The LIFSAVING Electrocardiogram

PART II

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

19th CONGRESS MIAMI

American College of Cardiology
Accreditation Services
(formerly The Society of Cardiovascular Patient Care)

May 25-27,2016

scpc.org/Congress

Elements of Sudden Cardiac Death Prevention Programs

The American College of Cardiology Accreditation Services

19th Congress – Miami, FL – May 25, 2016

Wayne Ruppert, CVT, CCCC, NREMT-P

To download presentation in PDF: visit: www.ECGtraining.org select: "Downloads - PDF"

Brief, focused ECG excerpts from 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%**

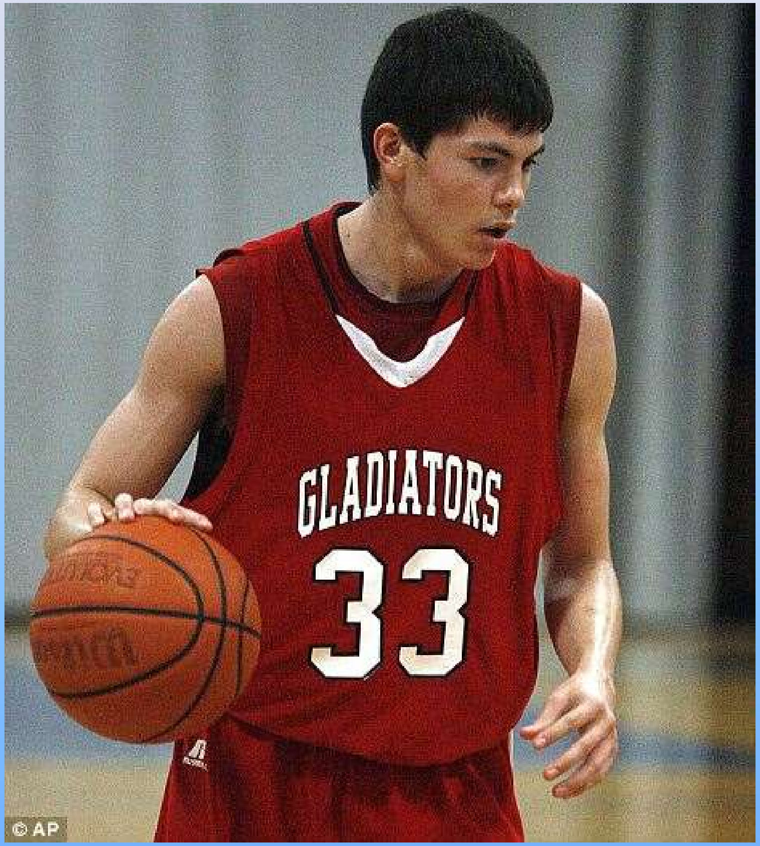
Most ACS Patients are over age 30.

Meet the typical patients affected by LQTS . . . .

High School Athlete Dies After Collapsing At Practice

August 15, 2011 11:28 PM

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



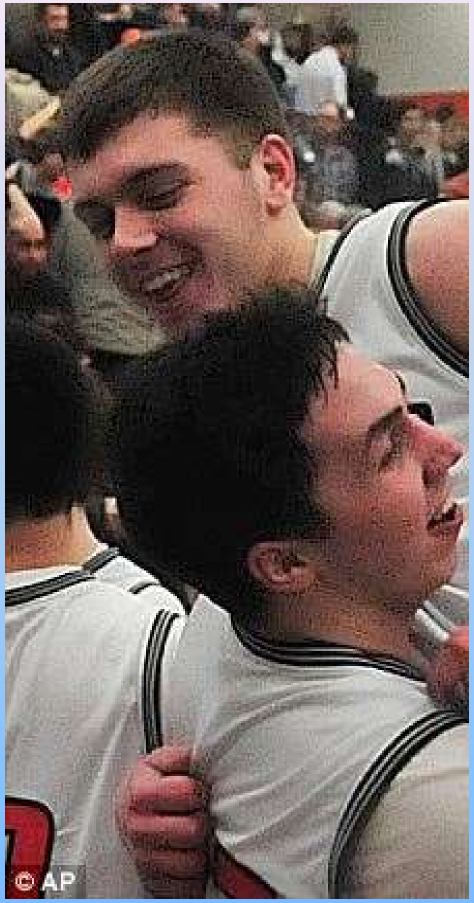
By DAILY MAIL REPORTER
UPDATED: 12:03 EST, 14 March 2011

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."***

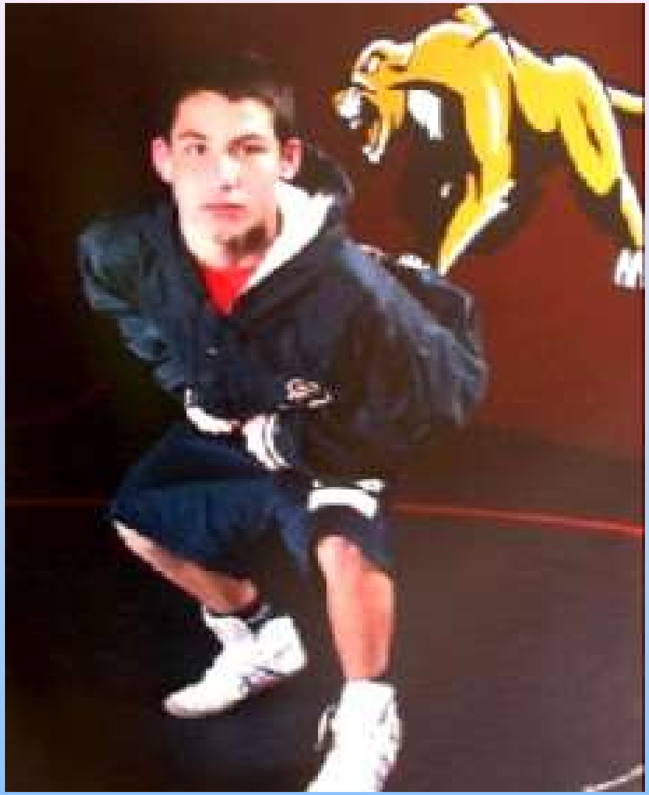
High school quarterback Reggie Garrett threw his second touchdown pass of the night, walked off the field, and collapsed from sudden cardiac arrest. He died in the ambulance on the way to the hospital in West Orange, Texas.

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 23
Dade City, Florida
February 1, 2015

Your Hometown News Source • Dade City News
February 12, 2015 • 7B dadecitynews.net

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 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.

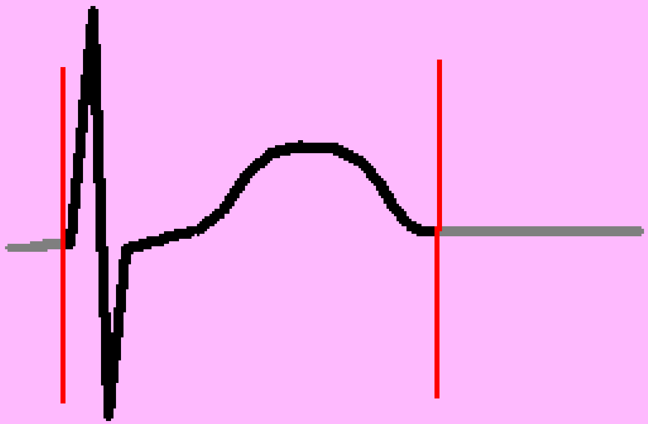
Sudden death was the first indication that she suffered from a cardiac condition.   At the current time, her specific diagnosis is unknown.

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

"As Healthcare Professionals, we have an obligation to implement programs, practices, protocols, policies and procedures designed to eliminate the needless mortality of SADS in our communities."

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:

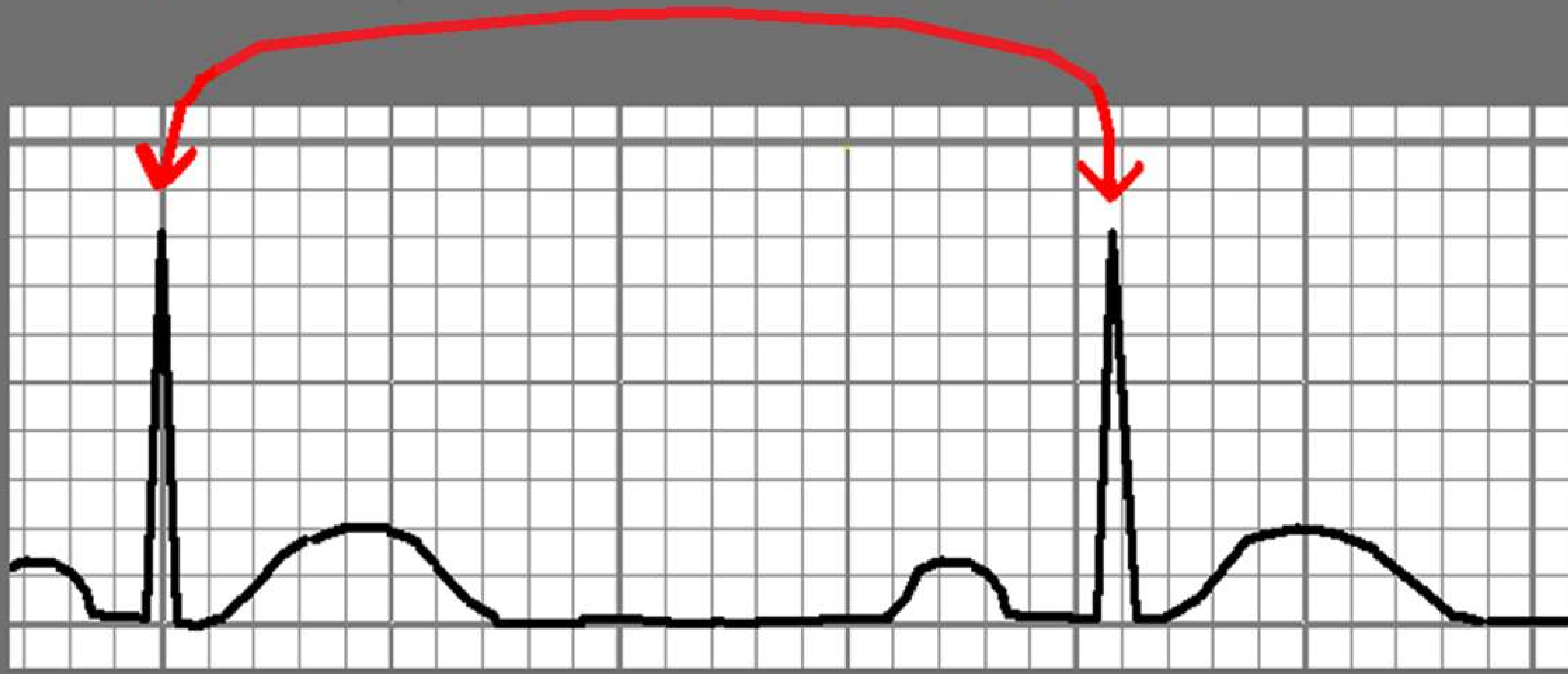
QT CORRECTION FORMULAS:

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

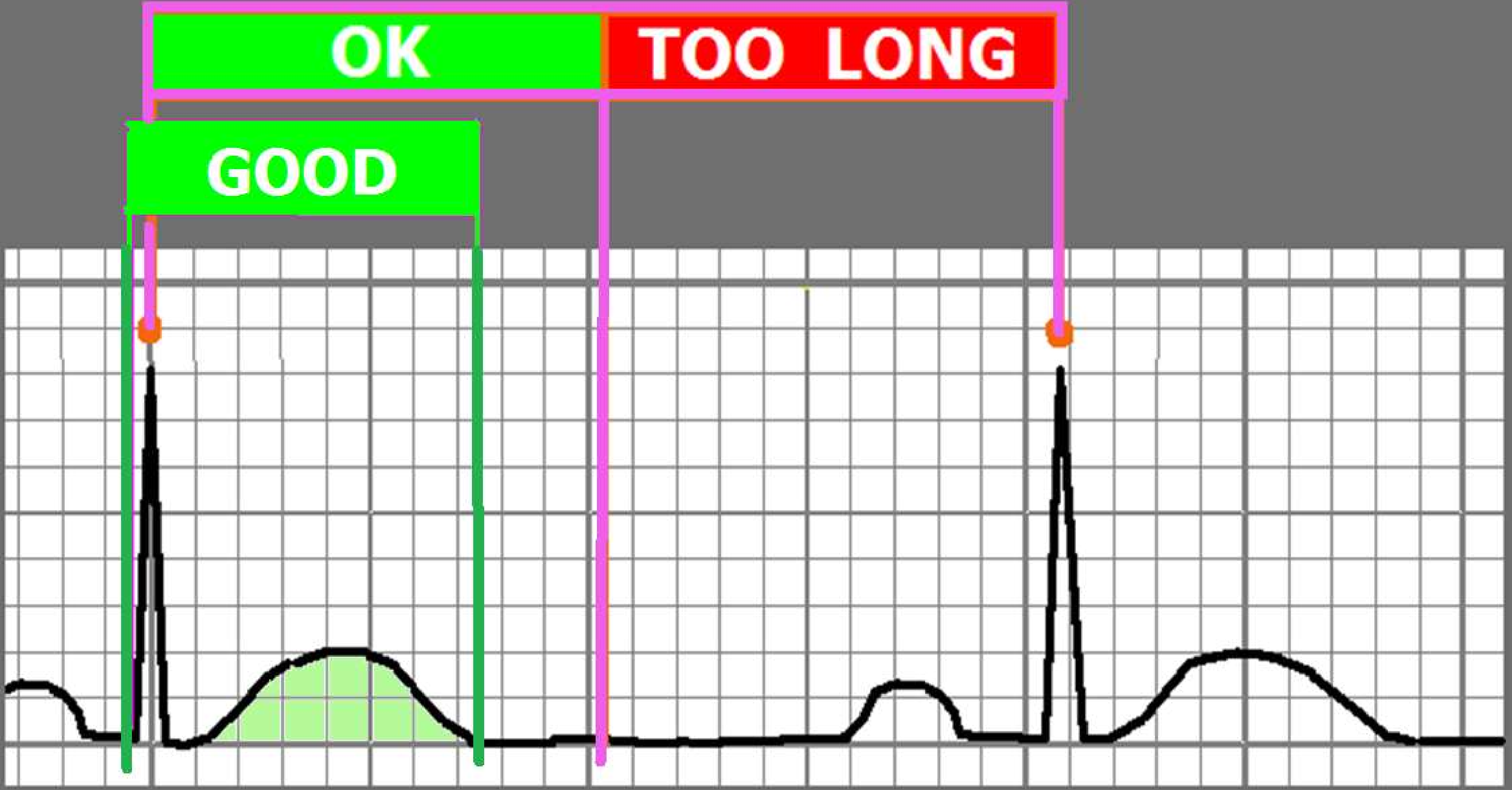
DETERMINING Q-T INTERVAL LIMITS

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



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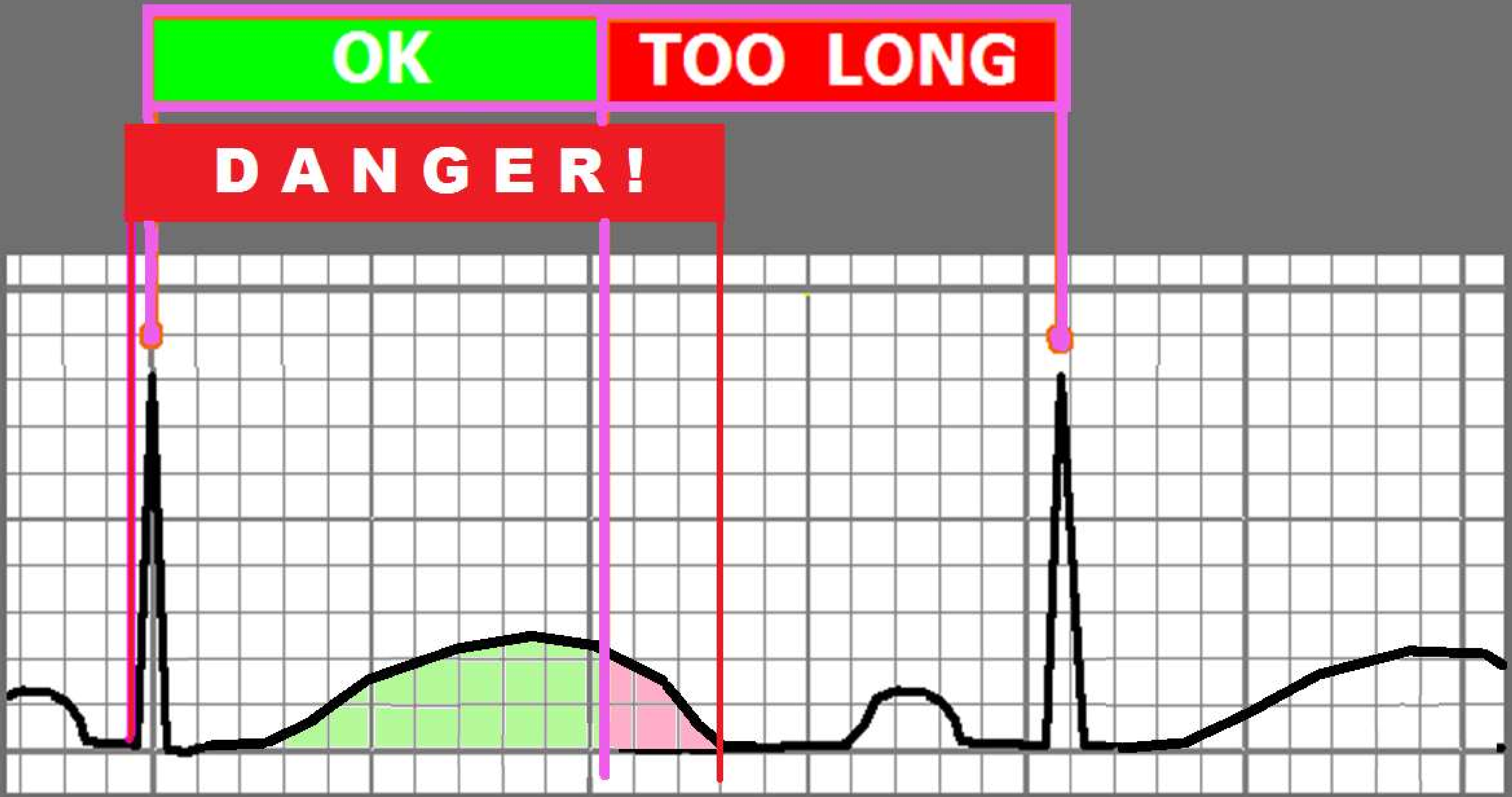


Determining the QT / QTc

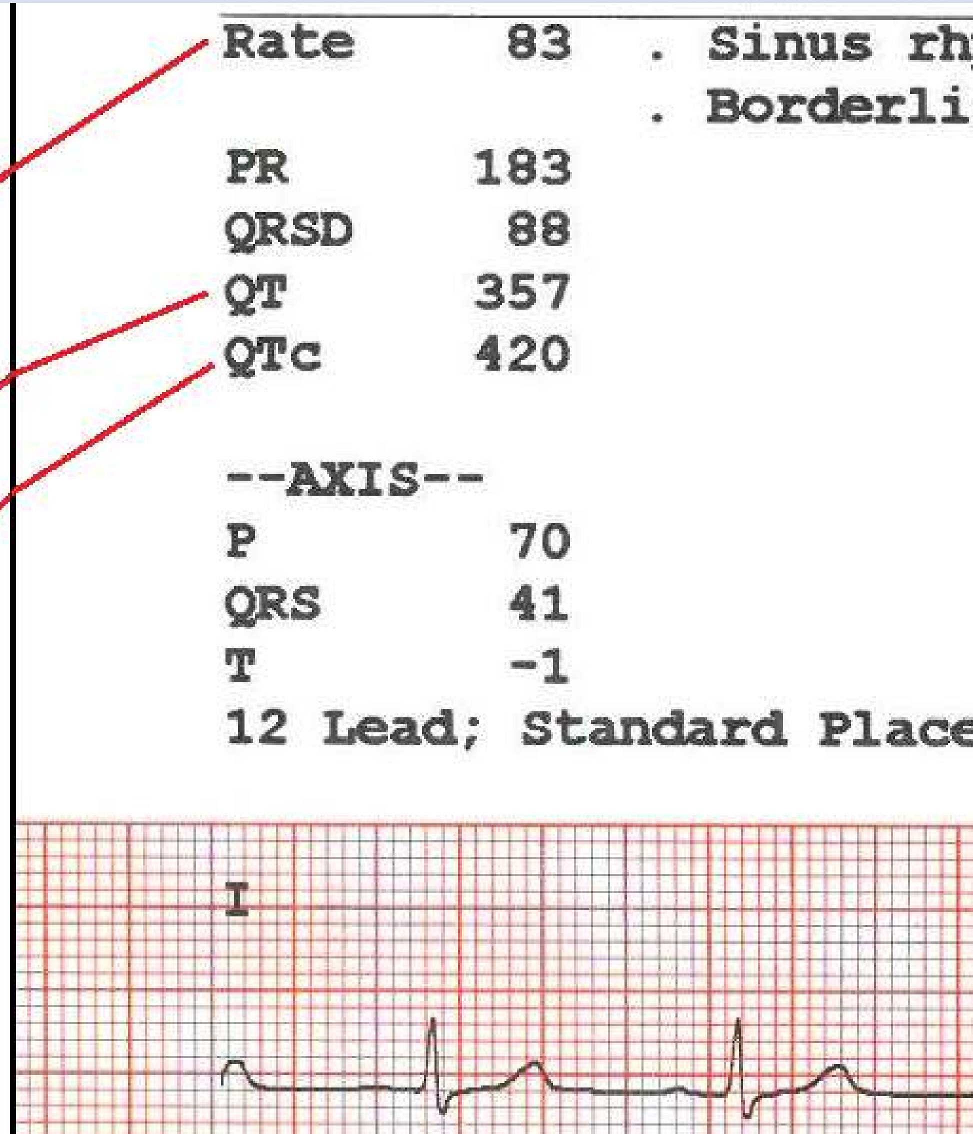
Method 1 – 12 Lead ECG Report:

Standard 12 Lead ECG printout . . .

Heart Rate = 83

QT Interval = 357

QTc = 420

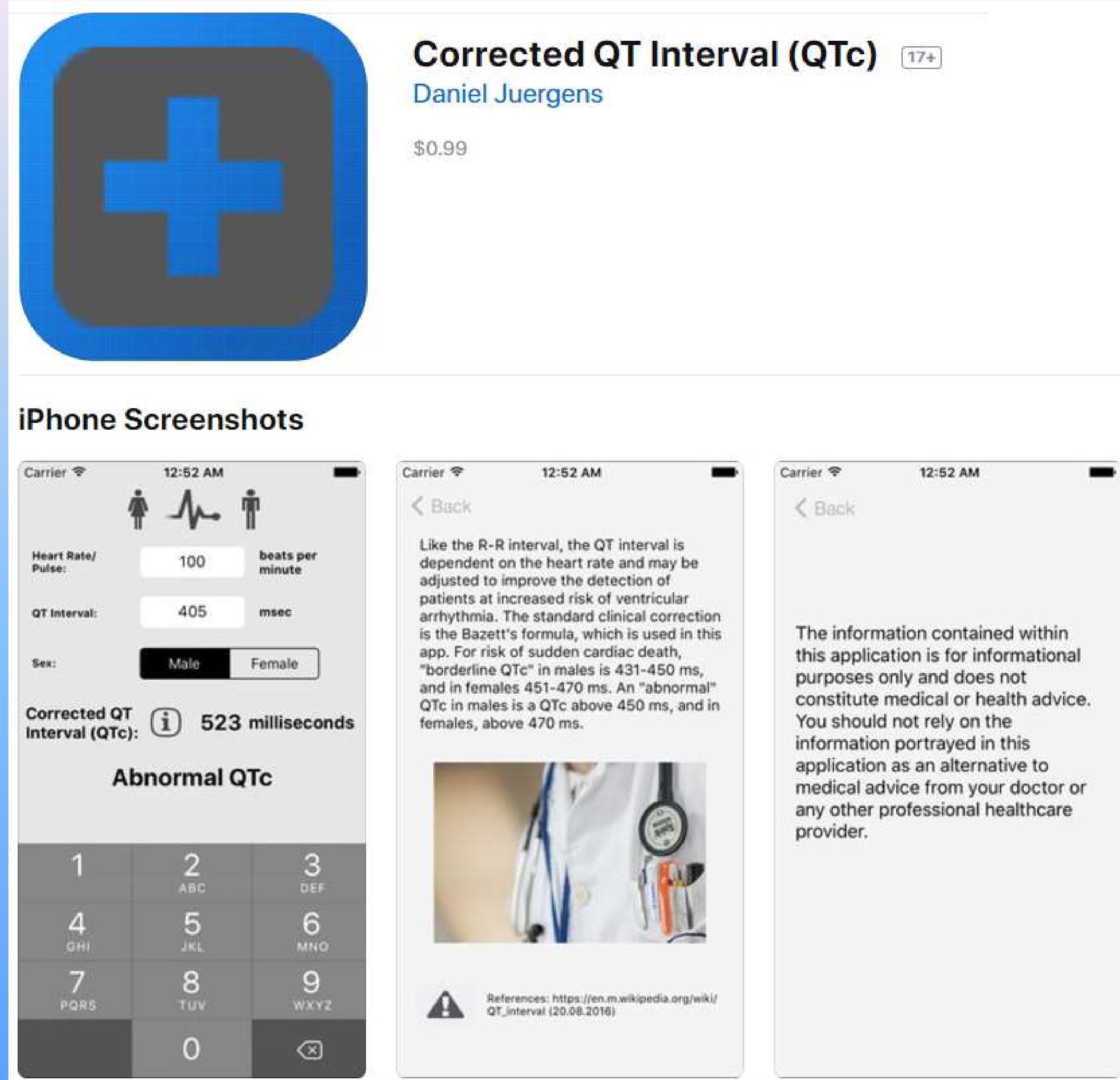


Determining the QTc

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:

theheart.org Medscape

Calculators ‣ Heart and Chest, Critical Care

QT Interval Correction (EKG)

Share

Input:

QT Interval	310	msec
Heart Rate	88	bpm

Results:

RR Interval	682	msec
QTI Corrected	375	msec

Decimal Precision: 0

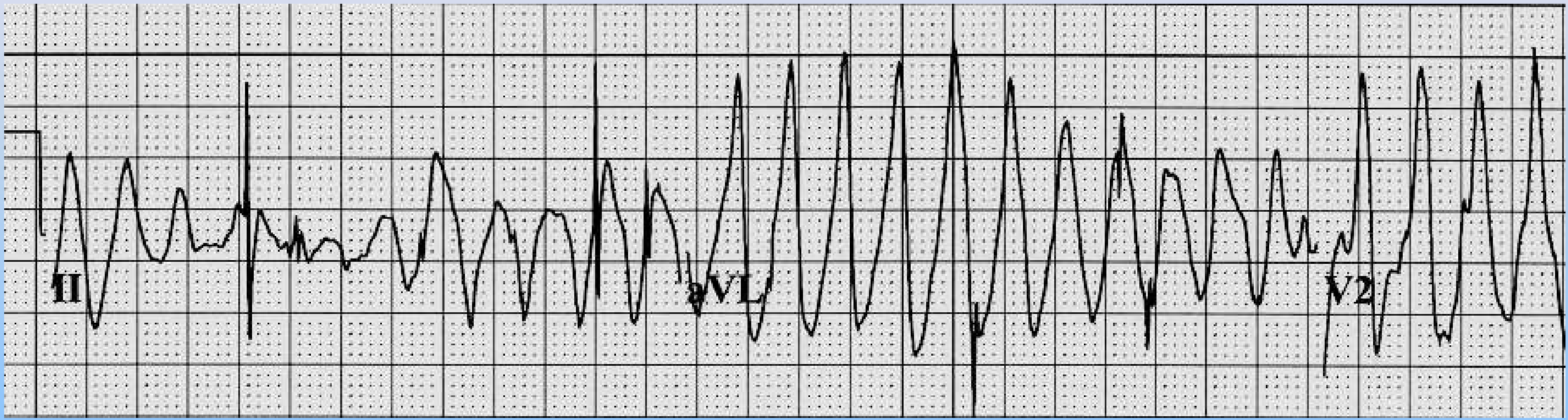
Our patient's QTc = 375 ms.

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*



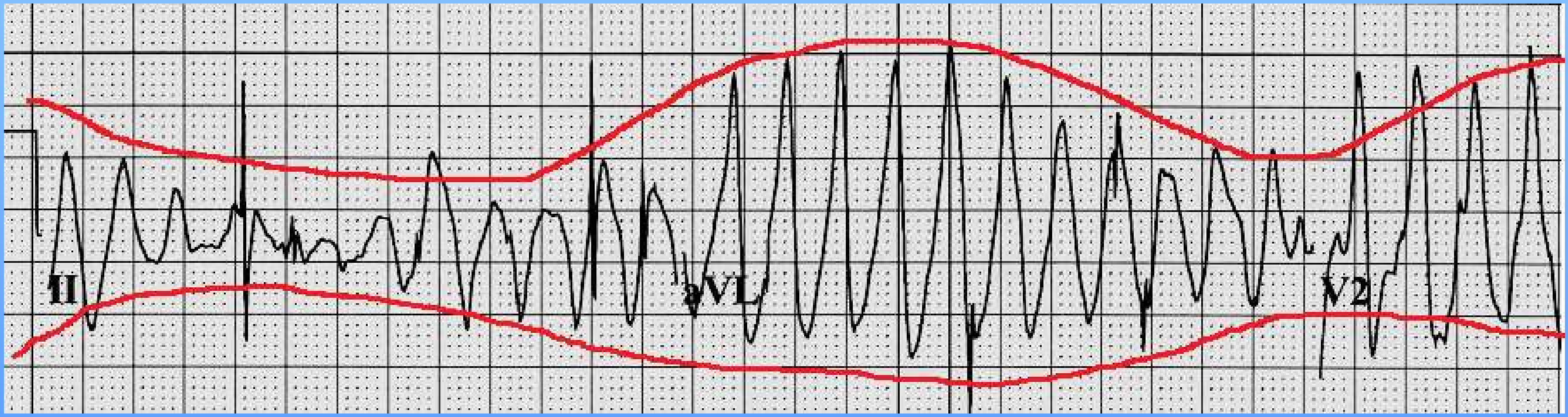
Torsades de Pointes (TdP) – HEMODYNAMICS:

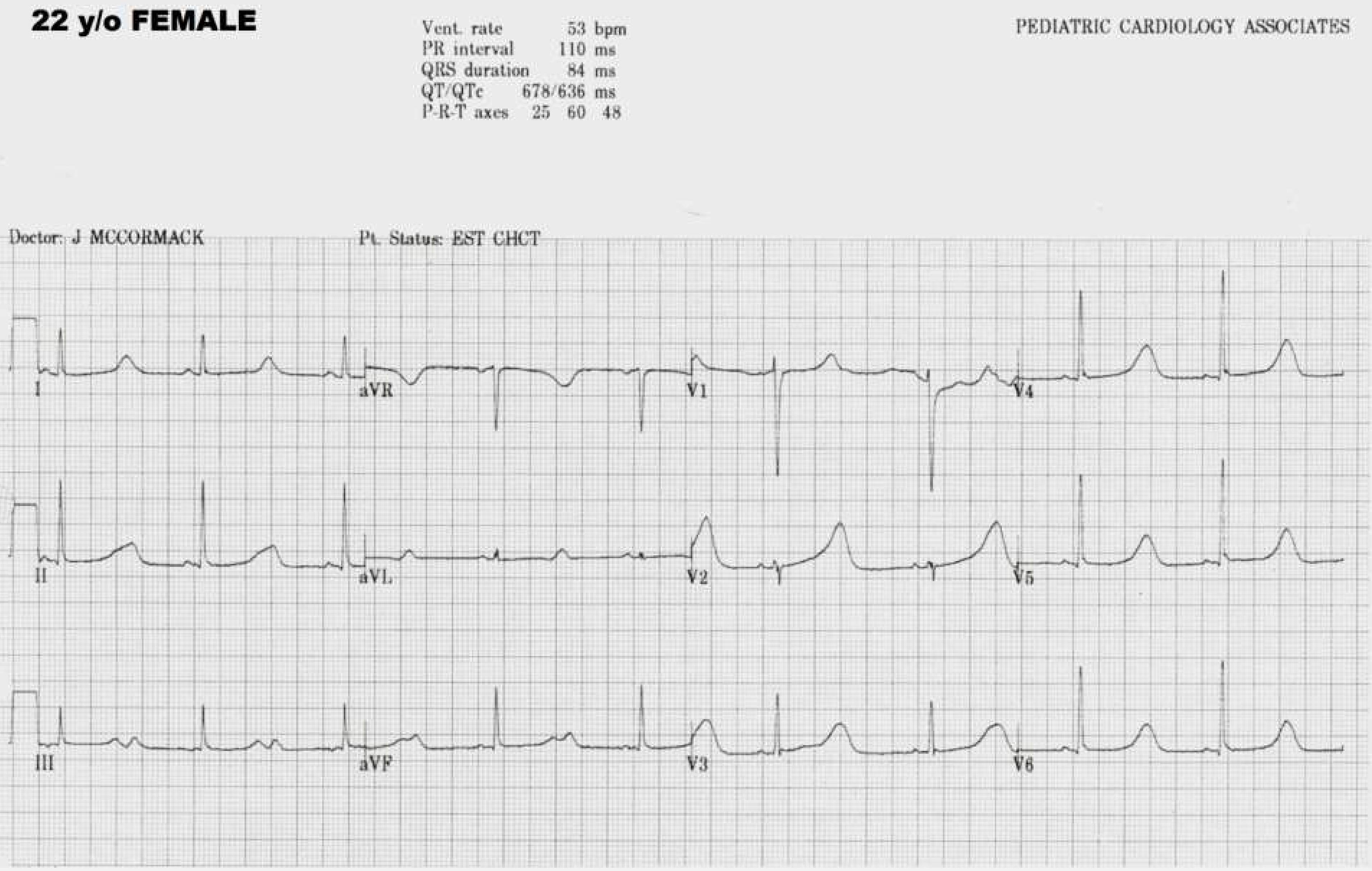
- **Decreased – to – NO Cardiac Output**
- **Often patient PULSELESS during episode**
- **Patients often report SYNCOPE when TdP self-terminates.**
- **May DETERIORATE into VENTRICULAR FIBRILLATION and CARDIAC ARREST. ("Sudden Death")**

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

resembles . . . . .

a piece of Twisted Ribbon !





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

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***

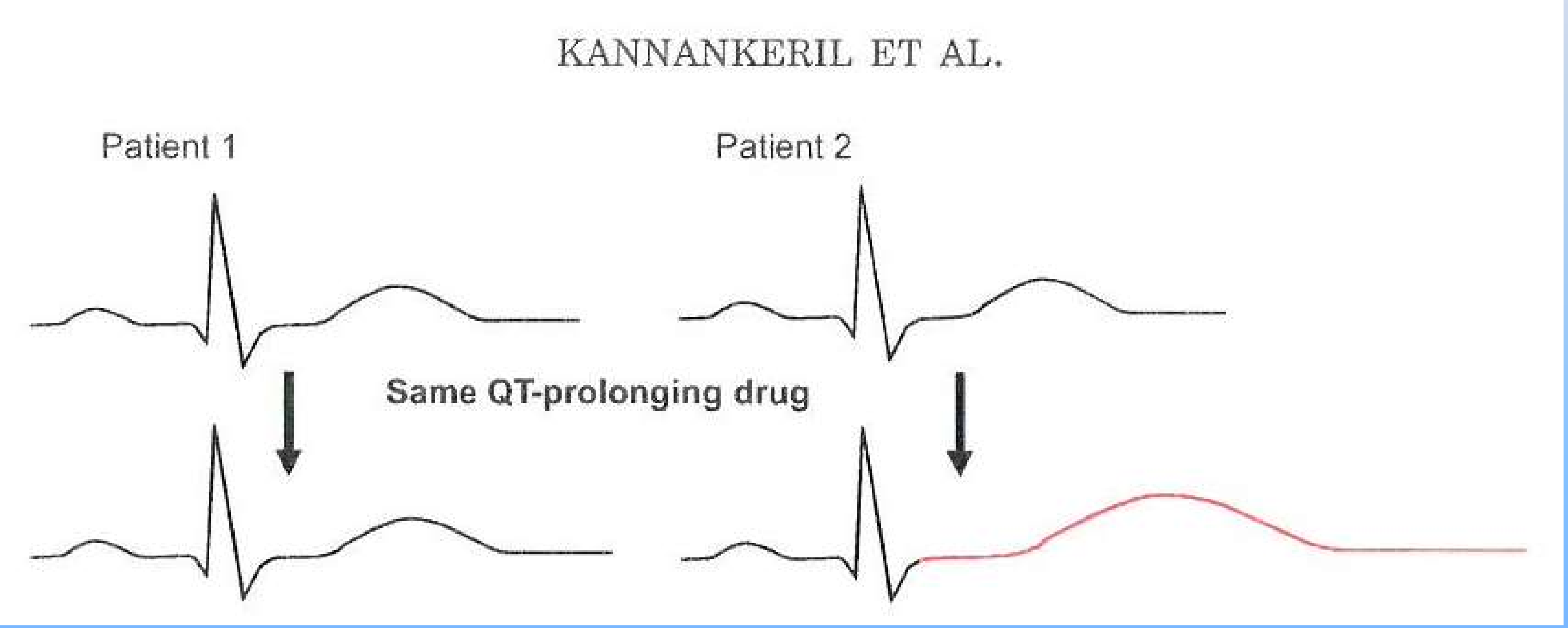
Commonly used QT prolonging meds include:

-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:



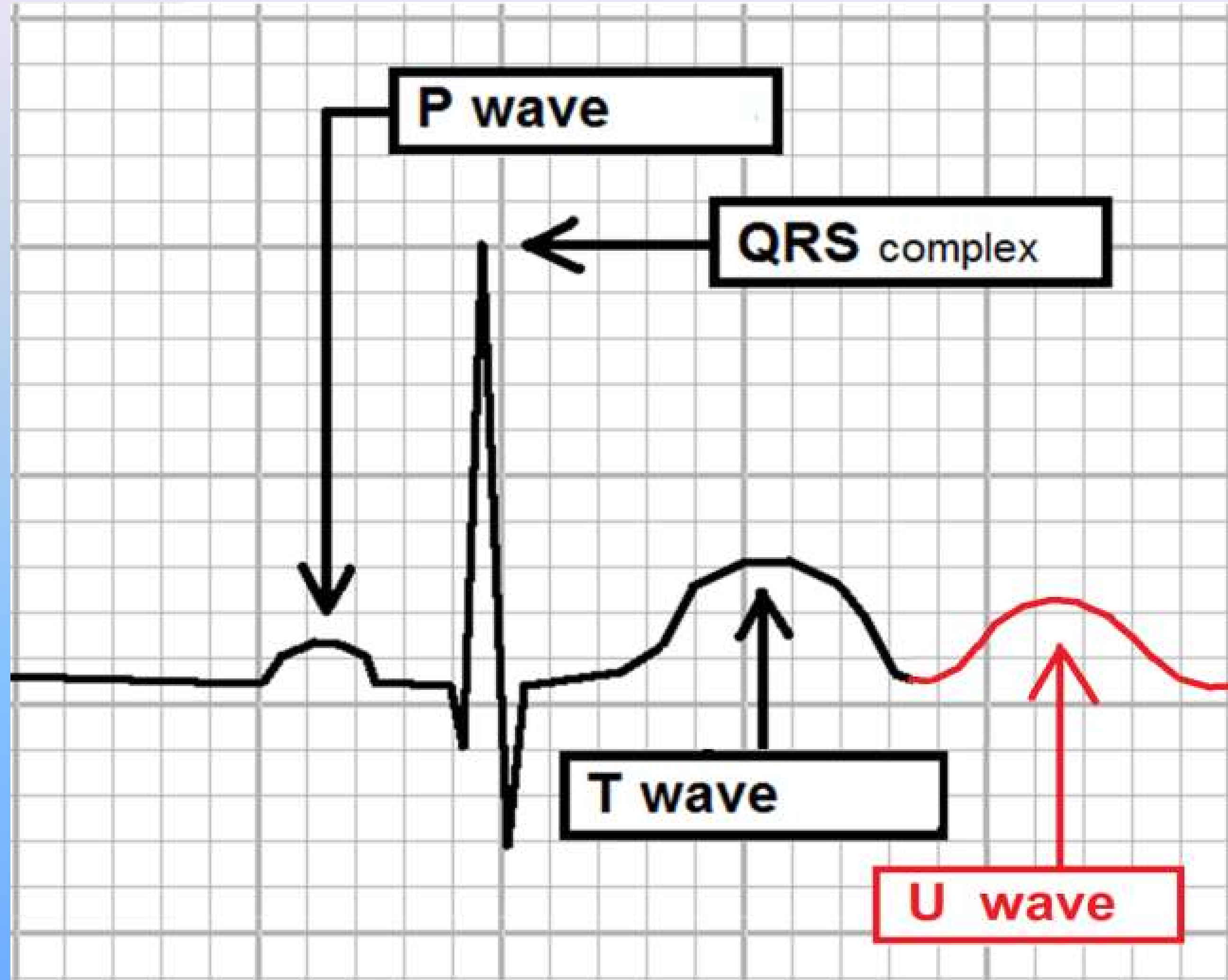
Click here for link to paper by Kannankeril et al (2010 Pharmacological Reviews) that describes genetic susceptibility described above.

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***
  - **Hypocalcemia***
  - **QT prolonging medications***
  - **Increased intracranial pressure***
  - **Hypothermia***
  - **Digitalis** (usually *shortens* the QT Interval)

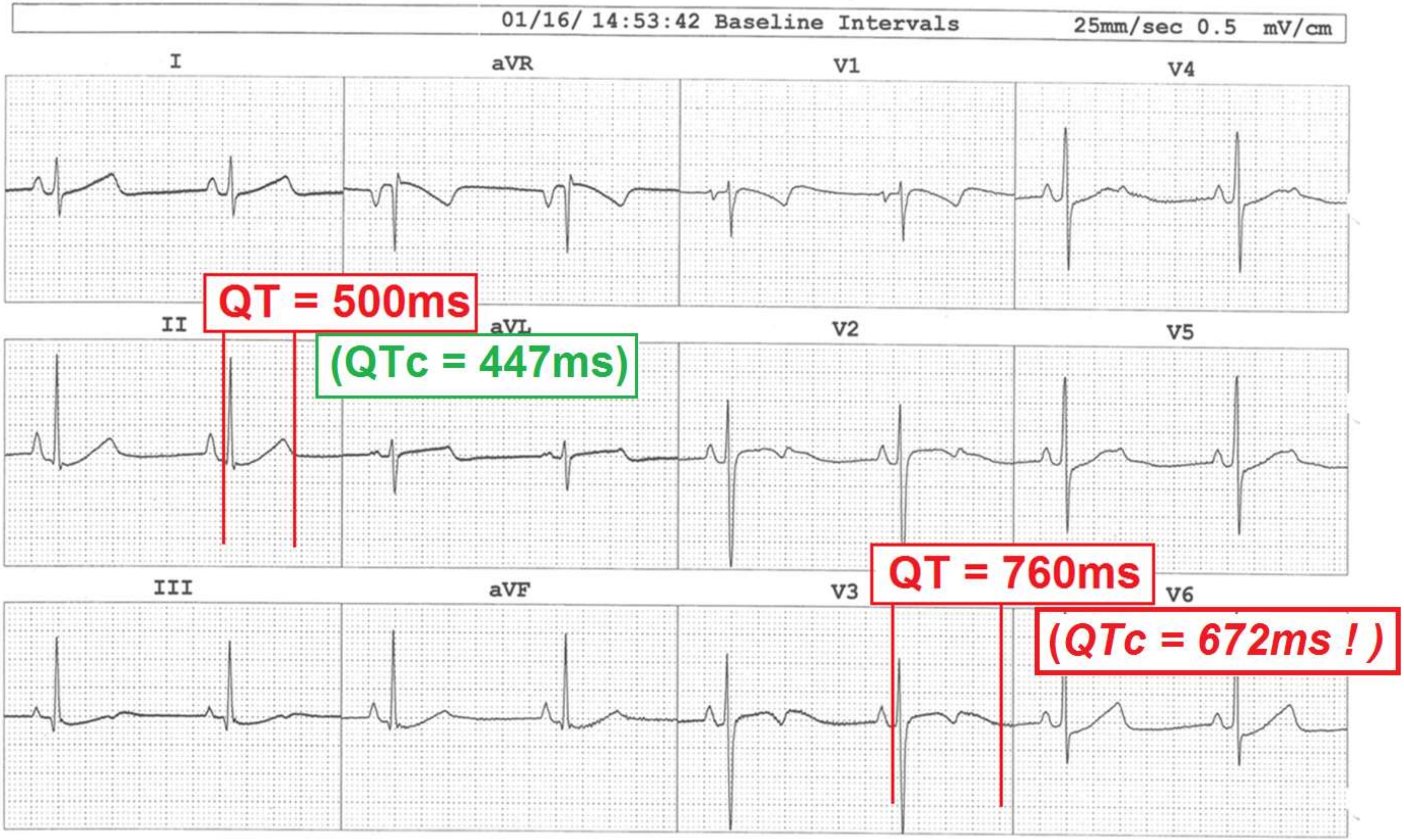
**** These are also causes of QT interval prolongation.***

Abnormal U Waves

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

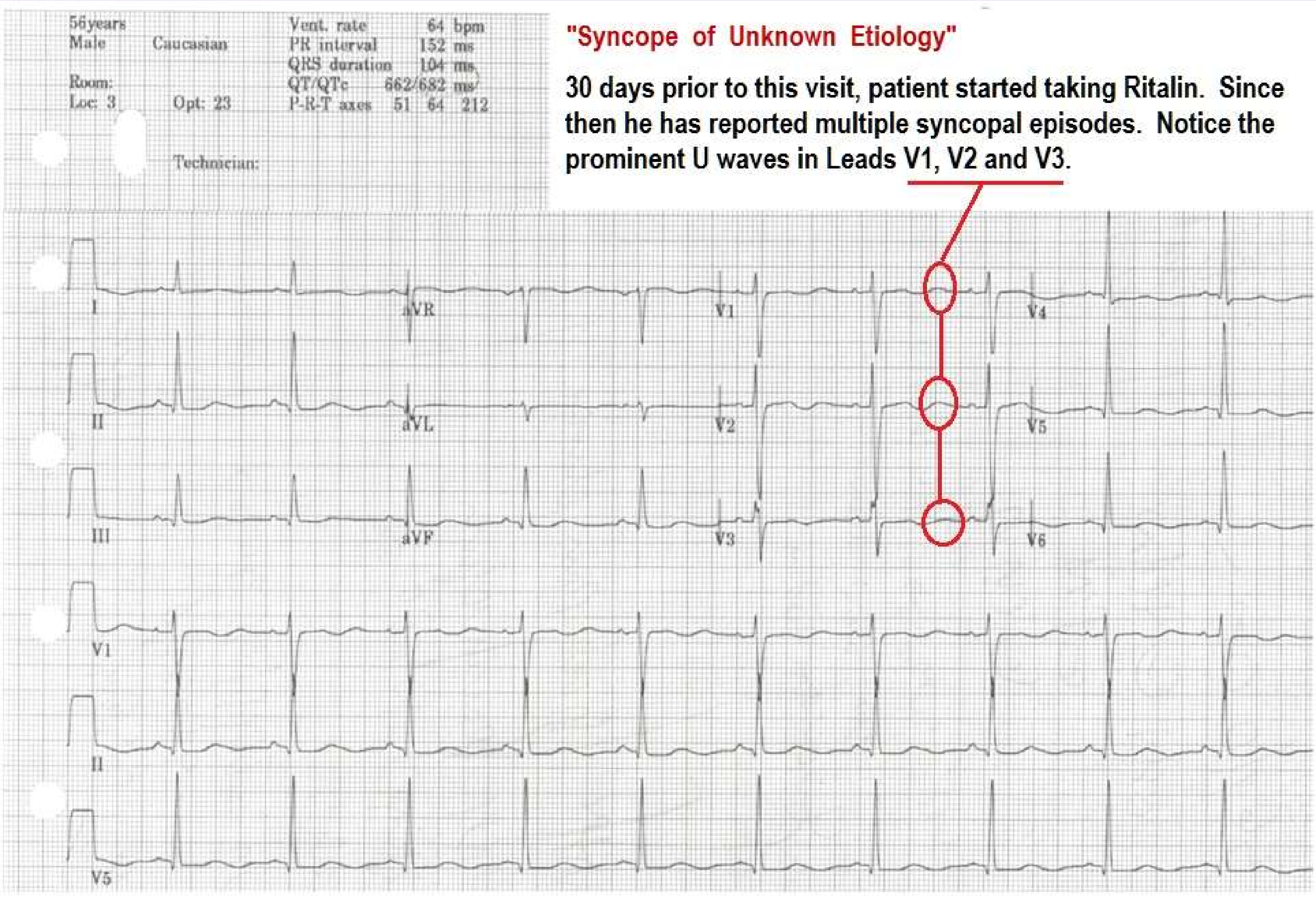
- **U wave 100% (or more) the size of the T wave.**
- **U wave is INVERTED (opposite polarity of T wave)**
- **U wave merged with the T wave**

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.

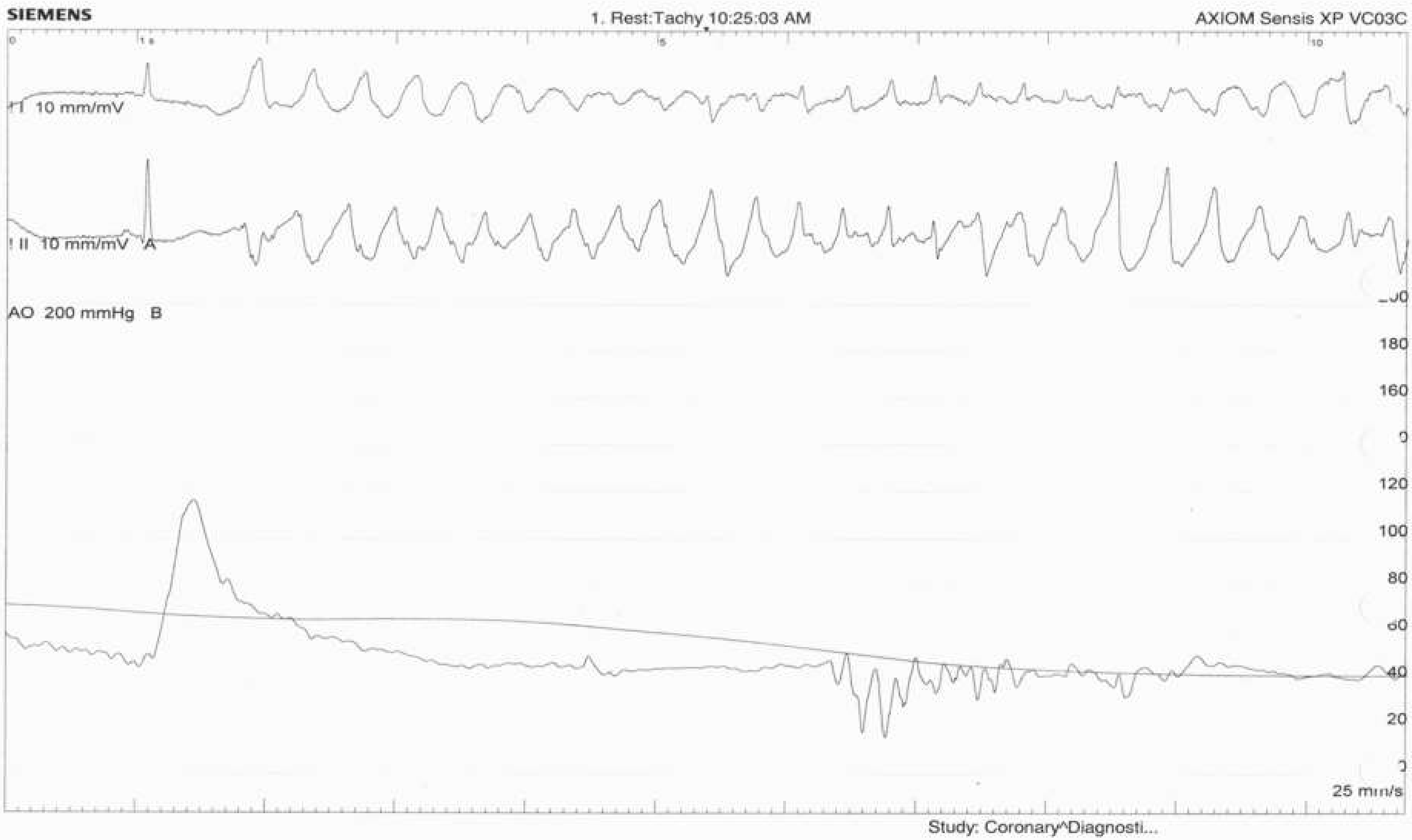


This ECG illustrates the degree of variation that can be noted between different leads on the 12 Lead ECG. ALWAY measure the QT Interval in the lead with the GREATEST value.

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

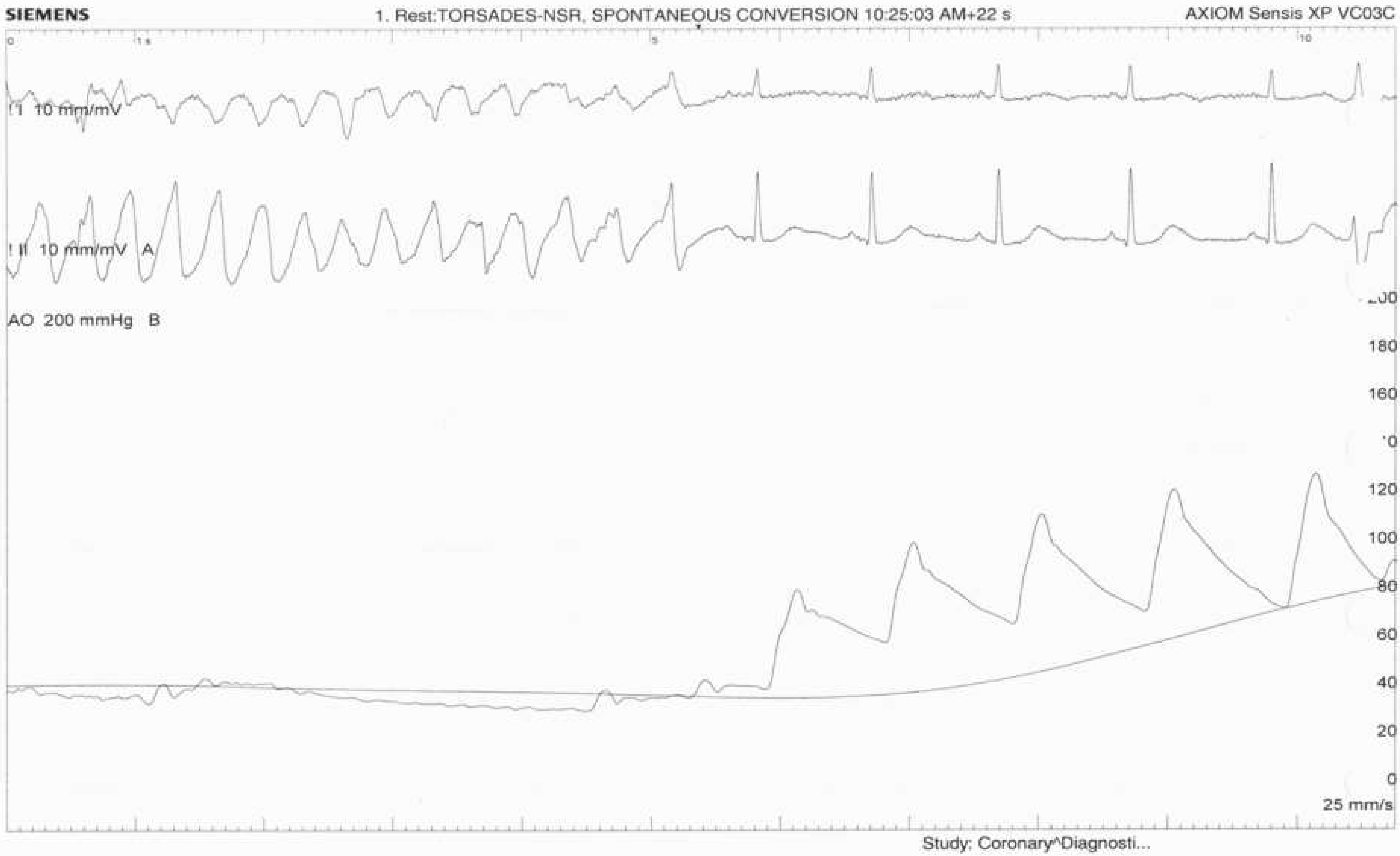


Medication induced LQTS with TdP and Cardiac Arrest - Case Study: 56 year old male



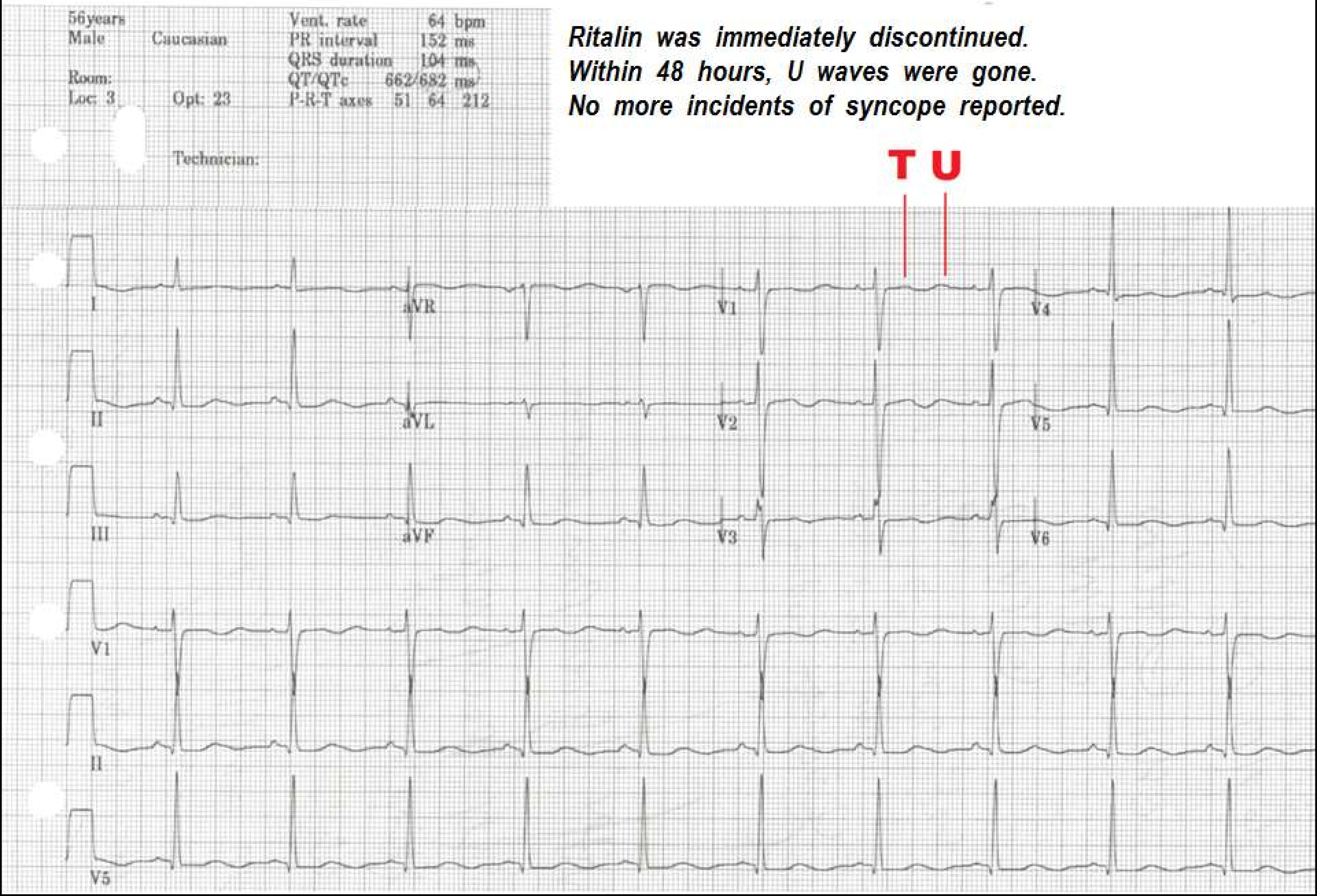
Run of Torsades de Pointes occurred during Cardiac Catheterization . . .

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

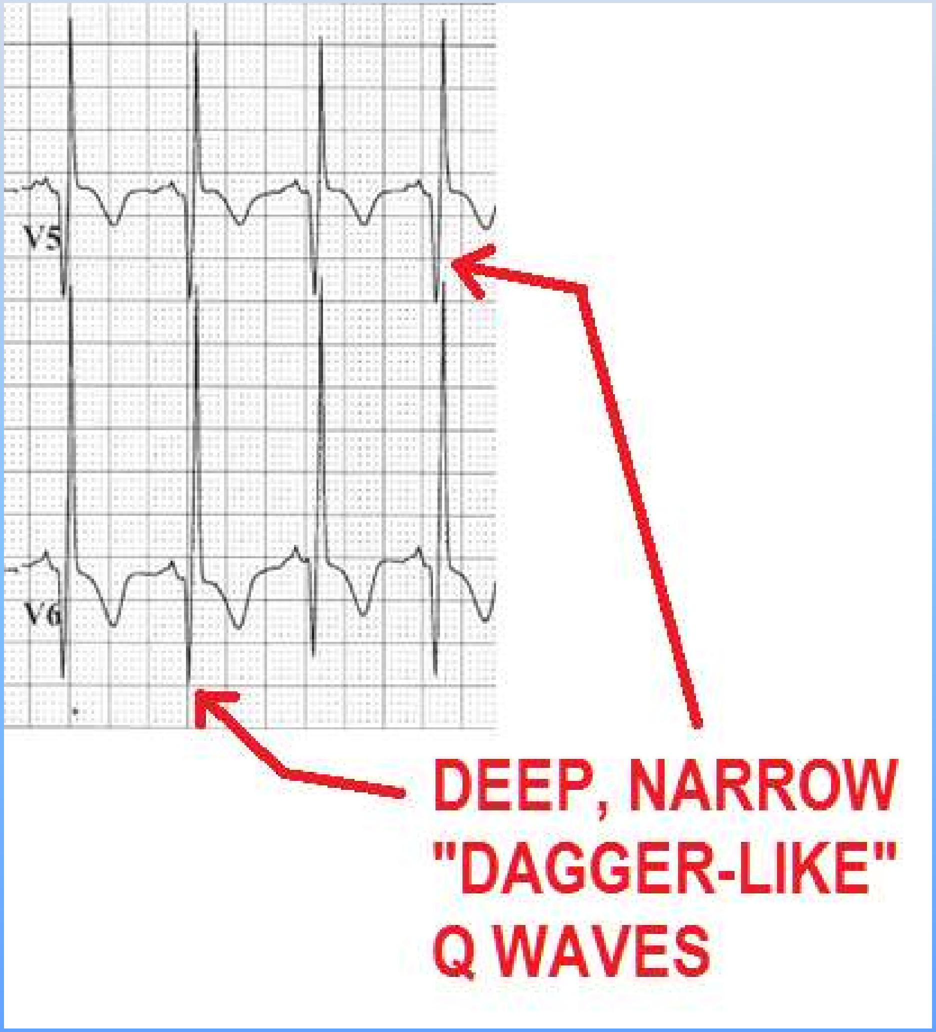


Ritalin was immediately discontinued.
Within 48 hours, U waves were gone.
No more incidents of syncope reported.

ECG Indicators: Hypertrophic Cardiomyopathy

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

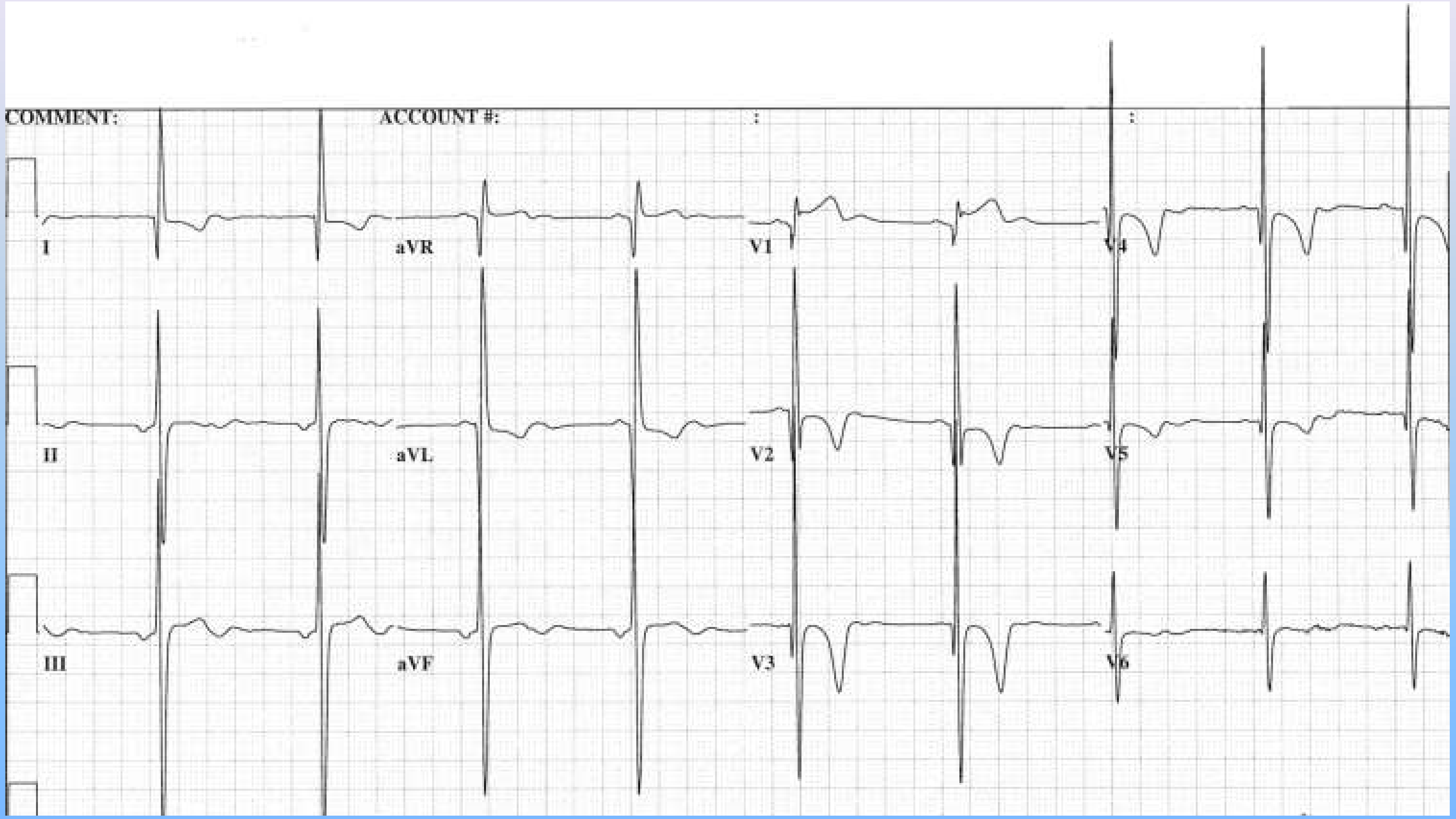
ECG Indicators: Hypertrophic Cardiomyopathy



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

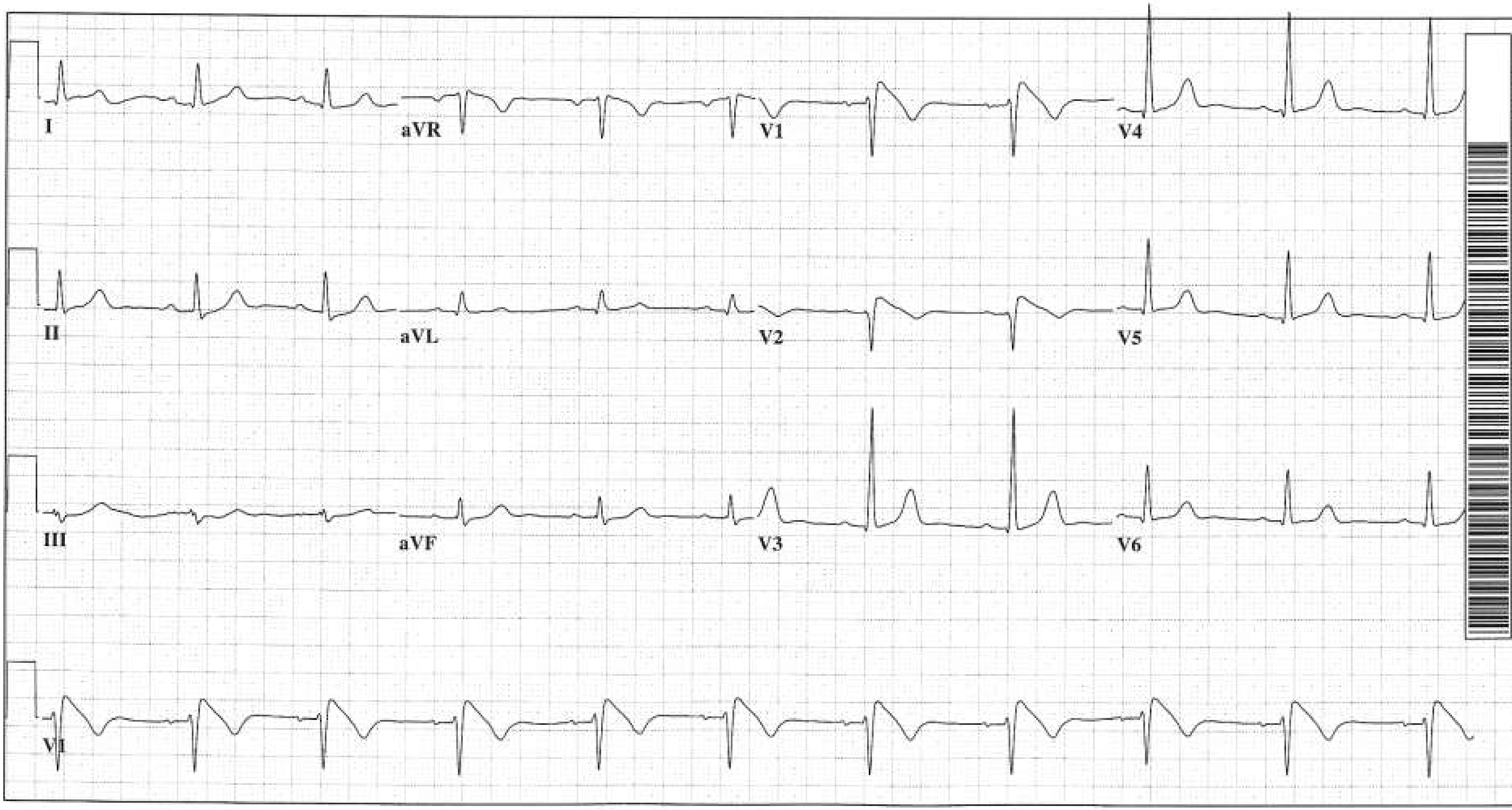
Room:C4A
Loc:3 Option:23

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

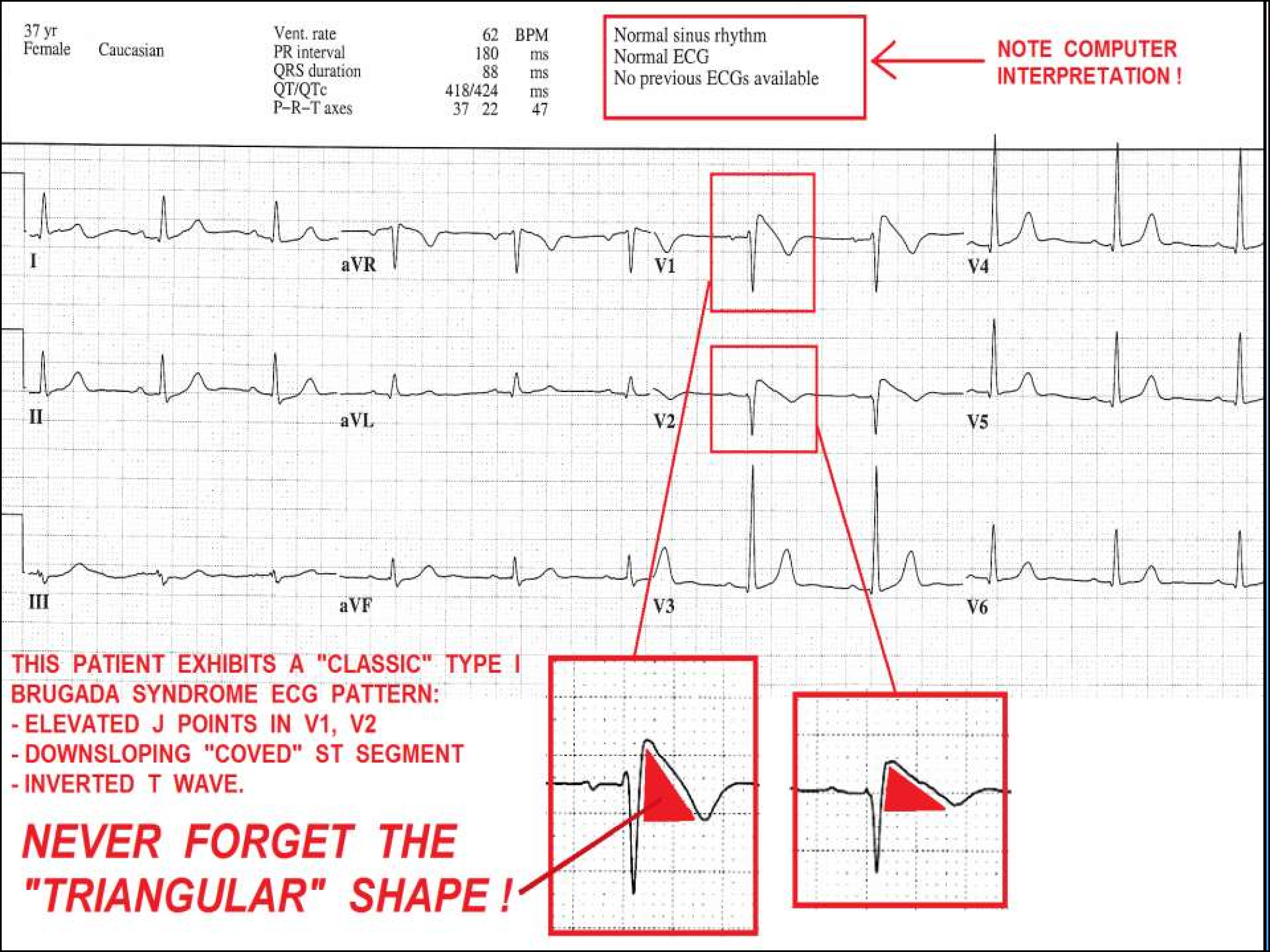
Technician:

Referred by:



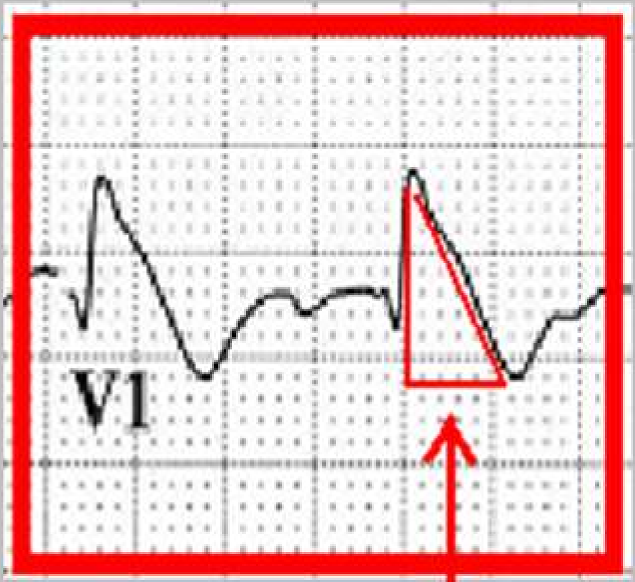
25mm/s 10mm/mV 40Hz 005D 12SL 235 CID: 3

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

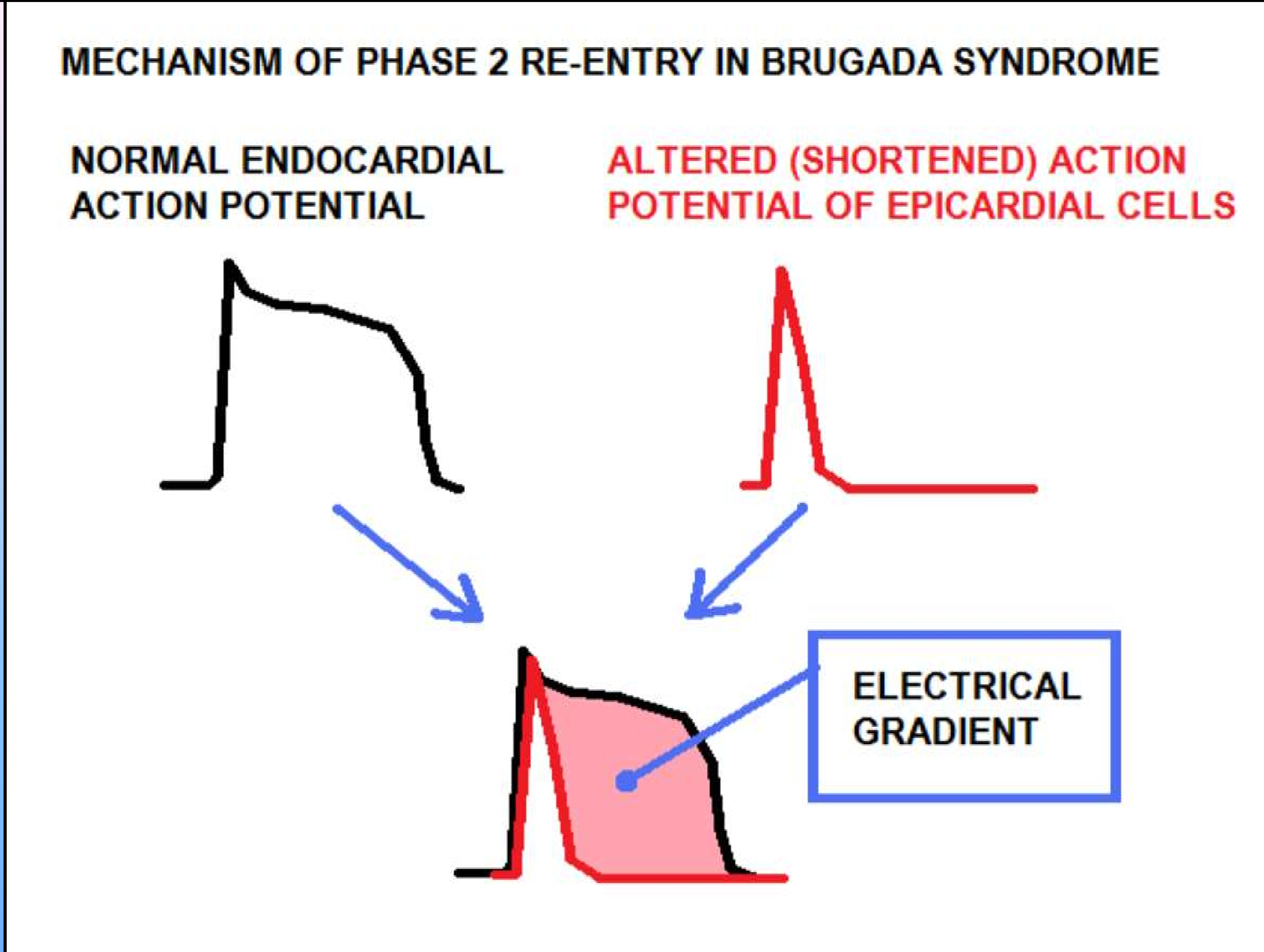
PATTERNS of S-T ELEVATION :

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.

