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Long-term outcome of the atrioventricular node ablation and pacemaker implantation for symptomatic refractory atrial fibrillation

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Aims To investigate long-term outcome and to determine predictors of development of heart failure (HF) in patients with atrioventricular (AV) node ablation and permanent right ventricular pacing because of symptomatic refractory atrial fibrillation (AF).
Background Atrioventricular node ablation and subsequent permanent pacing is a well-established therapy for patients with AF. Long-term right ventricular pacing may induce HF.
Methods and results In 121 (45 with previous HF) patients with drug refractory AF, AV node ablation and implantation of a pacemaker was performed. At baseline and after a mean follow-up of 4.3 $\pm$ 3.3 years, New York Heart Association (NYHA) functional class for HF and left ventricular (LV) and atrial diameters were assessed. During and at the end of follow-up, hospitalizations for HF, mortality, and quality of life were assessed using the SF-36 and an AVN-specific questionnaire. No significant changes in NYHA functional class (87 vs. 77% in NYHA I/II at baseline vs. end of follow-up) and LV end diastolic diameter (51 $\pm$ 7 vs. 52 $\pm$ 8 mm) were observed. Left ventricular end systolic diameter decreased (from 37 $\pm$ 9 to 34 $\pm$ 7 mm, $P = 0.03$) and fractional shortening improved (from 28 $\pm$ 10 to 34 $\pm$ 9, $P = 0.02$) in all patients and in patients with previous HF, but not in patients without previous HF. Hospitalizations for HF occurred in 24 patients (20%), predominantly those with previous HF. All-cause mortality occurred in 31 (26%) patients. At the end of follow-up, quality of life was comparable with the control group.
Conclusion Long-term outcome of AV node ablation and permanent pacing is good. Atrioventricular node ablation remains a treatment option for AF.

Introduction

Randomized controlled trials have shown that rate-control therapy is not inferior to rhythm-control therapy.[1,2] Therefore, rate control is adopted more frequently as first-choice therapy. Rate control, however, is not always easy to achieve. In the AFFIRM study, frequent dose adjustments and medication changes were needed, and the strict rate-control target was only achieved in 70% of all patients.[3] Atrioventricular (AV) node ablation and permanent pacing is a well-established therapeutic strategy for atrial fibrillation (AF) and provides highly efficient rate control. It improves symptoms in selected patients.[4–7] Previously, this approach was more often performed, as atrial catheter ablation was not a therapeutic option at that time.[8] However, even in these days, AV node ablation remains an important treatment option in patients in whom rhythm control is ineffective or causing severe adverse effects.[9,10] However, this approach has several limitations, including lifelong right ventricle pacing. Recent studies in patients with an implantable cardioverter defibrillator and left ventricular (LV) dysfunction and studies in patients with initially normal LV function have shown that right ventricular pacing may be associated with an increased risk of impairment of cardiac function and heart failure (HF) and may induce LV dyssynchrony and more AF.[11–16] It is still unclear whether cardiac resynchronization therapy may be beneficial in these patients. A recent meta-analysis reported that cardiac resynchronization therapy in stead of right ventricular pacing may improve outcome, especially in patients with permanent AF and HF.[17]

The purpose of this study was to investigate long-term outcome and to determine predictors of development of

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HF in patients with AV node ablation and permanent right ventricular pacing because of symptomatic refractory AF.

Methods and patients

Patient population

The present patient population consists of 121 consecutive patients with AF, including 45 (37%) patients with a previous hospitalization for HF. All patients underwent a pacemaker implantation and subsequently a successful radiofrequency catheter AV node ablation at the University Medical Center Groningen, Groningen, The Netherlands, between January 1997 and January 2007. Indications for AV node ablation were severely symptomatic paroxysmal or permanent AF despite the use of at least two antiarrhythmic drugs, or severe adverse events on rate- or rhythm-control drugs. Exclusion criteria were New York Heart Association (NYHA) functional class IV and age $<$18 years.

