Who Needs the Cath Lab/Cards Consult?

A guideline from the Steve Smith's EKG Blog and the EMCrit Podcast

Activate the Lab for *unambiguous* STEMI (only clear STEMIs have a 90 minute CMS mandate)

Get Cardiology or Interventional Consultation for more complicated cases: difficult ECGs, subtle ST elevation, ST depression with ongoing symptoms, STEMI "Equivalents".

This requires a systematic approach, with buy-in from Cardiology that they will respond immediately to such requests for help. What do they get out of it? Fewer false positive activations and more activations for the subtle cases that need it.

Know that the ACC/AHA guidelines for NonSTEMI recommend < 2 hour cath for: 1) refractory ischemia 2) ischemia with hemodynyamic or electrical instability

> *Proviso: Many cardiologists do not understand these subtle ECG findings or pseudo-STEMI patterns. You must be a strong advocate! If you are worried, get serial ECGs, compare with an old ECG, and get a high quality contrast echocardiogram exam.* ***Persistent*** *occlusion of a significant epicardial coronary artery will nearly always have a wall motion abnormality if the echo quality is good, is done with contrast, and is read by an expert.*

I. **ACC/AHA Criteria**

ST-elevation at the J point in 2 contiguous leads that reaches the following thresholds:[1]

- Men < 40 years of age: 2.5 mm in V2-V3 and 1 mm in all other leads
- Men > 40 years of age: 2 mm in V2-V3 and 1 mm in all other leads
- Women: 1.5 mm in V2-V3 and 1 mm in all other leads

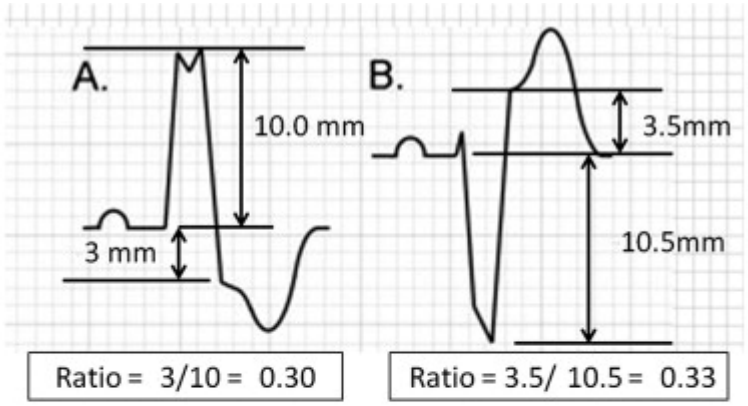
these criteria are only 45% sensitive for MI as measured by CK-MB, and about 70% sensitive for acute coronary occlusion, with perhaps 85% specificity. Beware of early repolarization, LVH, and LV aneurysm as false positives. Beware of subtle ST elevation as false negatives. Other less specific but more sensitive criteria require "new" ST elevation.

II. Left Bundle Branch Block

New LBBB alone is not an indication for cath lab activation. MI may also present in the context of old LBBB. Therefore, in stable patients, determine if there is a concordant ST segment, or an excessively discordant ST segment (see figure) and then use the algorithm below:

Activate if any of these three:[2]

1. In an unstable patient (hypotensive, Acute Pulmonary Edema, electrical instability, or looks sick)[3]
2. Sgarbossa Criteria (1 of the following)[4,5]
    - Concordant ST-segment elevation of 1 mm in at least 1 lead
    - Concordant ST-segment depression of at least 1 mm in leads V1 to V3
3. They have Smith-Modified Sgarbossa criteria[5] Any single lead with at least 1 mm of discordant ST elevation that is >/= 25% of the preceding S-wave. This does not apply to extreme tachycardia, pulmonary edema, hypertension, as they may have falsely elevated ST segment, although such ill patients should have a low threshold for cath lab activation anyway. Stabilize patient before assessing the ECG, if possible.