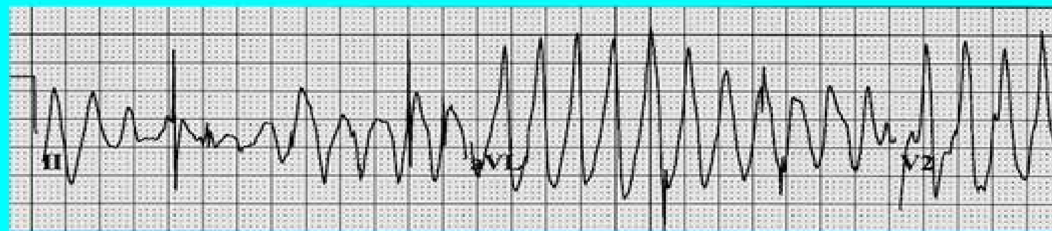
Brugada / Long QT Syndromes cause:

Torsades de Pointes:

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

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.

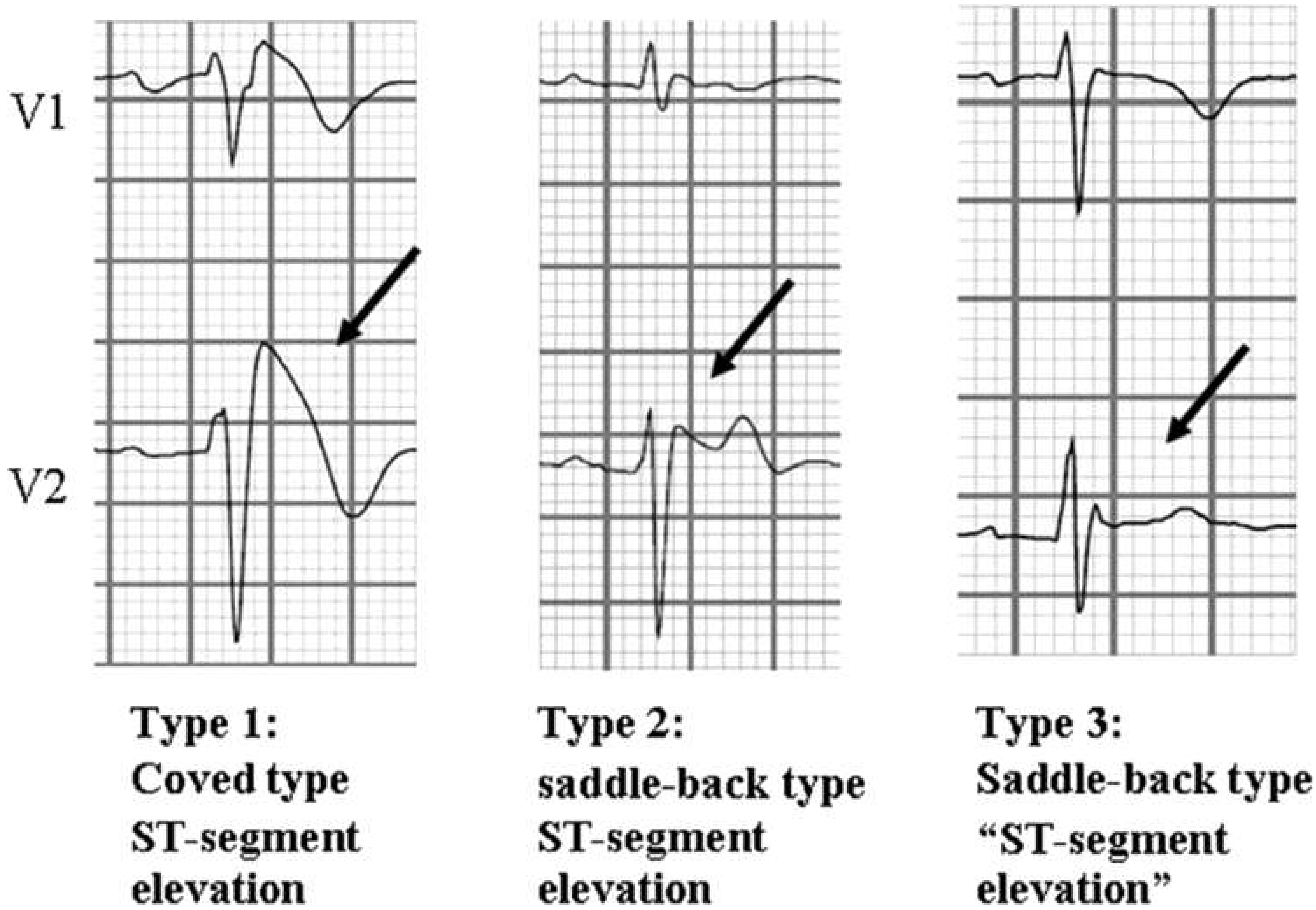
DO NOT give PROCAINAMIDE, AMIODARONE, or SOTALOL to patients with TORSADES or POLYMORPHIC VT !!!

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.

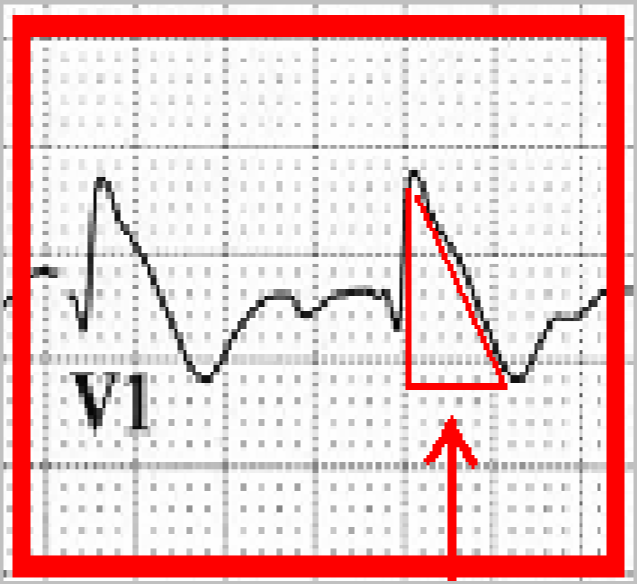
ECG abnormality diagnostic or suspected of Brugada syndrome.



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

American Heart Association

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.

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.**

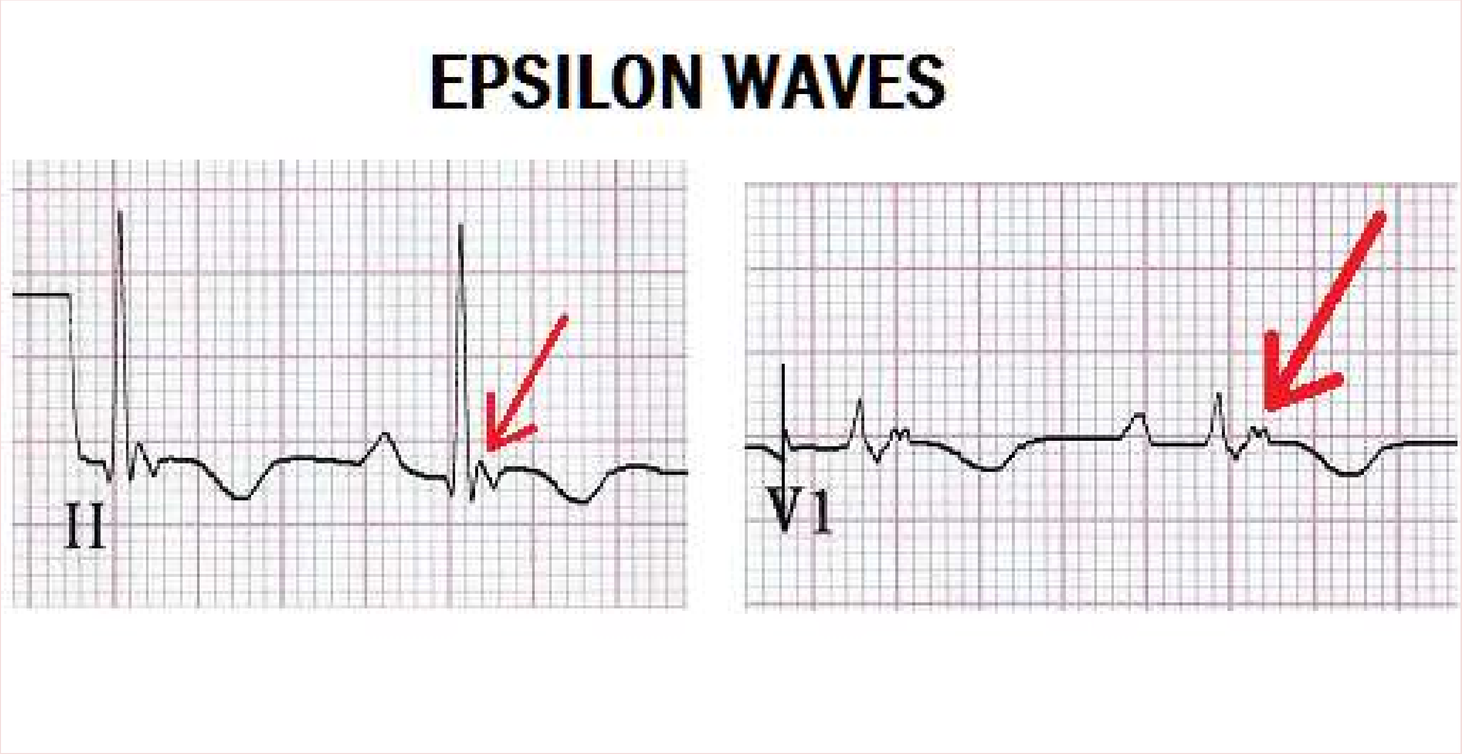
- **Estimated to affect approximately 1 in 5,000 people overall.**

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

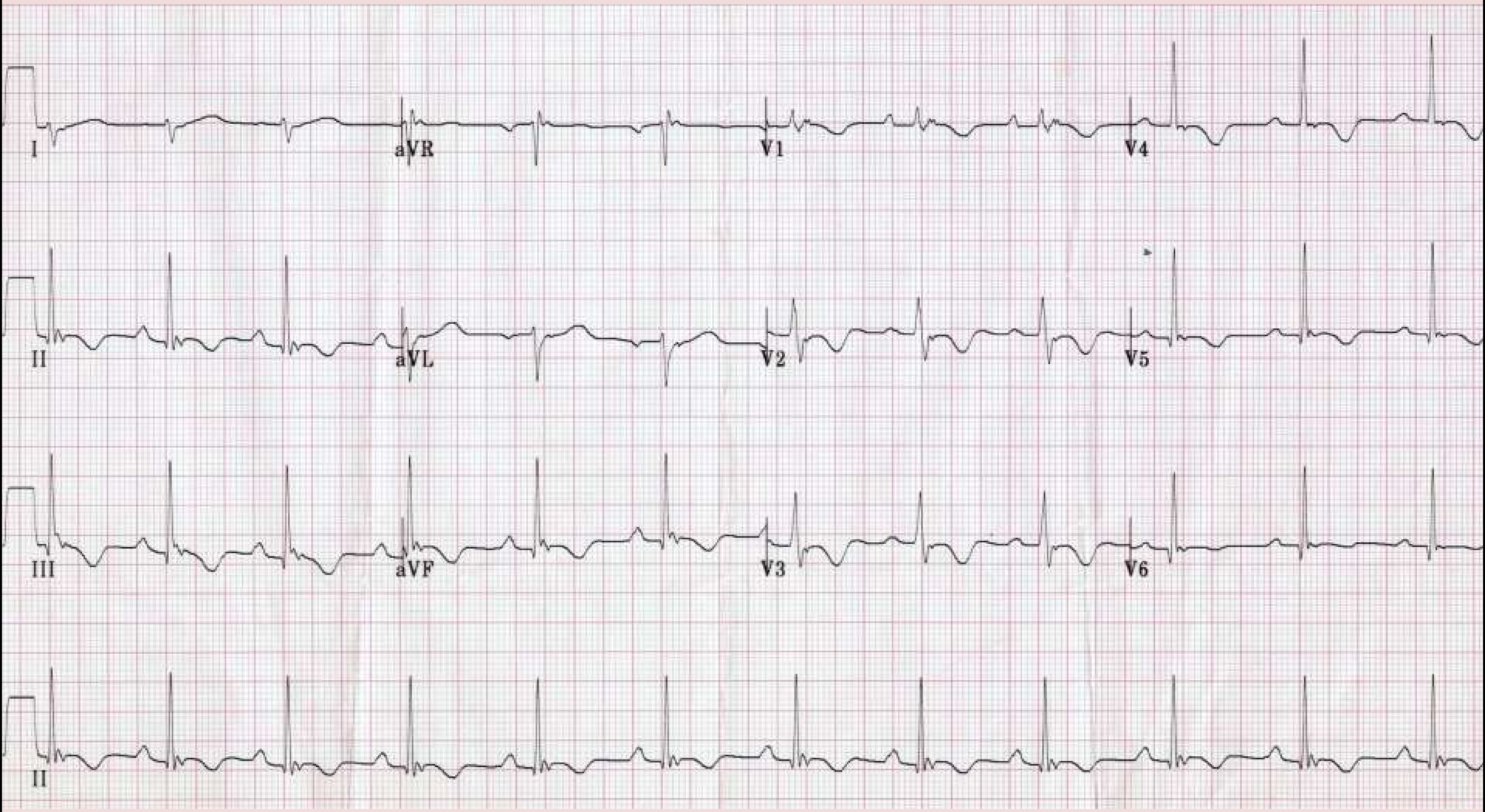
Arrhythmogenic Right Ventricular (RV) Cardiomyopathy and/or Dysplasia:

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

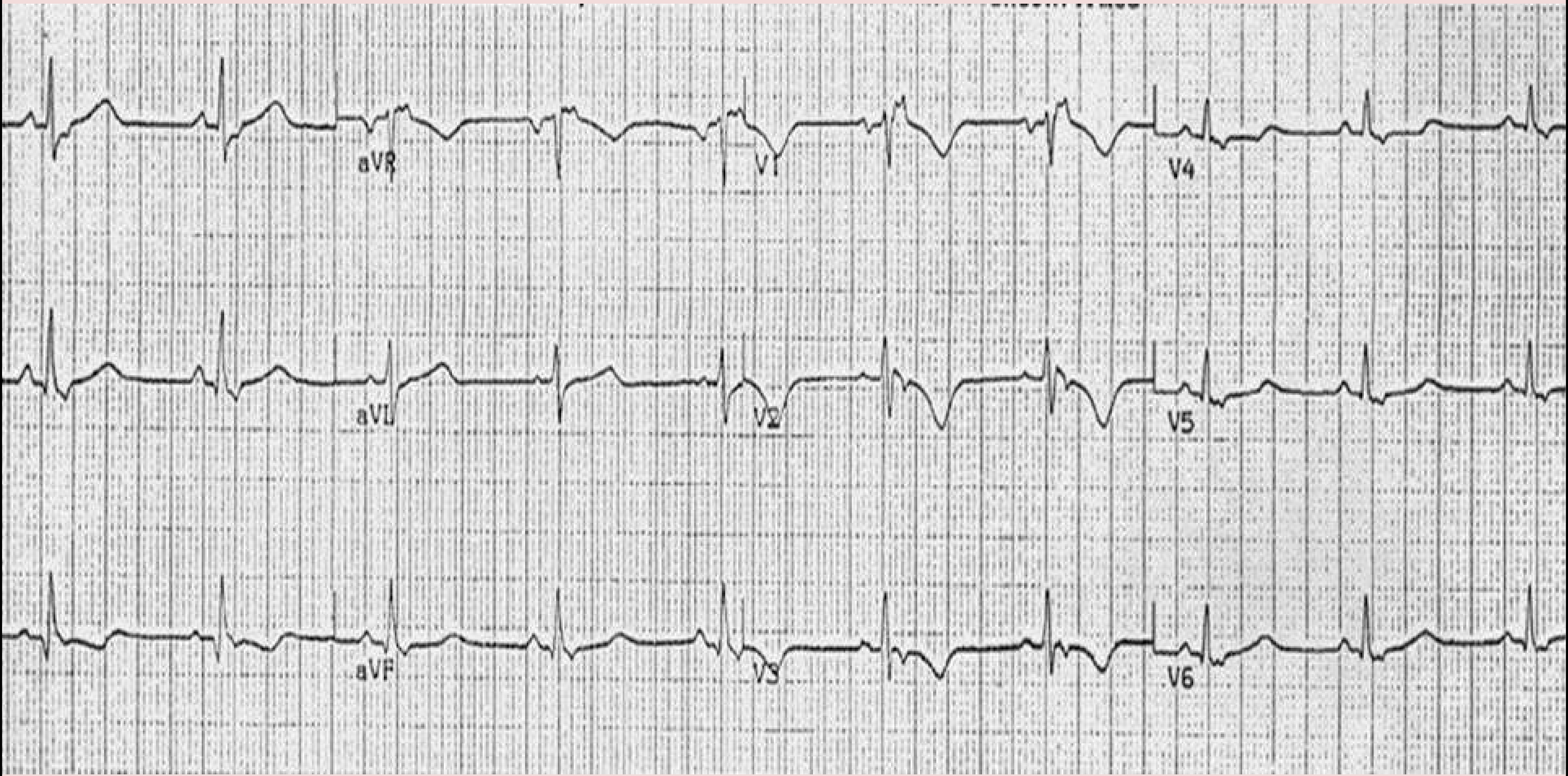


ARVD ECG 1

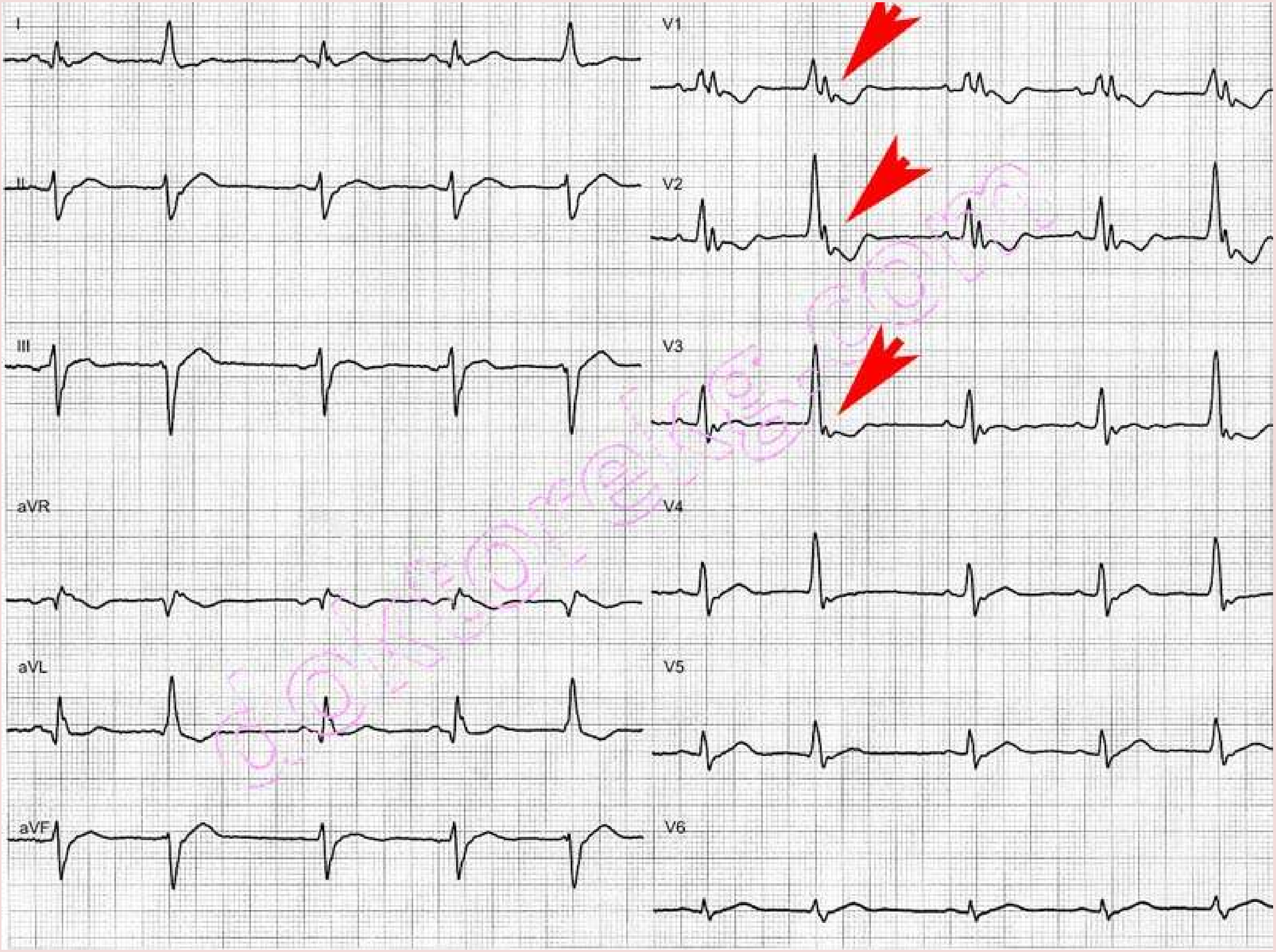


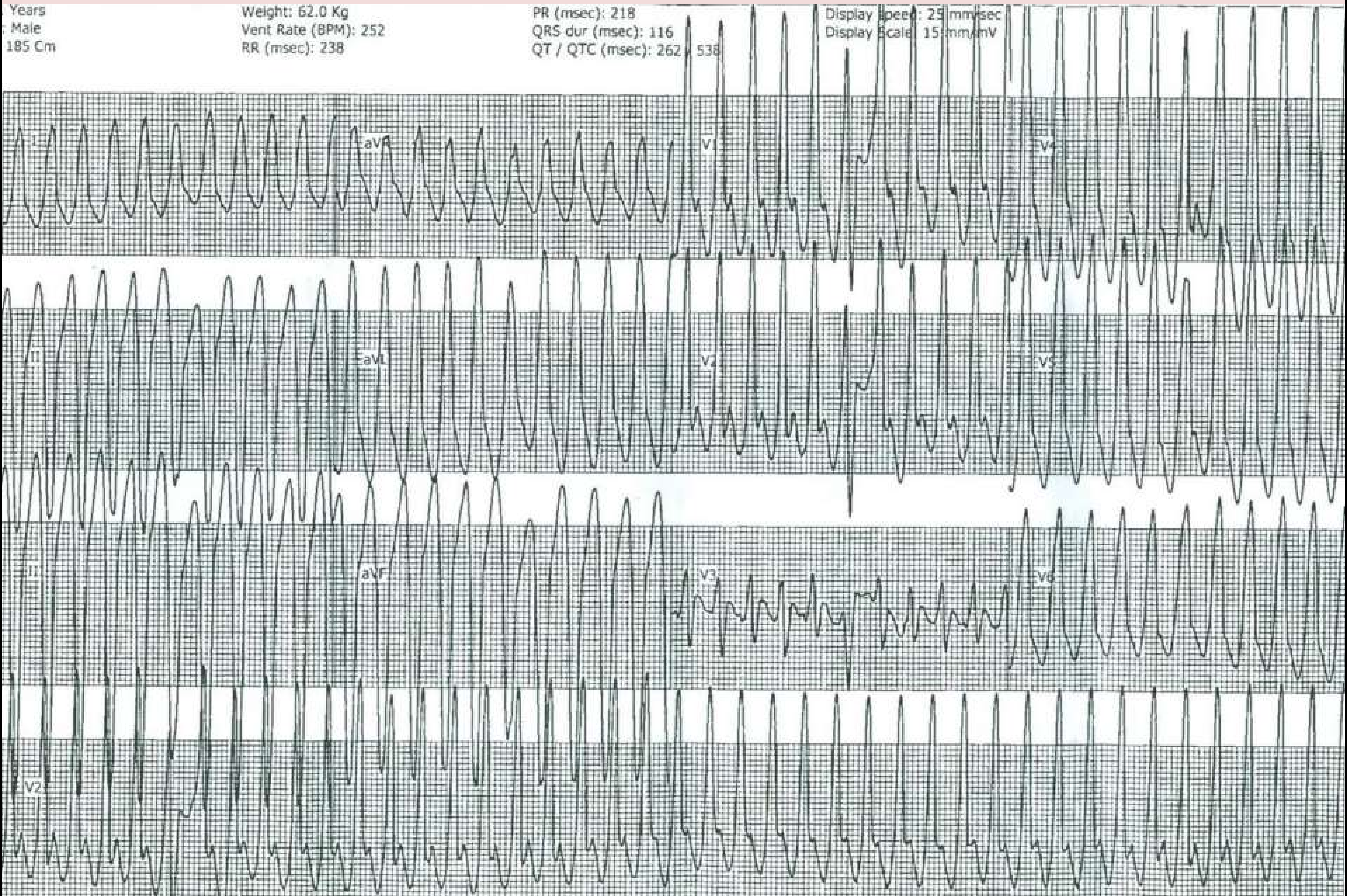
1. "Incomplete RBBB" Pattern
2. V1, V2 Rs pattern
3. Inverted T waves, symmetrical, - Global
4. Epsilon's waves

ARVD ECG 2

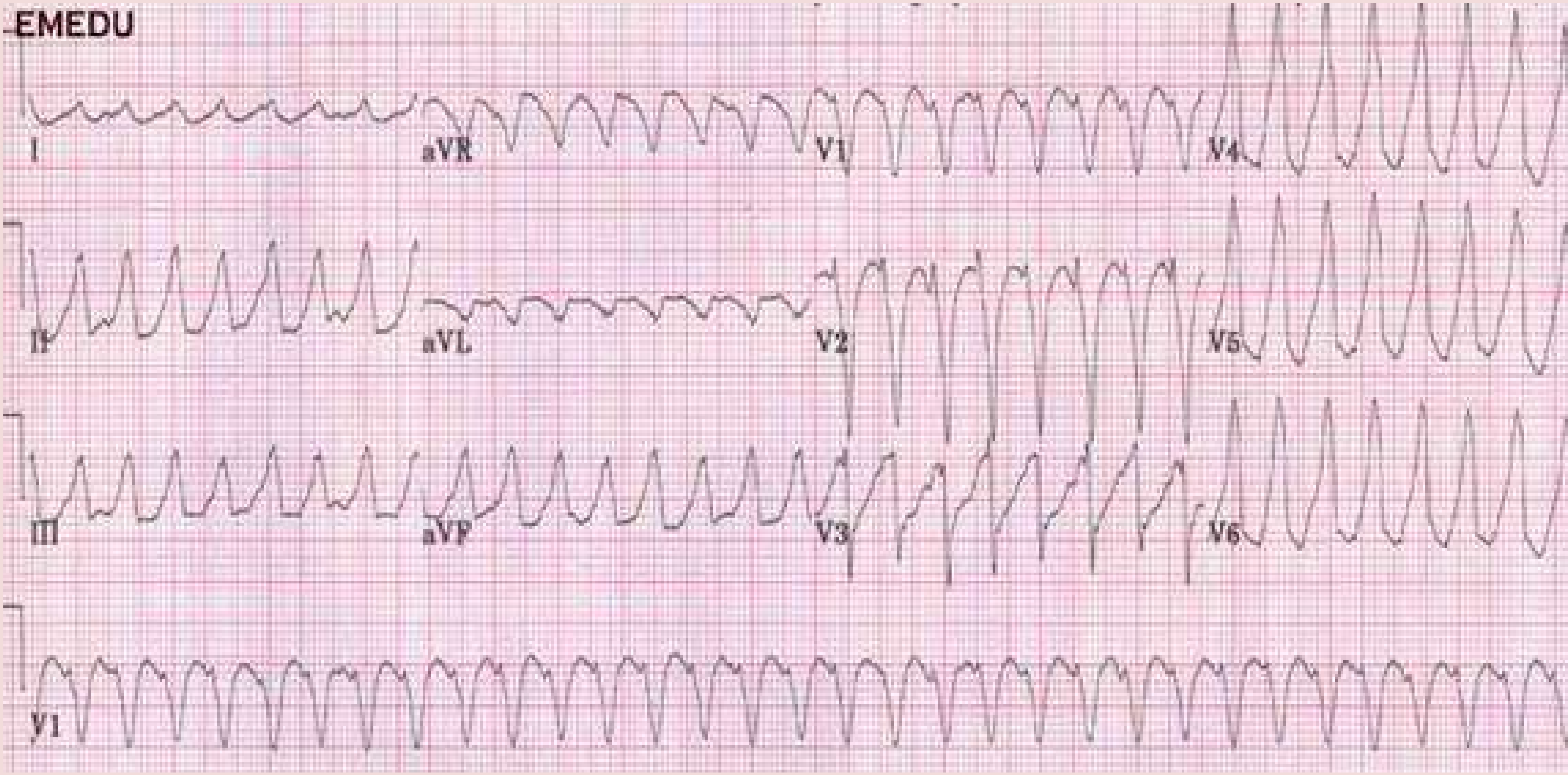


1. "Incomplete RBBB" Pattern
2. V1, V2 Rs pattern
3. Inverted T waves, symmetrical, - Global
4. Epsilon's waves


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


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 Scale
aVR
V1
V4
II
aVL
V2
V5
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

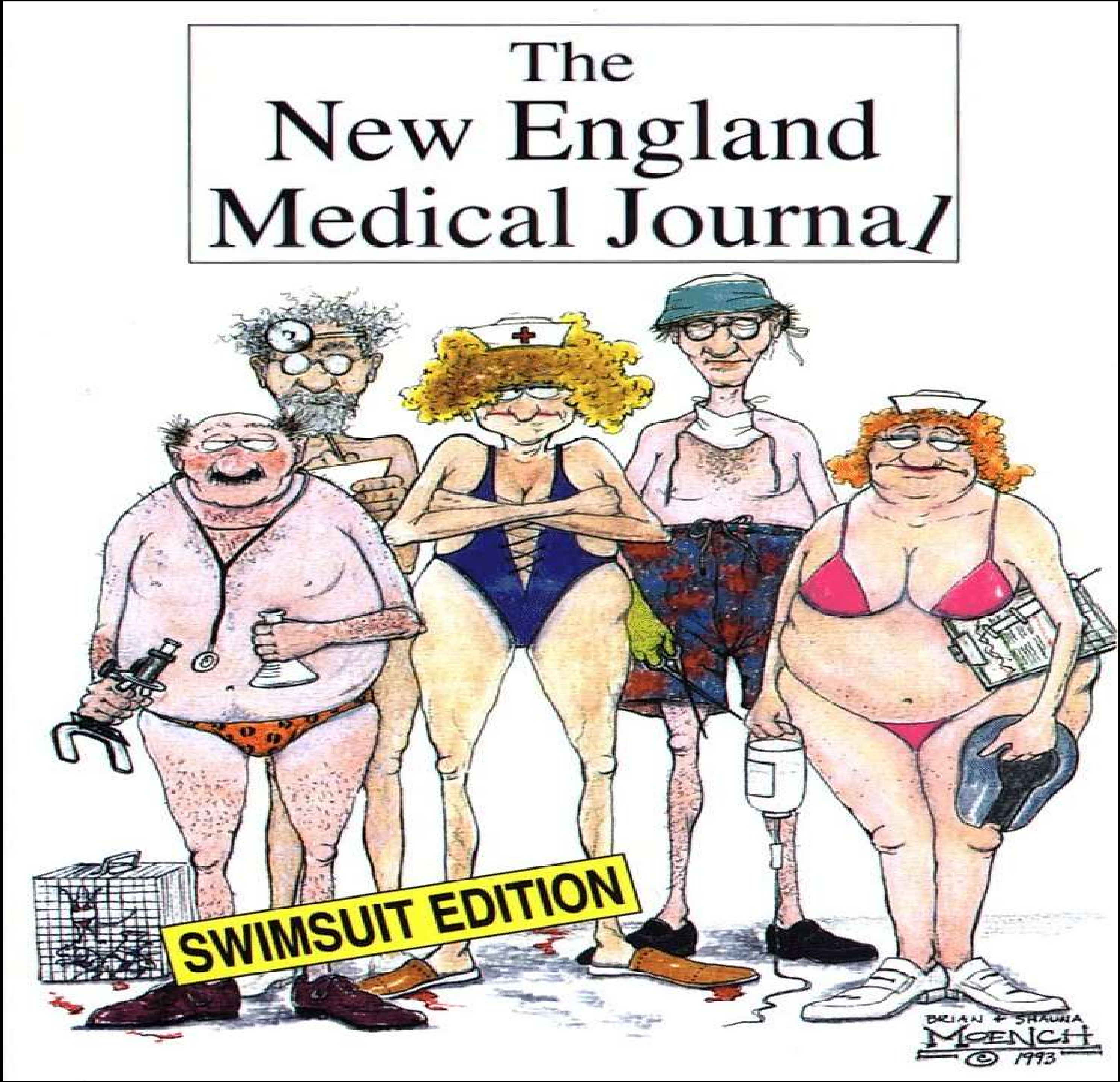
Other Reference Sources:

www.JACC.org

http://circ.ahajournals.org/

SADS
FOUNDATION
SUDDEN ARRHYTHMIA DEATH SYNDROMES

www.SADS.org


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

THE ACUTE CORONARY SYNDROMES

- STEMI
- NSTEMI
- UNSTABLE ANGINA / OBSTRUCTIVE C.A.D.

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

H = chest pain = 2

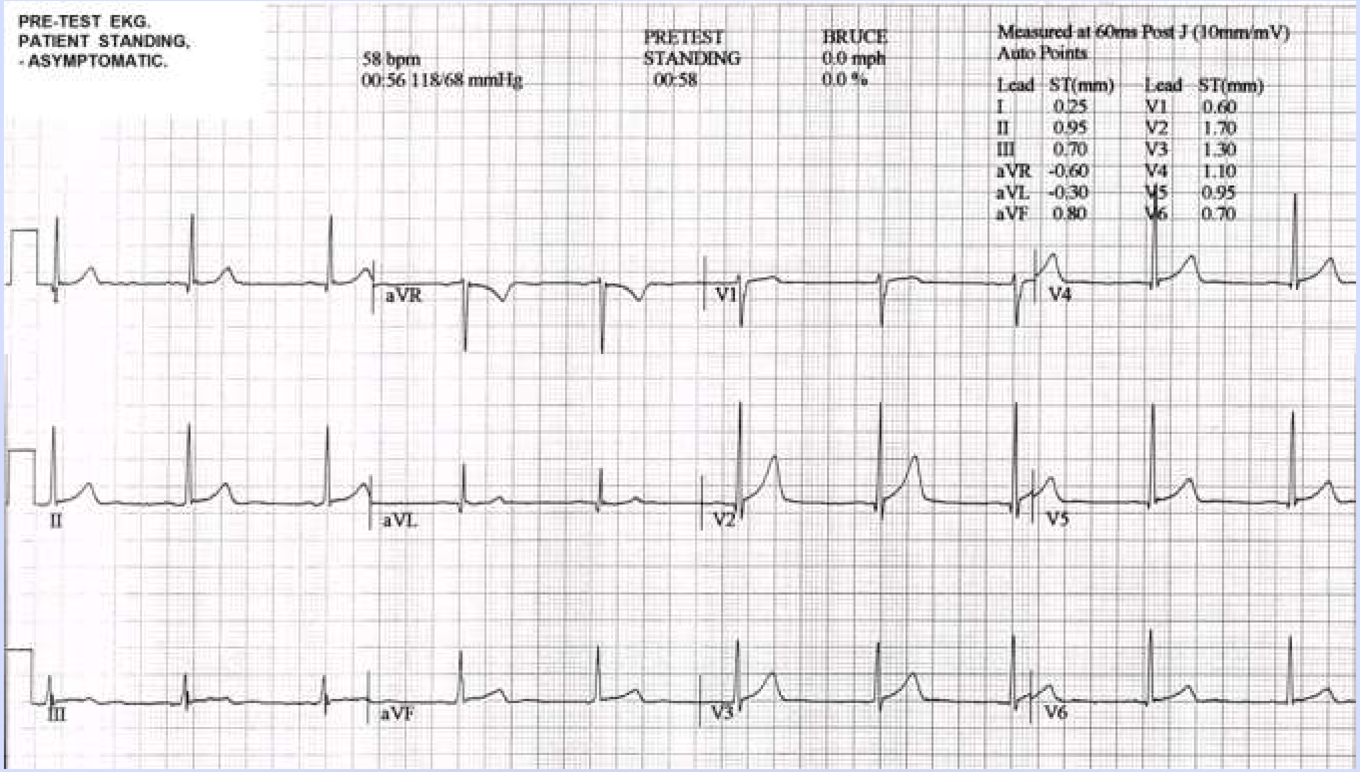
E = ECG normal = 0

A = 63 = 1

R = 3 risk fctors = 2

T = Trop. NL = 0

HEART Score: = 5



2 patients with the above ECG.

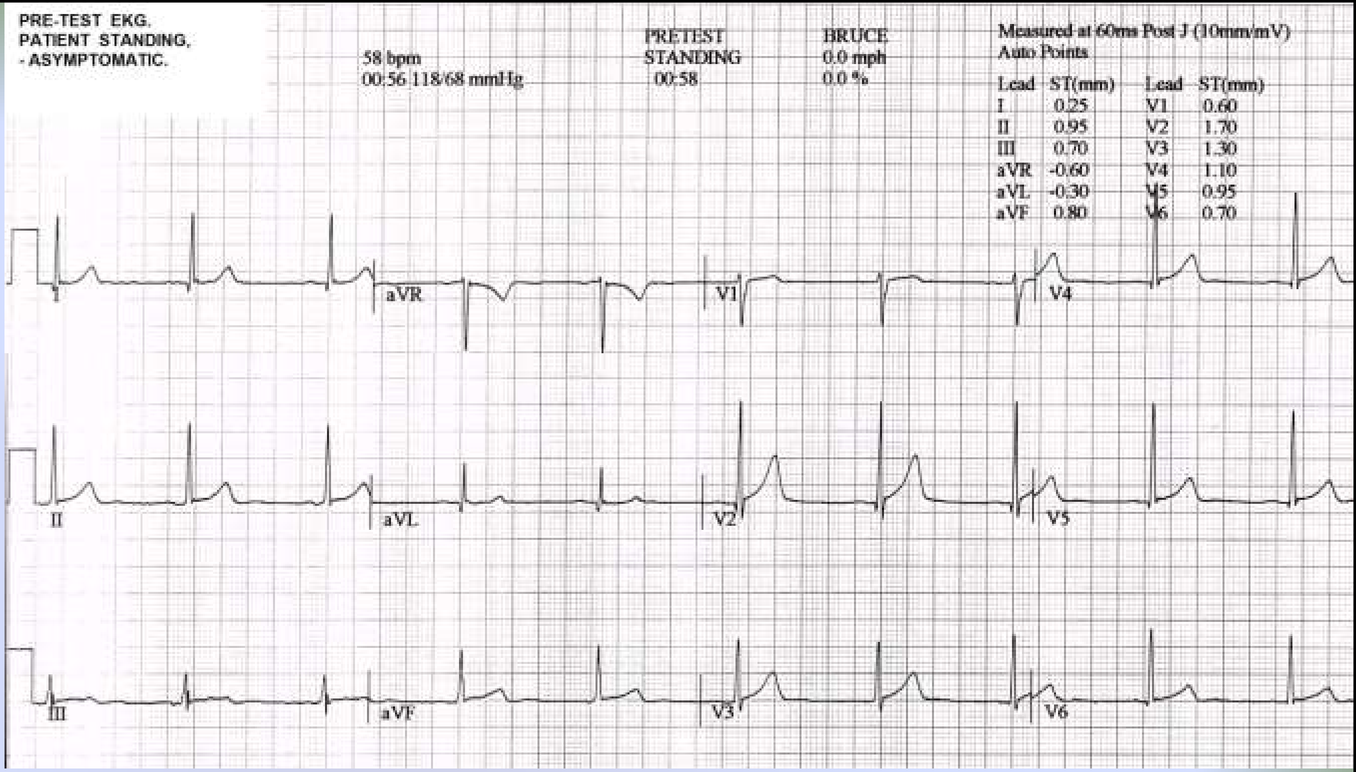
- Patient 1 HEART Score of "0"
- Patient 2 HEART Score of "5"

Should they get the same care ??

PROBLEMS WITH SENSITIVITY . . .

NORMAL ECG.

But . . . . .



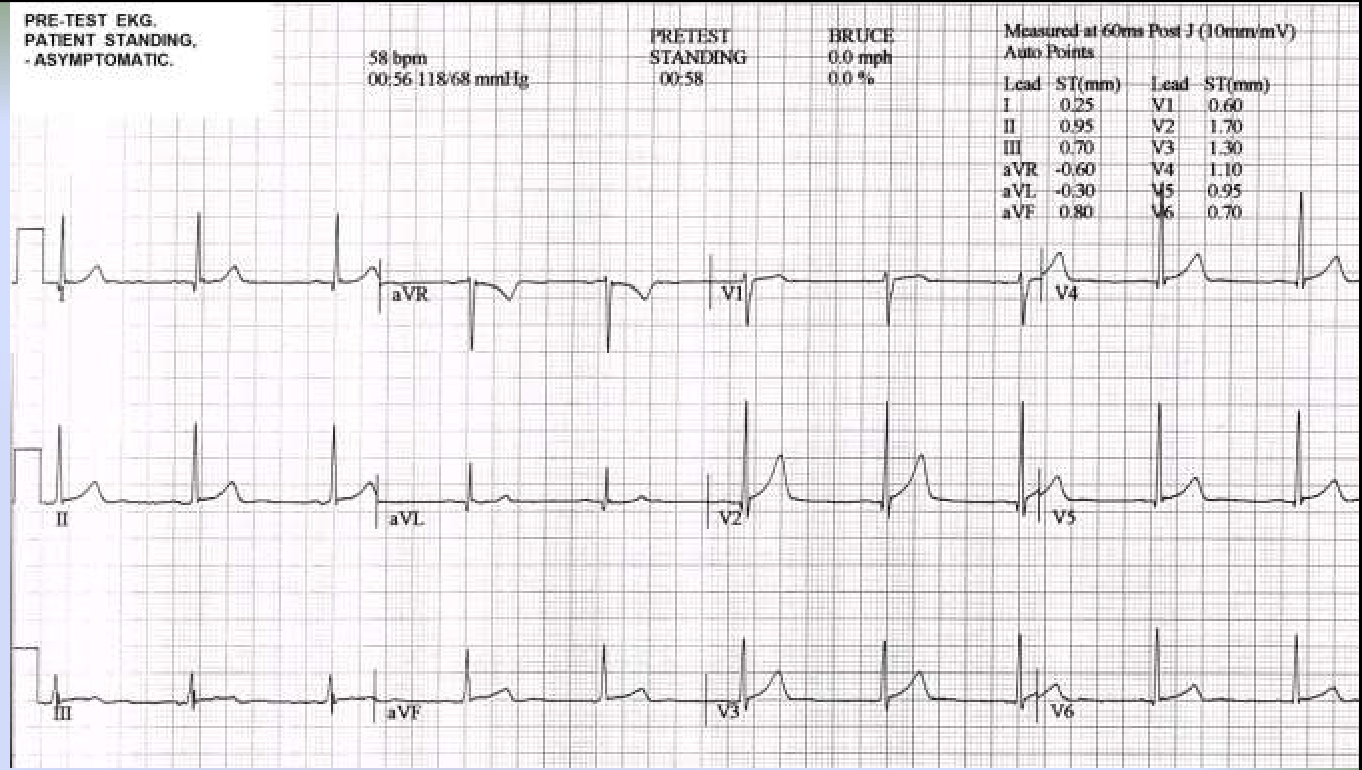
His HEART Score = 5

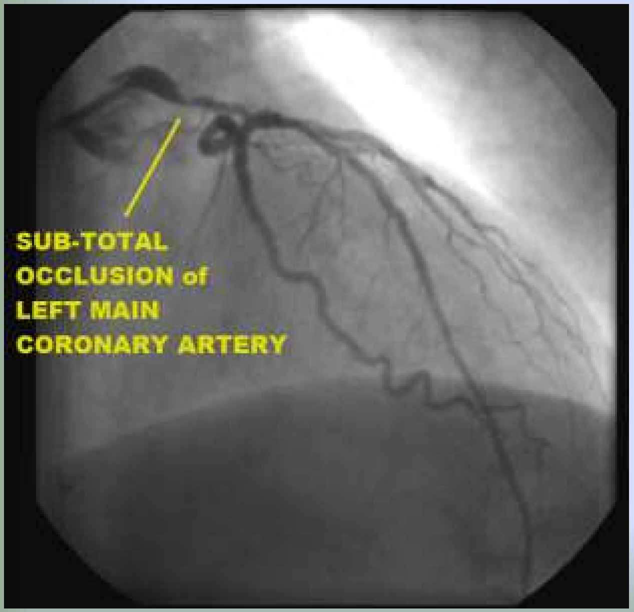
What could that possibly mean?

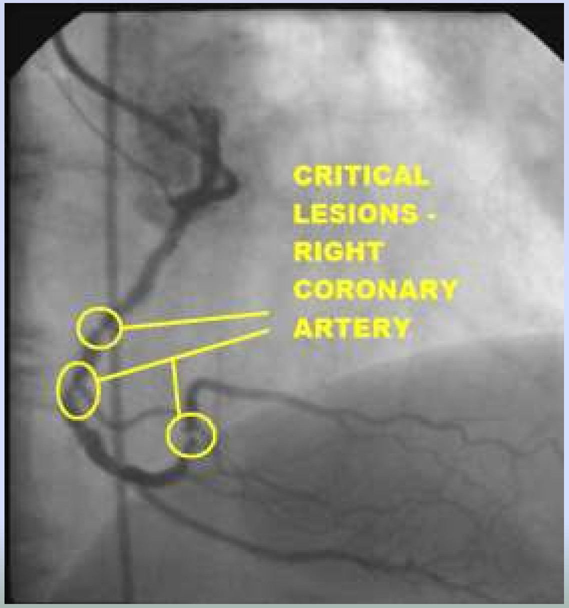
PROBLEMS WITH SENSITIVITY . . .

NORMAL ECG.

But . . . . .



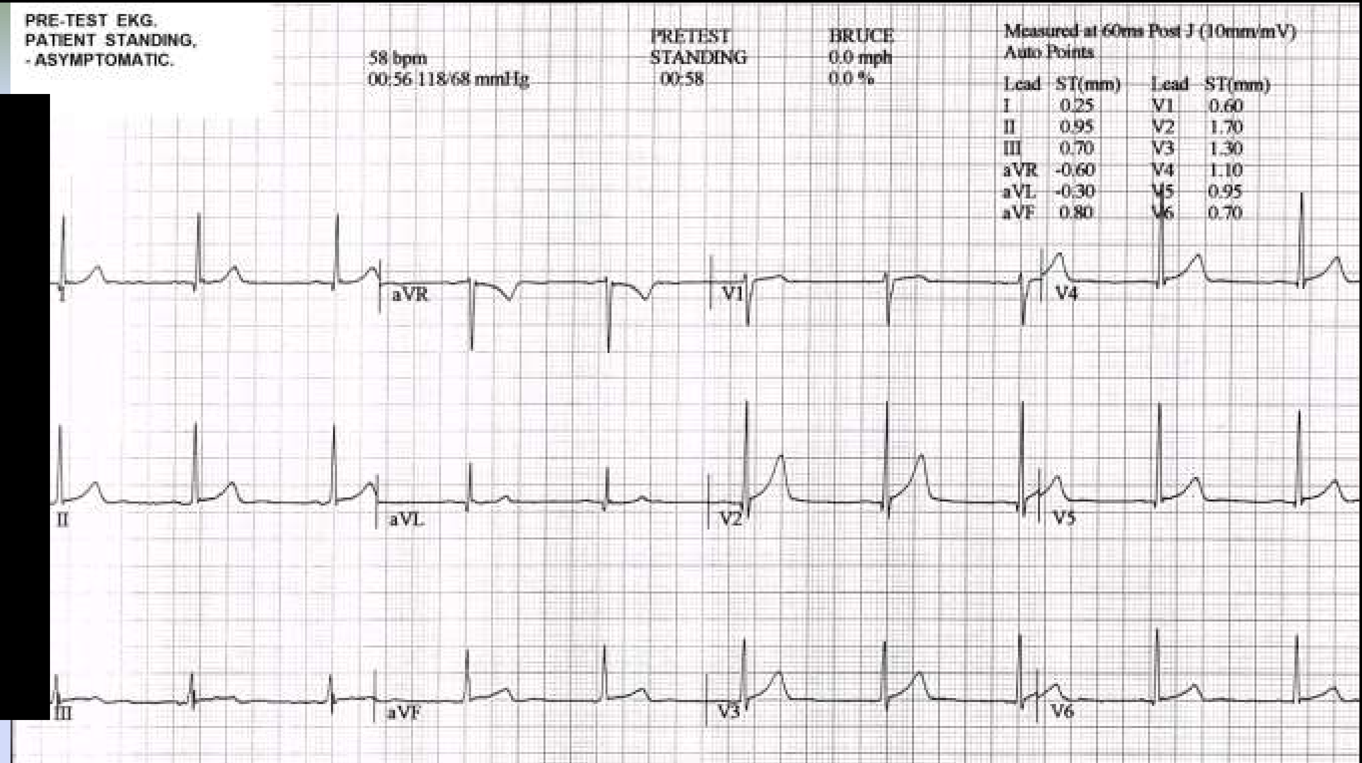


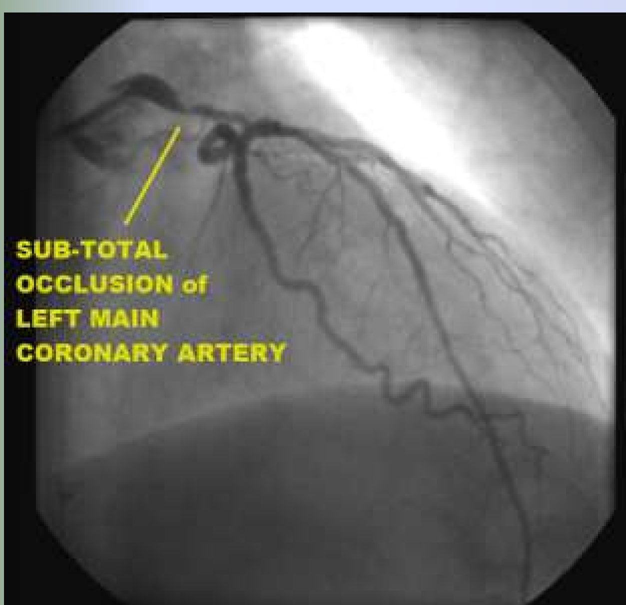


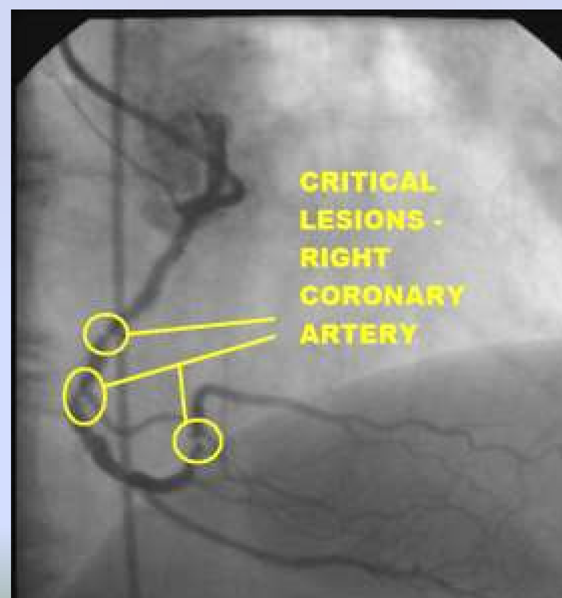
LETHAL TRIPLE VESSEL DISEASE

HEART SCORE:

5







Outcome:

Successful Emergency Bypass Surgery

TYPICAL SYPTOMS of

ACUTE CORNARY SYNDROME:

✓ CHEST PAIN - *DESCRIBED AS . . .*

- "HEAVINESS, PRESSURE, DULL PAIN, TIGHTNESS"
- CENTERED IN CHEST, SUBSTERNAL
- MAY RADIATE TO SHOULDERS, JAW, NECK, LEFT or RIGHT ARM
- NOT EFFECTED by:
  - MOVEMENT
  - POSITION
  - DEEP INSPIRATION

✓ SHORTNESS OF BREATH

- MAY or MAY NOT BE PRESENT

✓ NAUSEA / VOMITING

- MAY or MAY NOT BE PRESENT

INFARCTION

- - - "*Classic Symptoms*" - - -

☞ QUICK ASSESSMENT "SHORT FORM"

- ☑ SUBSTERNAL CHEST PAIN
  (HAVE PATIENT POINT TO WORST PAIN)
- ☑ DESCRIBED AS "DULL PAIN," "PRESSURE," or "HEAVINESS"
- ☑ DOES NOT CHANGE WITH DEEP BREATH

stable angina

1. SYMPTOMS START DURING PHYSICAL EXERTION.
2. SYMPTOMS ARE "PREDICTABLE"

VS.

unstable angina

1. SYMPTOMS MAY START AT ANY TIME, EVEN DURING REST
2. SYMPTOMS ARE *NEW*, *DIFFERENT*, or *WORSE* THAN PREVIOUS EPISODES

BEWARE of the patient with

"INTERMITTENT CHEST PAIN" . . . .

ATYPICAL SYMPTOMS of ACS

? ? ?

Acute MI patients who present without chest pain* are SHREWD:

Stroke (previous history of)
Heart failure (previous history of)
Race (non-white)
Elderly (age 75+)
Women
Diabetes mellitus

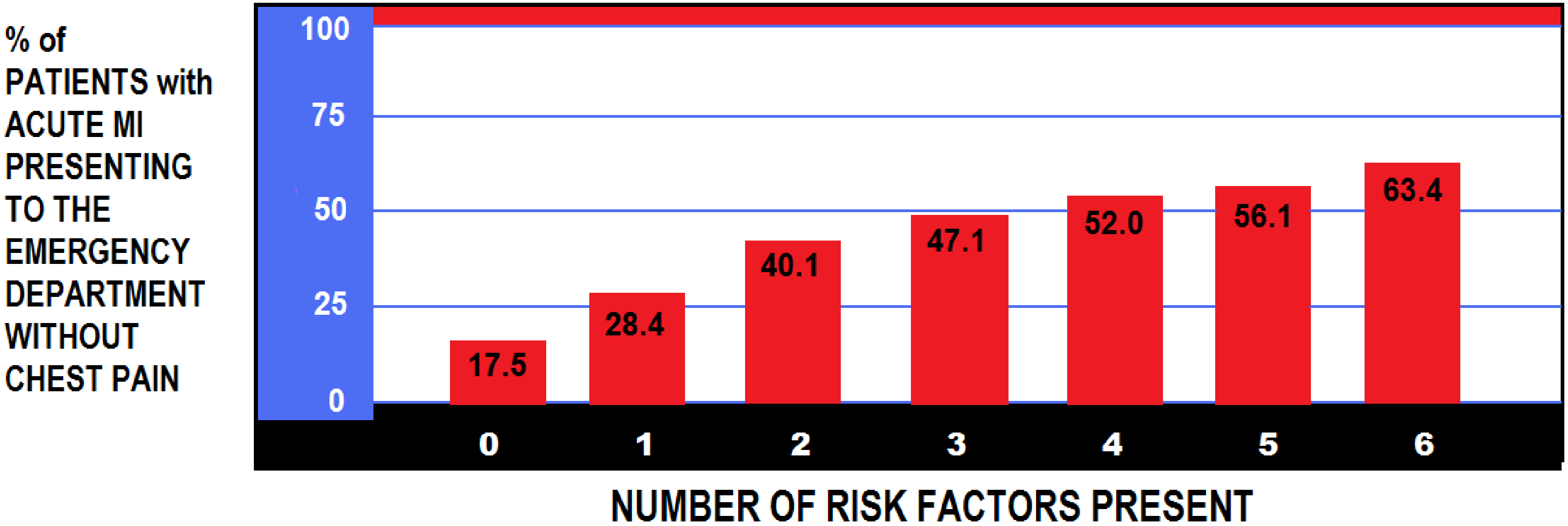
* The information listed in the table to the immediate left resulted from a study conducted by John G. Canto, MD, MSPH, et. al., of the University of Alabama. The study consisted of 434,877 patients diagnosed with AMI between 1994 and 1998 in 1,674 US hospitals. Study results were published in the Journal of the American Medical Association (JAMA) on June 28, 2000, Vol. 283, No. 24, pages 3223-3229

Common atypical complaints associated with AMI without chest pain include:

Malaise (weakness)
Indigestion
Nausea
Dizziness
Syncope

Fatigue
Abdominal pain
Cold sweats
Elevated heart rate
Dsypnea

Effect of Having Multiple Risk Factors for AMI Without Chest Pain



RISK FACTORS INCLUDE: Stroke (previous), Heart failure (previous), Race (non-white), Elderly (age 75+), Women, Diabtetes

DATA SOURCE: J. CANTO, MD, MSPH, et al, JAMA 2000 ; 283 : 3223 - 3229

WOMEN'S MAJOR SYMPTOMS *PRIOR* TO THEIR HEART ATTACK:

▪ UNUSUAL FATIGUE	71 %
▪ SLEEP DISTURBANCE	48 %
▪ SOB	42 %
▪ INDIGESTION	39 %
▪ ANXIETY	36 %

APPROXIMATELY 78 % OF WOMEN REPORTED EXPERIENCING AT LEAST ONE OF THESE SYMPTOMS FOR MORE THAN ONE MONTH EITHER DAILY OR SEVERAL TIMES PER WEEK PRIOR TO THEIR MI.

WOMEN'S MAJOR SYMPTOMS DURING THEIR HEART ATTACK:

SHORTNESS OF BREATH	58 %
WEAKNESS	55 %
UNUSUAL FATIGUE	43 %
COLD SWEAT	39 %
DIZZINESS	39 %

43 % HAD NO CHEST PAIN AT ANY TIME DURING THEIR MI !

Circulation, 2003:108;2619-2623

E = ECG

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

T = Troponin

- **2 Points:** 3 X Normal
- **1 Point:** **>**1 - <3 times normal limit
- **0 Points**: up to normal limit

OBTAINING
THE
12 LEAD ECG

And have it interpreted by a physician or mid-level provider ***…within 10 minutes !***

Evaluating the ECG for ACS:

A TWO-STEP process:

Evaluating the ECG for ACS:

A TWO-STEP process:

STEP 1: Evaluate QRS Width

Evaluating the ECG for ACS:

A TWO-STEP process:

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

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

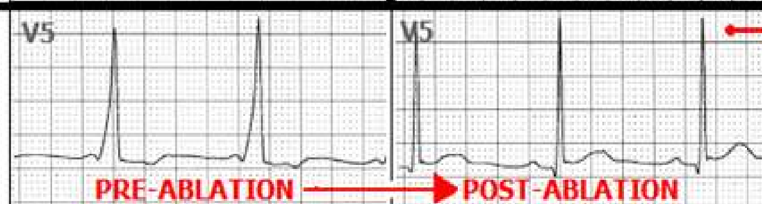
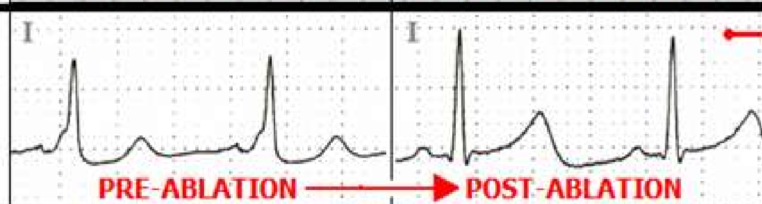
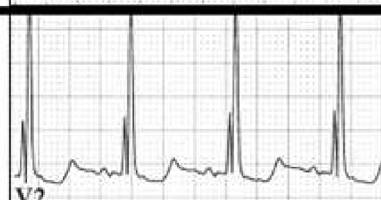
STEP 1 – evaluate QRS width:

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

STEP 1 – evaluate QRS width:

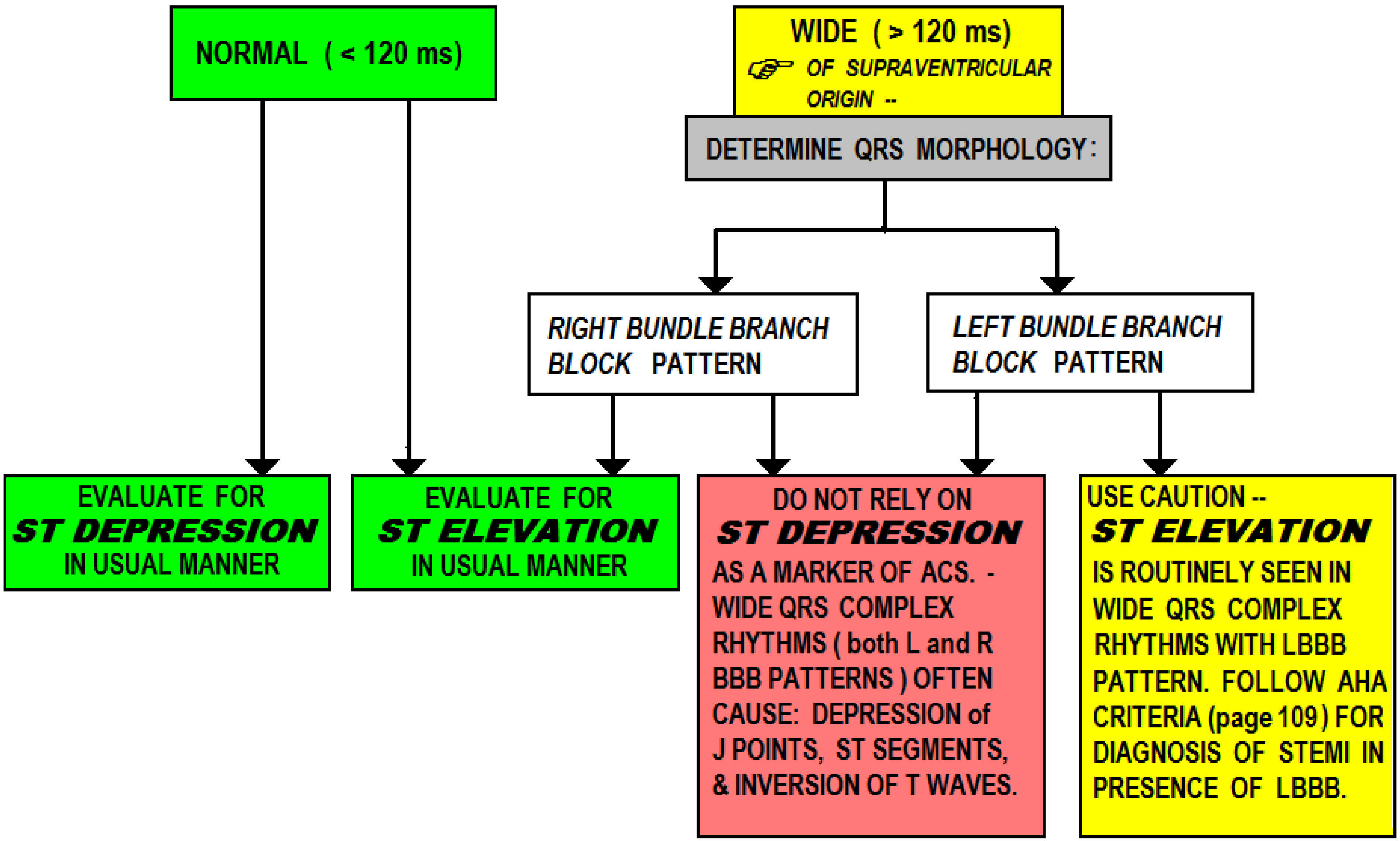
- **QRS is ABNORMALLY WIDE (>120 ms),**
  - **indicates DEPOLARIZATION ABNORMALITY (e.g. "bundle branch block, Wolff-Parkinson-White Syndrome, etc).**
  - **DEPOLARIZATION ABNORMALITIES in turn cause REPOLARIZATION ABNORMALITIES, which alters the: *J Points, ST-Segments and/or T Waves.***

CONDITIONS THAT INCREASE QRS DURATION RESULT IN SECONDARY REPOLARIZATION ABNORMALITIES:

RIGHT BUNDLE BRANCH BLOCK			LEFT BUNDLE BRANCH BLOCK
W-P-W BYPASS TRACT, LEFT LATERAL WALL 49 y/o MALE			SAME PATIENT AS ON LEFT - IMMEDIATELY AFTER RF ABLATION OF BYPASS TRACT
W-P-W BYPASS TRACT, RIGHT ANTERIOR/ LATERAL WALL 14 y/o MALE			SAME PATIENT AS ON LEFT - IMMEDIATELY AFTER RF ABLATION OF BYPASS TRACT
PACEMAKER - RIGHT VENTRICULAR APEX			PACEMAKER TURNED OFF HERE
RIGHT VENTRICULAR HYPERTROPHY (Strain Pattern)			LEFT VENTRICULAR HYPERTROPHY (Strain Pattern)
VENTRICULAR TACHYCARDIA FOCUS: LEFT FASICULAR, 17 y/o FEMALE			VENTRICULAR TACHYCARDIA- FOCUS: RIGHT VENTRICULAR APEX

Evaluating the ECG for ACS:

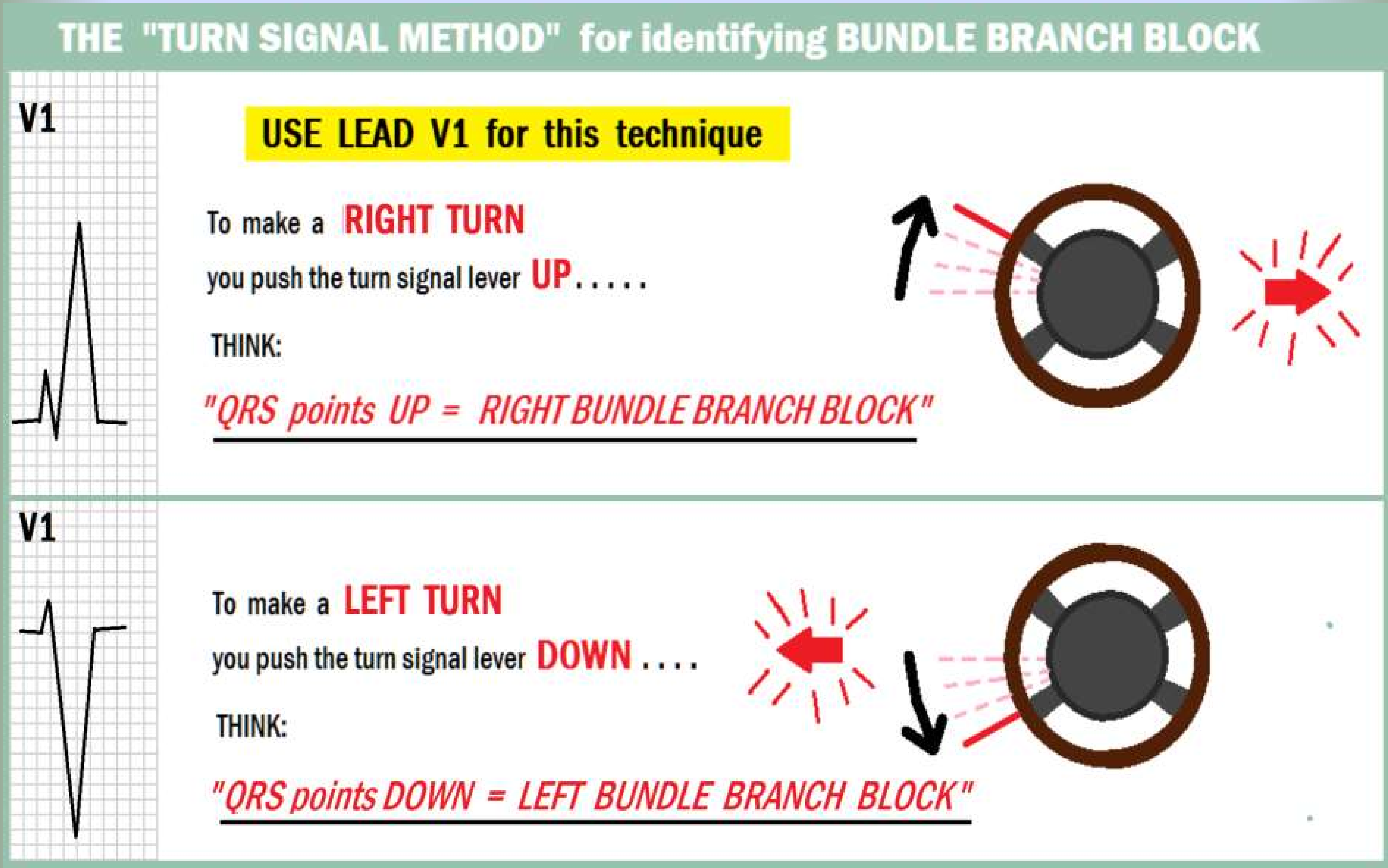
STEP 1 - EVALUATE WIDTH OF QRS:



Wide QRS present: QRSd > 120ms

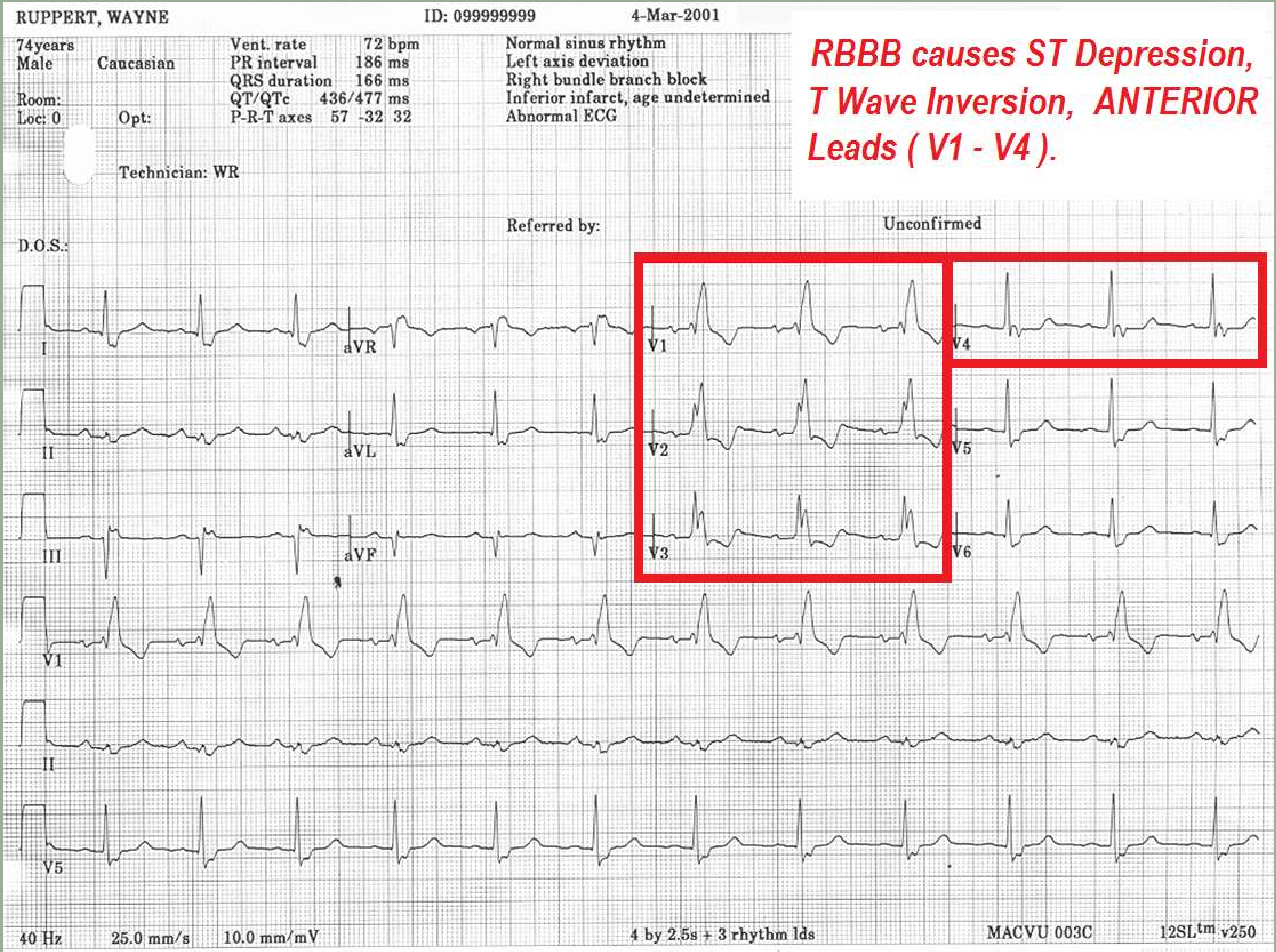
- **Determine RIGHT vs. LEFT Bundle Branch Block Pattern**

Simple "Turn Signal Method" . . .



