SVT Types and Treatement

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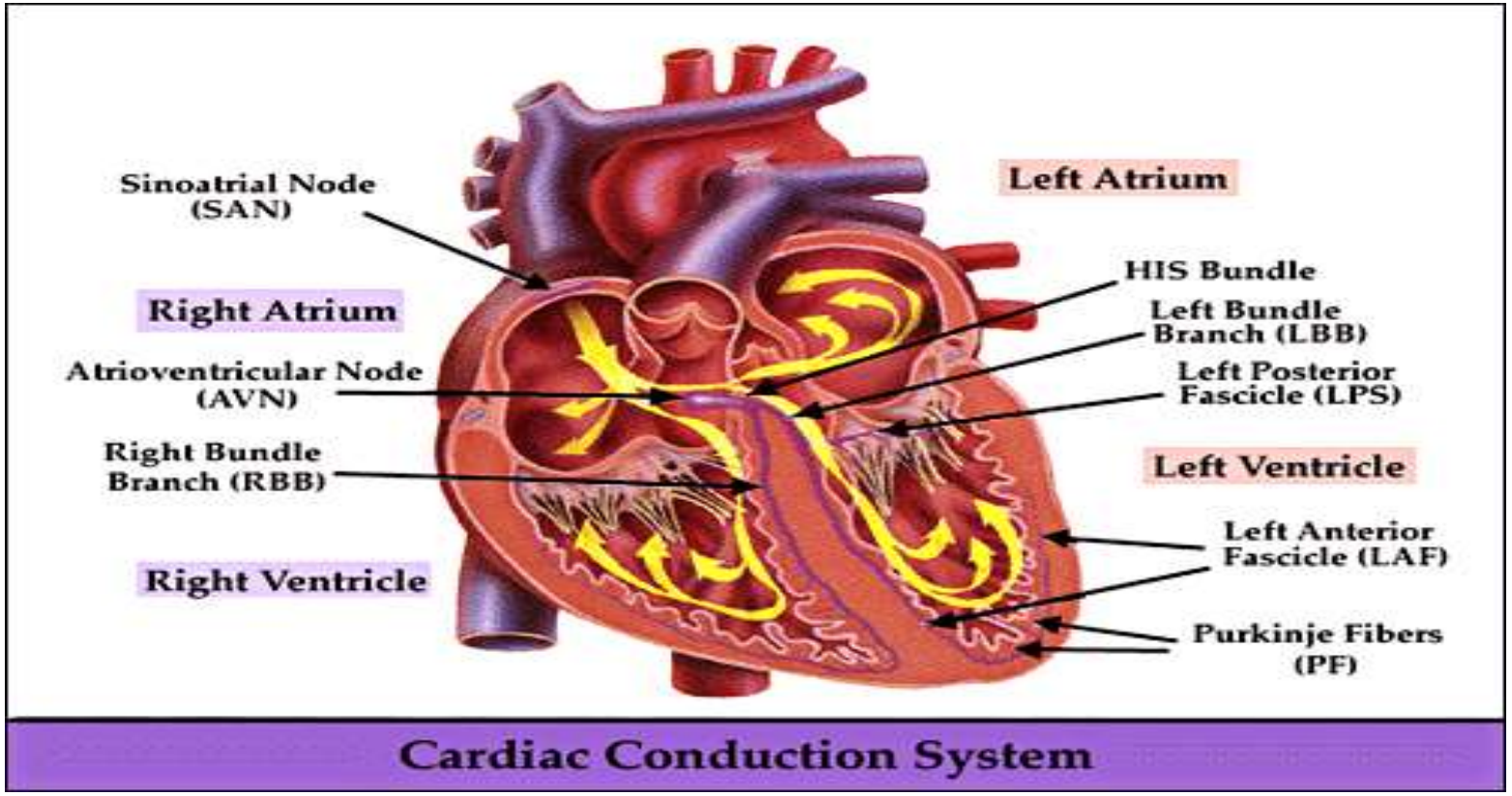
Learning Objectives

- Following the presentation, the participant should be able to:

1. Distinguish and explain the types of SVT.
2. Recognize patterns of SVT on ECG.
3. Understand indications and basics of invasive EP studies.
4. Review various pharmacologic agents role in treating SVT's.

Electrophysiology

- Contraction of heart is normally the result of well-orchestrated electromechanical system.



Orderly Function

- Orderly function of system is maintained:
  - by the domination of heart rate by a single pulse generator known as pacemaker
  - by the relatively fast & uniform conduction of electrical signal via specialized conduction pathways
  - by relatively long & uniform duration of electrical signal relative to its velocity of conduction through these pathways
  - these things assure uniform electrical excitation & contraction of the heart

Define arrhythmia

- Any disturbance in the normal sequence of impulse generation & conduction in the heart
- Arrhythmias may occur with or without underlying heart disease.

Action potential

- Cardiac muscle has some similarities to skeletal muscle, as well as important unique properties. Like skeletal myocytes (and axons for that matter), a given cardiac myocyte has a negative membrane potential when at rest. A notable difference between skeletal and cardiac myocytes is how each elevates the myoplasmic Ca^{2+} to induce contraction.
- When skeletal muscle is stimulated by somatic motor axons, influx of Na^{+} quickly depolarizes the skeletal myocyte and triggers calcium release from the sarcoplasmic reticulum. In cardiac myocytes, the release of Ca^{2+} from the sarcoplasmic reticulum is induced by Ca^{2+} influx into the cell through voltage-gated calcium channels on the sarcolemma. This phenomenon is called calcium-inducted calcium release and increases the myoplasmic free Ca^{2+} concentration causing muscle contraction.
- In both muscle types, after a delay, (the absolute refractory period), potassium channels reopen and the resulting flow of K^{+} out of the cell causes repolarization to the resting state. The voltage gated sodium channels in the cardiac sarcolemma are generally triggered by an influx in sodium during the "0" phase of the action potential.

More action potential

- Once the cell is electrically stimulated (typically by an electric current from an adjacent cell), it begins a sequence of actions involving the influx and efflux of multiple cations and anions that together produce the action potential of the cell, propagating the electrical stimulation to the cells that lie adjacent to it. In this fashion, an electrical stimulation is conducted from one cell to all the cells that are adjacent to it, to all the cells of the heart.

SA nodal cells

- Note that there are important physiological differences between nodal cells and ventricular cells; the specific differences in ion channels and mechanisms of polarization give rise to unique properties of SA node cells, most importantly the spontaneous depolarizations (cardiac muscle automaticity) necessary for the SA node's pacemaker activity.

A normal heart beat

- The electrical impulse begins at the SA node, also called the heart's natural pacemaker. The SA node is a cluster of specialized cells, located in the right atrium. The SA node produces the electrical impulses that set the rate and rhythm of your heartbeat. The impulse spreads through the walls of the right and left atria, causing them to contract, forcing blood into the ventricles.
- The impulse then reaches the atrioventricular (AV) node, which acts as an electrical bridge allowing impulses to travel from the atria to the ventricles. There is a short delay before the impulse travels on to the ventricles.
- From the AV node, the impulse travels through a pathway of fibers called the HIS-Purkinje system. This network sends the impulse into the muscular walls of the ventricles and causes them to contract. This contraction forces blood out of the heart to the lungs and body.
- The SA node fires another impulse and the cycle begins again.

Mechanisms of Arrhythmia

- 3 basic causes
  - suppression or enhancement of initiation or propagation of action potential
  - ectopic pacemaker activity
  - reentry of action potential into a pathway through which its already passed

  May be more than one of these things happening at a time to create a particular arrhythmia

Ectopic Pacemaker

- Enhanced automaticity of any part of the cardiac conduction system may result in initiation of an impulse faster than normal SA node.
- If this happens occasionally, a premature contraction will occur. The type depends on where it originates (PVC, PAC, etc)
- If there is rapid, sustained firing of the ectopic focus, a tachyarrhythmia will be produced.