Procedure of pacemaker implantation and atrioventricular node ablation

All patients had a DDD-R or a VVI-R pacemaker implanted before treatment, for paroxysmal or permanent AF, respectively. The ventricular pacing lead was routinely placed in the right ventricular apex and the atrial lead in the right atrial appendage. Standard techniques were used for radiofrequency ablation of the AV node.[18,19] AV node ablation was performed principally (94%) with a quadripolar thermocouple ablation catheter (Biosense Webster Inc., Diamond Bar, CA, USA, curve D or F), accessed through the femoral vein. Ablation was performed using a conventional right-sided approach in every case with radiofrequency energy. Patients were subsequently followed-up in the outpatient clinic. Routinely, pacing was performed in the VVI-R or DDD-R mode with a lower rate of 90 bpm during the first months to prevent any clinical problem related to the sudden reduction of heart rate.[20] Thereafter, the lower rate was decreased to 60–80 bpm.

Data collection

Patient history and baseline characteristics were obtained using patient medical records and the centralized patient record system. All AV node ablation parameters were retrieved from computerized operation reports. In all patients, NYHA functional class was determined before ablation, as well as a transthoracic echocardiography and a 12-lead electrocardiogram were performed. Heart failure at baseline was determined as previous hospitalization for HF. Renal function was assessed by serum creatinine and calculation of the glomerular filtration rate using the simplified modification of diet renal disease equation [$186 \times \text{serum creatinine}^{-1.154}$ (mg/dL) $\times$ $\text{age}^{-0.203}$ (years) $\times$ 0.742 if female].

Follow-up

All patients were seen every 6 months at the outpatient department. Severe cardiovascular complications during follow-up were determined, including hospitalizations for HF and all-cause mortality. Heart failure during follow-up was defined as hospitalization for HF. Both the time of death and the mode of death were determined using information from the treating physicians, or general practitioners. In case of incomplete data, a national registry was used. For that reason, we cannot provide complete data on the cause of cardiovascular mortality. Changes in NYHA functional class, changes in antiarrhythmic drug use, and changes in echocardiographic parameters between baseline and follow-up were assessed. Duration of follow-up was calculated from the time of AV node ablation to death or to the date when the last follow-up data were obtained.

Quality of life

At the end of follow-up, quality of life was assessed using the Medical Outcomes Study Short-Form Health Survey (SF-36) questionnaire.[21] The SF-36 questionnaire is a standardized, validated, generic health survey that has been frequently used in arrhythmia studies.[21] The SF-36 has been translated and validated in the Netherlands.[22] It contains items to assess physical health (e.g. general health perception, physical functioning, role limitations due to physical problems, and bodily pain) and mental health (social functioning, role limitations due to emotional problems, mental health, and vitality). The quality of life of AV node ablation patients was compared with a control population with a comparable age and gender distribution. In addition, we compiled a specific AV node ablation survey form with concise questions on the topic in order to assess additional information about the AV node ablation procedure, previously used for patients who underwent Maze surgery.[23]

Statistical analysis

Descriptive statistics are presented as the mean $\pm$ SD or median (range) for continuous variables and counts with percentages for categorical variables. In case of normally distributed variables, the Student *t*-test, otherwise the Mann–Whitney *U*-test, was used. Paired *t*-tests were used for the comparison of the study population at a different follow-up time. Kaplan–Meier estimates were performed to study the occurrence of hospitalization of HF and all-cause mortality during follow-up in the study population. Adjusted hazard ratios were calculated using Cox proportional hazards regression models. Linearity of the continuous variables with respect to the response variable was assessed by determining the quartiles of their distribution. If no linearity was demonstrated, the variable was further categorized, primarily the median value or on the basis of clinical relevance. All univariate predictors with $P < 0.1$ were tested in a multivariate model, including age, gender, NYHA class, underlying heart disease, renal function, drug therapy, and echocardiographic measurements. In the multivariate model, a variable was excluded when $P \geq 0.05$. A stepwise approach was used, and first-line interactions were investigated. In all analyses, a value of $P < 0.05$ was considered statistically significant.

