Common Supraventricular Tachycardias (SVT)

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Disclosures

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- ATTENDEES!
Objectives

- Be able to recognize the presentation, mechanism, and management of common supraventricular tachycardias.
Incidence of Common SVT

A. Incidence of All Common Heart Rhythm Abnormalities

B. Incidence of All Heart Rhythm Abnormalities During EP Studies
What do SVT’s look like?

A. Sinus Rhythm
B. Supraventricular Tachycardia (SVT)
C. Atrial Fibrillation
D. Ventricular Tachycardia (VT)
E. Atrial Flutter
What are the symptoms of SVT’s?

- Palpitations are the sensation of an irregular, fast, uncomfortable, or strong heartbeat.
  - Over 600,000 patients present to Emergency Departments each year in the United States (Probst et al. 2014) because of palpitations.
  - One in four of these patients will be admitted to the hospital for further care and roughly a third of patients will be diagnosed with a heart condition.
Cardiac Conduction System

SA Node

AV Node

Left Bundle Branch

Right Bundle
AV Node Reentrant Tachycardia (AVNRT)

• Most common SVT that we induce during EP studies.

• 15-30% of population has “dual AV-node physiology.”
  – Most day-to-day conduction is from “fast” AV node pathway.
  – Patients with “dual AV node physiology” may occasional use the “slow” AV node pathway.
ECG Findings with AVNRT

• Generally normal resting ECG
ECG Findings with AVNRT

• ECG in SVT usually narrow QRS with pseudo-
  r’ in V1.
Why does typical AVNRT have P wave buried at end of QRS?
Mechanism of AVNRT

- **Typical AVNRT**: down slow AVN pathway and retrograde fast AVN pathway.
- **Atypical AVNRT (shown)**: down fast AVN pathway and retrograde slow AVN pathway.
Management of AVNRT

AVNRT

Symptomatic

Reassess symptoms during follow-up

Clinical follow-up without treatment (Class IIa)

No or minimally symptomatic

Ablation candidate, pt prefers ablation

Self-administration of beta blockers, diltiazem, or verapamil in pts with infrequent, well-tolerated episodes of AVNRT (Class IIb)

Yes

Ablation

No

Slow-pathway catheter ablation (Class I)

Beta blockers, diltiazem, or verapamil (Class I)

If ineffective, consider ablation

If ineffective

Flecainide or propafenone (in the absence of SHD) (Class IIIa)

Amiodarone, digoxin, dofetilide, or sotalol (Class IIb)

If ineffective, consider ablation
Mechanism of Atrial Tachycardia

- Focus often in the lateral right atrium overdrive suppresses the sinus rhythm and dictates heart rate.
- Adenosine generally does not terminate but may cause AV block so you can identify atrial tachycardia morphology.
ECG of Atrial Tachycardia

Figure 2. Anatomic distribution of tachycardia foci and tachycardia P waves at the atrioventricular valvular annuli. AV = atrioventricular; HBE = His bundle electrogram; MV = mitral valve; other abbreviations as in Figure 1.

- Taken from Kistler et al, “P wave morphology in focal atrial tachycardia,” JACC, V. 48, No. 5 (2006)
What is an accessory pathway?

- The heart’s cardiomyocytes develop fibrous tissue between the atria and ventricle during the gestational stages of the seventh and twelfth week. The normal process of closure between the atria and ventricle does not occur and these accessory fibers allow conduction to occur between the two, outside of the normal pathway of the AVN.

- In normal conduction through the AVN there is a delay; however, there is no such delay in the conduction system of a patient with a conducting AP, in which the conduction through the AP can be anterograde, retrograde, or both.
Mechanism of Atrioventricular Reentrant Tachycardia (AVRT)

- **Orthodromic AVRT (shown):** down AV node and retrograde AP.
- **Antidromic AVRT:** down AP and retrograde AV node. This will be a wide complex (preexcited) tachycardia.
Ventricular Pacing: Eccentric Retrograde Conduction via Accessory Pathway

- Pacing the ventricle and earliest atrial activation distal coronary sinus.

