

Synchronized DC Precordial Shock for Arrhythmias

Safe New Technique To Establish Normal Rhythm May
Be Utilized on an Elective or an Emergency Basis

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Synchronized precordial direct current shock has been applied in the treatment of sustained arrhythmia 84 times in 62 patients. Sinus rhythm was restored in 90% of the patients in 85% of the attempts. Single and multiple shocks were tolerated without significant electrocardiographic abnormality, aside from change in rhythm, or transaminase rise. No pulmonary or systemic emboli occurred during 71 reversions to sinus rhythm although anticoagulants had been withheld in more than half the patients. Complications were limited to the development of other arrhythmias in three patients. Synchronized direct current precordial shock is a safe, simple, and effective therapy of ectopic arrhythmia which may be utilized on an elective or emergency basis. The results are immediately apparent.

ESTABLISHING NORMAL RHYTHM in the patient with ectopic arrhythmia may be a difficult task. When sedation or vagal stimulation are ineffective, successful use of the commonly utilized drugs, digitalis, quinidine sulfate, or procainamide hydrochloride, often taxes the skill and patience of the physician. None are without significant hazard. The toxic effects of the antiarrhythmic drugs are to a large extent no more than an excess of the hoped for therapeutic effect. Adequate trial of a drug in an arrhythmia may demand dosage to the point of toxicity.

Atrioventricular dissociation and ventricular automaticity induced by digitalis are well known.

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Both quinidine sulfate and procainamide hydrochloride slow ventricular conduction, depress myocardial function, and both are peripheral vasodilators. Any one of these effects may be deleterious in patients with organic cardiac disease.

The ever present possibility of adverse drug reaction, and the necessity for tedious, and to the patient, often expensive, titration of dose and response in the treatment of arrhythmia, has stimulated a search for new techniques. Brief precordial electric shock to abolish ventricular fibrillation is an established form of therapy.¹ Several reports have described the application of precordial electric shock in the treatment of ventricular and supraventricular arrhythmias.²⁻⁴ Lown et al have investigated in detail the effects on the heart of direct current and alternating current shock in the experimental animal and presented evidence that direct current shock is more effective and less damaging than alternating current.⁵ More recently, the use of synchronized direct current shock in the treatment of arrhythmia in 19 patients by Lown et al⁶ has created considerable interest.

This report describes the clinical experiences in the application of direct current precordial shock synchronized to the nonvulnerable period of the ventricle in the treatment of arrhythmia in 84 instances.

Materials and Methods

Direct current precordial shock was administered from a Lown cardioverter. This unit, which is calibrated in units of energy or watt seconds, delivers an underdamped direct current discharge of 2.5 milliseconds duration from a 16 microfarad (μ f) condenser.⁵ Except in two patients with supraventricular tachycardia, all shocks were administered, utilizing a synchronizer circuit, 0.07 sec after the onset of the QRS complex to avoid the vulnerable period of the ventricle.⁵ Unless the reversion was

