Management and Treatment of Supraventricular Tachycardias.

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No disclosures.

* Off label drug use discussed.
Outline

* What is SVT and types of SVT, focus on PSVT
* Mechanism
* Epidemiology and clinical presentation
* Diagnostic evaluation
* Treatment
Normal sinus rhythm

Intracardiac tracings show the normal intervals between:
- initiation of atrial depolarization $A$
- His bundle activation $H$
- ventricular depolarization $V$
- $AH + HV = PR$ interval
Supraventricular tachycardia

- Atrial Fibrillation (A Fib)
- Atrial Flutter (A Flutter)
- AV nodal reentry tachycardia (AVNRT)
- AV reentry tachycardia (AVRT) - the WPW(Wolff-Parkinson White) Syndrome
- Atrial tachycardia (AT)
- Inappropriate Sinus Tachycardia
- Paroxysmal Junctional reciprocating tachycardia (PJRT)
- Junctional ectopic tachycardia (JET)
- Multifocal atrial tachycardia (MAT)
Introduction to supraventricular arrhythmias

Supraventricular Arrhythmias

- Atrial Fibrillation
- Paroxysmal supraventricular tachycardias (PSVT)
  - AV nodal reentry tachycardia (AVNRT)
  - AV reentry tachycardia (AVRT)
    - WPW
    - AV reentry over concealed bypass tract
  - Atrial Tachycardia
- Atrial Flutter
- Other

Paroxysmal Supraventricular Tachycardia
SVT Mechanism

Two basic mechanisms

• 1. Abnormal impulse conduction (Reentry)

AVNRT Dual AV node physiology
- both fast and slow conduction pathways are present in the AV node
- rapidly conducting tissue has a long recovery time
  - fast boat, long wake
- slow-conducting pathway has a relatively short recovery time
  - slow boats can follow more closely
AVNRT Normal Sinus Rhythm

During sinus beats:
- Conduction occurs via fast pathway.
- Conduction via slow pathway is blocked.
AVNRT Sinus beat
• labeled $S_1$

Premature Atrial Contraction (PAC)
• labeled $S_2$
• blocked in fast pathway
• the slow pathway may permit reentry into the AV node
  - short recovery time
  - depolarizes both atria and ventricles
AVNRT: Initiation of tachycardia

Retrograde P-waves in leads I, II, V1-V3
AVRT: Wolff-Parkinson-White syndrome: Preexcitation

ECG requirements for diagnosis of WPW syndrome

• P-R interval < 120 ms
• Normal P wave vector (to exclude junctional rhythm)
• Presence of a delta wave
• QRS duration > 100 ms
Supraventricular tachycardia
• can be initiated by a closely coupled premature atrial complex (PAC)
• blocks in the accessory pathway
• but conducts through the AV node
• retrograde conduction via accessory pathway
• inverted P wave produced by retrograde conduction visible in the inferior ECG leads
Atrial Flutter: Typical counter-clockwise atrial flutter

On the ECG, note the saw-tooth shaped P wave, negative in leads II, III, and aVF, which indicates the retrograde conduction up the atrial septum, consistent with counter-clockwise flutter.
SVT Mechanism

Two basic mechanisms

1. Abnormal impulse conduction (Reentry)

2. Abnormal impulse formation.
   (Automaticity or triggered activity)

Atrial tachycardia: Ectopic foci of right and left atrial origin

Atrial tachycardia
• defined as a focal tachycardia originating in atrial muscle other than the sinus or AV nodes
• may have single or multiple foci, capable of autonomous depolarization at rapid rates

Note that on the ECG, the P-waves (arrows) are clearly discernible, and that the PR interval is normal.
Mechanism of PSVT

• **Reentry**- approx 90% of all SVT.
  
  Either reentry within the AVN (60%) or using an accessory bypass tract (30%). Almost all reentrant SVTs are started with a PAC.

