ECG Primer for the Cath: What Does a Tall R Wave in V1 mean? The Four Categories Approach

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Your cath patient is in the lab and the electrocardiogram (ECG) shows a tall R wave in V1 (defined as an R wave amplitude that is greater than that of the S wave). What should you be thinking about and what is the differential for this finding?

A tall R wave in V1 has many etiologies. It can be hard to remember them all, especially since prior approaches emphasized memorization over understanding. But there is a better way.

Tall R Wave in V1: The Four Categories

Let's take a logical approach to the meaning of a tall R wave in V1. First, understand that V1 is the only right-sided lead in the standard 12-lead ECG, and therefore, a tall R wave in V1 represents increased net rightward depolarization. Second, remember that the causes of increased right-sided depolarization can be divided into four mechanistic categories: (1) increased mass, (2) decreased mass, (3) electrical, and (4) technical. Third, within each of these categories, there is a common and uncommon etiology (Table 1). We call this the four mechanistic categories approach. The categories are:

1. Increased mass

The mechanism here is straightforward. Increased right-sided muscle mass results in increased rightward depolarization that manifests as a tall R wave in V1. The common etiology is hypertrophic cardiomyopathy. The uncommon etiology is right ventricular hypertrophy (which can occur when there is strain on the right side of the heart, such as in pulmonary hypertension, pulmonary embolism, pulmonic stenosis, etc.).

2. Decreased mass

The mechanism here is also straightforward, if slightly less so. The amplitude of an ECG wave reflects the sum of all the electrical vectors in the myocardium at a certain time. For example, if there were two vectors of simultaneous depolarization, one rightward and one leftward, the final ECG wave will be in the direction of the greater vector, with amplitude equal to difference between the two. If the leftward vector decreases in magnitude, it will show up on the surface ECG as a higher amplitude rightward depolarization. This is the mechanism by which decreased muscle mass can result in a tall R wave in V1. The common etiology is a posterior myocardial infarction (MI). The uncommon etiology is muscular dystrophy (including myotonic dystrophy and Duchenne muscular dystrophy). Both etiologies trigger decreased leftward depolarization, resulting in the sum of all the depolarization being more rightward and causing a tall R wave in V1.

3. Electrical

The common etiology is a right bundle branch block (RBBB). The uncommon etiology is Wolff-Parkinson-White (WPW) syndrome (there are other, even less common etiologies here, too, which include Brugada syndrome and arrhythmogenic right ventricular dysplasia/cardiomyopathy [ARVD/C]). In these cases, instead of the normal, fast ventricular depolarization through the conduction system, which includes the right bundle branch, there is slow left-to-right depolarization through ventricular myocardium, resulting in later, slower, rightward depolarization, and thus a tall R wave in V1.

4. Technical

The common etiology is lead reversal, specifically V1-V3 lead reversal. In this case, the tall R wave in "V1" is actually the R wave in V3, which represents normal left ventricular depolarization. The uncommon etiology is dextrocardia, in which the heart is rightward oriented, so the net electrical forces are opposite in direction as normal and therefore rightward in sum, resulting in a tall R wave in V1.

The final cause of tall R wave in V1 is a normal variant, especially in a younger person, where it is often also associated with T-wave inversions in V1-V3. When this finding persists into adulthood, it is sometimes called a persistent juvenile T-wave pattern. This doesn't neatly fit into a category, but it is easy enough to remember.

The specific features of each cause of tall R wave in V1 can be deduced by understanding the physiology of each underlying diagnosis. Let's consider some illustrative cases.

Example Cases

Case #1. The diagnosis is right bundle branch block (RBBB) (Figure 1). Features include:

- QRS >120ms – caused by slow depolarization through the ventricular myocardium, rather than fast depolarization through the conduction system.
- RSR' morphology in V1-V3 – caused by the progression of initial right to left depolarization through the septum (R), left ventricular depolarization through the left bundle (S), and right ventricular depolarization through the ventricular myocardium (R').
- Slurred S wave in I, aVL, V5, V6 – representing slow, rightward depolarization through the right ventricular myocardium that shows up as a negative-amplitude wave in the lateral leads.

