MANAGEMENT AND TREATMENT OF ATRIAL FIBRILLATION

FARHAT S. KHAIRALLAH, MD, FHRS, FACC
SCOPE OF ATRIAL FIBRILLATION (AF)

- 30-35 million people with AF worldwide
- Prevalence in adults ~ 3%
- Incidence is higher in developed countries
- One in 4 middle-aged adults in U.S. and Europe will develop AF
- Increasing prevalence
  - Better detection
  - Aging population and incidence of factors that contribute to AF
    - age, hypertension, obesity, atherosclerotic disease, renal insufficiency

(Circulation. 2014;130:2071-2104.)
COST

- U.S. (2008) $6-26 billion spent on care related to AF
  - Related to complication from stroke
  - Hospitalizations
  - Other

- 1% of healthcare spending in UK
**AF SYMPTOMS CLINICAL PRESENTATION**

**Symptomatic patients**
- Fast and irregular beats / palpitations
  - May be associated with slow heart rates in setting of conduction abnormalities due to disease or acquired condition such as use of medication
- Fatigue
- Shortness of breath
- Chest discomfort
- Complication of Afib
  - Heart failure
  - Stroke
  - Angina in patients with coronary disease

**Asymptomatic patients**
- Noted on physical exam
- Routine screening ECG or during diagnostic study performed for other reason
- Peri or intraoperative states
# Symptom Assessment

<table>
<thead>
<tr>
<th>Modified EHRA score</th>
<th>Symptoms</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>None</td>
<td>AF does not cause any symptoms.</td>
</tr>
<tr>
<td>2a</td>
<td>Mild</td>
<td>Normal daily activity not affected by symptoms related to AF.</td>
</tr>
<tr>
<td>2b</td>
<td>Moderate</td>
<td>Normal daily activity not affected by symptoms related to AF, but patient troubled by symptoms.</td>
</tr>
<tr>
<td>3</td>
<td>Severe</td>
<td>Normal daily activity affected by symptoms related to AF.</td>
</tr>
<tr>
<td>4</td>
<td>Disabling</td>
<td>Normal daily activity discontinued.</td>
</tr>
</tbody>
</table>
## CARDIOVASCULAR MORBIDITY AND MORTALITY ASSOCIATED WITH AF

<table>
<thead>
<tr>
<th>Event</th>
<th>Association with AF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>Increased mortality, especially cardiovascular mortality due to sudden death, heart failure or stroke.</td>
</tr>
<tr>
<td>Stroke</td>
<td>20–30% of all strokes are due to AF. A growing number of patients with stroke are diagnosed with ‘silent’, paroxysmal AF.</td>
</tr>
<tr>
<td>Hospitalizations</td>
<td>10–40% of AF patients are hospitalized every year.</td>
</tr>
<tr>
<td>Quality of life</td>
<td>Quality of life is impaired in AF patients independent of other cardiovascular conditions.</td>
</tr>
<tr>
<td>Left ventricular dysfunction and heart failure</td>
<td>Left ventricular dysfunction is found in 20–30% of all AF patients. AF causes or aggravates LV dysfunction in many AF patients, while others have completely preserved LV function despite long-standing AF.</td>
</tr>
<tr>
<td>Cognitive decline and vascular dementia</td>
<td>Cognitive decline and vascular dementia can develop even in anticoagulated AF patients. Brain white matter lesions are more common in AF patients than in patients without AF.</td>
</tr>
</tbody>
</table>
- Early onset AF has strong heritable component
- Few patients have rare monogenetic channopathies or inherited cardiomyopathies that are associated with increase incidence of AF
- 1/3 of AF patients carry common genetic variants that predispose to AF e.g. SNP
  - Some of these genetic variants may modify the risk of AF up to 7 fold
  - Some genetic variants are associated with increase risk of stroke
- Genetic markers in the future may help in selection of diagnostic tests and treatment recommendations
Incidence and prevalence is lower in women
Risk of death associated with AF in women is equal or higher
Risk of stroke in women is higher, especially at older age
Symptoms can be more intense for women
“effective diagnostic and treatment tools should be offered equally to women and men”
MAJOR MECHANISMS CAUSING AF
> 90% success rates for many types of SVT following single ablation procedure
LEFT ATRIAL REMODELING

