AAPA Bootcamp 2023
Hospital Diagnostics Workshop

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Disclosure

- I/we have no relevant relationships with ineligible companies to disclose within the past 24 months.

Our team!




Overarching objectives for the whole session

- At the conclusion of the session participants should be able to:
  1. Independently and systematically review ECGs that will be encountered in hospital practice
  2. Independently and systematically review chest radiographs that will be encountered in hospital practice
  3. Interpret common acid base, liver test and antibody testing encountered in hospital practice

Let’s meet our patient

Theresa S. Wift

- 67-year-old morbidly obese female with a past medical history of cirrhosis due to chronic hepatitis C virus infection, obstructive sleep apnea without CPAP use, hypertension and hyperlipidemia presenting with shortness of breath, cough, chest pain, and abdominal pain. You are called to come evaluate and admit the patient to the hospital.
- As you walk into the room, you see T. S. Wift sitting up on the bed with a continuous albuterol nebulizer running and multiple colleagues walking out including the lab tech, ECG tech, and x-ray tech, all saying they just got the labs, ECG, and chest x-ray the ED ordered.

Chest X-Ray Interpretation

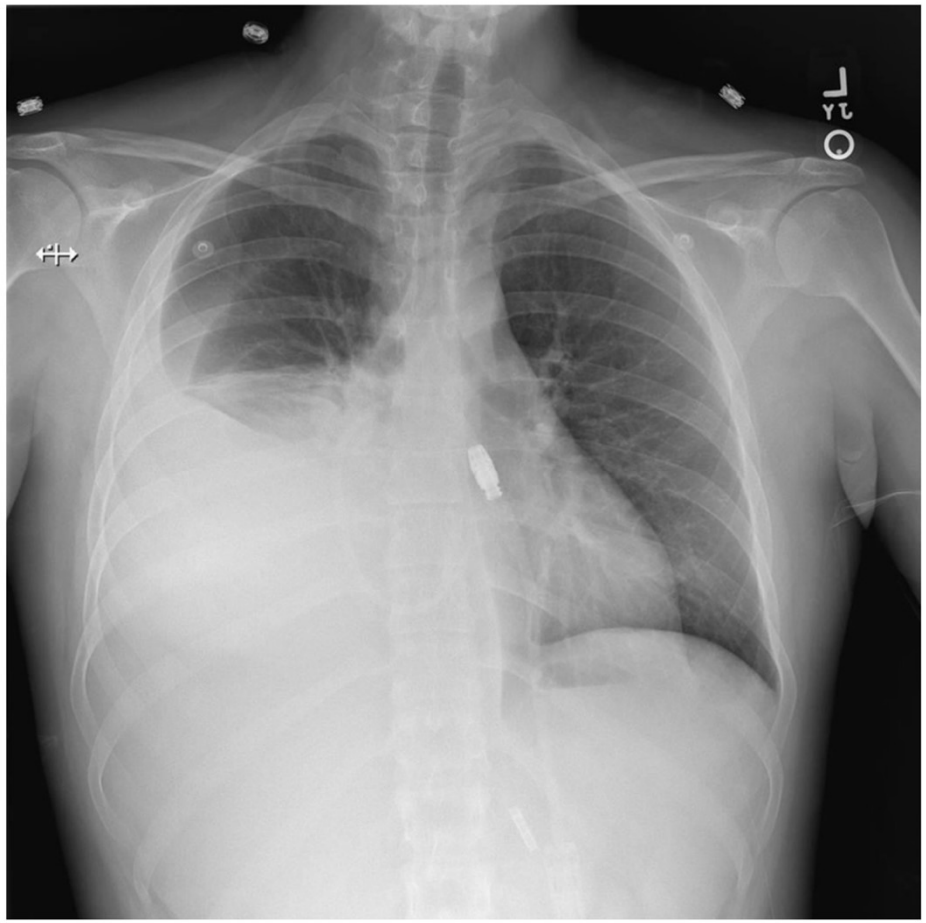
Frannie Lorenzi, MMS, PA-C
Assistant Professor
University of Colorado Anschutz School of Medicine

Objectives

- Identify techniques and common terminology used for chest radiographs
- Learn a standardized sequence of chest x-ray interpretation
- Recognize common chest radiograph abnormalities

Bring it back to our case…

- Theresa S. Wift is a 67-year-old morbidly obese female with a past medical history of cirrhosis due to chronic hepatitis C virus infection, obstructive sleep apnea without CPAP use, hypertension and hyperlipidemia presenting with shortness of breath, cough, chest pain, and abdominal pain. You are called to come evaluate and admit the patient to the hospital.
- We’ve working up some of her labs, now the chest Xray returns
- As you are reviewing the Chest Xray, you are getting more information from T. S. Wift. She states, "Call it what you want... I can't breathe, I'm breathless... I'm feel so cold that I can't even stay warm in my cardigan!"

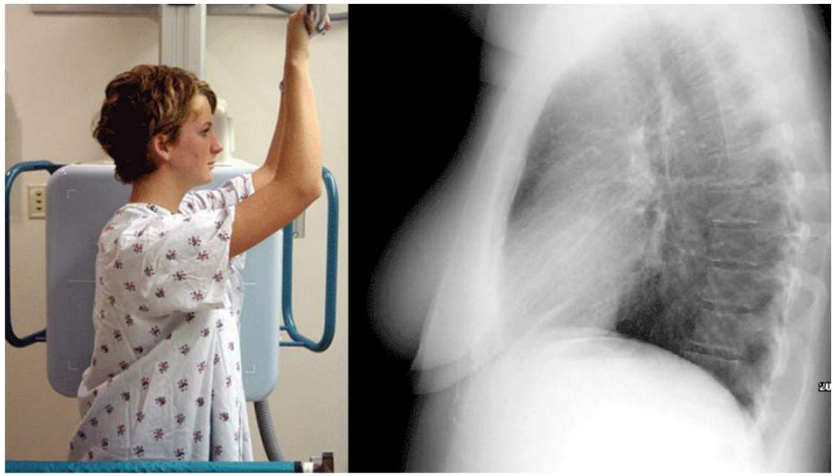


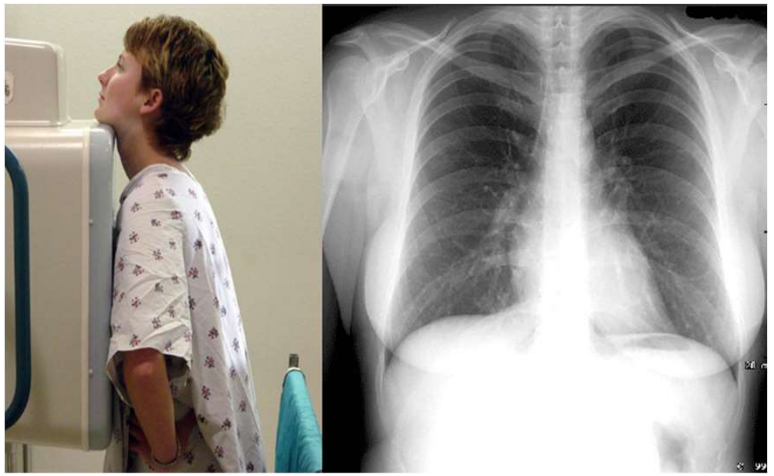
Source: Allaham H, Hudhud D, Salzer W. [redacted]: a peritoneal dialysis dilemma. BMJ Case Rep. 2018.

CXR Techniques: Posterior-Anterior & Lateral

This is a “Standard XRay”

1. Beams pass from from posterior to anterior
2. Heart size is better estimated because it is closer to the detector

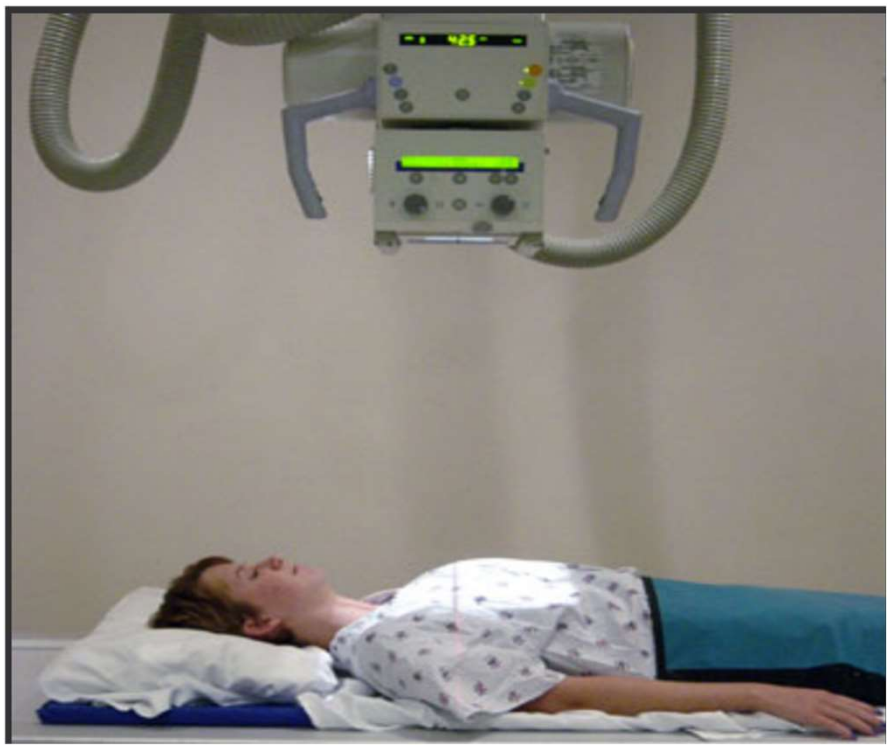




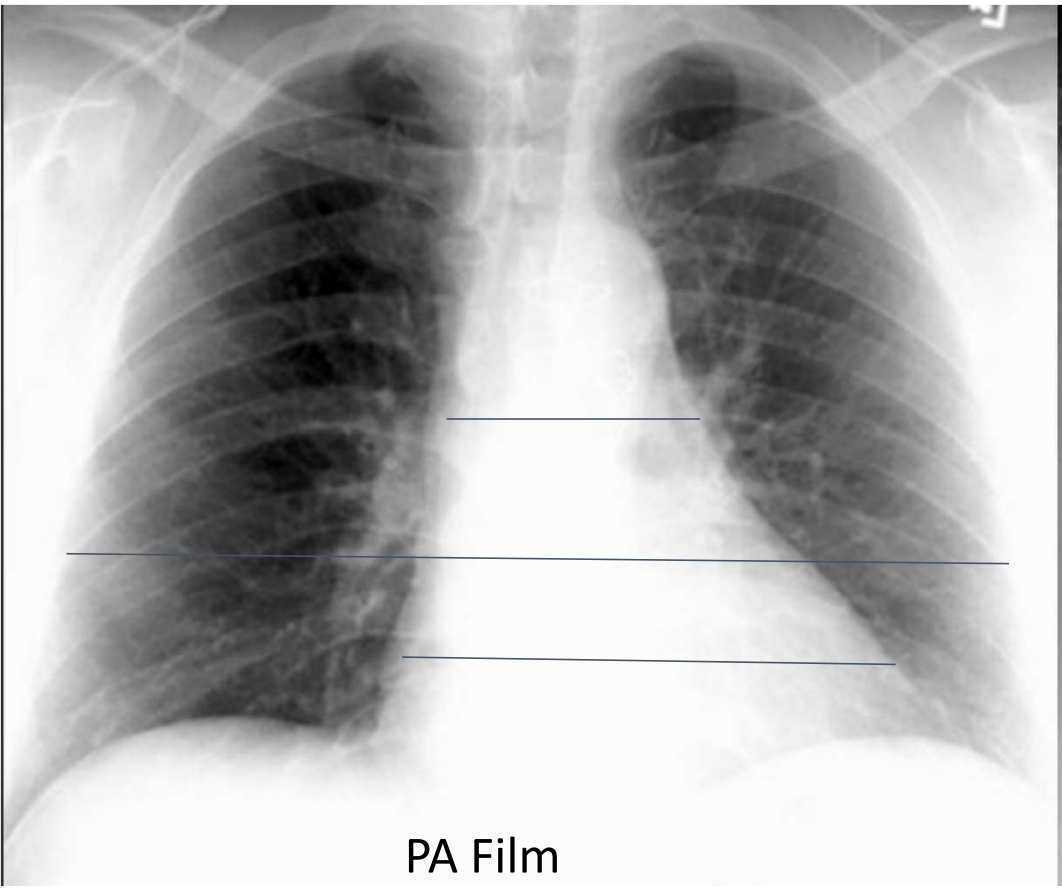
Techniques: Anterior-Posterior

This is what a lot of our patients will get when they are laying in bed or too ill to go down for CXR.

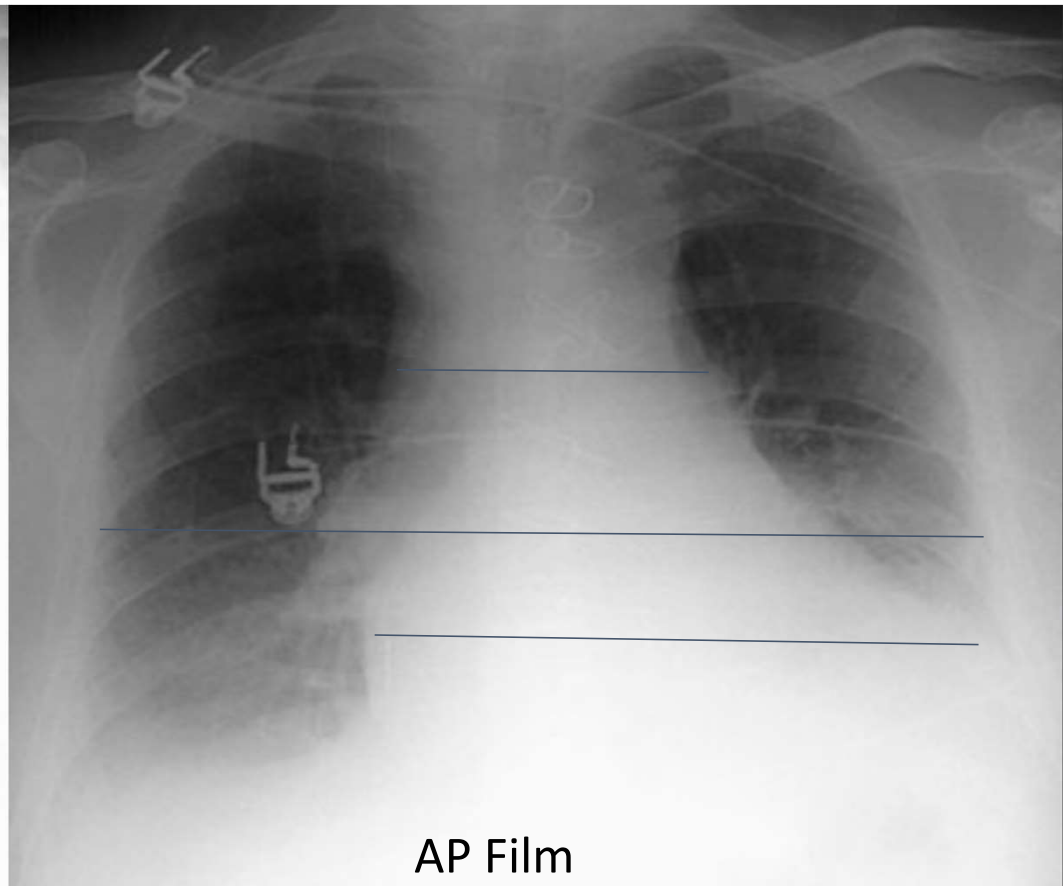
1. Beams pass anterior to posterior
2. Patients are closer to the beam and heart is further away from the detector so heart is magnified



Heart size difference

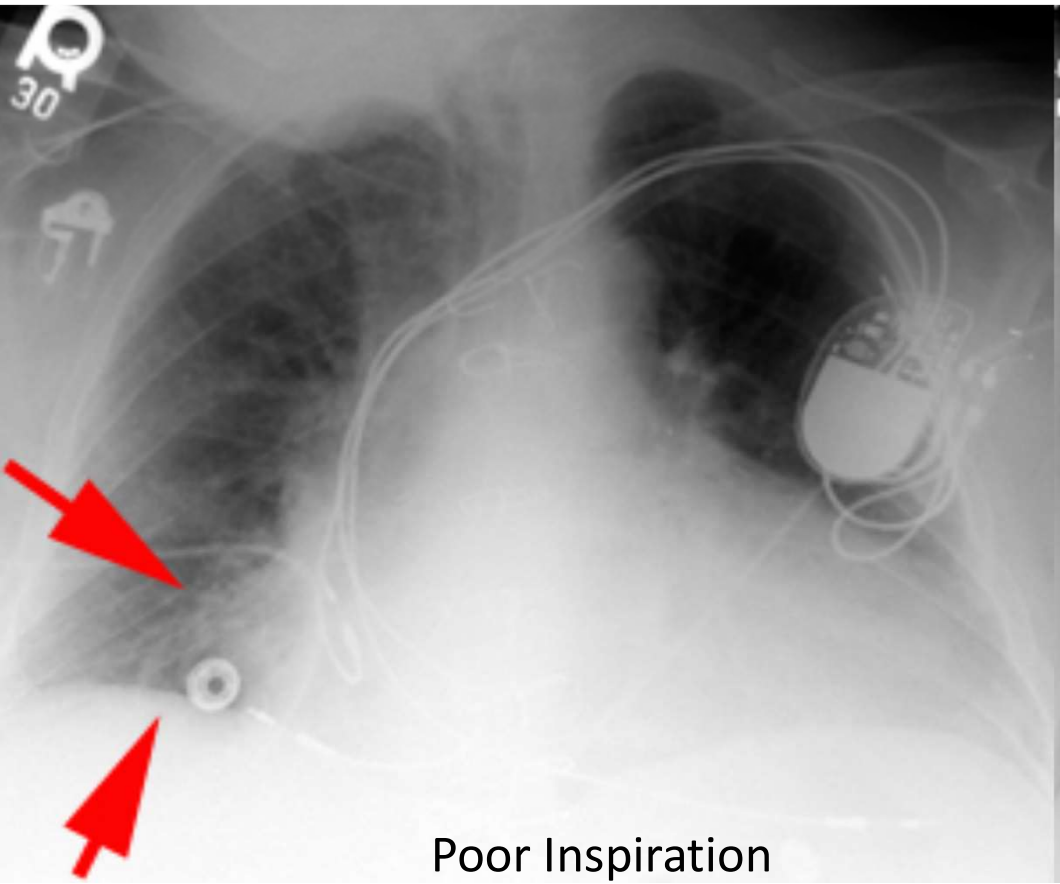


PA Film

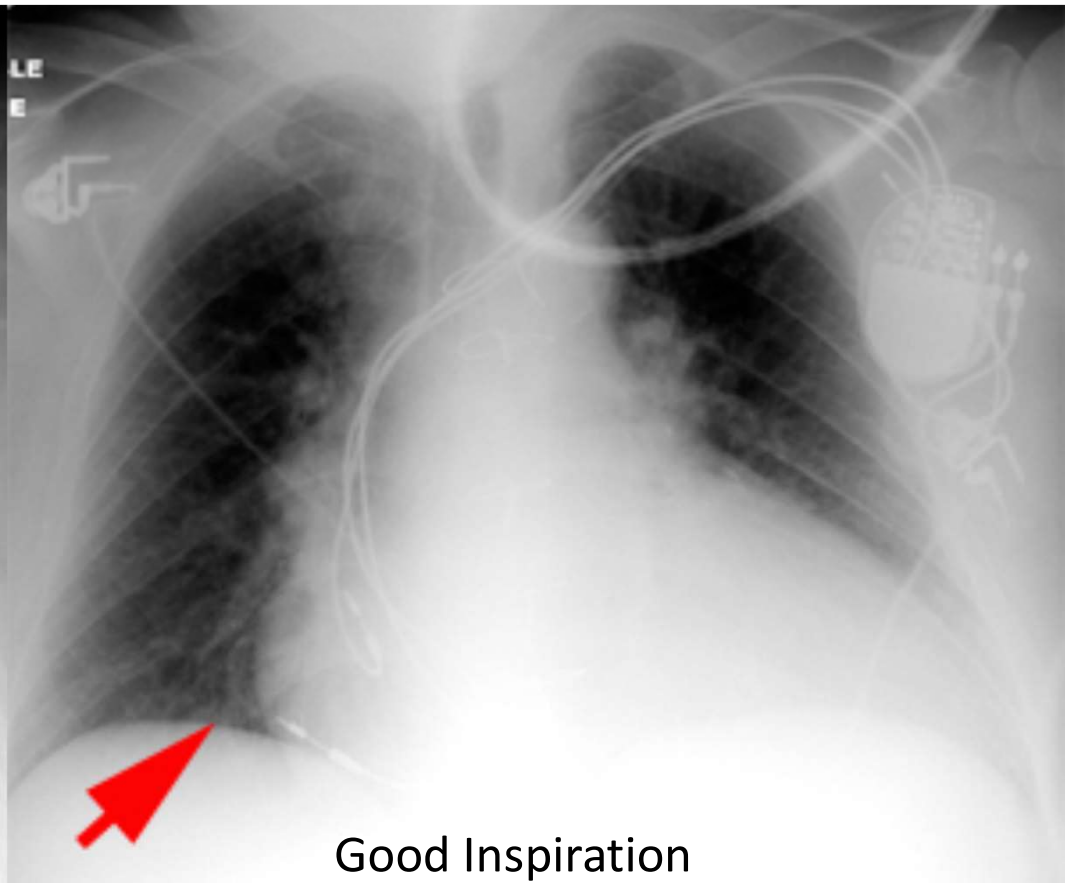


AP Film

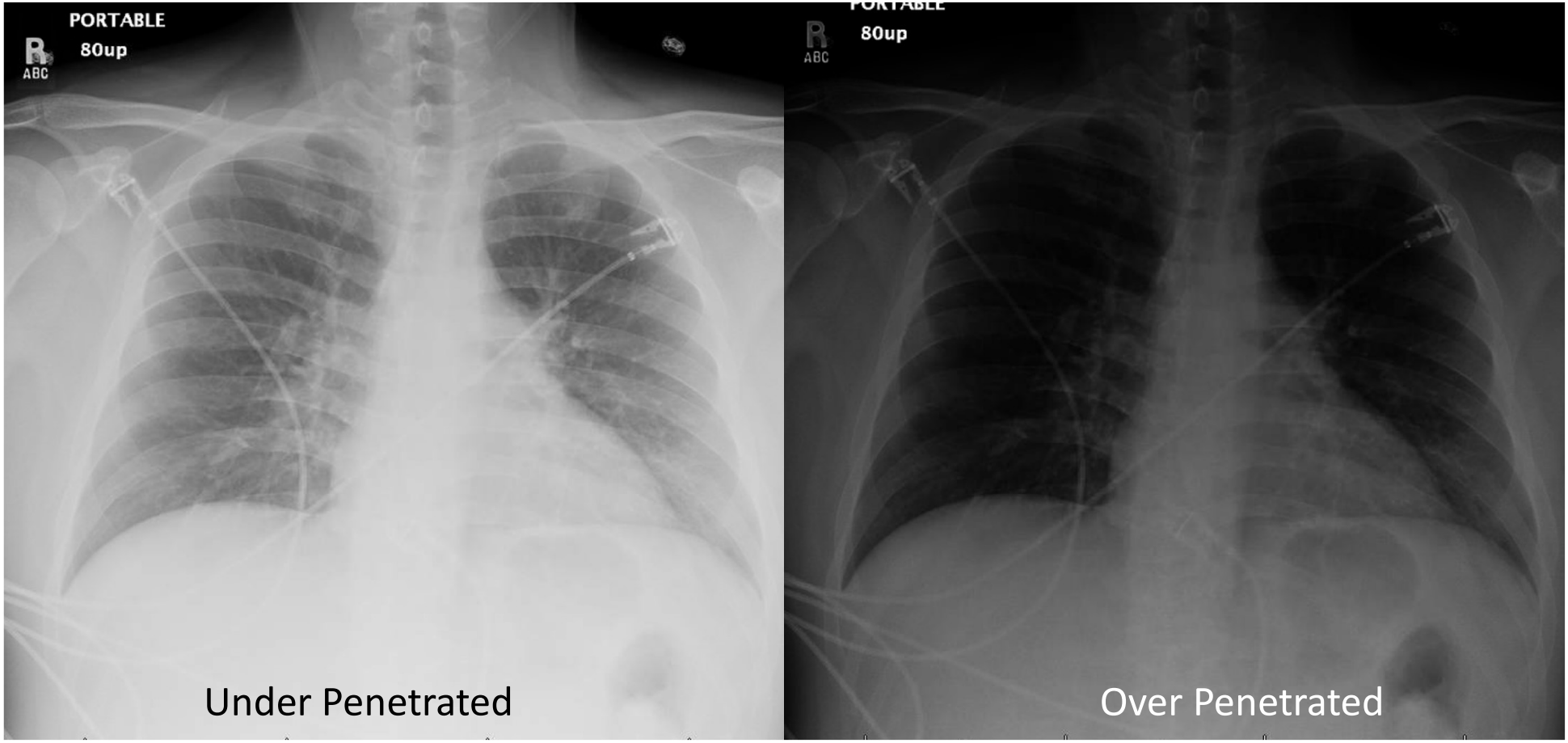
Inspiration Differences



Poor Inspiration



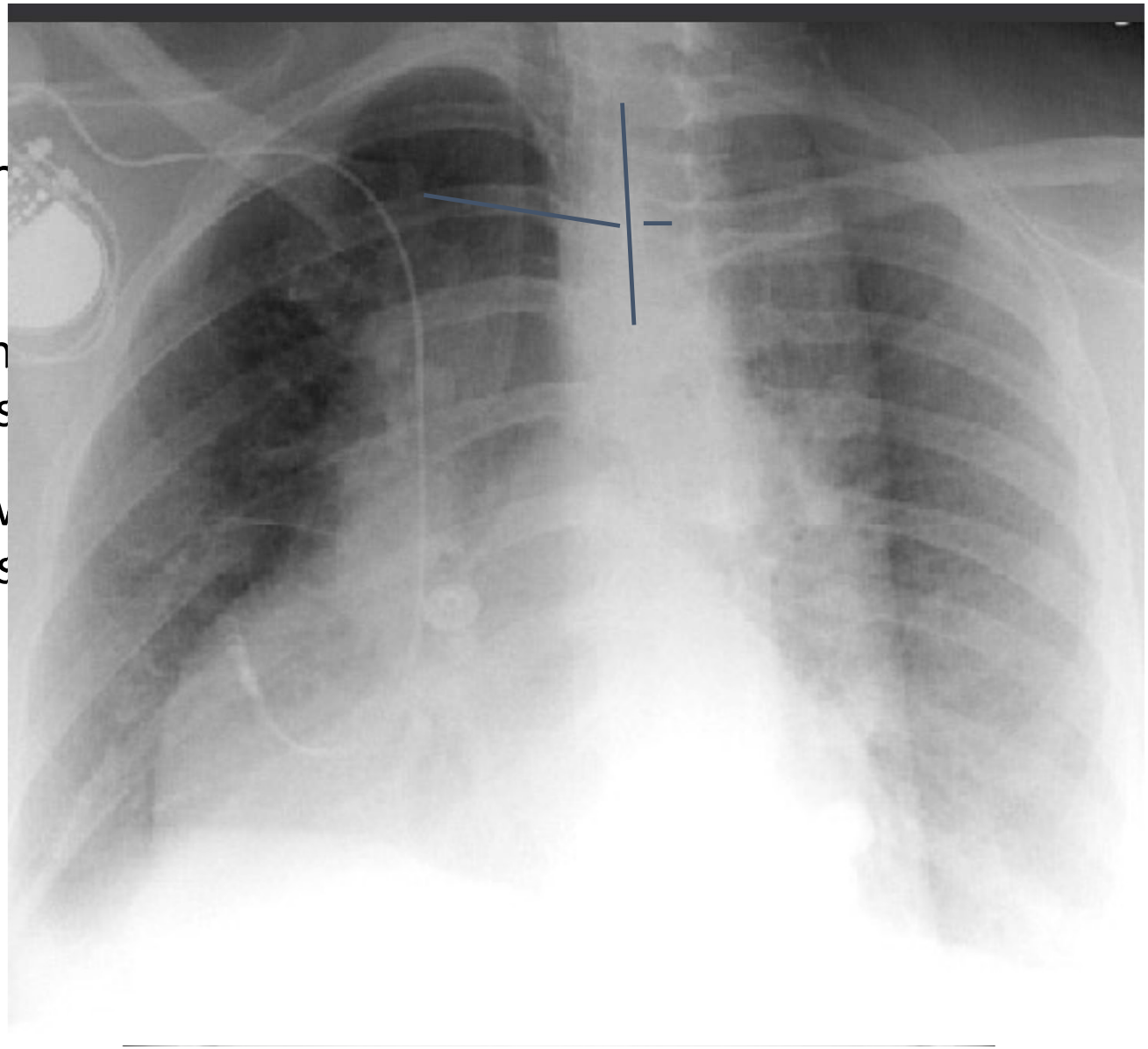
Good Inspiration

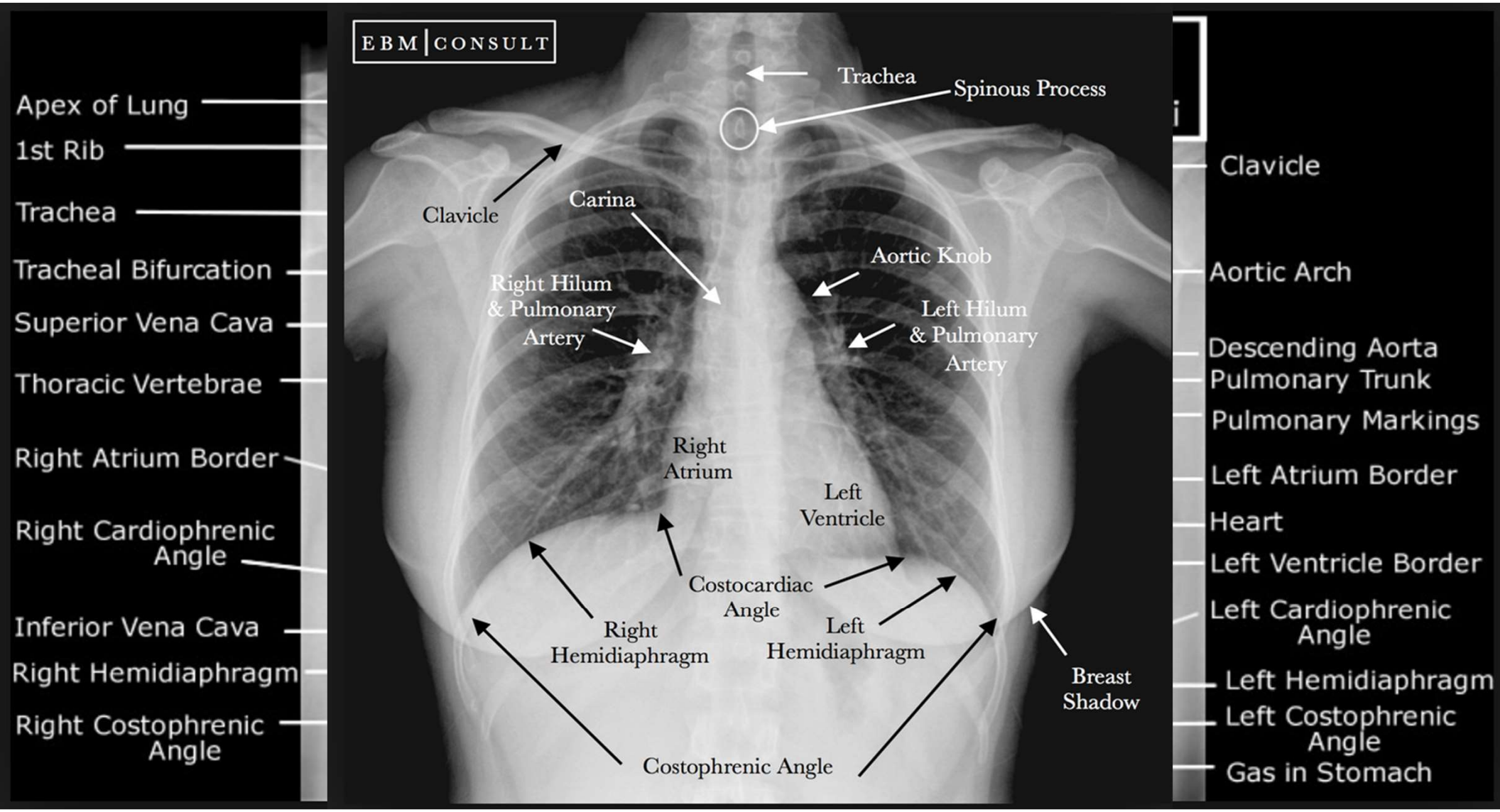

PORTABLE
80up
ABC
Under Penetrated
80up
ABC
Over Penetrated

Rotation

--The spin
--Clavicles

Why do w
-It's




EBM | CONSULT
Apex of Lung
1st Rib
Trachea
Tracheal Bifurcation
Superior Vena Cava
Thoracic Vertebrae
Right Atrium Border
Right Cardiophrenic Angle
Inferior Vena Cava
Right Hemidiaphragm
Right Costophrenic Angle
Trachea
Spinous Process
Clavicle
Carina
Aortic Knob
Right Hilum & Pulmonary Artery
Left Hilum & Pulmonary Artery
Right Atrium
Left Ventricle
Costocardiac Angle
Right Hemidiaphragm
Left Hemidiaphragm
Breast Shadow
Costophrenic Angle
Clavicle
Aortic Arch
Descending Aorta
Pulmonary Trunk
Pulmonary Markings
Left Atrium Border
Heart
Left Ventricle Border
Left Cardiophrenic Angle
Left Hemidiaphragm
Left Costophrenic Angle
Gas in Stomach

