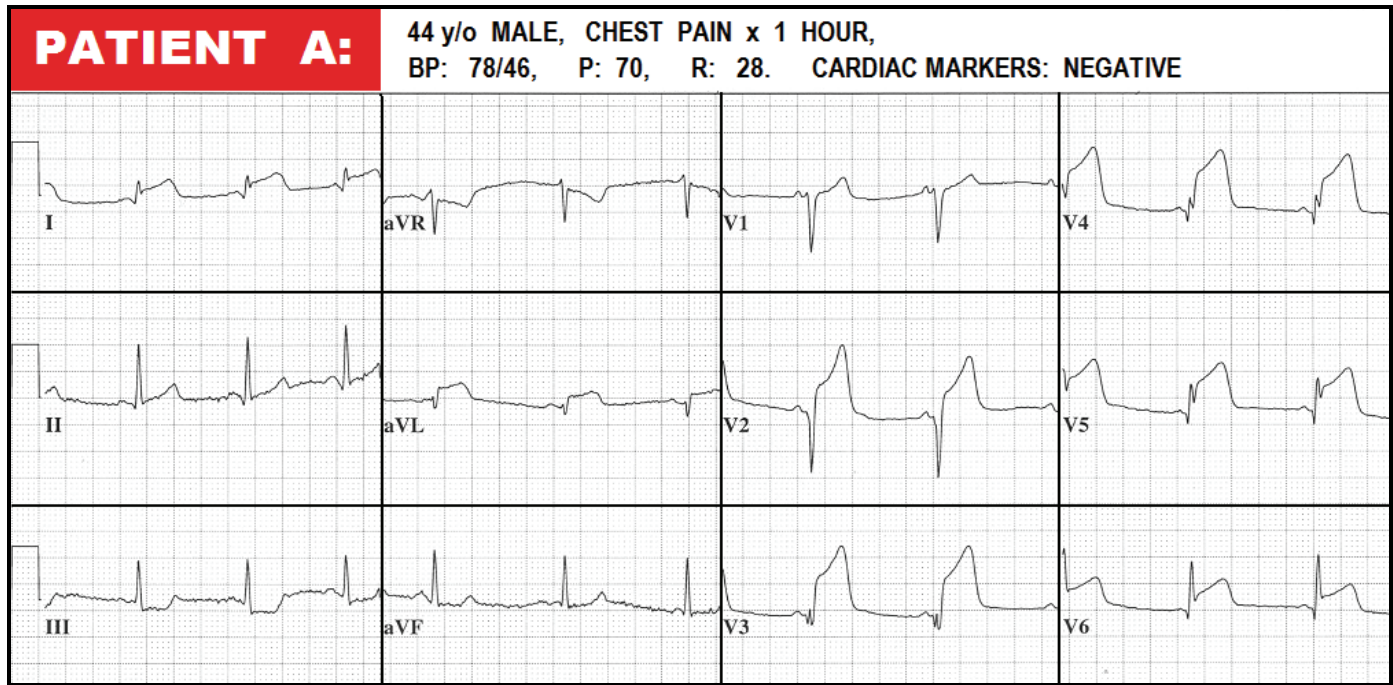


### CASE STUDY 4: CRITICAL DECISIONS SCENARIO

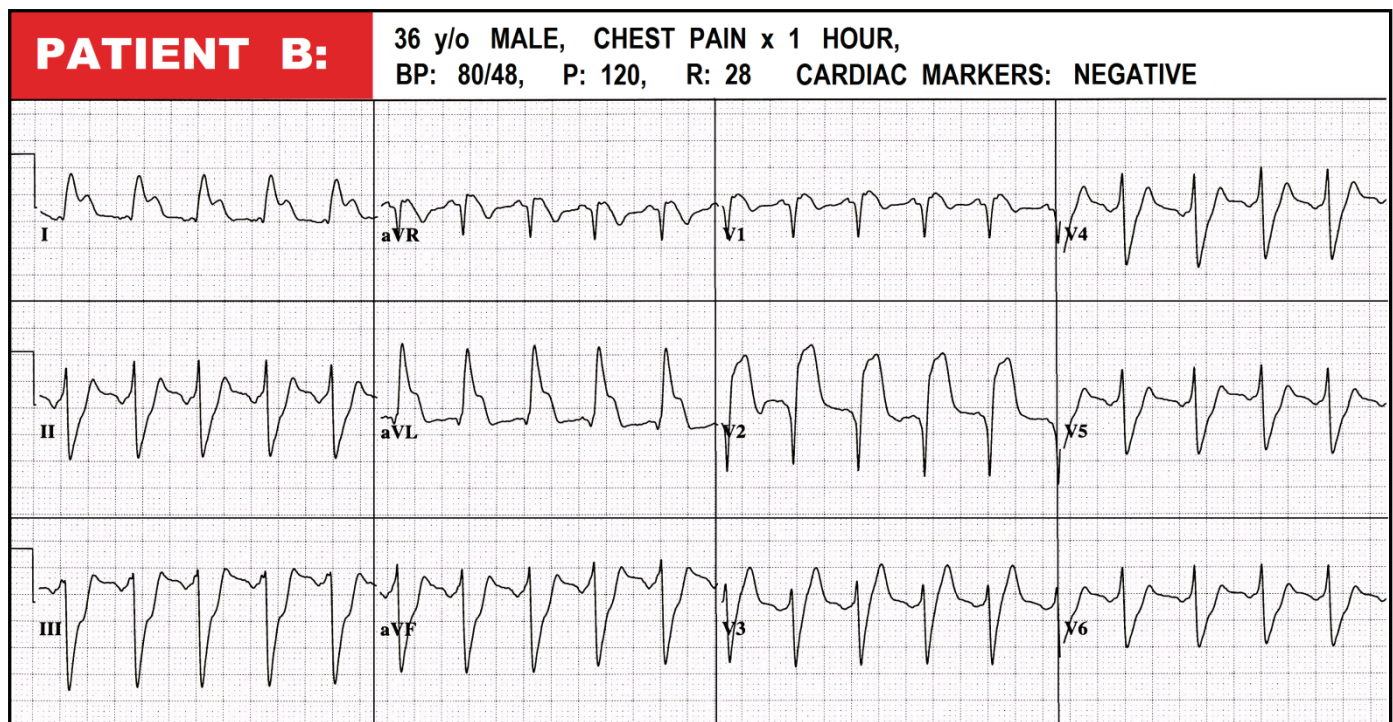
As per current AHA recommendations, your hospital's policy is to send every STEMI patient to the Cardiac Catheterization Lab for emergency PCI.

You are the ranking medical officer on duty in the ED when two acute STEMI patients arrive, ten minutes apart. The Cath Lab has one lab open, and can take ONE patient immediately. Both patients duration of symptoms and state of hemodynamic stability are similar.

**QUESTION 1: WHICH PATIENT WOULD YOU SEND TO SEND TO THE CATH LAB, STAT?**



-----OR-----



The Cath Lab Coordinator advises you they won't be able to accommodate the second patient until they've finished with the first. She informs you this will take at least *one hour, probably longer*.

**QUESTION 2: WHAT WILL YOU DO WITH THE PATIENT WHO DOES NOT GO TO THE CATH LAB FIRST?**

- A. Wait for the Cath Lab to finish the first patient, then send the second patient, while managing the patient's hemodynamic stability in the ED
- B. Administer thrombolytic therapy STAT in the ED, if the patient has no absolute contraindications
- C. Transfer the patient to another facility that is PCI capable.

