Clinical Science

New Method for Terminating Cardiac Arrhythmias

Use of Synchronized Capacitor Discharge

Bernard Lown, M.D., Raghavan Amarasingham, M.B., B.S., and Jose Neuman, M.D., Boston

THE ECTOPIC TACHYCARDIAS are currently treated by either vagal stimulation or drugs. The 3 most effective drugs are quinidine, procainamide hydrochloride, and the digitalis glycosides. When the ectopic mechanism drives the ventricles at rates above 160 per minute, cardiac output falls and coronary blood flow is compromised. This is most likely to occur with ventricular tachycardia which constitutes a serious cardiac emergency requiring immediate treatment. Frequently, however, the arrhythmia cannot be terminated promptly. Reversion with drugs generally involves a time-consuming biologic titration. Since it is impossible in any one patient to predict either the effective or the toxic dose, small increments of antiarrhythmic drugs are given at frequent intervals until a therapeutic end point is reached. The interval between doses is determined by the gravity of the patient's illness as well as by the rapidity of action of the particular agent. It may thus take minutes, days, or even weeks to terminate an ectopic rhythm.

The use of antiarrhythmic drugs may be associated with additional problems. Quinidine and procainamide hydrochloride lower peripheral resistance, depress cardiac contractility and excitability, prolong atrioventricular and intraventricular conduction, and induce a host of other untoward reactions. The digitalis glycosides also may provoke serious toxic reactions. At times, an ectopic arrhythmia is unresponsive to all drugs, even when these are used in combination and given in massive doses. When the refractory arrhythmia is of atrial origin, slowing of the ventricular rate can still be achieved by means of digitalization. This is not the case when the refractory mechanism is of ventricular origin. Survival is then threatened.

From the Department of Nutrition, Harvard School of Public Health, and the Medical Clinics, Peter Bent Brigham Hospital.

Presented in part before the 54th Annual Meeting of the Society for Clinical Investigation, April 30, 1962.

The requirements for an ideal method for abolishing atrial and ventricular arrhythmias can be readily spelled out. The ectopic mechanism should be controlled instantly and consistently. There should be no depression of the normal cardiac pacemakers, no prolongation of conductivity, nor impairment of myocardial contractility. Other vital structures should not be injured. Furthermore, the method should be simple in application.

The purpose of this report is to introduce a method for terminating certain cardiac arrhythmias which fulfills these requirements. The experimental studies which led to this new method and preliminary clinical results will be presented.

Experimental Studies

Alternating Current Countershock (AC).—Theoretically, electrical countershock of the heart would be an ideal form of antiarrhythmic therapy, if it were safe. By simultaneous depolarization of all cardiac fibers the ectopic focus or foci could be extinguished, permitting the sinus node to resume as pacemaker. AC countershock has been widely used and is regarded as the method of choice for defibrillating the heart. Alexander and co-workers [1] successfully employed AC countershock across the intact chest for treating paroxysmal ventricular tachycardia. Subsequently, Zoll and Linenthal [2] and Paul and Miller [3] reported on the use of AC countershock for controlling not only ventricular but also certain atrial arrhythmias.

The elective clinical use of AC for treating arrhythmias must be weighed against the potential hazard to the heart of the countershock itself. The effects of AC upon the normal cardiovascular apparatus were therefore evaluated in animals with sinus rhythm.[4] It was found that numerous and serious arrhythmias accompanied the use of AC. Ventricular fibrillation occurred once in every 5

shocks. Atrial fibrillation followed 70% of test shocks. Repeated AC countershocks administered to normal dogs resulted in sequential electrocardiographic changes of acute anterolateral myocardial infarction in 90% of animals. During a week of follow-up study there was a 35% mortality.[4] It was concluded that the use of AC countershock across the intact chest involves a substantial hazard to the heart.

Clinical data on the use of AC in adults with arrhythmias other than ventricular fibrillation tend to confirm some of these experimental observations. Data are available on 12 patients. The response of 8 of these was reported by Zoll and Linenthal.[2] Four patients were studied by one of the authors. The abnormal mechanisms consisted of ventricular tachycardia in 8, arrhythmias of either supraventricular or ventricular origin in 2, of nodal tachycardia in 1, and of atrial fibrillation in 1. In 4 patients, ventricular fibrillation followed the use of AC countershock; additional shocks had to be given to restore a normal mechanism. One of these patients died as a result of cardiac standstill induced by countershock. The use of AC provoked other arrhythmias as well, including A-V nodal rhythm, ventricular standstill, atrial flutter, and showers of atrial and ventricular premature beats, some of long duration. Six of the 12 patients required multiple shocks, and one each required 4, 5, and 6 successive discharges for terminating the rhythm disorder.

These experimental and clinical findings indicate that AC countershock is not a safe method for treating cardiac arrhythmias.

