SYMPOSIUM
Cardiac Arrhythmias
(Part 6)

Present Status of Electroversion in the Management of Cardiac Dysrhythmias

By Leon Resnekov

SUMMARY
The theoretical and practical considerations of electrical reversion of cardiac dysrhythmias are reviewed and a comparison made between AC and DC defibrillation, indicating the superiority of DC under all circumstances. The indications for, immediate and late results of DC shock for atrial and ventricular dysrhythmias are presented, the complication of such treatment reviewed, and the need for anticoagulant cover, anesthesia, and drug therapy preceding and following the electrical treatment discussed. Each patient requires individual assessment, particularly those with chronic rhythm disturbances, especially atrial fibrillation, in whom electrical energy settings in excess of 300 joules are rarely indicated for the risk of complications becomes progressively higher as the energy setting is increased and the length of time sinus rhythm persists in this group of patients may be short. Patients with acute rhythm disturbances, however, with potentially serious hemodynamic consequences should be treated with maximum electric energies if needed. Caution is also advised in patients with coronary heart disease, atrial fibrillation, and a slow ventricular rate, even in the absence of digoxin, patients with rapidly changing rhythm disturbances, those who cannot maintain sinus rhythm for a significant period of time despite drug therapy, patients with the "sick sinus syndrome," those with atrial fibrillation of more than 5 years standing with a cardiothoracic ratio exceeding 55%, and patients in lone atrial fibrillation. Heavily digitalized patients in general should have their electroversion postponed if possible, but if not, they should be protected against serious ventricular rhythm disturbances immediately after the shock by an intravenous dose of lidocaine, phenylhydantoin, or procaine amide immediately before and the initial energy setting should be reduced to 5 joules. Quinidine or some other antidyssrhythmic drug may be needed in an attempt to maintain sinus rhythm after successful electroversion, but even when controlled with adequate blood levels, results are poor.

Additional Indexing Words:
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Antidyssrhythmic drugs
DC defibrillation
Digitalis
Sick sinus syndrome
Anticoagulants

Drug therapy, often successful in treating cardiac dysrhythmias, has many limitations which cause serious disadvantages in its routine use. There is, for example, no universal agreement regarding the choice of drug nor any standardized dosage scheme applicable to all patients. Furthermore, the patient has to be kept under close observation over several days while the dose given and effect caused are titrated. Only a small margin separates the therapeutic from toxic effects, and many of the antidyssrhythmic drugs also have important negative inotropic and dromotropic effects. Should toxic manifestations emerge, they may be more serious than the original rhythm disturbance for which the drug was given, and

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Paradoxically, drug overdosage may well depress the normal sinus mechanism, thereby inhibiting reversion to sinus rhythm.

In contrast, an electric shock across the chest and myocardium causes momentary depolarization of the majority of heart fibers, terminates the ectopic rhythm, and allows the sinus node to be reestablished as the normal pacemaker.

Early History

In papers dealing with the effects of electric current on the myocardium, Prevost and Battelli noted almost as an afterthought that direct current shock across the heart would end ventricular fibrillation in dogs. This important fact continued generally unrecognized or seemed largely forgotten until Kouwenhoven, Hooker, and Langworthy and Ferris, King, Spence, and Williams undertook detailed studies on the effects of electricity on the heart and reported the following important conclusions: (1) current rather than voltage is a proper criterion for shock intensity, (2) the passage of electrical current across the heart may precipitate ventricular fibrillation even in the absence of any recognizable myocardial damage, and (3) death will follow unless this is successfully treated by another shock within a few minutes.

Subsequently, pioneer work into the use of capacitor discharge (direct current) for clinical application was undertaken in the Soviet Union between 1935 and 1950; these studies can be regarded as an important inspiration to later investigators, including Peleska of Czechoslovakia, Tsukerman of the Soviet Union, and Lown and his group in this country.

Alternating or Direct Current

Whether alternating or direct current is used, electrical defibrillation requires a high-energy impulse of short duration to be passed across the myocardium, either between two concave paddles closely applied to the heart (internal defibrillation) or through the chest wall using two flat paddles (external defibrillation). The total electrical energy delivered to the heart muscle depends not only on the electrical current, but also on the resistance of the heart, bony cage, and skin to its passage.

