Arrhythmias Related to Cardioversion

By L. Lemberg, M.D., A. Castellanos, Jr., M.D., J. Swenson, M.D., and A. Gosselin, M.D.

The use of electric current has proved to be an effective clinical method for terminating cardiac arrhythmias.\(^1\)\(^-\)\(^10\) External countershock with use of alternating current was introduced in 1956 by Zoll and co-workers\(^1\) for the treatment of ventricular fibrillation. In addition, these authors predicted the use of this procedure in terminating other arrhythmias as well. In recent years the use of direct current has been advocated by Lown and co-workers as a successful method of abolishing ventricular and supraventricular disorders of rhythm (cardioversion).\(^3\),\(^4\),\(^6\)

The growing importance of this recent therapeutic approach is attested by the increasing number of patients who have undergone successful electric countershock. It has been useful in terminating acute as well as chronic atrial fibrillation.\(^1\)\(^-\)\(^6\) The percentage of conversions of atrial fibrillation has been high, but at times reversion to sinus rhythm is only transient. Following conversion of atrial fibrillation there is an interval lasting several minutes which is considered highly significant because of frequent arrhythmias in this period. Some of these are able to induce recurrence of atrial fibrillation and thus are responsible for the transitory nature of reversions. The purpose of this report is to study the incidence of arrhythmias immediately after conversion and to corroborate the efficacy of this method of terminating atrial fibrillation. The mechanism by which these arrhythmias are able to induce the recurrence of atrial fibrillation, as well as the possibilities of preventing their appearance will be discussed.

Material and Methods

DC countershock was used in terminating 101 episodes of atrial fibrillation in 86 patients, their ages ranging from 17 to 86 years. Thirty-nine had atherosclerotic heart disease, 32 predominant or pure mitral stenosis, eight pure or predominant mitral insufficiency, and seven multiple valvular lesions. The procedure was performed on 13 occasions after cardiac surgery. In those with rheumatic heart disease, the diagnosis was made by cardiac catheterization and angiocardiography. All patients had cardiac enlargement and were compensated at the time of conversion. All but three were on maintenance doses of digitalis. They had been on anticoagulants for at least 10 days prior to conversion and none had experienced any episodes of embolization. The estimated duration of atrial fibrillation is shown in table 1. The patients early in the study had received very little (less than 1 Gm.) or no quinidine, but the last 27 were premedicated with 0.3 Gm. every 2 hours for four doses. Synchronized electric countershock of direct current \(^*\) was performed 6 to 8 hours after the first dose. The mechanism and function of this instrument, as well as the necessary precautions for its use have been described by Lown et al.\(^3\),\(^6\) The initial energy setting was 100 watt-seconds. With failure of conversion the setting was increased progressively to 150, 200, 300, and 400 watt-seconds. In the first 30 episodes of countershock maximal energy settings of 400 watt-seconds were not used because of brief duration of anesthesia. There were four cases of failure to convert the arrhythmia in this initial group.

Anesthesia was produced with methohexital sodium (Brevital), a short-acting barbiturate. The drug was diluted to 10 mg. per ml., and administered at a rate of 1 ml. per second. The total amount required per patient was 50 mg. but at times as much as 100 mg. were given. The patient was usually fully awake in 4 or 5 minutes. Routinely, the effects of the shock were observed on the oscilloscope, while a permanent graph was recorded on a standard electrocardiographic ma-

\(^*\) Cardioverter, manufactured by American Optical Company.
Table 1

<table>
<thead>
<tr>
<th>Duration of Atrial Fibrillation * (Episodes)</th>
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<tbody>
<tr>
<td>Less than 6 months</td>
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<tr>
<td>6 months-1 year</td>
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<tr>
<td>1-2 years</td>
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<td>2-5 years</td>
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<td>5-10 years</td>
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<td>More than 10 years</td>
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*In eight cases the exact duration of the arrhythmia could not be determined.

chine. Bipolar lead II or chest lead V₁ was used; the latter is least affected by the electric current or movements of the patient. All cases were continuously monitored for at least 15 minutes after the shock. Following conversion the patients were placed on maintenance quinidine therapy.

