Evidence-Based Medicine: Clinical Utility of Conventional and High-Sensitivity Cardiac Troponin Levels

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CUSOM

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CAMPBELL UNIVERSITY | Jerry M. Wallace School of Osteopathic Medicine

Clinical Utility of Conventional and of high-sensitivity cardiac troponins: Objectives

- Define cardiac troponins (cTn).
- Define high-sensitivity cardiac troponins (hs-cTn).
- Describe how hs-cTn levels are used to diagnose acute MI.
- List the differential diagnosis of an elevated hs-cTn.
- Discuss how hs-cTn is used to dx MI in acute CHF or CKD.
- Describe how hs-cTn levels are incorporated into clinical decision making.

Let’s see some patients . . .

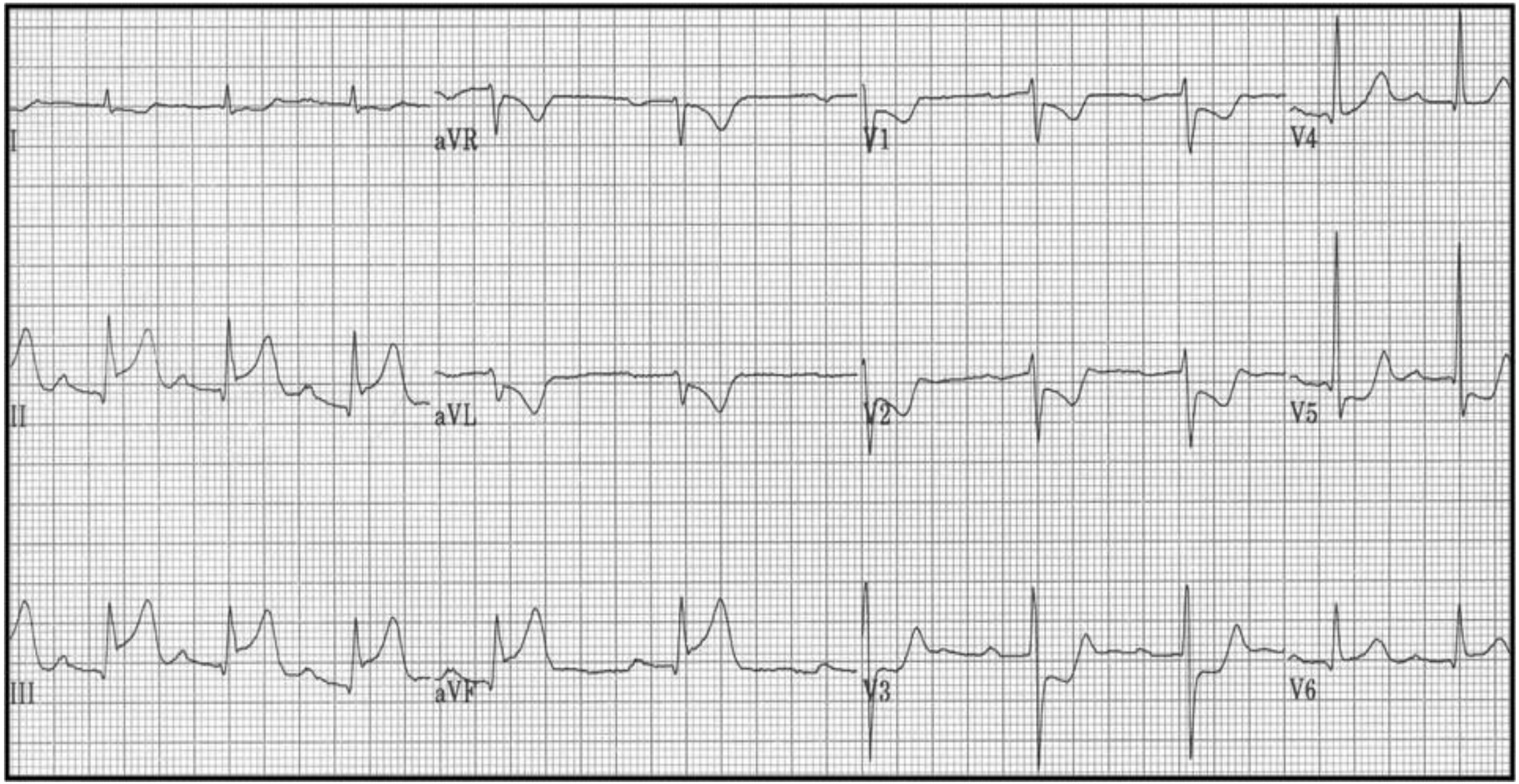
Patient 1

A 65-year-old woman with a history of HTN and tobacco abuse presents with intermittent nausea and vomiting with shortness of breath for the last 12 hours.

On exam, she appears mildly short of breath and diaphoretic.
Vital signs: bp 110/60 p 116 RR 28 temp 98.6F O2sat 92% on RA
HEENT: dry mucous membranes
Car: r/r/r, tachycardic without murmur. JVP 7 cm (normal)
Lungs: bilateral rales
Abd: Soft with mild epigastric tenderness, no rebound and no organomegaly
Extr: no edema. Dp pulses 1+ bilaterally

CBC: wbc is elevated at 15,000 with a left shift
H/H are normal
Chem-8 is significant for a low potassium of 3.1

An EKG is performed. . .



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Serial high-sensitivity cardiac troponin I (hs-cTI) are as follows:

Time from presentation	**0**	**3 hours**	**6 hours**
hs-cTnI (ng/L) Normal < 14 ng/L	12	246	180

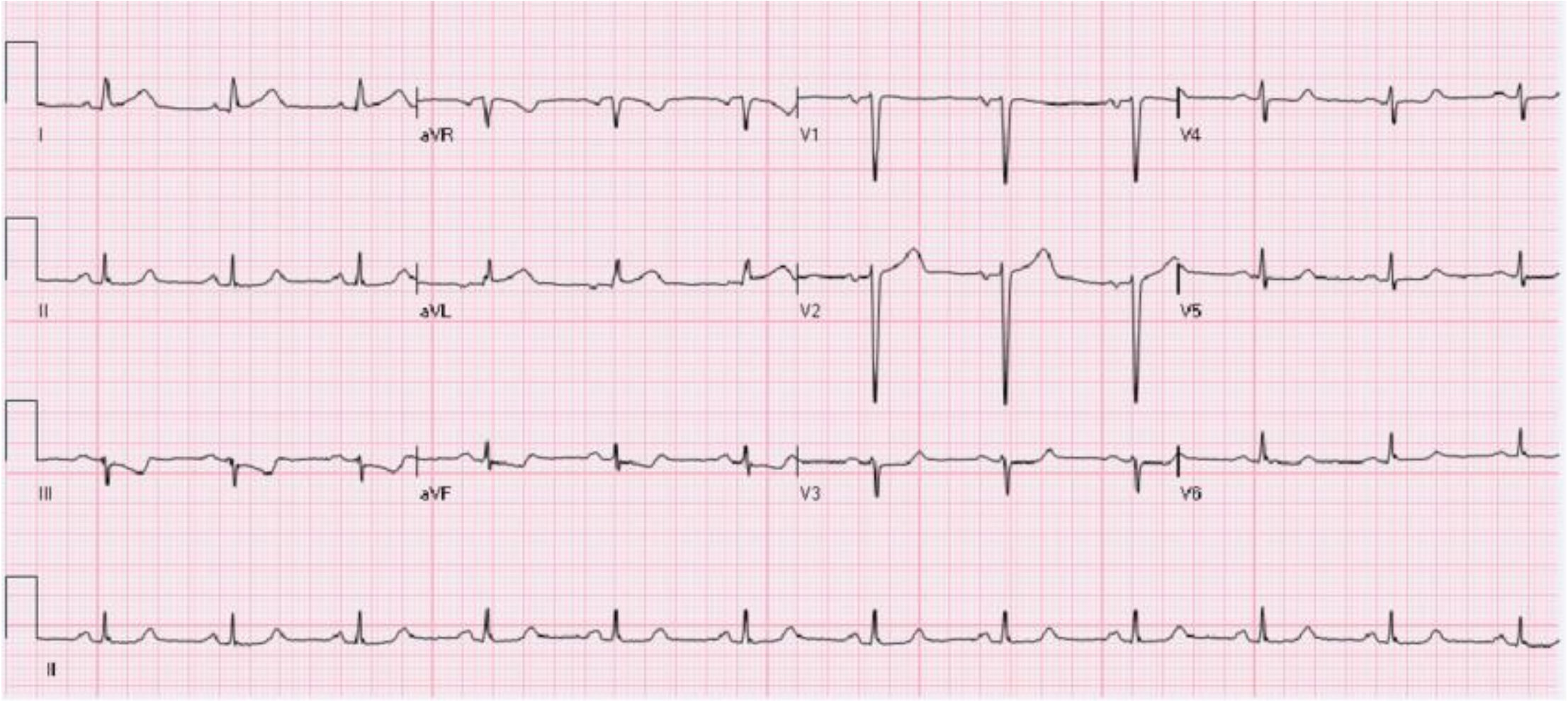
Patient 2

A 68-year-old man with a hx of tobacco abuse, HTN and type 2 diabetes presents to the ED with a two week history of worsening fatigue with exertion. He describes central chest pressure with radiation to his neck and left arm with nausea and shortness of breath. Walking up more than 10 steps at home precipates these symptoms. Resting about 10 minutes relieves these symptoms.

His appetite and weight have not changed recently. He denies fever, cough, changes in bowel movement or changes in urination.

On exam, he is resting comfortably.
Vital signs: bp 130/92 p 86 RR 14 temp 98.6 F O2 sat 95% on RA
JVP 8 cm (normal)
Car: r/r/r with a 3/6 systolic murmur at the right upper sternal border radiating to the neck
Lungs: CTA
Extr: no edema: dp pulses trace B/L

An EKG is performed . . .