Direct Current Countershock (DC).—A new electronic device was thereupon developed, employing direct current discharge. After extensive preliminary tests, a model was adopted which utilized a 16-microfarad capacitor in series with an inductor and delivered an underdamped discharge of 2.5 milliseconds duration. It was calibrated in energy units, or watt-seconds.[4] In order to establish whether or not this type of capacitor discharge would depolarize the entire heart, it was tested as a cardiac defibrillator. Transthoracic DC countershock was found to be 100% effective in controlling 550 episodes of ventricular fibrillation in 20 dogs. All animals survived. Furthermore, DC countershock controlled ventricular fibrillation in both animals and men when AC failed to restore a normal mechanism.[4,5]

As in the study of AC, the effects of DC shock upon the normal heart were investigated. Arrhythmias were noted infrequently after the use of DC in animals with normal sinus rhythm. Atrial fibrillation was never observed. Ventricular fibrillation occurred after 1.6% of the countershocks. Sequential electrocardiographic changes compatible with myocardial infarction were noted in one-third of the animals. None of the dogs died.

These experiments demonstrate that DC countershock consistently and uniformly depolarizes the heart. The findings indicate that DC is safer than AC countershock.

The Vulnerable Period.—The occurrence in normal animals of ventricular fibrillation after nearly 2% of random DC shocks precluded the elective clinical use of this particular method. Physiologists have long known that the ventricular cycle is not uniformly susceptible to fibrillation. A vulnerable period has been identified in late systole, coinciding in time with the T wave of the conventional electrocardiogram.[6-8]

Ventricular fibrillation occurring after DC discharge seemed to be a random phenomenon. It was possible that the arrhythmia was caused by a shock sporadically striking the vulnerable period of the cardiac cycle. An electronic synchronizer was therefore developed which permitted delivery of DC shocks through a time-delay circuit triggered by the R wave of the electrocardiogram.[9] This synchronized direct current could be discharged at any preselected point in the cardiac cycle.

A systematic exploration of the cardiac cycle at 10-millisecond intervals was then carried out with test shocks delivered across the closed chest. Analysis of 3,500 such synchronized shocks indicated that discharges outside the T wave did not result in ventricular fibrillation. When the shock was delivered during the T wave, the incidence of ventricular fibrillation rose to 35%. A systematic exploration of the T wave interval established that ventricular fibrillation could be induced only during a limited period of 30 milliseconds just preceding the apex of this wave. An appropriate test shock placed within this vulnerable period consistently produced ventricular fibrillation. Similar stimuli applied at any other instant of the cardiac cycle did not induce fibrillation. A period of vulnerability was also demonstrated for the atrium. It was located during inscription of the downslope of the R wave or the S wave. Discharge during this phase of the cardiac cycle resulted in atrial fibrillation.

These studies demonstrate that a properly synchronized, direct current discharge can depolarize the heart with complete avoidance of either ventricular or atrial fibrillation.

Clinical Experience

Ventricular Tachycardia.—Synchronized cardiac depolarization, employing a DC countershock device, was initially limited to patients with drug-refractory ventricular tachycardia. To date, this method has been employed in 5 patients having 9 episodes of this arrhythmia. Each patient had received large and even toxic doses of either procainamide hydrochloride, quinidine, or both, without avail. In 2 patients, Nos. 4 and 5 (Table 1), with recurrent episodes of ventricular tachycardia

Table 1.—Ventricular Tachycardia Treated with Synchronized Cardiac Depolarization

Patient	Age, Yrs.	Episodes, No.	Underlying Heart Disease	Clinical Condition	Duration Vent. Tachy.	Drugs Used	Shocks, No.	Energy Level (Watt-Seconds)	Results	Complications
1.	63	1	Acute M.I.	Shock, Pulmonary edema	4 days	Quinidine, Procainamide Hcl, Potassium	1	100	NSR	None
2.	85	1	Acute M.I.	Shock, Pulmonary edema	11 days+	Quinidine, Procainamide Hcl, Potassium, Digitalis	1	100	NSR	None
3.	31	1	Old M.I.	Good	15 days	Quinidine, Procainamide Hcl	1	100	NSR	None
4.	62	2	Old M.I.	Shock	2 hr.	Quinidine, Procainamide Hcl, Potassium	1	100	NSR	None*
				Shock	5 hr.		1	100	NSR	None
5.	62	4	Old M.I.	Shock	1-5 hr.	Procainamide Hcl, Quinidine	1	100	NSR	None
				Good			1	100	NSR	None
				Good			1	100	NSR	None
				Good			1	50	NSR	None

* Ventricular premature beats occurred after reversion but disappeared on discontinuing l-norepinephrine infusion.
M.I., myocardial infarction.
NSR, normal sinus rhythm.

previously unresponsive to antiarrhythmic agents, a test dose of 1 gm. of procainamide hydrochloride was administered by intravenous injection during a period of one hour before DC countershock was attempted.