Results

Patient characteristics

A total of 121 patients with AF were included (*Table 1*), 45 (37%) with a previous hospitalization for HF. Patients with a previous hospitalization for HF were older and had more severe underlying heart disease and impaired renal function (*Table 1*).

Treatment

A single-chamber VVIR pacemaker was implanted in 77 (64%), 13 patients with paroxysmal AF and 64 patients with permanent AF. In 44 (36%) patients, a DDDR pacemaker was implanted, 28 patients with paroxysmal AF and 16 patients with permanent AF ($P < 0.001$). Initial AV node ablation was successful in 111 (92%) patients. A second attempt for ablation was needed in 10 (8%) patients. Eventually, complete AV block was achieved in all patients. Both pacemaker implantation and AV node ablation were uneventful in all patients.

Follow-up

The mean follow-up was 4.3 $\pm$ 3.3 years. At the end of follow-up, patients treated with class I, III, and IV

Table 1 Patients characteristics

Characteristics	Total population ($n = 121$)	Previous hospitalization for HF ($n = 45$)	No previous hospitalization for HF ($n = 76$)	*P*-value
Gender, *n* (%)				1.0
Male	59 (49)	22 (49)	37 (49)	
Female	62 (51)	23 (51)	39 (51)	
Age, years	65 ± 11	70 ± 10	62 ± 12	<0.001
Type of AF, *n* (%)				0.1
Paroxysmal	41 (34)	11 (24)	30 (40)	
Permanent	80 (66)	34 (76)	46 (60)	
Total duration of AF, years	8.4 ± 6.3	8.1 ± 6.7	8.6 ± 6.0	0.7
Number of antiarrhythmic drugs used	2.9 ± 0.9	2.5 ± 0.8	3.0 ± 0.8	0.002
Previous cardioversion, *n* (%)	73 (60)	33 (73)	40 (53)	0.07
Number of cardioversions per patient	1 (0–10)	1 (0–9)	1 (0–10)	0.5
Hypertension, *n* (%)	55 (45)	22 (49)	33 (45)	0.7
Coronary artery disease, *n* (%)	25 (21)	16 (36)	9 (12)	0.005
Significant valve disease, *n* (%)	51 (42)	25 (56)	26 (34)	0.04
Mitral valve disease	45 (37)	23 (51)	22 (29)	0.03
Aortic valve disease	10 (8)	6 (13)	4 (5)	0.2
Previous cardiac surgery, *n* (%)	23 (19)	14 (31)	9 (12)	0.02
Diabetes mellitus, *n* (%)	13 (11)	7 (16)	6 (8)	0.2
Lone AF, *n* (%)	17 (14)	− (0)	17 (22)	<0.001
AF-related complaints, *n* (%)				
Palpitations	70 (57)	25 (56)	45 (59)	0.3
Dyspnoea	51 (42)	26 (58)	25 (33)	0.03
Fatigue	58 (48)	24 (53)	34 (45)	0.7
Dizziness	16 (13)	5 (11)	11 (15)	0.6
Angina	14 (12)	4 (9)	10 (13)	0.6
NYHA functional class, *n* (%)				0.08
I	40 (33)	8 (18)	32 (42)	
II	65 (54)	32 (71)	33 (43)	
III	16 (13)	5 (11)	11 (15)	
Electrocardiogram				
AF at baseline, *n* (%)	96 (79)	42 (93)	54 (71)	0.003
Heart rate (at rest), bpm	94 ± 23	101 ± 23	90 ± 21	0.01
Blood pressure, mmHg				
Systolic	133 ± 19	126 ± 16	136 ± 20	0.049
Diastolic	83 ± 11	81 ± 11	84 ± 11	0.2
LV end diastolic diameter mm)	51 ± 7	54 ± 6	50 ± 7	0.06
LV end systolic diameter (mm)	37 ± 9	42 ± 7	34 ± 8	0.03
Fractional shortening, %	28 ± 10	22 ± 8	32 ± 9	0.005
Creatinine, μmol/L	104 ± 29	113 ± 33	99 ± 26	0.01
Glomerular filtration rate, mL/min/1.73 m^2	62 (19–129)	55 ± 14	65 ± 18	0.001

