Managing STEMI Mimics in the Prehospital Environment

Brandon Oto, NREMT-B

The Problem: Most STE is not MI

… and most providers can't tell the difference

"Unfortunately, STE is a not an uncommon finding on the ECG of the chest pain patient; its cause infrequently involves AMI."

Brady et al., **Electrocardiographic ST-segment elevation: correct identification of acute myocardial infarction (AMI) and non-AMI syndromes by emergency physicians** *(Acad Emerg Med 2001; 8(4): 349-360)*

How common?

*Brady et al**., Cause of ST segment abnormality in ED chest pain patients** (Am J Emerg Med 2001 Jan;19 (1):25-8)*

- Retrospective review of ED charts over 3-month period
- Looked at 902 adults with cc "chest pain"
- Looked for STE in contiguous leads, >1mm limb leads, >2mm precordials
- Compared final diagnoses, MI vs. other

Results

Only 15% of STE patients had MI!

85% had non-MI diagnosis

What were they?

- Left Ventricular Hypertrophy — 25%
- Left Bundle Branch Block — 15%
- **AMI — 15%**
- Benign Early Repolarization — 12%
- Right Bundle Branch Block — 5%
- Nonspecific BBB — 5%
- Ventricular aneurysm — 3%
- Pericarditis — 1%
- Undefined/unknown — 17%

STEMI-mimic was **6 times** as likely as STEMI!
MI was not even **single** most likely cause!

In other words:

Any monkey can recognize ST elevation
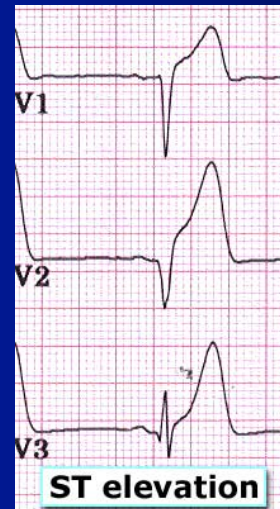
STEMI recognition and diagnosis requires distinguishing MI from non-ischemic causes

How do we do at this?

Bad!

51% false-positive rate

> *Sejersten et al.* ***Comparison of the Ability of Paramedics With That of Cardiologists in Diagnosing ST-Segment Elevation Acute Myocardial Infarction in Patients With Acute Chest Pain*** *(Am J Cardiol 2002 Nov 1;90(9):995-8)*

Half of our diagnoses were wrong

Why does it matter?

Maybe it doesn't!

Overtriage is better than undertriage...

Maybe it's better to play it safe...

But there *are* downsides

Downsides of false positive STEMI diagnoses

- **Compromises systems**. Cath labs that can't rely on you eventually won't activate for you. Increased D2B times.

- **Weakens true positives**. If you lack confidence in recognizing mimics, you'll lack confidence in making the call for true STEMI.

- **Degrades professional respect**. Field providers that do their job right encourage further development of progressive EMS-hospital interfaces.

More downsides of false positive STEMI diagnoses

- **Missed alternate diagnoses**. Some non-MI diagnoses are also critical – think aortic dissection, hyperkalemia, etc. "Call it STEMI" is not always "playing it safe."

- **Wrong treatment**. Nitro? Aspirin? Fibrinolytics? Getting it right affects field *and* hospital treatment.

- **Wrong transport destination.** Unnecessarily bypassing non-PCI hospitals damages continuity of care, burdens families, antagonizes facilities.

More downsides of false positive STEMI diagnoses

- **Stretches limited resources and causes iatrogenic harm**.

  - o Inappropriate activation from the field will *not* always be reversed by ED/cardiology upon arrival due to liability

  - o Unnecessary activations tax staff and facilities, divert resources from other patients

  - o Unnecessary angiography (or worse, thrombolysis) can cause renal/vascular damage

  - o Wrongly rushing the patient to the cath lab means they aren't getting treated for their *real* problems

More downsides of false positive STEMI diagnoses

"... the issue of false-positive catheterization laboratory activation remains a significant concern because unnecessary emergency coronary angiography is not without risk to the patient and may impose a burden on limited human and physical catheterization laboratory resources."

Larson et al, *"False-Positive" Cardiac Catheterization Laboratory Activation Among Patients With Suspected ST-Segment Elevation Myocardial Infarction.* JAMA. 2007;298(23):2754-2760.

What are our tools for addressing this?

- **Clinical correlation**. Any suspicious ECG findings should be matched against patient presentation and physical exam.

- **History and risk factors**. Does hx supports MI – smoker, diabetic, hypertensive, aspirin use, etc?

- **Old ECGs**. Extremely valuable tool when available for establishing baseline.

- **Serial ECGs**. Repeat 12-leads may reveal dynamic changes with time/treatment.

More tools for addressing this

- **Supportive ECG findings**. Subtler red flags (pink flags?) that further support or undermine diagnosis of STEMI.

- **Expert consultation**. Upload or interface with medical control, where available.

- **Computer interpretation**. Automatic algorithms provide a "virtual consult," an always-available second opinion.
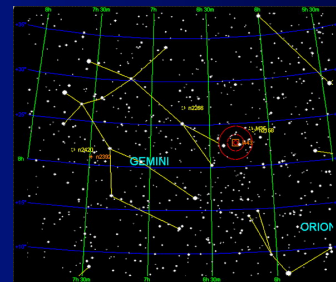
More tools for addressing this

But wait!

- **No** clinical sign/symptoms are completely reliable
- **No** ECG findings are completely reliable
- Hx regularly fools us

The answer?

You must look at the whole picture
Diagnosis is based on a
constellation of datapoints –
not any one finding!

Computer interpretations

Useless? Infallible?

Neither! Just another tool.

Understanding its strengths and weaknesses is essential to effective use

GE Marquette 12SL

Developed in 1980, continuously updated since

Used in most cardiac monitors including Zoll, Lifepack, most in-hospital monitors – but *not* the Philips MRx

*** ACUTE MI SUSPECTED ***

~100% specific!
~61% sensitive

Massel D et al., **Strict reliance on a computer algorithm or measurable ST segment criteria may lead to errors in thrombolytic therapy eligibility.** *Am Heart J 2000 Aug; 140(2) 2216*

GE Marquette 12SL

Bottom line:

*** ACUTE MI SUSPECTED ***

Not very <u>sensitive</u>
But very <u>specific</u>
Exactly what we need!

Great tool for screening out false positives

GE Marquette 12SL

Caveats:

- Very dependent on data quality!
  - Stable baseline
  - No artifact
  - Proper electrode placement
  - Zoll *may* warn you: "Poor data quality, interpretation may be adversely affected"

- Fooled by SVT – distrust interpretations with HR > 100

- Can be fooled by PR depression

GE Marquette 12SL

More on data quality:

- Proper precordial placement
- Limb electrodes go **on the limbs**
- Manage shivering
- Park the truck if necessary
- Prevent patient movement
- May need to prepare skin – shave, dry, tincture?
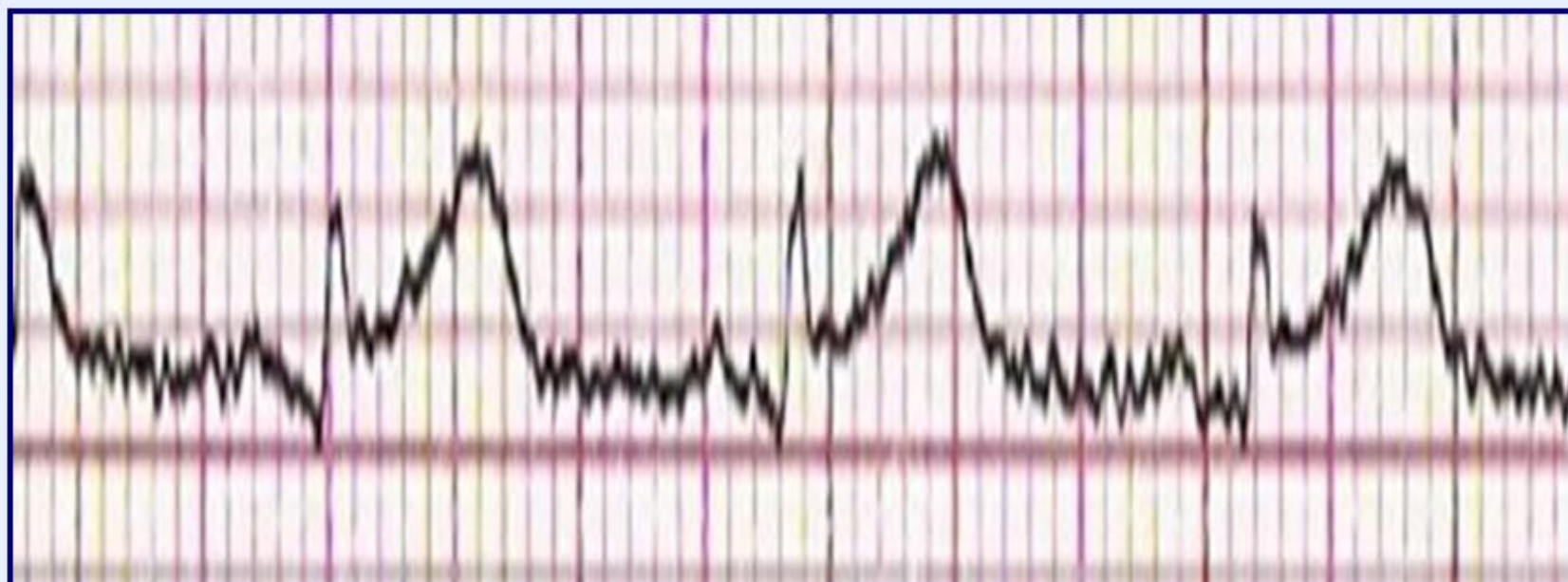- Undress fully from waist up when appropriate
- Stay organized

Signs that point to MI

Your basic model for STEMI should be:

Significant ST elevation in contiguous leads, with reciprocal changes, in the setting of clinical correlation

---

- <u>Significant</u> means significant relative to the QRS amplitude; microvoltages = small STE!

- <u>Contiguous</u> means: know your coronary arteries to understand which occlusions make anatomical sense ("go together")

Signs that point to MI

Your basic model for STEMI should be:

Significant ST elevation in contiguous leads, with reciprocal changes, in the setting of clinical correlation

---

- Reciprocal changes are the **most** valuable addition to your basic criteria for increased specificity (90+%). Look closely; changes may be subtle.

*Akhras, et al. **Reciprocal change in ST segment in acute myocardial infarction: correlation with findings on exercise electrocardiography and coronary angiography.** Br Med J (Clin Res Ed). 1985 June 29; 290(6486): 1931–1934.*

*Otto, et al. **Evaluation of ST segment elevation criteria for the prehospital electrocardiographic diagnosis for acute myocardial infarction.** Ann Emerg Med. 1994 Jan;23(1):17-24.*

Signs that point to MI

Your basic model for STEMI should be:

Significant ST elevation in contiguous leads, with reciprocal changes, in the setting of clinical correlation

---

- <u>Reciprocal changes</u> may also be your only indication of posterior-wall MI. Have a high index of suspicion and consider V7–V9.

A riddle: why does every patient seem to breathe 16 times a minute?

Signs that point to MI

Your basic model for STEMI should be:

Significant ST elevation in contiguous leads, with reciprocal changes, in the setting of clinical correlation

---

- Clinical correlation means the classics (CP, dyspnea, N/V, etc.) but also everything from dandruff to microdeckia. Be open!
- 1/4 – 1/3 may present with unusual or no complaints. Elderly, diabetics, females, cardiac hx more likely to present atypically, and have **worse** prognosis.

Pope et al. **Missed Diagnoses of Acute Cardiac Ischemia in the Emergency Department**. *N Engl J Med 2000; 342:1163-1170*
Kannel. **Silent myocardial ischemia and infarction: insights from the Framingham Study**. *Cardiol Clin. 1986 Nov;4(4):583-91.*

More signs that point to MI

- **ST morphology**. Most benign elevation presents with concave (scooped) ST segments; convex (rounded) elevation is a fairly specific indicator of MI.
- For ST depression, flip it over, same thing
- 97% specific; 77% sensitive
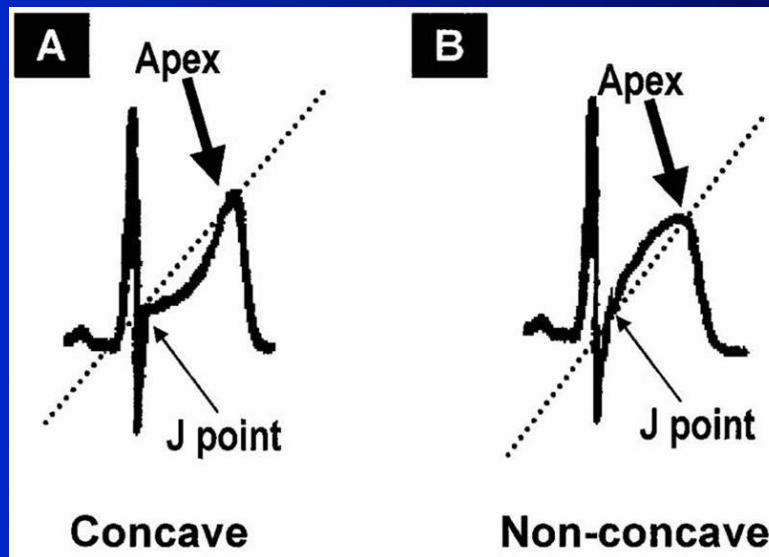
*Brady et al. **Electrocardiographic ST-segment elevation: the diagnosis of acute myocardial infarction by morphologic analysis of the ST segment.** Acad Emerg Med. 2001 Oct;8(10):961-7.*

More signs that point to MI

- **Changes on serial ECGs**. ACS is a dynamic process of supply/demand imbalance; consecutive 12-leads should reveal ongoing changes. Mimics are typically electrically stable.
  - When possible, obtain an initial ECG prior to treatment; oxygen/nitro may erase ischemic changes.
  - Perform serial recordings and watch for evolution over time; subtle becomes obvious, NSTEMI becomes STEMI, etc. *Any* changes are suspicious.
  - One of the **best** tools for distinguishing STEMI vs. mimics!

