Prognosis of Atrial Arrhythmias Treated by Electrical Counter Shock Therapy
A Three-Year Follow-Up

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Since the introduction of closed chest synchronized DC shock treatment for atrial fibrillation by Lown in 1962 (Lown, Amarasingham, and Newman, 1962a; Lown et al., 1962b) many thousands of patients have undergone this treatment. Complications have occurred on occasion. Arrhythmias, some fatal, have been reported by Ross (1964), Morris et al. (1964), Killip (1963), and Turner and Towers (1965). In addition, Lown, et al. (1963), Rabbino, Likoff, and Dreifus (1964), Oram and Davies (1964), and Navab and La Due (1965) reported that arterial emboli might occur. The incidence of myocardial damage has been documented by Sussman, Woldenberg, and Cohen (1964), Turner and Towers (1964), and Resnekov and McDonald (1965, 1967). Pulmonary oedema has been reported by Resnekov and McDonald (1965), Palomeho (1965), and Honey, Nicholls, and Towers (1965); hypotension by Turner and Towers (1965); and the emergence of digitalis toxicity after conversion by Gilbert and Cuddy (1965). However, the over-all incidence of serious side effects or permanent sequelae is very low. Recurrence of the arrhythmia is frequent.

In the following paper the results, complications, and follow-up of 149 patients defibrillated between May 1964 and July 1967 are presented.

Subjects and Methods

The majority of patients were from the United Bristol Hospitals, but some were admitted from outlying hospitals. After treatment they were followed up at six-monthly intervals. Thus the follow-up period varies from 6 months to 3½ years. The results may best be analysed with the patients divided into aetiological groups.

A Cardiac Recorders external-internal DC defibrillator was used in all cases. Energy settings of 60, 120, and 200 joules were used for conversion. Only very occasionally was an energy setting higher than 200 joules used. General anaesthesia was administered in all cases, with thiopentone and nitrous oxide being the agents preferred. Occasionally methohexitone was substituted for thiopentone. No antiarrhythmic drugs were administered either before or after conversions. All patients with a history of emboli were anticoagulated, as were the great majority of those with mitral valve disease. Only a minority of the other patients were on anticoagulants.

Results

Sinus rhythm was restored in 123 patients (83%). However, a true appreciation of the results may be gained from the various divisions in the Table.

Of 66 patients with mitral stenosis who were treated, 48 had atrial fibrillation before their operation; in the other 18 it was a post-operative complication. Sinus rhythm was restored in 52 patients, but in 29 of these the arrhythmias had recurred 6 months later. In a further 4, atrial fibrillation recurred within a year and in 1 more after 1 year. Only 10 patients persisted in sinus rhythm for more than a year or more. Of 44 patients followed for 2 years or more, 5 remain in sinus rhythm. The question might be asked whether long-term successes would have been more frequent had the patients been defibrillated at a longer interval after their operation. The series does include 7 patients who were treated on a second occasion some long time after their operation when atrial fibrillation had recurred.

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after successful treatment. Of these 7, 6 reverted to atrial fibrillation a second time, and this experience suggests that repeated defibrillation is not worth while.

In all, 34 patients without evidence of underlying heart disease were treated, 29 men and 5 women. Of these, 12 were treated for atrial flutter and 22 for atrial fibrillation. All but 1 of the 12 with flutter converted to sinus rhythm at an energy setting of 60 joules: the one exception required a shock of 120 joules. Of the 12 patients, 5 were in sinus rhythm at the last follow-up; 2 had required a second treatment, and 1 of these had reverted to atrial flutter for a third time. Of the 22 patients with atrial fibrillation, 4 failed to convert, and in 2 of these energy settings of 300 joules were used. Of the 18 patients who were successfully restored to sinus rhythm, sinus rhythm persisted for 1 year in 6, and for 2 years in 3 of the 16 followed for that length of time.

Thirteen patients with controlled thyrotoxicosis were defibrillated, 7 men and 6 women. Of these, 11 reverted to sinus rhythm and 2 did not. Of the 11, 2 were found to be fibrillating at follow-up. Of the 9 successes, 7 have been in sinus rhythm for more than one year; 3 of the 9 successful patients required shocks of 200 joules or more.

Nine patients with an arrhythmia due to cor pulmonale were defibrillated; 7 of these had atrial flutter and 2 had atrial fibrillation. All reverted to sinus rhythm; 7 of these were in sinus rhythm at follow-up for more than one year, only one had reverted to the previous arrhythmia. One patient in this group required a shock of 200 joules; all the others converted at 120 joules or less.

