Risk stratification and perioperative management of atrial fibrillation

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Left Atrial Appendage
Atrial Fibrillation and Thrombus Formation

Stroke in AF patients ≈ Appendage-related stroke

Prognostic Implications

Stroke, Mortality

Risk Ratio

<table>
<thead>
<tr>
<th>Stroke</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Framingham</td>
<td>Whitehall (no Heart Disease)</td>
</tr>
<tr>
<td>Whitehall Regional Heart Study</td>
<td>Manitoba</td>
</tr>
</tbody>
</table>
CHADS\textsubscript{2}–CHA\textsubscript{2}DS\textsubscript{2}–VASc Scores
Risk of Stroke in Atrial Fibrillation

What is the role of the left atrial appendage in determining stroke risk?

**Adjusted Stroke Rate (\% per y)**

CHADS\textsubscript{2}
- Congestive HF
- Hypertension
- Age ≥75 y
- Diabetes mellitus
- Stroke/TIA/TE
- Maximum score

CHA\textsubscript{2}DS\textsubscript{2}–VASc
- Congestive HF
- Hypertension
- Age ≥75 y
- Diabetes mellitus
- Stroke/TIA/TE
- Vascular disease (prior MI, PAD, or aortic plaque)
- Age 65–74 y
- Sex category (i.e., female sex)
- Maximum score

J Am Coll Cardiol. 2014;64(21):2246-2280. doi:10.1016/j.jacc.2014.03.021
Risk of LAA-related stroke

CHA$_2$DS$_2$-VASc Scores: Not specific

- CHA$_2$DS$_2$-VASc predicts risk of ischemic stroke in the ABSENCE of AF. *(Atherosclerosis. 2014 Dec;237(2):504-13.)*
Risk of LAA-related stroke

**CHA\textsubscript{2}DS\textsubscript{2}-VASc Scores: Not specific**

<table>
<thead>
<tr>
<th>CHA\textsubscript{2}DS\textsubscript{2}-VASc score 5</th>
<th>CHA\textsubscript{2}DS\textsubscript{2}-VASc score 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Sixty-six year-old (1)</td>
<td>• Sixty-six year-old (1)</td>
</tr>
<tr>
<td>• Female (1)</td>
<td>• Prior strokes (2)</td>
</tr>
<tr>
<td>• Diabetic (1)</td>
<td>• Ischemic cardiomyopathy with CHF (1)</td>
</tr>
<tr>
<td>• Hypertensive (1)</td>
<td>• Extensive, mobile atheromatous plaque in the aortic arch (1)</td>
</tr>
<tr>
<td>• Ca score of 450 (1)</td>
<td>• Persistent AF post CABG, cardioverted without recurrence</td>
</tr>
<tr>
<td>• Persistent AF for 2 years</td>
<td></td>
</tr>
<tr>
<td>• TEE prior to cardioversion showing LAA thrombus, resolved 1 month later</td>
<td></td>
</tr>
</tbody>
</table>
TEE specific findings in Nonvalvular atrial fibrillation associated with increase risk of stroke

**SPAF III**

- LAA Antegrade peak flow less than 20 cm/s
- Spontaneous echo contrast
- LAA thrombus

Electrical isolation of LAA needs OAC and consider LAA closure
LAA-related stroke risk?


[Diagram showing stroke rate comparison between Chicken Wing and Non-Chicken Wing groups.]
Extreme LAA Features

Are there benefits of anticoagulation beyond the LAA?

• SPAF study (*Neurology*. 1993; 43: 32–6):
  – 65% of strokes in atrial fibrillation classified as cardioembolic.
  – Up to 25% of strokes can be related to intrinsic cerebrovascular disease

• AF associations “procoagulant systemic state”:

• 4. Are there other diagnoses: DVT, PE
Risks of Stroke Prevention
Warfarin vs Watchman