Early and continuous prehospital ECGs can play a **crucial** role in eventual care!

More signs that point to MI

- **Changes from old ECGs**. When available (from facility or patient), previous 12-leads can establish a baseline -- but this only proves changes **since the time of that tracing**.

Sgarbossa's Criteria

Aka

The rule of appropriate discordance

Aka

The gift that keeps on giving

Originally developed in 1996 as system for ruling in MI in setting of LBBB

Sgarbossa et al. **Electrocardiographic Diagnosis of Evolving Acute Myocardial Infarction in the Presence of Left Bundle-Branch Block**. *N Engl J Med 1996; 334:481-487February 22, 1996*
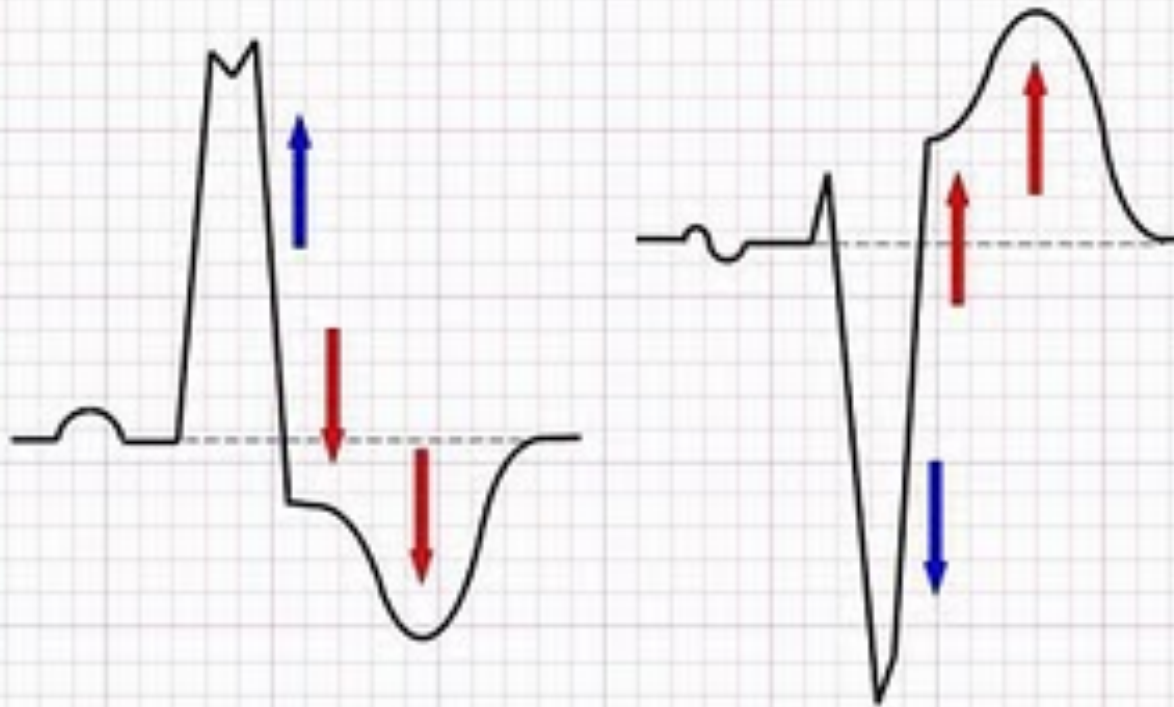
Sgarbossa's Criteria

The rule of appropriate discordance:

- **A QRS with a positive terminal deflection should be followed by a negatively shifted ST/T (ST depression, T wave inversion)**

- **A QRS with a negative terminal deflection should be followed by a positively deflected ST/T (ST elevation, upright T wave)**

- **ST elevation/depression should be proportional to the size of the QRS**
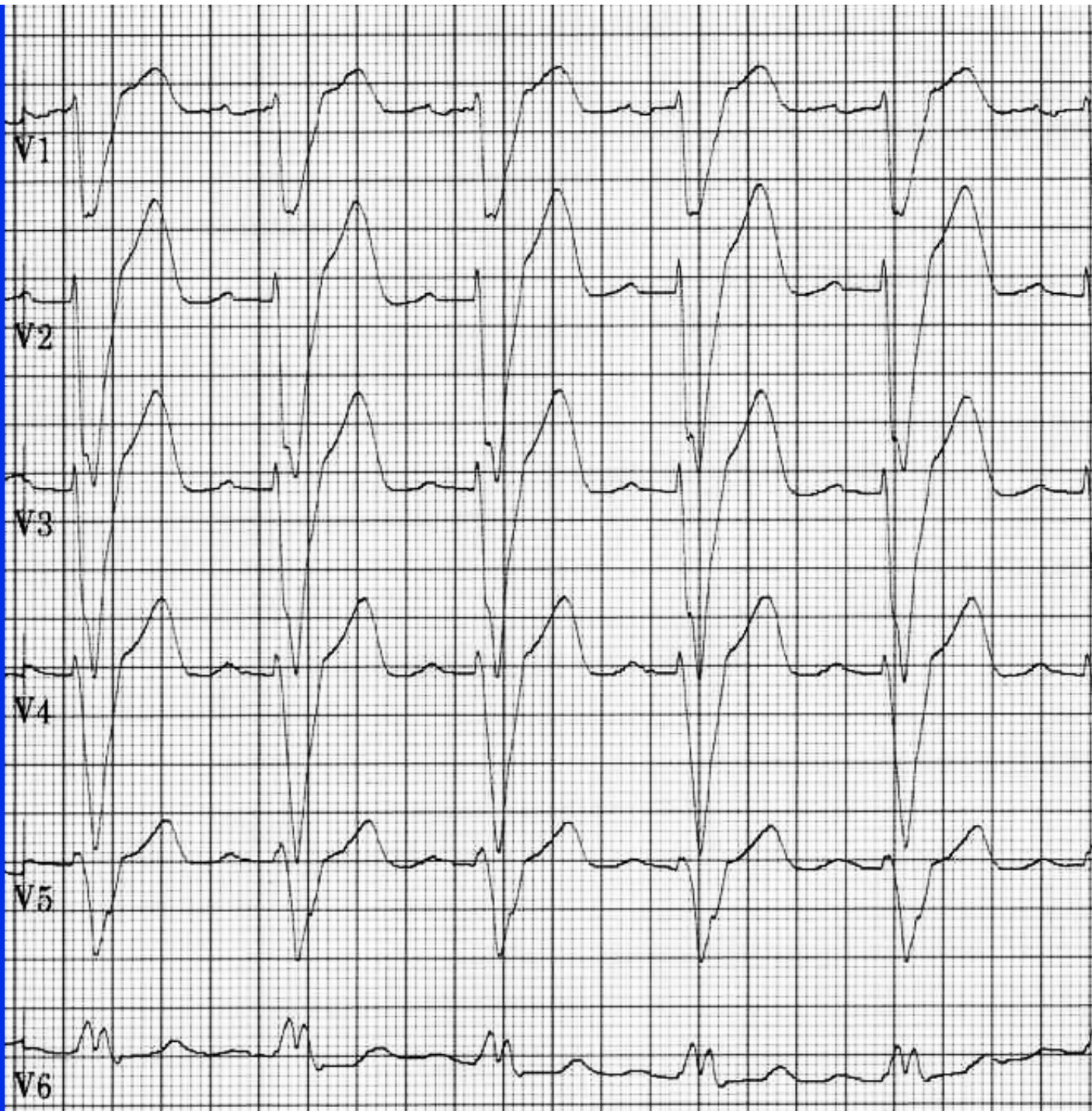
ST Segment and T Wave Discordance
Normal for LBBB / Paced Rhythm

ems12lead.blogspot.com

Sgarbossa's Criteria

Deviations from this suggest MI!

Sgarbossa's Criteria

This principle applies to:

- **LBBB**
- **RBBB**
- **LVH** ("strain pattern")
- **Paced ventricular rhythms**
- **Non-paced ventricular rhythms (including PVCs)**
- **WPW and other preexcitation**

How useful!

Sgarbossa's Criteria

The actual criteria

In the setting of chest pain and LBBB:

- Concordant STE ≥1mm in any lead with a positive QRS (5 points)
- Concordant ST depression ≥1mm in V1, V2, or V3 (3 points)
- Discordant ST elevation ≥5mm in any lead with negative QRS

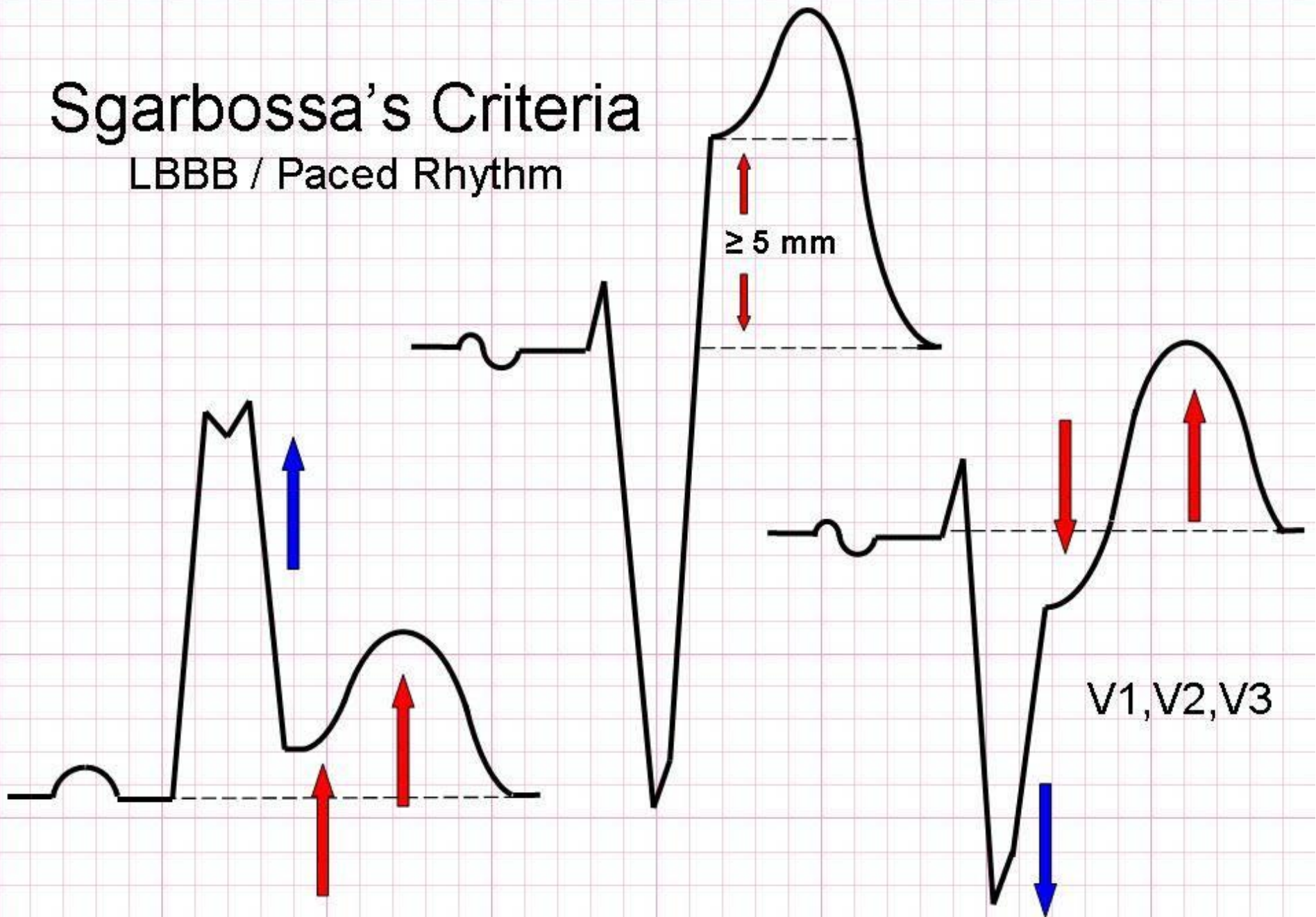## Sum up the points:

≥3 is 98% specific for MI, 20% sensitive
≥2 is 61–100% specific, 20–79% sensitive

Tabas et al. **Electrocardiographic criteria for detecting acute myocardial infarction in patients with left bundle branch block: a meta-analysis**. *Ann Emerg Med. 2008 Oct;52(4):329-336.e1. Epub 2008 Mar 17.*

Sgarbossa's Criteria
LBBB / Paced Rhythm

≥ 5 mm

V1,V2,V3

ems12lead.blogspot.com

Sgarbossa's Criteria

"Discordant ST elevation ≥5mm in any lead with negative QRS"