Non-specific T wave changes

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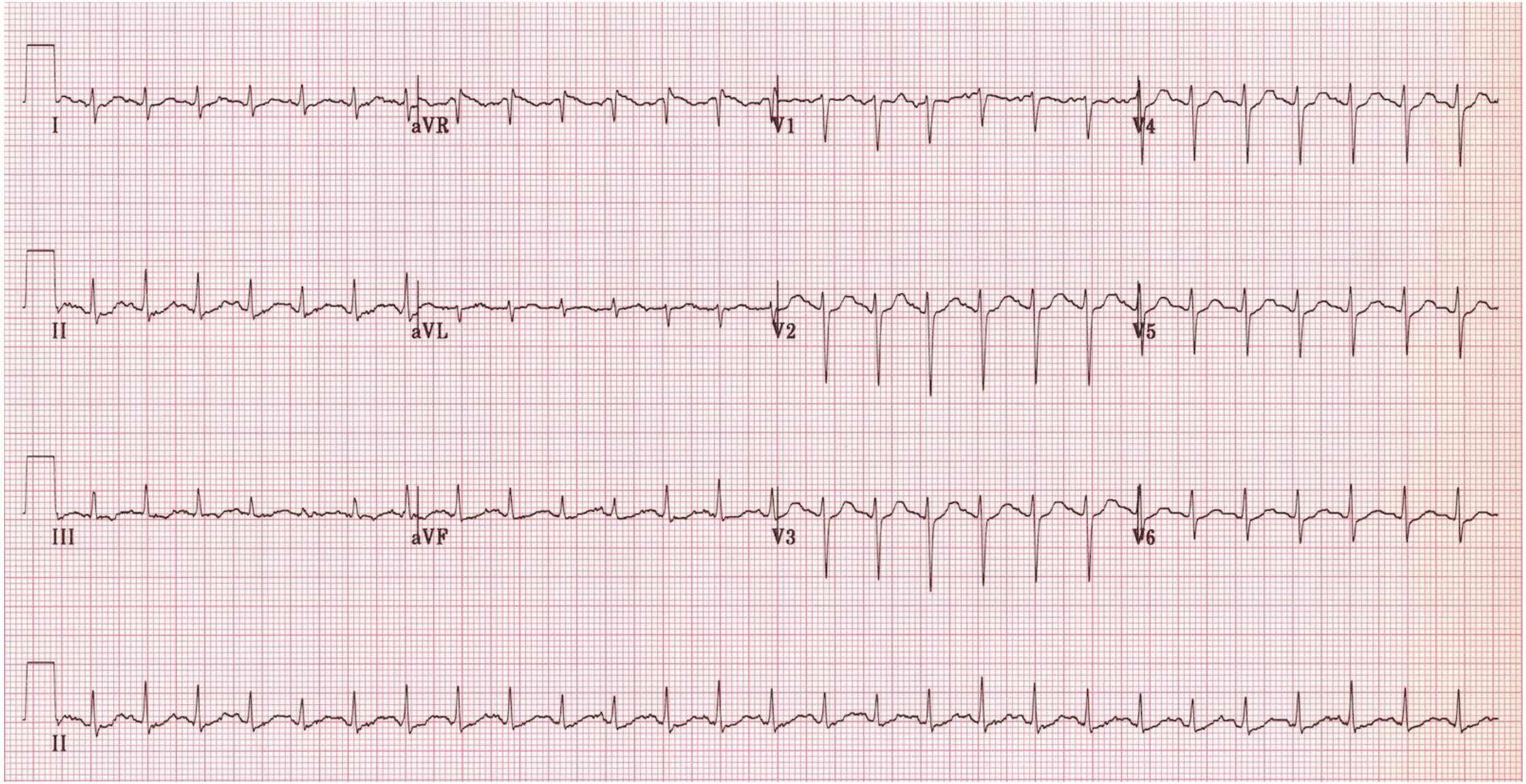
Patient 3

A 72-year-old man residing in a nursing home presents to the ED with delirium. He has a hx CAD with MI in 1998, CKD, and a cholecystectomy in 2001 and a splenectomy in the 1980's. He is arousable to sternal rub only and has rapid shallow breathing.

On examination, vitals: bp 110/70 p 150 RR 30 , and temp 100.8F with 02 sat 86% on RA. He has equal pupils with roving eye movements, a stiff neck, lungs with scattered rhonchi, tachycardia with a soft systolic murmur at the right upper sternal border and a nontender abdomen.

A CXR shows RLL and LUL infiltrates. A head CT is negative.

An EKG is performed . . .



Sinus tachycardia

Serial high-sensitivity cardiac troponin I (hs-cTI) are as follows:

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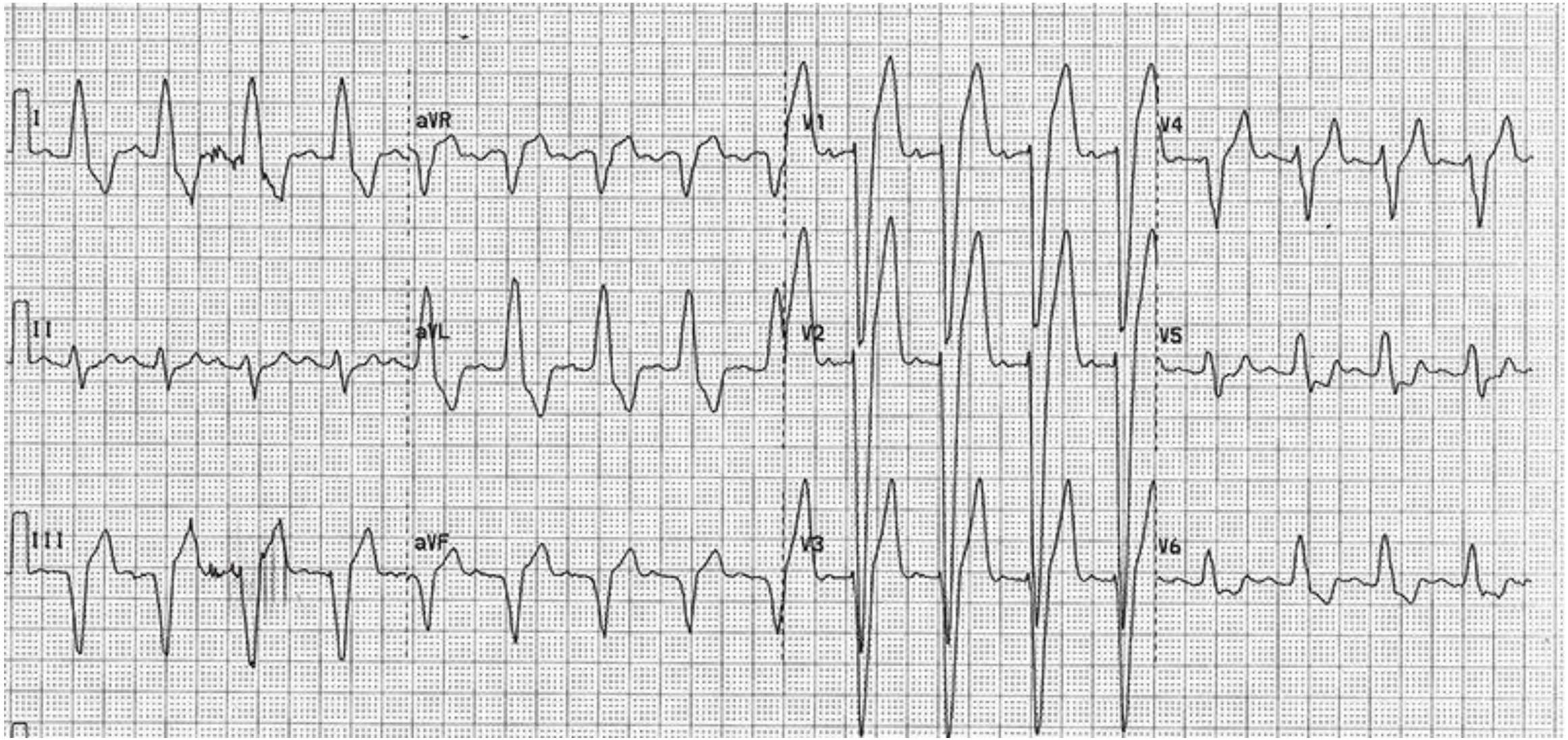
Patient 4

A 65-year-old man with history of HTN, hyperlipidemia and HFrEF presents with shortness of breath for the last 24 hours.

On exam: vital signs: blood pressure 100/60 p 96 RR 28 afebrile. O2 sat 90% on RA
car: r/r/r with a 3/6 holosystolic murmur at the left sternal border radiating to the left axilla. JVP to angle of mandible
lungs: bilateral rales.
Extremities: 2+ edema bilaterally

An EKG is performed . . .



Sinus tachycardia, left bundle-branch block, LVH

Serial high-sensitivity cardiac troponin I (hs-cTI) are as follows:

