A 12-Lead ECG That Teaches Us a LOT!

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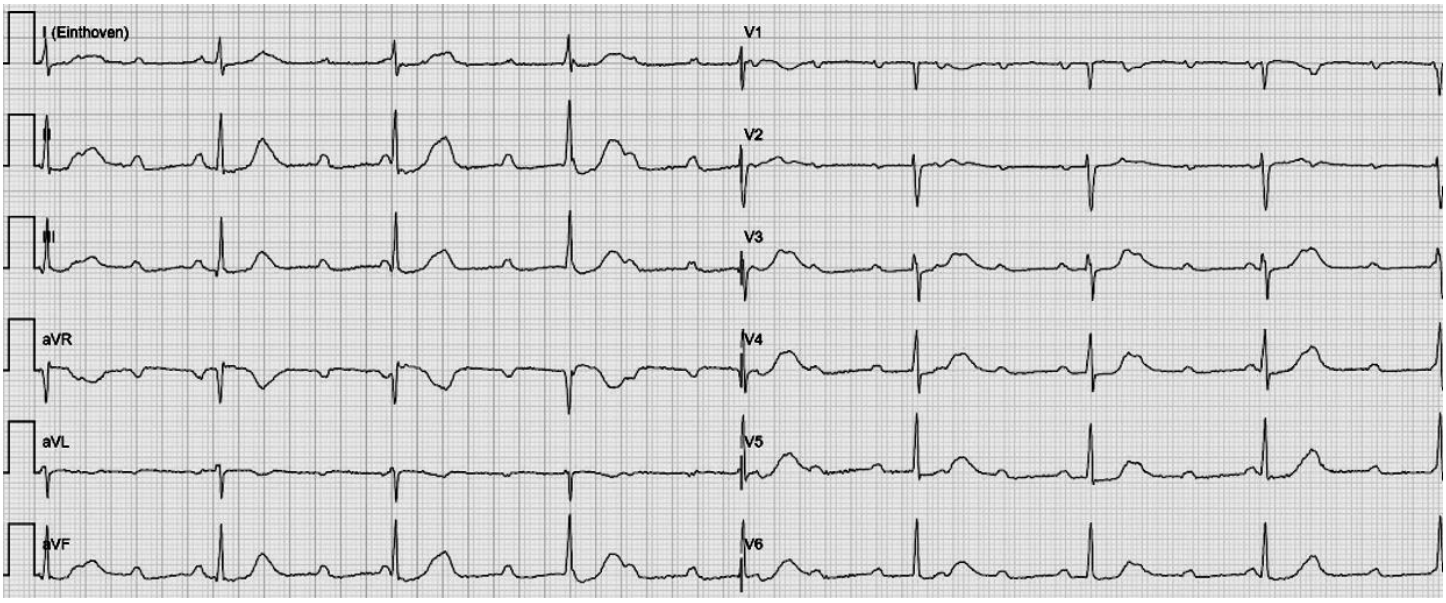

Figure 1

The problem on this ECG should should be very obvious…

If you said "AV dissociation" you would be correct – but you are only *reading* the ECG. Remember: AV dissociation is an *electrocardiographic symptom* – it tells us nothing about the patient nor gives any hint regarding what we should do for that patient. It's like telling someone who has come to see you because of a temperature of 103° F that they have a fever!

You have to *interpret* what the ECG is telling you. So, if you said "AV dissociation due to third degree AV block with a junctional escape rhythm" then you have interpreted the ECG.

But any good electrocardiographer would then be prompted to ask, "*Why* is this patient having a third degree AV block with a junctional escape rhythm?" The first thing you should consider is an *inferior transmural ischemia*. Remember: RCA (and sometimes LCx) occlusions cause *AV blocks* while LAD occlusions cause *bundle branch blocks* (usually RBBB). The left bundle branch immediately fans out into a spider's web of fibers. For an LAD occlusion to cause a complete LBBB, it would have to wipe out most of the left ventricle. An LAD occlusion resulting in a focal ischemia directly on the left main bundle (which is only about 1 cm in length) is a very rare occurrence!

Is there any hint of a cause of the third degree AV block? Yes, there is! First, I see no convincing evidence of an acute transmural ischemia. Of course, it is quite possible for a *complete* coronary occlusion to occur without *any* initial ECG changes. I personally have seen this happen – many times! Even if the patient weren't complaining of chest pain, I

would still initiate a workup for acute myocardial ischemia. It is also possible for certain people, such as diabetics, to have an MI *without any reported chest pain*. I've also seen *that* happen – a few times.

The teaching value of this ECG lies in the repolarization. Look at those T waves. It's like looking at a zoo! There are so many different shapes and sizes of T waves! Look at the shapes that the T waves assume when the P wave is on either the upslope or downslope and also when it is very near, or exactly on, the peak of the T wave.

The P wave is the *only* deflection that can hide (or *try* to hide) in a T wave. Think about it. If a QRS is superimposed on a T wave, it would be like an elephant trying to hide behind a sapling! There's no way you are going to miss a QRS complex! The only other deflection left to consider is a U wave – but that would be an impossibility because the U wave is also a repolarization phenomenon that *must* follow a T wave. It can *never* be superimposed on it (although it *can* merge with the termination of the T wave).

Did you notice that at times the T wave appears to become *slightly larger* but never slightly smaller? That's because the P wave is either adding *positive voltage* to a *positive deflection* or, in the case of Leads aVR, aVL and V1, adding *negative voltage* to a *negative deflection*. On the infrequent occasion (but not on *this* ECG) that an inverted (negative) P wave is superimposed on an upright (positive) T wave, the positive and negative voltages offset each other and the T wave will actually become *slightly smaller*.

Now, why is there a third degree AV block? Look closely at the ST segments in the inferior leads. All are depressed, but look even more closely at the *shape* of those ST segments! It looks as though someone has taken an ice cream dipper and scooped out a portion of the baseline. I suspect digitalis is the culprit here. This also brings up three more questions.

First, digitalis is notorious for causing junctional rhythms. Did it have any effect on the junctional escape rhythm? I certainly don't think so. Digitalis is notorious for causing *accelerated* junctional rhythms, but a junctional *escape* rhythm is definitely *physiologic*.

Second, doesn't digitalis toxicity result in sinus bradycardia? Yes it does… but you have to consider something else here. This patient has just experienced going from a normal heart rate to a rate around 40/minute! That likely caused a blood pressure drop and a surge of adrenaline to increase the heart rate, likely overriding the effect of the digitalis on the sinus node.

Third, could the digitalis have caused an atrial tachycardia resulting in the rapid atrial rate? Digitoxicity is also infamous for causing paroxysmal atrial tachycardia. The atrial rate here is around 115/minute. That's really too slow to be concerned about atrial tachycardia.