- The most controversial rule due to the lowest specificity

- The majority of problems arise with large amplitudes (LVH), when 5mm may be normal discordance
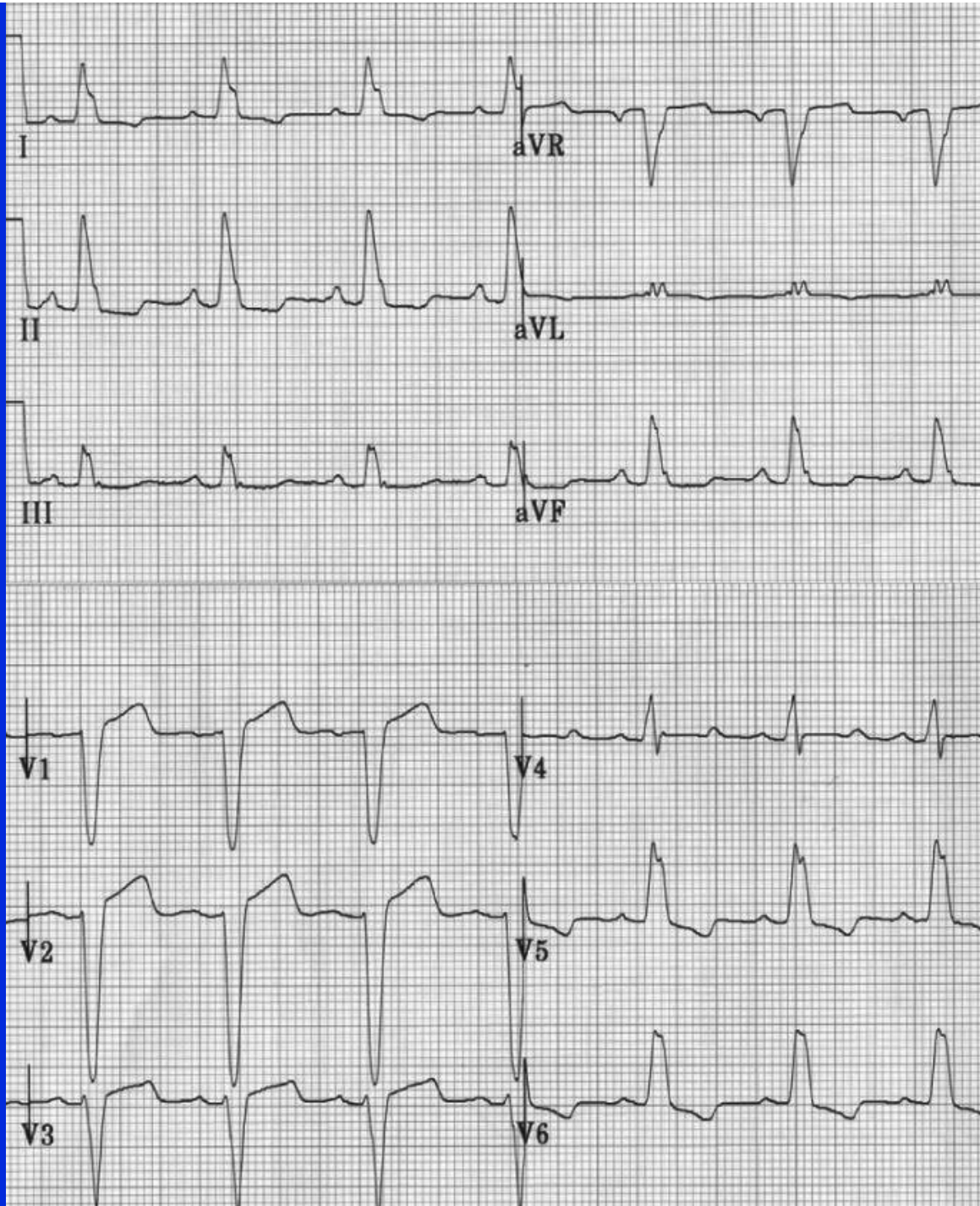
Sgarbossa's Criteria

Smith's Modification

Make it proportional!
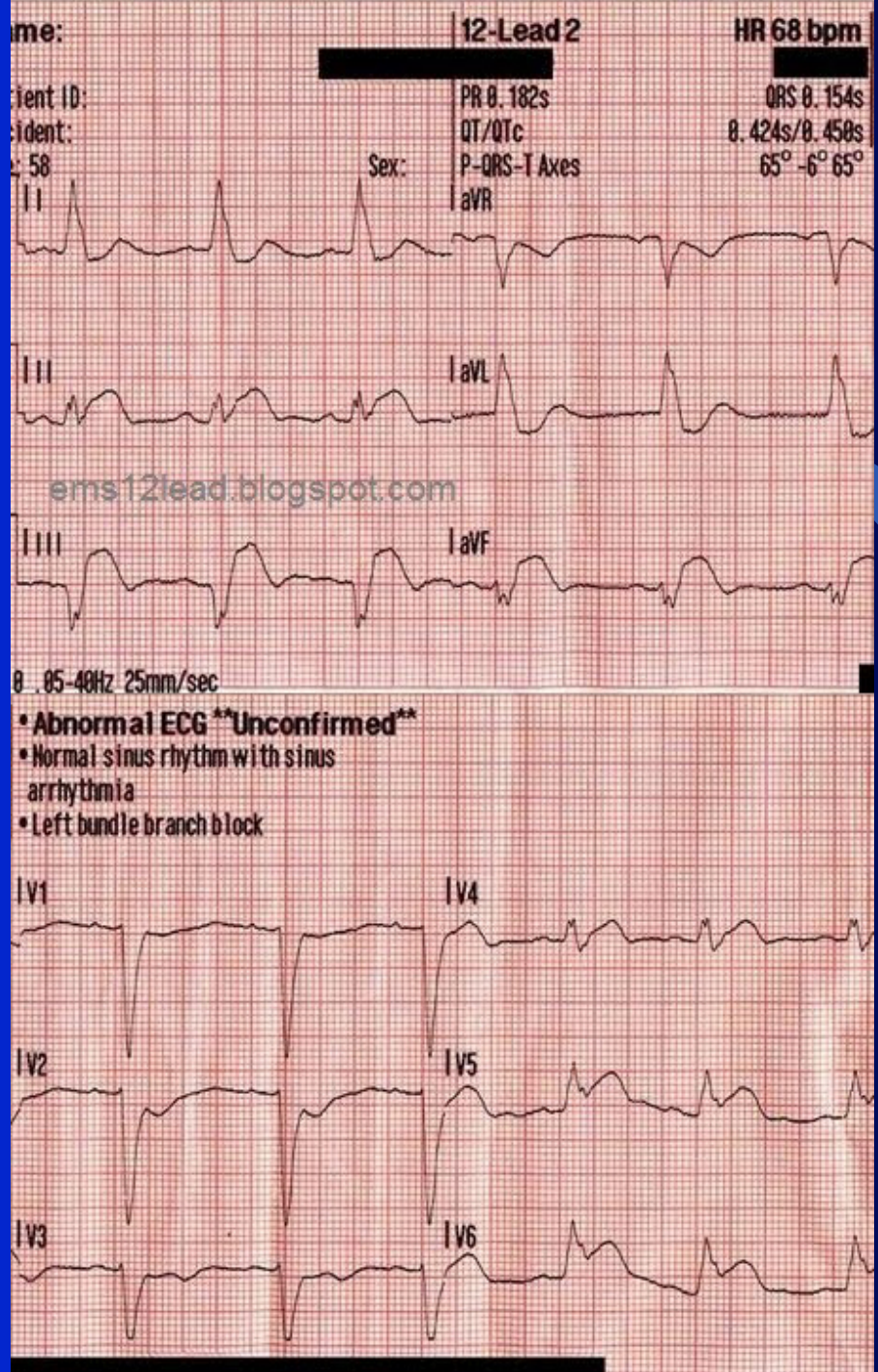
"Discordant ST elevation ≥.2 (1/5) of terminal S wave"

- Reduces false positives from large QRS
- May reduce false negatives from small QRS (microvoltages)

*Smith et al. (2012) **Diagnosis of ST-Elevation Myocardial Infarction in the Presence of Left Bundle Branch Block With the ST-Elevation to S-Wave Ratio in a Modified Sgarbossa Rule**. Ann Emerg Med. 2012 Aug 31 Epub*

ame: 12-Lead 2   HR 68 bpm

tient ID: PR 0.182s   QRS 0.154s
ident: QT/QTc   0.424s/0.450s
: 58   Sex: P-QRS-T Axes   65° -6° 65°
I I   I aVR

I II   I aVL

ems12lead.blogspot.com

I III   I aVF

0 .05-40Hz 25mm/sec

* Abnormal ECG **Unconfirmed**
* Normal sinus rhythm with sinus
  arrhythmia
* Left bundle branch block

I V1   I V4

I V2   I V5

I V3   I V6

Sgarbossa's Criteria

Modified or not, has the sensitivity/specificity of Sgarbossa been studied for any rhythm except LBBB?

Paced ventricular rhythms (similar results)

… and nothing else!

Does it work for LVH, etc?

Yes!

Use the principles – don't sweat the numbers

Now, bring in the mimics!

Left Ventricular Hypertrophy

- Single most common STEMI mimic
- May or may not exhibit ST changes
- Sgarbossa works well when it does
- Can be difficult to determine true size of QRS (overprinting or clipping)
- Tip: True anterior STEMI almost **never** present in setting of profound LVH
- Tip: ST segment **may** be benignly convex
- Voltage criteria generally irrelevant to question of MI

Dr. Smith, unpublished

Name:
ID:
Patient ID:
Incident:
Age: 44

12-Lead 1
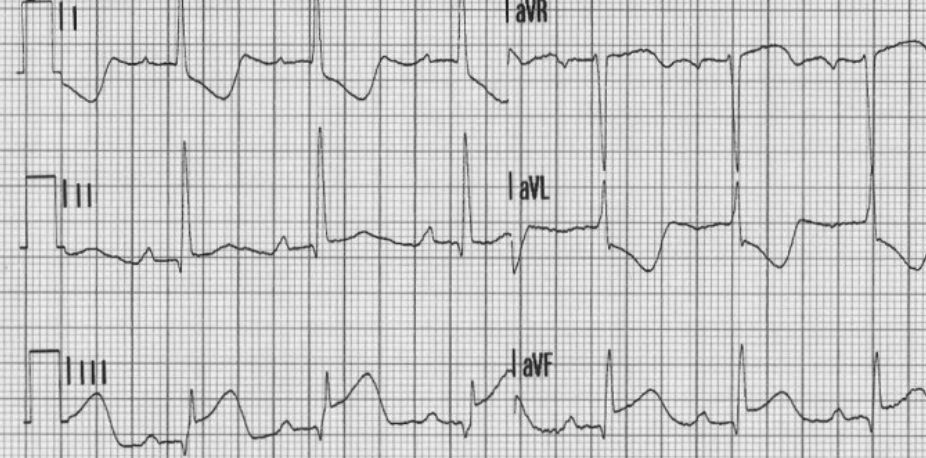
Sex:

PR 0.222s
QT/QTc
P-QRS-T Axes

HR 77 bpm
14:08:23
QRS 0.092s
0.404s/0.457s
72° 42° 138°

I

II
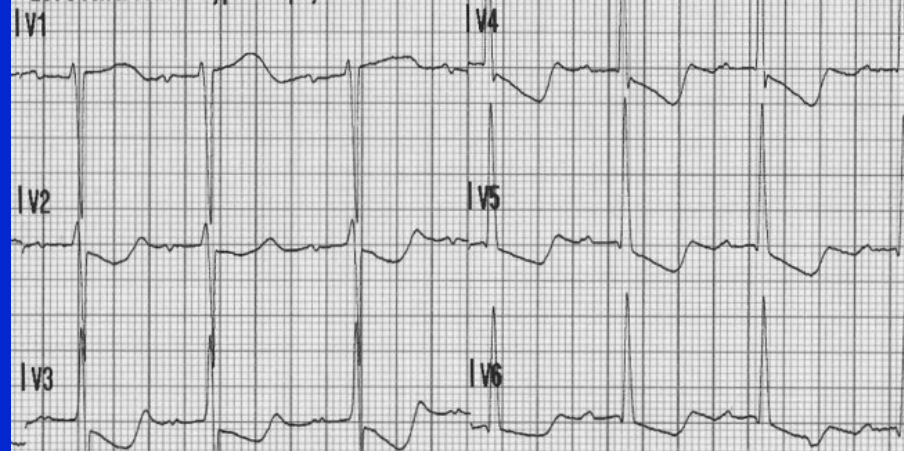
III

aVR

aVL

aVF

ems12lead.com

• *** ACUTE MI SUSPECTED ***
• Abnormal ECG **Unconfirmed**
• Normal sinus rhythm with 1st degree AV block
• Left ventricular hypertrophy with

repolarization abnormality
• ST elevation consider inferior injury or acute infarct

V1

V2

V3

V4

V5

V6

Left Bundle Branch Block

A big can of worms.

- Second most common STEMI mimic
- Traditionally either considered "nondiagnostic," or *de facto* evidence of MI.

AHA still recommends fibrinolysis/TPA:
"… in patients with [signs/symptoms of ACS and] … new or presumably new left bundle branch block …"

Why?
"… because they have the highest mortality rate when LBBB is due to extensive AMI"

2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Part 10: Acute Coronary Syndromes

Left Bundle Branch Block

(The pathophysiology here probably stems from the fact that a LAD occlusion proximal enough to involve the bundle branches is endangering a great deal of myocardium.)