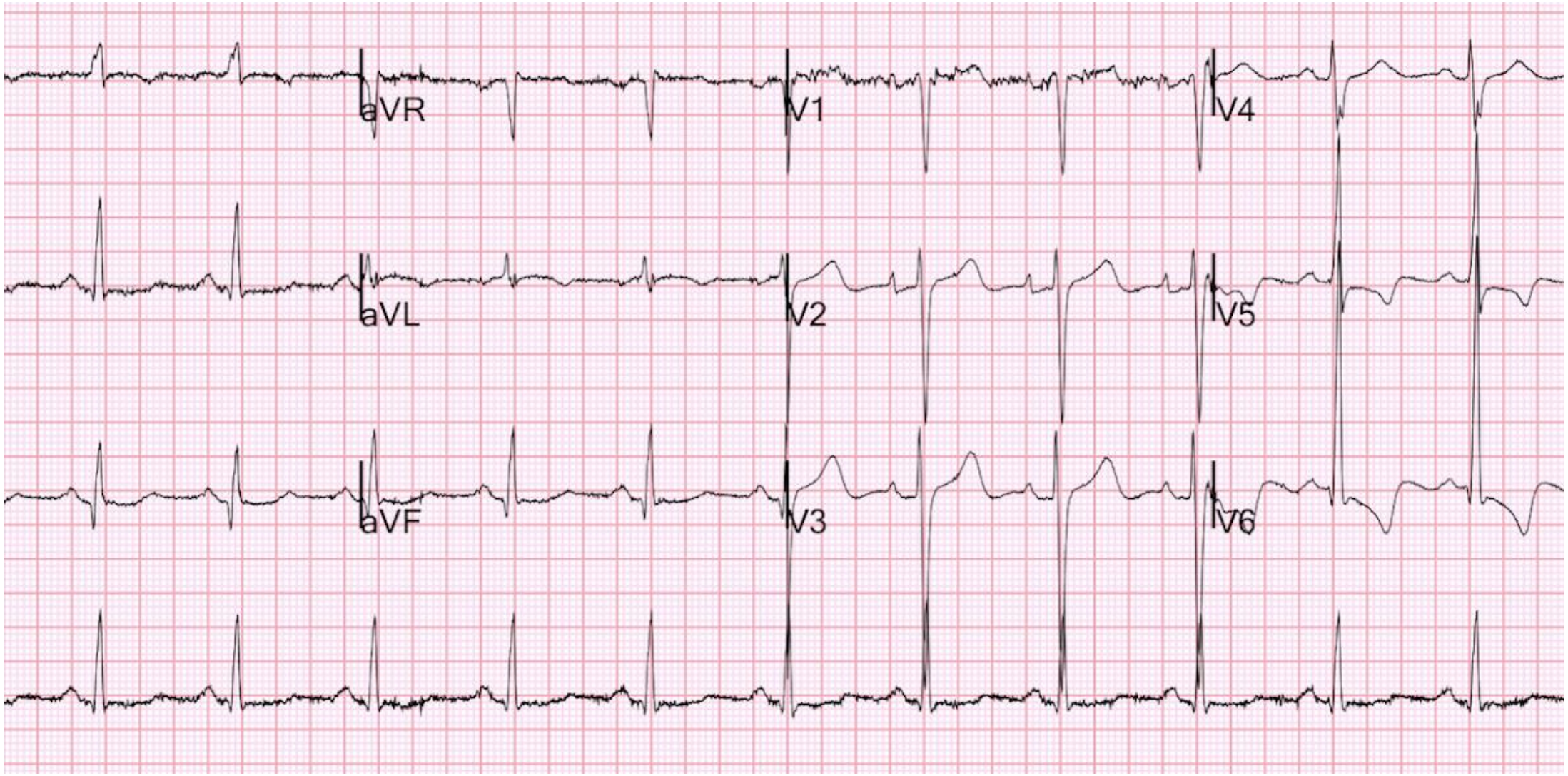
Time from presentation	**0**	**3 hours**	**6 hours**
hs-cTnI (ng/L) Normal < 14 ng/L	25	60	48

Patient 5

You are called to see a 61-year-old man with ESRD on hemodialysis three times per week to evaluate him for new onset shortness of breath. He describes forgetting to take his bp medication yesterday. He denies chest discomfort but is slightly nauseated.

On exam, vitals: bp 190/110 p 100 RR 22 temp 97.1F
JVP 9 cm at 45 degrees (upper limit of normal)
Car: r/r/r with S4
Lungs: bibasilar rales
Abd: soft, nontender
Extr: L arm with AV fistula with strong thrill and no warmth or erythema Legs with 1+ edema B/L and dp pulses trace

An EKG is performed . . .



Sinus rhythm, non- specific ST and T-wave changes

After one hour of dialysis, his shortness of breath resolves . . .

Serial high-sensitivity cardiac troponin I (hs-cTI) are as follows:

Time from presentation	**0**	**3 hours**	**6 hours**
hs-cTnI (ng/L) Normal < 14 ng/L	43	39	45

Clinical Utility of Conventional and of high-sensitivity cardiac troponins: Objectives

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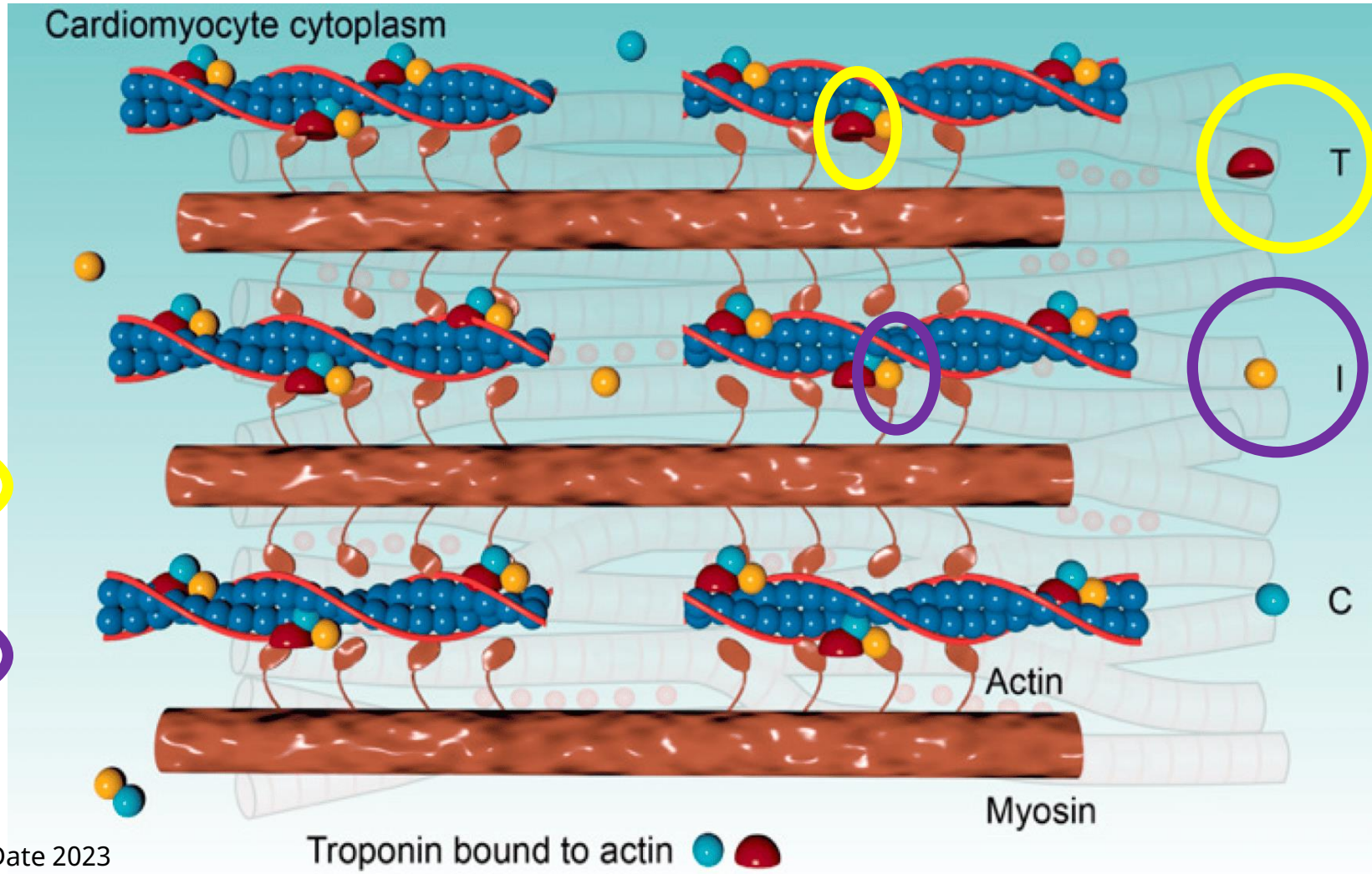
What are Troponins?

- Regulatory proteins
- Unique to heart
- Control interaction of actin and myosin via calcium
- Cardiac troponin T: cTnT
- Cardiac troponin I: cTnI

Troponin I and Troponin T have about the same clinical utility.



Allan S Jaffe, MD, David A Morrow, MD, MPH, UpToDate 2023

Int. J. Mol. Sci. **2019**, *20*(13), 3351; https://doi.org/10.3390/ijms20133351

What are troponins?

- Unique to the heart

- However, *some* patients with chronic skeletal muscle diseases have **chronic** elevations of cTnT with a **normal** cTnI

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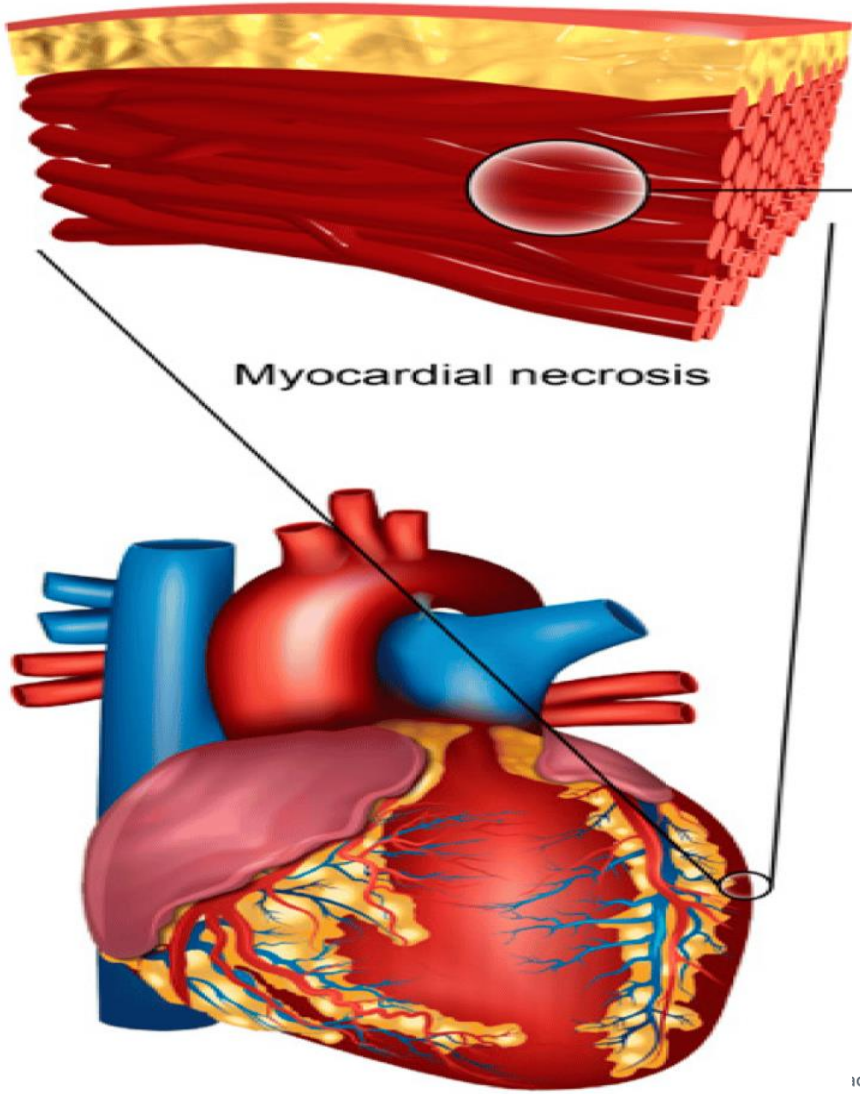
How are troponins used clinically to diagnose myocardial infarction?



