Supraventricular Tachycardia: Returning to a Regular *Rhythm*

Western

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Norah's Butterflies

- Norah, 38, is a business executive.
- She presents with recurring palpitations which she explains feel like her heart jumping out of her chest with a feeling of fullness in her neck.
- Norah gets mild shortness of breath and feels lightheaded with the palpitations, but has no chest pain, nor does she lose consciousness.
- She has had four episodes in the last five years that lasted up to 20 minutes each.
- A recent fifth episode lasted two hours. A visit to the ER showed a supraventricular tachycardia at 210 beats per minute which converted to a sinus rhythm with six mg of intravenous (IV) adenosine.
- Norah suffers from paroxysmal supraventricular tachycardia likely due to atrioventricular (AV) node reentry or AV reentry.

Table 1

Key historical questions

- Pattern of onset of symptoms (sudden or gradual)?
- Associated symptoms?
- Cardiac history, including functional capacity?
- Relation of symptoms with exertion?
- Episode triggers?
- Family history of sudden death?
- Degree of disruption to daily activities?

Supraventricular tachycardia (SVT) is broadly defined as a narrow-complex tachyarrhythmia (QRS width < 120 milli-seconds). Using this definition, SVTs may affect up to one per cent of the population.[1] By convention, atrial fibrillation and atrial flutter are often categorized separately from SVTs, such as atrial tachycardia, atrioventricular (AV) reciprocating tachycardia (AVRT) and AV nodal reciprocating tachycardia (AVNRT). Ignoring the more common atrial fibrillation and atrial flutter, the prevalence of SVTs is approximately 0.2% and 0.3% in the general population.[2] Details of atrial fibrillation and atrial flutter will not be discussed in this review.

Clinical features

SVT may present in a variety of ways, though typically, the patient complains of abrupt onset of palpitations, with varying accompanying symptoms. Common symptoms include:

- chest tightness,
- shortness of breath,
- lightheadedness and
- rarely, syncope.

Most types of SVT begin and terminate abruptly. By contrast, sinus tachycardia or atrial tachycardia tend to be less paroxysmal, with rhythm change occurring in a more gradual manner. Stereotypic triggers—such as caffeine, alcohol, or exertion—are rarely found despite common perception. Rather, autonomic maneuvers, such as postural change, can trigger the initiation of an SVT with a re-entrant mechanism. The patient interview is an opportunity to gain a sense of the symptomatic burden of the SVT, as the

impact to one's quality of life will often dictate management. Table 1 summarizes key historical data.

Approach and differential diagnosis

A practical classification of SVT is based on establishing the role of the AV node in the tachycardia, as either AV node-dependent or AV node-independent (Figure 1). This scheme allows AV node blocking maneuvers to assist with diagnosis. In a hemodynamically stable patient, diagnostic maneuvers include physiologic (*e.g.,* carotid massage) and pharmacologic (*e.g.,* intravenous [IV] adenosine) means of temporarily blocking conduction through the AV node. Given its very short half-life, a six mg to 12 mg IV push of adenosine is quite safe. However, common contraindications include severe obstructive or reactive airway disease.

The response of the SVT to such maneuvers is often diagnostic and therefore, a rhythm strip should be recorded during the diagnostic maneuvers. Figure 2 demonstrates the expected responses of an AV node-dependent (AV node re-entry) and independent (atrial flutter) SVT to AV nodal blockade with adenosine. AV node-dependent tachycardias cease abruptly whereas AV node-independent tachycardias demonstrate decreased AV node conduction; however, the underlying tachycardia persists.

FAQ

What is the best way to record an episode of heart palpitations?

Although Holter monitoring is widely available, symptoms must recur in the 24 hours to 48 hours from when the device is worn. Telephone transmitters (*i.e.*, event recorders) or external loop recorders are much higher yield because they prolong the duration of monitoring and are arranged by a specialist who is familiar with local availability.