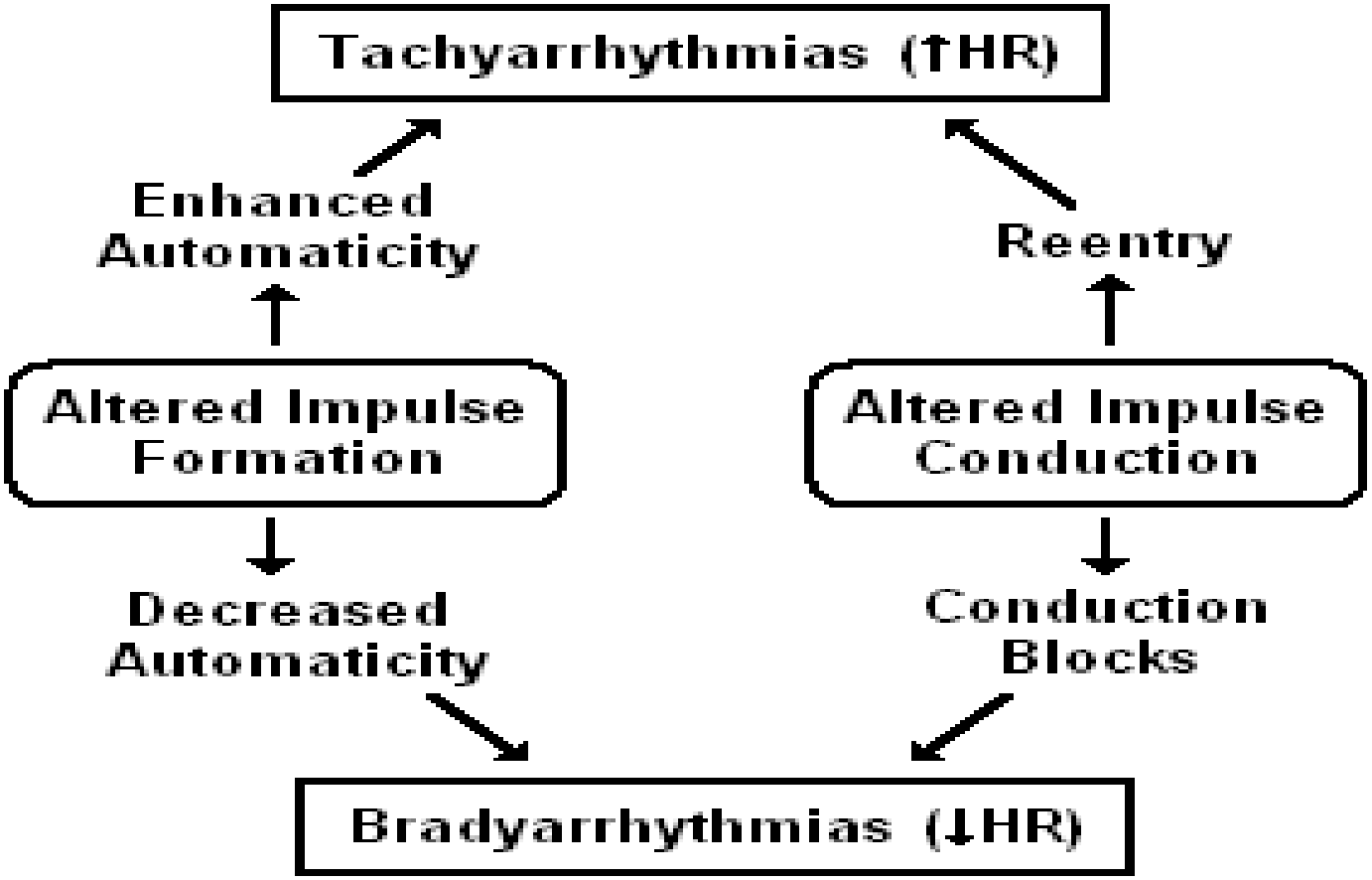
Reentry

- Alterations in refractory period of adjacent pathways & of the velocity of the impulse through them may allow retrograde conduction of AP through one of the pathways.
- Sustained reentry implies either unusual pathways for conduction of AP or poorly functioning & thus slowly conducting myocardium.

More about mechanisms

- Triggered activity and abnormal automaticity are particularly prone to occur along the crista terminalis in the RA.
- LA foci near or within the pulmonary veins are also common.
- Intra-atrial re-entry is more common in patients with underlying heart disease and associated atrial scars that form zones of slow conduction and boundaries that promote re-entry.
- Re-entry around surgical scars is another common mechanism.

Mechanisms of Arrhythmogenesis



Normal Conduction

- The electrical impulse originates in the sinus node. From there, it spreads across both atria, causing the atria to contract. As the electrical impulse passes through the atria, it generates the so-called "P" wave on the ECG.

More conduction

- The specialized AV conduction system consists of the AV node (AVN), the "His bundle," and the right and left bundle branches (RBB and LBB). The AV node conducts the electrical impulse very slowly, and passes it to the His bundle. The His bundle penetrates the AV disk, and passes the signal to the right and left bundle branches. The right and left bundle branches, in turn, send the electrical impulse to the right and left ventricles, respectively. LBB itself splits into the left anterior fascicle (LAF) and the left posterior fascicle (LPF).
- Because the impulse travels only very slowly through the AV node, there is a pause in the electrical activity on the ECG, referred to as the PR interval.

Final conduction

- The electrical impulse spreads throughout the right and left ventricles, causing these chambers to contract. As the electrical signal travels through the ventricles, it generates the “QRS complex” on the ECG.
- The T wave corresponds to ventricular repolarization.

Supraventricular Tachycardia

- Among most common conditions encountered in cardiology.
- Includes any tachycardia with signal originating above the ventricles.
- This includes AV nodal tachycardias.
- Because of the abrupt onset and termination of the reentrant SVT, the nonspecific term paroxysmal SVT has been used to describe these tachyarrhythmias

Types of SVT

- Premature atrial contractions (PACs)
- Paroxysmal supraventricular tachycardia (PSVT)
- Accessory pathway tachycardia (such as Wolff-Parkinson-White syndrome)
- Atrial tachycardia
- Atrial fibrillation
- Atrial flutter

SVT's

- Supraventricular arrhythmias are the most common cause of palpitations in patients who do not have structural heart disease.
- It is important to ascertain precipitating factors, the frequency of events, prior medical therapy, the duration of the tachycardia, whether it is sudden or gradual in onset, regular or irregular, and whether vagal maneuvers effectively terminate it.

Table 3. Predisposing or Precipitating Factors for Patients With Palpitations

Noncardiac Causes
Nicotine, alcohol, caffeine
Physical or mental stress
Hyperthyroidism
Premenstrual or menstrual
Electrolyte disturbance
Certain drugs (antiarrhythmic, antidepressant, antibiotic drugs; stimulants; antihistamines; appetite suppressants)
Anemia
Anxiety or hypovolemia
Fever, infection
Lack of sleep
Cardiac Causes
Coronary artery disease; old myocardial infarction, especially for ventricular tachycardias
Congestive heart failure
Cardiomyopathy
Valvular disease
Congenital heart disease
Other conditions that may cause myocardial scarring (ie, sarcoidosis, tuberculosis)
Primary electrical disorders (ie, long QT syndrome, Brugada syndrome)
Accessory pathways

Definitions

- Antegrade - conduction from atria to the ventricles
- Retrograde - conduction from ventricles to atrium

- Orthodromic – antegrade down AV & retrograde down accessory
- Antidromic - antegrade conduction down the accessory tract and then retrograde reentry of the normal pathway

3 most common types of PSVT

- Atrioventricular node reentrant tachycardia (AVNRT)
- Atrioventricular reentrant tachycardia (AVRT)
- Atrial tachycardia

AVNRT

- Also sometimes referred to as a junctional reciprocating tachycardia.
- It involves a reentry circuit forming just next to or within the AV node itself. The circuit most often involves two tiny pathways one faster than the other, within the AV node.
- Because the AV node is immediately between the atria and the ventricle, the re-entry circuit often stimulates both, meaning that a retrogradely conducted p-wave is buried within or occurs just *after* the regular, narrow QRS complexes.

AVRT

- Also results from a reentry circuit, although one physically much larger than AVNRT. One portion of the circuit is usually the AV node, and the other, an abnormal accessory pathway from the atria to the ventricle. WPW Syndrome is a relatively common abnormality with an accessory pathway, the Bundle of Kent crossing the A-V valvular ring.

AVRT

In orthodromic AVRT, atrial impulses are conducted down through the AV node and retrogradely re-enter the atrium via the accessory pathway. A distinguishing characteristic of orthodromic AVRT can therefore be a p-wave that follows each of its regular, narrow QRS complexes, due to retrograde conduction.

In antidromic AVRT, atrial impulses are conducted down through the accessory pathway and re-enter the atrium retrogradely via the AV node. Because the accessory pathway initiates conduction in the ventricles outside of the bundle of His, the QRS complex in antidromic AVRT is often wider than usual, with a delta wave.

Atrial Tachycardia

- Tachycardia resultant from one ectopic foci within the atria, distinguished by a consistent p-wave of abnormal morphology that fall before a narrow, regular QRS complex.

Differential Dx of Narrow Complex Tachycardia

- Most common presentation is palpitations
- ECG shows narrow QRS complex tachycardia

- Typical pt with AVNRT or AVRT presents with periods of palpitations that occur with sudden onset and abruptly end.
- During palpitations, pt may describe light-headedness, anginal-type chest pressure, or uneasy sensation in neck.
- Syncope is rare, but can occur in elderly pts.