An identical procedure was employed in each patient. Reversion was performed in the operating recovery room. No premedication was given. The patient was anesthetized with intravenous thiopental sodium. The synchronizer was preset to deliver the direct current discharge at 20 milliseconds after the peak of the R wave. This time was well before the presumed ventricular vulnerable period. Heart action was continuously monitored on an oscilloscope screen. As soon as unconsciousness was induced, 2 electrodes covered with a thick layer of conductive jelly were closely applied to the chest wall with pressure. One electrode was positioned in the left midaxillary line at the level of the fifth interspace and the other immediately to the right of the sternum at about the second intercostal space. The 2 paddles were held by the operator who released the discharge by a foot switch. A single twitch of skeletal muscles and a slight movement of the arms indicated the passage of electrical current. The patient was generally unconscious for 2 to 5 minutes. Oral antiarrhythmic maintenance therapy was started shortly after the cardiac depolarization.

Results

All 9 episodes of ventricular tachycardia were successfully reverted with a single synchronized DC discharge. The exact mechanism of reversion was obscured by the massive electrical field developed by the countershock. However, a normal sinus mechanism was observed in each patient within 2 to 3 seconds (Fig. 1). Cardiac arrhythmia was noted after only one of the reversions. This occurred in patient No. 4 (Table 1) who had showers of multiform ventricular ectopic beats accompanied by brief paroxysms of ventricular tachycardia. In this patient, during the ventricular tachycardia, the blood pressure could not be raised above 80/70 mm. Hg, even with large doses of levarterenol bitartrate. One minute after restoration of sinus rhythm the blood pressure was recorded as 170/110 mm. Hg. Stopping the catechol amine infusion caused the pressure to recede to normotensive levels and the arrhythmia disappeared. It is likely that the enhanced ventricular excitability was provoked by levarterenol bitartrate administration rather than by the electric shock.

No other untoward effects were noted. No patient showed any cutaneous burns at the site of electrode application. Objective manifestations of heart failure receded within several minutes. Most striking was the abrupt transformation in the pa-

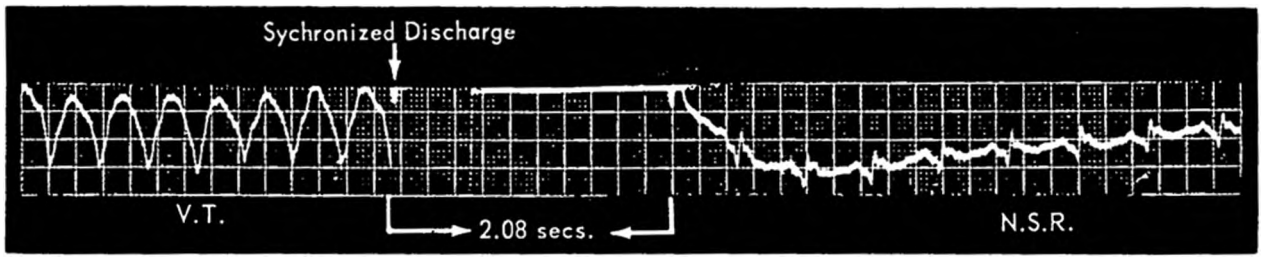

Fig. 1.—Patient No. 5 (Table 1) with ventricular tachycardia. Single capacitor discharge of 100-watt-seconds programmed to fall at nadir of S waves restores sinus rhythm. First normal complex appears in slightly over 2 seconds after shock.

tient's demeanor from morbid anxiety to that of euphoric exhilaration. The following report of a case is illustrative of this experience.

Report of a Case

A 63-year-old woman experienced an abrupt onset of severe crushing chest pain. She continued to do housework for about 2 weeks but noted increased breathlessness. Four days before she was hospitalized, rapid heart action was detected. Treatment consisted of small doses of digitalis and injections of mercurials. When admitted to the Peter Bent Brigham Hospital she was in acute respiratory distress and was breathing at a rate of 35 per minute. Neck veins were engorged with clearly visible atrial waves, in the jugular pulse occurring at a rate slower than in the apical pulse. Rales were present in both lung fields and extended up to the apices. The heart rate was 200 beats per minute and regular. Multiple sounds were audible. M_1 was widely split; P_2 was diminished and split. No murmurs were heard. Blood pressure was 110/90 mm. Hg. The abdomen was tense and distended. There was moderate peripheral edema. The electrocardiogram showed a bizarre QRS pattern with atrioventricular dissociation.

The diagnosis was ventricular tachycardia occurring in the wake of an acute myocardial infarction. Treatment was started with procainamide hydrochloride. After 750 mg. intravenously the ventricular rate slowed to 100 but reversion did not occur. This slowing was accompanied by profound hypotension. Forty milliequivalents of potassium chloride was then administered without effect. Finally, intravenous quinidine was given. After 800 mg., marked intraventricular block developed (Fig. 2). Blood pressure was difficult to maintain even with pressor amines. It was then decided to attempt countershock reversion.

