Atrial Fibrillation
Case

**ID:** 25 y o female psych nurse (post-night shift)

**RFR:** left-sided hemiparesis & expressive aphasia

**PMHx:**
- Cardiomyopathy

**CRFs:** non-smoker, no DM, no HTN, no CHOL, no FHX

**Meds:**
- Amiodarone 200 mg OD
- OCP (x 2 mths)
HPI:
• ~1 wk ago, felt some mild tingling/weakness in left fingertips that subsequently resolved
• Presented with 8 hr hx of left-sided hemiparesis & expressive aphasia.
• Pt was feeling extremely restless & was having difficulty relaxing.

P/E:
VS: stable
General: thin, agitated, obvious left-sided facial droop & arm paresis
HEENT: PERL, weakness of Left side
CVS/Resp/Abdo: unremarkable
Neuro: Reflexes (2/4), N tone, N sensation, Power 0/5 left arm
(no cooperation for cerebellar testing or cranial nerve testing)
Investigations:
ECG: NSR
CXR: heart size upper limits of normal
Labs: microcytic anemia
CT: no acute bleed or infarct (small hypodensity in left internal capsule)
MRI: no acute abnormality

Addendum:
• Notes from cardiologist revealed Dx of PAF
• Stopped taking ASA ~ 6 mths ago
Agenda

1. Conduction system
2. Epidemiology
3. Etiology
4. Classification
5. Diagnosis
6. Complications
7. Management
Conduction System
Heart Conduction System

- SA node
- Internodal tracts
- AV node
- His Bundle
- Bundle branches
- Purkinje fibres
SA Node Overdrive Suppression

* Beat generated outside normal pacemaker is an ectopic beat - site of beat = ectopic foci
Phase 0 denotes ventricular depolarization. This is seen on the ECG as the beginning of the QRS complex.

Phase 1 denotes the initial rapid repolarization due to closing of fast sodium channels. This is seen as the large drop in mV on the ECG.

Phase 2 represents the plateau stage during which inflow and outflow currents are balanced. The ECG returns to baseline.

Phase 3 is repolarization. Potassium channels open and calcium closes. The ECG shows the repolarizing T wave.

Phase 4 is the recover phase. Both the muscle tracing and ECG return to baseline levels.

Once the myocardial cells have depolarized, there is a period in which they are refractory to further depolarizations.

During phase 1 and most of phase 2, the depolarized cells cannot invoke another action potential. This is due to continued inactivation of sodium channels.

In phase 3, the sodium channels are reactivated and limited numbers of cells may be depolarized. An effective refractory period exists. In this phase, individual cells may be depolarized, but they are unable to propagate an action potential.
AF: atrial fibrillatory waves (350-600 impulses/min → irregularly irregular ventricular response of 90 up to 140-170 bpm)
Epidemiology
Epidemiology

- AF is a common arrhythmia (most common sustained tachyarrhythmia)
- Clinically important b/c pts at increased risk of mortality (1.5 - 1.9-fold in the Framingham study)

Risk Factors In Atrial Fibrillation (ATRIA) Study. *Jama* 2001;285:2370

- CSS of ~1.9 million subjects in a health maintenance organization. Results:
  - AF primarily disease of older adults, overall prevalence ~1%; 70% were at least 65 y & 45% were >75 y
  - Prevalence of AF ranged from 0.1% < 55y to 9% in those >80 y
  - Prevalence: males > females (1.1 vs 0.8%), a difference seen in every age group
- Pathogenesis?
Etiology/Classification
ATRIAL FIBRILLATION

CARDIAC
- Valvular
- Cardiomyopathy
- CAD/HTN
- Conduction system
- Pericardial disease
- Cardiothoracic Sx
- Intracardiac mass
- Congenital heart disease

NON-CARDIAC
- Pulmonary
- Neurologic
- Toxic/Metabolic
Classification

• AF can occur in normal heart & with presence of organic disease

ACC/AHA/ESC Classification:
1. Paroxysmal AF (self-terminating) generally lasts < 7 d (usually < 24 h) & may be recurrent
2. Persistent AF (fails to self-terminate) lasts for > 7 d, may also be paroxysmal if it recurs after reversion (recurrent if > 2 episodes)
3. Permanent AF lasts for more than 1 y & cardioversion has not been attempted or has failed
4. “Lone” AF describes paroxysmal, persistent, or permanent AF in individuals w/o structural heart disease

