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**Title**

Sinus Rhythm Restores Ventricular Function in Patients with Cardiomyopathy and No Late Gadolinium Enhancement On Cardiac Magnetic Resonance Imaging who undergo Catheter Ablation for Atrial Fibrillation

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**Abstract**

**Introduction:** Atrial fibrillation (AF) and systolic heart failure (HF) frequently coexist. Restoration of sinus rhythm by catheter ablation may result in a variable improvement in left ventricular (LV) function. Late gadolinium enhancement (LGE) on cardiac magnetic resonance (CMR) imaging identifies irreversible structural change and may predict incomplete recovery of LV function. We prospectively selected AF and symptomatic HF patients without LV LGE and report the impact of AF ablation on LV function.

**Methods and Results:** Patients with AF and symptomatic HF (LV EF<50%) resistant to at least one antiarrhythmic drug and prior electrical cardioversion underwent contrast-enhanced CMR. LGE negative patients underwent pulmonary vein isolation and left atrial roof line with continued antiarrhythmic medications until follow-up CMR 6 months post-ablation. Sixteen patients (aged 55±12 years, mean AF duration 46±41 months, LA size 44±13 ml/m$^2$) underwent AF ablation. At 6 months 15 of 16 patients maintained sinus rhythm and underwent CMR. LV EF increased from 40±10% at baseline to 60±6% ($p<0.001$) and LV end systolic volume index decreased from 52±12 to 36±9 ml/m$^2$ ($p<0.001$). Left atrial size decreased from 44±13 to 36±11 ml/m$^2$ ($p<0.01$).

**Conclusion:** In patients with AF and LV dysfunction in the absence of LGE on CMR, ventricular function normalizes following the restoration of sinus rhythm.

CMR may assist in the selection of AF–HF patients most likely to benefit from catheter ablation.

**Keywords:**

Atrial fibrillation

Ablation

Heart failure

Cardiac magnetic resonance

Late gadolinium enhancement

## Introduction

Atrial fibrillation (AF) occurs in up to 30% of patients with left ventricular (LV) dysfunction, and its prevalence increases with severity of heart failure (HF) symptoms.[1] Restoration of sinus rhythm may lead to recovery of LV function, however, pharmacologic options are limited and AF recurrence is common following electrical cardioversion.[2] Catheter ablation of AF in patients with combined AF and systolic HF (AF-HF) improves LV function,[3-8] exercise capacity and quality of life.[3-5] Despite its apparent efficacy, AF ablation has not been widely applied to this rapidly growing population.[3-6] Barriers to the its wider use include the challenge of selecting patients most likely to benefit, a more complex ablation strategy which may be required in persistent AF, the lower success rate of pulmonary vein isolation alone and the risk of complications.[9, 10]

The AF-CHF trial randomised AF-HF patients to rate control versus rhythm control and did not demonstrate a difference in cardiovascular death, worsening HF, or stroke.[11] However, inclusion criteria permitted recruitment of patients with a low AF burden and few patients underwent catheter ablation. In contrast, catheter ablation studies have been retrospective single centre studies in highly selected patients showing remarkable improvements in LV function. Although ventricular function generally improves with the restoration of sinus rhythm the magnitude of recovery is variable.[10, 12]

Late-gadolinium enhancement (LGE) on contrast-enhanced cardiac magnetic resonance (CMR) imaging identifies regional scar and irreversible structural heart disease.[13, 14] Leong et al demonstrated minimal response to medical therapy in patients with newly-diagnosed dilated cardiomyopathy and ventricular LGE on CMR.[15] The present study aimed to determine the impact of the restoration of sinus rhythm by catheter ablation on LV function by selecting AF-HF patients without LGE on CMR.

## Methods

### *Study Population*

The study was performed at the Alfred Hospital in Melbourne, Australia from January 2009 to December 2011 and was approved by the Alfred Hospital Human Research Ethics Committee. Consecutive patients referred for consideration of AF ablation who were resistant or intolerant to at least one antiarrhythmic agent, had failed at least one attempt at direct current reversion (DCR) and had New York Heart Association (NYHA) class II or greater symptoms despite 6 months of optimal medical therapy were recruited if they had an EF of <50% in the absence LV LGE on contrast-enhanced CMR. Optimal medical therapy included titration of AV nodal blocking agents according to ventricular rate response to AF as assessed by 24 h Holter monitoring.

