READING vs. INTERPRETING an ECG

Discussion

Jerry W. Jones, MD FACEP FAAEM

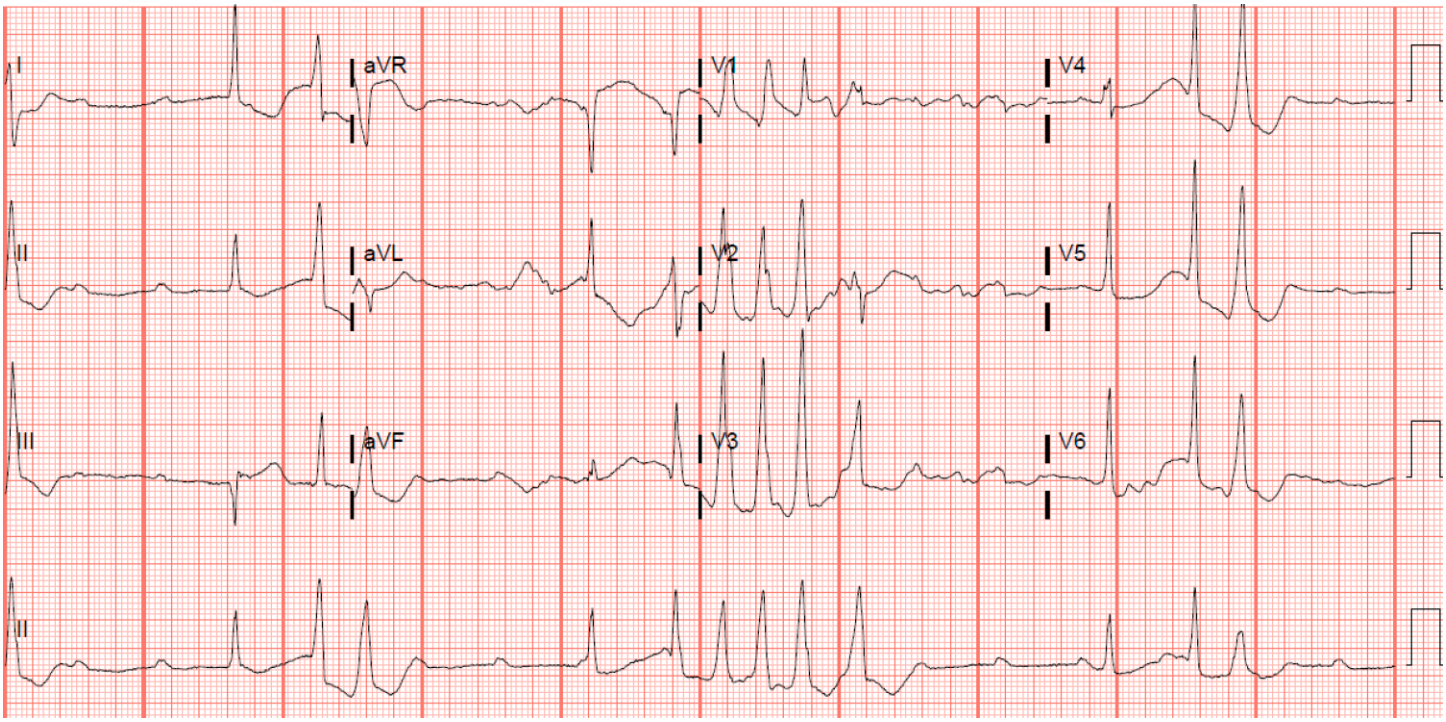

Figure 1

The *base* rhythm is *sinus*. No matter how awful the ECG may appear, we always look to see if there are regular P waves. If so, the *base* rhythm is sinus *no matter how many other dysrhytmias may be present*. If any *regular* rhythm is *faster* than the sinus base rhythm, then we call that the *prevailing* rhythm. But the sinus rhythm remains the *base rhythm*.

Some of the QRS complexes appear normal and narrow… but they have no relation to the P waves, which appear at regular intervals (though many are concurrent with QRS complexes). So, there is a *third degree AV block* present. When you mention *first*, *second* or *third* degree blocks, you must always stipulate *where the block is occurring* because first, second and third degree blocks can occur in the sinus node, the AV node, in the bundle branches and in the exit paths for ectopic pacemakers.

We also see some wider, ectopic beats appearing in short bursts or clusters. Although the narrow – presumably junctional escape – beats have no relationship with the P waves, these short busts of ventricular ectopy DO have a relationship with a certain deflection. Do you see which one?

They are related to the T waves. Each burst (or *salvo*) of ventricular ectopy occurs just after the peak of the T wave - the infamous “danger zone” of the T wave. This is a short period of time in which there is maximal dispersion of repolarization present in the ventricular wall. Different levels of the myocardium have repolarized to different degrees providing a rich substrate for reentrant ventricular tachydysrhythmias.

But this area is also known as *Phase 3 of the action potential*. A slow heart rate (i.e., ventricular rate) allows more time for calcium to enter the myocytes during Phases 2 and 3. In order to reduce this intracellular calcium load, the sodium-calcium exchanger (NCX) transports one Ca^{++} out of the cell in exchange for three Na^{+} entering the cell. If enough Ca^{++} has entered the cell, the increased influx of Na^{+} can “trigger” another action potential producing an ectopic QRS! This is what is called *triggered activity* – one of the three sources of dysrhythmias! It’s one that many of you may not have heard about because it’s a bit more complicated than automaticity or reentry.

Slow heart rates contribute to triggered activity. Hypokalemia – even mild hypokalemia – can also predispose to these potentially lethal dysrhythmias. Ventricular rhythms caused by triggered activity are very unstable rhythms in most cases and last only a few beats – as you can see on this ECG. But they can also be the source of a *polymorphic ventricular tachycardia* called *torsade de pointes!*

Since there is *no sustained* tachycardia here, treatment will be *preventive*. The first thing you need to do is increase the ventricular rate above 60 – 70 beats/minute to allow less time for calcium entry during Phases 2 and 3. You should also check the serum potassium level. If it is *low* – or even *low normal* – give enough potassium to raise the level to *mid* or *high normal*.

When you see short, non-sustained runs of ventricular tachycardia, think in terms of *preventive* measures – not so much *rescue* measures (which may produce immediate results). That is what you are doing when you give magnesium to a patient with a possible torsade de pointes: the magnesium isn’t going to STOP *anything* acutely; but, once the tachycardia *has* stopped, it will make it much more difficult for it to resume.