* Classification applies to episodes of AF lasting > 30 s & are unrelated to reversible cause
Diagnosis
Diagnosis

Hx:
• Define symptoms associated with AF, the clinical “pattern”, onset date, freq/duration of AF, any precipitating causes & modes of termination
• Presence of HD or potential reversible causes (hyperthyroidism)

P/E:
• Variation in intensity of S1
• Absent a waves on JVP
• Irregularly irregular ventricular rhythm (with fast rates, a pulse deficit may appear - apical rate > radial rate (pulse deficit)
Investigations

- ECG (LVH, preexcitation, BBB, old MI, P, RR, QRS, QT)
- CXR (useful to assess lungs, vasculature, cardiac outline)
- Echo (chamber size, function, VHD, LVH, pressures)
- Assess for hyperthyroidism (measure TSH, free T4)
MANAGEMENT

Atrial fibrillation
General treatment issues

- Rhythm control
  - Reversion to NSR & maintenance of NSR
- Rate control
  - Meds to control the VR in chronic AF
- Choosing b/w rhythm & rate control
- Prevention of systemic embolization
Rhythm control

• Reversion to NSR
  - Synchronized external DC cardioversion
  - Pharmacologic cardioversion

• Timing of cardioversion
  - In patients with AF more than 48 h or unknown duration, should be delayed until proper anticoagulation 3-4 w or TEE has excluded A. thrombi
Rhythm control

- DC cardioversion in
  - Hemodynamically unstable in the setting of short duration AF
  - Overall success rate of 75-97%
  - Related inversely to duration of AF and Left atrial size.
Rhythm control

• Medical cardioversion
  - Class 1A:
    • Quinidine, procainamide, disopyramide
  - Class 1C: flecainide, propafenone
  - Class 3: Amiodarone, sotalol, ibutilide

• Comparison of class 1A or 1C versus class 3 shown no difference in effect.
• Choice depends on duration of AF
Rhythm control

• Maintenance of NSR
  - Only 20-30% with successful CV maintain NSR without chronic antiarrhythmic therapy.
  - More in pts with AF < 1 year, no LAE, and a reversible cause of AF

• Class 1A, 1C, 3 useful for maintenance
  - Flacainide: in no or minimal heart disease
  - Amiodarone: reduced LVEF or HF
  - Sotalol: coronary heart dis.
Rate control

- Slowing of Av nodal conduction by:
  - B-blocker, CC blocker,
  - Digoxin: in CHF or hypotension
  - Amidarone: in those who are not CV to NSR
Rate vs Rhythm control

- Major clinical trials (AFFIRM & RACE) reached two major conclusions:
  - Significant trend toward a lower incidence of the primary end point with rate control.
  - Embolic events occur with equal frequency regardless of rate or rhythm control
    - Occur most often after warfarin stopped or INR is subtherapeutic.
Rate vs rhythm control

• Thus, both rhythm & rate control are acceptable approaches and both require anticoagulation.

• The trials provide supportive evidence for the rate control option in most patients.
Recent Onset AF

- **Spontaneous reversion:**
  - The incidence is related to duration of arrhythmia (<72h).

- **Urgent cardioversion:**
  - Active ischemia
  - Significant hypotension
  - Severe manifestation of CHF
  - Pre-excitation syndrome

- **Restoration of NSR takes precedence over TE risk.**
Rate and rhythm control

- **Rate control**
  - With β-blocker, CC blocker or Digoxin
- **Elective CV (electrical, medical)**
  - Depends on duration of AF and presence of reversible etiologic factors.
- **Immediate CV**
  - Low risk for systemic embolization
  - CV can be done after systemic Heparinization.
  - Aspirin for the first AF with spontaneous conversion and warfarin for 4 w to all other pts.
Cardioembolic stroke

• Assoc’d morbidity and mortality largely d/t thromboembolism (esp. stroke)