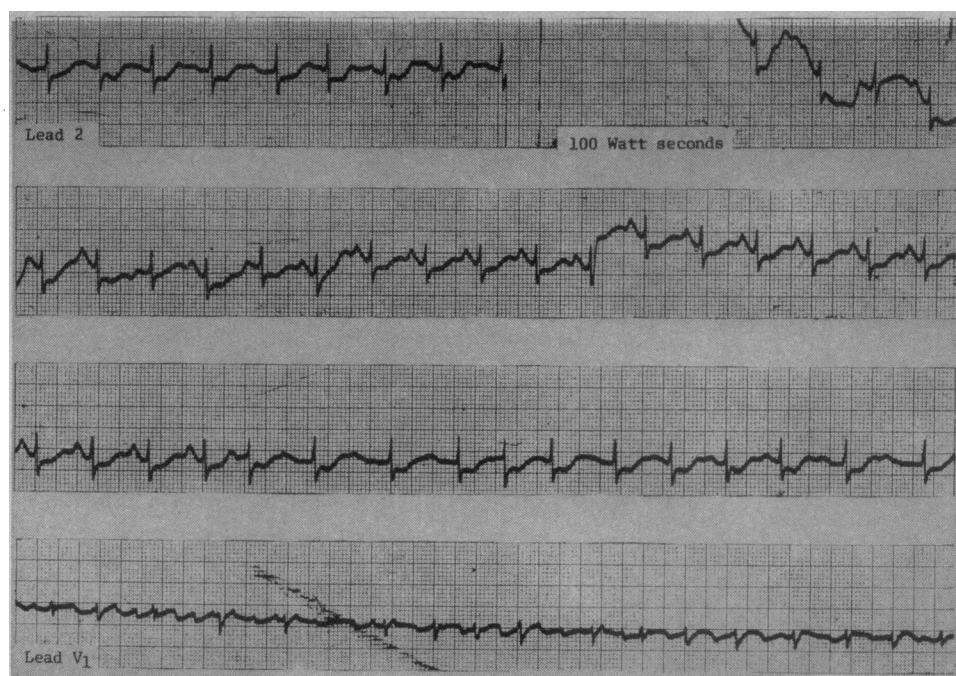


Fig 1.—Continuous electrocardiogram during reversion of atrial fibrillation, present ten years, to sinus rhythm in 62-year-old woman, 5 months after mitral valvuloplasty. First beats discernible after displaced writing arm returns are sinus. Fifth P wave in third line is premature and followed by atrial fibrillation, confirmed in lead V₁. Second shock, not shown, again resulted in normal rhythm which persisted.

deemed an emergency procedure, the patients were considered for shock only when optimal medical condition had been established. A routine procedure that permitted occasional variations was established.

Nothing was given by mouth after midnight save 0.4 gm of quinidine sulfate in the early morning. The procedure was carried out in the recovery room. An electrocardiogram was obtained immediately before and after shock therapy. The electrocardiogram was monitored on an oscilloscope and on a direct writer throughout the procedure. Synchronization of the direct current shock to the preselected segment of the cardiac cycle was routinely checked prior to each procedure. The paddle electrodes were approximated and the main cable of the electrocardiogram lead wound around one of the paddle handles. Discharge of the full capacity of the converter unit through the paddles as an electrocardiogram was recorded produced a sharp spike on the tracing at the moment of discharge.

The patient was given thiopental sodium intravenously in a dosage ranging from 75 to 400 mg. When the patient was lightly anesthetized the paddle electrodes, liberally covered with electrode paste, were applied across the precordium and the direct current shock administered. One electrode was placed on the chest in the region of the apex of the heart and the other at the base of the heart to the right or left of the sternum. Usually, consciousness was regained within 3 to 5 min. Patients were observed for approximately 1 hour after reversion and then returned to their rooms. Prophylactic antiarrhythmia therapy was continued following reversion.

Results

Direct current precordial electric shock synchronized from the electrocardiogram to a pre-selected area of the cardiac cycle has been utilized to treat arrhythmias in 62 patients on 84 separate occasions. Sinus rhythm was produced in 56 patients and in 71 reversion attempts. Successful reversions were accomplished in atrial tachycardia, atrial tachycardia with block, atrial flutter, atrial fibrillation, and ventricular tachycardia. The table lists the experiences with the various arrhythmias.

Electrocardiograms.—When reversion was successful the first few beats discernible following the shock usually revealed sinus rhythm (Fig 1). Occasional atrial premature contractions occurred following reversion in the majority of patients with atrial arrhythmias, but they usually disappeared within a few minutes. A short period of nodal rhythm in the immediate postreversion period was not uncommon (Fig 2). Ventricular premature contractions occurred sporadically in ten patients after reversion. All patients having ventricular premature

Synchronized DC Countershock for Treatment of Arrhythmias

Rhythm	Patients	Attempts	Success: NSR	Failure
Atrial tachycardia	3	4	2	2
2:1 Atrial tachycardia ...	4	6	4	2
Atrial flutter	7	8	7	1
Atrial fibrillation	46	59	53	6
Ventricular tachycardia ..	6	8	6	2
Totals	66*	84†	71	13

* Four patients had two different supraventricular arrhythmias and are counted twice.

