The Effects of External Electric Currents on the Heart

Control of Cardiac Rhythm and Induction and Termination of Cardiac Arrhythmias

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Clinically, application of electric currents to the heart has been limited to defibrillation in the operating room when the heart is exposed. In this paper technics are described for the external application of stimulating and countershock currents. They have been used successfully in man to terminate ventricular standstill from any cause and to stop ventricular tachycardia or fibrillation. For experimental purposes an additional technic has been developed for producing various cardiac arrhythmias by rapid external stimulation of the heart.

Three types of cardiac effects have long been known to result from the direct application of electric currents to the exposed heart: (1) stimulation of ectopic beats, (2) induction of arrhythmias, and (3) termination of arrhythmias. These procedures have been limited in experimental and clinical use because of the necessity of opening the chest. In this paper, technics are described whereby the same cardiac effects can be obtained when the currents are applied externally.

1. Single or repetitive electric stimuli applied directly to the heart are known to produce single ectopic beats or to assume control of cardiac rhythm. We have shown that electric stimulation applied externally across the unopened chest in man also produces effective cardiac beats and constitutes a valuable means of cardiac resuscitation from ventricular standstill.1-5 Evidence is presented here that the external electric stimulator acts as an intrinsic cardiac pacemaker.

2. Repetitive electric stimuli have also been applied directly to the exposed heart to produce supraventricular and ventricular arrhythmias for experimental purposes.6,9 We are presenting here a new technic of inducing these arrhythmias by electric stimulation of the heart across the unopened chest in the experimental animal with very rapid, monophasic impulses. This method of producing arrhythmias may prove valuable for physiologic and pharmacologic studies.

3. Electric countershock across the exposed heart has become the accepted therapy for ventricular fibrillation.9,10 In the experimental animal, ventricular fibrillation has also been terminated by external application of electric countershock across the unopened chest.11,15 In this report we also show that countershock electric current of the same type that has long been used in open-chest defibrillation will, when applied across the unopened chest, instantaneously terminate atrial, nodal, and ventricular tachycardia as well as atrial and ventricular fibrillation. This technic of external countershock has been applied successfully for the first time to resuscitate a patient from ventricular fibrillation.

Methods

Studies on control of the cardiac rhythm by an external electric cardiac pacemaker and on external countershock defibrillation were performed primarily on 22 normal, adult mongrel dogs.

The data concerning the induction and termination of supraventricular and ventricular tachycardia...
were obtained in these dogs, in 5 normal domestic pigs (4 to 8 weeks of age and weighing 15 to 30 pounds), and mainly in 14 pigs previously subjected to ligation of the terminal portion of the left circumflex coronary artery. All pigs survived this ligation and were fully active within 24 hours. Postmortem examination showed small areas of myocardial infarction and significant interarterial coronary anastomoses.16

We have also observed the effects of external electric stimulation in over 70 patients and of external countershock in 5 patients to whom these currents were applied for therapeutic purposes.

Figure 1 shows the optimum characteristics of the 2 forms of electric current that were used for external stimulation and countershock of the heart.

**Stimulating (Pacemaking) Current.** A modified clinical cardiac pacemaker* with an extended range of rates of stimulation provided monophasic, rounded impulses of 3 msec. duration. The low internal impedance of the instrument (approximately 50 ohms) permitted adequate power output even across low body resistances. The stimuli were applied at rates of 60 to 1200/min. across external or esophageal electrodes. In the animals, surface electrodes (3 cm. in diameter, of brass with satin-nickel plating, and covered with conductive paste) or subcutaneous needle electrodes were placed on the right and left anterolateral chest walls in the cardiac area; at times a surface electrode was paired with an esophageal wire electrode at atrial or ventricular levels. In man, 2 surface electrodes were placed a few inches apart near the cardiac apex.

**Countershock (Defibrillating) Current.** Alternating current (60 cycle, 0.15 second, 180 to 720 volts) was applied across the unopened chest with large electrodes.* A special 6:1 isolation step-up transformer and a variable autotransformer* were used to convert the 120-volt line current to 0 to 720 volts. The duration of the countershock was fixed by a suitable condenser in a relay circuit similar to that described by MacKay, Mooslin, and Leeds.17 The step-up transformer and the power-relay contacts were designed to carry approximately 12,000 watts (15 amperes at 720 volts) for 0.15 sec., repeated at intervals of 1 sec. In the animals, the countershock electrodes (10 by 15 cm. tin plates) were covered with electro paste and applied firmly to the anterolateral chest walls in the cardiac area with a rubber strap to insure optimum electric contact. More recently, in man, in order to provide more rapid and convenient application of the countershock, copper electrodes, 7.5 cm. in diameter, mounted on heavily insulated handles, were held firmly in place on the chest wall, one to the left of the lower end of the sternum and the other lateral to the apex. Care was taken not to touch the electrodes or the animal's or patient's body during application of the high voltages.