- Morphologic changes at cellular, tissue, chamber levels
- Functional changes
  - Strain, LA ejection fraction, pressure/volume relationships
- Electrical remodeling
  - Cellular ion currents, ectopy, conduction abnormalities
LA REMODELING: DELAYED ENHANCEMENT CMR CORRELATION WITH ELECTROANATOMIC MAPPING

**FIGURE 1** Light Microscopy of Crista Terminalis Specimen

Light microscopy of a cristal terminalis specimen in a patient in (A) sinus rhythm and without a history of atrial fibrillation (AF) showing no significant fibrosis, and (B) persistent AF showing a high level of fibrosis. Courtesy of Pyotr Platonov, MD, and Lubov Mitrofanova, MD.

DE-CMR correlates with electroanatomic mapping acquired by invasive electrophysiological study to identify patterns of low voltage (defined by the boundary of white lines) in posterior (PA) and right anterior oblique (RAO) views. (A) DE-CMR maximum intensity projection. (B) Color 3D models improve dynamic range and illuminate enhancement patterns. (C) Electroanatomic map. Reprinted with permission from Oakes et al. (130).
ELECTROPHYSIOLOGICAL MECHANISMS

**Electrophysiological Mechanisms**

- **Microreentrant circuits**
  - Sueda

- **PV foci**
  - Haisaguerre
  - NEJM 1998

- **LOM**
  - Hwang
  - Circulation 2000

- **Vagal Ganglia**
  - Pappone
  - Circulation 2004

- **Dominant Spiral Wave**
  - Mandapati
  - Circulation 2000

**A** Focal Activation

**B** Multiple Wavelets
AF IS DIFFERENT IN DIFFERENT PATIENTS
AF PATTERNS

- **Paroxysmal**
- **Persistent**
- **Long-standing persistent**
- **Permanent**

<table>
<thead>
<tr>
<th>AF pattern</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>First diagnosed AF</strong></td>
<td>AF that has not been diagnosed before, irrespective of the duration of the arrhythmia or the presence and severity of AF-related symptoms.</td>
</tr>
<tr>
<td><strong>Paroxysmal AF</strong></td>
<td>Self-terminating, in most cases within 48 hours. Some AF paroxysms may continue for up to 7 days. AF episodes that are cardioverted within 7 days should be considered paroxysmal.</td>
</tr>
<tr>
<td><strong>Persistent AF</strong></td>
<td>AF that lasts longer than 7 days, including episodes that are terminated by cardioversion, either with drugs or by direct current cardioversion, after 7 days or more.</td>
</tr>
<tr>
<td><strong>Long-standing persistent AF</strong></td>
<td>Continuous AF lasting for ≥1 year when it is decided to adopt a rhythm control strategy.</td>
</tr>
<tr>
<td><strong>Permanent AF</strong></td>
<td>AF that is accepted by the patient (and physician). Hence, rhythm control interventions are, by definition, not pursued in patients with permanent AF. Should a rhythm control strategy be adopted, the arrhythmia would be re-classified as 'long-standing persistent AF'.</td>
</tr>
</tbody>
</table>

