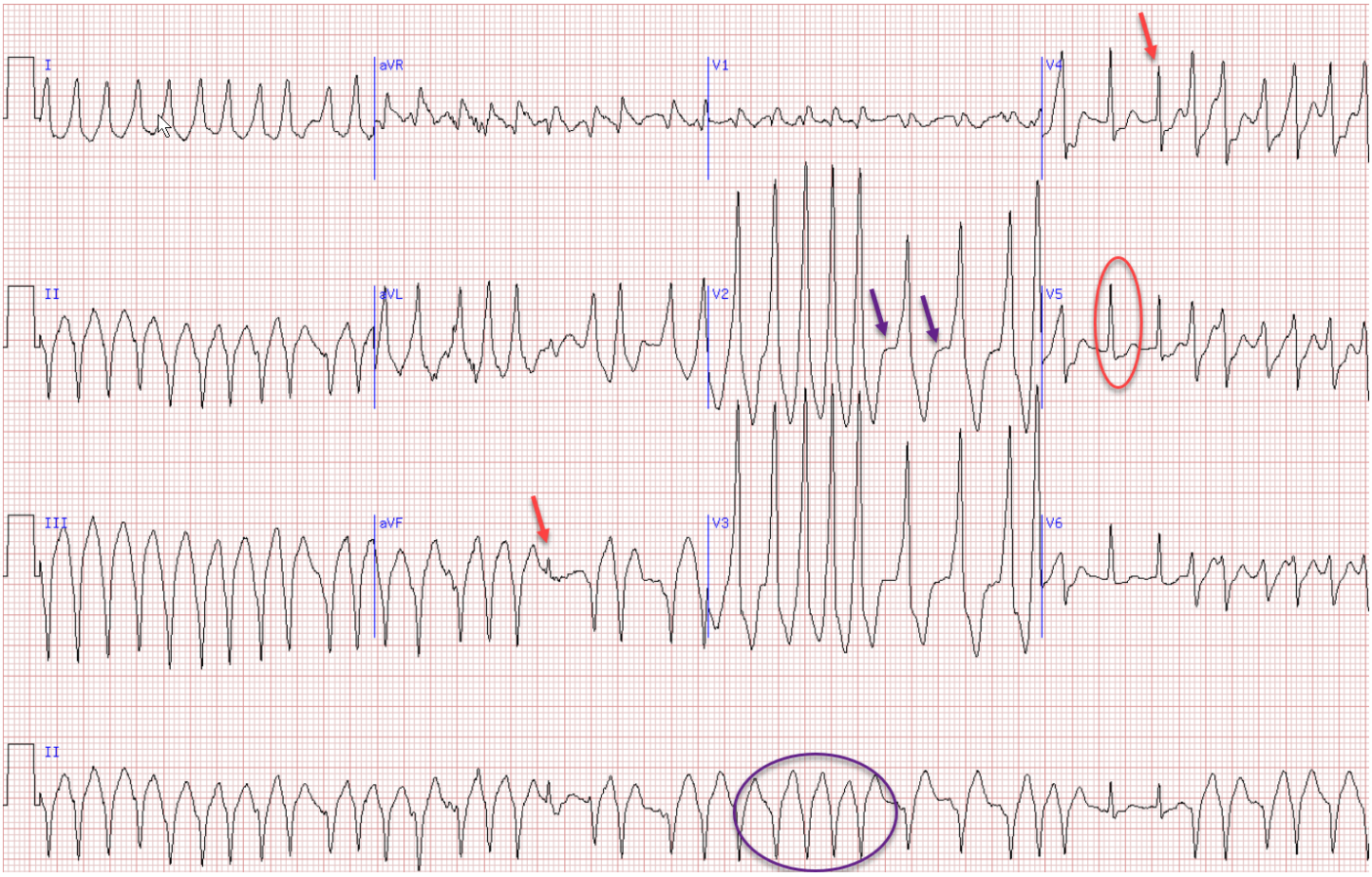
# A Wide and Irregularly Irregular Rhythm
# What Could Be MORE Dangerous Than Ventricular Tachycardia?

**Jerry W. Jones, MD FACEP FAAEM**




This ECG discussion is aimed at the more advanced ECG nerds but even newbie nerds can learn from it.

