LEFT BUNDLE BRANCH BLOCK AND ACUTE CORONARY SYNDROME

On Hellespont, guilty of true-love's blood,
In view and opposite two cities stood,
Sea-borderers, disjoined by Neptune's might;
The one Abydos, the other Sestos hight.
At Sestos Hero dwelt; Hero the fair;

Whom young Apollo courted for her hair,
And offered as a dower his burning throne,
Where she should sit for men to gaze upon...

... Amorous Leander, beautiful and young,
(whose tragedy divine Musaeus sung,)
Dwelt at Abydos; since him dwelt there none
For whom succeeding times make greater moan.
His dangling tresses, that were never shorn,
Had they been cut, and unto Colchis borne,
Would have allured the vent'rous youth of Greece
To hazard more than for the golden fleece...

Christopher Marlowe, Hero and Leander, c. 1593

Hero was a young priestess in the temple of the goddess Aphrodite, who lived in Sestos on the shores of the Hellespont. She was in love with Leander who lived in Abydos on the opposite bank of the Hellespont. Because of Hero's position of priestess of Aphrodite she was not permitted to marry or even to see Leander as she had to remain a virgin. So every night, in secret Leander would swim across the strait to be with Hero in her tower where she lived. Hero would guide Leander's way with a flaming torch. They would lie in each other's arms for a few hours before Leander would have to swim back to Abydos before dawn. It happened that a great storm arose one winter's night, which extinguished Hero's torch. She could not relight it no matter how much she frantically tried. Without the torch light to guide him, Leander became disoriented in the towering waves and tragically drowned. Hero found his body washed up on the shore the next morning. So distraught was she that she threw herself off the balcony of her tower and killed herself, dashed on the rocks far below.

The story of Hero and Leander is one of the most sorrowful in Greek mythology. When we encounter a patient with suspected STEMI, but who has a pre-existing LBBB we find ourselves in a most treacherous and uncertain situation. Fortunately we have a light to guide us in the form of the Sgarbossa Criteria. Sadly however, these criteria are, like Hero's guiding torch, not one hundred percent reliable.
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**Introduction**

Patients with a suspected acute coronary syndrome and left bundle branch block (LBBB) present a difficult diagnostic and therapeutic problem, as the presence of a complete LBBB makes the ECG diagnosis of myocardial infarction difficult.

The determination of myocardial infarction in patients with LBBB is additionally important as these patients may be at higher risk of death from myocardial infarction and congestive heart failure, as compared with patients without bundle branch block.

Traditional guidelines recommend that patients with new (or presumed new) LBBB undergo early reperfusion therapy; however only a minority of these patients with LBBB are ultimately diagnosed with acute myocardial infarction - regardless of LBBB chronicity, and a significant proportion of patients will not have an occluded culprit artery at cardiac catheterization.

Current thinking therefore suggests a more judicious diagnostic approach among clinically stable patients with LBBB who do not have electrocardiographic findings highly specific for ST-segment elevation myocardial infarction.

The decision to intervene therefore should not be solely based on the presence of a typical LBBB in isolation, but rather on a combination of one or more of:

- Clinical features:
- LBBB with associated specific ECG changes according to the Sgarbossa Criteria.
- Echocardiography
- Serum troponin levels

**Pathophysiology**

In contrast to the right bundle branch, which is a discrete structure that can be injured acutely with a small focal insult, the left bundle branch is a large and diffuse structure and so typically requires a much larger insult to lead to acute injury.

When a new LBBB is caused by myocardial infarction, the site of infarction is usually anterior or anteroseptal, with the infarction involving a large segment of myocardium. Inferior or posterior infarctions do not commonly result in a new LBBB. It may occur if there is involvement of the more proximal portion of the conduction system supplied by the atrioventricular nodal artery.

Most cases of LBBB in myocardial infarction however are not the result of focal infarctions, because either a discrete lesion just distal to the bundle of His or extensive myocardial...
damage involving a large portion of the distal conduction system including both fascicles would be required to cause the LBBB. Although LBBB can occur de novo in myocardial infarction, it is more often a pre-existing marker of underlying structural heart disease, and thus is more reflective of the patient’s baseline cardiovascular risk.

The chronicity of LBBB is not possible without reviewing previous ECGs, as the onset of LBBB usually is asymptomatic.

LBBB in myocardial infarction may be transient or permanent, although most cases of permanent LBBB are not the result of an acute transmural infarction, because true myocardial infarction associated LBBB results in very high mortality!