Wide QRS present:
(QRSd > 120ms)

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


RBBB causes ST Depression, T Wave Inversion, ANTERIOR Leads (V1 - V4).
RUPPERT, WAYNE
ID: 099999999
4-Mar-2001
74years
Male
Caucasian
Room:
Loc: 0
Opt:
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
Technician: WR
Referred by:
Unconfirmed
D.O.S.:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
40 Hz
25.0 mm/s
10.0 mm/mV
4 by 2.5s + 3 rhythm lds
MACVU 003C
12SLtm v250

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

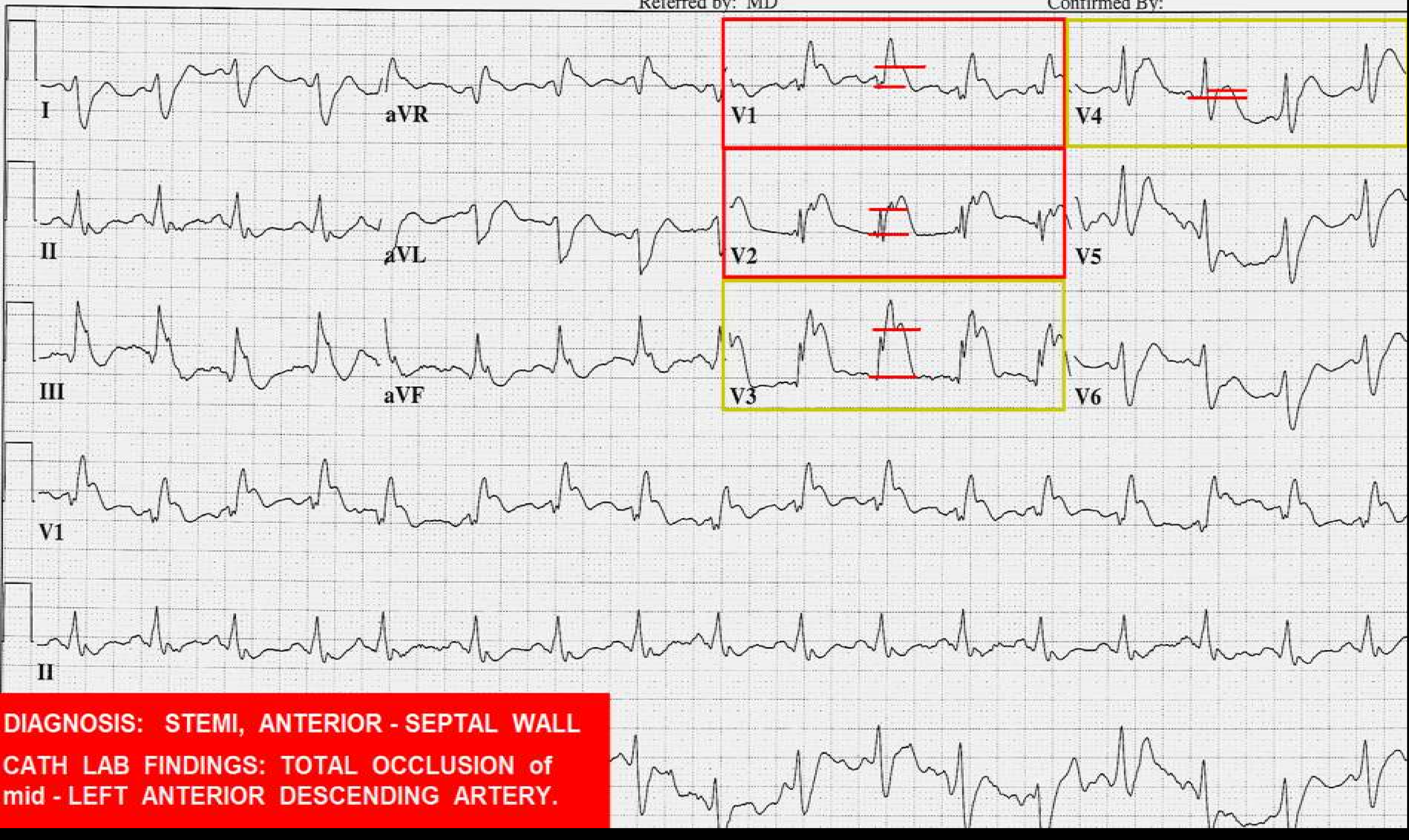
Room:ATL
Loc:3 Option:23

Technician: W Ruppert

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 ...

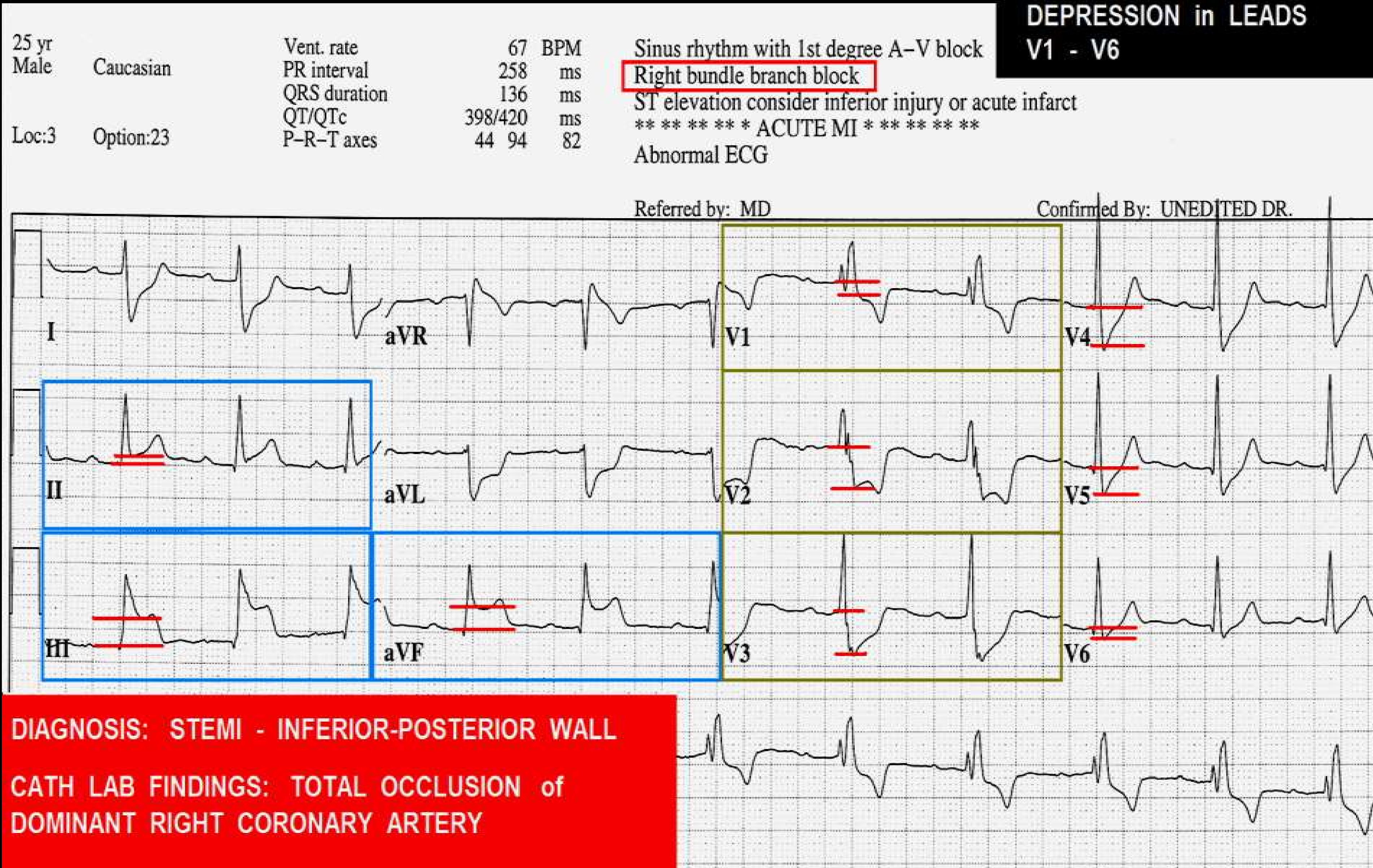
Referred by: MD Confirmed By:



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
Male Caucasian

Room:CS-19
Loc:6 Option:41

Vent. rate	110	BPM
PR interval	170	ms
QRS duration	148	ms
QT/QTc	366/495	ms
P-R-T axes	57 19	69

Sinus tachycardia
Right bundle branch block
Lateral infarct , possibly acute
Inferior infarct , possibly acute
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***

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

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- ***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*

A.H.A. ACLS GUIDELINES

1. If patient has a CONFIRMED HISTORY of LBBB, rely on:
   - CARDIAC MARKERS
   - SYMPTOMS
   - RISK FACTOR PROFILE
   - HIGH INDEX OF SUSPICION

   for diagnosis of STEMI

2. If patient has:

   a) previously NORMAL ECGs (no LBBB)

   -- or --

   b) no old ECGs available for comparison

   consider diagnosis as STEMI until proven otherwise.

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:

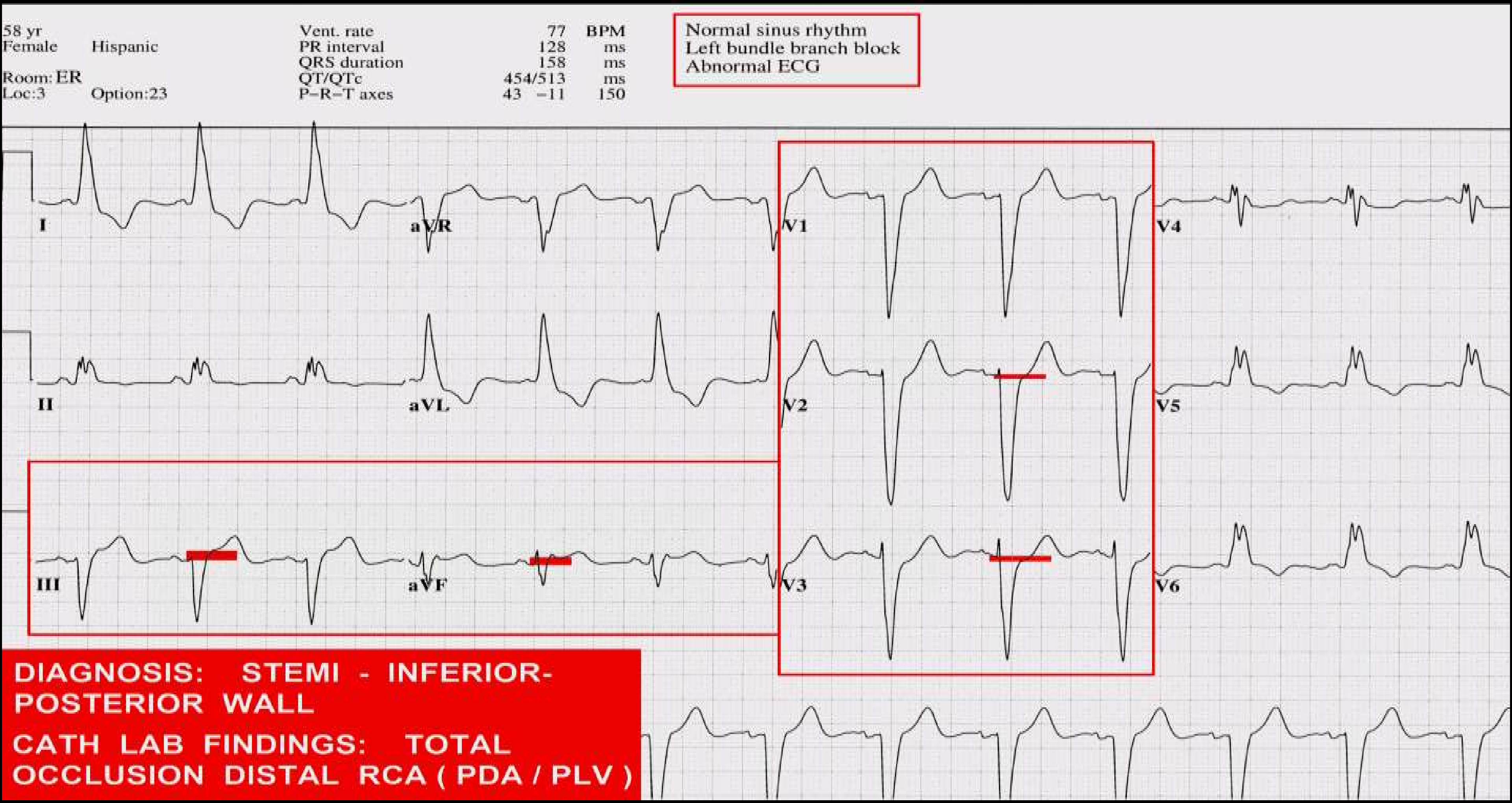
HELPFUL INDICATORS FOR ECG DIAGNOSIS OF STEMI in the presence of LBBB:

- ST ELEVATION > 5 mm
- COMPARE J POINT, ST SEGMENTS and T WAVES of previous ECG with LBBB to NEW ECG.
- CONVEX ST SEGMENT = poss. MI
  CONCAVE ST SEGMENT = normal
- CONCORDANT ST changes (1 mm or > ST DEPRESSION V1 - V3 or ST ELEVATION LEADS II, III, AVF)
- ST ELEVATION in LEADS II, III, and/or AVF

N. ENGL. J. MED v 348; p933 - 940 - Zimetbaum, et. al.

“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

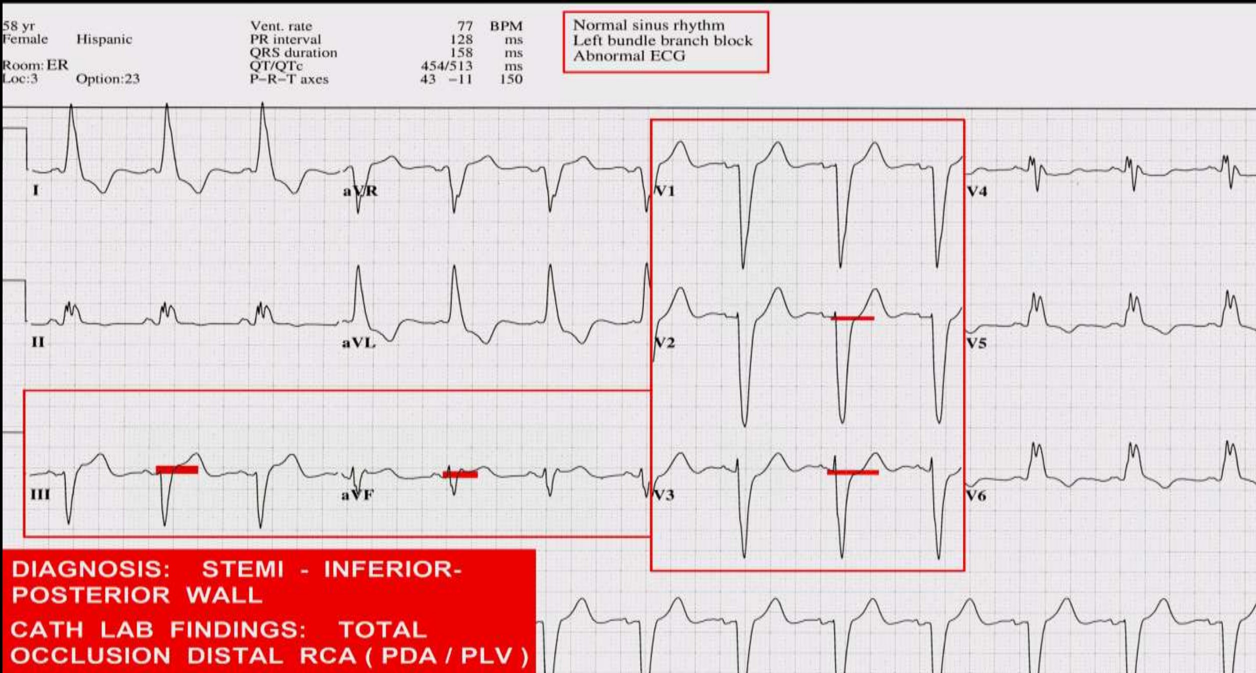
LBBB with CHEST PAIN - CASE 1: PRESENTING EKG



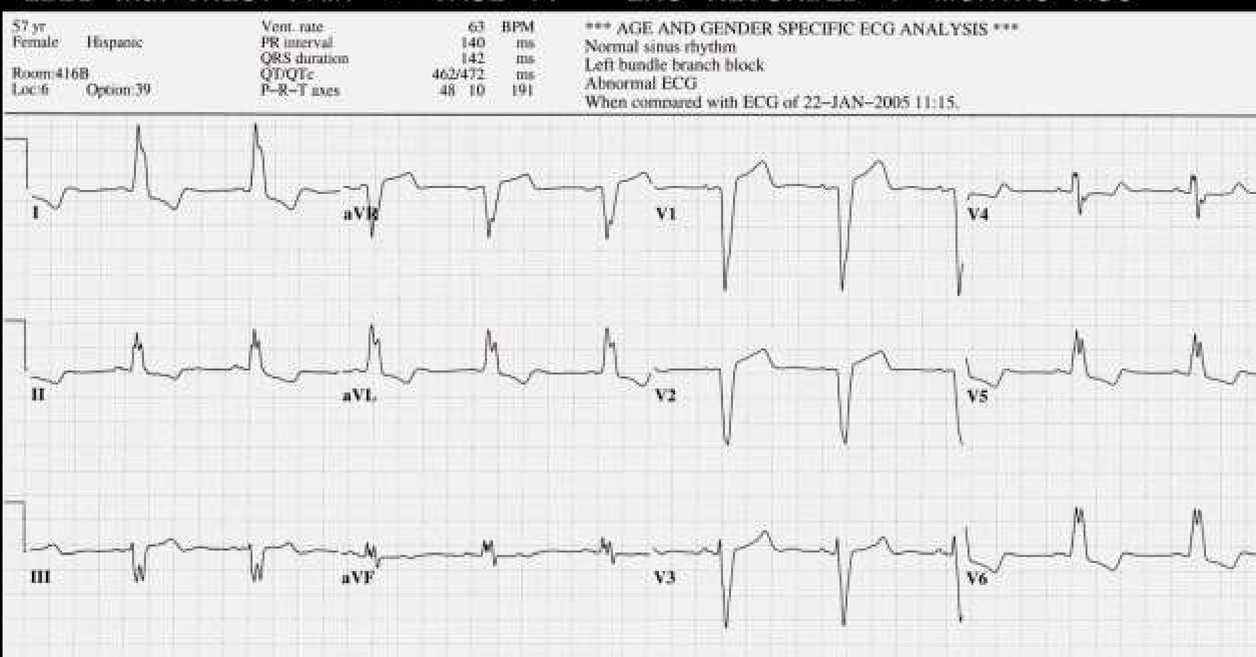
DIAGNOSIS: STEMI - INFERIOR-POSTERIOR WALL

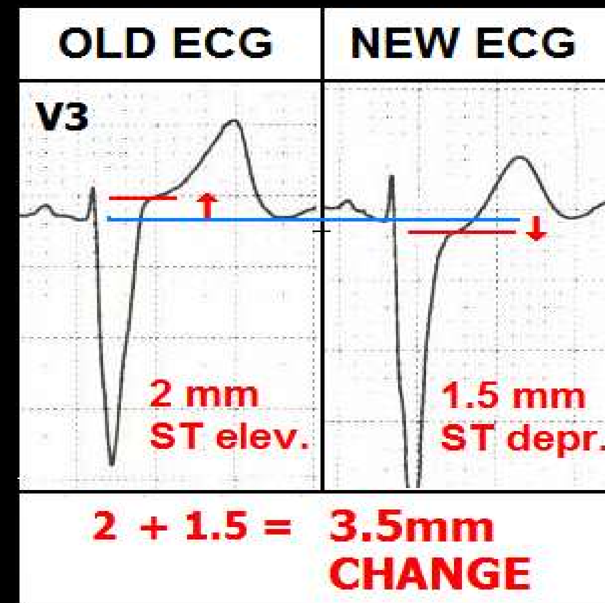
CATH LAB FINDINGS: TOTAL OCCLUSION DISTAL RCA (PDA / PLV)

LBBB with CHEST PAIN - CASE 1: PRESENTING EKG

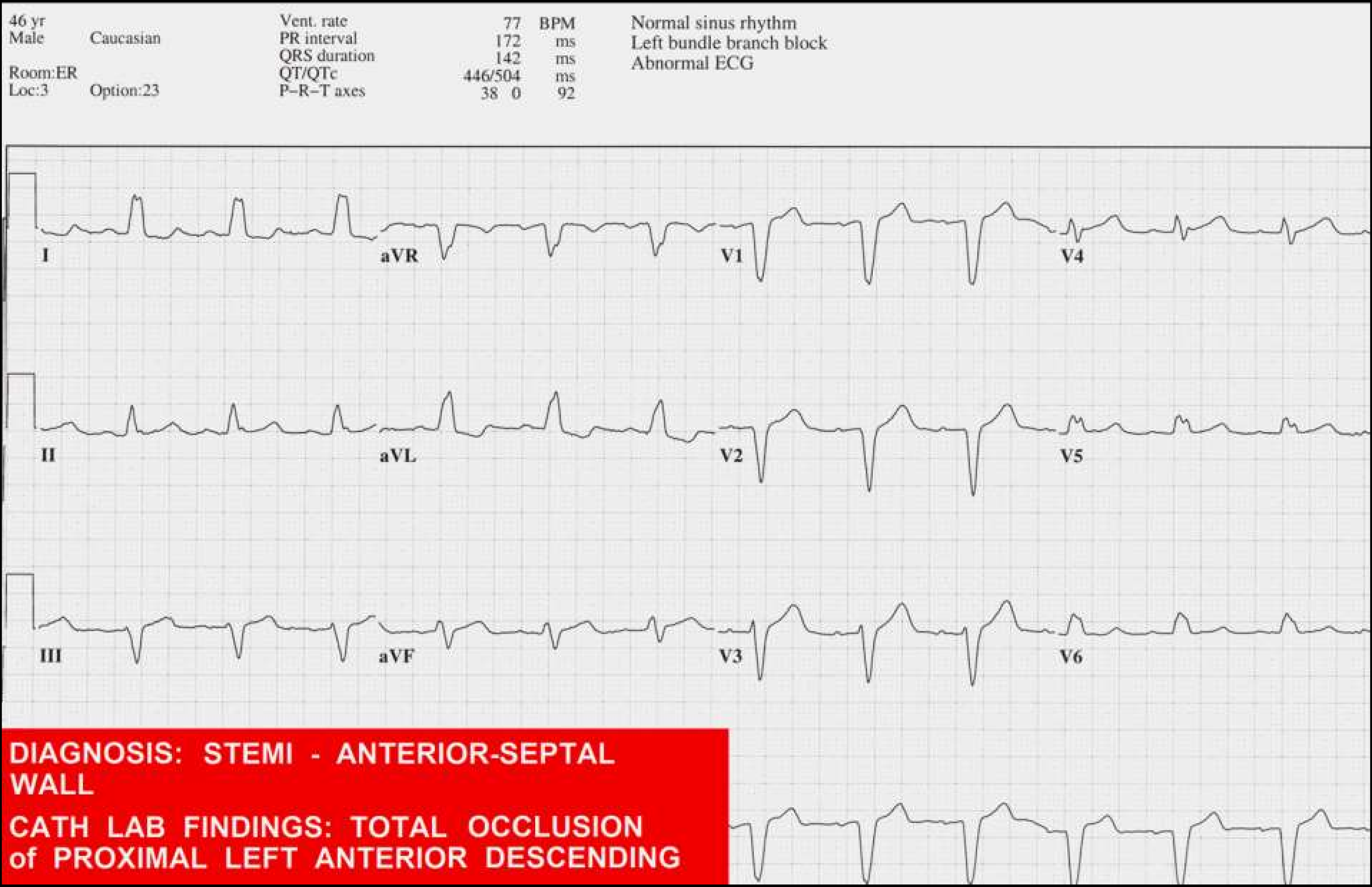


LBBB with CHEST PAIN - CASE 1: EKG RECORDED 7 MONTHS AGO





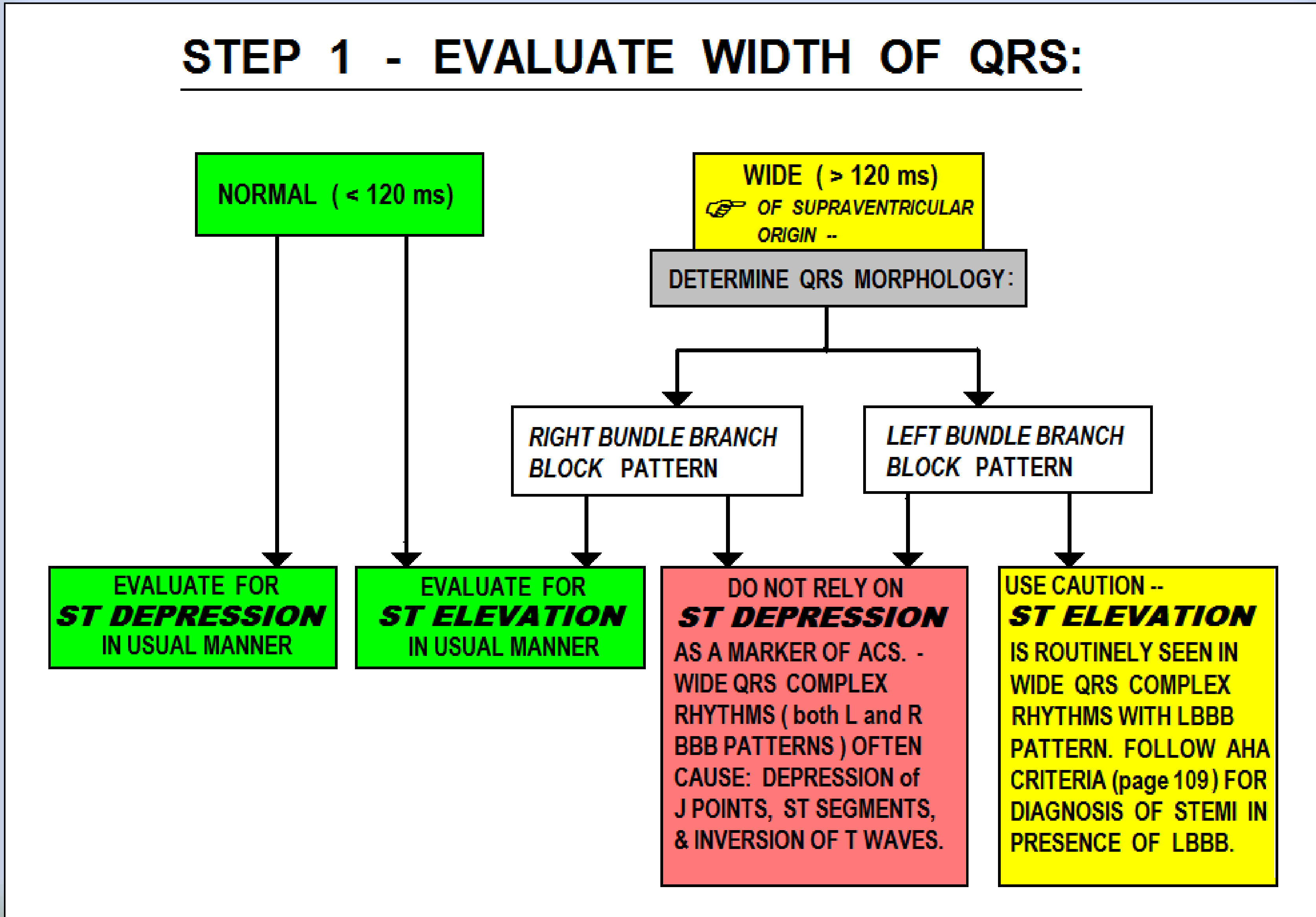
LBBB with CHEST PAIN - CASE 2: *NEW ONSET of LBBB*



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)

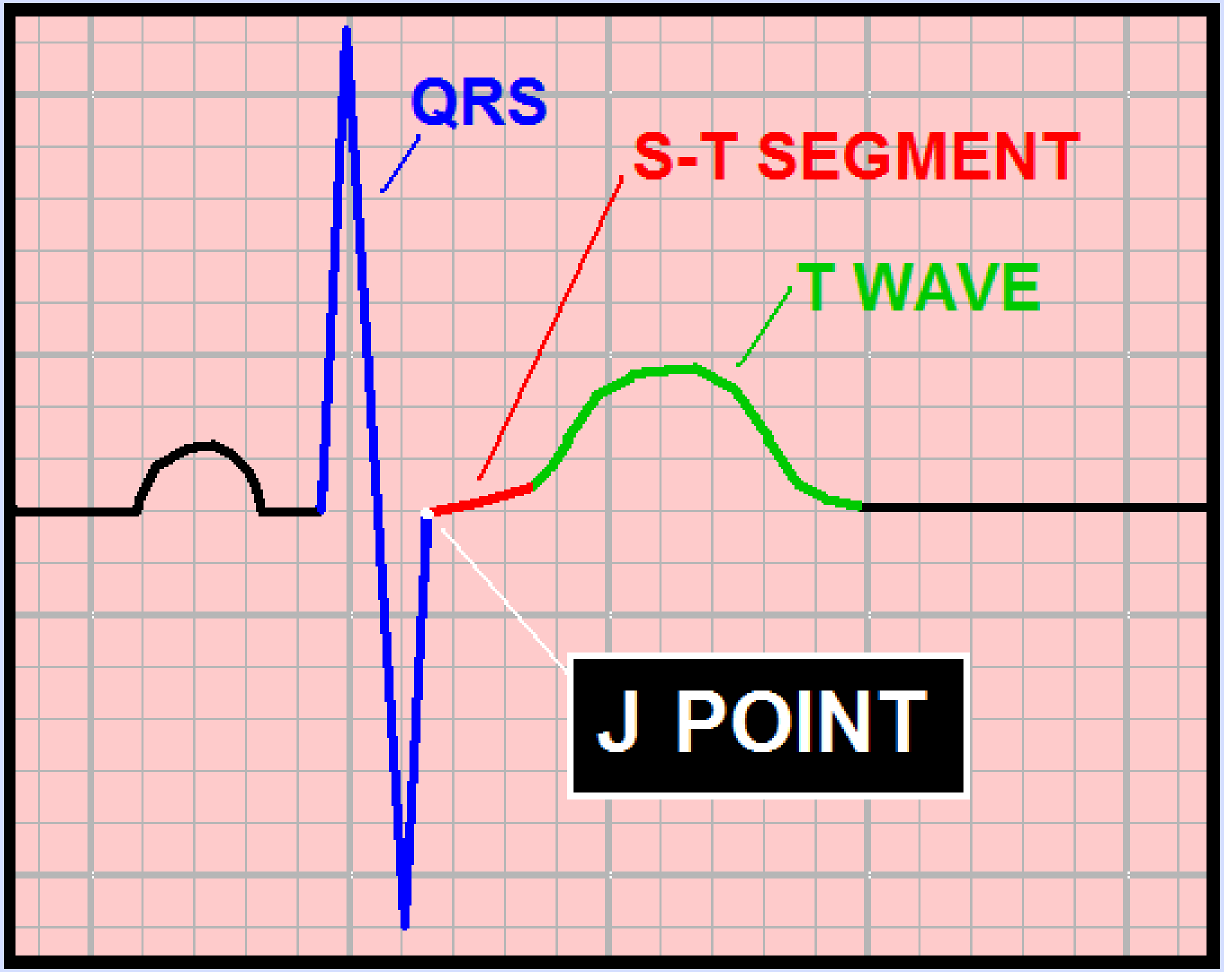
STEP 2 - EVALUATE the EKG for ACS

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

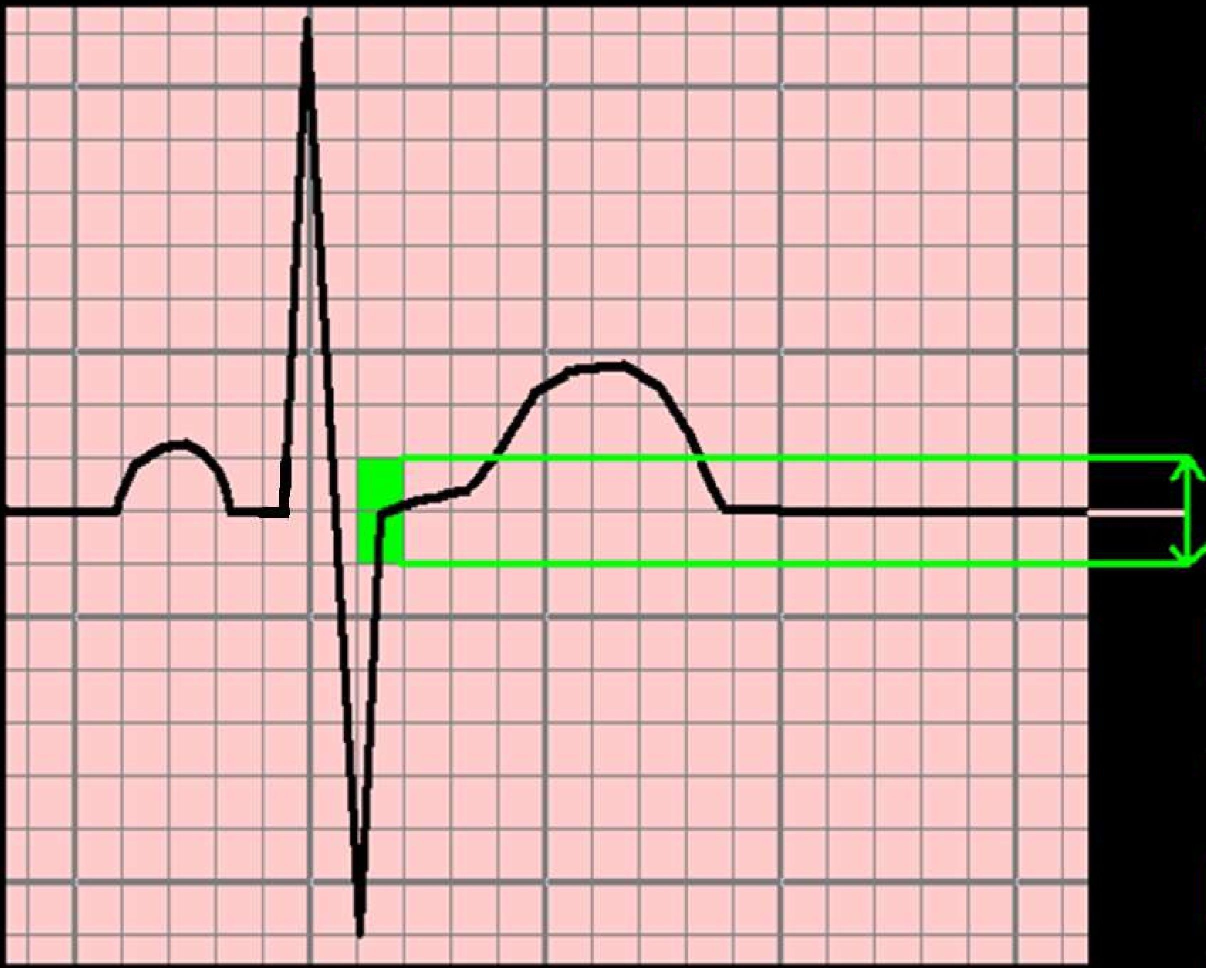
- WHEN QRS WIDTH IS NORMAL (< 120 ms)

ASSESS:

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

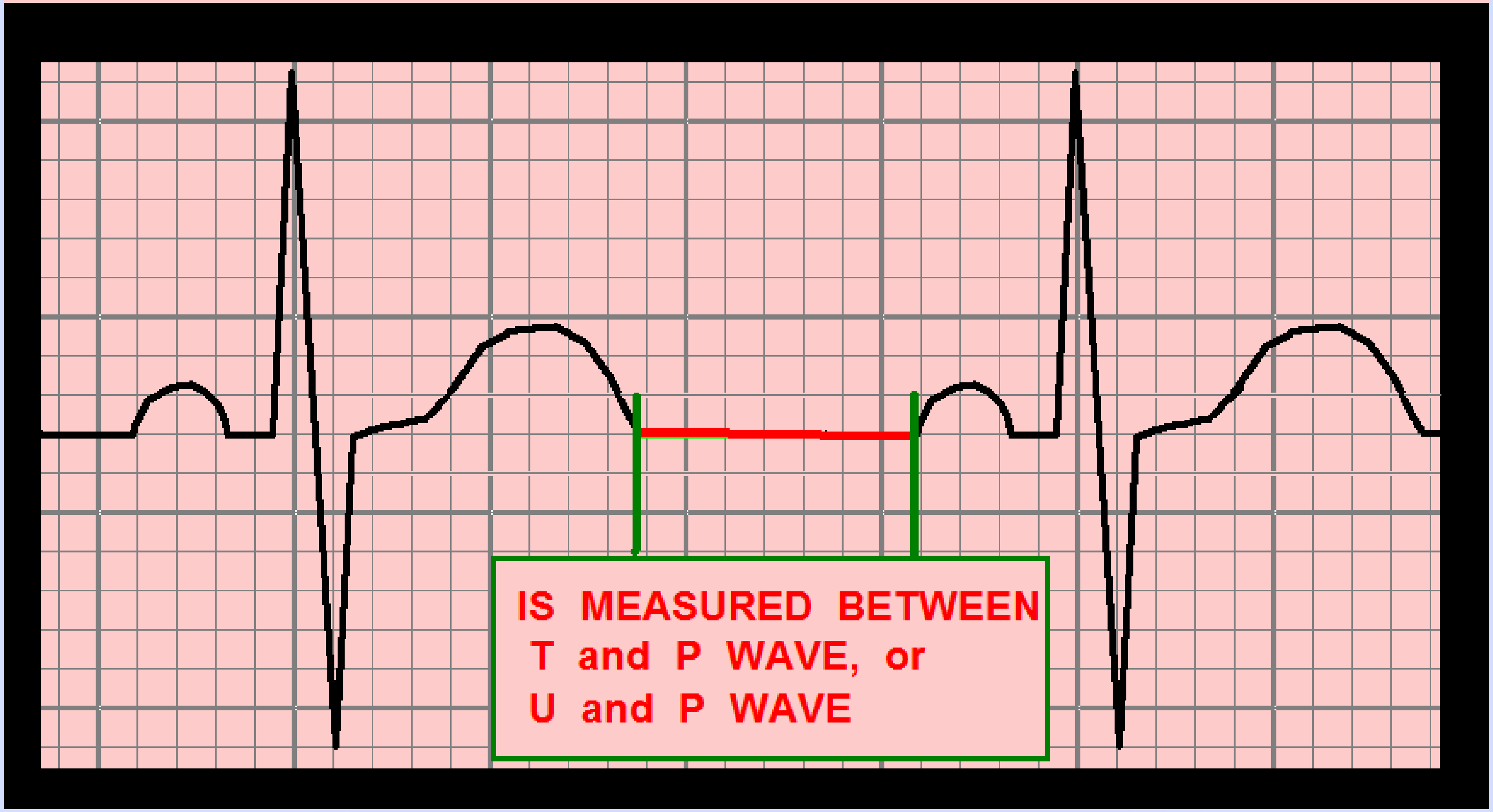
☞ *in EVERY LEAD EXCEPT aVR ! !*

THE J POINT SHOULD BE . .



WITHIN
1 mm
ABOVE
OR
BELOW
the
ISOELECTRIC
LINE

THE ISOELECTRIC LINE



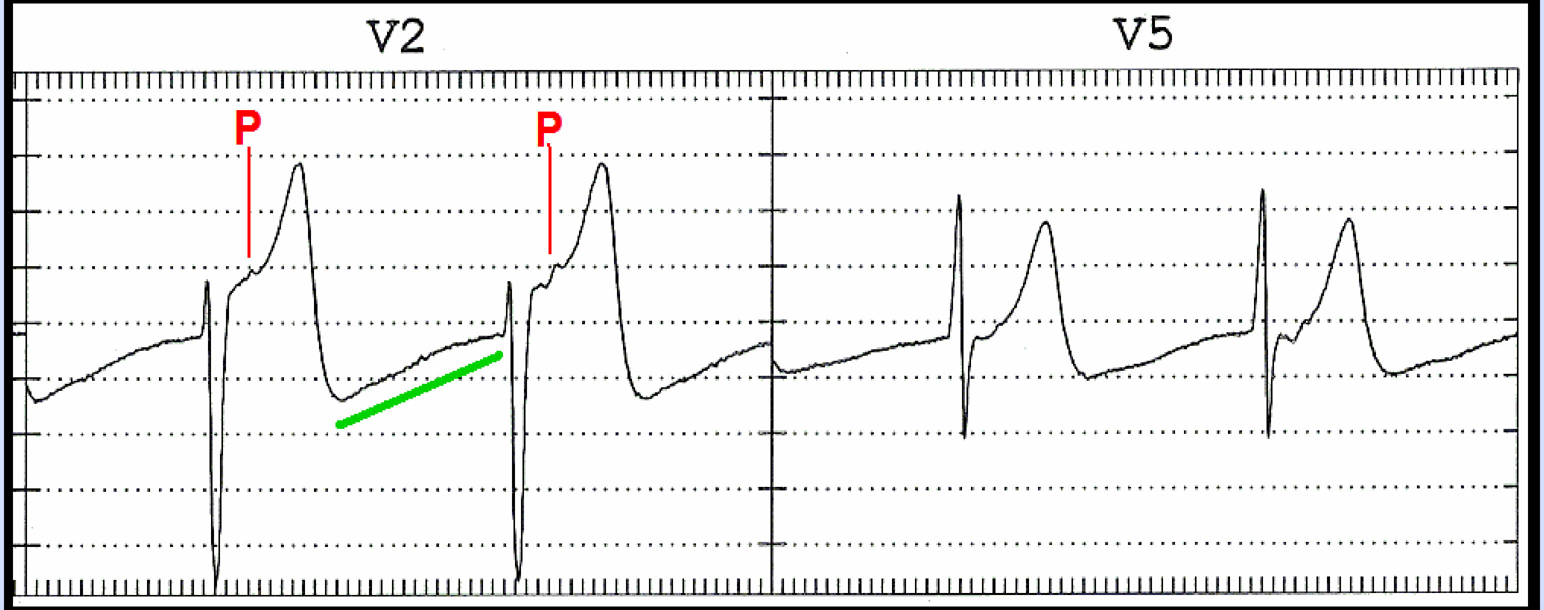
. . .the "flat line" between ECG complexes, when there is no detectable electrical activity . . .

The Isoelectric Line -

it's not always isoelectric !

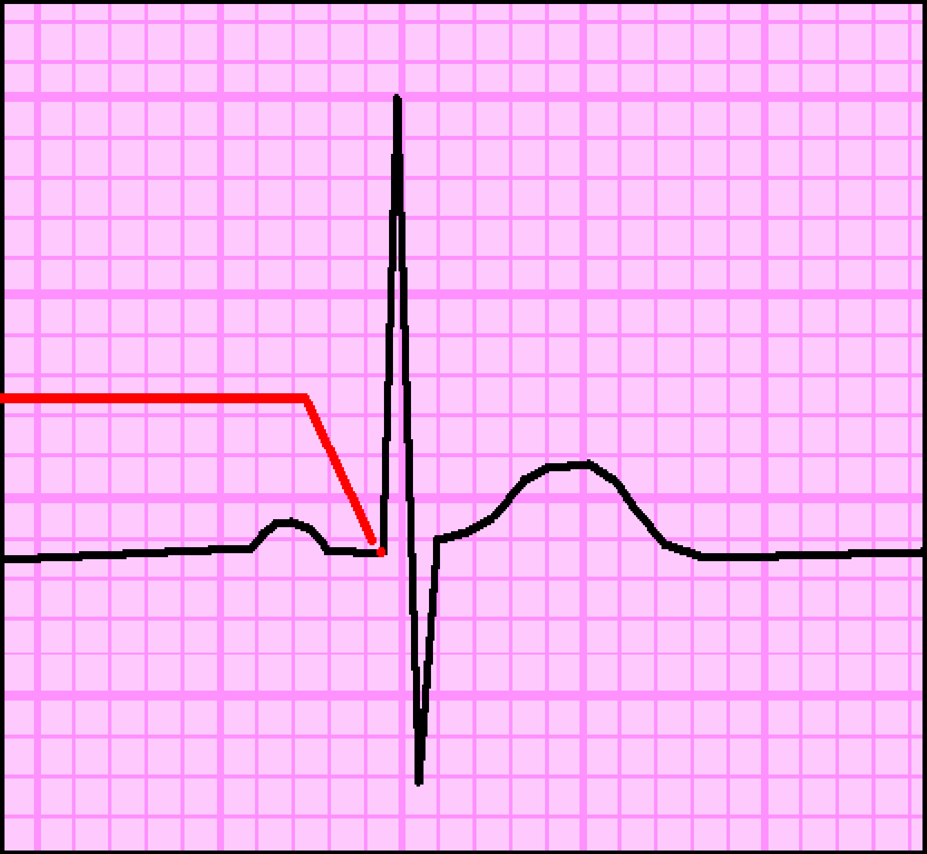
THE ISOELECTRIC LINE

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

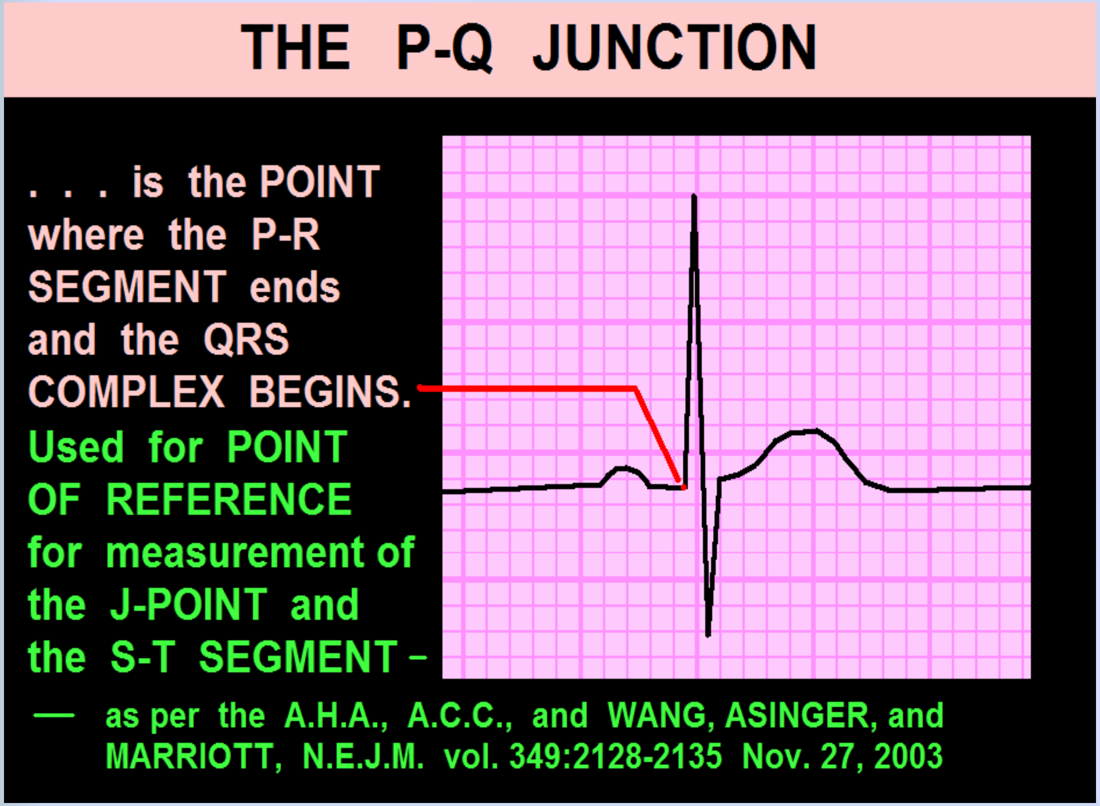
. . . is the POINT where the P-R SEGMENT ends and the QRS COMPLEX BEGINS.



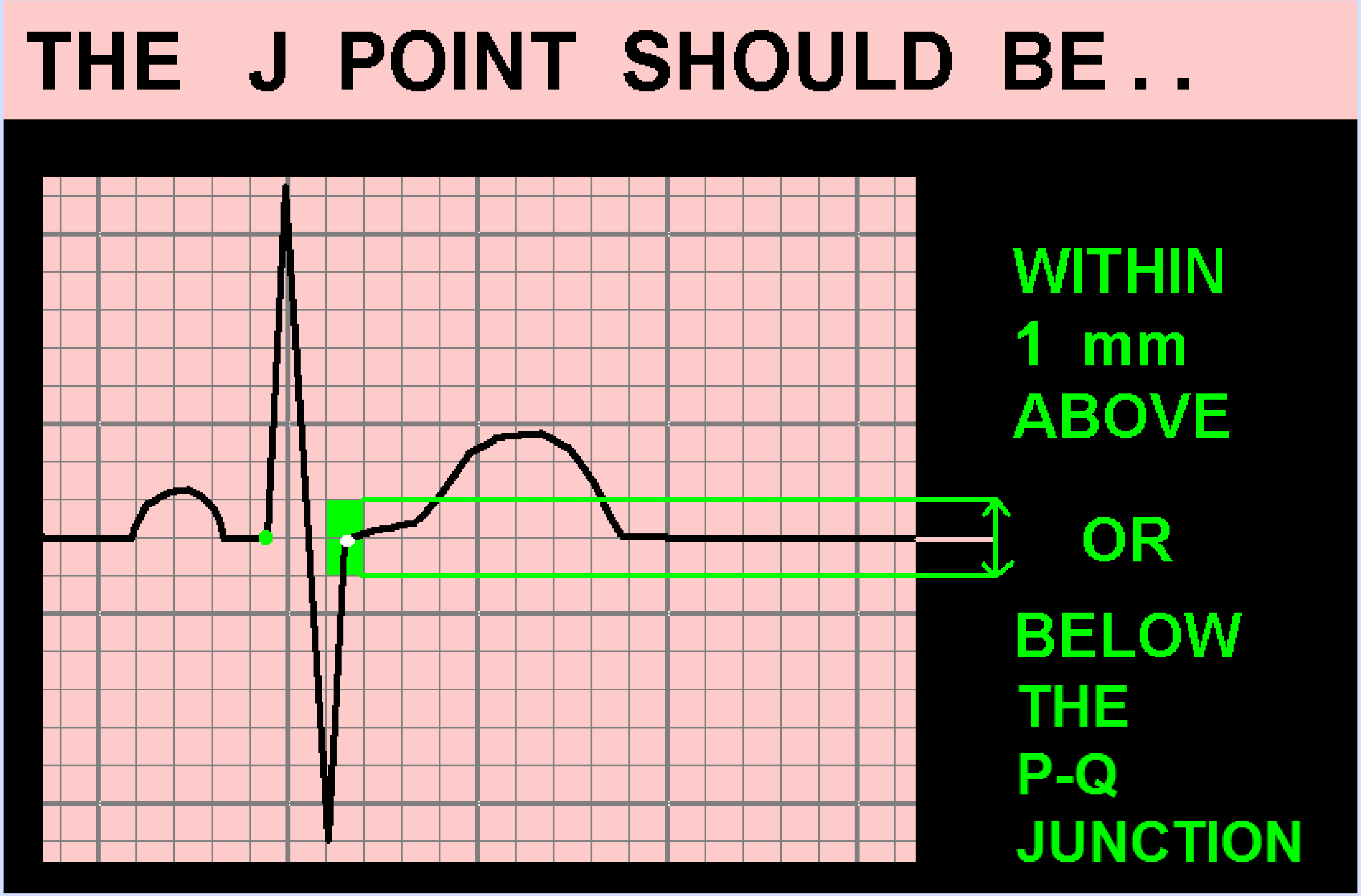
Used for POINT OF REFERENCE for measurement of the J-POINT and the S-T SEGMENT –

— 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

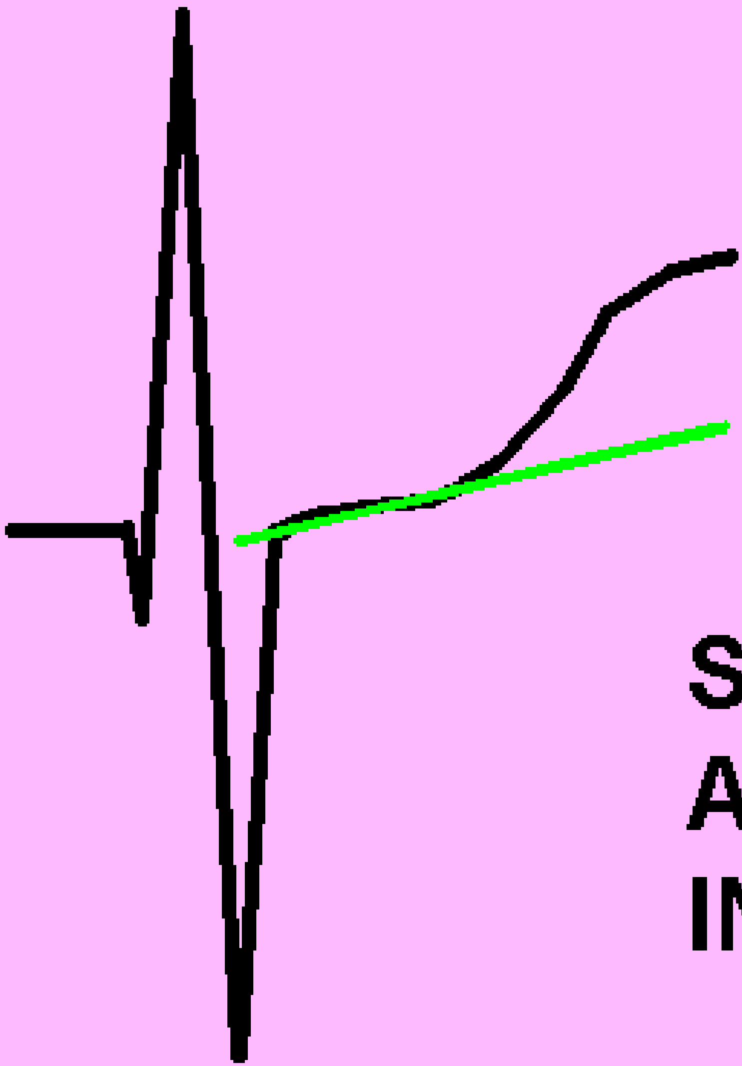
Use the P-Q junction as a reference point for measuring the J Point and ST-Segment when “iso-electric line is not iso-electric !



Defining NORMAL:

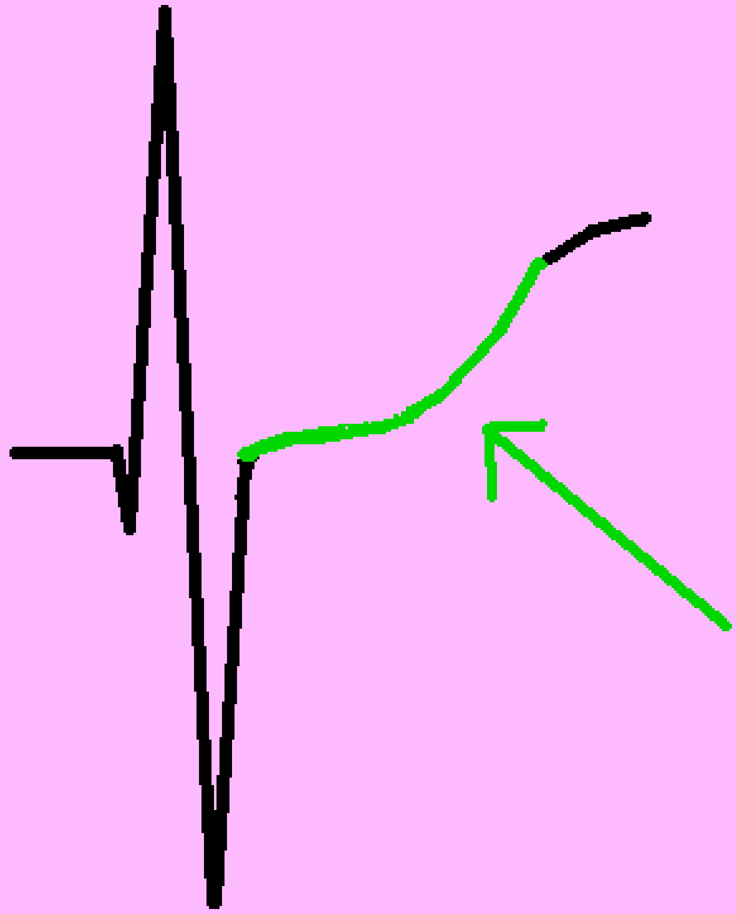


THE S-T SEGMENT



SHOULD HAVE
A "SLIGHT POSITIVE"
INCLINATION

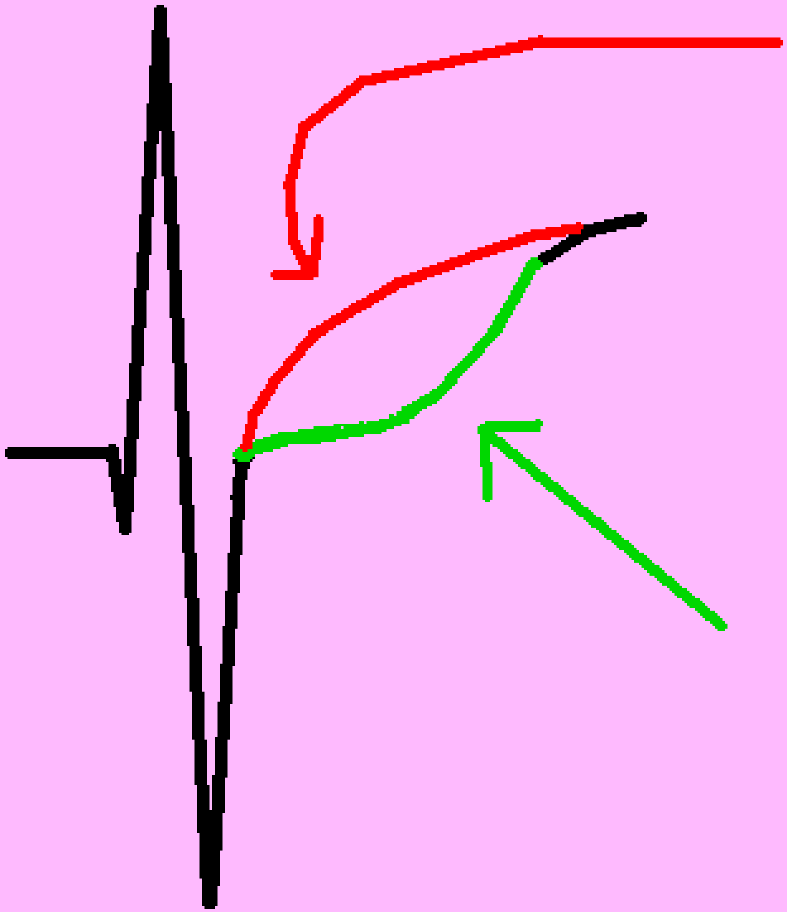
THE S-T SEGMENT



SHOULD BE
"CONCAVE" IN
SHAPE . . .

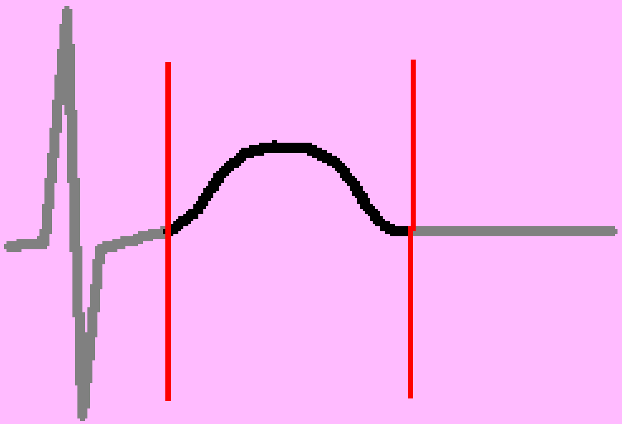
THE S-T SEGMENT

AS OPPOSED TO "CONVEX" IN SHAPE



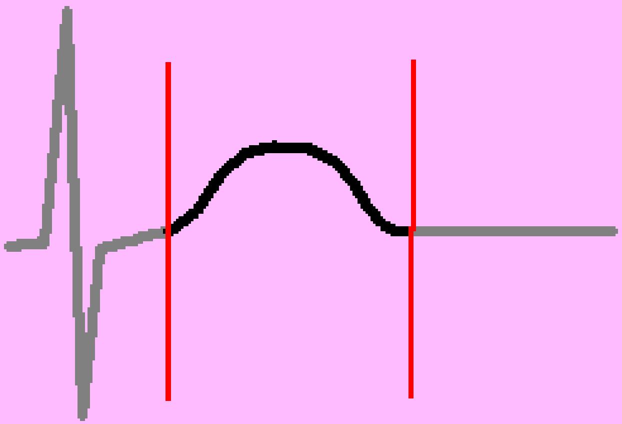
SHOULD BE "CONCAVE" IN SHAPE . . .

THE T WAVE



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

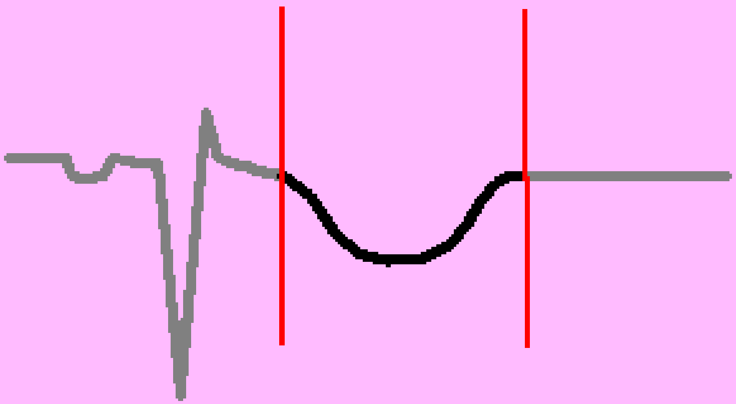
THE T WAVE



- 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

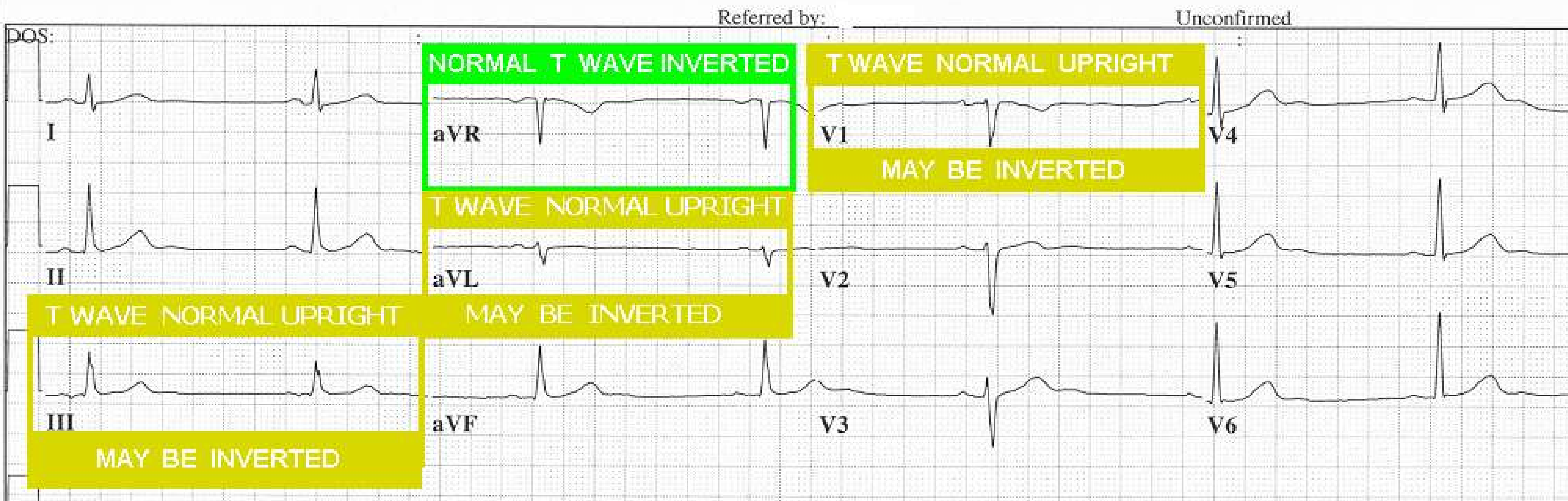
EVERYTHING

IS

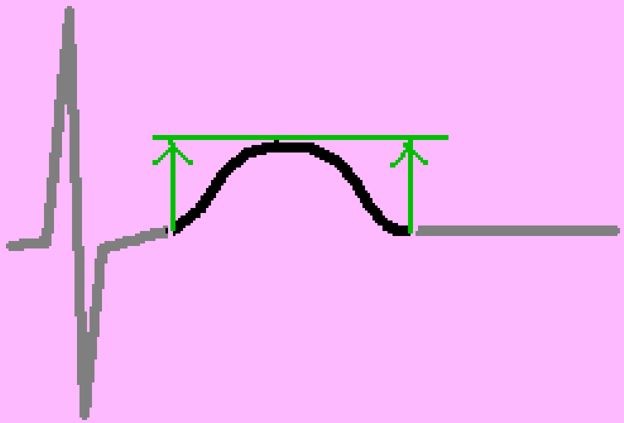
"UPSIDE-DOWN"

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 ! !***

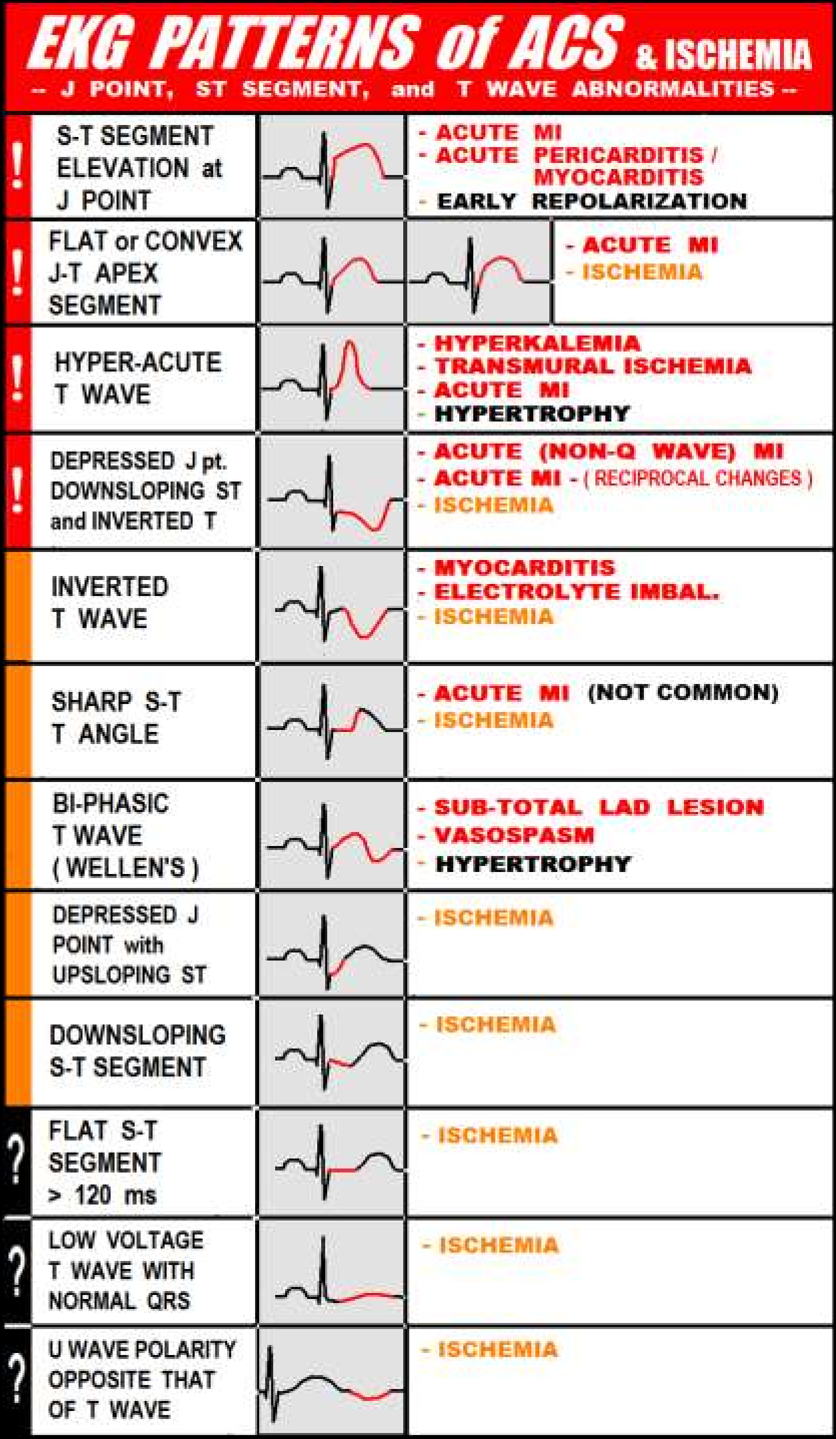
ECG Indicators of ACS in Patients with *Normal Width* QRS Complexes (QRS duration < 120 ms)

Multiple patterns of **ABNORMAL:**

- J Point
- ST-Segment
- T Wave

configurations may indicate ACS.

