

Supraventricular Tachycardias

Abnormal circulating impulse in a relatively normal heart, causing a regular narrow-complex tachycardia.

FACTOIDS:

Incidence is 35 per 100,000
Prevalence is 2.5 per 1000

SYMPTOMS:

- syncope
- dizziness, light-headedness
- palpitations
- shortness of breath
- panic, anxiety

SIGNS:

- Ridiculous fast pulse rate
- Sometimes pallor
- Most of the time, no signs.

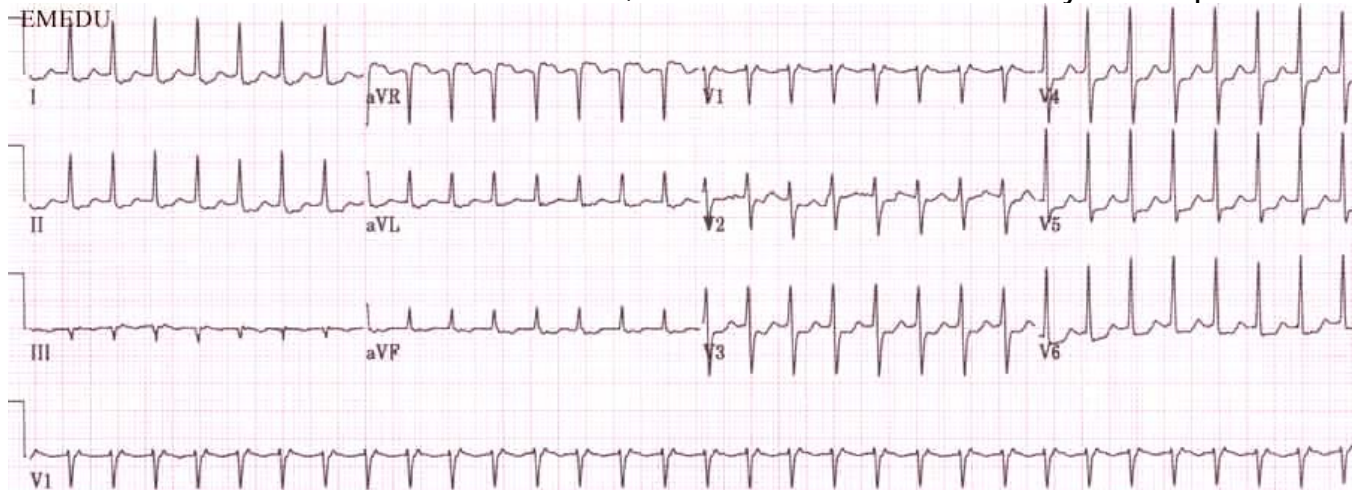
ASSESS IMMEDIATELY: is this patient hemodynamically stable? Are they crumbly enough to crash right now? Do they have a crappy heart already?

Associated historical evidence:

- This patient is a coffee glutton, cocaine fiend, or otherwise given to excess with stimulants
- Alcohol consumptions is a risk factor- causes both atrial arrhythmias and SVT
- Hyperthyroidism

ECG findings: by the time you saunter up to the patient, an ECG is usually ready for you to look at.

- 90% will have a NARROW QRS COMPLEX, with a rate of about 200. There may be ST depression.



Investigating the cause: to consider later...

- CHEST XRAY to look for gross abnormalities, eg. hugely dilated heart
- ECHOCARDIOGRAPHY to look for structural heart disease in detail
- ELECTROPHYSIOLOGY is usually something you do with a view to ablate an accessory pathway

In the emergency department, you will find yourself ordering the following:

- ECG
- Chest X ray
- CK, Troponin
- EUC and CMP
- TFT

You will give them oxygen.

DIAGNOSIS: Which narrow-complex tachycardia is it?

So, you wander up to the bedside, following the frantic beeping of the ECG monitor.

IS THE PATIENT STABLE? Or has the blood pressure suddenly dropped, and they are unconscious?

- Narrow complex tachy? Stop diagnosing them and give a DC shock.