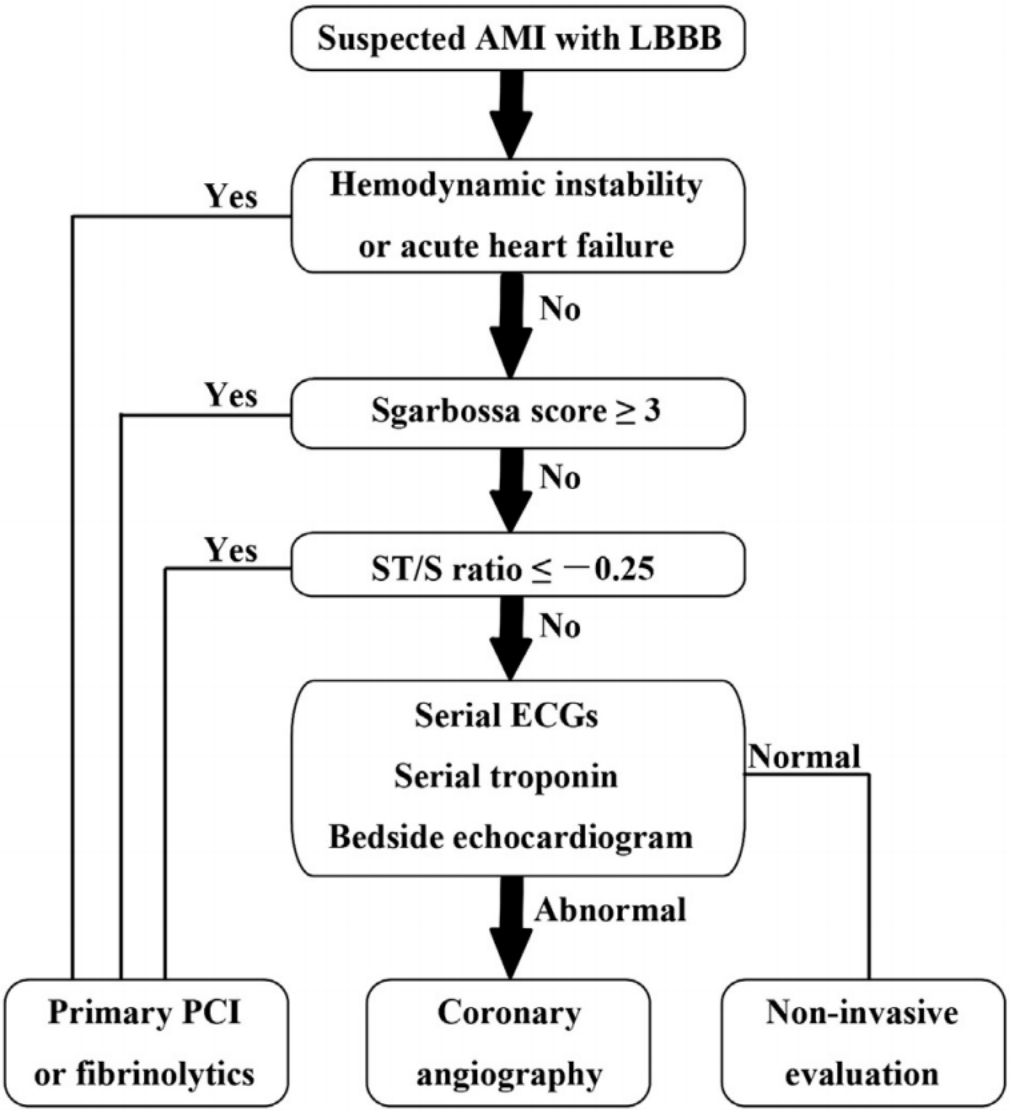


Sgarbossa-Smith Modification
Sgar(Sm)

Take absolute size of R or S Wave in leads with discordance. Take absolute size of ST Deviation (Dev). If Dev / (R or S) is > 0.25 in any one lead then Sgar(SM) is positive

Annals of Emergency Medicine Volume 60, Issue 6 , Pages 766-776, December 2012

Here is a nifty algorithm from Cai and Sgarbossa (2)
(https://www.mcfarlandclinic.com/file.cfm/media/cms/The_left_bundlebranch_block_puzzle__D98A0CC95BA67.pdf



Diagnosis and triage algorithm for patients with suspected AMI and LBBB.

III. **New Right Bundle Branch Block + LAFB**

New RBBB + LAFB is a very bad sign. It is highly associated with proximal LAD occlusion and bad outcomes.

See this paper by Widimsky et al[6], which shows the high association of RBBB, especially with LAFB, with LAD occlusion. **Furthermore, among 35 patients with acute left main coronary artery occlusion, 9 presented with RBBB (mostly with LAH) on the admission ECG. If there is STEMI, there will be pathologic ST elevation, but it may be downsloping and very difficult to discern, and can only be done with accurate identification of the end of the QRS.**

See:

http://hqmeded-ecg.blogspot.com/2014/11/chest-pain-and-right-bundle-branch-block.html

http://hqmeded-ecg.blogspot.com/2010/11/wide-complex-tachycardia-its-really.html

IV. Inferior Wall MI

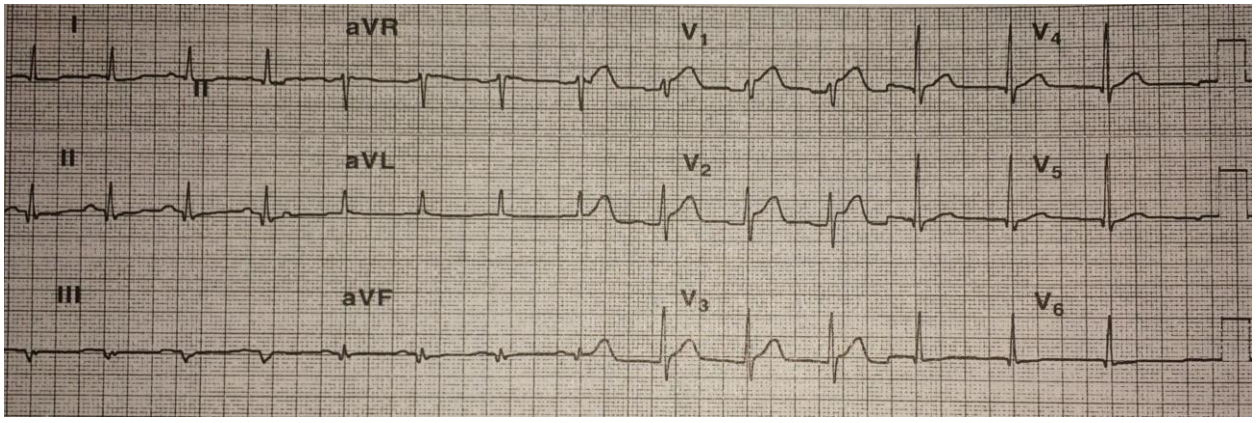
- Elevation of any degree in two contiguous leads (II, III, or aVF) with any amount of ST segment depression in aVL is highly suspicious for inferior MI. If there are well developed QS-waves, it is likely to be old MI with persistent ST elevation. LV aneurysm, LVH, WPW, and LBBB all have repolarization abnormalities that produce reciprocal ST depression in aVL. If these are not present, ST depression in aVL is highly sensitive and specific for acute inferior MI.