Exclusion criteria were: an implanted pacemaker or defibrillator precluding CMR, an alternative explanation for cardiomyopathy, left atrial (LA) diameter greater than 55 mm, anticipated cardiac transplantation in less than six months, or life expectancy less than one year.

AF was categorized as paroxysmal where episodes were self-terminating within 7 days or persistent where episodes lasted longer than 7 days or required electrical cardioversion. All recruits underwent repeat contrast-enhanced CMR 6 months after initial ablation.

*Cardiac Magnetic Resonance Imaging*

Pre- and post-ablation CMR was performed using a clinical 1.5-T MRI scanner (Signa HD 1.5-T, GE Healthcare, Wisconsin, USA). Sequences were acquired during breath-holds of 10 to 15 s. Initial cine CMR sequences were performed in 3 standard long-axis (4-, 3- and 2-chamber views) and short-axis slices (basal, mid, and apical), kept identical for each subsequent sequence throughout the CMR examination.[16] From an end-diastolic, 4-chamber, long-axis view, 5 equally spaced short-axis slices were planned such that the 2 outer slices lined up with the tip of the apex and the mitral annulus. The 2 outer slices were then deleted, leaving 3 slices corresponding to typical basal, mid, and apical short-axis views. To calculate LV volume and function, a contiguous short-axis steady state free precession stack was acquired (8 mm slice thickness, no gap), extending from the mitral valve annulus to the LV apex.

Delayed hyperenhancement was obtained in both long- and short-axis views 10 min after a bolus (0.2 mmol/kg bodyweight to a maximum of 20 mmol) of gadolinium-diethylene triamine penta-acetic acid (Magnevist, Schering, Germany) to identify regional fibrosis using a $T_1$ weighted inversion-recovery gradient echo technique (repetition time: 7.1 ms, echo time: 3.1 ms, inversion time individually determined to null the myocardial signal; range: 180 to 250 ms, slice thickness: 8 mm, matrix: 256 × 192, number of acquisitions: 2). For CMR scans performed prior to AF ablation, contrast-enhanced left atriography was performed prior to LGE imaging to generate an LA model for use with a 3D catheter navigation system at the time of ablation.

*Evaluation of LV function and regional fibrosis*

Volumetric analysis of the LV from which EF was derived was performed using the summation of discs method. LA volume was determined using the area-length method. Regional fibrosis was identified by LGE within the LV myocardium, defined quantitatively by a myocardial post-contrast signal intensity 5 standard deviations above that within a reference region of remote non-scarred myocardium within the same slice (Figure 1). Myocardial LGE was defined as being present only if it was identified in two orthogonal views.

*Catheter Ablation*

Patients underwent AF ablation within 72 h of baseline CMR. Antiarrhythmic drugs were not discontinued in preparation for the procedure. Warfarin with a target INR of 2 to 3 was administered for 4 weeks prior to the procedure and catheter ablation was performed on warfarin.

Under general anesthesia, transoesophageal echocardiography was performed to exclude LA thrombus and assist transseptal access. Using a femoral venous approach, a decapolar catheter was placed in the coronary sinus and a hexapolar catheter at the His position. Double transseptal access was obtained with standard techniques using a BRK1 needle and SL1 sheathes (St Jude Medical, MN, USA), and intravenous heparin was administered with a target activated clotting time of 350. A duodecapolar circular mapping catheter (Reflexion Spiral XX, St Jude Medical) was used to guide pulmonary vein isolation, and a 4 mm externally irrigated-tip catheter (Contact Therapy Cool Path Duo, St Jude Medical) was used for mapping and ablation. The left atriogram acquired by CMR was segmented and registered into the electroanatomic mapping system (EnSite NavX, St Jude Medical) as previously described.[17]