antiarrhythmic drug therapy had diminished (*Table 2*). Almost all patients (94%) used oral anticoagulation during complete follow-up. At the end of follow-up, most patients were in NYHA functional class I [43 (36%)] and II [37 (31%)], whereas only a few patients were in class III [14 (12%)] and IV [1 (1%)]. No significant changes in NYHA functional class between baseline and end of follow-up were observed ($P = 0.097$).

In the total population and in patients with previous HF, the LV end systolic diameter decreased and fractional shortening improved (*Table 3*). Left ventricular end diastolic diameter did not change. In patients without previous HF, no changes in LV diameters and fractional shortening were observed. Left and right atrial diameters increased in all groups during follow-up (*Table 3*). A marked deterioration of fractional shortening was observed in six patients, all with previous HF.

Hospitalization for heart failure and all-cause mortality

Hospitalization for HF occurred in 24 (20%) patients, predominantly in patients with HF ($n = 15$, *Figure 1*). Multivariate regression analyses revealed previous hospitalization for HF, coronary artery disease, and male gender use as independent predictors for the occurrence of hospitalizations for HF during follow-up (*Table 4*). All-cause mortality occurred in 31 (26%) patients during follow-up, including cardiovascular mortality in 12 patients (10%). In patients with HF, all-cause mortality occurred in 14 patients (31%), including

Table 2 Medication during the study

	Before AV node ablation	After AV node ablation	End of follow-up
Antiarrhythmic drug, *n* (%)			
Class I	2 (6)	− (0)	− (0)
Beta-blocker	26 (77)	22 (65)	19 (56)
Class III	− (0)	− (0)	1 (3)
Class IV	17 (50)	7 (21)	3 (9)
ACE-inhibitor or ARB, *n* (%)	17 (50)	15 (44)	19 (56)
Digoxin, *n* (%)	10 (30)	4 (12)	4 (12)
Oral anticoagulation, *n* (%)	30 (88)	29 (85)	32 (94)
Aspirin, *n* (%)	3 (9)	1 (3)	1 (3)

ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker.

cardiovascular mortality in 6 patients (13%). Regression analyses revealed age $>$ 67 years as the only predictor for all-cause mortality (*Table 4*).

Quality of life

Fifty of the living patients filled out the SF-36 questionnaire. Comparison of these patients with an age- and sex-matched group of healthy controls revealed a comparable quality of life on seven of the eight scales. Atrioventricular node ablation patients had a higher score on the vitality scale (52 vs. 64, $P = 0.048$). The specific AV node ablation survey was completed by 43 patients (*Table 5*). The majority of the patients affirmed, in retrospect, their initial choice for catheter ablation intervention for AF and would recommend it to patients with equal complaints.

Discussion

The present study shows that in patients with AV node ablation and pacemaker implantation, LV function and functional class do not deteriorate after long-term follow-up, even in patients with previous HF. Heart failure necessitating hospitalization, however, predominantly occurred in the latter patients.

