**Introduction: Supraventricular Tachycardia**

Supraventricular tachycardia (SVT) is a generic term that refers to any abnormally rapid heart rhythm that originates above the bundle of His in the conduction system. It is characterized by a sudden onset and termination of a regular, narrow QRS complex tachycardia. The heart rate during SVT is typically between 150-250 beats per minute. SVT is caused by either abnormal automaticity, triggered activity, or reentry. The most common mechanisms of SVT are atrioventricular nodal reentrant tachycardia (AVNRT) and atrioventricular reentrant tachycardia (AVRT) involving an accessory pathway. Other less common forms include atrial tachycardia, sinus node reentrant tachycardia, and intra-atrial reentrant tachycardia.Both utilize a reentrant circuit that allows rapid activation of the atria and ventricles. AVNRT involves reentry within the AV node, while AVRT involves an accessory pathway that connects the atria and ventricles outside of the AV node. This chapter will focus specifically on AV nodal reentrant tachycardia.

**Clinical Presentation**

Patients with AVNRT typically present with sudden onset palpitations, often described as a "pounding" or "racing" sensation in the chest. Episodes most often start and stop abruptly, lasting from a few seconds to hours. Other associated symptoms may include dizziness, lightheadedness, dyspnea, chest pain, and anxiety. Syncope can occur but is less common than in AVRT. Some patients may be asymptomatic and the tachycardia is discovered incidentally on ECG.

AVNRT can occur at any age but is most prevalent in middle-aged adults, with a mean age of onset around 50 years. There is a strong female predominance, with 60-80% of cases occurring in women. Many patients have no structural heart disease, however AVNRT can also occur in patients with underlying cardiovascular conditions. Triggering factors include caffeine, alcohol, exercise, emotional stress, fever, electrolyte imbalances, and medications including digoxin, beta-agonists, and antiarrhythmics.

On physical exam during an acute episode, typical findings include a rapid but regular pulse rate, normal blood pressure, and no murmurs, rubs, or gallops. Jugular venous pressure may be elevated due to impaired ventricular filling time. Abrupt termination of the episode may result in a brief pause before resumption of normal sinus rhythm. Some patients may develop hypotension or other signs of hemodynamic instability during prolonged episodes.

**Pathophysology**

The pathogenesis of AVNRT involves the presence of dual atrioventricular (AV) nodal pathways with different electrophysiological properties. The AV node has an unusual structure, with parallel tracks of conducting tissue that allow various routes for impulses to travel from the atria to the ventricles. One pathway has a longer refractory period but faster antegrade conduction velocity (fast pathway). The other has a shorter refractory period but slower antegrade conduction (slow pathway).

During normal sinus rhythm, impulses are conducted antegrade (from atria to ventricles) primarily through the fast pathway due to its faster conduction velocity. Retrograde conduction (from ventricles back to atria) occurs through the slow pathway. AVNRT occurs when a premature impulse traveling down the slow pathway finds the fast pathway still refractory. Instead of colliding with refractory tissue, the impulse conducts retrograde up the fast pathway, setting up a reentrant circuit. This allows repetitive activation circulating within the AV node itself.

The reentrant circuit utilizes the slow pathway for antegrade conduction towards the ventricles, and the fast pathway for retrograde conduction back to the atria. This is known as "slow-fast" AVNRT, which accounts for 95% of cases. The opposite pattern, "fast-slow" AVNRT, is less common. In either case, the end result is ineffective pumping due to the ventricles being bombarded by impulses coming down from the AV node, leading to the symptoms of rapid heart rate, palpitations, and hemodynamic instability in some patients.

**Diagnostic Approach**

**ECG**

On 12-lead ECG during AVNRT, the rhythm is regular with normal QRS complexes and ventricle rates typically between 140-250 bpm. The most characteristic ECG finding is RP interval (time from QRS to P wave) usually less than half the R-R interval, although not always. Retrograde P waves are typically not visible, buried in the QRS complex. Other findings may include pseudo r prime (r') in V1, pseudo S waves in inferior leads, and pseudo Q waves in aVL. However, AVNRT ECG patterns have significant variability between patients and episodes.

