Atrial Fibrillation
Where We’ve Been and What Lies Ahead

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Patient Presentation

• A 76 year old male with history of hypertension and hyperlipidemia presents for routine examination. You detect an irregular pulse and heart rate. An EKG confirms atrial fibrillation with a heart rate of 85.

• New Diagnosis of Atrial Fibrillation
  – What tests?
  – What medications?
Atrial Fibrillation

• Most common sustained arrhythmia
• Estimated 2.3 million patients in the United States
• Incidence of 3.8% in patients >60 years
• Incidence of 9% is patients >80 years
• Increases relative risk of death 1.3-2x
<table>
<thead>
<tr>
<th>Cardiac causes</th>
<th>Noncardiac causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertensive heart disease</td>
<td>Autonomically mediated (sympathetic or parasympathetic)</td>
</tr>
<tr>
<td>Valvular disease</td>
<td>Toxin exposure</td>
</tr>
<tr>
<td>Coronary heart disease</td>
<td>Endocrinopathy (especially thyroid disease)</td>
</tr>
<tr>
<td>Cardiomyopathy (all forms)</td>
<td>Pulmonary disease</td>
</tr>
<tr>
<td>Pericardial disease</td>
<td>Neurologic disorders</td>
</tr>
<tr>
<td>Intracardiac masses</td>
<td>Idiopathic</td>
</tr>
<tr>
<td>Electrical disease</td>
<td></td>
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<tr>
<td>Sinus node dysfunction</td>
<td></td>
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<tr>
<td>Tachycardia-induced</td>
<td></td>
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<tr>
<td>Familial</td>
<td></td>
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<tr>
<td>Cardiothoracic surgery</td>
<td></td>
</tr>
<tr>
<td>Congenital heart disease</td>
<td></td>
</tr>
</tbody>
</table>
Etiology - Hypertensive

• Hypertensive heart disease
  – Accounts for about 50% of cases in developed countries
  – May be due to LA dilatation secondary to decreased LV compliance
  – Associated CAD
Etiology - Valvular

• Valvular Heart Disease
  – Mitral stenosis due to rheumatic disease
  – Increased stroke risk – 20% of patients with AF and MS will have embolic event
  – Stroke risk 3-7x that of sinus rhythm with MS
  – AF is infrequent with isolated Aortic stenosis
Etiology - Surgery

• Cardiac Surgery
  – Common complication of cardiac surgery
  – 20-40% incidence following CABG, often postoperative days 2-8
  – Risk of AF following surgery
    • Elderly
    • Prior AF
    • Right coronary artery stenosis
    • Beta blockers discontinued preoperatively
Etiology - Other

• Thyroid disease
  – Occurs in 20-25% of elderly with thyrotoxicosis
  – About 1% of new onset AF is due to hyperthyroidism

• Alcohol
  – Common cause of AF
  – Seen in up to 60% of binge drinkers
  – AF episodes coincide with heavy intake

• Cardiomyopathy
  – AF present in 28% of patients with hypertrophic cardiomyopathy
  – AF occurs in 20% of those with dilated cardiomyopathy

• Familial
  – Autosomal dominant – chromosome 10q22-q24

Crawford
Mechanism of Atrial Fibrillation

MECHANISMS OF ATRIAL FIBRILLATION

- Multiple simultaneous re-entrant circuits
- Rapid single circuit or focal source
Sinus Rhythm vs. Atrial Fibrillation

http://www.heartpoint.com/
Clinical Classification

- Acute – AF related to transient or reversible cause, or 1st episode of AF
- Chronic
  - Paroxysmal – self-terminating AF
  - Sustained
    - Persistent – AF that can be cardioverted
    - Permanent – AF that is resistant to cardioversion or inappropriate for cardioversion

Situational variants
  - Vagal mediated – occurs at night or after meals
  - Adrenergic mediated – AF during exercise, stress

Paroxysmal AF may become chronic
  (8% at 1 year, 18% at 4 years)
Symptoms

• Asymptomatic – discovered by auscultation, pulse palpation, EKG, or Holter

• Major symptoms
  – Heart Failure
  – Angina
  – Hypotension
  – Presyncope
  – Syncope – usually with pre-excitation, hypertrophic cardiomyopathy, or aortic stenosis
  – Stroke
  – Systemic Embolization
Symptoms