### Risk for Bleeding

**Table 3**

<table>
<thead>
<tr>
<th>Score</th>
<th>Variable</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>H</td>
<td>Hypertension</td>
<td>1 point</td>
</tr>
<tr>
<td>A</td>
<td>Abnormal renal or hepatic function</td>
<td>1–2 points</td>
</tr>
<tr>
<td>S</td>
<td>Prior stroke</td>
<td>1 point</td>
</tr>
<tr>
<td>B</td>
<td>Bleeding</td>
<td>1 point</td>
</tr>
<tr>
<td>L</td>
<td>Labile INR values</td>
<td>1 point</td>
</tr>
<tr>
<td>E</td>
<td>Elderly, i.e., over age 65</td>
<td>1 point</td>
</tr>
<tr>
<td>D</td>
<td>Concomitant use of other drugs or alcohol</td>
<td>1–2 points</td>
</tr>
</tbody>
</table>

**Definition of the HAS-BLED score, with point distribution**

- **HAS-BLED Score**
  - 0: 0.9%
  - 1: 3.4%
  - 2: 4.1%
  - 3: 5.8%
  - 4: 8.9%
  - 5: 9.1%
  - 6–9: Insufficient data

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*INR, International Normalized Ratio.*  
*1 Modified from the guidelines of the European Society of Cardiology (3)*
Risks of Stroke Prevention
Warfarin vs NOACs

%/year

- RE-LY (Dabigatran 150 mg)
- ARISTOTLE (Apixaban)
- ROCKET-AF (Rivaroxaban)
- ENGAGE-AF (Edoxaban)

<table>
<thead>
<tr>
<th></th>
<th>Warfarin</th>
<th>NOAC</th>
</tr>
</thead>
<tbody>
<tr>
<td>ISCHEMIC STROKE</td>
<td>1.2</td>
<td>1.42</td>
</tr>
<tr>
<td>HEMORRHAGIC STROKE</td>
<td>0.38</td>
<td>0.47</td>
</tr>
<tr>
<td>BLEEDING</td>
<td>0.1</td>
<td>0.26</td>
</tr>
<tr>
<td>MORTALITY</td>
<td>4.35</td>
<td>3.52</td>
</tr>
</tbody>
</table>

Price, Valderrábano. *Circulation* 2014;130:202-12
Stroke prevention strategies

• Systemic anticoagulation
  – Warfarin
  – NOACs

• LAA closure
  – Watchman and other devices
  – Lariat
  – Atri-clip

• Selecting the right strategy requires individualization of risks/benefits!
Preventing Strokes in AF patients

Individualizing Risk: 4 questions

• 1. What are the causes of stroke risk in this patient?
  • AF-related vs AF unrelated stroke (LAA-related vs LAA unrelated)

• 2. What are the risks of stroke prevention strategies?
  – Bleeding risk Vs CHA\textsubscript{2}DS\textsubscript{2}-VASc
  – Hemorrhagic stroke risk
  – Procedural risk

• 3. Are there benefits of anticoagulation besides preventing LAA thrombus in AF?

• 4. What is the prior patient’s experience on anticoagulation?
Thanks!!
Perioperative management of atrial fibrillation
Perioperative risk stroke management in patients with atrial fibrillation

