Ablate and pace revisited: long term survival and predictors of permanent atrial fibrillation

A Queiroga, H J Marshall, M Clune, M D Gammage

Objective: To assess long term mortality and identify factors associated with the development of permanent atrial fibrillation after atrioventricular (AV) node ablation for drug refractory paroxysmal atrial fibrillation.

Design: Retrospective cohort study.

Setting: UK tertiary centre teaching hospital.

Patients: Patients admitted to the University Hospital Birmingham between January 1995 and December 2000.

Interventions: AV node ablation and dual chamber mode switching pacing.

Main outcome measures: Long term mortality and predictors of permanent atrial fibrillation, assessed through Kaplan-Meier curves and logistic regression.

Results: 114 patients (1995–2000) were included: age (mean (SD)), 65 (9) years; 55 (48%) male; left atrial diameter 4 (1) cm; left ventricular end diastolic diameter 5 (1) cm; ejection fraction 54 (17)%.

Indications for AV node ablation were paroxysmal atrial fibrillation in 95 (83%) and paroxysmal atrial fibrillation/flutter in 19 (17%). The survival curve showed a low overall mortality after 72 months (10.5%). Fifty two per cent of patients progressed to permanent atrial fibrillation within 72 months. There was no difference in progression to permanency between paroxysmal atrial fibrillation and paroxysmal atrial fibrillation/flutter (log rank 0.06, p = 0.8). Logistic regression did not show any association between the variables collected and the development of permanent atrial fibrillation, although age over 80 years showed a trend (p = 0.07).

Conclusions: Ablate and pace is associated with a low overall mortality. No predictors of permanent atrial fibrillation were identified, but 48% of patients were still in sinus rhythm at 72 months. These results support the use of dual chamber pacing for paroxysmal atrial fibrillation patients after ablate and pace.

Atrial fibrillation is the most common sustained cardiac rhythm disturbance, occurring in 0.5% of the population at the age of 50 years and in almost 9% of individuals after the eighth decade. Since the modern description of this re-entrant arrhythmia by Moe and colleagues, several different methods of treatment have been proposed. The mainstay of treatment is still pharmacological, but very often patients do not tolerate the haemodynamic consequences of the poorly controlled ventricular rates that occur during atrial fibrillation.

Atrioventricular (AV) node ablation and pacing is the only truly validated non-pharmacological strategy to treat paroxysmal atrial fibrillation. However, this form of treatment is palliative and is not a true cure. Its benefits are mainly on the patients' symptoms, as the arrhythmia persists. Short term progression to permanent atrial fibrillation is common (32% within two months), suggesting that resources may be wasted on unnecessary dual chamber systems.

It is not known which patients are most likely to develop permanent atrial fibrillation in the long term, or, if such patients could be identified, whether they should be given VVIR pacemakers routinely, or indeed whether other forms of pacing might improve their prognosis.

METHODS

Data collection

A six year retrospective analysis (1995 to 2000) was carried out in our unit on patients undergoing AV node ablation plus implantation of a dual chamber mode switching pacemaker for drug refractory paroxysmal atrial fibrillation. AV node ablation was undertaken with insertion of two quadripolar catheters (Cordis) through two femoral vein sheaths. A fixed curve catheter was placed in the right ventricular apex for back up pacing. A deflectable long reach catheter was used to map the proximal AV node. This technique has been described elsewhere. The acceptable site showed balanced atrial and ventricular electrograms plus a sharp His deflection. Radiofrequency energy was applied until complete heart block was achieved with a narrow complex escape rhythm (rate 40–50 beats/min). Pacemaker implantation was done immediately after ablation, and all antiarrhythmic drug treatment was discontinued after the procedure. The atrial lead was positioned in the right atrial appendage, and the ventricular lead in the right ventricular apex. Pacemakers were routinely programmed to DDDR, lower rate 70 beats/min, mode switching on.

Population data were obtained from the patients' medical notes. Variables collected included age, sex, antiarrhythmic drug history, history of direct current (DC) cardioversion, and presence of ischaemic heart disease, valvular heart disease, cardiomyopathy, hypertension, diabetes, or thyroid dysfunction. The date and cause of any deaths were also recorded. Echocardiographic variables were recorded, including left atrial diameter, left ventricular end diastolic diameter (LVEDD), and ejection fraction. Pacing notes and electrophysiology logbooks were examined for data on the development of permanent atrial fibrillation and the presence of atrial fibrillation at the time of ablation. The presence of permanent atrial fibrillation was assessed through atrial rate histograms in Medtronic/St Jude.
Medical pacemakers and through the percentage of pathological atrial rates in Vitatron pacemakers. Pathological atrial rates were defined as any atrial rates above 200 beats/min.

Statistical approach
Continuous variables were expressed as mean (SD). Overall mortality and survival free from permanent atrial fibrillation were evaluated with Kaplan-Meier curves. The significance between the different indications for AV node ablation (paroxysmal atrial fibrillation and paroxysmal atrial fibrillation/flutter) was calculated using the log rank test. Binary logistic regression was used to identify patients at higher risk of developing permanent atrial fibrillation after ablation. All calculations were done with 95% confidence intervals, and a probability value of p < 0.05 was considered significant.

RESULTS
In all, 114 cases were included in the database. The population baseline characteristics are summarised in Table 1. Indications for AV node ablation were paroxysmal atrial fibrillation in 95 patients (83.3%) and paroxysmal atrial fibrillation/flutter in the remaining 19 (16.7%).