Role of atrioventricular node ablation in 2008

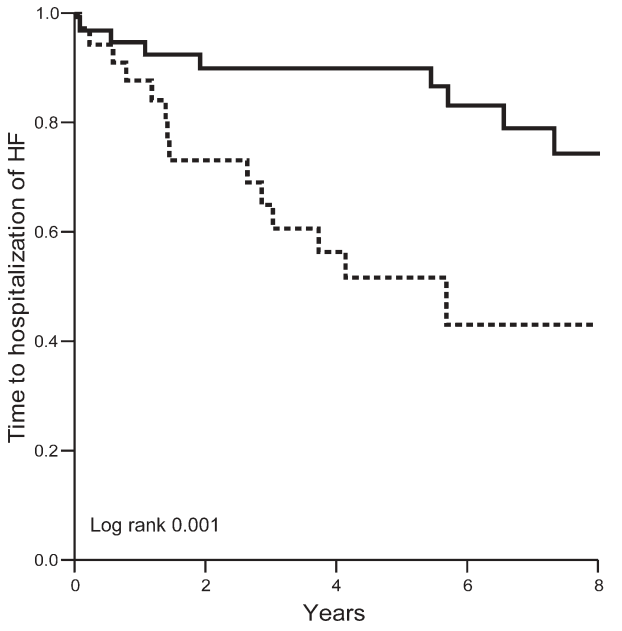
Previously, AV node ablation followed by permanent right ventricular pacing was often performed in symptomatic AF patients after failure on antiarrhythmic drugs.[7,24] At present, AV node ablation is less frequently performed. This is caused by a high success rate of atrial ablation that cures patients from AF[8] and concerns about its safety because of its potential to induce HF.

Atrioventricular node ablation effectively controls the ventricular rate and reduces symptoms and the need for drugs.[4–7,25] However, it also has several limitations. Continuous permanent right ventricular pacing may deteriorate LV function and increase the risk on HF.[11–13,26] At

Table 3 Echocardiographic measurements before atrioventricular node ablation and at the end of follow-up[a]

	Before ablation	End of follow-up	*P*-value
Total population			
Left atrial length, parasternal view, mm	44 ± 8	48 ± 9	<0.001
Left atrial length, apical view, mm	67 ± 10	71 ± 11	<0.001
Left atrial width, apical view, mm	46 ± 10	47 ± 7	0.2
Right atrial length, apical view, mm	60 ± 9	67 ± 8	<0.001
LV end diastolic diameter, mm	51 ± 7	52 ± 8	0.9
LV end systolic diameter, mm	37 ± 9	34 ± 7	0.03
Fractional shortening, %	28 ± 10	34 ± 9	0.02
Septal thickness, mm	10 ± 2	10 ± 2	0.7
Posterior wall thickness, mm	10 ± 2	10 ± 2	0.9
Previous hospitalization for HF			
Left atrial length, parasternal view, mm	46 ± 4	52 ± 7	0.001
Left atrial length, apical view, mm	71 ± 9	75 ± 9	0.2
Left atrial width, apical view, mm	50 ± 6	51 ± 4	0.7
Right atrial length, apical view, mm	63 ± 7	71 ± 7	0.03
LV end diastolic diameter, mm	54 ± 6	52 ± 11	0.6
LV end systolic diameter, mm	42 ± 7	34 ± 8	0.005
Fractional shortening, %	22 ± 8	34 ± 9	0.009
Septal thickness, mm	11 ± 2	11 ± 2	1.00
Posterior wall thickness, mm	10 ± 1	10 ± 2	0.9
No previous hospitalization for HF			
Left atrial length, parasternal view, mm	42 ± 9	46 ± 9	0.04
Left atrial length, apical view, mm	65 ± 9	69 ± 10	0.05
Left atrial width, apical view, mm	41 ± 10	48 ± 12	0.08
Right atrial length, apical view, mm	57 ± 9	66 ± 9	0.001
LV end diastolic diameter, mm	50 ± 7	51 ± 7	0.5
LV end systolic diameter, mm	34 ± 8	34 ± 7	0.7
Fractional shortening, %	32 ± 9	34 ± 8	0.5
Septal thickness, mm	9 ± 2	9 ± 2	0.6
Posterior wall thickness, mm	9 ± 2	9 ± 1	0.9

[a]Echocardiographic data at baseline and at the end of follow-up was available in 59 patients.