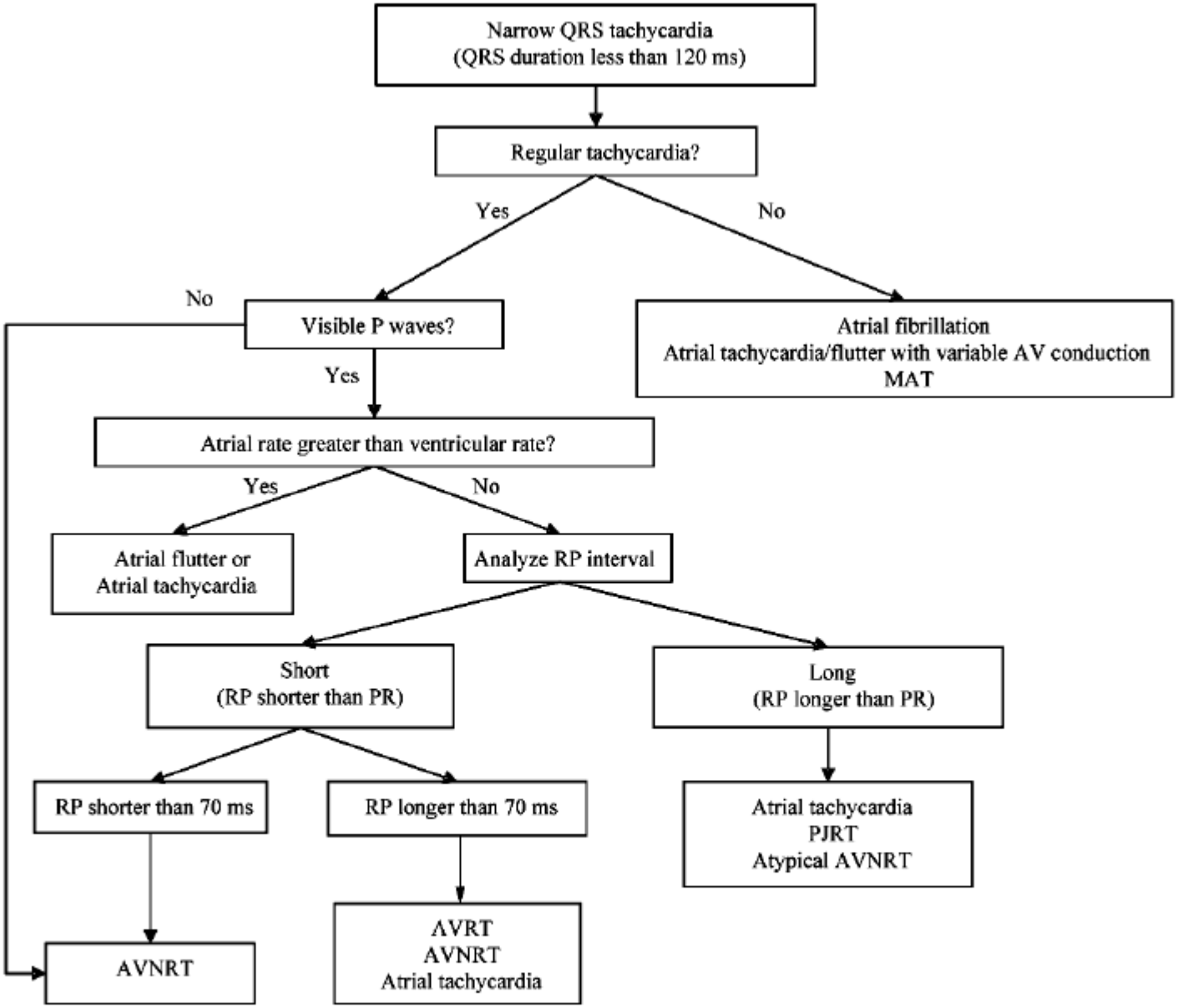


Figure 3. Differential diagnosis for narrow QRS tachycardia. Patients with focal junctional tachycardia may mimic the pattern of slow-fast AVNRT and may show AV dissociation and/or marked irregularity in the junctional rate. AV indicates atrioventricular; AVNRT, atrioventricular nodal reciprocating tachycardia; AVRT, atrioventricular reciprocating tachycardia; MAT, multifocal atrial tachycardia; ms, milliseconds; PJRT, permanent form of junctional reciprocating tachycardia; QRS, ventricular activation on electrocardiogram.

ECG Differentiation

- Regular narrow complex tachycardia identified:
  - 1st step determine if P waves exist
  - Is it closer to preceding or succeeding QRS - (thus short or long RP tachycardias)

RP Interval

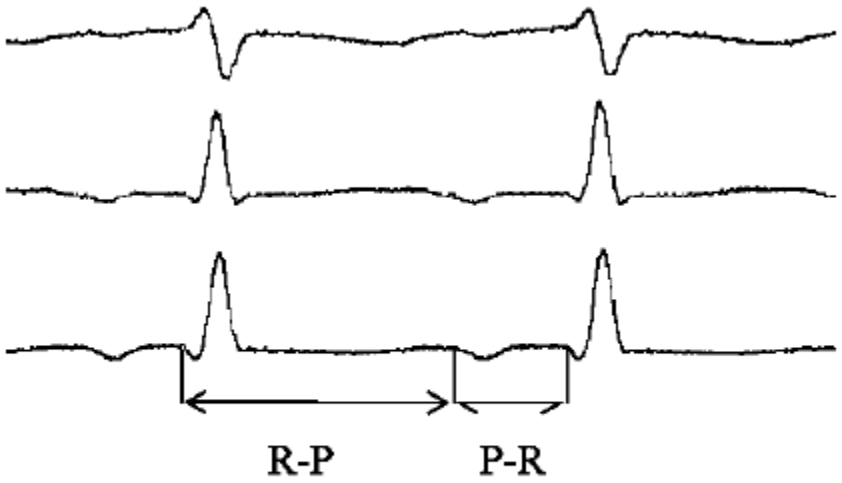


Figure 5. ECG tracing with limb leads I, II, and III, showing an RP (initial R to initial P) interval longer than the PR interval. The P wave differs from the sinus P wave. ECG indicates electrocardiogram.

RP

- Short RP tachycardia indicates relatively fast retrograde activation of atrium (orthodromic AVRT) or near simultaneous activation of atrium & ventricle (AVNRT & junctional tachycardias)
- Similar to sinus tachycardia, AT has long RP interval b/c there is no retrograde activation of atrium from ventricle.
- Once P wave is noted, if it occurs within 1st half of RR interval (short RP interval), AVNRT & orthodromic AVRT should be considered
- If PR interval is shorter than RP interval, AT is likely.

AVNRT

- AV nodal re-entrant tachycardia is the most common form of paroxysmal SVT.
- The initial presentation of AV node re-entry occurs most often in the fourth and fifth decades, although it can manifest at any age.
- It is more common in women than in men.

AVNRT

- AV node located on intra-atrial septum close to tricuspid annulus. Atrial myocardium connects electrically to the AV node at distinct sites.
- Sinus node impulse travels superior to fossa ovalis and posterior to eustachian ridge to reach AV node.
- These fibers are “fast pathway - alpha” to AV node.
- 2nd method of reaching AV node is anterior (more ventricular) to eustachian ridge from coronary sinus region.
- These inferior fibers from coronary sinus to AV node are “slow pathway - beta”.
- B/c of these 2 distinct atrial connections to AV node, reentrant tachycardia is possible.

Mechanism of AVNRT

- AV nodal re-entry results from a re-entrant circuit characterized by two functionally discrete pathways that incorporate the compact AV node and are distinguished by their rates of conduction.
- The fast pathway conducts more rapidly than the slow pathway, but recovery of the fast pathway takes more time because it has a longer refractory period.
- Although the tissues that comprise these pathways are not completely delineated, the fast pathway is located in the anterior septum and includes the compact AV node.
- The slow pathway is located along the septal aspect of the tricuspid annulus posterior to the compact portion of the AV node.

More AVNRT

- The tachycardia is initiated when an appropriately timed atrial premature complex is blocked in the fast pathway (longer refractory period) and conducts in the slow pathway (shorter refractory period).
- While the impulse conducts to the ventricle in the slow pathway (antegrade conduction), the fast pathway recovers so that the impulse can conduct retrograde up the fast pathway to the atrium and the atrial end of the slow pathway (retrograde conduction).
- This sets up the reentrant circuit.

Typical AVNRT

- Again, antegrade conduction here is slow while retrograde is fast
- Therefore, atrial activity begins soon after ventricular activation, which can create an inability to see P waves of ACG.

AVNRT

- AVNRT from a common point near AV node, activation of atrium & ventricle is near simultaneous.
- Therefore, very short RP interval (sometimes even 0 or negative), are possible.

AVNRT continues

- In addition to the typical mechanism of AVNRT, atypical AV nodal reentry can occur in the opposite direction, with antegrade conduction in the fast pathway and retrograde conduction in the slow pathway.
- Long RP, short RP, inverted P waves in inferior leads.
- Less commonly, the reentrant circuit can be over 2 slow pathways, the so-called slow-slow AV node reentry.

ECG Findings Again

- Evaluation usually reveals a supraventricular origin of QRS complexes at rates of 150-250 bpm and a regular rhythm.
- The QRS complex usually narrows unless a conduction abnormality is present or is functionally induced from the rapid heart rate.
- P waves are not usually seen because they are buried within the QRS complex. A pseudo R prime may be seen in V_1, or pseudo S waves may be seen in leads II, III, or aVF. The onset is abrupt with an atrial premature complex, which conducts with a prolonged PR interval.
- The PR interval may shorten over the first few beats at onset, or it may lengthen during last few beats preceding termination of the tachycardia.
- Abrupt termination occurs with a retrograde P wave, sometimes followed by a brief period of asystole or bradycardia.

EP Studies

- EP studies for SVT generally require the placement of several multipolar electrode catheters via peripheral veins.
- In most cases, catheters are placed in the high RA, the His bundle region, the coronary sinus, and the RV apex.
- The purpose of the coronary sinus catheter is to monitor activation of the LA and LV.
- Most venous access is obtained via the femoral veins. In some instances, the subclavian, internal jugular, or antecubital vein may be used for the coronary sinus catheter.

More About EP

- The ablation catheter must be positioned on the mitral annulus to ablate left-sided accessory pathways.
- This may be achieved by a retrograde aortic approach or transseptal catheterization of the LA.
- It is the relative timing of electrical activation recorded from the different electrodes that is critical in determining the mechanism of an arrhythmia and the location of its critical components.

