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Treatment of Cardiac Arrhythmias with Synchronized Electrical Countershock

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ABSTRACT

Synchronized electrical countershock is an intriguing new method for the treatment of ectopic tachycardias. The authors applied this treatment to 20 patients with chronic atrial fibrillation and, in 17 patients, sinus rhythm was restored immediately. An additional four patients with atrial flutter were successfully converted to sinus rhythm. One patient developed a hemiplegia two weeks after cardioversion. No other untoward side effects were observed. In two patients with ventricular fibrillation electrical countershock terminated the arrhythmia. After successful cardioversion of atrial fibrillation, a maintenance dose of quinidine is given to help maintain sinus rhythm. In spite of this precaution, one-half of the patients reverted to atrial fibrillation within a month. The quinidine was administered for two to three days in advance of cardioversion; on this regimen, 10 of 34 patients reverted to sinus rhythm on quinidine alone and did not require countershock. The exact place of this treatment of cardiac arrhythmias has not yet been clearly defined.

A METHOD for correcting ectopic tachycardias with direct current shock has been recently described by Lown.[1] This method, which has been termed "cardioversion", has been used successfully in cases of atrial fibrillation, atrial flutter, some atrial tachycardias and some ventricular arrhythmias as well.

The principle is to deliver an electrical current across the heart muscle which stimulates (or depolarizes) all the heart muscle fibres at the same time. Following this, all the fibres are at rest together and the ectopic focus is abolished. Then, since the sinus node has the greatest inherent rhythmicity, it re-establishes itself as pacemaker for the heart. A suitable current is delivered through electrodes on the surface of the intact chest. It has been shown by Lown that serious ventricular arrhythmias may occur when an electric shock falls within a certain vulnerable phase of the heart cycle. This vulnerable period coincides with the end of the upstroke of the T wave in the electrocardiogram. For this reason, it is essential that the countershock be synchronized to fall within a safe part of the cycle. To ensure this, a synchronizer§ in the circuit delivers the impulse in response to the R wave of the patient's own electrocardiogram.

METHOD

Arrhythmias corrected by electrical countershock are prone to recur. For this reason, a maintenance dose of quinidine sulfate is usually prescribed and it is customary to begin this two or three days prior to cardioversion. The dose of quinidine is adjusted according to tolerance, and in this series it varied from .2 g. (3 grains) three times daily to .4 (6 grains) every eight hours.

The patient is attached to the apparatus by electrodes on the arms and legs and the patient's electrocardiogram (ECG) is visualized continuously on the built-in cathode-ray oscilloscope. To this is connected a direct-writing electrocardiograph to provide a permanent record of all events. Several test shocks are observed and recorded to ensure

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§Equipment used in this study was the Cardiac Synchronizer manufactured by Corbin Farnsworth, Inc., Palo Alto, Calif., U.S.A.

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that the discharge is properly synchronized within the safe phase of the heart cycle.

Before the electrode paddles are applied to the patient's chest, the patient is anesthetized with thiopental sodium. The electrode paddles are coated with a thick layer of electrode paste and held firmly on the patient's chest—one in the second intercostal space to the right of the sternum and the other in the fifth intercostal space in the left mid-axillary line. The trigger is not released until the ECG baseline is stable, since an artefact or ventricular ectopic beat with a tall T wave could conceivably trigger the shock during the dangerous phase of the heart cycle. To avoid accidental electrocution of the operator or other attendants, care is taken that no one is in contact with the patient or the surface of the electrode paddles. When the discharge occurs, there is a mild convulsive jerk of the patient's extremities. Following the shock, the electrocardiographic tracing is lost for two or three seconds, after which it returns to the screen. If the electrocardiogram shows a return of sinus rhythm, the patient is allowed to awaken. If not, the shock is repeated at a higher energy setting. The period of anesthesia is about five minutes for each shock. After conversion to sinus rhythm, the patient remains in hospital overnight and is discharged on a maintenance dose of quinidine and, usually, digitalis.

