BRAVERA HEALTH

Bayfront Health Seven Rivers, Crystal River, FL

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Bayfront Health Spring Hill, Spring Hill, FL

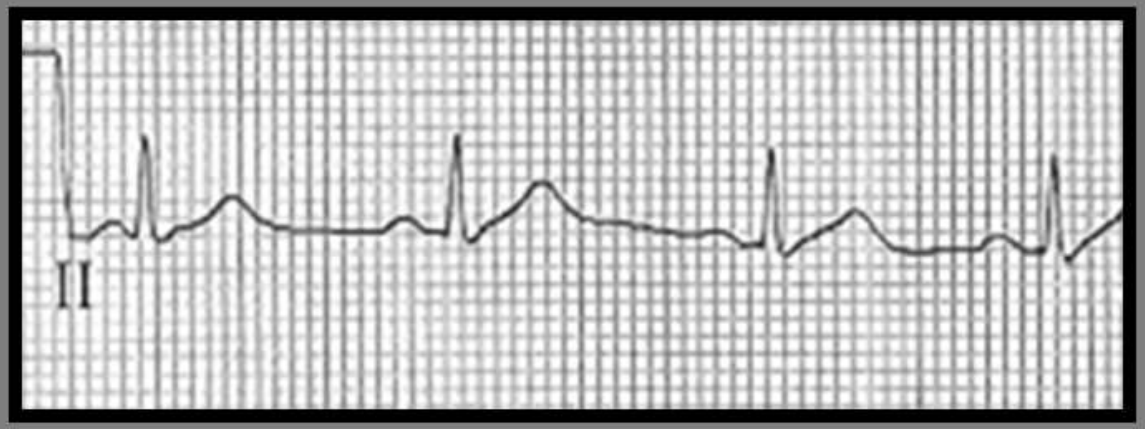
# The Lifesaving 12 Lead ECG: PCFR

**Wayne W Ruppert, CVT, CCCC, NREMT-P**
**Regional Director of Clinical Outreach &**
**Cardiovascular Accredited Programs:**
Chest Pain Center, Heart Failure

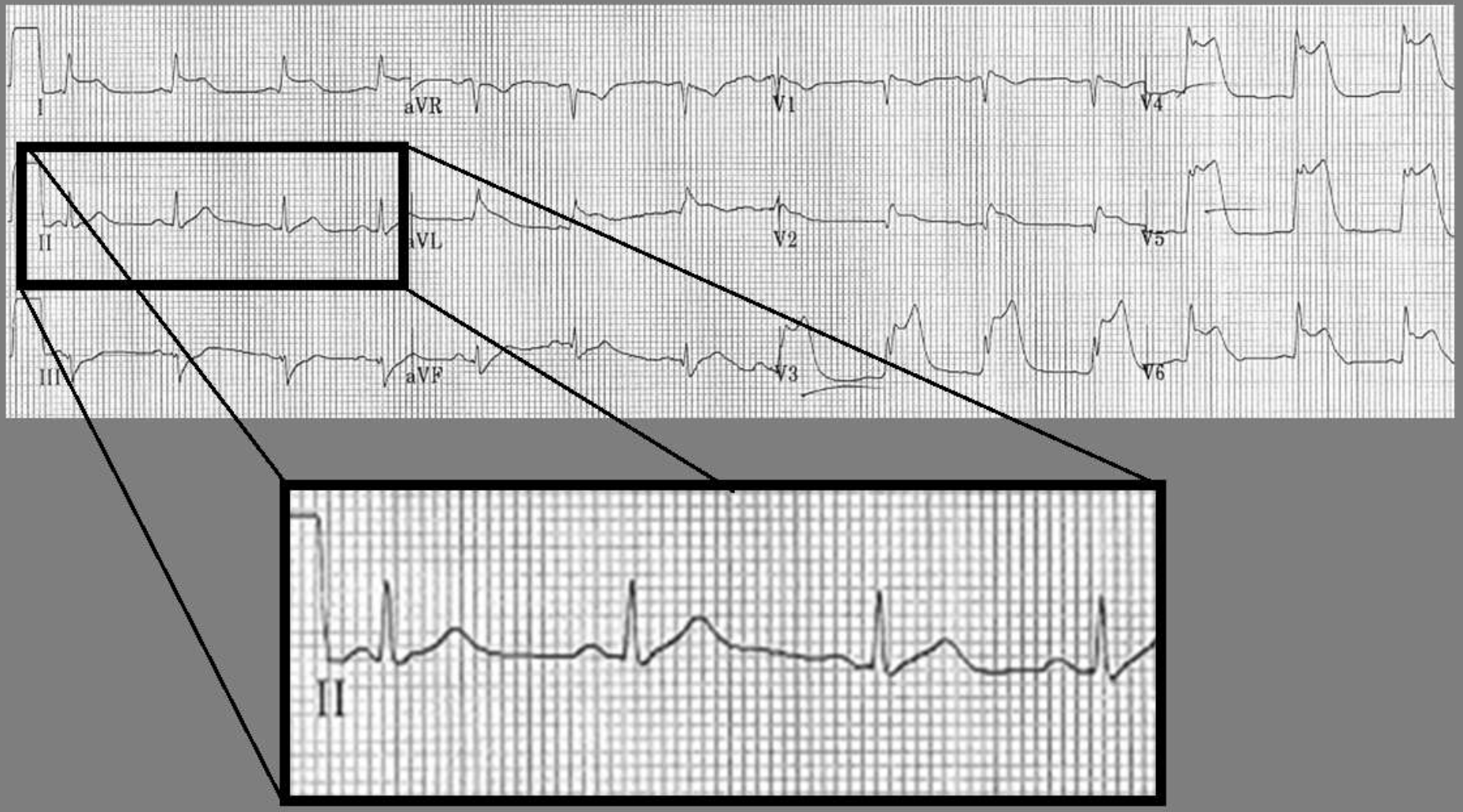
# Welcome !


Paramedics Christ Megoulas and Wayne Ruppert, Hershey, PA Fire Department, 1982

# 49 year old male - "Crushing chest pressure" . . . .



# 49 year old male - "Crushing chest pressure" . . . .



# Wayne Ruppert - Bio:

- Cardiovascular Coordinator 2012-present (coordinated 4 successful accreditations)
- Interventional Cardiovascular / Electrophysiology Technologist, 1995-Present. (Approx 13,000 patients)
- Author of: "**12 Lead ECG Interpretation in Acute Coronary Syndrome with Case Studies from the Cardiac Cath Lab**," 2010, TriGen publishing / Ingram Books
- Author of: "**STEMI Assistant**," 2014, TriGen publishing / Ingram Books
- Florida Nursing CE Provider # 50-12998
- 12 Lead ECG Instructor, 1994-present (multiple hospitals, USF College of Medicine 1994)
- Website: www.ECGtraining.org

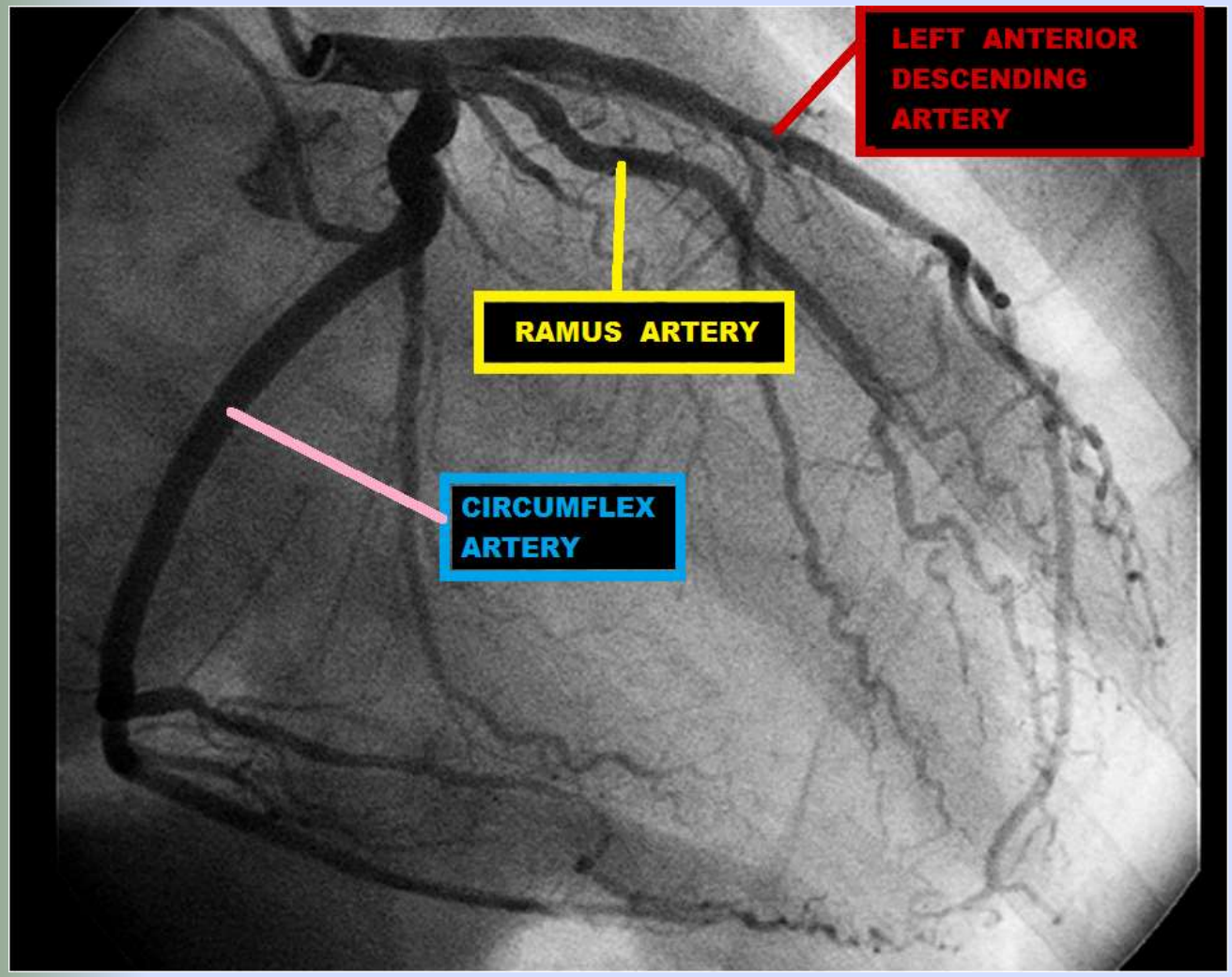
# Source of Curriculum:

- Case Studies from Cardiac Catheterization and Electrophysiology Labs, 1996 – Present

# 13,000 – 15,000 EP and Cath Lab cases between 1996 - Today

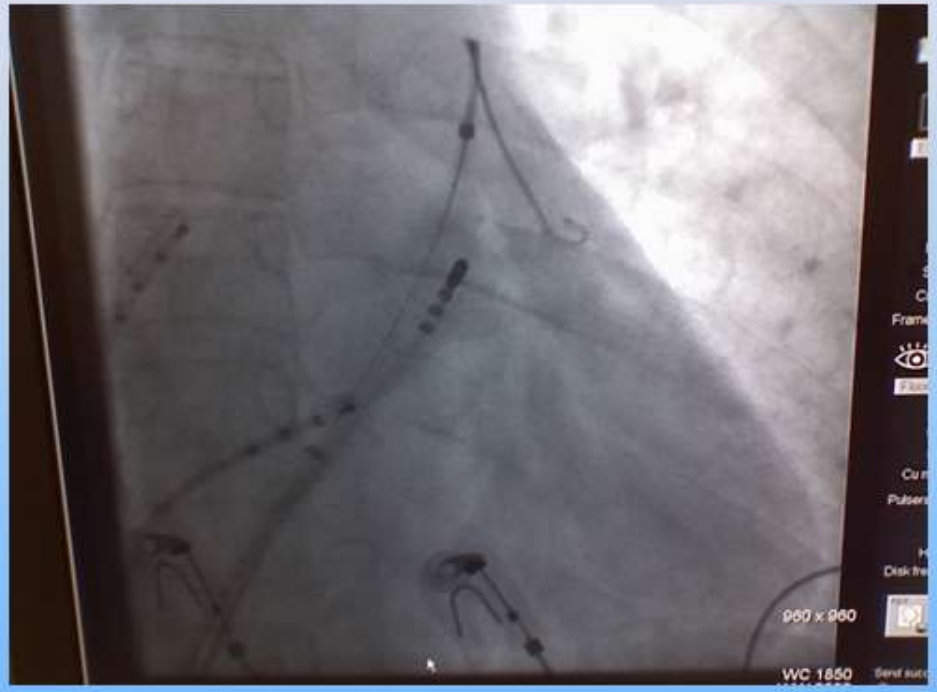

Wayne Ruppert and Dr. James Irwin, St Joseph's Hospital, Tampa, 7/29/2004

# Cardiac Cath Lab Advantage:




Correlation of ECG leads with SPECIFIC cardiac anatomic structures.

# Electrophysiology Lab Case Studies

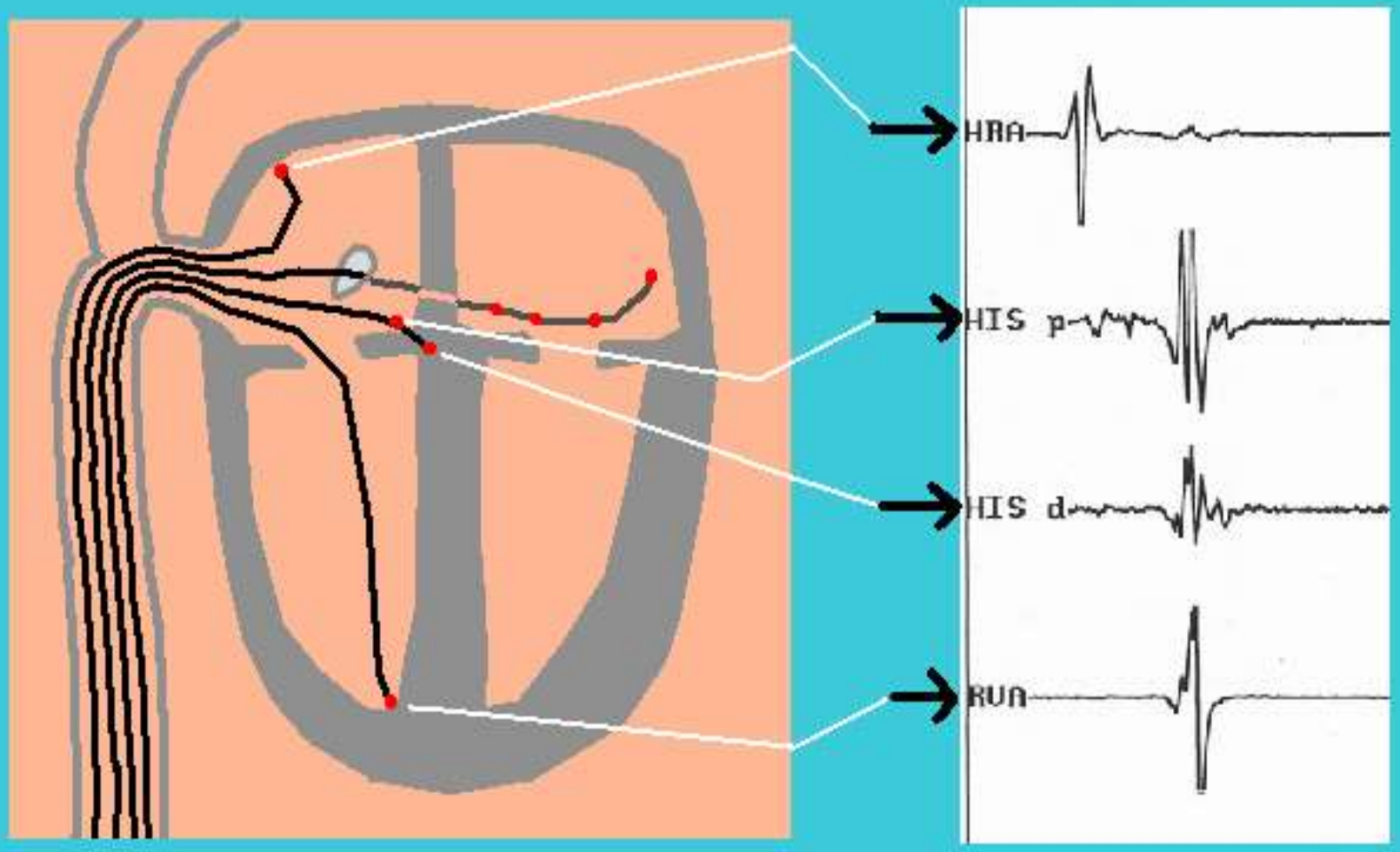


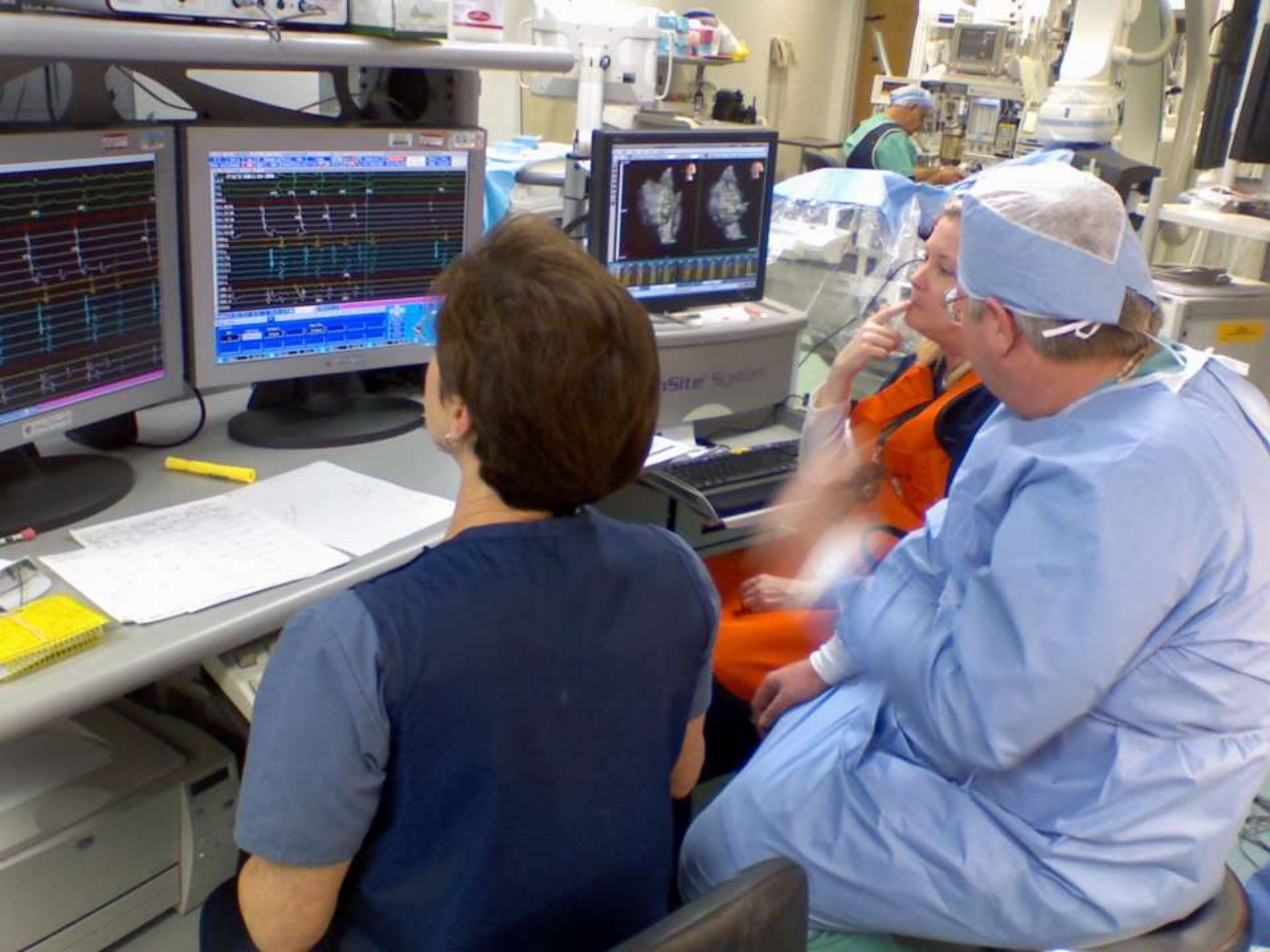

EP Catheters within the heart used for obtaining the Electrogram (the "internal ECG") Tracing and for Pace-mapping, an integral component of an EP study

Author Wayne Ruppert conducting Pace-mapping during EP study at the St Joseph's Hospital Heart Institute, Pediatric Electrophysiology Program, Tampa, FL in 2004

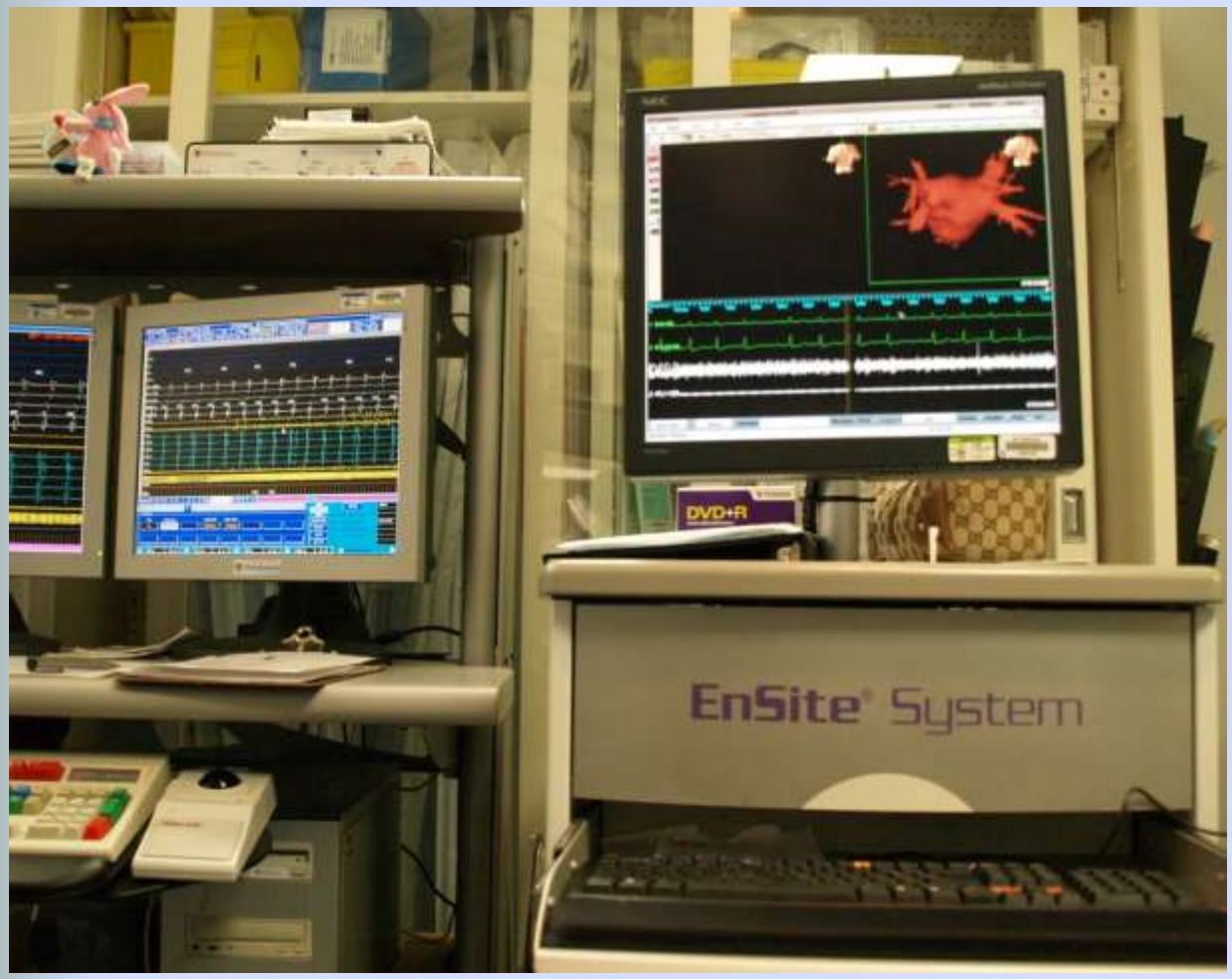
# THE ELECTROGRAM - THE INTERNAL EKG

## THE ELECTROPHYSIOLOGY ( EP ) LAB

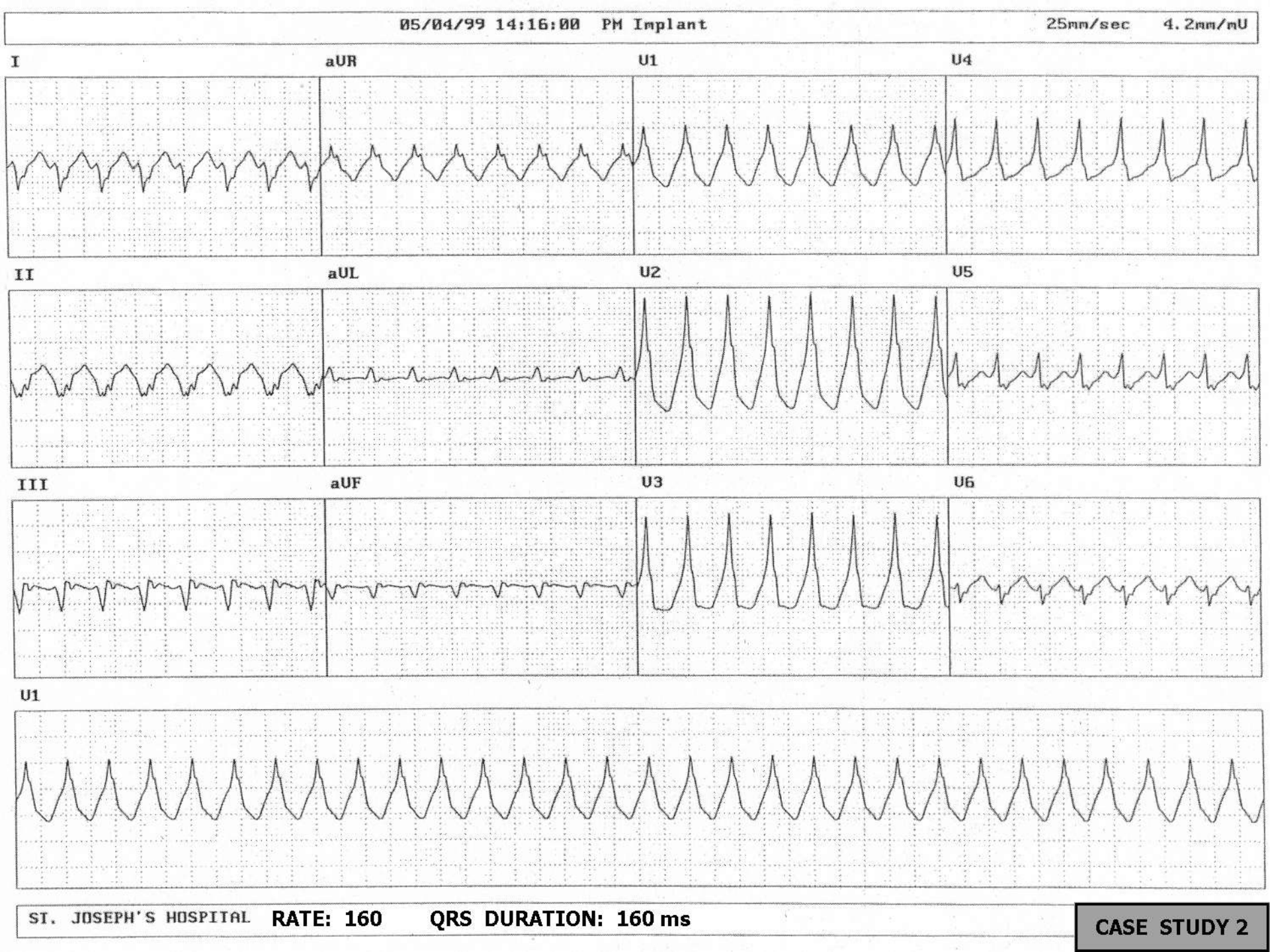


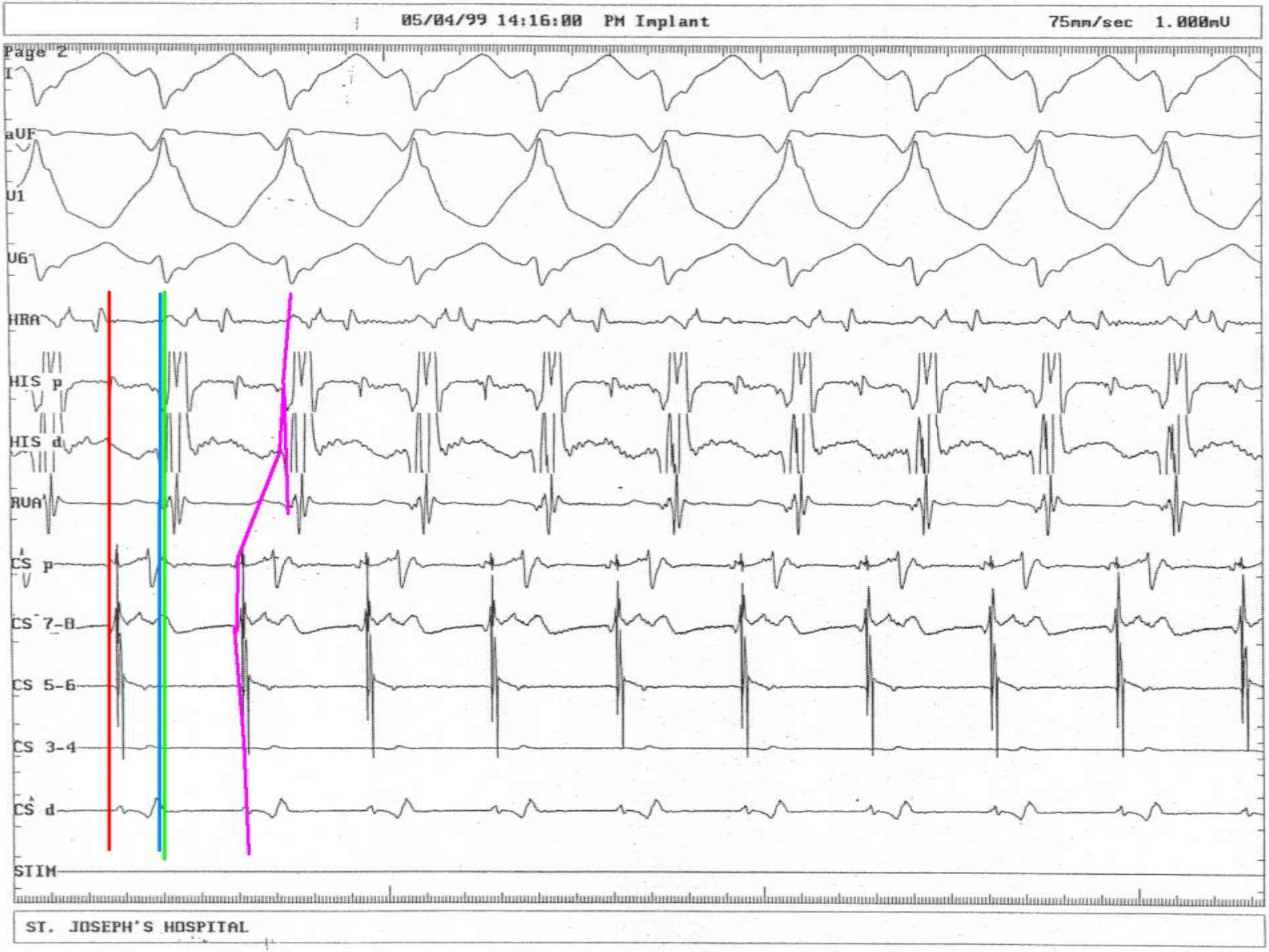


# EP Lab  Advantage:




Correlation of ECG derived diagnosis with true intra-cardiac electrogram acquired diagnosis.


05/04/99 14:16:00 PM Implant
25mm/sec 4.2mm/mV
I
aUR
U1
U4
II
aUL
U2
U5
III
aUF
U3
U6
U1
ST. JOSEPH'S HOSPITAL
RATE: 160
QRS DURATION: 160 ms
CASE STUDY 2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75mm/sec 1.000mU
Page 2
I
aVF
V1
V6
HRA
HIS p
HIS d
RVA
CS p
CS 7-8
CS 5-6
CS 3-4
CS d
STIM
ST. JOSEPH'S HOSPITAL

# Source of Curriculum:

- Case Studies from Cardiac Catheterization and Electrophysiology Labs, 1996 – Present
- Current Evidence-based Research
  - Journal of the American College of Cardiology (JACC)
  - American Heart Association (AHA) Circulation
  - ACC/AHA Guidelines
  - New England Journal of Medicine

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  - ACC/AHA Guidelines
  - New England Journal of Medicine
- Two peer reviewed, published textbooks

In the CARDIAC CATHETERIZATION LAB, we read our patients' 12 Lead ECGs and then evaluate their coronary arteries and ventricular function during angiography. Stated in plain English, *we rapidly learn how to correlate 12 lead ECG findings with what's really going on inside our patients' hearts.* Seeing ECGs from this perspective adds a new dimension to understanding the complex pathophysiologies of cardiovascular disease.

This book prepares you to:

- INTERPRET 12 Lead ECGs.
- ASSIMILATE DATA derived from the 12 Lead ECG into a comprehensive patient evaluation process designed to maximize diagnostic accuracy, while taking into consideration the 12 Lead ECGs inherent LACK of SENSITIVITY and SPECIFICITY.
- IDENTIFY 13 PATTERNS associated with myocardial ischemia and infarction, including the most subtle ECG changes often missed by clinicians and the ECG machine's computerized interpretation software.
- CORRELATE each lead of the ECG with specific regions of the heart – and the CORONARY ARTERIAL DISTRIBUTION that commonly supplies it. In cases of STEMI, this knowledge prepares you to ANTICIPATE the FAILURE OF CRITICAL CARDIAC STRUCTURES – often *BEFORE THEY FAIL.*

**For those who need to master essential material quickly, this book has been written with an expedited learning" feature, *designed to make learning as easy as 1 2 3:***

1. **READ the YELLOW HIGHLIGHTED TEXT**
2. **STUDY the GRAPHIC IMAGES, PICTURES and ECGs**
3. **CORRECTLY ANSWER the REVIEW QUESTIONS at the end of each section.**

This is an invaluable resource for every medical professional who evaluates patients and reads their 12 lead ECGs:

- *Fellows in Emergency, Cardiology, and Family Medicine*
- *Medical Residents*
- *Veteran Physicians wanting a good review in ACS patient evaluation*
- *Physician Assistants and Nurse Practitioners*
- *Emergency Department Nurses*
- *Coronary Care Unit and Cardiac Telemetry Nurses*
- *Walk-in Clinic Physicians and Nurses*
- *Paramedics*

*"I think this book will be a wonderful addition to the textbooks that are already available, with a fresh perspective'"*

**Joseph P. Ornato, MD, FACP, FACC, FACEP**
- Professor and Chairman, Department of Emergency Medicine
Medical College of Virginia/Virginia Commonwealth University
- Medical Director, Richmond Ambulance Authority,
Richmond, Virginia

*"This book integrates academic ECG principles with real-world clinical practice by incorporation of well chosen cath lab case studies into its curriculum. This combination lets readers see patients and their ECGs through the eyes of an experienced cath lab interventionalist, and provides a balanced approach to patient evaluation that compensates for the ECGs inherent lack of sensitivity and specificity. I highly recommend this book for all Emergency Medicine and Cardiology Fellows. For experienced clinicians, it's a superb review."*

**Humberto Coto, MD, FACP, FACC**
- Chief of Interventional Cardiology
St. Joseph's Hospital
Tampa, Florida

ISBN 978-0-9829172-1-3
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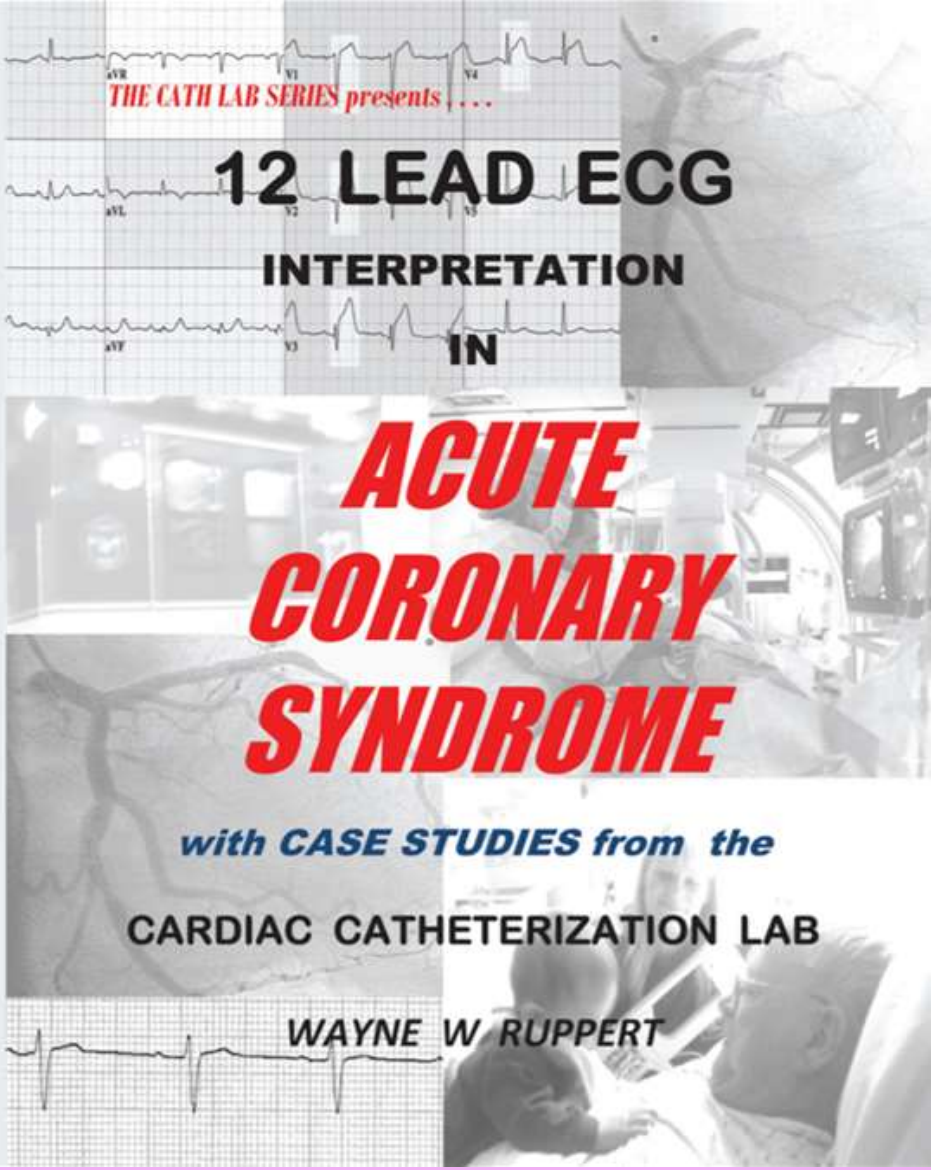
9 780982 917213

12 LEAD ECG INTERPRETATION in ACUTE CORONARY SYNDROME with CASE STUDIES from the CATH LAB – WAYNE RUPPERT




[Amazon.com](#)

[Barnes and Noble](#)

# TEXTBOOK REVIEWED BY:

Joseph P. Ornato, MD, FACP, FACEP, FACC, Professor and Chairman, Department of Emergency Medicine, Medical College of Virginia-Virginia Commonwealth University

Humberto Coto, MD, FACP, FACC, Chief of Cardiology, St. Joseph's Hospital

Matthew Glover, MD, FACP, FACC, Interventional Cardiologist, St. Joseph's Hospital

Xavier Prida, MD, FACP, FACC, Interventional Cardiologist, St. Joseph's Hospital

Charles Sand, MD, FACP, FACEP, Emergency Department Physician, St. Joseph's Hospital

Printed and Marketed Worldwide by The Ingram Book Company
2010 - Current

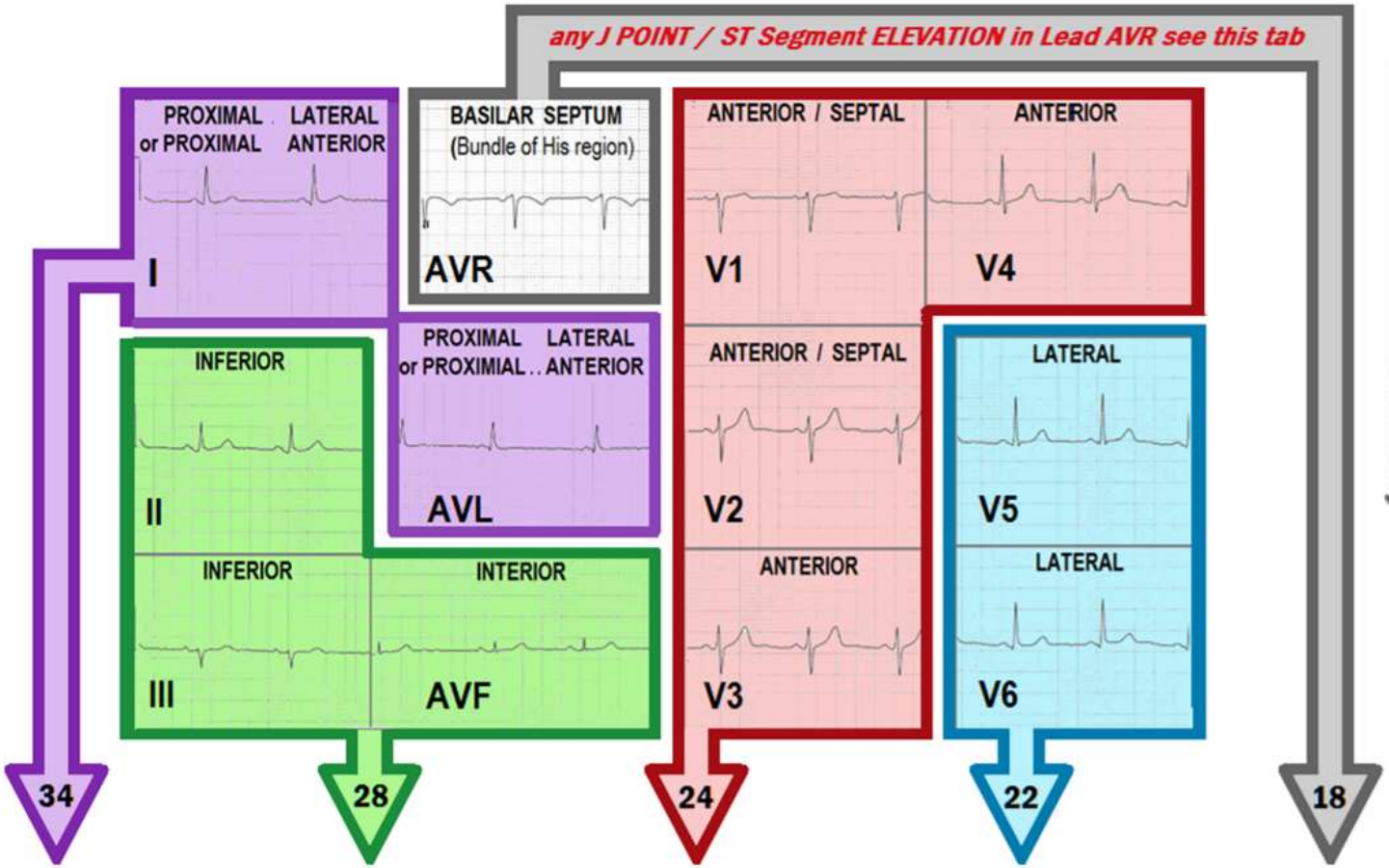
# STEMI Assistant

by Wayne Ruppert

**UNIVERSAL ACS PATIENT MANAGEMENT ALGORITHM**
**- - - See PAGE ONE - - -**

**Select LEAD SET with HIGHEST ST ELEVATION and open to associated page . . .**

CRASH CART EMERGENCY REFERENCE




Bayfront Health Dade City

# TEXTBOOK REVIEWED BY:

Barbra Backus, MD, PhD Inventor of "The HEART Score," University Medical Center, Utrech, Netherlands

Michael R. Gunderson, National Director, Clinical and Health IT, American Heart Association

Anna Ek, AACC, BSN, RN Accreditation Review Specialist, The American College of Cardiology

William Parker, PharmD, CGP, Director of Pharmacy, Bayfront Dade City

Printed and Marketed Worldwide by The Ingram Book Company
2010 - Current

# STEMI Assistant

Tutorial Video

Free download – electronic copy (PDF file)

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Wayne.ruppert@braverahealth.com

# The Lifesaving 12 Lead ECG Course:

**Is a condensed curriculum focused on acute conditions which are associated with a high degree of morbidity and mortality:**

# The Lifesaving 12 Lead ECG Course:

**Is a condensed curriculum focused on acute conditions which are associated with a high degree of morbidity and mortality:**

1. **Acute Coronary Syndromes**
   - **STEMI (pre-infarction, acute & evolving / old MI)**
   - **NSTEMI**
   - **Unstable Angina**
   - **Low Risk Chest Pain**

# The Lifesaving 12 Lead ECG Course:

**Is a condensed curriculum focused on acute conditions which are associated with a high degree of morbidity and mortality:**

2. **Sudden Cardiac Death Syndromes**
   - **Long QT Syndrome (Congenital & Drug Induced)**
   - **Brugada Syndrome**
   - **Cardiomyopathy (Hypertrophic and other)**
   - **Arrhythmogenic Right Ventricular Dysplasia**
   - **Wolff-Parkinson-White Syndrome**
   - **Catecholinergic Polymorphic Ventricular Tachy.**

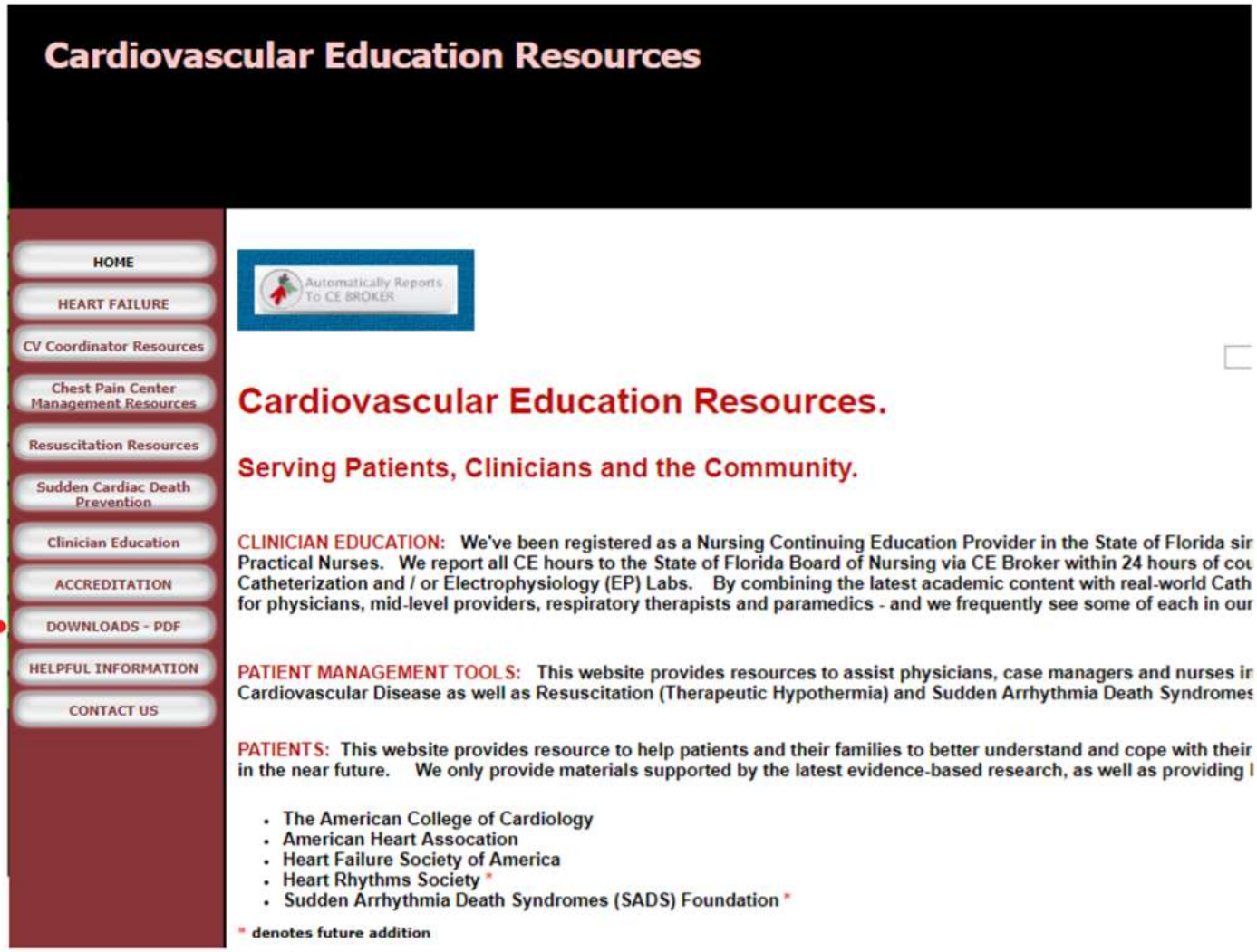
# Helpful Web Resources:

www.practicalclinicalskills.com

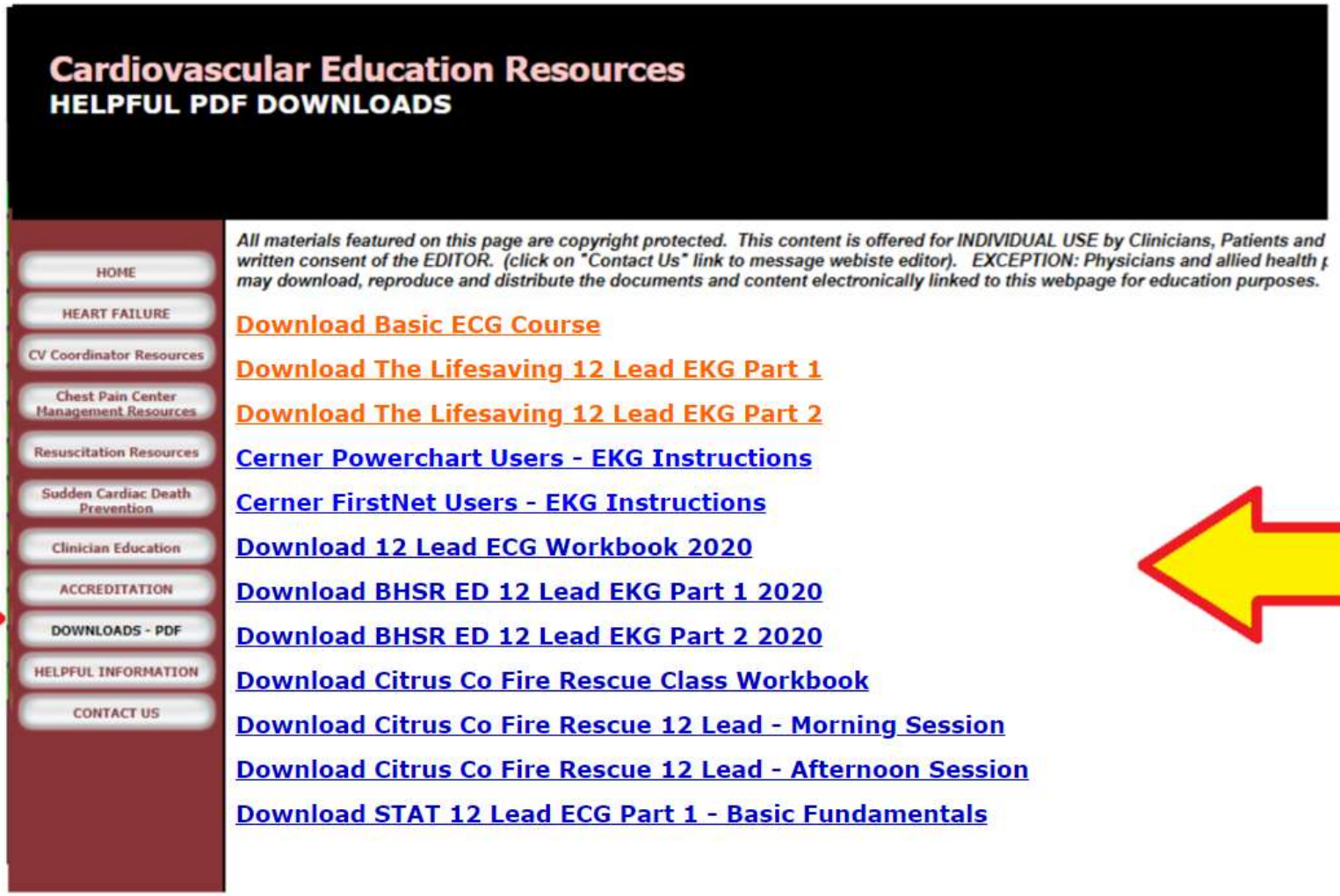
www.skillstat.com/tools/ecg-simulator

www.ECGtraining.org

1. Go to: www.ECGtraining.org
2. Select "Downloads PDF" from menu bar



1. Go to: www.ECGtraining.org
2. Select "Downloads PDF" from menu bar
3. Select your courses



# The EKG in PERSPECTIVE

1. Much development in the 1950s and 60s, and at that time, EKGs were the primary diagnostic tool.

2. Today we have better diagnostic tools (e.g. ECHO, CARDIAC CATH, EP STUDIES) that sometimes conflict with traditional EKG-made diagnoses.

3. Some EKG findings are more accurate and reliable than others .

***AND . . .***

# *Sometimes, ECGs LIE to us !*

# *ECGs and USED CAR SALESMEN often have MUCH in common !*

# The EKG in PERSPECTIVE

## PROBLEMS WITH EKGs . . .

↓ SENSITIVITY

( FALSE NEGATIVES )

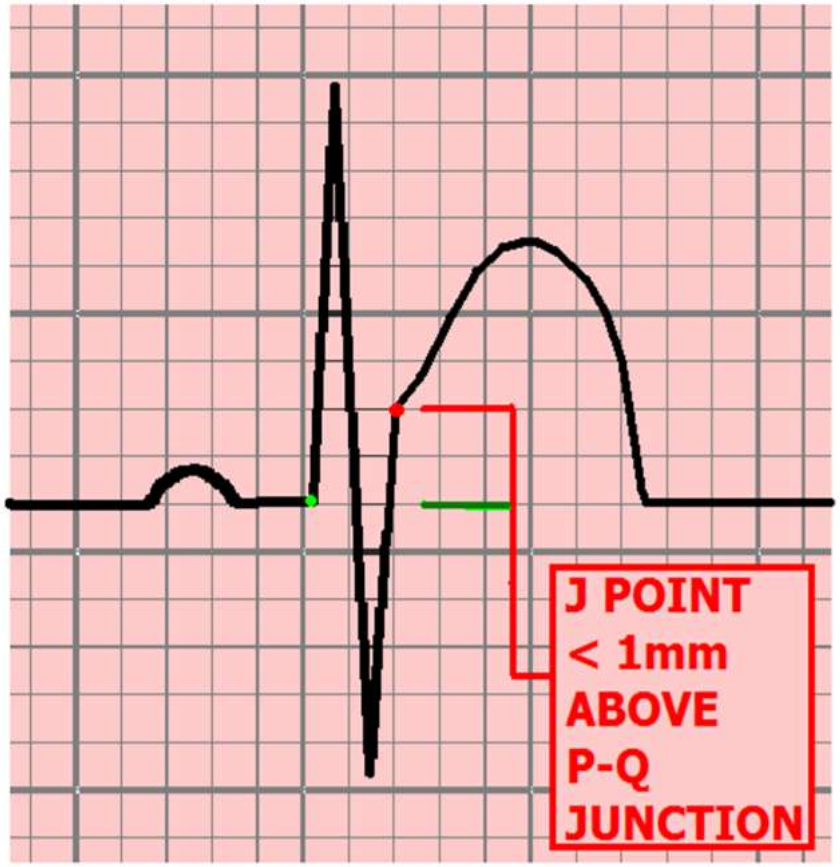
↓ SPECIFICITY

( FALSE POSITIVES )

*AND . . .*

# PROBLEMS WITH SPECIFICITY . . .

## S-T SEGMENT ELEVATION - COMMON ETIOLOGIES:




CONDITION:

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

# ST-Segment Elevation in Normal Circumstances and in Various Conditions

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

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

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

The NEW ENGLAND JOURNAL of MEDICINE

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

1North (06)

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

--AXIS--

| | |
|---|---|
| P | 59 |
| QRS | 14 |
| T | 33 |

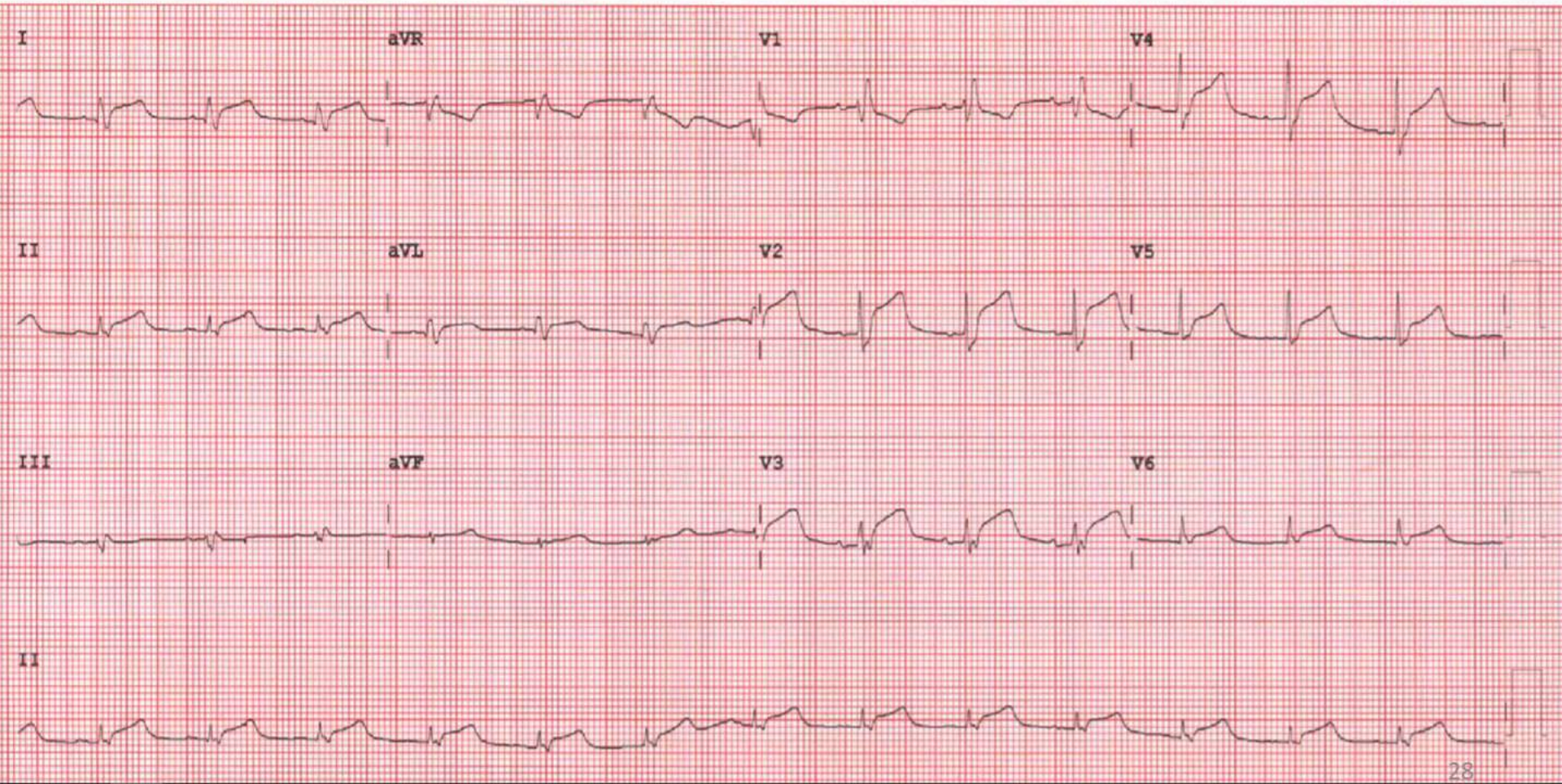
12 Lead; Standard Placement

- ABNORMAL ECG -

>>> Acute MI <<<

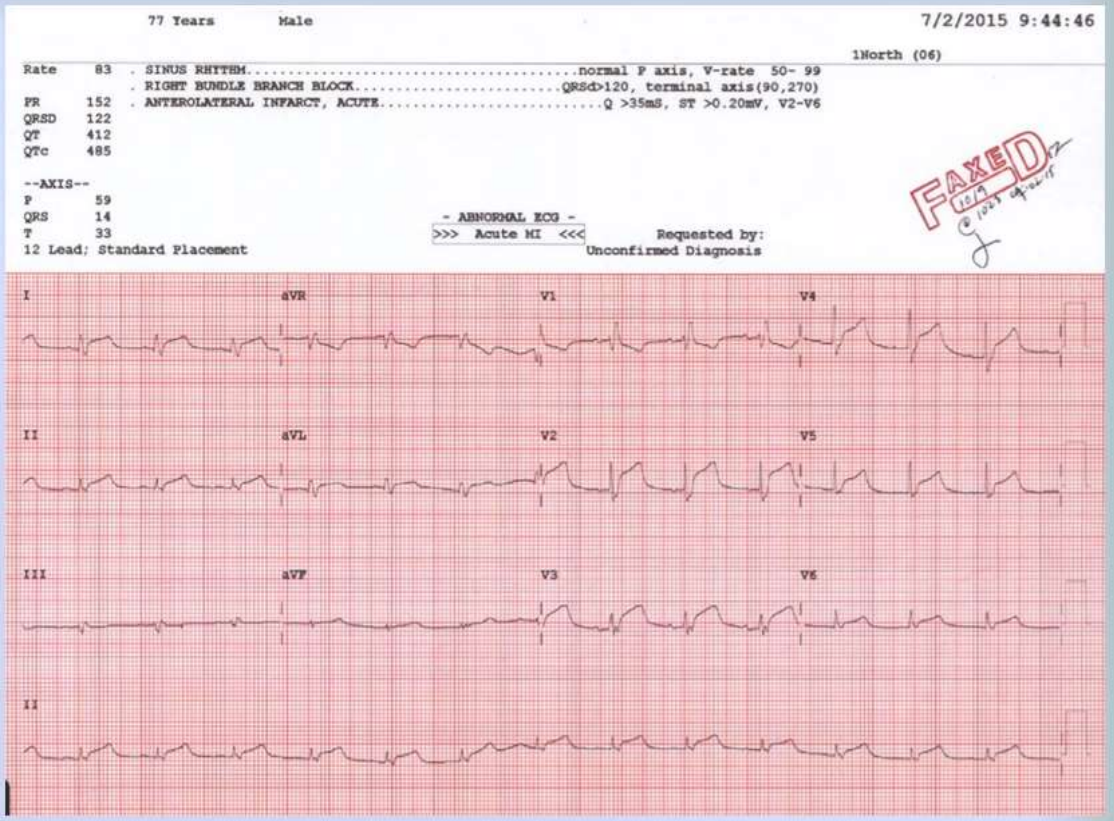
Requested by:
Unconfirmed Diagnosis

FAXED 10/9 @ 1023 cf 10.2.15



**Patient:**

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



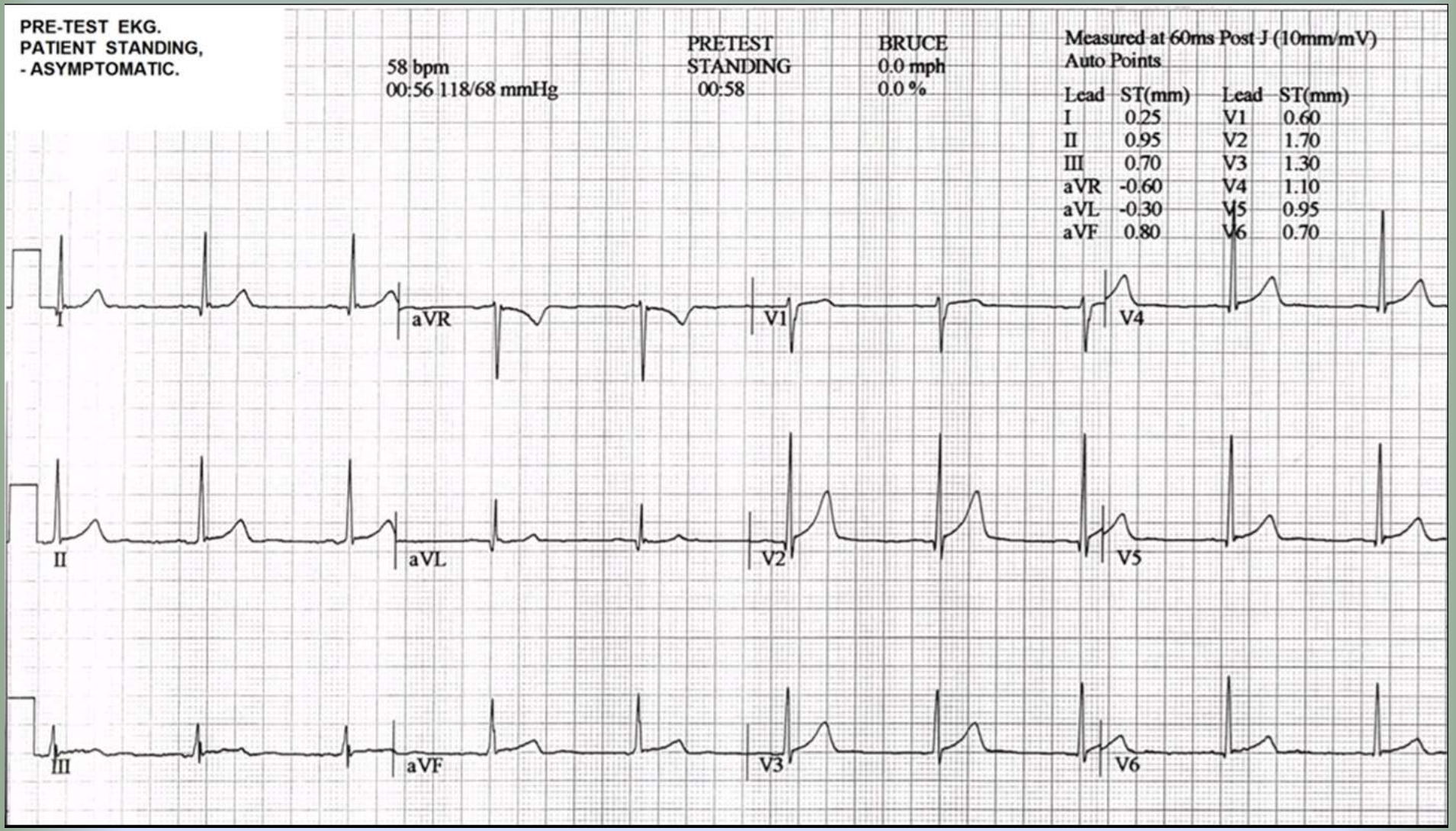

**EARLY REPOLARIZATION. This degree of ST Elevation in early repolarization is VERY RARE: The only such ECG I have seen in approximately 13,000 cardiac catheterizations.**

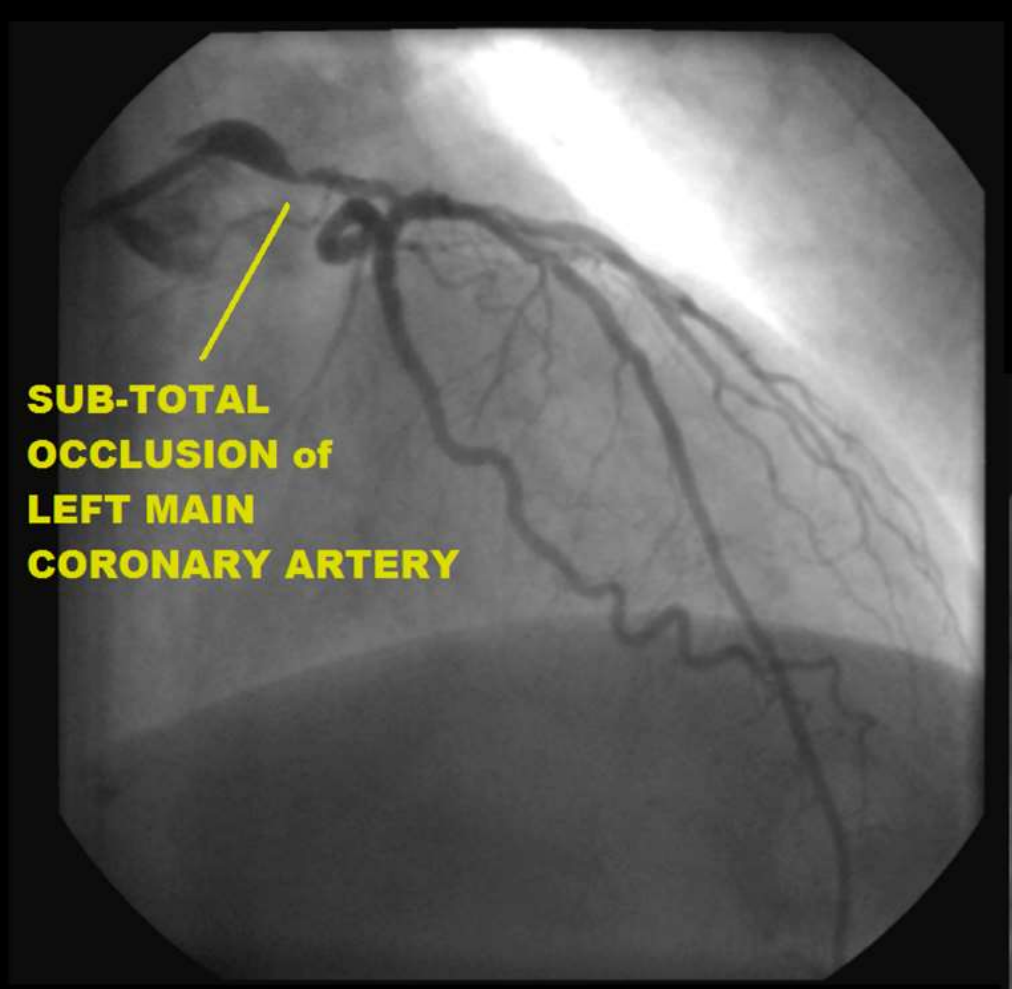
# EKGs in PERSPECTIVE, con't:

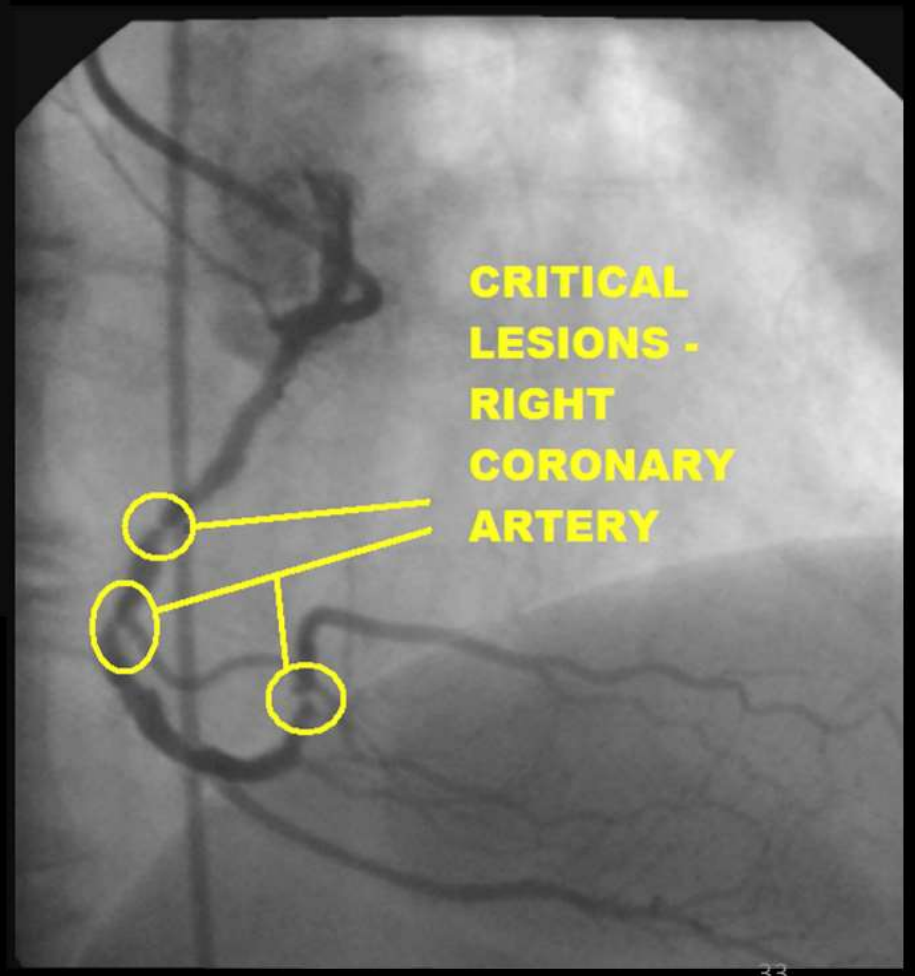
**One of the MOST MISLEADING scenarios of all is when the EKG APPEARS PERFECTLY NORMAL . . .**

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

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

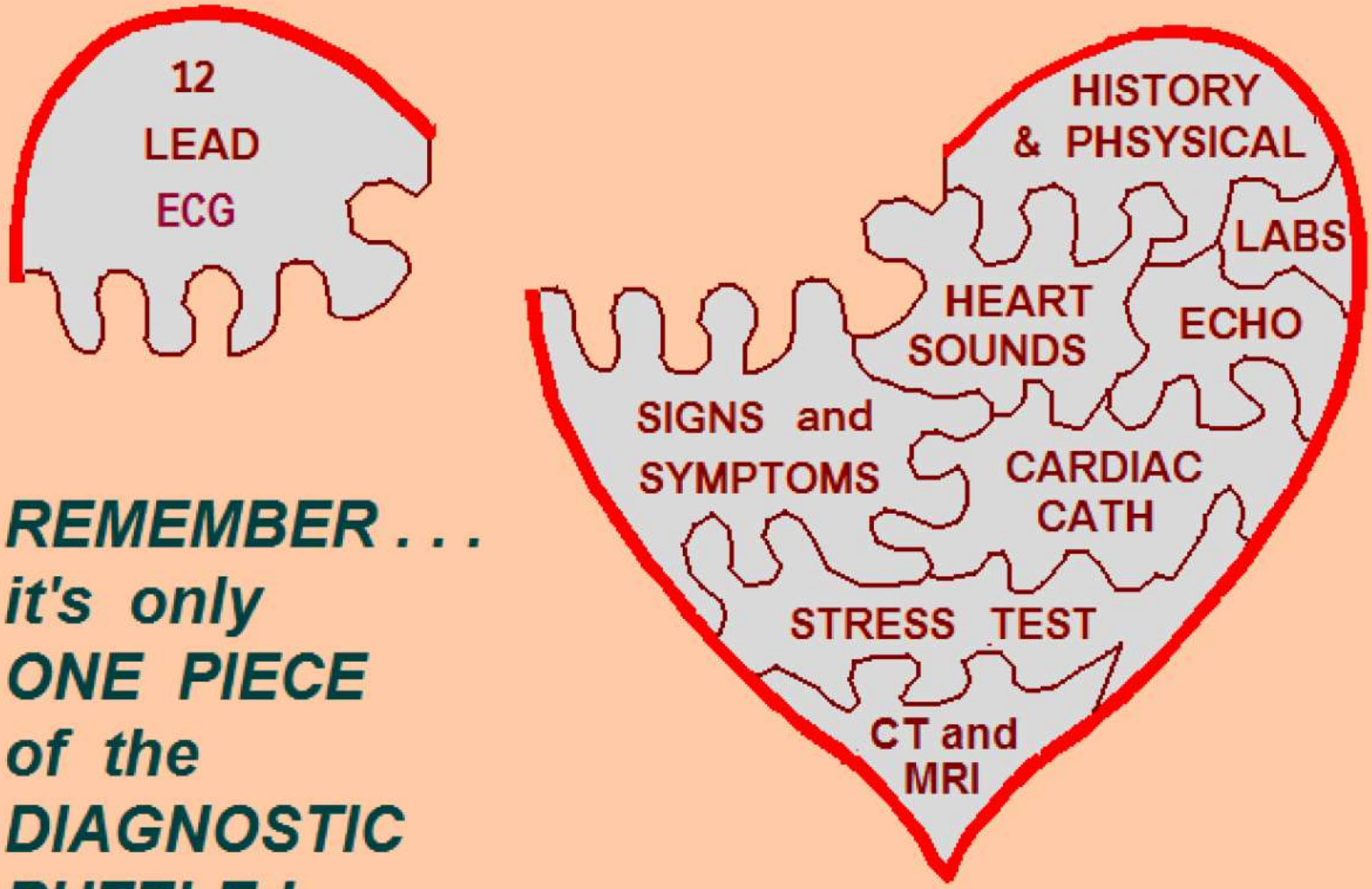

PRE-TEST EKG.
PATIENT STANDING,
- ASYMPTOMATIC.
58 bpm
00:56 118/68 mmHg
PRETEST
STANDING
00:58
BRUCE
0.0 mph
0.0 %
Measured at 60ms Post J (10mm/mV)
Auto Points
Lead ST(mm) Lead ST(mm)
I 0.25 V1 0.60
II 0.95 V2 1.70
III 0.70 V3 1.30
aVR -0.60 V4 1.10
aVL -0.30 V5 0.95
aVF 0.80 V6 0.70
I
aVR
V1
V4
II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


SUB-TOTAL
OCCLUSION of
LEFT MAIN
CORONARY ARTERY


CRITICAL
LESIONS -
RIGHT
CORONARY
ARTERY

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

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




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

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

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

# HEART

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

***Risk factors for atherosclerotic disease:**

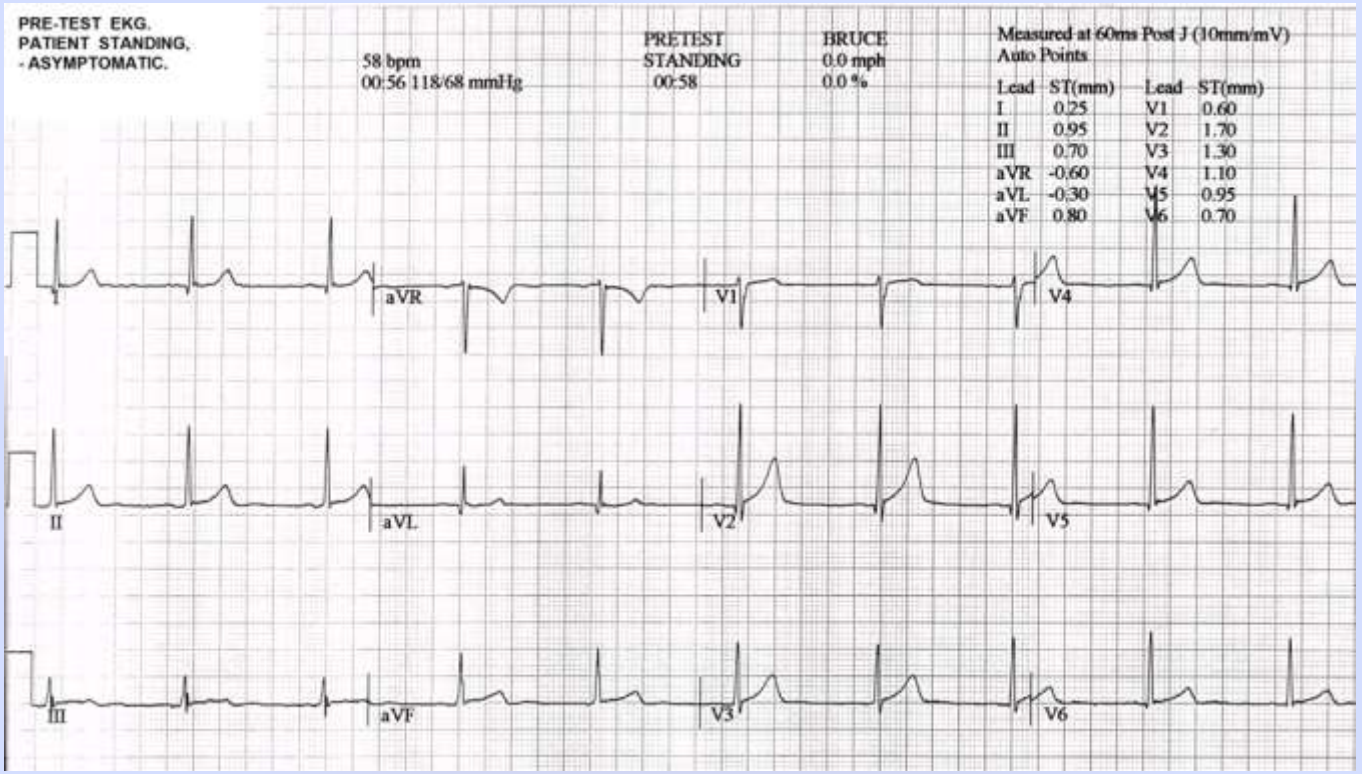
| | |
|---|---|
| Hypercholesterolemia | Cigarette smoking |
| Hypertension | Positive family history |
| Diabetes Mellitus | Obesity |

# HEART Score

- Used in cases where “you’re NOT SURE if this is CARDIAC - or - SOMETHING ELSE.”
- NOT used for OBVIOUS Cardiac cases
- Per Dr. Barbara Backus, HEART Score Inventor:
  - “if you calculate a HEART Score during STEMI, you are WASTING VALUABLE TIME !”

# HEART Score

CASE STUDY that's PERFECT for the HEART Score:




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

# H = HISTORY

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

# 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

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

# R = Risk Factors for CAD

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

# RISK FACTORS

## for the development of

## CORONARY ARTERY DISEASE:

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

per the AMERICAN HEART ASSOCIATION

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

# T = Troponin

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

# HEART

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

***Risk factors for atherosclerotic disease:**

| | |
|---|---|
| Hypercholesterolemia | Cigarette smoking |
| Hypertension | Positive family history |
| Diabetes Mellitus | Obesity |

**H** = chest pain = 2

**E** = ECG normal = 0

**A** = 63 = 1

**R** = 3 risk fctors = 2

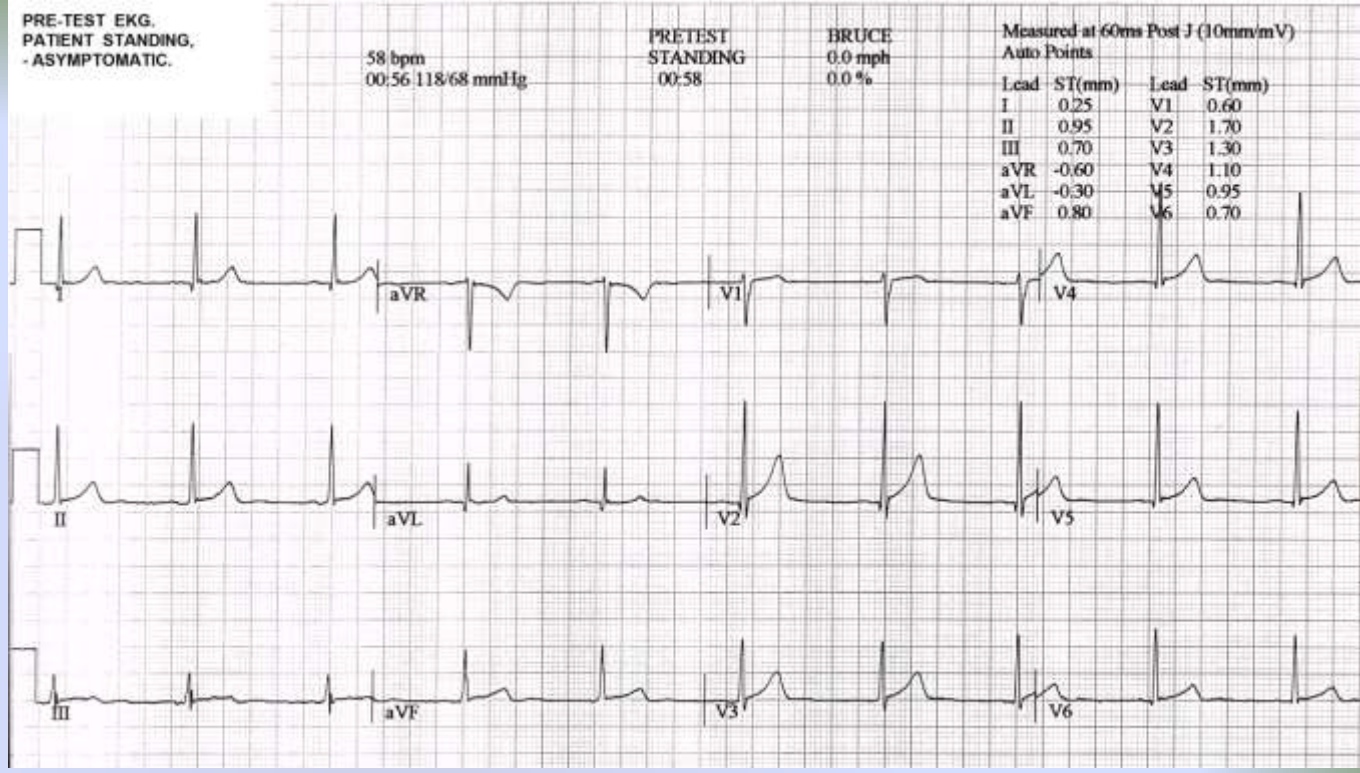
**T** = Trop. NL = 0

**HEART Score: = 5**

**PROBLEMS WITH SENSITIVITY . . .**

# NORMAL ECG.

But . . . . .



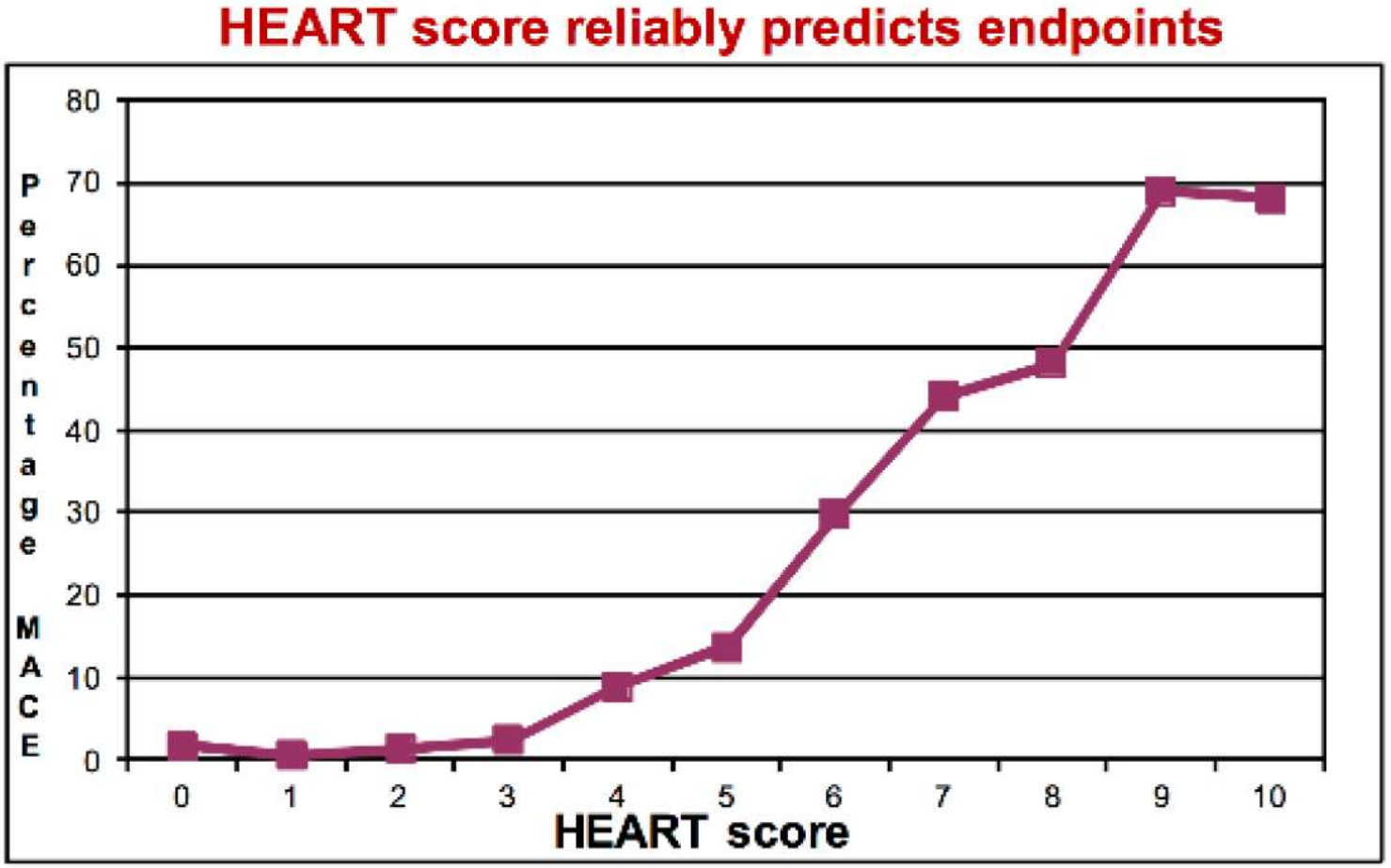

His HEART Score = 5

What could that possibly mean?