• **Atrial focus**- approx 10%. **Automaticity** or **Triggered activity**
Epidemiology

- Incidence: 35 cases per 100,000 persons per year.
- Prevalence: 2.25 per 1000 (excluding atrial fibrillation, atrial flutter, and multifocal atrial tachycardia).
- Increases with age.
- Not usually associated with structural heart disease.
- More common in Females 2:1

Symptoms

• Palpitations: with or without a trigger.
• Feeling of heart pounding in the chest and neck.
• Anxiety, light-headedness, dyspnea and psychological stress is common.
• Syncope and chest pain are uncommon, but may indicate CAD or significant structural heart disease, especially in older patients.

Focused physical exam.
- Vitals, orthostatic BP
- Cardiac: Murmurs, Gallops and JVP, Cannon A waves
- Respiratory: Rales
- Endocrine: Thyroid.
Blood work

- Chemistry:
- CBC:
- TSH:
- BNP and Cardiac enzymes
Diagnostic Investigations

Resting 12 Lead EKG.

EKG during symptoms is usually diagnostic.

Echocardiogram- “may be helpful” and “should be considered”.

Diagnostic Investigations

Holter / Event monitoring:

in the absence of severe symptoms and there is no concern for A Fib in the setting of WPW.

Diagnostic EP study


Clinical history of palpitations
12-Lead ECG (sinus rhythm)

Pre-excitation

(exclude heart disease)

Yes

Suspect AVRT

No

Assess arrhythmia pattern by clinical history

History of syncope?

Yes

Sustained regular palpitations

Refer to arrhythmia specialist

No

Irregular palpitations

Suspect AF, MAT or atrial flutter with variable AV conduction

Event monitor and follow-up
Reasons to consider a specialist consultation.

- WPW EKG+ palpitations.
- WPW EKG and irregular palpitations.
- Severe symptoms: syncope / chest pain / severe dyspnea.
- Wide complex tachycardia of unknown origin.

Treatment of SVT

ED management

Stable or Unstable?

• Altered Mental Status
• Hypotension
• Chest Pain
• Acute SOB
ED Management

Stable narrow complex tachycardia

- Vagal maneuvers
- Adenosine

ED Management

Stable narrow complex tachycardia

- Vagal maneuvers
- Adenosine
- IV Metoprolol / Verapamil / Diltiazem

Stable wide complex tachycardia

If the diagnosis is certainly a SVT:
- Vagal maneuvers
- Adenosine
- IV Metoprolol / Verapamil / Diltiazem

If the diagnosis is unclear: treat as VT.
- IV amiodarone / IV lidocaine/ IV procainamide.

BRUGADA criteria

Figure 1. Twelve-lead surface ECG (25 mm/s) of an irregular wide QRS-complex tachycardia during atrial fibrillation in the presence of a rapidly conducting accessory pathway.

Adenosine, Betablockers and Calcium Channel blockers are contraindicated.

IV Ibutilide, Procainamide or Amiodarone are treatment of Choice.

Blank R et al. Circulation. 2007;115:e469-e471
Atrial tachycardia.

- Adenosine infusion and use of IV metoprolol and or Diltiazem/Verapamil will make the diagnosis of atrial tachycardia but in majority of cases will not terminate it.

- Rate or rhythm control can be attempted.

ED Management

Options include:

* Observation with avoidance of triggers and treatment of reversible causes.

* Medical therapy.

* EP study and ablation.
Long term management

- Clinical factors: Symptom severity, frequency, effect on QoL, response to medical therapy and patient preference.

- High risk features: Syncope or symptomatic WPW: electrophysiology study and ablation is preferable.

- High risk occupations, ablation is preferred.

Long term management

AVNRT

* Ablation: Class I indication.
AVNRT can be cured permanently with catheter ablation, using radio frequency to heat and destroy the cells in the slow pathway, creating a permanent line of block.
Success rates for slow pathway ablation are over 95% and rate of AV block requiring pacemaker implantation are less than 1%

ACC/AHA Guidelines 2003
Mitrani RD, JACC 1993
Medical therapy for AVNRT.