† Fourteen patients had two conversion attempts and two patients had three conversion attempts for the same arrhythmia.

contractions after shock also had them prior to direct current shock. In two patients ventricular premature contractions with bigeminy which appeared to reflect over-digitalization developed immediately after reversion. They were successfully treated by withholding digitalis and giving potassium supplements.

With three exceptions, electrocardiograms obtained immediately and serially following precordial direct current shock showed no change from control tracings aside from changes in rhythm. In a 14-year-old boy with atrial tachycardia in whom reversion was attempted twice (*vide infra*), transient elevation of the ST segments in a right precordial lead occurred for approximately 20 sec after shocks of 200 and 400 watt sec. Serial transaminases remained normal. Right bundle-branch block occurred transiently in two other patients.

Roughly one-third of the patients had received standard drug therapy with digitalis, quinidine sulfate, or procainamide hydrochloride in an unsuccessful attempt to induce sinus rhythm. All but four patients were fully digitalized. Electrical reversion was performed 72 times, but was considered an emergency procedure in 12 instances.

Report of Cases

CASE 1.—Sudden tachycardia, dyspnea, and weakness developed in a 65-year-old man. He had previously suffered two documented myocardial infarctions. He was treated at another hospital with digoxin and procainamide hydrochloride without effect. Seven days after the onset of arrhythmia he was transferred to The New York Hospital. The patient's blood pressure was 90/60 mm Hg. There was evidence of congestive heart failure.

An electrocardiogram confirmed the diagnosis of ventricular tachycardia. The ventricular rate was 187 and the atrial rate 80 beats per minute. After 150 mg of thiopental sodium, given intravenously, a precordial direct current shock of 150 watt sec timed to the R wave was followed by immediate restoration of normal rhythm (Fig 3). The blood pressure rose to 130/90 mm Hg. An electrocardiogram obtained after restoration of sinus rhythm was unchanged when compared to tracings taken prior to the onset of the arrhythmia. Recovery was uneventful.

Diagnoses and Selection

By far the largest group of patients had rheumatic heart disease and atrial flutter or fibrillation. Other diagnoses included ischemic heart disease, acute myocardial infarction, ventricular aneurysm, hypertensive heart disease, myocardopathy, and treated thyrotoxicosis. Selection of patients for elective electrical reversion depended on a variety of factors, including progression of symptoms, onset of symptomatic arrhythmia, and recent or even remote successful mitral valvuloplasty.

Neither heart size nor duration of arrhythmia influenced the decision to attempt reversion. Duration of arrhythmia varied from a few hours in some patients to more than 15 years in others with successful results at both extremes. Heart sizes varied from normal to very large, with massive left atrial

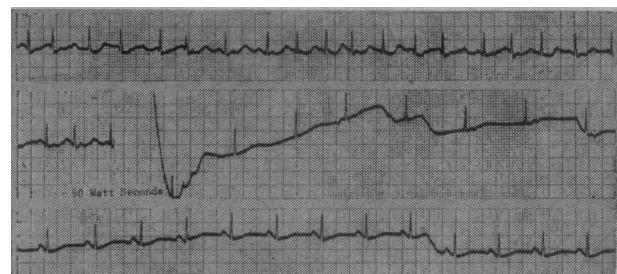


Fig 2.—Continuous electrocardiogram during direct current reversion of atrial fibrillation present 7 days in woman, age 46, with mitral stenosis. Nodal rhythm initially follows atrial depolarization but is superseded by faster sinus discharge.

enlargement. Success was obtained in eight patients with extremely large hearts. The improvement in exercise tolerance in many patients whose normal rhythm was restored and maintained was gratifying.

CASE 2.—A 55-year-old man had undergone mitral valvulotomy 15 and 12 years previously. Atrial fibrillation had persisted for at least 10 years. Several attempts at reversion with quinidine sulfate had been unsuccessful. Despite adequate digitalization, exercise tolerance was severely curtailed by angina, tachycardia, dyspnea, and fatigue. Hemodynamic studies revealed a low cardiac output and moderate mitral stenosis. The heart was slightly enlarged.