Ablation was performed to encircle the left and right sided pulmonary veins (PVs) in pairs 1 to 2 cm from their ostia, except at the anterior aspect of the left PVs, where ablation was performed along the ridge between the LA appendage and the PV ostia. Power was limited to 30 watts reduced to 25W on the posterior wall and temperature to 50°C with energy delivered. The PVs were continuously assessed for electrical disconnection with the endpoint of bidirectional PV-LA block. If AF continued following PV electrical isolation a roof line joining the superior aspects of each wide encirclement ablation ring was deployed followed by electrical cardioversion. Roof line block was confirmed as previously described.[18] If at any stage AF organized to atrial tachycardia (AT), activation mapping and targeted ablation was performed. The procedure was completed with a cavotricuspid isthmus ablation (40 Watts, 45°C) in all patients requiring cardioversion and also in patients where typical atrial flutter had been previously documented. Bidirectional conduction block was confirmed by differential pacing techniques and by the demonstration of widely spaced double potentials along the CTI. A 30-minute waiting period following PVI was strictly employed. Adenosine 18 mg was administered intravenously with the spiral catheter positioned sequentially in the superior PVs to assess for transient PV reconnection.

*Post ablation management*

Following the procedure, patients remained in hospital for 2 nights with cardiac monitoring while warfarin was continued. Patients returned for clinical review at 6 weeks, 3 months, and 6 months, or more frequently if clinically indicated. Electrocardiography and 24 h Holter monitoring was performed at 3 and 6 months post ablation, with patients advised to attend for event monitoring if experiencing palpitations. Arrhythmia recurrence was defined as any documented atrial tachycardia or atrial fibrillation episode lasting greater than 30 seconds that occurred after a 6 week post-ablation blanking period based on symptoms, electrocardiography, and Holter monitor evaluation. A repeat procedure was offered to patients with recurrent atrial arrhythmia who required ongoing antiarrhythmic medication beyond 3 months. A repeat procedure involved re-isolation of PVs and a more extensive approach involving a combination of linear ablation and targeting of fractionated signals. Atrial tachycardias were mapped and ablated using conventional techniques previously described.[19]

Cardiac MRI was repeated six months following successful AF ablation. In patients with AF recurrence the MRI was delayed until 6 months after the second procedure.

*Statistical Analysis*

All data are expressed as mean ± standard deviation (SD). Comparisons between pre- and post-ablation CMR indices were performed using the Student's t test after the Kolmogorov–Smirnov test confirmed normal distribution. A p value of <0.05 was considered significant, and all reported p values are 2-tailed. Analyses were conducted using SPSS software (version 17, SPSS, Chicago, Illinois).

## Results

*Subject Characteristics*

Of 21 patients with reduced LVEF on CMR undergoing AF ablation, 5 patients were excluded due to the presence of fibrosis, yielding a total of 16 consecutive patients who were analyzed in the present study. Subject characteristics are shown in Table 1. Median AF duration was 28 months (interquartile range 14 to 51 months) and no patient had bundle branch block on ECG.

*Procedural characteristics*

Successful pulmonary vein isolation was achieved in all 16 patients. Roof line ablation was performed in all patients with conduction block confirmed in 15/16 (94%). DCR was performed in 10/16 patients for ongoing AF at the end of the procedure. Mean procedural duration was 208±59 min and mean fluoroscopy time 29±9 min. There were no significant complications. Single procedure success was achieved in 14/16 patients at 6 months follow-up with 14/16 on antiarrhythmic medications (amiodarone in 8 and sotalol in 6). Repeat procedures were performed in 2 patients (perimitral atrial flutter in one, and repeat AF ablation for recurrent persistent AF in one).