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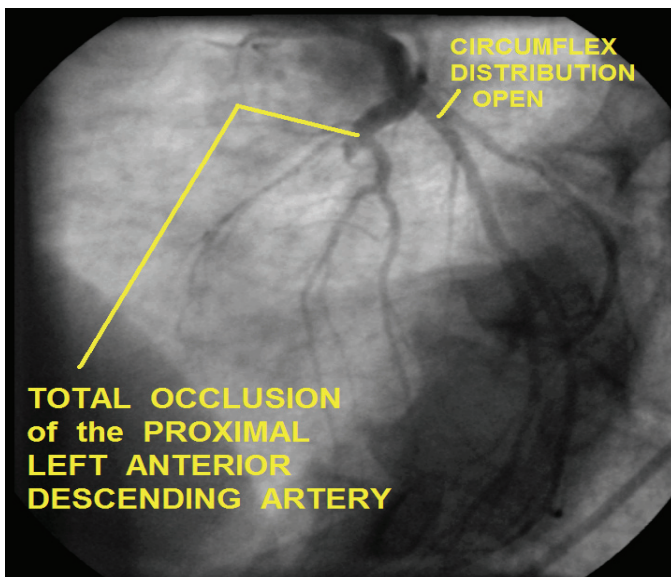
**ANSWER to QUESTION 1:**     *PATIENT B should be sent to the Cath Lab FIRST.*

**Rationale:** Based on the 12 Lead ECGs, both patients are suffering from *acute Anterior-Lateral Wall STEMI*. However the markers for *identifying the culprit artery*, present on each ECG, indicate:

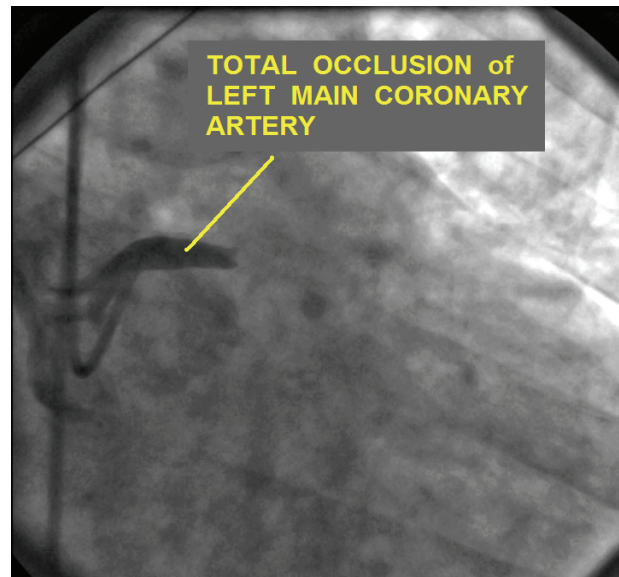
- **PATIENT A has a proximal occlusion of the LEFT ANTERIOR DESCENDING ARTERY (LAD). The LAD supplies approximately 40-50% of the Left Ventricular Muscle Mass.**
- **PATIENT B has a total occlusion of the LEFT MAIN CORONARY ARTERY (LMCA). The LMCA supplies up to 75% of the LV Muscle Mass in RIGHT DOMINANT Coronary artery systems, and up to 100% of the LV Muscle Mass in LEFT DOMINANT Coronary artery systems.**

Total LMCA obstructions involve occlusions of both the LAD and Circumflex arterial distributions, and are approximately three times more likely to result in cardiogenic shock and death than an isolated LAD occlusion. Angiography for both patients is shown below:

**PATIENT A – (LAD Occlusion):**



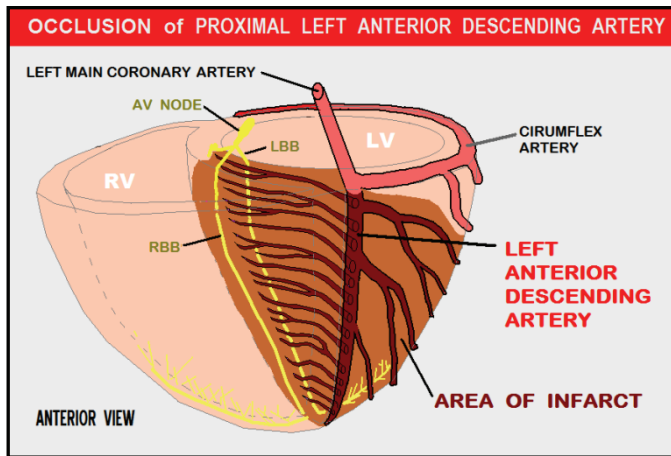
**PATIENT B – (LMCA Occlusion):**



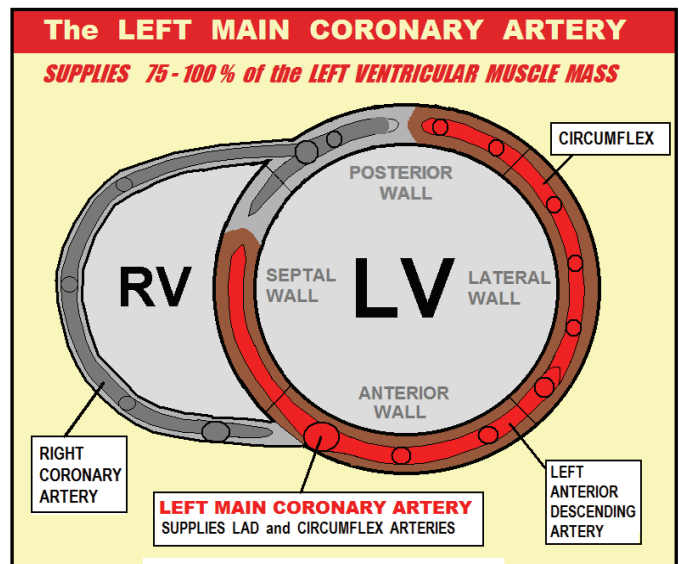
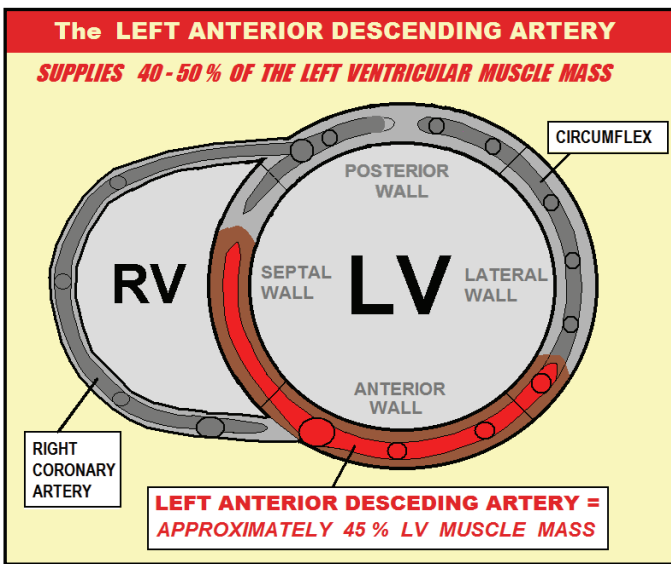
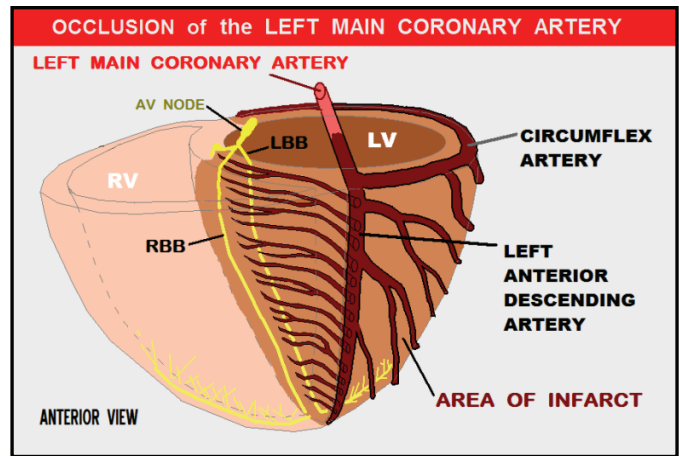