After reversion to normal sinus rhythm with a precordial direct current shock of 150 watt seconds, exercise tolerance improved dramatically. Angina and disproportionate tachycardia no longer developed after exertion. For the first time in more than 10 years the patient felt well enough to seek steady employment. Hemodynamic studies at rest and during exercise, to be summarized elsewhere, objectively documented the striking improvement.

Multiple Attempts

In 18 patients reversion was attempted on two separate occasions. It was successful both times in 11 patients, successful the first but not the second in three, successful the second but not the first time in two, and failed both times in three. Two patients successfully underwent reversion for atrial fibrillation three times, 2 weeks and 3 months apart. Because of the marked symptomatic improvement with sinus rhythm these two patients and their physicians were strongly motivated to reconvert and readjust preventive drug dosage when relapse occurred.

Half of the patients received more than one shock. The average for the total series was 2.0 shocks per patient, but as many as six were administered to a single patient. Usually, the first shock was in the low energy range, 100 watt seconds or less. If this was not successful a medium energy shock between 100 and 300 watt seconds was given. If this was not successful the full output of the instrument, 400 watt seconds was applied.

Failure to Restore Sinus Rhythm

Direct current shock failed to produce sinus rhythm in six of 62 patients, or 10% of the patient

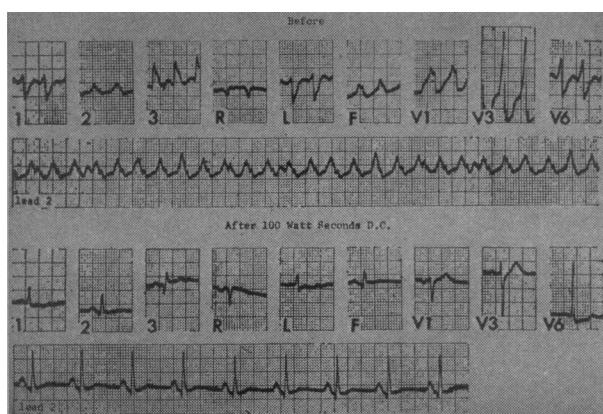


Fig 3.—Tracings immediately before and after reversion of ventricular tachycardia, present 7 days, to normal rhythm with a single synchronized direct current shock of 100 watt seconds in 65-year-old man with previous myocardial infarction. Postshock electrocardiogram is unchanged from prearrhythmic tracings.

total. Of the 84 attempts, 13, or 15% failed. Factors which may have influenced the failure to produce normal rhythm were variable. Inability to effect sinus rhythm in two patients with atrial tachycardia may have been related to concomitant catechol amine therapy. When the amines were withdrawn one patient reverted to normal rhythm spontaneously and the other patient responded to a second attempt with direct current shock.

Atrial tachycardia with block in young boy did not respond to multiple shocks and it was surmised that the patient had a primary abnormality of pacemaker tissue. Two of the three patients with atrial flutter or fibrillation in whom shock failed to produce sinus rhythm had very large left atria secondary to rheumatic mitral disease.

One other patient, a woman aged 67, had only a moderately enlarged heart and no evidence of valvular involvement. Failure to revert ventricular tachycardia in one case was associated with sarcoidosis of the heart, and in another case with advanced uremia. In the latter, direct current shock early during peritoneal dialysis was not successful but produced normal rhythm several hours later when the dialysis had been completed. Brief case reports describe our experiences with these patients.

CASE 3.—A 30-year-old woman had severe mitral regurgitation and a giant left atrium complicated by atrial fibrillation for 3 years. Seventeen days after successful insertion of a mitral valve prosthesis, reversion was attempted with shocks of 30, 150, 300, and 400 watt seconds without success.

CASE 4.—A 54-year-old salesman had ventricular tachycardia from varying foci at different ventricular rates for 3 weeks. Chest roentgenograms showed nodular streaking in both lungs. His condition deteriorated and electrical reversion was attempted. The ventricular rate was 187 and the atrial rate 82 beats per minute. Despite shocks of 200 and 400 watt seconds the rhythm did not change. During the next 24 hours 5.0 gm of procainamide hydrochloride were administered intravenously with slowing of the ventricular rate, but the arrhythmia persisted. Autopsy revealed diffuse

sarcoidosis and gross sarcoid infiltration in the ventricular septum in the region of the main bundle.

