Non-pharmacological interventions in atrial fibrillation

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Presenter Disclosure Information

The presenter has received honoraria for participation in lectures and advisory boards from the following pharmaceutical and biotechnology companies:

- AstraZeneca,
- Bard,
- Bayer Healthcare,
- Boehringer Ingelheim,
- Boston Scientific,
- Bristol-Myers Squibb,
- ELPEN,
- Galenica,
- Lilly,
- Medtronic,
- Menarini,
- MSD,
- Pfizer,
- Sanofi,
- Servier,
- StJude,
- Unifarma,
- Vianex.
“Οκόσα φάρμακα ουκ ιήται, σίδηρος ιήται. Όσσα σίδηρος ουκ ιήται, πυρ ιήται, όσσα δε πυρ ουκ ιήται ταύτα χρη νομίζειν ανίητα”

Ιπποκράτης (460-377 π.χ.)
3.1 Incidence and prevalence of atrial fibrillation

In 2010, the estimated numbers of men and women with AF worldwide were 20.9 million and 12.6 million, respectively, with higher incidence and prevalence rates in developed countries. One in four middle-aged adults in Europe and the US will develop AF. By 2030, 14–17 million AF patients are anticipated in the European Union, with 120,000–215,000 newly diagnosed patients per year. Estimates suggest an AF prevalence of approximately 3% in adults aged 20 years or older, with greater prevalence in older persons and in patients with conditions such as hypertension, heart failure, coronary artery disease (CAD), valvular heart disease, obesity, diabetes mellitus, or chronic kidney disease (CKD). The increase in AF prevalence can be attributed both to better detection of silent AF, alongside increasing age and conditions predisposing to AF.
ΕΠΙΠΟΛΑΣΜΟΣ ΚΟΛΠΙΚΗΣ ΜΑΡΜΑΡΥΓΗΣ ΣΤΟΝ ΕΛΛΗΝΙΚΟ ΠΛΗΘΥΣΜΟ (>14 ΕΤΩΝ)
ΔΕΔΟΜΕΝΑ ΑΠΟ ΤΟ ΠΡΟΓΡΑΜΜΑ ΠΡΟΛΗΨΗΣ ΤΟΥ ΕΛΙΚΑΡ

Συνολικός αριθμός συμμετεχόντων: 44.956 άτομα > 14 ετών
Δεδομένα για ΚΜ από 2011 ως 2013: 6970 ερωτηματολόγια

πληθυσμός > 14 ετών: 9148309 (84,80%)

<table>
<thead>
<tr>
<th></th>
<th>Αριθμός ασθενών</th>
<th>Κολπική Μαρμαρυγή (&gt;14 ετών)</th>
<th>ΚΜ στο σύνολο του πληθυσμού</th>
<th>Κολπική Μαρμαρυγή (&gt;75 ετών)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2011</td>
<td>3150</td>
<td>3,5%</td>
<td>2,9%</td>
<td>11%</td>
</tr>
<tr>
<td>2012</td>
<td>1570</td>
<td>3,9%</td>
<td>3,3%</td>
<td>11,5%</td>
</tr>
<tr>
<td>2013</td>
<td>2250</td>
<td>3,4%</td>
<td>2,9%</td>
<td>10,5%</td>
</tr>
</tbody>
</table>
Rate vs. rhythm control and adverse outcomes among European patients with atrial fibrillation

Yanish Purmah\textsuperscript{1\dagger}, Marco Proietti\textsuperscript{1,2\dagger}, Cecilé Laroche\textsuperscript{3}, Michal Mazurek\textsuperscript{1,4}, Dimitrios Tahmatzidis\textsuperscript{5}, Giuseppe Boriani\textsuperscript{6,7}, Salvatore Novo\textsuperscript{8}, and Gregory Y.H. Lip\textsuperscript{1,9\ast} on behalf of the EORP-AF General Pilot Registry Investigators