• Minor symptoms
  – Palpations
  – Racing heart
  – Fatigue
  – Light-headedness
  – Increased urination
  – Shortness of breath
### Initial Evaluation

**INITIAL EVALUATION OF PATIENTS WITH ATRIAL FIBRILLATION**

**Minimum evaluation**
- History and physical exam
- ECG
- Chest X-ray
- Echocardiogram
- Laboratory studies – thyroid, renal function

**Optional studies**
- Exercise testing or ambulatory ECG
- Transesophageal echocardiogram
- Electrophysiologic study

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History

- Symptoms
- Sustained or intermittent
- Complications
- Precipitating factors
- Relief of symptoms
- Duration/Frequency
- Prior treatment
Blood Tests

• Complete Blood Count
• Electrolytes
• Renal function
• Thyroid function
Chest X-ray

• May show congenital heart disease (ASD)
• Heart Size
• Presence of heart failure
• Coexisting intrathoracic pathology
EKG

- Rapid baseline oscillations
- Irregularly Irregular ventricular rate
- Absence of P waves
- ? Etiology
  - Left ventricular hypertrophy
  - Prior myocardial infarction
  - Pre-excitation
Atrial Fibrillation on EKG

http://www.ecglibrary.com/af_fast.html
Echocardiography

• Structural heart disease?
  – Valvular abnormalities
  – Congenital defects
  – Chamber size
    • Significant left atrial enlargement reduces success of cardioversion and long term maintenance of sinus rhythm. Also, LAE may increase risk of stroke
  – Pericardial thickening or effusion
  – Ventricular function
Electrophysiologic testing

- Limited role
- Atrial flutter or Supraventricular tachycardia is cause of atrial fibrillation
- Other symptoms (pre-excitation, sinus node dysfunction, syncope) need clarification
- Focal source amenable to ablation
Other studies

• Exercise stress testing
  – Anginal symptoms during episodic atrial fibrillation with rapid ventricular response or independent of atrial fibrillation
  – Assess for rate control during drug therapy

• Cardiac catheterization
  – Usually only indicated if symptoms or noninvasive tests suggest active ischemia
Pre-Management Assessment

• Are there any other associated arrhythmias or conduction abnormalities?
  – Pre-excitation
  – AV block

• Are there predisposing factors? Are they reversible or preventable?

• Is there a need for urgent intervention?
  – Hemodynamic instability

• Is there a need for rhythm control, or is rate control sufficient?
Acute Atrial Fibrillation Management

- Hemodynamic compromise – DC cardioversion
- Consider IV Heparin
- Rate control
  - Beta blockers
  - Calcium Channel Blockers
  - Digoxin
- Cardioversion if <48 hours duration
Paroxysmal Atrial Fibrillation Management

• Goals
  – Reduce frequency of paroxysms
  – Control rate during paroxysms
  – Prevent thromboembolism

• Digoxin may increase frequency and duration of paroxysms

• Calcium channel and Beta blockers may control ventricular rate, but not reduce frequency of attacks

• Antiarrhythmic therapy
  – Flecainide or propafenone considered in absence of structural heart disease
  – “Pill in the pocket” strategy
Chronic Atrial Fibrillation Management

• Underlying etiology?
• Rate control vs rhythm control
  – Heart rate 60-80 at rest, 90-115 during moderate exercise
• Thromboembolic prophylaxis
Risks for cardioversion failure and failure to maintain sinus rhythm

- Advanced age
- Duration of atrial fibrillation
  - Unlikely to maintain sinus rhythm when atrial fibrillation > 2 years duration
- Uncontrolled hypertension
- Severity of structural heart disease
  - Severe left atrial dilatation
- Other systemic diseases
Cardioversion

- Thromboembolism rare when AF duration is <48 hours
- When AF duration is >48 hours, thromboembolism occurs in 7% when no anticoagulation is used
- Most embolic events occur in 1st week after cardioversion
TEE before cardioversion

- Atrial fibrillation >48h or unknown
- Used to minimize duration of atrial fibrillation or reduce total anticoagulation time
- Evaluate for thrombus in the left atrial appendage
- If no thrombus, then may cardiovert followed by anticoagulation x 4 weeks
- If thrombus present, anticoagulation x 4 weeks then re-evaluate with TEE
Thrombus in left atrial appendage