V. Right Ventricular Infarction?

Think of RV MI in any inferior MI, especially if it is due to an RCA occlusion (any ST depression in lead I is fairly sensitive and specific for RCA as the infarct artery). If the RCA is the infarct artery, consider RV MI especially if there is hypotension. It often shows as ST elevation in lead V1, and a right side ECG with ST elevation in right-sided leads, especially V4R, is diagnostic.

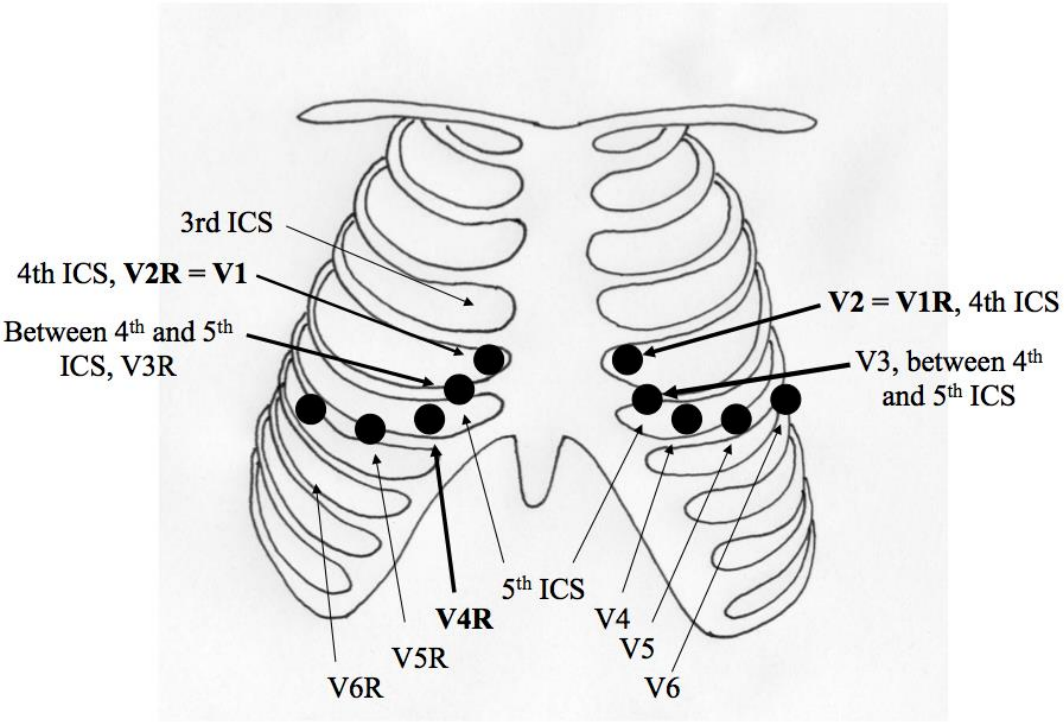
Isolated RV Infarction: It certainly exists, but is rare. It may occur with 1) isolated RV marginal branch occlusion or 2) old inferior MI (inferior wall is already dead) with NEW proximal occlusion of RCA. Also, even if all flow is cut off to RV marginal branch, there is usually good collateral flow from LAD. So most proximal RCA occlusions do not result in RV infarct physiology.

See this example of a patient with chest pain. He has old inferior MI (see Q-waves) with new ST elevation in (left-sided) V1-V3 due to new proximal RCA occlusion. This example may also be called a "pseudoanteroseptal MI" because the ST elevation extends out to V2 and V3



Look for any ST elevation in V1: this is fairly sensitive and specific for RV STEMI. Sensitivity is greatly reduced if there is ST depression in V2 and V3 (posterior STEMI), which attenuates the ST elevation which would otherwise show in V1

Consider adding leads V3R & V4R (5th intercostal space, mirror image of lead V4—)



Suggested cut point, though very dependent on proportion (R-wave amplitude): **Elevation of 0.5 mm in V3R/V4R**

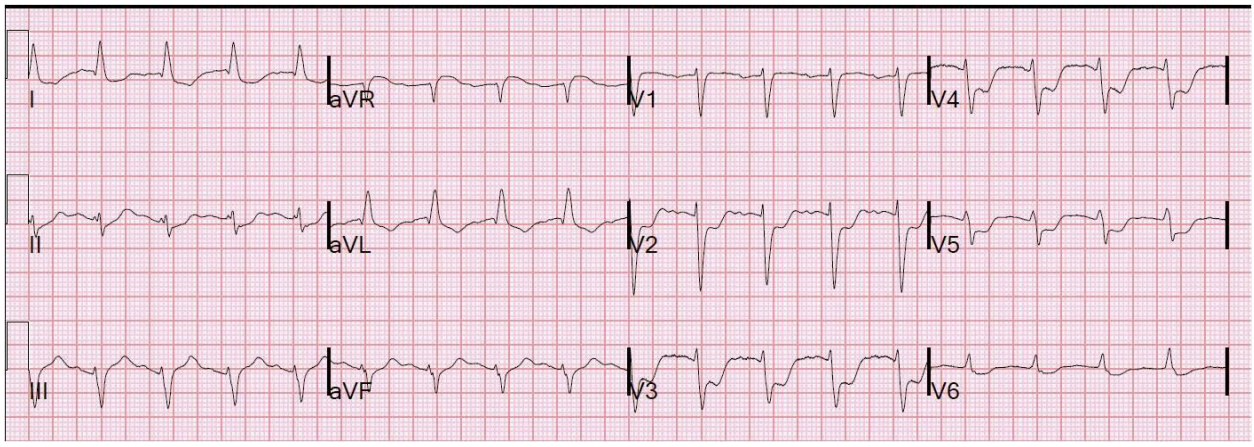
Myth: ~~When presenting with Inferior MI - Disproportionate ST segment elevation with greater ST elevation in lead III than in lead II is pathognomonic for an RVMI~~.[7] *This is completely false. This finding is sensitive, but not at all specific and not at all pathognomonic. STE in III greater than II suggests RCA over circumflex (by no means absolute). All RV MI come from RCA occlusion, but only from RCA occlusion proximal to the RV marginal branch and ONLY if the RV is not also supplied by the LAD, which it often is. Thus, STE in III > II says nothing about RV MI except that the RCA is involved. Proximal RCA occlusion does not imply RV MI physiology by itself. A minority of proximal RCA occlusions have RV MI physiology.*

Take home: RV MI usually has ST elevation in V1 UNLESS there is concomitant ST depression in V2 (posterior MI which attenuates the STE in V1).

