Control of Heart Action
by Electrical
and Mechanical Means

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ELECTRIC CURRENTS have long been known to have important effects on the heart, but only recently have they been used for therapeutic purposes in man. Two important clinical applications have now been established: electric stimulation of the heart for the production of effective beats, and electric countershock for the termination of ventricular fibrillation and other tachycardias.

Electric currents for stimulation or countershock may be applied either directly via electrodes touching the myocardium or indirectly with electrodes at some distance from the heart, usually externally on the chest wall. Direct electric stimulation of the heart is a familiar laboratory technic. It provides control of the heart rate in many physiologic and pharmacologic experiments, and much of our knowledge of cardiac excitability has developed from it. The clinical use of direct electric cardiac stimulation via a percutaneous needle electrode in the atrium for resuscitation from cardiac arrest was suggested by Hyman in 1932 (1). Electric stimulation of the heart was first applied successfully in man in 1952 externally by way of electrodes on the chest wall (2).
EXTERNAL CARDIAC STIMULATION

INDICATIONS

External electric stimulation of the heart is effective for the emergency resuscitation of patients from ventricular standstill of any etiology (Fig. 1). It has been used successfully in Stokes-Adams disease, in acute myocardial infarction, in reflex vagal standstill, in standstill due to cardiac drugs and in unexpected standstill during anesthesia, surgery and other therapeutic and diagnostic procedures (3).

After emergency resuscitation, intrinsic ventricular activity at times fails to reappear promptly, whereupon continued stimulation is necessary to maintain the heart beat. Under

Fig. 1.—Emergency resuscitation from ventricular standstill by external electric stimulation. Continuous electrocardiogram (lead aVF at half standardization) from patient with Stokes-Adams disease shows at first complete heart block with idioventricular beats (R) followed by ventricular standstill. Electric stimuli (E) terminated the standstill by producing ventricular responses (V). A spontaneous idioventricular beat (the first R in the fourth row) interrupted the regular externally paced rhythm; the next stimulus did not produce a ventricular response, i.e., it fell in the refractory period. With cessation of stimulation the idioventricular pacemaker resumed control. (Reprinted by permission of the American Heart Association, Inc., from Zoll, P. M., and Linenthal, A. J.: External and internal electric cardiac pacemakers, Circulation 28:455, 1963.)
these circumstances, external stimulation has been applied continuously for as long as 16 days. During this period, while the patient is being kept alive by electric stimulation, attempts can be made to arouse intrinsic ventricular activity with the careful, deliberate use of sympathomimetic amines. For this purpose, the intravenous administration of dilute solutions of epinephrine and isoproterenol has been found to be most effective and safe (4).

Finally, external electric stimulation has also been found valuable in patients with Stokes-Adams disease to prevent abnormal ventricular irritability, ranging from multifocal beats to tachycardia and fibrillation. Acceleration of the ventricular rate above a critical level, whether by electric stimulation or by sympathomimetic amines, prevents these manifestations of ventricular irritability and maintains a regular ventricular rhythm (5). The critical levels above which ventricular irritability is controlled vary somewhat from patient to patient and from time to time in the same patient: they usually range between 40 and 60 beats per minute, but are occasionally higher. Electric stimulation, however, does not terminate paroxysms of ventricular tachycardia or fibrillation that have already begun; countershock is often necessary for this purpose.

**Characteristics of Electric Stimuli**

Extensive experimental and clinical experiences have indicated the optimal features of the electric stimuli, that are the same for external as for direct stimulation except for the threshold levels. Monophasic, rounded waves were found to be more effective than a variety of other monophasic and biphasic wave forms (sinusoidal, spike and rectilinear). A duration of 2–3 milliseconds has been found to be most effective and safest: shorter impulses require larger currents to stimulate the heart; longer impulses do not significantly lower the current required and do increase the risk of multiple or repetitive responses to a single stimulus. The current and voltage of effective stimuli applied externally ranged in our patients from 50–200 milliamperes and from 15–100 volts.
Technic of Application

