



## COMMENTARY

# Reading Electrocardiograms “Blind”

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Of course, a blind person cannot read an ECG. By blinded, I mean, reading the ECG without knowing the details of the patient’s condition. As a cardiologist, I read one hundred fifty to several hundred electrocardiograms daily without knowing the patient. In actual fact, reading the electrocardiogram without knowing the patient’s condition can be boring, which may lead to sloppiness, but in some instances, other than learning what the heart rate is from the electrocardiogram, one can make a diagnosis.

Thus with few exceptions, the electrocardiogram is not as useful to the ordering physician as it would be if interpreted in the context of the patient’s symptoms and other findings e.g. Physical exam etc. I also read ECGs of patients that I have evaluated clinically, and find that the ECGs can help with decision making when interpreted in the context of the patient’s condition. e.g. acute myocardial infarction in a patient with ST segment elevation and troponin release.

## Atrial Fibrillation

Atrial fibrillation is the commonest rhythm disturbance admitted to hospital or seen in the clinic. Atrial fibrillation is one of several diagnoses that can be made without knowing the patient’s condition. The rate can be fast or slow and the rhythm is usually irregular. The trigger for the atrial fibrillation can be due to many causes, e.g. alcohol consumption, or other triggers, none of which are available to the

“blinded” ECG reader. The hallmark of atrial fibrillation on the ECG is completely disorganized atrial electrical activity manifested as not seeing any P Waves, with irregular QRS complexes.

## Sinus Bradycardia

Sinus rhythm with heart rate less than 60 beats per minute. I tend to think of hypothyroidism in these patients, but usually that disease is not present.

## Narrow Complex Tachycardia

Another ECG that is easily diagnosed is sinus tachycardia. Unfortunately, many conditions, unknown to the reader are related to sinus tachycardia. e.g. hyperthyroidism. Sinus tachycardia is diagnosed if there is sinus rhythm and the heart rate is greater than 100 bpm.

Another common narrow complex tachycardia is A-V nodal re-entrant tachycardia. This rhythm is diagnosed if the P wave is seen after the QRS (sometimes obscured by the QRS). On many ECGs the ventricular rate is too fast to discern the underlying atrial rhythm, in which case the only interpretation that can be made is supraventricular tachycardia.

## Wide Complex Tachycardia

The commonest wide complex tachycardia with regular rhythm is ventricular tachycardia. In this group of rarely asymptomatic patients, I am inclined to make the diagnosis of VT, unless some other rhythm is proven. e.g. Pre-excitation.

Regular Wide Complex Tachycardia can be ventricular tachycardia or supraventricular tachycardia

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with aberrant conduction. There are many causes of wide complex regular tachycardia and there are several ECG patterns that are seen in patients with the diagnosis of VT. Unfortunately, these patterns relate to populations, and may or may not apply to the individual patient. To unravel the mystery of the etiology of a wide complex tachycardia i.e. VT vs. SVT with aberrant conduction, electrophysiologic study or use of agents such as adenosine, may be required, but in general, a wide complex tachycardia is ventricular, until proven otherwise. Evidence of AV dissociation (i.e. a regular sinus rate slower than/and unrelated to the QRS rate) and fusion or capture beats (isolated beats with a different morphology due to momentary AV nodal conduction of the atrial rhythm) are insensitive but highly specific findings for VT.

## Left Axis Deviation

Left axis deviation is compatible with many conditions but not diagnostic of any one of them.

## Low Voltage

Low voltage in the limb leads or precordial leads is not diagnostic of a specific disease, but I like to think of hypothyroidism or pericardial effusion when low voltage is found. Obesity is also a common cause of low voltage on the ECG.

## Prolonged QRS Duration

Probably the most common cause of prolonged QRS duration is RV pacing, but drugs e.g. quinidine, Left and Right bundle branch block also prolong the QRS duration.