*Cardiac MR Findings*

Left ventricular EF increased significantly from 40±10 to 60±6% with an absolute increase of 20±10% (Table 1, p<0.001). Ten patients were in AF at the time of CMR and the remainder in sinus rhythm. There was no significant difference in EF between patients in sinus rhythm versus AF at the time of CMR (45±5 vs 37±11%, p=ns). Marked improvement in EF was seen in 15/16 patients, as defined as an increase in EF by >20% or to a value of >55% (Figure 2). There was no significant difference in the absolute increase in EF between patients in sinus rhythm versus AF at the time of CMR (14±7 vs 24±10%, p=ns). All patients were in sinus rhythm at the 6-month CMR. One patient had incomplete recovery of LV function and had recurrent paroxysmal AF post ablation (individual patient details provided in supplementary table). Overall, LV end systolic volume index decreased from 52±12 to 36±9 ml/m$^2$ (p<0.001), without significant changes in LV end diastolic volume index (89±16 to 91±16 ml/m$^2$, p=0.9) or LV mass index (62±18 to 61±26 ml/m$^2$). Left atrial volume index decreased from 44±13 ml to 36±11 ml (p<0.01).

## Discussion

This study demonstrates recovery of LV systolic function following AF ablation in patients with systolic HF in the absence of ventricular LGE on CMR. There was an absolute increase in EF of 20±10% at follow up CMR with normalization of ventricular function in 15 of 16 patients. These findings were paralleled by improvements in LV end-systolic volume index and LA dimensions. Therefore the absence of ventricular LGE prior to AF ablation may be a useful indicator of patients who are likely to experience greatest improvement in systolic function with the restoration of sinus rhythm following catheter ablation.

*Atrial fibrillation and heart failure – the chicken or the egg*

AF and HF have emerged as epidemics over the last decade, accounting for an increasing proportion of the total burden of cardiovascular disease.[20] In clinical practice, AF and HF are encountered regularly and frequently occur in combination, where they are associated with adverse outcomes.[21, 22] Both conditions share common risk factors, and each has a propensity of causing the other.[1] The temporal sequence of AF and HF is often unclear when the index presentation is with both conditions. Pre-existing HF may promote AF through atrial stretch resulting in structural and electrical remodeling with the electrophysiologic sequelae of conduction slowing and block.[23] Conversely, AF may perpetuate HF through the loss of atrial transport, irregular rapid ventricular rhythm, and tachycardia-induced cardiomyopathy.[24] Although the restoration of sinus rhythm may be attractive, this is challenging with limited pharmacologic agents, significant AF recurrence, and randomized studies not supportive of a rhythm control strategy.

*Prior studies*

Although catheter ablation has been associated with significant improvements in LV function, the magnitude of improvement has been variable ranging from 9% to 21% in single centre studies.[3-8, 25-27] A recent meta-analysis of 9 such studies including a total of 354 patients found the mean increase in EF to be 11.1% (95% CI: 7.1 to 15.2) at follow-up, compared to 20% in our study. One study failed to demonstrate a significant improvement in LV function with catheter ablation, likely due to the inclusion of a substantial proportion (78%) of patients with coronary artery disease [3]. Meta-regression analysis demonstrated the incidence of coronary artery disease in the individual studies to be inversely related to the improvement of EF after ablation, suggesting that the presence of ischemic/infarcted substrate reduces the magnitude of LV recovery following ablation.[12]

A rapid ventricular response to AF is a well-recognized cause of reversible LV systolic dysfunction. In the landmark study by Hsu and co-workers a substantial improvement in LV EF from 35% to 50% was demonstrated as early as one month following AF ablation.[4] Improvements in ventricular function were present in patients irrespective of the adequacy of rate control or concomitant structural heart disease although the degree of recovery varied. In the present study all patients underwent holter monitoring to ensure adequacy of ventricular rate control prior to study enrollment.

Myocardial fibrosis is a hallmark of cardiomyopathy and is generally considered irreversible. Discrete scar may be seen in ischemic,[28] hypertrophic,[29] and idiopathic dilated cardiomyopathy with characteristic topography[14]. Contrast-enhanced CMR is a well-established technique that identifies regional ventricular fibrosis by the presence of LGE. In previous studies, the presence of LGE in patients with existing dilated cardiomyopathy has been established as an independent predictor of mortality and morbidity.[14, 30, 31] Leong et al reported the response to medical therapy in patients with newly diagnosed dilated cardiomyopathy who underwent CMR. Myocardial fibrosis was independently associated with a lack of improvement in ventricular function independent of clinical and echocardiographic indices.[15] Based on earlier studies we hypothesized that the absence of LGE may be predictive of recovery of left ventricular function with the restoration of sinus rhythm. The LVEF increased by an absolute 20±10% following AF ablation in LGE negative patients, and recovered by >20% or to a value of ≥55% in 15 of 16 patients.