The images below illustrate the differences between proximal LAD and LMCA occlusions:

**PATIENT A - (LAD occlusion):**



**PATIENT B - (LMCA occlusion):**



STEMI caused by LMCA occlusion is rare, and for obvious reasons, carries a high mortality rate. Most patients who survive total LMCA occlusions fit into one or both of the following categories:

- Have developed *collateral circulation* from the RCA to the LAD and CX arterial distributions, or have undergone previous coronary artery bypass surgery (CABG), and have patent bypass grafts around the native LMCA
- Are “Right Dominant,” with large RCA distributions, whom are in close proximity to a medical center where rapid diagnosis of STEMI and reperfusion can be achieved.

☞ If you had chosen to send PATIENT A to the Cath Lab first, you fall into the category of nearly every health care professional who was surveyed! Most chose PATIENT A over PATIENT B, because the 12 lead ECGs of acute proximal LAD occlusions are more impressive; they usually display significantly more ST elevation than the ECGs of total LMCA occlusions.

Next, we’ll take a look at some specific ECG indicators of STEMI caused by LMCA occlusion.

**ECG Clues . . .** for **IDENTIFYING STEMI CAUSED BY LEFT MAIN CORONARY ARTERY occlusion:**

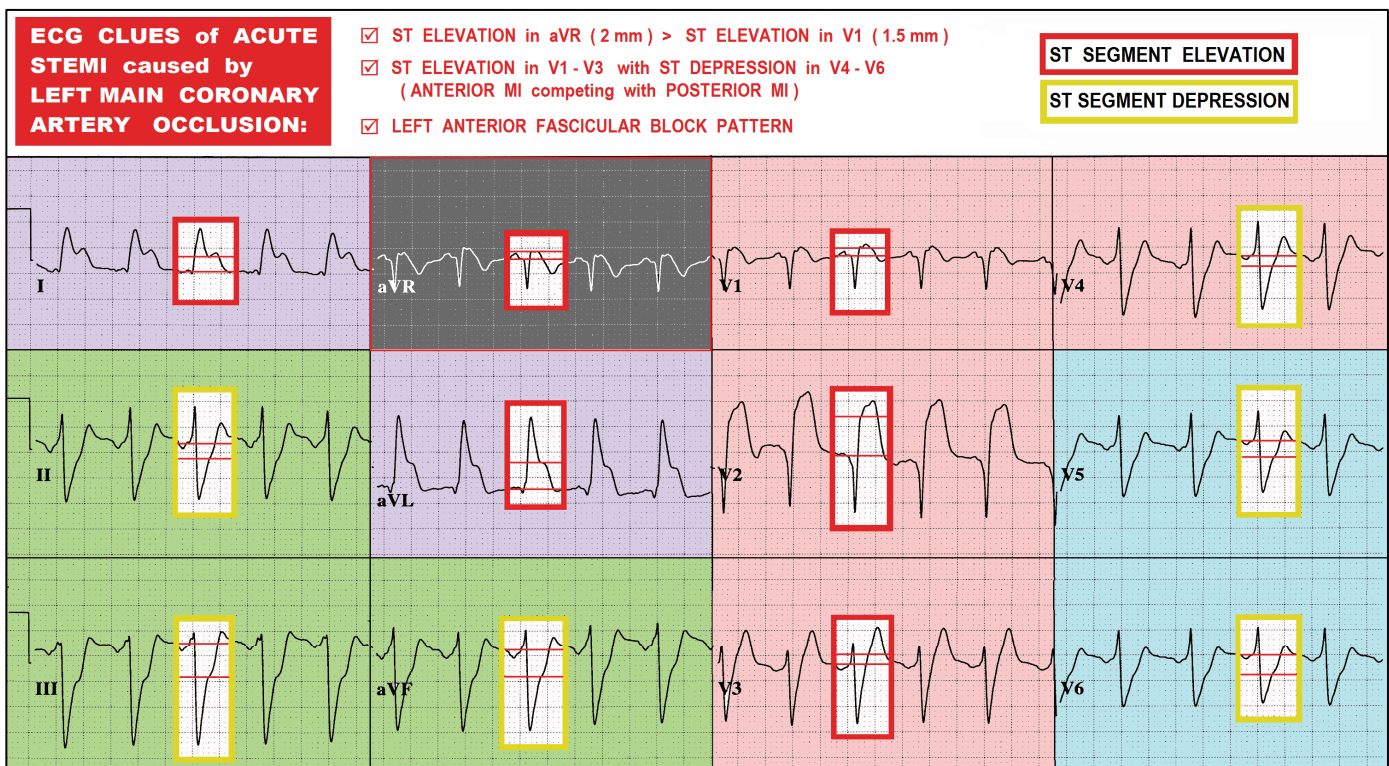
- ☑ ST ELEVATION in ANTERIOR LEADS (V1 - V4) and LATERAL LEADS (V5 & V6)
- ☑ ST DEPRESSION or ISOELECTRIC J POINTS may be seen in V LEADS . . . mainly V2 and/or V3 caused by *COMPETING FORCES* of ANTERIOR vs. POSTERIOR WALL MI.\*<sup>+</sup>
  - NOTE: it is very unusual to see ST DEPRESSION in V LEADS with isolated ANTERIOR WALL MI when caused by occluded LAD.
- ☑ ST ELEVATION in AVR is GREATER THAN ST ELEVATION in V1\*<sup>+</sup>
- ☑ ST ELEVATION in AVR GREATER THAN 0.5 mm
- ☑ ST ELEVATION in LEAD I and AVL (caused by NO FLOW to DIAGONAL / OBTUSE MARGINAL BRANCHES)\*
- ☑ ST DEPRESSION in LEADS II, III, and AVF. (in cases of LMCA occlusion of DOMINANT CIRCUMFLEX, leads II, III, and AVF may show ST ELEVATION or ISOELECTRIC J POINTS)\*<sup>+</sup>
- ☑ NEW / PRESUMABLY NEW RBBB, and/or LEFT ANTERIOR FASCICULAR BLOCK\*<sup>+</sup>