Table 1. The four Tall R wave categories.

Mechanism	Common	Uncommon
1. Increased mass	Hypertrophic cardiomyopathy (HCM)	Right ventricular hypertrophy
2. Decreased mass	Posterior myocardial infarction	Muscular dystrophy
3. Electrical	Right bundle branch block (RBBB)	Wolff-Parkinson-White (WPW)
4. Technical	V1-V3 lead reversal	Dextrocardia
(Other)	(Normal variant)	

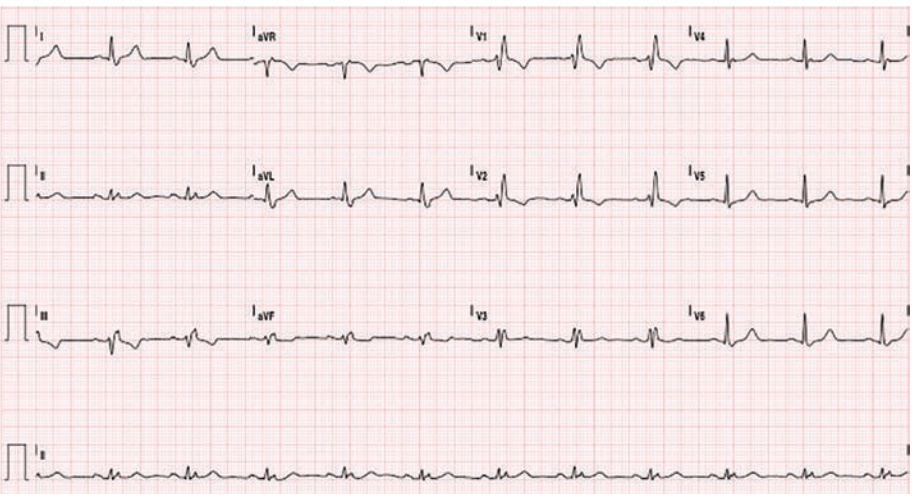

Figure 1. Right bundle branch block (RBBB).

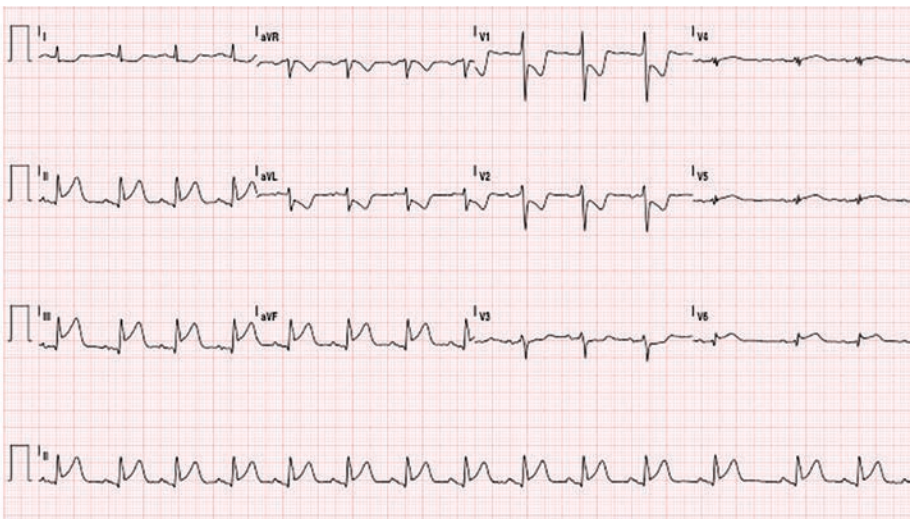

Figure 2. Posterior myocardial infarction (MI). In this case, it is also an inferior (inferior given the ST-elevations in II, III, and AVF) as well as lateral (given the ST-elevations in V5 and V6) MI.

Case #2. The diagnosis is posterior MI (Figure 2). Features include:

- Tall R wave in V1 or V2.
- ST depressions in the anterior leads, V1-V3. These are the equivalent of ST elevations from an infarcting posterior wall.
- ST-elevation in V7-V9 (not pictured). These are posterior leads that one can place below the scapula on the back of the patient.

