether it is a damped sine wave or a trapezoidal waveform defibrillator. Waveform also varies according to variation in resistance of the chest or heart of different subjects. The defibrillator has electrodes for precordial placement and/or direct heart placement for internal open-chest defibrillation, as shown in Fig. 8. Many defibrillators also have the capability for synchronizing the shock to the ECG; specifically, this entails application of the shock during the QRS component of the ECG. This is necessary for treating all arrhythmias other than ventricular fibrillation because a shock applied during the T wave of the electrocardiographic complex may cause ventricular fibrillation and result in the death of the patient.

A typical defibrillator will have a power supply and energy storage section, circuitry for a discharge, controls, and electrodes for applying current to the subject. Figure 9 shows a block diagram of these fundamental components as well as the synchronization section.

Some defibrillators are battery powered and small for portable use. Many have electrocardiographic-monitoring capabilities. Finally, a number of features may be included, such as impedance-detection circuitry to identify poor electrode placement, recording capabilities for determining the actual energy delivered by each shock, and various kinds of audible and visual alarms for identifying dangerous conditions. Most units have the monitoring capability and the synchronizing capability because this enables the physician to be certain of proper diagnosis and to avoid fibrillating the patient's ventricles with an unsynchronized shock.

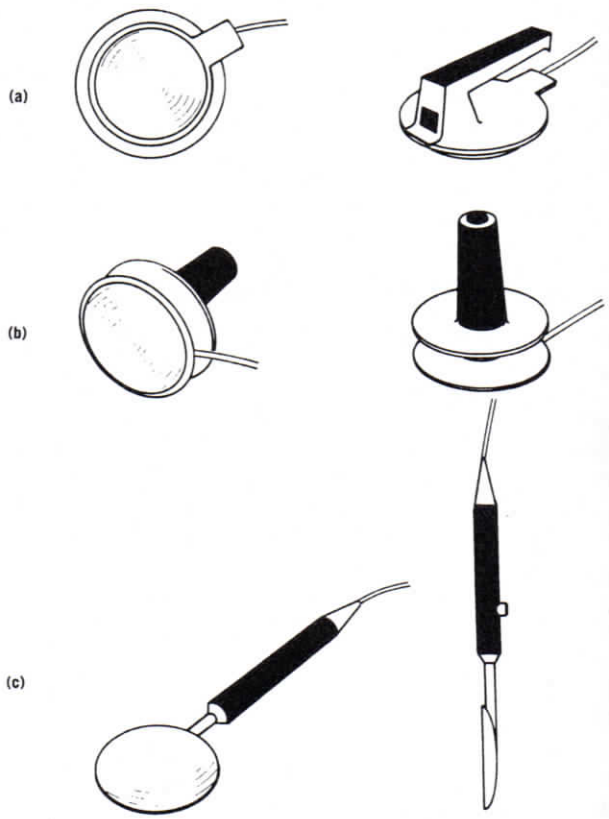

Figure 8. (a and b) Electrodes for precordial transchest defibrillation. (c) Electrodes for direct defibrillation.

ELECTRODES

Electrodes and their cables attach to the defibrillator to actually apply current through the body and heart to depolarize myocardial cells. Thoracic electrodes and direct heart electrodes usually consist of bare metal disks made of noncorrosive material. Size varies, with electrodes for transchest applica-

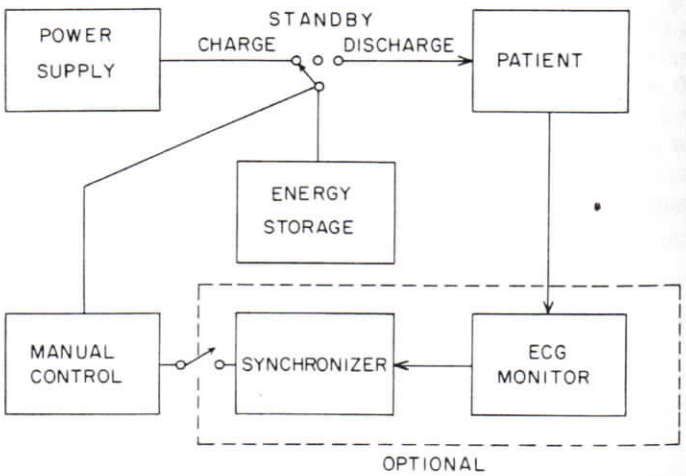

Figure 9. Block diagram of a defibrillator.