Results

Continuous electrocardiographic monitoring during conversion revealed that there was an interval following the shock during which a clear recording could not be obtained. It lasted from 2 to 5 seconds and was due to stylus wandering related to voltage overcharge of the electrocardiograph and also to thoracic muscle contractions and arm movements. DC countershock terminated 92 of the 101 episodes of atrial fibrillation in the 86 patients, an incidence of 91 per cent. Included in this group were two cases in which the arrhythmia was followed by an atrioventricular nodal rhythm and then recurrent atrial fibrillation without an intervening sinoatrial mechanism. The energies used in the successful cases are seen in table 2. Arrhythmias following countershock were classified according to whether they appeared before or after regular sinus rhythm developed (tables 3 and 4). Persistent atrial flutter (or atrial tachycardia) was seen following countershock of atrial fibrillation on four occasions (fig. 1) (table 3). These rapid paroxysms were reverted to sinus rhythm by an additional countershock. In 12 patients, atrial fibrillation was changed to atrioventricular dissociation with ventricular capture beats, the rate of both pacemakers being under 60 per minute, with sinus arrhythmia. Finally, in one patient, a passive atrioventricular nodal rhythm and an atrioventricular nodal tachycardia preceded the establishment of a regular sinoatrial mechanism (fig. 2). Several arrhythmias were noted immediately after conversion (table 4). Reversion to atrial fibrillation was observed six times and was probably triggered by premature atrial contractions in

Table 2

| Energies Employed in 92 Episodes Successfully Converted to Sinoatrial Rhythm |
|--------------------------------|---------|
| Watt-seconds | No. of episodes |
| 100          | 31       |
| 150          | 27       |
| 200          | 23       |
| 300          | 6        |
| 400          | 5        |

Table 3

<table>
<thead>
<tr>
<th>Arrhythmias Appearing after Countershock but Prior to Sinus Rhythm</th>
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<tbody>
<tr>
<td>Atrioventricular dissociation</td>
</tr>
<tr>
<td>Passive &quot;middle&quot; atrioventricular nodal rhythm</td>
</tr>
<tr>
<td>Atrioventricular nodal tachycardia</td>
</tr>
<tr>
<td>Atrial flutter or tachycardia</td>
</tr>
</tbody>
</table>

Table 4

<table>
<thead>
<tr>
<th>Arrhythmias Appearing after Conversion to Sinus Rhythm</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. quinidine</td>
</tr>
<tr>
<td>---------------</td>
</tr>
<tr>
<td>(74 episodes)</td>
</tr>
<tr>
<td>Atrial extrasystoles</td>
</tr>
<tr>
<td>Atrioventricular nodal extrasystoles or escapes</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
</tr>
<tr>
<td>Atrial flutter or tachycardia</td>
</tr>
<tr>
<td>Atrioventricular nodal tachycardia</td>
</tr>
<tr>
<td>Multifocal atrial arrhythmia</td>
</tr>
</tbody>
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Figure 1

Atrial fibrillation converted to atrial flutter (atrial rate: 230/minute) by DC current. Atrial shutter was abolished with additional countershock.

Figure 2

Atrioventricular nodal rhythm and nodal tachycardia appearing after countershock and before establishment of regular sinus rhythm.

Figure 3

Atrial fibrillation converted to sinus rhythm with atrial extrasystoles by DC countershock (upper strip). An atrial extrasystole reinitiated atrial fibrillation (lower strip) 5 minutes later.
four occasions (fig. 3) and by atrial flutter (rate of 300 per minute) two times (fig. 4). Rapid, ectopic, supraventricular disorders of rhythm were seen nine times (fig. 5). It was difficult to differentiate atrial flutter from atrial tachycardia in these paroxysms. For instance, in one subject with many runs of ectopic beats, the atrial rate varied from 135 to 270 per minute. Moreover, this as well as two others had irregular PP intervals within one single paroxysm. These could not be ascribed to ventriculophasic arrhythmia. The classic sawtooth appearance of atrial flutter was seen once when the atrial rate was only 167 per minute. No instances of ventricular tachycardia, ventricular fibrillation, or prolonged ventricular standstill were observed in the present series.