This is a wide-complex tachycardia. Well, most of the complexes are wide… *but not all!* There are a number of issues to be addressed on this ECG.

First, there are three kinds of QRS morphologies on this ECG. Look at the first four or five beats in Lead III. That is what I would call a *monomorphic* tachycardia (please note that I did not mention "ventricular"). There are also some narrow, normal-appearing beats with opposite axis orientations. And there are beats that look very, very similar to the normal beats, but they are just a little bit wider. You can see such a QRS in the red oval in V5. The QRS to the right of it is visibly thinner and looks completely normal.

Now, when I discuss ventricular tachycardias, I make two distinctions:

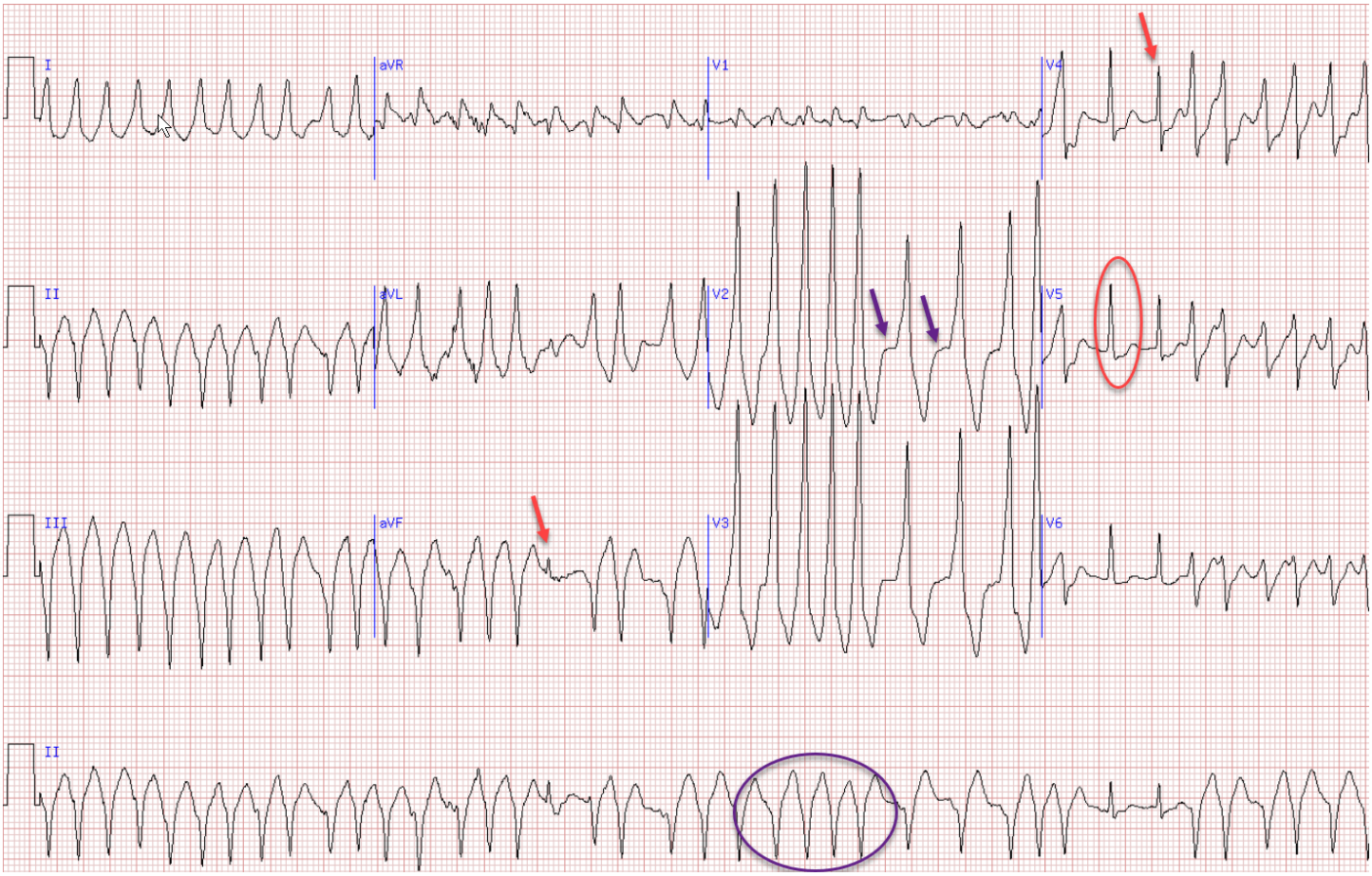
> There are many ways to categorize ventricular tachycardias and I use all of them in my teaching. One method is to divide VTs into *monomorphic* and *polymorphic* varieties. *Monomorphic* VTs almost always have a *very regular rhythm* and *polymorphic* VTs almost always have an *irregular rhythm*. Unless you are seeing hundreds of cases of VT every day, you can forget about the word "almost" in the previous sentence.


