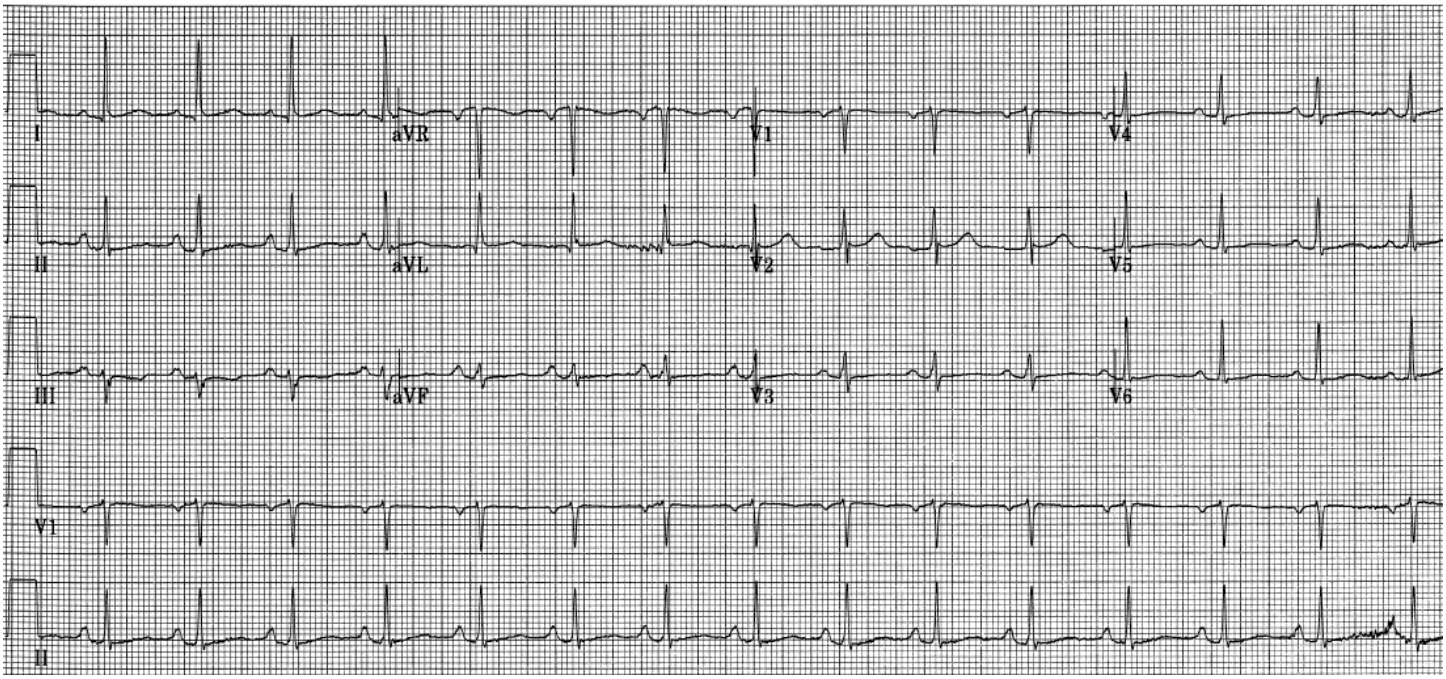
An “Abnormal ECG”

14-Nov-1961	Vent. rate	92 bpm	Normal sinus rhythm
Female	PR interval	170 ms	Possible Left atrial enlargement
	QRS duration	76 ms	Nonspecific T wave abnormality
	QT/QTc	374/462 ms	Prolonged QT
	P-R-T axes	72 17 18	Abnormal ECG



This is another randomly selected ECG from my personal library. It is from a female who was 50 y/o at the time the ECG was recorded (in 2011). We know nothing else about her.

This is *sinus rhythm* – P waves are upright in Leads I and II. This is an ***absolute requirement for sinus rhythm***. Everything else is just confirmatory. Lead II has the tallest P waves – confirmatory but not absolutely necessary for a diagnosis of sinus rhythm. The P waves in Lead V1 are NOT +/- biphasic; in fact, they are not biphasic at all. We only see a negative P wave. A classic +/- biphasic P wave would certainly add confirmatory evidence for sinus rhythm, but lack of that morphology does not rule it out. Please note that I did not mention anything about the P-R interval. That’s because ***the P-R interval has absolutely NOTHING to do with sinus rhythm***.