Discussion

The conversion rate of atrial fibrillation was 91 per cent, similar to that of Lown et al. (90 to 93 per cent)\(^6\,7\) and Killip (89 per cent)\(^10\). In four cases in which conversion failed, maximal energies of 400 watt-seconds were not used because of the brief anesthesia. The possibility of conversion with higher energies in these patients was not ascertained.

A review of the literature revealed a 71-per cent conversion rate with quinidine in atrial fibrillation of a total of 1,082 trials\(^11\). In another group of 500 cases treated by Goldman, successful reversions were obtained in 82 per cent.\(^11\) Yet, in judging the effectiveness of this drug in abolishing atrial fibrillation, one should consider the etiology of the underlying heart disease. A high incidence of success (80 to 87 per cent) was observed in atherosclerotic heart disease. Patients with predominant rheumatic mitral stenosis had a conversion rate of 50 to 55 per cent, and those with predominant mitral insufficiency, 20 to

![Figure 4](image1)

Atrial fibrillation converted to sinus rhythm with first-degree atrioventricular block by DC countershock (upper strip); reappearance of atrial fibrillation (preceded by a short run of atrial flutter) 7 minutes after cardioversion (lower strip).

![Figure 5](image2)

Paroxysmal atrial tachycardias appearing immediately after countershock. In the upper strip the paroxysm is initiated by a premature P wave different in configuration from those seen during the paroxysm. The primary (sinoatrial) pacemaker is depressed after the end of the tachycardia.
25 per cent. When these figures are compared with the results of electric countershock, 100 per cent of predominant mitral stenosis in Lown's cases and 91 per cent in the present group were converted to sinus rhythm. The efficacy of countershock and its superiority to quinidine are thus corroborated.

The incidence of arrhythmias before and after conversion was not negligible; they were transient, however, and did not jeopardize the lives of the patients. There were no serious ventricular arrhythmias in this series of elective patients when synchronized energies up to 400 watt-seconds were employed. Improper synchronization was responsible for the occurrence of ventricular fibrillation in one of Killip's cases and in one of our patients with ventricular tachycardia not included in this communication. The exact number of ectopic rhythms that may appear after countershock is not known because of the immediate brief interval of time during which a tracing is unobtainable.

Embolization was not observed in any of the patients. The only minor limitation of this procedure involves the unavoidable risks of anesthesia. It has been suggested that anesthesia is not required with energies less than 100 watt-seconds. A recent report of 14 cases of conversion without anesthesia indicates that the procedure can be employed with little or no discomfort.

The patients selected for conversion in this communication are by no means representative of the total population of the cases to be converted. Patients with refractory heart failure or with previous episodes of embolization were not selected. Similarly, there were few patients shocked after successful cardiac surgery, although these postoperative arrhythmias are easily terminated by countershock.

One of the basic problems encountered in this study was that of maintaining the sinoatrial mechanism. There were nine cases in which reversion was transient; in these, rapid tachycardias were frequent after countershock (table 4). Some were thought to be responsible for the recurrence of atrial fibrillation.

In many, the paroxysm appears to be initiated by a premature contraction that might have fallen in the vulnerable phase of atrial repolarization, thereby provoking atrial fibrillation (fig. 3), atrial flutter (fig. 4), or atrial tachycardia (fig. 5). This variable response in man is the counterpart of the experimental findings of Brooks et al., who found that if the strength of a stimulus applied during the vulnerable phase is increased gradually, either isolated extrasystoles, multiple responses, or paroxysms of flutter and fibrillation can occur.

