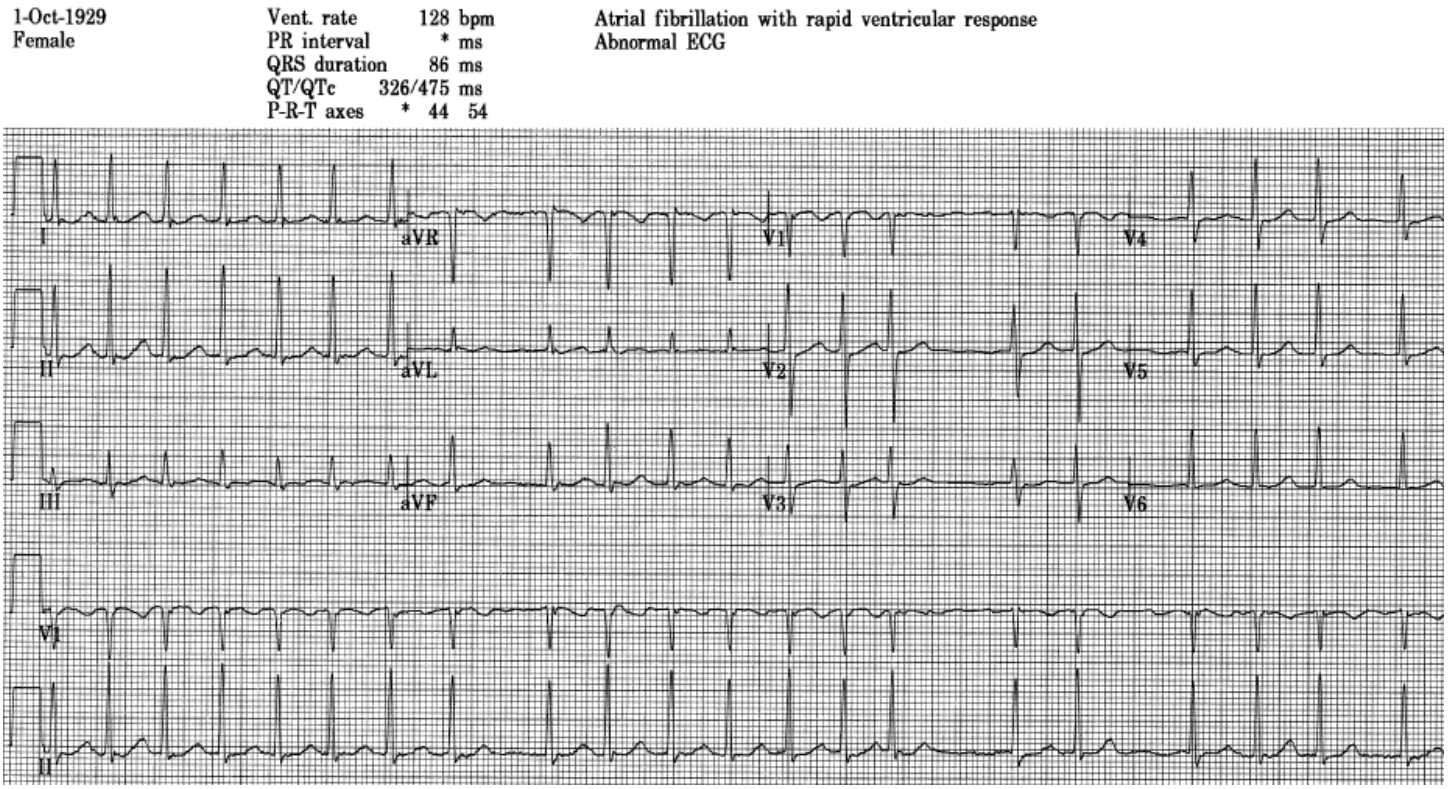
Good Grief! Was the Machine Interpretation Actually Correct?



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The machine interpretation says this is “atrial fibrillation with rapid ventricular response…” but is it, really?

And, whether it truly *is* atrial fibrillation – *or not!* – why is the rhythm so regular for a second or two and then… no ventricular activity at all?

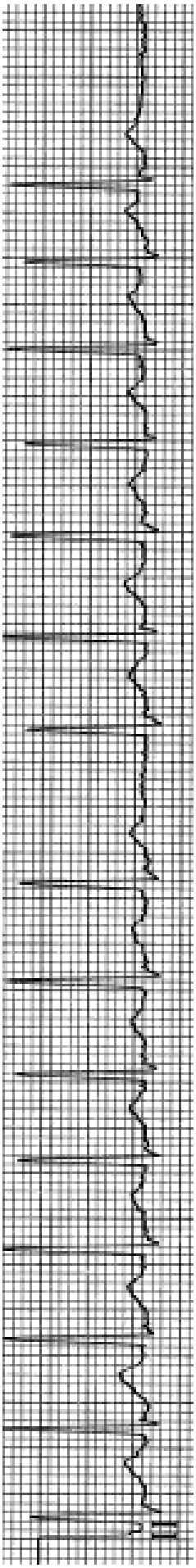
Let’s address the first question. I’ve always said that “very *slow* rhythms can appear *deceptively regular* and very *fast* rhythms can also appear *deceptively regular*. Had the pauses not been present, you may have tried to diagnose this tachydysrhythmia as an **AVNRT** or an **AVRT**. Perhaps even an **atrial tachycardia** with the P’ wave buried in the preceding T wave.

