Cardioversion Following Open-heart Valvular Surgery

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At the turn of the century, Prevost and Battelli (1899) observed that direct application to the dog's heart of a strong current of short duration stopped ventricular fibrillation. More than 30 years later, Hooker, Kouwenhoven, and Langworthy (1933) began a series of experiments utilizing an alternating current directly to the animal heart. This and subsequent studies (Ferris et al., 1936; Wiggers, 1940, etc.) laid the basis for successful defibrillation of a human heart by Beck, Pritchard, and Feil in 1947. However, it was not until 1956 that external defibrillation became a practical procedure (Zoll et al., 1956). And it is since the initiation by Lown, Amarasingham, and Neuman into the use of a synchronized direct current in 1962 that the procedure has gained a wide popularity.

The present paper is concerned with our experience with cardioversion following open-heart surgery.

SUBJECTS AND METHOD
The series consists of 26 subjects, 9 men and 17 women, ranging from 21 to 58 years in age.

All procedures were carried out in the post-absorptive state. Equipment for resuscitation was available in the room. An intravenous drip of 5 per cent glucose in water was started, and sodium pentothal solution (total dose ranging from 150 to 600 mg.) was injected through the tube until shallow unconsciousness was achieved. A Lown cardioverter (American Optical Company) was tested initially for synchronization of the peak of the “R” wave and the electrical discharge. After the initial setting of either 50, 75, or 100 watt seconds, the first electric shock was delivered. If this was unsuccessful, the following shock was given with an increment of 100 watt seconds. In the later 16 cases, 13 were successfully converted to sinus rhythm with only a single shock of 100 watt seconds.

Complete electrocardiographic tracings were obtained before and after the electrical shock. A long lead II or V1 was taken beginning just before and ending usually 30 seconds after the electrical shock. A longer strip was taken when ectopic rhythm was evident.

All patients were observed under electrocardiographic monitor for 15 minutes, during which time recovery from the anaesthetic usually took place.

For maintenance of normal sinus rhythm, quinidine sulphate was given in a dose of 0·2 to 0·3 g. four times a day, and quinidine gluconate 5 gr., was given at night before retiring. This treatment was started a day before cardioversion in the later cases of the series.

RESULTS
There were 25 patients with atrial fibrillation and one with atrial flutter. As shown in Table I, over half of them had pure or predominant mitral stenosis. There were 11 patients with Starr-Edwards valves, 10 mitral, and 1 aortic; and of the 10 with mitral prosthetic valves, there were 4 with mitral insufficiency and 6 with mitral stenosis with heavily calcified mitral valves.

The duration of the arrhythmia before cardioversion ranged from 24 hours to as long as 10 years. In 8 patients the duration was under 1 year and in 18 over 1 year. The interval between open-heart surgery and cardioversion ranged from 24 hours to 35 months. Cardioversion was performed within 3 weeks after operation in 13, 3 weeks to 12 months in 8, and 12 to 35 months in 5 patients.

Congestive heart failure was absent in all.
**TABLE I**

<table>
<thead>
<tr>
<th>Lesions</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pure or predominant MS</td>
<td>14</td>
</tr>
<tr>
<td>Pure or predominant MR</td>
<td>4</td>
</tr>
<tr>
<td>Predominant AS</td>
<td>2</td>
</tr>
<tr>
<td>Mixed valvular</td>
<td>6</td>
</tr>
<tr>
<td>Total</td>
<td>26</td>
</tr>
</tbody>
</table>

MS, mitral stenosis; MR, mitral regurgitation; AS, aortic stenosis.