There are also ST elevations in the inferior leads, II, III, and aVF, meaning this case is not just a posterior MI, but an inferior-posterior MI.

Case #3. The diagnosis is Wolff-Parkinson-White (Figure 3). Features include:

- Short PR interval (<120 ms) – caused by ventricular

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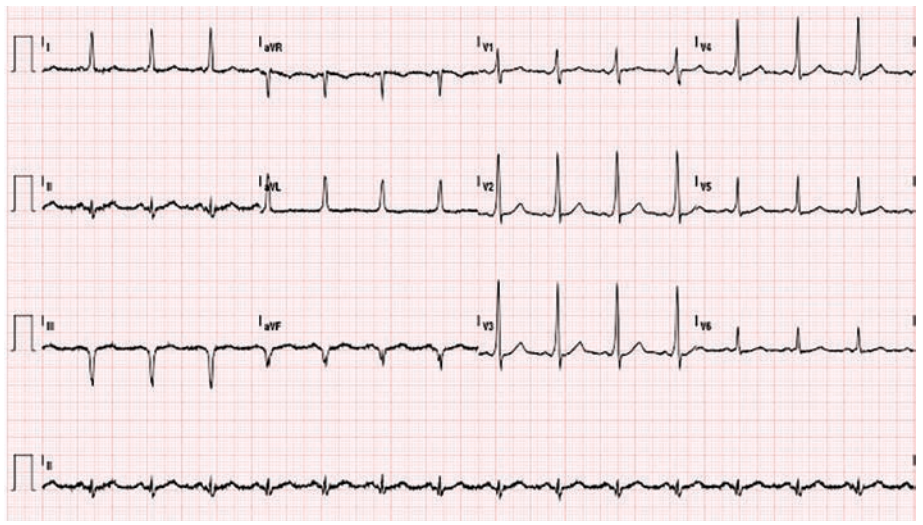

Figure 3. Wolff-Parkinson-White (WPW) syndrome.

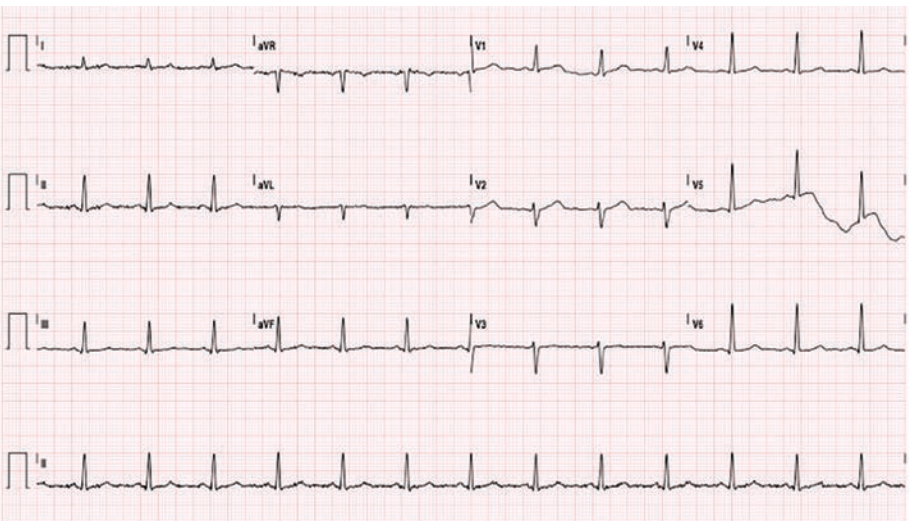

Figure 4. V1-V3 lead reversal.