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Anticoagulation and Cardioversion

<table>
<thead>
<tr>
<th>Duration of arrhythmia</th>
<th>Anticoagulation before cardioversion</th>
<th>Anticoagulation after cardioversion</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;48 hours</td>
<td>Not required</td>
<td>Optional based on risk for recurrence</td>
</tr>
<tr>
<td>&gt;48 hours</td>
<td>Warfarin to achieve INR of 2–3 for 3 weeks, or</td>
<td>Warfarin to achieve INR of 2–3 for &gt;4 weeks</td>
</tr>
<tr>
<td></td>
<td>Transesophageal echo-cardiogram negative for thrombus</td>
<td>Heparin, then warfarin to achieve INR of 2–3 for &gt;4 weeks</td>
</tr>
</tbody>
</table>
Electrical Cardioversion

• Synchronized
• 200 J monophasic or 125J biphasic
• Ibutilide or other class III antiarrythmic may facilitate cardioversion
• Reports of intracardiac shock or transthoracic shock up to 720J used in refractory cases
Nonembolic complications of electrical cardioversion

- Ventricular arrhythmia
- Sinus bradycardia
- Hypotension
- Pulmonary edema
- Skin burns
- Transient ST and T wave abnormalities
Torsades de Pointes
Following chemical cardioversion with ibutilide
Chemical cardioversion

- Many cases of new onset atrial fibrillation will spontaneously convert to sinus rhythm within 48 hours.

### Antiarrhythmic Drug Doses for Pharmacological Cardioversion and Prevention of Atrial Fibrillation Recurrences

<table>
<thead>
<tr>
<th>Class</th>
<th>Drug</th>
<th>iv or oral therapy for rapid conversion</th>
<th>Chronic oral drug therapy to prevent recurrence*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class IA drugs</td>
<td>Procainamide</td>
<td>500–1200mg iv over 30–60 minutes</td>
<td>2000–4000mg/day</td>
</tr>
<tr>
<td></td>
<td>Quinidine sulfate</td>
<td>Not recommended</td>
<td>600–1200mg/day</td>
</tr>
<tr>
<td></td>
<td>Disopyramide</td>
<td>Not recommended</td>
<td>450–600mg/day</td>
</tr>
<tr>
<td>Class IC drugs</td>
<td>Flecainide</td>
<td>1.5–3.0mg/kg iv over 10 minutes†</td>
<td>150–300mg/day</td>
</tr>
<tr>
<td></td>
<td></td>
<td>200–400mg po</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Propafenone</td>
<td>1.5–2mg/kg IV over 10–20 minutes†</td>
<td>400–600mg/day</td>
</tr>
<tr>
<td></td>
<td></td>
<td>300–450 mg po</td>
<td></td>
</tr>
<tr>
<td>Class III drugs</td>
<td>Ibutilide</td>
<td>1mg iv over 10 minutes, repeat once</td>
<td>Not available</td>
</tr>
<tr>
<td></td>
<td>Sotalol</td>
<td>Not recommended</td>
<td>160–320mg/day</td>
</tr>
<tr>
<td></td>
<td>Amiodarone</td>
<td>5–7mg/kg iv over 30 minutes then 1.2–1.8g/day</td>
<td>400–1200mg/day for 7 days, then taper to 100–300mg/day</td>
</tr>
<tr>
<td></td>
<td>Dofetilide</td>
<td>Loading not recommended</td>
<td>125–500μg q 12h</td>
</tr>
</tbody>
</table>
Antiarrhythmic therapy
Maintenance of sinus rhythm

• Amiodarone superior to sotalol and class I drugs
• Sotalol equivalent to class I drugs
• Risk of proarrythmia
  – Sinus node dysfunction or AV block
  – Class IA and III prolong QT interval
  – Class IA, IC, and amiodarone can cause atrial flutter; in absence of AV blockade may cause hemodynamic collapse with 1:1 conduction
Flecainide (IC) causing atrial flutter with 1:1 conduction
Rate Control