Remember, "IF IT'S NOT NORMAL, it's *ABNORMAL !*"



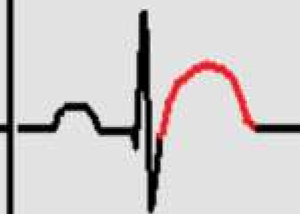
EKG PATTERNS of ACS & ISCHEMIA
-- J POINT, ST SEGMENT, and T WAVE ABNORMALITIES --

!	S-T SEGMENT ELEVATION at J POINT		- ACUTE MI - ACUTE PERICARDITIS / MYOCARDITIS - EARLY REPOLARIZATION
!	FLAT or CONVEX J-T APEX SEGMENT		- ACUTE MI - ISCHEMIA
!	HYPER-ACUTE T WAVE		- HYPERKALEMIA - TRANSMURAL ISCHEMIA - ACUTE MI - HYPERTROPHY
!	DEPRESSED J pt. DOWNSLOPING ST and INVERTED T		- ACUTE (NON-Q WAVE) MI - ACUTE MI - (RECIPROCAL CHANGES) - ISCHEMIA
	INVERTED T WAVE		- MYOCARDITIS - ELECTROLYTE IMBAL. - ISCHEMIA
	SHARP S-T T ANGLE		- ACUTE MI (NOT COMMON) - ISCHEMIA
	BI-PHASIC T WAVE (WELLEN'S)		- SUB-TOTAL LAD LESION - VASOSPASM - HYPERTROPHY
	DEPRESSED J POINT with UPSLOPING ST		- ISCHEMIA
	DOWNSLOPING S-T SEGMENT		- ISCHEMIA
?	FLAT S-T SEGMENT > 120 ms		- ISCHEMIA
?	LOW VOLTAGE T WAVE WITH NORMAL QRS		- ISCHEMIA
?	U WAVE POLARITY OPPOSITE THAT OF T WAVE		- ISCHEMIA

BOOK PAGE: 83

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**

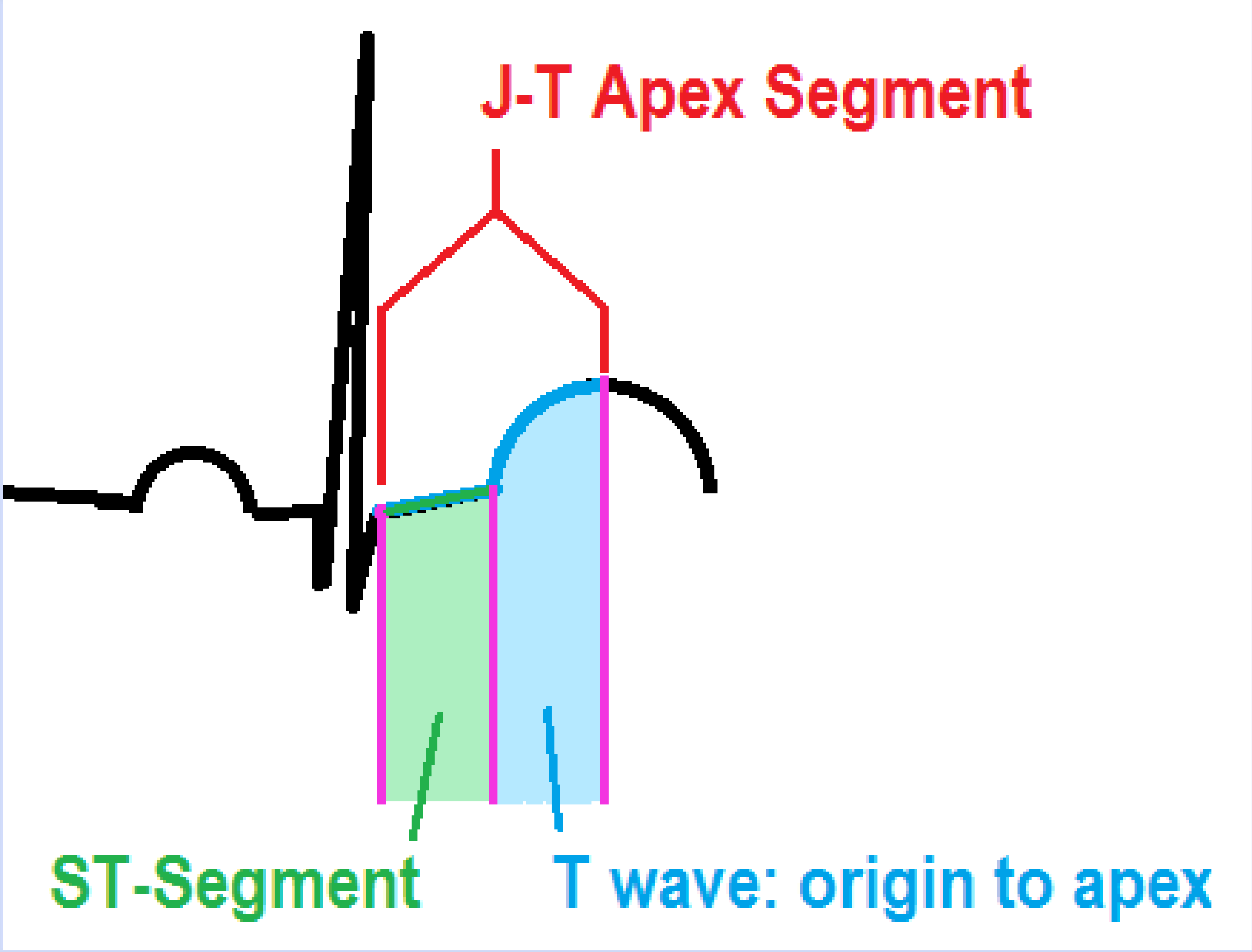
PATTERNS of ACS & ISCHEMIA

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

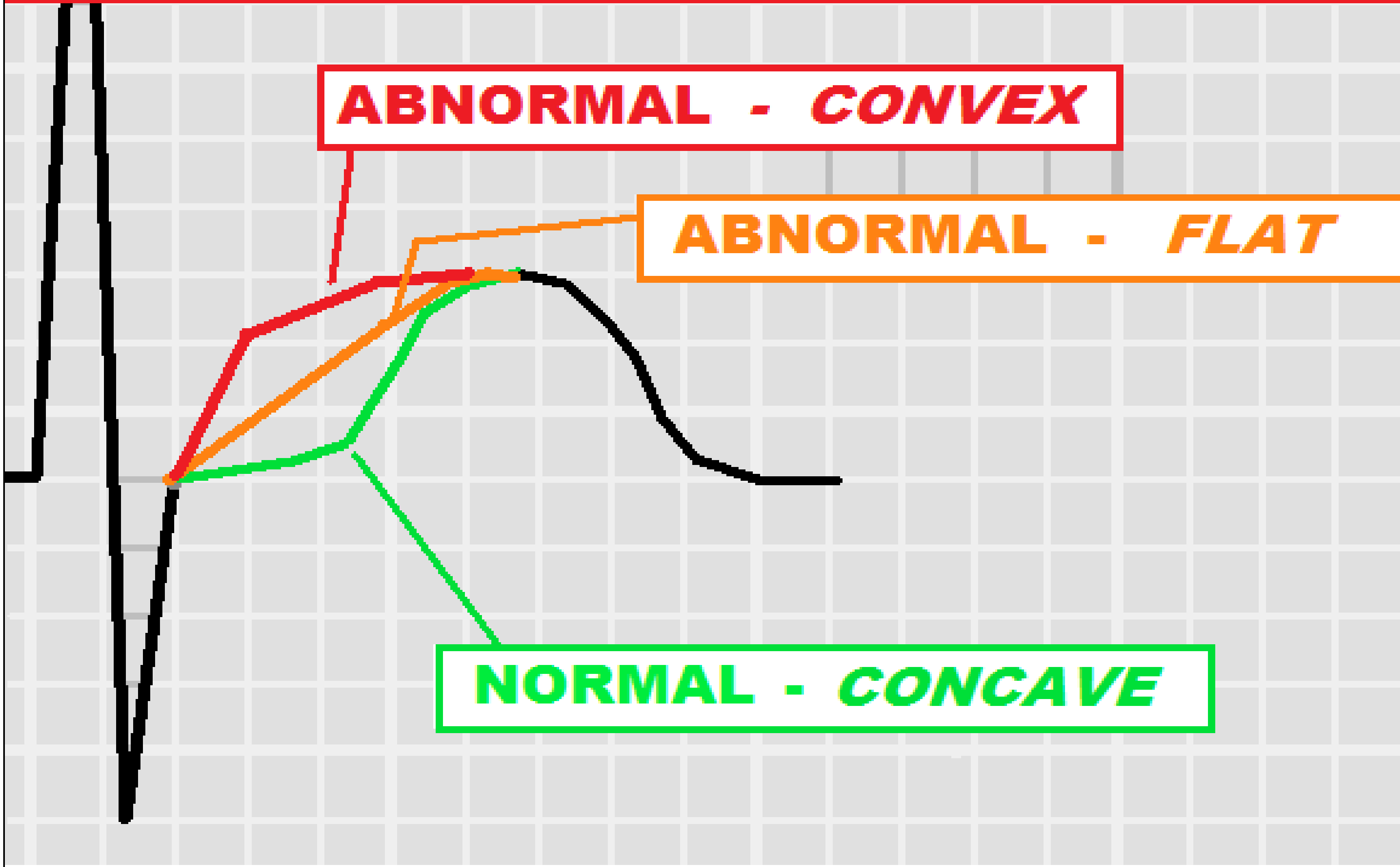
!	FLAT or CONVEX J-T APEX SEGMENT			***ACUTE MI*** *EARLY PHASE*
!	HYPER-ACUTE T WAVE		***ACUTE MI*** *EARLY PHASE*	
!	S-T SEGMENT ELEVATION at J POINT		***ACUTE MI***	
!	DEPRESSED J pt. DOWNSLOPING ST and INVERTED T		- **ACUTE (NON-Q WAVE) MI** - **ACUTE MI** - (RECIPROCAL CHANGES) - **ISCHEMIA**	

ECG 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

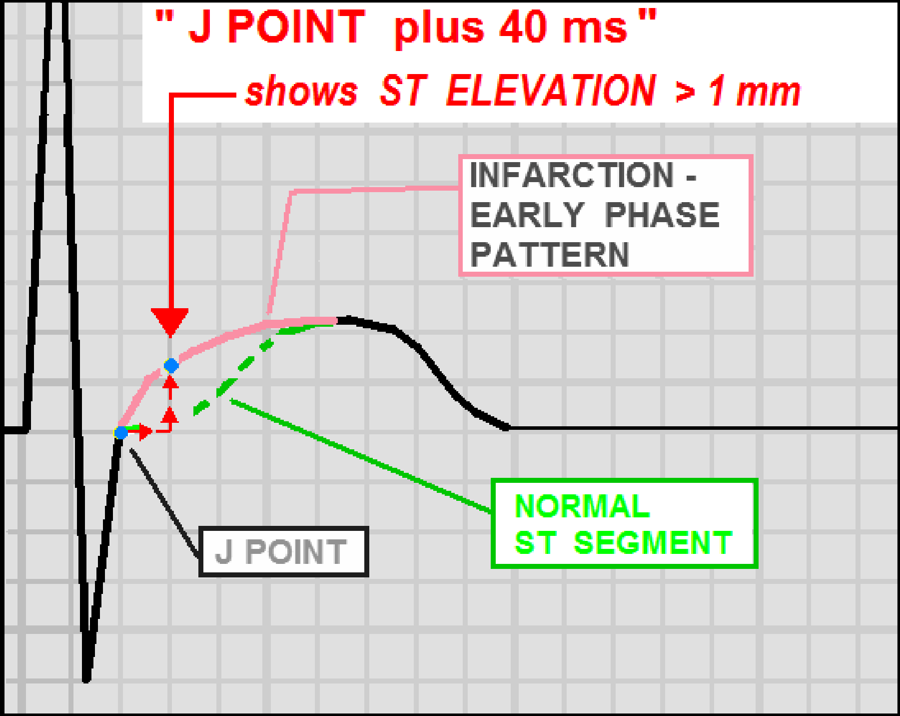


PATTERNS of EARLY INFARCTION

-- FLAT and CONVEX J-T APEX SEGMENTS

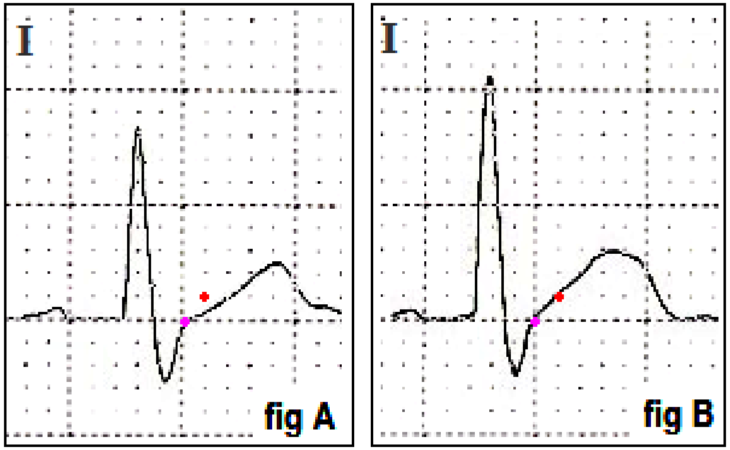
WHEN EVALUATING for ST SEGMENT ELEVATION . . . .

From:
AMERICAN HEART ASSOCIATION
ACLS 2005 REVISIONS



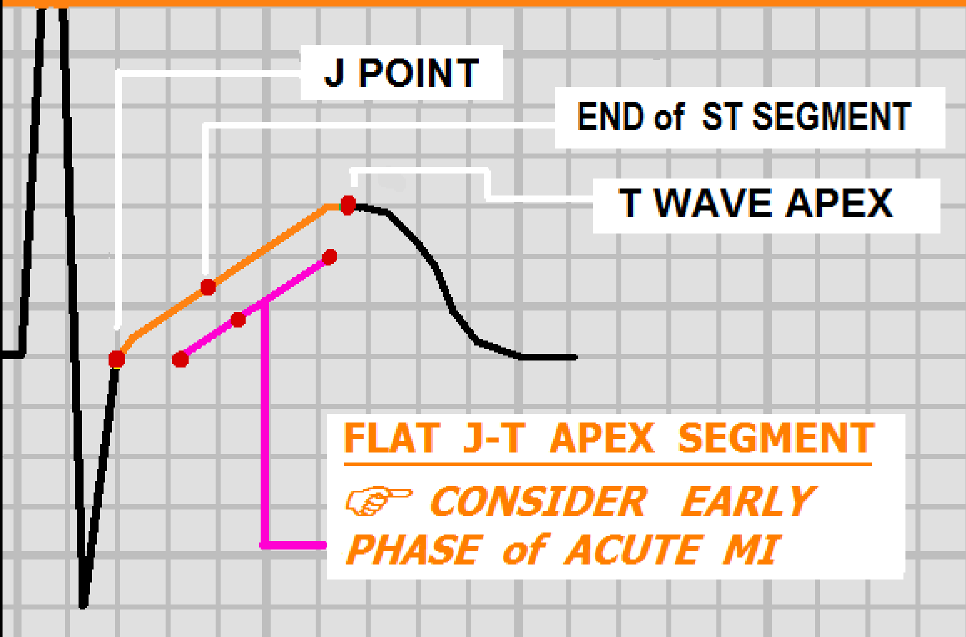
During NORMAL STATES of PERFUSION, the J POINT is ISOELECTRIC and the ST SEGMENT has a CONCAVE appearance. When measured 40 ms beyond the J POINT (noted by the RED DOT), the ST SEGMENT elevation is less than 1mm.

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).

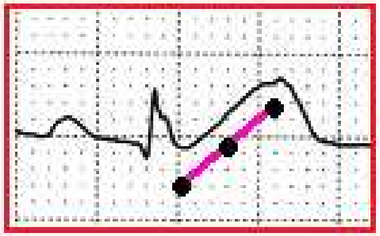


During a 20 second BALLOON OCCLUSION of the patient's LAD during routine PTCA, the ST segment assumes a CONVEX shape. When measured 40 ms beyond the J POINT, the ST segment is elevated > 1 mm. This phenomenon is seen routinely in the cath lab prior to the occurance of ST ELEVATION at the J POINT during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

ABNORMAL J-T APEX SEGMENT



LEAD II

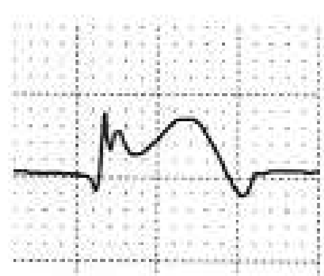


1839 hrs

41 y/o FEMALE

In ER C/O CHEST PAIN x 30 minutes.

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



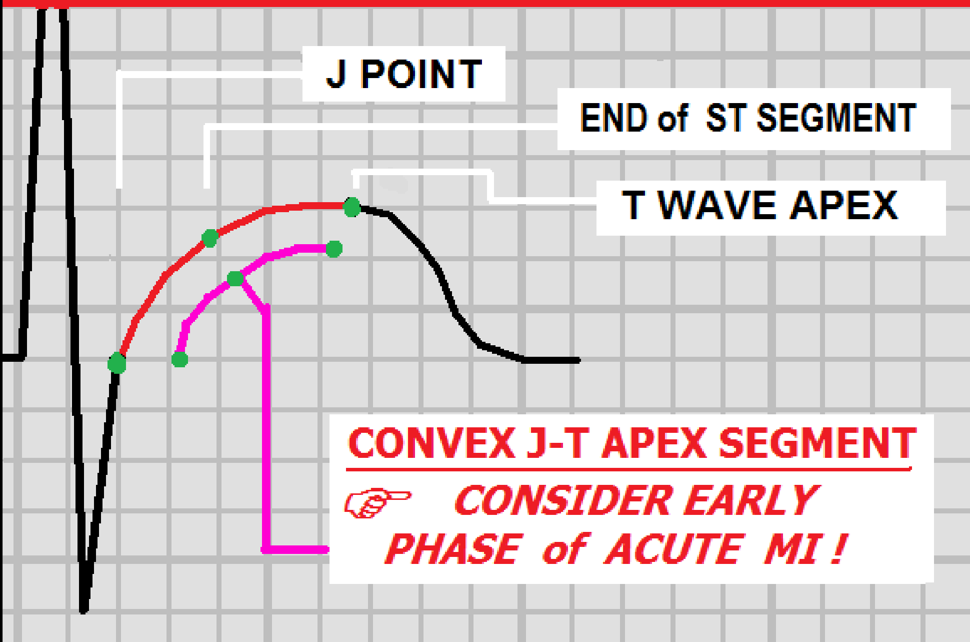
1850 hrs

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

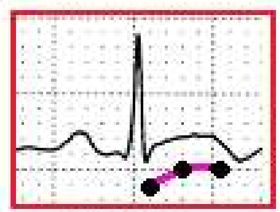
ABNORMAL J-T APEX SEGMENT



LEAD I

53 y/o MALE

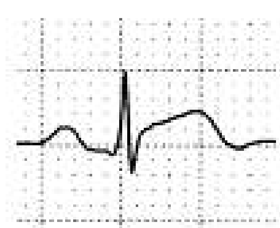
1 yr. PRIOR TO MI
NORMAL EKG
CONCAVE J - T APEX SEGMENT



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.

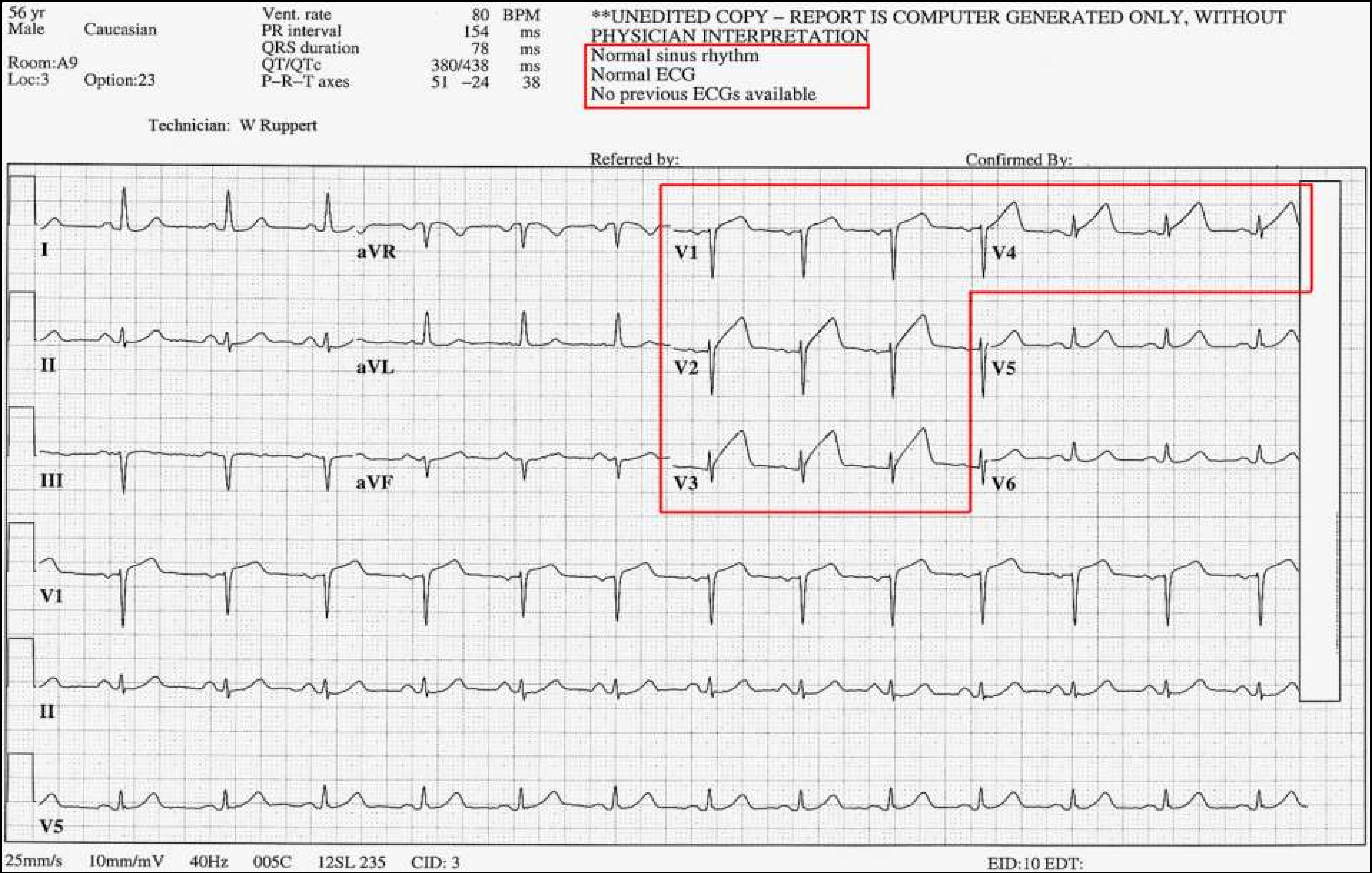
RISK FACTOR PROFILE:

- 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.

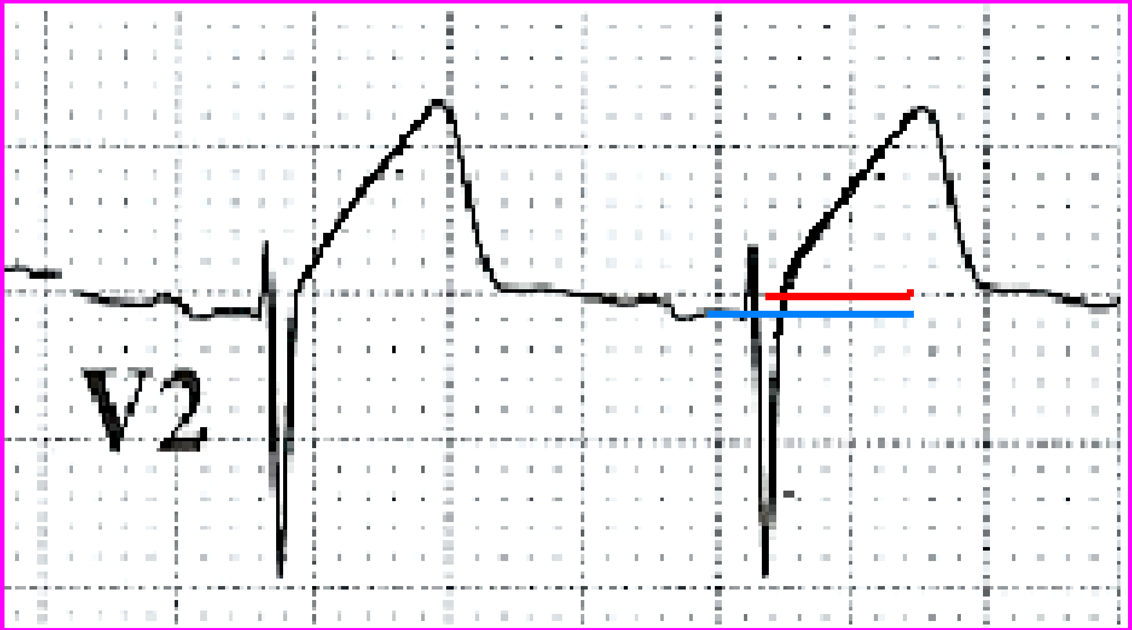
VITAL SIGNS: BP 142/94, P 80, R 20, SAO2 98%

LABS: JUST OBTAINED, RESULTS NOT AVAILABLE YET.



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

measurement of S-T elevation



S-T elevation at J point = 0.5 mm

ACUTE MI = S-T elev. > 1.0 mm

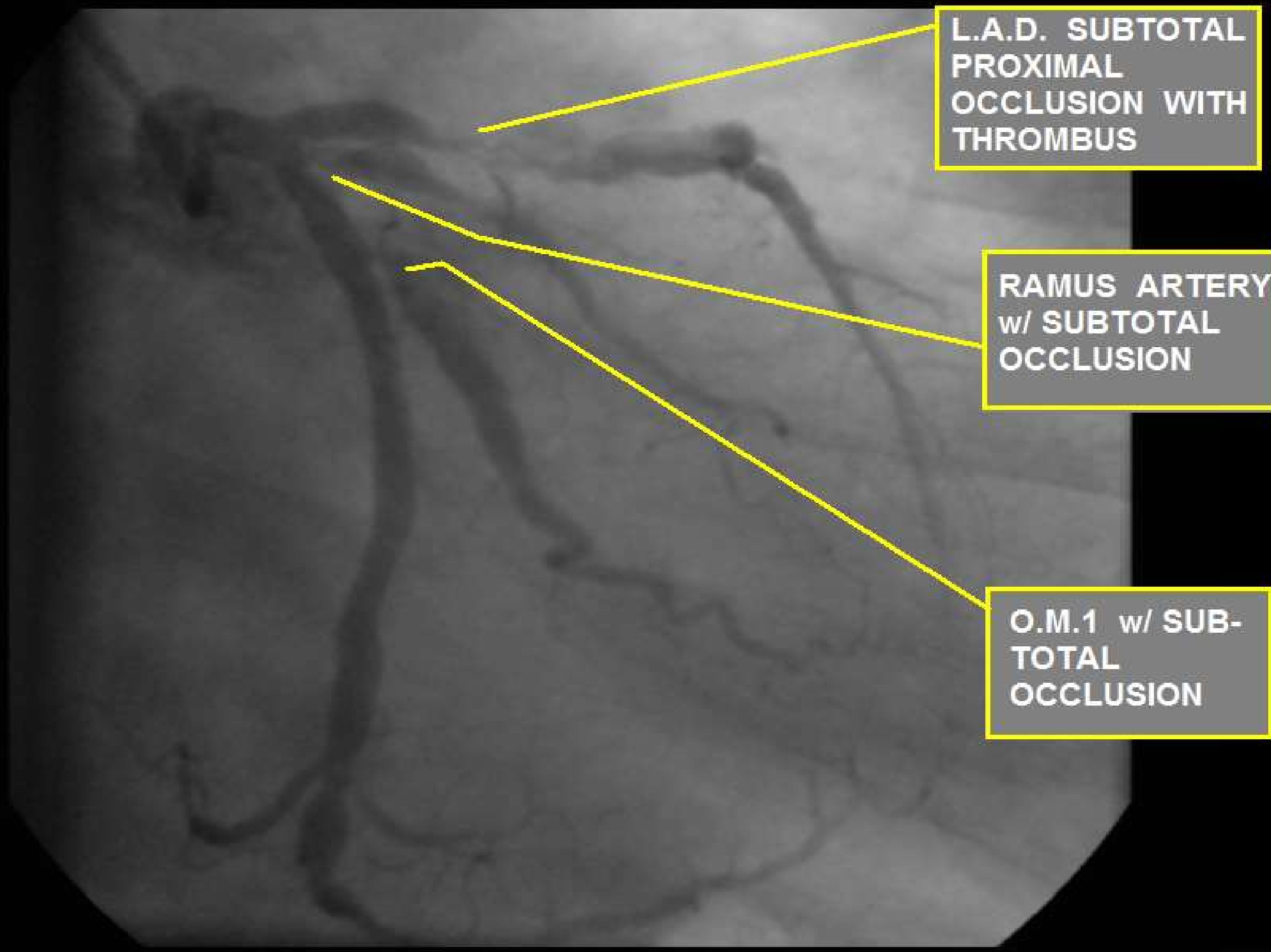
measurement of S-T elevation
by "J point + .04" method

S-T elevation at J point = 0.5 mm

S-T elevation at J + .04 = 2.0 mm

ACUTE MI = S-T elev. > 1.0 mm

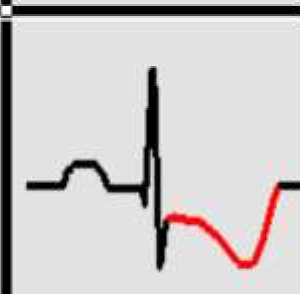
CASE STUDY: 56 y/o male with INTERMITTENT "CHEST HEAVINESS" . . . .



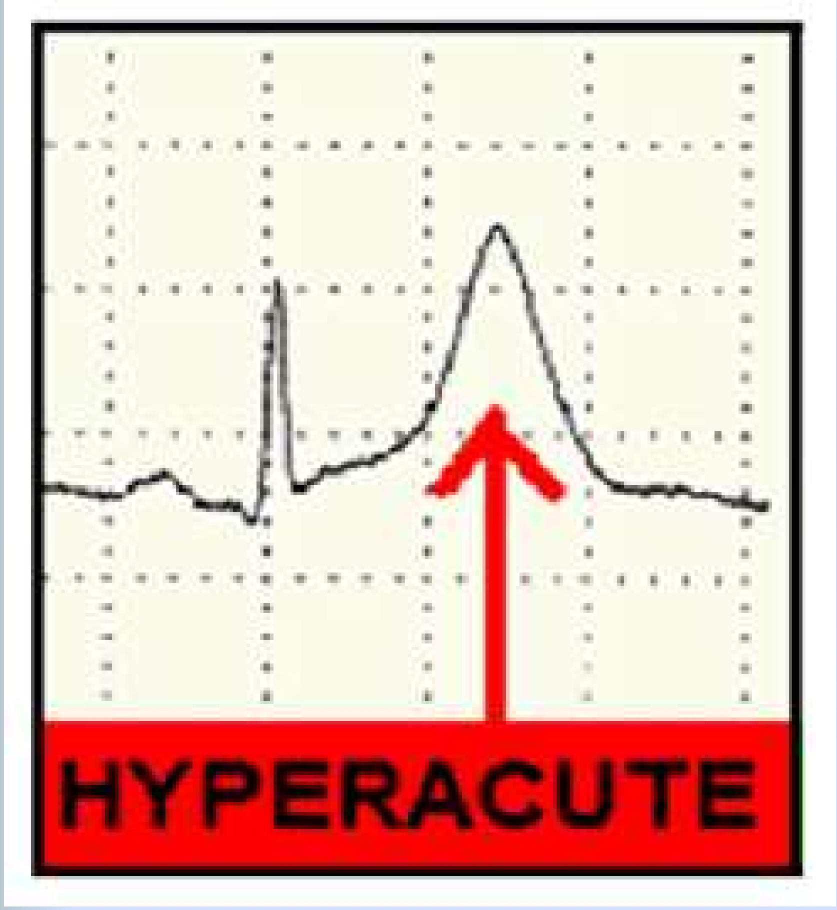
TREATMENT PLAN : EMERGENCY CORONARY ARTERY BYPASS SURGERY (4 VESSEL)

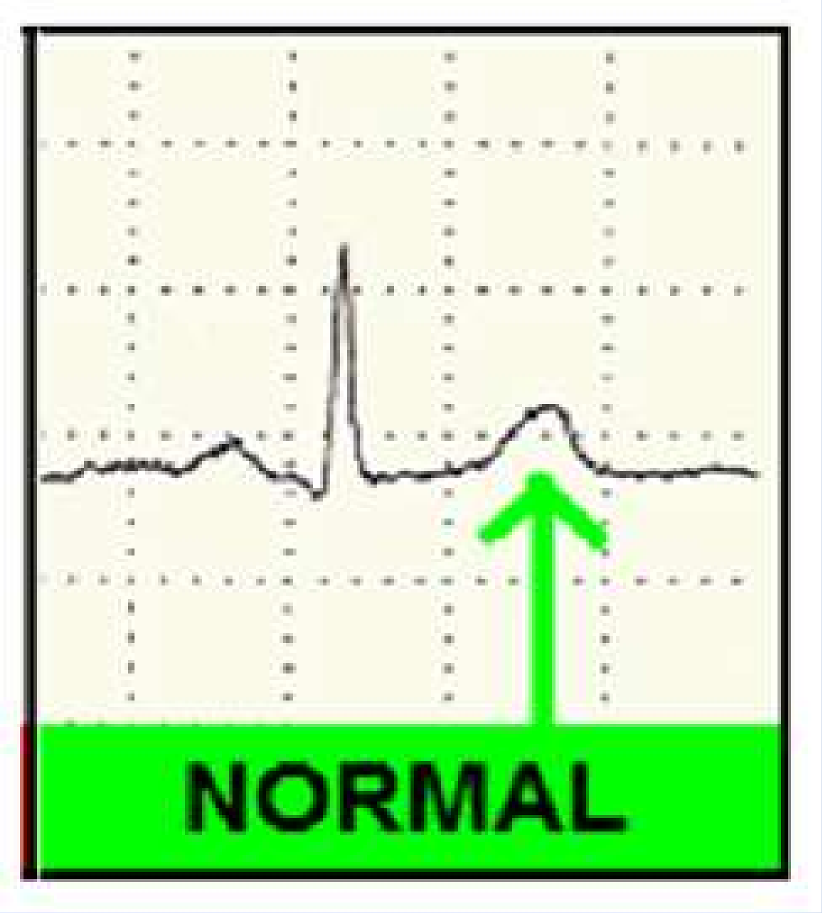
PATTERNS of ACS & ISCHEMIA

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

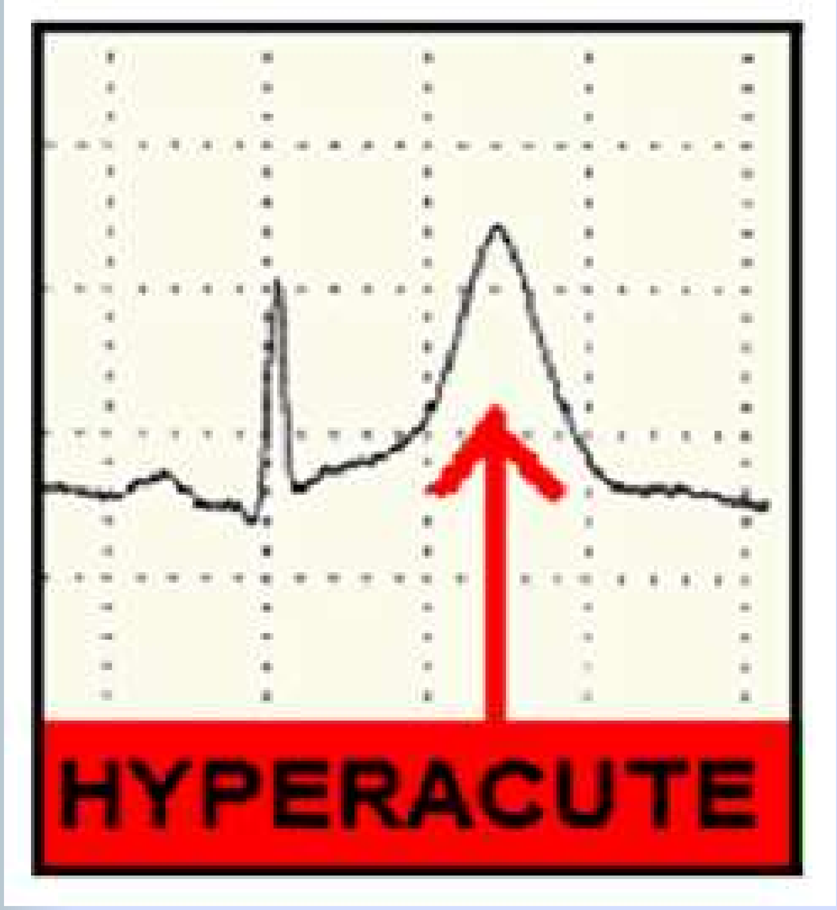
!	FLAT or CONVEX J-T APEX SEGMENT			**ACUTE MI** *EARLY PHASE*
!	HYPER-ACUTE T WAVE			**ACUTE MI** *EARLY PHASE*
!	S-T SEGMENT ELEVATION at J POINT			**ACUTE MI**
!	DEPRESSED J pt. DOWNSLOPING ST and INVERTED T			- **ACUTE (NON-Q WAVE) MI** - **ACUTE MI** - (RECIPROCAL CHANGES) - **ISCHEMIA**

T waves should not be HYPERACUTE



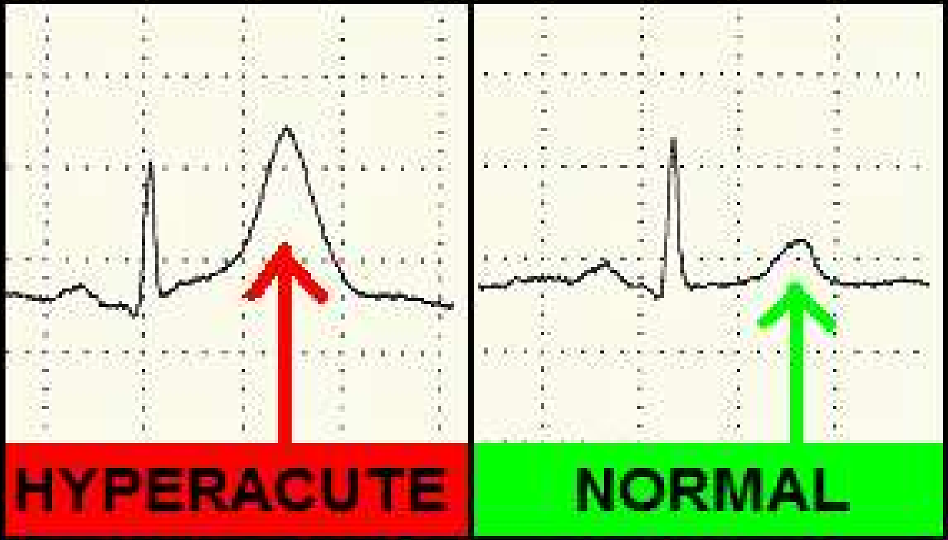


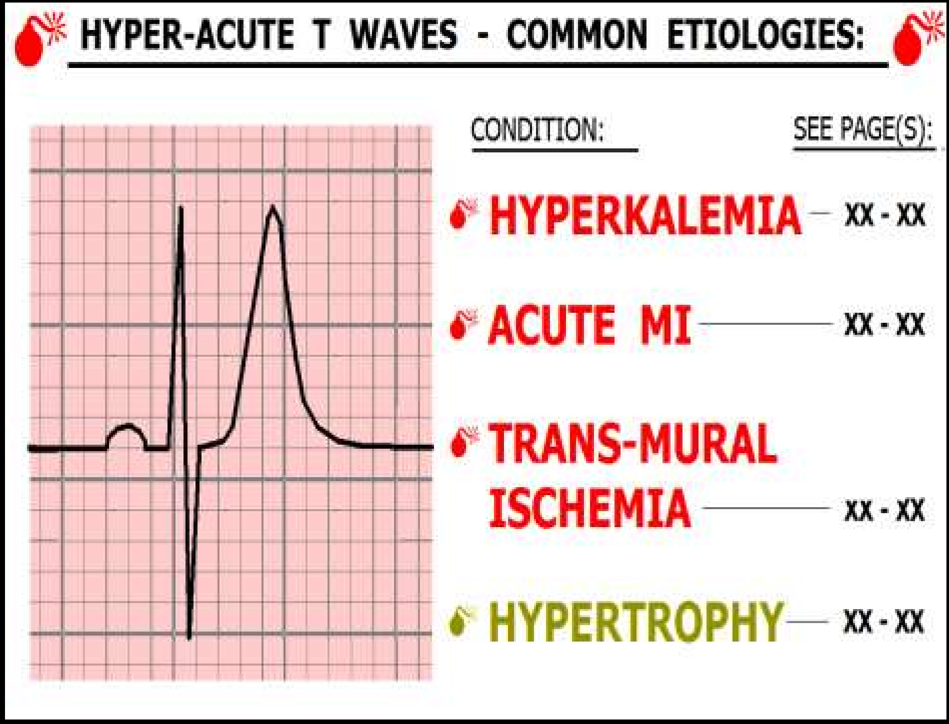
HYPERACUTE T Waves may indicate:

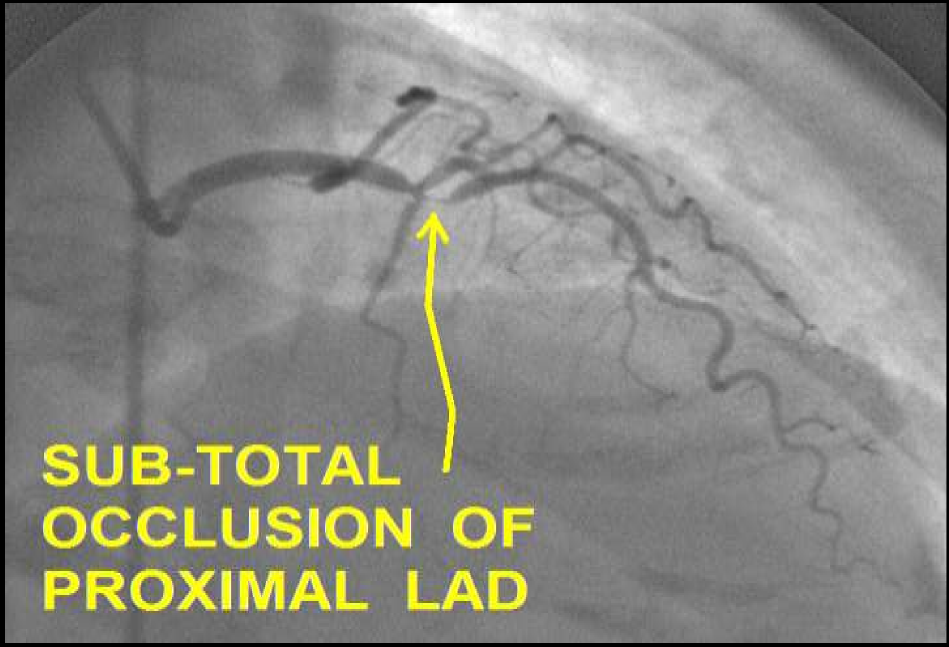


- **Early phase Acute MI**
- **Transmural ischemia** (usually seen in one region of the ECG)
- **Hyperkalemia** (seen globally across ECG)
- **Hypertrophy**

HYPERACUTE T WAVES







BOOK PAGE: 88

Helpful Clue: Hyper-Acute T Waves

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

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 1511

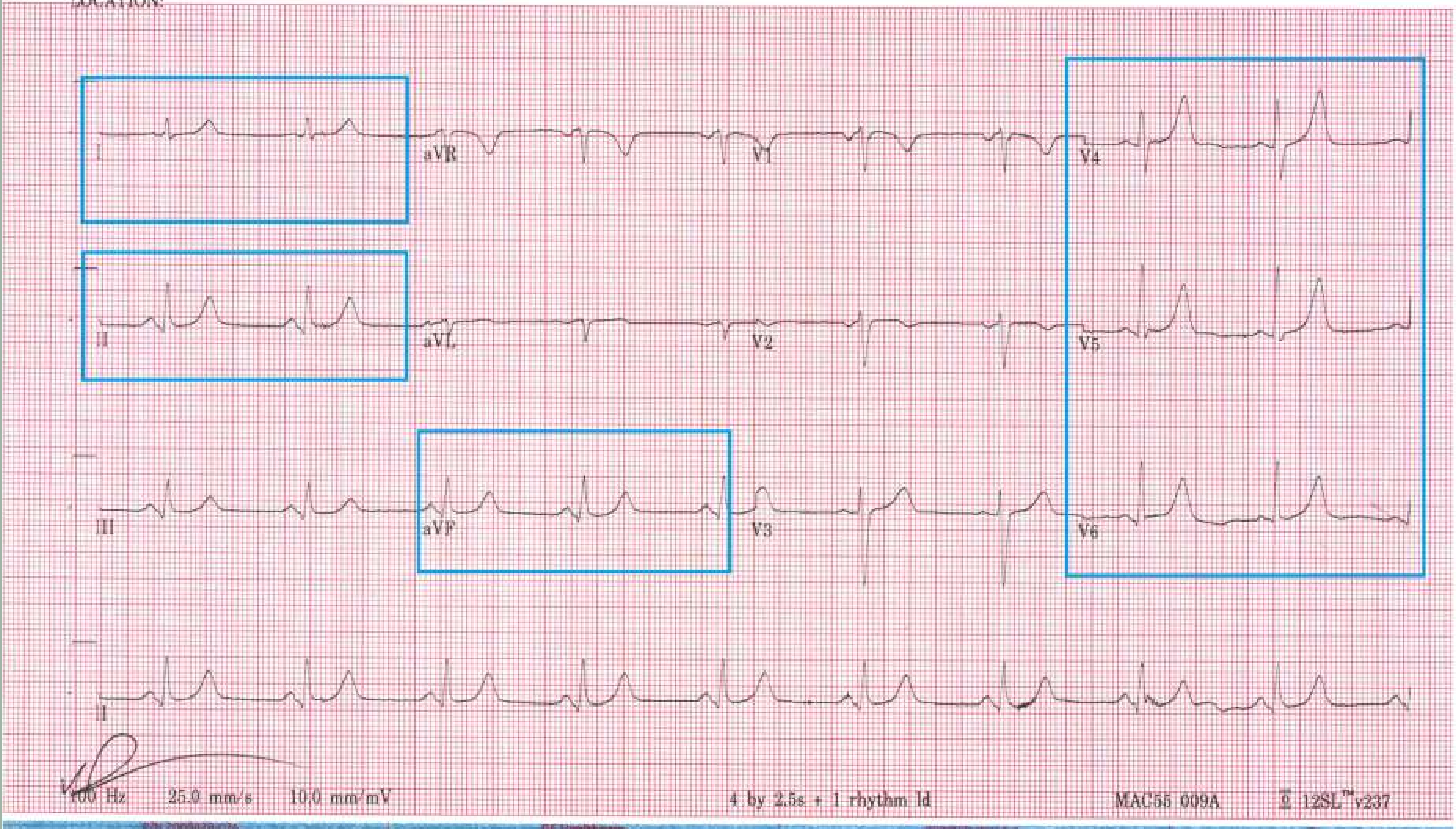
K+ = 6.7

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%

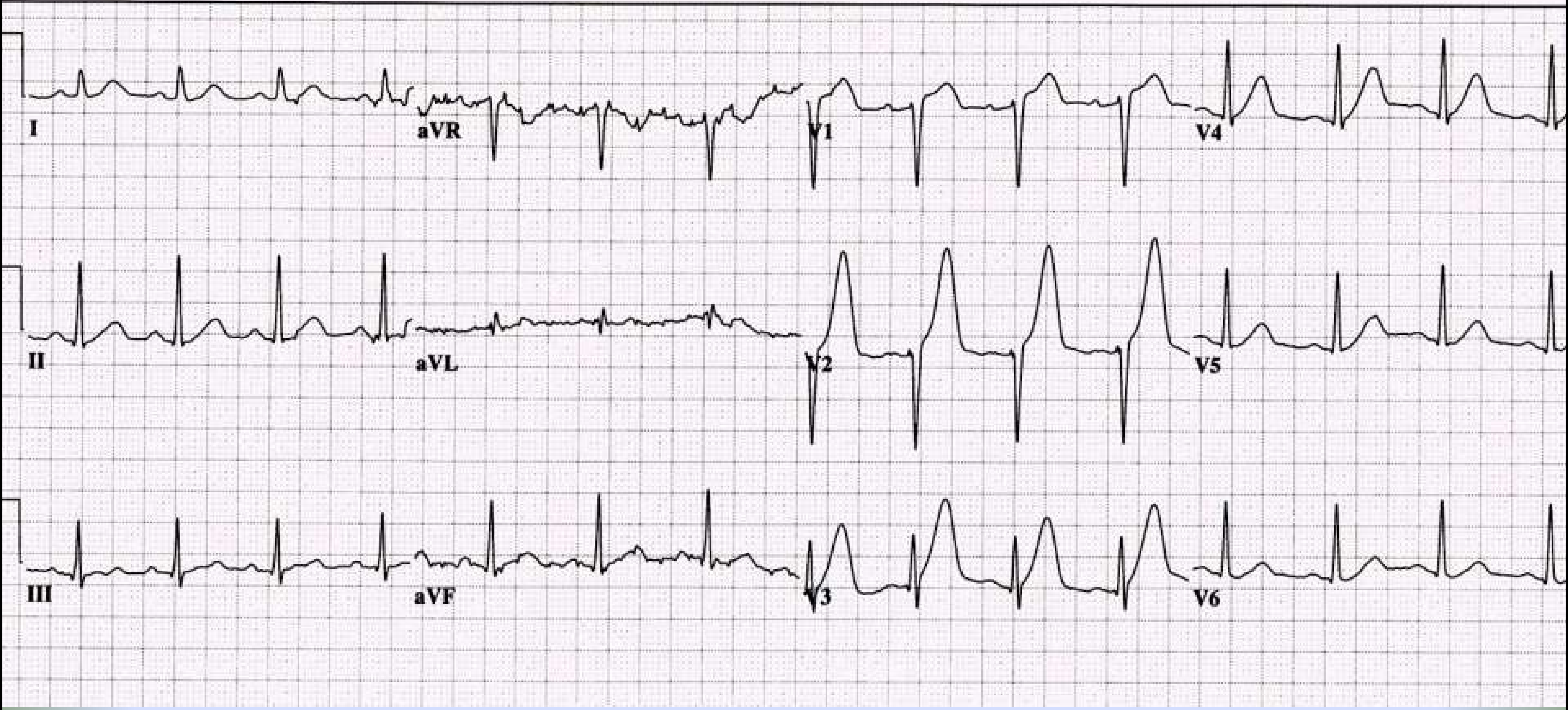
DIAGNOSTIC TESTING: 1st TROPONIN I - ultra: <0.07

30 yr
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

NOTE COMPUTER INTERPRETATION



30 yr
Male Black

Room: ER
Loc: Option:

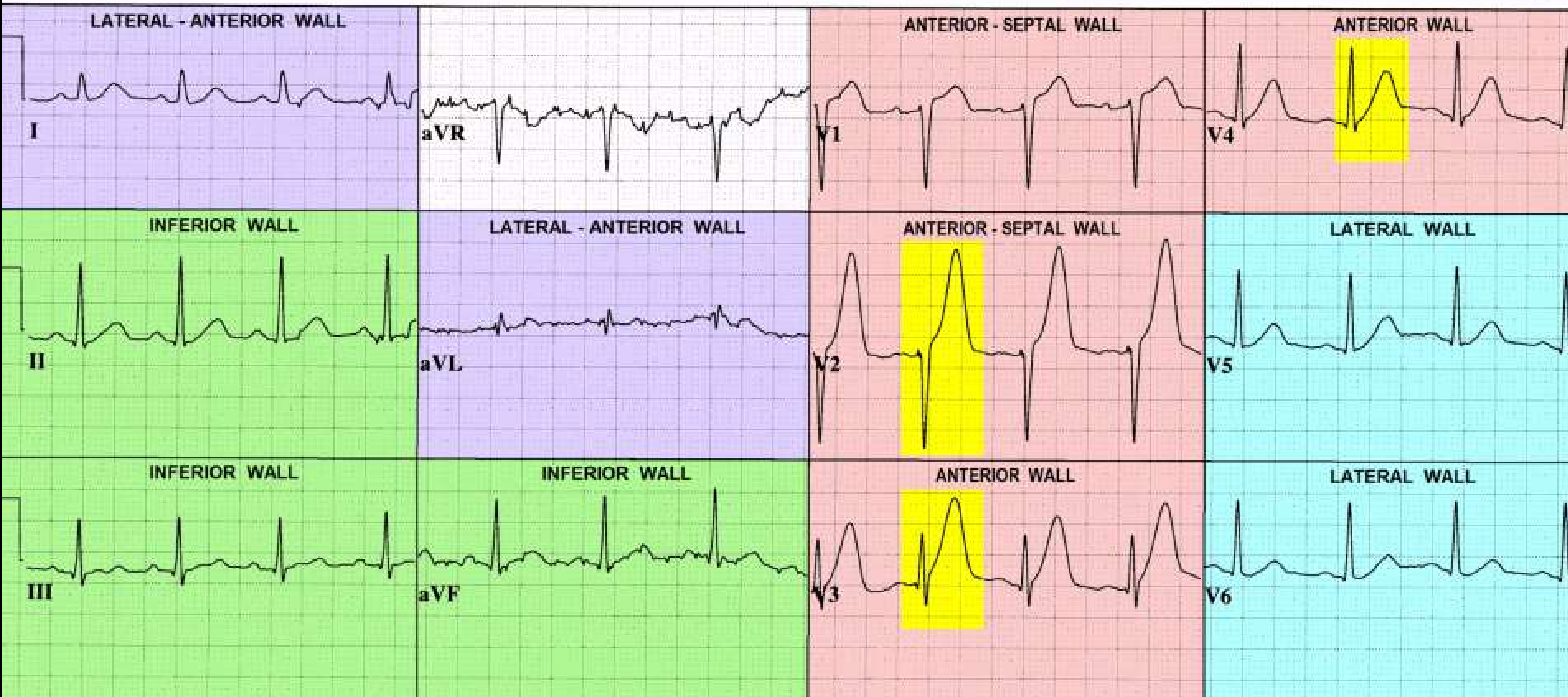
Vent. rate	88	BPM
PR interval	164	ms
QRS duration	90	ms
QT/QTc	370/447	ms
P–R–T axes	61 62	53

Normal sinus rhythm
Normal ECG
No previous ECGs available

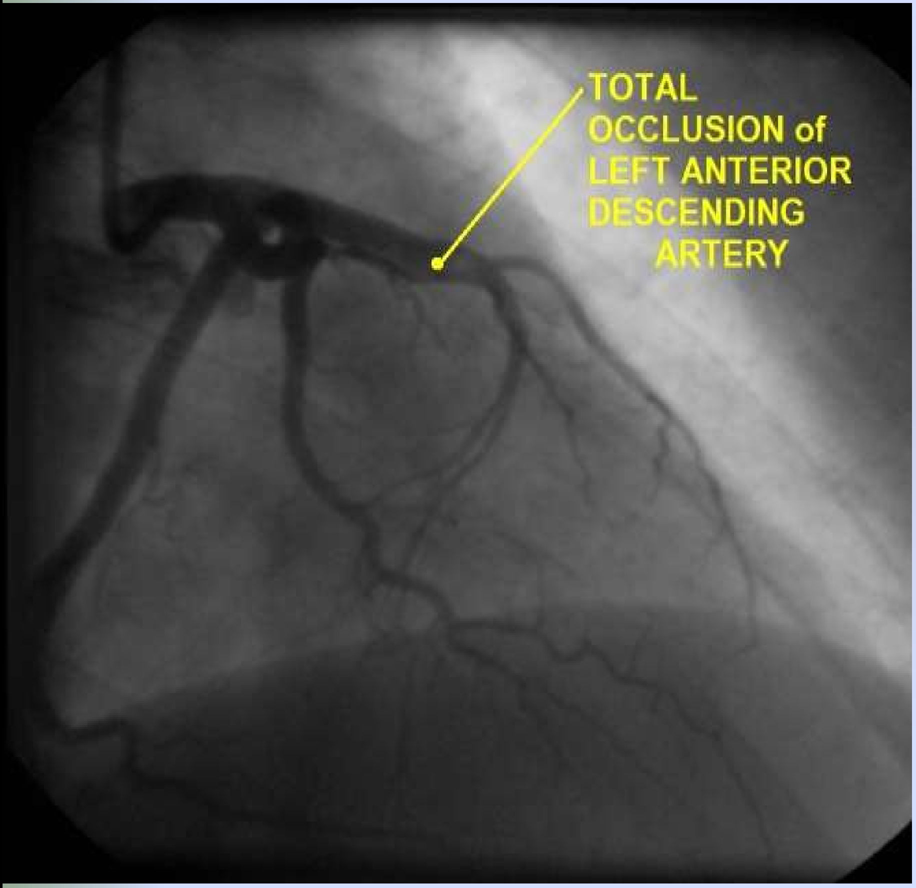
HIGHLIGHTED AREAS = HYPERACUTE T WAVES

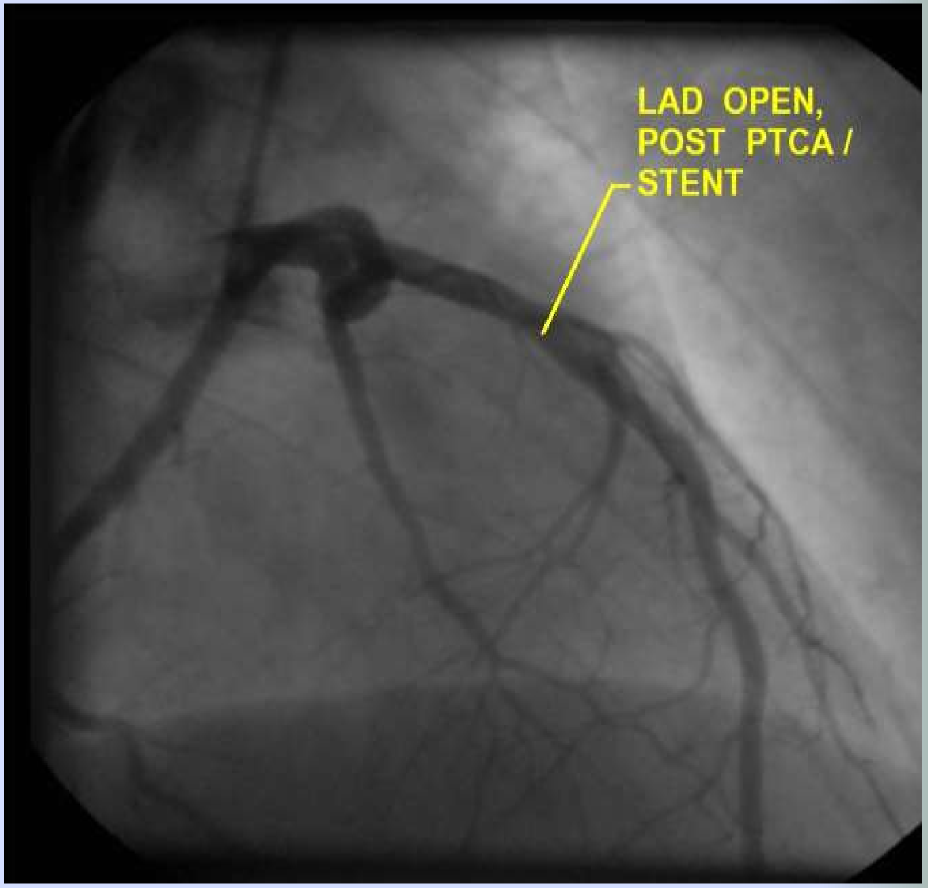
CORONARY ARTERIAL DISTRIBUTIONS:

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:

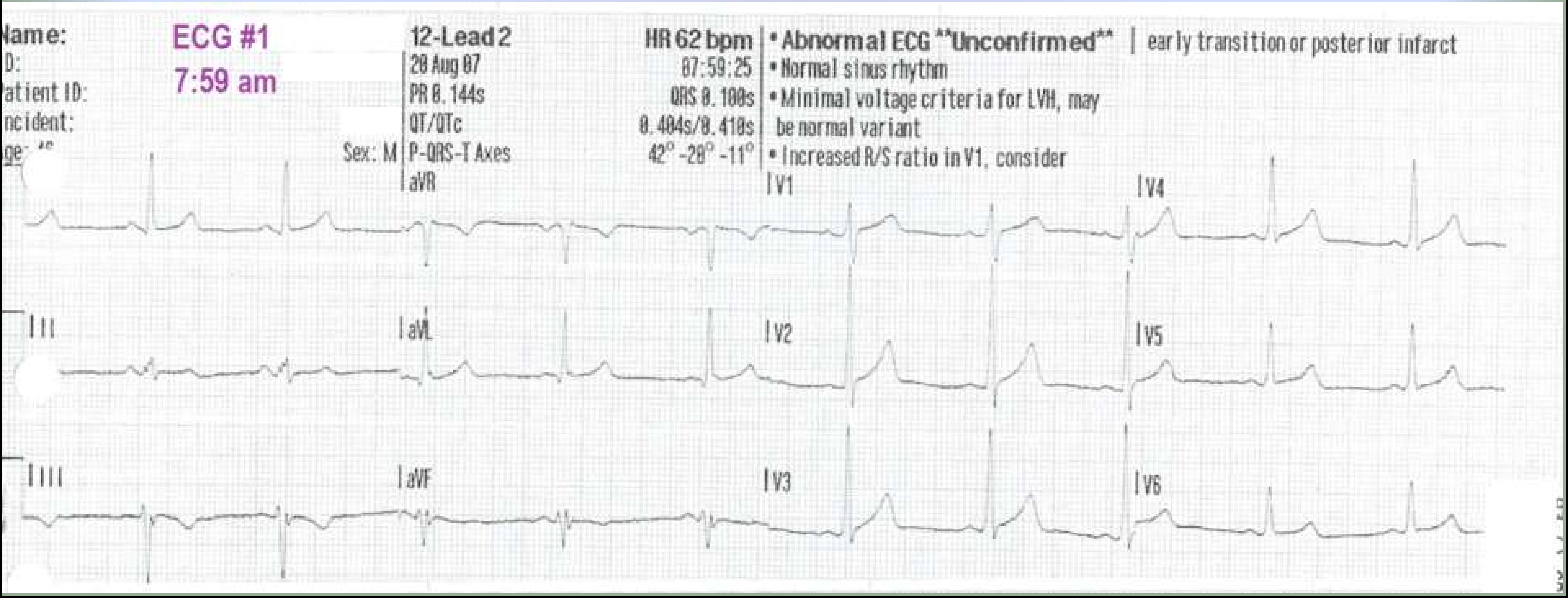
- Other than HEART RATE related variations (which affect intervals), ***J Points, ST-Segments and T Waves SHOULD NOT CHANGE.***

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.**

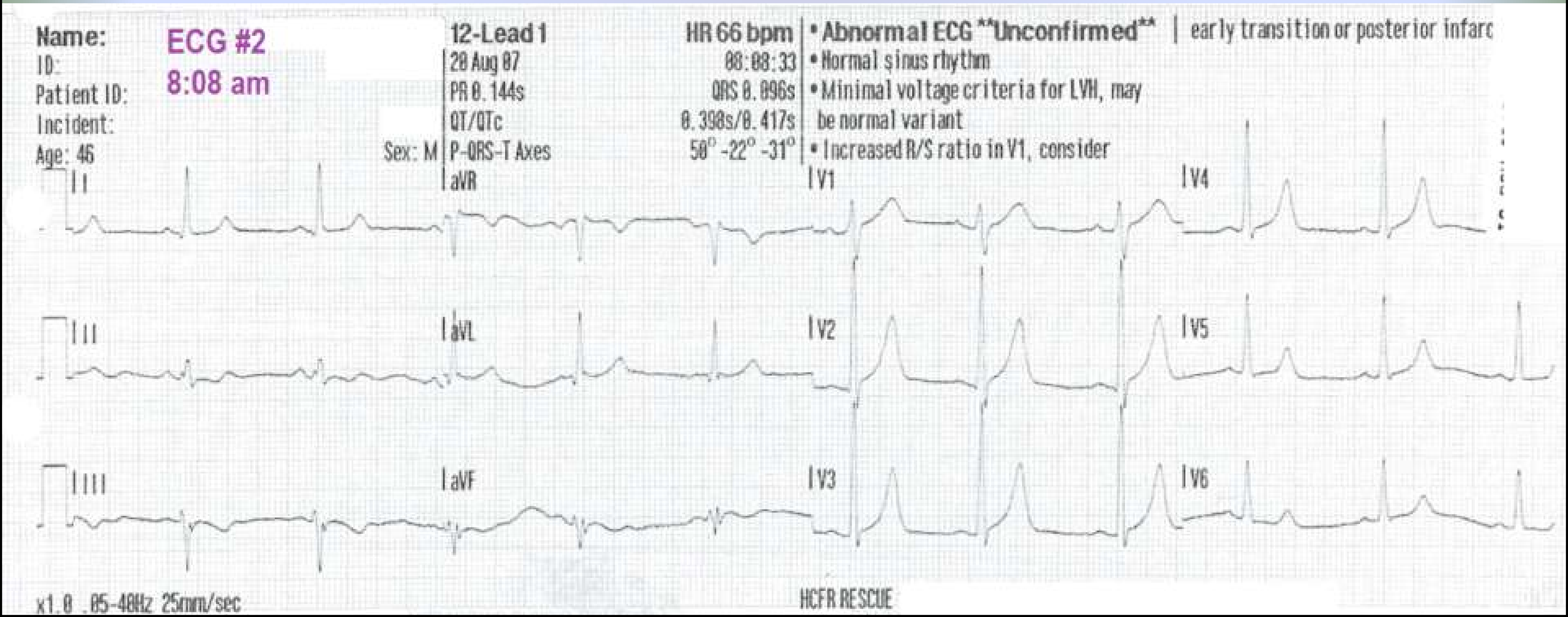
46 year old male

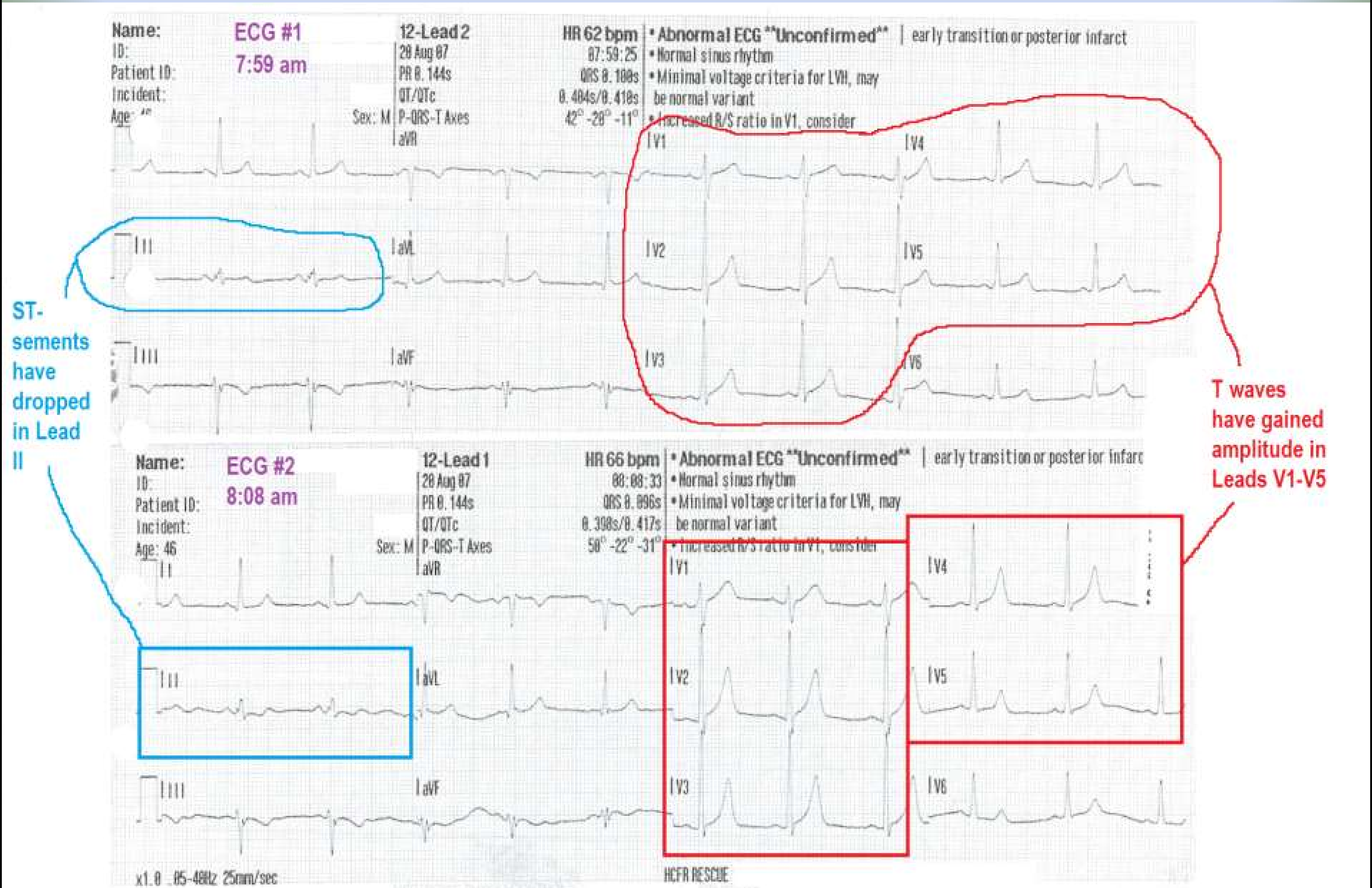
- Exertional dyspnea X "several weeks"
- Intermittent chest pressure X last 3 hours. Currently pain free.