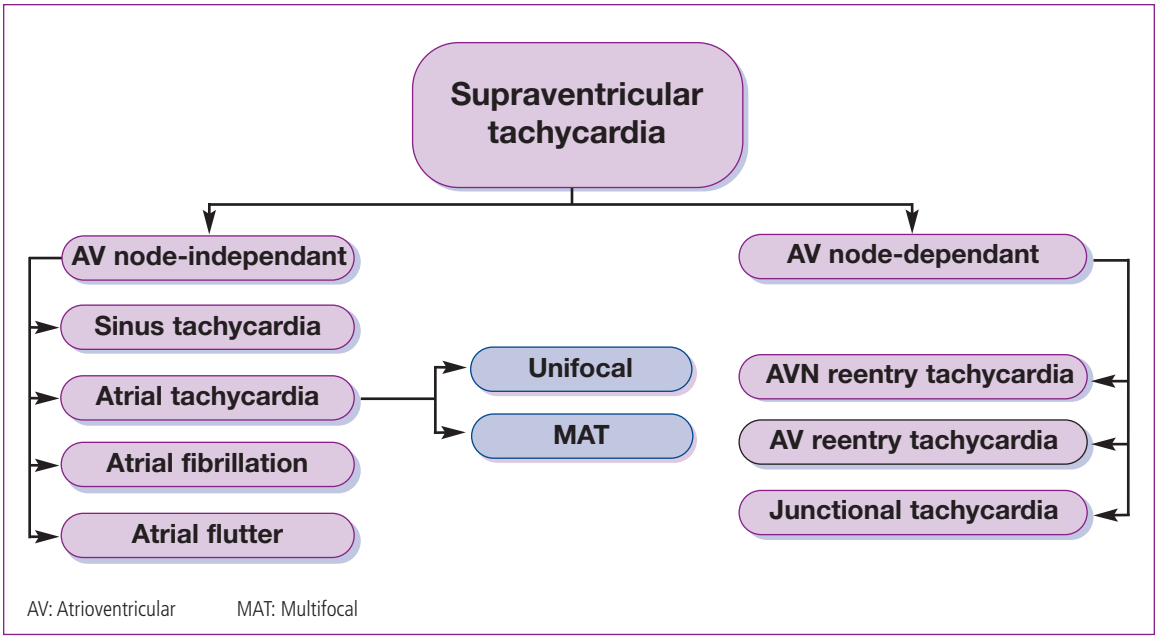

Figure 1. Classification of supraventricular tachycardias.