Much of the confusion in the earlier literature comparing alternating with direct current defibrillation relates to the infinite wave forms obtainable using direct current discharge. In contrast, the alternating current wave form is constant and determined at the power station as a sinusoidal-shaped impulse with a frequency of 60 Hz but at a variable voltage level. A minimal current of 1 amp is needed to bring all the heart muscles instantaneously to the same refractory point. The required voltage for internal defibrillation is 100 v and the power 100 w. Electric energy (power × time) is not synonymous with electrical current, and for internal defibrillation the energy needed would be about 20 joules (wsec). In contrast, for external defibrillation a sixfold current increase is used producing an energy of 360 joules.

With direct current defibrillators, the duration of the impulse is short (1.5-4 msec). The wave form is shaped by adding varying amounts of inductance to the circuit, thereby insuring minimal biologic damage to its passage across the tissues of the body, particularly the heart. The important difference between alternating and direct current defibrillation is that DC develops many times the power of AC. Direct current discharge, however, is much shorter than alternating current (3 msec vs 200 msec).

The first successful human defibrillation was reported by Beck, Pritchard, and Fell in 1947, and thereafter the convenience of AC defibrillation determined it as the standard method. Alternating current, however, was subsequently found to fail on many occasions when ventricular fibrillation was due to myocardial infarction, and also the need for electrically converting rhythm disturbances other than ventricular fibrillation. These two factors stimulated further work in the use of direct current.

Despite the fact that Lown and his colleagues reported the first AC termination of an organized rhythm disturbance, the experience of Zoll and Linenthal gave clear warning of the real risk of precipitating ventricular fibrillation and even death when alternating current was used in this way. Furthermore, severe deterioration of ventricular function follows transmyocardial AC shocks. By 1966 Nachlas et al. were able to show conclusively that DC was superior to AC in terminating experimental ventricular fibrillation.

From all this evidence, therefore, it can be concluded that alternating current is feasible for treating ventricular fibrillation, but that direct current is more effective; alternating current, however, cannot be recommended for electively treating atrial rhythm disturbances or ventricular tachycardia.

Synchronization

For many years a vulnerable period of ventricular excitability had been postulated in a wide variety of
animals and Wiggers and Wegria\textsuperscript{14} were able to show this to be 27 msec before end of systole in the dog, a similar period of atrial vulnerability has also been demonstrated.\textsuperscript{15} Nonuniform recovery from the refractory state occurs during the vulnerable period of the ventricle, and reentry of the depolarization wave and self-sustained activity are thereby favored. The risk of ventricular fibrillation following the shock should be reduced, therefore, by synchronizing with the R or S wave, thereby avoiding the apex of the T wave. It will be appreciated that it is almost impossible to avoid this phase when AC shocks are used because of their long duration. The chance occurrence of ventricular fibrillation following random unsynchronized DC shocks is about 2\% and Kreus, Salokannel, and Waris\textsuperscript{16} deliberately use no synchronization in clinical practice without dire consequence. The important point when no synchronizer is used, however, is to insure that sufficient energy is delivered so that a current of at least 1.5–2 amps passes across the heart; smaller energies may well be dangerous. Of course, when ventricular fibrillation is to be treated, the synchronizer must be switched out of the circuit.

**Clinical Use of Direct Current Capacitor Discharge for the Management of Cardiac Dysrhythmias**

The overall success rate for the DC termination of atrial and ventricular rhythm disturbances approaches 90\%.\textsuperscript{17–20} Even when determined efforts at conversion with drugs fail, direct current may often succeed.\textsuperscript{21} The correct choice of patient, meticulous attention to detail, correction of electrolyte imbalance, postponement of treatment in the presence of overdigitalization, proper synchronization, and choice of antidyssrhythmic agent immediately prior to and following treatment insure both immediate success and an absence of a high incidence of complications. The importance of meticulous attention to detail, however, cannot be overemphasized. Monthly checks of the apparatus should be made for overall electrical safety, including inspection of the wave form and measurement of the actual electrical energy following discharge across a 50-ohm load in the laboratory. Most apparatus provides a moving coil meter calibrated in joules (wsec). Models, however, are still in use, in which the desired energies are obtained by depressing a labeled switch, a highly undesirable feature. During actual patient treatment, the assisting personnel should not touch the patient, his bed, or any of the apparatus, for the large electrical field created at the moment of discharge may result in shocking any person in contact. The synchronizer should always be tested just before undertaking patient treatment by carefully examining the superimposed artifact on the ECG signal, or if this welcome refinement is not available, by discharging the capacitor away from the patient with the two paddles in close contact and their leads in apposition to the patient’s electrocardiogram leads. In this way an artifact is superimposed on the electrocardiogram tracing and can be examined for correct timing.