Systematic review

The BRIDGE Study

<table>
<thead>
<tr>
<th>Study or Subgroup</th>
<th>Bridging Events Total</th>
<th>No Bridging Events Total</th>
<th>Weight</th>
<th>Odds Ratio M-H, Random, 95% CI</th>
<th>Odds Ratio M-H, Random, 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daniels et al., 2009</td>
<td>36 342 18 213</td>
<td>9.8%</td>
<td>1.27 [0.70, 2.31]</td>
<td>1.27 [0.70, 2.31]</td>
<td></td>
</tr>
<tr>
<td>Dotan et al., 2002</td>
<td>2 20 1 20</td>
<td>3.7%</td>
<td>2.11 [0.16, 25.35]</td>
<td>2.11 [0.16, 25.35]</td>
<td></td>
</tr>
<tr>
<td>Erkan et al., 2010</td>
<td>11 44 21 1421</td>
<td>9.0%</td>
<td>22.22 [0.92, 49.81]</td>
<td>22.22 [0.92, 49.81]</td>
<td></td>
</tr>
<tr>
<td>Garcia et al., 2008</td>
<td>14 108 9 1185</td>
<td>8.8%</td>
<td>19.48 [0.21, 46.44]</td>
<td>19.48 [0.21, 46.44]</td>
<td></td>
</tr>
<tr>
<td>Ghanbari et al., 2010</td>
<td>6 29 3 74</td>
<td>6.5%</td>
<td>6.17 [1.43, 26.88]</td>
<td>6.17 [1.43, 26.88]</td>
<td></td>
</tr>
<tr>
<td>Jaffer et al., 2010</td>
<td>24 229 7 263</td>
<td>8.8%</td>
<td>4.28 [0.81, 10.14]</td>
<td>4.28 [0.81, 10.14]</td>
<td></td>
</tr>
<tr>
<td>Marquie et al., 2006</td>
<td>21 114 2 114</td>
<td>6.4%</td>
<td>12.65 [2.89, 55.34]</td>
<td>12.65 [2.89, 55.34]</td>
<td></td>
</tr>
<tr>
<td>McBane et al., 2010</td>
<td>34 514 5 261</td>
<td>1.4%</td>
<td>3.63 [1.40, 3.90]</td>
<td>3.63 [1.40, 3.90]</td>
<td></td>
</tr>
<tr>
<td>Robinson et al., 2009</td>
<td>20 113 3 35</td>
<td>7.2%</td>
<td>2.29 [0.64, 8.24]</td>
<td>2.29 [0.64, 8.24]</td>
<td></td>
</tr>
<tr>
<td>Tschenco et al., 2009</td>
<td>9 38 5 117</td>
<td>7.6%</td>
<td>6.95 [1.26, 22.33]</td>
<td>6.95 [1.26, 22.33]</td>
<td></td>
</tr>
<tr>
<td>Tompkins et al., 2010</td>
<td>23 165 15 513</td>
<td>9.5%</td>
<td>5.73 [1.29, 9.41]</td>
<td>5.73 [1.29, 9.41]</td>
<td></td>
</tr>
<tr>
<td>Varakakis et al., 2005</td>
<td>2 25 7 762</td>
<td>5.9%</td>
<td>9.38 [1.75, 47.64]</td>
<td>9.38 [1.75, 47.64]</td>
<td></td>
</tr>
<tr>
<td>Wysokinski et al., 2008</td>
<td>15 204 6 182</td>
<td>8.4%</td>
<td>2.33 [0.68, 8.13]</td>
<td>2.33 [0.68, 8.13]</td>
<td></td>
</tr>
<tr>
<td>Total (95% CI)</td>
<td>1935</td>
<td>5160 100.0%</td>
<td>5.40 [3.00, 9.74]</td>
<td>5.40 [3.00, 9.74]</td>
<td></td>
</tr>
<tr>
<td>Total events</td>
<td>217</td>
<td>102</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Heterogeneity: Tau² = 0.83, Chi² = 52.47, df = 12 (P < 0.00001); I² = 77%
Test for overall effect: Z = 5.61 (P < 0.00001)

Table 3. Study Outcomes.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>No Bridging (N = 916)</th>
<th>Bridging (N = 895)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary</td>
<td>number of patients (percent)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arterial thromboembolism</td>
<td>4 (0.4)</td>
<td>3 (0.3)</td>
<td>0.01*, 0.73†</td>
</tr>
<tr>
<td>Stroke</td>
<td>2 (0.2)</td>
<td>3 (0.3)</td>
<td></td>
</tr>
<tr>
<td>Transient ischemic attack</td>
<td>2 (0.2)</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Systemic embolism</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Major bleeding</td>
<td>12 (1.3)</td>
<td>29 (3.2)</td>
<td>0.005†</td>
</tr>
<tr>
<td>Secondary</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Death</td>
<td>5 (0.5)</td>
<td>4 (0.4)</td>
<td>0.88†</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>7 (0.8)</td>
<td>14 (1.6)</td>
<td>0.10†</td>
</tr>
<tr>
<td>Deep-vein thrombosis</td>
<td>0</td>
<td>1 (0.1)</td>
<td>0.25†</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>0</td>
<td>1 (0.1)</td>
<td>0.25†</td>
</tr>
<tr>
<td>Minor bleeding</td>
<td>110 (12.0)</td>
<td>187 (20.9)</td>
<td>&lt;0.001†</td>
</tr>
</tbody>
</table>

* P value for noninferiority.
† P value for superiority.