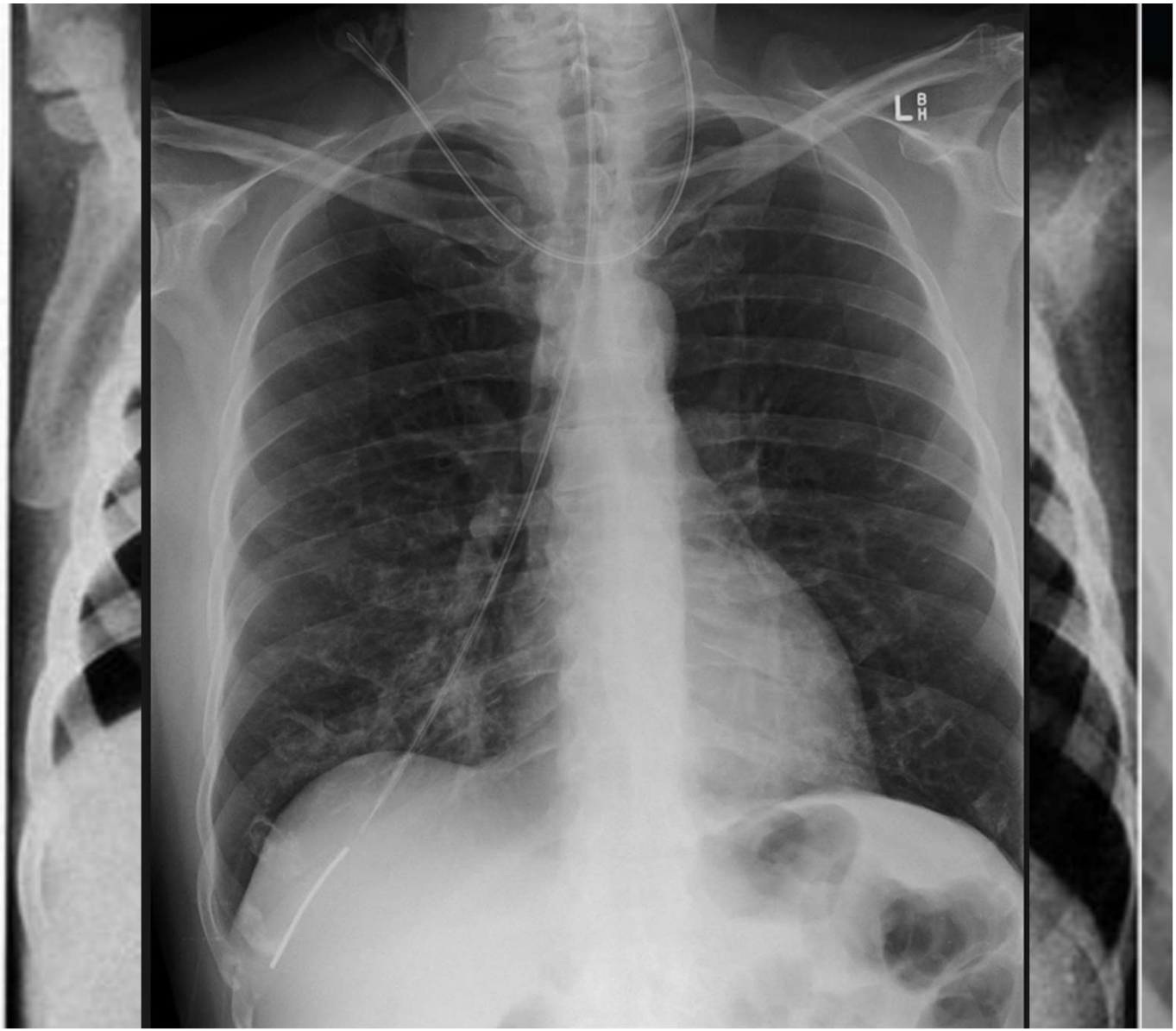
Anatomy & Interpretation!

M- Man made

A - Asses Quality, Airway
B - Bones, Soft Tissue
C - Cardiac
D - Diaphragms
F - (Lung) Field & Fissures
G - Great Vessels, Gastric Bubble
H - Hilum
I - Impression

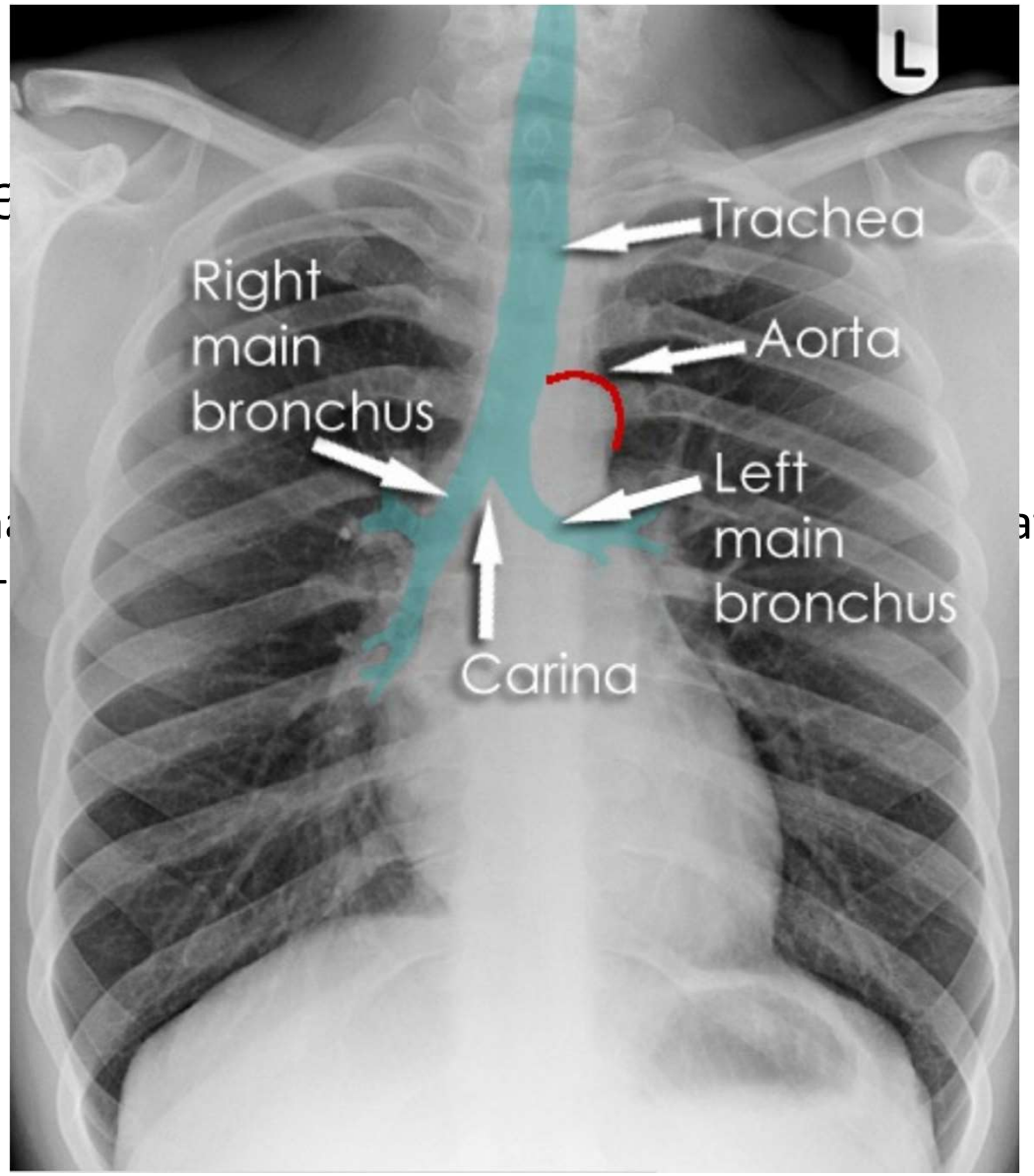
M is fo

Any forei



A is for Asse

1. Assess Ima ation
2. Airways --



B is for Bones

Bones

1. Ribs
2. Clavicle
3. Sternum
4. Vertebra
5. Shoulder Joint, Scapula

Soft Tissue

1. Breast Shadows
2. Skin Folds
3. Muscles

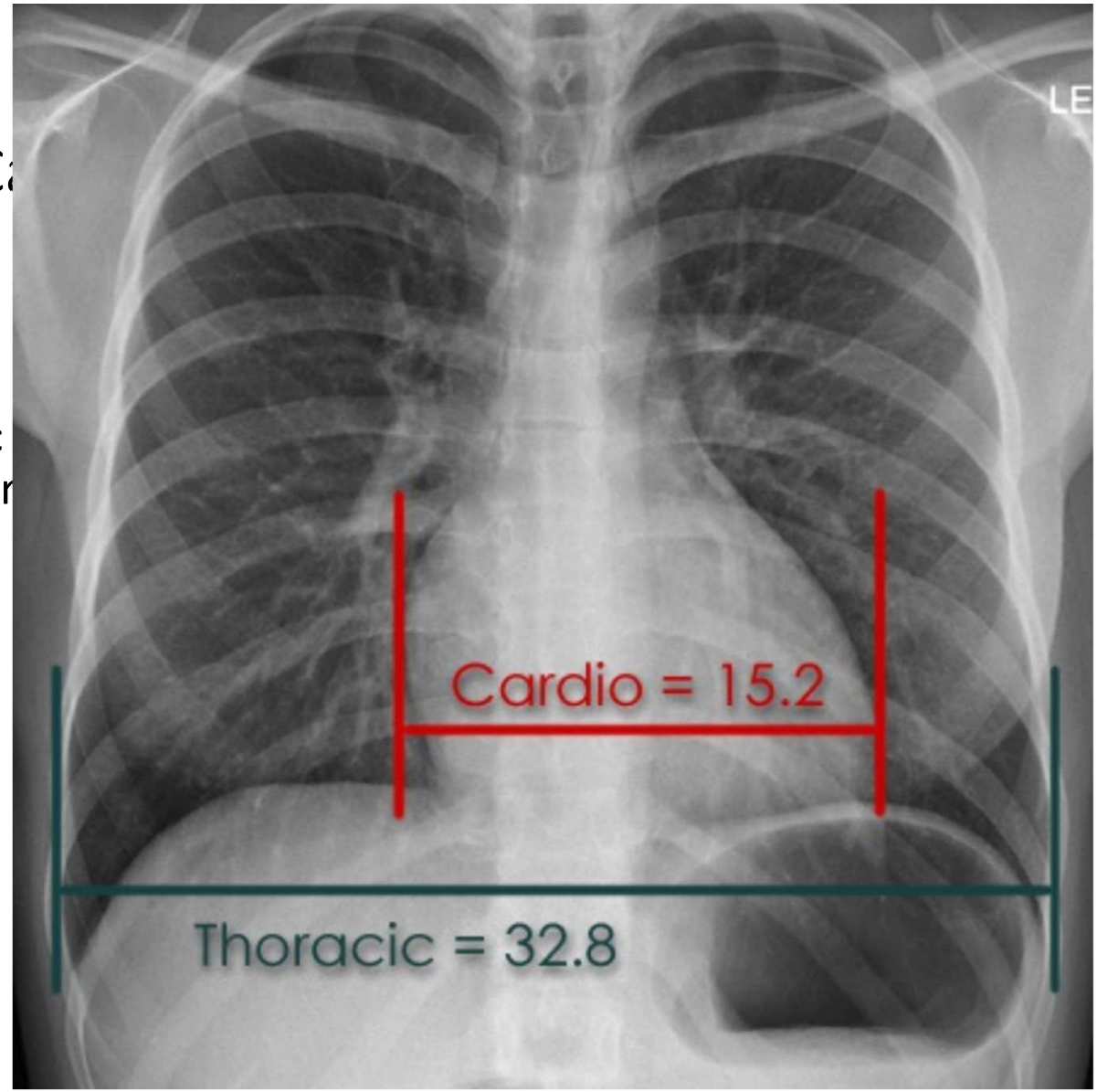
Check For:

1. Symmetry
2. Deformity
3. Fracture
4. Mass
5. Lytic Lesion

C is for C

Assess:

1. Cardiac
2. Contour



D is for Diaphragm

Each hemidiaphragm should be a rounded structure with a crisp white edge contrasted against the adjacent dark lung