*Study Limitations*

Although patient numbers were limited reflecting the small number of AF-HF patients suitable for AF ablation who could undergo CMR, the findings were uniform and highly significant. LGE positive patients were excluded from the present study as less likely to benefit from catheter ablation given discrete regional scar and as such this study does not provide information regarding the response of LGE positive patients to the restoration of sinus rhythm. The majority of patients continued antiarrhythmic medication post-ablation as the primary aim was to assess the recovery of LV function following the restoration of sinus rhythm rather than the efficacy of a stand alone ablation strategy.[32] The findings in the present study are hypothesis generating and provide a platform for a multicentre randomized study comparing medical therapy with catheter ablation in LGE positive and negative patients.

## Conclusion

In patients with AF and LV dysfunction in the absence of LGE on CMR, ventricular function normalizes following the restoration of sinus rhythm.

## Author Contributions

Concept/design: Ling LH, Taylor AJ, Kalman JM, Kistler PM. Data acquisition, analysis, and interpretation: all authors. Drafting article: Ling LH. Critical revision of the article: all authors. Statistics: Ling LH, Kistler PM. Approval of article: all authors.

## References


1. Balasubramaniam R, Kistler PM. Atrial fibrillation in heart failure: the chicken or the egg? Heart Apr 2009;95:535-539.
2. Pisters R, Nieuwlaat R, Prins MH, et al. Clinical correlates of immediate success and outcome at 1-year follow-up of real-world cardioversion of atrial fibrillation: the Euro Heart Survey. Europace May 2012;14:666-674.
3. Chen MS, Marrouche NF, Khaykin Y, et al. Pulmonary vein isolation for the treatment of atrial fibrillation in patients with impaired systolic function. J Am Coll Cardiol Mar 17 2004;43:1004-1009.
4. Hsu LF, Jais P, Sanders P, et al. Catheter ablation for atrial fibrillation in congestive heart failure. N Engl J Med Dec 2 2004;351:2373-2383.
5. Khan MN, Jais P, Cummings J, et al. Pulmonary-vein isolation for atrial fibrillation in patients with heart failure. N Engl J Med Oct 23 2008;359:1778-1785.
6. De Potter T, Berruezo A, Mont L, et al. Left ventricular systolic dysfunction by itself does not influence outcome of atrial fibrillation ablation. Europace Jan 2010;12:24-29.
7. Gentlesk PJ, Sauer WH, Gerstenfeld EP, et al. Reversal of left ventricular dysfunction following ablation of atrial fibrillation. J Cardiovasc Electrophysiol Jan 2007;18:9-14.
8. MacDonald MR, Connelly DT, Hawkins NM, et al. Radiofrequency ablation for persistent atrial fibrillation in patients with advanced heart failure and severe left ventricular systolic dysfunction: a randomised controlled trial. Heart May 2011;97:740-747.
9. Dagres N, Hindricks G, Kottkamp H, et al. Complications of atrial fibrillation ablation in a high-volume center in 1,000 procedures: still cause for concern? J Cardiovasc Electrophysiol Sep 2009;20:1014-1019.

**10.** Wilton SB, Fundytus A, Ghali WA, et al. Meta-analysis of the effectiveness and safety of catheter ablation of atrial fibrillation in patients with versus without left ventricular systolic dysfunction. Am J Cardiol Nov 1 2010;106:1284-1291.

**11.** Talajic M, Khairy P, Levesque S, et al. Maintenance of sinus rhythm and survival in patients with heart failure and atrial fibrillation. J Am Coll Cardiol Apr 27 2010;55:1796-1802.