The electrocardiographic response to external electric stimulation was monitored in all experiments with an oscilloscope† and recorded on a multichannel direct-writing electrocardiograph with paper speeds of 25 to 100 mm./sec.‡ Lead aV, was used. An input-limiting preamplifier‡ and the “Automatic switch” of the electrocardiograph, which shortens the time constant of the amplifier, were used at times during application of the large countershock currents to prevent wide swings of the electrocardiographic baseline and damage to the stylus.

In several experiments blood pressure tracings were recorded from the carotid artery or aorta with an electric pressure transducer.

**CONTROL OF CARDIAC RHYTHM BY ELECTRIC STIMULATION**

In the experimental animal and in man the electric stimulator acts as an externally controlled cardiac pacemaker, producing effective beats. The cardiac response to electric stimuli depends upon their frequency, intensity, and timing in the cardiac cycle. A stimulus above threshold in intensity that arrives in the responsive phase of the cardiac cycle produces an ectopic beat, either supraventricular or

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* The cardiac pacemaker and external defibrillator are manufactured by the Electrodyne Company, Norwood, Mass.


‡ Cathode-ray oscillograph, type 304-A, manufactured by Allen B. DuMont Laboratories, Inc., Clifton, N. J.

† Manufactured by the Sanborn Company, Cambridge, Mass.
ventricular in origin (fig. 2). The threshold for effective cardiac stimulation ranged from 45 to 60 volts in the pig, 40 to 100 volts in the dog, and 20 to 100 volts (50 to 200 milliamperes) in man.

With rates of stimulation slower than the intrinsic cardiac rhythm, intermittent ectopic beats are produced whenever the stimulus falls in a responsive phase of the cardiac cycle. The external electric pacemaker competes with the intrinsic cardiac pacemaker and all the phenomena of parasystole may appear (fig. 3).

At rates faster than the intrinsic pacemaker, the electric stimuli produce a regular, externally paced rhythm and mask the intrinsic cardiac activity (fig. 4). At frequencies of stimulation between 500 and 600/min. in the normal dog, the heart no longer responds to every stimulus and the externally paced rhythm becomes irregular; cessation of such rapid stimulation is usually followed by return of the intrinsic normal sinus rhythm.

In the normal pig regular 1:1 responses to the electric stimuli were recorded electrocardiographically at rates as high as 1000/min. (fig. 5). Simultaneous aortic pressure tracings showed small mechanical pulses synchronous with each electric cardiac response up to frequencies of 500/min. (fig. 5B).

With stimulation at 1000/min. (fig. 5C), pulse waves were not discernible, presumably because of insignificant stroke volumes. This absence of pulse waves confirmed the effectiveness of all the stimuli by indicating the suppression of the slower intrinsic pacemaker. The 1:1 electrocardiographic responses to the rapid
stimuli were of low voltage; nevertheless the effective stimuli and their responses differed in configuration from the ineffective stimuli that were seen in the immediately subsequent period of 2:1 response.

The location of the electrodes, the timing of the electric stimulus in the cardiac cycle, and the intensity of stimulation determine whether the externally paced response is supraventricular or ventricular in origin. With high location of the electrodes in the esophagus or on the chest, stimulation produces supraventricular beats; with lower positions the beats are ventricular; at intermediate positions both types of responses may occur. Stimuli occurring early in the responsive phase of the cardiac cycle produce supraventricular beats and late ones produce ventricular beats (fig. 2). If the intensity of an early stimulus that produces a supraventricular beat is increased, however, a ventricular complex may occur.

Figure 6 shows the only instance of supraventricular response that has been observed in man. Complete heart block developed during
Fig. 6. Ventricular and supraventricular stimulation (man). A. Ventricular response, V, to every stimulus, E. B. As the stimuli, E, are increased to threshold intensity, the slow idioventricular rhythm is replaced by the faster externally paced supraventricular beats, A. Note the lengthening E-A interval. C. Thirteen seconds later a Wenckebach phenomenon has appeared: the E-A interval increases until a beat is dropped and then shortens.