Another, implicit reason is because MI is difficult to definitively diagnose in the setting of LBBB.

Prehospitally, the result is that new LBBB is often assumed to be AMI.

Left Bundle Branch Block

Problem #1: How do you know it's "new"?

Answer: Patient history

Answer: Old ECGs

Answer: You can't

Left Bundle Branch Block

Problem #2: Even "new" LBBB is **not** an indicator of MI!

2008 observational study looked at three groups of ED patients with complaints of ACS:

- Those with new LBBB
- Those with old LBBB
- Those with no LBBB

Result: All groups had **equal** probability of AMI

Chang et al. **Lack of association between left bundle-branch block and acute myocardial infarction in symptomatic ED patients.** *American Journal of Emergency Medicine (2009) 27, 916–921*

Left Bundle Branch Block

In short: new LBBB **in the setting of AMI** may be an indicator of high risk

But it is **not an indicator of AMI!**

Diagnosis must still occur.

Left Bundle Branch Block

How?

You already know it:

- **Sgarbossa!** That's what it's for and we know it works.
- **Serial ECGs**. Uncomplicated LBBB is electrically stable.
- **Old ECGs**. Old LBBB vs. new does not change the probability of MI, but if there is an old LBBB, you can compare old vs. new to find acute changes

Right Bundle Branch Block

- Fifth most common STEMI mimic
- Nevertheless, usually easier to manage, as it often does **not** elevate the ST segment (T wave inversion and ST depression more common)
- Like LBBB, morbidity + mortality may be very high when AMI is associated with RBBB
- Principles are the same! Use your Sgarbossa, etc.; remember to base discordance on *terminal* QRS deflection
- Watch out for posterior reciprocal changes deepening your normal ST depression

ne:

12-Lead 5

HR 79 bpm
08:06:55

ent ID:
dent:
77

PR 0.134s
QT/QTc
Sex: P-QRS-T Axes

QRS 0.152s
0.418s/0.479s
-14° -43° -18°

I
II
III

aVR
aVL
aVF
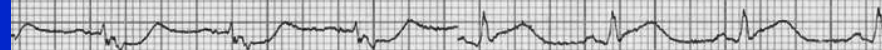
0.05-150Hz 25mm/sec

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• *** ACUTE MI SUSPECTED ***
• Abnormal ECG **Unconfirmed**
• Normal sinus rhythm
• Left axis deviation
• Right bundle branch block

• Anterolateral infarct, possibly acute
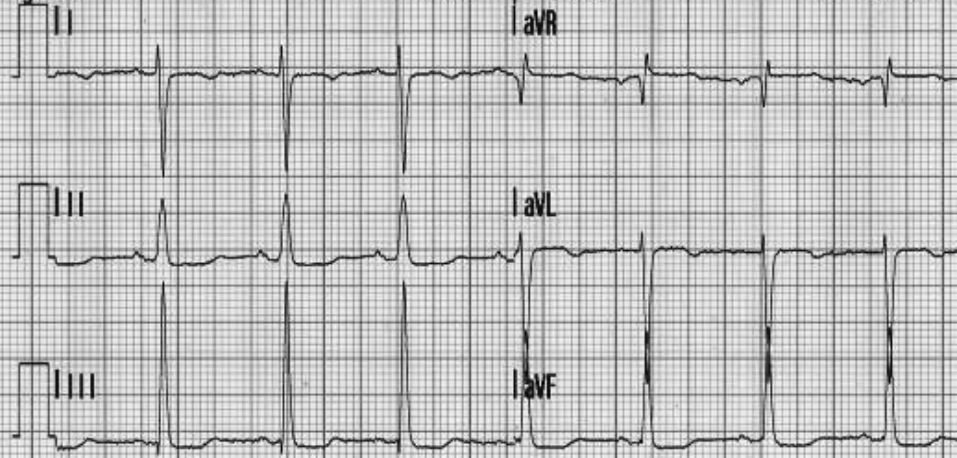
V1
V2
V3

V4
V5
V6
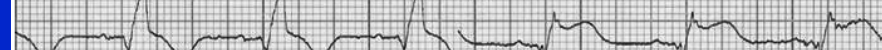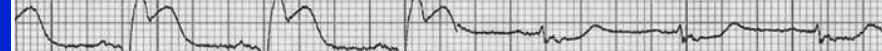
2 LEAD STA 5

Right Ventricular Hypertrophy

- **Second verse, same as the first**. Use your Sgarbossa!
- Only notable differences from LVH:
  - Generally most evident in V1–V3
  - Generally has right-axis deviation
- Presents similarly to posterior-wall MI with reciprocal changes, so keep an eye out

Name:
ID:
Patient ID:
Incident:
Age: 16   Sex:

12-Lead 3   HR 91 bpm
                                 12:55:29
PR 0.130s   QRS 0.088s
QT/QTc   0.338s/0.415s
P-QRS-T Axes   63° 121° 250°

I

aVR

II

aVL

III

aVF

v1 0 05 40Hz 25mm/sec

ems12lead.blogspot.com

- **Abnormal ECG **Unconfirmed****
- Normal sinus rhythm
- Right axis deviation
- Right ventricular hypertrophy with repolarization abnormality

- T wave abnormality, consider inferior ischemia

V1

V4

V2

V5

V3

V6
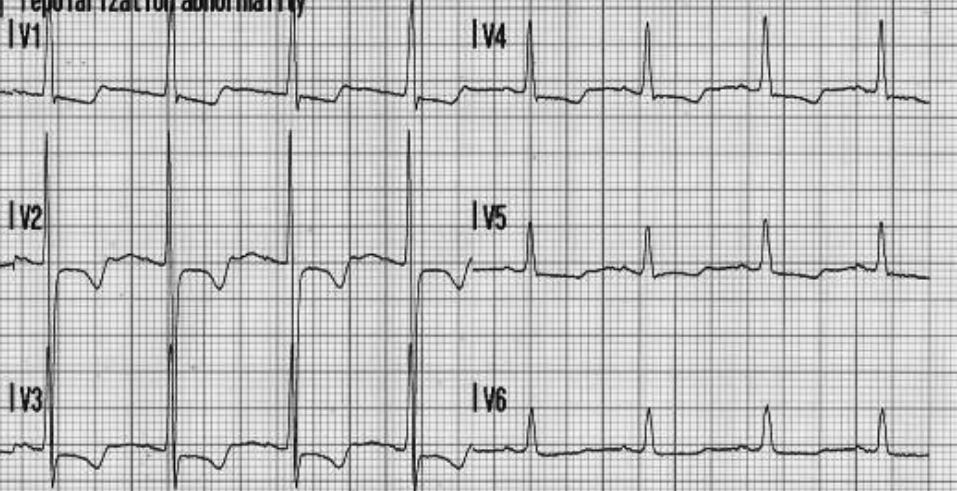
Benign Early Repolarization

Aka **Normal variant** aka **J-point elevation**

- Fourth most common STEMI mimic
- Some degree of benign variance **very** common in healthy population
- Most prevalent in young; decreases in frequency with age
- May be especially noticeable in athletes

The bad news: very common, non-specific ECG characteristics

The good news: changes are generally limited to a few specific features

Benign Early Repolarization

Typical ECG findings:

- ST elevation focused in anterior precordials (V2–V5)
- that is not too profound (<5mm)
- and is concave
- and may have a notched J-point.

Permissible ECG findings:

- Large, *slightly* asymmetrical T waves
- Some movement of ST segment or T wave on serial recordings

Brady, et al. **The Diagnosis: Benign Early Repolarization.** *Emergency Medicine News 23(12):30,36 (2001)*

Benign Early Repolarization

Should *not* be present (suggest MI):
- **Should be no** reciprocal changes!
- **Should be no** convex ST segments!
- **Should be no** pathological Q waves!
- **Should be no** acute evolution!
- **Should be no** clinical signs/symptoms!

In short, except for ST elevation and big T waves, it shouldn't look like MI!
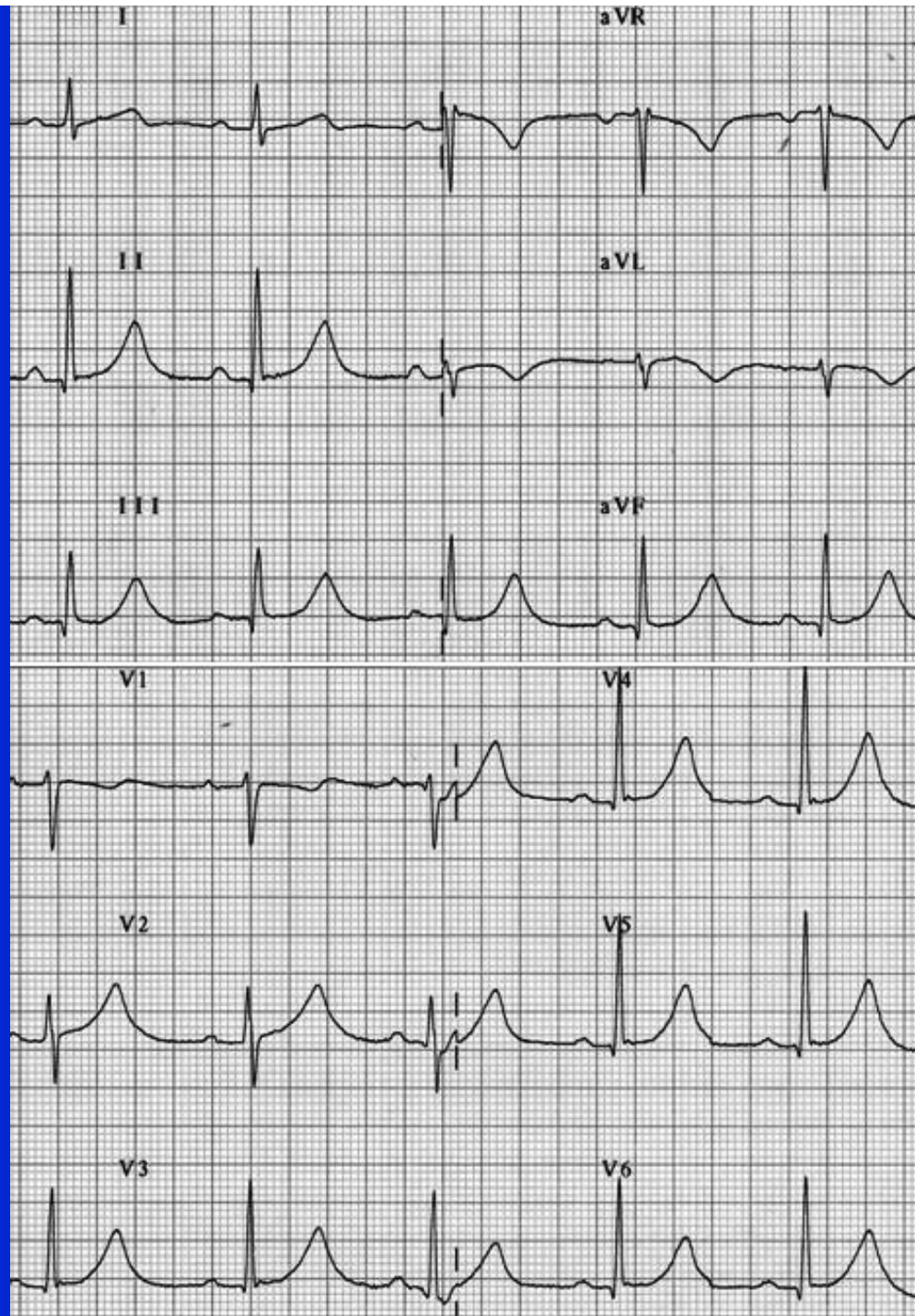
Benign Early Repolarization

Two more pearls to distinguish BER from anterior MI:

- BER is almost always associated with well-developed R waves
- BER is almost always associated with short QTc