<table>
<thead>
<tr>
<th>Author (year)</th>
<th>Patients (n)</th>
<th>Study Type</th>
<th>AF Monitoring</th>
<th>Follow-Up</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glotzer et al (2003)</td>
<td>312</td>
<td>Secondary analysis of multicenter RCT (MOST)</td>
<td>Dual-chamber PPM</td>
<td>Median 27 mo</td>
<td>10 patients (3.2%) developed stroke</td>
</tr>
<tr>
<td>Capucci et al (2005)</td>
<td>725</td>
<td>Prospective registry (AT500 Registry)</td>
<td>Dual-chamber PPM</td>
<td>Median 22 mo</td>
<td>14 patients (1.9%) developed a thromboembolic event (11 were stroke or TIA)</td>
</tr>
<tr>
<td>Botto et al (2009)</td>
<td>568</td>
<td>Prospective observational study</td>
<td>Dual-chamber PPM</td>
<td>1 y</td>
<td>14 patients (2.5%) developed stroke or systemic embolism</td>
</tr>
<tr>
<td>Glotzer et al (2009)</td>
<td>2486</td>
<td>Prospective observational study (TRENDS)</td>
<td>Dual-chamber PPM or ICD</td>
<td>Mean 1.4 y</td>
<td>Annual thromboembolic risk was 1.1% for patients with no AF, 1.1% for patients with AF episodes lasting &lt;5.5 h (low burden), and 2.1% for patients with AF episodes lasting ≥5.5 h (high burden)</td>
</tr>
<tr>
<td>Healey et al (2012)</td>
<td>2560</td>
<td>Primary analysis of RCT (ASSERT)</td>
<td>Dual-chamber PPM or ICD</td>
<td>Mean 2.5 y</td>
<td>Annual rate of thromboembolism 1.69% in patients with atrial tachyarrhythmia episodes lasting &gt;6 min compared with 0.69% in patients with episodes ≤6 min (HR 1.76, P=0.05)</td>
</tr>
</tbody>
</table>

30-day cumulative AF burden ≥10.8 h showed a trend toward association with an increased risk of thromboembolism (HR 2.22, P=0.06)
Prognostic Implications

Dementia

Bunch TJ et al *Heart Rhythm* 2010 Apr;7(4):433-7
Risk of LAA-related stroke

**CHA$_2$DS$_2$-VASc Scores: LAA vs Aortic plaque**

- **SPAF-TEE study**: Of 332 High-risk AF patients with CHF, prior stroke, female sex, Age >75. (One or more)

![Graph showing prevalence and reduction of Stroke Risk for LAA thrombus/contrast and Complex aortic plaque](image)

LA appendage closure

Endovascular

Epicardial

Courtesy of Randall Lee, MD
2. Risks of Stroke Prevention
Watchman Procedural Risks

Patients with Safety Event (%)

<table>
<thead>
<tr>
<th>Device</th>
<th>1st Half</th>
<th>2nd Half</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>PROTECT AF</td>
<td>9.9%</td>
<td>4.8%</td>
<td>4.1%</td>
</tr>
<tr>
<td>CAP</td>
<td>4.1%</td>
<td>4.1%</td>
<td>3.8%</td>
</tr>
<tr>
<td>PREVAIL</td>
<td>4.1%</td>
<td>4.1%</td>
<td>3.8%</td>
</tr>
<tr>
<td>CAP2</td>
<td>3.8%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Proportions of patients with safety event

Learning Curve


Thanks
What is Atrial Fibrillation?
Disorganized atrial electrical activity

Atrial Fibrillation: Future

Go AS et al JAMA 2001;285:2370-75
Definitions

Paroxysmal AF
• AF that terminates spontaneously or with intervention within 7 d of onset. Episodic may recur with variable frequency.

Persistent AF
• Continuous AF that is sustained >7 d.

Long-standing persistent AF
• Continuous AF >12 mo in duration.

Permanent AF
• The patient and clinician make a joint decision to stop further attempts to restore and/or maintain sinus rhythm. Acceptance of AF represents a therapeutic attitude on the part of the patient and clinician rather than an inherent pathophysiological attribute of AF. Acceptance of AF may change as symptoms, efficacy of therapeutic interventions, and patient and clinician preferences evolve.