CASE 5.—A 14-year-old boy was found to have an irregular pulse during examination by his school physician. An electrocardiogram showed atrial tachycardia at a rate of 177 beats per minute. There was no evidence of organic heart disease and an angiocardiogram was normal. Administration of digitalis resulted in a 2:1 atrioventricular block. Reversion was attempted twice and despite a total of six shocks, three of which were synchronized to 0.04 sec after the unblocked P wave with a maximum energy of 400 watt seconds, there was no change in the rhythm. A satisfactory diagnosis was not established, although it was postulated that the boy had a congenital arrhythmia.

Temporary Reversion

Sinus rhythm which persisted less than 5 min occurred in six patients (Fig 1). In three a second shock administered immediately after the observed relapse resulted in sinus rhythm which was maintained. In three patients repeated shocks were not followed by established sinus rhythm and the procedure was abandoned. An especially interesting case history is given below.

CASE 6.—A 46-year-old Negro man had had atrial fibrillation for 5 years. His heart disease was of an undiagnosed type characterized by polycythemia, anoxemia without hypercapnia, moderate enlargement of the heart, and no murmurs. A single shock of 300 watt seconds resulted in sinus rhythm which persisted 50 sec. Despite administration of 750 mg of procainamide hydrochloride intravenously and four more shocks during the next one-half hour, sinus rhythm was maintained no more than 75 sec at a time. Six days later, after daily doses of quinidine sulfate of 1.2 gm, 1.6 gm, and 2.0 gm, two successive shocks of 400 watt seconds resulted in sinus rhythm lasting 40 sec and 18 sec respectively.

Thus sinus rhythm followed direct current shock six or seven times, but did not persist despite co-incident drug therapy. It seems likely that failure of sinus rhythm to continue is evidence of atrial rather than pacemaker dysfunction.

Anticoagulation and Emboli.—All patients were carefully evaluated after reversion for evidence of emboli. In 73 successful reversions to sinus rhythm no embolic phenomenon occurred. Anticoagulant therapy with coumarin derivatives had been established prior to 33 reversions. Anticoagulants had not been given prior to 40 reversions. Before reversion, 24 patients with rheumatic heart disease, atrial fibrillation or flutter, and mitral stenosis received anticoagulants; 31 did not.

Transaminase.—Three patients had significant rises (more than 20 units) in SGOT after reversion. In one patient the elevation was associated with a fever induced by quinidine sulfate. In another very muscular patient the elevation followed five precordial shocks which produced spasm and subsequent tenderness of the left pectoralis muscle group. In the third patient the increased transaminase was not readily explained. It was not accompanied by other laboratory or electrocardiographic or physical evidence of myocardial damage.

Serum glutamic oxalacetic transaminase was

analyzed serially in 42 patients. Control levels averaged 27.0 ± 3.4 (standard error of the mean). Transaminase on the first day after reversion averaged 29.6 ± 3.8 , and 31.8 ± 4.3 on the second day. These differences are not significant. Thus, there is no evidence that direct current precordial shock influenced blood transaminase activity.

Abnormal Rhythm.—Direct current shock produced a rhythm other than sinus rhythm in three patients whose case histories are briefly outlined below.

CASE 7.—In a 63-year-old woman with hypertensive heart disease atrial tachycardia developed at a rate of 140. Conventional treatment was unsuccessful and her blood pressure required support with metaraminol bitartrate and levarterenol. A direct current shock under emergency conditions of 150 watt seconds timed to the R wave produced atrial tachycardia at a rate of 210 with 2:1 atrioventricular block. Four subsequent shocks of 300 and 400 watt seconds were ineffective. One week later, when catechol amine therapy was no longer required, a single direct current shock of 200 watt seconds resulted in sinus rhythm which persisted.

