# UNDERSTANDING ST EQUIVALENTS ON ECG PART II

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#### **ABSTRACT**

In reality, many acute myocardial infarction patients with ST deviations observed in other conditions in which the characteristics of these ECG do not meet the criteria of STEMI. They are called: ST equivalents. STEMI equivalents represent coronary occlusion without meeting the traditional STE criteria and are equally important to recognize in a timely fashion. Emergency physicians must know to involve interventional cardiologists for patients with dynamic ECG changes, persistent ischemic chest pain, hemodynamic instability, and STEMI equivalent patterns that require emergent PCI to minimize morbidity and mortality. This is the reason why we would like to propose this review to present some common ST equivalents for physicians and cardiologists to apply to the clinical practice. These are: de Winter ST/T complex, Wellens syndromes, ST-elevation in lead AVR, LBBB with Sgarbossa criteria, isolated posterior MI, T waves upright in V1. This review is the part two of the topic.

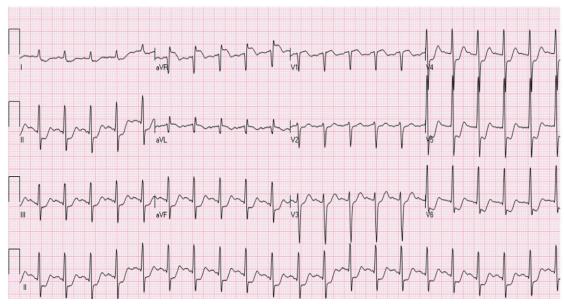
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#### 1. ST elevation in aVR

Lead aVR on electrocardiogram as a unipolar and augmented limb lead can present valuable and specific information from the right upper portion of the heart, including the basal portion of the interventricular septum and consequently, a transmural infarction in

this area theoretically produces ST elevation in lead aVR. This lead is electrically in opposition to leads I, II, aVL, and V3-V6, so when the ST in aVR is elevated, the ST in these leads may be depressed. In terms of anatomy, the left main coronary artery (LMCA) branches into the LAD and left circumflex arteries, supplying blood to most of the anterolateral and septal regions of the left ventricle. Due to myocardial ischemia or injury, LMCA narrowing reduces blood flow to a significant region of the heart, causing electrical abnormalities that are detectable by an ECG. The positive end of lead aVR points in the direction of the patient's right shoulder, so if the LMCA is obstructed/narrowed, the septal branches of the LAD would also be affected. It is thought that LMCA obstruction leads to the basal septal ischemia or injury, resulting in right superior pointing injury vector causing ST segment elevation in lead aVR. Gorgels et al. suggested that ischemia of the basal interventricular septum should cause ST elevation in leads aVR and V1. Thus, both proximal LAD obstruction and LMCA occlusion should cause ST segment elevation in leads aVR and V1 [3]. Hence, one of the two main mechanisms-diffuse antero-lateral subendocardial ischemia with reciprocal change in aVR or transmural infarction of the basal region of the heart-is thought to be the cause of STE-aVR. The clinical implication is that significant left main coronary artery disease or proximal LAD disease is indicated by STE in aVR (+/V1) in conjunction with ST depression in other leads. It is an urgent clinical situation that indicates a serious clinical condition, especially when the patient exhibits hemodynamic compromise [1, 4-6].



**Figure 1.** ECG showed sinus tachycardia with ST elevation in lead aVR and ST diffuse depression in leads V3-V6, II, III and aVF. The coronary angiography revealed significant LMCA stenosis [5].

# 2. LBBB with Sgarbossa criteria

Left bundle branch block (LBBB) is a common electrocardiographic (ECG) abnormality seen in patients whose normal cardiac conduction down both anterior and posterior left fascicles of the His-Purkinje system is compromised. Although LBBB is often associated with significant heart disease and is often the result of myocardial injury, strain or hypertrophy, it can also be seen in patients without any particular clinical disease. New onset LBBB in the proper setting of concerning clinical symptoms should always be considered a sign of pathology and can indicate myocardial infarction. New LBBB is considered an ST-segment elevation equivalent in patients presenting with chest pain. Although the QRS and ST segments of an ECG are traditionally regarded as uninterpretable in the presence of LBBB, emerging Sgarbossa criteria have been developed allowing some interpretation of ECGs despite LBBB [12, 13].

In the setting of acute myocardial infarction, there are a set of criteria called

Sgarbossa criteria which can be applied to the ECG to increase predictive value for or against myocardial infarction. These criteria are not as good as ST-segment elevation in the absence of LBBB. Their sensitivity is only 49%, but specificity is greater than 90%.

- 1. Concordant ST elevation greater than 1 mm in leads with a positive QRS complex (5 points)
- 2. Concordant ST depression greater than 1 mm in V1 to V3 (3 points)
- 3. Discordant ST elevation greater than 5 mm in leads in a negative QRS complex (2 points)

Three or more points means acute myocardial infarction [13].