The patient was taken to the recovery room and anesthesized with thiopental sodium. A single 100-watt-second synchronized discharge resulted in immediate restoration of sinus rhythm. Within 2 minutes the advanced degree of intraventricular block had completely disappeared. First degree heart block continued for several hours (Fig. 3). Blood pressure was immediately restored to normotensive levels. The pulmonary congestion receded rapidly and disappeared entirely within several minutes. The patient was awake within 2 minutes and was quite startled by the newly found sense of well-being.

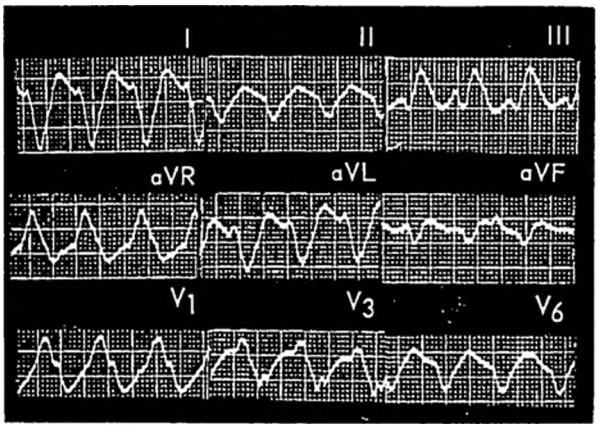

Fig. 2.—Patient No. 1 (Table 1) with ventricular tachycardia of 4 days' duration shows marked intraventricular block after treatment with antiarrhythmic drugs.

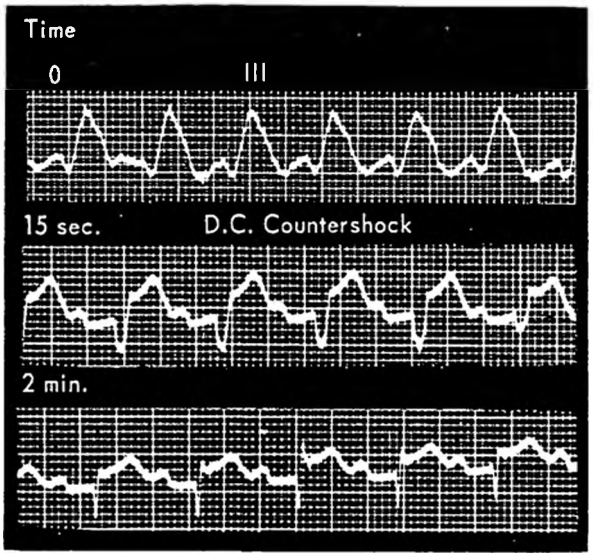

Fig. 3.—Same patient as in Figure 2. After single synchronized discharge, normal sinus rhythm is restored. Transient intraventricular block (middle strip) disappeared within 2 minutes.

Comment.—Ventricular tachycardia is a serious disorder which, if it persists, leads to death. It develops generally in patients with serious cardiac disease. The administration of antiarrhythmic agents may further compromise cardiac pumping action. This was clearly illustrated in the foregoing patient. Administration of moderate doses of both procainamide hydrochloride and quinidine resulted in deterioration of cardiac function, development of a marked degree of intraventricular block, and a fall in systemic pressure refractory to pressor agents. Administration of the various antiarrhythmic drugs required 5 hours. By contrast, use of synchronized countershock required but a few minutes of preparation, reversion was instantaneous, and there were no evident side effects. The results were identical in all 9 reversions.

Atrial Fibrillation.—The absence of complications after synchronized cardiac depolarization in patients with ventricular tachycardia prompted application of this method to less serious disorders of rhythm. Patients with chronic atrial fibrillation were then selected for treatment. In order to minimize possible untoward effects, reversion attempts were initially limited to patients undergoing mitral valve operation. Cardiac depolarization was carried out only if the left atrium was free of thrombi. Once valvuloplasty was accomplished, the chest retractors were removed, the chest wall was approximated, and the electrode paddles were applied directly to the intercostal muscles. No conducting paste was employed. After countershock, the chest was reopened, the atrial suture line was examined, and operation was completed. In patient No. 3 (Table 1), one of the electrodes was applied directly to the heart while the other electrode was placed on the lung.

Absence of undesirable side effects encouraged extension of this investigation to patients in whom

Table 2.—Atrial Fibrillation Treated with Synchronized Cardiac Depolarization

Patient	Mitral Operation	Duration Atrial Fibrillation	Shocks, No.	Energy Levels (Watt-Seconds)	Postreversion Arrhythmia	Reversion
1.	+	2 yr.	2	50, 100	0	Yes
2.	+	3 mo.	2	100, 30*	0	Yes
3.	+	5 yr.	1	50	0	Yes
4.	+	2 mo.	1	200	Nodal rhythm, 30 sec.	Yes
5.	+	1-2 yr.	4	50, 100, 200, 300	0	No
6.	+	5 yr.	3	100, 200 400	Ventricular bigeminy, 1 min.	Yes
7.	+	3 mo.	2	100, 200	0	Yes
8.	+	4 yr.	3	100, 200, 300	Sinus tachycardia	Yes
9.	+	5 yr.	2	100, 200	0	Yes
10.	+	5 mo.	4	100, 200, 250, 400	0	No
11.	0	1 mo.	1	100	Nodal rhythm, 30 sec.	Yes
12.†	0	1 mo.	1	100	0	Yes
13.	0	1 yr.	1	100	Rare VPB	Yes