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

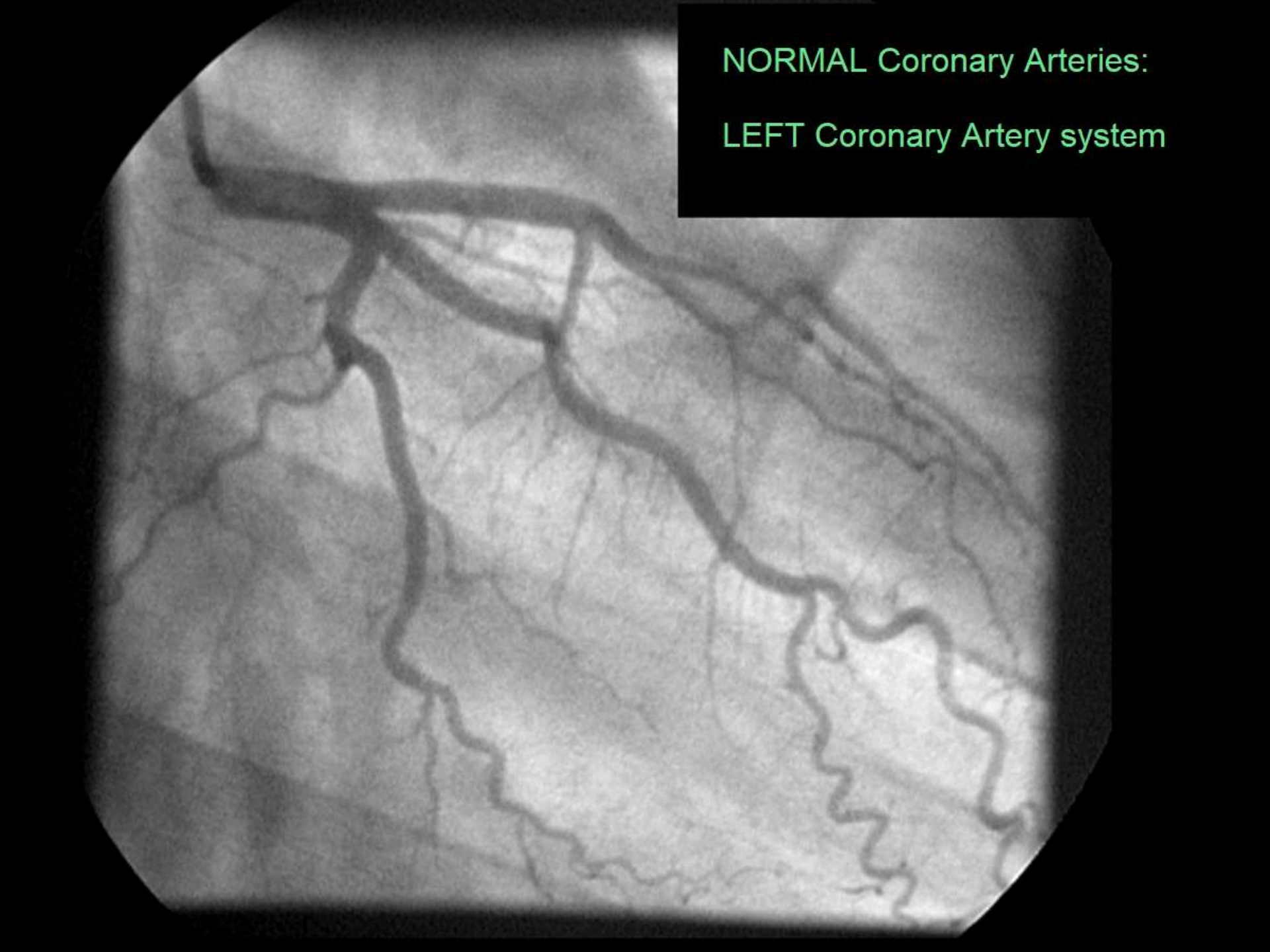
http://www.heartscore.nl/

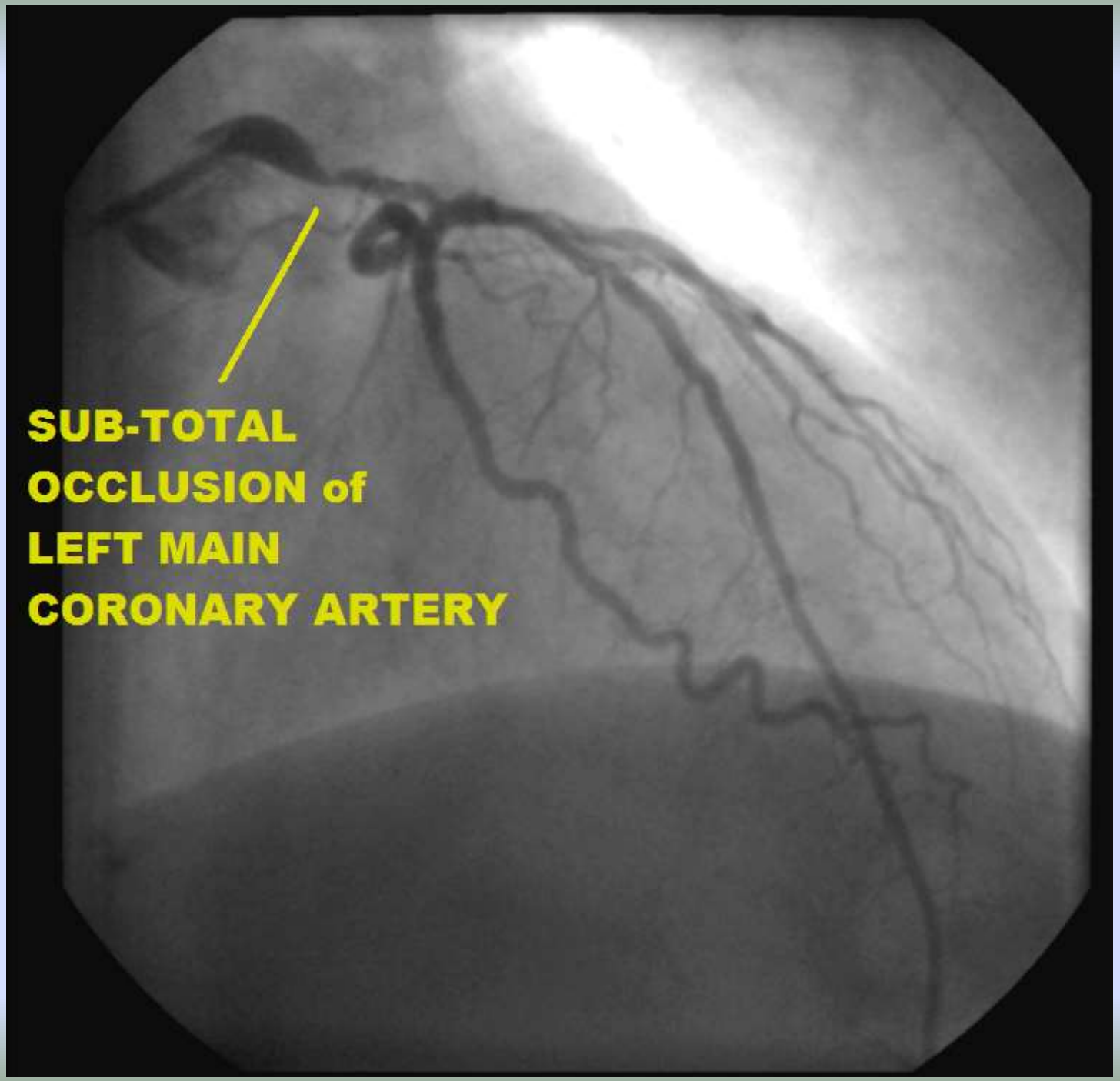
# Heart Score Reliability

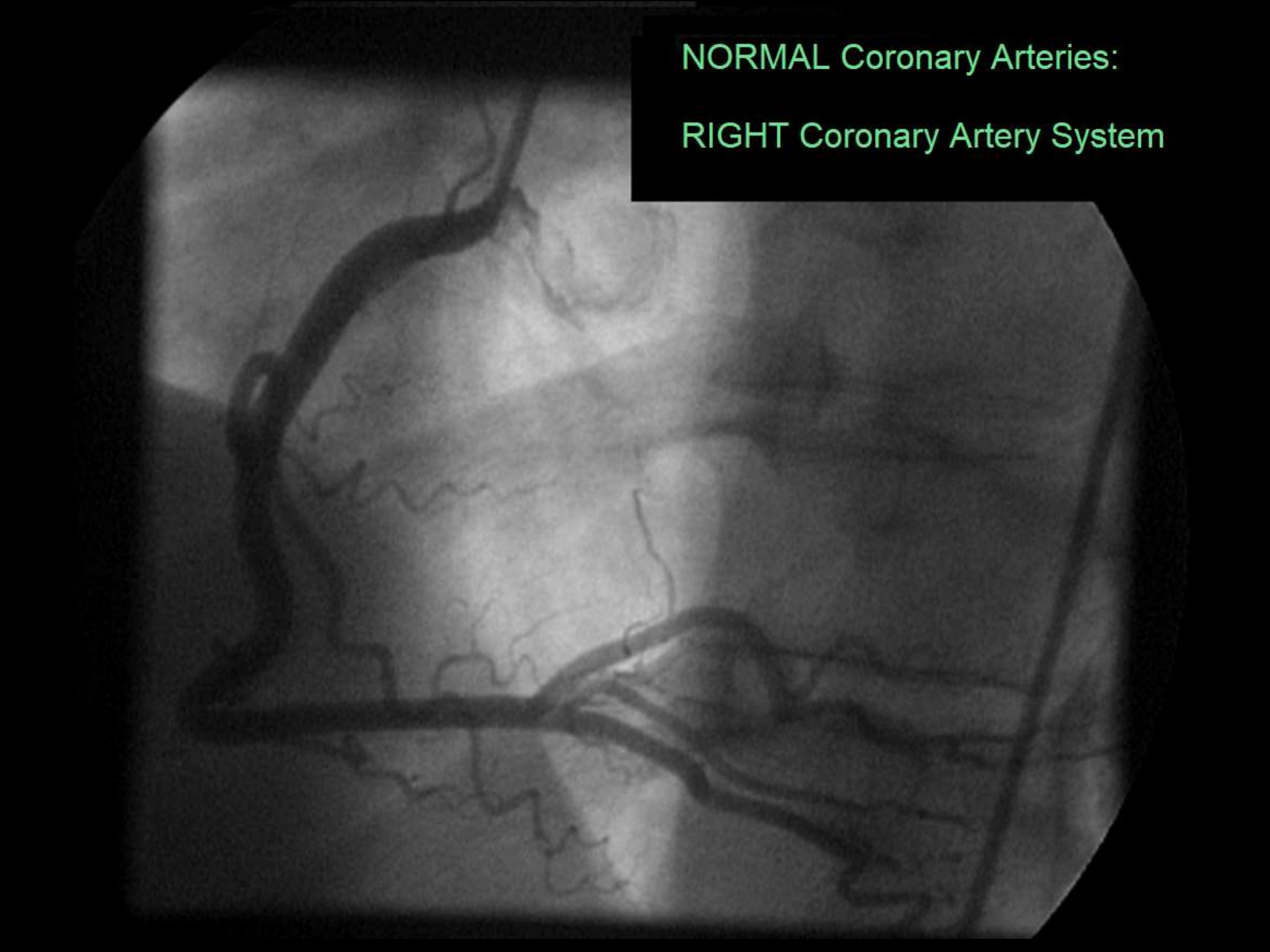


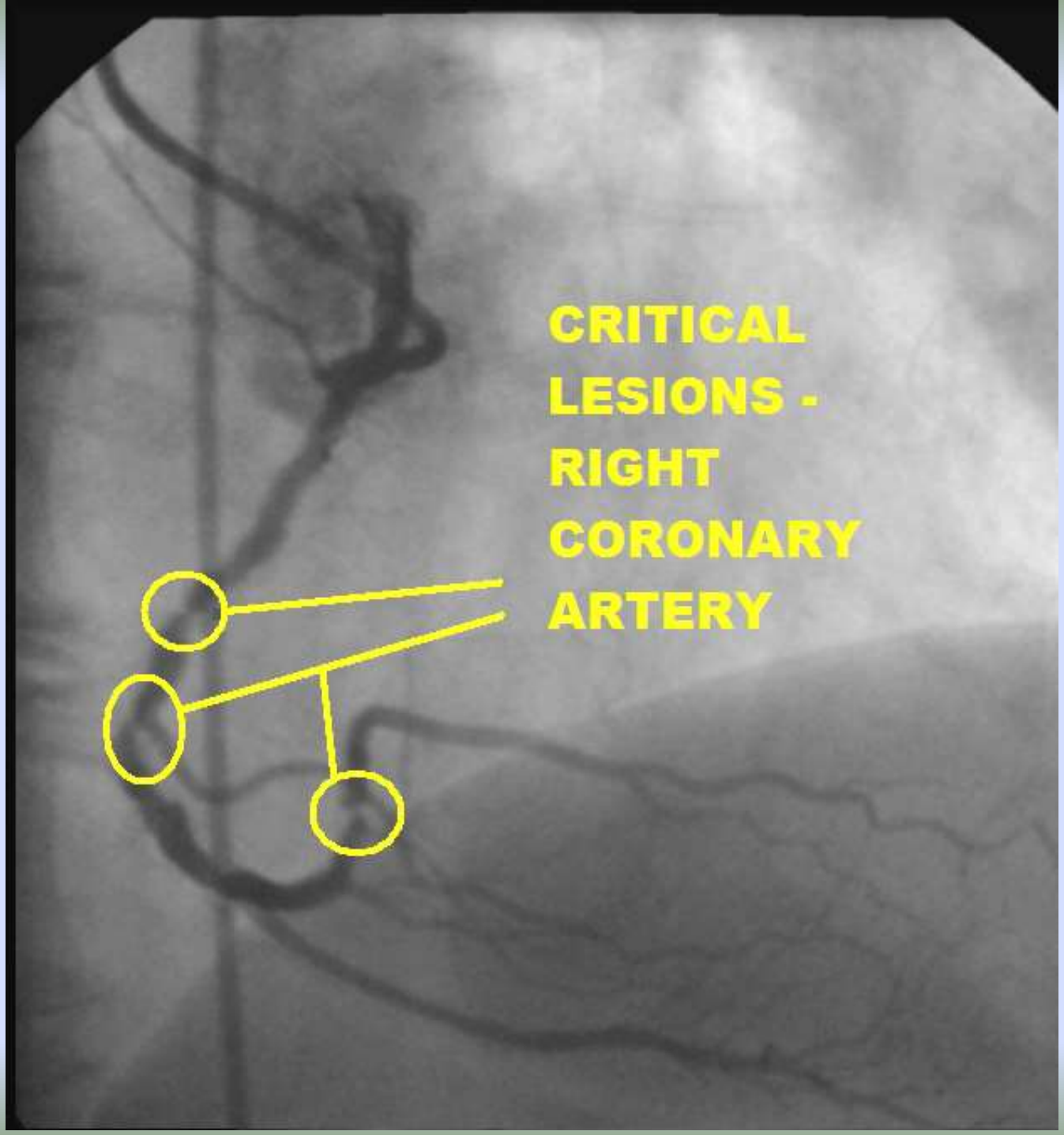
# Based on HEART SCORE:

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


NORMAL Coronary Arteries:
LEFT Coronary Artery system


SUB-TOTAL
OCCLUSION of
LEFT MAIN
CORONARY ARTERY

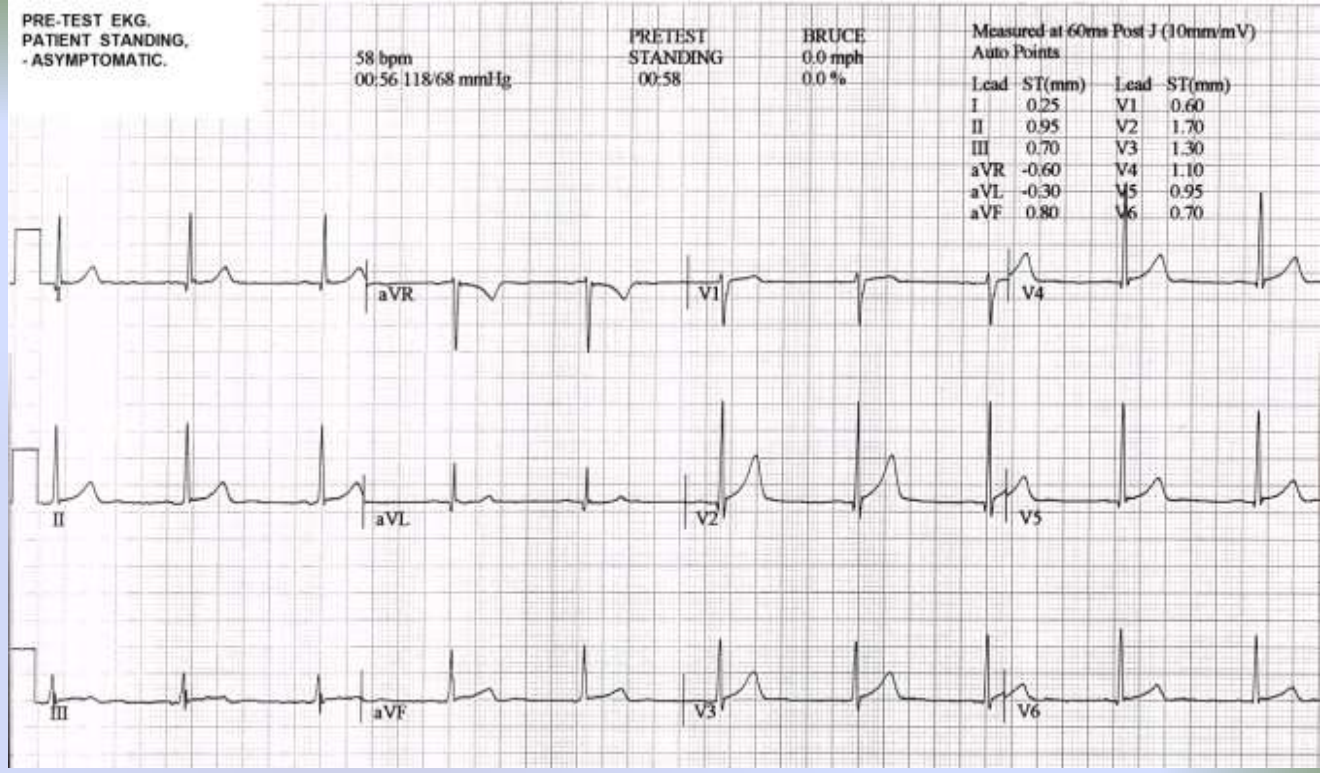

NORMAL Coronary Arteries:
RIGHT Coronary Artery System

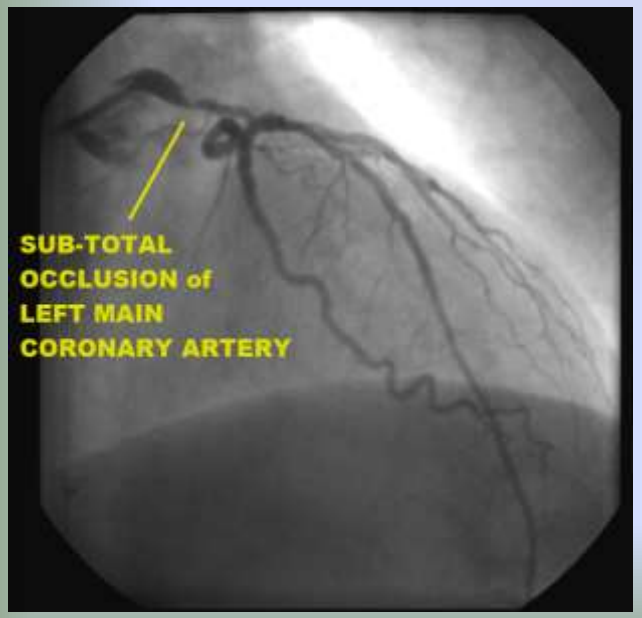

CRITICAL
LESIONS -
RIGHT
CORONARY
ARTERY

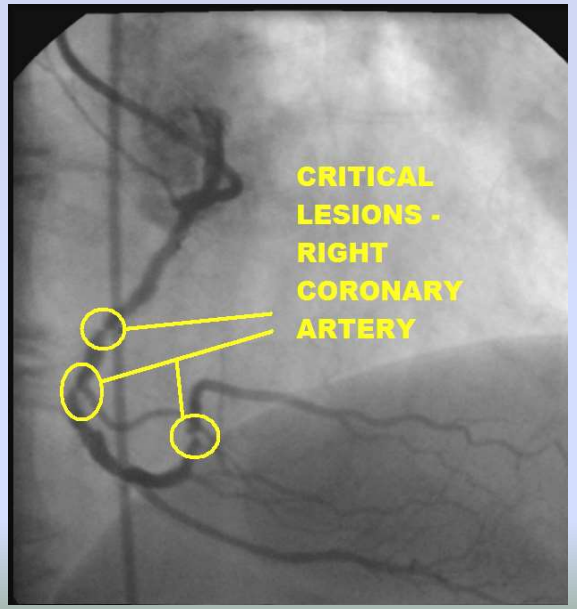
*“A classic example of a FALSE NEGATIVE 12 Lead ECG.”*

# Heart Score 5.

# Lethal Triple Vessel Disease =




***Emergency Triple Vessel Coronary Artery Bypass Surgery***

# US HEART Score Validation

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

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


Prehospital and Disaster Medicine

## Article contents



# Prehospital Modified HEART Score Predictive of 30-Day Adverse Cardiac Events

Published online by Cambridge University Press: **10 January 2018**

Jason P. Stopyra, William S. Harper, Tyson J. Higgins, Julia V. Prokesova, James E. Winslow, Robert D. Nelson, Roy L. Alson, Christopher A. Davis, Gregory B. Russell and Chadwick D. Miller

...Show all authors Show author details



## Abstract

### Introduction

The History, Electrocardiogram (ECG), Age, Risk Factors, and Troponin (HEART) score is a decision aid designed to risk stratify emergency department (ED) patients with acute chest pain. It has been validated for ED use, but it has yet to be evaluated in a prehospital setting.

### Hypothesis

# “HEART” – minus the “T”

| **H**istory | **Value** |
|---|---|
| Highly Suspicious | 2 |
| Moderately Suspicious | 1 |
| Slightly Suspicious | 0 |
| **ECG** | |
| Significant ST-Depression | 2 |
| Non-Specific Repolarization Disturbance | 1 |
| Normal | 0 |
| **A**ge | |
| ≥ 65 years | 2 |
| 45-65 years | 1 |
| ≤ 45 years | 0 |
| **R**isk Factors | |
| ≥ 3 Risk Factors or History of Atherosclerotic Disease | 2 |
| 1 or 2 Risk Factors | 1 |
| No Risk Factors Known | 0 |
| **Total** | **0-8** |
| “Low Risk” = 0-3 and Normal Troponin<br>“High Risk” = 4-8 **or** Troponin (>0.065 ng/ml) | |

# HEAR Score (Prehospital version)

- HEART – minus the "T" (troponin)
- HEAR Score validated in multiple studies.
- LINK to Cambridge University Journal article on "HEAR Score" for Prehospital use.
- SCORE 0 – 2 = "NEGATIVE"
- SCORE 2 - 4 = "Should be evaluated"
- SCORE 4+ = "Suspicious for ACS"

# HEART Pathway 12+

Chest pain. Risk-stratified.

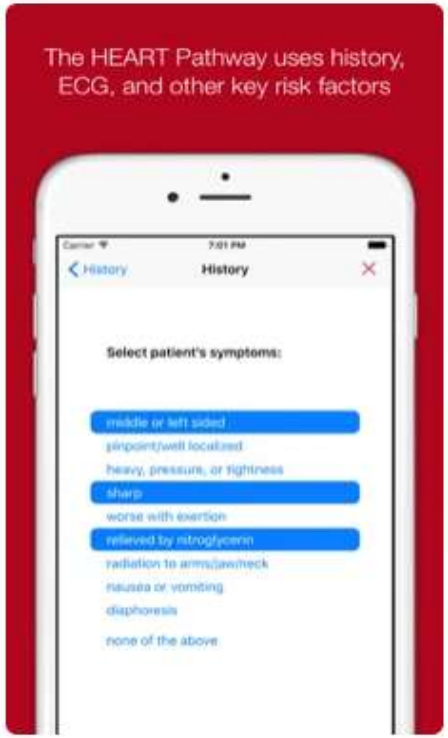
Impathiq

Designed for iPhone

★★★★½ 4.5 • 13 Ratings

Free

## iPhone Screenshots



# CARDIAC ANATOMY and PHYSIOLOGY "101"

**CARDIAC CELLS AT REST have POSITIVE charged IONS on the OUTSIDE of the cell membrane, and NEGATIVE charged IONS on the INSIDE**

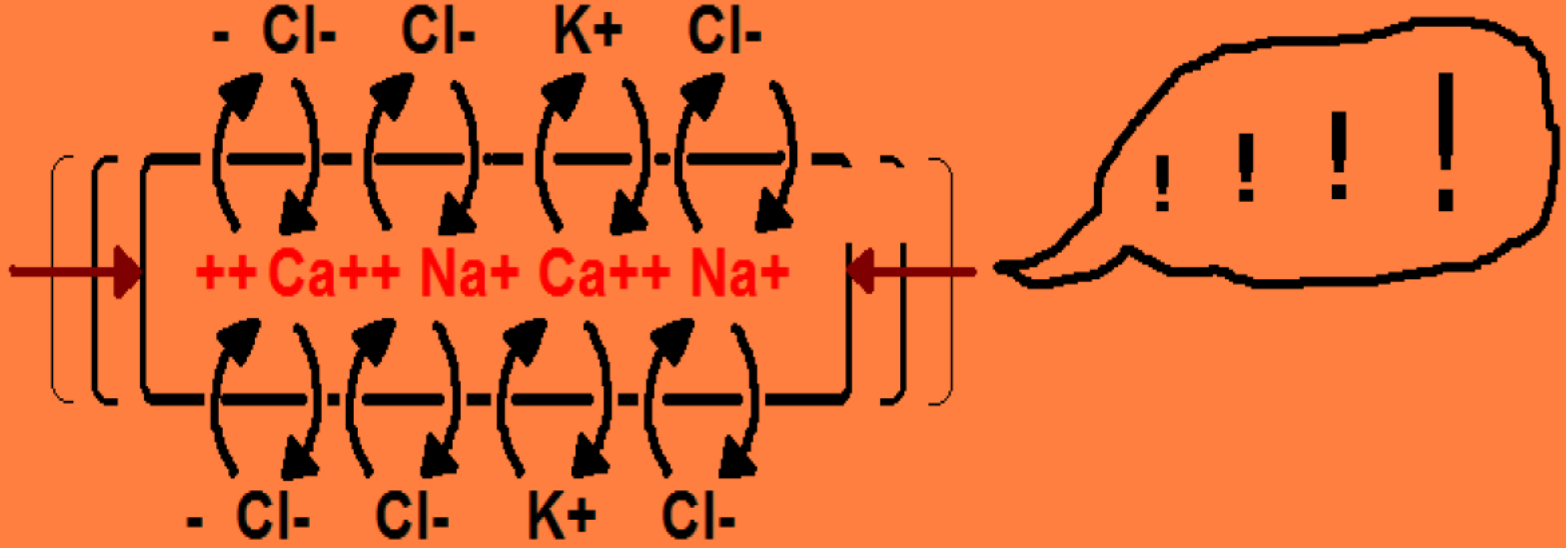
Ca++ Na+ Ca++ Na+ Ca++

Cl- Cl- K+ Cl- K+ Cl- K+ Cl-

Ca++ Na+ Ca++ Na+ Ca++

# CARDIAC ANATOMY and PHYSIOLOGY "101"

**. . . when the IONS shift . . . that is, the POSITIVE IONS that were on the outside TRADE PLACES with the NEGATIVE IONS that were on the INSIDE . . . .**




**. . . THE CELL CONTRACTS!**

# CARDIAC ANATOMY and PHYSIOLOGY "101"

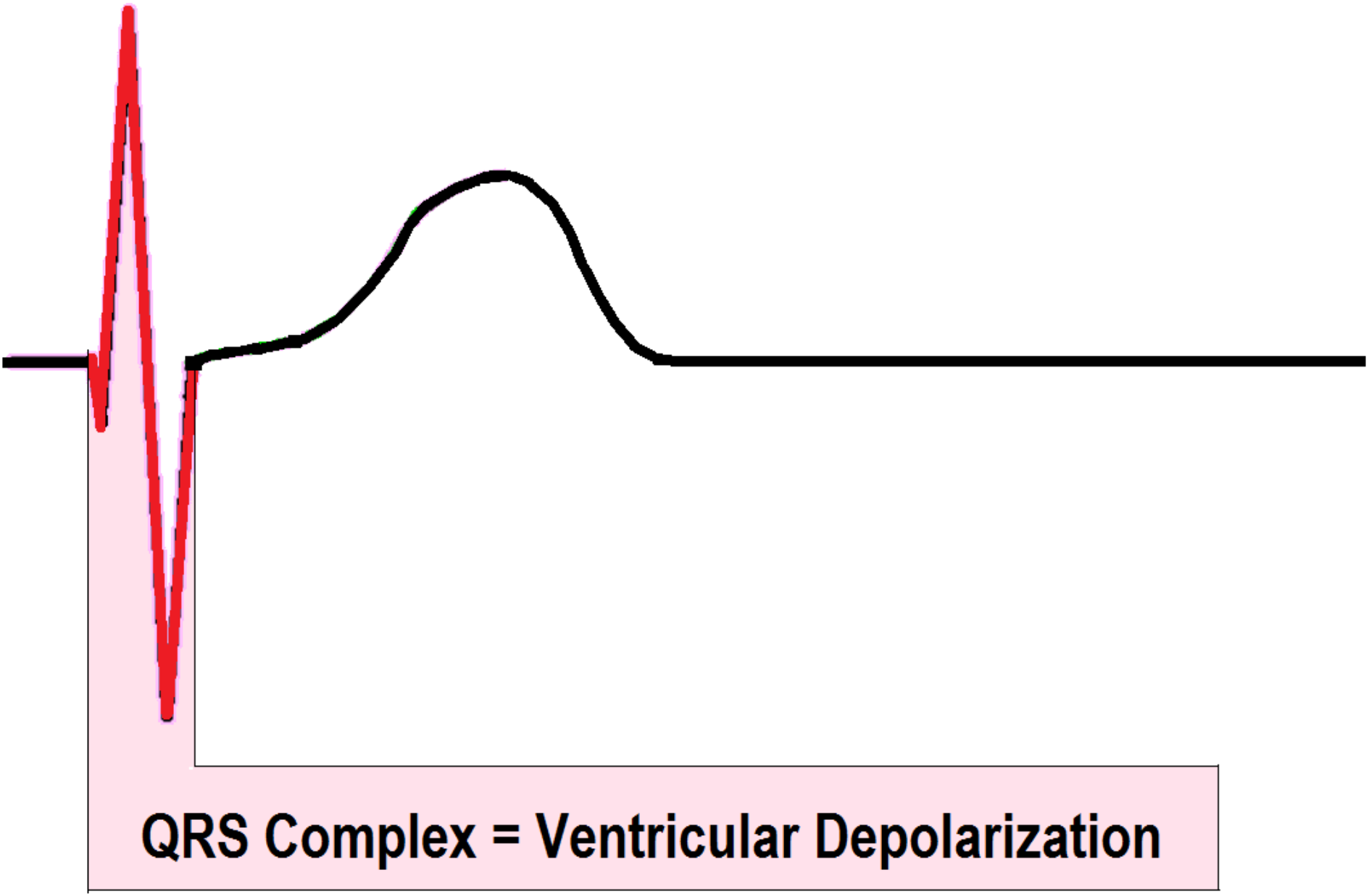
**THIS (OF COURSE) IS KNOW AS . . .**

## DEPOLARIZATION

**WHEN EVERYTHING IS WORKING PROPERLY, THE WAVE OF DEPOLARIZING CELLS CAUSES THE HEART TO CONTRACT, AND PUMP BLOOD TO THE LUNGS AND THE SYSTEMIC CIRCULATION**

# **Ventricular Depolarization**:

- Is represented by the **QRS Complex**

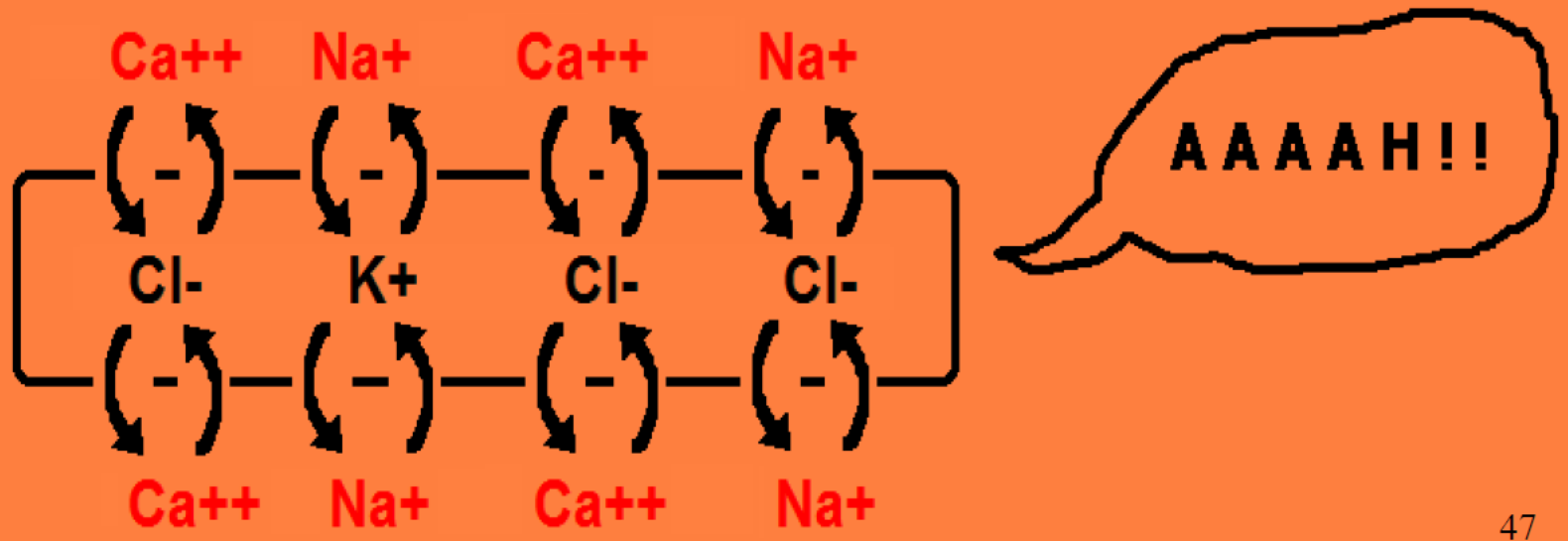

QRS Complex = Ventricular Depolarization

# CARDIAC ANATOMY and PHYSIOLOGY "101"

**AFTER DEPOLARIZATION, THE CELLS RELAX.**

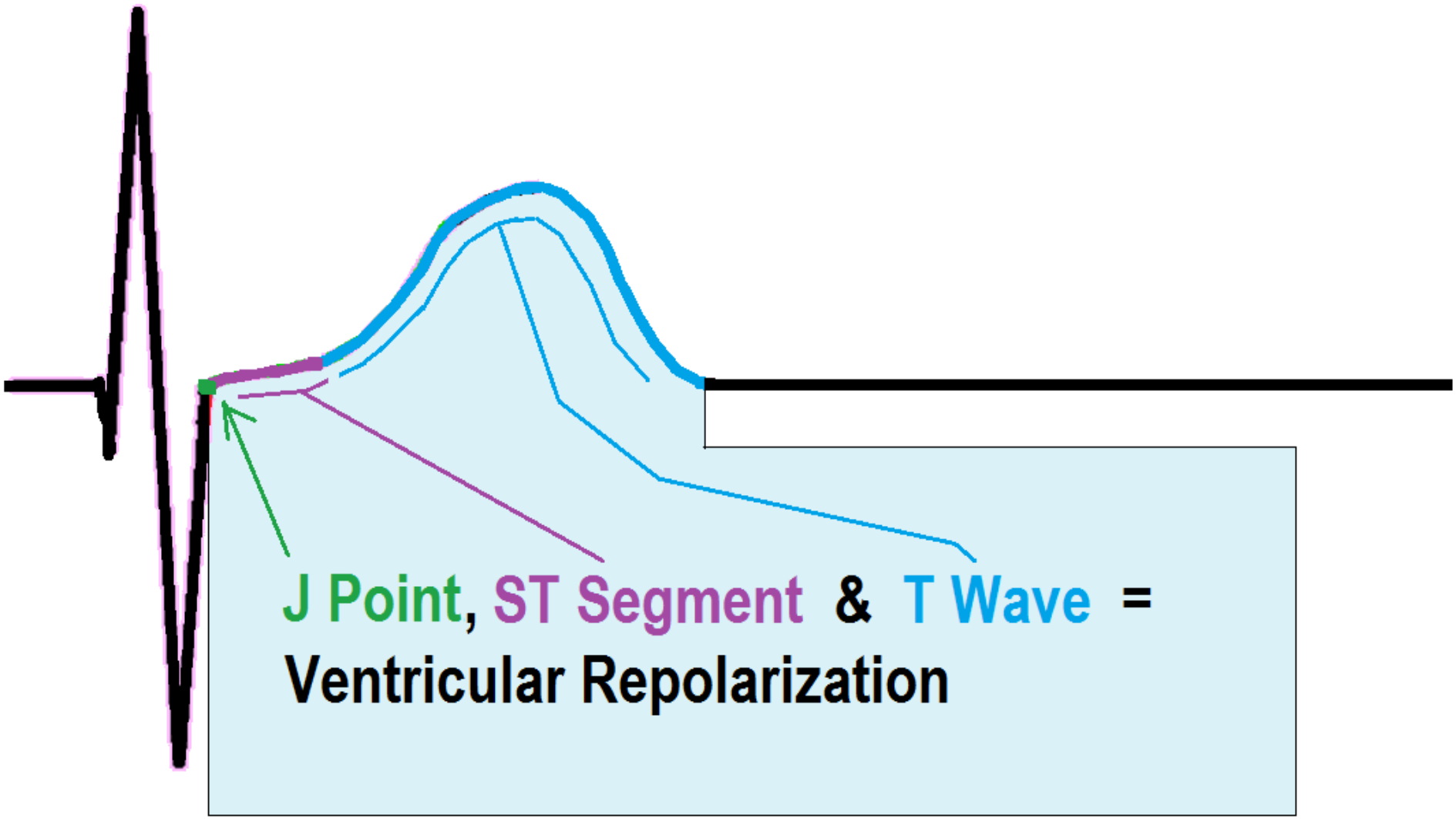
**THE IONS RETURN TO THEIR ORIGINAL POSITIONS --**

**THIS PROCESS IS KNOWN AS REPOLARIZATION**

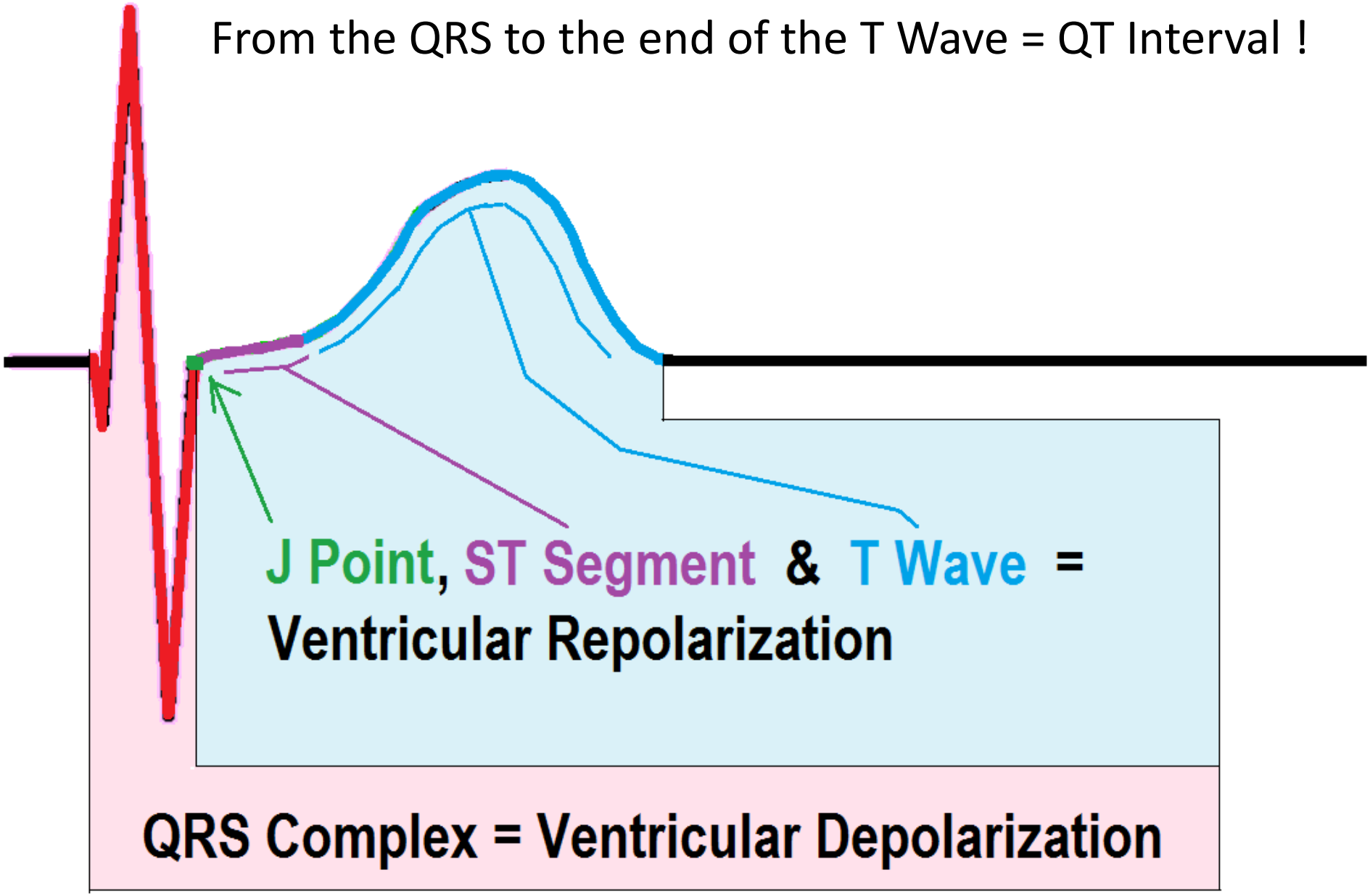


# **Repolarization** on the ECG:

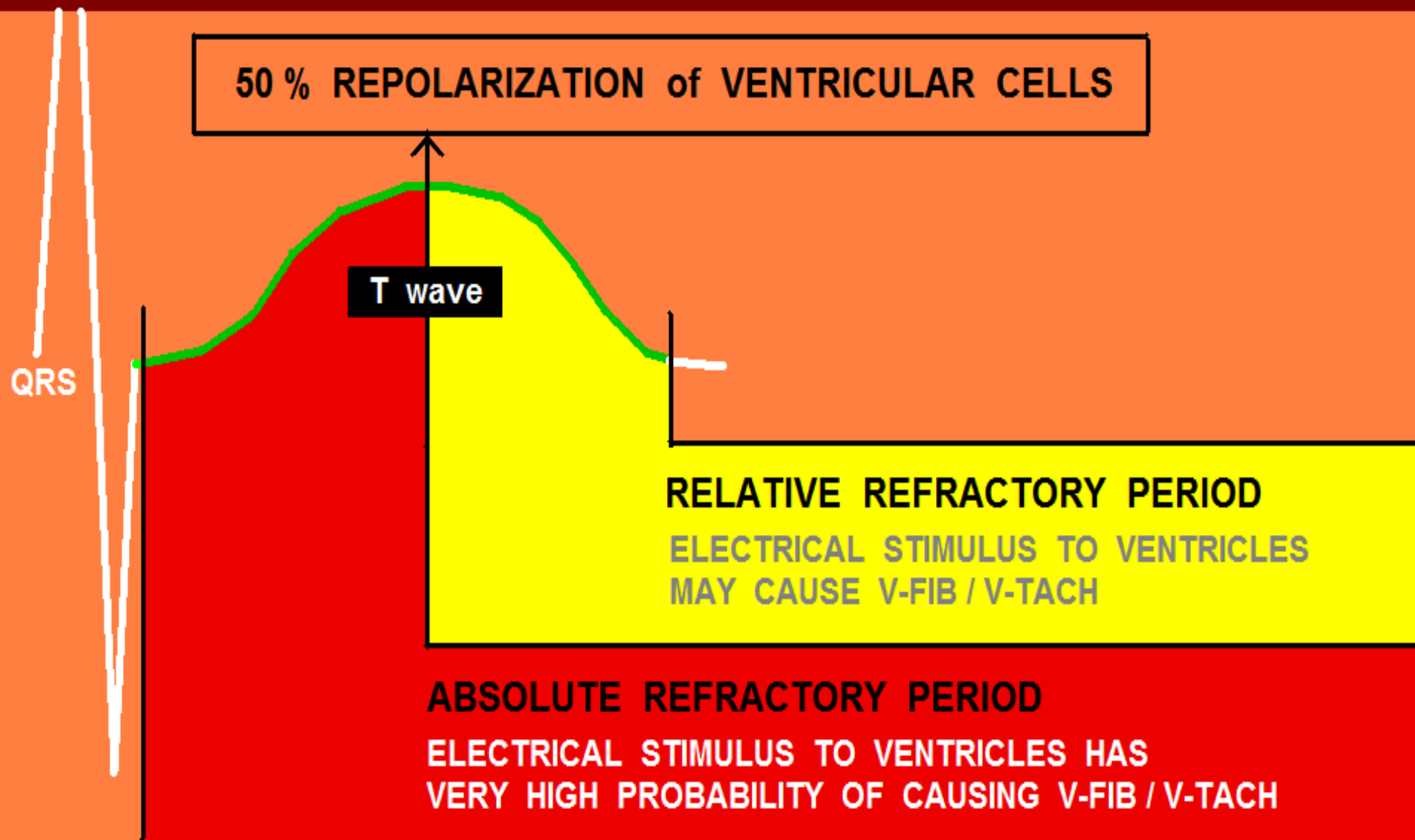
- Is represented by the:
  - **J Point**
  - **ST Segment**
  - **T Wave**


J Point, ST Segment & T Wave =
Ventricular Repolarization

From the QRS to the end of the T Wave = QT Interval !



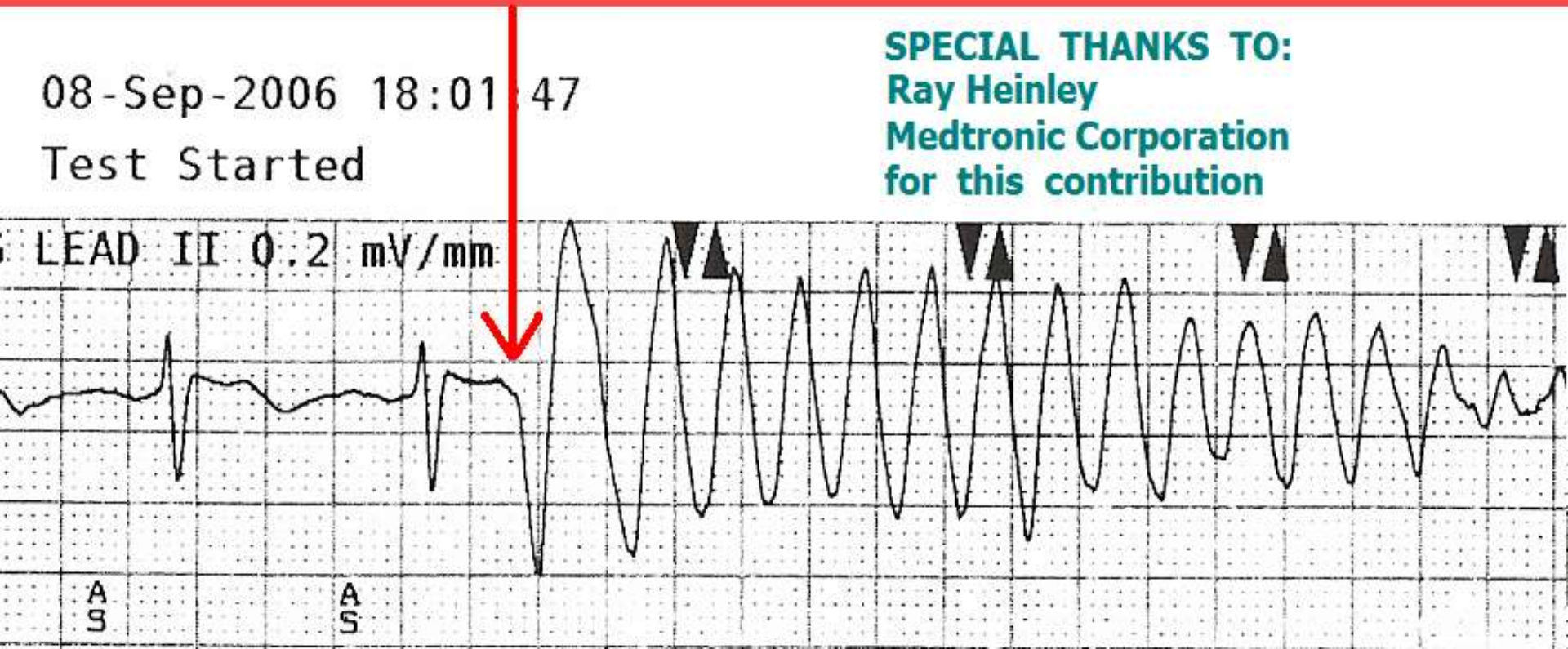
# CARDIAC ANATOMY and PHYSIOLOGY "101"



# ROUTINE TEST OF ICD

## ELECTRICAL IMPULSE ADMINISTERED DURING ABSOLUTE REFRACTORY PERIOD -- INDUCES VENTRICULAR FIBRILLATION

SPECIAL THANKS TO:
Ray Heinley
Medtronic Corporation
for this contribution



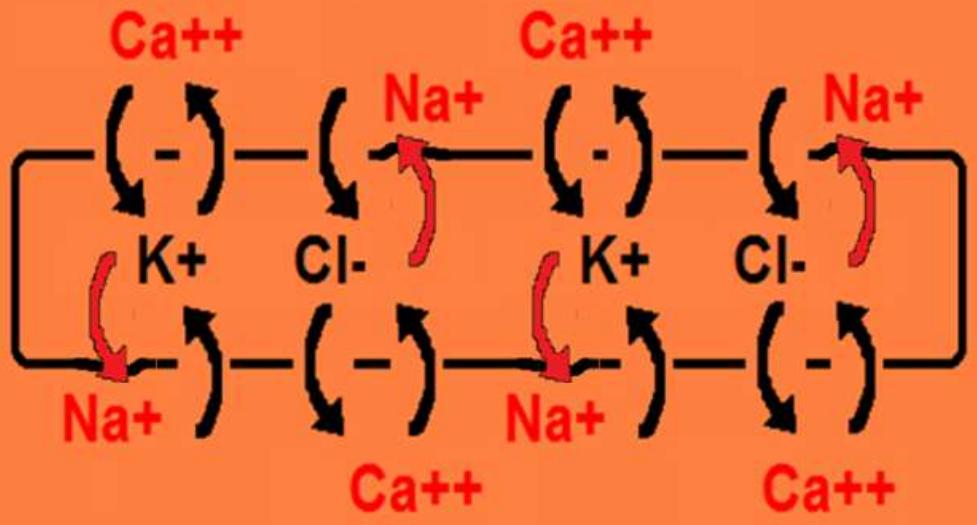
# QT Prolongation

- **Congenital** malformation of ion channels
- **Medications** or other substances alter Ion channel function

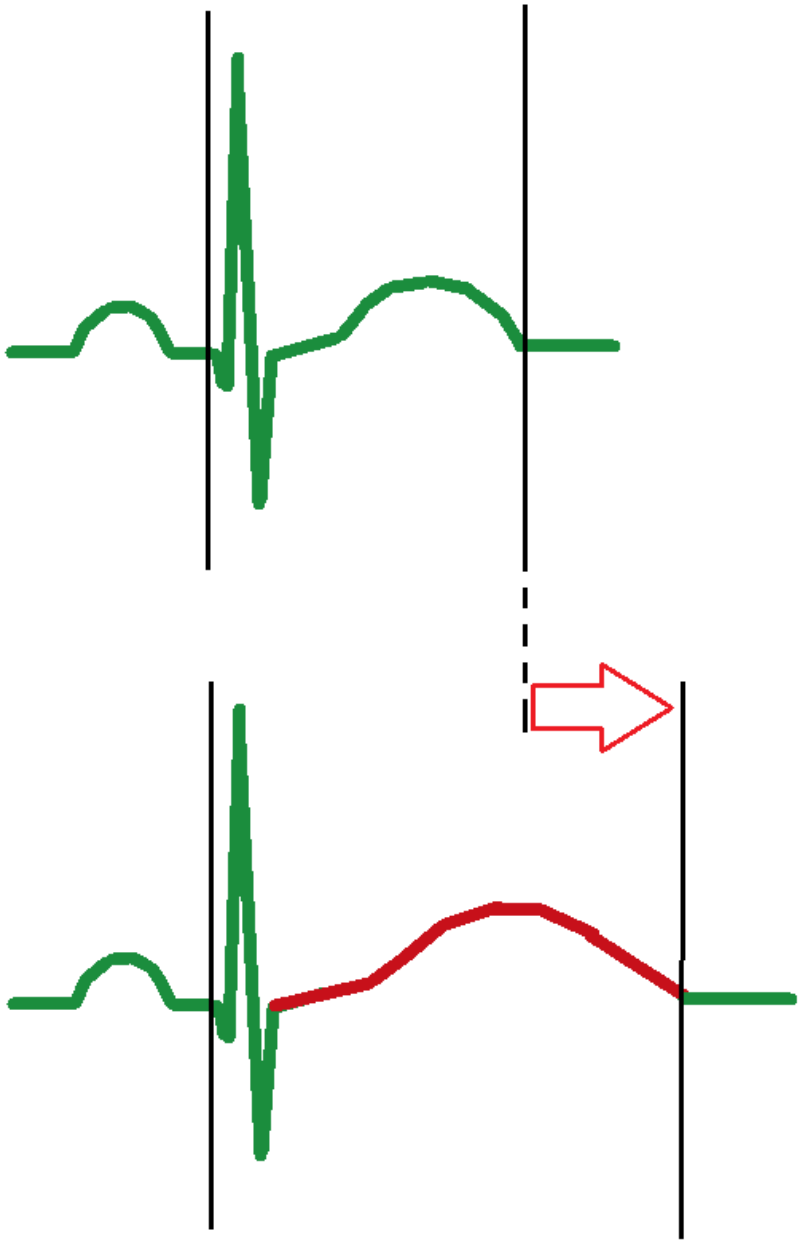
# CARDIAC ANATOMY and PHYSIOLOGY "101"

**When ION CHANNELS are MALFORMED, the abnormal channel shape may DELAY the transfer of IONS . . . . .**

**. . . . this can DELAY REPOLARIZATION, which will show on the ECG as "QT Prolongation"**



Normal
QT Interval

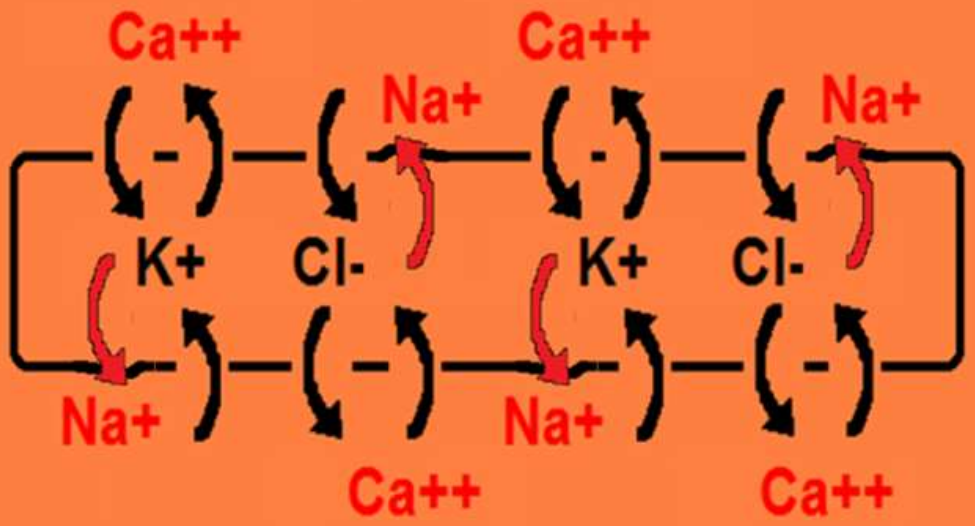


ABNORMAL
(prolonged)
QT Interval

# CARDIAC ANATOMY and PHYSIOLOGY "101"

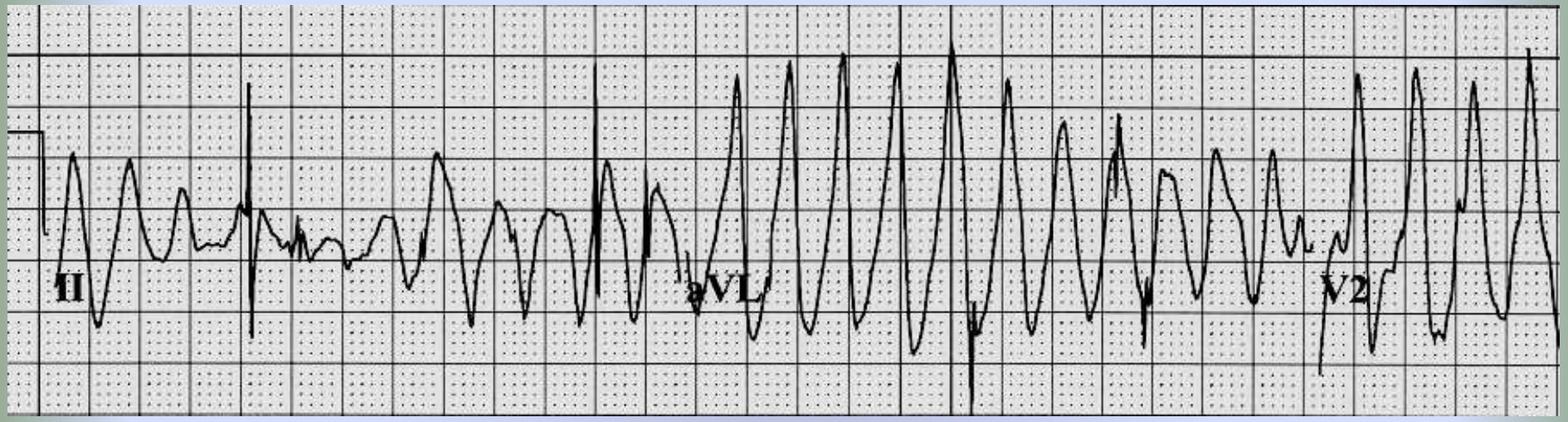
**When ION CHANNELS are MALFORMED, the abnormal channel shape may DELAY the transfer of IONS . . . . .**

**. . . . this can DELAY REPOLARIZATION, which will show on the ECG as "QT Prolongation"**



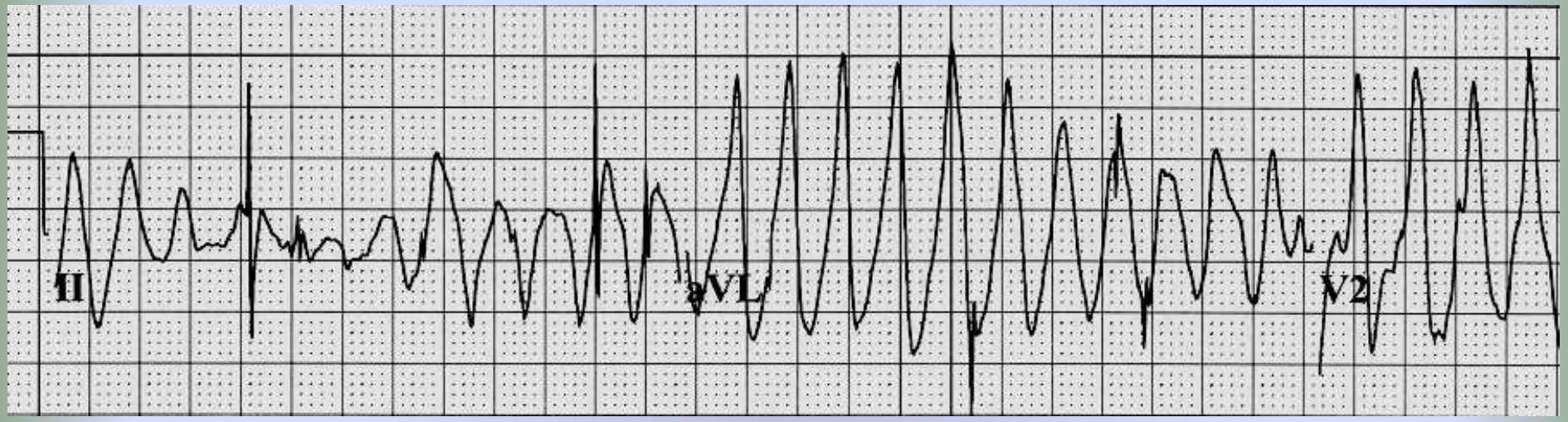

**which can lead to Torsades . . . Cardiac Arrest . . . and *SUDDEN DEATH.***

# *Torsades de Pointes (TdP)*




- **Common cause: QTc > 600 ms**
- Patients typically have little to no cardiac output when in this rhythm
- TdP may self-terminate or deteriorate into **VENTRICULAR FIBRILLATION**

# *Torsades de Pointes (TdP)*

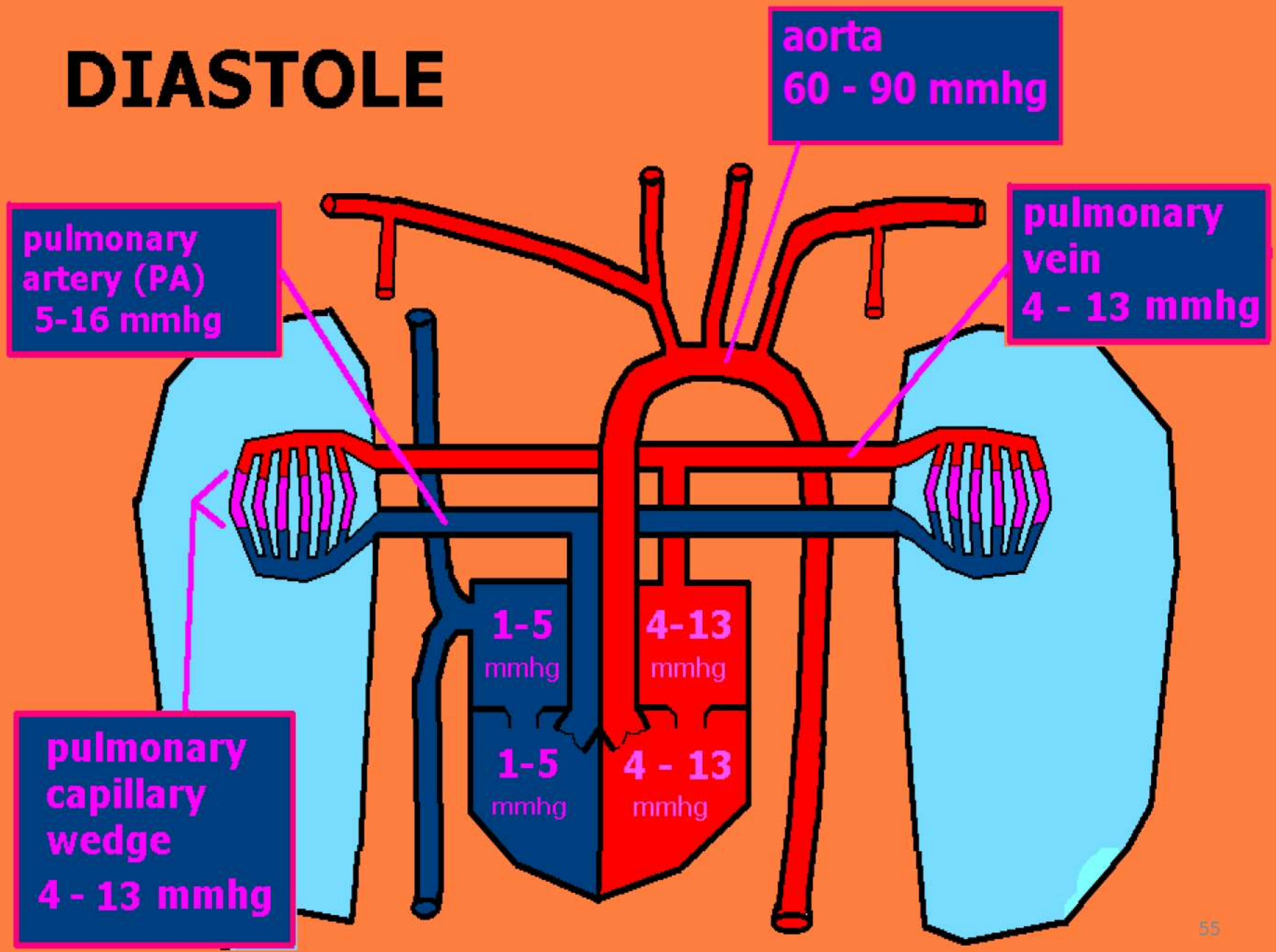



- Common cause:  QTc > 600 ms
- Patients typically have little to no cardiac output when in this rhythm
- TdP may self-terminate or deteriorate into **VENTRICULAR FIBRILLATION**

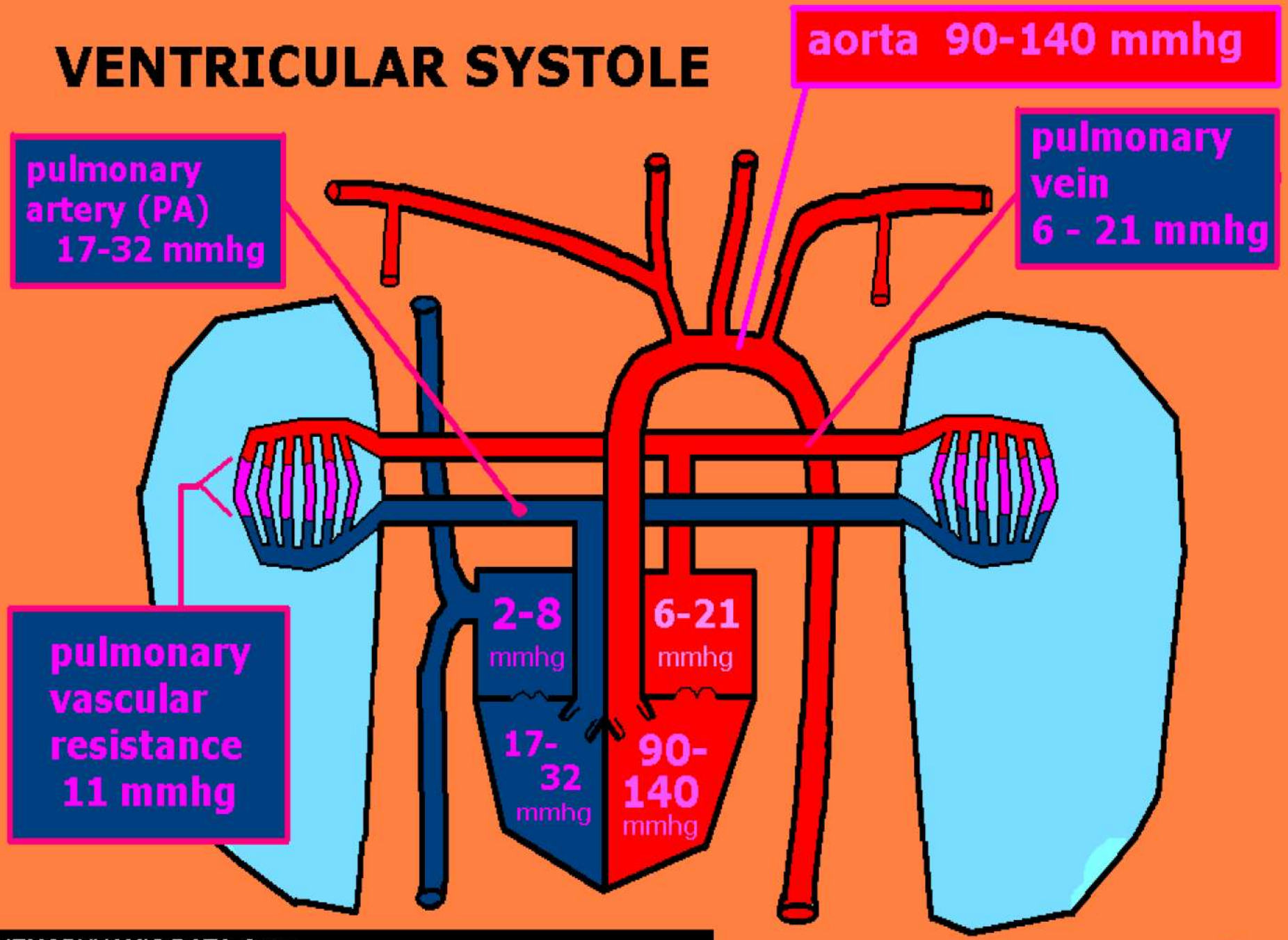
# “Cardiac A & P 101”

- Depolarization and Repolarization
- **Normal Pressures within Heart & Lungs**

# DIASTOLE

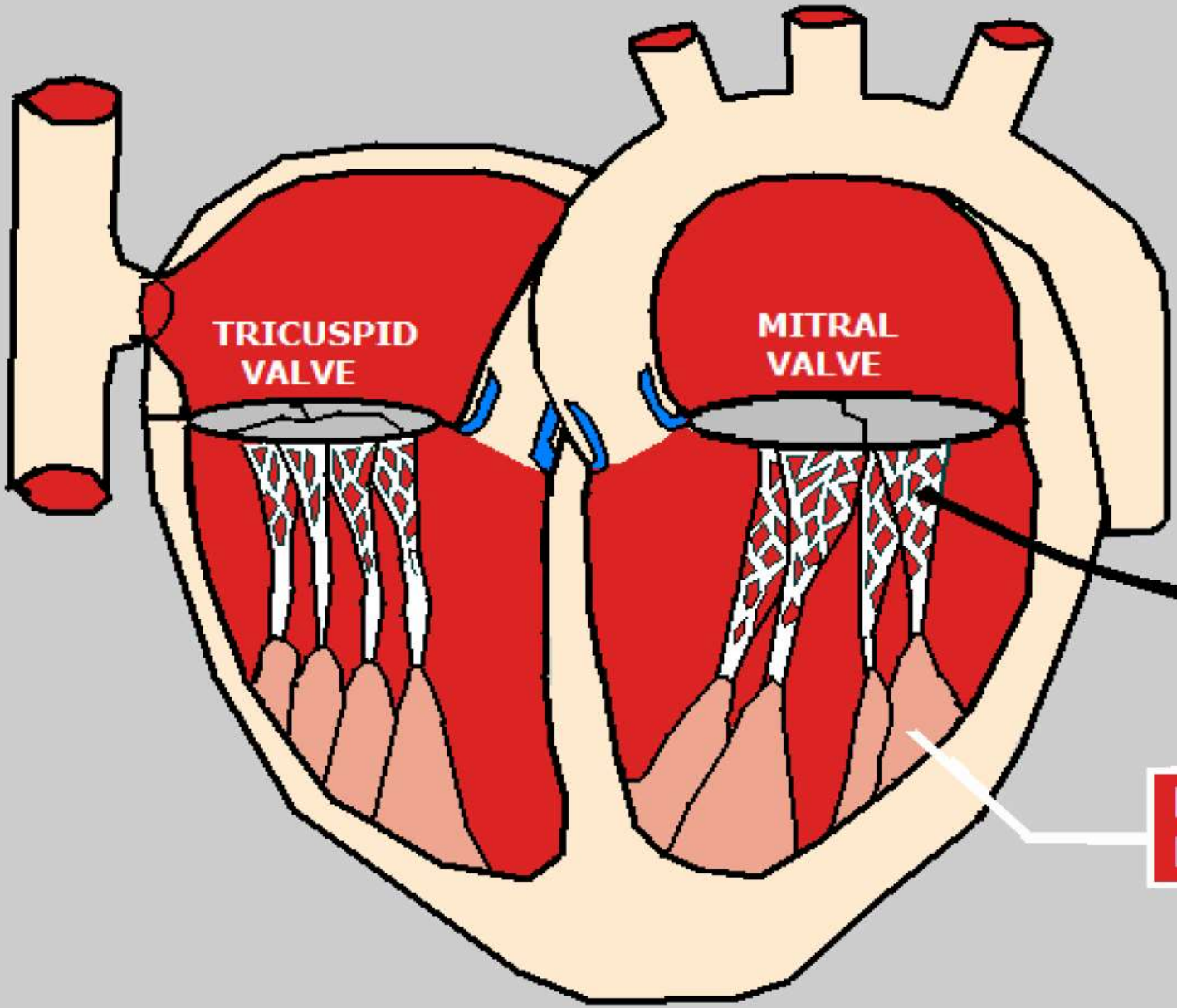

aorta
60 - 90 mmhg
pulmonary artery (PA)
5-16 mmhg
pulmonary vein
4 - 13 mmhg
1-5
mmhg
4-13
mmhg
1-5
mmhg
4 - 13
mmhg
pulmonary capillary wedge
4 - 13 mmhg

# VENTRICULAR SYSTOLE




HEMODYNAMIC DATA from:
"The Cardiac Catheterization Handbook," Morton J. Kearn, MD

# ATRIO-VENTRICULAR VALVES



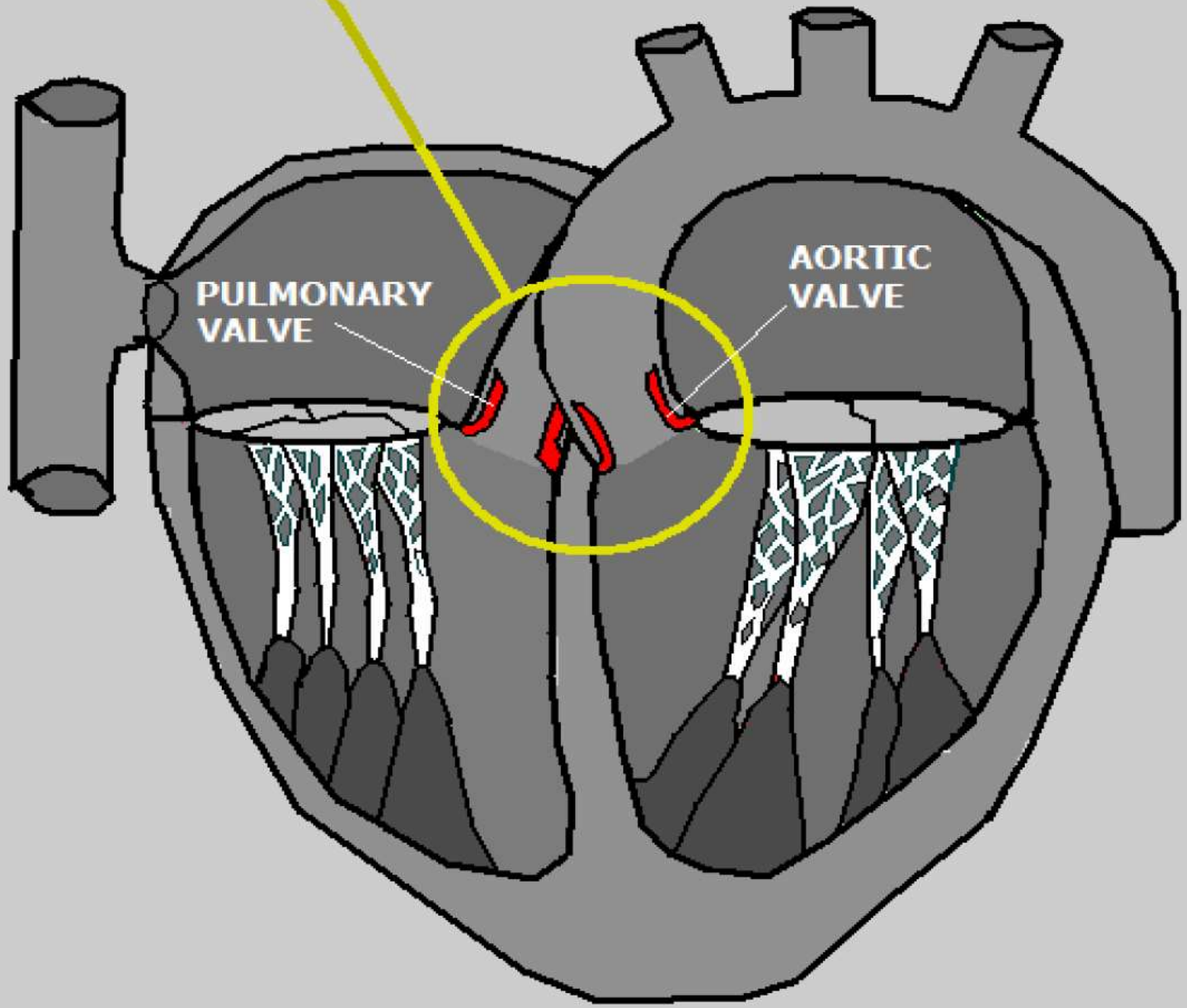

ARE SECURED TO THE ENDO-CARDIAL SURFACE BY THE

CHORDAE TENDINEAE

AND THE

PAPILLARY MUSCLES

# THE SEMILUNAR VALVES



# “Cardiac A & P 101”

- Heart Chambers
- Heart Electrical System & ECG Waveforms
- Depolarization and Repolarization
- Normal Pressures within Heart & Lungs
- Heart Valves
- **Heart Sounds Overview**

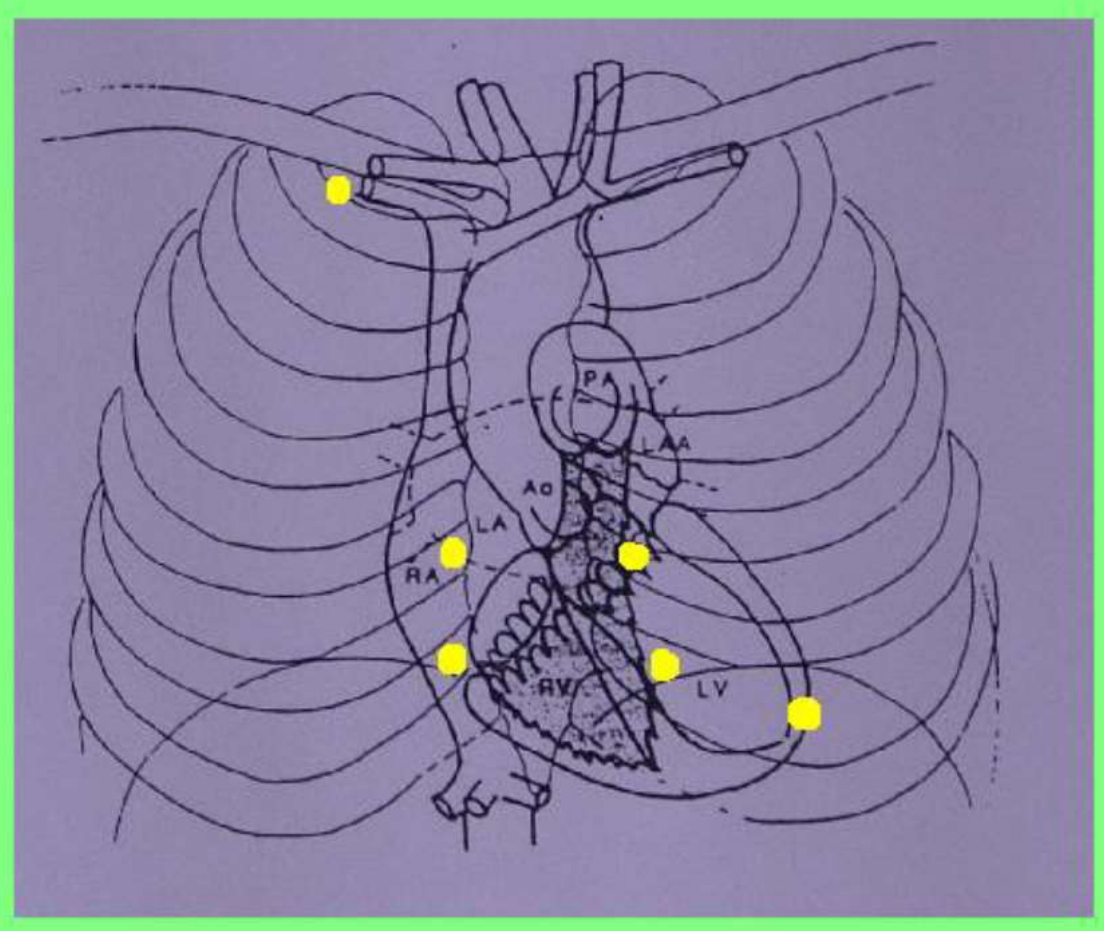
# VERY BASIC HEART SOUNDS ASSESSMENT

ABNORMAL EKG CHANGES THAT MAY PRESENT WITH ABNORMAL HEART SOUNDS:

- ACUTE MI
- CHAMBER HYPERTROPHY
- RECENT MI (NECROSIS)
- PERICARDITIS

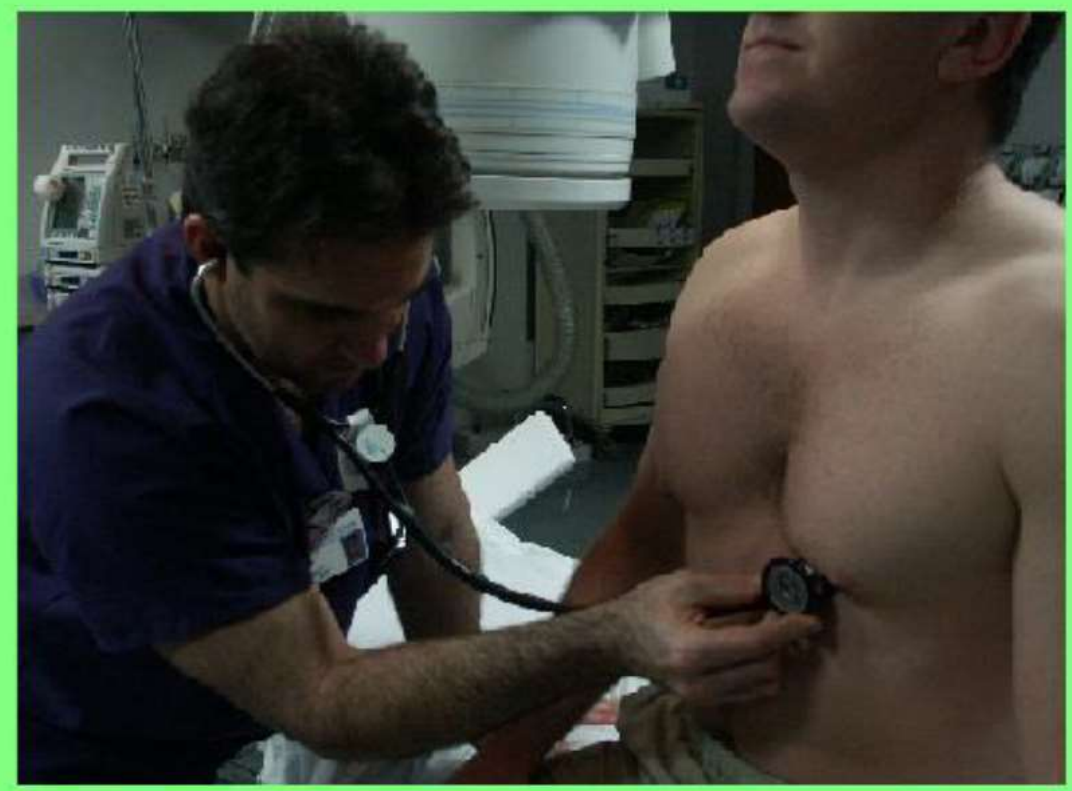
# HEART SOUNDS ASSESSMENT

# HEART SOUNDS ASSESSMENT



# VERY BASIC HEART SOUNDS ASSESSMENT

- Normal Heart Sounds
- Murmurs
  - systolic
  - diastolic
- Friction Rubs



SCOTT DAVIDSON, RN auscultating heart sounds at St. Joseph's Hospital Heart Institute Tampa, FL

# HEART SOUNDS ASSESSMENT

HEART SOUNDS ARE GENERATED BY THE SOUND OF THE HEART VALVES CLOSING.

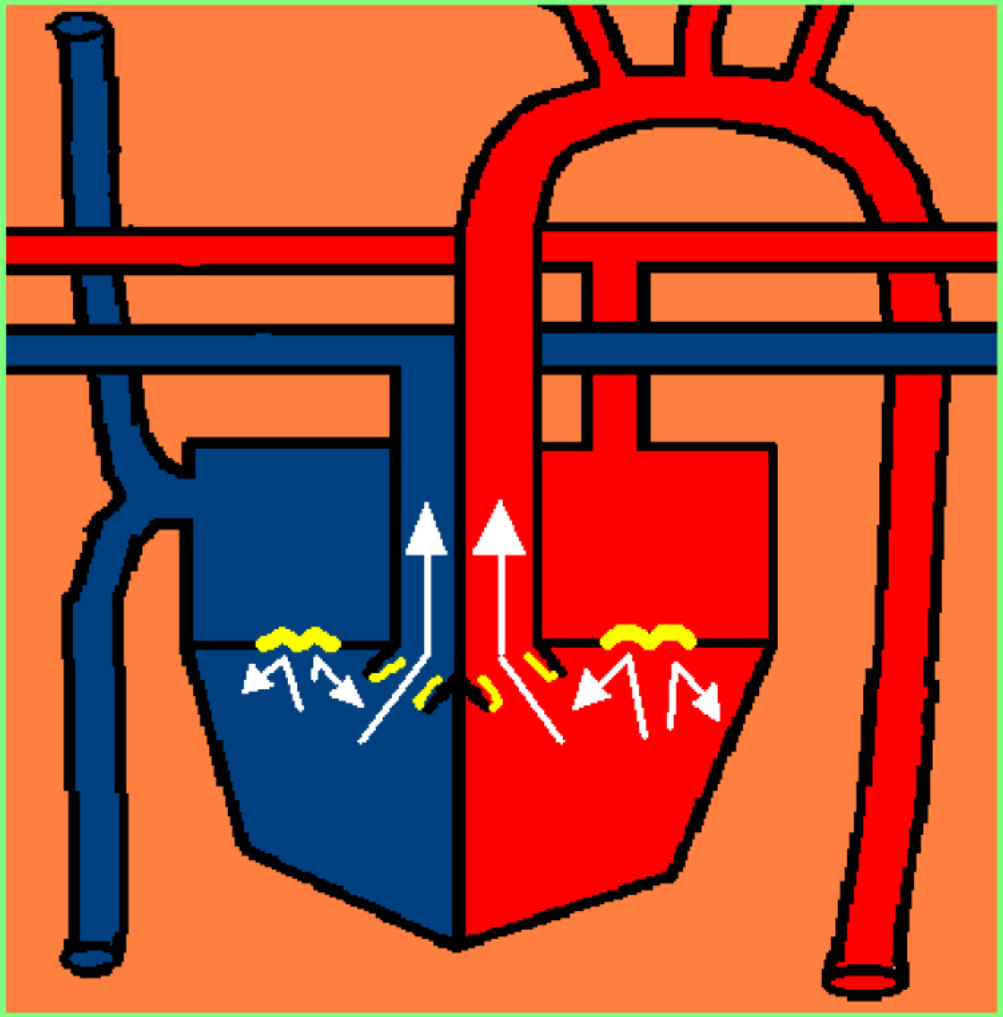
THERE ARE TWO NORMAL HEART SOUNDS, KNOWN AS: S-1 and S-2

WE OFTEN DESCRIBE THESE HEART SOUNDS AS "LUB - DUP"

# HEART SOUNDS ASSESSMENT

**S-1 BEGINNING OF SYSTOLE.**

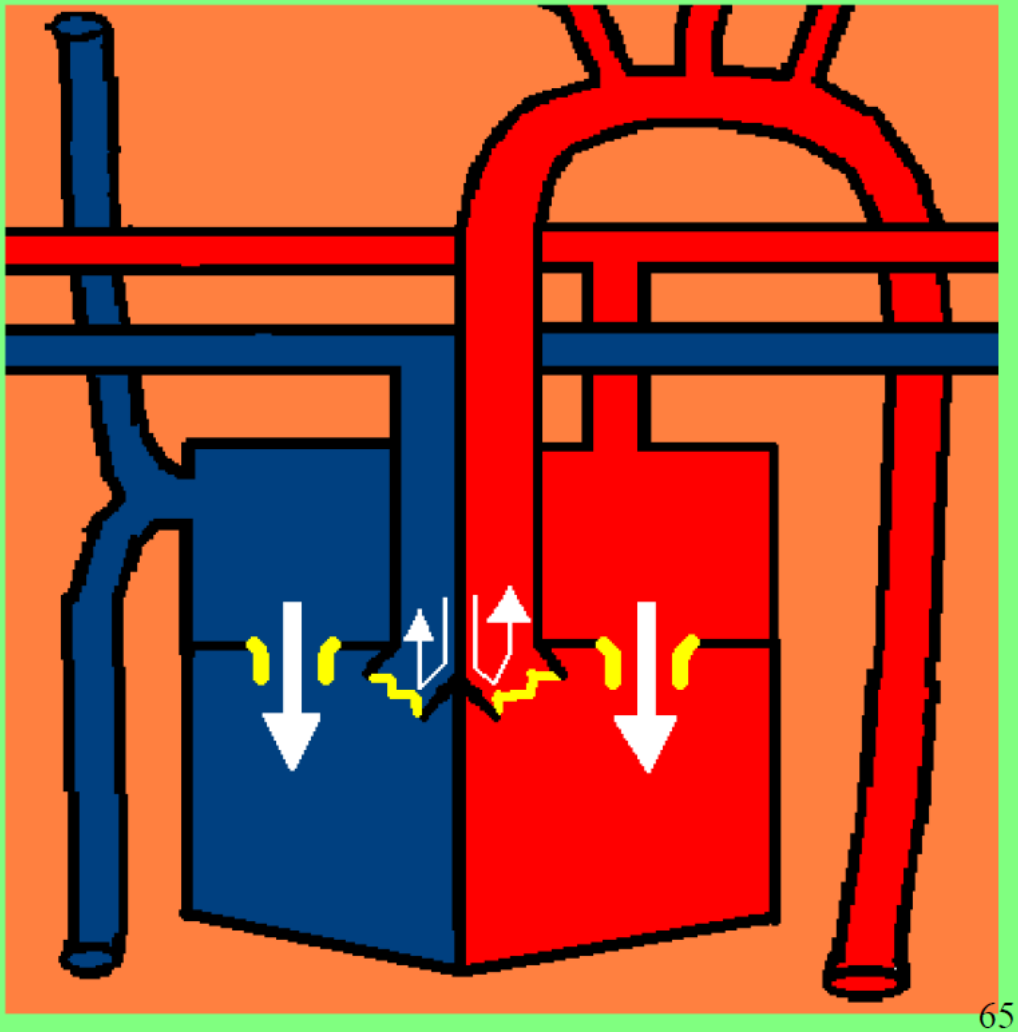
**SOUND OF THE MITRAL AND TRICUSPID VALVES CLOSING.**



# HEART SOUNDS ASSESSMENT

S-2 OCCURS AT THE END OF SYSTOLE (THE BEGINNING OF DIASTOLE).

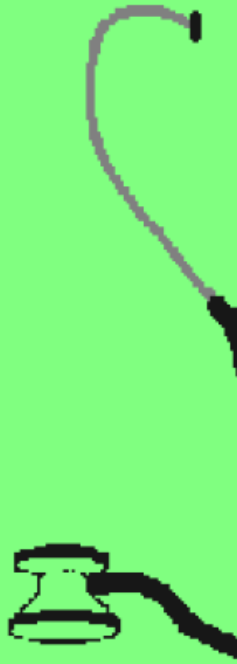
IT IS THE SOUND OF THE AORTIC AND PULMONARY VALVES CLOSING.



MURMUR = "SWOOSH"
SOUND CAUSED BY THE
SOUND OF TURBULENCE.