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\* Kurisu et al, HEART 2004, SEPTEMBER: 90 (9): 1059-1060  
<sup>+</sup> Yamaji et al, JACC vol. 38, No. 5, 2001, November 1, 2001:1348-54

A few comments on the information provided in the table shown above:

- The precordial leads can show ST elevation, ST depression, a combination of ST elevation and ST depression, or isoelectric ST segments. This is due to the competing forces of ANTERIOR WALL INFARCTION (ST elevation) vs. POSTERIOR WALL INFARCTION (ST depression).
- ST elevation of at least 0.5mm in lead aVR is reported in over 63% of the cases of acute LMCA occlusion described in current medical journals. ST elevation in lead aVR is due to ischemia of the basilar ventricular septum.
- In the ECGs of STEMI caused by LMCA occlusion that we present in this book, we note the pattern of Left Anterior Fascicular Block in every case.
- ALSO NOTE that in every one of our LEFT MAIN occlusion STEMI ECGs, there is either significant ST elevation or depression in EACH of the 12 leads.





Notice the similarities of the four ECGs shown below and on the next page. All are from FOUR DIFFERENT STEMI patients found to have total Left Main Coronary Artery occlusions in the Cardiac Cath Lab. **Commit these findings to memory, as your ability to rapidly identify and initiate reperfusion therapy in a patient suffering from STEMI caused by LMCA occlusion may have a significant impact on the patient's outcome.**

36 yr Male Caucasian Room:C- Loc:3	Vent. rate PR interval QRS duration QT/QTc P-R-T axes	123 96 130 310/443 * -53	BPM ms ms ms 43	Sinus tachycardia with short PR Left ventricular hypertrophy with QRS widening Cannot rule out Septal infarct , age undetermined Lateral injury pattern ***** ACUTE MI *****	<b>ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION</b>
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**ECG CLUES of ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION:**

- ST ELEVATION in leads I and aVL
- INCONSISTENCY of ST SEGMENT in leads V1 - V6 : V1 - V3 ST ELEVATION, V4 - V6 ST DEPRESSION (COMPETING FORCES of ANTERIOR vs. POSTERIOR M.I.)
- PATTERN of LEFT ANTERIOR FASCICULAR BLOCK ( POS. QRS lead I; NEG rS leads II, III )
- ST ELEVATION in lead aVR > 0.5 mm

43 yr Male	Vent. rate PR interval QRS duration QT/QTc P-R-T axes	183 * 106 240/418 * -34	BPM ms ms ms -18	Atrial fibrillation with rapid ventricular response with premature ventricular or aberrantly conducted complexes Left axis deviation ST elevation consider anterolateral injury or acute infarct ***** ACUTE MI *****	<b>ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION</b>
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**ECG CLUES of ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION:**

- ST ELEVATION in leads I and aVL
- INCONSISTENCY of ST SEGMENT in leads V1 - V6 : V1 - V2 ST ELEVATION, V3 - V6 ST DEPRESSION (COMPETING FORCES of ANTERIOR vs. POSTERIOR M.I.)
- PATTERN of LEFT ANTERIOR FASCICULAR BLOCK ( POS. QRS lead I; NEG rS leads II, III )



48 yr Male	Caucasian	Vent. rate PR interval QRS duration QT/QTc P-R-T axes	155 * 110 300/482 * -83 -34	<b>ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION</b>
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**ECG CLUES of ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION:**

- ST ELEVATION in LEADS I, aVL, V1 - V2, V4 - V6 with ST DEPRESSION in V3: (COMPETING FORCES of ANTERIOR vs. POSTERIOR M.I.)
- RIGHT BUNDLE BRANCH BLOCK PATTERN, with
- LEFT ANTERIOR FASCICULAR BLOCK PATTERN

Name: ID: Patient ID: Incident: Age: 37 Sex:	12-Lead 4 06 Oct 07 PR 0.154s QT/QTc P-QRS-T Axes aVR	HR 107 bpm 12:44:13 QRS 0.182s 0.332s/0.443s 89° -62° 44°	<ul style="list-style-type: none"> <li>• <b>*** ACUTE MI SUSPECTED ***</b></li> <li>• <b>Abnormal ECG *** Unconfirmed ***</b></li> <li>• Sinus tachycardia</li> <li>• Left anterior fascicular block</li> <li>• Cannot rule out Anteroseptal infarct.</li> </ul>	<b>ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION</b>
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**ECG CLUES of ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION:**

- ST ELEVATION in LEADS I, aVL, V1 - V6
- ST ELEVATION in aVR GREATER THAN 0.5 mm
- ST ELEVATION in aVR GREATER THAN LEAD V1
- LEFT ANTERIOR FASCICULAR BLOCK PATTERN

In the ECGs on this page, significant ST elevation is noted throughout the precordial leads. Both patients had large RCA distributions, which protected much of their posterior walls, leaving the *ST segment elevating forces* of Anterior Wall MI unopposed.

Conversely, in the ECGs on the previous page, ST elevation is noted only in leads V1 and V2. In leads V3 – V6, there is J point depression, with upsloping ST segments. ECGs on the previous page are examples of the “competing forces of concurrent ANTERIOR and POSTERIOR wall MI” -- both patients had large circumflex distributions supplying their posterior walls.



