Session 2

Bravera Health Seven Rivers

Bravera Health Brooksville

Bravera Citrus Hills

Bravera Health Spring Hill

The INTEGRATED LIFESAVING ECG

Wayne W Ruppert, CVT, CCCC, NREMT-P
Bravera Market Regional Cardiovascular Coordinator
Bravera Health Hospitals:
Brooksville – Spring Hill – Seven Rivers

Sometimes,

ECGs

LIE to us !

ECGs and USED CAR SALESMEN often have MUCH in common !

The EKG in PERSPECTIVE

PROBLEMS WITH EKGs . . .

↓ SENSITIVITY

(FALSE NEGATIVES)

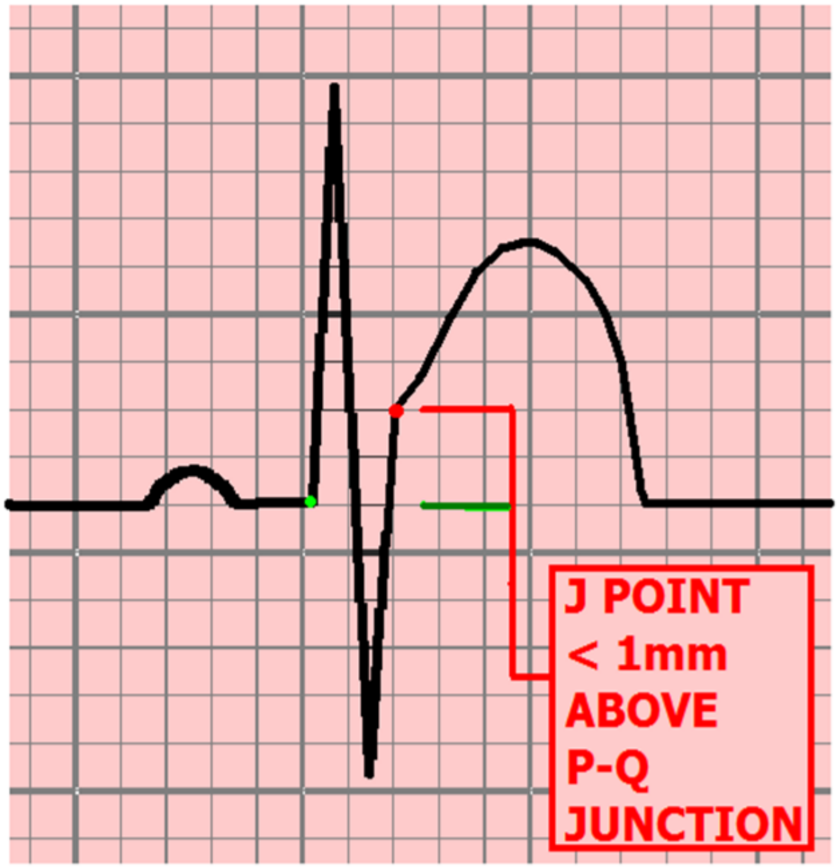
↓ SPECIFICITY

(FALSE POSITIVES)

AND . . .

PROBLEMS WITH SPECIFICITY . . .

S-T SEGMENT ELEVATION - COMMON ETIOLOGIES:



CONDITION:

- ACUTE INFARCTION
- HYPERKALEMIA
- BRUGADA SYNDROME
- PULMONARY EMBOLUS
- INTRACRANIAL BLEED
- MYOCARDITIS / PERICARDITIS
- L. VENT. HYPERTROPHY
- PRINZMETAL'S ANGINA
- L. BUNDLE BRANCH BLOCK
- PACED RHYTHM
- EARLY REPOLARIZATION & "MALE PATTERN" S-T ELEV.

ST-Segment Elevation in Normal Circumstances and in Various Conditions

Table 1. ST-Segment Elevation in Normal Circumstances and in Various Conditions.

Condition	Features
Normal (so-called male pattern)	Seen in approximately 90 percent of healthy young men; therefore, normal Elevation of 1–3 mm Most marked in V_2 Concave
Early repolarization	Most marked in V_4, with notching at J point Tall, upright T waves Reciprocal ST depression in aVR, not in aVL, when limb leads are involved
ST elevation of normal variant	Seen in V_3 through V_5 with inverted T waves Short QT, high QRS voltage
Left ventricular hypertrophy	Concave Other features of left ventricular hypertrophy
Left bundle-branch block	Concave ST-segment deviation discordant from the QRS
Acute pericarditis	Diffuse ST-segment elevation Reciprocal ST-segment depression in aVR, not in aVL Elevation seldom >5 mm PR-segment depression
Hyperkalemia	Other features of hyperkalemia present: Widened QRS and tall, peaked, tented T waves Low-amplitude or absent P waves ST segment usually downsloping
Brugada syndrome	rSR' in V_1 and V_2 ST-segment elevation in V_1 and V_2, typically downsloping
Pulmonary embolism	Changes simulating myocardial infarction seen often in both inferior and anteroseptal leads
Cardioversion	Striking ST-segment elevation, often >10 mm, but lasting only a minute or two immediately after direct-current shock
Prinzmetal's angina	Same as ST-segment elevation in infarction, but transient
Acute myocardial infarction	ST segment with a plateau or shoulder or upsloping Reciprocal behavior between aVL and III

Wang K et al. N Engl J Med 2003;349:2128-2135

The NEW ENGLAND JOURNAL of MEDICINE

77 Years Male 7/2/2015 9:44:46

1North (06)

Rate	83	. SINUS RHYTHM.....................................normal P axis, V-rate 50- 99
		. RIGHT BUNDLE BRANCH BLOCK...........................QRSd>120, terminal axis(90,270)
PR	152	. ANTEROLATERAL INFARCT, ACUTE.........................Q >35mS, ST >0.20mV, V2-V6
QRSD	122	
QT	412	
QTc	485	

--AXIS--

P	59
QRS	14
T	33

12 Lead; Standard Placement

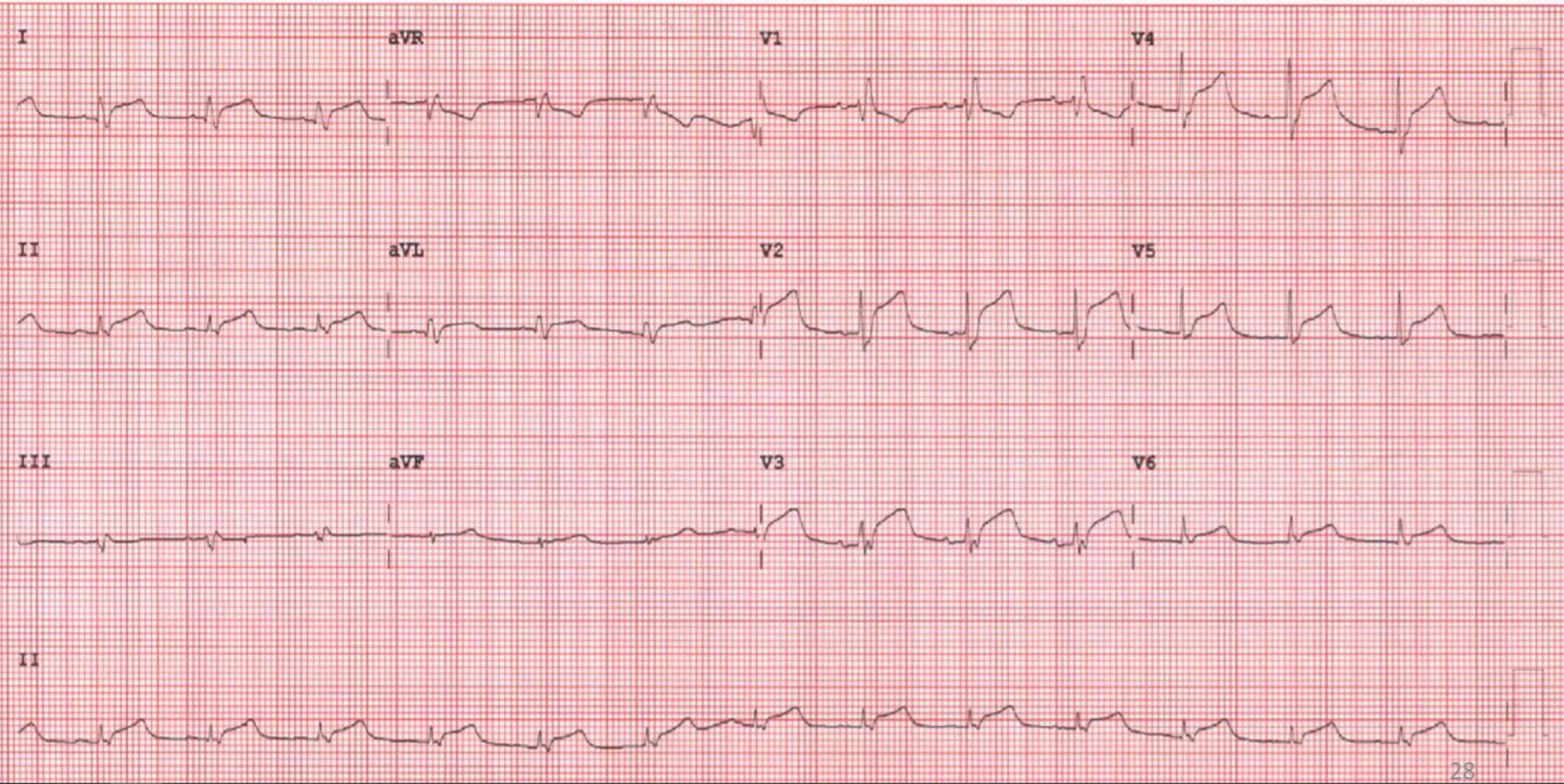
- ABNORMAL ECG -

>>> Acute MI <<<

Requested by:

Unconfirmed Diagnosis

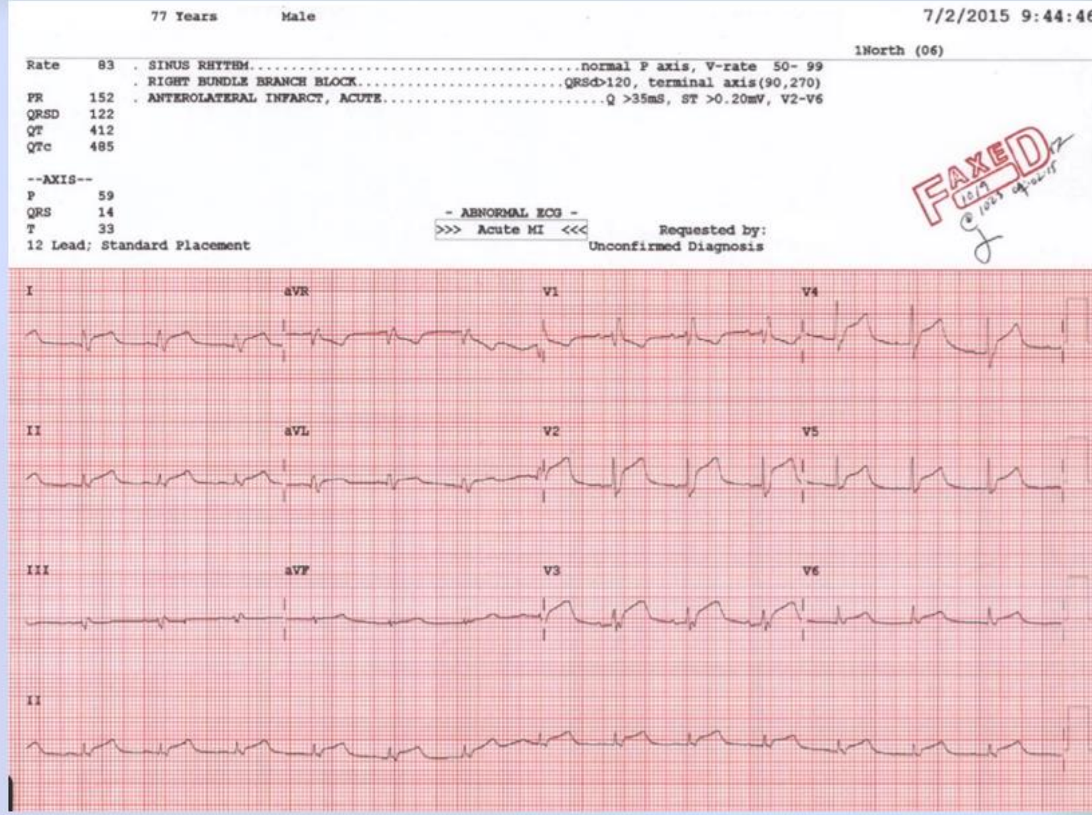
FAXED 1019 @ 1023 07.02.15



28

Patient:

- **Asymptomatic**
- **Troponin normal**
- **Cardiac Cath angiography = “no obstructive CAD.”**
- **Discharge diagnosis:**



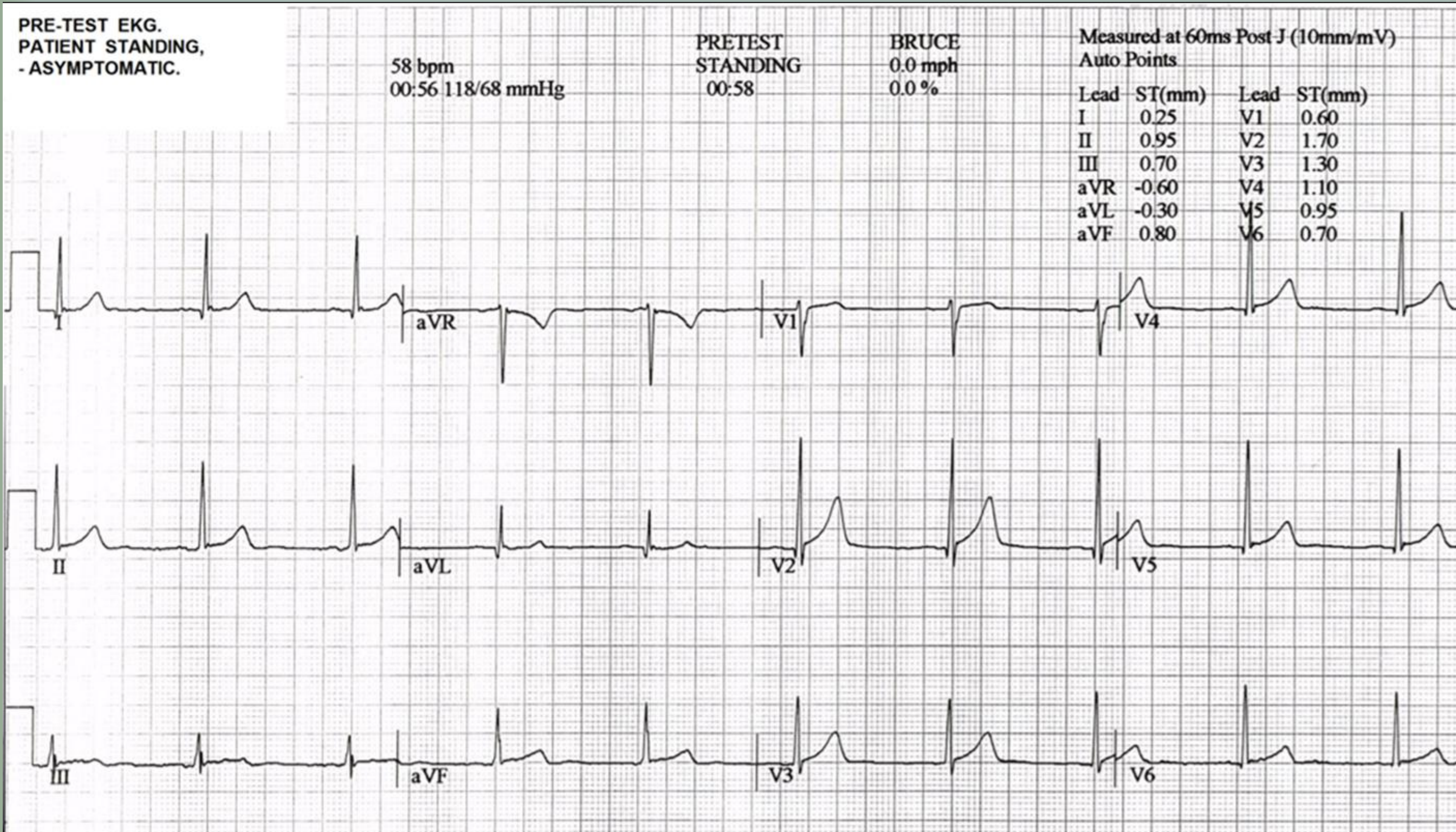
EARLY REPOLARIZATION. This degree of ST Elevation in early repolarization is VERY RARE: The only such ECG I have seen in approximately 13,000 cardiac catheterizations.

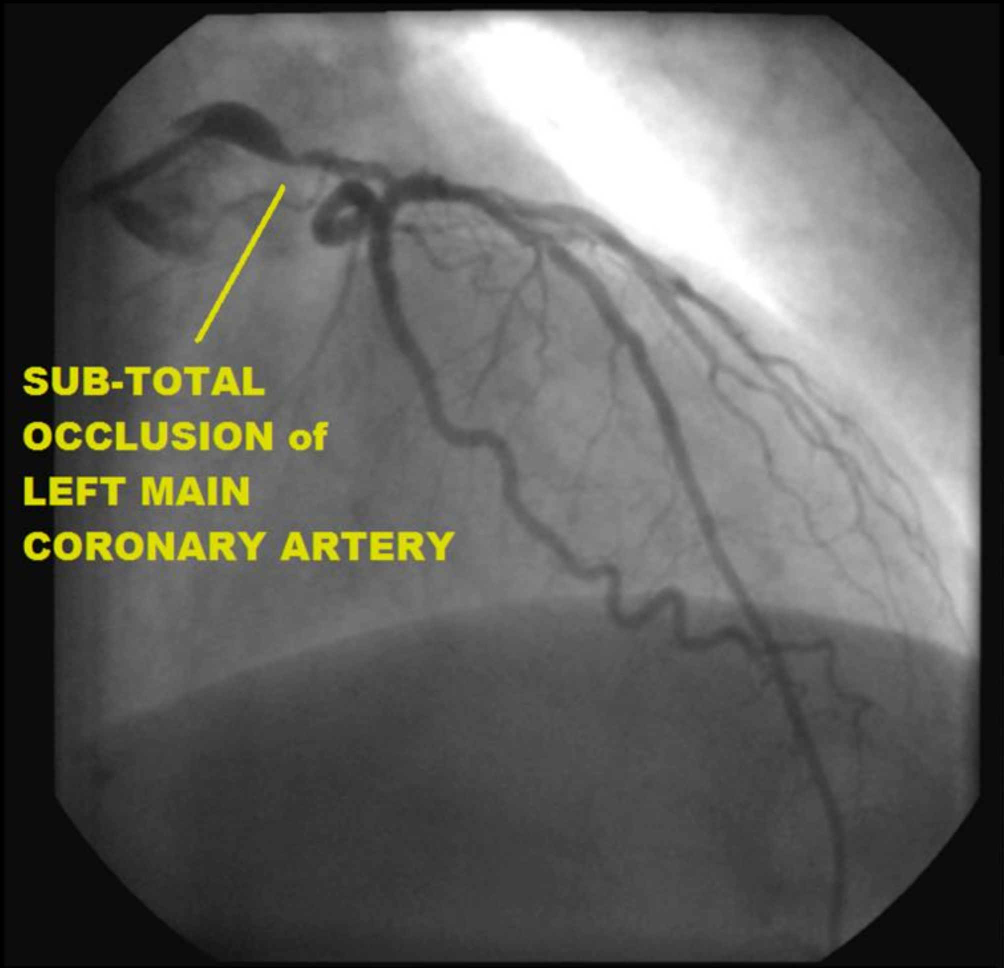
EKGs in PERSPECTIVE, con't:

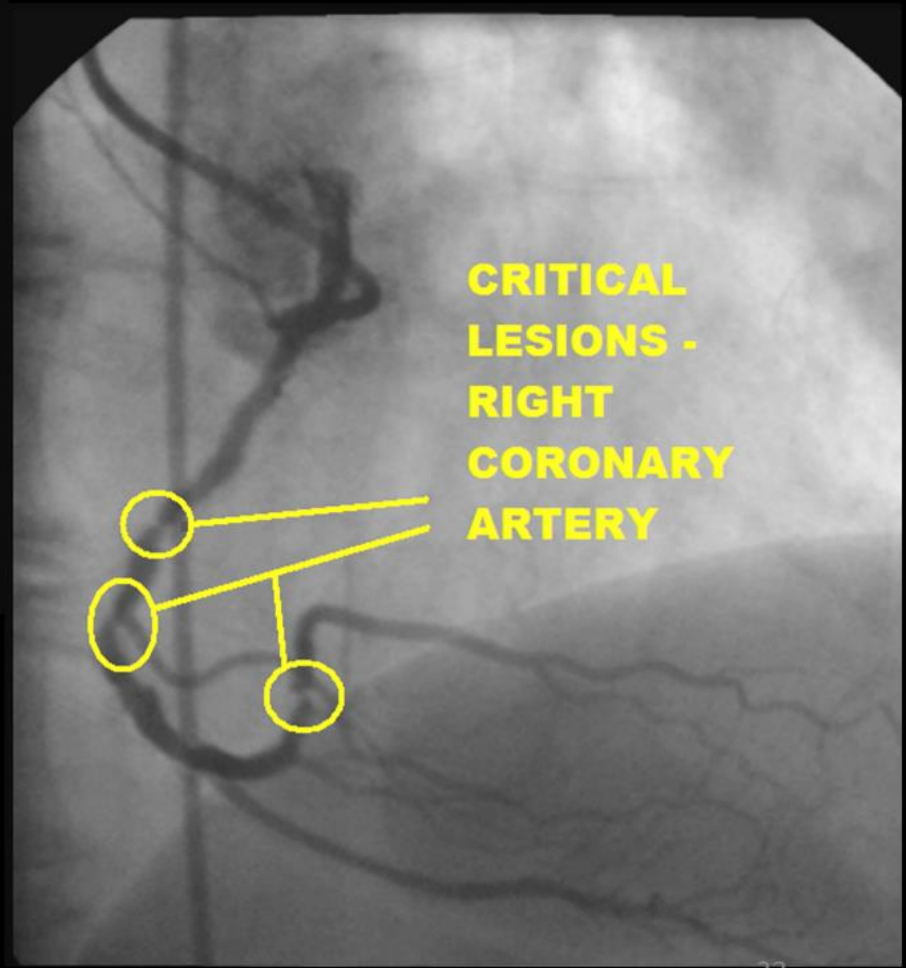
One of the MOST MISLEADING scenarios of all is when the EKG APPEARS PERFECTLY NORMAL . . .

. . . but MASKS serious, LIFE - THREATENING CONDITIONS.

that is why YOU must do a THOROUGH PATIENT EVALUATION . . . and have a HIGH INDEX OF SUSPICION ! ! !


PRE-TEST EKG.
PATIENT STANDING,
- ASYMPTOMATIC.
58 bpm
00:56 118/68 mmHg
PRETEST
STANDING
00:58
BRUCE
0.0 mph
0.0 %
Measured at 60ms Post J (10mm/mV)
Auto Points
Lead ST(mm) Lead ST(mm)
I 0.25 V1 0.60
II 0.95 V2 1.70
III 0.70 V3 1.30
aVR -0.60 V4 1.10
aVL -0.30 V5 0.95
aVF 0.80 V6 0.70
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

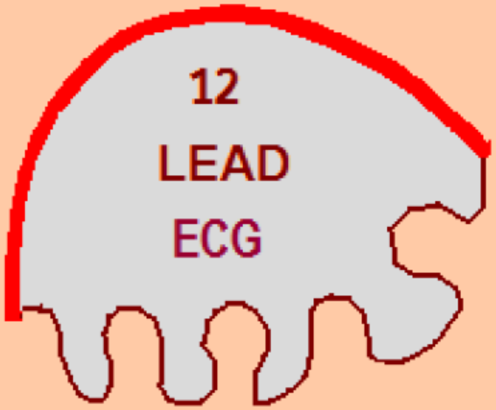

SUB-TOTAL
OCCLUSION of
LEFT MAIN
CORONARY ARTERY


CRITICAL
LESIONS -
RIGHT
CORONARY
ARTERY

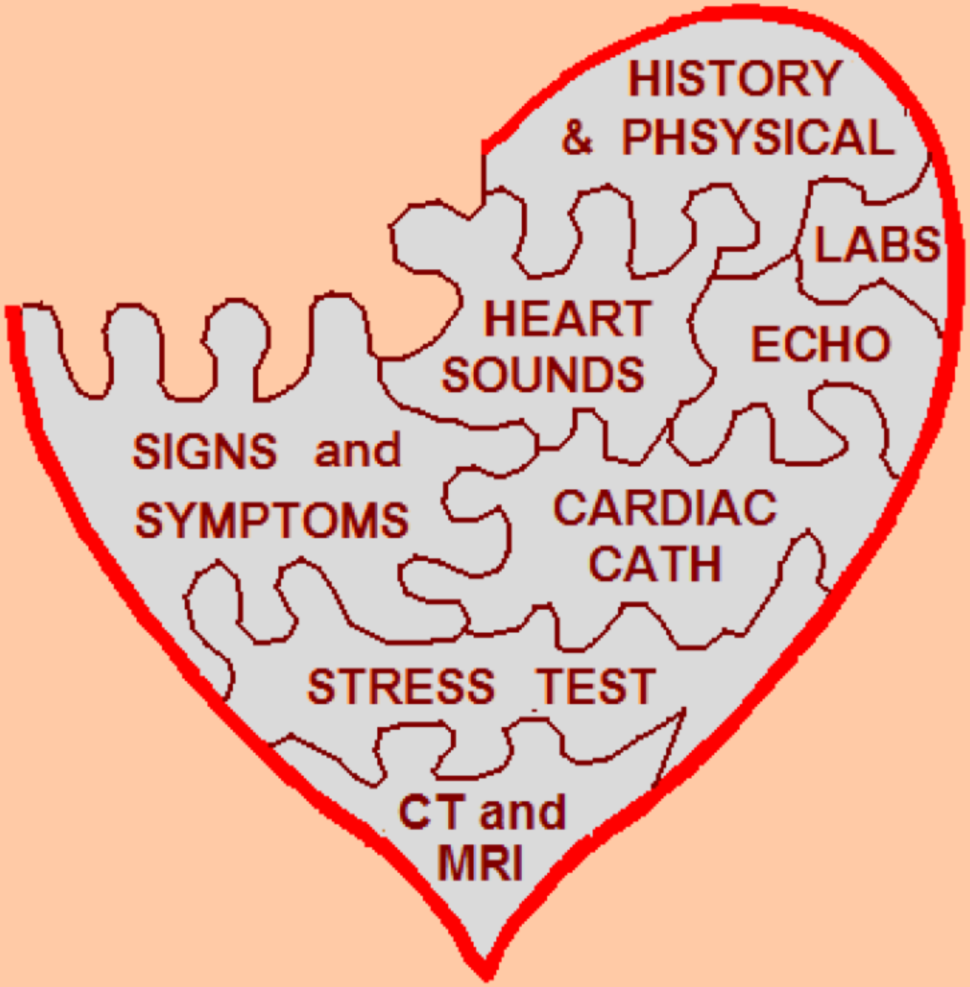
"From time to time, the EKG – derived diagnosis will be ***TOTALLY INCORRECT**."*

Despite the ECG's problematic issues with
Lack of Sensitivity
&
Lack of Specificity,
The 12 Lead ECG remains one of our QUICKEST, most cost-efficient front-line Triage Tools that we have today.

REMEMBER . . . . Keep the ECG Results in PROPER PERSPECTIVE . . . .



REMEMBER . . .
it's only
ONE PIECE
of the
DIAGNOSTIC
PUZZLE !



So how do we know when the ECG is telling us the truth ???

- ***We utilize ACS Risk Stratification*** ***to compensate for the ECG's lack of sensitivity and specificity, to aid us in clinical decision-making and to improve our diagnostic accuracy.***

HEART

HEART score for chest pain patients			
History	Highly suspicious	2	
	Moderately suspicious	1	
	Slightly suspicious	0	
ECG	Significant ST-deviation	2	
	Non specific repolarisation disturbance / LBTB / PM	1	
	Normal	0	
Age	≥ 65 years	2	
	> 45 and < 65 years	1	
	≤ 45 years	0	
Risk factors	≥ 3 risk factors or history of atherosclerotic disease*	2	
	1 or 2 risk factors	1	
	No risk factors known	0	
Troponin	≥ 3x normal limit	2	
	> 1 and < 3x normal limit	1	
	≤ 1x normal limit	0	
		Total	

***Risk factors for atherosclerotic disease:**

Hypercholesterolemia
Hypertension
Diabetes Mellitus
Cigarette smoking
Positive family history
Obesity

C-Statistic scores achieved in this study:

HEART: 0.83

TIMI: 0.75

GRACE: 0.70

C-Statistic interpretation:

A score of "1.00" would mean the score predicts outcome with 100% perfection. A score of 0.50 is the same as a "50/50 coin toss." A score of LESS THAN 0.50 means that the score predicts the opposite outcome.

US HEART Score Validation

- 1,070 observation unit patients at Wake Forest
- ☞ *Out performed clinician gestalt !*

Mahler et. al, Crit Path Cardiol, 2011
Mahler et. al, Int J Cardiol, 2013

App Store Preview

Open the Mac App Store to buy and download apps.

HEART Pathway 12+

Chest pain. Risk-stratified.

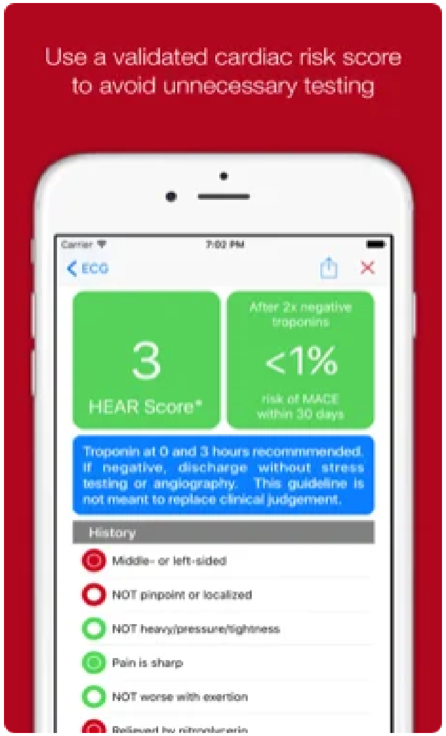
Impathiq

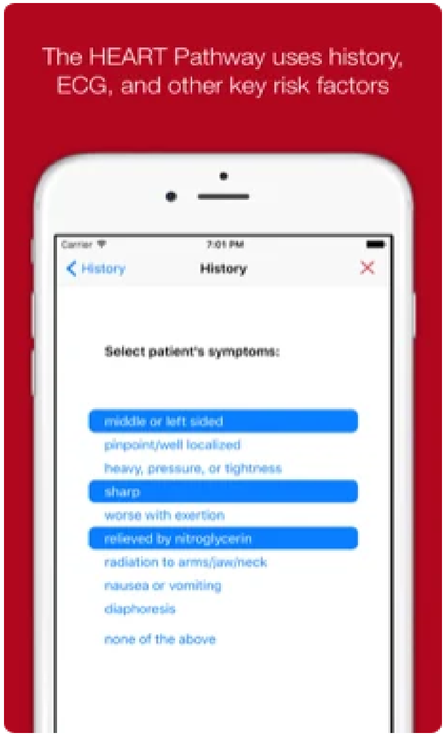
Designed for iPhone

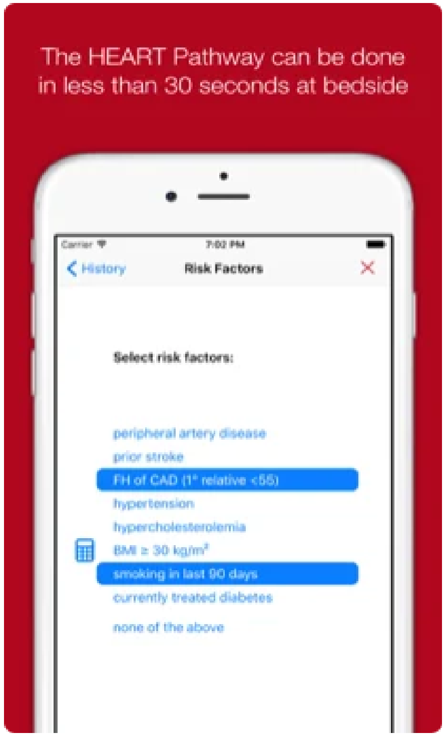
★★★★★ 4.5 • 13 Ratings

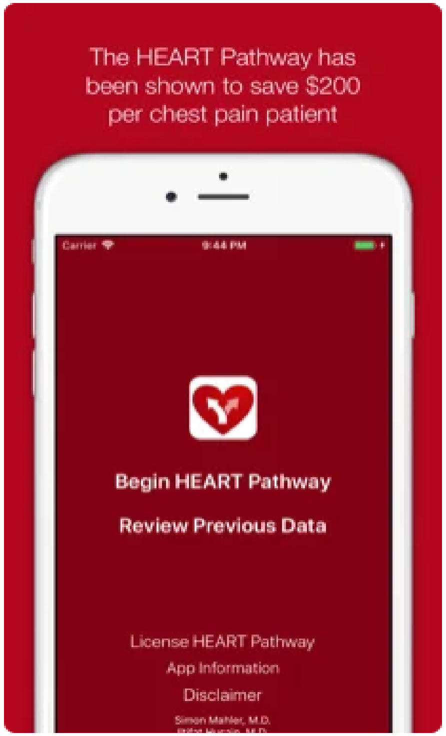
Free

iPhone Screenshots





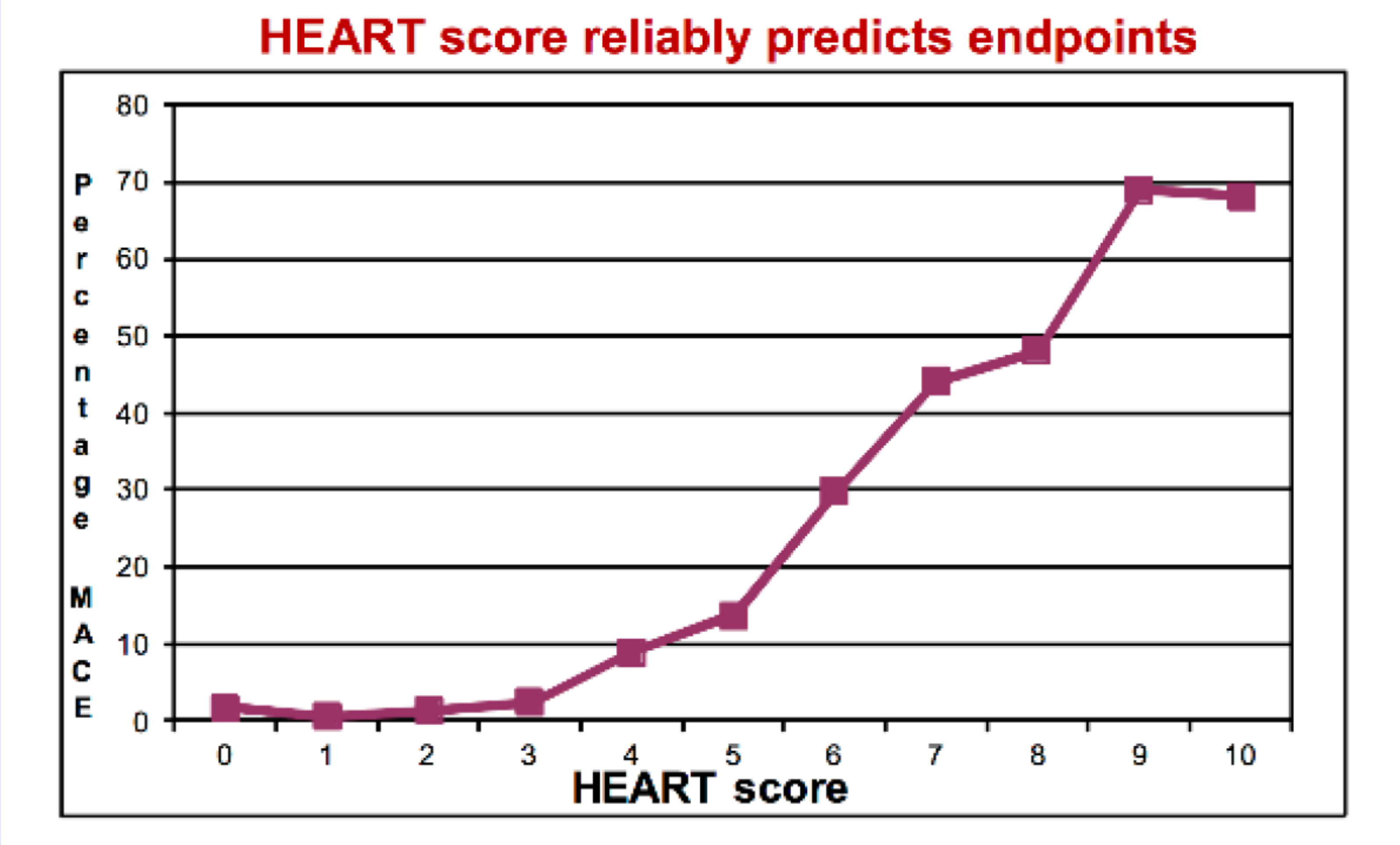




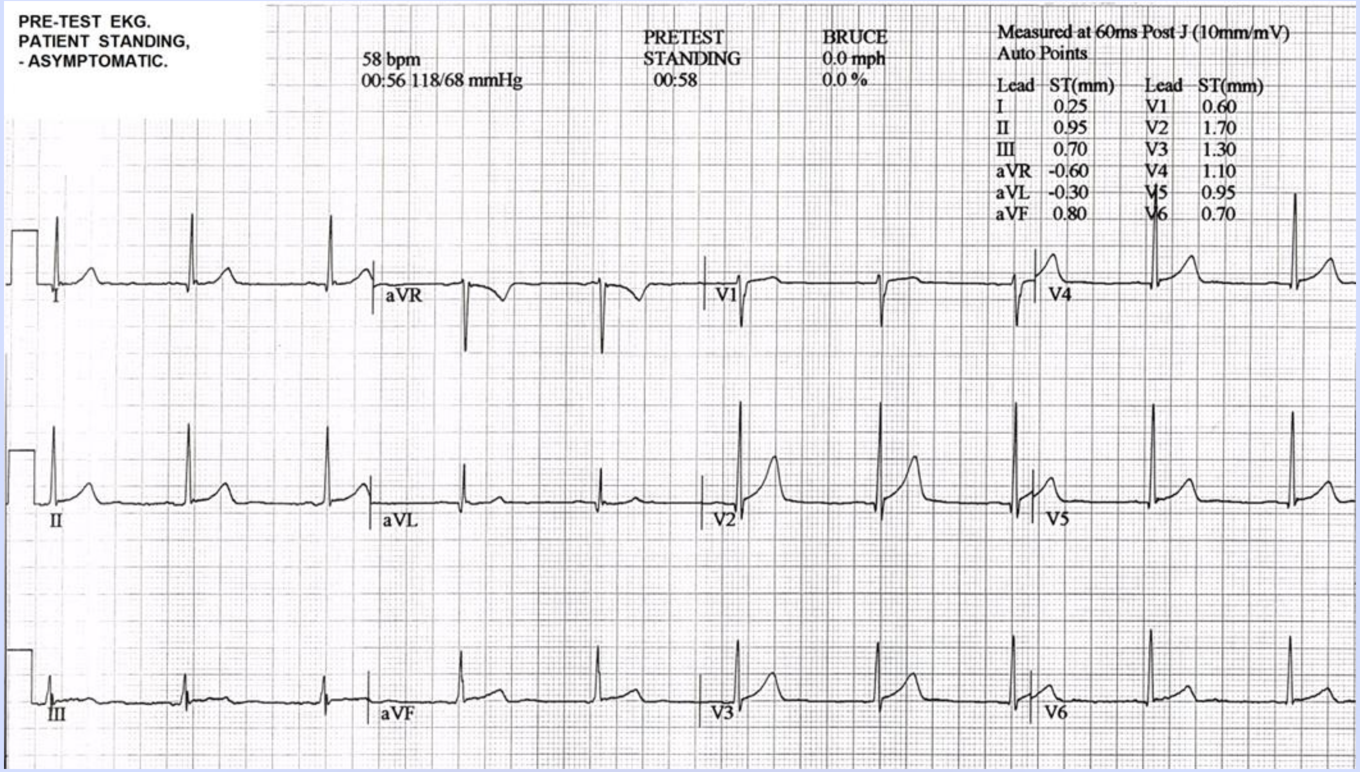
The HEART Score

Score	Common Diagnosis:	Disposition:
0-3	Low Risk Chest Pain	Early Discharge with referral
4-6	Low Risk Chest Pain Unstable Angina	Observation Unit or Admission Tele
7-10	Unstable Angina NSTEMI STEMI	Tele Admission ICU Admission STAT Cath Lab

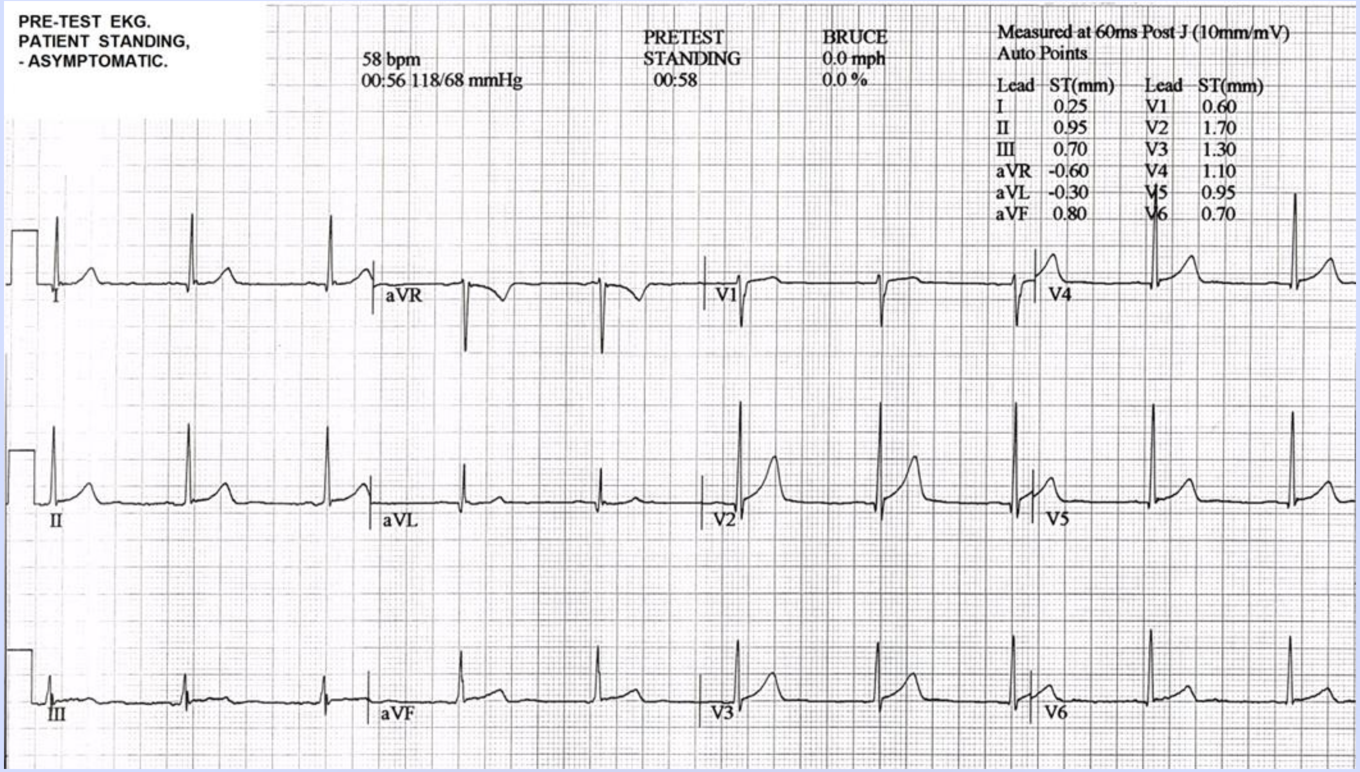
Heart Score Reliability



63 year old male complains of upper abdominal and chest pressure described as "indigestion"



63 year old male complains of upper abdominal and chest pressure described as “indigestion”



Send him home with a referral to see a cardiologist??

HEART

HEART score for chest pain patients			
History	Highly suspicious	2	
	Moderately suspicious	1	
	Slightly suspicious	0	
ECG	Significant ST-deviation	2	
	Non specific repolarisation disturbance / LBTB / PM	1	
	Normal	0	
Age	≥ 65 years	2	
	> 45 and < 65 years	1	
	≤ 45 years	0	
Risk factors	≥ 3 risk factors or history of atherosclerotic disease*	2	
	1 or 2 risk factors	1	
	No risk factors known	0	
Troponin	≥ 3x normal limit	2	
	> 1 and < 3x normal limit	1	
	≤ 1x normal limit	0	
		Total	

***Risk factors for atherosclerotic disease:**

Hypercholesterolemia	Cigarette smoking
Hypertension	Positive family history
Diabetes Mellitus	Obesity

H = HISTORY

- **<u>2 Points</u>**: "Suspicious" = Typical ACS Symptoms
- **<u>1 Point</u>**: "Moderately Suspicious" = Atypical ACS Symptoms
- **<u>0 Points</u>**: No Typical or Atypical Symptoms of ACS

E = ECG

- **<u>2 Points:</u>** ST Deviation (elevation or depression at the J point of 0.5mv or more)
- **<u>1 Point:</u>** Non-specific ST-T wave abnormalities / Non
- **<u>0 Points</u>**: Normal ECG

A = Age

- **<u>2 Points:</u>** Age 65 or more
- **<u>1 Point:</u>** Age 46 – 64
- **<u>0 Points</u>**: Age 45 or less

R = Risk Factors for CAD

- **2 Points:** 3 or more risk factors
- **1 Point:** 1 or 2 risk factors
- **0 Points**: No Risk Factors

RISK FACTORS

for the development of

CORONARY ARTERY DISEASE:

- HEREDITY
- ↑ LDL and ↓ HDL CHOLESTEROL PROFILES
- SMOKING
- DIABETES MELLITUS
- OBESITY
- PHYSICAL INACTIVITY
- HYPERTENSION
- AGE - OVER 65
- MALE
- HIGH STRESS

per the AMERICAN HEART ASSOCIATION

RISK FACTORS: Family history of CAD, elevated cholesterol, hypertension (3 Risk factors)

T = Troponin

- **2 Points:** 3 X Normal (> 0.056)
- **1 Point:** **>**1 - <3 (0.017 – 0.056)
- **0 Points**: up to normal limit (< 0.017)

HEART

HEART score for chest pain patients			
History	Highly suspicious	2	
	Moderately suspicious	1	
	Slightly suspicious	0	
ECG	Significant ST-deviation	2	
	Non specific repolarisation disturbance / LBTB / PM	1	
	Normal	0	
Age	≥ 65 years	2	
	> 45 and < 65 years	1	
	≤ 45 years	0	
Risk factors	≥ 3 risk factors or history of atherosclerotic disease*	2	
	1 or 2 risk factors	1	
	No risk factors known	0	
Troponin	≥ 3x normal limit	2	
	> 1 and < 3x normal limit	1	
	≤ 1x normal limit	0	
		Total	

***Risk factors for atherosclerotic disease:**

Hypercholesterolemia	Cigarette smoking
Hypertension	Positive family history
Diabetes Mellitus	Obesity

H = chest pain = 2

E = ECG normal = 0

A = 63 = 1

R = 3 risk fctors = 2

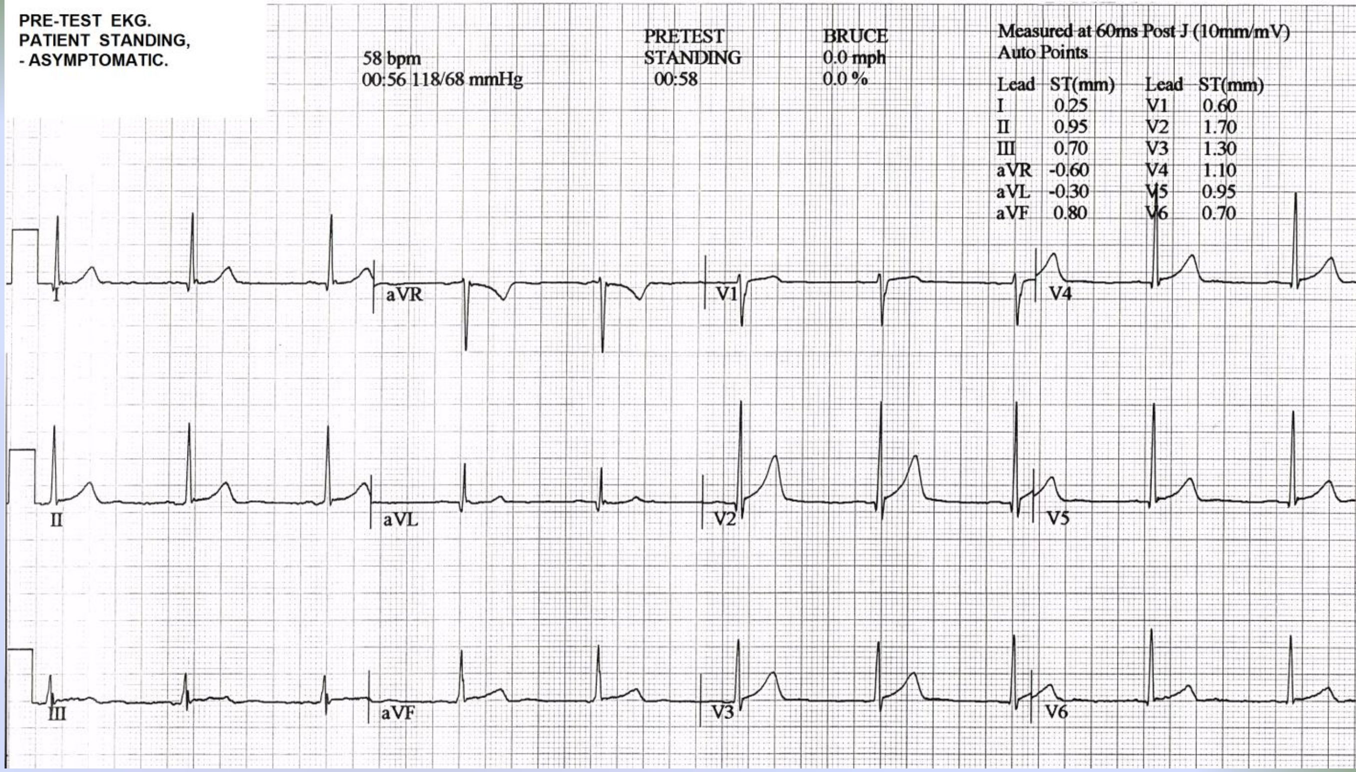
T = Trop. NL = 0

HEART Score: = 5

PROBLEMS WITH SENSITIVITY . . .

NORMAL ECG.

But . . . . .



His HEART Score = 5

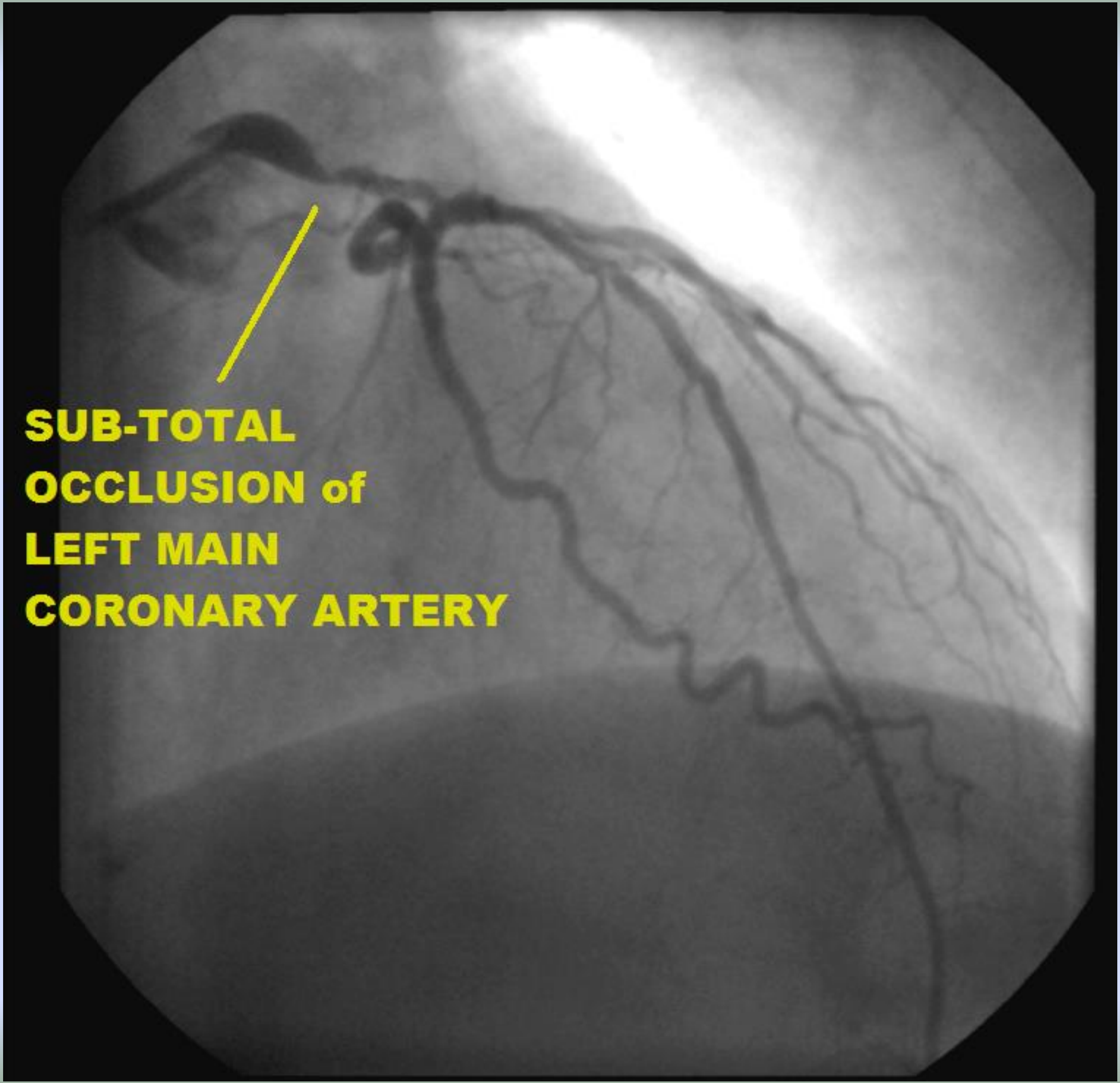
What could that possibly mean?

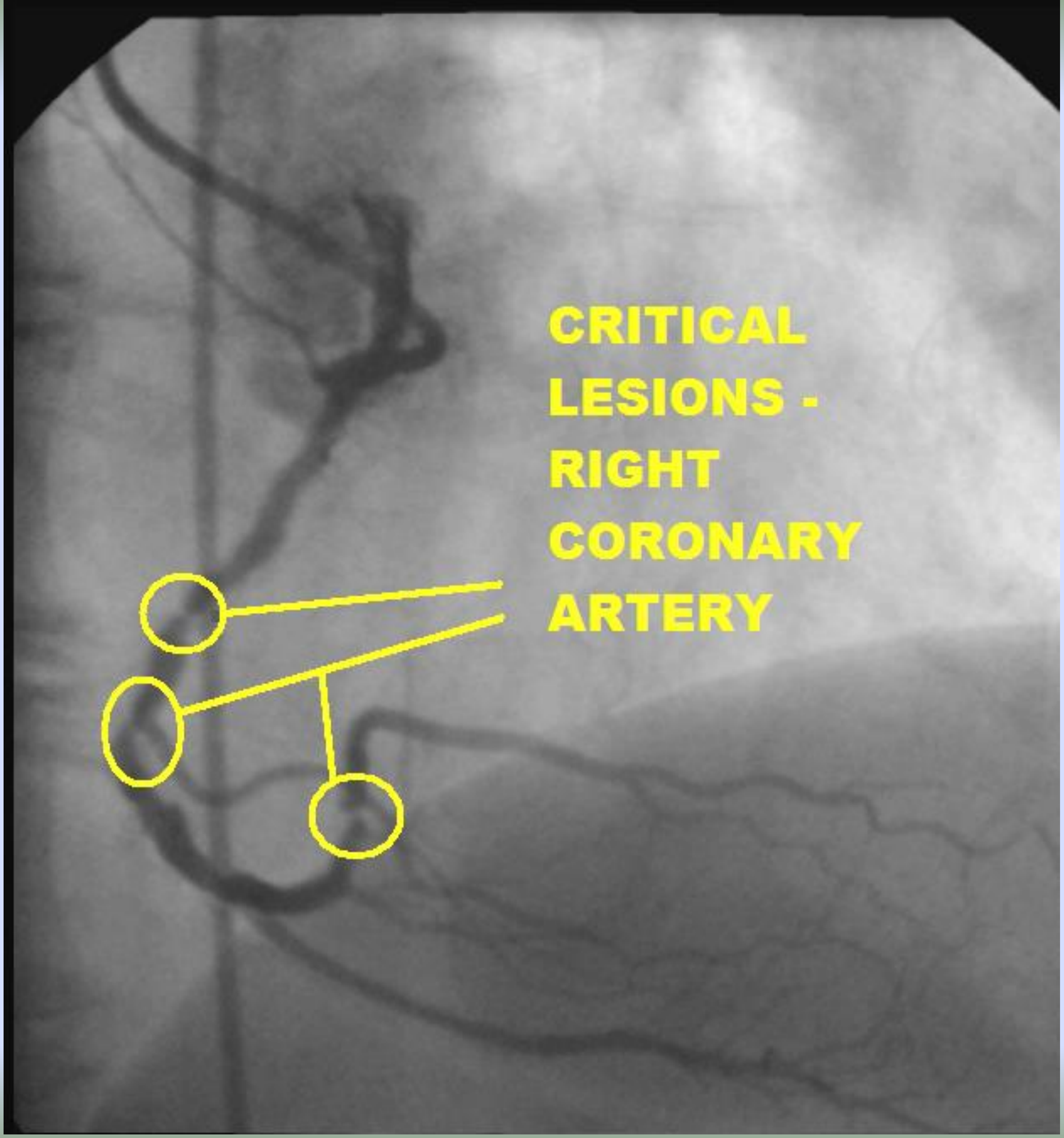
HEART Score	RISK	ACS Dx?	Proposed Management
0 - 3	LOW	Non-ACS	Discharge with follow-up / out-patient stress
4 - 6	Intermed.	Suspect: ACS, Obstructive CAD, Unstable Angina NSTEMI	Admit to hospital, Serial ECGs /Troponins aggressive diagnositic work-up (e.g. Cardiac Cath, CT coronary angio
7 - 10	HIGH	NSTEMI STEMI	STEMI= STAT PCI or thrombolytics. NSTEMI = "urgent" Cardiac Cath

http://www.heartscore.nl/

Based on HEART SCORE:

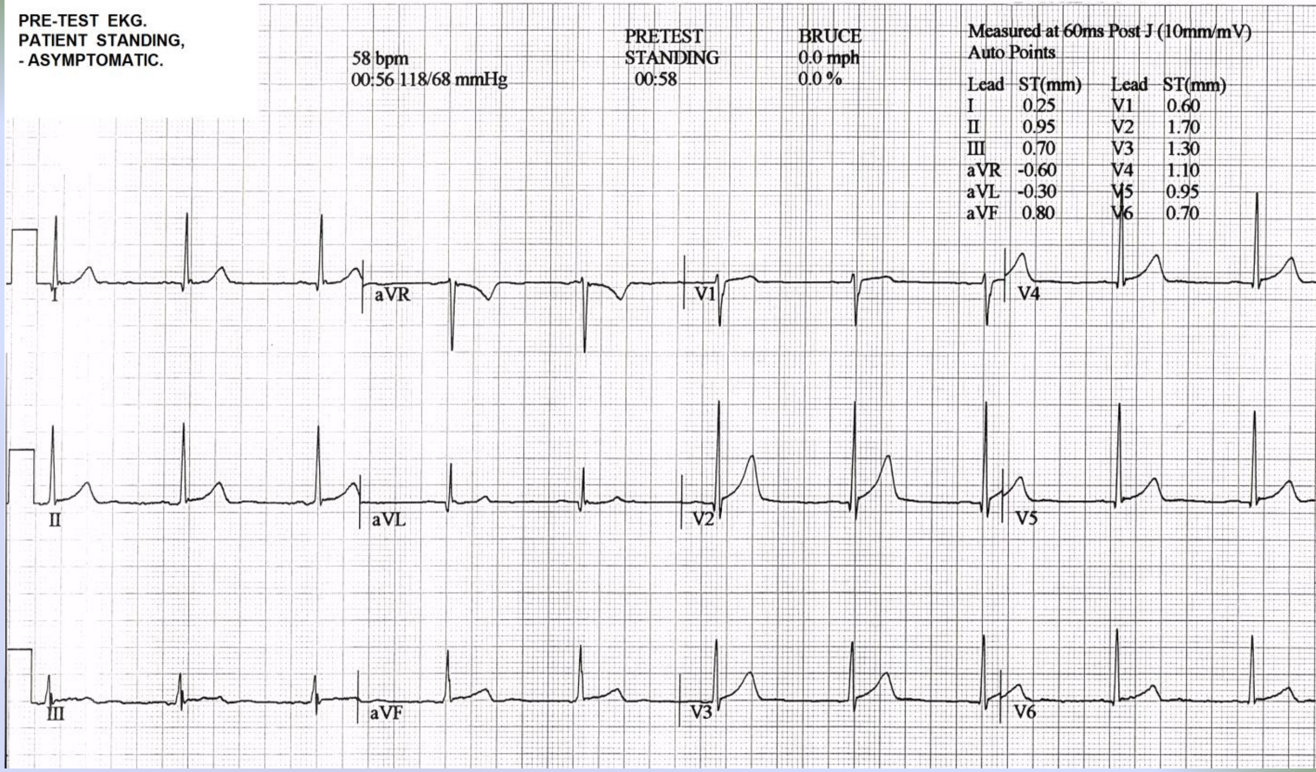
- Patient hospitalized as “Observation” status patient.
- Serial EKGs and Troponins were NEGATIVE.
- PATIENT FAILED STRESS TEST the next morning.
- Sent for a STAT Cardiac Cath…….

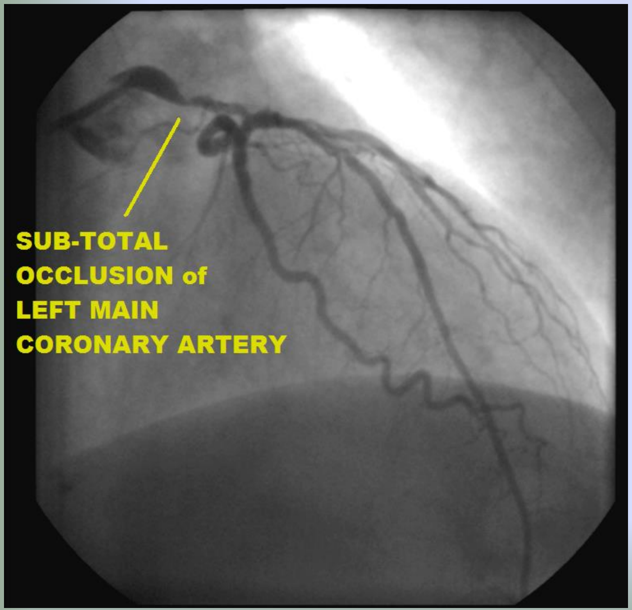

SUB-TOTAL
OCCLUSION of
LEFT MAIN
CORONARY ARTERY

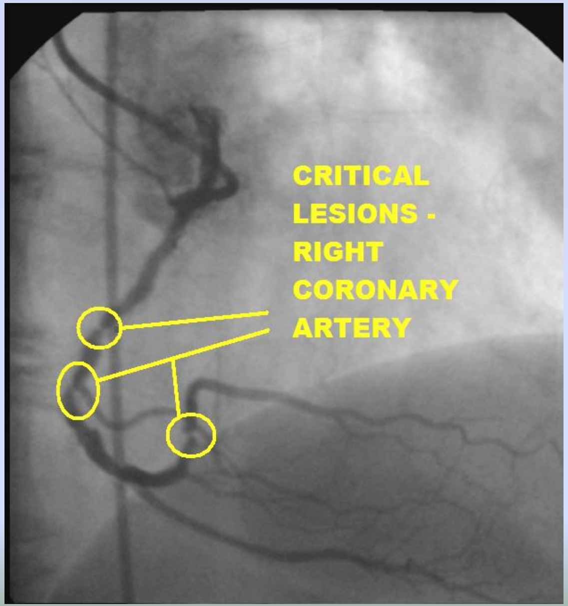

CRITICAL LESIONS - RIGHT CORONARY ARTERY

Heart Score 5.

Lethal Triple Vessel Disease =







Emergency Triple Vessel Coronary Artery Bypass Surgery

63 y/o male patient:

- The HEART Score guided physicians to admit the patient to Observation and do a cardiac work-up.
- Stress Test in the AM indicated “significant global ischemia.”
- Patient taken to Cath Lab where critical Triple-Vessel Disease was discovered
- Patient taken to STAT Open Heart Surgery.

stable angina	VS.	unstable angina
1. SYMPTOMS START DURING PHYSICAL EXERTION.		1. SYMPTOMS MAY START AT ANY TIME, EVEN DURING REST
2. SYMPTOMS ARE "PREDICTABLE"		2. SYMPTOMS ARE *NEW*, *DIFFERENT*, or *WORSE* THAN PREVIOUS EPISODES

BEWARE of the patient with
“INTERMITTENT CHEST PAIN” . . . .

Modified HEART Score for EMS

- Most EMS units don’t have access to “Troponin blood testing.”
- The “HEAR” Score (“HEART” – minus the Troponin) has been validated by recent a recent study conducted by Cambridge University.
- View Cambridge University Journal article about HEAR Score

Integrated ECG:

- HEMODYNAMIC STATUS
  - ABCs
  - Shock
- SYMPTOMS
  - Chest Pain / Pressure
  - Other ACS Symptoms
- ECG
  - **12 Lead**
  - Single Lead "rhythm strip"

Evaluating the ECG for ACS:

A TWO-STEP process:

Evaluating the ECG for ACS:

A TWO-STEP process:

STEP 1: <u>Evaluate QRS Width</u>

Evaluating the ECG for ACS:

A TWO-STEP process:

STEP 1: **Evaluate QRS Width**

STEP 2: **Evaluate J Points, ST-Segment and T waves in EVERY Lead**

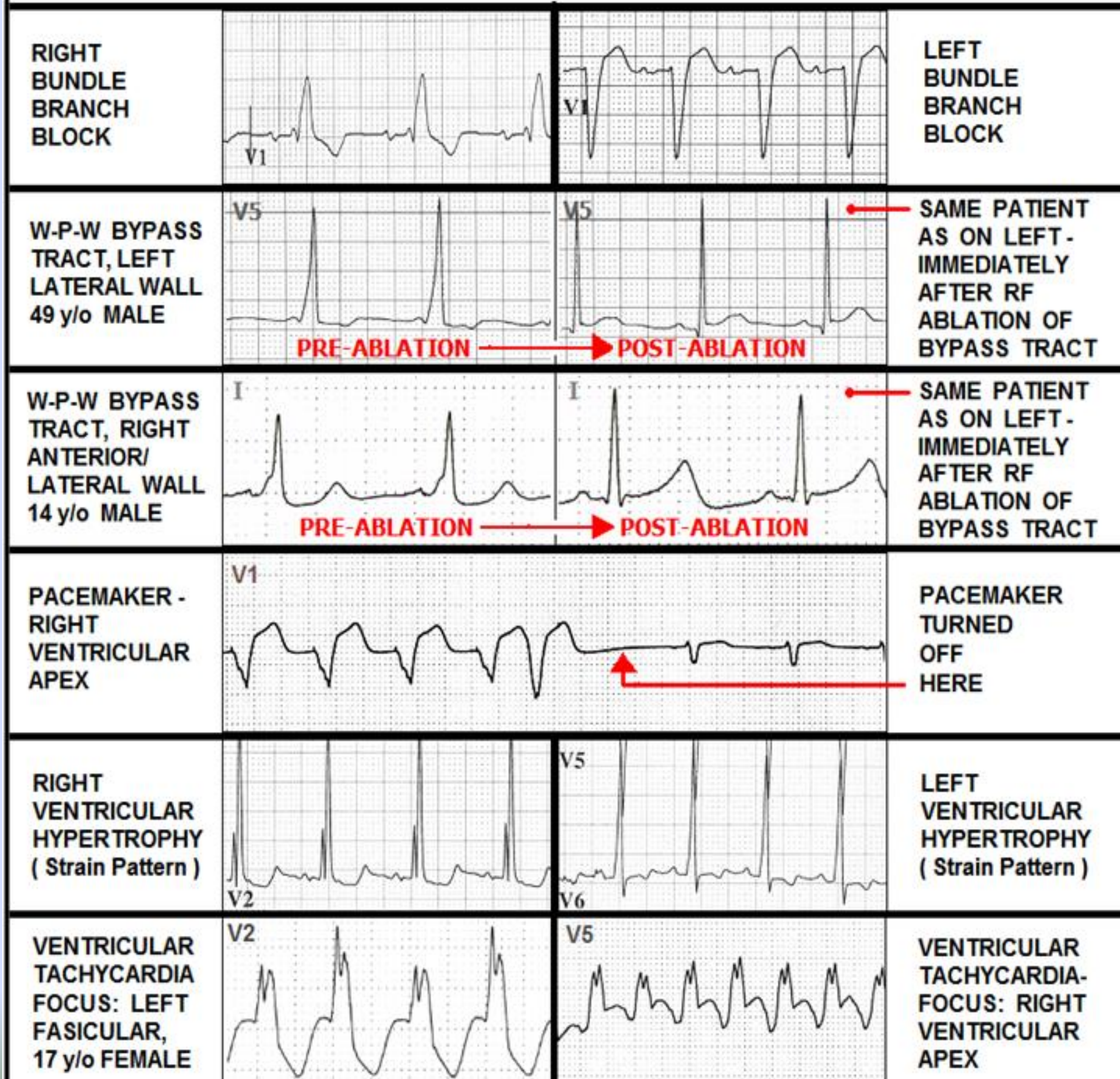
STEP 1 – evaluate QRS width:

- **QRS is ABNORMALLY WIDE (>120 ms),**
  - **indicates DEPOLARIZATION ABNORMALITY (e.g. “bundle branch block, Wolff-Parkinson-White Syndrome, etc).**

STEP 1 – evaluate QRS width:

- **QRS is ABNORMALLY WIDE (>120 ms),**
  - **indicates DEPOLARIZATION ABNORMALITY (e.g. "bundle branch block, Wolff-Parkinson-White Syndrome, etc).**
  - **DEPOLARIZATION ABNORMALITIES in turn cause REPOLARIZATION ABNORMALITIES, which alters the: *J Points, ST-Segments and/or T Waves.***

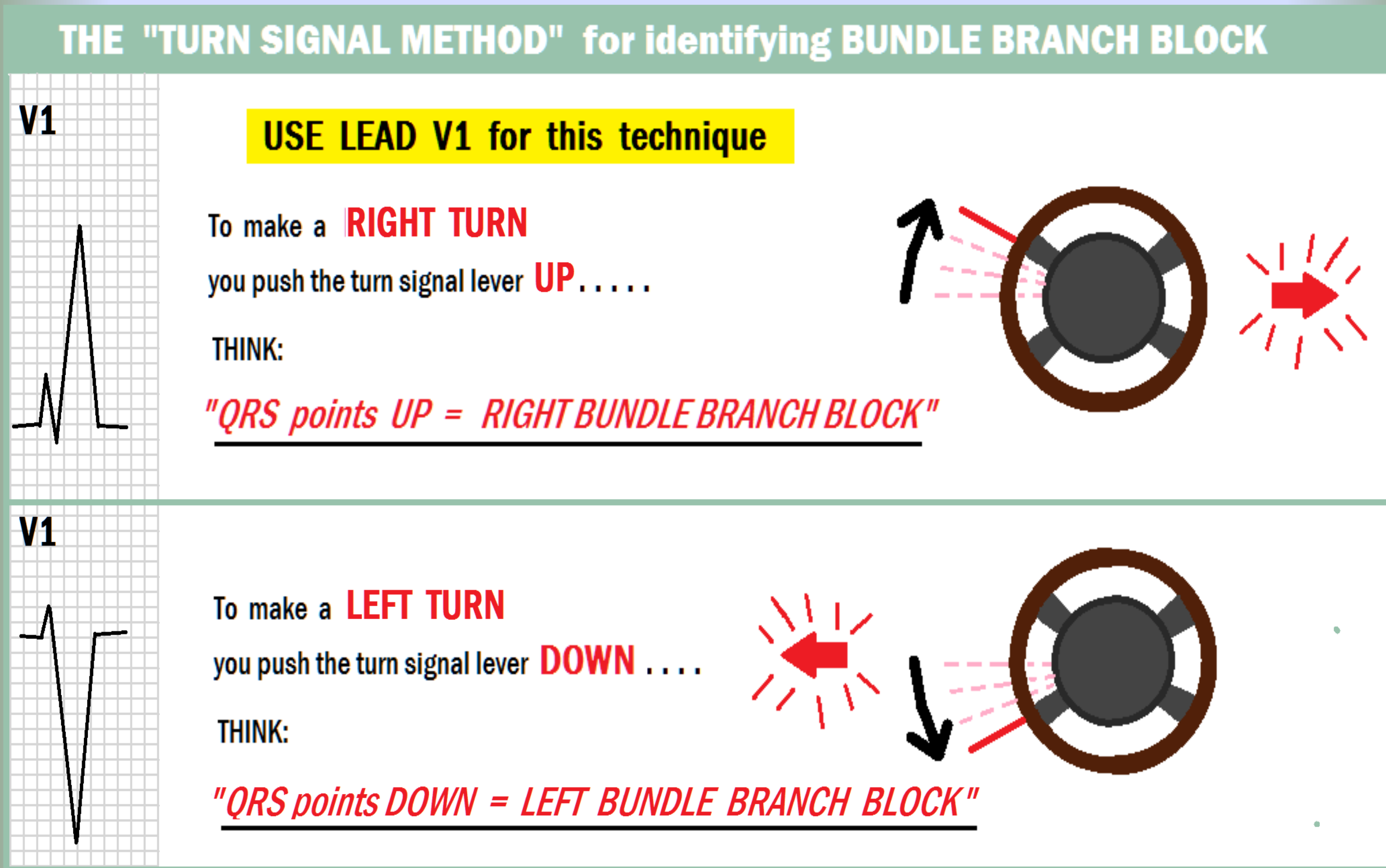
CONDITIONS THAT INCREASE QRS DURATION RESULT IN SECONDARY REPOLARIZATION ABNORMALITIES:



Wide QRS present:
QRSd > 120ms

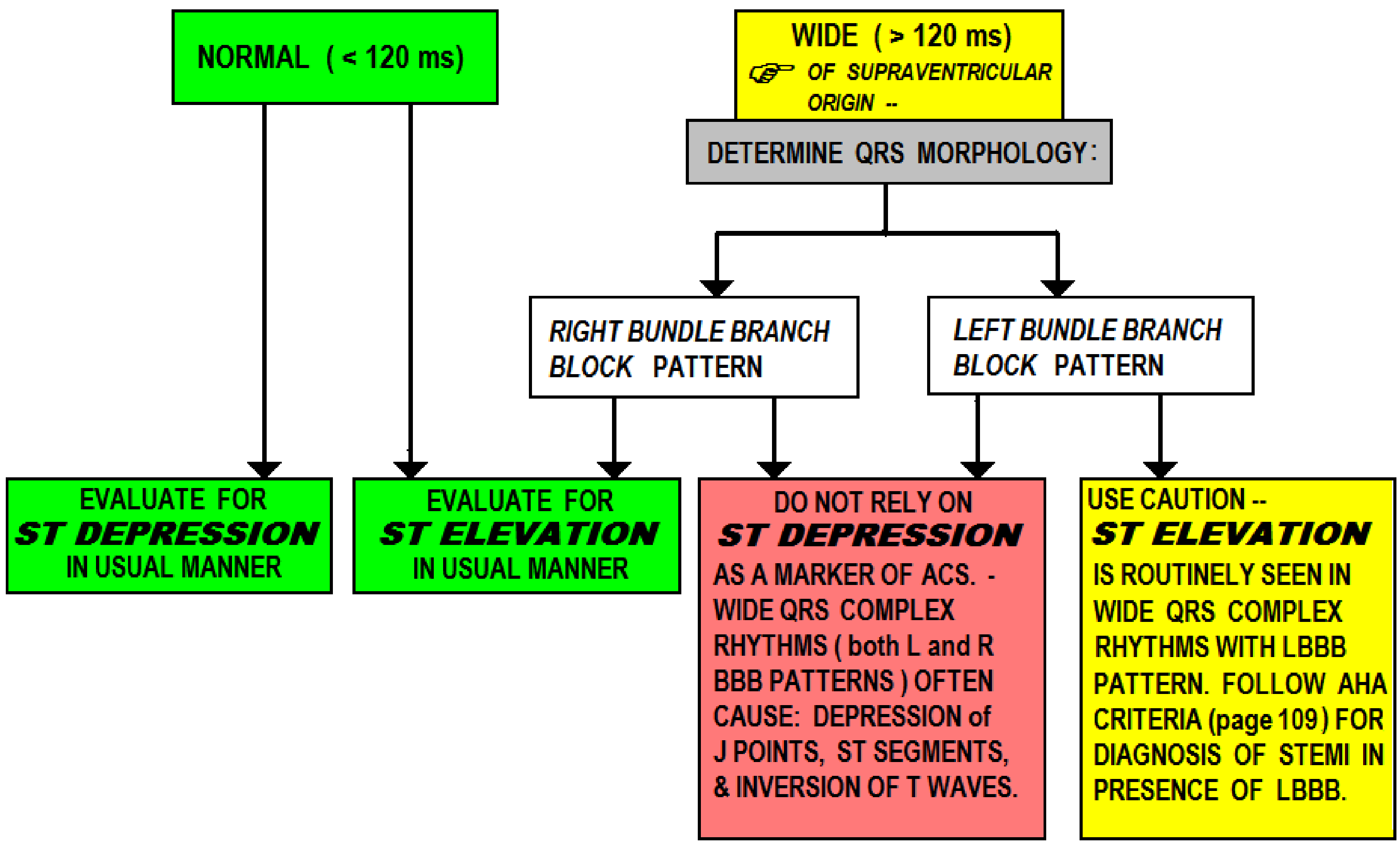
- **Determine RIGHT vs. LEFT Bundle Branch Block Pattern**

Simple “Turn Signal Method” . . .



Evaluating the ECG for ACS:

STEP 1 - EVALUATE WIDTH OF QRS:



Wide QRS present:
(QRSd > 120ms)

- **When RIGHT Bundle Branch Block pattern is present:**
  - **Precordial Leads typically demonstrate ST Depression and T wave Inversion**

RUPPERT, WAYNE ID: 099999999 4-Mar-2001

74years
Male Caucasian

Room:
Loc: 0 Opt:

Technician: WR

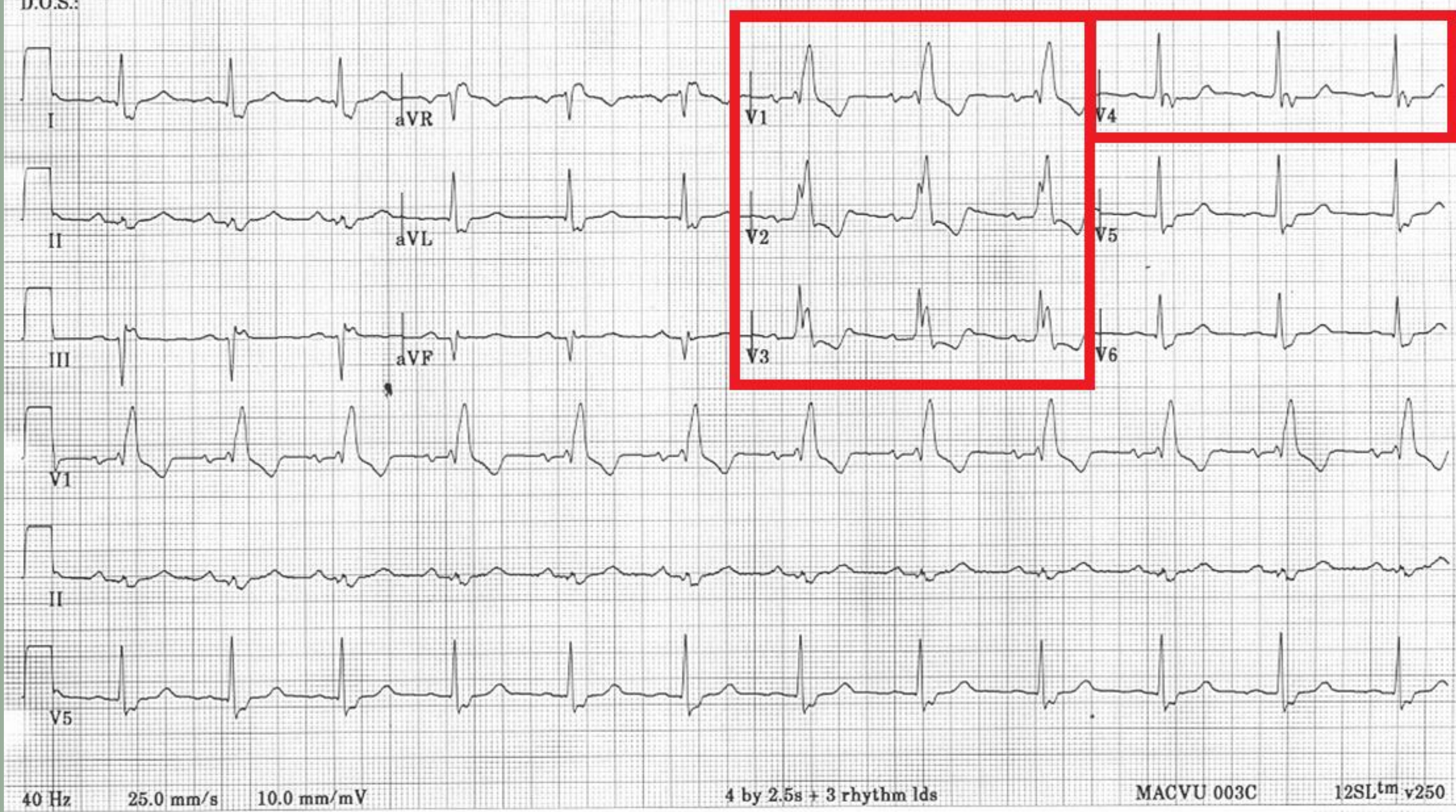
Vent. rate	72	bpm
PR interval	186	ms
QRS duration	166	ms
QT/QTc	436/477	ms
P-R-T axes	57 -32 32	

Normal sinus rhythm
Left axis deviation
Right bundle branch block
Inferior infarct, age undetermined
Abnormal ECG

RBBB causes ST Depression, T Wave Inversion, ANTERIOR Leads (V1 - V4).

Referred by: Unconfirmed

D.O.S.:



Wide QRS present:
(QRSd > 120ms)

- **When RIGHT Bundle Branch Block pattern is present:**
  - **Precordial Leads typically demonstrate ST Depression and T wave Inversion**
  - **DOES NOT MASK STEMI; *when ST Elevation is noted, CONSIDER STEMI ! !***

RBBB with CHEST PAIN - CASE 1 : ST ELEVATION IN LEADS V1 - V4

48 yr
Male Caucasian

Room:ATL
Loc:3 Option:23

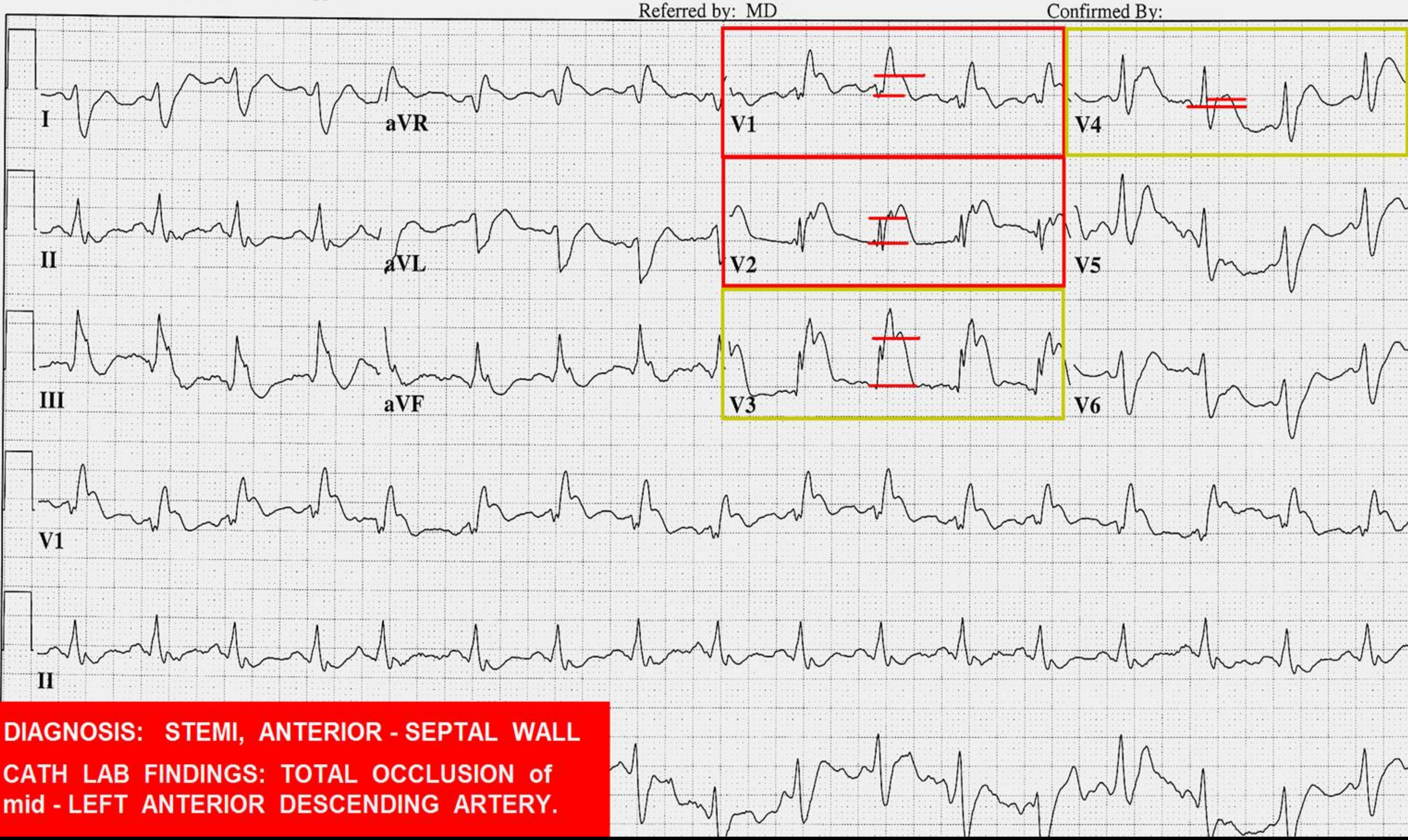
Vent. rate	102	BPM
PR interval	130	ms
QRS duration	168	ms
QT/QTc	400/521	ms
P–R–T axes	60 114	–19

Sinus tachycardia with Premature supraventricular complexes and Fusion complexes
Right bundle branch block
ST elevation consider anterior injury or acute infarct
** ** ** ** * ACUTE MI * ** ** ** **
Abnormal ECG ...

Technician: W Ruppert

Referred by: MD

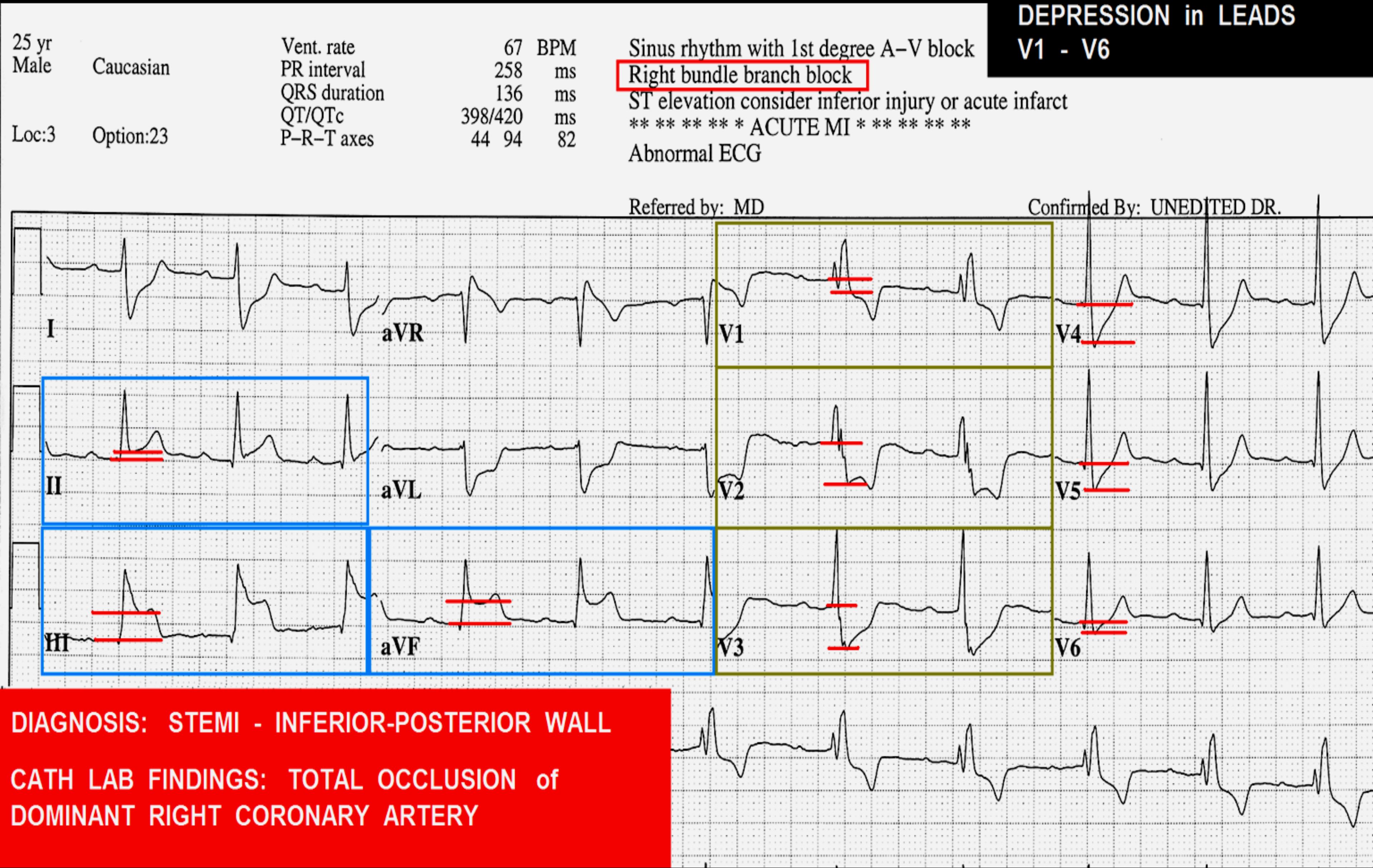
Confirmed By:



DIAGNOSIS: STEMI, ANTERIOR - SEPTAL WALL

CATH LAB FINDINGS: TOTAL OCCLUSION of mid - LEFT ANTERIOR DESCENDING ARTERY.