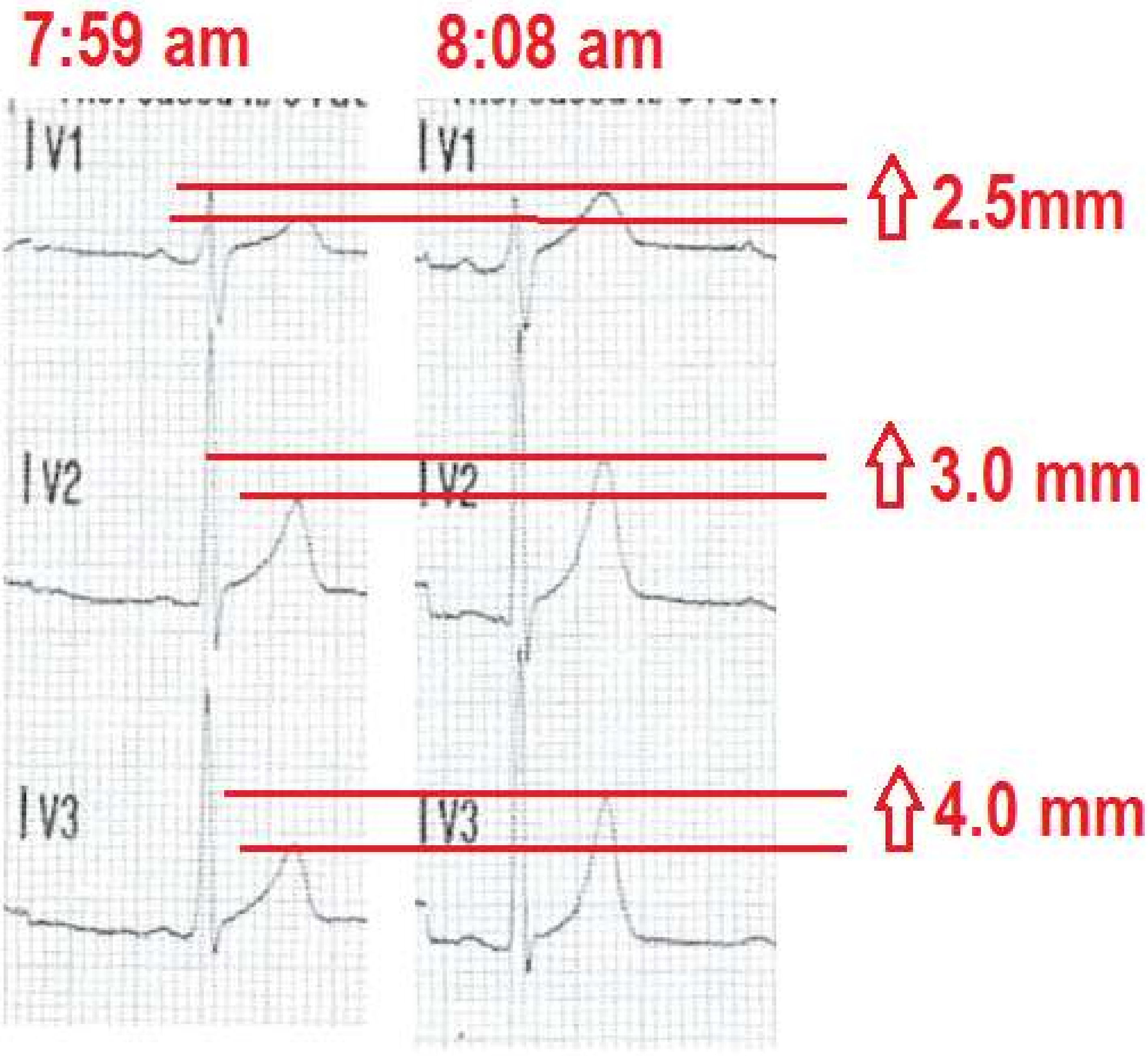
46 year old male: ECG 1

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





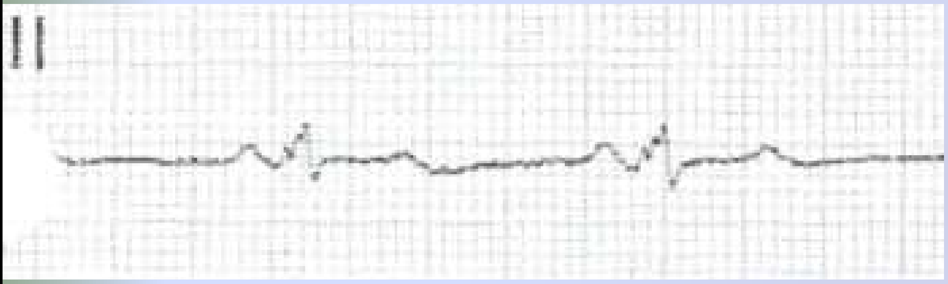
Name:
ID:
Patient ID:
Incident:
ECG #1
7:59 am
12-Lead 2
28 Aug 07
PR 0.144s
QT/QTc
Sex: M P-QRS-T Axes
HR 62 bpm
07:59:25
QRS 0.100s
0.404s/0.410s
42° -28° -11°
• Abnormal ECG **Unconfirmed**
• Normal sinus rhythm
• Minimal voltage criteria for LVH, may be normal variant
• Increased R/S ratio in V1, consider
early transition or posterior infarct
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
ST-sements have dropped in Lead II
T waves have gained amplitude in Leads V1-V5
Name:
ID:
Patient ID:
Incident:
Age: 46
ECG #2
8:08 am
12-Lead 1
28 Aug 07
PR 0.144s
QT/QTc
Sex: M P-QRS-T Axes
HR 66 bpm
08:08:33
QRS 0.096s
0.398s/0.417s
50° -22° -31°
• Abnormal ECG **Unconfirmed**
• Normal sinus rhythm
• Minimal voltage criteria for LVH, may be normal variant
early transition or posterior infarc
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
x1.0 .05-40Hz 25mm/sec
HCFR RESCUE



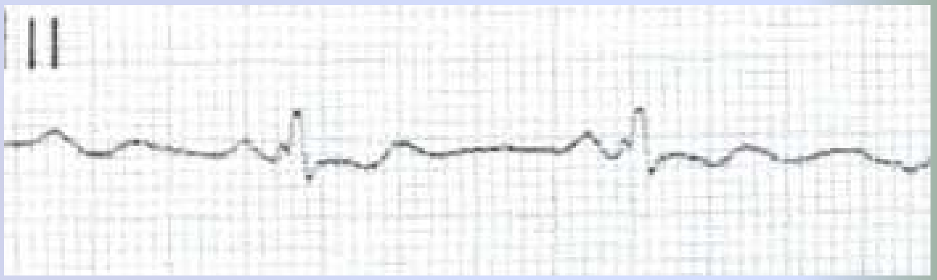
7:59 am
8:08 am
V1
V1
2.5mm
V2
V2
3.0 mm
V3
V3
4.0 mm

ST-Segment Depression

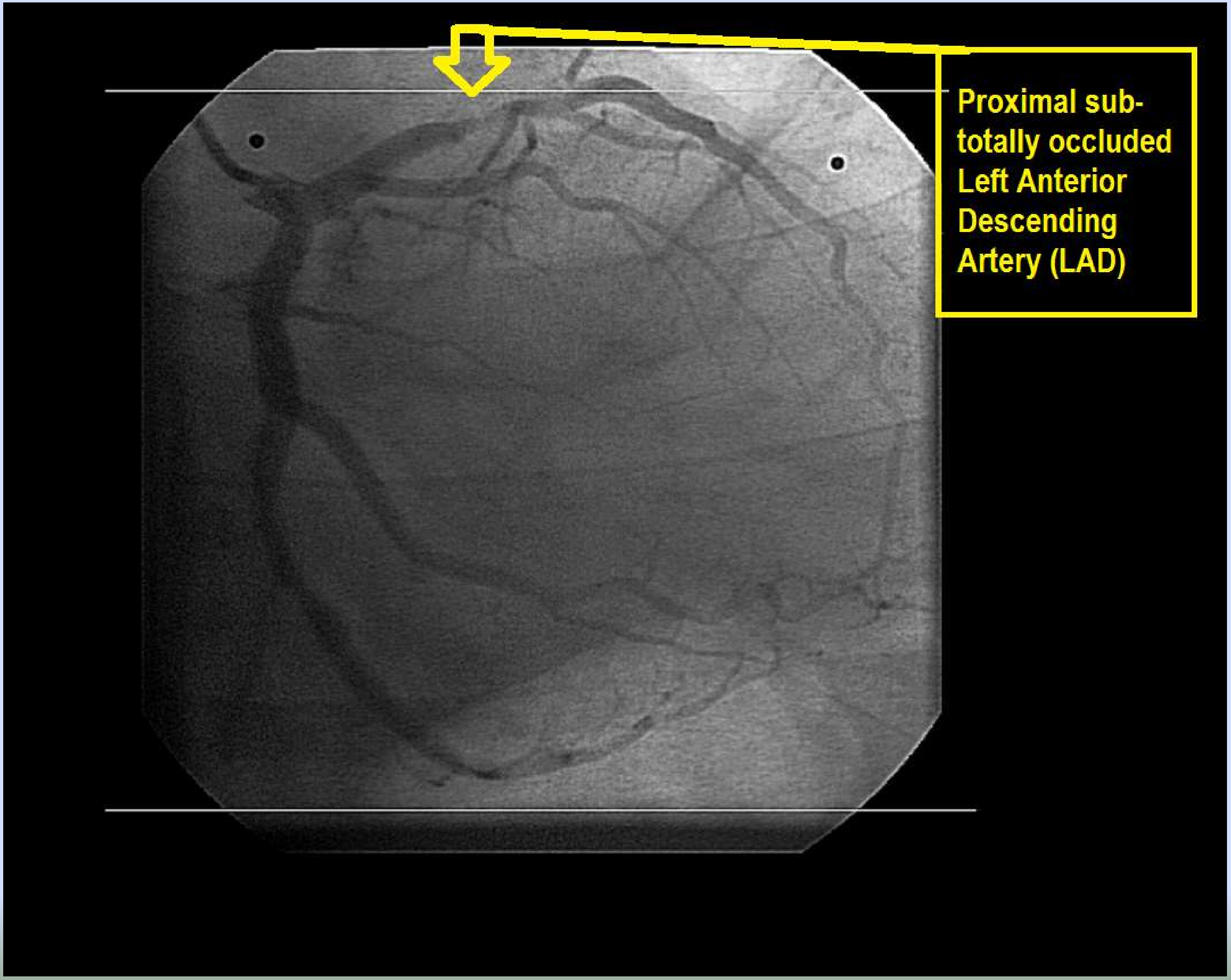
7:59 am



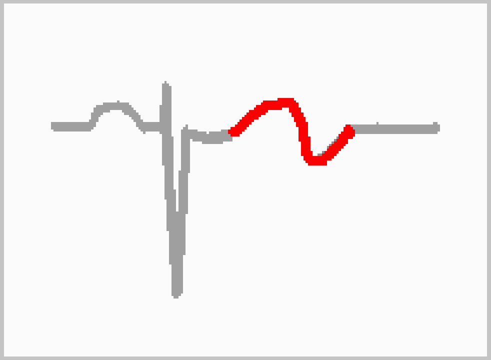
8:08 am



Cath Lab Angiography:



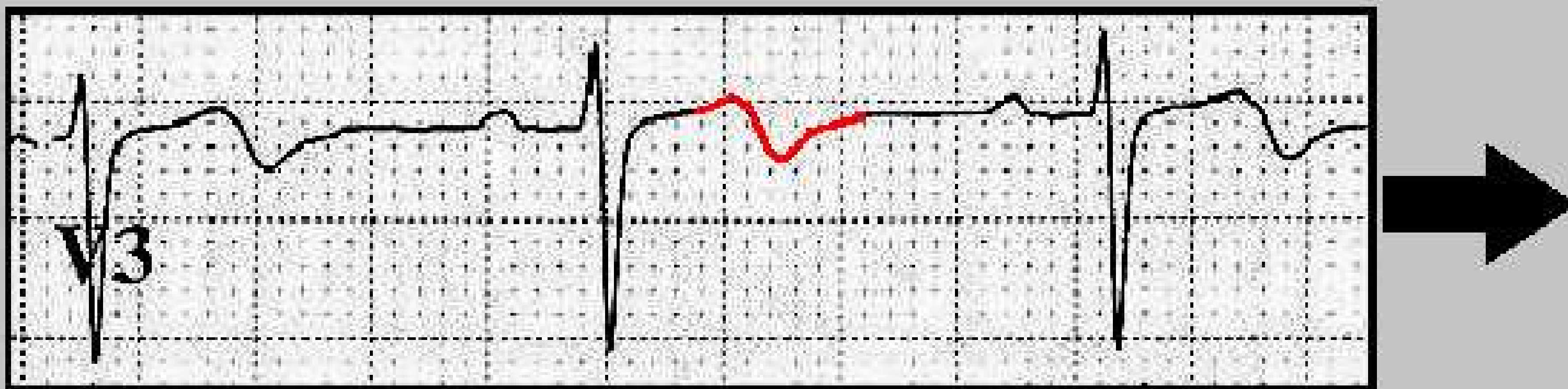
ISCHEMIA



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:

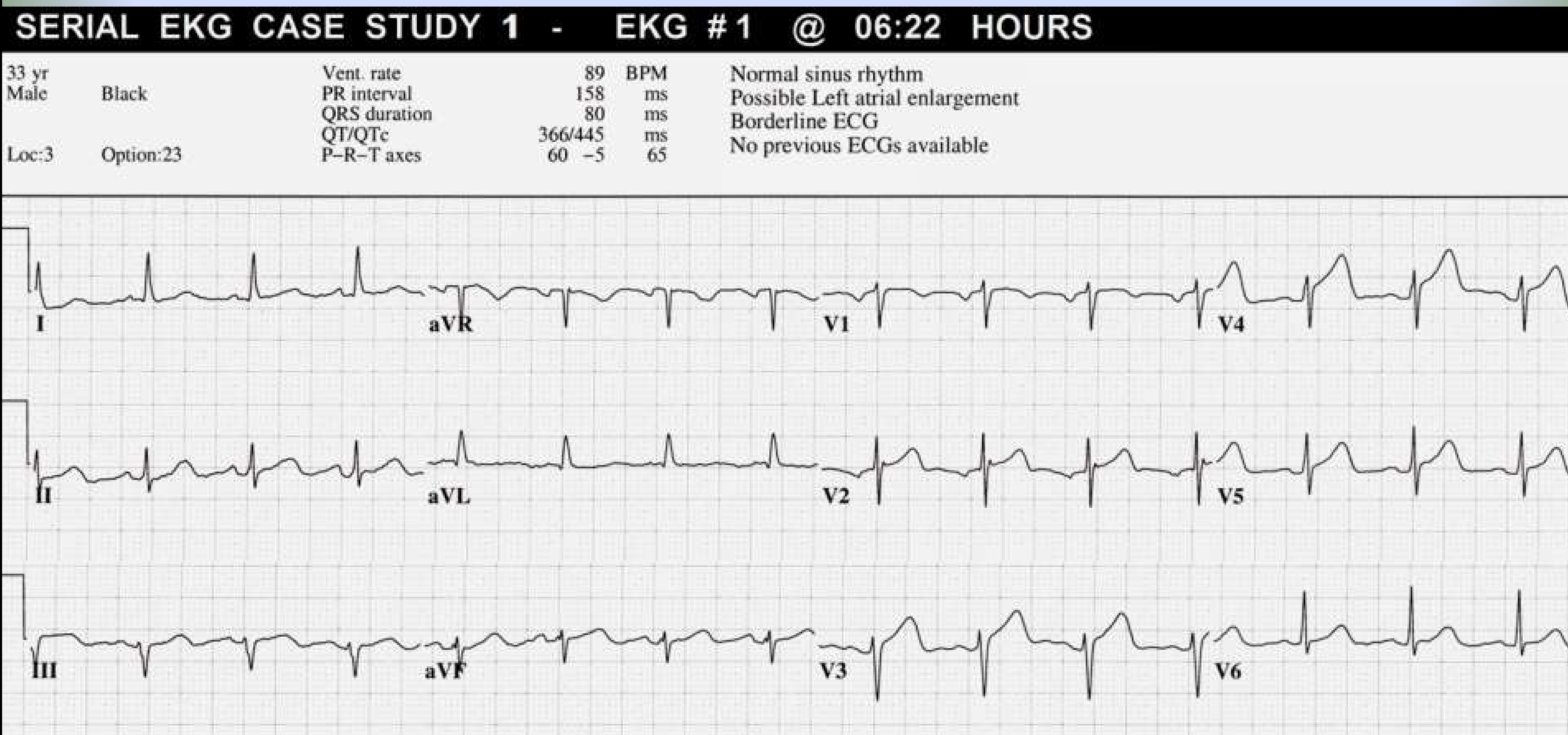
- Present in 14-18% of patients admitted with unstable angina
- 75% patients not treated developed extensive Anterior MI within 3 weeks.
- *Median Average time from presentation to Acute Myocardial Infarction – 8 days*

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

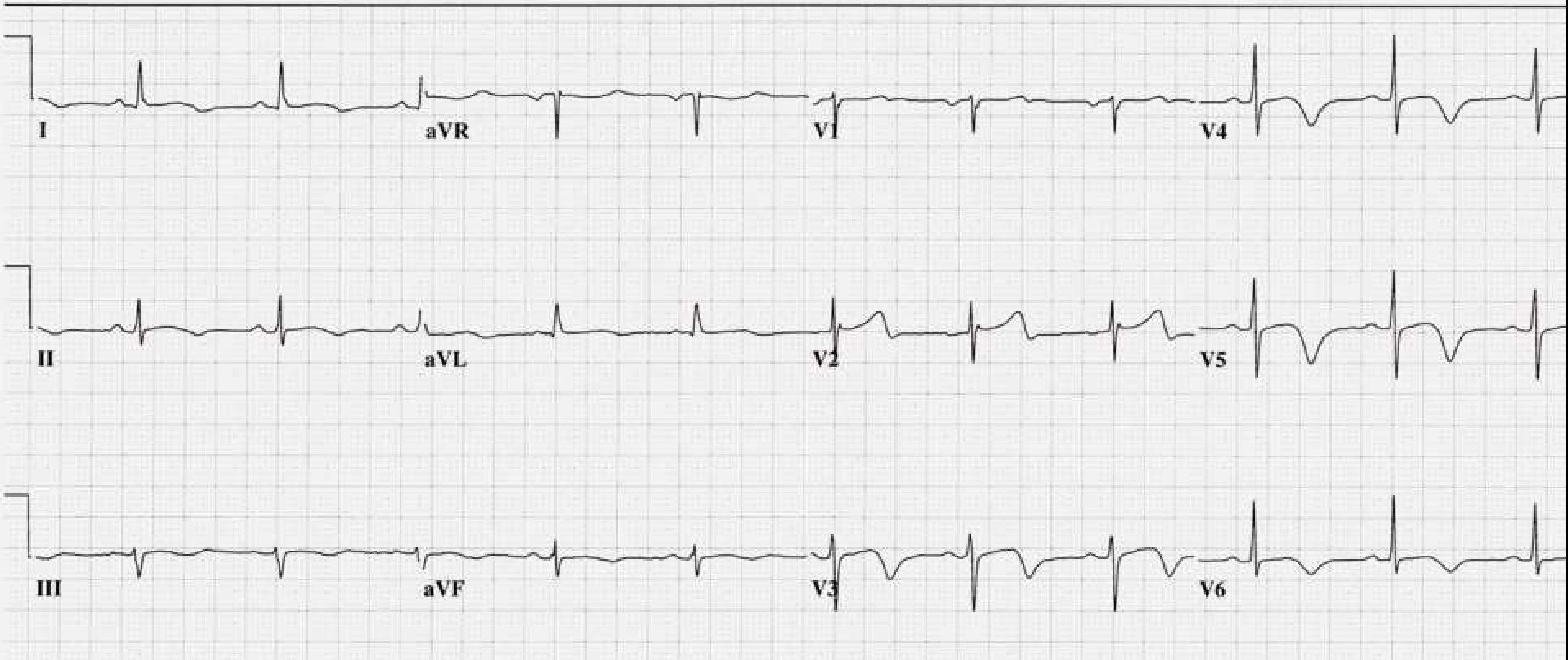
SERIAL EKG CASE STUDY 1 - EKG # 2 @ 09:42 HOURS

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

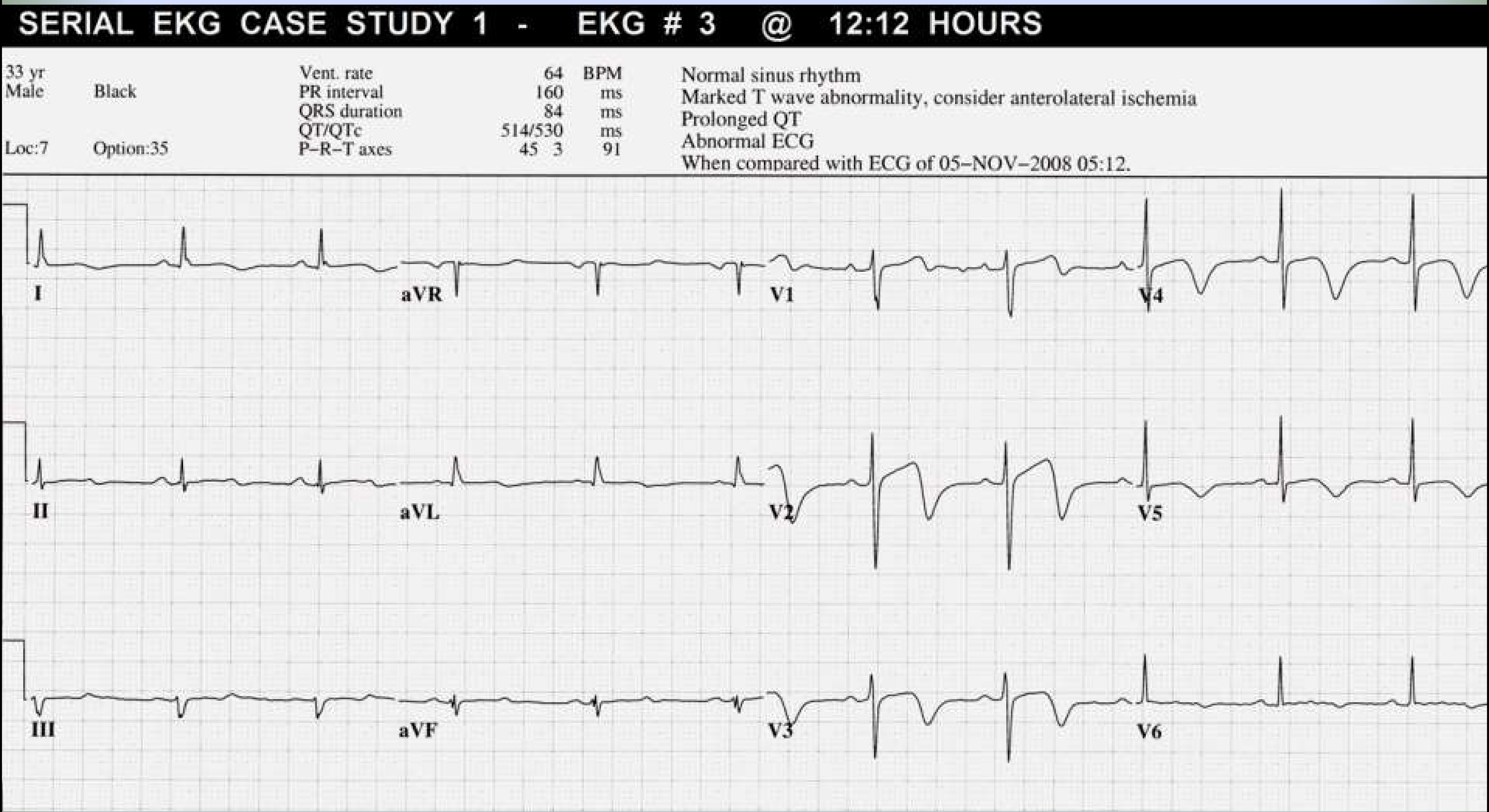
is the time for the

STAT CALL

to the

CARDIOLOGIST !!!!

Wellen’s Syndrome Case Study



Wellen's Syndrome Case Study

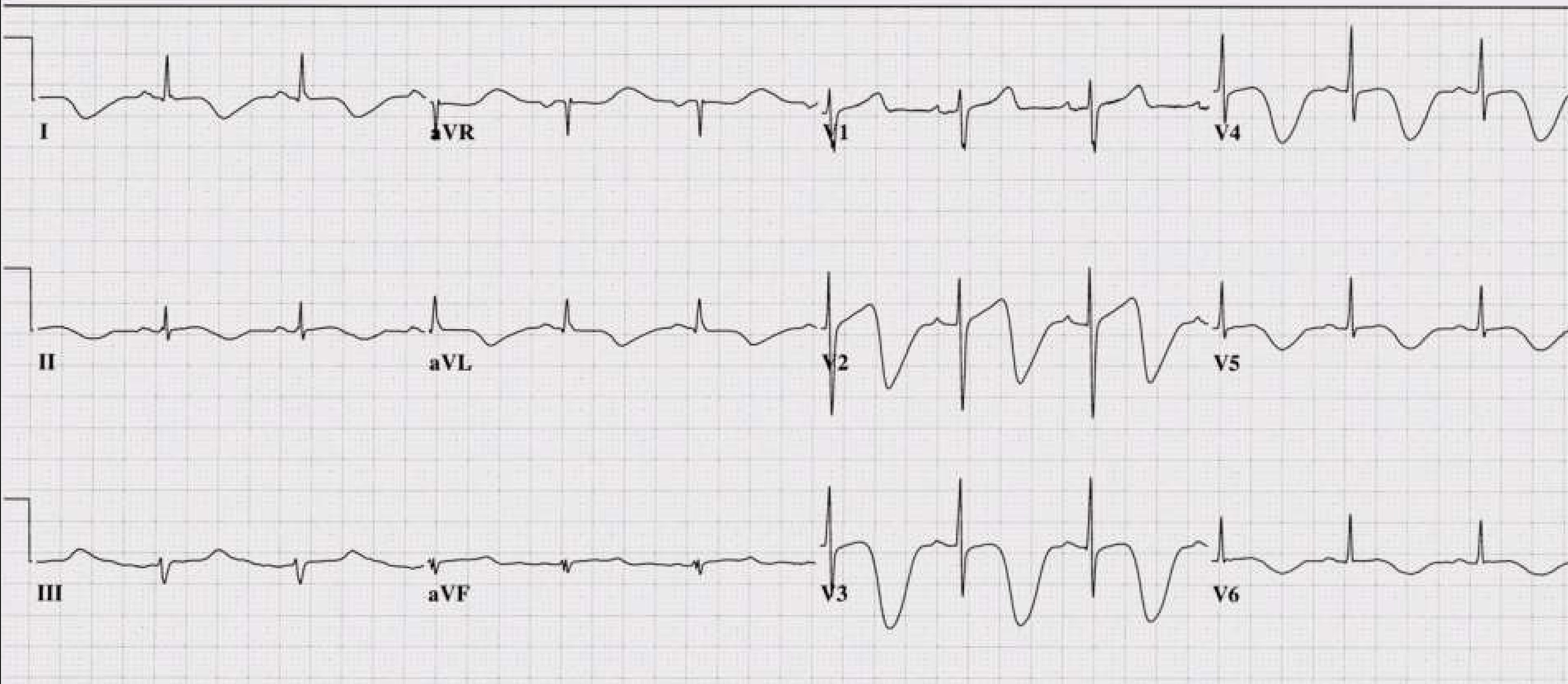
SERIAL EKG CASE STUDY 1 - EKG # 4 @ 15:37 HOURS

33 yr
Male Black

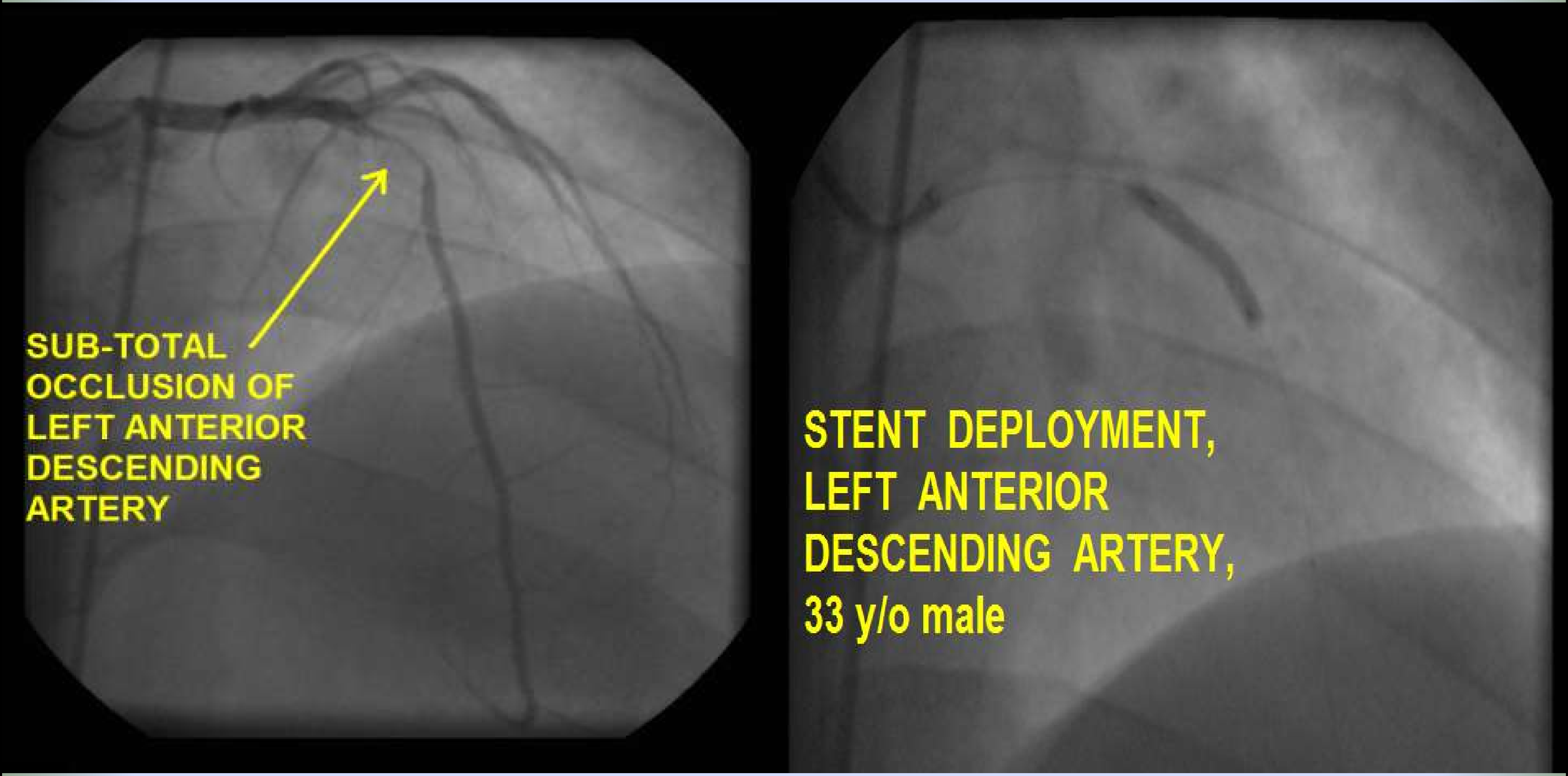
Room:405A
Loc:5 Option:39

Vent. rate	71	BPM
PR interval	144	ms
QRS duration	74	ms
QT/QTc	600/652	ms
P–R–T axes	20 1	160

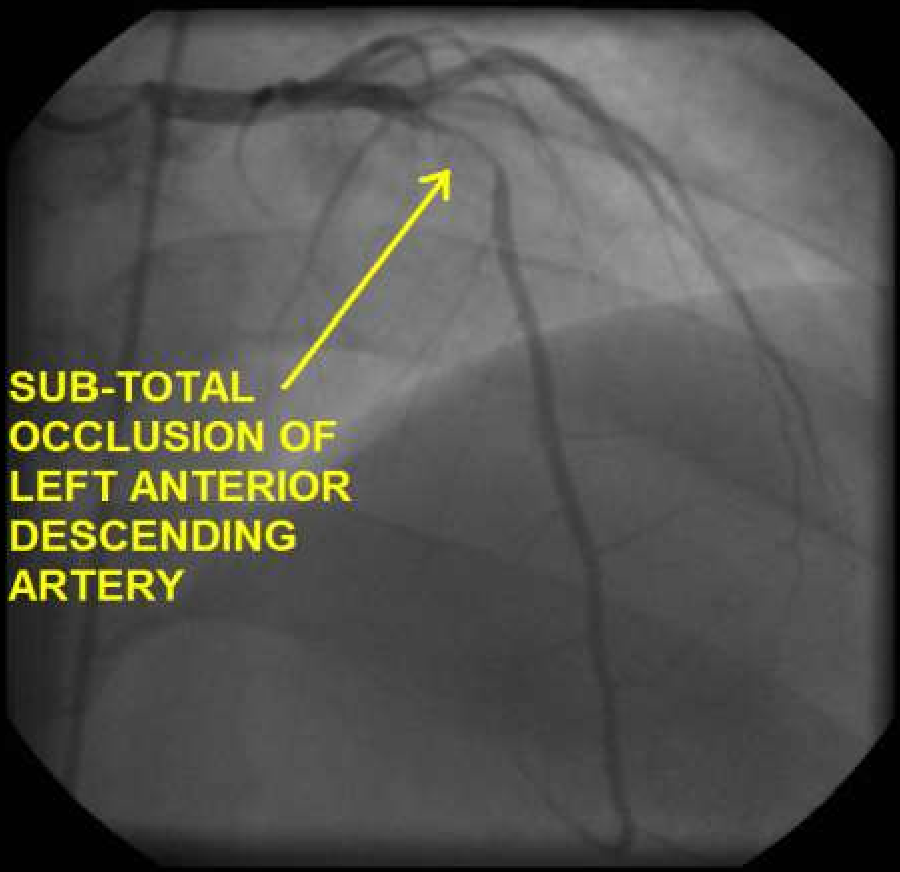
Normal sinus rhythm
Marked T wave abnormality, consider anterolateral ischemia
Prolonged QT
Abnormal ECG

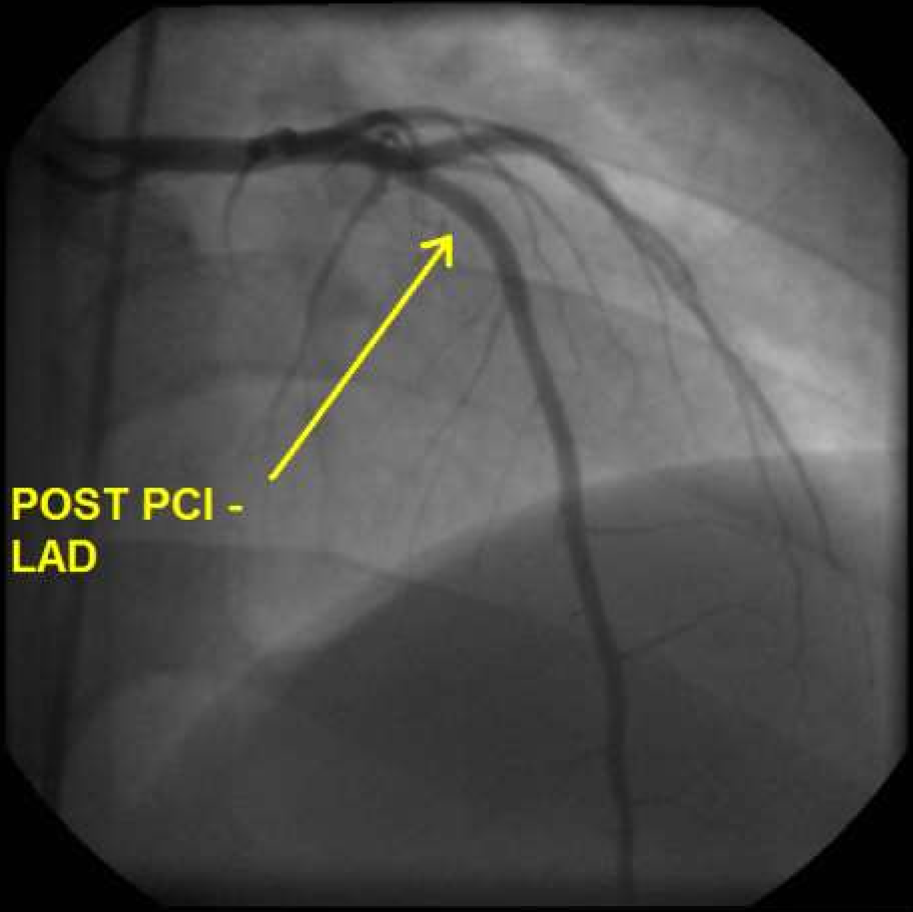


Wellen’s Syndrome Case Study



Wellen's Syndrome Case Study





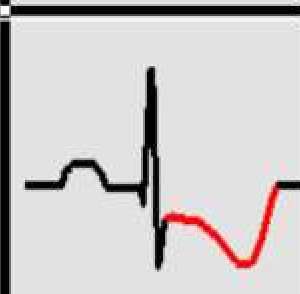
Additional Resources:

- Wellen’s Syndrome, NEJM case study

“NOWHERE”, NEW MEXICO, 1994

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**

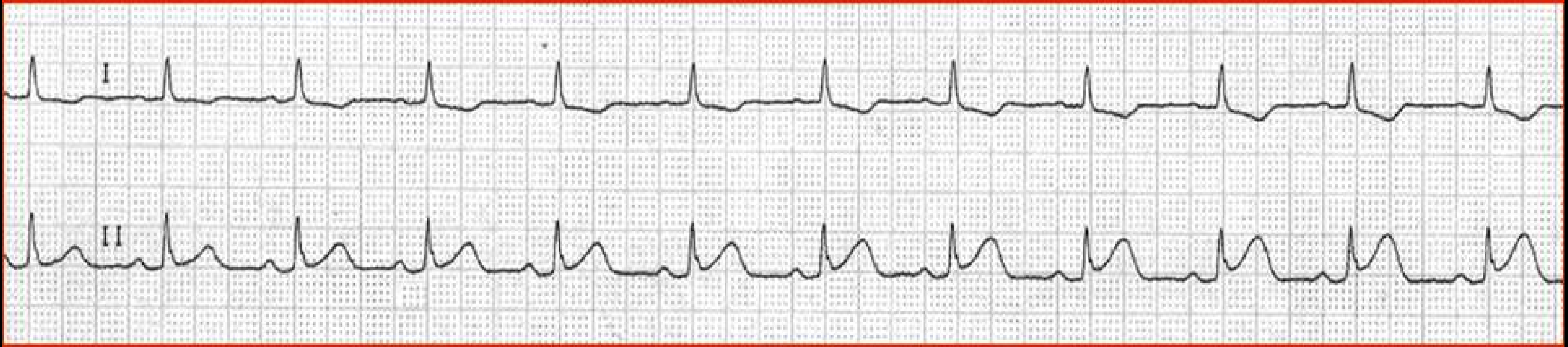
Abnormal ST Elevation Criteria: ACC/AHA 2009 "Standardization and Interpretation of the ECG, Part VI Acute Ischemia and Infarction," Galen Wagner, et al

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.

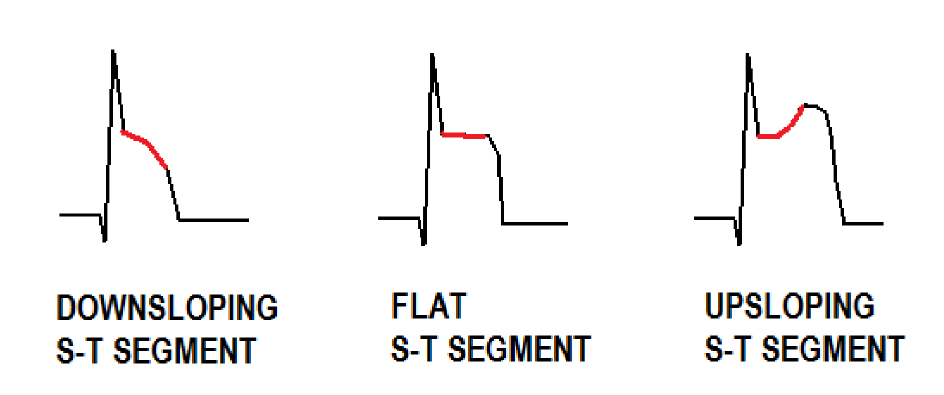
ST SEGMENT ELEVATION:

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



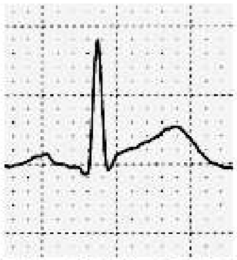
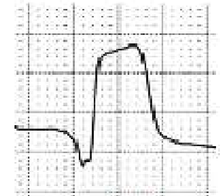
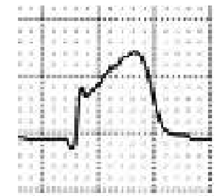
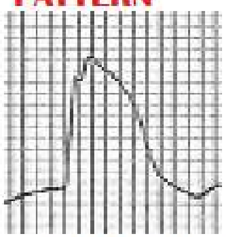
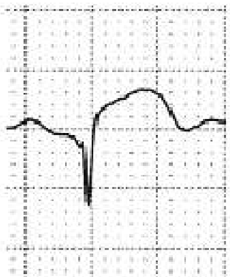
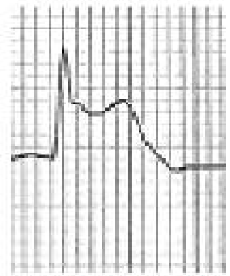
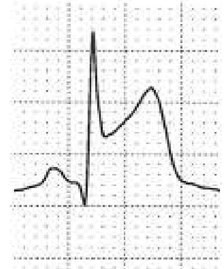
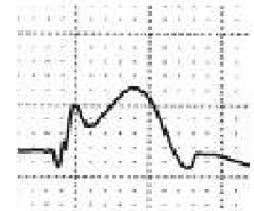
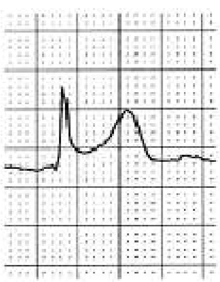
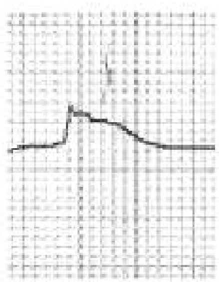
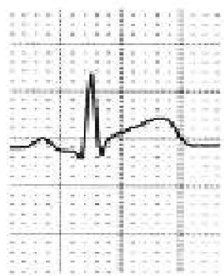
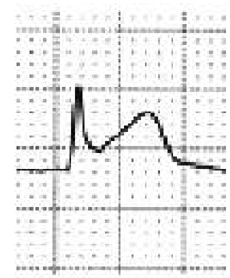
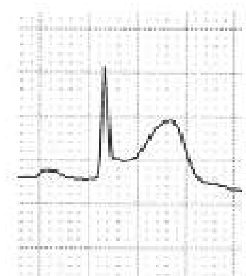
IN THIS CASE, a normal response to balloon occlusion of the RIGHT CORONARY ARTERY during PTCA in the CARDIAC CATH LAB

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.

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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

Lancaster County, Pennsylvania
Winter, 2002

STEMI

- **Correlation of ECG Leads with Coronary Arterial Anatomy and the STRUCTURES SERVED by the OCCLUDED ARTERY . . . .**

STEMI

- **Correlation of ECG Leads with Coronary Arterial Anatomy and the STRUCTURES SERVED by the OCCLUDED ARTERY . . . .**

. . . . . Will serve as a “crystal ball,” allowing you to ANTICIPATE complications of STEMI . . . .

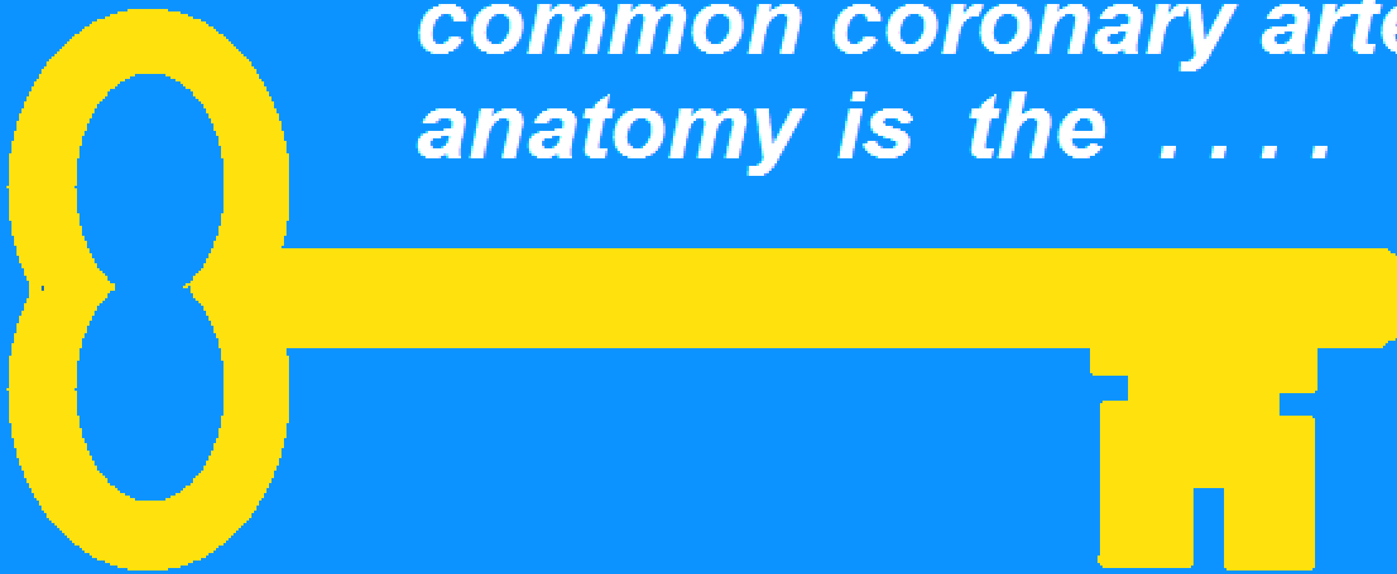
STEMI

- **Correlation of ECG Leads with Coronary Arterial Anatomy and the STRUCTURES SERVED by the OCCLUDED ARTERY . . . .**

. . . . . Will serve as a "crystal ball," allowing you to ANTICIPATE complications of STEMI . . . .

. . . . BEFORE they occur !!

"Having knowledge of common coronary artery anatomy is the . . . .



to understanding the PHYSIOLOGICAL CHANGES that occur during ACUTE MI."

"an INVALUABLE ASSET for ALL MEDICAL PROFESSIONALS who provide direct care to STEMI patients !"

INTERPRET THE EKG, THEN:

- IDENTIFY THE AREA OF THE HEART WITH A PROBLEM . . .
- RECALL THE ARTERY WHICH SERVES THAT REGION . . .
- RECALL OTHER STRUCTURES SERVED BY THAT ARTERY . . .
- ANTICIPATE FAILURE OF THOSE STRUCTURES . . .
- *INTERVENE APPROPRIATELY !*

STEMI Case Studies, excerpts from "[12 Lead ECG Interpretation in ACS with Case Studies from the Cardiac Cath Lab]."

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.

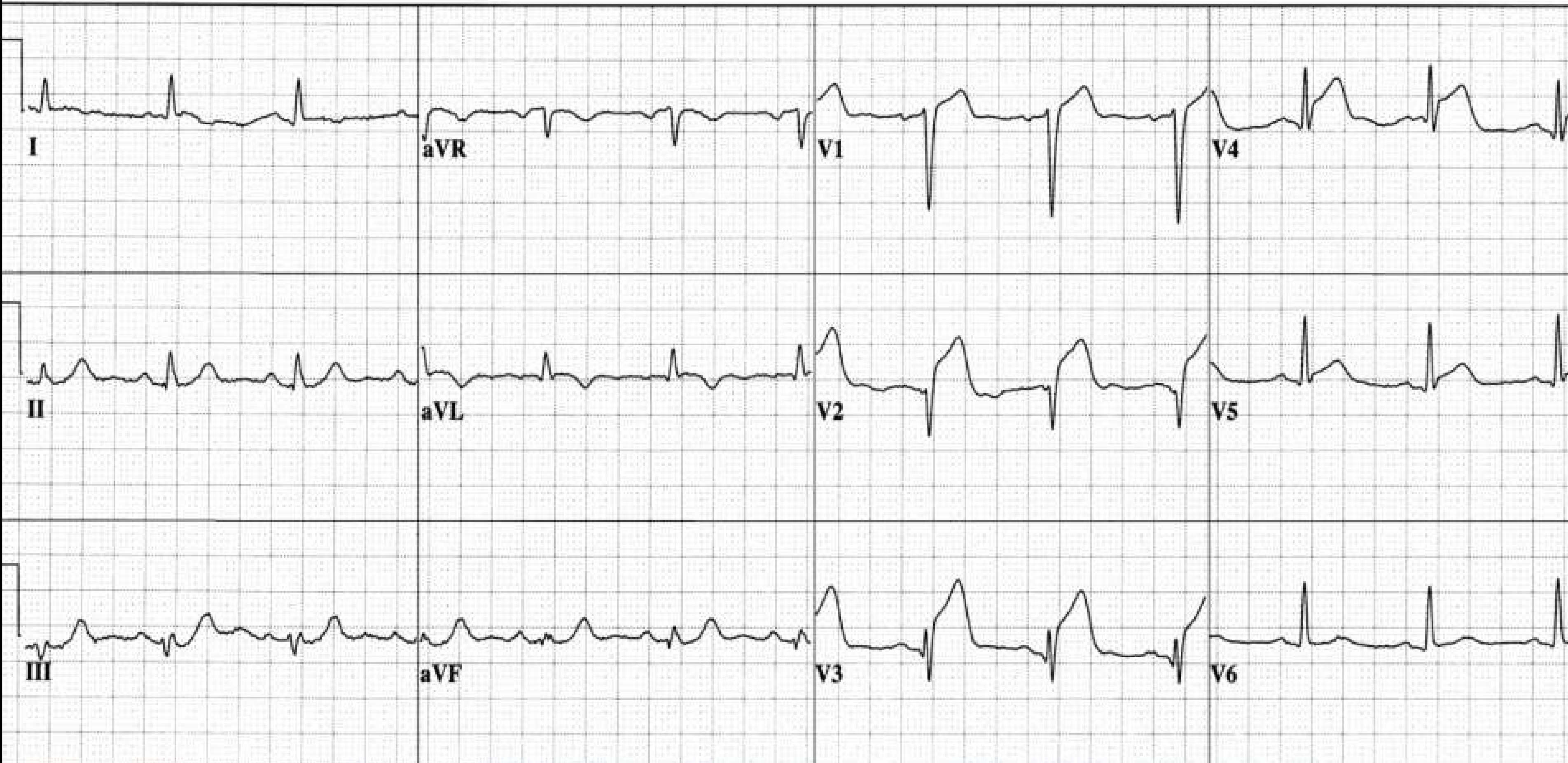
VITAL SIGNS: BP: 100/64, P: 75, R: 20, SAO2: 94%

LABS: FIRST TROPONIN: 6.4

72 yr		Vent. rate	75	BPM
Male	Caucasian	PR interval	162	ms
		QRS duration	98	ms
		QT/QTc	382/426	ms
Loc:3	Option:23	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

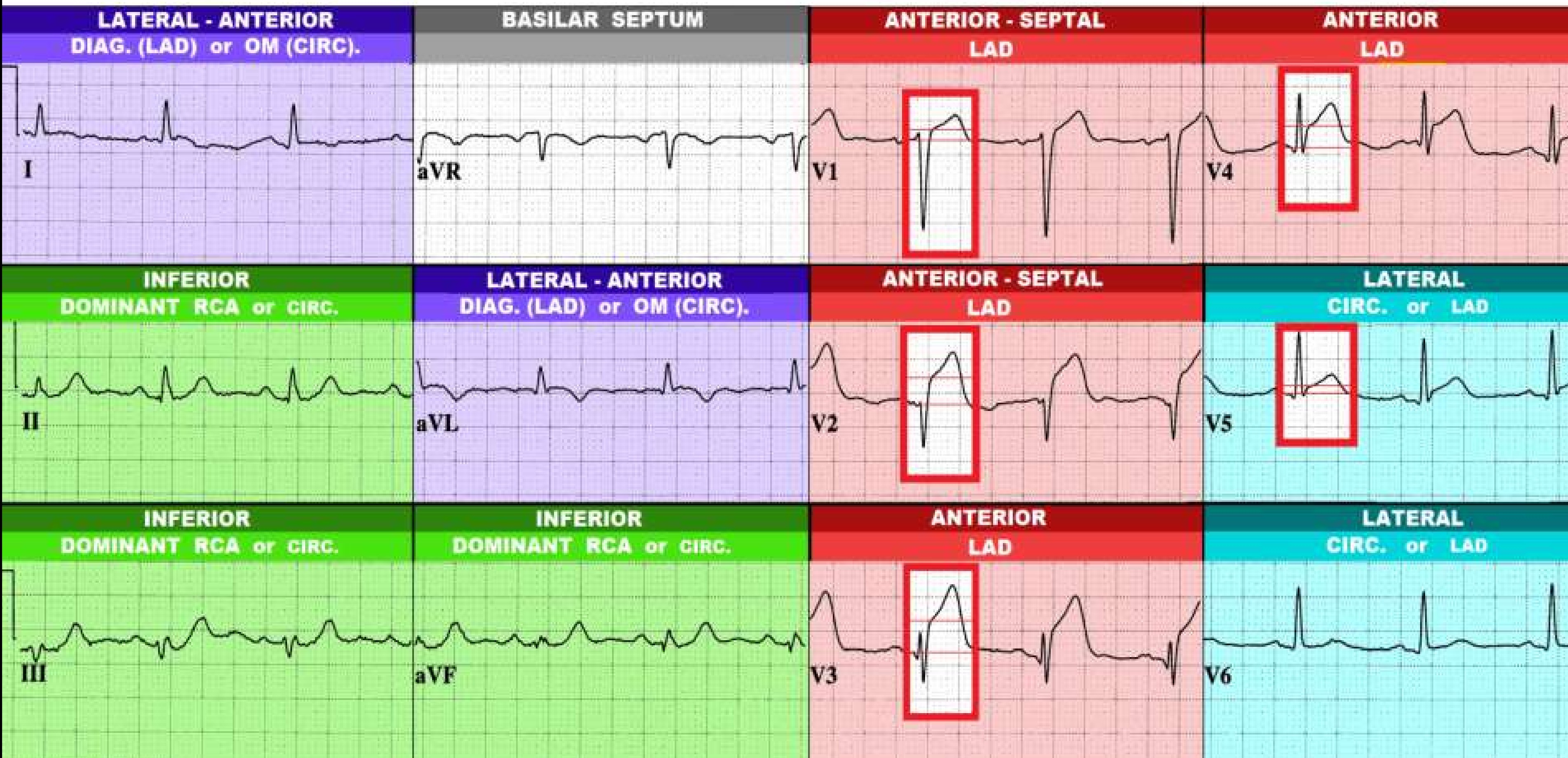


72 yr
Male Caucasian
Loc: Option:2

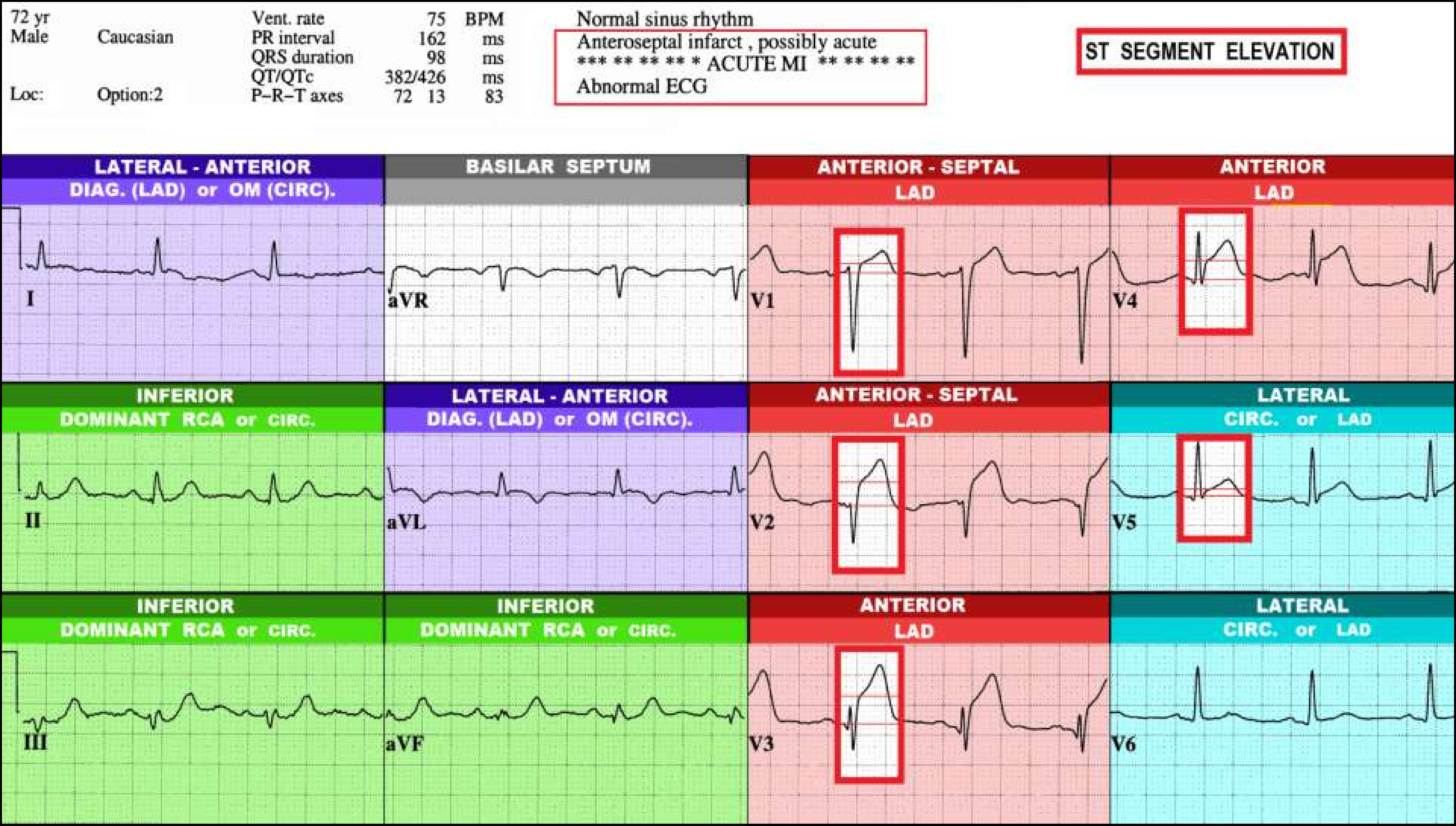
Vent. rate 75 BPM
PR interval 162 ms
QRS duration 98 ms
QT/QTc 382/426 ms
P–R–T axes 72 13 83

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

ST SEGMENT ELEVATION



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).
BASILAR SEPTUM
ANTERIOR - SEPTAL
LAD
ANTERIOR
LAD
I
aVR
V1
V4
INFERIOR
DOMINANT RCA or CIRC.
LATERAL - ANTERIOR
DIAG. (LAD) or OM (CIRC).
ANTERIOR - SEPTAL
LAD
LATERAL
CIRC. or LAD
II
aVL
V2
V5
INFERIOR
DOMINANT RCA or CIRC.
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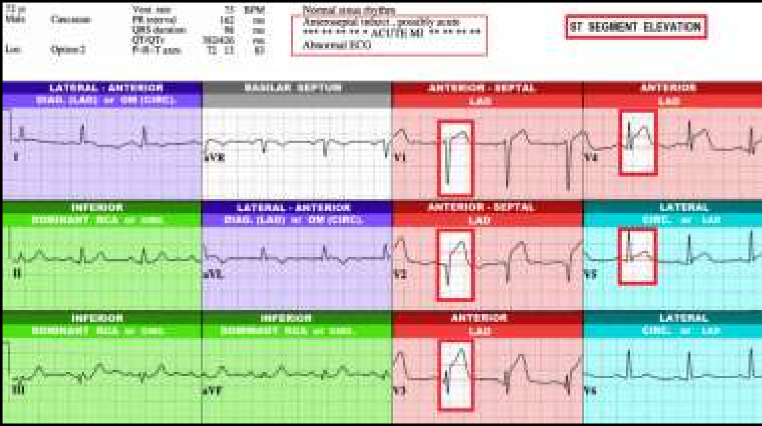
DOMINANT RCA or CIRC.
ANTERIOR
LAD
LATERAL
CIRC. or LAD
III
aVF
V3
V6

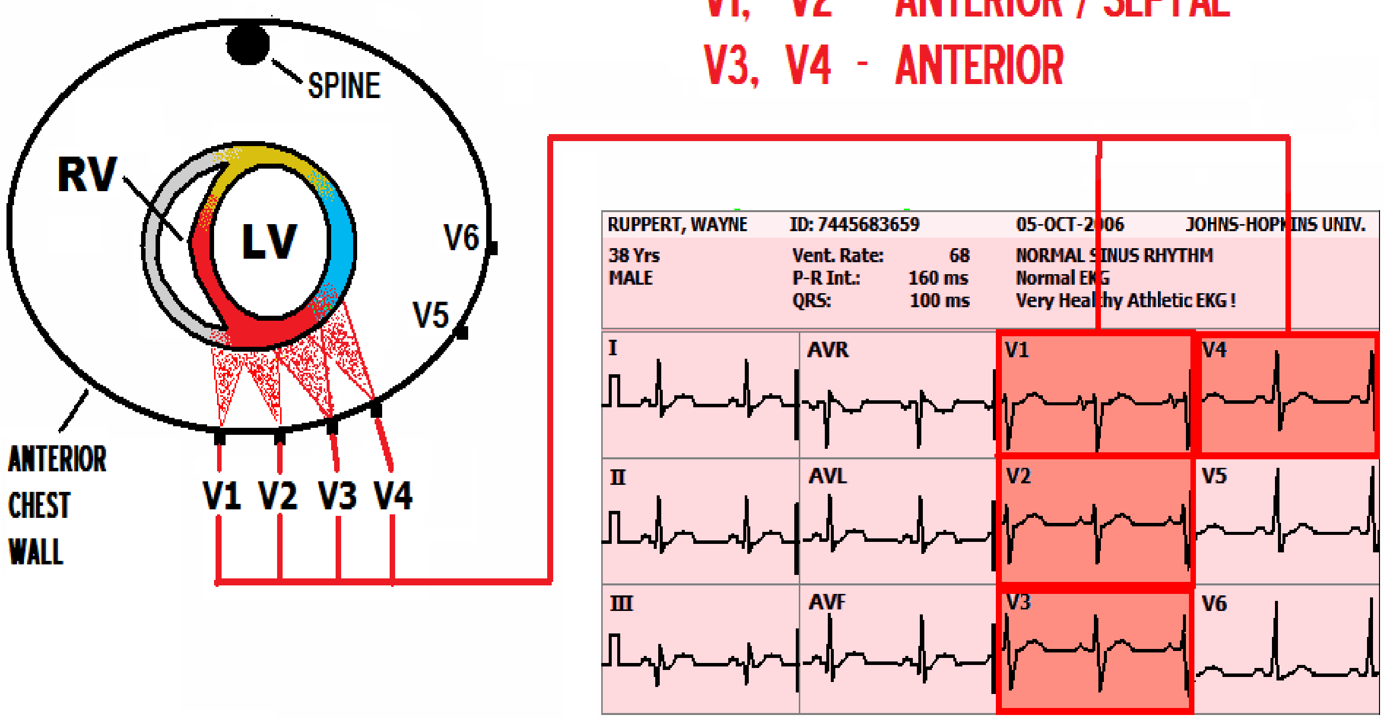


V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL

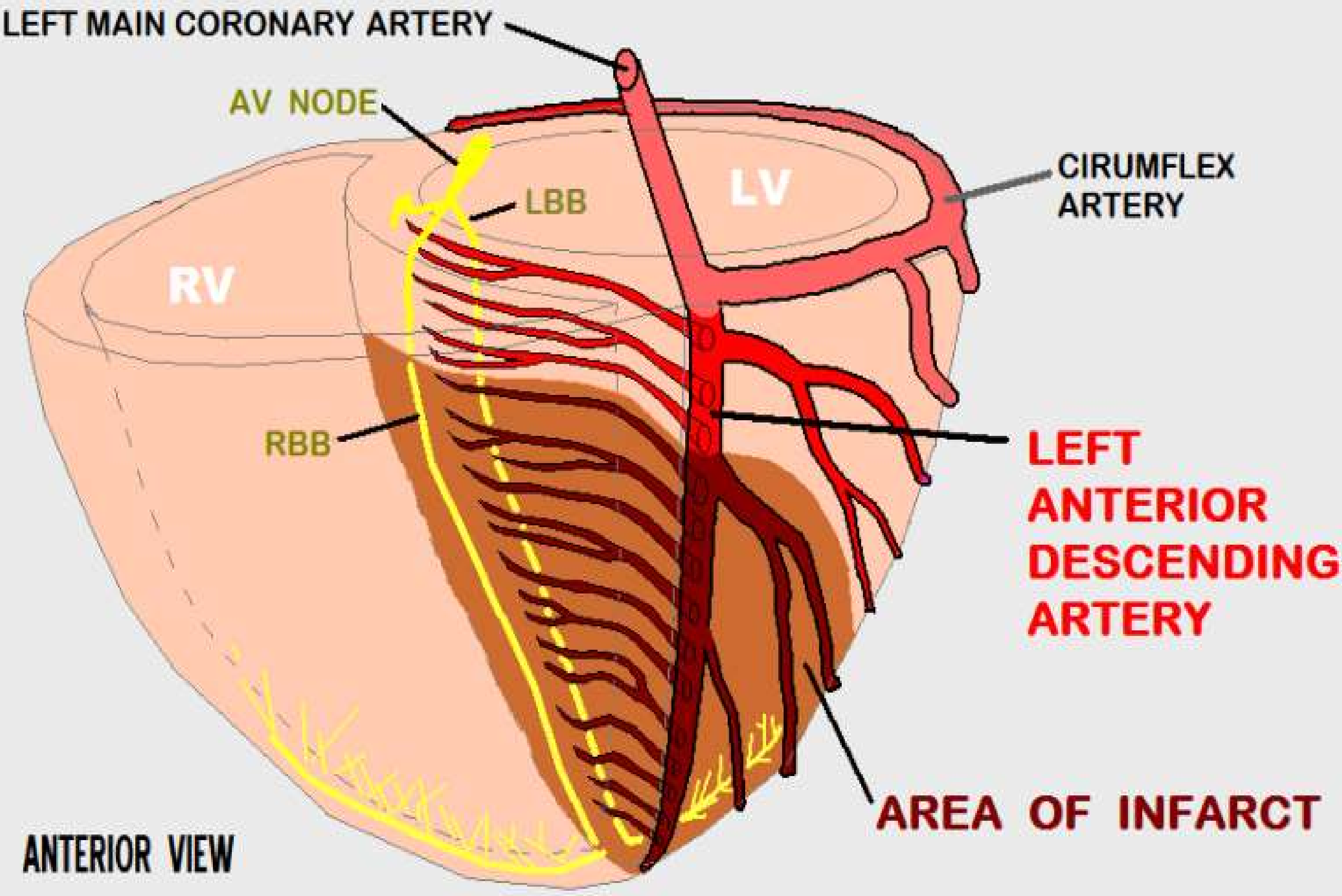
of the LEFT VENTRICLE

V1, V2 - ANTERIOR / SEPTAL

V3, V4 - ANTERIOR

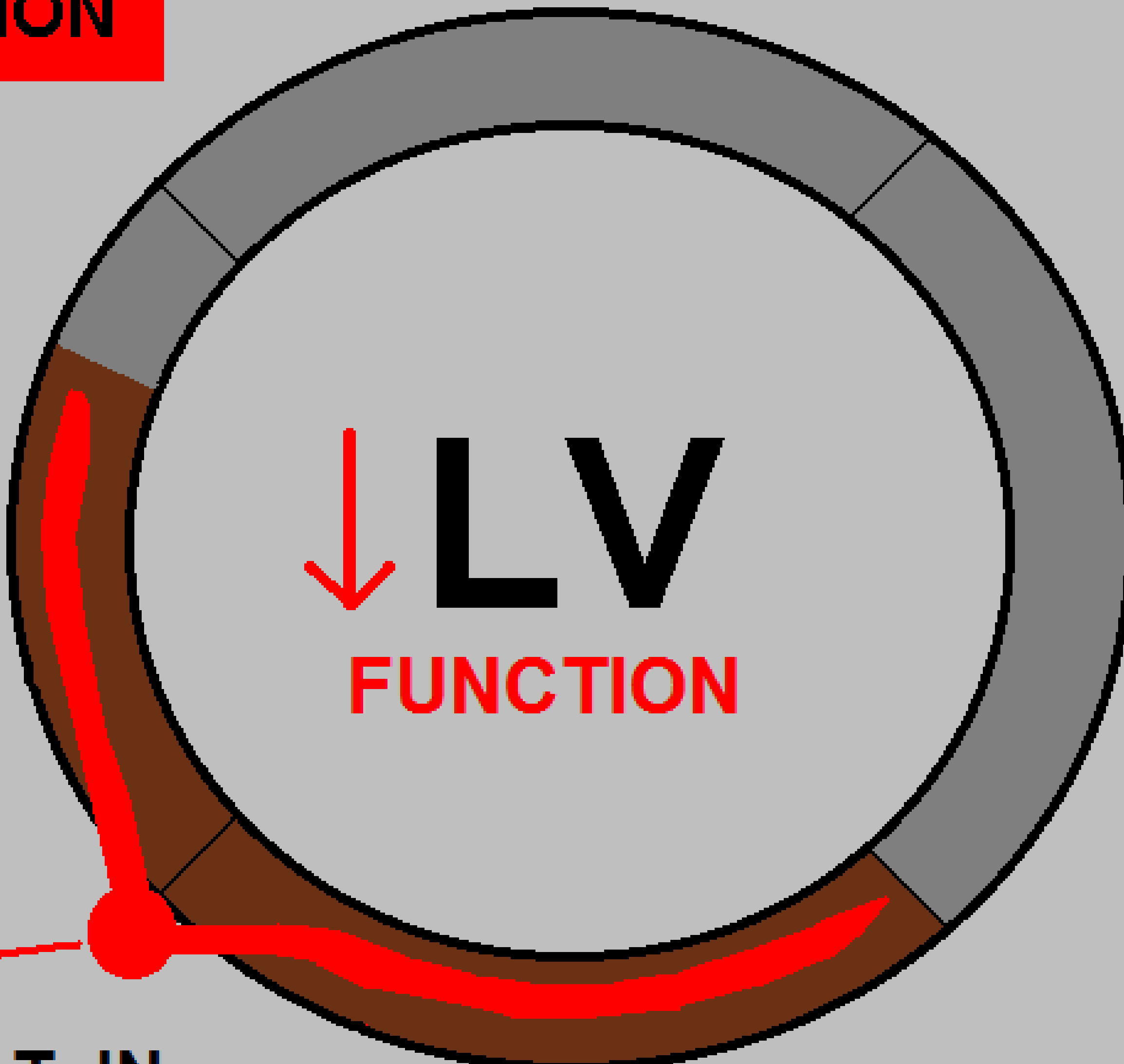


OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY



LAD DISTRIBUTION

35 - 45 % of LV MUSCLE MASS



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

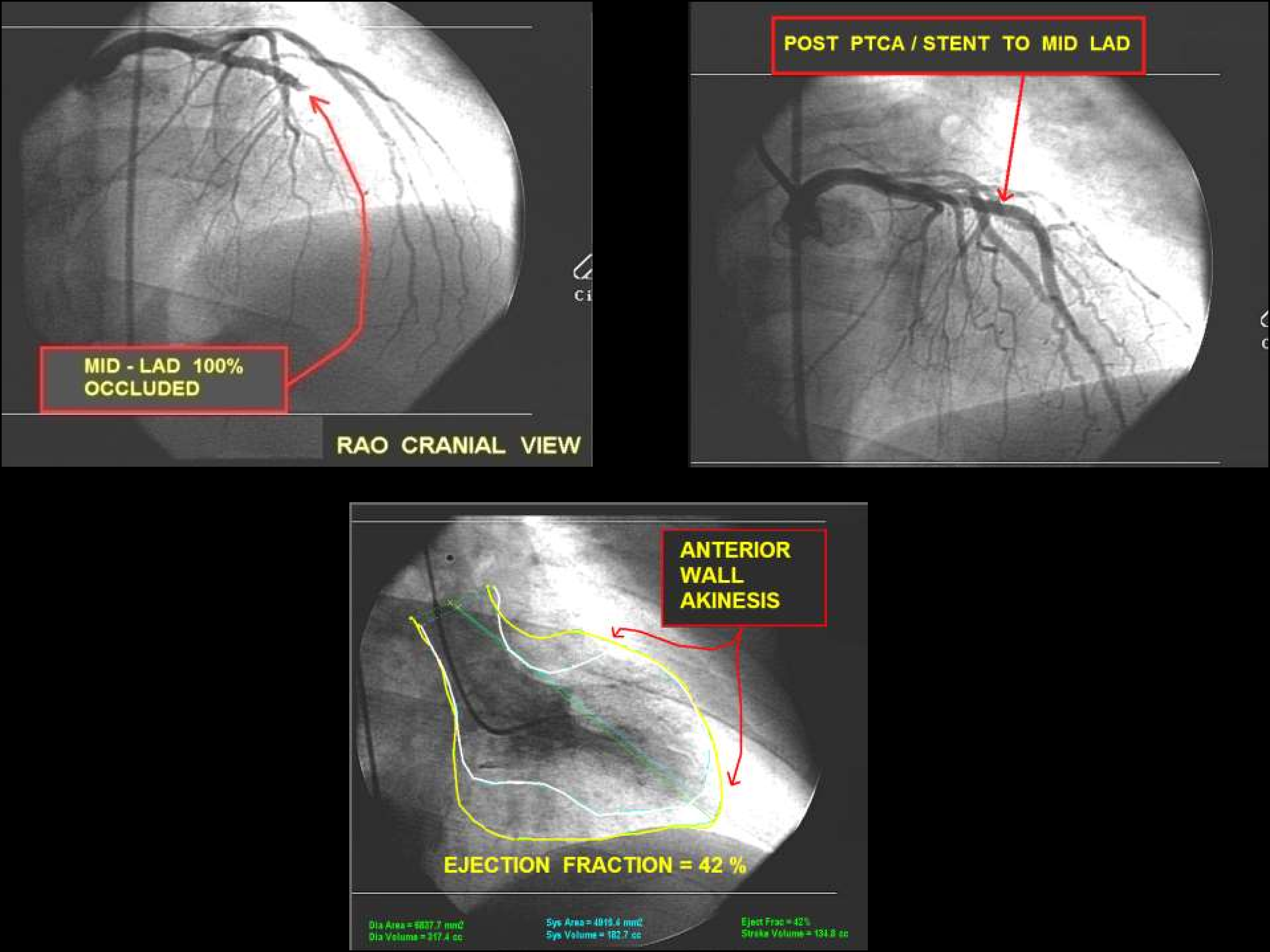
- **CARDIOGENIC SHOCK** ☠
- **PULMONARY EDEMA** ☠


400 mg
Dopamine
(1600 mcg/mL)
Dopamine Hydrochloride
Dopamine
and 5% Dextrose Injectio
250 mL
Baxter
500 mg
DOBUTamin
Hydrochlori
5% Dextros
(2000 mc
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 %
Sys Volume = 182.7 cc
Eject Frac = 42%

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

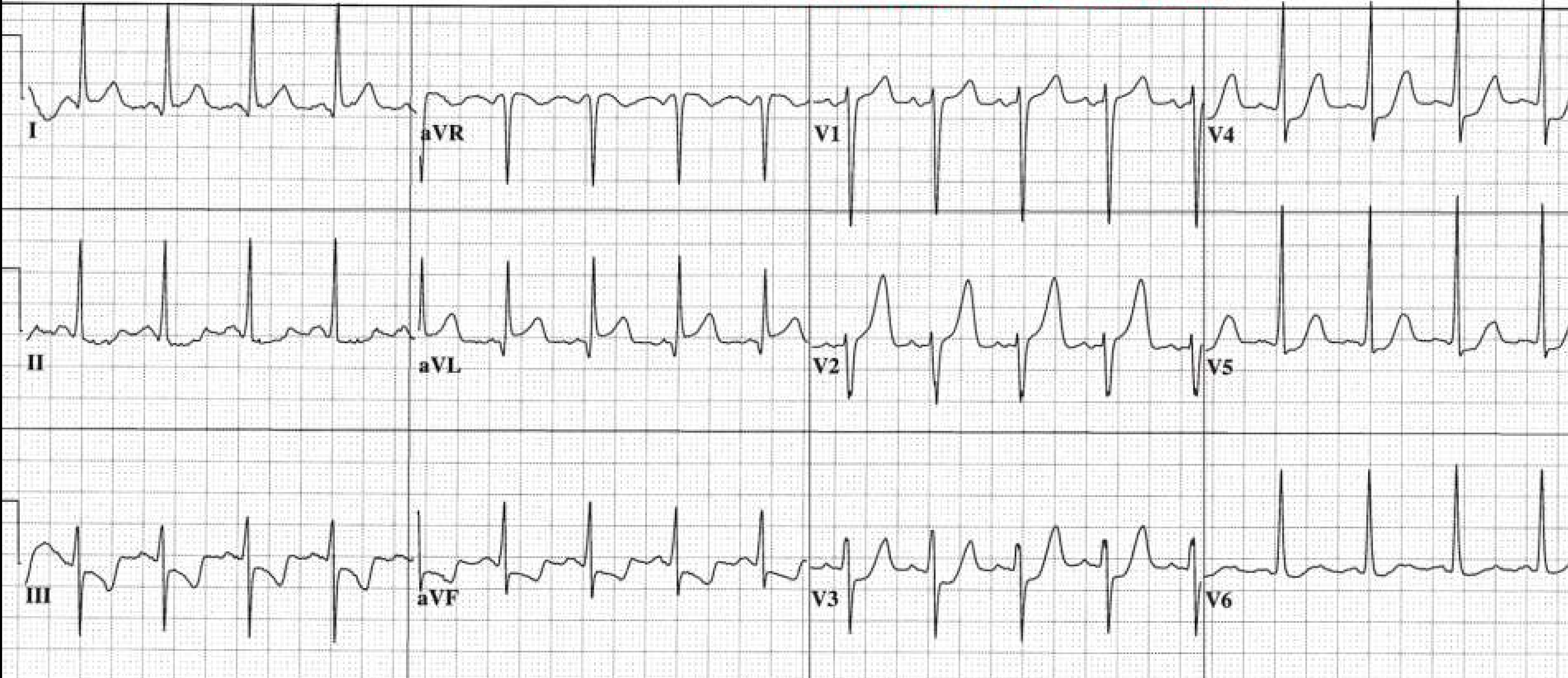
46 yr
Female

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

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

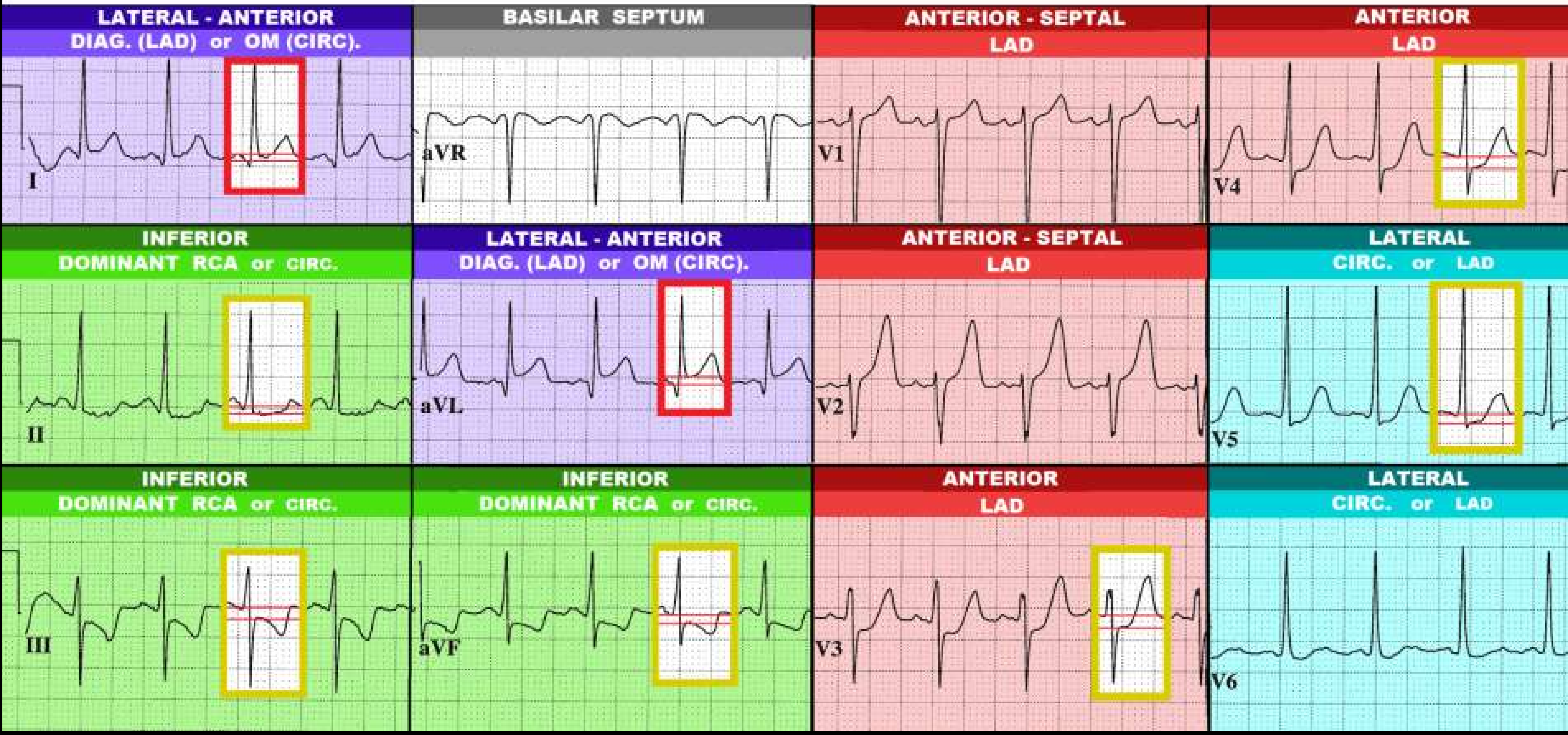
Room:ER

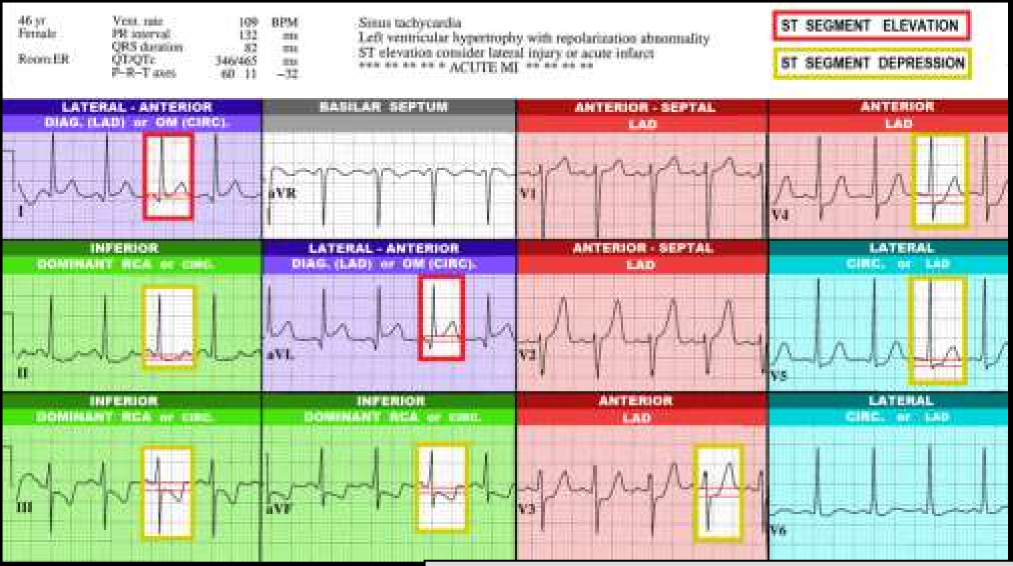
Vent. rate	109	BPM	
PR interval	132	ms	
QRS duration	82	ms	
QT/QTc	346/465	ms	
P–R–T axes	60	11	–32

Sinus tachycardia
Left ventricular hypertrophy with repolarization abnormality
ST elevation consider lateral injury or acute infarct
*** ** ** ** * ACUTE MI ** ** ** **