The P waves in Leads II and aVF are the largest on the tracing. Both leads (along with Lead III) have their positive electrode on the left foot. However, that doesn’t mean that they share the same positive pole because they all

PEARL #1

When someone – and especially someone who is an expert with ECGs – asks you, “What is the heart rate?” that is a trick question! There is an ATRIAL rate and a VENTRICULAR rate.

have different “negative” poles! Lead II has its positive pole at +60°, Lead aVF has its positive pole at +90° and Lead III has its positive pole at +120°. In this ECG, we can see that the P wave vector is pointing inferiorly because

it is inscribing large, positive P waves in Leads II and aVF (Lead III is slightly out of this range because its P waves are much smaller). Since the P waves in Leads II and aVF are almost the same size, we could assume that the P wave vector is located about halfway between the two leads. That would be +75°. When we look at the ECG machine's measurement, we see that it is +72°. Not bad! Whenever you see a P wave axis that is more rightward than +60°, *start suspecting COPD*.

PEARL #2

By convention, when a vector or axis moves TOWARD +90°, we say that it is moving RIGHTWARD or that the axis is VERTICAL. When a vector or axis moves up TOWARD 0°, we say it is moving LEFTWARD or that the axis is HORIZONTAL.

The ECG machine says there is *possible* left atrial *enlargement*. Today, we would normally say left atrial *abnormality*. The same pattern may appear with *dilatation*, *hypertrophy* or *interatrial conduction delay or block*. But why does it say *possible*? The negative component of the P wave in Lead V1 *barely* meets the criteria of 1 mm wide and 1 mm deep. However, this is not confirmed by the P wave in Lead II which has a *normal duration*. Left atrial abnormality is confirmed by a P wave in Lead II that is > 0.11 seconds. The lack of supporting evidence for left atrial abnormality in Lead II does not rule out a left atrial abnormality, but it makes the weak evidence in Lead V1 a bit more suspect. I agree with *possible* left atrial abnormality, but I will have another explanation at the end of this article.

The ECG machine also states "nonspecific T wave abnormality." Why does it say that? Well, does anything about the T waves on this ECG strike you as odd? Only one lead (Lead V2) has T waves of reasonable size. All the other T waves are virtually isoelectric. What condition do you think of first when the T waves are very small or isoelectric? Hypokalemia! Do you think this is hypokalemia? *Possibly*… but I would not be too quick to jump to that conclusion. Why? We all know that in the course of hypokalemia the T waves get smaller and smaller… but what else happens *at the same time?* The U waves get larger and larger! Do you see any U waves on this tracing? I don't.

The differential diagnosis for low voltage T waves is not too extensive, but the things I would consider next after hypokalemia would be hypomagnesemia, medication or drug interference with repolarization, and hypothyroidism. While it's possible conditions like hyperinflated lungs, pericardial or pleural effusion and morbid obesity could possibly contribute to this pattern, I think it would be most unusual for just the T waves to be affected. Almost all these questions could be answered with a good history and physical exam. But what if you can't do a history or physical and you see no explanation elsewhere on the ECG for the isoelectric T waves – what can you call it? What is your diagnosis? By default, it is *nonspecific T wave abnormality*. We see these comments so often, we tend to brush them aside and pay little attention. Always remember: *nonspecific* does NOT mean *trivial*, *unimportant,* or *benign*! Let me translate the phrase "*nonspecific T wave abnormality"* for you: "***There is something wrong here and I do not have the information necessary to determine exactly what it is. It may be benign – but it may NOT be benign at all! YOU must decide what it is!"*** During my career, I have seen ECGs labeled *nonspecific T wave abnormality* when the abnormality was actually **Jones's sign**. Therefore, whether the comment comes from the ECG machine or the cardiologist – pay close attention to *nonspecific T wave abnormality.*

The machine states the QT interval is prolonged and indeed it is. But the QTc is only 463. The upper limit for men is 440 and for women it is 460. We generally don't start worrying about imminent dysrhythmia complications until the QTc reaches around 500. However, with *any* prolonged QTc, it would be very prudent to

check all the patient's medications, explain the implications of a prolonged QTc and direct the patient to one of the excellent websites that give more information and also list prescription and non-prescription medications to avoid.

There is one other issue that is not mentioned in the machine interpretation. Look at Leads V1 – V3. Notice anything a bit unusual? I suspect that the electrodes for V1 and V2 have been placed too high causing changes in the P waves and the QRS morphologies. That is probably why Lead II shows no evidence of left atrial abnormality and V1 just shows minimal evidence with no positive component to the P wave. One would also have to ask, "WHY is there an atrial abnormality?" We know there is no interatrial conduction delay because the P waves in Lead II have a normal duration. It is also unlikely to be due to LVH because there is absolutely no indication of LVH on the ECG (though that certainly doesn't rule it out!). Although mitral stenosis is a possibility, we don't see too much of it anymore. These observations make lead misplacement a more likely cause.

This is definitely not an exhaustive review of any of the topics I have discussed here, but I hope it has guided you a bit in your ECG interpretation and given you some new perspectives.