The responses to stimulation in B and C are supraventricular in origin: (1) they differ in shape from the ventricular responses seen in A; (2) the E-A interval is longer than the E-V interval and is consistent with ativoventricular conduction time; and (3) Wenckebach-type block is a phenomenon of ativoventricular conduction.

Fig. 7. Bidirectional ventricular complexes produced by external electric stimulation (dog).

cardiac catheterization in an infant with severe cyanotic congenital heart disease and was followed by circulatory collapse. Circulation was maintained for 12 hours by external electric stimulation of the ventricles (fig. 6A). Terminally, however, atrial activity ceased and stimulation produced supraventricular beats with a Wenckebach atrioventricular block (fig. 6B, C).

The ventricular responses to repetitive stimuli may have different electrocardiographic configurations. Infrequent but striking examples are alternating mirror-image complexes (fig. 7), similar to the ominous bidirectional ventricular tachycardia occasionally seen in clinical digitalis intoxication.18

The external electric pacemaker generally produces an effective myocardial contraction as well as an electrocardiographic response. The blood pressures depend upon the state of the myocardium and the amount of filling of the ventricles during diastole and not upon whether the pacemaker is intrinsic or external, supraventricular or ventricular. At similar rates the blood pressures following externally paced beats are comparable with those from the intrinsic cardiac pacemaker (figs. 4 and 13C).

Recently Starzl, Gaertner, and Webb have concluded from experiments in dogs "that ventricular stimulation in the face of an intact conduction system is injurious."19 The one published electrocardiogram supporting this conclusion (their fig. 1c) shows irregular ventricular beats and arterial pulses during electric stimulation and is clearly just another example of competition between the sinoatrial and the electric pacemaker (our fig. 3). As these authors
themselves illustrate (their fig. 1d), however, stimulation at faster rates provides complete control of the ventricular rhythm and regular pulses. Their sweeping condemnation of ventricular stimulation in the presence of an intact conduction system is unwarranted, since it is based on the special circumstance of competition. During normal sinus rhythm in dogs, pigs, and man, external stimulation has taken over control of the heart and produced an effective circulation. Contrary to the suggested hazard of external cardiac stimulation in the presence of atrioventricular conduction,19 we have observed in dogs with normal sinus rhythm that effective circulation was maintained continuously without ill effect for many hours by the external electric pacemaker.

In the presence of myocardial depression from drugs, shock, anoxia, or myocardial ischemia, an effective myocardial contraction may not always follow every electrocardiographic ventricular response to external electric stimulation (fig. 8). This dissociation of electric and contractile activity is comparable to that seen in the dying heart when the intrinsic pacemaker produces action currents that do not evoke effective myocardial contractions.

No untoward cardiac effects of external electric stimulation were observed in hundreds of animal experiments and in over 70 patients. It did not produce multiple ectopic ventricular beats or ventricular fibrillation, although these disturbances are seen experimentally when electric stimulation is applied directly to the heart. Postmortem examinations in 12 patients and numerous animals showed no evidence of damage from the electric current to the heart or to neighboring structures. The only tissue damage from electric stimulation consisted of 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. The major untoward effects were chest pain and muscular twitch. The intensity of the pain and of the muscular contraction varied in different patients; in some it was negligible, in others it made continued stimulation difficult. Meperidine (Demerol) hydrochloride or paraldehyde usually made the discomfort tolerable and permitted continued stimulation. With prolonged stimulation the severity of the pain usually diminished markedly and less medication was required.

Electric stimulation has been clinically useful in cardiac arrest by producing an externally controlled ventricular rhythm in patients with Stokes-Adams disease, drug-induced standstill, reflex vagal standstill, and arrest occurring un-
expectedly during various procedures particularly under anesthesia.1-5

**Induction of Arrhythmias by Rapid Stimulation**

External stimulation of the heart at rapid rates of 500 to 1200/min. for periods of 10 to 60 sec. was applied to normal dogs, normal pigs, and pigs with previous ligation of the left circumflex coronary artery.

In the normal dogs, cessation of rapid stimulation was usually followed by return of normal sinus rhythm. Infrequently, however, atrial fibrillation followed (fig. 9). When rapid stimulation with consequent severe hypotension was prolonged (30 to 60 sec.), ventricular fibrillation often ensued, probably as a result of the marked cardiac ischemia. No other arrhythmias were observed in the dogs.