So maybe they are stable. Is the rhythm regular?

ADENOSINE

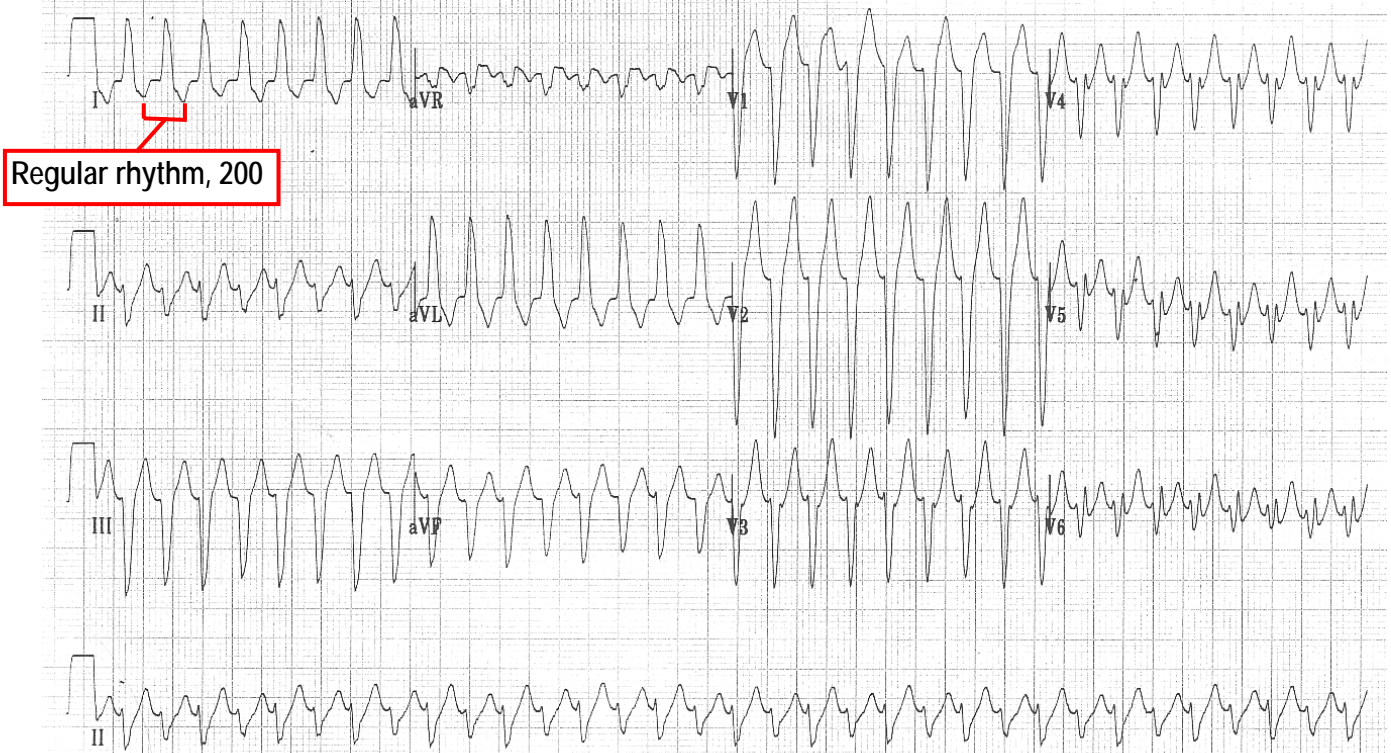
Should be OK; with an accessory pathway the chances of AF with 1:1 conduction are tiny; ...but... have the crash trolley parked at the end of the bed anyway. Chance favors the prepared man.

- Irregular rhythm? Can you see any P waves?
 - If not, its probably ATRIAL FIBRILLATION.
 - If you see any P waves, they are likely to have many weird morphologies, and so this is likely MULTIFOCAL ATRIAL TACHYCARDIA; this happens to people with huge dilated atria.
 - Regular rhythm? Detectable P waves?
 - Cant make out those P waves? Try adenosine. You will either convert it to sinus OR you will slow the ventricular rate to get a good look at the P waves.
- Or, if its WPW, you may induce a fatal VT or VF with adenosine. RARELY.