RBBB with CHEST PAIN - CASE 2: ST ELEVATION LEADS II, III, aVF - WITH RECIPROCAL ST DEPRESSION in LEADS V1 - V6



DIAGNOSIS: STEMI - INFERIOR-POSTERIOR WALL

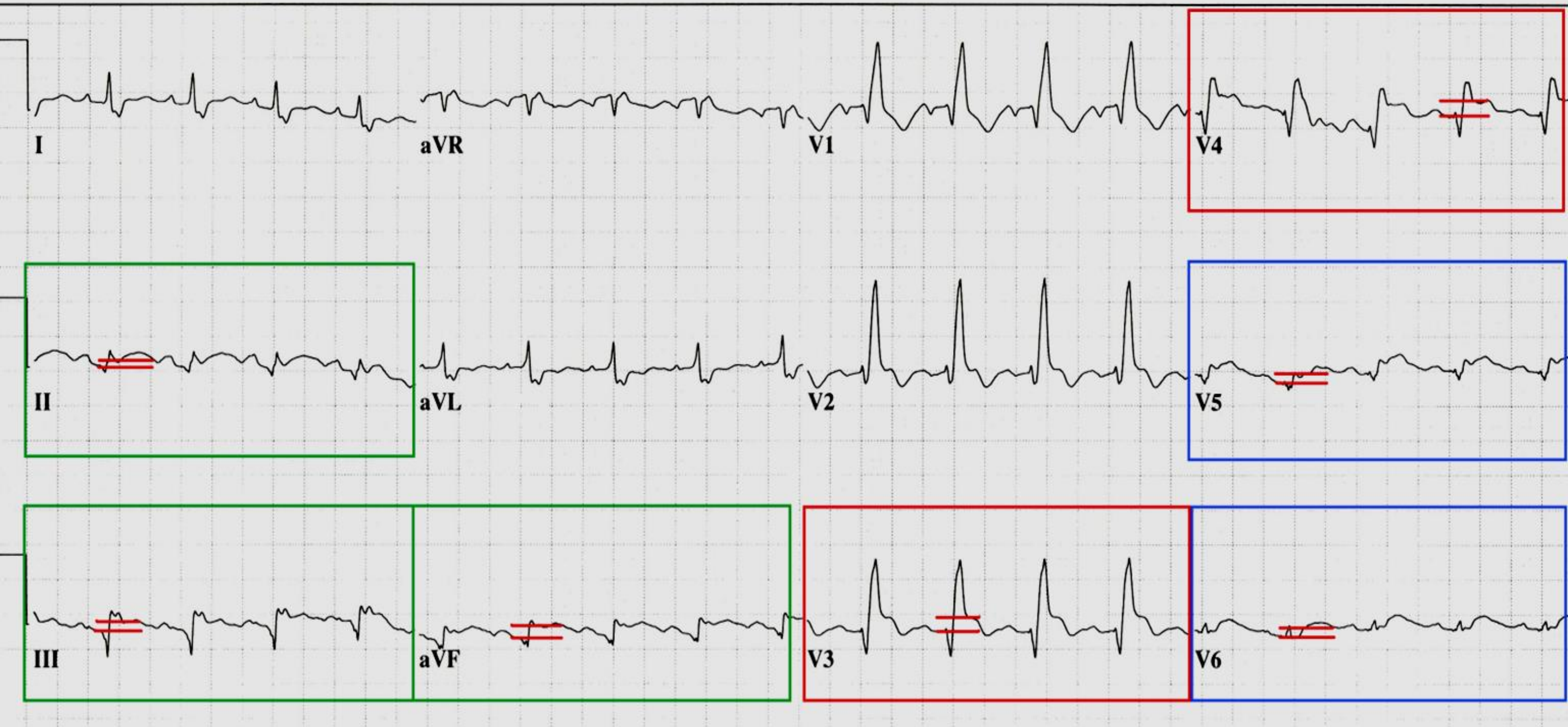
CATH LAB FINDINGS: TOTAL OCCLUSION of DOMINANT RIGHT CORONARY ARTERY

RBBB with CHEST PAIN - CASE 3: ST ELEVATION V3 - V6, II, III, aVF

75 yr		Vent. rate	110	BPM	Sinus tachycardia
Male	Caucasian	PR interval	170	ms	Right bundle branch block
		QRS duration	148	ms	Lateral infarct , possibly acute
Room:CS−19		QT/QTc	366/495	ms	Inferior infarct , possibly acute
Loc:6	Option:41	P−R−T axes	57 19	69	Anterior injury pattern
					Abnormal ECG

ACUTE LATERAL - INFERIOR - ANTERIOR AMI

CATH LAB FINDINGS: OCCLUDED VEIN GRAFT TO THE CIRCUMFLEX DISTRIBUTION (DOMINANT CIRCUMFLEX)



Wide QRS present:

(QRSd > 120ms)

- **When LBBB QRS pattern is present:**

Wide QRS present:
(QRSd > 120ms)

- **When LBBB QRS pattern is present:**
  - **ST-Segment Elevation is typically noted in Precordial Leads**

Wide QRS present:
(QRSd > 120ms)

- **When LBBB QRS pattern is present:**
  - **ST-Segment Elevation is typically noted in Precordial Leads**
  - ***Can cause up to 5mm of J Point Elevation in normally calibrated ECG (1mm=10mv)***

Wide QRS present:
(QRSd > 120ms)

- **When LBBB QRS pattern is present:**
  - **ST-Segment Elevation is typically noted in Precordial Leads**
  - ***Can cause up to 5mm of J Point Elevation in normally calibrated ECG (1mm=10mv)***
  - ***Does NOT typically cause ST elevation in INFERIOR Leads (II, III and AVF).***

Diagnosis of STEMI with LBBB pattern:

2013 ACC/AHA Guideline for Management of STEMI

- ***ST Elevation of 0.1mv (1mm) or more in leads with Positive Deflection QRS complexes***

Diagnosis of STEMI with LBBB pattern:

2013 ACC/AHA Guideline for Management of STEMI

- ***ST Elevation of 0.1mv (1mm) or more in leads with Positive Deflection QRS complexes***
- ***ST Elevation of 0.5mv (5mm) or more in leads with Negative Deflection QRS complexes***

Diagnosis of STEMI with LBBB pattern:

2013 ACC/AHA Guideline for Management of STEMI

- ***ST Elevation of 0.1mv (1mm) or more in leads with Positive Deflection QRS complexes***
- ***ST Elevation of 0.5mv (5mm) or more in leads with Negative Deflection QRS complexes***
- ***ST Segment Changes as compared with those of older ECGs with LBBB***

Diagnosis of STEMI with LBBB pattern:

2013 ACC/AHA Guideline for Management of STEMI

- ***ST Elevation of 0.1mv (1mm) or more in leads with Positive Deflection QRS complexes***
- ***ST Elevation of 0.5mv (5mm) or more in leads with Negative Deflection QRS complexes***
- ***ST Segment Changes as compared with those of older ECGs with LBBB***
- ***Convex ST Segment***

78 yr
Female Black

Room:ICU5
Loc:6 Option:19

Vent. rate	94	BPM
PR interval	202	ms
QRS duration	160	ms
QT/QTc	388/485	ms
P-R-T axes	91 -23	87

Normal sinus rhythm with occasional Premature ventricular complexes
Left bundle branch block
Abnormal ECG

- Normal arteries
- Normal LV Function
- No hypertrophy

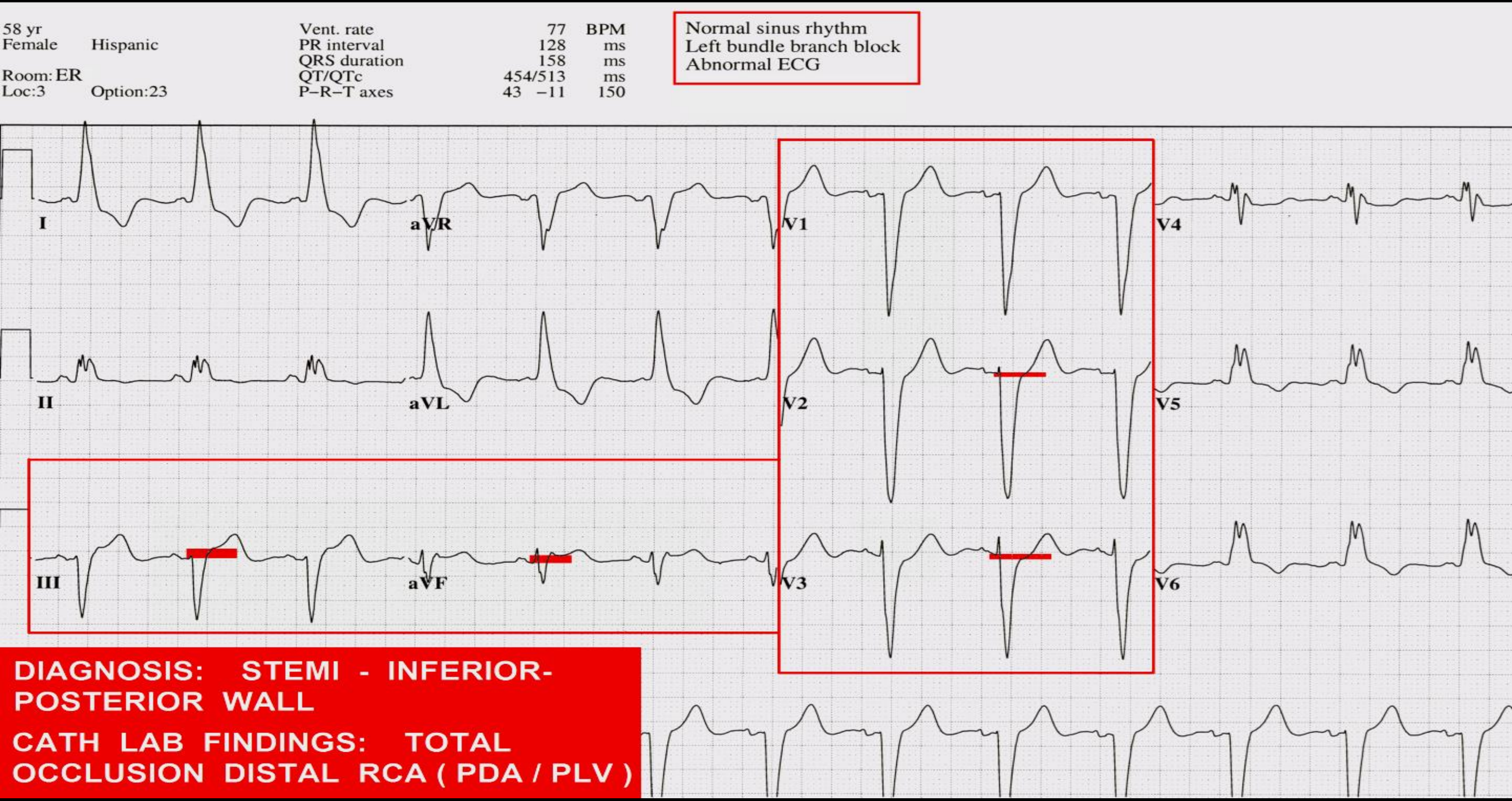
Technician: EKG CLASS #WR03602718

Referred by:

25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 11 EID:18 EDT:

“Electrocardiographic Diagnosis of Evolving Acute Myocardial Infarction in the Presence of Left Bundle-Branch Block” Birnbaum et al, N Engl J Med 1996; 334:481-487

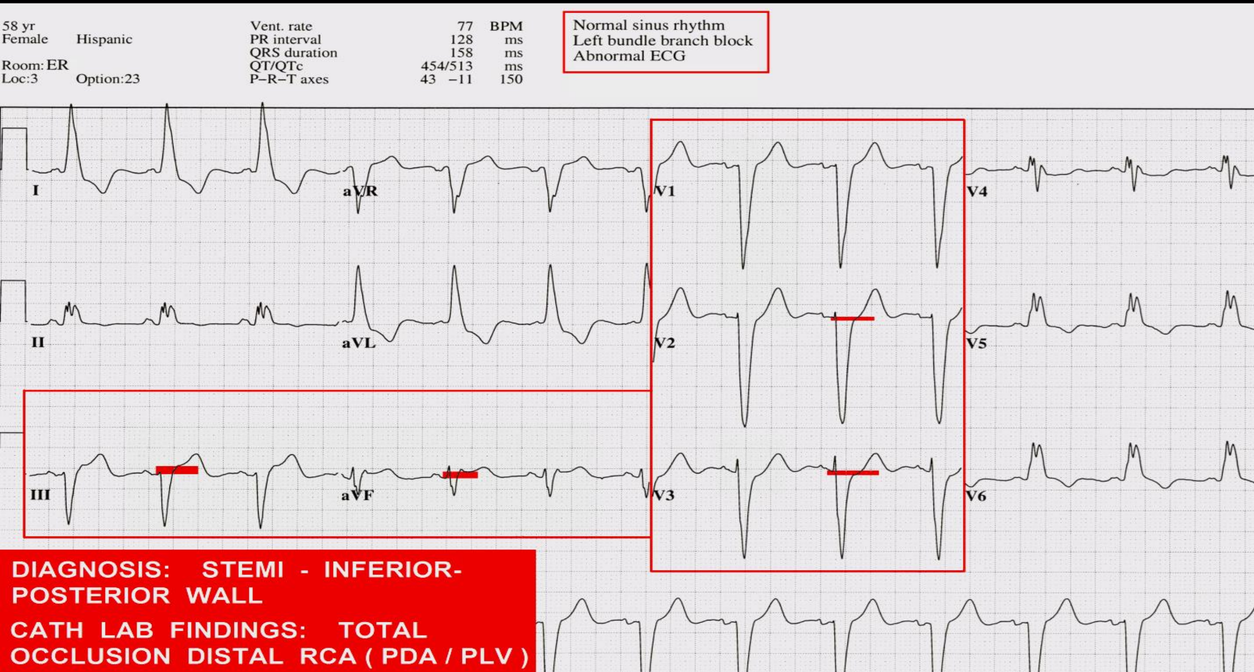
LBBB with CHEST PAIN - CASE 1 : PRESENTING EKG



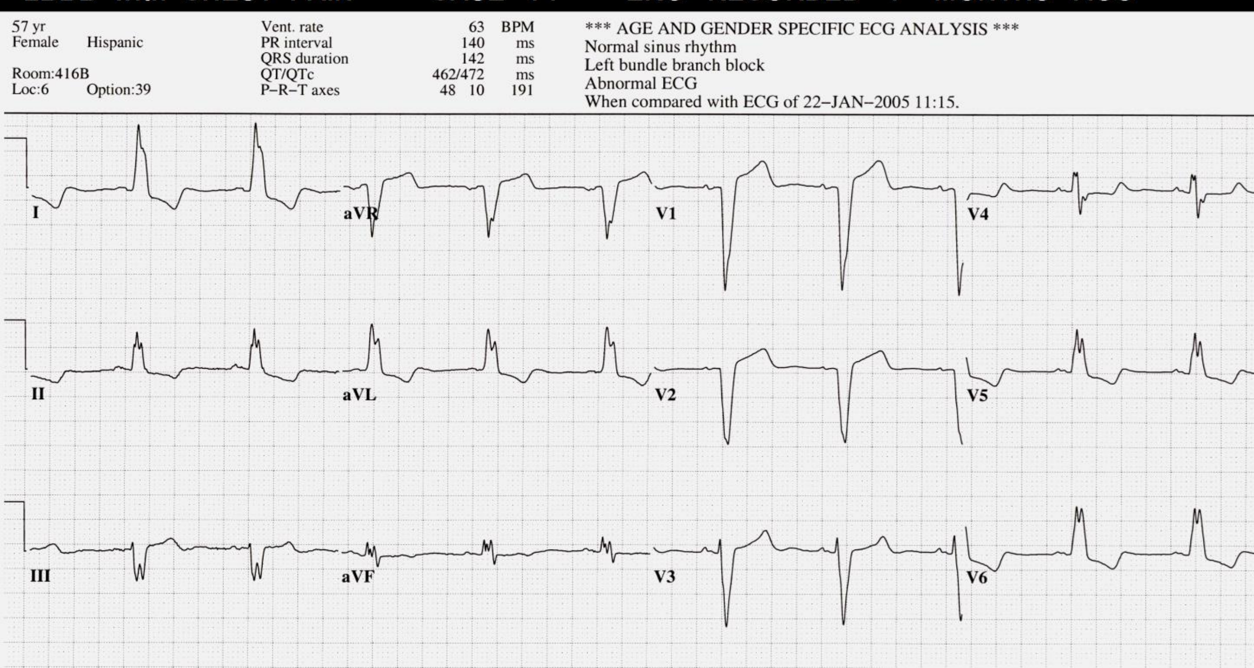
DIAGNOSIS: STEMI - INFERIOR-POSTERIOR WALL

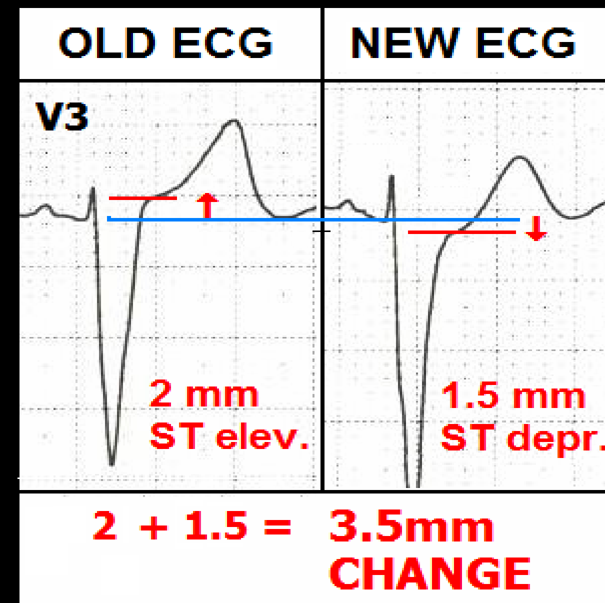
CATH LAB FINDINGS: TOTAL OCCLUSION DISTAL RCA (PDA / PLV)

LBBB with CHEST PAIN - CASE 1: PRESENTING EKG



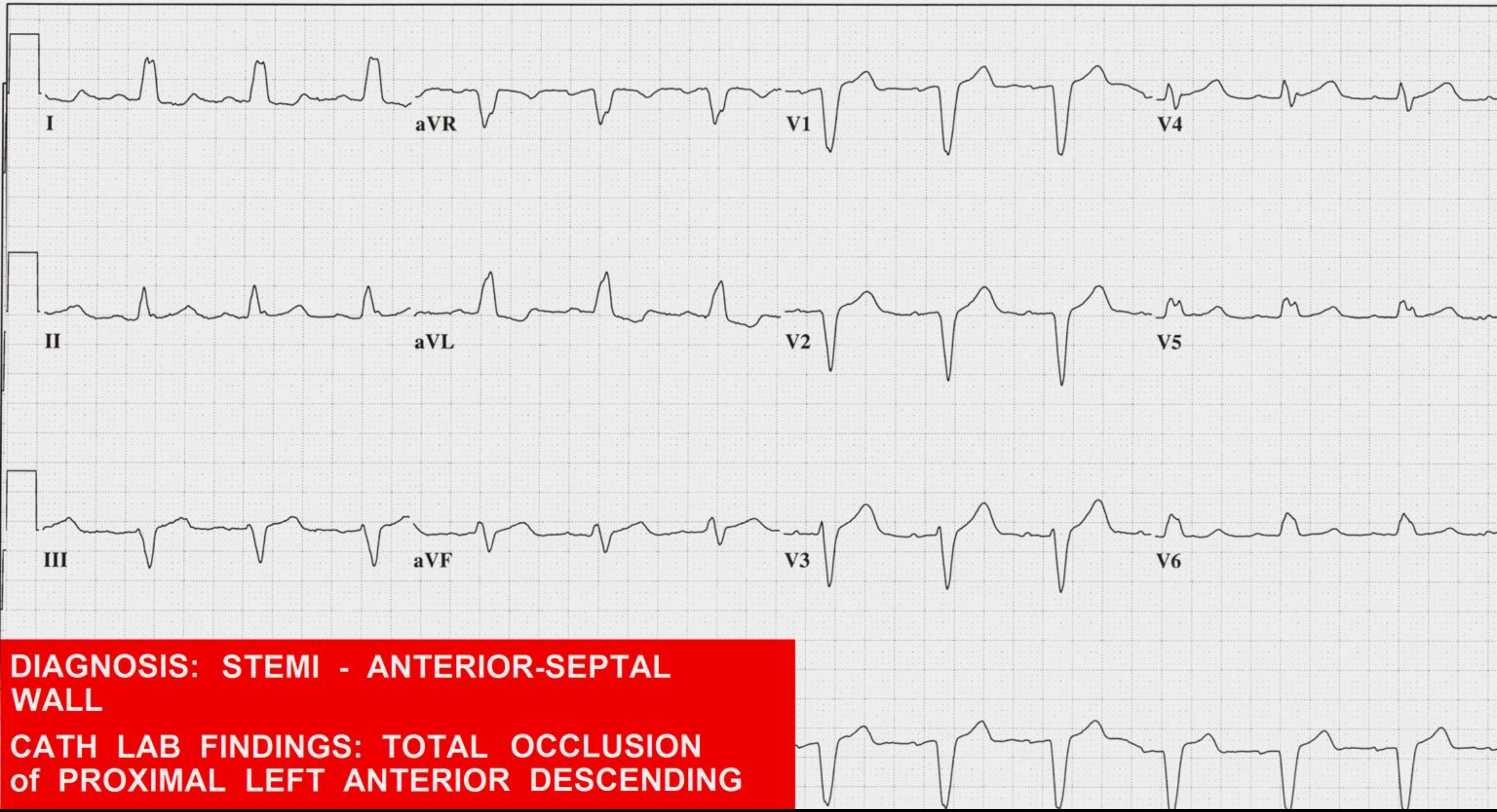
LBBB with CHEST PAIN - CASE 1: EKG RECORDED 7 MONTHS AGO





LBBB with CHEST PAIN - CASE 2: *NEW ONSET of LBBB*

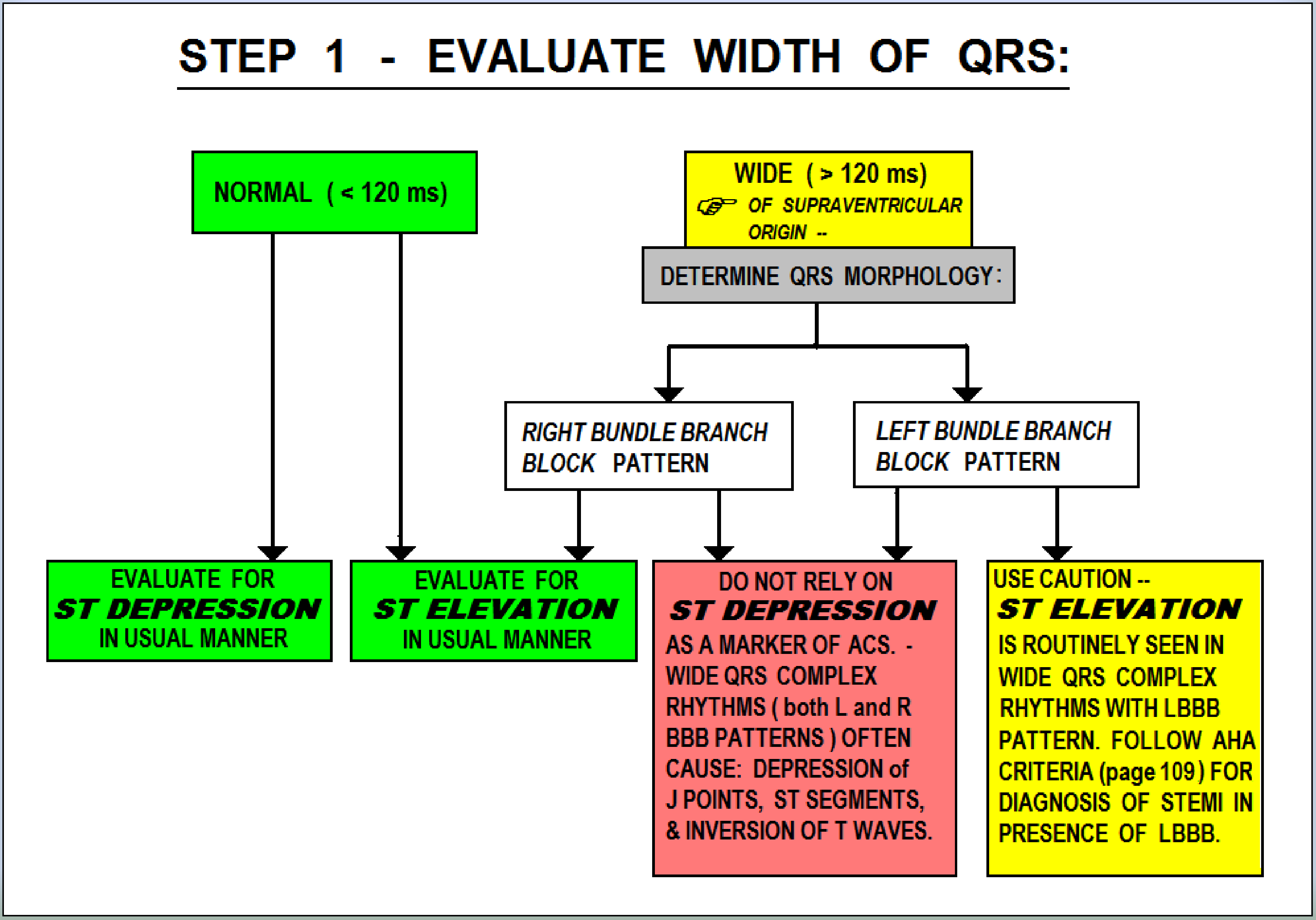
46 yr		Vent. rate	77	BPM	Normal sinus rhythm
Male	Caucasian	PR interval	172	ms	Left bundle branch block
		QRS duration	142	ms	Abnormal ECG
Room:ER		QT/QTc	446/504	ms	
Loc:3	Option:23	P–R–T axes	38 0	92	



DIAGNOSIS: STEMI - ANTERIOR-SEPTAL WALL

CATH LAB FINDINGS: TOTAL OCCLUSION of PROXIMAL LEFT ANTERIOR DESCENDING

Evaluating the ECG for ACS:



Evaluating the ECG for ACS:

Patients with Normal Width QRS

(QRSd < 120ms)

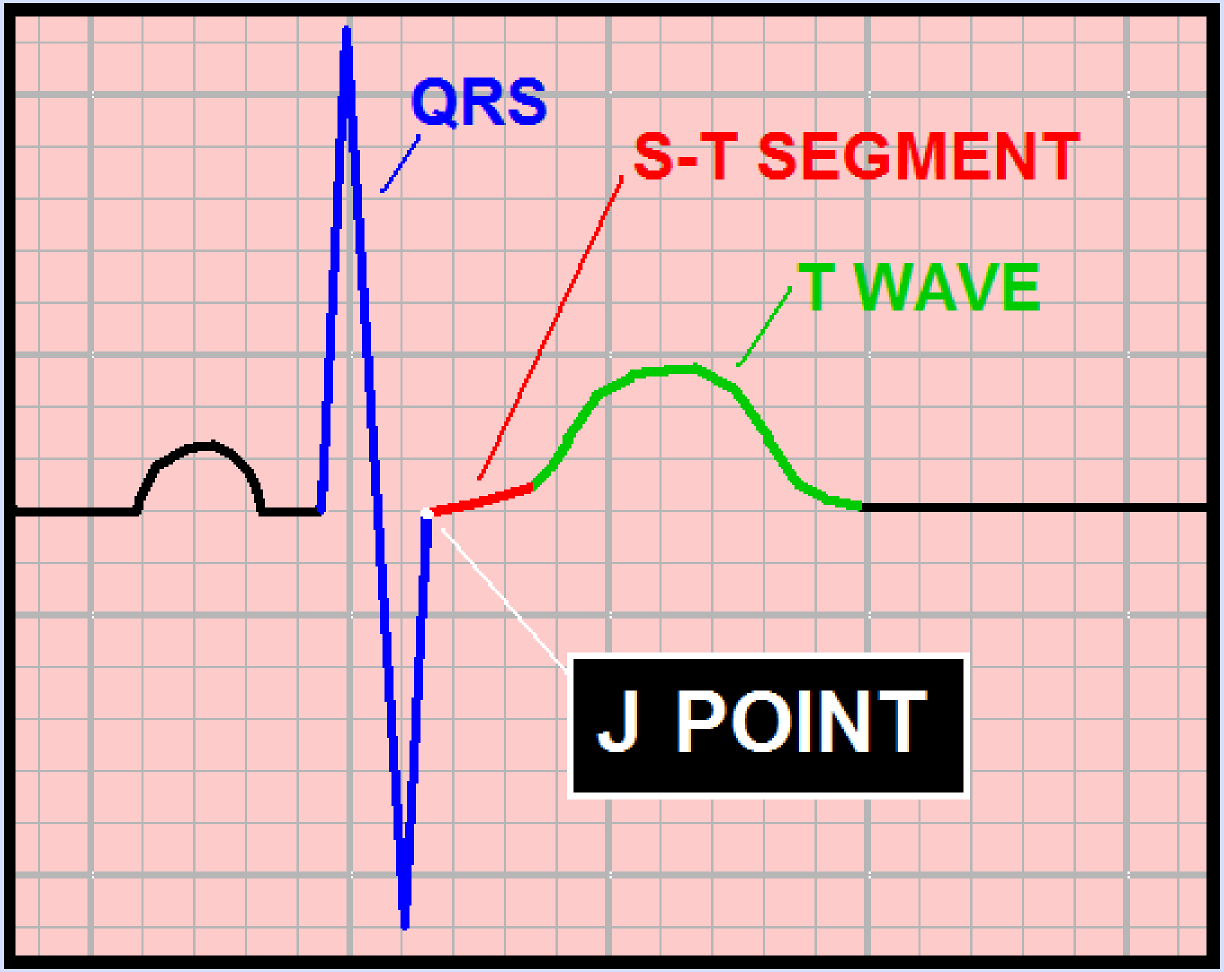
STEP 2 - EVALUATE the EKG for ACS

THE EKG MARKERS USED FOR DETERMINING THE PRESENCE OF ACUTE CORONARY SYNDROME INCLUDE:

- J POINTS
- ST SEGMENTS
- T WAVES

CAREFULLY SCRUTINIZE THESE MARKERS IN EVERY LEAD OF THE 12 LEAD EKG, TO DETERMINE IF THEY ARE *NORMAL* or *ABNORMAL*.

Defining NORMAL – QRS <120ms:



When QRS duration is NORMAL (< 120 ms):

NORMAL ST - T WAVES

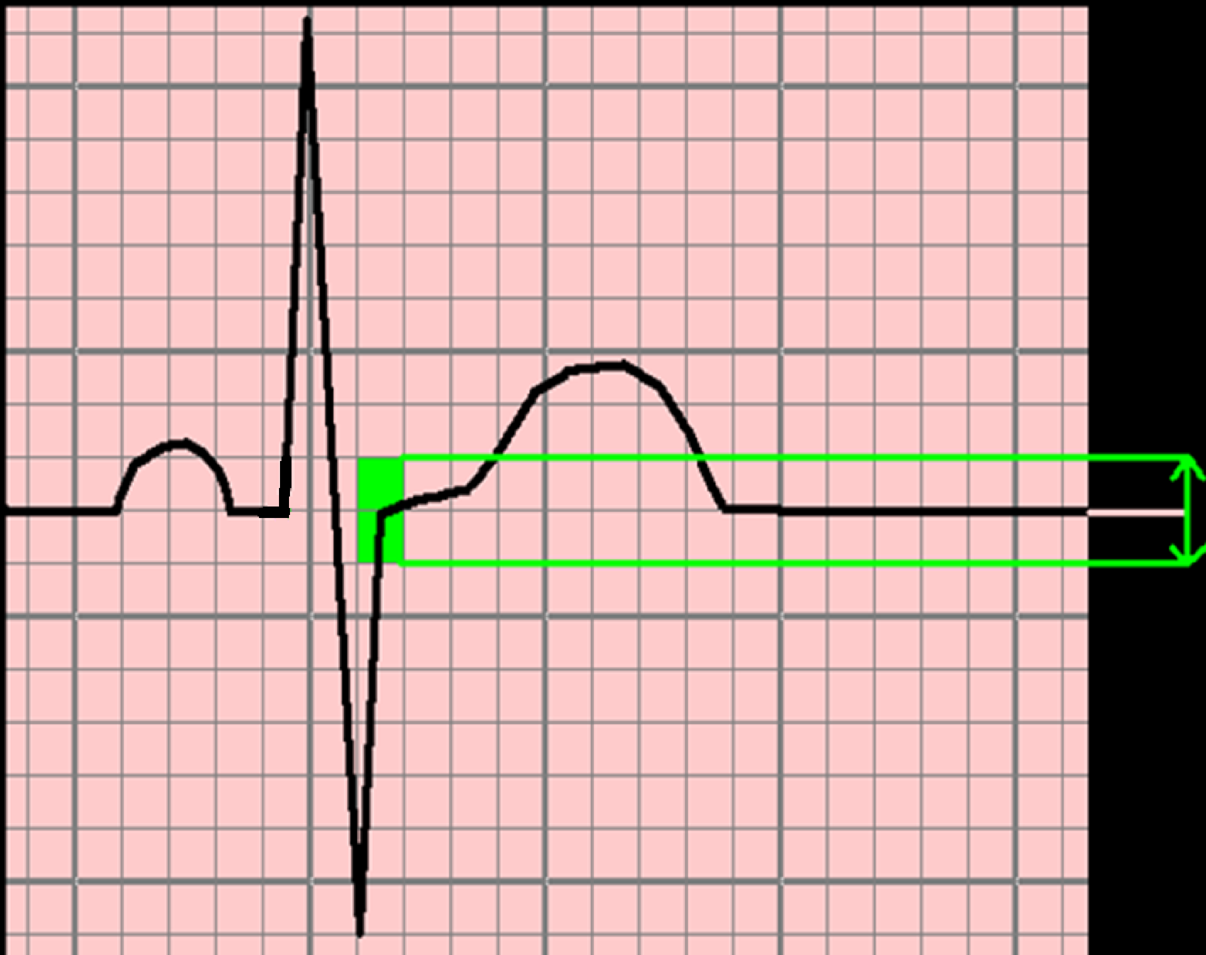
- WHEN QRS WIDTH IS NORMAL (< 120 ms)

ASSESS:

- J POINT: ISOELECTRIC (or < 1 mm dev.)
- ST SEG: SLIGHT, POSITIVE INCLINATION
- T WAVE: UPRIGHT, POSITIVE

☞ *in EVERY LEAD EXCEPT aVR ! !*

THE J POINT SHOULD BE . .

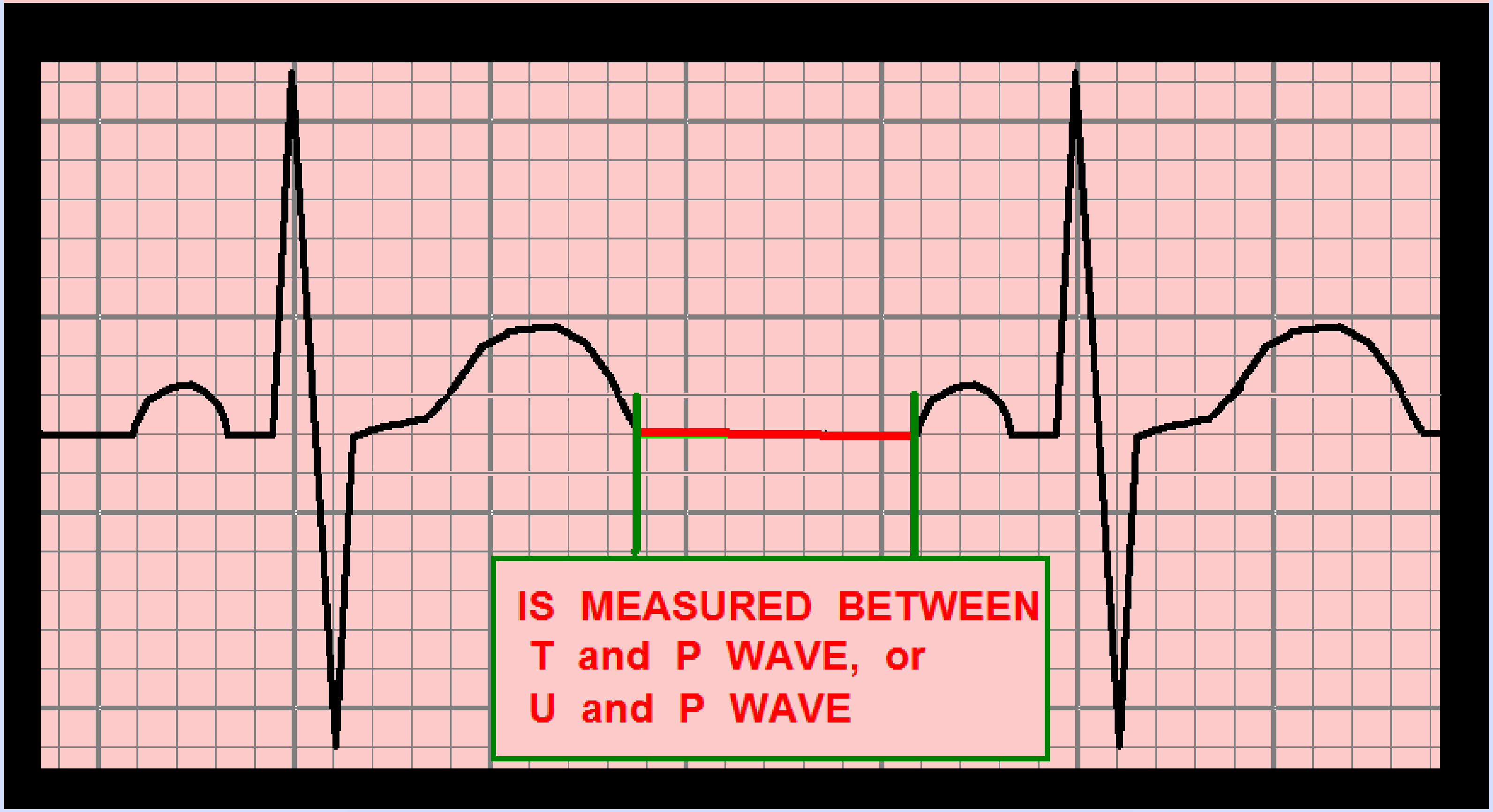


WITHIN
1 mm
ABOVE

OR

BELOW
the
ISOELECTRIC
LINE

THE ISOELECTRIC LINE

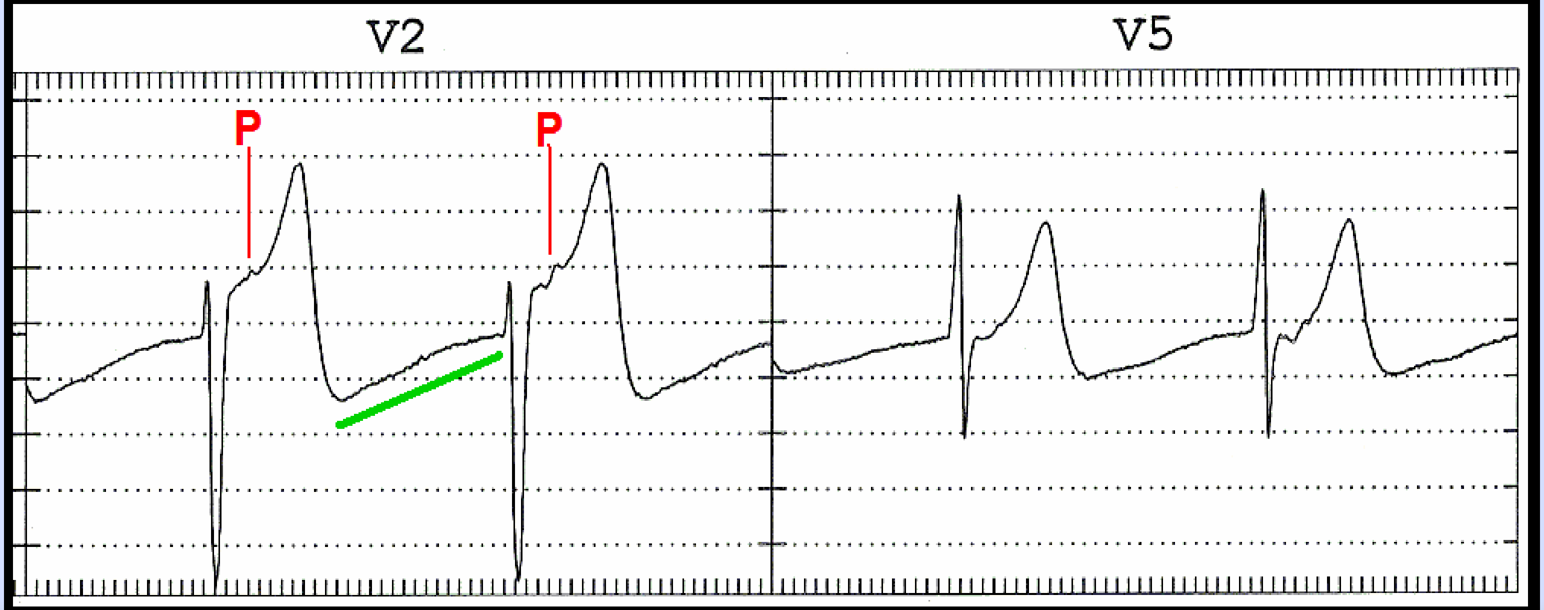


. . .the “flat line” between ECG complexes, when there is no detectable electrical activity . . .

The Isoelectric Line -
it's not always isoelectric !

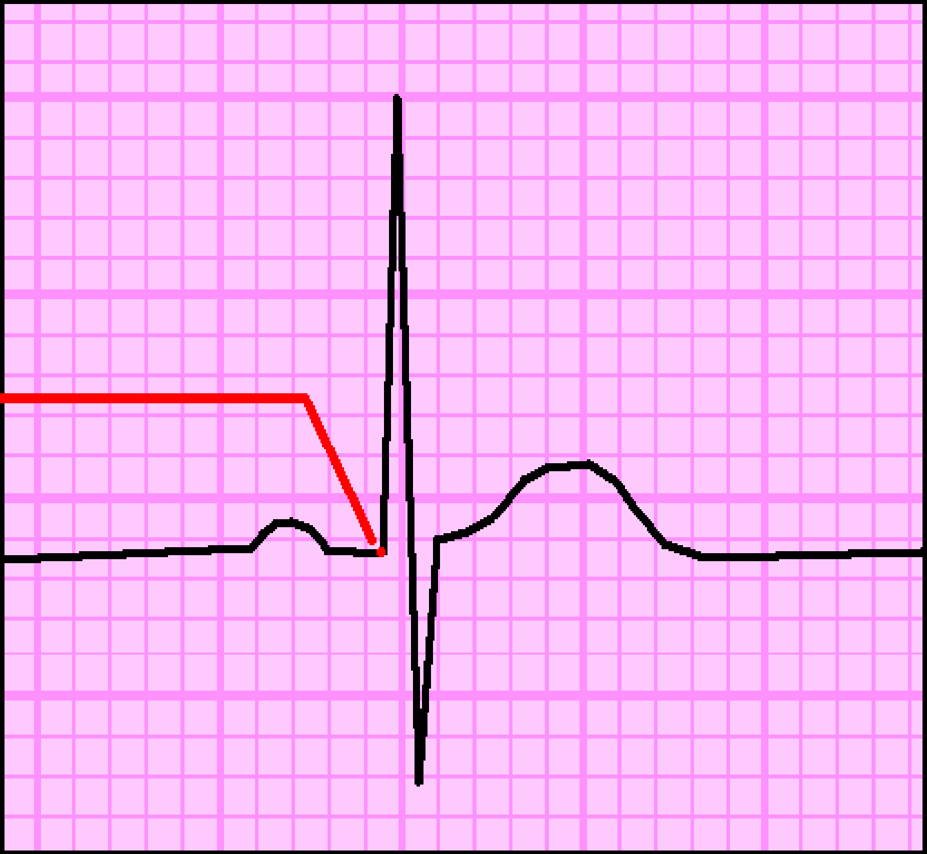
THE ISOELECTRIC LINE

EKG from 13 y/o girl in ACCELERATED JUNCTIONAL RHYTHM.
note: upsloping T-P interval, and P buried in T waves.



THE P-Q JUNCTION

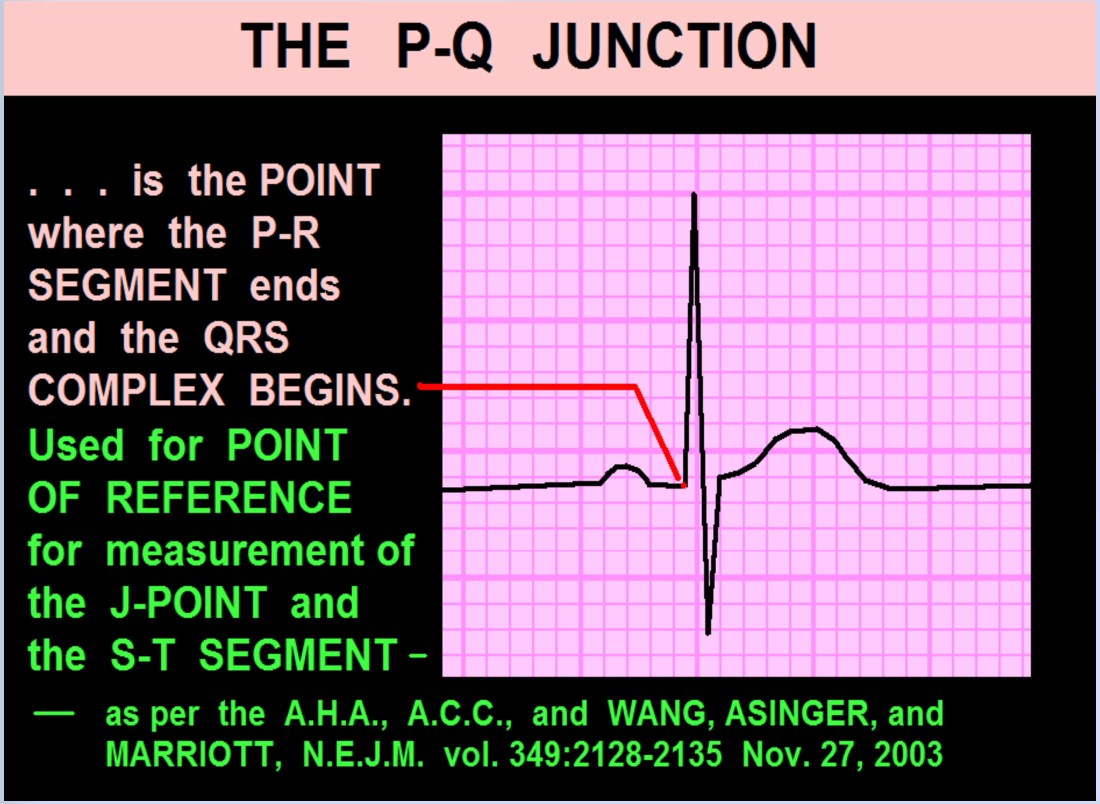
. . . is the POINT where the P-R SEGMENT ends and the QRS COMPLEX BEGINS.



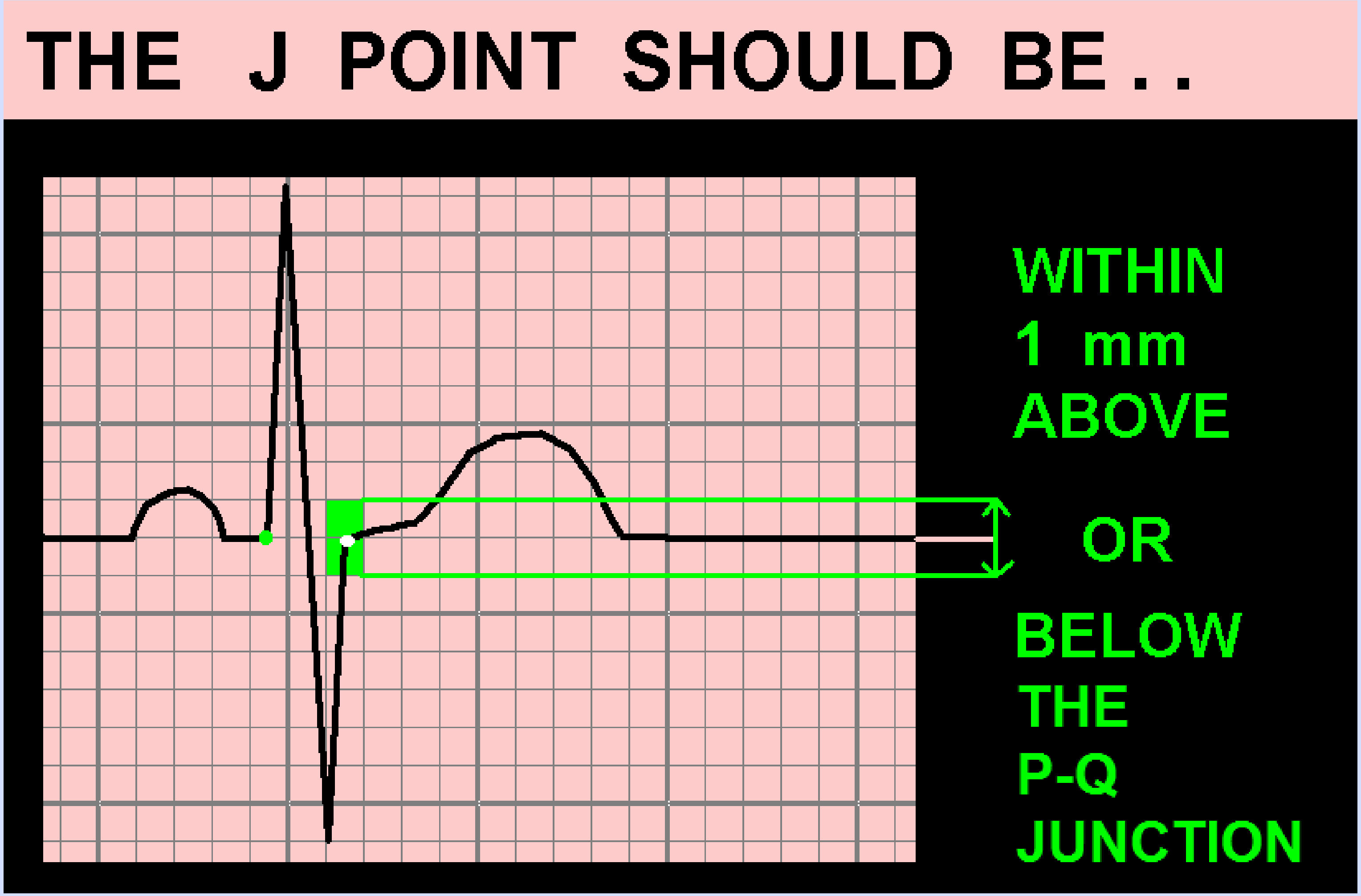
Used for POINT OF REFERENCE for measurement of the J-POINT and the S-T SEGMENT –

— as per the A.H.A., A.C.C., and WANG, ASINGER, and MARRIOTT, N.E.J.M. vol. 349:2128-2135 Nov. 27, 2003

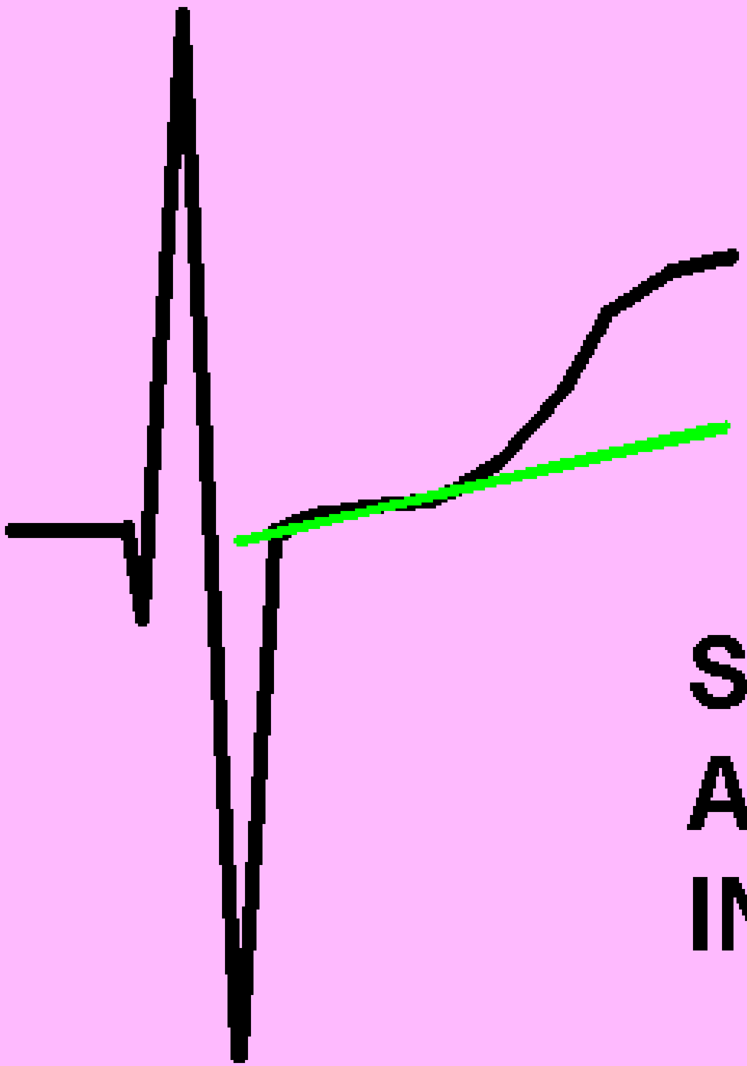
Use the P-Q junction as a reference point for measuring the J Point and ST-Segment when "iso-electric line is not iso-electric !



Defining NORMAL:

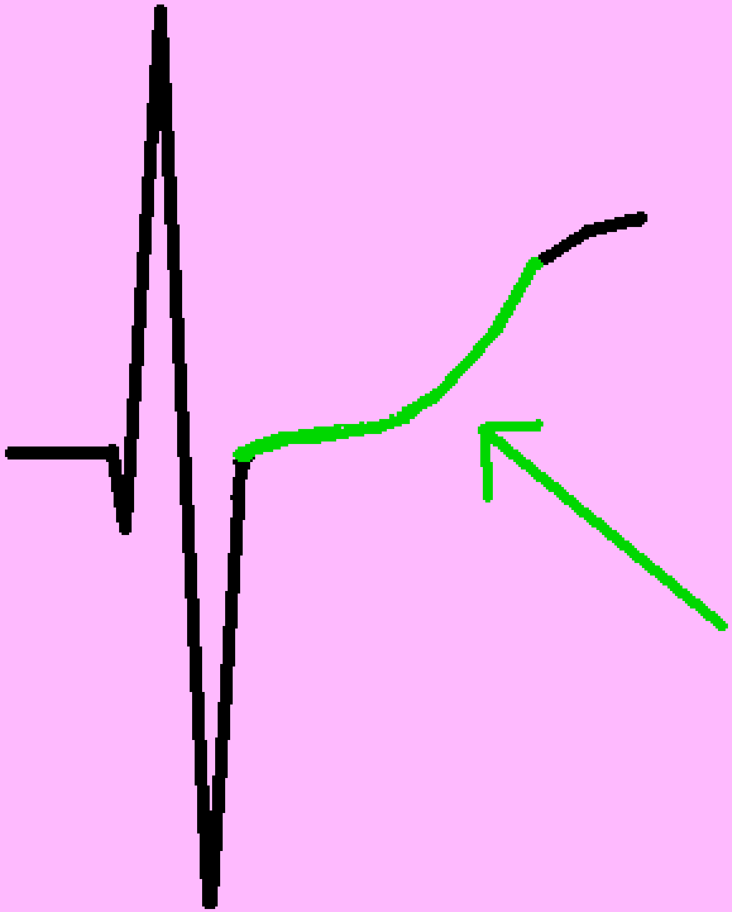


THE S-T SEGMENT



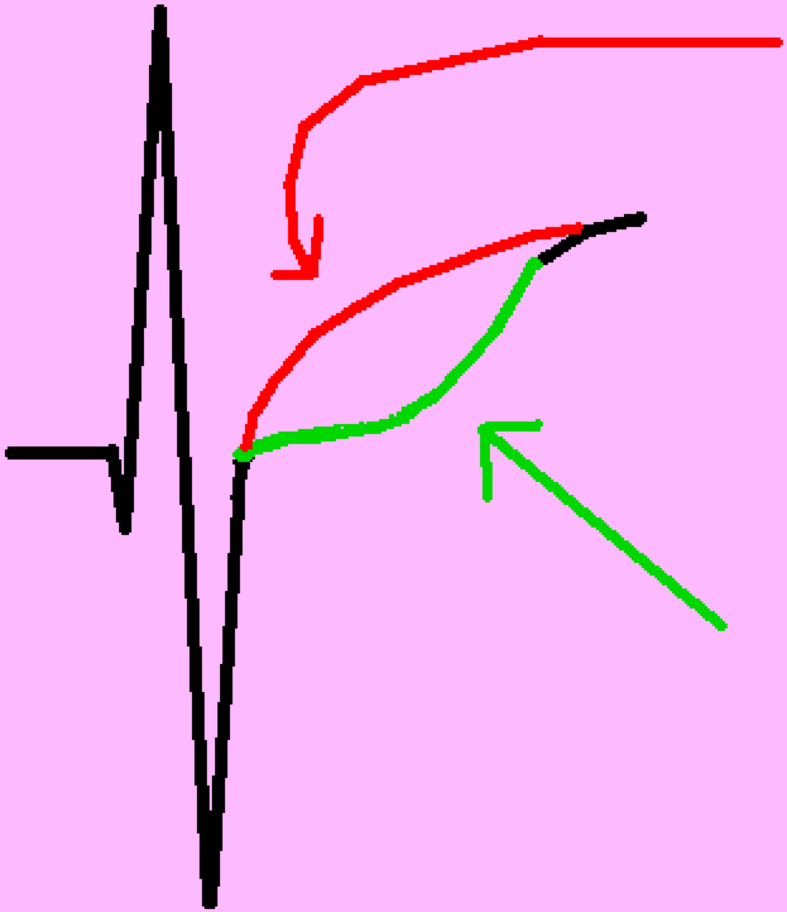
SHOULD HAVE
A "SLIGHT POSITIVE"
INCLINATION

THE S-T SEGMENT



SHOULD BE "CONCAVE" IN SHAPE . . .

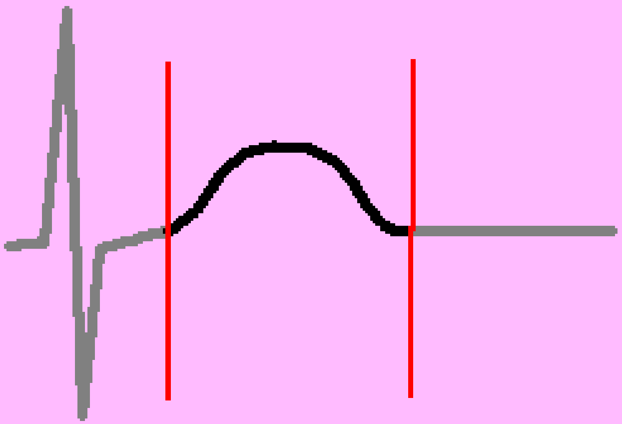
THE S-T SEGMENT



AS OPPOSED TO "CONVEX" IN SHAPE

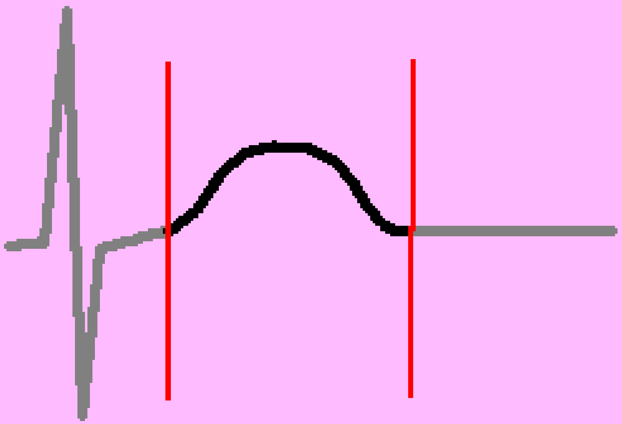
SHOULD BE "CONCAVE" IN SHAPE . . .

THE T WAVE



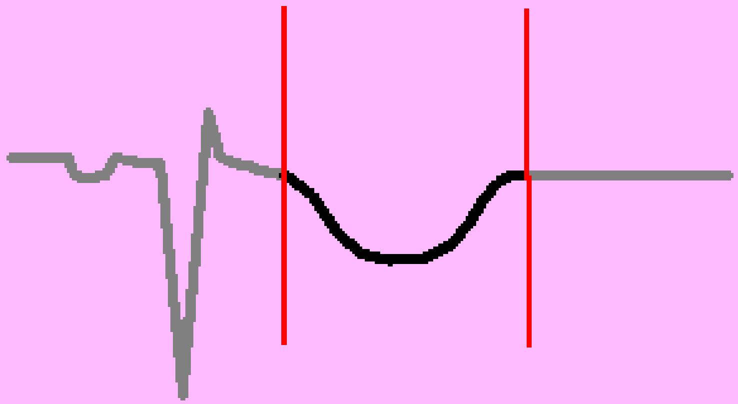
- SHOULD BE A "NICE," ROUNDED, CONVEX SHAPE
- SHOULD BE SYMMETRICAL

THE T WAVE



- SHOULD BE A "NICE," ROUNDED, CONVEX SHAPE
- SHOULD BE SYMMETRICAL
- SHOULD BE UPRIGHT IN ALL LEADS, EXCEPT AVR

THE T WAVE

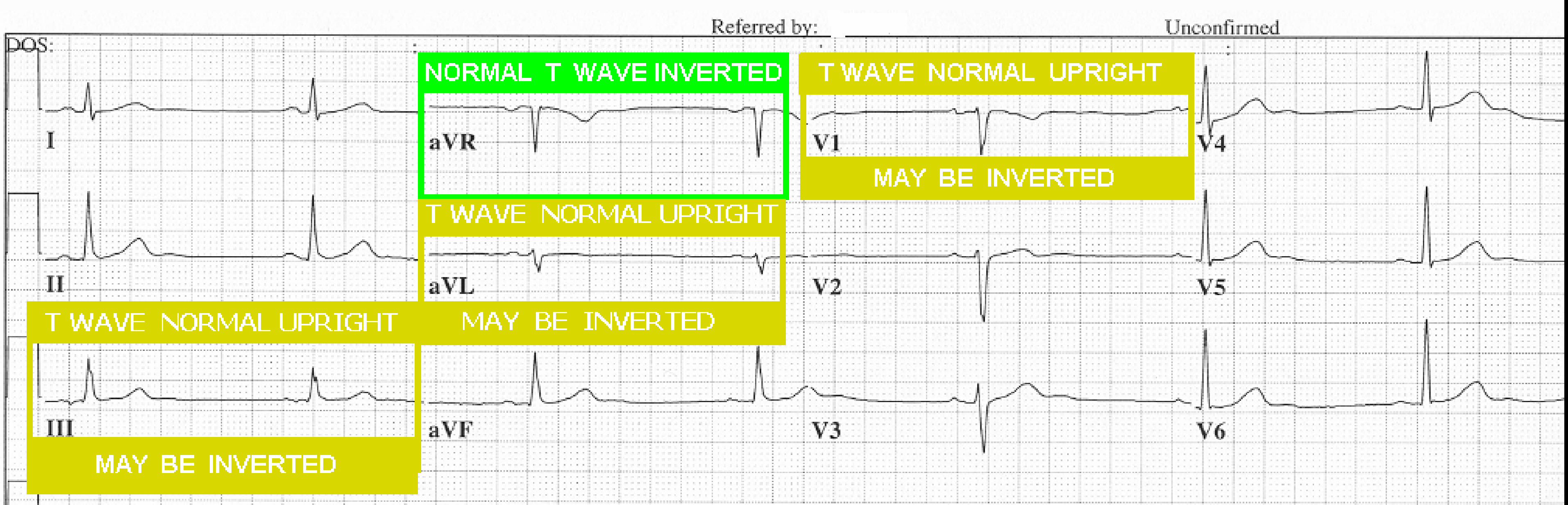


LEAD AVR

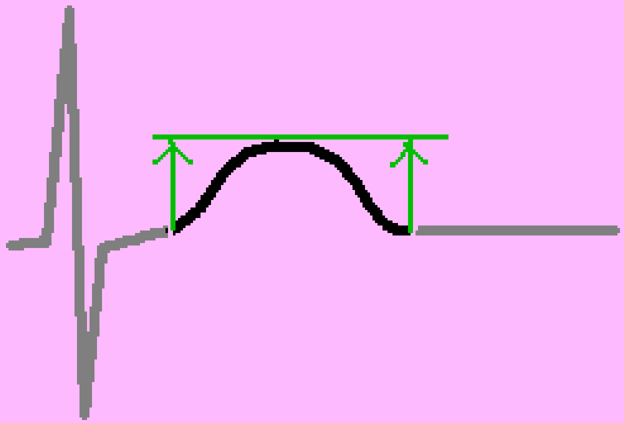
- REMEMBER, IN LEAD AVR *EVERYTHING* IS "UPSIDE-DOWN"

Normal Variants:
T Wave Inversion

Leads where the T WAVE may be INVERTED:



THE T WAVE



AMPLITUDE GUIDELINES:

- IN THE LIMB LEADS, SHOULD BE LESS THAN 1.0 mv (10 mm)
- IN THE PRECORDIAL LEADS, SHOULD BE LESS THAN 0.5 mv (5 mm)
- SHOULD NOT BE TALLER THAN R WAVE IN 2 OR MORE LEADS.

When QRS duration is NORMAL (< 120 ms):

NORMAL ST - T WAVES

- WHEN QRS WIDTH IS NORMAL (< 120 ms)

ASSESS:

- J POINT: ISOELECTRIC (or < 1 mm dev.)
- ST SEG: SLIGHT, POSITIVE INCLINATION
- T WAVE: UPRIGHT, POSITIVE

☞ *in EVERY LEAD EXCEPT aVR ! !*

ECG Indicators of ACS in Patients with *Normal Width* QRS Complexes (QRS duration < 120 ms)

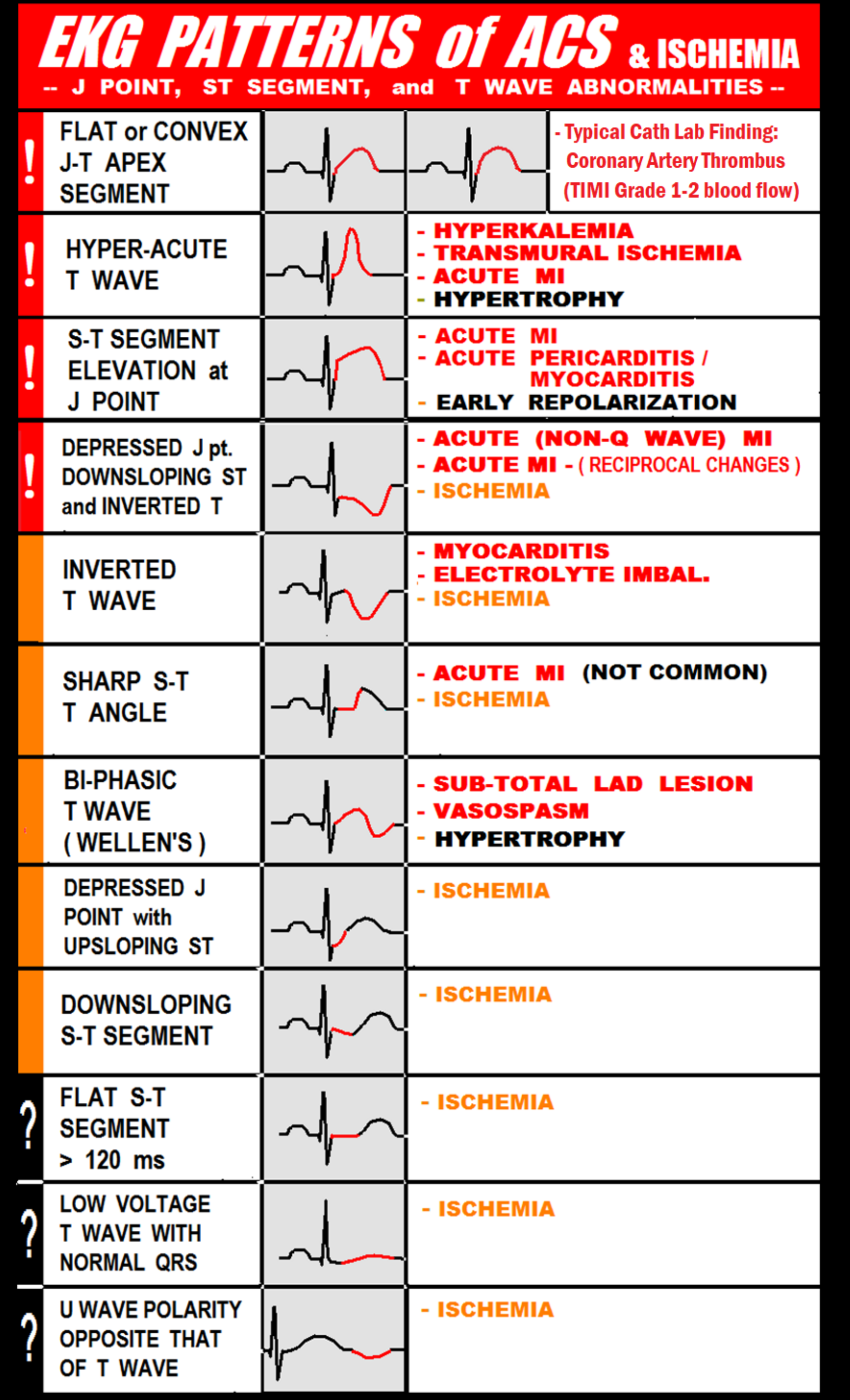
Multiple patterns of **ABNORMAL:**

- J Point
- ST-Segment
- T Wave

configurations may indicate ACS.

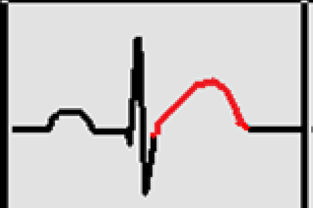
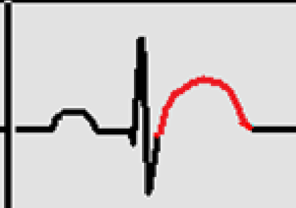
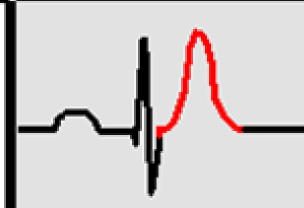
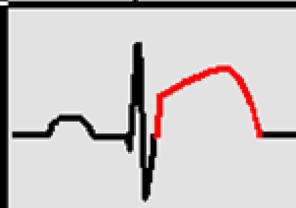
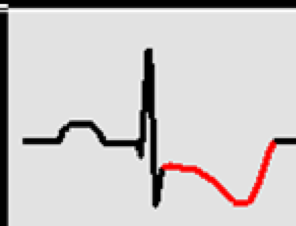
Remember, “IF IT’S NOT NORMAL, it’s *ABNORMAL !*”

BOOK PAGE: 83



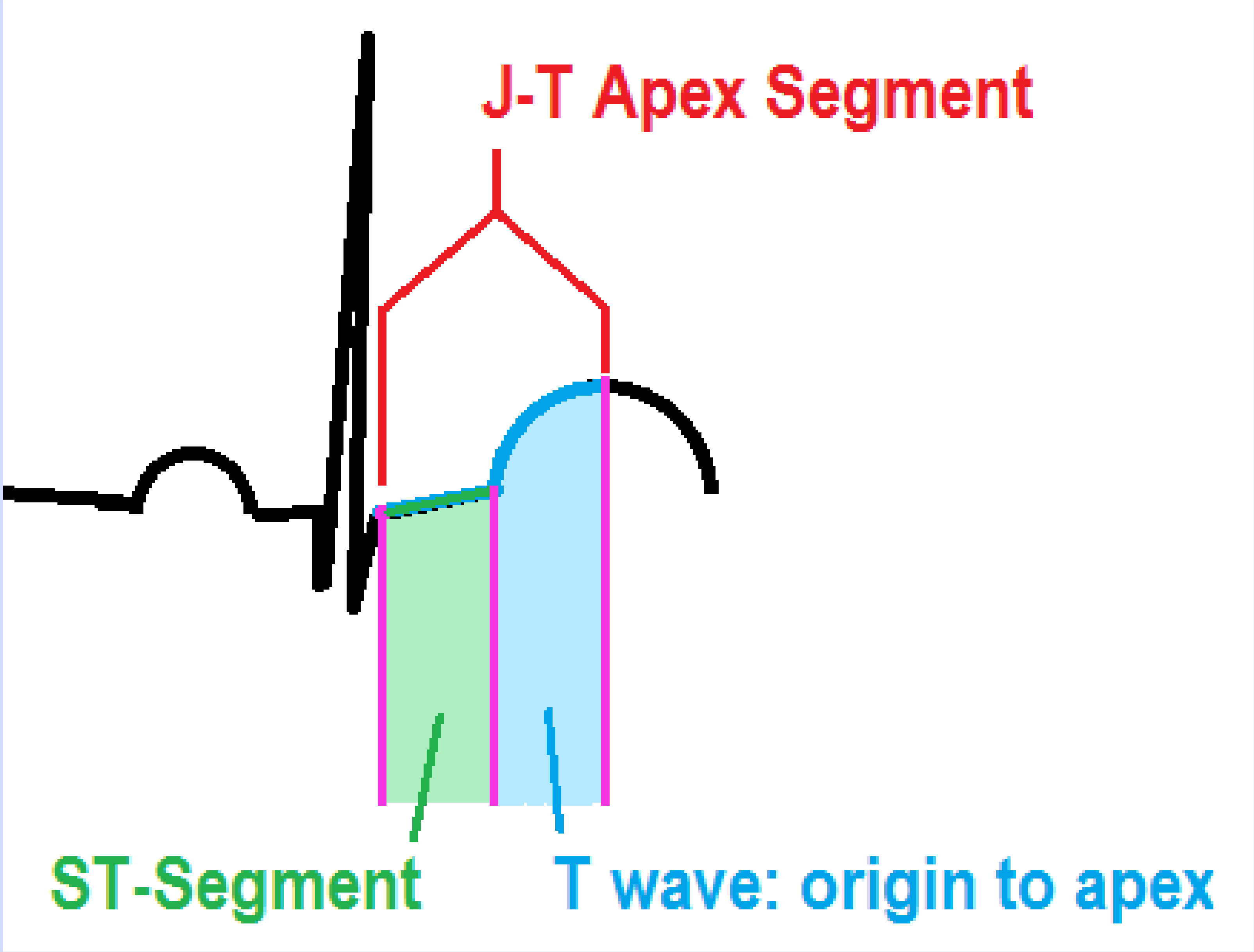
EKG PATTERNS of ACS & ISCHEMIA

-- J POINT, ST SEGMENT, and T WAVE ABNORMALITIES --

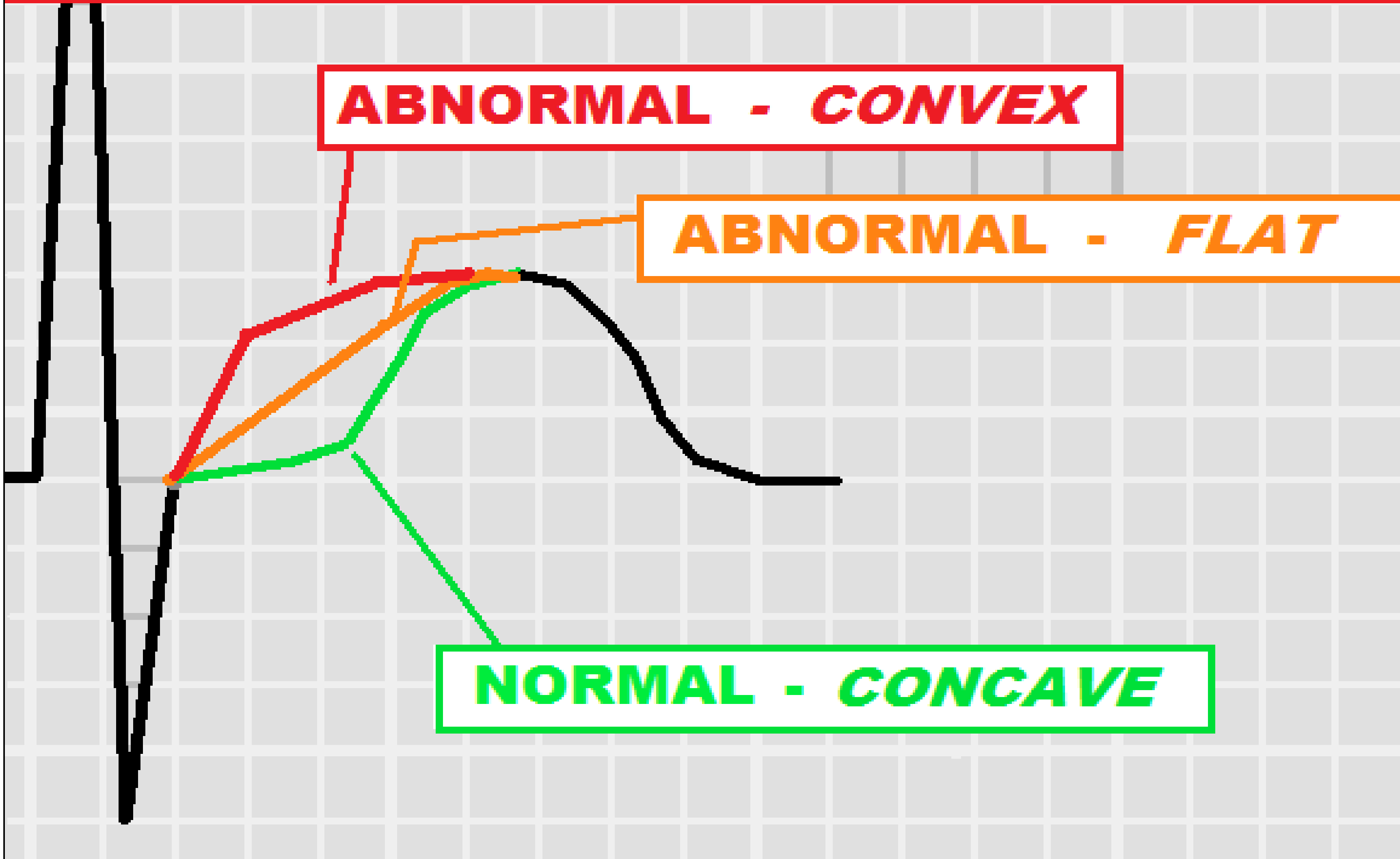
!	FLAT or CONVEX J-T APEX SEGMENT	 	- Typical Cath Lab Finding: Coronary Artery Thrombus (TIMI Grade 1-2 blood flow)
!	HYPER-ACUTE T WAVE		- HYPERKALEMIA - TRANSMURAL ISCHEMIA - ACUTE MI - HYPERTROPHY
!	S-T SEGMENT ELEVATION at J POINT		- ACUTE MI - ACUTE PERICARDITIS / MYOCARDITIS - EARLY REPOLARIZATION
!	DEPRESSED J pt. DOWNSLOPING ST and INVERTED T		- ACUTE (NON-Q WAVE) MI - ACUTE MI - (RECIPROCAL CHANGES) - ISCHEMIA

ECG Patterns associated with "EARLY PHASE MI:"

- ***J-T Apex abnormalities***
- ***Hyper-Acute T Waves***
- ***ST-T Wave Changes***


J-T Apex Segment
ST-Segment
T wave: origin to apex

J - T APEX SEGMENT VARIATIONS

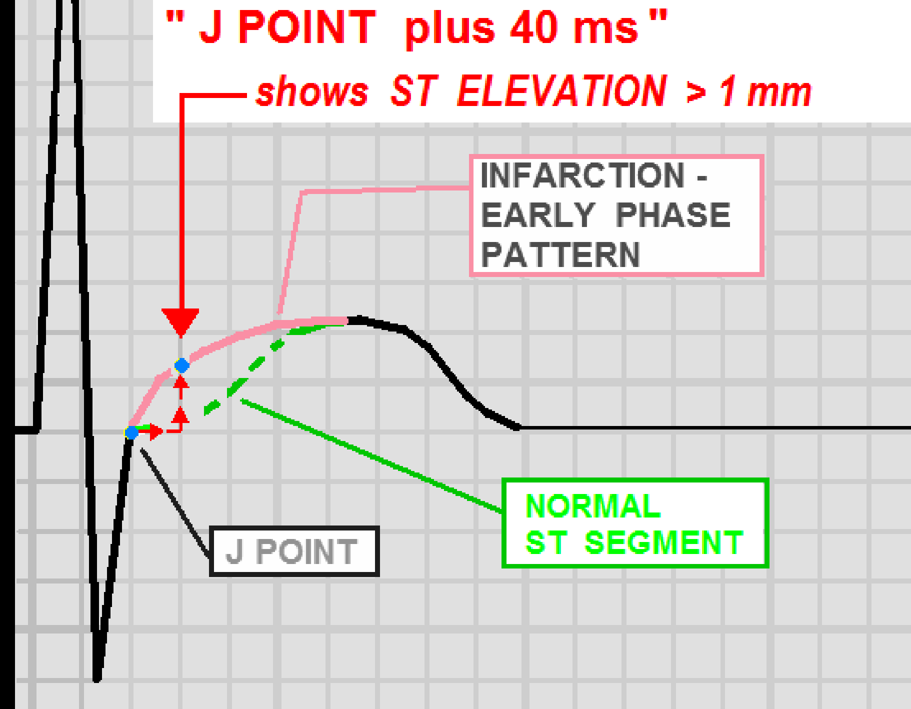


PATTERNS of EARLY INFARCTION

-- FLAT and CONVEX J-T APEX SEGMENTS

WHEN EVALUATING
for
ST SEGMENT
ELEVATION . . . .

From:
AMERICAN HEART ASSOCIATION
ACLS 2005 REVISIONS



During NORMAL STATES of PERFUSION, the J POINT is ISOELECTRIC and the ST SEGMENT has a CONCAVE appearance. When measured 40 ms beyond the J POINT (noted by the RED DOT), the ST SEGMENT elevation is less than 1mm.

During a 20 second BALLOON OCCLUSION of the patient's LAD during routine PTCA, the ST segment assumes a CONVEX shape. When measured 40 ms beyond the J POINT, the ST segment is elevated > 1 mm. This phenomenon is seen routinely in the cath lab prior to the occurance of ST ELEVATION at the J POINT during PTCA and STENTING.

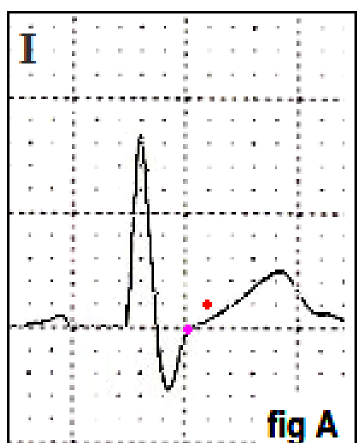


fig A

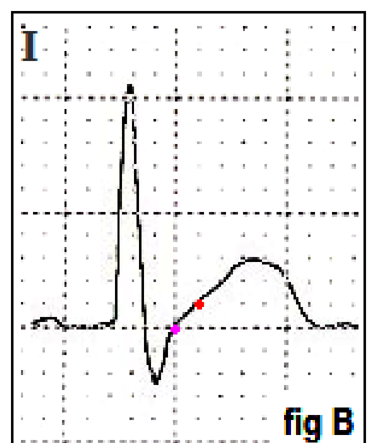
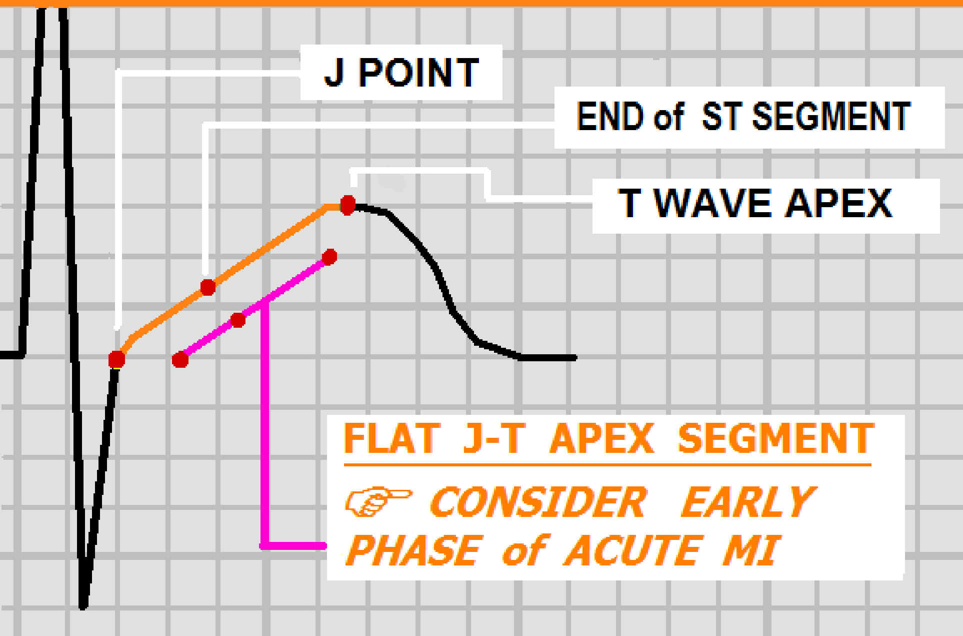


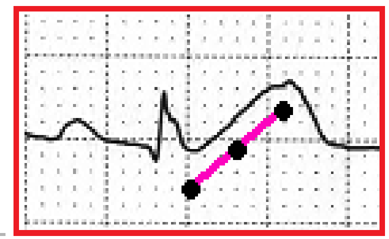
fig B

Both figures were recorded from a 54 year old male while resting (figure A), and during PTCA of the Left Anterior Descending artery (figure B).

ABNORMAL J-T APEX SEGMENT



LEAD II

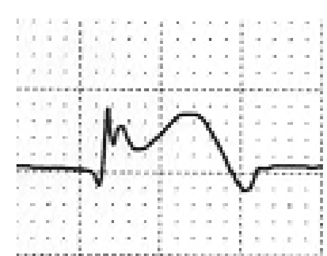


1839 hrs

41 y/o FEMALE

In ER C/O CHEST PAIN x 30 minutes.

- *FLAT J-T APEX SEGMENT*
- *NO ST ELEVATION at J POINT !*



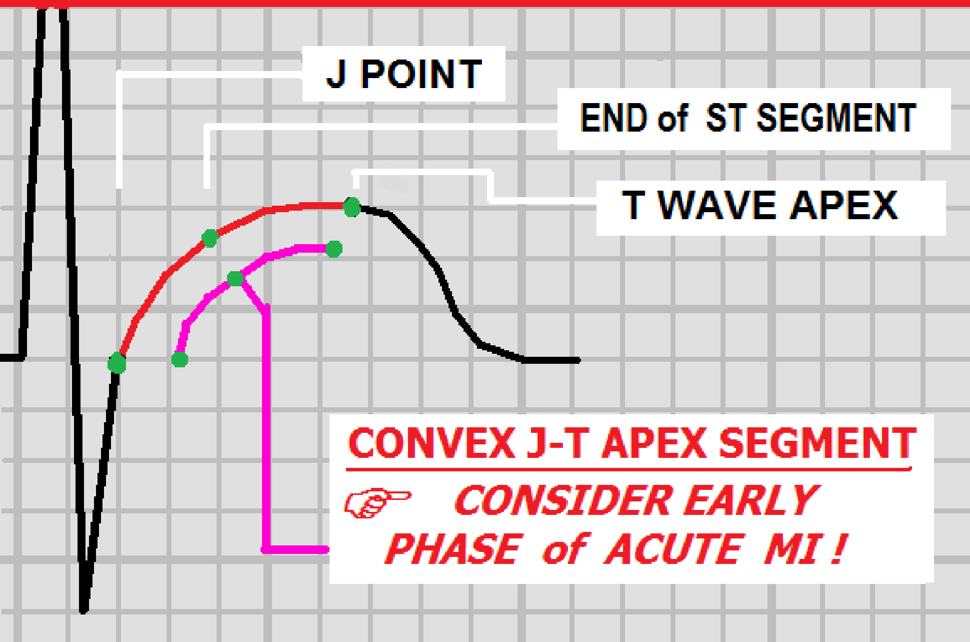
1850 hrs

STEMI - INFERIOR WALL

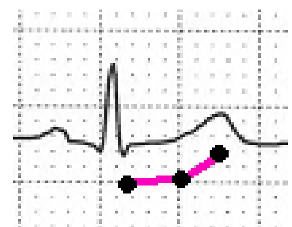
11 MINUTES LATER, S-T ELEVATION at the J POINT IS NOTED.