*One electrode on heart, the second on lung.
†Same patient as No. 11. VPB, ventricular premature beats.

reversion to normal sinus rhythm was indicated on medical grounds but could not be accomplished with quinidine. To date, 2 patients with 3 episodes of atrial fibrillation have been treated. The procedure was identical to that employed for patients with ventricular tachycardia. Since this was an elective undertaking, measures were taken to prevent postanesthetic complications. Food was withheld for 6 hours. One hour prior to the attempted reversion, a small dose of atropine was given subcutaneously.

Results.—Ten patients with atrial fibrillation who were undergoing mitral valve operation were treated with synchronized DC discharge. In 6 the arrhythmia was of long duration; in 3 of the 6 it had existed for about 5 years. Eight of the 10 patients were restored to sinus rhythm. Two patients remained in atrial fibrillation despite 4 synchronized shocks (Table 2). As in patients with ventricular tachycardia, reversion was nearly instantaneous. P waves were identifiable within 2 to 3 seconds. Immediately after the DC shock, arrhythmias occurred in 3 patients. In one, it was sinus tachycardia, the heart rate eventually being controlled with additional digitalis. In another, nodal rhythm developed which lasted 30 seconds, and in the third patient there was an episode of ventricular bigeminy lasting one minute. No other side effects were observed.

Two additional patients with atrial fibrillation refractory to large doses of quinidine were treated with synchronized cardiac depolarization. Both had mitral insufficiency. One patient had a recurrence of atrial fibrillation after one month and was again treated with cardiac depolarization. A single synchronized discharge across the intact chest reverted each of these 3 episodes to normal sinus rhythm. The electrode application was the same as that used in patients with ventricular tachycardia. The discharge was delivered within the terminal portion of the QRS complex. In one patient (No. 11) transient nodal rhythm developed prior to resumption of the sinus mechanism. The second patient had rare ventricular ectopic beats.

The following patient illustrates the experience with atrial fibrillation.

Report of a Case

A 54-year-old woman had been found to have a heart murmur during a second pregnancy. There was no history of acute rheumatic fever. Atrial fibrillation occurred 5 years before her admission to the hospital. She was free of any symptoms of cardiac decompensation. A year before hospitalization she experienced 2 successive embolic episodes, one to the lungs, the other to the lower right extremity. She was therefore treated with anticoagulants. Physical and

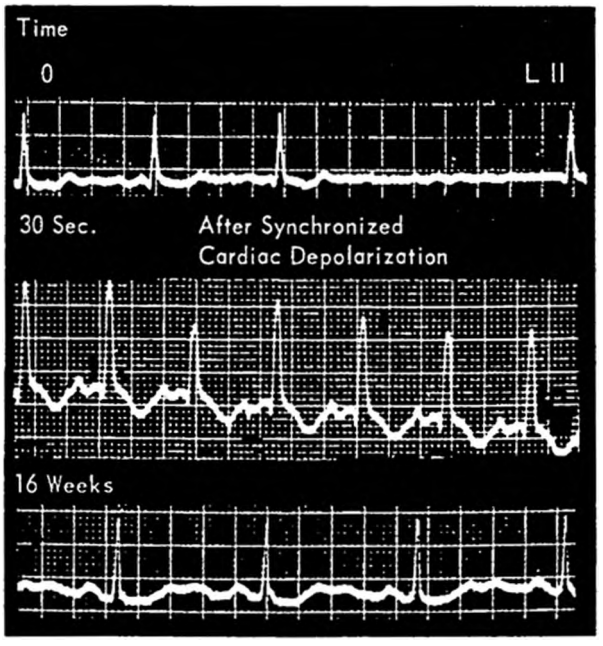

Fig. 4.—Atrial fibrillation of 5 years' duration. Patient No. 3 (Table 2) reverted with single 50-watt-second discharge. Intraventricular block and biphasic P waves (middle strip) disappeared within 1 minute. Normal sinus rhythm has persisted.

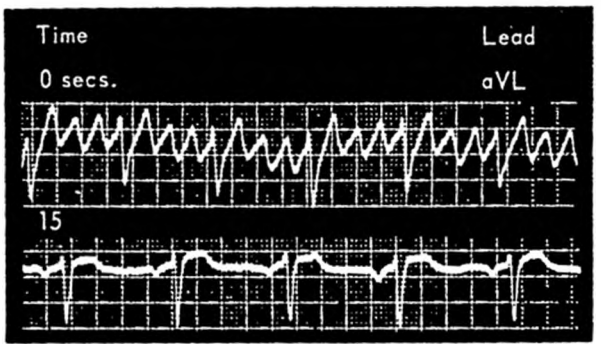

Fig. 5.—Chronic atrial flutter, not reverted with large doses of quinidine, responded to single synchronized discharge.