tion being in the range of 8–13 cm in diameter. Electrodes for direct defibrillation are usually 4–8 cm in diameter. There are two styles of electrodes for transchest application, the flatiron style [Fig. 8(a)] and the dagger style [Fig. 8(b)]. Electrodes for direct defibrillation usually resemble spoons mounted on the ends of long handles which can be easily inserted into a thoracic incision [Fig. 8(c)]. For transchest application, low-resistivity electrode interface material (gel) must be applied between the skin and the thoracic electrodes in order to establish a low-resistivity pathway for electric current flow. If this is not done, the high resistance may prevent defibrillation, and the high resistance generally produces skin burns due to the high impedance at the skin–electrode interface. Typically, transchest impedance to defibrillator shocks is from 25 to 150 Ω. Impedance for electrodes directly on the heart is typically from 20 to 40 Ω.

Preapplied electrosurgical dispersive electrodes have been used in transchest defibrillation after special modification to carry adequate current. They may be made of flexible conductive material with an adhesive perimeter or may be made of flexible metal foil covered with a conductive adhesive material to hold the electrodes to the skin. The electrodes can be applied ahead of time for use in patients at high risk, such as those in the coronary care unit. These preapplied electrodes have several advantages, including the fact that they save the time required to defibrillate by eliminating the need to apply conventional electrode gel, and they are safer, since the user does not have to touch the electrodes or the patient in order to apply the shock.

When electrodes are placed on the chest, they should be applied with either both electrodes on the anterior chest wall or with one on the anterior chest and the other between the scapulae of the back. In the former case, one electrode is placed over the apex beat area of the heart, which is about the fifth intercostal space in the midclavicular line on the left side of the chest. The other electrode should be placed in the second right intercostal space adjacent to the sternum. The placement for the anterior to posterior electrodes is to put one electrode over the cardiac apex beat and the second electrode under the back between the scapulae or just inferior to the left scapula.

Pediatric-size electrodes are available for defibrillation of pediatric patients; however, they are not usually needed, because fibrillation is relatively rare in small children and because it is possible to use adult-size electrodes on the pediatric patient if low energy levels are selected.

USES FOR THE DEFIBRILLATOR

There are basically three uses for defibrillators. The first is during direct surgical defibrillation. Frequently during cardiac surgery, the ventricles will spontaneously fibrillate or will be intentionally fibrillated by the surgeon. If the ventricles remain in fibrillation, it is necessary to defibrillate in order to bring the patient out of surgery successfully. The shocks are applied directly to the heart using 5–30 J. Higher energy shocks are damaging to the heart, and hence, modern defibrillators have a limit of 50-J output for use with internal electrodes. Energies required to defibrillate are typically 10 or 20 J, and defibrillation is successful in virtually all subjects. To discharge the shock, there may be an insulated push button on the defibrillator paddle, so that the surgeon can activate the defibrillator himself, or there may be a push button on the front panel of the defibrillator, which an assistant uses to apply the shock at the instruction of the surgeon.

Occasionally, direct defibrillation may be attempted with emergency ventricular fibrillation, such as occurs with a heart attack. This technique is less common than transchest defibrillation for emergencies, but is used in some institutions. The success rates for direct defibrillation are considerably lower during emergencies than during cardiac surgery, inasmuch as there is not the opportunity to control the environment, select the optimal time, and have the patient's heart perfused by cardiopulmonary bypass (extracorporeal circulation).

Cardioversion is the second use for a defibrillator. Cardioversion is application of synchronized, electrical shocks for treatment of arrhythmias other than ventricular fibrillation. This is virtually always a transchest shock. As stated previously, this includes atrial fibrillation, atrial flutter, and ventricular tachycardia. It is necessary to synchronize shocks to be certain that they do not fall during the T wave of the ventricular electrocardiographic complex, since this is associated with production of ventricular fibrillation, which is a potentially fatal arrhythmia. Cardioversion is usually attempted at energy settings of 5–20 J for ventricular tachycardia and atrial flutter and energies of 50–200 J for atrial fibrillation. In general, energies are low for the first shock, and the energy is increased gradually and only if previous shocks are unsuccessful in converting the arrhythmia to a functional rhythm. Frequently, cardioversion is used in combination with use of antiarrhythmic drugs, since cardioverting the heart into a normal rhythm usually will produce only transient success if the basic medical problem has not been corrected. Successful conversion to a regular, slower rhythm is the usual case, occurring in 60–95% of patients, depending on the arrhythmia being treated. However, atrial fibrillation is notorious for reverting back from the sinus rhythm to atrial fibrillation within a few hours, weeks, or months of successful cardioversion.