**12.** Dagres N, Varounis C, Gaspar T, et al. Catheter ablation for atrial fibrillation in patients with left ventricular systolic dysfunction. A systematic review and meta-analysis. J Card Fail Nov 2011;17:964-970.

**13.** Bello D, Shah DJ, Farah GM, et al. Gadolinium cardiovascular magnetic resonance predicts reversible myocardial dysfunction and remodeling in patients with heart failure undergoing beta-blocker therapy. Circulation Oct 21 2003;108:1945-1953.

**14.** Wu KC, Weiss RG, Thiemann DR, et al. Late gadolinium enhancement by cardiovascular magnetic resonance heralds an adverse prognosis in nonischemic cardiomyopathy. J Am Coll Cardiol Jun 24 2008;51:2414-2421.

**15.** Leong DP, Chakrabarty A, Shipp N, et al. Effects of myocardial fibrosis and ventricular dyssynchrony on response to therapy in new-presentation idiopathic dilated cardiomyopathy: insights from cardiovascular magnetic resonance and echocardiography. Eur Heart J Mar 2012;33:640-648.

**16.** Taylor AJ, Al-Saadi N, Abdel-Aty H, Schulz-Menger J, Messroghli DR, Friedrich MG. Detection of acutely impaired microvascular reperfusion after infarct angioplasty with magnetic resonance imaging. Circulation May 4 2004;109:2080-2085.

**17.** Kistler PM, Earley MJ, Harris S, et al. Validation of three-dimensional cardiac image integration: use of integrated CT image into electroanatomic mapping system to perform catheter ablation of atrial fibrillation. J Cardiovasc Electrophysiol Apr 2006;17:341-348.

**18.** Takahashi Y, Rotter M, Sanders P, et al. Left atrial linear ablation to modify the substrate of atrial fibrillation using a new nonfluoroscopic imaging system. PACE Jan 2005;28 Suppl 1:S90-93.

**19.** Rostock T, Drewitz I, Steven D, et al. Characterization, mapping, and catheter ablation of recurrent atrial tachycardias after stepwise ablation of long-lasting persistent atrial fibrillation. Circ Arrhythm Electrophysiol Apr 2010;3:160-169.

**20.** Braunwald E. Shattuck lecture--cardiovascular medicine at the turn of the millennium: triumphs, concerns, and opportunities. N Engl J Med Nov 6 1997;337:1360-1369.

**21.** Dries DL, Exner DV, Gersh BJ, Domanski MJ, Waclawiw MA, Stevenson LW. Atrial fibrillation is associated with an increased risk for mortality and heart failure progression in patients with asymptomatic and symptomatic left ventricular systolic dysfunction: a retrospective analysis of the SOLVD trials. Studies of Left Ventricular Dysfunction. J Am Coll Cardiol Sep 1998;32:695-703.

**22.** Olsson LG, Swedberg K, Ducharme A, et al. Atrial fibrillation and risk of clinical events in chronic heart failure with and without left ventricular systolic dysfunction: results from the Candesartan in Heart failure-Assessment of Reduction in Mortality and morbidity (CHARM) program. J Am Coll Cardiol May 16 2006;47:1997-2004.

**23.** Sanders P, Morton JB, Davidson NC, et al. Electrical remodeling of the atria in congestive heart failure: electrophysiological and electroanatomic mapping in humans. Circulation Sep 23 2003;108:1461-1468.

**24.** Avitall B, Bi J, Mykytsey A, Chicos A. Atrial and ventricular fibrosis induced by atrial fibrillation: evidence to support early rhythm control. Heart Rhythm Jun 2008;5:839-845.

**25.** Choi AD, Hematpour K, Kukin M, Mittal S, Steinberg JS. Ablation vs medical therapy in the setting of symptomatic atrial fibrillation and left ventricular dysfunction. Congest Heart Fail Jan-Feb 2010;16:10-14.

**26.** Efremidis M, Sideris A, Xydonas S, et al. Ablation of atrial fibrillation in patients with heart failure: reversal of atrial and ventricular remodelling. Hell J Cardiol Jan-Feb 2008;49:19-25.