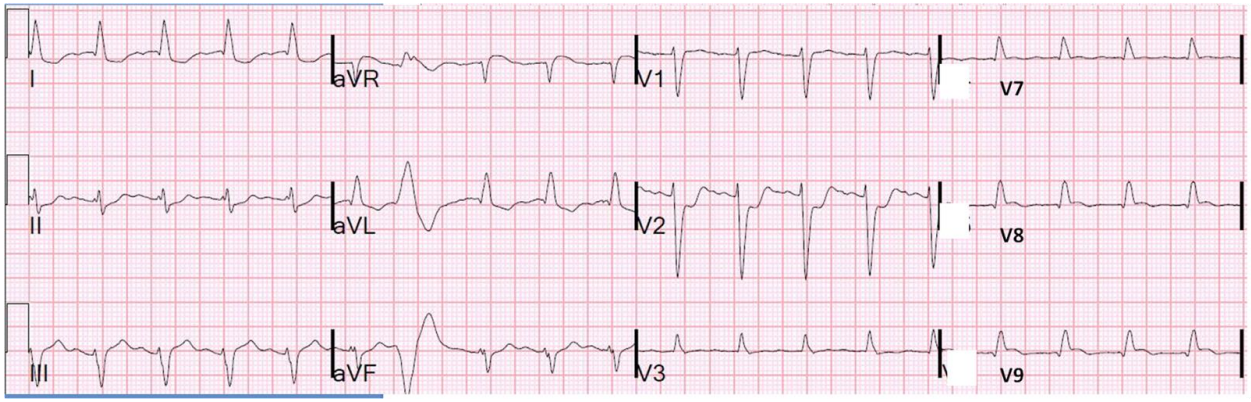
VI. Posterior MI (isolated)

- Precordial ST-depression ≥ 1 mm maximal in leads V1-V4. Appearance of tall R-waves in V1-V2 may be delayed.[8] Elevations of 0.5 mm or more in V8 and/or V9 add specificity, but may not be sensitive. (see Figure XXX a) standard leads and B) with posterior leads – note how the ST depression is profound, but the ST elevation on posterior leads is minimal. When the standard 12-lead is diagnostic, posterior leads may just confuse the issue by appearing to be negative (falsely)

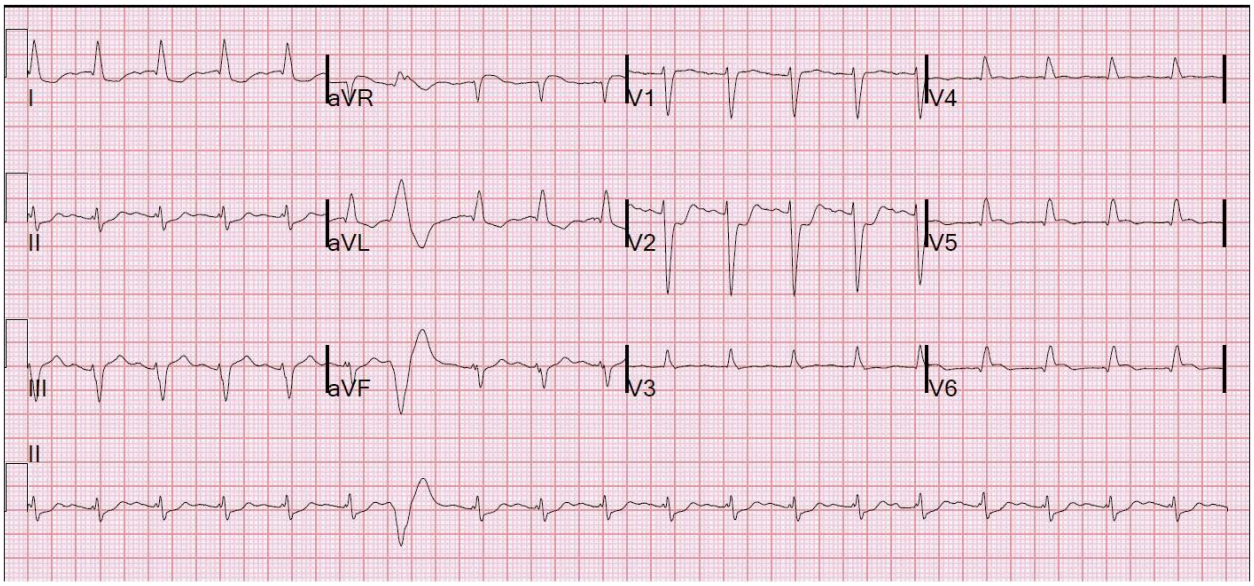
Here there is ST depression maximal in V2-V4, not V5, V6. This is posterior MI until proven otherwise.