Differential diagnoses to consider based on ECG include other SVTs like AVRT, atrial tachycardia, flutter or fibrillation. Adenosine can be diagnostic to help differentiate, as it will transiently block AV nodal conduction and terminate AVNRT. AVRT and atrial tachycardia will persist after adenosine.

**Ambulatory Monitoring**

Ambulatory ECG monitoring using Holter monitors or external loop recorders can help document tachycardia episodes. The advantage of ambulatory monitoring is ability to correlate timing of episodes with patient symptoms and activities. This can be useful in assessing frequency of recurrences, response to treatments, and triggering factors. Limitations include inconsistent patient compliance and short monitoring durations.

**Electrophysiology Study**

An electrophysiology study (EPS) involves catheter placement into the heart chambers to map electrical signals. Programmed electrical stimulation is used to induce AVNRT episodes in a controlled setting. EPS allows definitive diagnosis by directly assessing conduction through dual AV nodal pathways and mechanisms of tachycardia. It is also used to guide ablation therapy. However, EPS is an invasive procedure with some risks. It should be considered in patients when noninvasive methods fail to establish a clear diagnosis.

### ****Management - Overview****

The goals of managing AVNRT are to acutely terminate episodes and prevent recurrences. Acute treatment focuses on restoration of normal sinus rhythm, while long-term therapy aims to modify the arrhythmia substrate. Management options include:

* Vagal maneuvers
* Pharmacologic therapy
* Electrical cardioversion
* Catheter ablation

Vagal maneuvers and/or intravenous adenosine are recommended as first-line acute treatment in hemodynamically stable patients. Synchronized electrical cardioversion is indicated for those who are unstable. Long-term management options include pharmacotherapy with antiarrhythmic medications or catheter ablation. Catheter ablation is generally preferred over lifelong drug therapy given its high efficacy and low risk. However, antiarrhythmic medications may be considered in patients with infrequent recurrences or who are not candidates for ablation.

### ****Pharmacotherapy****

Various antiarrhythmic drugs can be used to help control AVNRT episodes, either for acute termination or chronic prevention. They work by modifying conduction or refractoriness in the reentrant circuit pathways.

#### Acute Treatment

Adenosine is the preferred agent for acute pharmacologic termination of AVNRT. It works by hyperpolarizing cells in the AV node, suppressing conduction through slow pathway cells. This interrupts the reentrant circuit and restores normal conduction. Adenosine is given as a rapid IV bolus, with onset of action within seconds and a very short half-life. It terminates AVNRT episodes in up to 90% of cases. Side effects are transient and include chest pain, flushing, dyspnea, and AV block.

* Adenosine
  + Dose: Dose: 6 mg rapid IV bolus followed by 12 mg x 2 if not successful
    - May need higher dose in patients ingesting signficant amount of caffeine
    - Caution in patients with heart transplant
    - Adenosine is often considered contraindicated in heart transplant patients due to parasympathetic denervation.
  + Administration
    - Stopcock method
    - Diluted method by McDowell et al
      * Rapid push of mixture of adenosine and 20-30 mL normal saline given as a rapid IV push
  + Side effects
    - Flushing
    - Feeling of doom
    - Temporary Asystole
    - Can cause severe bronchospasm in asthma patients.
* Calcium Channel Blockers
  + DIltiazem IV
    - 0.25 mg/kg IV bolusover 2 mins, may repeat with 0.35 mg/kg IV; follow with 5—15 mg/hour infusion
    - Side effects
      * Hypotension
    - Caution
      * Avoid in hypotension, heart failure, cardiomyopathy, and acute coronary syndromes
    - Convert SVT to normal sinus rhythm in 64% to 98% of patients
    - A Cochrane systematic review of 7 RCTs (622 patients) found similar rates of conversion to sinus rhythm with adenosine or calcium channel blockers (90% versus 93%) and no significant difference in hypotension.
  + Verapamil
    - 2.5—5 mg IV; may repeat up to total 20 mg
    - Side effects
      * Hypotension
    - Convert SVT to normal sinus rhythm in 64% to 98% of patients