Table 5  Cox regression analysis for all-cause death

<table>
<thead>
<tr>
<th>Variable</th>
<th>HR</th>
<th>95% CI</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (per year)</td>
<td>1.04</td>
<td>1.02--1.07</td>
<td>0.0012</td>
</tr>
<tr>
<td>Rate control (vs. rhythm control)</td>
<td>2.83</td>
<td>1.14--7.05</td>
<td>0.0256</td>
</tr>
<tr>
<td>Previous TIA</td>
<td>2.14</td>
<td>1.15--3.99</td>
<td>0.0159</td>
</tr>
<tr>
<td>Chronic heart failure</td>
<td>2.76</td>
<td>1.65--4.61</td>
<td>0.0001</td>
</tr>
<tr>
<td>Chronic kidney disease</td>
<td>2.01</td>
<td>1.31--3.09</td>
<td>0.0015</td>
</tr>
<tr>
<td>Diabetes</td>
<td>2.02</td>
<td>1.33--3.08</td>
<td>0.0010</td>
</tr>
<tr>
<td>Physical activity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None (ref.)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Occasional</td>
<td>0.40</td>
<td>0.23--0.67</td>
<td>0.0005</td>
</tr>
<tr>
<td>Regular</td>
<td>0.29</td>
<td>0.11--0.72</td>
<td>0.0080</td>
</tr>
<tr>
<td>Intense</td>
<td>0.65</td>
<td>0.16--2.70</td>
<td>0.5540</td>
</tr>
</tbody>
</table>

Figure 3  Kaplan–Meier curves for all-cause death according to baseline strategy.
Non-pharmacological interventions in atrial fibrillation

I. LAA closure

II. Pacing +/- Atrioventricular node ablation

III. Atrial Fibrillation ablation
Left Atrial Appendage Is Potentially Thrombogenic

«Increased LAA width and length correlates with thromboembolic risk»

«~90% of atrial thrombi occur in the LAA»
Devices for Transcatheter LAA Closure
(Clinical Introduction in Europe)

2001
PLAATO
(abandoned)

2002
Amplatzer
ASD/PFO/VSD
Occluders

2002
Watchman

2008
LAA Transcatheter Patch

2008
Amplatzer Cardiac Plug
Amulet

2011
Wavecrest

2015
Occlutech

2015
Ultrasept

2016
LAMbre

2016
pfm LAA Occluder
PROTECT-AF & PREVAIL Combined Analysis
Reduction in Major Bleeding (>6-mo)

➢ Late Major Bleeding was Reduced by 71%

Free of Major Bleeding Event (%)

HR = 0.29
p<0.001

/watchman
Warfarin

Aspirin

(as presented by Dr Reddy at ACC 2017)

Spurious Signal of Hemorrhagic stroke...

...more than just the play of chance

FDA Executive Summary

Prepared for the October 8, 2014 meeting of the Circulatory System Devices Panel

P130013

Boston Scientific WATCHMAN® Left Atrial Appendage Closure Therapy

(as presented by John Mandrola at ESC 2017)
After the FDA Review

7 vs 11

(as presented by John Mandrola at ESC 2017)
Single ASA therapy post-LAAO

Transcatheter left atrial appendage occlusion in patients with atrial fibrillation and a high bleeding risk using aspirin alone for post-implant antithrombotic therapy

Kasper Korsholm¹, MD; Kirsten Melgaard Nielsen¹, MD, PhD; Jesper Møller Jensen¹, MD, PhD; Henrik Kjærulf Jensen¹, MD, PhD, DMSæ; Grethe Andersen², MD, DMSæ; Jens Erik Nielsen-Kudsk¹, MD, DMSæ

- ACP or Amulet: 107 patients
- Single ASA therapy post-LAAO
- 2.3 years median
- DAT 1.9%
- 61% Stroke – 57% major bleeding reduction

EuroIntervention 2016
Major bleeding events with aspirin are similar to those caused by VKAs and NOACs.