EP Study for AVNRT

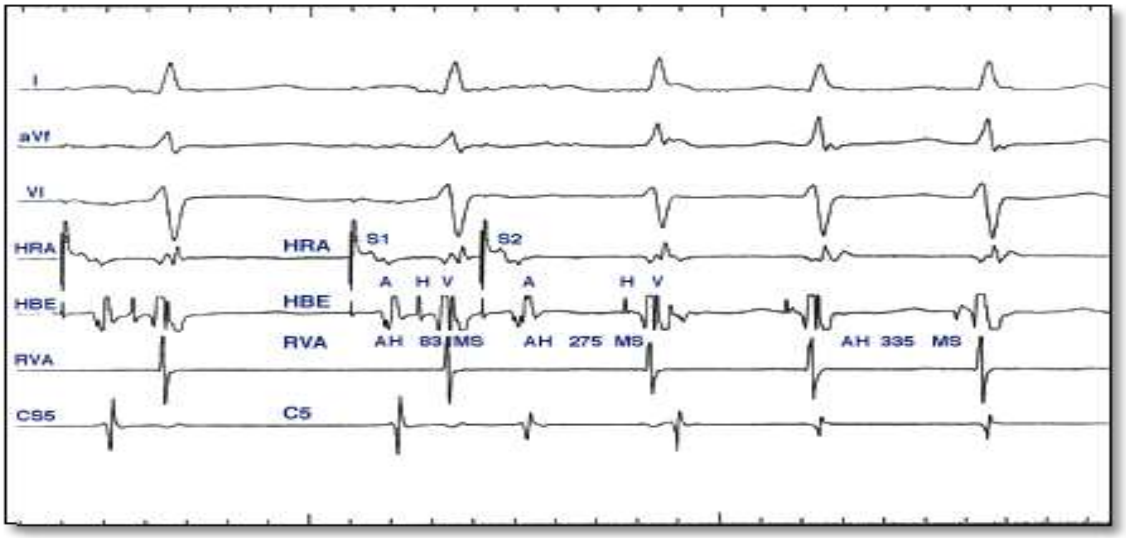
- In sinus rhythm, the fast pathway conducts preferentially through the AV node.
- AV nodal re-entrant tachycardia may be induced by an atrial extra stimulus when the fast pathway is still refractory. Under these circumstances, conduction to the ventricle may occur via the slow pathway if it has regained excitability. If conduction is sufficiently slow, the tissue comprising the fast pathway may recover and conduct the wavefront of activation back to the atria. Repetitive re-entry is established if the slow pathway is able to conduct this wavefront again.

EP Study AVNRT

- Thus, the typical form of AV node re-entry conducts antegradely over the slow pathway and retrogradely over the fast pathway. Activation of the ventricles occurs nearly simultaneously with the atria. Medications or interventions designed to affect conduction in these tissues can abolish the tachycardia.
- Because the His bundle is not a critical component of this circuit, it is possible (though very unusual) for AV node re-entry to persist with 2:1 conduction in His bundle. There is an atypical or uncommon form of AV node re-entry that utilizes the same circuit with the opposite sequence of activation. Antegrade conduction is observed over the fast pathway, and retrograde conduction utilizes the slow pathway. In these cases, activation of the atria occurs much later and corresponds in time with the middle-to-latter half of the R-R interval.

ECG leads I, aVF, and V_1 and intracardiac electrograms from the HRA, HBE, RVA, and CS recording the last two beats (S_1) of an eight-beat pacing drive, a programmed atrial extrastimulus (S_2), and AV nodal re-entrant supraventricular tachycardia (SVT). During S_1, AV conduction is through the fast pathway (AH 83 msec). In response to S_2, conduction blocks in the fast limb and occurs through the slow pathway (AH 275 msec) with re-excitation of the fast pathway and initiation of SVT characterized by simultaneous activation of the atria and ventricles.

A = atrial electrogram; HBE = His-bundle electrogram; V = ventricular electrogram; HRA = high RA; RVA = RV apex; CS = coronary sinus



EP Study Criteria for AVNRT

- The essential criteria for the diagnosis of typical AV nodal re-entry are:
  - 1) a critical prolongation in the AH interval that initiates the tachycardia
  - 2) earliest atrial activation in the His-bundle electrogram
  - 3) activation of the atria within 60 msec of the onset of the surface QRS complex

AVNRT - Review

- Extremely short RP interval
- Valsalva-like maneuvers terminate
- RFA targets slow pathway & is highly effective for eliminating episodes
- Junctional rhythm often occurs during successful ablation

Accessory Pathway Related Tachycardia

- Most common is reentrant - either orthodromic or antidromic AVRT.
- When accessory pathways conduct antegradely, the ECG is preexcited and this is a manifest pathway.
- In a concealed pathway, can only conduct retrogradely.
- Preexcitation: PR interval is short & initial deflection of QRS is abnormal & slurred

Orthodromic AVRT

- Most common symptomatic arrhythmia associated with accessory pathway (90% of arrhythmias in pts with WPW syndrome).
- Antegrade limb is AV node & normal His-Purkinje system, and an accessory pathway that conducts retrograde.
- Either PAC or PVC can incite this tachycardia.
- Termination may by result of AV nodal conduction “fatigue”, increased vagal tone from a vagal maneuver, or a premature extra systolic beat.

Orthodromic AVRT

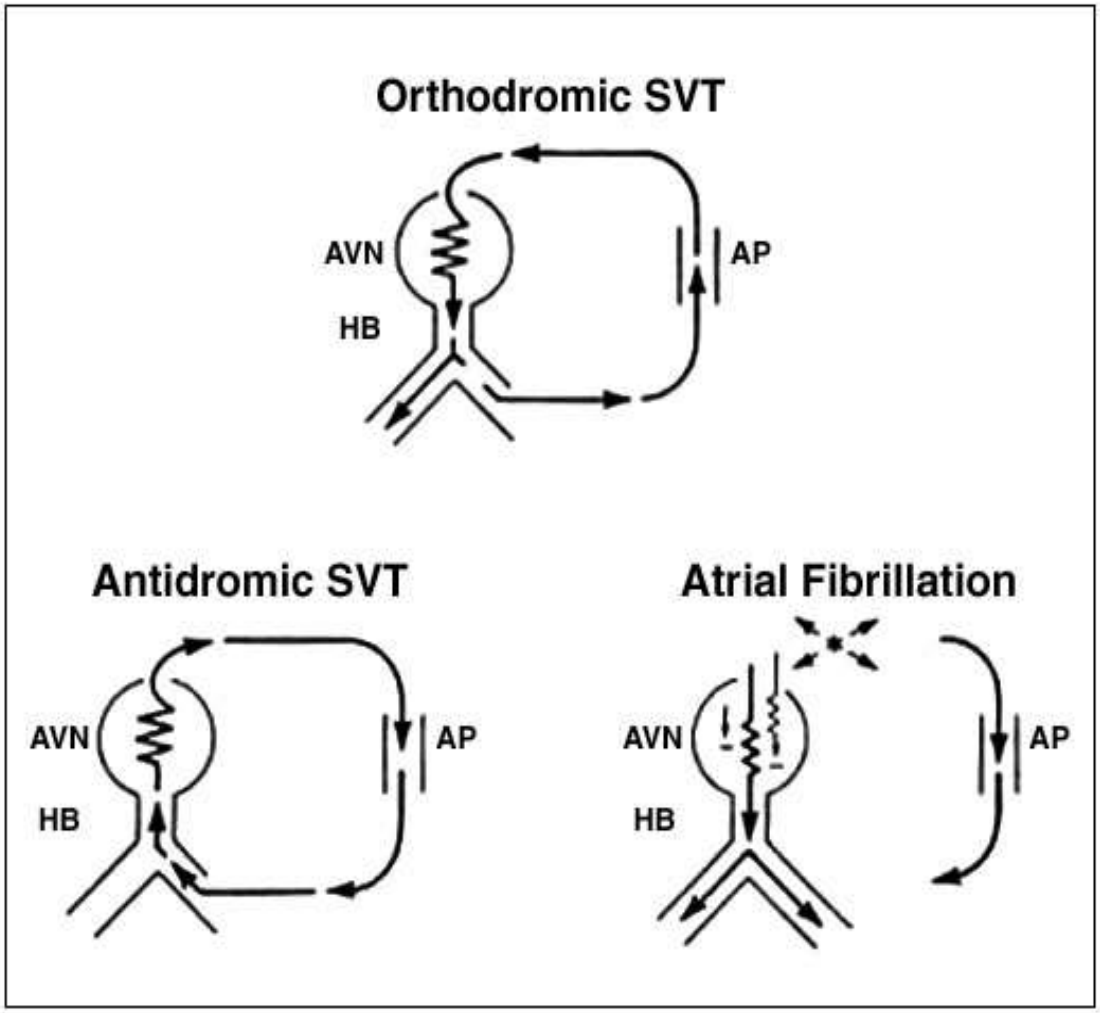
- Orthodromic AV re-entry is a macrore-entrant circuit involving the atria, normal AV conduction system, ventricles, and the accessory pathway.
- During orthodromic AV re-entry, antegrade conduction occurs through the AV node/His Purkinje system.
- Following activation of the ventricles, the wavefront of excitation continues retrogradely through the accessory pathway to the atria.
- The arrhythmia is terminated if conduction is blocked in either the AV node or the accessory pathway.

Orthodromic AVRT

- Orthodromic AVRT, a finite interval has to elapse between activation of ventricle by way of AV node & travel of electrical wave front through ventricle & back to atrium through accessory pathway.
- This interval is almost never less than 100mS.

Antidromic AVRT

- Antegrade limb of this circuit is accessory pathway. This is rare tachycardia.
- B/c earliest site of ventricular activation is ventricular myocardium instead of normal conduction system, the QRS complex is wide and maximally preexcited.
- Drugs that inhibit AV nodal conduction & vagal maneuvers with terminate this tachycardia also.