- **Digoxin**
  - Enhances vagal tone, prolongs AV nodal refractory period
  - Less effect during stress, fever, etc.
  - Onset of action several hours (even IV)
- **Beta Blockers**
  - Decrease resting heart rate and blunt HR response to exercise
  - May worsen vagally mediated atrial fibrillation
- **Calcium Channel Blockers**
  - Slow conduction in the AV node
  - Negative inotropes (especially verapamil)
## Medications for rate control

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Acute intravenous therapy</th>
<th>Chronic oral therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beta blockers</td>
<td>Metoprolol: 2.5–5mg every 5 minutes up to 15mg</td>
<td>50–200mg/day</td>
</tr>
<tr>
<td></td>
<td>Propranolol: 0.15mg/kg (1mg every 2 minutes)</td>
<td>40–240mg/day</td>
</tr>
<tr>
<td></td>
<td>Esmolol: 0.5mg bolus, then 0.05–0.2mg/kg per minute</td>
<td>NA</td>
</tr>
<tr>
<td></td>
<td>Pindolol: NA</td>
<td>7.5–30mg/day</td>
</tr>
<tr>
<td></td>
<td>Atenolol: 5mg over 5 minutes, repeat in 10 minutes</td>
<td>25–100mg/day</td>
</tr>
<tr>
<td></td>
<td>Nadolol: NA</td>
<td>20–80mg/day</td>
</tr>
<tr>
<td>Calcium channel</td>
<td>Verapamil: 0.075–0.15mg/kg over 2 minutes; 0.005mg/kg per minute</td>
<td>120–360mg/day</td>
</tr>
<tr>
<td>blockers</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Diltiazem: 0.25–0.35mg/kg followed by 5–15mg/hour</td>
<td>120–360mg/day</td>
</tr>
<tr>
<td>Cardiac glycoside</td>
<td>Digoxin: 0.75mg–1.5mg in divided doses over 12–24 hours</td>
<td>0.125mg–0.375mg/day</td>
</tr>
</tbody>
</table>
Nonpharmacologic Therapies for rate control

- Pacemaker therapy
- Catheter ablation of AV node
- Catheter and surgical ablation

- Reserved for patients refractory to standard medical management
Pacemaker therapy

• Typically used in setting of sinus node dysfunction or AV block
• In sick sinus syndrome, atrial pacing results in much less atrial fibrillation than ventricular pacing
• In permanent atrial fibrillation, VVIR is the pacing mode of choice
• For selected patients atrial defibrillators are available to sense AF and either pace rapidly or shock to convert to sinus rhythm
Catheter ablation of AV node

• For patients resistant to medical rate control

• Requires implantation of pacemaker at time of ablation
  – VVIR mode for permanent atrial fibrillation
  – DDDR with mode switching for paroxysmal atrial fibrillation

• Must still risk stratify for thromboembolism and anticoagulate if indicated!
Surgical ablation

• Maze procedure (and variants) – multiple linear incisions in both atria, excision of both atrial appendages, and isolation of pulmonary veins
  – Complications of fluid retention, atrial arrhythmia

• Radiofrequency – lesion made on endocardium via atriotomy during open heart surgery
Pulmonary Vein Ablation (Percutaneous)

- 1,171 Symptomatic patients with AF
- 589 to ablation, 582 to antiarrhythmic therapy (not randomized)
  - Ablation patients off coumadin after 4 weeks
  - Amiodarone, flecainide, propafenon, sotalol most common antiarrythmics
- Median followup 900 days
- Ablation improved Mortality (92% vs 86% at 3 years), Morbidity (Heart failure, CVA, AF recurrence, and Quality of Life scores
Worldwide Survey of Atrial Fibrillation Ablation

- 181 of 777 Worldwide centers surveyed
- 1995 – 18 procedures; 2002 – 5005 procedures
- Patient results
  - 52% asymptomatic without drugs
  - 24% asymptomatic with drugs
  - 27% required >1 procedure
  - 6% major complications
    - 0.05% death  1.22% Tamponade  0.28% Stroke
    - 1.6% Pulmonary vein stenosis

Atrial Fibrillation technique

- June 2005 German study
  - 50 patients circumferential PV ablation
  - 50 Segmental PV ablation
- Not much difference at 6 months
- Circumferential: More symptomatic Atrial Flutter
- Segmental: More pulmonary vein stenosis

