Prospective study of left ventricular function after radiofrequency ablation of atrioventricular junction in patients with atrial fibrillation

Magnus Edner, Kenneth Caidahl, Lennart Bergfeldt, Börje Darpö, Nils Edvardsson, Mårten Rosenqvist

Abstract

Background—In patients with drug resistant incessant supraventricular tachycardia, radiofrequency induced ablation of the atrioventricular junction and pacemaker implantation have hitherto been considered a treatment of last resort.

Objective—To assess the short and long term effects of ablation of the atrioventricular junction on systolic and diastolic left ventricular function in patients with atrial fibrillation with and without impaired left ventricular function.

Patients—29 patients (19 men; mean age 65 (SD 7) years (range 50–76)) undergoing ablation of the atrioventricular junction for drug refractory atrial fibrillation were examined a mean of 2, 65, and 216 days after ablation of the bundle of His.

Main outcome measures—Left ventricular ejection fraction and early filling deceleration times (Edec) were assessed by Doppler echocardiography after 1 to 2 hours of ventricular pacing at a rate of 80 beats/minute.

Results—In 14 patients with a left ventricular ejection fraction < 50% left ventricular ejection fraction increased significantly from 32% (11%) to 39% (11%) (65 days) and 45% (11%) (216 days) (P < 0.001); Edec increased from 142 (46) ms to 169 (57) ms (65 days) and 167 (56) ms (216 days) (P < 0.05). In 15 patients with an ejection fraction ≥ 50% at the initial examination no significant change in systolic function was observed.

Conclusions—In patients with left ventricular dysfunction long term improvement of systolic and diastolic left ventricular function was seen after ablation of the atrioventricular junction for rate control of atrial fibrillation. This procedure had no adverse effects on normal left ventricular function.

Keywords: atrial fibrillation; left ventricular function; ablation of atrioventricular junction

Left ventricular dysfunction and congestive heart failure may develop as a consequence of incessant tachycardia. In the clinical setting this has been observed especially in children and newborn infants. In adult patients with left ventricular dysfunction and atrial tachycardias, mostly atrial fibrillation, a causal relation is often obscure but highly likely. In selected cases direct current conversion or rate regulation by pharmacological treatment has been followed by improvement in systolic left ventricular function. However, prophylactic drug treatment after direct current conversion or to regulate the heart rate in chronic atrial fibrillation usually has negative inotropic effects, with the exception of digoxin and possibly amiodarone. Furthermore, such treatment sometimes fails or is intolerable. As an alternative, ablation of the atrioventricular junction has been introduced, initially using direct current shocks and subsequently radiofrequency energy. This treatment may also improve systolic function in patients with left ventricular dysfunction, at least in the short term of six weeks.

This prospective study was designed to assess the short and long term effects of radiofrequency ablation of the atrioventricular junction and pacemaker implantation on systolic and diastolic left ventricular function in patients with atrial fibrillation with and without impaired left ventricular function.

Patients and methods

Twenty nine patients (19 men) with a mean age of 65 (SD 7) years (range 50–76) were included. The patients had chronic or paroxysmal atrial fibrillation, and they had on average tried 3-7 (2–7) different antiarrhythmic drugs with insufficient symptom relief or rate regulation or adverse effects before ablation was considered. The patients were divided into two groups on the basis of the results of the first echocardiographic examination an average of two days after the procedure since we thought it important to have comparable heart rates. Fifteen patients in group A had normal systolic left ventricular function (ejection fraction ≥ 50%) and 14 in group B had impaired left ventricular function (ejection fraction < 50%). In group A two patients had valvar heart disease—one with an aortic valve prosthesis and one with a slight to moderate mitral regurgitation. In group B four patients had valvar heart disease—one had been operated on for an atrial septal defect, one had moderate mitral regurgitation, and two had a slight aortic stenosis. In group B eight patients...
Table 1  Baseline characteristics. Values are numbers of patients unless stated otherwise

<table>
<thead>
<tr>
<th></th>
<th>Group A (n = 15)</th>
<th>Group B (n = 14)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricular ejection fraction (%)</td>
<td>≥ 50</td>
<td>&lt; 50</td>
</tr>
<tr>
<td>Women/men</td>
<td>4/11</td>
<td>6/8</td>
</tr>
<tr>
<td>Mean (SD) age (years)</td>
<td>64 (11) (44-82)</td>
<td>68 (7) (52-77)</td>
</tr>
<tr>
<td>Atrial fibrillation:</td>
<td>Chronic</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Paroxysmal</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>Mean duration (years)</td>
<td>5-8 (0-2-20-3)</td>
</tr>
<tr>
<td></td>
<td>Dyspnoea</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>Syncope</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Palpitations</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>Ischaemic heart disease</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Valvar disease</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Congestive heart failure</td>
<td>1</td>
</tr>
</tbody>
</table>

had dilated cardiomyopathy; two of them had ischaemic heart disease, three had valvar heart disease, and in three no other cause was known. Baseline characteristics for the two groups are presented in table 1.