• Can occur in both paroxysmal and chronic AF

<table>
<thead>
<tr>
<th>Event</th>
<th>Avg Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Annual stroke rate not on warfarin</td>
<td>5%</td>
</tr>
<tr>
<td>Risk of death from stroke</td>
<td>25%</td>
</tr>
<tr>
<td>Risk of permanent disability from stroke</td>
<td>50%</td>
</tr>
</tbody>
</table>
Factors promoting thrombosis:

Virchow's Triad

- stasis
- Endothelial injury
- Hypercoagulability
Mechanisms of thrombogenesis

- LV dysfunction
- HTN
- Structural heart disease
- Cardioversion
- hypercoagulability
Prevalence of TE stroke in AF

- AF ↑ stroke risk 5X
  - AR of 4.5%/yr
  - Precise annual risk ranges from <1% to >12% depending on ECHO and clinical RF

- ↑ strikingly w/age
  - Nearly 1/2 of AF assoc’d strokes in pt >75yo

- 2° stroke incidence = 10-12% despite ASA
**Risk factors**

- **Independent predictors of ischemic stroke in non-valve atrial fibrillation**
  - **Consistent predictors**
    - Old age
    - HTN
    - Previous stroke or TIA
    - LV dysfunction* (on ECHO or recent clinical CHF)
  - **Inconsistent predictors**
    - DM
    - SBP >160 mm Hg
    - Women, especially >75yo
    - HRT
    - CAD

- **Factors which ↓ risk of stroke**
  - Moderate to severe mitral regurgitation
  - Regular alcohol use (>14 drinks in two weeks)
# Risk stratification

## Table 18. Published Risk-Stratification Schemes for Primary Prevention of Thromboembolism in Patients With Nonvalvular Atrial Fibrillation

<table>
<thead>
<tr>
<th>Source</th>
<th>High Risk</th>
<th>Intermediate Risk</th>
<th>Low Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrial Fibrillation Investigators (185)†</td>
<td>Age greater than or equal to 65 years</td>
<td></td>
<td>Age less than 65 years</td>
</tr>
<tr>
<td></td>
<td>History of hypertension</td>
<td></td>
<td>No high-risk features</td>
</tr>
<tr>
<td></td>
<td>Coronary artery disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Diabetes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>American College of Chest Physicians (473)</td>
<td>Age greater than 75 years</td>
<td></td>
<td>Age 65–75 years</td>
</tr>
<tr>
<td></td>
<td>History of hypertension</td>
<td></td>
<td>Diabetes</td>
</tr>
<tr>
<td></td>
<td>Left ventricular dysfunction‡</td>
<td></td>
<td>Coronary artery disease</td>
</tr>
<tr>
<td></td>
<td>More than 1 intermediate risk factor</td>
<td></td>
<td>Thyrotoxicosis</td>
</tr>
<tr>
<td>Stroke Prevention in Atrial Fibrillation (468)</td>
<td>Women greater than 75 years</td>
<td></td>
<td>History of hypertension</td>
</tr>
<tr>
<td></td>
<td>Systolic BP greater than 160 mm Hg</td>
<td></td>
<td>No high-risk features</td>
</tr>
<tr>
<td></td>
<td>Left ventricular dysfunction†</td>
<td></td>
<td>No history of hypertension</td>
</tr>
</tbody>
</table>

BF indicates blood pressure. Patients are classified on the basis of the presence or absence of any risk factor. Adapted with permission from Am J Med, Vol. 109, Pearce et al., Assessment of these schemes for stratifying stroke risk in patients with nonvalvular atrial fibrillation, pp. 45-51, © 2000, with permission, from Excerpta Medica, Inc.

*Patients with AF and prior thromboembolism are at high risk of stroke, and anticoagulation is indicated for secondary prevention in such cases.

†Did not distinguish high-risk from intermediate-risk patients.

‡Left ventricular dysfunction refers to moderate to severe wall motion abnormality assessed globally by 2-dimensional echocardiography, reduced ejection fraction, fractional shortening less than 0.25 by M-mode echocardiography, or clinical heart failure.
Echo

- TEE - the most sensitive and specific imaging technique for detection of LA and LAA thrombus.

- ECHO findings assoc’d w/TE in high-risk pts:
  - impaired LV fn on transthoracic echocardiography
  - thrombus
  - reduced velocity of blood flow in the LAA
  - complex atheromatous plaque in thoracic aorta on TEE.