**27.** Lutomsky BA, Rostock T, Koops A, et al. Catheter ablation of paroxysmal atrial fibrillation improves cardiac function: a prospective study on the impact of atrial fibrillation ablation on left ventricular function assessed by magnetic resonance imaging. Europace May 2008;10:593-599.

**28.** Fieno DS, Kim RJ, Chen EL, Lomasney JW, Klocke FJ, Judd RM. Contrast-enhanced magnetic resonance imaging of myocardium at risk: distinction between reversible and irreversible injury throughout infarct healing. J Am Coll Cardiol Nov 15 2000;36:1985-1991.

**29.** Moon JC, Reed E, Sheppard MN, et al. The histologic basis of late gadolinium enhancement cardiovascular magnetic resonance in hypertrophic cardiomyopathy. J Am Coll Cardiol Jun 16 2004;43:2260-2264.

**30.** Leyva F, Taylor RJ, Foley PW, et al. Left ventricular midwall fibrosis as a predictor of mortality and morbidity after cardiac resynchronization therapy in patients with nonischemic cardiomyopathy. J Am Coll Cardiol Oct 23 2012;60:1659-1667.

**31.** Assomull RG, Prasad SK, Lyne J, et al. Cardiovascular magnetic resonance, fibrosis, and prognosis in dilated cardiomyopathy. J Am Coll Cardiol Nov 21 2006;48:1977-1985.

**32.** Mulder AA, Wijffels MC, Wever EF, Kelder JC, Boersma LV. Arrhythmia detection after atrial fibrillation ablation: value of incremental monitoring time. PACE Feb 2012;35:164-169.

**Figure Legend**

Figure 1

Representative cardiac MRI delayed enhancement (DE): mid left ventricle short axis in 3 patients with normal/ no DE, focal inferior wall DE, and anteroseptal mid-wall DE.

Figure 2

Left ventricular ejection fraction increased from 40±10% on baseline cardiac magnetic resonance imaging (CMR) to 60±6% at 6 months post-atrial fibrillation ablation ($p<0.001$).

## Figures

*Figure 1: Representative cardiac MRI delayed enhancement (DE): mid left ventricle short axis in 3 patients with normal, focal inferior wall DE, and anteroseptal mid-wall DE.*

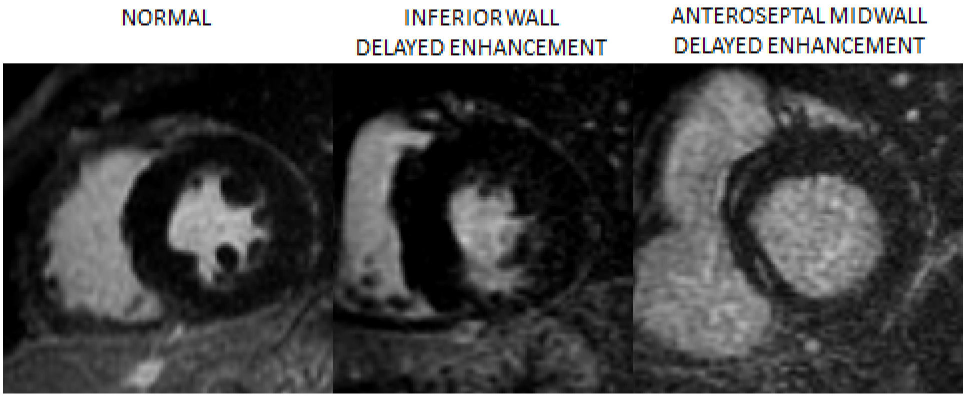
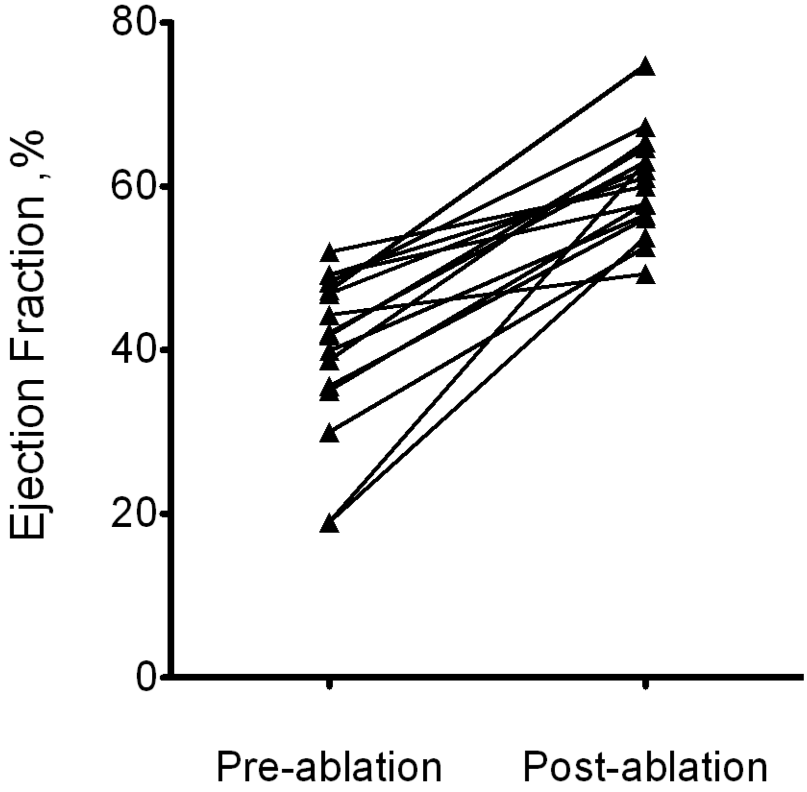