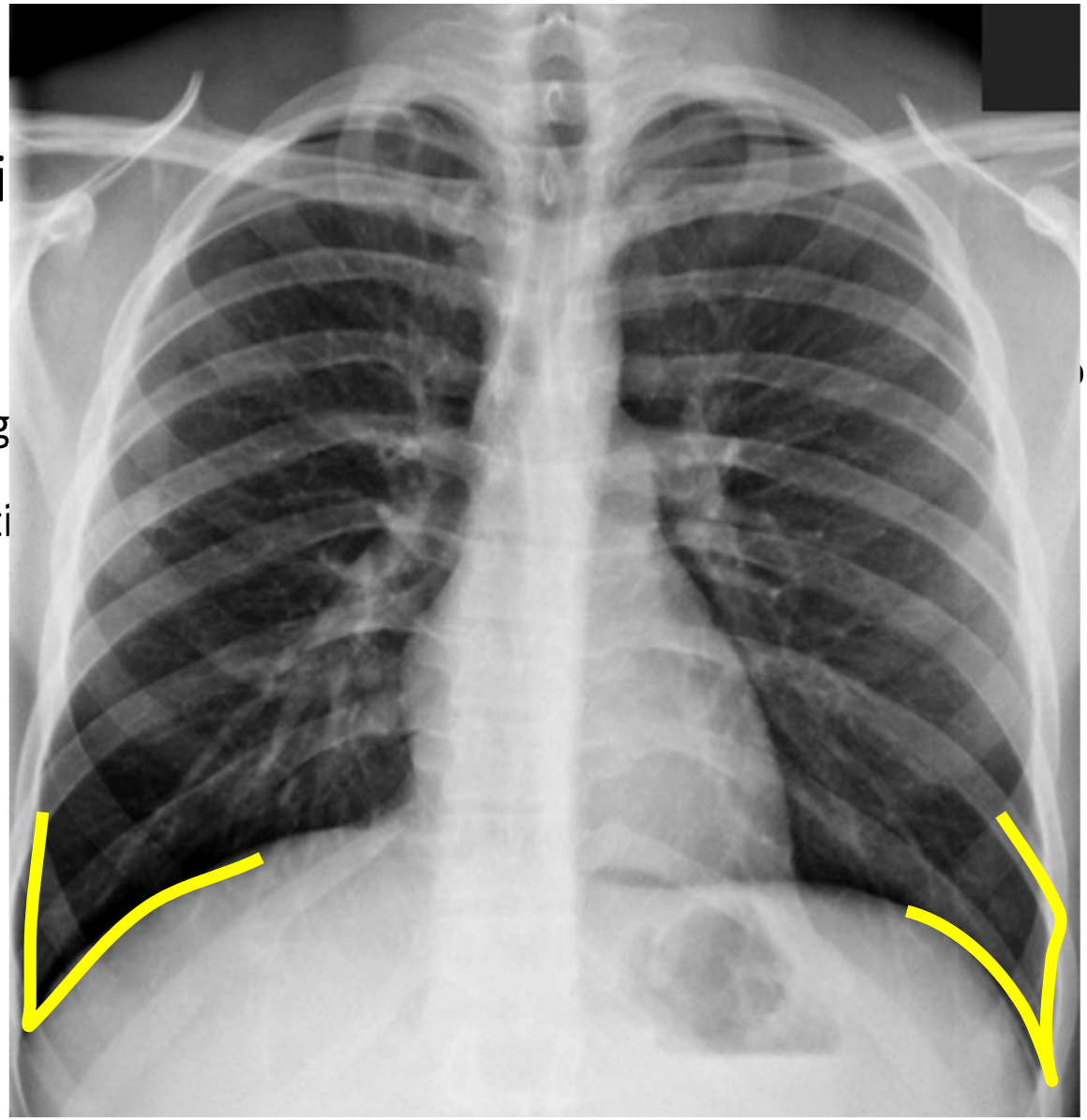
Right Diaphragm higher than the Left

Gastric Bubble usually under the left side

D is for Di

Costophrenic ome of each hemidiaphrag

-Blunti

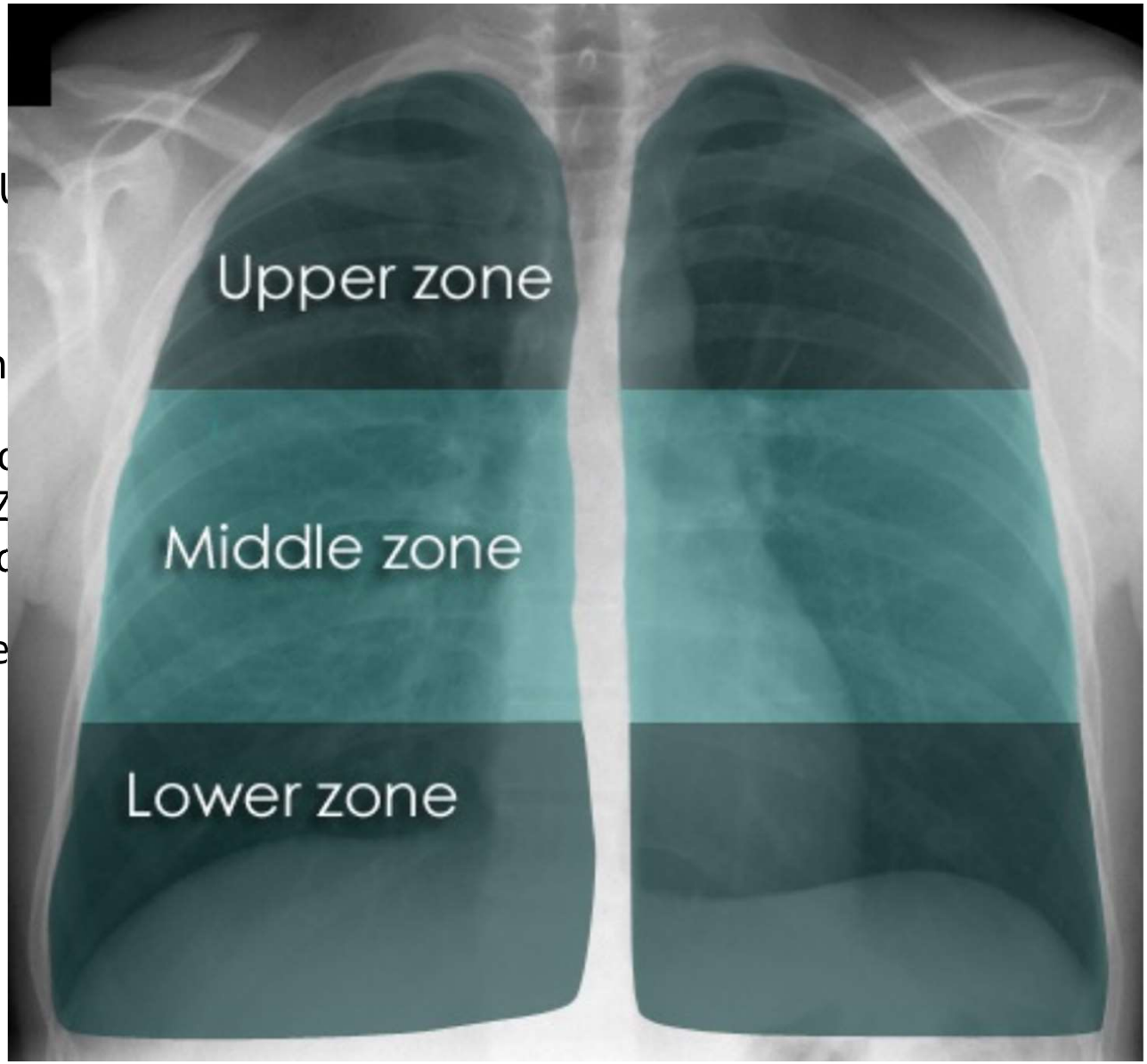


F is for (Lu

Let’s break th

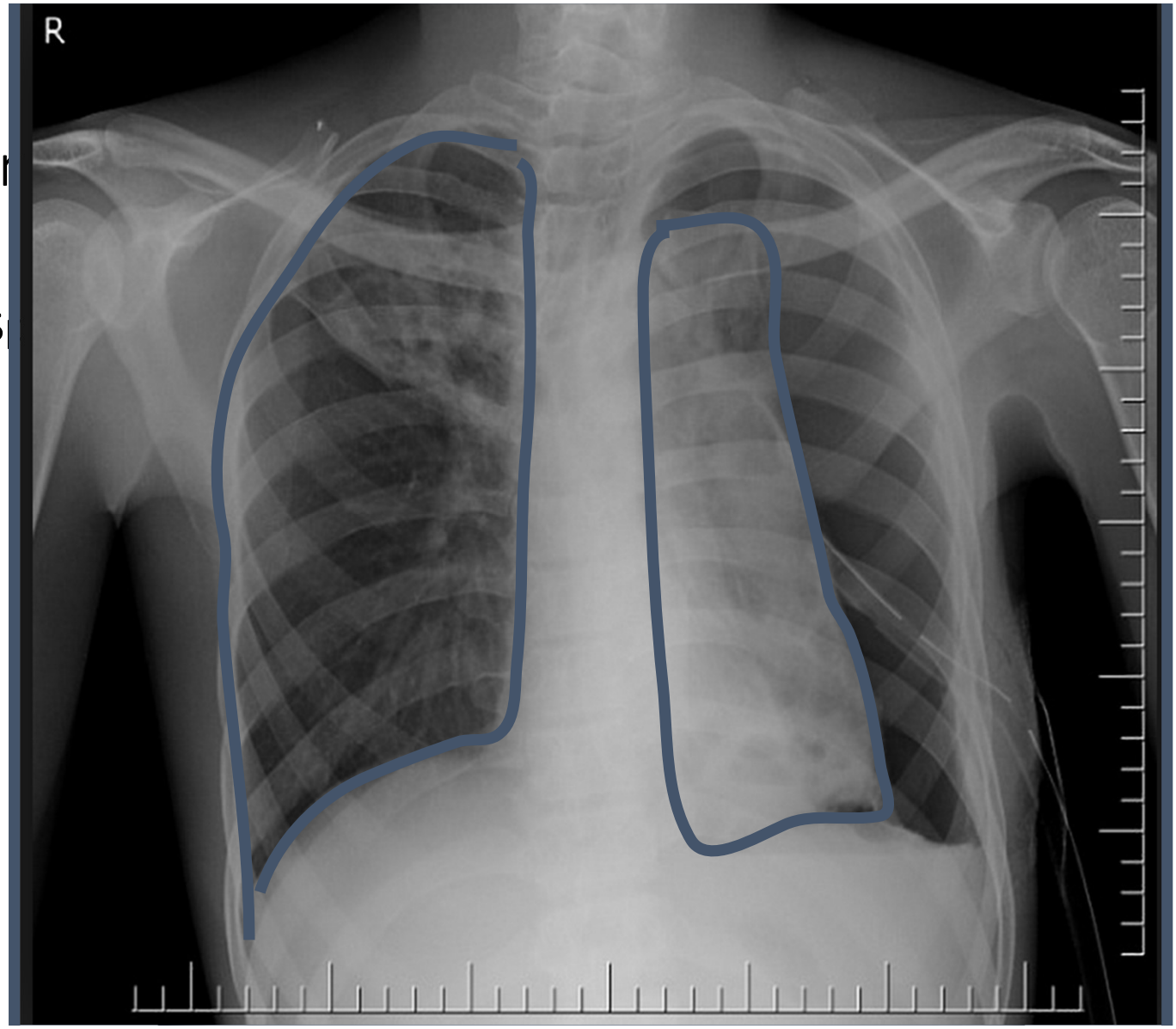
1. Upper Zo
2. Middle Z
3. Lower Zo

Let’s you take

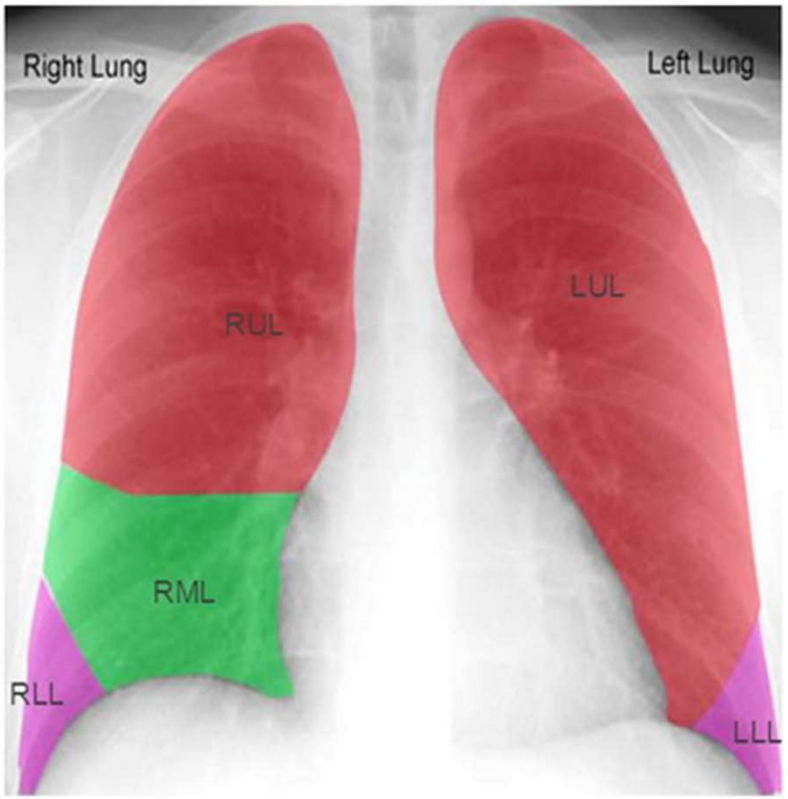


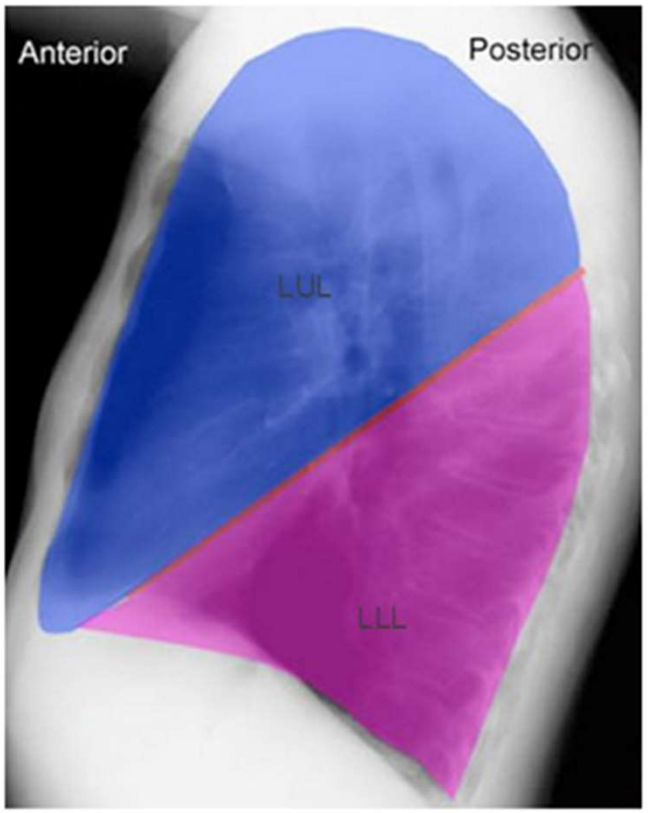
F is for

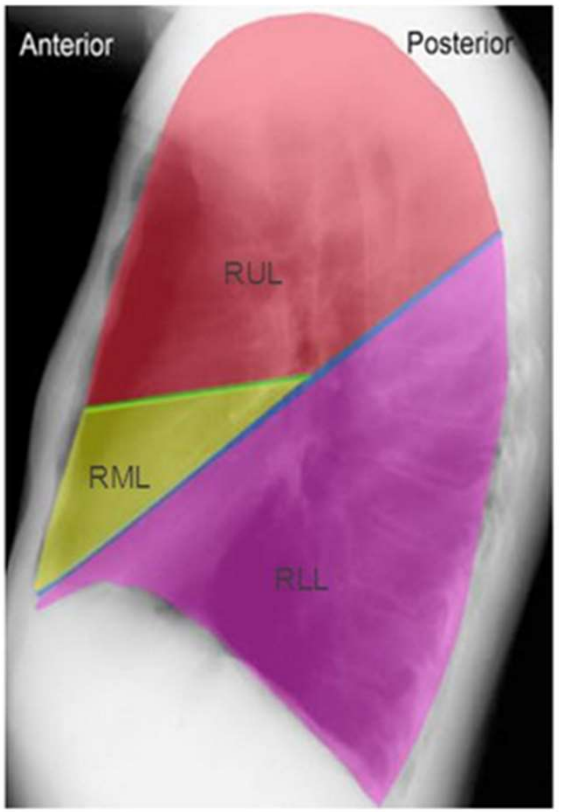
Pleural S

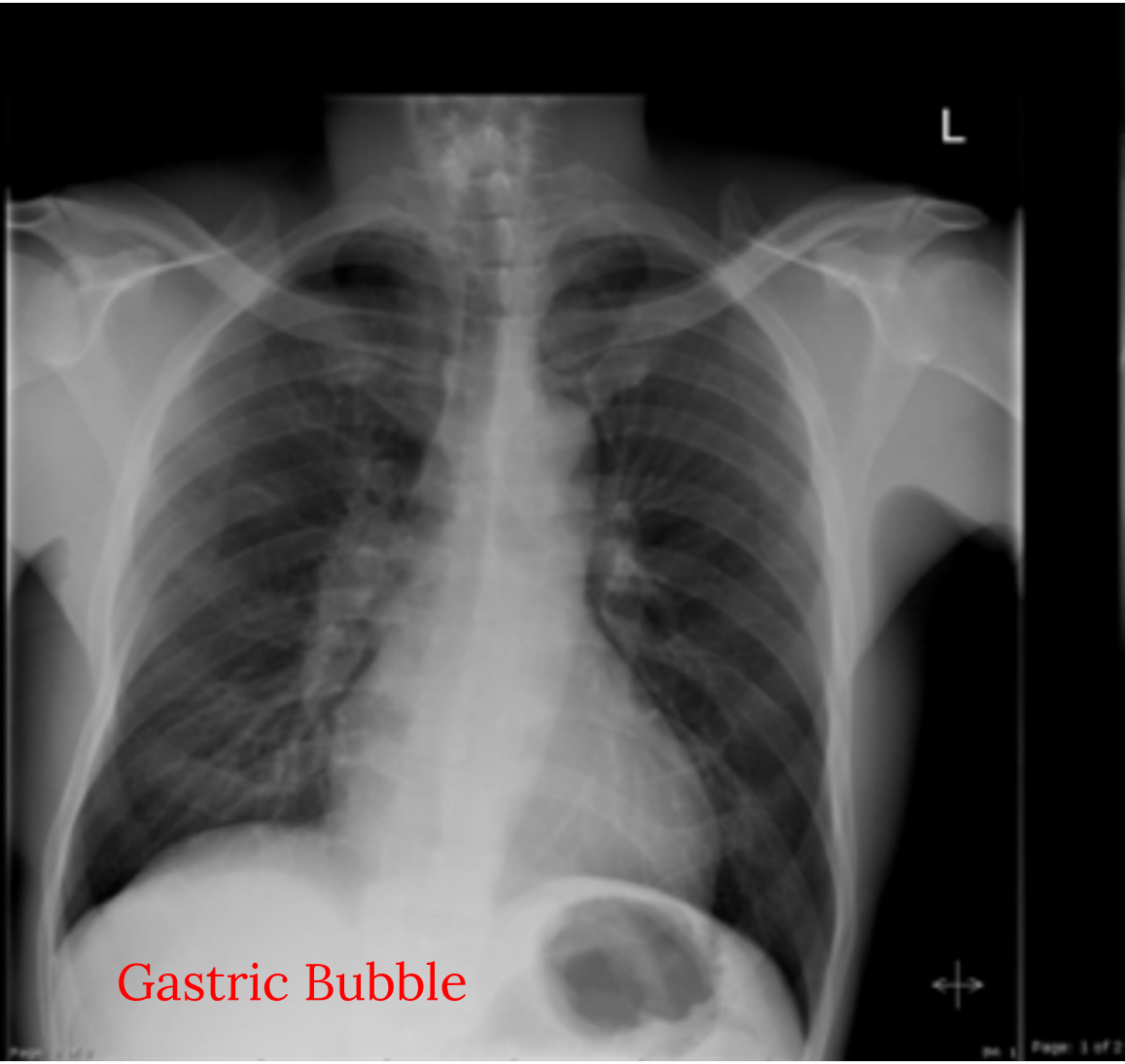


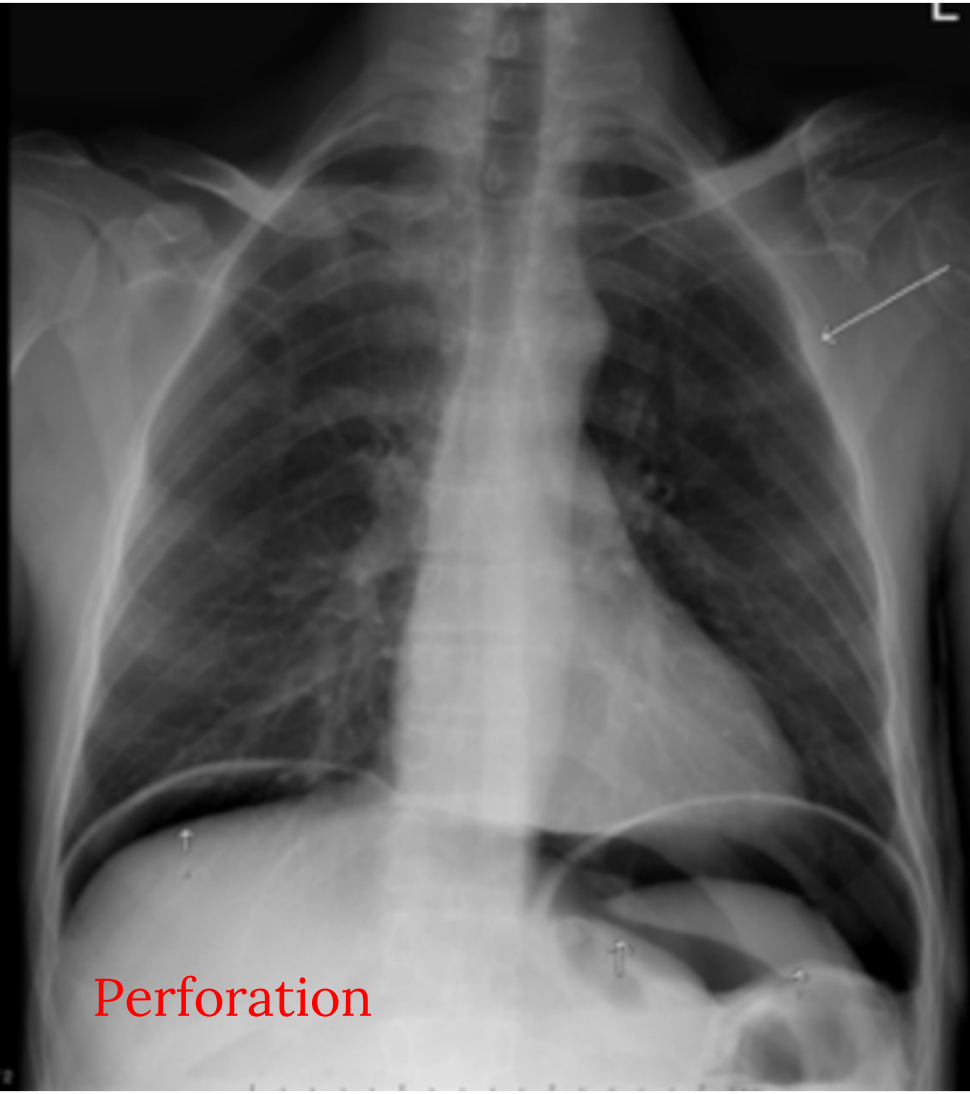
F is for (Lung) Fields, Fissures





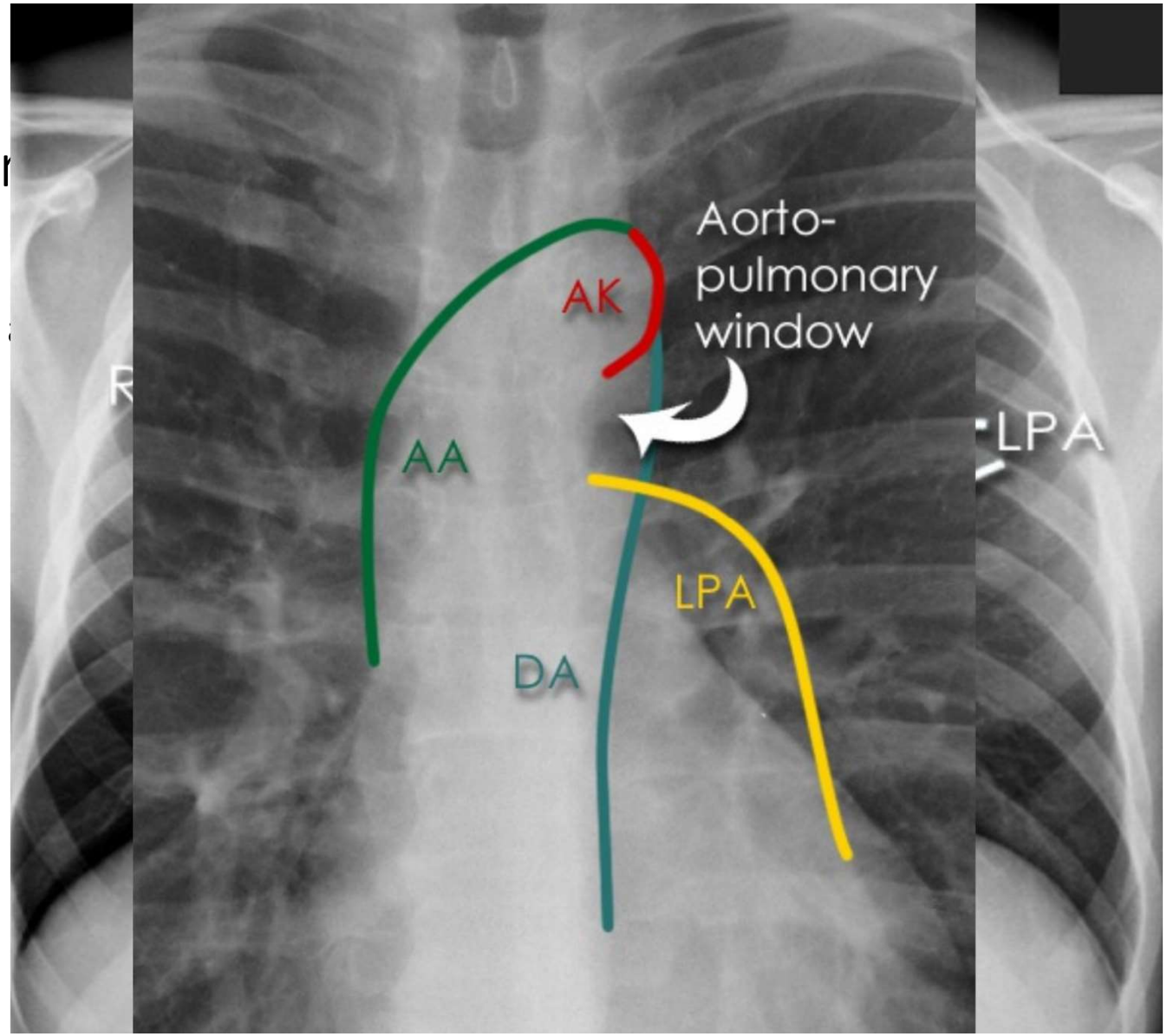



L
Gastric Bubble


Perforation

H is for

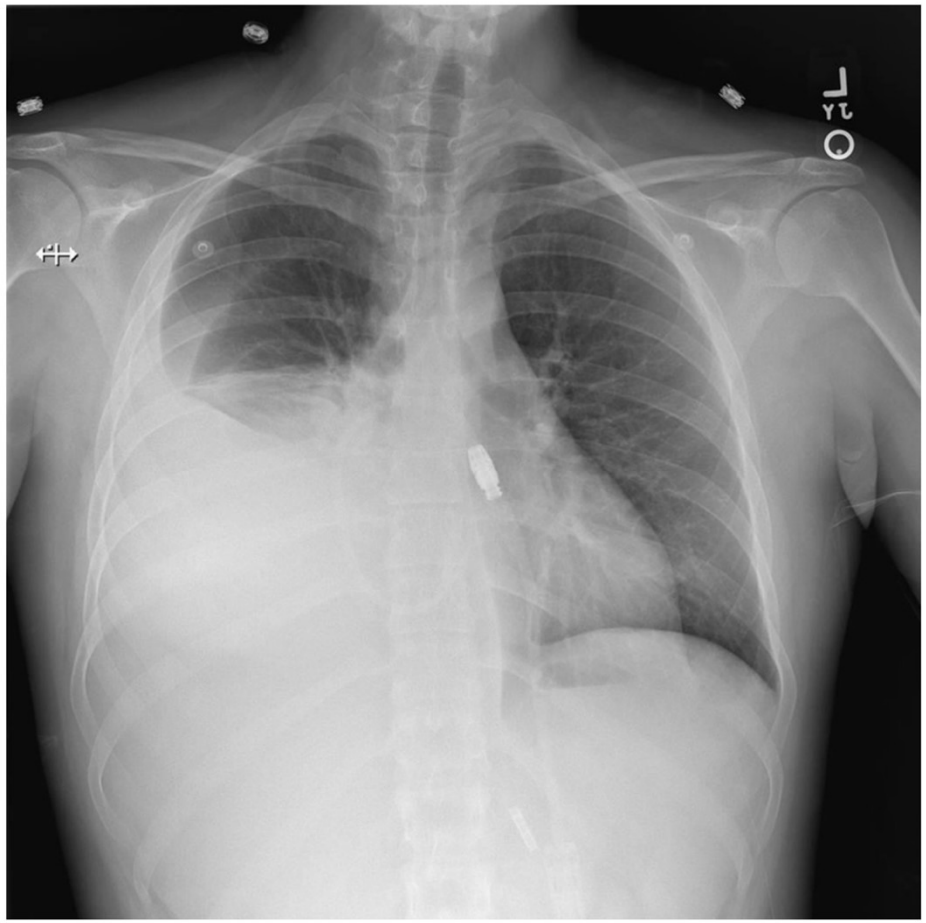
Complex



I is for Impression

Put your money down! Give me an impression!

Back to our case



Source: Allaham H, Hudhud D, Salzer W. [redacted]: a peritoneal dialysis dilemma. BMJ Case Rep. 2018.

So this is...

A. Anterior-Posterior with poor penetration
B. PA & Lateral with poor penetration
C. PA & Lateral with good penetration
D. Anterior-Posterior with good penetration

Do you see…

A. Enlarged cardiac silhouette, normal lung fields bilaterally
B. Normal cardiac size, Normal Left Lunch, Large Right sided effusion
C. Deviated trachea, Left sided effusion
D. Bilateral ground glass opacities

Let’s give our impression!
This is....

A. A collapsed Right sided lung
B. An enlarged liver in the right pleural space
C. Fluid overload from heart disease
D. Hepatic Hydrothorax

To sum it all up...

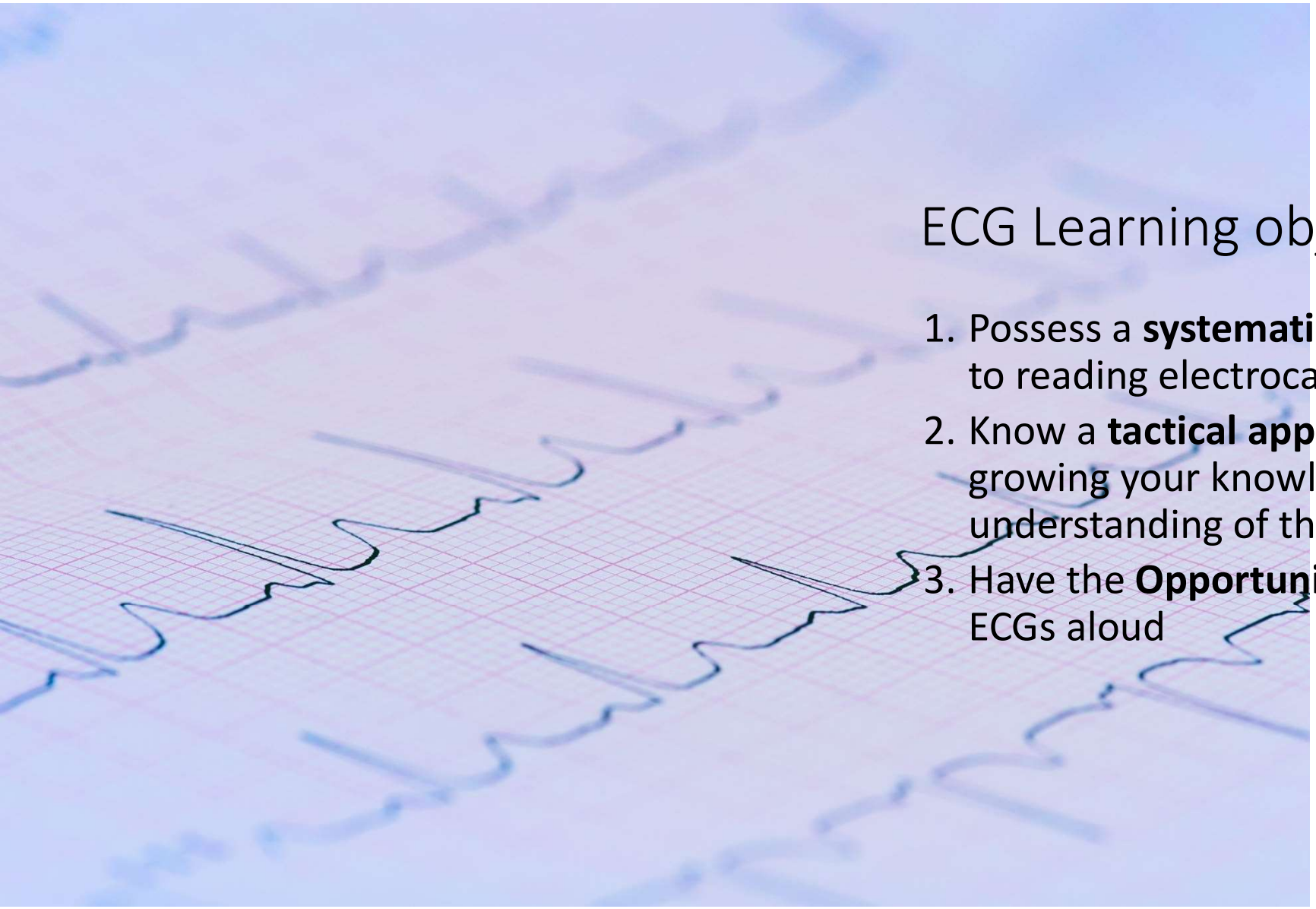
1. Chest Xrays are common in the hospitalized patient; the standard view is Posterior-Anterior & Lateral (P/A Lateral) which can better estimate the cardiac size
2. It is important to have a standardized system to consistently review Chest Xrays. The system covered here is Anatomy & Interpretation using M, A-I

References

1. Radiology Masterclass: https://www.radiologymasterclass.co.uk/
2. University of Virginia - Introduction to Chest Radiology: https://www.med-ed.virginia.edu/courses/rad/cxr/
3. Allaham H, Hudhud D, Salzer W. Right-sided hydrothorax: a peritoneal dialysis dilemma. BMJ Case Rep. 2018 May 26;2018:bcr2018225166. doi: 10.1136/bcr-2018-225166. PMID: 29804087; PMCID: PMC5976137.

ECG Session

Brian Wolfe, MD
Associate Professor of Medicine
University of Colorado Anschutz School of Medicine

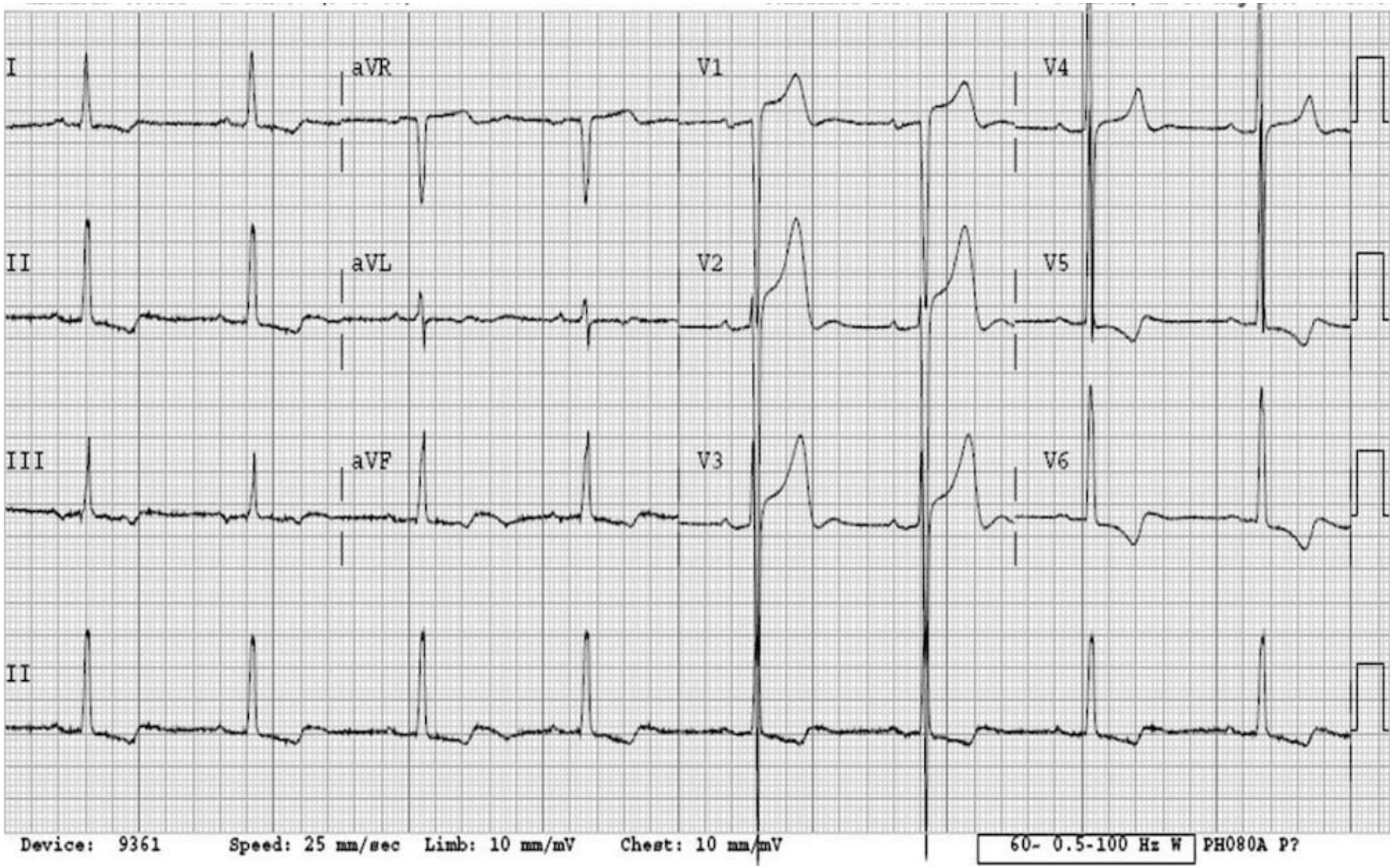


ECG Learning objectives

1. Possess a **systematic approach** to reading electrocardiograms
2. Know a **tactical approach** to growing your knowledge and understanding of this tool
3. Have the **Opportunity** to read ECGs aloud

Bring it back to our case…

- The ECG tech hands you Ms. T. S. Wift ECG...
- As you are reviewing the ECG she states, "Long story short, this chest pain just hits different. Don't blame me, I just can't shake it off"


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
Device: 9361
Speed: 25 mm/sec
Limb: 10 mm/mV
Chest: 10 mm/mV
60- 0.5-100 Hz W
PH080A P?

ECG Basics

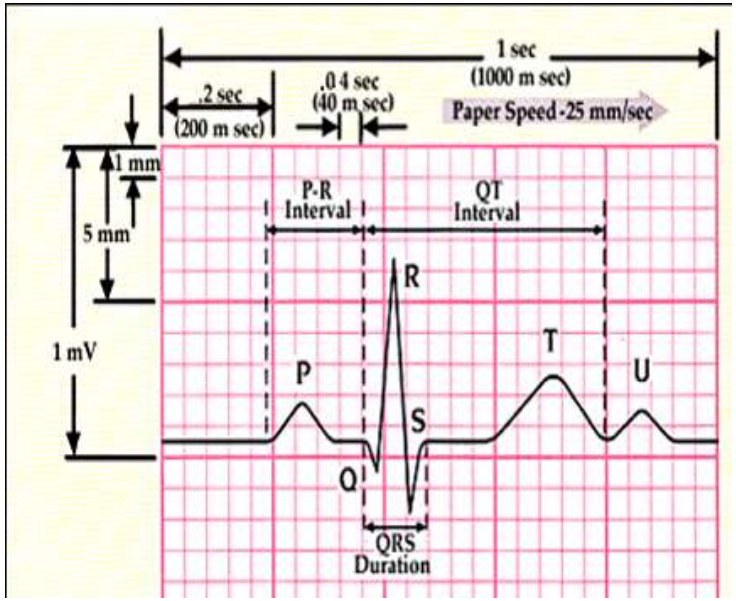
Lateral: Sm box = 0.04 s

Lrg box = 0.20 s

Typical ECG: 10 sec (25mm/s)

2.5 sec of each lead

Full 10s of lead II



ECG Basics

Intervals:

PR (begin P – begin QRS)

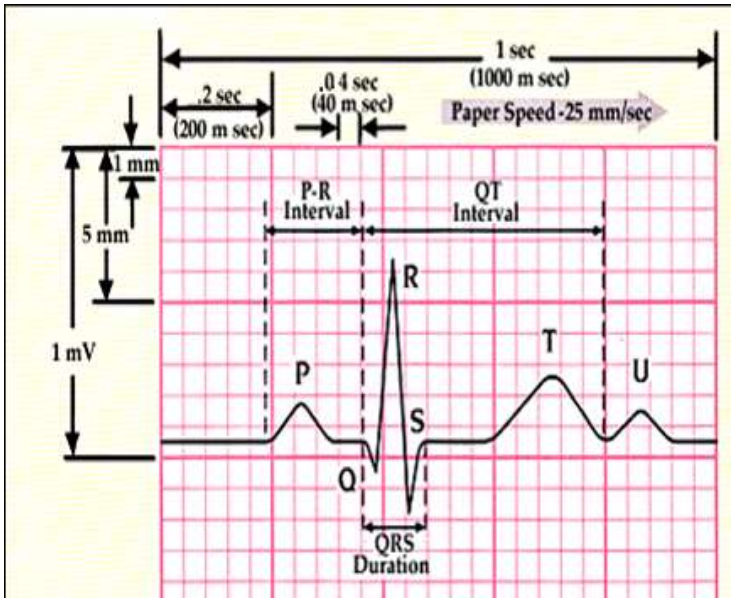
0.12 – 0.20

QRS (begin to end QRS)

0.08-0.12

QT (begin QRS – end T w)

QT**c** 470-480 ULN



Secrets about this session..

- This skill is important: if you excel at reading ECGs it will make a clinical difference
- You will not get good at ECGs from being taught in a lecture
- Lifetime of practice and “reading blind” and then double-checking your work
- So we are going to start today!

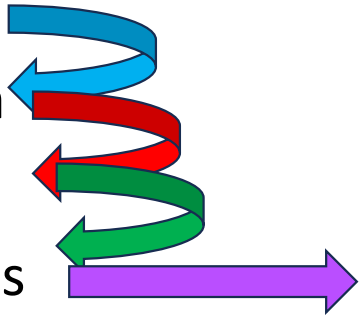
Cases: Gender

Titles shortened to:

- F for identifying as Female
- M for identifying as Male
- NB for identifying as non-binary
- Sex assigned at birth not important for any of the following cases

Reading

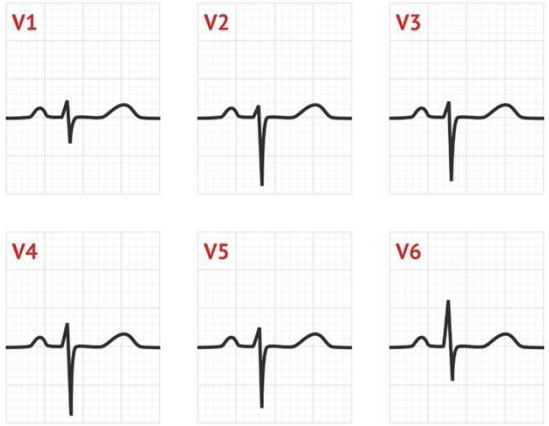
- Rate
- Rhythm
- Axis
- Intervals
- R-wave Progression
- ST/Twave/Qwaves - Ischemia
- Waveform (LVH, RVH, others)
- Diagnosis (if appropriate)



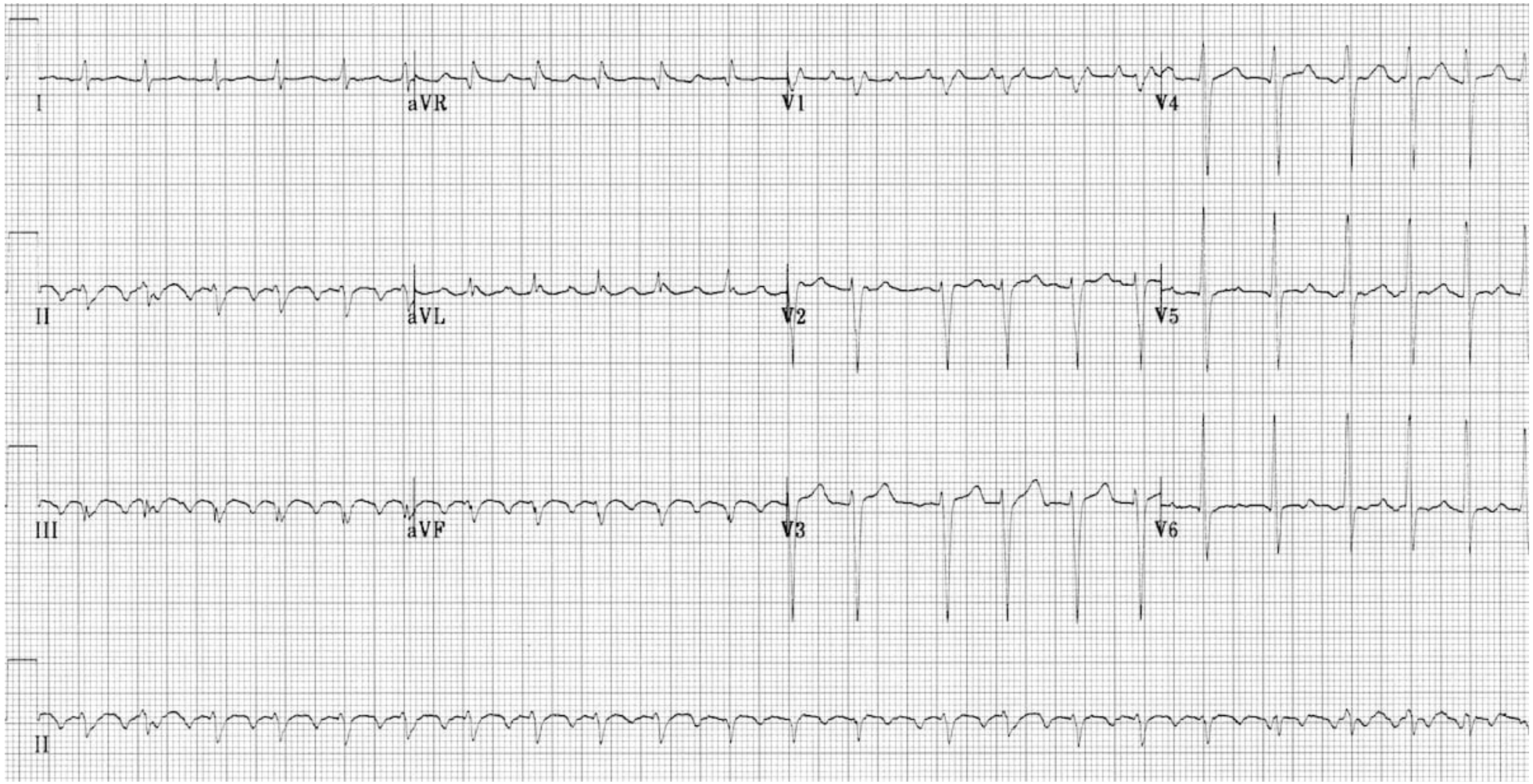
Prolonged PR interval:
1 deg AV nodal block