So, we have an irregularly irregular rhythm – there is no pattern to this rhythm.

When someone asks you what the rhythm is on an ECG or even just a rhythm strip, *that is a trick question!* There are always at least two rhythms on an ECG because every ECG is actually TWO ECGs recorded simultaneously on the same piece of paper – an *atrial* ECG and a *ventricular* ECG. Thus, there is an *atrial rhythm* and a *ventricular rhythm* and, obviously, they are not always synchronized. There can even be a third rhythm if a *parasystole* happens to be present. But back to this ECG…

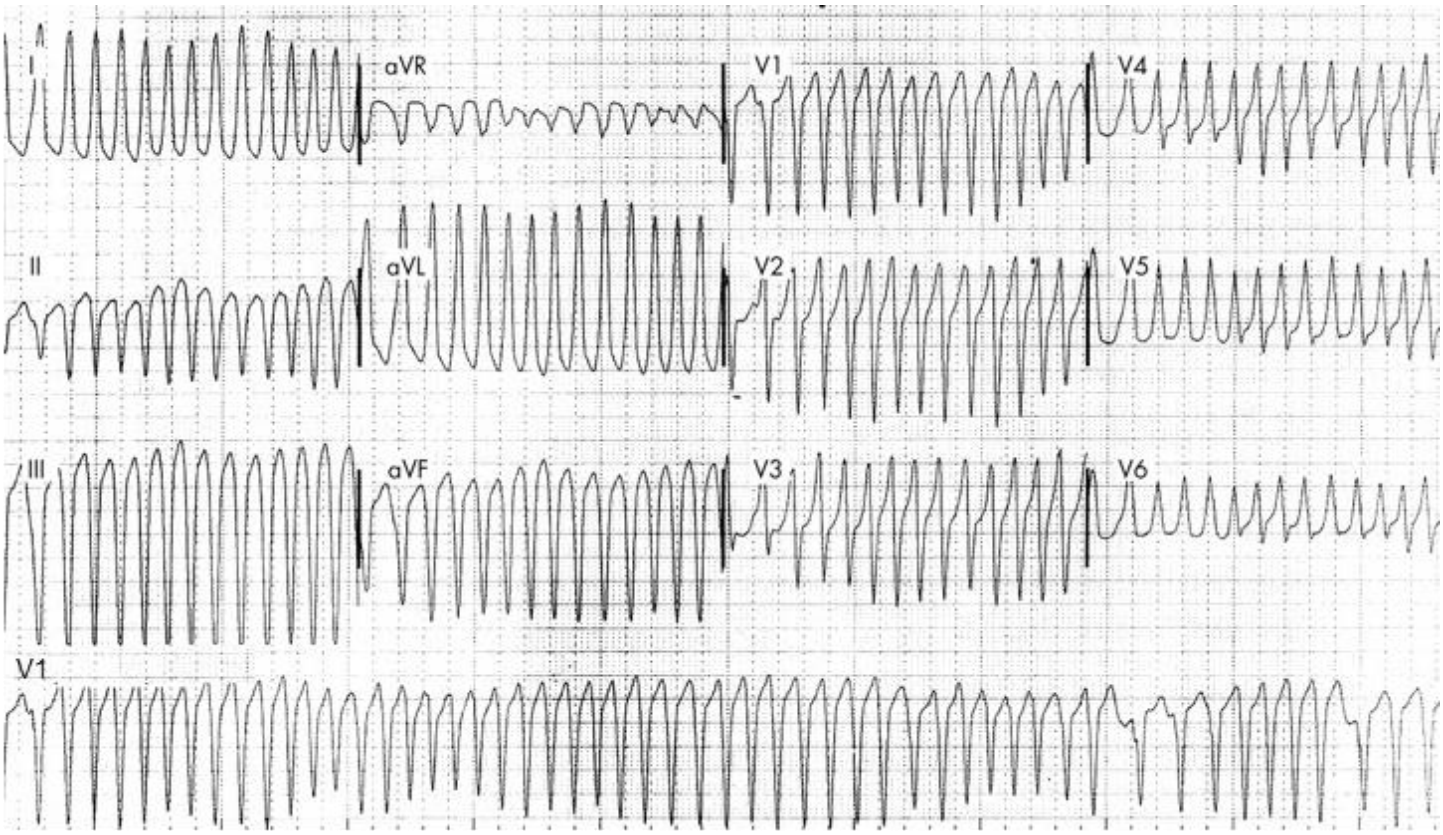
The rhythm is very *irregularly* irregular. There are many rhythms that can be *irregularly* irregular but, of course, *atrial fibrillation* is by far the most common and that's exactly what this is. Incidentally, a *regularly* irregular rhythm is an irregularity that repeats itself and we call that an *allorhythmia*; a Mobitz I AV block is a good example of an allorhythmia (or sometimes called just an allorhythm).