In 4 normal pigs, no arrhythmias were produced on repeated trials. In a fifth normal pig that was subjected to more prolonged stimulation with shorter rest intervals, 1 episode of ventricular tachycardia and many episodes of ventricular fibrillation were produced.

In 12 pigs, the terminal portion of the left circumflex coronary artery was ligated. Thereafter, atrial, nodal (fig. 10), and ventricular (fig. 11) tachycardia and atrial and ventricular fibrillation (fig. 13A) were readily produced by external stimulation. These arrhythmias were induced 88 times between the second and
EFFECTS OF EXTERNAL ELECTRIC CURRENTS ON HEART

FIG. 11. Induction and termination of ventricular tachycardia (pig).

### Table 1.—Induction and Termination of Cardiac Arrhythmias in Pigs with Coronary Artery Ligation

<table>
<thead>
<tr>
<th>Type of Arrhythmia</th>
<th>Induction Without stimulation</th>
<th>Induction With stimulation</th>
<th>Termination Spontaneous</th>
<th>Termination Countershock</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supraventricular tachycardia</td>
<td>1</td>
<td>6</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>0</td>
<td>13</td>
<td>8</td>
<td>5</td>
</tr>
<tr>
<td>Ventricular tachycardia</td>
<td>2</td>
<td>37</td>
<td>15</td>
<td>24</td>
</tr>
<tr>
<td>Ventricular fibrillation</td>
<td>1</td>
<td>32</td>
<td>1</td>
<td>32</td>
</tr>
</tbody>
</table>

* Severe anoxia, intracardiac epinephrine, or intravenous procaine amide was probably responsible for these 4 instances.

Figure 11 shows the induction and termination of ventricular tachycardia (pig) with an electric current of 240 V. The table (Table 1) illustrates the induction and termination of cardiac arrhythmias in pigs with coronary artery ligation.

### Termination of Arrhythmias by Countershock

We have stopped each kind of induced arrhythmia in the animals by the application of 60-cycle alternating current of 180 to 660 volts for 0.15 sec. across the closed chest.

Ventricular fibrillation was instantaneously terminated 69 times in dogs and 32 times in pigs. On numerous occasions defibrillation was unsuccessful because of inadequate countershock voltages, improper electrode placement, or poor condition of the animal from prolonged circulatory arrest or excessive countershocks. When the high-voltage countershocks were applied in rapid succession without adequate rest intervals, clear evidence of cardiac damage was manifested by abnormal QRS complexes, fall in arterial pressure, spontaneously recurrent fibrillation, and actual "cooking" of the heart muscle. Similar observations on external defibrillation have been made by others.11-15

Furthermore, we have been able to terminate experimentally induced atrial, nodal, and ventricular tachycardia and atrial fibrillation by externally applied countershock (Table 1). Although many of these arrhythmias stopped spontaneously, the efficacy of external countershock in terminating them was clearly established by the repeated observation of their instantaneous cessation upon application of the current (figs. 9-13). When countershocks of 180-300 volts were applied, termination of the arrhythmia usually occurred immediately.
arrhythmias was usually followed immediately by normal sinus rhythm. When higher voltages (300–660 volts) were used, atrial standstill, ventricular standstill, idioventricular beats, or atrioventricular block was often observed. These manifestations of interference with impulse formation and conduction were usually followed quickly by normal sinus rhythm.

In a few instances, ventricular standstill persisted following countershock, especially in hearts deprived by anoxia, prolonged circulatory collapse, or repeated high-voltage countershocks. Under such circumstances external electric stimulation often aroused effective ventricular responses and was followed by spontaneous ventricular activity. Figure 12 shows a sequence, repeatedly observed in 1 pig, of ventricular standstill after defibrillation, in which a single effective electric stimulus initiated spontaneous ventricular activity with subsequent resumption of normal sinus rhythm. Figure 13 illustrates prolonged ventricular standstill following countershock and final resuscitation by external electric stimulation and sympathomimetic drugs after 7 minutes of circulatory arrest.

External countershock defibrillation has also been accomplished in the human heart 12 times in 5 patients. In 2 patients ventricular fibrillation followed an acute myocardial infarction. After delays of at least 5 and 7 min. the ventricular fibrillation was terminated instantaneous by countershocks of 240 and 360 volts. Persistent ventricular standstill followed in both cases, however, and heart beats could not be stimulated by the external electric pacemaker.