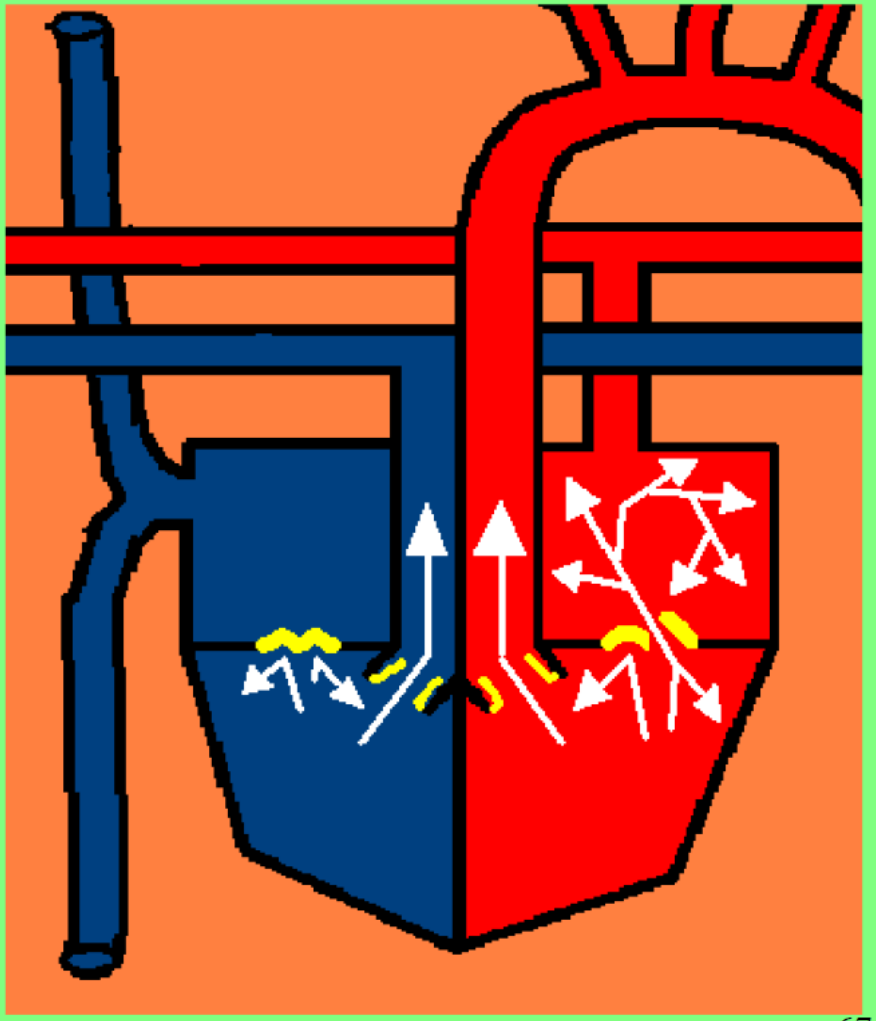
S-1 MURMUR SOUNDS LIKE:

"SWOOSH-DUB . . . . SWOOSH-
DUB . . . . SWOOSH-DUB . . . .
SWOOSH-DUB . . . "



# CAUSE OF SYSTOLIC (S 1) MURMUR

- **DAMAGE TO MITRAL and/or TRICUSPID VALVE(s)**
- **CAUSES REGURGITATION**



- **MOST SYSTOLIC MURMURS CAUSED BY MITRAL VALVE FAILURE.**

- **ACUTE MITRAL VALVE REGURGITATION IS A POTENTIALLY LETHAL COMPLICATION OF ACUTE / RECENT EXTENSIVE TRANSMURAL MI**

**ACUTE MITRAL VALVE RUPTURE USUALLY OCCURS 7-10 DAYS POST EXTENSIVE MI (e.g.: INFERIOR POSTERIOR LATERAL MI).**

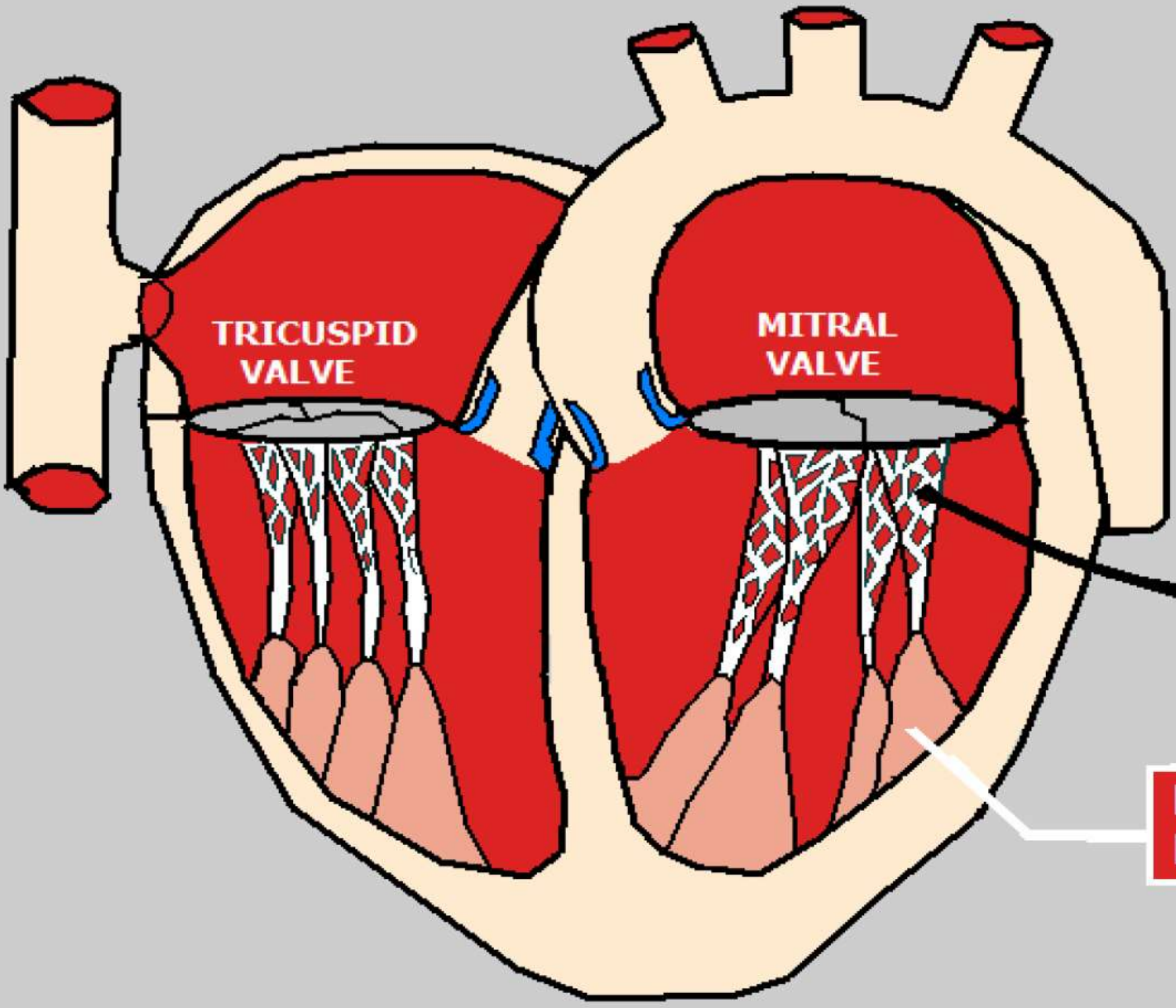
**ACUTE Mitral Valve REGURGITATION can be caused by EXTENSIVE "Multi-Site" Myocardial Infarction and Necrosis – which results in PAPILLARY MUSCLE NECROSIS and PAPILLARY MUSCLE TEAR.**

**Papillary muscles are attached to "multiple surfaces" . . . . .**

# A Common Cause of ACUTE MITRAL REGURGITATION is:

"Patients who are 7-10 days POST-EXTENSIVE MI," in cases where the "zone of infarction" is large (e.g. "inferior-posterior-lateral") and there was a delay in PCI resulting in large zone of necrosis.

# ATRIO-VENTRICULAR VALVES




ARE SECURED TO THE ENDO-CARDIAL SURFACE BY THE

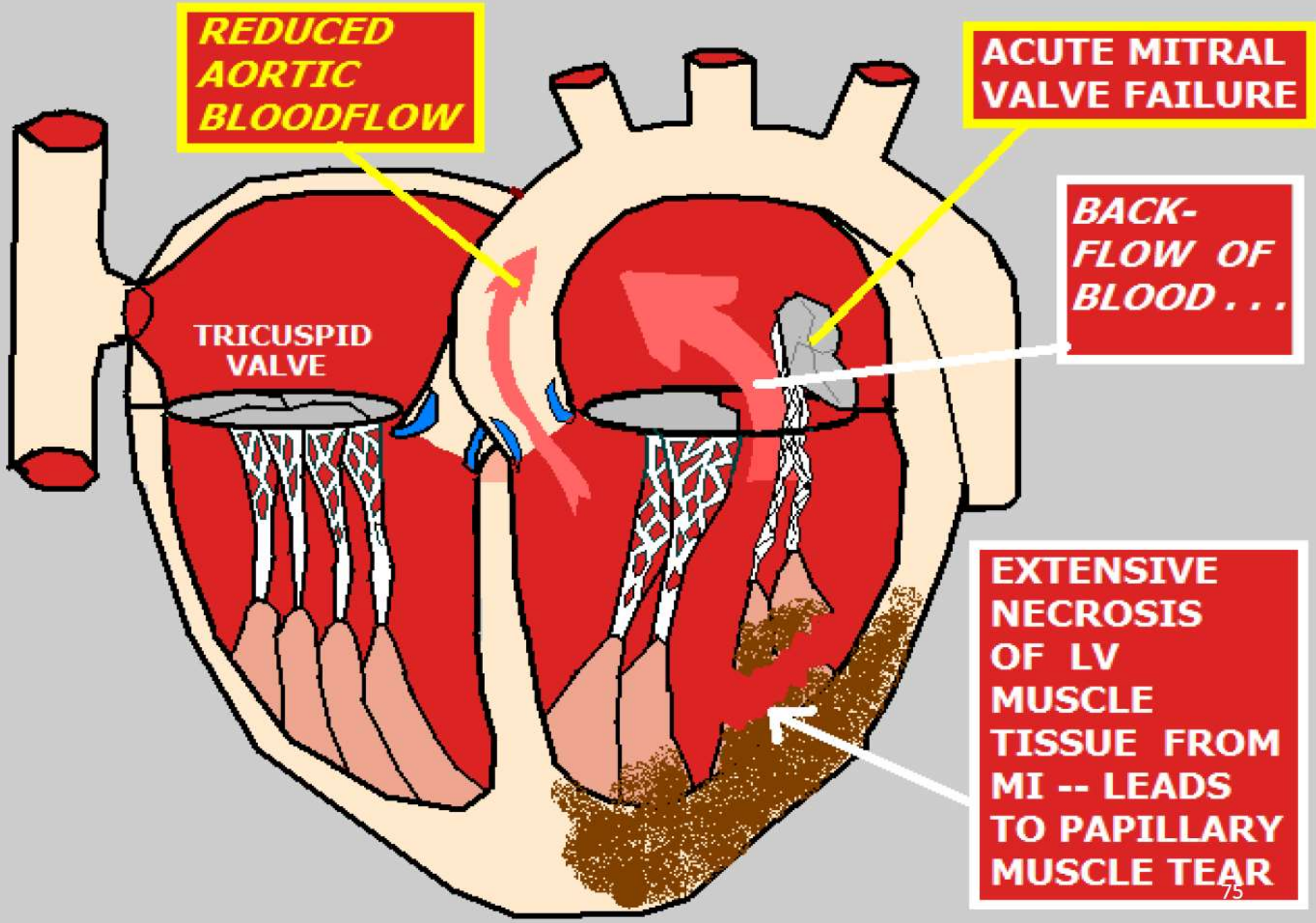
CHORDAE TENDINEAE

AND THE

PAPILLARY MUSCLES

# ACUTE MITRAL REGURGITATION

## DURING VENTRICULAR SYSTOLE



# Symptoms of Acute Mitral Regurgitation

- SHOCK
- PROFOUND HYPOTENSION
- PINK, FROTHY SPUTUM
- PULMONARY EDEMA
- SYSTOLIC (S1) MURMUR

***"SWOOSH – DUB……SWOOSH – DUB……SWOOSH – DUB…"***

# BASIC HEART SOUNDS ASSESSMENT

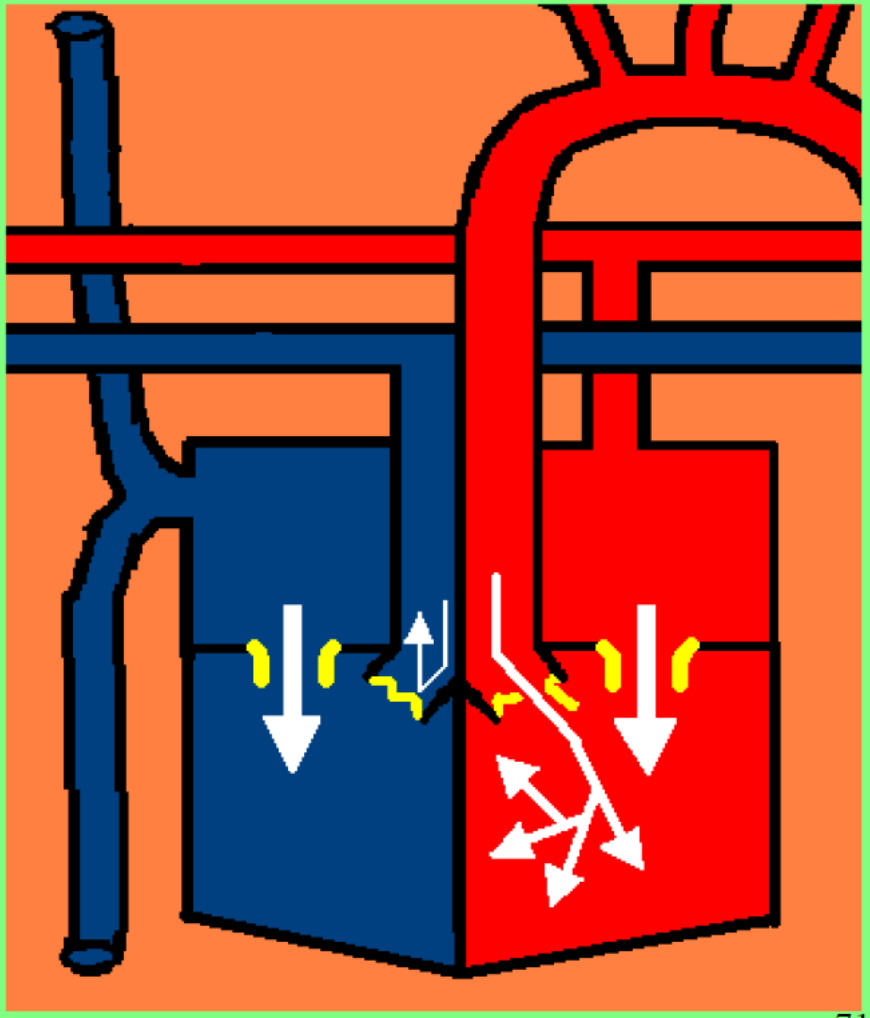
**MURMUR = "SWOOSH"
SOUND CAUSED BY THE
SOUND OF TURBULENCE.**

**S-2 MURMUR SOUNDS LIKE:**

**"LUB-SWOOSH . . . . LUB-SWOOSH
. . . .LUB-SWOOSH . . . . LUB-
SWOOSH . . . "**

# CAUSE OF DIASTOLIC (S2) MURMUR

- **DAMAGE TO AORTIC and/or PULMONIC VALVE(s)**
- **CAUSES REGURGITATION**



Chronic **Valvular REGURGITATION** (Leaky Valve) leads to elevated heart chamber pressures and DILITATION.

Chronic **Valvular STENOSIS** (“Creaky” Valve) leads to Cardiac Muscle STRAIN and HYPERTROPHY.

BOTH conditions, if untreated, eventually leads to **HEART FAILURE.**

# Heart Sounds: S3

- S3 sounds like: " kenTUCky . . . kenTUCky"
- Caused by: increased atrial pressure.
- S3 is associated with: **Heart Failure, Dilated Cardiomyopathy.**

# Heart Sounds: S4

- S4 sounds like: "TENnessee. . . TENnessee"
- Caused by: stiffened left ventricle.
- S4 is associated with: **Hypertension, Aortic Stenosis, Ischemic** or **Hypertrophic Cardiomyopathy.**

Access
University of Washington
Department of Medicine

---

Heart Sound Simulator

# BASIC HEART SOUNDS ASSESSMENT

## FRICTION RUB

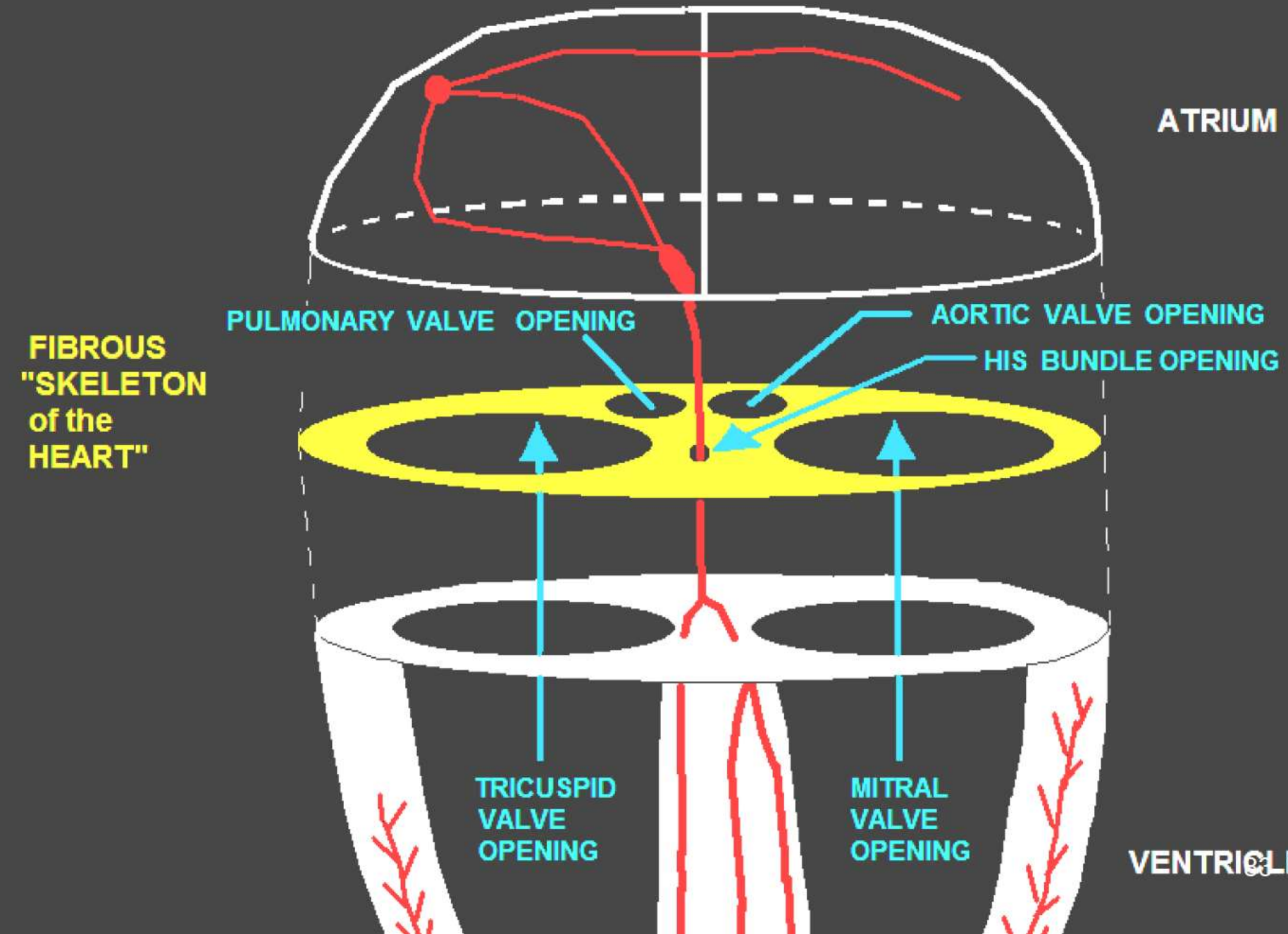
- ASSOCIATED WITH PERICARDITIS
- SOUNDS LIKE THE GENTLE RUBBING OF SANDPAPER
- HAS 3 COMPONENTS: SYSTOLIC, EARLY, and LATE DIASTOLIC

# BASIC HEART SOUNDS ASSESSMENT

## FRICTION RUB

- IS PRESENT IN MOST ACUTE TRANSMURAL MI PATIENTS
- MAY BE PRESENT WITHIN HOURS AFTER ONSET
- IS TRANSIENT -- MAY LAST FOR A FEW DAYS

# THE "SKELETON OF THE HEART"

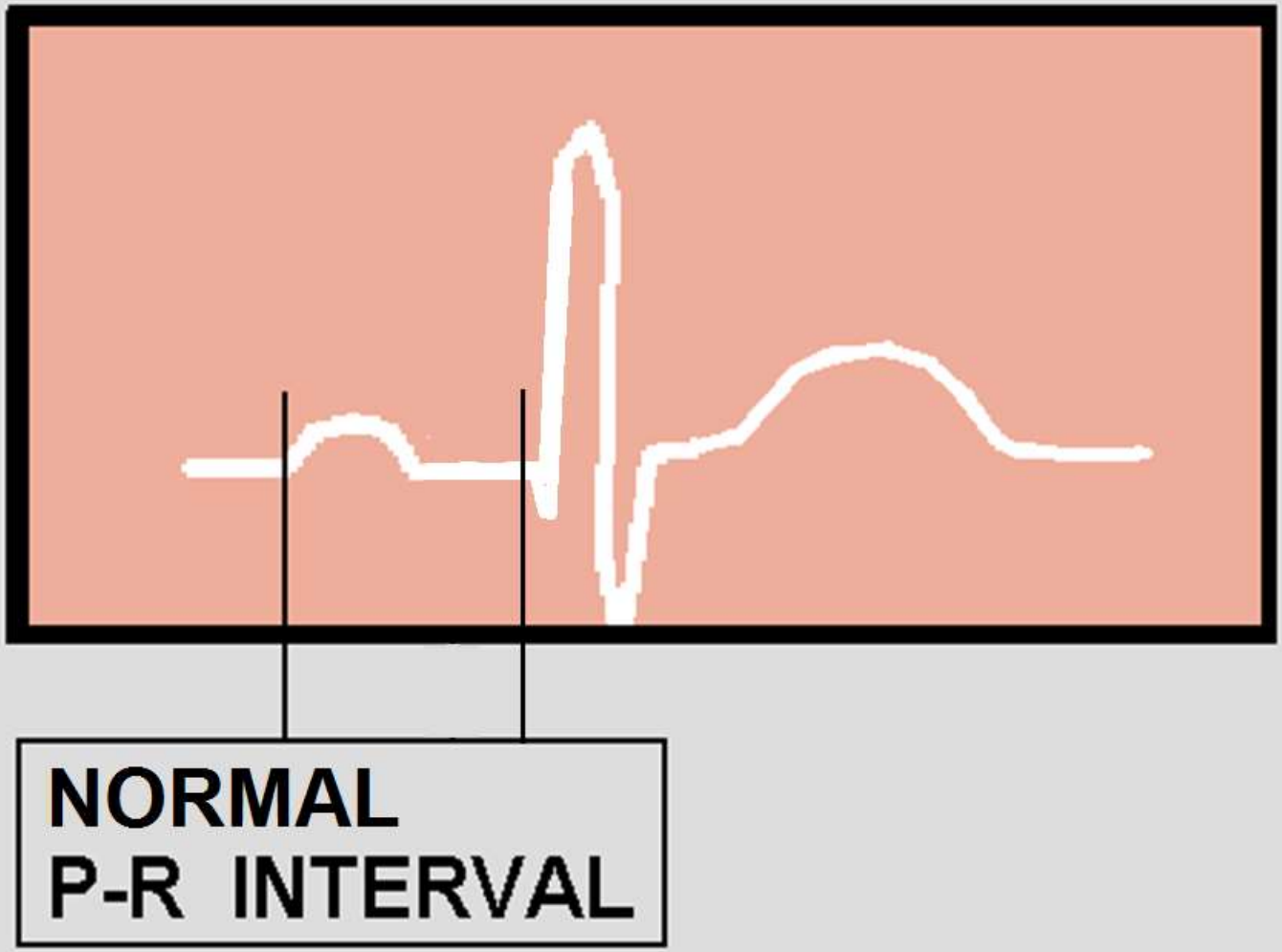


# Fibrous Skeleton of the Heart…

- Rarely taught ….. But it's so important to understanding cardiac function and ECGs…..
- It's a disk-shaped structure separating the atrium from the ventricles.
- Secures the heart valves.
- Acts as an electrical insulator, blocks electrical current……
- **An abnormal hole (BYPASS TRACT) allows current to "leak" between atrium and ventricles**

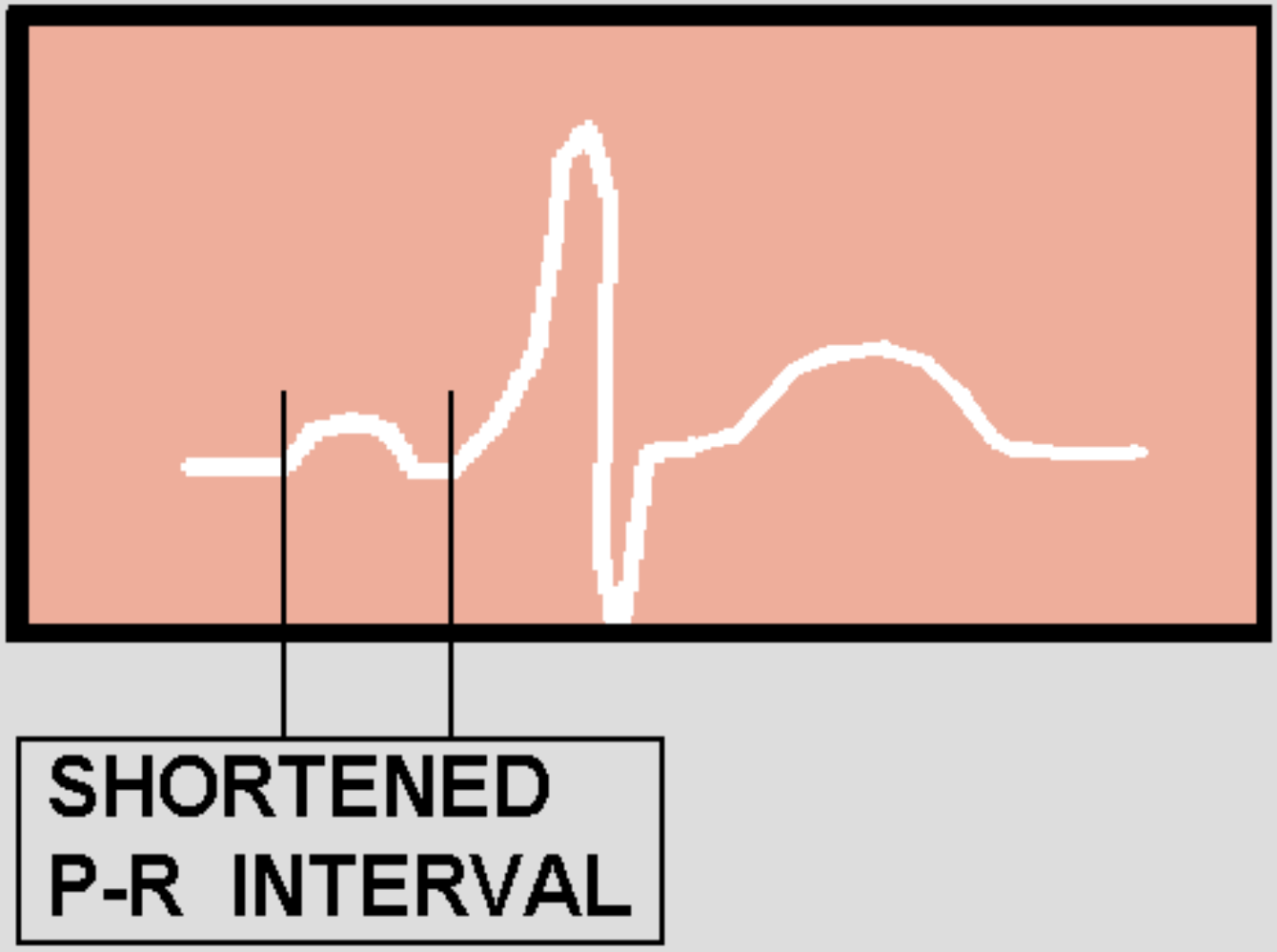
# WOLFF-PARKINSON-WHITE

## THE NORMAL ECG . . . .



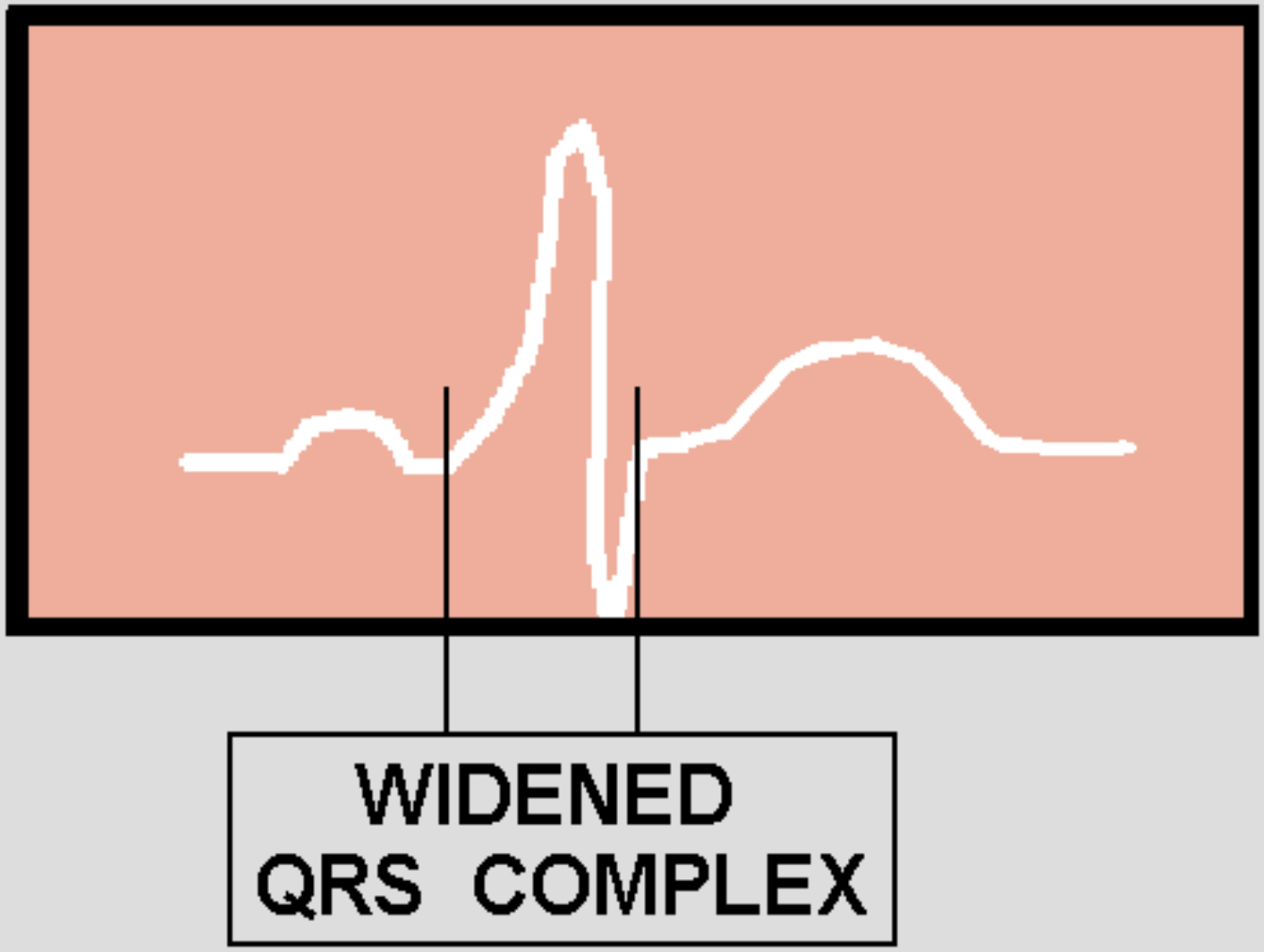
# WOLFF-PARKINSON-WHITE

## EKG CHARACTERISTICS



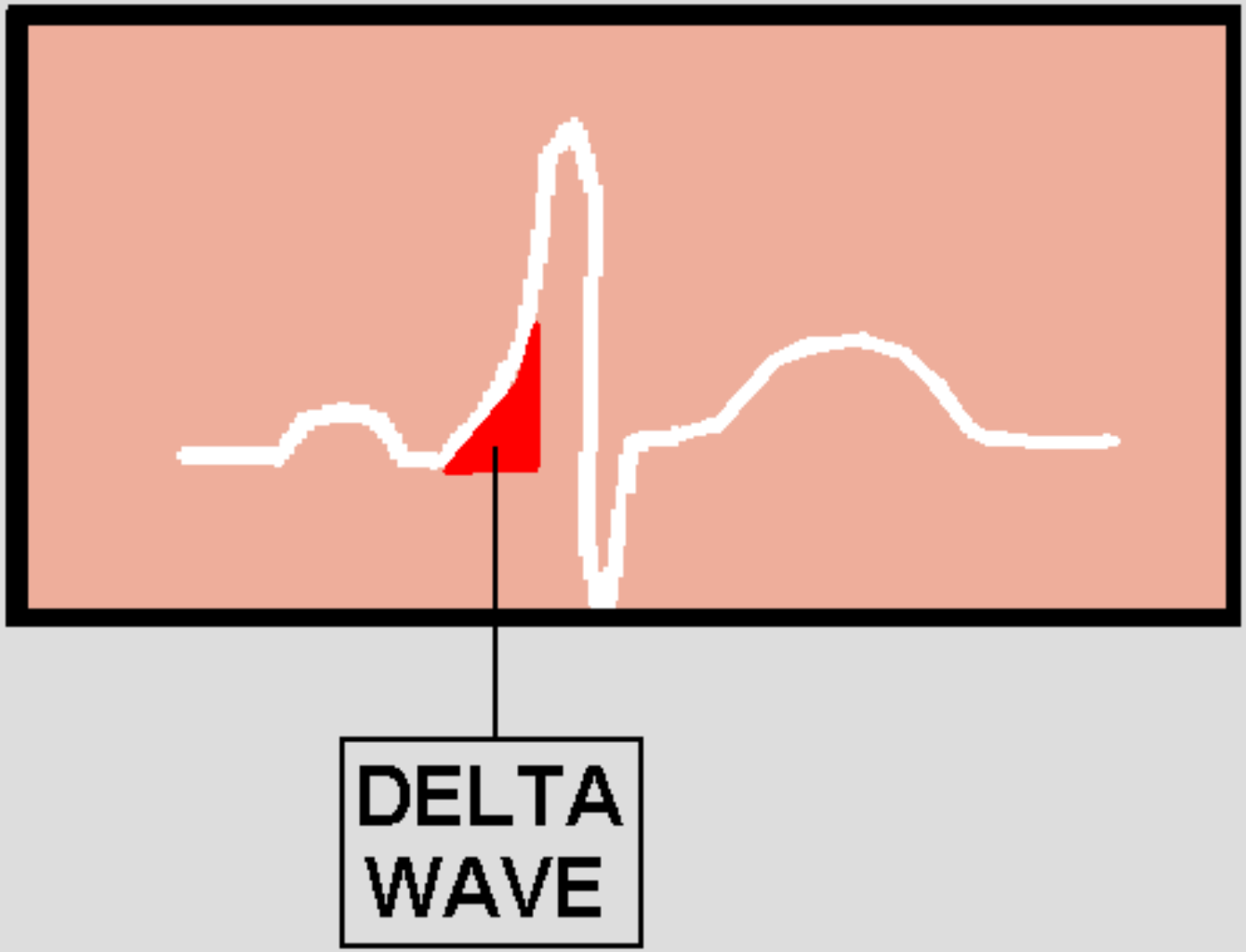
# WOLFF-PARKINSON-WHITE

## EKG CHARACTERISTICS



# WOLFF-PARKINSON-WHITE

## EKG CHARACTERISTICS



17-MAY-1997 15:32:09 ST. JOSEPH'S WOMEN'S-WOMEN' ROUTINE RETRIEVAL

16 yr
Female Caucasian

Room:REC
Loc:20 Option:50

| | | |
|---|---|---|
| Vent. rate | 92 | BPM |
| PR interval | 112 | ms |
| QRS duration | 118 | ms |
| QT/QTc | 356/440 | ms |
| P-R-T axes | 59 -22 | 107 |

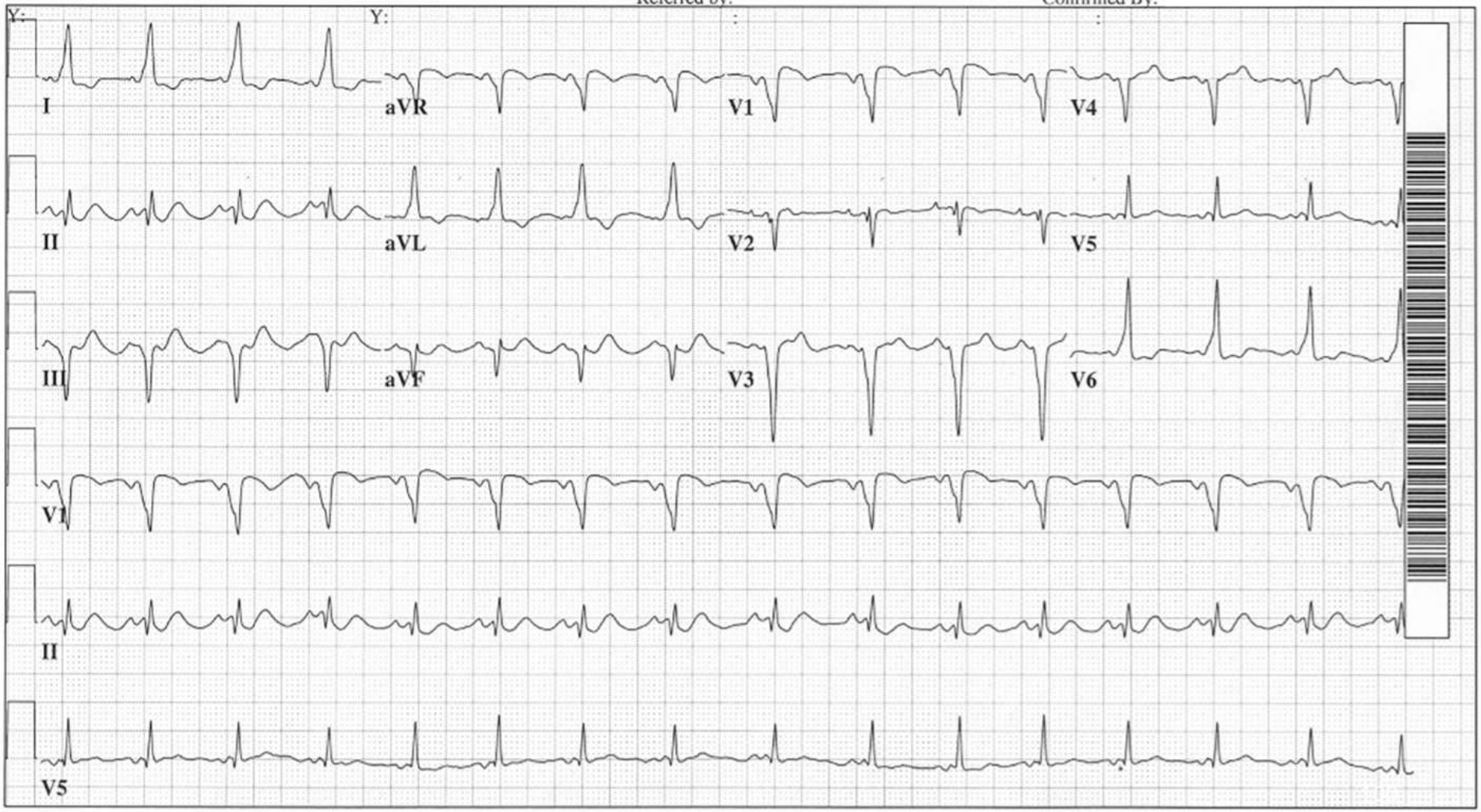
Normal sinus rhythm with sinus arrhythmia
Left atrial enlargement
Anterior infarct , age undetermined
Inferior infarct , age undetermined
ST & T wave abnormality, consider lateral ischemia
Wolff-Parkinson-White
Abnormal ECG
No previous ECGs available

History:Unknown
Technician: DP
Test ind:EKG

EKG CLASS #WR030100
60783

Referred by:

Confirmed By:




25mm/s 10mm/mV 40Hz 005C 12SL 250 CID: 12

EID:18 EDT: 16:01 17-MAY-1997 ORDER:

01-MAY-1999 04:14:17 ST. JOSEPH'S HOSPITAL-IN1464 ROUTINE RETRIEVAL

51 yr
Male Caucasian

Room:540
Loc:5 Option:28

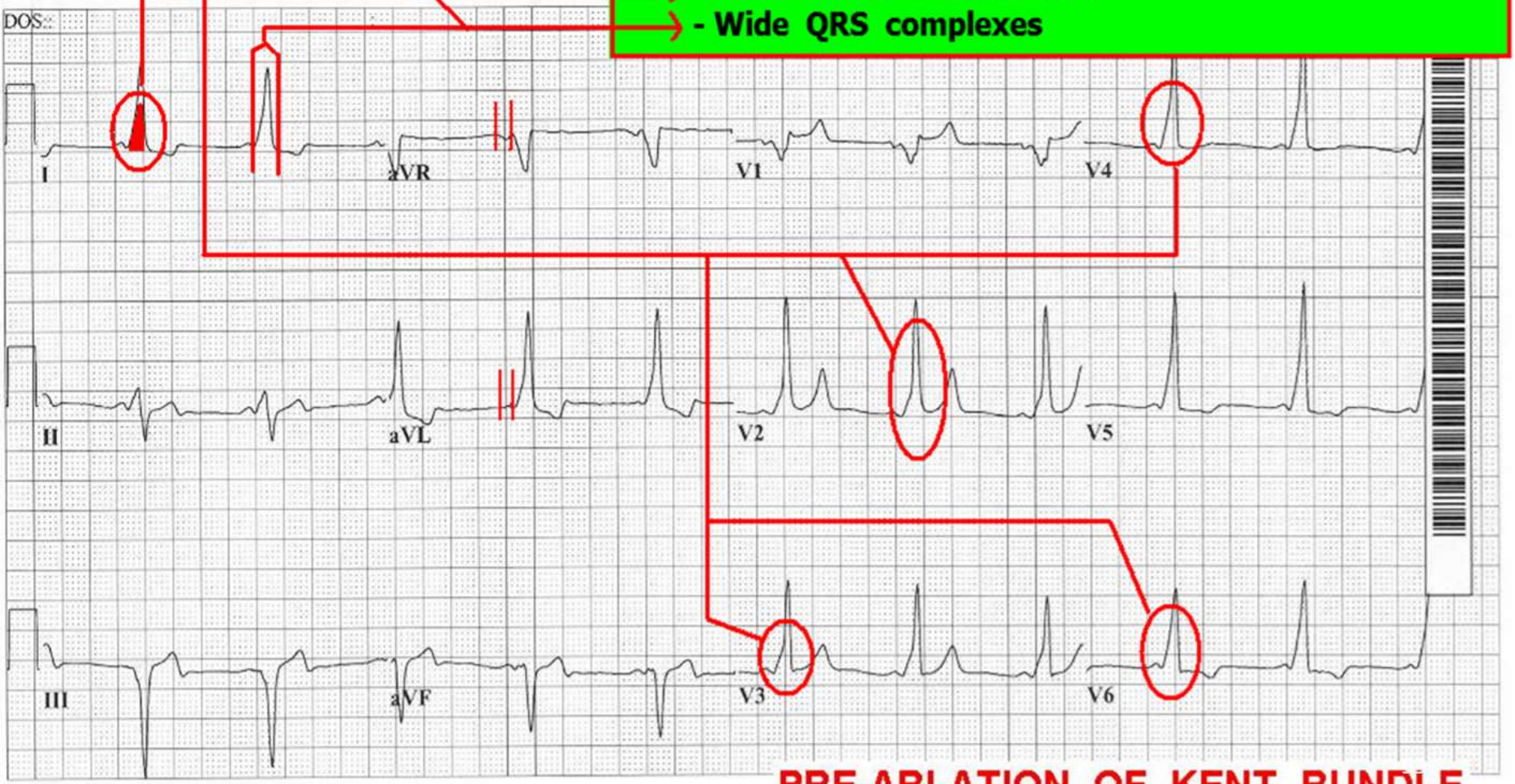
| | | |
|---|---|---|
| Vent. rate | 64 | BPM |
| PR interval | 110 | ms |
| QRS duration | 146 | ms |
| QT/QTc | 418/431 | ms |
| P-R-T axes | 50 -36 | 119 |

Normal sinus rhythm
Wolff-Parkinson-White
Abnormal ECG
No previous ECGs available

Technician: EKG CLASS #WR03696205

## 4. Wolff-Parkinson-White (WPW) type A

- Short P-R Interval
- Presence of Delta Waves
- Wide QRS complexes




25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 12

**PRE-ABLATION OF KENT BUNDLE**

16 yr
Female Caucasian

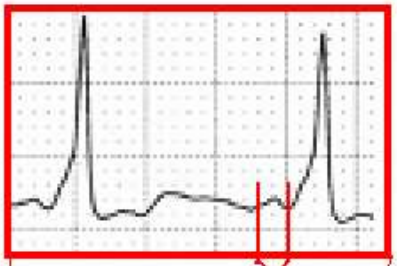
Room:REC
Loc:20 Option:50

| | | |
|---|---|---|
| Vent. rate | 92 | BPM |
| PR interval | 112 | ms |
| QRS duration | 118 | ms |
| QT/QTc | 356/440 | ms |
| P-R-T axes | 59 -22 | 107 |

Normal sinus rhythm with sinus arrhythmia
Wolff-Parkinson-White
Abnormal ECG
No previous ECGs available

History:Unknown
Technician: DP
Test ind:EKG

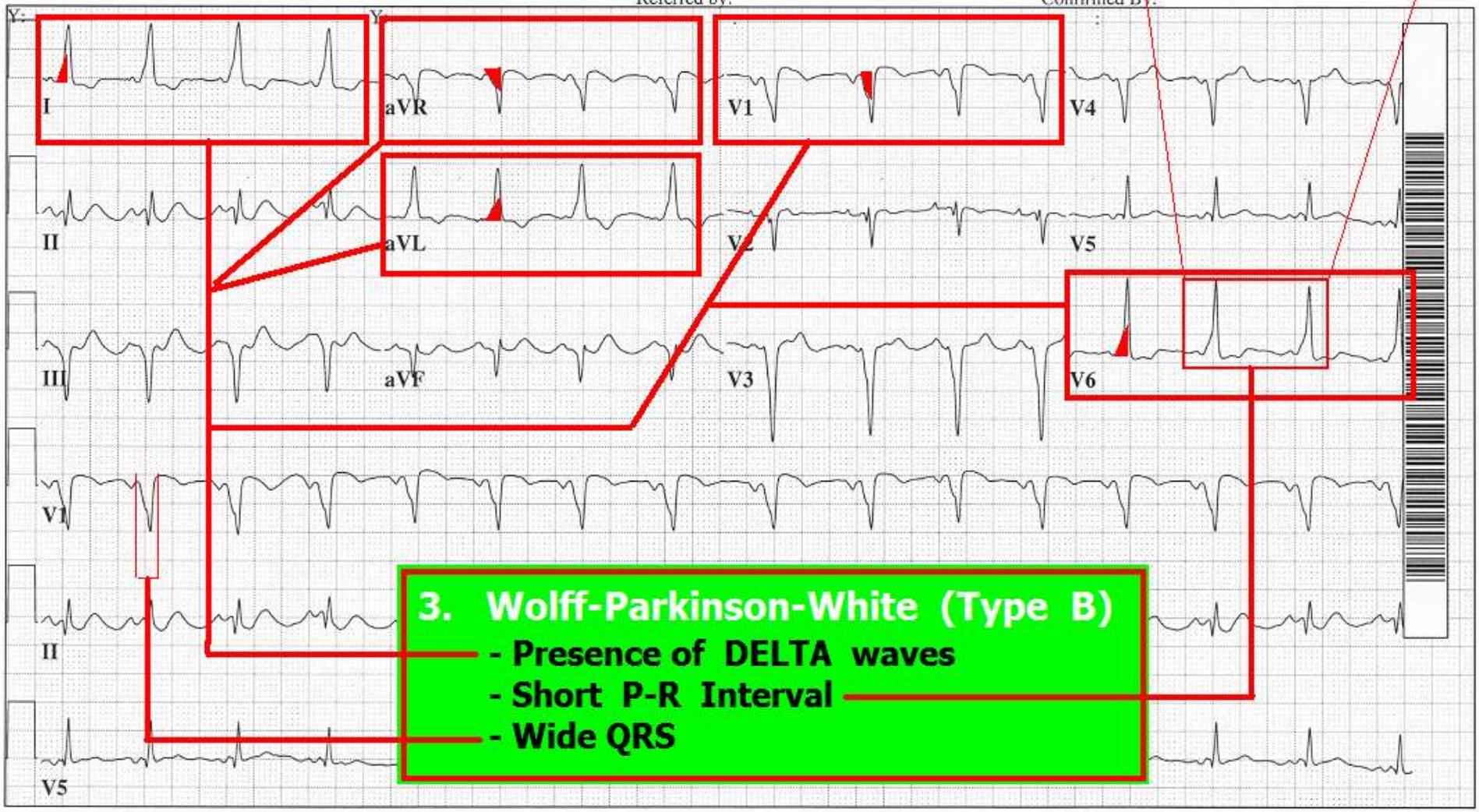
EKG CLASS #WR030100
60783



**P-R = .08**

Referred by:

Confirmed By:




**3. Wolff-Parkinson-White (Type B)**

- **Presence of DELTA waves**
- **Short P-R Interval**
- **Wide QRS**

25mm/s 10mm/mV 40Hz 005C 12SL 250 CID: 12

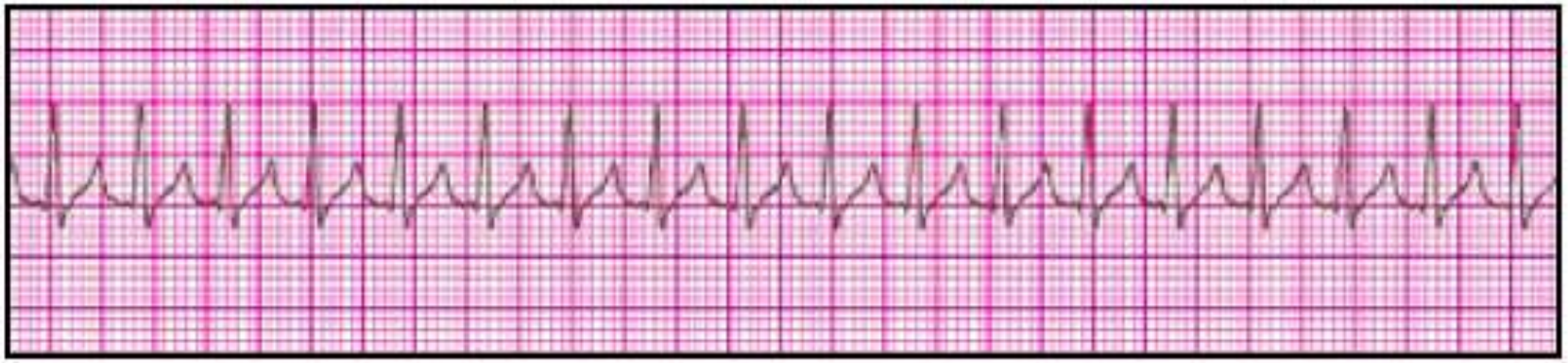
EID:18 EDT: 16:01 17-MAY-1997 ORDER:

W-P-W patients often experience Tachycardias:

- Narrow QRS Tachycardia (SVT)
- Wide QRS Tachycardia (mimics V-Tach.

The same patient can present with narrow QRS SVT, and at another time, Wide QRS Tachycardia …….

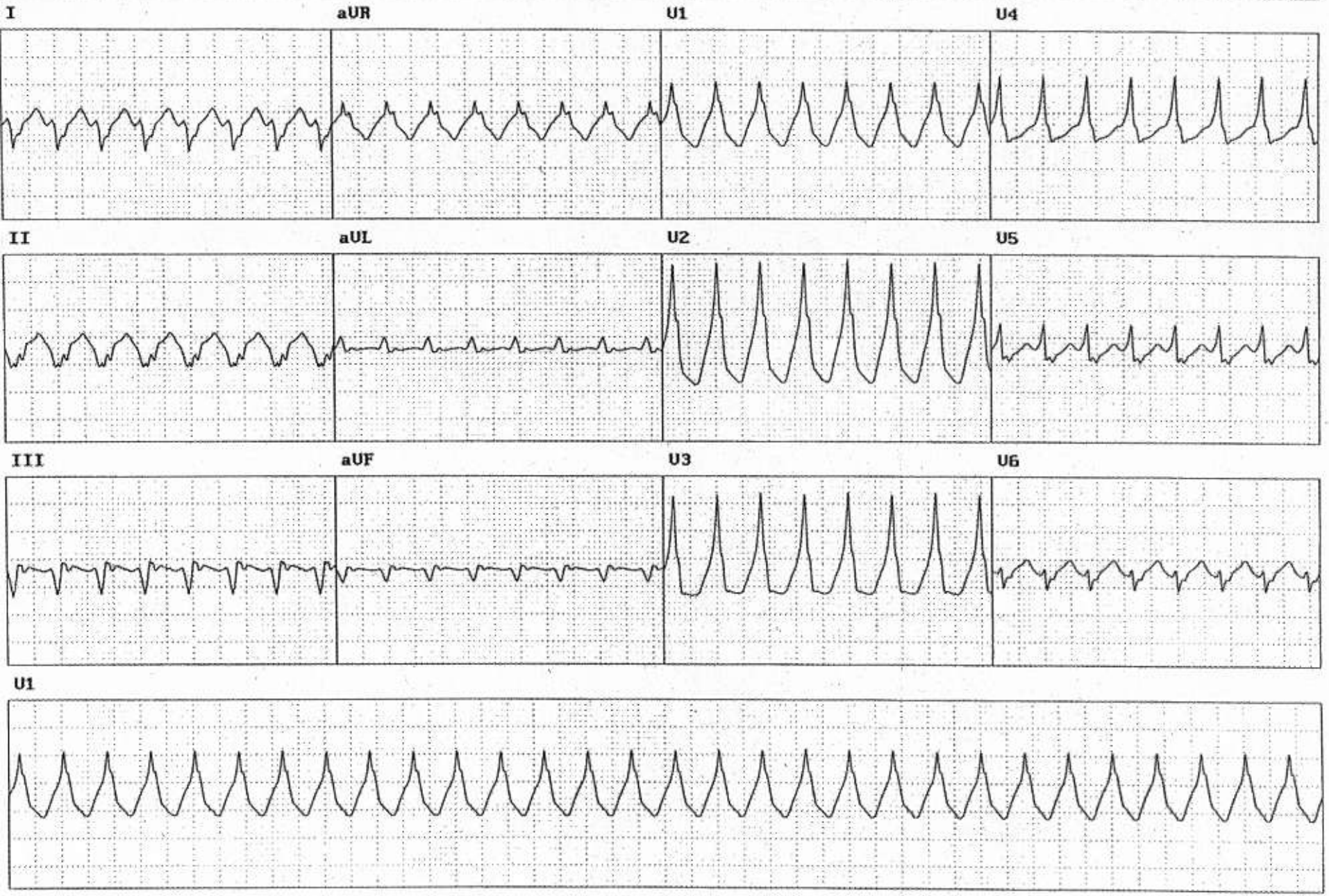
# THIS RHYTHM IS: SUPRAVENTRICULAR TACHYCARDIA (SVT)



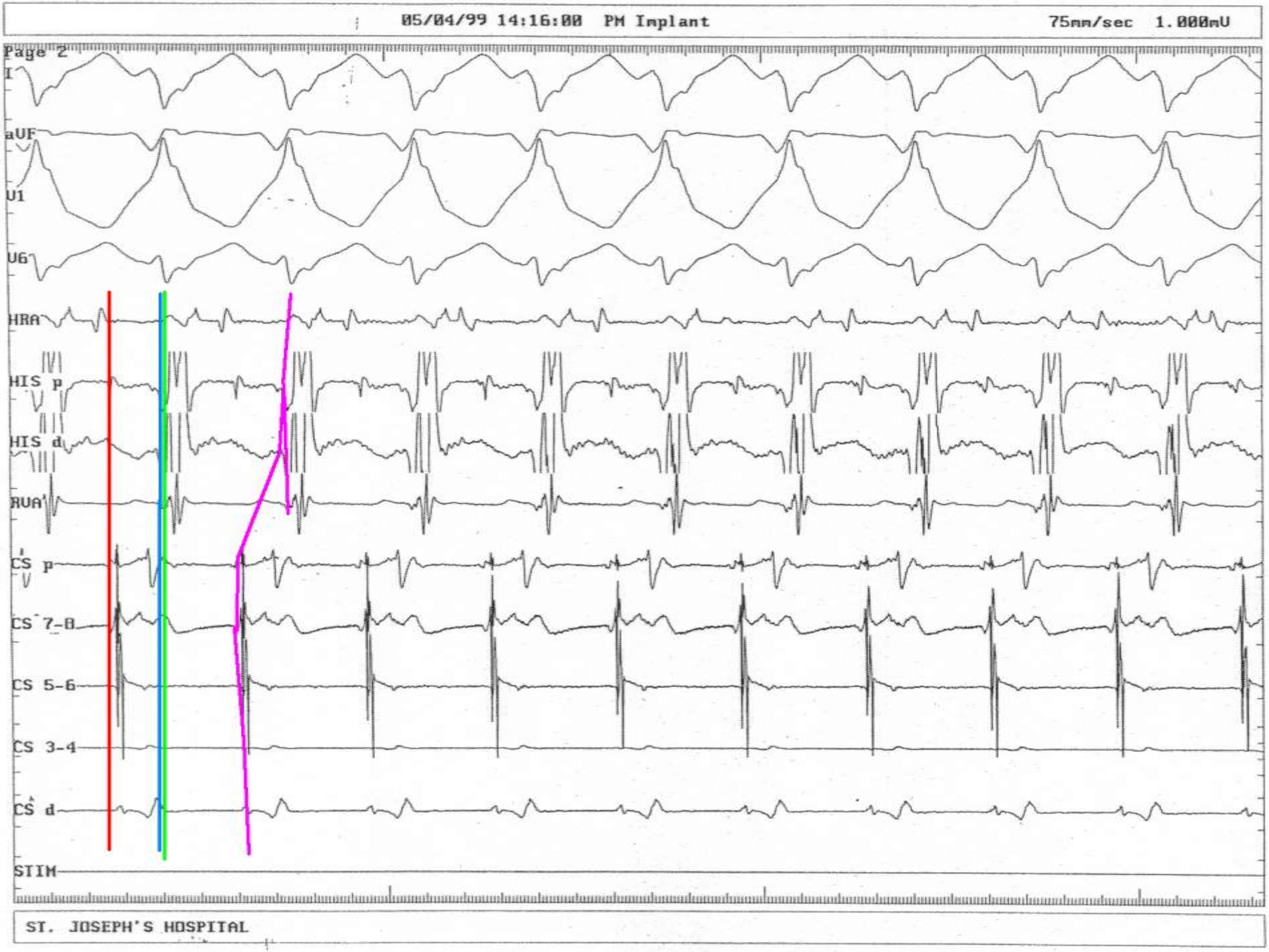
MAIN IDENTIFICATION CHARACTERISTIC(S): HEART RATE TOO FAST, USUALLY > 150. P WAVES MAY BE "BURIED" IN THE PRECEDING T WAVES. Pt USUALLY C/O "SUDDEN ONSET of HEART RACING," or "PALPITATIONS."

| | | |
|---|---|---|
| RATE ---------- | TACHYCARDIC (usually > 150) | |
| RHYTHM ---------- | REGULAR | |
| P-R INTERVAL ---- | NORMAL or ABNORMAL. | MAY BE IMPOSSIBLE TO SEE DUE TO P WAVE BURIED IN T WAVES |
| P:QRS RATIO ------ | 1:1 | |
| QRS INTERVAL ---- | NORMAL | |

05/04/99 14:16:00 25mm/sec 4.2mm/mU




ST. JOSEPH'S HOSPITAL W-P-W DOUBLE BY-PASS TRACT


05/04/99 14:16:00 PM Implant
75mm/sec 1.000mU
Page 2
I
aVF
V1
V6
HRA
HIS p
HIS d
RVA
CS p
CS 7-8
CS 5-6
CS 3-4
CS d
STIM
ST. JOSEPH'S HOSPITAL

**Patient Profile: Wolff-Parkinson-White:**

**- Typically Pediatric / Young Adult**

**- May not know they have it**

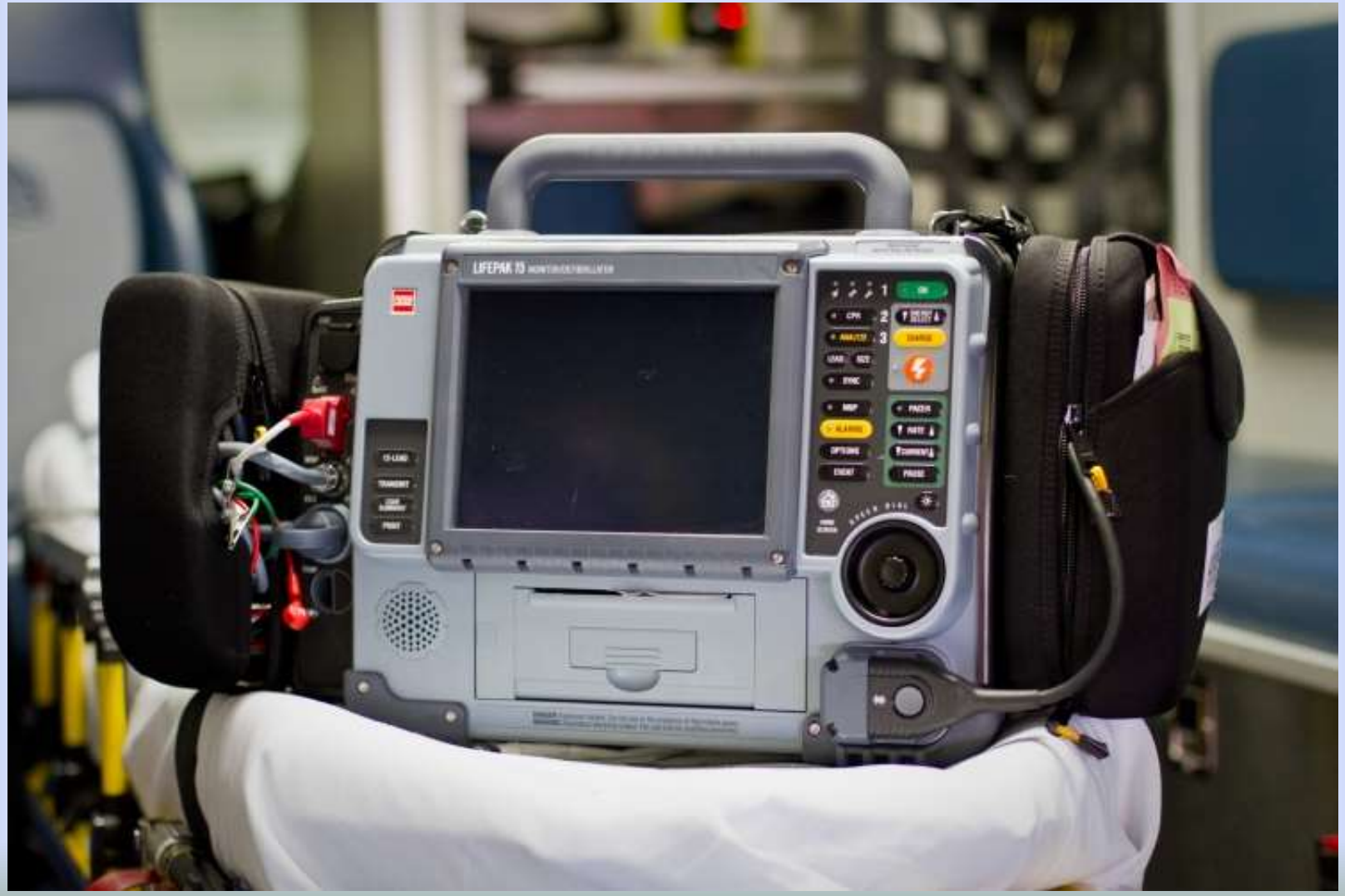
**- May experience episodes of "palpitations" or "Very Fast Heartbeat."**

**W-P-W may CAUSE A-fib with RVR.**

**Patients may present with symptoms of "palpitations," "heart racing," "light-headedness," or "passing out" . . . . . .**

OLD BARN, SHREWSBURY, PA – 2001

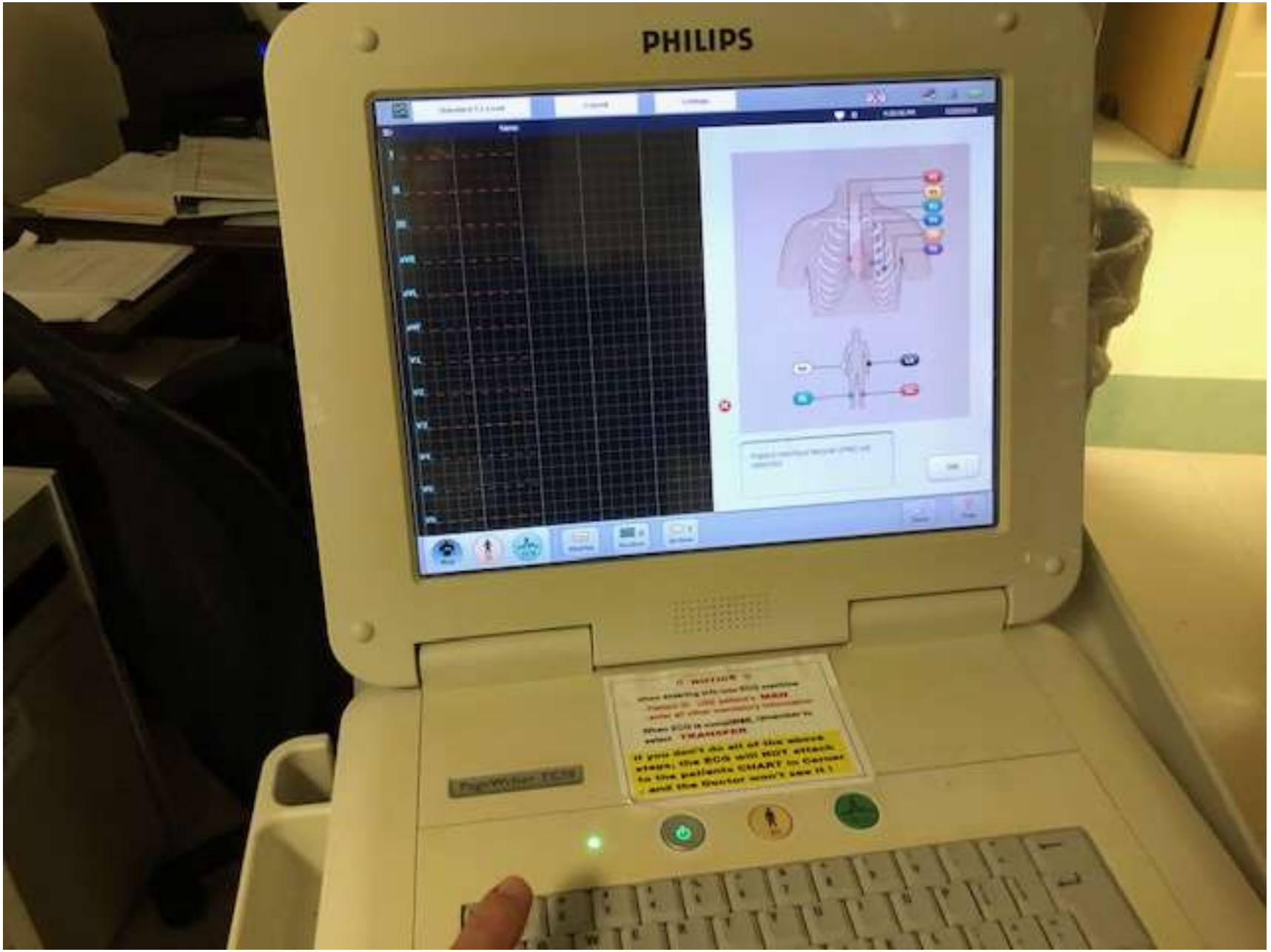
# EMS 12 Lead ECG



# 10 wires . . .

- 4 limb leads
- 6 chest (“V”) leads


Sanitas


PHILIPS

# OBTAINING
# THE
# 12 LEAD ECG

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

# Obtaining the 12 Lead ECG

- **Limb leads should be on the limbs.**

# Obtaining the 12 Lead ECG

- **Limb leads should be on the limbs.**
- **When emergency circumstances dictate that limb leads be placed on patient's torso, the words "LIMB LEADS ON PATIENT'S TORSO" should be noted on the ECG.**

# Obtaining the 12 Lead ECG

**Recent AHA/ACC/HRS literature indicates QRS AMPLITUDE, Q WAVE DURATION, AXIS and WAVEFORM DEFLECTION can be altered when limb leads are placed on the patient's torso (Mason-Likar lead placement).**

***Therefore every effort should be made to place limb leads on the limbs.***

# Recommendations for the Standardization and Interpretation of the Electrocardiogram

## Part I: The Electrocardiogram and Its Technology

affected by monitoring lead placement; however, tracings that use torso electrodes differ in important ways from the standard 12-lead ECG. In addition to body position differences that affect the ECG,[109] monitoring electrodes placed on the trunk do not provide standard limb leads, and distortion of the central terminal alters the augmented limb leads and the precordial leads.[110,111] Tracings with Mason-Likar and other alternative lead placement may affect QRS morphology more than repolarization compared with the standard ECG; these differences can include false-negative and false-positive infarction criteria.[81,112] Motion artifact of the limbs is a particular problem for routine recording in neonates, infants, and

**AHA/ACC/HRS Scientific Statement**

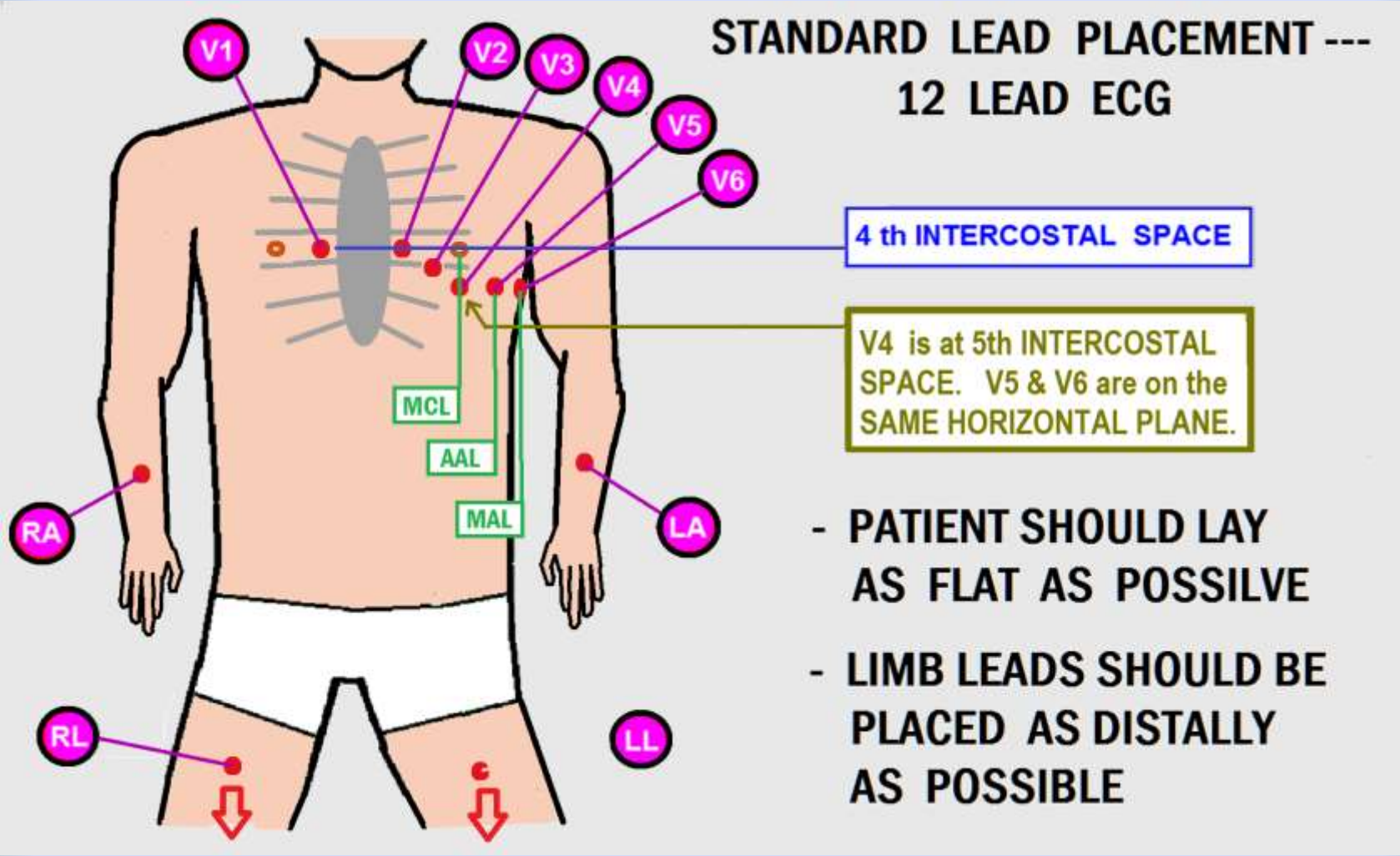
# Recommendations for the Standardization and Interpretation of the Electrocardiogram

## Part I: The Electrocardiogram and Its Technology

***Recommendations***

ECGs recorded with torso placement of the extremity electrodes cannot be considered equivalent to standard ECGs for all purposes and should not be used interchangeably with standard ECGs for serial comparison. Evaluation of the effect of torso placement of limb leads on waveform amplitudes and

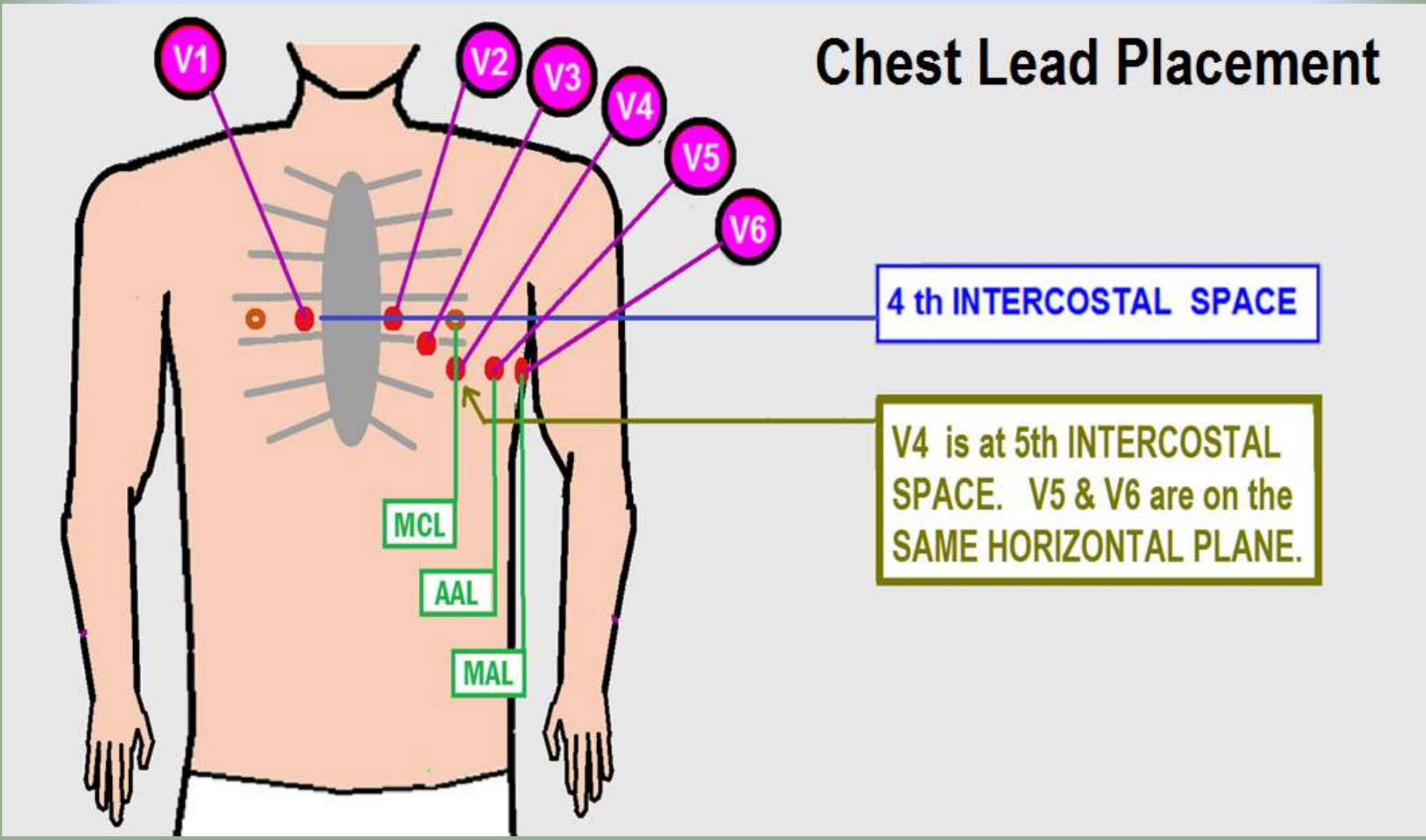
# Obtaining the 12 Lead ECG



# Leads V1 & V2 on 12 Lead ECG:

- **Proper lead placement of precordial Leads V1 and V2 are 4th intercostal space on opposite sides of the sternum.**
- **Incorrect placement of Leads V1 and V2 will result in: reduction of R wave amplitude (resulting in poor R wave progression) leading to misdiagnosis of previous anterior / septal infarction.**

# CORRECT Lead placement:



[redacted] [redacted] [redacted]

DOB [redacted] 75 Years Female (2)

Rate 76 . Sinus rhythm.....normal P axis, V-rate 50- 99

PR 161
QRSD 90
QT 350
QTc 394

--AXIS--
P 50
QRS 51
T 44

12 Lead; Standard Placement

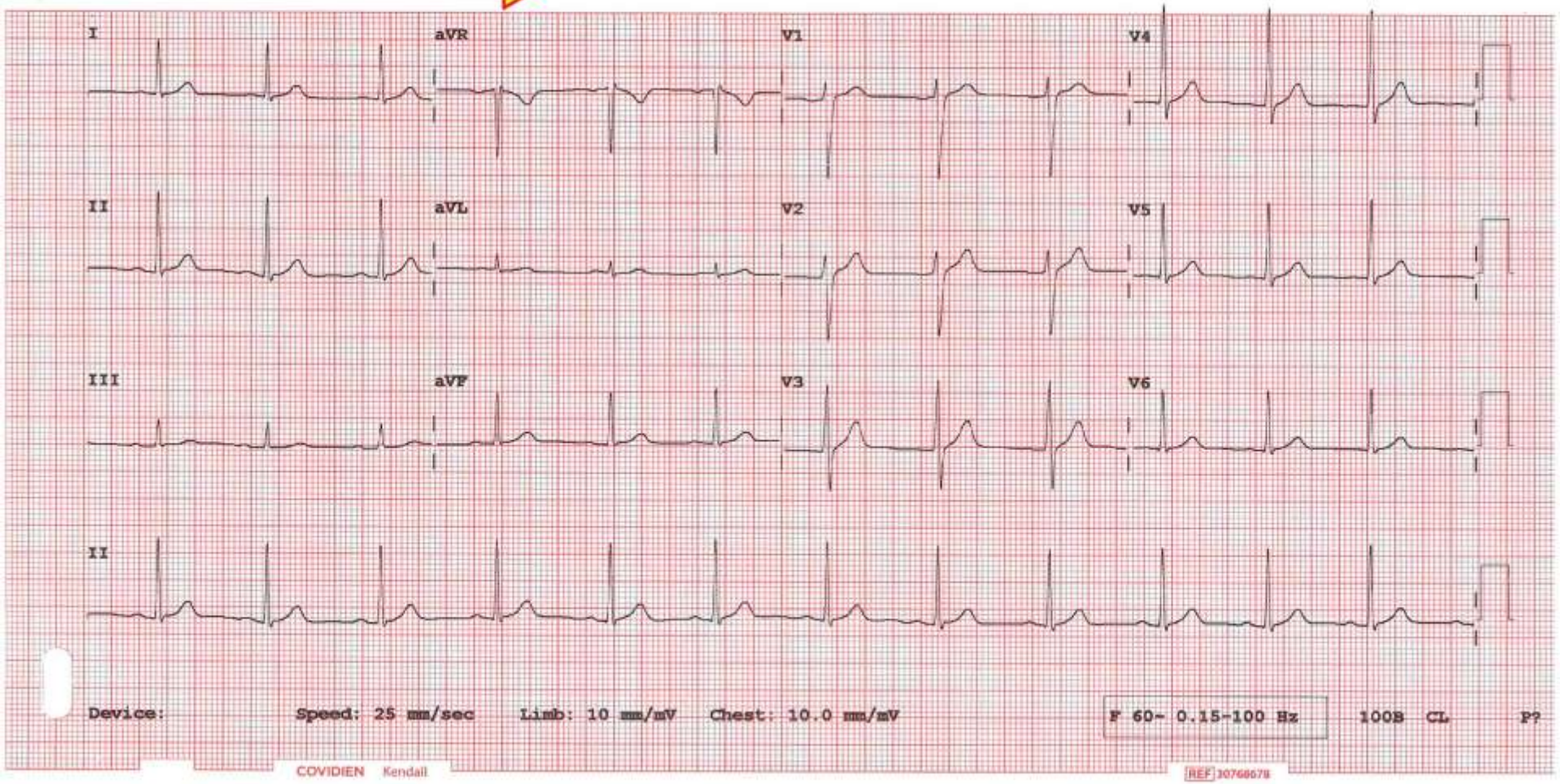
[redacted] TECH SD

[redacted]

- NORMAL ECG -

Unconfirmed Diagnosi

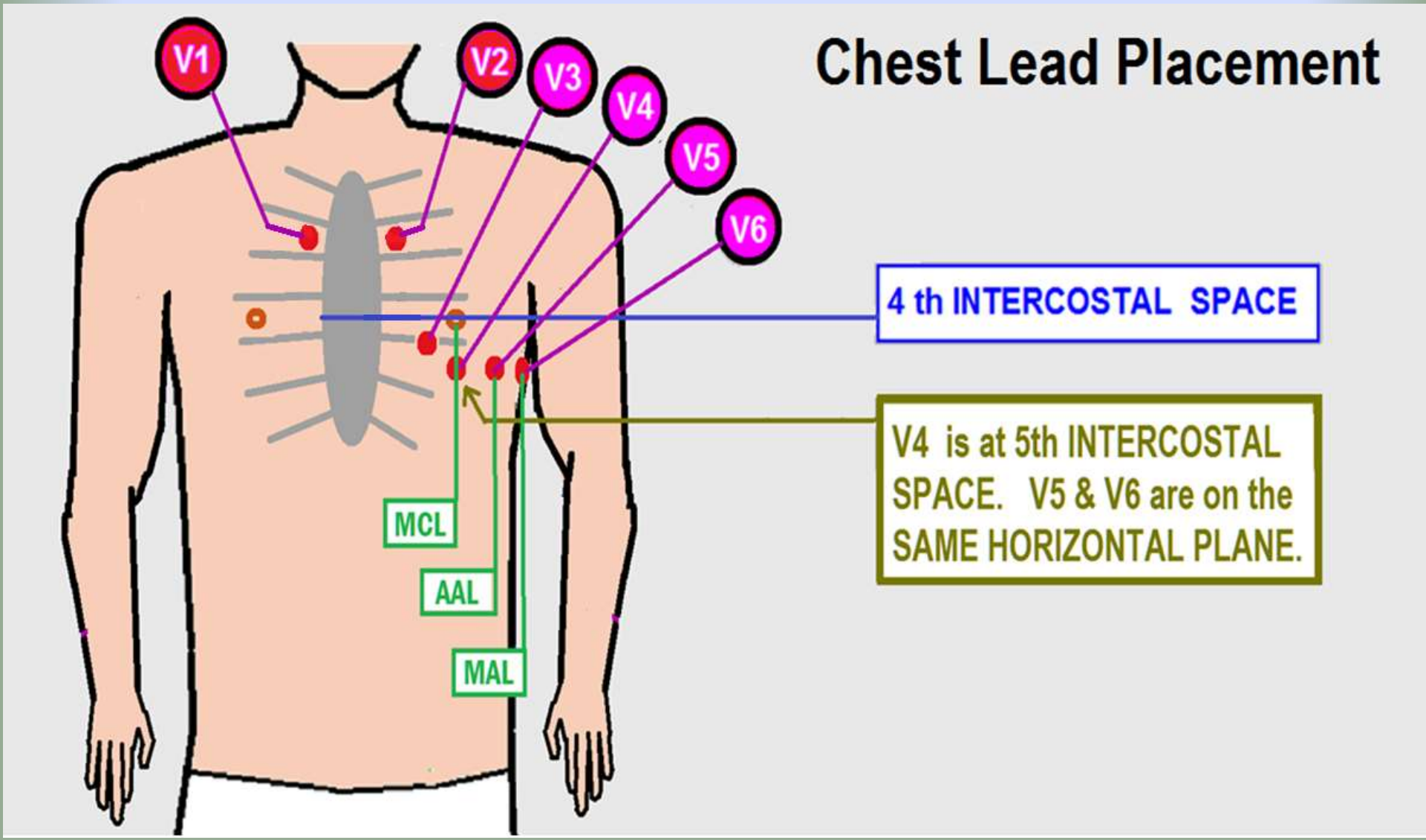
[redacted]




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

COVIDIEN Kendall REF 30768678

# **INCORRECT** Lead placement:



[redacted]

DOB [redacted]1988 30 Years [redacted] Female [redacted] 5:20:58 AM

(1)

3NE

Rate 89 . Sinus rhythm.....normal P axis. V-rate 50- 99

Anteroseptal infarct, age indeterminate.....Q >35mS

PR 157
QRSD 96
QT 365
QTc 445

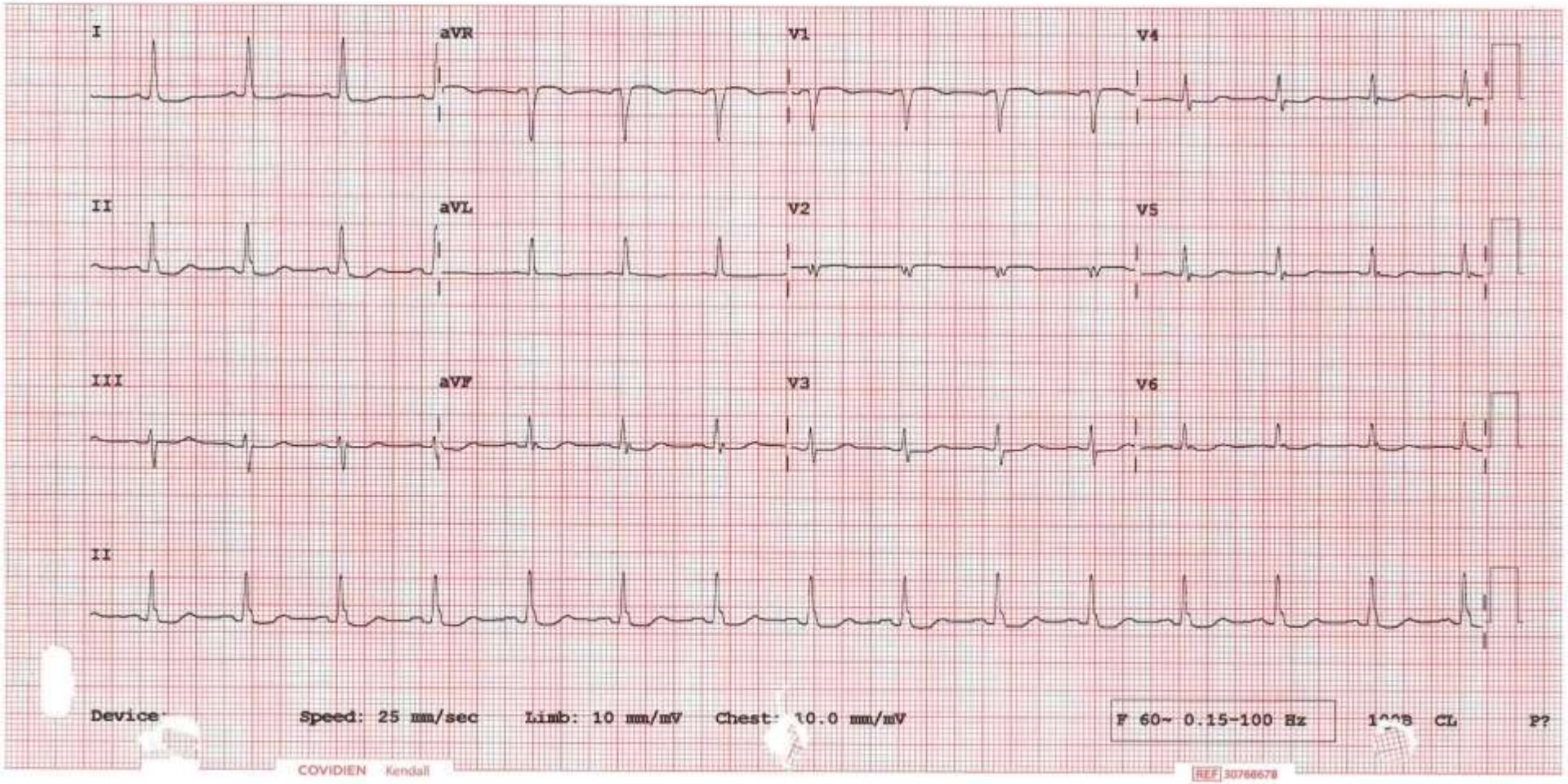
--AXIS--
P 46
QRS 24
T 86

12 Lead; Standard Placement

- ABNORMAL ECG -

Unconfirmed Diagnosis

[redacted]



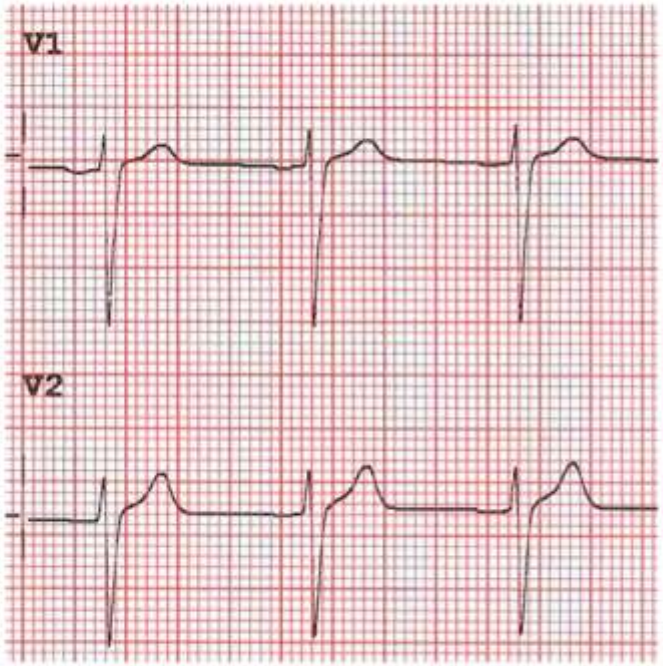
# AHA/ACC/HRS Scientific Statement

## Recommendations for the Standardization and Interpretation of the Electrocardiogram

### Part I: The Electrocardiogram and Its Technology

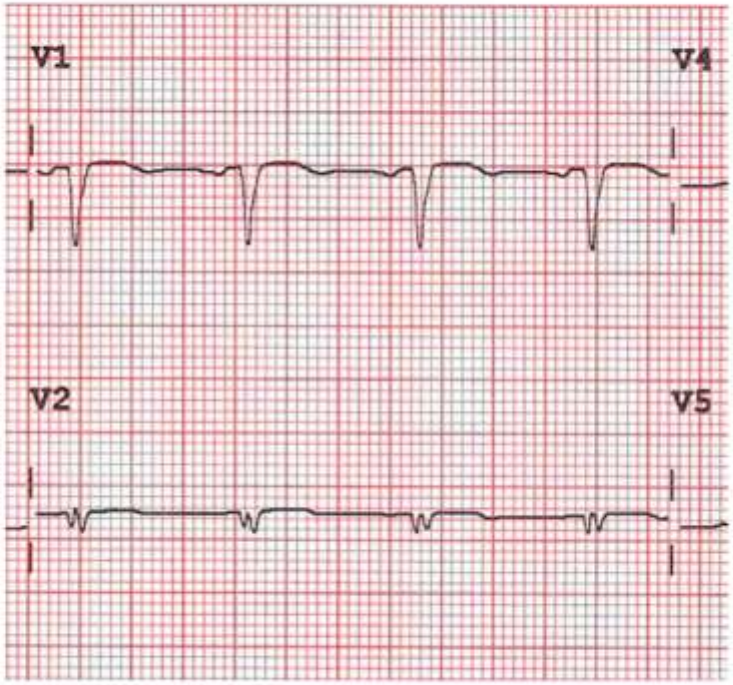
the often profound alterations in waveforms that can result from precordial electrode misplacement.[85,86] A common error is superior misplacement of $V_1$ and $V_2$ in the second or third intercostal space. This can result in reduction of initial R-wave amplitude in these leads, approximating 0.1 mV per interspace, which can cause poor R-wave progression or erroneous signs of anterior infarction.[87] Superior displacement of the $V_1$ and $V_2$ electrodes will often result in rSr′ complexes with T-wave inversion, resembling the complex in lead aVR. It also has been shown that in patients with low diaphragm position, as in obstructive pulmonary disease,[88,89]

## Correct Lead Placement




**RS = NO old MI**

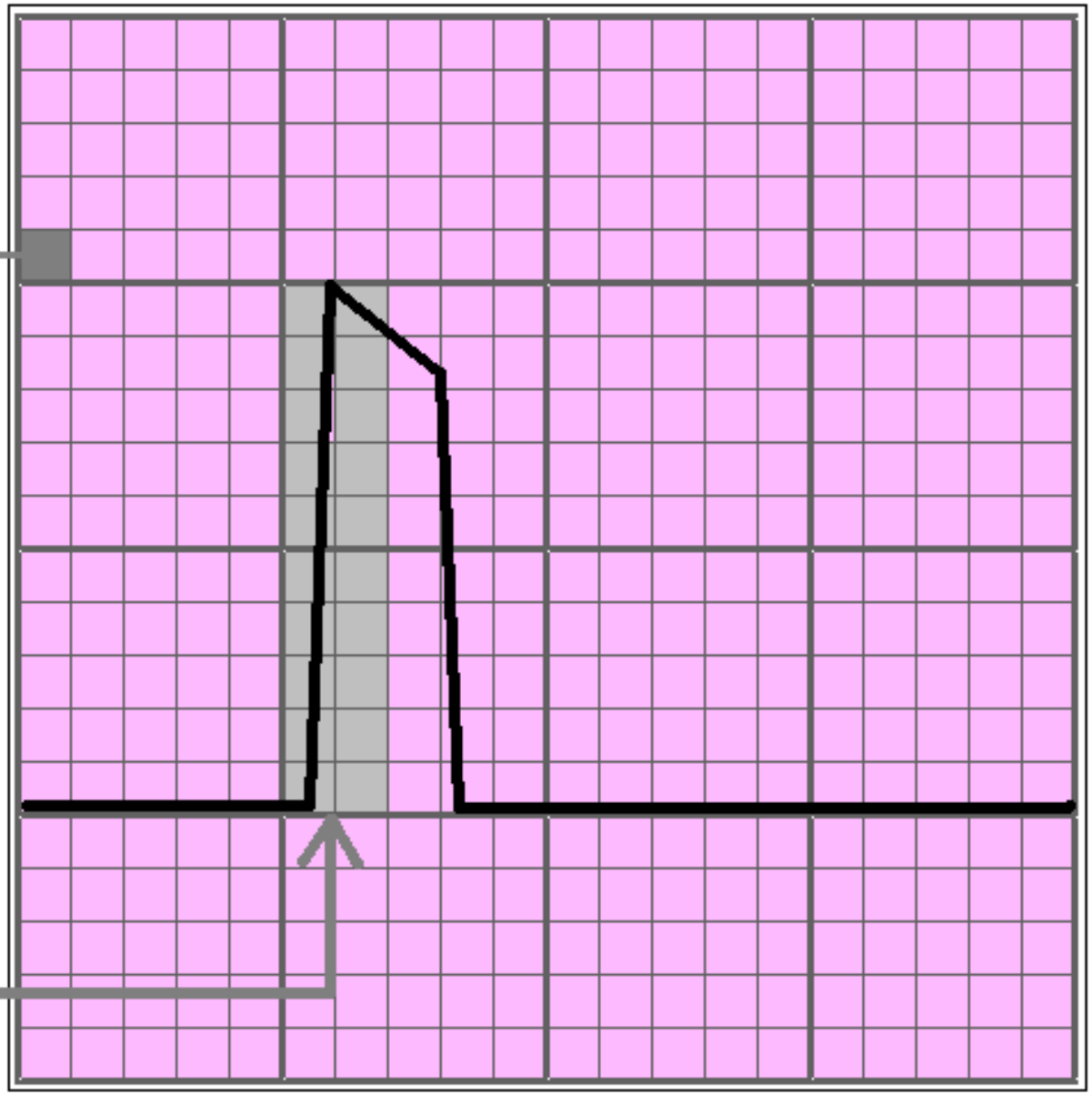
## Incorrect Lead Placement




**QS = old MI**

# ECG PAPER - THE VERTICAL AXIS:

- SMALL BOXES = 1mm SQUARES
- THE VERTICAL AXIS REPRESENTS AMPLITIUDE (VOLTAGE)
- IN VERTICAL DIRECTION, THERE ARE 5 SMALL BOXES IN EACH LARGE (5mm) BOX
- 1 mv CALIBRATION SPIKE = 10 mm



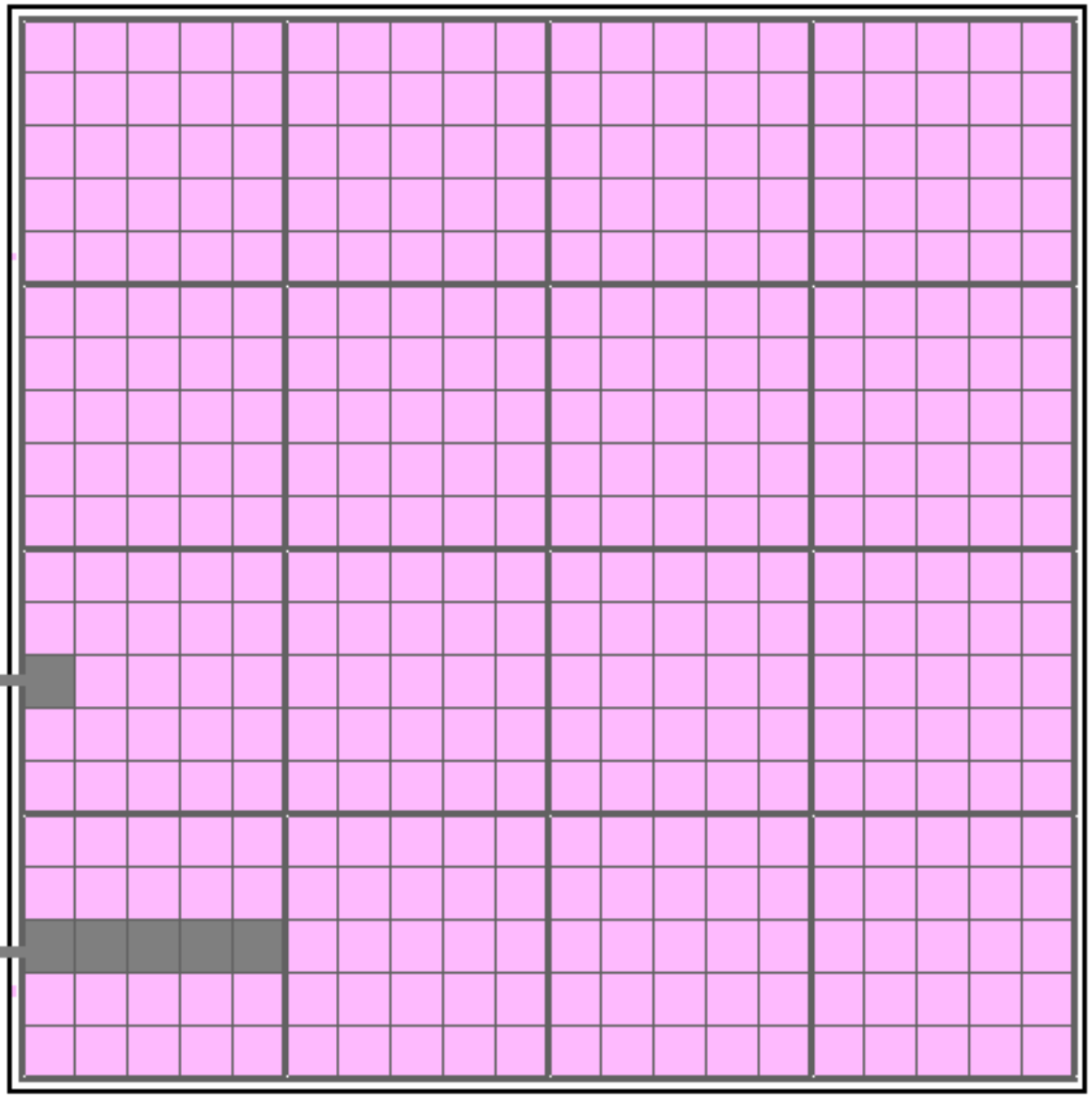
# ECG PAPER - THE HORIZONTAL AXIS:

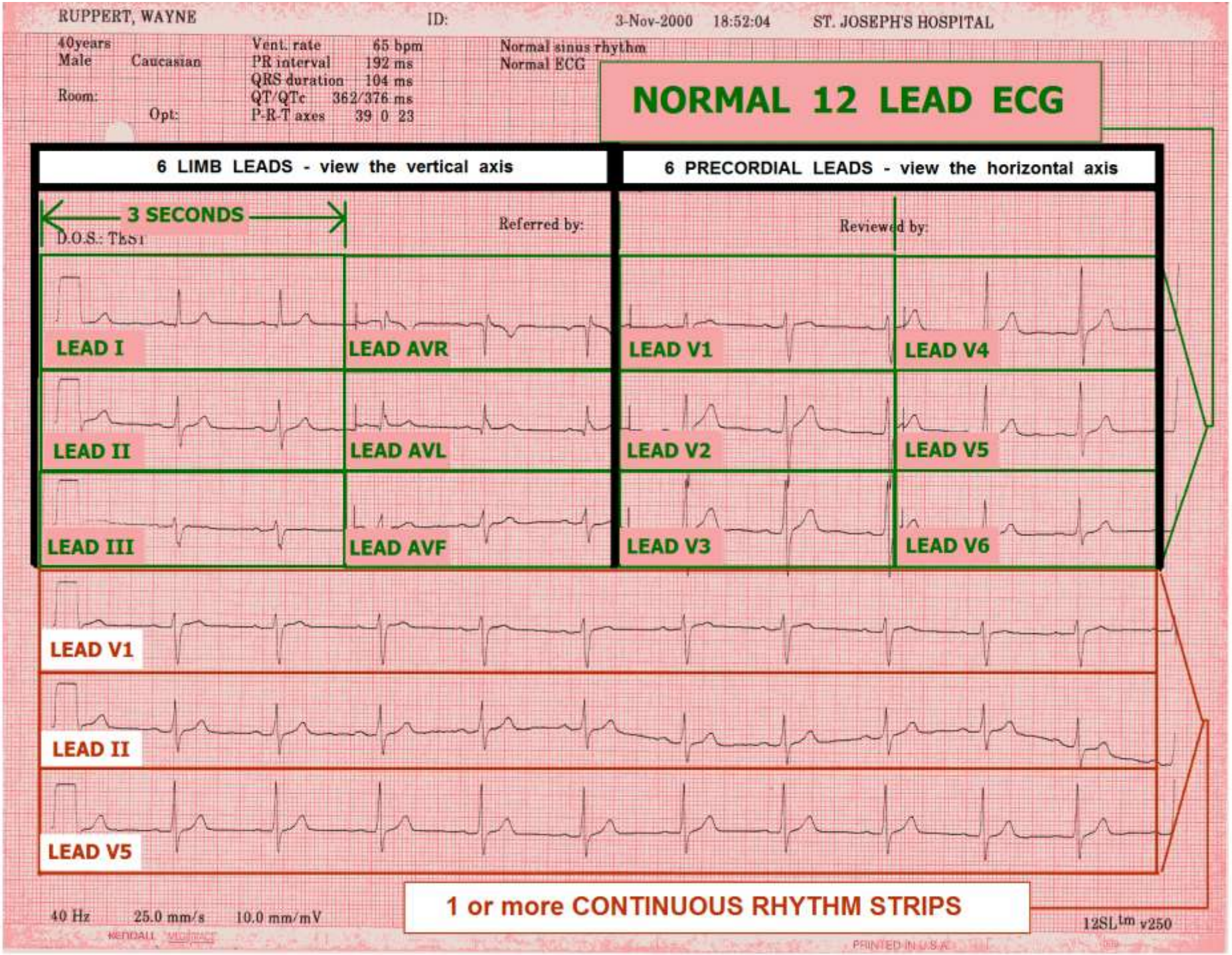
THE HORIZONTAL AXIS REPRESENTS TIME . . .