Nonvalvular AF
• AF in the absence of rheumatic mitral stenosis, a mechanical or bioprosthetic heart valve, or mitral valve repair.
Treatment Goals

• #1: Symptom suppression

• #2: Improve outcomes:
  – Prevent strokes
  – Prevent tachycardia-induced cardiomyopathy
  – Prevent dementia?
  – Reduce mortality?

• Approaches:
  – Rhythm control
  – Rate control/anticoagulation
## Rate control

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>COR</th>
<th>LOE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control ventricular rate using a beta blocker or nondihydropyridine calcium channel antagonist for paroxysmal, persistent, or permanent AF</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>IV beta blocker or nondihydropyridine calcium channel blocker is recommended to slow ventricular heart rate in the acute setting in patients without pre-excitation. In hemodynamically unstable patients, electrical cardioversion is indicated</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>For AF, assess heart rate control during exertion, adjusting pharmacological treatment as necessary</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>A heart rate control (resting heart rate &lt;80 bpm) strategy is reasonable for symptomatic management of AF</td>
<td>IIa</td>
<td>B</td>
</tr>
<tr>
<td>IV amiodarone can be useful for rate control in critically ill patients without pre-excitation</td>
<td>IIa</td>
<td>B</td>
</tr>
<tr>
<td>AV nodal ablation with permanent ventricular pacing is reasonable when pharmacological therapy is inadequate and rhythm control is not achievable</td>
<td>IIa</td>
<td>B</td>
</tr>
<tr>
<td>A lenient rate-control strategy (resting heart rate &lt;110 bpm) may be reasonable when patients remain asymptomatic and LV systolic function is preserved</td>
<td>IIb</td>
<td>B</td>
</tr>
<tr>
<td>Oral amiodarone may be useful for ventricular rate control when other measures are unsuccessful or contraindicated</td>
<td>IIb</td>
<td>C</td>
</tr>
<tr>
<td>AV nodal ablation should not be performed without prior attempts to achieve rate control with medications</td>
<td>III: Harm</td>
<td>C</td>
</tr>
<tr>
<td>Nondihydropyridine calcium channel antagonists should not be used in decompensated HF</td>
<td>III: Harm</td>
<td>C</td>
</tr>
<tr>
<td>With pre-excitation and AF, digoxin, nondihydropyridine calcium channel antagonists, or amiodarone should not be administered</td>
<td>III: Harm</td>
<td>B</td>
</tr>
<tr>
<td>Dronedarone should not be used to control ventricular rate with permanent AF</td>
<td>III: Harm</td>
<td>B</td>
</tr>
</tbody>
</table>
Rhythm control
Cardioversion – electrical or chemical

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>COR</th>
<th>LOE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prevention of thromboembolism</td>
<td></td>
<td></td>
</tr>
<tr>
<td>With AF or atrial flutter for ≥48 h, or unknown duration, anticoagulate with warfarin for at least 3 wk before and 4 wk after cardioversion</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>With AF or atrial flutter for &gt;48 h or unknown duration, requiring immediate cardioversion, anticoagulate as soon as possible and continue for at least 4 wk</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>With AF or atrial flutter &lt;48 h and high stroke risk, IV heparin or LMWH, or factor Xa or direct thrombin inhibitor, is recommended before or immediately after cardioversion, followed by long-term anticoagulation</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Following cardioversion of AF, long-term anticoagulation should be based on thromboembolic risk</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>With AF or atrial flutter for ≥48 h or unknown duration and no anticoagulation for preceding 3 wk, it is reasonable to perform TEE before cardioversion and then cardiovert if no LA thrombus is identified, provided anticoagulation is achieved before TEE and maintained after cardioversion for at least 4 wk</td>
<td>Ila</td>
<td>B</td>
</tr>
<tr>
<td>With AF or atrial flutter ≥48 h or unknown duration, anticoagulation with dabigatran, rivaroxaban, or apixaban is reasonable for ≥3 wk before and 4 wk after cardioversion</td>
<td>Ila</td>
<td>C</td>
</tr>
<tr>
<td>With AF or atrial flutter &lt;48 h and low thromboembolic risk, IV heparin, LMWH, a new oral anticoagulant, or no antithrombotic may be considered for cardioversion</td>
<td>Iib</td>
<td>C</td>
</tr>
<tr>
<td>Direct-current cardioversion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardioversion is recommended for AF or atrial flutter to restore sinus rhythm. If unsuccessful, cardioversion attempts may be repeated.</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Cardioversion is recommended for AF or atrial flutter with RVR, that does not respond to pharmacological therapies</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Cardioversion is recommended for AF or atrial flutter and pre-excitation with hemodynamic instability</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>It is reasonable to repeat cardioversion in persistent AF when sinus rhythm can be maintained for a clinically</td>
<td>Ila</td>
<td>C</td>
</tr>
</tbody>
</table>
Rhythm Control: AAD