Number of patients

No HF	76	36	32	21	11
HF	45	19	12	5	3

Figure 1 Kaplan–Meier survival curve of the time to hospitalization for heart failure in patients without and with previous hospitalization for heart failure (dotted line).

least in part, this seems related to the induction of LV dyssynchrony, even in patients with initially normal LV function.[14,15]

Outcome in our patient population was good, both in patients with and without previous HF. There was no deterioration of LV function. Hospitalizations for HF predominantly occurred in patients previously known with HF. More than 70% of patients were still alive after a follow-up of 5 years, which is in line with the data of Oczan *et al.*[25] In that study, long-term survival was worse in AF patients treated with AV node ablation compared with healthy controls, but comparable with AF patients treated with drug therapy. Furthermore, the present study shows that quality of life was comparable with healthy controls at the end of follow-up.

Previous studies showed dissimilar results with regard to the outcome of cardiac function. This may be related to different patient populations, small numbers of patients, and various follow-up durations in the respective studies. Especially, studies with shorter follow-ups show an improvement of cardiac function, predominantly in patients with an impaired cardiac function at baseline.[27,28] Rodriguez *et al.*[27] showed an improvement of cardiac function in patients with lone AF, with an LV ejection fraction at baseline below 50%. Comparable results were reported by Edner *et al.*[28] in patients with LV dysfunction. They showed, comparable with our data, no effect on cardiac function in patients with a normal LV function. In contrast, more recent reports showed a deterioration of cardiac function after long-term follow-up, also in patients with a normal LV function at baseline.[14,15,29] Szili-Torok *et al.*[29] demonstrated an impairment of LV ejection fraction after a follow-up of 3 months in a mixed group of 12 patients. After a mean follow-up of 4 years, Tops *et al.*[14] observed the occurrence of LV dyssynchrony in 49% ($n = 27$) of their patients. Concurrently, these patients worsened in HF symptoms and showed an impairment of LV function. In contrast, patients who did not develop LV dyssynchrony did not deteriorate in HF symptoms, LV function, or LV volumes. Vernooy *et al.*[15] investigated 55 patients and reported an impairment of LV function in patients with initially normal LV function. Their group consisted of only 28 patients, their follow-up, however, was long (7 years).

The aforementioned data indicate that cardiac function may impair owing to permanent right ventricular pacing inducing LV dyssynchrony, in accordance to data on continuous pacing in patients without AV node ablation. However, as also suggested by the study of Tops *et al.*, not every person is at risk. Ideally, patients at risk for the development of LV dyssynchrony and HF should be identified beforehand. In these patients, biventricular pacing may have beneficial effect.[17,30,31]

The value of biventricular pacing to reduce the risk on HF in these patients still remains to be proved. A recent meta-analysis of 3 randomized trials with 347 patients compared cardiac resynchronization therapy with right ventricular pacing in AF patients treated with AV nodal ablation. Patients included were both patients with permanent AF with drug refractory, severely symptomatic, and uncontrolled heart rates and patients with permanent AF in the setting of HF.[17] No differences in survival, stroke, hospitalization, exercise capacity, or healthcare costs were found. Cardiac resynchronization therapy, however, was associated with an improvement in ejection fraction in two of the three trials, predominantly in patients with permanent AF in the setting of HF.

In the present study, previous hospitalization of HF, coronary artery disease, and male gender were independent predictors of hospitalization for HF during follow-up. This suggests that progression of the underlying heart disease is a main factor in the development of HF. Nevertheless, HF also occurred in patients with a normal cardiac function at baseline.