Earliest activation in distal coronary sinus.
AVRT: Orthodromic versus Antidromic

**AP Localization:**
1. ± in I: Left lateral
2. QS in II: MCV left posteroseptal
3. ± in V1: Septal
4. Absent 1-3: R free wall (delta prior to completion of P wave)

A: Antidromic (down AP and up AVN) tachycardia, B: Orthodromic AVRT (down AV node and up L lat AP), C: SR preexcited.
Acute Treatment of Orthodromic AVRT

Orthodromic AVRT

Vagal maneuvers and/or IV adenosine (Class I)

If ineffective or not feasible

Hemodynamically stable

Pre-excitation on resting ECG

Yes

IV beta blockers, IV diltiazem, or IV verapamil (Class IIb)

If ineffective or not feasible

Synchronized Cardioversion* (Class I)

No

Synchronized cardioversion (Class I)

IV beta blockers, IV diltiazem, or IV verapamil (Class Ia)
Treatment of Orthodromic AVRT

Orthodromic AVRT

Pre-excitation on resting ECG

Yes

Ablation candidate, willing to undergo ablation

No

Ablation candidate, pt prefers ablation

Flecainide or propafenone (in the absence of SHD) (Class IIa)

Amiodarone, beta blockers, diltiazem, dofetilide, sotalol, or verapamil (Class IIb)

Catheter ablation (Class I)

Beta blockers, diltiazem, or verapamil (Class I)

Flecainide or propafenone (in the absence of SHD) (Class IIa)

Amiodarone, digoxin, dofetilide, or sotalol (Class IIb)

If ineffective, consider ablation

If ineffective, consider ablation
Mechanism of Atrial Flutter

- **Typical CTI atrial flutter**: atrial activity rotates counterclockwise around right atrium.
- **Atypical CTI atrial flutter (shown in animation)**: atrial activity rotates clockwise around

Cavotricuspid Isthmus: area between IVC and tricuspid valve that serves as “circuit” for atrial flutter.
Treatments for Atrial Flutter/Fibrillation

• Anticoagulate both fibrillation and flutter!
• Rate control and anticoagulation
• Rhythm control and anticoagulation
## Antiarrhythmics for SVT’s

<table>
<thead>
<tr>
<th>Vaughan-Williams Class</th>
<th>Medications</th>
<th>How it is used</th>
<th>Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (Sodium Channel Blockers)</td>
<td>Flecainide, Procainamide, Disopyramide, Quinidine</td>
<td>Atrial fibrillation, accessory pathways, ventricular arrhythmias</td>
<td>Could cause arrhythmias, dry mouth, decrease heart contractility, ECG abnormalities.</td>
</tr>
<tr>
<td>2 (Beta Blockers)</td>
<td>Metoprolol, carvedilol, propranolol, atenolol, bisoprolol</td>
<td>Heart attacks, coronary artery blockages, heart failure</td>
<td>Can make you feel lethargic or dizzy. May lower blood pressure and heart rate. Do not stop abruptly.</td>
</tr>
<tr>
<td>3 (Potassium Channel Blockers)</td>
<td>Amiodarone, sotalol, ibutilide, dofetilide, dronedarone</td>
<td>Atrial and ventricular arrhythmias</td>
<td>Could cause arrhythmias, fatigue, ECG abnormalities</td>
</tr>
<tr>
<td>4 (Calcium Channel Blockers)</td>
<td>Verapamil, diltiazem</td>
<td>Atrial arrhythmias</td>
<td>Constipation, lower extremity swelling.</td>
</tr>
</tbody>
</table>
Questions?
Afib Ablation Candidates

- Symptomatic Paroxysmal or Persistent Atrial Fibrillation
- Failure of Class IC or Class III agent
- Intolerance to Medical Therapy, Refusal of Medical Therapy
- Other Considerations:
  - Young patients with paroxysmal atrial fibrillation, in whom decades-long drug therapy is undesirable
  - Congestive Heart Failure due to tachycardia-induced cardiomyopathy, in whom drug choices are limited by the presence of CHF
- Limitations in Efficacy
  - Longstanding Persistent Atrial Fibrillation (>1 year)
  - Enlarged LA (>55 mm)
  - Age > 70 years
- Left atrial or Left atrial appendage thrombus is an absolute contraindication to atrial fibrillation ablation.

### Worldwide Experience with AF Ablation

**Table 3: Fatality Rates According to Type of Complication**

<table>
<thead>
<tr>
<th>Complication</th>
<th>Death/Overall Events</th>
<th>Rate (%)</th>
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</thead>
<tbody>
<tr>
<td>Tamponade</td>
<td>7/331</td>
<td>2.3</td>
</tr>
<tr>
<td>Atrioesophageal fistula</td>
<td>5/7</td>
<td>71.4</td>
</tr>
<tr>
<td>Massive pneumonia</td>
<td>2/2</td>
<td>100.0</td>
</tr>
</tbody>
</table>

- This is report from only **85 centers in the world**.
- **4.5% rate of major complications**.
- Carto-guided left atrial circumferential ablation (48%) and lasso-guided ostial electric disconnection (27%).
- Overall drug-free (with AAD) 18 month success rates were 75% (83%) and 63% (72%) for paroxysmal and persistent atrial fibrillation.