**AVERROES study**

![Bar chart showing major bleeding events with aspirin compared to VKAs and NOACs]

**Birmingham Atrial Fibrillation Treatment of the Aged Study (BAFTA, Mant et al., Lancet 2007)**

<table>
<thead>
<tr>
<th></th>
<th>Warfarin</th>
<th>Aspirin</th>
<th>Warfarin vs aspirin</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Death</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All causes</td>
<td>107</td>
<td>108</td>
<td>0.95 (0.72-1.26)</td>
</tr>
<tr>
<td>Fatal primary endpoint</td>
<td>15</td>
<td>23</td>
<td>0.63 (0.31-1.26)</td>
</tr>
<tr>
<td>Other vascular death*</td>
<td>41</td>
<td>34</td>
<td>1.16 (0.72-1.88)</td>
</tr>
<tr>
<td>Non-vascular death*</td>
<td>51</td>
<td>51</td>
<td>0.96 (0.64-1.45)</td>
</tr>
<tr>
<td><strong>Secondary vascular outcomes (fatal and non-fatal)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All strokes</td>
<td>33</td>
<td>61</td>
<td>0.52 (0.33-0.80)</td>
</tr>
<tr>
<td>All strokes plus TIA</td>
<td>40</td>
<td>70</td>
<td>0.55 (0.36-0.82)</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>15</td>
<td>15</td>
<td>0.96 (0.44-2.11)</td>
</tr>
<tr>
<td>Heart failure</td>
<td>38</td>
<td>23</td>
<td>1.59 (0.92-2.79)</td>
</tr>
<tr>
<td>Other vascular events†</td>
<td>34</td>
<td>45</td>
<td>0.71 (0.44-1.13)</td>
</tr>
<tr>
<td>All non-stroke vascular events</td>
<td>78</td>
<td>76</td>
<td>0.97 (0.70-1.35)</td>
</tr>
<tr>
<td><strong>Haemorrhage (fateful and non-fatal)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Major extracranial haemorrhage</td>
<td>18</td>
<td>20</td>
<td>0.87 (0.43-1.73)</td>
</tr>
<tr>
<td>Other hospital admission for haemorrhage</td>
<td>24</td>
<td>19</td>
<td>1.22 (0.64-2.36)</td>
</tr>
<tr>
<td>All major haemorrhages (including intracranial and haemorrhagic stroke)</td>
<td>25</td>
<td>25</td>
<td>0.96 (0.53-1.75)</td>
</tr>
<tr>
<td><strong>Composite outcomes</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Major vascular events (stroke, myocardial infarction, pulmonary embolus, t vascular death)</td>
<td>76</td>
<td>100</td>
<td>0.73 (0.53-0.99)</td>
</tr>
<tr>
<td>Primary events plus major haemorrhage</td>
<td>39</td>
<td>64</td>
<td>0.59 (0.38-0.89)</td>
</tr>
</tbody>
</table>

Analyses are censored at first event, so the composite outcomes are not the sum of the individual categories of event. *Includes deaths that occurred after non-fatal primary endpoints, including four deaths from stroke (as ‘other vascular death’). †Other events leading to hospital admission or death, such as angina, deep vein thrombosis, acute bowel ischaemia, pulmonary embolism, acute arrhythmia, and elective vascular surgery. ‡There were five pulmonary emboli, one in the warfarin group and four in the aspirin group.
Termination of anticoagulation therapy at 45 days after concomitant atrial fibrillation catheter ablation and left atrial appendage occlusion resulting in device-related thrombosis and stroke

Steven K Carlson, MD, Rahul N Doshi, MD, FACC, FHRS

From the Keck School of Medicine, University of Southern California, Los Angeles, California.

KEY TEACHING POINTS

- Concomitant atrial fibrillation catheter ablation and left atrial appendage occlusion is increasing in incidence.
- There is currently no consensus about the postprocedure anticoagulation regimen for concomitant atrial fibrillation catheter ablation and left atrial appendage occlusion.
- Based on available evidence, it appears that the current best practice is to treat with at least 2 months of oral anticoagulation followed by 4 months of aspirin and clopidogrel therapy and then lifelong aspirin therapy.
- A consensus statement on the postprocedure antithrombotic regimen for this procedure is needed quickly to prevent confusion among the health care community.