In this case, Posterior leads do not quite confirm posterior ST elevation:

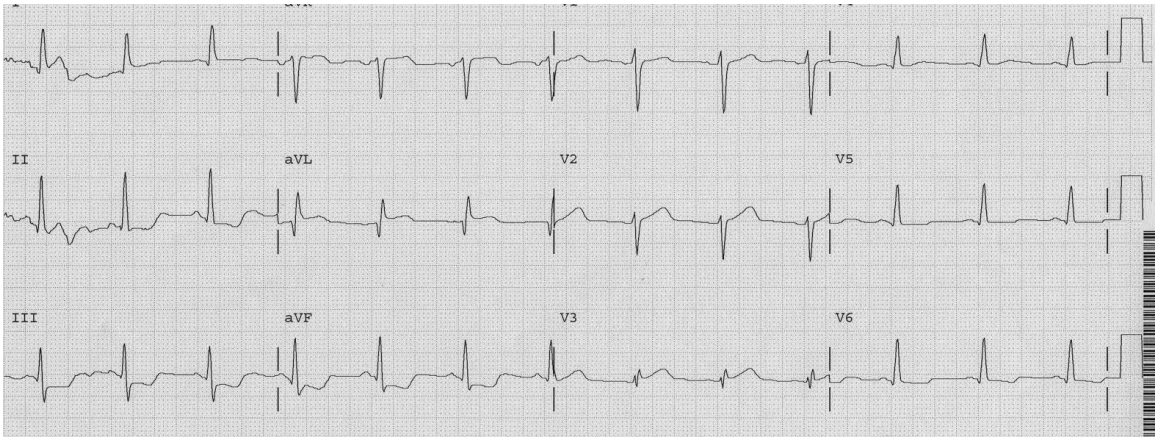


In the case below, posterior MI is confirmed with > 0.5 mm ST elevation in V7-V9, but just barely [posterior ECG only is shown (V4-V6 are really V7-V9), but you can see the right precordial ST depression in V2, which really is V2]



VII. High Lateral Wall MI

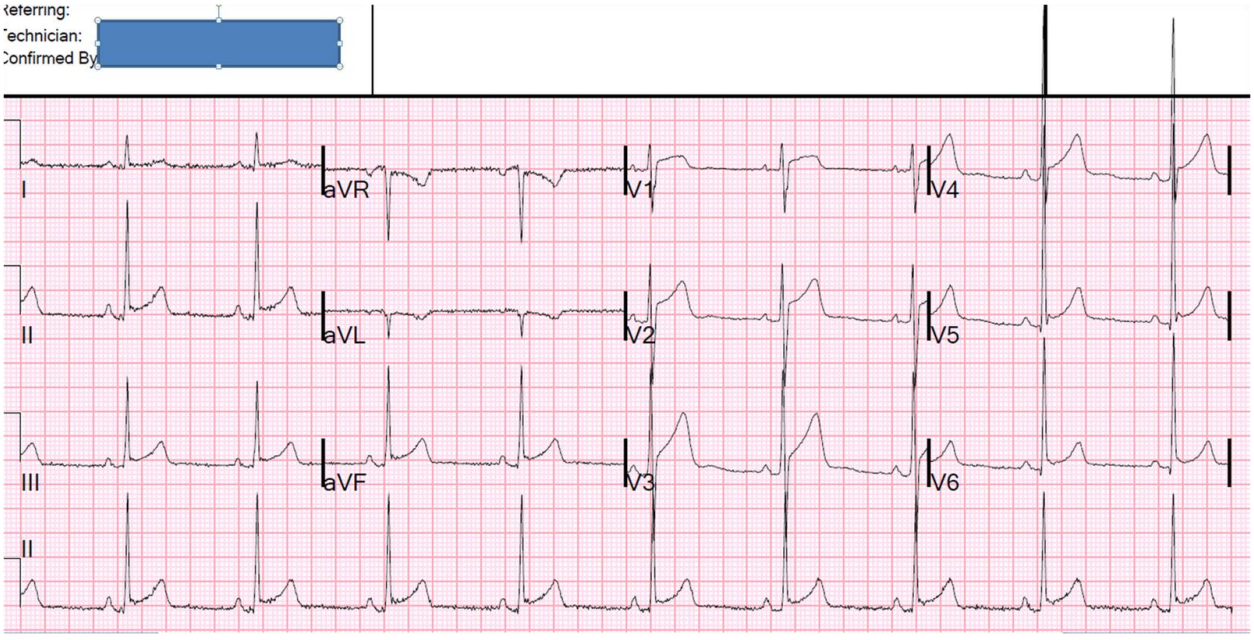
- Any degree of ST elevation in aVL with ST depressions in lead III (with or without II and aVF
- ST depression in II, III, aVF does NOT signify "inferior ischemia." It is usually a reciprocal manifestation of ST elevation in aVL, which may be very subtle. High lateral MI is often best seen by the reciprocal ST depression in leads ii, III, aVF.
- Here is a first Diagonal Occlusion:



VIII. Left Ventricular Hypertrophy

- When there is very high voltage in V1-V4, Anterior STEMI is very unusual, but not impossible
- Beware any concordant ST elevation. With LVH, the ST elevation is discordant to the deep S-wave in V1-V3 and the ST depression is discordant to a high voltage R-wave in V5, V6. V4 is variable in positivity.
- Look for changes from previous ECG.
- In those leads with problematic ST Elevation, if there is high voltage, then assess the ratio. I think 25% (Armstrong paper) is far too much, too insensitive especially for LAD occlusion. These problematic cases usually have at least one S-wave with a 30 mm amplitude. One rarely sees the ST elevation (at the J-point, relative to the PQ jct.) greater than 4 mm, for a ratio of 14%. If Steve must choose a ratio for these leads, he would choose 17%, or 1:6 ratio. 25% (Armstrong et al.) would require 8 mm of STE in such a case!