The three pauses rule out those three diagnoses almost preemptively.

So, what IS the diagnosis here? Could the ECG machine be (gasp!) *correct* for a change? Well, yes it is. That brings up the question: is the rhythm actually *regular* during the four tachycardia episodes?

The answer is… ***ALMOST!*** Of course, in this case, “almost” means “NO!”

Let’s take a closer look at the Lead II rhythm strip – but just the first two tachycardic episodes:



You will need some calipers for this. If you measure from the beginning of one R wave to the next you will see a fairly regular rhythm with only slight variations. The last R-R interval of the first tachycardic episode ends with a slightly *prolonged* R-R interval. Could this be a *third degree AV block* and a *junctional tachycardia with a Mobitz I exit block*?

Unlikely for two reasons:

1) *Junctional tachycardias due to enhanced or abnormal automaticity are very, very rare*. Another form – called PJRT* – it is almost always in the context of recent heart surgery for a congenital defect or digitalis toxicity. It is very unlikely that an 82 year old woman is finally getting surgery for a congenital heart defect (though not *totally* inconceivable) and there is no other evidence for the presence of digtoxicity.

2) The second episode ends with a *shortened* R-R interval, which is very contrary to a Mobitz I exit block – even an *atypical* Mobitz I exit block. The prime rule for ***ALL*** Mobitz I blocks – *typical* or *atypical* – is that (in *this* case) the last R-R interval *before* the pause is longer than the first R-R interval *after* the pause. If we were discussing a Mobitz I ***AV*** block, we would be talking about P-P intervals.

Actually, during the first episode, the R-R interval preceding the first pause is indeed *longer* than the first R-R interval following the pause! Does this *prove* a Mobitz I exit block as mentioned earlier?

Unfortunately not! During the second episode, we see that the last R-R interval *before* the pause is much shorter than the first R-R interval *after* the pause. These tachydysrhythmic episodes do not represent Mobitz I exit blocks. As you measure the R-R intervals, you will see that there are slight, subtle irregularities in the rhythm. This would not happen with a reentrant tachycardia (AVNRT, AVRT) and would usually only occur during atrial tachycardias at the *onset* and *offset* (termination) of the tachycardia. In the case of atrial tachycardia, the accelerating or decelerating sections would be more gradual, usually over about 4 – 8 beats.

The machine was *correct* – this is indeed ***atrial fibrillation with a rapid ventricular response***.

But what caused the pauses? There are two likely explanations which are not necessarily mutually exclusive…

First, we are likely seeing concealed conduction here, i.e., two many fibrillatory waves are trying to get through the AV node at the same time and are encountering an AV node or His bundle that has been rendered momentarily refractory.

Second, and equally likely, is that we are witnessing *overdrive suppression*. This patient is 82 years old, so we are aware that she doesn't have the heart of a 20 year old woman. It is not at all unusual to see a rapid tachycardia (and not necessarily a *regular* rapid tachycardia) end abruptly followed by a pause of various lengths before a rhythm (sinus or otherwise) resumes.

So, what IS *overdrive suppression*? As the heart beats more and more rapidly, or remains at a rapid rate for an extended length of time, the amount of intracellular Na^+ begins to increase because the transport system responsible for moving Na^+ out of the cell and K^+ back into the cell, has fallen behind. The Na^+/K^+ pump (Na^+-K^+ ATPase) must work harder and faster to eliminate the Na^+ from the cellular interior in a 3:2 exchange with K^+. You can see that this will result in a *repolarizing current* (more positively-charged ions leaving the cell than entering) that can eventually take the resting membrane potential (RMP) to a very, very negative level – much more negative than the usual -90 mV of the RMP. Eventually, the cell becomes so *hyperpolarized*, that it cannot reach threshold and depolarization ceases while the Na^+/K^+ pump catches up. Once the RMP is closer to its usual -90 mV, depolarization resumes and we see the appearance of QRS complexes once again. The Na^+/K^+ pump is active throughout the action potential, *even during Phase 0* (depolarization)!

The irregular irregularity of the rapid ventricular rhythm PLUS the lack of P waves during the pauses tells us that the tachydysrhythmia is *atrial fibrillation*. The pauses are, therefore, not likely to be due to an exit block of a subsidiary pacemaker activating below a 3rd degree AV block. Instead, it is likely due to a combination of concealed conduction and overdrive suppression in this 82 year old heart.

Whenever you encounter a rapid ventricular rate – due to any cause! – followed by a short period of ventricular asystole, you are witnessing *overdrive suppression*. Overdrive suppression is also another cause of Stokes-Adams attacks.

*The junctional tachycardia I am referring to is something called ***permanent junctional reciprocating tachycardia, or PJRT***. Actually, this tachycardia is neither *permanent* nor *junctional*. It is a form of AVRT conducting orthodromically through a decrementally-conducting accessory pathway (also a rarity!). The slow-down in the accessory pathway keeps the PJRT from self-terminating. Its rate tends to be slower than AVNRT or regular AVRT and it can be slightly irregular at times. But it is *very rare in adults* – especially those who have ***not*** just undergone surgery for a congenital heart defect.