- CATH LAB FINDINGS: TOTAL OCCLUSION of the RIGHT CORONARY ARTERY

ABNORMAL J-T APEX SEGMENT

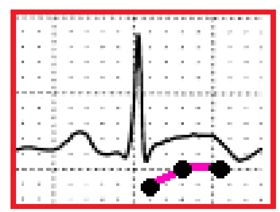


LEAD I



53 y/o MALE

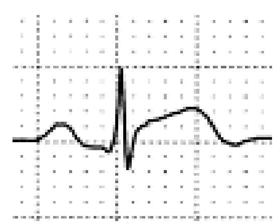
1 yr. PRIOR TO MI
NORMAL EKG
CONCAVE J - T APEX SEGMENT



0732 hrs

STEMI LATERAL WALL

- *CONVEX J-T APEX SEGMENT*
- MINIMAL ST ELEVATION at J POINT



0747 hrs

15 MINUTES LATER, S-T ELEVATION at the J POINT IS NOTED.

- CATH LAB FINDINGS: TOTAL OCCLUSION OF CIRCUMFLEX ARTERY

CASE STUDY: ABNORMAL J-T APEX SEGMENTS

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

56 y/o MALE presents to ED with complaint of "INTERMITTENT SUBSTERNAL & SUB-EPIGASTRIC PRESSURE" x 3 HOURS. PMHx of ESOPHAGEAL REFLUX. NO other significant past medical history.

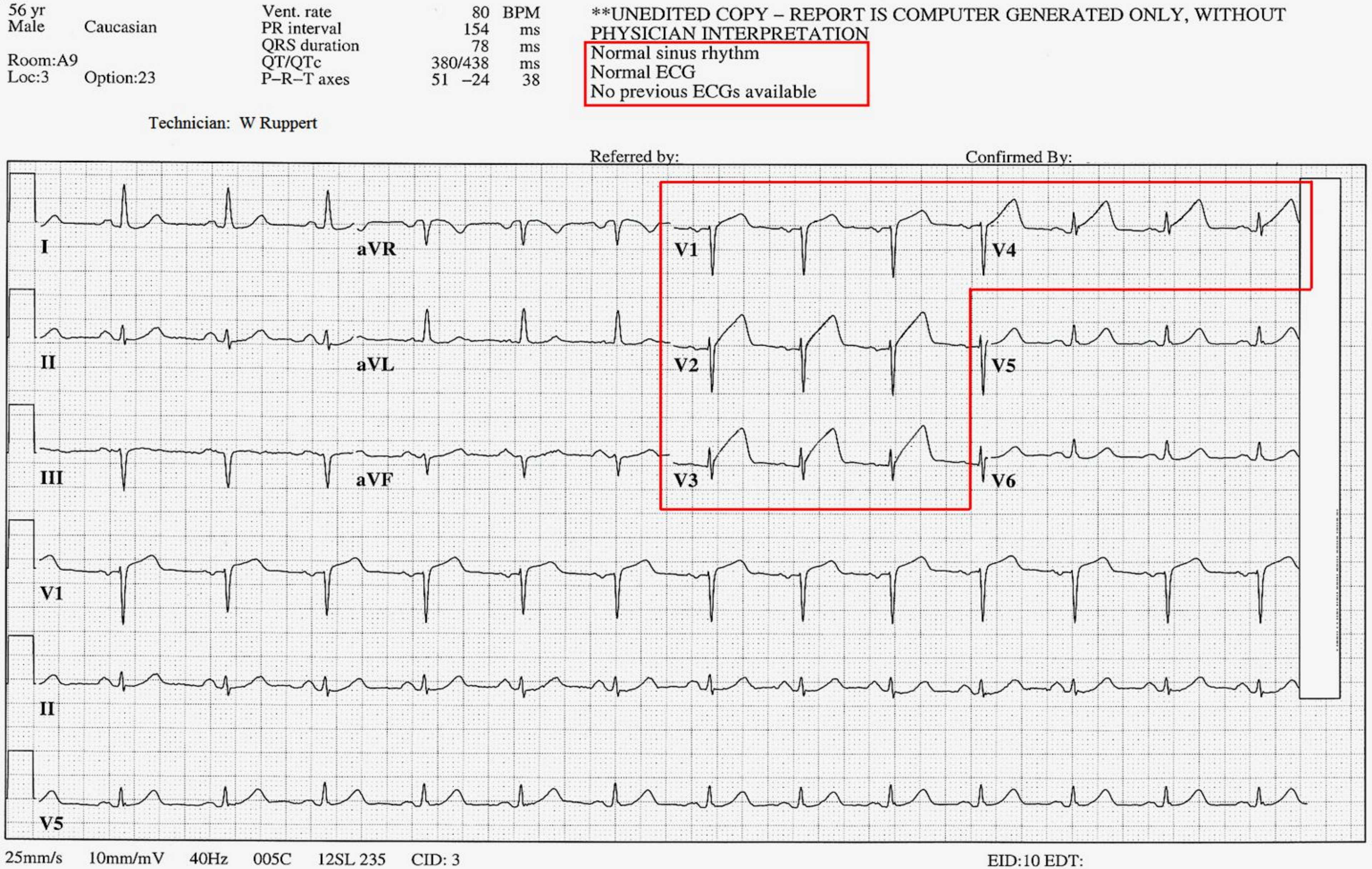
RISK FACTOR PROFILE:

- FAMILY HISTORY - father died of MI at age 62
- ☑ PREVIOUS CIGARETTE SMOKER - quit 15 years ago.
- ☑ CHOLESTEROL - DOES NOT KNOW; "never had it checked."
- ☑ OBESITY

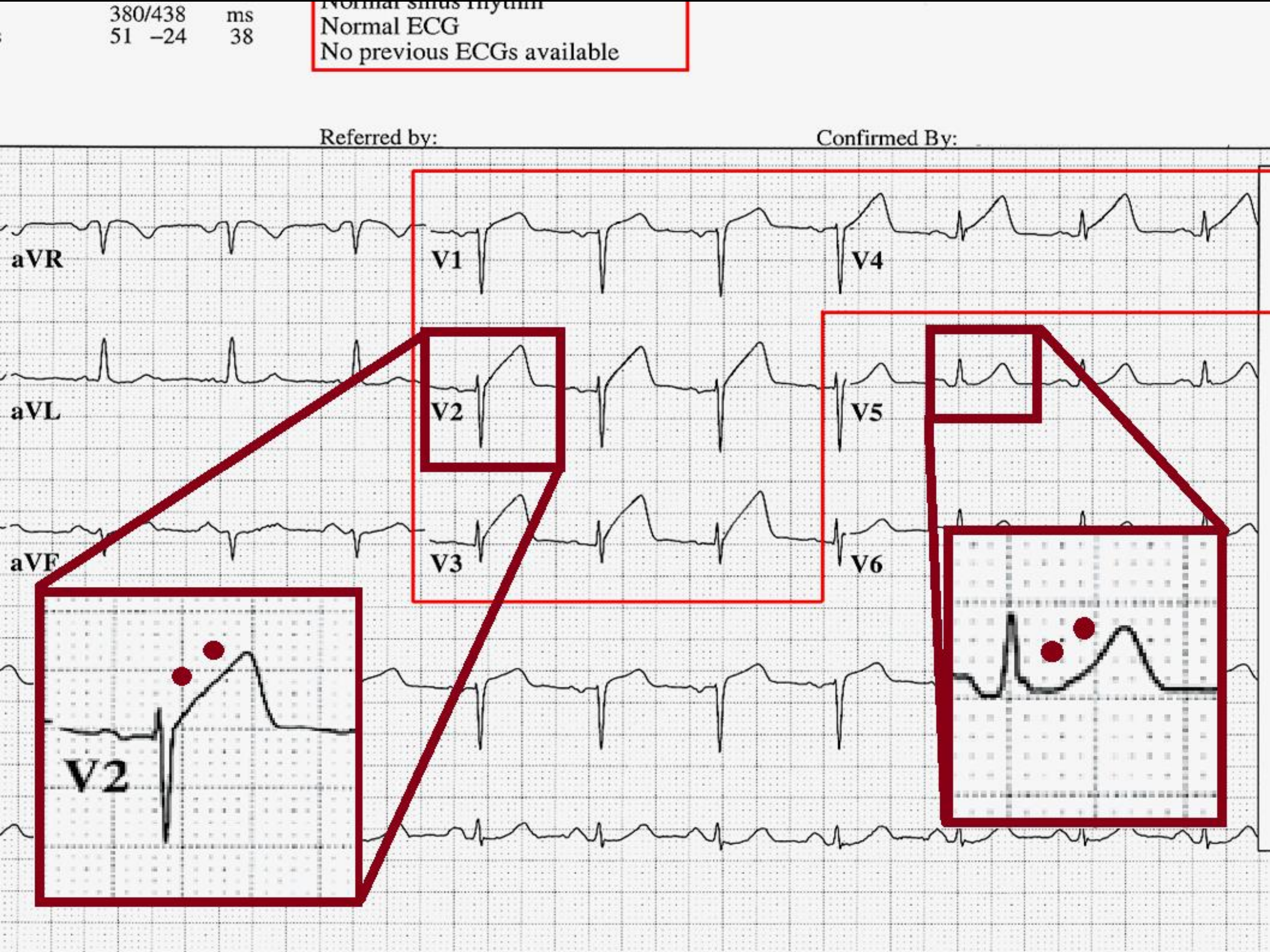
PHYSICAL EXAM: Patient supine on exam table, mildly anxious, currently complaining of "mild indigestion," skin is warm, pale, dry; REST OF EXAM is UNREMARKABLE.

VITAL SIGNS: BP 142/94, P 80, R 20, SAO2 98%

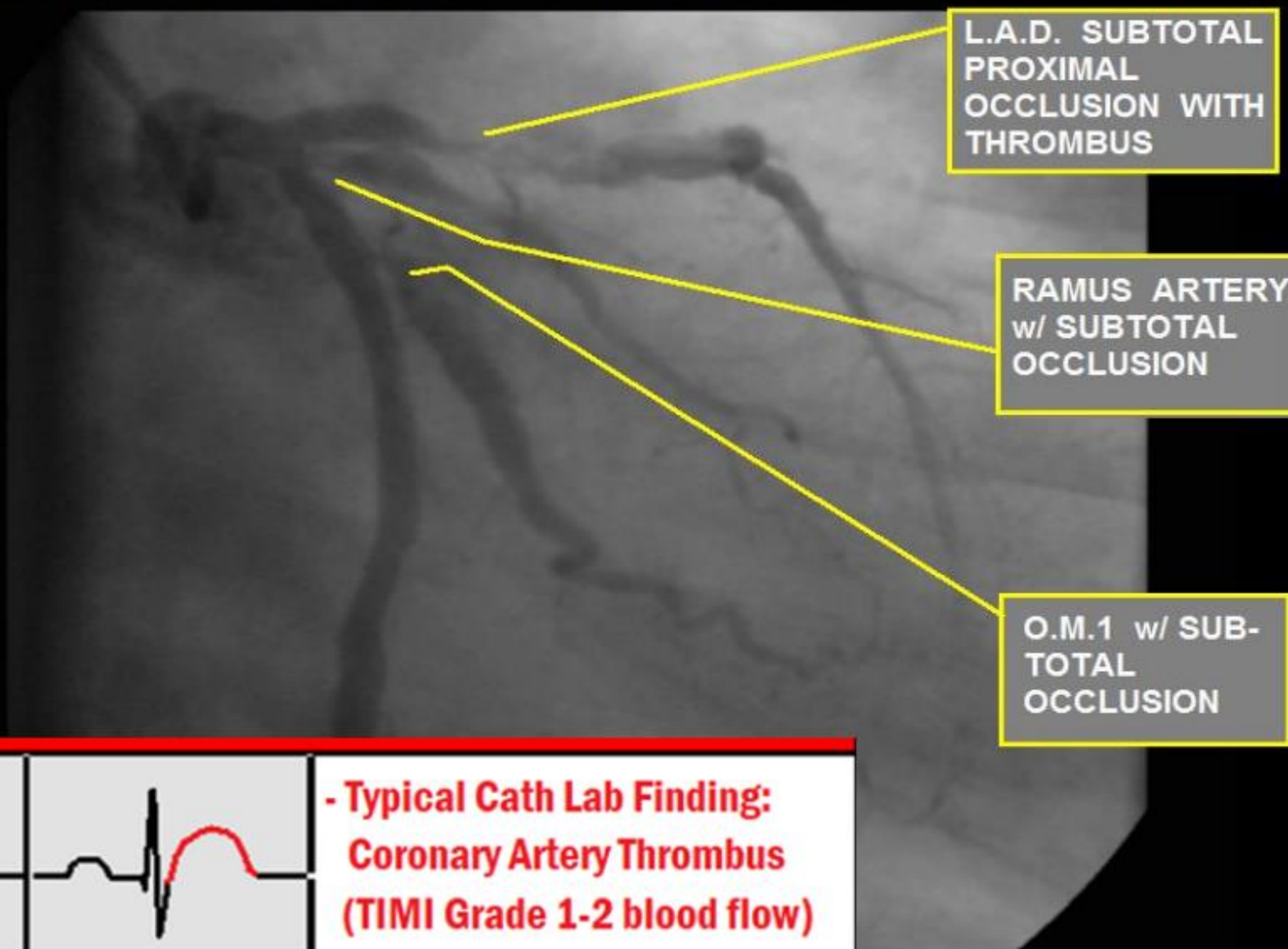
LABS: JUST OBTAINED, RESULTS NOT AVAILABLE YET.

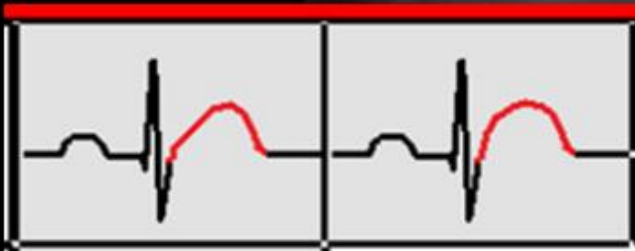


ECG COMPUTER DOES NOT NOTICE THE CONVEX J-T APEX SEGMENTS !


380/438 ms
51 –24 38
Normal sinus rhythm
Normal ECG
No previous ECGs available
Referred by:
Confirmed By:
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
V2

CASE STUDY: 56 y/o male with INTERMITTENT "CHEST HEAVINESS" . . . .



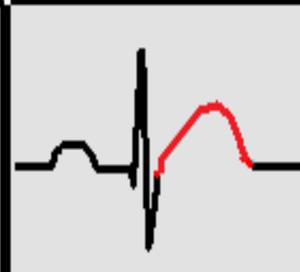
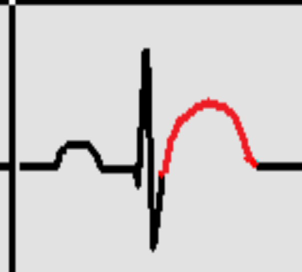
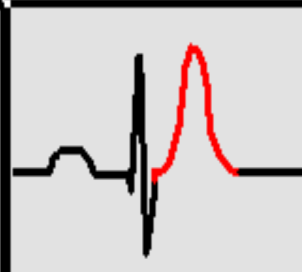
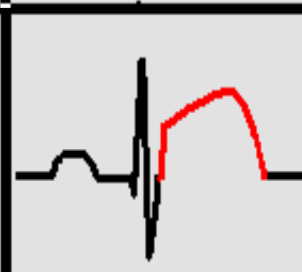
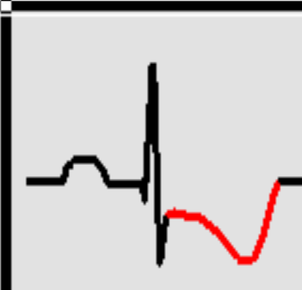


- Typical Cath Lab Finding: Coronary Artery Thrombus (TIMI Grade 1-2 blood flow)

TREATMENT PLAN : EMERGENCY CORONARY ARTERY BYPASS SURGERY (4 VESSEL)

PATTERNS of ACS & ISCHEMIA

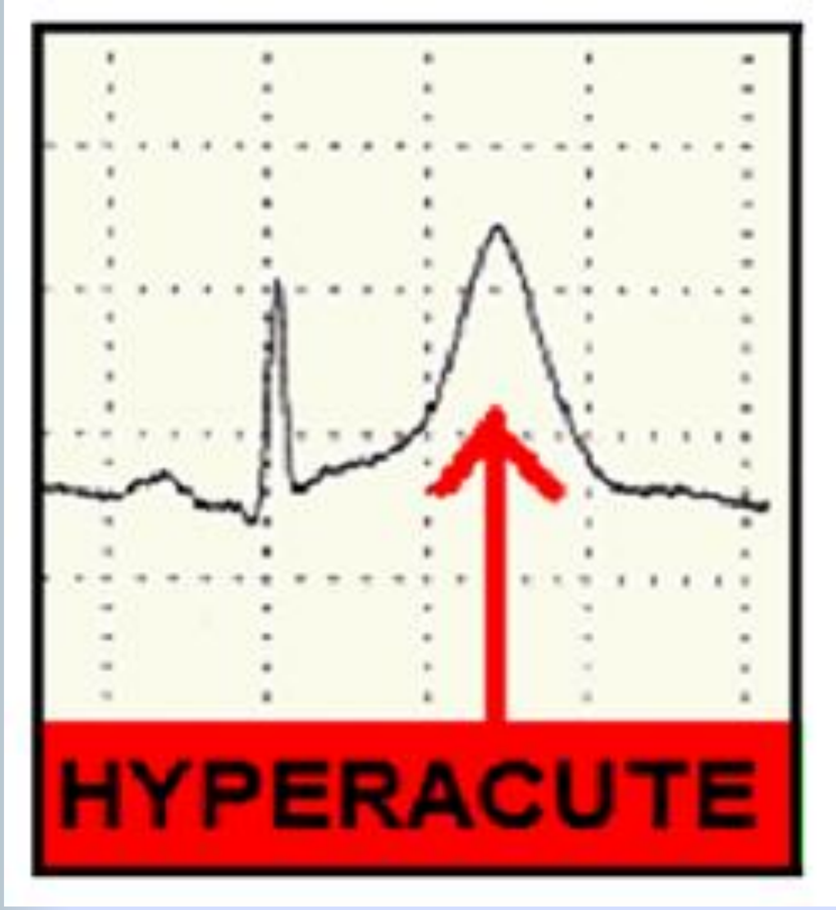
-- J POINT, ST SEGMENT, and T WAVE ABNORMALITIES --

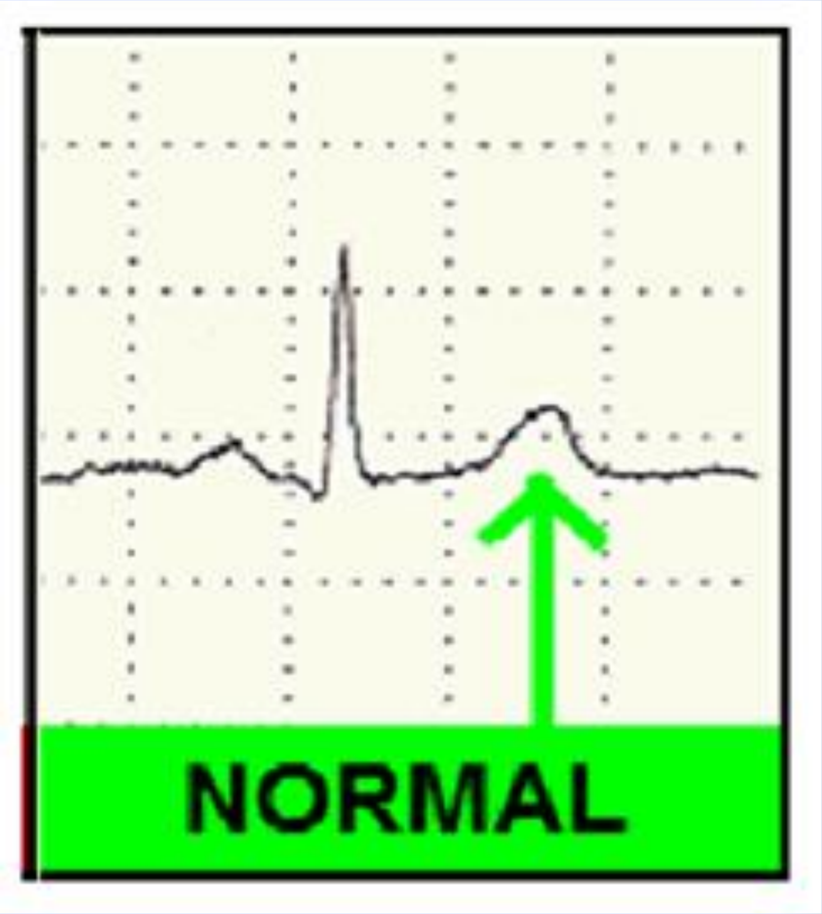
!	FLAT or CONVEX J-T APEX SEGMENT	 	***ACUTE MI*** *EARLY PHASE*
!	HYPER-ACUTE T WAVE		***ACUTE MI*** *EARLY PHASE*
!	S-T SEGMENT ELEVATION at J POINT		***ACUTE MI***
!	DEPRESSED J pt. DOWNSLOPING ST and INVERTED T		- **ACUTE (NON-Q WAVE) MI** - **ACUTE MI** - (RECIPROCAL CHANGES) - **ISCHEMIA**

HYPER-ACUTE T WAVES - COMMON ETIOLOGIES:

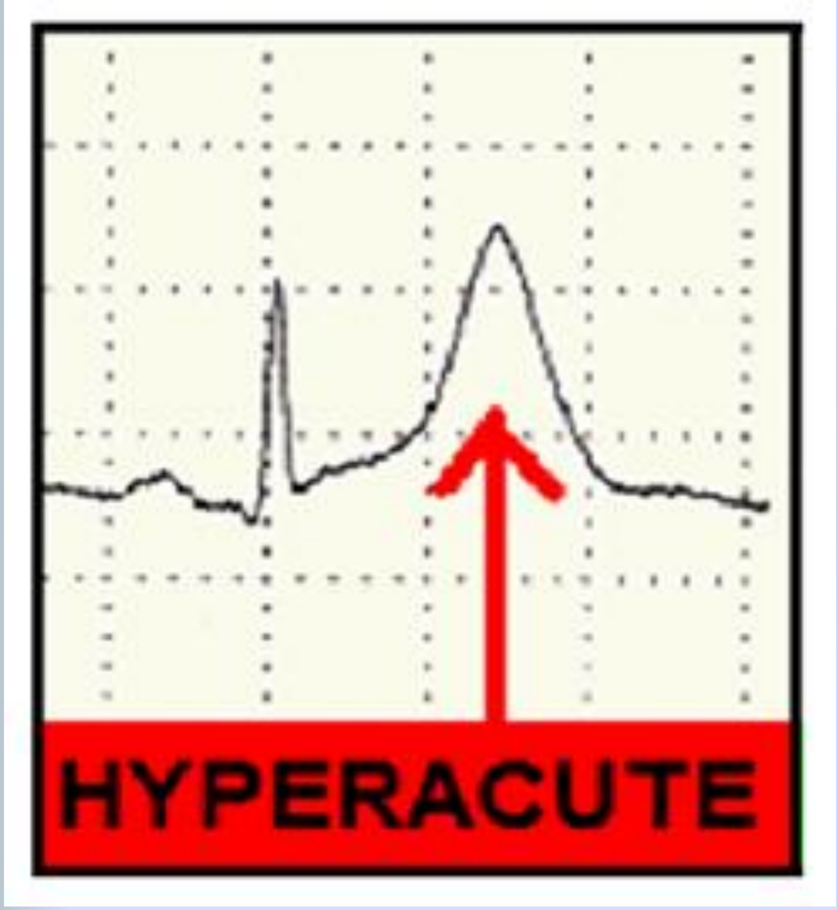
CONDITION:	SEE PAGE(S):
HYPERKALEMIA	XX - XX
ACUTE MI	XX - XX
TRANS-MURAL ISCHEMIA	XX - XX
HYPERTROPHY	XX - XX

T waves should not be HYPERACUTE



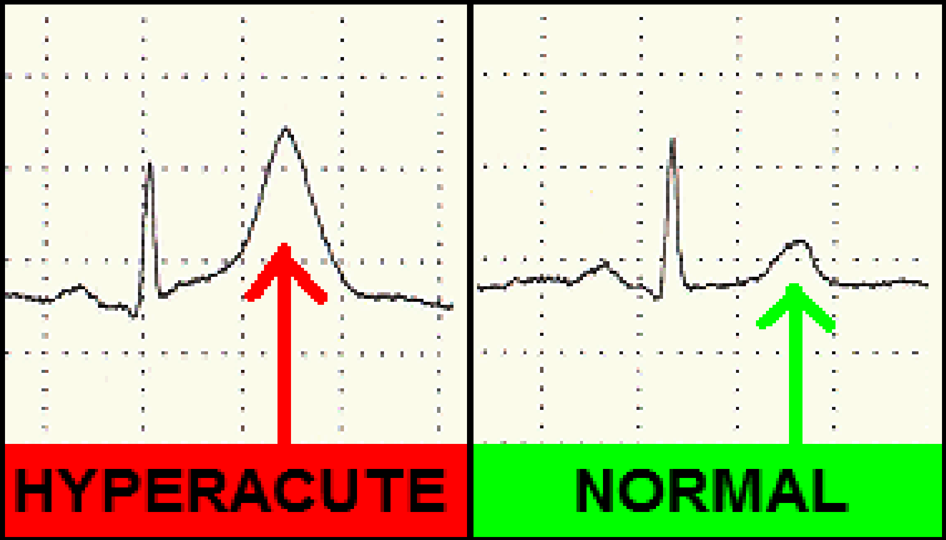


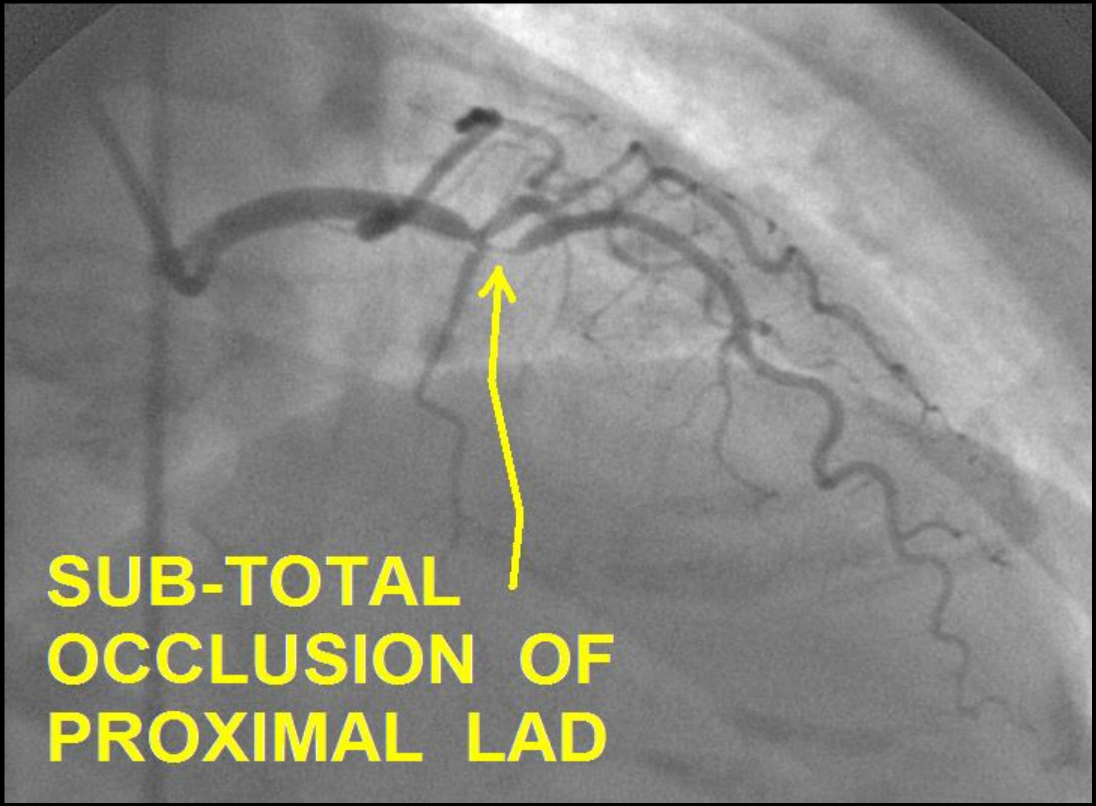
HYPERACUTE T Waves may indicate:



- **Early phase Acute MI**
- **Transmural ischemia** (usually seen in one region of the ECG)
- **Hyperkalemia** (seen globally across ECG)
- **Hypertrophy**

HYPERACUTE T WAVES





Helpful Clue: Hyper-Acute T Waves

- **GLOBAL Hyper-acute T Waves** (in leads viewing multiple myocardial regions / arterial distributions) **favors HYPERKALEMIA**

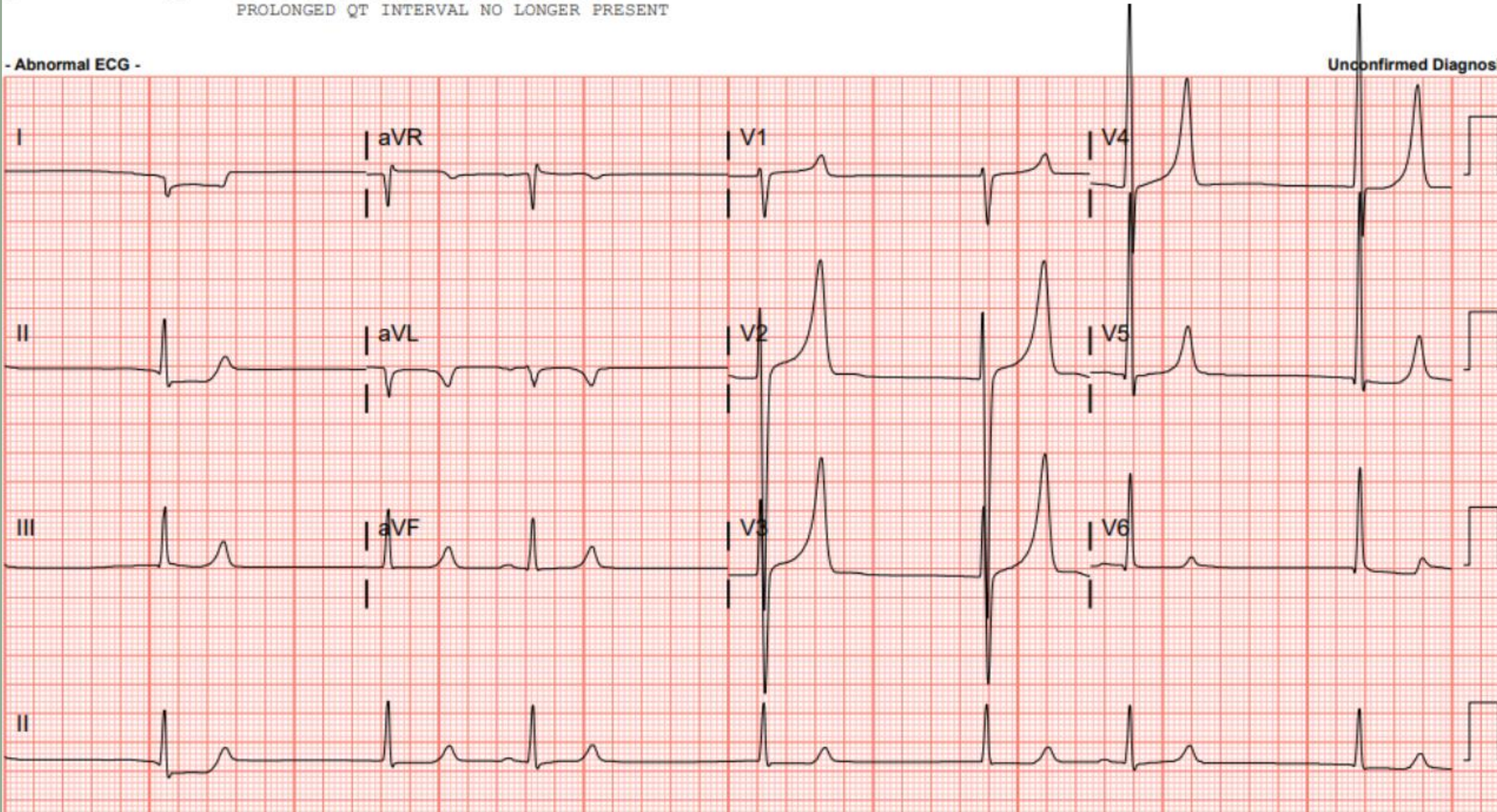
Rate	39	Right and left arm electrode reversal, interpretation assumes no reversal
PR	500	Sinus bradycardia
QRSd	117	Atrial premature complexes
QT	549	LVH with IVCD and secondary repol abnrm
QTc	443	Anterior ST elevation, probably due to LVH
--Axis--		COMPARED TO ECG 02/24/2020 21:46:48
P	0	SINUS BRADYCARDIA NOW PRESENT
QRS	96	INTRAVENTRICULAR CONDUCTION DELAY NOW PRESENT
T	117	ST (T WAVE) DEVIATION NOW PRESENT
		PROLONGED QT INTERVAL NO LONGER PRESENT

Req Provider: ONIER VILLARREA

K+ = 7.9

- Abnormal ECG -

Unconfirmed Diagnosis



ID: 23-Nov-

REGIONAL MEDICAL CENTER

55years
Female Caucasian

Room:

Vent. rate 57 bpm
PR interval 150 ms
QRS duration 102 ms
QT/QTc 472/459 ms
P-R-T axes 76 70 58

Sinus bradyc a
Possible Left atrial enlargement
Borderline ECG

ER ATTENDING REVIEW
NO STEMI
TIME 1311

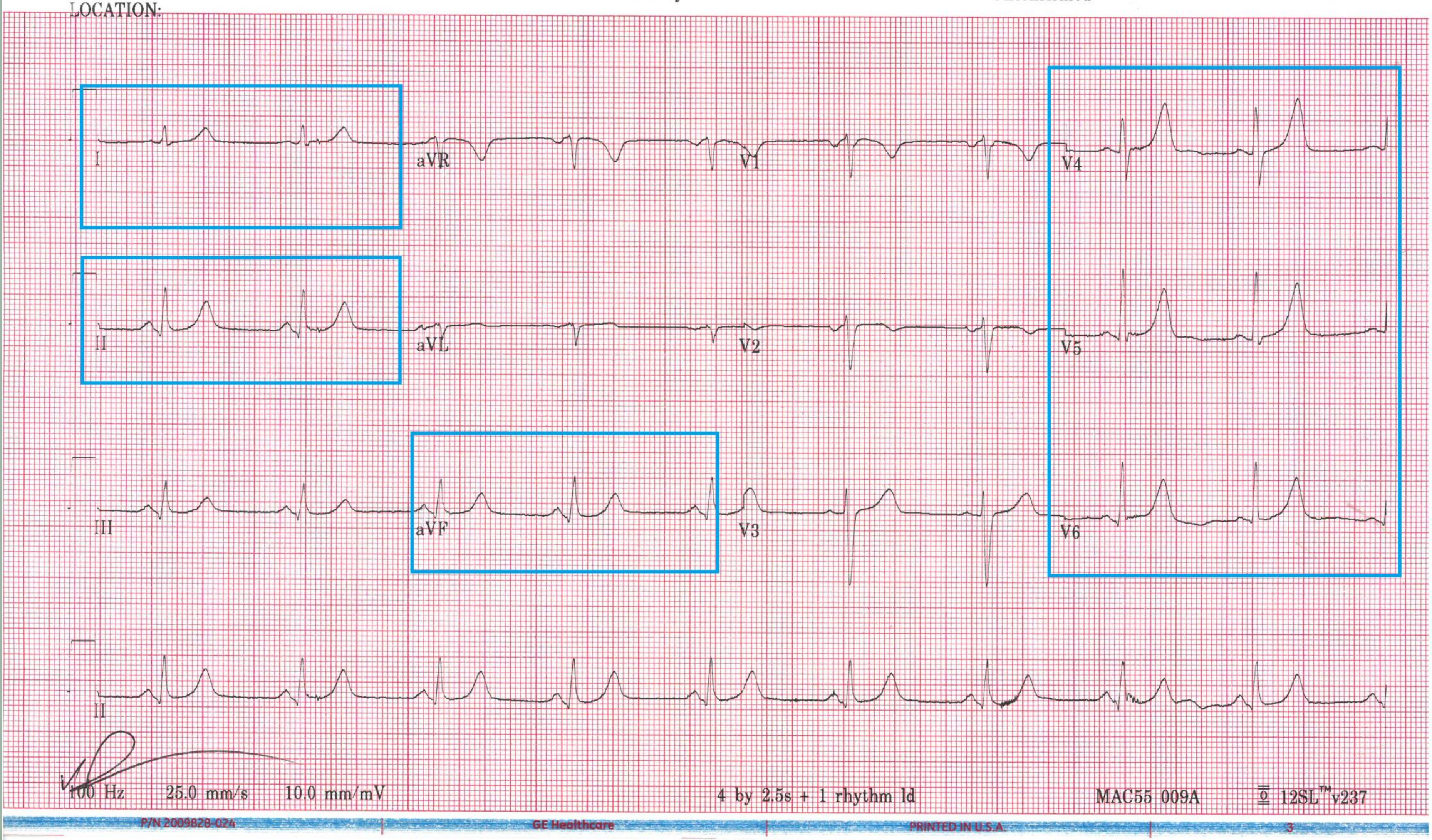
K+ = 6.7

Technician:
Test ind:

Referred by:

Unconfirmed

LOCATION:



Helpful Clue: Hyper-Acute T Waves

- **GLOBAL Hyper-acute T Waves** (in leads viewing multiple myocardial regions / arterial distributions) **favors HYPERKALEMIA**

- **Hyper-acute T Wave noted in ONE ARTERIAL DISTRIBUTION** (Anterior / Lateral / Inferior) favors TRANSMURAL ISCHEMIA / Early Phase Acute MI

CASE STUDY: HYPERACUTE T WAVES

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

30 y/o male presents to ER via EMS, c/o sudden onset of dull chest pain x 40 min. Pain level varies, not effected by position, movement or deep inspiration. No associated symptoms.

RISK FACTOR PROFILE: NONE. CHOLESTEROL UNKNOWN.

PHYSICAL EXAM: Patient is supine on exam table, CAO x 4, anxious, restless, skin pale, cool, dry. Patient c/o chest pressure, "7" on 1 - 10 scale, uneffected by position, movement, deep inspiration. Lungs clear. HS: NL S1, S2, no rubs, murmurs, gallops

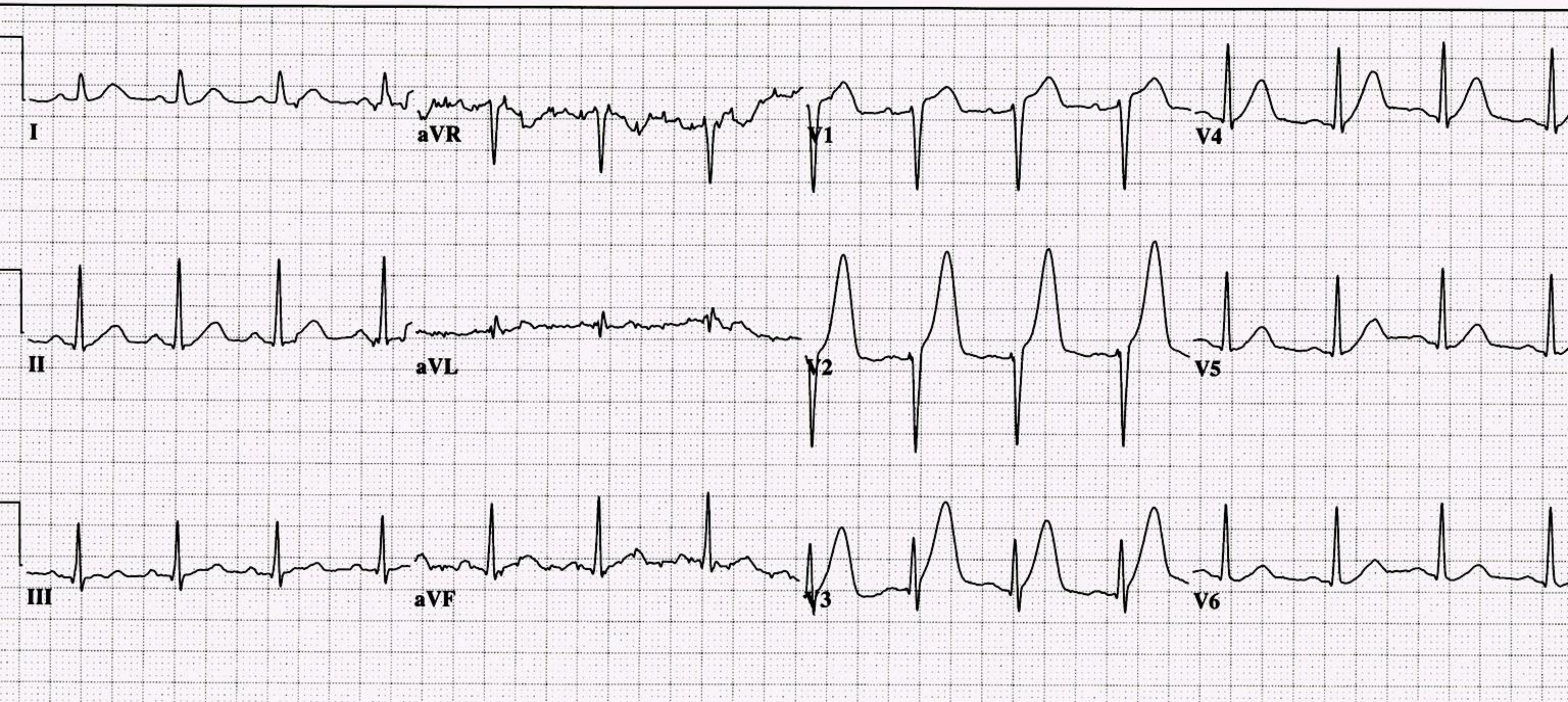
VITAL SIGNS: BP 136/88 P 90 R 20 SAO2 98%

DIAGNOSTIC TESTING: 1st TROPONIN I - ultra: <0.07

30 yr		Vent. rate	88	BPM
Male	Black	PR interval	164	ms
		QRS duration	90	ms
Room: ER		QT/QTc	370/447	ms
Loc:	Option:	P–R–T axes	61 62	53

Normal sinus rhythm
Normal ECG
No previous ECGs available

← NOTE COMPUTER INTERPRETATION



30 yr
Male Black

Room: ER
Loc: Option:

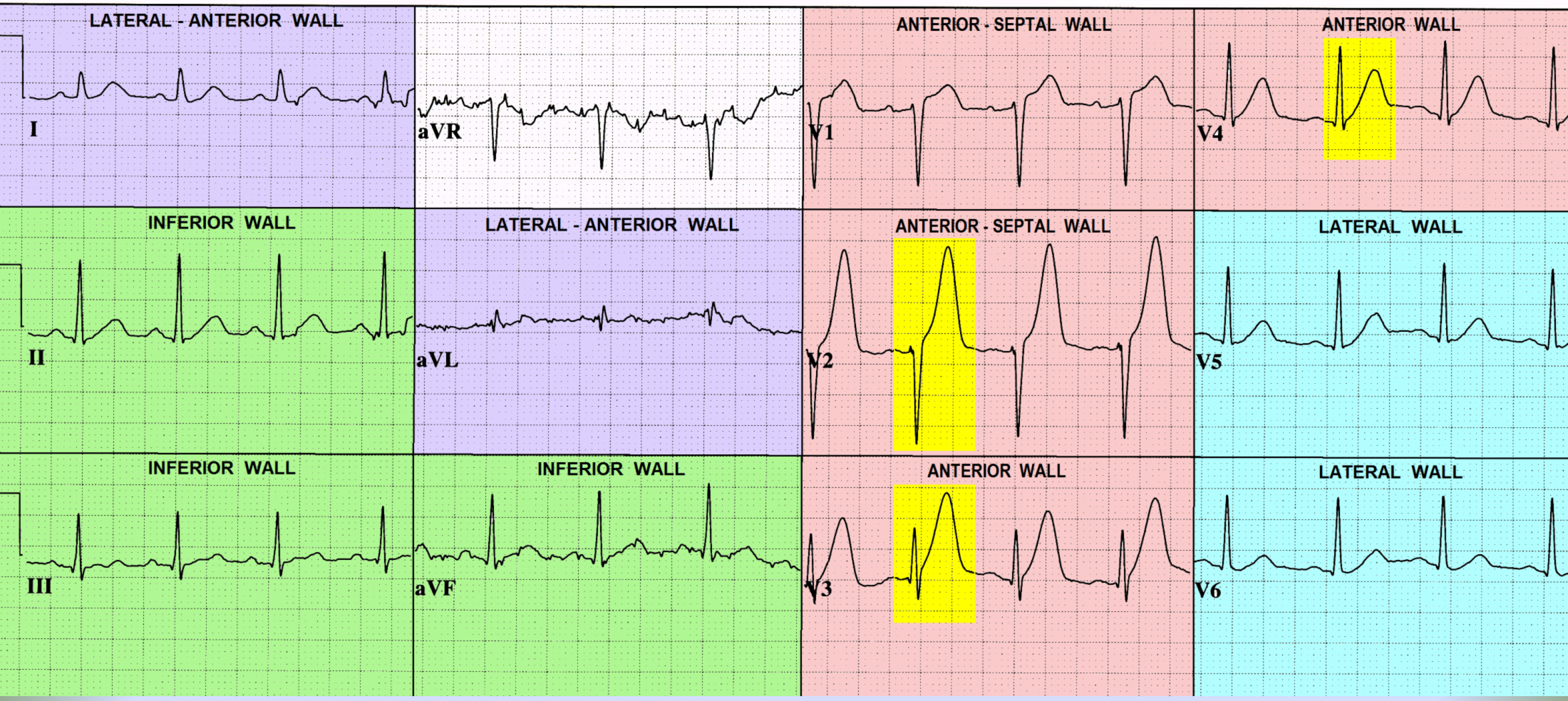
Vent. rate	88	BPM
PR interval	164	ms
QRS duration	90	ms
QT/QTc	370/447	ms
P–R–T axes	61 62	53

Normal sinus rhythm
Normal ECG
No previous ECGs available

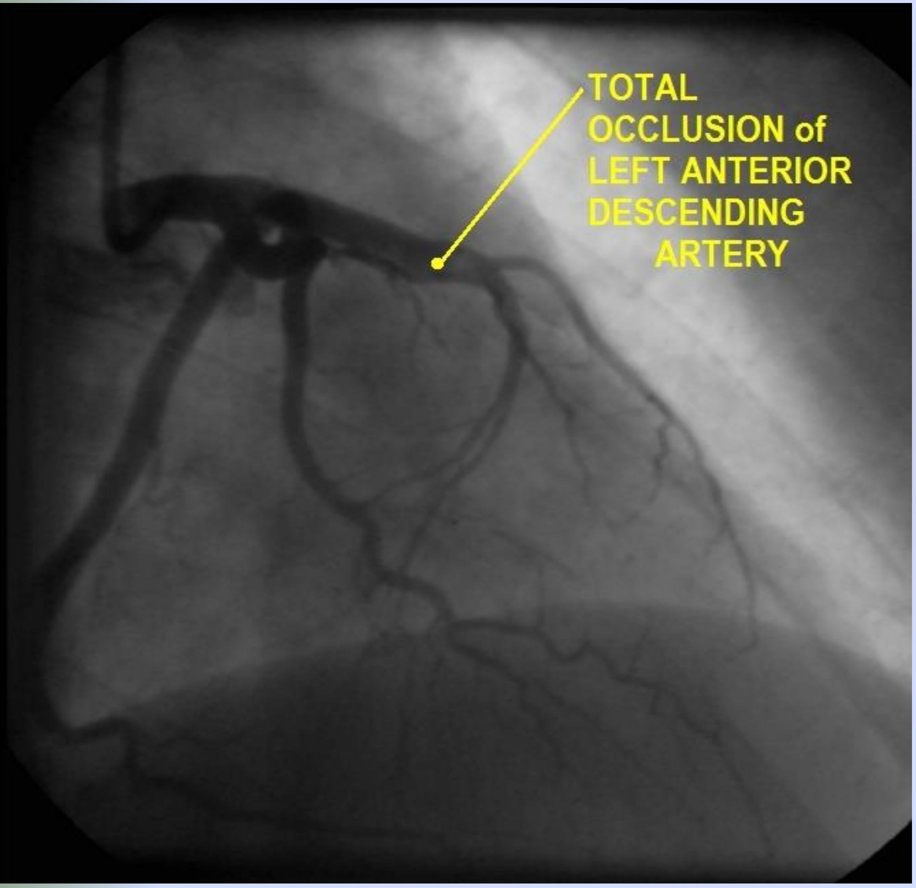
HIGHLIGHTED AREAS = HYPERACUTE T WAVES

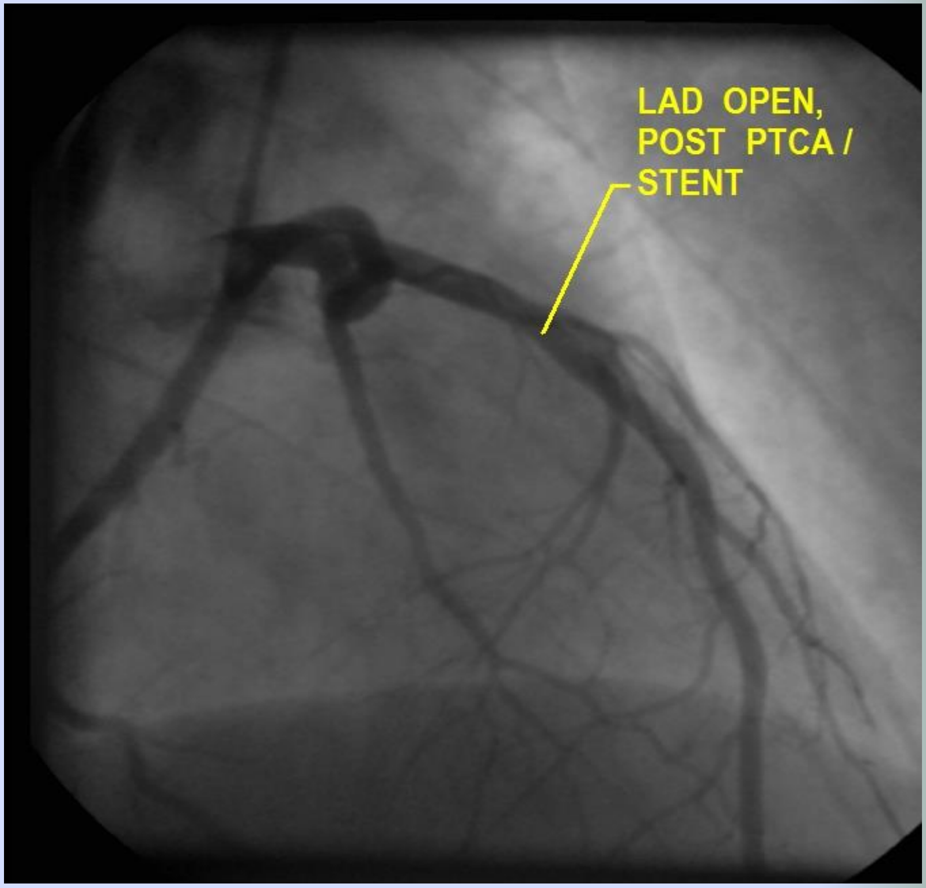
CORONARY ARTERIAL DISTRIBUTIONS:

V1 - V4 = LEFT ANTERIOR DESCENDING (LAD)
I, AVL = DIAGONAL (DIAG) off the LAD or OBTUSE MARGINAL (OM) off CIRCUMFLEX (CX)
V5, V6 = CIRCUMFLEX
II, III, AVF = RIGHT CORONARY ARTERY or CX



Cath Lab findings:





Dynamic ST-T Wave Changes:

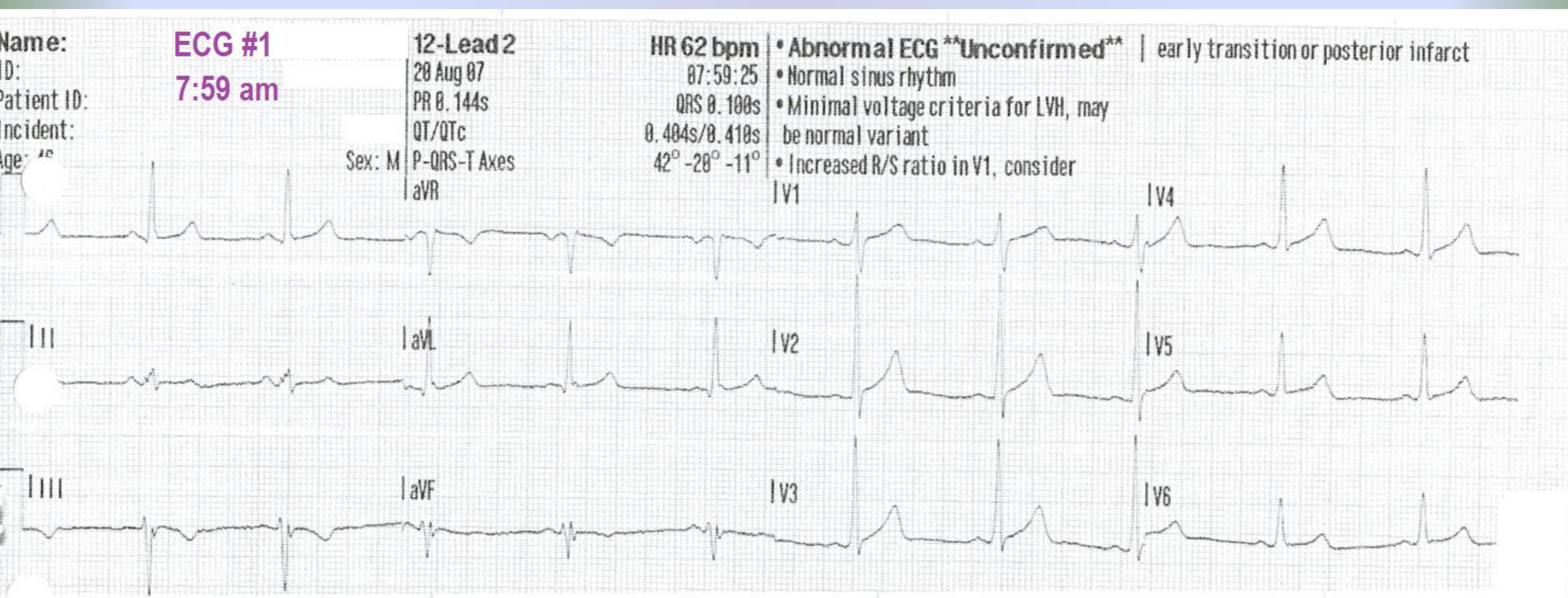
- Other than HEART RATE related variations (which affect intervals), ***J Points, ST-Segments and T Waves SHOULD NOT CHANGE.***

Dynamic ST-T Wave Changes:

- Other than HEART RATE related variations (which affect intervals), ***J Points, ST-Segments and T Waves SHOULD NOT CHANGE.***
- **When changes to J Points, ST-Segments and/or T waves are NOTED, consider EVOLVING MYOCARDIAL ISCHEMIA and/or EARLY PHASE MI, until proven otherwise.**

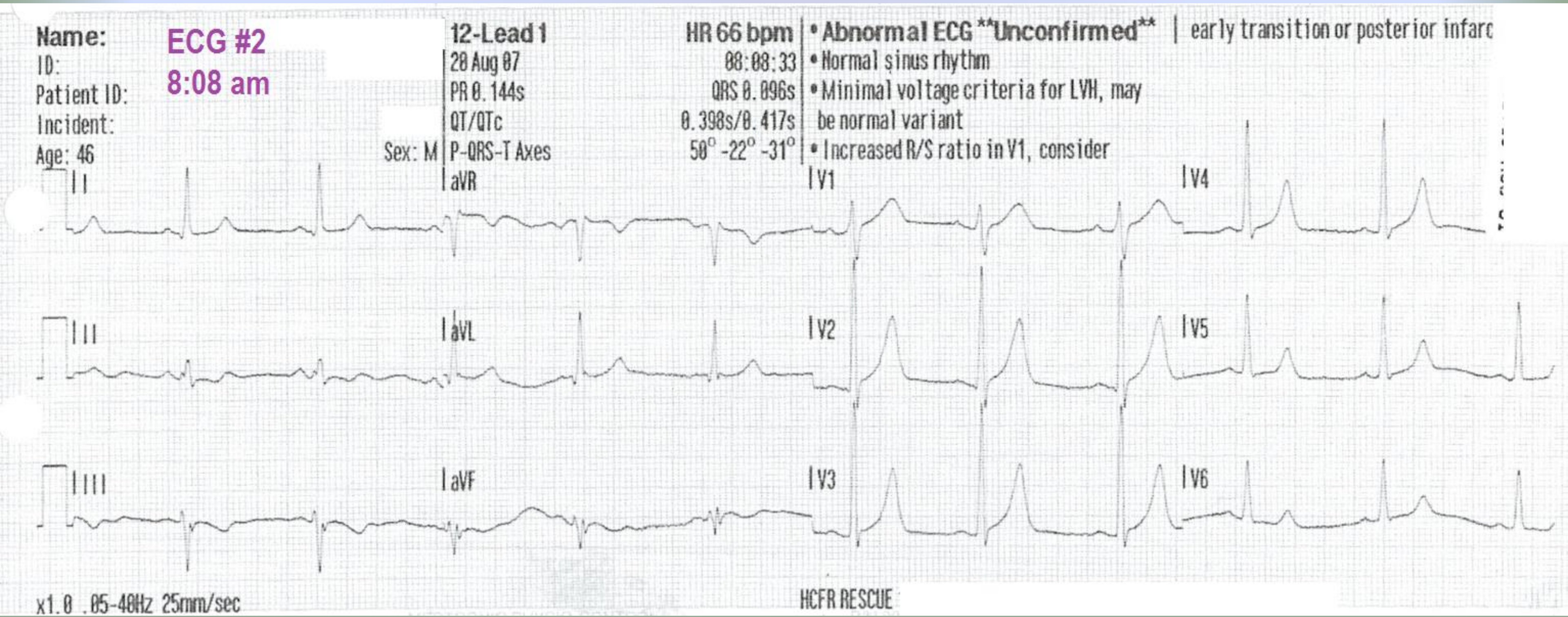
46 year old male

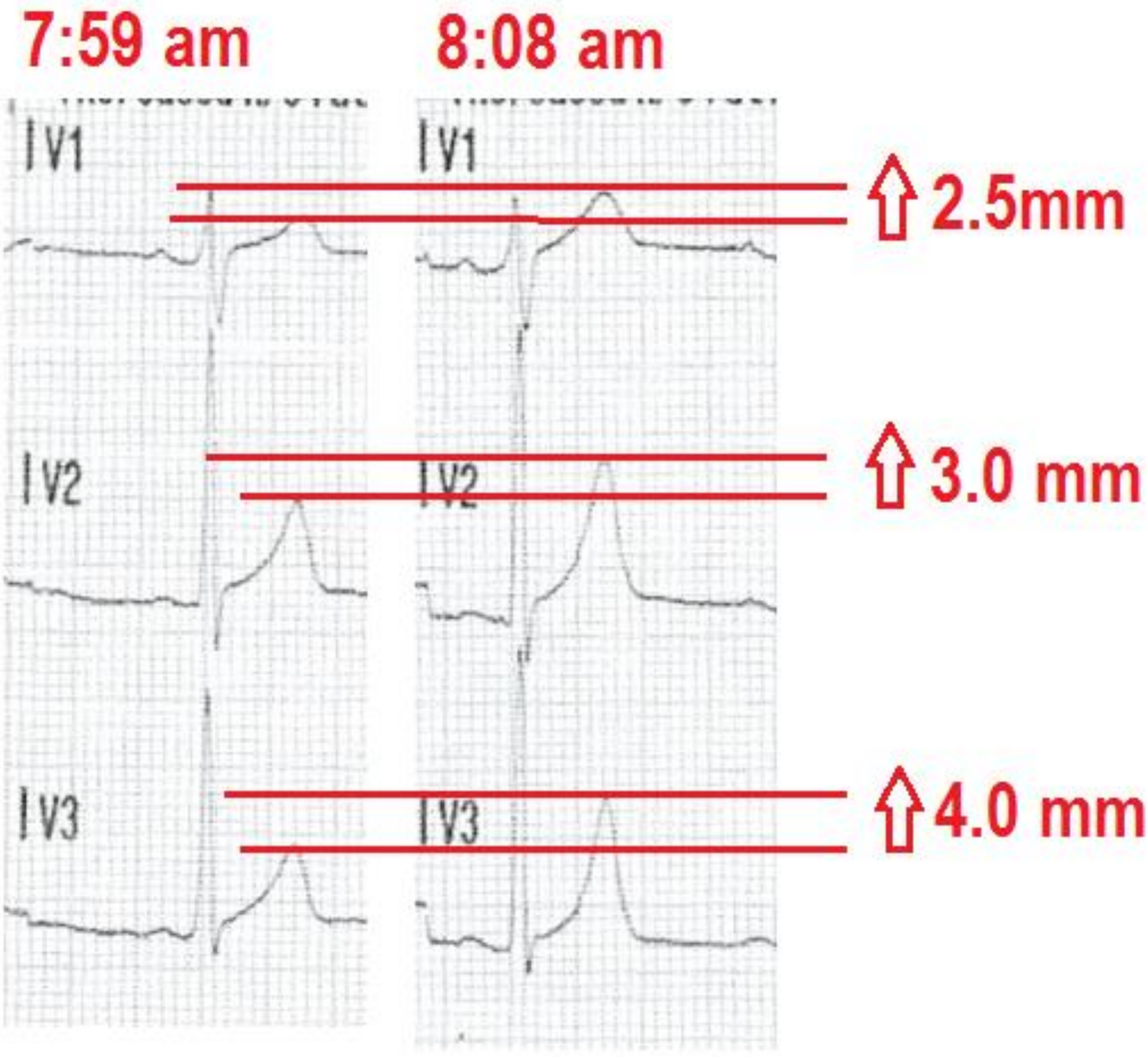
- Exertional dyspnea X “several weeks”
- Intermittent chest pressure X last 3 hours. Currently pain free.



46 year old male: ECG 1

- Chest pressure has returned, "5" on 1-10 scale.   2nd ECG obtained due to "change in symptoms":

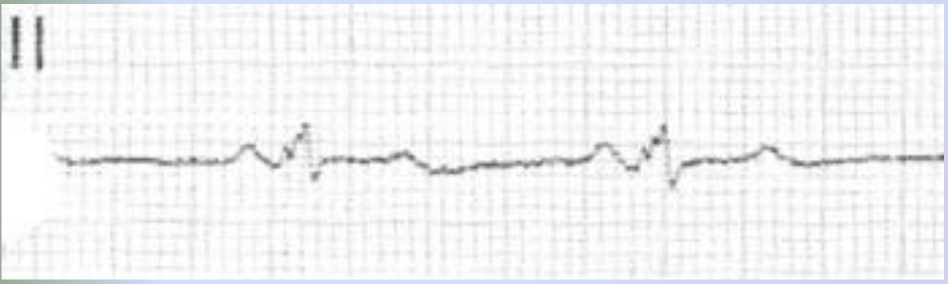



7:59 am
8:08 am
V1
V2
V3
2.5mm
3.0 mm
4.0 mm

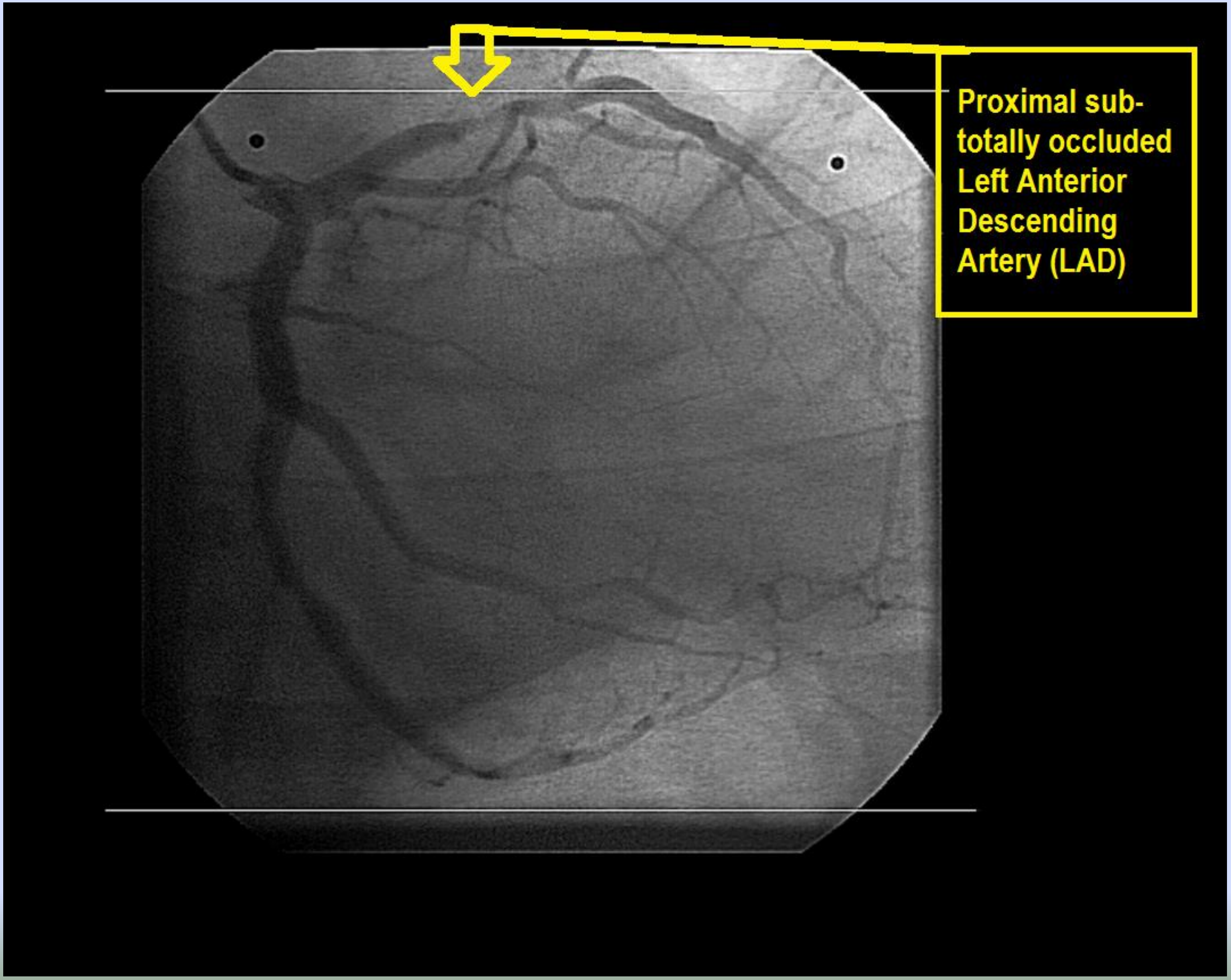
ST-Segment Depression

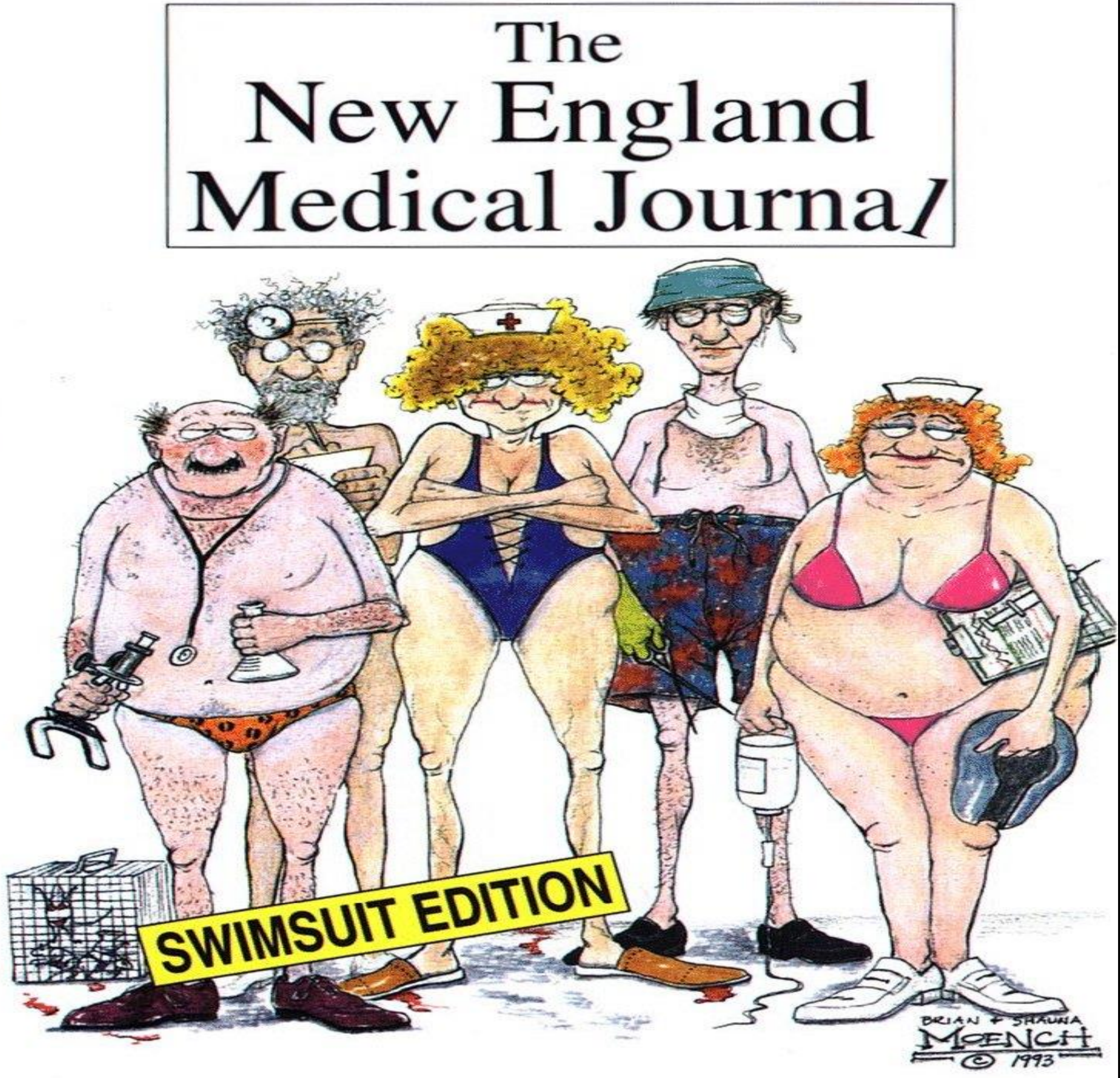
7:59 am

8:08 am



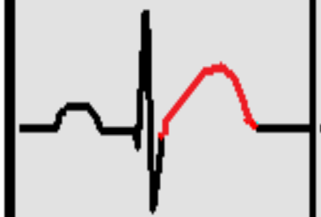
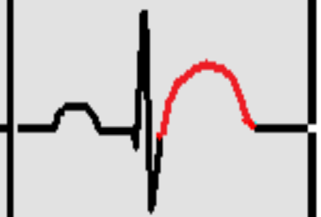
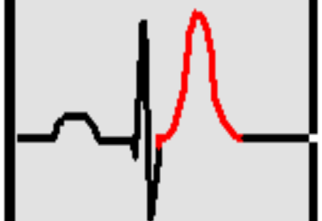
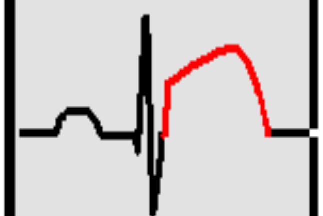
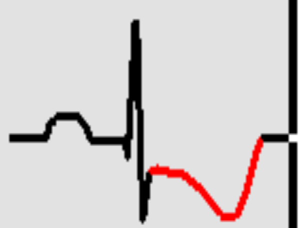
Cath Lab Angiography:




The New England Medical Journal
SWIMSUIT EDITION
BRIAN & SHAUNA MOENCH
© 1993

PATTERNS of ACS & ISCHEMIA

-- J POINT, ST SEGMENT, and T WAVE ABNORMALITIES --

!	FLAT or CONVEX J-T APEX SEGMENT			ACUTE MI EARLY PHASE
!	HYPER-ACUTE T WAVE			ACUTE MI EARLY PHASE
!	S-T SEGMENT ELEVATION at J POINT			ACUTE MI
!	DEPRESSED J pt. DOWNSLOPING ST and INVERTED T			- ACUTE (NON-Q WAVE) MI - ACUTE MI - (RECIPROCAL CHANGES) - ISCHEMIA

ECG CRITERIA for DIAGNOSIS of STEMI:

(ST ELEVATION @ J POINT)

***LEADS V2 and V3**:

MALES AGE 40 and up ------ 2.0 mm

(MALES LESS THAN 40------- 2.5 mm)

FEMALES ---------------------- 1.5 mm

ALL OTHER LEADS: 1.0 mm or more,

in TWO or more

CONTIGUOUS LEADS

* **P. Rautaharju et al, "Standardization and Interpretation of the ECG," JACC 2009;(53)No.11:982-991**

STEMI Criteria for 18 Lead ECGs:

Right-Sided Chest Leads

(V3R – V6R): 0.5 mm

Posterior Chest Leads

(V7 – V9): 0.5 mm

*** P. Rautaharju et al, "Standardization and Interpretation of the ECG," JACC 2009;(53)No.11:982-991**

Abnormal ST Elevation Criteria: ACC/AHA 2009 "Standardization and Interpretation of the ECG, Part VI Acute Ischemia and Infarction," Galen Wagner, et al

Recommendations

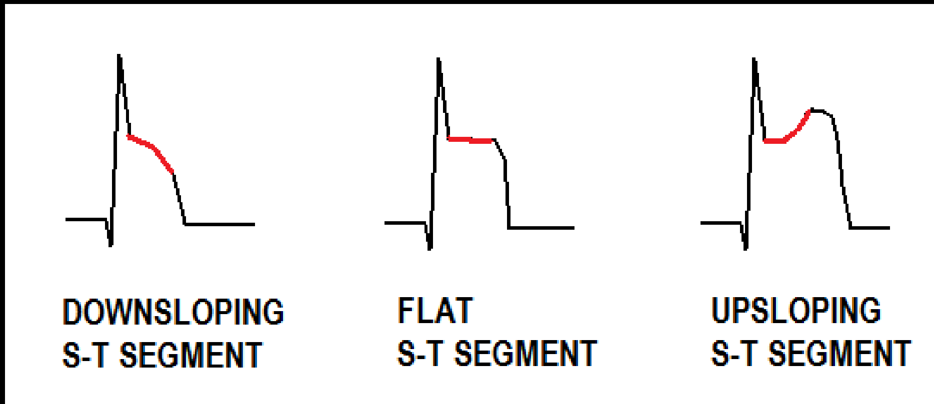
1. For men 40 years of age and older, the threshold value for abnormal J-point elevation should be 0.2 mV (2 mm) in leads V_2 and V_3 and 0.1 mV (1 mm) in all other leads.
2. For men less than 40 years of age, the threshold values for abnormal J-point elevation in leads V_2 and V_3 should be 0.25 mV (2.5 mm).
3. For women, the threshold value for abnormal J-point elevation should be 0.15 mV (1.5 mm) in leads V_2 and V_3 and greater than 0.1 mV (1 mm) in all other leads.
4. For men and women, the threshold for abnormal J-point elevation in V_3R and V_4R should be 0.05 mV (0.5 mm), except for males less than 30 years of age, for whom 0.1 mV (1 mm) is more appropriate.
5. For men and women, the threshold value for abnormal J-point elevation in V_7 through V_9 should be 0.05 mV (0.5 mm).
6. For men and women of all ages, the threshold value for abnormal J-point depression should be −0.05 mV (−0.5 mm) in leads V_2 and V_3 and −0.1 mV (−1 mm) in all other leads.

ST SEGMENT ELEVATION:

S-T SEGMENTS ELEVATE WITHIN SECONDS OF CORONARY ARTERY OCCLUSION:

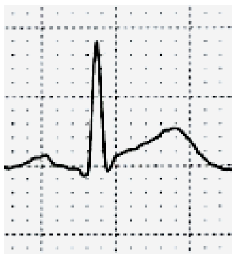
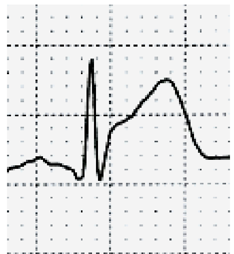
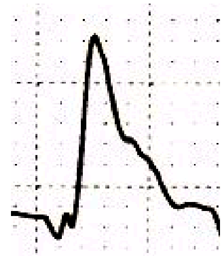
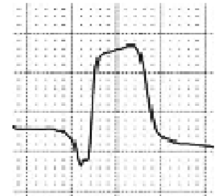
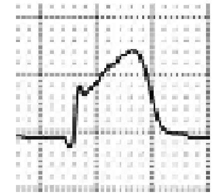
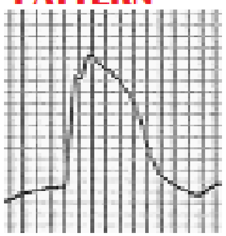
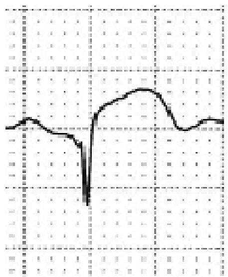
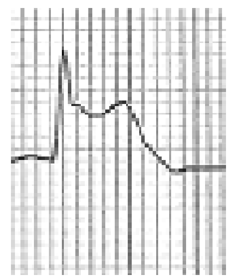
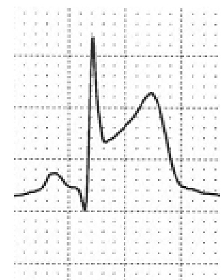
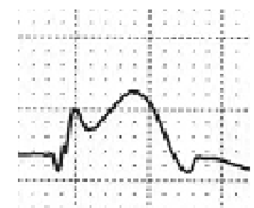
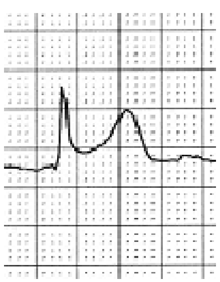
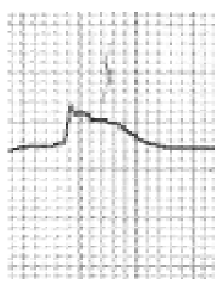
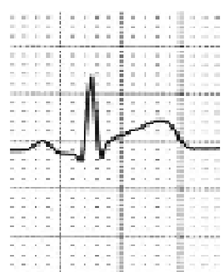
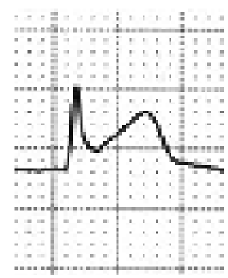
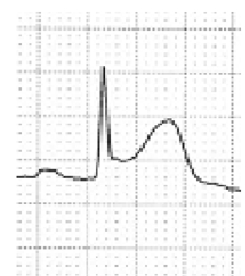
IN THIS CASE, a normal response to balloon occlusion of the RIGHT CORONARY ARTERY during PTCA in the CARDIAC CATH LAB

3 COMMON PATTERNS of ST SEGMENT ELEVATION From ACUTE MI:



ST SEGMENT ELEVATION in ACUTE MI:

The following samples are from patients with ACUTE MI, as confirmed by discovery of total arterial occlusion in the Cardiac Cath Lab:

 V5 - ANTERIOR LATERAL MI	 V4 - ANTERIOR LATERAL MI	 aVL - ANTERIOR LATERAL MI	"TOOMBSTONE" PATTERN  V2 - ANTERIOR LATERAL MI	"FIREMAN'S HAT" PATTERN  V3- ANTERIOR LATERAL MI
"TOOMBSTONE" PATTERN  V4- ANTERIOR LATERAL MI	 V5- ANTERIOR LATERAL MI	 V5-ANTERIOR LATERAL MI	 II - INFERIOR POSTERIOR MI	"FIREMAN'S HAT" PATTERN  aVF- INFERIOR POSTERIOR MI
 III-INFERIOR MI	 III-INFERIOR POSTERIOR MI	 III-INFERIOR MI	 III-INFERIOR MI	 II- INFERIOR POSTERIOR MI

Reciprocal S-T Segment Depression *may* or *may not* be present during STEMI.

Reciprocal S-T Segment Depression *may* or *may not* be present during STEMI.

The presence of S-T Depression on an EKG which exhibits significant S-T elevation is a fairly reliable indicator that STEMI is the diagnosis.

Reciprocal S-T Segment Depression *may* or *may not* be present during STEMI.

The presence of S-T Depression on an EKG which exhibits significant S-T elevation is a fairly reliable indicator that STEMI is the diagnosis.

However the *lack of Reciprocal S-T Depression* DOES NOT rule out STEMI.

ACUTE MI

COMPLICATIONS TO ANTICIPATE FOR ALL MI PATIENTS :

- LETHAL DYSRHYTHMIAS
- CARDIAC ARREST
- FAILURE OF STRUCTURE(S) SERVED BY THE BLOCKED ARTERY

CASE STUDY 1 - STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

72 y/o male, c/o CHEST "HEAVINESS," started 20 minutes before calling 911. Pain is "8" on 1-10 scale, also c/o mild shortness of breath. Has had same pain "intermittently" x 2 weeks.

RISK FACTOR PROFILE:

- FAMILY HISTORY - father died of MI at age 77
- FORMER CIGARETTE SMOKER - smoked for 30 year - quit 27 years ago
- DIABETES - oral meds and diet controlled
- HIGH CHOLESTEROL - controlled with STATIN meds
- AGE: OVER 65

PHYSICAL EXAM: Patient calm, alert, oriented X 4, skin cool, dry, pale. No JVD, Lungs clear bilaterally. Heart sounds normal S1, S2. No peripheral edema.

VITAL SIGNS: BP: 100/64, P: 75, R: 20, SAO2: 94%

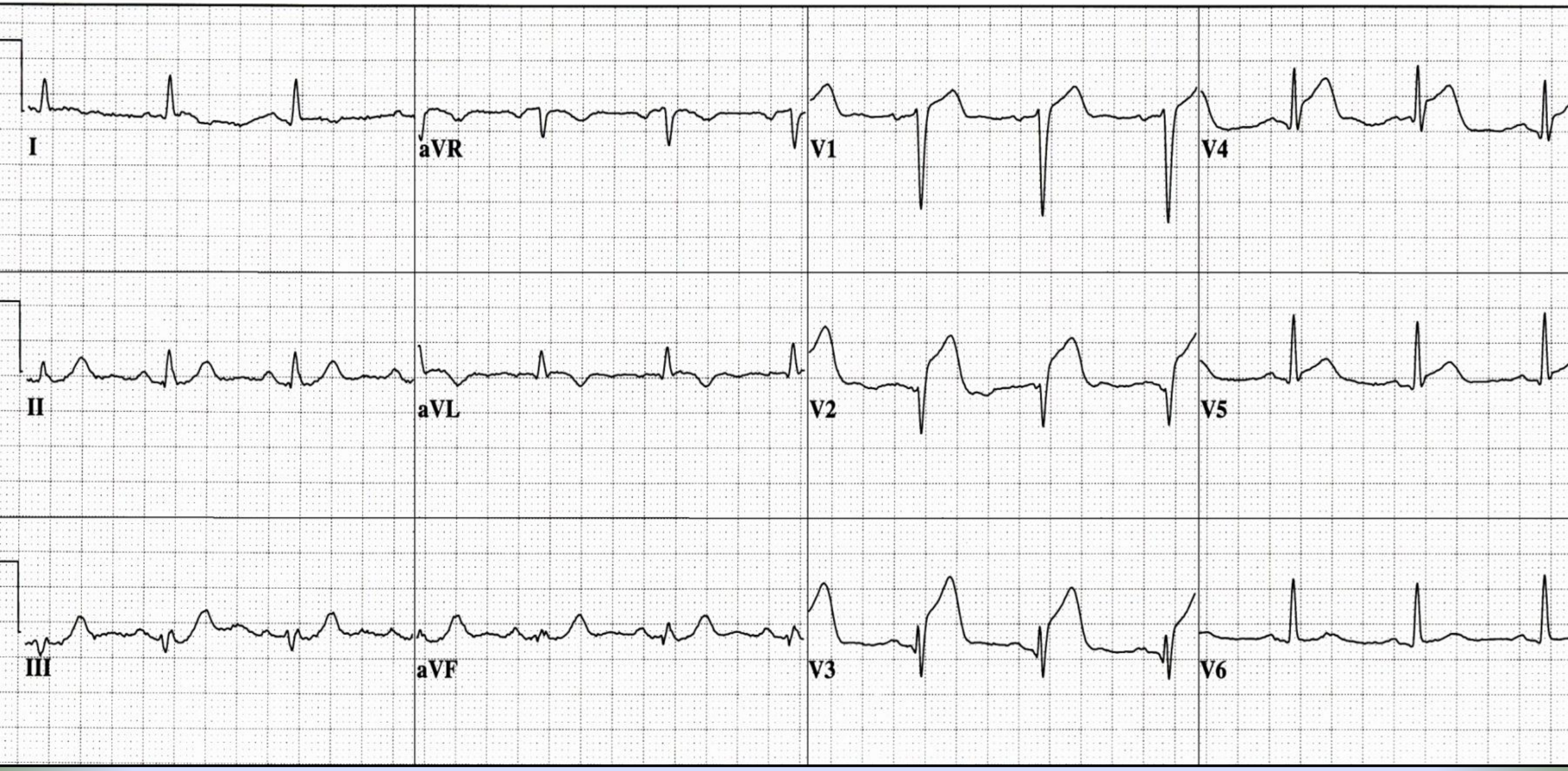
LABS: FIRST TROPONIN: 6.4

72 yr
Male Caucasian
Loc:3 Option:23

Vent. rate	75	BPM
PR interval	162	ms
QRS duration	98	ms
QT/QTc	382/426	ms
P–R–T axes	72 13	83

EVALUATE EKG for indicators of ACS:

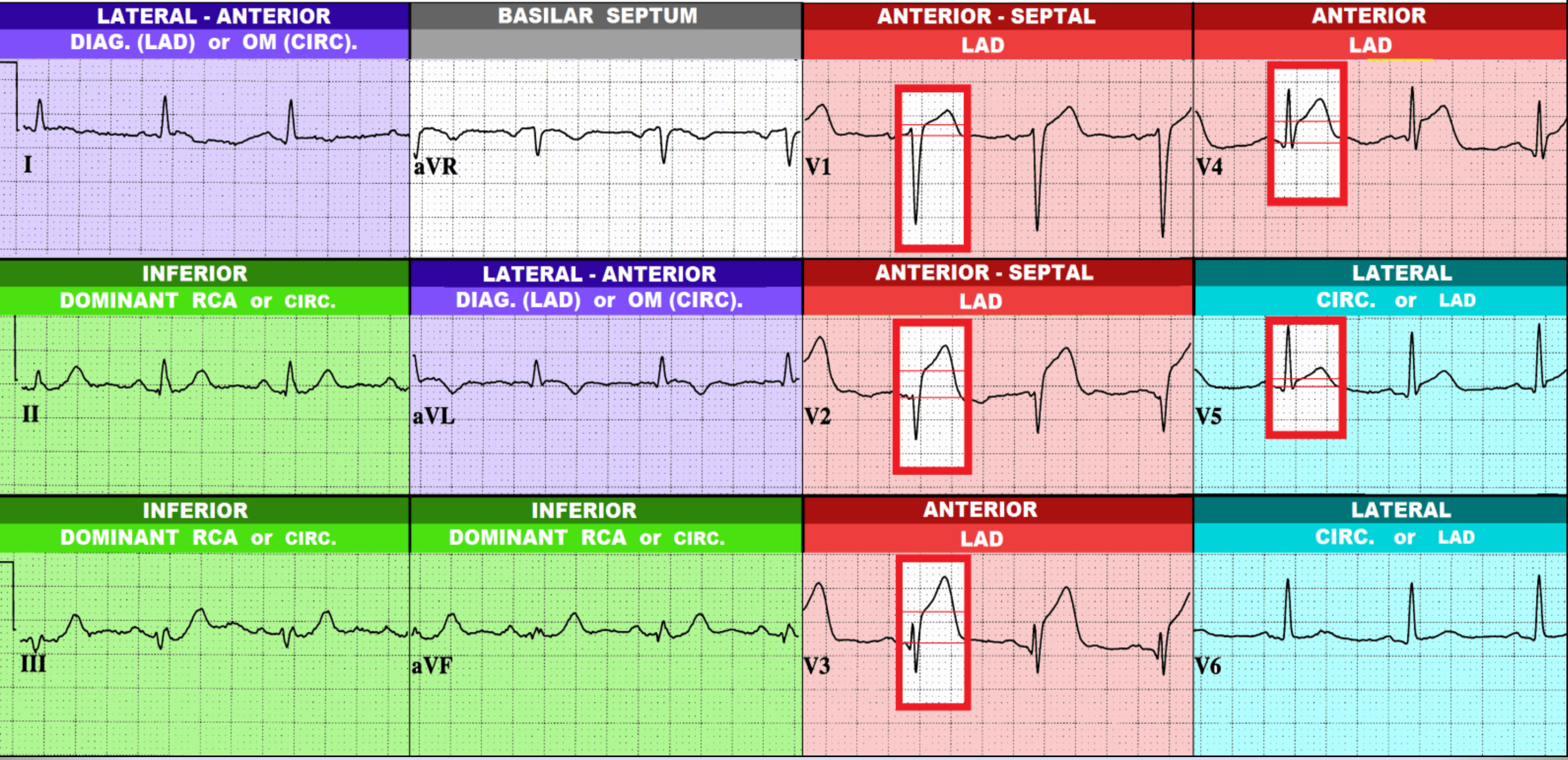
- ST SEGMENT ELEVATION / DEPRESSION
- HYPERACUTE T WAVES
- CONVEX ST SEGMENTS
- OTHER ST SEGMENT / T WAVE ABNORMALITIES



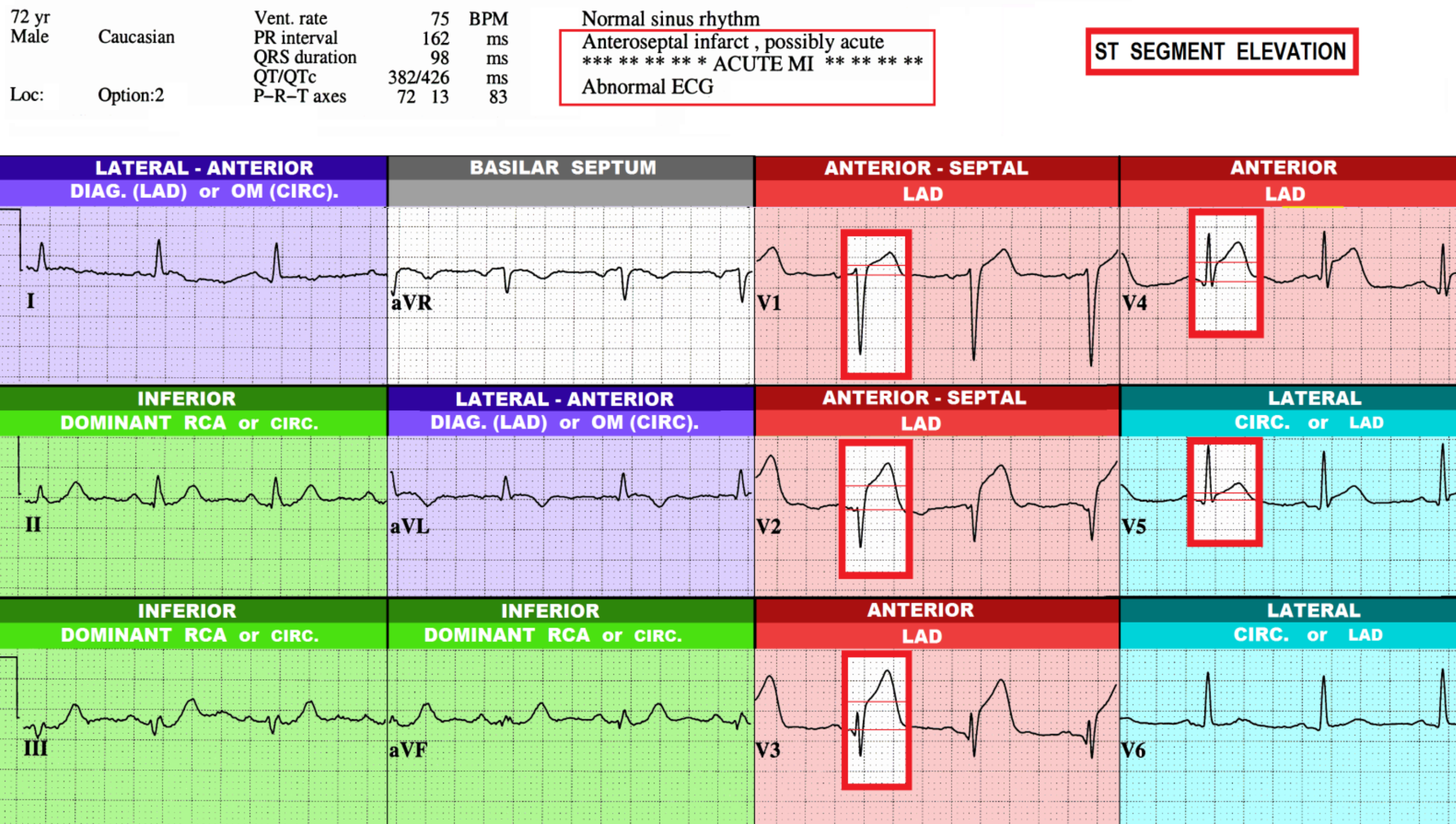
72 yr		Vent. rate	75	BPM
Male	Caucasian	PR interval	162	ms
		QRS duration	98	ms
		QT/QTc	382/426	ms
Loc:	Option:2	P–R–T axes	72 13	83

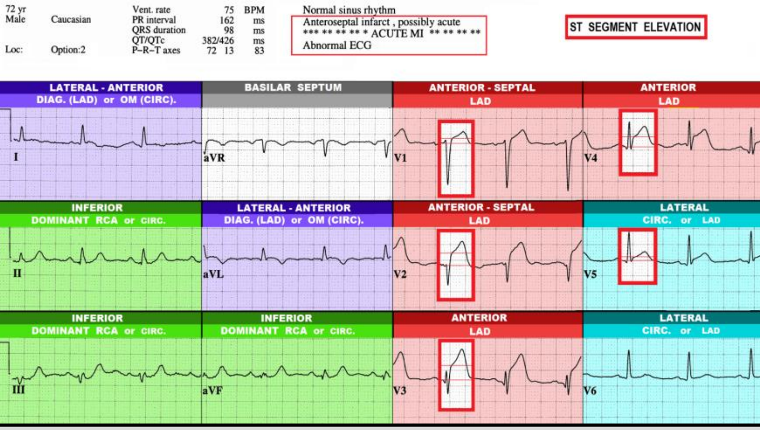
Normal sinus rhythm
Anteroseptal infarct , possibly acute
*** ** ** ** * ACUTE MI ** ** ** **
Abnormal ECG

ST SEGMENT ELEVATION



Note: There is NO Reciprocal ST Depression on this STEMI ECG !