## Prolonged QT Interval

QT interval prolongation determined by the computer may be erroneous but may be drug induced or congenital and cannot be ignored. My approach to this diagnosis is to estimate the QT interval and if it is greater than  $\frac{1}{2}$  the RR interval, I consider it abnormal. Electrolyte deficiencies, particularly potassium, magnesium, and calcium, are common reversible causes of QT-prolongation.

## Prominent R Wave in Lead V1

Prominent R wave in lead V1 may be due to right bundle branch block if the QRS is prolonged or may be seen if the precordial leads are misplaced. Right ventricular hypertrophy can also produce a prominent R-wave in V1.

## Poor R Wave Progression

Delayed precordial transition may be associated with myocardial infarction, but is not diagnostic and may be due to misplaced leads. Patients with left anterior fascicular block will usually have delayed R-wave progression across the precordial leads.

## Peaked T Waves

Peaked T waves are not diagnostic but the first thing that comes to my mind when I see peaked T waves is hyperkalemia, but other causes can do it as well. e.g. early myocardial infarction, acidosis etc.

## Right Axis Deviation

Usually the cause of Right axis deviation is right ventricular hypertrophy, which is usually due to high RV pressure.

## ST Segment Elevation

The commoner causes for this condition are 1. acute myocardial infarction, 2. coronary vasospasm, 3. Pericarditis and 4. benign early Repolarization. The contour of the elevated ST-segment (convex vs. concave) and the specific leads involved (localized vs. diffuse) can be suggestive of one etiology over another. However, reading the ECG without the clinical story and other lab tests makes it impossible to distinguish the cause of ST segment elevation.

## Non-Specific ST and T Wave Changes

As the label suggests, this finding does not rule in or exclude any particular diagnosis, but is extremely

common. They can be seen in a wide range of scenarios, from healthy asymptomatic patients to acute myocardial infarctions. The clinical context, and ideally, comparison with a prior ECG, are both key in order to determine the significance of nonspecific ST-changes.

## Left Ventricular Hypertrophy

LVH determined by ECG has a low positive predictive value to prospectively detect LVH by echo, but is sensitive in patients with known LVH measured by cardiac ultrasound or Cardiac MR. In fact, there are 37 different ECG criteria [1] which suggest a lack of consensus for diagnosis of LVH by electrocardiography. My favorite ECG abnormality to suggest LVH is a prominent R wave in AVL >11 mm. When increased voltage criteria are present along with repolarization abnormalities (i.e. t-wave inversion in leads I, aVL, V5-V6) and/or P negative V1, positive predictive value for Echo determined left ventricular hypertrophy increases.

## Left Bundle Branch Block

LBBB is Compatible with many conditions but not diagnostic of a particular cause of the abnormality. E.g. could be a myocardial infarction if not seen in previous ECGs.

## Right Bundle Branch block

Usually not associated with cardiac conditions with the exceptions of secundum ASD in adults.

## Heart Block

*First degree;* Prolonged P-R interval

*Second degree;*

Type 1; Mobitz 1, Wenckebach-progressive prolongation of the PR interval until a non conducted P Wave occurs. Progressive PR-prolongation is sometimes very subtle and is best visualized by comparing the PR intervals of the beats before and after the non-conducted P-wave.

Type 2; Mobitz 2-fixed block, e.g. 2:1 block

*Third degree;* complete heart block (no relationship of the P wave to the QRS) often associated with wide complex Left ventricular beat.

## Conclusion

My advice is to not make clinical conclusions based on reading the ECG without knowing the patient's condition. Just read the abnormal findings on the ECG. The ECG is best utilized when the clinical situation is known. Let the primary physician taking care of the patient make the clinical decisions based on the ECG and consultants opinion if requested by the primary physician.

## REFERENCE

1. Peguero JG, Presti SL, Perez J, Issa O, Brenes JC, Tolentino A. | Electrocardiographic criteria for the diagnosis of Left Ventricular | Hypertrophy. J Am Coll Cardiol 2017;69:1694–703.