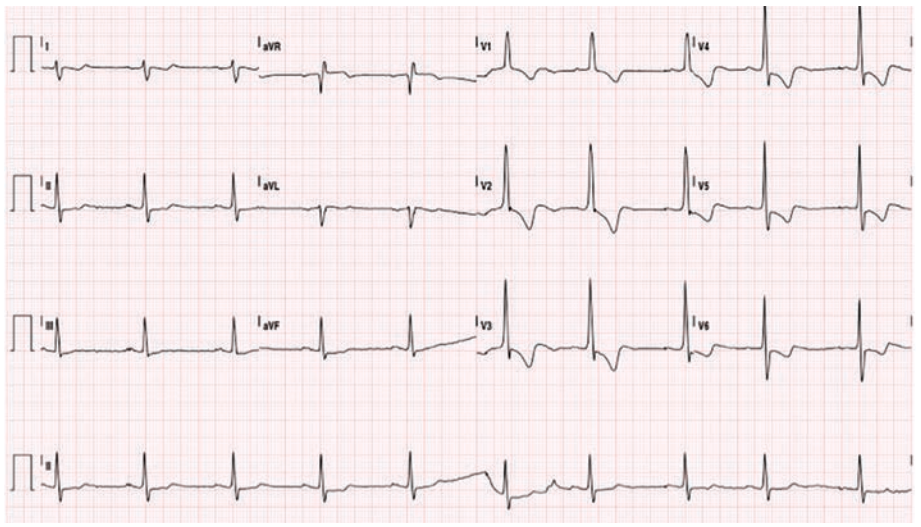

Figure 5. Right ventricular hypertrophy.

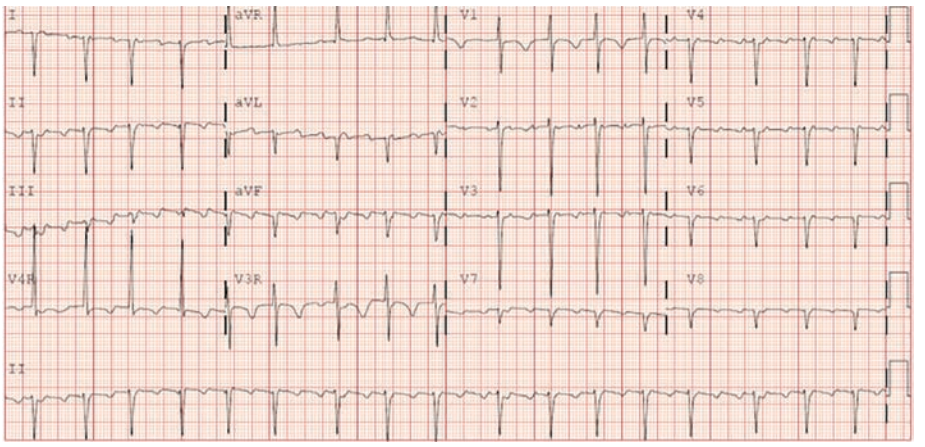

Figure 7. Dextrocardia. There is also atrial flutter, which need not occur with dextrocardia, but is present here.

preexcitation by conduction through an accessory pathway.
- Delta waves – also caused by preexcitation.

From an electrophysiology perspective, this case involves conduction through a left-sided accessory pathway. That conduction leads to early left-to-rightward depolarization and results in a positive deflection in V1. The depolarization through this accessory pathway leads it to depolarize the ventricular myocardium directly, which is slow-conducting as opposed to the fast-conducting His-Purkinje system. Thus, the positive deflection has a slow upslope, resulting in the signature "delta wave," a slow, triangle-shaped, upward depolarization preceding the steep upstroke of the regular QRS complex. It is best seen above in V1-V3.

Case #4. The diagnosis is V1-V3 lead reversal (Figure 4). It is easily identified, as V3 has a normal V1 morphology, in which there is a dominant S wave. Also, there is no physiology in which the R wave progression makes sense; it is not possible for there to be increased rightward amplitudes (as in "V1" above), then slightly rightward amplitudes (as in V2), then more rightward amplitudes again (as in "V3"), then significantly more leftward amplitudes again (as in V4-V6). The only way to explain this aberrant R wave progression is via technical error, specifically, V1-V3 lead reversal.