Seven patients with atrial septal defect were treated. Of these, 3 patients had atrial fibrillation and 3 had flutter, the remaining patient had both rhythms on occasions. Of these 7 patients, 6 converted to sinus rhythm after a shock of less than 200 joules; 3 patients were in sinus rhythm at follow-up; only 1 of these had been followed up for more than one year. The patient who failed to convert in this group had an ostium primum defect.

Four patients who had had an aortic Starr Edwards valve inserted were defibrillated. All converted; these 4 have remained in sinus rhythm for more than one year.

Five patients with hypertension were treated; 3 were successful and 2 failed, 2 remaining in sinus rhythm for more than one year, and 1 reverting to atrial fibrillation.

Five patients with ischaemic heart disease were treated; 3 converted to sinus rhythm and 2 failed.

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TABLE

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>Age range (yr.)</th>
<th>Rhythm</th>
<th>Total No. of cases</th>
<th>No. of men</th>
<th>No. of women</th>
<th>No. of successes</th>
<th>No. of failures</th>
<th>No. in sinus rhythm at end of 1 year</th>
<th>No. in sinus rhythm at end of 2 years</th>
<th>Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral stenosis (post-valvotomy)</td>
<td>24–72</td>
<td>Atrial fibrillation</td>
<td>66</td>
<td>16</td>
<td>50</td>
<td>52 (79%)</td>
<td>14</td>
<td>12 (18%)</td>
<td>5 (11%) followed by 3 (33%)</td>
<td>1 cerebral embolus 1 arrhythmia</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>26–73</td>
<td>Atrial fibrillation</td>
<td>22</td>
<td>20</td>
<td>2</td>
<td>19 (90%)</td>
<td>5</td>
<td>6 (50%)</td>
<td>11 (50%)</td>
<td></td>
</tr>
<tr>
<td>Thyrotoxicosis (treated)</td>
<td>50–60</td>
<td>Atrial fibrillation</td>
<td>13</td>
<td>7</td>
<td>6</td>
<td>11 (85%)</td>
<td>2</td>
<td>7 (9 followed up)</td>
<td>24 followed by 11 (50%)</td>
<td>1 cerebral embolus 1 arrhythmia</td>
</tr>
<tr>
<td>Cor pulmonale</td>
<td>56–72</td>
<td>Atrial fibrillation</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>2 (100%)</td>
<td>0</td>
<td>0 (100%)</td>
<td>6 (7 followed up)</td>
<td></td>
</tr>
<tr>
<td>Atrial septal defect</td>
<td>5 mth–55</td>
<td>Atrial fibrillation</td>
<td>3</td>
<td>3</td>
<td>0</td>
<td>3 (100%)</td>
<td>6</td>
<td>1 (20%)</td>
<td>1 (20%)</td>
<td>1 death 2 days after defibrillation</td>
</tr>
<tr>
<td>Aortic valve replacement</td>
<td>22–65</td>
<td>Atrial fibrillation</td>
<td>4</td>
<td>4</td>
<td>0</td>
<td>4 (100%)</td>
<td>0</td>
<td>4 (100%)</td>
<td>0 (100%)</td>
<td></td>
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<tr>
<td>Hypertension</td>
<td>51–62</td>
<td>Atrial fibrillation</td>
<td>5</td>
<td>4</td>
<td>1</td>
<td>3 (100%)</td>
<td>2</td>
<td>2 (100%)</td>
<td>2 (100%)</td>
<td></td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>44–65</td>
<td>Atrial fibrillation</td>
<td>5</td>
<td>5</td>
<td>0</td>
<td>3 (100%)</td>
<td>2</td>
<td>2 (100%)</td>
<td>2 (100%)</td>
<td></td>
</tr>
<tr>
<td>Cardiomyopathy</td>
<td>26–51</td>
<td>Atrial fibrillation</td>
<td>5</td>
<td>4</td>
<td>1</td>
<td>4 (100%)</td>
<td>1</td>
<td>3 (100%)</td>
<td>1 (25%)</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td>149</td>
<td>81</td>
<td>68</td>
<td>123 (83%)</td>
<td>27</td>
<td>18 (18%)</td>
<td>6 (4%)</td>
<td></td>
</tr>
</tbody>
</table>
Of these, 2 have remained in sinus rhythm for more than one year, and 1 has reverted to fibrillation.

Six patients with cardiomyopathy underwent treatment and 5 converted to sinus rhythm. Of these, 3 remained in sinus rhythm for more than one year; 2 have reverted to atrial fibrillation.