72 yr
Male
Caucasian
Loc:
Option:2
Vent. rate 75 BPM
PR interval 162 ms
QRS duration 98 ms
QT/QTc 382/426 ms
P–R–T axes 72 13 83
Normal sinus rhythm
Anteroseptal infarct , possibly acute
*** ** ** ** * ACUTE MI ** ** ** **
Abnormal ECG
ST SEGMENT ELEVATION
LATERAL - ANTERIOR
DIAG. (LAD) or OM (CIRC).
I
BASILAR SEPTUM
aVR
ANTERIOR - SEPTAL
LAD
V1
ANTERIOR
LAD
V4
INFERIOR
DOMINANT RCA or CIRC.
II
LATERAL - ANTERIOR
DIAG. (LAD) or OM (CIRC).
aVL
ANTERIOR - SEPTAL
LAD
V2
LATERAL
CIRC. or LAD
V5
INFERIOR
DOMINANT RCA or CIRC.
III
INFERIOR
DOMINANT RCA or CIRC.
aVF
ANTERIOR
LAD
V3
LATERAL
CIRC. or LAD
V6

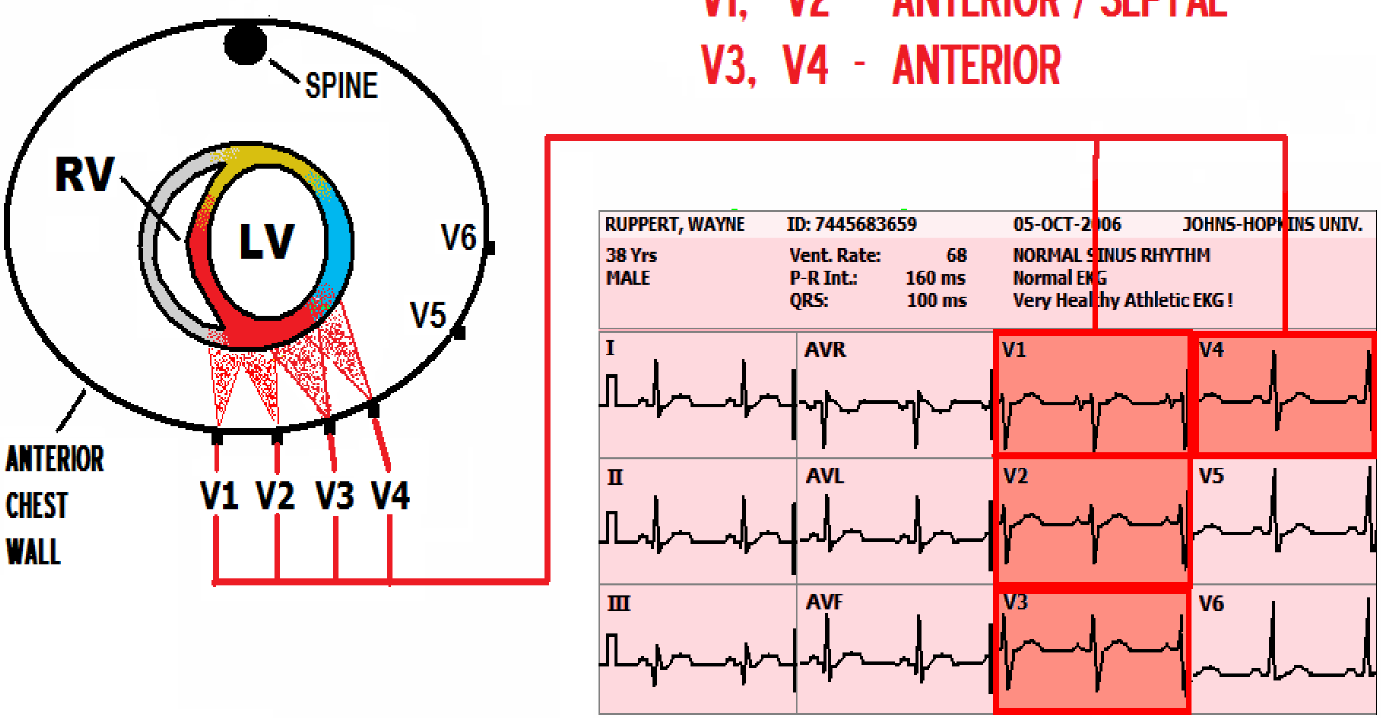


V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL

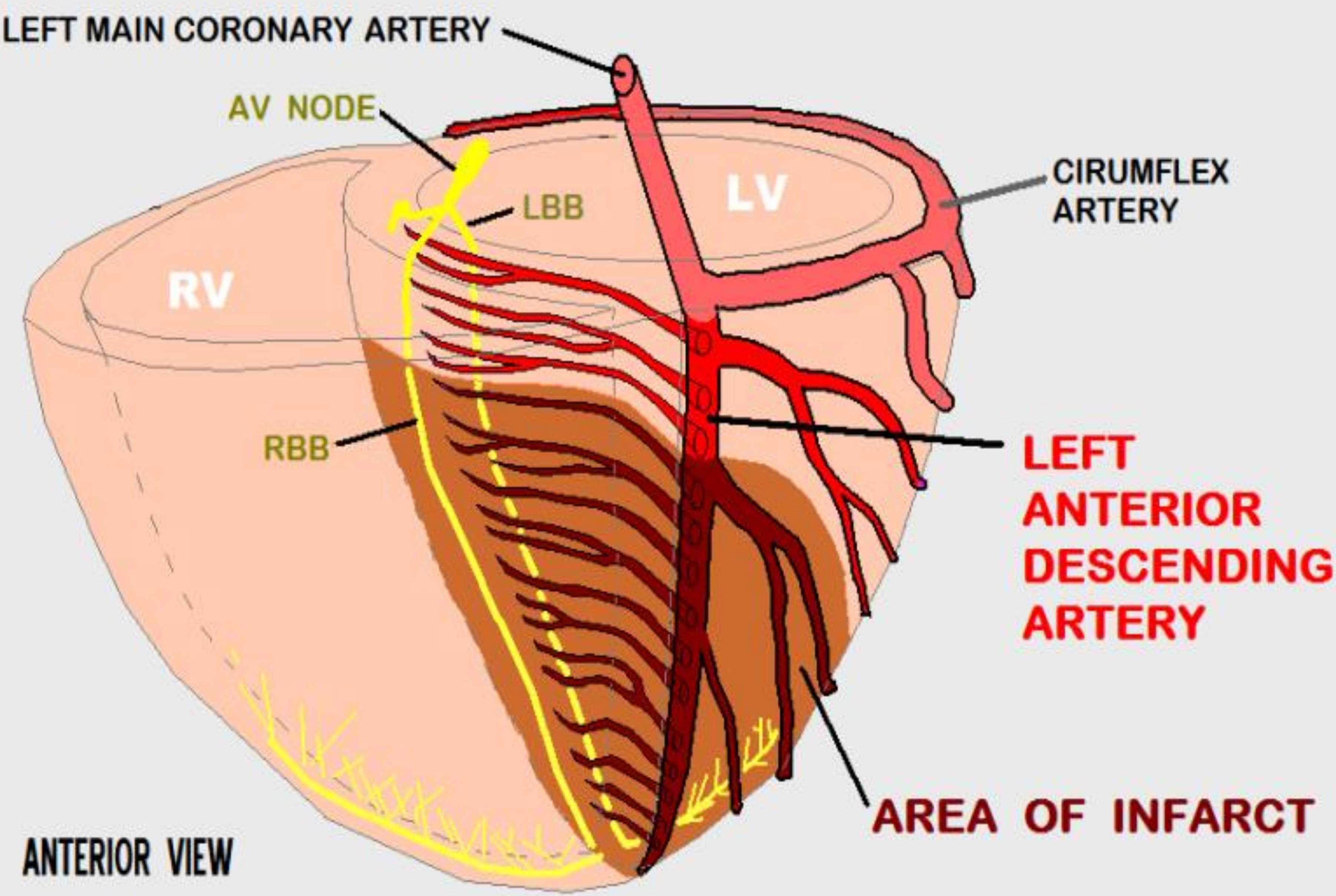
of the LEFT VENTRICLE

V1, V2 - ANTERIOR / SEPTAL

V3, V4 - ANTERIOR

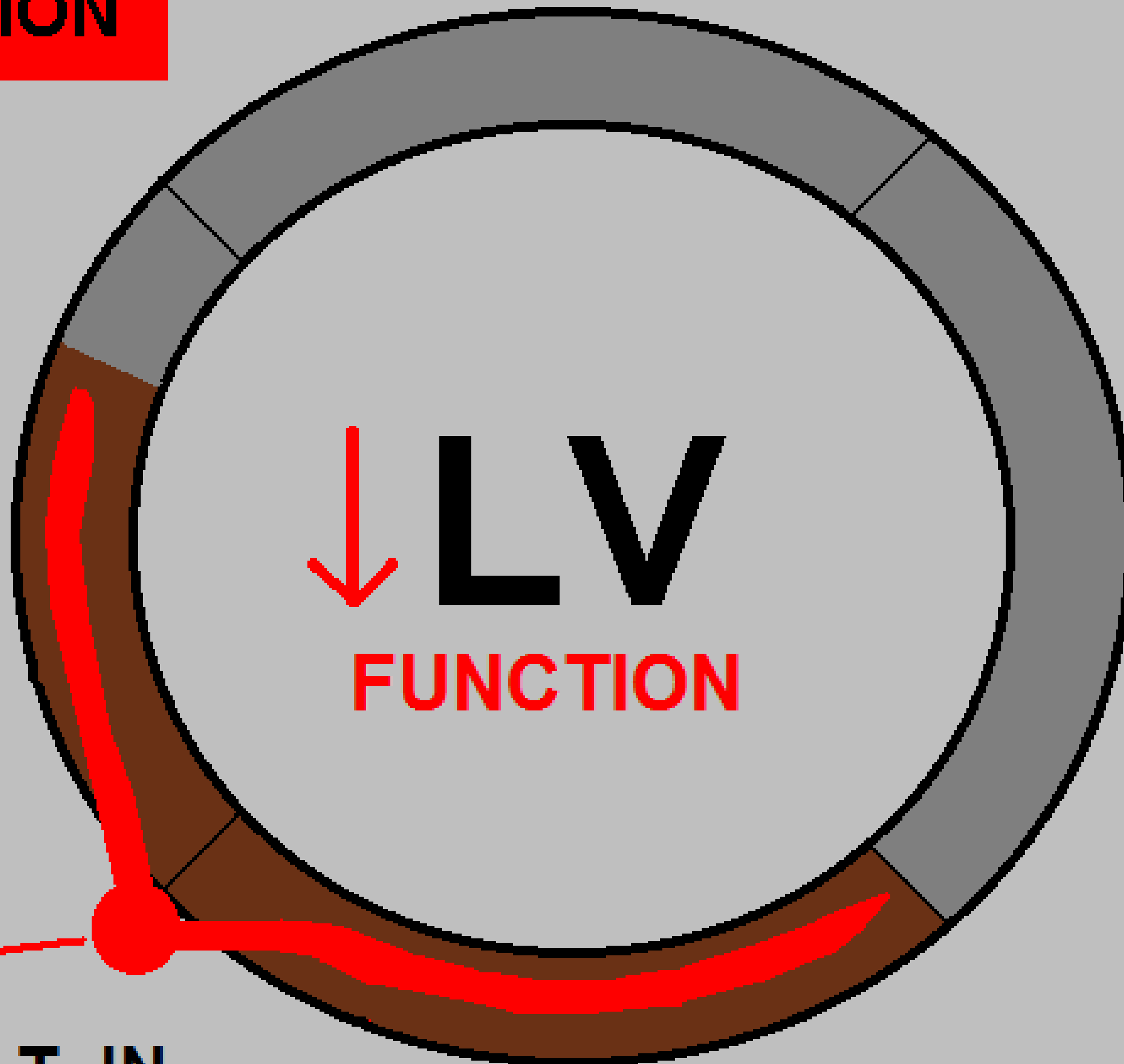


OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY



LAD DISTRIBUTION

35 - 45 % of LV MUSCLE MASS



A BLOCKAGE OF THE LAD CAN RESULT IN LV PUMP FAILURE --

- CARDIOGENIC SHOCK ☠
- PULMONARY EDEMA ☠


400 mg
Dopamine
(1600 mcg/mL)
Dopamine Hydrochloride
and 5% Dextrose Injection USP
250 mL
Baxter
500 mg Total
DOBUTamine
Hydrochloride
5% Dextrose
250 mL
Baxter

LEFT ANTERIOR DESCENDING ARTERY (LAD)

- ANTERIOR WALL OF LEFT VENTRICLE

35 - 45 % OF LEFT VENTRICLE MUSCLE MASS

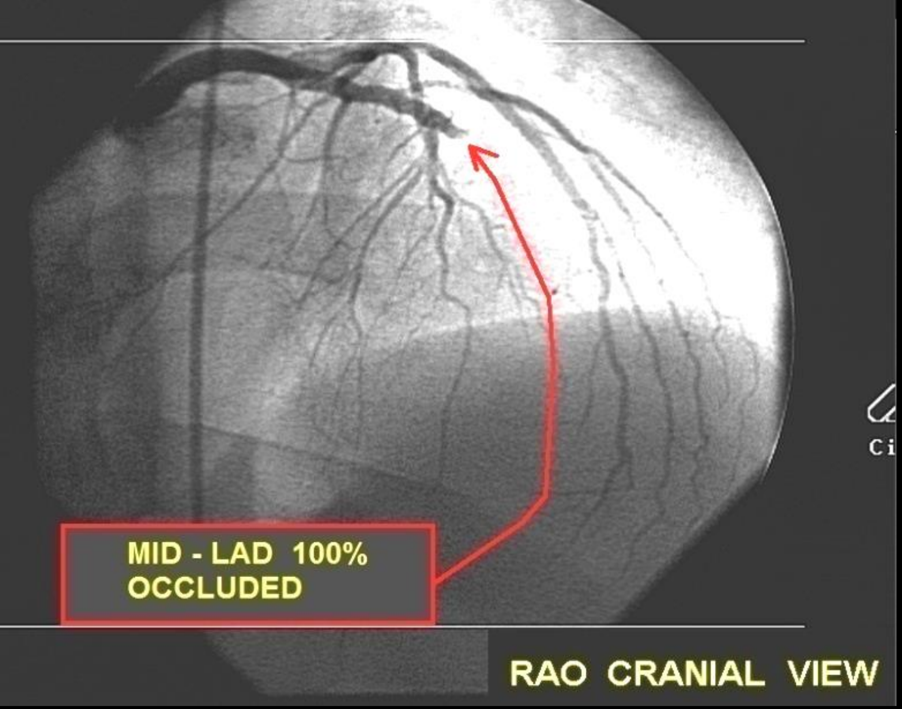
- SEPTUM, ANTERIOR 2/3

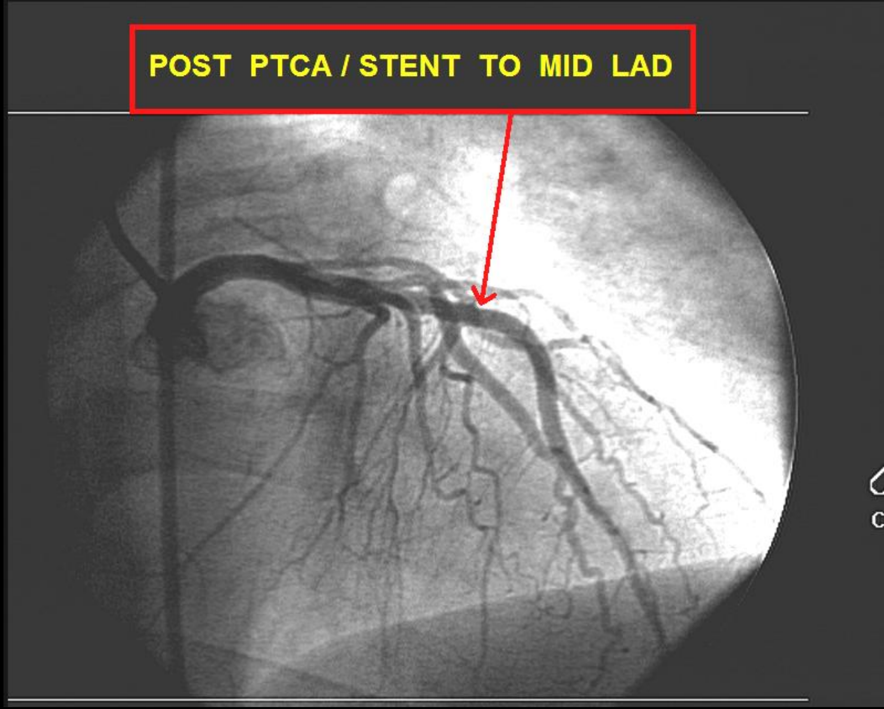
BUNDLE BRANCHES

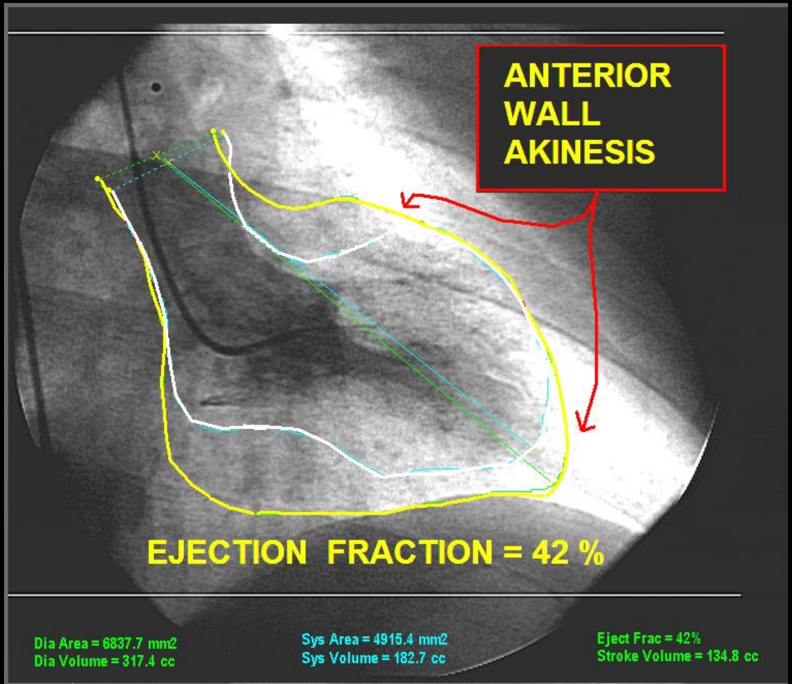
- ANTERIOR-MEDIAL PAPILLARY MUSCLE

ANTICIPATED COMPLICATIONS of ANTERIOR-SEPTAL WALL STEMI & POSSIBLE INDICATED INTERVENTIONS:	
- CARDIAC ARREST	BCLS / ACLS
- CARDIAC DYSRHYTHMIAS (VT / VF)	ACLS (antiarrhythmics)
- PUMP FAILURE with CARDIOGENIC SHOCK	INOTROPE THERAPY: -DOPAMINE / DOBUTAMINE / LEVOPHED - INTRA-AORTIC BALLOON PUMP (use caution with fluid challenges due to PULMONARY EDEMA)

- PULMONARY EDEMA	- CPAP - ET INTUBATION (use caution with dieuretics due to pump failure and hypotension)
- 3rd DEGREE HEART BLOCK - NOT RESPONSIVE TO ATROPINE	TRANSCUTANEOUS or TRANSVENOUS PACING


MID - LAD 100%
OCCLUDED
RAO CRANIAL VIEW


POST PTCA / STENT TO MID LAD


ANTERIOR
WALL
AKINESIS
EJECTION FRACTION = 42 %
Dia Area = 6837.7 mm2
Dia Volume = 317.4 cc
Sys Area = 4915.4 mm2
Sys Volume = 182.7 cc
Eject Frac = 42%
Stroke Volume = 134.8 cc

CASE STUDY 2: STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

46 y/o Female walks into ED TRIAGE, with chief complaint of EPIGASTRIC PAIN, NAUSEA and WEAKNESS. Symptoms have been intermittent for last two days. She was awakened early this morning with the above symptoms, which are now PERSISTENT.

RISK FACTOR PROFILE:

- FAMILY HISTORY - father died of CAD, older brother had CABG, age 39
- DIABETES - diet controlled
- HYPERTENSION

PHYSICAL EXAM: Pt. CAOx4, anxious, SKIN cold, clammy, diaphoretic. No JVD. Lungs: clear, bilaterally. Heart Sounds: Normal S1, S2.

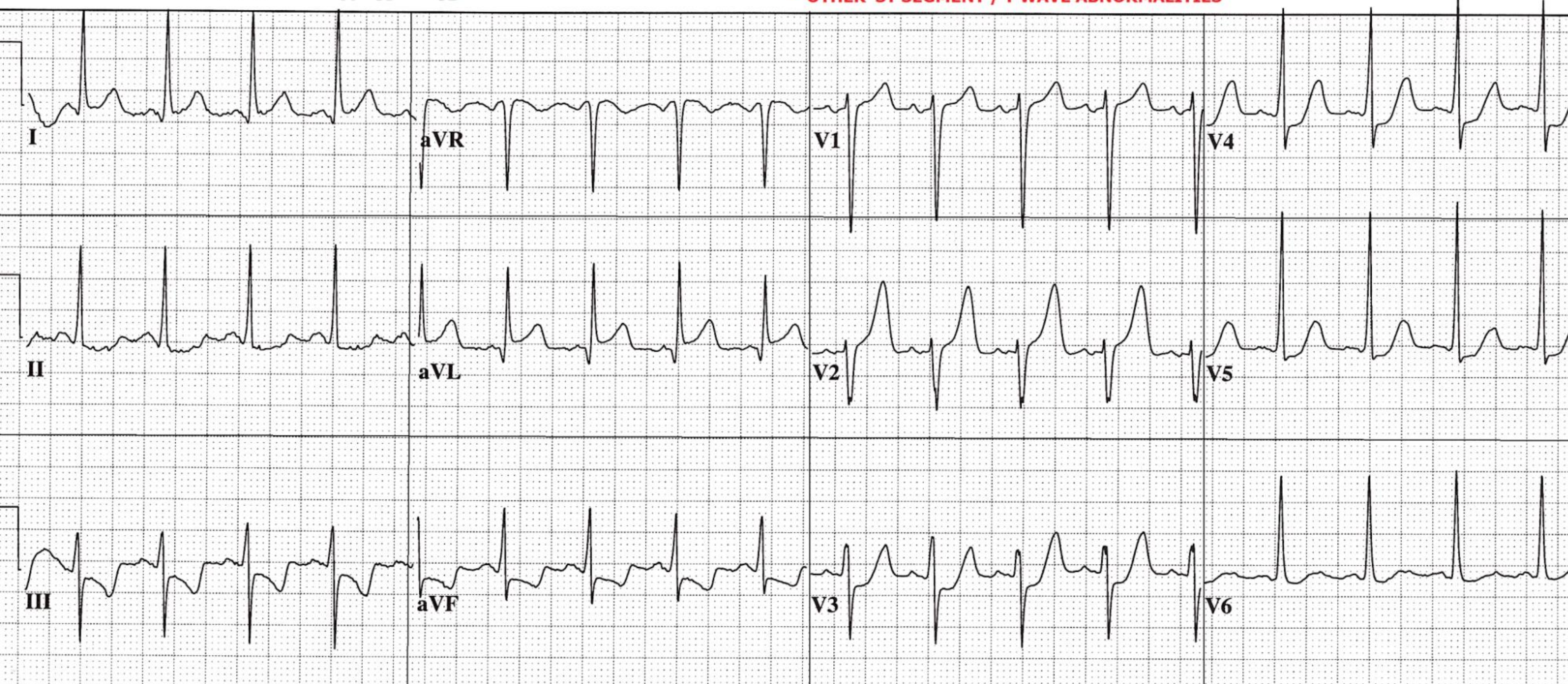
VITAL SIGNS: BP: 168/98, P: 110, R: 24, SAO2: 97% on O2 4 LPM via nasal canula

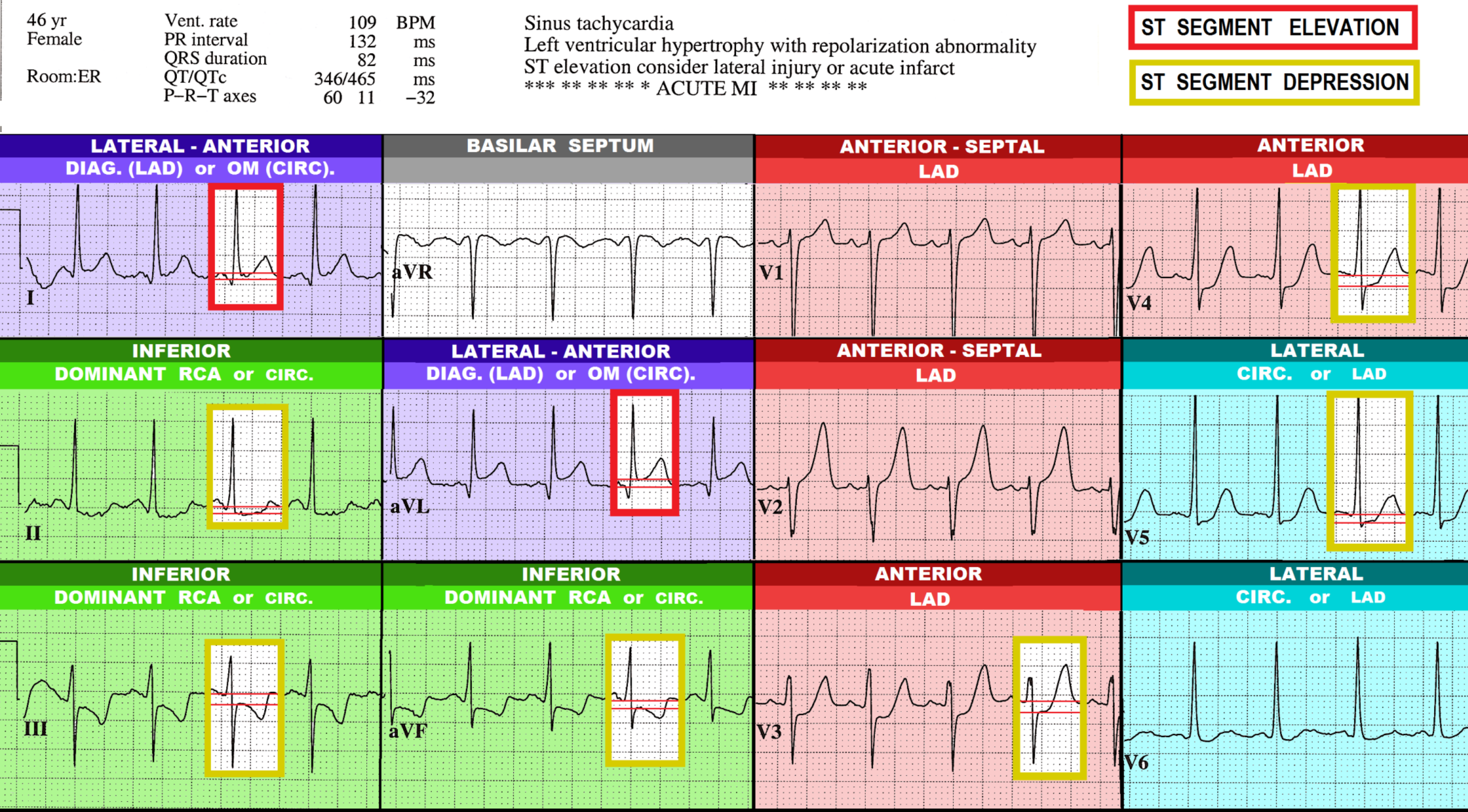
LABS: TROPONIN ultra = 2.8

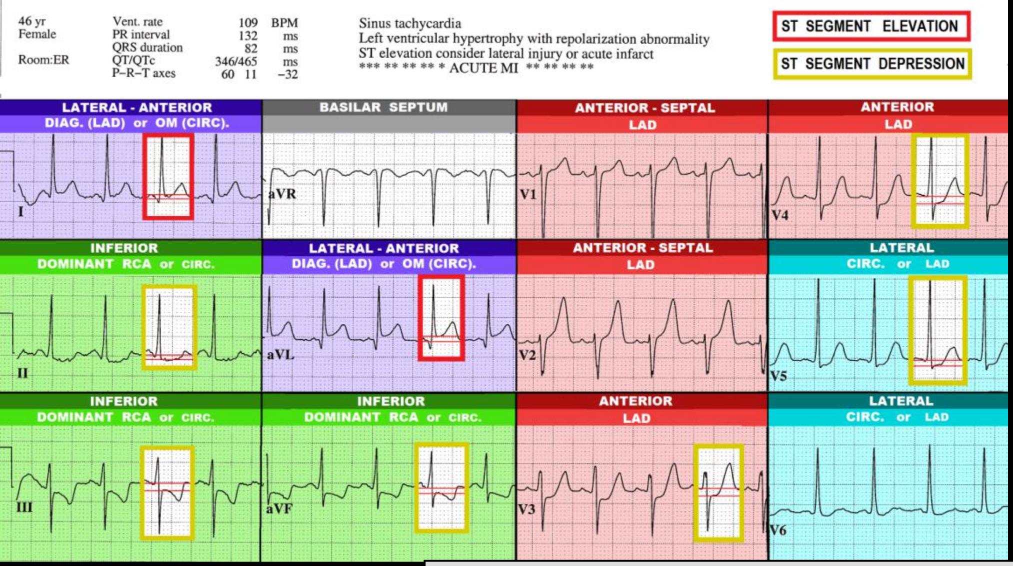
46 yr	Vent. rate	109	BPM
Female	PR interval	132	ms
	QRS duration	82	ms
Room:ER	QT/QTc	346/465	ms
	P–R–T axes	60 11	–32

EVALUATE EKG for indicators of ACS:

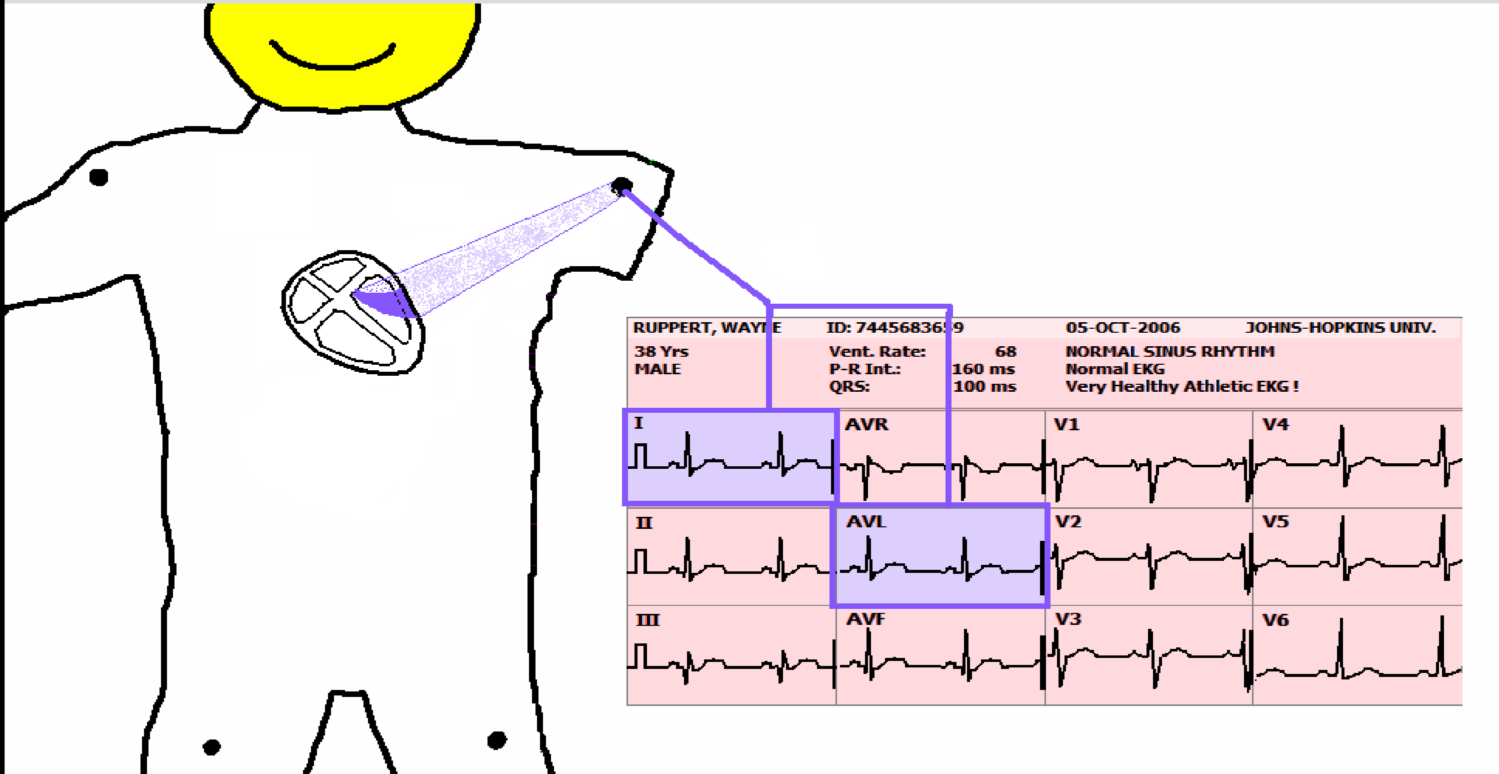
- ST SEGMENT ELEVATION / DEPRESSION
- HYPERACUTE T WAVES
- CONVEX ST SEGMENTS
- OTHER ST SEGMENT / T WAVE ABNORMALITIES

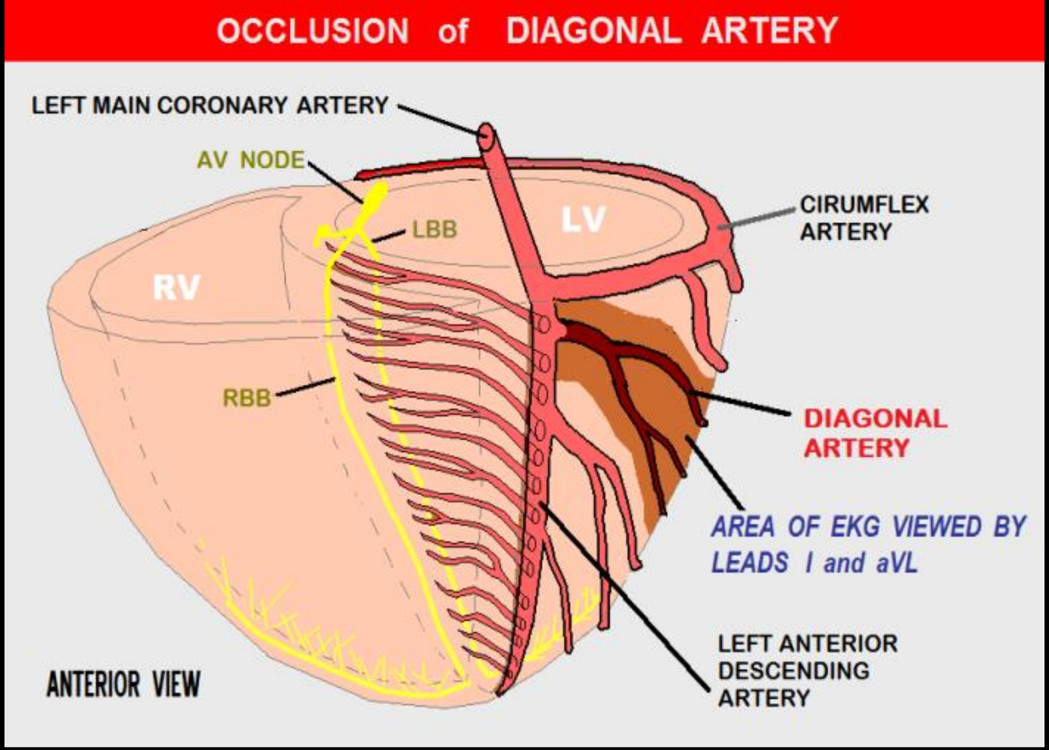


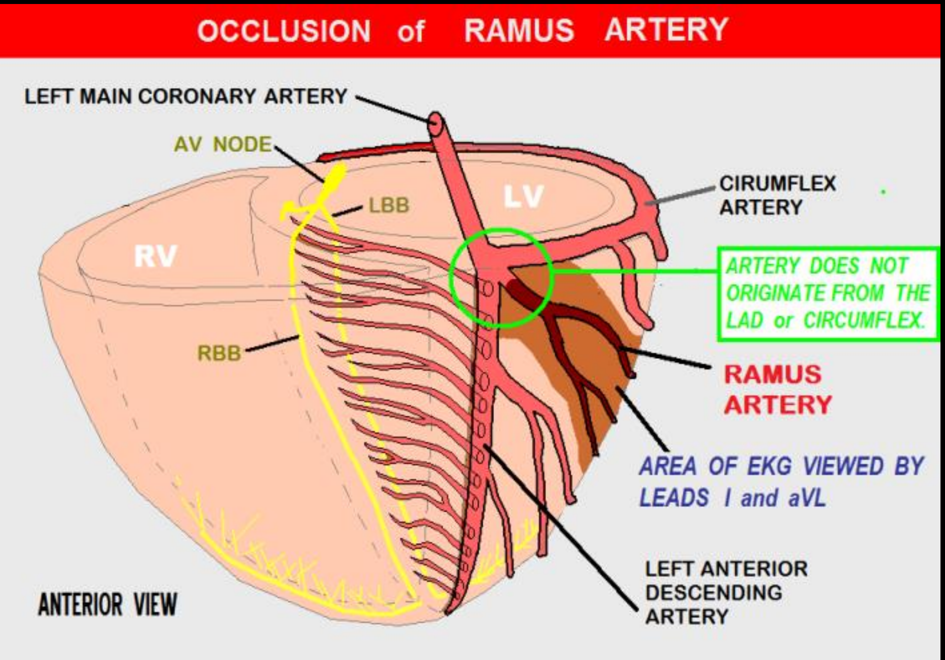

46 yr
Female
Room:ER
Vent. rate 109 BPM
PR interval 132 ms
QRS duration 82 ms
QT/QTc 346/465 ms
P–R–T axes 60 11 –32
Sinus tachycardia
Left ventricular hypertrophy with repolarization abnormality
ST elevation consider lateral injury or acute infarct
*** ** ** ** * ACUTE MI ** ** ** **
ST SEGMENT ELEVATION
ST SEGMENT DEPRESSION
LATERAL - ANTERIOR
DIAG. (LAD) or OM (CIRC).
I
BASILAR SEPTUM
aVR
ANTERIOR - SEPTAL
LAD
V1
ANTERIOR
LAD
V4
INFERIOR
DOMINANT RCA or CIRC.
II
LATERAL - ANTERIOR
DIAG. (LAD) or OM (CIRC).
aVL
ANTERIOR - SEPTAL
LAD
V2
LATERAL
CIRC. or LAD
V5
INFERIOR
DOMINANT RCA or CIRC.
III
INFERIOR
DOMINANT RCA or CIRC.
aVF
ANTERIOR
LAD
V3
LATERAL
CIRC. or LAD
V6

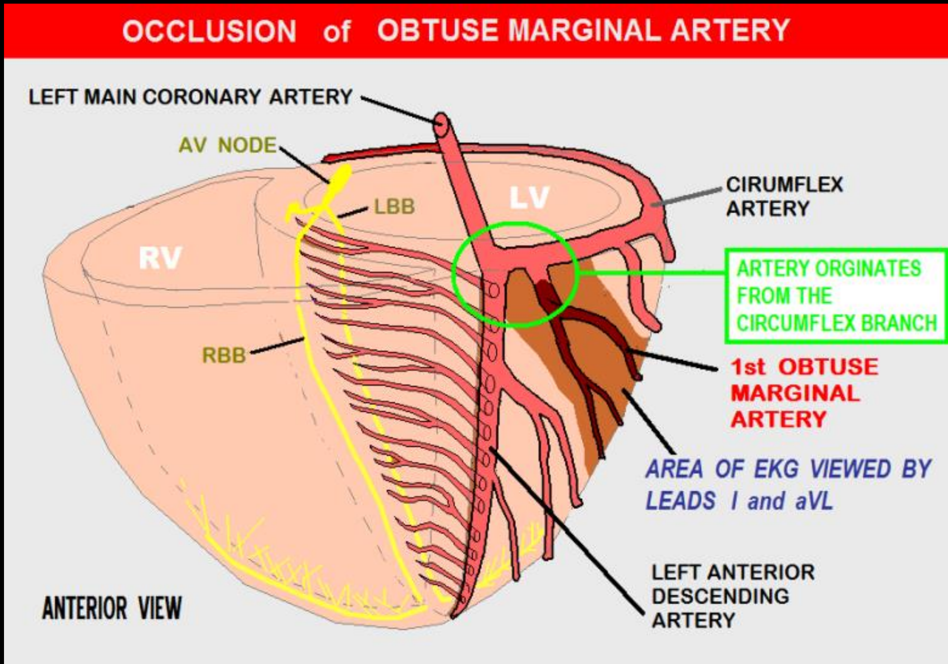


LEADS I and aVL view the ANTERIOR-LATERAL JUNCTION

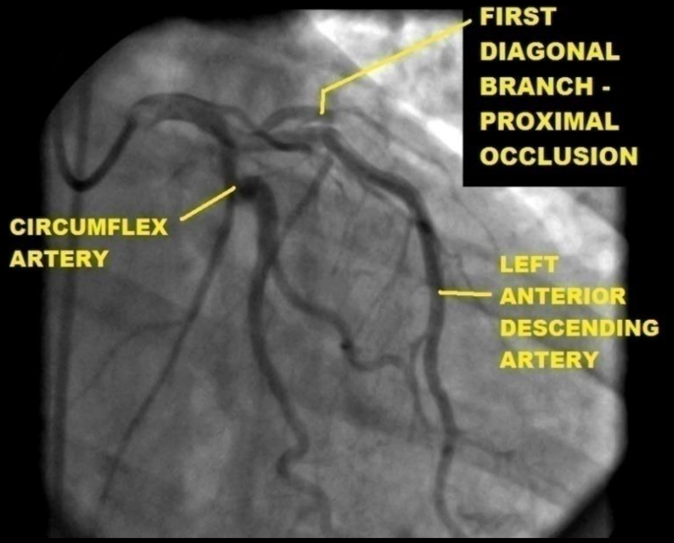


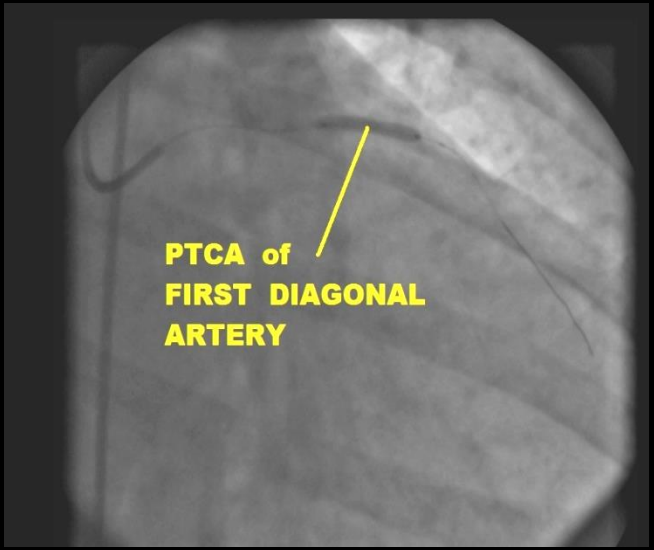

OCCLUSION of DIAGONAL ARTERY
LEFT MAIN CORONARY ARTERY
AV NODE
LBB
LV
CIRUMFLEX ARTERY
RV
RBB
DIAGONAL ARTERY
AREA OF EKG VIEWED BY LEADS I and aVL
LEFT ANTERIOR DESCENDING ARTERY
ANTERIOR VIEW

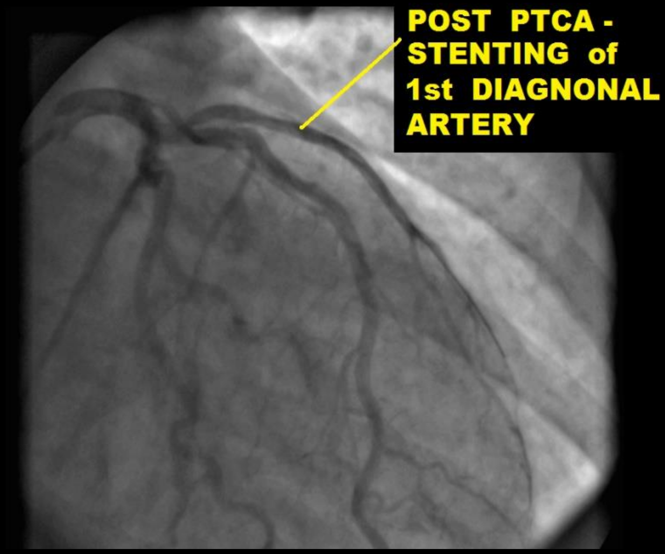

OCCLUSION of RAMUS ARTERY
LEFT MAIN CORONARY ARTERY
AV NODE
LBB
LV
CIRUMFLEX ARTERY
RV
ARTERY DOES NOT ORIGINATE FROM THE LAD or CIRCUMFLEX.
RBB
RAMUS ARTERY
AREA OF EKG VIEWED BY LEADS I and aVL
LEFT ANTERIOR DESCENDING ARTERY
ANTERIOR VIEW


OCCLUSION of OBTUSE MARGINAL ARTERY
LEFT MAIN CORONARY ARTERY
AV NODE
LBB
LV
CIRUMFLEX ARTERY
RV
ARTERY ORGINATES FROM THE CIRCUMFLEX BRANCH
RBB
1st OBTUSE MARGINAL ARTERY
AREA OF EKG VIEWED BY LEADS I and aVL
LEFT ANTERIOR DESCENDING ARTERY
ANTERIOR VIEW

CASE PROGRESSION: As the patient was being prepared for transport to the Cardiac Cath Lab, she experienced an episode of Ventricular Fibrillation.







11111 11
Born 1/ 1941 77 Years

[redacted]
Acct# [redacted] MR# [redacted]
ONIER VILLARREAL
Adm: [redacted] 2018 DOB: [redacted]
SEVEN RIVERS RMC
[redacted]

3/16/2018 1:31:57 PM
Seven Rivers Reg al

Rate	69	. SINUS RHYT. [redacted] ....normal P axis, V-rate 50- 99	Room: er11
		. LEFT ATRIAL ABNORMALITY.................................P,P'>60mS, <-0.15mV V1	
PR	180	. LEFT ANTERIOR FASCICULAR BLOCK......................axis(240,-40), init forces inf	
QRSD	94	. [redacted]	
QT	436		
QTc	467		

--AXIS--

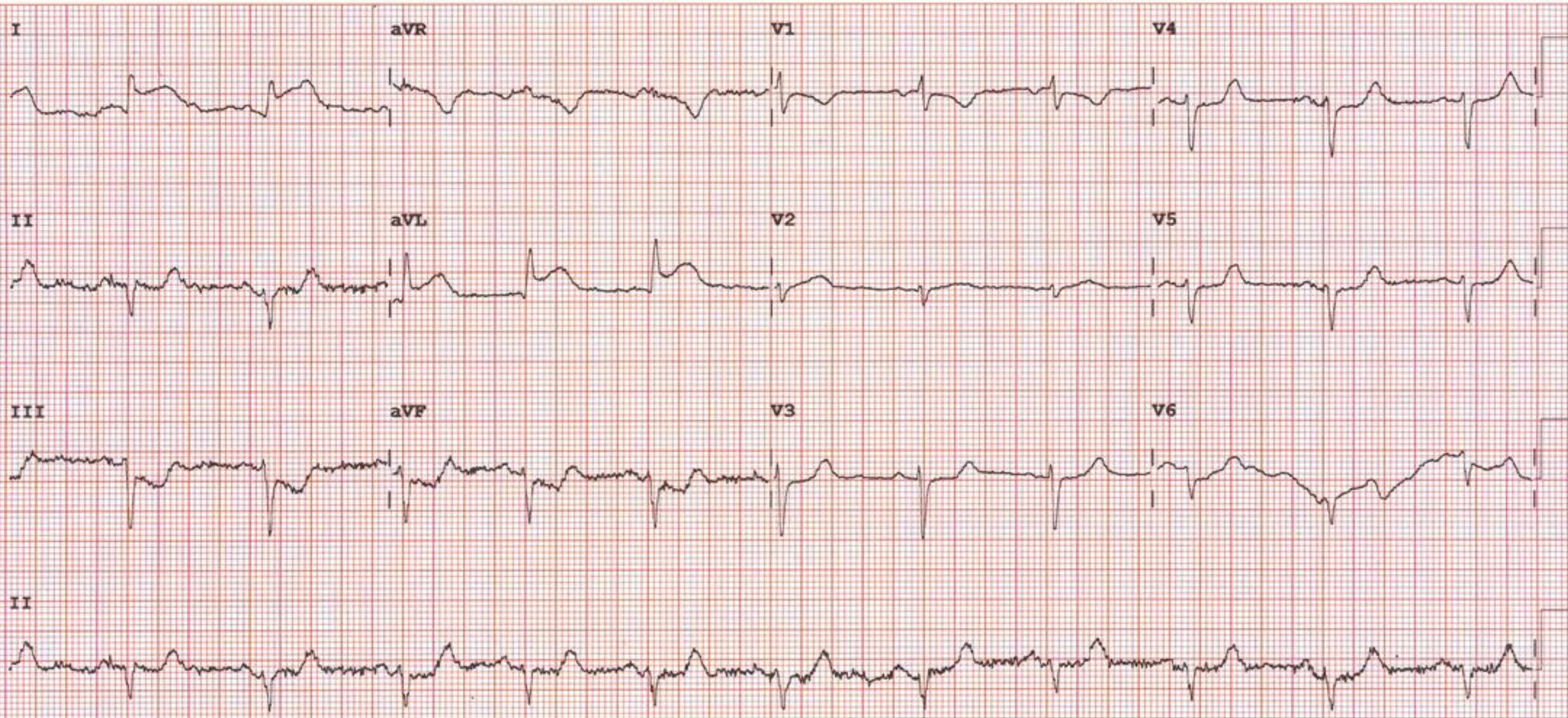
P	56
QRS	-51
T	-7

- ABNORMAL ECG -

12 Lead; Standard Placement

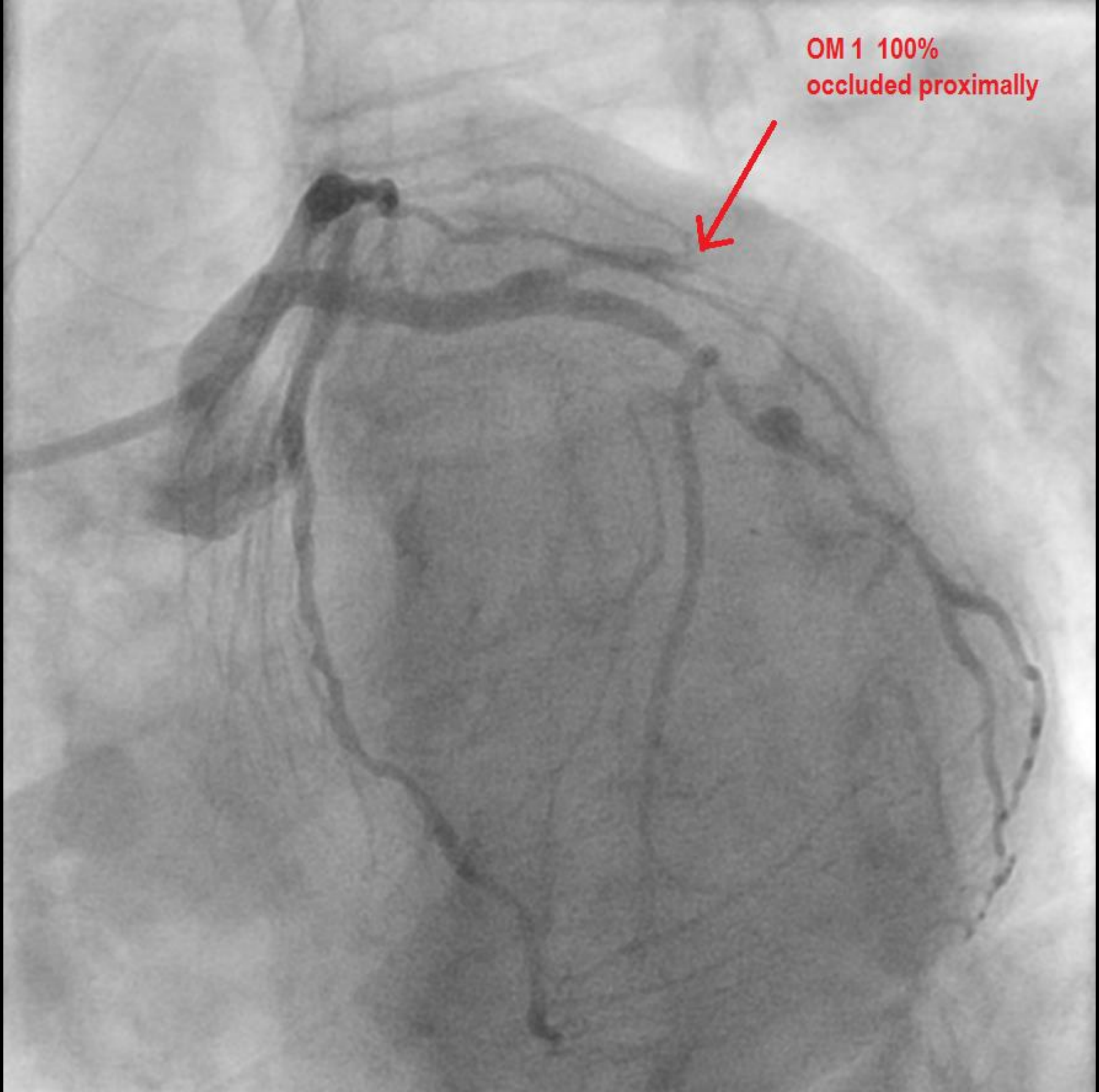
Unconfirmed Diagnosis

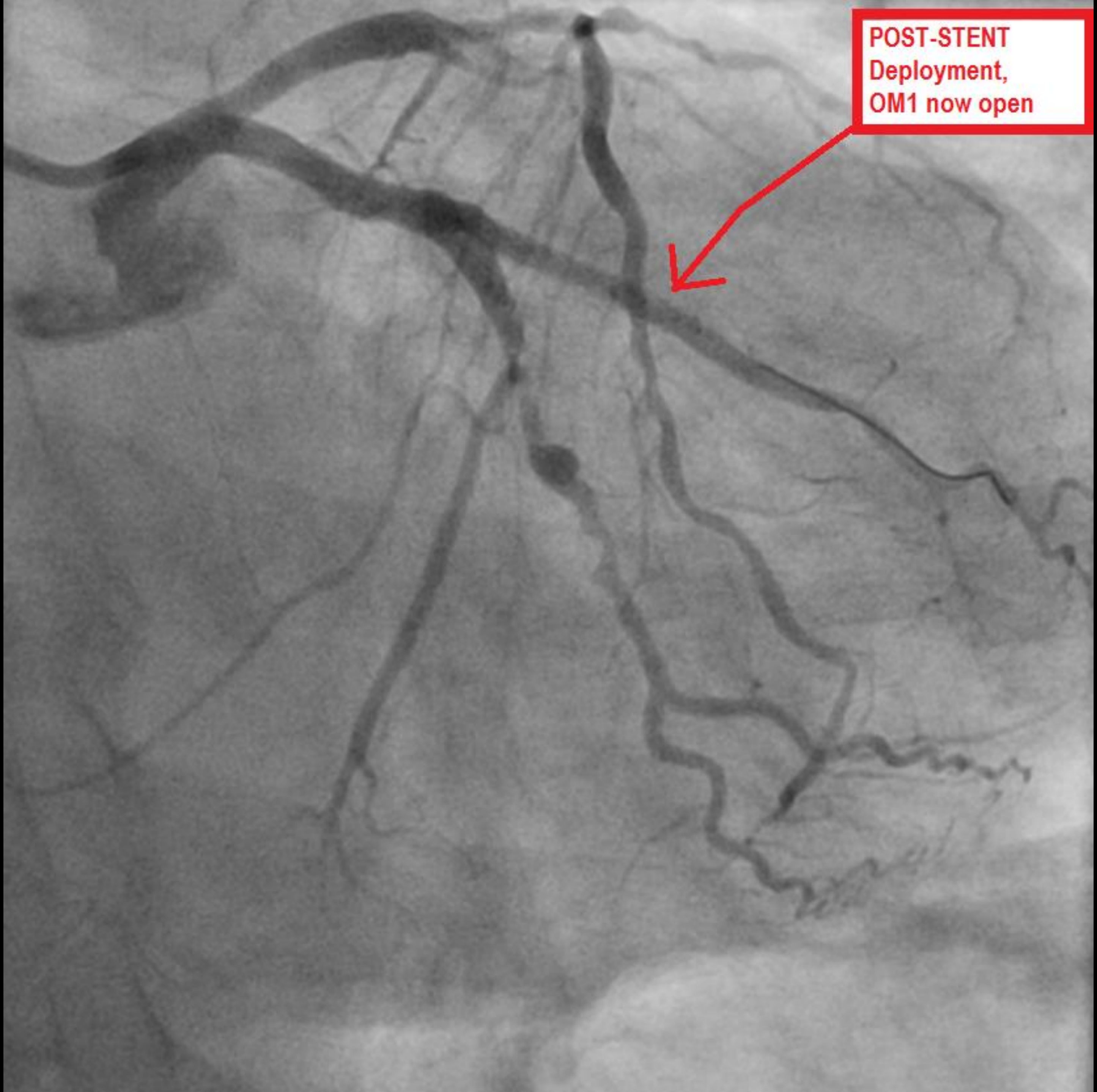
Physician
Date [redacted]
Time 1331
STEMI YES NO



Device: Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10.0 mm/mV F 60~ 0.15-100 Hz PH090A L P?

1711 COVIDIEN Kendall REF 30768678


OM 1 100%
occluded proximally


POST-STENT
Deployment,
OM1 now open

CASE STUDY 3: STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

29 y/o male presents to the ER c/o "HEAVY CHEST PRESSURE" x 30 minutes. The patient states he was playing football with friends after eating a large meal. Pt. also c/o nausea. Denies DIB.

RISK FACTOR PROFILE:

- FAMILY HISTORY - father died of MI age 46
- CURRENT CIGARETTE SMOKER
- "MILD" HYPERTENSION - untreated
- ☑ CHOLESTEROL - unknown - "never had it checked."

PHYSICAL EXAM: Patient alert, oriented X 4, skin cool, dry, pale. Patient restless. No JVD, Lungs clear bilaterally. Heart sounds normal S1, S2. No peripheral edema.

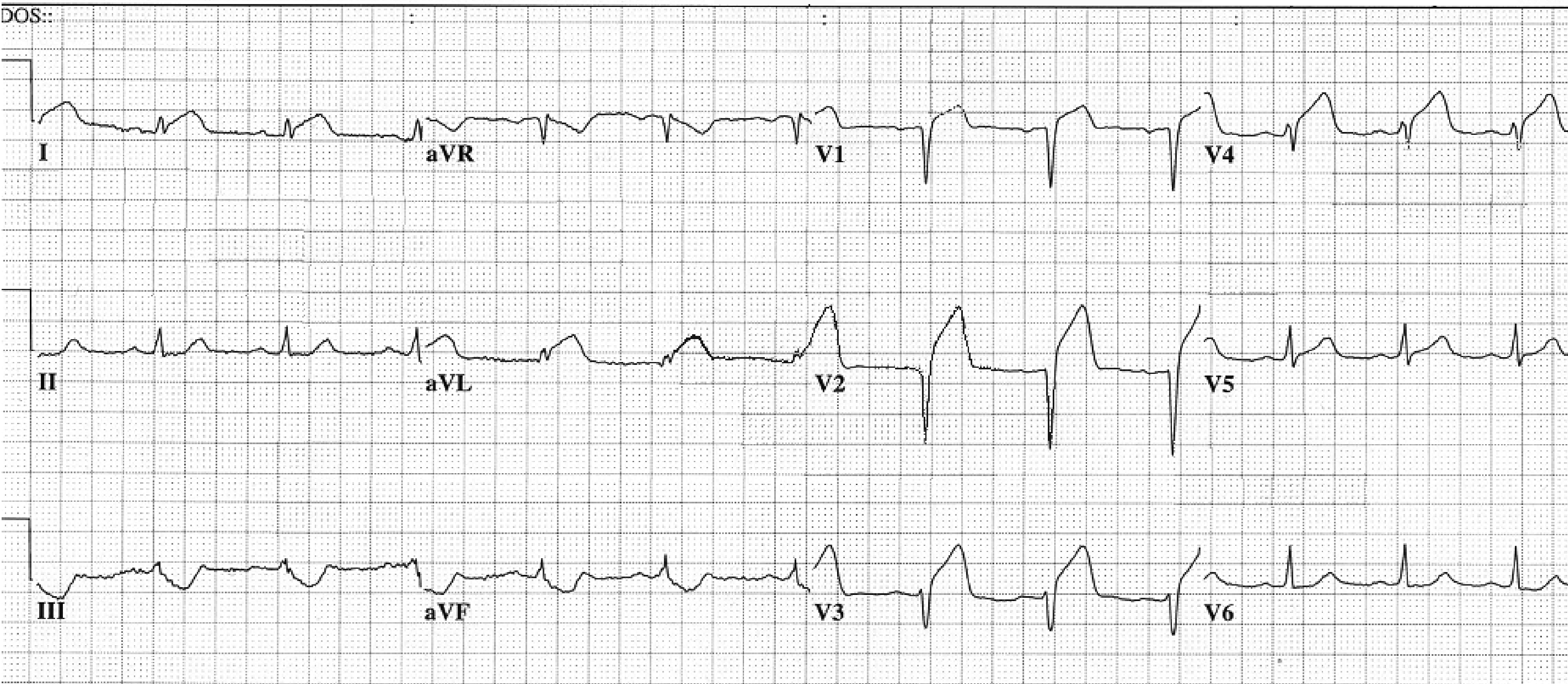
VITAL SIGNS: BP: 104/78, P: 76, R: 20, SAO2: 96%

LABS: INITIAL CARDIAC MARKERS - NEGATIVE

29 yr		Vent. rate	75	BPM		
Male	Caucasian	PR interval	176	ms		
		QRS duration	90	ms		
		QT/QTc	362/404	ms		
Loc:3	Option:20	P–R–T axes	70 50	–11	14:07	Hours

EVALUATE the EKG for signs of ACS:

- ST SEGMENT ELEVATION / DEPRESSION
- HYPERACUTE T WAVES
- CONVEX / FLAT ST SEGMENTS
- OTHER ST - T WAVE ABNORMALITIES



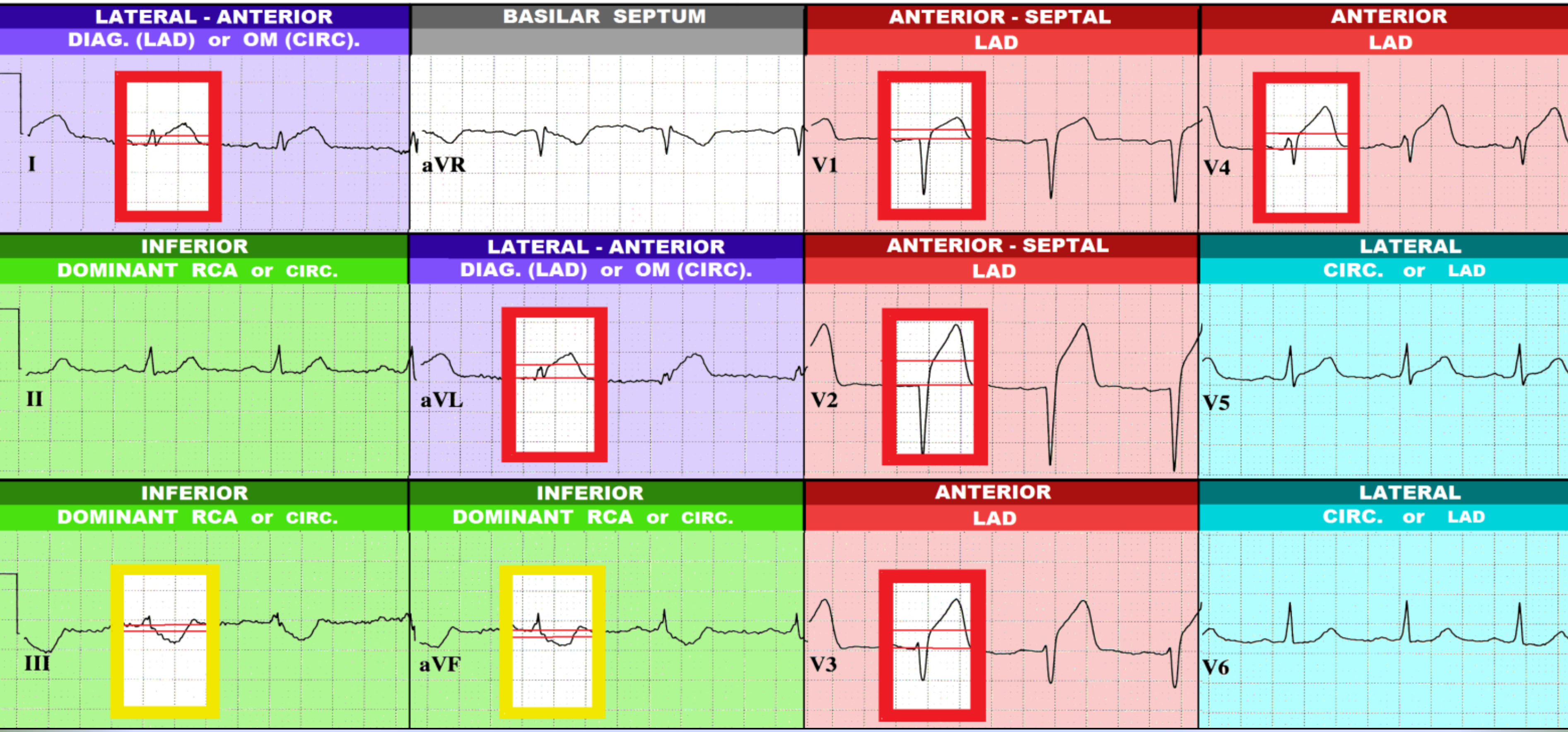
29 yr
Male Caucasian

Vent. rate	75	BPM
PR interval	176	ms
QRS duration	90	ms
QT/QTc	362/404	ms
P–R–T axes	70 50	–11

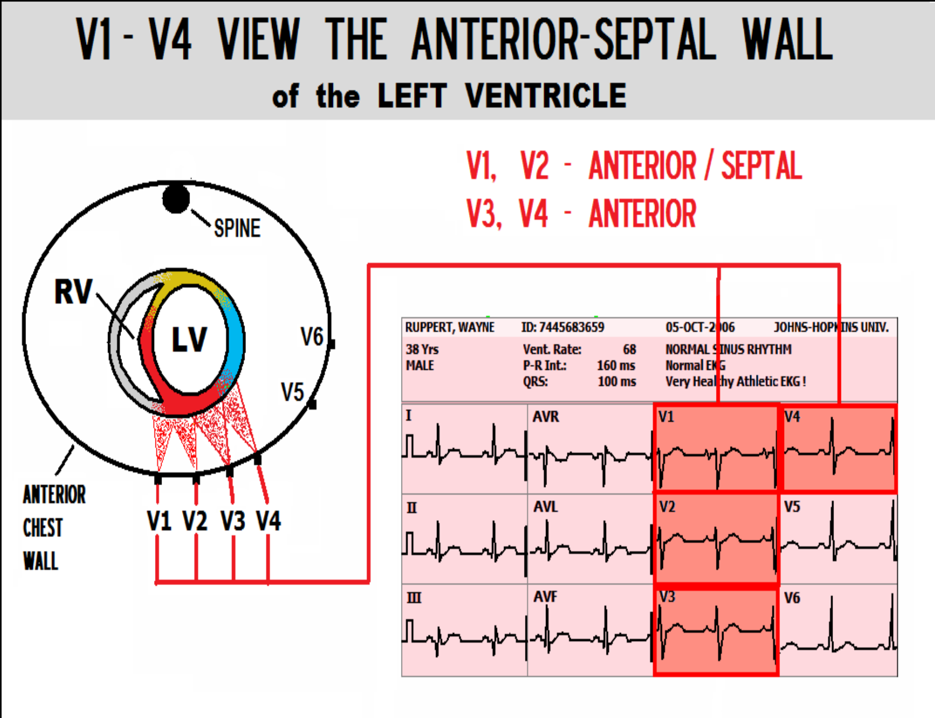
Normal sinus rhythm
Septal infarct , possibly acute
Anterolateral injury pattern
*** ** ** ** * ACUTE MI ** ** ** **
Abnormal ECG

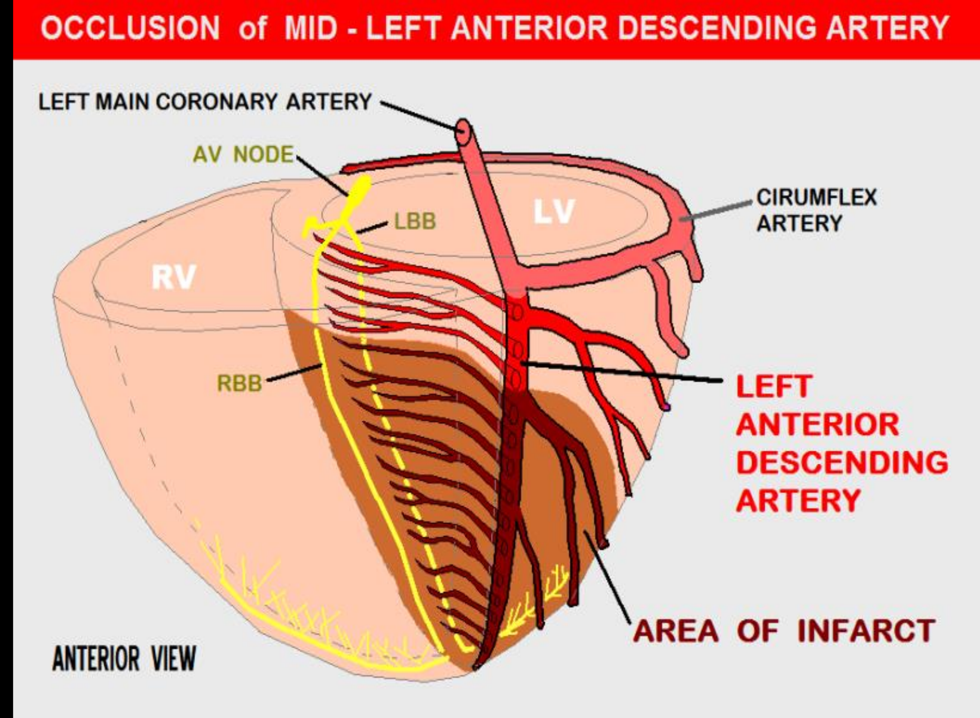
ST SEGMENT ELEVATION

ST SEGMENT DEPRESSION

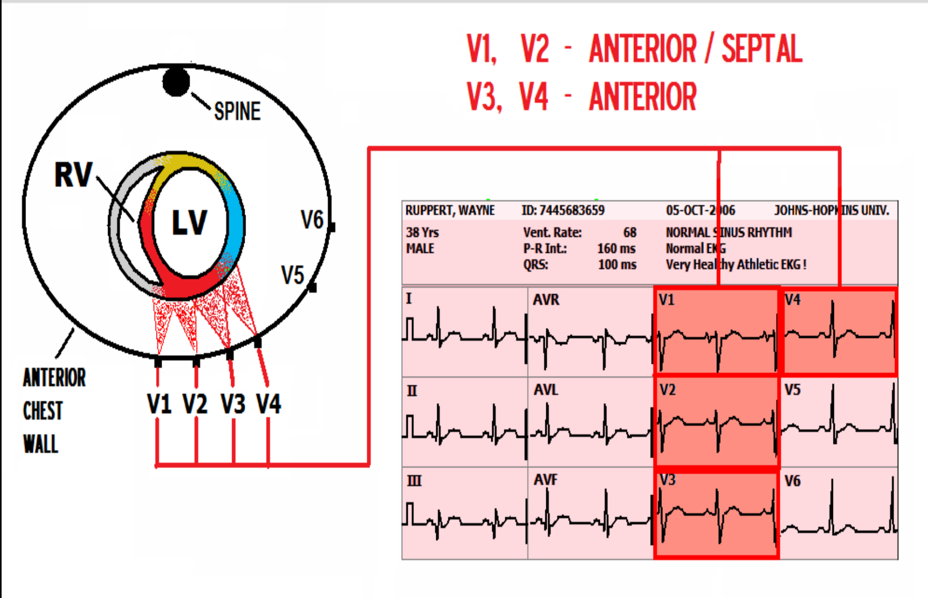


- **Reciprocal ST Depression is NOW PRESENT**
- **Additional ST Elevation is present in Leads I, AVL**

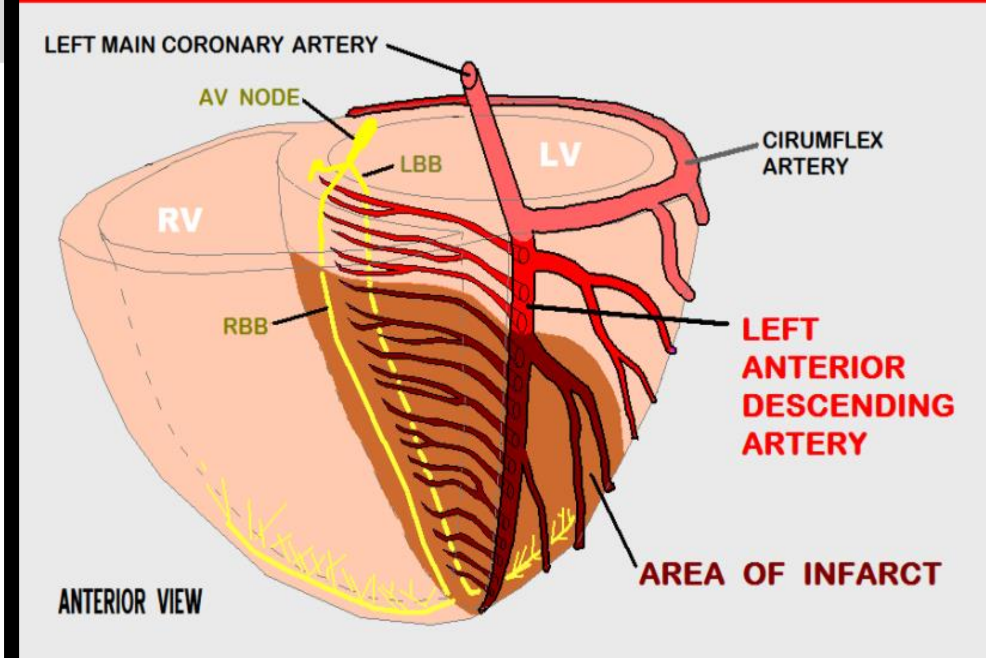

V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL
of the LEFT VENTRICLE
V1, V2 - ANTERIOR / SEPTAL
V3, V4 - ANTERIOR
SPINE
RV
LV
V6
V5
ANTERIOR CHEST WALL
V1 V2 V3 V4
RUPPERT, WAYNE
ID: 7445683659
38 Yrs
MALE
Vent. Rate: 68
P-R Int.: 160 ms
QRS: 100 ms
NORMAL SINUS RHYTHM
Normal EKG
I
AVR
V1
V4
II
AVL
V2
V5
III
AVF
V3
V6


OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY
LEFT MAIN CORONARY ARTERY
AV NODE
LBB
LV
CIRUMFLEX ARTERY
RV
RBB
LEFT ANTERIOR DESCENDING ARTERY
AREA OF INFARCT
ANTERIOR VIEW

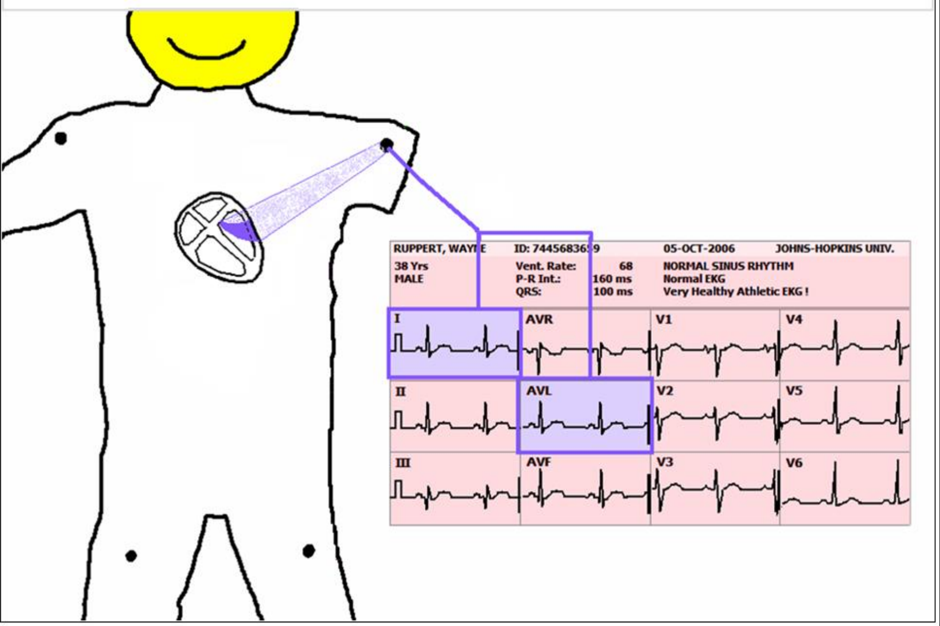
V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL of the LEFT VENTRICLE



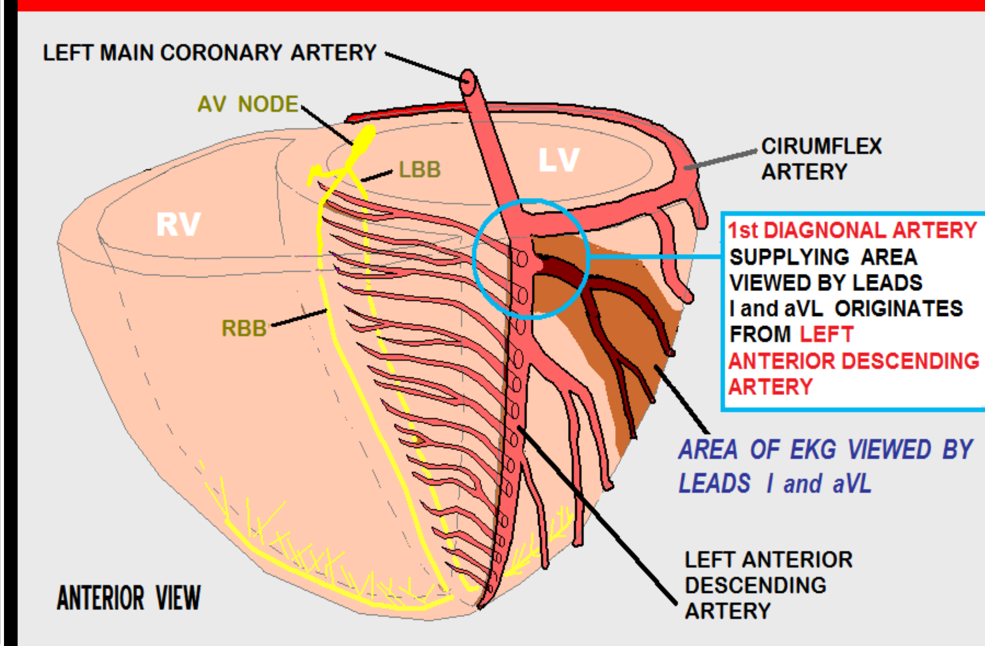
OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY

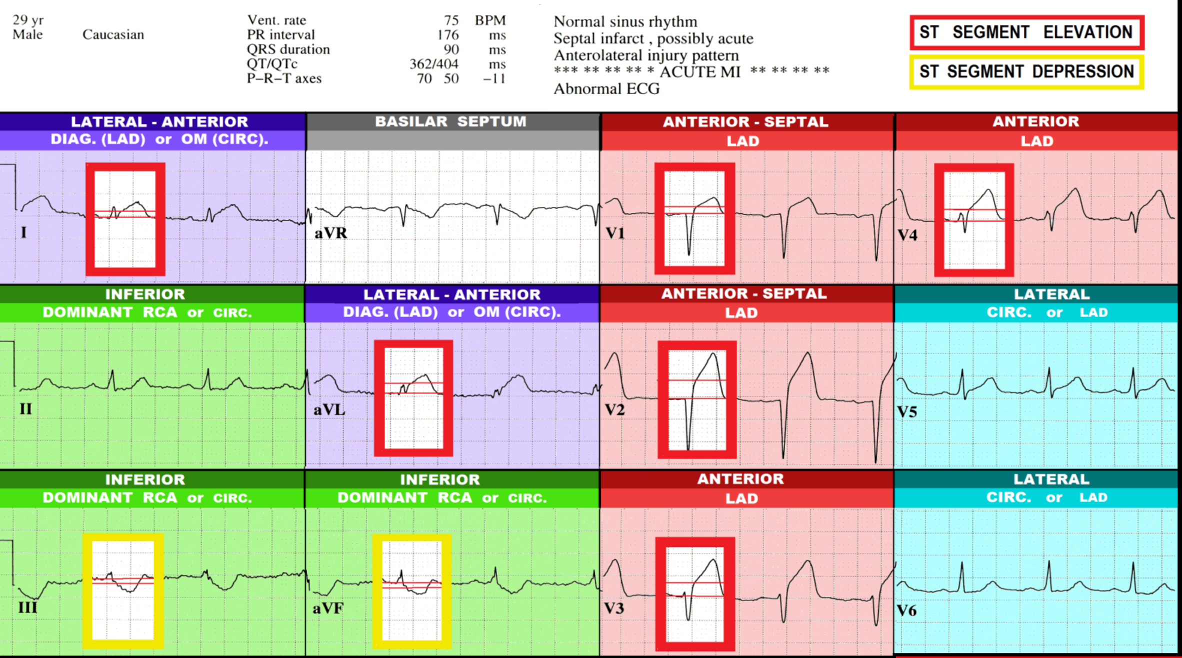


Leads I & AVL view the ANTERIOR-LATERAL JUNCTION

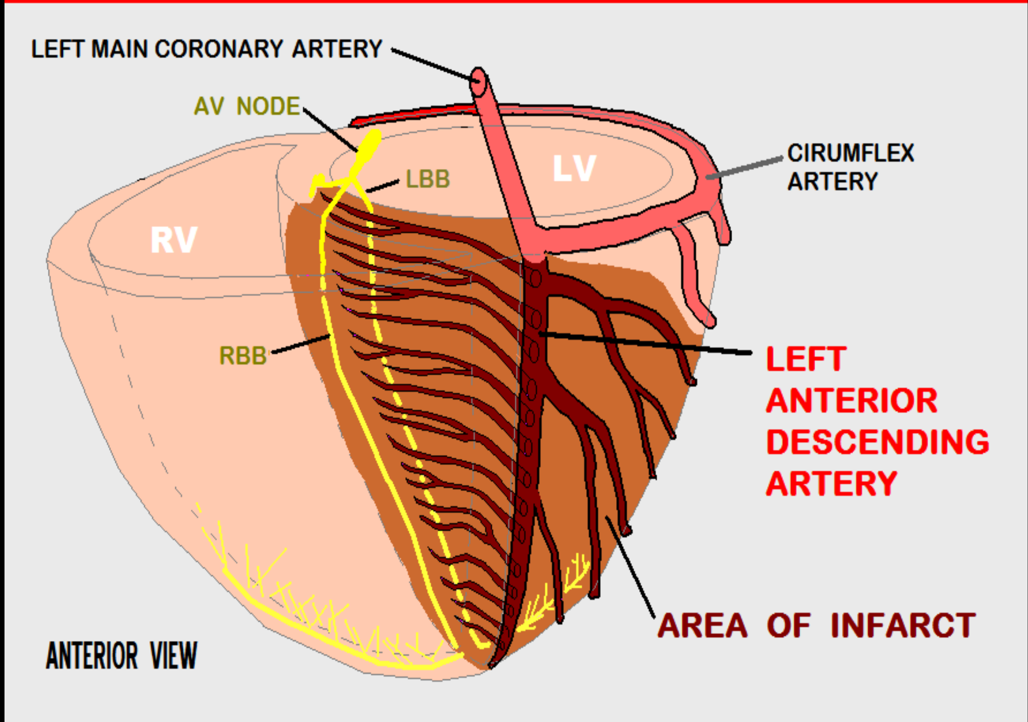


OCCLUSION of DIAGONAL ARTERY

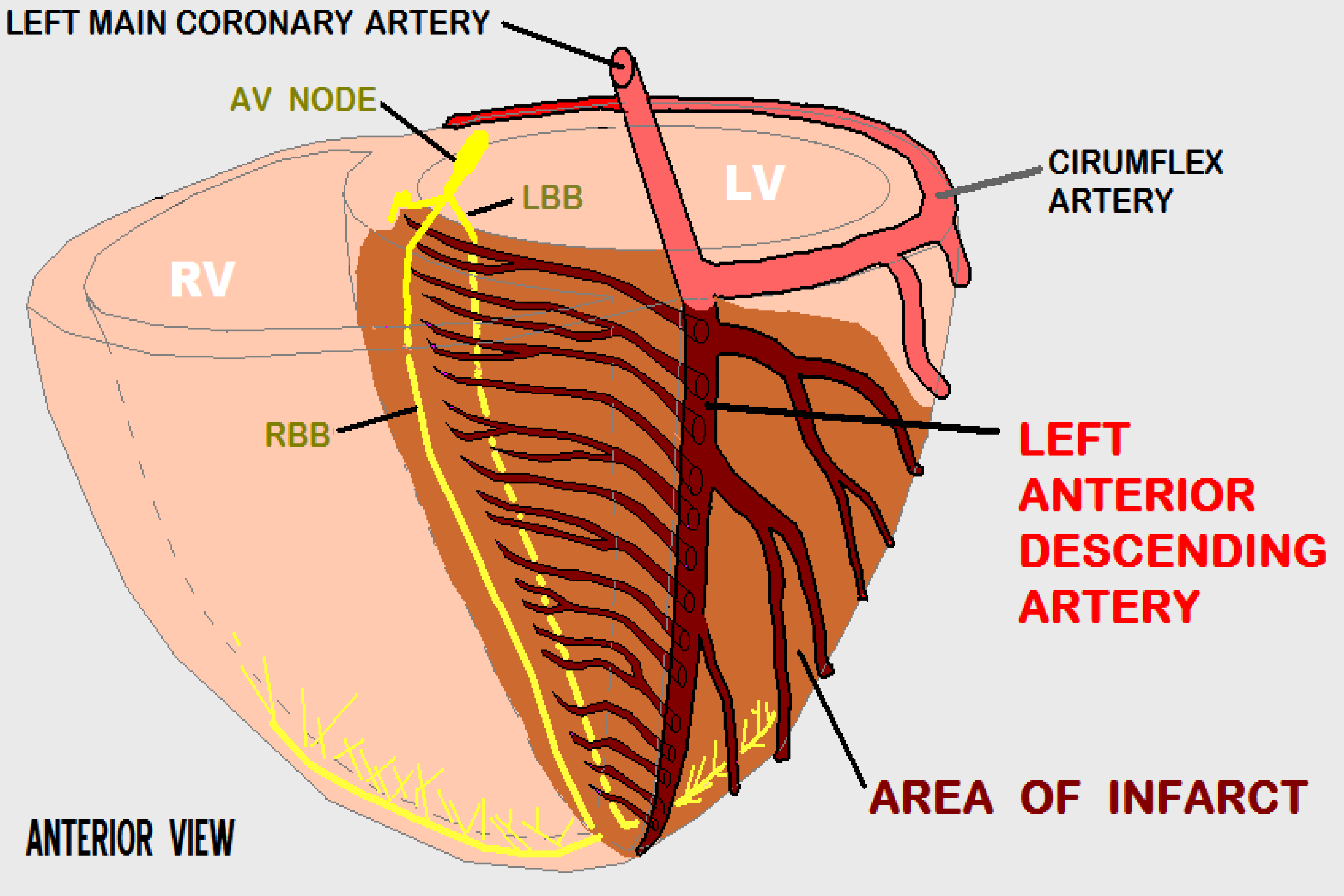




OCCLUSION of PROXIMAL LEFT ANTERIOR DESCENDING ARTERY



OCCLUSION of PROXIMAL LEFT ANTERIOR DESCENDING ARTERY

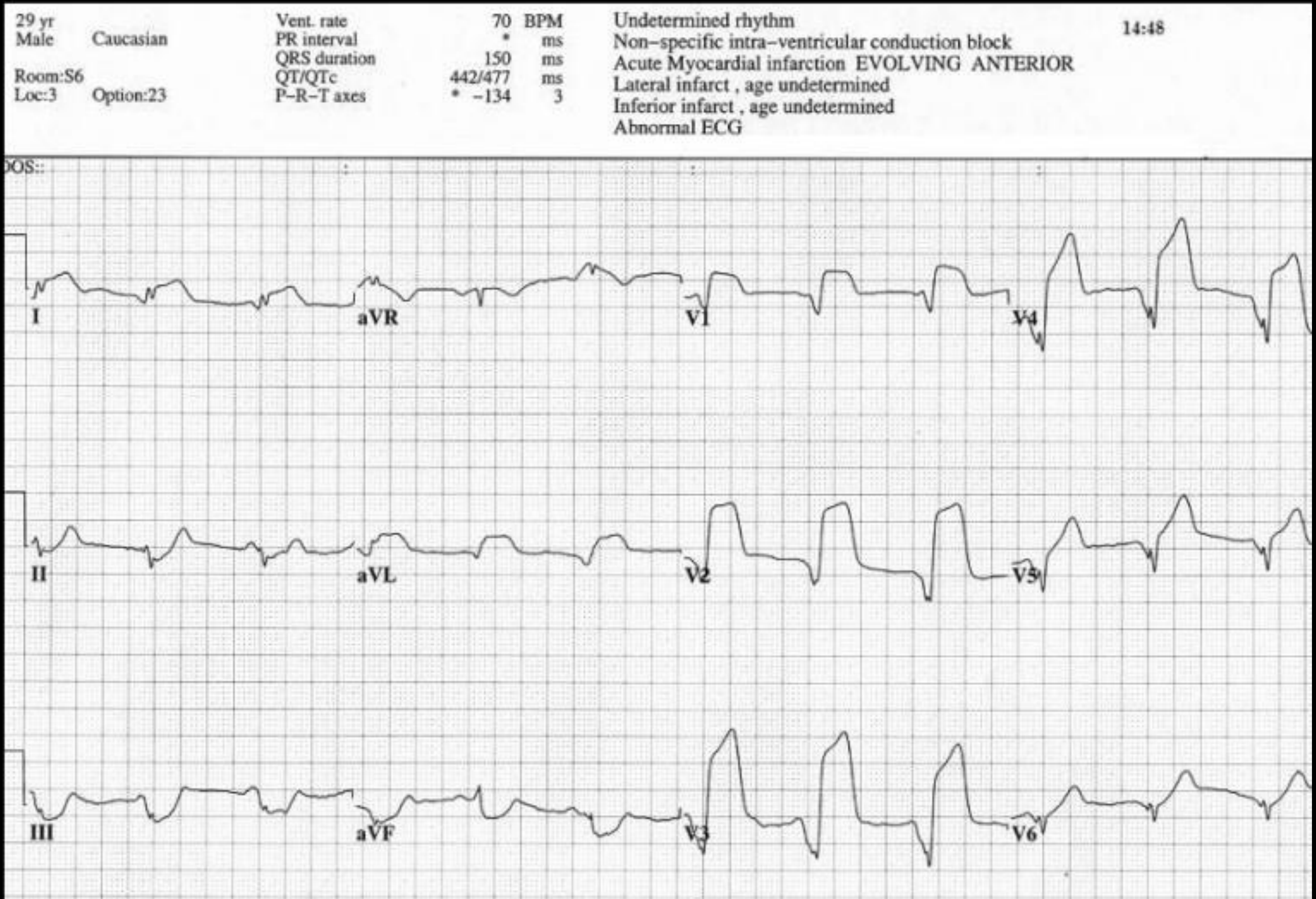


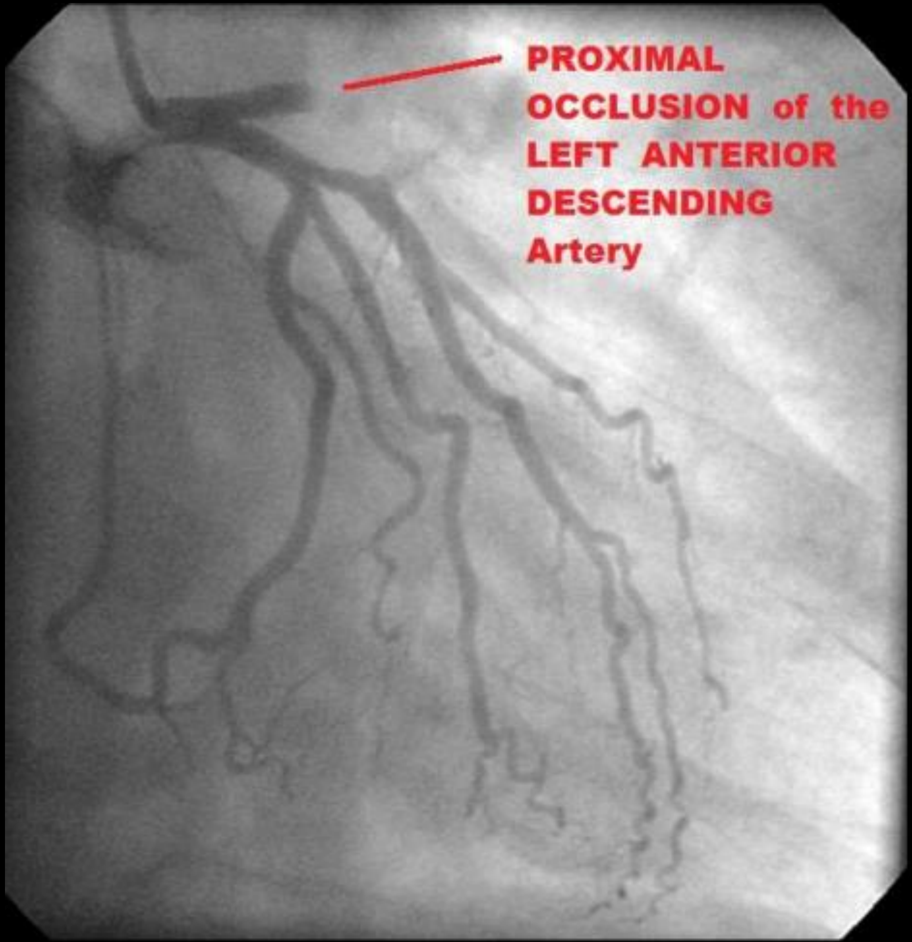
ANTICIPATED COMPLICATIONS of ANTERIOR-SEPTAL WALL STEMI & POSSIBLE INDICATED INTERVENTIONS:	
- CARDIAC ARREST	BCLS / ACLS
- CARDIAC DYSRHYTHMIAS (VT / VF)	ACLS (antiarrhythmics)
- PUMP FAILURE with CARDIOGENIC SHOCK	INOTROPE THERAPY: -DOPAMINE / DOBUTAMINE / LEVOPHED - INTRA-AORTIC BALLOON PUMP (use caution with fluid challenges due to PULMONARY EDEMA)

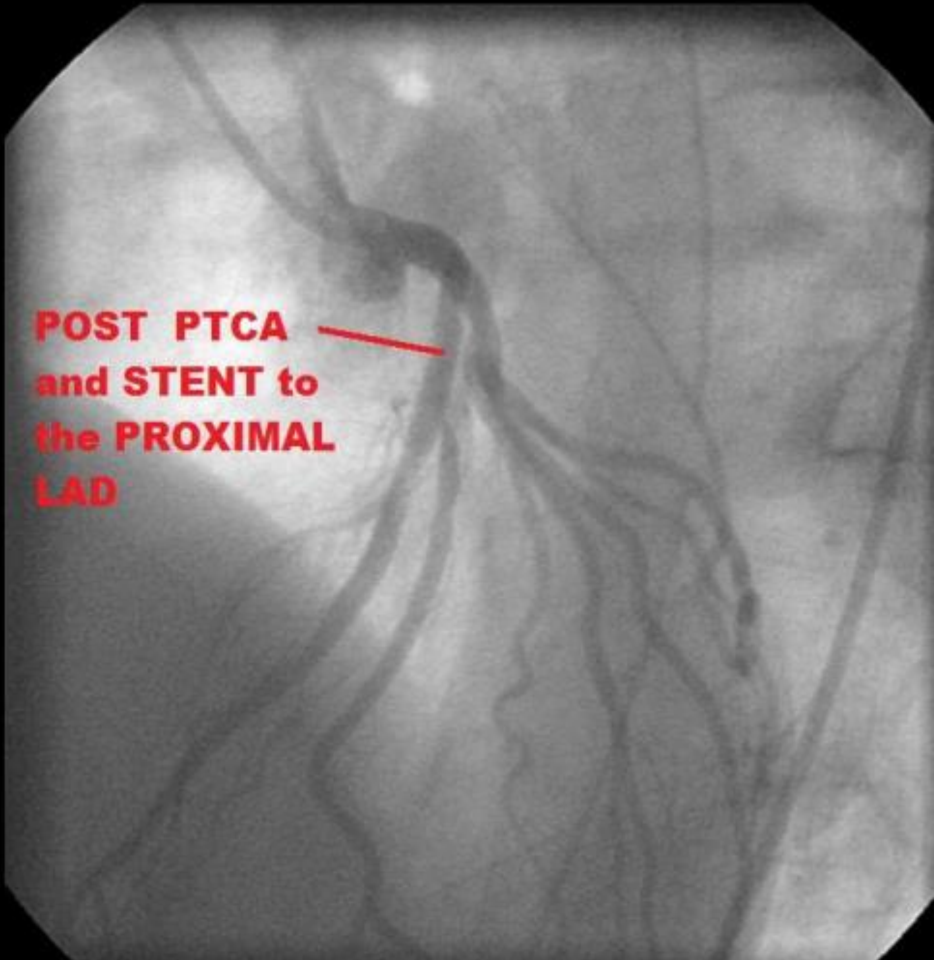
- PULMONARY EDEMA	- CPAP - ET INTUBATION (use caution with dieuretics due to pump failure and hypotension)
- 3rd DEGREE HEART BLOCK - NOT RESPONSIVE TO ATROPINE	TRANSCUTANEOUS or TRANSVENOUS PACING

WHILE AWAITING THE CATH TEAM, THE PATIENT BEGAN VOMITING. SKIN BECAME ASHEN & DIAPHORETIC. REPEAT BP = 50/30.

-WHAT THERAPEUTIC INTERVENTIONS SHOULD BE IMPLMENTED AT THIS POINT ?

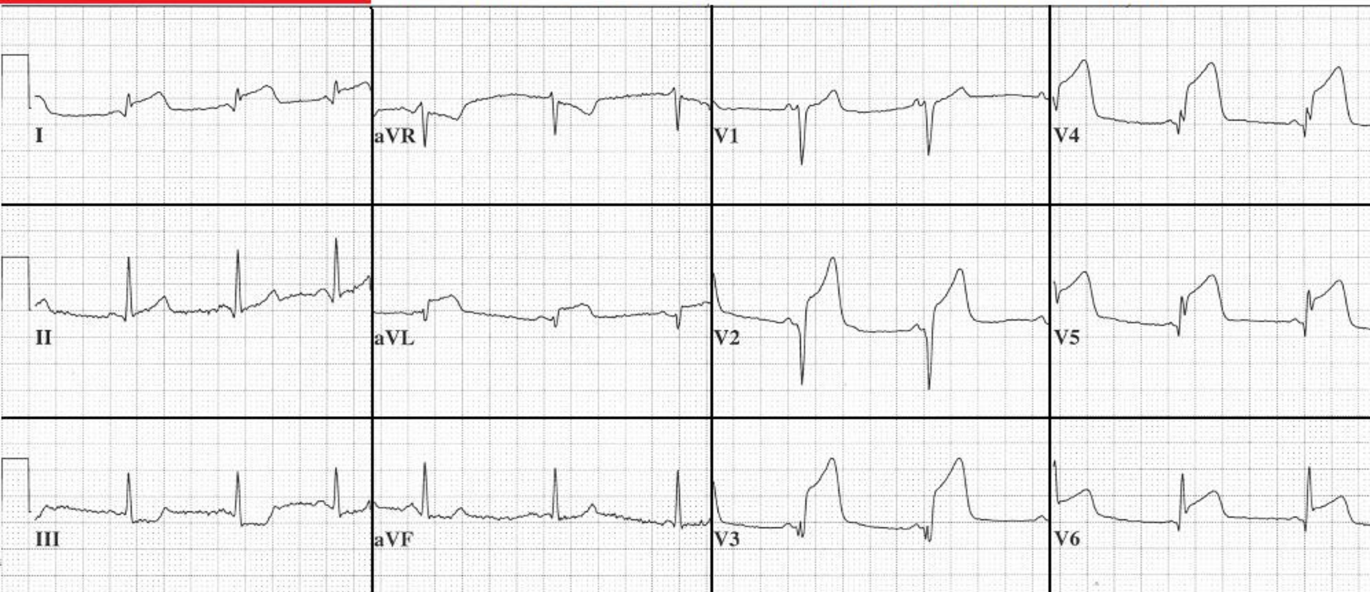



PROXIMAL
OCCLUSION of the
LEFT ANTERIOR
DESCENDING
Artery


POST PTCA
and STENT to
the PROXIMAL
LAD

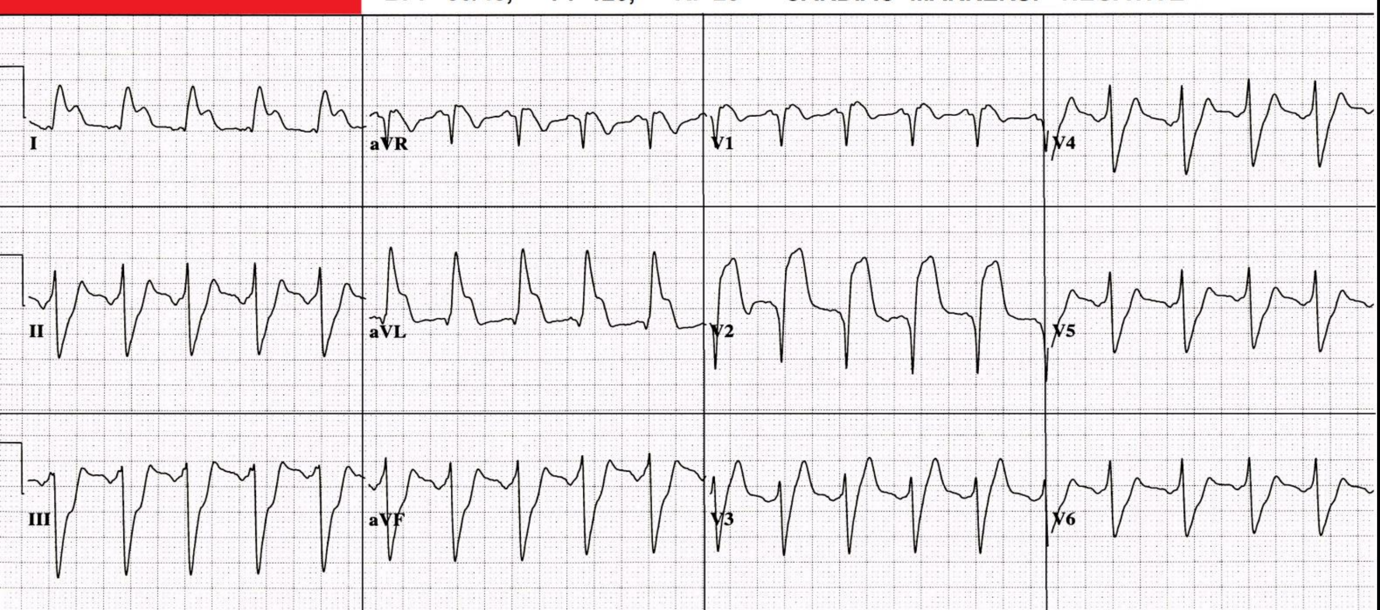
PATIENT A:

44 y/o MALE, CHEST PAIN x 1 HOUR,
BP: 78/46, P: 70, R: 28. CARDIAC MARKERS: NEGATIVE



PATIENT B:

36 y/o MALE, CHEST PAIN x 1 HOUR,
BP: 80/48, P: 120, R: 28 CARDIAC MARKERS: NEGATIVE



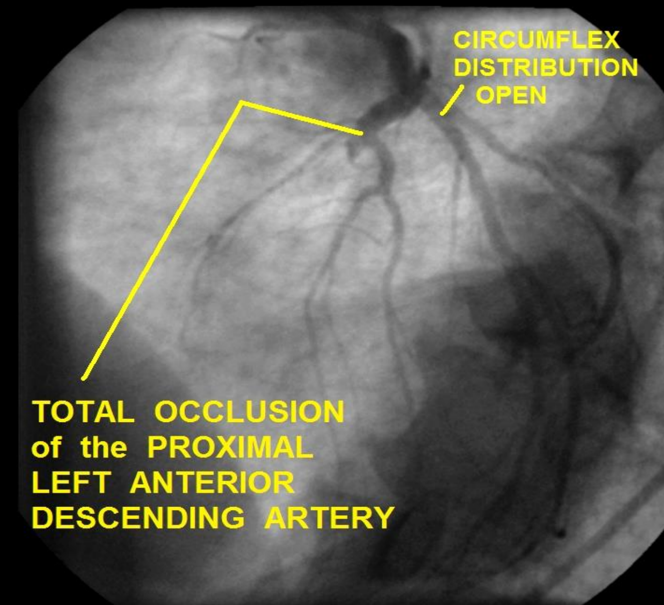
WHO SHOULD GO TO THE CATH LAB FIRST ?

And . . . .

WHAT WOULD YOU DO WITH THE PATIENT WHO DID NOT GO TO THE CATH LAB ?

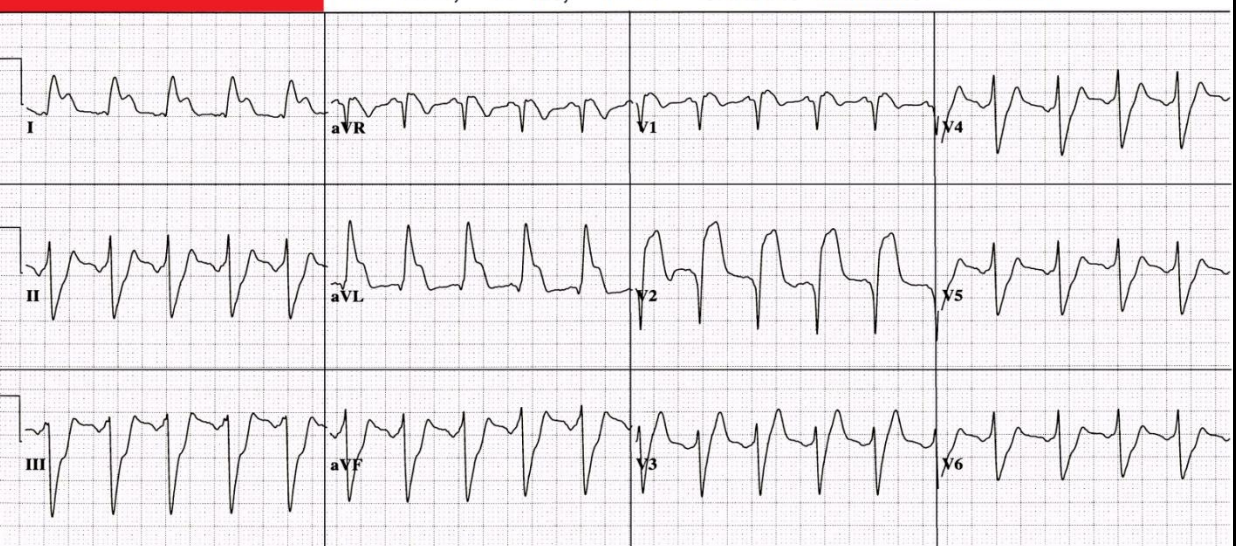
PATIENT A:

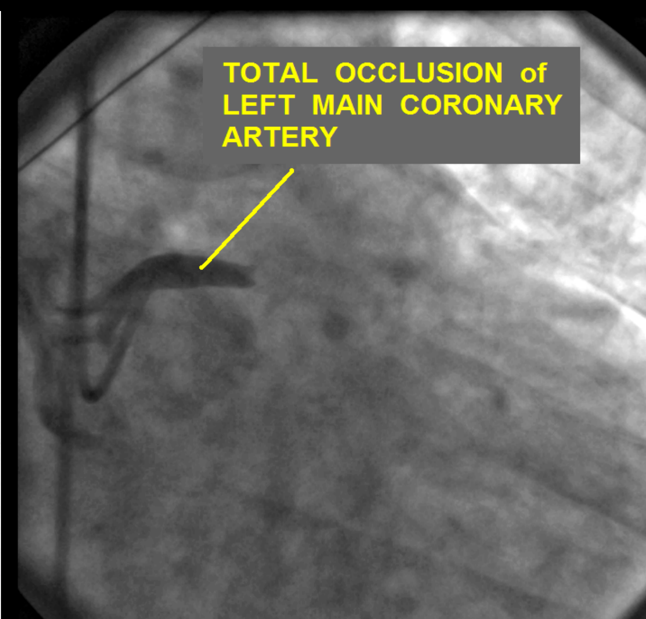
44 y/o MALE, CHEST PAIN x 1 HOUR,
BP: 78/46, P: 70, R: 28. CARDIAC MARKERS: NEGATIVE



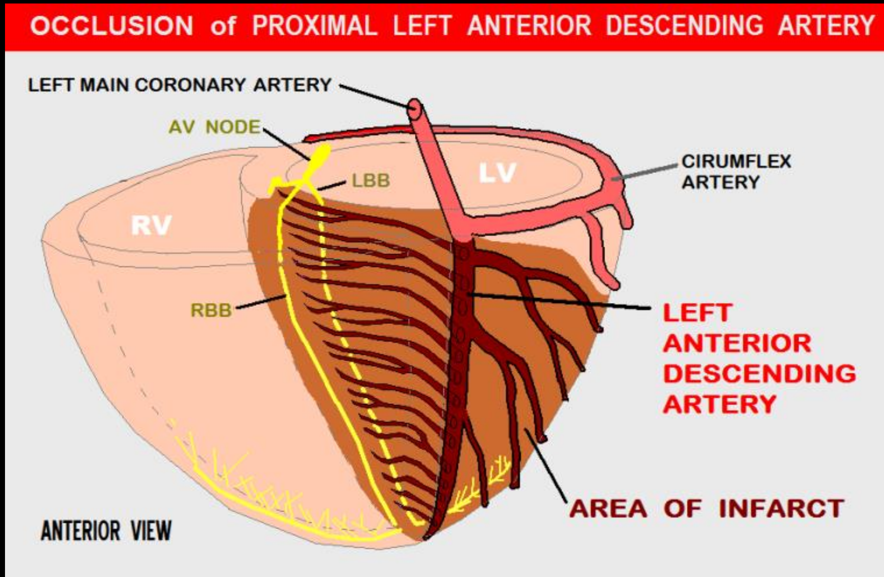
PATIENT B:

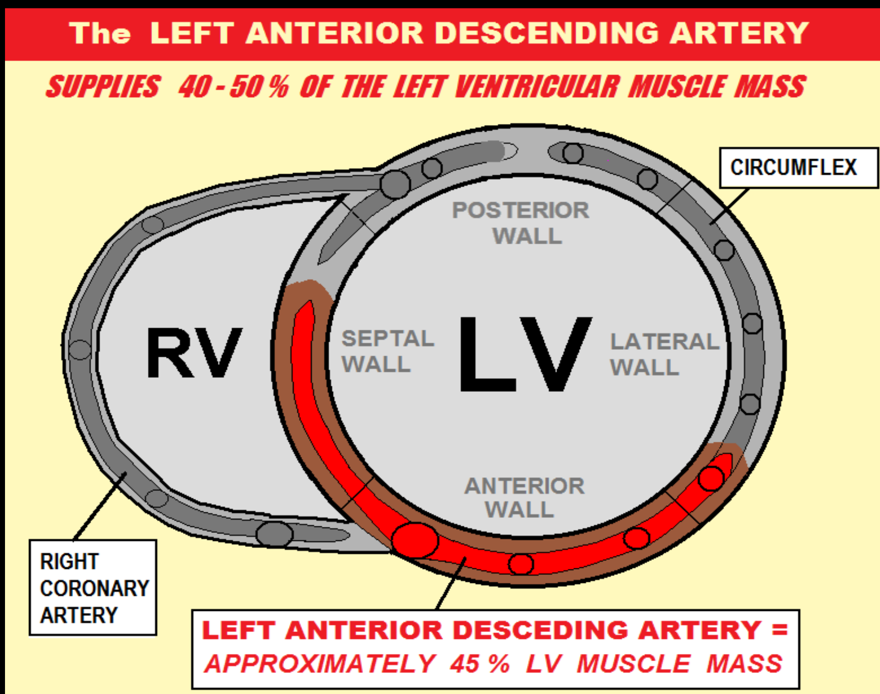
36 y/o MALE, CHEST PAIN x 1 HOUR,
BP: 80/48, P: 120, R: 28 CARDIAC MARKERS: NEGATIVE



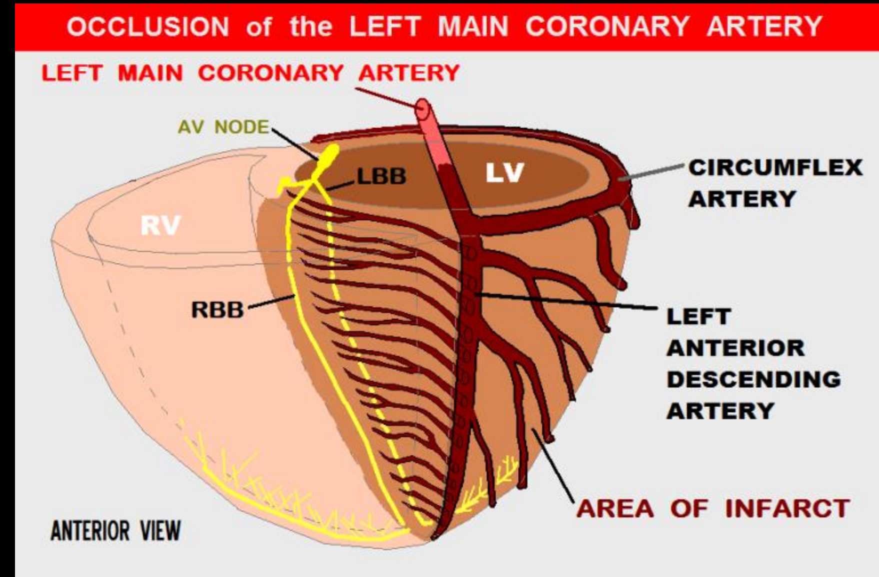


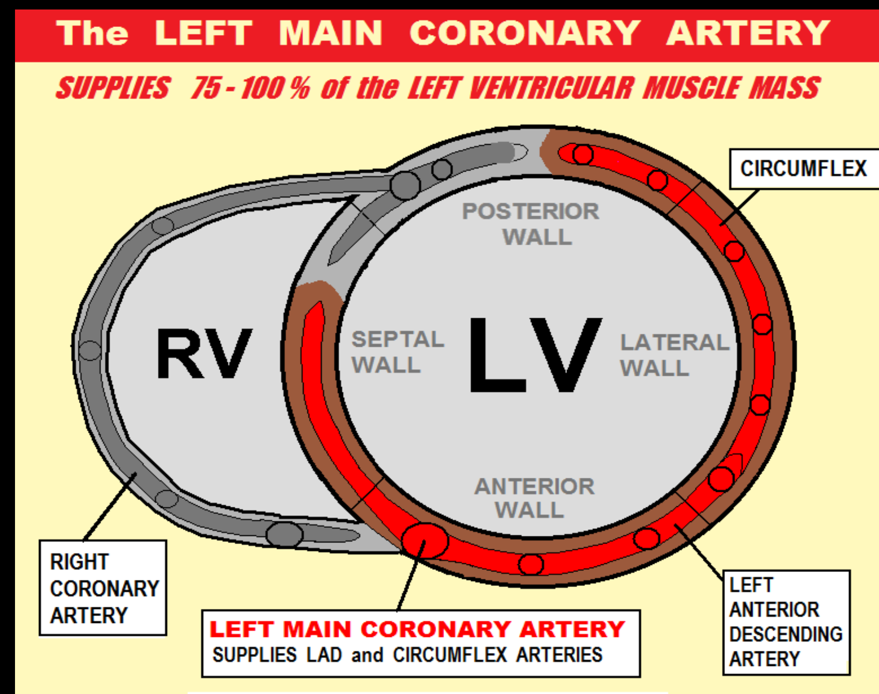
PATIENT A:





PATIENT B:



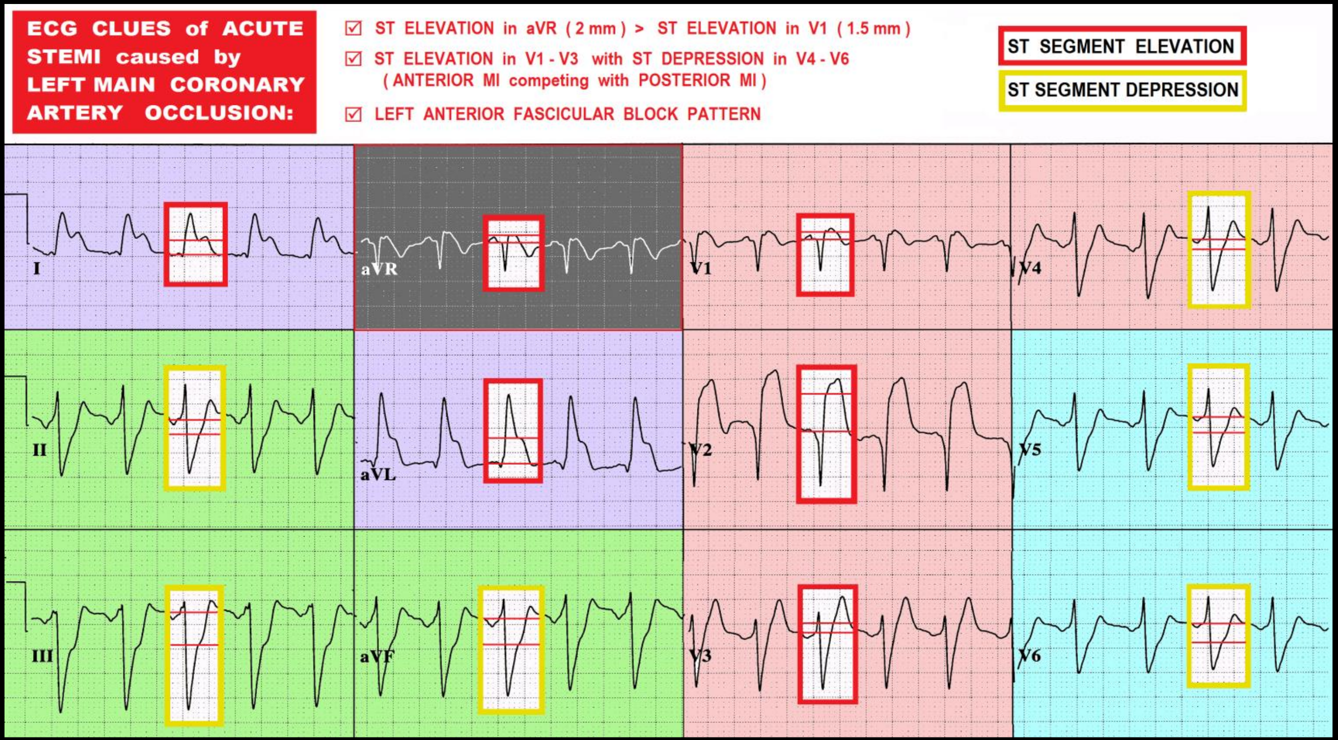


ECG Clues . . . for IDENTIFYING STEMI CAUSED BY LEFT MAIN CORONARY ARTERY occlusion:

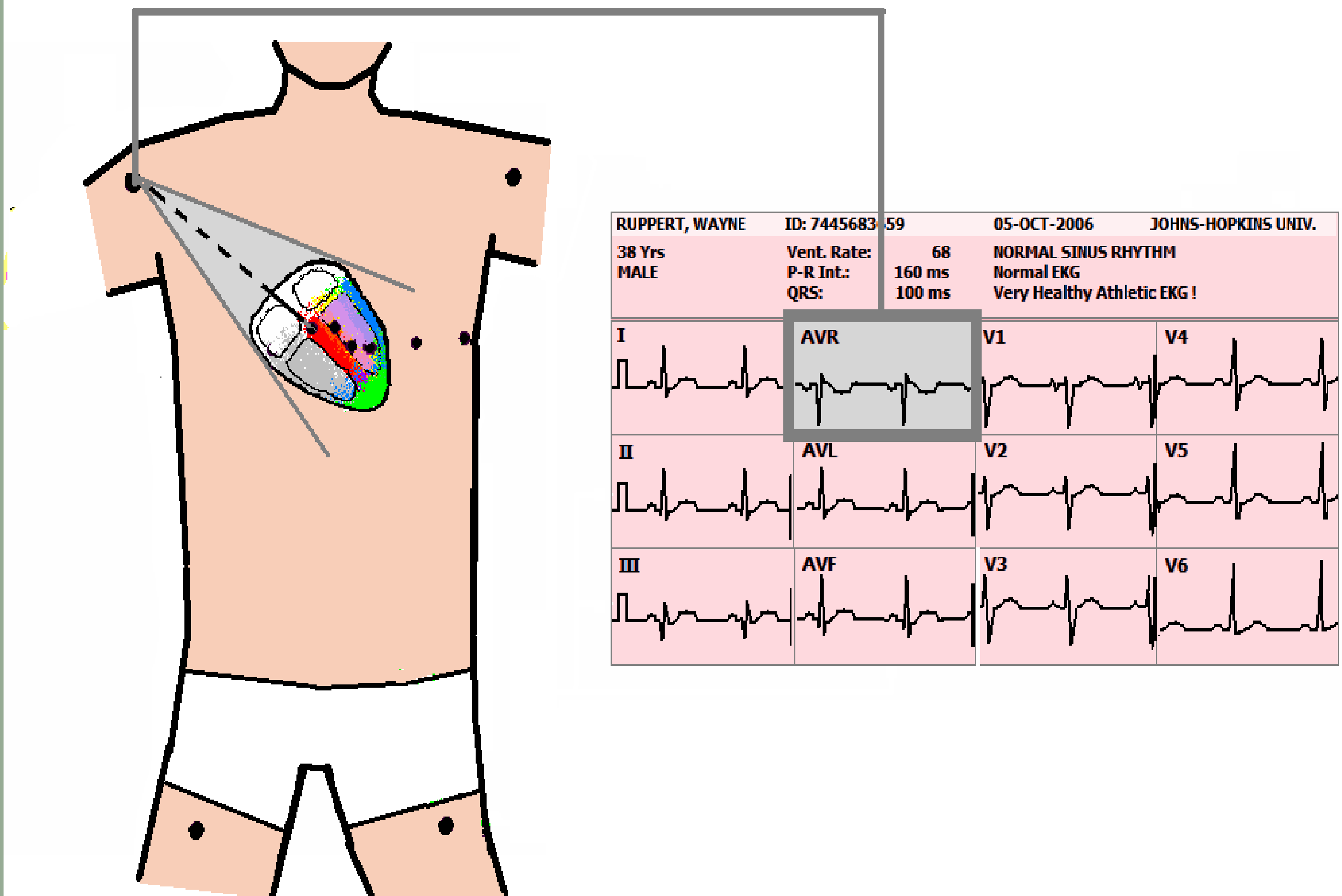
- ☑ ST ELEVATION in ANTERIOR LEADS (V1 - V4) and LATERAL LEADS (V5 & V6)
- ☑ ST DEPRESSION or ISOELCTRIC J POINTS may be seen in V LEADS . . . . mainly V2 and/or V3 *caused by COMPETING FORCES of ANTERIOR vs. POSTERIOR WALL MI.* *+
  - → NOTE: it is very unusual to see ST DEPRESSION in V LEADS with isolated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

* Kurisu et al, HEART 2004, SEPTEMBER: 90 (9): 1059-1060

\+ Yamaji et al, JACC vol. 38, No. 5, 2001, November 1, 2001:1348-54

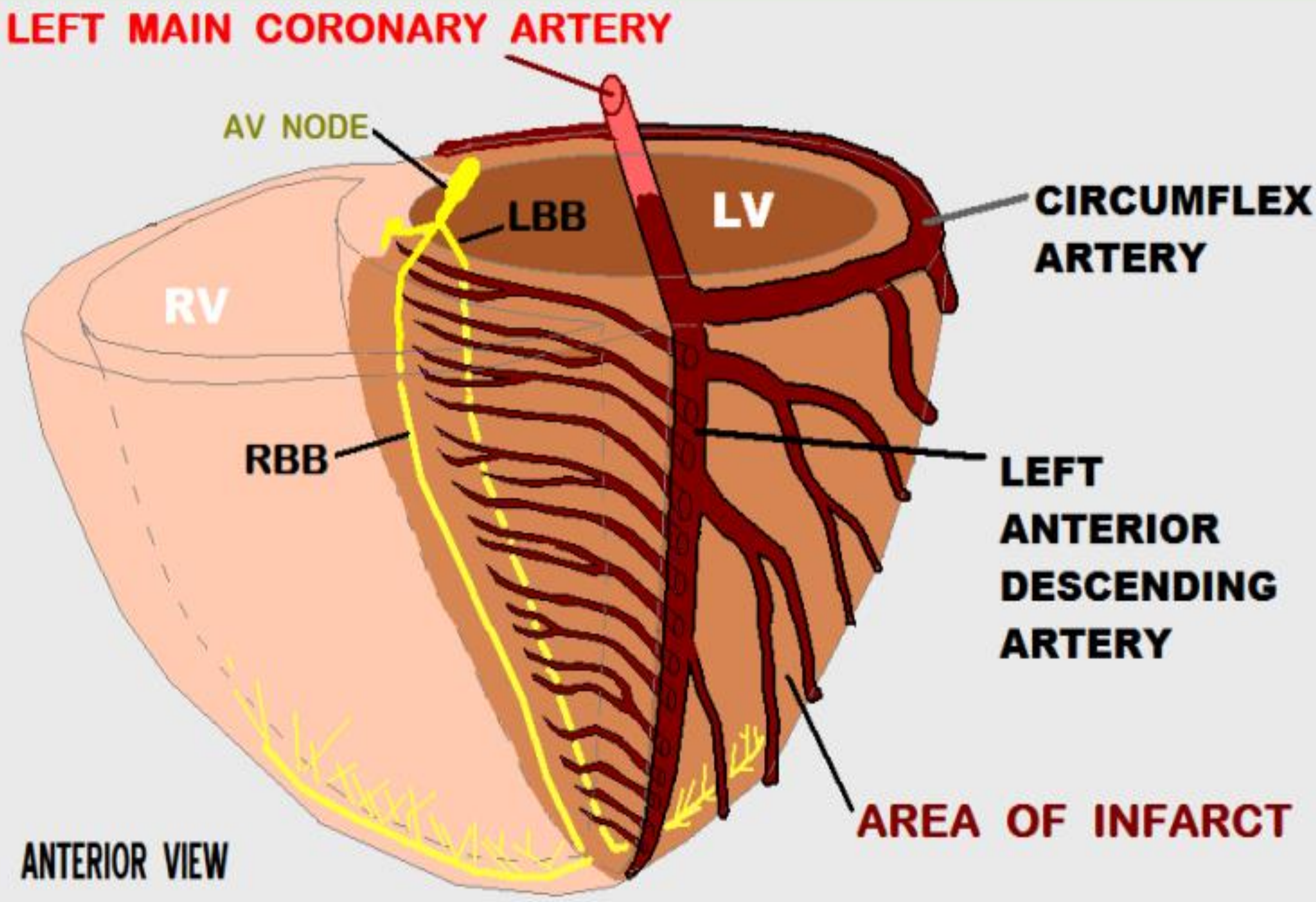


Lead AVR Views the BASILAR SEPTUM (region of the Bundle of His)

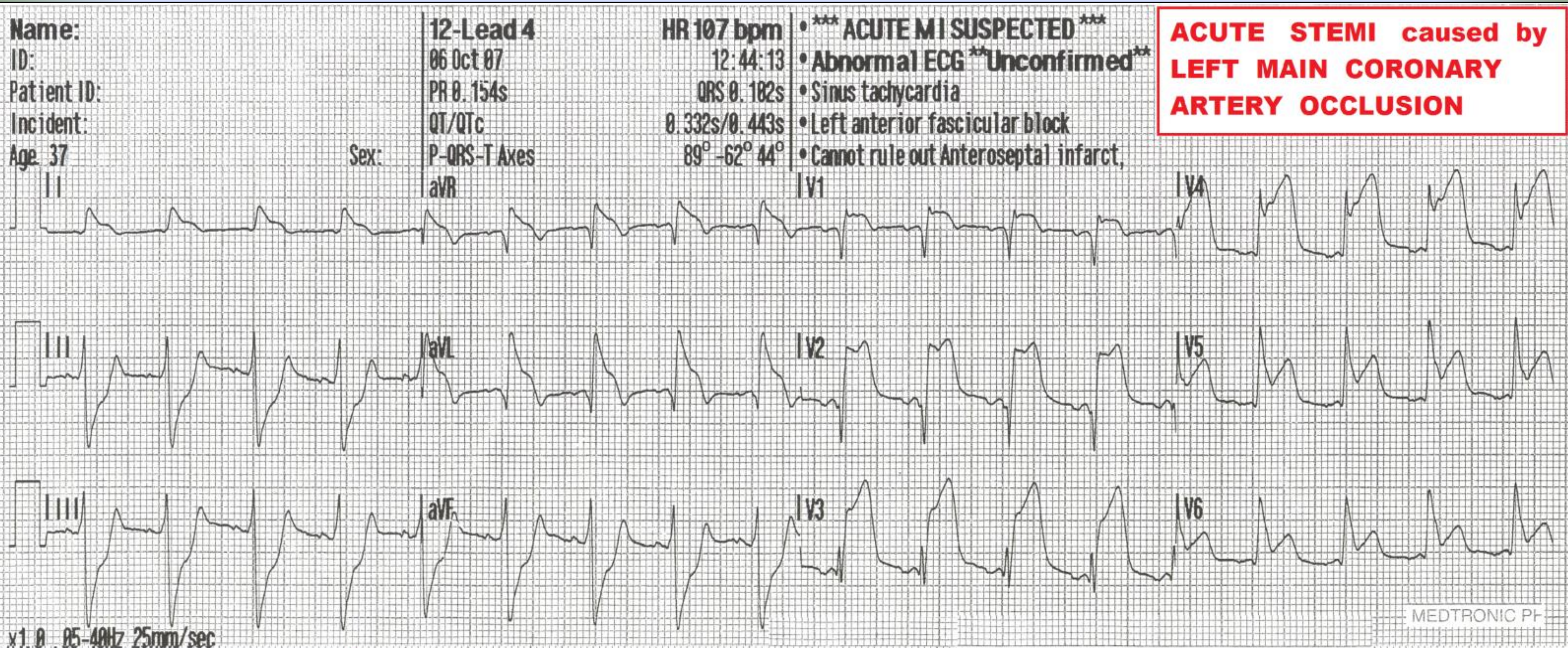


In STEMI with ST-Segment Elevation in Lead AVR,
This is indicative of
Left Main Coronary Artery Occlusion . . .

OCCLUSION of the LEFT MAIN CORONARY ARTERY



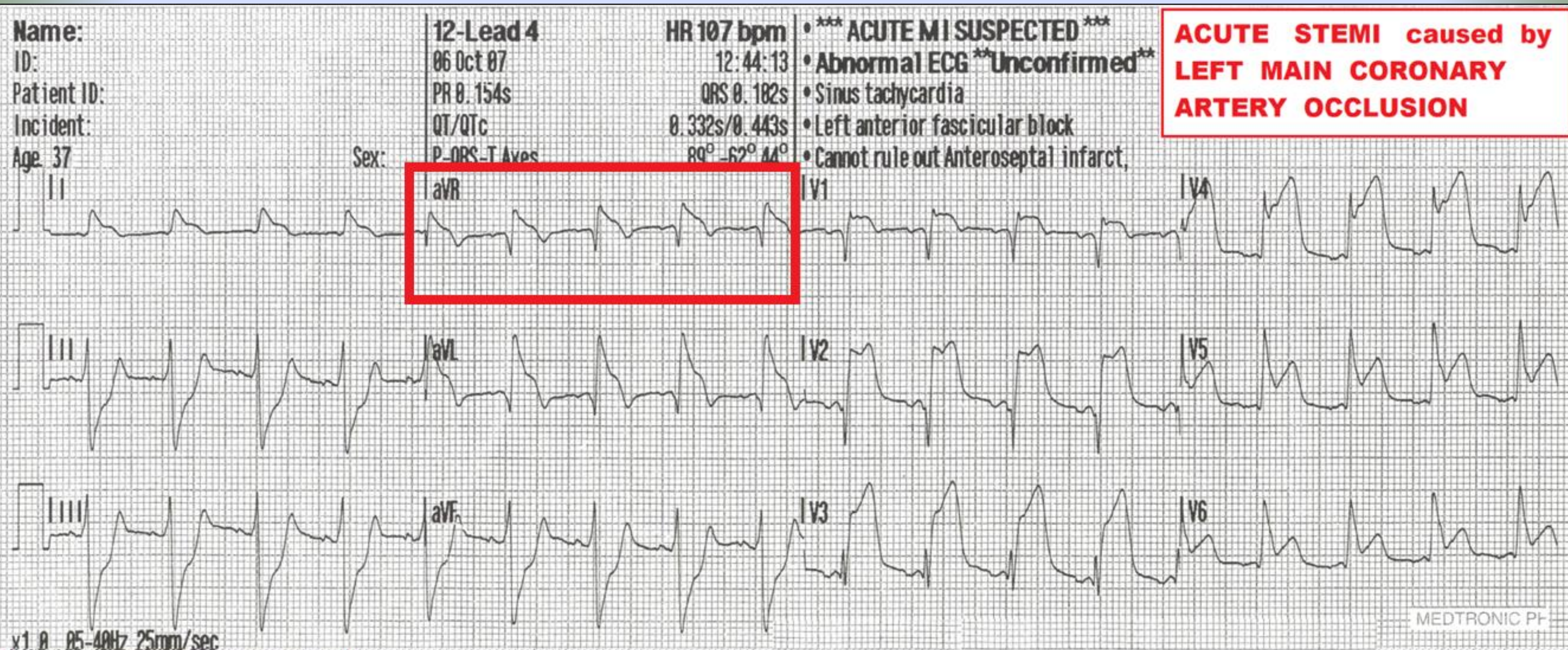
ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION



ECG CLUES of ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION:

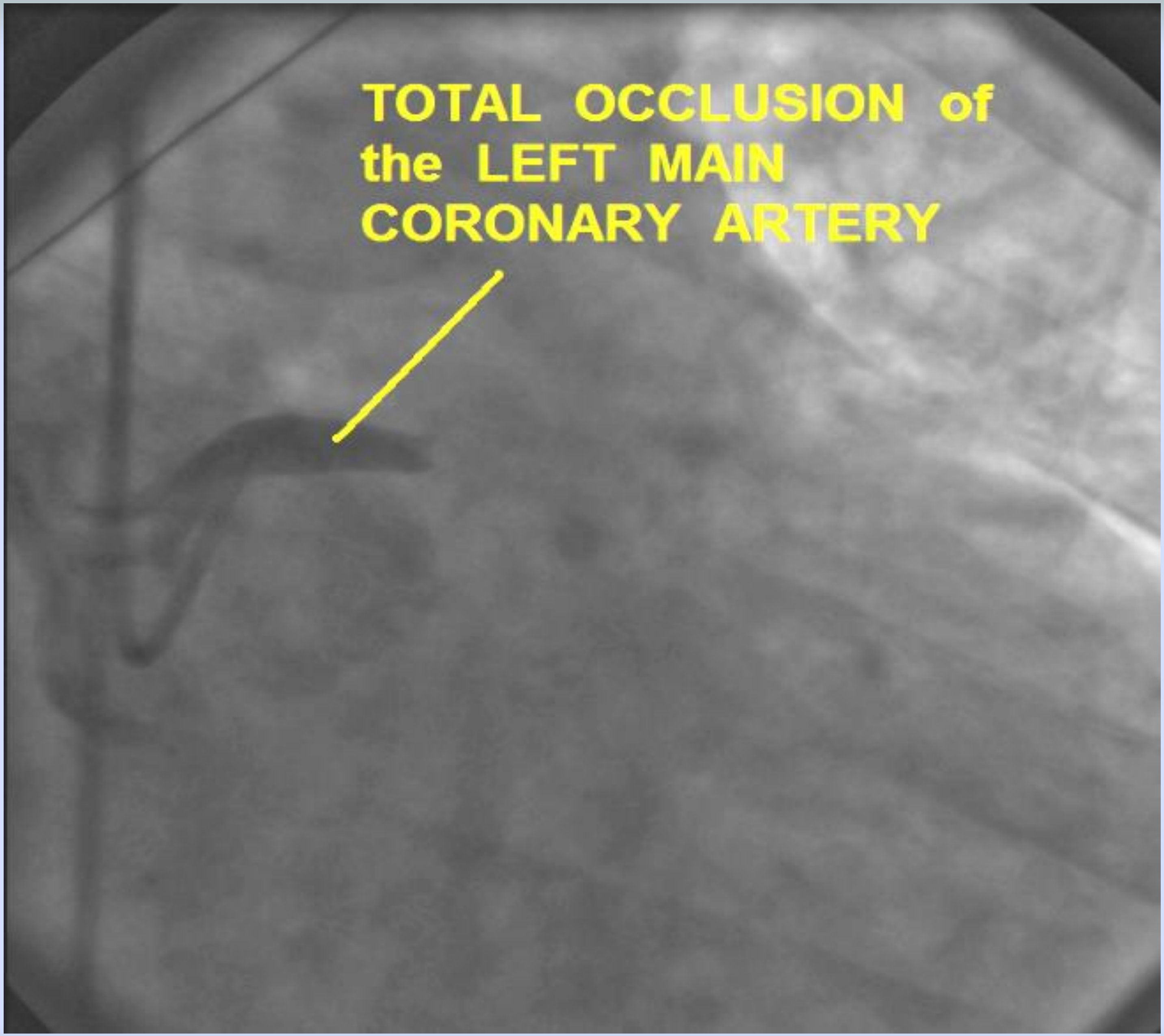
- ☑ ST ELEVATION in LEADS I, aVL, V1 - V6
- ☑ ST ELEVATION in aVR GREATER THAN 0.5 mm
- ☑ ST ELEVATION in aVR GREATER THAN LEAD V1
- ☑ LEFT ANTERIOR FASCICULAR BLOCK PATTERN

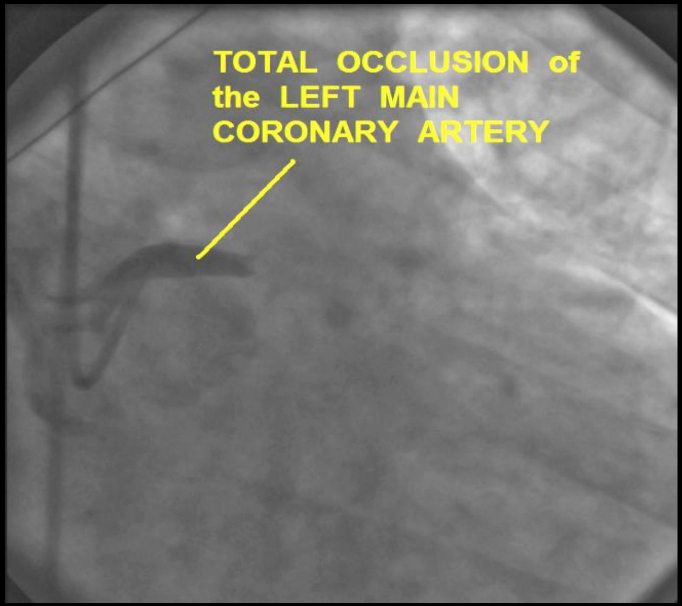
ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION

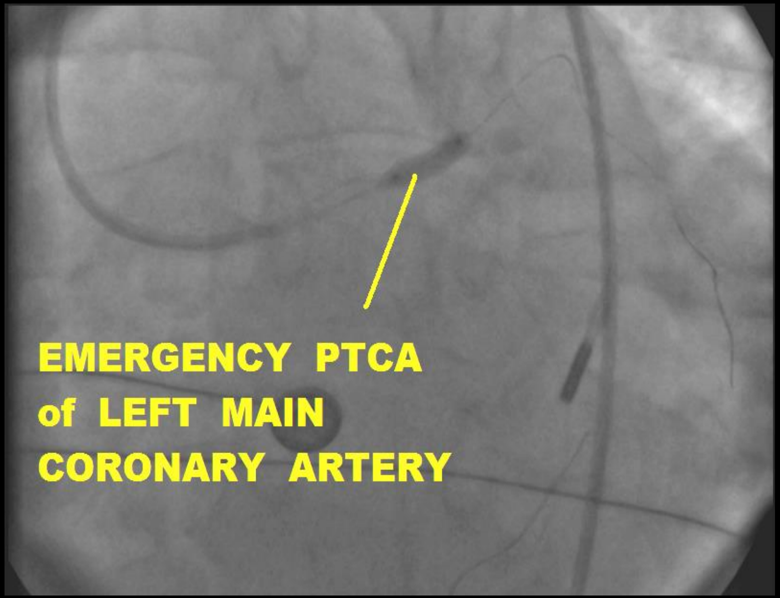


ECG CLUES of ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION:

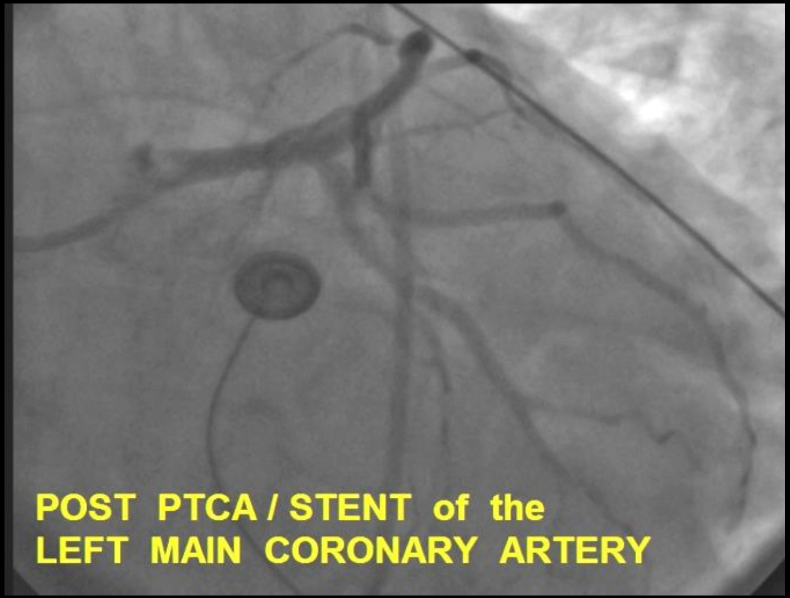
- ☑ ST ELEVATION in LEADS I, aVL, V1 - V6
- ☑ ST ELEVATION in aVR GREATER THAN 0.5 mm
- ☑ ST ELEVATION in aVR GREATER THAN LEAD V1
- ☑ LEFT ANTERIOR FASCICULAR BLOCK PATTERN


TOTAL OCCLUSION of
the LEFT MAIN
CORONARY ARTERY





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

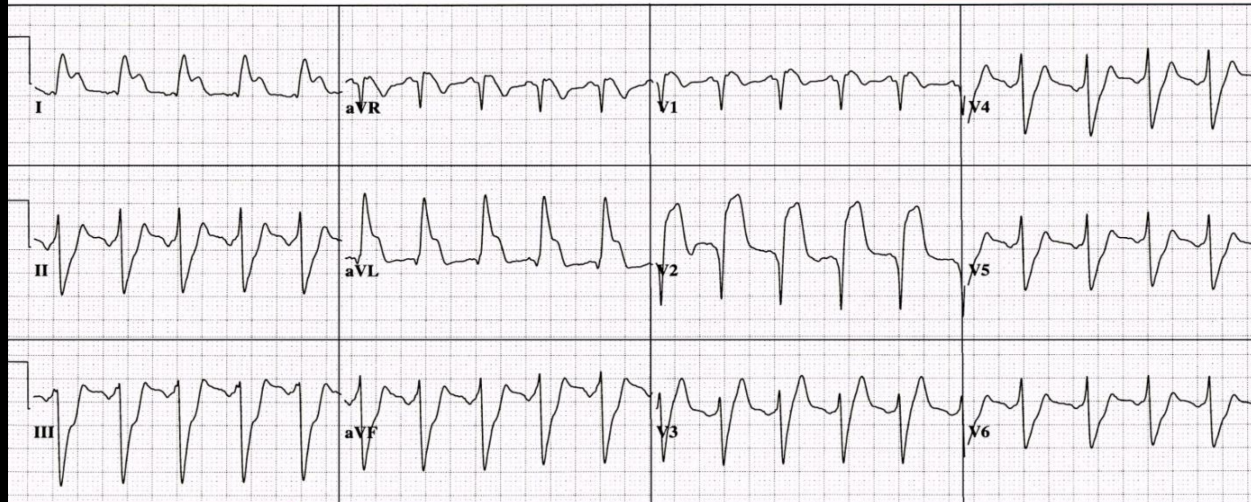


36 yr
Male Caucasian
Room:C-
Loc:3

Vent. rate	123	BPM
PR interval	96	ms
QRS duration	130	ms
QT/QTc	310/443	ms
P–R–T axes	* –53	43

Sinus tachycardia with short PR
Left ventricular hypertrophy with QRS widening
Cannot rule out Septal infarct , age undetermined
Lateral injury pattern
*** ** ** ** * ACUTE MI ** ** ** **

ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION



ECG CLUES of ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION:

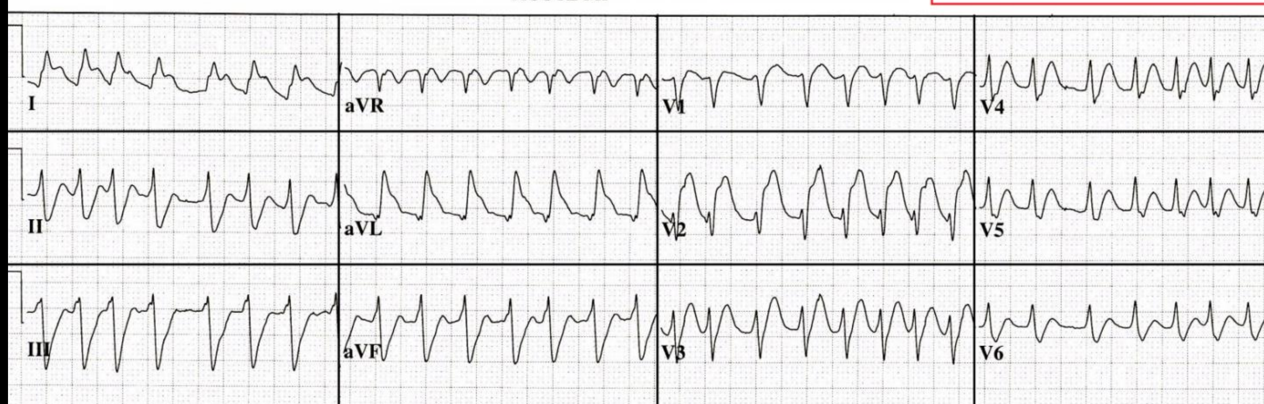
- ☑ ST ELEVATION in leads I and aVL
- ☑ INCONSISTENCY of ST SEGEMENT in leads V1 - V6 : V1 - V3 ST ELEVATION, V4 - V6 ST DEPRESSION (COMPETING FORCES of ANTERIOR vs. POSTERIOR M.I.)
- ☑ PATTERN of LEFT ANTERIOR FASCICULAR BLOCK (POS. QRS lead I; NEG rS leads II, III)
- ☑ ST ELEVATION in lead aVR > 0.5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

43 yr
Male

Vent. rate	183	BPM
PR interval	*	ms
QRS duration	106	ms
QT/QTc	240/418	ms
P–R–T axes	* –34	–18

Atrial fibrillation with rapid ventricular response
with premature ventricular or aberrantly conducted complexes
Left axis deviation
ST elevation consider anterolateral injury or acute infarct
** ** ** ** * ACUTE MI * ** ** ** **

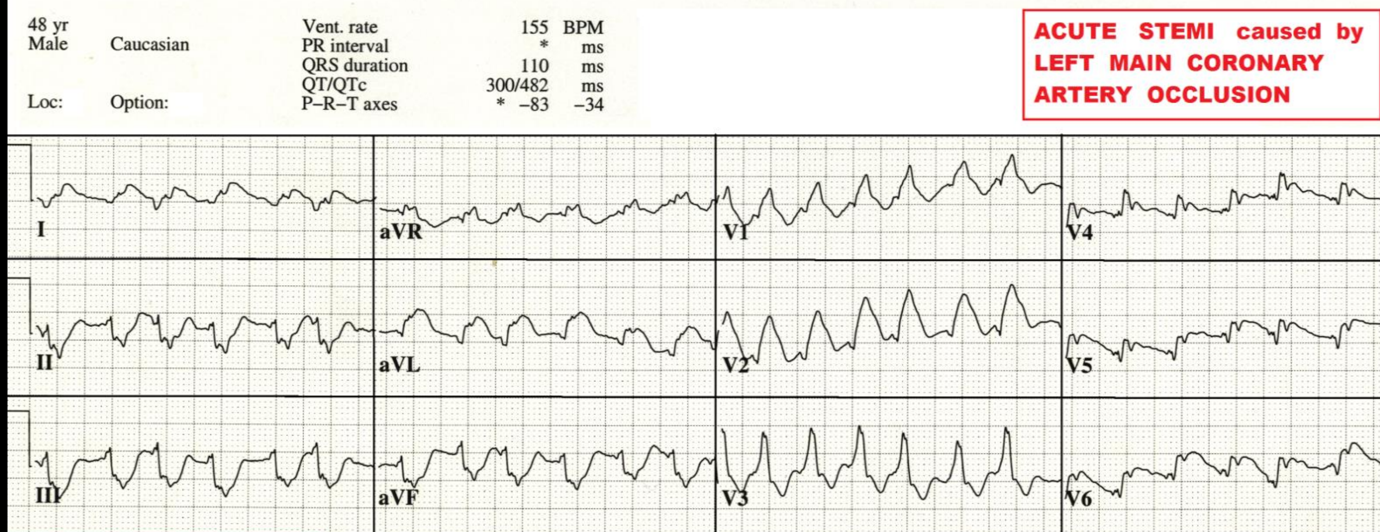
ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION



ECG CLUES of ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION:

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

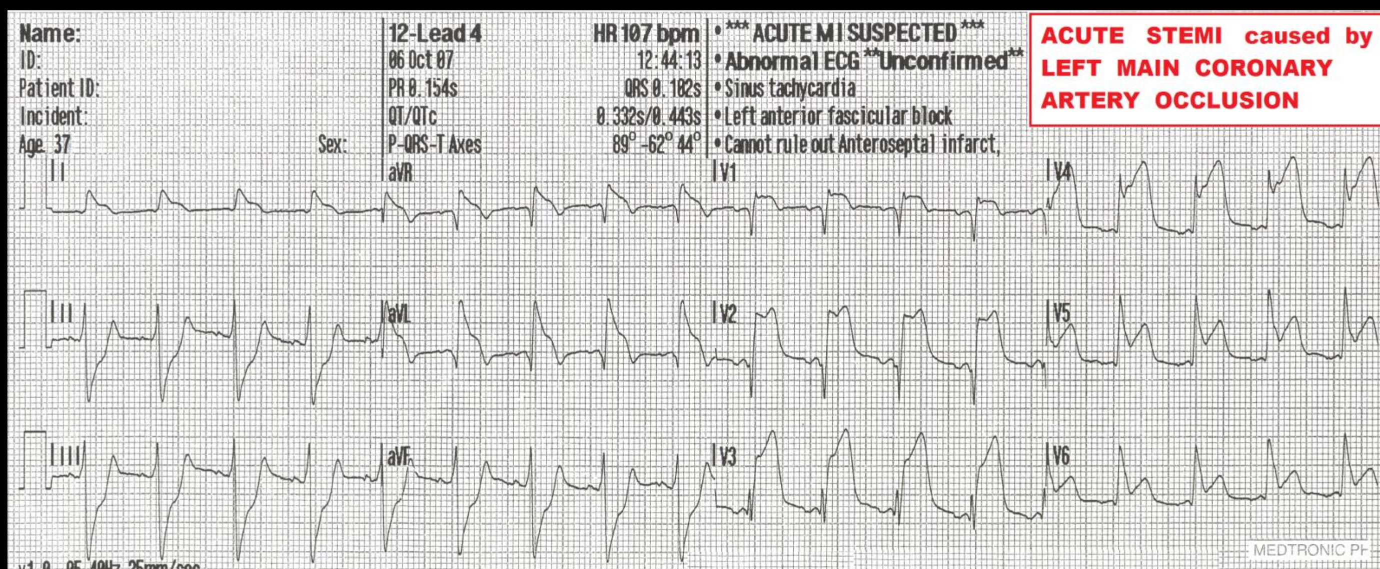
ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION



ECG CLUES of ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION:

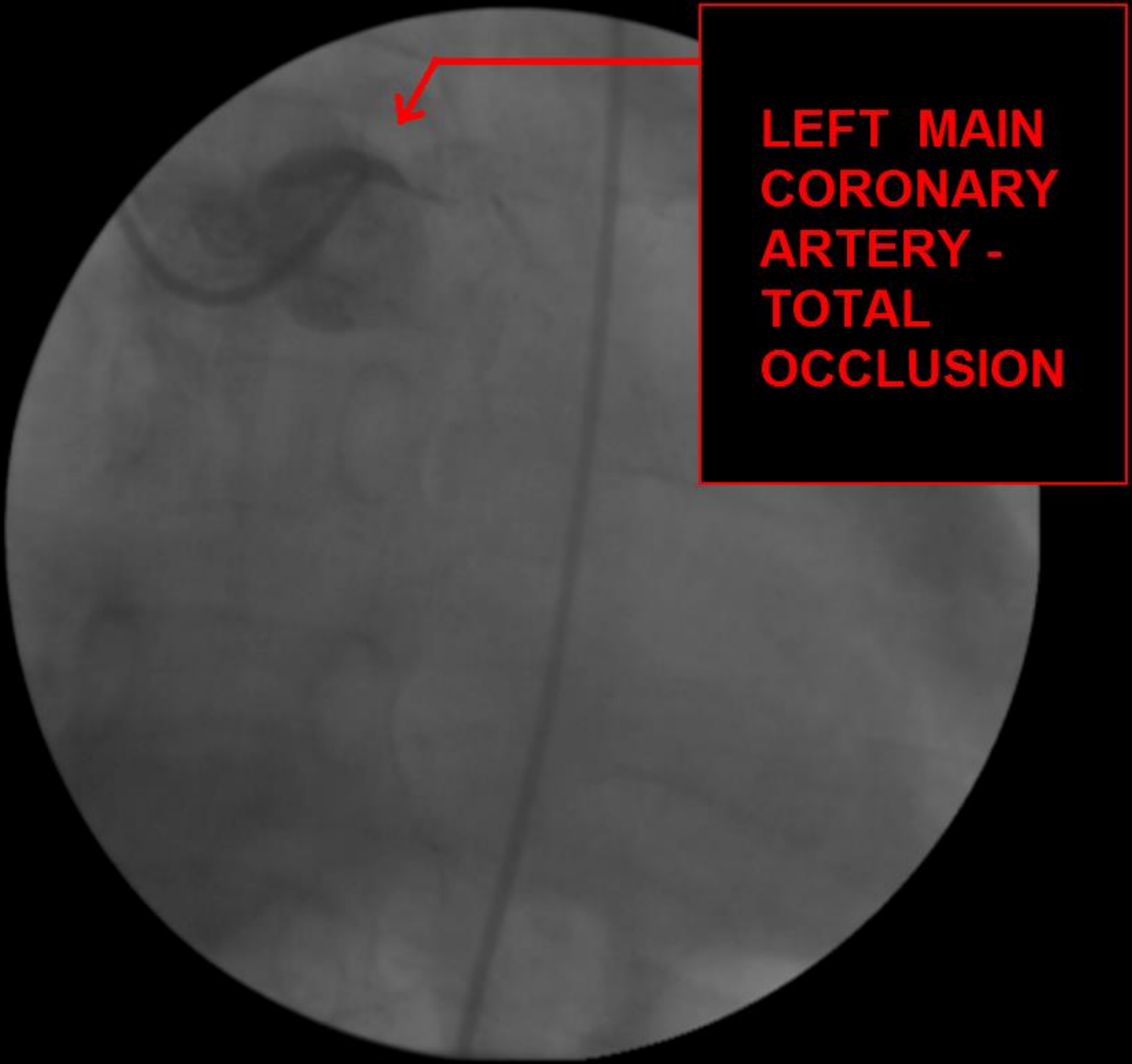
- ☑ ST ELEVATION in LEADS I, aVL, V1 - V2, V4 - V6 with ST DEPRESSION in V3: (COMPETING FORCES of ANTERIOR vs. POSTERIOR M.I.)
- ☑ RIGHT BUNDLE BRANCH BLOCK PATTERN, with
- ☑ LEFT ANTERIOR FASCICULAR BLOCK PATTERN

ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION



ECG CLUES of ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION:

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


LEFT MAIN
CORONARY
ARTERY -
TOTAL
OCCLUSION

CASE STUDY 4: CRITICAL DECISIONS SCENARIO

CONCLUSIONS:

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

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

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

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

CASE STUDY 7 - STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

46 yr. old MALE arrives in ER, C/O SUDDEN ONSET OF CHEST PRESSURE 45 MINUTES AGO. PAIN IS CONSTANT, PRESSURE-LIKE, AND NOT EFFECTED BY POSITION, MOVEMENT or DEEP INSPIRATION. ALSO C/O D.I.B.

RISK FACTOR PROFILE:

- CURRENT CIGARTTE SMOKER x 18 YEARS
- HYPERTENSION
- HIGH LDL CHOLESTEROL

PHYSICAL EXAM: Patient is alert & oriented x 4, skin warm, dry, color normal. Non-anxious Lungs clear, normal S1, S2. No JVD, No ankle edema.

VITAL SIGNS: BP: 136/88 P: 88 R: 20 SAO2: 100% on 4 LPM O2

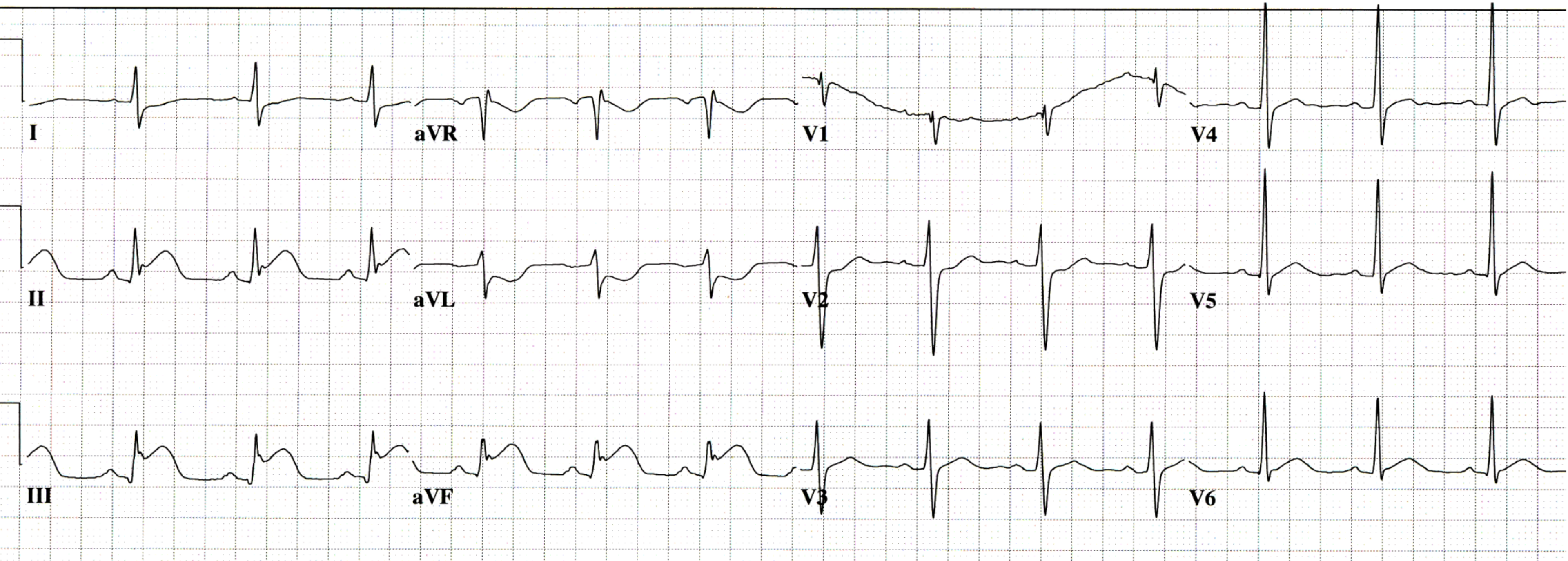
LABS: TROPONIN: < .04

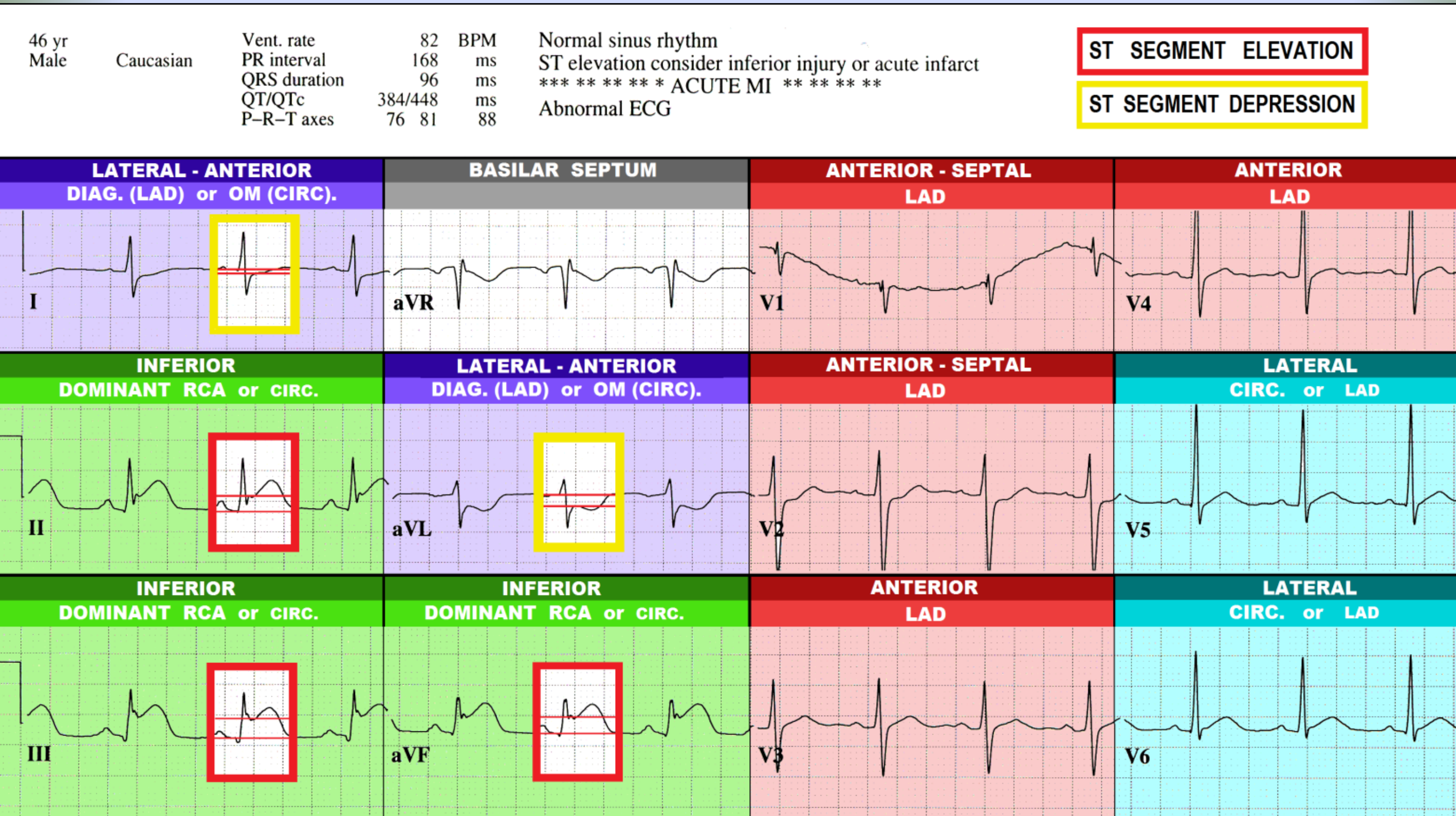
46 yr
Male Caucasian
Loc:3 Option:23

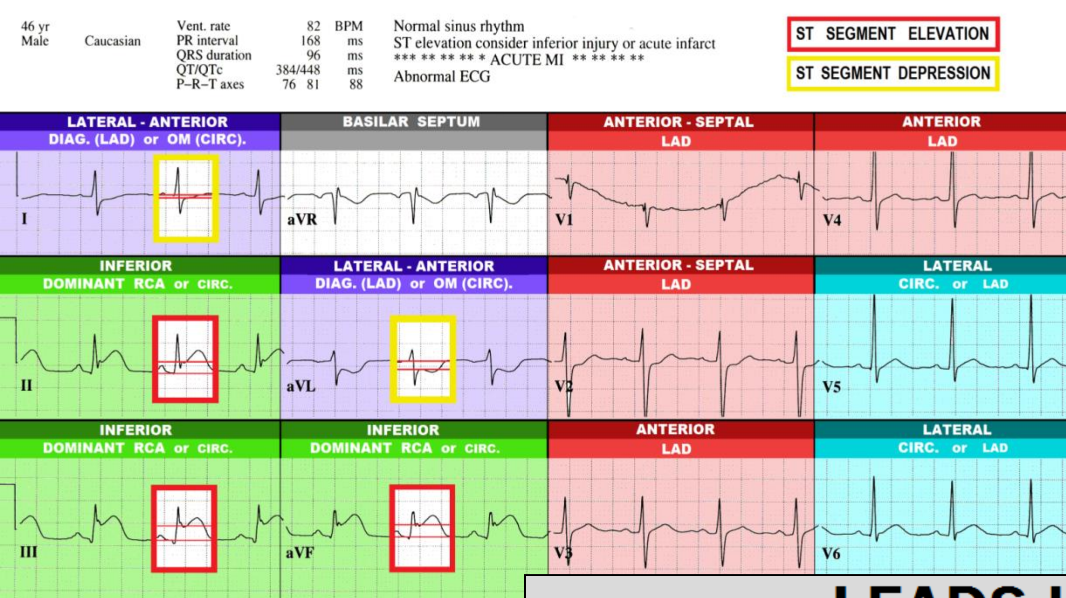
Vent. rate	82	BPM
PR interval	168	ms
QRS duration	96	ms
QT/QTc	384/448	ms
P–R–T axes	76 81	88

☞ **EVALUATE EKG for indicators of ACS:**

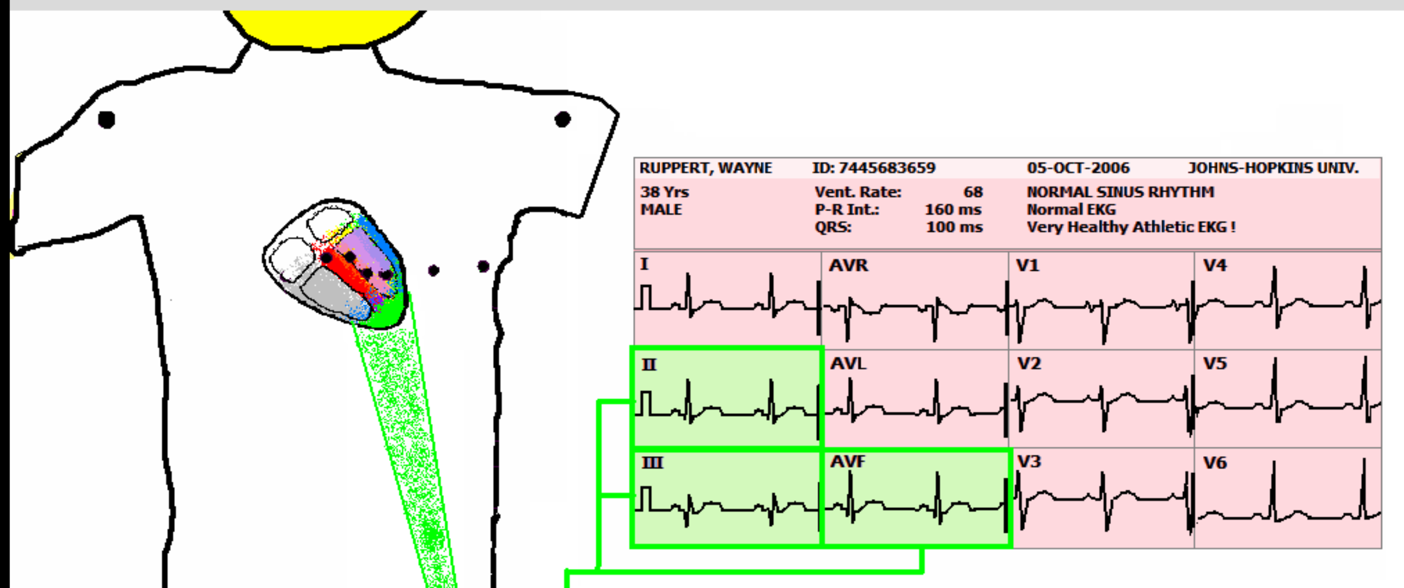
- ST SEGMENT ELEVATION / DEPRESSION
- HYPERACUTE T WAVES
- CONVEX ST SEGMENTS
- OTHER ST SEGMENT / T WAVE ABNORMALITIES




46 yr
Male
Caucasian
Vent. rate 82 BPM
PR interval 168 ms
QRS duration 96 ms
QT/QTc 384/448 ms
P–R–T axes 76 81 88
Normal sinus rhythm
ST elevation consider inferior injury or acute infarct
*** ** ** ** * ACUTE MI ** ** ** **
Abnormal ECG
ST SEGMENT ELEVATION
ST SEGMENT DEPRESSION
LATERAL - ANTERIOR
DIAG. (LAD) or OM (CIRC).
I
BASILAR SEPTUM
aVR
ANTERIOR - SEPTAL
LAD
V1
ANTERIOR
LAD
V4
INFERIOR
DOMINANT RCA or CIRC.
II
LATERAL - ANTERIOR
DIAG. (LAD) or OM (CIRC).
aVL
ANTERIOR - SEPTAL
LAD
V2
LATERAL
CIRC. or LAD
V5
INFERIOR
DOMINANT RCA or CIRC.
III
INFERIOR
DOMINANT RCA or CIRC.
aVF
ANTERIOR
LAD
V3
LATERAL
CIRC. or LAD
V6

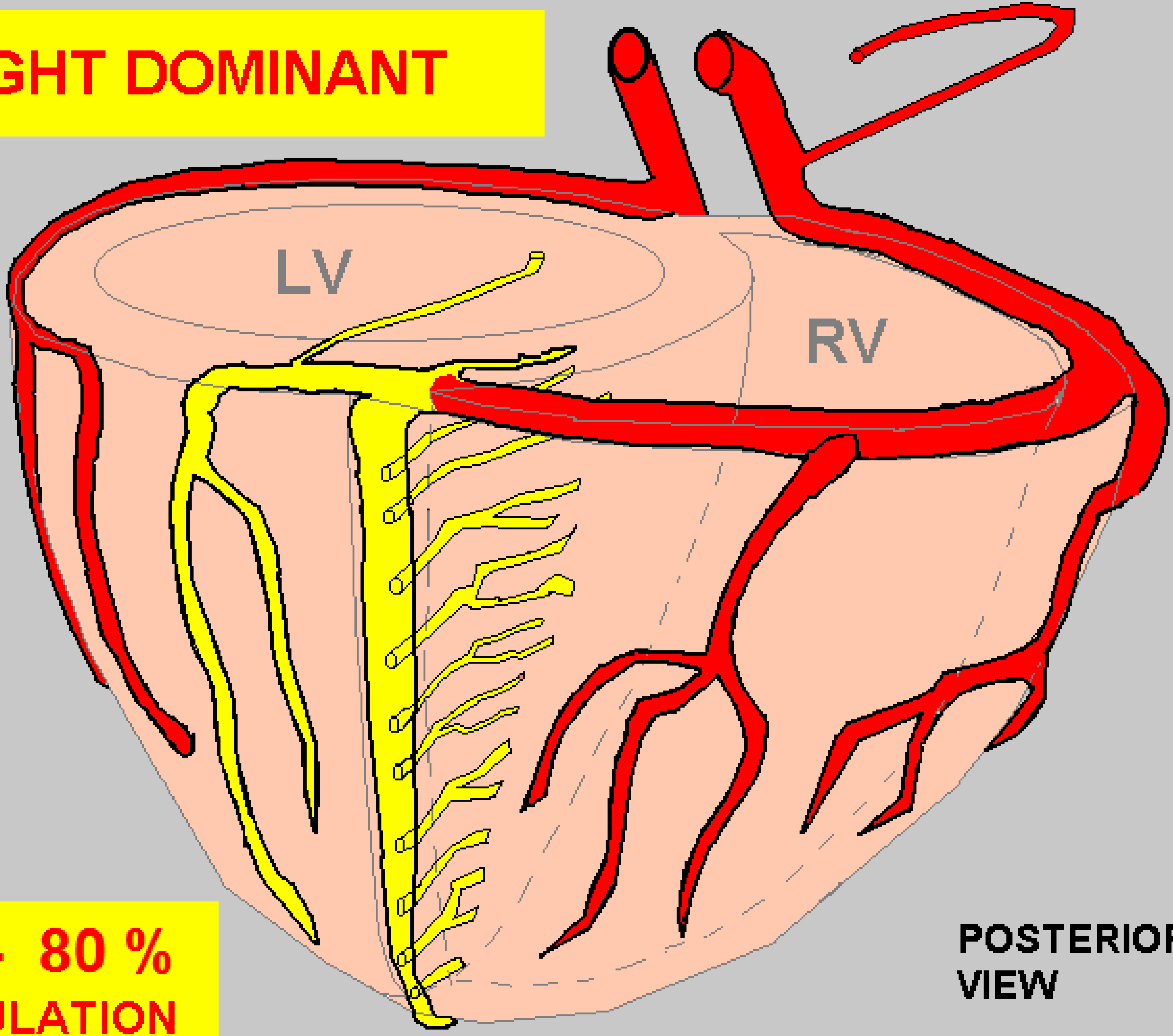


LEADS II, III, and aVF VIEW
INFERIOR WALL of the LEFT VENTRICLE



FED by the RCA (75 - 80 % pop)
or the CIRCUMFLEX (10 - 15 %)

RIGHT DOMINANT



75 - 80 %
POPULATION

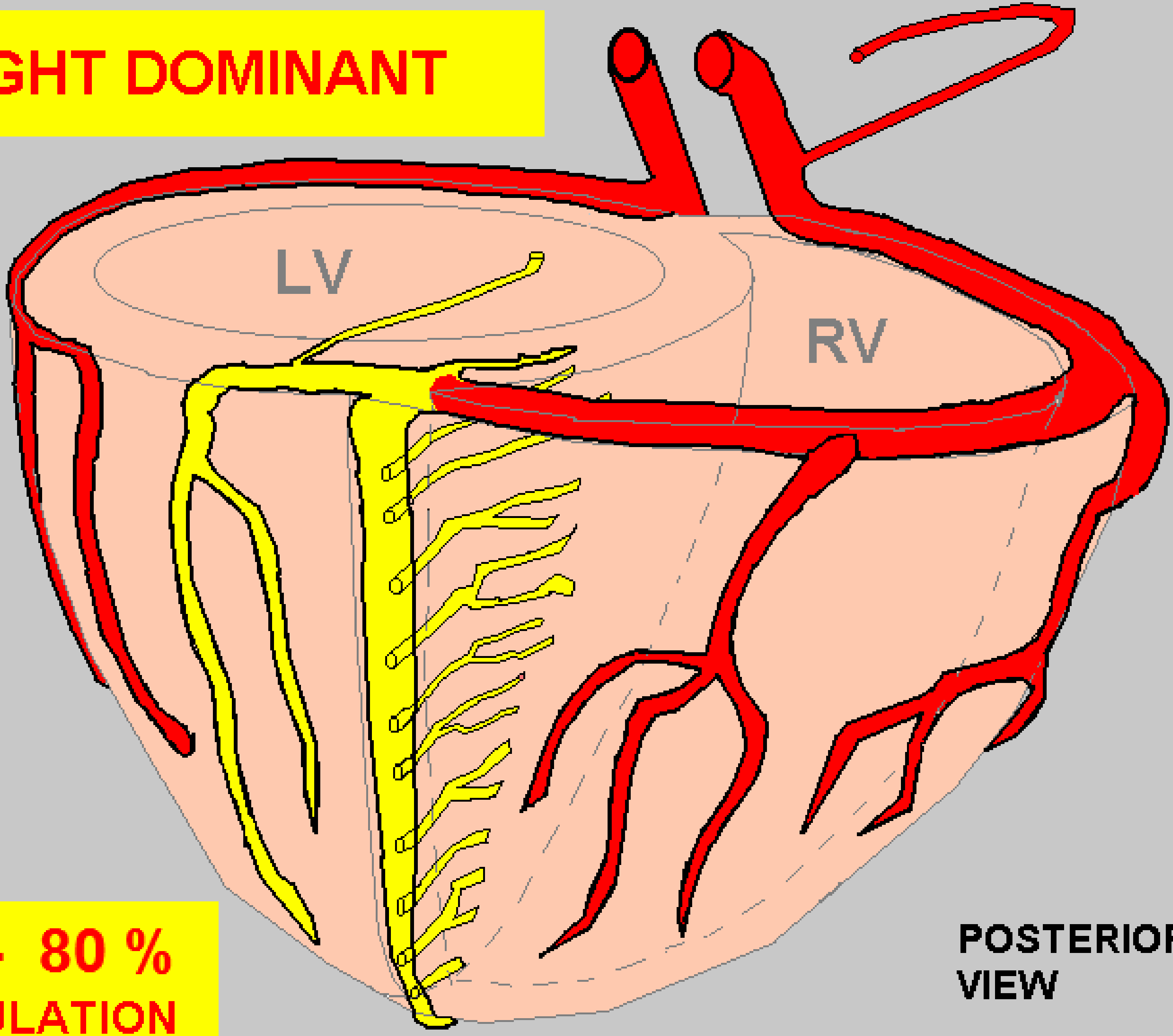
HELPFUL HINT . . . MEMORIZE THIS !