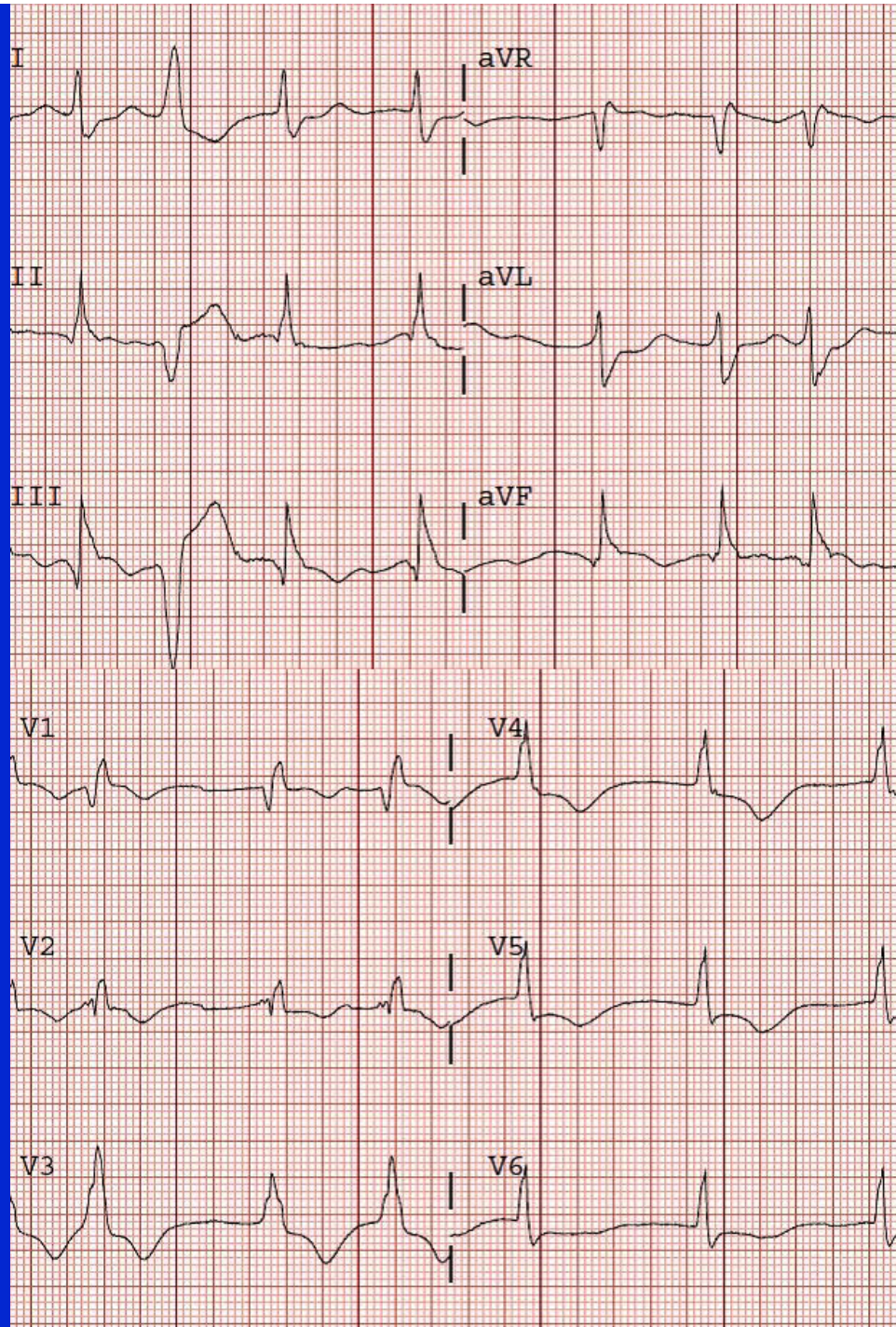**So, if any two of the following are true, it is MI with sensitivity/specificity of 90%:**

- R wave in V4 <13mm
- QTc >392
- ST elevation (measured 1.5 boxes after J point) more than 2mm

Smith et al. *Electrocardiographic Differentiation of Early Repolarization From Subtle Anterior ST-Segment Elevation Myocardial Infarction.* Ann Emerg Med. 2012 Jul;60(1):45-56.e2. Epub 2012 Apr 19
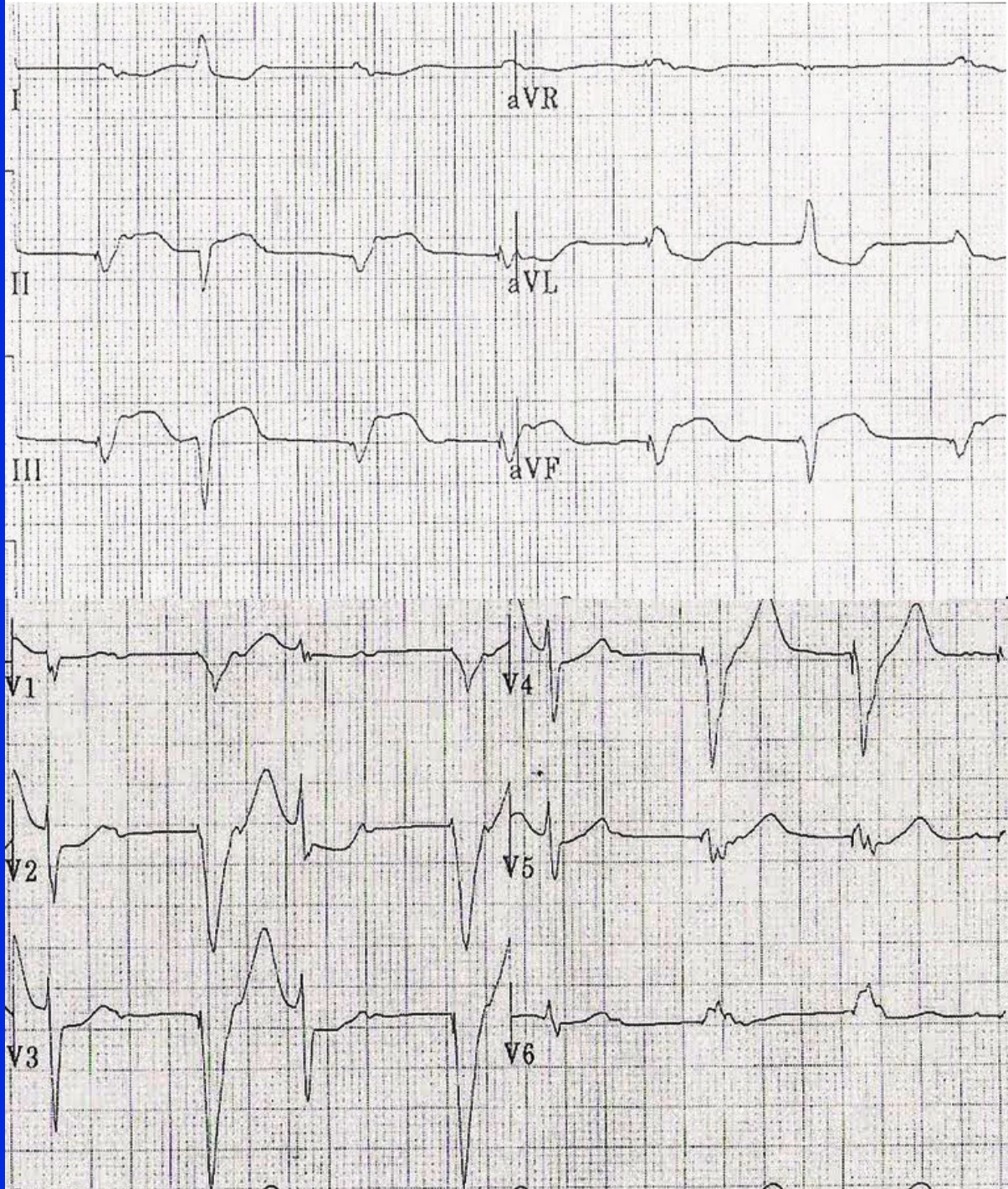
Ventricular Rhythms

- Paced ventricular rhythms the **only** rhythm other than LBBB with empirical support for Sgarbossa's criteria
- Do people with pacemakers have heart attacks? . . .
- **Nothing new here** – use the rules – follow serial ECGs – use your head
- Atypical pacer lead placement (not in apex of right ventricle – or biventricular) may not fit this model

Sgarbossa et al. **Early Electrocardiographic Diagnosis of Acute Myocardial Infarction in the Presence of Ventricular Paced Rhythm**. *Am J Cardiol, 1996; 77: 423–424.*

Left Ventricular Aneurysm

- Refers to persistent aftereffects of a previous MI, which often includes chronically-elevated ST segment
- May or may not actually entail aneurysm (bulging of a weakened section of myocardium)
- Can present essentially **identical** to MI since that's what it was!
- Somewhat more common in anterior leads
- Look into the past: PMH includes prior MI? Old ECG available?

Left Ventricular Aneurysm

Biggest clues:

- **Q waves!** This is an old infarct, and should therefore exhibit pathological Q waves. Look for substantial negative "QS" waves with no notable R wave.

- **No exaggeration of T waves**. As there is no acute ischemia, T waves should not be hyperacute

Left Ventricular Aneurysm

In other words: compare QS depth to T height. The first should be relatively large, the second relatively small. Otherwise suspect AMI.

If you want numbers: amplitude of T wave should be no more than .36 (~1/3) of QS depth in each lead V1–V4. If ratio is greater, suspect AMI.

*Smith SW. **T/QRS ratio best distinguishes ventricular aneurysm from anterior myocardial infarction**. Am J Emerg Med. 2005 May;23(3):279-87.*
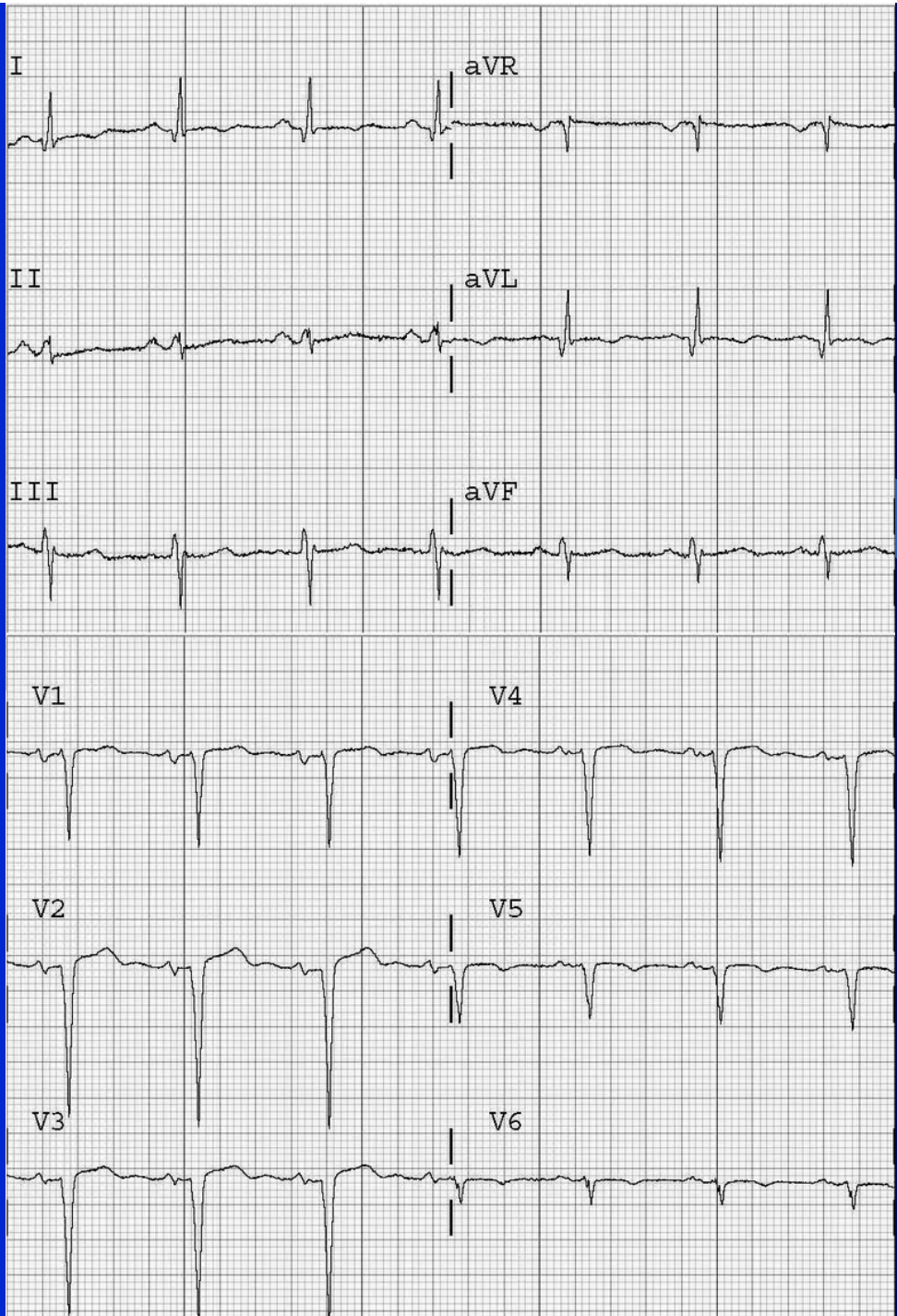
Pericarditis

- Inflammation of the pericardial sac, often infectious
- Clinical signs are suggestive although not specific:
  - Chest pain that may be sharp and pleuritic
  - Pain is alleviated by sitting up or leaning forward
  - May be accompanied signs/symptoms of infection (fever, etc.)
  - Friction rub appreciated on auscultation
  - No relief with nitro

Pericarditis

Initial ECG findings:
- Diffuse, nonlocalized ST elevation, often in all leads except V1 and aVR (which will be depressed instead)
- No reciprocal changes!
- Widespread PR depression
- Morphology resembles BER – generally modest STE that is concave, often notched, with substantial T waves

Generally, approach it similarly to BER! Your main clues are global ST elevation and PR depression, with no supportive signs of ACS like reciprocal changes.