No Structural Heart Disease

- Dofetilide
- Dronedarone
- Flecaïnide
- Propafenone
- Sotalol

→ Catheter ablation

→ Amiodarone

Structural Heart Disease

- CAD
- HF

- Dofetilide
- Dronedarone
- Sotalol

→ Catheter ablation

→ Amiodarone

Amiodarone

J Am Coll Cardiol. 2014;64(21):2246-2280. doi:10.1016/j.jacc.2014.03.021
Atrial Fibrillation Mechanisms:
*It is not so clear*

Calkins et al *Heart Rhythm* 2012
Atrial Fibrillation Ablation Strategies

Calkins et al *Heart Rhythm* 2012
Strategies and targets

- Pulmonary vein isolation
- Wide area circumferential ablation
- Antral isolation
- Complex and fractionated potential ablation
- Ganglionic vagal ablation
- Left atrial posterior linear ablation
- Mitral isthmus linear ablation

- Ectopic foci from the pulmonary veins
- Vagal innervation
- Triggers from the vein of Marshall
- Rotors in the posterior left atrium
- Elimination of iatrogenic flutter
- Rotor-anchoring and wavebreak sites

• ROTOR AND FOCAL ACTIVATION MAPPING
# Table 3  Recommendations regarding ablation technique

- Ablation strategies that target the PVs and/or PV antrum are the cornerstone for most AF ablation procedures.
- If the PVs are targeted, electrical isolation should be the goal.
- Achievement of electrical isolation requires, at a minimum, assessment and demonstration of entrance block into the PV.
- Monitoring for PV reconduction for 20 minutes following initial PV isolation should be considered.
- For surgical PV isolation, entrance and/or exit block should be demonstrated.
- Careful identification of the PV ostia is mandatory to avoid ablation within the PVs.
- If a focal trigger is identified outside a PV at the time of an AF ablation procedure, ablation of that focal trigger should be considered.
- If additional linear lesions are applied, operators should consider using mapping and pacing maneuvers to assess for line completeness.
- Ablation of the cavo-tricuspid isthmus is recommended in patients with a history of typical atrial flutter or inducible cavo-tricuspid isthmus dependent atrial flutter.
- If patients with longstanding persistent AF are approached, operators should consider more extensive ablation based on linear lesions or complex fractionated electrograms.
- It is recommended that RF power be reduced when creating lesions along the posterior wall near the esophagus.

Calkins et al et al *Heart Rhythm* 2012
Symptom control

Ablation as first-line?

Wazni et al JAMA 2005;293:2634

Radiofrequency Ablation vs Antiarrhythmic Drugs as First-line Treatment of Symptomatic Atrial Fibrillation
A Randomized Trial

Nielsen et al NEJM 2012;367:1587

Primary endpoint: Symptomatic AF

Morillo et al JAMA 2014;311:692

Primary endpoint: Time to documented atrial tachyarrhythmia

Primary endpoint: AF burden

The NEW ENGLAND JOURNAL of MEDICINE

Radiofrequency Ablation as Initial Therapy in Paroxysmal Atrial Fibrillation


Editorial page 679
Supplemental content at jama.com
Decreased symptomatic AF

Wazni et al JAMA 2005;293:2634
Decreased AF burden?
Nielsen et al *NEJM* 2012;367:1587
Decreased AF burden?