When possible, the antiarrhythmic treatment was withheld 1 to 2 days before the ablation. Drugs used at the initial echocardiographic examination and during follow up are listed in table 2.

**Radiofrequency ablation**

The patient was brought to the catheter laboratory having fasted and been sedated. Under local anaesthesia, a 6F multipolar electrode catheter was inserted into the femoral vein and advanced to the right ventricular apex; it was connected to an external stimulator for back up pacing. A 4 mm tip electrode catheter (Polaris, Mansfield-Webster Catheters, Watertown, MA, USA) with a deflectable curve was advanced in the same way and positioned across the tricuspid valve. This catheter was used to record the largest His bundle, which served as a marker for an appropriate ablation site. Radiofrequency current was generated by a commercially available electrosurgical device (model RFG-3B, Radionics, Burlington, MA, USA) that delivers energy at a frequency of 300 kHz. The energy was delivered between the tip electrode, which was positioned as proximal as possible, and a cutaneous disposable patch electrode that was applied to the patient's back. It was delivered at 30 W for 60 s. If complete heart block was not achieved the catheter was repositioned and the electrode was applied again. A left sided approach by means of the femoral artery and retrograde catherisation of the aortic valve was used in a few cases. After the induction of complete heart block, temporary ventricular pacing was effected for 10 minutes. Pacing was then stopped to verify that complete heart block was still present. After complete heart block had been confirmed the patient received a permanent pacemaker. To avoid ventricular tachyarrhythmias induced by bradycardia, the pacemaker was programmed for a rate of 80 beats/minute during the first week. Every patient underwent preoperative and postoperative (within 24 hours) echocardiography to establish that myocardial or valvar damage had not been induced by ablation.

**Echocardiographic measurements**

All measurements were performed under standardised conditions after 1 to 2 hours of ventricular pacing at 80 beats/minute on three occasions: before leaving hospital and one and six months after ablation.

The ultrasound equipment was Acuson 128 (Mountain View, CA, USA) and Vingmed CFM 750 (Vingmed Sound, Horten, Norway). The left atrial size and area and the left ventricular volumes and ejection fractions were measured according to the recommendations of the American Society of Echocardiography. Biplane volumes were calculated from area tracings using the disc summation method (modified Simpson's rule), and dedicated computer equipment and software (Vingmed Sound, Horten, Norway). Ejection fractions were calculated as the mean of three separate measurements (diastolic

**Table 2  Drugs taken at baseline and as follow up echocardiographic examinations. Values are number of patients**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Baseline</th>
<th>Follow up</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group A</td>
<td>Group B</td>
</tr>
<tr>
<td></td>
<td>Group A</td>
<td>Group B</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>β blocker</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Calcium antagonist</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Diogoxin</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Soratal</td>
<td></td>
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</tr>
</tbody>
</table>

**Table 3  Mean (SD) echocardiographic measurements after ablation**

<table>
<thead>
<tr>
<th></th>
<th>2 Days</th>
<th>65 Days</th>
<th>216 Days</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group A</td>
<td>Group B</td>
<td>Group A</td>
</tr>
<tr>
<td>Left atrium:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mm</td>
<td>44 (7)*</td>
<td>50 (6)</td>
<td>46 (7)</td>
</tr>
<tr>
<td>cm²</td>
<td>23 (5)*</td>
<td>30 (7)</td>
<td>23 (5)</td>
</tr>
<tr>
<td>EFSS</td>
<td>8 (3)*</td>
<td>19 (10)</td>
<td>8 (5)</td>
</tr>
<tr>
<td>Volume diastole (ml)</td>
<td>80 (23)*</td>
<td>142 (83)</td>
<td>81 (17)</td>
</tr>
<tr>
<td>Volume systole (ml)</td>
<td>34 (11)*</td>
<td>102 (71)</td>
<td>35 (10)</td>
</tr>
<tr>
<td>ESVI (ml/m²)</td>
<td>19 (5)*</td>
<td>49 (31)</td>
<td>17 (4)</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>58 (5)*</td>
<td>32 (11)</td>
<td>57 (6)</td>
</tr>
<tr>
<td>Change in stroke volume (ml)</td>
<td>-1.2 (10)</td>
<td>15.5 (14)</td>
<td>-0.6 (11)</td>
</tr>
<tr>
<td>Emax (cm/s)</td>
<td>85 (20)</td>
<td>82 (17)</td>
<td>86 (24)</td>
</tr>
<tr>
<td>Edece (cm)</td>
<td>156 (37)</td>
<td>142 (46)</td>
<td>166 (34)</td>
</tr>
<tr>
<td>IVRT (ms)</td>
<td>74 (26)</td>
<td>81 (19)</td>
<td>78 (24)</td>
</tr>
</tbody>
</table>
Left ventricular function after radiofrequency ablation of atrioventricular junction for atrial fibrillation

