Be it resolved that all patients with heart failure and atrial fibrillation should be cardioverted at least once.

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Disclosures

- None in respect of this talk
What this talk is *not* about

- Catheter ablation strategies for AF
- Tachymyopathy
- Internal defibrillation (HF patients with ICD)
- Long term pharmacologic therapy with anti-arrhythmics (which may increase side effects and overall risk)
- HFpEF and AF (data on HF and AF are confined to HFrEF)
What this talk is about

- Giving heart failure patients in atrial fibrillation at least one attempt at cardioversion to sinus rhythm to improve ventricular systolic and diastolic function
ARS Question

Please pick option below most applicable to your practice:

1) I try at least one cardioversion in all HF patients with AF
2) I do not attempt cardioversion in HF patients with AF
3) I attempt cardioversion depending on the HF patient characteristics
pick option below most applicable to your practice:

I try at least one cardioversion in all HF patients with AF

I do not attempt cardioversion in HF patients with AF

I attempt a cardioversion depending on the HF patient...

Start the presentation to activate live content

If you see this message in presentation mode, install the add-in or get help at PollEv.com/app
AF begets HF and HF begets AF

Cycle of interdependence between HF and AF

- Loss of atrial systole
- Decreased diastolic filling interval
- Decreased cardiac output
- Increased end-diastolic pressure
- RAAS/neurohormonal activation

- Left atrial stretch
- Increased atrial pressure
- Increased atrial size
- Atrial fibrosis

- Tachycardia
- Irregular conduction

- Increased focal triggers
- Conduction slowing
- Shortened atrial effective refractory period
- Increased action potential duration heterogeneity

Kotecha D, Piccini JP. Eur Heart J 2015; 36: 3250-7
What is the Evidence?
A COMPARISON OF RATE CONTROL AND RHYTHM CONTROL IN PATIENTS WITH RECURRENT PERSISTENT ATRIAL FIBRILLATION


FOR THE RATE CONTROL VersUS ELECTRICAL CARDIOVERSION FOR PERSISTENT ATRIAL FIBRILLATION STUDY GROUP

ABSTRACT

Background Maintenance of sinus rhythm is the main therapeutic goal in patients with atrial fibrillation. However, recurrences of atrial fibrillation and side effects of antiarrhythmic drugs offset the benefits of sinus rhythm. We hypothesized that ventricular rate control is not inferior to the maintenance of sinus rhythm for the treatment of atrial fibrillation.

Methods We randomly assigned 522 patients who had persistent atrial fibrillation after a previous electrical cardioversion to receive treatment aimed at rate control or rhythm control. Patients in the rate-control group received oral anticoagulant drugs and rate-slowing medication. Patients in the rhythm-control group underwent serial cardioversions and received antiarrhythmic drugs and oral anticoagulant drugs. The endpoint was a composite of death from cardiovascular causes, heart failure, thromboembolic complications, bleeding, implantation of a pacemaker, and severe adverse effects of drugs.

Results After a mean (±SD) of 2.3±0.6 years, 39 percent of the 266 patients in the rhythm-control group had sinus rhythm, as compared with 10 percent of the 256 patients in the rate-control group. The primary end point occurred in 44 patients (17.2 percent) in the rate-control group and in 60 (22.6 percent) in the rhythm-control group. The 90 percent (two-sided) upper boundary of the absolute difference in the primary end point was 0.4 percent (the prespecified criterion for noninferiority was 10 percent or less). The distribution of the various components of the primary end point was similar in the rate-control and rhythm-control groups.

Conclusions Rate control is not inferior to rhythm control for the prevention of death and morbidity from cardiovascular causes and may be appropriate therapy in patients with a recurrence of persistent atrial fibrillation after electrical cardioversion. (N Engl J Med 2002;347:1834–40.)

Atrial fibrillation is not a benign condition.1,2 For many clinicians, maintenance of sinus rhythm is the main therapeutic goal. In patients with persistent atrial fibrillation, repeated electrical cardioversion and prophylactic antiarrhythmic drugs are used to maintain sinus rhythm.3 However, frequent recurrences of atrial fibrillation and adverse effects of drugs decrease the potential benefits of electrical cardioversion.4,6 Also, the beneficial effects of rhythm control may be nullified by life-threatening cardiovascular events. Such events may be related not to the rhythm but, rather, to underlying cardiovascular abnormalities.4 Since the rhythm is not the main determinant of the prognosis, it is questionable whether rhythm control is better than ventricular rate control.7,8 We performed a randomized, prospective study to compare the long-term effects of rate control with those of rhythm control, using electrical cardioversion for persistent atrial fibrillation. Our hypothesis was that rate control is not inferior to rhythm control for the treatment of persistent atrial fibrillation.