Normal R wave progression:

Poor R wave progression:



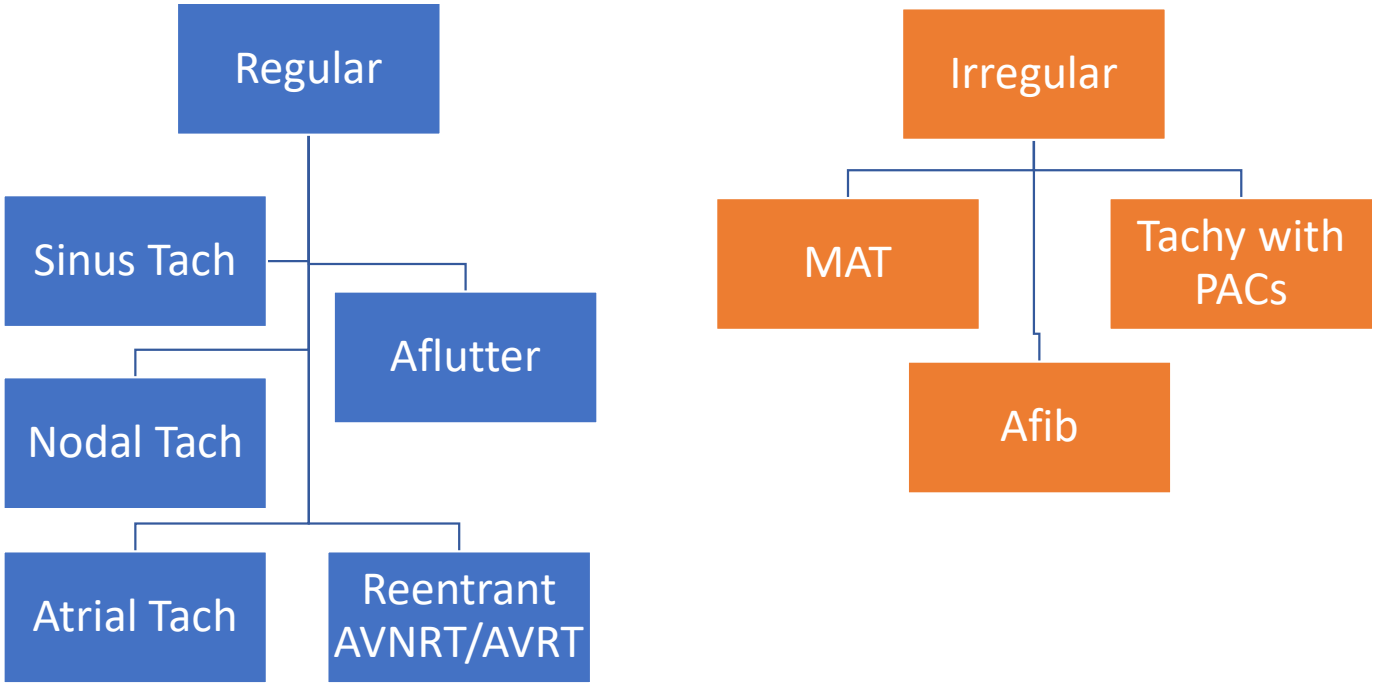
72 yo F w/SOB



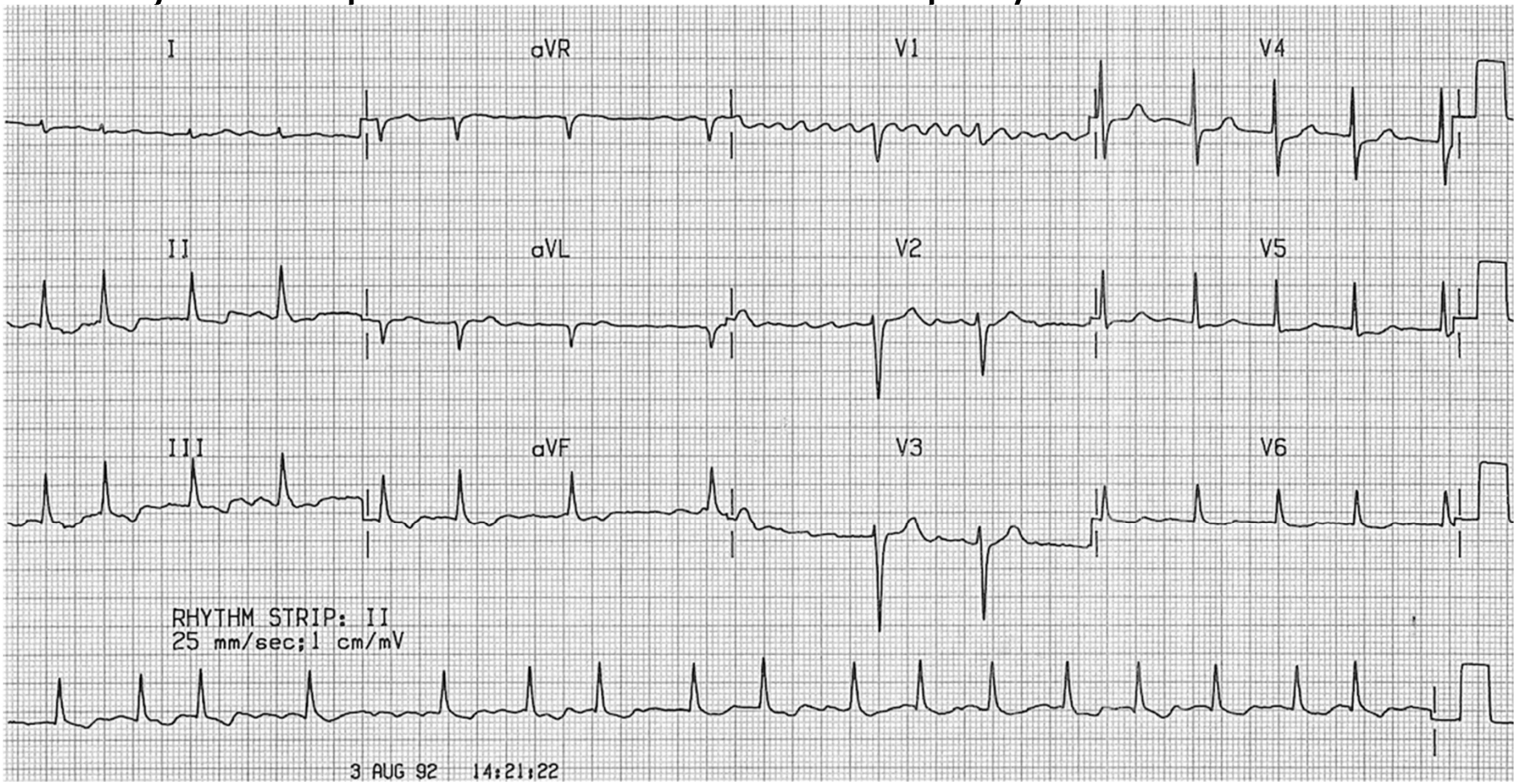
Reading

- Rate
- Rhythm → Narrow Complex Tachycardia
- Axis
- Intervals
- R-wave Progression
- ST/Twave/Qwaves
- Waveform
- Diagnosis (if appropriate)

Narrow Complex Tachycardia



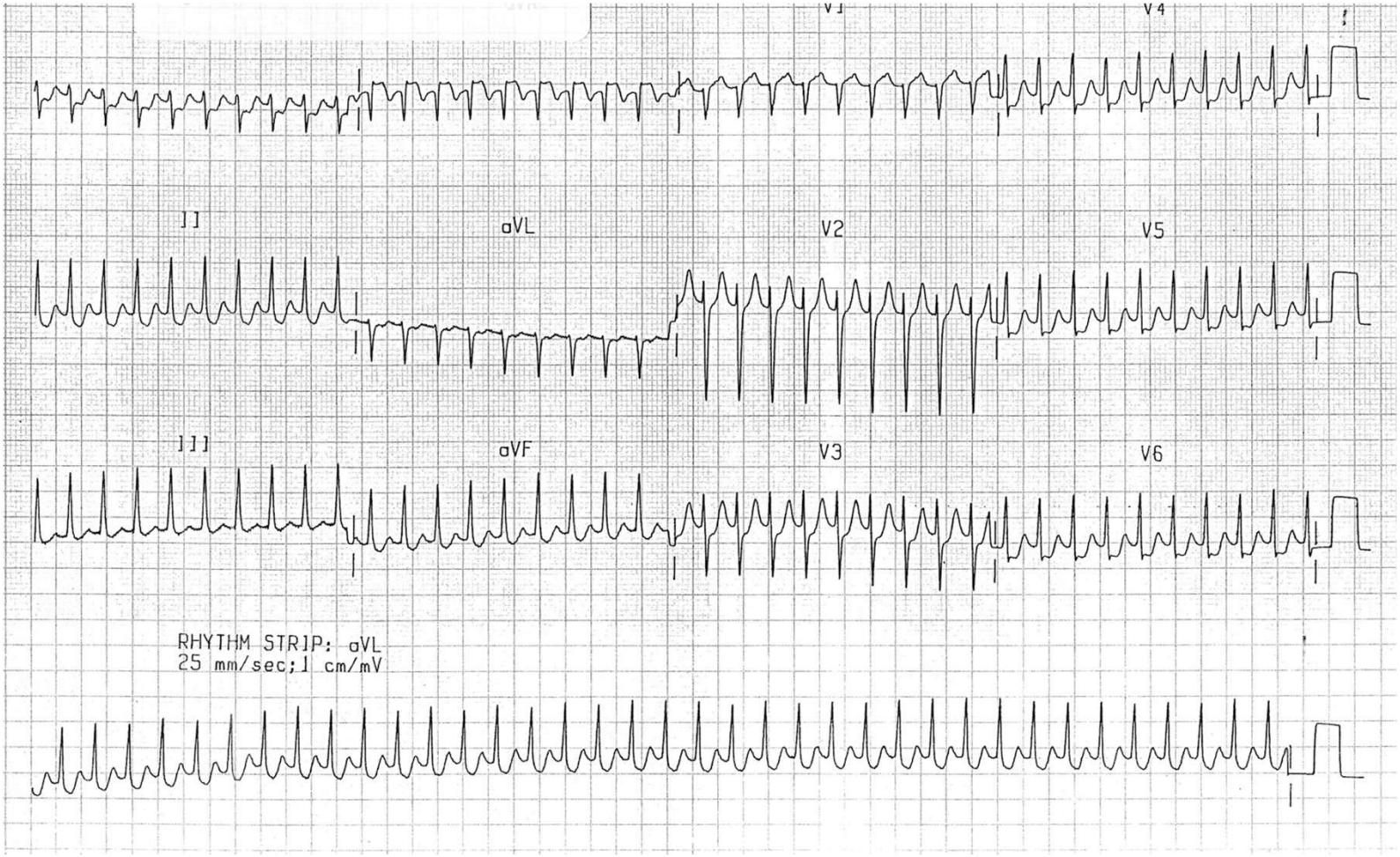
45 yo NB person for normal physical



Atrial Fibrillation

- Classic irregular narrow complex tachycardia

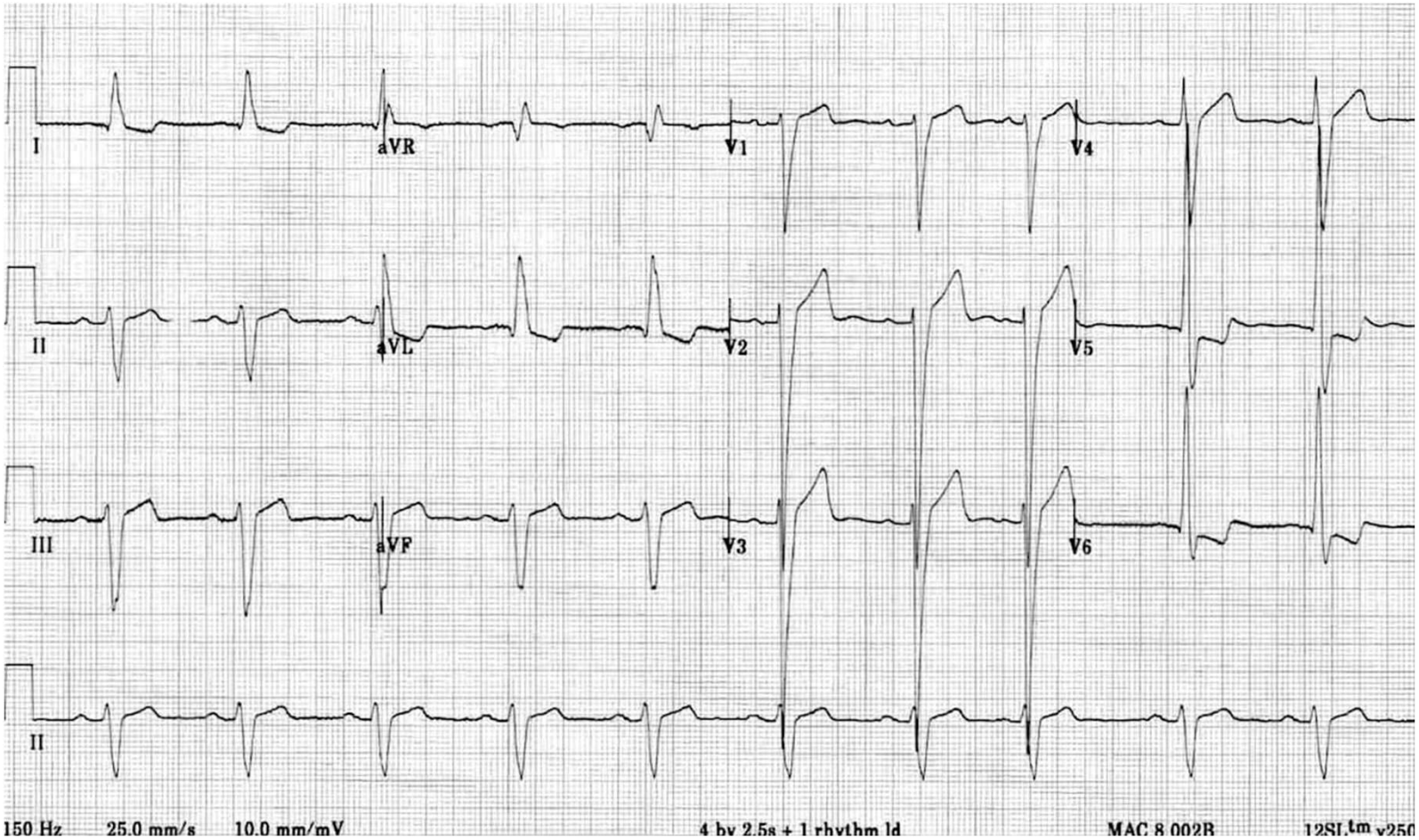
18 yo M athlete w/SOB



AVNRT (reentrant)

- Narrow complex tachycardia
- Can be very fast, much faster than Sinus tach which typically is <220-age max heart rate
- Retrograde P-waves can sometimes be seen

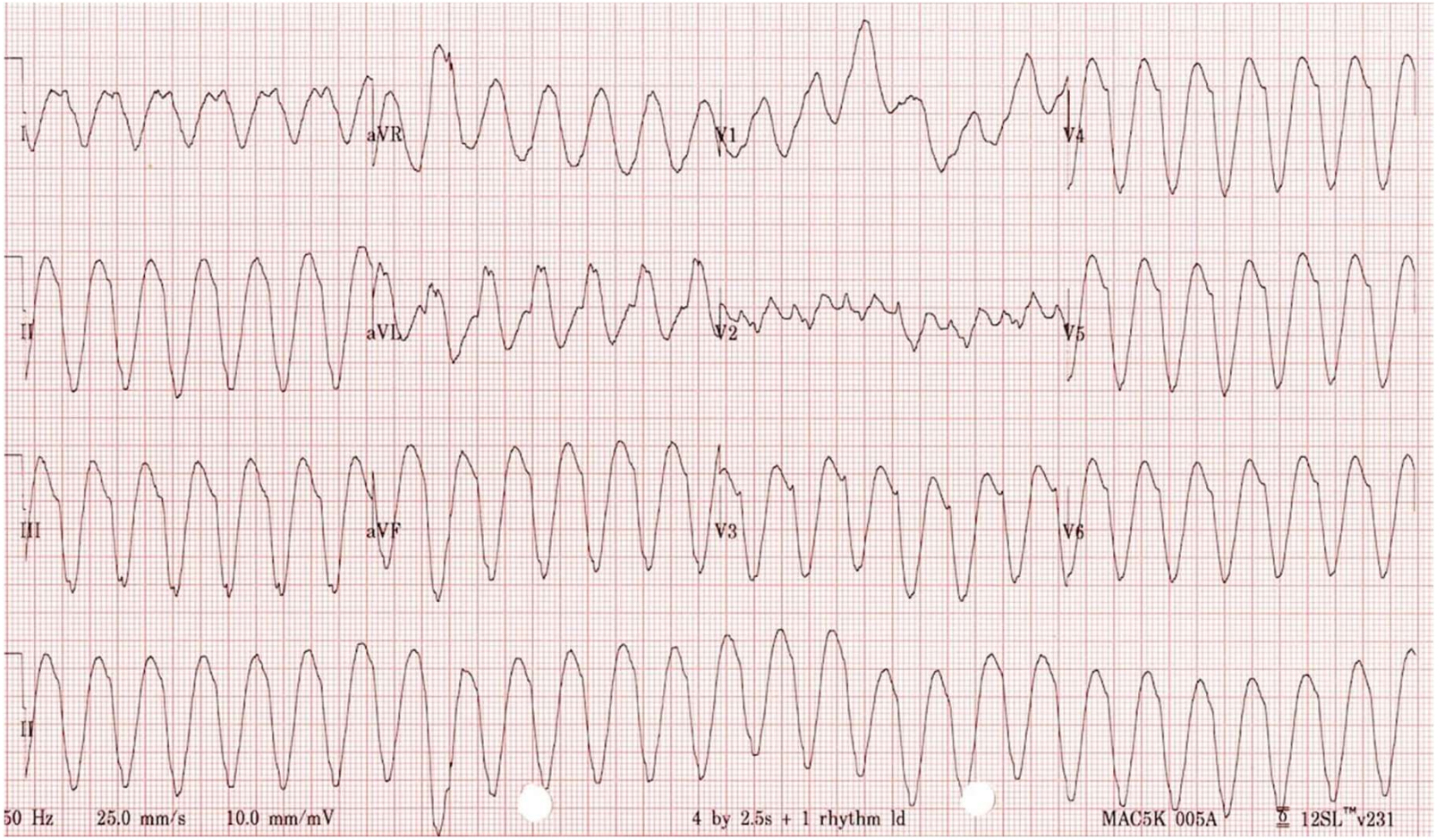
45 NB w/atypical CP



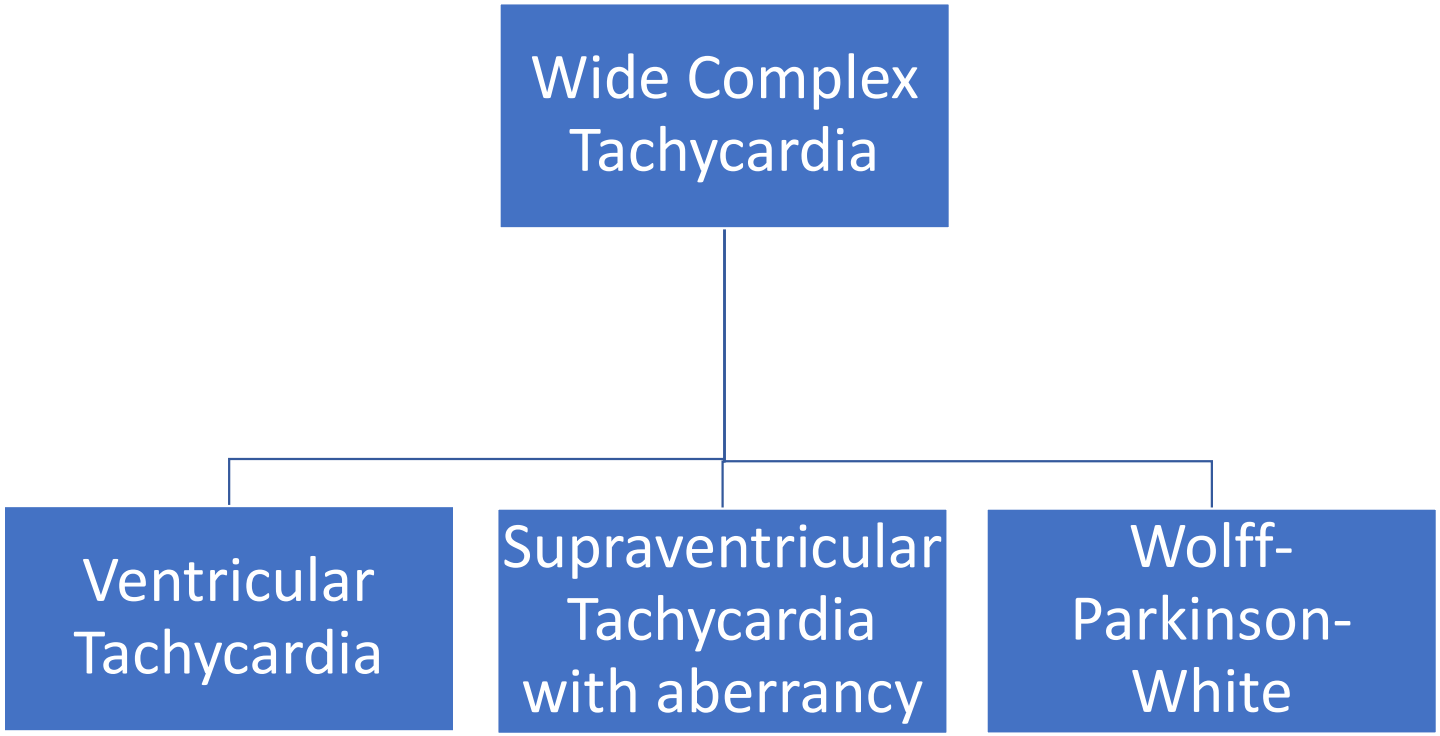
Left ventricular hypertrophy (LVH)

- Markedly increased LV voltages: huge precordial R and S waves that overlap with the adjacent leads (SV2 + RV6 >> 35 mm).
- R-wave peak time > 50 ms in V5-6 with associated QRS broadening.
- LV strain pattern with ST depression and T-wave inversions in I, aVL and V5-6.
- ST elevation in V1-3.
- Prominent U waves in V1-3.
- Left axis deviation.

56 yo unconscious man



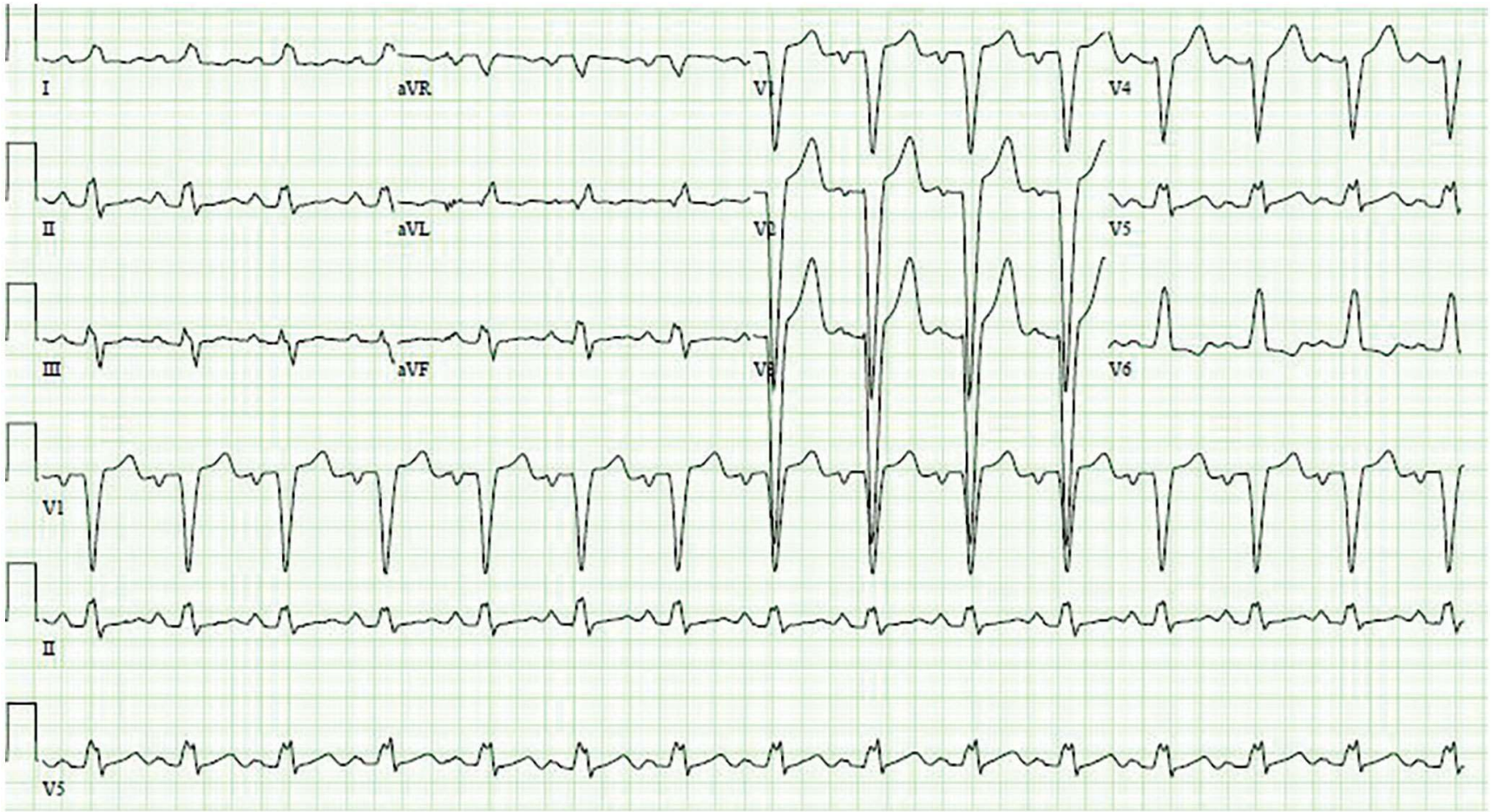
Wide Complex Tachycardia



Ventricular Tachycardia - characteristics

- Very broad complexes (>160ms)
- AV dissociation (P and QRS complexes at different rates)
- Capture beats — occur when the sinoatrial node transiently ‘captures’ the ventricles, in the midst of AV dissociation, to produce a QRS complex of normal duration
- Fusion beats — occur when a sinus and ventricular beat coincide to produce a hybrid complex of intermediate morphology
- Positive or negative concordance throughout the chest leads, i.e. leads V1-6 show entirely positive (R) or entirely negative (QS) complexes, with no RS complexes seen

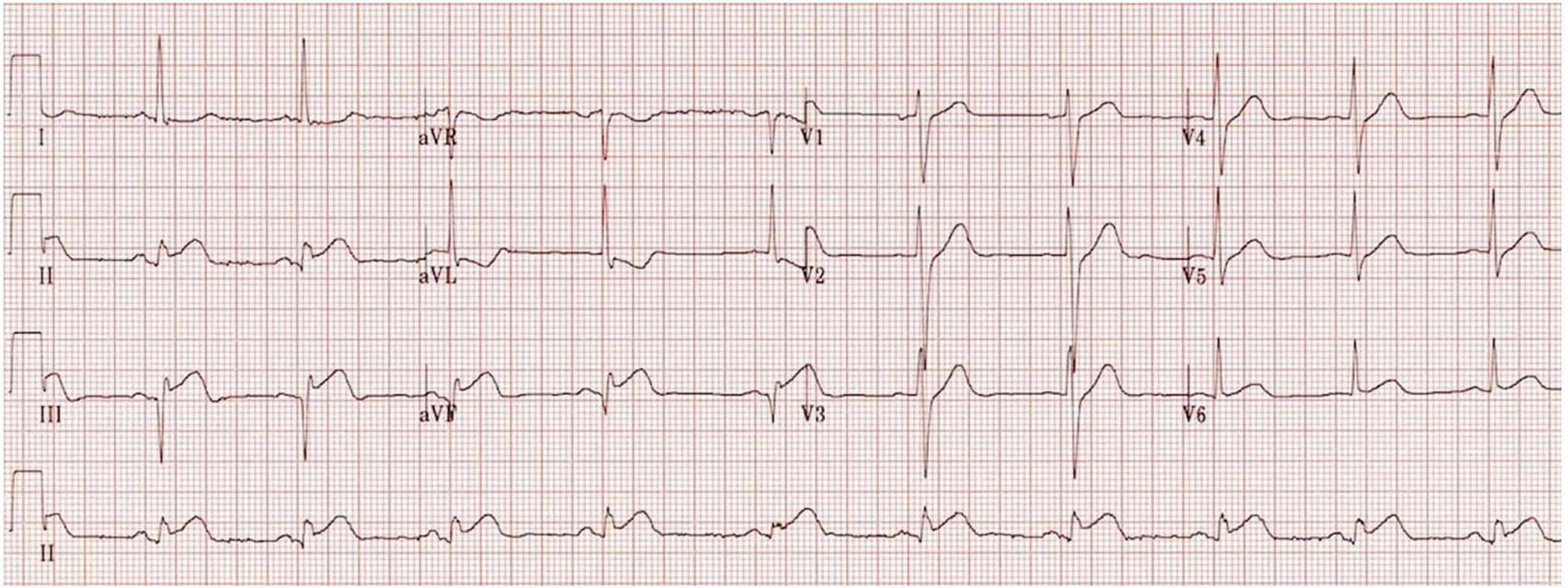
59 yo M in preop clinic



Left Bundle Branch Block

- QRS >120 msec
- Typical LBBB pattern:
  - Largely negative forces in V1
  - Positive forces in I and V6
- Expected repol abnormalities:
  - Disconcordant ST and T wave changes
    (if QRS forces negative, then ST elevation, e.g.)