Patients

Figure 1: Mean (SD) ejection fractions in patients with initial ejection fraction ≥ 50% (group A) and < 50% (group B) 2, 65, and 216 days after ablation of the atrioventricular junction and pacemaker implantation.

Figure 2: Individual ejection fractions in groups A and B two days after ablation of atrioventricular junction and pacemaker implantation and during follow-up.

Results

ABLATION OF ATRIOVENTRICULAR JUNCTION

A right sided approach was used in 26 patients. A mean of 8.3 applications (9.2; range 1–36) over 358 s (400; range 10–1325) were needed to obtain complete heart block in 27 patients; a modification of conduction through the atrioventricular junction made two of the patients dependent on a pacemaker. Seventeen patients received ablation during permanent atrial fibrillation. The pacemaker used was DDD-R in one patient, VVI in six patients, and VVI-R in 22. Post-operative echocardiographic examinations did not disclose any signs of myocardial or valvar damage induced by ablation. One patient with a carotid artery stenosis and a history of previous transient cerebral ischaemia had a cerebral embolus without persistent neurological sequele on the day after the ablation, probably caused by decreased anticoagulation intensity.

CLINICAL FOLLOW UP

Four patients in each group were examined only twice. Three patients died, one of a cerebral tumour, one suddenly three months after the procedure, and the last of progressive heart failure. One patient did not have acceptable echo views and four patients were not accessible. The initial echocardiographic examination was performed 2 (1–8) days after ablation, and follow up examinations after 65 (20–153) and 216 (130–339) days.

During follow up all patients remained in complete heart block. In group A nine patients had paroxysmal fibrillation at baseline, but during follow up underlying sinus rhythm was observed in only two patients. In group B all three patients with paroxysmal atrial fibrillation showed underlying atrial fibrillation during follow up. The DDD-R pacemaker in one patient had to be reprogrammed to VVI-R because the patient developed permanent atrial fibrillation.

ECHOCARDIOGRAPHIC FINDINGS DURING FOLLOW UP

The left atrium was significantly larger in patients in group B compared with those in group A at the initial examination (P < 0.05). The left atrial diameter was unchanged, but the area decreased in group B after the ablation (P < 0.05). The initial E point septal separation was also significantly larger in group B than in group A (P < 0.05). In group A no change was observed, but in group B a decrease was noted after the ablation (P < 0.05) (table 3).
In 26 patients (90%) apical biplane volumes were obtained and in the remaining three single plane volumes. The end diastolic and end systolic volumes were significantly larger in group B at the initial examination (P < 0.05 and P < 0.01). In group A no significant changes in ejection fraction, end systolic, or end diastolic volumes were observed. In contrast, there was a significant (40%, P < 0.001) increase in ejection fraction after ablation in group B. There was a similar trend also for end systolic volume and end systolic volume index, but these changes were not significant (P = 0.13 and P = 0.07). End diastolic volume did not change after ablation (figure 1, table 3). Individual data are given in figure 2. There was a significant increase in stroke volume in group B (P < 0.05; figure 3, table 3).

There were no significant differences in Emax, Edec, or isovolumetric relaxation time between groups A and B at initial examination. No changes in Emax or isovolumetric relaxation time were observed after the procedure in either group. In both groups Edec increased in group A after 216 days and in group B after 65 days (P < 0.05) (figure 4, table 3). Individual data are given in figure 5.