Orthodromic SVT
AVN
HB
AP
Antidromic SVT
AVN
HB
AP
Atrial Fibrillation
AVN
HB
AP

Embryology of WPW

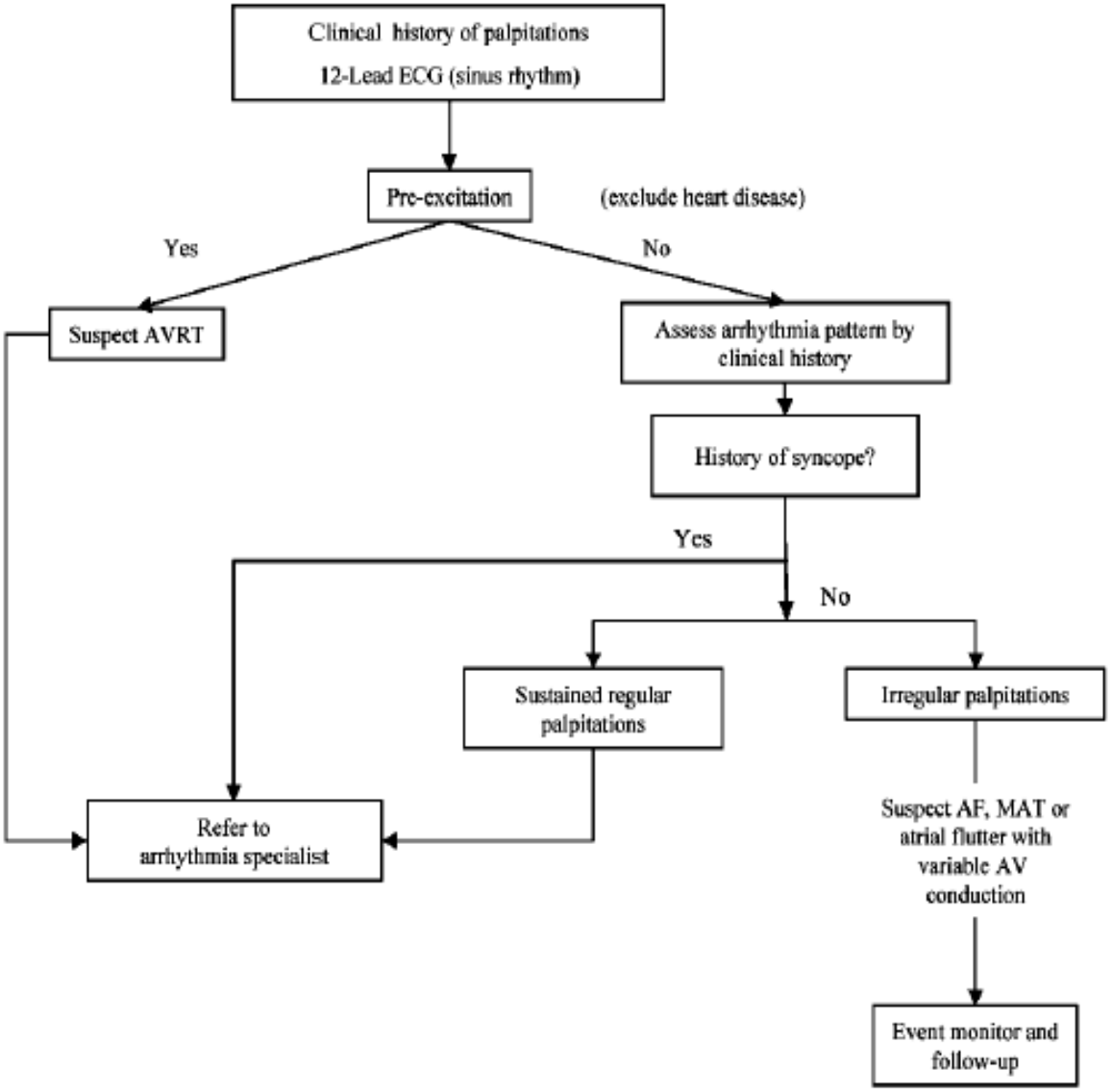
- During cardiac embryogenesis, the atrial and ventricular myocardium are contiguous. The subsequent invagination of the atrial and ventricular septa and formation of the annulus fibrosis normally sever all AV connections except for the AV node/His bundle.
- Persistent strands of myocardium that bridge the annulus fibrosus are the anatomic substrate that causes WPW syndrome. These fibers, which have been termed “accessory pathways,” may be located on either side of the heart or within the septum.
- Accessory pathways may be capable of conducting antegradely from the atrium to the ventricle and retrogradely from the ventricle to the atrium or both.

Preexcited ECG

- In sinus rhythm, every ventricular activation is fusion between accessory & “normal” AV nodal conduction.
- B/c AV nodal conduction usually slower, initial portion of QRS reflects this abnormal ventricular activation (delta wave).
- RBBB pattern is seen in left-sided (+ R wave in V1) & LBBB pattern seen in right-sided (QS complex in V1) accessory pathways.

More Accessory Pathway

- Accessory pathways are referred to as manifest or concealed based on whether they are evident on ECGs recorded during sinus rhythm
- When sinus rhythm is present in patients with manifest accessory pathways, the ventricles are activated through both the normal conduction system and the accessory pathway
- In contrast to manifest accessory pathways, concealed accessory pathways conduct retrogradely, but not antegradely.
- Ventricular activation is normal in patients with concealed accessory pathways, so the ECG never demonstrates ventricular pre-excitation.

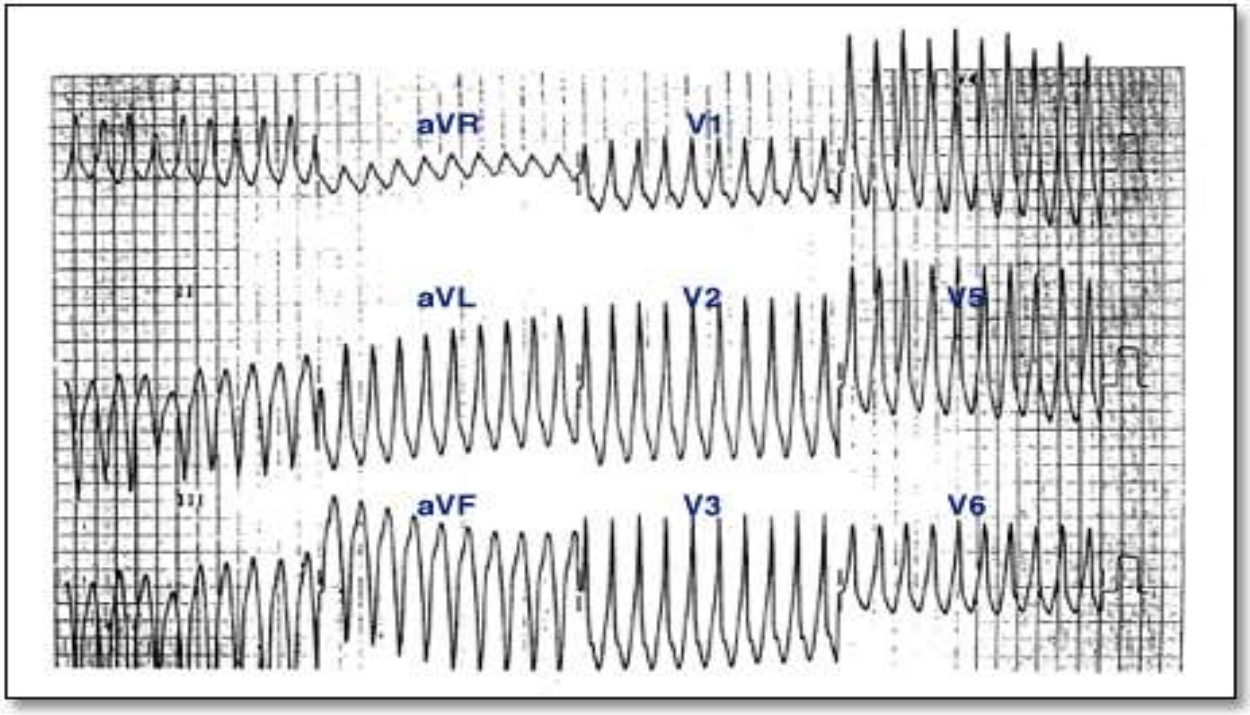

Clinical history of palpitations
12-Lead ECG (sinus rhythm)
Pre-excitation
(exclude heart disease)
Yes
No
Suspect AVRT
Assess arrhythmia pattern by clinical history
History of syncope?
Yes
No
Sustained regular palpitations
Irregular palpitations
Refer to arrhythmia specialist
Suspect AF, MAT or atrial flutter with variable AV conduction
Event monitor and follow-up

SVT with Wide QRS Complex

- SVT's can present with wide complex QRS if
  - 1. Bundle branch block exists during tachycardia
  - 2. Antegrade conduction by way of accessory bypass tract

ECG During Antidromic AV Re-Entry

The QRS complex shows maximal pre-excitation, and the R-R intervals are regular.



Wide QRS b/c BBB

- Wider QRS may obscure retrograde P wave in very short RP tachycardias (like AVNRT)
- Differentiation from ventricular tachycardia can be very difficult, are some guidelines to assist.

Wide QRS b/c Antegrade Preexcitation

- “Mirror image” of orthodromic AVRT may occur, called antidromic tachycardia.
- In this cause, antegrade conduction is by accessory pathway & retrograde conduction is through AV conduction system.
- Ventricular activation occurs directly into ventricular myocardium & bypasses specialized conduction tissue, QRS is therefore wide.

Wide QRS b/c Antegrade Preexcitation

- B/c most accessory pathways insert into base of heart, the QRS vector goes from base to apex - results in concordant + QRS complexes in anterior chest leads.
- This tachycardia is regular, AV nodal blocking agents terminate this arrhythmia.

