What Is the Diagnosis Here (and Don't Say "SVT") Discussion

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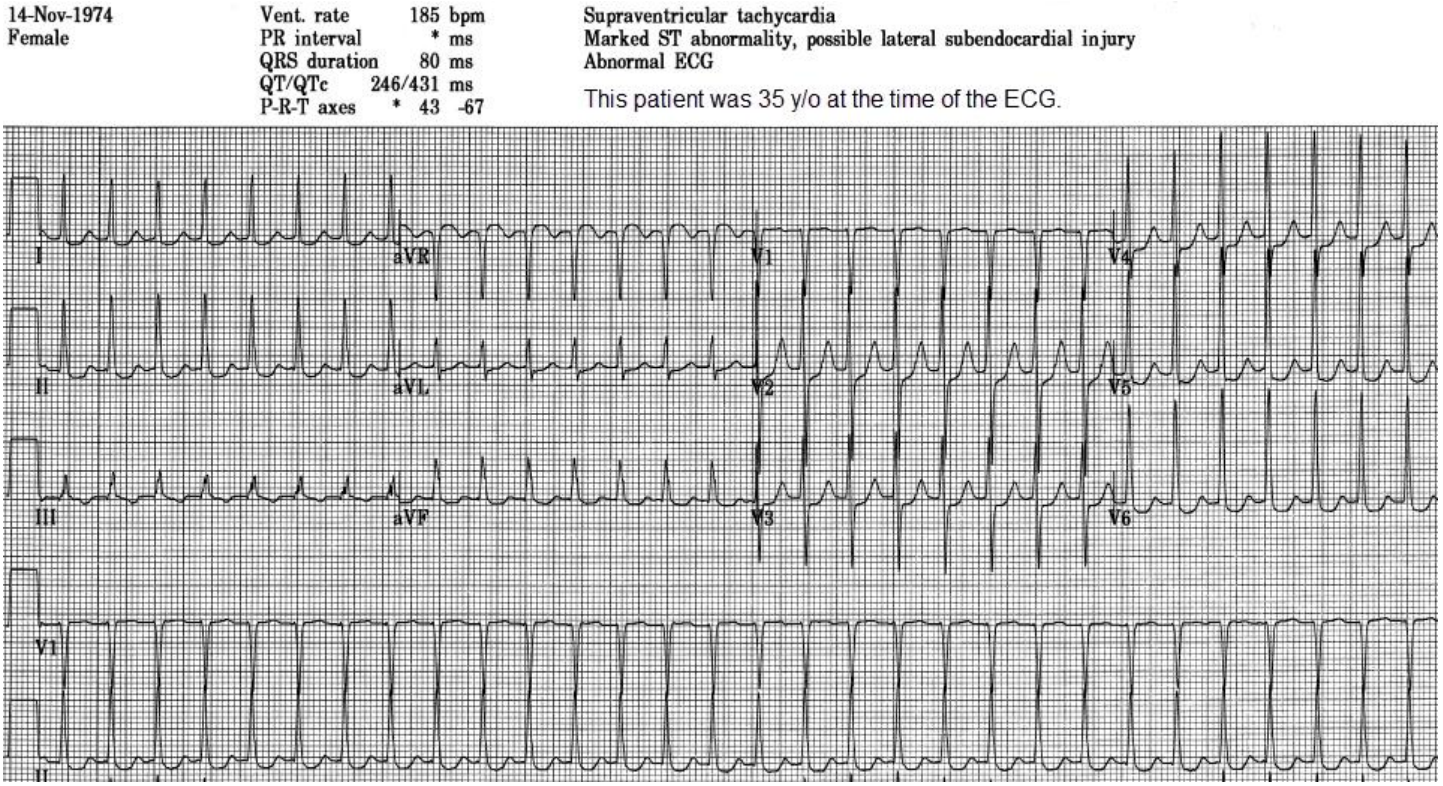

Figure 1

Regarding the **FIRST QUESTION**…

The only one of the three regular, narrow-complex tachycardias that is benign is **AVNRT** – and that's *only* in otherwise healthy subjects. How long could an 85 y/o patient with advanced COPD and coronary vascular disease last with a ventricular rate of 185/min?

AVRT should never be written off as *totally* benign because of the accessory pathway. If the AVRT involves the AV node in the reentry circuit, then it is not as likely to cause major problems for the patient. The AV node will help limit the tachycardia due to its property of decremental conduction. If it proceeds to develop a protective 2:1 block – *that will terminate the AVRT*. The greatest problem arises when there is more than one accessory pathway (not too unusual) which will allow extremely rapid ventricular rates without the protection of the AV node.

But the real danger of the AVRT (and, as you will see, the atrial tachycardia, too) is NOT the development of a *reentrant* circuit but rather the use of the accessory pathway as a *bystander* pathway. It acts simply as an "open door" from the atria to the ventricles. This allows tachydysrhythmias such as atrial fibrillation, atrial flutter and atrial tachycardia to enter the ventricles at very high rates. This will frequently cause the ventricles to fibrillate.

So you see, those three regular, narrow-complex tachycardias are not as benign as you may have thought they are and even though TWO of them can be managed the same way in the ER, they need a DIAGNOSIS – *not* a garbage can!

Regarding the **SECOND QUESTION**…

The ECG machine diagnoses "…possible lateral subendocardial injury." Subendocardial ischemia is non-localizing on the ECG. Therefore, *inferior* subendocardial ischemia or *lateral* subendocardial ischemia and all other attempts to localize the subendocardial ischemia are simply a waste of time. Just say *subendocardial ischemia* and leave it at that. You will have reported the issue and made your point.

Now do NOT confuse the ST depression of *subendocardial ischemia* with the ST depression of a *reciprocal change*. If you don't understand the difference, visit my website to learn more about ***The Masterclass in Advanced Electrocardiography*** which will be presented in **Houston, Texas May 4-7, 2026** and in **Strasbourg, France September 21-24, 2026**. The Strasbourg Masterclass will be conducted in English.

Regarding the **THIRD QUESTION**…

For years we thought that the ST depression that appeared during many regular, narrow-complex tachycardias was caused by a failure to adequately restore the ionic concentrations of the myocytes between each contraction. That seemed intuitive but the question remained: if the ionic balance was disturbed to the point of creating (at times) a very significant ST depression – how was it able to completely disappear within ONE HEARTBEAT following restoration of sinus rhythm?

Now, articles have appeared saying that ionic imbalance was never the cause of the ST depression – the ST depression is an illusion created by the presence of the atrial T wave (the Ta wave)! But I have a bit of a problem with that…

In the ventricles, the QRS and the T wave are usually – but certainly not *always* – located on the same side of the baseline. That's because oppositely charged vectors are traveling in opposite directions. But in the atria, the P wave (the atrial equivalent of the QRS) and its Ta wave are ALWAYS on opposite sides of the baseline – <u>ALWAYS!</u> So how is it that the

inverted, retrograde P ′ waves located in the inferior leads (Leads II, III and aVF) are followed by *inverted* “Ta waves?” I think we need to keep looking for answers.