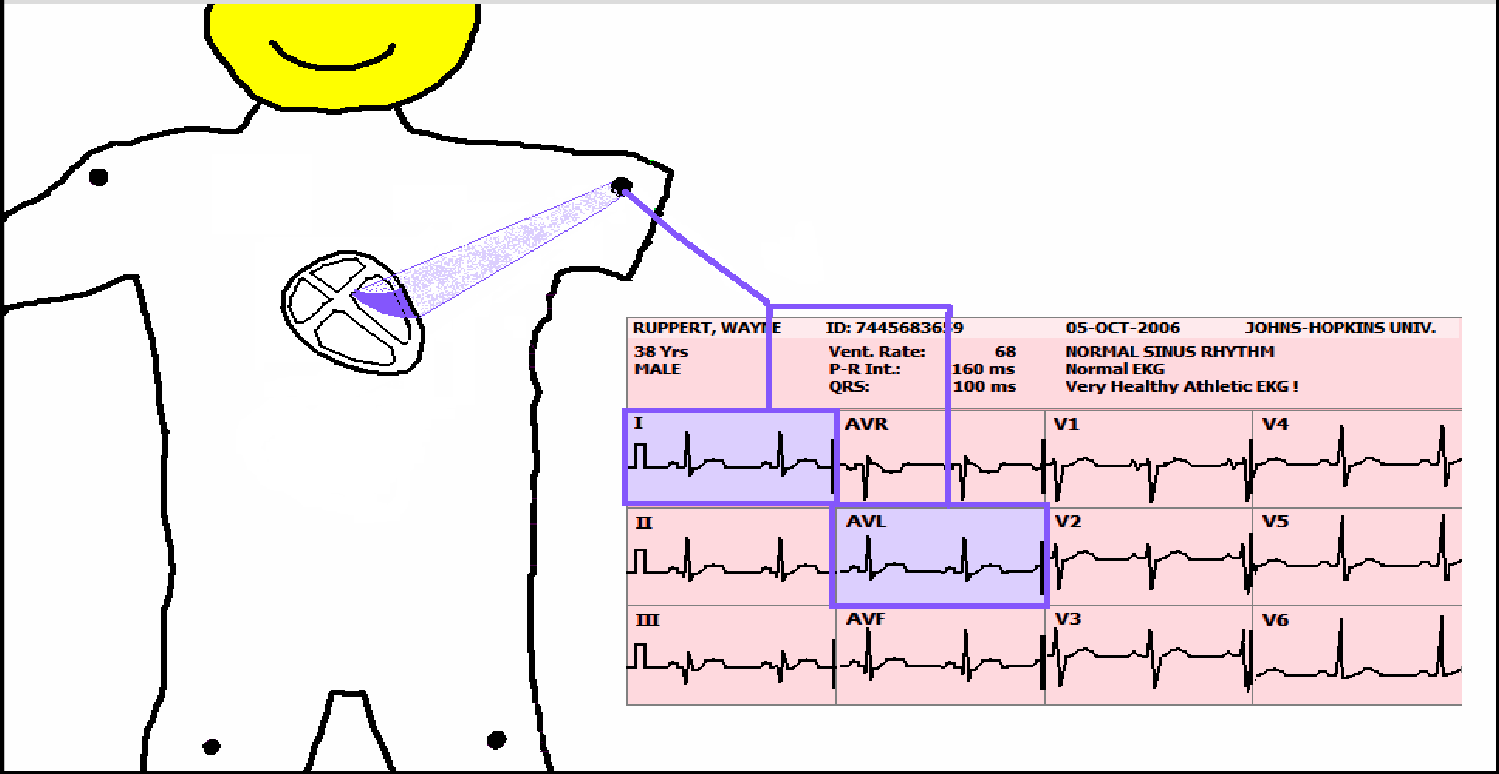
ST SEGMENT ELEVATION

ST SEGMENT DEPRESSION

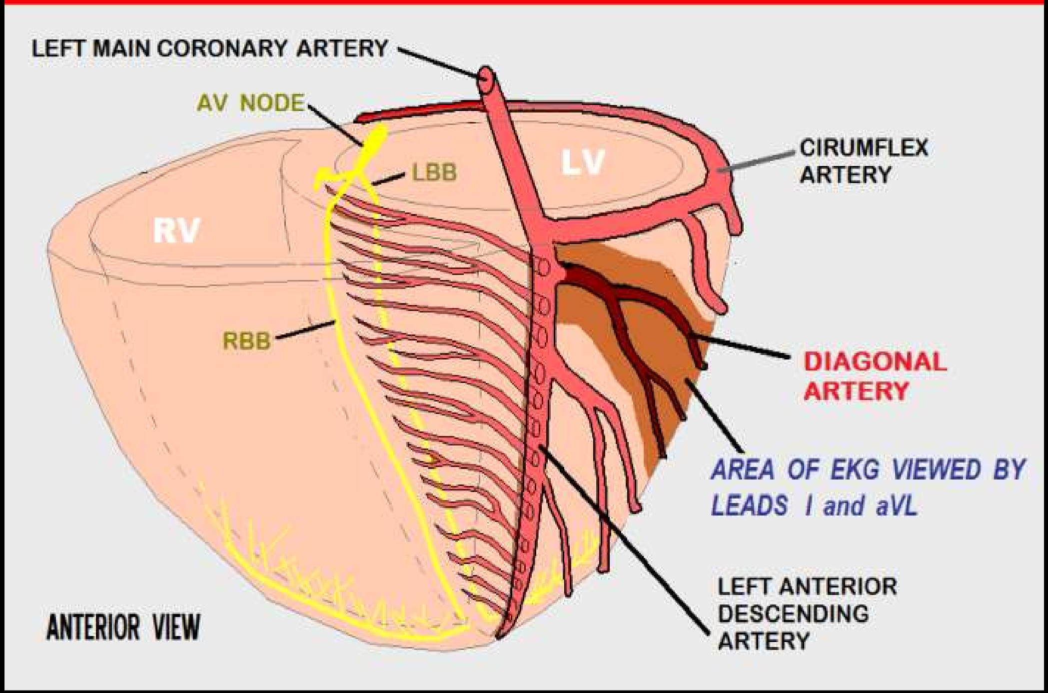




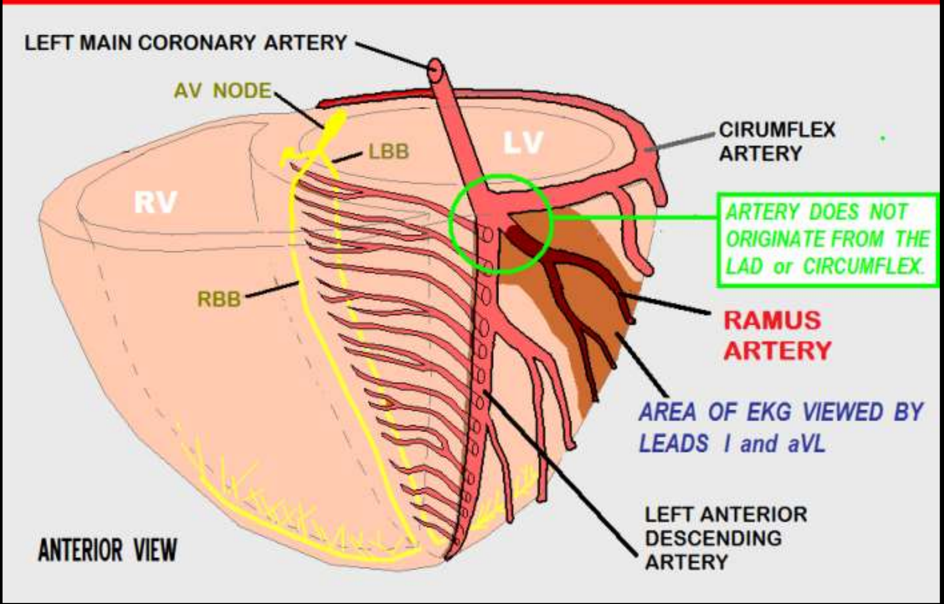
LEADS I and aVL view the ANTERIOR-LATERAL JUNCTION



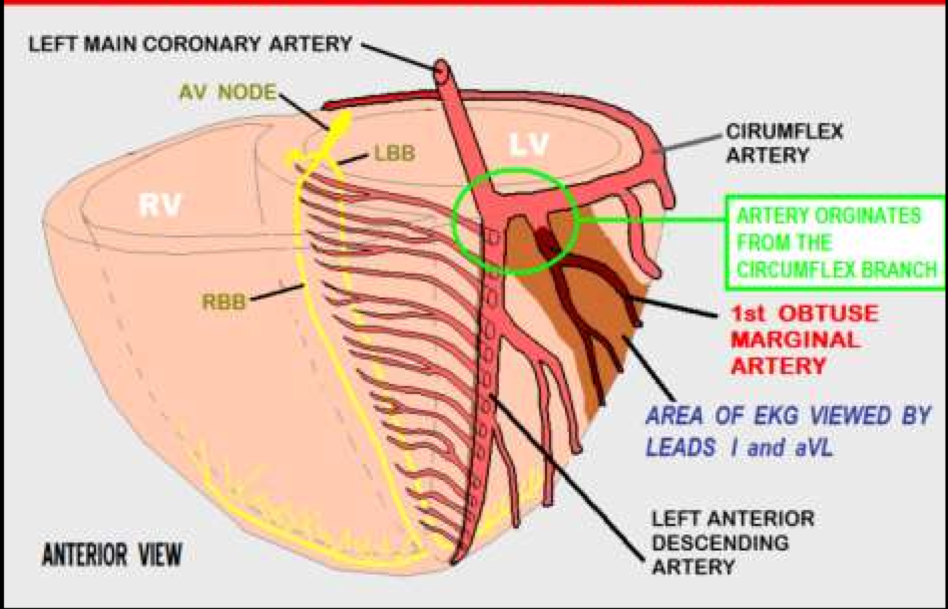
OCCLUSION of DIAGONAL ARTERY



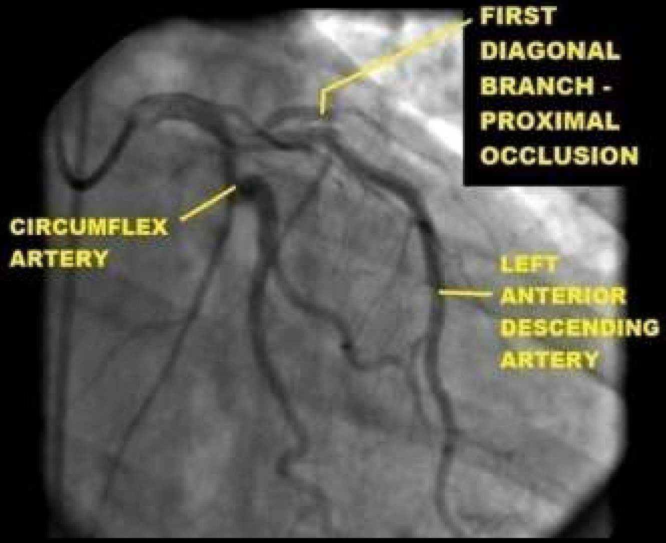
OCCLUSION of RAMUS ARTERY

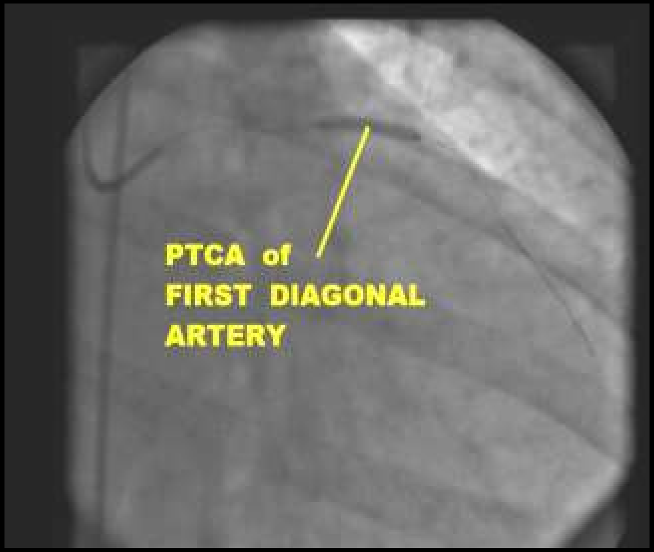


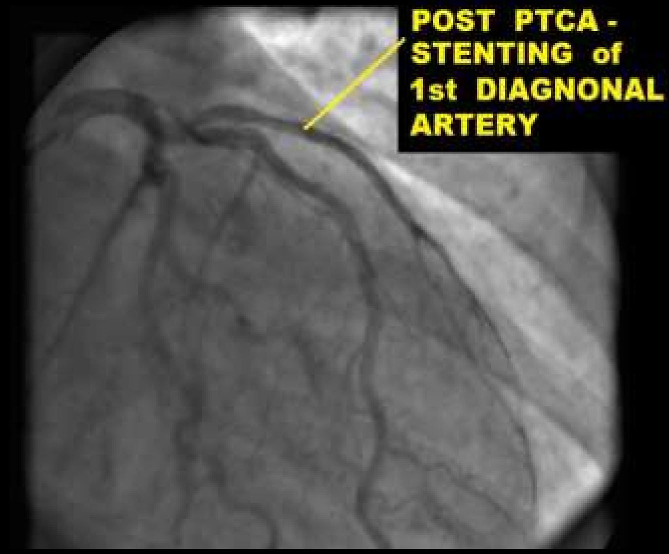
OCCLUSION of OBTUSE MARGINAL ARTERY

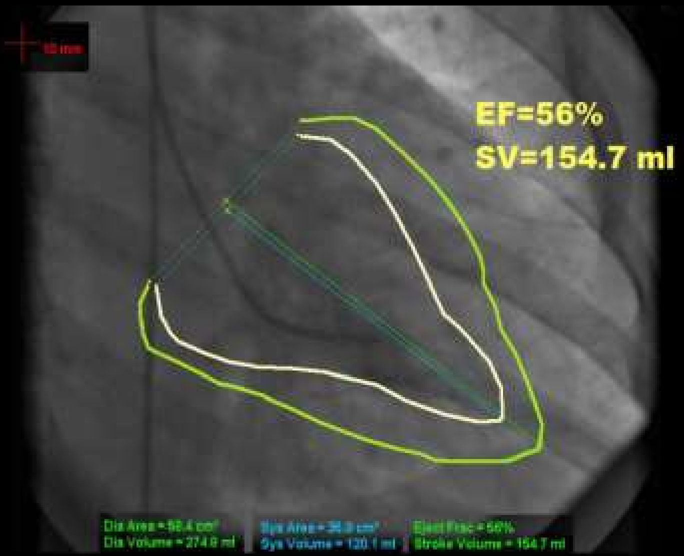


CASE PROGRESSION: As the patient was being prepared for transport to the Cardiac Cath Lab, she experienced an episode of Ventricular Fibrillation.









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%

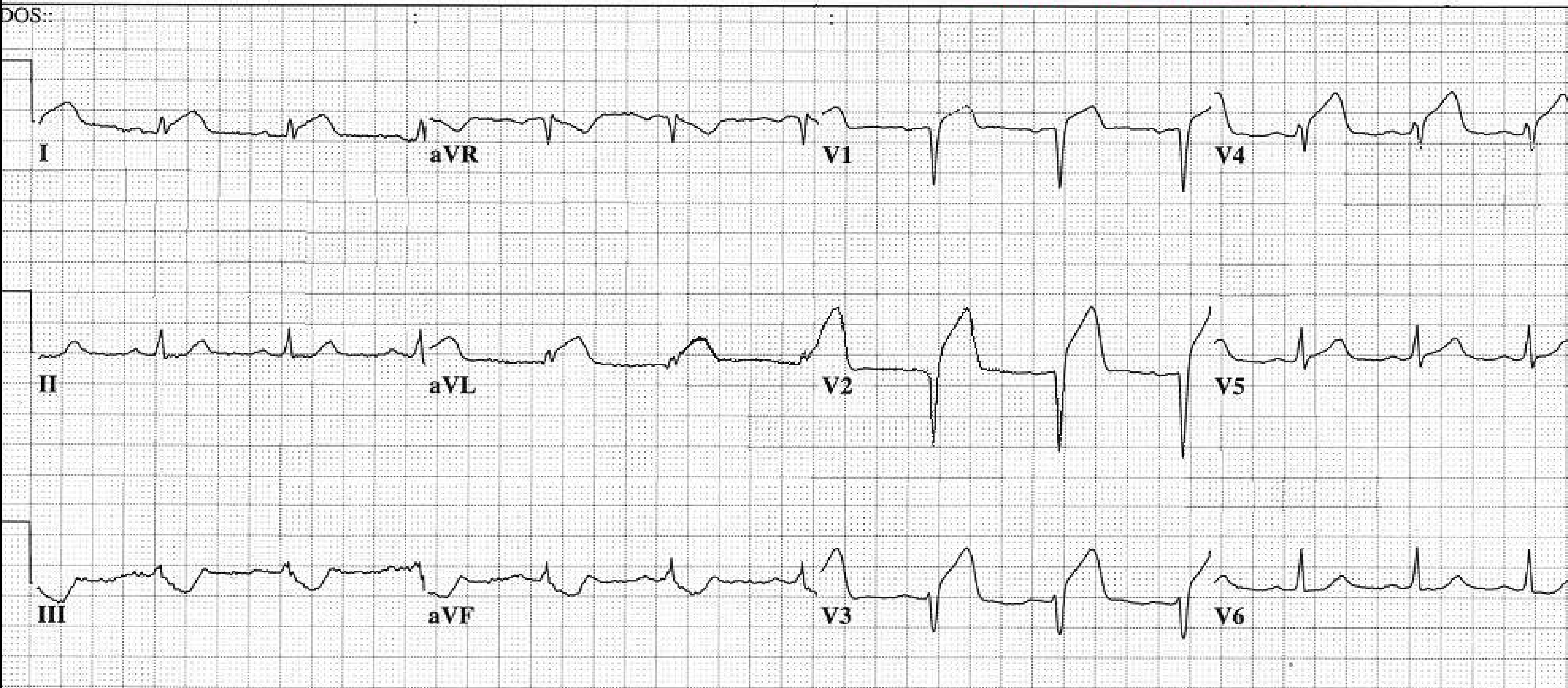
LABS: INITIAL CARDIAC MARKERS - NEGATIVE

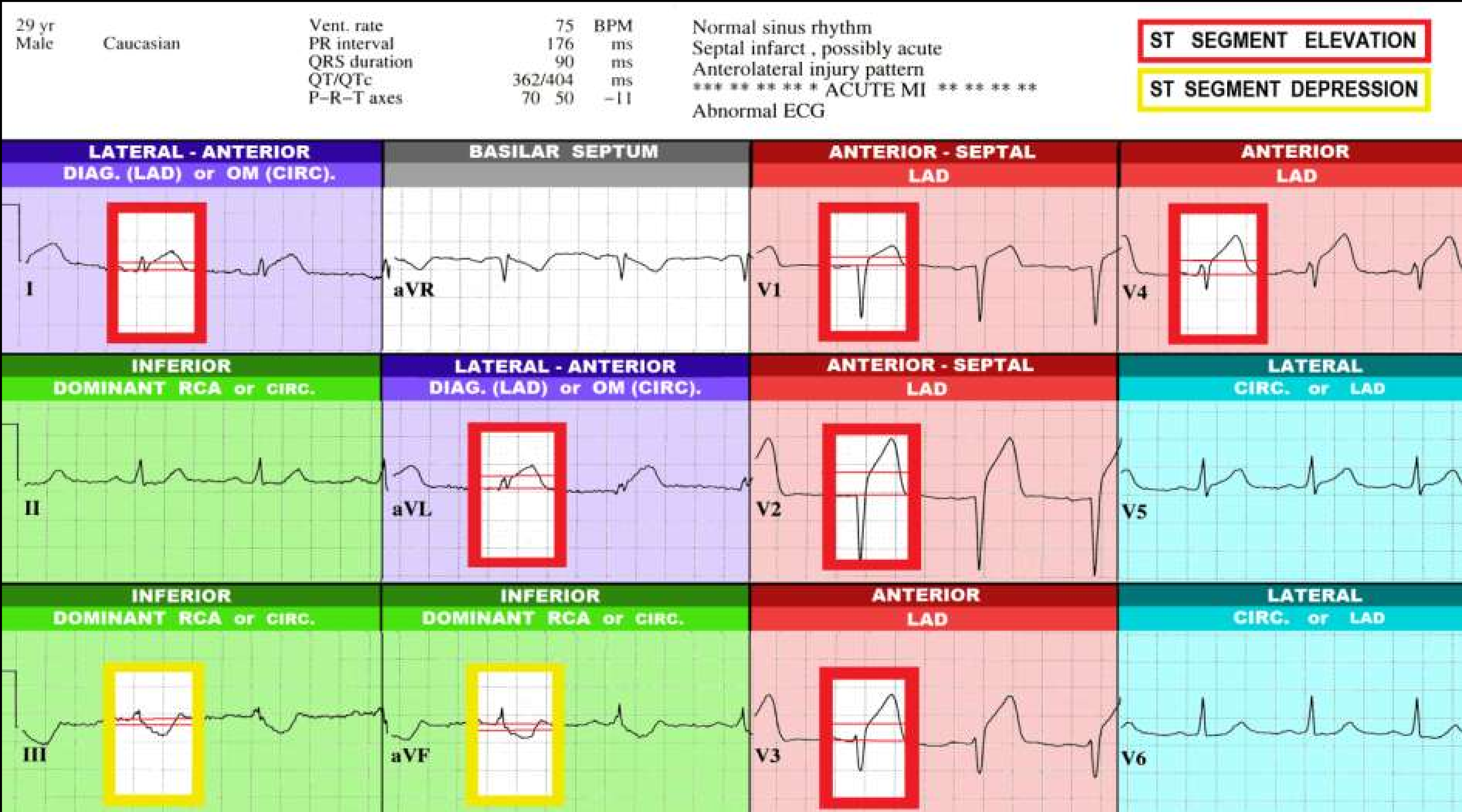
29 yr
Male Caucasian
Loc:3 Option:20

Vent. rate	75		BPM	
PR interval	176		ms	
QRS duration	90		ms	
QT/QTc	362/404		ms	
P–R–T axes	70	50	–11	14:07 Hours

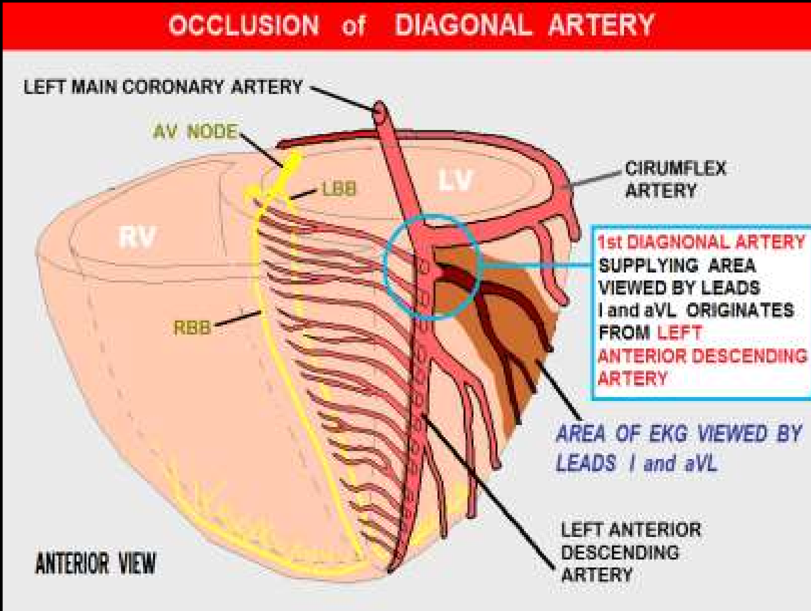
EVALUATE the EKG for signs of ACS:

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

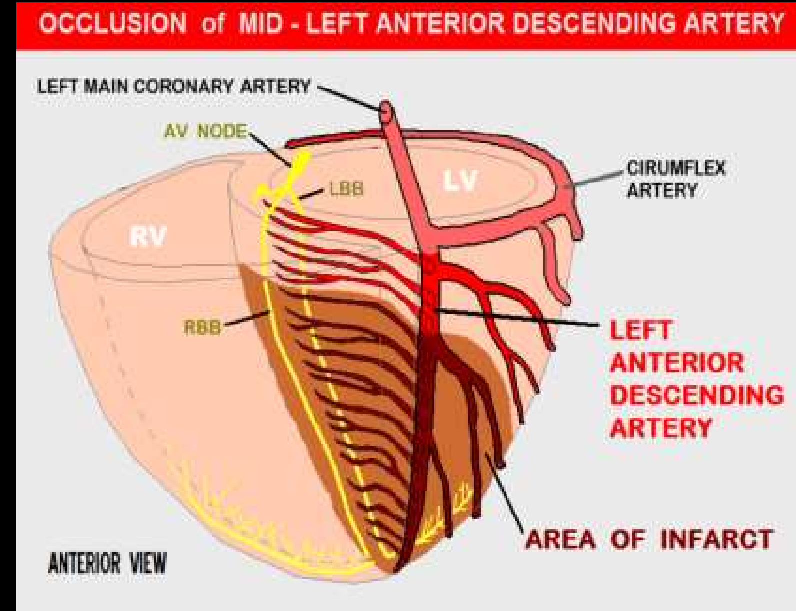



29 yr
Male
Caucasian
Vent. rate 75 BPM
PR interval 176 ms
QRS duration 90 ms
QT/QTc 362/404 ms
P-R-T axes 70 50 -11
Normal sinus rhythm
Septal infarct , possibly acute
Anterolateral injury pattern
*** ** ** ** * ACUTE MI ** ** ** **
Abnormal ECG
ST SEGMENT ELEVATION
ST SEGMENT DEPRESSION
LATERAL - ANTERIOR
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

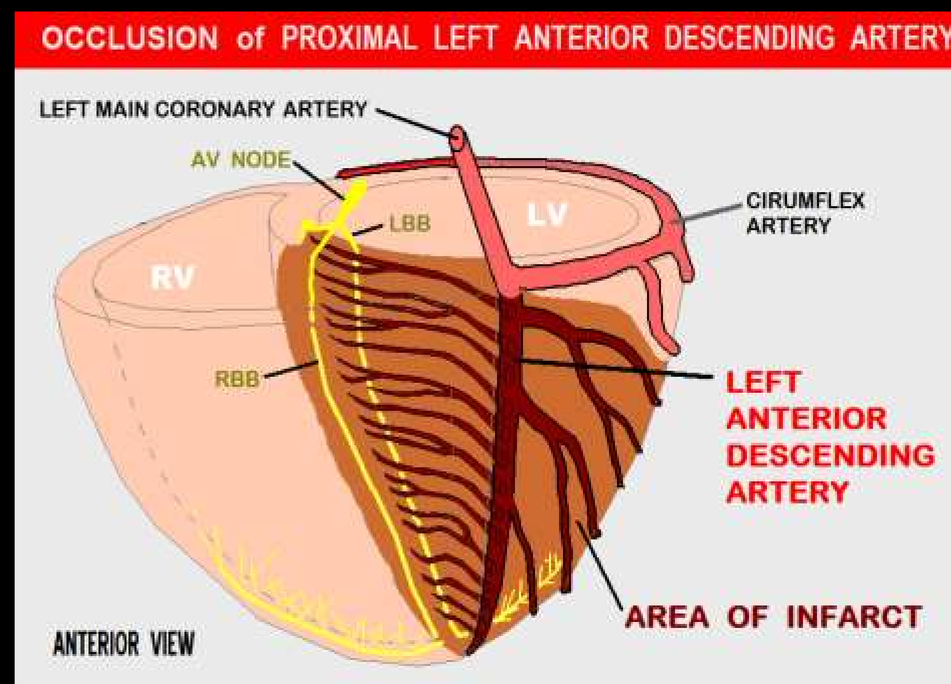
- **Reciprocal ST Depression is NOW PRESENT**
- **Additional ST Elevation is present in Leads I, AVL**

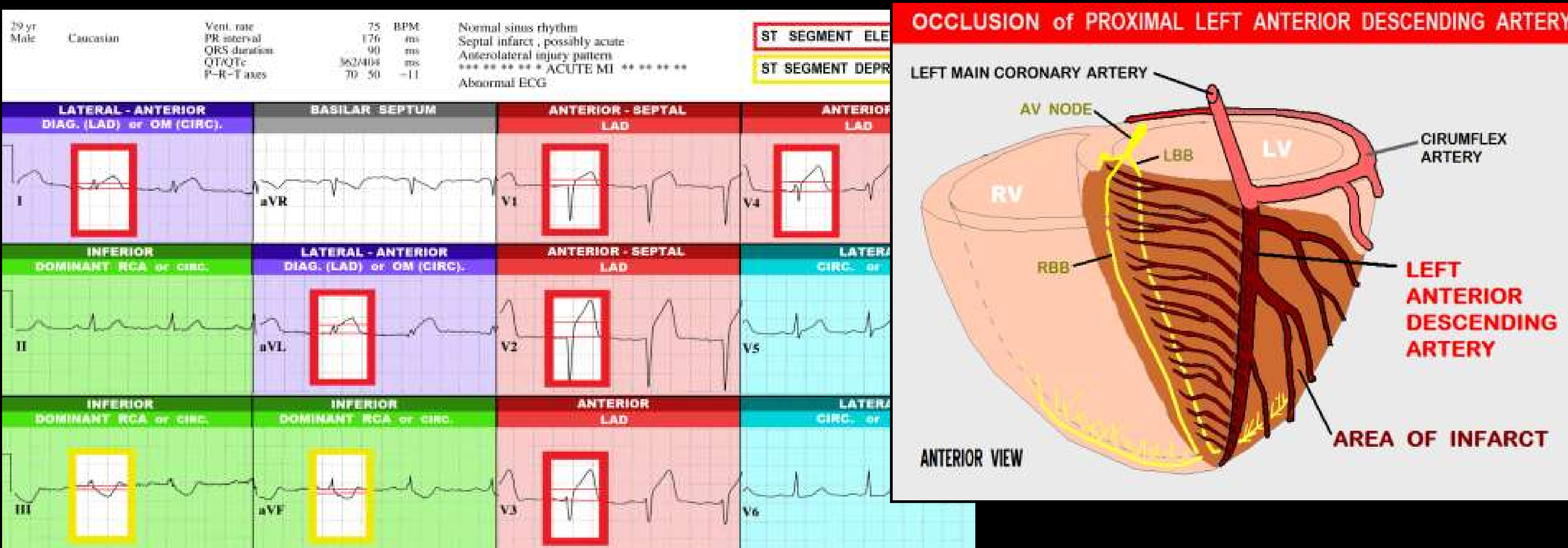

OCCLUSION of DIAGONAL ARTERY
LEFT MAIN CORONARY ARTERY
AV NODE
LBB
LV
RV
CIRUMFLEX ARTERY
1st DIAGNONAL ARTERY SUPPLYING AREA VIEWED BY LEADS I and aVL ORIGINATES FROM LEFT ANTERIOR DESCENDING ARTERY
RBB
AREA OF EKG VIEWED BY LEADS I and aVL
LEFT ANTERIOR DESCENDING ARTERY
ANTERIOR VIEW

+


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

=


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



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 ELE
ST SEGMENT DEPR
LATERAL - ANTERIOR
DIAG. (LAD) or OM (CIRC).
BASILAR SEPTUM
ANTERIOR - SEPTAL
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ANTERIOR
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LATERA
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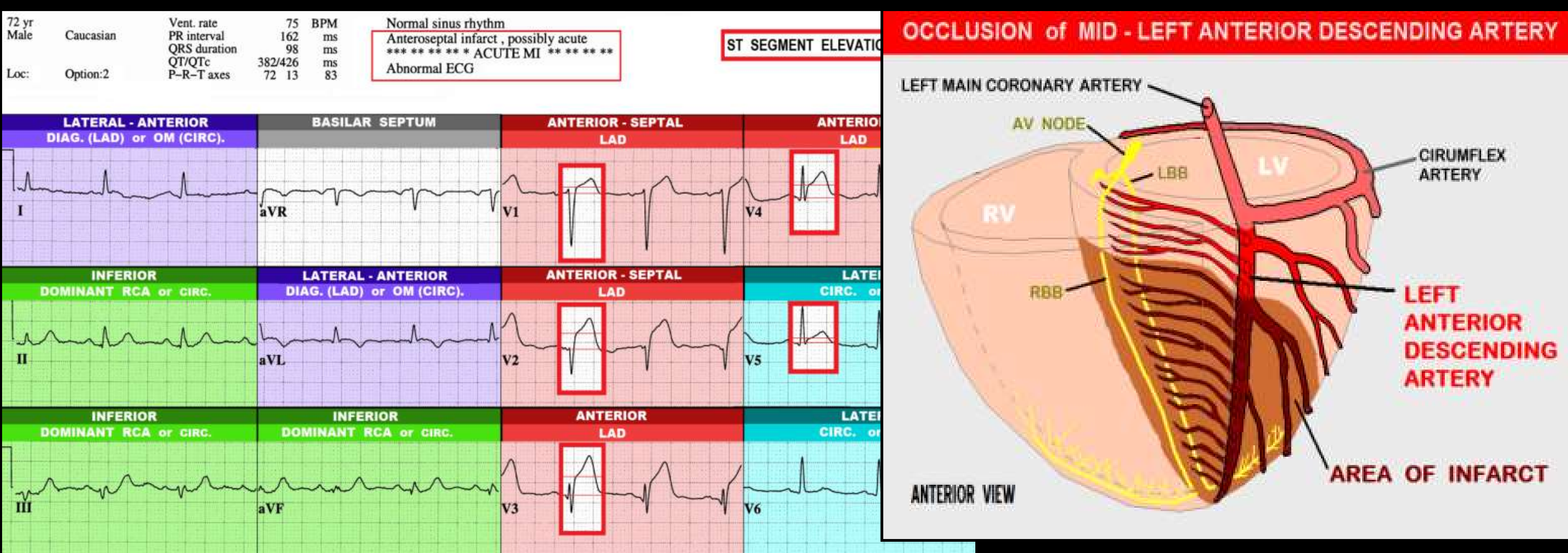
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
OCCLUSION of PROXIMAL LEFT ANTERIOR DESCENDING ARTERY
LEFT MAIN CORONARY ARTERY
AV NODE
LBB
LV
CIRUMFLEX ARTERY
RV
RBB
LEFT ANTERIOR DESCENDING ARTERY
AREA OF INFARCT
ANTERIOR VIEW



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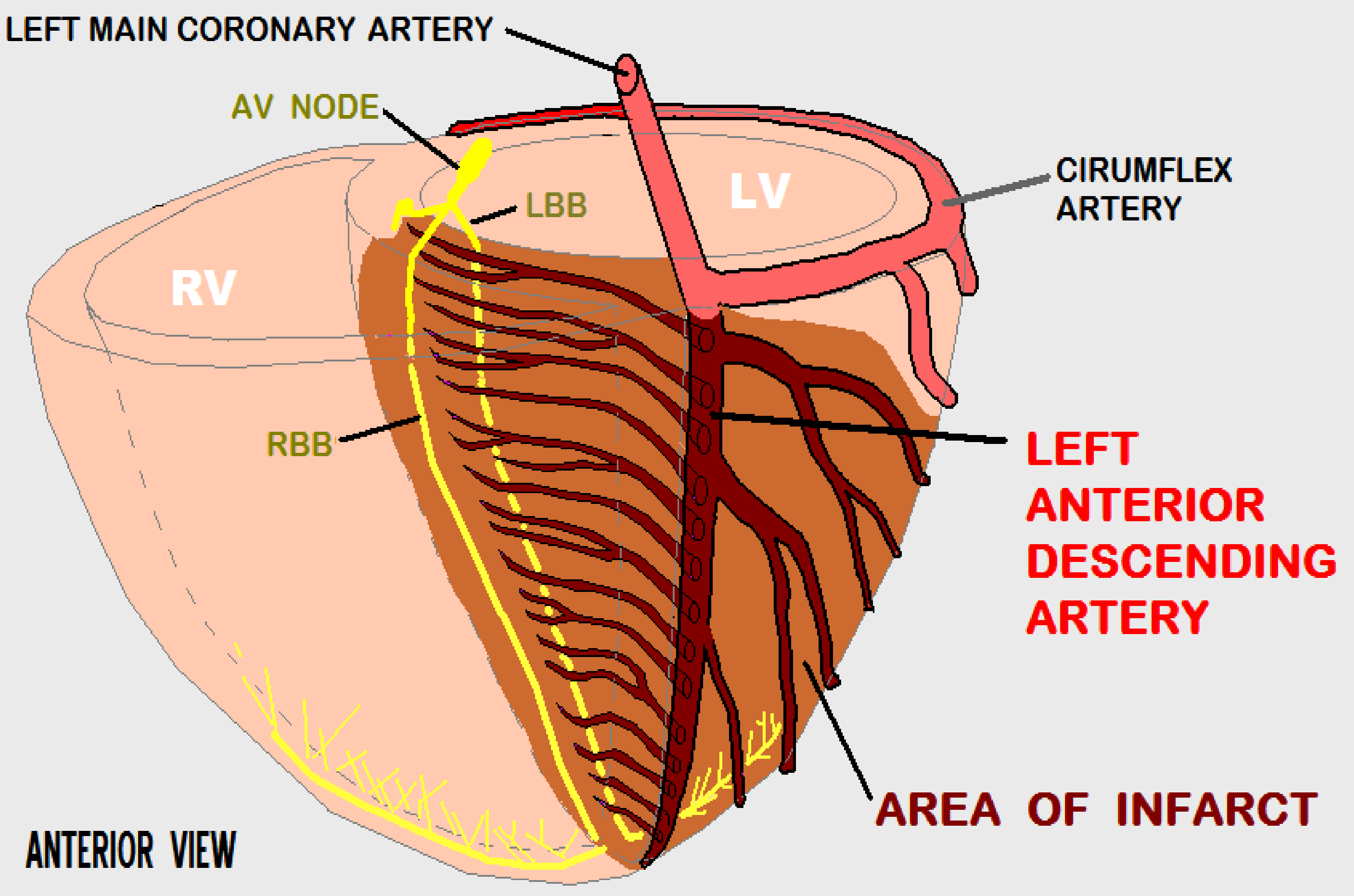
CIRUMFLEX ARTERY
RV
RBB
LEFT ANTERIOR DESCENDING ARTERY
AREA OF INFARCT
ANTERIOR VIEW

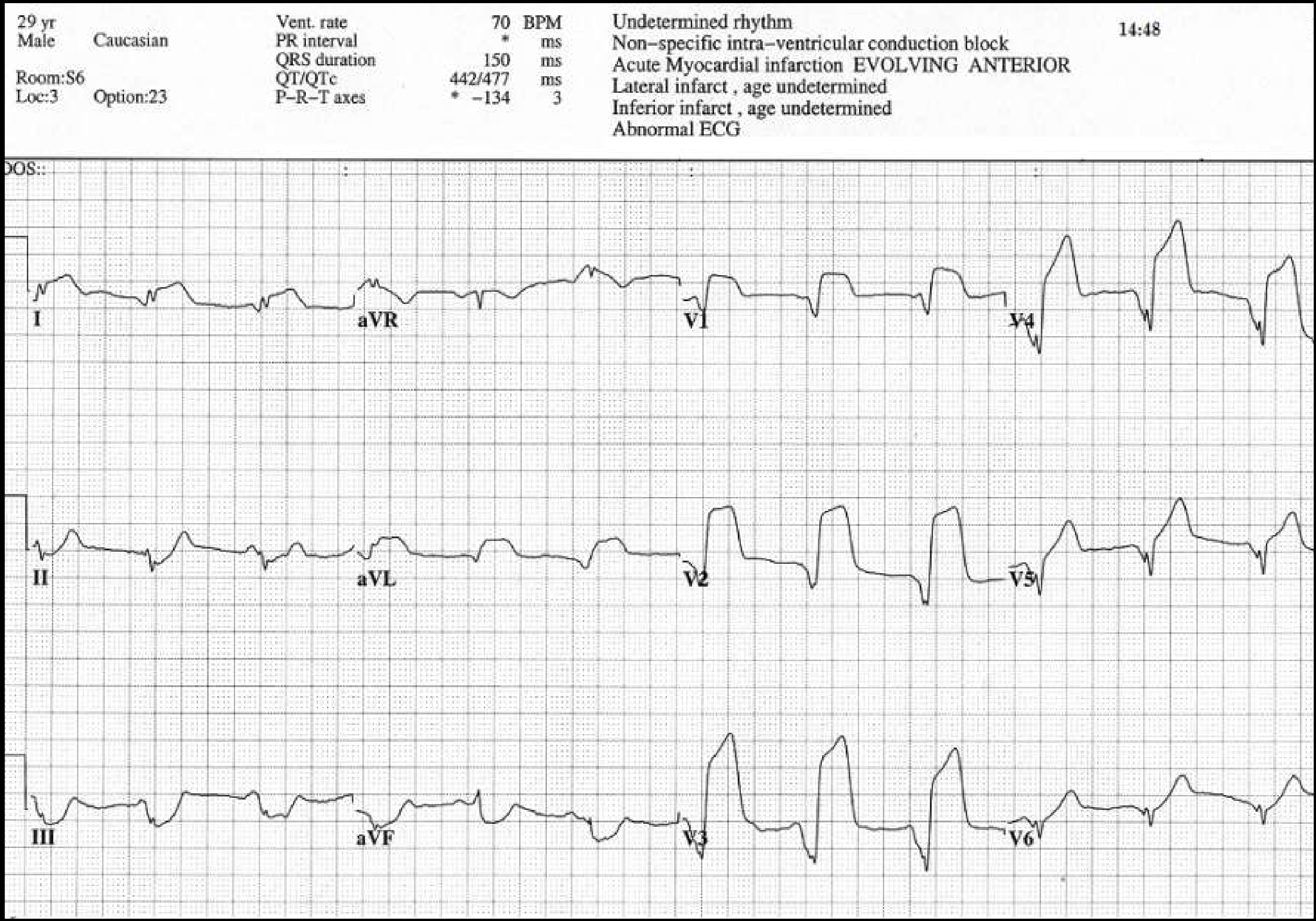
OCCLUSION of PROXIMAL LEFT ANTERIOR DESCENDING ARTERY

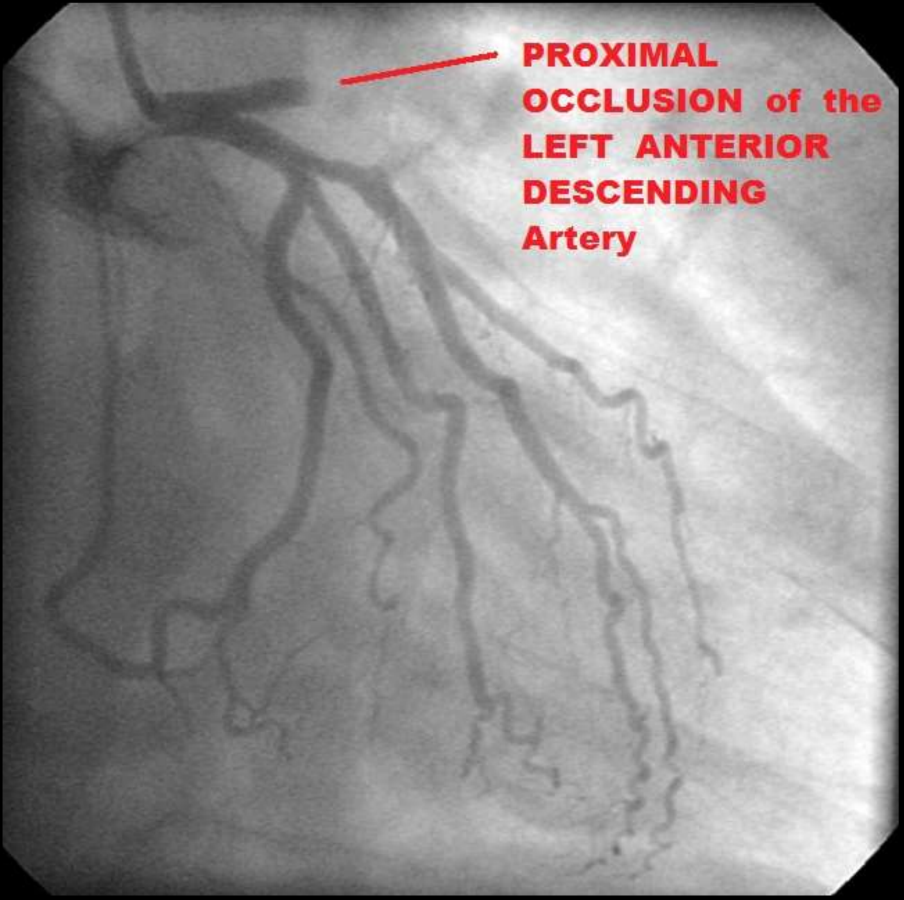


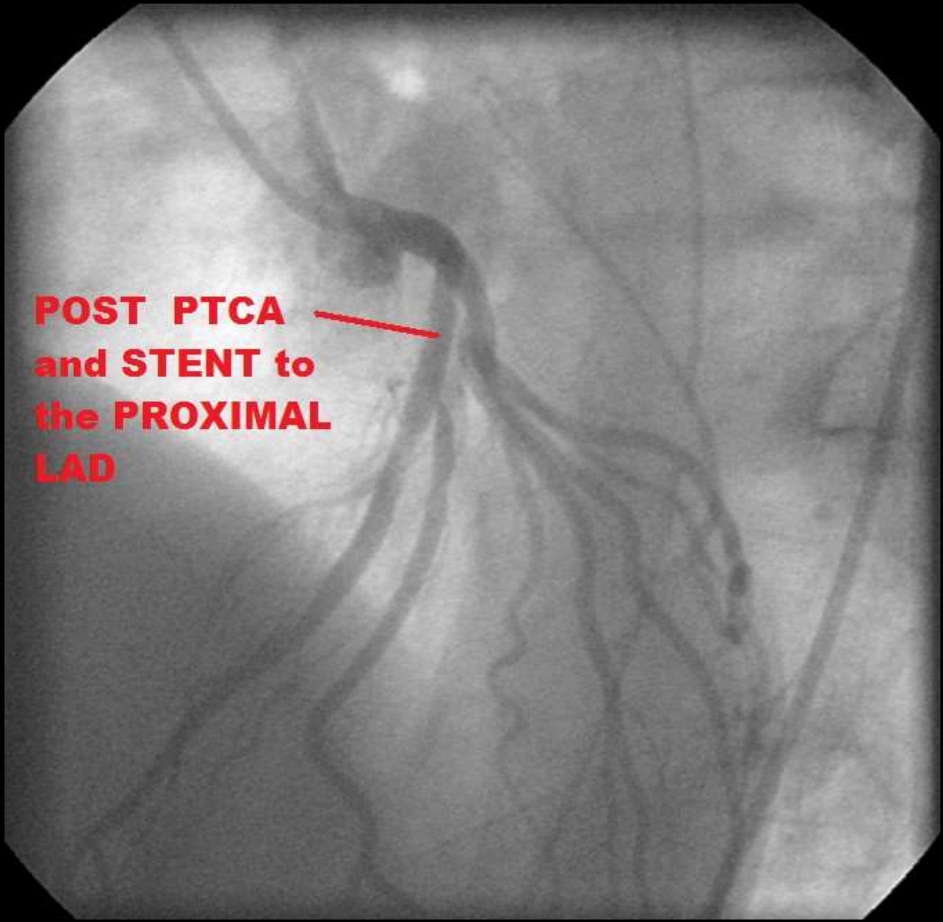
ANTICIPATED COMPLICATIONS of ANTERIOR-SEPTAL WALL STEMI & POSSIBLE INDICATED INTERVENTIONS:	
- CARDIAC ARREST	BCLS / ACLS
- CARDIAC DYSRHYTHMIAS (VT / VF)	ACLS (antiarrhythmics)
- PUMP FAILURE with CARDIOGENIC SHOCK	INOTROPE THERAPY: -DOPAMINE / DOBUTAMINE / LEVOPHED - INTRA-AORTIC BALLOON PUMP (use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

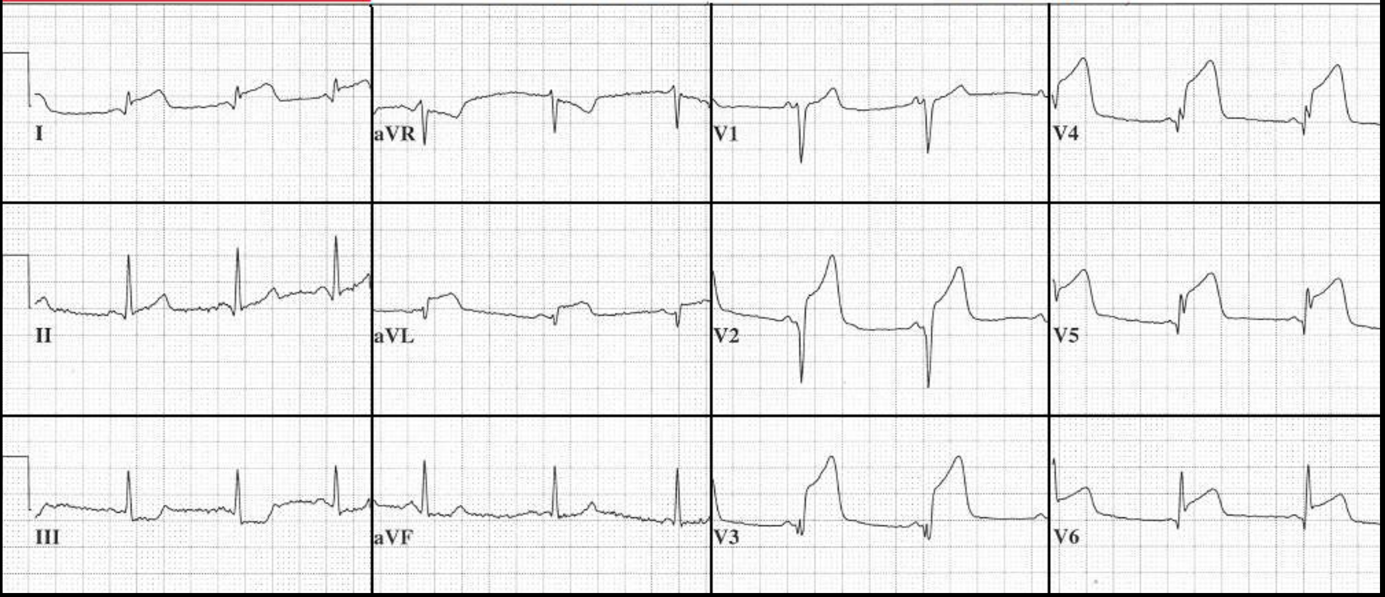
CASE STUDY 4: CRITICAL DECISIONS SCENARIO

As per current AHA recommendations, your hospital's policy is to send every STEMI patient to the Cardiac Catheterization Lab for emergency PCI.

You are the ranking medical officer on duty in the ED when two acute STEMI patients arrive, ten minutes apart. The Cath Lab has one lab open, and can take ONE patient immediately. Both patients duration of symptoms and state of hemodynamic stability are similar.

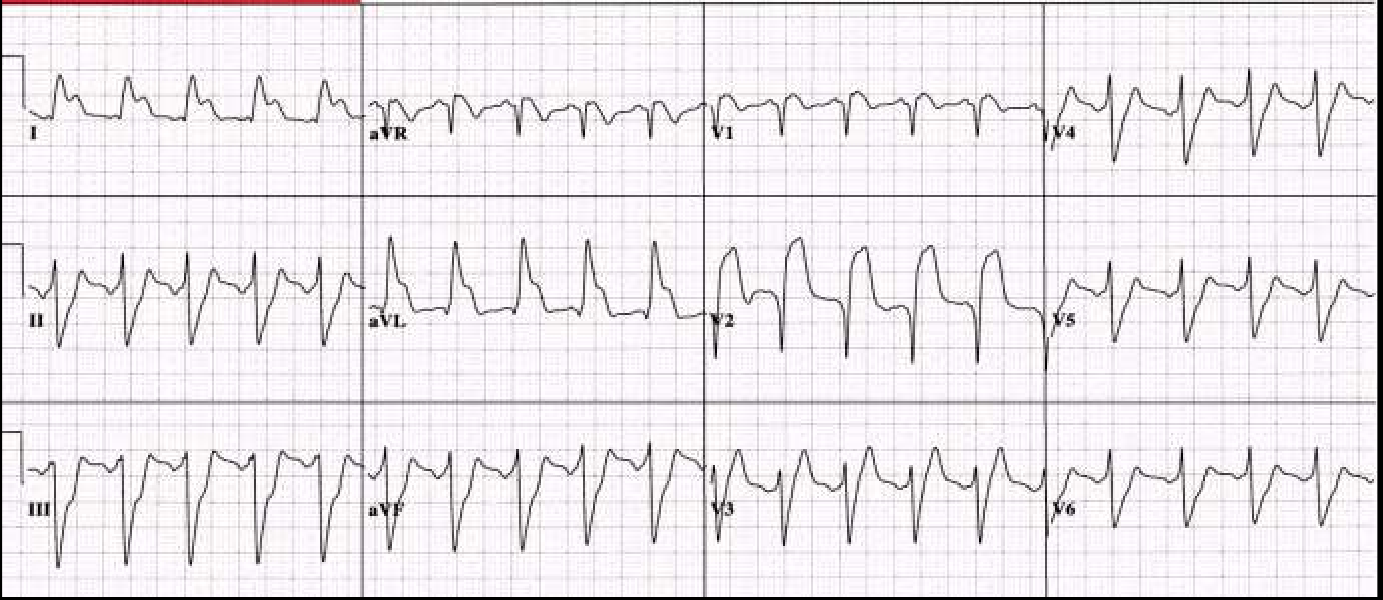
PATIENT A:

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

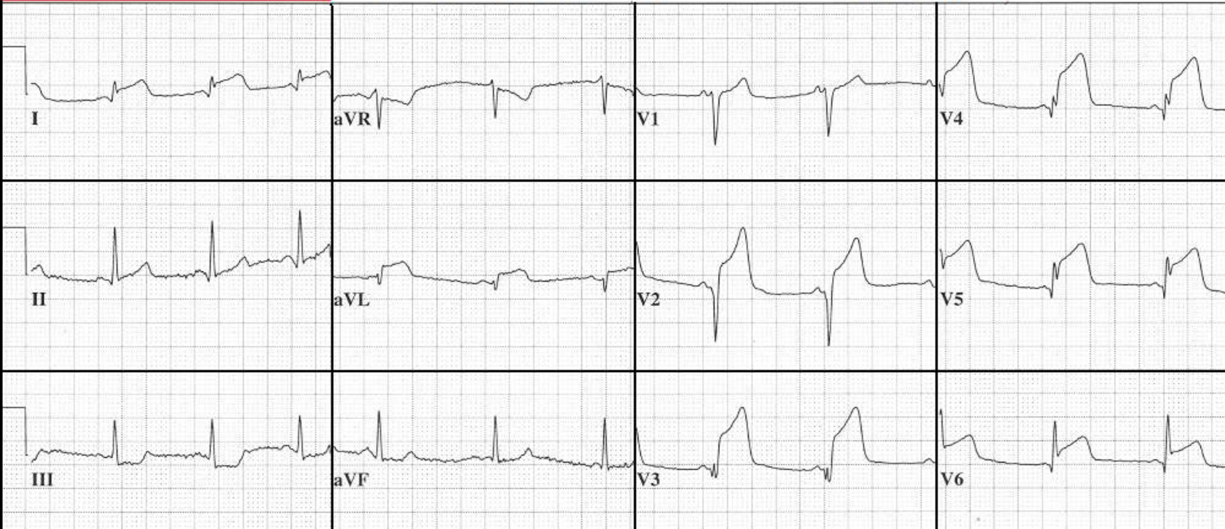


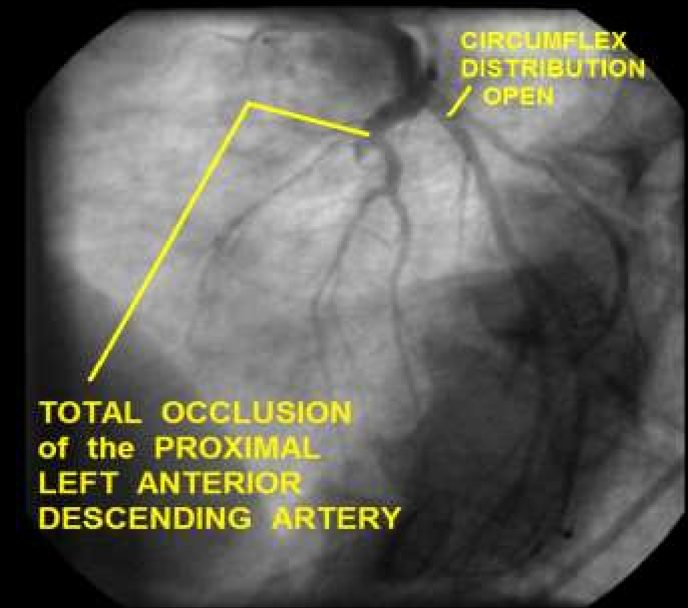
WHO SHOULD GO TO THE CATH LAB FIRST ?

And . . . .

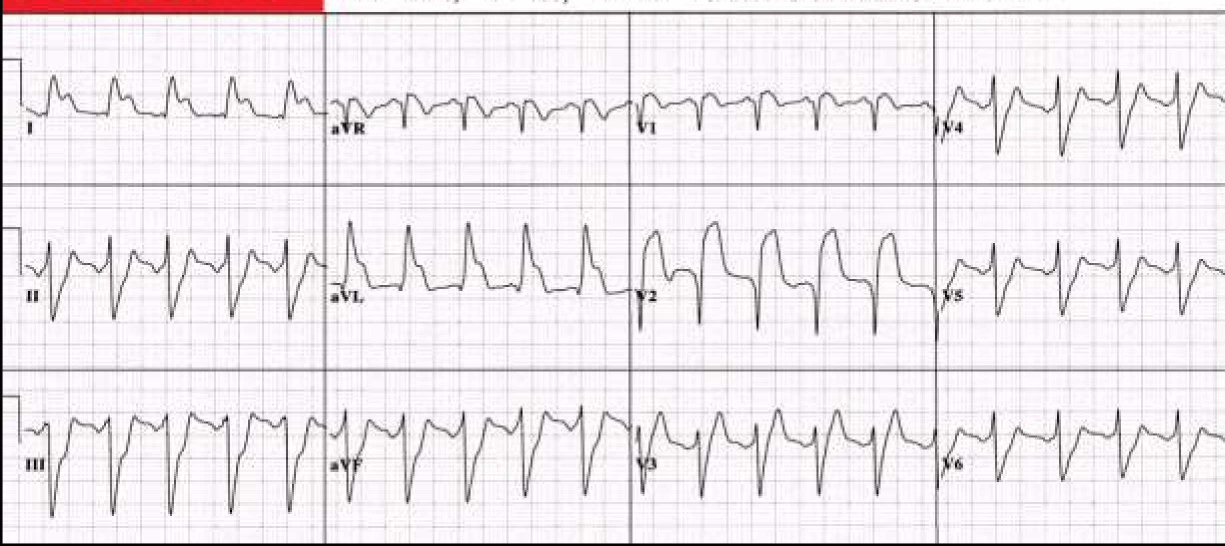
WHAT WOULD YOU DO WITH THE PATIENT WHO DID NOT GO TO THE CATH LAB ?

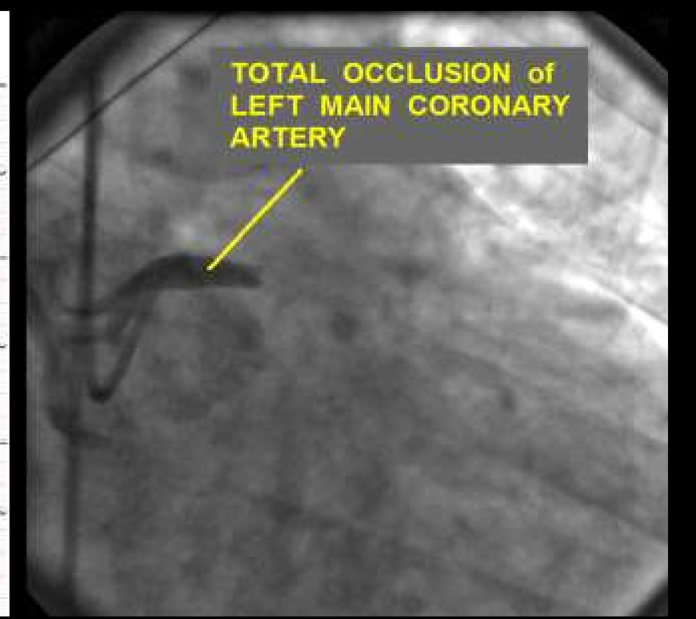
PATIENT A: 44 y/o MALE, CHEST PAIN x 1 HOUR,
BP: 78/46, P: 70, R: 28. CARDIAC MARKERS: NEGATIVE



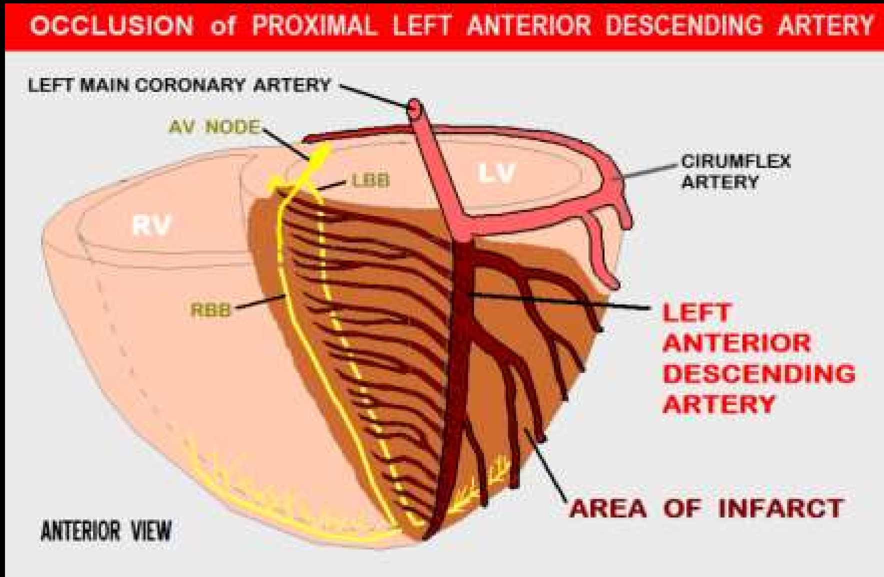


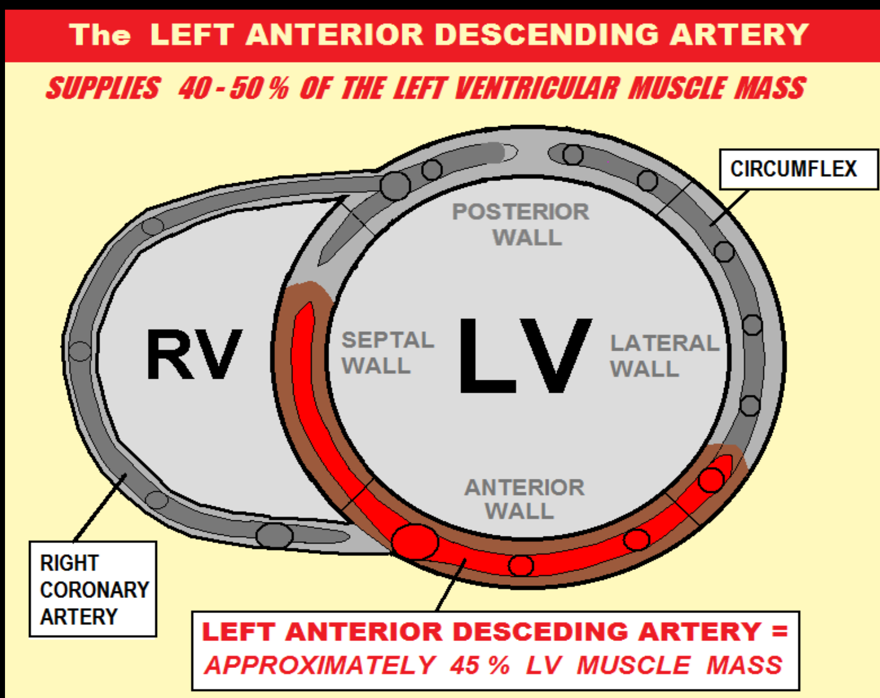
PATIENT B: 36 y/o MALE, CHEST PAIN x 1 HOUR,
BP: 80/48, P: 120, R: 28 CARDIAC MARKERS: NEGATIVE



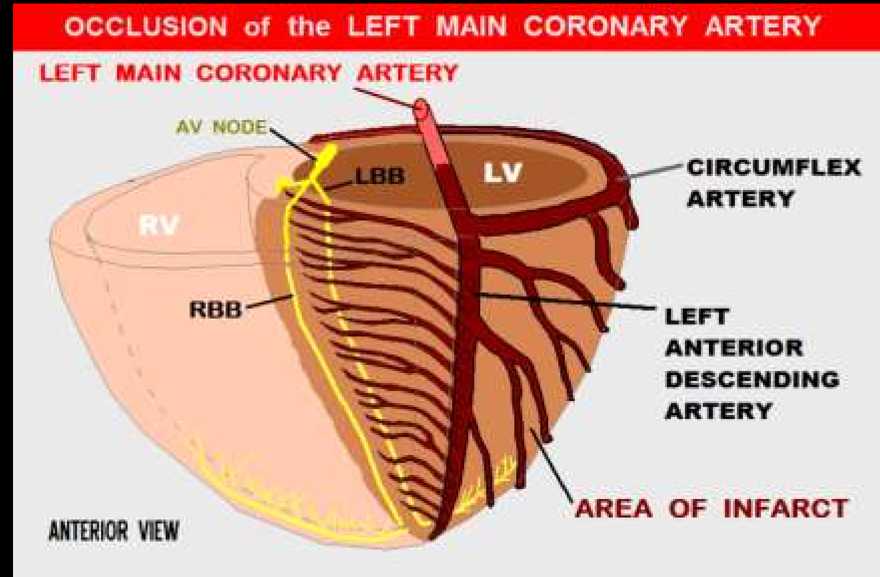


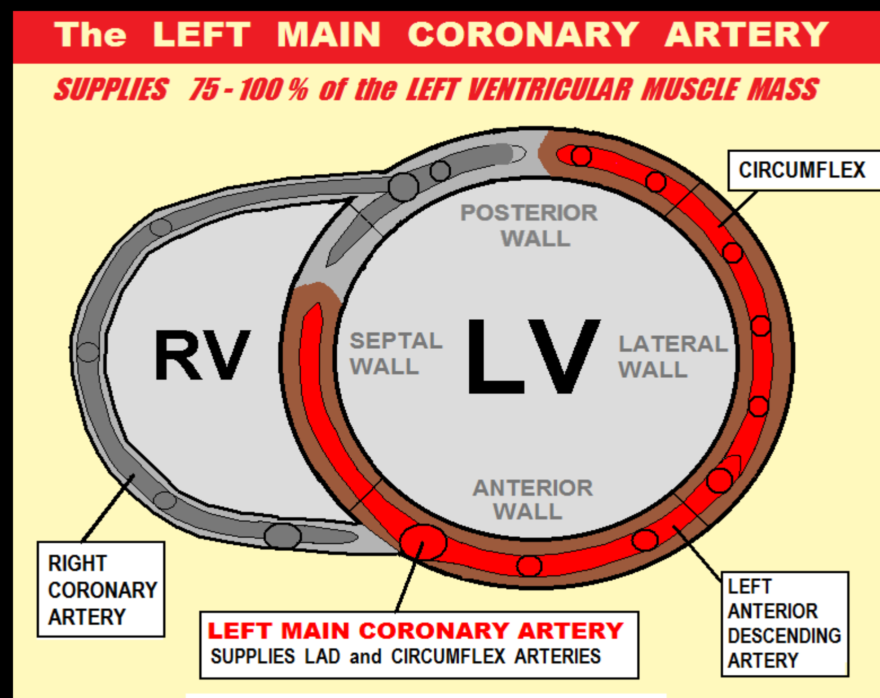
PATIENT A:





PATIENT B:



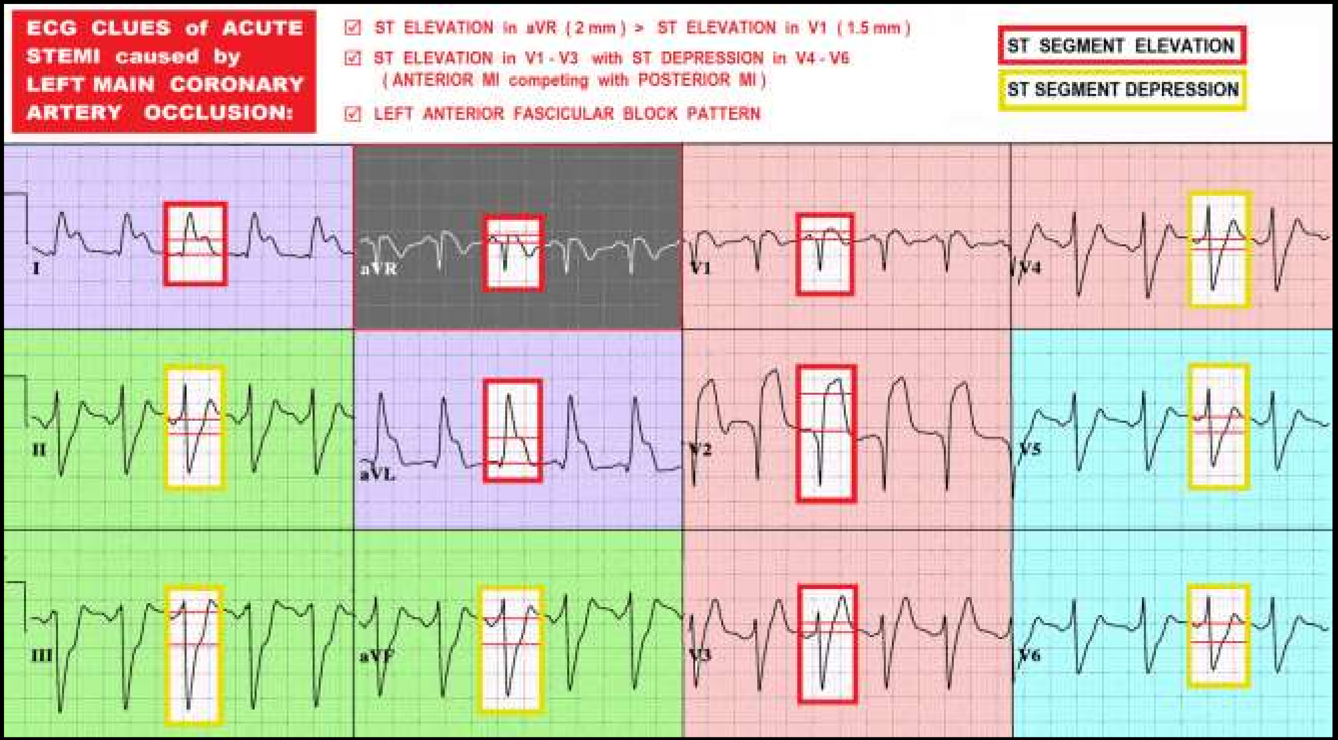


ECG Clues . . . for IDENTIFYING STEMI CAUSED BY LEFT MAIN CORONARY ARTERY occlusion:

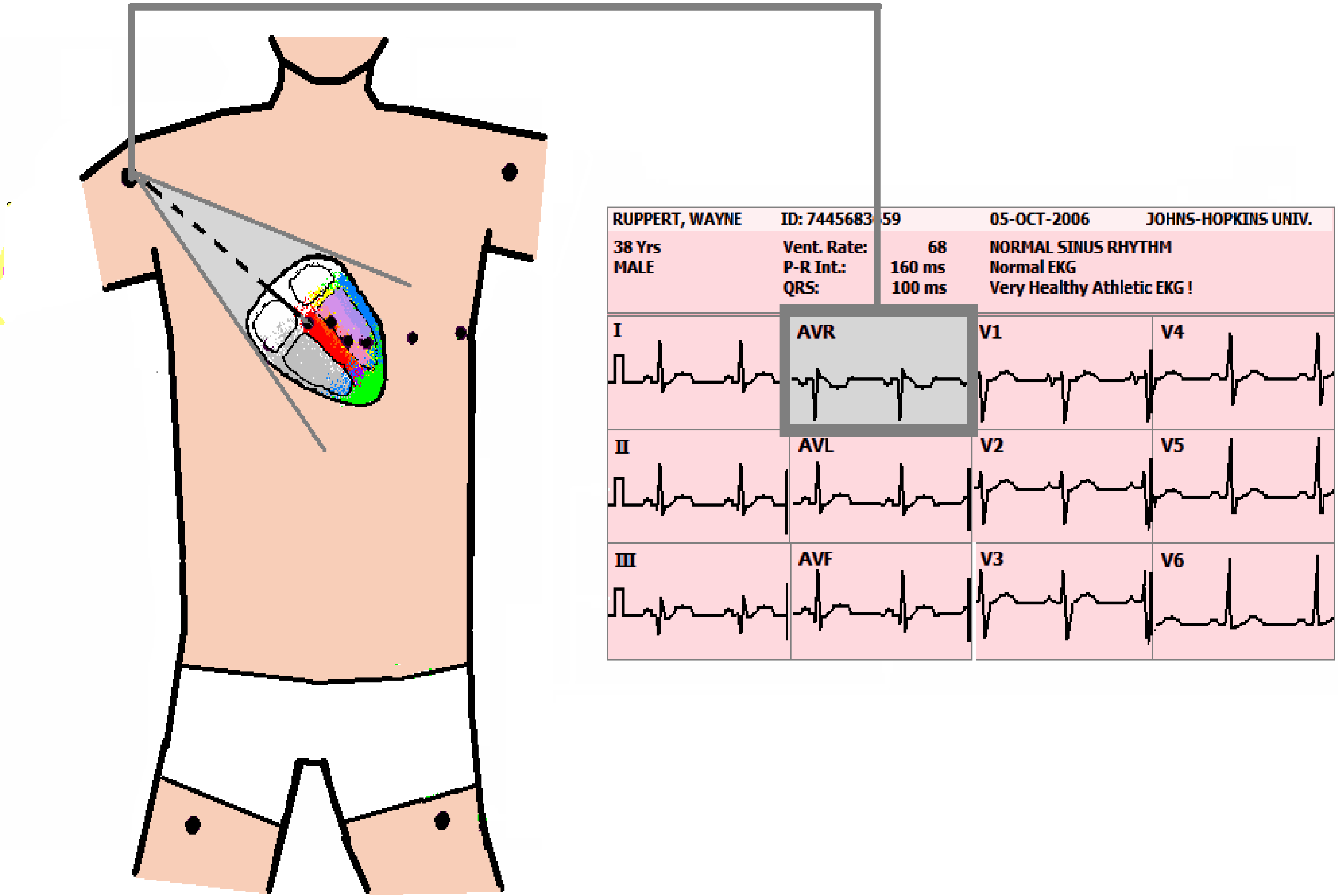
- ☑ ST ELEVATION in ANTERIOR LEADS (V1 - V4) and LATERAL LEADS (V5 & V6)
- ☑ ST DEPRESSION or ISOELCTRIC J POINTS may be seen in V LEADS . . . . mainly V2 and/or V3 *caused by COMPETING FORCES of ANTERIOR vs. POSTERIOR WALL MI.* *+
  - → NOTE: it is very unusual to see ST DEPRESSION in V LEADS with isolated 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