...warfarin was discontinued because she was out from her procedure for 45 days
The WATCHMAN should only be used in patients who:
• have atrial fibrillation not related to heart valve disease
• are at increased risk for a stroke
• are recommended for blood thinning medicines
• are suitable for warfarin
• have an appropriate reason to seek a non-drug alternative to warfarin
".... after PCI in anticoagulation/TAH contraindicated patients"

European Heart Journal
doi:10.1093/eurheartj/ehu278
Non-pharmacological interventions in atrial fibrillation

I. LAA closure

II. Pacing +/- Atrioventricular node ablation

III. Atrial Fibrillation ablation
Increased duration, dispersion and variance of P wave duration are common in patients with atrial fibrillation

(P dispersion and P variation predict Atrial Fibrillation)

Shorter P waves are associated with lower prevalence of Atrial Fibrillation

Can we Prevent AF by Alternate site Pacing?

Atrial Septal Pacing? (ASPECT)
Multisite Pacing? (DAPPAF)
ATRIAL PACING ALGORITHMS TO PREVENT ATRIAL FIBRILLATION

‘Atrial Preference Pacing’ algorithm (Boston Scientific/Guidant) devices increases the atrial pacing rate when sensed atrial events occur.

‘Atrial Rate Stabilization’ algorithm (Medtronic/Vitatron) the atria are electively paced after a PAC to avoid short-long cycles.

‘Atrial Fibrillation Suppression’ algorithm, (SJM) atrial pacing rate increases when two native P waves (consecutive or not) are sensed in a sensing window.

ADOPT, AOP, AFTherapy study, ASSERT, CAOP

❖ Patient discomfort from high-rate atrial pacing
❖ Risk of tachycardia-induced cardiomyopathy
### Atrioventricular node ablation + pacing

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class</th>
<th>Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrioventricular node ablation should be considered to control heart rate in patients unresponsive or intolerant to intensive rate and rhythm control therapy, accepting that these patients will become pacemaker dependent.</td>
<td>IIa</td>
<td>B</td>
</tr>
</tbody>
</table>

...Modern era.... potential for advanced rhythm control strategies such as catheter ablation, surgical ablation or hybrid approaches, thereby reducing the need for permanent pacemaker implantation.
Non-pharmacological interventions in atrial fibrillation

I. LAA closure

II. Pacing +/- Atrioventricular node ablation

III. Atrial Fibrillation ablation
Initiation of long term rhythm control therapy in symptomatic patients with atrial fibrillation
Mixed treatment comparison of dronedarone, amiodarone, sotalol, flecainide, and propafenone, for the management of atrial fibrillation.

Effect of anti-arrhythmic drugs on all-cause mortality in studies involving >100 patients in either arm.

- **Dronedarone**: 0.85 (0.67, 1.09) \( P = 0.165 \)
- **Amiodarone**: 2.73 (1.00, 7.41) \( P = 0.049 \)
- **Sotalol**: 4.32 (1.59, 11.70) \( P = 0.013 \)

Incidence of proarrhythmics events:

- **Dronedarone**: 1.45 (1.02, 2.08) \( P = 0.043 \)
- **Propafenone**: 4.06 (1.13, 14.52) \( P = 0.035 \)
- **Amiodarone**: 5.45 (0.89, 42.93) \( P = 0.095 \)
- **Sotalol**: 6.44 (1.03, 40.24) \( P = 0.047 \)
- **Flecainide**: 6.77 (0.85, 54.02) \( P = 0.067 \)

*Figure 9* Mixed treatment comparison analysis: effect of anti-arrhythmic drugs on incidence of proarrhythmic events, odds ratio, and 95% confidence intervals.
Pulmonary veins can be triggers for atrial fibrillation initiation.
ΠΛΕΟΝΕΚΤΗΜΑΤΑ ΜΕΘΟΔΩΝ (RF vs Cryo)