The third patient had an irregular tachycardia and circulatory collapse for over 15 min., when ventricular fibrillation ensued. After 1 minute of ventricular fibrillation, external electric countershock was applied. Initial shocks of 240 and 360 volts were ineffective. On 6 occasions thereafter, countershocks of 480 to 720 volts defibrillated the ventricles (fig. 14). Each time, however, fibrillation recurred within 2 to 30 sec., following periods of cardiac standstill or ventricular beats. Adequate respiratory exchange was not effected, circulatory collapse persisted, and the patient died.

The fourth patient developed ventricular tachycardia and then ventricular fibrillation as a result of digitoxin overdosage. External countershock of 240 volts stopped the fibrillation. A ventricular rhythm ensued and persisted for 5½ min., but the patient remained apneic, unresponsive, and pulseless, and then died.

The fifth patient had active Stokes-Adams disease. He was resuscitated from ventricular fibrillation on 3 occasions by external countershocks of 270 to 360 volts. Tachycardia occurred twice after defibrillation; once it stopped spontaneously, and once it was terminated by countershock. Ventricular standstill occurred in each episode and external electric stimulation evoked effective ventricular beats, which were followed shortly by spontaneous ventricular rhythm and complete recovery.

These high, potentially dangerous countershock voltages have been applied without accident to the medical personnel. The procedure also appeared safe for the subject; no neurologic sequelae, or cardiac or local tissue
Fig. 13. Induction of ventricular fibrillation, defibrillation, and resuscitation from subsequent cardiac arrest (pig). A. Ventricular fibrillation was induced by rapid stimulation (600/min.), persisted for 2 minutes, was terminated by 240-volt countershock, and was followed by cardiac arrest. B. External electric stimulation applied during the period of arrest produced ventricular responses and increasing arterial pulse pressure. The dependence of these responses on the external cardiac pacemaker is shown by their disappearance with interruption of stimulation or with reduction of its voltage to ineffective levels. C. Seven minutes after the onset of ventricular fibrillation, during a test interruption of external stimulation, normal sinus rhythm returned. Early and later in the recovery period, the external pacemaker produced beats with pressures equal to those from the sinoatrial pacemaker.
damage was observed in numerous animals and in the 1 surviving patient.

The termination of atrial fibrillation and supraventricular and ventricular tachycardia by this technic has wide clinical potentialities. External countershock may prove useful in desperate cases resistant to drug therapy, by providing a means of immediate termination of the arrhythmia without the dangers of massive drug dosage.

The demonstrated efficacy of external countershock in stopping ventricular fibrillation in man provides a readily available and safe technic for stopping this usually fatal arrhythmia. Outside the operating room and in seriously ill patients in whom thoracotomy and cardiac massage are particularly inadvisable,22 external countershock may offer the only effective therapy. In the operating room, external electric stimulation and defibrillation comprise a combined technic whereby patients with unexpected cardiac arrest may be resuscitated from ventricular standstill or fibrillation before recourse to the more formidable and traumatic procedure of cardiac massage. Continuous cardiac monitoring* in the operating room would enhance the likelihood of successful external resuscitation by providing immediate recognition of the onset and mechanism of the arrest.

**Summary**

Electric stimuli applied externally across the unopened chest produced effective cardiac beats and controlled cardiac rhythm in dogs, pigs, and man. Supraventricular and ventricular tachycardia and fibrillation have been readily induced by very rapid external electric stimulation (over 500/min.) in pigs with previous coronary artery ligation. Countershock electric current applied externally has instantaneously terminated all these arrhythmias. Resuscitation of patients from ventricular standstill and ventricular fibrillation by these external technics has been accomplished.

**SUMMARIO IN INTERLINGUA**

Stimulos electric, applicate externemente a transverso le non-aperte thorace, produceva efficace pulsos cardiac e controlava le rhythmno cardiac in canes, porcos, e humanos. Tachycardia e fibrillation supraventricular e ventricular esseva promptemente inducite per le rapidissime electrostimulation externe (plus que 500 per minuta) in porcos con previemente effe-ctuate ligationes del arteria coronari. Le choc contrari effectuate per le application externe de currente electric terminava instantaneamente omne iste arrhythmias. Le resuscitation de patientes ab arresto ventricular e ab fibrillation ventriculares esseva effectuate per medio de iste technicas externe.

**REFERENCES**


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22 Harken, D. E.: Personal communication.


The principal innovation of therapy discussed is Wallgren's successful use of beer as a diuretic in heart failure. The author states "I can think of many patients who will welcome this news."

McKusick
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Circulation. 1956;14:745-756
doi: 10.1161/01.CIR.14.5.745

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1956 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/14/5/745

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