- Other ECHO signs, such as LA diameter and fibrocalcific endocardial abnormalities, n invariably assoc’d with TE and may interact with other factors.

- Role of TEE:
  - No role for routine TEE in pts with effective anticoagulation >3wks prior to CV.
  - However, consider TEE prior to CV for those at increased risk of LA thrombi
    - Rheumatic mitral valve ds, recent TE, poor LV fn

- ACUTE trial:
  - Compared TEE guided vs conventional approach
    - No difference b/w two groups in incidence of stroke, TIA, embolic events w/n 8wks of CV.
Anticoagulation

- Well established that antithrombotic therapy confers thromboprophylaxis in AF
- **Warfarin** (adjusted dose)
  - Recent meta-analysis - ↓ stroke by 60%
    - ARR of 3%/yr for 1° prevention (NNT 33)
    - ARR of 8%/yr for 2° prevention (NNT 13)

Anticoagulation

- Aspirin
  - ↓ stroke by 20%
  - ARR 1.5%/yr for 1° and 2.5%/yr for 2° prevention

Anticoagulation

- Relative to ASA, warfarin ↓ risk by ~40%

- Overall, warfarin (dosed to maintain INR 2-3) is significantly more effective than ASA in pts at high risk of stroke

- Benefit of ASA more for non-cardioembolic strokes

- no role for mini-dose warfarin (1mg/d regardless of INR), alone or in combination with anti-platelet drugs or ASA

Anticoagulation

- Full dose anticoagulation carries risk of major bleeding, incl. ICH

- Meta-analysis of initial 5 primary prevention trials suggests risk of hemorrhagic stroke only marginally ↑ from 0.1% to 0.3%/yr

- Higher rates of major hemorrhage seen in elderly pts and those w/higher intensity anticoagulation
Major Bleeding During Antithrombotic Therapy in Atrial Fibrillation Trials*†

<table>
<thead>
<tr>
<th>Study</th>
<th>Rate, percent/year</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All¥</td>
</tr>
<tr>
<td></td>
<td>Intracranial</td>
</tr>
<tr>
<td></td>
<td>Aspirin</td>
</tr>
<tr>
<td></td>
<td>Placebo</td>
</tr>
<tr>
<td>Atrial Fibrillation, Aspirin, and Anticoagulation (AFASAK 1)</td>
<td>0.5</td>
</tr>
<tr>
<td></td>
<td>0.2</td>
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<td></td>
<td>0.2</td>
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<td></td>
<td>0</td>
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<tr>
<td>Boston Area Anticoagulation Trial for Atrial Fibrillation (BAATAF)</td>
<td>1.6</td>
</tr>
<tr>
<td></td>
<td>0.2</td>
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<tr>
<td></td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>1.6‡</td>
</tr>
<tr>
<td>Stroke Prevention in Atrial Fibrillation (SPAF I)</td>
<td>1.5</td>
</tr>
<tr>
<td></td>
<td>0.8</td>
</tr>
<tr>
<td></td>
<td>1.9</td>
</tr>
<tr>
<td></td>
<td>1.8</td>
</tr>
<tr>
<td>Canadian Atrial Fibrillation Anticoagulation</td>
<td>2.1</td>
</tr>
<tr>
<td></td>
<td>0.4</td>
</tr>
<tr>
<td></td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>0.5</td>
</tr>
<tr>
<td>Stroke Prevention in Nonrheumatic Atrial Fibrillation</td>
<td>1.4</td>
</tr>
<tr>
<td></td>
<td>0.2</td>
</tr>
<tr>
<td></td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>1.1</td>
</tr>
<tr>
<td>European Atrial Fibrillation Trial</td>
<td>2.6</td>
</tr>
<tr>
<td></td>
<td>0</td>
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<tr>
<td></td>
<td>0.9</td>
</tr>
<tr>
<td></td>
<td>-</td>
</tr>
<tr>
<td>SPAF II</td>
<td></td>
</tr>
<tr>
<td>≤75 y</td>
<td>1.6</td>
</tr>
<tr>
<td></td>
<td>0.5</td>
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<tr>
<td></td>
<td>0.9</td>
</tr>
<tr>
<td></td>
<td>-</td>
</tr>
<tr>
<td>&gt;75 y</td>
<td>4.2</td>
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<tr>
<td></td>
<td>1.8</td>
</tr>
<tr>
<td></td>
<td>1.6</td>
</tr>
<tr>
<td></td>
<td>-</td>
</tr>
<tr>
<td>SPAF III</td>
<td>2.1</td>
</tr>
<tr>
<td></td>
<td>0.5</td>
</tr>
<tr>
<td></td>
<td>2.4§</td>
</tr>
<tr>
<td></td>
<td>-</td>
</tr>
<tr>
<td>AFASAK 2</td>
<td>1.1</td>
</tr>
<tr>
<td></td>
<td>0.6</td>
</tr>
<tr>
<td></td>
<td>1.4</td>
</tr>
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<td>-</td>
</tr>
</tbody>
</table>