**TABLE II**

<table>
<thead>
<tr>
<th>Grading</th>
<th>Over-all heart size</th>
<th>Left atrium</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>1+</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>2+</td>
<td>12</td>
<td>13</td>
</tr>
<tr>
<td>3+</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>4+</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>26</td>
<td>26</td>
</tr>
</tbody>
</table>

**TABLE III**

<table>
<thead>
<tr>
<th>Case No</th>
<th>Age (yr.)</th>
<th>Sex</th>
<th>Lesion</th>
<th>Duration of AF (yr.)</th>
<th>Interval*</th>
<th>Embolism</th>
<th>Chest radiograph</th>
<th>Prosthetic valve</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>37</td>
<td>F</td>
<td>MR</td>
<td>10</td>
<td>9 mth.</td>
<td>Pre- and post-op.</td>
<td>3+</td>
<td>Mitral</td>
</tr>
<tr>
<td>20</td>
<td>46</td>
<td>F</td>
<td>MR</td>
<td>7</td>
<td>2 mth.</td>
<td>Pre- and post-op.</td>
<td>2+</td>
<td>Mitral</td>
</tr>
<tr>
<td>29</td>
<td>58</td>
<td>M</td>
<td>MR</td>
<td>7</td>
<td>11 dy.</td>
<td>None</td>
<td>3+</td>
<td>Mitral</td>
</tr>
</tbody>
</table>

* Interval between open-heart surgery and cardioversion.
AF, atrial fibrillation; MR, mitral regurgitation.

A history of systemic embolization before cardioversion was found in 8, namely, 6 pre-operative and 2 pre- and post-operative. The latter 2 failed to respond to cardioversion. One other patient developed a cerebral embolism 4 months after a successful cardioversion. It is not certain whether the patient was in atrial fibrillation at the time of the embolic episode. Subsequent follow-up revealed the patient in sinus rhythm.

The heart size was radiographically evaluated in terms of left atrial and over-all cardiac enlargement (on the basis of 0 to 4 plus). Slight to moderate enlargement was present in the majority (Table II).

Before cardioversion, digitalis was given to all patients and quinidine to 14. Of the latter, 3 attained temporary conversion.

Anticoagulants were being taken by 12 patients at the time of cardioversion: a history of systemic embolization was present in 7 of them.

Thirty cardioversions were performed on 26 subjects, 4 of whom had 2 procedures. An average of 1.6 electric shocks was given on each cardioversion. The electrical energy required for each shock ranged from 50 to 400 watt seconds, the majority being under 200 watt seconds, with maximal distribution at 120 watt seconds.

The procedure was successful in 25 out of 30 attempts at conversion (83%) and in all but 3 patients (88%). No further attempt was made for these 3 failures (Table III). Follow-up data were available in 18 among 23 patients who responded to cardioversion (Table IV). Of these 18, 10 had no known recurrence of arrhythmia; 3 showed recurrences of arrhythmia, which were terminated by recardioversion in 1 and by an increase in quinidine dosage in 2; recardioversion was not attempted in the remaining 5 recurrences. Two patients showed quinidine intolerance. One patient developed recurrent atrial fibrillation even though the quinidine dosage was increased to 0.4 g. four times a day in addition to quinidine gluconate 5 gr. daily. One patient had a history of two successful courses of

**TABLE IV**

<table>
<thead>
<tr>
<th>Outcome</th>
<th>No. of cases (26)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Failure</td>
<td>3 (12%)</td>
</tr>
<tr>
<td>Success</td>
<td>23 (88%)</td>
</tr>
<tr>
<td>Follow-up data available</td>
<td>18</td>
</tr>
<tr>
<td>In sinus rhythm</td>
<td>13</td>
</tr>
<tr>
<td>No recurrence</td>
<td>10</td>
</tr>
<tr>
<td>Required recardioversion</td>
<td>1</td>
</tr>
<tr>
<td>Required increase in quinidine dosage</td>
<td>2</td>
</tr>
<tr>
<td>In arrhythmia (no further attempt of conversion)</td>
<td>5</td>
</tr>
<tr>
<td>Follow-up data not available</td>
<td>5</td>
</tr>
</tbody>
</table>
quintidine therapy over two years before cardioversion, yet had recurrent atrial fibrillation, even on adequate maintenance dose of quintidine. The remaining patient was not available for treatment (Table V).