NOW THAT YOU CAN SEE SOME P WAVES:

- Atrial rate of 250, sawtooth pattern? Its FLUTTER.
- Totally normal P waves?? Is got to be a SA node reentrant tachycardia.
- Abnormal (maybe inverted) P waves and prolonged RP interval? It has to be an ATRIAL TACHYCARDIA, i.e. some ectopic focus sending impulses which are conducted 1:1
- RETROGRADE P waves and SHORT RP interval? Its an AV reentry tachycardia with an accessory pathway, or AV nodal reentrant tachycardia.
- Retrograde Ps and a LONG RP interval? – its AV reentry tachy with a slow-conducting accessory pathway
- NO ATRIAL ACTIVITY?? It was probably atrial fibrillation, or an ectopic junctional tachycardia..

Here's one from real life. pt. hemodynamically stable, conscious, and known to have a "silent" reentry pathway, which was investigated with EPS only a day ago - but not ablated owing to its proximity to the AV node.



The above arrhythmia was terminated with a Valsalva manoeuvre in emergency. Despite having an accessory pathway, the pt. reported this being managed with adenosine in the past, with good effect.

MANAGEMENT

- If they are unconscious because of their SVT, you need to DC-shock them immediately.
- Otherwise, in an otherwise well person, you are permitted to try any number of measures:

VAGAL MANOEUVRES: the idea is to increase vagal tone. Increased parasympathetic input will slow conduction through the AV node; acetylcholine causes an increased permeability to potassium which leaks out of the conducting fibers, taking positive charge with it and thus hyperpolarizing them.

- **carotid massage:** one side at a time
- **Valsalva manoeuvre:** exhale against a closed epiglottis, like clearing "popped" ears
- **Ice pack on the face:** works best in children
- **Precordial Thump:** a dangerous and out-of-date practice. May induce arrest. May be hilarious.

DC CARDIOVERSION

Cardioversion vs. defibrillation: cardioversion is synchronized to the QRS complexes, whereas defibrillation just intrudes rudely and randomly into the electrical activity of the heart.

THE THEORY: when you zap someone, you suddenly depolarize the whole of the excitable muscle, and that includes the aberrant reentry circuit. Once you depolarize everything, you put the whole of the myocardium into a refractive period, and so the next thing to fire should be the normal SA nodal pacemaker, kickstarting a normal sequence of conduction.

PHARMACOLOGICAL MEASURES: because the vagal manoeuvres only work 25% of the time

Mighty ADENOSINE – both diagnostic and therapeutic value

Antidromic SVT is much less common than orthodromic, even in accessory pathway scenarios.

Causes a transient AV node block. The node just stops working for a few seconds. Mediated via the A1 receptor, adenosine inhibits adenylyl cyclase, reducing cAMP and thus causing potassium to flood out of the cell, reducing its positive ion content and thus hyperpolarizing it. This is basically what the vagus nerve does, but it bypasses the whole acetylcholine business. Interestingly, these are the same receptors which caffeine binds to.

- Given 6 to 8mg IV, VERY QUICKLY (in 2 seconds) because it gets metabolized very quickly in the bloodstream.
- If ineffective the first time, give a larger dose (12 to 16mg)
- 90% of SVT will be terminated by the second larger dose.
- Just make sure resus equipment is around, and you're confident in intubation.
- In instances of atrial flutter, adenosine will block conduction long enough for you to get a good flutter trace in lead V1, thereby confirming the diagnosis of flutter.