The triggering of tachycardias by ectopic impulses is not rare in clinical electrocardiography. For example, premature atrial contractions preceding and probably initiating atrial flutter and atrial fibrillation have been reported. As stated previously, when the onset of an atrial tachycardia is recorded, the arrhythmia is initiated by a premature contraction. The first extrasystole of the paroxysm (fig. 5) may have a different configuration from the rest. Scherf et al. believed that this indicates that they arise in a different center, although aberrant retrograde conduction of the first beat cannot be definitely ruled out.

Some authors are of the opinion that paroxysmal atrial tachycardia is produced by a reciprocal rhythm involving the atrioventricular node. They have assumed that retrograde conduction of an atrioventricular nodal or ventricular extrasystole can use only part of the atrioventricular conducting system, thereby permitting alternate pathways for a reentry mechanism. Regardless of the fundamental mechanism responsible for rapid ectopic supraventricular rhythms, it is possible that a basic step in their prevention should be directed toward preventing or suppressing premature contractions. Atrial and ventricular extrasystoles are common but innocuous in the absence of organic heart disease; in atherosclerotic or rheumatic mitral valvular disease they may herald the onset of fibrillation or flutter.

It may be theorized that when incoordinate electrical activity of the atria has been
present for long periods of time, susceptibility to recurrent fibrillation is enhanced, not only immediately after conversion, but in later stages as well. For even though the ectopic rhythm was abolished, the factors that led to fibrillation (left atrial disease) still persist. This, of course, does not apply to rheumatic patients who had successful valvular surgery prior to countershock.

Precordication with quinidine prior to countershock would theoretically be expected to reduce the incidence of immediate postconversion arrhythmias by depressing ectopic atrial activity. In addition it has been shown that quinidine raises the threshold for atrial fibrillation in the experimental animal. In our series there were 27 patients who had received 1.2 Gm. of quinidine before countershock. In this group there were only five instances of postconversion arrhythmia. Atrial tachycardia and flutter occurred four times, and atrial fibrillation only once. The incidence of the latter rhythm was low when compared to that of patients who were not premedicated with quinidine (table 4). Two other patients were of interest in this respect. One had atrial flutter and the other atrial fibrillation in the immediate postconversion period. These arrhythmias did not occur when quinidine was administered (by the above method) prior to their second countershock. More experience is needed, however, to establish the most effective dose of quinidine to prevent the occurrence of arrhythmias appearing after conversion to sinus rhythm. On the other hand, it is probable that the mechanism of the ectopic beats appearing before sinus rhythm is different from that of arrhythmias following conversion to sinus rhythm. Atrioventricular dissociation or nodal rhythm can occur transiently after treatment of atrial tachycardia, atrial flutter, or fibrillation with quinidine or procaine amide. According to Katz and Pick, this is due to depression of the primary (and sometimes of the secondary) pacemaker by the ectopic rhythm per se, or by the antiarrhythmic drug. In this respect it should be emphasized that digitalis, which was administered to most of our patients, is a greater depressant of the sinoatrial node than quinidine. A more likely explanation is that a diseased sinoatrial node or a dilated left atrium is more prone to develop supraventricular arrhythmias after conversion. Two of the five cases with atherosclerotic heart disease in which a slow atrioventricular nodal rhythm appeared after countershock never developed sinus activity. Tracings obtained 6 hours later revealed recurrent atrial fibrillation. The transitional period was not recorded. These patients had not been premedicated with quinidine, and their maintenance doses of digitalis (0.25 mg. of digoxin daily) was not considered to be clinically excessive. It is thus likely that in these cases sinus node depression was not drug induced.