The pacemaker is attached to the patient by two output wires connected to circular electrodes, 3 cm. in diameter. The electrodes are placed over the precordium, one at or outside the cardiac apex (V4 to V6 position) and the other to the left of the sternum at or above the apical level (V2 to V4 position). The electrodes may be moved and their polarity may be reversed to find the lowest threshold of stimulation and the clearest electrocardiographic recording. The electrodes should be moved and cleaned several times a day to minimize local skin irritation. Good electric contact is made with electrode paste or jelly, and the electrodes are held in place with plastic handles or with a rubber strap encircling the chest. To prevent ineffective stimulation due to short circuit or high resistance at the skin surface, the electrodes should be at least 3 inches apart, they should be rubbed with sandpaper until bright and free of oxide formation, the skin between them should be clean and dry, the electrode paste should be rubbed vigorously on the skin and the paste should be reapplied when it dries; alcohol and electrode creams are not satisfactory.

During anesthesia, subcutaneous needles attached to the patient cable with Luer-lock connections are often used as electrodes for stimulation and monitoring because their application is simpler. During thoracic surgery, as for pacemaker implantation, long needles insulated except at the tips may be used to keep the connecting wires away from the sterile field.

Satisfactory electrocardiograms are usually obtainable during stimulation with selection of a suitable lead. To determine the threshold of effective stimulation, the frequency is set above the intrinsic ventricular rate, or about 60 per minute in case of ventricular standstill, and the amplitude is increased quickly from zero until the stimuli become effective (Fig. 2). When effective, the stimuli produce electrocardiographically demonstrable ventricular responses and synchronous peripheral pulses. The electric stimuli may be lowered below threshold intensity, may be varied in rate and may be interrupted momentarily to demonstrate the dependence of the ventricular
Electrocardiographic demonstration of the effectiveness of external electric stimulation; lead aVF at half standardization. A, electric stimuli (E) were increased in intensity until they produced ventricular responses (V) that replaced the slower idioventricular beats (R). B, variations in the rate of stimulation produced corresponding variations in the rate of responses. C, interruption of stimulation was followed by a ventricular pause of 4.64 seconds before intrinsic ventricular beats reappeared. D, subsequently, no intrinsic ventricular activity appeared when stimulation was interrupted for 5.48 seconds. (Reprinted by permission of the American Heart Association, Inc., from Zoll, P. M., Linenthal, A. J., and Norman, L. R.: Treatment of Stokes-Adams disease by external electric stimulation of the heart, Circulation 9:486, 1954.)

Responses upon them. When an intrinsic ventricular rhythm is present, its replacement by the faster, externally paced ventricular complexes is additional evidence of the effectiveness of the electric stimuli. Thereafter, effective cardiac stimulation is maintained with amplitudes somewhat above the threshold level.

In the emergency of ventricular standstill, there is not time to attach an electrocardiograph. Resuscitation of the patient, however, or palpation of synchronous pulses is satisfactory evidence of the efficacy of stimulation.
Untoward Effects of External Stimulation

No untoward cardiac effects of external electric stimulation have been observed. Repetitive, multifocal ventricular beats, ventricular tachycardia or ventricular fibrillation have not been produced. We have found no evidence of damage from the electric currents to the heart or to neighboring structures at necropsy.

The major untoward effects are chest pain and muscular twitch. The intensity of the pain and of the muscular contraction varies in different patients; in some it is negligible, in others it makes continued stimulation difficult. Meperidine hydrochloride (Demerol) or paraldehyde usually makes the discomfort tolerable and permits continued stimulation. Local infiltration with procaine hydrochloride under the electrodes does not reduce the pain. With prolonged stimulation the severity of the pain usually diminishes markedly and less medication is required. The muscular twitch is greatly reduced by curariform drugs. The only tissue damage from electric stimulation has been superficial ulcerations under the chest electrodes in patients treated for a day or more. This problem has been minimized by frequent, small changes in the positions of the electrodes and meticulous care of the skin. Flexible adhesive or soft rubber electrodes greatly reduce skin trauma; although useful for long-term monitoring, they are unsuitable for stimulation because of their relatively high electric resistance.