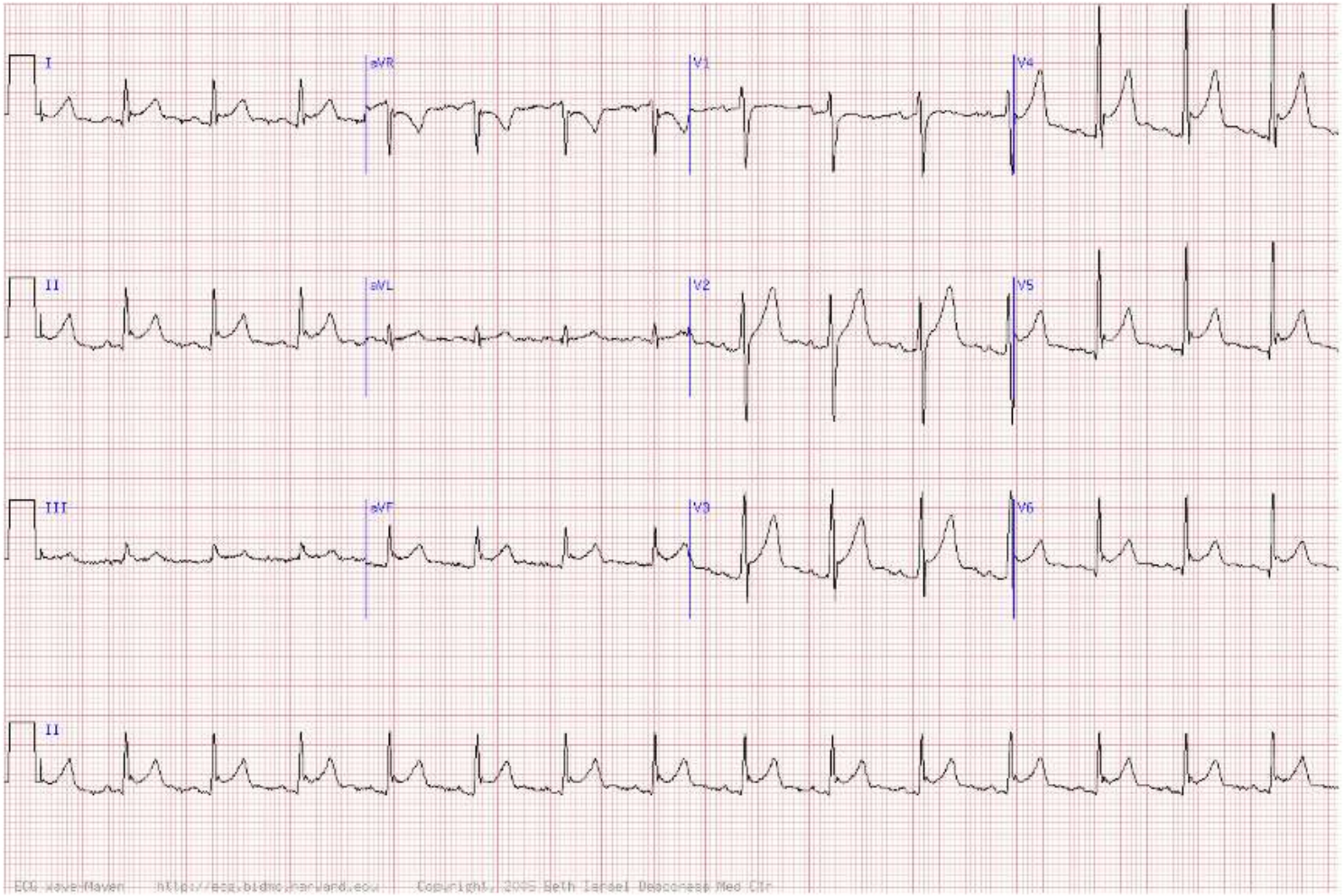
63 yo M w chest pain



STEMI

- Inferior STEMI
- ST elevation in II, III and aVF.
- Q-wave formation in III and aVF.
- Reciprocal ST depression and T wave inversion in aVL
- ST elevation in lead II = lead III and absent reciprocal change in lead I (isoelectric ST segment) suggest a circumflex artery occlusion

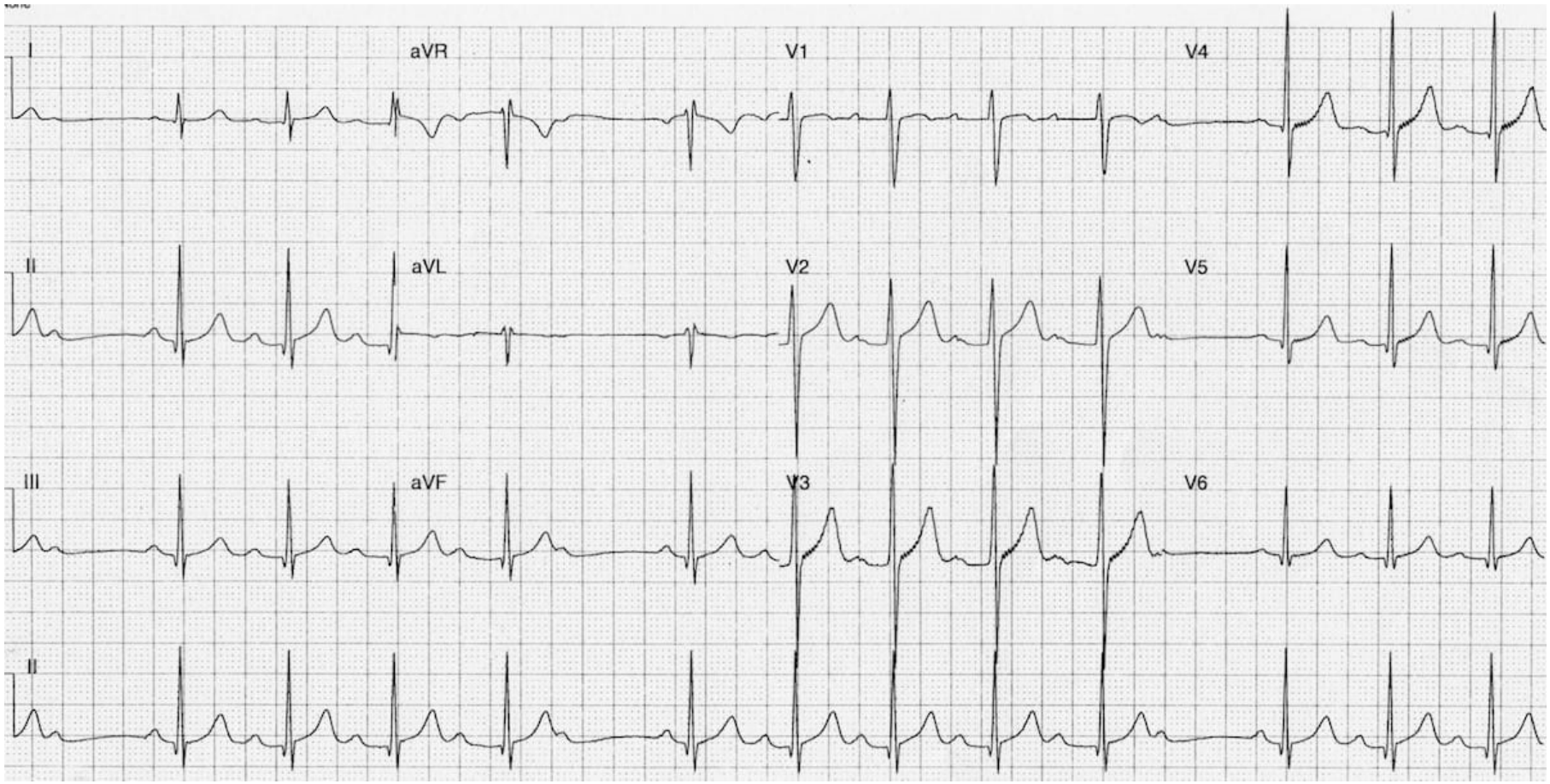
32 yo NB w/chest pain



Pericarditis

- Widespread ST elevations
- Depressed P waves
- V6: ST:T wave amplitude ratio exceeds 0.25

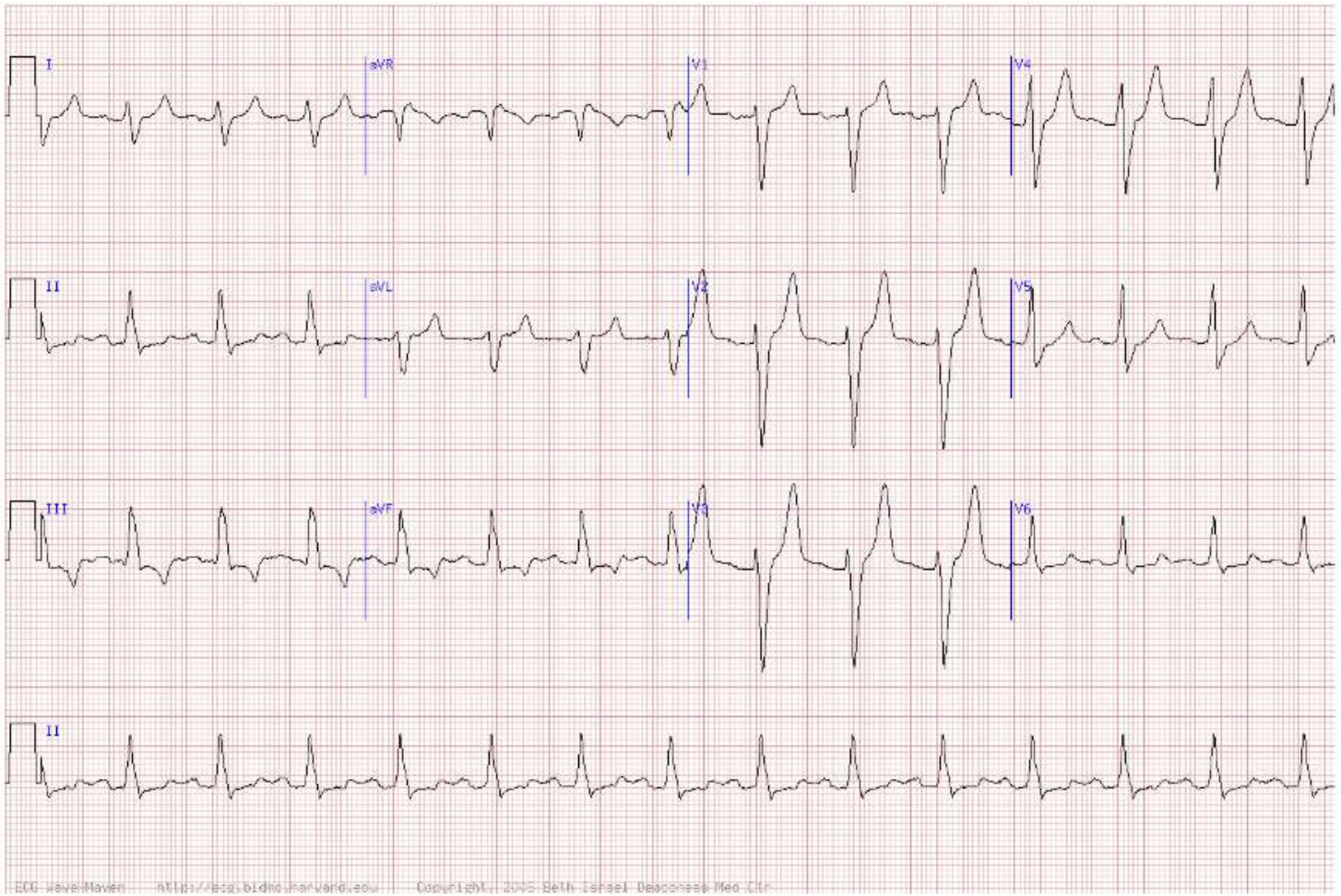
65 yo M with lightheadedness



2nd Degree, Mobitz I AV block

- QRS complexes clustered in groups, separated by non-conducted P waves.
- The P:QRS conduction ratio varies from 5:4 to 6:5.
- Note the difference in PR interval between the first and last QRS complex of each group.

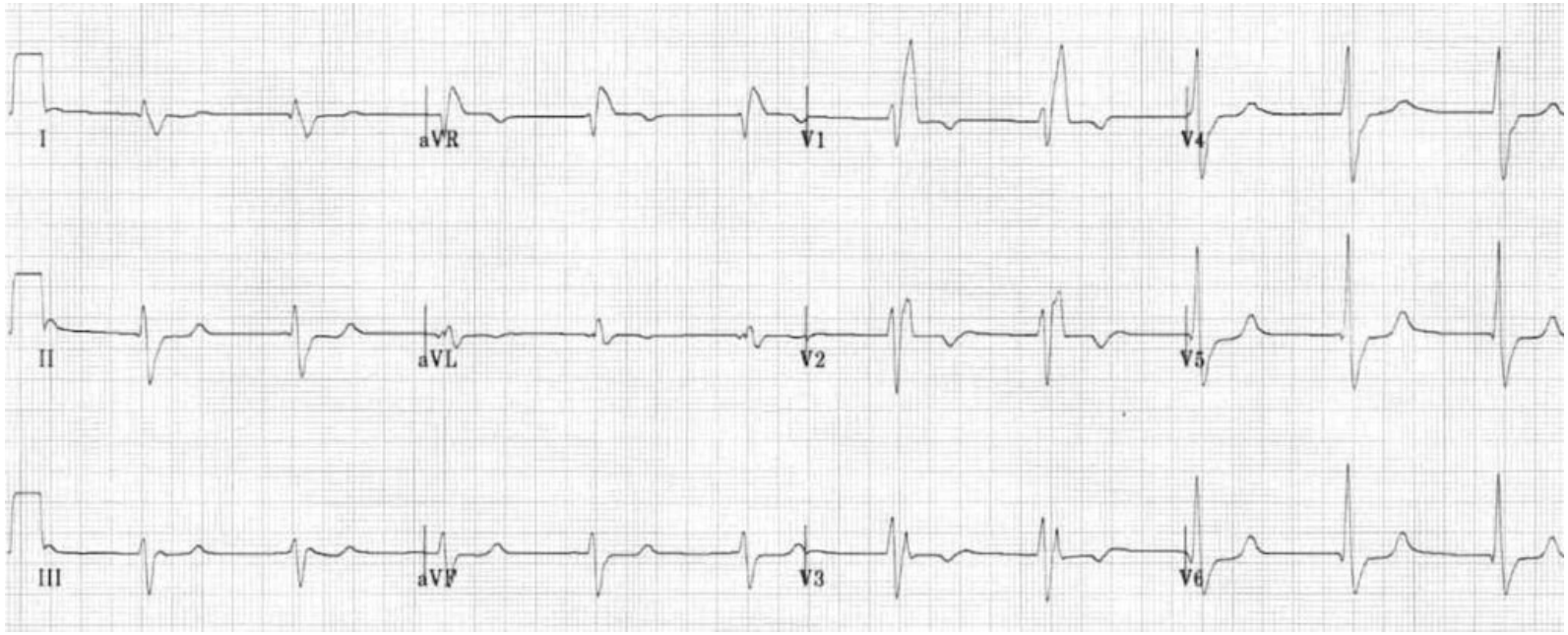
45 yo F weakness/nausea



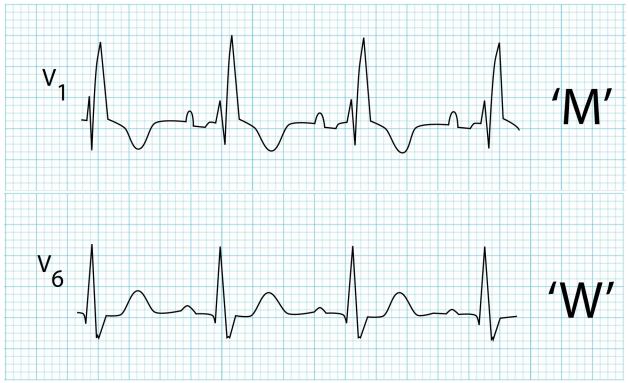
Hyperkalemia

- The serum potassium was 8.9 mEq/L. Note the wide QRS with tall ("peaked" or "tented") T waves in V2-V4. The PR interval is prolonged. Left atrial abnormality is also present.

47 yo NB w/pneumonia



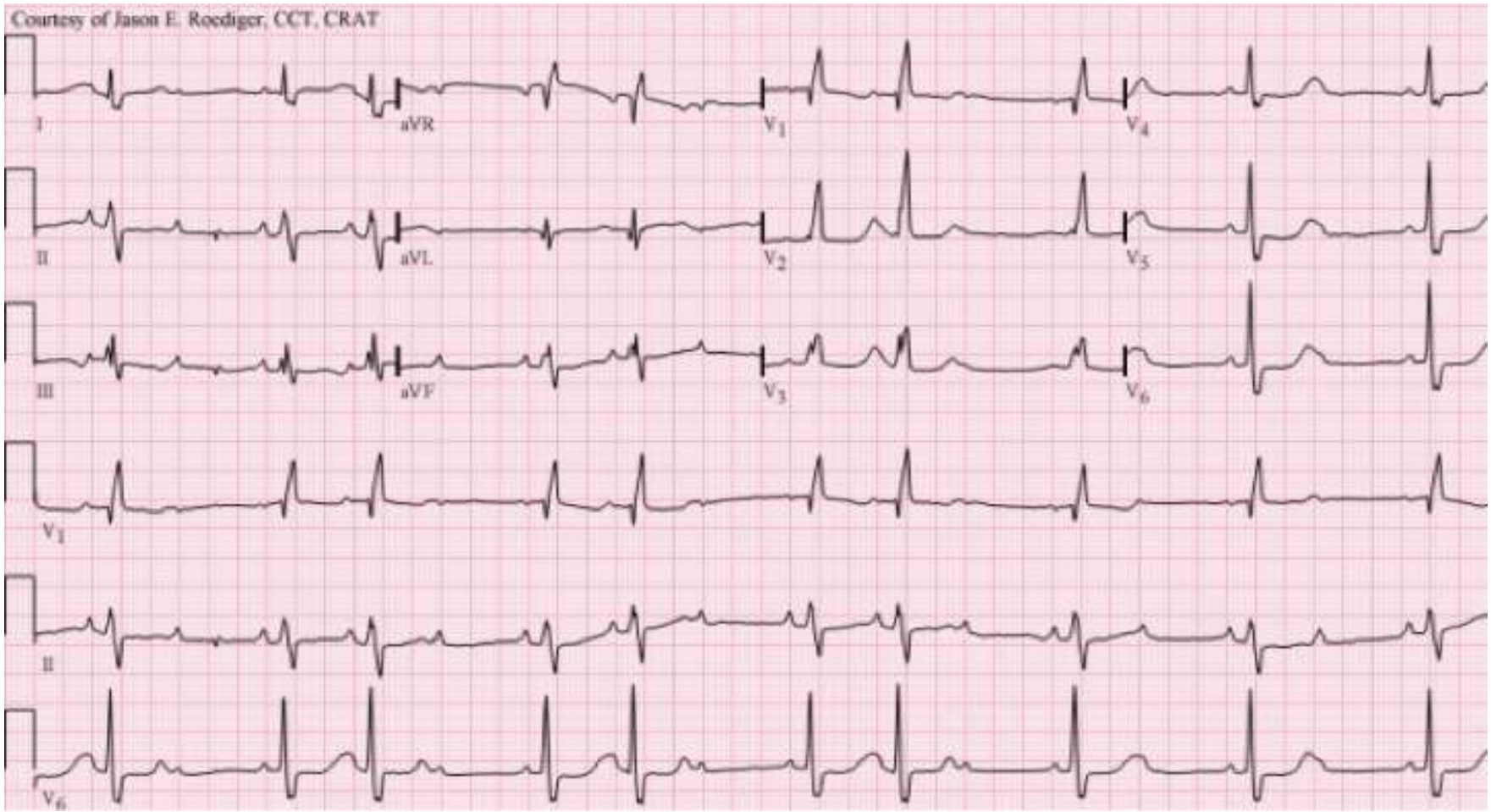
- QRS duration > 120ms
- RSR' pattern in V1-3 ("M-shaped" QRS complex)
- Wide, slurred S wave in lateral leads (I, aVL, V5-6)



RBBB Ddx

- Right ventricular hypertrophy / cor pulmonale
- Pulmonary embolus
- Ischemic heart disease
- Rheumatic heart disease
- Congenital heart disease (e.g. atrial septal defect)
- Myocarditis
- Cardiomyopathy
- Lenègre-Lev disease: primary degenerative disease (fibrosis) of the conducting system

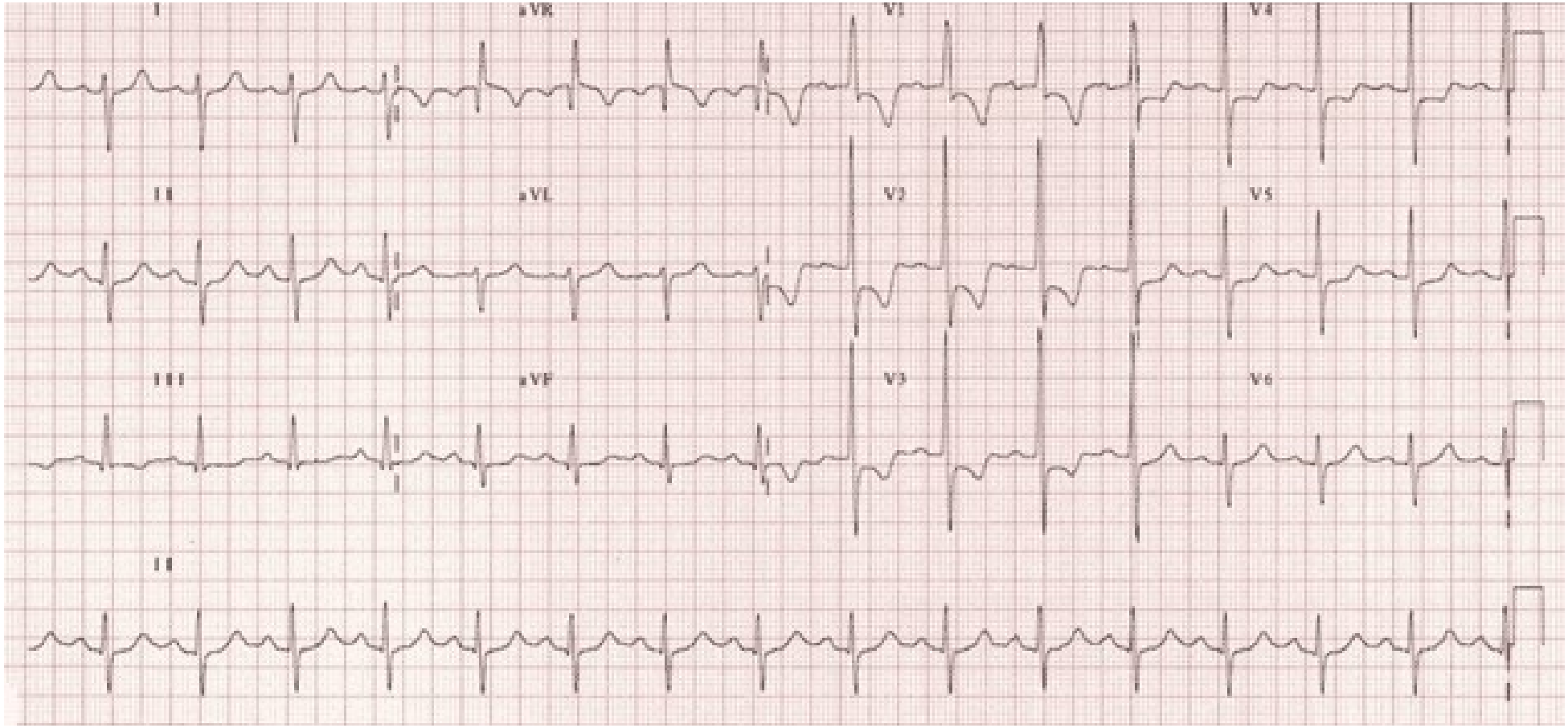
65 yo F w/dizziness



2nd Degree Block, Mobitz II

- 2:1 condution
- Normal PR interval
- Other conduction ab

63 yo W with SOB



Early R wave prog - RVH

Diagnostic criteria for RVH

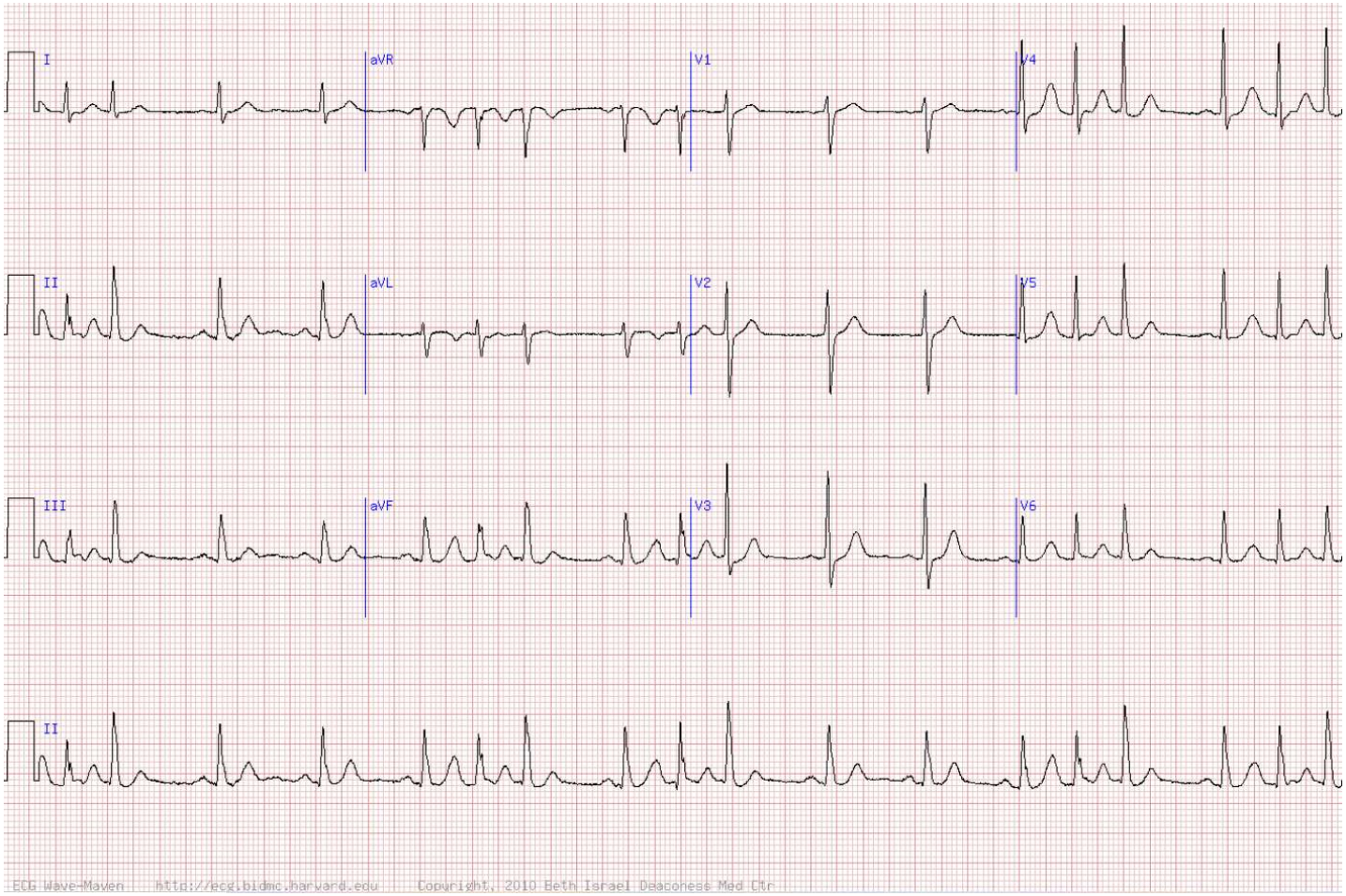
- QRS duration < 120 ms.
- Right axis deviation > +110°
- Dominant R wave in lead V1 ⩾7 mm

Supporting criteria for RVH

- ST segment depression/T wave inversion in anterior/inferior leads
- Deep S waves in leads V5, V6, I, and aVL (clockwise rotation)
- P pulmonale
- S1, S2, S3 pattern = far right axis deviation

Note: QRS needs to be <120 so that we don't attribute findings 2/2 RBBB.