Table 3.—Over-all Summary of Treatment of Arrhythmias with Synchronized Cardiac Depolarization

Arrhythmia	Patients, No.	Episodes, No.	Reversions to Normal Sinus Rhythm	Shocks, No.	Complications
Ventricular Tachycardia	5	9	9	9	0
Atrial Fibrillation During Mitral Operation	10	10	8	24	0
Elective	2	3	3	3	0
Atrial Flutter	1	1	1	1	0
Supraventricular Tachycardia	1	2	2	2	0
Total	19	25	23	39	0

laboratory findings were consistent with tight mitral stenosis. At operation the mitral valve area was estimated to be 0.6 square centimeters. Immediately after fracture of the mitral valve, the chest wall was approximated and the 2 electrode paddles were placed subcutaneously at the extremes of the incision directly on the intercostal musculature. One was positioned posteriorly at the left infrascapular area and the other one was placed at the left sternal border at about the fourth intercostal space. A single 50-watt synchronized discharge resulted in the nearly instantaneous resumption of sinus rhythm (Fig. 4). She was promptly started on maintenance quinidine therapy. Now, 9 months after reversion, she remains in sinus rhythm.

Comment.—This patient had chronic atrial fibrillation for many years. In view of the long duration of arrhythmia, it is unlikely that restoration of a normal mechanism with quinidine would have been attempted. With cardiac depolarization, sinus rhythm was readily restored after a single discharge. This patient differed from the other patients treated during mitral operation, all but one of whom required more than one shock.

The patients treated during valvular operation required not only more shocks but also shocks at higher energy levels. Thus, 24 shocks were employed in these 10 patients. A single 100-watt-second discharge was, however, uniformly successful in the 15 episodes of arrhythmia treated outside the operating room. This discrepancy between those treated in and out of the operating room is probably the result of a difference in electrode placement. The application of the electrode paddles directly on the intercostal muscle and the presence of blood in the operative field may have served to short circuit the current and divert the energy from the heart. This factor may also account for the 2 failures even after shocks of 300- and 400-watt-seconds.

Other Atrial Arrhythmias.—The depolarizer was also used in treating one episode of chronic atrial flutter and 2 episodes of double tachycardia. The patient with flutter was a 21-year-old man who one year earlier had had an atrial septal defect corrected. At operation he had normal sinus rhythm; one year later the rhythm was atrial flutter. Quinidine in increasing amounts, up to 1.5 gm. in a single dose, did not restore a normal mechanism but induced nausea, vomiting, dizziness, headache, and a slowing of the atrial flutter rate from 280 to 200 per minute. A single 100-watt-second synchronized discharge resulted in sinus rhythm (Fig. 5).

A second patient who experienced many episodes of repetitive paroxysmal tachycardia of atrial, nodal, and ventricular origin was seen because of a recurrence of arrhythmia. The mechanism was a double tachycardia with one focus in the atrium and the second in the AV node. He was already receiving large doses of antiarrhythmic medication. A single 100-watt-second shock restored normal sinus rhythm. Fifteen minutes later, however, the arrhythmia recurred. He was therefore digitalized and treated once more with countershock. A single discharge again re-established sinus rhythm which then persisted.

In summary, 19 patients with 25 episodes of arrhythmias of ventricular and atrial origin were treated with synchronized cardiac depolarization (Table 3). Thirty-nine shocks were employed. Reversion to normal sinus mechanism was achieved in 23 of the 25 patients. In none of the patients were there any immediate complications or delayed untoward reactions.

Comment

The method described for treating certain arrhythmias is based on 2 suppositions: first, that the factors which initiate a ventricular or atrial tachycardia are different from those which sustain it, and second, and a corollary to the first, that once an ectopic site is momentarily extinguished, the sinus node will take over as the cardiac pacemaker. Paroxysmal tachycardia, including atrial flutter and fibrillation, frequently result from an interaction of factors, many of which are transient. When the arrhythmia is established, the mechanism becomes self-sustaining. This may result from the presence of a re-entry pathway which the ectopic wave front of depolarization can traverse indefinitely. In theory, when the heart is completely depolarized, this pathway is abolished and with it the arrhythmia. Depolarization of the entire heart can be accomplished readily with electric countershock administered across the intact chest. A major

deterrent to its clinical use has been the hazard of provoking ventricular fibrillation. Synchronized DC shock obviates this danger both by using capacitor discharge as the source of electrical energy and by releasing the shock outside the vulnerable period of the cardiac cycle.