In group A mitral regurgitation was graded 0-5 (0-2) two days after ablation and 0-5 (0-3) 216 days afterwards. The corresponding values for tricuspid regurgitation were 0-7 (0-3) and 1-0 (0-5) (P < 0.05). In group B the corresponding values for mitral regurgitation were 0-8 (0-3) and 0-8 (0-2) and for tricuspid regurgitation 0-6 (0-3) and 0-9 (0-5) (P < 0.05).

**Discussion**

This prospective study evaluates the long term consequences on left ventricular function after ablation of the atrioventricular junction because of drug refractory atrial fibrillation. Importantly, no discernible negative haemodynamic effect was seen in patients with normal left ventricular function. In patients with impaired left ventricular function a long term improvement of both systolic and diastolic left ventricular function was seen.

**PROCEDURE RELATED FACTORS AFFECTING VENTRICULAR FUNCTION**

The induction of complete heart block and the subsequent pacemaker treatment causes several haemodynamic changes that might influence the outcome in individual patients. These include downregulation of the heart rate, a change from irregular to regular rhythm, and discontinuation of drugs exerting a negative inotropic influence on the left ventricle. These factors should all be positive in respect of left ventricular function.

On the other hand, permanent or intermittent loss of atrioventricular synchrony and an abnormal activation pattern caused by right ventricular pacing, discontinuation of digoxin, and perhaps myocardial injury induced by ablation could work in the opposite direction.

Atrial tachycardia itself has long been suggested as responsible for cardiac dilatation and progressive congestive heart failure. In dogs paced at 190 beats/minute for three months left ventricular ejection fraction decreased from 49% to 29%. After termination of pacing left ventricular systolic function returned to normal within eight weeks. This was confirmed in another study on pigs paced at 240 beats/minute for three weeks. However, diastolic dysfunction still persisted four weeks after pacing had been stopped. In newborn pigs three weeks of left atrial pacing tachycardia caused dilated cardiomyopathy.

Pharmacological control of ventricular rate in patients initially believed to have idiopathic dilated cardiomyopathy with secondary atrial fibrillation resulted in improvement in left ventricular function and complete resolution of symptoms of heart failure. Thus, control of ventricular rate in our patients with drug resistant atrial fibrillation and impaired left ventricular function probably played an important part in improving their left ventricular function. Whether there is a relation between heart rate during arrhythmia and left ventricular dysfunction has to be evaluated in future studies.

Whether irregular heart rhythm in itself can cause impaired myocardial function is unclear. Recently Hariman et al reported the effect of regular ventricular pacing in anaesthetised dogs in which atrial fibrillation had been induced by rapid atrial pacing. During constant ventricular pacing at a rate similar to the ventricular response during fibrillation a small but significant increase of the cardiac output was observed. This phenomenon was more obvious at fast ventricular rate.

We do not believe that withdrawal of antiarrhythmic agents with negative inotropic effects was a major cause for the observed improvement in left ventricular function in our patients. In our group with impaired left ventricular function was taking sotalol at the initial echocardiographic examinations, and he was not taking it at the following echocardiographic examinations. His left ventricular ejection fraction improved at both follow up examinations (43%, 51%, 60%). Six patients in group A and three in group B were initially taking amiodarone. When given orally, however, as in our patients, amiodarone is believed to have little if any negative inotropic action.

**FACTORS INFLUENCING EJECTION FRACTION**

Ejection fraction is dependent on several determinants. Dimensions, heart rate, type of atrial rhythm, synchronism, and age were sufficiently controlled for in this study. The study was not, however, designed to control for other important determinants of ejection fraction such as preload, afterload, contractility, and compliance. Thus we can only speculate about the reasons for the improvement in systolic left ventricular function.

**POSSIBLE EFFECTS OF VENTRICULAR PACING**

As we have said, ventricular apical pacing
could also cause negative haemodynamic effects. Several recent studies have emphasised that a preserved normal ventricular activation pattern is a prerequisite for an optimal left ventricular function.\(^{22, 26}\) As a consequence, right ventricular apical pacing might impair left ventricular function. This effect seems to be independent of the presence or absence of atrioventricular synchrony.\(^{12}\) Furthermore, animal studies have suggested that long term ventricular pacing might cause structural changes in the myocardium.\(^{27, 28}\) Despite this potentially deleterious effect of ventricular pacing we did not observe any significantly negative effects of ventricular pacing in our patients with normal left ventricular function. It should, however, be remembered that the follow up period as well as the number of patients is limited in our study.