Direct Cardiac Stimulation

Indications

Long-term electric stimulation of the heart is valuable for the complete prevention of recurrent Stokes-Adams attacks, that are often not controlled by drugs. External stimulation is unsatisfactory for the long term because of pain and skin irritation. Direct cardiac stimulation with electrodes on or in the heart requires a current of low intensity and obviates these difficulties. For assured prevention of seizures, whether due to ventricular standstill or fibrillation, the unreliable intrinsic
ventricular pacemaker can be replaced by a reliable electric one to drive the ventricle continuously and indefinitely. In view of the complete unpredictability and lethal potentiality of Stokes-Adams attacks, a single episode not due to a transient cause (e.g., acute myocardial infarction, digitalis, toxicity) is a compelling indication for long-term electric stimulation. The degree and variability of the A-V block and the severity, frequency, type of arrhythmia and response to drugs of the seizures do not affect this indication. Even one minor spell in a patient with a long P-R interval is enough.

Long-term stimulation at normal rates is also indicated for patients with heart block who suffer from diminished cardiac output due to slow ventricular rates. Inadequate output may be manifest by congestive heart-failure, diminished exercise tolerance, angina pectoris, inadequate renal function and poor cerebral status.

Paired-pulse stimulation by endocardial catheter electrodes has been suggested as a means of reducing the number of contractions and of improving the cardiac output in tachycardias (6). Its clinical application is still greatly limited and it should be regarded as a highly experimental technic because of its uncertain effectiveness and the risk of ventricular fibrillation.

METHODS OF PLACING ELECTRODES

Direct stimulation can be accomplished by the placement of electrodes in the myocardium at surgical exposure or blindly by percutaneous puncture or by introducing a catheter electrode into the right ventricular cavity by way of a peripheral vein. Although it seems simplest, placement of a myocardial electrode by percutaneous puncture is not widely used even for emergencies because it carries the risks of damage to a coronary vessel, pericardial tamponade, ventricular fibrillation, infection and ineffective stimulation.

Endocardial catheter electrodes provide a means of direct cardiac stimulation without the need for thoracotomy. This approach has been used for temporary control and, with implanted pacemakers, for the long-term prevention of Stokes-
Adams attacks (7, 8). Difficulty with placement of the catheter, sudden ineffective stimulation from its displacement or breakage, infection, thrombophlebitis and embolism, interference with tricuspid valve closure, cardiac perforation and ventricular fibrillation from the accidental transmission of small external currents occur with significant frequency. Consequently, we prefer to avoid this technic if possible. Stokes-Adams attacks can usually be controlled temporarily by the intravenous administration of dilute solutions of epinephrine or isoproterenol. Long-term control is best achieved by implantation of myocardial electrodes at open thoracotomy or, in poor risk patients, extrapleurally by a parasternal or subcostal approach. Surgical implantation of myocardial electrodes has been the most widely used method, now in thousands of patients (9, 10). We have used this technic in 115 patients in the last 5 years (Fig. 3).

Fig. 3.—Direct cardiac stimulation by implanted pacemaker. Lead 1—top, complete heart block and slow idioventricular pacemaker (R) before surgery; middle, electric stimuli (E) from the artificial pacemaker produced regular ventricular responses (V); bottom, competition between conducted sino-atrial beats (R) and the artificial pacemaker. The fourth ventricular complex is a fusion beat. (Reproduced by permission of the Annals of Surgery 154:340, 1961.)
Myocardial Electrodes

The initial difficulties with direct cardiac stimulation resulted from progressively rising thresholds due to foreign body tissue reaction, which in effect separated the electrodes from excitable myocardium. This problem has been solved with electrodes of various metals, particularly platinum, and with meticulous avoidance of contamination of the electrodes or the site of implantation with foreign matter of any kind.

The remaining problem with myocardial electrodes is wire breakage, which is still encountered with all electrodes currently in use; breakage results from corrosion, fretting and flexion fatigue. Continual improvements are being made in the design of electrodes so that the goal of reliable performance for the many years that patients may need them now appears in reach.

Power Supply

The most widely used pacemakers consist of completely implantable units that include mercury batteries. In our system, the batteries have lasted from 16 to 46 months, but recent improvement in battery manufacture should make them more reliable. Our system is so designed that when the batteries fail, the rate of stimulation slows slightly; this change is not hemodynamically significant but it is useful in suggesting that the pacemaker be replaced promptly. In some systems of different design, failing batteries lead to acceleration that may be dangerous; such "runaway pacemakers" have produced ventricular tachycardia and fibrillation.