Case #5. The diagnosis is right ventricular hypertrophy (Figure 5). Features include:
- Tall R wave in V1 (R>S, or R wave >7 mm).
- S in V5 or V6 >7 mm – representing depolarization of an enlarged RV.
- Right axis deviation >110.
- Usually a drop in the R to S ratio across the precordium.
- QRS <120 mm (in other words, not caused by RBBB).

Supporting criteria include:
- Right ventricular strain pattern, which includes the ST depressions and T-wave inversion in V1-V3.
- S1Q3T3 pattern.
- Right atrial abnormality.

Not all of the above are seen in the example ECG, but there is a tall R wave in V1, right axis deviation, and a prominent right ventricular strain pattern, as represented by the T-wave inversion in the precordial leads.

Case #6. The diagnosis is Duchenne muscular dystrophy (Figure 6).[1] Features include:
- Tall R wave in V1.
- Deep Q waves in the lateral leads (V4-V6).

Supplemental features include a short PR interval and sinus tachycardia.

Case #7. The diagnosis is dextrocardia (Figure 7). Features include:
- Right axis deviation.
- Progressively decreasing R wave progression in the precordial leads.
- Positive R wave in aVR.
- Negative p wave and negative QRS in I and AVL (although this is not present in this case, since there is atrial flutter as well.)

Conclusion

Remembering the differential diagnosis for a tall R-wave in V1 has, historically, been difficult. But it doesn't need to be. The four mechanistic categories approach is straightforward. Divide the differential into four categories by mechanism, and remember a common and an uncommon etiology for each mechanism: (1) increased muscle mass, including hypertrophic cardiomyopathy (common) and right ventricular hypertrophy (uncommon); (2) decreased muscle mass, including posterior infarction and muscular dystrophy; (3) electrical, including RBBB and WPW syndrome; and (4) technical, including V1-V3 lead reversal and dextrocardia. Deduce the specific diagnosis through an understanding how the mechanism of each diagnosis leads to its signature ECG findings. ■

Reference

1. Slucka C. The electrocardiogram in duchenne progressive muscular dystrophy. *Circulation*. 1968; 38: 933-940.

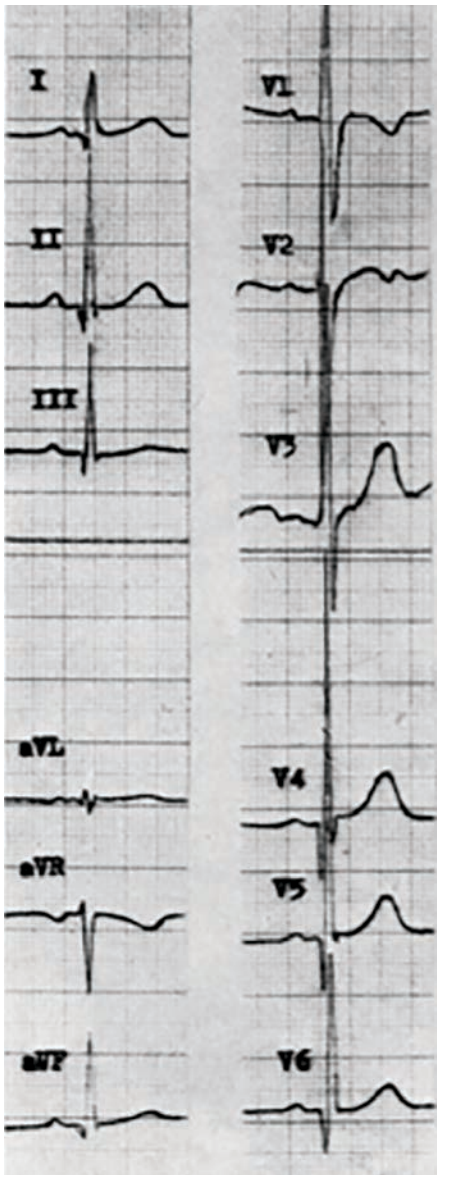

Figure 6. Duchenne muscular dystrophy. This example ECG is reproduced from an early article describing this finding. Reprinted with permission from Slucka C. The electrocardiogram in duchenne progressive muscular dystrophy. *Circulation*. 1968;38:933-940.