*(Circulation. 2014;130:2071-2104.)*
## CLINICAL SUBTYPES OF AF

<table>
<thead>
<tr>
<th>AF type</th>
<th>Clinical presentation</th>
<th>Possible pathophysiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>AF secondary to structural heart disease</td>
<td>AF in patients with systolic/diastolic dysfunction, or structural disease.</td>
<td>Structural remodelling; activation of autonomic/renin-angiotensin system.</td>
</tr>
<tr>
<td>Focal AF</td>
<td>Patients with coarse paroxysmal AF; often highly symptomatic and younger.</td>
<td>Localized triggers, in most cases originating from pulmonary veins.</td>
</tr>
<tr>
<td>Polygenic AF</td>
<td>Common gene variants associated with early onset AF.</td>
<td>Currently under study.</td>
</tr>
<tr>
<td>Postoperative AF</td>
<td>New onset after major (typically cardiac) surgery.</td>
<td>Acute perioperative factors and pre-existing substrate for AF.</td>
</tr>
<tr>
<td>AF with mitral stenosis or prosthetic valves</td>
<td>AF in patients with mitral stenosis, mitral valve surgery and other valvular disease.</td>
<td>Left atrial pressure (stenosis) and volume (regurgitation) load.</td>
</tr>
<tr>
<td>AF in athletes</td>
<td>Usually paroxysmal, related to duration/intensity of training.</td>
<td>Increased vagal tone and atrial volume.</td>
</tr>
<tr>
<td>Monogenic AF</td>
<td>AF in inherited cardiomyopathies and channelopathies.</td>
<td>Arrhythmogenic mechanisms responsible for sudden death.</td>
</tr>
</tbody>
</table>
GENERAL APPROACH TO TREATMENT

**Treatment**
- Acute rate and rhythm control (e.g., β-blockers, cardioversion)
- Manage precipitating factors
- Assess stroke risk
- Assess heart rate
- Assess symptoms
- Lifestyle changes, treatment of underlying cardiovascular conditions
- Oral anticoagulation in patients at risk for stroke
- Rate control therapy
- Antiarrhythmic drugs, cardioversion, catheter ablation, AF surgery

**Desired outcome**
- Haemodynamic stability
- Cardiovascular risk reduction
- Stroke prevention
- Symptom improvement, preservation of LV function
- Symptom improvement

**Patient benefit**
- Improved life expectancy
- Improved quality of life, autonomy, social functioning
**Clinical conditions**

- Haemodynamic instability.
- Uncontrollable heart rate.
- Symptomatic bradycardia not amenable to reduction of rate control agents.
- Severe angina or worsening left ventricular function.
- Transient ischaemic attack or stroke.
ACHIEVING OPTIMAL MANAGEMENT IN AF PATIENTS

- Provision of all therapy options
- Measurable high service quality
- Multidisciplinary service, shared decision making
- Service accessible for all patients

Optimal AF management
## INTEGRATED AF MANAGEMENT

<table>
<thead>
<tr>
<th>Patient Involvement</th>
<th>Multidisciplinary teams</th>
<th>Technology tools</th>
<th>Access to all treatment options for AF</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Central role in care process.</td>
<td>• Physicians (general physicians, cardiology and stroke AF specialists, surgeons) and</td>
<td>• Information on AF.</td>
<td>• Structured support for lifestyle changes.</td>
</tr>
<tr>
<td>• Patient education.</td>
<td>allied health professionals work in a collaborative practice model.</td>
<td>• Clinical decision support.</td>
<td>• Anticoagulation.</td>
</tr>
<tr>
<td>• Encouragement and empowerment for self-management.</td>
<td>• Efficient mix of communication skills, education, and experience.</td>
<td>• Checklist and communication tools</td>
<td>• Rate control.</td>
</tr>
<tr>
<td>• Advice and education on lifestyle and risk factor management.</td>
<td>• Working together in a multi-disciplinary chronic AF care team.</td>
<td>• Used by healthcare professionals and patients.</td>
<td>• Antiarrhythmic drugs.</td>
</tr>
<tr>
<td>• Shared decision making.</td>
<td></td>
<td>• Monitoring of therapy adherence and effectiveness.</td>
<td>• Catheter and surgical interventions (ablation, LAA occluder, AF surgery, etc.).</td>
</tr>
<tr>
<td>• Informed, involved, empowered patient.</td>
<td></td>
<td>• Navigation system to support decision making in treatment team.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Complex management decisions underpinned by an AF Heart Team.</td>
</tr>
</tbody>
</table>
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OBESITY