* Major bleeding was defined as bleeding requiring transfusion of blood or leading to permanent disability or to death. The rate was calculated as number of events per year of treatment.

† Includes intracranial bleeding events.

‡ Aspirin voluntary.

§ Aspirin plus warfarin international normalized ratio of the prothrombin time ratio, 1.2–1.5.

† Reproduced with permission from Gullo, AL, Koefoed, BG, Peterson, P, Arch Intern Med 1999; 159:1322.
**Anticoagulation**

- 2 major settings:
  1) During cardioversion to NSR

  **ACCP/ACC/AHA guidelines:**
  - AF>48h → oral anticoagulation (warfarin) X 3-4 wks prior to and >4wks following cardioversion
  - Also recommended in pt w/
    - Valvular ds, LV dysfn, recent TE, and unknown duration
  - Recommended target INR=2.5

  2) Chronic or paroxysmal AF
# American College of Chest Physicians Recommendations for Anticoagulation in Chronic Nonvalvular Atrial Fibrillation

<table>
<thead>
<tr>
<th>Risk group/status</th>
<th>Annual risk, percent</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>1</td>
<td>Aspirin (325 mg/day)</td>
</tr>
<tr>
<td>One moderate risk factor</td>
<td>1-4</td>
<td>Aspirin or warfarin (target INR 2.5; range 2.0-3.0)</td>
</tr>
<tr>
<td>High or &gt; 1 moderate risk factor</td>
<td>8-12</td>
<td>Warfarin (target INR 2.5; range 2.0-3.0)</td>
</tr>
</tbody>
</table>

* In patients considered for long-term oral anticoagulation, the recommendations for warfarin therapy only if there are no contraindications; the combined use of aspirin and low-fixed-dose warfarin is not recommended. Aspirin therapy if adjusted-dose warfarin contraindicated or declined by patient and there are no contraindications for aspirin.

Echocardiography not required for routine assessment but can refine clinical risk stratification

◊ Patients are classified into the following risk groups:

- **High risk** — patients with risk factors including prior transient ischemic attack, systematic embolus or stroke; history of hypertension; clinical evidence of valve disease (rheumatic mitral valve disease or prosthetic heart valve); heart failure or impaired left ventricular function on echocardiography; thyroid disease; or age ≥75.

- **Moderate risk** — patients age 65 to 75 years, diabetes, or coronary heart disease with preserved left ventricular function

- **Low risk** — patient under age 65 with no clinical or echocardiographic evidence of cardiovascular disease (no history of embolism, hypertension, diabetes, or other clinical risk factors)


Data from Lip, GYP, Lancet 1999; 353:4.
ACCP guidelines for antithrombotic Tx in non-valvar AF

- **Assess risk, and reassess regularly**
- **High risk (annual stroke risk = 8-12%)**
  - previous TIA or stroke
  - 75yo w/DM or HTN
  - clinical evidence of valve disease, heart failure, thyroid disease, and poor LV fn on ECHO*
  - **Tx:** Warfarin (target INR 2-3) if no contraindications.

- **Moderate risk (annual stroke risk=4%)**
  - <65yo w/clinical RF: DM, HTN, PVD, IHD
  - >65yo not in high risk group
  - **Tx:** Either Warfarin (INR 2-3) or ASA 75-300 mg daily. (insufficient clear cut evidence, ∴ Tx may be decided on individual cases).