Paddles used should always be of adequate size, for if they are too small the electric current density is extremely high and the possibility of myocardial damage very real.\textsuperscript{22} It has been reported that the anterior and posterior paddle position for external defibrillation significantly lowers the electric energy needed for electroversion;\textsuperscript{23} others, however, could not confirm this.\textsuperscript{24} Indeed, experimental work\textsuperscript{25} has shown that anterior positioning of both paddles in the longitudinal plane delivers more current to the heart. The failure to note more striking clinical differences with changes in paddle positioning is probably due to the excess electrical energies which are consistently used to achieve electroversion. The added safety of a flat posterior paddle supported only by the weight of the patient and untouched by the operator is advantageous, however, and the anteroposterior position is therefore recommended.

Digoxin or other digitalis preparations should be withheld for 24–28 hours before treatment if at all possible. If the treatment cannot be postponed but has to be undertaken in the presence of heavy digitalization, the initial energy setting should be markedly reduced to 5 joules for an adult, and an intravenous injection of 50 mg lidocaine, 50–100 mg phenylhydantoin, or 50–100 mg of procaine amide should always precede the shock. Many use quinidine or some other antidyssrhythmic agent routinely before administering the shock, but their efficacy in reducing energy requirements or in helping to maintain sinus rhythm thereafter is questionable.\textsuperscript{26} General anesthesia is not mandatory, and amnesia produced by diazepam 5–10 mg i.v., can now be recommended.\textsuperscript{27}

Treatment should be undertaken in an area fully equipped for cardiac monitoring and resuscitation, including emergency pacemaking. The heart rate should be displayed on a tachometer, and the electrocardiogram constantly on an oscilloscope, and preferably recorded as a direct tracing as well.
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A short strip of V₁ before administering the shock is advantageous for subsequent comparison, as immediately after the shock P waves can be difficult to detect in the standard limb leads, even when sinus rhythm is present. The skin should be prepared by a liberal application of ECG paste rubbed well in to reduce electrical resistance, and thereby prevent painful skin burns. Great care must be taken to insure that no part of the patient’s skin is in direct contact with the metal of the trolley or the metal of the bed on which he is lying.

With either two anterior, or preferably an anterior and a posterior paddle, small energies should be administered first, and if unavailing, shocks are repeated at increased energy level settings. For an adult, an initial setting of 25-50 joules is satisfactory, increasing in 25-50-joule steps as needed. If heavy digitalization is present, an initial shock of 5 joules is appropriate. Should ectopic beats follow the first shock, 50 mg lidocaine should be given intravenously and as a routine before any patient known to be heavily digitalized.

The initial setting for a child is 5-10 joules delivered across appropriately sized pediatric paddles and increasing by 5-10-joule increments.

There should be great reluctance to exceed an energy setting level of 300 joules in an adult being treated for a chronic rhythm disturbance, although the situation is quite different when an acute rhythm disturbance producing serious hemodynamic effects is present; maximal energies (400 joules) should now be used if needed.

The initial setting for treating ventricular fibrillation (no synchronizer in circuit) in an adult is 200 joules, increasing by 100-joule steps to a maximum of 400 joules.

Appropriate settings for internal defibrillation (special paddles) are 20-100 joules in 20-joule increments for an adult, and 5-50 joules in 5-joule increments for a child.

Immediately after the shock with the development of sinus rhythm or failure to achieve sinus rhythm after optimal energy, the amnesic drug is discontinued and a 12-lead electrocardiogram recorded. The electrocardiogram should be monitored for the next 24 hours or longer if need be, and records of blood pressure should be taken every half hour if hypotension occurs.

Individual Rhythm disturbances

Atrial Fibrillation

Atrial fibrillation is the most common rhythm disturbance to be treated, but lone atrial fibrillation deserves special mention, for the success rate of its electroversion is low, the incidence of complications high, and the length of time during which sinus rhythm persists is often disappointingly short. Initial success rates for atrial fibrillation are in general around 90%, irrespective of the underlying cause; for lone atrial fibrillation it is only 79%. Successful electroversion is not related to age or sex, type of heart disease (lone atrial fibrillation accepted), overall body size, nor to the size of the f waves in lead V₁ of the electrocardiogram, but does depend on the duration of the rhythm disturbance, being less than 50% when atrial fibrillation has been present for 5 years or longer. Similarly, a cardiothoracic ratio of 50% or more and selective enlargement of the left atrium lessen the chance of success.

Every patient, therefore, with chronic atrial fibrillation requires individual assessment to determine whether treatment is worthwhile. Nevertheless, hemodynamic benefit may be achieved in sinus rhythm, especially when patients are studied at progressive exercise loads. Certain groups of patients, particularly those with severe mitral regurgitation, may only be maintained free of cardiac failure by repeated electric terminations of episodes of atrial fibrillation.