Ablation Free Drug Free AF Freedom

Follow-up (Days)

Total AF Freedom

Follow-up (Days)

Group
- WL $\leq 10\%$
- WL 3 - 9\%
- WL $< 3\%$ or Gain
- Reverse remodeling of atria is seen with use of CPAP
- Decrease recurrences of AF after cardioversion and/or ablation
- Decrease risk of AF
- Screening for sleep apnea should be considered in AF patients with risk factors
- Sleep apnea is common in heart failure reduced ejection fraction
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AF IS ASSOCIATED WITH INCREASE RISK OF STROKE AND MORTALITY

Stroke rate classified by age group.
THROMBUS HAPPENS
Other risk factors to consider

- Left atrial enlargement
- Left ventricular hypertrophy
- Hypertrophic cardiomyopathy
- Prothrombotic states
- Hormone replacement
- Low velocities on TEE
INCREASED ADVERSE OUTCOMES IN MORE PERSISTENT FORMS OF AF

ENGAGE AF TIMI 48 trial
Pre-specified analysis of the Edoxaban v. warfarin no inferiority trial

(Circ Arrhythm Electrophysiol. 2017;10:e004267.)
**CHA₂DS₂-VASC**
- Hypertension
- Age 75

**HAS-BLED**
- Alcohol
- Age
- Hypertension

(Circulation. 2014;130:2071-2104.)

**HAS-BLED**

- Alcohol
- Age
- Hypertension

**Modifiable bleeding risk factors**
- Hypertension (especially when systolic blood pressure is >160 mmHg)
- Labile INR or time in therapeutic range <40% in patients on vitamin K antagonists
- Medication predisposing to bleeding such as antithrombotic drugs and non-steroidal anti-inflammatory drugs
- Excessive alcohol (>8 drinks/week)
- Potentially modifiable bleeding risk factors
  - Atrial fibrillation
  - Impaired renal function
  - Impaired liver function
  - Reduced platelet count or function

**Non-modifiable bleeding risk factors**
- Age (>65 years) (≥75 years)
- History of major bleeding
- Previous stroke
- Dialysis-dependent kidney disease or renal transplant
- Cirrhotic liver disease
- Malignancy
- Genetic factors
- Biomarker-based bleeding risk factors
  - High-sensitivity troponin
  - Growth differentiation factor-15
  - Serum creatinine/estimated GFR
HOW MUCH AF IS ENOUGH TO BE DANGEROUS?

- Unknown at this time
- Atrial high rate episodes (AHRE) occur in 10-15% of patients with pacer or implantable ICDs
- AHRE episodes are associated with higher risk of stroke although many strokes are not temporally related to the actual AHRE
- At this time unclear how to treat these patients
  - ARTESia, NOAH-AFNET 6
APPROACHES TO STROKE PREVENTION IN AF

- Coumadin/warfarin
- New generation anticoagulants, novel anticoagulants, direct oral antithrombotic agents
- (Antiplatelet agents)
- Device therapy
  - Endovascular
  - Epicardial approaches
  - Surgical
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- Improved quality of life, autonomy, social functioning

Tallahassee Memorial Healthcare
Southern Medical Group, P.A.
- 60-100 BPM at rest is reasonable as long as no other significant issues or symptoms

- RACE II no major difference between <80 BPM at rest and <110 BPM with moderate exercise v. <110 BPM at rest
APPROACH TO SELECTING DRUG THERAPY FOR VENTRICULAR RATE CONTROL*

Atrial Fibrillation

- No Other CV Disease
  - Beta blocker
  - Diltiazem
  - Verapamil

- Hypertension or HFpEF
  - Beta blocker
  - Diltiazem
  - Verapamil

- LV Dysfunction or HF
  - Beta blocker†
  - Digoxin‡

- COPD
  - Beta blocker
  - Diltiazem
  - Verapamil

Amiodarone§
RATE CONTROL WITH PACER AND AV JUNCTION ABLATION
GENERAL APPROACH TO TREATMENT

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RHYTHM CONTROL

- Medications/antiarrhythmic medications

- Invasive
  - Catheter based techniques
  - Surgical
  - Hybrid
# ANTI-ARRHYTHMIC THERAPY (LONG TERM)