Pericarditis

The disease process produces four distinct stages on the ECG over hours/days:

- Stage 1: ST elevation
- Stage 2: Normalization
- Stage 3: T wave inversion
- Stage 4: Normalization

Depending on when you show up, you may see any of these.
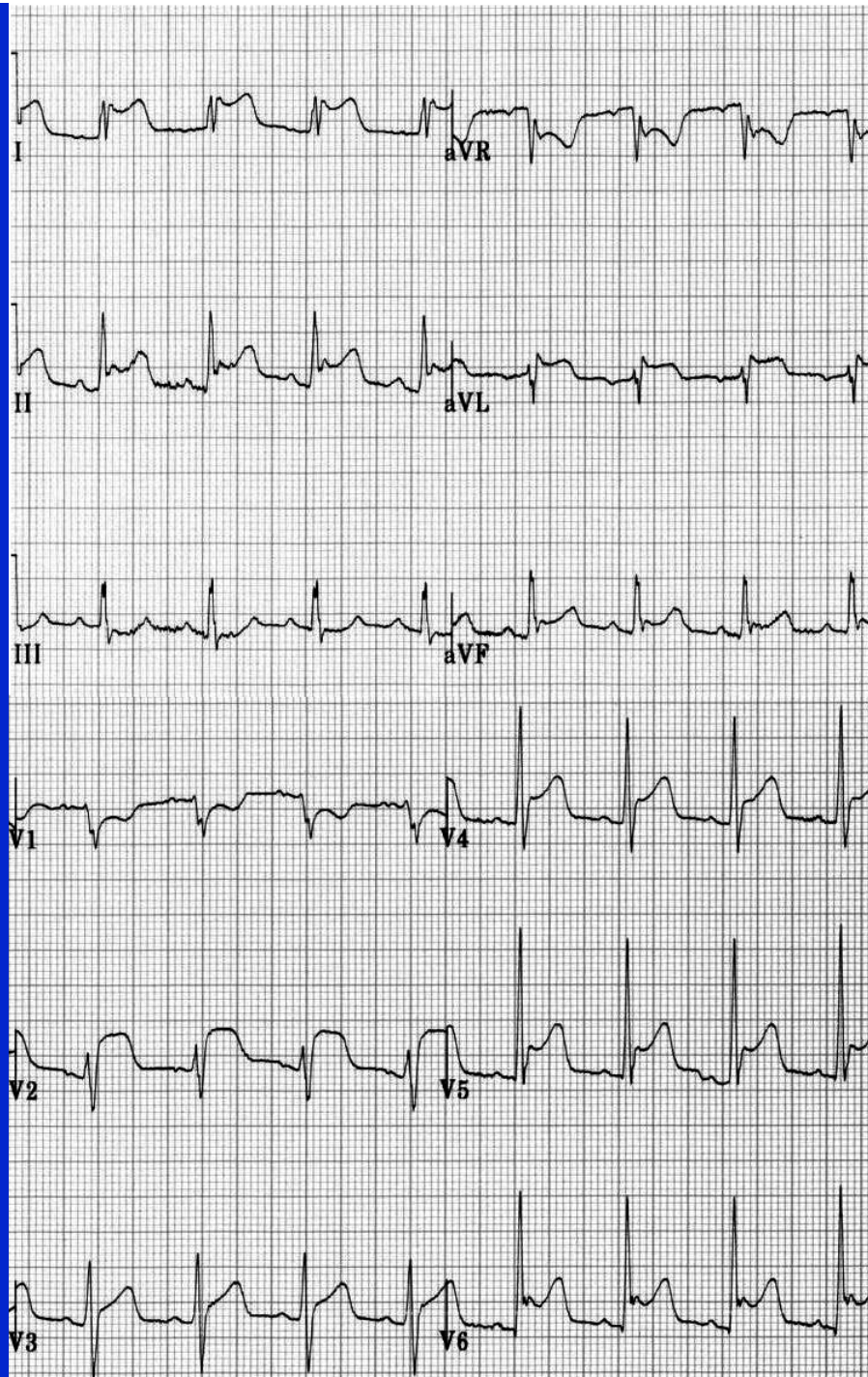
Pericarditis

WARNING! WARNING!

A much more sinister cause can closely resemble pericarditis: occlusion of the LCA (left main) or acute three-vessel disease, causing injury to nearly the entire left ventricle

Pericarditis

If you see widespread ST elevation (*or* depression) and start to think pericarditis, consider two things:

Pericarditis

- **Clinical context.** These patients will usually look terrible and so will their rhythms.

- **aVR.** Pericarditis should show ST depression in aVR; LCA patients typically exhibit ST **elevation** instead.

   Elevation in aVR greater than in V1 may be 80%+ predictive for LCA occlusion

*Yamaji, et al. **Prediction of acute left main coronary artery obstruction by 12-lead electrocardiography**. J Am Coll Cardiol, 2001; 38:1348-1354*

Pericarditis

Despite their acuity, LCA patients need a facility with PCI and CABG – thrombolysis is not beneficial

Mortality can be over 70%

Do your best!

That does it for the common mimics!

How about some of the lesser players?

Brugada Syndrome

- Distinct combination of ECG findings found to be a risk factor for sudden polymorphic VT or VF
- Initially described in 1992, well-studied since and found to have a genetic factor
- Only confirmed cause of Sudden Unexpected Death Syndrome among young males in Southeast Asia
- Risk of sudden arrhythmia and possible death is variable (worse if there is hx of syncope), but may be upward of 8% per year; treatment is EP study + ICD

*Brugada et al. **Long-Term Follow-Up of Individuals With the Electrocardiographic Pattern of Right Bundle-Branch Block and ST-Segment Elevation in Precordial Leads V1 to V3**. Circulation. 2002;105:73.*
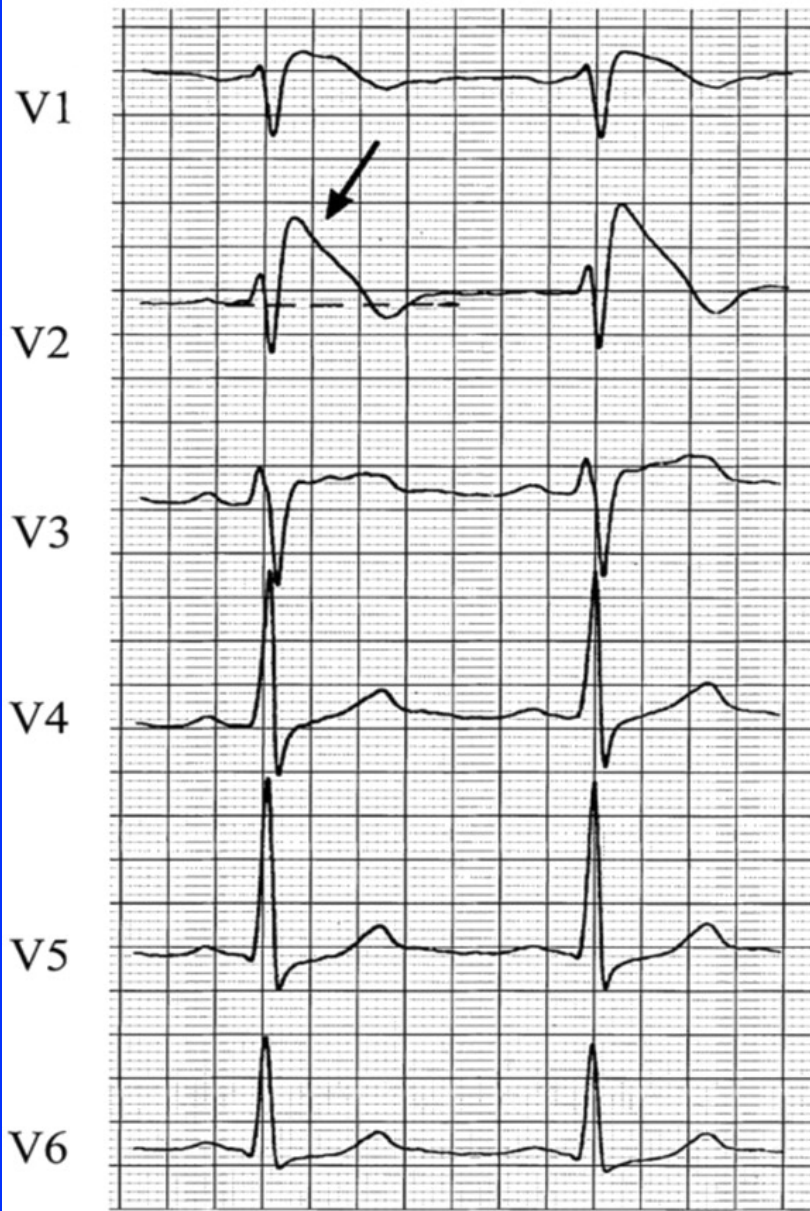
Brugada Syndrome

ECG findings:
- ST elevation in V1–V2, sometimes V3, with particular morphology
- RBBB or similar appearance
- Appearance may fluctuate over time
- ECG changes may be inducible with certain antiarrhythmics

Hallmark morphology is most visible in V1 and V2

	5/2/99	13/2/99	
	type 1	type 2	type 3

V1

V2

V3

V4

V5

V6

1 mV

500ms

Brugada Syndrome

Bottom line:

- Learn the basic morphology
- Screen for it on all ECGs
- Make it part of your differential for syncope in young, healthy patients
- If suspected, monitor continuously and transport to cardiac care

Brugada is the "evil twin" of BER. . . your 12-lead may be the only chance to catch a young, asymptomatic Brugada patient early

Brain injury and intracranial hemorrhage

- Head injury (traumatic and non-traumatic) can induce cardiac dysfunction in 50%–100% of cases
- Some degree of heart failure is the most common result
- Thought to involve myocardial "stunning," possibly resulting from neurogenically-mediated vasospasm
- Effects are typically transient, but not necessarily benign
- ECG signs are variable, can include ST changes and giant inverted "neurogenic" T waves; Osborne wave elevation is possible.

Jain, et al. **Management of Patients with Stunned Myocardium Associated with Subarachnoid Hemorrhage**. *American Journal of Neuroradiology 25:126-129, January 2004*

Brain injury and intracranial hemorrhage

Bottom line:

- Primary point is not to be confused if you see ECG abnormalities when assessing the brain injury patient. Consider the circumstances, but the likelihood of concomitant ACS is very low.

- Monitor for cardiogenic shock; patient may need cardiovascular support in immediate injury period.

Thoracic Aortic Dissection

- Diagnosing TAD is critical due to its high mortality and potential for rapid deterioration
- Complicated by its similarity in presentation to AMI, which can include ECG findings
- Distinguishing the two is *critical* due to radically different treatment paths (different destination facilities, surgery vs. thrombolysis, etc.)
- Mechanism for ECG changes is physical impingement on coronary arteries from retrograde dissection (i.e. it is true ischemia but not ACS)

Thoracic Aortic Dissection

Clinically:

- No signs/symptoms are highly diagnostic for TAD vs. AMI
- The best is patient description of pain: >80% will describe it as abrupt, maximal at onset, and "worst ever."
- 40–60% describe it as sharp or tearing/ ripping
- Vast majority localize the pain to the chest area (anterior or posterior)
- Hypertension is present in >70% of patients
- Pulse or BP differential *not* sensitive or specific

Thoracic Aortic Dissection

ECG findings:

- May present with LVH baseline due to prevalence of background hypertension in TAD patients

- 8% will show ST elevation

- 42% will show some form of ischemic changes, including ST depression or T wave inversion

  - 75% of the time in inferior leads
  - 25% of the time in lateral leads

Mattu, A et al. **Avoiding Common Errors in the Emergency Department**. 2010.
Schubert. **Thoracic aortic dissection: Distinguishing it from acute myocardial infarction**. Canadian Family Physician.
Available from http://www.cfpc.ca/cfp/2003/May/vol49-may-clinical-3.asp

Thoracic Aortic Dissection

Bottom line:
- A very difficult and high-stakes diagnosis that still has no good solutions
- Have a high index of suspicion; TAD should be on your differential for all chest pain patients
- If history and clinical picture leans toward TAD over MI, weigh the risks/benefits and get to a hospital fast

Prinzmetal (Vasospastic) Angina

- A still-largely-idiopathic disorder involving spontaneous vasoconstriction of the coronary vessels
- Distinguished from typical angina by its arbitrary onset, unrelated to exercise
- Often occurs in setting of CAD, but produces symptoms disproportionate to degree of stenosis
- Typically benign unless very severe, but in combination with CAD can contribute to poor outcomes

Prinzmetal (Vasospastic) Angina

ECG findings:

- ST elevation, typically slight (1mm or less) but occasionally severe; sometimes with T wave inversion

- Inverted U waves may be present

- Transient duration, generally relenting within several minutes

- ECG changes normalize with termination of episode, although some T wave inversion may persist

*Miwa et al. **Two electrocardiographic patterns with or without transient T-wave inversion during recovery periods of variant anginal attacks.** Jpn Circ J. 1983 Dec;47(12):1415-22.*

Prinzmetal (Vasospastic) Angina

Bottom line:

- Atypical or not, it's angina! It will look and smell like angina!

- Nitro will likely be very effective

- It'll pass – one more reason for serial ECGs. Signs/symptoms 10+ minutes start to point to AMI.