Several approaches have been tried to avoid implanted batteries that ultimately require replacement: they include implanted rechargeable batteries recharged across the intact chest wall from an external power source, and fuel cells and piezo-electric materials that convert bodily chemical and mechanical energy into electric energy. Although these power sources can produce cardiac stimulation, long-term, reliable performance has not been demonstrated and these technics, however promising, cannot be considered ready for clini-
cal application. Systems have also been developed with external components from which electric signals can be transmitted across the intact chest wall to internal radio receivers or induction coils. The implanted units are small and simple, and may even be placed directly on the heart so that component failure and breakage of connecting wires may be reduced. Despite their advantages, we consider such systems to be unacceptable because of the ease with which stimulation may be interrupted. Furthermore, preoccupation with the equipment and with its constant maintenance in precise position creates physical and psychologic hindrances to the normal activities of life.

**Risk of Ventricular Fibrillation from Competition**

The fear is often expressed that electric stimuli may produce repetitive responses and ventricular fibrillation when they fall in the vulnerable period after conducted beats or ectopic ventricular beats. As a result, A-V conduction of any degree has even been suggested to be a contraindication to implantation. Over a third of our patients have had such competition (Fig. 3). In our intensive studies of these patients, we have observed repetitive responses in only 3 patients: in 2 patients the phenomenon of repetitive response occurred transiently only during infusion of isoproterenol; in the third patient, the phenomenon was spontaneous and recurrent. Never did clinically significant ventricular tachycardia or fibrillation occur from this mechanism.

It must be recognized that the threshold for repetitive responses may at times be lowered by factors such as local myocardial ischemia or catecholamines, so that this hazard may not be entirely avoidable. Such arrhythmias should be most infrequent with rounded, monophasic stimuli of short duration (2 milliseconds) and 7.5 volts or less, at rates that usually suppress ectopic ventricular activity (70 per minute or more) and by careful placement of electrodes to minimize myocardial damage.

Considerable interest has been aroused by pacemakers in
which the stimuli are triggered by the P waves or appear “on
demand” after a fixed interval without a beat (11). Competi-
tion between the electric pacemaker and conducted beats is
avoided with these systems but in our view, competition is not
of clinical concern. The patients are not disturbed by the ir-
regularity, and repetitive response is rare and has never been
observed to produce clinically significant tachycardia or
fibrillation.

Furthermore, with P-wave-synchronized pacemakers, the
ventricular rate varies normally and the normal atrioven-
tricular sequence restores the contribution of atrial systole to
ventricular output. In acute observations comparing the stroke
outputs of individual beats, these physiologic refinements seem
highly advantageous but the average cardiac output over the
long-term is not greatly improved. Most of our patients with
asynchronous, fixed-rate pacemakers have been well without
cardiac symptoms at levels of activity compatible with their
genral physical condition. Many engage in vigorous physical
activity (water-skiing, swimming, hunting) and many un-
derwent severe stresses of normal pregnancy, major illnesses
and surgery without difficulty. Severe congestive failure was
usually completely controlled in our patients after implanta-
tion; in the few instances in which it persisted, extensive car-
diac or other diseases were adequate explanations.

The major disadvantage of P-wave-synchronized pacemak-
ers is their increased complexity with consequent increased
risk of component failure. This objection is supported by con-
tinuing experiences of instrument failures. In striking contrast
are the experiences with fixed-rate pacemakers; in our series,
for example, there have been only 2 component failures,
other than battery depletion or wire breakage, in the 177 in-
struments of the present type implanted since June 1961. It
should be kept in mind that the primary purpose of artificial
internal pacemakers is to provide completely reliable preven-
tion of Stokes-Adams attacks; certainly this basic objective
should not be compromised for minor secondary gains.

In addition to its important therapeutic value, electric stimu-
lation of the heart, both external and direct, has provided op-
opportunities for physiologic and pharmacologic studies, many of which have previously not been possible in man, and has led to revived interest in heart block and Stokes-Adams disease in the last decade. Such studies have included the relation of heart rate to cardiac output, effects of sympathomimetic amines on ventricular rhythmicity and atrioventricular conduction, depression of idioventricular pacemakers by rapid stimulation, delineation of a new curve of ventricular excitability, features of antegrade and retrograde atrioventricular conduction, an analysis of fusion beats and new insight into the nature of partial and complete A-V block (12). Advances in our knowledge of cardiac physiology, particularly in relation to arrhythmias, must surely come from these observations, that have their origin in this new electrical approach to the treatment of this long-recognized, somewhat uncommon and still poorly understood disease.