Overall mortality was 10.5% at 72 months (fig 1). Only one death was associated with an arrhythmic event and happened six months after the ablation. This patient had a past medical history of ischaemic heart disease, and the arrhythmic episode was associated with an acute coronary syndrome, as stated on the death certificate. A Kaplan-Meier curve showed no difference in mortality between subjects who had purely paroxysmal atrial fibrillation and those who had both atrial fibrillation and atrial flutter (log rank statistic 0.63, p = 0.43).

Although technical echocardiographic comments were available for most patients, only a quarter of the cases had full measurements in their echo reports. Analysis of echocardiographic variables was thus done separately. There was a trend towards an association between age over 80 years and progression to permanent atrial fibrillation, but this did not reach significance (score 3.29; p = 0.07). No other clinical variables were able to predict the progression to permanent atrial fibrillation in this study (Table 2). The echocardiographic variables also failed to predict progression to permanent atrial fibrillation (p = 0.11, p = 0.17, and p = 0.39 for left atrial diameter, LVEDD, and ejection fraction, respectively).

Table 1 Population baseline characteristics (n = 141)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years) (mean (SD))</td>
<td>65 (9)</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>55 (48.2%)</td>
</tr>
<tr>
<td>Female</td>
<td>59 (51.8%)</td>
</tr>
<tr>
<td>Indications for AV node ablation</td>
<td></td>
</tr>
<tr>
<td>Paroxysmal AF</td>
<td>95 (83.3%)</td>
</tr>
<tr>
<td>Paroxysmal AF/flutter</td>
<td>19 (16.7%)</td>
</tr>
<tr>
<td>AF at the time of ablation</td>
<td>23 (20.2%)</td>
</tr>
<tr>
<td>History of DC cardioversion</td>
<td>7 (6.1%)</td>
</tr>
<tr>
<td>Heart disease</td>
<td></td>
</tr>
<tr>
<td>Ischaemic</td>
<td>39 (34.2%)</td>
</tr>
<tr>
<td>Valvar</td>
<td>19 (16.7%)</td>
</tr>
<tr>
<td>Cardiomyopathy</td>
<td>32 (28.1%)</td>
</tr>
<tr>
<td>Systemic disease</td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>31 (27.2%)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>9 (7.9%)</td>
</tr>
<tr>
<td>Thyroid dysfunction</td>
<td>17 (14.9%)</td>
</tr>
<tr>
<td>Number of previous antiarrhythmic drugs (mean (SD))</td>
<td>4 (2)</td>
</tr>
<tr>
<td>Echo parameters (mean (SD))</td>
<td></td>
</tr>
<tr>
<td>LAD</td>
<td>4 (1) cm</td>
</tr>
<tr>
<td>LVEDD</td>
<td>5 (1) cm</td>
</tr>
<tr>
<td>EF</td>
<td>54 (17%)</td>
</tr>
</tbody>
</table>

AF, atrial fibrillation; DC, direct current; EF, ejection fraction; LAD, left atrial diameter; LVEDD, left ventricular end diastolic diameter.
DISCUSSION

AV node ablation and pacing is the standard treatment for drug refractory atrial fibrillation. Brignole and colleagues compared AV junction ablation and pacing with pharmacological treatment and showed that the former was associated with significantly lower scores for heart failure, palpitations, effort dyspnoea, exercise intolerance, and fatigueability. Palpitations were absent in 81% of the ablation group, compared with only 6% of the drug group. However, at the end of six months, permanent atrial fibrillation was present in 24% of subjects in the ablation group and in none of those in the drug group. Similar results were found in another prospective study conducted by Marshall and colleagues, who showed that AV node ablation and dual chamber pacing was better than pharmacological treatment in terms of overall symptoms, palpitations, and breathlessness. Dual chamber mode switch-
an age and sex matched atrial fibrillation control group treated only with antiarrhythmic drugs, and there was no difference in mortality between the two groups. The overall six-year mortality in the ablation group was 22.3%, however. In a meta-analysis, Wood and colleagues pooled data from prospective trials of AV node ablation and pacing and included a total of 1181 patients. Overall mortality at 12 months was 6.3%, which was comparable to the number of deaths in the population followed up in the stroke prevention in atrial fibrillation trial (6.7% at 1.3 years). It is possible that AV node ablation and pacing might have some positive impact on mortality, which could be explained by reversal of tachycardia. Ablation and pacing might have some positive impact on mortality, which could be explained by reversal of tachycardia.

Based on the evidence available, it can be stated that atrioventricular node ablation and permanent pacing does not have an adverse effect on long term survival. The inability to identify predictors of permanent atrial fibrillation after AV node ablation precludes any attempt to use VVI pacing in patients with paroxysmal atrial fibrillation. It also makes it difficult to assess which patients should be offered other treatments, such as overdrive pacing, to improve their prognosis. Overdrive pacing has been suggested as a useful tool in the management of paroxysmal atrial fibrillation, but further evidence is required. Its logical use would be before AV node ablation to avoid the irreversible damage to the electrical conduction system. Nevertheless, if this pacing mode is to be offered after ablation, some benefit to the quality of life would have to be demonstrated as well.

Conclusions
Ablate and pace is associated with a low overall mortality. No predictors of permanent atrial fibrillation could be identified from the variables analysed, but 48% of patients still had evidence of sinus rhythm after six years. These results support the routine use of dual chamber pacing for patients with drug refractory paroxysmal atrial fibrillation after AV node ablation.

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REFERENCES
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