<table>
<thead>
<tr>
<th>Antiarrhythmic Drug Name</th>
<th>Typical Dose</th>
<th>Timing</th>
<th>Potential Significant Adverse Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flecainide</td>
<td>50-200 mg</td>
<td>Every 12 h</td>
<td>Sinus node or atrioventricular node dysfunction, Atrial flutter with 1:1 ventricular response</td>
</tr>
<tr>
<td>Propafenone</td>
<td>150-300 mg (Immediate release)</td>
<td>Every 8 h</td>
<td>Sinus node or atrioventricular node dysfunction, Atrial flutter with 1:1 ventricular response</td>
</tr>
<tr>
<td></td>
<td>225-425 mg (extended release)</td>
<td>Every 12 h</td>
<td>Sinus node or atrioventricular node dysfunction, Atrial flutter with 1:1 ventricular response</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>Loading dose: 400-600 mg/d</td>
<td>For 4 wk</td>
<td>Sinus node or atrioventricular node dysfunction, Lung, liver, or thyroid toxicity, Prolonged QT interval (torsade de pointes ventricular tachycardia rare)</td>
</tr>
<tr>
<td></td>
<td>Maintenance dose: 200-300 mg</td>
<td>Daily</td>
<td>Sinus node or atrioventricular node dysfunction, Lung, liver, or thyroid toxicity, Prolonged QT interval (torsade de pointes ventricular tachycardia rare)</td>
</tr>
<tr>
<td>Dofetilide</td>
<td>125-500 µg</td>
<td>Every 12 h</td>
<td>Prolonged QT interval with torsade de pointes ventricular tachycardia</td>
</tr>
<tr>
<td>Dronedarone</td>
<td>400 mg</td>
<td>Every 12 h</td>
<td>Sinus node or atrioventricular node dysfunction, Prolonged QT interval (torsade de pointes ventricular tachycardia rare)</td>
</tr>
<tr>
<td>Sotalol</td>
<td>40-160 mg</td>
<td>Every 12 h</td>
<td>Sinus node or atrioventricular node dysfunction, Prolonged QT interval (torsade de pointes ventricular tachycardia rare)</td>
</tr>
</tbody>
</table>

*a* Indicates renal-based dosing.

*b* Renal dosing may require dosing every 24 hours instead.

About 50% efficacy at maintaining sinus rhythm.
INVASIVE RHYTHM CONTROL

Surgical Cox MAZE procedure developed in late 1980's

Catheter ablation of AF triggers mid to late

Catheter based pulmonary vein isolation cryoablation is developed and utilized over past 10
Cox MAZE procedure has evolved to utilize RF ablation and cryotherapy instead of cut and suture technique.

In most instances a MAZE is added to other cardiac surgery procedure.

Less invasive surgical options have been developed e.g. thoracoscopic ablation, stand alone or as hybrid approach.

Some procedures appear more effective than stand alone endovascular ablation.

More invasive, longer hospital stays, higher complication rates.

### Complication Rates

<table>
<thead>
<tr>
<th>Complication</th>
<th>Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conversion to sternotomy</td>
<td>0–1.6%</td>
</tr>
<tr>
<td>Pacemaker implantation</td>
<td>0–3.3%</td>
</tr>
<tr>
<td>Drainage for pneumothorax</td>
<td>0–3.3%</td>
</tr>
<tr>
<td>Pericardial tamponade</td>
<td>0–6.0%</td>
</tr>
<tr>
<td>Transient ischaemic attack</td>
<td>0–3.0%</td>
</tr>
</tbody>
</table>
Paroxysmal AF: ~ 70% success of freedom from significant symptomatic AF