Here is an Example of false positive ST elevation:



Here is the explanation: http://hqmeded-ecg.blogspot.com/2012/09/male-in-his-40s-with-chest-pressure-what.html

Here are some morphologies of false positive STE in LVH:

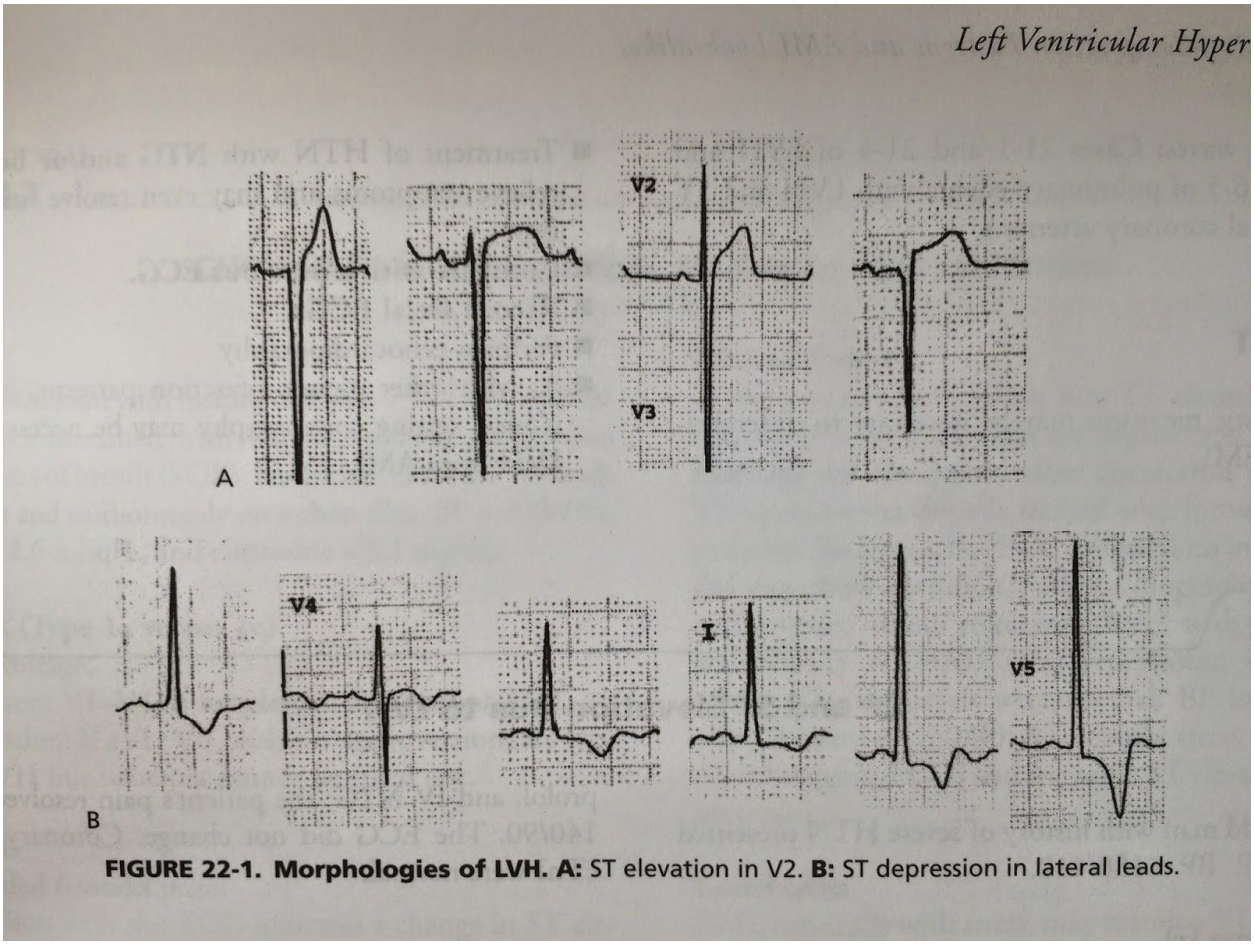
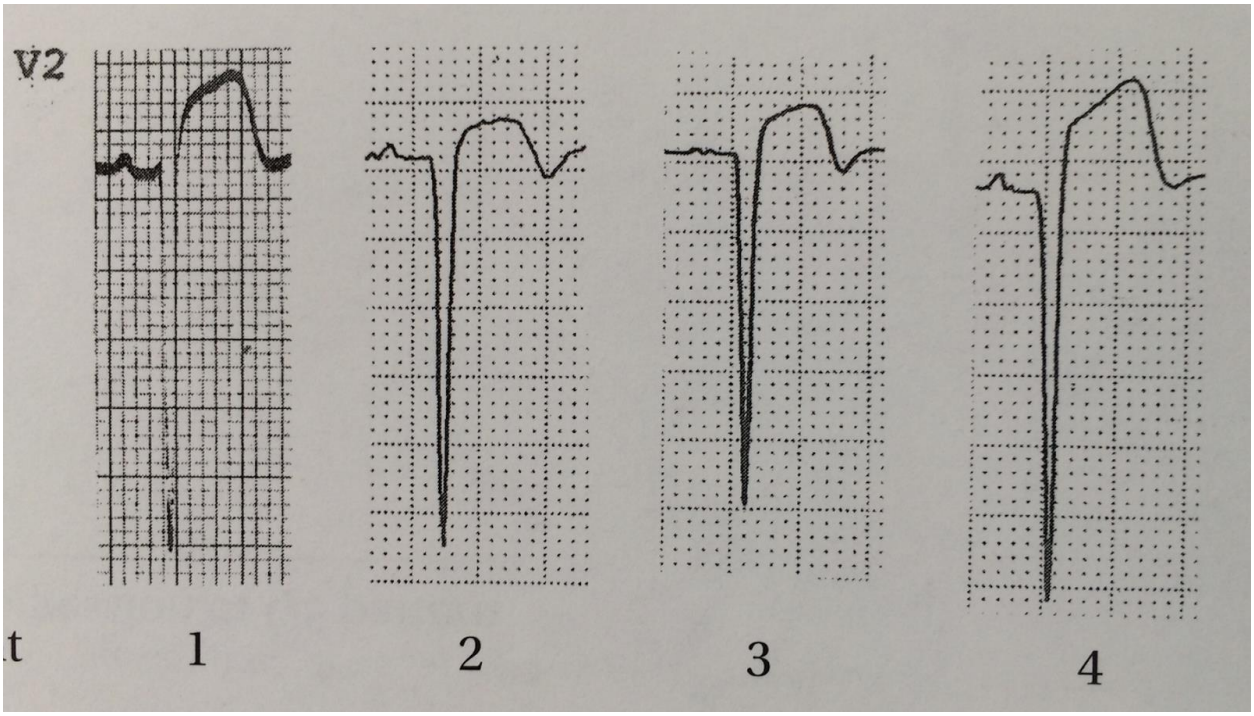