RIGHT CORONARY ARTERY (RCA)

RIGHT DOMINANT SYSTEMS

- RIGHT ATRIUM
- SINUS NODE (55% of the population)
- RIGHT VENTRICLE - 100 % of muscle mass
- LEFT VENTRICLE: 15 - 25 % of muscle mass
  - INFERIOR WALL
  - approx. 1/2 of POSTERIOR WALL
- AV NODE

RIGHT DOMINANT



75 - 80 %
POPULATION

A standard

12 LEAD EKG

Does NOT show the

RIGHT VENTRICLE

To see the

RIGHT VENTRICLE . . .

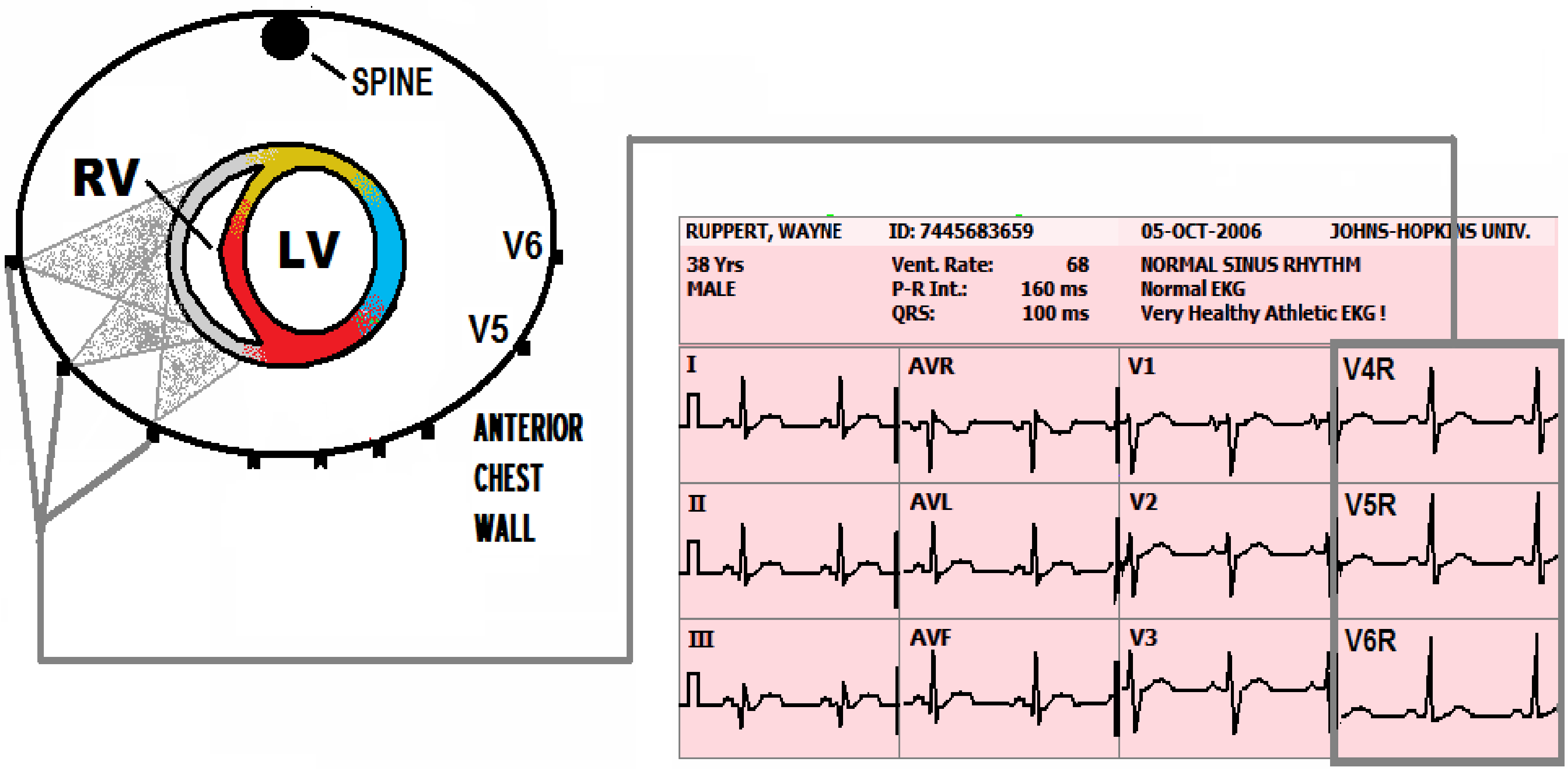
. . . such as in cases of

INFERIOR WALL M.I.

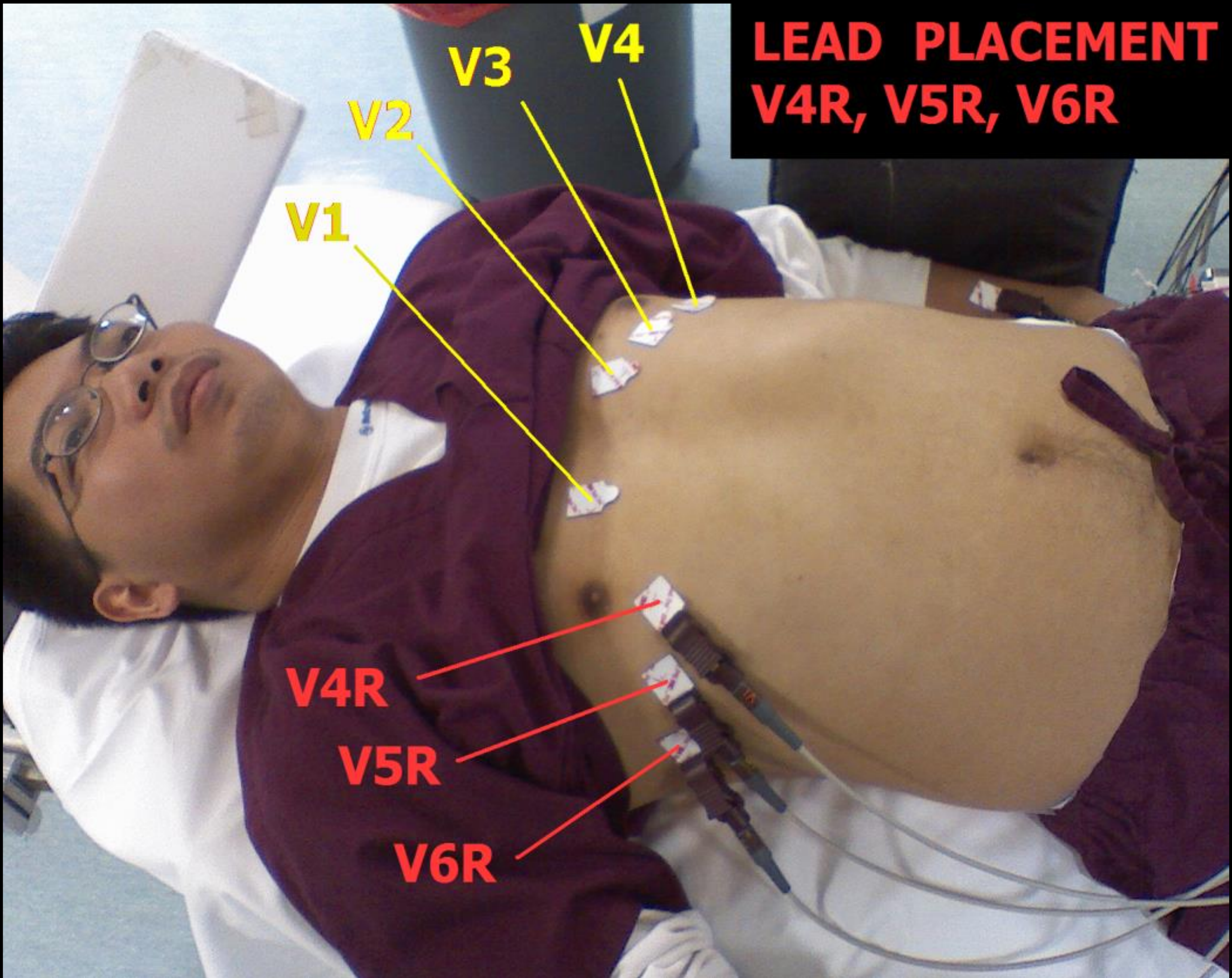
☞ You must do a

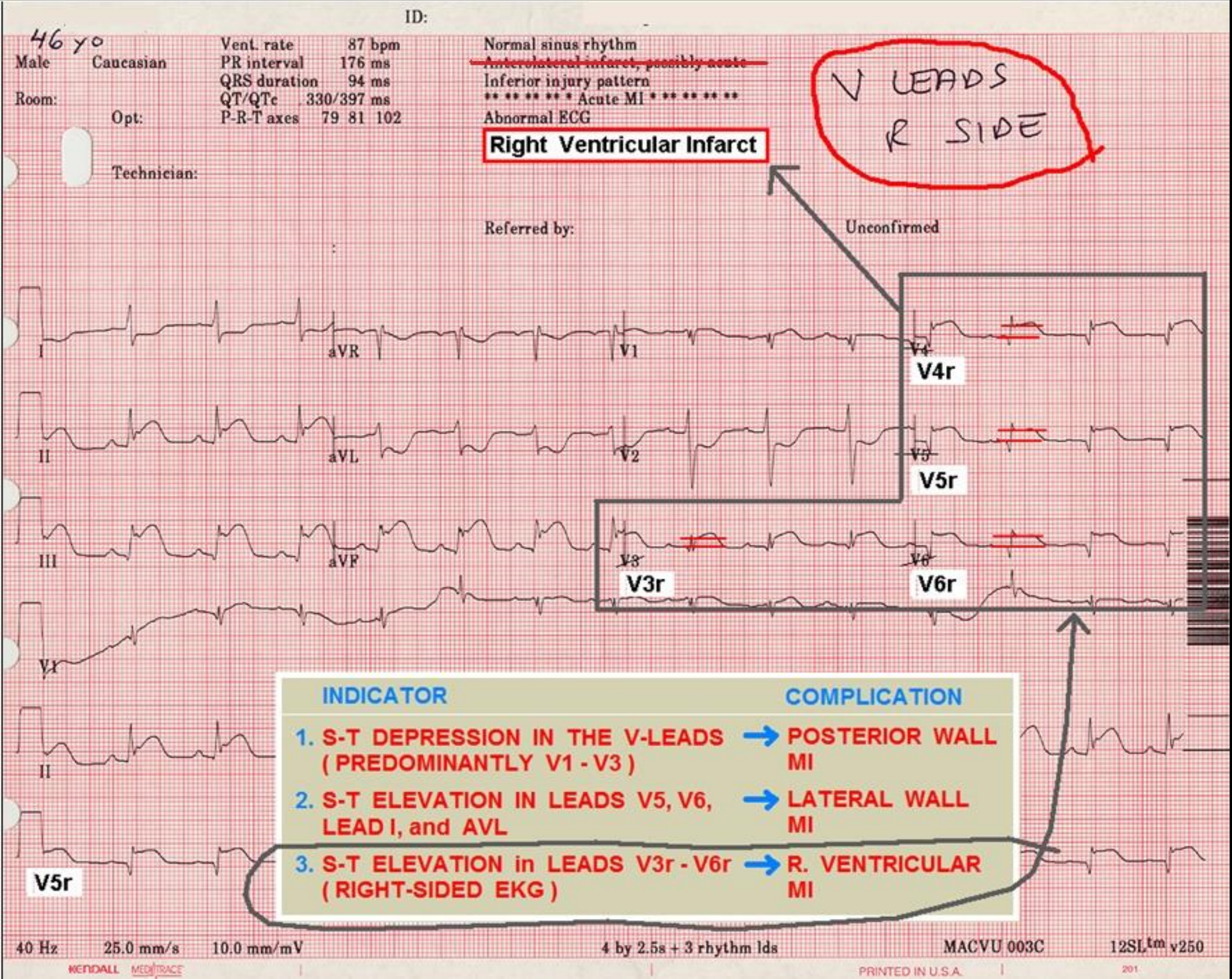
RIGHT - SIDED EKG ! !

V4R - V6R VIEW THE RIGHT VENTRICLE

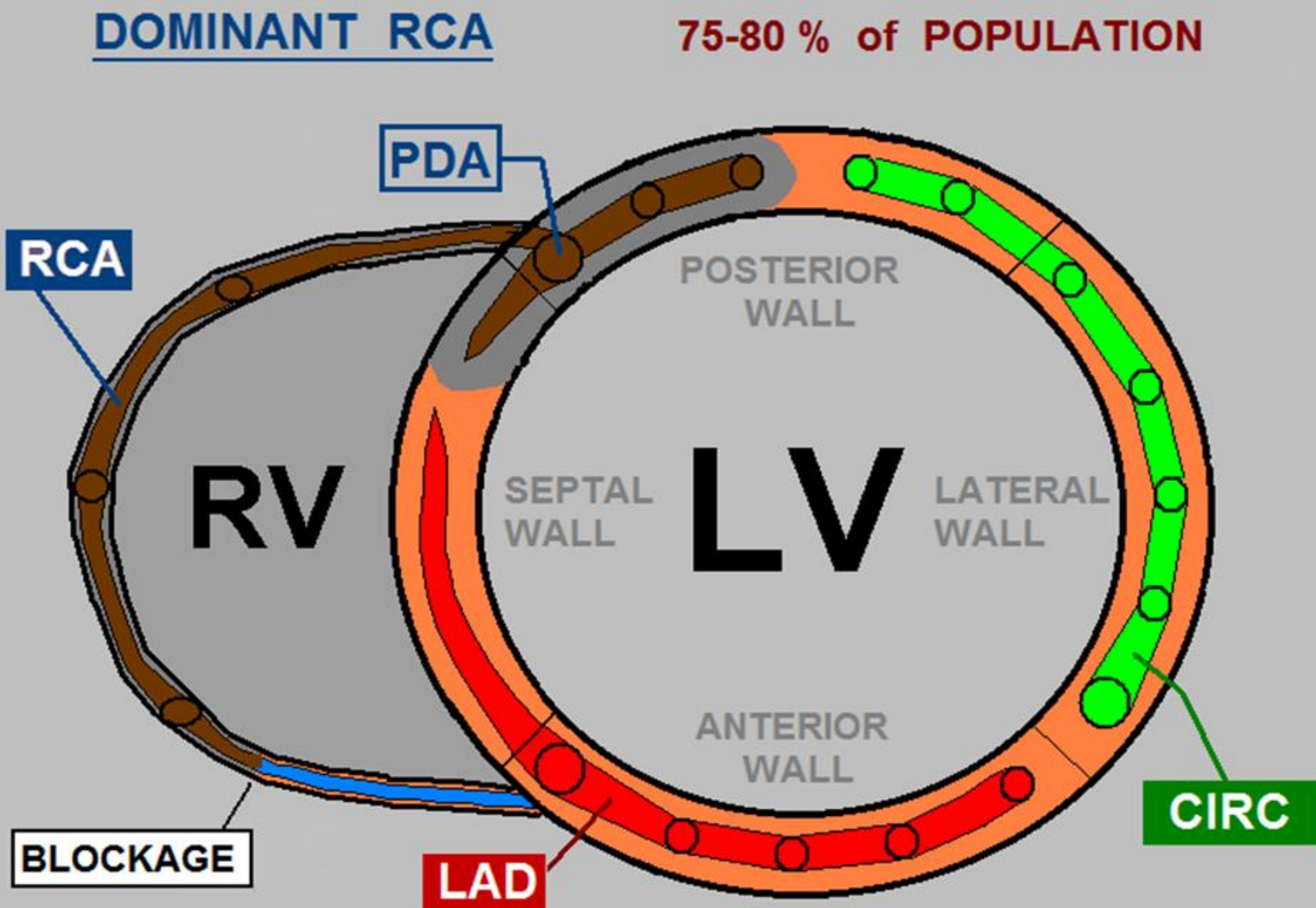


LEAD PLACEMENT V4R, V5R, V6R




ID:
46 yo
Male Caucasian
Room:
Opt:
Technician:
Vent. rate 87 bpm
PR interval 176 ms
QRS duration 94 ms
QT/QTc 330/397 ms
P-R-T axes 79 81 102
Normal sinus rhythm
~~Anterolateral infarct, possibly acute~~
Inferior injury pattern
** ** ** ** * Acute MI * ** ** ** **
Abnormal ECG
Right Ventricular Infarct
V LEADS R SIDE
Referred by:
Unconfirmed
I
aVR
V1
V4r
II
aVL
V2
V5r
III
aVF
V3r
V6r
V1
II
V5r
INDICATOR
COMPLICATION
1. S-T DEPRESSION IN THE V-LEADS (PREDOMINANTLY V1 - V3) → POSTERIOR WALL MI
2. S-T ELEVATION IN LEADS V5, V6, LEAD I, and AVL → LATERAL WALL MI
3. S-T ELEVATION in LEADS V3r - V6r (RIGHT-SIDED EKG) → R. VENTRICULAR MI
40 Hz
25.0 mm/s
10.0 mm/mV
4 by 2.5s + 3 rhythm lds
MACVU 003C
12SLtm v250
KENDALL MEDITRACE
PRINTED IN U.S.A.

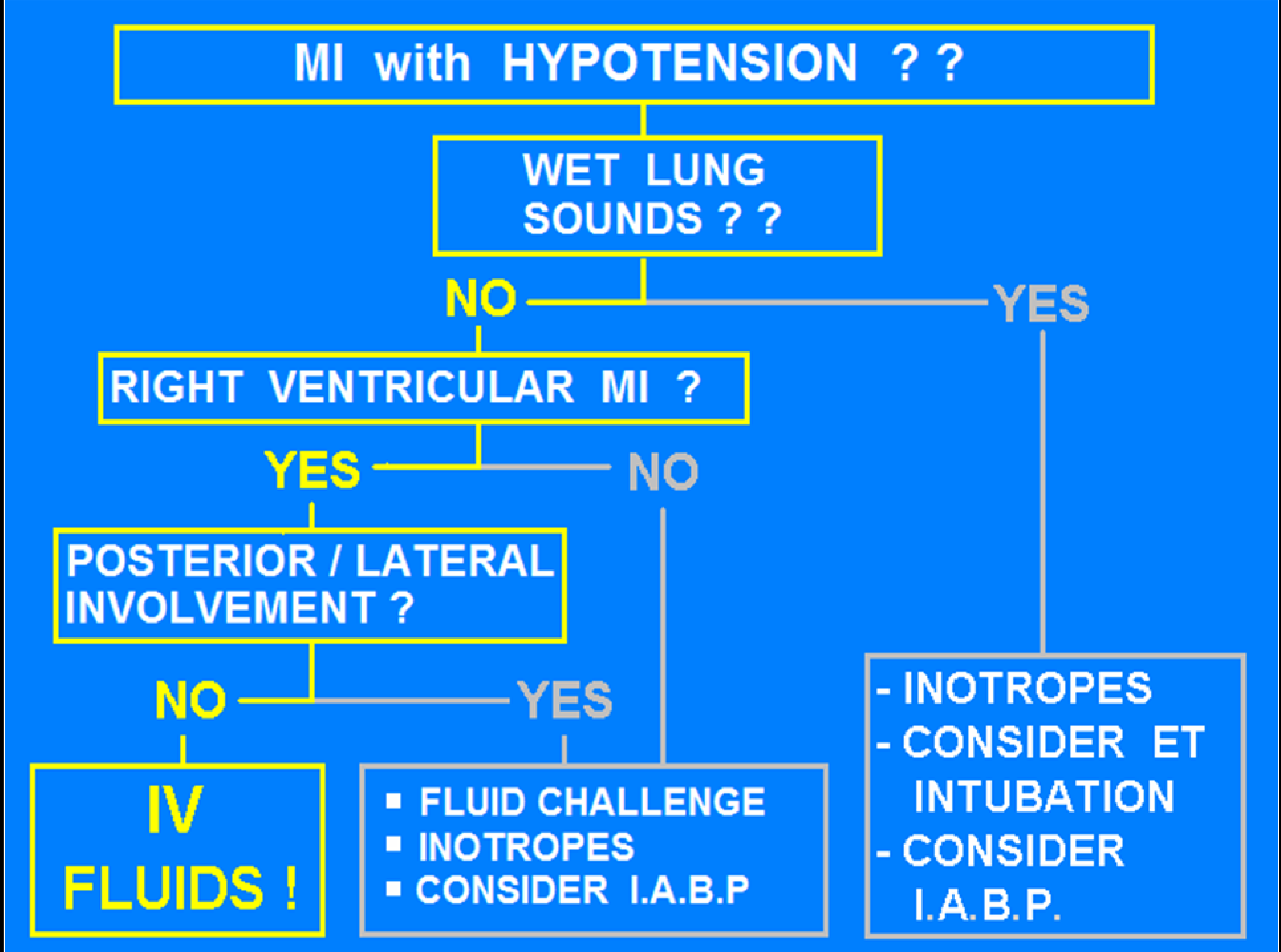
INFERIOR - RIGHT VENTRICULAR MI

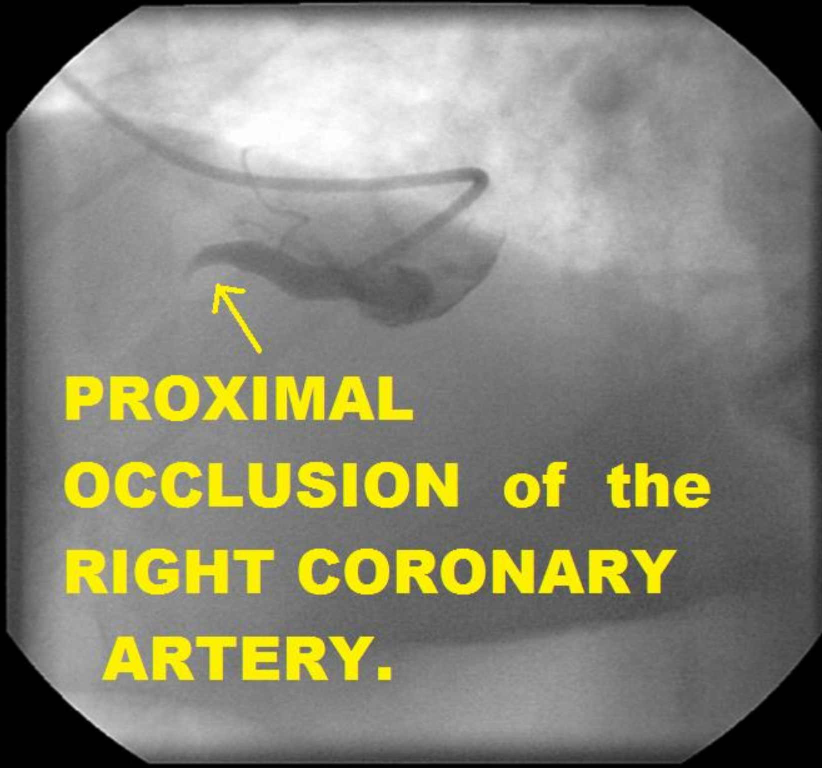


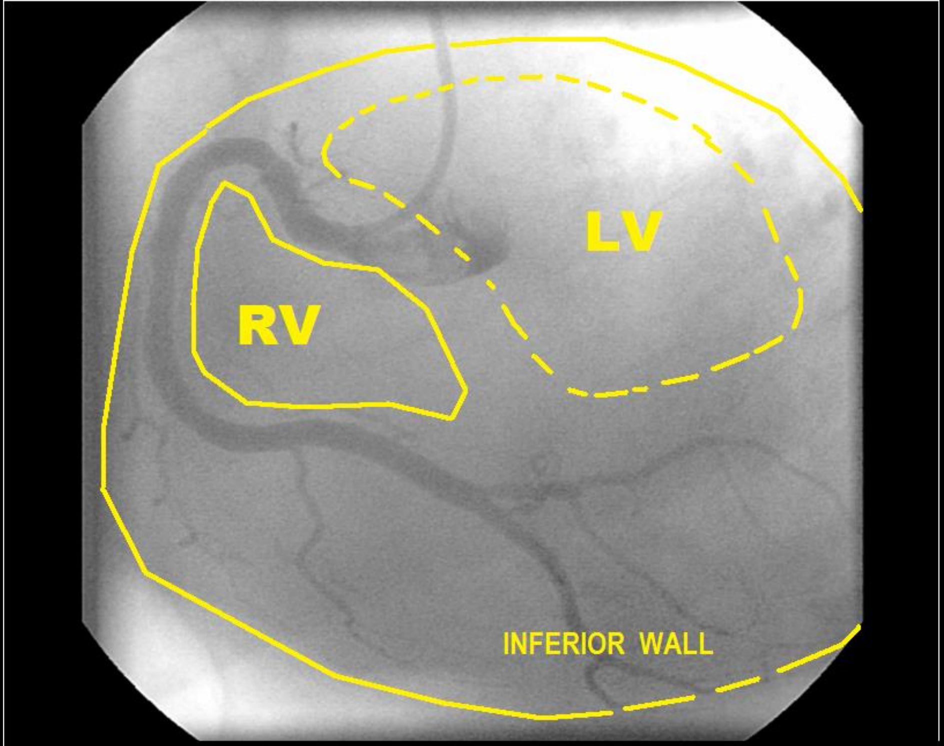
ANTICIPATED COMPLICATIONS of INFERIOR WALL STEMI secondary to
RCA Occlusion & POSSIBLE INDICATED INTERVENTIONS:

- CARDIAC ARREST	BCLS / ACLS
- CARDIAC DYSRHYTHMIAS (VT / VF)	ACLS (antiarrhythmics)
- SINUS BRADYCARDIA	ATROPINE 0.5mg, REPEAT as needed UP TO 3mg. (follow ACLS and/or UNIT protocols)
- HEART BLOCKS (1st, 2nd & 3rd Degree HB)	ATROPINE 0.5mg, REPEAT as needed UP TO 3mg, Transcutaneous Pacing, (follow ACLS and/or UNIT protocols)
- RIGHT VENTRICULAR MYOCARDIAL INFARCTION	- The standard 12 Lead ECG does NOT view the Right Ventricle. - You must do a RIGHT-SIDED ECG to see if RV MI is present. - Do NOT give any Inferior Wall STEMI patient NITRATES or DIURETICS until RV MI has been RULED OUT.

If this patient becomes HYPOTENSIVE . . . . .


MI with HYPOTENSION ? ?
WET LUNG SOUNDS ? ?
NO
YES
RIGHT VENTRICULAR MI ?
YES
NO
POSTERIOR / LATERAL INVOLVEMENT ?
NO
YES
IV FLUIDS !
▪ FLUID CHALLENGE
▪ INOTROPES
▪ CONSIDER I.A.B.P
- INOTROPES
- CONSIDER ET INTUBATION
- CONSIDER I.A.B.P.





POST PTCA / STENT DEPLOYMENT TO PROXIMAL RCA

IN ***EVERY*** CASE of

INFERIOR WALL STEMI

You must first *RULE OUT*

RIGHT VENTRICULAR MI

BEFORE giving any:

- NITROGLYCERIN
- Diuretics

Nitroglycerin & Diuretics
are
CLASS III CONTRINDICATED
in
RIGHT VENTRICULAR MI ! !*

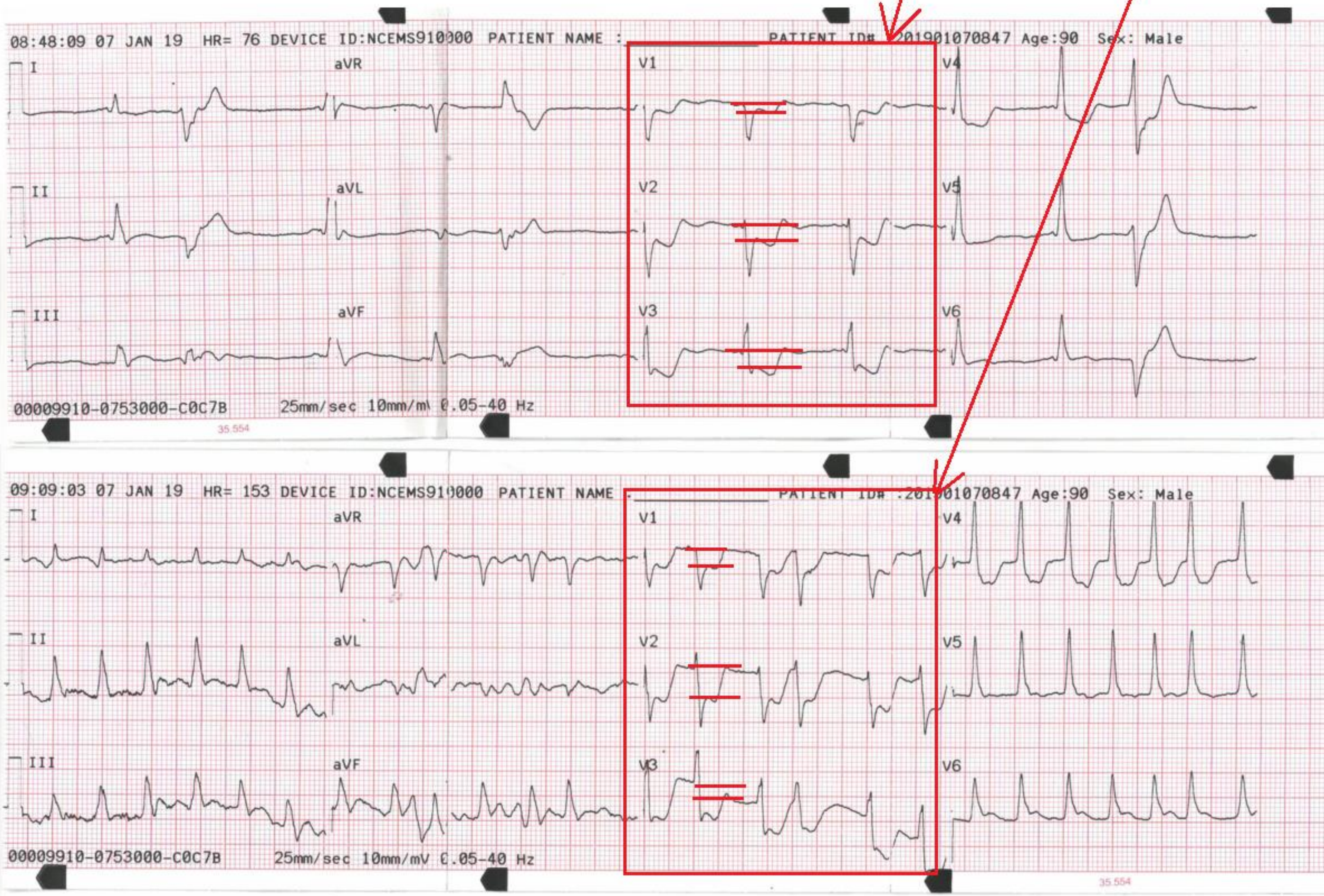
They precipitate SEVERE HYPOTENSION

*** A.H.A. ACLS 2010 / 2015**

Case Study- January 2019

- 79 y/o female complaining of “L arm pain, and minimal chest pain”
- EMS 12 Lead ECGs show ST Depression in Anterior Leads V1-V4. There is NO ST Elevation……

Two EMS 12 Lead ECGs: none show ST Elevation, but both show significant ST depression in Anterior Leads V1-V3.



Initial Exam in ED

- Upon arrival in ED, 12 Lead ECG confirmed EMS findings: ST Depression in Leads V1-V4.

Pat ID [redacted] 01/07/2019 09:19:35 [redacted] 79 yrs [redacted] Caucasian Female Account # [redacted]

Bayfront Health Seven Rivers ED

Dept	ED
Room	ED01
Tech	gp

RX
DX

Rate	153
PR	
QRSd	117
QT	260
QTc	415
--Axis--	
P	
QRS	73
T	78

```
Atrial fibrillation with rapid V-rate
Nonspecific intraventricular conduction delay
NO PREVIOUS ECG AVAILABLE FOR COMPARISON
```

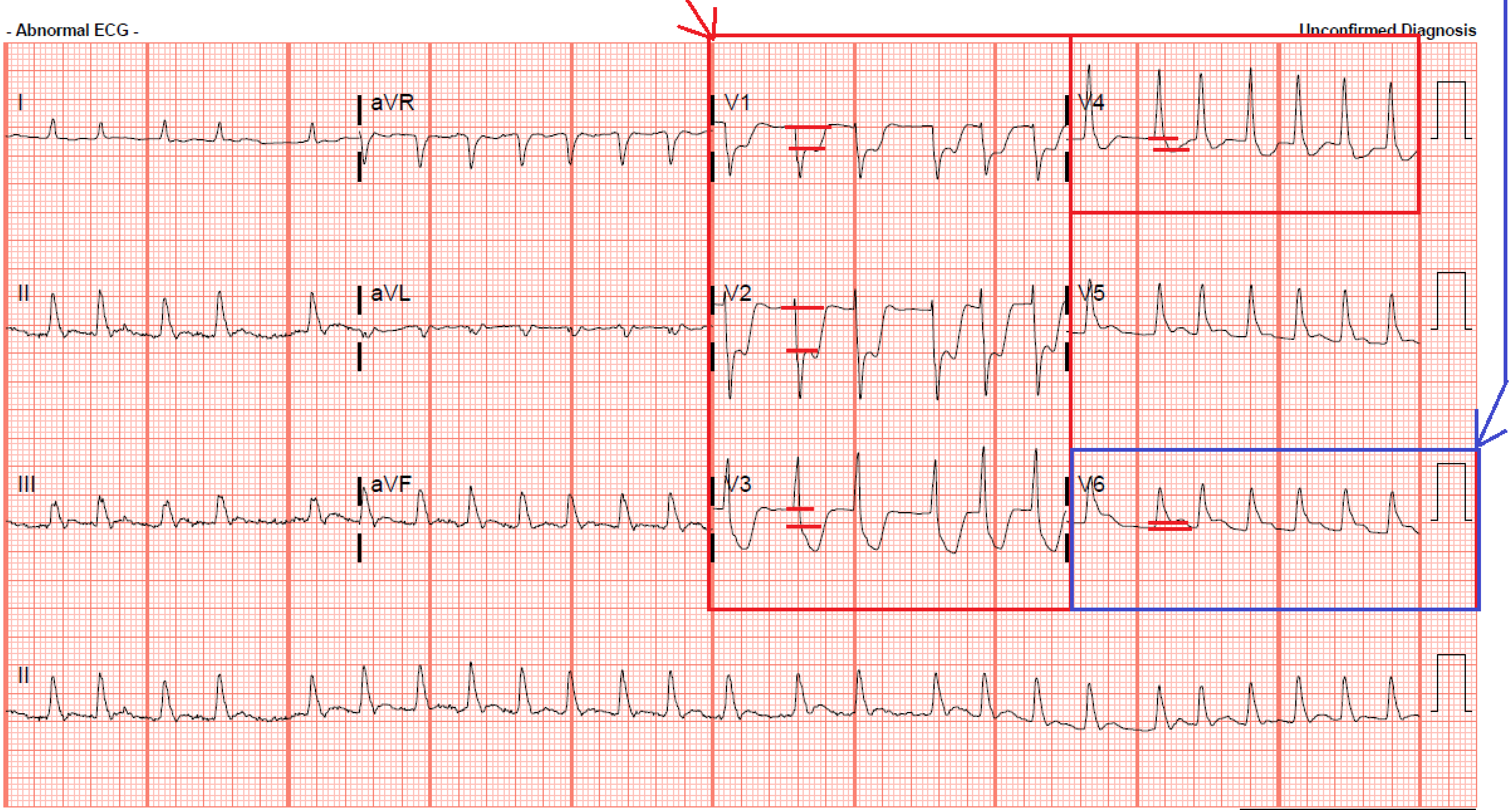
Req Provider:

ST Depression Leads V1 - V4

Minimal ST Elevation in Lead V6. (Does not meet STEMI Criteria)

- Abnormal ECG -

Unconfirmed Diagnosis



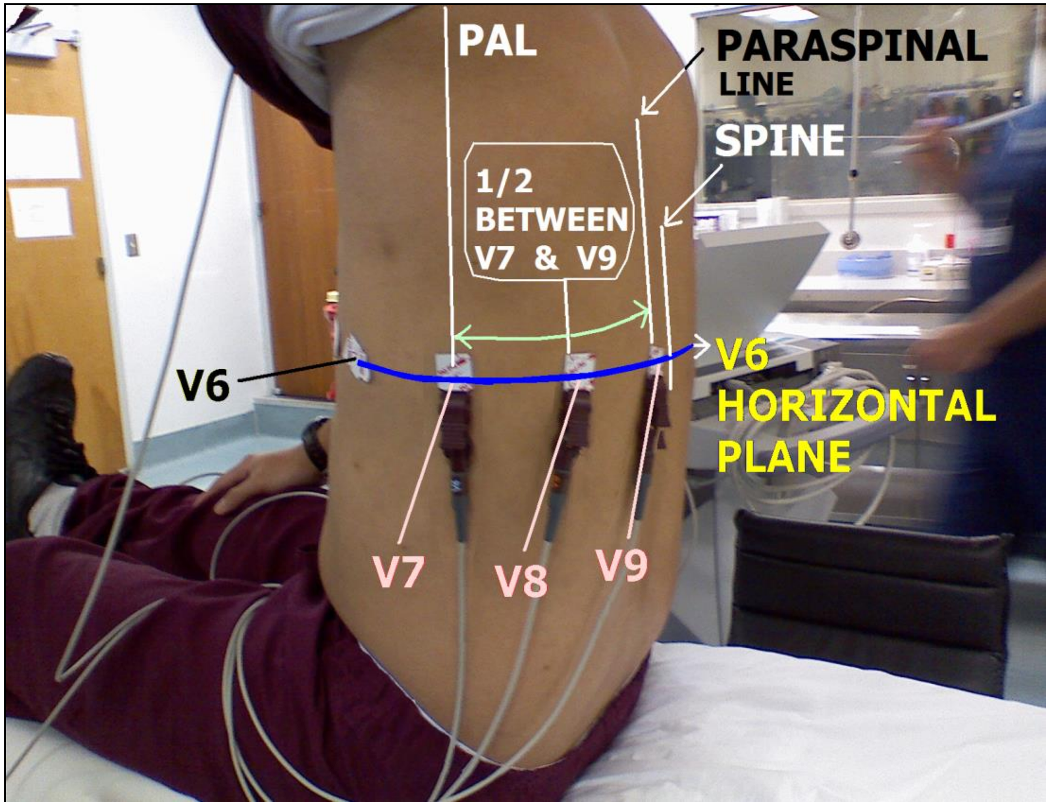
Philips PageWriter TC 25 mm/sec 10.00 mm/mV F 60~ 0.15 - 100 Hz

Causes of ST Depression V1-V4

- Anterior Wall ischemia
- Anterior Wall NSTEMI (partial wall thickness myocardial infarction)
- Posterior Wall STEMI

Continued Exam in the ED….

- Upon noting ST Depression in Anterior Leads, 3 leads were placed on the patient's back. The lead wires for V4, V5 and V6, were repositioned, as shown here:
- The "Posterior Lead ECG" is seen on the next slide……



Pat ID [redacted]
RX
DX

01/07/2019 09:23:29
[redacted] 79 yrs

[redacted]
Caucasian Female
Account # [redacted]

Bayfront Health Seven Rivers ED
Dept EDHD
Room EDH
Tech gp

Rate	133
PR	
QRSd	114
QT	337
QTc	502
--Axis--	
P	
QRS	77
T	121

Atrial fibrillation
~~Anterolateral infarct, acute~~
Prolonged QT interval
COMPARED TO ECG 01/07/2019 09:21:04
PROLONGED QT INTERVAL NOW PRESENT

**** Posterior Infarct - Acute ****

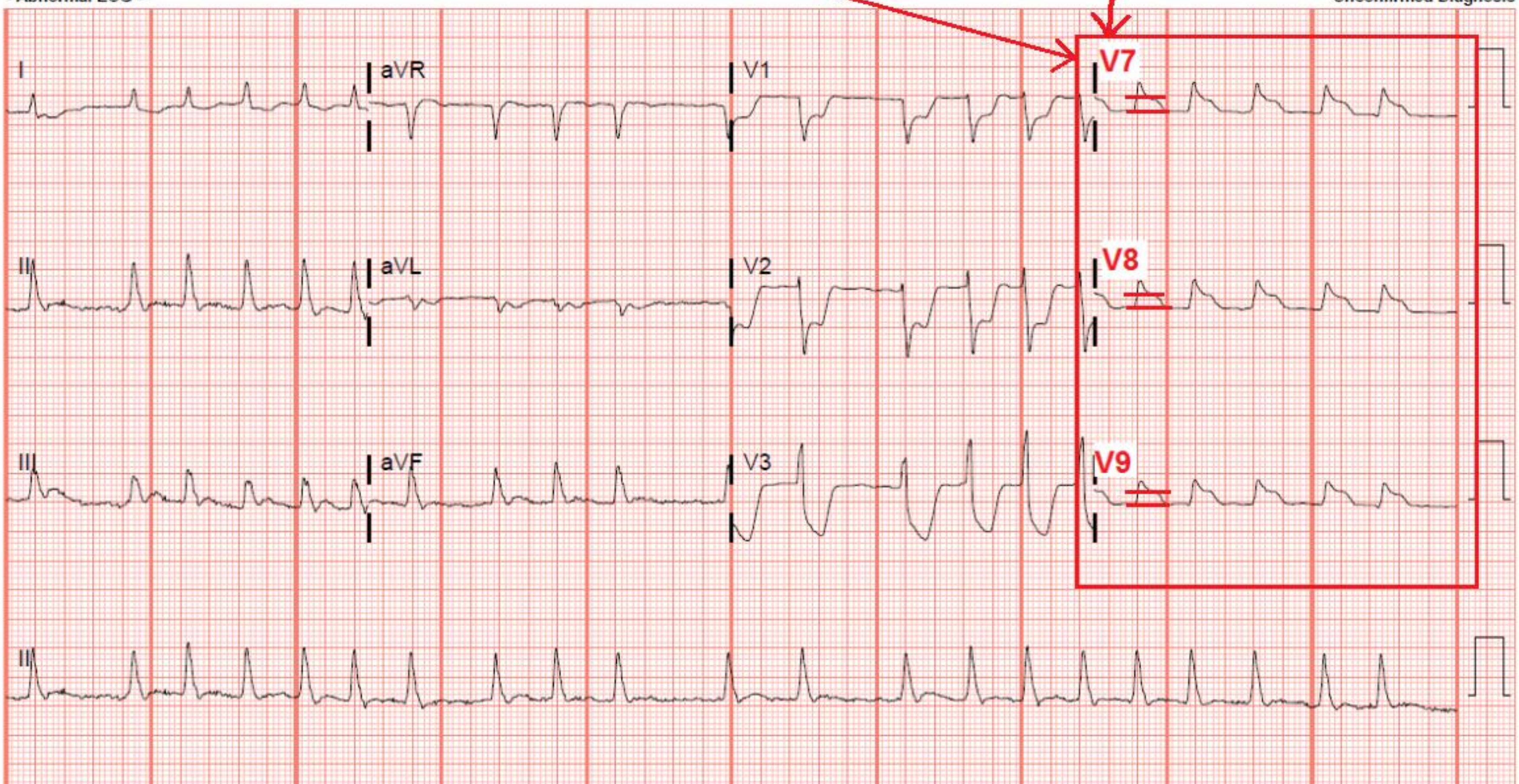
ACUTE POSTERIOR WALL STEMI

Req Provider: ONIER VILLARREAL

Chest leads V4-V6 repositioned to patient's back (Posterior Leads V7, V8 and V9) reveal ST Segement Elevation. Patient diagnosis changes from "possible NSTEMI" to "Acute STEMI."

- Abnormal ECG -

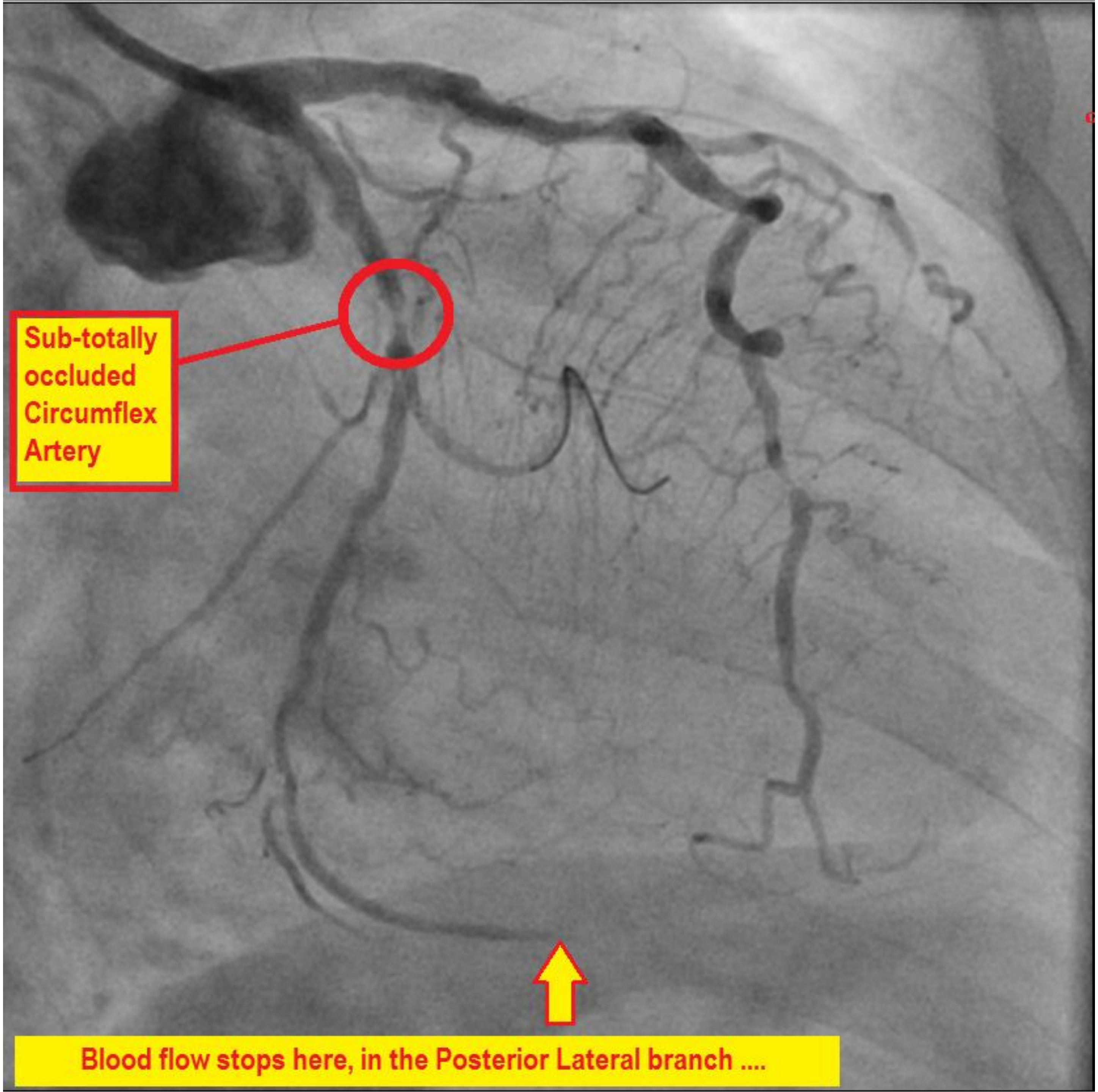
Unconfirmed Diagnosis

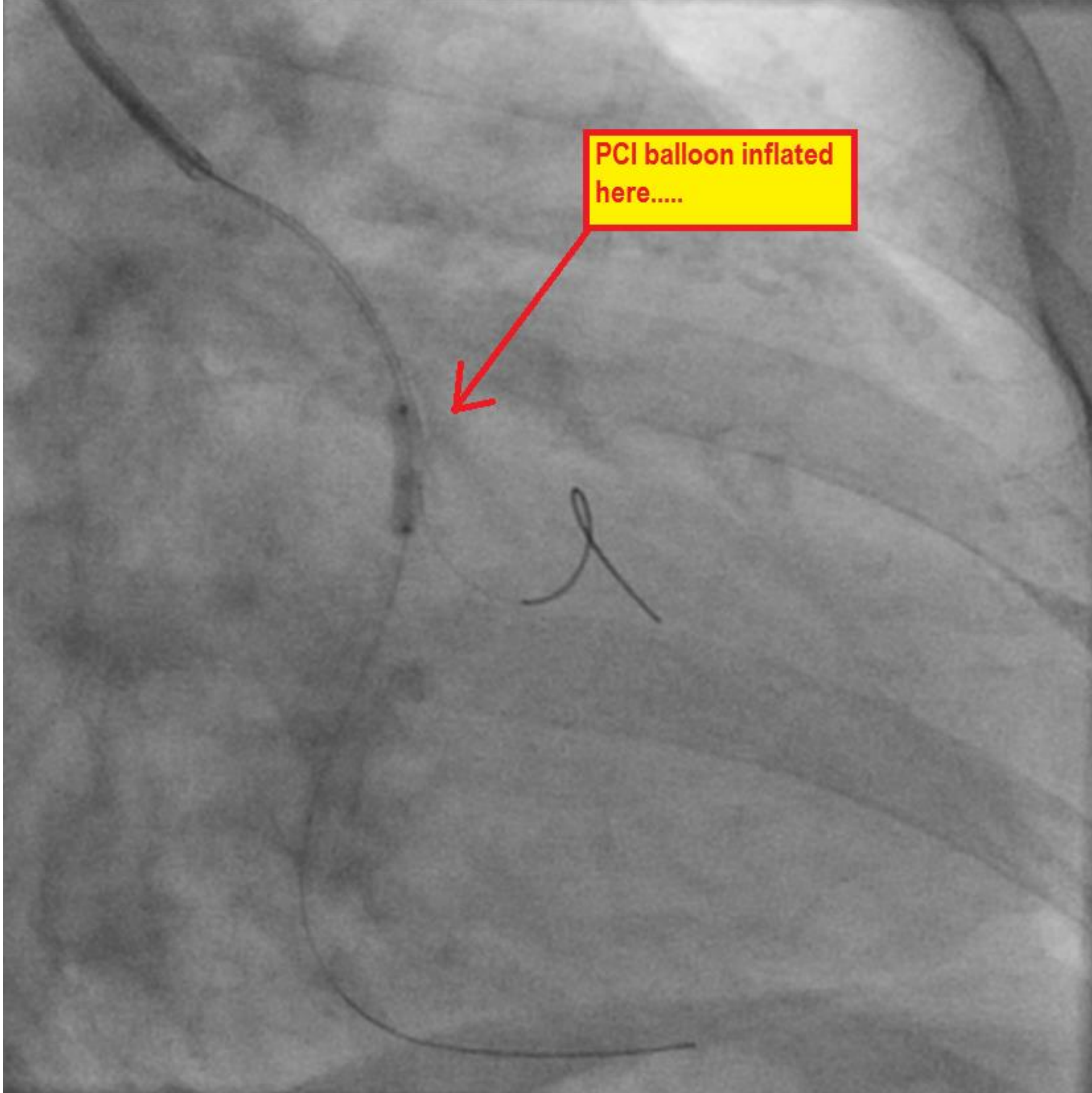


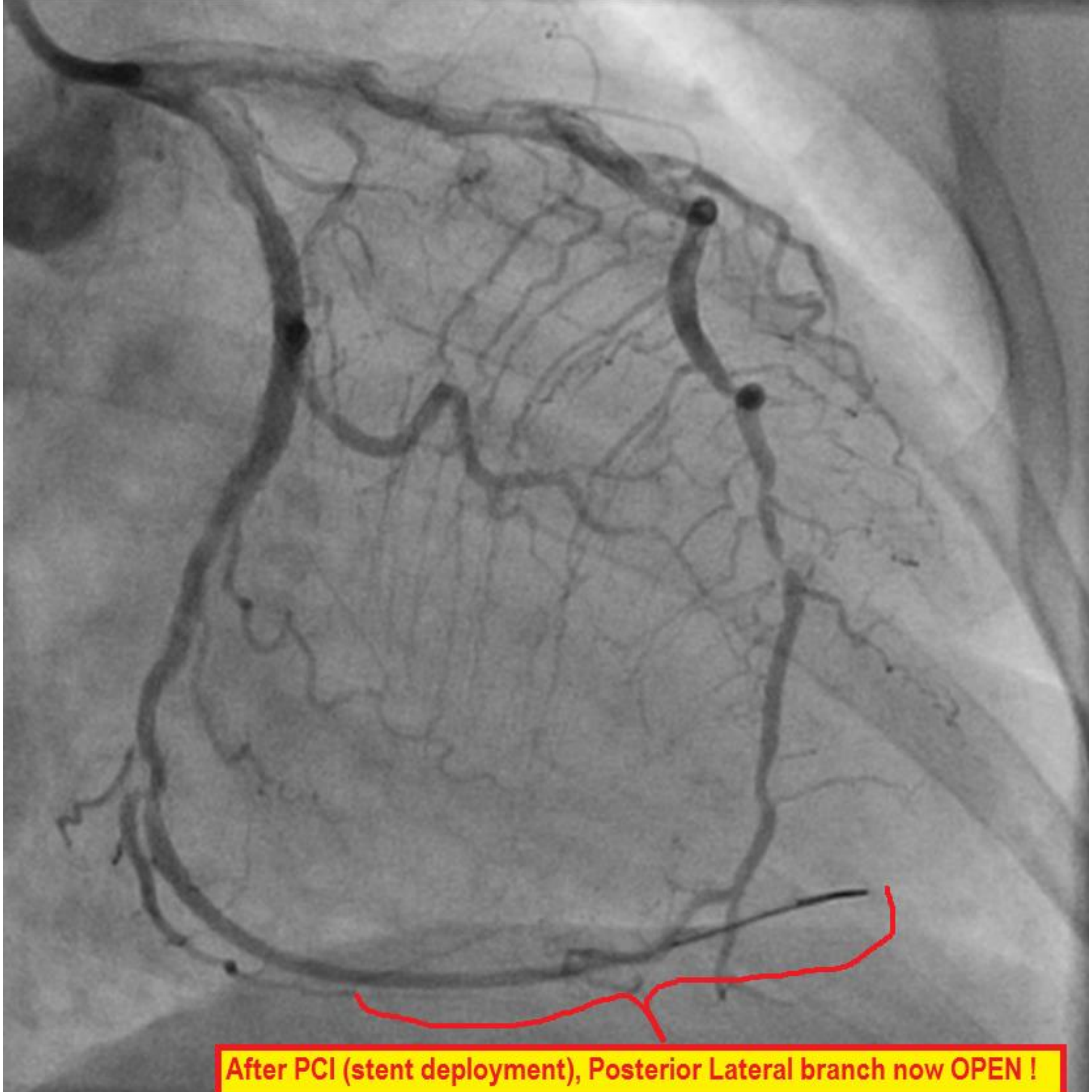
Philips PageWriter TC 25 mm/sec 10.00 mm/mV F 60~ 0.15 - 100 Hz

STEMI Alert !

Upon seeing “Significant ST Elevation in TWO or more CONTIGUOUS LEADS, the ED physician diagnosed “Posterior Wall STEMI,” a STEMI Alert was issued, and the patient was taken immediately to the cardiac cath lab, where the following images were obtained…….


Sub-totally occluded Circumflex Artery
Blood flow stops here, in the Posterior Lateral branch ....


PCI balloon inflated
here.....


After PCI (stent deployment), Posterior Lateral branch now OPEN !

SUMMARY

- Whenever ST Depression is noted in Anterior Leads (V1-V4), it could indicate that Acute Posterior Wall STEMI is present.

SUMMARY

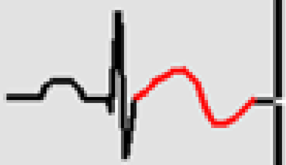
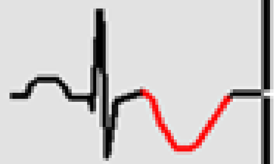
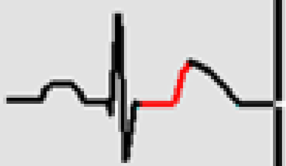
- Whenever ST Depression is noted in Anterior Leads (V1-V4), it could indicate that Acute Posterior Wall STEMI is present.
- To rule-out Posterior Wall STEMI, a "posterior lead ECG" (V7 – V9) must be obtained.

SUMMARY

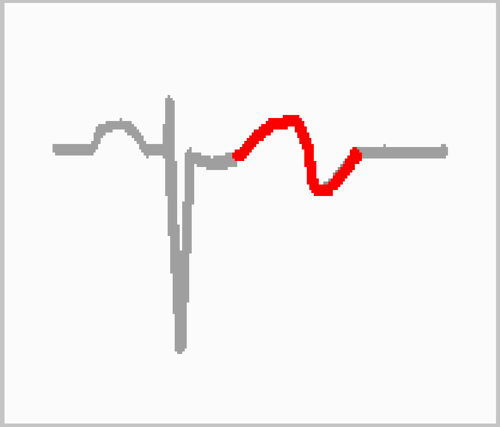
- Whenever ST Depression is noted in Anterior Leads (V1-V4), it could indicate that Acute Posterior Wall STEMI is present.
- To rule-out Posterior Wall STEMI, a "posterior lead ECG" (V7 – V9) must be obtained.
- In THIS CASE, **Posterior Wall STEMI** was diagnosed via Posterior Lead ECG.

SUMMARY

- Whenever ST Depression is noted in Anterior Leads (V1-V4), it could indicate that Acute Posterior Wall STEMI is present.
- To rule-out Posterior Wall STEMI, a "posterior lead ECG" (V7 – V9) must be obtained.
- In THIS CASE, Posterior Wall STEMI was diagnosed via Posterior Lead ECG.
- **STEMI Alert was issued, with a Door-to-PCI time of 53 minutes.**

BI-PHASIC T WAVE (WELLEN'S)		- SUB-TOTAL LAD LESION - VASOSPASM - HYPERTROPHY
INVERTED T WAVE		- MYOCARDITIS - ELECTROLYTE IMBAL. - ISCHEMIA
SHARP S-T T ANGLE		- ACUTE MI (NOT COMMON) - ISCHEMIA
DEPRESSED J POINT with UPSLOPING ST		- ISCHEMIA
DOWNSLOPING S-T SEGMENT		- ISCHEMIA

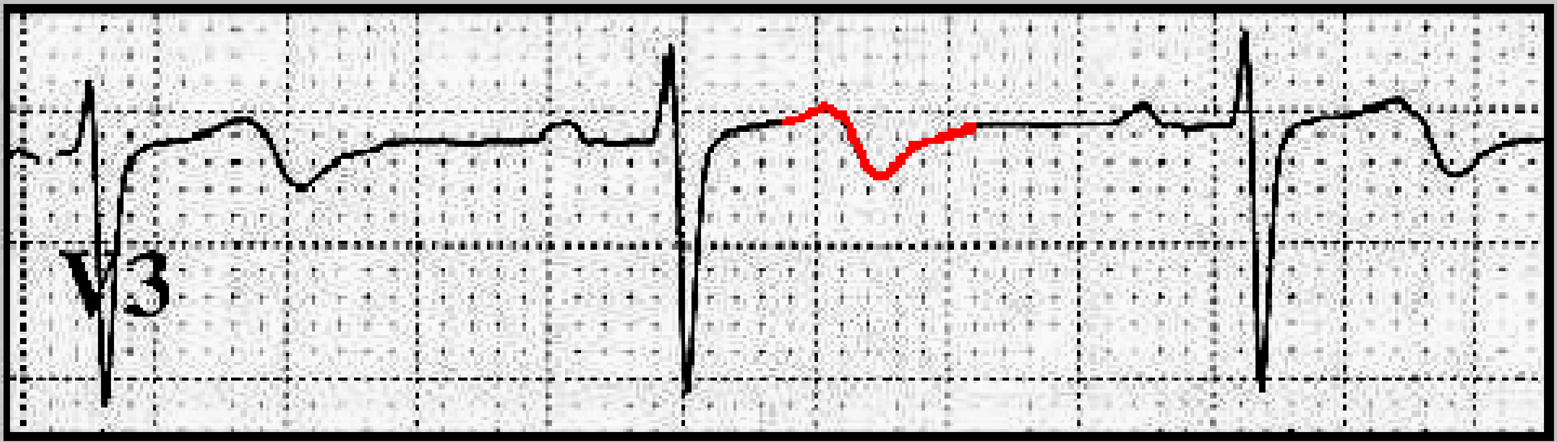
ISCHEMIA



BI-PHASIC T WAVE

- SUB-TOTAL OCCLUSION of LEFT ANTERIOR DESCENDING ARTERY (when noted in V1-V4)
- LEFT VENTRICULAR HYPERTROPHY
- COCAINE INDUCED VASOSPASM

BI-PHASIC T WAVES



58 y/o MALE WITH SUB-TOTAL OCCLUSIONS OF THE LEFT ANTERIOR DESCENDING ARTERY

58 y/o MALE WITH "WELLEN'S WARNING." PT HAS SUB-TOTALLY OCCLUDED LAD X 2

Classic "Wellen's Syndrome:"

- Characteristic T wave changes
  - Biphasic T waves
  - Inverted T waves
- History of anginal chest pain
- Normal or minimally elevated cardiac markers
- ECG without Q waves, without significant ST-segment elevation, and with normal precordial R-wave progression

Wellen’s Syndrome ETIOLOGY:

- Critical Lesion, Proximal LAD
- Coronary Artery Vasospasm
- Cocaine use (vasospasm)
- Increased myocardial oxygen demand
- Generalized Hypoxia / anemia / low H&H

Wellen’s Syndrome EPIDEMIOLOGY & PROGNOSIS:

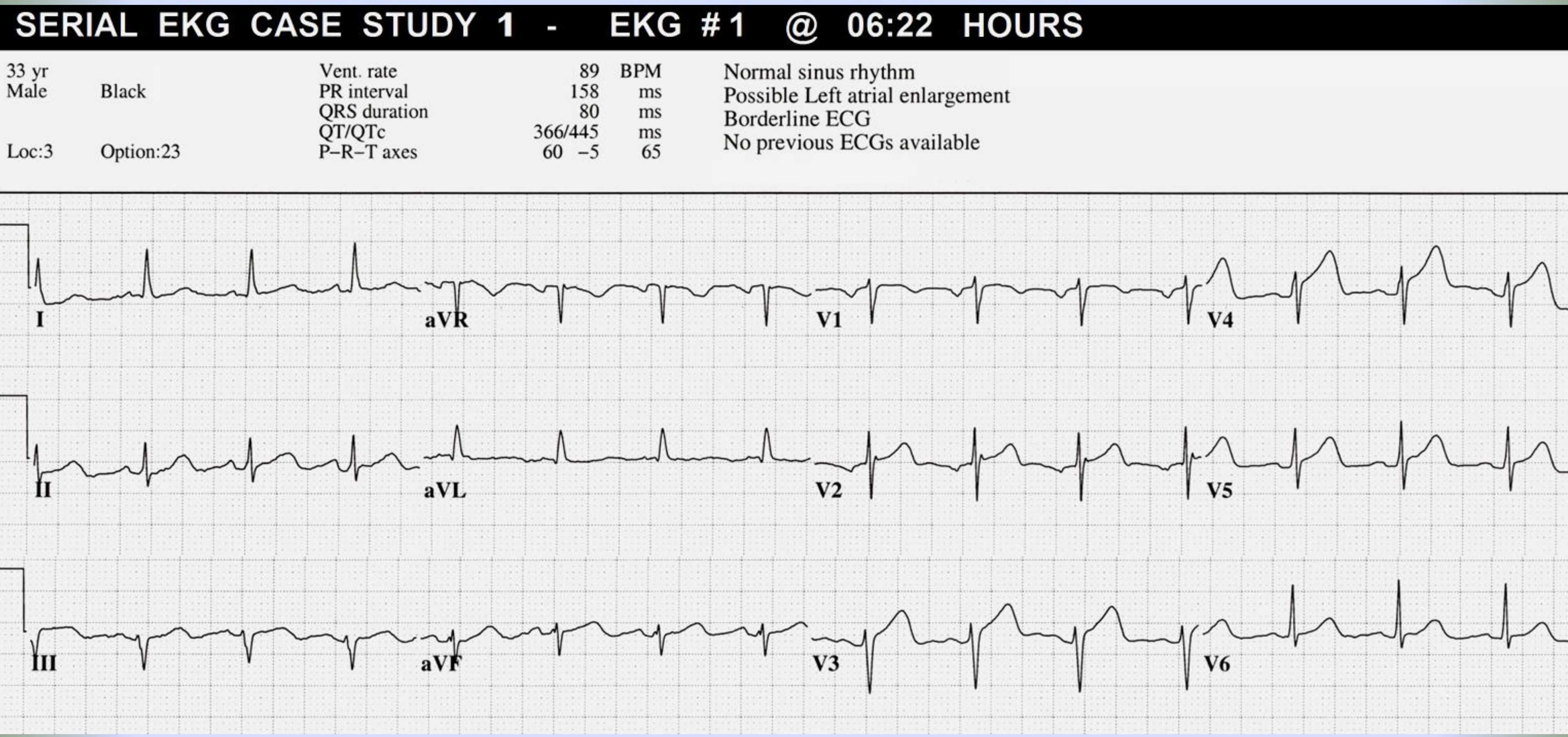
- **Present in 14-18% of patients admitted with unstable angina**
- **75% patients not treated developed extensive Anterior MI within 3 weeks.**
- ***Median Average time from presentation to Acute Myocardial Infarction – 8 days***

Sources: H Wellens et. Al, Am Heart J 1982; v103(4) 730-736

Wellen’s Syndrome Case Study

- 33 y/o male
- Chief complaint “sharp, pleuritic quality chest pain, intermittent, recent history lower respiratory infection with productive cough.”
- ED physician attributed the ST elevation in precordial leads to “early repolarization,” due to patient age, gender, race (African American) and concave nature of ST-segments.

Wellen’s Syndrome Case Study



Wellen's Syndrome Case Study

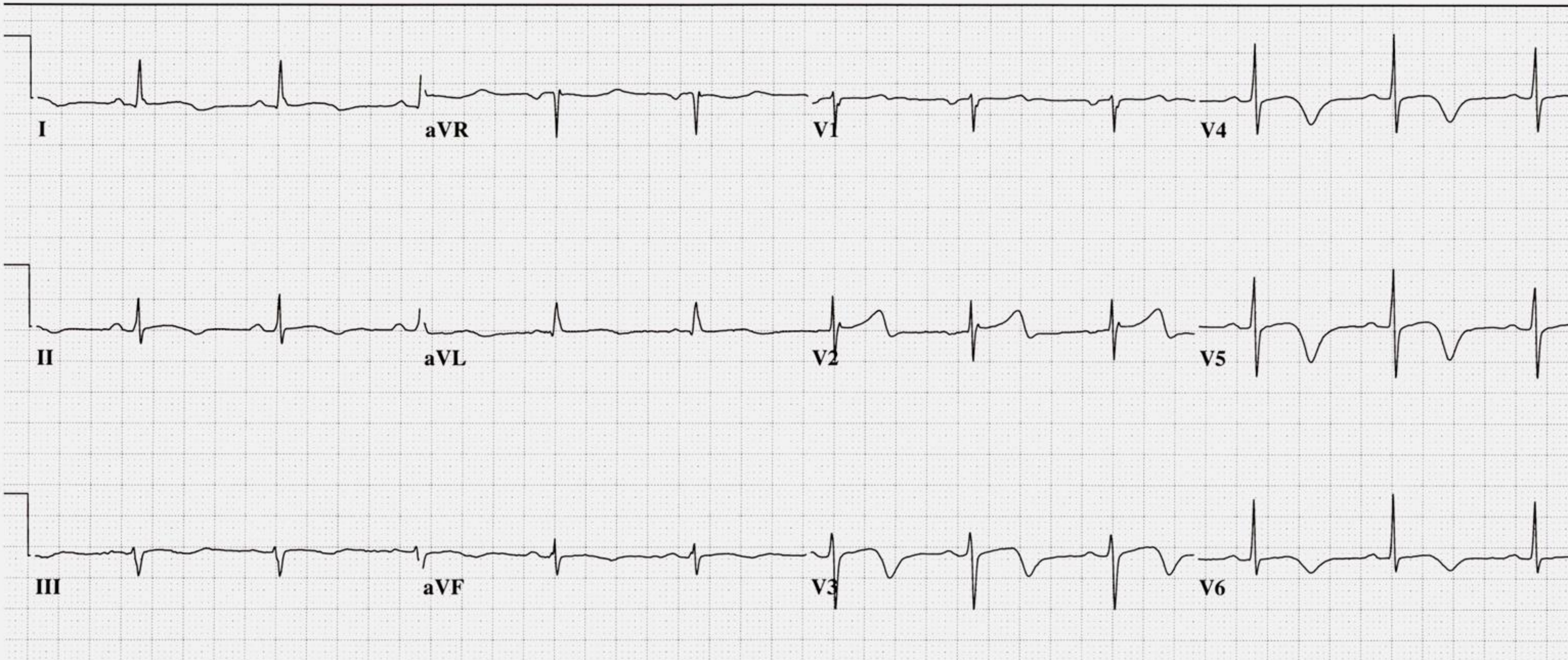
SERIAL EKG CASE STUDY 1 - EKG # 2 @ 09:42 HOURS

33 yr
Male Black

Room:A13
Loc:3 Option:23

Vent. rate	67	BPM
PR interval	160	ms
QRS duration	82	ms
QT/QTc	512/541	ms
P–R–T axes	44 0	54

"**UNEDITED COPY: REPORT IS COMPUTER GENERATED ONLY, WITHOUT PHYS
INTERPRETATION".
Normal sinus rhythm
T wave abnormality, consider anterolateral ischemia
Prolonged QT
Abnormal ECG



DYNAMIC ST-T Wave Changes ARE PRESENT !!

NOW

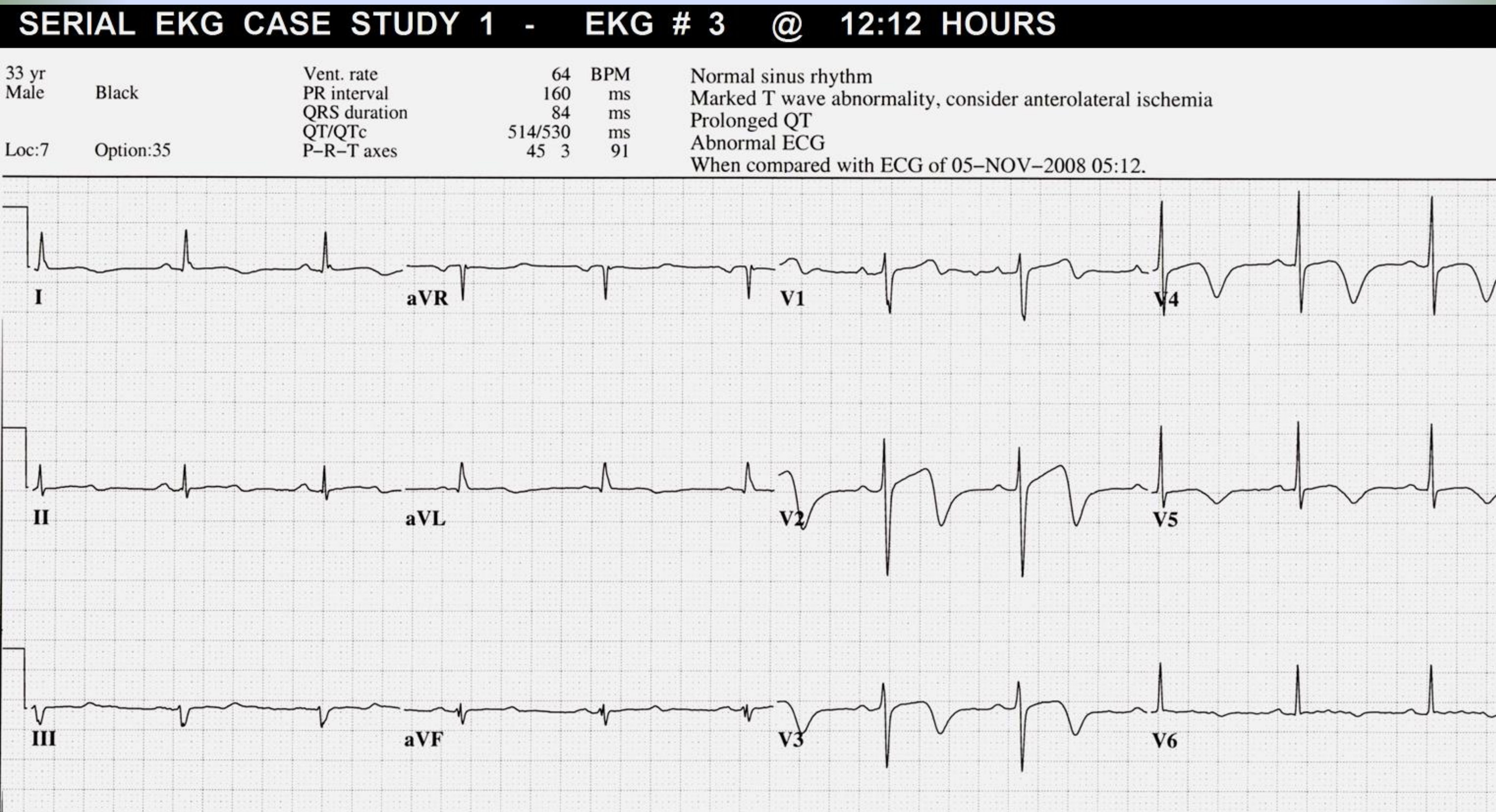
is the time for the

STAT CALL

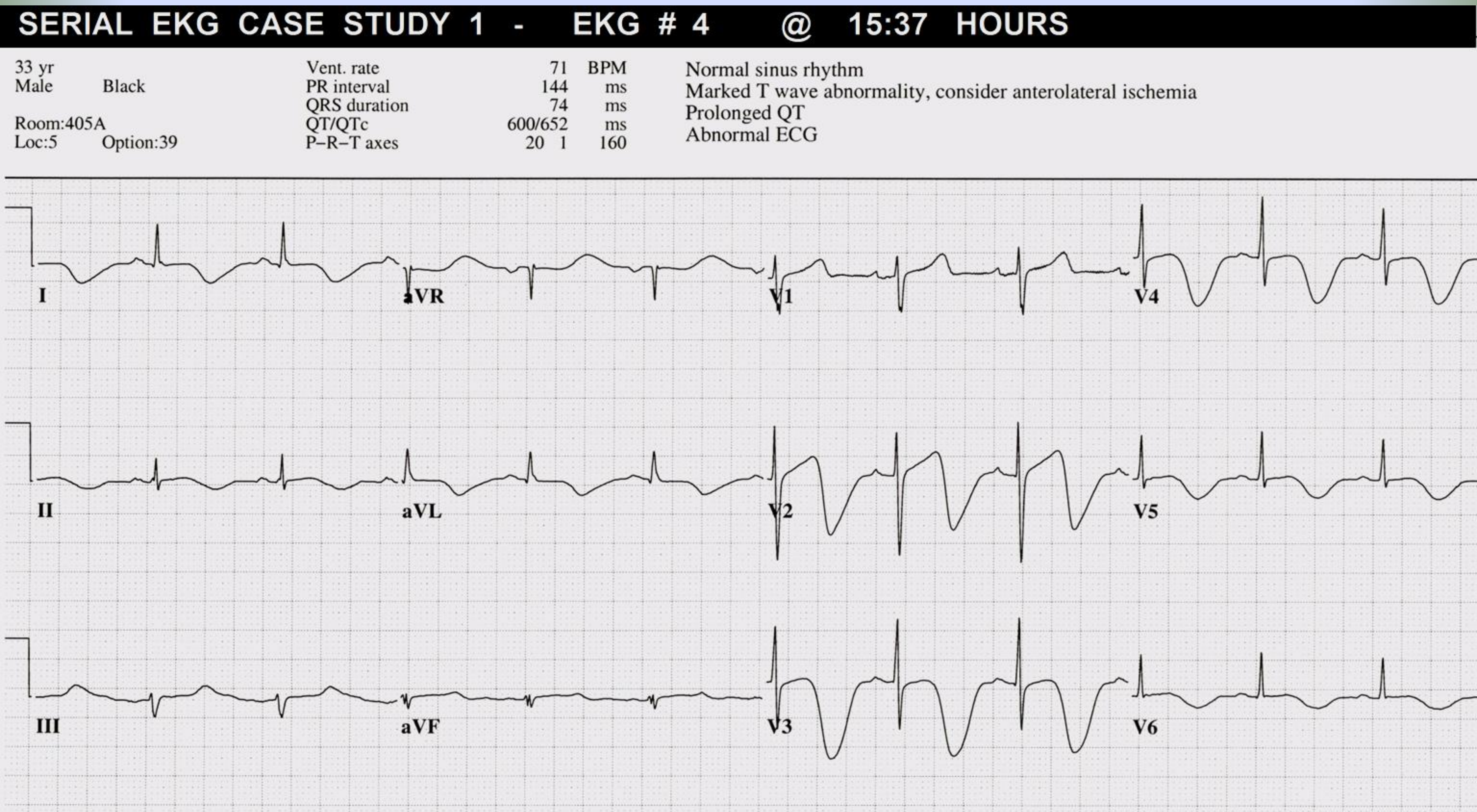
to the

CARDIOLOGIST !!!!

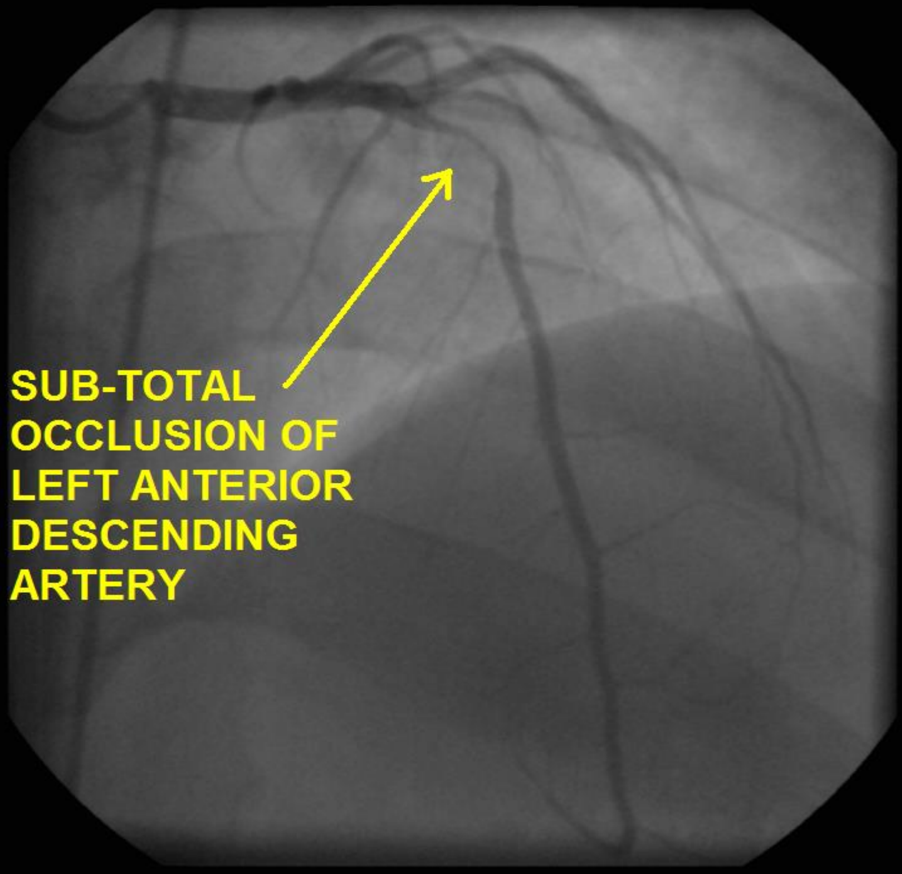
Wellen's Syndrome Case Study

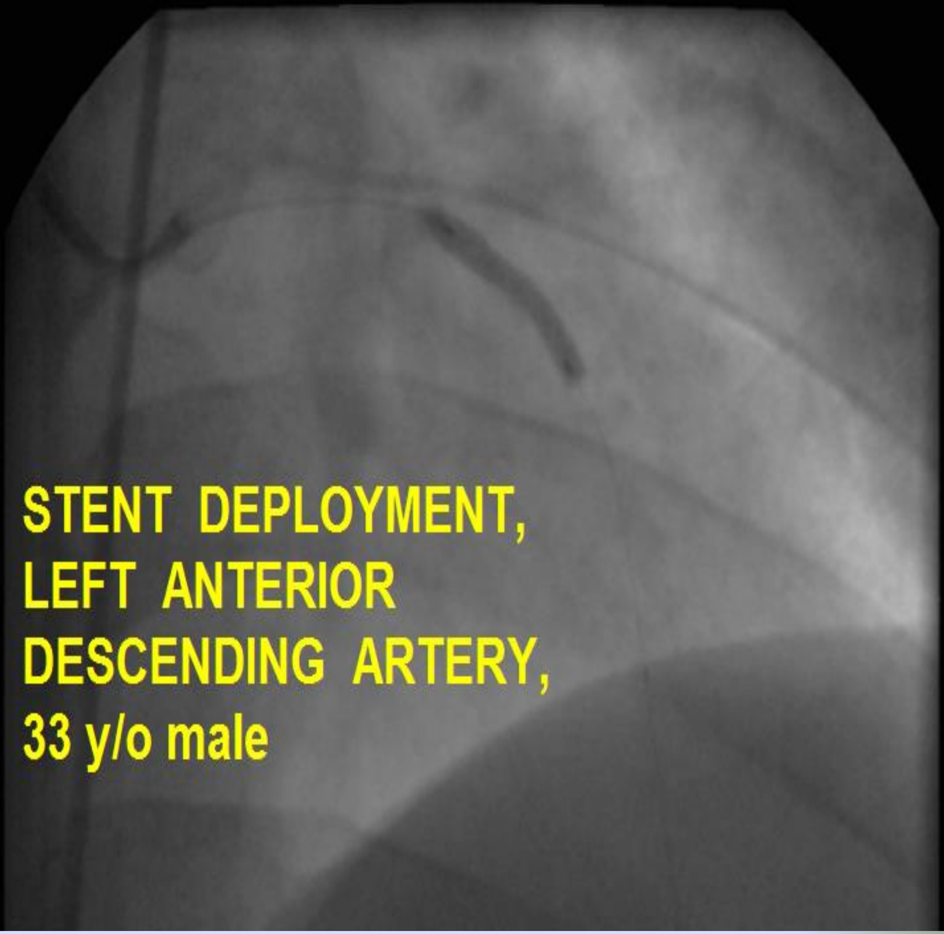


Wellen's Syndrome Case Study

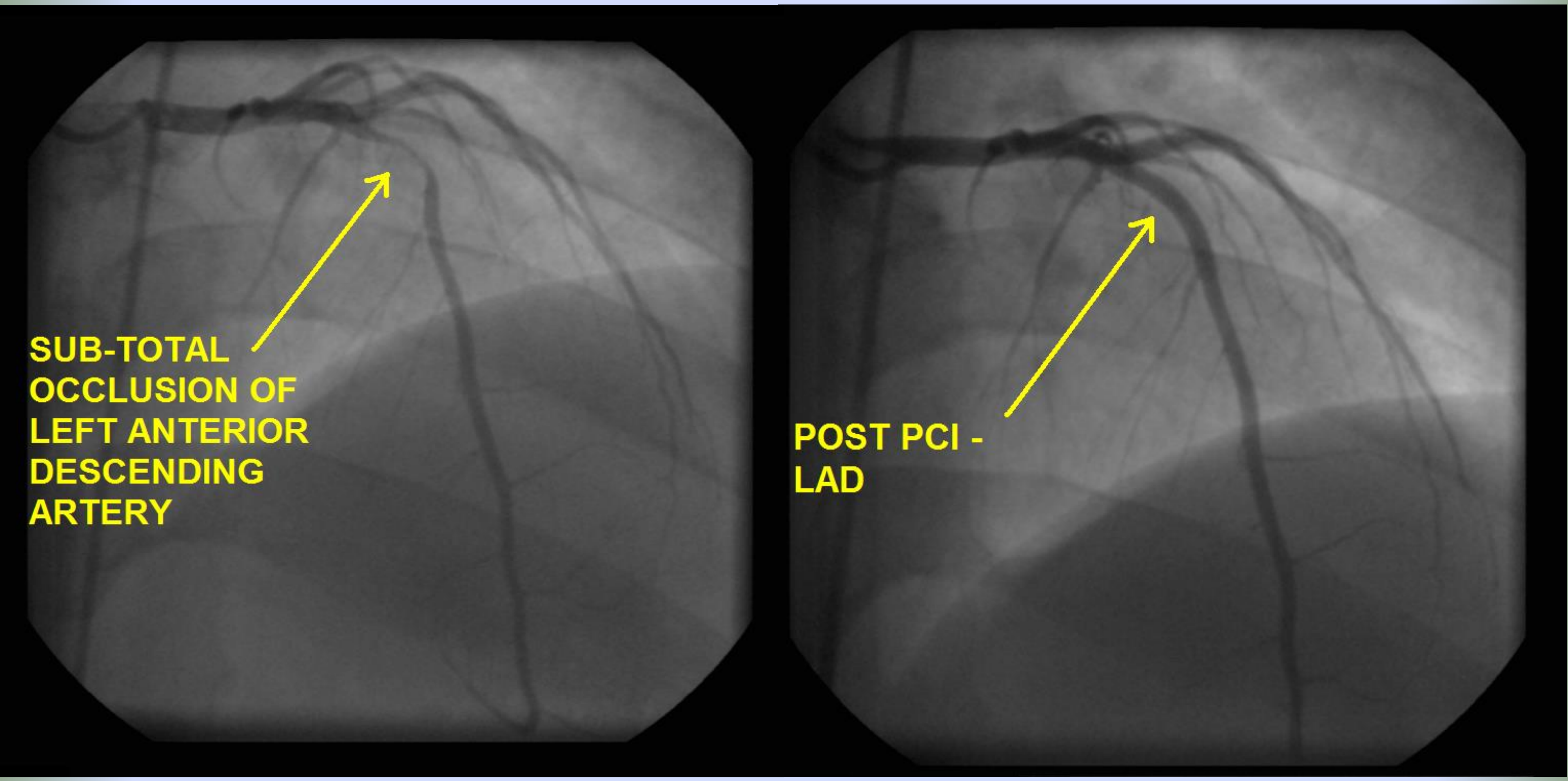


Wellen's Syndrome Case Study





Wellen’s Syndrome Case Study



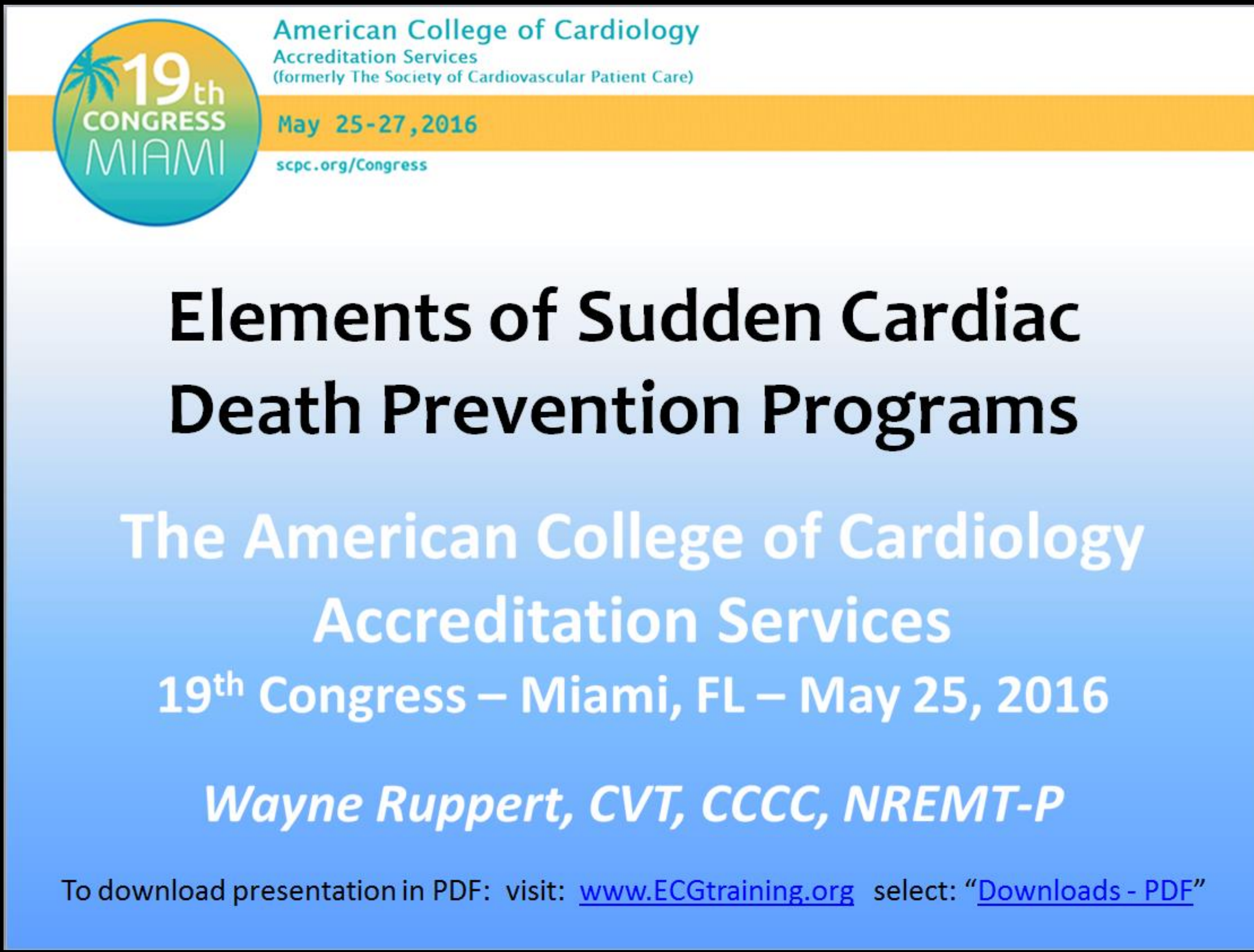
Additional Resources:

- [Wellen's Syndrome, NEJM case study]


Lancaster County, Pennsylvania
Winter, 2002

“NOWHERE”, NEW MEXICO, 1994

CLICK HERE to download “A SHORT Course in LONG QT Syndrome,” a focused excerpt from:



Brief, focused ECG excerpts from the presentation given by Wayne Ruppert at the “19th Congress,” American College of Cardiology Accreditation Services” national conference, on

MAY 25, 2016

Miami, FL ……….