RESULTS

To date, 34 patients with atrial arrhythmias have been scheduled for cardioversion. Of these, 10 reverted to sinus rhythm on the quinidine administered prior to cardioversion (Table I). In two of these patients atrial fibrillation recurred in spite of maintenance quinidine therapy. Another man, aged 66, died suddenly two days after reverting to sinus rhythm on 0.4 g. of quinidine every eight hours. This patient suffered a femoral artery embolus two months earlier and was admitted four weeks prior to death with a mesenteric embolus requiring bowel resection. He had been receiving oral anticoagulants for three weeks before quinidine was begun. Permission for autopsy was denied, and the nature of the underlying heart lesion and cause of death remain unknown.

TABLE I.—PATIENTS REVERTING TO SINUS RHYTHM ON QUINIDINE PRIOR TO CARDIOVERSION

Type of arrhythmia		*No. of patients*
Atrial fibrillation		9
Coronary heart disease	3	
Mitral valve disease	3	
Hypertensive heart disease	1	
Postoperative atrial defect	1	
Idiopathic	1	
Atrial flutter (alcoholic)		1
		10

In two instances sinus rhythm was not induced by cardioversion (Table II). In one, the anesthetic was poorly tolerated and anesthesia was discontinued after only one stimulus. The second patient developed increasingly frequent ventricular extrasystoles and it was feared these might lead to an improperly synchronized shock. Immediately after one of the early countershocks, the electrocardiogram monitor recorded a straight line suggesting cardiac standstill. This provided a few anxious moments until it was recognized that one of the patient's electrodes had become detached. Subsequent to this incident, we have routinely fastened the electrode cables to the skin with adhesive tape to reinforce the usual rubber straps.

TABLE II.—RESULTS OF CARDIOVERSION IN 24 CASES OF ATRIAL ARRHYTHMIAS*

Achieved sinus rhythm		21
—later resumed fibrillation	10	
—still in sinus rhythm	11	
Failed to achieve sinus rhythm		3

*Average follow-up: four weeks.

From Table II it is apparent that the ectopic rhythm returned in half the patients in the first four weeks after successful cardioversion. This has occurred in spite of continued suppressive drug therapy. In two patients, vomiting and diarrhea developed (presumably from excessive quinidine dosage), and during the intestinal upset, quinidine treatment was interrupted and atrial fibrillation appeared. It is possible that a more individualized and more closely supervised program of quinidine therapy, with the help of quinidine blood levels, would reduce this effect in the future. In Table III the heart diseases underlying the arrhythmias are listed. It is seen that there was a high incidence of mitral valve disease in this group of patients. Eight of the 10 patients who had an early return of arrhythmia had mitral disease. It is known that it is more difficult to sustain sinus rhythm in these patients with left atrial enlargement and the large proportion of these patients may account for the relatively high proportion of our patients who failed to remain in sinus rhythm.

TABLE III.—TYPE OF ARRHYTHMIA IN 24 PATIENTS SUBJECTED TO ELECTRICAL COUNTERSHOCK

		No. of patients
Atrial fibrillation		20
—Rheumatic mitral valve disease	17	
—Coronary heart disease	2	
—Idiopathic	1	
Atrial flutter		4

Atrial flutter was the abnormal rhythm in four cases, and each responded to the initial low-energy discharge. In eight cases of atrial fibrillation, two or more countershocks at increasing energy levels were required to produce sinus rhythm. It was our impression that atrial fibrillation with coarse atrial activity in the electrocardiogram responded more consistently to the initial shock at the low setting (80 watt-secs.).

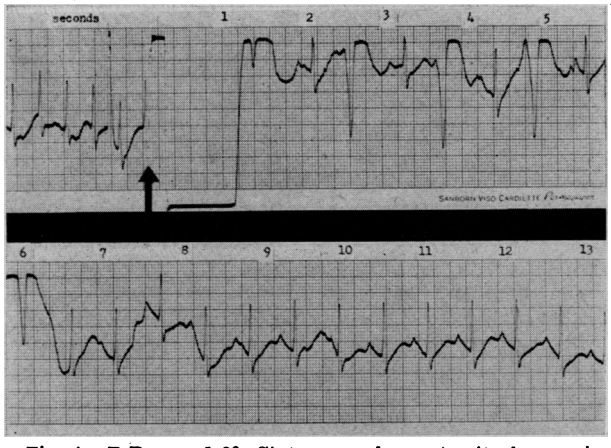

Fig. 1.—R.R., aged 39. Sixteen weeks post mitral commissurotomy. Rapid atrial fibrillation disrupted by countershock of 80 watt-seconds. Sinus rhythm apparent within two seconds but bigeminal rhythm observed until six seconds.