Nielsen et al. NEJM 2012;367:1587

AF elimination on 7-day Holter

<table>
<thead>
<tr>
<th>No Atrial Fibrillation on 7-Day Holter-Monitor Recording (no. of patients)</th>
<th>Ablation</th>
<th>Drug therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>61</td>
<td>66</td>
</tr>
<tr>
<td>3 Months</td>
<td>97</td>
<td>91</td>
</tr>
<tr>
<td>6 Months</td>
<td>112</td>
<td>103</td>
</tr>
<tr>
<td>12 Months</td>
<td>120</td>
<td>106</td>
</tr>
<tr>
<td>18 Months</td>
<td>122</td>
<td>109</td>
</tr>
<tr>
<td>24 Months</td>
<td>124</td>
<td>105</td>
</tr>
<tr>
<td>Cumulative</td>
<td>77</td>
<td>65</td>
</tr>
</tbody>
</table>

Whitney test

| P Value | 0.72 | 0.49 | 0.34 | 0.08 | 0.07 | 0.007 | 0.10 |

85% vs 71%
Decreased AF burden

Nielsen et al NEJM 2012;367:1587

90th percentile of AF burden dropped by half

9% vs. 18%; P=0.007
Reduced AF recurrence
Morillo et al JAMA 2014;311:692

A Primary efficacy outcome

B Time to first recurrence of symptomatic atrial tachyarrhythmias

All Recurrences in 54.5% (ablation) vs 72.1% (drugs)

Symptomatic Recurrence in 47% (ablation) vs 59% (drugs)
Goal #1: Symptom control

- Freedom from *symptomatic* atrial fibrillation-flutter:
  - FIRST LINE: No prior AAD:
    - 93% vs. 84%, *P*=0.01 (Nielsen et al NEJM 2012;367:1587)
      - AAD group: 36% of patients underwent ablation.
      - Nonsignificant effects up to 24 months
    - 87% vs 47%; *p*<0.001 (Wazni et al JAMA 2005;293;2634)
    - 53% vs 41%; *p*=0.03 (Morillo CA et al JAMA 2015;311;692)
  - Prior AAD:
    - STOP-AF: 81% vs 7%, (Packer et al JACC 2013;61;1713)
    - Thermocool AF: 70% vs 19% (Wilber et al JAMA. 2010;303:333)

*As first-line therapy, symptom control can be achieved in a substantial fraction of patients with drug therapy*
Symptom control at what price?

Complications

- Pericardial bleeding and Tamponade (1%)
- TIA/Stroke (0.5 – 1%)
- Atrio-esophageal fistula (0.01%)
- Phrenic nerve paralysis (0.1%)
- Pulmonary vein stenosis (0.5%)
- Laryngeal nerve paralysis
- Vascular access complications (0.5-1%)
- Gastroparesis
- Death (0.05-0.1%)
Goal #2: Stroke prevention

- No prospective data [CABANA trial]
- Observational studies:

Oral et al *Circulation* 2006;114:759
Stroke prevention
AF ablation ~ no AF

AF ablation, n=4212
AF no ablation, n=16848
No AF, n=16848

Bunch TJ et al *Heart Rhythm* 2013;10:1272
Stroke Prevention


**A**

Vascular events only

- Af patients with medical control
- Ablation patients without recurrence
- Ablation patients with recurrence

Log Rank Test: $P=0.004$

**B**

Stroke / TIA

- Af patients with medical control
- Ablation patients without recurrence
- Ablation patients with recurrence

Log Rank Test: $P=0.015$
Mortality

Ablation Beyond Symptoms: CABANA TRIAL
When is AF ablation appropriate? INDIVIDUALIZE!

AF HETEROGENEITY
- Symptoms
- AF burden
- Structural disease
- Prognostic impact:
  - Stroke (CHADS-VASc)
  - Dementia

AAD CHOICE AND SUCCESS
- Paroxysmal vs Persistent
- Structural disease
- Compliance long-term

ABLATION SUCCESS
- Paroxysmal vs Persistent
- Structural disease
- Risks
CARDIOVASCULAR FELLOWS’ BOOTCAMP

ATRIAL FIBRILLATION

Miguel Valderrábano

Division of Cardiac Electrophysiology, Department of Cardiology, Methodist DeBakey Heart and Vascular Center, Houston Methodist Hospital, Houston, TX
Selecting the Right Strategy for the Right Patient: NOAC, Warfarin, or LAA Closure?