METHODS

Study Design

Thirty-one centers in the Netherlands participated in the study. The institutional review boards at each participating hospital approved the study protocol, and all patients gave written informed consent. The study was conducted from June 1, 1998, until July 1, 2001. The follow-up period was at least two years. The study design is shown in Figure 1.

Only patients with recurrent persistent atrial fibrillation or flutter, in whom oral anticoagulation was not contraindicated, were included. Persistent atrial fibrillation and flutter were defined as non-self-terminating arrhythmia requiring electrical cardioversion to obtain sinus rhythm.9,10 Atrial flutter was defined as a supraventricular tachycardia with a regular atrial rhythm between 240 and 300 beats per minute. Patients were excluded if arrhythmia had lasted longer than one year. In addition to the usual exclusion criteria for studies of this type, we excluded patients with heart failure (New York Heart Association functional class III or IV), systemic illness, recent myocardial infarction, prolonged atrial fibrillation (i.e., >5 days), and atrial flutter with atrial fibrillation. After meeting the inclusion criteria, the patients were randomly assigned to the rate-control or rhythm-control group by use of computer-generated random numbers. The investigators were unaware of the randomization results. At the start of the study, the patients selected the group to which they wished to be assigned; those not assigned to the rate-control or rhythm-control groups were assigned to the other group.
Figure 2. Kaplan–Meier Curves for Event-free Survival in the Rate-Control and Rhythm-Control Groups.
**Table 2. Incidence of the Primary End Point and Its Components According to the Treatment Group.**

<table>
<thead>
<tr>
<th>END POINT</th>
<th>RATE CONTROL (N=256)</th>
<th>RHYTHM CONTROL (N=266)</th>
<th>ABSOLUTE DIFFERENCE (90% CI)†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Composite end point</td>
<td>44 (17.2)</td>
<td>60 (22.6)</td>
<td>-5.4 (-11.0 to 0.4)</td>
</tr>
<tr>
<td>Death from cardiovascular causes</td>
<td>18 (7.0)</td>
<td>18 (6.8)</td>
<td>0.2 (-3.4 to 3.9)</td>
</tr>
<tr>
<td>Heart failure</td>
<td>9 (3.5)</td>
<td>12 (4.5)</td>
<td>-1.0 (-3.8 to 1.8)</td>
</tr>
<tr>
<td>Thromboembolic complications</td>
<td>14 (5.5)</td>
<td>21 (7.9)</td>
<td>-2.4 (-6.0 to 1.2)</td>
</tr>
<tr>
<td>Bleeding</td>
<td>12 (4.7)</td>
<td>9 (3.4)</td>
<td>1.3 (-1.5 to 4.1)</td>
</tr>
<tr>
<td>Severe adverse effects of antiarrhythmic drugs</td>
<td>2 (0.8)</td>
<td>12 (4.5)</td>
<td>-3.7 (-6.0 to -1.4)</td>
</tr>
<tr>
<td>Implantation of a pacemaker</td>
<td>3 (1.2)</td>
<td>8 (3.0)</td>
<td>-1.8 (-3.9 to 0.2)</td>
</tr>
</tbody>
</table>
Rate control versus rhythm control for patients with persistent atrial fibrillation with mild to moderate heart failure: results from the RAte Control versus Electrical cardioversion (RACE) study


**BACKGROUND:** This study was conducted to compare rate- and rhythm-control therapy in patients with persistent atrial fibrillation (AF) and mild to moderate chronic heart failure (CHF). Rate control is not inferior to rhythm control in preventing mortality and morbidity in patients with AF. In CHF, this issue is still unsettled.

**METHODS:** In this predefined analysis of the RACE study, a total of 261 patients were in New York Heart Association (NYHA) classes II and III at baseline. These patients were analyzed. The primary end point was a composite of cardiovascular mortality, hospitalization for CHF, thromboembolic complications, bleeding, pacemaker implantation, and life-threatening drug side effects.