Why are the QRS complexes different and why do some have completely different axis orientations? The reason is that this patient's heart has two ways for the atrial impulses to access the ventricles: the *AV node* and an *accessory pathway*. How do I know that there is an accessory pathway? Look at the beats enclosed in the blue oval in the Lead II rhythm strip. Those beats have conducted to the ventricles at a rate that is very close to 300 beats/minute. Now think for a moment: have you ever seen the ECG of a patient in atrial fibrillation who is *not* on any medication for it? How fast was the ventricular rate? Probably around 120 beats/minute. During atrial fibrillation, the fibrillatory waves are occurring between 400 and 600 beats/minute. But there is so much competition for these beats to get through the AV node that they tend to pile up on one another and leave the




AV node too refractory to conduct more rapidly than about 120 beats/minute (remember the old motion picture comedies where two or three people would try to go through a door at the same time and then get stuck in the doorway? – same thing here). This "pile up" in the AV node is due to something called *concealed conduction*. Besides, the AV node conducts impulses in a *decremental* manner which means that *the faster the rate, the slower the conduction*. Bottom line: you are not going to have an AV node conducting any where close to 300 beats/minute without a lot of sympathomimetic drugs on board – and even then it would be very unlikely to conduct that fast – *especially if the patient is at rest!*

But an accessory pathway is often composed of atrial myocytes that form a bridge between the atrium and ventricle. Such cells would have no problem conducting at 300 beats/minute or even faster. So why isn't the entire ECG showing conduction at 300 beats/minute? The answer is that *the AV node is also conducting and those conducted beats are competing with the impulses being transmitted through the accessory pathway*. Plus, even the accessory pathway can also "block up" occasionally for the same reason. Now do you see how important it is NOT to give any AV nodal blocking drugs to a patient like this? Take a look at what could happen on the next page…




This is an example of a patient with atrial fibrillation going long periods without any AV nodal competition. The ventricles simply cannot sustain a rate this fast and will usually fibrillate after a very short time.

This is an irregularly irregular rhythm but here is a very valuable pearl for you…