❖ Εμπειρία
❖ Διαθεσιμότητα
❖ Ιδιαίτερη ανατομία πνευμονικών φλεβών
❖ Αντιμετώπιση επιπρόσθετων αρρυθμιών που απαντούν στη διάρκεια της επέμβασης (πχ. ισθμοεξαρτώμενος πτερυγισμός)
❖ Άμεσο κόστος

❖ Ταχεία αξιόπιστη απομόνωση ΠΦ
❖ Ταχύτερη εκμάθηση τεχνικής
❖ Υψηλή αποτελεσματικότητα επί «τυπικής» ανατομίας
❖ Έμμεσο κόστος
Atrial Fibrillation Ablation – Transeptal puncture
Catheter Ablation versus Standard conventional Treatment in patients with Left ventricular dysfunction and Atrial Fibrillation

The CASTLE-AF trial

Nassir F. Marrouche MD
on behalf the CASTLE AF Investigators
Catheter Ablation for Atrial Fibrillation with Heart Failure

Nassir F. Marrouche, M.D., Johannes Brachmann, M.D., Dietrich Andresen, M.D., Jürgen Siebels, M.D., Lucas Boersma, M.D., Luc Jordaens, M.D., Béla Merkely, M.D., Evgeny Pokushalov, M.D., Prashanthan Sanders, M.D., Jochen Proff, B.S., Heribert Schunkert, M.D., Hildegard Christ, M.D., Jürgen Vogt, M.D., and Dietmar Bänsch, M.D., for the CASTLE-AF Investigators*

ABSTRACT

BACKGROUND
Mortality and morbidity are higher among patients with atrial fibrillation and heart failure than among those with heart failure alone. Catheter ablation for atrial fibrillation has been proposed as a means of improving outcomes among patients with heart failure who are otherwise receiving appropriate treatment.
CASTLE-AF

Primary Endpoint

• All-cause mortality

• Worsening heart failure admissions

Secondary Endpoints

• All-cause mortality
• Worsening of heart failure admissions
• Cerebrovascular accidents
• Cardiovascular mortality
• Unplanned hospitalization due to cardiovascular reason
• All-cause hospitalization
• Quality of Life: Minnesota Living with Heart Failure and EuroQoL EQ-5D
• Exercise tolerance (6 minutes walk test)
• Number of delivered ICD shocks, and ATPs (appropriate/inappropriate)
• LVEF
• Time to first ICD shock, and time to first ATP
• Number of device detected VT/VF
• AF burden: cumulative duration of AF episodes
• AF free interval: time to first AF recurrence after 3 months blanking period post ablation
CASTLE-AF

Inclusion Criteria

- Symptomatic paroxysmal or persistent AF
- Failure or intolerance to ≥ 1 or unwillingness to take AAD
- LVEF ≤ 35%
- NYHA class ≥ II
- ICD/CRT-D with Home Monitoring capabilities already implanted due to primary or secondary prevention
Catheter Ablation for Atrial Fibrillation with Heart Failure

Table 2. Primary and Secondary Clinical End Points.*

<table>
<thead>
<tr>
<th>End Point</th>
<th>Ablation (N=179)</th>
<th>Medical Therapy (N=184)</th>
<th>Hazard Ratio (95% CI)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>number (percent)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primary†</td>
<td>51 (28.5)</td>
<td>82 (44.6)</td>
<td>0.62 (0.43–0.87)</td>
<td>0.007</td>
</tr>
<tr>
<td>Secondary</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Death from any cause</td>
<td>24 (13.4)</td>
<td>46 (25.0)</td>
<td>0.53 (0.32–0.86)</td>
<td>0.01</td>
</tr>
<tr>
<td>Heart-failure hospitalization</td>
<td>37 (20.7)</td>
<td>66 (35.9)</td>
<td>0.56 (0.37–0.83)</td>
<td>0.004</td>
</tr>
<tr>
<td>Cardiovascular death</td>
<td>20 (11.2)</td>
<td>41 (22.3)</td>
<td>0.49 (0.29–0.84)</td>
<td>0.009</td>
</tr>
<tr>
<td>Cardiovascular hospitalization</td>
<td>64 (35.8)</td>
<td>89 (48.4)</td>
<td>0.72 (0.52–0.99)</td>
<td>0.04</td>
</tr>
<tr>
<td>Hospitalization for any cause</td>
<td>114 (63.7)</td>
<td>122 (66.3)</td>
<td>0.99 (0.77–1.28)</td>
<td>0.96</td>
</tr>
<tr>
<td>Cerebrovascular accident</td>
<td>5 (2.8)</td>
<td>11 (6.0)</td>
<td>0.46 (0.16–1.33)</td>
<td>0.15</td>
</tr>
</tbody>
</table>