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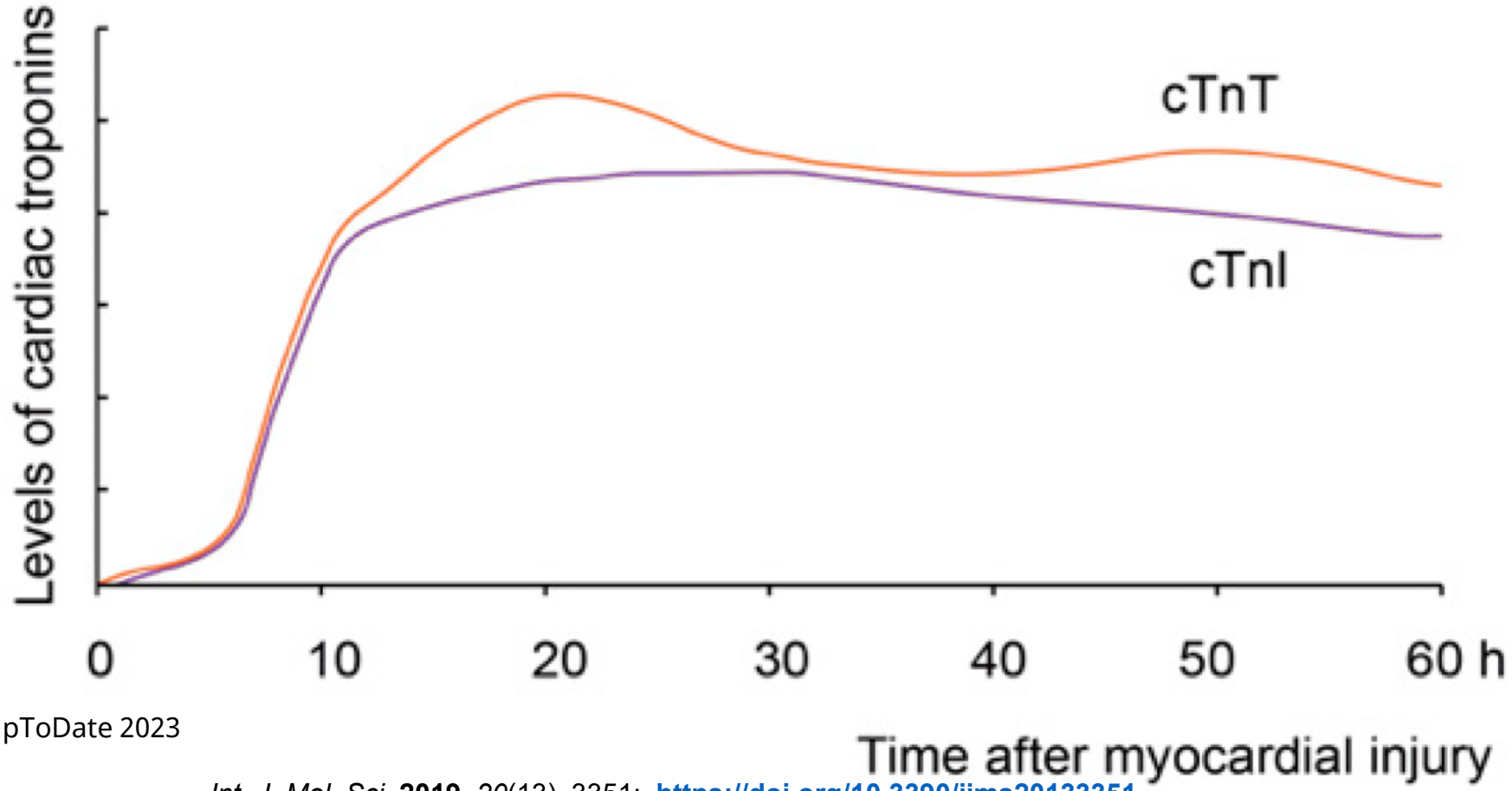
What is Troponin?

When cardiac muscle dies, myocyte integrity is compromised and multiple intracellular enyzmes and proteins are detectable in the circulation, including troponin . . .

Troponin levels begin to rise two to three hours after myocardial injury



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Measuring Troponin Levels

- Use of enzyme-linked immunosorbent assay (ELISA)
- ELISA uses monoclonal antibodies against cTnT or cTnI
- The upper limit of normal is defined as the 99th percentile in a given population

- **Original** troponin levels were measured in nanograms/ml with normal range of 0.0 to 0.04 ng/**ml**
- Normal Troponin I range per Cape Fear Valley Medical Center Lab is 0.000 to 0.045 ng/ml

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Measuring Troponin Levels

- **High-sensitivity** cardiac troponin levels (either hs-cTnI or hs-cTnT) detect levels several magnitudes lower than then original cTn

- Therefore, hs-cTn levels are reported as nanograms/**L**

- The upper limit of normal is defined as the 99th percentile in a given population

- Therefore the normal range of **high sensitivity** cardiac troponins is less than 14 ng/L in most labs.

- Note, that **most healthy people have detectable troponins** when high sensitivity assays are used

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Interpreting Elevated hs-cTn Levels

- hs-Tn above upper limit of normal (ie 99th percentile), usually 14 ng/L or
- If hs-cTn is below the 99th percentile, a change of 50% of upper limit of normal (ie. change of 7 ng/L from baseline)

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What is the ddx of an elevated high-sensivity cardiac troponin (hs-cTn)?

Injury related to primary myocardial ischemia

- Plaque rupture
- Intraluminal thrombus

Januzzi JL, et al. **JACC** 2019;73(9): 1059-1077

What is the ddx of an elevated high-sensivity cardiac troponin (hs-cTn)?

Injury related to myocardial oxygen supply/demand imbalance

- Tachy/bradyarrhythmia
- Aortic dissection or severe aortic valve disease
- Hypertrophic cardiomyopathy
- Cardiogenic, hypovolemic or septic shock

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What is the ddx of an elevated high-sensivity cardiac troponin (hs-cTn)?

Injury related to myocardial oxygen supply/demand imbalance

- Severe respiratory failure
- Severe anemia
- Hypertension with or without LVH
- Coronary endothelial dysfunction, spasm or dissection

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What is the ddx of an elevated high-sensivity cardiac troponin (hs-cTn)?

Injury not related to myocardial ischemia

- Cardiac contusion, surgery, ablation, pacing or defibrillation
- Rhabdomyolysis with cardiac involvement
- Myocarditis
- Cardiotoxic agents (ex: anthracyclines, Herceptin)

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What is the ddx of an elevated high-sensivity cardiac troponin (hs-cTn)?

Multifactorial or indeterminate myocardial injury

- Heart failure
- Stress cardiomyopathy
- Pulmonary embolism
- Pulmonary hypertension
- Sepsis
- Critical illness

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What is the ddx of an elevated high-sensivity cardiac troponin (hs-cTn)?

Multifactorial or indeterminate myocardial injury

- Renal failure
- Severe acute neurologic disease (ex: stroke, subarachnoid hemorrhage)
- Infiltrative cardiomyopathy (ex: amyloidosis, sarcoidosis)
- Strenuous exercise

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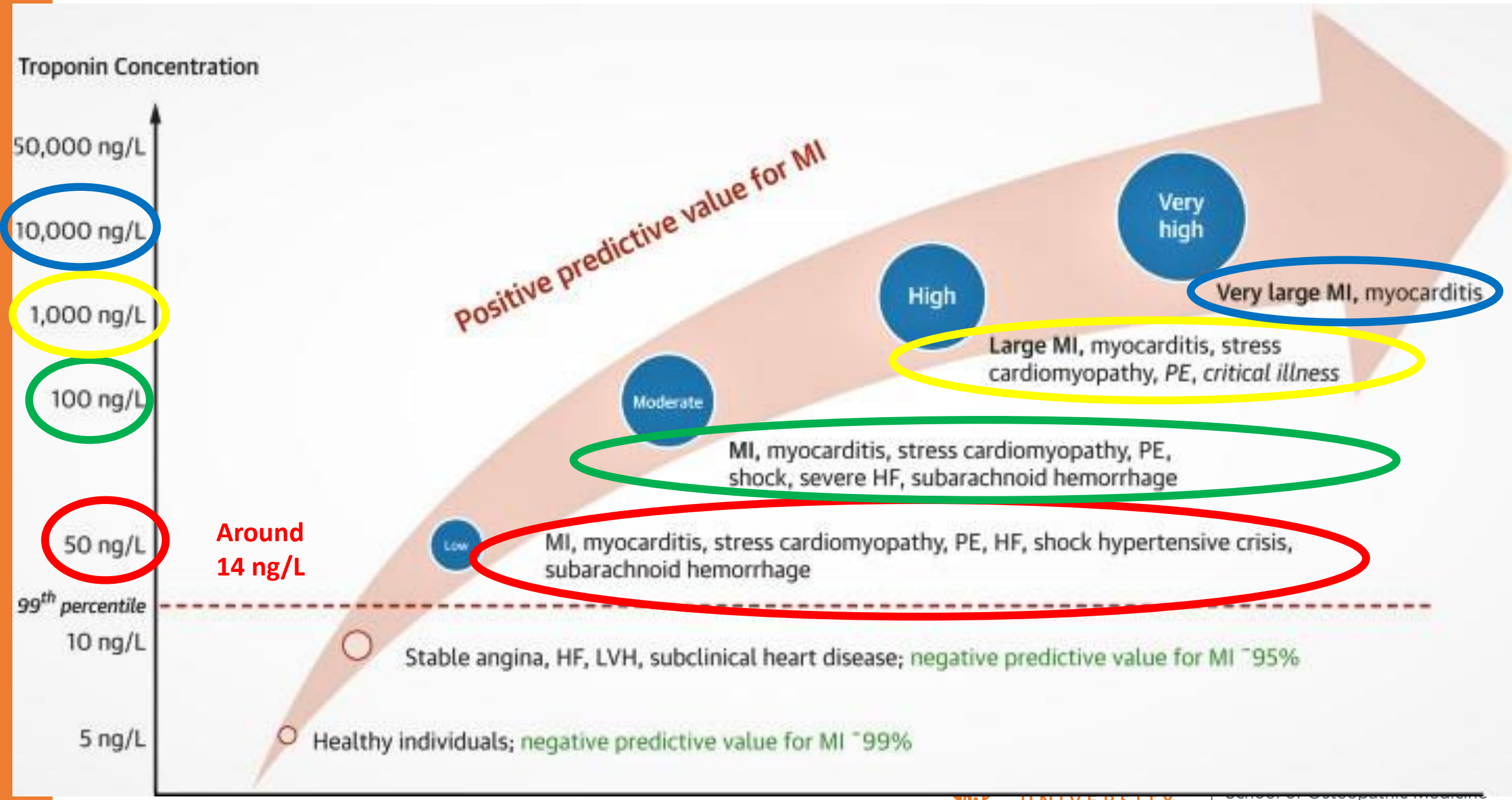
hs-cTn levels *alone* cannot distinguish among these diagnoses . . .