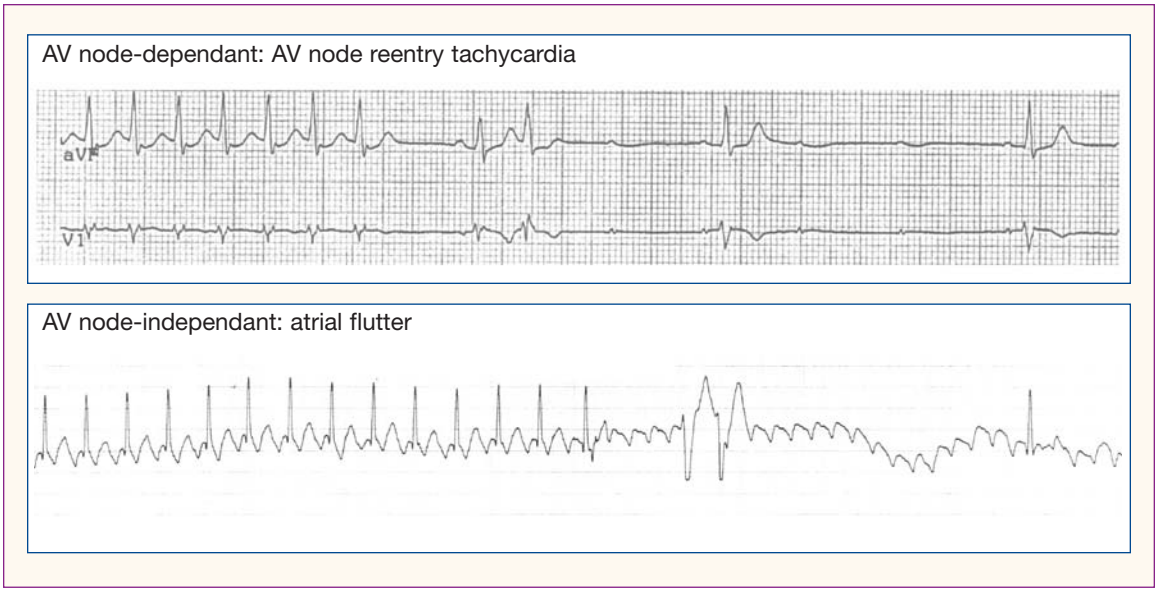

Figure 2. Response of regular SVTs to adenosine.
Adminstration of an IV adenosine bolus (six mg or 12 mg) causes abrupt termination of AV node-dependent tachycardias (upper), whereas AV node-independent tachycardias persist with slowed ventricular conduction (lower).

Long-term management

In general, SVTs are associated with an excellent prognosis in a patient with a structurally normal heart. Patient reassurance is the rule. An exception is the small risk of sudden death due to ventricular arrhythmias associated with Wolff-Parkinson-White (WPW) syndrome estimated at 0.1% per patient year.[3-4] Sudden death is postulated to occur due to rapid conduction of atrial fibrillation over the accessory pathway (Figure 3). Due to this small risk, patients with a suspected AV accessory pathway should have a discussion with a specialist regarding treatment options.

Pharmacologic treatments

In general, treatment of SVT is a quality of life intervention, based on frequency and severity of symptoms. Observation without therapy is acceptable if the patient is not excessively symptomatic.

In patients with AV node-dependent SVT seeking treatment, therapy targets AV node conduction to prevent future episodes.

FAQ

Who should I refer?

Patients presenting with:
a) Diagnostic dilemmas

b) Symptomatic SVT requiring long-term medical therapy

c) WPW pattern (symptomatic or asymptomatic)

d) Other ablation candidates
(refer to FAQ on page 76 for more information)

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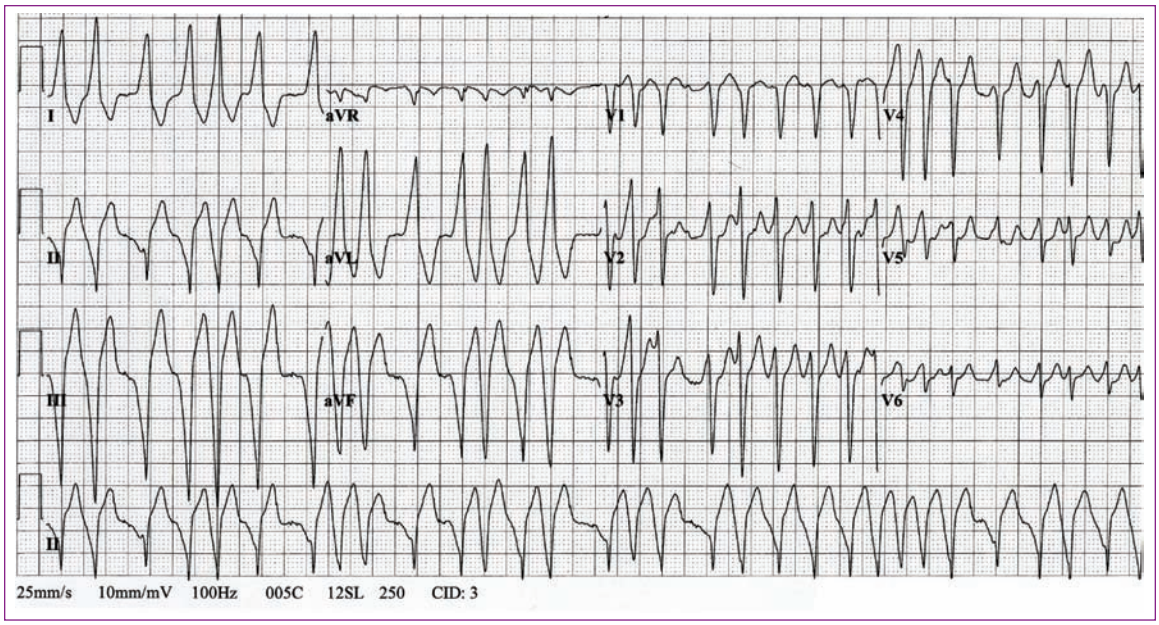