Karch et al., 2005
Rate control vs Rhythm Control

• Previous belief that maintenance of sinus rhythm improved morbidity and mortality
• Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM)
  – 4060 patients, at 3.5 years trend toward lower mortality in rate control group
• 2 smaller trials also showed no stroke or mortality benefit to rhythm control
  – Pharmacological Intervention in Atrial Fibrillation (PIAF)
  – Rate Control vs. Electrical Cardioversion (RACE)
• In asymptomatic patients, either strategy is acceptable
• Lesson from trials: Anticoagulation must be continued with rhythm control
Risk of Stroke

- Thromboembolic stroke, typically due to thrombus in the left atrial appendage
- Risk of stroke 5-9% per year among high risk patients on aspirin (not coumadin)
- Duration of episodes and overall atrial fibrillation burden have not been useful to assess stroke risk
## Stroke Prevention in Atrial Fibrillation Trials

<table>
<thead>
<tr>
<th>Trial</th>
<th>Time Interval</th>
<th>Main Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>SPAF I</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Warfarin vs. placebo</td>
<td>1987–1989</td>
<td>Warfarin substantially reduces stroke</td>
</tr>
<tr>
<td>Aspirin vs. placebo</td>
<td>1987–1990</td>
<td>Aspirin reduces stroke</td>
</tr>
<tr>
<td>SPAF II</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Warfarin vs. aspirin, age ≤ 75 y</td>
<td>1987–1992</td>
<td>Small absolute reduction in stroke by warfarin over aspirin in unselected patients</td>
</tr>
<tr>
<td>Warfarin vs. aspirin, age &gt; 75 y</td>
<td>1989–1992</td>
<td>High rate of intracranial bleeding with warfarin (INR, 2–4.5) in patients &gt;75 years of age offset reduction in ischemic stroke</td>
</tr>
<tr>
<td>SPAF III</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Warfarin INR 2–3 vs. aspirin plus low-intensity, fixed-dose warfarin in selected high-risk patients</td>
<td>1993–1995</td>
<td>Warfarin INR 2–3 offers large benefits over aspirin plus low-intensity, fixed-dose warfarin for high-risk patients</td>
</tr>
<tr>
<td>Aspirin-treated low-risk cohort</td>
<td>1993–1997</td>
<td>Patients whose stroke risk is low when given aspirin can be identified (validation of the SPAF risk stratification scheme)</td>
</tr>
</tbody>
</table>

* All were randomized trials, except the nonrandomized aspirin-treated low-risk cohort clinical trial in SPAF III, in which all participants were prescribed aspirin and followed to validate the stroke risk stratification scheme. INR = international normalized ratio; SPAF = Stroke Prevention in Atrial Fibrillation.

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# SPAF 3 Risk factors

## Table 3. Stroke Prevention in Atrial Fibrillation III Stroke Risk Stratification Scheme*

<table>
<thead>
<tr>
<th>Risk Strata and Criteria</th>
<th>Ischemic Stroke with Aspirin</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Derivation Cohort (n = 854)</td>
</tr>
<tr>
<td>High risk</td>
<td>5.9†</td>
</tr>
<tr>
<td>Previous stroke or transient ischemic attack</td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure &gt; 160 mm Hg</td>
<td></td>
</tr>
<tr>
<td>Heart failure†</td>
<td></td>
</tr>
<tr>
<td>Women &gt; 75 y</td>
<td></td>
</tr>
<tr>
<td>Moderate risk</td>
<td>2.8</td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
</tr>
<tr>
<td>No high-risk features</td>
<td></td>
</tr>
<tr>
<td>Low risk</td>
<td>1.0</td>
</tr>
<tr>
<td>No hypertension</td>
<td></td>
</tr>
<tr>
<td>No high-risk features</td>
<td></td>
</tr>
</tbody>
</table>

* SPAF = Stroke Prevention in Atrial Fibrillation.

† Excluding patients with previous stroke or transient ischemic attack, the annualized rates among remaining high-risk patients with atrial fibrillation (that is, for primary prevention) were 5.8% per year for the derivation data set (27), 5.3% per year for the test cohort (6), and 3.4% per year for the other clinical trials cohort (28).