- **Injury related to primary myocardial ischemia**
- **Injury not related to myocardial ischemia**
- **Injury related to myocardial oxygen supply/demand imbalance**
- **Multifactorial or indeterminate myocardial injury**

Clinicians need more information from other sources . . .


Troponin Concentration
50,000 ng/L
10,000 ng/L
1,000 ng/L
100 ng/L
50 ng/L
99th percentile
10 ng/L
5 ng/L
Positive predictive value for MI
Very high
High
Moderate
Low
Very large MI, myocarditis
Large MI, myocarditis, stress cardiomyopathy, PE, critical illness
MI, myocarditis, stress cardiomyopathy, PE, shock, severe HF, subarachnoid hemorrhage
MI, myocarditis, stress cardiomyopathy, PE, HF, shock hypertensive crisis, subarachnoid hemorrhage
Around 14 ng/L
Stable angina, HF, LVH, subclinical heart disease; negative predictive value for MI ~95%
Healthy individuals; negative predictive value for MI ~99%

"An abnormal hs-cTn is central to the diagnosis of AMI but MI is a clinical diagnosis that is not defined by troponin alone".

"An elevated hs-cTnI or T without other corroborating evidence is not sufficient for a diagnosis of AMI, even if a rise or fall is detected".

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What is a significant change in troponin consistent with AMI?

- Biochemical definition of AMI with serial troponins:
  - Level > 99th percentile for the troponin assay being used

  AND
  - A rise and fall in serial troponins must be confirmed

- If troponins remain < 99th percentile then troponin rise of 50% to 80% from baseline *may also suggest* AMI

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What is a significant change in troponin in acute CHF?

- hs-cTn levels above the 99% percentile are *common* in acute decompensated CHF
- Serial hs-cTn levels can also demonstrate a rise and fall as in AMI
- hs-cTn levels can rise and fall in acute CHF due to:
  - Myocardial ischemia
  - Myocardial stress
  - Cardiomyocyte apoptosis
- Rise and fall in hs-cTn from acute CHF predict:
  - Adverse ventricular remodeling
  - Future hospitalizations for CHF
  - Death

Januzzi JL, et al. **JACC** 2019;73(9): 1059-1077

What is a significant change in troponin in a patient with advanced kidney disease?

- hs-cTn levels are frequently > 99th percentile at *baseline* (ie. without acute MI) among patients with ESRD
- However, with *serial* testing, the absolute changes in hs-cTn levels are *similar* in AMI among patients with or without ESRD

Januzzi JL, et al. **JACC** 2019;73(9): 1059-1077

The other thing to keep in mind . . .

Diagnosis of coronary artery disease without infarction.

VS

Diagnosis of acute myocardial infarction.

Can be two different questions . . . .

In other words, a patient can have coronary artery disease and cardiac ischemia but NO myocardial infarction and therefore have unremarkable troponin levels.

The most important tool you have to diagnose coronary artery disease and/or acute myocardial infarction is

You

Diagnosis of Coronary Artery Disease

Pretest Probability of CAD

- Ask three questions:
  - Is the chest discomfort substernal?
  - Are the pt's symptoms precipitated by exertion?
  - Does the pt experience prompt relief (ie. Within 10 minutes) with rest or nitroglycerin?

Pretest Probability of CAD

- Classify chest symptoms:
  - Three of three questions “yes” = typical angina
  - Two or three “yes” = atypical angina
  - One or none “yes” = nonanginal chest pain

Pretest Prob of CAD: Women

Age (yrs)	Asymp (%)	Nonang (%)	Atypical (%)	Typical (%)
30-39	0.3	0.8	4.2	25.8
40-49	1.0	2.8	13.3	55.2
50-59	3.2	8.4	32.4	79.4
60-69	7.5	18.6	54.4	90.6

Gold standard of cardiac catheterization

Diagnostic Strategies for Common Medical Problems. Second Edition. Black et al (eds) Philadelphia: American College of Physicians 1999.

Pretest Prob of CAD:Men

Age (yrs)	Asymp (%)	Nonang (%)	Atypical (%)	Typical (%)
30-39	1.9	5.2	21.8	69.7
40-49	5.5	14.1	46.1	87.3
50-59	9.7	21.5	58.9	92.0
60-69	12.3	28.1	67.1	94.3

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Estimating the probability of acute coronary syndrome in the emergency department from clinical history and initial troponin level . . . The HEART Score

The HEART Score

HISTORY

Data	Score
Highly suspicious	2
Moderately suspicious	1
Slightly suspicious	0

ECG

Data	Score
Significant ST depression	2
Nonspecific repolarization disturbance	1
Normal	0

AGE

Data	Score
≥ 65 years	2
45-65 years	1
<45 years	0

CARDIAC RISK FACTORS*

Data	Score
3 or more or know CV dz	2
1 or 2	1
None	0

*bp > 140/90
*LDL > 159 mg/dl
*HDL < 40 mg/dl
*Diabetes mellitus
*Tobacco abuse
*Family history

TROPONIN

Data	Score
> 2X normal limit	2
1-2X normal limit	1
≤ normal limit	0

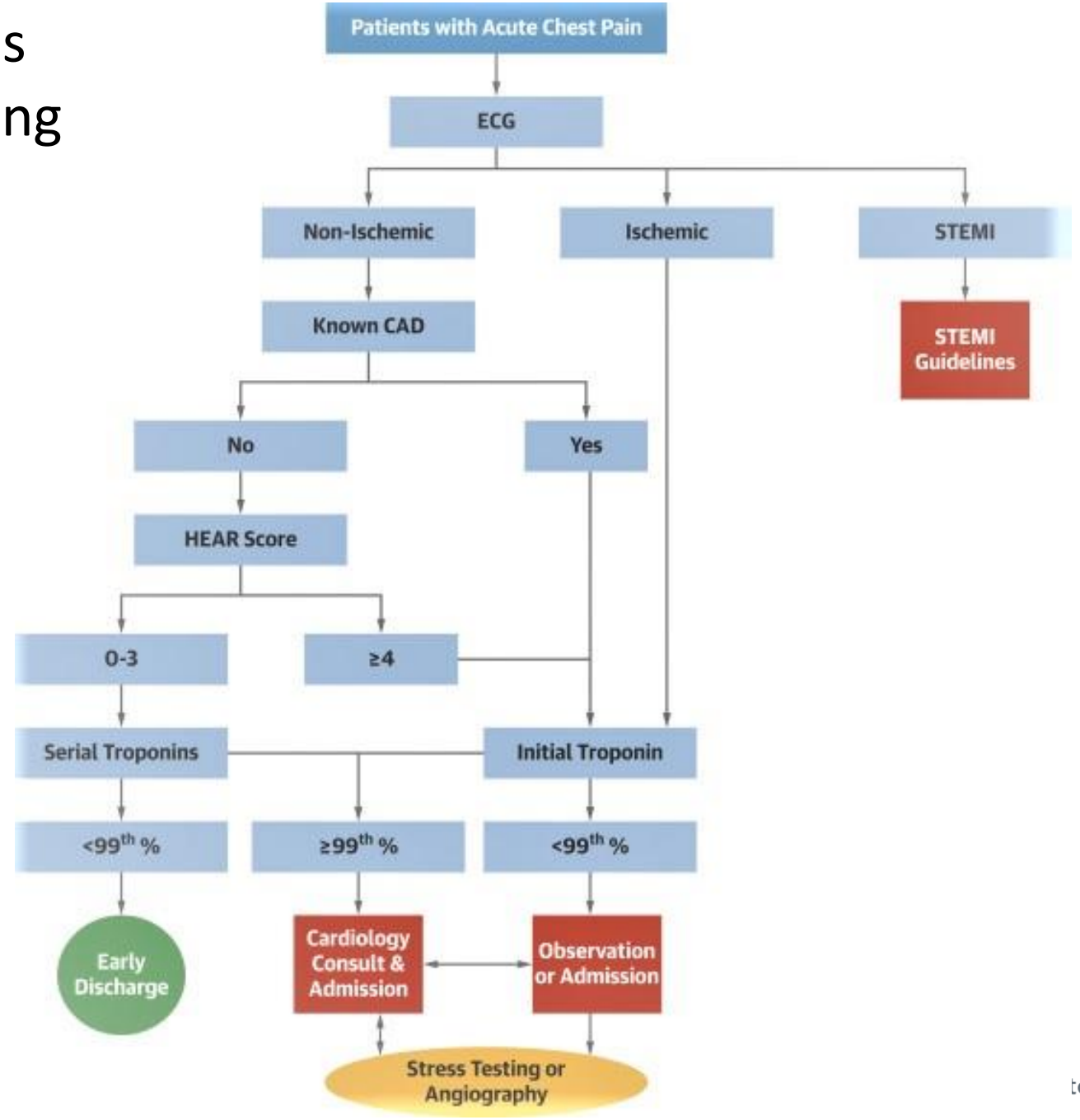
Heart score and risk for ACS:

0 to 3 = low

4 to 6 = moderate

≥ 7= high

Incorporation of Troponins into clinical decision-making



Back to our patients . . .