Faerestrand et al compared the incidence of valvar insufficiency in patients with ventricular or atrioventricular synchronous pacing.\(^{29}\) During long term follow up they found an increased incidence of both tricuspid and mitral regurgitation in patients with ventricular pacing.

It has recently been shown that the ventricular response during atrial fibrillation can be modified by radiofrequency ablation of the posterior area of the right atrium, which corresponds anatomically to the region that is supposed to be the site for slow posterior pathways in patients with atrioventricular nodal re-entrant tachycardia.\(^{30, 31}\) If these data can be confirmed this might represent a new approach to modify the ventricular rate in atrial fibrillation without the need for permanent ventricular pacing.

Finally, the radiofrequency ablation procedure itself could impair left ventricular function. This seems, however, unlikely as radiofrequency ablation produces only a small well circumscribed area of coagulation necrosis.\(^{32}\) In addition, Twidale et al did not observe any negative short term effects on left ventricular function after the procedure,\(^{15}\) in accordance with the results of this study.

Left ventricular function improved in 11 of the 14 patients with initially impaired function. In one patient left ventricular function did not improve and in two patients it deteriorated. The patient in whom it was unchanged had dilated cardiomyopathy, chronic atrial fibrillation, and a mitral valve prosthesis. The two patients in whom deterioration was observed both had ischaemic heart disease and dilated cardiomyopathy; one also had chronic and the other paroxysmal atrial fibrillation. A lack of beneficial effect on left ventricular function in patients with concomitant ischaemic heart disease is in accordance with previous observations.\(^{11}\)

Thus, among patients with a low ejection fraction most benefited from the procedure. Improvement was observed in four out of five patients with an ejection fraction below 30%.

**Diastolic Function**

With increasing age left ventricular relaxation progressively slows and left ventricular filling...
in early diastole gradually decreases. In our patients' age group the mitral deceleration time is approximately 200–220 ms. Twenty-four of our patients had an Edede below the normal range. In 15 of them (63%) deceleration time increased, in two it was unchanged, and in seven it decreased. Three patients were within the normal range at the first examination two days after ablation. Although both of them remained within the range, one showed a further increase. In one patient deceleration time remained above the normal range.

PREVIOUS STUDIES
Since 1990 five studies, most of them retrospective, have described a positive effect of atrioventricular junctional ablation on left ventricular systolic myocardial function (table 4). In one study this was also confirmed by radionucleide angiography. In the retrospective study by Rodriguez et al four different ablation techniques were used. The described relation between arrhythmia duration and left ventricular dysfunction was, however, not confirmed in our study.

Conclusions
Radiofrequency ablation of the atrioventricular junction and implantation of a pacemaker seems to be a realistic option in patients with rapid atrial fibrillation resistant to pharmacological control or in whom intolerable adverse effects develop. It offers not only symptom relief but also the possibility of long term improvement of impaired left ventricular function.

This study was supported by grants from the Karolinska Institute and the Swedish Society of Medicine.

<table>
<thead>
<tr>
<th>Reference</th>
<th>No of patients</th>
<th>Study design</th>
<th>No of patients with EF &lt; 50% or FS &gt; 27%</th>
<th>No of patients improving</th>
<th>Average % improvement in EF or FS</th>
<th>Length of follow up (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rosengrant, et al1</td>
<td>5</td>
<td>Retrospective</td>
<td>5</td>
<td>4</td>
<td>66</td>
<td>31</td>
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<tr>
<td>Heinz, et al1</td>
<td>10</td>
<td>Prospective</td>
<td>5 (FS)</td>
<td>5</td>
<td>44 (FS)</td>
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<tr>
<td>Twidale, et al12</td>
<td>14</td>
<td>Not stated</td>
<td>10</td>
<td>14</td>
<td>14</td>
<td>1-5</td>
</tr>
<tr>
<td>Rodriguez, et al11</td>
<td>30</td>
<td>Retrospective</td>
<td>12</td>
<td>11</td>
<td>22</td>
<td>12</td>
</tr>
<tr>
<td>Current study</td>
<td>29</td>
<td>Prospective</td>
<td>14</td>
<td>11</td>
<td>40</td>
<td>7</td>
</tr>
</tbody>
</table>

EP, ejection fraction; FS, fraction shortening.

18 Tomita M, Spinale FG, Crawford F, Zile MR. Changes in left ventricular volume, mass, and function during the development and regression of supraventricular tachycardia-induced cardiomyopathy. Circulation 1991;83:635-44.
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Br Heart J 1995 74: 261-267
doi: 10.1136/hrt.74.3.261

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