ELECTRIC COUNTERSHOCK

Electric countershock applied directly across the exposed heart for the termination of ventricular fibrillation was developed by Prevost and Battelli in 1899 (13) and was first applied successfully in man in 1947 by Beck, Pritchard and Feil (14). This technic required thoracotomy and was usually combined with direct cardiac compression or "massage." This formidable procedure was often undertaken with reluctance or not used at all. External countershock was first applied successfully in man in 1955 for the termination of ventricular fibrillation and tachycardia (Fig. 4) (15). The effectiveness of this procedure together with external electric stimulation and external cardiac compression has greatly improved the management of cardiac arrest due to either ventricular fibrillation or standstill and has made thoracotomy necessary only rarely for this emergency. Direct countershock is still performed at times when the chest is already open. External countershock has subsequently become widely used for the elective termination of tachycardias of all kinds (Fig. 5); the great success of this application constitutes a major advance in the treatment of cardiac arrhythmias (16).
**Fig. 4.**—Continuous tracing showing the usual idioventricular pacemaker, ventricular fibrillation, ineffective AC countershock of 240 volts and successful countershock of 300 volts with return of idioventricular rhythm. (Reproduced by permission of the New England J. Med. 262:108, 1960.)

**Fig. 5.**—Continuous tracing showing successful termination of ventricular tachycardia (167 per min.) by 450-volt AC countershock followed by a few A-V nodal beats and then normal sinus rhythm.

**Indications**

The clinical decision to terminate a rapid arrhythmia is based on an evaluation of the disturbances it produces, balanced against the likelihood of success, the risks and the difficulties of the undertaking. The indications to terminate an arrhythmia may vary widely from the desperate emergency of ventricular fibrillation and the urgent manifestations of hypotension, pulmonary edema and cardiac pain to the problematical prevention of embolization in an asymptomatic patient and the relief of minor palpitation. The termination of arrhythmias involves either the application of external countershock or the administration of an anti-arrhythmic drug. External countershock is almost always successful and immediately so, whereas drugs are more uncertain and usually much...
slower. In the emergency of ventricular fibrillation, countershock obviously should be chosen and applied without hesitation. In lesser but still desperate arrhythmias, however, an intravenous drug (e.g., procainamide) may be effective within minutes, long before arrangements can be made for anesthesia and countershock. The risk of serious arrhythmia and even death is present both with countershock and with drugs; comparison is difficult because the risks vary with the clinical situation, with the choice and dose of drug and, perhaps with the type, strength and number of countershocks. In view of the unpredictable and potentially fatal complications of any type of treatment, termination of an arrhythmia should not be undertaken without adequate clinical need and without appropriate precautions. Difficulties both in the application of countershock and in the administration of drugs vary widely from case to case so that generalizations are hard to draw. Usually, however, the prompt and highly successful action and the low risk of external countershock make it a relatively simple and satisfactory procedure. It is indicated when the patient's condition is desperate or intolerable and drug therapy is ineffective, too slow or otherwise inadvisable. It may well be safer at times than the administration of large doses of anti-arrhythmic drugs.

**Characteristics of Countershock Current**

Many variations have been proposed in the features of the electric current needed for countershock. Two major types of current, alternating and direct, are in general use at the present time.

The AC countershocks that we use consist of 60-cycle alternating current of 0.15-second duration and of 150-750 volts; across the usually low output resistance of about 50 ohms, the current flow is about 3-15 amperes (External AC defibrillator manufactured by Electrodyne Company, Westwood, Massachusetts). The frequency, duration and voltage were chosen after extensive experimental studies to determine the optimal features with which countershocks terminate arrhythmias with
minimal risks of ventricular fibrillation and of other cardiac damage.