‡ Congestive heart failure within the previous 3 months or left ventricular fractional shortening of ≤ 25% by precordial echocardiography.

<table>
<thead>
<tr>
<th>Risk Strata</th>
<th>Stroke Rate with Aspirin, %/y</th>
<th>Relative Risk Reduction: Warfarin vs. Aspirin, %†</th>
<th>NNT&lt;sub&gt;B&lt;/sub&gt;‡</th>
<th>General Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous stroke or transient ischemic attack</td>
<td>10</td>
<td>60</td>
<td>17</td>
<td>Warfarin (INR, 2–3)</td>
</tr>
<tr>
<td>Primary prevention</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High risk</td>
<td>&gt; 4</td>
<td>55</td>
<td>35</td>
<td>Warfarin (INR, 2–3)</td>
</tr>
<tr>
<td>Moderate risk</td>
<td>2–4</td>
<td>45</td>
<td>75</td>
<td>Warfarin or aspirin§</td>
</tr>
<tr>
<td>Low risk</td>
<td>&lt; 2</td>
<td>35</td>
<td>&gt; 200</td>
<td>Aspirin (81–325 mg/d)</td>
</tr>
</tbody>
</table>

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Risks of anticoagulation

- Intracranial hemorrhage – 0.1-0.3%/year
- Risk of major bleed – about 2%/year
  - 13-33% risk of death from major bleed
  - 15% risk of morbidity from major bleed
Anticoagulation Recommendations

- Anticoagulate all Valvular associated Atrial Fibrillation
- Assess risk factors (and review annually)
  - Prior TIA or stroke
  - Hypertension
  - Heart failure or Left ventricular dysfunction
  - Diabetes mellitus
  - * Clinical coronary artery disease (not included as a risk factor in ACCP guidelines)
Prognosis

• Framingham study
  – Men: Odds ratio of death 1.5
  – Women: Odds ratio of death 1.9

• Greatest impact on those with advanced heart disease or other comorbidity
Atrial Flutter

- Digoxin, Beta Blockers, Calcium Channel blockers for rate control
- Electrical cardioversion (synchronized, 25-100J) preferred over medications
- Antiarrythmic drugs have modest effect at preventing atrial flutter
- “Typical” atrial flutter very amenable to catheter ablation
Typical Atrial Flutter
Mechanism of Typical Atrial Flutter
So What’s New with Atrial Fibrillation?

- Recognition of upper pulmonary veins as major Atrial fibrillation focus (foci)
- Potential for Atrial Fibrillation Ablation
  - More effective for paroxysmal or persistent atrial fibrillation, rather than chronic sustained AF (25% success)
- Better antiarrhythmics?
  - AVEO118 is a novel K+ channel blocker that prolongs atrial refractory period without affecting the ventricles
- Novel direct thrombin inhibitors
  - Ximelagatran showed efficacy equivalent to coumadin in stroke prophylaxis for atrial fibrillation (SPORTIF III, SPORTIF V trials), however the FDA advisory panel recommended **against** drug approval due to hepatic toxicity
- Mechanical occlusion of left atrial appendage
- “Pill in the Pocket” for paroxysmal atrial fibrillation
  - Propafenone or flecainide for outpatient, episodic use
Left Ventricle
Flap Opened in Posterolateral Wall

Left Atrium and Ventricle
Sectioned with Mitral Valve Cut Away

Note: broken line indicates level of origin of tricuspid valve
Left Atrial Appendage Occlusion
Percutaneous Left Atrial Appendage Transcatheter Occlusion (PLAATO)

- 15 patients with AF, high risk of stroke, poor coumadin candidates
- 1 month follow-up, implant stable by TEE
Back to the patient

- A 76 year old male with hypertension and hyperlipidemia diagnosed with asymptomatic atrial fibrillation, heart rate 85
  - EKG (done)
  - Echocardiography
  - Chest Xray
  - CBC, Renal panel, TSH
  - Rate control with Beta blocker (asymptomatic)
    - Assess adequacy with exercise monitoring if needed
    - May consider cardioversion after 3 weeks of anticoagulation
  - Anticoagulation with coumadin
  - Consider ischemic workup
References