- May not be distinguishable from aborted or "stuttering" MI ("winking and blinking"), and those patients *do* need cardiac care, so play it safe

Wolf-Parkinson-White Syndrome

- Accessory pathway allows preexcitation of ventricles outside of standard conduction pathways
- Due to transverse conduction, morphology resembles BBB, and produces STE for same reason
- Risk factor for sudden death in the setting of arrhythmia (reentry or conduction of A-fib)
- Diagnosis is important for two reasons:
  - Distinguishing it from STEMI
 - Providing appropriate anti-arrhythmic treatment

Wolf-Parkinson-White Syndrome

ECG findings:
- Slurred initial entrance to QRS ("delta wave")
- Short PR (<120ms)
- Wide or borderline wide QRS
- Often resembles LVH and is confused with it
- ST elevation generally discordant with QRS

Wolf-Parkinson-White Syndrome

Bottom line:

- Start by recognizing the preexcitation
- Sgarbossa actually works in most cases, but not reliably – each accessory pathway is different and conduction is unique. Approach it like LVH but keep an open mind.
- If diagnosis of WPW is clear, be skeptical about STEMI; symptoms are much more likely related to arrhythmia than to MI.
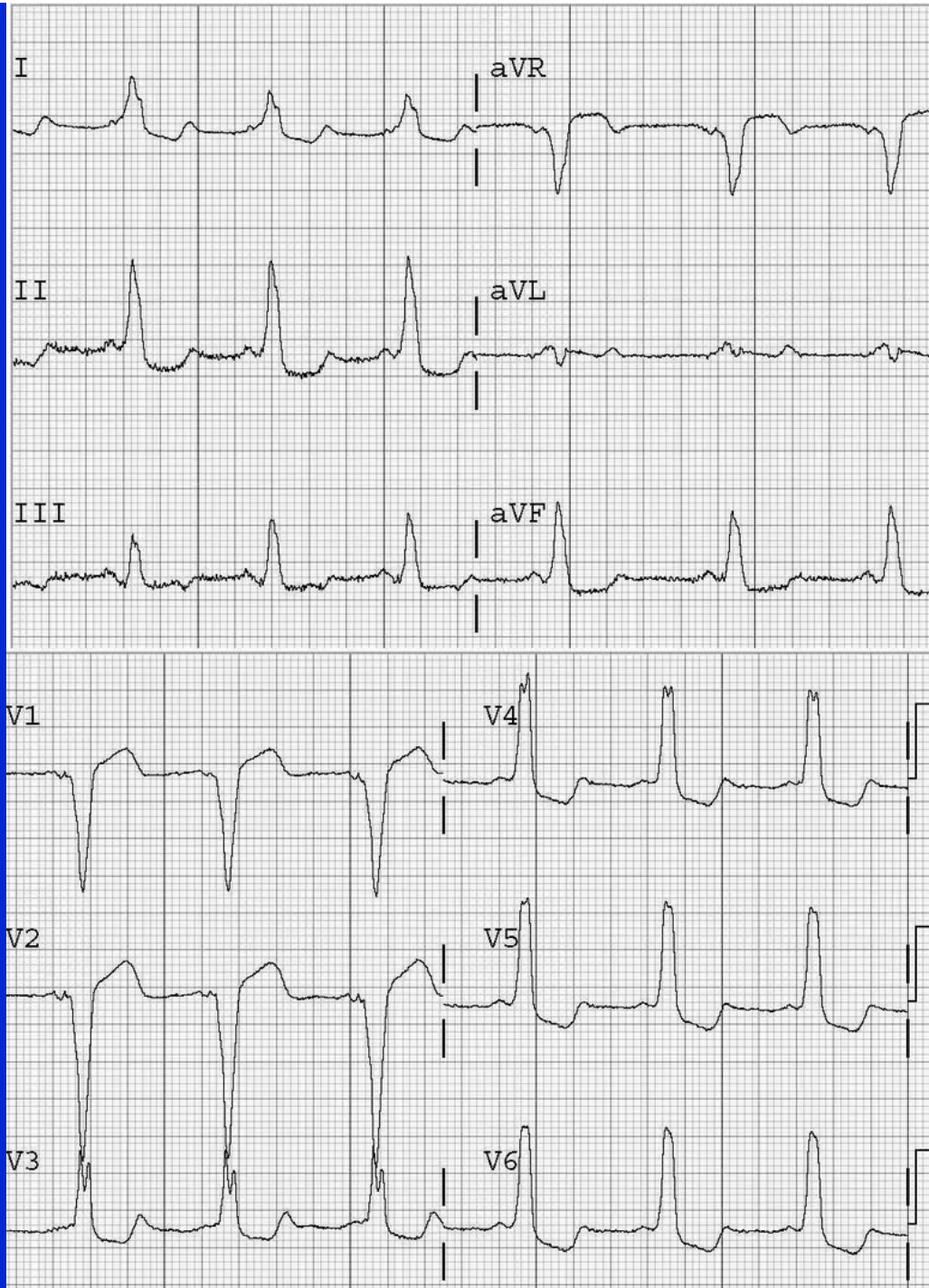
Hyperkalemia

- Manifests with ECG findings that can resemble ACS
- ECG presentation may correlate unreliably with level of serum potassium
- May require immediate field treatment and management for arrhythmias
- History should be highly suggestive! Renal insufficiency is a major risk factor. Also consider meds and major soft tissue trauma (crush syndrome, burns) as potential causes.
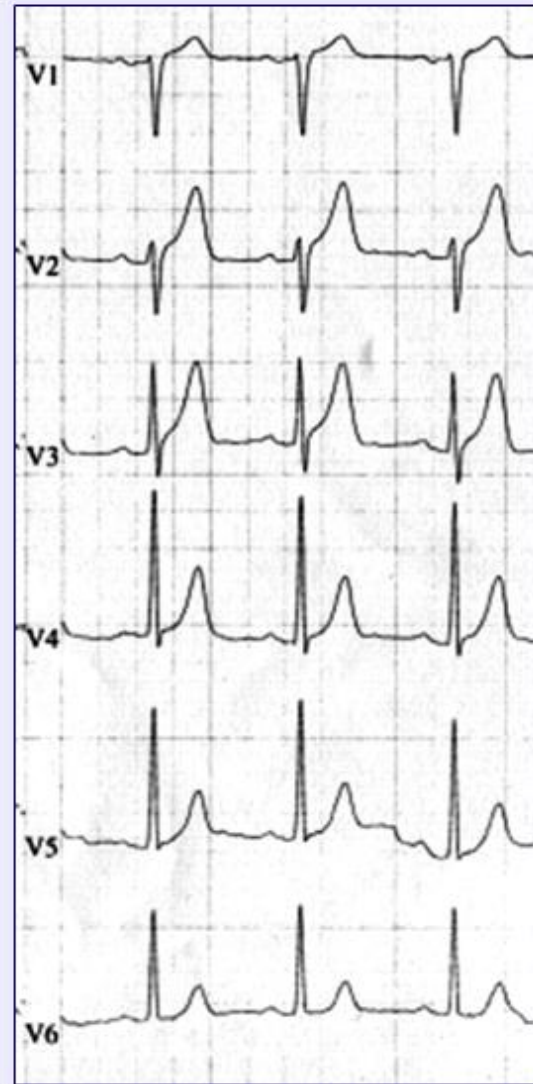
Hyperkalemia

ECG findings:

- Early sign is hyperacute T waves, which classically appear:
    - Fairly symmetric
    - Narrow at the base and slim
    - With a "sharp" point
    - With a concave ST segment

- As it progresses, the QRS and T start to widen and merge, which both can cause apparent ST elevation (or depression if QRS is positive) and can start to hide the narrowness of the T waves
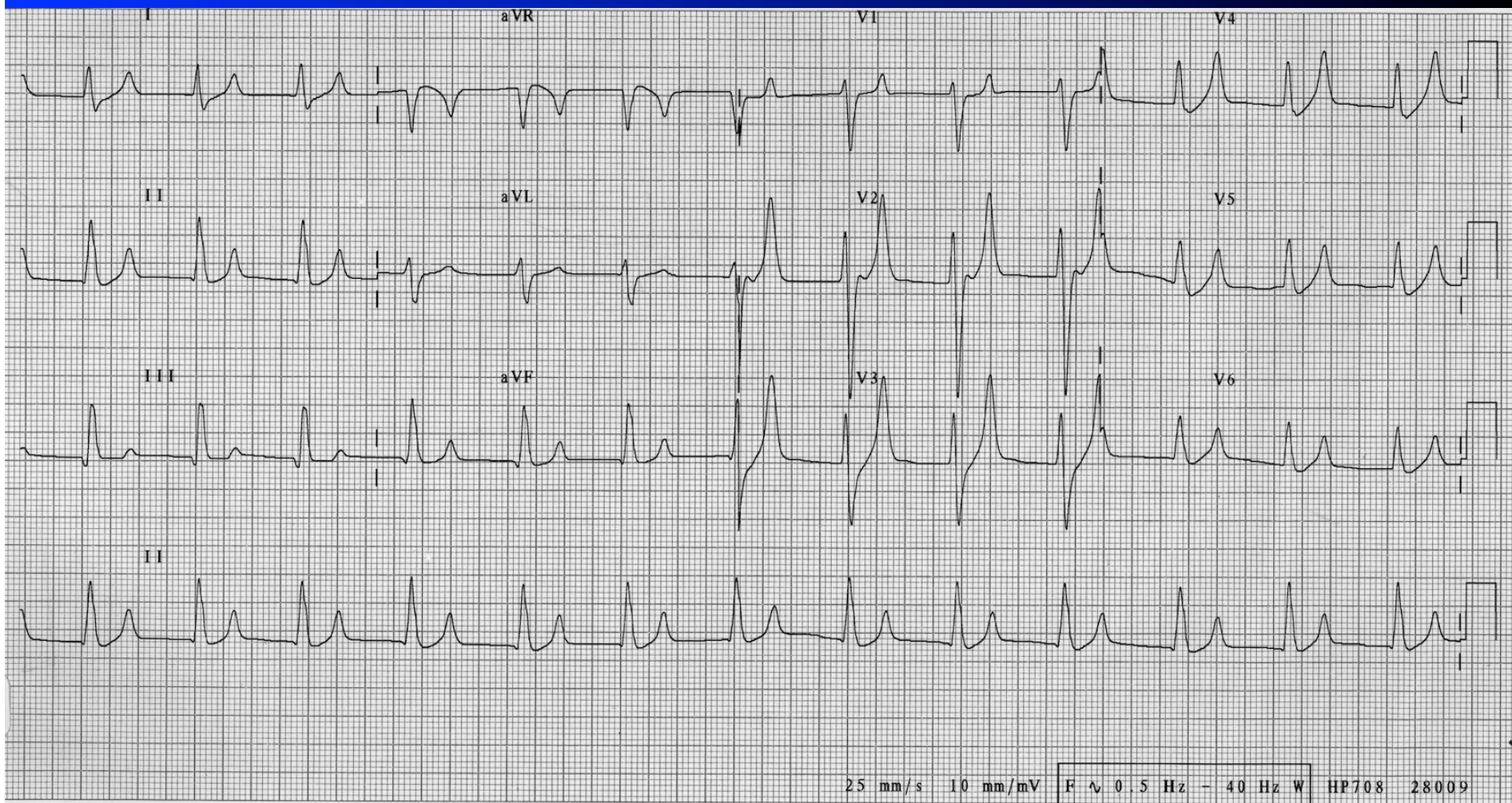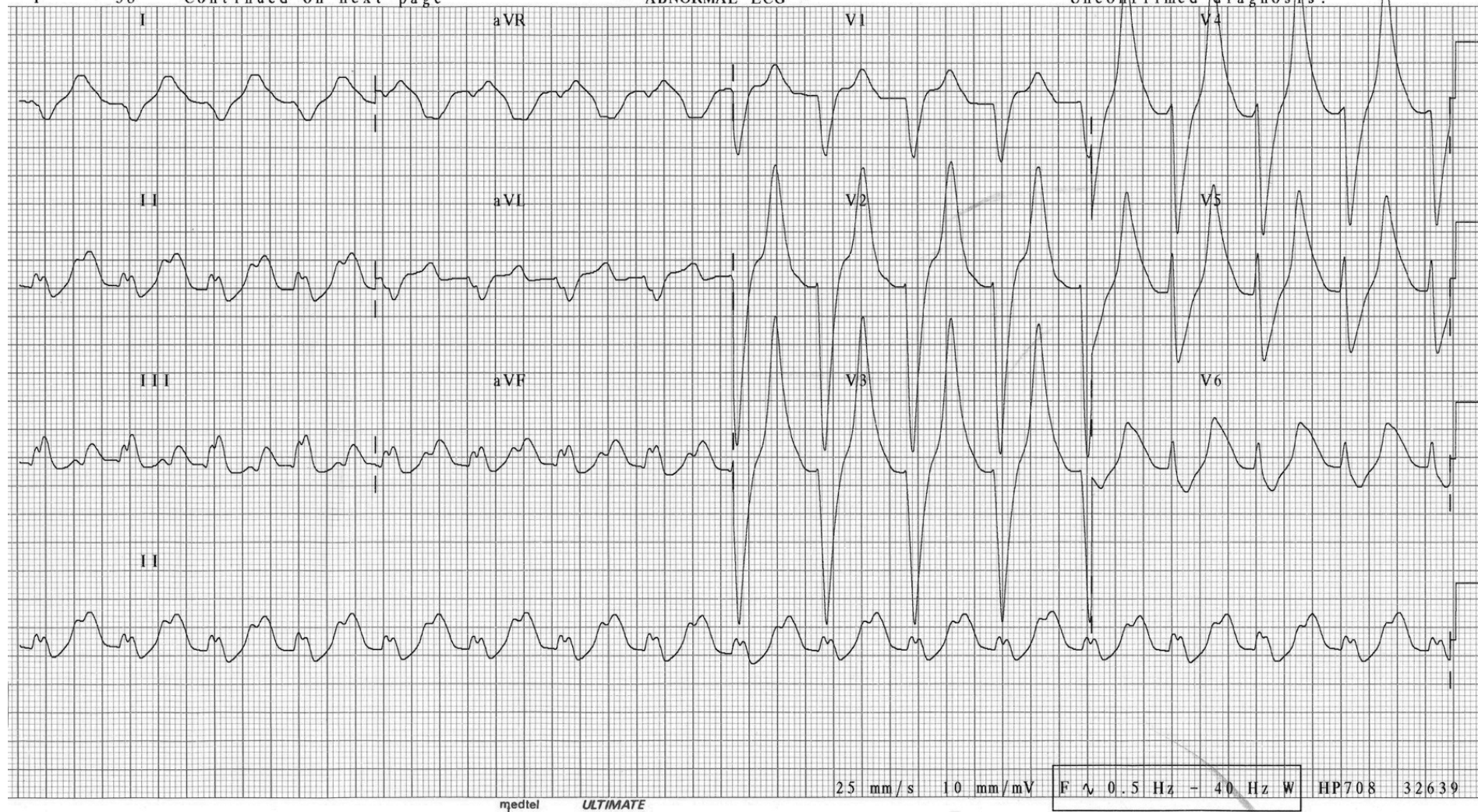
Hyperkalemia