**Clinical Assessment**

Important aspects of the clinical assessment include:

- Assessing the patient’s **risk factors** for CVS disease
- Assessing the patient’s acute presentation for clinical features suggestive of a possible myocardial infarction:
  - Chest pain, (nature).
  - Pallor/ ashen appearance
  - Patient apprehension
  - Diaphoresis
  - Abnormal vital signs
  - Evidence of acute heart failure

**Investigations**

*Blood tests*

1. FBE
2. U&Es/ glucose
3. Troponin I

Others as clinically indicated.
ECG: The Sgarbossa Criteria

The three classical Sgarbossa ECG criteria for myocardial infarction in the presence of a LBBB.

The presence of a complete LBBB makes the diagnosis of myocardial infarction by ECG difficult.

Sgarbossa’s Criteria can be used in cases of patients with a pre-existing LBBB a pacemaker or a new LBBB, to determine the likelihood of myocardial infarction (or more correctly - a STEMI equivalent) and hence aid in the decision of whether or not to pursue reperfusion therapy.

There are 3 independent Sgarbossa ECG signs of myocardial infarction during LBBB in patients with chest pain or history of CAD:

- **Concordant ST elevation ≥ 1 mm in *any* lead with a positive QRS**  (5 points)
- **ST depression ≥ 1 mm in any of V1 to V3**  (3 points)
- **Discordant ST elevation ≥ 5 mm in any lead with a negative QRS**  (2 points)

The higher the number of total points up to 10, the more likely the diagnosis of myocardial infarction.

For zero points there will still be a 16% chance of myocardial infarction.
For 10 points there will be an almost 100% chance of myocardial infarction.

For practical purposes a score of **3 or more** is generally treated as myocardial infarction, (or STEMI equivalent). A score less than this means the diagnosis is much less certain and **further evaluation** will be necessary.

The Sgarbossa’s Criteria have been found to have good specificity, but only moderate sensitivity.

*Modified Sgarbossa Rule*

Recently a revised rule has been proposed, in which the **third** Sgarbossa component (i.e excessively discordant ST-segment elevation as defined by ≥ 5 mm of ST-segment elevation in the setting of a negative QRS) is replaced by a different, more sensitive and specific measurement.  

This new rule is defined as a proportion of ST-segment elevation to S-wave depth (i.e the ST/S ratio) - as applied to **any** lead.

Replacement of the absolute ST-elevation measurement of greater than or equal to 5 mm in the third component of the Sgarbossa rule with an ST/S ratio less than - 0.25 greatly improves diagnostic utility of the rule for STEMI.

An **unweighted** rule using this criterion resulted in excellent prediction for acute coronary occlusion. *An example of the calculation is shown below:*

![Image](image_url)

Abnormal, excessive discordance, with the ST segment and T wave in the opposite direction from QRS. Method of measurement: ST segment is measured at the J point, relative to the PR segment. R wave and S wave are also measured relative to the PR segment.

*One drawback to this modification, however is its complexity!*
Echocardiography

When uncertainty exists, then an echocardiogram to look for focal wall motion abnormalities will suggest myocardial infarction.

Management

Suggested algorithm for suspected Myocardial Infarction and LBBB

Patients with a suspected ACS in the setting of LBBB represent a much more heterogeneous population than STEMI without BBB and present unique diagnostic and therapeutic challenges to the clinician.

Most patients will not have a myocardial infarction regardless of LBBB chronicity and likely would not benefit from urgent reperfusion therapy.

Current expert opinion suggests a more judicious approach to diagnosis among hemodynamically and clinically stable patients with LBBB who do not have ECG
findings highly specific for STEMI. This is may be achieved by patient selection via echocardiography and/ or serial troponin measurements.

**Disposition**

Patients with chest pain fulfilling, clinical, echocardiographic, biomarker or Sgarbossa criteria on ECG, (old or newer versions thereof), should be treated as STEMI equivalents.

Note also that patients with presumed new LBBB, who subsequently do not prove to have a STEMI equivalent, (or non-STEMI) - still have increased risk of mortality and so should have appropriately timely investigation and follow-up.
Appendix 1

Anatomy of the Bundle Branches:

The left bundle branch comprises the main left bundle which divides in the distal anterior and posterior fascicles. LBBB resulting from a myocardial infarction requires a lesion just distal to the bundle of His (1) or extensive myocardial damage involving a large portion of the distal conduction system that includes both the anterior and posterior fascicles (2 and 3). ¹
References


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