

Supraventricular Tachycardia

Supraventricular tachycardia (SVT) is a rapid heart rate that originates in the top chambers of the heart called the atria. This is a relatively common entity that effects 570,000 people in the United States with an estimated 89,000 new cases reported annually. While the average age of onset of symptomatic SVT is 57 years old, it can affect people at any age. The incidence and prevalence increases with increasing age. Women are twice as likely as men to experience this arrhythmia. More than a third of patients have no associated cardiovascular disease. Three mechanisms predominate in SVT (excluding atrial fibrillation and atrial flutter, which will not be part of this discussion). The most common etiology is atrioventricular nodal reentrant tachycardia (AVNRT) which constitutes 56% of cases. Atrioventricular reentrant tachycardia (AVRT) utilizes an accessory pathway (an extra electrical connection between the top and bottom heart chambers) and accounts for 27%. Atrial tachycardia and other more uncommon arrhythmias (junctional tachycardia and inappropriate sinus tachycardia) are responsible for the remaining 17%.

SVT is usually regular with a heart rate of 160 to 200 beats per minute, but can range from 80 to 240 beats per minute. These arrhythmias are usually not life-threatening. Symptoms include palpitations that can last seconds to hours, a pounding sensation in the chest or neck, shortness of breath, anxiety, a feeling as though one may pass-out and uncommonly, loss of consciousness. In very rare instances, SVT can result in cardiac arrest when the arrhythmia is atrial fibrillation or flutter conducted rapidly over an accessory pathway to the bottom heart chambers (ventricles). Most patients are not able to identify a clear trigger for the episodes. However, possible precipitators include substances such as caffeine, alcohol and nicotine, psychological stress, lack of sleep, dehydration and illnesses such as anemia, hyperthyroidism, asthma and emphysema and acute infectious illnesses such as pneumonia, upper respiratory tract infections or urinary tract infections. In women, menstruation and pregnancy can precipitate episodes. Tachycardia episodes have a circadian pattern and are more likely to happen during the day than at night.

Conduction in the normal heart is initiated in the sinus node, is conducted through the atrial tissue to the AV node, then into the His bundle and out to the left and right fascicular bundles and finally to the ventricular muscle. The figure below illustrates the normal cardiac conduction system.

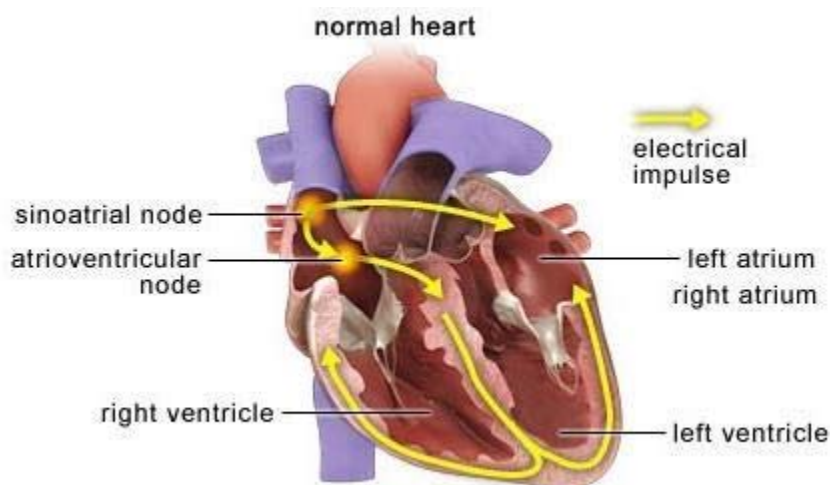


Figure adapted from The British United Provident Association Limited website at http://hcd2.bupa.co.uk/fact_sheets/html/supraventricular_tachycardia.html

Atrial tachycardia is a regular tachycardia that is initiated in the upper chamber (atrium) and does not involve the sinus node or the AV node. The mechanism can either be reentry or automaticity. Like with AVNRT or AVRT, a reentrant mechanism involves a circuit that is continually activated causing electrical impulses to go around and around the circuit perpetuating tachycardia. This usually occurs in the setting of atrial scar, commonly from prior surgery, such that the macroreentrant circuit goes around and around the scar. Automatic arrhythmias are generated from a single focus within the atrium that continually fires (much like the sinus node, although at faster rates) precipitating tachycardia. Approximately two thirds occur from the right atrium and the remaining from the left. The figure below illustrates an automatic atrial tachycardia.