*Endpoints were assessed in a prespecified analysis plan.
†Atrial tachycardia episodes were not included as an endpoint as they were not specifically defined in the protocol.

Catheter Ablation for Atrial Fibrillation with Heart Failure

A Death or Hospitalization for Worsening Heart Failure

Hazard ratio, 0.62 (95% CI, 0.43–0.87)
P<0.007 by Cox regression
P=0.006 by log-rank test

No. at Risk
Ablation 179 141 114 76 58 22
Medical therapy 184 145 111 70 48 12

Marrouche H, et al. New Eng J Med 2018;378(5);417-427
Catheter Ablation for Atrial Fibrillation with Heart Failure

C Hospitalization for Worsening Heart Failure

- Probability of Hospital Admission
- Months of Follow-up
- No. at Risk
  - Ablation: 179, 141, 114, 76, 58, 22
  - Medical therapy: 184, 145, 111, 70, 48, 12

Hazard ratio, 0.56 (95% CI, 0.37–0.83)
P – 0.004 by Cox regression
P – 0.004 by log-rank test
Catheter Ablation for Atrial Fibrillation with Heart Failure

B  Death from Any Cause

Hazard ratio, 0.53 (95% CI, 0.32–0.86)
P = 0.01 by Cox regression
P = 0.009 by log-rank test

No. at Risk
Ablation 179 154 130 94 71 27
Medical therapy 184 168 138 97 63 19
Conclusions of CASTLE AF

➢ **Catheter ablation** of atrial fibrillation in patients with heart failure is associated with *improved all-cause mortality* and *fewer admissions for worsening heart failure* when compared to conventional standard of care treatment.

➢ **Catheter ablation** of atrial fibrillation in patients with heart failure is also associated with *improved cardiovascular mortality* and *hospitalization* when compared to conventional standard of care treatment.
Atrial fibrillation ablation patients have long-term stroke rates similar to patients without atrial fibrillation regardless of CHADS2 score

T. Jared Bunch, MD,* Heidi T. May, PhD, MSPH,* Tami L. Bair, BS,* J. Peter Weiss, MD,* Brian G. Crandall, MD,* Jeffrey S. Osborn, MD,* Charles Mallender, MD,* Jeffrey L. Anderson, MD,† Brent J. Muhlestein, MD,† Donald L. Lappe, MD,* John D. Day, MD, FHRS†

From the *Intermountain Heart Institute, Intermountain Medical Center, Murray, Utah, and †Department of Medicine, University of Utah, Salt Lake City, Utah.