STANDARD SPEED FOR RECORDING ADULT EKGs = 25 mm / SECOND

EACH 1mm BOX = .04 SECONDS, or 40 MILLISECONDS (40 ms)

5 SMALL BOXES = .20 SECONDS, or 200 MILLISECONDS (200 ms)




RUPPERT, WAYNE
ID:
3-Nov-2000 18:52:04
ST. JOSEPH'S HOSPITAL
40years
Male
Caucasian
Room:
Opt:
Vent. rate 65 bpm
PR interval 192 ms
QRS duration 104 ms
QT/QTc 362/376 ms
P-R-T axes 39 0 23
Normal sinus rhythm
Normal ECG
NORMAL 12 LEAD ECG
6 LIMB LEADS - view the vertical axis
6 PRECORDIAL LEADS - view the horizontal axis
3 SECONDS
D.O.S.: Tbs1
Referred by:
Reviewed by:
LEAD I
LEAD AVR
LEAD V1
LEAD V4
LEAD II
LEAD AVL
LEAD V2
LEAD V5
LEAD III
LEAD AVF
LEAD V3
LEAD V6
LEAD V1
LEAD II
LEAD V5
40 Hz
25.0 mm/s
10.0 mm/mV
1 or more CONTINUOUS RHYTHM STRIPS
12SL tm v250
KENDALL
PRINTED IN U.S.A.

[redacted]

DOB [redacted] 75 Years

**Evaluate this EKG for each of the "Normal" criteria seen on the NEXT 2 SLIDES . . . . . .**

Rate 76 . Sinus rhythm.

PR 161
QRSD 90
QT 350
QTc 394

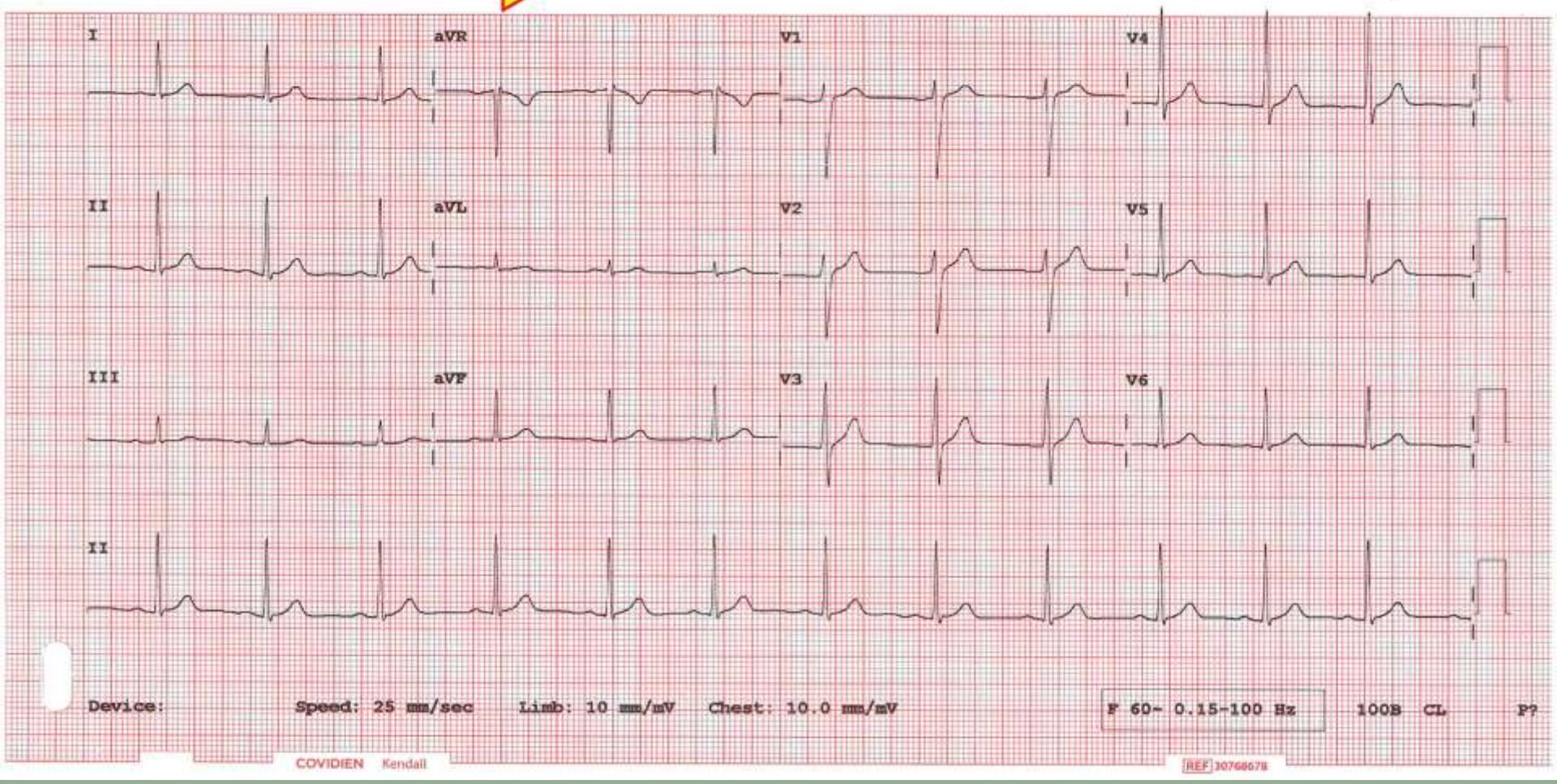
--AXIS--
P 50
QRS 51
T 44

12 Lead; Standard Placement

- NORMAL ECG -

Unconfirmed Diagnosi

[redacted]



# The Normal 12 Lead EKG

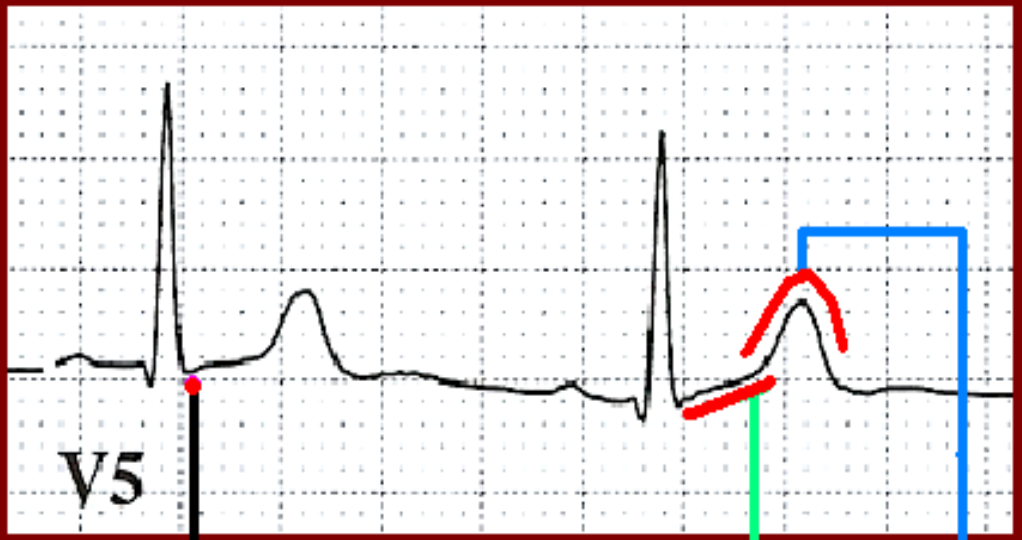
- NSR (rate 60-100, regular rhythm)
- P Waves upright all leads except aVR
- P Waves inverted lead aVR, possibly V1
- QRS upright Leads I, II, III, aVL, aVF, V5, V6
- QRS inverted Leads aVR, V1, V2
- QRS biphasic: Leads V3, V4
- P wave size:  up to 2mm tall, 2.5mm long
- QRS height Limb Leads: 5-15mm tall
- QRS height V Leads 10-15mm tall
- QRS width: not to exceed 3mm  (120 ms)
- Overall QRS Amplitude:  not greater than 30mm

# The Normal 12 Lead EKG

- T waves – Upright all Leads except aVR
- T wave – Inverted in Lead aVR
- (everything is inverted in lead aVR)
- T wave MAY be inverted (as a normal variant) in Leads III and aVL.
- Overall QRS Amplitude:  not greater than 30mm

# NORMAL ST - T WAVES

- WHEN QRS WIDTH IS NORMAL ( < 120 ms )




## *ASSESS:*

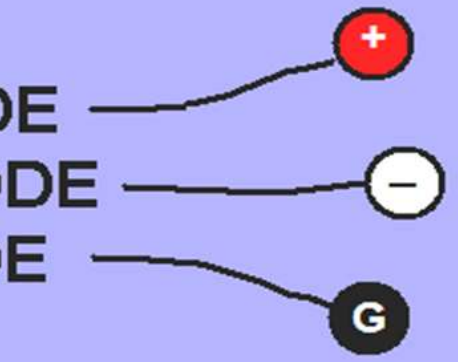
- J POINT: ISOELECTRIC ( or < 1 mm dev. )
- ST SEG: SLIGHT, POSITIVE INCLINATION
- T WAVE: UPRIGHT, POSITIVE

☞ *in EVERY LEAD EXCEPT aVR ! !*

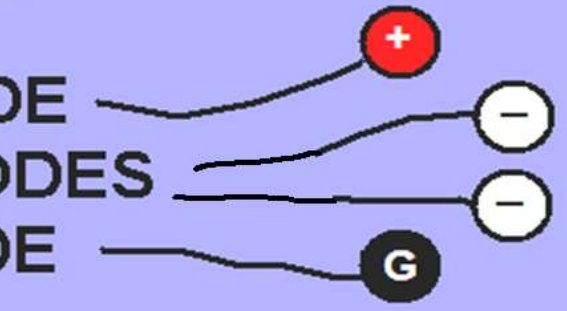
# THE ECG MACHINE

STANDARD 12 LEADS - USES 10 WIRES
( 6 CHEST and 4 LIMB )

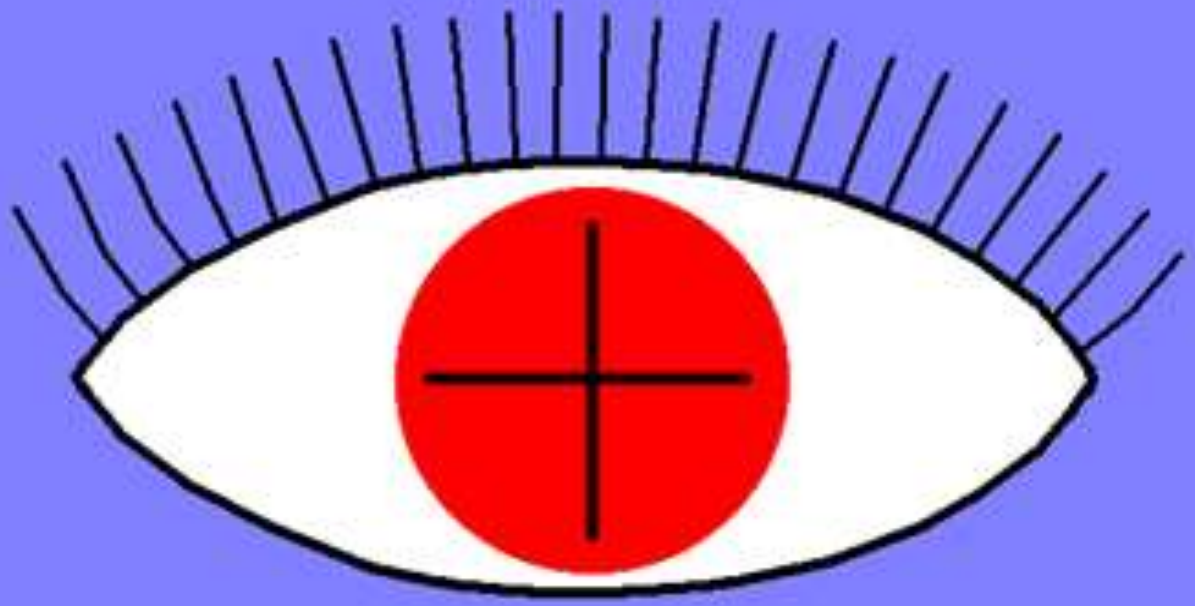
- LEADS I, II, III, and V1, V2, V3, V4, V5, V6



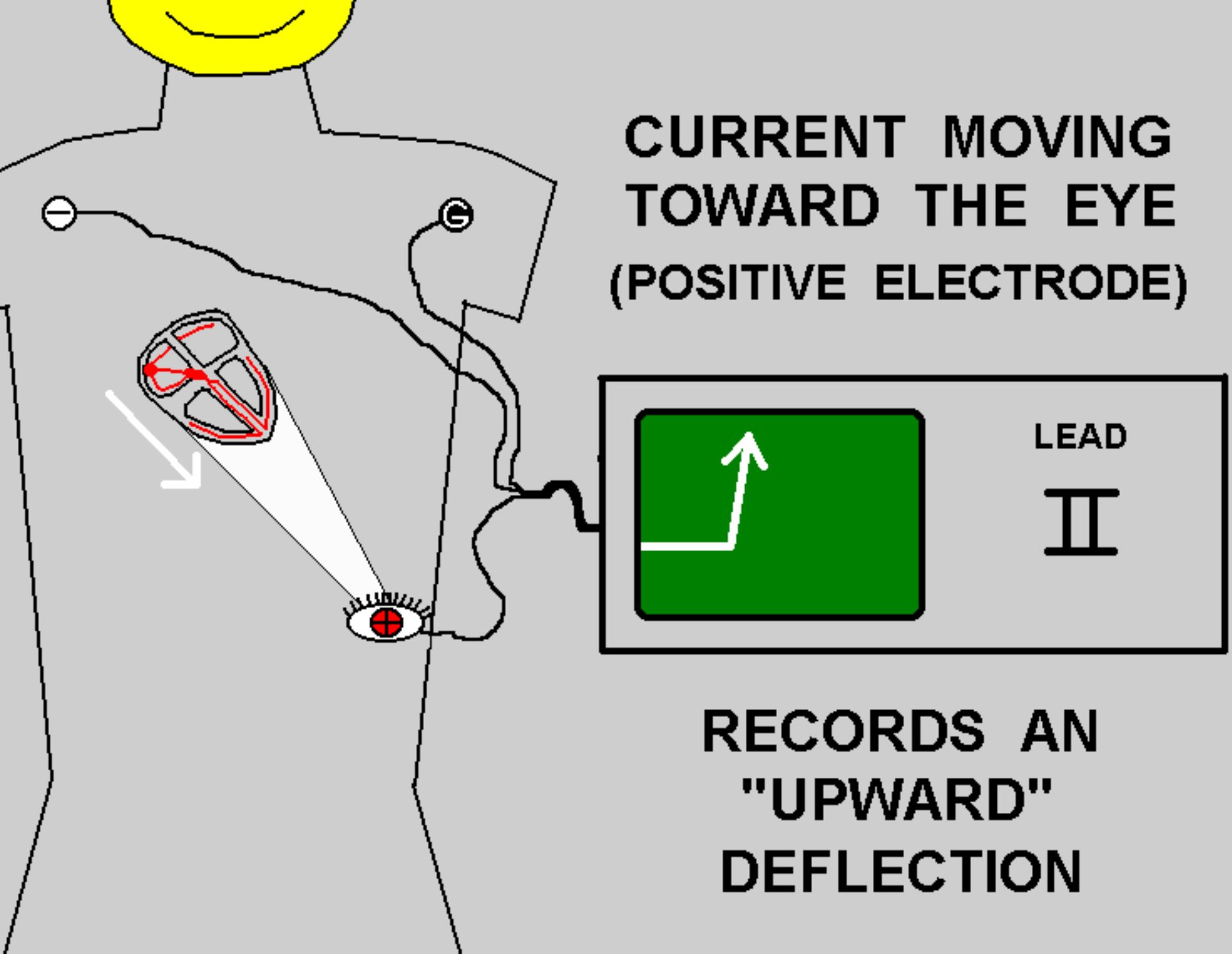

- LEADS AVR, AVL, and AVF

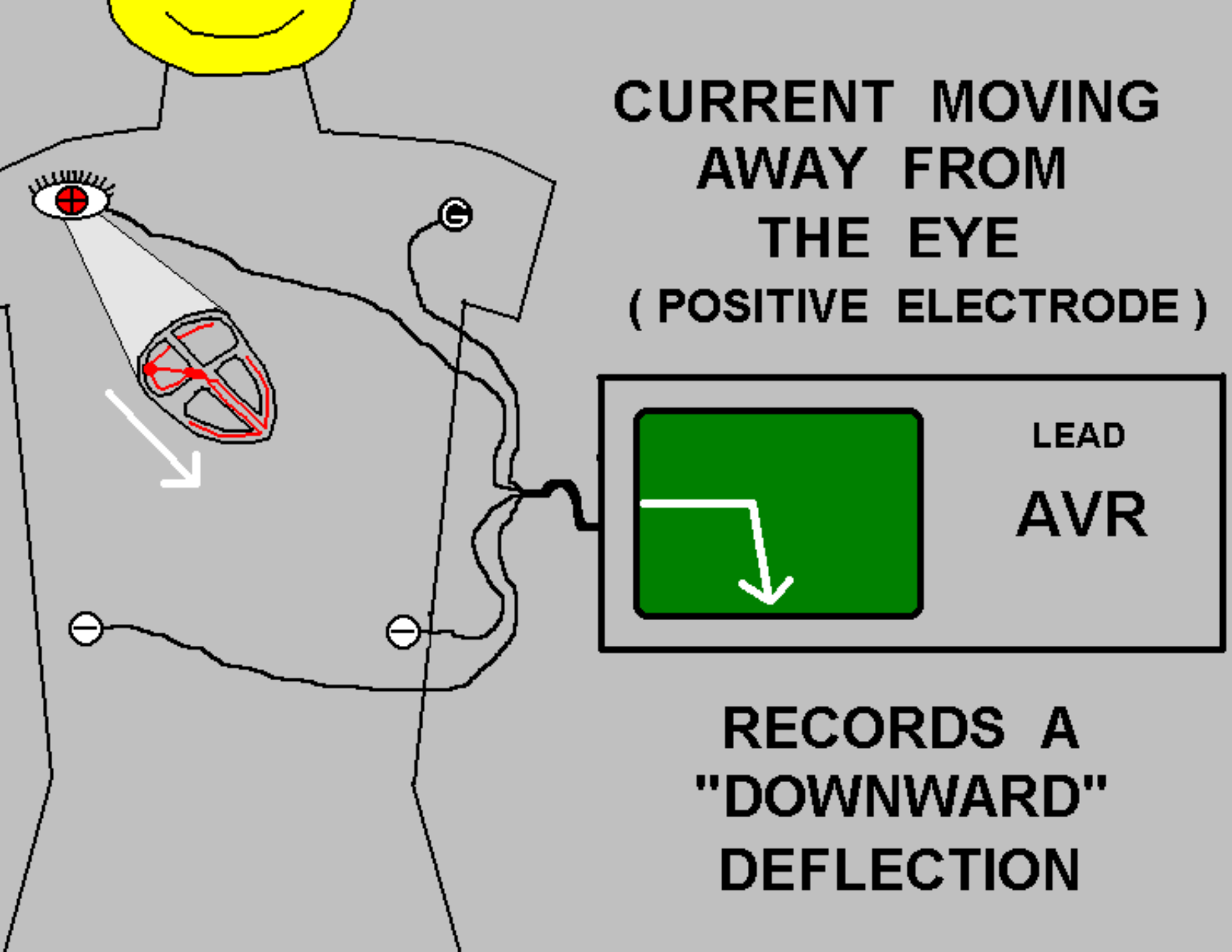


# THE POSITIVE ELECTRODE

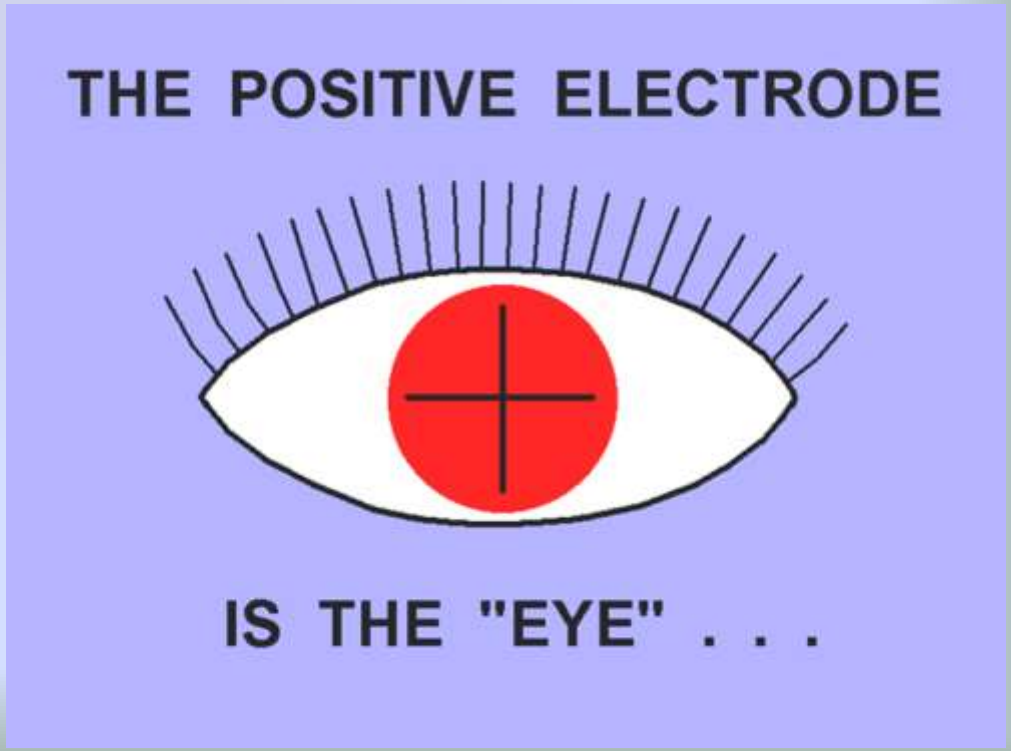


# IS THE "EYE" . . .


CURRENT MOVING
TOWARD THE EYE
(POSITIVE ELECTRODE)
LEAD
II
RECORDS AN
"UPWARD"
DEFLECTION

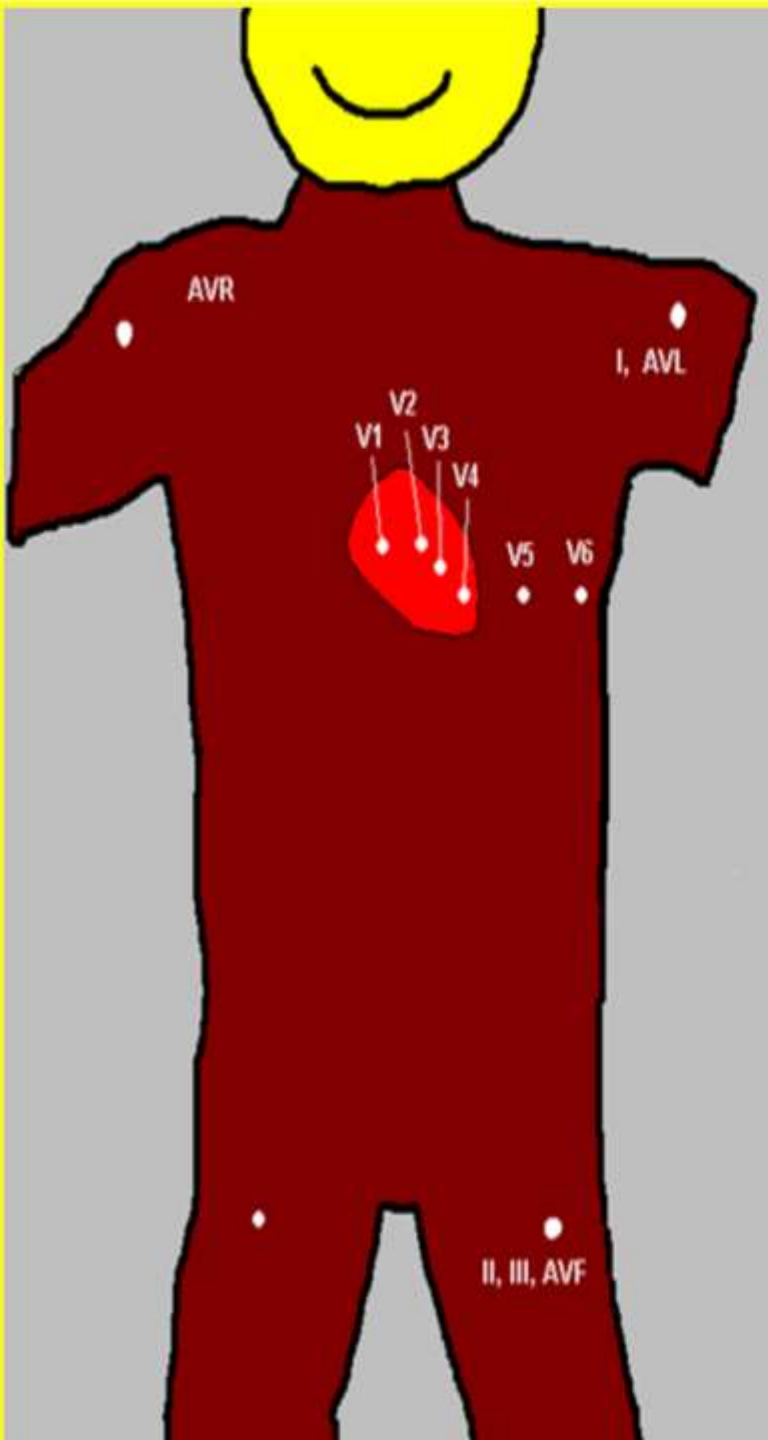

CURRENT MOVING AWAY FROM THE EYE
( POSITIVE ELECTRODE )
LEAD
AVR
RECORDS A "DOWNWARD" DEFLECTION

# *What part of the HEART does each lead SEE ?*



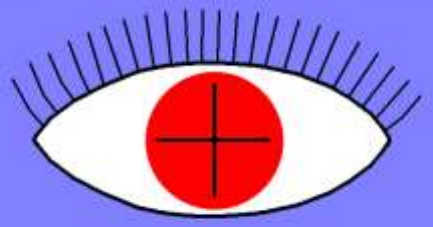
***Imagine a body made of clear glass, with only a HEART inside. We dip this body in liquid chocolate, and then scratch holes in each spot where we normally place the ECG leads . . . . .***

# AREAS VIEWED by 12 LEAD ECG



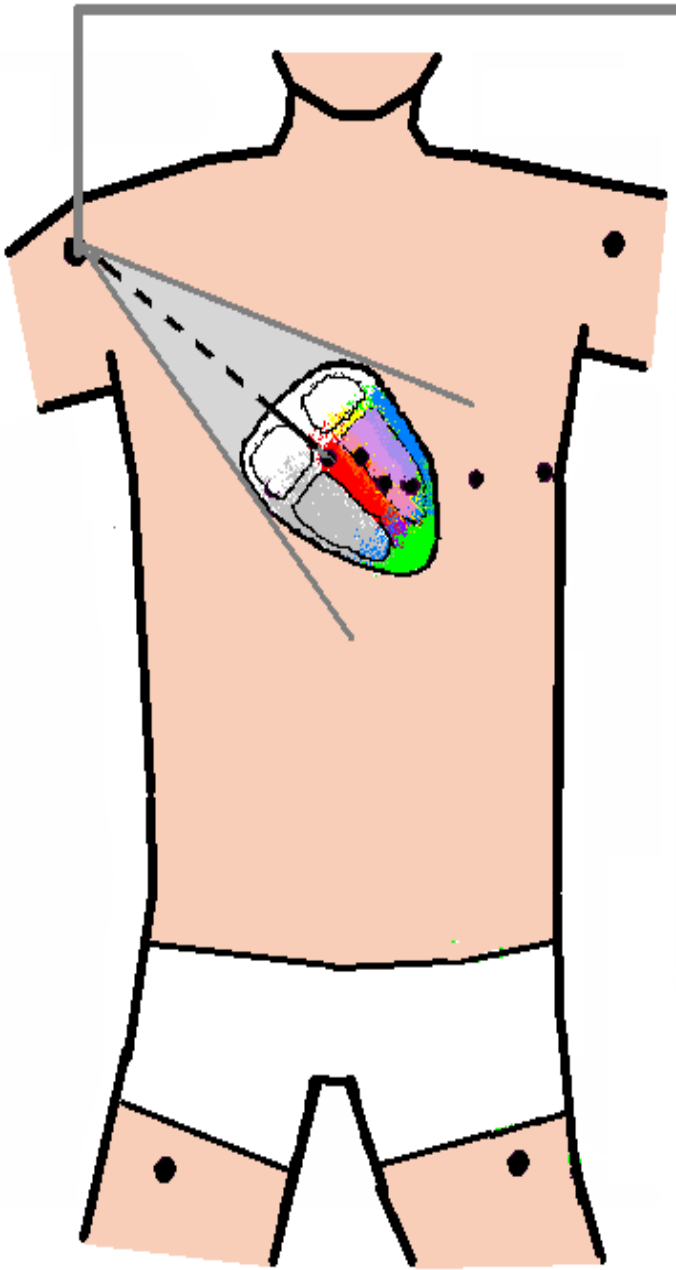

| |
|---|
| AVR |
| AVL, I |
| V1, V2 |
| |
| |
| V3, V4 |
| V5, V6 |
| II, III, AVF |

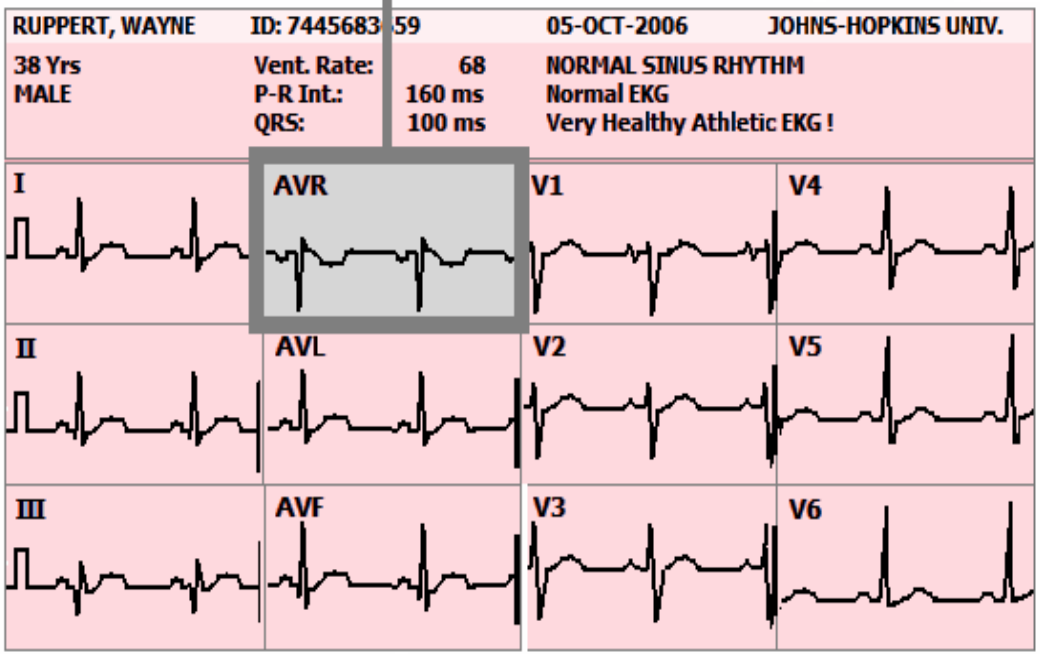
THE POSITIVE ELECTRODE



IS THE "EYE" . . .

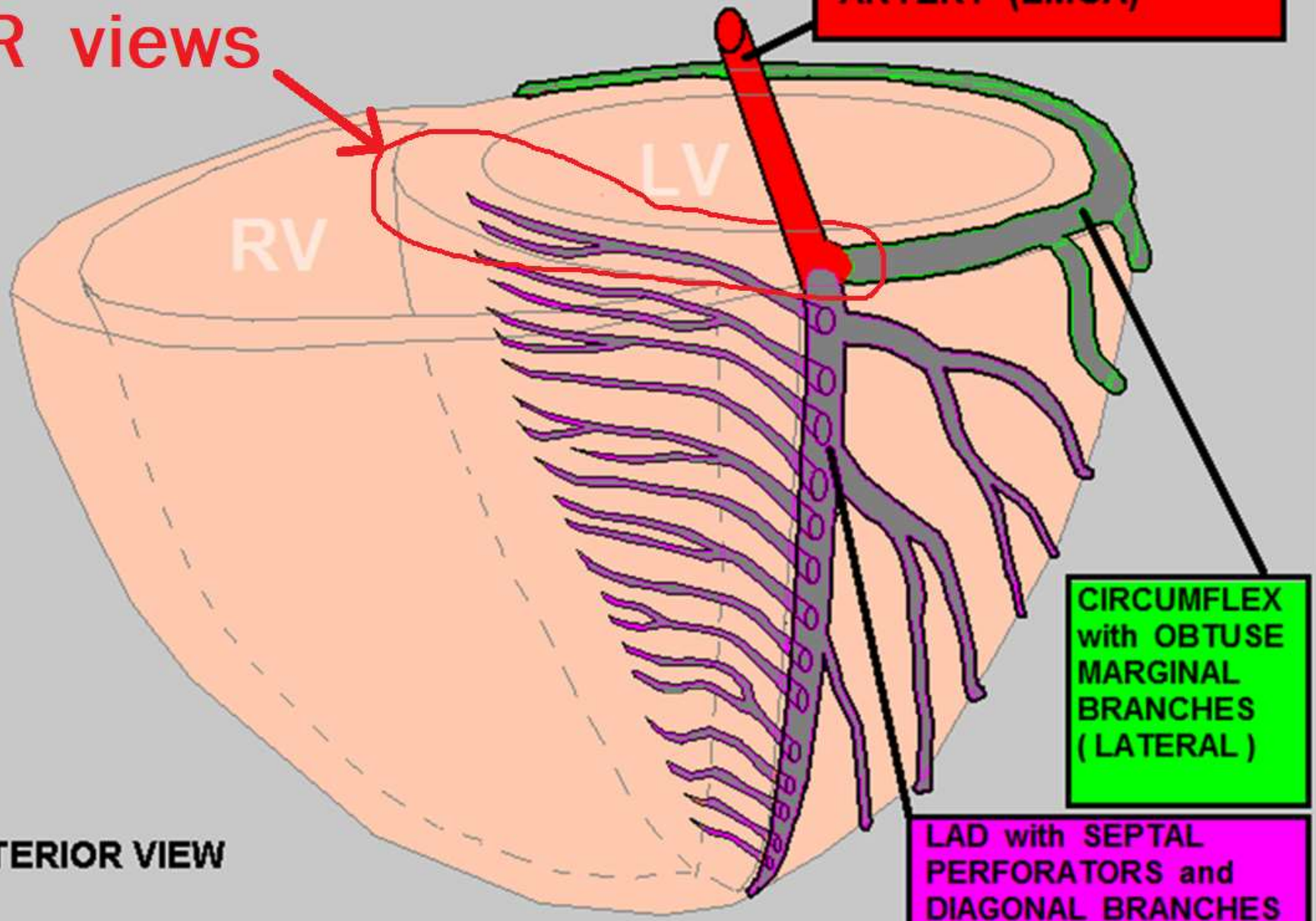
# Lead AVR Views the BASILAR SEPTUM (region of the Bundle of His)



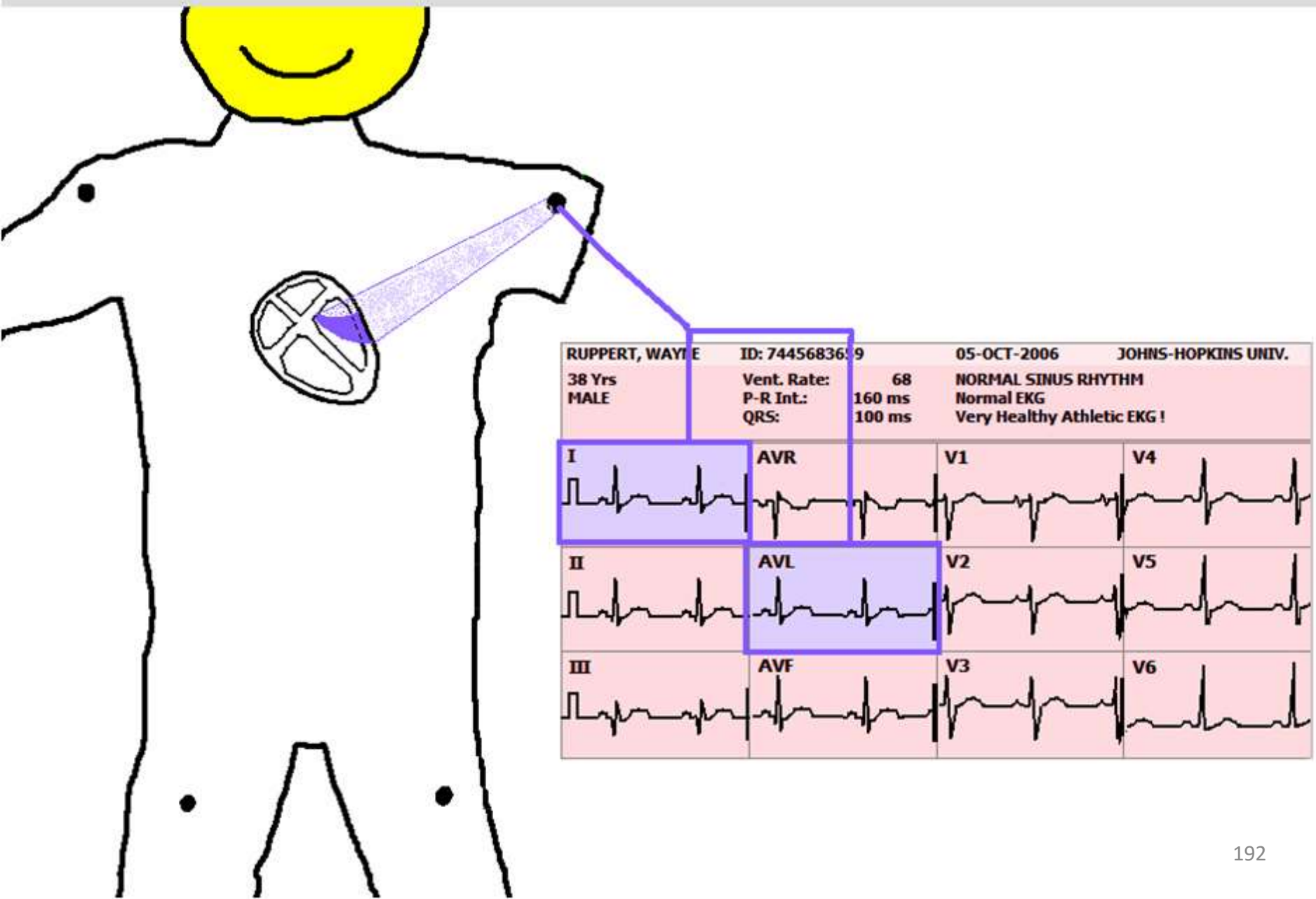


# LEFT CORONARY ARTERY SYSTEM

## AVR views

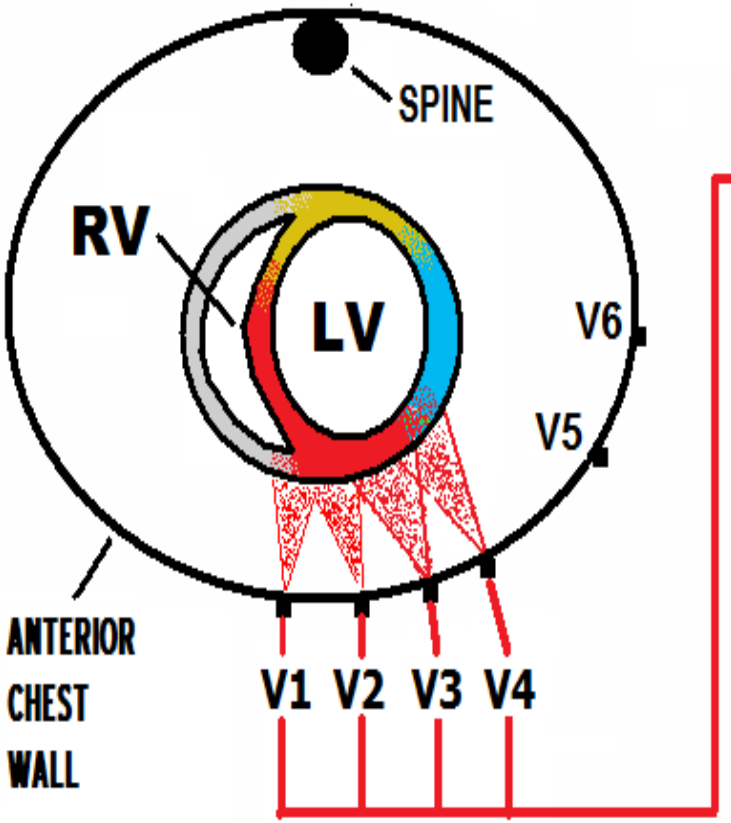


# LEADS I and aVL VIEW the LATERAL - ANTERIOR WALL



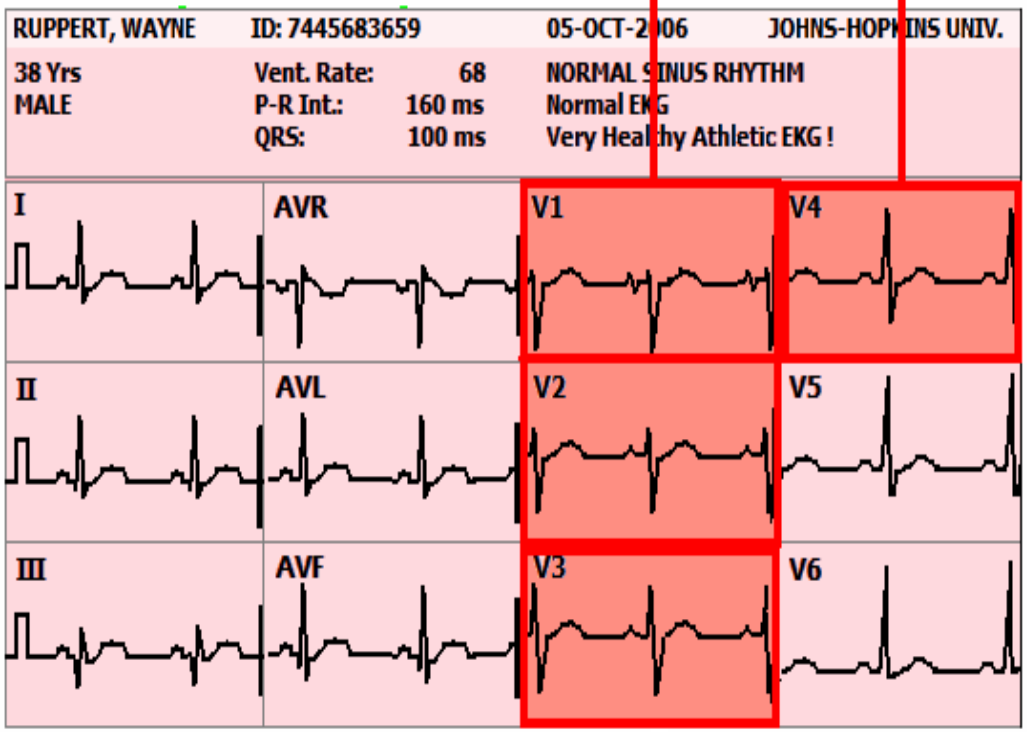
# V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL

## of the LEFT VENTRICLE



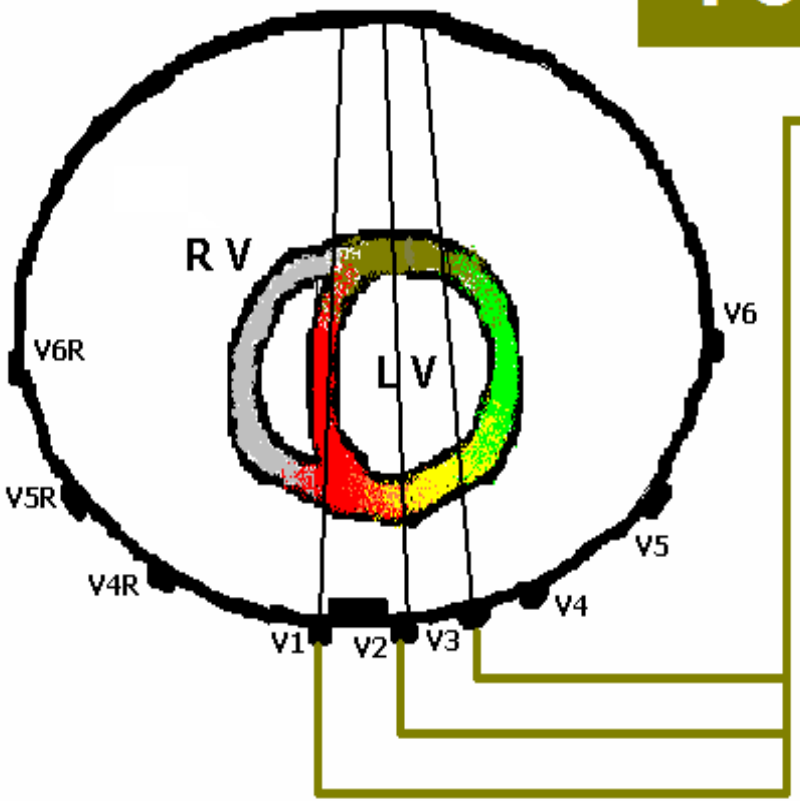

V1, V2 - ANTERIOR / SEPTAL

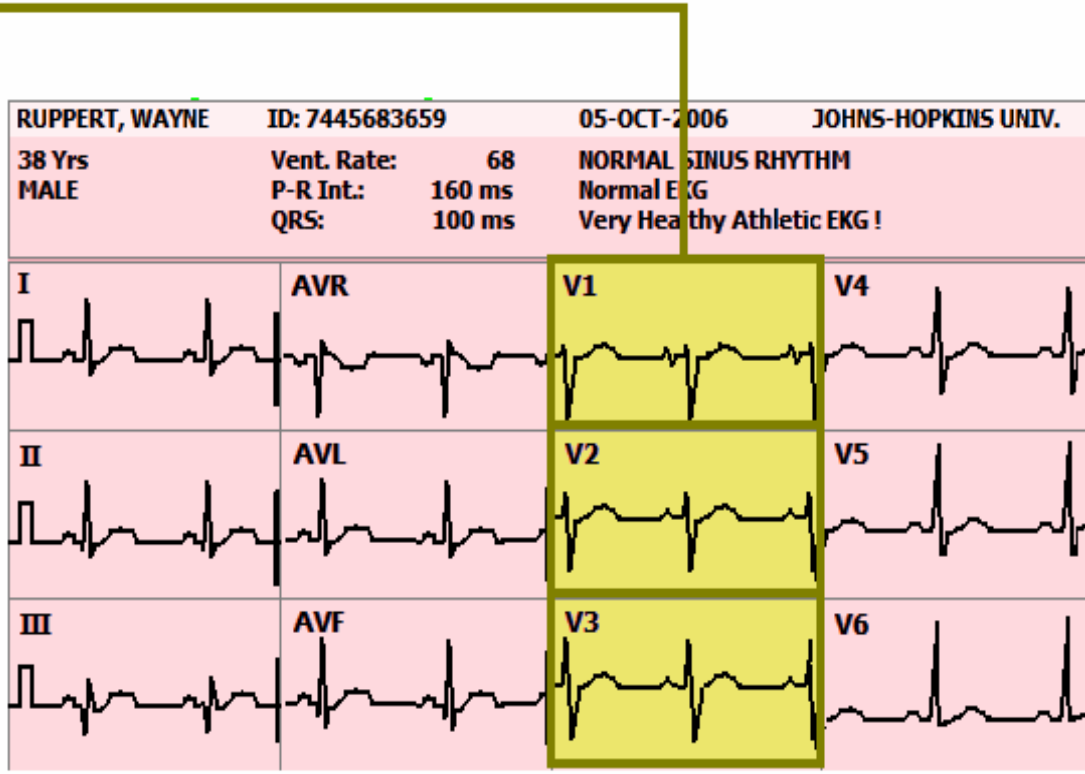
V3, V4 - ANTERIOR



# LEADS V1 - V3 *view the*

## POSTERIOR WALL



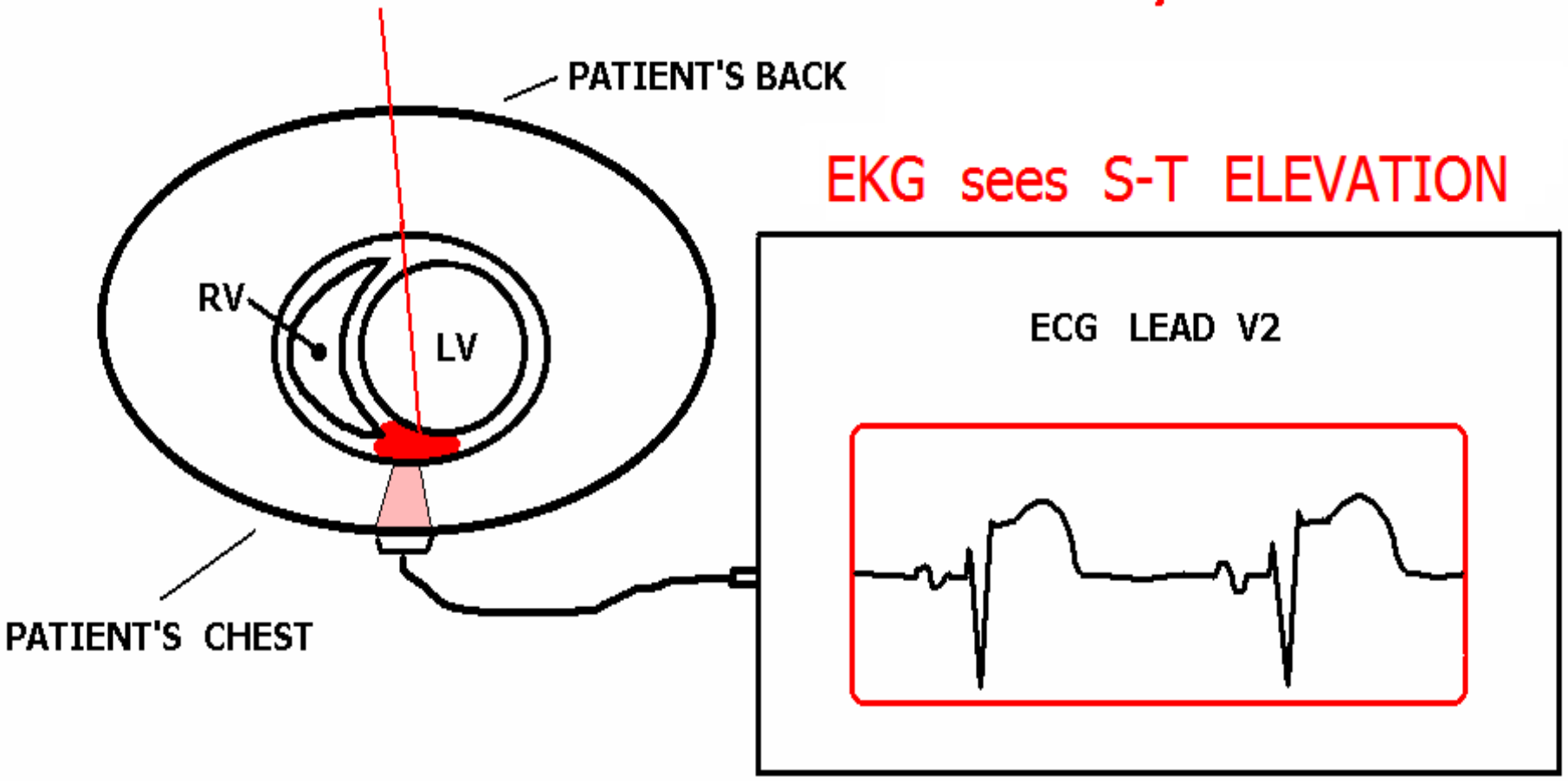




## *via* RECIPROCAL CHANGES.

# HOW EKG VIEWS INDICATIVE CHANGES

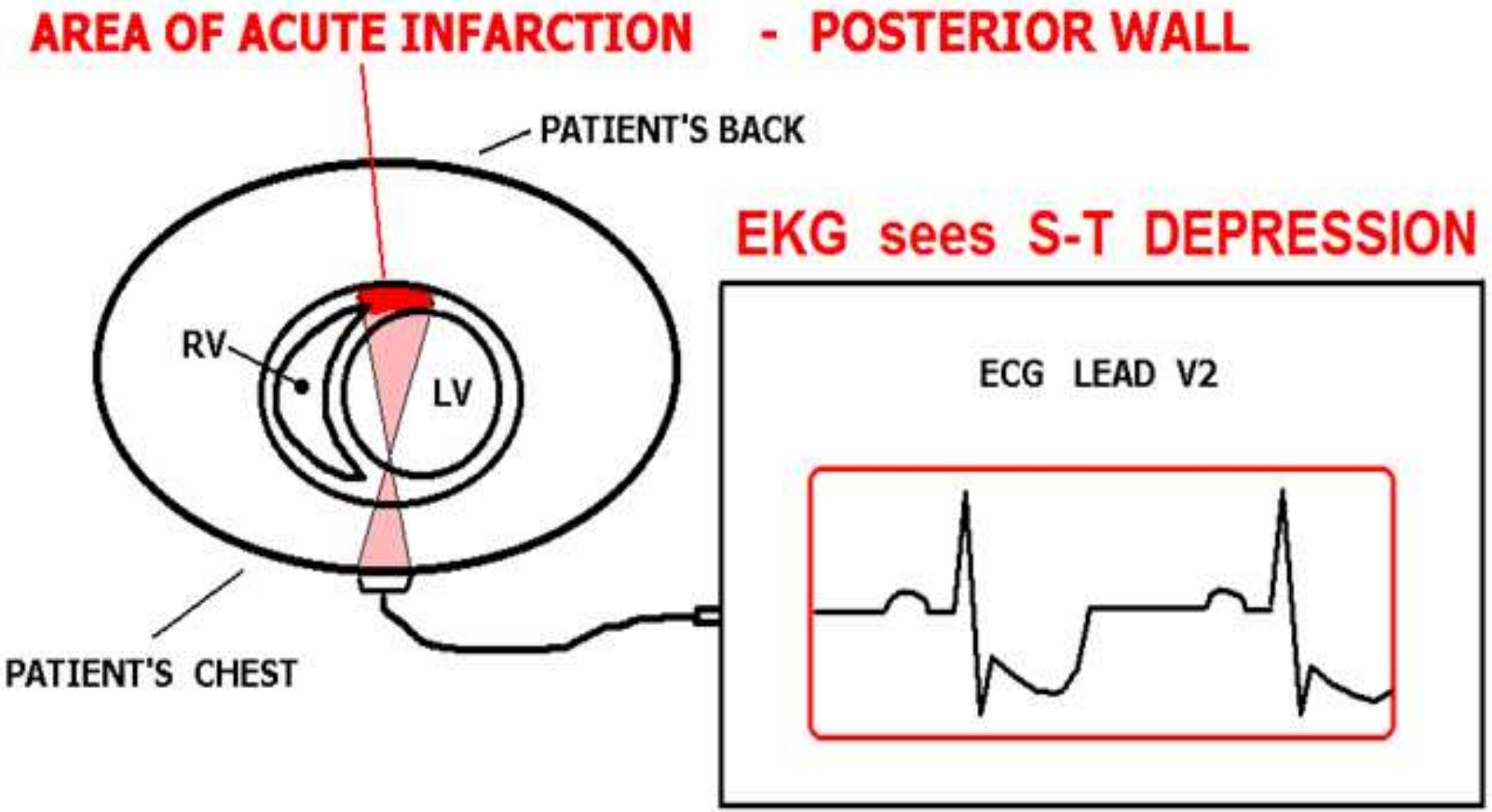
**EXAMPLE:**

**AREA OF ACUTE INFARCTION - ANTERIOR/SEPTAL**

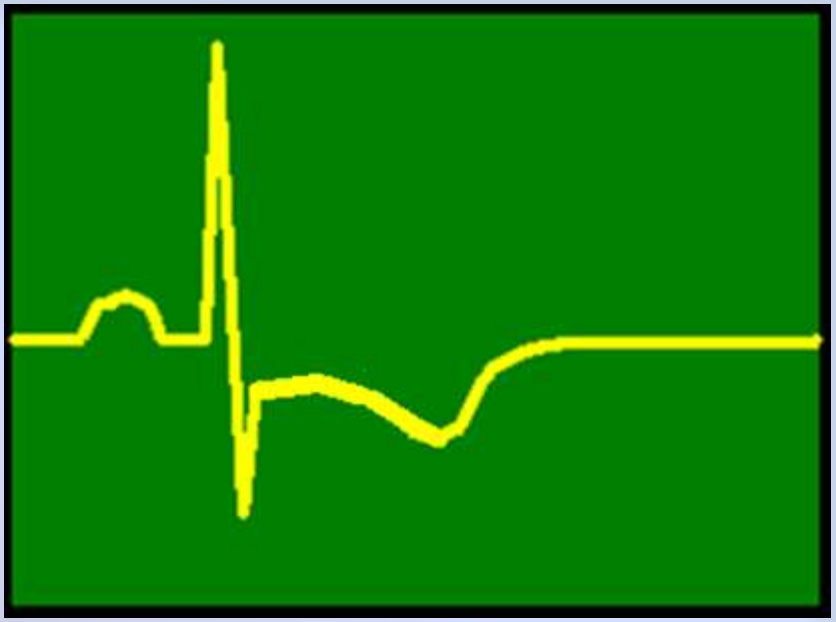


# HOW EKG VIEWS RECIPROCAL CHANGES

**EXAMPLE:**



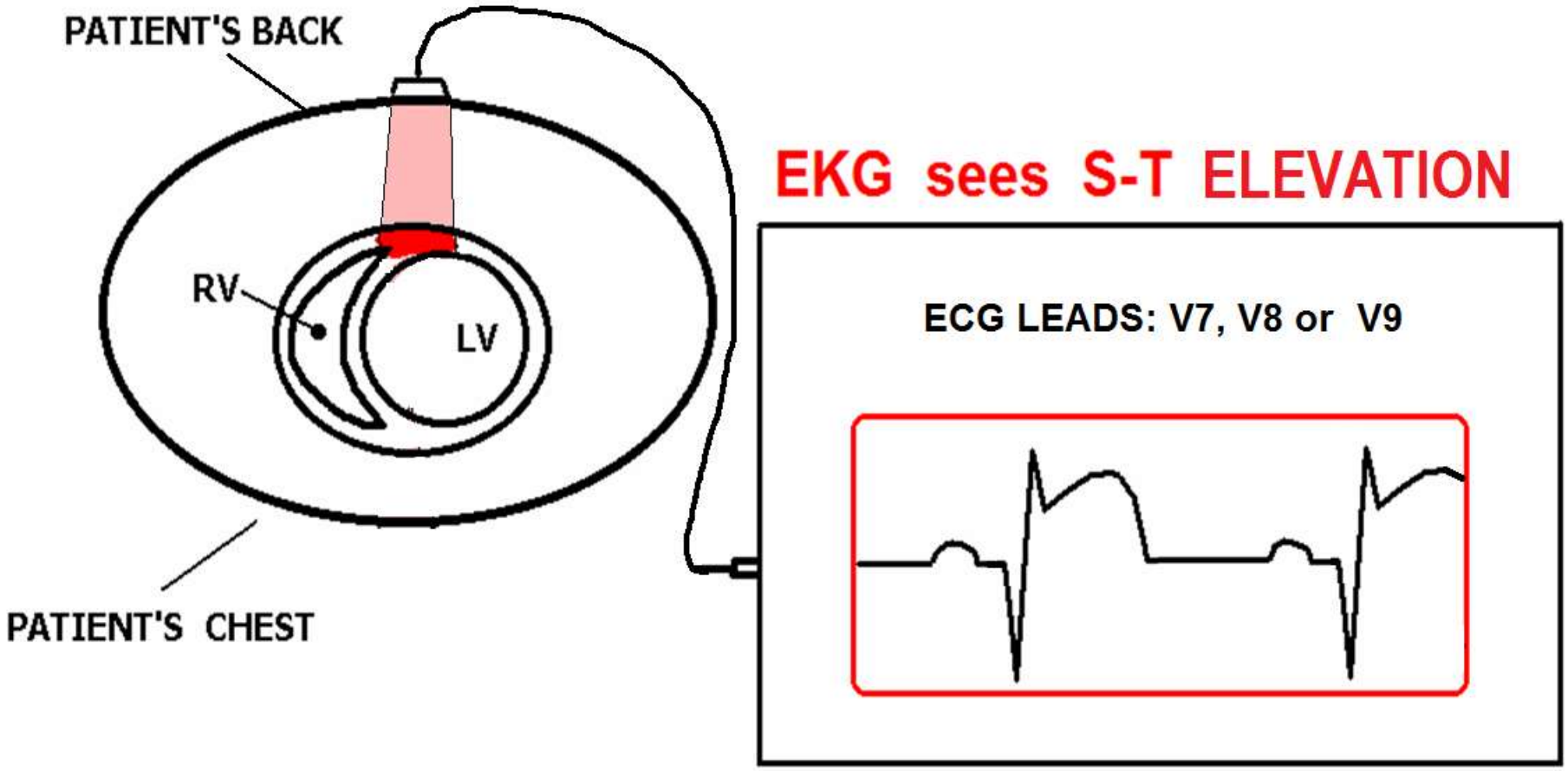
# ST Depression can indicate:



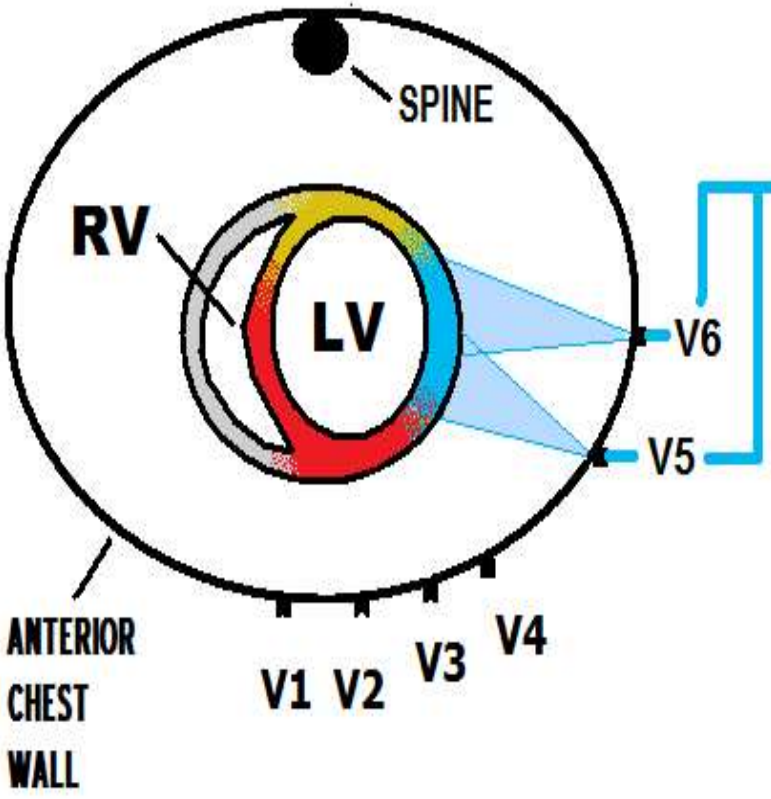
- ISCHEMIA
- “Partial-wall thickness” MI  (NSTEMI)
- STEMI (in the opposite side of the heart)
- Other things (like RBBB, certain medications, etc).

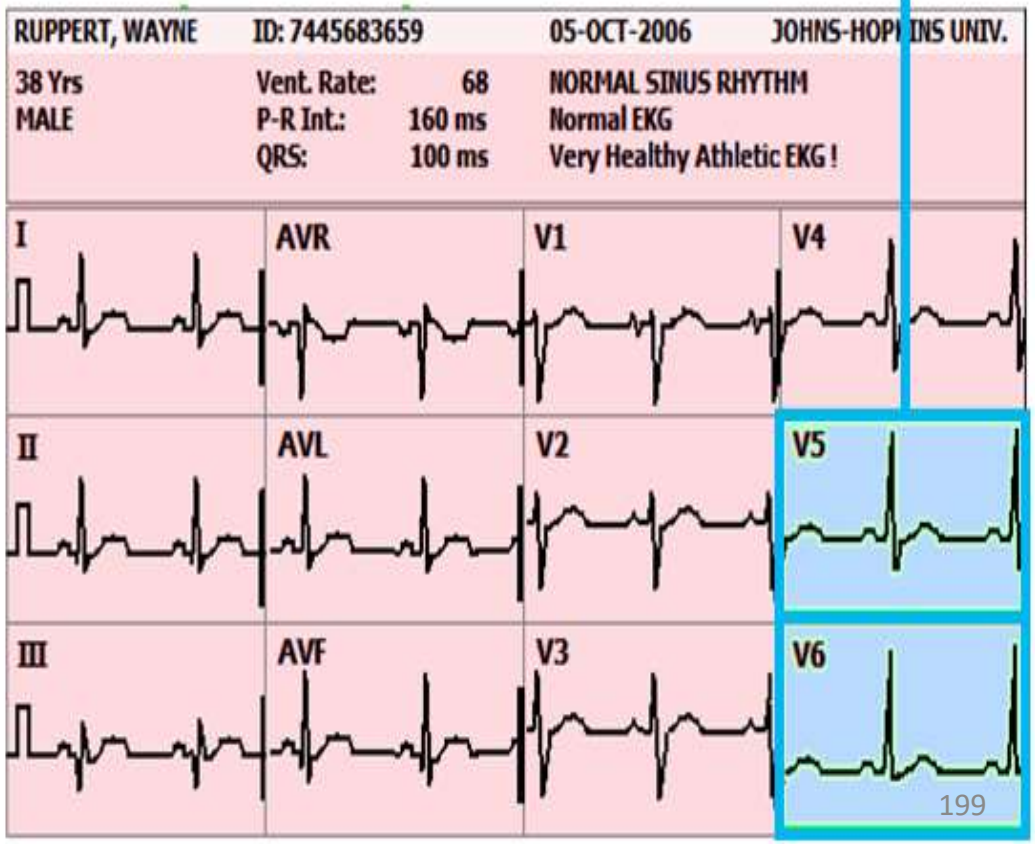
# If we put ECG leads on the BACK of a PATIENT who is having an

## ACUTE POSTERIOR WALL MI . . . . .



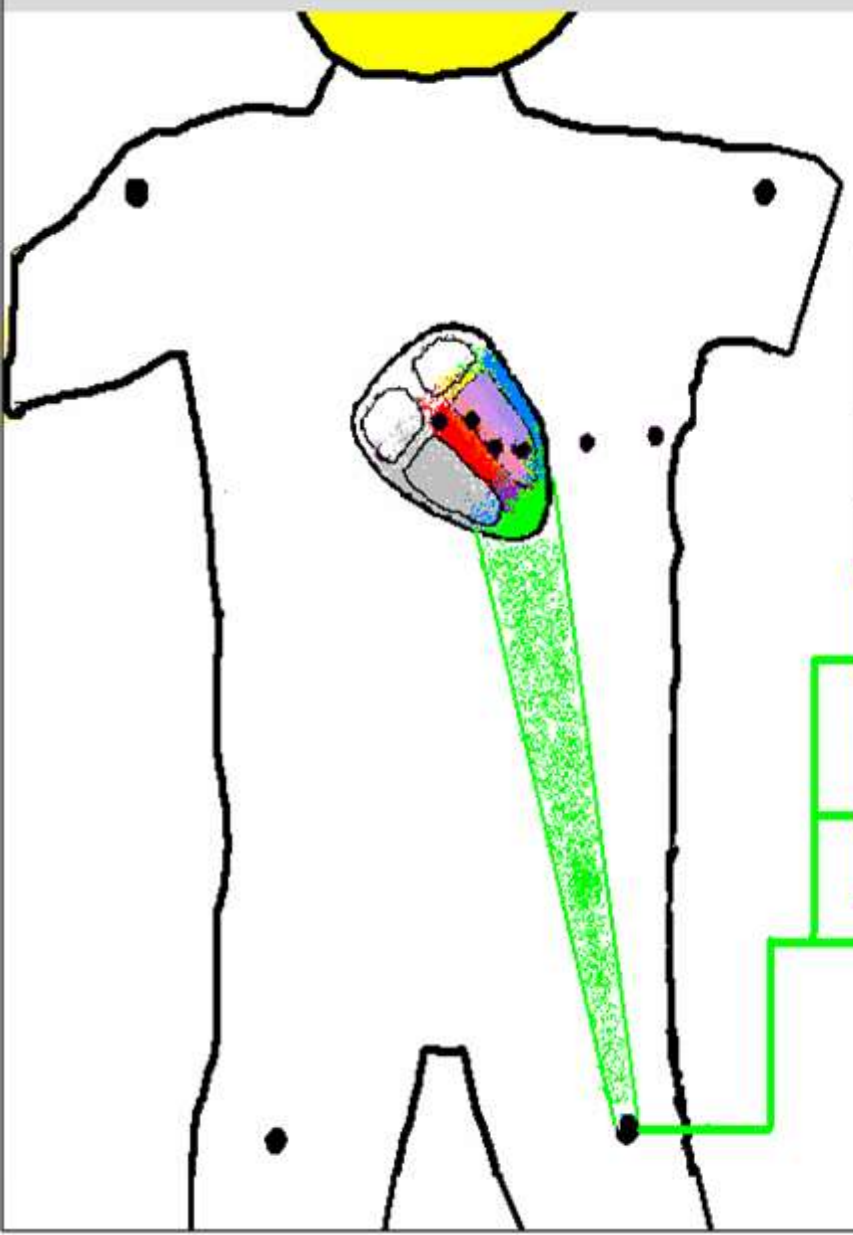
# V5 - V6 VIEW THE LATERAL WALL
## of the LEFT VENTRICLE

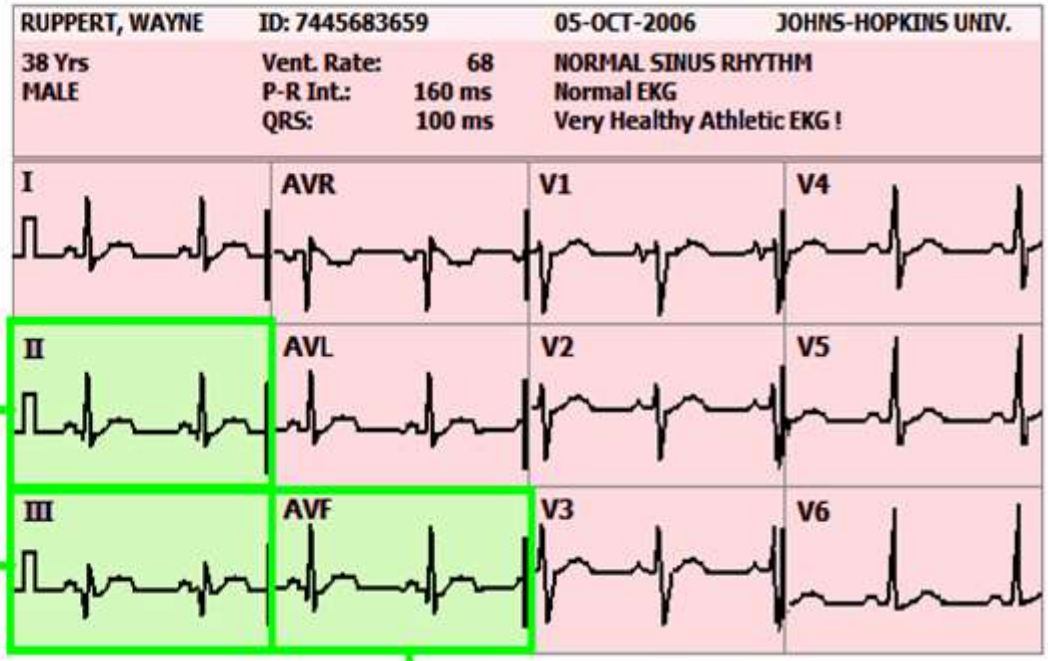


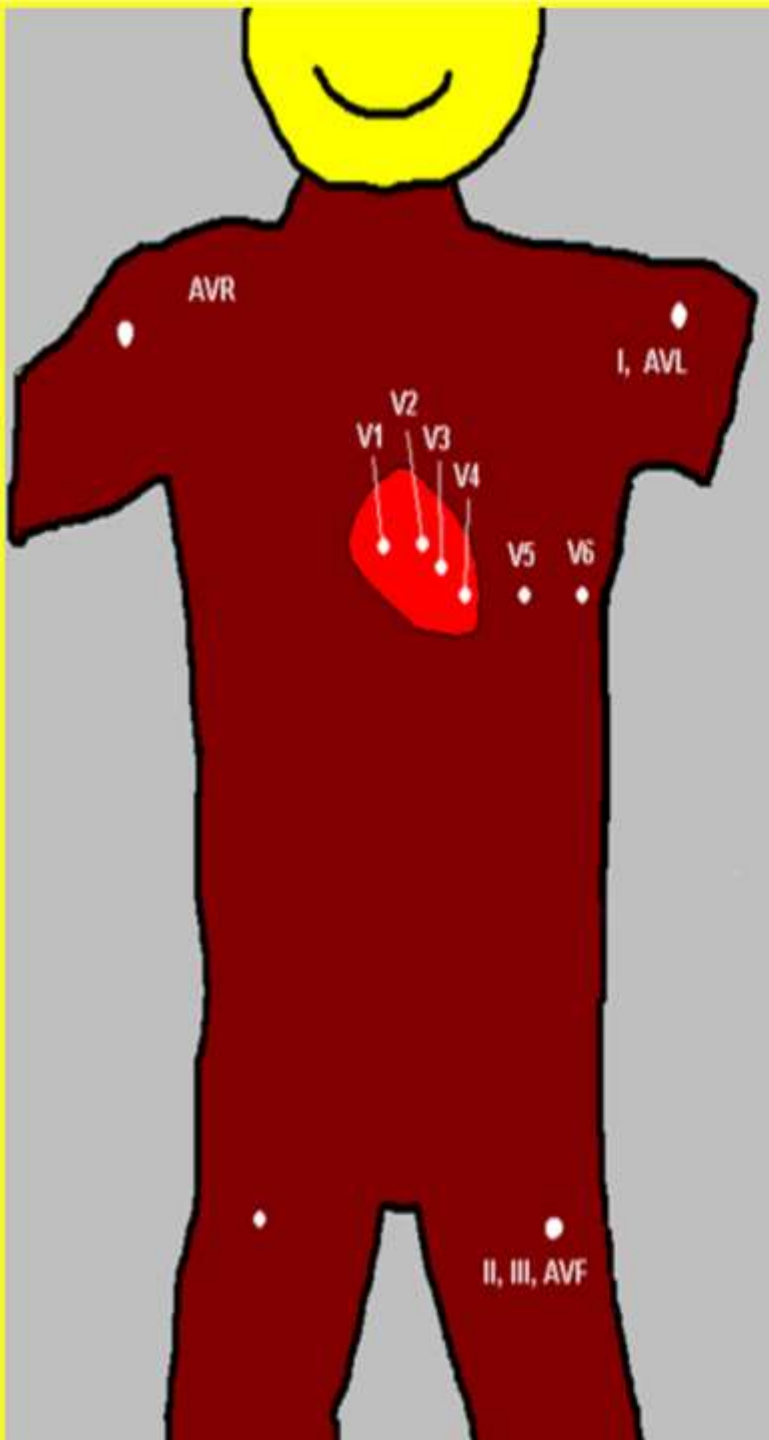



# LEADS II, III, and aVF VIEW

## INFERIOR WALL of the LEFT VENTRICLE







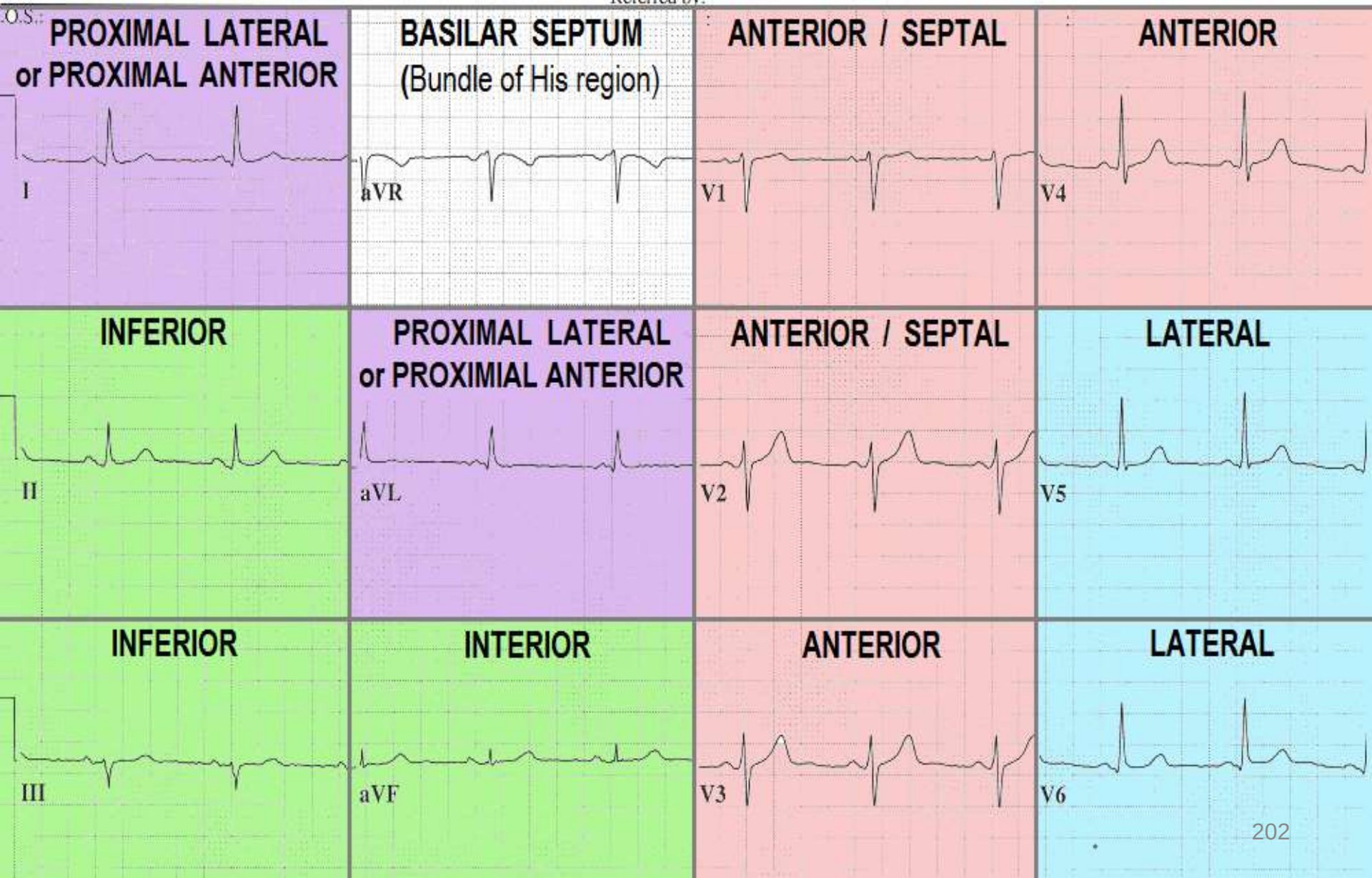

# AREAS VIEWED by 12 LEAD ECG

| | |
|---|---|
| AVR | *BASILAR SEPTAL* |
| AVL, I | LATERAL ANTERIOR |
| V1, V2 | ANTERIOR |
| | SEPTAL |
| | POSTERIOR (recip.) |
| V3, V4 | ANTERIOR |
| V5, V6 | LATERAL |
| II, III, AVF | INFERIOR |

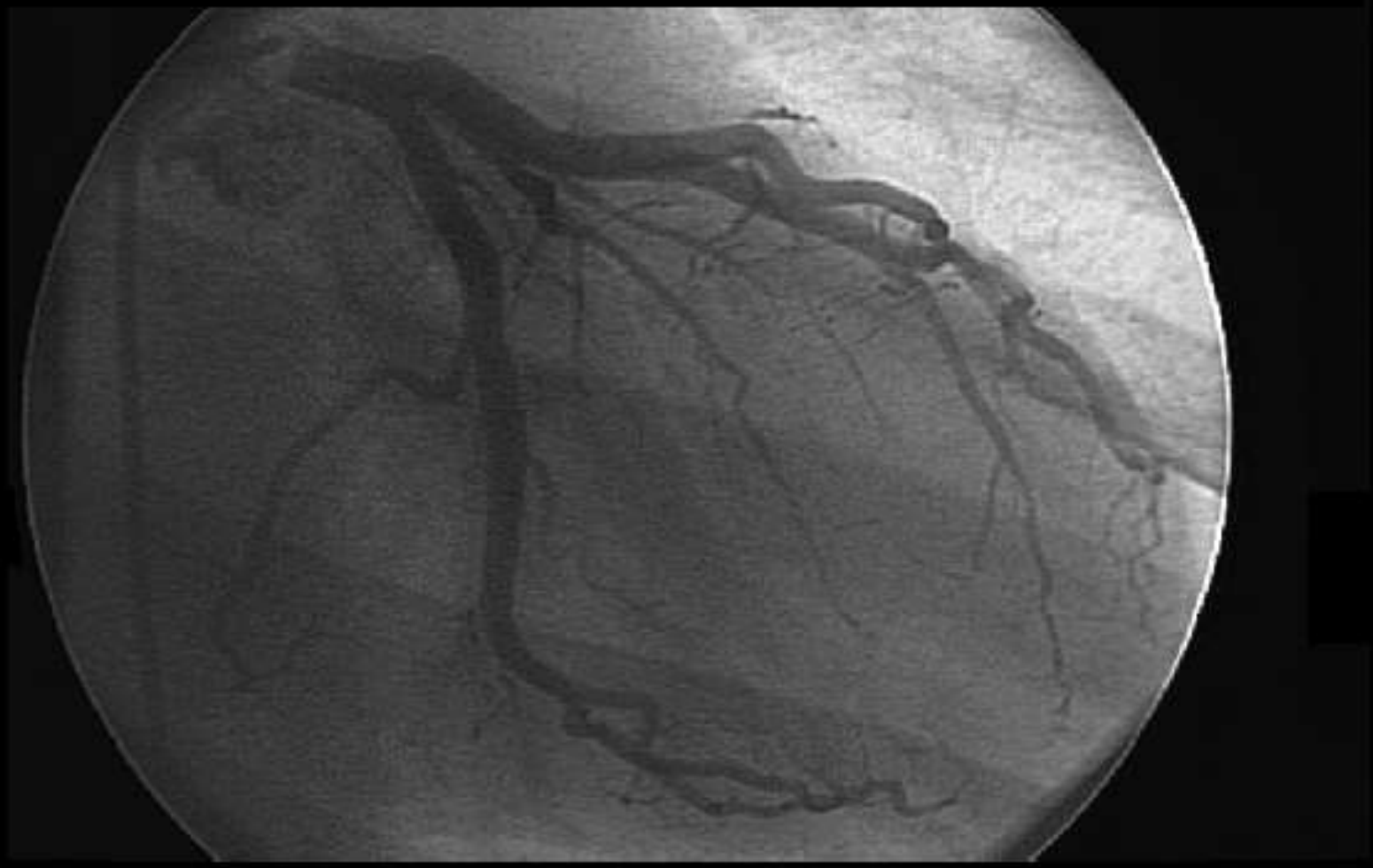
| | | |
|---|---|---|
| Vent. rate | 64 | BPM |
| PR interval | 130 | ms |
| QRS duration | 96 | ms |
| QT/QTc | 396/408 | ms |
| P–R–T axes | 40 11 | 61 |

Normal sinus rhythm
Normal ECG
No previous ECGs available

Referred by:

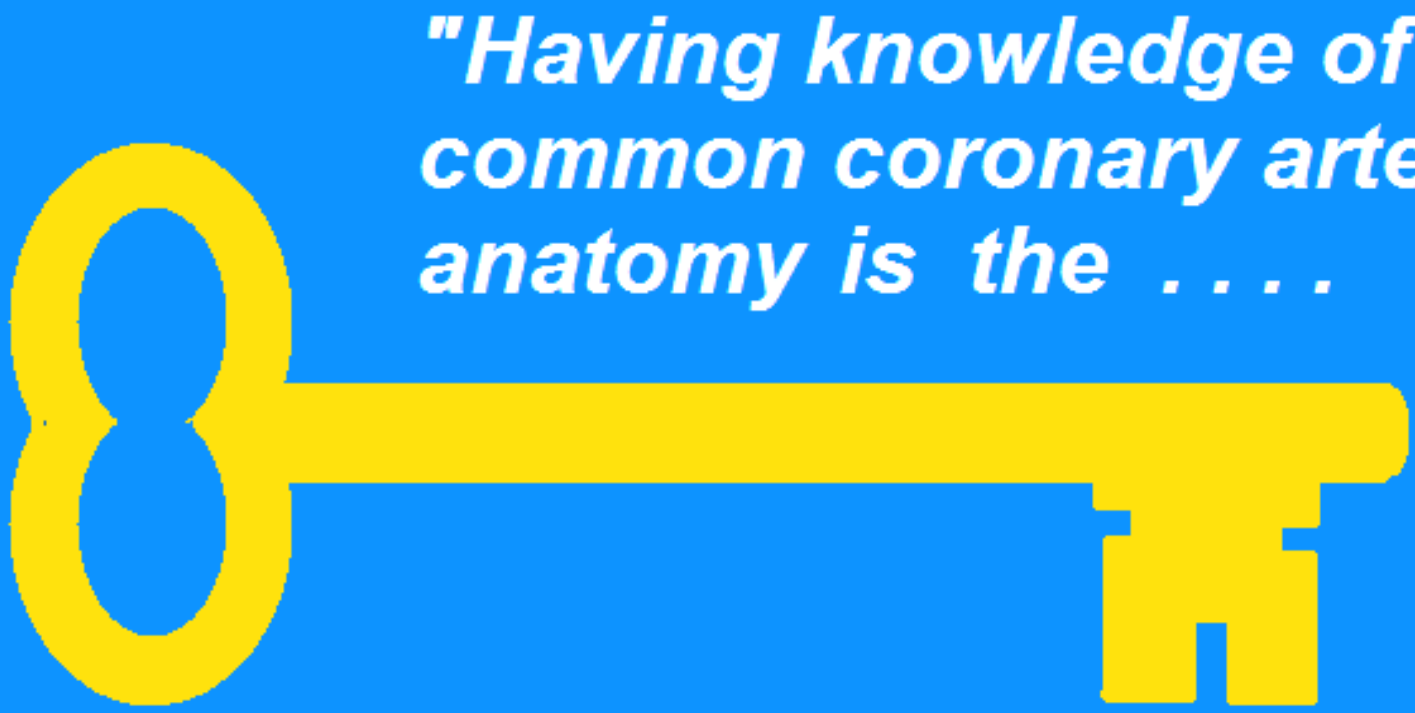


# THE CORONARY



# ARTERIES

# STRUCTURES SERVED BY THE CORONARY ARTERIES




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

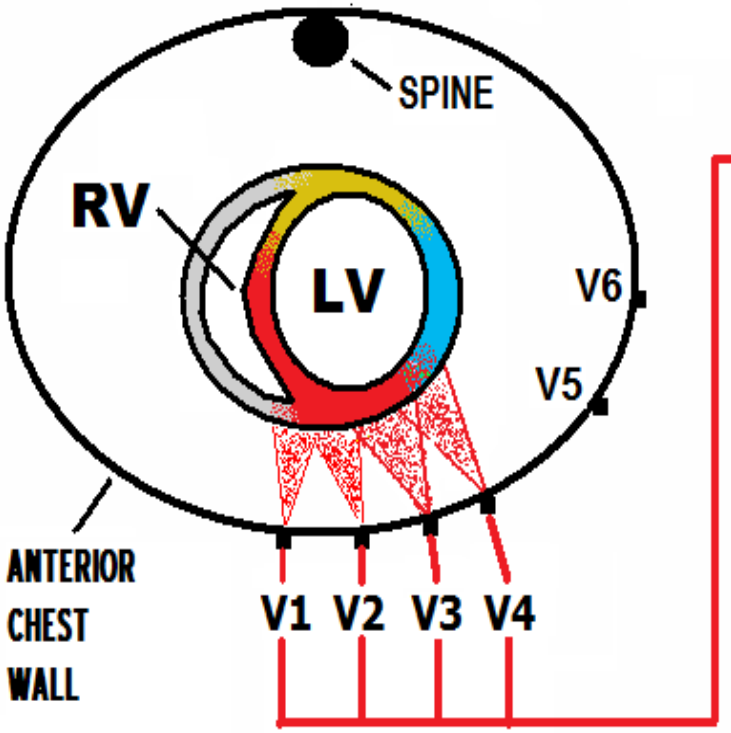
# The 12 Lead ECG becomes your "crystal ball !!"