Prevost and Batelli [10] who, in 1899, introduced alternating current countershock were also the first to employ direct current for defibrillating the heart. This latter form of countershock never gained wide acceptance, partly because of the decisive experiments of Kouwenhoven and co-workers [11] who clearly demonstrated that alternating current countershock is highly effective in defibrillating the heart. In part, this lack of acceptance was also a result of the very nature of capacitor discharge, for, by merely changing the parameters of the discharge circuit, a capacitor can yield an infinite variety of wave forms. At the present time there is no physiologic basis for predicting a wave form optimal for defibrillation. Development of an effective instrument, therefore, becomes a tedious trial-and-error procedure in which values for capacitance and inductance are randomly changed in relation to one another. It is not surprising that the use of instruments with different electrical properties have yielded contradictory results. While Gurvitch and Yuniev in the Soviet Union [12,13] and Peleška [14] in Czechoslovakia found capacitor discharge effective in defibrillating the heart, Guyton and Satterfield [15] as well as Kouwenhoven and Milnor [16] noted poor and inconsistent results.

As long as defibrillation was but rarely attempted, the problem of finding an optimal form of electric countershock did not press for solution. The remarkable demonstration by Kouwenhoven and co-workers [17] that blood flow to vital organs can be maintained in the arrested heart by repetitive compression of the lower portion of the sternum has reopened this question. In recent experiments,[4] capacitor discharge and alternating current countershock were compared in animals with normal sinus rhythm. It was found that capacitor discharge was a more effective and safer method than the latter. As compared with alternating current, it was associated with fewer arrhythmias, less evidence of myocardial injury, and a lower mortality. Furthermore, the brief pulse duration of the capacitor discharge permits release of the shock at any predetermined interval of the cardiac cycle. The vulnerable periods of the atria and ventricle can therefore be avoided, thereby preventing production of either ventricular or atrial fibrillation. For the first time, a relatively safe method has been developed for treating certain ectopic tachycardias.

Synchronized cardiac depolarization is a nearly ideal method for terminating selected cardiac arrhythmias. It is simple to employ, results are immediate, there is no evident depression of cardiac rhythmicity or contractility, and there are no side effects. One limitation is the need for anesthesia. Whether this can be obviated entirely or substituted by the use of sedatives or narcotic drugs is at present uncertain. Even if the need for anesthesia remains a permanent feature of this method, the remarkable clinical results thus far achieved indicate that synchronized countershock will find application in the treatment of intractable arrhythmias.

When is synchronized cardiac depolarization to be used? It should be employed in the treatment of tachycardias of atrial, nodal, or ventricular origin that have not responded to antiarrhythmic drugs. In the case of ventricular tachycardia, at present we initiate treatment with intravenous procainamide hydrochloride. If this drug induces a significant drop in blood pressure or if it fails to restore a normal rhythm after a total dose of 1 gm., further use of the drug is withheld and synchronized depolarization is utilized.

Results to date seem encouraging. The full scope of application of this method will be determined by more extensive clinical studies. A note of caution is in order; experience of physicians with other so-called wonder methods of treatment has amply demonstrated a significant gap between early promise and ultimate fulfillment. No method of treatment should gain unquestioned acceptance until the "late returns" are in!

Summary

A new method for treating ectopic tachycardias has been developed. The heart is depolarized instantly within a safe part of the cardiac cycle, thereby extinguishing the ectopic site and permitting resumption of sinus rhythm without danger of ventricular fibrillation.

The instrument on which this method is based consists of 2 electronic components, a capacitor-discharge unit, which delivers a brief single pulse with enough energy to depolarize the heart within the intact chest, and a time-delay circuit triggered by the voltage of the R wave of the surface electrocardiogram, which permits delivery of the discharge at any predetermined point of the cardiac cycle.

The instrument has been employed to treat 25 episodes of arrhythmia in 19 patients. The arrhythmias consisted of ventricular tachycardia in 9, chronic atrial fibrillation in 13, chronic atrial flutter in one, and double tachycardia in 2.

Twenty-three of the 25 episodes of arrhythmia were successfully reverted. The 2 failures were in patients with chronic atrial fibrillation, in whom reversion was attempted immediately after completion of mitral valvular operation. There were no immediate or late complications.

The application of this new method of synchronized cardiac depolarization for treating cardiac rhythm disorders is discussed.

Since submission of this article for publication, 10 additional patients with various arrhythmias have been successfully treated.

1 Shattuck St., Boston 15 (Dr. Lown).

The authors are indebted to Dr. Dwight E. Harken who provided the bulk of the patients who had atrial fibrillation. We are also grateful to Drs. S. A. Levine, H. D. Black, W. Taylor, and H. D. Levine for making their patients available for this study.

This study was supported in part by grants in aid from the John A. Hartford Memorial Fund, the National Heart Institute, the Nutrition Foundation, Inc., and the Fund for Research and Teaching, Department of Nutrition.