Prevalence
SADS Foundation Stats:

- Each year in the United States, 350,000 Americans die suddenly and unexpectedly due to cardiac arrhythmias. Almost 4,000 of them are young people under age 35. (CDC 2002)
- In 30%–50% of sudden cardiac deaths, it is the first clinically identified expression of heart disease
- 10-12% of Sudden Infant Death Syndrome (SIDS) cases are due to Long QT Syndrome.
- LQTS is now known to be 3 times more common in the US than childhood leukemia.
- 1 in 200,000 high school athletes in the US will die suddenly, most without any prior symptoms—*JAMA 1996; 276*

The SADS Conditions:

- Hypertrophic Cardiomyopathy (HCM)
- Long QT Syndrome (LQTS)
- Short QT Syndrome (SQTS)
- Brugada Syndrome (BrS)
- Arrhythmogenic Right Ventricular Dysplasia (ARVD)
- Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)
- Wolff-Parkinson-White (WPW) Syndrome
- Commotio Cordis
- Less-common conditions (e.g. Marfans, Ehlers-Danlos, Loeys-Dietz Syndromes)

Estimated SADS Prevalence in US Population:

• HCM:	1/500	*J Am Coll Cardiol*. 2014;64
• BrS:	1/2,500	SADS Foundation
• LQTS:	1/2,500	Lenhart,SE 2007 AHA Circ
• ARVD:	1/10,000	SADS Foundation
• CPVT:	1/10,000	US Nat'l Library of Medicine
• WPW:	1/1,000	Circulation.2011; 124: 746-757

Prevalence

Sudden Deaths in Young Competitive Athletes

B Maron et al; AHA Circulation.2009; 119: 1085-1092

Analysis, causes of 1866 Deaths in the US, 1980 –2006:

- **Cardiovascular: 56%**
- **Traumatic: 22%**
- **Commotio Cordis: 3%**
- **Heat Stroke: 2%**
- **Other: 17%**

Most ACS Cardiac Arrest Patients are over age 30.

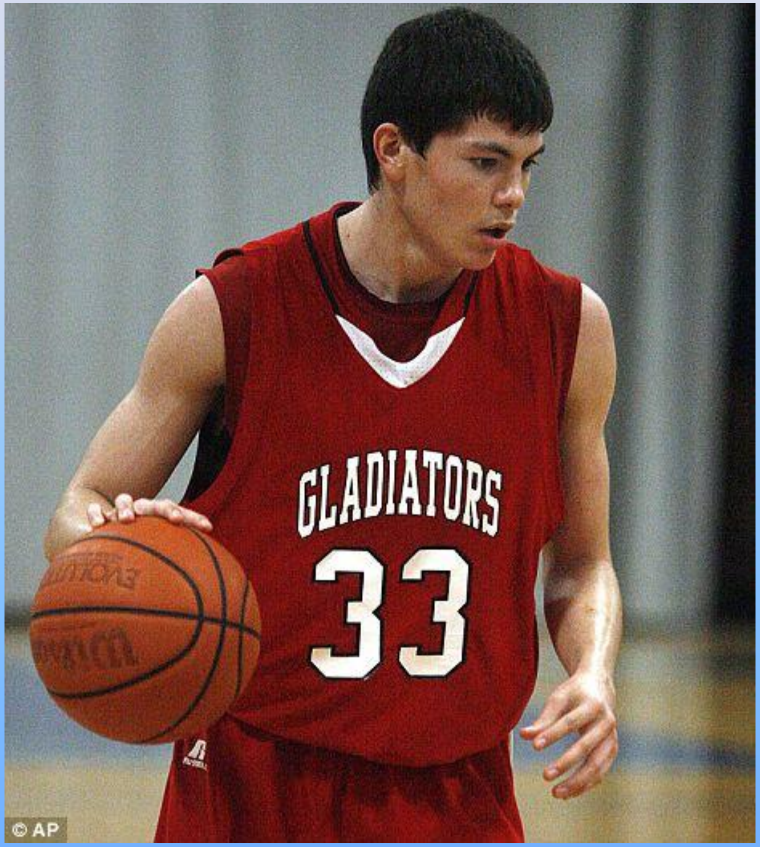
Meet the typical Cardiac Arrest patients affected by SADS . . . .

High School Athlete Dies After Collapsing AtPractice

August 15, 2011 11:28 PM

Share on email**17**

Teen basketball player collapses and dies on court - third school boy sportsman to do so in less than a month



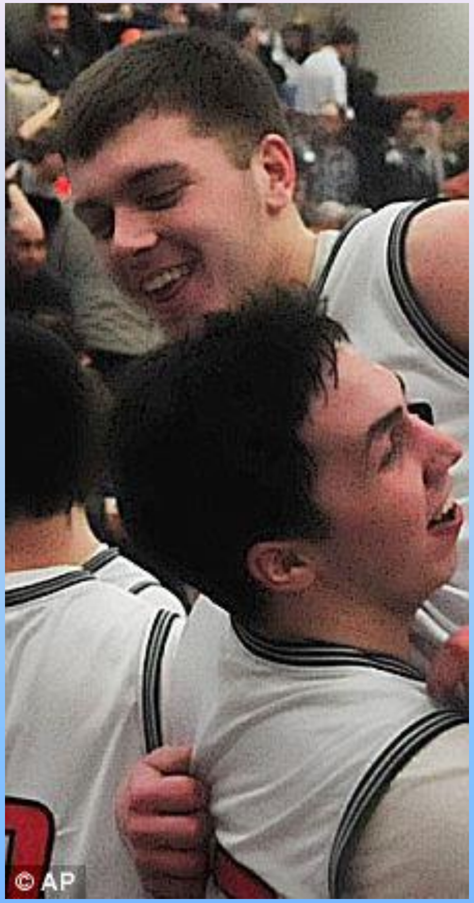
By DAILY MAIL REPORTER
UPDATED: 12:03 EST, 14 March 2011

A teenage basketball player has become the third school boy sports man in less than a month to collapse and die while playing.
Roma High School junior Robert Garza, 16, was playing in the AAU tournament on Saturday with the Hoopsters, a South Texas club team, when he collapsed without any warning.

His death follows that of Wes Leonard, who died of cardiac arrest from an enlarged heart on March 3 and

Matthew Hammerdorfer,17,
who collapsed after taking a tackle to the chest at a rugby match near Denver last week.

Sudden: The death of Robert Garza is the third such school boy death in the last month. The other two both had heart conditions



Tragedy: The death comes only weeks after that of Wes Leonard (right top) and Matthew Hammerdorfer, who collapsed after taking a school rugby match near Denver

Ray-Pec student collapses and dies during track practice
Posted, 2015-03-05
Kansas City Star

A senior at Raymore-Peculiar High School collapsed during track practice Wednesday and died at a hospital, according to school officials. … Click to Continue »

Family and friends mourn popular Boonsboro High School athlete

Michaela Grove 'was just a good kid that didn't follow the crowd, and people liked that'

July 24, 2013|By DAVE McMILLION | davem@herald-mail.com

Family members and friends of a popular Boonsboro High School athlete are mourning her death after she collapsed at a camp in Mercersburg, Pa., on Monday evening.

Michaela Grove’s mother, Brenda Grove, said she believes her 16-year-old daughter was involved in a tug-of-war competition at Camp Tohiglo when she fell to the ground in cardiac arrest.

Greg Moyer, 15

Greg Moyer collapsed and died of sudden cardiac arrest while playing in a high school basketball game in East Stroudsburg, Pennsylvania. His school did not have a automated external defibrillator available and there were no nearby emergency medical services.

Afterwards, a nurse at the hospital emergency room suggested to Greg's parents that they start a fund to help locals schools get AEDs. The Moyers are now involved in AED projects statewide, and Greg's mother, Rachel Moyer, has traveled as far as Hawaii to advocate for school AED legislation and donate AEDs

"Princess George" died at age 3 of sudden cardiac arrest brought on by an undiagnosed heart condition. At the suggestion of the doctor who saw "George" in the emergency room, her brother was subsequently tested for heart problems. He was diagnosed with a heart condition that is, fortunately, treatable.

Jennifer Lynn Balma, their mother, notes that "George" never showed any symptoms of cardiac problems — *until the day she suddenly stopped breathing.*

Olivia Corinne Hoff, 14

Olivia died at age 14 from sudden cardiac arrest attributed to **Long QT Syndrome.** **The condition was undiagnosed. Olivia, a high school freshman involved in sports and cheerleading, suffered cardiac arrest during the night. Her mother found her unresponsive and called 911. Olivia was subsequently hospitalized, but did not survive.**

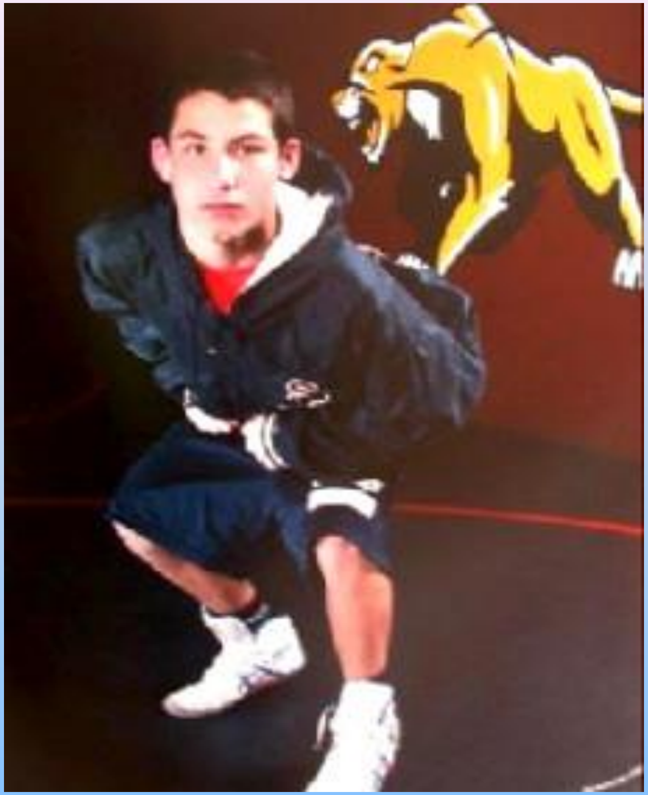
Her mother, Corinne Ruiz, wrote: **"Today, 6 years later, I cry for my daughter every day. Not a day goes by that I don't ask myself:** ***If only I had been told that there are screening tests or preventative treatments."***

High school quarterback Reggie Garrett threw his second touchdown pass of the night, walked off the field, and [collapsed from sudden cardiac arrest](). He died in the ambulance on the way to the hospital in West Orange, Texas.
In the news coverage following Garrett's death, Dallas station WFAA.com urged cardiac screening for high school athletes.

Zachary Shrah, 16

High school football player Zachary Schrah collapsed and died of sudden cardiac arrest during football practice in Plano, Texas. His mother, Karen Schrah, has become an advocate for legislation mandating heart screenings as a part of student physicals.

Zachary's death had an impact on the community at large. Heart Hospital Baylor Plano now offers low-cost ECGs and echocardiograms for the area's student athletes.



Eric Paredes, a two-sport high school athlete, had an enlarged heart. But no one knew about it until it was too late. His father, Hector Paredes, found Eric on the kitchen floor, unconscious and not breathing. He administered CPR, but was unable to revive him. Eric died of sudden cardiac arrest.

In Eric's memory, the family has organized electrocardiogram (EKG) screening for other students at Eric's San Diego area high school.

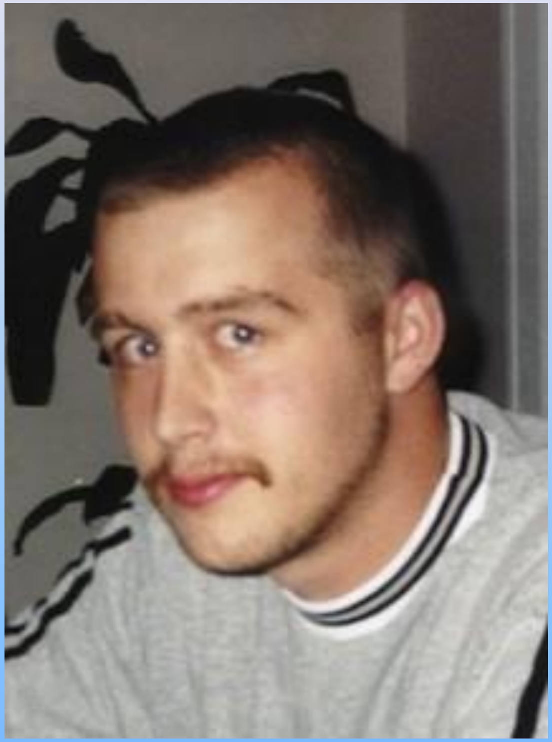
In 2005, Chicago conservationist and wildlife educator Max Schewitz died of sudden cardiac arrhythmia. Since then, the Max Schewitz Foundation, created by his parents, has provided free electrocardiograms (EKGs) for more than 10,000 Chicago-area students through a Screen for Teens program.

According to media reports, the screenings have identified 142 teens who are considered at-risk for sudden cardiac death because of cardiac conditions.

Nick Varrenti, 16

Nick Varrenti played in two high school football games — varsity and junior varsity — on Labor Day weekend. A day later, he suffered sudden cardiac arrest and died. His family learned later that Nick had lived with an undiagnosed heart condition, hypertrophic cardiomyopathy.
Nick's parents created the Nick of Time Foundation, which is dedicated to education schools, athletes, and communities about sudden cardiac arrest, public access defibrillator (PAD) programs, and cardiac screenings.

Jimmy Brackett, 22, and Crissy Brackett, 21



The hereditary cardiac disease [Long QT Syndrome] ran in Jackie Renfrow's family, *but she had no idea about it until two of her children died from sudden cardiac arrest.*

Brandon athlete dies after collapsing at practice

TAMPA — A Brandon High School senior Milo Meeks died Saturday, one day after conditioning with the basketball team

"This is mind blowing," said Ben Bromley, the junior varsity and assistant varsity basketball coach at Armwood.

Jeremy Twining,
age 21
Dade City, Florida
February 1, 2015

Obituaries

Jeremy Grant Twining

TWINING, Jeremy Grant, 21, of Dade City, joined his savior Jesus in Heaven on Feb. 1, 2015. He was born May 31, 1993. He graduated from Pasco High School and was studying Criminal Justice at Liberty University. He is survived by his parents, John and Julie Twining of Dade City; siblings, Jonathan, Jessica and James Twining of Dade City; girlfriend, Lydia Tucker of Temple Terrace; paternal grandparents, Dave and Shirley Twining of Tampa; maternal grandparents, Edna Margaret Neatherly of Tampa and Earl and Ginger Hornsby of Cromwell, Conn.; and countless aunts, uncles, and cousins. Jeremy will always be remembered for his contagious laugh, his huge caring heart, and his love for his Lord and Savior Jesus Christ. A private graveside service was held Feb. 6 from the Florida National Cemetery in Bushnell. A memorial service was held at First Baptist Church of Dade City on Feb. 7. In lieu of flowers make send donations to the Sudden Arrhythmia Death Foundation at SADS.org. Hodges Family Funeral Home was in charge of arrangements.

. . . . And on a more personal note:

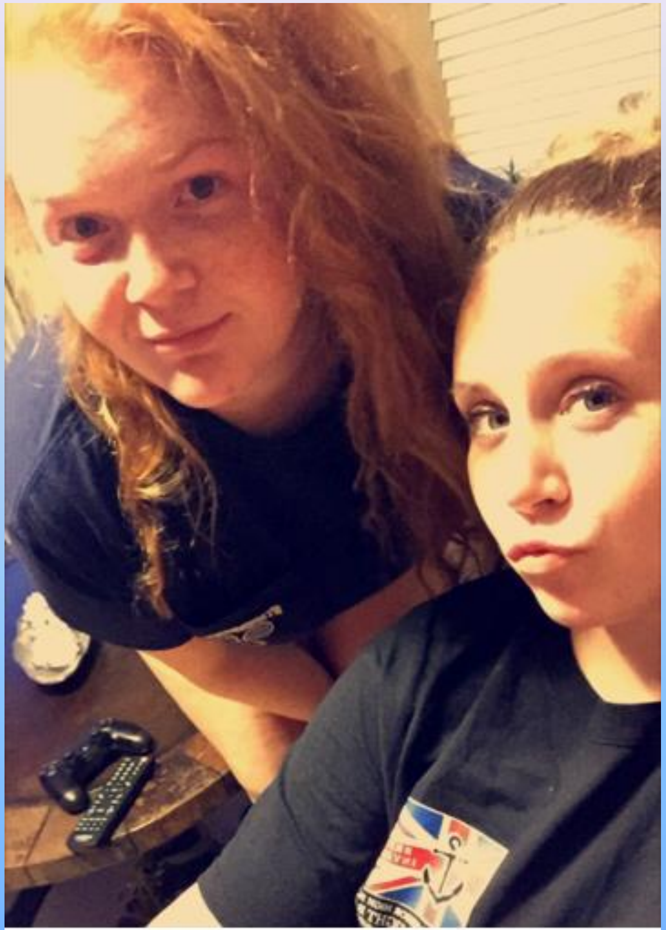
This slide added April 27, 2016:

Yesterday, a good friend of my step-daughter collapsed during a tennis game in the Carrollwood community of Tampa, Florida. She was 16 years old.

A physician bystander started CPR, but since no AED was available, she did not survive.

Sudden death was the first indication that she suffered from a cardiac condition. At the current time, her specific diagnosis is unknown.

Entry 5/2/2016: I was advised that the cause of cardiac arrest was Hypertrophic Cardiomyopathy.

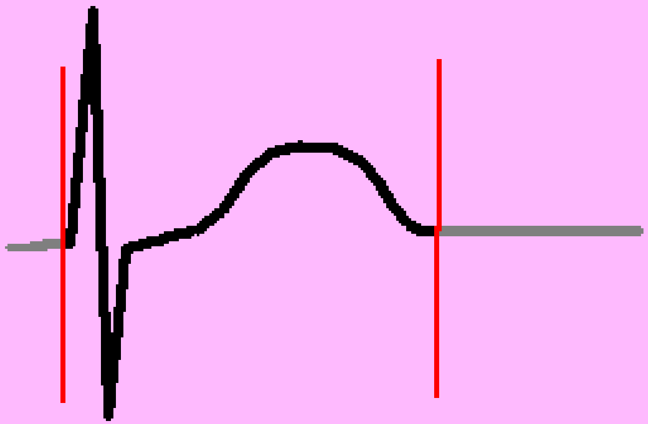


My step-daughter, Caitlin Cameron (right) with her friend, also named Caitlin (left) who collapsed and died during a tennis match on 4/26/16

“Healthcare organizations have an obligation to implement programs, practices, protocols, policies and procedures designed to eliminate the needless mortality of SADS in our communities.”

"Healthcare professionals who evaluate young patients have an obligation to be aware of risk factors, signs and symptoms of patients with potential SADS conditions. Those who read ECGs should be aware of the subtle ECG identifiers of SADS conditions."

THE Q - T INTERVAL



- BEGINNING OF QRS COMPLEX TO THE END OF THE T WAVE

- NORMAL VALUES VARY BASED ON HEART RATE
- SEVERAL WAYS TO DETERMINE NORMAL LIMITS

THE *QTc INTERVAL

* QTc = Q-T interval,
corrected for heart rate

HEART RATE	MALE	FEMALE
150	0.25	0.28
125	0.26	0.29
100	0.31	0.34
93	0.32	0.35
83	0.34	0.37
71	0.37	0.40
60	0.40	0.44
50	0.44	0.48
43	0.47	0.51

Annals of Internal Medicine, 1988 109:905.

Determining the QTc

Manual calculation:

QT CORRECTION FORMULAS:

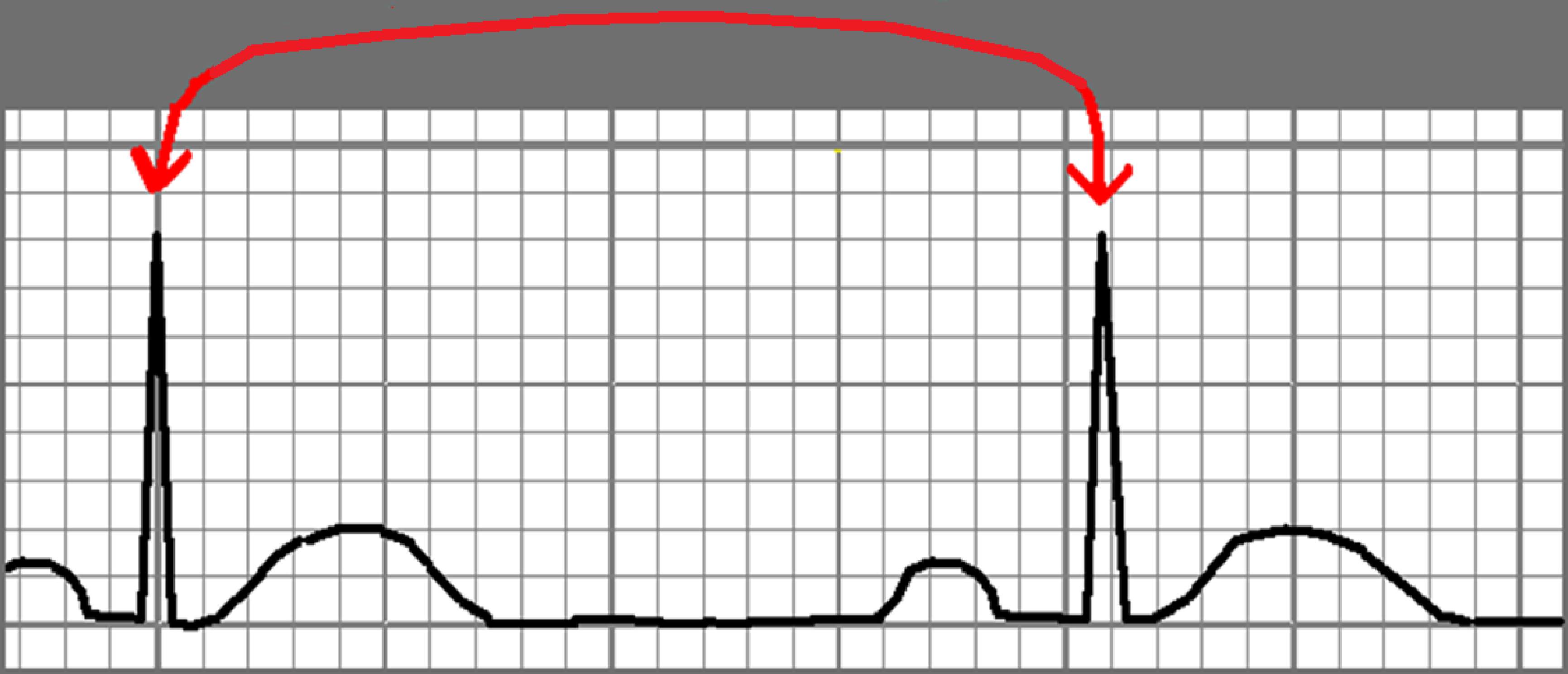
Bazett's	QTc=QT/√ RR
Fredericia	QTc=QT/(RR)1/3
Framingham	QTc=QT+0.154(1-RR)
Rautaharju	QTp=656/(1+HR/100)

DETERMINING Q-T INTERVAL LIMITS

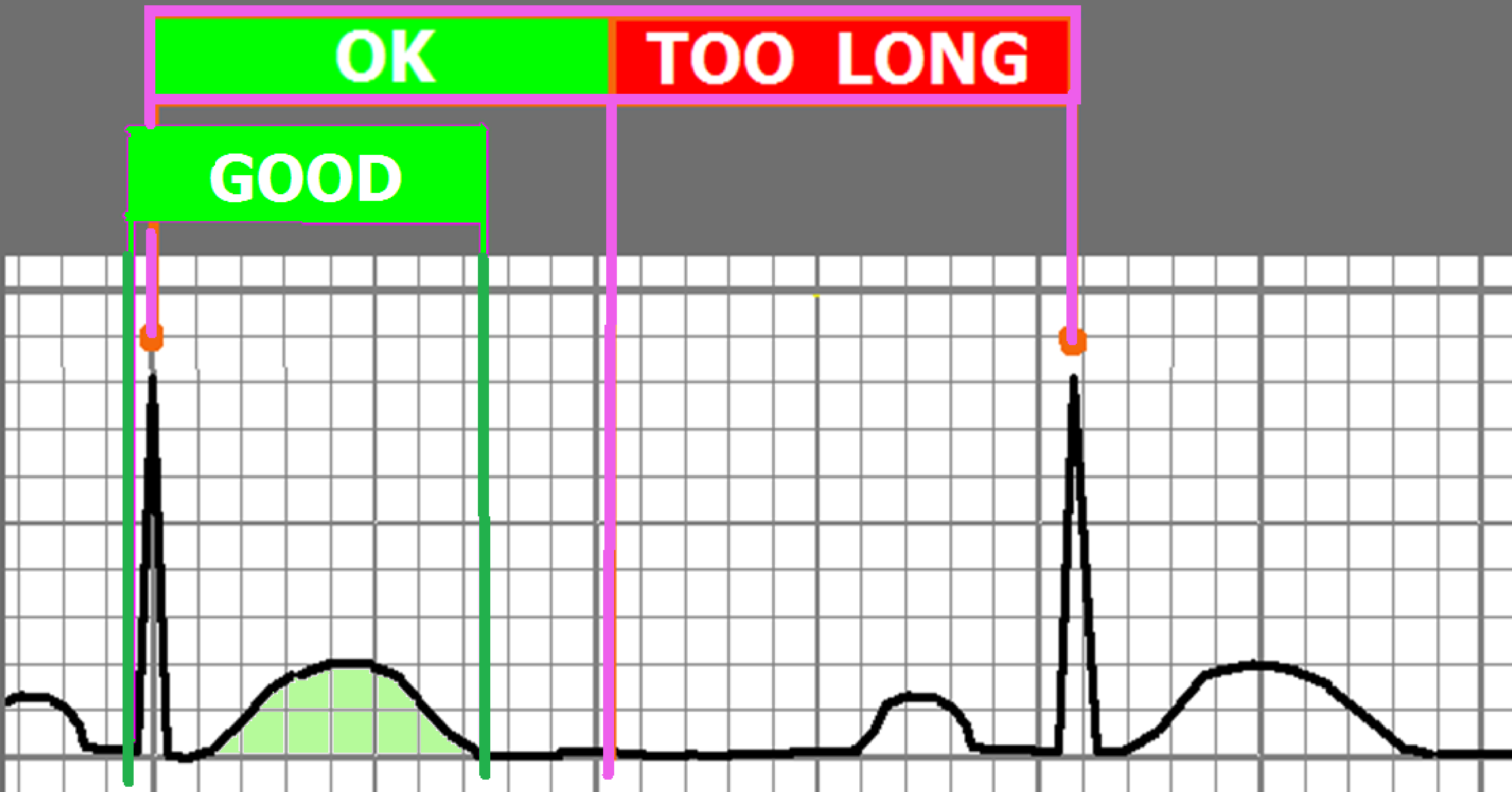
THE "QUICK PEEK" METHOD

☞ **Relatively accurate method to quickly identify patients with abnormal QT Intervals.**

- Applies to patients with normal heart rates (60-100) and narrow QRS (QRSd <120ms)



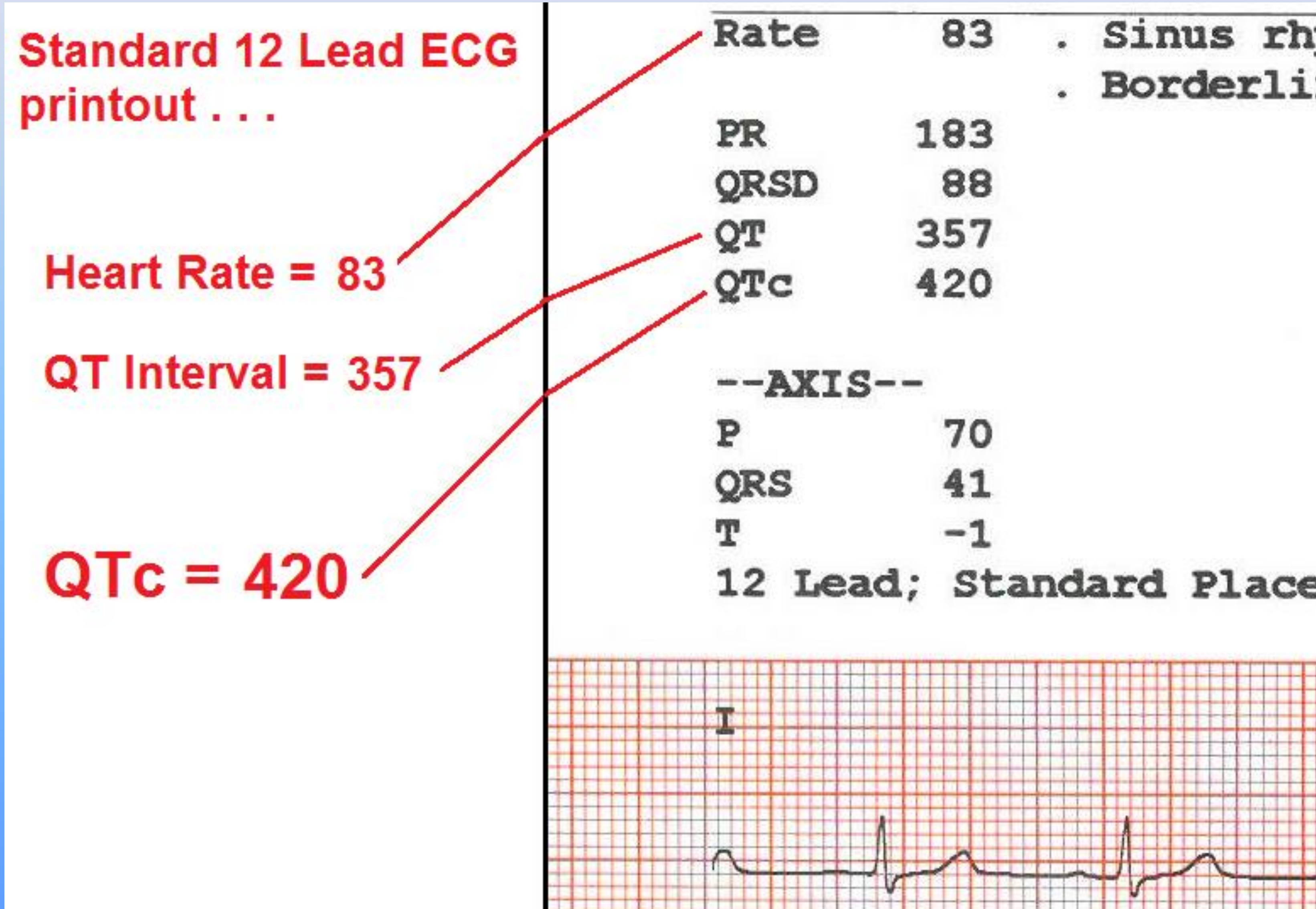
The Q - T Interval should be LESS THAN 1/2 the R - R Interval



The Q - T Interval
should be LESS THAN 1/2 the
R - R Interval

Determining the QT / QTc

Method 1 – 12 Lead ECG Report:

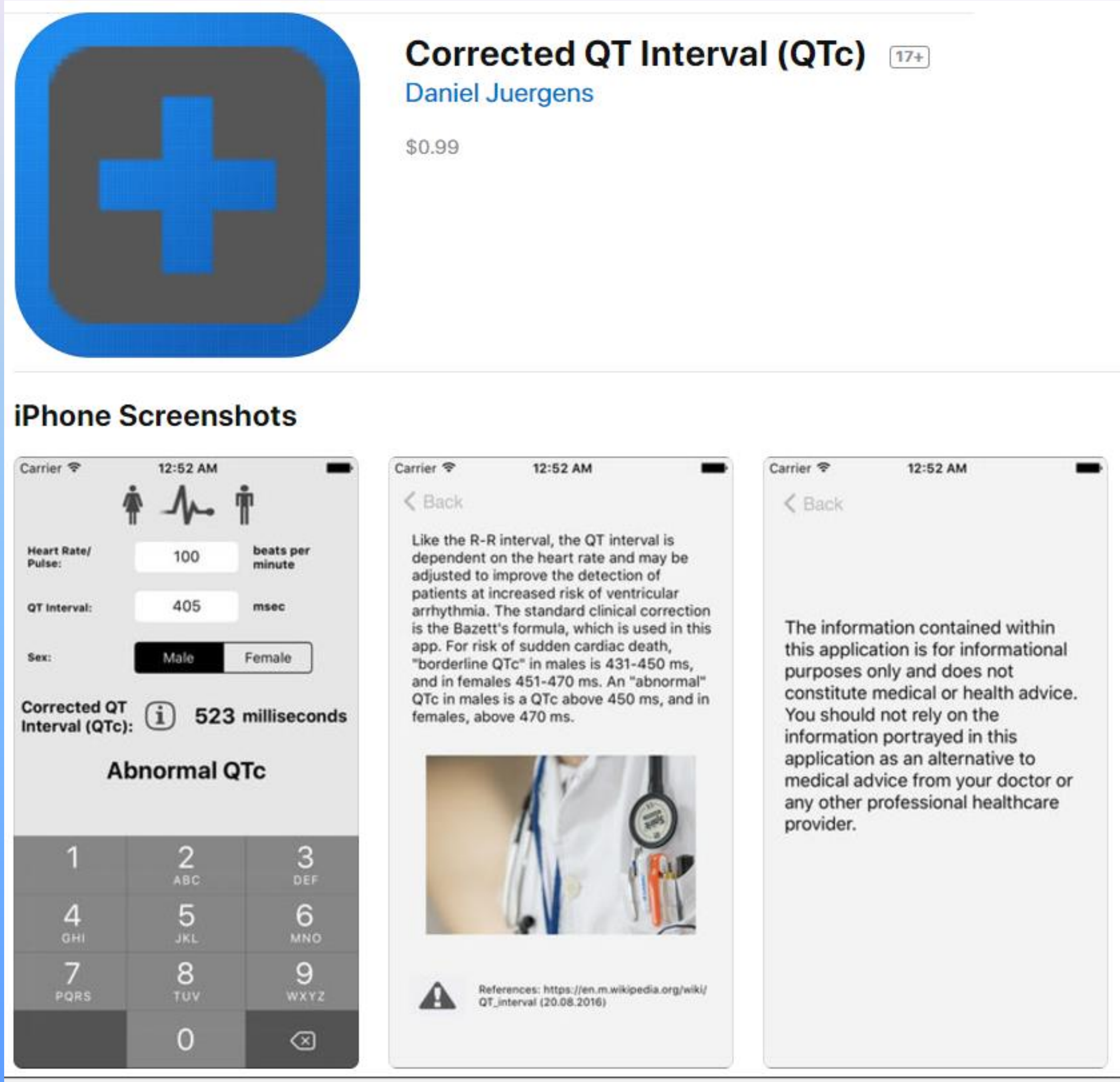


Determining the QTc

Method 4, Use a Smartphone App:

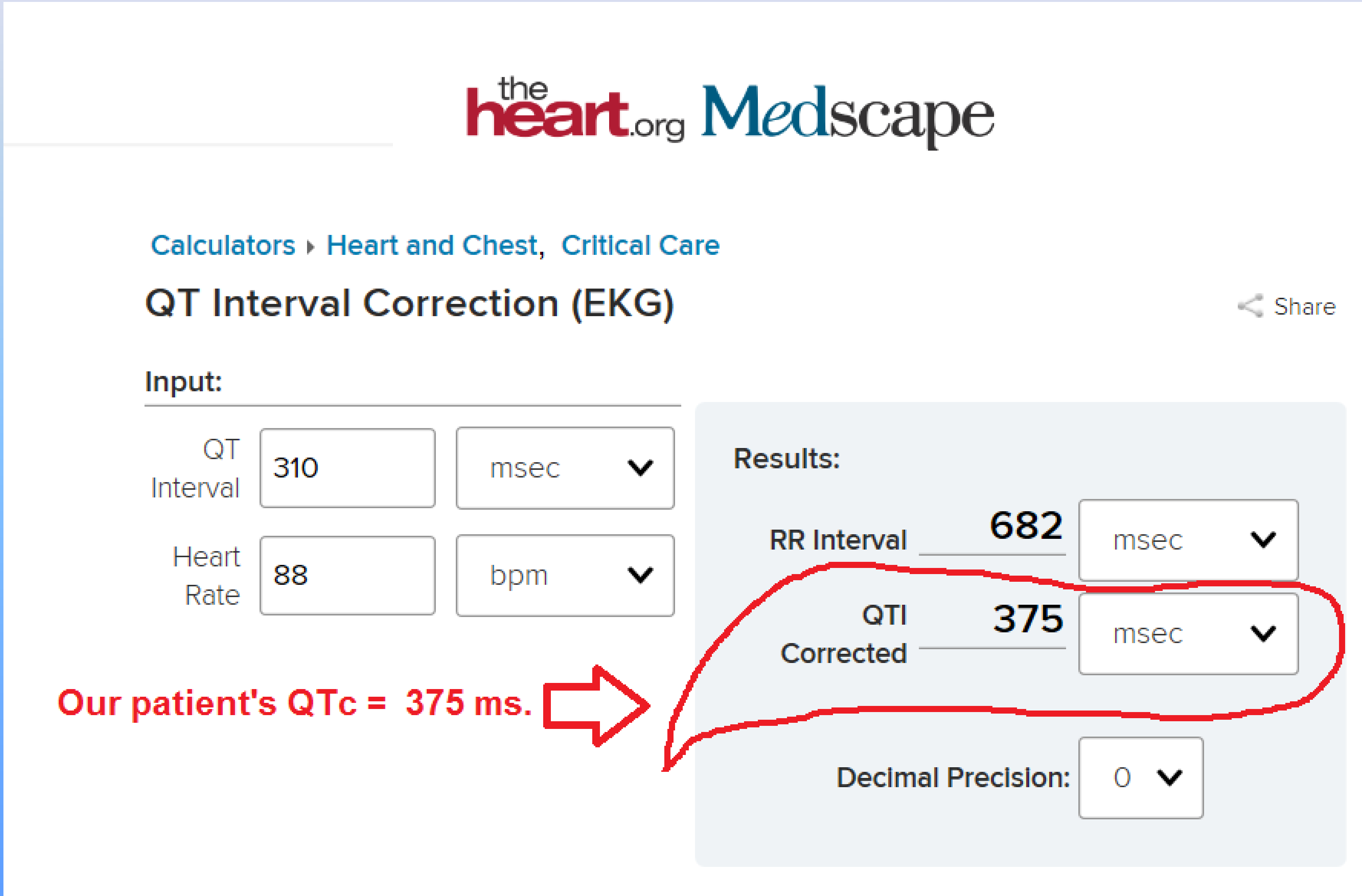
- **iPhone**
  - https://itunes.apple.com/us/app/corrected-qt-interval-qtc/id1146177765?mt=8
- **Android**
  - https://play.google.com/store/apps/details?id=com.medsam.qtccalculator&hl=en

"There's an APP for that!"



Determining the QTc

Method 3, Use a Web-based App:



QTc Values:

Too Short:	**< 390 ms**
Normal	
-Males:	**390 - 450 ms**
-Females:	**390 - 460 ms**
Borderline High	
-Males:	**450 - 500 ms**
-Females:	**460 - 500 ms**
High (All Genders):	**500 - 600 ms**
Critical High	
(associated with TdP):	**600 + ms**

SOURCE: "ACC/AHA/HRS Recommendations for Standardization and Interpretation of the ECG, Part IV: The ST Segment, T and U Waves, and the QT Interval" Rautaharju et al 2009

Dysrhythmia Associated with Mortality, Triggered by LQTS: *Torsades de Pointes*

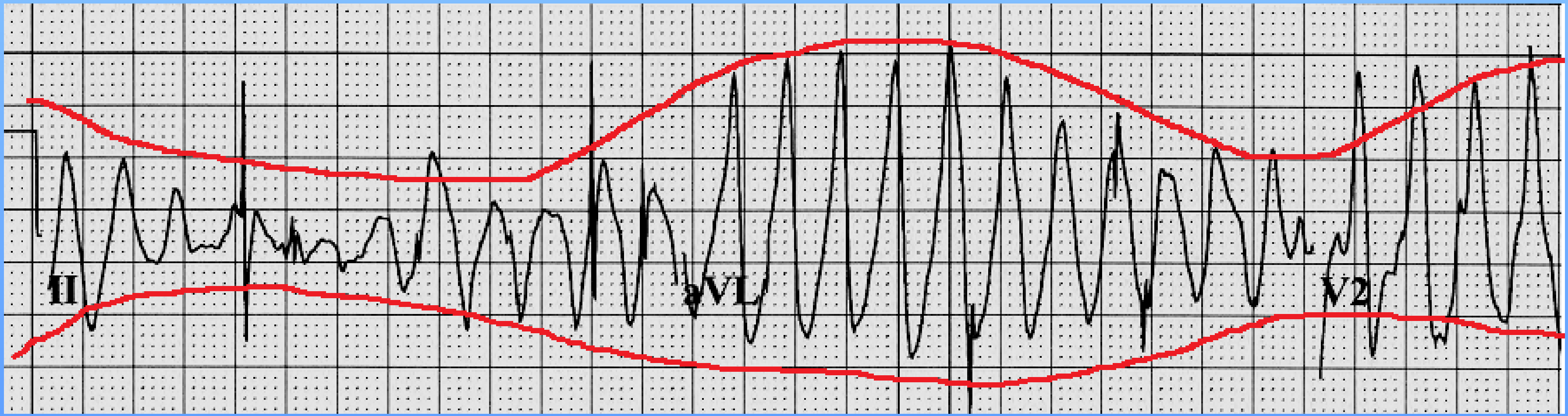
Torsades de Pointes (TdP) – HEMODYNAMICS:

- Decreased – to – NO Cardiac Output
- Often patient PULSELESS during episode
- Patients often report SYNCOPE when TdP self-terminates.
- May DETERIORATE into VENTRICULAR FIBRILLATION and CARDIAC ARREST. ("Sudden Death")

ECG Characteristics of TdP: The QRS Pattern of *Torsades de Pointes*

resembles . . . . .

a piece of Twisted Ribbon !

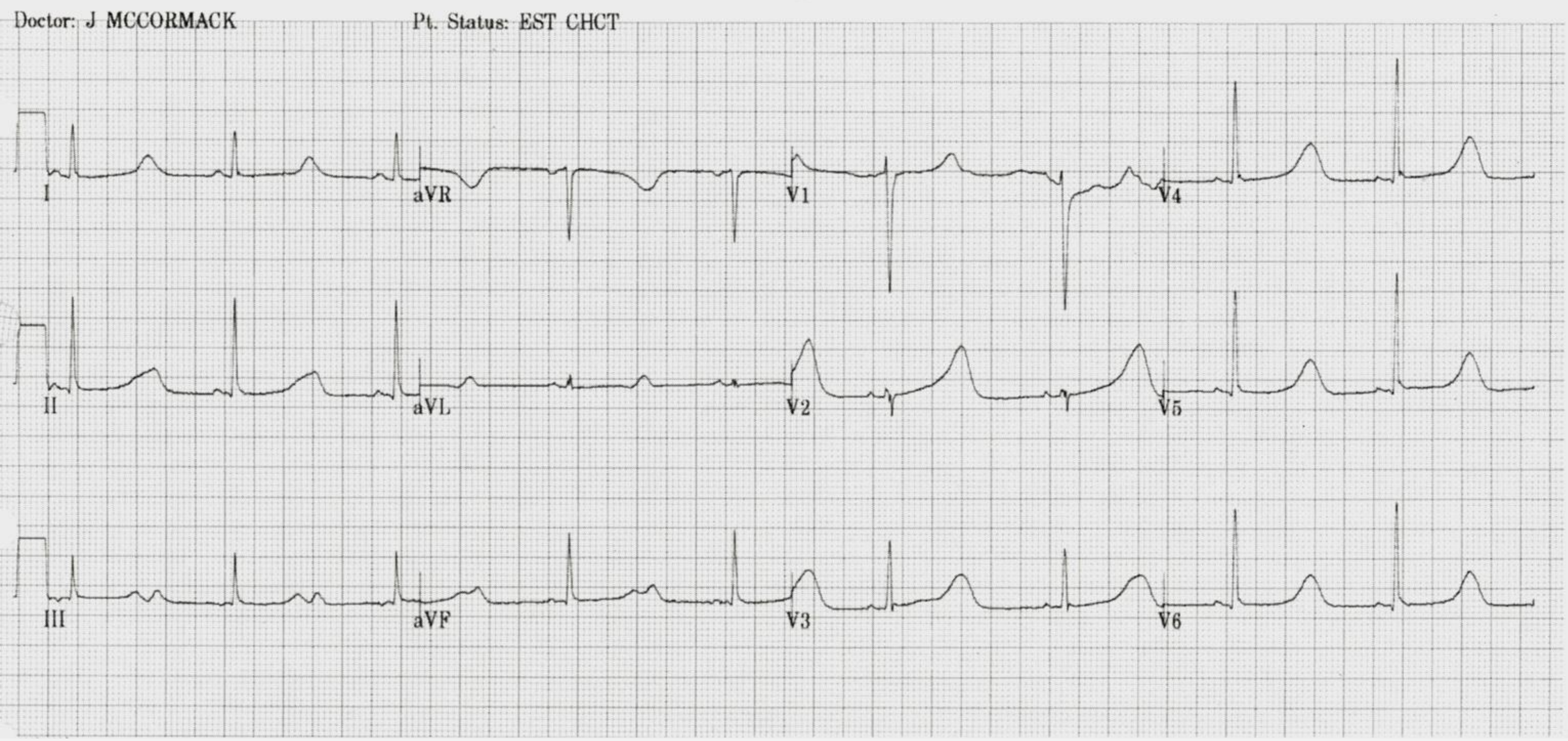


22 y/o FEMALE

Vent. rate 53 bpm
PR interval 110 ms
QRS duration 84 ms
QT/QTc 678/636 ms
P-R-T axes 25 60 48

PEDIATRIC CARDIOLOGY ASSOCIATES

Chief Complaint: “Grand-Mal Seizures”
…. With NO postictal phase!

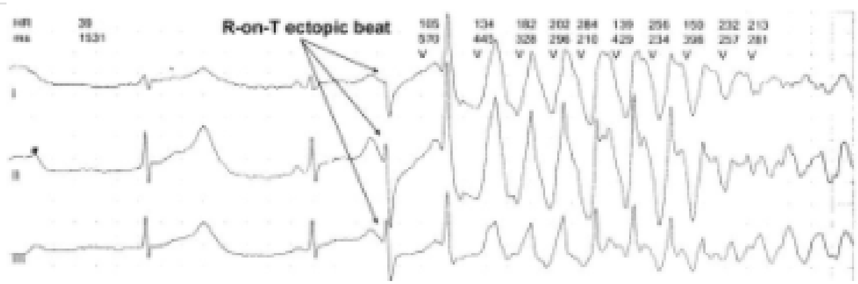


WHEN THE “QUICK PEEK” METHOD for QT INTERAL EVALUATION IS APPLIED TO THE ABOVE ECG, WHAT IS THE RESULT?

Long QT Syndromes and Torsade de Pointes

Gan-Xin Yan

I. Long QT syndrome: What every physician needs to know.



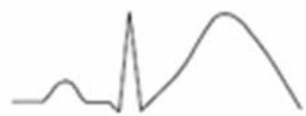
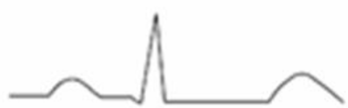
Long QT syndrome (LQTS) is an inherited disorder of delayed ventricular repolarization characterized by a prolonged QT interval on electrocardiography (ECG) and a propensity to torsades de pointes (TdP). TdP by definition is: (1) a polymorphic ventricular tachycardia that occurs specifically under conditions of QT prolongation; and (2) it is almost always initiated by R-on-T ectopic beats. Clinical manifestations of TdP include syncope (fainting), seizure (epilepsy), or sudden cardiac death. As shown in Figure 1, an episode of sustained TdP was recorded in a patient aged 13 years with LQTS type 2. The episode during which the boy had "seizures" was triggered by the alarm clock in the early morning.

Figure 1:

Torsade de pointes in a long QT syndrome type 2 patient.

GENETICALLY ACQUIRED LONG QT SYNDROMES:

ECG PATTERNS of 3 MOST COMMON VARIATIONS:

Type	Current	Functional Effect	Frequency Among LQTS	ECG[12,13]	Triggers Lethal Cardiac Event[10]	Penetrance*
LQTS1	K	↓	30%-35%		Exercise (68%) Emotional Stress (14%) Sleep, Repose (9%) Others (19%)	62%
LQTS2	K	↓	25%-30%		Exercise (29%) Emotional Stress (49%) Sleep, Repose (22%)	75%
LQTS3	Na	↑	5%-10%		Exercise (4%) Emotional Stress (12%) Sleep, Repose (64%) Others (20%)	90%

Etiology of Long QT Syndromes:

Congenital (14 known subtypes)

Genetic mutation results in abnormalities of cellular ion channels

Acquired

Drug Induced

Metabolic/electrolyte induced

Very low energy diets / anorexia

CNS & Autonomic nervous system disorders

Miscellaneous

Coronary Artery Disease

Mitral Valve Prolapse

PROLONGED Q - T INTERVAL

THINK:

- CHECK K+ AND MAG LEVELS
- POSSIBILITY OF TORSADES

PROLONGED Q - T INTERVAL

THINK:

- CHECK K+ AND MAG LEVELS
- POSSIBILITY OF TORSADES

- QUESTION MEDS THAT PROLONG Q-T

QT Prolongation -- *STAT Intervention:*

☞ *Avoidance of Meds that are known to prolong the QT Interval. Click here for current list from CREDIBLEMEDS.ORG*

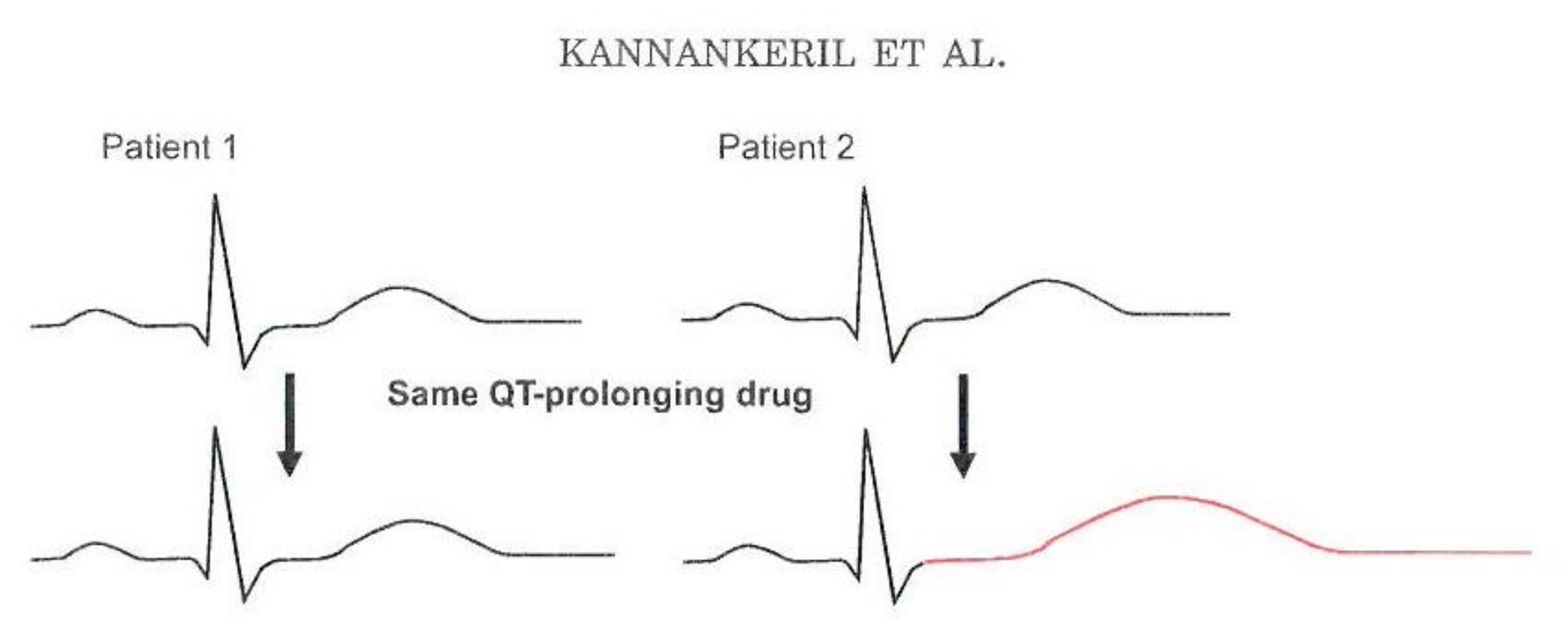
Commonly used QT prolonging meds include:

-Amiodarone	**-Ritalin**
-Procainamide	**-Pseudophedrine**
-Levaquin	**-Haloperidol**
-Erythromycin	**-Thorazine**
-Norpace	**-Propulcid**
-Tequin	**-Zofran**
-Benadryl	**-Ilbutilide**

and MANY more!

PATIENT 1: NORMAL

PATIENT 2: Genetic susceptibility; sensitivity to QT prolonging drugs:



Click here for link to paper by Kannankeril et al (2010 Pharmacological Reviews) that describes genetic susceptibility described above.

Policy, Procedure and Protocol

Bayfront Health Dade City

Policy Title: QTc Interval Monitoring Department: Pharmacy	Function Team: Medication Management Effective Date: 11/15
Prepared by: William Parker, PharmD, CGP, Derek Harmeson, RN, BSN; Wayne Ruppert, CVT, CCCC	
Date(s) Reviewed: 11/15	Date(s) Revised: N/A
Approvals: __X__P&T __X__MEC	

1. PURPOSE:

   1.1. To establish a protocol and process by which the Pharmacy and Nursing departments can monitor QTc intervals in patients at high risk for QTc prolongation and subsequently decrease the risk for sudden cardiac death

2. POLICY:

   2.1. The Policy, Procedure and Protocol will be utilized selectively and appropriately by the Pharmacy and Nursing staff in order to evaluate and monitor patients at high risk for QTc prolongation and decrease their risk for arrhythmias and sudden cardiac death

Click here to download QTc Interval Monitoring Policy

Bayfront Health
Dade City

Results of QTc Monitoring Protocol - Trial - March 8 - March 22

In patients with QTc 500 or more (indicated by red arrow →), QT prolonging drugs were discontinued and substituted with non-QT prolonging medications.

PATIENT:	3/8/2016	3/9/2016	3/10/2016	3/11/2016	3/14/2016	3/15/2016	3/16/2016	3/17/2016	3/18/2016	3/21/2016	3/22/2016
A	389	400									
B	425	437									
→ C	469	479	528	470	630	500	480				
D	465	426	400	370	470						
→ E	559	495	480								
F	418										
G			370	420	460	420	460				
H			390	420							
I			416	430							
J			400	400							
K			435								
L			410	400	430	410	440	420	478	430	
→ M					510						
N					480						
O					470						
→ P					500						
Q						400	420	400	413		
R						440					
S						430	440	460			
T							400	480			
U								430			
V									491		
W									441	440	440
→ X											530
Y											460
Z											390

QTc	Men	Women
Abnormal	>450	>460
Panic	500+	500+

QTc Medications - Monitoring Protocol
developed by: William Parker, Director of Pharmacy, Bayfront Health Dade City
Derek Harmeson, Director of ICU/CPCU
Wayne Ruppert, Cardiovascular Coordinator, Bayfront Health Dade City

Bayfront Health Dade City is a 120 bed community hospital with an accredited chest pain center and an interventional cardiac catheterization program in Dade City, Florida.

Click for link to: "Predicting the Unpredictable; Drug-Induced QT Prolongation and Torsades de Pointes: *J Am Coll Cardiol*. 2016;67(13):1639-1650

Click for link to "AHA ACC Scientific Statement: Prevention of Torsades de Pointes in the Hospital Setting," AHA Circulation 2010;

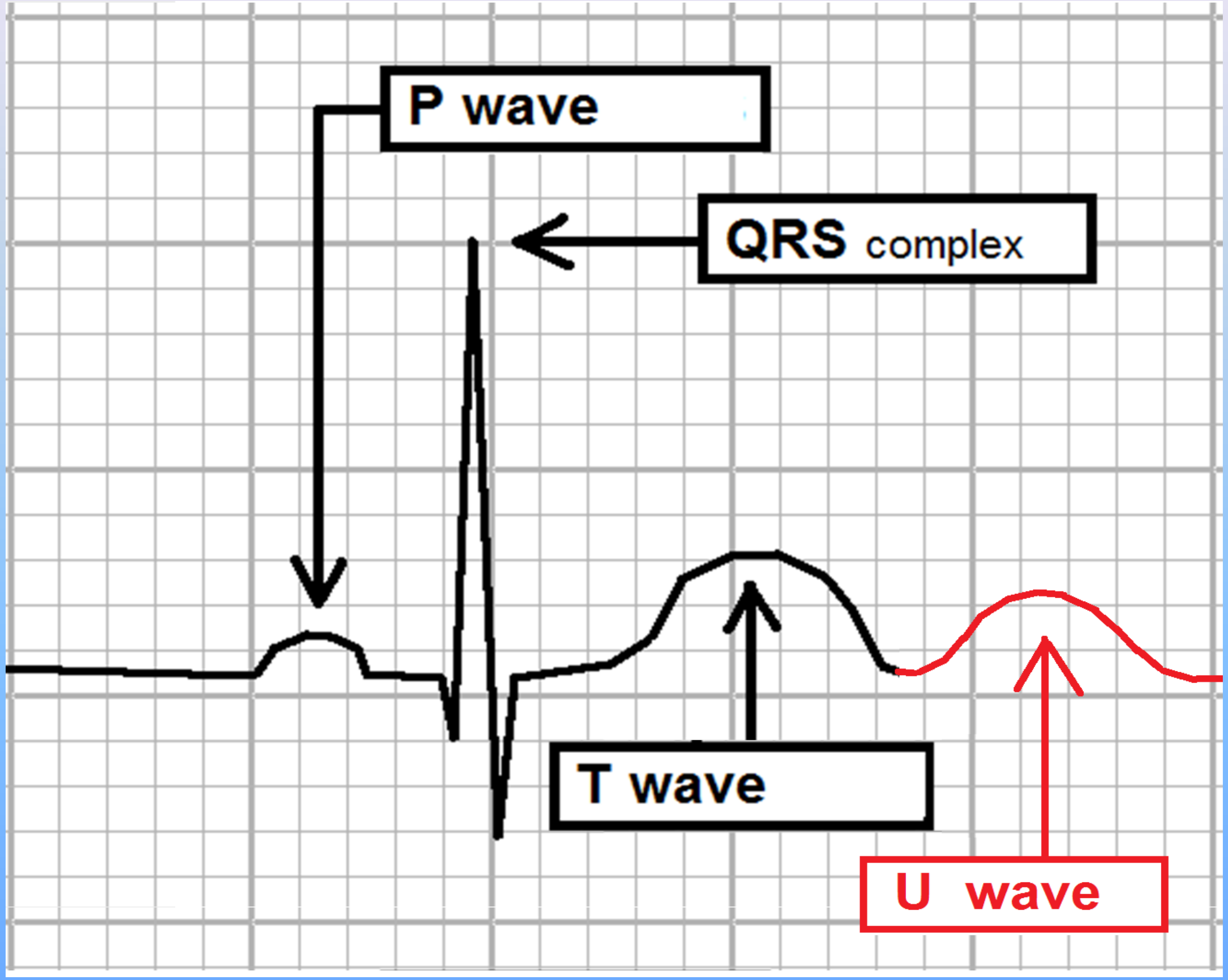
Click for link to hospital model policy & procedure for: "QT Prolonging Medications; QT interval monitoring"

U Waves

Occasionally an extra wave is noted after each T wave.

It typically resembles

"a secondary T wave."



When present on the ECG, this "extra" waveform is referred to as a " U Wave."

U Waves . . .

- Common U wave Etiology:
  - **Hypomagnesemia***
  - **Hypokalemia***
  - **Hypercalcemia***
  - **QT prolonging medications***
  - **Increased intracranial pressure***
  - **Hypothermia***
  - **Digitalis** (usually *shortens* the QT Interval)

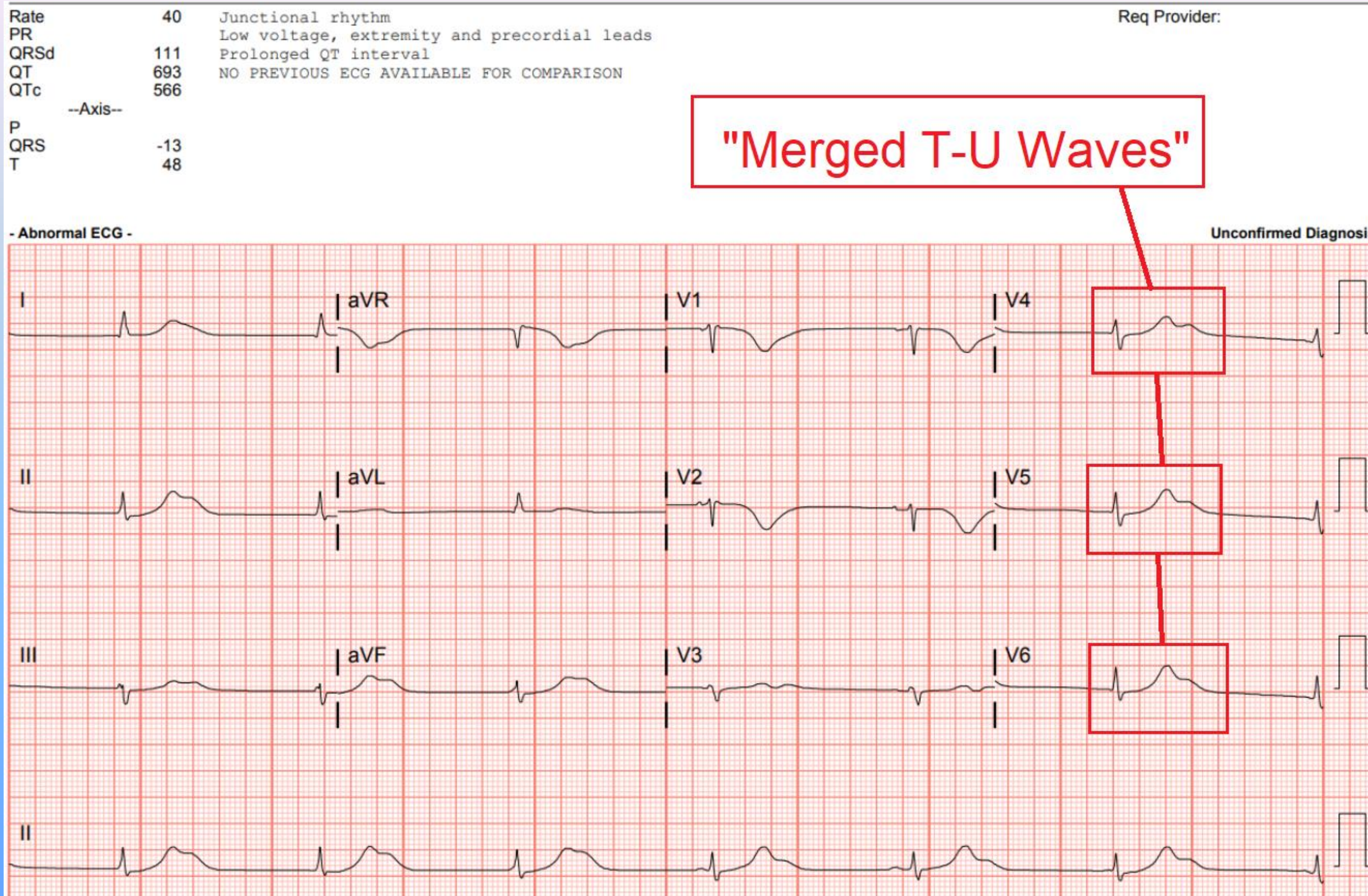
*** *These are also causes of QT interval prolongation.***

Abnormal U Waves

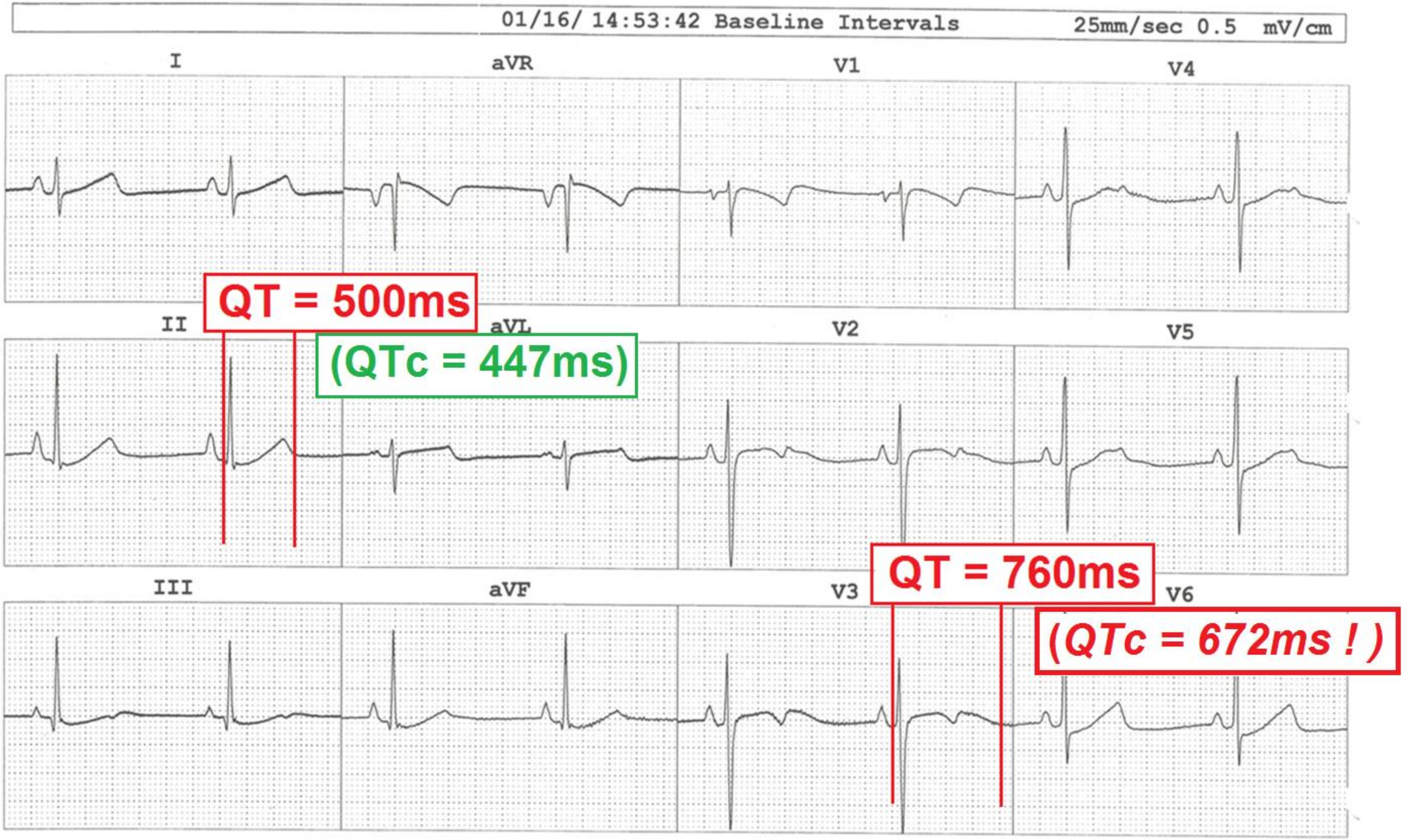
INCLUDE the U Wave in the QT Interval measurement **when any one or more criteria are present:**

- **U wave 100% (or more) the size of the T wave.**
- **U wave is INVERTED (opposite polarity of T wave)**
- **U wave merged with the T wave**

EVIDENCE SOURCE:
ACC/AHA/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram Part IV: The ST Segment, T and U Waves, and the QT Interval.

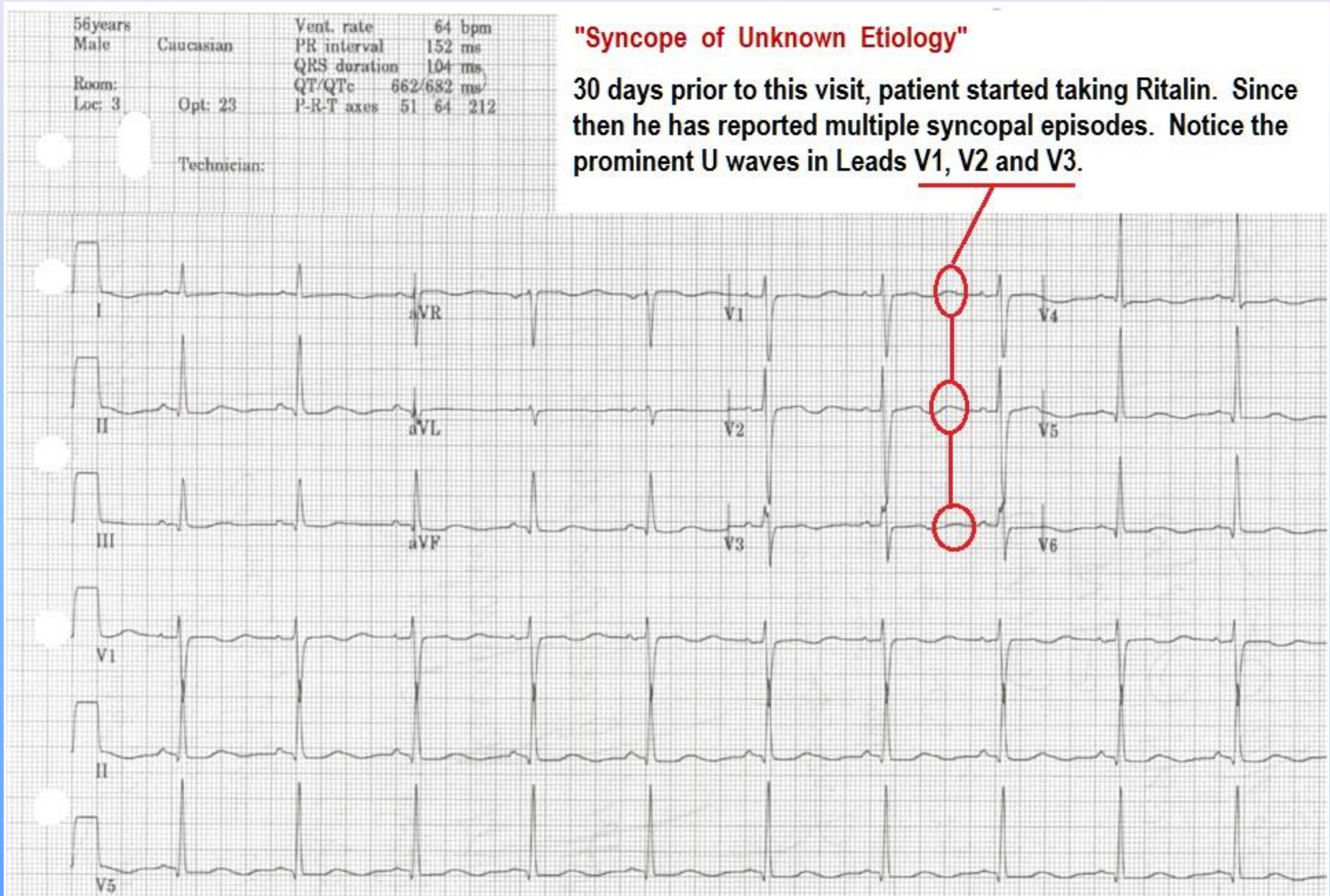


Rate 40
PR
QRSd 111
QT 693
QTc 566
--Axis--
P
QRS -13
T 48
Junctional rhythm
Low voltage, extremity and precordial leads
Prolonged QT interval
NO PREVIOUS ECG AVAILABLE FOR COMPARISON
Req Provider:
"Merged T-U Waves"
- Abnormal ECG -
Unconfirmed Diagnosis
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

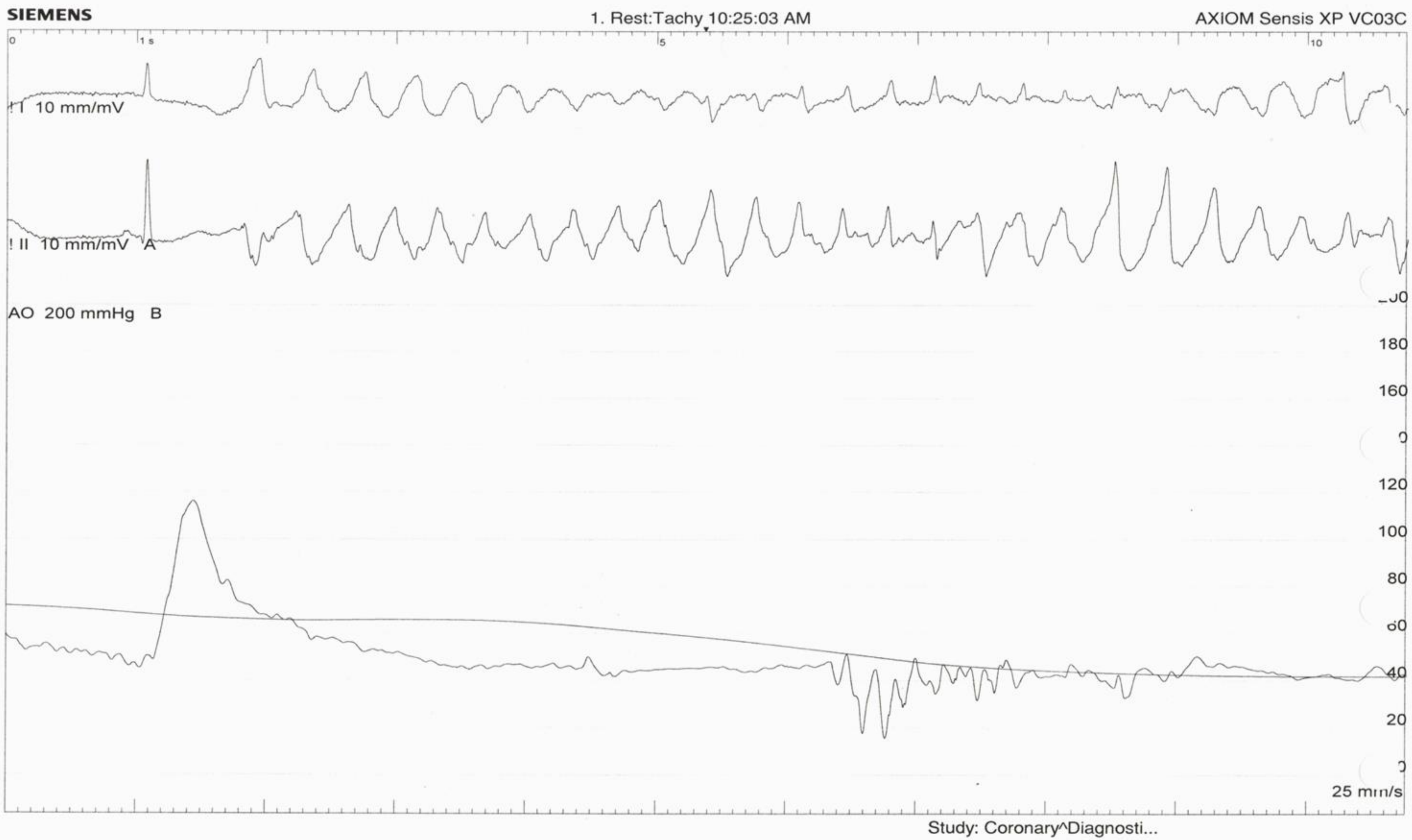


This ECG illustrates the degree of variation that can be noted between different leads on the 12 Lead ECG. ALWAY measure the QT Interval in the lead with the GREATEST value.

Medication induced LQTS with TdP and Cardiac Arrest - Case Study: 56 year old male

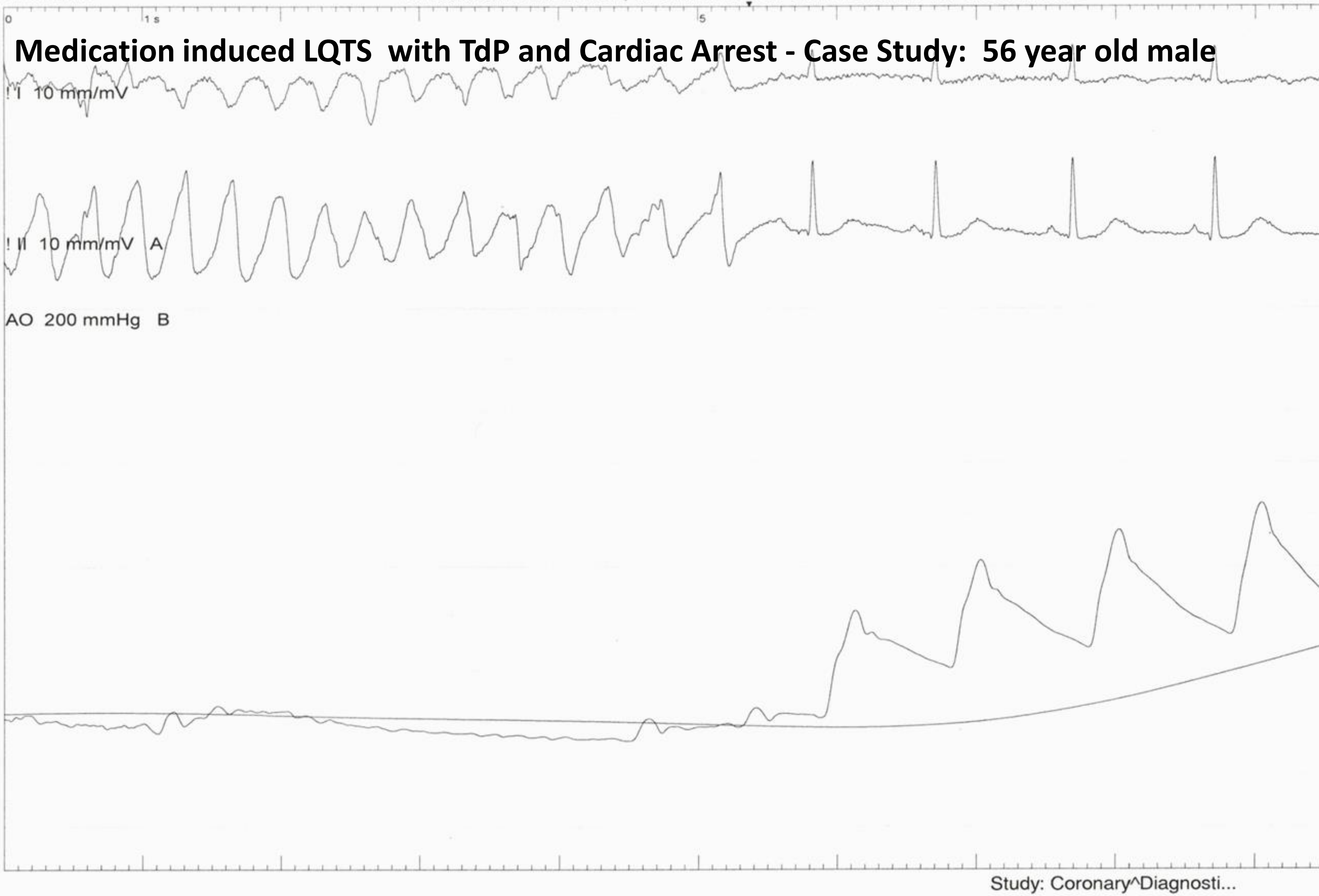


Medication induced LQTS with TdP and Cardiac Arrest - Case Study: 56 year old male



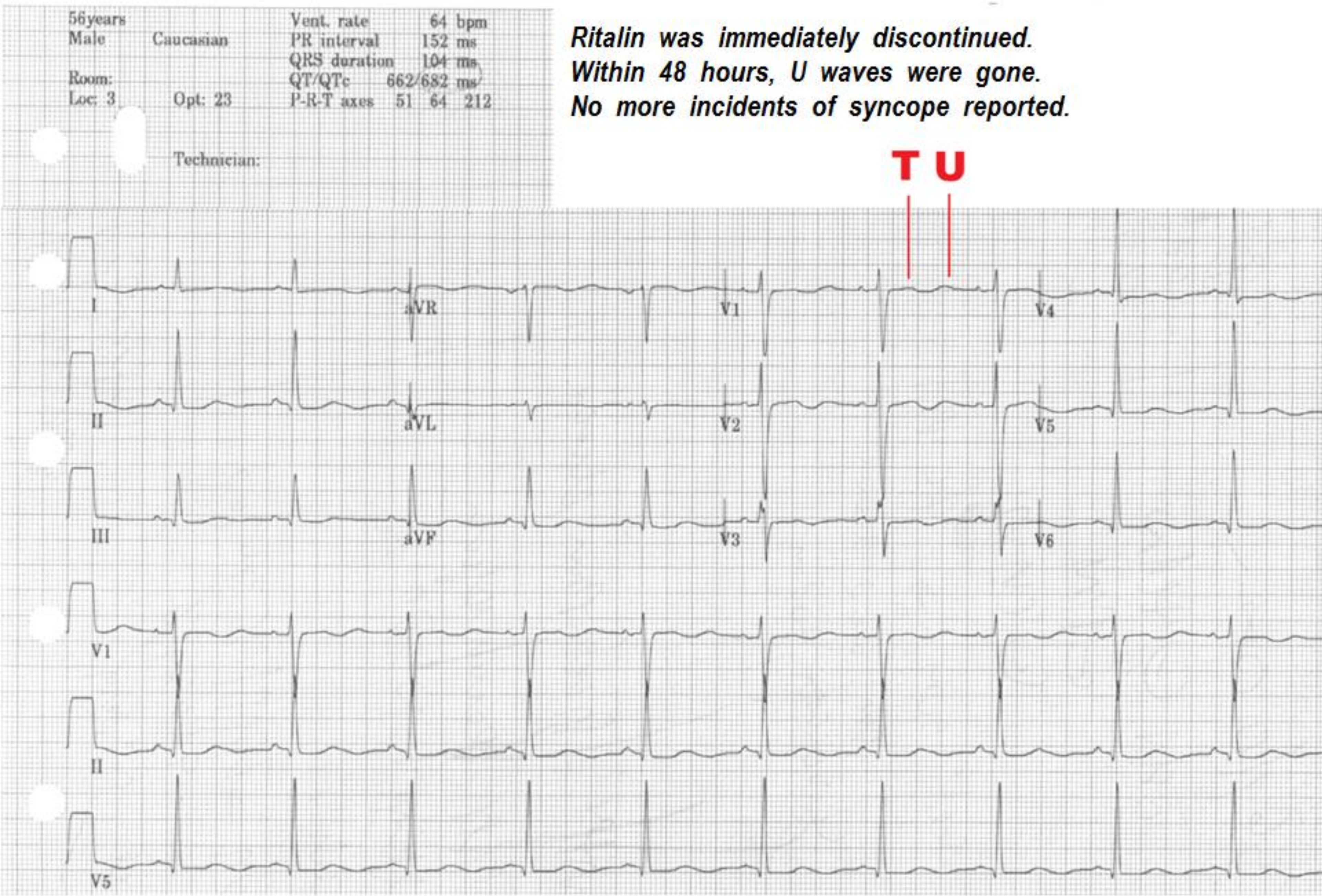
Run of Torsades de Pointes occurred during Cardiac Catheterization . . .

Medication induced LQTS with TdP and Cardiac Arrest - Case Study: 56 year old male



Torsades de Pointes self-terminates just before aborted Defibrillation

Medication induced LQTS with TdP and Cardiac Arrest - Case Study: 56 year old male



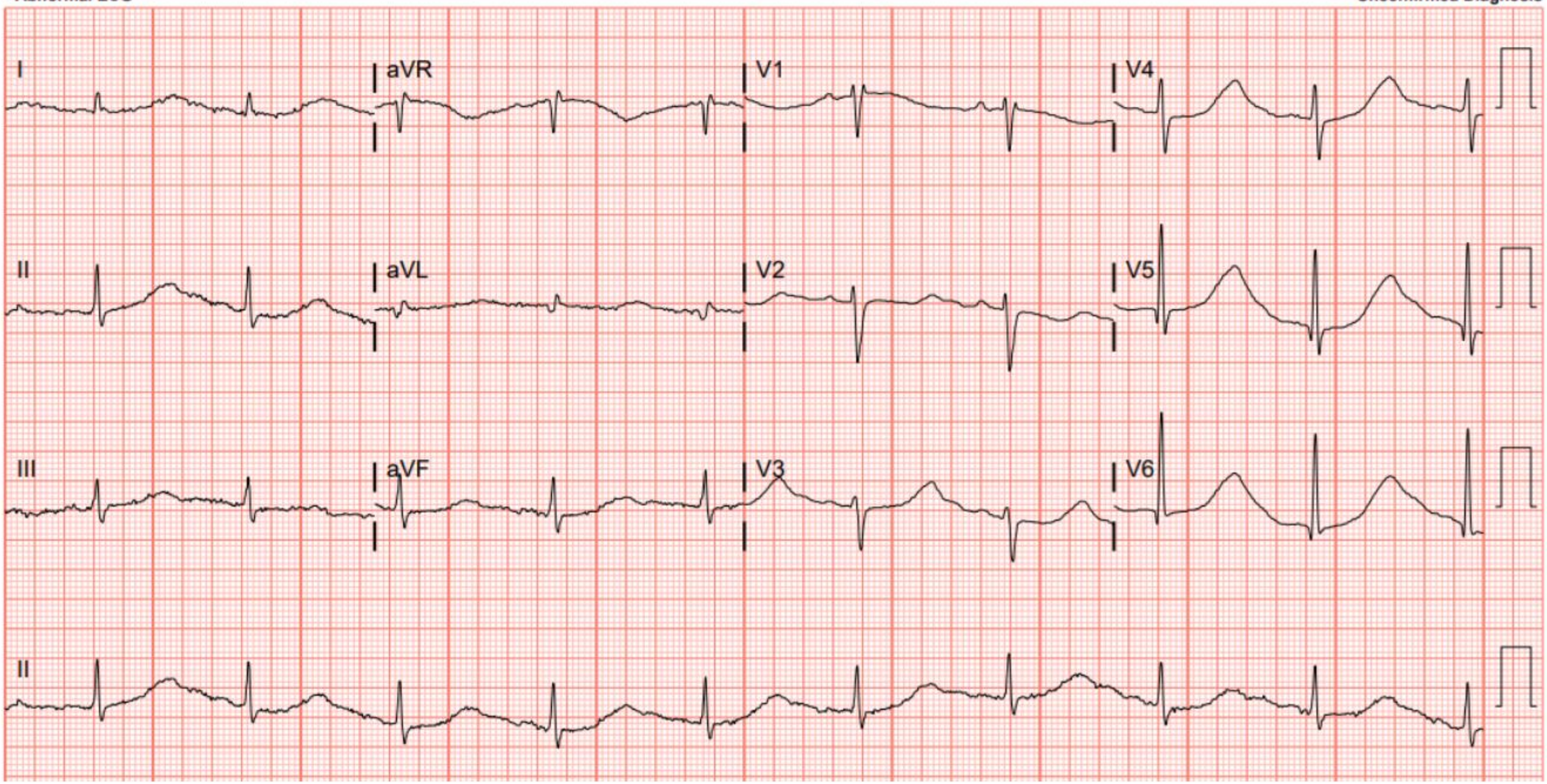
Ritalin was immediately discontinued.
Within 48 hours, U waves were gone.
No more incidents of syncope reported.