CONTRAINDICATIONS:

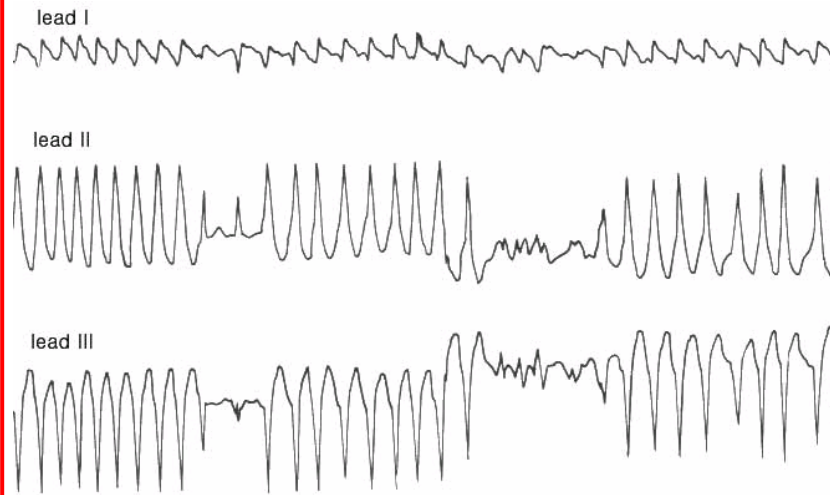
- **ACCESSORY PATHWAY!** Adenosine also shortens the refractory period. It can induce atrial fibrillation. If you have WPW or something similar, adenosine might induce ventricular fibrillation. THIS IS VERY RARE.

Most senior ED staff just give adenosine to WPW anyway. You can always bring out the cardioverter and zap them.

- Asthma or severe COPD (may cause bronchospasm)
- Sick sinus syndrome
- Broad complex tachycardia

So you used adenosine anyway. After you were told not to. That's just fine. Vast majority of the time you will actually get away with it. Only very occasionally you might induce AF in a patient with an accessory pathway.

This is what happens.. The patient is looking slightly unwell.



Now what are you going to do..

For all intents and purposes this is VF; through technically its AF with 1:1 conduction.

Normal drugs wont work: blocking the AV node just makes the accessory pathway take over more of the conduction role. **The rate would actually increase.**

Two strategies: target the accessory pathway to slow its rate of conduction, or just cardiovert the whole thing and hope the AV node takes over the role of conduction (DC shock should silence ectopic atrial foci). **Procainamide** seems to be the agent of choice in the US. **Antipodes prefer Flecainide.**

This is very rare and requires a DC shock.

The DC current is an effective rescue measure; so in reality... you can always just give adenosine, and if everything goes to hell- there is always the defibrillator.

Second line agents

Verapamil

You wouldn't use any of these in patients with severe hypotension, history of congestive heart failure, WPW or some sort of heart block.

- Calcium channel blocker; blocking the calcium channels will slow the rate of AV node conduction and prolong the AV refractory period. LV function will also be depressed, so this is not any good in people with low LVEF.
- Usually given as a slow bolus of 5-10mg IV, with constant (preferably arterial line) blood pressure monitoring.

Beta-blockers

- **Beta-blockade slows the rate of sinus pacemakers, or any ectopic pacemakers. It also increases the refractory period of the AV node, and it will slow conduction of retrograde or anterograde accessory pathways.**

Third line agents

Class 1a: Procainamide

Class 1c: Flecainide and Propafenone

Fast sodium channel blockers, best for VT; they will lengthen the refractory period of the accessory pathway and sometimes terminate AF.

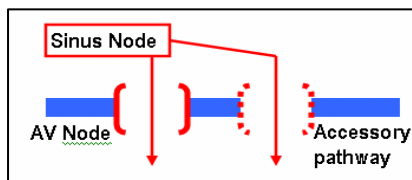
In WPW with atrial fibrillation, these drugs are used if DC cardioversion is impossible.

IN SVT with definite preexcitation, these are SECOND LINE AGENTS.

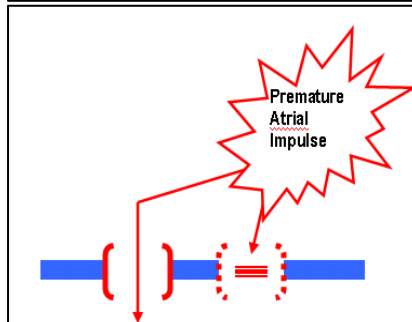
Wolff-Parkinson-White: Verapamil, digoxin and adenosine are said to be contraindicated.