**RESULTS:** After 2.3 +/- 0.6 years, the primary end point occurred in 29 (22.3%) of the 130 rate-control patients and in 32 (24.4%) of the 131 rhythm-control patients. More cardiovascular deaths, hospitalization for CHF, and bleeding occurred under rate control. Thromboembolic complications, drug side effects, and pacemaker implantation were more frequent under rhythm control. Quality of life did not differ between strategies. In patients successfully treated with rhythm control, the prevalence of end points was not different from those who were in AF at study end. However, the type of end point was different: mortality, bleeding, hospitalization for heart failure, and pacemaker implantation occurred less frequently.

**CONCLUSIONS:** In patients with mild to moderate CHF, rate control is not inferior to rhythm control. However, if sinus rhythm can be maintained, outcome may be improved. A prospective randomized trial is necessary to confirm these results.
A COMPARISON OF RATE CONTROL AND RHYTHM CONTROL IN PATIENTS WITH ATRIAL FIBRILLATION

THE ATRIAL FIBRILLATION FOLLOW-UP INVESTIGATION OF RHYTHM MANAGEMENT (AFFIRM) INVESTIGATORS*

ABSTRACT

Background There are two approaches to the treatment of atrial fibrillation: one is cardioversion and treatment with antiarrhythmic drugs to maintain sinus rhythm, and the other is the use of rate-controlling drugs, allowing atrial fibrillation to persist. In both approaches, the use of anticoagulant drugs is recommended.

Methods We conducted a randomized, multicenter comparison of these two treatment strategies in patients with atrial fibrillation and a high risk of stroke or death. The primary end point was overall mortality.

Results A total of 4060 patients (mean ± SD age, 69.7 ± 9.0 years) were enrolled in the study; 70.8 percent had a history of hypertension, and 38.2 percent had coronary artery disease. Of the 3311 patients with echocardiograms, the left atrium was enlarged in 64.7 percent and left ventricular function was depressed in 26.0 percent. There were 356 deaths among the patients assigned to rhythm-control therapy and 310 deaths among those assigned to rate-control therapy (mortality at five years, 23.8 percent and 21.3 percent, respectively; hazard ratio, 1.15 [95 percent confidence interval, 0.99 to 1.34]; P = 0.08). More patients in the rhythm-control group than in the rate-control group were hospitalized, and there were more adverse drug effects in the rhythm-control group as well. In both groups, the majority of strokes occurred after warfarin had been stopped or when the international normalized ratio was subtherapeutic.

Conclusions Management of atrial fibrillation with the rhythm-control strategy offers no survival advantage over the rate-control strategy, and there are potential advantages, such as a lower risk of adverse drug effects, with the rate-control strategy. Anticoagulation should be continued in this group of high-risk patients. (N Engl J Med 2002;347:1825-33.)

Address reprint requests to the AFFIRM Clinical Trial Center, Axio Research, 2601 4th Ave., Ste. 200, Seattle, WA 98121, or to leong@axioresearch.com.

Rhythm-Control Strategy

In the rhythm-control group, the antiarrhythmic drug used was chosen by the treating physician.\textsuperscript{24,25} Attempts to maintain sinus rhythm could include cardioversion as necessary. The following drugs were acceptable for use, according to the protocol: amiodarone, disopyramide, flecainide, moricizine, procainamide, propafenone, quinidine, sotalol, and combinations of these drugs. When dofetilide became available, it also could be used. Specific guidelines for the use of antiarrhythmic drugs were imposed.\textsuperscript{22,26}
<table>
<thead>
<tr>
<th>DRUG</th>
<th>RATE-CONTROL GROUP</th>
<th>RHYTHM-CONTROL GROUP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>USED DRUG FOR INITIAL THERAPY</td>
<td>USED DRUG AT ANY TIME</td>
</tr>
</tbody>
</table>
|                 | no. of patients (%) | no. of patients (%) |}

**Rate control**

- Data available: 1957 (48.5) 2027 (70.6) 1266 (32.9) 2033 (54.4)
- Digoxin: 949 (48.5) 1432 (70.6) 417 (32.9) 1106 (54.4)
- Beta-blocker: 915 (46.8) 1380 (68.1) 276 (21.8) 1008 (49.6)
- Diltiazem: 583 (29.8) 935 (46.1) 198 (15.6) 610 (30.0)
- Verapamil: 187 (9.6) 340 (16.8) 56 (4.4) 204 (10.0)