Acute MI

Chest 2004; 125:1561-1576

Potassium 7.1

25 mm/s 10 mm/mV F ∿ 0.5 Hz − 40 Hz W HP708 32639

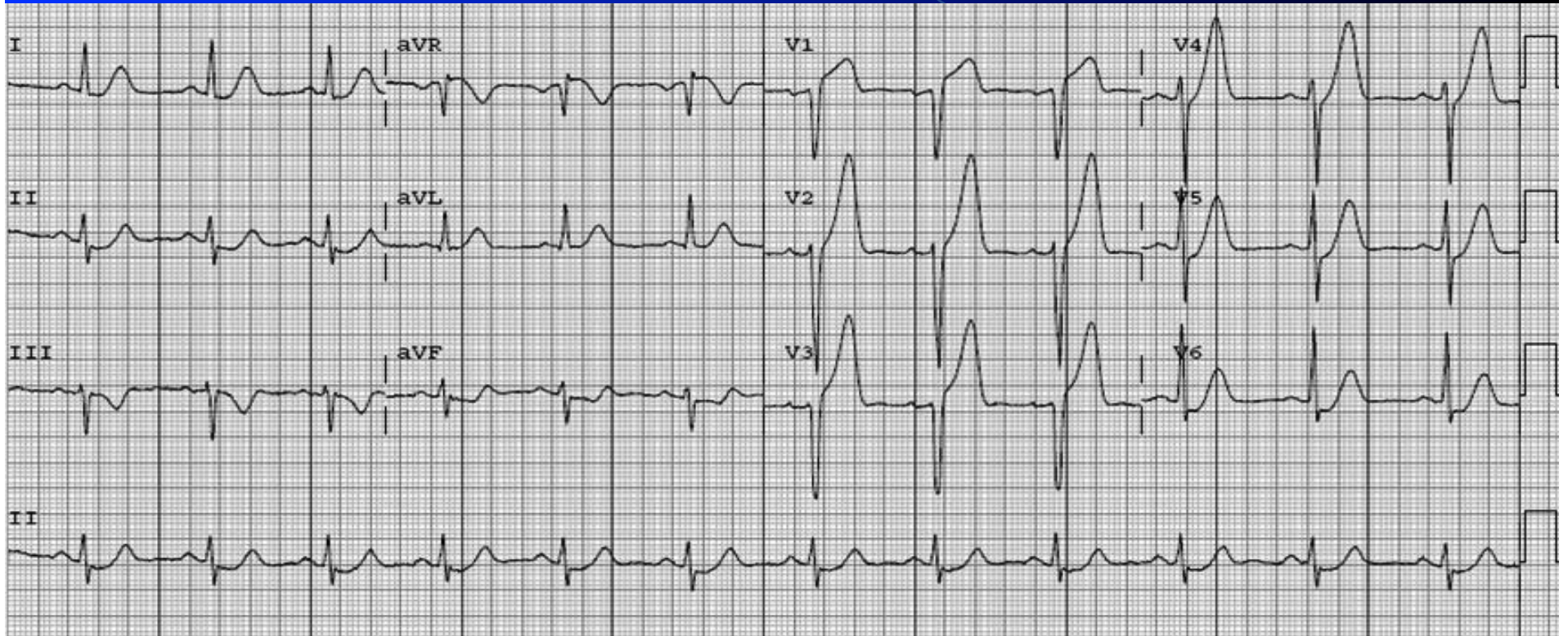medtel ULTIMATE
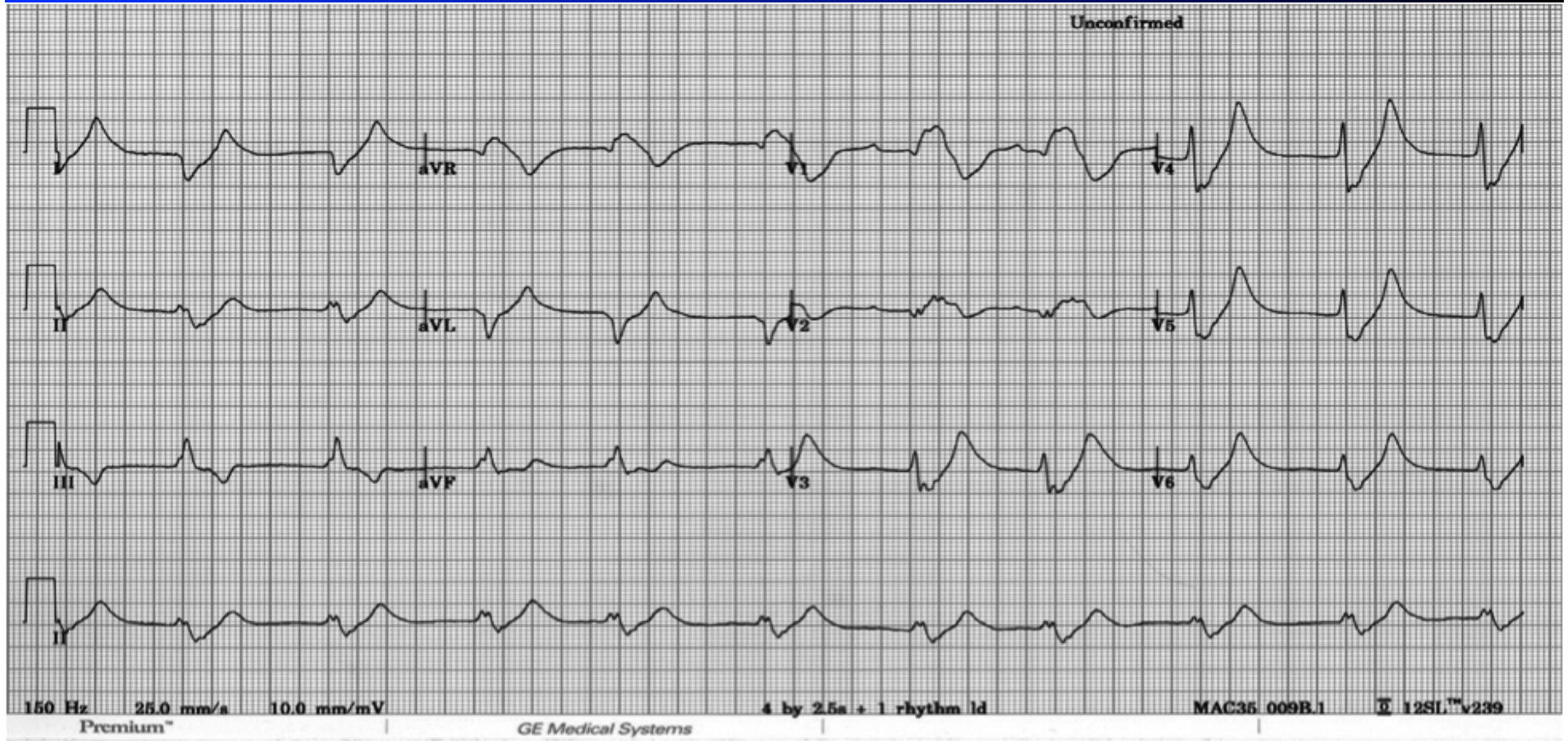
Potassium 8.5

Potassium 9+

AMI

Hyperkalemia

Bottom line:
- Hyperkalemia should be in your differential for every known dialysis patient with general complaints or altered mental status
- Most diagnostic early ECG change is T wave morphology: peaked and narrow
- More advanced stages may be less clear, but by then (as QRS begins to resemble BBB or ventricular rhythm) it should be obvious there is something other than AMI going on
- Guard against arrhythmias and manage acutely (calcium, bicarb, fluids, etc.)

Tako-Tsubo Cardiomyopathy
Aka **Stress Cardiomyopathy**
Transient Apical Ballooning
"Ampulla" Cardiomyopathy
or **Broken Heart Syndrome**

- An interesting acute cardiac disorder, with growing recent awareness; first described in Japan
- 90% of cases occur in post-menopausal women, often without substantial cardiac risk factors

Tako-Tsubo Cardiomyopathy

- Involves a transient myocardial stunning of the LV apex, causing the heart at systole to resemble an "octopus pot" (tako-tsubo)
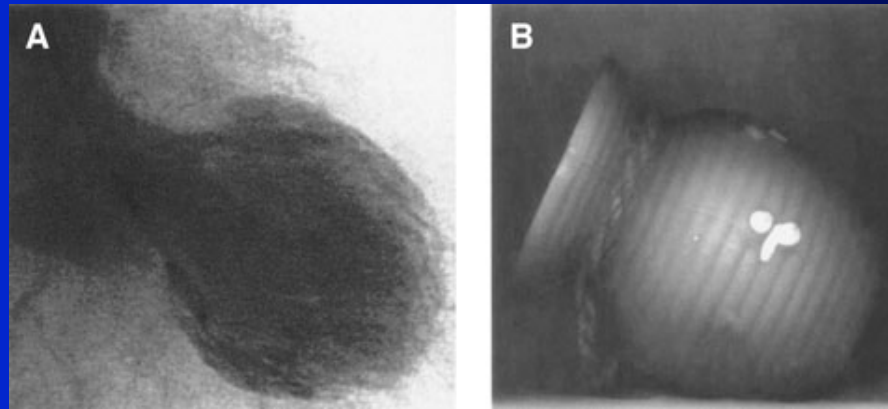
- Its hallmark feature is an onset provoked (in >2/3 of cases) by stress: emotional (bad news, near-drowning), physical (trauma), chemical (drugs)

Tako-Tsubo Cardiomyopathy

- Can resemble AMI in nearly every respect: clinically, ECG, biomarkers. Primary diagnosis occurs when no acute occlusions are found on angio.

- ECG changes may resemble Prinzmetal's, with modest ST elevation and bizarre T wave inversion

- Mechanism not understood; theories include vasospasm, aborted MI, and various neurogenic pathways

- Provoking stressor can be acute or chronic

Tako-Tsubo Cardiomyopathy

- Primary harm is from acute heart failure (LVEF often ~40%)

- Like cardiac stunning secondary to brain injury, prognosis is generally excellent if the acute period is survived; no true myocardial damage has been incurred

- Patients may require cardiovascular support if cardiogenic shock exhibits

Tako-Tsubo Cardiomyopathy

Bottom line:

- Sudden onset of ACS-like symptoms after some form of stress should make you suspicious

- Nevertheless there is *no* way to rule out true MI; most patients will be going to the cath lab regardless

*Tomich, et al. **Cardiomyopathy, Takotsubo**. eMedicine. http://emedicine.medscape.com/article/1513631-overview*

And that's that!

Bringing it all together

So how do you make sense of 1,000,000 different mimics?

- **Start with the patient history and background. Is this patient young or old? History of CAD, risk factors, past procedures? Is there a provoking event? How likely is a congenital pathology? Is there an old ECG available?**

- **Consider the complaint. Is it typical for MI or more consistent with another etiology?**

Bringing it all together

- Obtain an early 12-lead, before treatment if possible. Are there ST or T-wave changes? How profound? What is their morphology?

- Are there reciprocal changes?

- What does the computer think?

- Does the ECG support an alternate diagnosis? Would it explain the chief complaint? Is it more or less likely than MI? If likely, what is the probability of a comorbid MI?

Bringing it all together

- Is there any chance of an alternate diagnosis that is as or more pressing than STEMI? (Aortic dissection, advanced hyperkalemia, etc.)

- Obtain serial ECGs to guide or confirm diagnosis.

- Weigh risk/benefit of transport destinations. Consult medical control as appropriate.

- Provide your findings to the receiving staff.

That's all, folks! Go home!
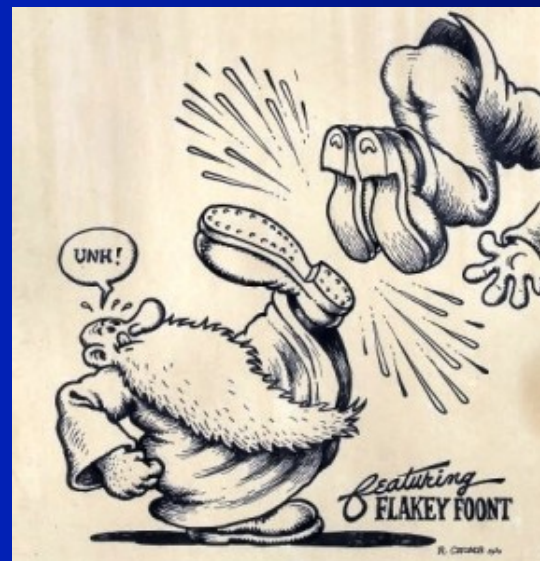
Thanks to:

- **Tom Bouthillet, FF/EMT-P, of ems12lead.com, for much of the information, many of the example ECGs, and the spiritual inspiration for this material**

- **Dr. Stephen Smith for numerous ECGs and many of the unpublished evidence-based rules provided**

- **Dr. Brady, Dr. Sgarbossa, and the other researchers whose work is heavily featured**