The third use for defibrillators is emergency defibrillation. In this case, the defibrillation shock is applied transchest, more commonly using precordial electrodes, but sometimes using anterior-to-posterior electrodes. Higher energy levels are required for this situation, with an initial shock of 200 J being followed by shocks of 300 or 360 J if the lower energy (i.e., 200 J) is unsuccessful. Because emergency ventricular fibrillation is associated with total cessation of perfusion, it is necessary to use cardiopulmonary resuscitation (CPR) if a defibrillator shock cannot be applied immediately. Cardiopulmonary resuscitation will forestall the death of the patient for a few minutes, while a defibrillator is being obtained. Use of drugs and other adjunctive therapy in combination with ventricular defibrillation is referred to as advanced life support (ALS).

AUTOMATIC IMPLANTABLE DEFIBRILLATORS

Totally automatic, fully implantable defibrillators have been developed. These resemble pacemakers, but treat tachycardias rather than bradycardias, and have much more powerful electrical shock output up to 30 J. Both ventricular fibrillation and ventricular tachycardia are treated, because of the rapid fatality of ventricular fibrillation and the high frequency with which ventricular tachycardia proceeds to ventricular fibrilla-

tion. These devices have been found to greatly reduce mortality in patients who are known to be at very high risk for sudden cardiac death syndrome. The implantable defibrillator contains sensors to detect cardiac activity, a signal-processing and algorithm circuit for making proper diagnosis of the tachyarrhythmia, and appropriate power supply and energy storage sections. Finally, electrodes are implanted in, on, or near the heart for delivery of the shock itself. Automatic implantable defibrillator components include the electrocardiographic sensing leads, the pulse generator (which also contains the signal-processing algorithm sections), and the current application electrodes (which may be placed on the epicardium or in the superior vena cava and right ventricle). The waveform consists of one or two truncated exponential decay waveform(s).

AUTOMATIC EXTERNAL DEFIBRILLATORS

An automatic external defibrillator is a device combining the electrodes and output features of the standard external defibrillator and a diagnostic circuit for automatically determining need to apply a shock. It can be used by paramedical personnel for emergency defibrillation.

TISSUE ABLATION

Finally, defibrillators may be used for destroying myocardial tissue in an effort to eliminate troublesome areas of the conduction system or latent pacemakers. The general strategy is to place a catheter in the heart, using fluoroscopy or electrocardiographic mapping. After placing the catheter in the area which is deemed undesirable, a defibrillator shock of a few hundred joules is passed through the catheter, producing a high-current-density, high-voltage shock, which damages the target tissue. In this way, the AV conduction pathways can be destroyed to protect the ventricles from life-threatening atrial rhythm disorders, or ventricular ectopic pacemaker sites can be destroyed to control ventricular tachycardia and ventricular fibrillation.

SIGNIFICANCE

Defibrillators are very frequently used and widely available for those trained in their use. They must be maintained in fail-safe operating condition due to the emergency nature of their use.

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DENTISTRY, BIOMATERIALS FOR.

See BIOMATERIALS FOR DENTISTRY.

DIFFERENTIAL COUNTS, AUTOMATED

The human body is an inviting target for many microorganisms looking for a boundless food supply and a place to multiply. To protect itself from invasion by these creatures, the body has many defense systems. The primary defense is mechanical, consisting of the skin and the lining cells (mucous membrane) of the respiratory, alimentary, and urogenital tracts. As long as these tissues remain intact, they block the entry of microorganisms into the body. Unfortunately, breaks in these barriers or the appearance of an especially aggressive intruder results in penetration. Once past this cordon defense, however, the intruder arouses a counteroffensive by activating the immune system. The system includes two closely interrelated components, humoral and cellular. The humoral component consists of soluble proteins (antibodies) which combine with the offending organism, either to neutralize it directly or to render it vulnerable to destruction. The cellular component of the immune system is comprised of many different cell types, which are known collectively as white cells (leukocytes), or sometimes, not quite correctly, as white blood cells (see Cell Counters, Blood). Unlike red blood cells, which reside and function only within the blood vessels, white cells use the bloodstream merely as a means of transportation to the sites where they are needed. Although the average life span of a white cell is approximately 2 weeks, the transit time through the bloodstream is a matter of only a few hours. By far, most white cells exist outside of the blood vessels, sequestered in such organs as the bone marrow, liver, lungs, gastrointestinal tract, spleen, and striated muscles. It is, nevertheless, the blood which is most often sampled to study white cells.

The leukocytes present in the normal circulating blood are classified into major groups known as granulocytes, lymphocytes, and monocytes (1). Each of these groups has been subclassified, depending on their physical appearance, staining characteristics, and enzyme activity. There are neutrophilic,