- **Low risk (annual risk=1%)**
  - <65yp w/o hx embolism, HTN, DM or other clinical RF
  - **Tx:** Give ASA 75-300 mg daily

- *Echo not needed for routine risk assessment but refines clinical risk stratification in case of moderate or severe left ventricular dysfunction and valve disease. A large atrium per se is not an independent risk factor on multivariate analysis*
Table 19. Risk-Based Approach to Antithrombotic Therapy in Patients With Atrial Fibrillation

<table>
<thead>
<tr>
<th>Patient Features</th>
<th>Antithrombotic Therapy</th>
<th>Grade of Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age less than 60 years, no heart disease (lone AF)</td>
<td>Aspirin (325 mg per day) or no therapy</td>
<td>I</td>
</tr>
<tr>
<td>Age less than 60 years, heart disease but no risk factors*</td>
<td>Aspirin (325 mg per day)</td>
<td>I</td>
</tr>
<tr>
<td>Age greater than or equal to 60 years, no risk factors*</td>
<td>Aspirin (325 mg per day)</td>
<td>I</td>
</tr>
<tr>
<td>Age greater than or equal to 60 years with diabetes mellitus or CAD</td>
<td>Oral anticoagulation (INR 2.0–3.0) or addition of aspirin, 81–162 mg per day is optional</td>
<td>IIb</td>
</tr>
<tr>
<td>Age greater than or equal to 75 years, especially women</td>
<td>Oral anticoagulation (INR ≈ 2.0)</td>
<td>I</td>
</tr>
<tr>
<td>HF</td>
<td>Oral anticoagulation (INR 2.0–3.0)</td>
<td>I</td>
</tr>
<tr>
<td>LV ejection fraction less than or equal to 0.35, thyrotoxicosis, and hypertension</td>
<td>Oral anticoagulation (INR 2.5–3.5 or higher may be appropriate)</td>
<td>I</td>
</tr>
<tr>
<td>Rheumatic heart disease (mitral stenosis)</td>
<td>Oral anticoagulation</td>
<td>I</td>
</tr>
<tr>
<td>Prosthetic heart valves</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prior thromboembolism</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Persistent atrial thrombus on TEE</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

AF indicates atrial fibrillation; HF, heart failure; INR, international normalized ratio; LV, left ventricular; CAD, coronary artery disease; and TEE, transesophageal echocardiography.

*Risk factors for thromboembolism include HF, LV ejection fraction less than 0.35, and history of hypertension.

ACC/AHA/ESC recommendations
General Recommendations

(J Am Cardiol 2001;38:1231)

• **Class I**
  - Antithrombotic tx (warfarin or ASA) for all pts w/AF, except w/lone AF
  - Individualize treatment based on FR
  - Target INR = 2-3 in high risk pts unless contraindicated
  - ASA 325mg daily in low-risk pt or w/contraindications for anticoagulation
  - Warfarin for pts w/AF and valvular ds

• **Class II**
  - Target INR = 2 (1.6-2.5) in pts >75yo w/risk of bleed but w/o frank contraindications
  - Antithrombotic tx in atrial flutter managed as AF
  - Select antithrombotic tx by same criteria irrespective of pattern of AF
Long term outcome

- AF is a risk factor for increased mortality in otherwise healthy older people and those with coexisting CVD.

- Increased risk was more pronounced in coexistence of CVD worsens the prognosis, doubling the CV mortality.
Impression/Plan:

- 25 y o female with previous hx of cardiomyopathy & PAF presented with 8 hr hx of aphasia & left-sided hemiparesis, probable Dx of thromboembolic event.

- She was admitted & monitored. She remained stable & over the subsequent 12 hrs, her symptoms resolved completely with the exception of a mild residual numbness in her left fingertips.

- Discussed importance of ASA Rx & discussed use of an alternate anti-platelet drug, combo, or warfarin.
Bottom Line
Bottom Line

(EXIT) AF = erratic atrial rhythm → irregular ventricular response

(EXIT) ↓ CO → Atrial Thrombus formation → Embolization

(EXIT) Complications = Stroke, HF, Coronary thrombus, Embolism, Death

(EXIT) Management = Rate, Rhythm, Cardiovert, Anticoagulate, Ablate