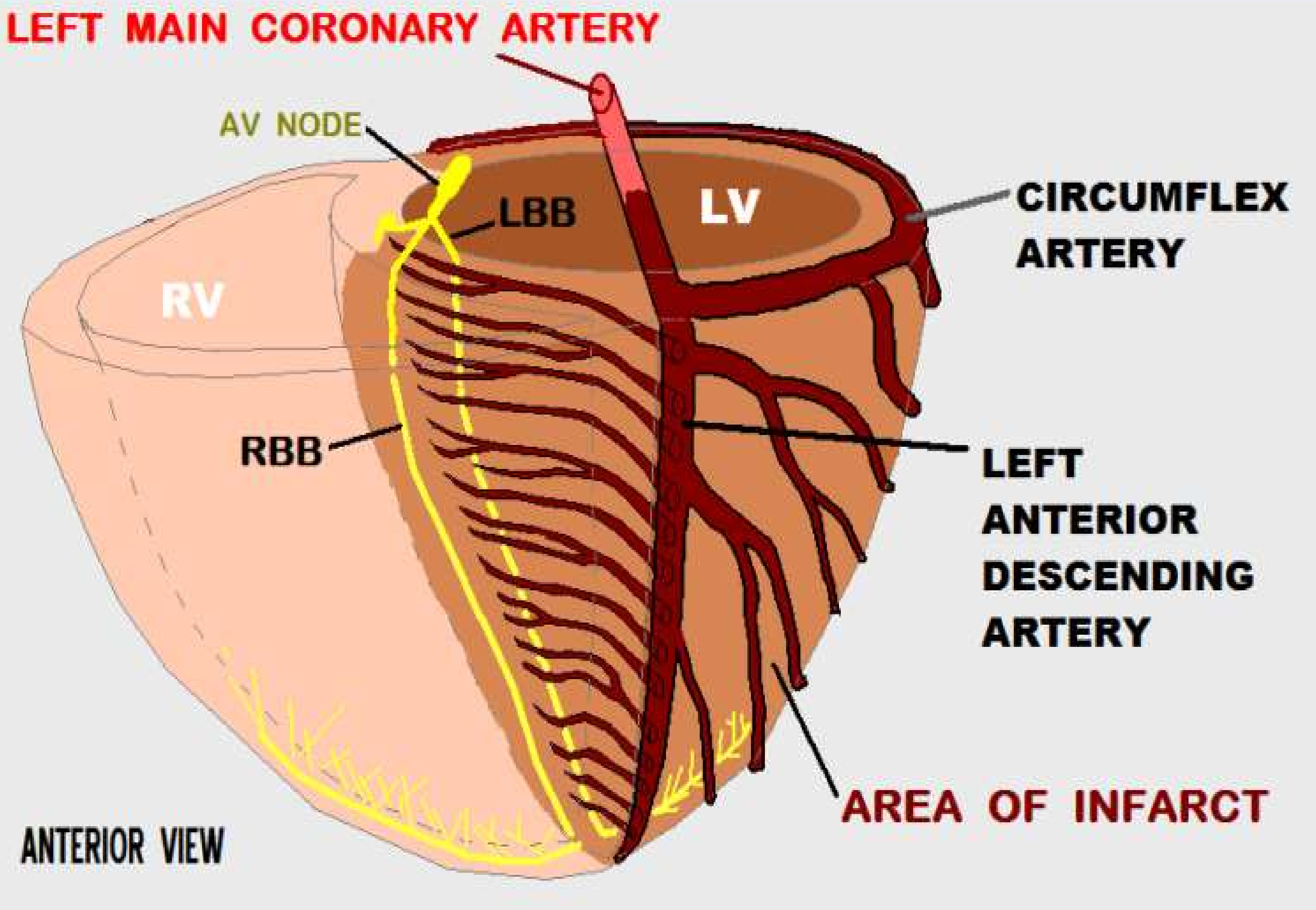


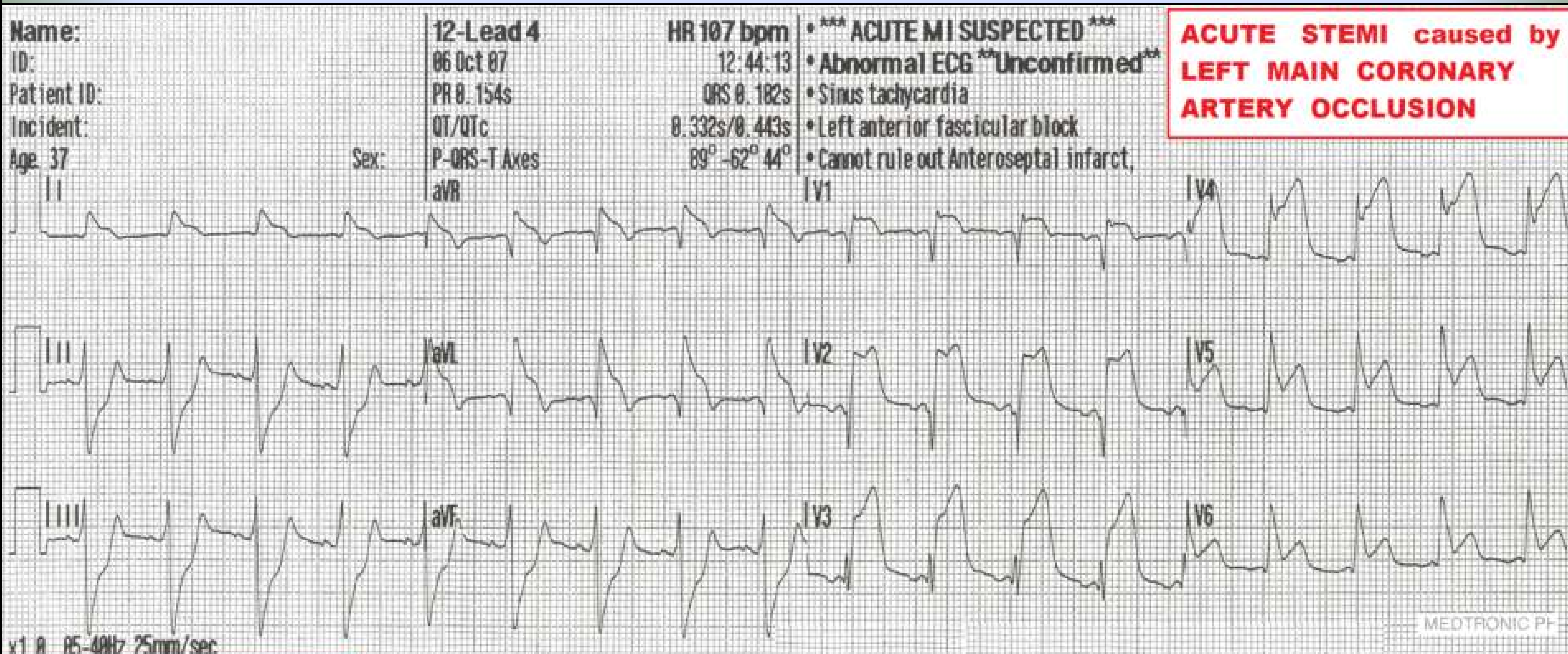
Lead AVR Views the BASILAR SEPTUM (region of the Bundle of His)



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

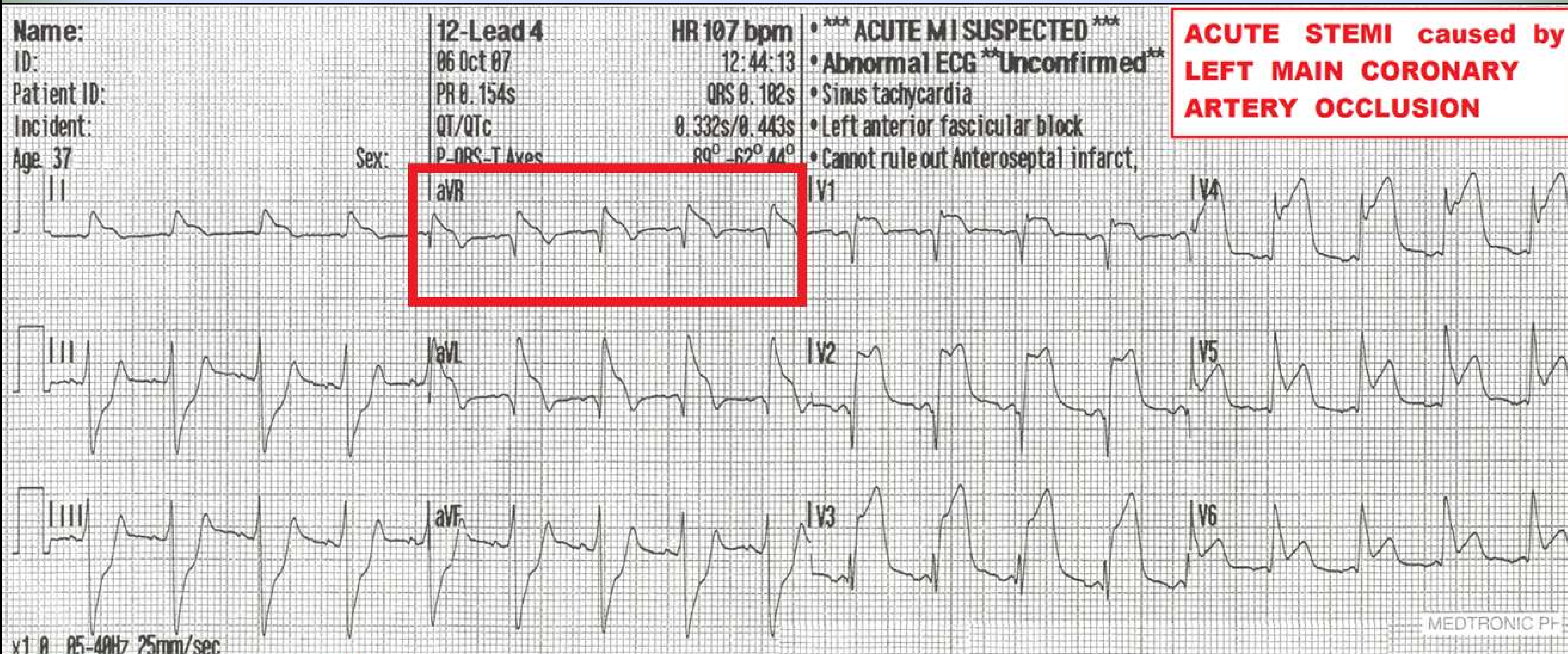




ECG CLUES of ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION:

- ☑ 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
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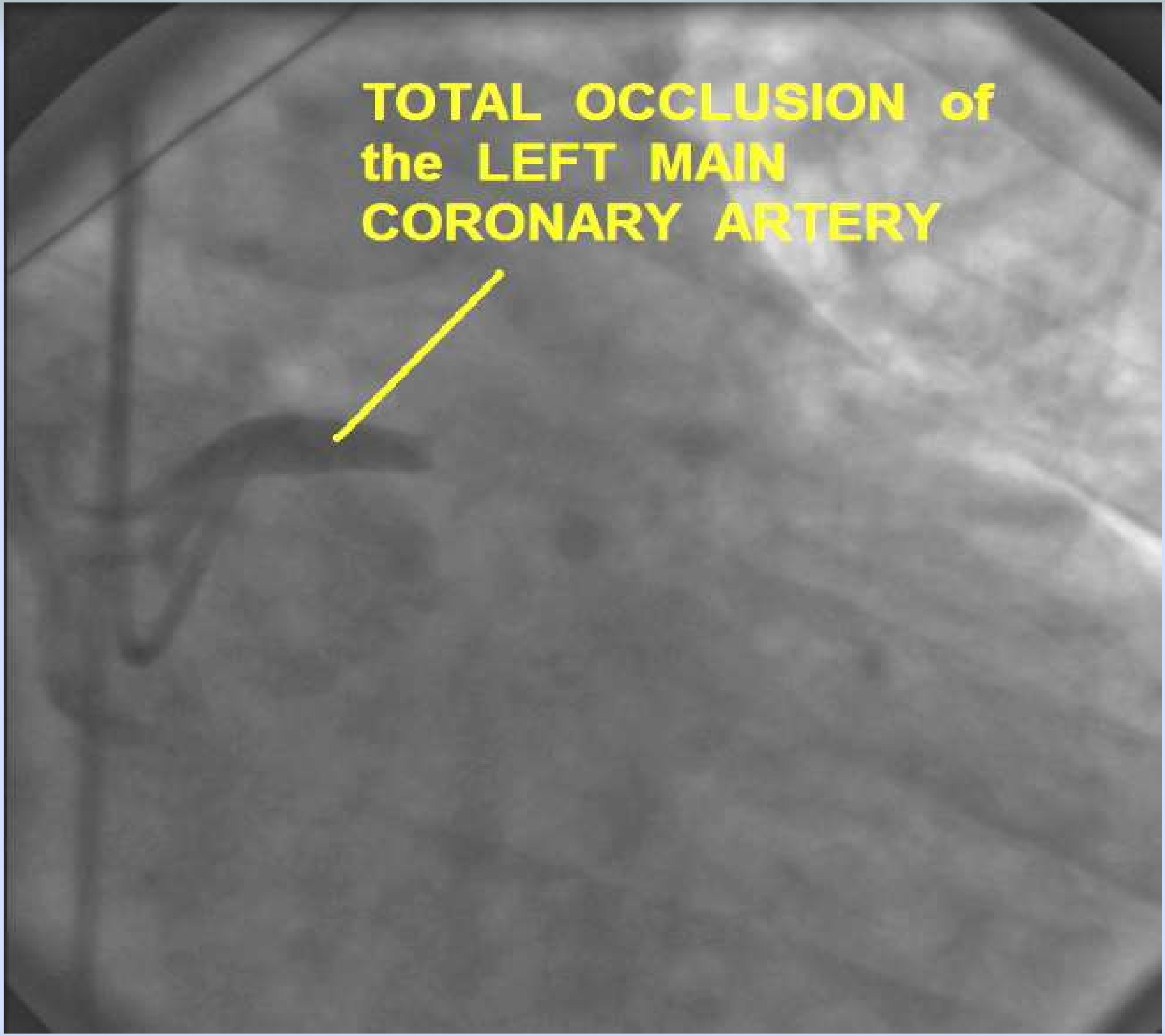
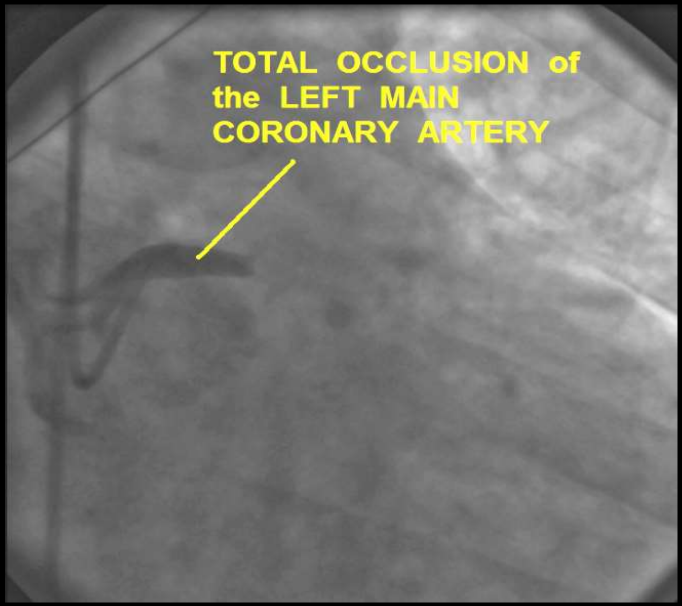
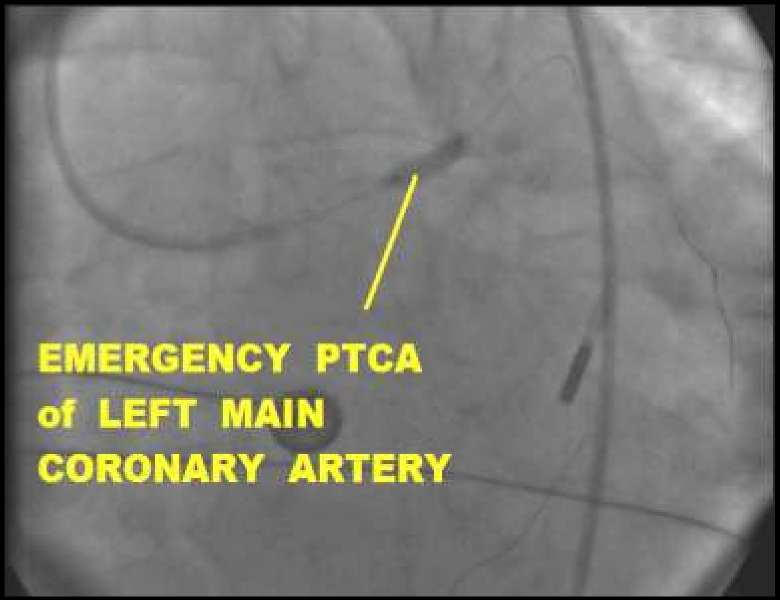
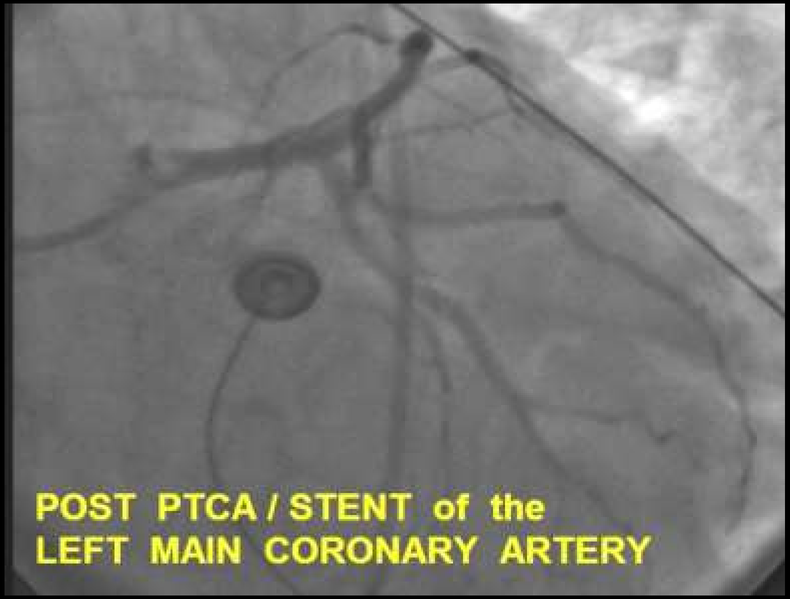

TOTAL OCCLUSION of
the LEFT MAIN
CORONARY ARTERY







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.

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.*

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

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- ☑ 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.* *+
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- ☑ ST ELEVATION in AVR is GREATER THAN ST ELEVATION in V1*+
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- ☑ ST ELEVATION in LEAD I and AVL (caused by NO FLOW to DIAGONAL / OBTUSE MARGINAL BRANCHES)*
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+ Yamaji et al, JACC vol. 38, No. 5, 2001, November 1, 2001:1348-54

[Yamaji et al, JACC vol 38, No 5, 2001: 1348-54]

[Electrocardiogram patterns in acute left main occlusion: J Electrocardiol. 2008 Nov-Dec;41(6):626-9.]

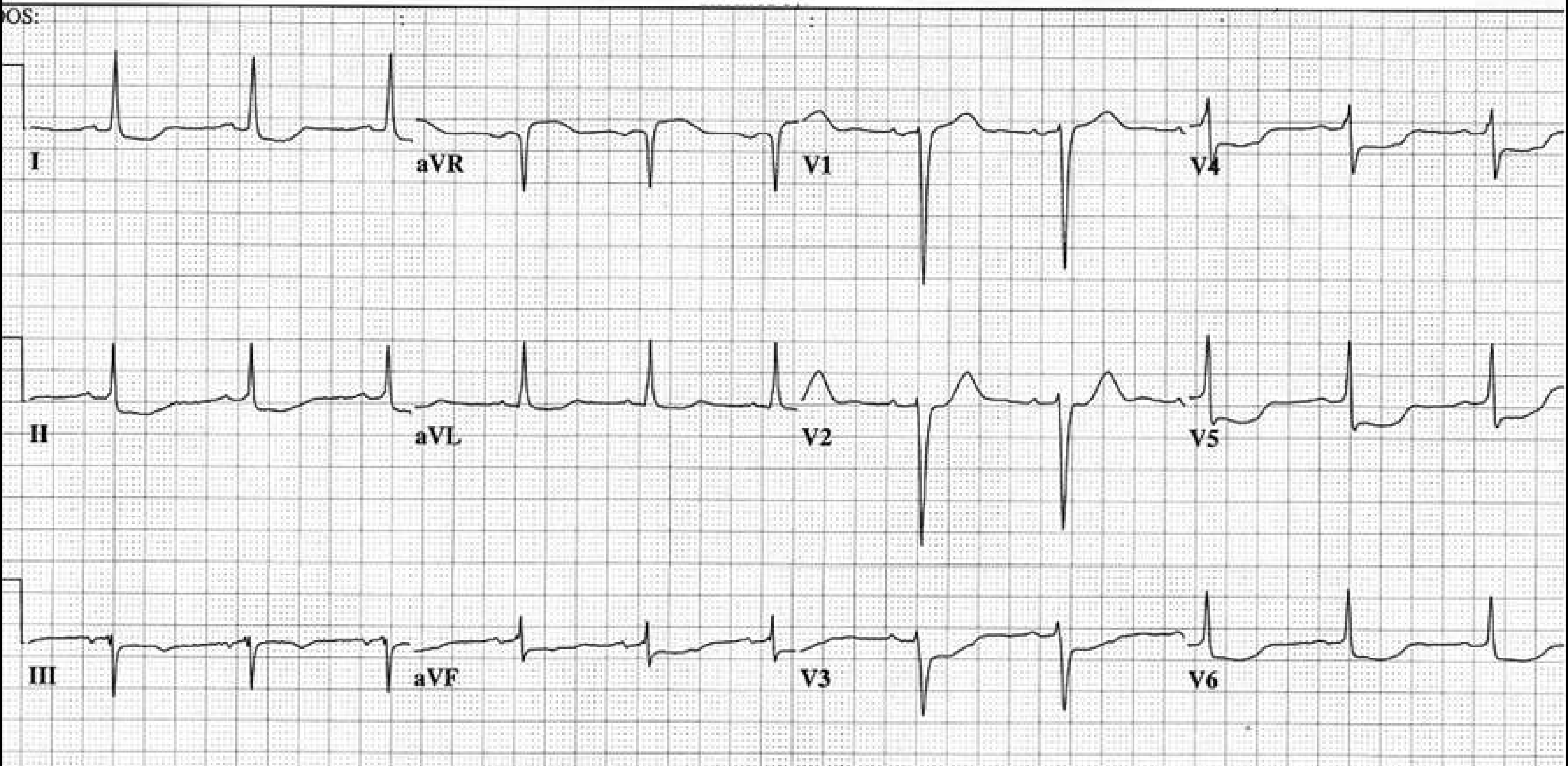
In patients without STEMI, ST Elevation in AVR, when seen with global indications of ischemia (ST Depression in 8 leads or more), is indicative of advanced multi-vessel disease or significant Left Main Coronary Artery stenosis

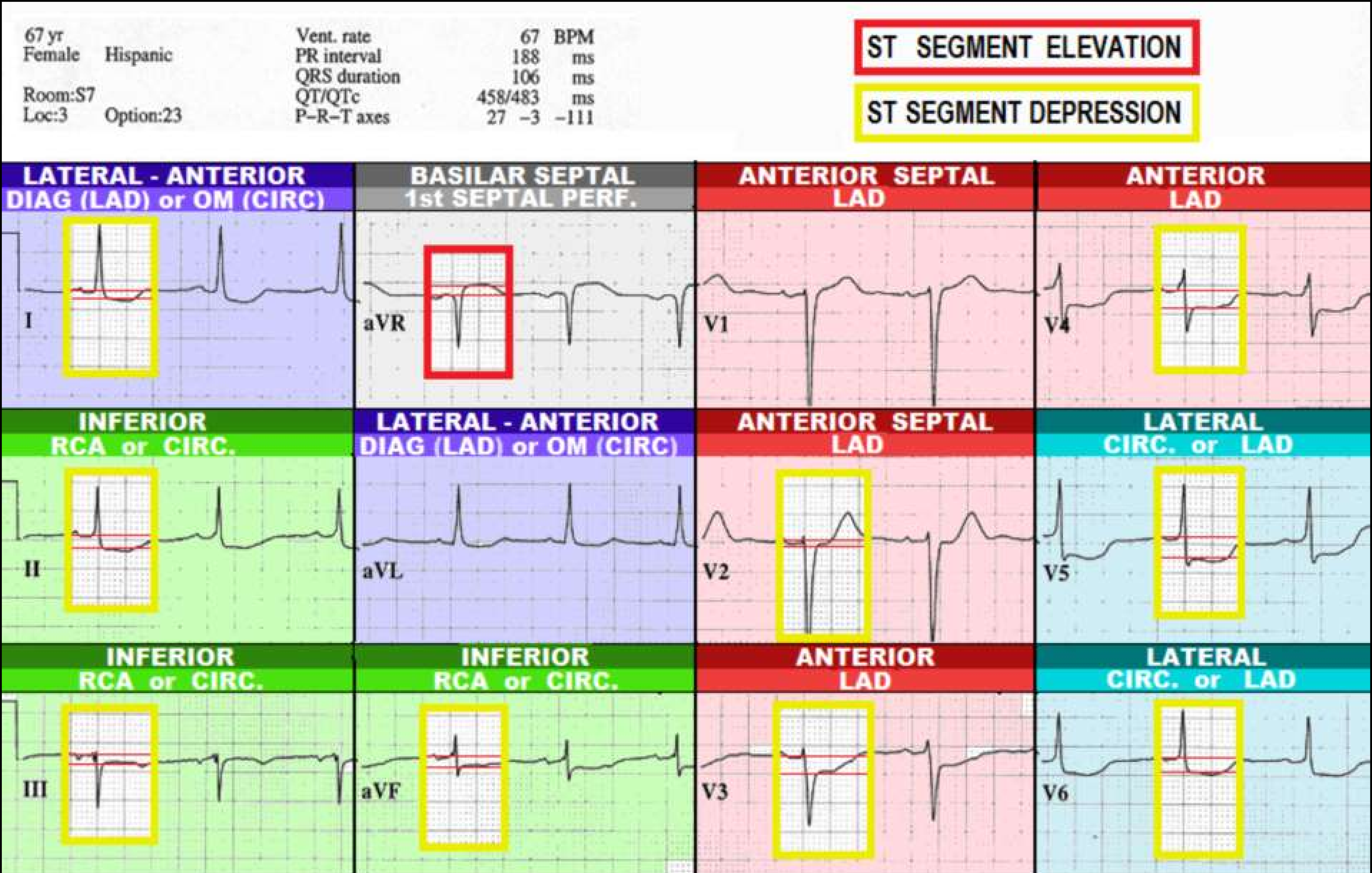
“In patients with:

- **Angina at rest**
- **ST Elevation in AVR and ST Depression in 8 or more ECG leads (global ischemia), it is reported with a *75% predictive accuracy* of 3-vessel or left main coronary artery stenosis” . . .**

- Wagner et al, **2009 ACC/AHA Standardization and Interpretation of the ECG, Part VI, ACS**.

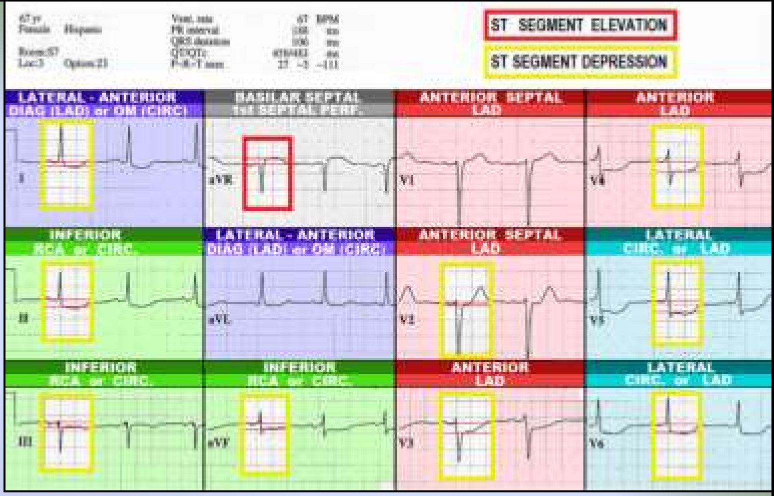
67 yr		Vent. rate	67	BPM
Female	Hispanic	PR interval	188	ms
		QRS duration	106	ms
Room:S7		QT/QTc	458/483	ms
Loc:3	Option:23	P–R–T axes	27 –3	–111

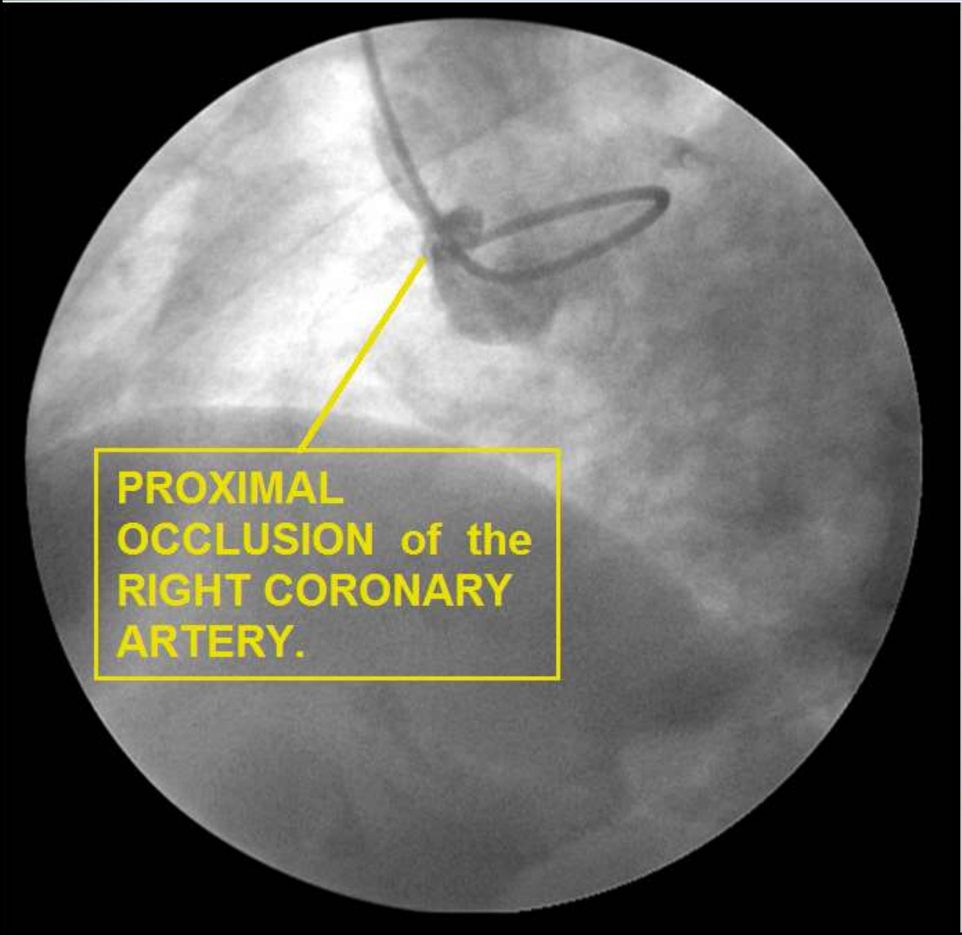


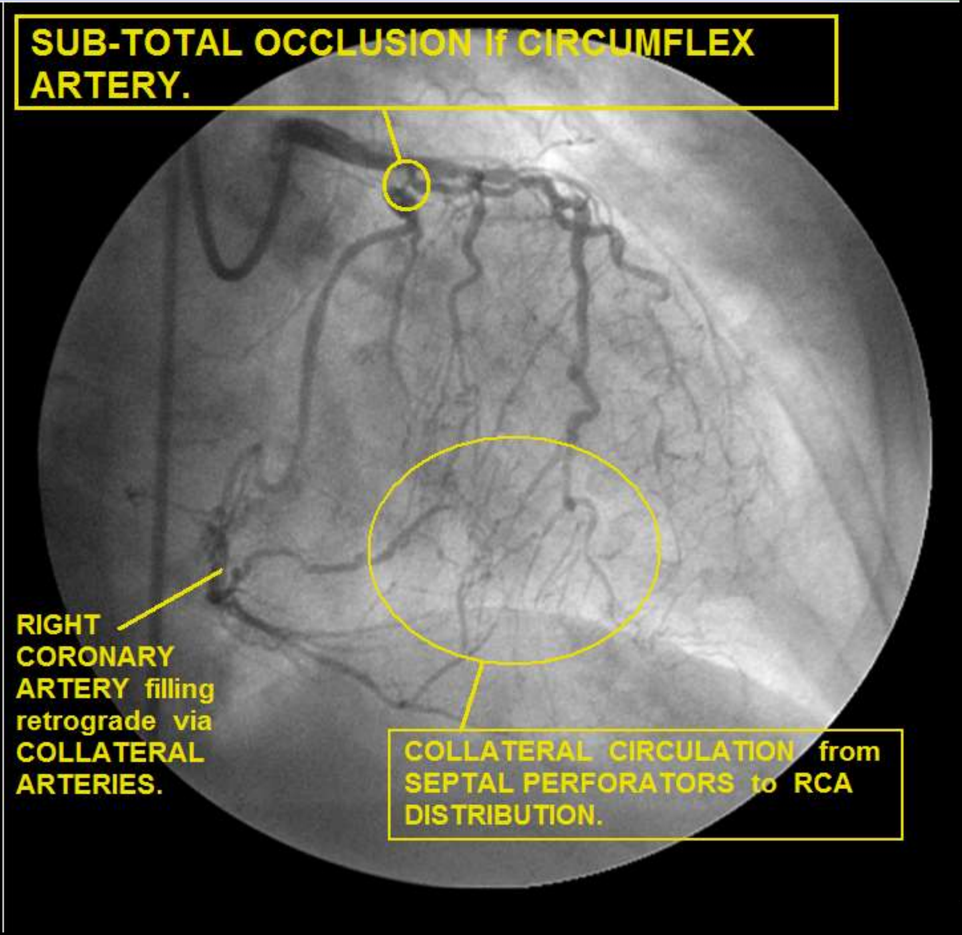


67 yr
Female Hispanic
Room:S7
Loc:3 Option:23
Vent. rate 67 BPM
PR interval 188 ms
QRS duration 106 ms
QT/QTc 458/483 ms
P-R-T axes 27 -3 -111
ST SEGMENT ELEVATION
ST SEGMENT DEPRESSION
LATERAL - ANTERIOR
DIAG (LAD) or OM (CIRC)
I
BASILAR SEPTAL
1st SEPTAL PERF.
aVR
ANTERIOR SEPTAL
LAD
V1
ANTERIOR
LAD
V4
INFERIOR
RCA or CIRC.
II
LATERAL - ANTERIOR
DIAG (LAD) or OM (CIRC)
aVL
ANTERIOR SEPTAL
LAD
V2
LATERAL
CIRC. or LAD
V5
INFERIOR
RCA or CIRC.
III
INFERIOR
RCA or CIRC.
aVF
ANTERIOR
LAD
V3
LATERAL
CIRC. or LAD
V6

Critical Triple Vessel Disease = *STAT Coronary Artery Bypass Surgery*







ANTICIPATED COMPLICATIONS of GLOBAL ISCHEMIA with POSSIBLE NSTEMI -- INTERVENTIONS to be CONSIDERED:

Patients with CHEST PAIN at REST and this ECG presentation have a 75% incidence of severe LMCA STENOSIS and/or TRIPLE - VESSEL DISEASE -- in such cases Coronary Artery Bypass Surgery (CABG) is frequently indicated.	PREHOSPITAL: if patient has no hospital preference consider transport to Chest Pain Center WITH Open Heart Surgery capabilities IF nearby. HOSPITAL: consider use of SHORT-ACTING intravenous GP IIb/IIIa receptor agonists
- ACTIVE CHEST PAIN	ACUTE CHEST PAIN PROTOCOL
- ISCHEMIA - CONSIDER DYSRHYTHMIAS	ACLS PROTOCOL
- INCREASED PROBABILITY of IMMINENT MYOCARDIAL INFARCTION	1. AGGRESSIVE SERIAL TROPONIN and SERIAL ECG PROTOCOLS (2014 AHA /ACC / NSTE-ACS Guidelines) 2. Positive TROPONIN: consider STAT / early Cardiac Catheterization

Excerpt from ***STEMI Assistant***

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.

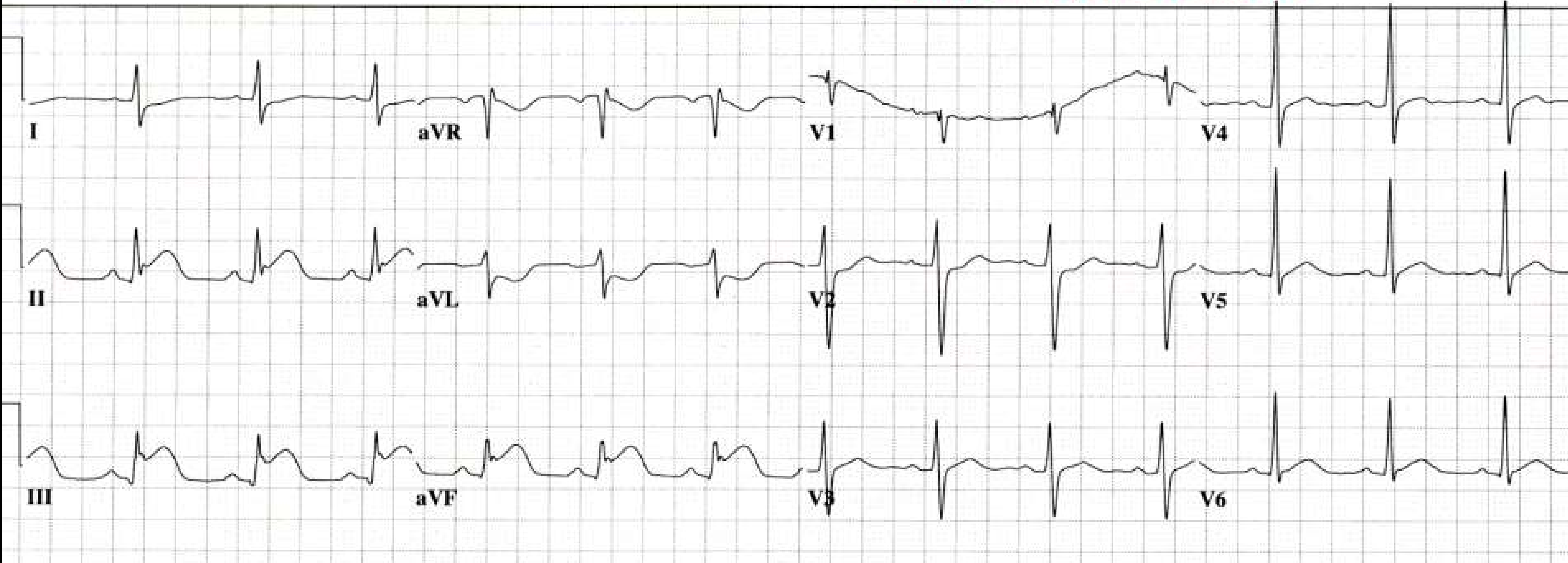
VITAL SIGNS: BP: 136/88 P: 88 R: 20 SAO2: 100% on 4 LPM O2

LABS: TROPONIN: < .04

46 yr		Vent. rate	82	BPM
Male	Caucasian	PR interval	168	ms
		QRS duration	96	ms
		QT/QTc	384/448	ms
Loc:3	Option:23	P–R–T axes	76 81	88

EVALUATE EKG for indicators of ACS:

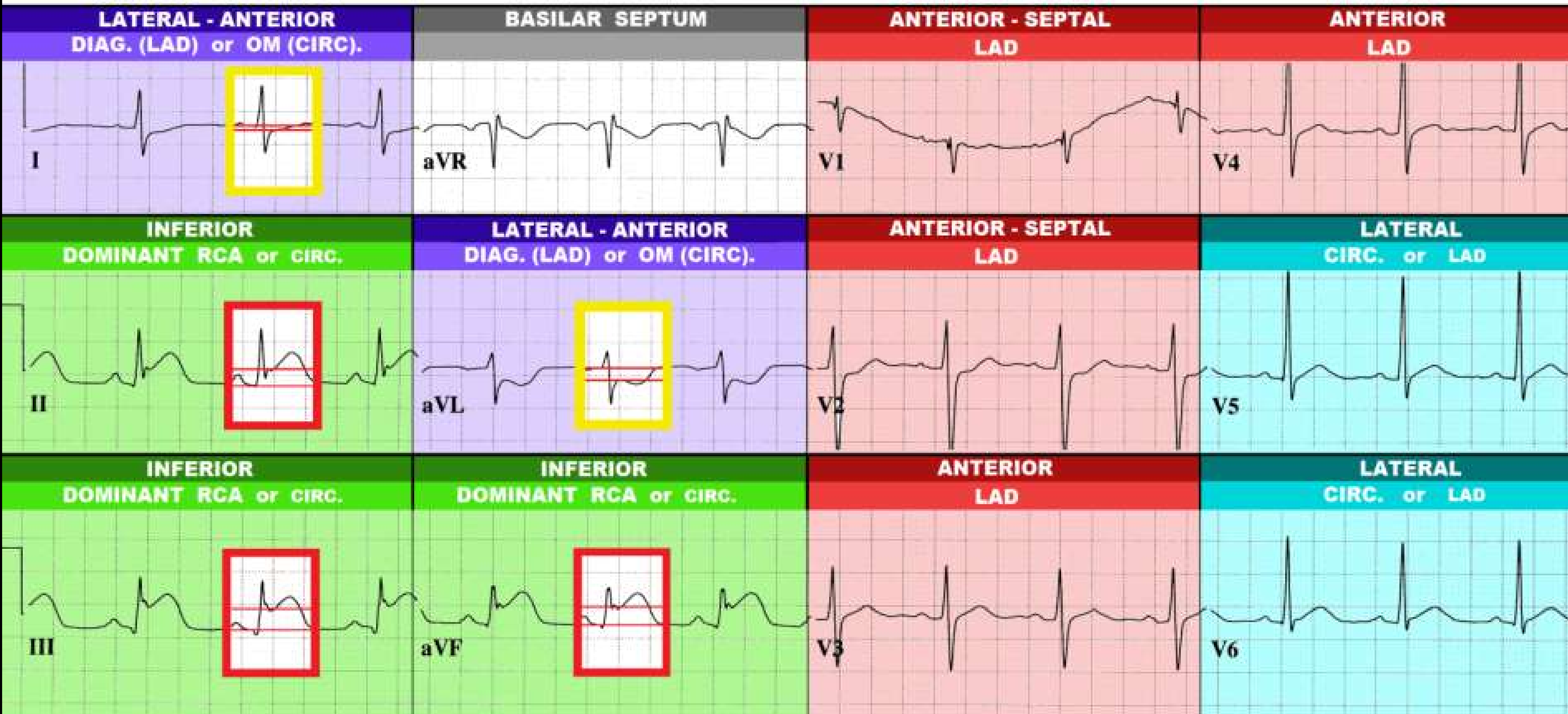
- ST SEGMENT ELEVATION / DEPRESSION
- HYPERACUTE T WAVES
- CONVEX ST SEGMENTS
- OTHER ST SEGMENT / T WAVE ABNORMALITIES

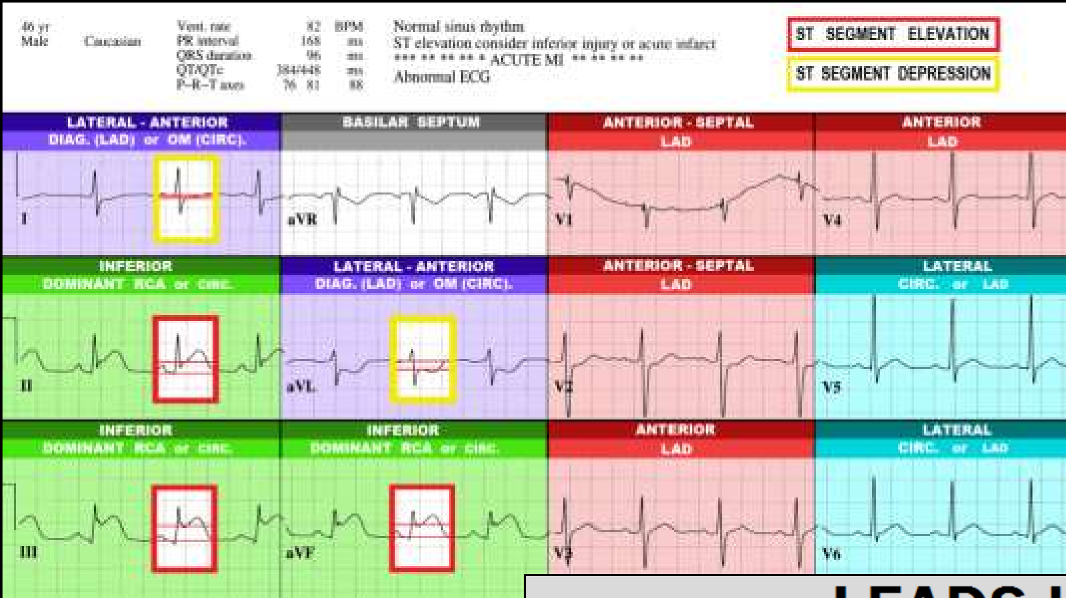


46 yr Male	Caucasian	Vent. rate PR interval QRS duration QT/QTc P–R–T axes	82 168 96 384/448 76 81	BPM ms ms ms 88	Normal sinus rhythm ST elevation consider inferior injury or acute infarct *** ** ** ** * ACUTE MI ** ** ** ** Abnormal ECG

ST SEGMENT ELEVATION

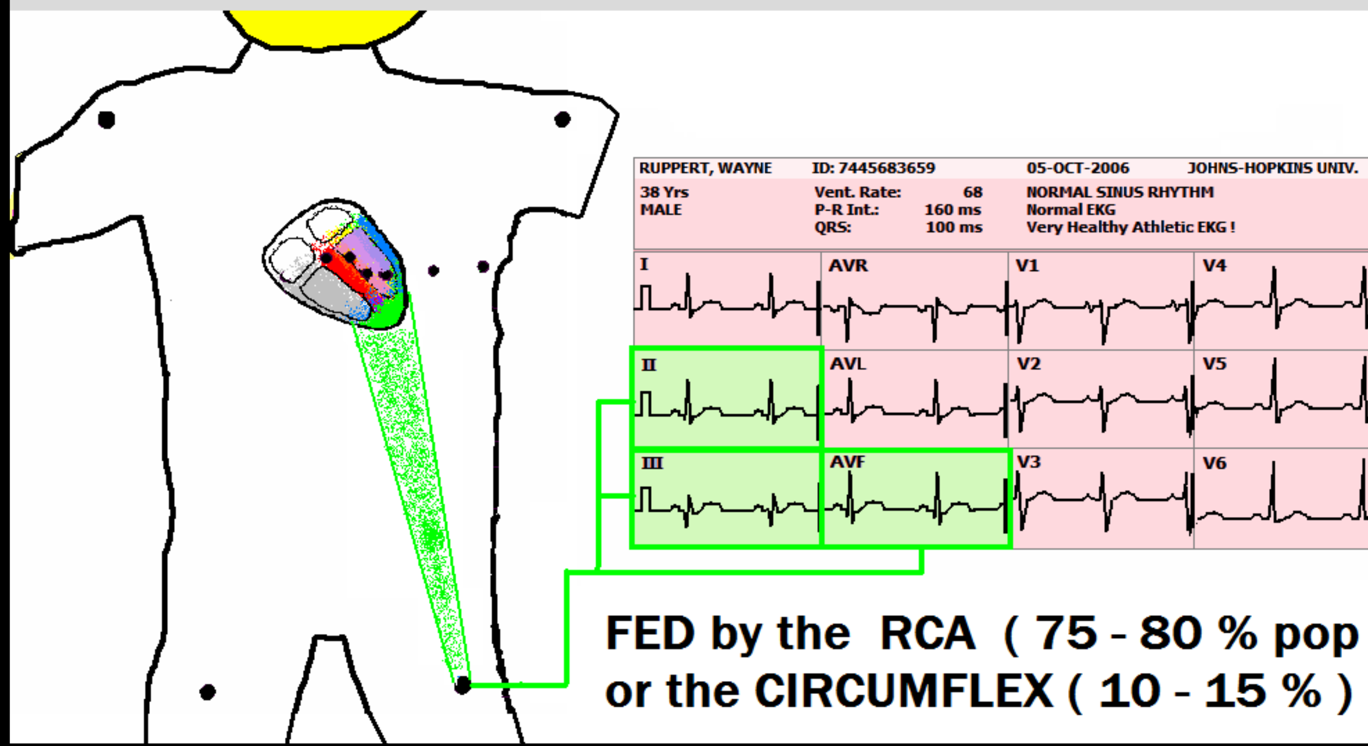
ST SEGMENT DEPRESSION





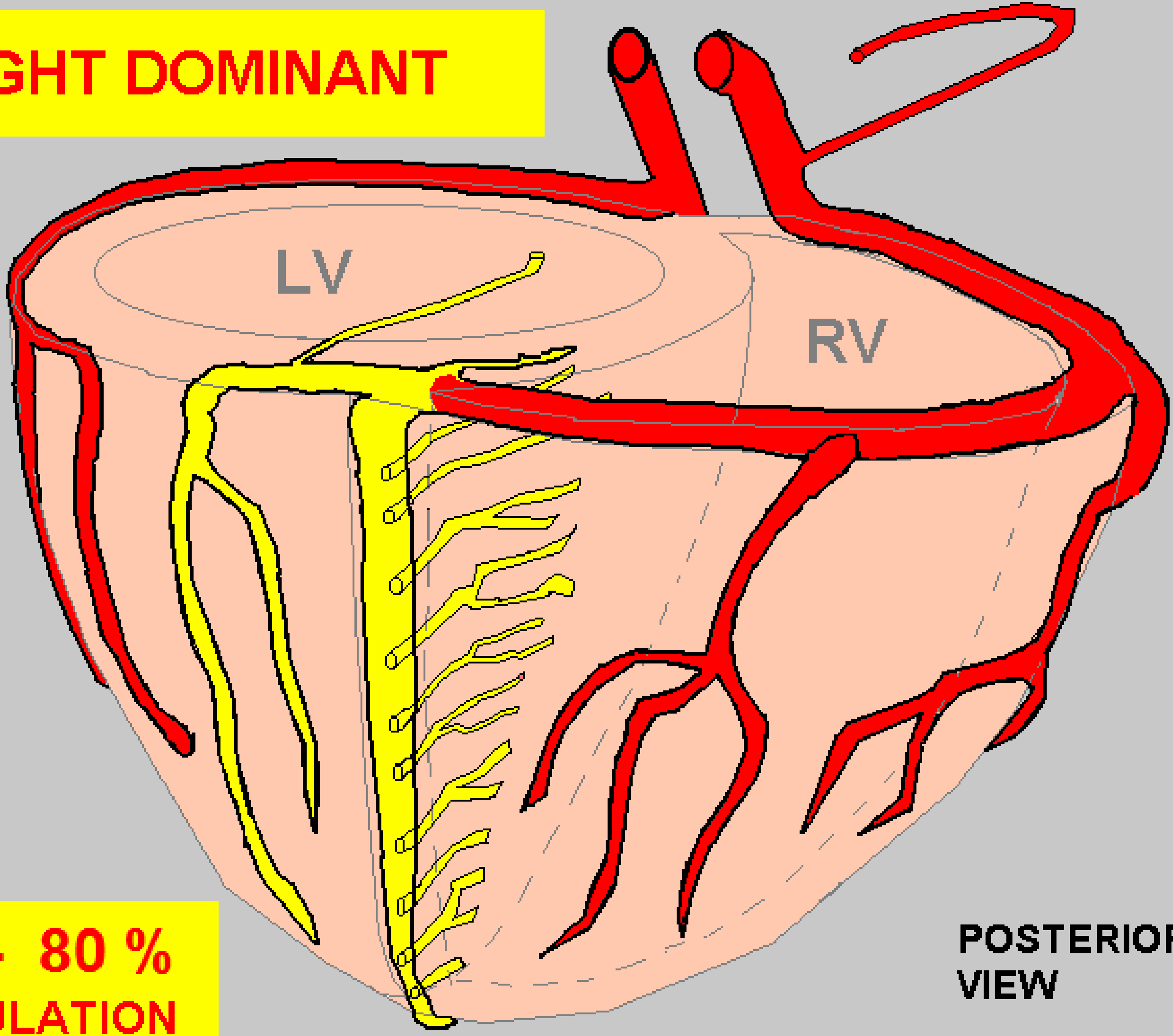
LEADS II, III, and aVF VIEW

INFERIOR WALL of the LEFT VENTRICLE



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

RIGHT DOMINANT



POSTERIOR VIEW

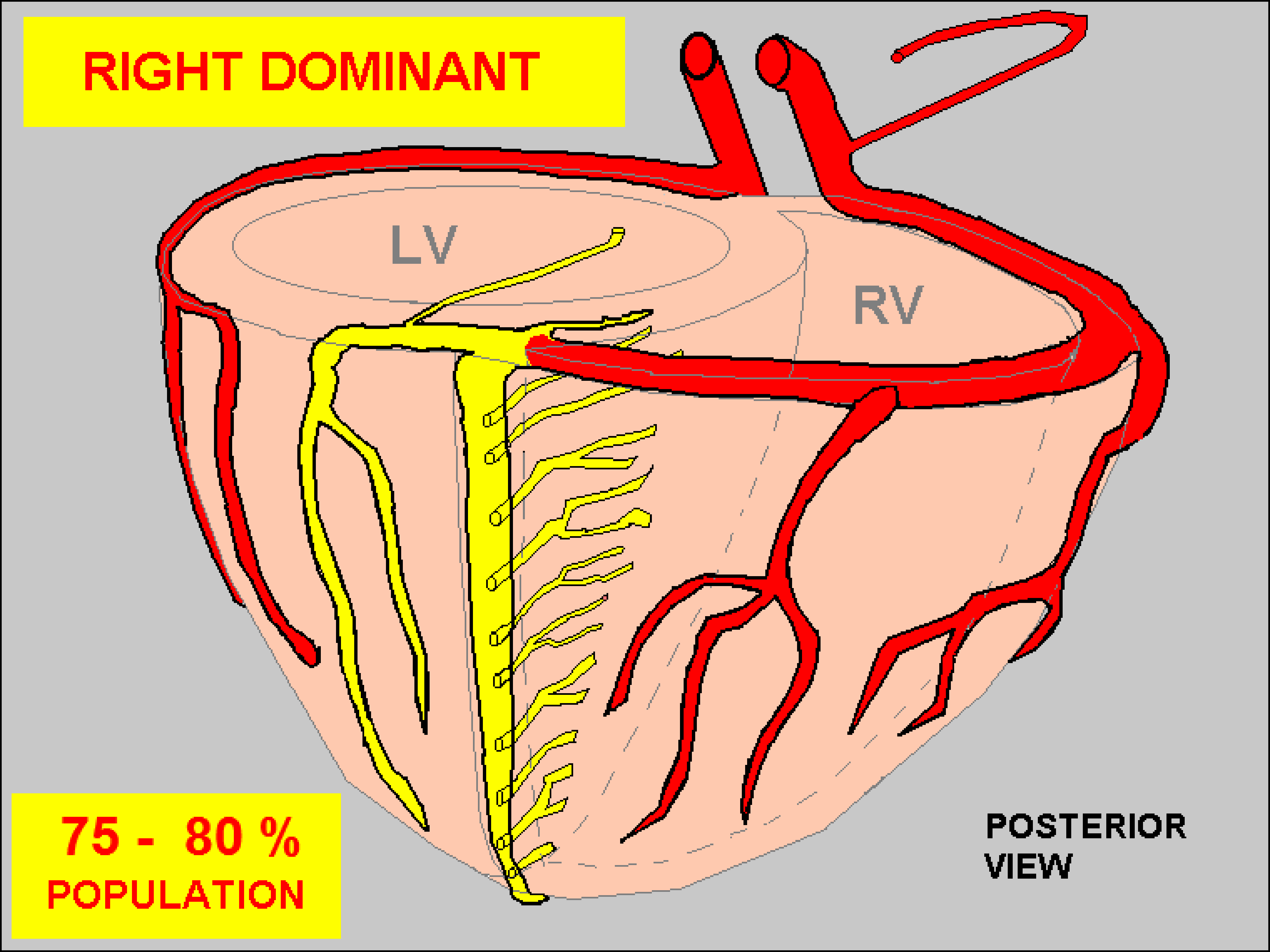
75 - 80 %
POPULATION

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
LV
RV
75 - 80 %
POPULATION
POSTERIOR
VIEW

A standard

12 LEAD EKG

Does NOT show the

RIGHT VENTRICLE

To see the

RIGHT VENTRICLE . . .

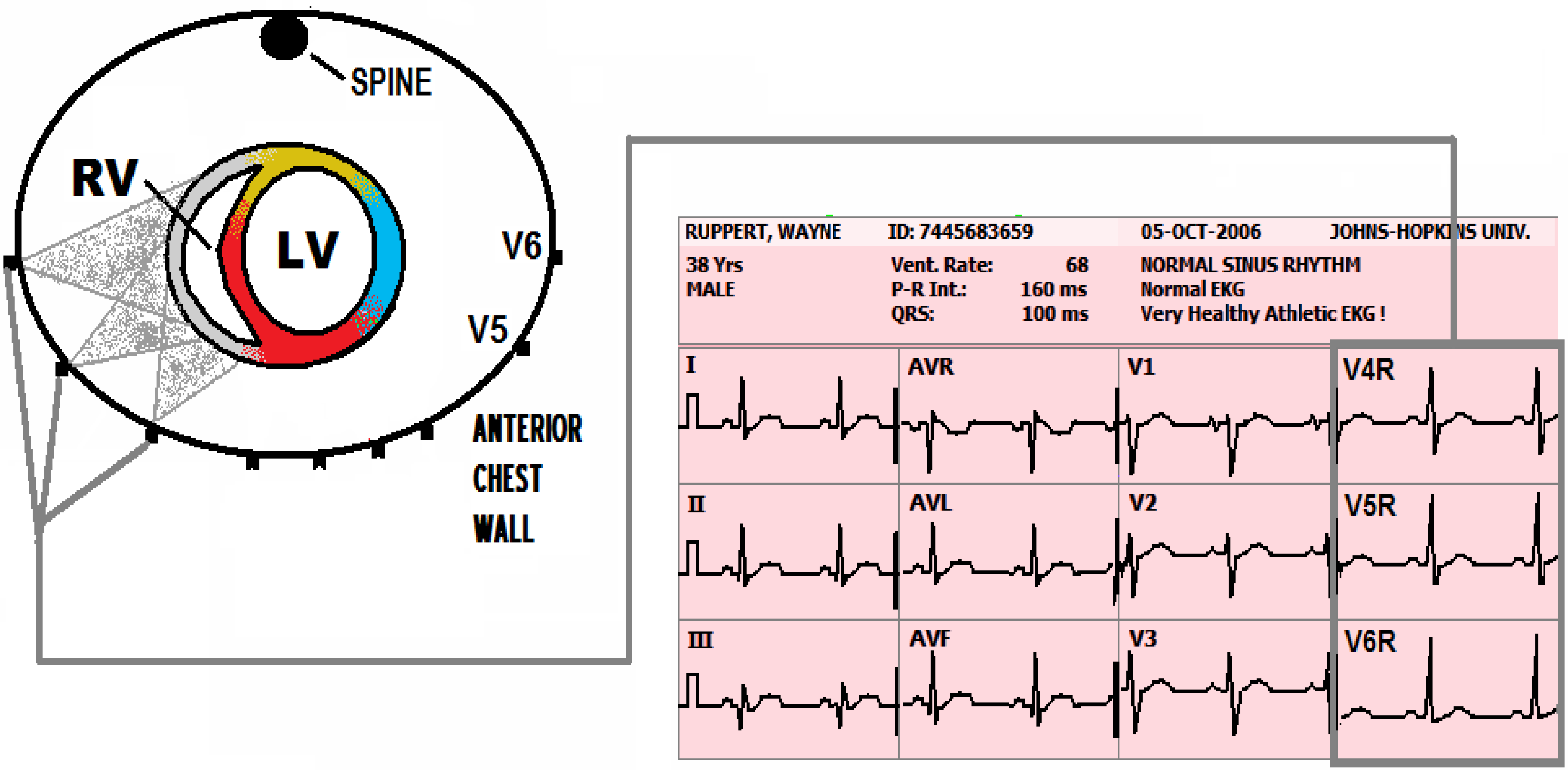
. . . such as in cases of

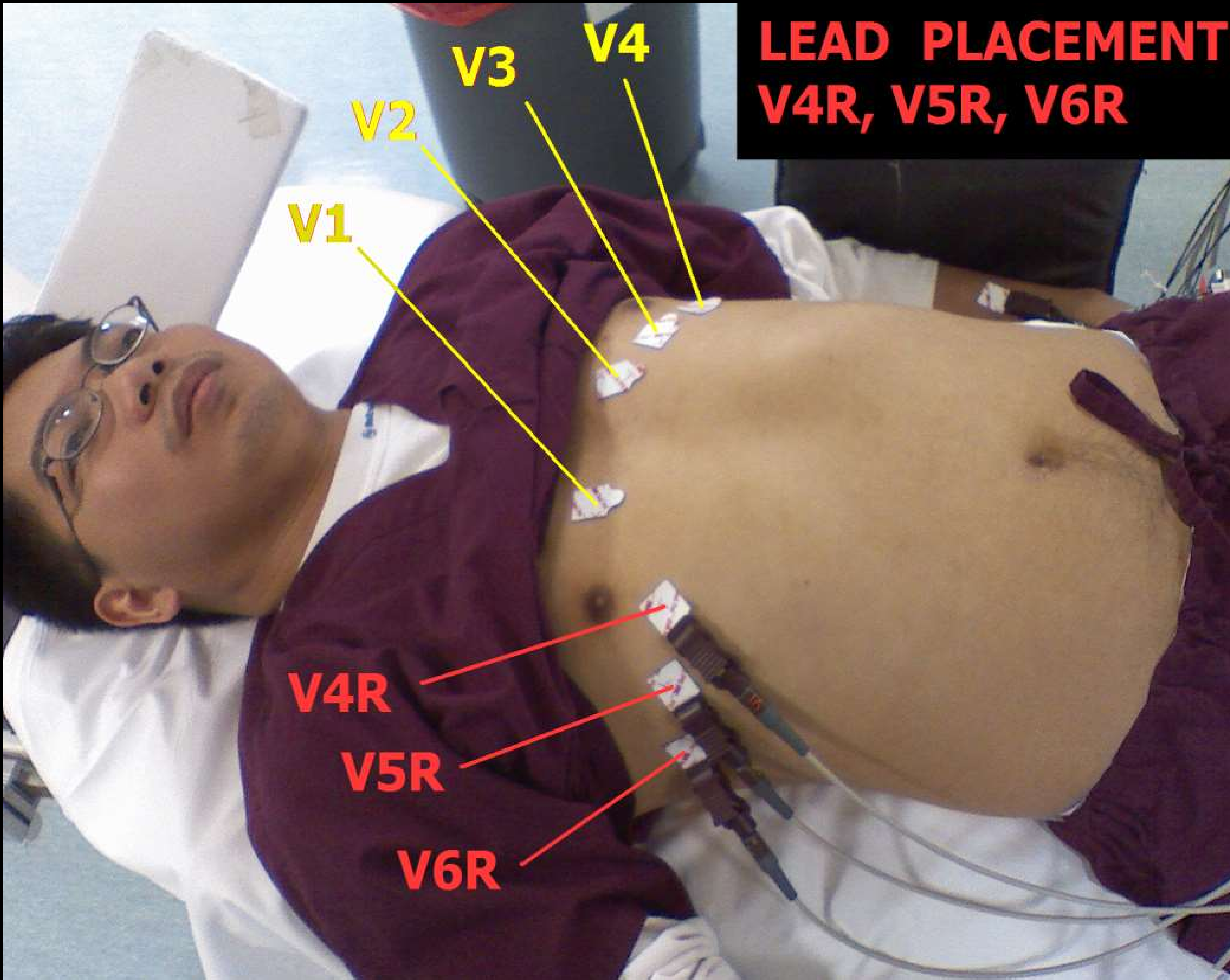
INFERIOR WALL M.I.

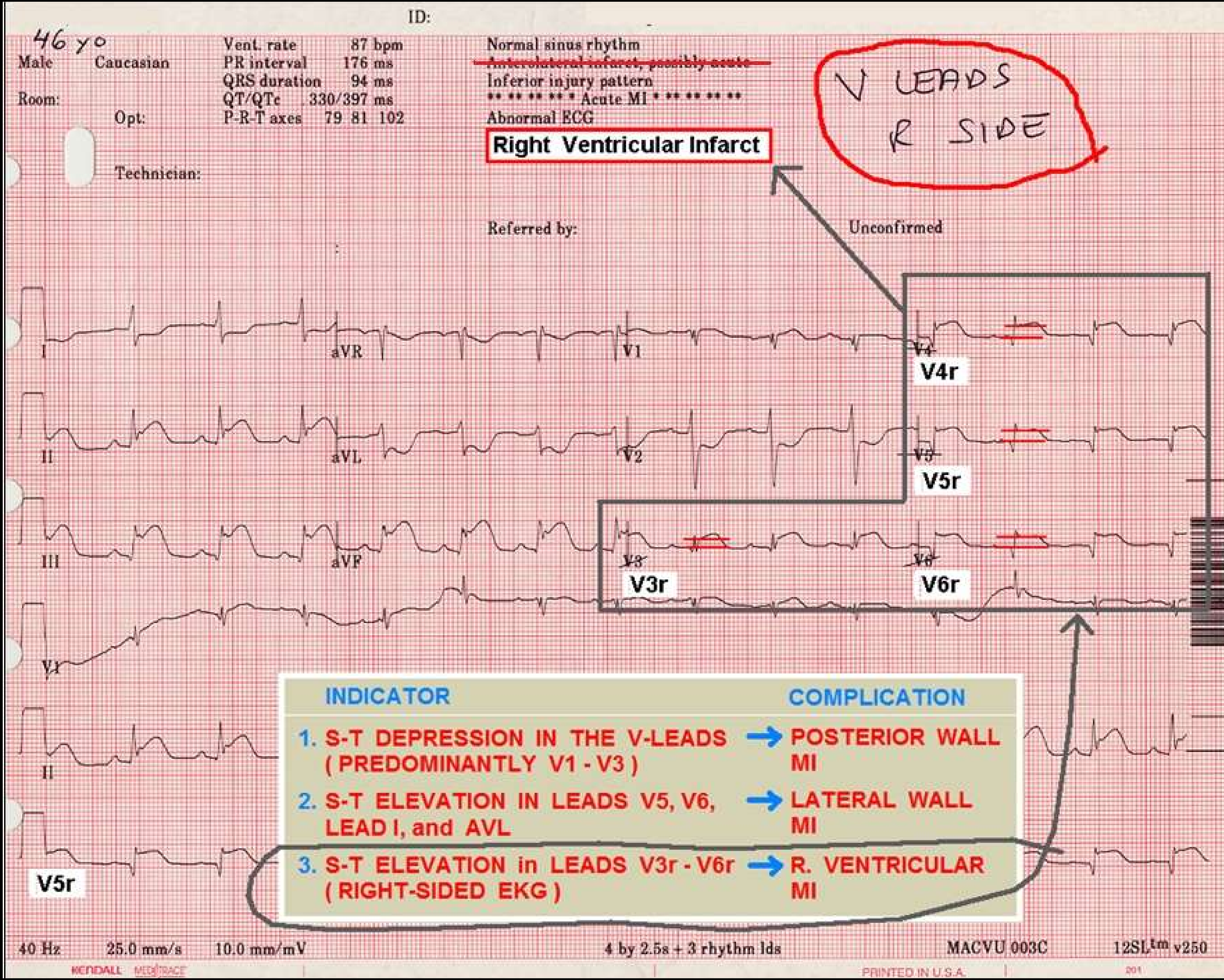
☞ You must do a

RIGHT - SIDED EKG ! !

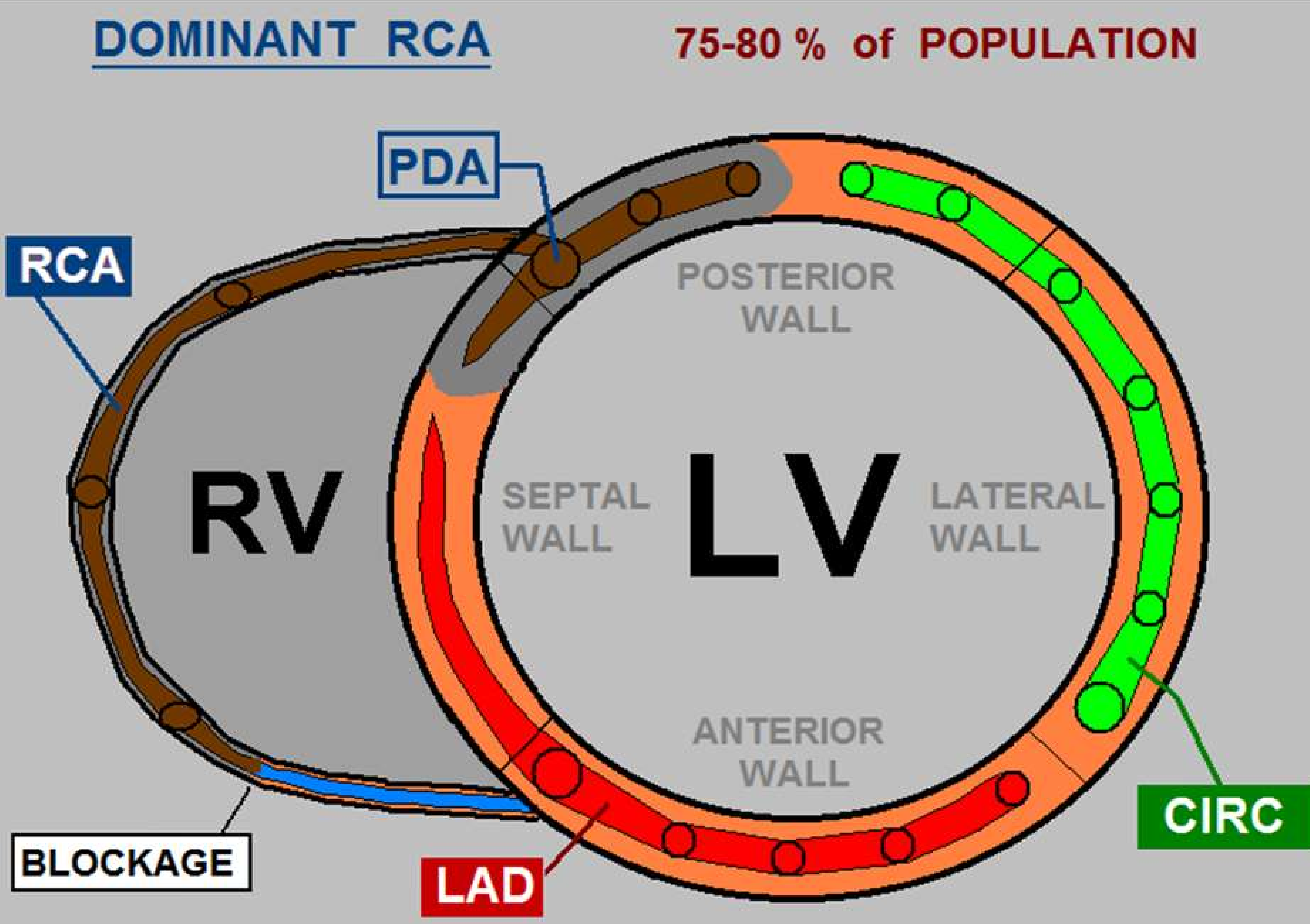
V4R - V6R VIEW THE RIGHT VENTRICLE




LEAD PLACEMENT
V4R, V5R, V6R
V1
V2
V3
V4
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.

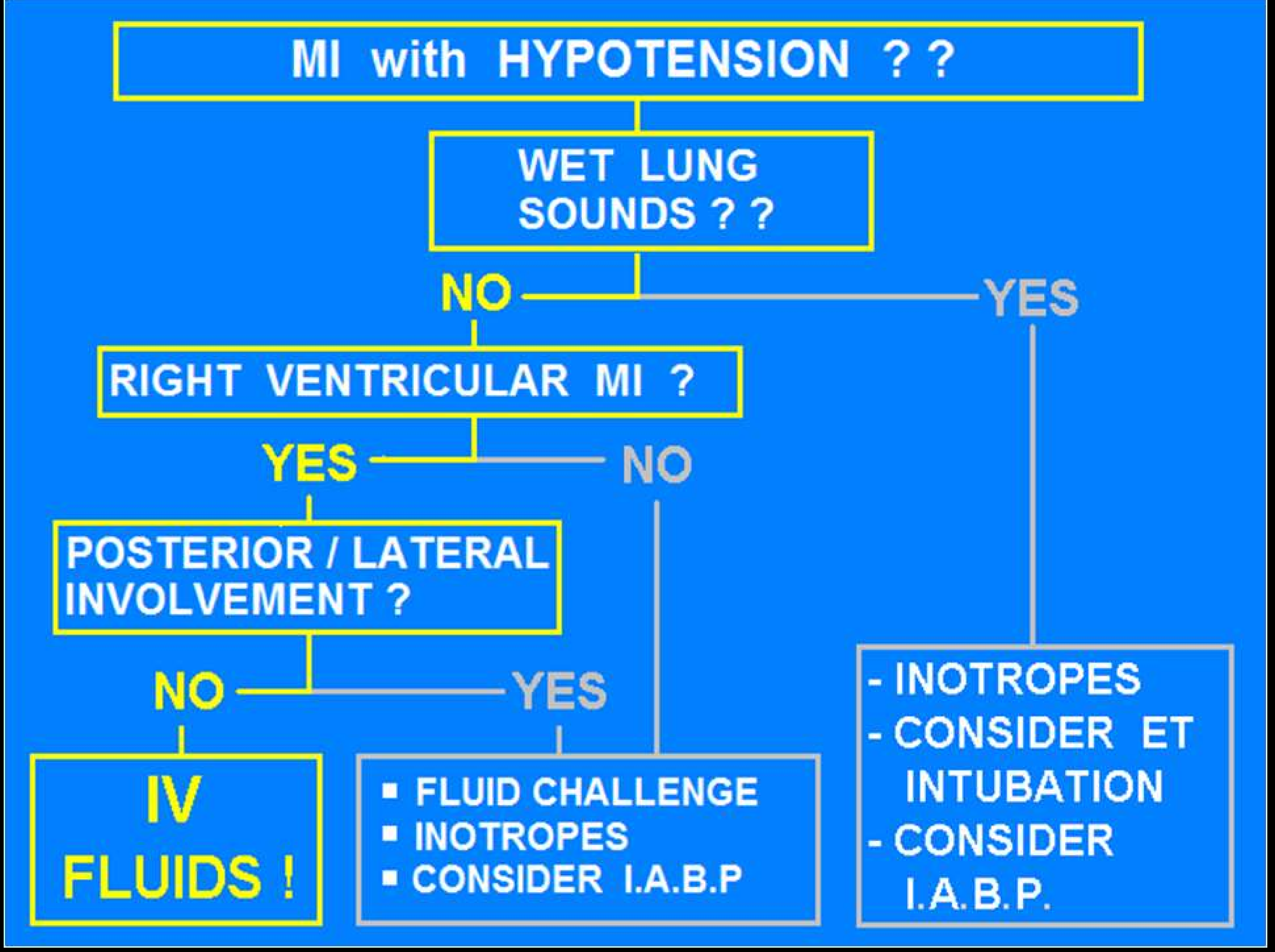
INFERIOR - RIGHT VENTRICULAR MI

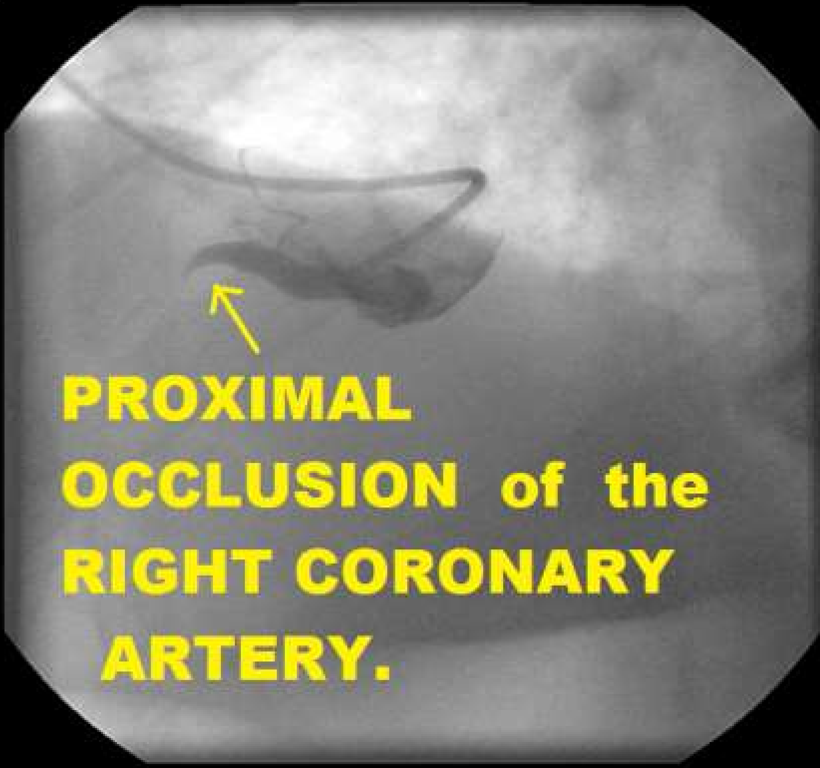


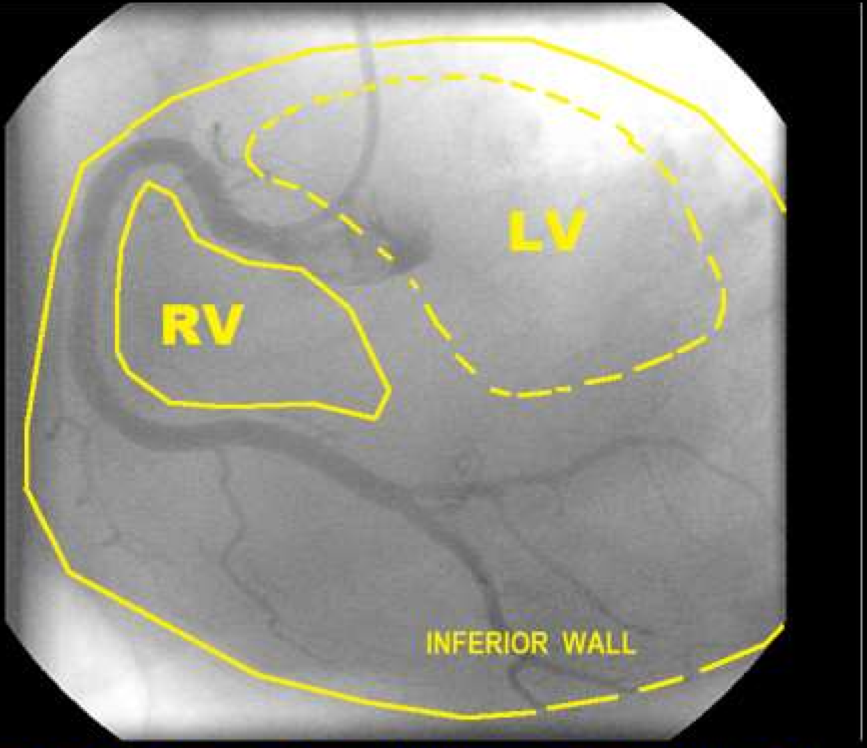
ANTICIPATED COMPLICATIONS of INFERIOR WALL STEMI secondary to RCA Occlusion & POSSIBLE INDICATED INTERVENTIONS:

- 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.
- POSTERIOR WALL INFARCTION	- POSTERIOR WALL MI presents on the 12 Lead ECG as ST DEPRESSION in Leads V1 - V3. - POSTERIOR WALL MI is NOT PRESENT ON THIS ECG.