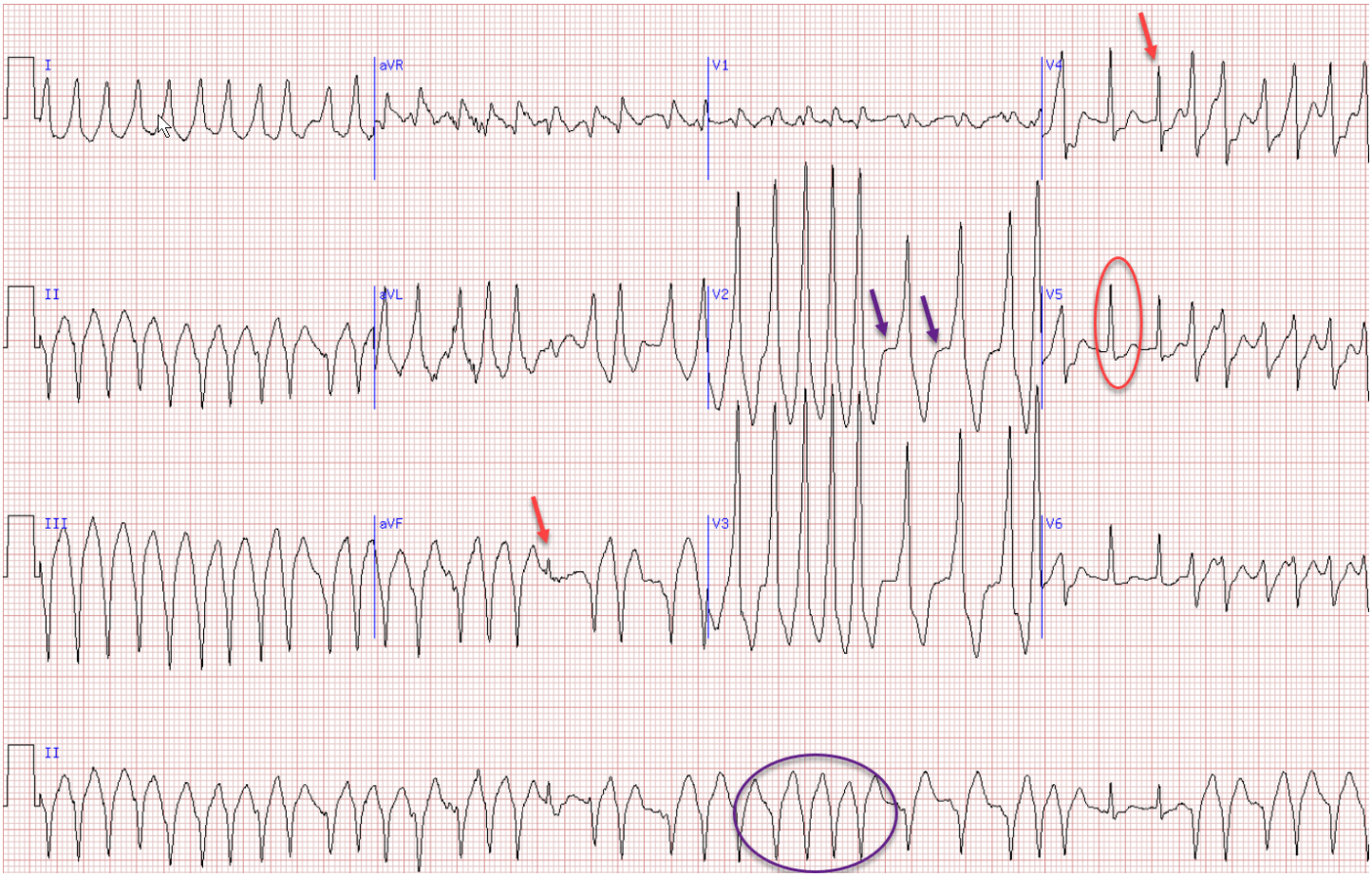
## *PEARL*

*Very **fast** ventricular rates and very **slow** ventricular rates can look quite regular at first glance. Be very, very careful when interpreting both situations!*

Go back to our original ECG for a moment and look at the blue arrows. Those arrows are pointing at "right shoulders" preceding some of the QRS intervals. Here's another pearl for you…