The most generally used DC countershock is a somewhat underdamped discharge from a 16-microfarad capacitor through a 100-millihenry inductance with a resultant, largely monophasic wave of 2.5–3.0-millisecond average duration (17). The intensity of the shock usually ranges from 50–400 watt-seconds (joules) with peak values of up to 3,500 volts and 75 amperes (External DC defibrillators manufactured by Electrodyne Company, Westwood, Massachusetts and American Optical Company, Buffalo, New York).

**Technic of Application**

Large electrodes, 8.5 cm. in diameter, are placed on the chest wall in the V_e and V_g or V_s positions so that the heart lies in the electrical field between them. Other placements with oblique or anteroposterior axes seem to offer no advantage. Certain technical details must be carefully observed if countershock, whether AC or DC, is to be effective: (1) the electrical resistance between the electrodes must be low (gray oxide film, that slowly forms on the electrode surface and increases the resistance, must be removed with fine sandpaper); (2) highly conductive electrode paste must be applied generously; and (3) the electrodes must be pressed firmly with their entire surface against the chest wall. To avoid loss of current across a surface shunt, the skin between the electrodes must be wiped clean and dry. Small caliber or long extension cords constitute significant resistances and must be avoided with AC instruments, which require large instantaneous current flow; this precaution does not apply to DC instruments.

Continuous monitoring of the cardiac rhythm with an electrocardiograph and oscilloscope is necessary for observation of the effects of the countershock and for management of subsequent arrhythmias. An external electric pacemaker and appropriate drugs for intravenous administration should be at hand. Conscious patients should be put to sleep with a short-acting anesthetic agent such as intravenous thiopental;
the procedure is, therefore, usually performed in the operating room for ease and safety of anesthesia. Even in our desperately ill patients the brief light anesthesia has not been of significance.

RESULTS

Since 1955, we have used external AC countershock over a thousand times for the termination of ventricular fibrillation. Although effective cardiac action was frequently not restored in the anoxic, moribund patients, the fibrillation was always terminated with voltages of 150–750 volts when the technic was properly applied. Other arrhythmias were terminated 333 times in 80 patients (Table 1). In only one instance, a patient with congenital heart disease and atrial fibrillation, properly applied AC countershock failed to terminate the arrhythmia; in this case DC countershocks, even of 400 joules, were also ineffective. Most of these patients were desperately ill with circulatory collapse or congestive failure, and many had suffered recent myocardial infarction. Ventricular tachycardia was the most common arrhythmia and the need of termination was usually urgent. In a smaller number of instances, atrial fibrillation and flutter were terminated, usually for comparatively minor indications. With termination of the arrhythmia there was generally prompt and often dramatic improvement in the patient’s clinical condition, with clearing of congestive failure and recovery from circulatory collapse.

Direct-current countershock has been widely used since 1962, particularly for the electric termination of atrial fibrillation and flutter. Its effectiveness is generally reported as 90–95%.

<table>
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<tr>
<th>TABLE 1. AC COUNTERSHOCK FOR ARRHYTHMIAS (1955-1966)</th>
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<tr>
<td>ARRHYTHMIA</td>
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<tr>
<td>Ventricular Fibrillation</td>
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<tr>
<td>Atrial fibrillation</td>
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<td>Atrial flutter</td>
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<tr>
<td>Supraventricular tachycardia</td>
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<tr>
<td>Tachycardia, ? type</td>
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<tr>
<td>Ventricular tachycardia</td>
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but these figures may well be too low because prompt recurrence of arrhythmia with lack of clinical benefit has often been counted as a failure of the countershock to terminate the arrhythmia at all (Fig. 6).

It would appear from these considerations that both AC and DC countershocks are almost always effective in terminating fibrillation and tachycardia of any kind and that there is little to choose between them in this regard. Recurrence is a major remaining problem in the clinical management of tachycardias. The frequency of recurrence of all types of arrhythmias is high in our own and in most reported series, after intervals ranging from seconds to years. Recurrence depends on the clinical state of the patient, the underlying cardiac disorder, precipitating factors and the responsiveness to anti-arrhythmic drugs rather than on the means by which the arrhythmia is terminated. Relapse of atrial fibrillation, for example, within 1 year after countershock varies from 50-90% and is similar to previous experiences following quinidine conversion. The clinical value of terminating a tachycardia must be assessed in the light of the severity of the circulatory disturbance produced by the tachycardia and the interval before recurrence. Desperate arrhythmias such as ventricular fibrillation and tachycardia must obviously be terminated promptly without regard for the question of recurrence. Even with repeated epi-

Fig. 6.—Continuous tracing showing successful termination of atrial fibrillation by 400-joule DC countershock but with return of atrial fibrillation in 13 seconds.
sodes, a short interruption may provide opportunity for improving the cardiac status and preventing further recurrence. On the other hand, with less serious arrhythmias like atrial fibrillation, recurrences even after several months may negate the value of the treatment.