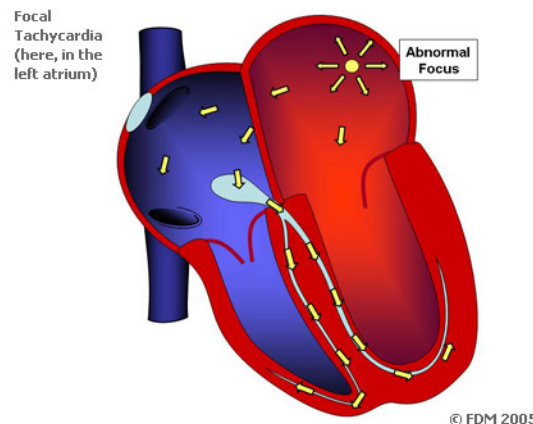


Figure adapted from the London Arrhythmia Centre website at <http://www.londonarrhythmiacentre.co.uk/diagnosis-svt-atrial-tachycardia.html>

Atrioventricular nodal reentrant tachycardia (AVNRT) is a reentrant arrhythmia involving a microcircuit within the AV node, which is the “junction box” connecting the electrical system of the top chambers to that of the bottom chambers. Up to 20% of the population has two pathways within their AV node. An extra beat (usually from the top chamber) can initiate a situation in which the electrical impulse goes down one pathway and back up the other pathway initiating a circuit that goes around and around the AV node causing AVNRT. The circuit can go either way around the node causing either typical or atypical AVNRT. The treatment for both is the same. The figure below demonstrates AVNRT.

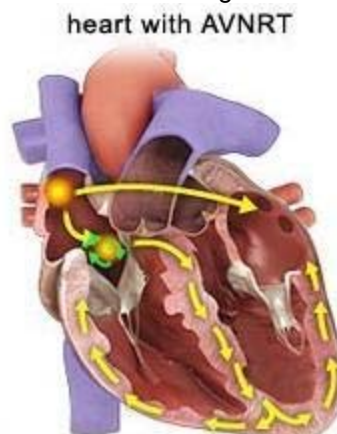


Figure adapted from The British United Provident Association Limited website at http://hcd2.bupa.co.uk/fact_sheets/html/supraventricular_tachycardia.html

Atrioventricular reentrant tachycardia (AVRT) utilizes an accessory pathway, which is an abnormal electrical connection between the top and bottom chambers. This pathway is formed and maintained during embryologic development. Thus, people who have these pathways are born with them. In the general population, 0.15 to 0.25% of people have an accessory pathway demonstrated on baseline electrocardiogram. Less than 2% of this population will develop symptomatic tachycardia (Wolff Parkinson White syndrome). As indicated above, there is a rare incidence of cardiac arrest in people with accessory pathways. It is estimated that approximately 2% of patients with symptomatic accessory pathway mediated tachycardia will suffer cardiac arrest, or less than 0.0001% of the general population. Accessory pathways occur around the tricuspid (right side) and mitral (left side) valves (the valves between the top and bottom chambers). Seventy to eighty percent occur on the left side, with the remaining occurring on the right side. These pathways may conduct from the top to the bottom chamber, from the bottom to the top chamber or both. As with AVNRT, an extra beat (from either the top or bottom chamber) can initiate a perpetuating reentrant circuit that goes up the accessory pathway and down the AV node (orthodromic), or vice versa (antidromic), causing AVRT. Up to 20% of people with accessory pathways have more than one pathway. It is important to note that people can have accessory pathways which are not involved in the specific tachycardia that they suffer from. These are called bystander pathways. Below is a diagram demonstrating AVRT.