- Single drug success rates are 30-50%.

- Metoprolol, Diltiazem, Verapamil: Class I
- Flecainide, propafenone or sotalol: Class IIa
- Digoxin: IIb.
- Amiodarone: IIb.

ACC/AHA/HRS Guidelines for supraventricular arrhythmias, 2003
The incidence of sudden cardiac death in patients with the WPW syndrome has been estimated to range from 0.15% to 0.39% over 3- to 10-year follow-up.

“supports the concept of liberal indications for catheter ablation”.

ACC/AHA/HRS Guidelines for supraventricular arrhythmias, 2003
Long term treatment of AVRT

Catheter ablation for AVRT / WPW.

Class I: symptomatic SVT related to pathway

Class IIa: asymptomatic pathway.

ACC/AHA/HRS Guidelines for supraventricular arrhythmias, 2003
Radio frequency ablation of the accessory pathway is often indicated in patients with WPW who are at risk of sudden death due to atrial fibrillation with a rapid ventricular response via the bypass tract.

Note the disappearance of the preexcitation delta wave in the QRS with catheter ablation.
Catheter ablation for AVRT / WPW.

Acute success rate: > 95% and delayed recurrence rate of 5%.

Risk: Mortality rate 0% to 0.2%
Morbidity: 4.4% complication rate.

ACC/AHA/HRS Guidelines for supraventricular arrhythmias, 2003
Long term treatment of AVRT

Medical therapy for AVRT / WPW.

There are no controlled trials of drug prophylaxis involving patients with AVRT.

ACC/AHA/HRS Guidelines for supraventricular arrhythmias, 2003
Long term treatment of AVRT

Medical therapy for AVRT / WPW.

Metoprolol, Flecainide, propafenone, sotalol: Class IIa for well tolerated AVRT.

In the presence of WPW: Diltiazem, Verapamil and digoxin should not be used. (Class III).

ACC/AHA/HRS Guidelines for supraventricular arrhythmias, 2003
Symptomatic recurrent AT:
Ablation, Beta blockers and Ca Channel Blockers: Class I
Class IC and Class III antiarrhythmics: Class IIa

ACC/AHA/HRS Guidelines for supraventricular arrhythmias, 2003
Atrial tachycardia: Mapping and ablation of ectopic focus

The atrial focus is identified as the location showing earliest depolarization. The ablation catheter (ABL) has located a point in the right atrium with depolarization 35 ms before high right atrial activation (HRA). This is the point chosen for ablation.
Atrial Flutter: Catheter ablation

Ablation in the tricuspid isthmus creates a line of block that interrupts the flutter circuit. Subsequent pacing from the coronary sinus demonstrates bidirectional block along the line of ablation.
Thank You!

QUESTIONs?
Clinical series of radiofrequency catheter ablation of accessory pathways have been published with excellent overall results.\textsuperscript{38} Experienced electrophysiology laboratories routinely achieve success rates of 95 percent in the ablation of accessory pathways, with recurrence rates of less than 5 percent.\textsuperscript{39} With improved knowledge of atrio-ventricular nodal anatomy and the advent of cryotherapy ablation, the current rate of symptomatic heart block is 0.5 to 1 percent.\textsuperscript{40}
Mechanisms

Onset of SVT
Classification

- AV Node Dependent-
  Reentry- approx 90% of all SVT. Either reentry within the AVN (60%) or using an accessory bypass tract (30%).

- AV Node Independent-
  Atrial focus- approx 10%. Automaticity or Triggered activity.

- Thinking of SVT in this way can help with treatment.
MANAGEMENT

Acute Management:

**Goal** is to control the heart rate and prevent hemodynamic collapse.
Normal conduction.
AV Node Rentry Tachycardia

Colucci et al. AFP. October 2010.
AV Rentry Tachycardia

Colucci et al. AFP. October 2010.