Wide QRS b/c Antegrade Preexcitation

- Second circumstance where this occurs is when primary arrhythmia is not dependent on accessory pathway, but conducts to ventricle via the accessory pathway.
- Example: atrial fibrillation that is conducting to ventricles via accessory & AV node results in wide QRS complex tachycardia.
- AV nodal blocking agents DO NOT terminate this arrhythmia, will give greater degree of preexcitation.

Wide QRS Complex

- Orthodromic AVRT: antegrade conduction over AV node & retrograde over accessory pathway: results in narrow complex tachycardia unless bundle branch block exists
- Antidromic AVRT: antegrade conduction over accessory pathway & retrograde through AV node: results in wide complex tachycardia

EP Study for Preexcitation

- The diagnosis of ventricular pre-excitation is based on detection of delta waves and a short HV interval during sinus rhythm.
- Ventricular pacing demonstrates earliest retrograde atrial activation at the site of the accessory pathway that differs from activation by the His bundle.

EP Study for Preexcitation

- The criteria for orthodromic AV re-entryinclude:
  - 1) antegrade conduction through the AV node/His bundle
  - 2) eccentric retrograde atrial activation through the accessory pathway
  - 3) pre-excitation of the atria during SVT without a change in the activation sequence by premature ventricular extrastimuli introduced at a time when the His bundle is refractory
  - 4) an increase in the VA interval with bundle branch block ipsilateral to the accessory pathway.

EP Study for Preexcitation

- The criteria for diagnosis of antidromic AV re-entry include:
  - 1) eccentric ventricular activation with a QRS morphology identical to that obtained during atrial pacing at a cycle length that produces maximal pre-excitation
  - 2) a 1:1 relationship between the atria and ventricles
  - 3) demonstration that the ventricle is an essential component of the re-entrant circuit, by terminating the tachycardia with a premature ventricular extrastimulus without depolarizing the His bundle or atria
  - 4) demonstration that the sequence of retrograde atrial activation during ventricular pacing is identical to that during the tachycardia.

Atrial Tachycardia

- Includes various conditions such as automatic AT, macroreentrant AT, scar-related AT, atrial flutters.
- Automatic AT usually presents with long RP tachycardia.
- A single site located anywhere in atria exhibits inherent automaticity at a cycle length shorter than that of sinus node.
- P wave morphology pattern depends on exact site of origin.
- AV nodal blocking agents do not terminate the tachycardia.

Atrial Tachycardia

- With automatic AT, symptoms are often more gradual and get rapid over time.
- Offset also gradual.
- Pts with AT sometimes find a particular maneuver or position provokes symptoms.
- Pts can tap out a very regular rhythm.

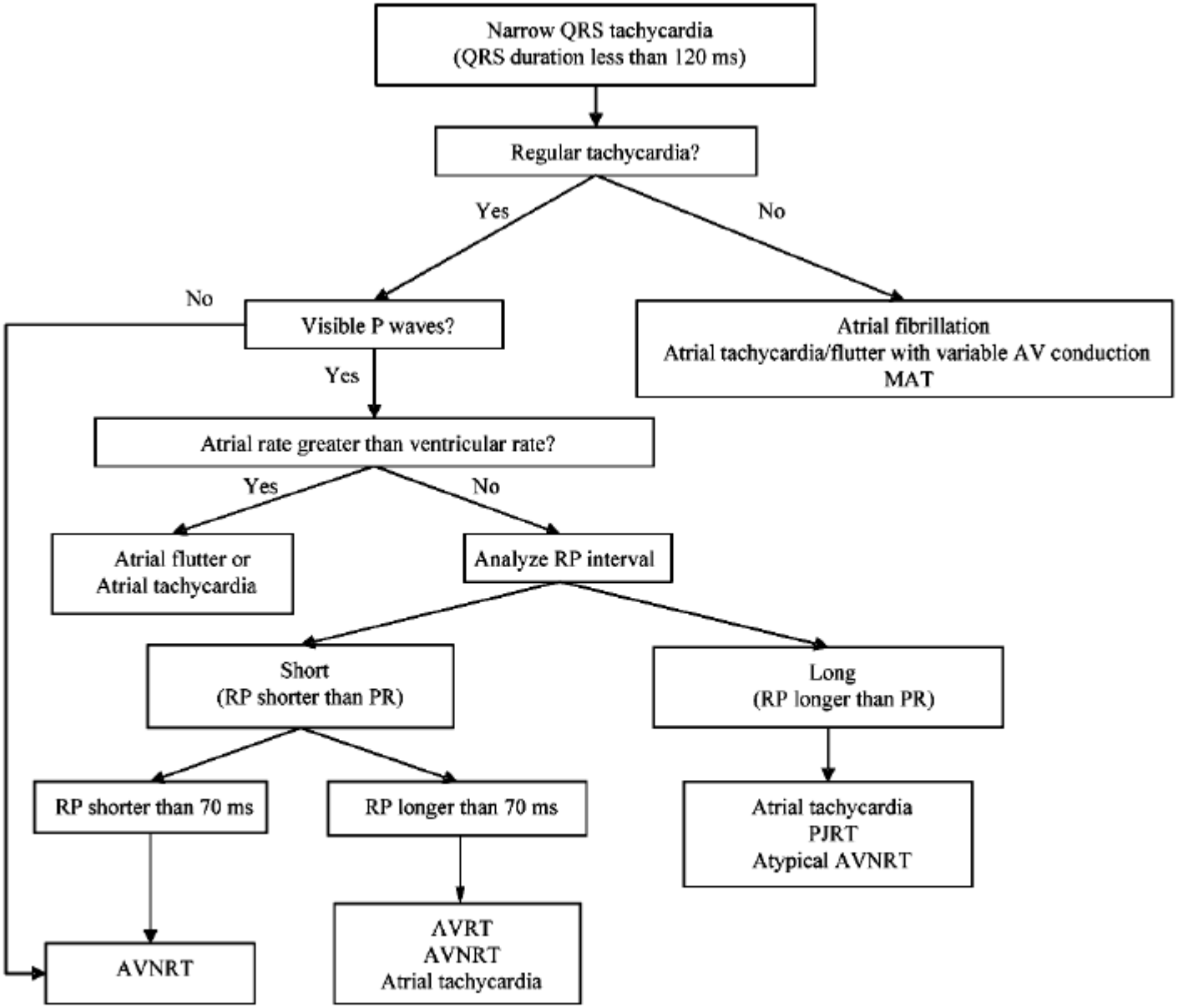
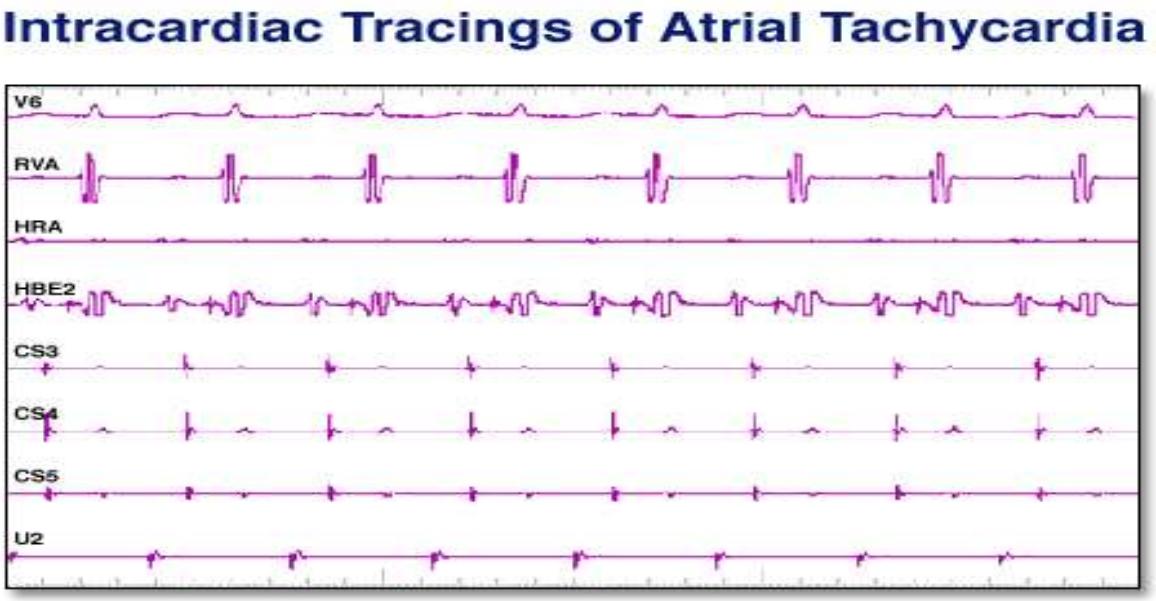


Figure 3. Differential diagnosis for narrow QRS tachycardia. Patients with focal junctional tachycardia may mimic the pattern of slow-fast AVNRT and may show AV dissociation and/or marked irregularity in the junctional rate. AV indicates atrioventricular; AVNRT, atrioventricular nodal reciprocating tachycardia; AVRT, atrioventricular reciprocating tachycardia; MAT, multifocal atrial tachycardia; ms, milliseconds; PJRT, permanent form of junctional reciprocating tachycardia; QRS, ventricular activation on electrocardiogram.

An example of atrial tachycardia, with earliest atrial activation at the mapping catheter (U2), which was located in the low lateral RA. Ablation at this site eliminated the tachycardia. From top to bottom are ECG lead V_6 and intracardiac tracings from the RV apex (RVA), high RA (HRA), distal His bundle (HBE2), proximal to distal coronary sinus (CS3-5), and low lateral RA.



