Arrhythmias (I)
Supraventricular Tachycardias

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Disclosures

• None
Short R-P Tachycardia

REGULAR with 1:1 P/R relationship

- RP > PR
- RP < PR
- RP = PR

• AVRT
• AVNRT
• AT

Re-entry

- Requirements
  - Two pathways
  - Unidirectional block
  - Area of slow conduction

- AVNRT
  - “dual AV node physiology”
    - “fast pathway” – conducts rapidly, long refractory period
    - “slow pathway” – conducts slowly, short refractory period
18 year old male with palpitations
Baseline EKG

Slide courtesy of William Stevenson
WPW characteristics:

- P-R interval < 120 ms
- QRS duration > 100 ms
- Normal P wave vector
- Presence of a delta wave
  - can be (+) or (-)

Pathways can be “Concealed”
Accessory Pathways

• Abnormal pathway of conduction which bypasses the insulation of AV groove
• Nondecremental in 99%
  – Antegrade bidirectional (manifest) – (84%)
  – Retrograde - (concealed) ~10%
  – Antegrade only – (<5%)
• Atrial arrhythmias –
  – AF in 15-30% of WPW
  – Atrial Flutter 5%
  – AVRT (ortho, anti)
• Management –
  – Catheter ablation if high risk
  – Catheter ablation if symptomatic
Accessory Pathways, Atrium, Ventricle
+ fast sodium current - Class I drugs
  procainamide, flecainide, propafenone
+ K current - Class III drugs
  amiodarone, sotalol
+/- sympathetic stimulation
  beta-blockers

AV node
+ acetylcholine sensitive
  vagotonic maneuvers
+ calcium sensitive
  verapamil, diltiazem
+ sympathetic stimulation
  beta-blockers

Slide courtesy of William Stevenson

72 year old man with palpitations

Slide courtesy of William Stevenson
...after i.v. adenosine

Slide courtesy of William Stevenson
Typical Atrial Flutter

- Counterclockwise right atrial circuit
- Cavo-tricuspid isthmus dependent

Atrial Flutter

- Challenging to rate control
  - Typically requires high doses of nodal agents
    - Risk of severe bradycardia at cardioversion
  - 3:1 or higher degree block (off meds) generally means the AVN is diseased
  - When rate control IS achieved, the level of drugs required can result in severe bradycardia when cardioversion occurs

- Ablation can be first-line management
  - Better long-term freedom from AFL than with drugs
  - Decreases risk of subsequent AF (which otherwise occurs in over 50%)
What's the rhythm?

Same patient…
Automaticity can be provoked

Automaticity can be protective
Atrial Fibrillation

- Chaotic atrial electrical activity with high-rate fibrillatory potentials
- Irregularly irregular ventricular complexes

Rate Control Options

- Beta blockers
  - Most effective class in AFFIRM
  - Negative inotropic effects
- Calcium channel blockers
  - Only drugs shown to improve QOL & exercise tolerance
  - Negative inotropic effects
- Digoxin
  - Should not be the sole agent in paroxysmal AF
- (Pacemaker)
- (AV Junction ablation)

Writing Group, 2011 ACCF/AHA/HRS Focused Update on AF, Circulation 2011.
AV Node Ablation

• Improved symptom control and quality of life, but no difference in functional capacity\(^1\)
  – Low (<1%) morbidity/mortality
  • Historically, sudden death in ~2% of patients post-procedure\(^2\)
    – Repolarization abnormalities exacerbated by sudden change in HR
    – PPM failure without escape
    – Unrelated ventricular arrhythmias
  – Improved mortality in patients with CHF and AF receiving CRT\(^3\)


Pop Quiz

A 75 year old man presents with atrial fibrillation with ventricular rate ~110. After initiation of flecainide, atrial fibrillation organizes into atrial flutter at an atrial rate of ~260. What is the predicted change in the ventricular rate given the reduction in atrial rate?

a) Ventricular rate will increase
b) Ventricular rate will decrease
c) Ventricular rate will not change
d) Ventricular rate is so variable in AF that it’s a meaningless distinction
Pop Quiz

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AV node

“decremental conduction”

• At increasing atrial rates, conduction in the AV node will slow
  – the node protects the ventricles from being driven too fast
  – “concealed conduction” that blocks within the node renders it transiently refractory
• the vast majority of bypass tracts lack this property – they’re “non-decremental”
Atrial Fibrillation in WPW

NO nodal agents!!
Drugs of choice:
Procainamide
Ibutilide
Consider DCCV

The other end of the spectrum
Post DCCV

Syncope from AF?