Modified Sgarbossa criteria were introduced by Smith et al in 2012 and validated in 2015, replaced the third original Sgarbossa criteria with an ST/S ratio less than -0.25. The sensitivity of the modified criteria increases to 80% without affecting specificity. Furthermore, the modified Sgarbossa criteria do not utilize a point system, instead, it only requires 1 of 3 criteria to be considered positive.

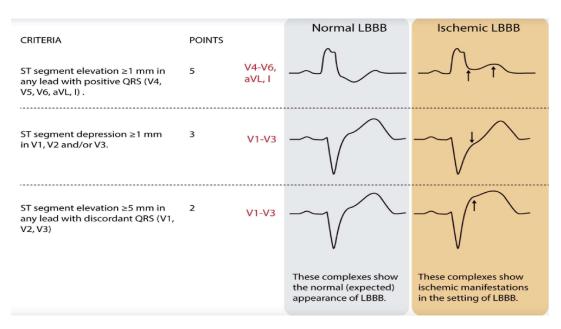


Figure 2. Sgarbossa criteria [13]

The reason of the replacement of the criteria 3 in Sgarbossa criteria was that the anterior STEMI caused by acute left anterior descending artery occlusion results in ST-segment elevation in leads V1 to V4, as well as in any or all of leads V5, V6, I, and aVL when occlusion is proximal to the first diagonal artery. In left bundle branch block, the normal discordance results in ST-segment elevation in leads V1 to V4 at baseline. Therefore, in the setting of a mid left anterior descending artery occlusion, the diagnosis of STEMI will rely exclusively on excessive discordance in leads V1 to V4. Sgarbossa's weighted criteria give only 2 points for excessive discordance and thus will "miss" a large number of anterior STEMIs, as they did in Smith's study [12, 14].

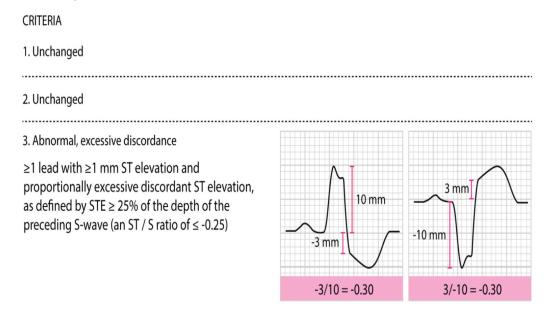


Figure 3. Modified Sgarbossa criteria [14]

#### 3. Posterior MI

Posterior MIs are easily missed because of the absence of any STE. Posterior involvement is estimated to occur in 15-21% of all acute myocardial infarctions<sup>8</sup> and in isolation ~3% of the time, typically due to occlusion of the left circumflex or right coronary arteries. Since there are no leads that look directly at the posterior wall, the associated ECG changes are reciprocal changes seen in the anterior leads V1-V3.7 Typical ECG changes include STD (reciprocal STE), tall R-waves (reciprocal Q-waves), and prominent positive T-waves (reciprocal terminal T-wave inversions). A posterior ECG should be obtained, and STE  $\geq$  0.5 mm ( $\geq$  1 mm in men < 40 years) in V7, V8, or V9 is diagnostic of a posterior MI. Note that the absence of elevations in the posterior leads does not exclude a posterior MI [2, 7].

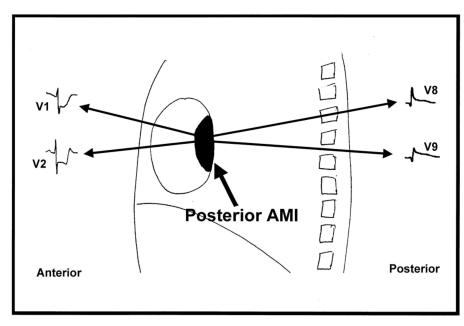
The ECG criteria of Acute MI of the Posterior Wall:

Standard 12-Lead ECG:

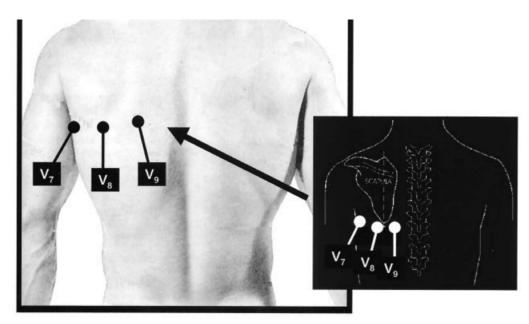
- ST segment depression\* (Horizontal > Downsloping/Upsloping)
  - Prominent R wave\*
  - R/S wave ratio > 1.0 in lead V2 only
  - Prominent, upright T wave\*

- Combination of horizontal ST segment depression with upright T wave\*
- Co-existing acute inferior or lateral MI *Additional-Lead ECG* (Posterior Leads V7, V8 & V9) >/= 1 mm ST segment elevation