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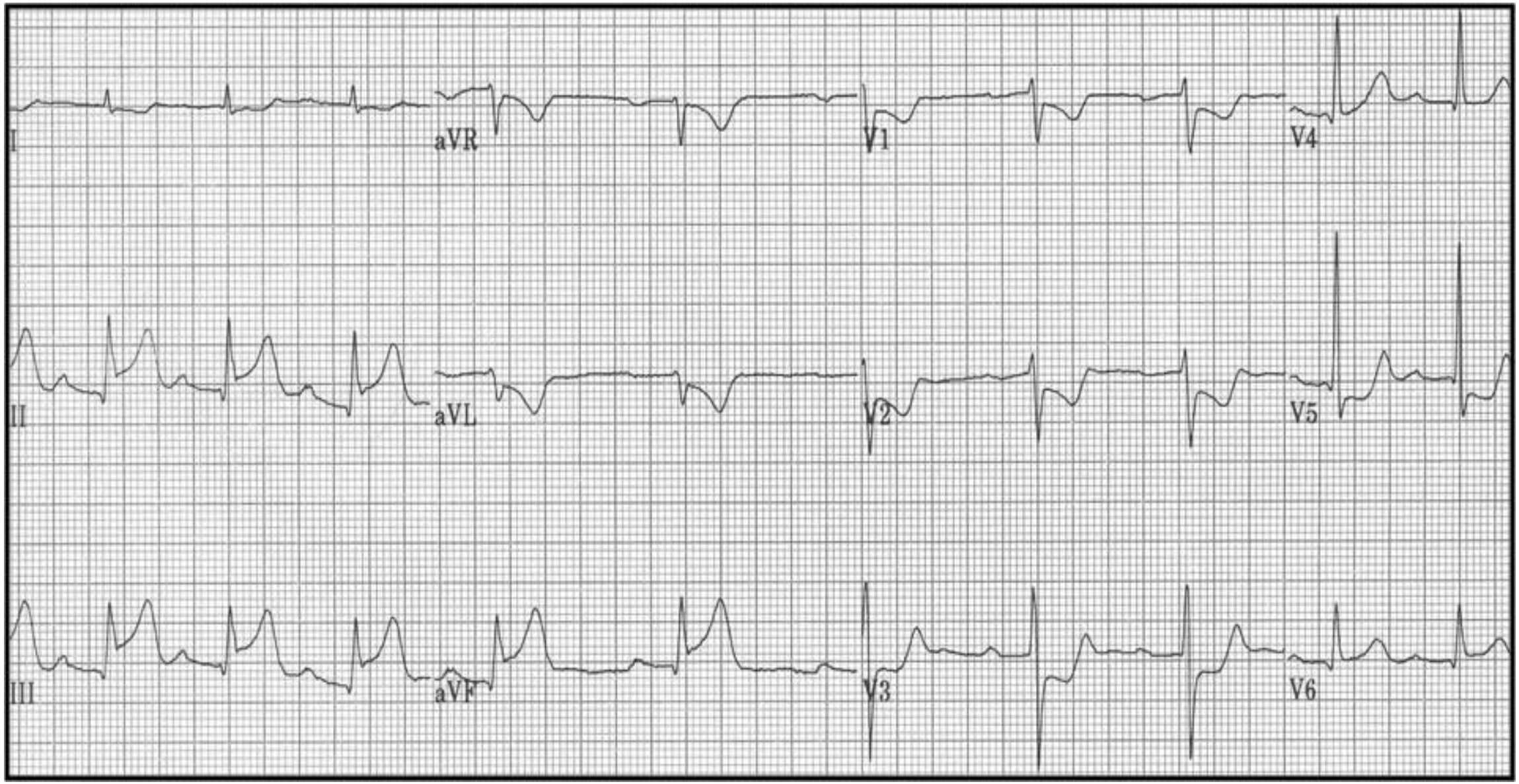

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The patient is sent emergently to the catheterization lab for percutaneous coronary intervention . . .

Patient 1: Elevated troponin due to inferior wall STEMI from coronary thrombosis

Patient 2

A 68-year-old man with a hx of tobacco abuse, HTN and type 2 diabetes presents to the ED with a two week history of worsening fatigue with exertion. He describes central chest pressure with radiation to his neck and left arm with nausea and shortness of breath. Walking up more than 10 steps at home precipates these symptoms. Resting about 10 minutes relieves these symptoms.

His appetite and weight have not changed recently. He denies fever, cough, changes in bowel movement or changes in urination.

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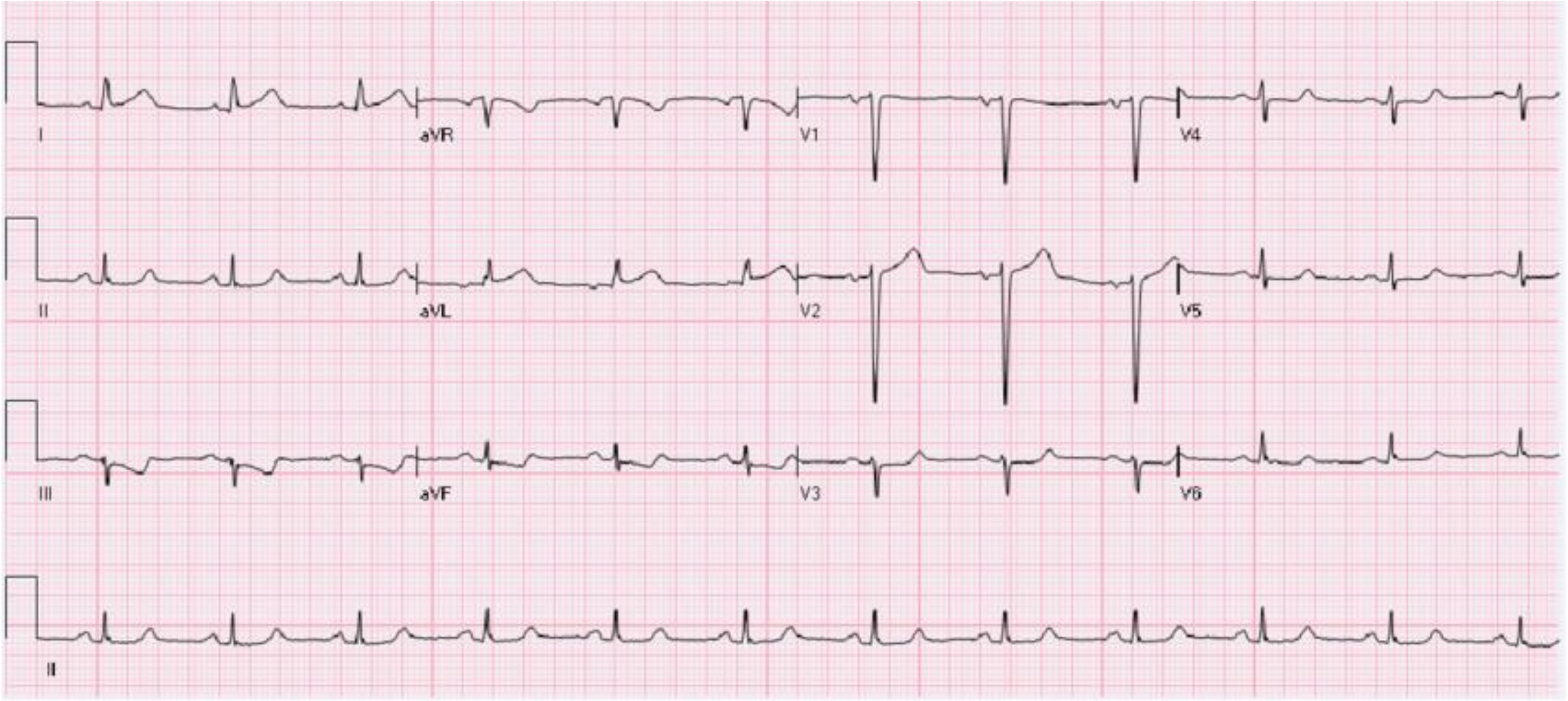

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Data	Score
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1 or 2	1
None	0

*bp > 140/90
*LDL > 159 mg/dl
*HDL < 40 mg/dl
*Diabetes mellitus
*Tobacco abuse
*Family history

TROPONIN

Data	Score
> 2X normal limit	2
1-2X normal limit	1
< normal limit	0

Heart score and risk for ACS:

0 to 3 = low

4 to 6 = moderate

≥ 7= high

The patient is referred to the cardiac catheterization lab. I left heart cath reveals multivessel disease and he is then referred for CABG.