EKG example of AT

Algorithm - Most likely scenarios

- P waves seen within or just after QRS: AVNRT. B/c of very short RP with AVNRT, P wave may give a deflection at end of QRS, giving appearance of incomplete RBBB in V1.
- Short RP tachycardia, but P waves 100mS or more after QRS: orthodromic AVRT.
- Long RP tachycardia: AT.

Differential Diagnosis of Wide-Complex Tachycardia

- VT
- SVT with aberrancy (atrial fibrillation/flutter)-i.e. BB Block
- Antidromic AV reentry –i.e. antegrade via WPW accessory pathway
- Atrial fibrillation, atrial flutter, atrial tachycardia, or AV nodal reentry in setting of WPW with rapid conduction down accessory pathway that is activated as a bystander-I.e. not an integral part of the circuit.

Brugada Criteria for Dx of VT

- Lack of an RS complex in the precordial leads
- Whether the longest interval in any precordial lead from the beginning of the R wave to the deepest part of the S wave when an RS complex is present is greater than 100 ms
- Whether atrioventricular dissociation is present
- Whether both leads V1 and V6 fulfilled classic criteria for ventricular tachycardia.

Major Features in DDx of SVT with aberrancy vs VT

- Supports SVT
  - Slowing or termination by vagal tone
  - Onset with premature P wave
  - RP interval <100mS
  - P & QRS rate & rhythm linked to suggest ventricular activation depends on atrial discharge
  - Long-short cycle sequence
- Supports VT
  - Fusion beats
  - Capture beats
  - AV dissociation
  - P & QRS rate & rhythm linked to suggest that atrial activation depends on ventricular discharge
  - “compensatory” pause
  - L axis deviation
  - QRS duration >140mS

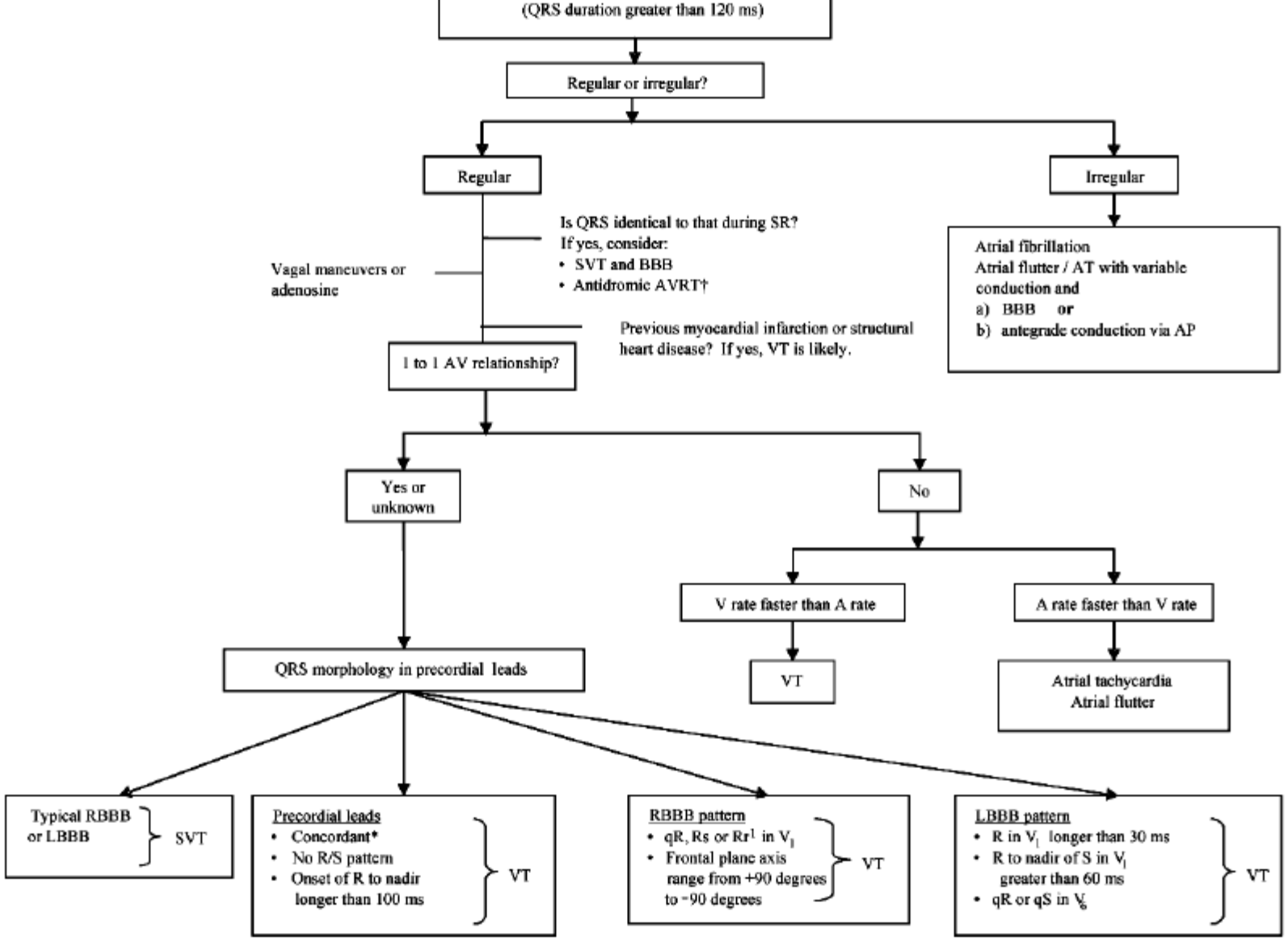


Figure 7. Differential diagnosis for wide QRS-complex tachycardia (greater than 120 ms). A QRS conduction delay during sinus rhythm, when available for comparison, reduces the value of QRS morphology analysis. Adenosine should be used with caution when the diagnosis is unclear because it may produce VF in patients with coronary artery disease and AF with a rapid ventricular rate in pre-excited tachycardias. Various adenosine responses are shown in Fig. 6. *Concordant indicates that all precordial leads show either positive or negative deflections. Fusion complexes are diagnostic of VT. †In pre-excited tachycardias, the QRS is generally wider (ie, more pre-excited) compared with sinus rhythm. A indicates atrial; AF, atrial fibrillation; AP, accessory pathway; AT, atrial tachycardia; AV, atrioventricular; AVRT, atrioventricular reciprocating tachycardia; BBB, bundle-branch block; LBBB, left bundle-

Response to Adenosine

- The administration of adenosine during SVT may be a useful diagnostic as well as therapeutic intervention.
- Adenosine may unmask atrial flutter when the diagnosis is not readily apparent from the ECG.
- It is particularly effective for treatment of arrhythmias that depend on the AV node, such as AV node re-entry or orthodromic AV re-entry. The response of atrial tachycardias is variable. Sometimes adenosine terminates these arrhythmias and other times it reveals the P-wave morphology by creating AV block.
- It is also used to differentiate VT from SVT with aberrant conduction. VT is rarely affected by adenosine, but SVT will either terminate or be exposed by transient AV block. Adenosine may not have any effect if it is administered slowly through a peripheral vein or if the patient has consumed caffeine.