**DISCUSSION**

The results obtained in this series of patients show again the effectiveness and relative safety of terminating attacks of atrial fibrillation or flutter by the electrical method. When digitalization fails to restore sinus rhythm in patients with these arrhythmias we have no doubt that electrical treatment is to be preferred to the use of quinidine or other anti-arrhythmic agents. Previous work (Gilbert and Cuddy, 1965) has shown that it is essential to omit digitalis for at least 24 hours before conversion. This prevents the emergence of slow arrhythmias due to over-digitalization, which may not have been evident in the presence of fibrillation or flutter.

**Complications.** In contrast to most other reported series we have used lower energy settings, and have only exceeded 200 joules on 22 occasions. This may well account for the fact that we have not observed any incident of severe hypotension, pulmonary oedema, or evidence of myocardial damage as recorded by the electrocardiogram after conversion. Two dangers must always be borne in mind at the time of the rhythm change. The first is the emergence of some other arrhythmia; the second is the occurrence of systemic emboli. It must be assumed that the one death in this series was due to the emergence of an arrhythmia (atrial flutter) two days after successful restoration of sinus rhythm, as there was no other explanation to be found at necropsy; an atrial septal defect of ostium secundum type was present. There is no way of telling the relation between his electrical treatment and subsequent death. Three other patients in the series developed arrhythmias after treatment. There were two incidents of nodal rhythm which caused no serious haemodynamic disturbances, and one other patient with an atrial septal defect developed a slow atrial arrhythmia which reverted to atrial flutter two months after defibrillation. We had no instances of ventricular fibrillation such as those reported by Resnekov and McDonald (1967), or Ross (1964). It is noteworthy that Lown (1967) had no single episode of ventricular fibrillation or asystole in a series of 1088 shocks. Two of our patients developed systemic embolism in the days immediately after successful defibrillation. Neither of these patients had mitral valve disease and neither had previously been anticoagulated. Both made a good recovery.

**Analysis of Results.**

(i) **Mitral stenosis.** Previous authors have shown that most patients with mitral stenosis and atrial fibrillation can be restored to sinus rhythm if sufficiently high voltages are used (Kahn et al., 1967; Reale, 1965). Age does not influence results, and success is particularly likely if atrial fibrillation has not been present for long periods of time (Bell, Pugh, and Dunn 1967). Our experience is the same. The mean age of those who converted was $44 \pm 8$ (p = 0·05) years, and of those who failed $49 \pm 8$ (p = 0·05) years. Similarly, of those who failed to convert all but one had been in atrial fibrillation for more than one year. On the other hand, the majority of patients reverted to atrial fibrillation, and of the 52 in our series in whom sinus rhythm was successfully restored, fibrillation had restarted in 40 before the end of one year. Of the 44 patients followed for more than two years, 5 (11%) remain in sinus rhythm. Selzer et al. (1965) had a similar long-term success rate (21%) in patients who had been fibrillating for some time before valvotomy.

One would hope that the 12 patients who maintained sinus rhythm for one year would give some indication of the factors that are favourable for restoring and maintaining sinus rhythm. An analysis of the cases shows some pointers in this direction. Thus, of the 12 successes, 7 were from the group of 18 who developed fibrillation after operation while 5 had developed fibrillation for varying periods before their operations (2, 3, 5, 12, and 18 months). Age was not an important factor. It is perhaps noteworthy that the 12 long-term successes all returned to sinus rhythm with currents of 120 joules and less.

It remains, therefore, very difficult to make a firm recommendation about the use of cardioversion for the treatment of atrial fibrillation after valvotomy. Clearly, the main considerations are twofold: does the patient gain important haemodynamic benefit from being in sinus rhythm, and is the incidence of systemic embolism diminished by the presence of sinus rhythm? Kahn et al. (1967) found that the cardiac output was increased by 22 per cent after the establishment of sinus rhythm in a series of patients who had undergone a mitral valvotomy. Reale (1965) found that the increase in cardiac output varied, but on average the increase was only 7 per cent. He considered that the response to exercise was considerably enhanced. However, the advan-
tages of sinus rhythm in mitral stenosis are not accepted by all authorities. The haemodynamic benefit of sinus rhythm is not likely to be very important in this context, and in our view this consideration would not justify repeated attempts to restore sinus rhythm by electrical treatment under anaesthesia. Similarly, we have failed to show any important gain with regard to systemic emboli. Though we have followed 66 patients with mitral stenosis for a mean period of two and a quarter years, only one has developed systemic embolism during the period of observation. This one incident occurred in a patient who had been successfully converted to sinus rhythm and then reverted to atrial fibrillation. It is clear, however, from our study that sinus rhythm is more likely to be restored in patients in whom atrial fibrillation has not been present for more than a year before valvotomy, and our present practice is, therefore, to offer electrical treatment to these patients, with the knowledge that only a minority will continue in sinus rhythm. High energy loads should probably not be employed.