Miguel Valderrábano

Division of Cardiac Electrophysiology, Department of Cardiology, Methodist DeBakey Heart and Vascular Center, Houston Methodist Hospital, Houston, TX

ACC 2016
2. Risks of Stroke Prevention
Bleeding on Warfarin vs Watchman

Free of Major Bleeding Event (%)

<table>
<thead>
<tr>
<th>Time (days)</th>
<th>Warfarin +Aspirin</th>
<th>Warfarin +Aspirin</th>
<th>Aspirin+ Clopidogrel</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>100</td>
<td>100</td>
<td>70</td>
</tr>
<tr>
<td>7</td>
<td>90</td>
<td>90</td>
<td>90</td>
</tr>
<tr>
<td>8</td>
<td>90</td>
<td>90</td>
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</tr>
<tr>
<td>45</td>
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<td>46</td>
<td>90</td>
<td>90</td>
<td>90</td>
</tr>
<tr>
<td>180</td>
<td>90</td>
<td>90</td>
<td>90</td>
</tr>
</tbody>
</table>

Definition of bleeding: Serious bleeding event that required intervention or hospitalization according to adjudication committee

HR = 0.29  
p<0.001

71% Relative Reduction In Major Bleeding after cessation of anti-thrombotics
Making decisions

- Extreme risk: LAA thrombus, other diagnoses requiring anticoagulation

First choice
- Financial constrains
- Stable INRs
- No bleeding
- Good tolerance

Bleeding
- Stroke on anticoagulation
- Poor tolerance
- Hemorrhagic stroke
- Procedural candidacy
- High LAA-risk

NOACs
Warfarin
Watchman
## Specific disease recommendations

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>COR</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hypertrophic cardiomyopathy</strong></td>
<td></td>
</tr>
<tr>
<td>Anticoagulation is indicated in HCM with AF independent of the CHA2DS2-VASc score</td>
<td>I</td>
</tr>
<tr>
<td>Antiarrhythmic drugs can be useful to prevent recurrent AF in HCM. Amiodarone or disopyramide combined with a beta blocker or nondihydropyridine calcium channel antagonist are reasonable</td>
<td>IIa</td>
</tr>
<tr>
<td>AF catheter ablation can be beneficial for HCM to facilitate a rhythm-control strategy when antiarrhythmics fail or are not tolerated</td>
<td>IIa</td>
</tr>
<tr>
<td>Sotalol, dofetilide, and dronedarone may be considered for a rhythm-control strategy in HCM</td>
<td>IIb</td>
</tr>
<tr>
<td><strong>AF complicating ACS</strong></td>
<td></td>
</tr>
<tr>
<td>Urgent cardioversion of new-onset AF in the setting of ACS is recommended for patients with hemodynamic compromise, ongoing ischemia, or inadequate rate control</td>
<td>I</td>
</tr>
<tr>
<td>IV beta blockers are recommended to slow RVR with ACS and no HF, hemodynamic instability, or bronchospasm</td>
<td>I</td>
</tr>
<tr>
<td>With ACS and AF with CHA2DS2-VASc score ≥2, anticoagulation with warfarin is recommended unless contraindicated</td>
<td>I</td>
</tr>
<tr>
<td>Amiodarone or digoxin may be considered to slow RVR with ACS and AF and severe LV dysfunction and HF or hemodynamic instability</td>
<td>IIb</td>
</tr>
<tr>
<td>Nondihydropyridine calcium antagonists might be considered to slow RVR with ACS and AF only in the absence of significant HF or hemodynamic instability</td>
<td>IIb</td>
</tr>
<tr>
<td><strong>Hyperthyroidism</strong></td>
<td></td>
</tr>
<tr>
<td>Beta blockers are recommended to control ventricular rate with AF complicating thyrotoxicosis unless contraindicated</td>
<td>I</td>
</tr>
</tbody>
</table>