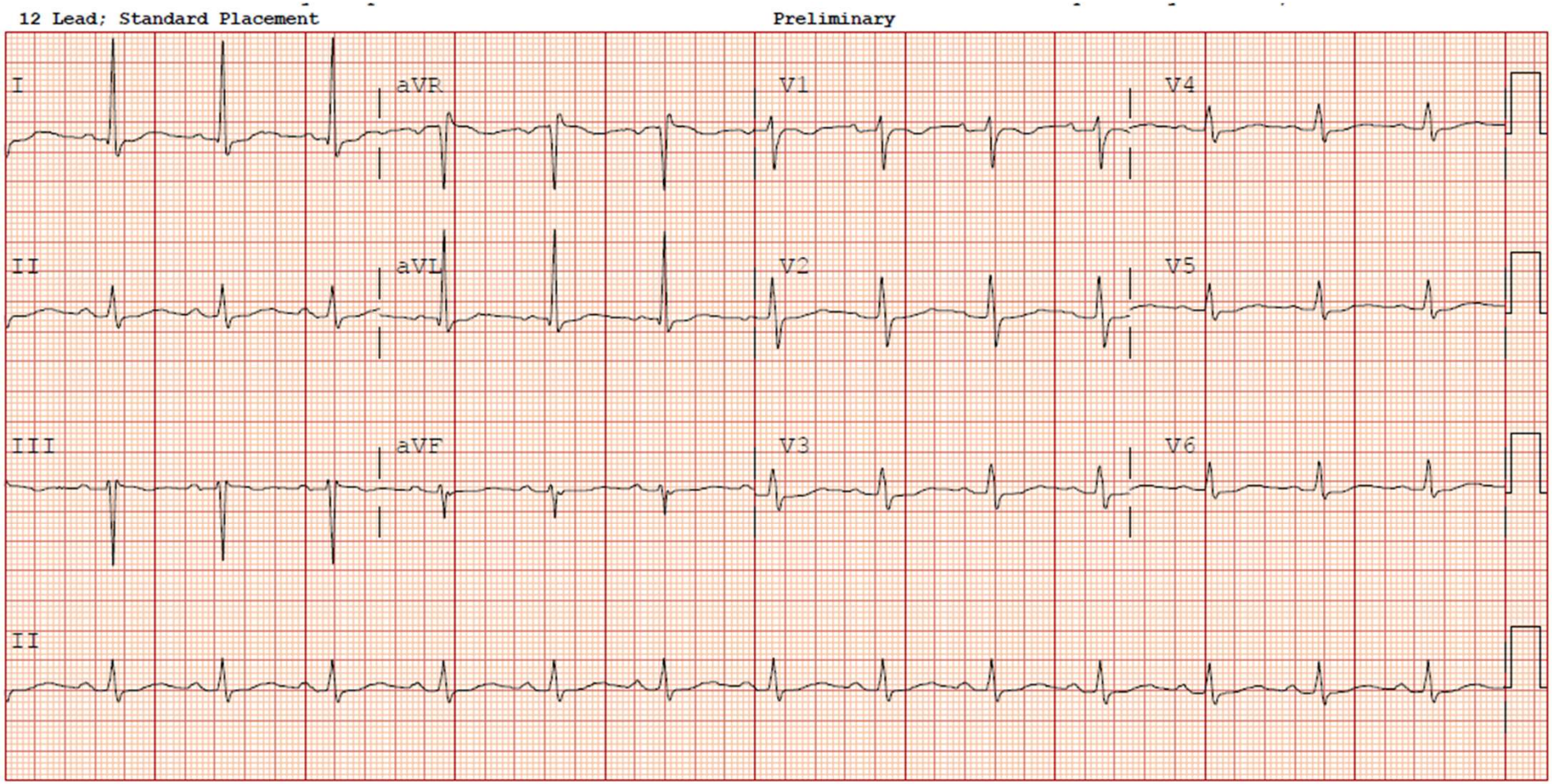
25 yo NB with irreg heartbeat



Premature Atrial Complexes (PACs)

- P-wave followed by QRS
- Earlier than expected by rate

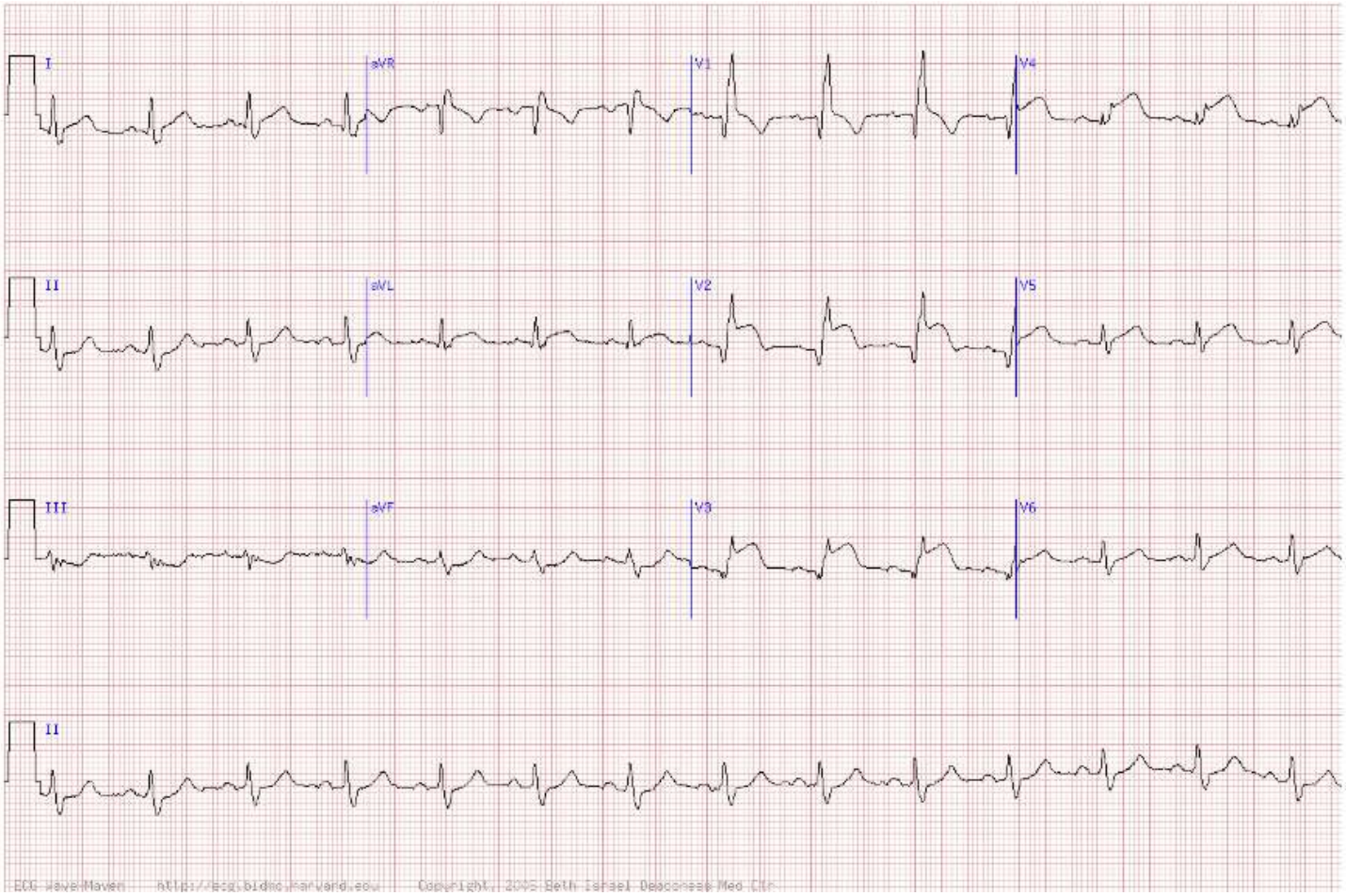
36 yo M referred for MDD



Long QTc

- Measured from beginning of QRS to end of T
- Note there is a correction should the patient have a bundle branch block (not present in this case)

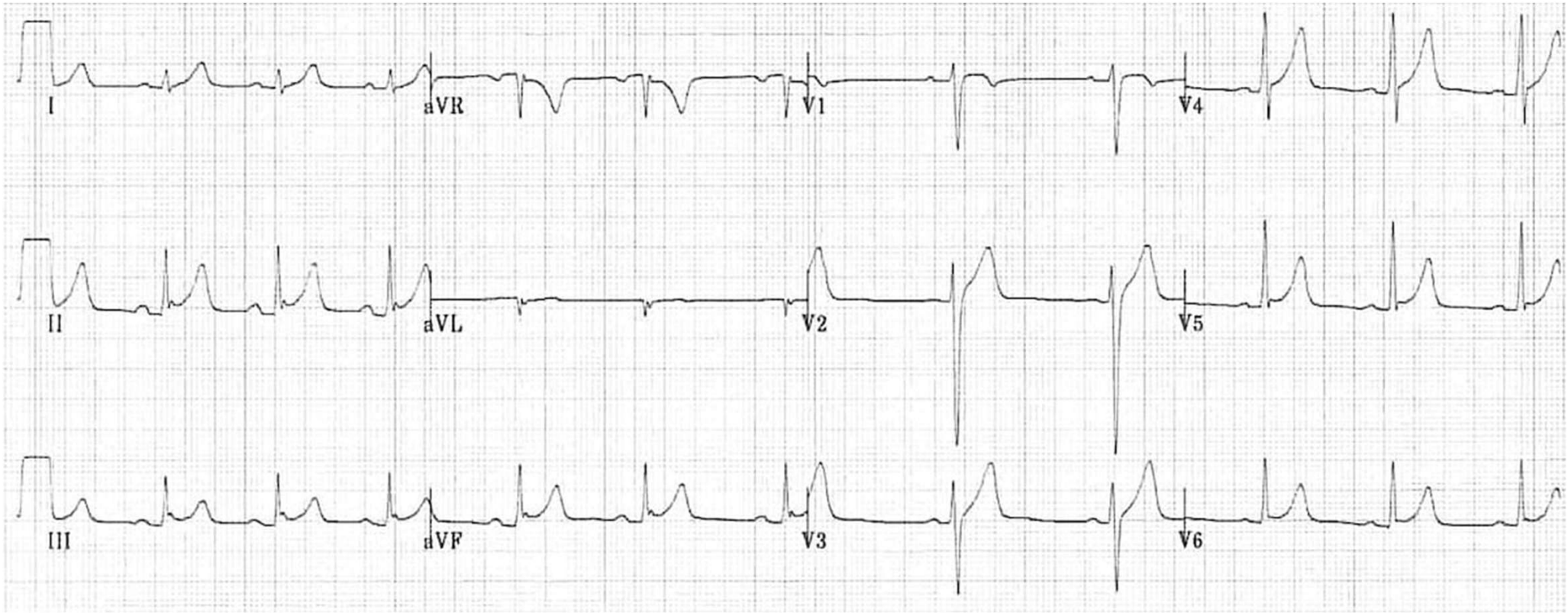
78 yo M with chest pressure



STEMI

- Anterolateral MI
- Reciprocal changes (to lateral STE's) in inferior leads

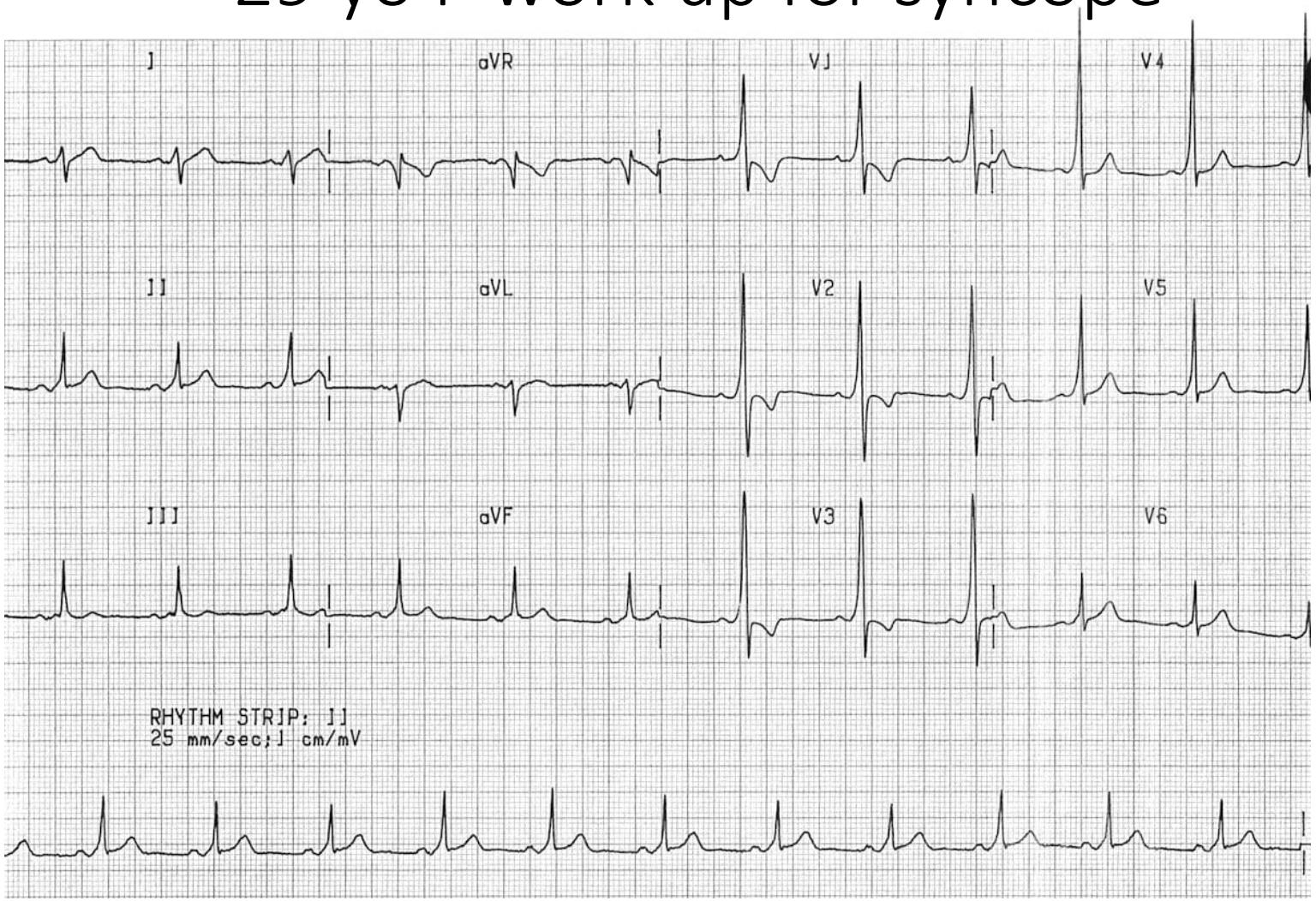
47 yo F w/HTN preop



Benign repolarization

- There is generalized concave ST elevation in the precordial (V2-6) and limb leads (I, II, III, aVF).
- J-point notching is evident in the inferior leads (II, III and aVF).
- There are prominent, slightly asymmetrical T waves that are concordant with the main vector of the QRS complexes.

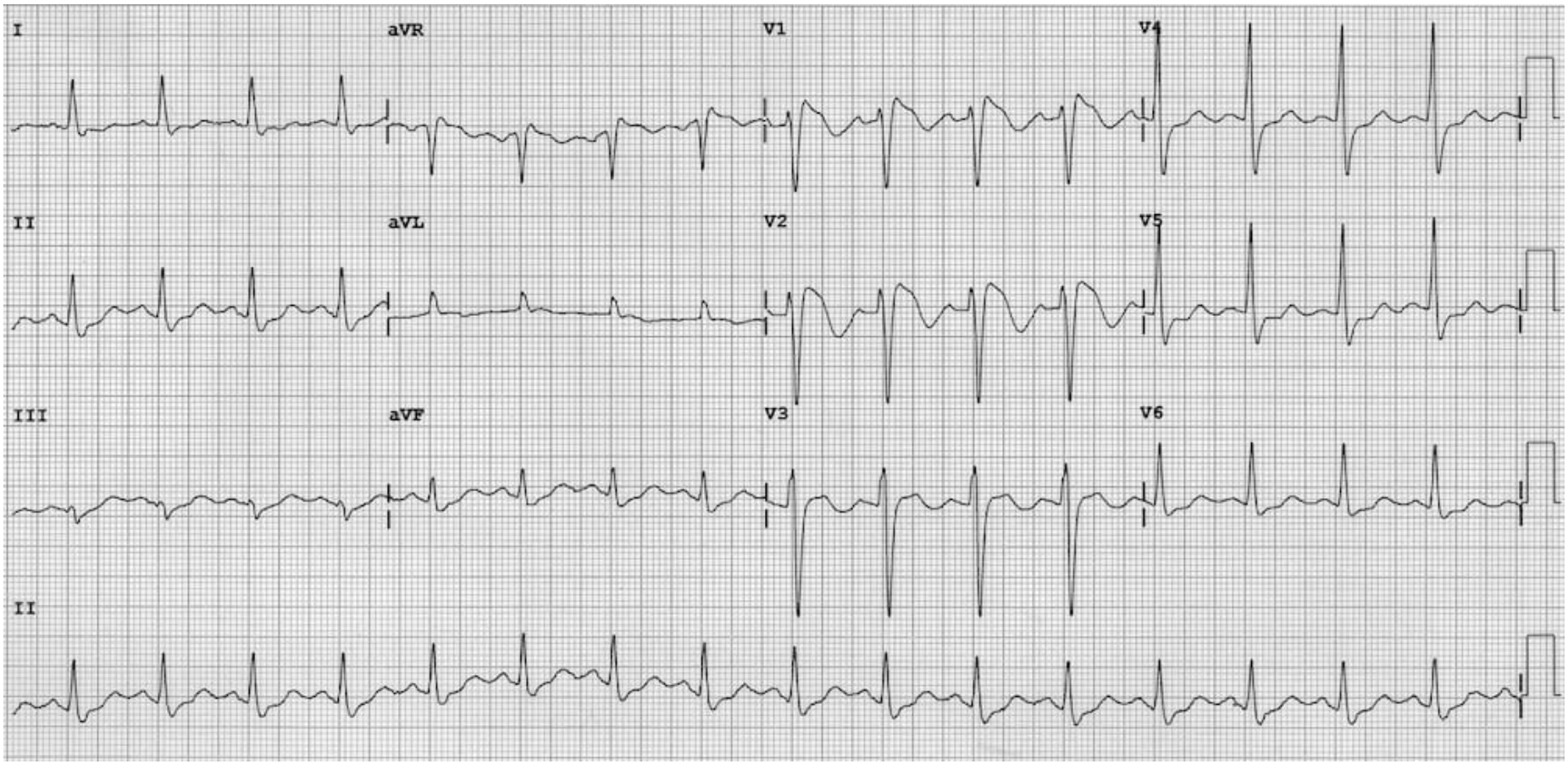
29 yo F work up for syncope



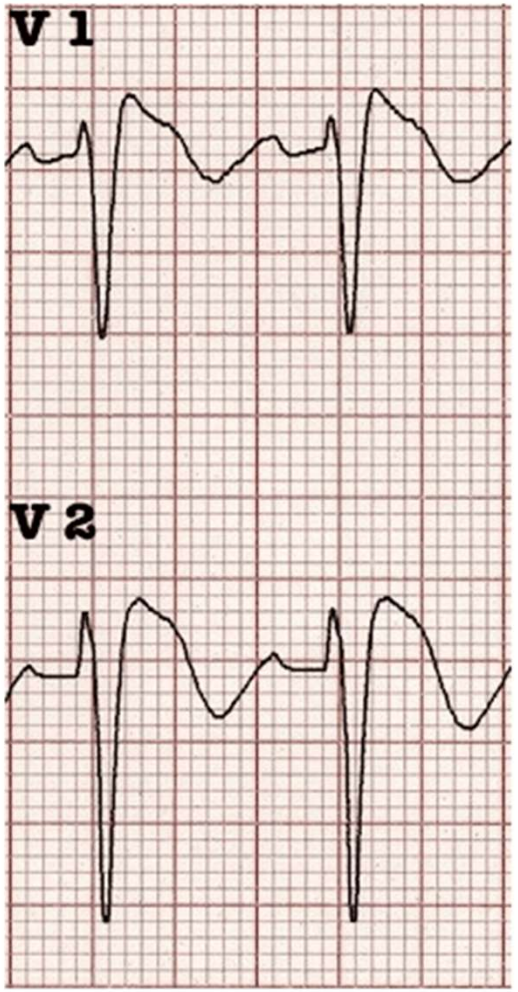
WPW

- Sinus rhythm with a very short PR interval (< 120 ms).
- Broad QRS complexes with a slurred upstroke to the QRS complex — the delta wave.
- **Dominant R wave in V1** — this pattern is known as "**Type A" WPW** and is associated with a **left-sided accessory pathway**.
- Tall R waves and inverted T waves in V1-3 mimicking right ventricular hypertrophy — these changes are due to WPW and do not indicate underlying RVH.
- Negative delta wave in aVL simulating the Q waves of lateral infarction — this is referred to as the **"pseudo-infarction" pattern**
- Type A WPW pattern with dominant R wave in V1 and right precordial T-wave inversions simulating RVH.

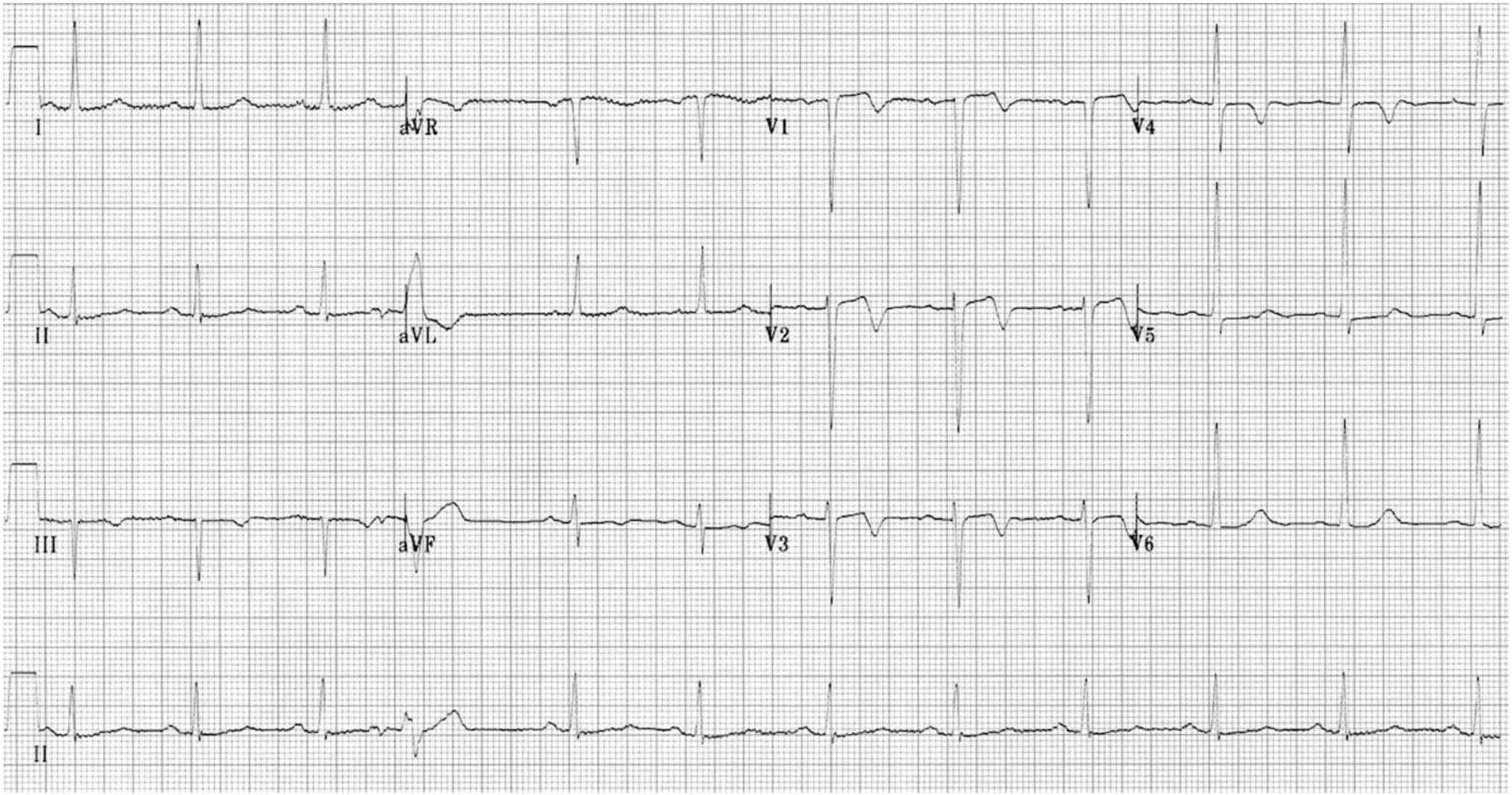
45 yo F with preop ECG



There is ST elevation and partial RBBB in V1-2 with a coved morphology — the “Brugada sign”.



68 yo M in ED with CP

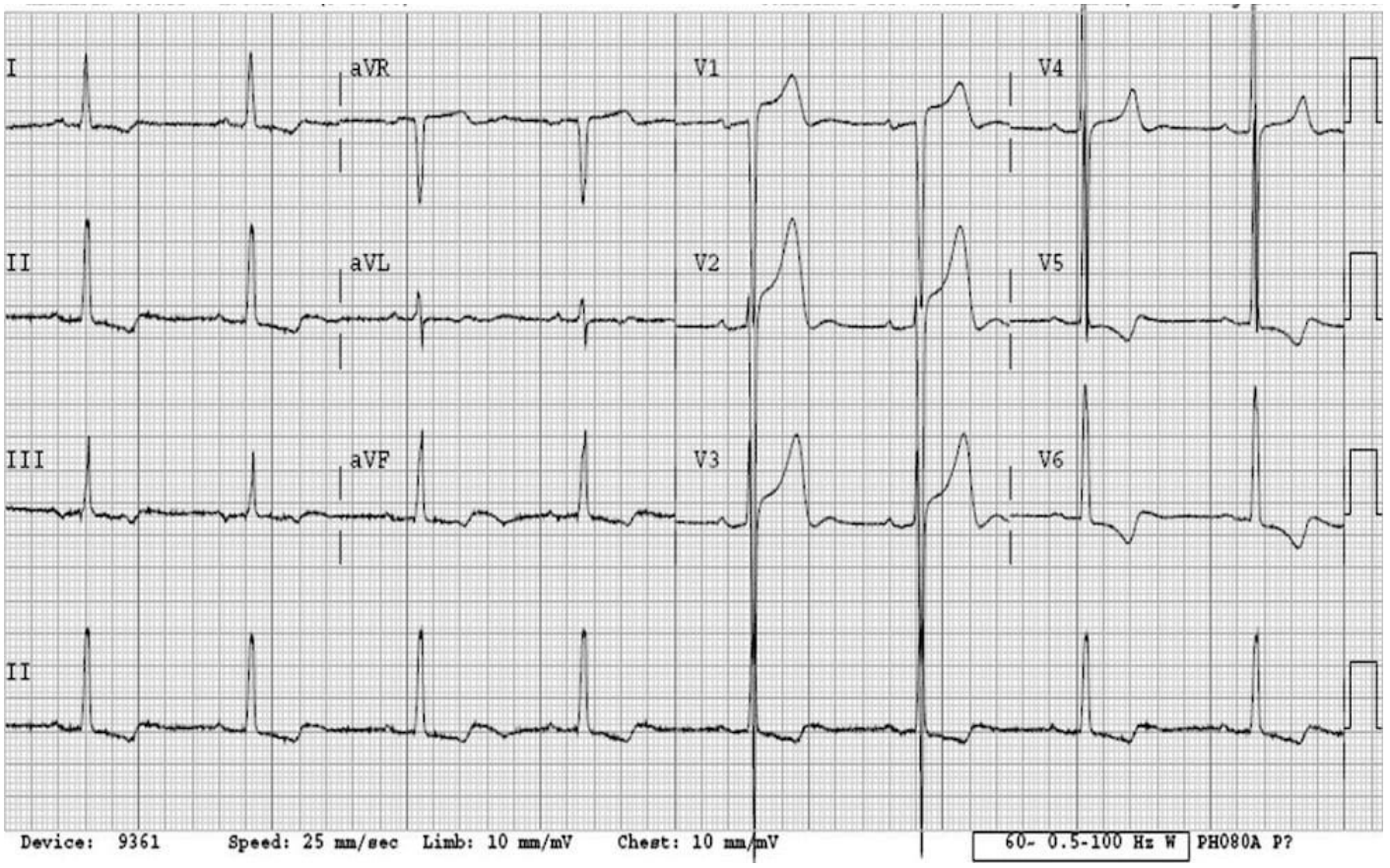


Wellen's Pattern

- There are abnormal T waves in V1-4 — biphasic in V1-3 and inverted in V4.
- This pattern is and is highly specific for a critical stenosis of the proximal LAD artery
- Type A Wellen's Pattern

T. S. Wift's ECG

- **Rate**: ~50
- **Rhythm**: NSR
- **Axis**: normal axis
- **Intervals**:
  PR 205 -> 1deg AVB
- QRS 100
- QT 440 (normal QTc)
- **RVP:** normal
- **Ischemia:** ST elevations in anteroseptal leads, ST depressions in intero/lateral leads, "strain pattern"
- **Waveform:** voltage -> c/w LVH
- **Dx:** likely simply LVH with repolarization abnormalities



To sum it all up...

1. Reading ECGs is a skill that takes real world practice. You will need to force yourself to read these using a systematic approach to gain this skill
2. A common approach to reading ECGs is rate, rhythm, axis, intervals, R-wave progression, ST/Twave/Qwave, waveform

References

ECGs drawn from:

1. Life in the Fast Lane: https://litfl.com/library/
2. Deidentified personal ECG library

Acid-Base abnormalities

Rudy Moravek, PA-C
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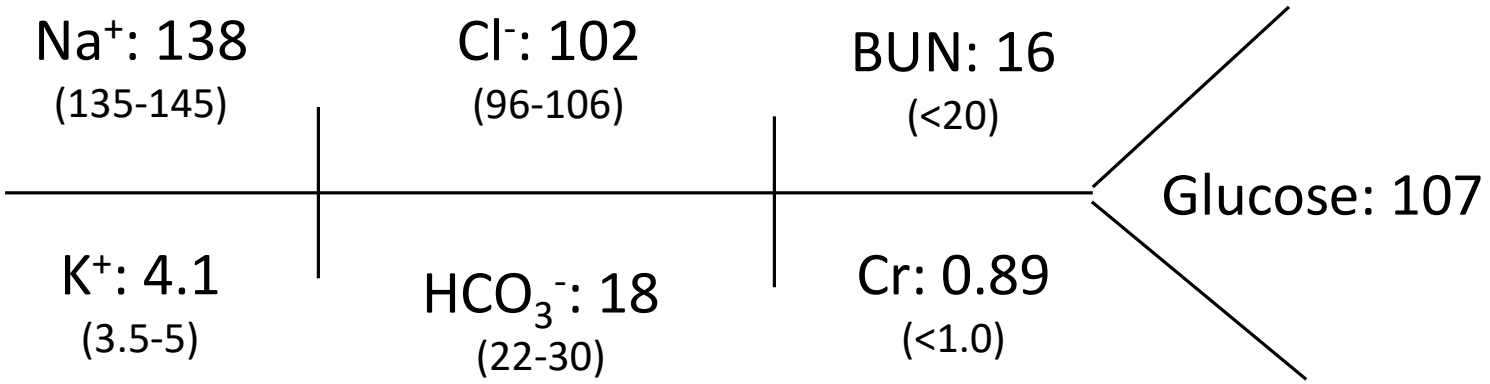
University of Colorado Anschutz School of Medicine

Learning objectives

- Have a three-step approach to assessing acid-base disorders
- Leave with an improved understanding of the Anion Gap
- Know there are several different causes of elevated lactate
- Improved comfort with formulating a differential diagnosis for various Acid-Base disorder

- As you are getting your history from T. S. Wift, you get a ”ping” on the computer that some labs are resulting. You attempt to be efficient and check the EMR…
- While you are reviewing the BMP, she claims, "Call it what you want, my body is in a glitch and I must have some bad blood"



What is the abnormality seen on the BMP?

A. Anion Gap Metabolic Acidosis
B. Metabolic Alkalosis
C. Non-anion Gap Metabolic Acidosis
D. I have no idea

Metabolic Acidosis

- Drop in systemic pH (due to increase in $[H^+]$) from one of three reasons…
  1. Increase in metabolic acids
  2. Loss of systemic bicarbonate
  3. Decreased secretion of metabolic acids from the kidneys

What test can we get to further work up the Acid-Base problem?

A. Lactate
B. Urinalysis
C. Repeat the BMP and hope for a different result
D. Arterial Blood Gas

T. S. Wift's ABG

pH: 7.31 (7.35-7.45) / CO_2: 40 (35-45) / O_2: 88 (80-100) / HCO_3^-: 18 (21-27)

What the heck to I do with this???

TRY TO KEEP IT SIMPLE!!!

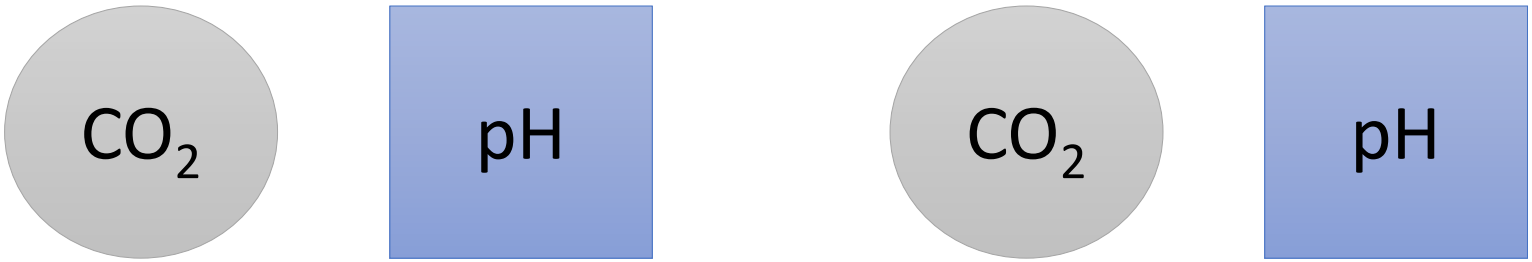
Basic approach to Acid-Base abnormalities…

Three steps using the ABG…

1. Assess for primary disorder
2. Assess for compensation
3. If the primary disorder is a metabolic acidosis, assess the anion gap

Step 1. Assess for the primary disorder

- Quick and easy…
  1. Acidosis vs Alkalosis (is the pH high/alkalotic vs low/acidotic)
  2. Look at the CO_2
     - Just remember, “when in ROME…”
       - Respiratory Opposite, Metabolic Equal



So what is our primary disorder?

pH: 7.31 (7.35-7.45)	CO_2: 40 (35-45)	O_2: 88 (80-100)	HCO_3^-: 18 (21-27)

Acidosis

Metabolic

METABOLIC ACIDOSIS

Assess for compensation

- Metabolic Acidosis: Increase in ventilation --> decrease in PCO_2
  - $PCO_2 = 1.5 \text{ X } [HCO_3\text{-}] + 8 \pm 2$ mmHg
- Metabolic Alkalosis: Decrease in ventilation --> increase in PCO_2
  - $PCO_2 = 40 + 0.7 \text{ X } (HCO_3^- - 24) \pm 5$ mmHg
- Respiratory Acidosis: Decrease in renal HCO_3^- loss --> increase in serum $[HCO_3^-]$
  - Acute: 24-48 hours: $[HCO_3^-]$ rise of 1 mEq/L per 10 mmHg rise in PCO_2 from 40
  - Chronic: >48 hours: $[HCO_3^-]$ rise of 3.5 mEq/L per 10 mmHg rise in PCO_2 from 40
- Respiratory Alkalosis: Increase in renal HCO_3^- loss --> decrease in serum $[HCO_3^-]$
  - Acute: 24-48 hours: $[HCO_3^-]$ drop of 2 mEq/L per 10 mmHg drop in PCO_2 from 40
  - Chronic: >48 hours: $[HCO_3^-]$ drop of 5 mEq/L per 10 mmHg drop in PCO_2 from 40

Is T. S. Wift compensated?