Patient 2: Cardiac ischemia without infarction

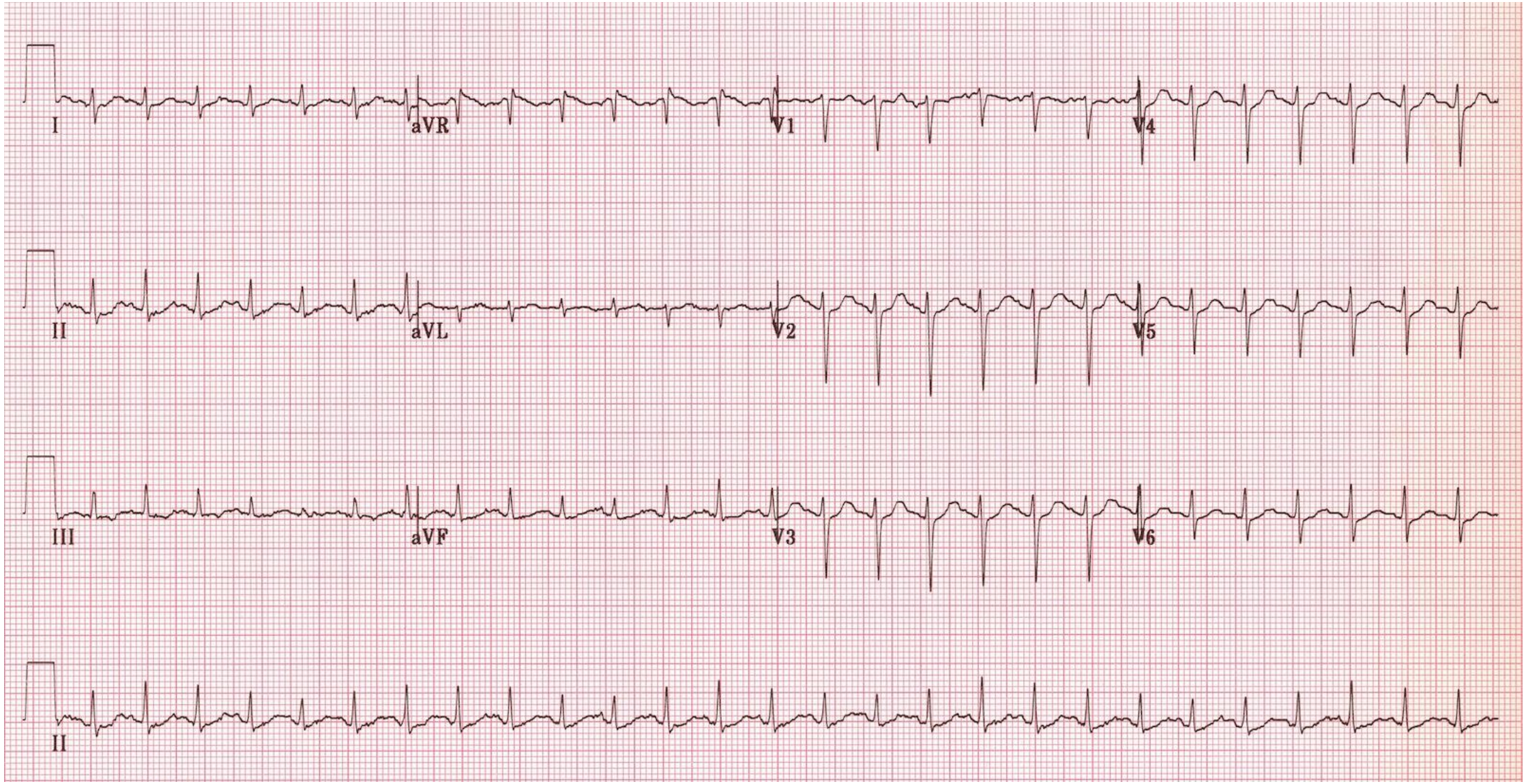
Patient 3

A 72-year-old man residing in a nursing home presents to the ED with delirium. He has a hx CAD with MI in 1998, CKD, and a cholecystectomy in 2001 and a splenectomy in the 1980's. He is arousable to sternal rub only and has rapid shallow breathing.

On examination, vitals: bp 110/70 p 150 RR 30 , and temp 100.8F with 02 sat 86% on RA. He has equal pupils with roving eye movements, a stiff neck, lungs with scattered rhonchi, tachycardia with a soft systolic murmur at the right upper sternal border and a nontender abdomen.

A CXR shows RLL and LUL infiltrates. A head CT is negative.

An EKG is performed . . .



Sinus tachycardia

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Persistent elevation of hs-cTnI without rise and fall

The patient is stabilized in the ICU and treated for septic shock.

Four days later, a 2-D echocardiogram shows normal LV systolic function with LVEF 55% and no significant valvular disease.

Two weeks later before discharge, the patient has an adenosine cardiolite with reveals normal LVEF and no evidence of ischemia or infarction.

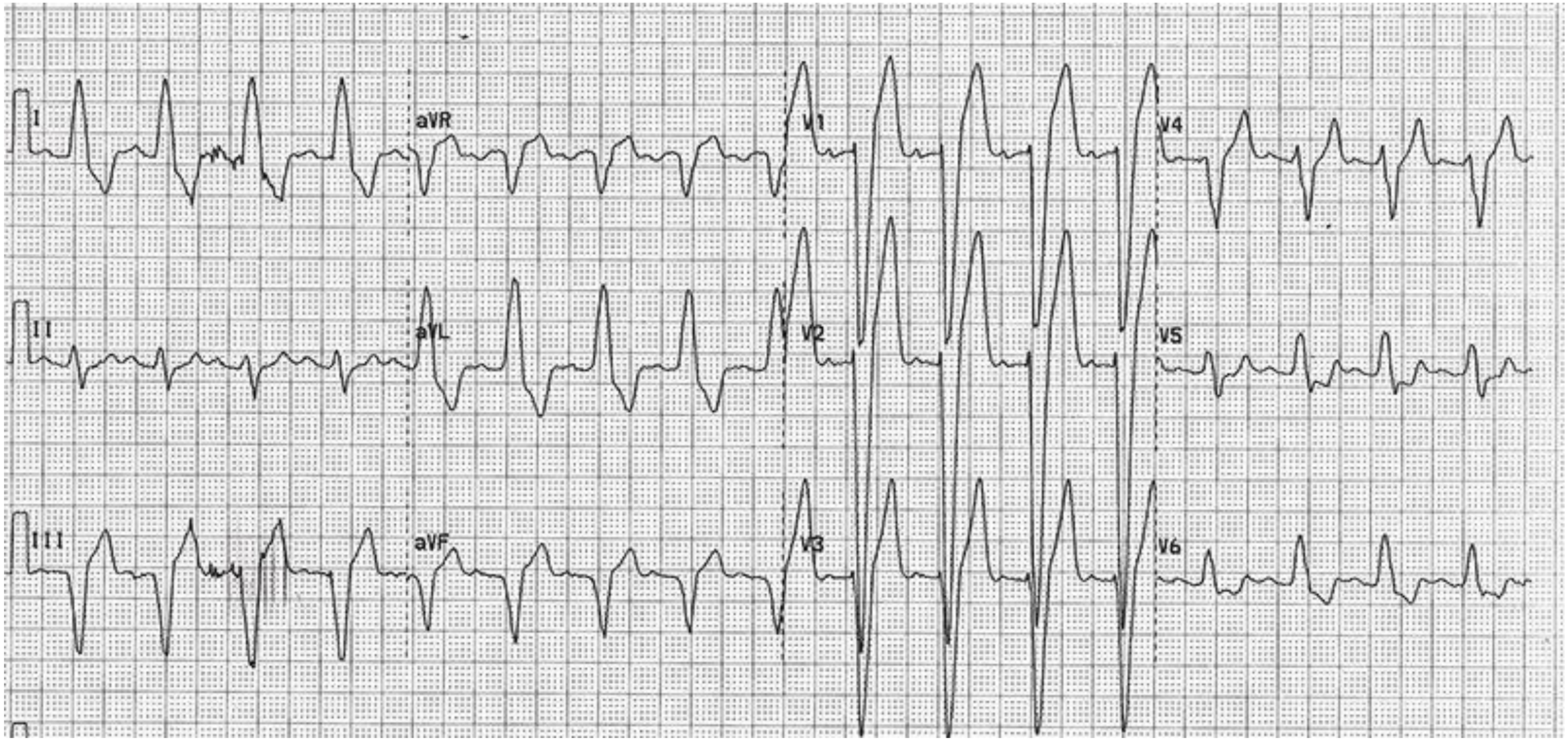
Patient 3: Elevated troponin from myocardial oxygen supply and demand mismatch from sepsis. No evidence of obstructive CAD.

Patient 4

A 65-year-old man with history of HTN, hyperlipidemia and HFrEF presents with shortness of breath for the last 24 hours.

On exam: vital signs: blood pressure 100/60 p 96 RR 28 afebrile. O2 sat 90% on RA
car: r/r/r with a 3/6 holosystolic murmur at the left sternal border radiating to the left axilla. JVP to angle of mandible
lungs: bilateral rales.
Extremities: 2+ edema bilaterally

An EKG is performed . . .



Sinus tachycardia, left bundle-branch block, LVH

Serial high-sensitivity cardiac troponin I (hs-cTI) are as follows:

Time from presentation	**0**	**3 hours**	**6 hours**
hs-cTnI (ng/L) Normal < 14 ng/L	25	60	48

Elevated hs-cTn with significant rise and fall . . .

He is treated for acute on chronic HFrEF.

He undergoes left heart catheterization which demonstrates non-obstructive CAD for which medical therapy is recommended. Left ventriculogram confirms global hypokinesis with LVEF 35%. In addition, he has severe mitral regurgitation.

He then referred for surgical mitral valve repair and cardiac pacer (ie. resynchronization therapy and AICD placement.

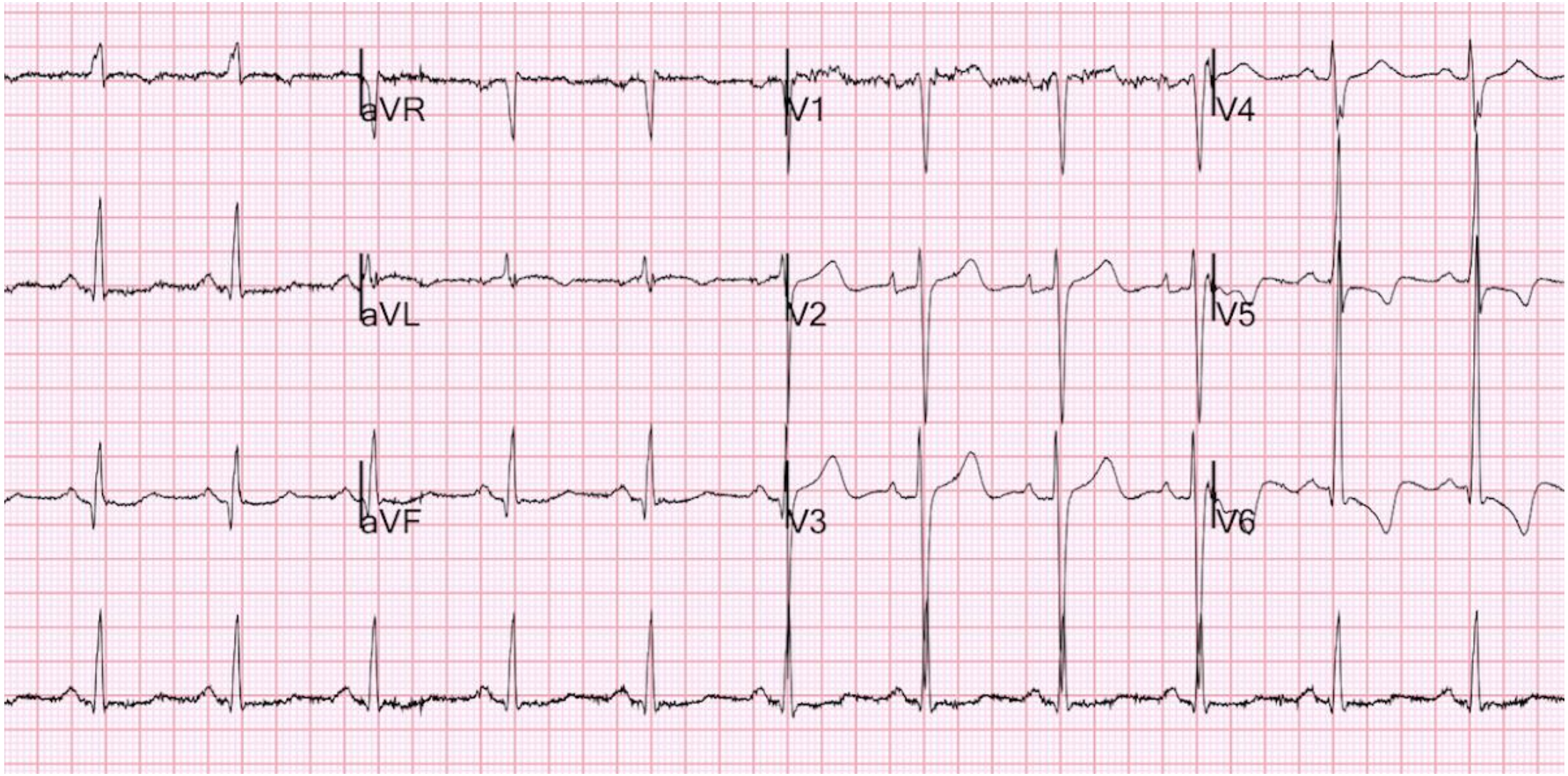
Patient 4: Elevated troponin from acute CHF in the setting of known HFrEF. No evidence of obstructive CAD.