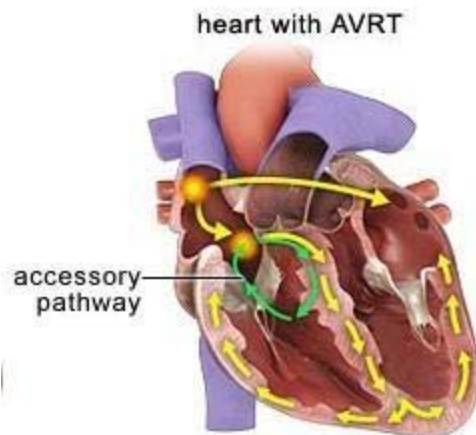


Figure adapted from The British United Provident Association Limited website at http://hcd2.bupa.co.uk/fact_sheets/html/supraventricular_tachycardia.html

An electrophysiology (EP) study is often used to determine the exact mechanism of the arrhythmia and to determine the risk of sudden cardiac death in patients whose arrhythmia mechanism involves an accessory pathway. This procedure is performed in the electrophysiology laboratory. It involves advancing two to four catheters through the veins in the groin (femoral veins) up to the heart. This is done with the use of fluoroscopic imaging, a form of X-ray. These catheters allow us to map the conduction system of the heart and to pace the heart to induce the abnormal heart rhythm such that we can characterize it. Sometimes, the arrhythmia can not be initiated with pacing alone and we use medications to speed the heart rate and increase the ability to initiate the abnormal heart rhythm. The figure below shows a fluoroscopic image with the typical placement of catheters in the heart for an EP study.

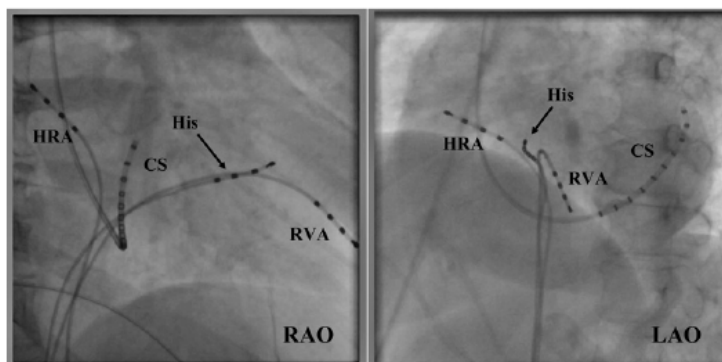


Figure adapted from Lee KW, Badhwar N and Scheinman MM. Supraventricular Tachycardia—Part I. *Curr Probl Cardiol* 2008;33:467-546.

Long-term treatment of SVT can include management with medications or a more invasive approach with catheter ablation. Medical management commonly includes treatment with one or more atrioventricular (AV) nodal blocking agents such as beta blockers (metoprolol, propranolol, atenolol) or calcium channel blockers (diltiazem, verapamil). Antiarrhythmic medications, such as flecainide and propafenone, may also be effective. However, catheter ablation is often considered early in the management plan because it is highly effective and is a relatively low risk procedure in most patients. In patients with symptomatic AVRT (Wolff Parkinson White syndrome), catheter ablation is the recommended first-line therapy.