Immediate post-cardioversion electrocardiograms were available in 22 patients, of whom 20 were successfully converted to sinus rhythm. Among these 20, 7 revealed neither ectopic rhythm nor conduction defect, while the remaining 13 had either or both (Table VI, and Fig. 1, 2, and 3). The ectopic rhythm, in most instances, disappeared in about 15 minutes. First degree A-V block persisted in all affected cases for as long as 7 months. In one, nodal rhythm dominated the initial 7 seconds after the electrical discharge, followed by the appearance of sinus mechanism. The latter could not be maintained due to slowing of sinus discharge culminating in nodal escape. The rhythm fluctuated between nodal rhythm, sinus rhythm, and atrial fibrillation and finally settled to the latter (Fig. 4).

There was no embolic phenomenon related to cardioversion. The only untoward effects of the procedure were a mild apnoea following intravenous administration of sodium pentothal in one, and erythema over the areas of electrode application in almost all patients.

**DISCUSSION**

The benefit of conversion of atrial fibrillation to sinus rhythm is manifold. Wetherbee, Brown, and Holzman (1952) demonstrated less increase in the ventricular rate after exercise in sinus rhythm than in atrial fibrillation. Kory and Meneely (1951) and Broch and Müller (1957) observed an increase in cardiac output following conversion of atrial fibrillation to sinus rhythm. And since the association of atrial fibrillation with thromboembolism is much more frequent than that of sinus rhythm with the latter (Hay and Levine, 1942; Jordan, Scheifley, and Edwards, 1951; Daley et al., 1951; Fraser and Turner, 1955), the incidence of systemic embolism may be expected to decrease after conversion to sinus rhythm.

Where systemic embolism is associated with mitral stenosis, its incidence is reduced following mitral commissurotomy (Greenwood, Aldridge, and

![Fig. 1.—Establishment of sinus rhythm following the shock. No ectopic rhythm or A-V block.](http://heart.bmj.com/)}
McKelvey, 1963). In the case of mitral valve prosthesis, however, embolism has been shown to occur in significantly high percentage (Effler, Favaloro, and Groves, 1965). The inherent structure of the prosthetic valve probably contributes considerably in the causation of the embolism. The presence of atrial fibrillation undoubtedly adds to the risk of embolism. Even for this reason only, conversion to sinus rhythm is worth while after insertion of the prosthetic valve.

The treatment of atrial fibrillation has been much facilitated since the introduction of the direct current countershock by Lown et al. in 1962. The treatment designated as "cardioversion" has many advantages not enjoyed by conversion with quinidine. It is more effective in the sense that a higher conversion rate can be obtained, including those patients who had not responded to quinidine previously. The effect is immediate. It is practically harmless to the myocardium, as opposed to the depressing effects commonly associated with the use of the anti-arrhythmic drugs, notably quinidine.

We have undertaken to convert atrial fibrillation and flutter in those who had open-heart surgery for aortic or mitral valvular disease. All but one had atrial fibrillation, and initial success was obtained in 88 per cent, which approximates to those experienced by others (Oram et al., 1963; Lown, 1964; Morris et al., 1964; Lemberg et al., 1964).
The interval between the open-heart surgery and cardioversion appears to have no influence on the chance of conversion, for success was evident in both extremes, namely, 24 hours and 32 months. In the former, the procedure was prompted by impending cardiac failure, associated with atrial flutter with rapid ventricular rate, not responding to a large dose of digitalis. Due to potential embolism during the immediate post-operative period, and especially those that might occur at the time of conversion, cardioversion should probably be carried out as an elective procedure at least a week after operation. If termination of arrhythmia is necessary because of deterioration of cardiac function, then cardioversion may be carried out sooner.

It is probably not a mere coincidence that all 3 failures occurred in those who had the longest duration of atrial fibrillation (7, 7, and 10 years, respectively) and had a Starr-Edwards mitral valve inserted for correction of mitral regurgitation. Two of these had noticeable left atrial enlargement. In none were previous attempts at conversion with quinidine successful. The wisdom of routine cardioversion in similar cases may be argued. However, if there is a history of systemic embolization, re-establishment of sinus rhythm may reduce the risk of further embolic episodes.