Persistent AF: ~ 50% success

Late recurrences are possible and may represent progression of disease state → implications regarding anticoagulation
ACHILLES HEEL IN AF MANAGEMENT
ADDITIONAL TARGETS AND STRATEGIES USED IN ABLATION

- Pre-procedural risk stratification for recurrences
  - Utah score
  - FACM score
- Complex fractionated electrograms
- Ganglionic plexi
- Box isolation of fibrotic areas
- Spatiotemporal mapping
- Rotor mapping
  - Pre-procedure
  - Intra-procedural
Reconnection to an area of triggers
  - Pulmonary vein

Progression of abnormal substrate and disease state

Formation of abnormal conduction areas leading to re-entry tachycardias/atrial flutters
CHOICE OF FURTHER RHYTHM CONTROL STRATEGY FOLLOWING TREATMENT FAILURE

Selection of further rhythm control therapy after therapy failure to improve symptoms of AF

- Failure of dronedarone, flecainide, propafenone, or sotalol
  - Patient choice
    - Amiodarone (Ia)
    - another AAD (Ila)

- Failure of amiodarone
  - Patient choice
    - Catheter Ablation (Ia/IIaB)

- Failure of catheter ablation
  - Patient choice
    - Hybrid therapy (IIaC)
    - Repeat ablation (Ia/IIaB)
    - another AAD (Ila)

Patient choice informed by AF Heart Team

- AF surgery (IIaC)
- Rate control (IB)
- Hybrid therapy (IIaC)
## ABLATION COMPLICATIONS

<table>
<thead>
<tr>
<th>Complication severity</th>
<th>Complication type</th>
<th>Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Life-threatening complications</td>
<td>Periprocedural death</td>
<td>&lt;0.2%</td>
</tr>
<tr>
<td></td>
<td>Oesophageal injury (perforation/fistula)</td>
<td>&lt;0.5%</td>
</tr>
<tr>
<td></td>
<td>Periprocedural stroke (including TIA/air embolism)</td>
<td>&lt;1%</td>
</tr>
<tr>
<td></td>
<td>Cardiac tamponade</td>
<td>1–2%</td>
</tr>
<tr>
<td>Severe complications</td>
<td>Pulmonary vein stenosis</td>
<td>&lt;1%</td>
</tr>
<tr>
<td></td>
<td>Persistent phrenic nerve palsy</td>
<td>1–2%</td>
</tr>
<tr>
<td></td>
<td>Vascular complications</td>
<td>2–4%</td>
</tr>
<tr>
<td></td>
<td>Other severe complications</td>
<td>≈1%</td>
</tr>
<tr>
<td>Other moderate or minor complications</td>
<td></td>
<td>1–2%</td>
</tr>
<tr>
<td>Unknown significance</td>
<td>Asymptomatic cerebral embolism (silent stroke)</td>
<td>5–20%</td>
</tr>
<tr>
<td></td>
<td>Radiation exposure</td>
<td></td>
</tr>
</tbody>
</table>
# Antiarrhythmic Therapy vs. Ablation

## Catheter Ablation vs Antiarrhythmic Drugs in First-Line Therapy

<table>
<thead>
<tr>
<th>Source</th>
<th>No. of Patients</th>
<th>End Point Description</th>
<th>AF Recurrence, %&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Catheter Ablation</th>
<th>Antiarrhythmic Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wazni et al.,&lt;sup&gt;19&lt;/sup&gt; 2005</td>
<td>70</td>
<td>1-y AF recurrence</td>
<td>13</td>
<td>63</td>
<td></td>
</tr>
<tr>
<td>Cosseds Nielsen et al,&lt;sup&gt;20&lt;/sup&gt; 2012</td>
<td>254</td>
<td>Cumulative AF burden&lt;sup&gt;b&lt;/sup&gt;</td>
<td>13</td>
<td>19</td>
<td></td>
</tr>
<tr>
<td>Mortillo et al,&lt;sup&gt;21&lt;/sup&gt; 2014</td>
<td>127</td>
<td>2-y AF recurrence</td>
<td>54.5</td>
<td>72.1</td>
<td></td>
</tr>
</tbody>
</table>