**Rhythm control**

- Data available: 1265 (0.2) 2027 (10.2) 1960 (37.5) 2033 (62.8)
- Amiodarone: 2 (0.2) 207 (10.2) 735 (37.5) 1277 (62.8)
- Sotalol: 1 (0.1) 84 (4.1) 612 (31.2) 841 (41.4)
- Propafenone: 2 (0.2) 45 (2.2) 183 (9.3) 294 (14.5)
- Procainamide: 0 30 (1.5) 103 (5.3) 173 (8.5)
- Quinidine: 2 (0.2) 14 (0.7) 92 (4.7) 151 (7.4)
- Flecainide: 0 29 (1.4) 88 (4.5) 169 (8.3)
- Disopyramide: 0 7 (0.3) 42 (2.1) 87 (4.3)
- Moricizine: 0 2 (0.1) 14 (0.7) 35 (1.7)
- Dofetilide: 0 5 (0.2) 0 13 (0.6)
<table>
<thead>
<tr>
<th>Years</th>
<th>Rhythm control</th>
<th>Rate control</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>1</td>
<td>80 (4)</td>
<td>78 (4)</td>
</tr>
<tr>
<td>2</td>
<td>175 (9)</td>
<td>148 (7)</td>
</tr>
<tr>
<td>3</td>
<td>257 (13)</td>
<td>210 (11)</td>
</tr>
<tr>
<td>4</td>
<td>314 (18)</td>
<td>275 (16)</td>
</tr>
<tr>
<td>5</td>
<td>352 (24)</td>
<td>306 (21)</td>
</tr>
</tbody>
</table>

**Figure 1.** Cumulative Mortality from Any Cause in the Rhythm-Control Group and the Rate-Control Group.

Time zero is the day of randomization. Data have been truncated at five years.
<table>
<thead>
<tr>
<th>EVENT</th>
<th>OVERALL (N = 4060)</th>
<th>RATE-CONTROL GROUP (N = 2027)</th>
<th>RHYTHM-CONTROL GROUP (N = 2033)</th>
<th>P VALUE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary end point (death)</td>
<td>666 (26.3)</td>
<td>310 (25.9)</td>
<td>356 (26.7)</td>
<td>0.08†</td>
</tr>
<tr>
<td>Secondary end point (composite of death, disabling stroke, disabling anoxic encephalopathy, major bleeding, and cardiac arrest)</td>
<td>861 (32.3)</td>
<td>416 (32.7)</td>
<td>445 (32.0)</td>
<td>0.33</td>
</tr>
<tr>
<td>Torsade de pointes</td>
<td>14 (0.5)</td>
<td>2 (0.2)‡</td>
<td>12 (0.8)</td>
<td>0.007</td>
</tr>
<tr>
<td>Sustained ventricular tachycardia</td>
<td>15 (0.6)</td>
<td>9 (0.7)</td>
<td>6 (0.6)</td>
<td>0.44</td>
</tr>
<tr>
<td>Cardiac arrest followed by resuscitation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ventricular fibrillation or ventricular tachycardia</td>
<td>19 (0.6)</td>
<td>10 (0.7)</td>
<td>9 (0.5)</td>
<td>0.83</td>
</tr>
<tr>
<td>Pulseless electrical activity, bradycardia, or other rhythm</td>
<td>10 (0.3)</td>
<td>1 (&lt;0.1)</td>
<td>9 (0.6)</td>
<td>0.01</td>
</tr>
<tr>
<td>Central nervous system event</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>211 (8.2)</td>
<td>105 (7.4)</td>
<td>106 (8.9)</td>
<td>0.93</td>
</tr>
<tr>
<td>Ischemic stroke§</td>
<td>157 (6.3)</td>
<td>77 (5.5)</td>
<td>80 (7.1)</td>
<td>0.79</td>
</tr>
<tr>
<td>After discontinuation of warfarin</td>
<td>69</td>
<td>25</td>
<td>44</td>
<td></td>
</tr>
<tr>
<td>During warfarin but with INR &lt; 2.0</td>
<td>44</td>
<td>27</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td>Concurrent atrial fibrillation</td>
<td>67</td>
<td>42</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>Primary intracerebral hemorrhage</td>
<td>34 (1.2)</td>
<td>18 (1.1)</td>
<td>16 (1.3)</td>
<td>0.73</td>
</tr>
<tr>
<td>Subdural or subarachnoid hemorrhage</td>
<td>24 (0.8)</td>
<td>11 (0.8)</td>
<td>13 (0.8)</td>
<td>0.68</td>
</tr>
<tr>
<td>Disabling anoxic encephalopathy</td>
<td>9 (0.3)</td>
<td>4 (0.2)</td>
<td>5 (0.4)</td>
<td>0.74</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>140 (5.5)</td>
<td>67 (4.9)</td>
<td>73 (6.1)</td>
<td>0.60</td>
</tr>
<tr>
<td>Hemorrhage not involving the central nervous system</td>
<td>203 (7.3)</td>
<td>107 (7.7)</td>
<td>96 (6.9)</td>
<td>0.44</td>
</tr>
<tr>
<td>Systemic embolism</td>
<td>16 (0.5)</td>
<td>9 (0.5)</td>
<td>7 (0.4)</td>
<td>0.62</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>8 (0.3)</td>
<td>2 (0.1)</td>
<td>6 (0.5)</td>
<td>0.16</td>
</tr>
<tr>
<td>Hospitalization after base line</td>
<td>2594 (76.6)</td>
<td>1220 (73.0)</td>
<td>1374 (80.1)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>
Guglin M, Chen R, Curtis AB. Sinus rhythm is associated with fewer heart failure symptoms: Insights from the AFFIRM trial. Heart Rhythm 2010;7:596-601