Quality of life

At the end of follow-up, quality-of-life scores in our study population were comparable with those of age- and gender-matched healthy control groups. No difference could be demonstrated for the study population at seven of the eight subscales of the SF-36. Quality-of-life questionnaire compared with the healthy controls. This in agreement with previous AV node ablation studies.[6] Pharmacological rate-control approaches, as used in the major rate- vs. rhythm-control trials, are not able to improve quality of life of patients with permanent AF to the level of healthy controls.[32]

Limitations

The present analysis was retrospective in design and has therefore important limitations. This design in combination with the relatively small numbers of patients included precludes definite conclusions. Cause–effect relationship cannot be demonstrated and the present data are thus only hypothesis generating. Left ventricular ejection fraction measurements were not available in the majority of

Table 4 Independent determinants of hospitalization for heart failure and all-cause mortality over time

	Univariate HR (95% CI)	Multivariate HR (95% CI)	*P*-value
Hospitalization for HF			
Previous hospitalization for HF	3.8 (1.6–8.7)	3.3 (1.4–7.9)	0.007
Coronary artery disease	3.6 (1.6–8.0)	2.5 (1.1–5.8)	0.03
Male gender	2.8 (1.2–6.8)	3.0 (1.2–7.2)	0.02
HF at baseline	4.6 (1.1–19.6)		
CABG surgery	3.9 (1.5–10.6)		
Hypertension	2.4 (1.0–5.5)		
Valve disease	2.4 (1.0–5.4)		
Glomerular filtration rate < 60 mL/min/1.73 m^2	2.8 (1.8–6.9)		
Creatinine > 97 μmol/L	3.1 (1.2–8.0)		
Systolic blood pressure < 130 mmHg	3.9 (1.3–11.1)		
Age > 67 years	4.2 (1.7–10.6)		
Statin use	3.0 (1.3–6.9)		
ACE-I/ARB use	2.7 (1.2–6.4)		
All-cause mortality			
Age > 67 years	3.0 (1.2–7.8)	3.0 (1.2–7.8)	0.02
Valve disease	2.2 (0.9–5.4)		
Diuretics use	2.4 (0.9–6.0)		

ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CABG, coronary artery bypass graft; CI, confidence interval; HR, hazard ratio.

Table 5 Results of atrioventricular node ablation-specific questionnaire

	Yes, *n* (%)	No, *n* (%)	No opinion, *n* (%)
In your opinion, do you have normal (sinus) rhythm at the moment?	36 (84)	7 (16)	0
Has it been worthwhile to undergo the AV node ablation procedure?	39 (91)	4 (9)	0
Should the operation have been offered to you at an earlier stage?	23 (53)	20 (47)	0
In retrospect, would you undergo the AV node ablation procedure again?	36 (84)	6 (14)	1 (2)
Would you recommend the AV node ablation procedure to another patient with comparable complaints?	38 (89)	4 (9)	1 (2)
Has your need for cardiovascular medication lessened as a result of the AV node ablation procedure?	22 (51)	21 (49)	0
Are you still actively working, e.g. did you maintain your pre-operative job?	17 (40)	25 (58)	1 (2)
If no, were you forced to stop working as a result of your cardiac condition?	17 (40)	22 (51)	4 (9)

patients. Although the follow-up was relatively long, we cannot exclude that in our patients LV dysfunction may have been observed in case of a longer follow-up. Quality of life and the specific AV node ablation survey were not performed in all patients. A selection bias may therefore have been introduced.

Conclusions

Our study underlines the role of AV node ablation for the treatment of AF. Long-term follow-up is good in many patients. However, HF may occur also in patients with normal cardiac function at baseline. Probably, this relates to both progression of the underlying disease and permanent right ventricular pacing. The beneficial effects of biventricular pacing seem obvious but remain to be proved. In our opinion, nowadays, candidates for AV node ablation are patients with permanent symptomatic AF and poor control of the ventricular rate despite pharmacological therapy or patients with severe adverse effects of rate-control drugs.

Conflict of interest: none declared.

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