This patient was treated early in the series and synchronization was adjusted for the R wave and not for the P wave. Persistence of the tachycardia in the first attempt at reversion may have been related to failure to depolarize the atria because of improper synchronization or the administration of catechol amine therapy. When the latter was withdrawn reversion was successful.

CASE 8.—Atrial fibrillation and a cerebral embolus developed in a 64-year-old woman 6 months prior to reversion. She had a history of slow nodal rhythm. There was no evidence of valvular disease or myocardial infarction. An electrocardiogram was compatible with incomplete left bundle-branch block. A direct current shock of 200 watt seconds was followed by nodal rhythm at a rate of 40 with occasional sinus beats. Intermittent doses of atropine were required for 36 hours to maintain an adequate ventricular rate. Following potassium depletion and alkalinization she reverted to atrial fibrillation.

The history of nodal rhythm and failure of a sinus pacemaker to become established after depolarization of the atria suggests that the patient had a "dead" sinus node. A relation between disease of the artery to the sinus node and the genesis of atrial arrhythmias has been suggested recently.⁷

CASE 9.—A 39-year-old woman with mitral stenosis fainted in a movie. She was found to have atrial fibrillation and left bundle-branch block. A course of quinidine sulfate failed to induce sinus rhythm. Her symptoms increased and 3 months later direct current reversion was attempted under pentothal anesthesia. A control electrocardiogram showed occasional premature ventricular contractions. A shock of 50 watt seconds caused no change.

A second shock of 150 watt seconds was apparently triggered during the ST segment by an artifact and was followed by ventricular fibrillation (Fig 4, *left*). Defibrillation was immediately accomplished with a shock of 300 watt seconds. Atrial fibrillation persisted and 5 min later a shock of 400 watt seconds was administered 0.07 sec after the onset of the QRS complex. Ventricular fibrillation again appeared (Fig 4, *right*). The patient was immediately defibril-

lated with a fifth shock of 400 watt seconds. The procedure was abandoned. There was no sequelae and serial transaminase determinations were normal. The electrocardiogram was unchanged.

This experience emphasizes the fact that a potential hazard of any form of shock therapy to the heart is ventricular fibrillation. Fortunately, treatment of fibrillation may be readily accomplished by a second shock. The source of the artifact could not be determined. Possibly it was associated with a movement by the patient.

Comment

Precordial direct current shock triggered to a preselected moment of the cardiac cycle appears to be a safe, simple, and effective method for the treatment of abnormal cardiac rhythms. When the proper instrumentation is available, precordial shock may be applied quickly. The results of therapy are immediately apparent. The necessity of undertaking a tedious titration of dosage and response, as is so often the case with drug therapy, may be avoided.

Use of anesthesia has not presented a serious obstacle. Seriously ill patients receive minimal amounts of thiopental sodium and no untoward effects have been observed. Intubation has not been required. It is not certain that anesthesia is necessary in all cases. (Dr. Richard J. Stock has treated ten patients with synchronized direct current shock without general anesthesia.⁸)

Complications.—The only complications encountered in the present series were related to the generation of other arrhythmias in three patients. In each instance the induced arrhythmia responded to appropriate therapy, including further direct current shock. One episode of induced rapid atrial tachycardia was probably related in part to coincident catechol amine therapy. In one patient, failure of sinus rhythm to override the lower pacemaker from the atrioventricular node strongly suggests intrinsic abnormality of the normal pacemaker tissue. One can readily imagine the catastrophe that might have resulted if a depressant drug such as quinidine sulfate had been selected to attempt normal rhythm. Indeed, it is possible that functional abnormality of pacemaker tissue coupled with suppression of compensatory mechanisms may explain some of the serious arrhythmias which have been encountered in the past with the use of depressant drugs.