Rate	58	Sinus rhythm
PR	185	IVCD, consider atypical RBBB
QRSd	126	Baseline wander in lead(s) V2,V3,V4,V6
QT	668	COMPARED TO ECG 07/22/2020 16:56:59
QTc	657	SINUS RHYTHM NOW PRESENT
--Axis--		
P	107	
QRS	61	
T	45	

Req Provider: Rafael Santiago-Aponte

- Abnormal ECG -

Unconfirmed Diagnosis

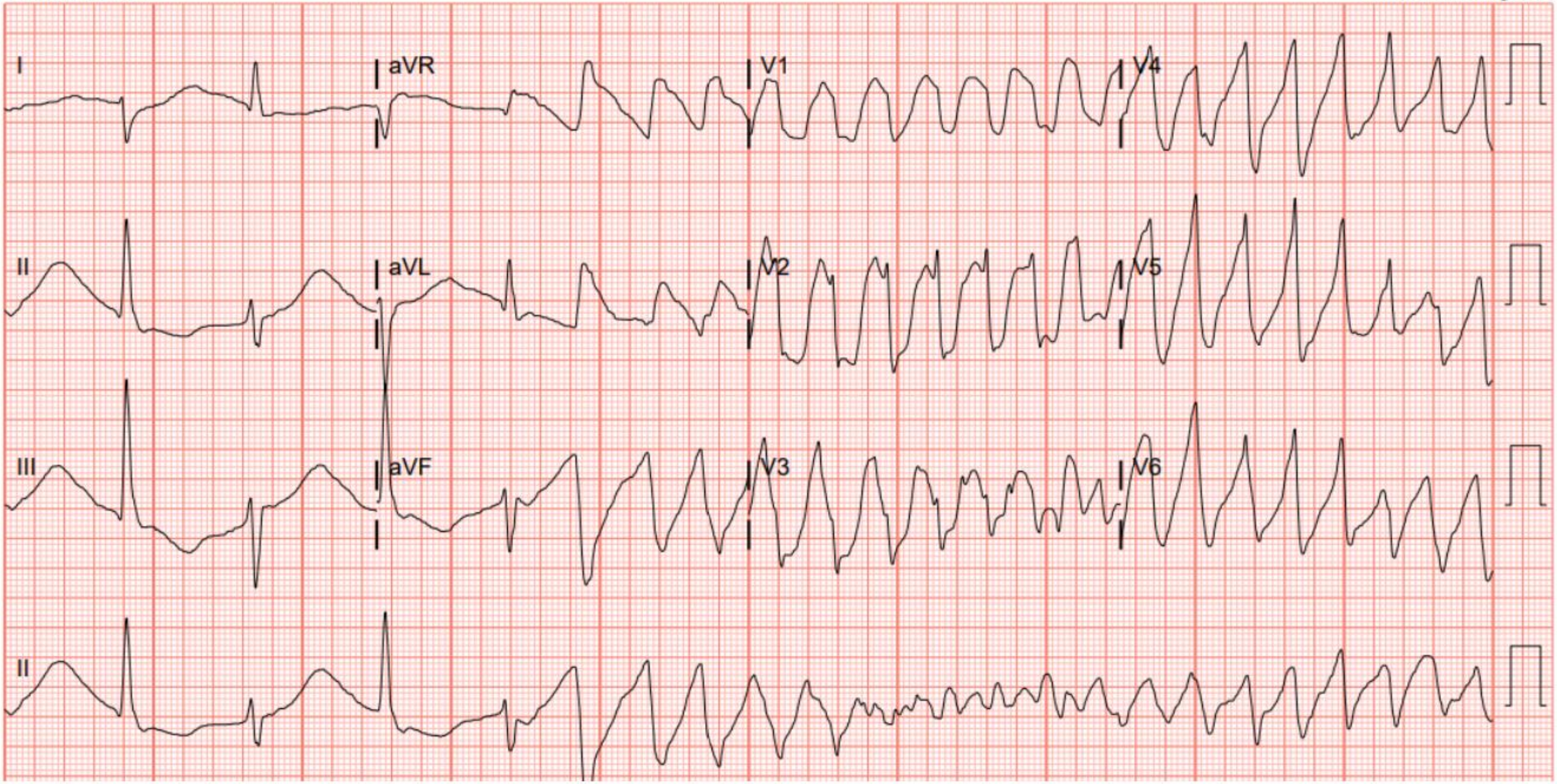


Rate	156	Atrial fibrillation
PR		Ventricular tachycardia, unsustained
QRSd	138	RBBB and LPFB
QT	456	Repol abnrm suggests ischemia, diffuse leads
QTc	735	Baseline wander in lead(s) II,III,aVR,aVF,V1,V2,V3,V4
--Axis--		COMPARED TO ECG 07/22/2020 15:32:52
P		ATRIAL FIBRILLATION NOW PRESENT
QRS	102	VENTRICULAR TACHYCARDIA NOW PRESENT
T	185	LEFT POSTERIOR FASCICULAR BLOCK NOW PRESENT
		RIGHT BUNDLE-BRANCH BLOCK NOW PRESENT
		POSSIBLE ISCHEMIA NOW PRESENT
		PROLONGED QT INTERVAL NO LONGER PRESENT

Req Provider: Rafael Santiago-Aponte

- Abnormal ECG -

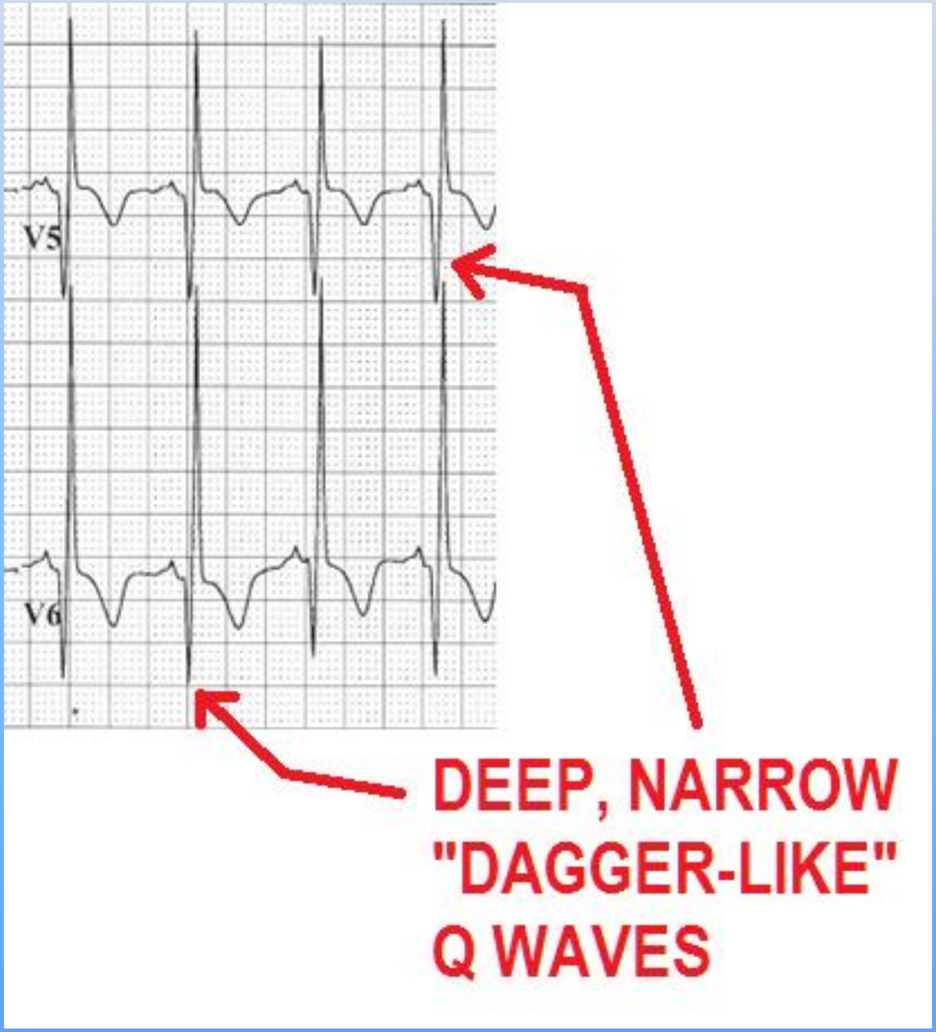
Unconfirmed Diagnosis



ECG Indicators: Hypertrophic Cardiomyopathy

- ECG may be normal
- Deep, narrow (dagger-like) Q waves

ECG Indicators: Hypertrophic Cardiomyopathy



ECG Indicators: Hypertrophic Cardiomyopathy

- ECG may be normal
- Deep, narrow (dagger-like) Q waves
- Inverted T waves in multiple regions
- Left Ventricular and possibly Left Atrial Hypertrophy

Hypertrophic Cardiomyopathy (HCM)

12 Lead ECG Traits:

- **QRS Height -- exceeds normal size, "spearing through QRS" in other leads**
- **Inverted T waves appear in multiple regions (ANTERIOR, LATERAL)**
- **BiPHASIC T waves in Inferior Leads.**
- **T WAVES are SYMMETRICAL .**

ECG Indicators: Brugada Syndrome

IS THERE ANYTHING ABNORMAL WITH THIS EKG ?

37 yr
Female Caucasian

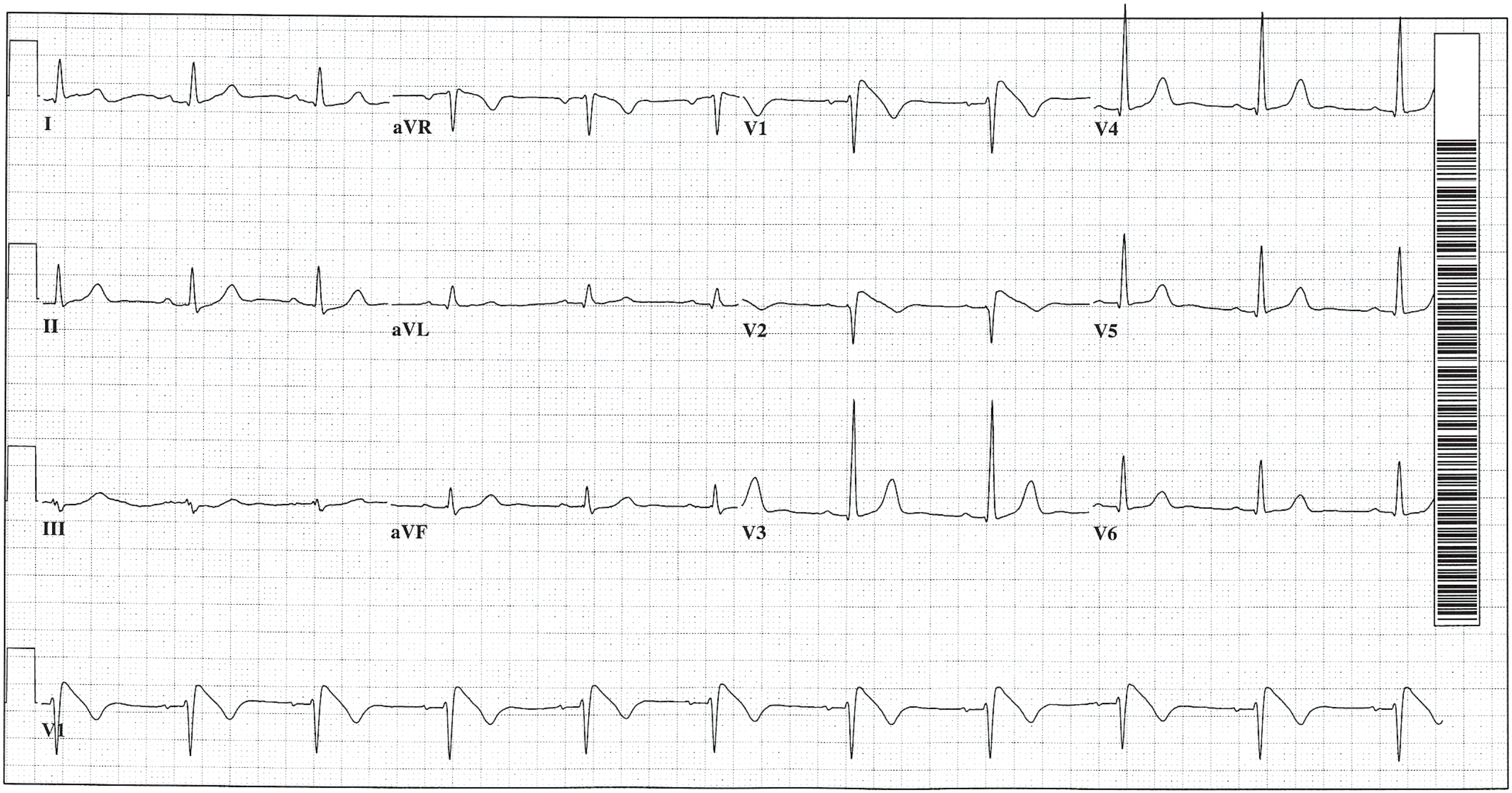
Room:C4A
Loc:3 Option:23

Vent. rate	62	BPM
PR interval	180	ms
QRS duration	88	ms
QT/QTc	418/424	ms
P–R–T axes	37 22	47

Normal sinus rhythm
Normal ECG
No previous ECGs available

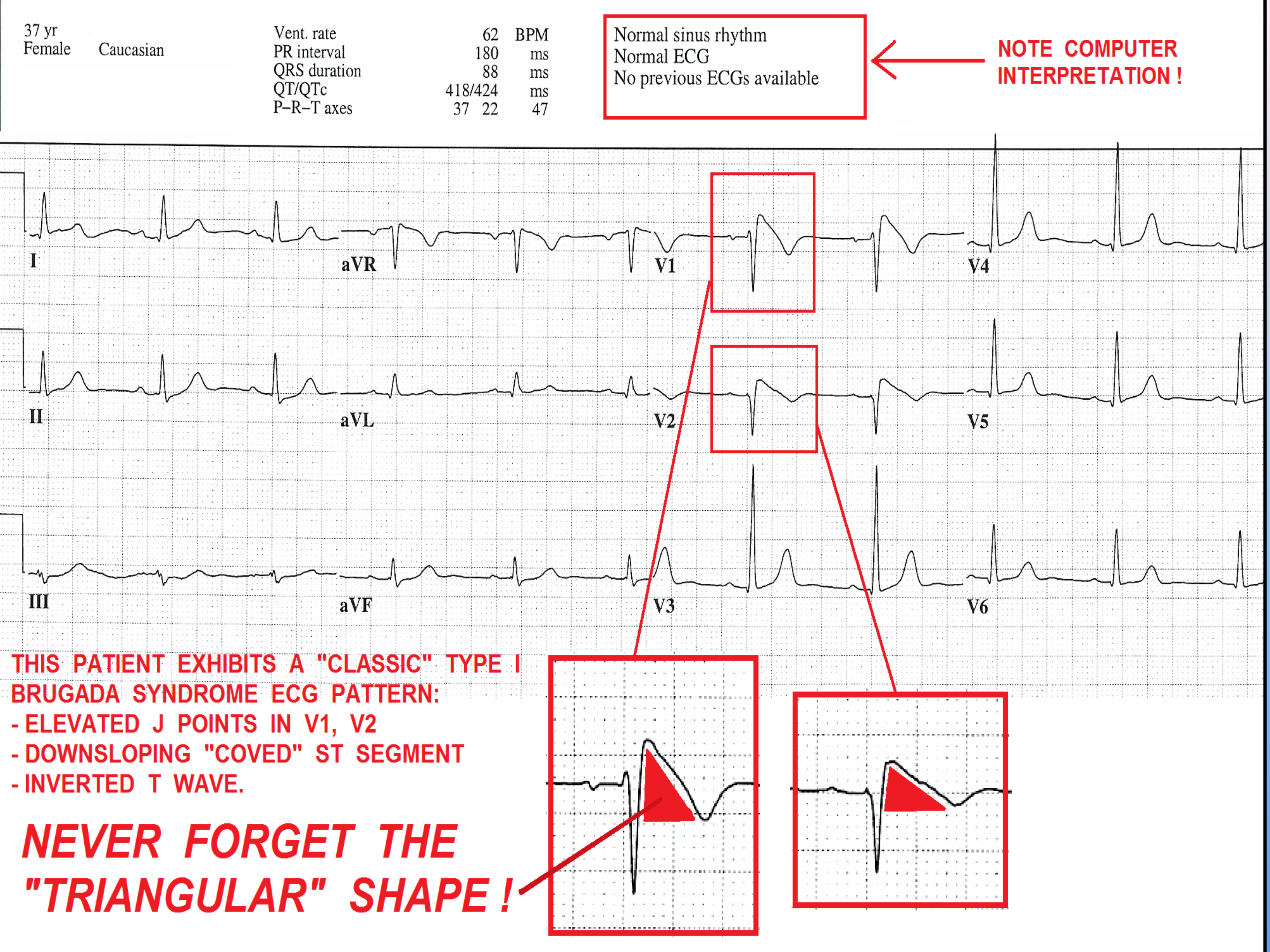
Technician:

Referred by:

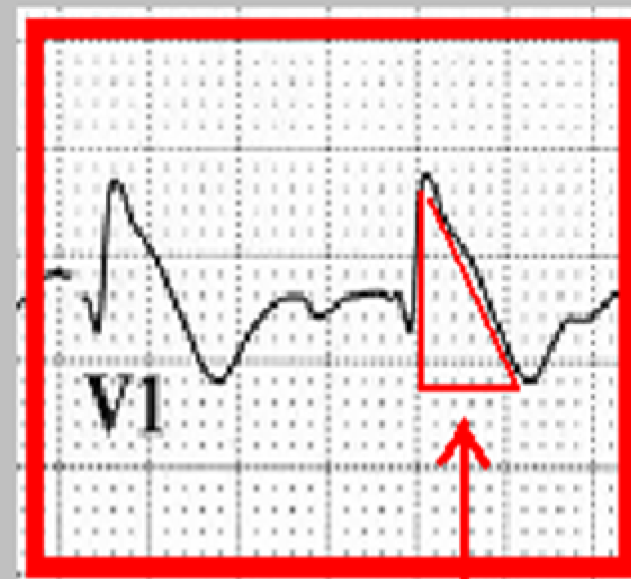


25mm/s 10mm/mV 40Hz 005D 12SL 235 CID: 3

EID:46 EDT:


37 yr
Female Caucasian
Vent. rate 62 BPM
PR interval 180 ms
QRS duration 88 ms
QT/QTc 418/424 ms
P–R–T axes 37 22 47
Normal sinus rhythm
Normal ECG
No previous ECGs available
NOTE COMPUTER INTERPRETATION !
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
THIS PATIENT EXHIBITS A "CLASSIC" TYPE I BRUGADA SYNDROME ECG PATTERN:
- ELEVATED J POINTS IN V1, V2
- DOWNSLOPING "COVED" ST SEGMENT
- INVERTED T WAVE.
NEVER FORGET THE "TRIANGULAR" SHAPE !

BRUGADA SYNDROME



1. RBBB PATTERN
2. J POINT ELEVATION V1, V2 and possibly V3
3. DOWNWARD SLOPING S-T SEGMENT
4. INVERTED T WAVE
5. GIVES S-T SEGMENT A "TRIANGULAR" APPEARANCE

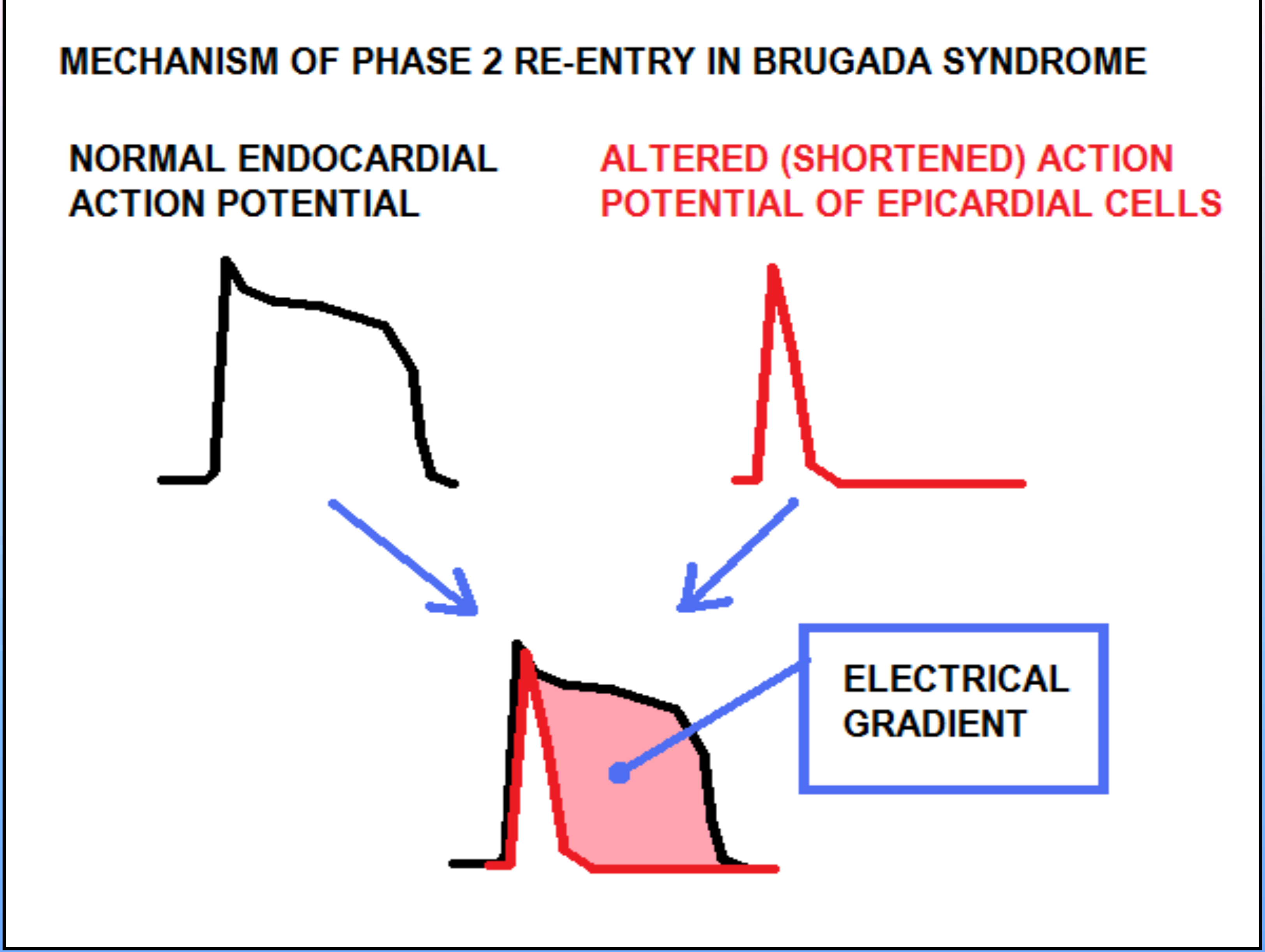
PATTERNS of S-T ELEVATION :

BEWARE of the

**" TRIANGULAR "
SHAPED S-T SEGMENT
IN V1, V2, and some-
times also in V3 . . .**

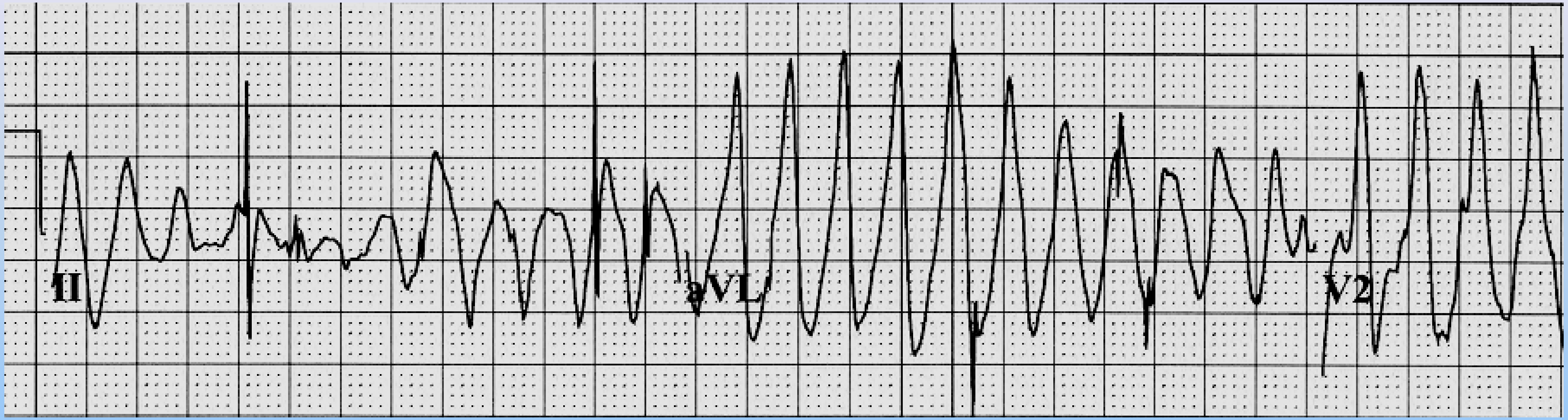
THINK - -

BRUGADA SYNDROME



Trigger for Torsades de Pointes – ECTOPIC BEAT during The "ELECTRICAL GRADIENT" phase shown above.

Brugada / Long QT Syndromes cause:



Torsades de Pointes:

- **Decreased – to – NO Cardiac Output**
- **Often patient PULSELESS during episode**
- **Causes SYNCOPE**
- **Often DETERIORATES into VENTRICULAR FIBRILLATION and CARDIAC ARREST.**

TREATMENT OF TORSADES de POINTES per AHA ACLS 2015:

-TRANSIENT: MAGNESIUM SULFATE 1 – 2 gm IV infusion over 5 – 60 minutes.

-PERSISTENT, PATIENT UNSTABLE: DEFIBRILLATION

-CARDIAC ARREST: FOLLOW Ventricular Fibrillation Algorithm. Consider Mag Sulfate as your Antiarrhythmic of choice.

WIDE COMPLEX TACHYCARDIA
TORSADES de POINTES

(QRS > 120 ms)

ABC s

NO PULSE	PULSE - UNSTABLE	PULSE - STABLE
GO TO V - FIB ALGORITHM !	▪ IMMEDIATE DEFIBRILLATION START IV CONSIDER SEDATION	▪ O2 / IV / EKG ▪ MAGNESIUM INFUSION 1 - 2 gm OVER 5 - 60 min,

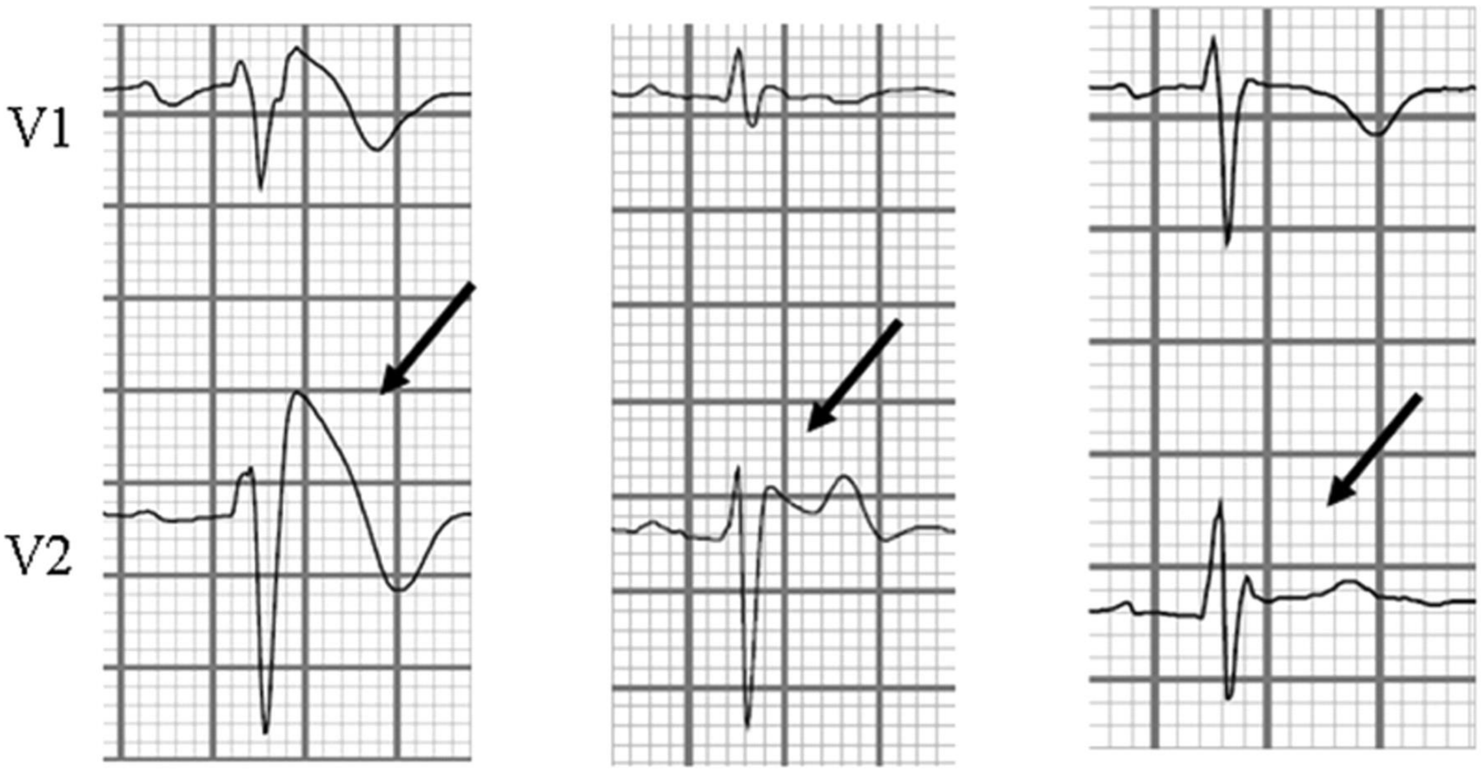
DO NOT give PROCAINAMIDE, AMIODARONE, or SOTALOL to patients with TORSADES or POLYMORPHIC VT !!!

OTHER CONSIDERATIONS:

- EVALUATE BASELINE ECG RHYTHM FOR PRONGED Q-T INVERVAL.
- EVALUATE PATIENT'S MEDS FOR Q-T PROLONGING DRUGS
  . . . if PATIENT HAS BEEN RECEIVING ANY Q-T PROLONGING DRUGS, IMMEDIATELY DISCONTINUE AND CONTACT PHYSICIAN STAT.
- EVALUATE PATIENT HISTORY FOR PREVIOUS EVENTS OF "SYNCOPE OF UNKOWN ETIOLOGY"
- EVALUATE PATIENT FOR FAMILY HISTORY FOR SUDDEN CARDIAC DEATH

REPORT ANY ABNORMAL FINDINGS TO PHYSICIAN.

ECG abnormality diagnostic or suspected of Brugada syndrome.



Type 1:
Coved type
ST-segment
elevation

Type 2:
saddle-back type
ST-segment
elevation

Type 3:
Saddle-back type
“ST-segment
elevation”

Yuka Mizusawa, and Arthur A.M. Wilde Circ Arrhythm Electrophysiol. 2012;5:606-616

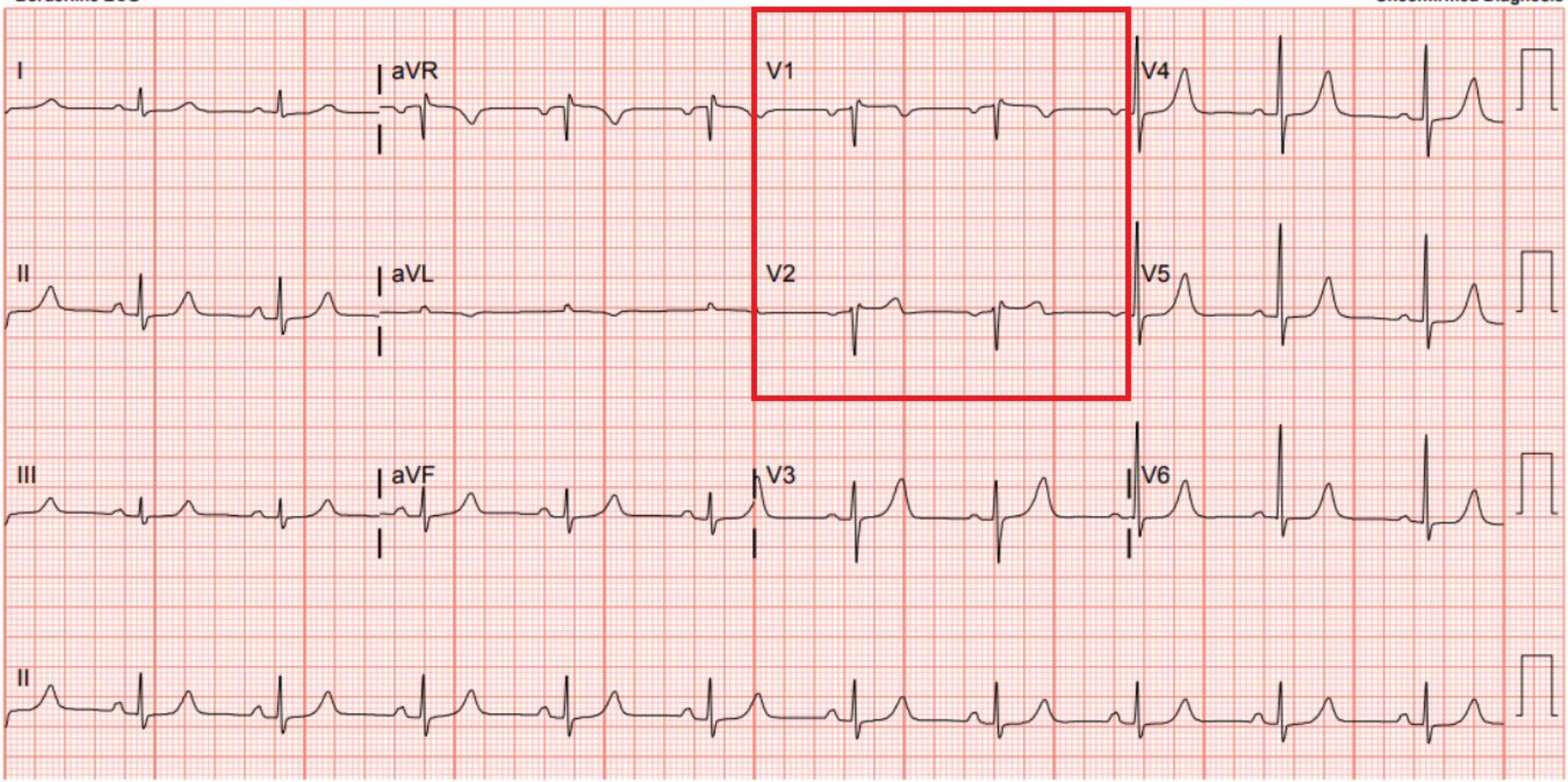
Rate	63
PR	168
QRSd	85
QT	440
QTc	451
--Axis--	
P	66
QRS	27
T	67

Sinus rhythm
Probable left atrial enlargement
RSR' in V1 or V2, right VCD or RVH
COMPARED TO ECG 09/27/2019 02:43:44
RIGHT VENTRICULAR HYPERTROPHY NOW PRESENT

Req Provider: R POTU

Brugada Syndrome: Type 2 ECG
Waveforms: "Saddleback" ST-T Waves
V1 & V2

- Borderline ECG - **Unconfirmed Diagnosis**



For those who think *"Brugada Syndrome? – that kind of stuff doesn't happen here"……….*

Pat ID [redacted] 10/04/2019 10:56:31 [redacted]
[redacted]
Female
Account #
RX
DX

Bayfront Health Seven Rivers ED
Dept
Room
Tech tb

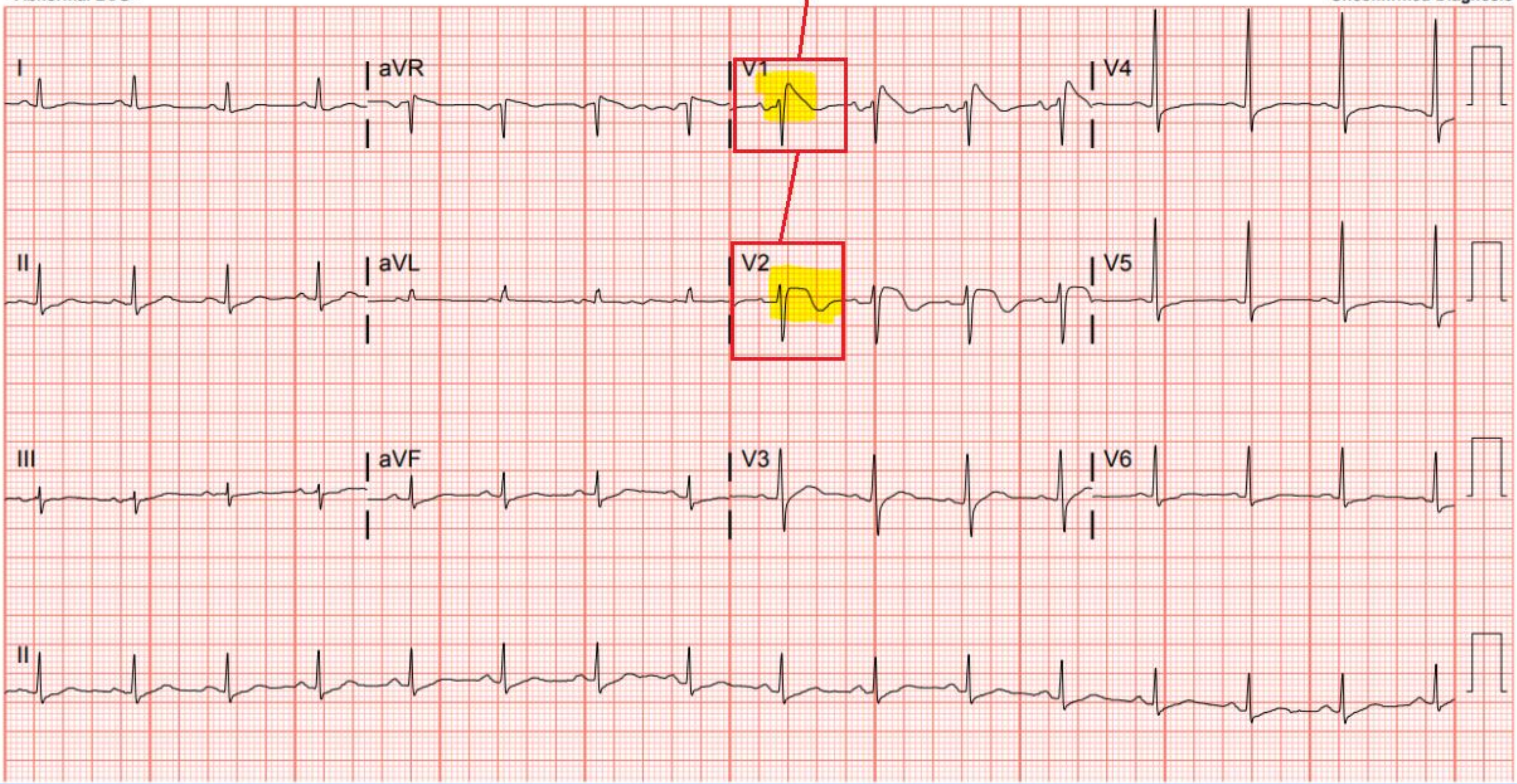
Rate	93	Sinus rhythm
PR	150	Probable left atrial enlargement
QRSd	66	Anteroseptal infarct, acute
QT	419	Prolonged QT interval
QTc	522	Baseline wander in lead(s) II,III,aVR,aVL,aVF
--Axis--		COMPARED TO ECG 10/04/2019 10:50:56
P	42	PROLONGED QT INTERVAL NOW PRESENT
QRS	6	
T	47	

Req Provider:

Notice the ECG computer has no idea what it's looking at . . .

- Abnormal ECG -

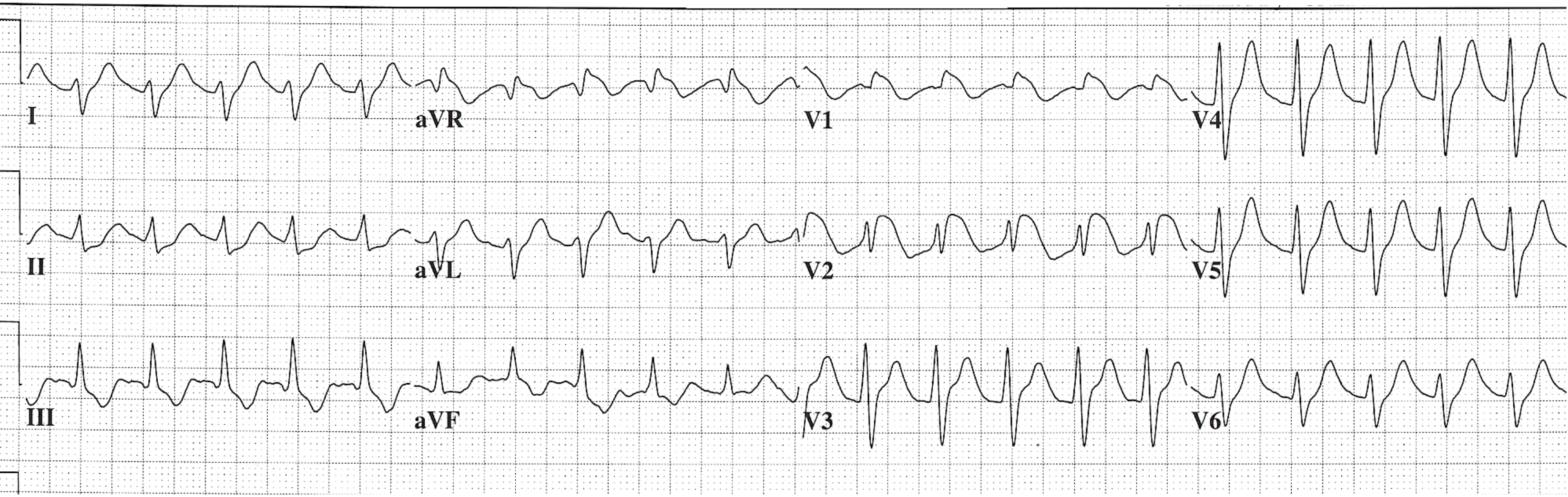
Unconfirmed Diagnosis



33 y/o FEMALE

Vent. rate	129	BPM
PR interval	*	ms
QRS duration	112	ms
QT/QTc	398/583	ms
P–R–T axes	* 121	–2

Undetermined rhythm
Incomplete right bundle branch block
Right ventricular hypertrophy
ST elevation consider anterior injury or acute infarct
** ** ** ** * ACUTE MI * ** ** ** **
Abnormal ECG
No previous ECGs available



PT. BROUGHT TO EMERGENCY DEPARTMENT BY EMS AFTER SUFFERING SPONTANEOUS CARDIAC ARREST. PATIENT DID NOT EXPERIENCE ANY SYMPTOMS PRIOR TO COLLAPSE. HAD SEVERAL EPISODES OF NEAR-SYNCOPE IN THE PAST 10 YEARS. CARDIAC CATHETERIZATION REVEALED NO EVIDENCE OF CARDIOVASCULAR DISEASE. NORMAL LV FUNCTION.

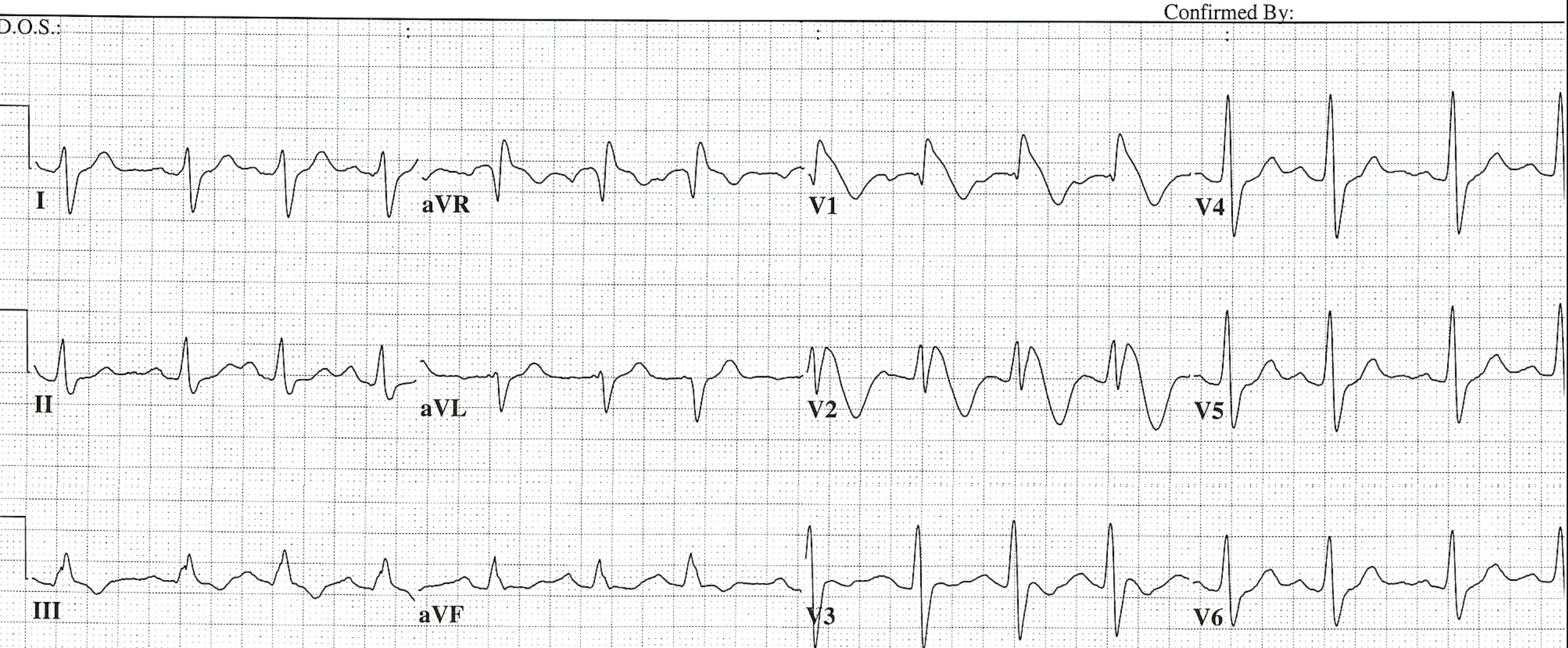
DIAGNOSIS: BRUGADA SYNDROME. PT. RECEIVED ICD PRIOR TO HOSPITAL DISCHARGE.

VISIT: www.BRUGADA.org FOR MORE INFORMATION.

42 y/o FEMALE

Vent. rate	86	BPM
PR interval	200	ms
QRS duration	148	ms
QT/QTc	414/495	ms
P–R–T axes	64 114	17

Normal sinus rhythm with sinus arrhythmia
Right bundle branch block
ST elevation consider anterior injury or acute infarct
** ** ** ** * ACUTE MI * ** ** ** **
Abnormal ECG
No previous ECGs available

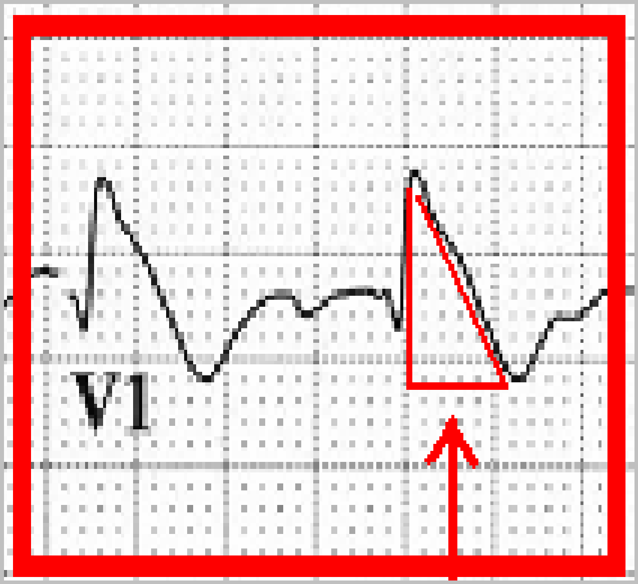


BRUGADA SYNDROME.

PATIENT HAD HISTORY of SYNCOPE of UNKNOWN ETIOLOGY.
FAMILY HISTORY of SUDDEN DEATH of YOUNG, HEALTHY ADULTS.
VISIT: www.BRUGADA.org FOR MORE INFORMATION !

THE ONLY KNOWN TREATMENT FOR BRUGADA SYNDROME is IMPLANTATION of an ICD. THIS PATIENT HAD ICD IMPLANTED PRIOR TO HOSPITAL DISCHARGE.

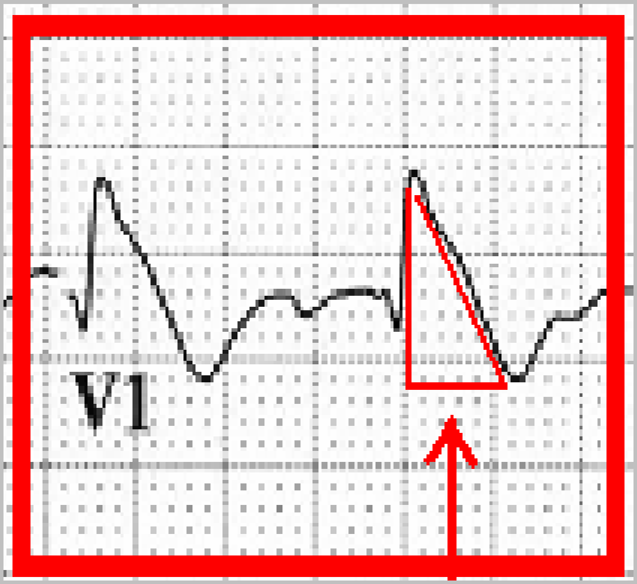
BRUGADA SYNDROME



- GENETIC DISORDER - GENE SCN5A, which encodes CARDIAC SODIUM CHANNELS.
- CAUSES EARLY RIGHT VENTRICULAR SUB-EPICARDIAL REPOLARIZATION
- CAUSES RUNS OF TORSADES de POINTES, and SUDDEN DEATH from TORSADES and V-FIB.
- IS BELIEVED TO CAUSE 4 - 12 % of ALL SUDDEN DEATHS, and 50 % of ALL CARDIAC DEATHS where pt. has a STRUCTUALLY NORMAL HEART.

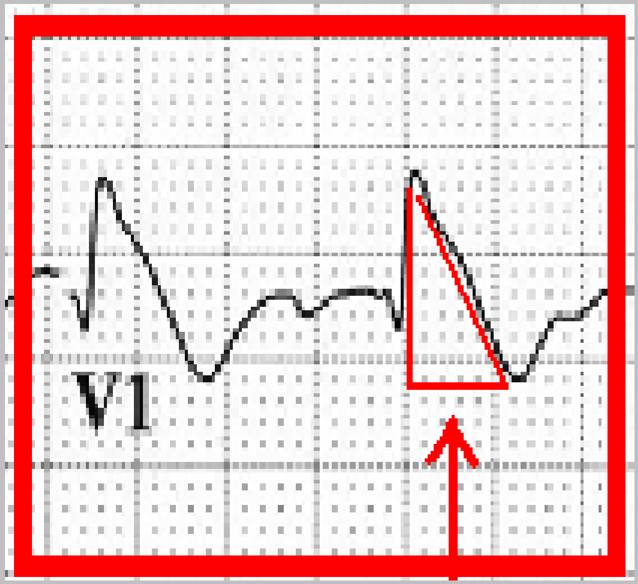
BRUGADA SYNDROME

- SEVERAL VARIATIONS of this disorder are known to exist.
- CONCEALED and NON-CONCEALED.
- The NON-CONCEALED version HAS THE V1-V3 abnormality VISIBLE at all times.



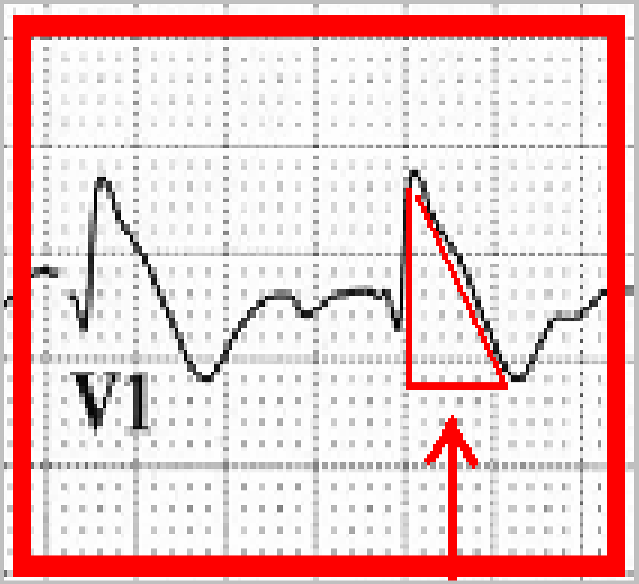
- The CONCEALED version - pt. has a NORMAL EKG at most times - a DRUG STUDY, an EP STUDY, and / or GENETIC TESTING must be done to rule out or confirm diagnosis.

BRUGADA SYNDROME



- YOUNG MALES of SOUTHEAST ASIAN DESCENT are in HIGH RISK GROUP, however this disorder affects ANY RACE or GENDER.
- BRUGADA SYNDROME is HEREDITARY.
- SUSPECT BRUGADA SYNDROME in patients with FAMILY HISTORY of BRUGADA / SUDDEN DEATH, and/or TORSADES.

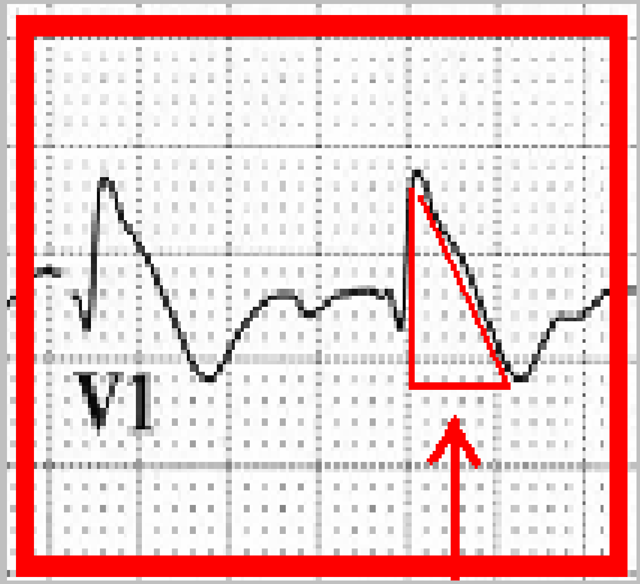
BRUGADA SYNDROME - TESTING



- For CONCEALED cases, a drug study of AJMALINE, FLECAINIDE, or PROCAINAMIDE can UNMASK the "tell-tale" TRIANGULAR COMPLEXES of V1 and V2.
- IN EP STUDIES, a PROLONGED H-V INTERVAL may be observed.
- GENETIC TESTING is performed by THE RAMON A. BRUGADA FOUNDATION.

BRUGADA SYNDROME - TREATMENT

ICD implantation is the only known effective treatment to date.



www.BRUGADA.org

Arrhythmogenic Right Ventricular Dysplasia

- **A genetically acquired myocardial disease associated with paroxysmal ventricular arrhythmias and sudden cardiac death.**

- **Characterized pathologically by fibro-fatty replacement of the right ventricular myocardium.**

- **The second most common cause of sudden cardiac death in young people (after HOCM), causing *up to 20% of sudden cardiac deaths in patients < 35 yrs of age*.**

- **Typically inherited as an autosomal dominant trait, with variable penetrance and expression (there is an autosomal recessive form calledNaxos Disease, which is associated with woolly hair and skin changes).**

- **More common in men than women (3:1) and in people of Italian or Greek descent.**

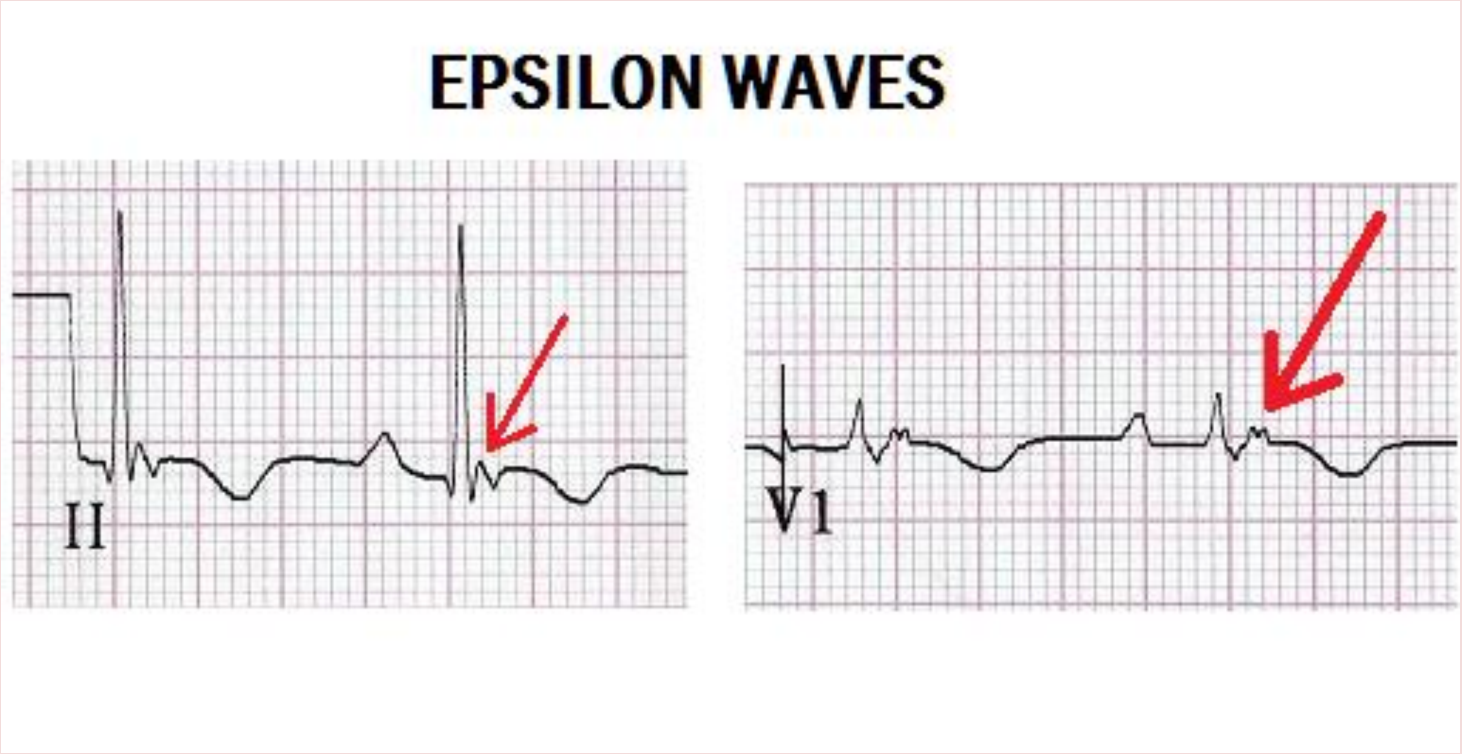
- **Estimated to affect approximately 1 in 5,000 people overall.**

From: 2014 ACC/AHA Guideline on Perioperative Cardiovascular Evaluation and Management of Patients Undergoing Noncardiac Surgery: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

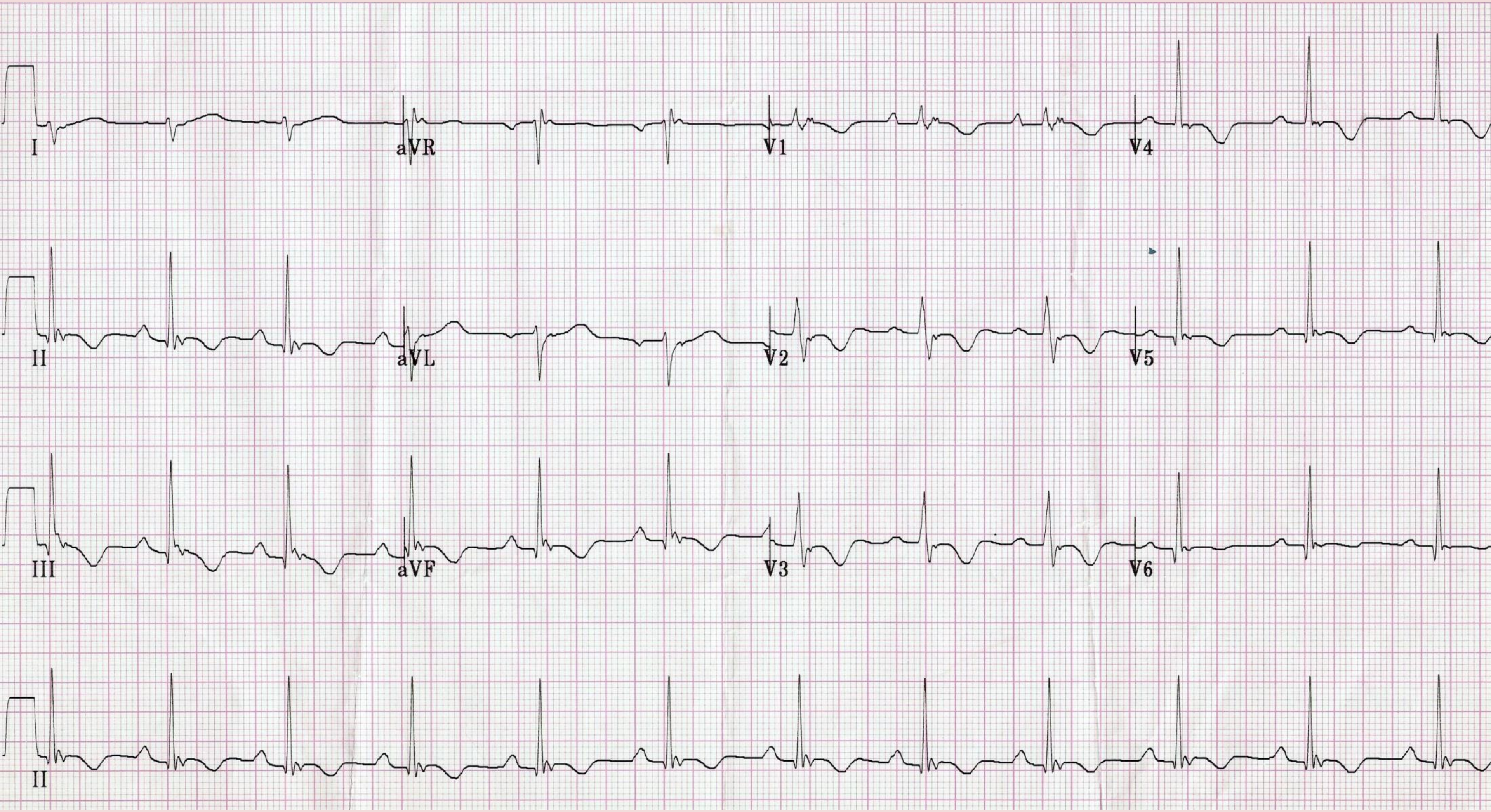
Arrhythmogenic Right Ventricular (RV) Cardiomyopathy and/or Dysplasia:

In 1 autopsy study examining a series of 200 cases of sudden death associated with arrhythmogenic RV cardiomyopathy and/or dysplasia, death occurred in 9.5% of cases during the perioperative period. This emphasizes the importance of close perioperative evaluation and monitoring of these patients for ventricular arrhythmia. Most of these patients require cardiac electrophysiologist involvement and consideration for an implantable cardioverter-defibrillator (ICD) for long-term management.

ARVD – 12 Lead ECG Indicators

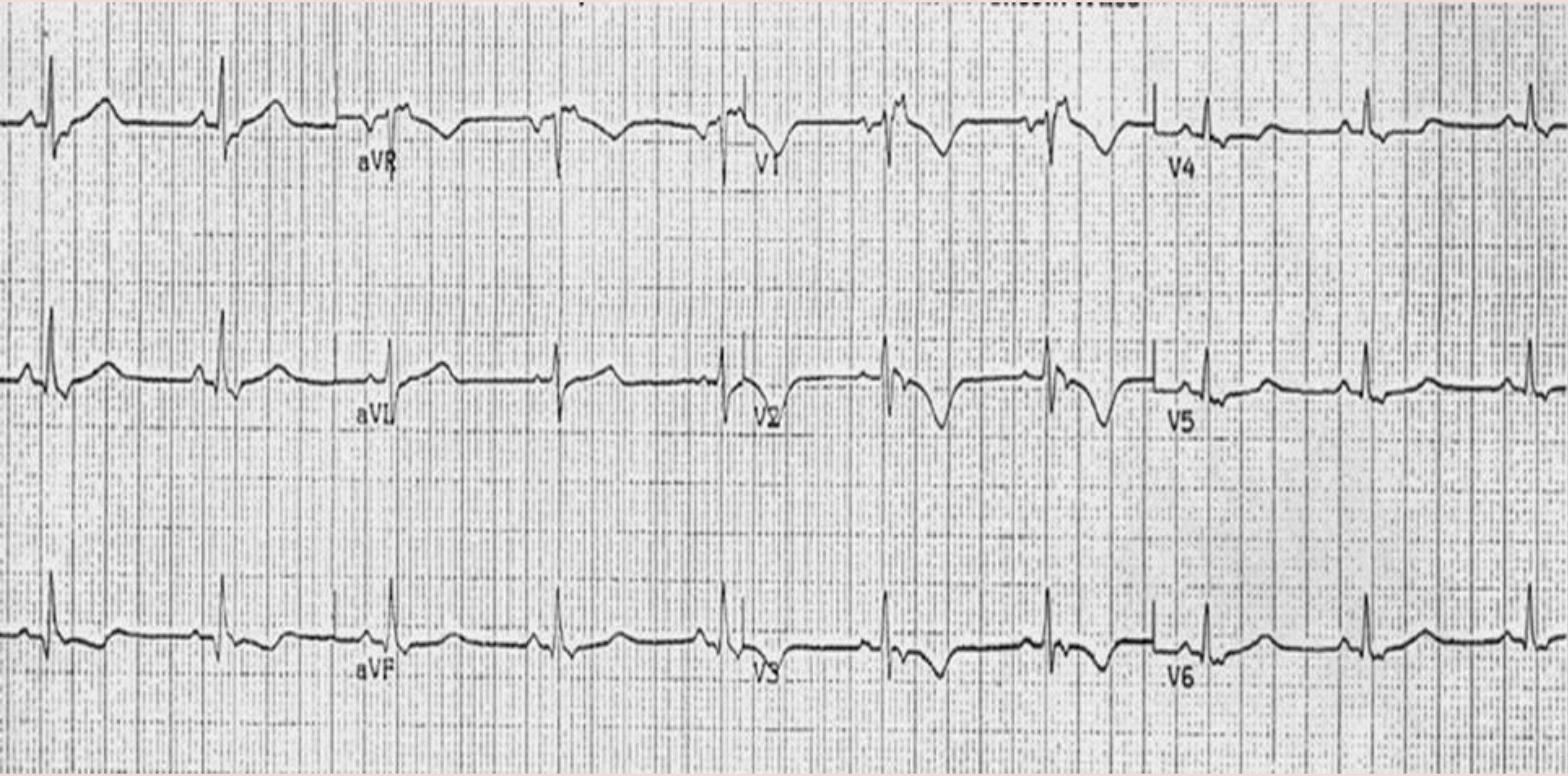


ARVD ECG 1



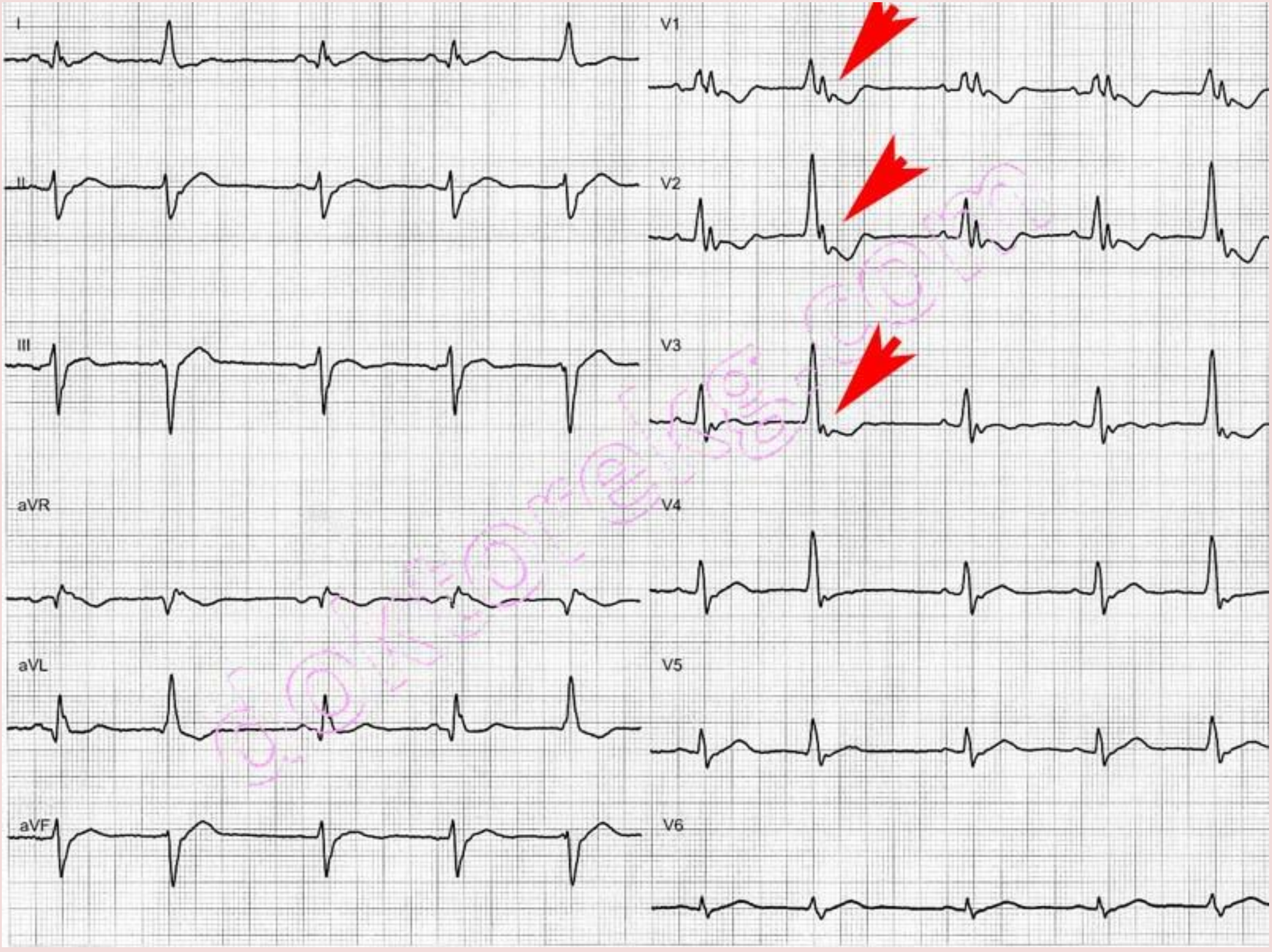
1. “Incomplete RBBB” Pattern
2. V1, V2 Rs pattern
3. Inverted T waves, symmetrical, - Global
4. Epsilon’s waves

ARVD ECG 2



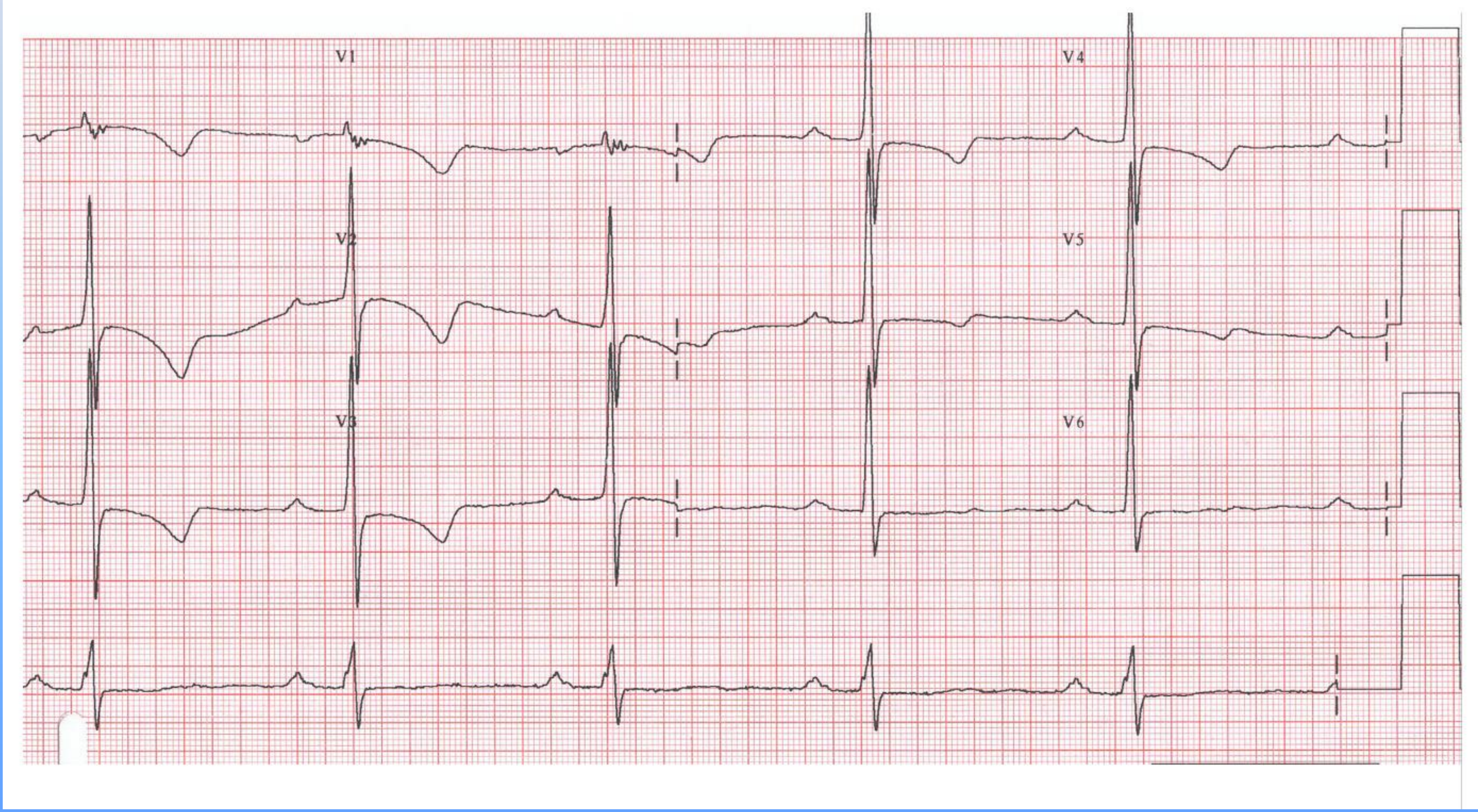
1. "Incomplete RBBB" Pattern
2. V1, V2 Rs pattern
3. Inverted T waves, symmetrical, - Global

4. Epsilon's waves

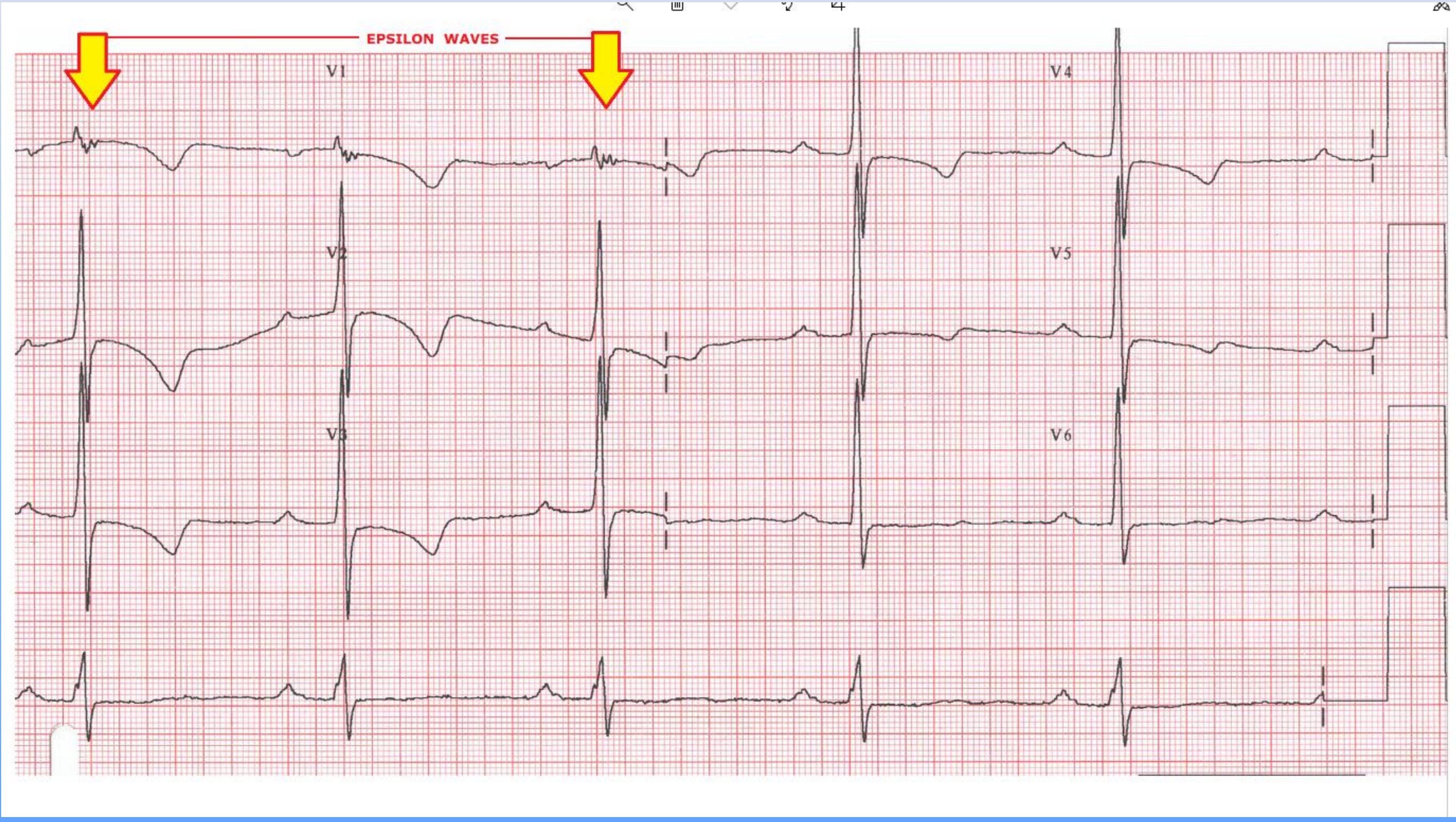


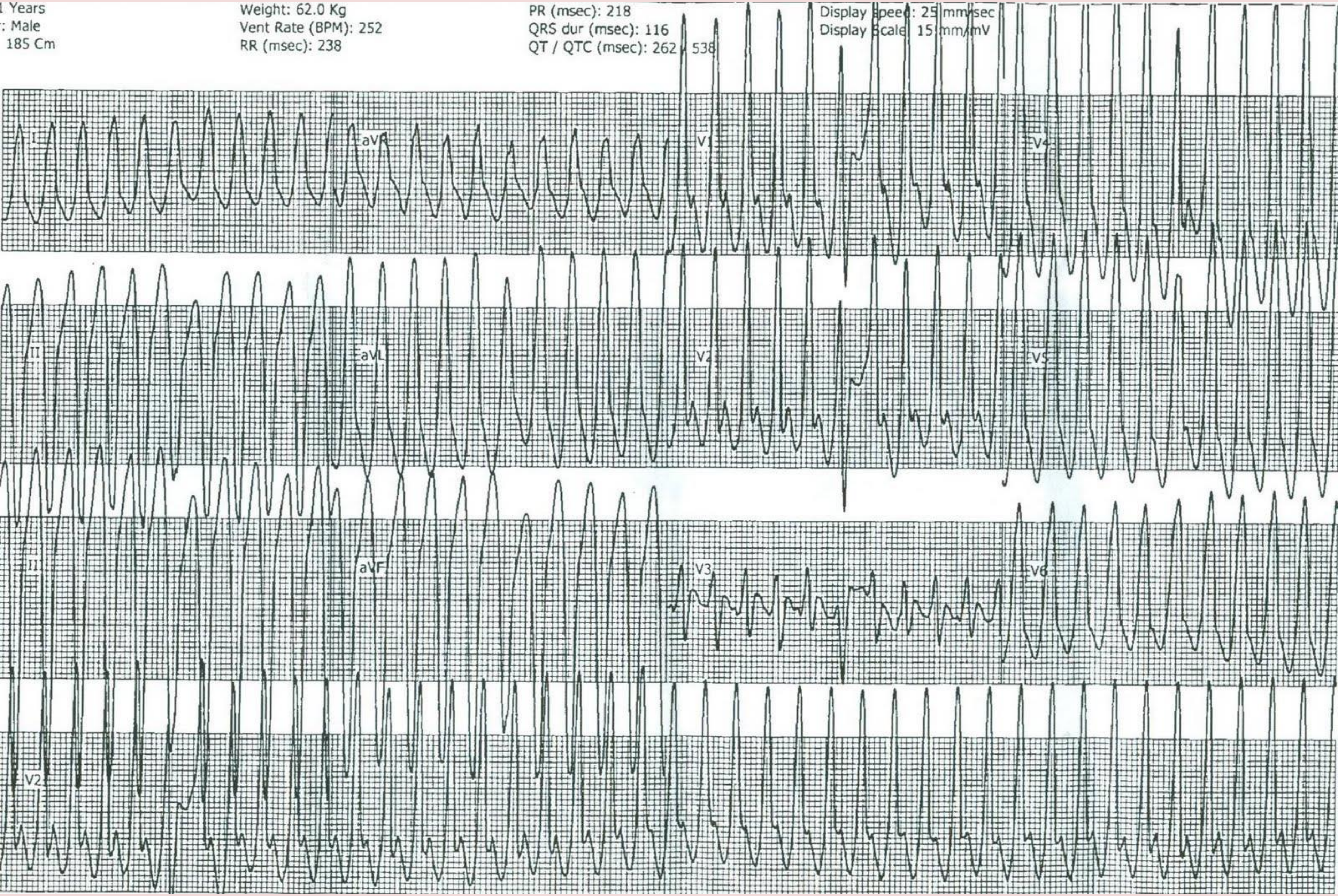
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

Would you spot the Epsilon's Waves?

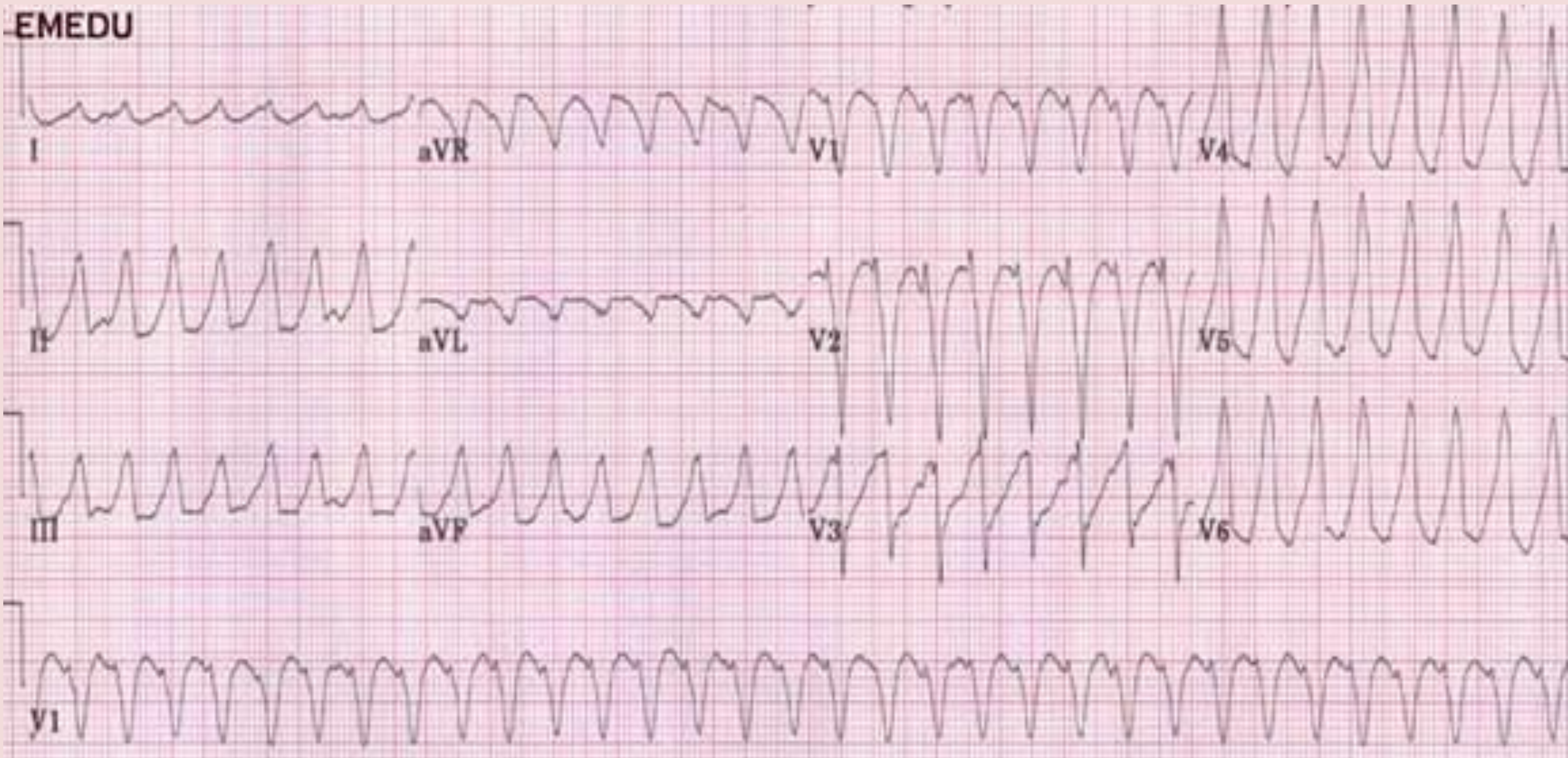


BHSR Patient – Epsilon's Waves




Years
: Male
185 Cm
Weight: 62.0 Kg
Vent Rate (BPM): 252
RR (msec): 238
PR (msec): 218
QRS dur (msec): 116
QT / QTC (msec): 262 / 538
Display speed: 25 mm/sec
Display Scale: 15 mm/mV
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V2

ARVD INDUCED VT



Evidence Based Reference Sources

- **2016 ACC** Interassociation Consensus Statement on Cardiovascular Care of College Student-Athletes
- **2014 AHA/ACC Scientific Statement**: Assessment of the 12-Lead ECG as a Screening Test for Detection of Cardiovascular Disease in Healthy General Populations of Young People (12–25 Years of Age)
- **AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram: Part IV: The ST Segment, T and U Waves, and the QT Interval : Circulation 2009 119: e241-e250**
- **AHA Circulation: Inherited Arrhythmias; Basic Science for Clinicians**
- **AHA ACC Scientific Statement Prevention of Torsade de Pointes in Hospital Settings**
- **AHA ACC QTc Behavior During Exercise and Genetic Testing for the Long-QT Syndrome**
- **Pharmacology Review: Drug Induced Long QT Syndromes**

Evidence Based Reference Sources, cont’

- HRS/EHRA/APHRS Expert Consensus Statement on the Diagnosis and Management of Patients with Inherited Primary Arrhythmia Syndromes
- Genetic Determinants of Sudden Cardiac Death: AHA Circulation.2008; 118: 1854-1863
- AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram: Part III: Intraventricular Conduction Disturbances
- AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram : Part V: Electrocardiogram Changes Associated With Cardiac Chamber Hypertrophy
- Arrhythmogenic Disorders of Genetic Origin; Brugada Syndrome: Circulation: Arrhythmia and Electrophysiology.2012; 5: 606-616

Other Reference Sources:

www.JACC.org

http://circ.ahajournals.org/

SADS FOUNDATION
SUDDEN ARRHYTHMIA DEATH SYNDROMES

www.SADS.org

My top two reasons for giving everything in life the best I have to offer.

YOU MADE IT !!!

Any

???