put together by Alex Yartsev: Sorry if i used your images or data and forgot to reference you. Tell me who you are. aleksei.igorevich@gmail.com

Wolff-Parkinson-White and Arrhythmias

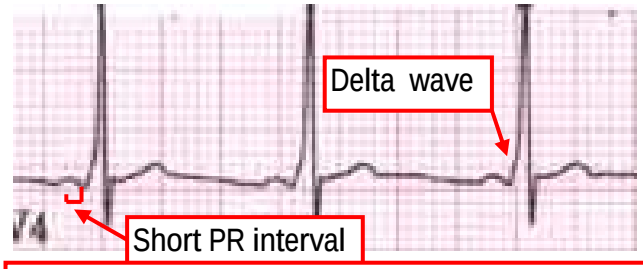
Prevalence is 0.15 to 0.25% of the population; or about 0.5% among first-degree relatives. **Predistposition to arrhythmias favours AV reentrant tachy.** An ectopic atrial beat can produce an orthodromic SVT if it is nearer to the AV node, and an antodromic SVT if it is near the accessory pathway.

The atria are behaving ; they politely conduct the impulse from the SA node along the usual fast conduits. The P wave, therefore, is normal.

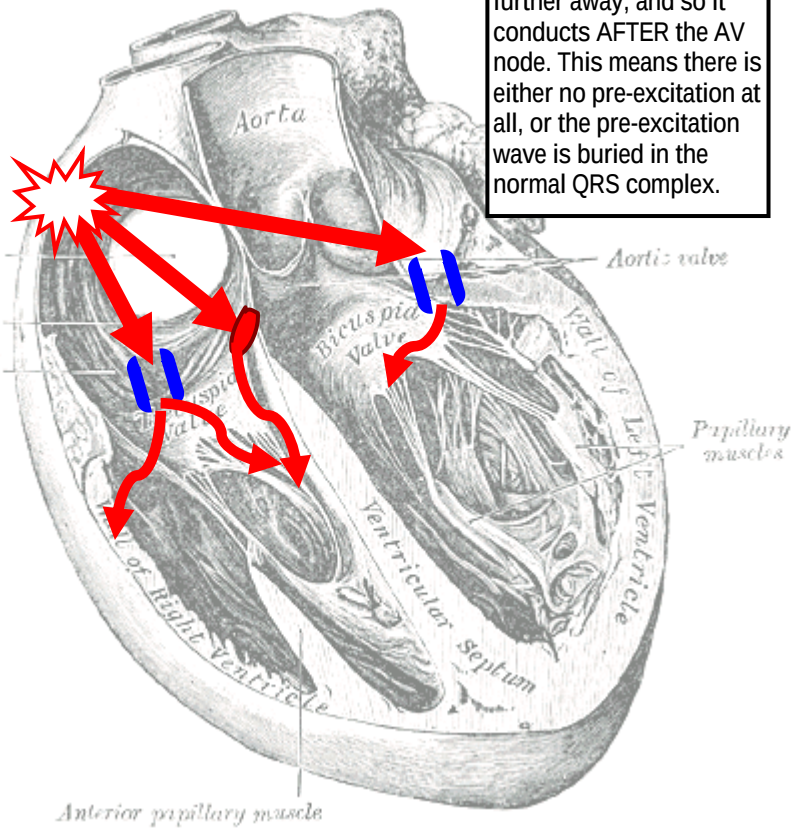
The Bundle of Kent is an accessory pathway which is closer to the SA node. It conducts the impulse first, BEFORE the AV node has a chance.

Because some portion of the ventricle depolarizes BEFORE the rest, the QRS upstroke is "slurred". This is a Delta Wave.

Because the Bundle of Kent is closer to the SA node it will trigger a QRS complex to begin sooner, and so the PR interval is abnormally shortened.



A Left Lateral pathway is further away, and so it conducts AFTER the AV node. This means there is either no pre-excitation at all, or the pre-excitation wave is buried in the normal QRS complex.



WPW and SVT

Remember: if the complexes are narrow, its orthodromic. If they are wide and with delta-waves, its antidromic. Does that really matter? No. Of course not. If you know this patient has WPW, you can give them adenosine fearlessly; VF is rare and you can just shock them anyway, so why mess around with toxic class 1a agents?...

WPW and atrial fibrillation

AF is disturbingly common in WPW- 10 to 30% of patients will have it at some point. Having AVRT predisposes one to AF in this situation because the reentry circuit via the accessory pathway can cause the atria to contract quite randomly (after all, the accessory pathway is not a serious part of the conducting system, and it doesn't link into any sort of conduction pathways- its just going to excite any old patch of atrium).

The ECG will throw you off. The conduction rate is roughly 1:1.5; the QRS rate is about 180 to 200. It is hard to tell that its irregularly irregular. The QRS complexes will be a mixture of pre-excited delta-waving ones, and normal-looking narrow ones. If the accessory pathway has a short refractory period, it will conduct more often and therefore there will be more broad complexes than narrow ones. The shorter the refractory period of the accessory pathway, the broader the QRS. The broader the QRS, the greater the chance of this thing degenerating into ventricular fibrillation.

REMEMBER: the wider the QRS, the more chance of VF.

WPW and atrial flutter

Just like atrial fibrillation, flutter can conduct via the bundle of Kent. There will be 1:1 conduction. Ventricular rate will approach 300. Because this is an antidromic way of conducting impulses, the QRS complexes will be broad and there will be delta waves. Unlike AF, the rate runs with a metronome-like regularity.

WPW and ventricular fibrillation

So, in AF with WPW conduction, the rate of ventricular contraction is increased, and the regularity is decreased. This fractionates the wavefront of ventricular depolarization. Soon enough, there are numerous wavefronts all moving around the ventricle. This is ventricular fibrillation. If you block the AV node, occasionally the accessory pathway will launch the ventricles into this. It's a known, and extremely uncommon, complication of adenosine.