Despite meticulous attention to detail, ventricular fibrillation was produced twice in one patient. No errors in technique were recognized. Routine check of the synchronizer circuit, as outlined under methods, insured proper function of the apparatus and proper position of all switches. An artifact apparently triggered the shock which occurred during the vulnerable period and produced ventricular fibrillation. A similar artifact has not been

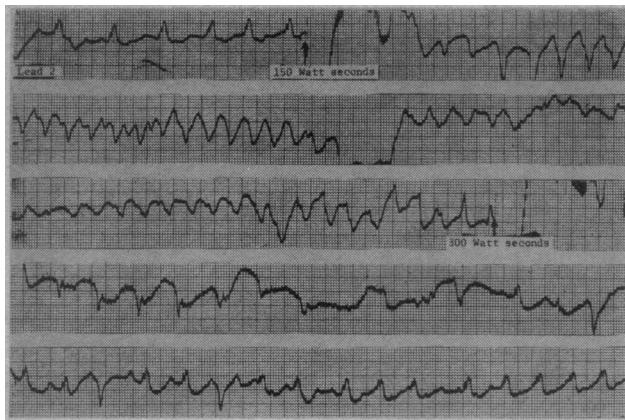


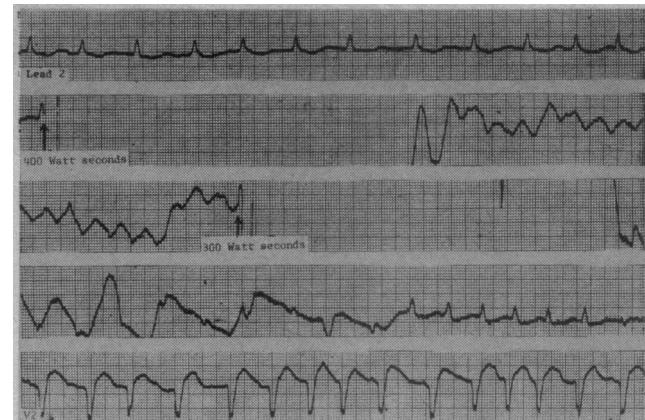
Fig 4.—Left. Attempted reversion of atrial fibrillation in a 39-year-old woman with mitral stenosis and left bundle-branch block. First shock, triggered by artifact, occurred during vulnerable period and was followed by ventricular tachycardia and fibrillation.

noted in the more than 170 synchronized shocks that have been given with permanent recording of the electrocardiogram during the procedure in the present series. After defibrillation another direct current shock occurring 0.07 sec after the onset of the QRS complex again produced ventricular fibrillation.

The cause of the second episode of ventricular fibrillation in this patient after a properly timed shock is not clear. Further experience will be needed to determine whether or not certain clinical combinations (such as bundle-branch block and ventricular premature contractions) or other unrecognized factors will predispose the development of ventricular arrhythmias following synchronized direct current shock.

Vulnerable Period.—Electric shocks to the heart during the vulnerable period may cause ventricular fibrillation.^{6, 9-11} Lown et al systematically explored the vulnerability of the ventricle to fibrillation by administering direct current shocks throughout the cardiac cycle.⁶ They found that fibrillation occurred only when the shock was given during the first portion of the T wave. The ventricles were resistant to fibrillation when shocked during other parts of the cardiac cycle. For safe utilization of the technique of precordial shock for arrhythmias it is axiomatic that the electrical discharge be accurately timed for delivery during the safe period by a synchronizer circuit. Failure to take this precaution may be disastrous.

Direct Current.—The use of direct current rather than alternating current for myocardial depolarization in man represents a departure from previous experience in this country, although a commercially available direct current defibrillator was available in the USSR in 1954.¹² Conflicting opinions comparing various modes of cardiac shock have accumulated during the past half century.^{5, 12} Lown et al recently compared the effectiveness and safety of synchronized alternating current and direct current



Second shock followed by few sinus beats and then atrial fibrillation. Right. Several minutes later properly synchronized direct current shock produced ventricular fibrillation which was terminated by another shock.

discharges in the fibrillating, hypothermic dog.⁵ They found that direct current was consistently more effective in defibrillating the heart and caused less myocardial damage than alternating current.

On the basis of this evidence and the redefinition of the vulnerable period, Lown et al introduced the use of synchronized direct current precordial shock for the treatment of ectopic arrhythmias.⁶ Negovskii has pointed out "that the voltage and strength of the current defibrillating the heart as a single impulse are practically the same as the voltage and strength of the alternating current required to defibrillate the heart."¹² Thus energy beyond one-half cycle of alternating current is not needed and may be harmful.