Figure 3. Atrial fibrillation in Wolff-Parkinson White (WPW) syndrome.
Wide complex polymorphic irregular tachycardia representing atrial fibrillation with variable conduction through the accessory pathway. Treatment of choice is IV procainamide if stable and electrical cardioversion if unstable. AV node blocking agents are contraindicated.

All patients should be counseled on:

- vagal maneuvers such as Valsalva,
- forceful coughing or
- the splashing cold water on the face.

First-line medications, which may be titrated up as symptoms require, include:

- ß-blockers,
- calcium channel blockers and
- digoxin.

Once again, the only exception would be patients with pre-excitation on ECG where calcium channel blockers and digoxin should be avoided.

In patients with an AV node-independent SVT, treatments are more varied. In focal atrial tachycardia, beta-blockers may be helpful. Otherwise, Class Ic (*e.g.*, propafenone 150 mg to 300 mg, three times daily or flecainide 50 mg to 150 mg, twice daily) or Class III antiarrhythmics (*e.g.,* sotalol 80 mg to 160 mg, twice daily) can be attempted. In multifocal atrial tachycardia and sinus tachycardia, the arrhythmia is most often secondary to another process and management should be focused on finding the underlying cause (*e.g.,* infection, hyperthyroidism, lung disease, *etc.*).

FAQ

Are there patients with SVT in whom it would not be safe to start an AV node blocking agent?

Routine contraindications apply (e.g., bronchospasm, bradycardia or hypotension for beta-blockers). Additionally, the patient with WPW and atrial fibrillation warrants early referral.

What investigations do I need to send to the specialist?

The most important information is a copy of the documented arrhythmia, plus any rhythm strips during adenosine administration, if performed. An echocardiogram is often helpful.

FAQ

Is ablation considered first-line therapy in anyone?

Symptomatic atrial flutter, nodal reciprocating tachycardia and reciprocating tachycardia, can be referred early due to the excellent risk-to-benefit ratio of ablation; although, medications can be attempted first. Ablation is not a first-line therapy for atrial fibrillation or atrial tachycardia.

Take-home message

1. SVTs are generally benign diagnoses with treatments based on symptom-burden.
2. SVTs should be approached as AV node-dependent and AV node-independent when a diagnostic maneuver is performed during an episode.
3. AV nodal blocking agents are generally good empiric first-line agents, with the exception of WPW syndrome with atrial fibrillation.
4. Potentially curative ablation procedures are available for atrial flutter, AVRT and AVNRT, but they should be considered in atrial fibrillation and atrial tachycardia only after the failure of medical therapy.

Invasive treatments

Percutaneous procedures in the electrophysiology laboratory have advanced tremendously over the last two decades. Catheters are inserted into the heart via the femoral and subclavian veins under local or general anesthetic, or less commonly, from the femoral artery. Diagnostic testing and curative ablation, using radio frequency energy, is performed, targeting and eliminating the responsible tissue. Ablation has now become routine for most reentrant arrhythmias, with cure rates of 95% or greater for AV node reentry, AV reentry and atrial flutter. Focal atrial tachycardia and atrial fibrillation ablations are also performed, with a more modest success rate of 70%. Complication rates are in the range of one per cent to two per cent and depend on the location of the ablation target.

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References
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