YES **NO**

- Metabolic Acidosis: $PCO_2 = 1.5 \times [HCO_3^-] + 8 \pm 2$ mmHg

pH: 7.31 (7.35-7.45)	CO_2: 40 (35-45)	O_2: 88 (80-100)	HCO_3^-: 18 (21-27)

- $PCO_2 = 1.5 \times 18 + 8$ → 35 ± 2

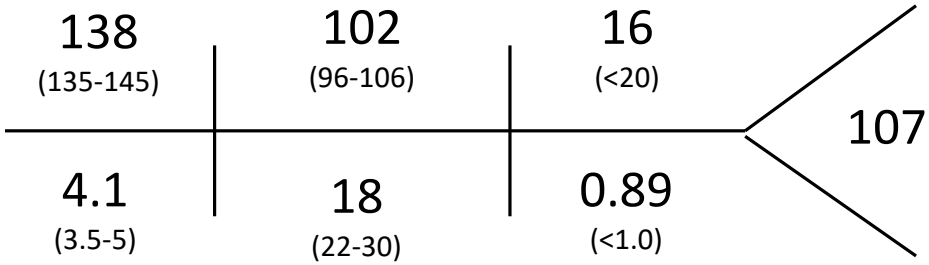
What is our Acid-Base disorder?

A. Metabolic Acidosis without Anion Gap with Respiratory Alkalosis
B. Metabolic Acidosis without Anion Gap
C. Metabolic Acidosis with Anion Gap with Respiratory Acidosis
D. Metabolic Acidosis with Anion Gap

If the primary disorder is Metabolic Acidosis, assess the anion gap

Anion Gap = $(Na^+) – [(HCO_3^-) + (Cl^-)]$

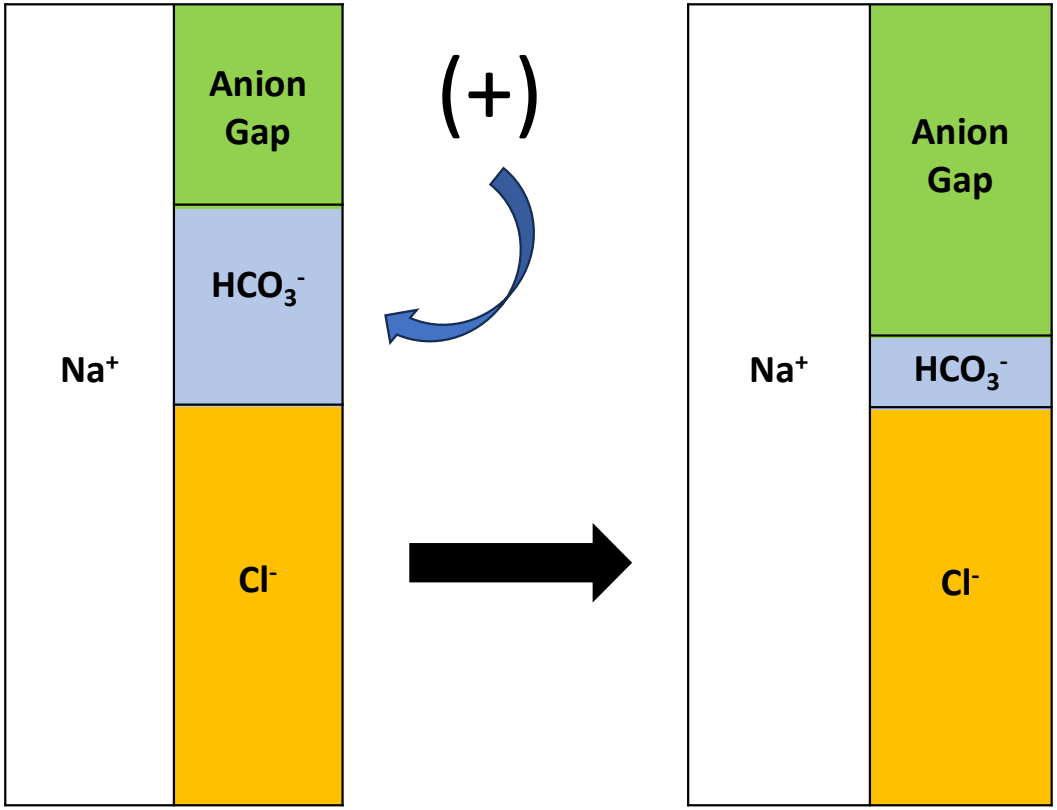
Normal range: 6-12 mmol/L



Some may add (K^+) to the (Na^+) but often this is not necessary given plasma concentration of Na^+ (135-145) compared to K^+ (3.5-5.0)

What the heck is the anion gap?!?

Unmeasured cation/acid is present which is then buffered by HCO_3^-, leading to consumption of systemic HCO_3^-



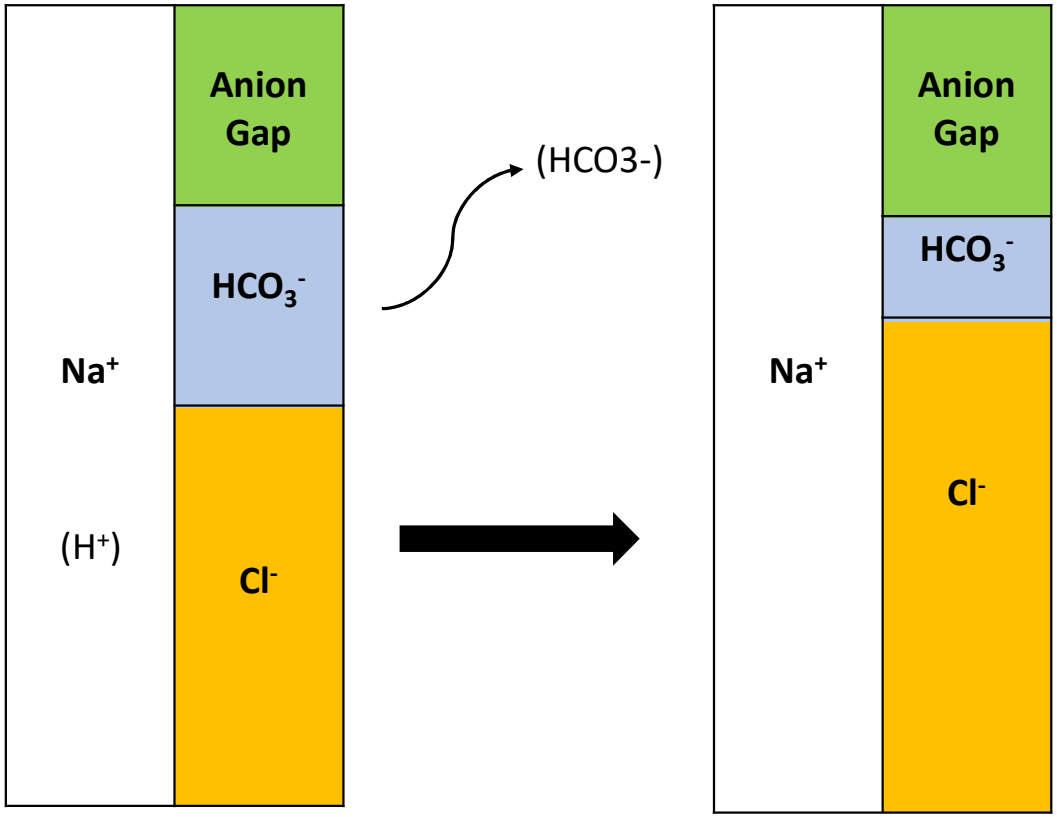
Don't forget about Albumin...

- Albumin carries a negative charge and is one of the most prominent proteins in the body. Because of this you may need to adjust your anion gap based on the albumin level
  - Rule of thumb... expected anion gap will fall 2.5 mEq/L for every 1.0 g/dL of albumin below normal

Corrected Anion Gap = (Measured Serum Anion Gap) + (2.5 x [4.5 - measured serum albumin])

Well then, what the heck is a non-anion gap?!?

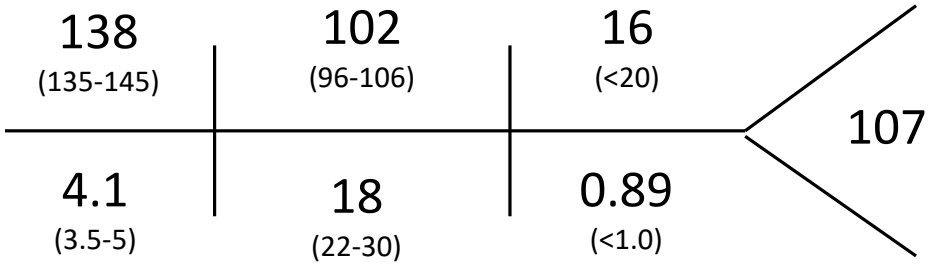
Abnormal loss of HCO_3^- or retention of H^+ causing a shifting of Cl^- to the plasma space



If the primary disorder is Metabolic Acidosis, assess the anion gap

Anion Gap = $(Na^+) – [(HCO_3^-) + (Cl^-)]$

Normal range: 6-12 mmol/L



For T. S. Wift: 138 – (18 + 102) = 18

What workup can we get to assess the etiology for an Anion Gap Metabolic Acidosis?

A. Blood cultures, urinalysis with culture, respiratory culture
B. Urinalysis, Lactate, Salicylate level, BMP
C. Troponin, BNP, Lactate
D. Blood culture, Troponin, Salicylate level

Why do we care about the Anion Gap?

It's all about the differential!

Anion Gap (MUDPILES or GOLDMARK)

Methanol	Glycols
Uremia	Oxoproline
DKA	L-lactate
Propylene Glycol	D-lactate
Isoniazid/Iron	Methanol
Lactic Acidosis	Aspirin
Ethylene Glycol	Renal failure
Salicylates	Ketosis

Non-anion Gap

Loss of HCO_3^-

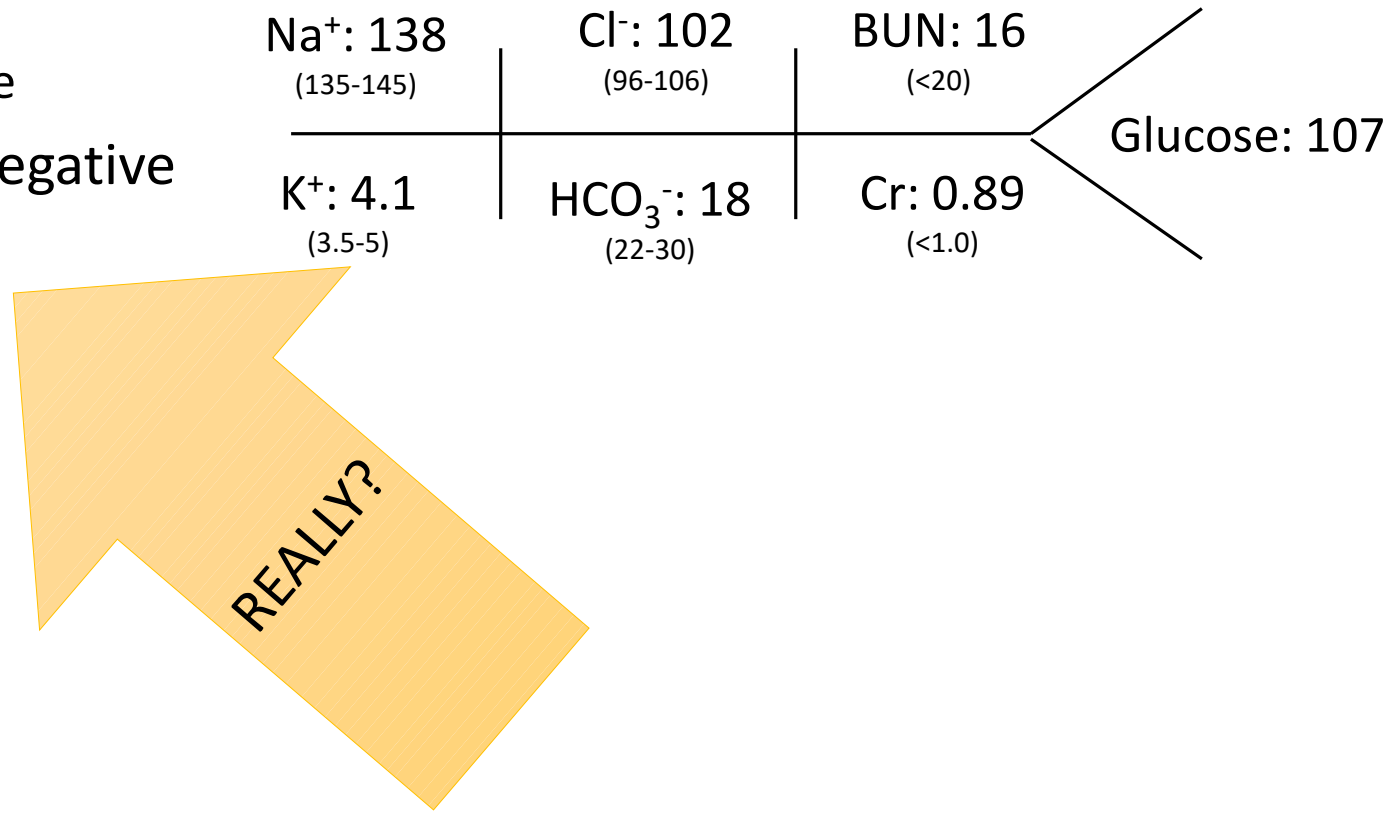
- GI loss (diarrhea)
- Drugs: Acetazolamide
- RTA Type II

Decreased secretion of H^+

- Renal failure
- RTA Type I or IV

What does our workup reveal?

- Urinalysis
  - Ketones: Negative
- Salicylate Level: Negative
- Lactate: 4.1 (<2.2)



Always good to have a differential!

Type A Lactic Acidosis

Decreased tissue perfusion leading to change in cellular metabolism to an anaerobic process

- Sepsis
- Cardiac failure
- Hypovolemia

Type B Lactic Acidosis

Impaired cellular metabolism leading to accumulation of Lactic Acid

- Diabetes/DKA
- Malignancy
- Alcohol use
- Drugs for HIV
- β_2 agonist
- Thiamine deficiency

All this on Metabolic Acidosis, what about Metabolic Alkalosis?

- Usually due either a loss of H^+, or a net gain of HCO_3^-
  - Loss of H^+
    - Diuretic therapy using Loop Diuretics
    - Excessive vomiting
    - Patient with Nasogastric Tube to suction for prolonged time
  - Gain of HCO_3^-
    - Chronic respiratory acidosis (hypercapnia)

It seems like there is still something that we have not discussed…

- Remember what our overall Acid-Base problem was???

Metabolic Acidosis with Anion Gap with Respiratory Acidosis

Respiratory Acidosis/Hypercapnia

- Accumulation of systemic CO_2 due to impair alveolar ventilation
- Thankfully, since this is a talk on Acid-Base disorders and not respiratory failure, we can be brief on this topic…

Acute vs Chronic Hypercapnia

Acute (pH low), HCO_3^- normal

- Impaired respiratory center: opioid narcosis, CVA
- Acute exacerbation of lung disease
- Chest wall injury --> splinting
- Respiratory muscle weakness
- Airway obstruction

Chronic (pH normal to low end of normal), HCO_3^- normal to high

- Most often due to COPD, other restrictive lung disease, or undertreated hypoventilation state such as OSA/OHS

Respiratory Alkalosis in a single slide…

- Often occurs acutely so may not see compensatory decrease in HCO_3^-
- Often associated with hyper-ventilatory states
  - Anxiety
  - Pain
  - Sepsis/Severe fever
  - Salicylate toxicity
  - Encephalitis
  - Can occur in ventilated patients who have respiratory rate turned up

Let's bring it all together!

pH: 7.31 CO_2: 40 HCO_3^-: 18

Based off Winter's Formula: expected CO_2 33-37 but measured was 40

Anion gap: 18

Urinalysis: negative for ketones

Salicylate level: negative

Cr: 0.89 Glucose: 107 Lactate: 4.1

Metabolic Acidosis with Anion Gap with Respiratory Acidosis

Lactic Acidosis

Type B Lactic acidosis due to β-2 agonist treatment from the continuous albuterol nebulizer

Chronic respiratory acidosis due to Obesity Hypoventilation Syndrome and untreated Obstructive Sleep Apnea

To sum it all up...

1. Keep it simple! You can have a three-step approach to acid-base disorders
   I. Determine primary disorder
   II. Assess for compensation
   III. If metabolic acidosis is present, check the anion gap
2. The anion gap evaluates if there is an unmeasured acid being buffered/consuming HCO_3^-
3. There are two types of lactic acidosis (Type A and Type B) with different etiologies
4. Knowing you to assess for the anion gap, and there are different types of lactic acidosis, help formulate a broad differential diagnosis for acid-base abnormalities

References

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Approach to Abnormal Liver Function Tests

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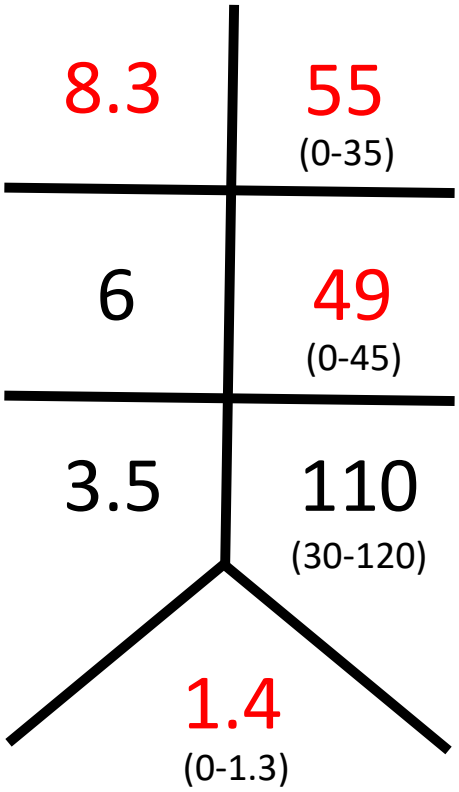
Objectives

- Cite which labs are markers of liver injury and dysfunction
- Identify the patterns of liver injury and associated differential diagnoses
- Recall common differential diagnoses for degree of AST and ALT elevation

T. S. Wift's hepatic function panel on admission was abnormal. What is your first step to pinpointing the cause of these abnormalities?

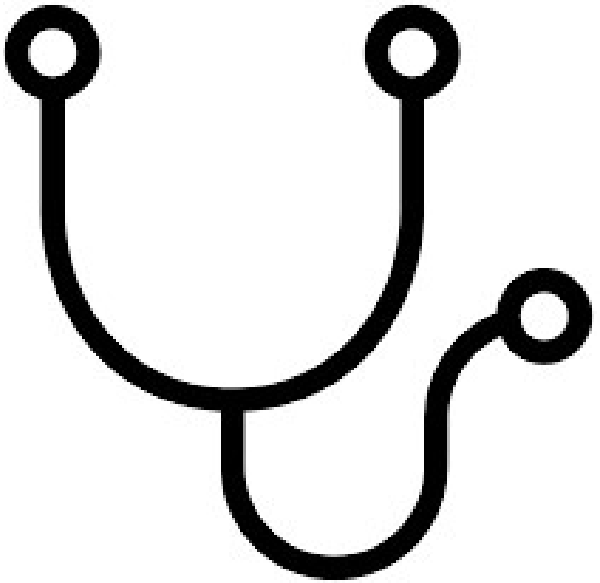
A. Grab additional labs immediately (e.g. acute viral hepatitis panel, Tylenol level, alcohol level)
B. Nothing, her AST/ALT are only mildly elevated
C. Review her history and laboratory trends
D. Obtain RUQ abdominal US



History & Physical Exam

- History
  - Medications
  - Medical history
  - Social history
  - Family history

- Physical Exam
  - Stigmata of chronic liver disease

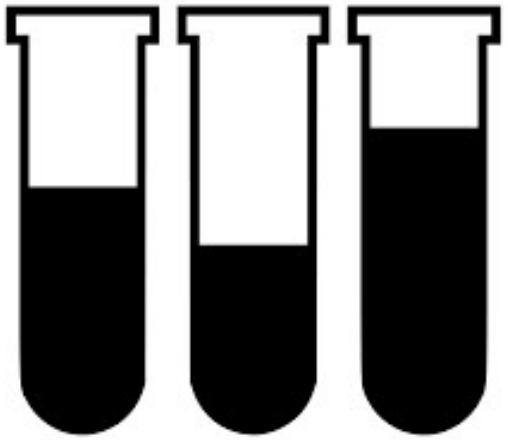


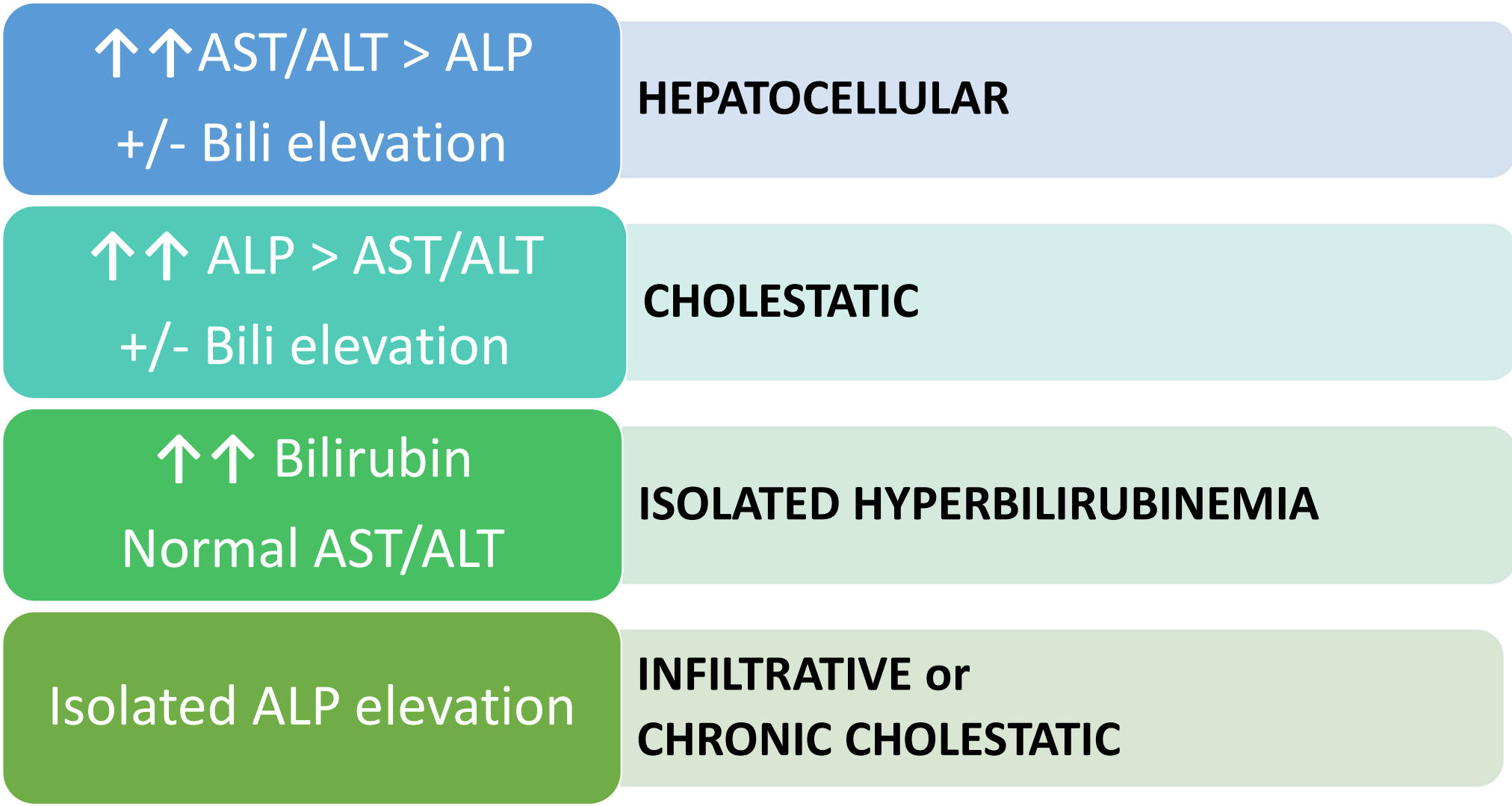
What lab values are markers of liver injury and dysfunction?

A. AST, ALT, Alk Phos
B. Bilirubin
C. Albumin and INR
D. A and B
E. All the above

Liver “Function” Tests

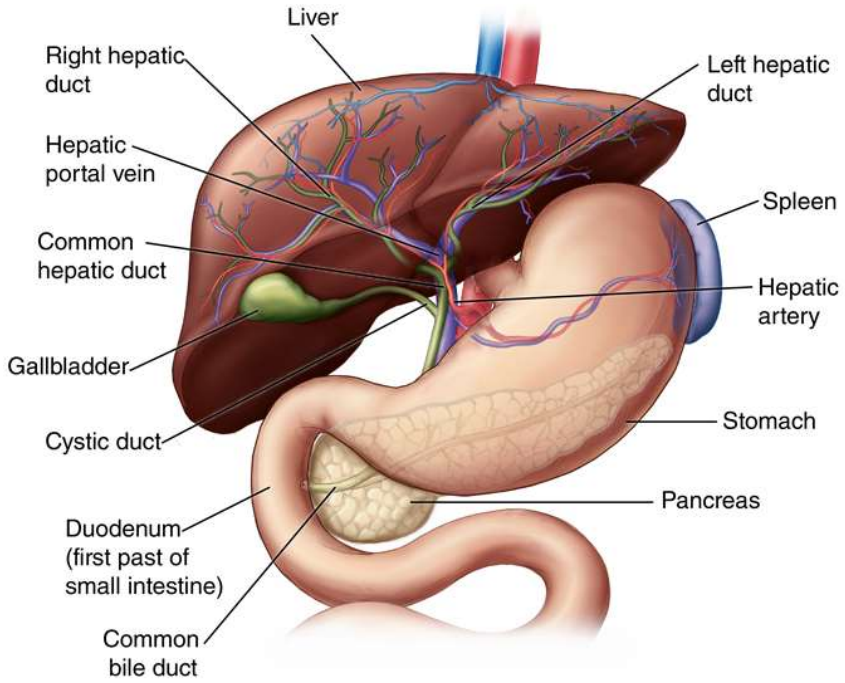
- Hepatic enzymes
  - AST
  - ALT
  - Alk Phos
- Synthetic Function
  - INR
  - Albumin
- Bilirubin
  - Unconjugated (indirect)
  - Conjugated (direct)




↑↑AST/ALT > ALP
+/- Bili elevation
HEPATOCELLULAR
↑↑ ALP > AST/ALT
+/- Bili elevation
CHOLESTATIC
↑↑ Bilirubin
Normal AST/ALT
ISOLATED HYPERBILIRUBINEMIA
Isolated ALP elevation
INFILTRATIVE or
CHRONIC CHOLESTATIC

R Factor

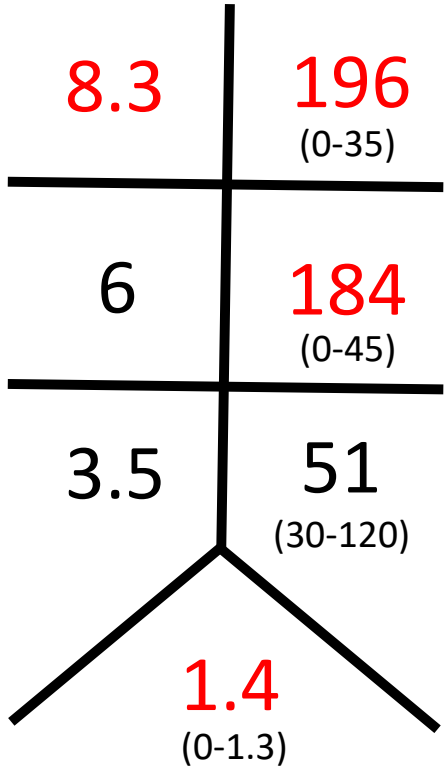
- < 2 → Cholestatic
- > 5 → Hepatocellular
- 2-5 → Mixed



Hepatocellular	Cholestatic	Hyperbilirubinemia
• Viral hepatitis • Alcohol • Drugs/Toxins • Autoimmune • NAFLD • Hereditary • Budd Chiari • Shock Liver • HELLP Syndrome	• Biliary Obstruction • Malignancy • PBC • PSC • Pregnancy • Pancreatitis	• Hemolysis • Gilbert's Syndrome • Meds • Other rare genetic conditions

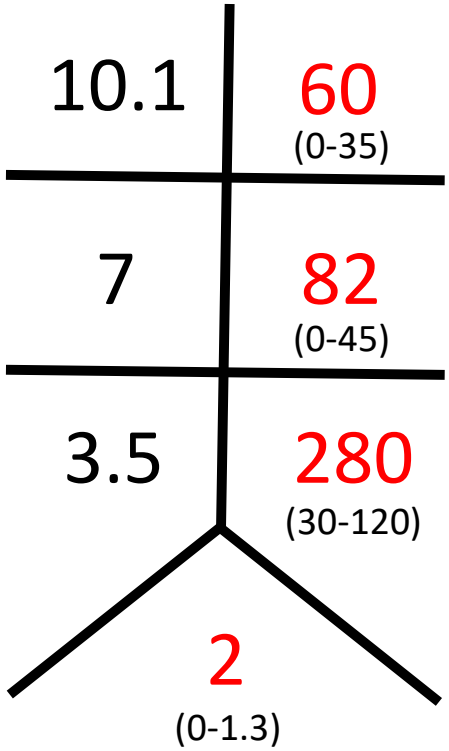
How would you describe the pattern of the following labs?

A. Hepatocellular
B. Mixed
C. Cholestatic

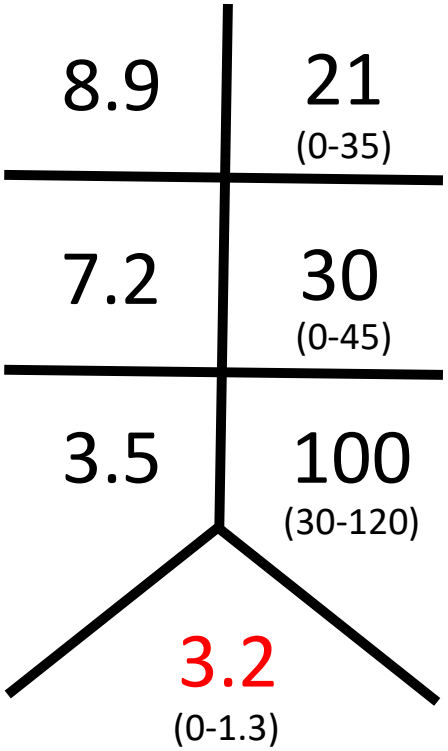


It's nice to have a friend like Hillary Stone who arrives to the hospital to visit T. S. Wift. While eating a hamburger, she develops sudden RUQ abdominal pain and goes to the ED to be evaluated. What would you expect to see on her labs?