The recurrence rate was 35 per cent (8 in 23) during the follow-up period of 10 months. Except for 2 who had the earliest recurrences, namely, a few minutes and four days, respectively, all cases recurred in between 1 and 7 months. The average duration of atrial fibrillation in these 6 patients was 4 years. There were 3 patients with prosthetic valves among these who deserve comment. All were in their late forties or in the sixth decade, though only one of them sustained chronic atrial fibrillation before cardioversion. In the other 2, one developed atrial fibrillation, and the other developed atrial flutter during the immediate post-operative period. It is possible that previous rheumatic process and long-standing mitral valvular disease rendered particularly the left atrium vulnerable to operative trauma in precipitating atrial arrhythmia.
The results of cardioversion in patients with prosthetic valves may not seem promising. Of 11 patients, 6 either failed or showed recurrences, and these are the patients who had either mitral regurgitation or mitral stenosis with heavily calcified mitral valves. The left atrium in these patients may be highly susceptible to atrial fibrillation. Most likely, these factors rather than the presence of the prosthetic valve itself predispose to the outcome of cardioversion.

Embolicus occurs infrequently as a complication of cardioversion. Its incidence has been reported at 2–3 per cent in larger series (Oram et al., 1963; Morris et al., 1964; Lemberg et al., 1964). In our series, none occurred during or immediately after cardioversion.

As the experience accumulates it has become evident that cardioversion may be attended by serious arrhythmia not expected before the procedure. It is obvious that failure in the sinoatrial node to resume pacemaking activity following electric depolarization of the heart will result in the dominance of the lower pacemaker (Killip, 1963). Similarly, the presence of A–V block may culminate in ventricular arrhythmia and asystole following cardioversion, especially with previous administration of antiarrhythmic agents (Dreifus, Rabbino, and Watanabe, 1964). These changes in sinoatrial and A–V nodes may result from rheumatic activity, arteriosclerotic process, over-digitalization, or a combination of these. Digitalis has been implicated in the occurrence of serious arrhythmia following cardioversion. It has been proposed that digitalis will be withdrawn in all cases for a few days before the procedure (Gilbert and Cuddy, 1964). In view of dormant digitalis toxicity in some cases and of the possibility of its potentiation following cardioversion, this precaution may be justified.

The maintenance of sinus rhythm after conversion depends upon various factors, e.g., type and duration of arrhythmia, type of valvular lesion, size of the left atrium, and adequacy of quinidine administration (Lown, 1964; Morris et al., 1964). Those intolerant to quinidine are poor candidates, so are those who failed to maintain sinus rhythm on a reasonable dose of quinidine after a successful conversion. One patient in this series was placed on pronestyl instead of quinidine because of his intolerance to the latter. Although cardioversion was successful, atrial fibrillation recurred in a month while the patient was on pronestyl.

**SUMMARY**

Twenty-six patients underwent cardioversion for atrial fibrillation and flutter following open-heart valvular surgery. In 11 of these, a Starr–Edwards valve prosthesis was inserted.

The procedure was successful in 23 (88%) and unsuccessful in 3 (12%). There were 8 recurrences over a period of 10 months. Three failures and 3 recurrences occurred in those who had a prosthetic valve. In this series, prosthetic valves were employed in patients with mitral regurgitation (4), aortic regurgitation (1), and mitral stenosis with heavily calcified mitral valve (6). It is felt that factors associated with the underlying lesion rather than with the valve itself were responsible for these results. Proper selection of cases will increase the rate of successful cardioversion. Relative frequency of systemic embolism following the insertion of the prosthetic valves, especially in cases with a history of previous embolization, strongly justifies the attempt at cardioversion for atrial fibrillation and flutter.

**REFERENCES**


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