Generic and Trade Names of Drugs

Procainamide hydrochloride—*Pronestyl Hydrochloride.*

Levarterenol bitartrate—*Levophed Bitartrate.*

References

1. Alexander, S.; Kleiger, R.; and Lown, B.: Use of External Electric Countershock in Treatment of Ventricular Tachycardia, *JAMA* 177:916-918 (Sept. 30) 1961.

2. Zoll, P. M., and Linenthal, A. J.: Termination of Refractory Tachycardia by External Countershock, *Circulation* 25:596-603 (April) 1962.

3. Paul, M. H., and Miller, R. A.: External Electrical Termination of Supraventricular Arrhythmias in Congenital Heart Disease, *Circulation* 25:604-609 (April) 1962.

4. Lown, B., et al.: Comparison of Alternating Current with Direct Current Electroshock Across Closed Chest, unpublished data.

5. Lefemine, A. A., et al.: Defibrillation by Direct and Alternating Currents at Normal and Reduced Temperature, unpublished data.

6. King, B. G.: *Effect of Electric Shock on Heart Action with Special Reference to Varying Susceptibility in Different Parts of Cardiac Cycle,* Columbia University, Doctoral Thesis, New York City: Aberdeen Press (May) 1934.

7. Ferris, L. P., et al.: Effect of Electric Shock on Heart, *Electrical Engineering* 55:498-515 (May) 1936.

8. Wiggers, C. J., and Wegria, R.: Ventricular Fibrillation Due to Single, Localized Induction and Condenser Shocks Applied During Vulnerable Phase of Ventricular Systole, *Amer J Physiol* 128:500-505 (Oct. 28) 1939.

9. Lown, B., et al.: Use of Synchronized Direct Current Countershock in Treatment of Cardiac Arrhythmias, presented before 54th Annual Meeting, American Society for Clinical Investigation, April 30, 1962.

10. Prevost, J. L., and Battelli, F.: La Mort par les Courants Electriques-Courants Alternitifs à Haute Tension, *J Physiol Path Gen* 1:427-430, 1899.

11. Kouwenhoven, W. B.; Hooker, D. R.; and Langworthy, O. R.: Current Flowing Through Heart Under Conditions of Electric Shock, *Amer J Physiol* 100:344-350 (Jan. 12) 1932.

12. Gurvich, N. L., and Yuniev, G. S.: Restoration of Regular Rhythm in Mammalian Fibrillating Heart, *Am Rev Soviet Med* 3:236-239 (Feb.) 1946.

13. Gurvich, N. L., and Yuniev, G. S.: Restoration of Heart Rhythm During Fibrillation by Condenser Discharge, *Am Rev Soviet Med* 4:252-256 (Feb.) 1947.

14. Peleška, B.: Transthoracic and Direct Defibrillation, *Rozhl Chir* 26:731-753 (Nov.) 1957.

15. Guyton, A. C., and Satterfield, J.: Factors Concerned in Electrical Defibrillation of Heart Particularly Through Unopened Chest, *Amer J Physiol* 167:81-87 (Oct.) 1951.

16. Kouwenhoven, W. B., and Milnor, W. R.: Treatment of Ventricular Fibrillation Using Capacitor Discharge, *J Appl Physiol* 7:253-257 (Nov.) 1954.

17. Kouwenhoven, W. B.; Jude, J. R.; and Knickerbocker, G. G.: Closed-Chest Cardiac Massage, *JAMA* 173:1064-1067 (July 9) 1960.

GALLOP RHYTHM AND ANAPEST RHYTHM.—The gallop stroke is diastolic and is due to the beginning of sudden tension in the ventricular wall as a result of the blood flow into the cavity. It is more pronounced if the wall is not distensible and the failure of distensibility may depend either on a sclerotic thickening of the heart wall (hypertrophy due to Bright's disease) or to decrease in muscular tonicity. Since the wall, by virtue of its own elasticity, is no longer able to resist the inflow of blood, it is placed under tension precisely at the same moment that this occurs.

The gallop can originate in all cases where the elastic resistance of the wall encroaches on muscular tonicity, either by an increase of the first factor, or a diminution of the second.

It was observed in a goodly number of acute diseases, chiefly in typhoid fever, and also in cachectic subjects, in whom cardiac function is embarrassed. It occurs either in a constant or more pronounced manner in ventricular hypertrophy of Bright's disease, or on the other hand, accompanies dilatation of the right heart of hepatogastro-intestinal origin. It is therefore an important sign owing to the latent or insidious character of the latter diseases.

The name, gallop rhythm, was first introduced by Bouillaud and should be used for the phenomenon to which it applies; the phenomenon does not, however, always maintain the character of the gallop of a horse.

One could apply the name of murmur of diastolic shock in all the cases in which the above mentioned theory is applicable and reserve the term, gallop rhythm, specifically for the type, in which the *anapest* rhythm occurs.—Potain, M.: Theory of Gallop Rhythm, *Classic of Cardiology,* New York City: Henry Schuman, Inc., Dover Publications, Inc., 1941.