*Figure 2: Ejection Fraction at Baseline and at 6 Months post-AF ablation*



**Table**

*Table 1: Subject characteristics*

| | |
|---|---|
| **General characteristics** | |
| Age, years | 52±11 |
| Male gender, n (%) | 14 (88) |
| Mean resting heart rate, bpm | 71±15 |
| NYHA class II to IV | 16 (100) |
| Paroxysmal AF | 4 (25) |
| Persistent AF | 12 (75) |
| AF duration, months (mean, range) | 39 (1-156) |
| AF ablation performed in SR | 6 (38) |
| AF ablation performed in AF < 1 year | 2 (12) |
| AF ablation performed in AF > 1 year | 8 (50) |
| **Co-morbidities, n (%)** | |
| Diabetes | 0 (0) |
| Hypertension | 6 (38) |
| Ischaemic heart disease | 1 (6) |
| **Cardiac medications, n (%)** | |
| ACE-inhibitor/ARB | 9 (56) |
| β-blocker | 13 (81) |
| Calcium channel blockers | 0 (0) |
| Aldosterone antagonist | 0 (0) |
| Digoxin | 3 (19) |
| Frusemide | 3 (19) |
| Warfarin | 11 (69) |
| AADs tried (mean±SD) | 1.6±0.7 |
| Sotalol | 9 (56) |
| Flecainide | 8 (50) |
| Amiodarone | 5 (31) |
| **Baseline CMR findings** | |
| LV ejection fraction (%) | 40±10 |
| LV end diastolic volume index (ml/m$^2$) | 89±16 |
| LV end systolic volume index (ml/m$^2$) | 52±12 |
| LV mass index (g/m$^2$) | 62±18 |
| LA volume index (ml/m$^2$) | 44±13 |
| **Post-ablation CMR findings** | |

| | |
|---|---|
| LV ejection fraction (%) | 60±6* |
| LV end diastolic volume index (ml/m$^2$) | 91±16 |
| LV end systolic volume index (ml/m$^2$) | 36±9* |
| LV mass index (g/m$^2$) | 61±26 |
| LA volume index (ml/m$^2$) | 36±11* |

*$p<0.01$ for comparisons between post-ablation and baseline CMR findings. ARB – angiotensin receptor blocker; AAD – antiarrhythmic drug; CMR – cardiac magnetic resonance; LA – left atrial; LV – left ventricular.