Four cases of atrial tachycardia or flutter appeared before sinus rhythm. Three instances were observed when atrial flutter followed atrial fibrillation, immediately after countershock. An interesting feature was observed in one case (from another series *) when atrial flutter with a rate of 240 per minute converted to an atrial rate of 107 per minute after countershock (fig. 6). The center of impulse formation of the lower rate was not the sinoatrial node, since a second shock converted this slower tachycardia to sinus rhythm with a change in atrial rate and P-wave configuration. It thus appears that two effective responses can be elicited by DC countershock in fibrillation or flutter. The first is more frequent, abolishes the arrhythmia, and establishes a sinus rhythm. The second is rare, and converts fibrillation into flutter or slows the atrial rate from that usually seen with flutter to that of atrial tachycardia. Thus progressive decrements in ectopic atrial rates can occur with countershock rather than the expected conversion to sinus rhythm. These findings lead to a possible explanation of the mechanisms by which external electric countershock acts in patients with

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* Thirty-nine episodes of atrial flutter have been treated in our department with 100-per cent conversion to sinus rhythm.

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![Graph](image)

**Figure 6**

Atrial flutter (rate 230 per minute) converted to an ectopic atrial tachycardia (rate 105 per minute). Second countershock established sinus rhythm.

Atrial fibrillation. If the intensity is sufficient to excite all fibers at a given instance, the arrhythmia can be arrested, and the sinus node assumes its role as pacemaker. If the intensity of the shock is weak and thus insufficient to depolarize the atria completely, fibrillation can persist. At times, however, incomplete depolarization of the atria by lesser intensities may result in a lengthening of the circulating impulse, thereby producing slower atrial rates (figs. 1 and 6). Therefore it appears that the transition of fibrillation to flutter with quinidine therapy as well as the reduction in the rate of flutter with added increments of quinidine is a pharmacologic effect that can be imitated in electric countershock.

**Summary**

DC countershock abolished 92 of 101 episodes of atrial fibrillation in 86 patients, an incidence of 91 per cent. Supraventricular arrhythmias were not infrequent immediately after countershock. These were transient and did not complicate the procedure nor were they hazardous to the patient. On two occasions a slow atrioventricular nodal rhythm appeared followed by recurrence of atrial fibrillation a few hours later. Sinus node activity did not return in these patients. Ventricular tachycardia, fibrillation, or standstill did not occur in this series.

The following arrhythmias were observed immediately after countershock but prior to the establishment of a regular sinus rhythm: atrioventricular dissociation, 12 times; passive atrioventricular nodal rhythm, five times; atrioventricular nodal tachycardia, five times; atrial flutter or tachycardia, four times.

The conversion of fibrillation to flutter by countershock implies that the effect of the electric current was merely to shorten the length of the circulating wave, a phenomenon that can also be observed in the treatment of atrial fibrillation with quinidine.

Disorders of rhythm, probably having a different mechanism, were also observed after countershock had established a regular sinus pacemaker. In the group not treated with quinidine prior to countershock (74 episodes) atrial extrasystoles were seen 26 times (35 per cent); atrioventricular nodal extrasystoles or escapes, 18 times (24.3 per cent); atrial flutter or tachycardia, six times (8.1 per cent); atrial fibrillation, eight times (18 per cent); atrioventricular nodal tachycardias, twice (2.6 per cent); and a bizarre, multifocal atrial arrhythmia, once (1.3 per cent). The arrhythmias considered to be responsible for the recurrence of atrial fibrillation and also the possible mechanisms involved were discussed.

Pretreatment with quinidine was effective in reducing the incidence of arrhythmias oc-
curring after conversion (27 episodes), atrial extrasystoles six times (22 per cent), atrioventricular nodal extrasystoles or escapes six times (22 per cent); atrial flutter or tachycardia three times (11 per cent); atrial fibrillation once (3.7 per cent); and atrioventricular nodal tachycardia once (3.7 per cent).

References

The Pulse Rate

Although the ancient Egyptians, according to the Ebers papyrus (1600 B.C.), paid attention to the pulse, and the Chinese in the fifth century B.C. attached great importance to the characters of the pulse, of which they recognized three thousand varieties, and Herophilus (300 B.C.) of Alexandria counted the pulse with his water clock or clepsydra, its rate apart from its other characters did not attract any general interest in Europe until long after Harvey's time.—Sir HUMPHRY DAVY ROLLESTON. The Harveian Oration. Great Britain, Cambridge University Press, 1928, p. 81.
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