# 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 !***

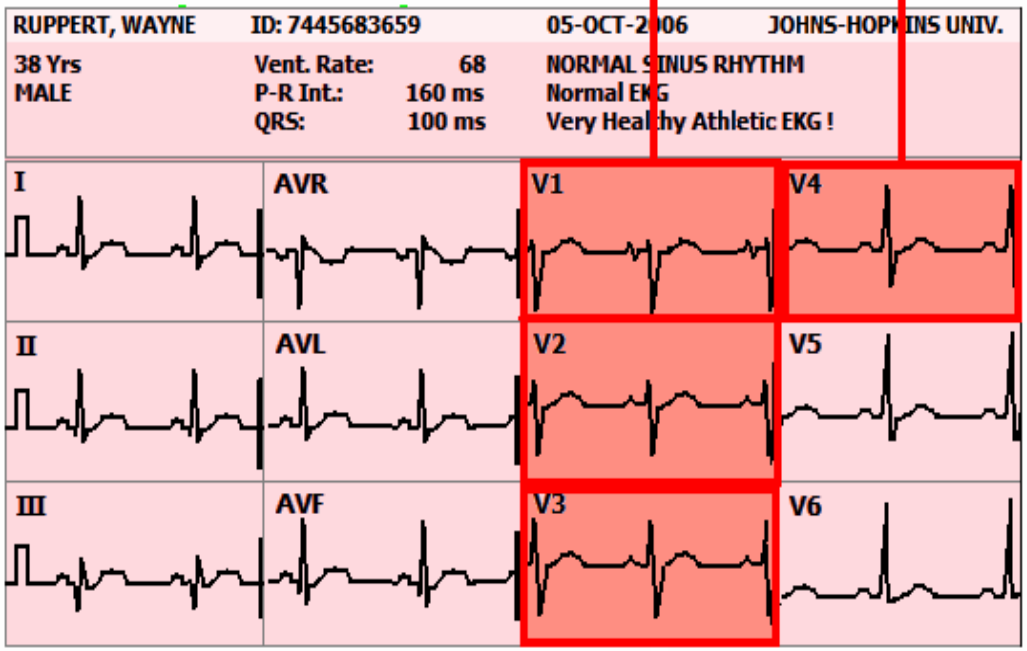
# V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL

## of the LEFT VENTRICLE



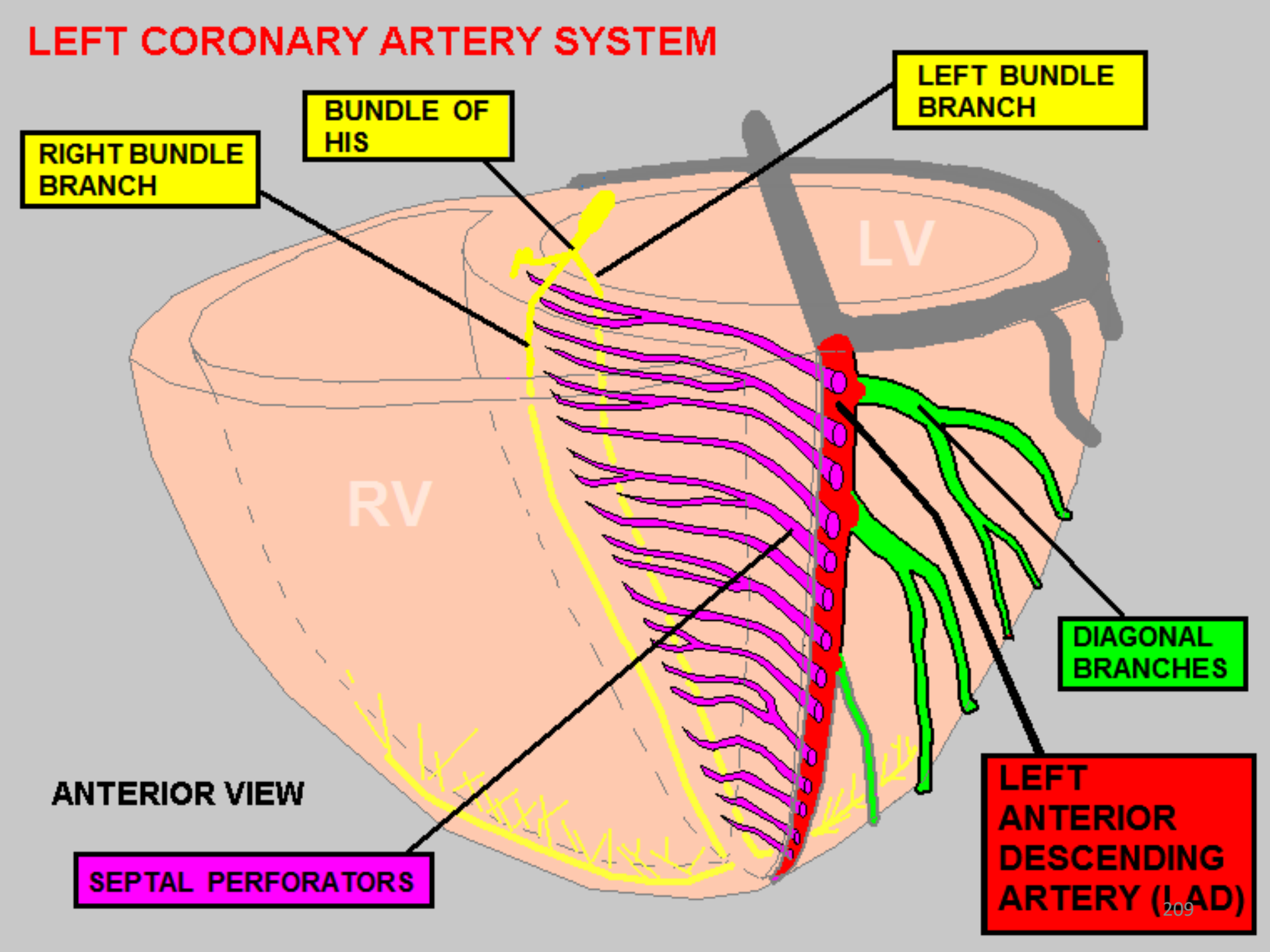

V1, V2 - ANTERIOR / SEPTAL

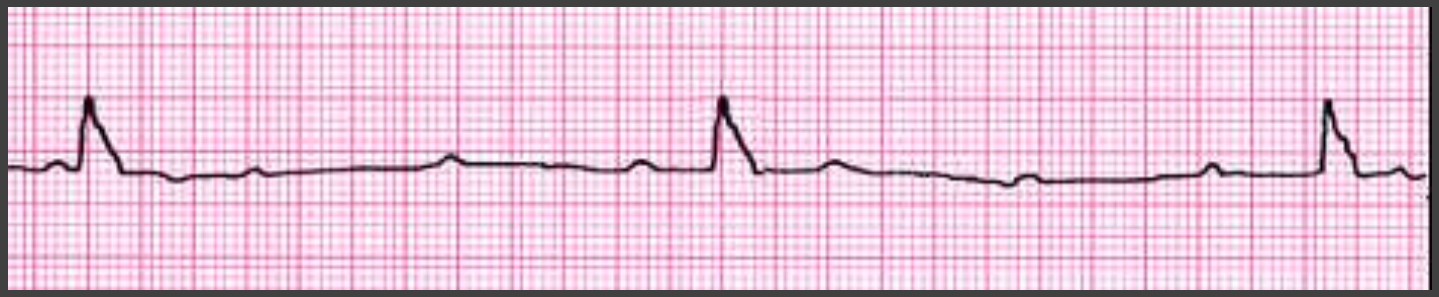
V3, V4 - ANTERIOR

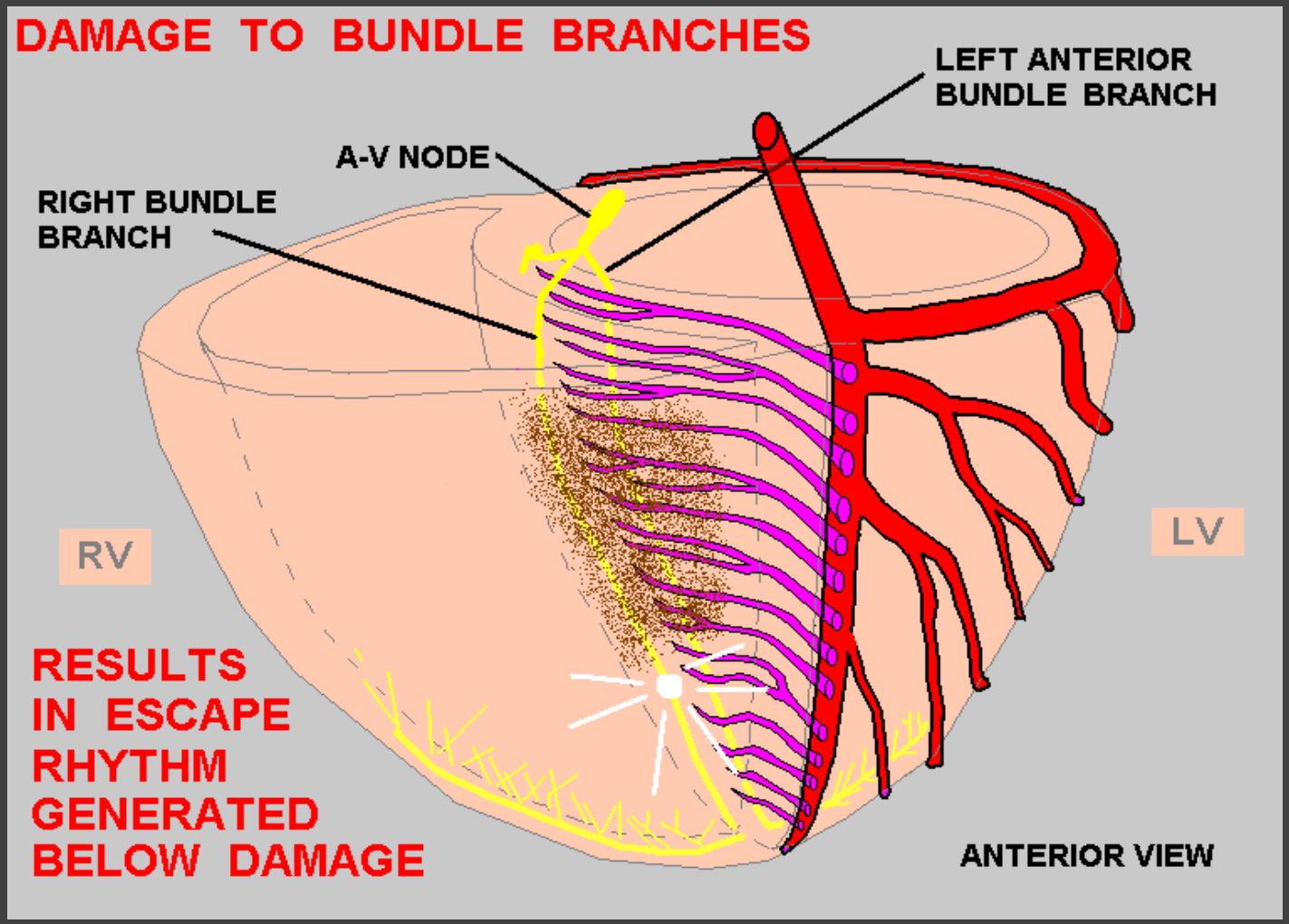



**Which Coronary Artery typically Supplies the ANTERIOR WALL ?**

# LEFT CORONARY ARTERY SYSTEM



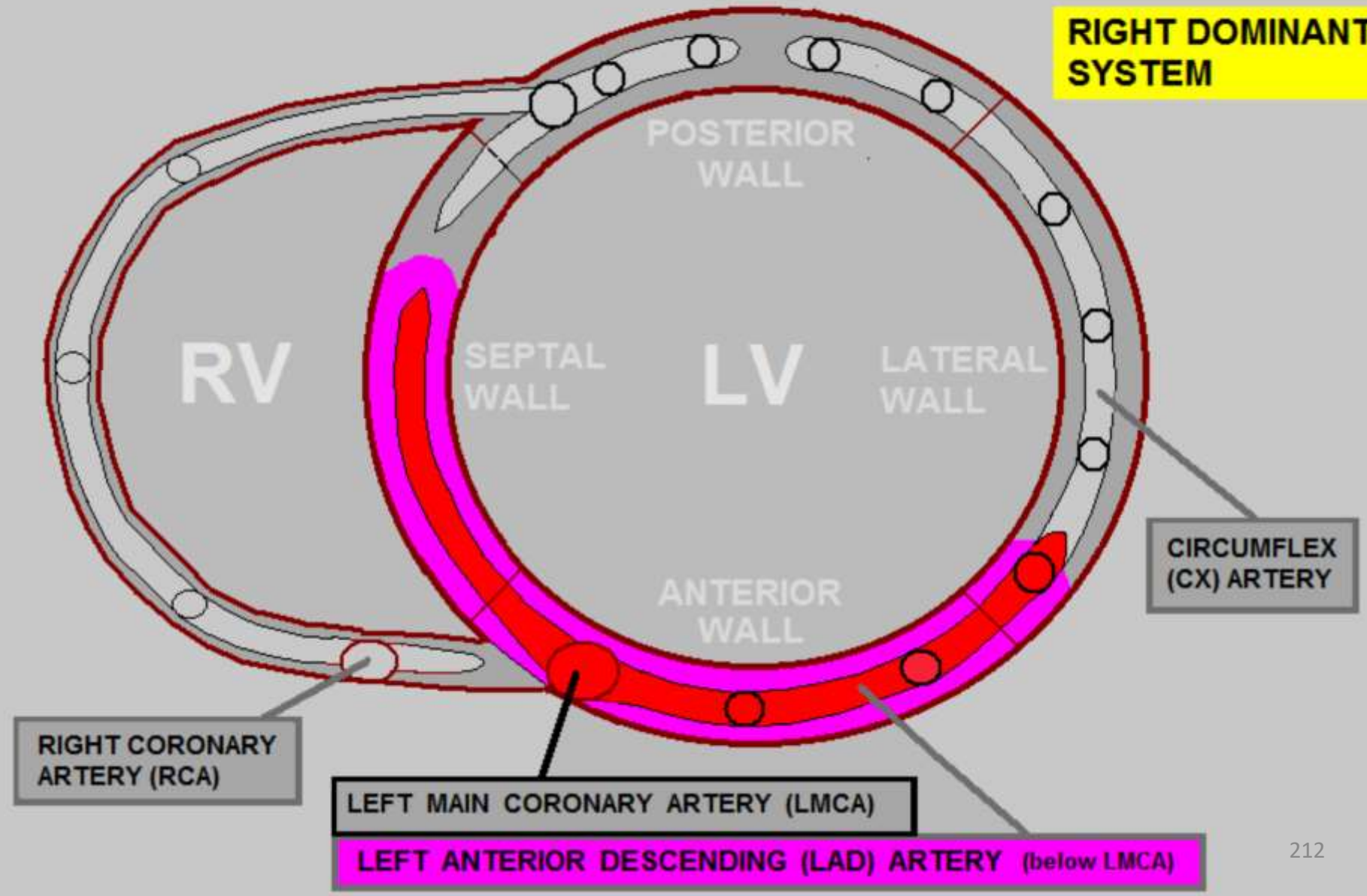



DAMAGE TO BUNDLE BRANCHES
LEFT ANTERIOR BUNDLE BRANCH
A-V NODE
RIGHT BUNDLE BRANCH
RV
LV
RESULTS IN ESCAPE RHYTHM GENERATED BELOW DAMAGE
ANTERIOR VIEW

*cutaway view of the*

# LEFT ANTERIOR DESCENDING ARTERY (LAD)

## SUPPLIES APPROX. 35 - 45% of the LV MUSCLE MASS



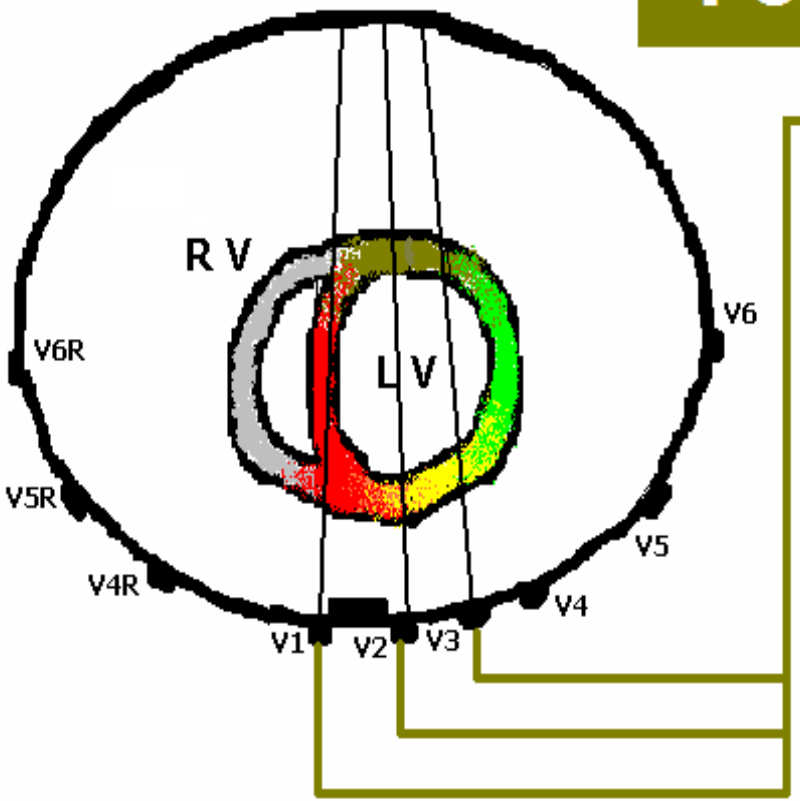
HELPFUL HINT . . . MEMORIZE THIS !

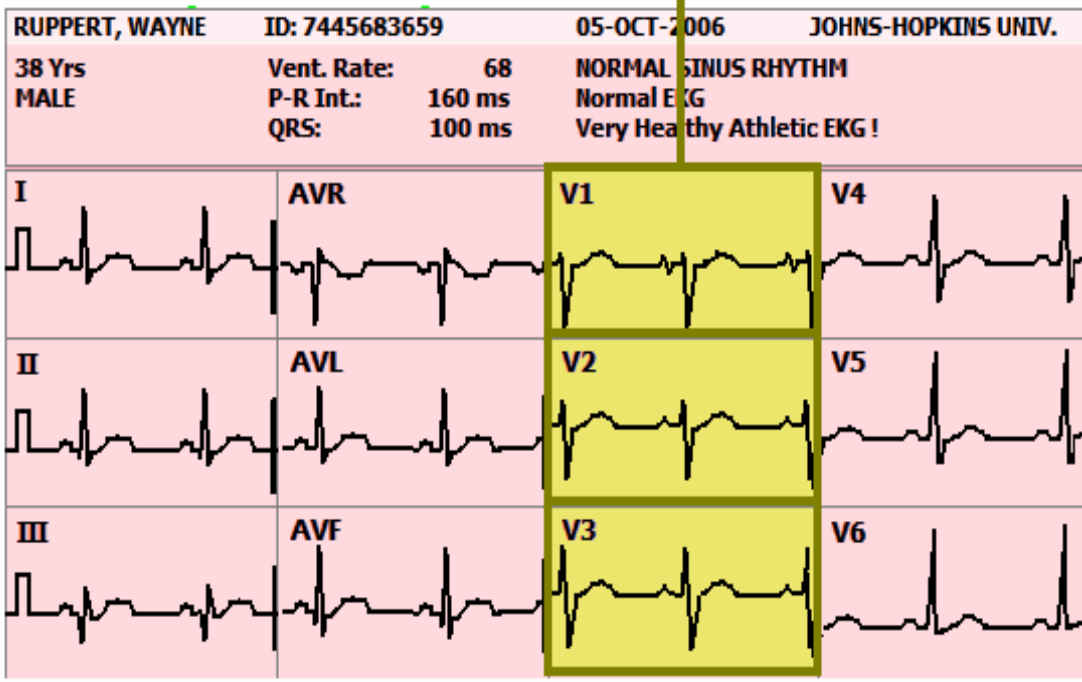
# LEFT ANTERIOR DESCENDING ARTERY (LAD)

- BUNDLE OF HIS
- BUNDLE BRANCHES ( )
- 35 - 45 % OF LV MUSCLE MASS
  - ANTERIOR WALL
  - SEPTAL WALL ( anterior 2/3 )

# LEADS V1 - V3 *view the*

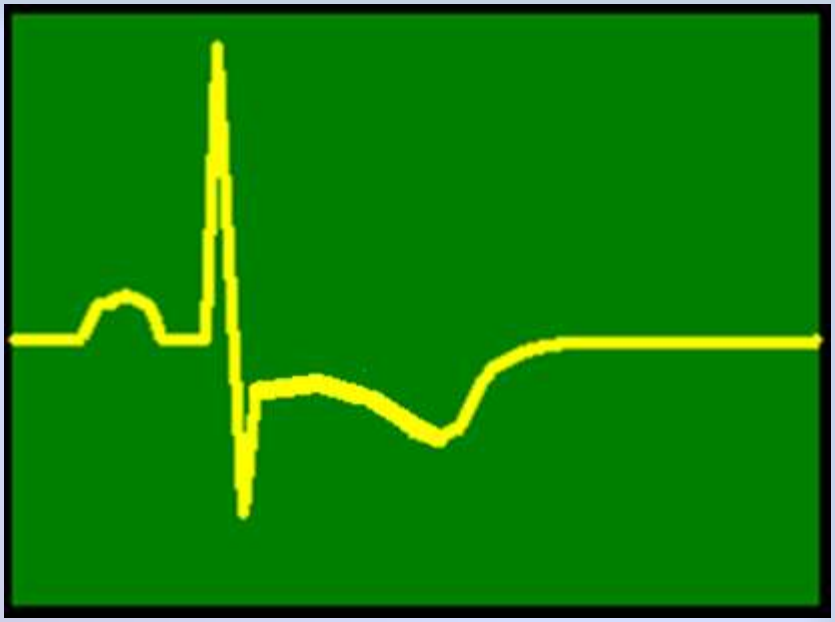
## POSTERIOR WALL



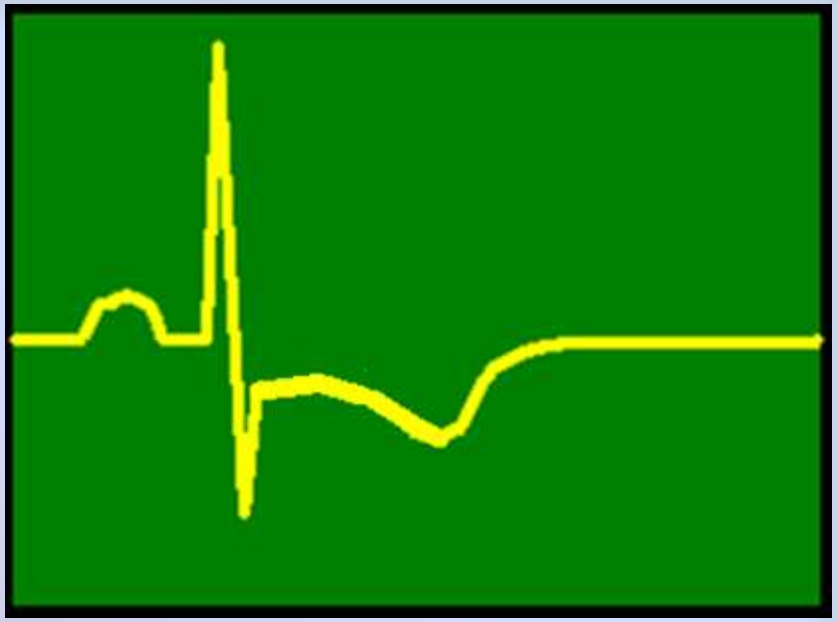

## *via* RECIPROCAL CHANGES.

# ST Depression in Leads V1 – V4:



- **Direct view of ISCHEMIA  (anterior wall)**

# ST Depression in Leads V1 – V4:

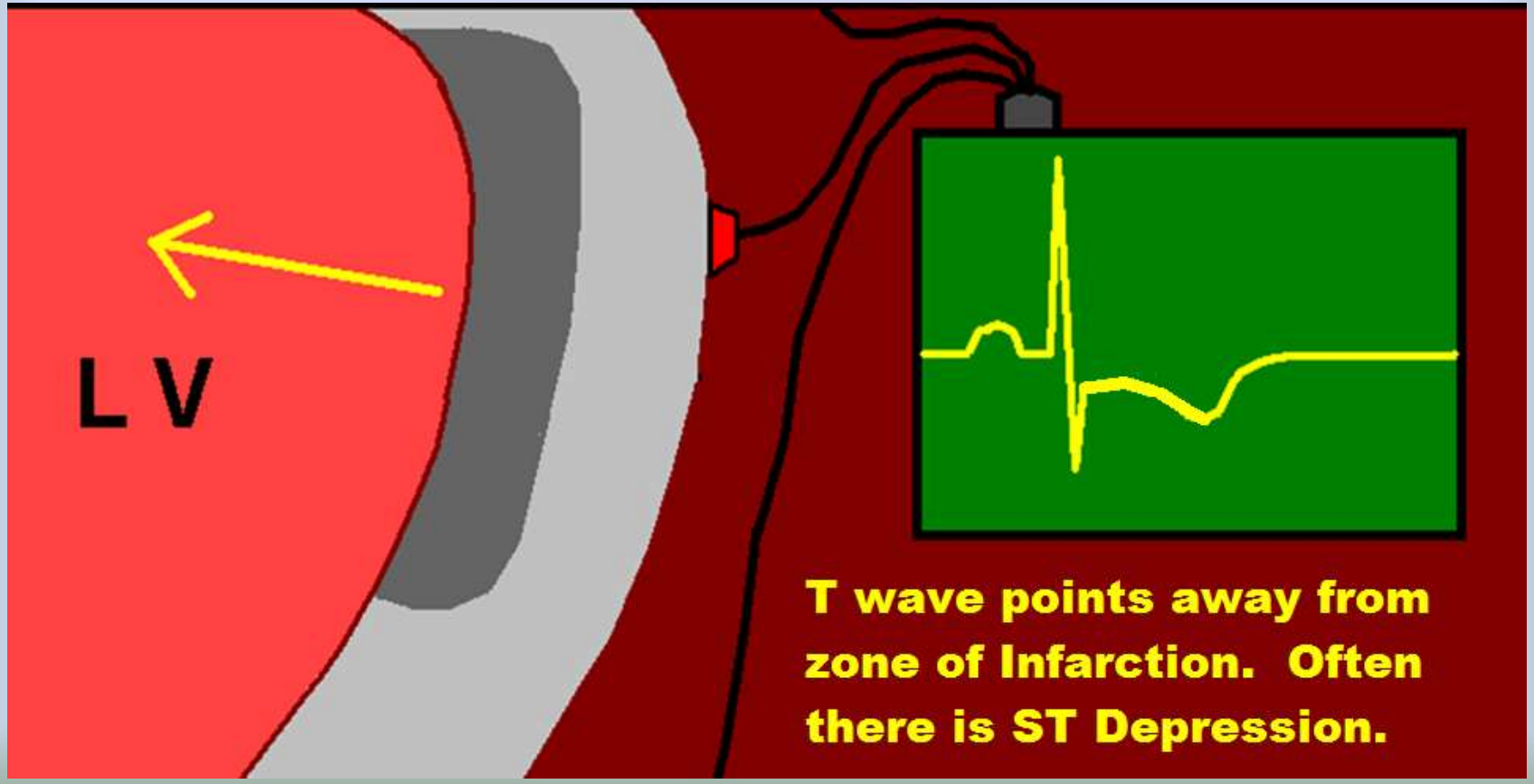


- Direct view of ISCHEMIA (anterior wall)
- Direct view of NSTEMI (anterior wall)

# Non-STEMI (NSTEMI)

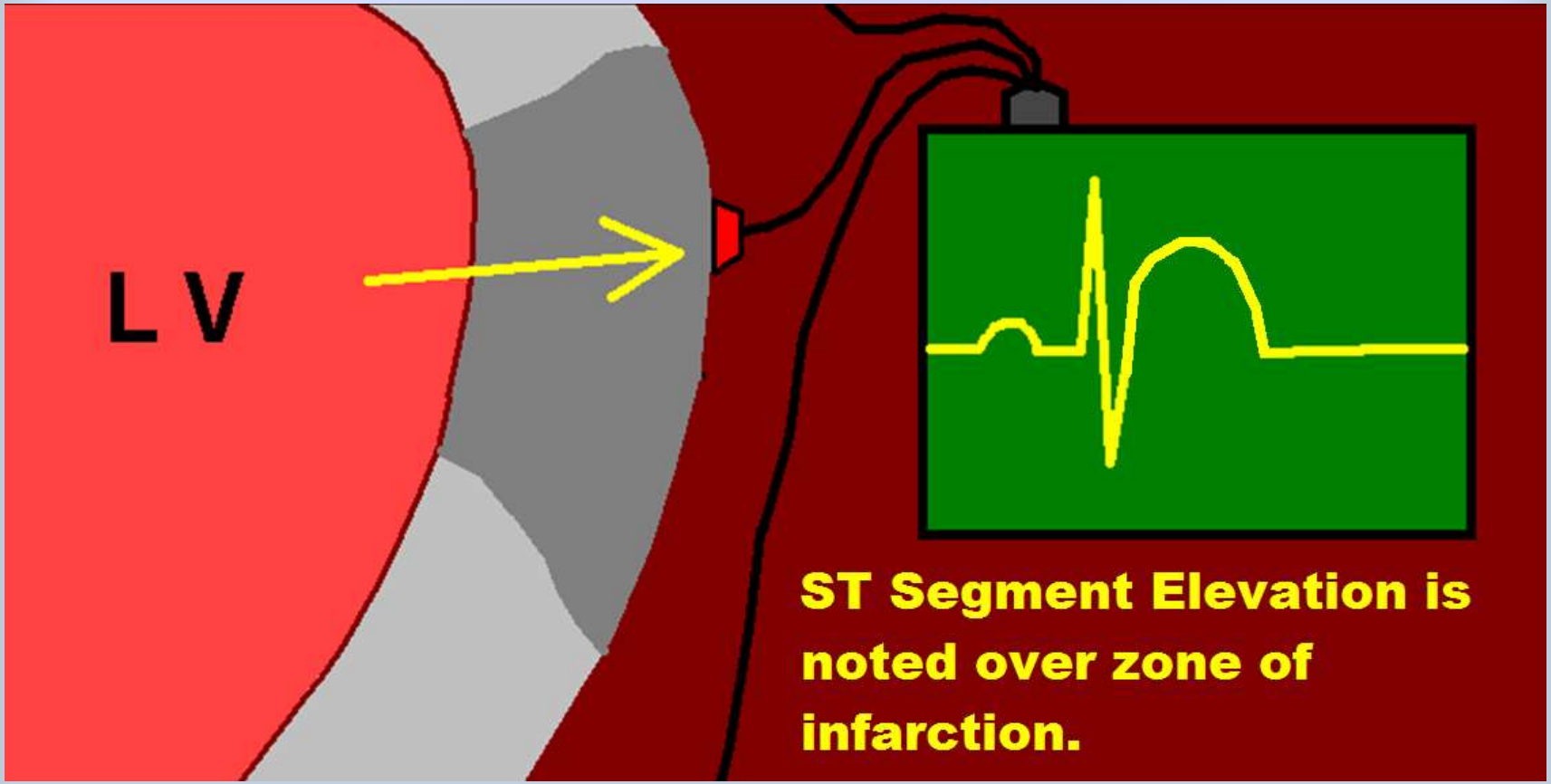
Non-ST Segment Elevation Myocardial Infarction.

"sub-endocardial MI" . . . "partial wall thickness"

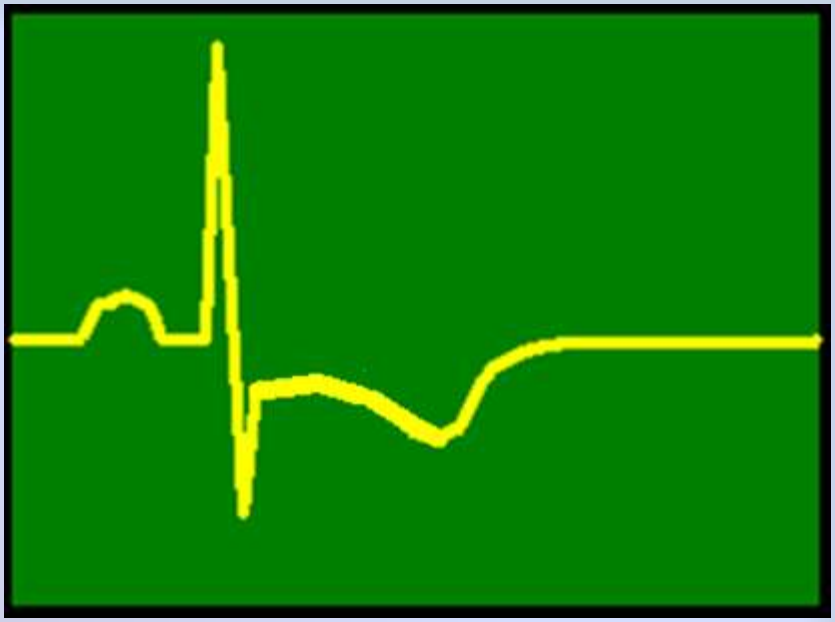


# STEMI

- ST Segment Elevation Myocardial Infarction.



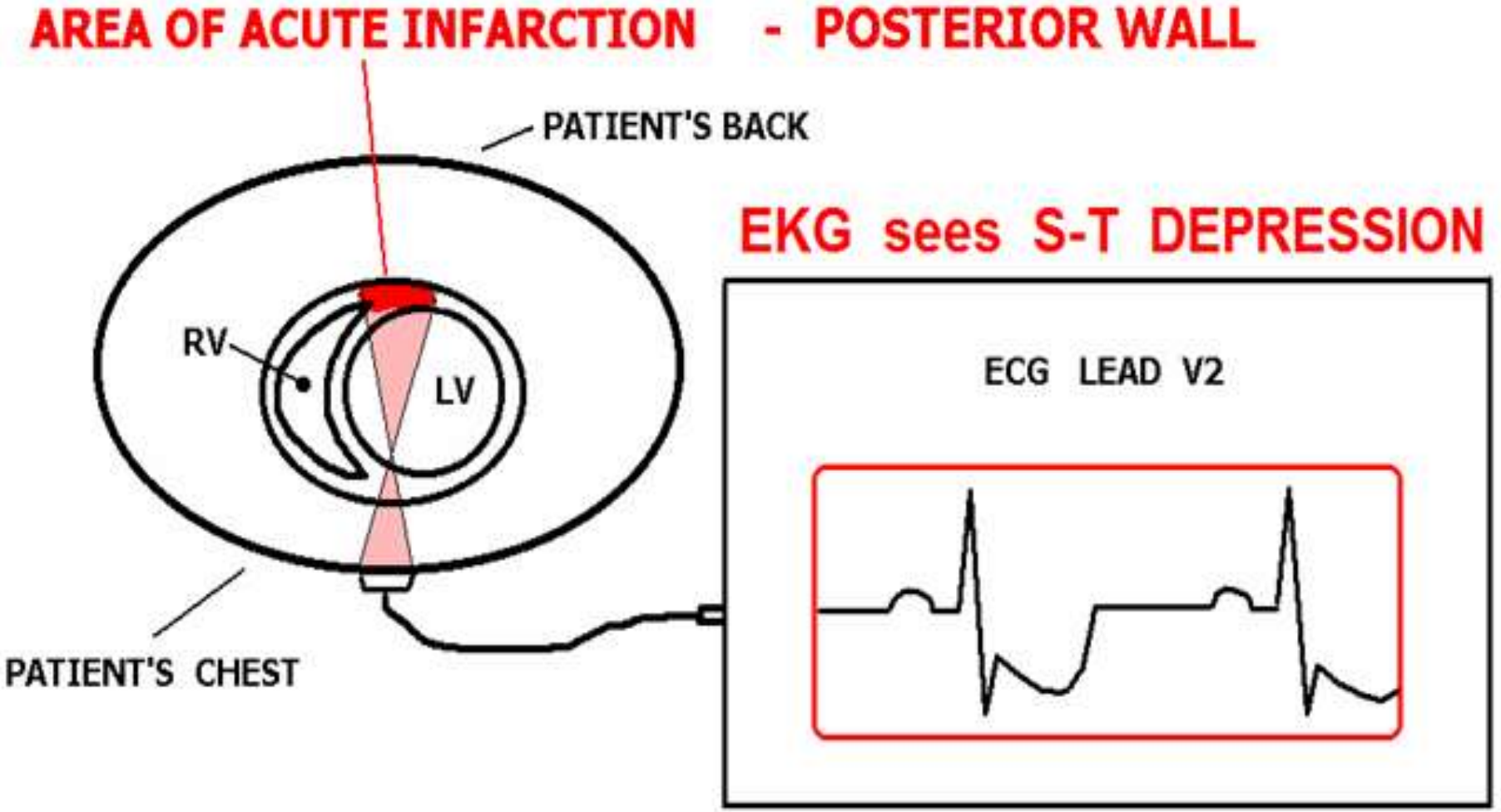
# ST Depression in Leads V1 – V4:



- Direct view of ISCHEMIA (anterior wall)
- Direct view of NSTEMI (anterior wall)
- Reciprocal view of STEMI (opposite side of heart - posterior wall)

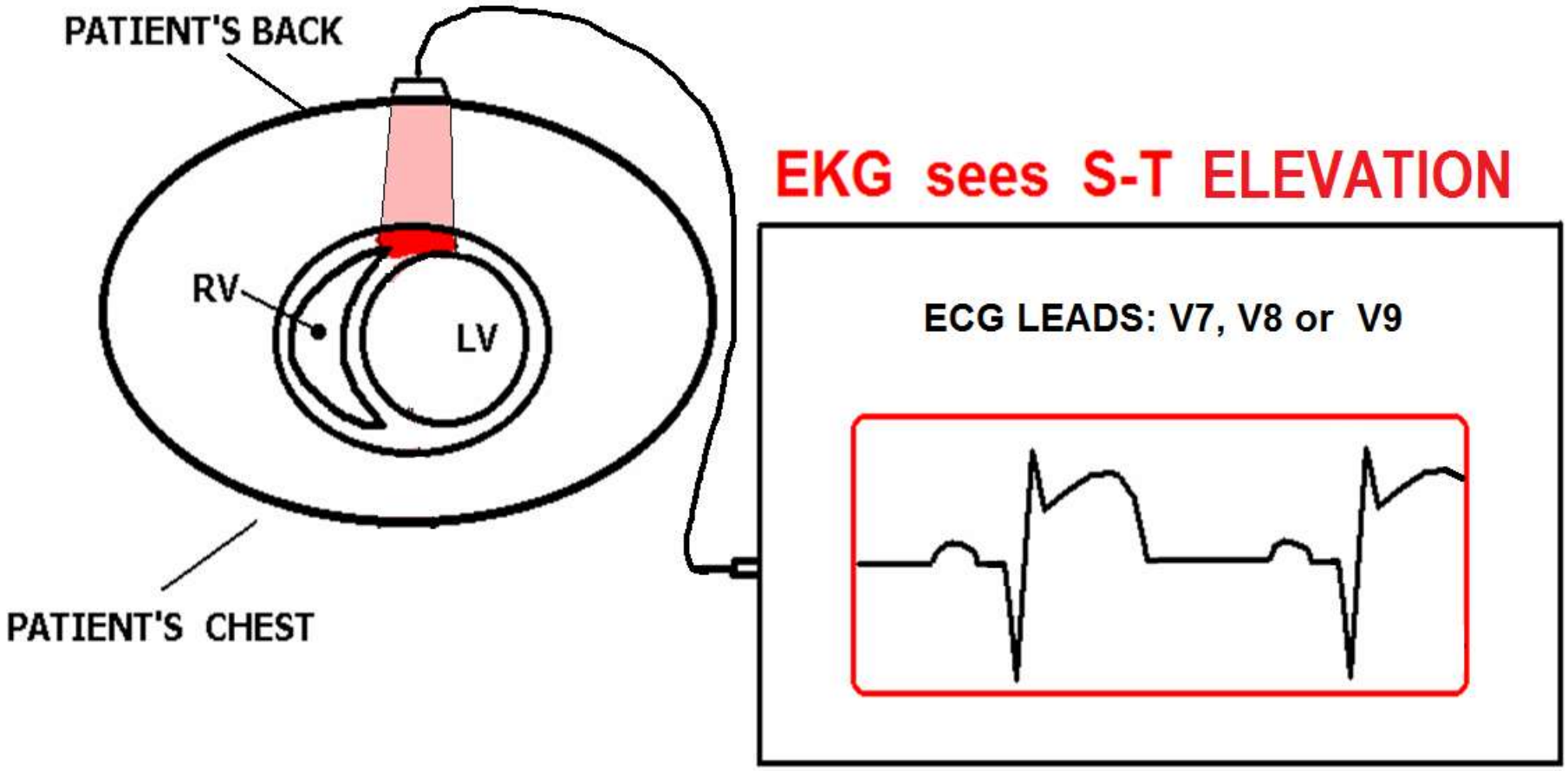
# HOW EKG VIEWS RECIPROCAL CHANGES

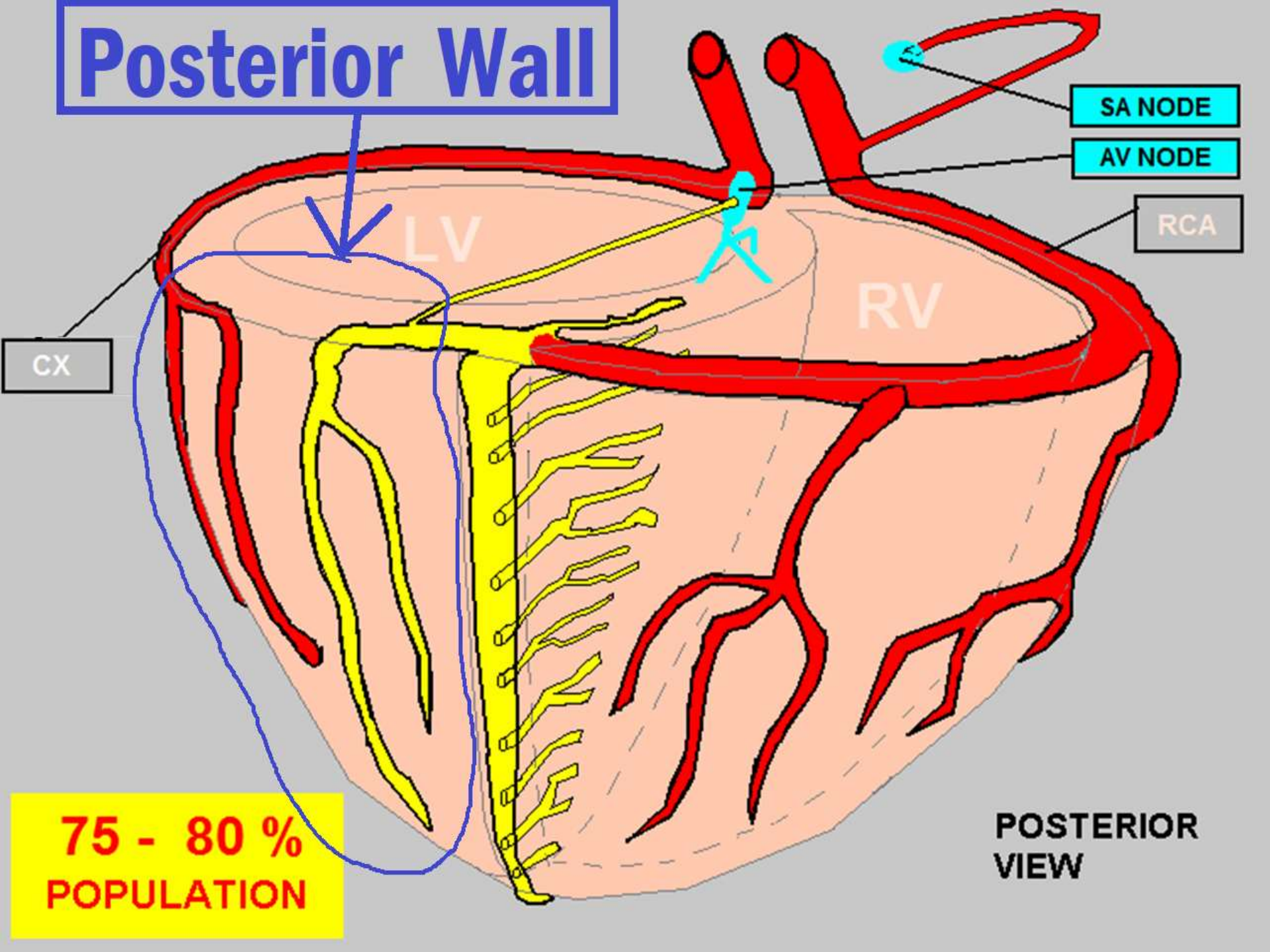
**EXAMPLE:**



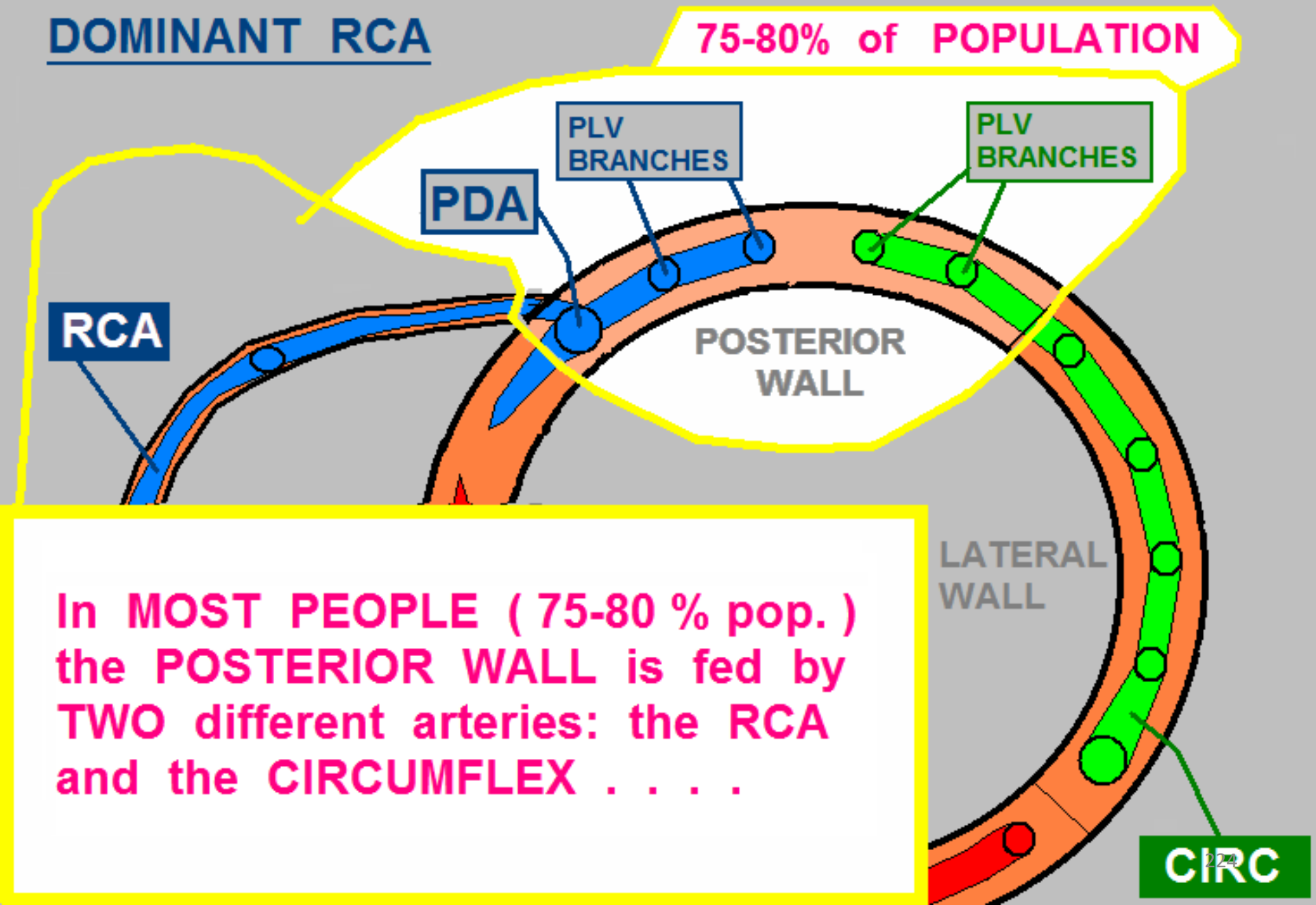
# If we put ECG leads on the BACK of a PATIENT who is having an

## ACUTE POSTERIOR WALL MI . . . . .



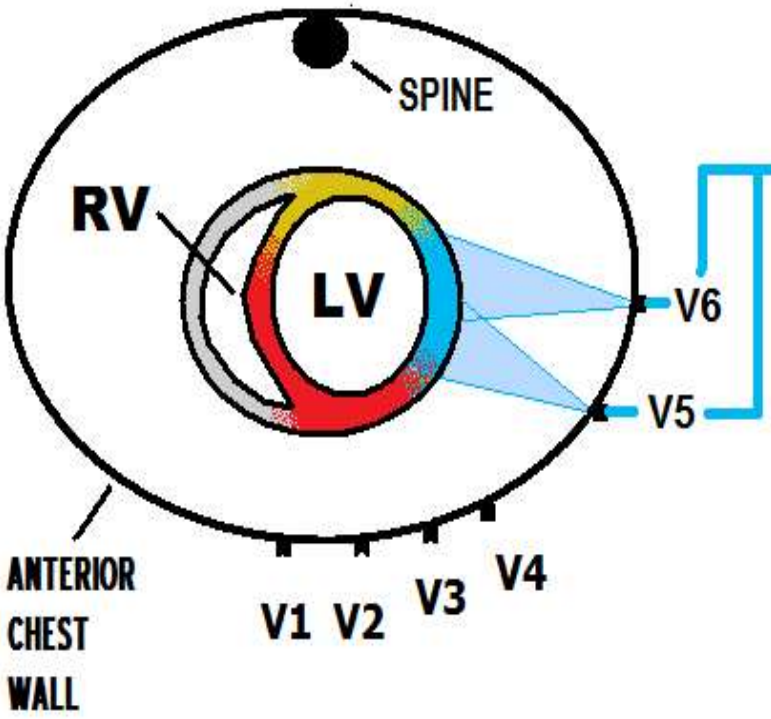

Posterior Wall
SA NODE
AV NODE
RCA
LV
RV
CX
75 - 80 %
POPULATION
POSTERIOR
VIEW

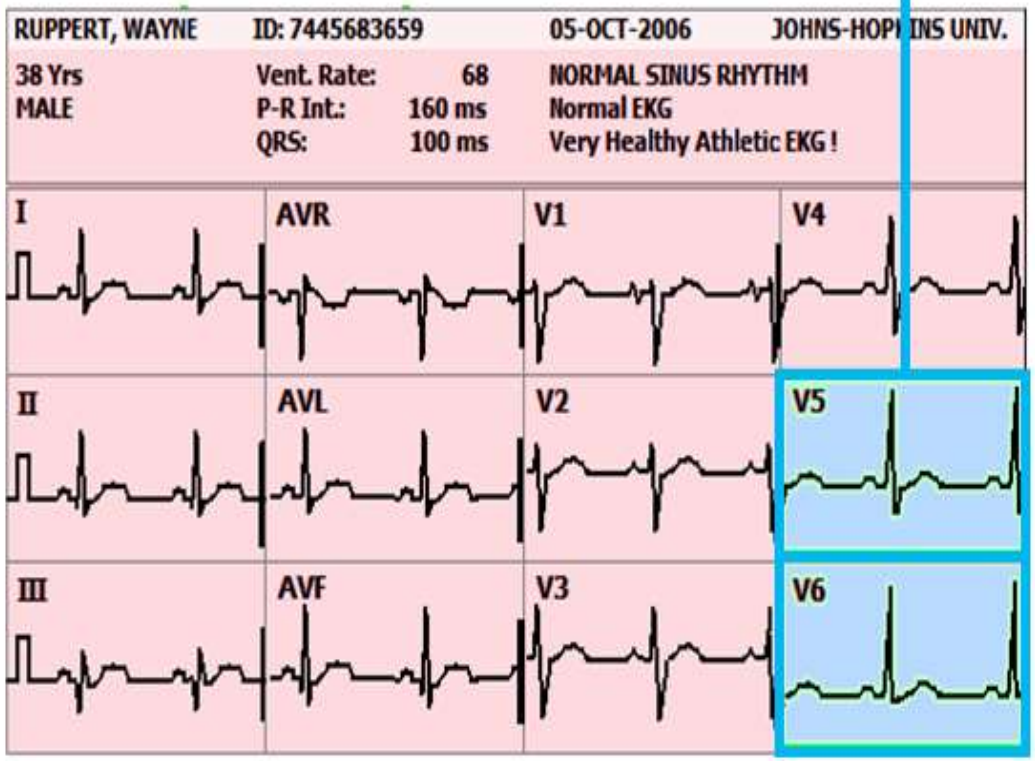
# POSTERIOR WALL BLOOD SUPPLY




In MOST PEOPLE ( 75-80 % pop. ) the POSTERIOR WALL is fed by TWO different arteries: the RCA and the CIRCUMFLEX . . . .

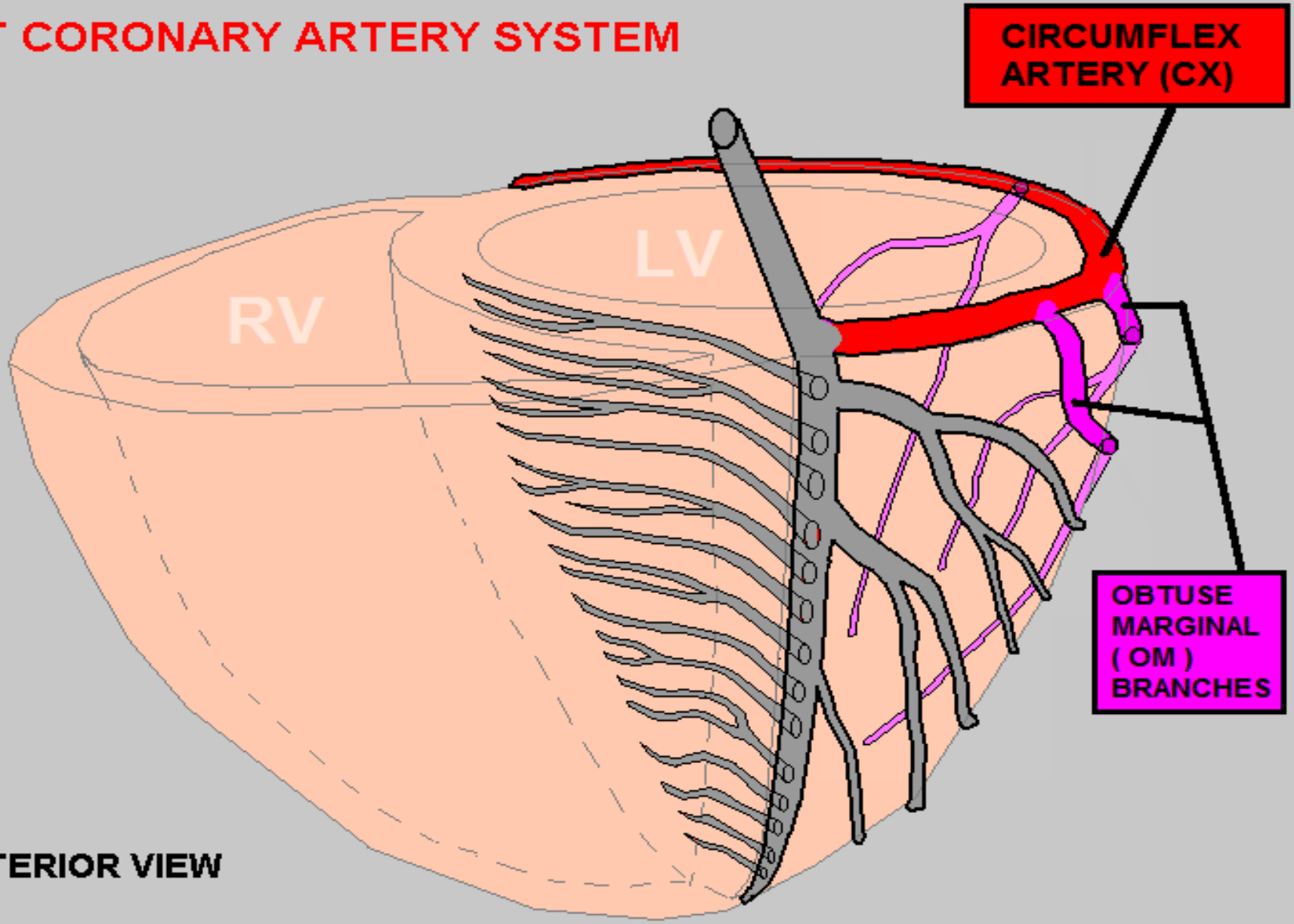
# V5 - V6 VIEW THE LATERAL WALL
## of the LEFT VENTRICLE




**Which Coronary Artery typically Supplies the LATERAL WALL ?**

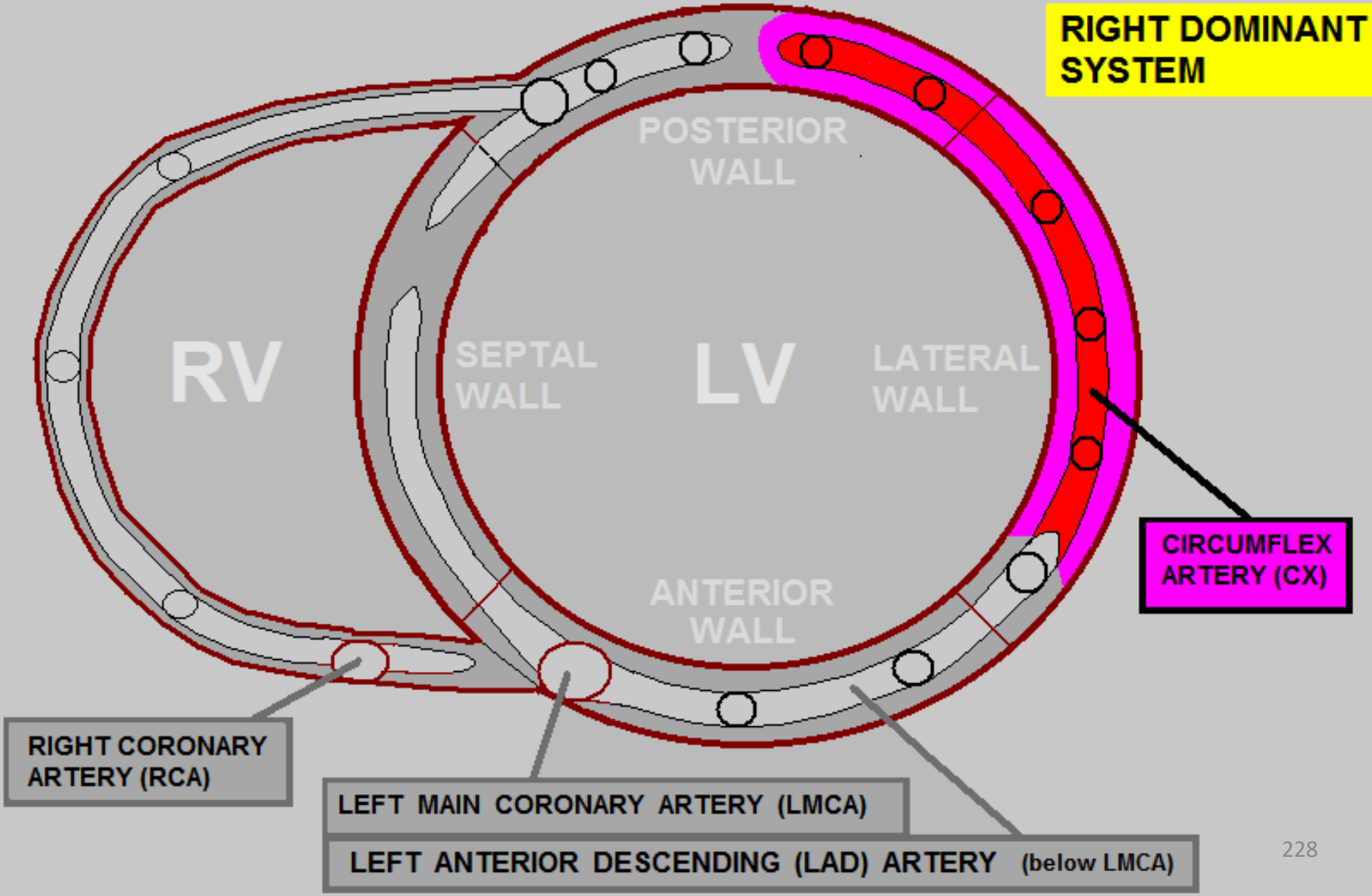
# LEFT CORONARY ARTERY SYSTEM



*cutaway view of the*

# CIRCUMFLEX ARTERY (CX) DISTRIBUTION

## SUPPLIES 20 - 30 % of the LV MUSCLE MASS



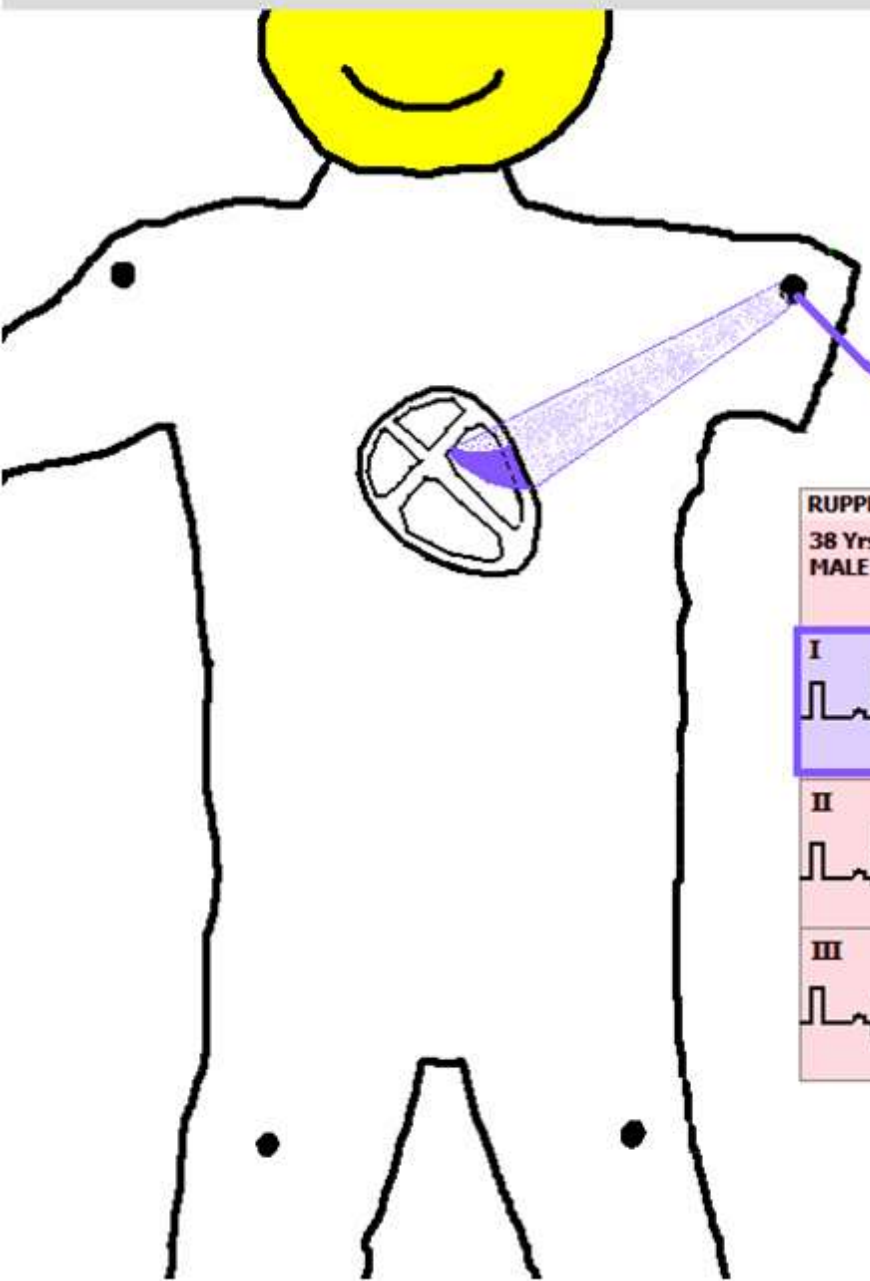
HELPFUL HINT . . . MEMORIZE THIS !

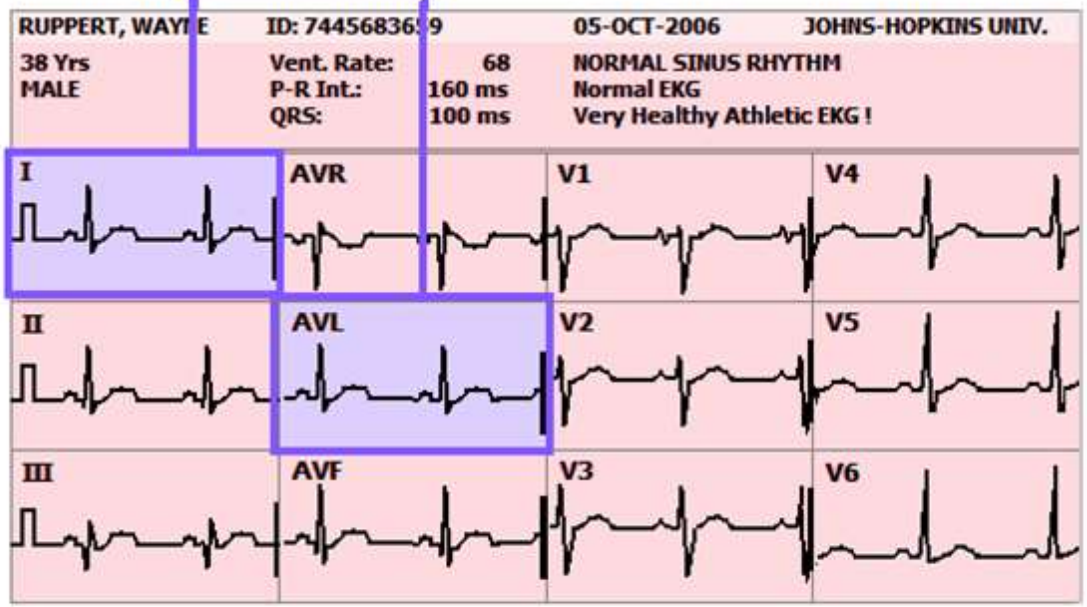
# CIRCUMFLEX ARTERY ( CX )

RIGHT DOMINANT SYSTEMS

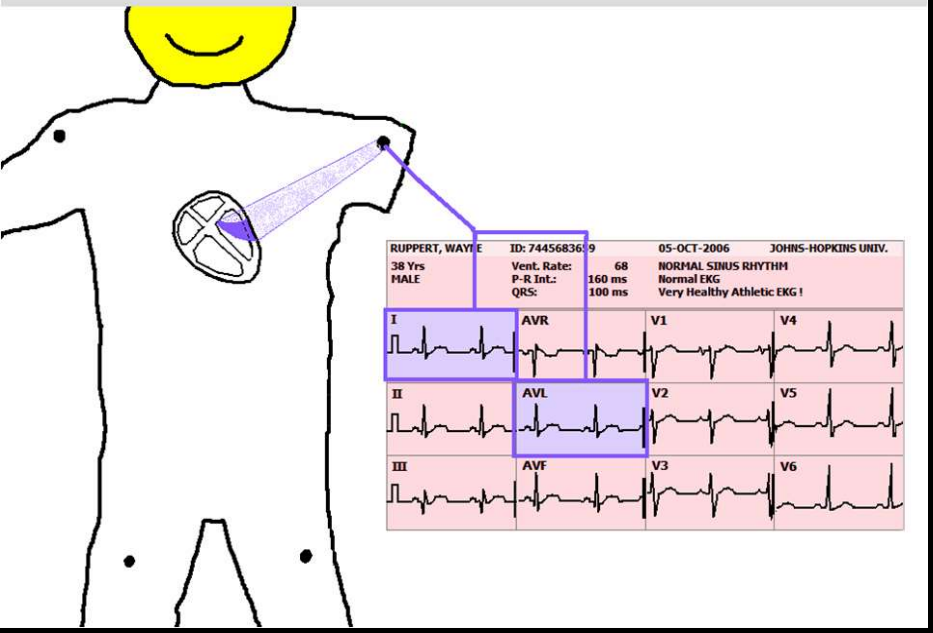
- LEFT ATRIUM
- SINUS NODE ( 5% of the population )
- LEFT VENTRICLE: 20 - 30 % of muscle mass
  - LATERAL WALL
  - up to 1/2 of POSTERIOR WALL

# LEADS I and aVL VIEW the LATERAL - ANTERIOR WALL

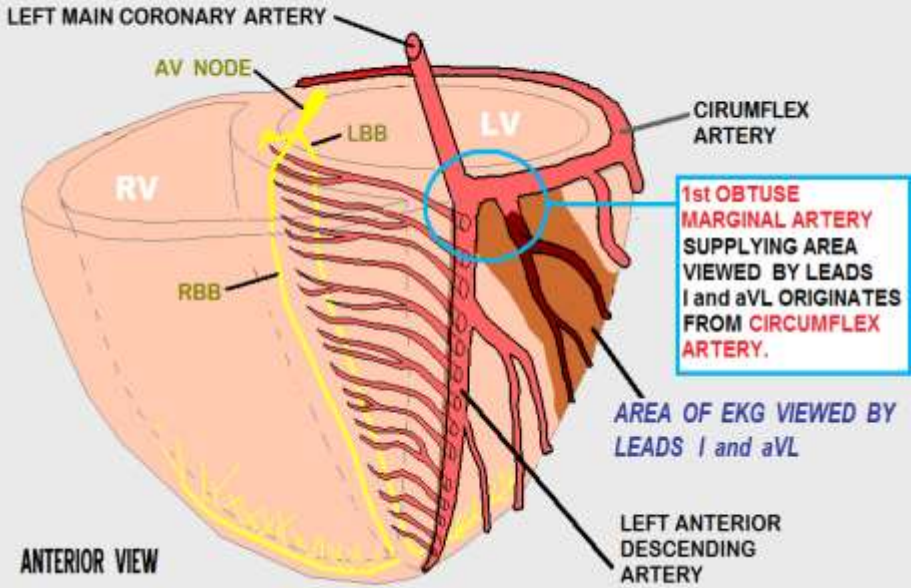




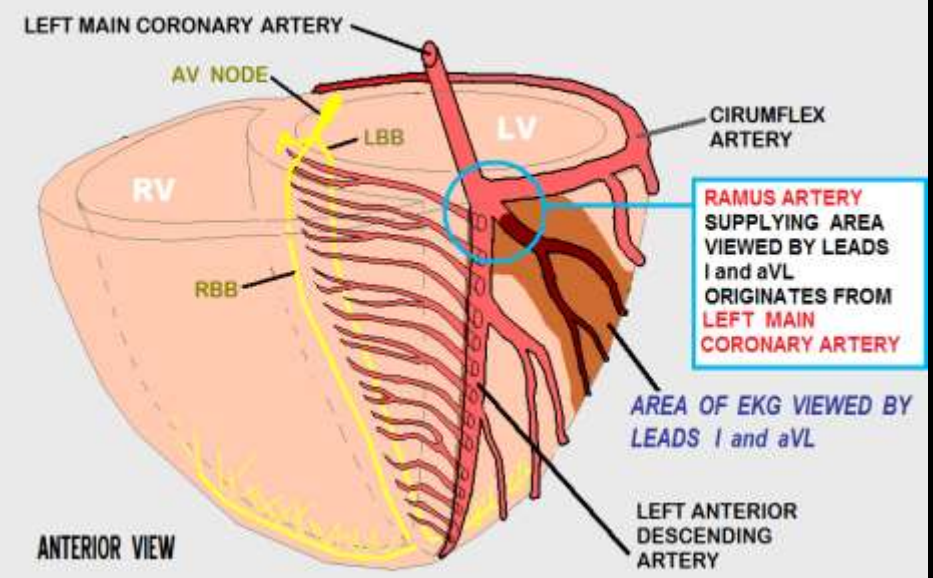
## LEADS I and aVL VIEW the LATERAL - ANTERIOR WALL



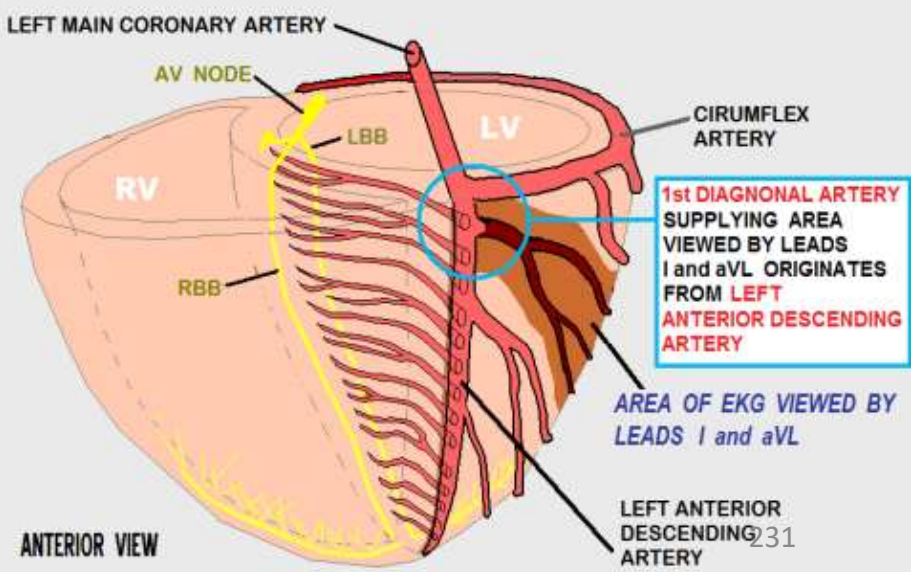

## OCCLUSION of OBTUSE MARGINAL ARTERY

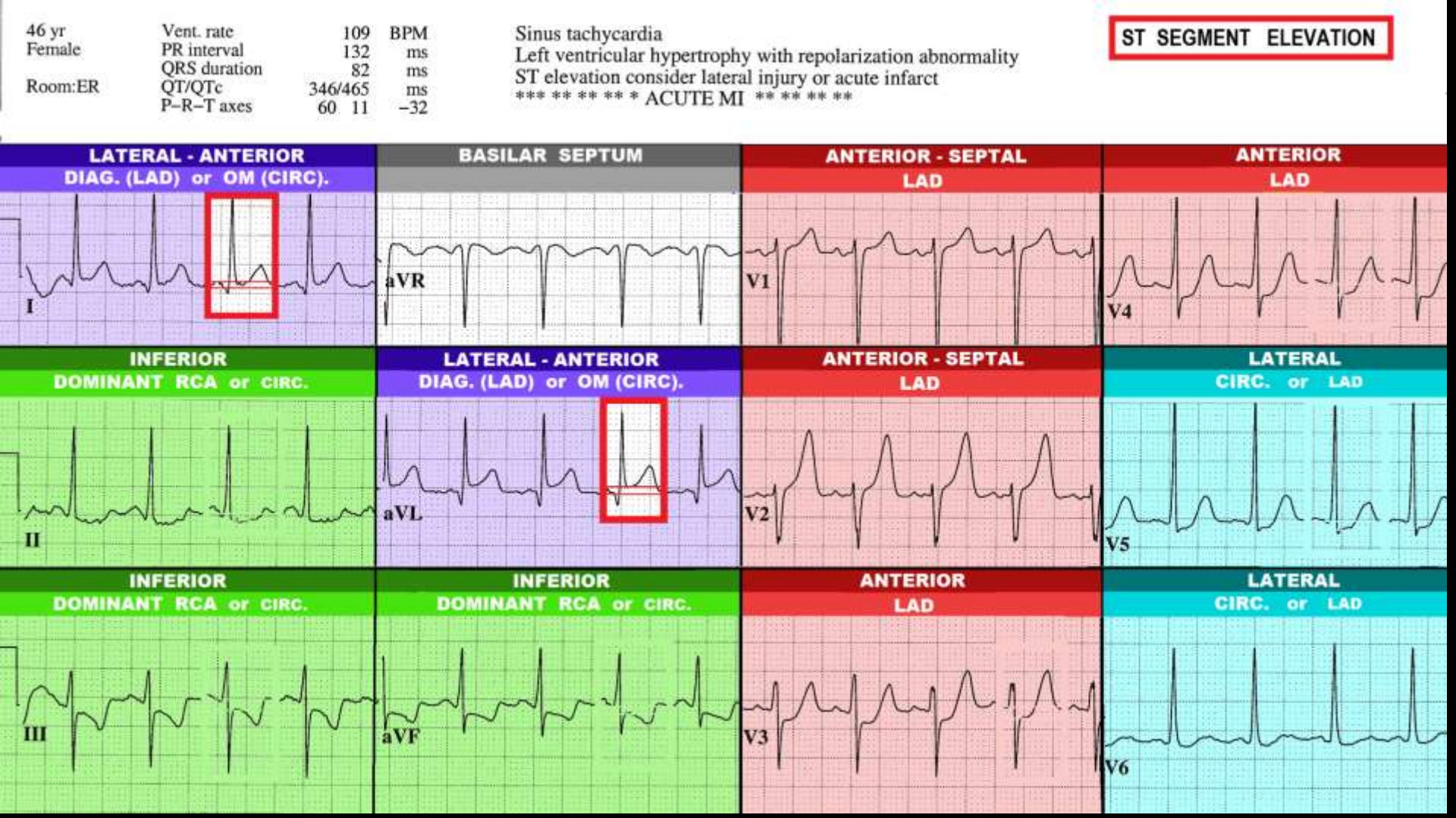



## OCCLUSION of RAMUS ARTERY




## OCCLUSION of DIAGONAL ARTERY

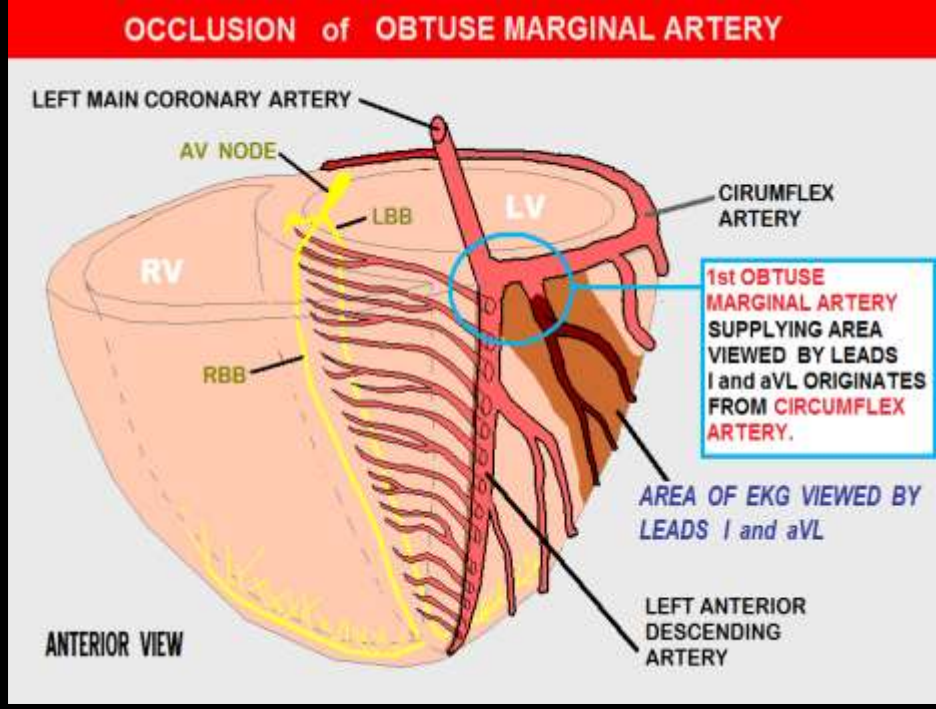


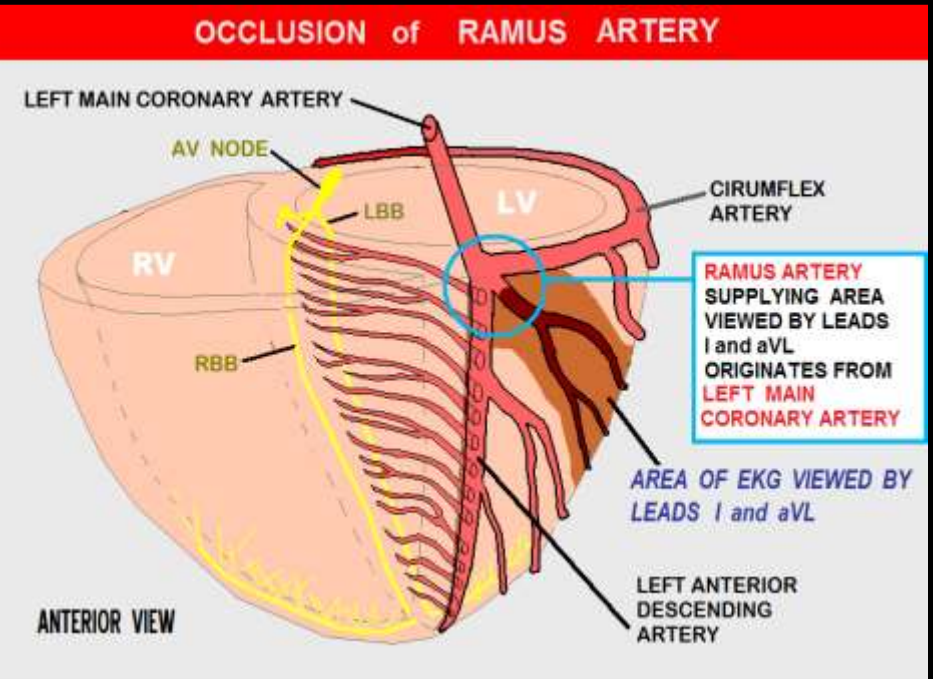


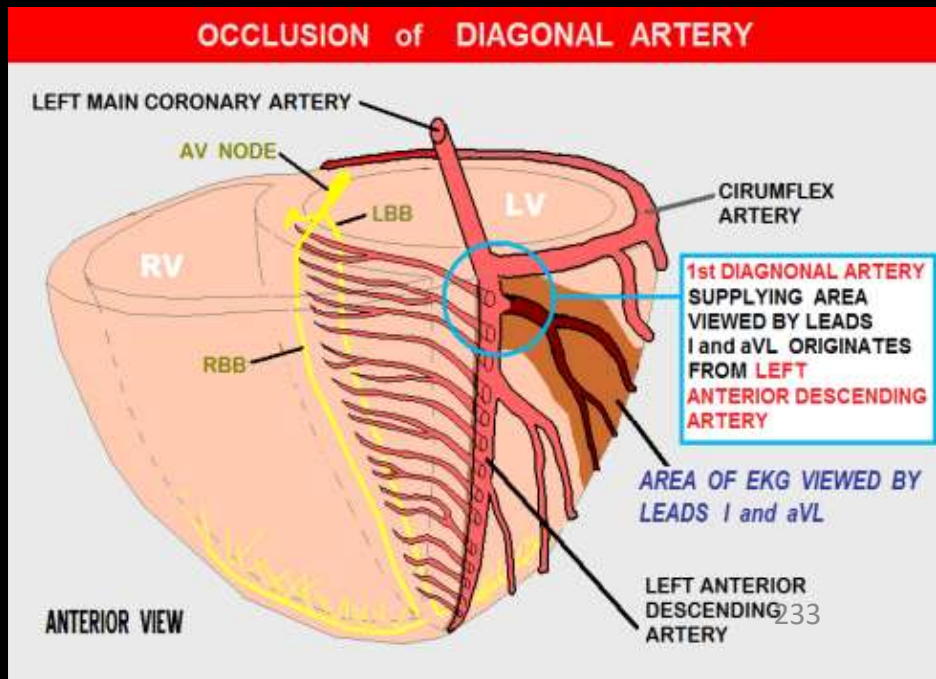

# ST Segment elevation ONLY in Leads I and aVL

**ST Elevation isolated to Leads I and aVL - usually indicates the "Culprit Artery" is most likely One of the following:**