FIGURE 22-1. Morphologies of LVH. A: ST elevation in V2. **B:** ST depression in lateral leads.

Here are some Morphologies that DR. Wang says are STEMI in LVH:



I am sure of the 1st and 4th, and pretty sure of the 2nd and 3rd.

IX. LAD Occlusion vs. Anterior Early Repolarization (Normal Variant ST Elevation in Precordial leads)

It is critical to use it only when the differential diagnosis is subtle LAD occlusion vs. anterior early repol. Thus, there must be ST Elevation of at least 1 mm. If there is LVH, it may not apply. If there are features that make LAD occlusion obvious (inferior or anterior ST depression, convexity, terminal QRS distortion, Q-waves), then the equation MAY NOT apply. These kinds of cases were excluded from the study as obvious anterior STEMI. [9]

- (1.196 x STE at 60 ms after the J-point in V3 in mm) + (0.059 x computerized QTc) - (0.326 x R-wave Amplitude in V4 in mm). QTc is the computer measurement.
- RAV4 = R-wave amplitude, in mm, in lead V4.
- ST elevation (STE) is measured at 60 milliseconds after the J-point, relative to the PR segment, in millimeters.

A value greater than 23.4 is quite sensitive and specific for LAD occlusion (sensitivity 86%, specificity 91%. At a cutoff of 22.0, the sensitivity is 96% with a specificity of 81%

Use the calculator at [hqmeded-ecg.blogspot.com]. See applet down right side of blog, or free iPHONE app "subtleSTEMI".

There are many examples of its use on the blog (http://hqmeded-ecg.blogspot.com/search?q=early+repol+formula).

Here is one good example: http://hqmeded-ecg.blogspot.com/2013/01/male-in-his-40s-with-chest-pain.html

X. de Winter ST/T-wave complex (a form of hyperacute T-wave with depressed ST takeoff)

- ST depression >1 mm up-sloping at the J-point in leads V1-V6[8]
- Tall T waves and up-sloping ST depression do not necessarily evolve into ST elevation. Precordial T waves are tall, upright, symmetric
- Normal QRS duration
- Associated with extremely tight acute thrombotic LAD stenosis/subtotal occlusion or occlusion with minimal collateral flow.
- Here are many examples:
    - http://hqmeded-ecg.blogspot.com/2014/04/chest-pain.html

XI. Hyper-acute T-waves (HATW) (Large T waves, large in proportion to the QRS!)

- Generally prudent to perform serial ECGs, because true HATW generally morph quickly into a classic STEMI pattern[8] or resolve quickly if they are "on the way down" after spontaneous reperfusion
- Hyperkalemia is another common cause of tall T waves. If in doubt, measure the K!
- Here are some examples:
    - http://hqmeded-ecg.blogspot.com/2009/02/hyperacute-t-waves.html

XII. Elevation in aVR with diffuse ST Depressions

Usually indicates left-main or LAD insufficiency (not necessarily occlusion, as so many of us have thought). aVR elevation with ST depressions in a patient whose pain is controlled by medical management whose ST deviation largely resolves, and is hemodynamically and electrically stable, can be managed medically with extremely vigilant observation, continuous 12-lead ECG monitoring, and next day Cath.

The new 2013 ACC/AHA guidelines give this as an indication for thrombolytic therapy. This is the first time they have recognized that the studies prohibiting thrombolytics for ST depression did not include this high risk group.

XIII. STEMI vs. Left Ventricular Aneurysm (old MI with Persistent ST elevation)

They are differentiated based on the T-wave amplitude (specifically T/QRS ratio), and the principle that an acute MI, in contrast to an old or subacute MI, has large upright T-waves. We have both derived and validated this rule. Suspect it when there are QS-waves in V2 and V3.

If there is one lead of V1-V4 with a ratio of positive T-wave amplitude to total QRS amplitude (T/QRS ratio) that is > 0.36, it is highly likely to be acute MI. If < 0.36, it is highly likely to be LV aneurysm. Subacute MI (chest pain duration > 6 hours) is a possible false negative.

see: http://hqmeded-ecg.blogspot.com/2009/08/persistent-st-elevation-after-previous.html

Inferior aneurysm is much more difficult to diagnose, as acute inferior MI can have Q-waves that mimic aneurysm. Assume it is acute unless historical or echo data prove otherwise.

XIV. What if it looks like a STEMI, but chest pain has been of long duration, or there are Q-waves?

Q-waves may appear within the 1st hour of pain onset. If the patient still has chest pain, they should go to the lab.