Unless sinus rhythm is needed to maintain the circulation or the mechanical efficiency of prosthetic heart valves, electroversion should be postponed until patients are convalescing following open- or closed-heart surgery. Reversion to atrial fibrillation in the immediate postoperative phase is almost universal if open-chest electroversion is undertaken at the time of surgery, and atrial fibrillation with a ventricular rate controlled by digoxin is often to be preferred to rapidly changing rhythms in the postoperative phase.

The following groups of patients are not suitable for the electroversion of atrial fibrillation, which should be attempted only under unusual circumstances: (1) Coronary heart disease with atrial fibrillation in which the basic ventricular rate is slow, even in the absence of digoxin; (2) patients not able to maintain sinus rhythm for more than a brief period, even when maintained on an adequate dose of an antidysrhythmic drug; (3) patients presenting with variable atrial rhythm disturbances in rapid succession; (4) patients with the “sick sinus syndrome”. If electroversion is done during the rapid atrial phase of the syndrome, a transvenous pacemaker should first be positioned in the
right ventricle, for there is a serious risk of severe bradycardia or complete asystole following the shock; (5) patients with chronic valvar heart disease and long-standing atrial fibrillation (more than 5 years), considerable enlargement of the heart (cardiothoracic ratio more than 55%), and in whom cardiac surgery is not contemplated; and (6) patients with lone atrial fibrillation.

**Atrial Flutter**

The success rate is more than 90%, and the average energy level setting required for electroversion is only 50 joules. Unlike lone atrial fibrillation, patients with idiopathic atrial flutter may still be successfully reverted by direct-current shock at low energy level settings, even when the rhythm disturbance is known to have been present for several years; furthermore, sinus rhythm is frequently maintained for a significant period of time.25

**Paroxysmal Atrial Tachycardia**

Simple vagal stimulation and drug therapy remain the initial treatment of choice and when used for resistant PAT, electroversion succeeds in about 80% of cases. It should only be used for digitalis-induced supraventricular or junctional tachycardias under the most exceptional circumstances, for the risk of ventricular fibrillation being precipitated is high.31

**Ventricular Tachycardia**

This is a particularly gratifying dysrhythmia to treat; the initial success rate is 97% and electric energies needed are low, the average setting being 50 joules. There should therefore be no hesitation in recommending electroversion for any ventricular tachycardia which fails to respond quickly to emergency drug therapy. Caution has to be advised, however, when ventricular tachycardia is digitalis-induced; electroversion can now only be recommended under the most exceptional circumstances and drug therapy is the treatment of choice.

**Ventricular Fibrillation**

Controlled clinical trials of direct current (unsynchronized) vs alternating current for defibrillation are difficult to design, but there is good animal experimental work to indicate the superiority of DC,18 and in clinical practice successful resuscitation (patient leaving hospital) has been reported in more than 50% of patients treated by DC shock and well conducted principles of resuscitation.32 The initial energy setting should be 200 joules, increasing by 100-joule increments if needed.

**Complications**

Complications occur more frequently than initially predicted, and do not only relate to drugs given to maintain sinus rhythm. Indeed, an incidence of 14.5% among 220 consecutive patients has been reported;33 these did not include minor complications such as superficial skin burns due to poor preparation of the skin or transient rhythm disturbances after the shock. Included, however, were the following.

**Raised Levels of the Serum Enzymes LDH and CPK (10%)**

Their origin, however, is still uncertain. Mandecki et al.34 consider skeletal muscle damage as the cause, but as other signs of myocardial damage frequently coexist when the serum enzyme levels are raised, the matter awaits definitive study.

**Hypotension (3%)**

Hypotension is more common when higher electric energies are used, and may persist for several hours, frequently requiring no particular intervention, but the patient should be carefully observed.

**ECG Evidence of Myocardial Damage (3%)**

Patterns of myocardial infarction may follow the shock and persist for many months; they are most common following electroversion at high energy settings.

**Pulmonary and Systemic Emboli (1.4%)**

The incidence is similar to that following quinidine conversion of rhythm disturbances but does indicate the need for anticoagulant cover (see below).

**Ventricular Rhythm Disturbances**

Ventricular premature beats and tachycardia are common at low energy settings when the patient is heavily digitalized, and at high energy level settings even in the absence of any digitalis preparation. Should a ventricular dysrhythmia follow the first shock, an intravenous antidysrhythmic drug, as previously recommended, should be given before increasing to high energy settings.