## Catheter Ablation vs Antiarrhythmic Drugs in Second-Line Therapy

<table>
<thead>
<tr>
<th>Source</th>
<th>No. of Patients</th>
<th>End Point Description</th>
<th>AF Recurrence, %&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Catheter Ablation</th>
<th>Antiarrhythmic Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jais et al.,&lt;sup&gt;22&lt;/sup&gt; 2008</td>
<td>112</td>
<td>1-y AF recurrence</td>
<td>11</td>
<td>77</td>
<td></td>
</tr>
<tr>
<td>Wilber et al,&lt;sup&gt;23&lt;/sup&gt; 2010</td>
<td>157</td>
<td>9-mo AF recurrence</td>
<td>34</td>
<td>84</td>
<td></td>
</tr>
<tr>
<td>Fieker et al,&lt;sup&gt;24&lt;/sup&gt; 2013</td>
<td>245</td>
<td>1-y AF recurrence</td>
<td>30.1</td>
<td>92.7</td>
<td></td>
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<tr>
<td>Mont et al,&lt;sup&gt;25&lt;/sup&gt; 2014</td>
<td>146</td>
<td>1-y AF recurrence</td>
<td>29.6</td>
<td>56.3</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviation: AF, atrial fibrillation.

<sup>a</sup>AF burden was expressed as % of time in AF, 90th percentile.

<sup>b</sup>The difference in outcome was statistically significant for all comparisons of catheter ablation vs antiarrhythmic drugs except Cosseds Nielsen.<sup>20</sup>

<sup>c</sup>Patients in this study were diagnosed with persistent AF.
Indicated for maintenance of sinus rhythm for improvement in quality of life

Performed by expert operators

Complications associated with ablations have been termed similar to adverse outcomes associated with AADs

No data available to date on hard cardiovascular outcomes: death, cardiovascular death, discontinuation of anticoagulation

Most of the available data is in paroxysmal AF patients

Available data also favors ablation to antiarrhythmic in maintaining sinus rhythm in persistent forms of AF
STRATEGIES FOR RHYTHM CONTROL IN PATIENTS WITH PAROXYSMAL AND PERSISTENT AF

No Structural Heart Disease

- Dofetilide
- Dronedarone
- Flecaïnide
- Propafenone
- Sotalol

→ Catheter ablation

→ Amiodarone

Structural Heart Disease

- CAD
- HF

→ Catheter ablation

→ Amiodarone

→ Dofetilide
→ Dronedarone
→ Sotalol
Traditional trails of rhythm control have had neutral outcome

Modern rhythm control
- Early intervention in afib
- Catheter ablation
- Combination therapy antiarrhythmics and catheter ablation
  - EAST –AFNET 4, CABANA

Rhythm control is indicated for improvement in QOL/symptoms
In general exercise reduces risk of AF by promoting cardiovascular health

Intense endurance sports increase risk of AF later in life

Volume overload on atrium

Vagal tone
CONCLUSIONS

- Common problem, with significant associated morbidity, mortality and healthcare costs
- Complex rhythm with multiple biologic inputs that contribute to its pathogenesis
- For many patients AF is a chronic condition
- Well informed and empowered patients who take active role in their care tend to do better
- AF heart teams which include cardiologists, invasive electrophysiologist, surgeons, and other educators can play an important role in helping patients understand their options and achieve realistic treatment goals
General cardiologists with experience treating AF
Dedicated cardiologists with expertise in advanced imaging modalities
4 invasive electrophysiologists with expertise in complex ablation, device therapy
Availability of left atrial exclusion procedures (transcatheter and surgical)
Surgical and hybrid ablation
AF coordinator nurse
Involvement in clinical studies