**Background:** The AFFIRM trial demonstrated no mortality benefit from a rhythm control strategy compared with rate control of atrial fibrillation (AF). However, AF is associated with greater morbidity and mortality and poorer functional status than sinus rhythm, which is more likely to be achieved with a rhythm control strategy.

**Objective** This study sought to compare heart failure (HF) symptoms in the different AFFIRM (Atrial Fibrillation Follow-Up Investigation of Rhythm Management) subgroups depending on the prevalence of AF or sinus rhythm throughout the follow-up period.

**Methods:** This study analyzed a limited-access dataset from the AFFIRM trial.

**Results:** Symptomatic HF was more common in the rate control than in the rhythm control arm. On analysis based on actual rhythm, New York Heart Association functional status was the best in patients who were in stable sinus rhythm, worse if they were consistently in AF, and much worse if they were changed back and forth between the rhythm control and rate control strategies. Patients in all groups had fewer HF symptoms and required less HF medications when they were in sinus rhythm compared with AF, except for those who crossed over from the rhythm to the rate control strategy.

**Conclusion:** Patients in the rhythm control arm had fewer HF symptoms than those in the rate control arm. Stable sinus rhythm was associated with the best functional status. Patients who are the most symptomatic in AF but are unable to maintain normal sinus rhythm if treated by the means used in the AFFIRM trial may be candidates for other treatment options, such as ablation.
Rhythm Control versus Rate Control for Atrial Fibrillation and Heart Failure

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Kerry L. Lee, Ph.D., Martial G. Bourassa, M.D., J. Malcolm O. Arnold, M.D., Alfred E. Buxton, M.D.,
A. John Camm, M.D., Stuart J. Connolly, M.D., Marc Dubuc, M.D., Anique Ducharme, M.D., M.Sc.,
Peter G. Guerra, M.D., Stefan H. Hohnloser, M.D., Jean Lambert, Ph.D., Jean-Yves Le Heuzey, M.D.,
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Lynne Warner Stevenson, M.D., William G. Stevenson, M.D., Bernard Thibault, M.D., and Albert L. Waldo, M.D.,
for the Atrial Fibrillation and Congestive Heart Failure Investigators*

ABSTRACT

BACKGROUND

It is common practice to restore and maintain sinus rhythm in patients with atrial fibrillation and heart failure. This approach is based in part on data indicating that atrial fibrillation is a predictor of death in patients with heart failure and suggesting that the suppression of atrial fibrillation may favorably affect the outcome. However, the benefits and risks of this approach have not been adequately studied.

METHODS

We conducted a multicenter, randomized trial comparing the maintenance of sinus rhythm (rhythm control) with control of the ventricular rate (rate control) in patients with a left ventricular ejection fraction of 35% or less, symptoms of congestive heart failure, and a history of atrial fibrillation. The primary outcome was the time to death from cardiovascular causes.