Patients were enrolled from the large ongoing prospective Intermountain Atrial Fibrillation Study and were followed for at least 3 years.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Baseline demographics of the study population</th>
</tr>
</thead>
<tbody>
<tr>
<td>Characteristic</td>
<td>No AF (n = 16,848)</td>
</tr>
<tr>
<td>Age (y)</td>
<td>64.1 ± 13.0</td>
</tr>
<tr>
<td>Sex male</td>
<td>60.8%</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>58.4%</td>
</tr>
<tr>
<td>Hypertension</td>
<td>41.2%</td>
</tr>
<tr>
<td>Diabetes</td>
<td>19.0%</td>
</tr>
<tr>
<td>Heart failure</td>
<td>14.5%</td>
</tr>
<tr>
<td>Renal failure</td>
<td>5.6%</td>
</tr>
<tr>
<td>MI history</td>
<td>10.0%</td>
</tr>
<tr>
<td>TIA history</td>
<td>4.0%</td>
</tr>
<tr>
<td>CVA history</td>
<td>4.4%</td>
</tr>
<tr>
<td>Valve history</td>
<td>10.9%</td>
</tr>
<tr>
<td>CHADS2 score</td>
<td>0: 41.0%</td>
</tr>
<tr>
<td></td>
<td>1: 28.3%</td>
</tr>
<tr>
<td></td>
<td>2: 17.9%</td>
</tr>
<tr>
<td></td>
<td>3: 8.6%</td>
</tr>
<tr>
<td></td>
<td>4: 8.6%</td>
</tr>
<tr>
<td></td>
<td>5: 1.2%</td>
</tr>
<tr>
<td></td>
<td>6: 0.2%</td>
</tr>
<tr>
<td>EF (n = 10,004)</td>
<td>60.0 ± 16.0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Age-based long-term stroke rates among AF patients who underwent ablation compared to those AF patients who did not undergo ablation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>AF, no ablation</td>
</tr>
<tr>
<td>&lt; 60, n = 5638</td>
<td>3.6%</td>
</tr>
<tr>
<td>60–69, n = 5804</td>
<td>5.6%</td>
</tr>
<tr>
<td>70–79, n = 7082</td>
<td>8.7%</td>
</tr>
<tr>
<td>≥ 80, n = 2536</td>
<td>8.6%</td>
</tr>
</tbody>
</table>

Log rank p < .0001

Days To CVA
Radiofrequency catheter ablation has become an established treatment option for the management of patients with atrial fibrillation (AF). Although the concept of a rhythm control strategy devoid of the adverse events related to antiarrhythmic treatment seems highly attractive, further steps are needed in order to improve our understanding of our therapeutic efficacy. Furthermore, the increased cost of candidates also mandates the evaluation of this invasive treatment against the existing evidence pertaining to cost-effectiveness of AF catheter ablation (see Figure 1). The cost-effectiveness plane.

The value of innovation should not be underestimated.

Impact of Risk Factor and Weight Management on AF Ablation Outcomes

The schematic demonstrates the natural progression of the atrial fibrillation (AF) substrate and its impact on the maintenance of sinus rhythm (blue). Risk factor management has been demonstrated to reduce the burden of AF and also improve the outcomes of catheter ablation (salmon).

RESULTS: There were no differences in baseline characteristics, number of procedures, or follow-up duration between the groups (p = NS). RFM resulted in greater reductions in weight (p = 0.002) and blood pressure (p = 0.006), and better glycemic control (p = 0.001) and lipid profiles (p = 0.01). At follow-up, AF frequency, duration, symptoms, and symptom severity decreased more in the RFM group compared with the control group (all p < 0.001). Single-procedure drug-unassisted arrhythmia-free survival was greater in RFM patients compared with control subjects (p < 0.001). Multiple-procedure arrhythmia-free survival was markedly better in RFM patients compared with control subjects (p < 0.001), with 16% and 42.4%, respectively, using antiarrhythmic drugs (p = 0.004). On multivariate analysis, type of AF (p < 0.001) and RFM (hazard ratio 4.8 [95% confidence interval: 2.04 to 11.4]; p < 0.001) were independent predictors of arrhythmia-free survival.

CONCLUSIONS: Aggressive RFM improved the long-term success of AF ablation. This study underscores the importance of therapy directed at the primary promoters of the AF substrate to facilitate rhythm control strategies.
Ευχαριστώ την ομάδα ηλεκτροφυσιολογίας του Ερρίκος Ντυνάν Hospital Center

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- Παστρωμάς Σ.
- Συκιώτης Α.
- Κουρκούτη Π.
- Ταμπάκης Κ.

Νοσηλευτές
- Λιβιτσάνου Γ.
- Αλεξοπούλου Γ.
- Γουργιώτη Ζ.
- Καμμένος Σ.
- Κληματσούδας Β.
- Μαυροδήμου Ν.

Αναισθησιολόγοι
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- Ροζάκης Δ.

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