See: http://hqmeded-ecg.blogspot.com/2014/08/9-hours-of-chest-pain-and-deep-q-waves.html

XV. Unrelieved Pain with NSTEMI

"The False STEMI/NonSTEMI dichotomy"

ST depressions or positive troponin, or clinical diagnosis of angina with ongoing symptoms not relieved by medical management

STEMI: unequivocal, needs emergent reperfusion

Non-STEMI: Does not need reperfusion until 24-36 hours, right?

Wrong! There are Non-STEMIs that need the cath lab now. The dichotomy is false.

ACC recognizes this in their recommendations, but they are buried in there:

> *Patients with objective evidence of ischemia (high risk like h/o CAD with typical pain, or ischemic ECG, subtle ST elevation, or + trop) and persistent ischemia (usually persistent pain, but also persistent ischemia on the ECG) in spite of maximal medical therapy (antiplatelet, antithrombotic, IV nitro) need to go to the cath lab immediately.*

Evidence? No randomized trials, but there are 5 trials of emergent PCI for Non-STEMI. Few know that all of them either excluded patients with persistent ischemia ***or*** showed benefit of emergent PCI. The largest trial by far, the TIMACS trial (http://www.nejm.org/doi/full/10.1056/nejmoa0807986), by Mehta et al. -- they did not even state in their methods that they excluded patients with refractory ischemia. Steve had to personally contact Dr. Mehta to find that out. He said he "Cannot Imagine that any investigator would have enrolled a patient with refractory ischemia." Early cath was also average of 14 hours. They did find that for those who were "high risk," as defined by GRACE score > 140, this so-called "early" cath did result in better outcomes.

Also, there are 4 studies on the condition of the infarct artery in patient with "NonSTEMI" who get their cath the next day: they all show that about 25% have an occluded artery at the time of cath 24 hours later, that they have higher biomarkers, worse LV function, and higher mortality than the "NonSTEMI" patients with an open artery.

So treatment is simple: Objective evidence of ischemia (including, but not limited to, ST depression and subtle ST elevation), and you can't control it: go to the cath lab. Not every NonSTEMI is alike.

Steve's shop now has an intermediate path other than the typical STEMI or Not a STEMI dichotomy that they call "Pathway B". They page the on-call cardiologist for cases that might need emergent PCI, but are not STEMI, and they emergently (within 5 minutes) help the ED doc assess whether there is need for it, especially with emergent formal contrast ultrasound. They are very responsive, and experience has shown so many unexpectedly occluded arteries that they are now very aggressive.

Get a Cardiology Consult and/or Admit to Cardiology Bed where the patient can be carefully monitored

I. Wellens

This is a high risk LAD lesion that was occluded at the time of the pain but is now open. The lesion may close off at any moment and this may initially be asymptomatic. If emergent cath is not undertaken, maximal antiplatelet and antithrombotic therapy and **continuous 12-lead ST segment monitoring** is essential to prevent re-occlusion and to detect it if it occurs.

See this post:

http://hqmeded-ecg.blogspot.com/2013/11/why-we-need-12-lead-st-segment.html

Pattern A is biphasic T-wave (Up and then Down)

Pattern B is deeply inverted T-waves

Both should only be seen while the patient is chest pain free. Unfortunately, there are many false positives, especially in patients with LVH and hypertension. Wellens' is a syndrome (ECG and high risk clinical presentation, and pain free)

II. Transient STEMI

Ownbey M. et al. **Prevalence and interventional outcomes of patients with resolution of ST-segment elevation between prehospital and in-hospital ECG.** Prehosp Emerg Care 18(2);174-9. Apr-Jun 2014.

They found 293 total cases of prehospital STEMI, but could only find all the relevant records in 83 cases (28%). ST Resolution (STR) by the time of ED arrival occurred in 18 of 83 cases. There were no differences between STR and non-STR cases in prehospital vital signs or treatments. 95% of patients underwent cardiac catheterization with a mean door-to-needle time of 57 minutes (interquartile range 43-71). Comparing STR and non-STR cases, significant lesions (greater than or equal to 50%) were found in 94 and 97% of patients ($p = 0.6$), and subtotal or total lesions (greater than or equal to 95%) were found in 63% and 85% ($p = 0.1$), respectively.

Meisel SR, et al. **Transient ST-elevation myocardial infarction: clinical course with intense medical therapy and early invasive approach, and comparison with persistent ST-elevation myocardial infarction.** Am Heart J 155(5):848.

They studied 1244 consecutive STEMI patients. 63 (5%) had Transient STEMI (TSTEMI): Patients with Transient STEMI were treated with intravenous isosorbide dinitrate, aspirin, and clopidogrel, and/or with glycoprotein IIb/IIIa inhibitors. Coronary angiography performed 1.5 days after admission demonstrated no obstructive lesion or single-vessel obstructive disease in 43 patients (70%). PCI was performed in 48 patients (77%), and 8 patients (13%) were referred to surgery. Left ventricular ejection fraction was within normal limits, and peak creatine kinase was mildly elevated. Transient STEMI was associated with less myocardial damage, less extensive coronary artery disease, higher thrombolysis in myocardial infarction flow grade in culprit artery, and better cardiac function. These data suggest that immediate intense medical therapy with an early invasive approach is an appropriate therapy in patients with Transient STEMI.

III. Any of the above that you or consults feel do not require emergent PCI

Additional Key Points

Subtle ST Elevation is STILL BAD! (PMID 25458652)

References

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7. Ondrus T, Kanovsky J, Novotny T, Andrsova I, Spinar J, Kala P. Right ventricular myocardial infarction: From pathophysiology to prognosis. Experimental and clinical cardiology 2013;18:27-30.
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