RESULTS

A total of 1376 patients were enrolled (682 in the rhythm-control group and 694 in the rate-control group) and were followed for a mean of 37 months. Of these patients, 182 (27%) in the rhythm-control group died from cardiovascular causes, as compared with 175 (25%) in the rate-control group (hazard ratio in the rhythm-control group, 1.06; 95% confidence interval, 0.86 to 1.30; P = 0.59 by the log-rank test). Secondary outcomes were similar in the two groups, including death from any cause (3% in the rhythm-control group and 3% in the rate-control group), stroke (3% and 4%, respectively), worsening heart failure (28% and 31%), and the composite of death from cardiovascular causes, stroke, or worsening heart failure (43% and 46%). There were also no significant differences favoring either strategy in any predefined subgroup.

CONCLUSIONS

In patients with atrial fibrillation and congestive heart failure, a routine strategy of rhythm control does not reduce the rate of death from cardiovascular causes, as compared with a rate-control strategy. (ClinicalTrials.gov number, NCT00597077.)

*Investigators and committees in the Atrial Fibrillation and Congestive Heart Failure trial are listed in the Appendix.


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H3T 1C8, Canada, or at droy@icm.mhri.

*
Rhythm Control

Aggressive therapy to prevent atrial fibrillation was recommended for patients in the rhythm-control group. Electrical cardioversion was recommended within 6 weeks after randomization in patients who did not have conversion to sinus rhythm after antiarrhythmic drug therapy. If necessary, a second cardioversion was recommended within 3 months after enrollment. Additional cardioversions were recommended for subsequent recurrences of atrial fibrillation. Amiodarone was the drug of choice for the maintenance of sinus rhythm, and either sotalol or dofetilide was used if required. The installation of a permanent pacemaker was recommended if bradycardia prevented the use of antiarrhythmic drugs. Patients who did not have a response to antiarrhythmic drug therapy could be referred for nonpharmacologic therapy.
Figure 2. Kaplan–Meier Estimates of Death from Cardiovascular Causes (Primary Outcome).

Among 1376 patients with atrial fibrillation and congestive heart failure who were followed for a mean of 37 months, 182 patients (27%) in the rhythm-control group died from cardiovascular causes, as compared with 175 patients (25%) in the rate-control group (hazard ratio, 1.06; 95% confidence interval, 0.86 to 1.30).
Lack of prevention of heart failure by serial electrical cardioversion in patients with persistent atrial fibrillation.


**DESIGN:** Non-randomised controlled trial; cohort; case series; mean (SD) follow up duration 3.4 (1.6) years.

**SETTING:** Tertiary care centre.

**SUBJECTS:** Consecutive sampling of 342 patients with persistent atrial fibrillation (defined as > 24 hours duration) considered eligible for electrical cardioversion.

**INTERVENTIONS:** Serial electrical cardioversions and serial antiarrhythmic drug treatment, after identification and treatment of underlying cardiovascular disease.

**OUTCOMES:** heart failure complications: development or progression of heart failure requiring the institution or addition of drug treatment, hospital admission, or death from heart failure.

**RESULTS:** Development or progression of heart failure occurred in 38 patients (11%), and 22 patients (6%) died from heart failure. These complications were related to the presence of coronary artery disease (p < 0.001, risk ratio 3.2, 95% confidence interval (CI) 1.6 to 6.5), rheumatic heart disease (p < 0.001, risk ratio 5.0, 95% CI 2.4 to 10.2), cardiomyopathy (p < 0.001, risk ratio 5.0, 95% CI 2.0 to 12.4), atrial fibrillation for < 3 months (p = 0.04, risk ratio 2.0, 95% CI 1.0 to 3.7), and poor exercise tolerance (New York Heart Association class III at inclusion, p < 0.001, risk ratio 3.5, 95% CI 1.9 to 6.7). No heart failure complications were observed in patients with lone atrial fibrillation.

**CONCLUSIONS:** Aggressive serial electrical cardioversion does not prevent heart failure complications in patients with persistent atrial fibrillation. These complications are predominantly observed in patients with more severe underlying cardiovascular disease. Randomised comparison with rate control treatment is needed to define the optimal treatment for persistent atrial fibrillation in relation to heart failure.

Effects of sinus rhythm maintenance on left heart function after electrical cardioversion of atrial fibrillation: implications for tachycardia-induced cardiomyopathy. Can J Cardiol 2015;31:36-43.

METHODS: Consecutive AF patients who were to undergo electrical cardioversion (ECV) were enrolled. Patients with unstable or acute heart failure, severe valvular diseases, recent open-heart surgery, major disorders, or an unsuccessful ECV were excluded. Transthoracic echocardiography, including 3-dimensional left atrial and ventricular volume acquisitions, was performed 1-2 hours before and after ECV, and 4-6 weeks later.