The cause of an arrhythmia, e.g., digitalis toxicity or acute myocardial infarction, is not an important consideration in the decision to apply countershock. It has been suggested that desperate arrhythmias due to digitalis toxicity cannot be terminated or will recur promptly, since countershock does not affect the cause. We have, however, observed several instances of digitalis-induced tachycardia that were successfully handled with countershock. Obviously, countershock does not remove the cause of any arrhythmia.

Risks

Avoidance of injury to attendant personnel is a primary consideration in the design of the equipment and in the technic of application. The output circuit of the instruments should be isolated from the ground to prevent a shock to anyone touching the instrument or the patient. Personnel should be warned to avoid contact with the patient and the instrument, and one person should not hold both electrodes. In addition, foot switches should be avoided, since they permit too easy a discharge of the countershock at an improper time. Parenthetically, it should be noted that AC instruments are much simpler and sturdier, and are much safer to handle and repair than DC ones.

Countershock applied directly to the heart is known to produce myocardial damage in the form of an electric burn, particularly with poor contact of the electrode with the cardiac surface. From the time of the introduction of external countershock there has been concern about the risk of myocardial damage from it. Many studies of changes in enzymes, local temperature and electrocardiographic complexes have given conflicting results, but no indisputable evidence of significant cardiac damage. Clinical experience with both AC and DC
countershock, now in thousands of cases, has established the safety of the technic in this respect, with no manifestations of cardiac injury or diminished cardiac function. Indeed, repeated countershocks have been reported in 6 patients, 4 with AC and 2 with DC, from 20 to over 300 times, and no resulting cardiac damage was subsequently found at autopsy. One possible exception is an experience recently reported of 4 patients who suffered acute pulmonary edema after DC countershock; the pathogenesis of the pulmonary edema and its relevance to the point under discussion are not clear (18).

The production of ventricular fibrillation has been a major concern in the application of countershock for lesser arrhythmias. Indeed, ventricular fibrillation was produced 4 times with countershocks of less than 450 volts in the first 307 episodes in our series, but another countershock terminated the fibrillation promptly every time. It is known that relatively small AC and DC countershock might produce ventricular fibrillation whereas larger ones would not do so and, moreover, would terminate fibrillation. Accordingly, we now begin with countershocks of 450 volts and have not produced ventricular fibrillation in the last year.

To reduce the risk of ventricular fibrillation, DC countershock, synchronized to fall before the vulnerable period, was introduced (17). The vulnerable period has long been recognized as part of the relative refractory period (on the descending limb of the T wave) when a strong electric stimulus often causes repetitive beats, tachycardia and fibrillation. Although synchronized DC countershocks are clearly less likely to produce ventricular fibrillation than unsynchronized ones, this risk is not entirely obviated: a number of instances of ventricular fibrillation produced by properly synchronized DC countershocks have occurred. The risk of ventricular fibrillation appears to be small with either high-voltage AC or synchronized DC countershocks; nevertheless, countershock of either kind should be undertaken only if the operator recognizes this risk and is prepared for it.

Many arrhythmias other than ventricular fibrillation occur frequently after countershock, both AC and DC. Atrial and
ventricular premature beats, singly, in bigeminy and even in bursts of 2 or 3 multifocal beats often occur immediately after the countershock and usually last seconds to only a few minutes. Anti-arrhythmic drugs such as quinidine are often given before the countershock to diminish this irritability as well as to prevent recurrence of the initial arrhythmia; agents like procainamide and lidocaine (Xylocaine) may be given intravenously after the countershock for rapid effect. Deep forced inspiration with a breathing bag followed by an abrupt expiration often abolishes bigeminy (this maneuver was initially suggested by Dr. Robert Smith, Anesthesiologist-in-Chief at the Children's Hospital Medical Center, Boston).