## CASE STUDY 4: CRITICAL DECISIONS SCENARIO

### CONCLUSIONS:

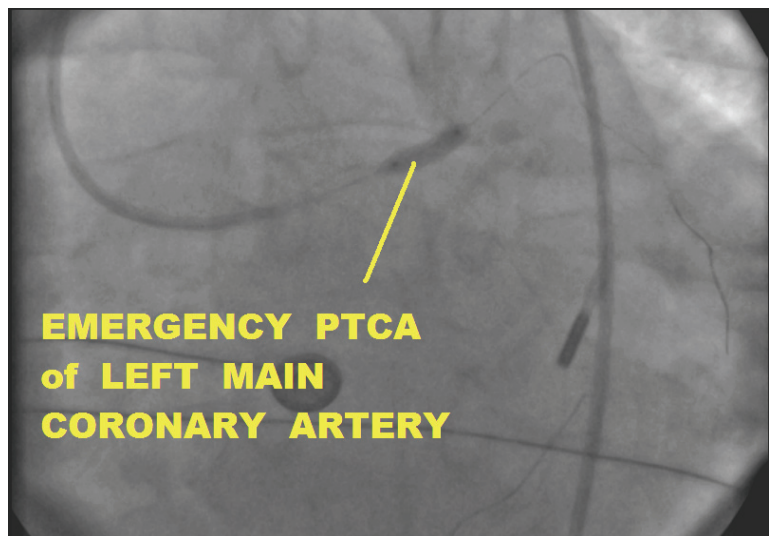
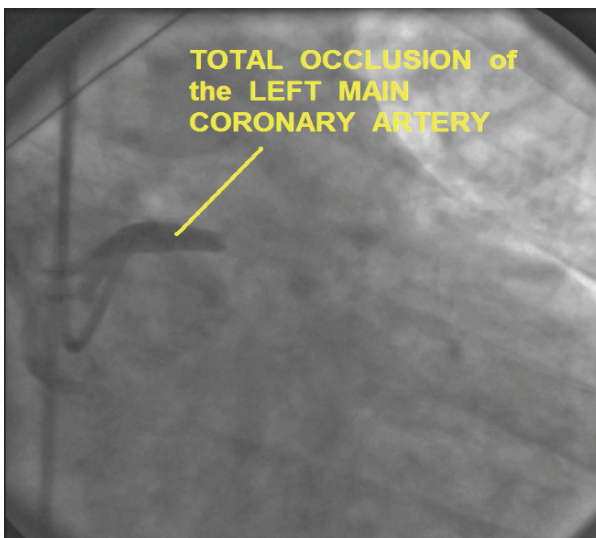
**QUESTION 1:** WHICH PATIENT SHOULD BE TAKEN FIRST FOR IMMEDIATE CARDIAC CATHETERIZATION for EMERGENCY PCI ?

**ANSWER:** PATIENT B was taken emergently to the Cardiac Cath Lab - both the ED physician and the Interventional Cardiologist correctly identified the EKG patterns of LMCA occlusion.

**QUESTION 2:** WHAT COURSE OF ACTION SHOULD BE TAKEN WITH THE PATIENT NOT CHOSEN TO BE SENT TO THE CATH LAB FIRST?

**ANSWER:** PATIENT A received thrombolytic therapy in the ED. It was determined that THROMBOLYTIC THERAPY would achieve the FASTEST ROUTE to REPERFUSION --  
-- *by at least 60 minutes.*

PATIENT B underwent emergency thrombectomy, PTCA and stenting of his Left Main Coronary Artery with a drug-eluting stent in the Cardiac Catheterization Lab. His angiography is shown below:



Despite the dismal mortality rate associated with STEMI from total LMCA occlusion, this patient survived and was later discharged. His EF is estimated at approximately 30%. He received an ICD, and is currently stable.

His survival of this ordeal is no doubt attributed to his quick arrival in the ED after his symptoms started, the rapid diagnosis of his condition, and his expedient triage to the cardiac catheterization lab for emergency PCI.

Case contributed by Humberto Coto, MD, FACC