Transaminase.—Neither the transaminase data nor serial electrocardiograms revealed evidence of significant myocardial damage following direct current precordial shock. Whether single or repeated shocks may transiently influence myocardial function must await more precise data, but the advantages of a technique for treating sustained arrhythmia which does not adversely effect the myocardium are apparent.

Embolization.—No evidence of embolization during direct current reversion occurred in the present series, although anticoagulants were withheld in more than half the patients. Embolization has long been considered a risk in the treatment of arrhythmias, especially atrial fibrillation. Atrial thrombus, however, can only be correlated with atrial fibrillation in patients with rheumatic heart disease.¹³ Embolization during quinidine sulfate therapy may be related in part to the variable effects of the drug on ventricular rate and atrial contraction which develop during the hours or days of continued drug effect. In the overwhelming majority of patients, direct current precordial shock is all or none: either sinus rhythm supervenes or the arrhythmia persists.

Persistent Arrhythmia.—As further experience accumulates, careful analysis of the patients who fail

to develop sinus rhythm after precordial shock may provide insight into the pathogenesis of arrhythmias. One group of patients with atrial fibrillation probably have intrinsic disease of the sinus node and are unable to generate a regular pacemaker discharge following atrial depolarization. Others have sinus rhythm for short periods after shock but soon revert to atrial fibrillation, suggesting that either the pacemaker is unable to sustain active discharge or the atria are unable to maintain a coordinated beat. The repeated production of sinus rhythm in several patients followed by the abrupt appearance of atrial fibrillation without change in pacemaker rate tends to favor the thesis of an atrial abnormality. A third group, illustrated by the patient with sarcoidosis, have abnormality of the conduction pathways and atrial or ventricular automaticity.

Both the sites for placement of the electrodes in the chest and the maximum energy selected for attempted depolarization of the heart have been empirically determined. It is possible that failure to produce sinus rhythm in some patients is related to improper technique rather than intrinsic cardiac abnormality. The atria are posterolateral-antero-medial in orientation and similar orientation in electrode placement may be effective in refractory cases. Thickness of the chest wall and myocardium may influence energy required for depolarization. Some refractory arrhythmias may require more than the current maximum 400 watt seconds for termination. (A patient treated by Dr. B. Lown for refractory ventricular tachycardia required 800 watt seconds for reversion to sinus rhythm.¹⁴) Variations in technique which may influence the success rate are being investigated.

Maintenance.—Maintenance of normal rhythm depends on a variety of factors and is a problem separate from the feasibility of electrical reversion. This is especially true of the patients with atrial flutter or fibrillation. Most have organic heart dis-

ease and cardiac enlargement. Interplay of myocardial and hemodynamic abnormalities may be of variable importance in the genesis of the arrhythmia. The physician must decide whether maintenance of sinus rhythm represents a worthwhile achievement for the patient. Since the majority of patients require long-term suppressive doses of quinidine sulphate or similar drugs and since adjustment of drug therapy may require a period of trial and error with occasional relapse of arrhythmia and re-reversion with direct current shock, the motivation of the patient and the physician in maintaining sinus rhythm is paramount. To date (May, 1963, with longest follow-up 8 months), approximately one-third of the patients reverted from atrial fibrillation or flutter have reverted to the original arrhythmia. In at least one-half of this group, relapse was associated with inadequate prophylactic therapy. Few patients have been successfully maintained in sinus rhythm on less than 1.2 gm of quinidine sulfate daily. The patient's inability to tolerate quinidine sulfate or similar drugs may limit successful application of long-term suppressive therapy.

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Generic and Trade Names of Drugs

Quinidine sulfate—Asarum compound, Quinicardine, Quinidate, Quinidex.
 Procainamide hydrochloride—Pronestyl Hydrochloride.
 Metaraminol bitartrate—Aramine Bitartrate.
 Levarterenol bitartrate—Levophed Bitartrate.
 Digoxin—Lanoxin.

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