Although in many cases there was prompt restoration of sinus rhythm, on several occasions abnormal and bizarre P waves were seen with varying PR intervals in the first few minutes after countershock. Ventricular premature beats were also common at this stage (Fig. 1). This early instability of the heart rhythm has led us to allow at least four or five minutes for proper assessment of the response. Painless erythema at the site of electrode application is regularly seen, but this fades in a few hours. One of the patients in this series developed a hemiplegia two weeks after cardioversion. Others[1, 2] have encountered little difficulty with this procedure, and only two other systemic emboli have been reported.[3] Because of the possible risk of embolism in patients with atrial fibrillation and mitral valve disease, anticoagulants are given (usually on an outpatient basis) for two to three weeks preceding cardioversion and are continued for a few days thereafter.

In addition to its use in atrial arrhythmias, direct current countershock is applicable in cases of ventricular tachycardia and ventricular fibrillation. We have successfully treated one patient with ventricular fibrillation following acute myocardial infarction. In a second patient, who developed ventricular fibrillation after open-heart surgery for muscular septal hypertrophy, the arrhythmia did not respond to 30 successive internal applications of A.C. current but did respond to the second D.C. countershock applied externally. As yet, we have had no personal experience in the treatment of ventricular tachycardia by this means.

DISCUSSION

The indications for cardioversion are not yet well defined. Limitations of this method include the need for an anesthetic and the prolonged use of quinidine which involve both expense and the possibility of toxicity. Recurrence of the arrhythmia is frequent and in our experience occurred in half the patients within four weeks. Cardioversion was not required in 10 of 34 cases because quinidine in maintenance doses converted the arrhythmia. It is possible that if the quinidine is started farther in advance of cardioversion a higher proportion of patients will convert to sinus rhythm without countershock. At any rate, renewed interest in both immediate and long-term effects of quinidine will be a profitable dividend of this new form of treatment.

Cardioversion is a simple and effective method for correcting ectopic tachycardia. Although the method is apparently free of serious risk, occasionally serious arrhythmias develop,[3] and personnel and facilities for cardiac resuscitation should be close at hand before cardioversion is attempted. The results of this method are apparent at once, and prolonged hospitalization is avoided. It is our current practice to admit these patients to hospital for 24-hour observation following the procedure.

At present, indications for cardioversion appear to be the presence of atrial fibrillation with rapid ventricular rate despite digitalis, refractory heart failure associated with atrial fibrillation, and in most instances in which it is known that fibrillation is of recent onset.

The asymptomatic patient with fibrillation easily controlled by digitalis is less clearly a candidate for reversion. Cardioversion is especially satisfactory for the correction of atrial flutter. In ventricular tachycardia it should be tried early if initial medical treatment is not successful. In ventricular fibrillation the D.C. defibrillator is used without synchronization. Early experience suggests that it is preferable to A.C. defibrillation in this serious arrhythmia.

SUMMARY

Synchronized direct current countershock (cardioversion) is a satisfactory method of restoring normal sinus rhythm in patients with atrial flutter and atrial fibrillation. Successful conversion occurred in 21 of 24 patients treated by this method.

Aside from one patient who suffered a hemiplegia two weeks after this treatment, no complications were encountered in this series of cases.

The method described is simple and requires only brief anesthesia and a short period of observation in hospital.

Long-term quinidine therapy is apparently necessary to sustain sinus rhythm following cardioversion.

One-third of our patients converted to sinus rhythm when quinidine was given *prior* to cardioversion.

Nine of 17 patients reverted to atrial fibrillation within one month of initially successful cardioversion.

The exact role of cardioversion in the treatment of cardiac arrhythmias remains to be clearly defined. Some of the indications and limitations have been discussed.

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