If an ablation procedure is to be done, this is usually performed at the time of the EP study. One of the catheters we use emits high frequency radio waves from its tip which are transmitted to the cardiac tissue in the form of heat. This allows us to burn areas in the heart that precipitate the abnormal heart rhythms. These burns create scars. Electricity can not travel through a scar. Therefore, this leaves the "ablated" tissue unable to induce or sustain the arrhythmia. For the treatment of AVNRT, catheter ablation is very effective with reported immediate success rates up to 99% and long-term arrhythmia recurrence rates of 1 to 5%. As the ablation is performed near the AV node there is a 0.4 to 1% risk of AV block requiring implantation of a permanent pacemaker. We use sophisticated 3-dimensional mapping systems which allow for precise localization of our ablation increasing the safety of this procedure. In cases where the ablation target area is very close to the AV node, we can use a different ablation method called cryoablation. This technique freezes the tissue, rather than burning it. These lesions are more reversible and tend to carry less of a risk of permanent AV nodal damage. The trade-off is that cryoablation is associated with lower immediate success rates (91%) and higher recurrence rates.

Catheter ablation of AVRT is also highly effective with an immediate success rate of 90 to 97% with a recurrence rate of up to 6%. In patients with pathways near the AV node, the risk of nodal damage and the need for pacemaker implantation is similar to that of AVNRT ablation. Again, several precautions are taken to ensure the least possible risk of damage to the AV node. When the pathway is located on the mitral valve annulus it is necessary to access the left side of the heart. This is done either by a transseptal puncture (poking a hole through the wall that separates the two top chambers) or entering the left side from the aorta via the femoral artery. Ablating in the left side of the heart adds the additional risk of stroke that is not present when ablating on the right side. For this reason, high dose anticoagulation is used during the procedure when the left side is accessed. These blood thinners can increase the risk of bleeding during left sided procedures. If ablation is performed on the left side, the patient is required to take 325mg of aspirin everyday for 6 to 8 weeks to decrease the risk of stroke. Overall, the procedure of accessory pathway ablation has a complication rate of 1 to 4% with a procedure-related death rate of 0.2%.

The method for ablating atrial tachycardia depends on the mechanism of the arrhythmia. Targeting the abnormal focus is used in automatic ATs. Reentrant AT ablation involves defining the circuit (and usually the atrial scar) and ablating a critical area within the circuit necessary to propagate the arrhythmia. Ablation of atrial tachycardia has an immediate success rate of 68-90% with recurrence rates of 5 to 20%. In the case of recurrence, a second ablation procedure is usually highly effective. As with AVNRT and AVRT ablation, there is a risk of AV nodal damage when ablating near the AV node, as well as risk of stroke if the target for ablation is on the left side of the heart. There is also a small risk of damage to the nerve that supplies the diaphragm when ablating atrial tachycardias in the right atrium. This could result in dysfunction of the diaphragm causing shortness of breath. Again, great care is taken when ablating in this area and pacing maneuvers are performed to identify the risk of damage to the phrenic nerve during ablation.

In addition to the risks discussed above (AV nodal damage requiring pacemaker placement, stroke when accessing the left side of the heart and phrenic nerve injury resulting in diaphragm dysfunction) other risks of EP study and ablation include bleeding or infection in the groin area, damage to the vessels and heart during catheter placement, manipulation and ablation, heart attack and very rarely death. The overall risk of major complications with ablation of SVT is 1 to 3%.

For more information on supraventricular tachycardia, we recommend the following articles available online and through our office:

Lee KW, Badhwar N and Scheinman MM. Supraventricular Tachycardia—Part I. *Curr Probl Cardiol* 2008;33:467-546.

Lee KW, Badhwar N and Scheinman MM. Supraventricular Tachycardia—Part II: History, Presentation, Mechanism and Treatment. *Curr Probl Cardiol* 2008;33:557-622.

Nakagawa H and Jackman WM. Catheter Ablation of Paroxysmal Supraventricular Tachycardia. *Circulation*. 2007;116:2465-2478.

Fox DJ, Tischenko A, Krahn AD et al. Supraventricular Tachycardia: Diagnosis and Management. *Mayo Clinic Proc*. 2008;83(12):1400-1411.

Patient information is also available on the website of the Heart Rhythm Society at www.HRSonline.org.