Patient 5

You are called to see a 61-year-old man with ESRD on hemodialysis three times per week to evaluate him for new onset shortness of breath. He describes forgetting to take his bp medication yesterday. He denies chest discomfort but is slightly nauseated.

On exam, vitals: bp 190/110 p 100 RR 22 temp 97.1F
JVP 9 cm at 45 degrees (upper limit of normal)
Car: r/r/r with S4
Lungs: bibasilar rales
Abd: soft, nontender
Extr: L arm with AV fistula with strong thrill and no warmth or erythema Legs with 1+ edema B/L and dp pulses trace

An EKG is performed . . .



Sinus rhythm, non- specific ST and T-wave changes

After one hour of dialysis, his shortness of breath resolves . . .

Serial high-sensitivity cardiac troponin I (hs-cTI) are as follows:

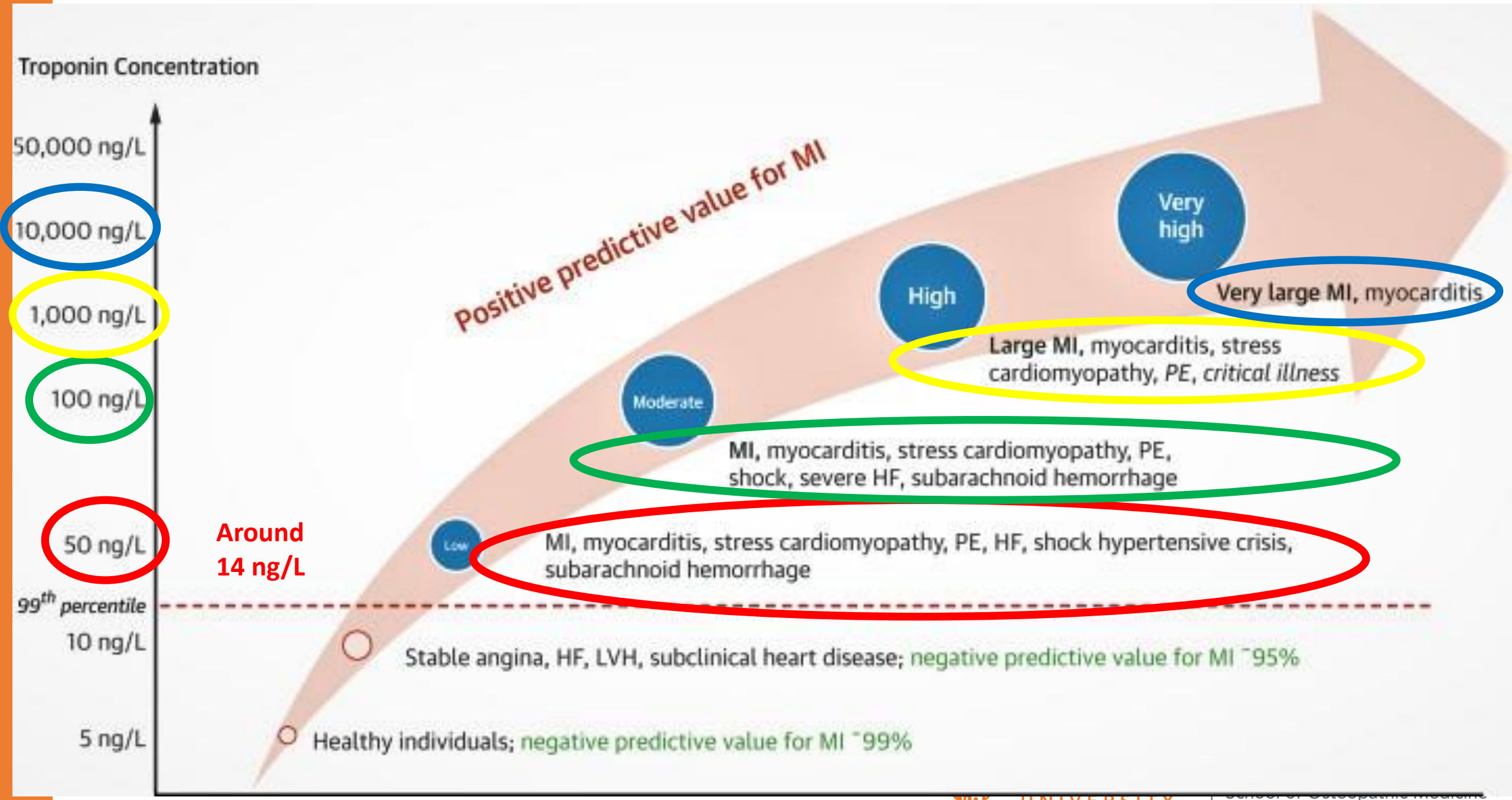
Time from presentation	**0**	**3 hours**	**6 hours**
hs-cTnI (ng/L) Normal < 14 ng/L	43	39	45

Chronically elevated hs-cTn without significant rise or fall . . .

Patient 5: Elevated troponin from CKD. Transient SOB from fluid overload. Consider work up for cardiac ischemia if not done in the last two years.

Clinical Utility of Conventional and High-Sensitivity Cardiac Troponin Levels: Summary and Conclusions

- Troponins are regulatory proteins, unique to heart which control interaction of actin and myosin via calcium
- Troponin levels begin to rise two to three hours after myocardial injury
- **High-sensitivity** cardiac troponin levels (either hs-cTnI or hs-cTnT) detect levels several magnitudes lower than then original cTn
- Upper limit of normal of hs-cTn is the 99% percentile which in most populations is around 14 ng/L


Troponin Concentration
50,000 ng/L
10,000 ng/L
1,000 ng/L
100 ng/L
50 ng/L
99th percentile
10 ng/L
5 ng/L
Positive predictive value for MI
Very high
High
Moderate
Low
Very large MI, myocarditis
Large MI, myocarditis, stress cardiomyopathy, PE, critical illness
MI, myocarditis, stress cardiomyopathy, PE, shock, severe HF, subarachnoid hemorrhage
MI, myocarditis, stress cardiomyopathy, PE, HF, shock hypertensive crisis, subarachnoid hemorrhage
Around 14 ng/L
Stable angina, HF, LVH, subclinical heart disease; negative predictive value for MI ~95%
Healthy individuals; negative predictive value for MI ~99%

Clinical Utility of Conventional and High-Sensitivity Cardiac Troponin Levels: Summary and Conclusions

- Biochemical definition of AMI with serial troponins:
  - Level > 99th percentile for the troponin assay being used

  AND
  - A rise and fall in serial troponins must be confirmed

- If troponins remain < 99th percentile then troponin rise of 50% to 80% from baseline *may also suggest* AMI

Clinical Utility of Conventional and High-Sensitivity Cardiac Troponin Levels: Summary and Conclusions

- Troponin levels are necessary *but not sufficient* to diagnose AMI or ACS
- There is a wide ddx of elevated troponin levels . . .including rise and fall of hs-cTn in acute CHF and chronic elevations in ESRD

Clinical Utility of Conventional and High-Sensitivity Cardiac Troponin Levels: Summary and Conclusions

hs-cTn levels *alone* cannot distinguish among these diagnoses . . .

- **Injury related to primary myocardial ischemia**
- **Injury related to myocardial oxygen supply/demand imbalance**
- **Injury not related to myocardial ischemia**
- **Multifactorial or indeterminate myocardial injury**

Clinical Utility of Conventional and High-Sensitivity Cardiac Troponin Levels: Summary and Conclusions

Diagnosis of coronary artery disease without infarction.

VS

Diagnosis of acute myocardial infarction.

Can be two different questions . . . .

In other words, a patient can have coronary artery disease and cardiac ischemia but NO myocardial infarction and therefore have unremarkable troponin levels.

Clinical Utility of Conventional and High-Sensitivity Cardiac Troponin Levels: Summary and Conclusions

The most important tool you have to diagnose coronary artery disease and/or acute myocardial infarction is

You

Clinical Utility of Conventional and High-Sensitivity Cardiac Troponin Levels: Summary and Conclusions

- Clinically estimate the pretest probability of CAD
- Use tools like the HEART score to aid clinical decision-making
- Always think of a ddx for your patient presentation and troponin results:
  - AMI/ACS
  - Sepsis
  - PE
  - Myocarditis
- Use information from other sources to make final diagnoses
  - Echocardiogram
  - Stress testing
  - Left heart catheterization

Questions?

Thank you!