RESULTS: In 73 patients (77% male, 66 ± 11 years), ECV resulted in an immediate increase in LVEF (from 43 [interquartile range (IQR), 33-50%] to 48 [IQR, 40-53%]; P < 0.0001). Four to 6 weeks after ECV, ejection fraction increased further in patients who remained in sinus rhythm (SR) (n = 55) to 55 (IQR, 44-62%); P < 0.001. In patients with AF relapse, LVEF returned to values comparable to pre-ECV (n = 18) (44 [IQR, 32-51%]; P = 0.03). The atrial emptying fraction did not significantly change immediately after ECV (n = 69; from 20 [IQR, 13-25]% to 20 [IQR, 15-28]%; P = 0.14). Only patients who remained in SR showed an increase in atrial emptying fraction after 4-6 weeks (n = 51; to 37 [IQR, 26-48%]; P < 0.0001 vs post-ECV).

CONCLUSIONS: Immediate improvement in LVEF after ECV explains approximately 50% of total LVEF increase over time. However, in SR, LVEF, and atrial function continuously increase over 4-6 weeks after ECV. This might be attributable to recovery of tachycardia-induced cardiomyopathy.
# Upcoming trials in AF and HF

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Kotecha D, Piccini JP. Eur Heart J 2015; 36: 3250-7
Proposed approach to management of AF in HF patients

Kotecha D, Piccini JP. Eur Heart J 2015; 36: 3250-7
Take Home Message

1. One attempt at cardioversion is warranted in patients with AF and HF
2. Multiple cardioversions are not indicated, and may be harmful
3. Chronic anti-arrhythmic pharmacotherapy may be harmful
4. One try at cardioversion to sinus rhythm may improve cardiac function and decrease HF outcomes in patients with AF and HF, and has few downsides
Thank you

The David Braley Cardiac, Vascular, & Stroke Research Institute
McMaster University/Hamilton Health Sciences
Hamilton, Ontario, Canada
Betablockers in HF and SR vs. AF

A. All-cause mortality: sinus rhythm

- HR 0.73 (95% CI 0.67–0.80); P < 0.001

B. All-cause mortality: atrial fibrillation

- HR 0.97 (95% CI 0.83–1.14); P = 0.73

Decreased Mortality With Beta-Blockers in Patients With Heart Failure and Coexisting Atrial Fibrillation: An AF-CHF Substudy


OBJECTIVES: The impact of beta-blockers on mortality and hospitalizations was assessed in the largest randomized trial of patients with both atrial fibrillation (AF) and heart failure with a reduced ejection fraction (HFrEF): the Atrial Fibrillation-Congestive Heart Failure trial.

BACKGROUND: Although beta-blockers are the cornerstone of therapy for HFrEF, a recent patient-level meta-analysis cast doubt on their efficacy in patients with coexisting AF.

METHODS: From 1,376 subjects randomized in the AF-CHF trial, those without beta-blockers at baseline were propensity matched to a maximum of 2 exposed patients. Primary analyses respected the intention-to-treat principle. In on-treatment sensitivity analyses, beta-blocker status was modeled as a time-dependent covariate.

RESULTS: Baseline characteristics were comparable among the matched cohorts (mean age 70 ± 11 years, 81% male, and mean left ventricular ejection fraction 27 ± 6%). During a median follow-up of 37 months, beta-blockers were associated with significantly lower all-cause mortality (hazard ratio [HR]: 0.721, 95% confidence interval [CI]: 0.549 to 0.945; p = 0.0180) but not hospitalizations (HR: 0.886; 95% CI: 0.715 to 1.100; p = 0.2232). Similar results were obtained in sensitivity analyses that modeled beta-blockers as a time-dependent variable (HR: 0.668 for all-cause mortality; 95% CI: 0.511 to 0.874; p = 0.0032; HR: 0.814 for hospitalizations; 95% CI: 0.653 to 1.014; p = 0.0658). There were no significant interactions between beta-blockers and patterns (i.e., persistent vs. paroxysmal) or burden of AF with respect to mortality or hospitalizations.

CONCLUSIONS: In propensity-matched analyses, beta-blockers were associated with significantly lower mortality but not hospitalizations in patients with HFrEF and AF, irrespective of the pattern or burden of AF. These results support current evidence-based recommendations for beta-blockers in patients with HFrEF, whether or not they have associated AF.