- **DC Cardiovert these people as a first line treatment.**
- **Only if that fails, move onto Class 1c or 1a fast sodium channel blockers**

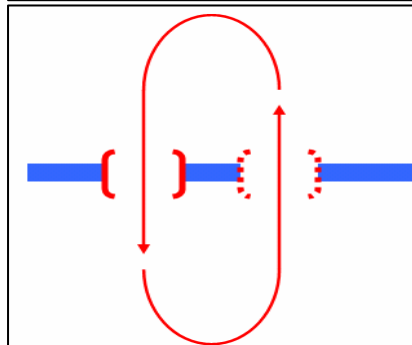
PATHOPHYSIOLOGY OF REENTRY CIRCUITS



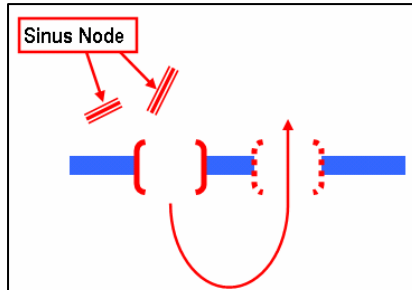
First, the sinoatrial node fires as normal; the impulse travels through both the AV node and the accessory pathway. **Pre-excitation** is the phenomenon of seeing some of the ventricle, triggered by the accessory pathway, start to depolarize before the bulk of it; this causes a **delta wave**.



Then, an accessory pacemaker – some random atrial ectopic – fires a premature P wave, a premature atrial impulse. The accessory pathway is still in the refractory period, because it had just conducted a proper sinoatrial impulse. So it cannot conduct the ectopic premature impulse just yet. This impulse can only be conducted through the AV node.



The ectopic impulse, conducted through the AV node and the ventricle, takes its time to get back to the reentry circuit area: but the time it gets there, the accessory pathway is no longer refractory and can now be excited again. The same for the atrium. The sinoatrial node hasn't fired yet – all this business happens between beats. So the re-entering impulse can once again be conducted via the atria to the AV node, and then to the ventricles, thereby setting up a whole separate (and faster) rhythm.

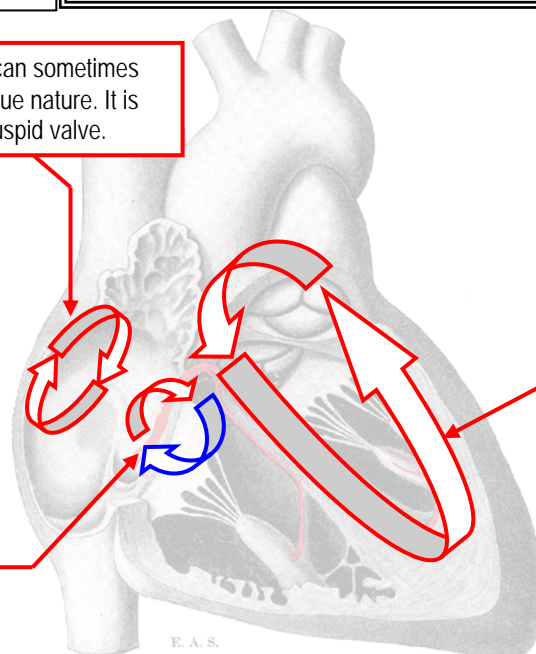


With the conducting system in revolt, the sinoatrial node struggles for control, still firing regularly – but its impulses meet a useless refractory atrial wall, and so its commands are rudely ignored.

Atrial Flutter with 2:1 conduction can sometimes look like SVT; adenosine reveals its true nature. It is usually a large circuit, around the tricuspid valve.

AV nodal reentrant tachycardia: The AV node is normally a one-way street, but in this instance, it becomes two pathways: one **fast**, one **slow**. The slow pathway conducts the impulse to the beginning of the fast pathway; by the time the impulse got there, the beginning of the slow pathway is already ready to receive an impulse. The fast pathway conducts the impulse back up into the beginning of the slow pathway. And so it continues.