• Rare…
  – Patients who are preload dependent
    • HCM
    • Aortic stenosis
  – Patients with an accessory pathway
  – Patients with high-grade AV block
  – Patients with sinus node dysfunction (offset pauses)
Rhythm Control

• On a population level, large-scale clinical trials suggest that a “rhythm control” strategy will NOT…
  – Improve quality of life
  – Decrease CHF exacerbations/hospitalizations
  – Decrease stroke risk

• Drugs slow conduction, alter refractoriness, and/or decrease automaticity of cardiac tissue
  – Effects may be restricted to specific tissue (e.g., Class IB)
  – Drugs altering conduction &/or refractory periods are also proarrhythmic
  – Efficacy is on the order of 30-60%

Sodium channel blockers

• Class IA (Procainamide) –
  – historical drug of choice in patients with WPW

• Class IC (Flecainide, Propafenone)
  – Slowed conduction can paradoxically increase rates – require concomitant nodal agent
  – Proarrhythmic (CAST trial, increased mortality post-MI) - We do not use these drugs in patients with CHF, significant LVH, or CAD
Potassium Channel Blockers (Class III)

- Sotalol, Dofetilide – prolong QTc and can cause torsades
- Amiodarone – most effective, most toxic
- Dronedarone – controversial...

Rhythm Control Decision Tree

No ♥ Dz → Hypertension → LVH ≥ 14mm

No… → Dronedarone, Flecaïnide, Propafenone, Sotalol

Yes… → Amiodarone

CAD → Dofetilide, Dronedarone, Sotalol

CHF → Amiodarone, Dofetilide

PVI
AF Ablation

- Catheter Pulmonary Vein Isolation
  - No role in the acute management of AF
  - Elective procedure after failure of medical therapy
  - One yr success rates ~60-80% (~2 mo “blanking period”) 
    - Success rates improve with multiple ablations
    - “Reconnections” occur
    - atypical flutters (re-entrant around RFA lines)
  - Risks: Stroke ~1%, Tamponade ~1%, Death 0.1-0.7%
- Surgical Ablation
  - 3rd line approach currently (after RFA)
  - Technical advances continue

Strokes and AF

- Multifactorial – atrial stasis, hypercoagulability (?local vs systemic), endothelial dysfunction
- Risk increases with Age
  - associated w/ LAE, reduced LAA flow velocity, and “smoke”
- 48 hours – not as magic as we’d like!
- “Atrial stunning” – observed after ANY type of cardioversion (spontaneous, drug, DCCV)
  - Rapid recovery in days, but can last up to 4 weeks
  - Increased duration AF predicts increased duration stunning

Anticoagulation

- Consideration is INDEPENDENT of “Rhythm Control”
  - Ablation is not (yet) curative
  - Drugs generally will fail given enough time
- Risk is a continuum
  - “Moderate” risk patients benefit from therapeutic anticoagulation
  - Rare major risk factors don’t appear in the scoring (e.g., mitral stenosis, prosthetic valve)
- “Paroxysmal” AF is not lower risk
- Given the higher risk peri-cardioversion, anticoagulation peri-cardioversion is necessary independent of risk score

CHADS$_2$

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**CHA\textsubscript{2}DS\textsubscript{2}-VASc**

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<th>Description</th>
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<td>S\textsubscript{2}</td>
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<td>A</td>
<td>Age 65-74 years</td>
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**Novel Anticoagulants**

- Growing list of options without consensus on which drug for who…
  - Dabigatran –
    - controversy over low-dose, higher risk in elderly, increase in MI, GI side effects
    - Requires renal dosing
  - Rivaroxaban –
    - Noninferior, higher risk population in study
    - Increased events at study end (no bridge, blinding maintained)
  - Apixaban –
    - Not yet approved
Universal issues with new agents

- Insurance / cost issues
- Lack of reversal agent
- Truly long-term follow-up
- Periprocedural management
- How to document/ensure compliance
  - DCCV

A 74 year old woman with hypertension, prior TIA, and persistent atrial fibrillation undergoes catheter ablation of AF (PVI).

Which of the following is correct?

1. Anticoagulation must be held periprocedurally
2. Anticoagulation is no longer required (ablation is curative)
3. Anticoagulation should be given for 2 months postprocedure and then stopped
4. Anticoagulation is only needed if a rhythm control agent is required post-ablation
5. Anticoagulation should be continued indefinitely
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