## *PEARL*

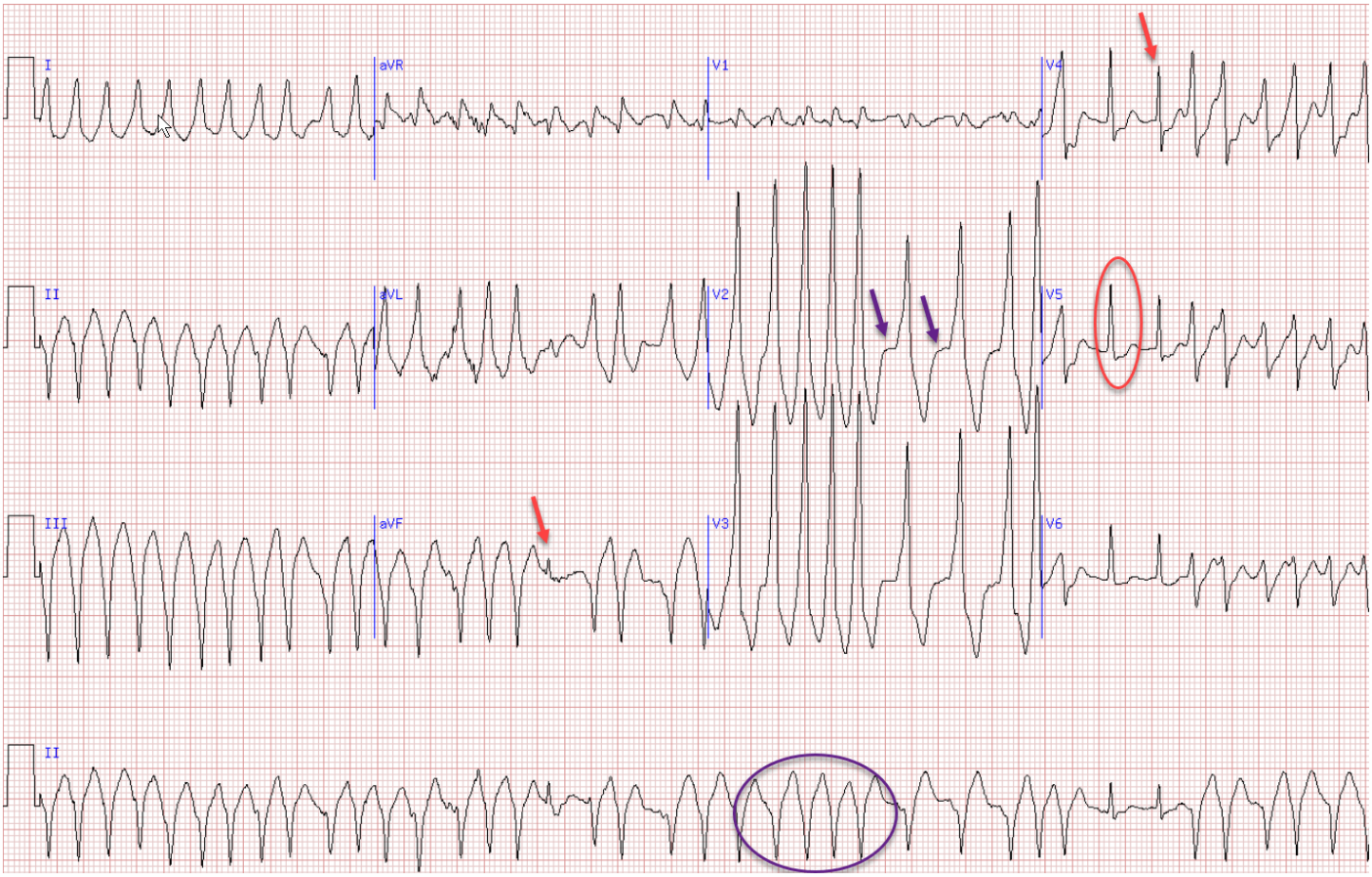
When you are interpreting a wide complex tachycardia and see some QRS complexes with "right shoulders" and some (or most) without, you are very likely viewing atrial fibrillation.




If you look at the QRS in Lead V4 with the red arrow, you will see a normal, AV node-conducted QRS. The QRS that just precedes it (red oval in Lead V5) is also an AV node-conducted beat, but it's just a little bit wider (this is more apparent in V4). Why is that? Is that a "fusion" beat? Unlikely, because a fusion beat under these circumstances would most likely result in a delta wave. That's not the case here. This is likely a slightly aberrantly conducted beat that resulted in just a minimal amount of delay in one of the bundle branches (probably right bundle branch). By the following beat, the right bundle is able to conduct completely normally.