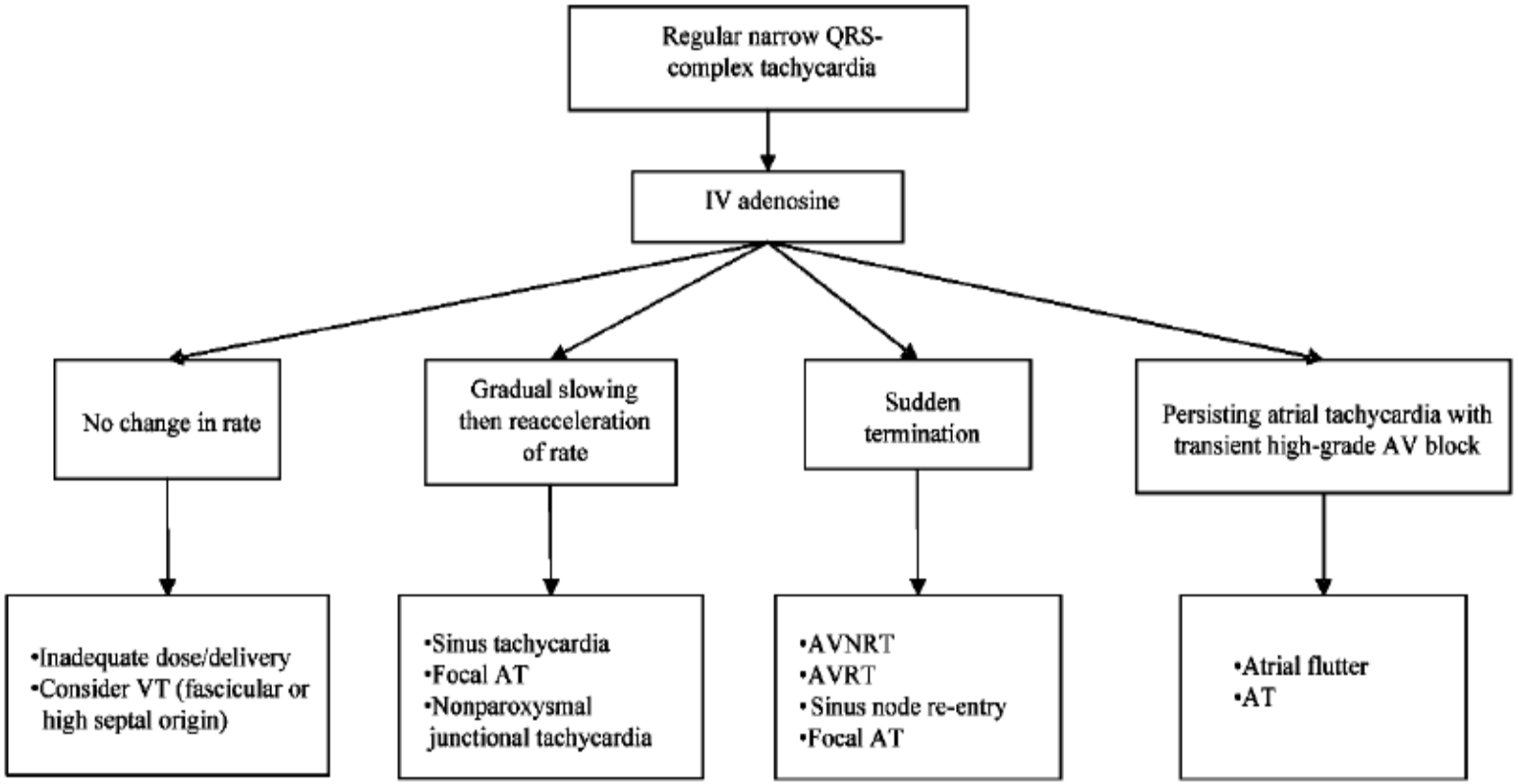


Figure 6. Responses of narrow complex tachycardias to adenosine. AT indicates atrial tachycardia; AV, atrioventricular; AVNRT, atrioventricular nodal reciprocating tachycardia; AVRT, atrioventricular reciprocating tachycardia; IV, intravenous; QRS, ventricular activation on electrocardiogram; VT, ventricular tachycardia.

Ablation

- The diagnostic portion of the EP study identifies myocardial tissue that is critical to the pathophysiology of the arrhythmia.
- A special catheter is positioned at this site, and a small amount of tissue is selectively destroyed by one or more applications of RF energy, which is a high-frequency (500 kHz) alternating electrical current that does not directly stimulate muscle or nerves. The impedance of tissue to electrical current causes an increase in tissue temperature that results in coagulation necrosis.
- Many arrhythmias can be cured by ablating a small focal point of tissue that is critical to the pathophysiology of the arrhythmia. Elimination of accessory pathways in patients with WPW or ectopic atrial tachycardias are examples of problems that can be ablated by a single 60-second application of RF energy.

Risks of Ablation

- 1) cardiac perforation
- 2) vascular injury
- 3) valvular damage
- 4) stroke
- 5) iatrogenic heart block

Questions

- A 28-year-old woman with a history of WPW syndrome comes to the ER with a rapid heart rate associated with shortness of breath, chest discomfort, and near syncope that began 30 minutes ago. She has a history of palpitations that can usually be terminated by performing a Valsalva maneuver, but she was unable to terminate this episode. Her BP is 75/40 mm Hg. She is dyspneic, ashen, and diaphoretic. The ECG shows a rapid, irregular tachycardia that has a rate of 240 bpm with a QRS that has variable morphologies.
- Which of the following is the most appropriate management?
- A. IV digoxin.
- B. IV verapamil.
- C. Esmolol.
- D. IV procainamide.
- E. Immediate sedation and cardioversion

The correct answer is E.

- This patient has a history of WPW syndrome and ECG features characteristic of AF with rapid conduction over an accessory pathway. The variable QRS morphology reflects beat-to-beat differences in the extent of ventricular activation by the accessory pathway. Immediate sedation and cardioversion is most appropriate for patients with severe symptoms and hypotension.
- IV digoxin and verapamil are contraindicated in patients with manifest accessory pathways and AF because they can accelerate the ventricular rate and provoke VF. Esmolol would not slow conduction over the accessory pathway and would lower the BP further. IV procainamide would be likely to block conduction over the accessory pathway and reduce the ventricular rate. It would be a good choice in a patient with mild symptoms who was hemodynamically stable, but it would not be a first choice in this case because it takes about 30

  minutes to administer and would worsen hypotension.

Question

- A 60-year-old woman with a history of palpitations comes to the ER after one hour of symptoms consisting of a rapid heart rate, light-headedness, and mild dyspnea. Her ECG shows SVT with a regular, narrow QRS and a rate of 160 bpm. Transient 2:1 AV block is observed during carotid sinus massage, but the tachycardia is not terminated. It resolved spontaneously before adenosine is administered.
- Which of the following is the most likely cause of the tachycardia?
- A. Atrial tachycardia.
- B. AV nodal re-entry.
- C. Orthodromic AV re-entry.
- D. Antidromic AV re-entry.

The correct answer is A.

- The mechanism of atrial tachycardias does not depend on AV node conduction, so 2:1 AV block could be observed in response to carotid sinus massage without interrupting the tachycardia.
- AV nodal re-entry can persist with block below the His bundle, but this is an exceptionally rare observation. Carotid sinus massage, which affects conduction through the AV node, would either terminate AV node re-entry, cause a transient reduction in rate, or have no effect. Re-entry mediated by accessory pathways and Mahaim fibers depends on a 1:1 relationship between the atria and the ventricles. These possibilities were excluded by the response of 2:1 AV block.

Question

- A 19-year-old man comes to the ER with palpitations, shortness of breath, and light-headedness that began two hours ago. He has had palpitations on several other occasions that are sudden in onset and could be terminated by holding his breath and bearing down. The ECG shows a regular narrow QRS tachycardia with retrograde P waves in the early portion of the ST segment. During monitoring on telemetry, the tachycardia changed from a rate of 200 bpm to 160 bpm when left bundle branch aberration was observed. His arrhythmia is terminated with the administration of adenosine. The ECG recorded during sinus rhythm is normal. Which of the following is the most likely cause of this patient's tachycardia?
- A. AV nodal re-entry.
- B. Atrial tachycardia.
- C. A left-sided concealed accessory pathway mediating orthodromic AV re-entry.
- D. Atrial flutter.
- E. A right-sided concealed accessory pathway mediating orthodromic AV

The correct answer is C

- Orthodromic AV re-entry is characterized by a regular tachycardia with a narrow QRS and a retrograde P wave in the early ST segment. When bundle branch block occurs on the same side as the accessory pathway, the rate of the tachycardia usually becomes slower because the re-entrant circuit becomes longer. In this case, LBBB caused the rate of the tachycardia to decrease, which indicates that the accessory pathway is located on the left side. The accessory pathway is concealed because pre-excitation was not observed during sinus rhythm.
- The rate of AV nodal re-entry would not be affected by bundle branch block, and the P wave is usually not visible because it is buried within the QRS complex. Atrial tachycardia and atrial flutter are generally not terminated by Valsalva maneuvers, often persist when adenosine is administered, and the rate is not affected by bundle branch block. The tachycardia would not have become slower with LBBB if the accessory pathway were located on the right side of the heart.

Question

- An 8-year-old boy with severe asthma develops SVT after using his inhaler. He comes to the ER because he cannot terminate the arrhythmia with Valsalva maneuvers. His heart rate is 220 bpm, with a BP of 110/70. Marked wheezing is heard on auscultation of the chest. The ECG shows SVT with a narrow QRS. No P waves are evident in the recording. Carotid sinus massage fails to terminate his arrhythmia.

- Which of the following is the most appropriate management?
- A. Adenosine.
- B. IV metoprolol.
- C. IV diltiazem.
- D. High-dose oral loading with propafenone.
- E. Immediate cardioversion.

The correct answer is C.

- IV diltiazem is an effective treatment for re-entrant SVT that includes the AV node in its circuit. The description of the ECG is characteristic of AV node re-entry, which responds well to diltiazem.
- Adenosine can provoke bronchospasm in patients with severe reactive airway disease. It would be contraindicated in a patient with severe asthma who was wheezing at the time of examination.
- Metoprolol and propafenone would be contraindicated in an asthmatic.
- Immediate cardioversion would not be the first choice in a hemodynamically stable patient.

Question

- A 76-year-old man with CAD, heart failure, and chronic renal failure has recurrent SVT despite treatment with beta-blockers and calcium channel blockers. He declines to undergo an EP study for further evaluation and treatment of this problem. His arrhythmia occurs several times during dialysis and causes hypotension.
- Which of the following is the most appropriate pharmacotherapy?
- A. Procainamide.
- B. Amiodarone.
- C. Flecainide.
- D. Sotalol.
- E. Propafenone.

The correct answer is B.

- Although amiodarone is not approved for treatment of supraventricular arrhythmias, it is commonly used for this purpose. It is the appropriate choice for selected patients. Low doses of amiodarone are very effective for treatment of SVT, and the risk of adverse effects is acceptable in a patient this age.
- Procainamide has a high incidence of GI side effects and drug-induced lupus. It prolongs repolarization and has a 1-3% incidence of torsade de pointes. Although it can be used in patients with renal failure by adjusting the dosage and monitoring levels, it is not as effective as amiodarone and is more difficult to use in patients with renal failure.
- Flecainide and propafenone are contraindicated in patients with CAD and heart failure because of their negative inotropic and proarrhythmic effects. Dosage adjustment is required in patients with renal failure because they are excreted by the kidneys.
- Sotalol is a negative inotrope and must be used cautiously in patients with heart failure. It is also cleared by the kidneys, which requires careful dosage adjustment and monitoring in patients with renal failure to avoid excessive QT prolongation and induction of torsade de pointes.