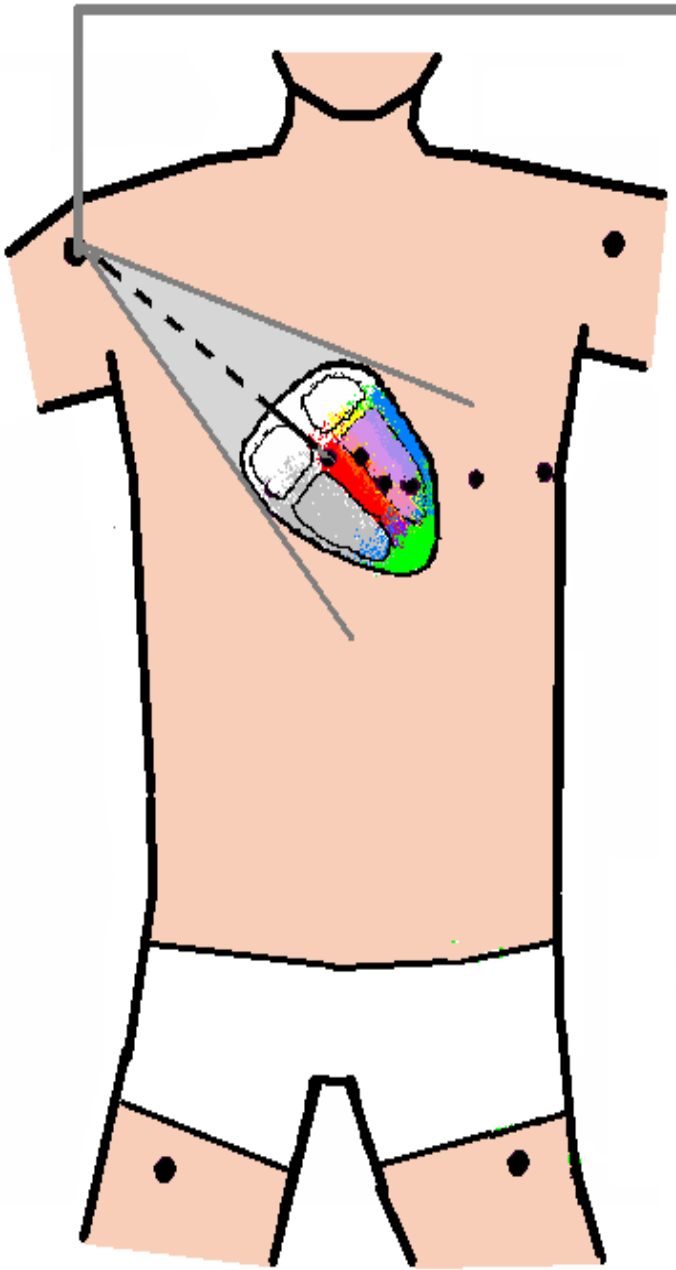
- **RAMUS BRANCH**
- **1st DIAGONAL off of LAD**
- **1st OBTUSE MARGINAL off of CIRCUMFLEX**

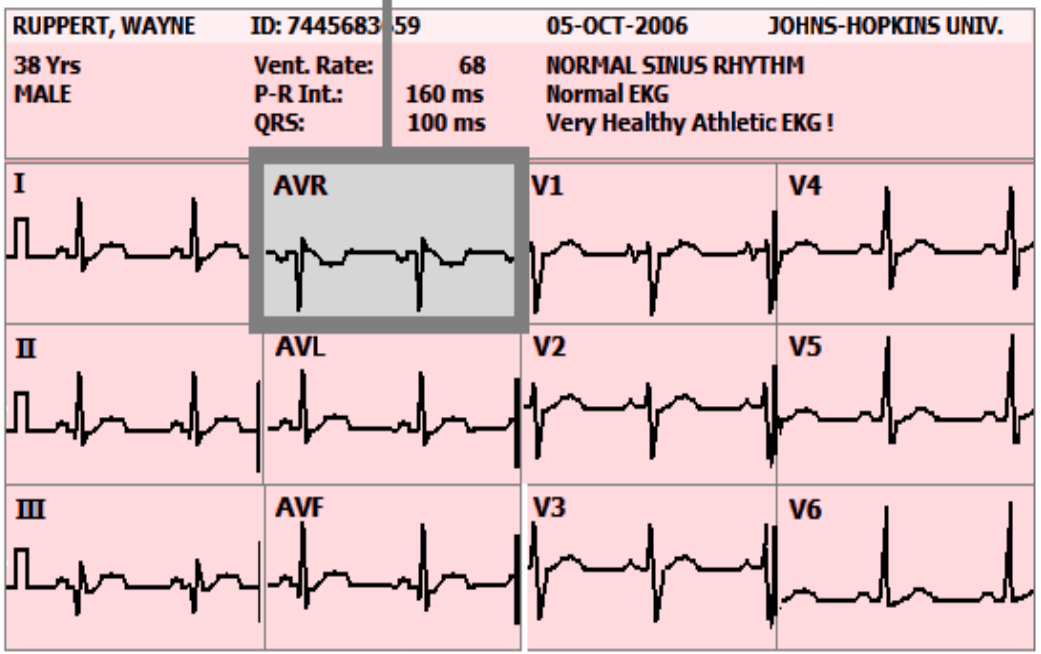


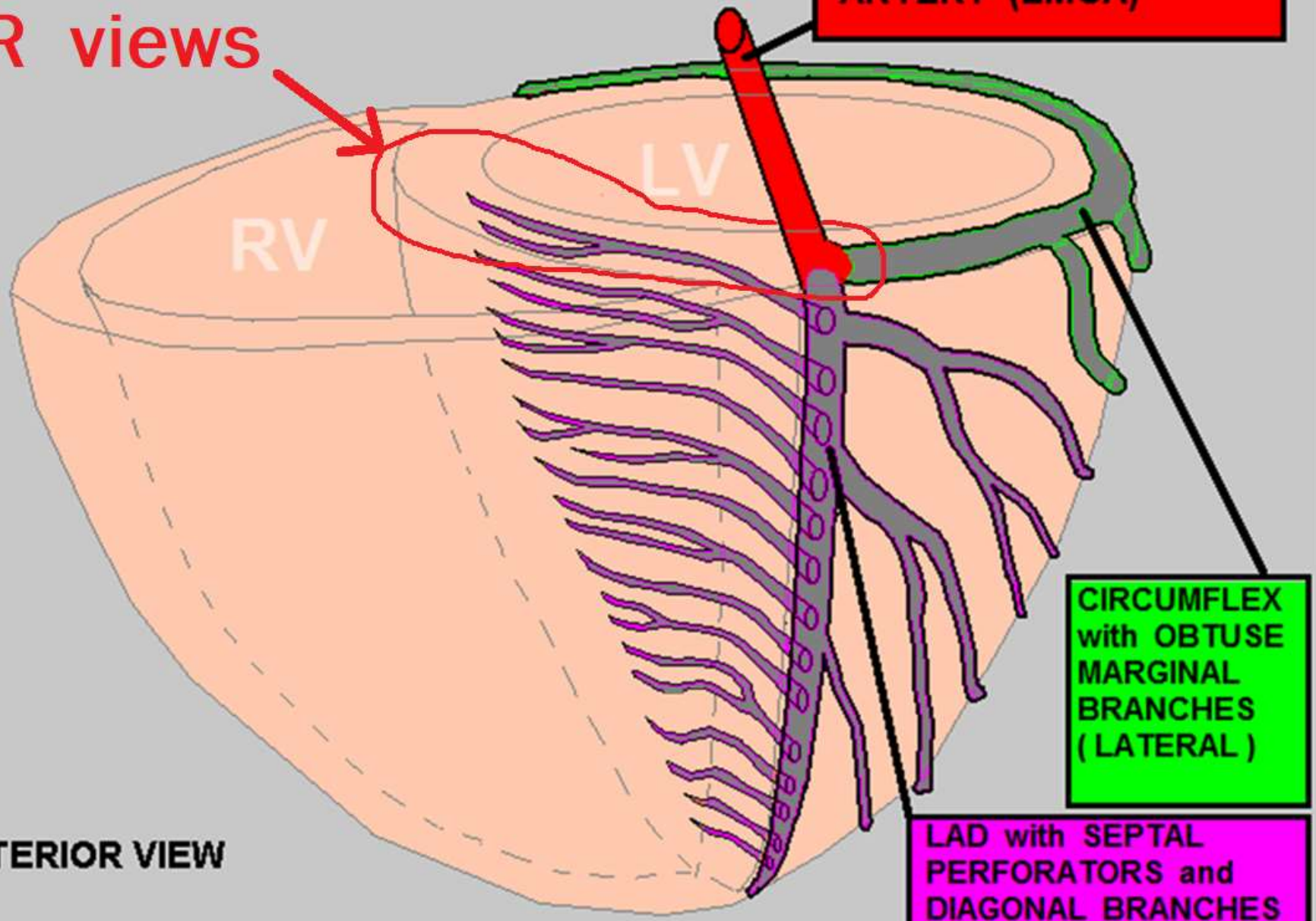
# Lead AVR Views the BASILAR SEPTUM (region of the Bundle of His)





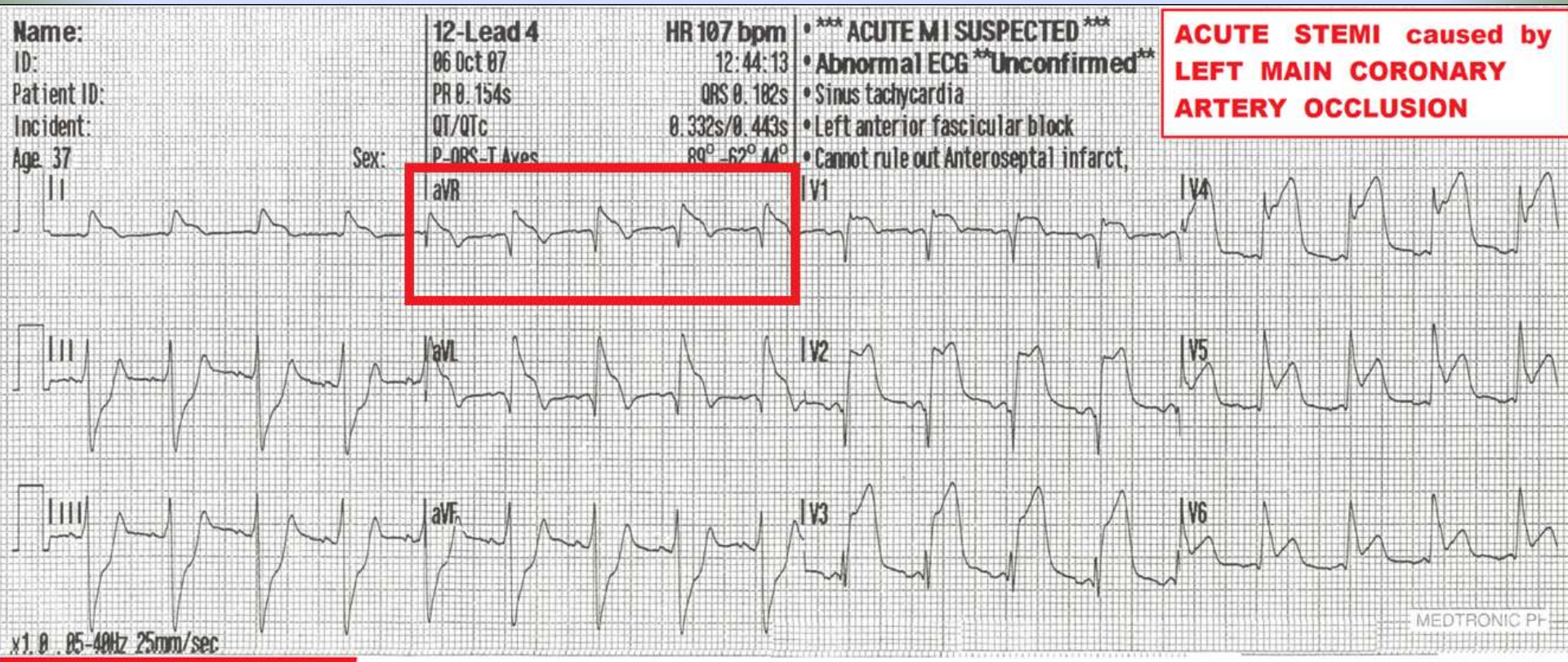
# LEFT CORONARY ARTERY SYSTEM

## AVR views



When LEAD AVR shows ST Elevation:

- **STEMI:** consider occlusion of the Left Main Coronary Artery.

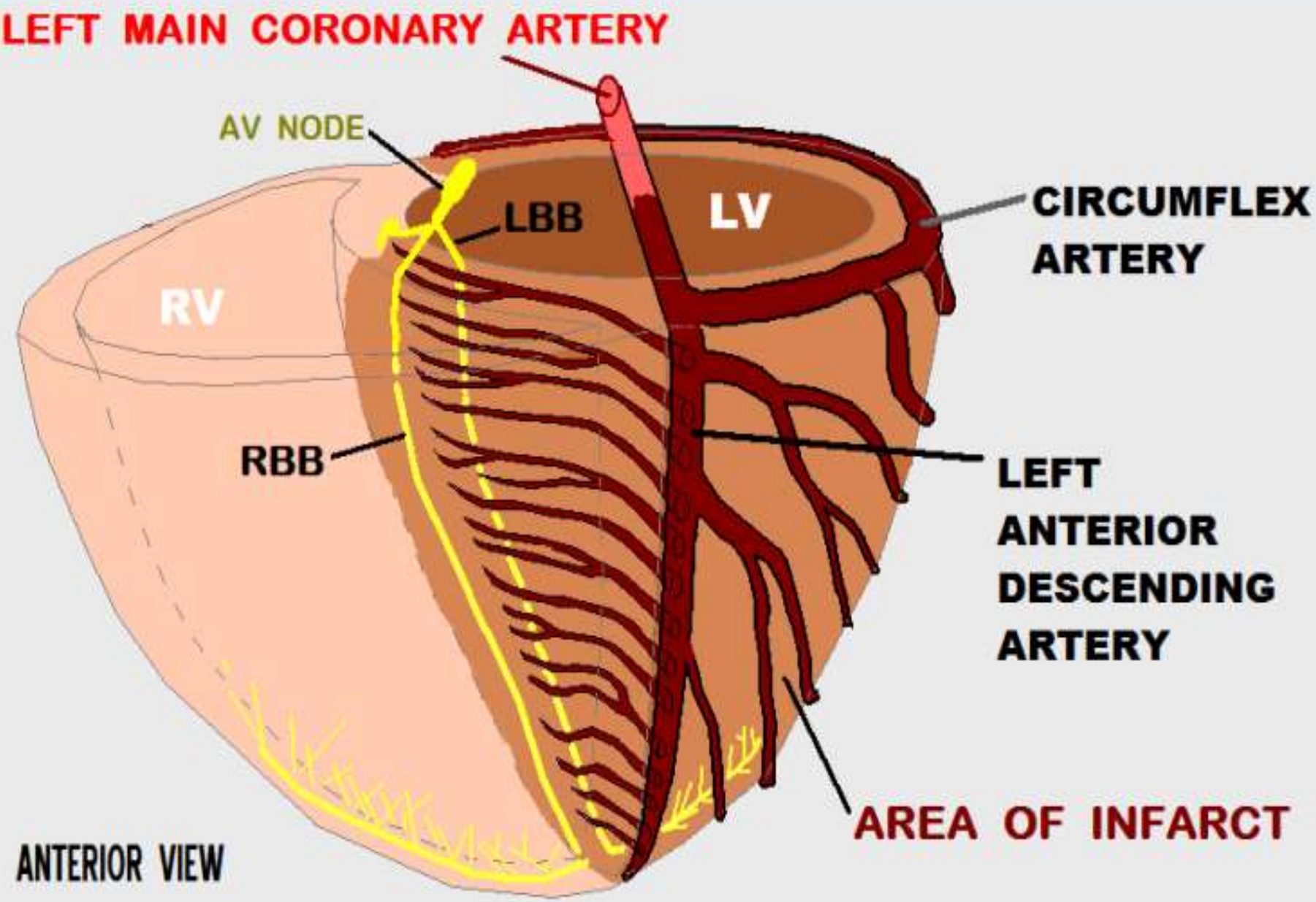


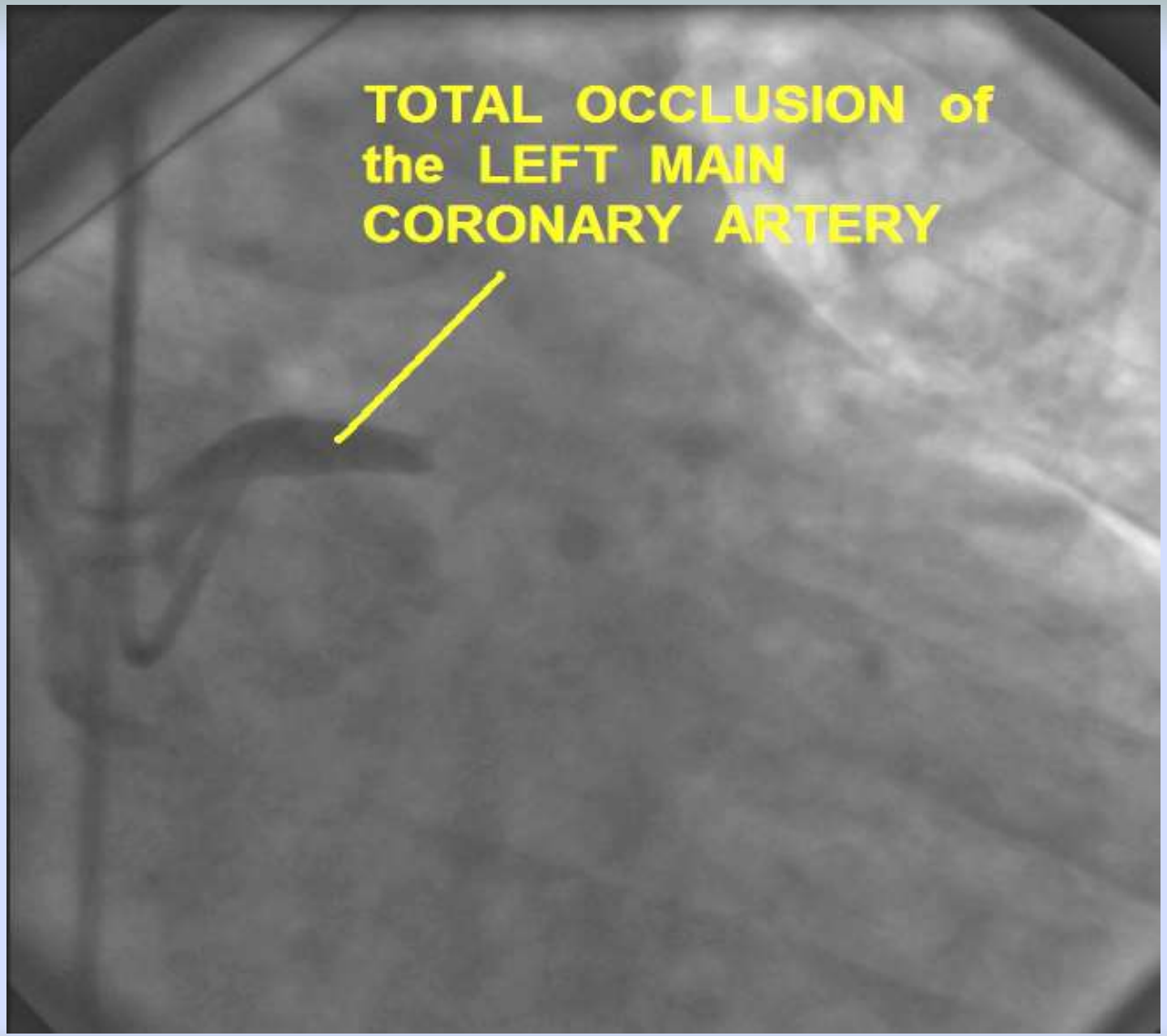

**ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION**

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

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

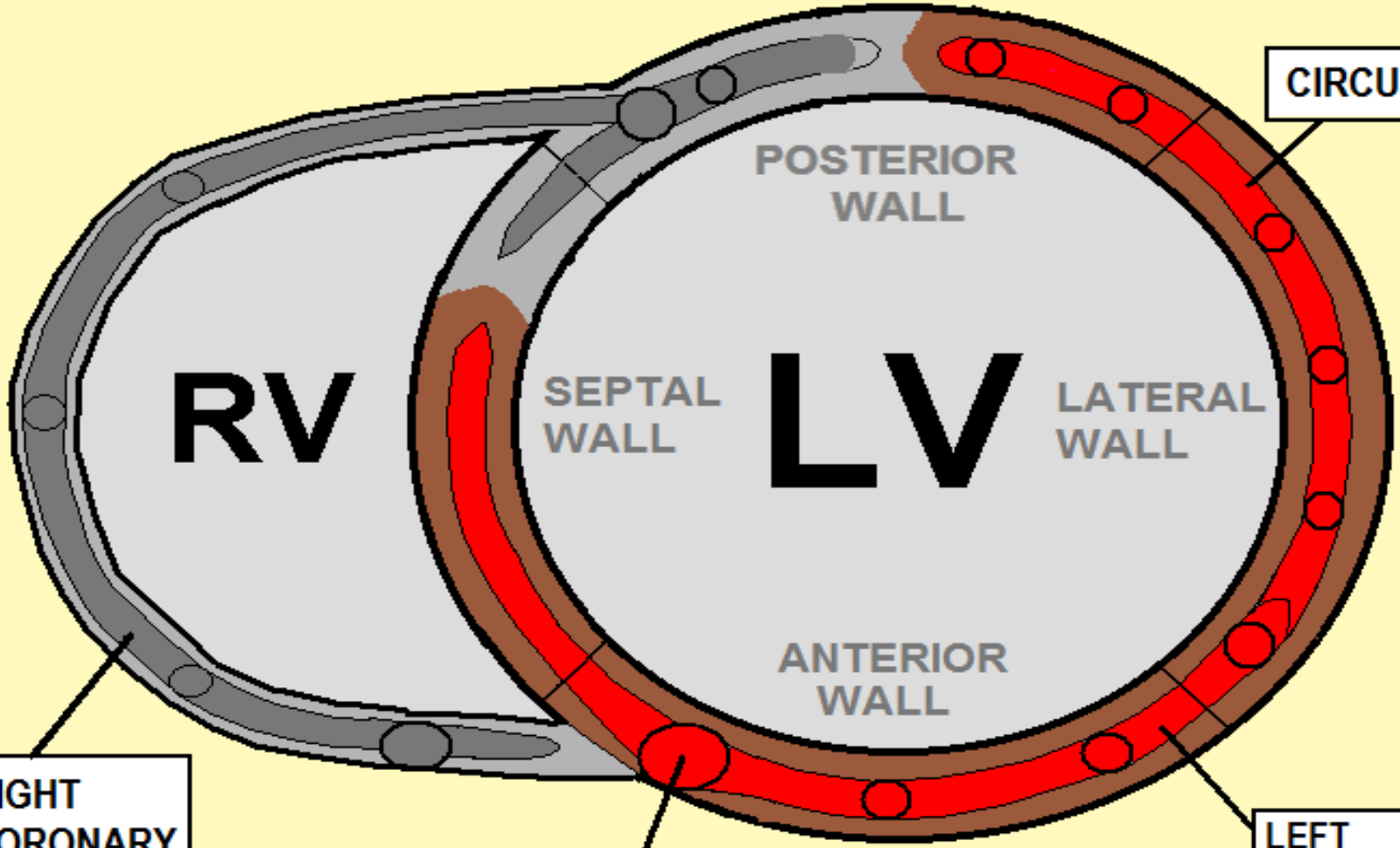
# OCCLUSION of the LEFT MAIN CORONARY ARTERY




TOTAL OCCLUSION of
the LEFT MAIN
CORONARY ARTERY

# The LEFT MAIN CORONARY ARTERY

## *SUPPLIES 75 - 100 % of the LEFT VENTRICULAR MUSCLE MASS*



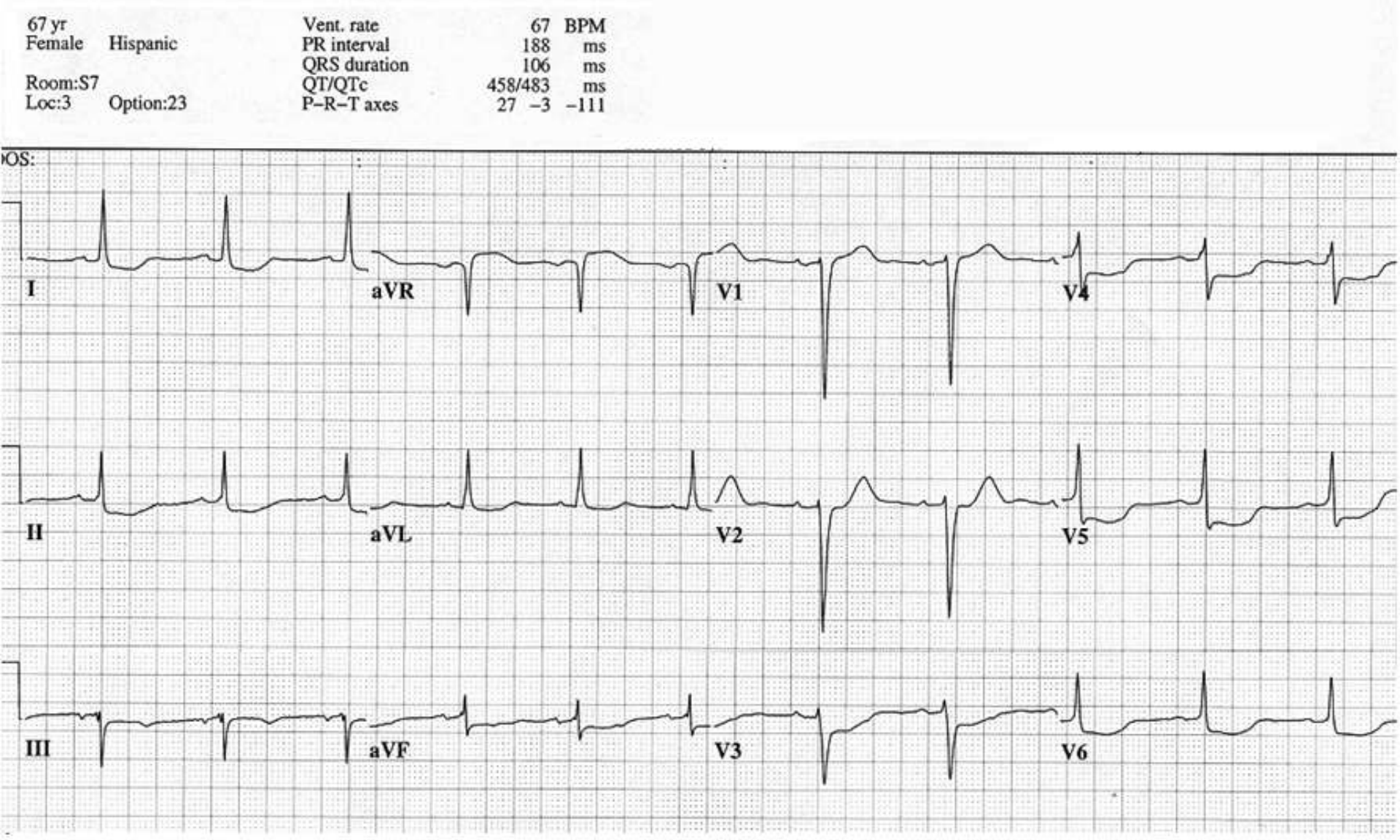
When LEAD AVR shows ST Elevation:

- **STEMI:** consider occlusion of the Left Main Coronary Artery.

When LEAD AVR shows ST Elevation:

- **STEMI:** consider occlusion of the Left Main Coronary Artery.
- **NSTEMI** and **Unstable Angina** consider LMCA Occlusion – or TRIPLE VESSEL DISEASE

**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**


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

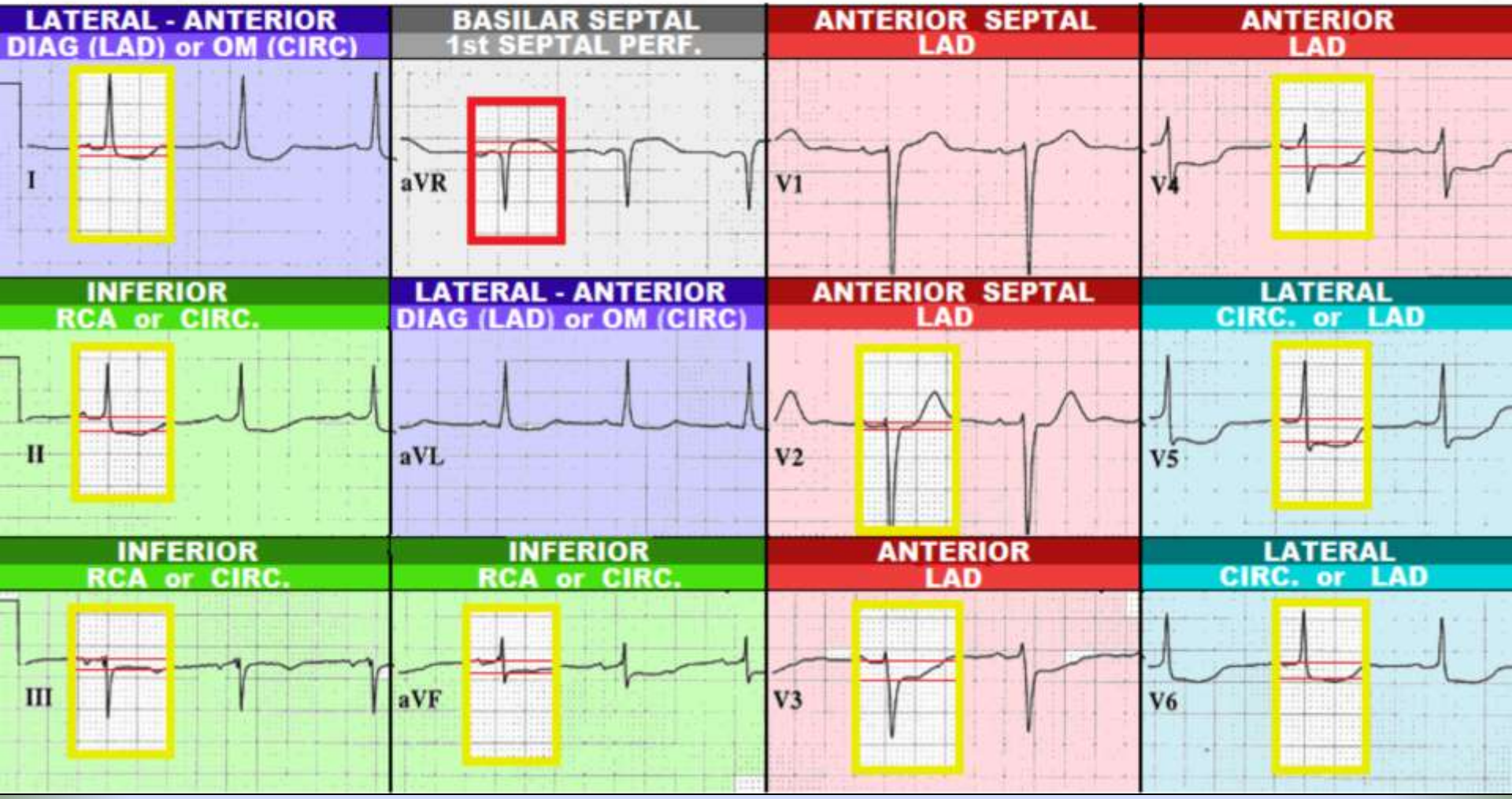
67 yr
Female Hispanic

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**

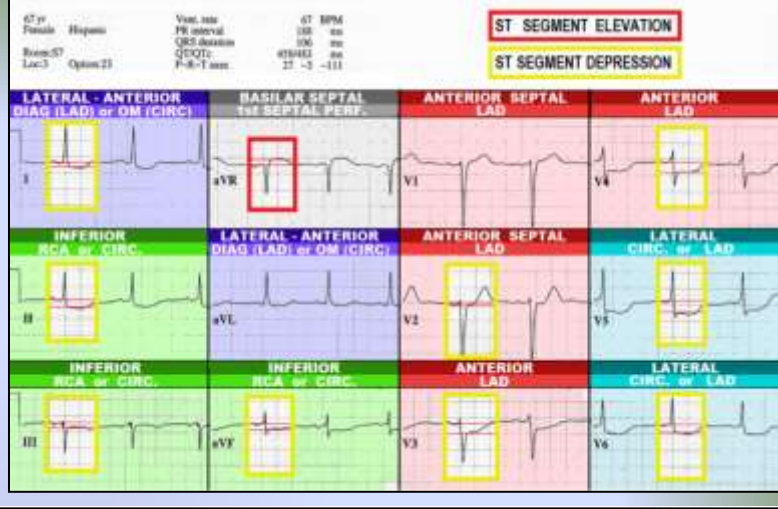
**ST SEGMENT DEPRESSION**

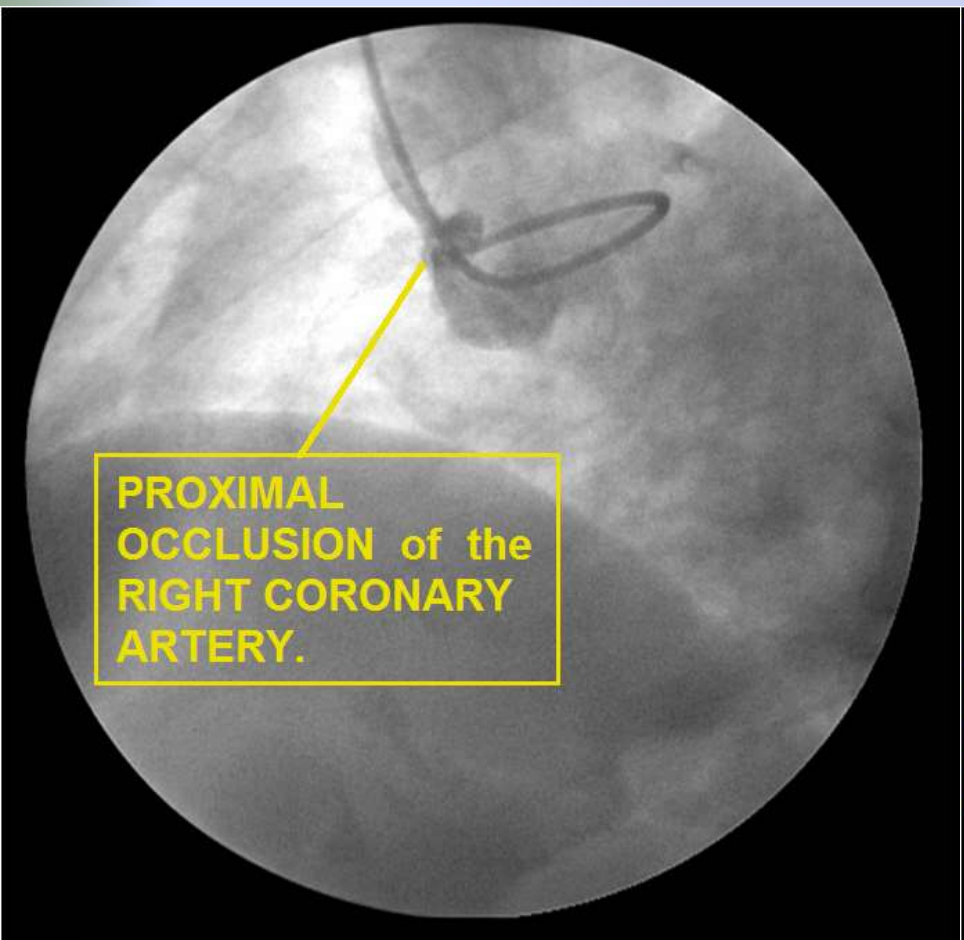


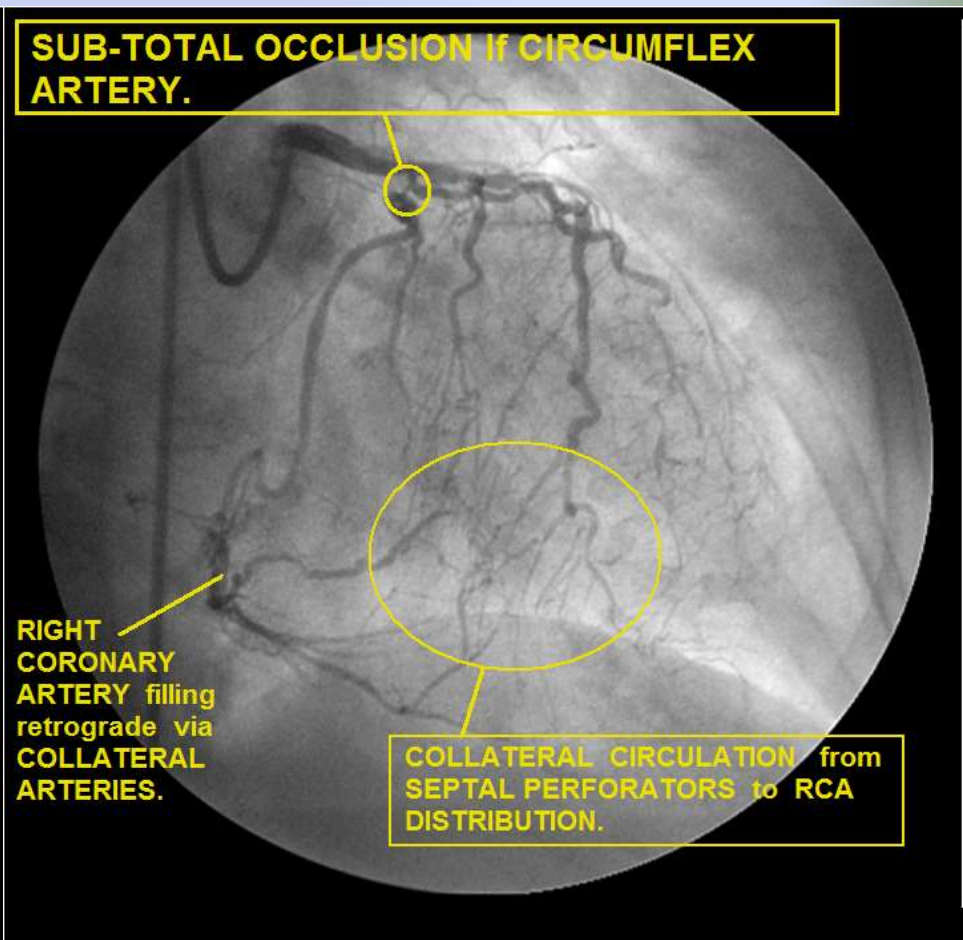
# GLOBAL ISCHEMIA

- **ST Elevation Lead aVR**
- **ST Depression in 8 or more other Leads**
- **Indicates either SUB-TOTALLY OCCLUDED LEFT MAIN CORONARY ARTERY – or – TRIPLE VESSEL DISEASE.**
- ***MOST PATIENTS WITH THIS ECG PRESENTATION REQUIRE OPEN HEART SURGERY.***

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



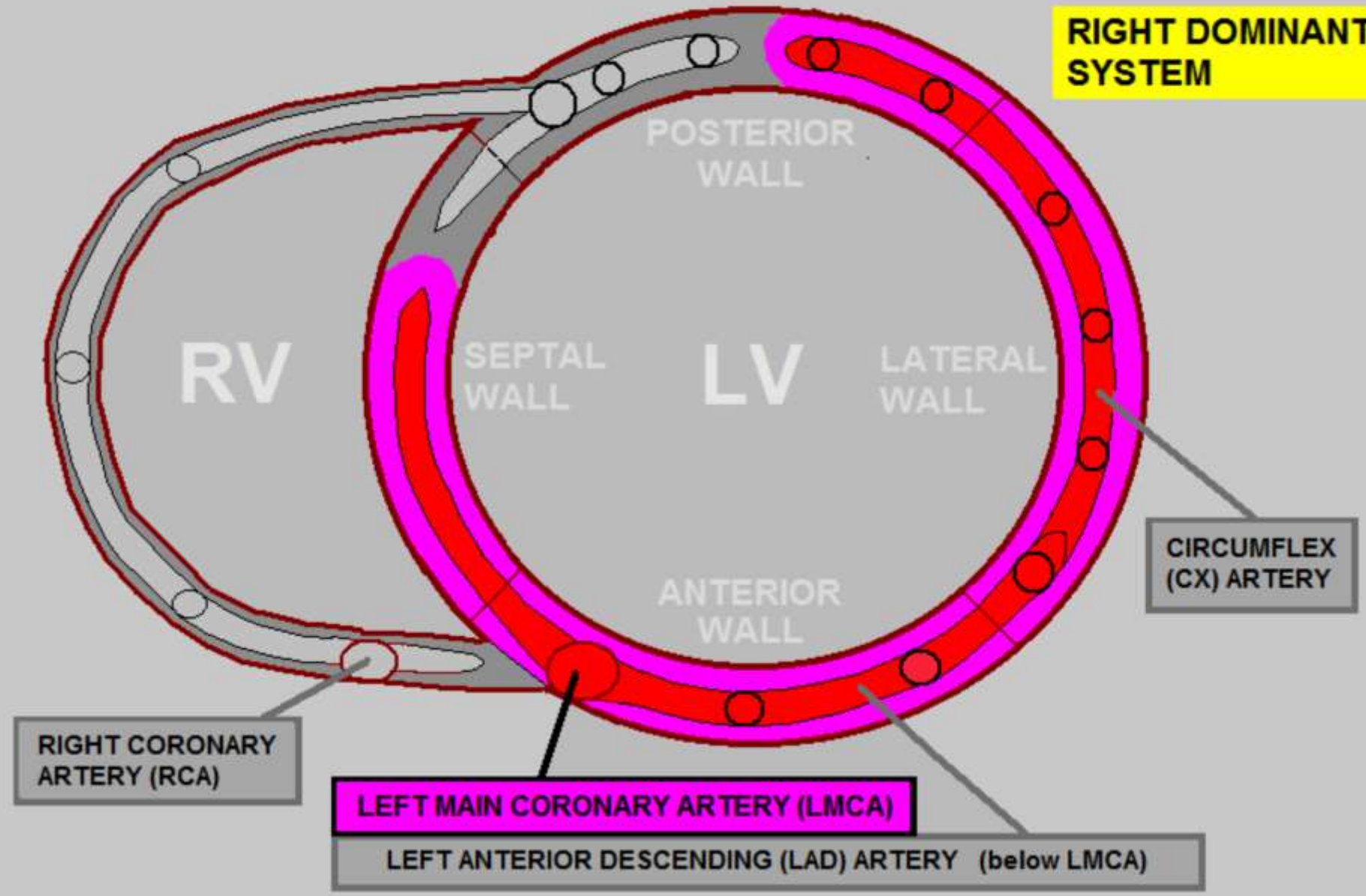



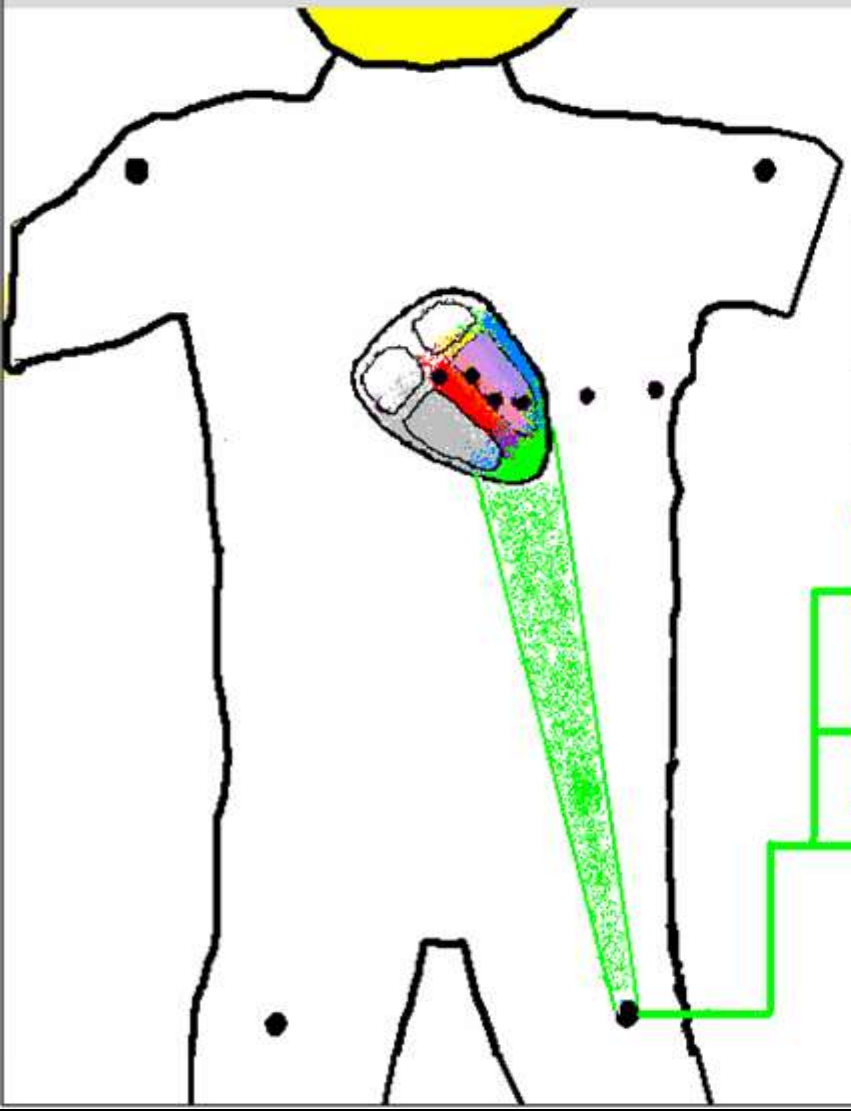
*cutaway view of the*

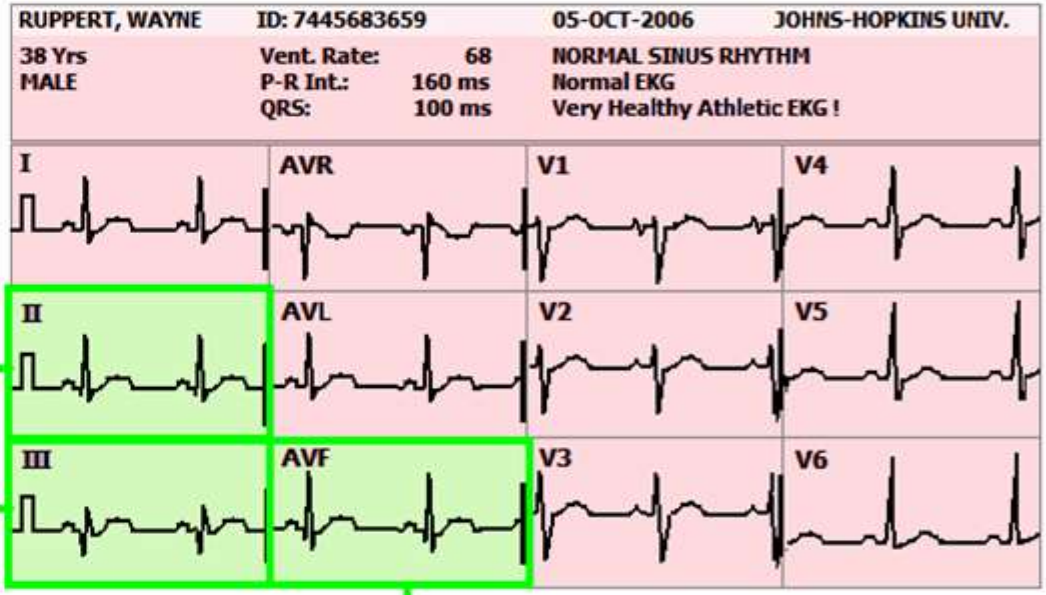
# LEFT MAIN CORONARY ARTERY (LMCA)

## SUPPLIES APPROXIMATELY 75% OF LV MUSCLE MASS

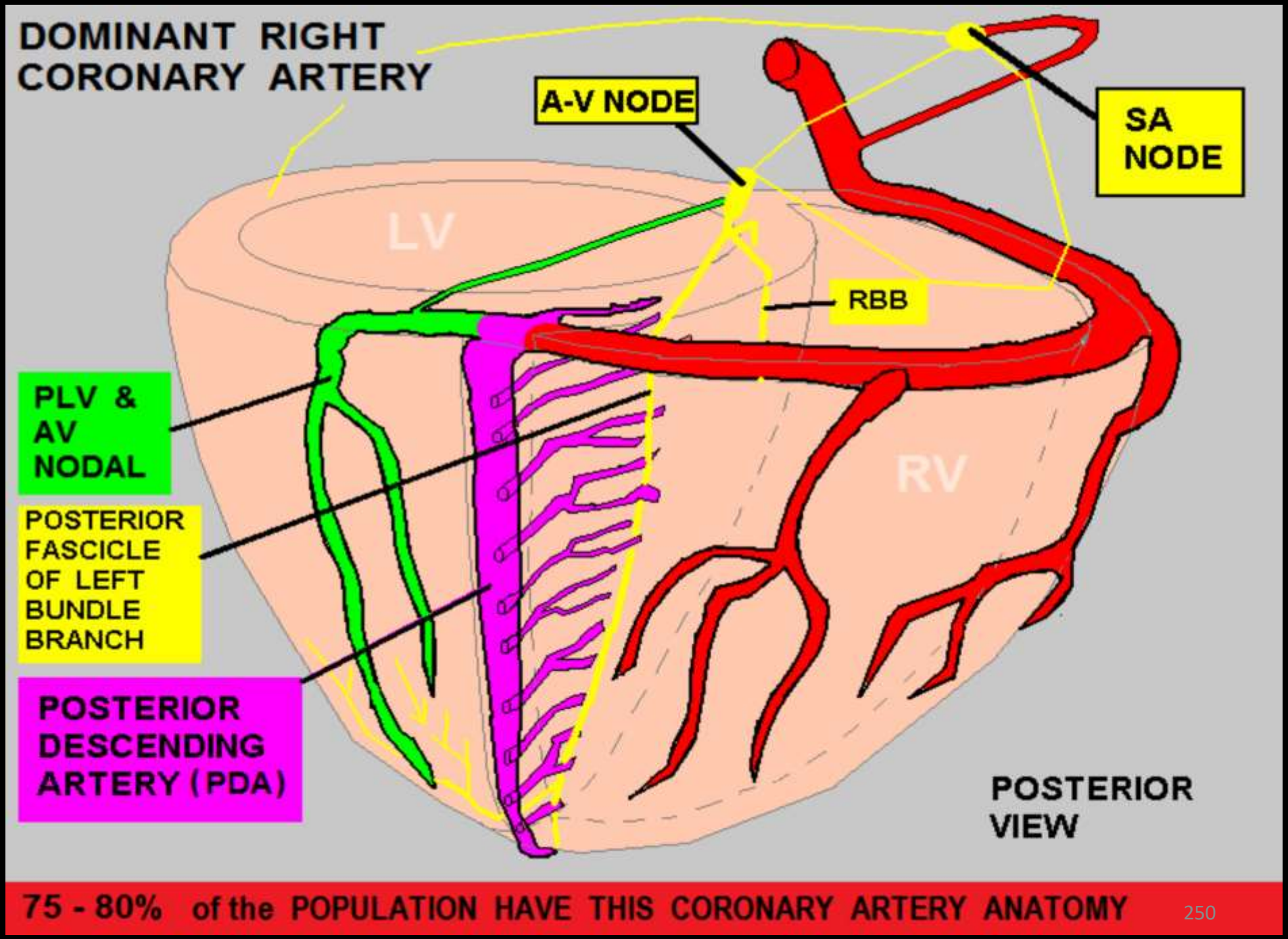


# LEADS II, III, and aVF VIEW
## INFERIOR WALL of the LEFT VENTRICLE






Which CORONARY ARTERY usually supplies the INFERIOR WALL?

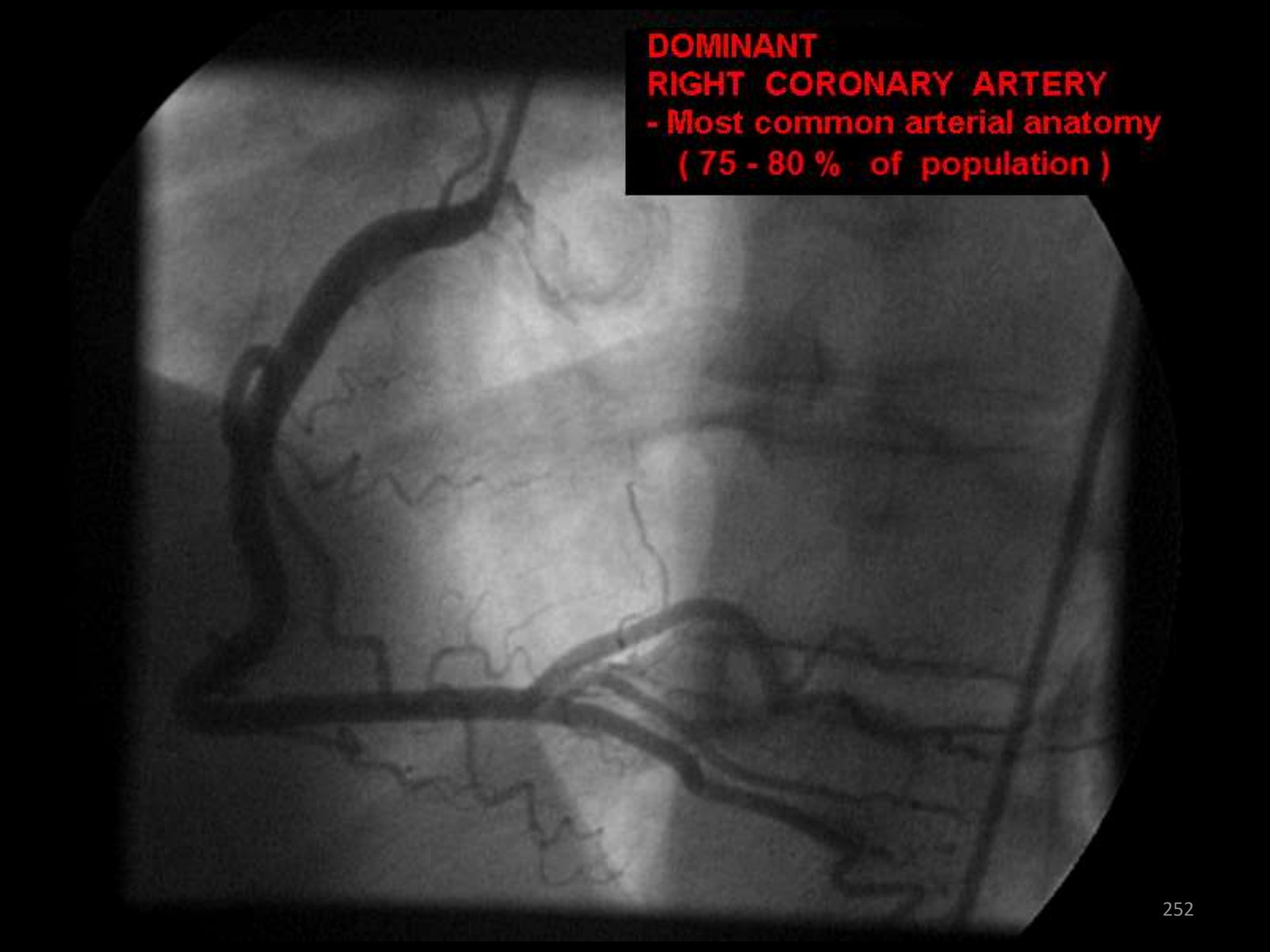

DOMINANT RIGHT CORONARY ARTERY
A-V NODE
SA NODE
LV
RBB
RV
PLV & AV NODAL
POSTERIOR FASCICLE OF LEFT BUNDLE BRANCH
POSTERIOR DESCENDING ARTERY (PDA)
POSTERIOR VIEW
75 - 80% of the POPULATION HAVE THIS CORONARY ARTERY ANATOMY

HELPFUL HINT . . . MEMORIZE THIS !

# RIGHT CORONARY ARTERY ( RCA )

RIGHT DOMINANT SYSTEMS

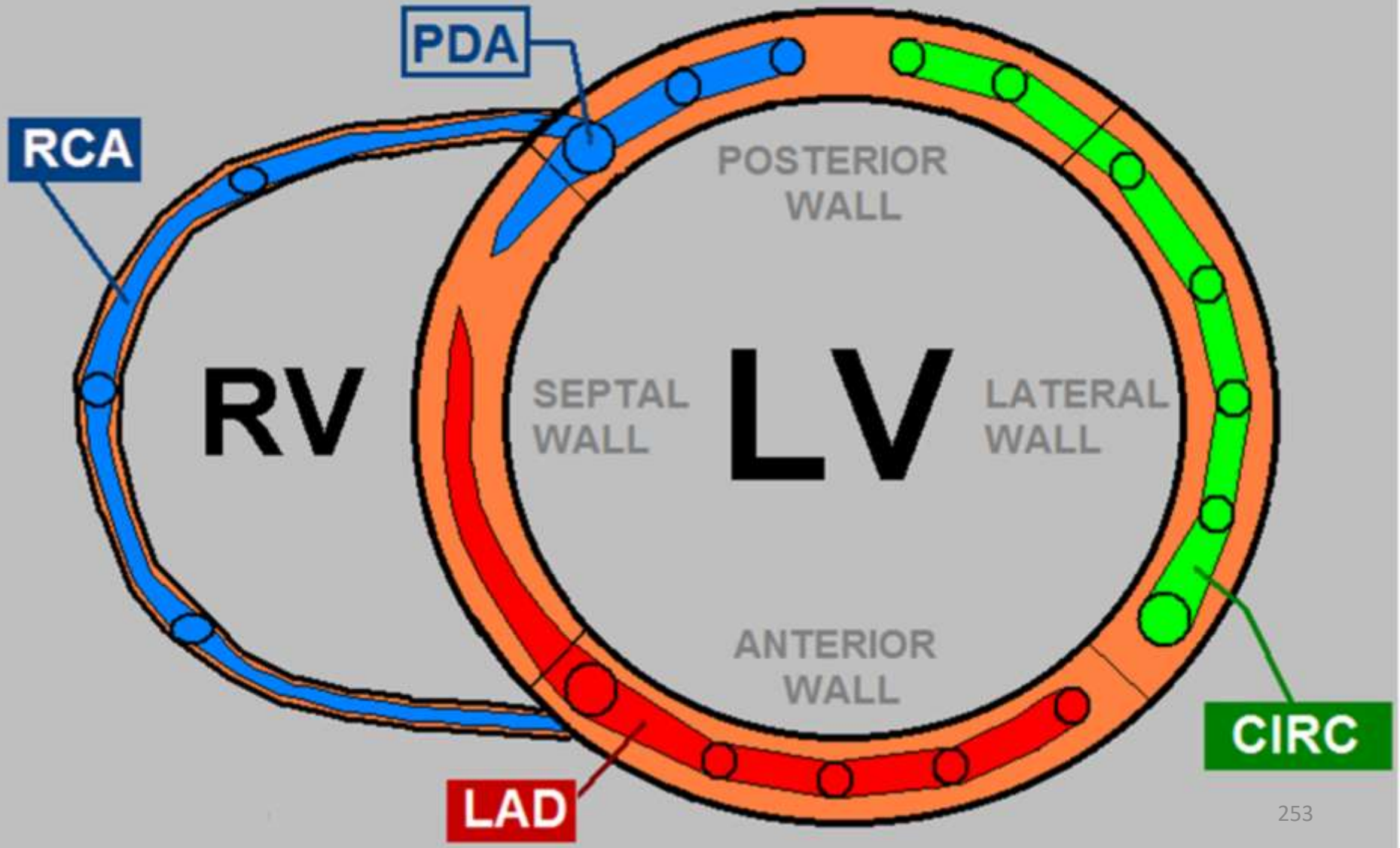
- RIGHT ATRIUM
- SINUS NODE ( 55% of the population )
- RIGHT VENTRICLE - 100 % of muscle mass
- LEFT VENTRICLE: 15 - 25 % of muscle mass
  - INFERIOR WALL
  - approx. 1/2 of POSTERIOR WALL
- AV NODE


DOMINANT
RIGHT CORONARY ARTERY
- Most common arterial anatomy
( 75 - 80 % of population )

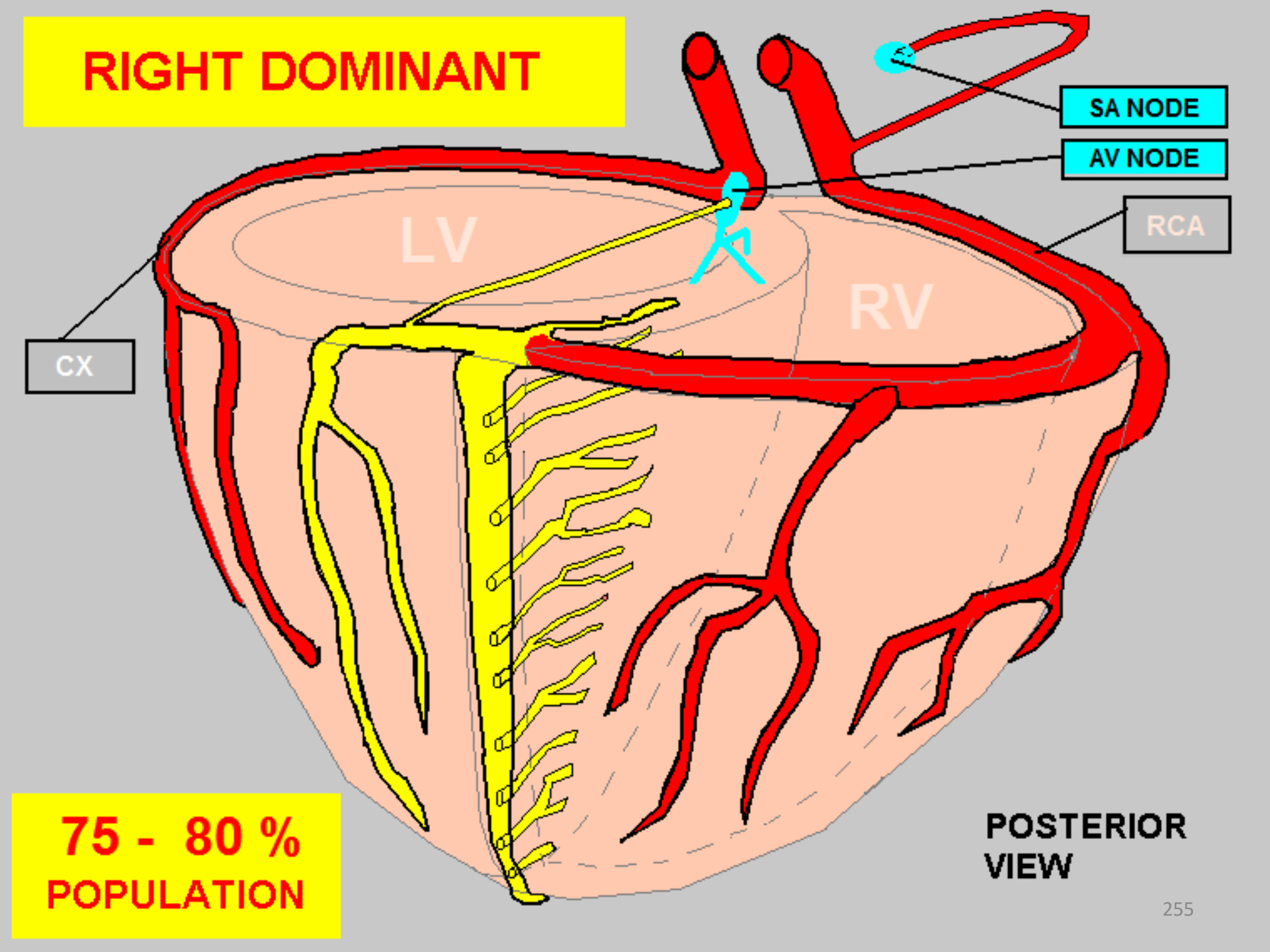
# ARTERIAL DISTRIBUTION - MYOCARDIUM

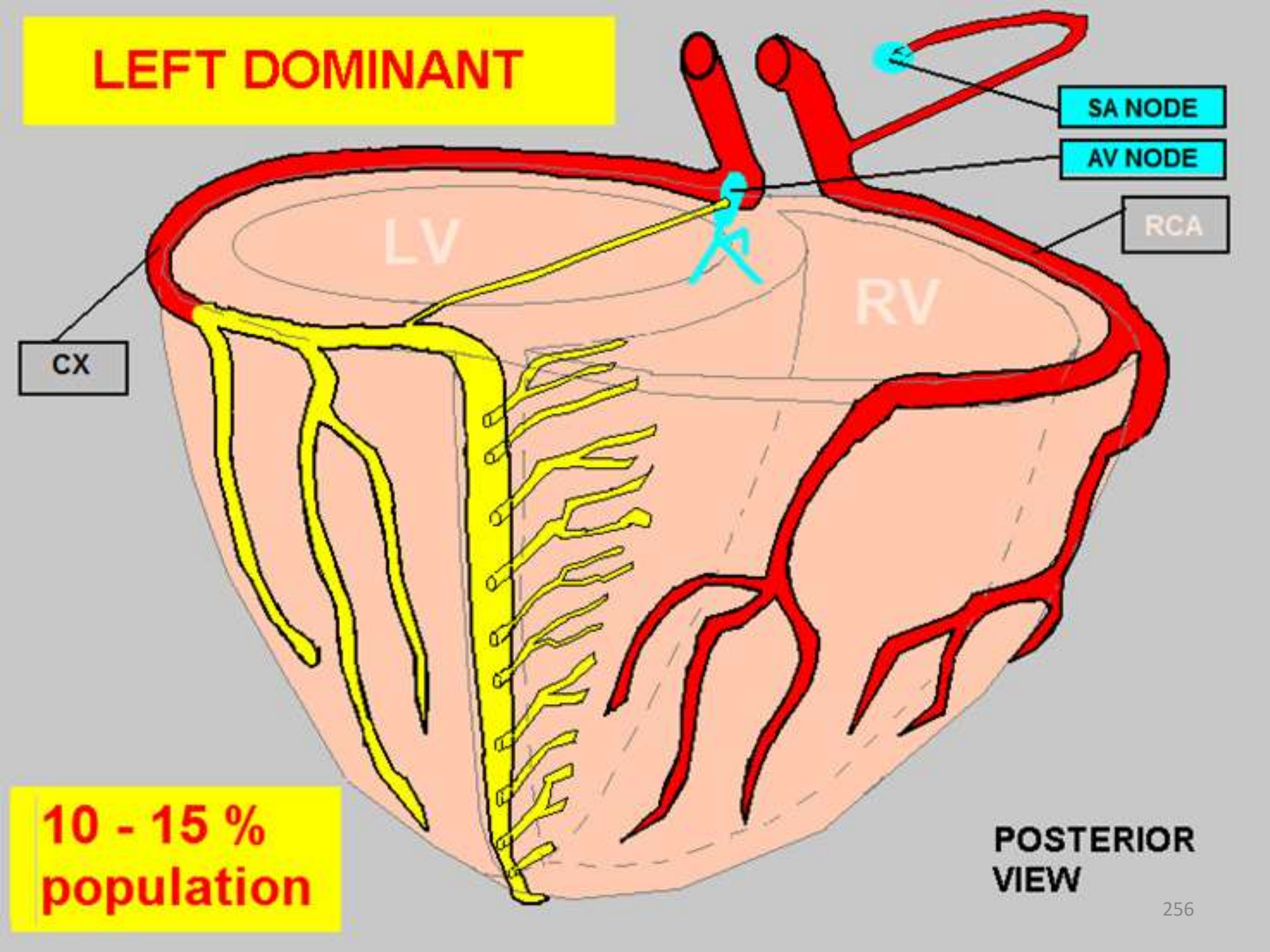
## DOMINANT RCA

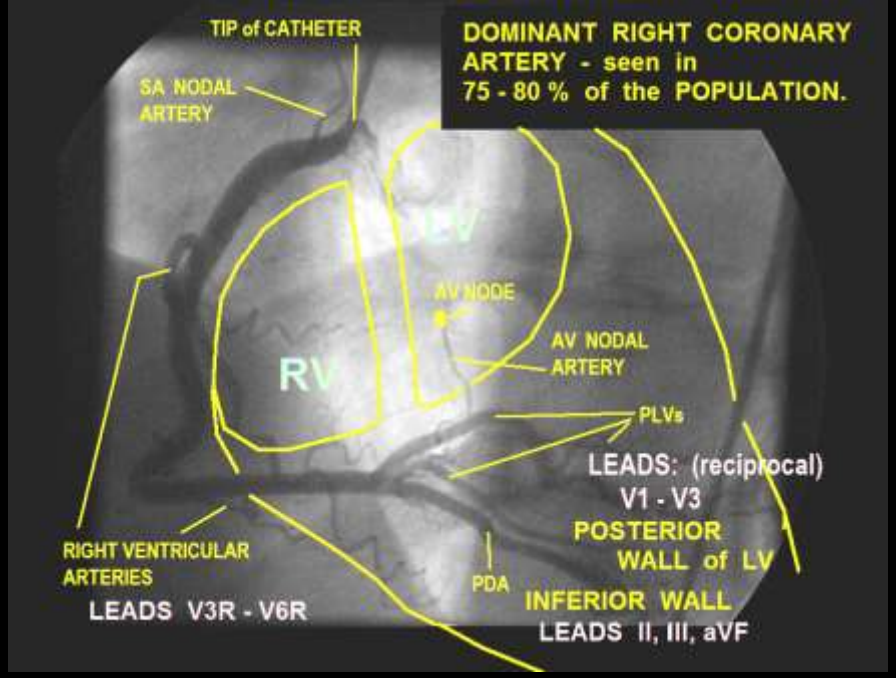
75-80 % of POPULATION

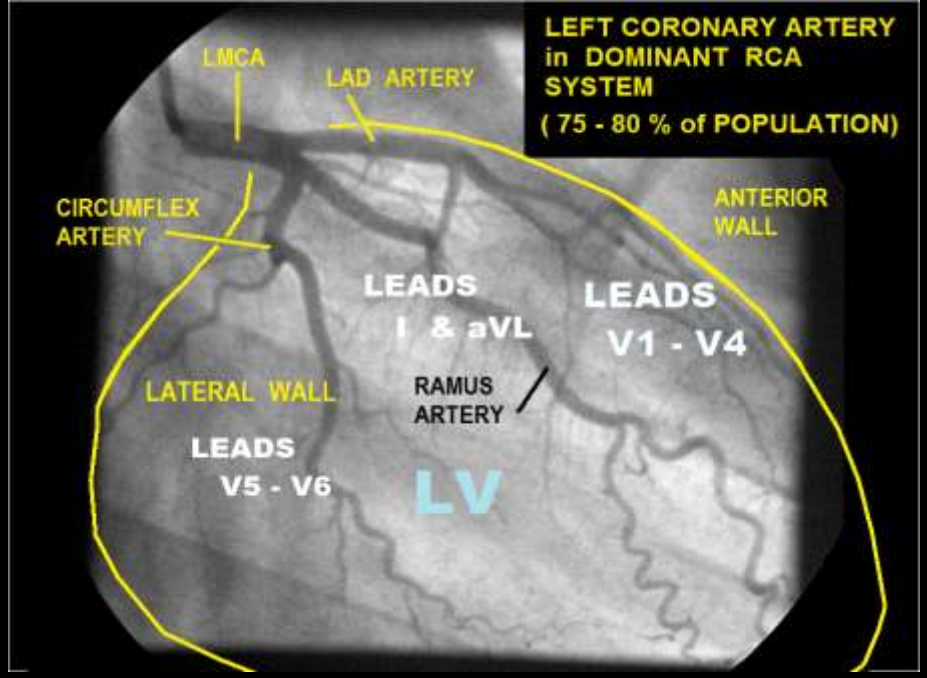


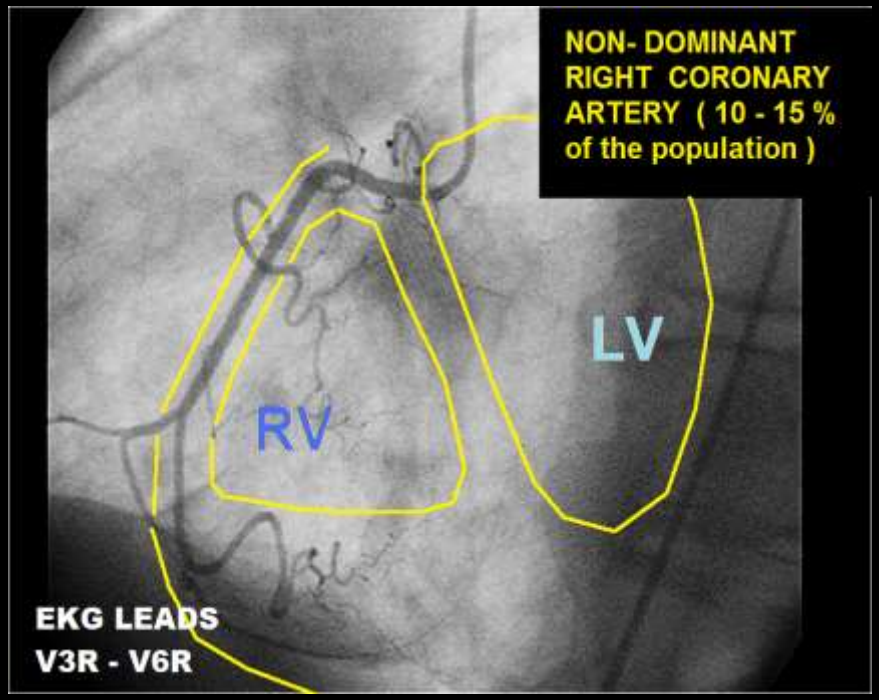
**So if the Right Coronary Artery Is DOMINANT in 75 – 80% of the POPULATION, what accounts for the Other 20 – 25% ??**

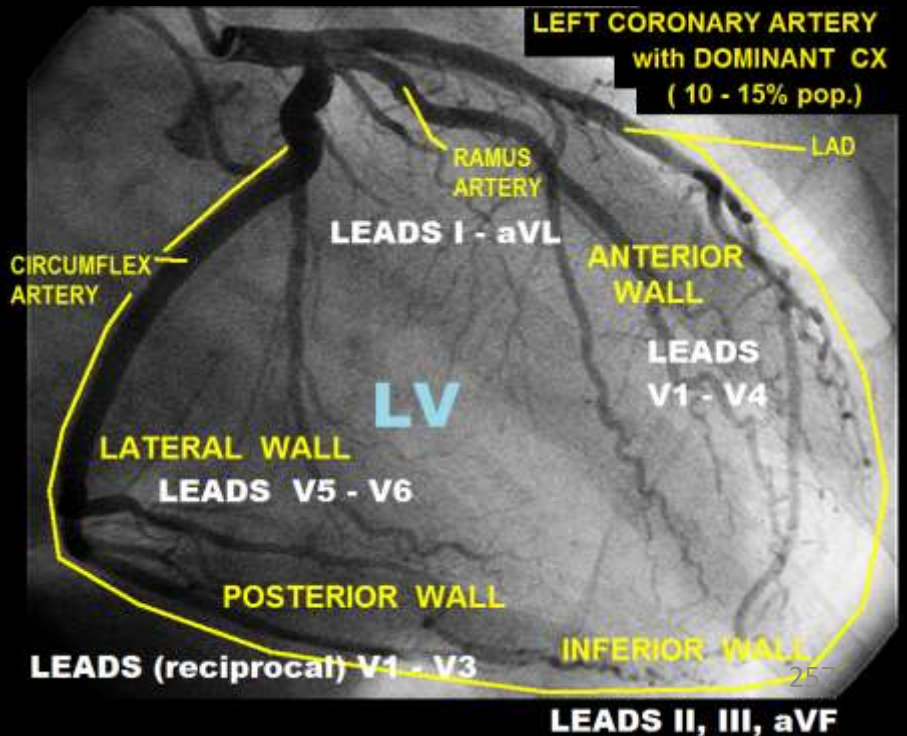

RIGHT DOMINANT
SA NODE
AV NODE
RCA
LV
RV
CX
75 - 80 %
POPULATION
POSTERIOR
VIEW

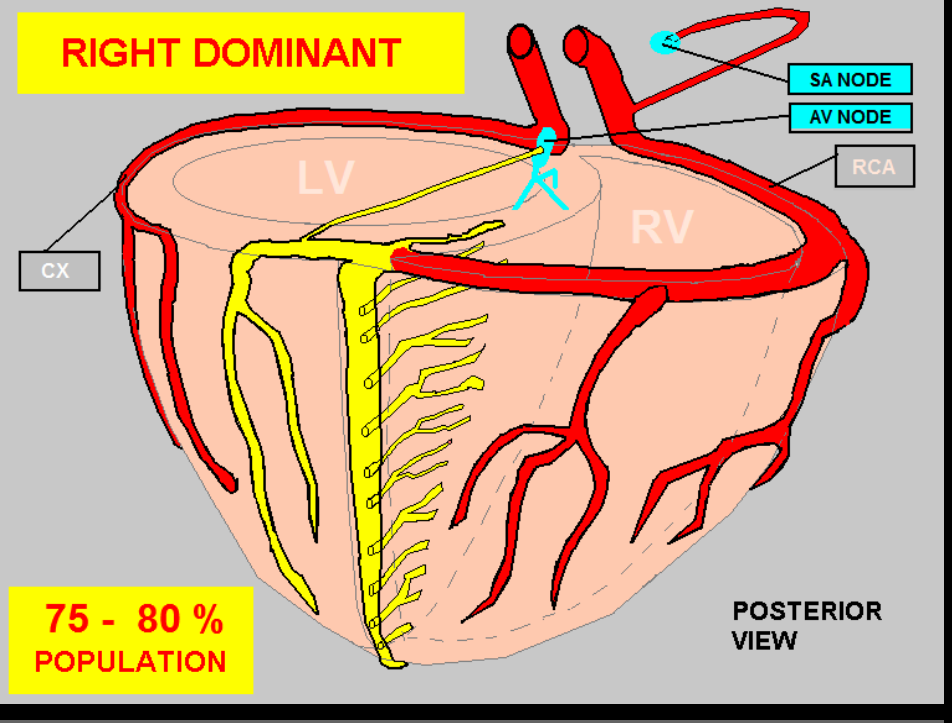

LEFT DOMINANT
SA NODE
AV NODE
RCA
LV
RV
CX
10 - 15 %
population
POSTERIOR
VIEW

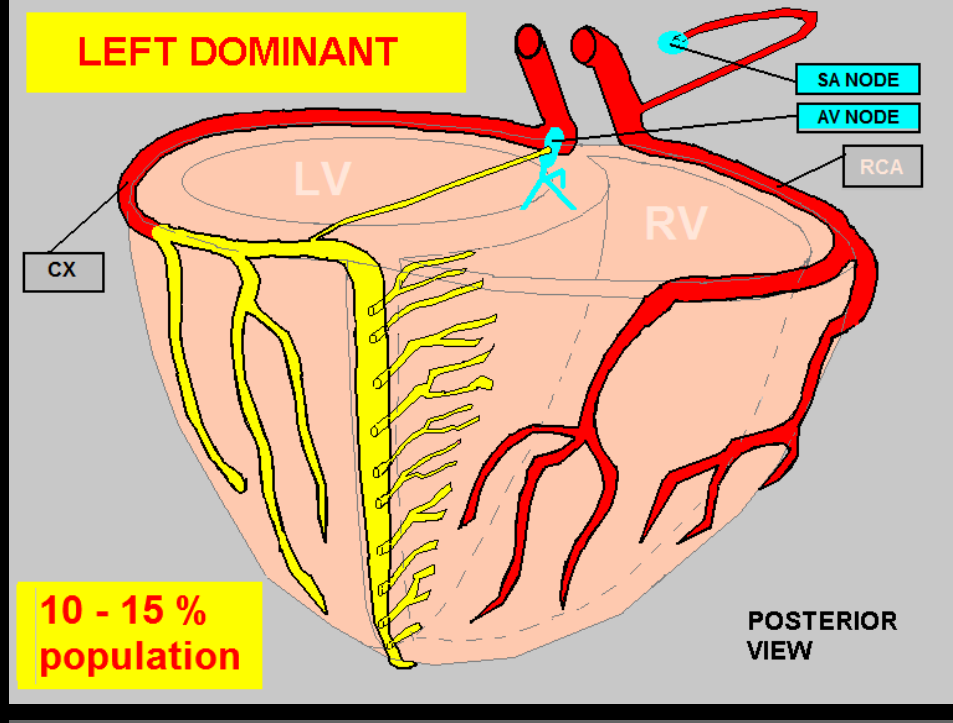

TIP of CATHETER
SA NODAL ARTERY
DOMINANT RIGHT CORONARY ARTERY - seen in 75 - 80 % of the POPULATION.
AV NODE
AV NODAL ARTERY
RV
PLVs
LEADS: (reciprocal) V1 - V3
POSTERIOR WALL of LV
RIGHT VENTRICULAR ARTERIES
PDA
INFERIOR WALL
LEADS II, III, aVF
LEADS V3R - V6R

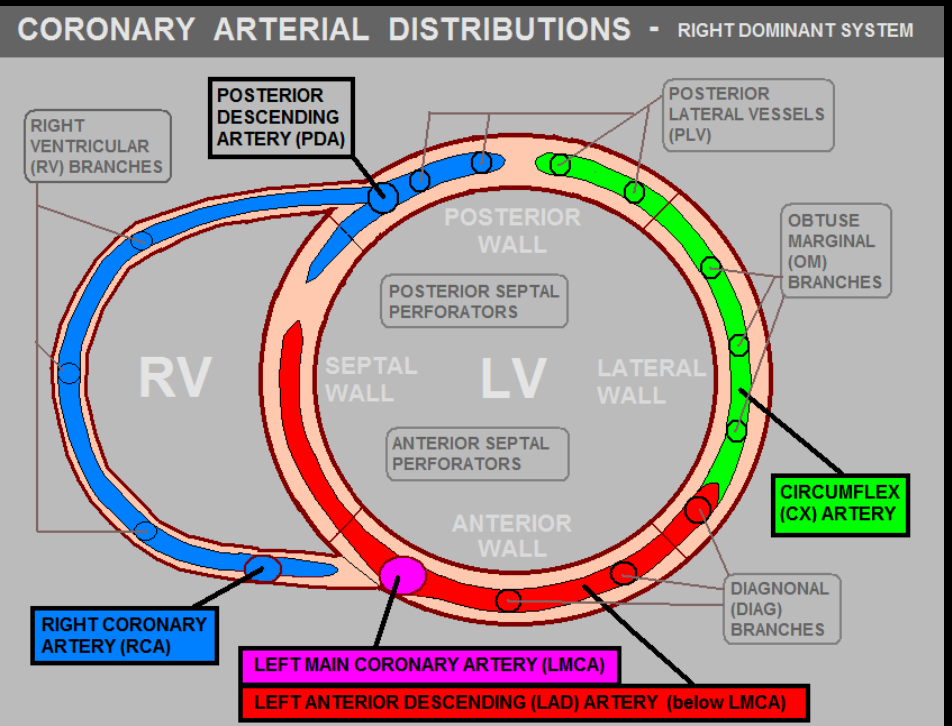

LEFT CORONARY ARTERY in DOMINANT RCA SYSTEM
( 75 - 80 % of POPULATION)
LMCA
LAD ARTERY
CIRCUMFLEX ARTERY
ANTERIOR WALL
LEADS I & aVL
LEADS V1 - V4
LATERAL WALL
RAMUS ARTERY
LEADS V5 - V6
LV

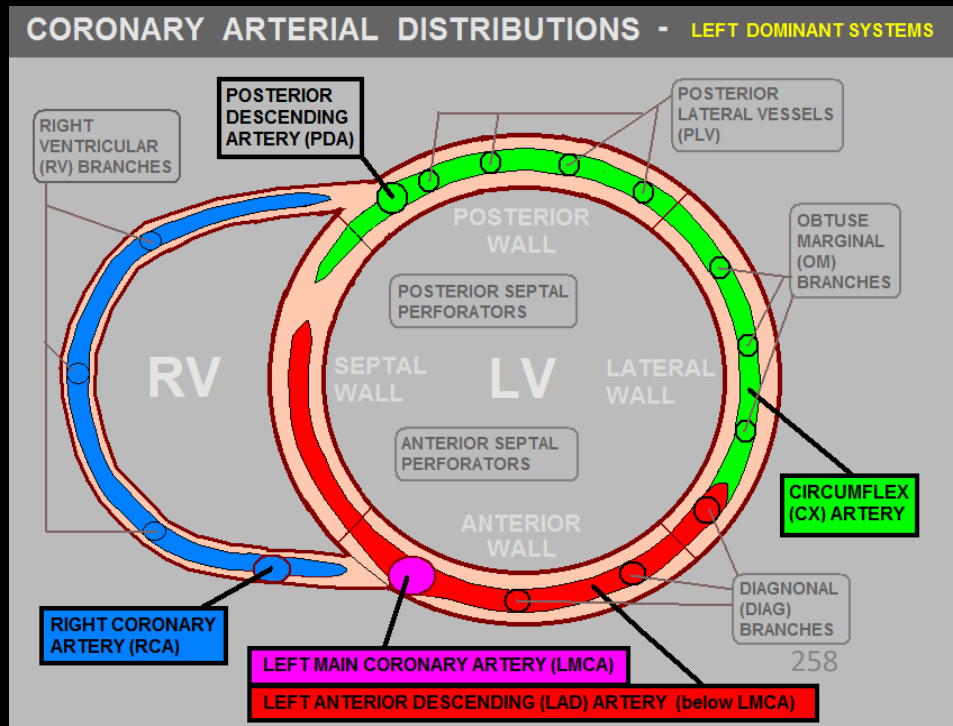

NON- DOMINANT RIGHT CORONARY ARTERY ( 10 - 15 % of the population )
LV
RV
EKG LEADS V3R - V6R


LEFT CORONARY ARTERY with DOMINANT CX ( 10 - 15% pop.)
RAMUS ARTERY
LAD
LEADS I - aVL
CIRCUMFLEX ARTERY
ANTERIOR WALL
LEADS V1 - V4
LV
LATERAL WALL
LEADS V5 - V6
POSTERIOR WALL
INFERIOR WALL
LEADS (reciprocal) V1 - V3
LEADS II, III, aVF


RIGHT DOMINANT
SA NODE
AV NODE
RCA
CX
LV
RV
75 - 80 %
POPULATION
POSTERIOR
VIEW


LEFT DOMINANT
SA NODE
AV NODE
RCA
CX
LV
RV
10 - 15 %
population
POSTERIOR
VIEW


CORONARY ARTERIAL DISTRIBUTIONS - RIGHT DOMINANT SYSTEM
POSTERIOR DESCENDING ARTERY (PDA)
POSTERIOR LATERAL VESSELS (PLV)
RIGHT VENTRICULAR (RV) BRANCHES
OBTUSE MARGINAL (OM) BRANCHES
POSTERIOR WALL
POSTERIOR SEPTAL PERFORATORS
RV
SEPTAL WALL
LV
LATERAL WALL
ANTERIOR SEPTAL PERFORATORS
CIRCUMFLEX (CX) ARTERY
ANTERIOR WALL
DIAGNONAL (DIAG) BRANCHES
RIGHT CORONARY ARTERY (RCA)
LEFT MAIN CORONARY ARTERY (LMCA)
LEFT ANTERIOR DESCENDING (LAD) ARTERY (below LMCA)


CORONARY ARTERIAL DISTRIBUTIONS - LEFT DOMINANT SYSTEMS
POSTERIOR DESCENDING ARTERY (PDA)
POSTERIOR LATERAL VESSELS (PLV)
RIGHT VENTRICULAR (RV) BRANCHES
OBTUSE MARGINAL (OM) BRANCHES
POSTERIOR WALL
POSTERIOR SEPTAL PERFORATORS
RV
SEPTAL WALL
LV
LATERAL WALL
ANTERIOR SEPTAL PERFORATORS
CIRCUMFLEX (CX) ARTERY
ANTERIOR WALL
DIAGNONAL (DIAG) BRANCHES
RIGHT CORONARY ARTERY (RCA)
LEFT MAIN CORONARY ARTERY (LMCA)
LEFT ANTERIOR DESCENDING (LAD) ARTERY (below LMCA)

Pat ID [redacted]
01/20/2021 07:46:46
08/17/1955 65 yrs
Account # [redacted]
RX
DX