(ii) Aortic valve replacement. The 4 patients in this series who developed atrial fibrillation as a complication of aortic valve replacement all converted successfully and all remained in sinus rhythm. It is clearly in this sort of situation that electrical termination of atrial fibrillation is most strongly indicated.

(iii) Atrial septal defect. Only 7 patients with atrial septal defect have been treated. In 6 of the 7, sinus rhythm was restored with energy settings of less than 200 joules. Nevertheless, we have some reservation about the wisdom of the use of electrical treatment to arrest supraventricular arrhythmias in this condition. The one death in the series was in this group, and the most likely explanation of his death was the probable re-emergence of atrial flutter, perhaps with a 1:1 ventricular response. In one other patient a slow atrial arrhythmia which reverted to atrial flutter only after two months gave rise to some concern. In another two the arrhythmia began again.

(iv) Treated thyrotoxicosis. The results in this group clearly indicate that termination of atrial fibrillation by electrical treatment is reasonable and correct.

(v) Cor pulmonale. The feature of this group is that 7 of the 9 patients had atrial flutter. The results confirm the value in this group.

(vi) Ischaemic heart disease, hypertension, and cardiomyopathy. The numbers treated in each of these groups are all small, and generally our feeling is that little is to be expected from termination of atrial fibrillation in these patients.

(vii) Idiopathic heart disease. The criteria for including a patient in the idiopathic group were as follows: (a) All denied chest pain under any circumstances. (b) None had evidence of valvular disease or hypertension. (c) None had detectable enlargement of the heart on x-ray. (d) None had the electrocardiographic features of ischaemic heart disease. The fact that the majority of these patients were men suggests that ischaemic heart disease may perhaps be responsible for the arrhythmia, but we are impressed by the fact that in the three-year follow-up none of these patients have developed either the symptoms or the electrocardiographic changes of ischaemic heart disease. It does, therefore, seem possible that this arrhythmia may represent a disturbance of the excitatory tissue of the atrium, and that generalized ischaemia may not be the commonly precipitating factor. Ageing must in part be responsible. There may be a parallel here with the aetiology of atrioventricular block, which has been shown not to be commonly associated with extensive coronary artery disease (Smith and Zoob, 1961).

The results in our series differ conspicuously from those of Resnekov and McDonald (1967) who report 33 patients with lone fibrillation, of whom only 4 remained in sinus rhythm for a significant length of time after conversion. Half of our patients followed for one year or more have remained in sinus rhythm. These results are closer to those of Radford and Evans (1968) who had a 25 per cent two-year maintenance of sinus rhythm after conversion in a group of 20 lone fibrillators.

Age did not appear to influence results. The mean age of those who maintained sinus rhythm for a year or more was 54 ± 7 (p = 0.05), and the mean age of those who reverted to atrial fibrillation in less than one year was 52 ± 14.

Again it is difficult to decide how much benefit the patients have received from this form of treatment. In general, they all claim a sense of well-being when they change from atrial fibrillation to sinus rhythm. Systemic embolism in patients without underlying heart disease is a comparatively rare event. The most serious embolism in the whole series occurred in one of these patients before treatment, but in the follow-up period the only systemic embolism that occurred was in one patient three days after successful restoration of sinus rhythm. In the follow-up period, none of the
patients whose arrhythmia had restarted had in any important way become unwell, and it therefore remains an open question whether the restoration of sinus rhythm in these patients is worth while.

**CONCLUSIONS**

Electrical countershock therapy is a highly effective method for arresting atrial fibrillation or atrial flutter. Important complications are few provided relatively low energy settings are used. Further prolonged follow-up is necessary to determine the value of the treatment in the different conditions in which the arrhythmia is found.

**SUMMARY**

A series of 149 patients with atrial arrhythmia subjected to DC electroconversion is reviewed. Of these patients, 83 per cent converted to sinus rhythm, emphasizing the effectiveness of the procedure. The treated patients were followed up for varying periods of 6 months to 3 years. It is shown that patients with cor pulmonale, treated thyrotoxicosis, and Starr-Edwards aortic valves are eminently suitable for the procedure. The immediate results achieved in patients who have had mitral valvotomies are good, 79 per cent converting, but follow-up showed that only 19 per cent of those followed for one or more years maintained sinus rhythm. In those with idiopathic atrial fibrillation or flutter, immediate results are excellent, and of 21 followed up for one year or more 11 were in sinus rhythm. However, it is emphasized that further long-term follow-up is necessary in these patients, as it is in those with mitral stenosis, if definite recommendations as to the value of conversion are to be made. Serious complications occurred in only 3 patients.

**REFERENCES**


Prognosis of atrial arrhythmias treated by electrical counter shock therapy. A three-year follow-up.

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