Depression of rhythmicity also appears occasionally in the form of atrioventricular nodal rhythm, minor degrees of A-V block and even ventricular standstill. All these disturbances are usually short, but atrioventricular nodal rhythm has lasted for 2 days, and one moribund patient was observed who could not be resuscitated from persistent ventricular standstill. Atropine may be useful in terminating persistent atrioventricular nodal rhythm.

Prolonged bigeminy, A-V block and A-V nodal rhythm have been attributed to digitalis toxicity that became apparent only after countershock. For this reason digitalis is ordinarily withheld, if possible, for a few days before electric countershock.

CARDIAC MONITORING

Crucial to the successful resuscitation of patients from cardiac arrest is prompt recognition of the onset of the emergency. The interval between circulatory arrest and the resumption of effective ventricular output must be brief, preferably less than 1 minute, if cerebral and cardiac function is to return unimpaired. Cardiac arrest may pass unrecognized and untreated in unattended or anesthetized patients unless a cardiac monitor immediately signals the emergency.

Ideally, a cardiac monitor should be easy to apply, should give an audible alarm with the onset of arrest, should demonstrate the mechanism of arrest, whether ventricular standstill
or fibrillation and should be combined with a pacemaker to provide means of immediate, even automatic, external electric stimulation in case of standstill (19). Such monitors are used to great advantage in patients when there is a special threat of cardiac arrest, as in acute myocardial infarction, Stokes-Adams disease, anesthesia, cardiac arrhythmias and cardiac catheterization.

**PROGRAM FOR CARDIAC ARREST**

Equally important to the success of resuscitation is the prompt institution of a pre-arranged and well rehearsed program of action (Table 2). The emergency restoration of circulation may involve external electric stimulation for ventricular standstill, external electric countershock for fibrillation, manual blows to the precordium, external cardiac massage, cardiac puncture and, in appropriate circumstances, even thoracotomy with direct cardiac massage. The sequence of

<table>
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<th>TABLE 2. PROGRAM FOR CARDIAC ARREST</th>
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<td><strong>Program for Cardiac Arrest</strong></td>
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<tr>
<td>1. Emergency restoration of circulation</td>
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<td>Precordial blow</td>
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<tr>
<td>External electric stimulation or countershock</td>
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<tr>
<td>External cardiac massage</td>
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<tr>
<td>Cardiac puncture and intracardiac epinephrine</td>
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<tr>
<td>Thoracotomy and massage</td>
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<tr>
<td>if other methods fail</td>
</tr>
<tr>
<td>if adequate help is available</td>
</tr>
<tr>
<td>if prognosis is favorable:</td>
</tr>
<tr>
<td>no irreversible cerebral damage</td>
</tr>
<tr>
<td>general condition good</td>
</tr>
<tr>
<td>Artificial respiration</td>
</tr>
<tr>
<td>2. Restoration of intrinsic cardiac rhythm</td>
</tr>
<tr>
<td>Electrocardiograph or monitor</td>
</tr>
<tr>
<td>Electric stimulation or countershock</td>
</tr>
<tr>
<td>Drugs: epinephrine, isoproterenol, procainamide, calcium salts, norepinephrine, sodium bicarbonate</td>
</tr>
<tr>
<td>3. Prevention of recurrent episodes</td>
</tr>
<tr>
<td>Cardiac monitor</td>
</tr>
<tr>
<td>Intravenous epinephrine, isoproterenol, atropine</td>
</tr>
<tr>
<td>Oral ephedrine and isoproterenol</td>
</tr>
<tr>
<td>Internal electric pacemaker</td>
</tr>
</tbody>
</table>
the procedures depends upon their relative safety, speed of applicability and mechanism of the arrest. Artificial respiration may also be necessary in the event of respiratory arrest.

After circulation has been restored, two additional problems may arise: effective intrinsic cardiac rhythm may not return promptly, and cardiac arrest may recur. The permanent prevention of recurrent episodes is a specially difficult problem in Stokes-Adams disease and often requires long-term direct cardiac stimulation with internal electric pacemakers.

REFERENCES