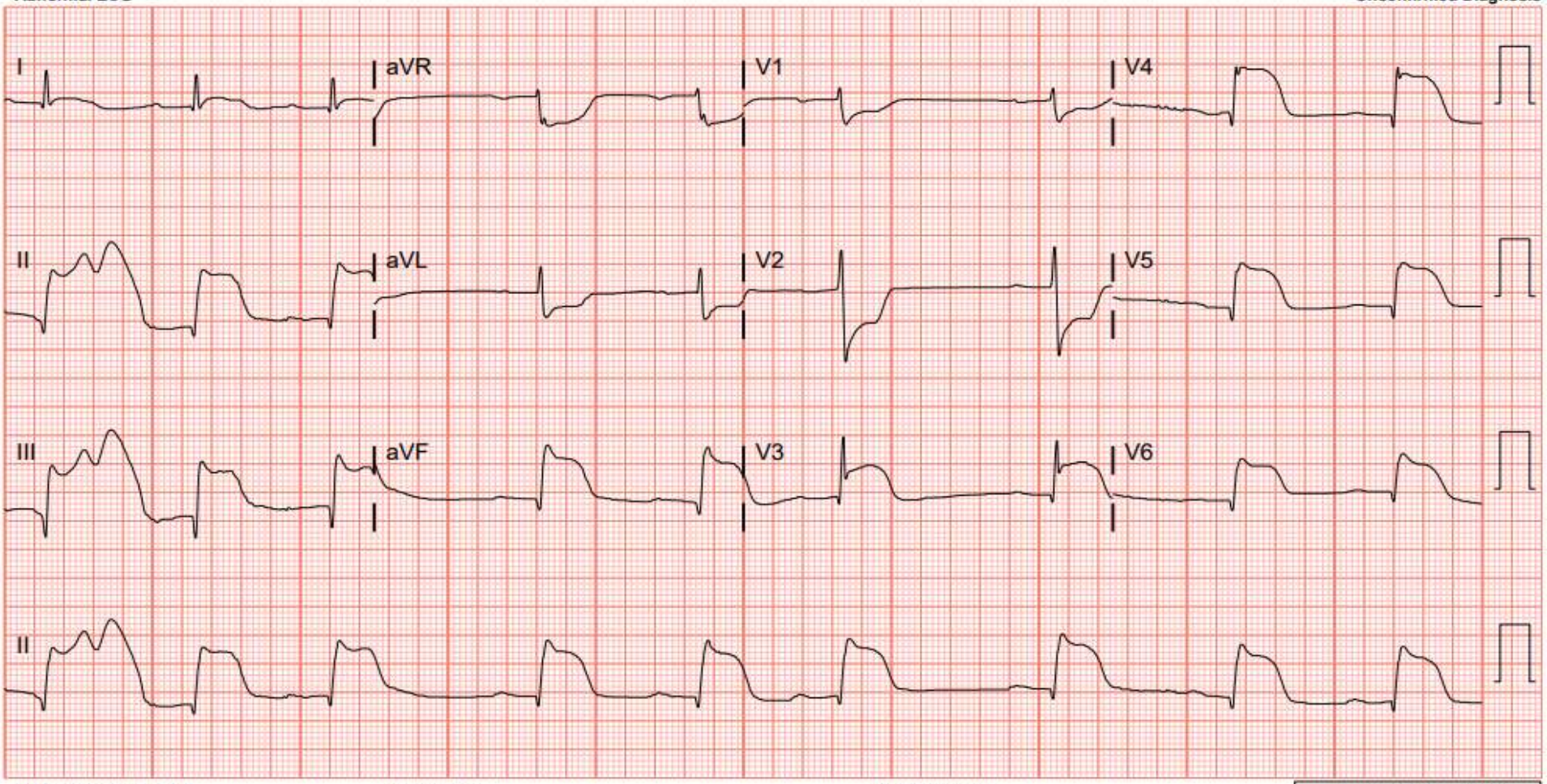
Bayfront Health Seven Rivers ED
Dept ED
Room EDWR
Tech mg

| | | |
|---|---|---|
| Rate | 54 | Sinus or ectopic atrial rhythm |
| PR | 329 | Atrial premature complex |
| QRSd | 139 | Prolonged PR interval |
| QT | 437 | Nonspecific intraventricular conduction delay |
| QTc | 415 | Inferoposterior infarct, acute (LCx) |
| --Axis-- | | Anterolateral infarct, acute |
| P | -83 | Baseline wander in lead(s) V3,V4 |
| QRS | 80 | NO PREVIOUS ECG AVAILABLE FOR COMPARISON |
| T | 77 | |

Req Provider: Xandus Chen

**- Abnormal ECG -**

**Unconfirmed Diagnosis**




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

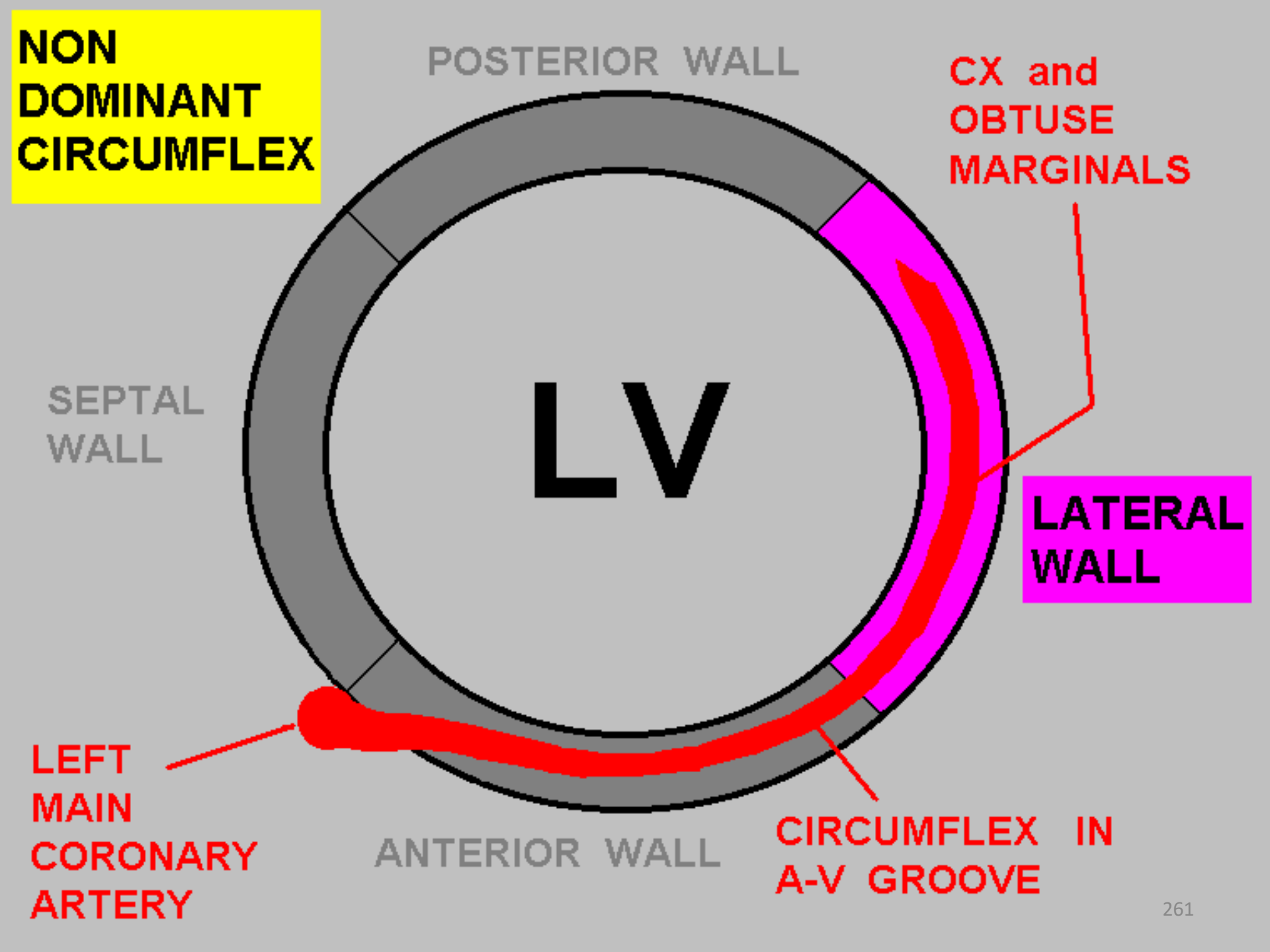
# CIRCUMFLEX ARTERY ( CX )

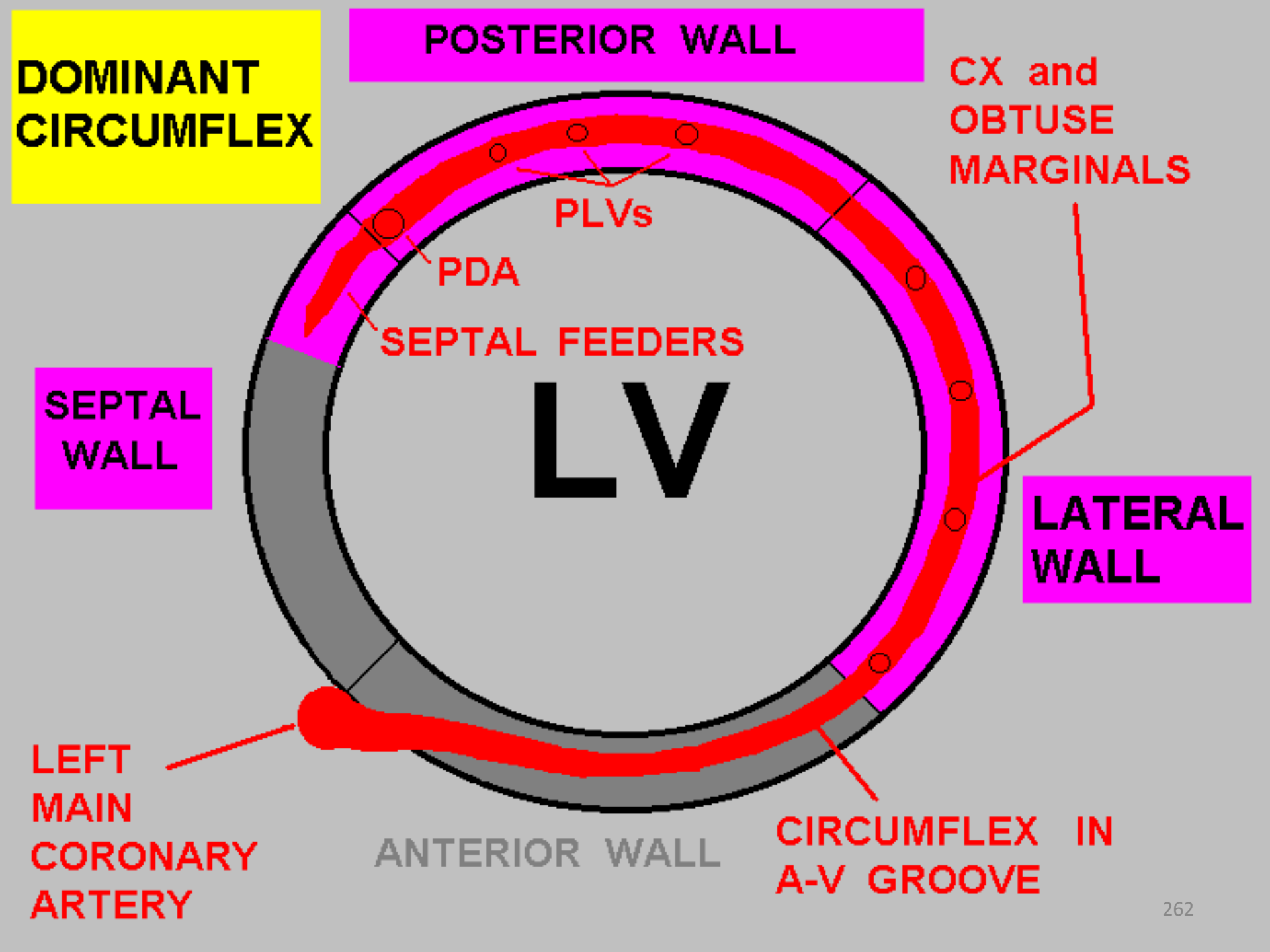
- NON-DOMINANT CX:

CX = 15 - 30% OF LV MASS

- DOMINANT CX:

| | | |
|---|---|---|
| CX | = | 15 - 30% OF LV MASS |
| + PDA | = | 15 - 25% OF LV MASS |
| TOTAL | | 30 - 55% OF LV MASS |

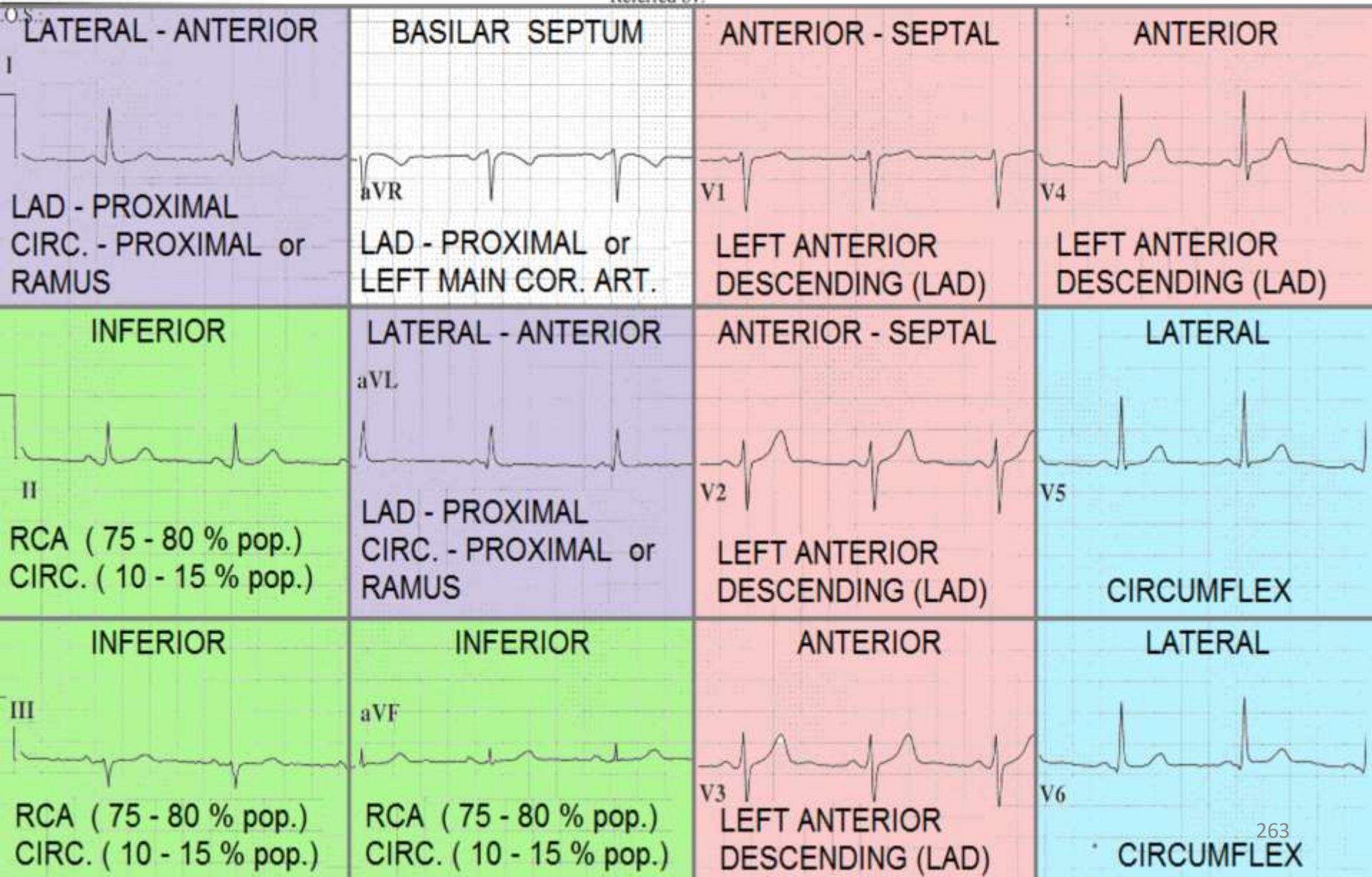

NON DOMINANT CIRCUMFLEX
POSTERIOR WALL
CX and OBTUSE MARGINALS
SEPTAL WALL
LV
LATERAL WALL
LEFT MAIN CORONARY ARTERY
ANTERIOR WALL
CIRCUMFLEX IN A-V GROOVE


DOMINANT CIRCUMFLEX
POSTERIOR WALL
CX and OBTUSE MARGINALS
PLVs
PDA
SEPTAL FEEDERS
LV
SEPTAL WALL
LATERAL WALL
LEFT MAIN CORONARY ARTERY
ANTERIOR WALL
CIRCUMFLEX IN A-V GROOVE

| | | |
|---|---|---|
| Vent. rate | 64 | BPM |
| PR interval | 130 | ms |
| QRS duration | 96 | ms |
| QT/QTc | 396/408 | ms |
| P–R–T axes | 40 11 | 61 |

Normal sinus rhythm
Normal ECG
No previous ECGs available

Referred by:

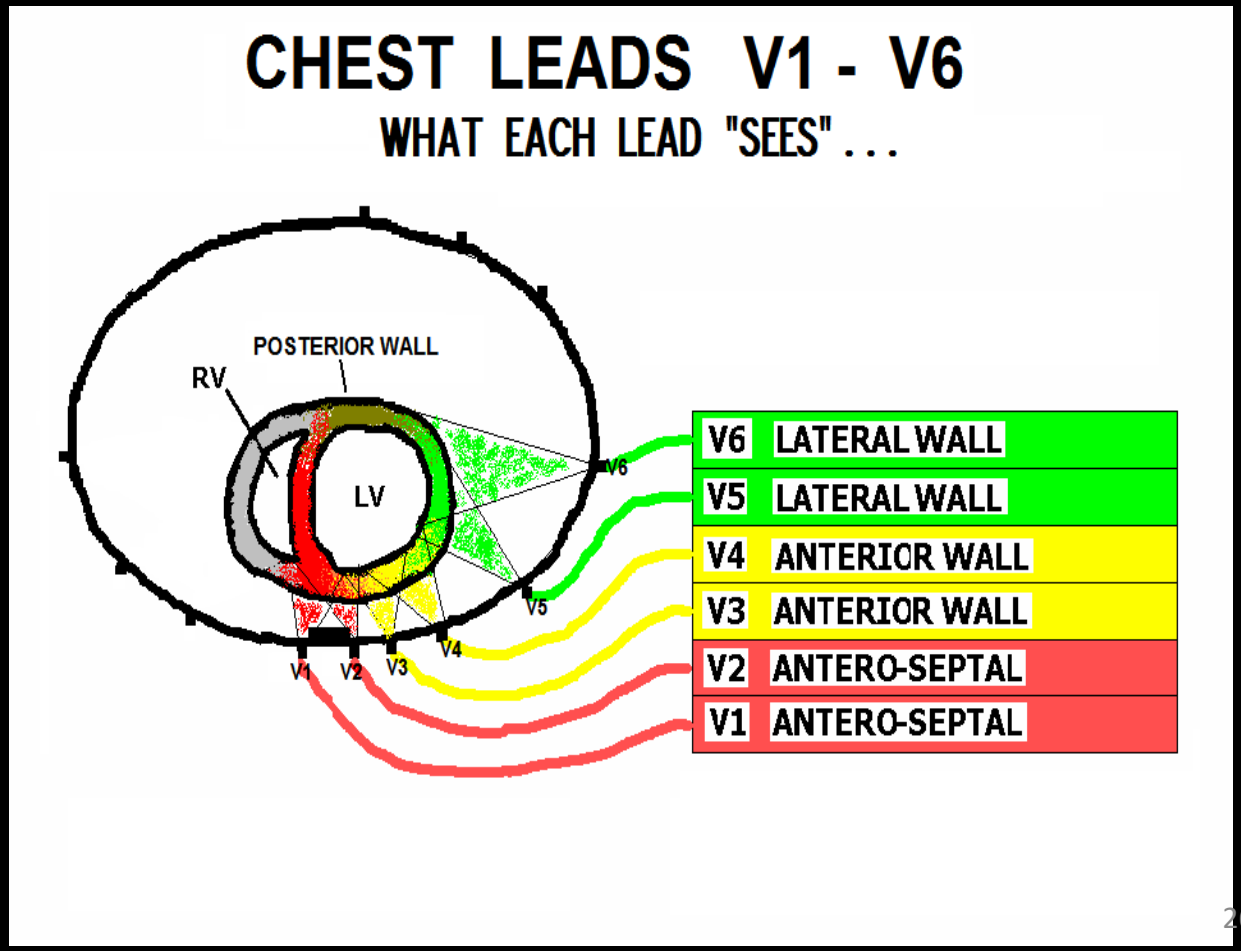


# The 12 Lead ECG
# Has TWO major BLIND SPOTS . . . . .

The POSTERIOR WALL

&

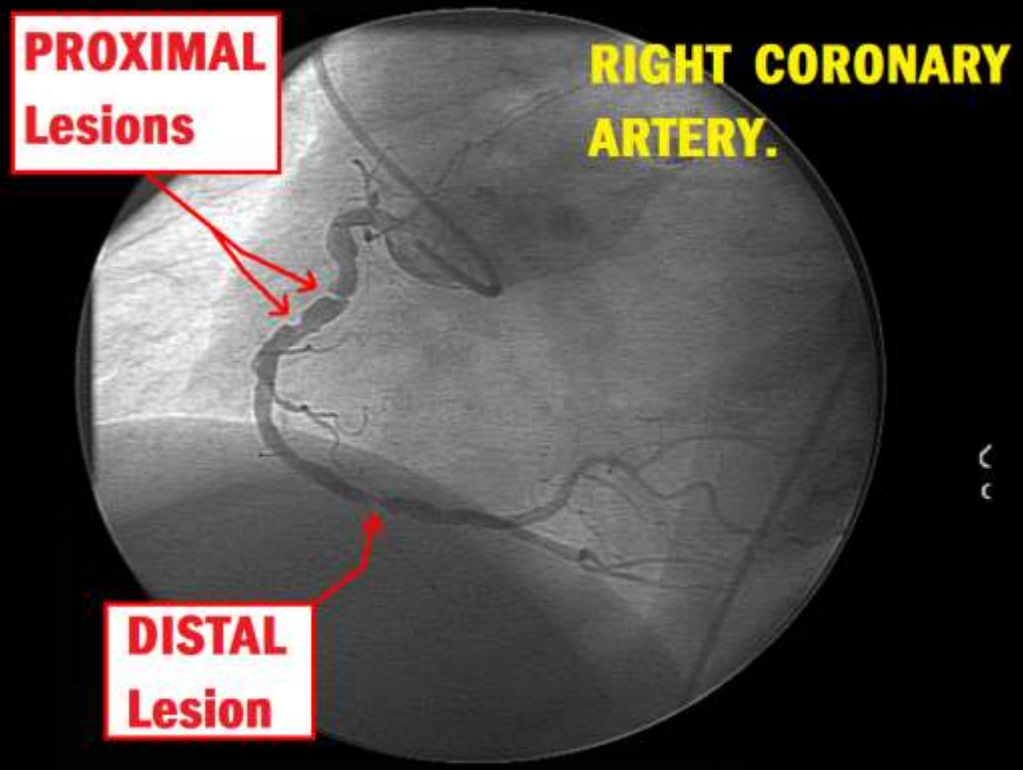
RIGHT VENTRICLE

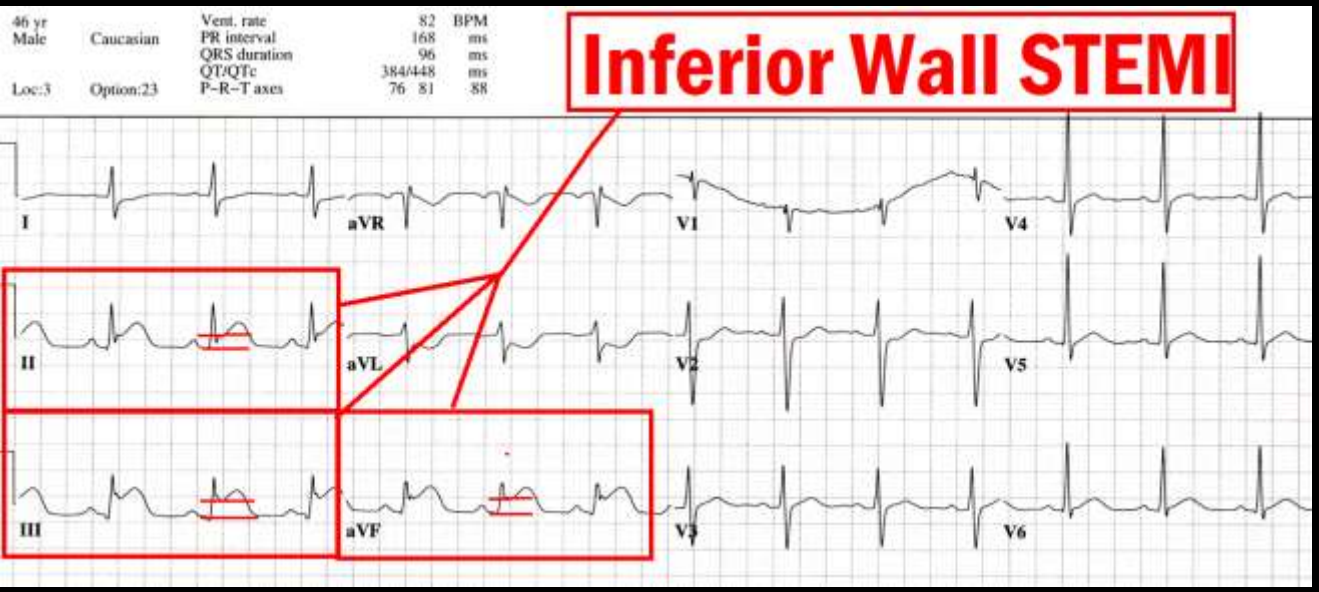


# When do we need to see the Right Ventricle?

- All Patient with INFERIOR WALL STEMI (ST Elevation in Leads II, III, aVF ).

When you see an EKG with **ST Elevation in Leads II, III and AVF** (Inferior Wall STEMI) – you cannot tell if the blockage is in the PROXIMAL RCA – or the DISTAL RCA.



To see the

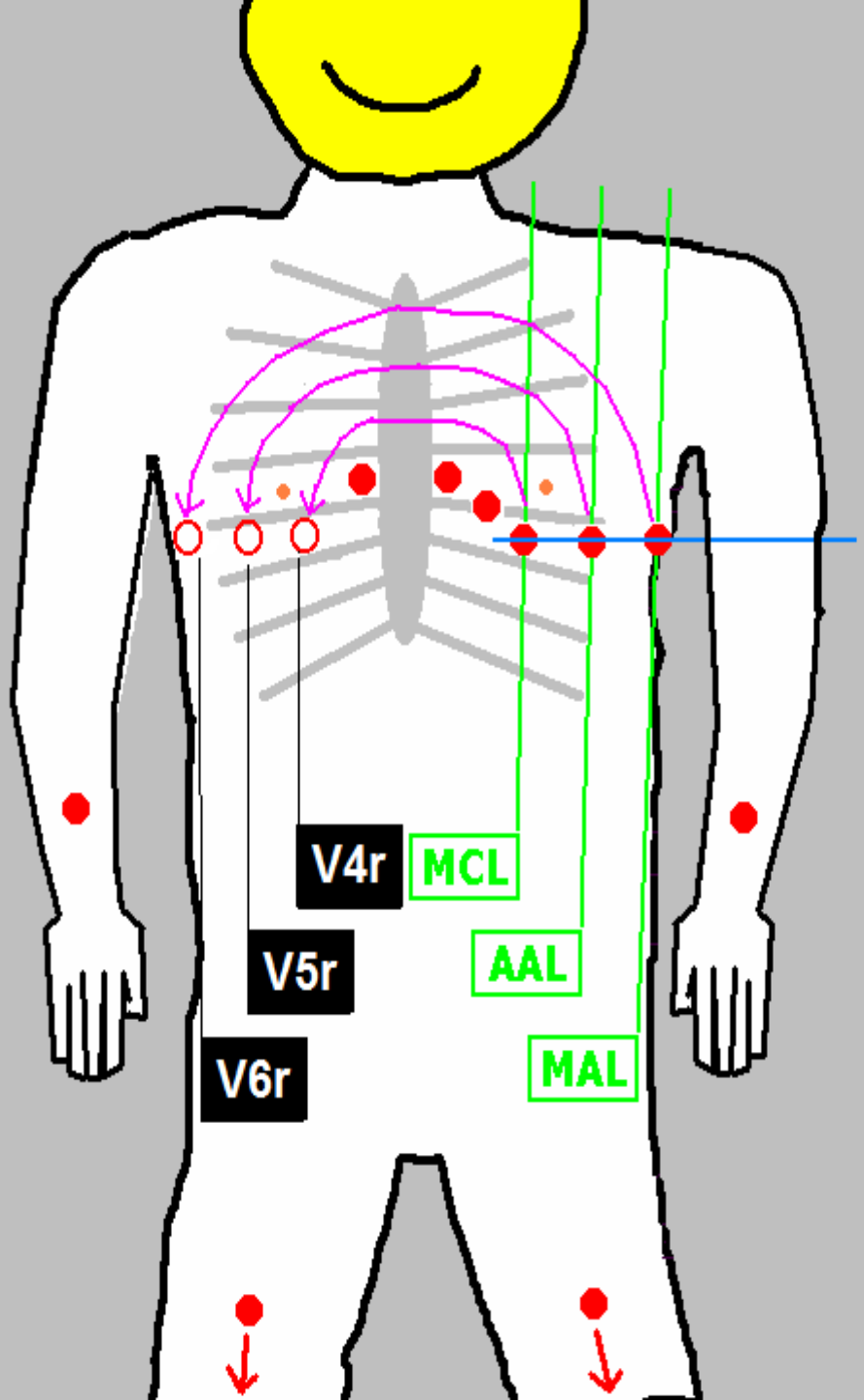
RIGHT VENTRICLE . . .

. . . such as in cases of

# INFERIOR WALL M.I.

☞ You must do a

# RIGHT - SIDED EKG ! !

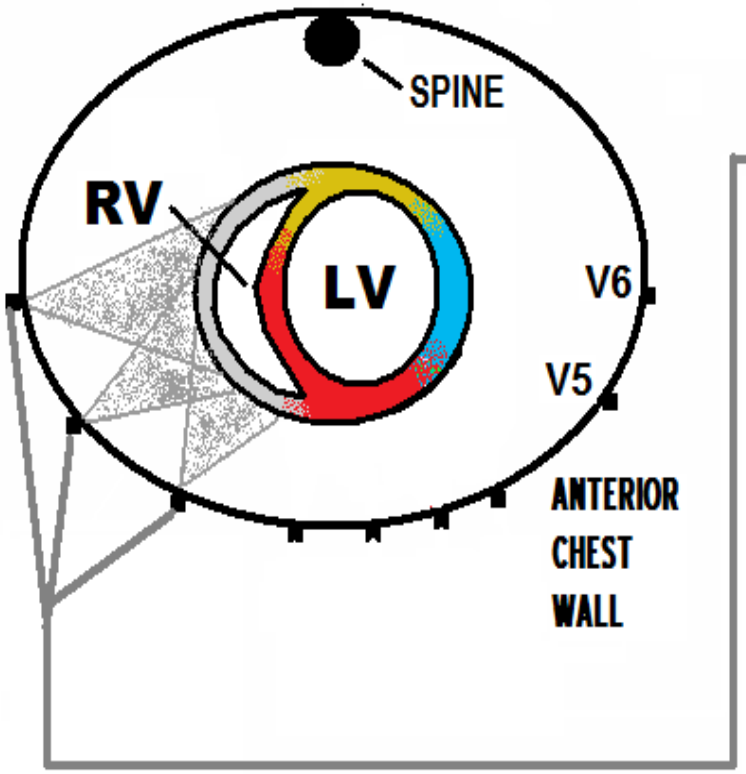


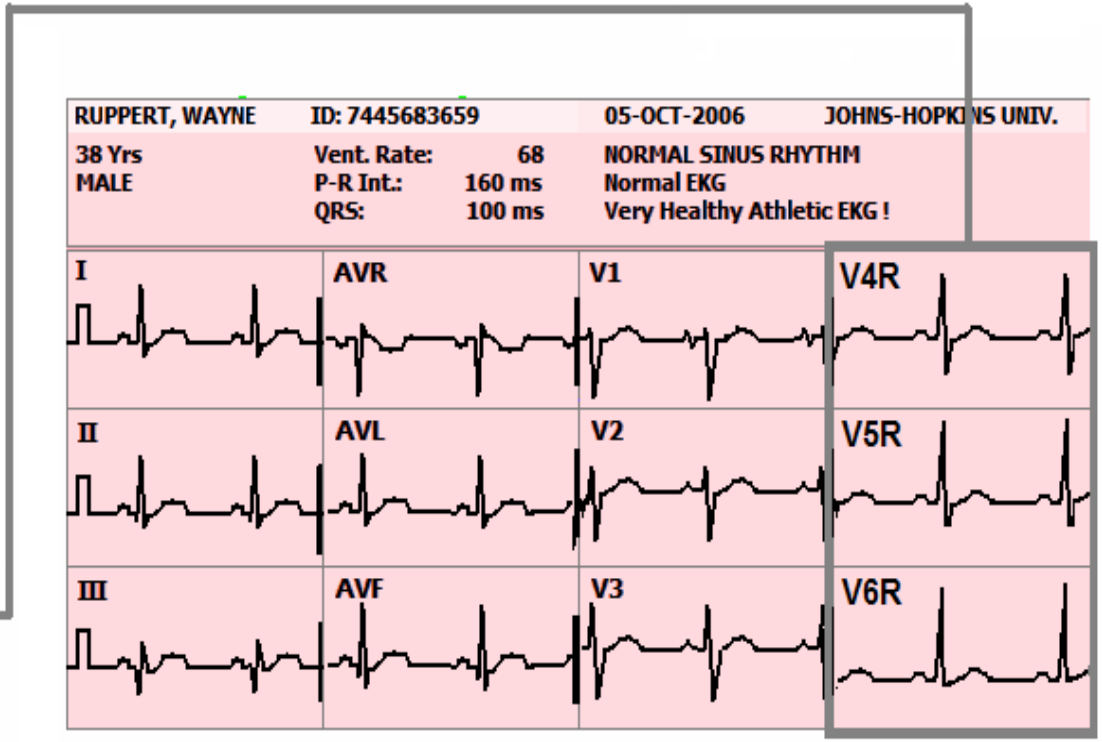

To do a
RIGHT - SIDED EKG . .

MOVE leads
V4, V5, and V6

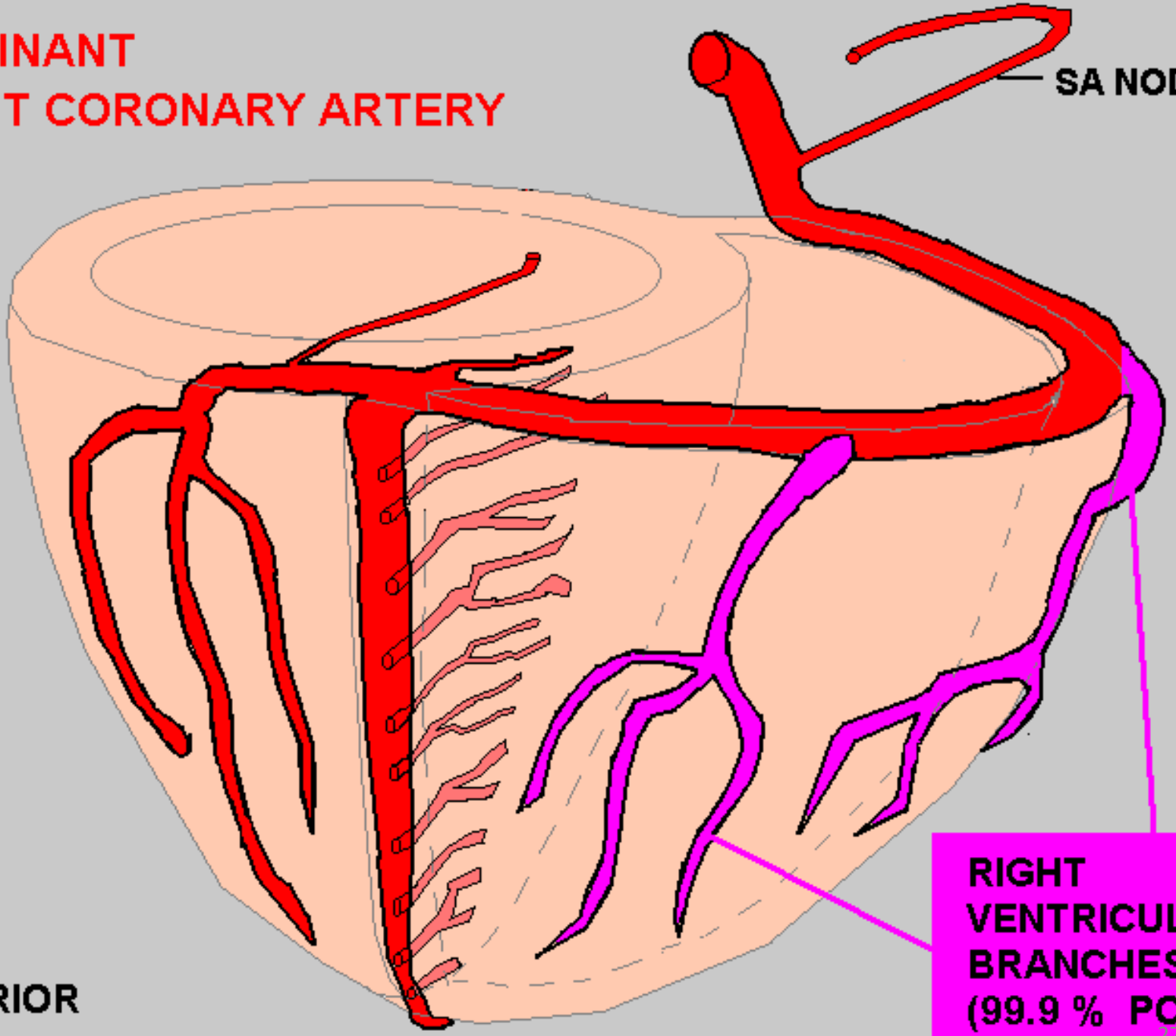
to the corresponding placement on the RIGHT SIDE of patient's chest . . .

# V4R - V6R VIEW THE RIGHT VENTRICLE



# DOMINANT RIGHT CORONARY ARTERY



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

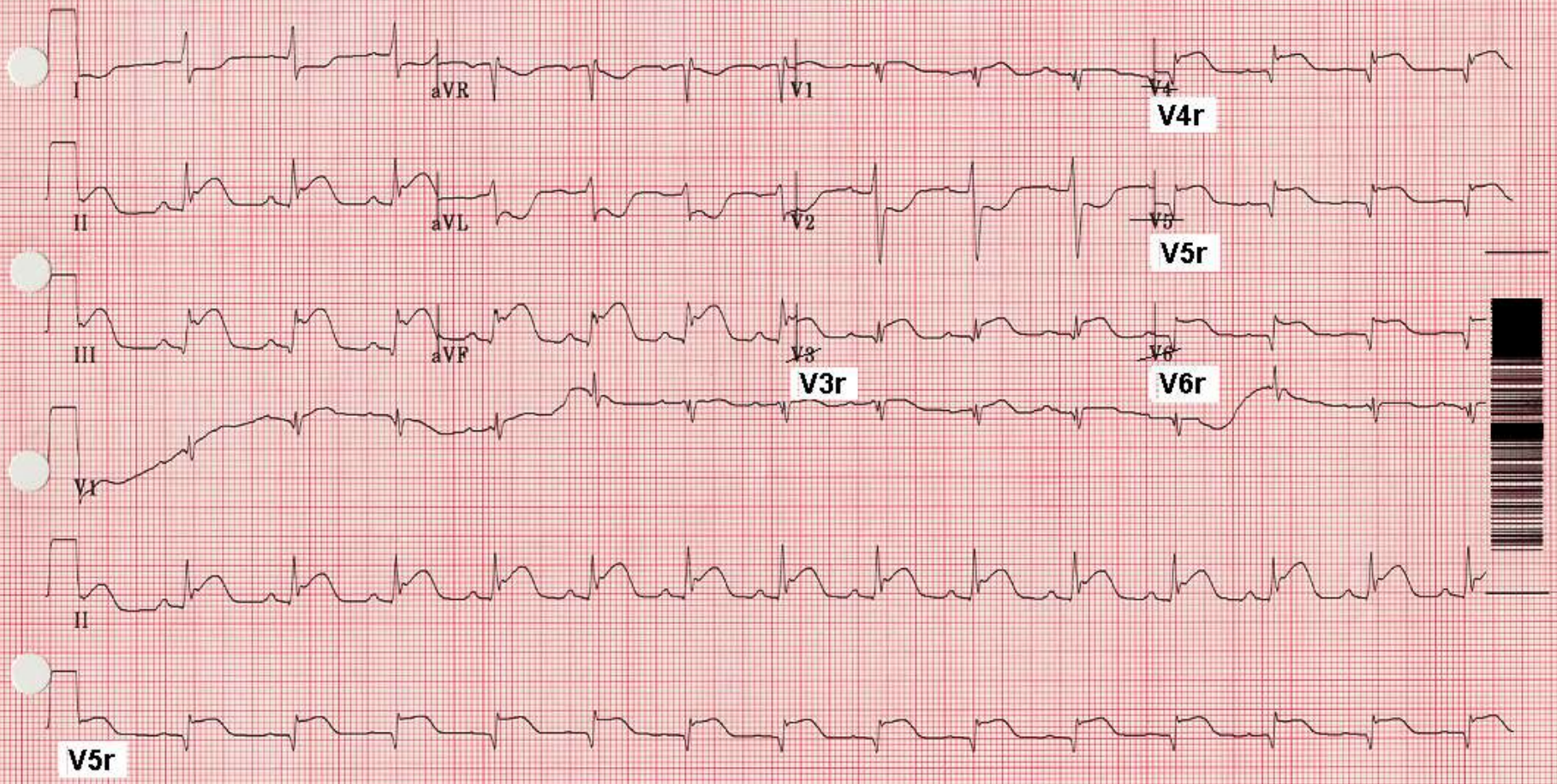
Normal sinus rhythm
~~Anterolateral infarct, possibly acute~~
Inferior injury pattern
** ** ** ** * Acute MI * ** ** ** **
Abnormal ECG

**Right Ventricular Infarct**

V LEADS
R SIDE

Referred by:

Unconfirmed




40 Hz 25.0 mm/s 10.0 mm/mV 4 by 2.5s + 3 rhythm lds MACVU 003C 12SL[tm] v250

# When do we need to see the Posterior Wall?

- Any time a patient presents with symptoms of ACS and the 12 Lead ECG shows ST Depression in Leads V1, V2, V3 and/or V4.

Whenever you see

# ST DEPRESSION in Leads V1 - V4

you must do a

# POSTERIOR LEAD ECG

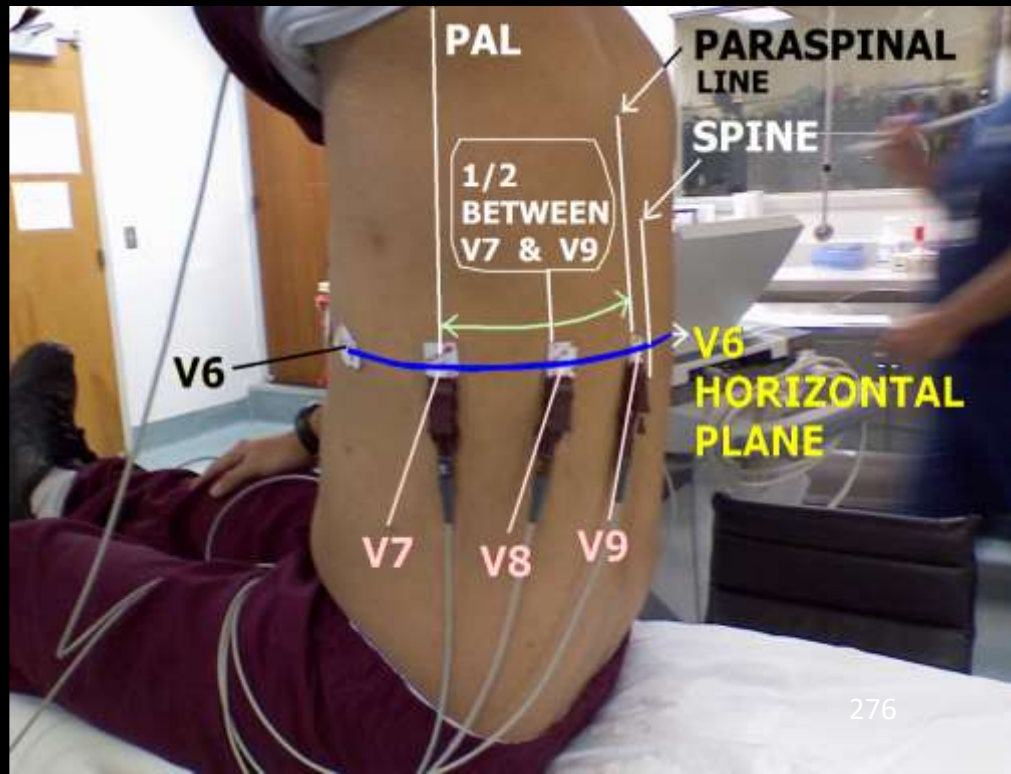
( V7 - V9 )

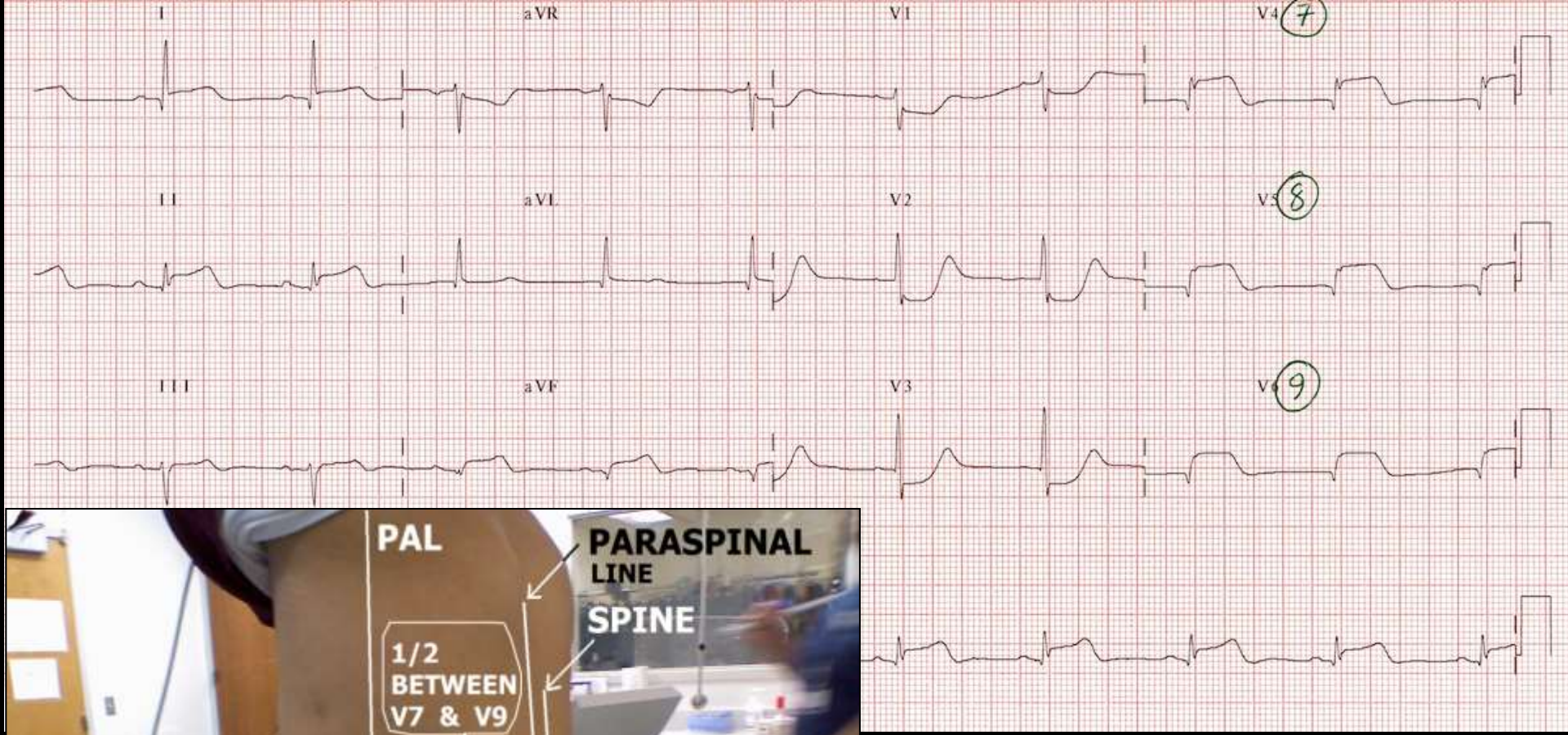
to see if you Patient is having a

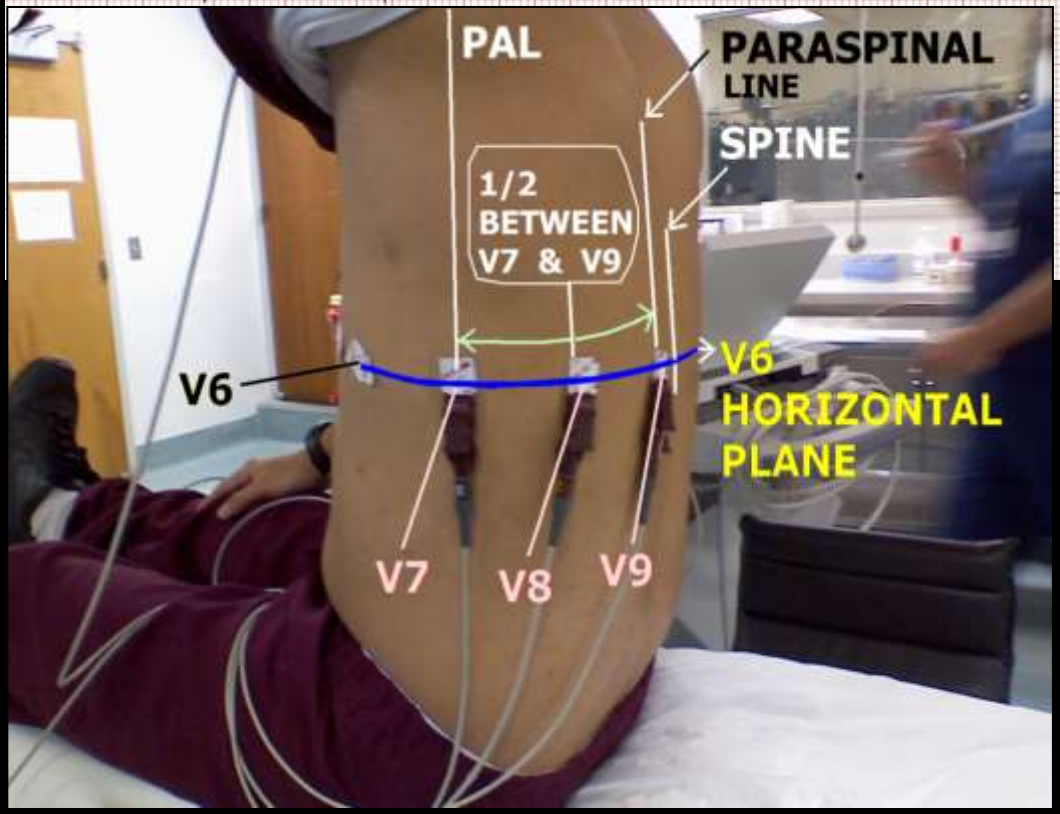
# POSTERIOR WALL STEMI

Whenever your patient's ECG exhibits ST DEPRESSION in any of the ANTERIOR LEADS (V1-V4), CONSIDER the possibility of POSTERIOR WALL STEMI ! !

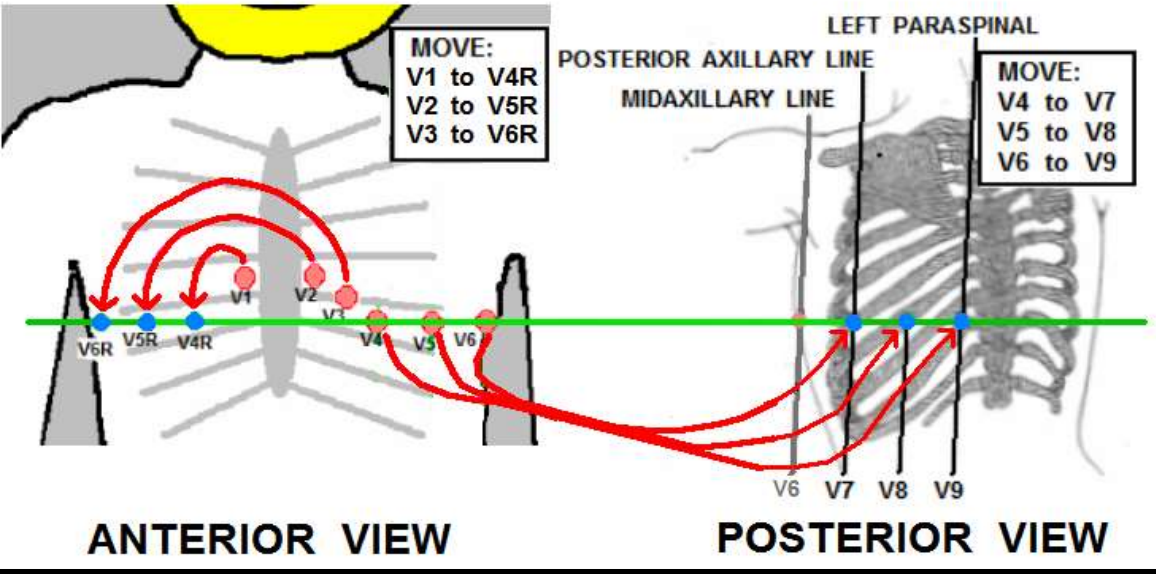
. . . To DIGANOSE Posterior Wall STEMI, we should see LEADS V7 – V9 !!

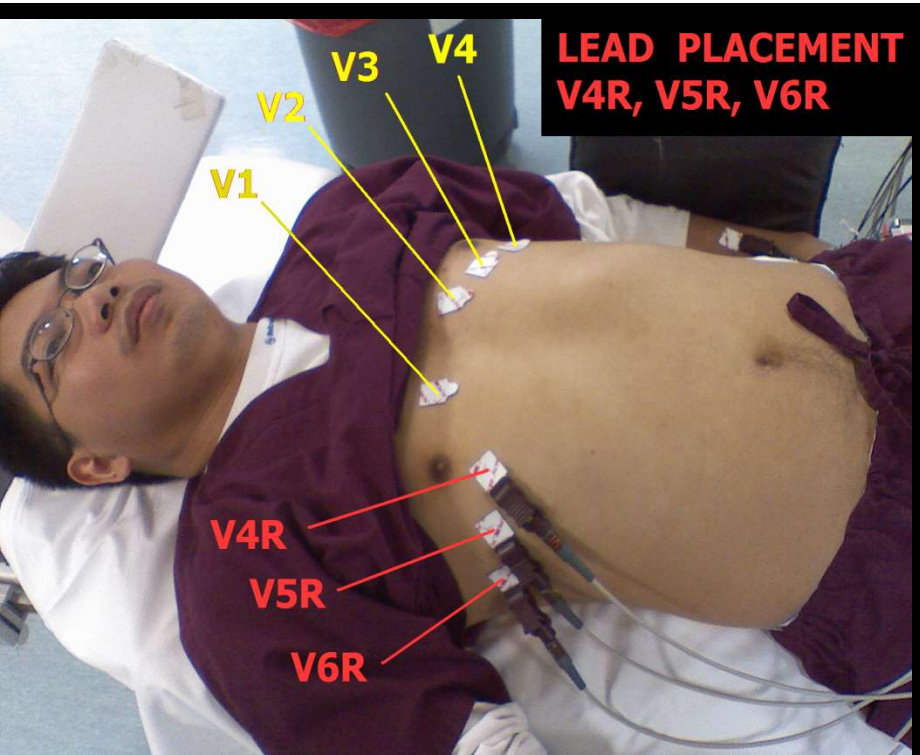


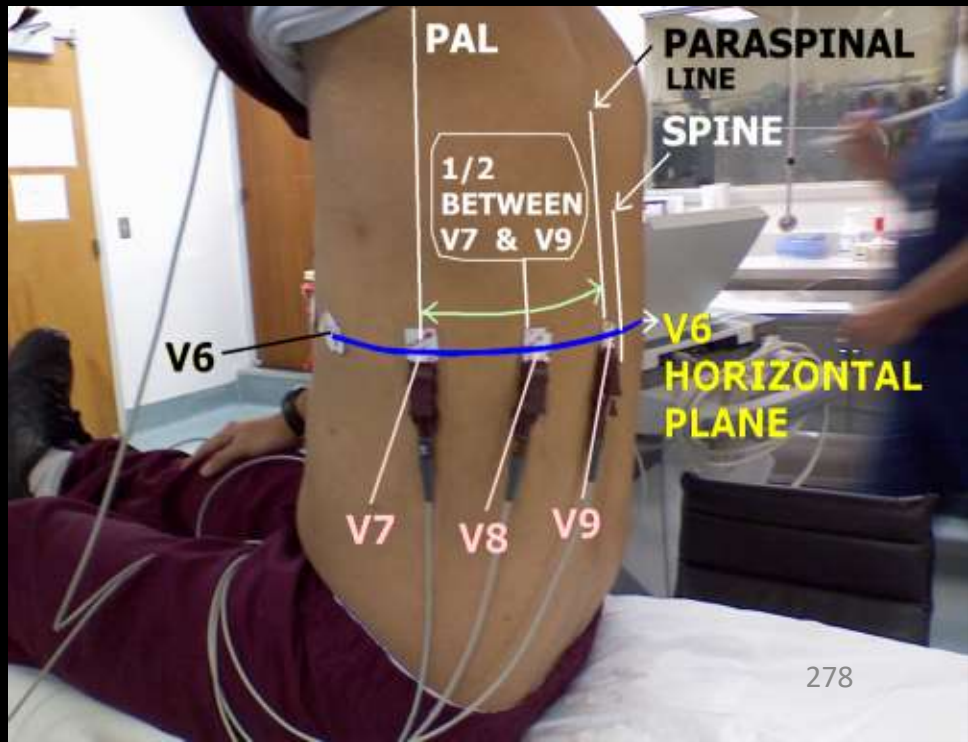

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

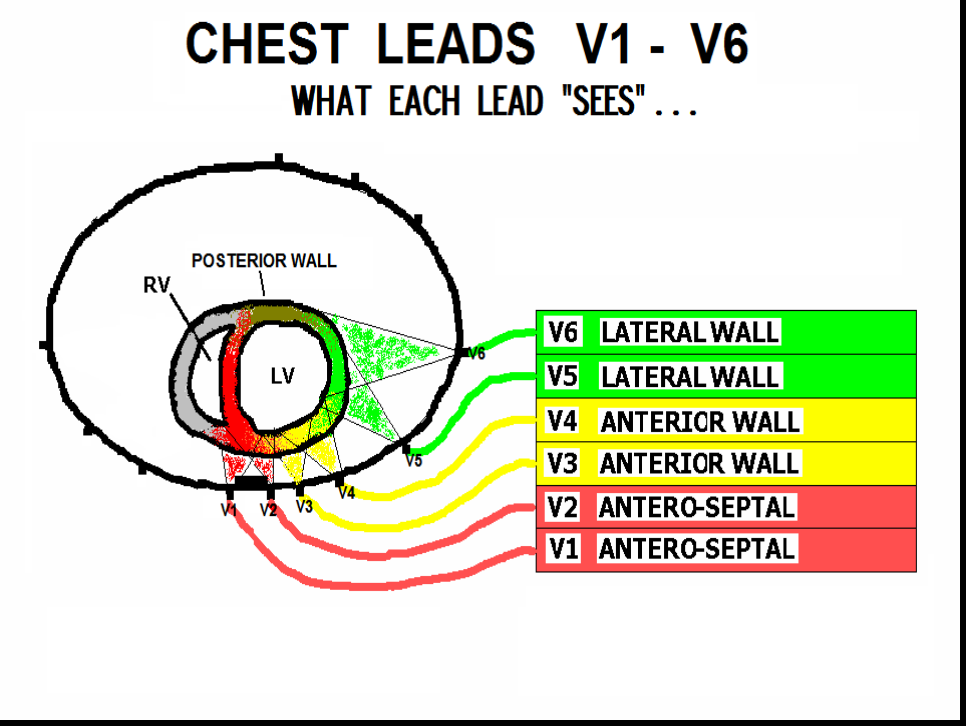

PAL
PARASPINAL LINE
SPINE
1/2 BETWEEN V7 & V9
V6
V6 HORIZONTAL PLANE
V7
V8
V9

# HOW TO REPOSITION 6 CHEST LEADS to OBTAIN 3 R VENTRICLE and 3 POSTERIOR LEADS



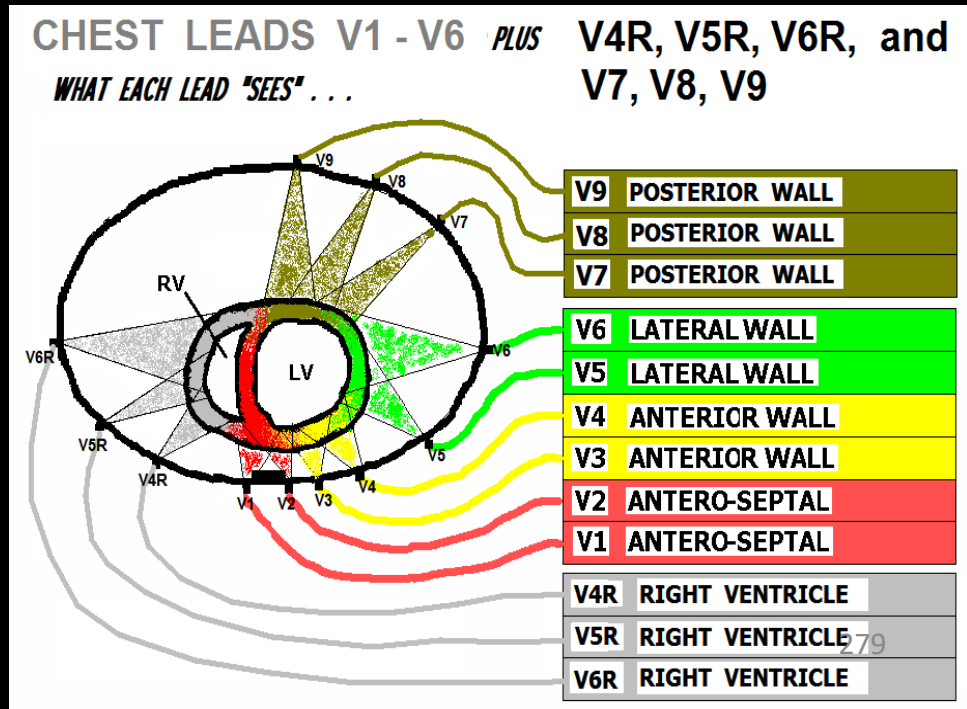






⇦ The 12 Lead ECG

The 18 Lead ECG ⇨



34years
Male Asian

Room:

Opt:

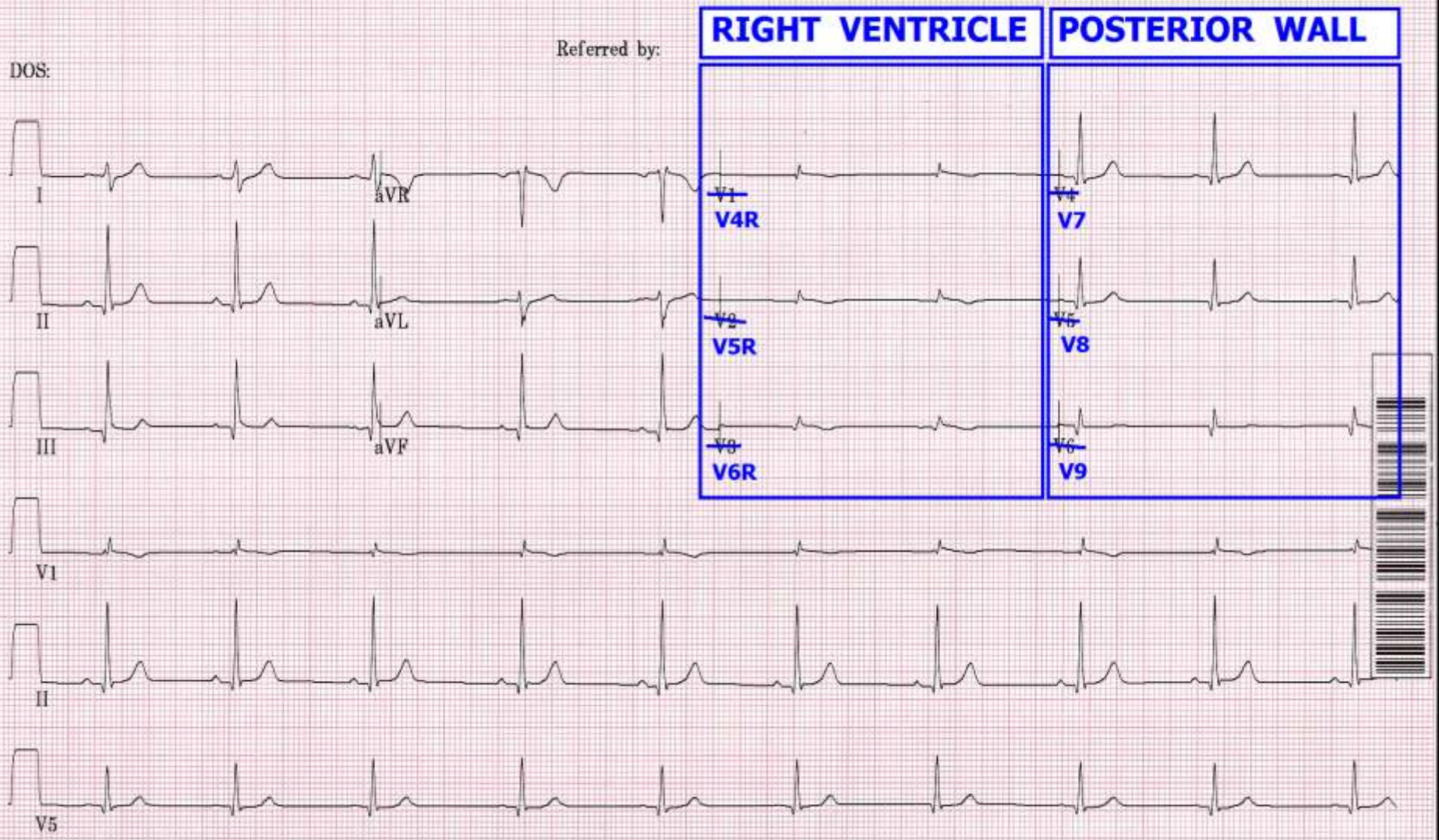
Vent. rate 58 bpm
PR interval 146 ms
QRS duration 82 ms
QT/QTc 372/365 ms
P-R-T axes 29 82 50

Sinus bradycardia
~~RSR' or QR pattern in V1 suggests right ventricular conduction delay~~
~~Cannot rule out Anteroseptal infarct, age undetermined~~
~~Abnormal ECG~~

Technician: WR

Referred by:

DOS:




40 Hz 25.0 mm/s 10.0 mm/mV 4 by 2.5s + 3 rhythm lds MAC5K 007A.2 12SL™v235

POSTERIOR WALL MI usually accompanies INFERIOR and/or LATERAL WALL MI !!!

POSTERIOR WALL MI usually accompanies INFERIOR and/or LATERAL WALL MI !!!

*. . . . On rare occasions, we see isolated cases of POSTERIOR WALL MI*

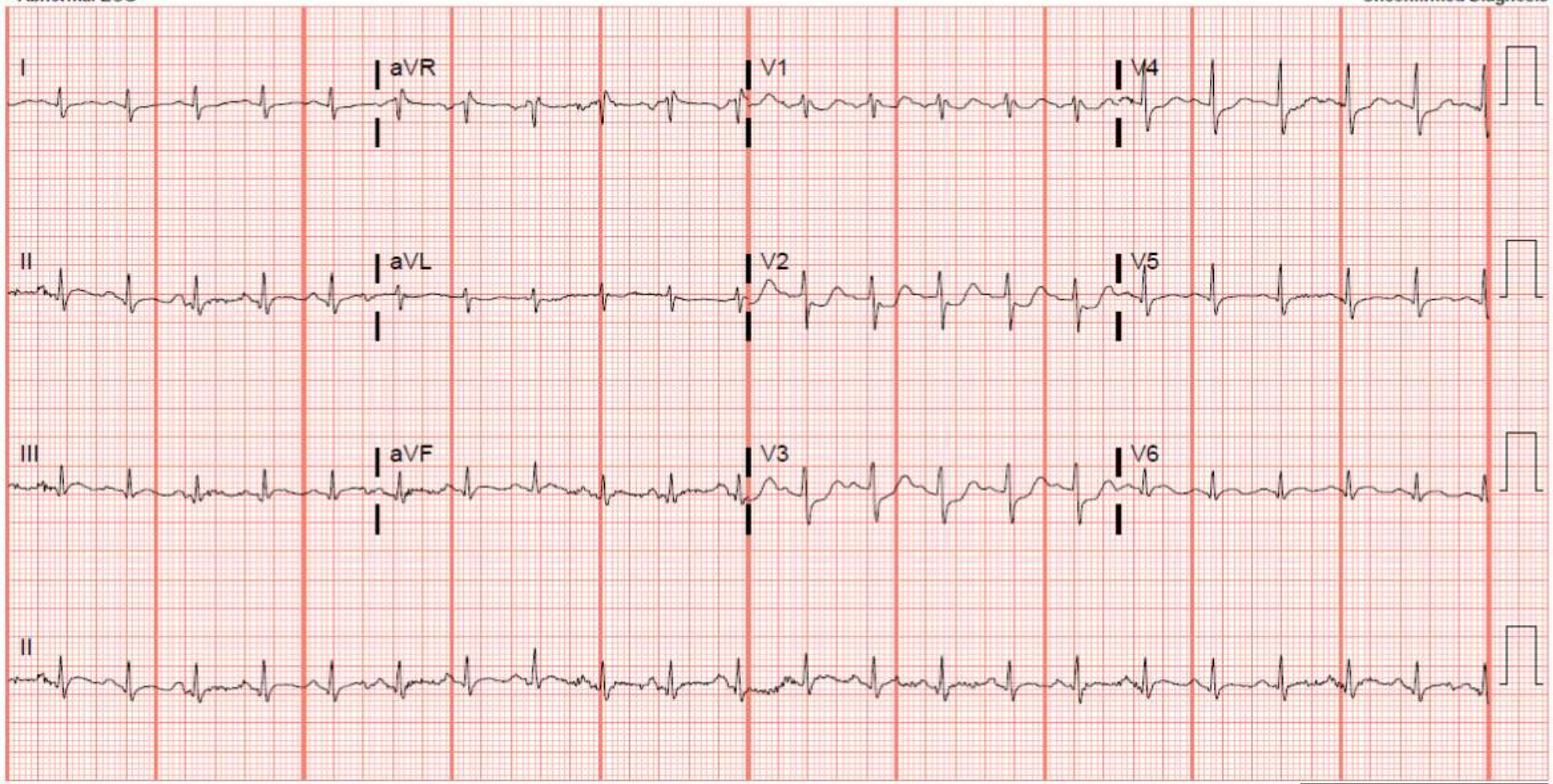
Pat ID [redacted]
[redacted] 2019 22:07:54
[redacted] 46 yrs
[redacted]
Caucasian Female
Account # [redacted]
Bayfront Health Seven Rivers ED
Dept ED
Room [redacted]
Tech LDC
RX
DX

| | | |
|---|---|---|
| Rate | 131 | Sinus tachycardia |
| PR | 128 | Probable inferior infarct, old |
| QRSd | 92 | Posterior infarct, acute (LCx) |
| QT | 317 | ST depression V1-V3, suggest recording posterior leads |
| QTc | 468 | NO PREVIOUS ECG AVAILABLE FOR COMPARISON |
| --Axis-- | | |
| P | 65 | |
| QRS | 83 | |
| T | 132 | |

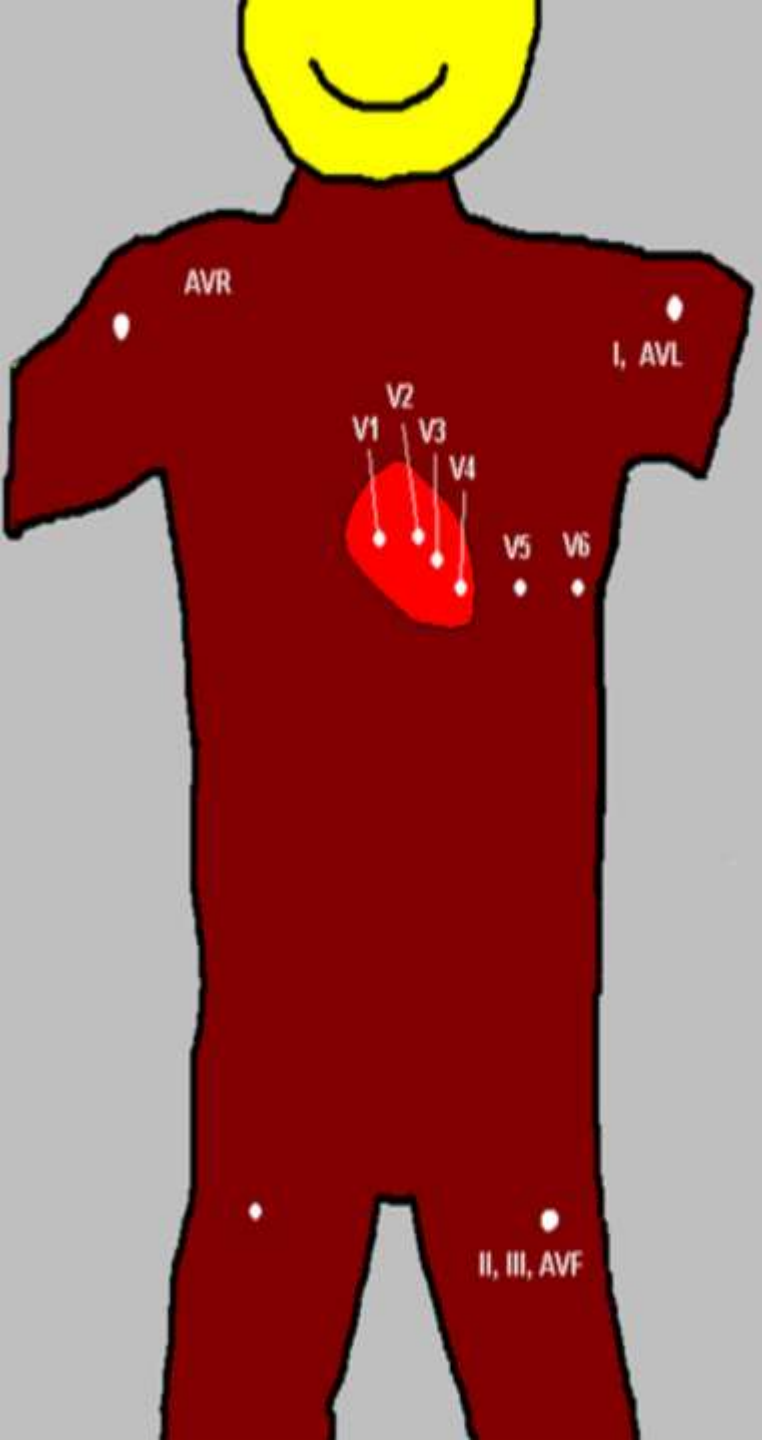
Req Provider: CHARLES NOLES

- Abnormal ECG -

Unconfirmed Diagnosis




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




| AREAS VIEWED by 12 LEAD ECG | | + | TYPICAL CORONARY ARTERIAL DISTRIBUTION |
|---|---|---|---|
| AVR | *BASILAR SEPTAL* | → | 1st SEPTAL PERFORATOR |
| AVL, I | LATERAL ANTERIOR | → | 1st DIAGONAL or RAMUS or 1st OBTUSE MARGINAL |
| V1, V2 | ANTERIOR | → | LEFT ANTERIOR DESCENDING |
| | SEPTAL | → | LEFT ANTERIOR DESCENDING |
| | POSTERIOR (recip.) | → | POSTERIOR LATERAL VESSELS |
| V3, V4 | ANTERIOR | → | LEFT ANTERIOR DESCENDING |
| V5, V6 | LATERAL | → | CIRCUMFLEX |
| II, III, AVF | INFERIOR | → | RIGHT CORONARY ARTERY or CIRCUMFLEX |

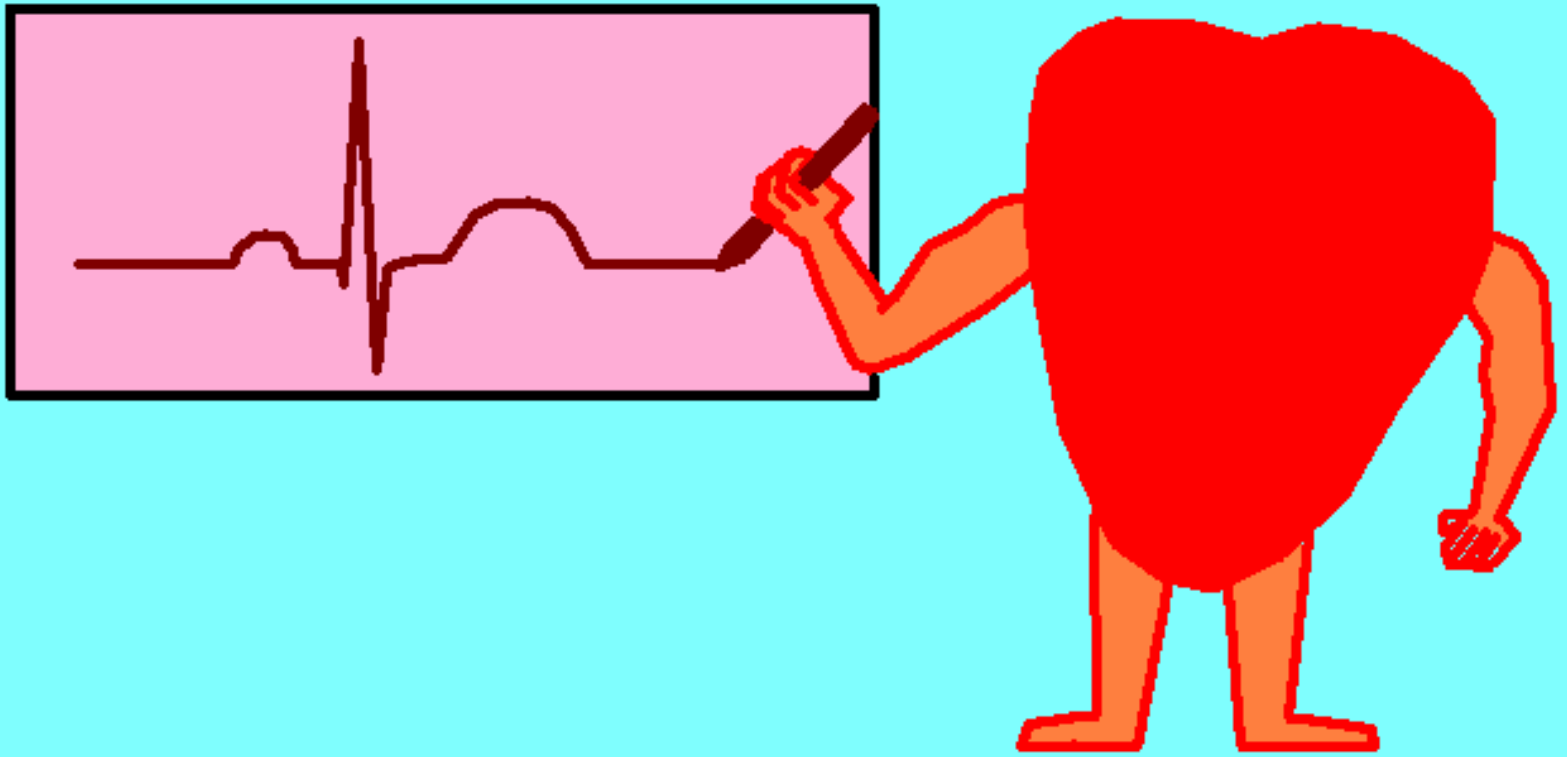
# RIGHT DOMINANT and LEFT DOMINANT systems account for approximately 90 % of the population. . . . .

The other 10% of the population have a wide diversity of coronary arterial anatomies. Please see the DOWNLOADABLE PDF version of this presentation to view this optional material !!

“ROAD TO FOREVER,” Rt 385, Oklahoma panhandle, 1994

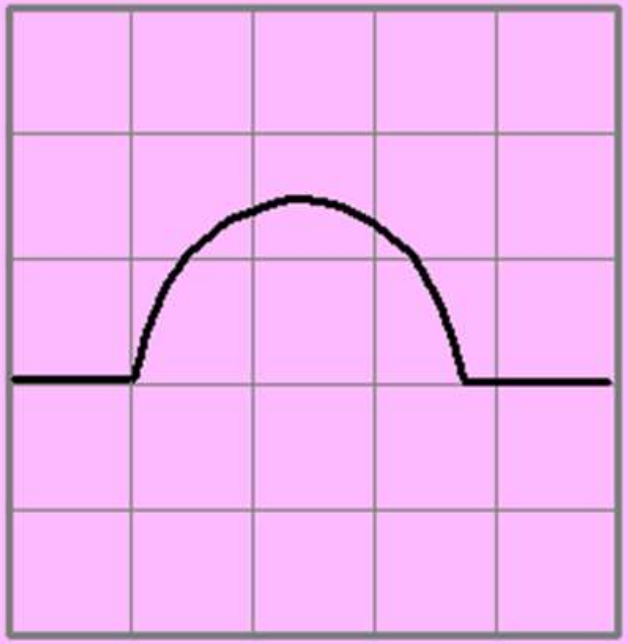
# PUTTING IT ALL ON PAPER . . .

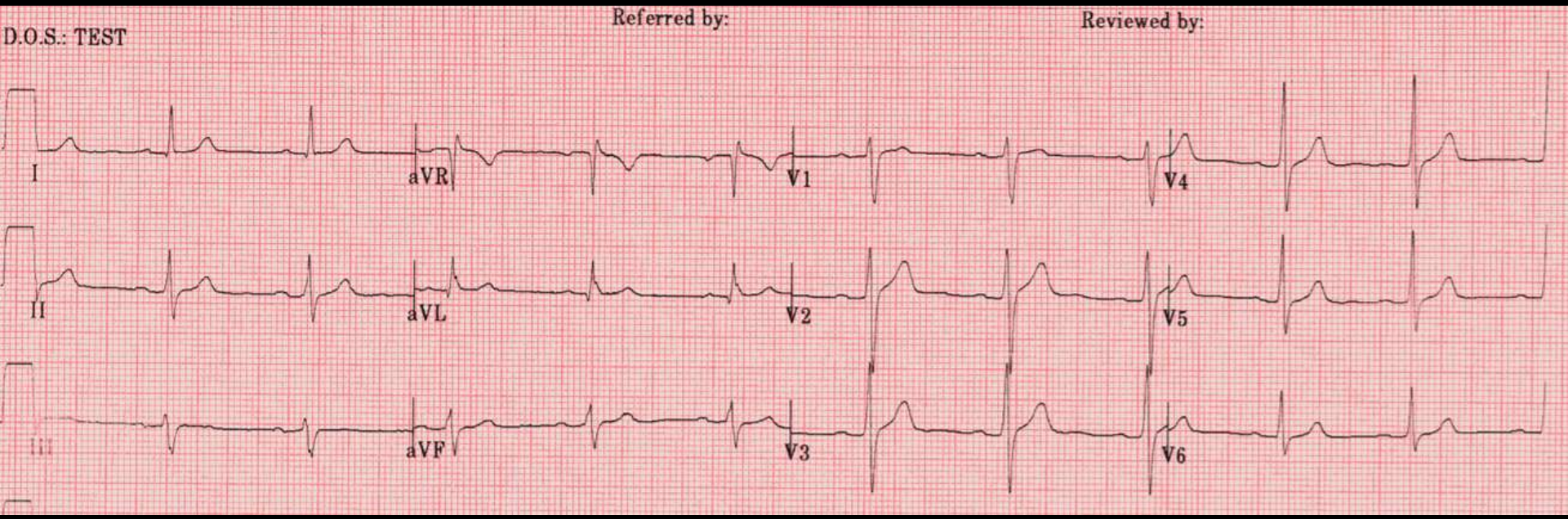
***WAVEFORMS and INTERVALS . . .***



# THE P WAVE

- SHOULD BE UPRIGHT, CONVEX-SHAPED DOME IN ALL LEADS EXCEPT AVR and V1

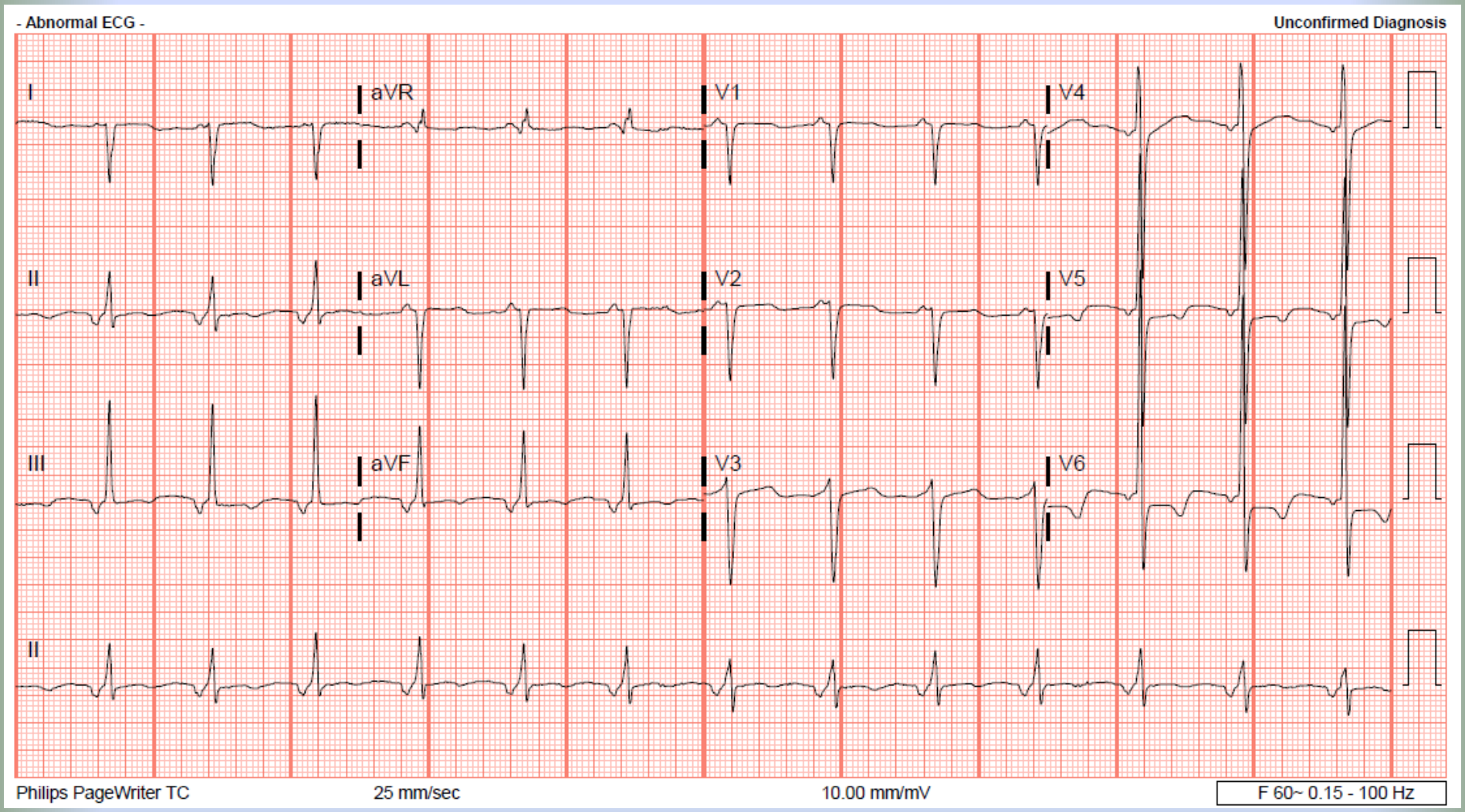



D.O.S.: TEST
Referred by:
Reviewed by:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

# P Wave Axis

- P waves with abnormal axis (“not pointing in the right direction”) may signify ectopic atrial beats.
- When P waves are inverted in most leads with an abnormally short P-R interval (<120ms) the origin of the rhythm may be the AV node (Junctional Rhythm).