OK, then… is the normal beat a *capture beat*? No, it isn't. A capture beat represents an atrial impulse transmitted through the AV node during a ventricular tachycardia strongly suggesting – *but NOT proving!* – a ventricular ectopic focus (i.e., ventricular tachycardia). What we see in this ECG is simply a normally transmitted beat while the accessory pathway is momentarily blocked. And, as you can see, transmission through the accessory pathway quickly resumes. Be careful: there is a big difference between a *capture beat* (which strongly suggests an ectopic ventricular focus) and a *capture**d*** *beat* (which indicates normal transmission through the AV node *and nothing more*). A capture beat will appear at a cycle length that is shorter than the prevailing ectopic rhythm.

Now, why did I mention the part about *monomorphic* VT and *polymorphic* VT at the beginning? A bit of history – for many years it was taught that ventricular tachycardia was a very irregular rhythm. The reason was that these earlier ECG interpreters were seeing 1) polymorphic VT and 2) atrial fibrillation being transmitted over an accessory pathway. In the case of polymorphic VT, they were correct, but not in the case of atrial fibrillation. *A regular wide monomorphic tachycardia between 150 and 200 beats/minute is much more likely to be ventricular tachycardia than any other rhythm (from my own experience in 40 years of reading ECGs)*.




Now, for my last point, let's look back at the title of this article: "What could be more dangerous than ventricular tachycardia?"

When an accessory pathway is present, there are two ways a rapid tachycardia can develop.

First, there can be a *macro reentry circuit*: the initial atrial impulse enters the ventricle through the AV node and then exits back into the atria via the accessory pathway, activates the atria in a retrograde manner and then reenters the AV node. This is called an *orthodromic atrioventricular reentrant tachycardia (AVRT)*. The QRS complexes will be narrow unless there is a pre-existing or acceleration-dependent bundle branch block.

Second, the atrial impulse may be transmitted through the accessory pathway first and then it exits the ventricles through the AV node, re-excites the atria and then enters the ventricles once again via the accessory pathway. This will result in a wide complex tachycardia called an *antidromic AVRT*. As scary as these tachycardias may appear on the ECG, they are both *nodal-dependent* dysrhythmias in that *both tachycardias must go through the AV node at some point to sustain the rhythm*. This makes treatment very easy because all one has to do is block the AV node and that will immediately end the dysrhythmia. Plus, the AV node itself is a limiting factor for the tachycardia due to its decremental conduction.

Third, there may be more than one accessory pathway present which would allow entry into and exit from the ventricle without the control exerted by the AV node.

All of the above are reentrant rhythms and will result in a regular rhythm – whether the QRS complexes are wide or narrow.

But in this ECG, we have a fourth *different and much, much more dangerous situation*. In this case, the accessory pathway is not being used as part of an AVRT reentrant circuit but, instead, as a *bystander* pathway. The rapid transmission of the impulses entering the ventricles is not going through the AV node, so this is *not a nodal-dependent dysrhythmia*. There are, however, impulses entering through the AV node and the competition of those beats with the ones coming through the accessory pathway is probably what is keeping the ECG from looking like the one on page 3. Also, there will be some pleomorphism of the QRS complexes – as during *any* atrial fibrillation – due to the variations in refractoriness of the ventricular conduction system.

Giving any medication that would block the AV node – as one would do for an AVRT – would be absolutely disastrous in this case and possibly lethal as well.

Want to learn more about dysrhythmias? Come join us in **Houston, Texas June 9 – 12, 2025** for a live, in-person 4-day interactive course in advanced cardiac dysrhythmias: ***The Masterclass in Advanced Dysrhythmias***. This is NOT a beginner's course nor is it a review of the usual dysrhythmias taught in ACLS courses. This course deals with the complex dysrhythmias that occur as a combination of blocks, concealed conduction, reentry, triggered activity, transmural dispersion of repolarization and various other sources of rhythm disturbances.

The Masterclasses are kept small to allow for more interaction by all participants. They are very casual and informal and you can ask questions or make comments at any time. Everyone will have the opportunity to analyze complex ECGs during class while being guided and prompted by Dr. Jones, an internationally recognized instructor in advanced electrocardiography.

**I teach advanced electrocardiography because when someone's LIFE is in YOUR hands, introductory skills are NOT enough!**

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