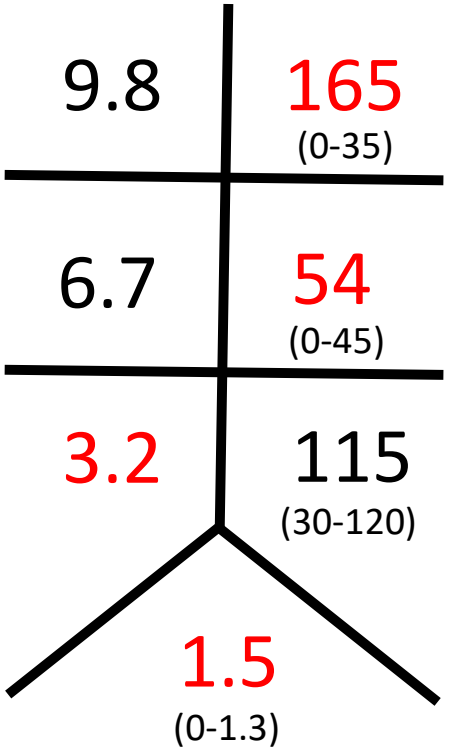
A.



B.



C.



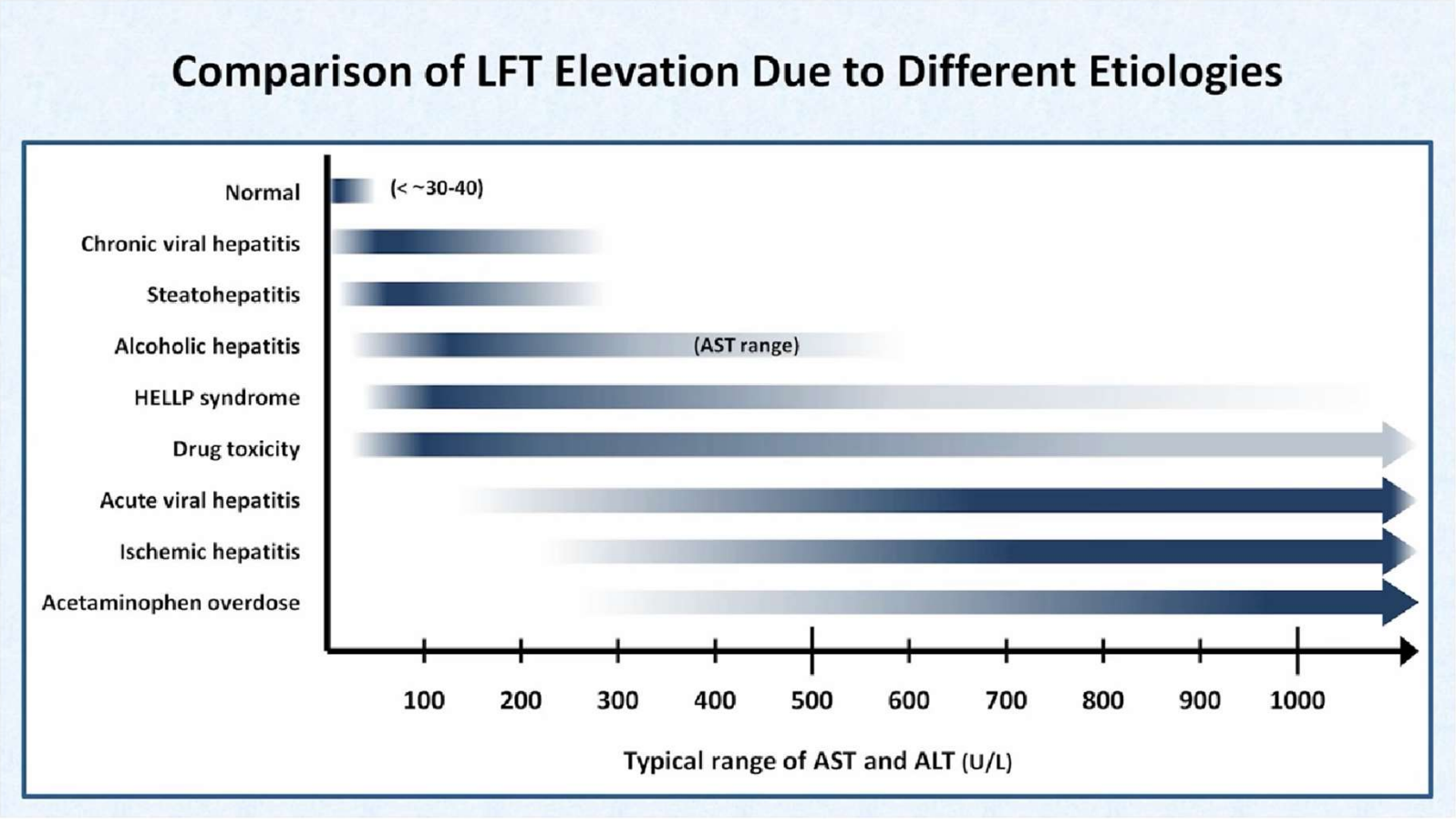
While hospitalized, T. S. Wift develops jaundice and her total bilirubin is found to be 11. What is your immediate next step?

A. Nothing this is a normal finding of chronic HCV cirrhosis
B. Fractionate total bilirubin
C. Review her medications

While completing T. S. Wift's medication reconciliation on admission, she endorses taking Tylenol 1000mg Q6H, DayQuil and NyQuil for URI symptoms and Excedrin for migraines. There is concern for Tylenol toxicity. How elevated would you expect her liver enzymes (AST, ALT) to be?

A. Less than 5 times the upper limit of normal
B. Greater than 50 times the upper limit of normal
C. Greater than 25 times the upper limit of normal

Degree of Injury



Strong, *Interpretation of LFTs* 2014

An appropriate differential for mild elevations (no more than 5x the upper limit of normal) of liver enzymes would be...

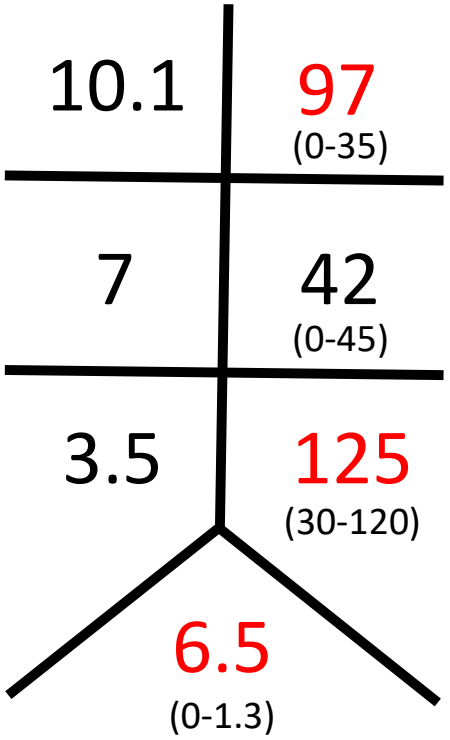
A. Chronic hepatitis, NAFLD, and cirrhosis
B. Acute viral hepatitis, ischemic hepatitis
C. Tylenol overdose, shock liver

AST/ALT Ratio Considerations

- AST : ALT > 2 → EtOH liver disease
- AST : ALT >> 5 → Not hepatic in origin (e.g. rhabdomyolysis, MI)

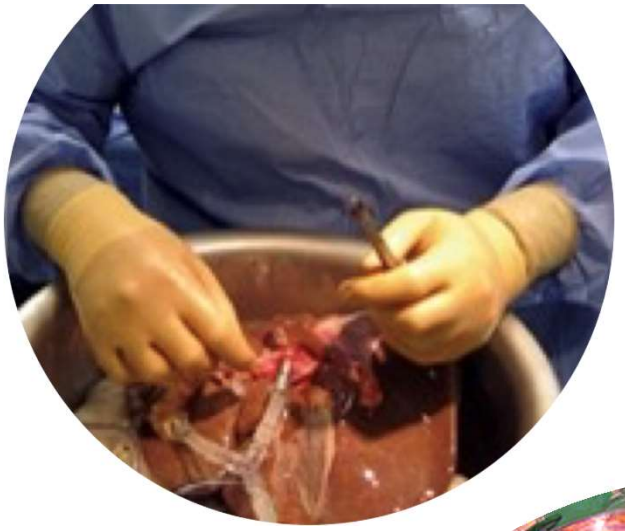
Fast forward 5 years. T. S. Wift was lost to follow up and never received treatment for her HCV. She presents with a R hepatic mass concerning for HCC. How would you describe the pattern of liver injury?

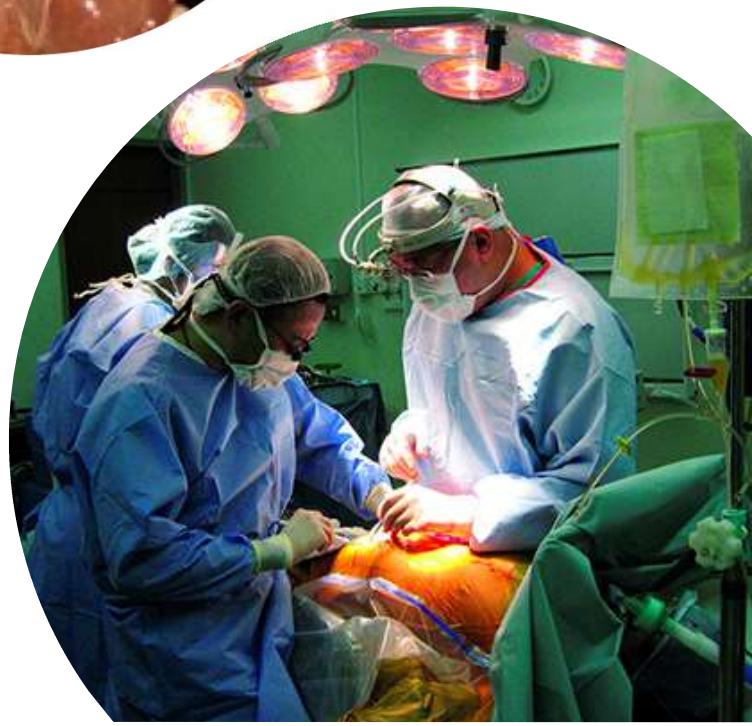
A. Hepatocellular
B. Cholestatic
C. Mixed



Acute Liver Failure

- Acute
- Elevated liver enzymes
- INR > 1.5
- Hepatic encephalopathy
- No known pre-existing liver disease





To sum it up...

1. A variety of lab values can give you indication of liver injury and/or dysfunction
   I. Injury: AST/ALT/ALP
   II. Dysfunction: INR, Albumin, Bilirubin
2. Pattern of liver injury can be divided into Hepatocellular (AST/ALT > ALP) and Cholestatic (ALP > AST/ALT)
3. Your differential for liver injury should be guided by pattern of injury and level or lab elevation

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Viral Hepatitis Serology

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Objectives

- Identify which patient populations are at high risk and should be evaluated for hepatitis A, B, and C (HAV, HBV, HCV)
- Recall which serologic tests to obtain in order to accurately diagnosis HAV, HBV, and HCV
- Interpret serologic testing for HAV, HBV, HCV

Why It's Important

- High incident of new viral hepatitis infections yearly in the US
- Causes thousands of deaths in the US annually
- Leading cause of hepatocellular carcinoma

Symptomatology

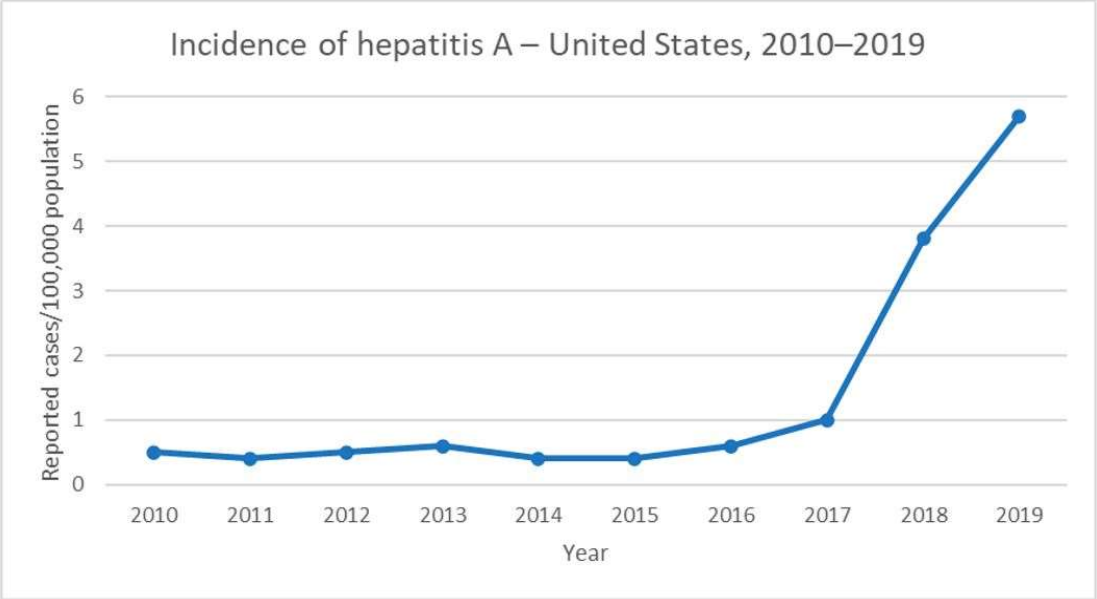
- Jaundice
- Fever
- Anorexia
- Malaise
- Nausea
- Vomiting
- Abdominal pain
- Joint pain
- Dark urine
- Clay colored stool
- Diarrhea

Who To Test

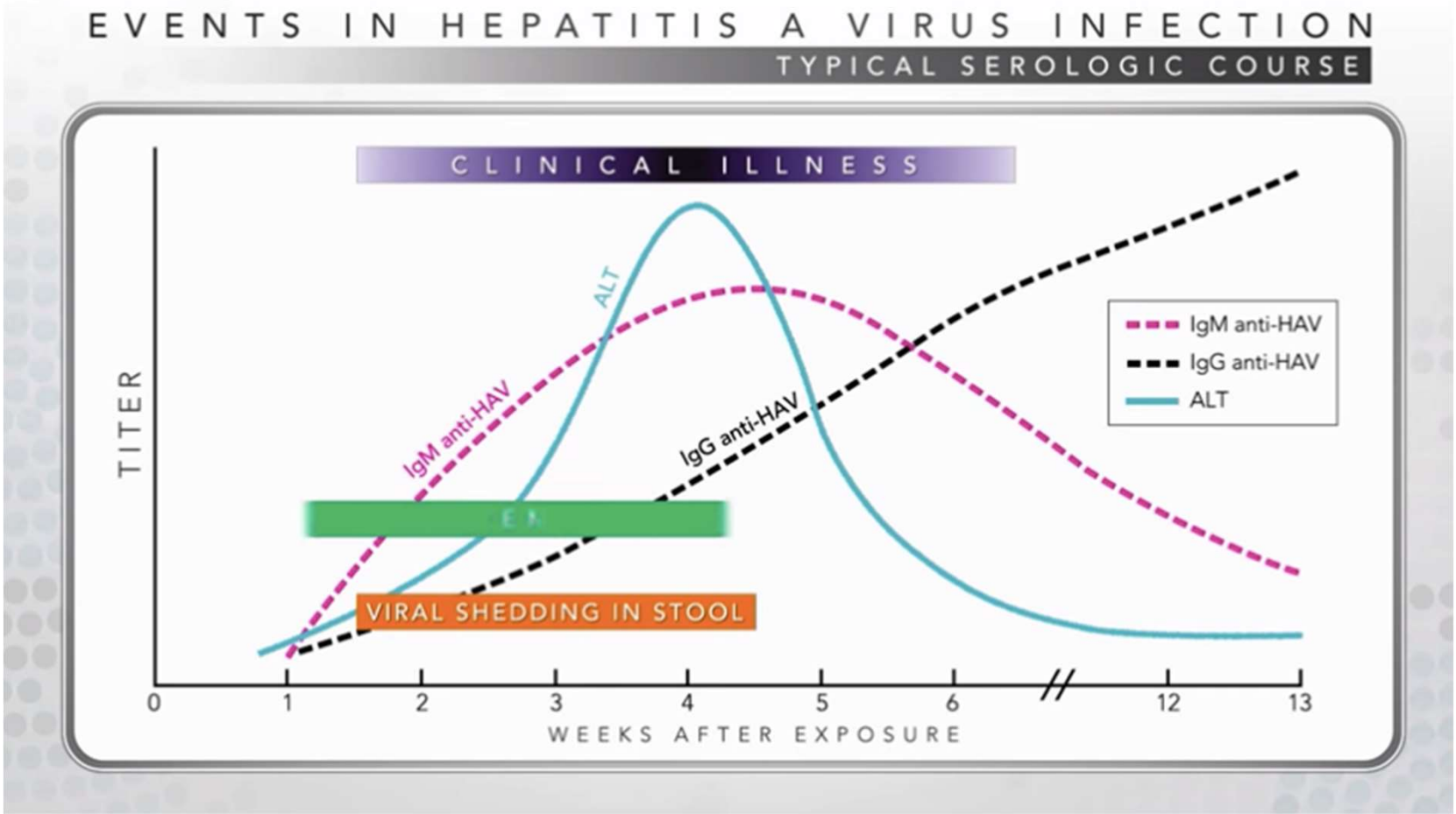
- Those who request testing or adults who have never been tested in their lifetime
- Presenting with common symptomatology
- IV Drug Use
- Experiencing homelessness
- International travel
- Incarceration
- Sexual contact history
- Pregnancy
- Infants born to a mother with HCV, HBV
- Exposure

Hepatitis A

- Transmission → fecal/oral
- Self limited, does not become chronic
- Infection = lifelong immunity
- Preventable by vaccination



(CDC, *Hepatitis A: FAQs, statistics, data & guidelines* 2020)



(CDC, *Hepatitis A: CDC Viral Serology Training* 2015)

Hepatitis A

Diagnosis	Anti-HAV	IgM	IgG
Active hepatitis A	+	+	-
Recovered hepatitis A	+	-	+

As you are obtaining T. S. Wift's history she endorses several years of humanitarian aid work in Africa when she was fearless and in her 20s. Due to presenting symptoms and newly obtained history, Ms. T. S. Wift is tested for hepatitis A.

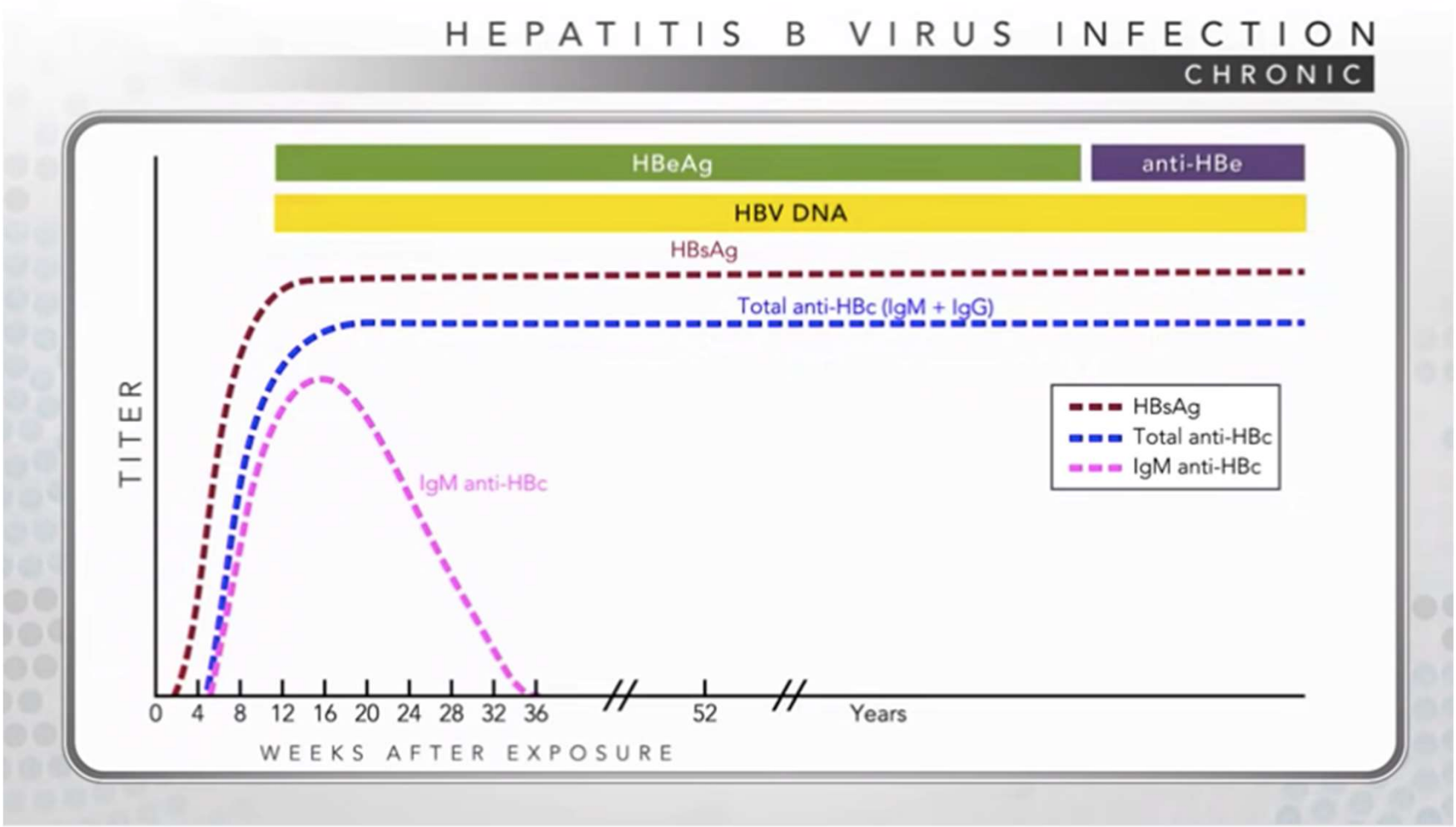
Anti-HAV + HAV IgM – HAV IgG +

Which of the following is the correct interpretation?

A. Acute hepatitis A
B. Chronic hepatitis A
C. Recovered hepatitis A
D. Lifelong immunity
E. More testing is needed
F. Both C and D

Hepatitis B

- Transmission → Person to person via bodily fluids
- Can become chronic
- Vaccine preventable



(CDC, *Hepatitis B: CDC Viral Serology Training* 2015)

Hepatitis B

Diagnosis	HBsAg	Anti-HBc	IgM Anti-HBc	Anti-HBs
Susceptible to Disease	-	-	-	-
Immune due to natural infection	-	+	-	+
Immune due to vaccination	-	-	-	+
Acutely infected	+	+	+	-
Chronically infected	+	+	-	-

Because of her known history of HCV, T. S. Wift is tested for HBV.

HBsAg – Anti-HBc + IgM Anti-HBc – Anti- HBs +

The correct interpretation of these results are:

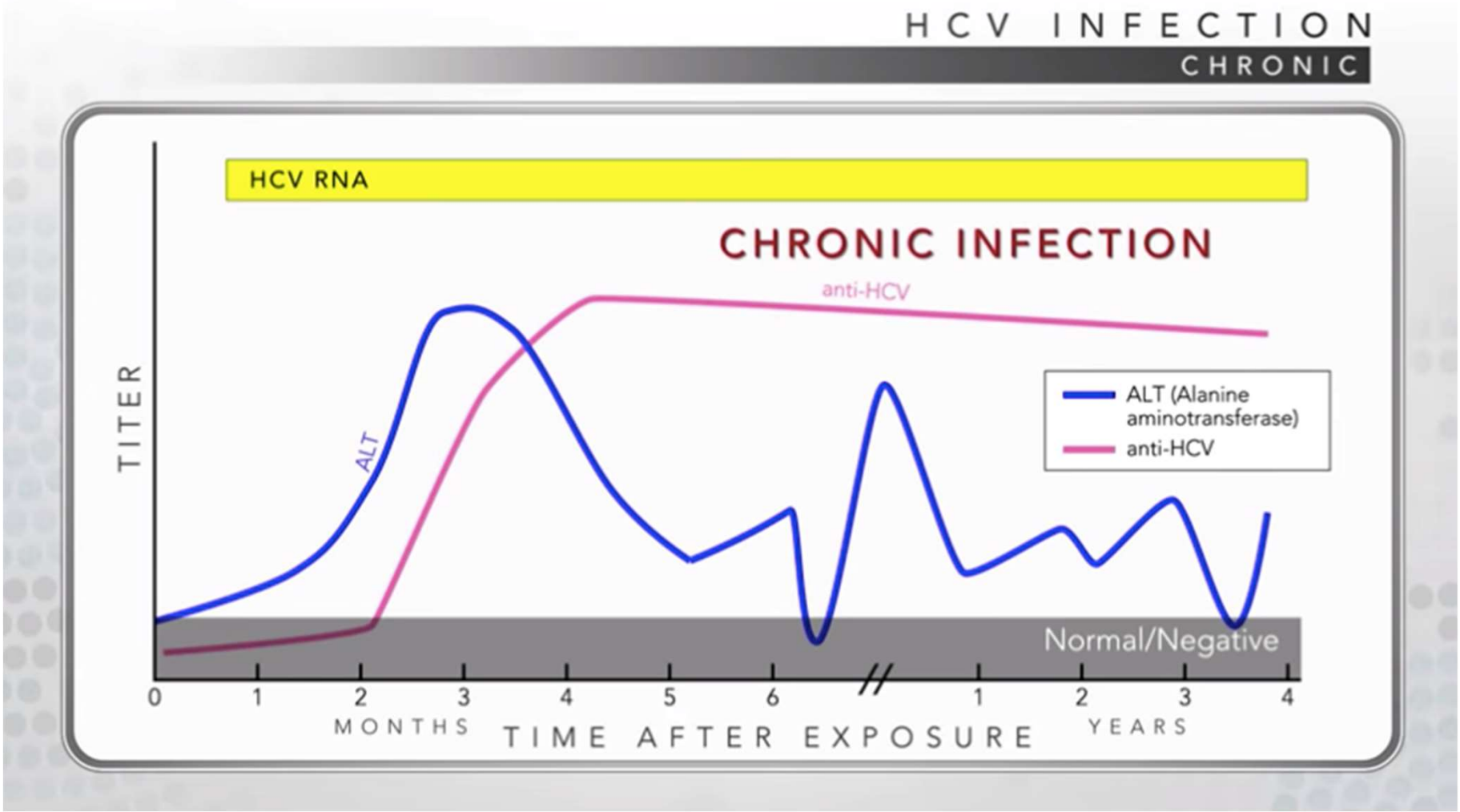
A. Previous infection, with recovery
B. Immunity from vaccination
C. Acute HBV
D. Chronic HBV

In the event T. S. Wift did not have immunity and she became acutely infected with HBV, you would expect her serologic results to be

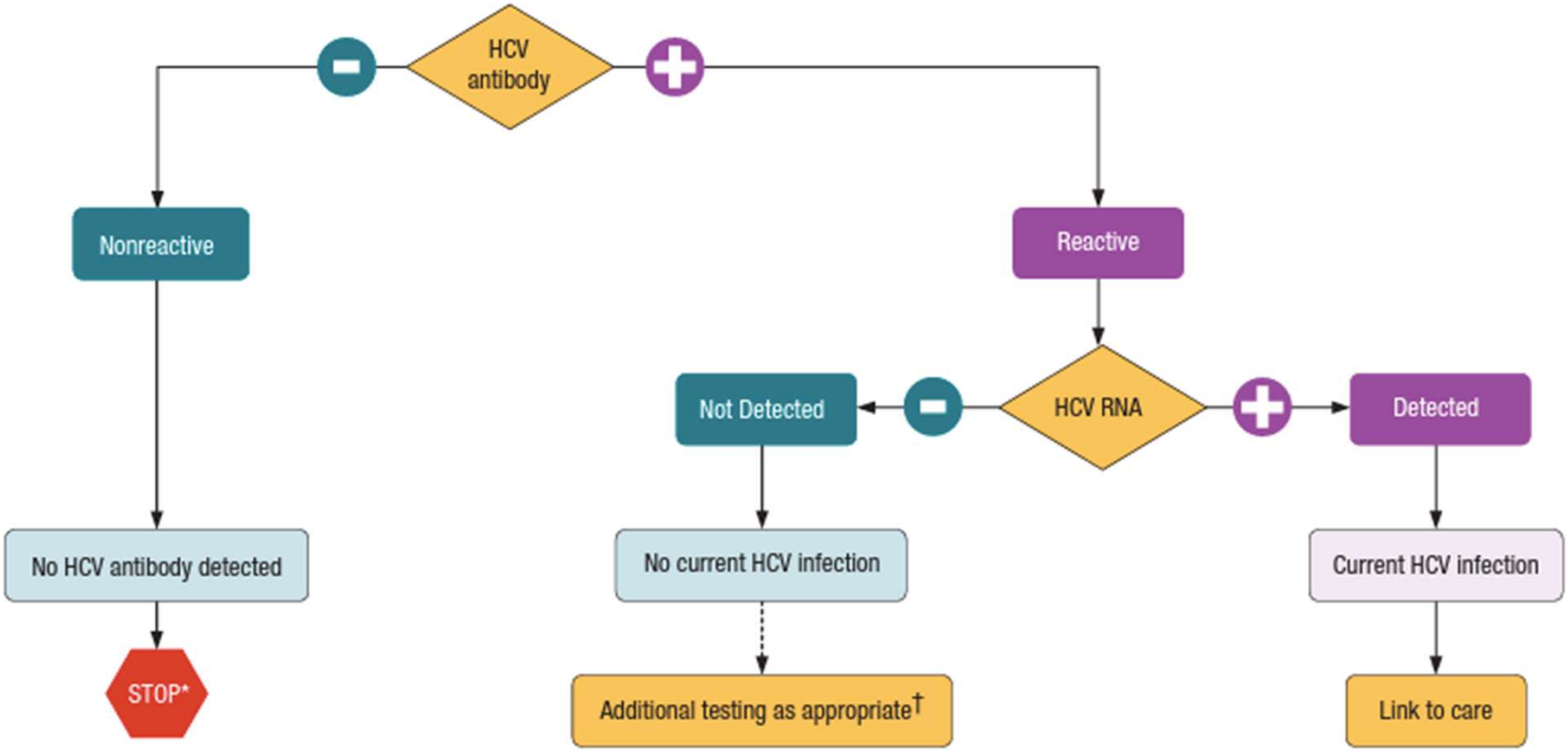
A. HBsAg + Anti-HBc + IgM – anti-HBs –
B. HBsAg + Anti-HBc + IgM + anti-HBs +
C. HBsAg + Anti-HBc + IgM + anti-HBs –

Hepatitis C

- Transmission → Blood or bodily fluids
- Acute or chronic
- Prior infection ≠ immunity
- No vaccine available
- Antiviral treatment available



(CDC, *Hepatitis C: CDC Viral Serology Training* 2015)



(CDC, *Testing for HCV Infection* 2013)

Hepatitis C

Diagnosis	Anti-HCV	HCV RNA
Active hepatitis C infection	+	+
No active infection	+	-

T. S. Wift's diagnosis of chronic HCV is confirmed by

A. We do not need confirmation, she told us she has chronic HCV
B. Anti HCV – , HCV RNA +
C. Anti HCV +
D. Anti HCV + HCV RNA +

To sum it all up...

1. Due to wide and rising prevalence, there are several factors to be considered when assessing who to test for viral hepatitis
2. Different strains of viral hepatitis require different tests
    I. HAV: Anti-HAV, IgM, IgG
    II. HBV: HBsAg, Anti-HBc, IgM Anti-HBc, Anti-HBs
    III. HCV: Anti-HCV, HCV RNA

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Thank you for your time and attention!