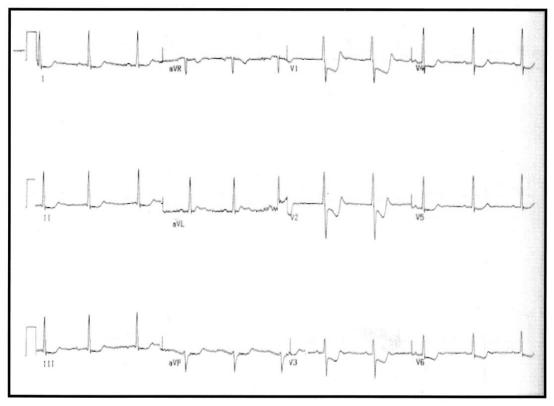
From the perspective of the standard 12-lead ECG, the "typical" electrocardiographic findings indicative of acute transmural myocardial infarction will be reversed. This reversal results from the fact that the endocardial surface of the posterior wall faces the anterior precordial leads (V1 through V3) in the standard 12-lead ECG. In other words, STD, prominent R waves, and upright T waves in leads V1 through V3 - "when reversed"-represent STE, Q waves, and T wave inversions, respectively, of acute PMI. If one considers the "reversed nature" of these electrocardiographic abnormalities when applied to the posterior wall, the findings assume a more recognizable, ominous meaning. In the setting of acute PMI (Figure 10), the 12lead ECG in the right precordial chest leads (V1 and V2) will display STD with a large R wave; if viewed from the posterior perspective of the thorax, these same findings are "reversed" and indicate acute transmural infarction of the posterior wall of the left ventricle, with the easily discerned STE [2]



**Figure 4.** The posterior perspective of the thorax [2]



**Figure 5.** Placement of the posterior thoracic leads: Lead V8 is placed on the patient's left back at the tip of the scapula. Lead V9 is placed halfway between lead V8 and the left paraspinal muscles. Lead V7 is used by some clinicians and is placed at the level of lead V6 at the posterior axillary line. [2]



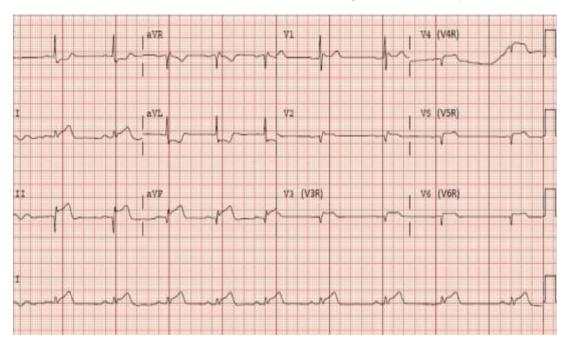
**Figure 6.** ST depression in leads I, II, aVF, and V1 to V6, the ST depression in leads V1 to V3 with prominent R waves [2].

### 4. T upright in V1

The standard 12-lead ECG provides information on the left ventricle but yields limited information on the right side of the heart. Leads V<sub>1</sub> and V<sub>2</sub> on the standard ECG provide only a partial view of the right ventricle free wall. Upright T wave in lead V1 was defined as a positive deflection of 0.15 mV or greater [8, 9]. Normally the T wave is inverted or flat in lead V1. An upright T wave in V1 may be considered a normal variant. However assuming that there is no ventricular hypertrophy or LBBB, a large, upright T wave in V1 may be abnormal if:

- The T wave in V1 is taller than the T wave in V6 (This is referred to as a loss in precordial T wave balance) and/or
  - The upright T wave in V1 is new [10]

T wave upright in V1 could be explained by the repolarization of the posterior wall secondary to ischemia. The ECG findings suggestive of right ventricular myocardial infarction (RVMI) on the standard 12-lead ECG include ST elevation in leads II, III, and aVF with reciprocal ST depression in the lateral leads. Characteristically in RVMI, the ST elevation in lead III is greater than in lead II, and the ST elevation in lead aVF is greater than the ST depression in lead V<sub>2</sub>. Right-sided precordial leads are critical to the evaluation of suspected RVMI. Using right-sided precordial leads, ST-segment elevation in lead  $V_AR \ge 1.0 \text{ mm}$ is diagnostic of RVMI. The ECG finding of ST elevation in lead V<sub>4</sub>R for diagnosis of RVMI has 100% sensitivity, 87% specificity, and 92% predictive accuracy [9, 11].



**Figure 7.** ECG showed upright T wave in lead V1 and ST elevation in leads V4R, V5R, V6R [9]

### **CONCLUSIONS**

It is crucial to remember that an ECG is not 100% sensitive for coronary occlusion and that the absence of ST elevation does not rule

out an myocardial infarction. Understanding these ST equivalents on ECG plays an important role in management patients with acute coronary syndrome.

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