**Pulmonary Edema and Increase in Heart Size**

These may occur in 3% of patients coming on within 1–3 hours of treatment.38 Unlike other major complications, they occur only in patients actually reverted to sinus rhythm. While Lown considers
pulmonary emboli as a possible cause, others believe that following electroversion there is considerable depression of the mechanical function of the left atrium. Any additional obstruction to flow across the mitral valve, any lessening of left ventricular wall compliance or left ventricular dysfunction will aggravate the situation and may result in pulmonary edema.

The incidence of complications relates to the energy level setting, being 6% at 150 joules but rising to exceed 30% at a setting of 400 joules. One can conclude, therefore, that there is rarely an indication for exceeding a 300-joules setting in patients presenting with long-standing rhythm disturbances, particularly atrial fibrillation of more than 5 years’ duration.

**Follow-up Studies**

While the initial success rate is remarkably good, direct current shock is disappointing in long-term follow-up studies, particularly when atrial fibrillation is treated. Reversion to atrial fibrillation is most likely by the end of the first month of electroversion, and not infrequently occurs within the first day. Those in whom atrial fibrillation has persisted for more than 3 years, those with significant underlying heart disease, or with considerable overall enlargement of the heart are particularly liable to revert to atrial fibrillation. Those with lone atrial fibrillation are exceptional; they do not remain in sinus rhythm even in the absence of these unfavorable circumstances.

**Quinidine and Other Antidysrhythmic Drugs**

Rossi and Lown reported that quinidine preceding direct current shock improves the chances of persistent sinus rhythm, diminishes the electric energy needed for the conversion, and reduces the incidence of postconversion rhythm disturbances. Studies such as these are difficult to control, and other groups have been unable to confirm their conclusion. Several groups, however, have investigated the use of drugs alone or in combination to maintain sinus rhythm. After transmyocardial DC shock, acetylecholine and catecholamines are liberated, and their combined effects may precipitate ectopic rhythms, permit their continuance, and either prevent the emergence of sinus rhythm or curtail its duration. Szekely reported that quinidine, procaine amide, and propranolol do not necessarily reduce the instance of DC shock-induced rhythm disturbances, except for those directly related to digitalis. Furthermore, their clinical and experimental observations suggest that the toxic cardiac effects of these drugs may even be enhanced by high energy electric current; they therefore advise caution in their routine use. More recently, quinidine bisulfate as a long-acting preparation has been extensively investigated in an attempt to maintain sinus rhythm following DC shock. Even when a long-acting preparation is controlled with adequate blood levels, sinus rhythm persists only for a slightly longer period of time, and furthermore, the incidence of complications with quinidine may still be high.

It can be concluded, nevertheless, that quinidine or some other antidysrhythmic drug should be considered for any patient who has reverted to his original rhythm disturbance, and in whom sinus rhythm is important. The chances of sinus rhythm persisting, however, are not good, even when adequate blood levels are maintained.

**Digitalis and Electroversion**

Lown, Kleiger, and Williams reported that in experimental animals the DC threshold for the emergence of ventricular tachycardia fell to 0.2 joules in the presence of heavy digitalization. In man, the counterpart of these observations is demonstrated by ventricular premature beats, tachycardia, or even fibrillation being precipitated by the DC shock in heavily digitalized patients. In the presence of a known digitalis-induced rhythm disturbance, electroversion should therefore rarely be used, for despite the very occasional record of a successful outcome, fatal ventricular fibrillation may follow the shock, even when properly synchronized.

**Anticoagulants and Electroversion**

The need for and efficiency of anticoagulant protection has been the subject of much debate, in the absence of any well-controlled study. Such a study has now been reported demonstrating a statistical benefit in a group of patients who had electroversion under anticoagulant control. Unless a contraindication to its use is present, all patients going forward to electroversion should be protected by anticoagulant therapy, particularly patients with recent cardiac infarction, chronic coronary heart disease, mitral valvar disease, cardiomyopathy, and prosthetic heart valves when a coumadin derivative should be given or heparin used if the indication for DC shock is urgent. As the risk of reversion to the original rhythm disturbance is highest within the first month of treatment, it is wise to maintain oral
anticoagulant therapy for at least 4 weeks after successful DC shock, when it may be discontinued unless the underlying heart disease requires its continuance.

Electric defibrillation of the heart, particularly synchronized DC shock, is an exciting advance but further effort is needed to uncover the basic mechanisms of rhythm disturbances, thereby encouraging the development of more physiologic approaches to their treatment and particularly to the maintenance of sinus rhythm thereafter.

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