60%



"Orthodromic" AV reentrant tachycardia

If you have an accessory pathway, this is your tachycardia. The impulse travels just like in the pictures above: from atrium, to AV node, to ventricles, and back to the atrium via the accessory pathway.

Antidromic tachycardias follow the opposite route: to the ventricles via the accessory pathway, then to the AV node, and then back to the atrium. The QRS complex in these is typically broad, and therefore very similar to a proper ventricular tachycardia.

30%

CAUSES OF SVT

AV nodal reentry circuit – 60% of cases

Another 30% are due to an accessory pathway.

10% are focal atrial causes, ectopic pacemakers of some sort.

RARE SPECIES:

These do not involve the AV node or accessory pathways!

Don't get me wrong: those pathways might exist, but they don't participate in the primary mechanism.

- **Inappropriate Sinus Tachycardia**
 - Poorly understood; seems like something in the SA node has gone wrong. May be a dysautonomic thing, but we just don't know.
 - TOTALLY NORMAL RHYTHM! ECG looks like a sinus tachy; just with a very high rate.
 - Occasionally you may discover an inappropriately increased tachycardia response to exercise or an elevated resting heart rate.
 - Typically, these patients don't have any heart disease or hyperthyroidism, and are not coke fiends.
Treatment is the usual - vagus, adenosine, verapamil... Slow AV conduction and you Slow the ventricular response; increase vagal tone and you slow down the SA node.
- **Sinus node reentrant tachycardia:**
 - This is initiated in much the same way as any other sinus beat; except the sinoatrial node keeps sending impulses. The atria, behaving normally, transmit these to the rest of the heart, and the P wave looks completely normal.
Most of the time this is caused either by a reentry circuit within the SA node itself, or by a reentry circuit which lies just next to the node- either way, the SA nodes' own impulse triggers further impulses.
- rate is usually 100 to 150 bpm. Treatment is the usual - vagus, adenosine, verapamil...
- **Intraatrial reentry tachycardia:**
 - usually initiated with a premature atrial ectopic, and so the P wave looks abnormal. In V1, it will be negatively deflected if it starts in the right atrium, and positively deflected if it starts in the left atrium
 - This sometimes happens after a large atrial incision, in cardiac surgery.
- Treatment, once again, is just the usual.
- **Ectopic Junctional tachycardia and nonparoxysmal junctional tachycardia:**
 - A weird tachycardia, arising from the AV bundle- NOT the AV node! So there's no reentry to speak of. Seems to be a matter of increased automaticity.
 - Occasionally this is the result of digoxin toxicity or a big heart attack.
 - This is also a congenital condition affecting neonates.
- Adenosine is useless: AV node is not involved. Some trials recommend amiodarone.

STRUCTURAL DEFECTS

- **Hypertrophic cardiomyopathy:**
 - These people often get transient and asymptomatic SVT events.
- **Ebsteins' Anomaly:**
 - This is a congenital abnormality, where the tricuspid leaflets are malformed, and there is some degree of "atrialisation" of the right ventricle.
 - 20% of these people end up with an accessory pathway, seeing as the normal fibrous insulation between the atria and ventricles is so disturbed and abnormal.

Etienne Delacrétaiz, M.D. Supraventricular Tachycardia N Engl J Med march 9, 2006;354:1039-51.

Henry Gray (1825–1861). Anatomy of the Human Body. 1918. Fig. 518

Emergency Medicine Education Online <http://www.emedu.org/ecg/givemall.php>

Amiodarone for post-operative junctional ectopic tachycardia Kevin Plumpton, Robert Justo, Nikolaus Haas *Cardiol Young* 2005; 15: 13–18. Also UP TO DATE, of course.

Pharmacologic Therapy of Supraventricular Tachycardia - Bruce B. Lerman, MD

<http://www.hrsonline.org/Education/SelfStudy/Articles/lerman.cfm>

Kister PM, Pharmacological management of tachycardia. *Australian Family Physician* Vol. 36, No. 7, July 2007