β-Adrenergic Blockers

* Metoprolol
  + 2.5–5 mg over 2 min, up to 3 doses
  + Avoid in decompensated heart failure
* Esmolol
  + 500 μg/kg IV over 1 min bolus then 50–300 μg/kg per min
  + Short duration of action; avoid in decompensated heart failure

Choice of agent depends on factors like presence of structural heart disease, kidney function, and side effect profile. Most patients require combination therapy with two medications from different classes to adequately control recurrences. Frequent monitoring is recommended to assess efficacy and minimize potential toxicity.

### ****Tips for Board Exam Questions:****

* AVNRT is the most common cause of regular, narrow complex tachycardia
* It involves reentry within the AV node through dual pathways
* Sudden onset/offset of palpitations is typical
* Adenosine transiently blocks AV nodal conduction to terminate AVNRT
* Ablation of slow pathway is curative

### ****Key Guidelines and Evidence****

* 2015 ACC/AHA/HRS Guidelines for the Management of Adult Patients With Supraventricular Tachycardia recommend:
  + Vagal maneuvers and/or IV adenosine for initial management of stable AVNRT
  + Synchronized cardioversion if unstable
  + EPS for diagnosis if noninvasive monitoring unclear
  + Catheter ablation of slow pathway as preferred long-term therapy
  + Antiarrhythmic medications reasonable for certain patients

* Part 3: adult Advanced Cardiac Life Support: 2020 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care.
  + 2. Adenosine is recommended for acute treatment in patients with SVT at a regular rate.
    - COR 1, LOE B-R
  + 3. IV diltiazem or verapamil can be effective for acute treatment in patients with hemodynamically stable SVT at a regular rate.
    - COR 2a, LOE B-R
  + 4. IV β-adrenergic blockers are reasonable for acute treatment in patients with hemodynamically stable SVT at a regular rate.
    - COR 2a, LOE LD

**Key Stuides**

* Alabed S, Sabouni A, Providencia R, Atallah E, Qintar M, Chico TJ. Adenosine versus intravenous calcium channel antagonists for supraventricular tachycardia. Cochrane Database Syst Rev. 2017;10:CD005154.
  + Viewed randomised controlled trials (RCTs) that compare effects of adenosine versus CCAs in terminating SVT.
  + Moderate-quality evidence shows no differences in the number of people reverting to sinus rhythm who were treated with adenosine or CCA (89.7% vs 92.9%; OR 1.51, 95% confidence interval (CI) 0.85 to 2.68; participants = 622; studies = 7; I2 = 36%).

### ****Clinical Scenarios****

Scenario 1

A 63-year-old woman presents to the ED with sudden onset rapid palpitations that woke her from sleep. ECG shows a regular narrow complex tachycardia at 170 bpm. Vagal maneuvers do not terminate the episode but 6 mg IV adenosine converts the rhythm to normal sinus. What is the likely diagnosis and what long-term management options would be recommended?

* This clinical presentation is classic for AVNRT - sudden onset, rapid regular palpitations terminated by adenosine. Long-term options would be catheter ablation of the slow pathway or antiarrhythmic medications if ablation is deferred.

### ****Summary****

* AV nodal reentrant tachycardia is the most common type of paroxysmal supraventricular tachycardia
* It involves reentry through dual AV nodal pathways with different electrophysiologic properties
* Patients present with sudden onset/offset palpitations, normal QRS complexes, heart rates 140-250 bpm
* Adenosine terminates AVNRT by transiently blocking AV nodal conduction
* Definitive treatment is catheter ablation of the slow pathway within the AV node
* Antiarrhythmic medications like beta blockers and non-DHP CCBs agents can also be used for rhythm control
* Acute management focuses on terminating episodes, while long-term therapy aims to prevent recurrences

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