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

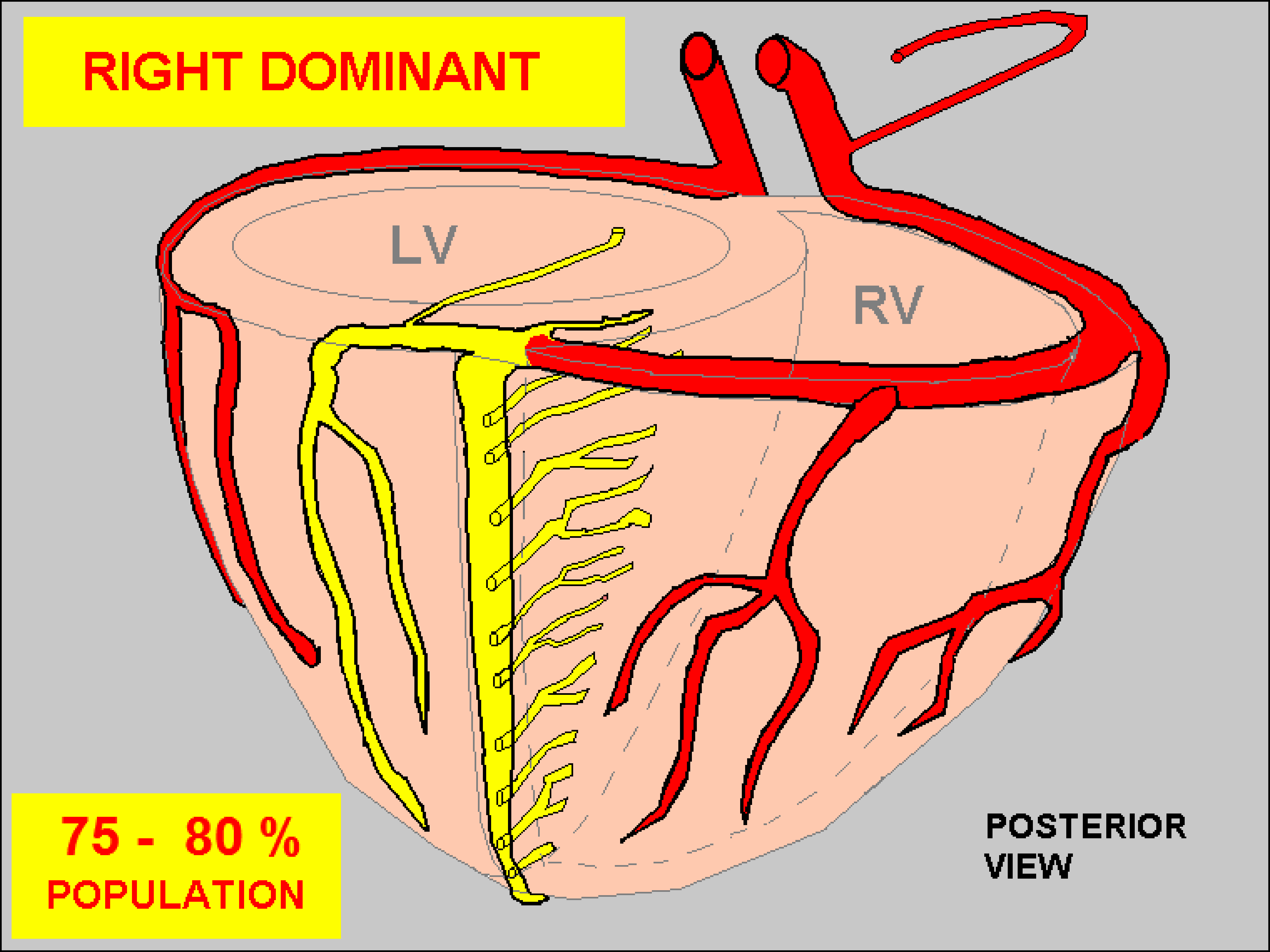
CLASS III CONTRINDICATED

in

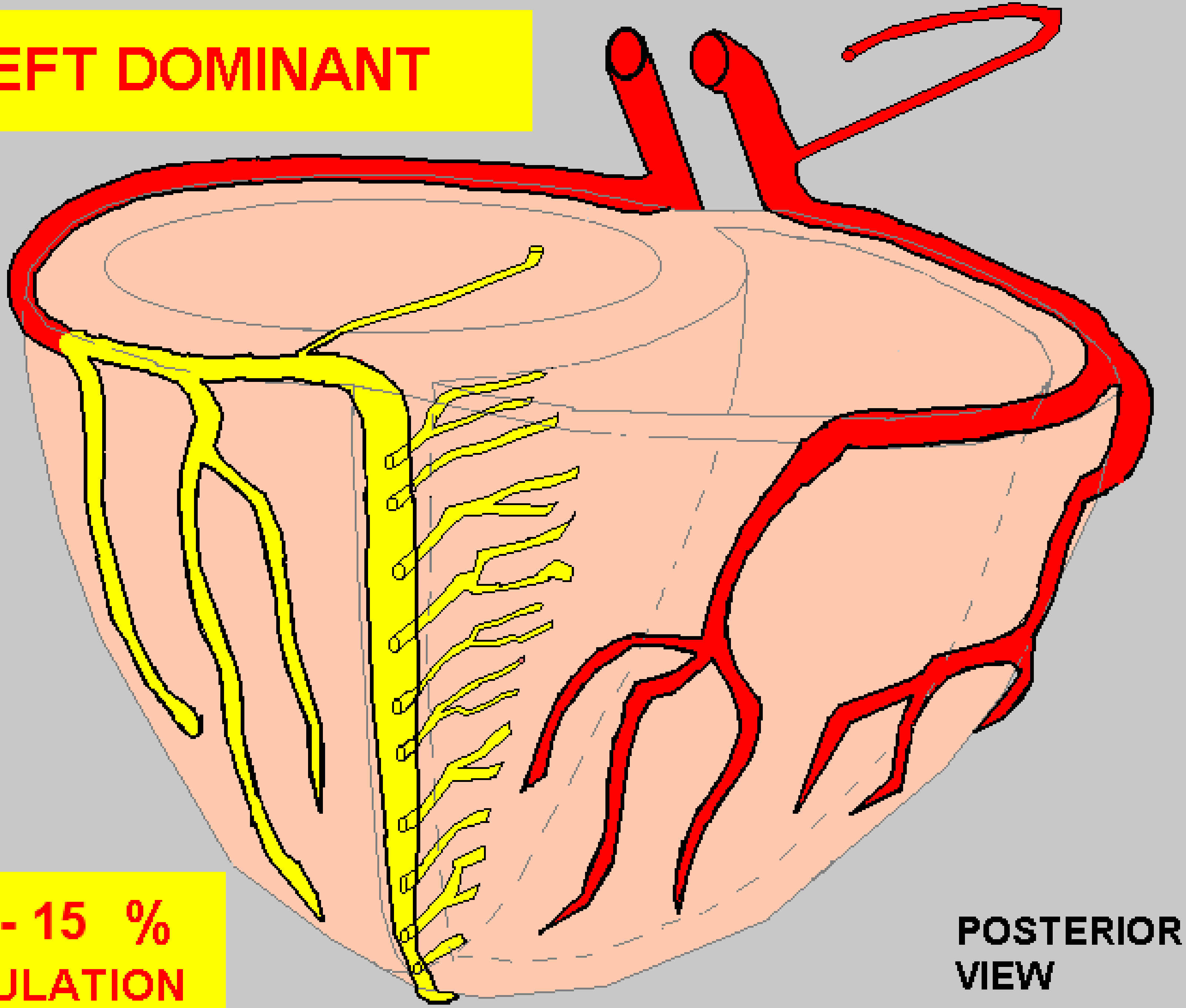
RIGHT VENTRICULAR MI ! !*

They precipitate SEVERE HYPOTENSION

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


RIGHT DOMINANT
LV
RV
75 - 80 %
POPULATION
POSTERIOR
VIEW

LEFT DOMINANT



10 - 15 %
POPULATION

CASE STUDY 9 - STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

42 y/o MALE arrived via EMS, c/o "HEAVY CHEST PRESSURE," SHORTNESS of BREATH X 40 min.
He has experienced V-FIB and been DEFIBRILLATED multiple times

RISK FACTOR PROFILE:

- CIGARETTE SMOKER
- HYPERTENSION
- HIGH LDL CHOLESTEROL

PHYSICAL EXAM: Patient is alert & oriented x 4, ANXIOUS, with COOL, PALE, DIAPHORETIC SKIN. C/O NAUSEA, and is VOMITING. LUNG SOUNDS: COARSE CRACKLES, BASES, bilaterally

VITAL SIGNS: BP: 80/40 P: 70 R: 32 SAO2: 92 % on 15 LPM O2

LABS: TROPONIN: < .04

SHOCK ASSESSMENT

LOC:	ANXIOUS RESTLESS LETHARGIC UNCONSCIOUS	AWAKE ALERT & ORIENTED
SKIN:	PALE / ASHEN CYANOTIC COOL DIAPHORETIC	NORMAL HUE WARM DRY
BREATHING:	TACHYPNEA	NORMAL
PULSE:	WEAK / THREADY TOO FAST or SLOW	STRONG
STATUS:	SHOCK	NORMAL

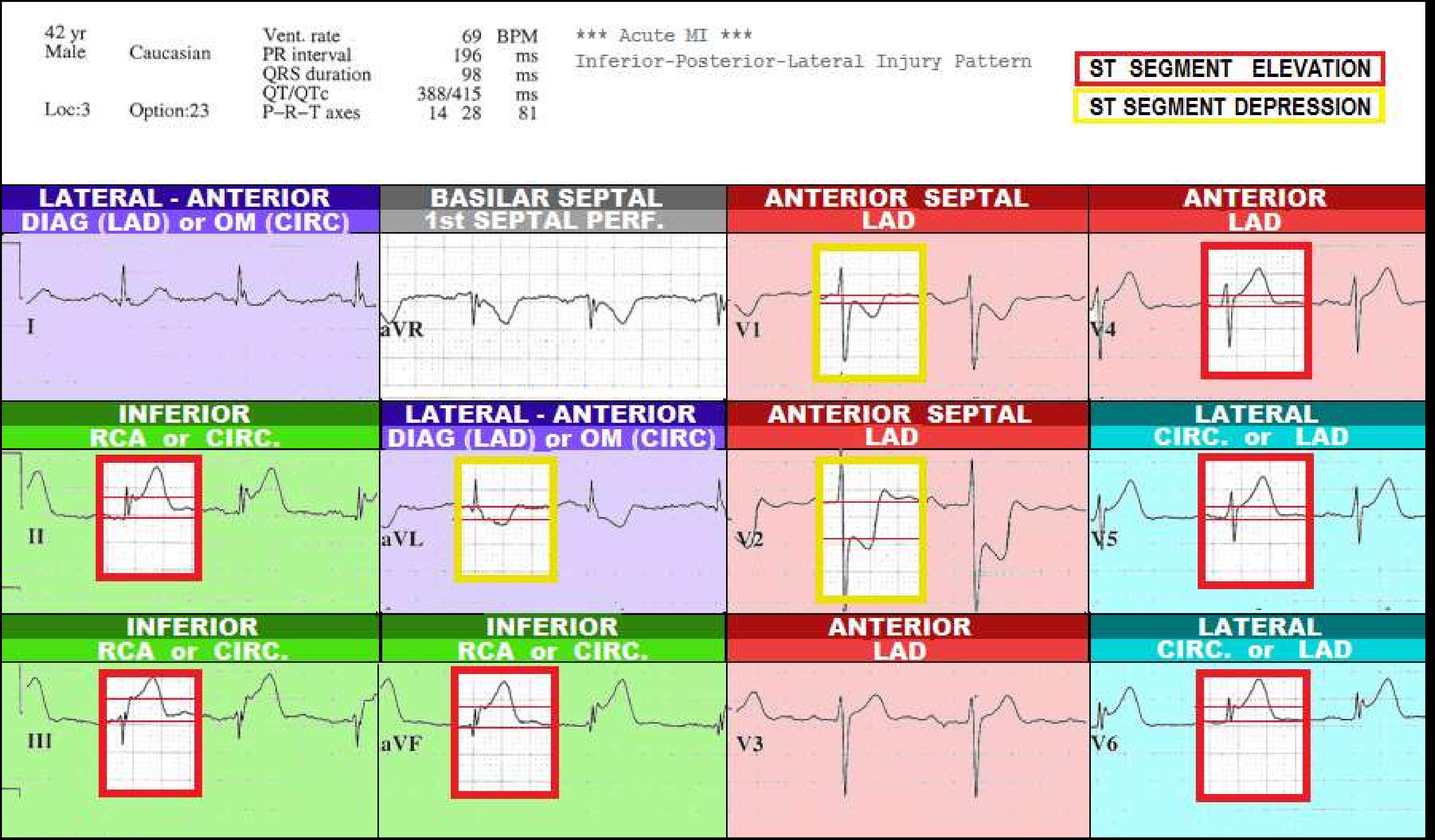
42 yr Male	Caucasian	Vent. rate	69	BPM
		PR interval	196	ms
		QRS duration	98	ms
		QT/QTc	388/415	ms
Loc:3	Option:23	P–R–T axes	14 28	81

EVALUATE EKG for indicators of ACS:

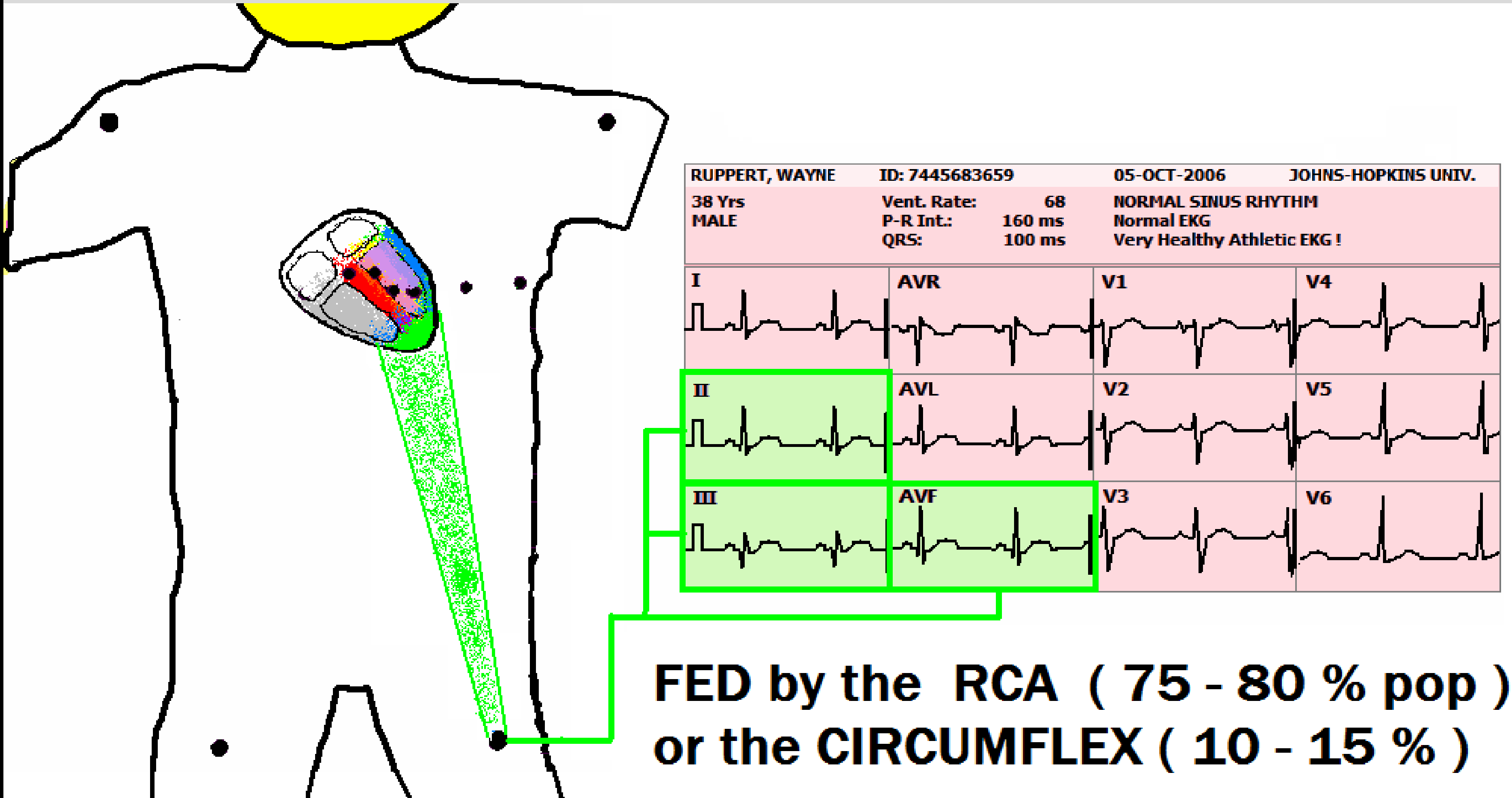
- ST SEGMENT ELEVATION / DEPRESSION
- HYPERACUTE T WAVES
- CONVEX ST SEGMENTS
- OTHER ST SEGMENT / T WAVE ABNORMALITIES

CASE STUDY QUESTIONS:

NOTE LEADS WITH ST ELEVATION:	**NOTE LEADS WITH ST DEPRESSION:**
WHAT IS THE SUSPECTED DIAGNOSIS ?	
WHAT IS THE "CULPRIT ARTERY" -- if applicable ?	
LIST ANY CRITICAL STRUCTURES COMPROMISED:	**LIST ANY POTENTIAL COMPLICATIONS:**


42 yr
Male
Caucasian
Loc:3
Option:23
Vent. rate 69 BPM
PR interval 196 ms
QRS duration 98 ms
QT/QTc 388/415 ms
P–R–T axes 14 28 81
*** Acute MI ***
Inferior-Posterior-Lateral Injury Pattern
ST SEGMENT ELEVATION
ST SEGMENT DEPRESSION
LATERAL - ANTERIOR
DIAG (LAD) or OM (CIRC)
I
BASILAR SEPTAL
1st SEPTAL PERF.
aVR
ANTERIOR SEPTAL
LAD
V1
ANTERIOR
LAD
V4
INFERIOR
RCA or CIRC.
II
LATERAL - ANTERIOR
DIAG (LAD) or OM (CIRC)
aVL
ANTERIOR SEPTAL
LAD
V2
LATERAL
CIRC. or LAD
V5
INFERIOR
RCA or CIRC.
III
INFERIOR
RCA or CIRC.
aVF
ANTERIOR
LAD
V3
LATERAL
CIRC. or LAD
V6

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



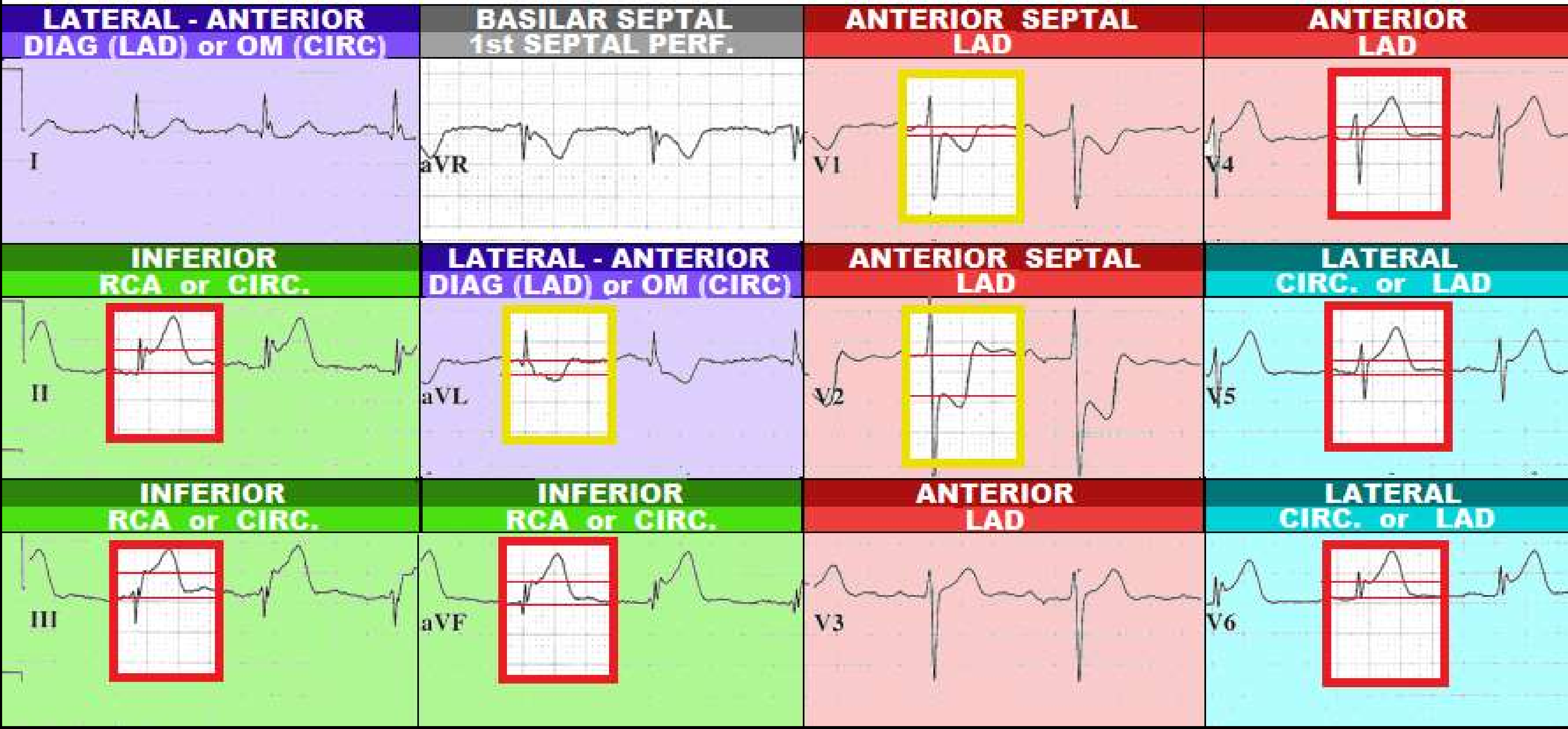
FED by the RCA (75 - 80 % pop)
or the CIRCUMFLEX (10 - 15 %)

42 yr
Male Caucasian
Loc:3 Option:23

Vent. rate 69 BPM
PR interval 196 ms
QRS duration 98 ms
QT/QTc 388/415 ms
P–R–T axes 14 28 81

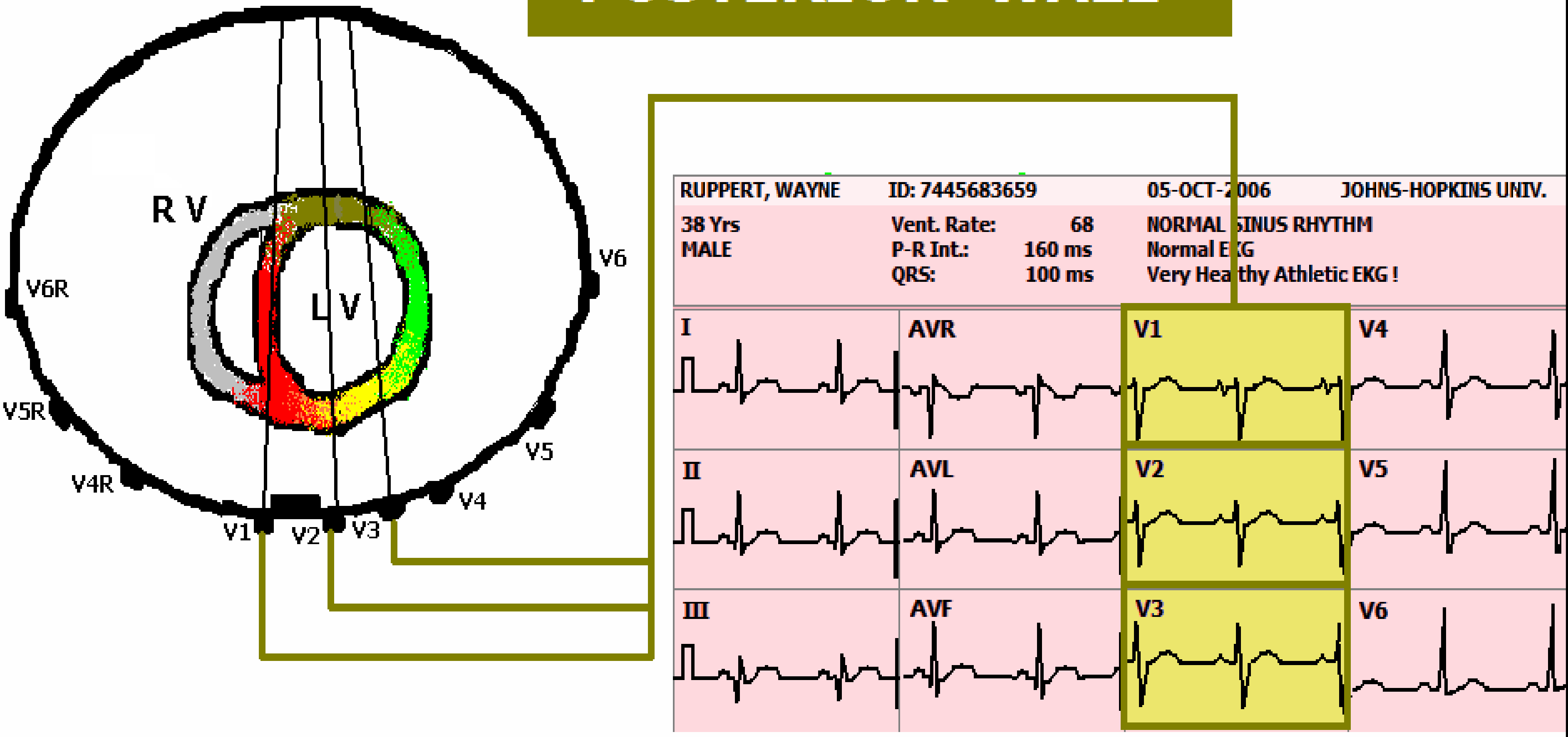
*** Acute MI ***
Inferior-Posterior-Lateral Injury Pattern

ST SEGMENT ELEVATION
ST SEGMENT DEPRESSION



LEADS V1 - V3 *view the*

POSTERIOR WALL

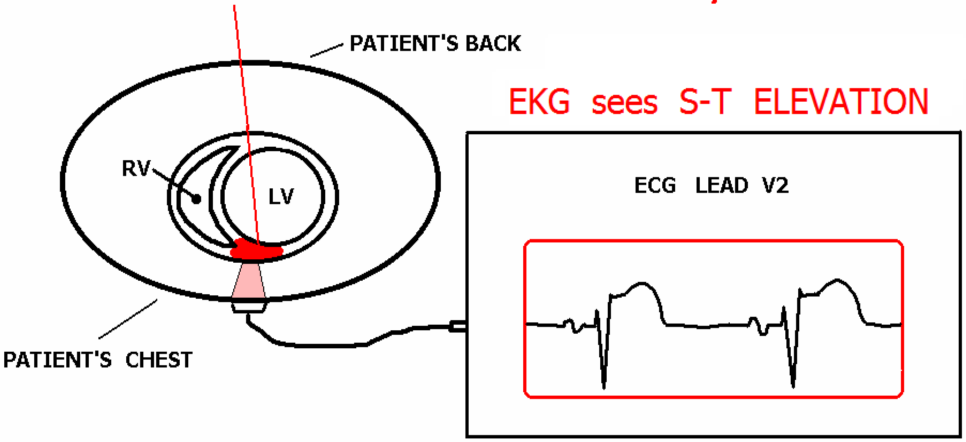


via RECIPROCAL CHANGES.

HOW EKG VIEWS INDICATIVE CHANGES

EXAMPLE:

AREA OF ACUTE INFARCTION - ANTERIOR/SEPTAL



HOW EKG VIEWS INDICATIVE CHANGES

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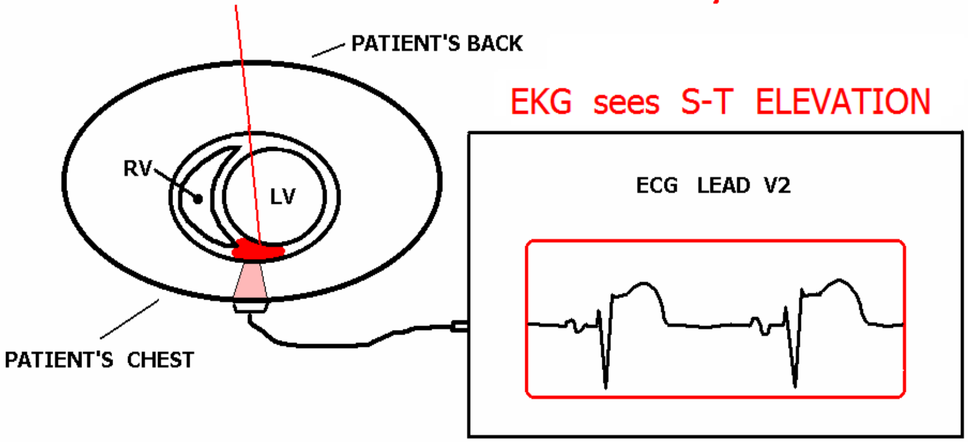
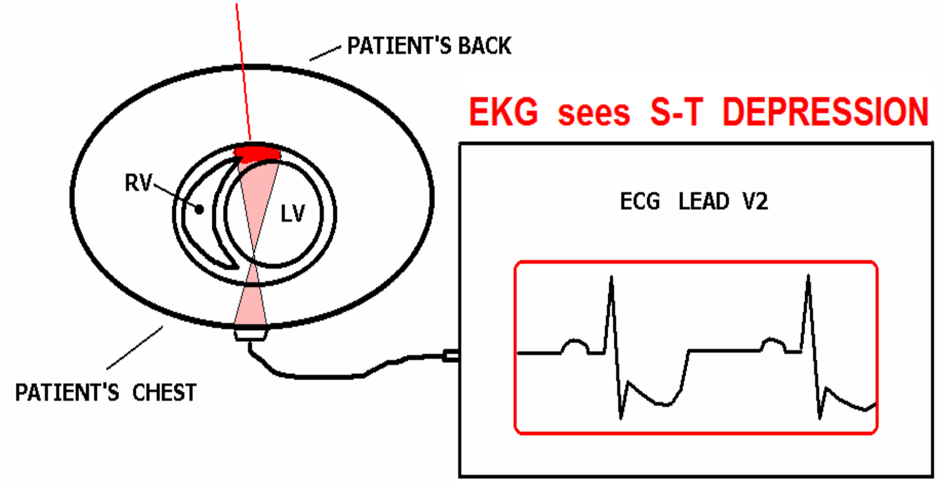
AREA OF ACUTE INFARCTION - ANTERIOR/SEPTAL

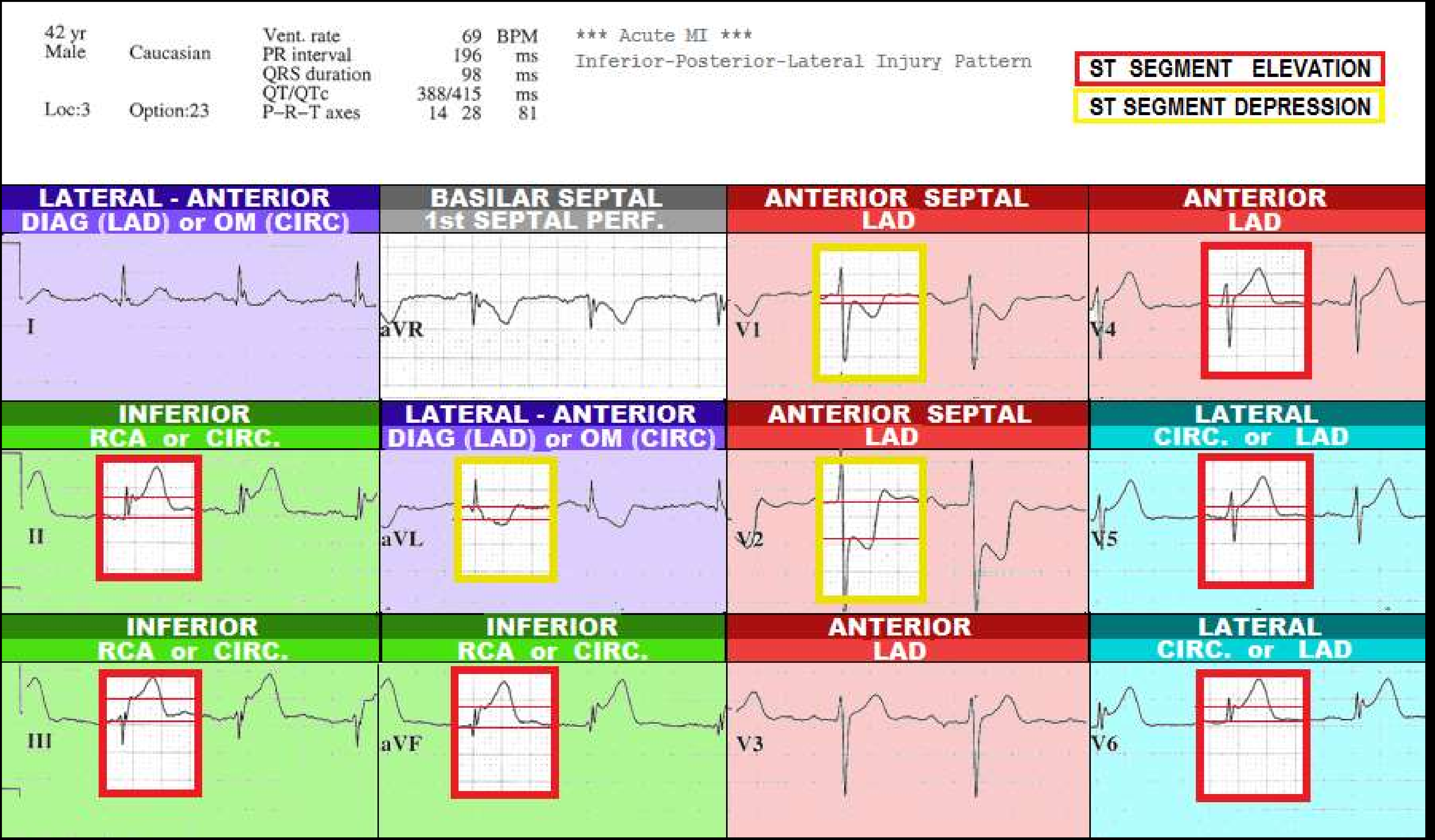


HOW EKG VIEWS RECIPROCAL CHANGES

EXAMPLE:

AREA OF ACUTE INFARCTION - POSTERIOR WALL





42 yr
Male
Caucasian
Loc:3
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Vent. rate 69 BPM
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*** Acute MI ***
Inferior-Posterior-Lateral Injury Pattern
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III
INFERIOR
RCA or CIRC.
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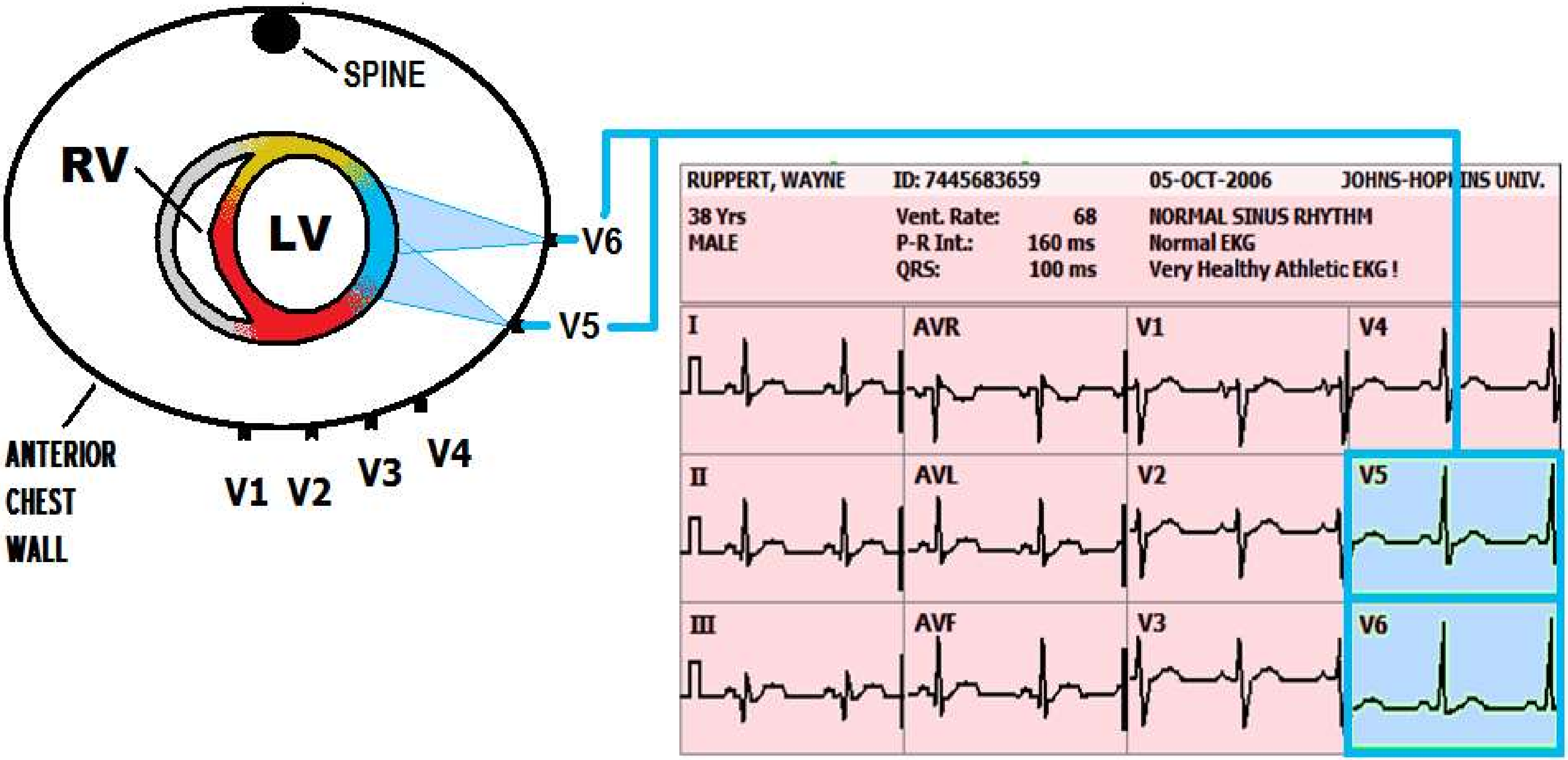
ANTERIOR
LAD
V3
LATERAL
CIRC. or LAD
V6

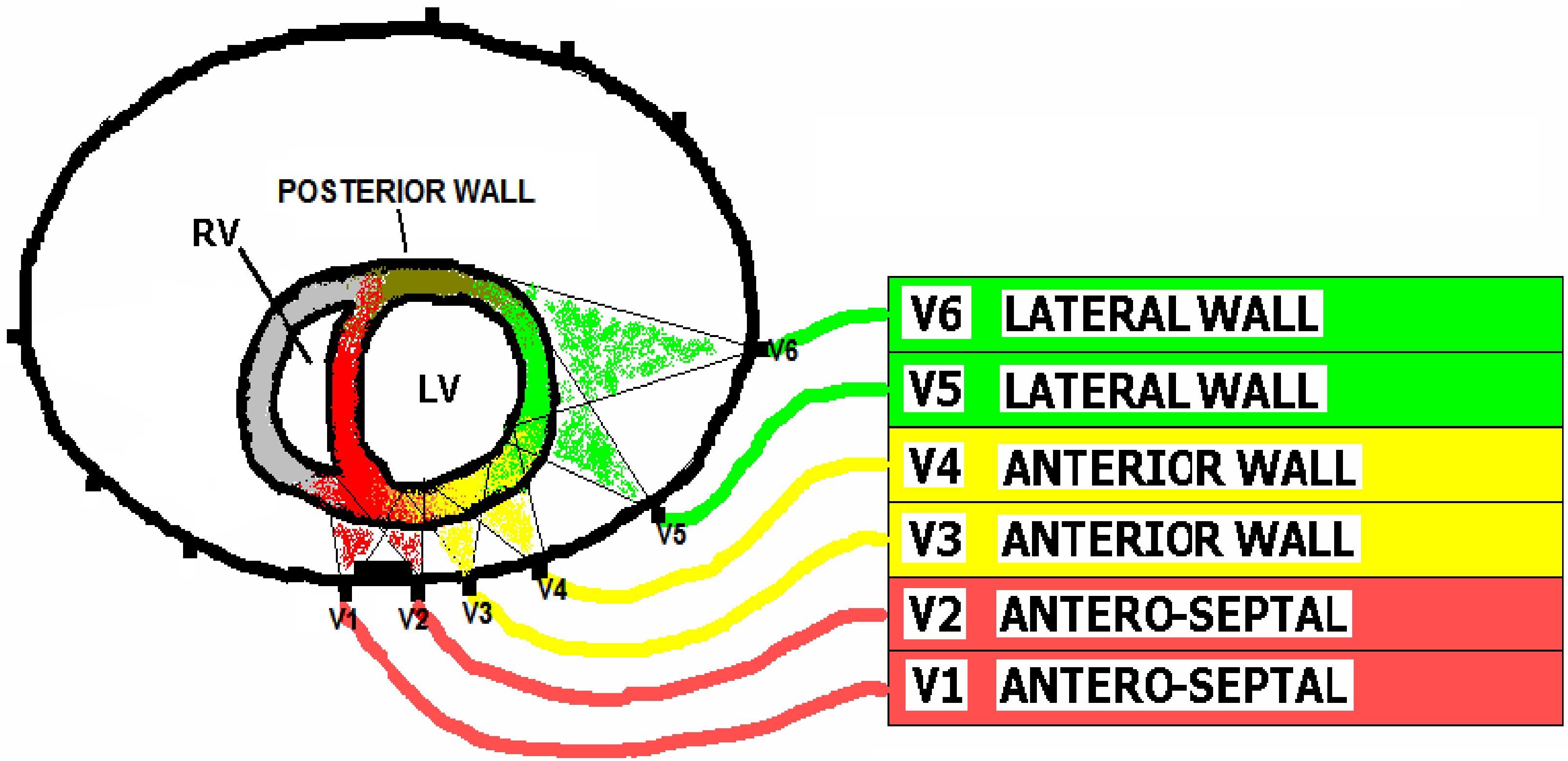
V5 - V6 VIEW THE LATERAL WALL
of the LEFT VENTRICLE



THE 12 LEAD ECG HAS TWO MAJOR BLIND SPOTS . .

CHEST LEADS V1 - V6

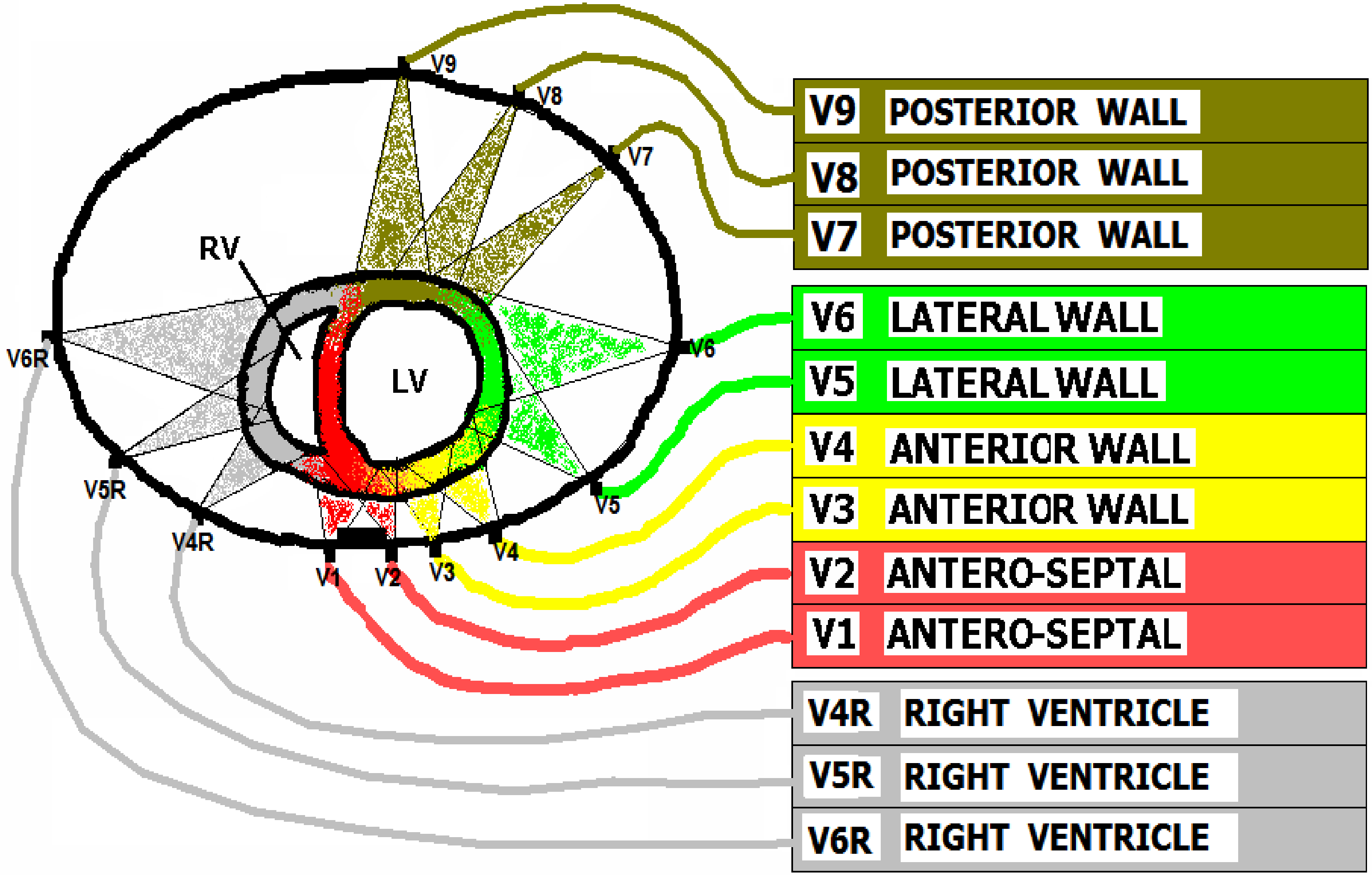
WHAT EACH LEAD "SEES" . . .



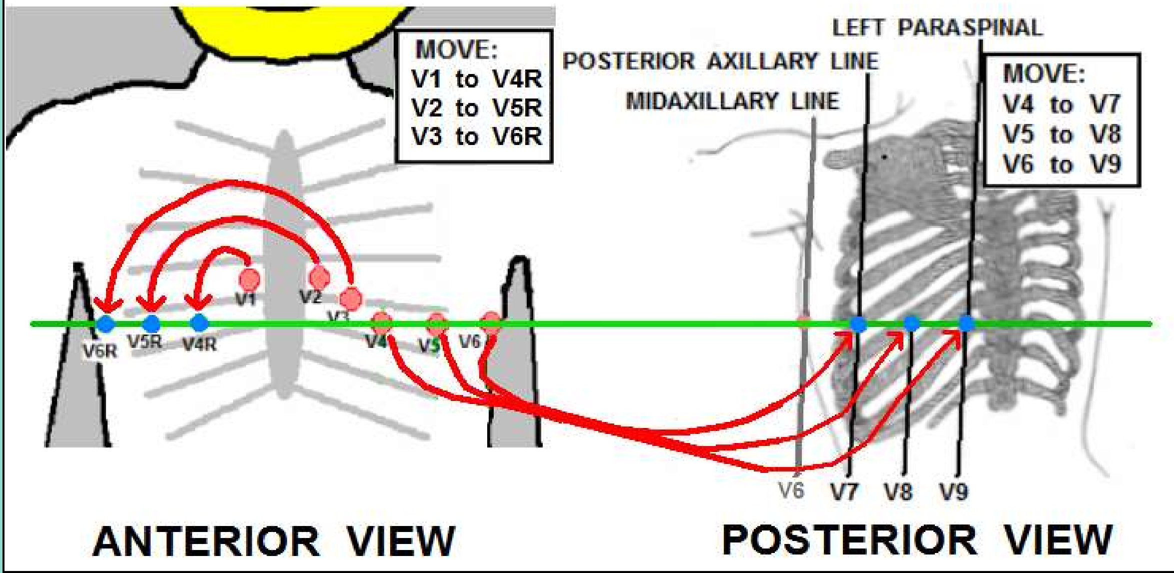
THE 18 LEAD ECG COVERS THE ENTIRE HEART . .

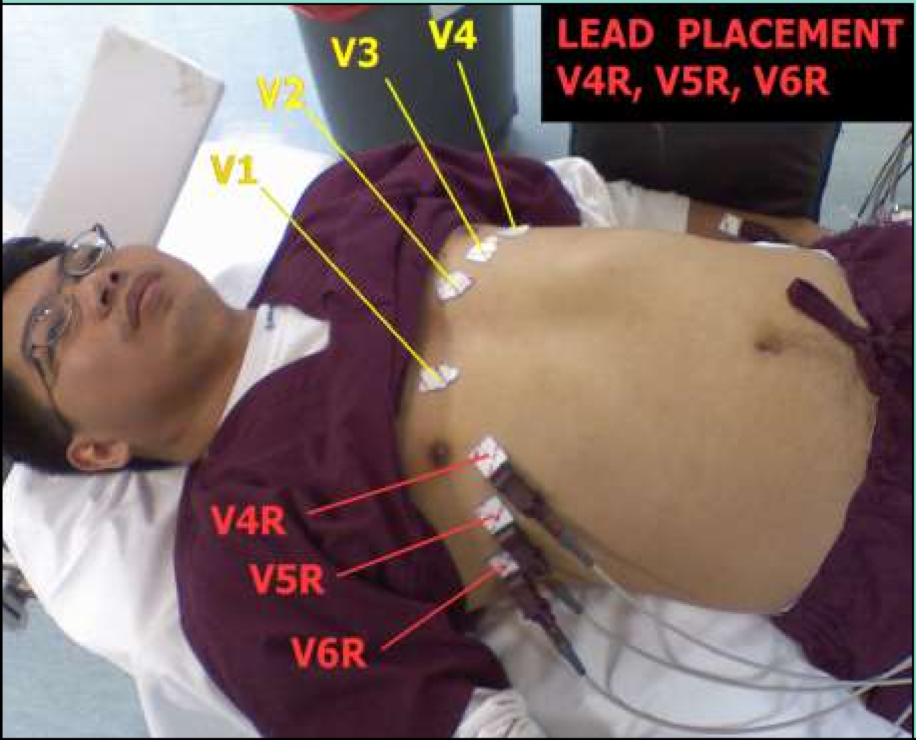
CHEST LEADS V1 - V6 *PLUS* V4R, V5R, V6R, and V7, V8, V9

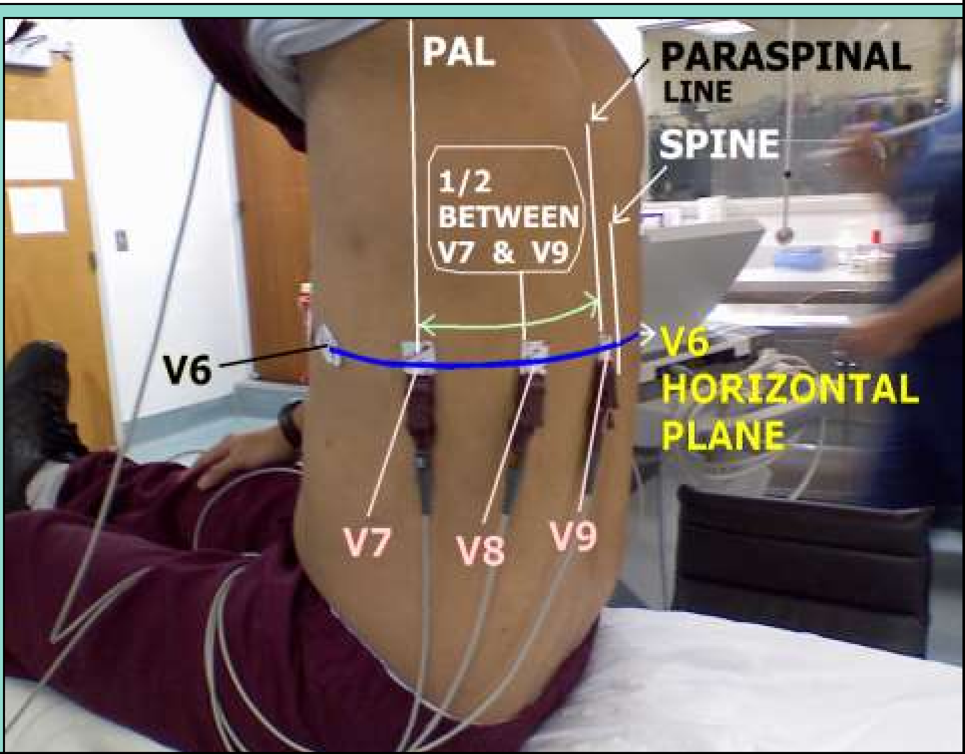
WHAT EACH LEAD "SEES" . . .



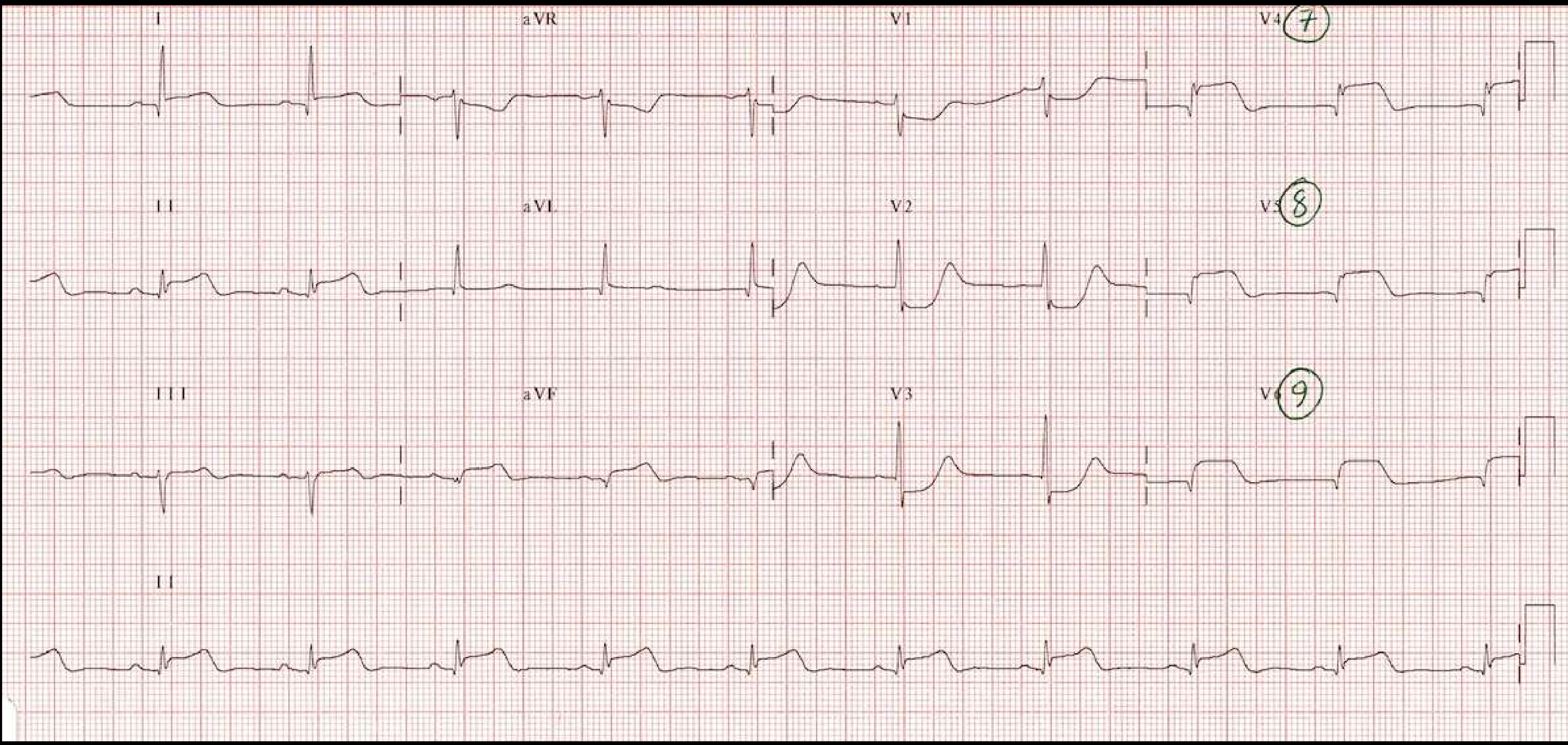
To do 18 Lead ECG with 12 Lead machine – after you obtain 12 Lead, reposition CHEST LEADS to this configuration, then print !





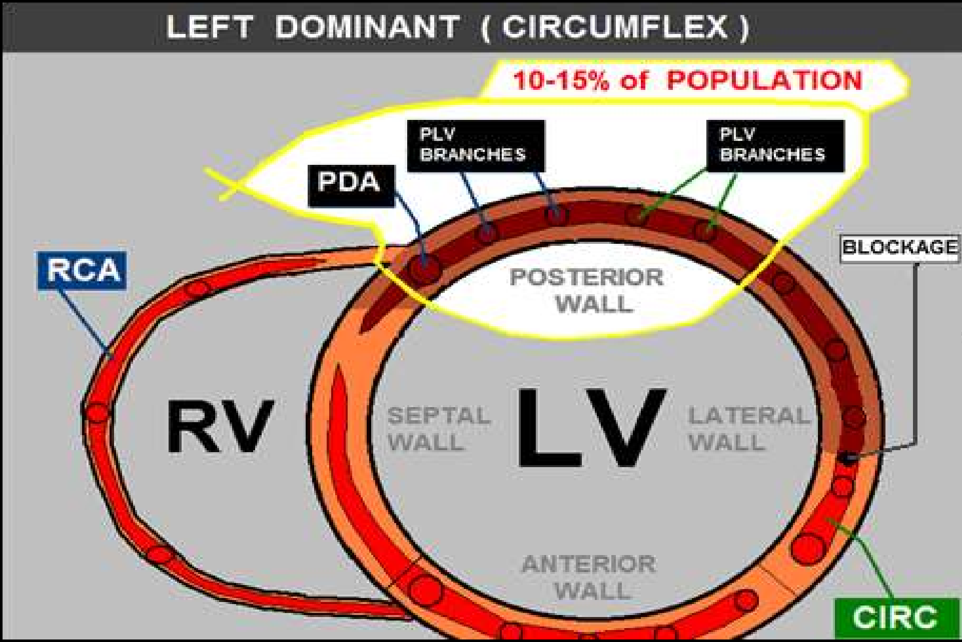


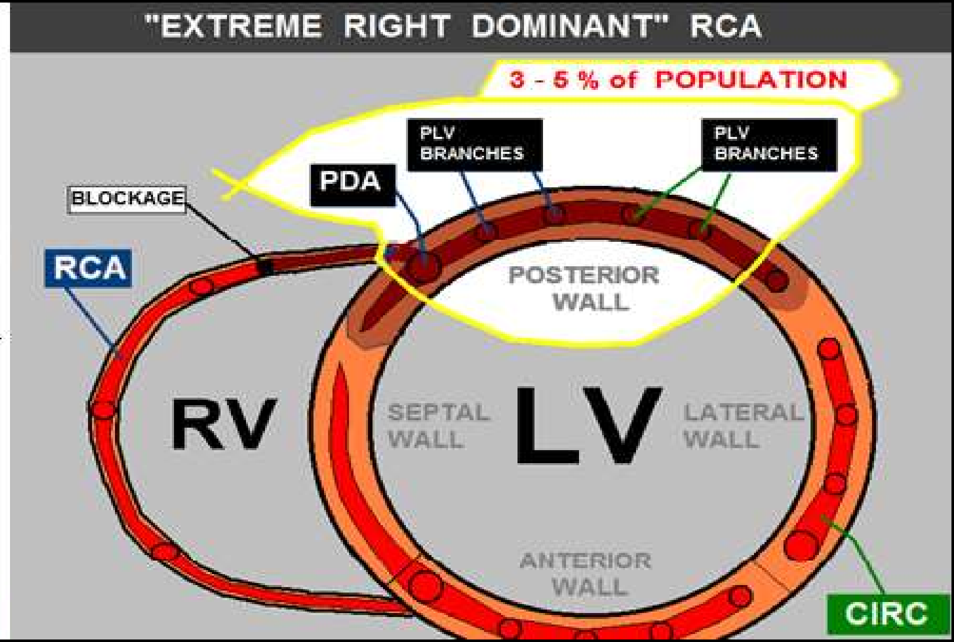
Posterior wall STEMI – ST Elevation V7 – V9

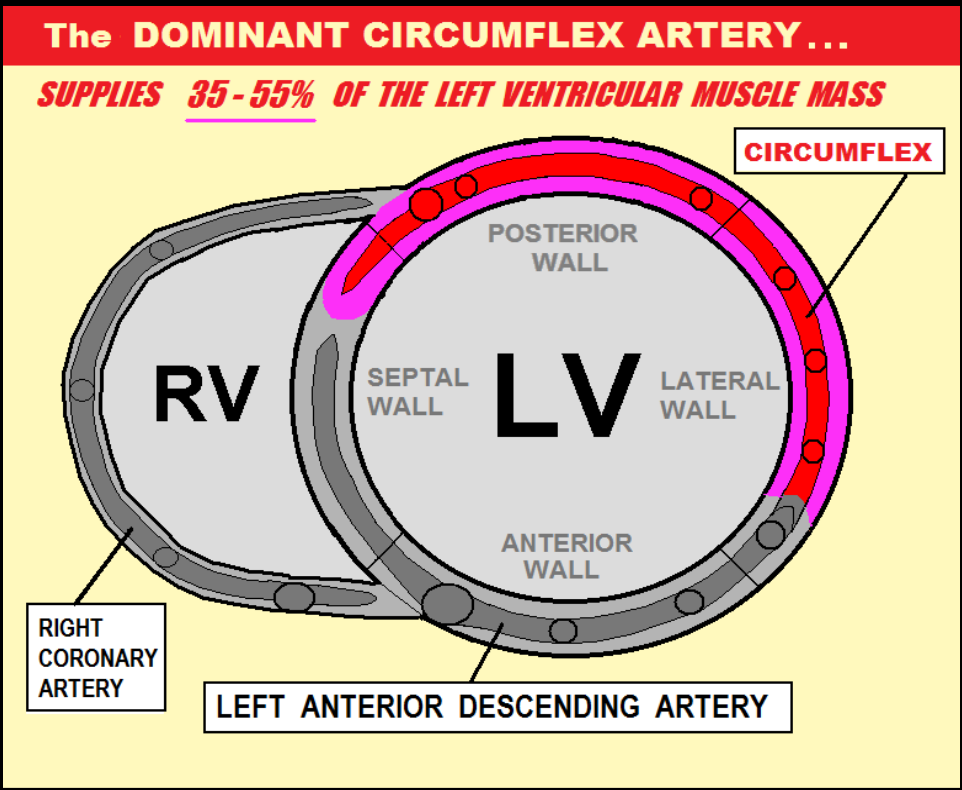


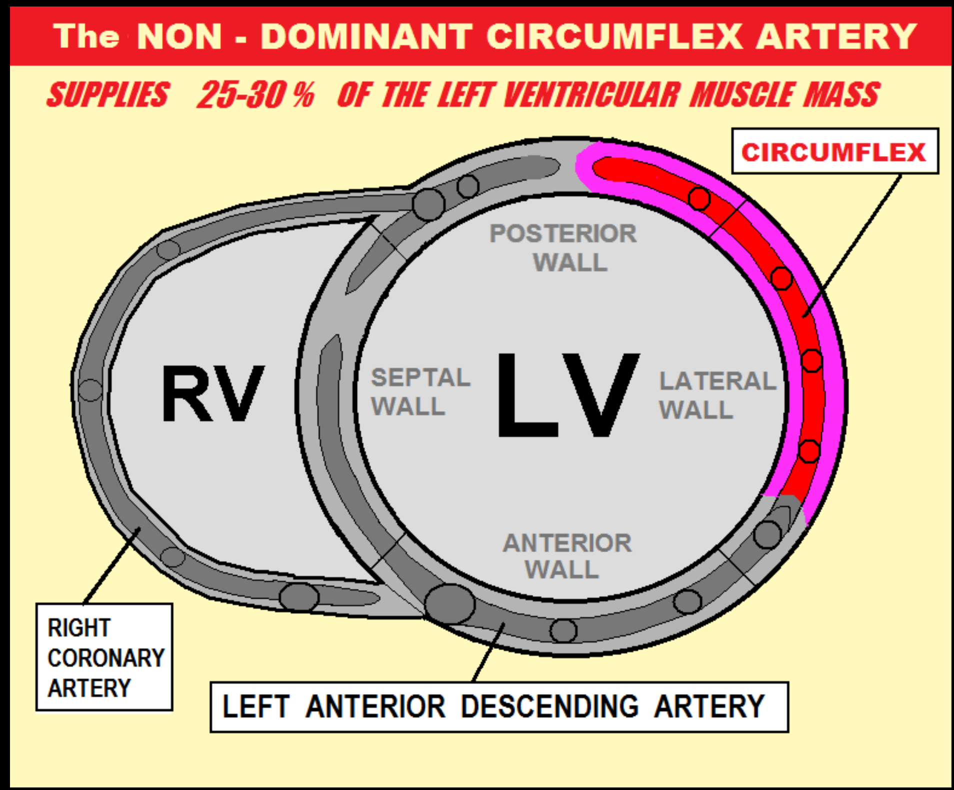
INDICATIONS for 18 Lead ECG include:

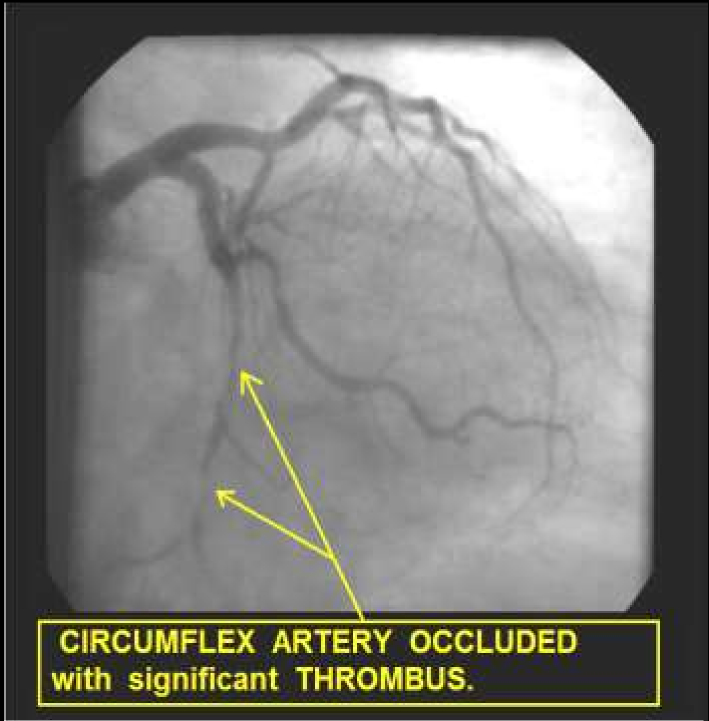
- **INFERIOR WALL MI**
- **ST Depression in LEADS V1-V4**

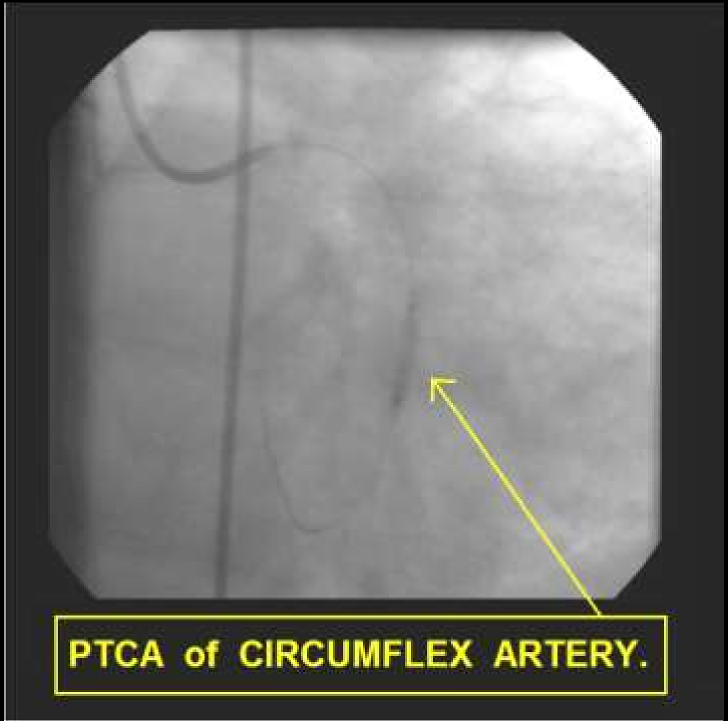

LEFT DOMINANT (CIRCUMFLEX)
10-15% of POPULATION
PLV BRANCHES
PLV BRANCHES
PDA
RCA
BLOCKAGE
POSTERIOR WALL
RV
SEPTAL WALL
LV
LATERAL WALL
ANTERIOR WALL
CIRC

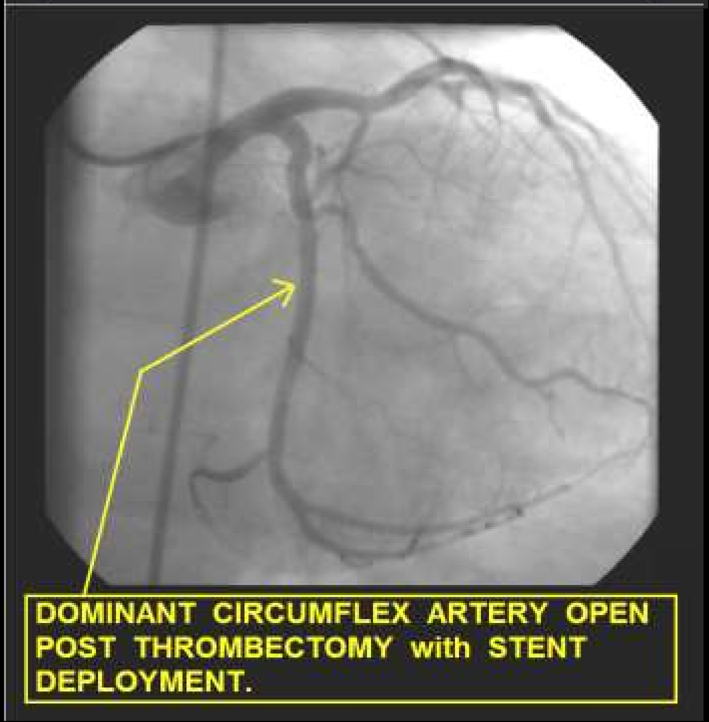

"EXTREME RIGHT DOMINANT" RCA
3 - 5 % of POPULATION
PLV BRANCHES
PLV BRANCHES
PDA
BLOCKAGE
RCA
POSTERIOR WALL
RV
SEPTAL WALL
LV
LATERAL WALL
ANTERIOR WALL
CIRC

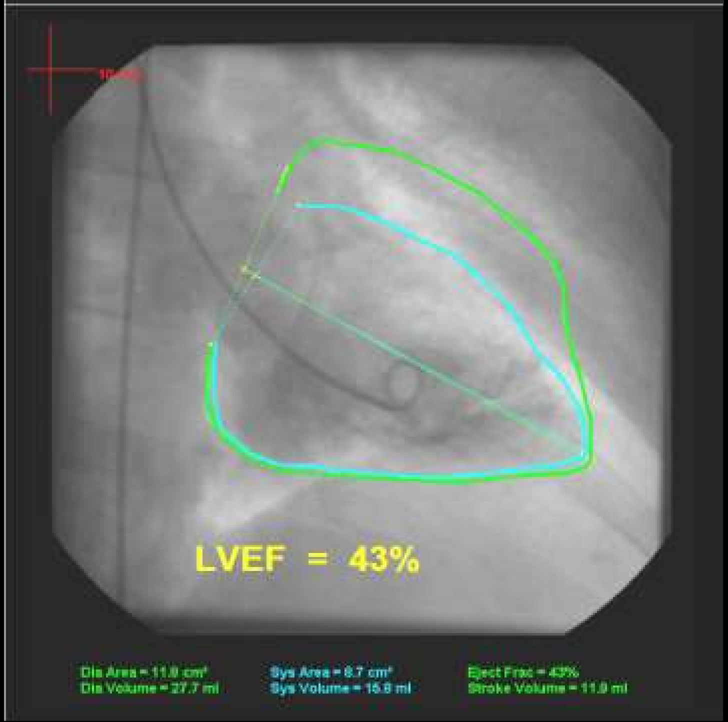

The DOMINANT CIRCUMFLEX ARTERY . . .
SUPPLIES 35 - 55% OF THE LEFT VENTRICULAR MUSCLE MASS
CIRCUMFLEX
POSTERIOR WALL
RV
SEPTAL WALL
LV
LATERAL WALL
ANTERIOR WALL
RIGHT CORONARY ARTERY
LEFT ANTERIOR DESCENDING ARTERY


The NON - DOMINANT CIRCUMFLEX ARTERY
SUPPLIES 25-30 % OF THE LEFT VENTRICULAR MUSCLE MASS
CIRCUMFLEX
POSTERIOR WALL
RV
SEPTAL WALL
LV
LATERAL WALL
ANTERIOR WALL
RIGHT CORONARY ARTERY
LEFT ANTERIOR DESCENDING ARTERY


CIRCUMFLEX ARTERY OCCLUDED
with significant THROMBUS.


PTCA of CIRCUMFLEX ARTERY.


DOMINANT CIRCUMFLEX ARTERY OPEN
POST THROMBECTOMY with STENT
DEPLOYMENT.


LVEF = 43%

YOU MADE IT !!!

Any

???

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