# Inverted P waves & short P-R interval:

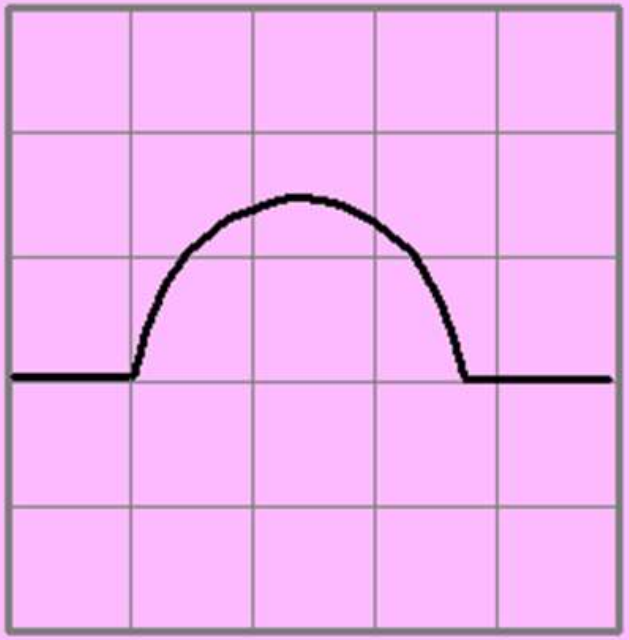


# Evaluate P Wave for Atrial  Hypertrophy

- Evaluate amplitude and duration in Lead II

# THE P WAVE

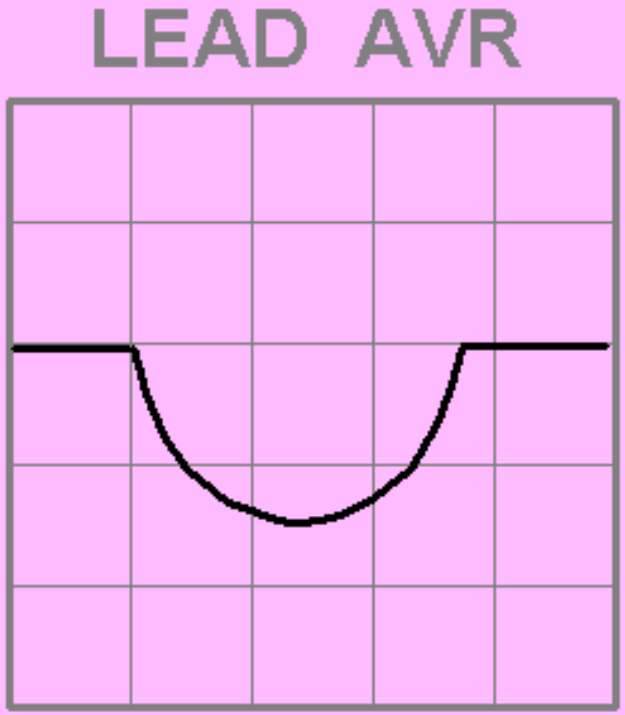
- SHOULD BE UPRIGHT, CONVEX-SHAPED DOME IN ALL LEADS EXCEPT AVR and V1



- SHOULD BE LESS THAN .2 mv (2 mm) HIGH
- SHOULD BE LESS THAN 100 ms (2.5mm) LONG

# THE P WAVE

- SHOULD BE INVERTED IN LEAD AVR



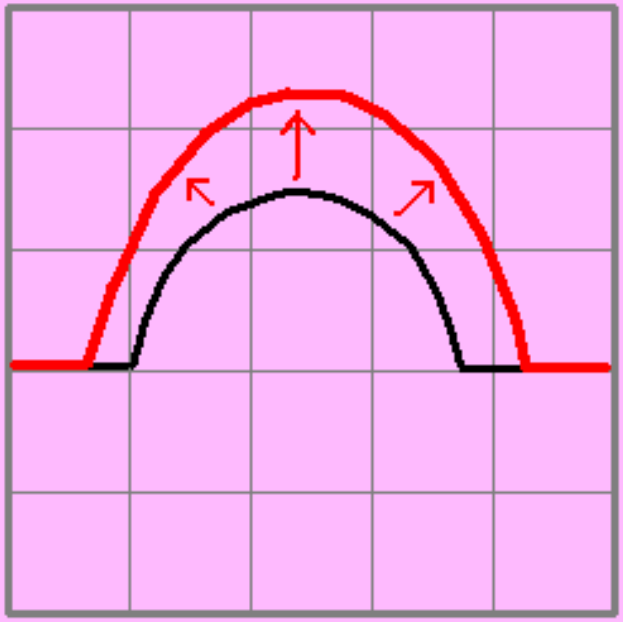
# THE P WAVE

When the P WAVE

is

***TOO LARGE***

We think of



***ATRIAL HYPERTROPHY***

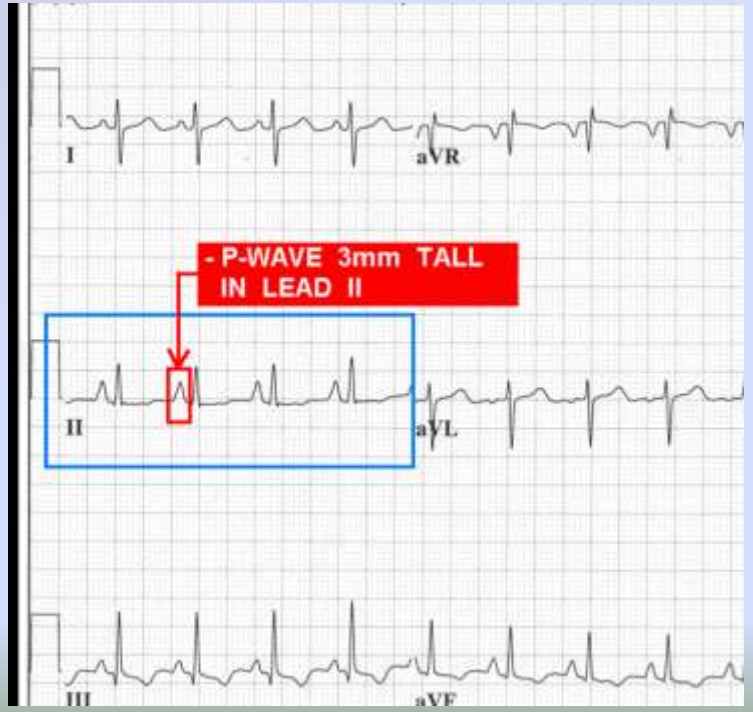
( SPECIFIC CRITERIA FOR ATRIAL HYPERTROPHY IS DISCUSSED IN MORE DETAIL IN THE "CHAMBER HYPERTROPHY" SECTION )

# Evaluate P Wave for Atrial Hypertrophy

- Evaluate amplitude and duration in ***Lead II***

# Evaluate P Wave for Atrial Hypertrophy

- Evaluate amplitude and duration in Lead II
- If the P wave it “too tall (>2mm) or too long (>2.5mm)” in Lead II, ***then go to Lead V1*** to evaluate P wave . . . .



# THE P WAVE

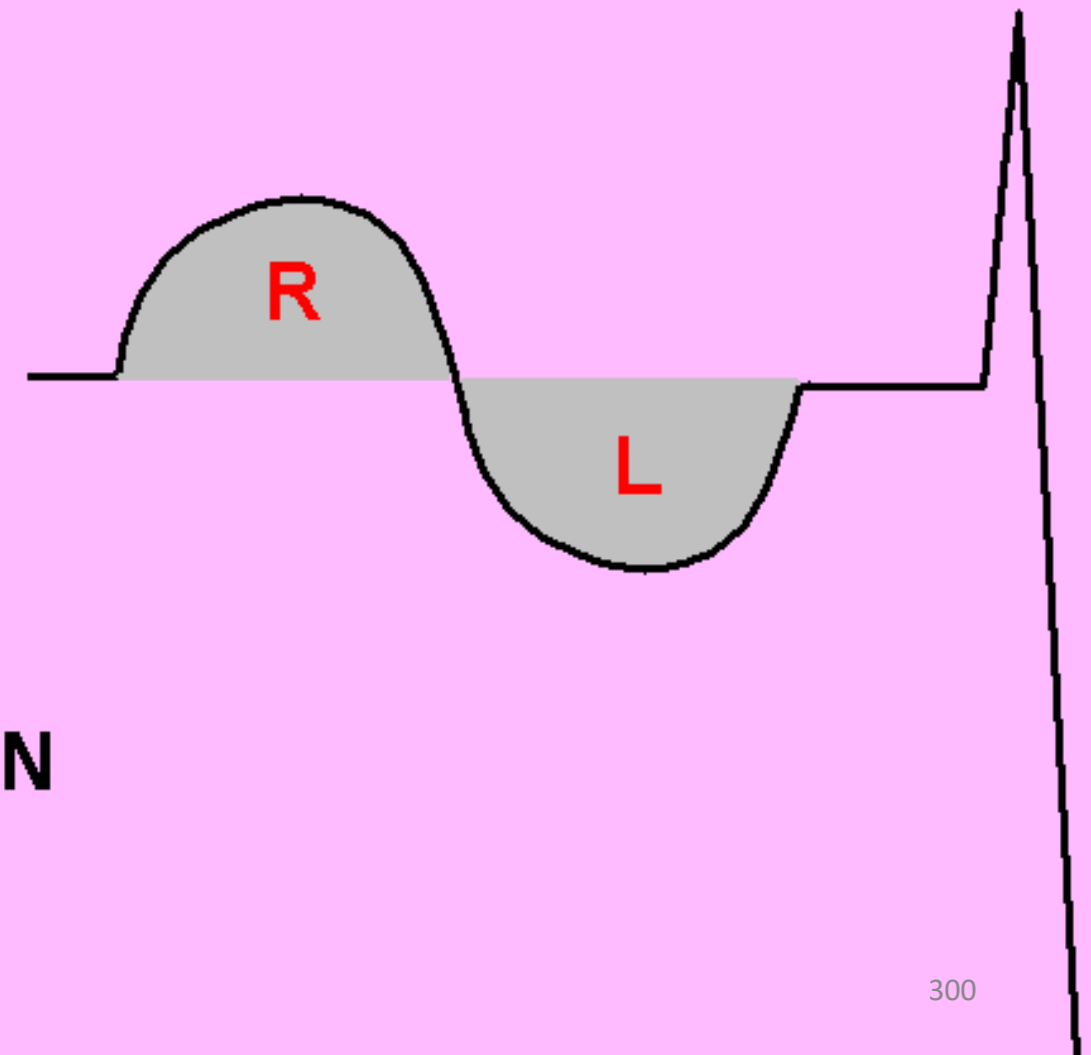
## IN LEAD V1 MAY BE:

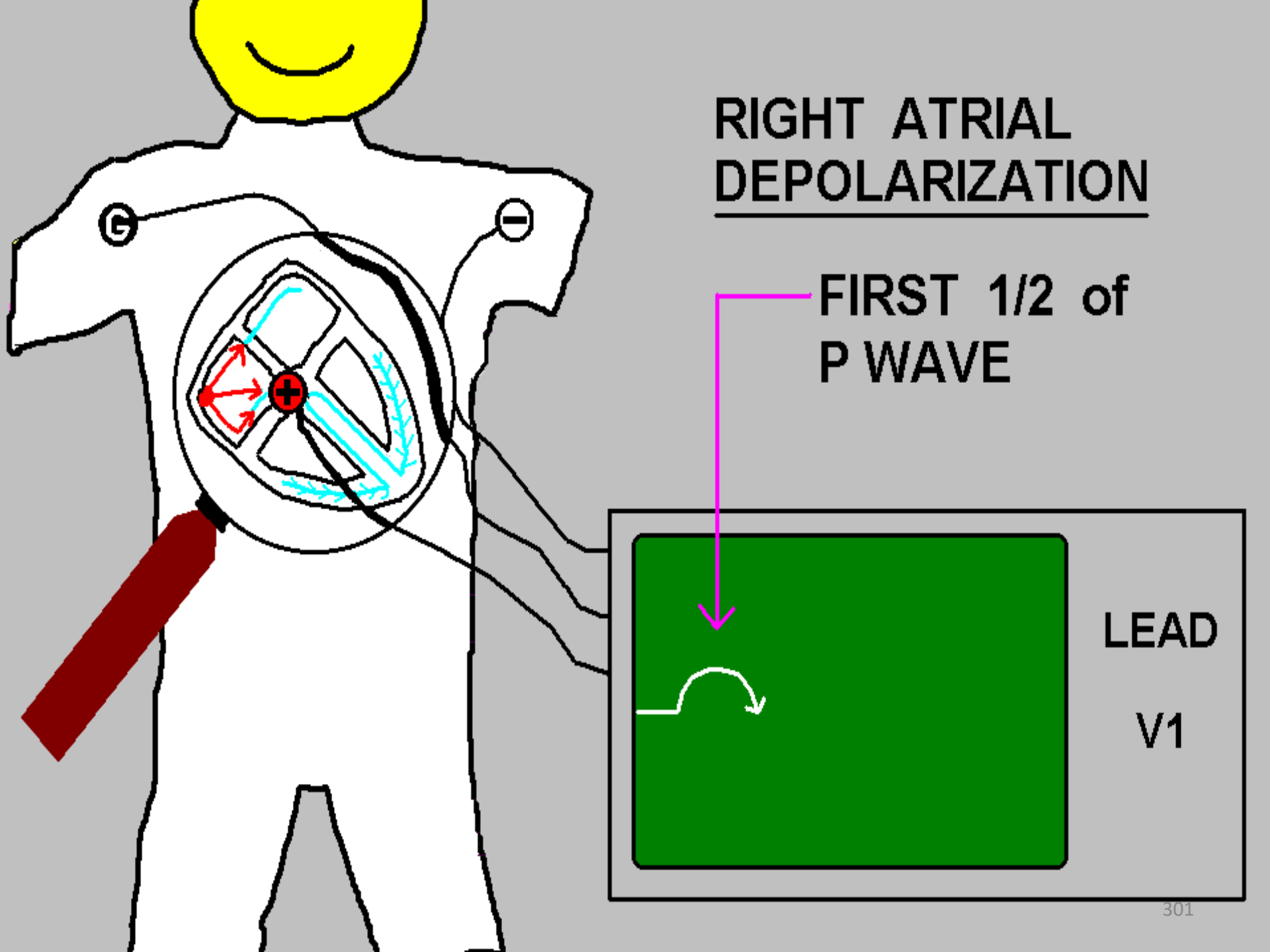
- POSITIVE

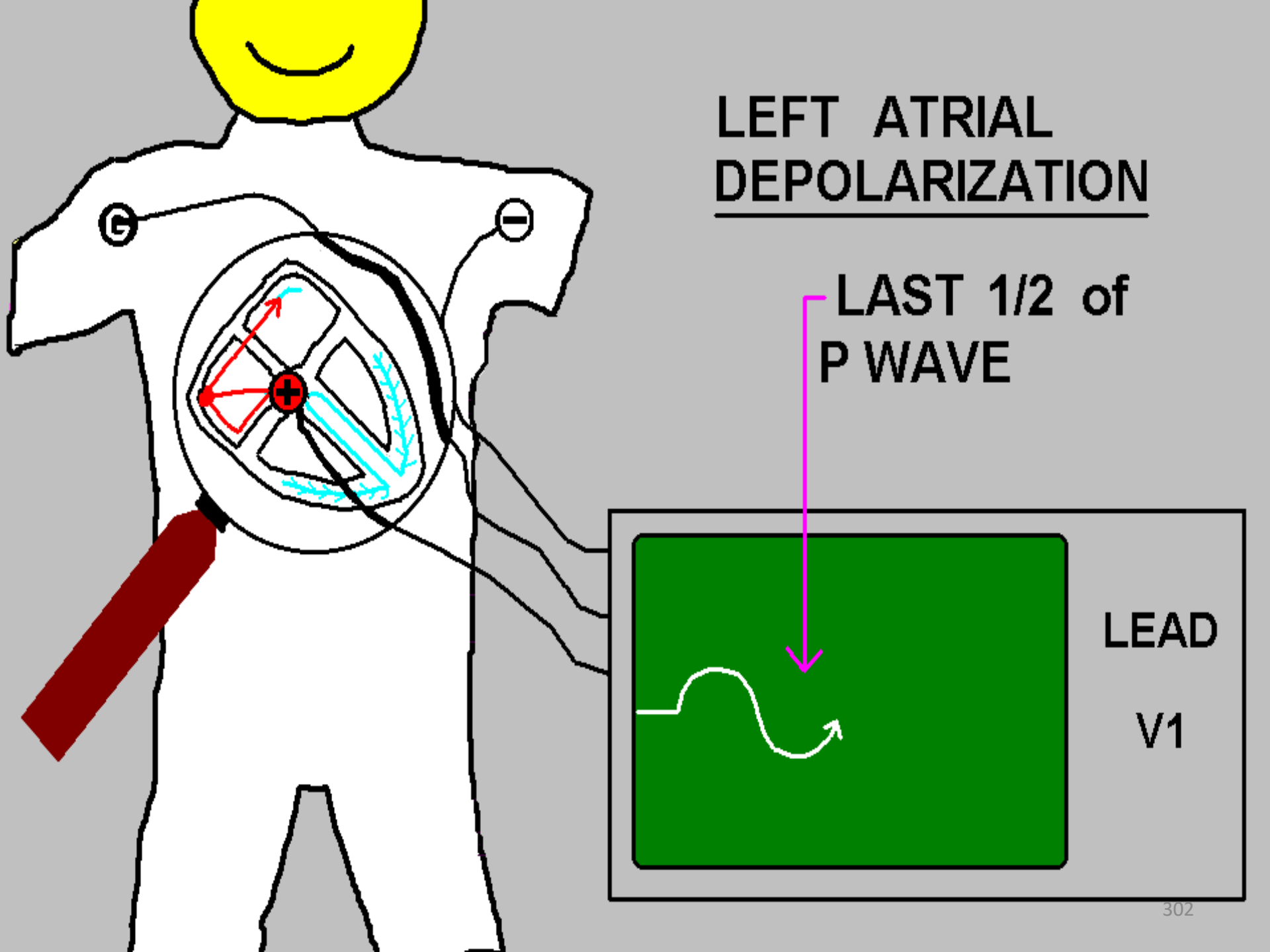
- OR BI-PHASIC

# THE P WAVE

- WHEN THE P WAVE IS BI-PHASIC IN V1, IT DISPLAYS BOTH R and L ATRIAL DEPOLARIZATION



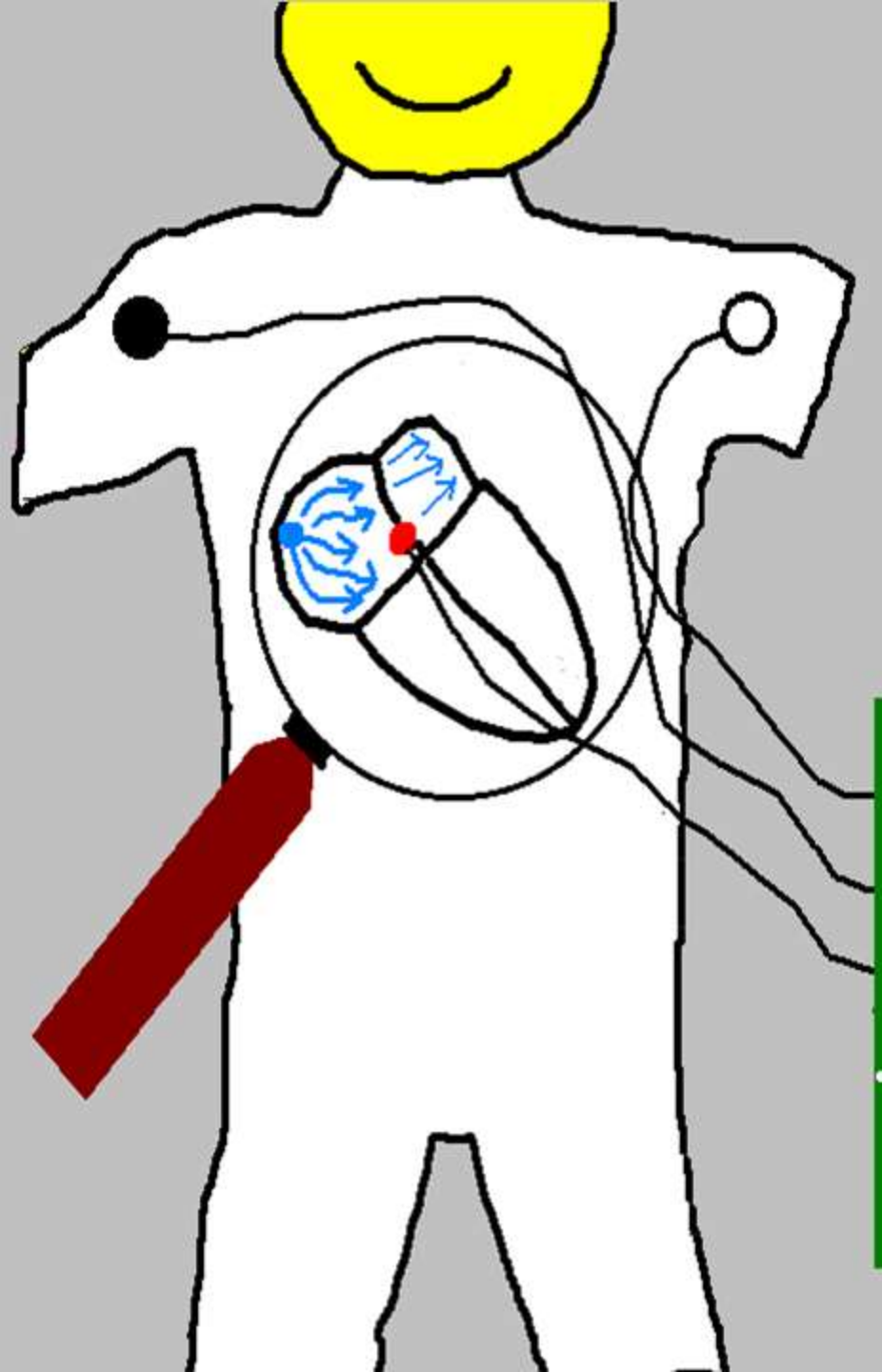

RIGHT ATRIAL
DEPOLARIZATION
FIRST 1/2 of
P WAVE
LEAD
V1


LEFT ATRIAL
DEPOLARIZATION
LAST 1/2 of
P WAVE
LEAD
V1

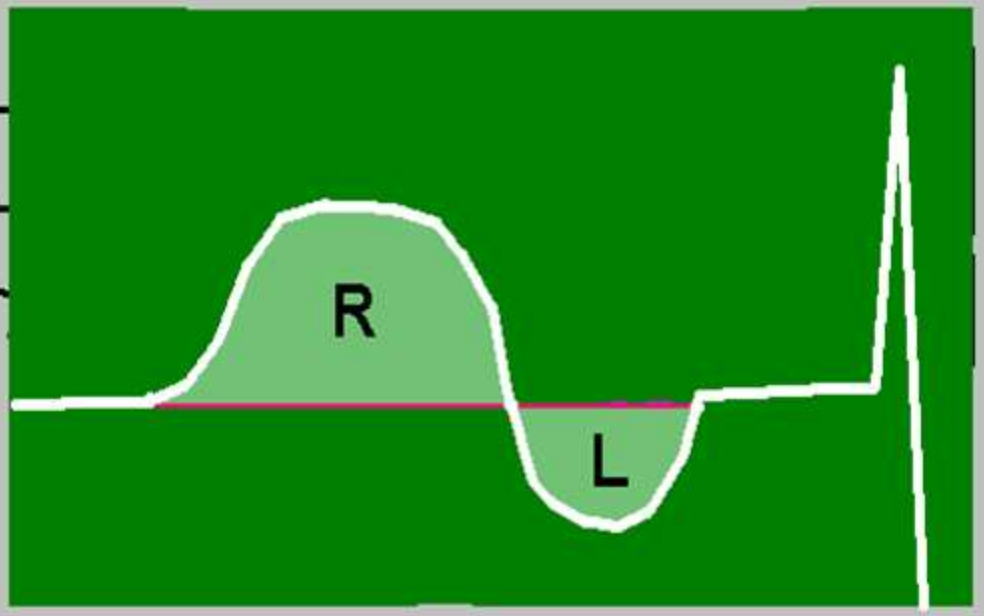
# Evaluate P Wave for Atrial Hypertrophy

- Evaluate amplitude and duration in Lead II
- If the P wave it “too tall (>2mm) or too long (>2.5mm)” in Lead II, ***then go to lead V1*** to evaluate P wave.
- In Lead V1, if the first half (positive deflection) of the P wave is LARGER than the second half (negative deflection) it suggests RIGHT ATRIAL HYPERTROPHY (RAH).

# RIGHT ATRIAL ENLARGEMENT



## P-WAVE IN V1



02-DEC-1998 00:24:45 ST. JOSEPH'S HOSPITAL-ER ROUTINE RETRIEVAL

29 yr
Male Black

Room:ER
Loc:3 Option:28

| | | |
|---|---|---|
| Vent. rate | 107 | BPM |
| PR interval | 132 | ms |
| QRS duration | 80 | ms |
| QT/QTc | 310/413 | ms |
| P-R-T axes | 67 105 | -32 |

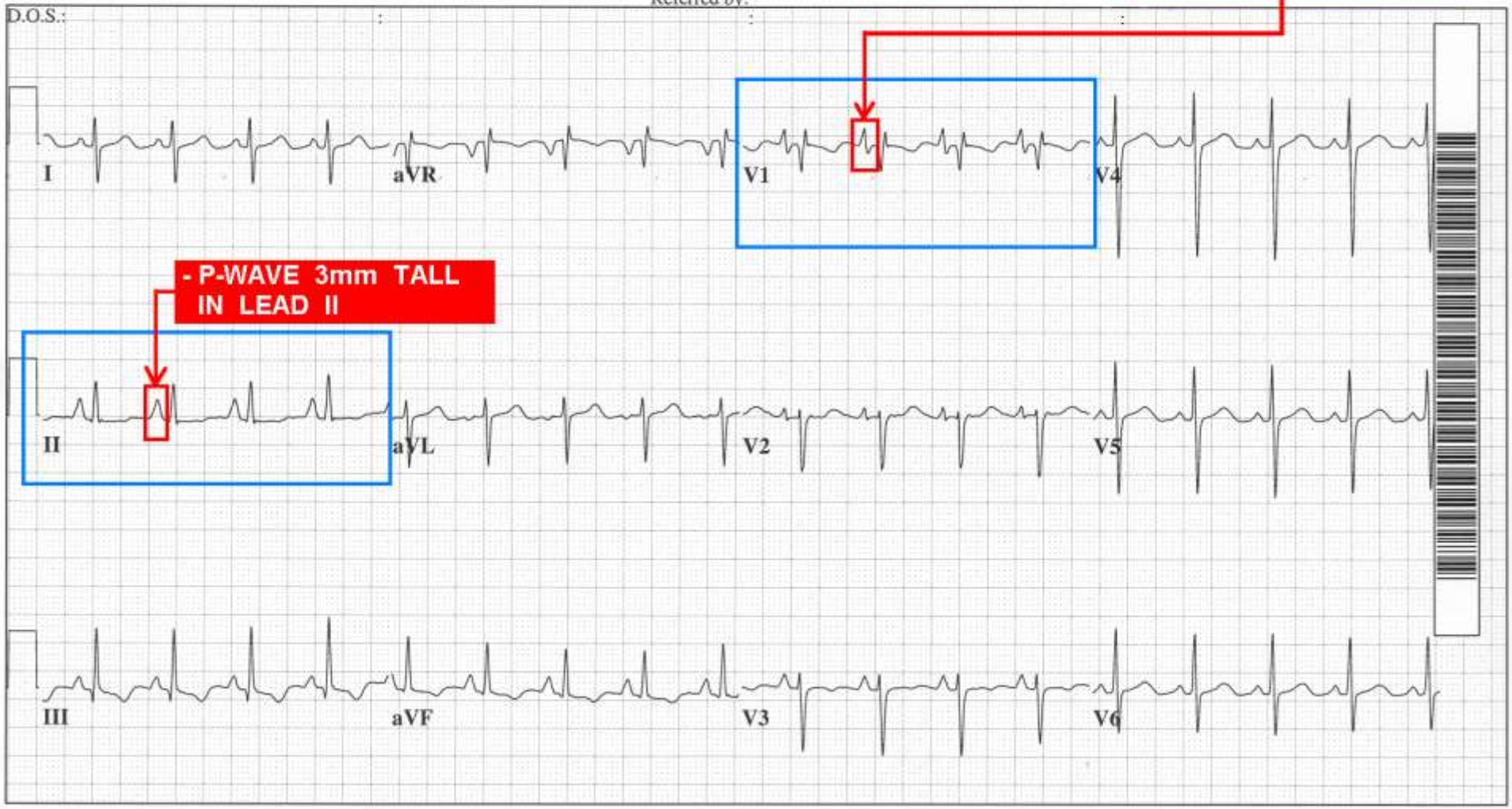
Sinus tachycardia
Right atrial enlargement
Rightward axis
Pulmonary disease pattern
RSR' or QR pattern in V1 suggests right ventricular
T wave abnormality, consider inferior ischemia
Abnormal ECG
When compared with ECG of 01-OCT-1998 21:45
T wave inversion more evident in Inferior leads ...

Referred by:

**EKG CLASS #WR03446043**

- POSITIVE DEFLECTION TALLER (more dominant) IN LEAD V1

- P-WAVE 3mm TALL IN LEAD II



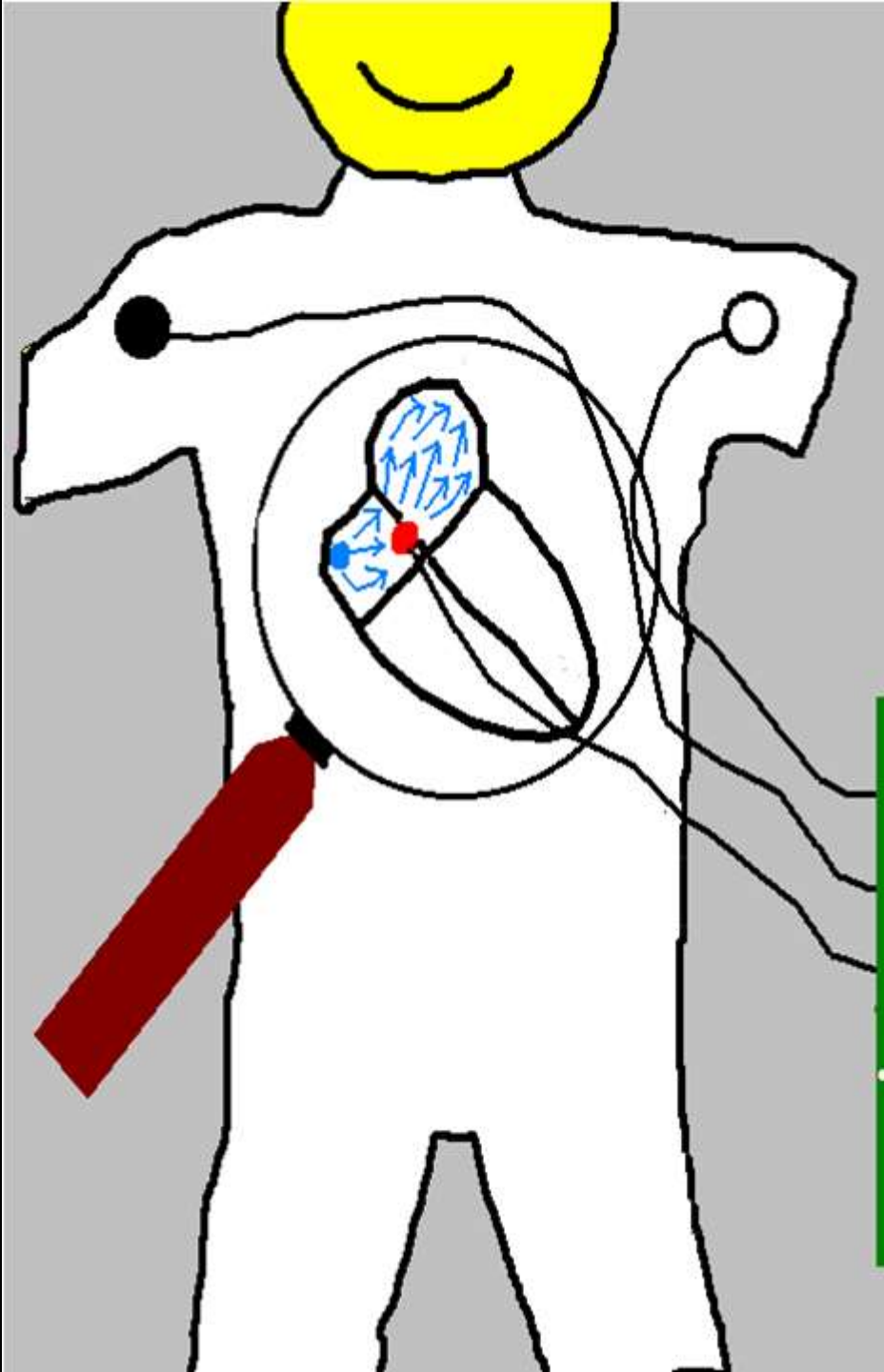

25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 13

EID:17 EDT: 07:28 02-DEC-1998 ORDER:

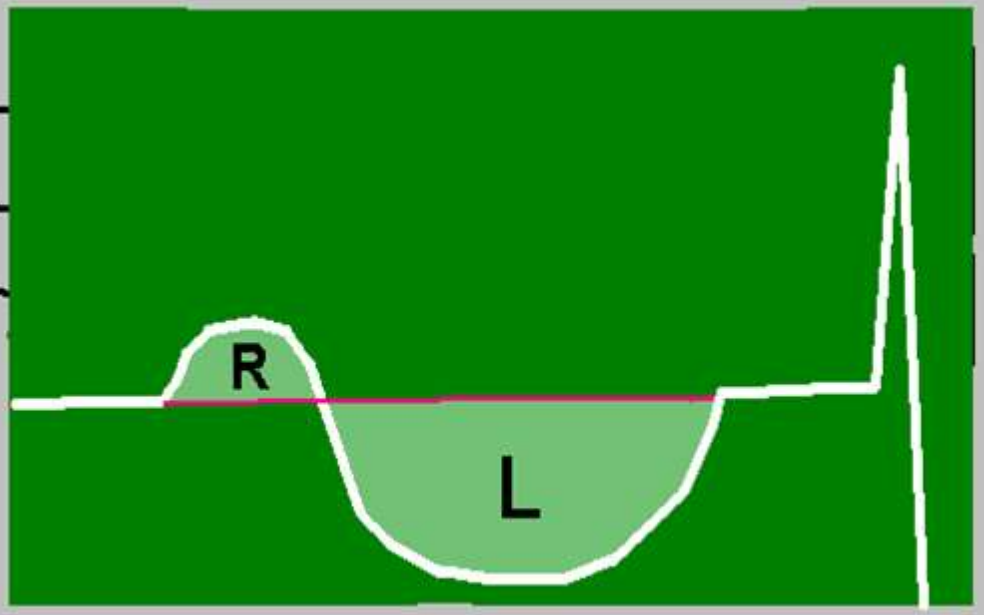
# Evaluate P Wave for Atrial Hypertrophy

- Evaluate amplitude and duration in Lead II
- If the P wave it “too tall (>2mm) or too long (>2.5mm)” in Lead II, ***then go to lead V1*** to evaluate P wave.
- In Lead V1, if the first half (positive deflection) of the P wave is LARGER than the second half (negative deflection) it suggests RAH. If the second half (negative deflection) is larger, it suggests LEFT ATRIAL HYPERTROPY (LAH).

# LEFT ATRIAL ENLARGEMENT



## P-WAVE IN V1



01–MAY–1998 03:09:15 ST. JOSEPH'S HOSPITAL–ER ROUTINE RETRIEVAL

77 yr
Male Caucasian

Room:S 1
Loc:3 Option:10

| | | |
|---|---|---|
| Vent. rate | 106 | BPM |
| PR interval | 170 | ms |
| QRS duration | 104 | ms |
| QT/QTc | 374/496 | ms |
| P–R–T axes | 67 –66 | 70 |

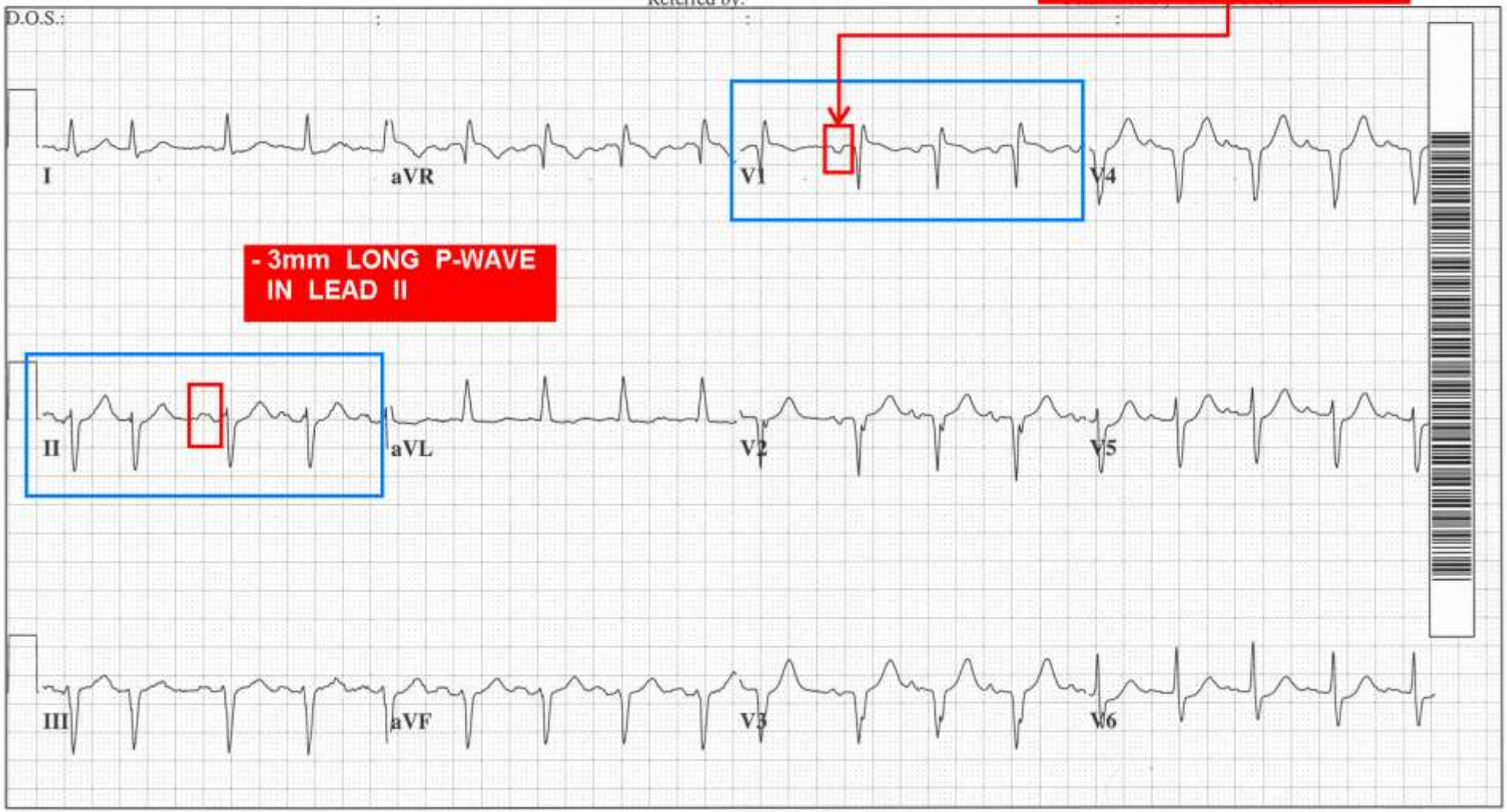
Sinus tachycardia with occasional Premature supraventricular complexes
Left atrial enlargement
Left axis deviation
Incomplete right bundle branch block
Anteroseptal infarct , age undetermined
Abnormal ECG
No previous ECGs available

**EKG CLASS #WR03651849**

**- NEGATIVE DEFLECTION P-WAVE IN LEAD V1**

Referred by:

D.O.S.:




**- 3mm LONG P-WAVE IN LEAD II**

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

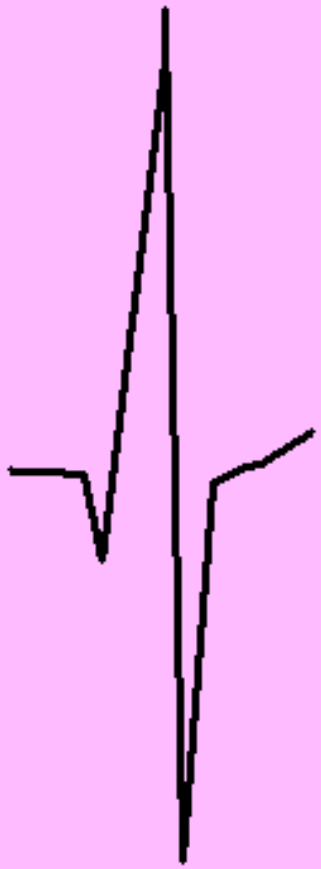
EID:17 EDT: 14:12 02–MAY–1998 ORDER:

# THE QRS COMPLEX

- MAY BE POSITIVE, NEGATIVE, OR BI- PHASIC, BASED ON THE LEAD VIEWED
- TOTAL WIDTH SHOULD BE LESS THAN 120 ms / or .12

# THE QRS COMPLEX

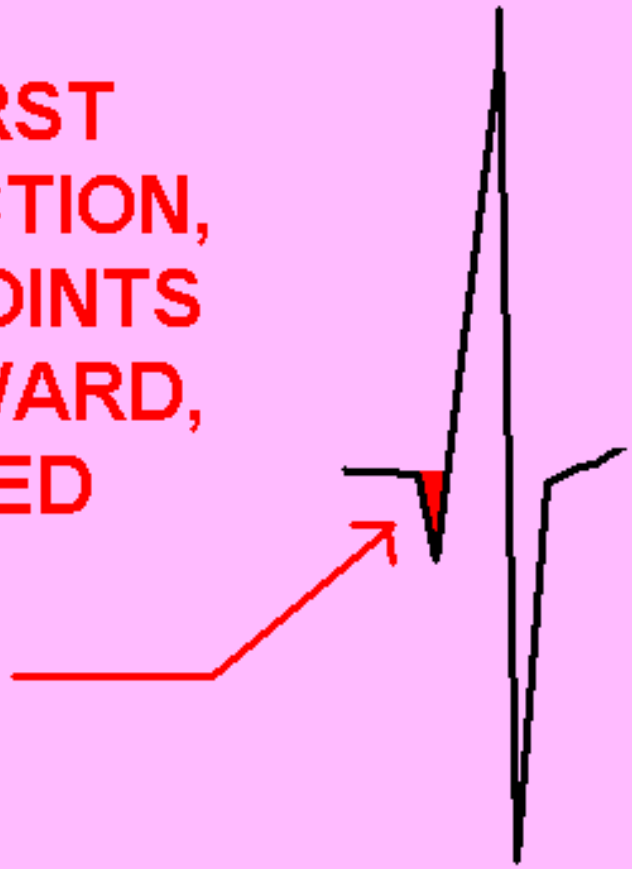
THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .



# THE QRS COMPLEX

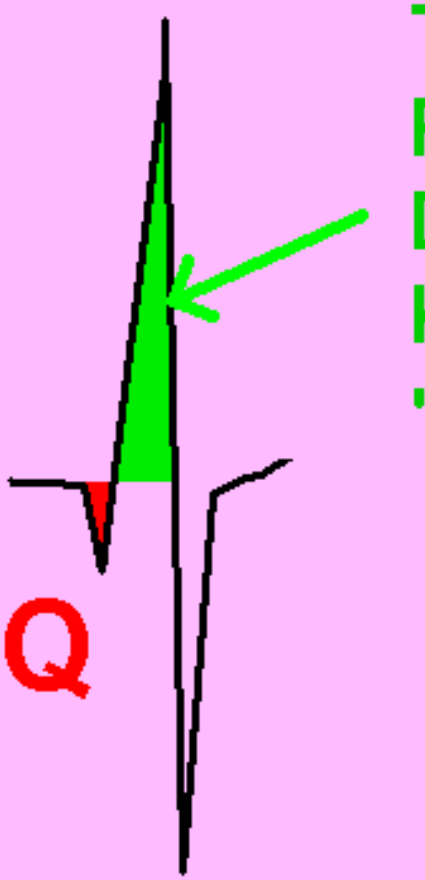
THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .

THE FIRST DEFLECTION, IF IT POINTS DOWNWARD, IS NAMED THE "Q WAVE"



# THE QRS COMPLEX

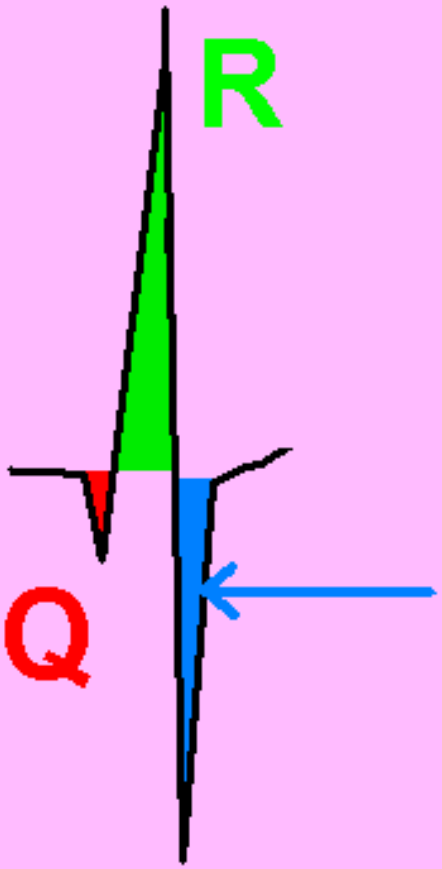
THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .




THE FIRST POSITIVE DEFECTION IS KNOW AS THE "R" WAVE

# THE QRS COMPLEX

THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .

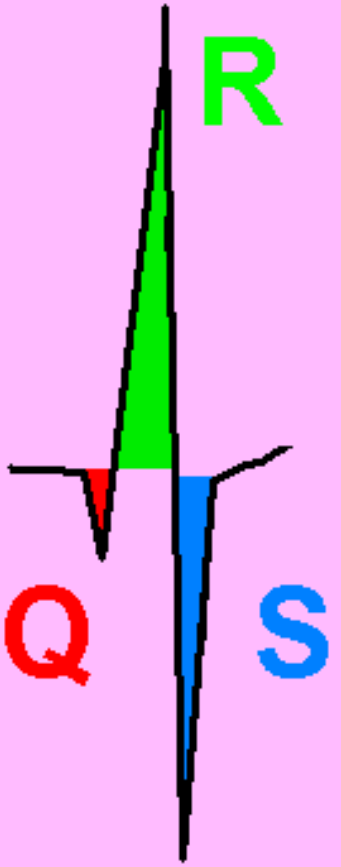



A NEGATIVE DEFLECTION AFTER THE R WAVE IS CALLED THE " S " WAVE

# THE QRS COMPLEX

THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .

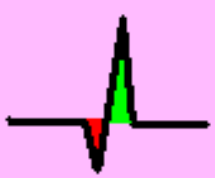
AND IS THE ONLY TRUE "QRS" COMPLEX




SOME OF THE OTHER VARIATIONS INCLUDE . . . .

# THE QRS COMPLEX

## WHAT ARE THESE COMPLEXES ??



qR

RS

R

Rsr'

Qr

QS

qRSr'

qR

rS

Rr'

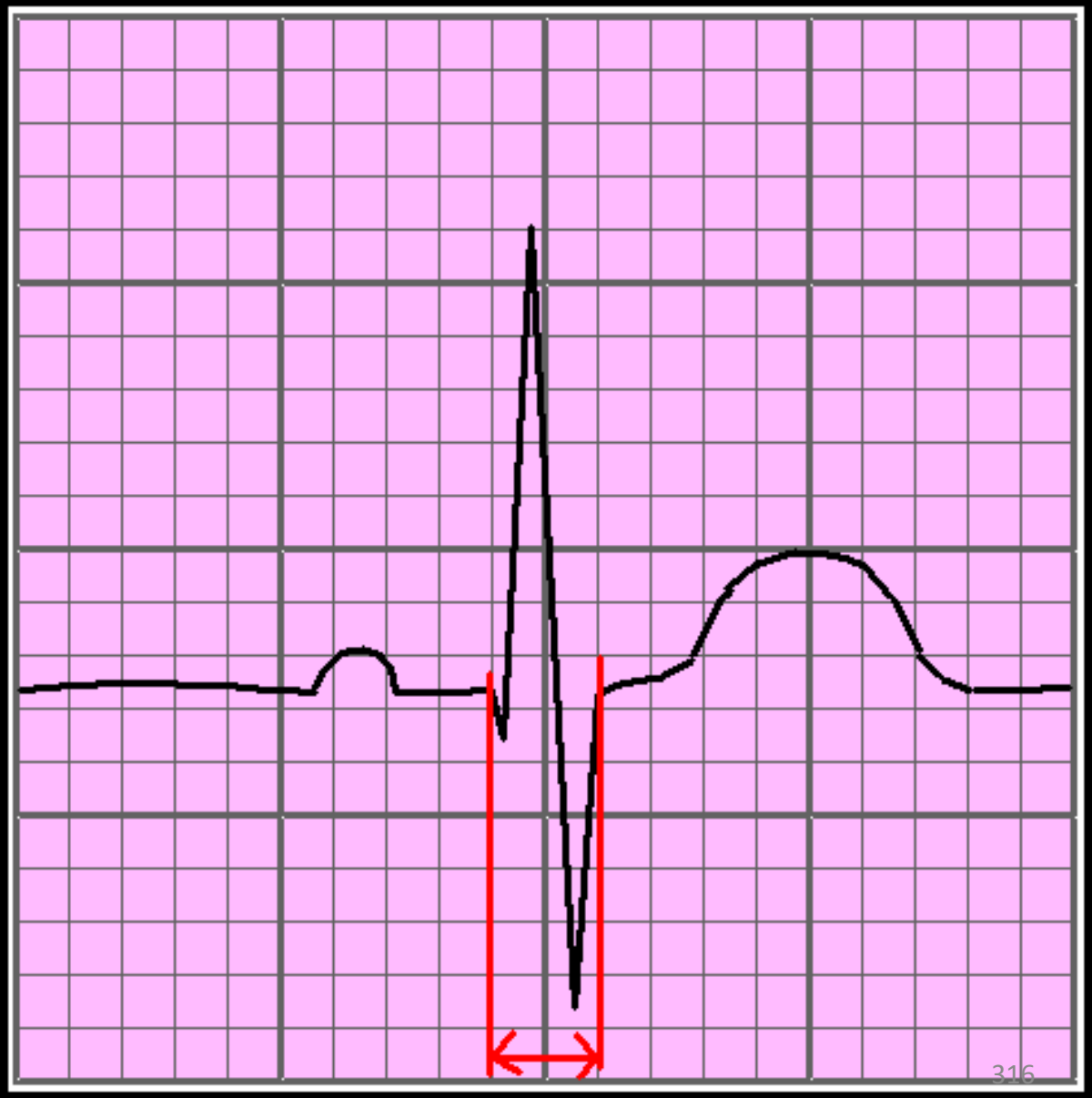
# QRS INTERVAL

LESS THAN

.12

OR

120 mSEC



# QRS COMPLEX TOO WIDE

WIDER THAN 120 mSEC

THINK:

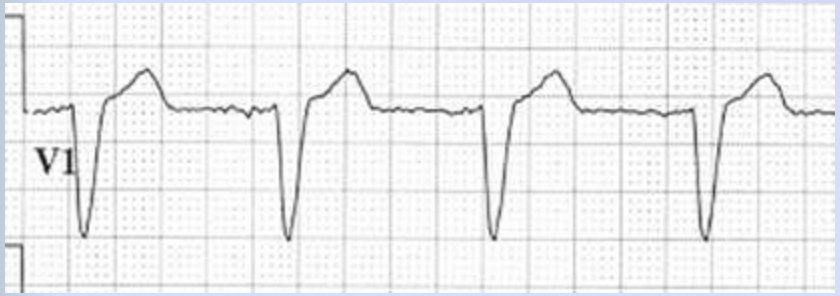
- BUNDLE BRANCH BLOCK
- VENTRICULAR COMPEX (ES)
- PACED RHYTHM
- L VENTRICULAR HYPERTROPHY
- ELECTROLYTE IMBAL. ( $\uparrow K^+$ $\downarrow Ca^{++}$ )
- DELTA WAVE (PRE-EXCITATION)

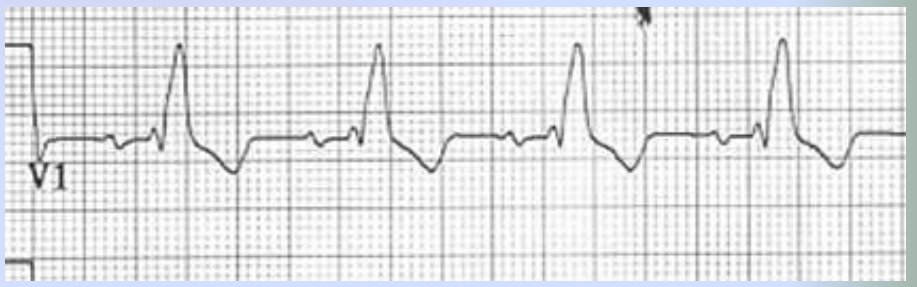
# When the QRS is WIDE (> 3mm):

- **If you KNOW the Rhythm is originating ABOVE the Ventricles (such as NSR or any Supraventricular Rhythm) – you should determine if the QRS has a RIGHT or LEFT Bundle Branch Block morphology.**

# Normal Sinus and Other "Supraventricular Rhythms" with WIDE QRS ( > 120 ms )

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

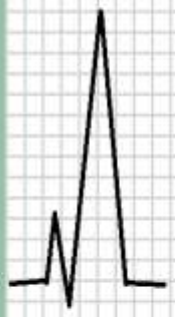


# Simple “Turn Signal Method” . . .

## THE "TURN SIGNAL METHOD" for identifying BUNDLE BRANCH BLOCK

V1



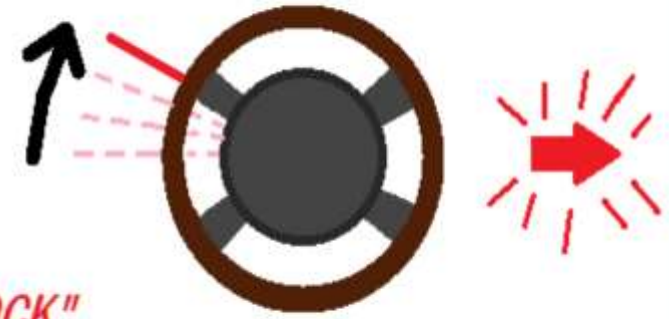
**USE LEAD V1 for this technique**

To make a **RIGHT TURN**

you push the turn signal lever **UP**. . . . .

THINK:

*"QRS points UP = RIGHT BUNDLE BRANCH BLOCK"*



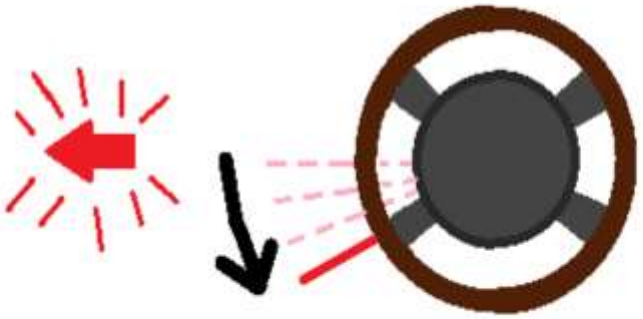
V1

To make a **LEFT TURN**

you push the turn signal lever **DOWN** . . . .

THINK:

*"QRS points DOWN = LEFT BUNDLE BRANCH BLOCK"*



RUPPERT, WAYNE ID: 099999999 4-Mar-2001

74years Male Caucasian
Room:
Loc: 0 Opt:
Technician: WR

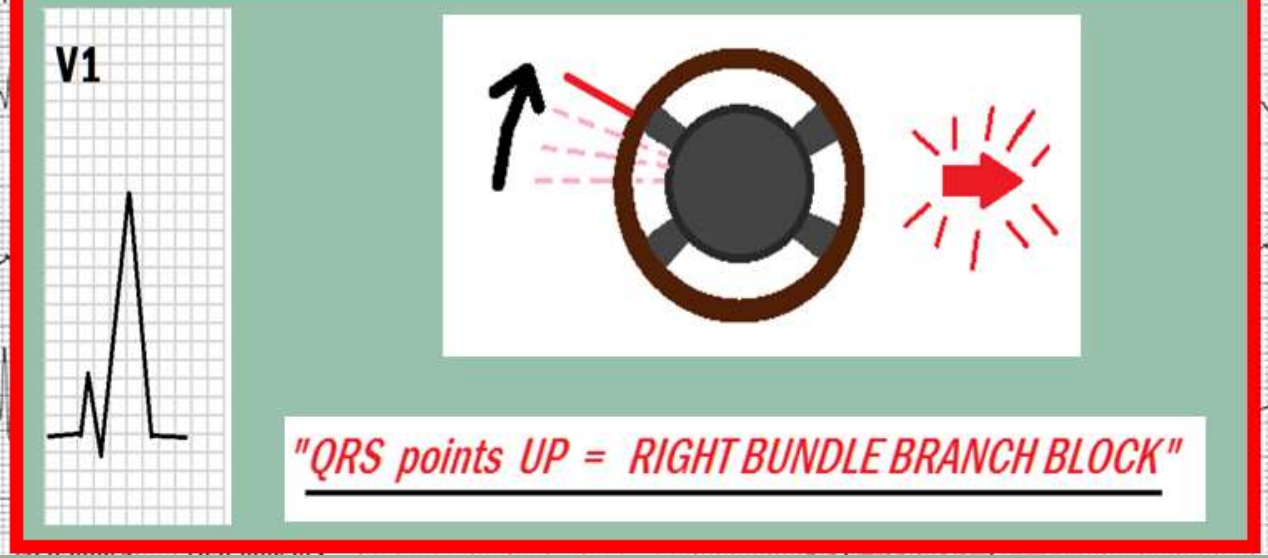
| | | |
|---|---|---|
| Vent. rate | 72 | bpm |
| PR interval | 186 | ms |
| QRS duration | 166 | ms |
| QT/QTc | 436/477 | ms |
| P-R-T axes | 57 -32 32 | |

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

# USE LEAD V1 for this technique

D.O.S.:

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




40 Hz MACVU 003C 12SL$^{tm}$ v250

09:16:40

74 yr
Female Caucasian

Loc:7 Option:35

| | | |
|---|---|---|
| Vent. rate | 64 | BPM |
| PR interval | 188 | ms |
| QRS duration | 152 | ms |
| QT/QTc | 472/486 | ms |
| P-R-T axes | 78 3 | 106 |

Normal sinus rhythm
Left bundle branch block
Abnormal ECG
When compared with ECG of 28-MAY-2003 06:36,

EKG #WD03029959

Technician: WW

# USE LEAD V1 for this technique

Referred by:

Confirmed By:

D.O.S.:

I aVR V1 V4

II aVL V2 V5

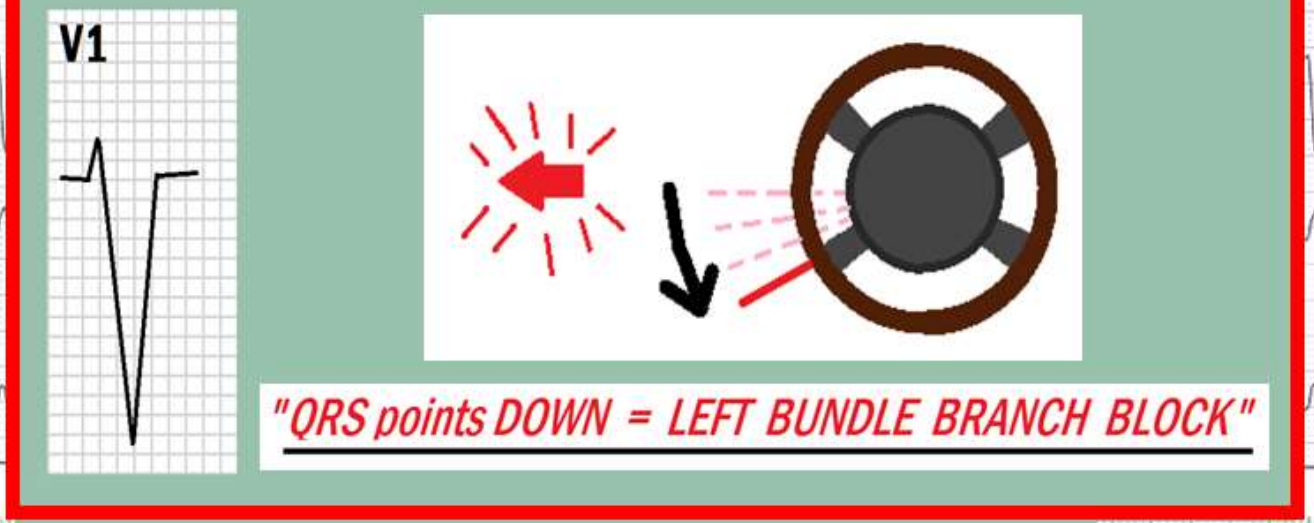
III aVF V3 V6

V1

II

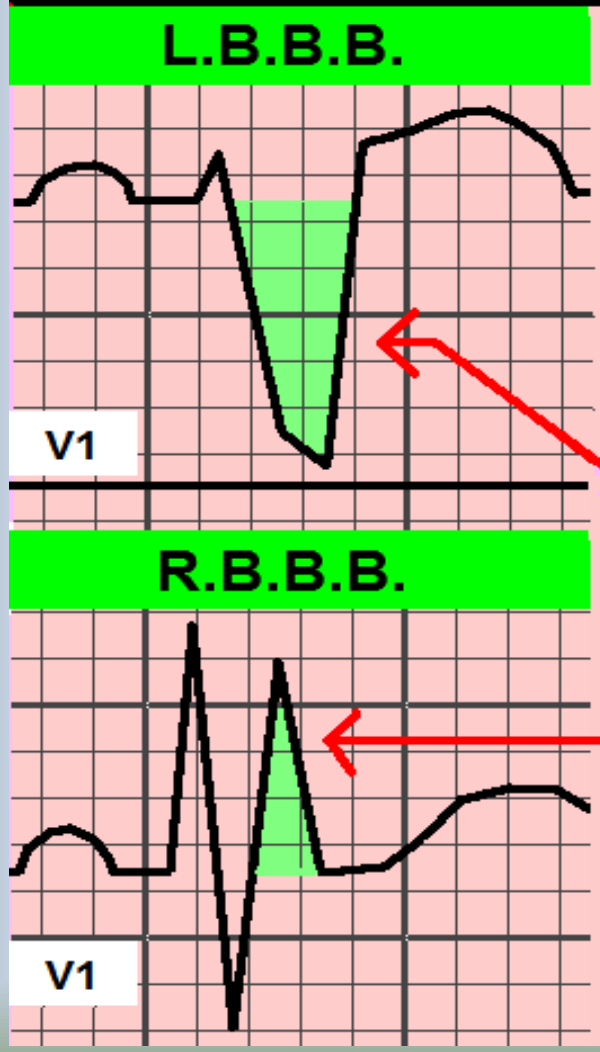
V5

25mm/s 10mm/mV



# “Terminal Phase of QRS Method”. . .

## DIAGNOSING BUNDLE BRANCH BLOCK




### USING LEAD V1

- QRS WIDER THAN 120 ms
- BEAT IS SUPRAVENTRICULAR IN ORIGIN
- TERMINAL PHASE OF QRS COMPLEX (LAST DEFLECTION)

NEGATIVE = LEFT BUNDLE BRANCH BLOCK

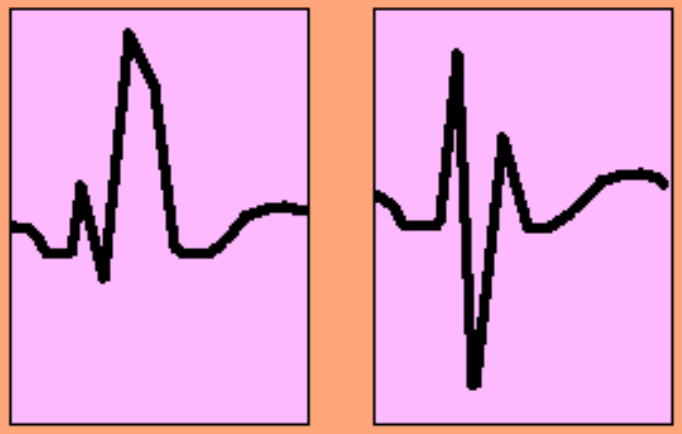
POSITIVE = RIGHT BUNDLE BRANCH BLOCK

# DIAGNOSING BUNDLE BRANCH BLOCK

## USING LEADS V1, V2, and V5, V6:

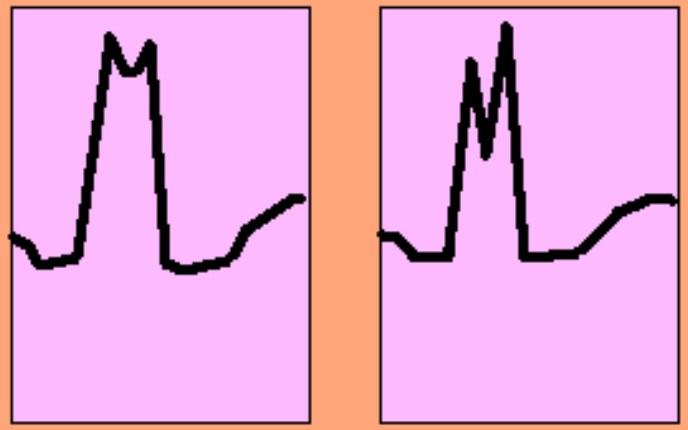
### LOCATING RsR' or RR' COMPLEXES:

V1 V2



RIGHT BUNDLE BRANCH BLOCK

V5 V6



LEFT BUNDLE BRANCH BLOCK

# THE QRS COMPLEX

## QRS HEIGHT

is a reflection of the QRS AMPLITUDE.

The NORMAL QRS AMPLITUDE varies from one lead to another . . .

# THE QRS COMPLEX

## QRS AMPLITUDE

is influenced by:

- age
- physical fitness
- body size
- conduction system disorders
- chamber hypertrophy

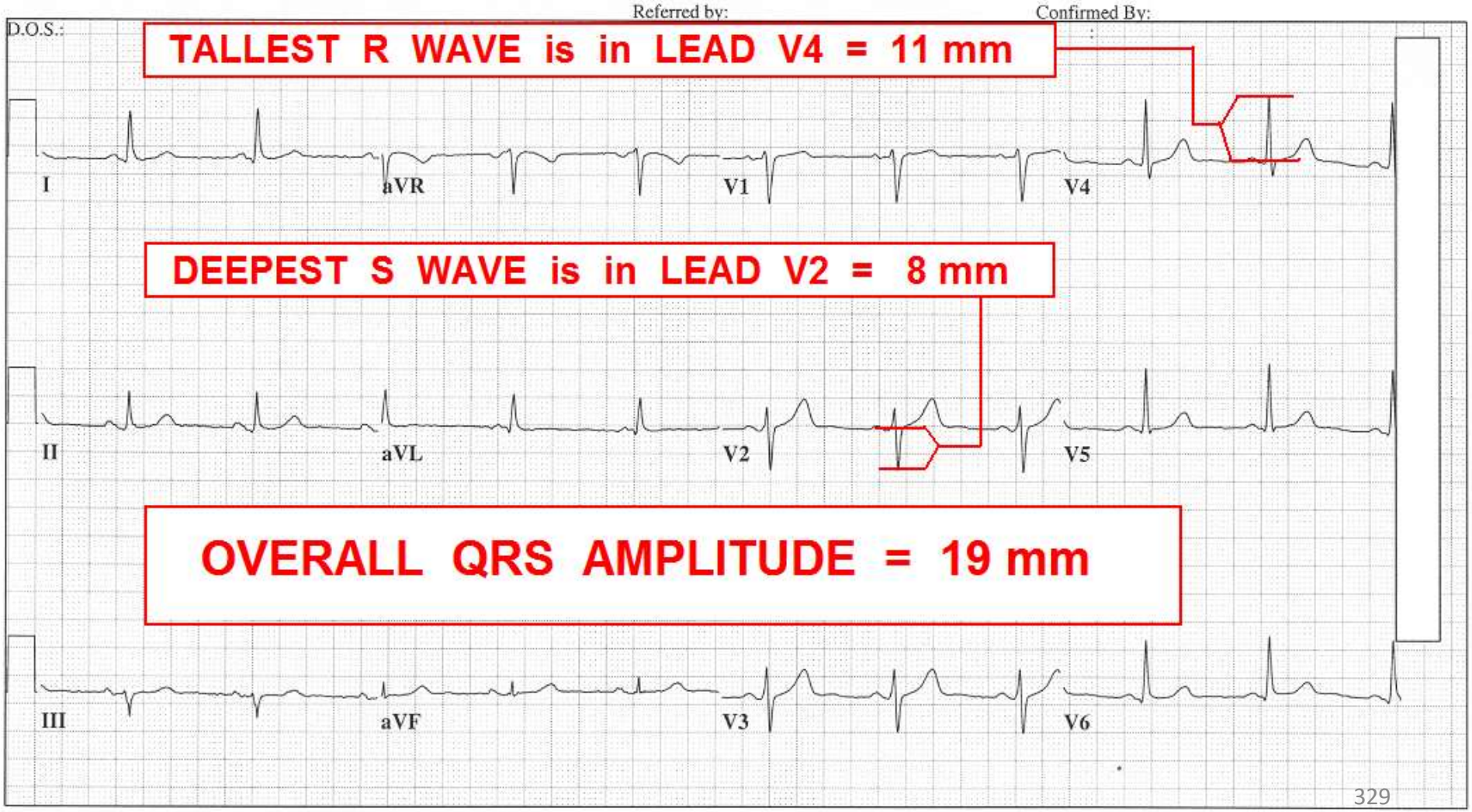
# THE QRS COMPLEX

## QRS AMPLITUDE

is measured by finding the TALLEST POSITIVE DEFLECTION ( R WAVE ) and the DEEPEST NEGATIVE DEFLECTION ( S WAVE ) on the 12 LEAD EKG and ADDING THE VALUES TOGETHER

# MEASURING THE "OVERALL QRS AMPLITUDE"

**Add the SIZE of the TALLEST R WAVE to the SIZE of the DEEPEST S WAVE . . . .**



# THE QRS COMPLEX

## QRS AMPLITUDE

**MAXIMUM NORMAL VALUES are difficult to define due to differences in PATIENT AGE, BODY SIZE, and FITNESS.**

**HOWEVER A GENERAL VALUE GUIDELINE IS: 3.0 mV ( 30 mm on normally calibrated EKG )**

# OVERALL QRS AMPLITUDE TOO HIGH:

## ( GREATER THAN 3.0 mV / 30 mm )

## THINK:

- VENTRICULAR HYPERTROPHY

# Hypertrophy “Cheats”:

- **WHEN QRS COMPLEX(ES) “SPEAR” OUTSIDE OF THEIR SPACE.**
- **WHEN QRS COMPLEXES SPEAR THROUGH OTHER LEADS ! . . . . . .**

14–JUL–1997 14:30:58 ST. JOSEPH'S HOSPITAL–ER ROUTINE RETRIEVAL

17 yr
Male Black

Room:ER
Loc:3 Option:16

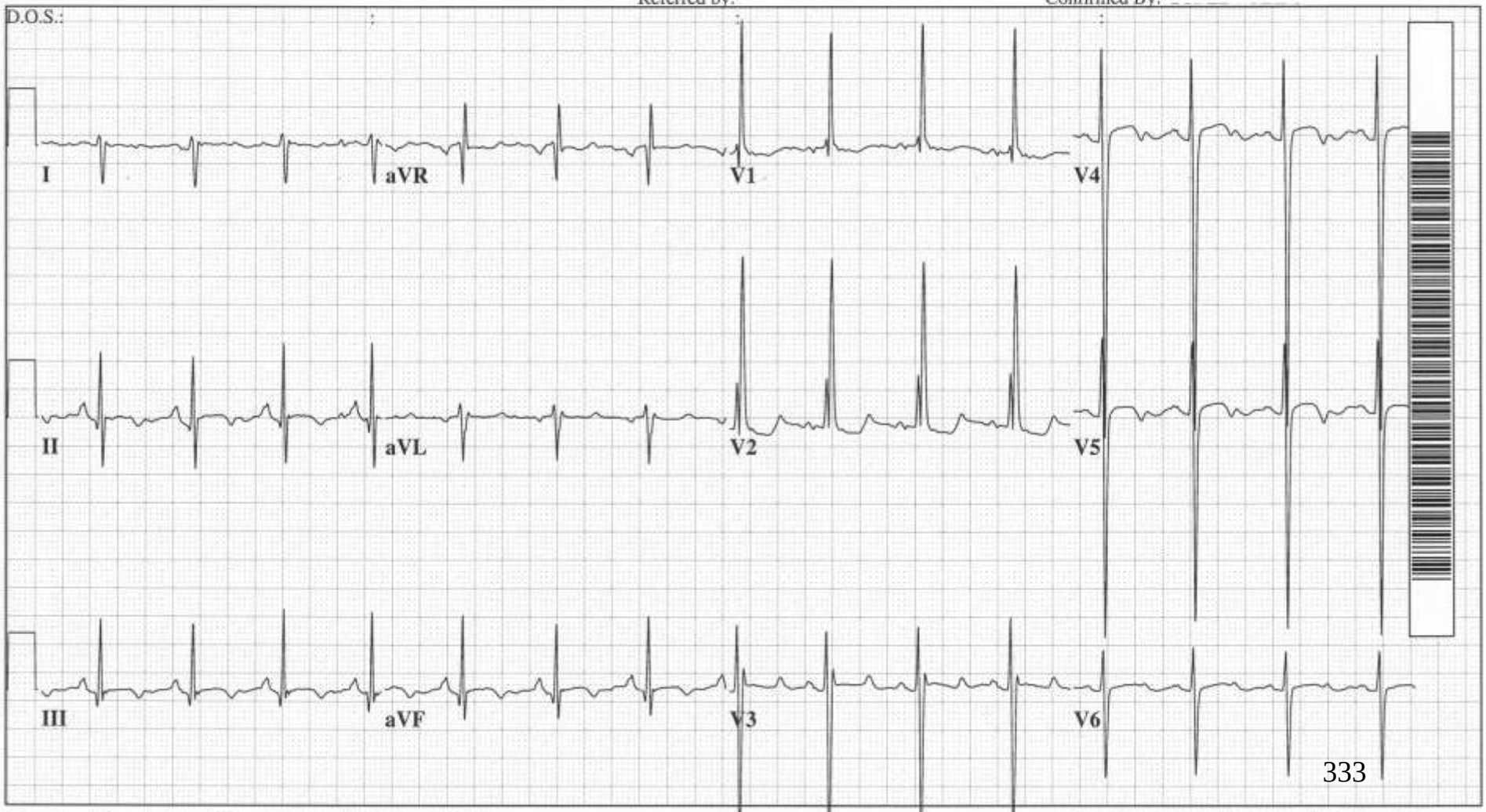
| | | |
|---|---|---|
| Vent. rate | 90 | BPM |
| PR interval | 136 | ms |
| QRS duration | 94 | ms |
| QT/QTc | 378/462 | ms |
| P–R–T axes | 77 123 | 58 |

Normal sinus rhythm
Right atrial enlargement
Right axis deviation
Incomplete right bundle branch block , plus right ventricular hypertrophy
NORMAL SINUS INFERIOR LATERAL CHANGES
Abnormal ECG

**EKG CLASS #WRO3616941**

Referred by: Confirmed By:

D.O.S.:




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

EID:11 EDT: 17:04 15–JUL–1997 ORDER:

19-JUN-1997 22:28:08 ST. JOSEPH'S HOSPITAL-ER ROUTINE RETRIEVAL

53 yr
Male Caucasian

Room:ER S3
Loc:3 Option:18

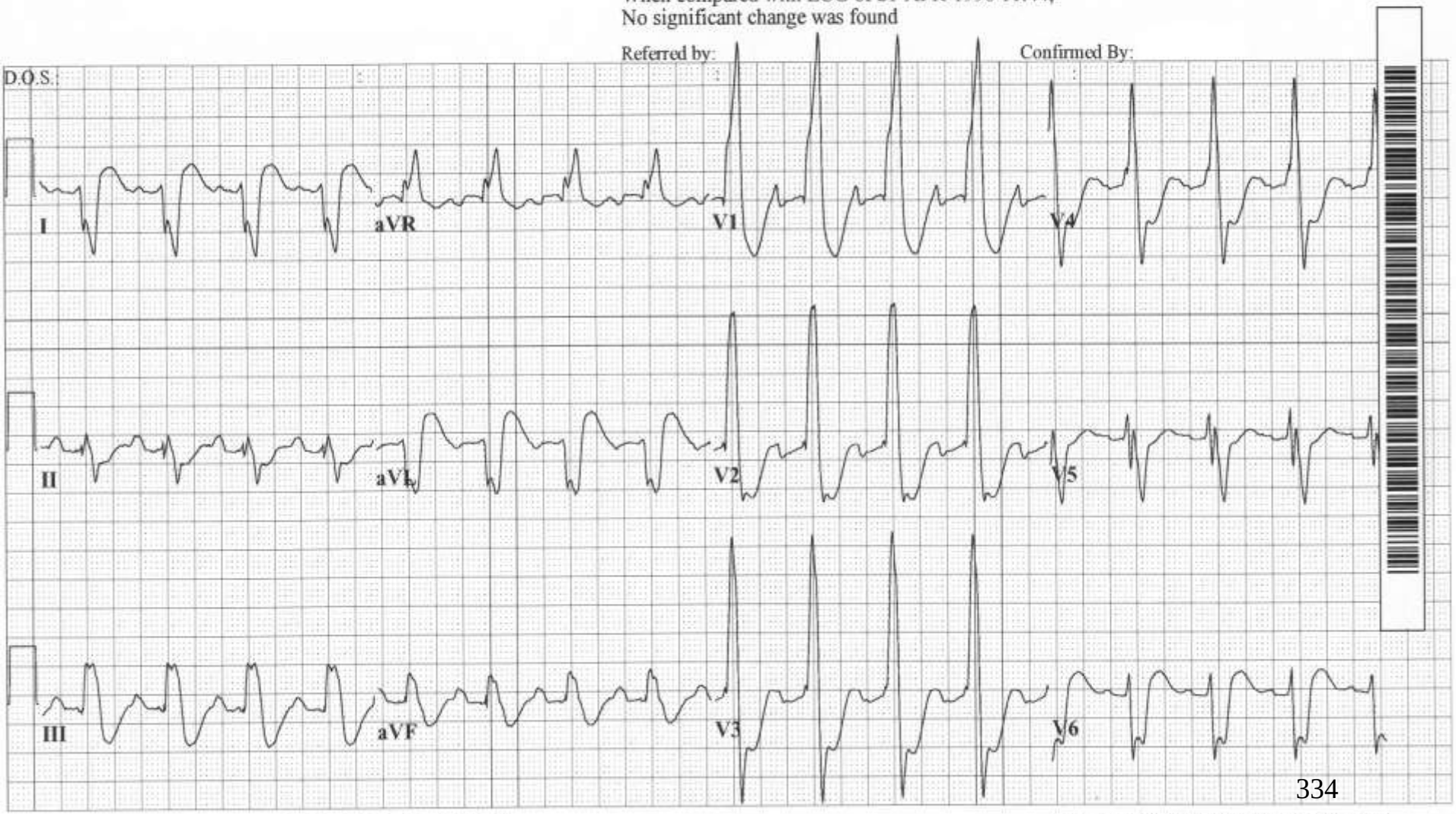
| | | |
|---|---|---|
| Vent. rate | 100 | BPM |
| PR interval | 198 | ms |
| QRS duration | 186 | ms |
| QT/QTc | 380/490 | ms |
| P-R-T axes | 79 163 -20 | |

Normal sinus rhythm
Left atrial enlargement
Right bundle branch block , plus right ventricular hypertrophy
Left posterior fascicular block
*** Bifascicular block ***
NONSPECIFIC ST CHANGES
Abnormal ECG
When compared with ECG of 21-APR-1996 11:44,
No significant change was found

**EKG CLASS #WR03028722**

Referred by: Confirmed By:

D.O.S.:






25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 10

EID:13 EDT: 23:01 20-JUN-1997 ORDER:

18–NOV–2002 02:15:46 ST. JOSEPH'S HOSPITAL–ER 1ST PREVIOUS

53 yr
Male Black

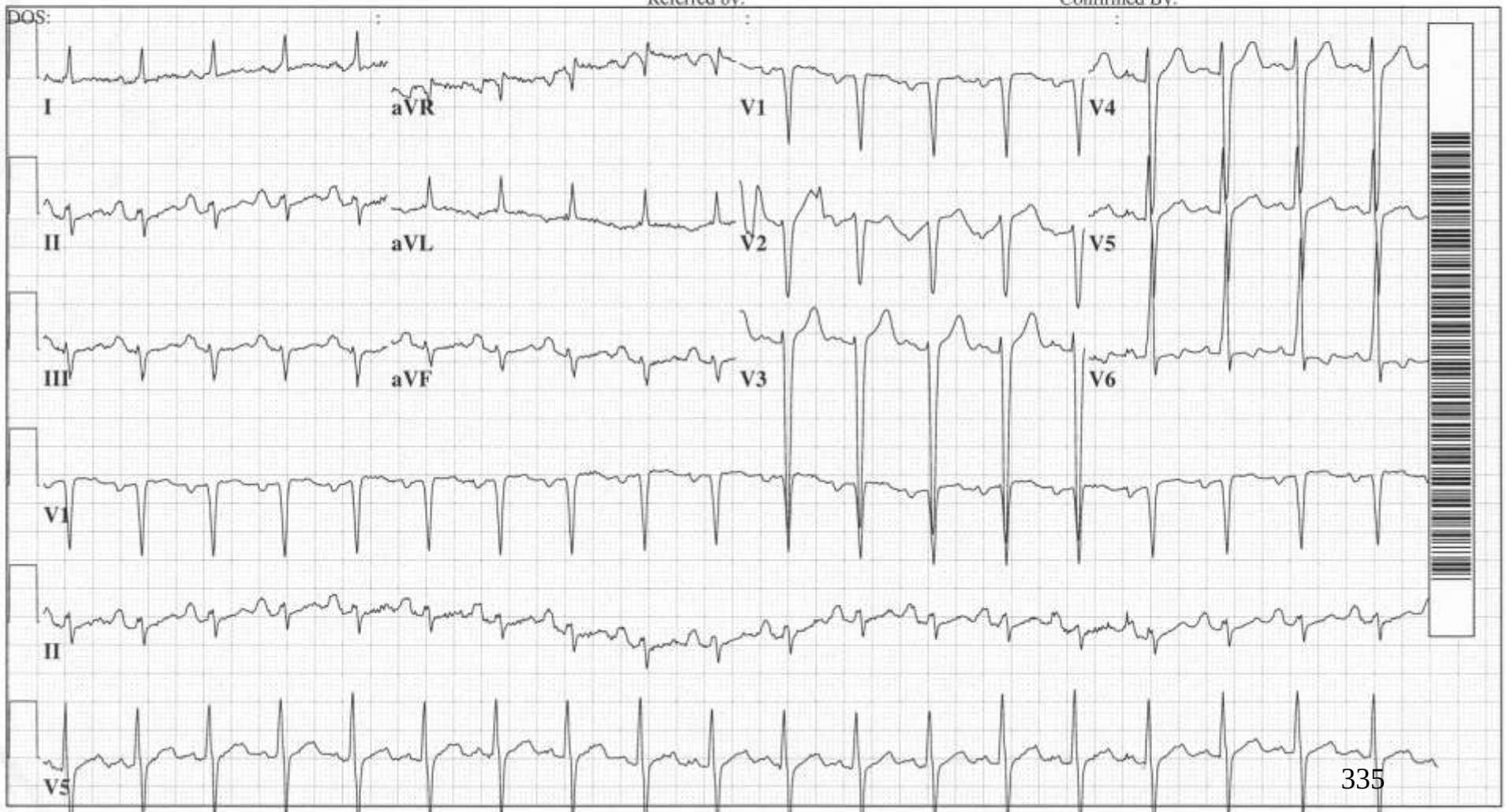
Room:ER
Loc:3 Option:23

| | | |
|---|---|---|
| Vent. rate | 115 | BPM |
| PR interval | 160 | ms |
| QRS duration | 92 | ms |
| QT/QTc | 316/437 | ms |
| P–R–T axes | 76 –39 | 59 |

**UNEDITED COPY – REPORT IS COMPUTER GENERATED ONLY, WITHOUT PHYSICIAN INTERPRETATION
Sinus tachycardia
Possible Left atrial enlargement
Left axis deviation
Left ventricular hypertrophy
Abnormal ECG
No previous ECGs available

**EKG CLASS #WR03896717**

Referred by: Confirmed By:




25mm/s 10mm/mV 40Hz 005C I2SL 233 CID: 7 EID:28 EDT: 11:27 18–NOV–2002 ORDER:

10–APR–2003 11:49:36 ST. JOSEPH'S HOSPITAL–CCR ROUTINE RETRIEVAL

61 yr
Male Black

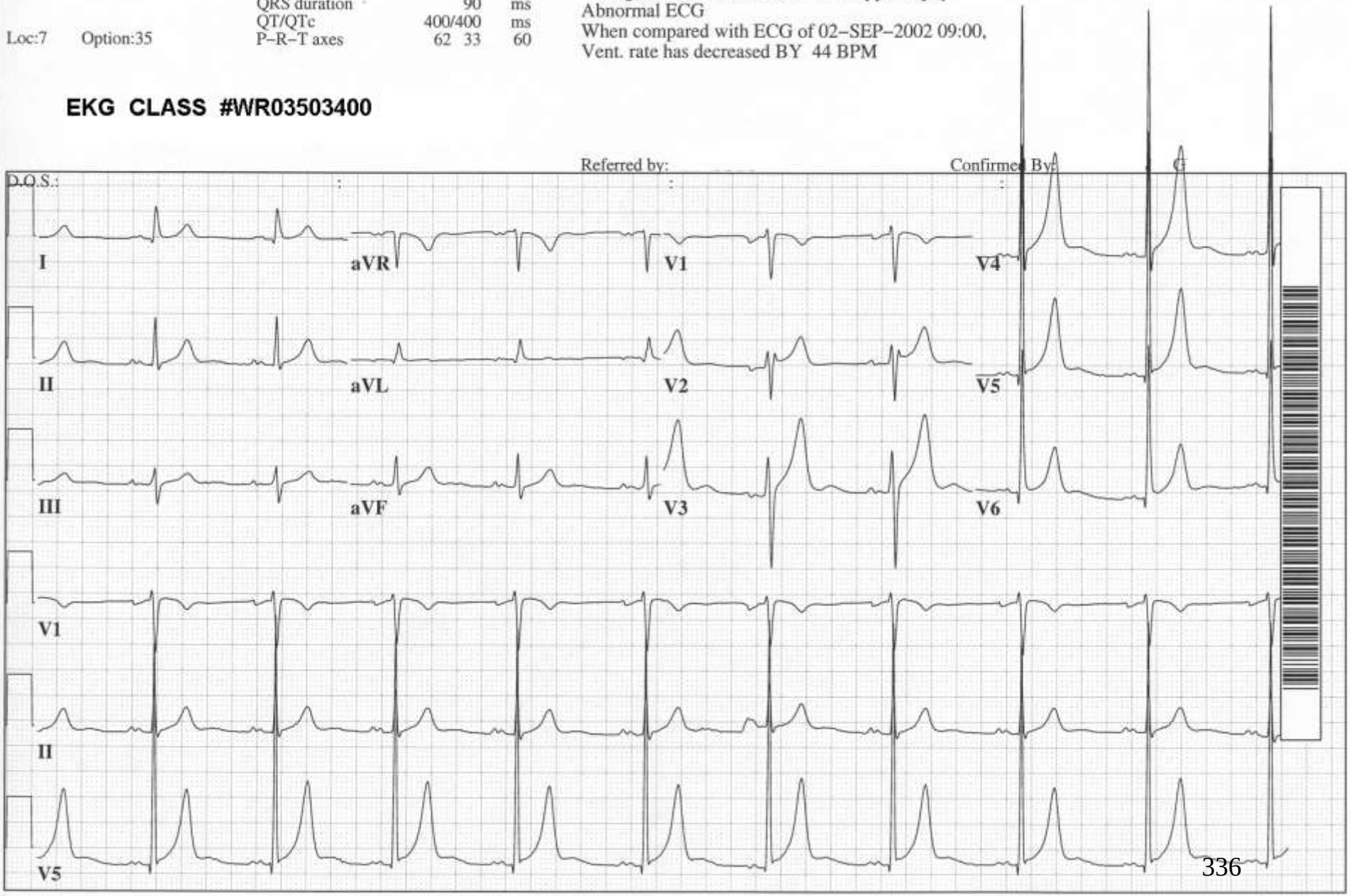
Loc:7 Option:35

| | | |
|---|---|---|
| Vent. rate | 60 | BPM |
| PR interval | 176 | ms |
| QRS duration | 90 | ms |
| QT/QTc | 400/400 | ms |
| P–R–T axes | 62 33 | 60 |

Normal sinus rhythm
Voltage criteria for left ventricular hypertrophy
Abnormal ECG
When compared with ECG of 02–SEP–2002 09:00,
Vent. rate has decreased BY 44 BPM

**EKG CLASS #WR03503400**

Referred by: Confirmed By:






25mm/s 10mm/mV 40Hz 005C 12SL 229 CID: 0

EID:28 EDT: 10:43 14–APR–2003 ORDER:

# THE QRS COMPLEX

## QRS AMPLITUDE

**CRITERIA FOR MINIMUM AMPLITUDE:**

**Abnormally LOW QRS VOLTAGE occurs when the OVERALL QRS is:**

**≤ 0.5 mV IN ANY LIMB LEAD**

**— *and* —**

**≤ 1.0 mV IN ANY PRECORDIAL LEAD**

# OVERALL QRS AMPLITUDE TOO LOW:

## ( VERTICAL QRS SIZE )

## THINK ( in absence of obvious OBESITY ) :

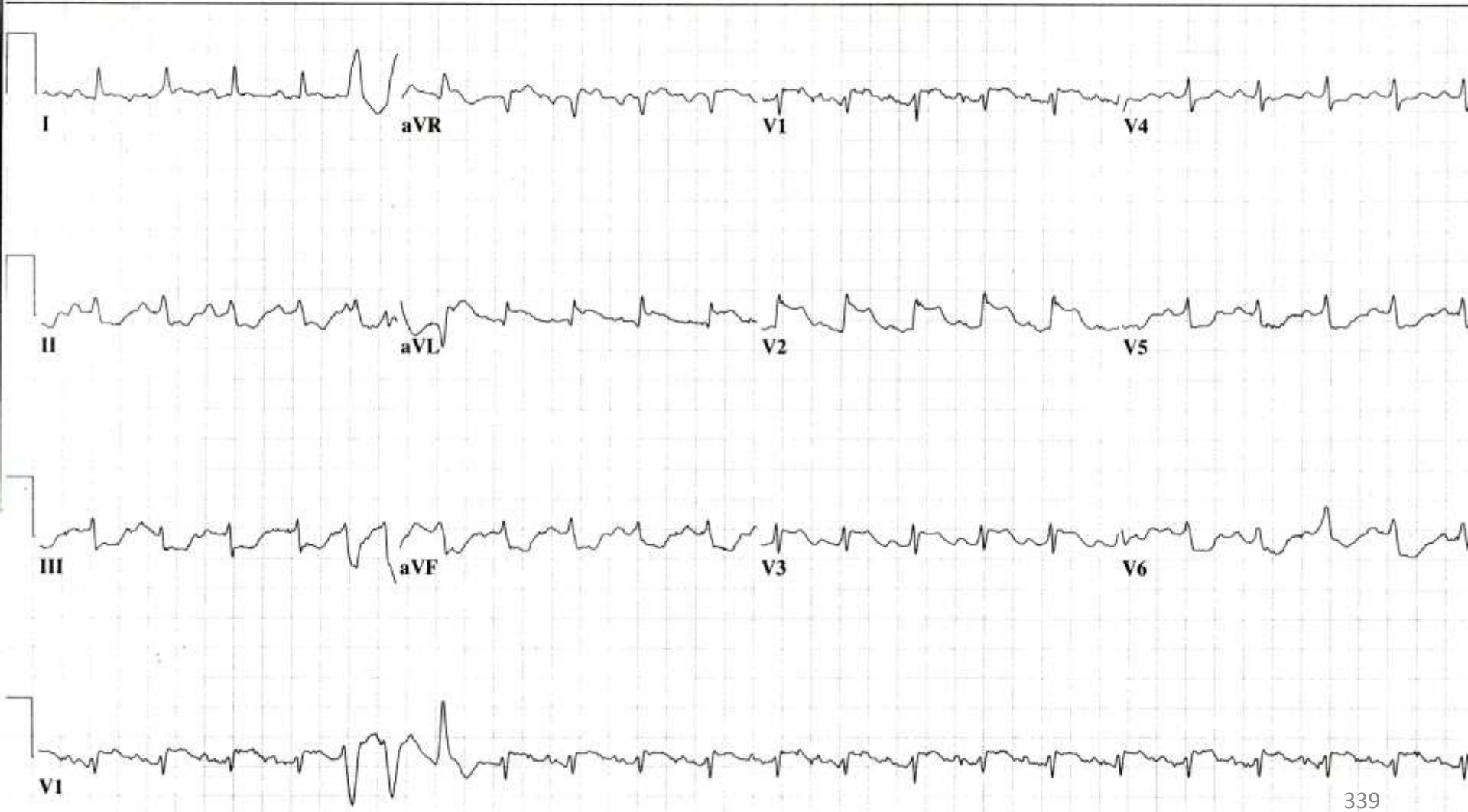
- **MYOCARDITIS / CONSTRICTIVE PERICARDITIS**
- **EFFUSIONS / TAMPONADE**
- **COPD c HYPERINFLATION**
- **AMYLOIDOSIS** ( abnormal protein accumulation in organs )
- **SCLERODERMA** ( abnormal hardening of skin )
- **HEMACHROMOTOSIS** ( excessive iron buildup in blood / organs )
- **MYXEDEMA** ( thyroid disorder )

33 yr
Female Black

Room:ATL
Loc:3 Option:23

| | | |
|---|---|---|
| Vent. rate | 132 | BPM |
| PR interval | 154 | ms |
| QRS duration | 76 | ms |
| QT/QTc | 282/417 | ms |
| P-R-T axes | 51 17 | -80 |

"**unedited copy: report is computer generated only, without physician interpretation".
*** Age and gender specific ECG analysis ***
Sinus tachycardia with occasional , and consecutive
Premature ventricular complexes
Low voltage QRS
ST elevation consider anterolateral injury or acute infarct
*** ** ** ** * ACUTE MI ** ** ** **
Abnormal ECG
No previous ECGs available

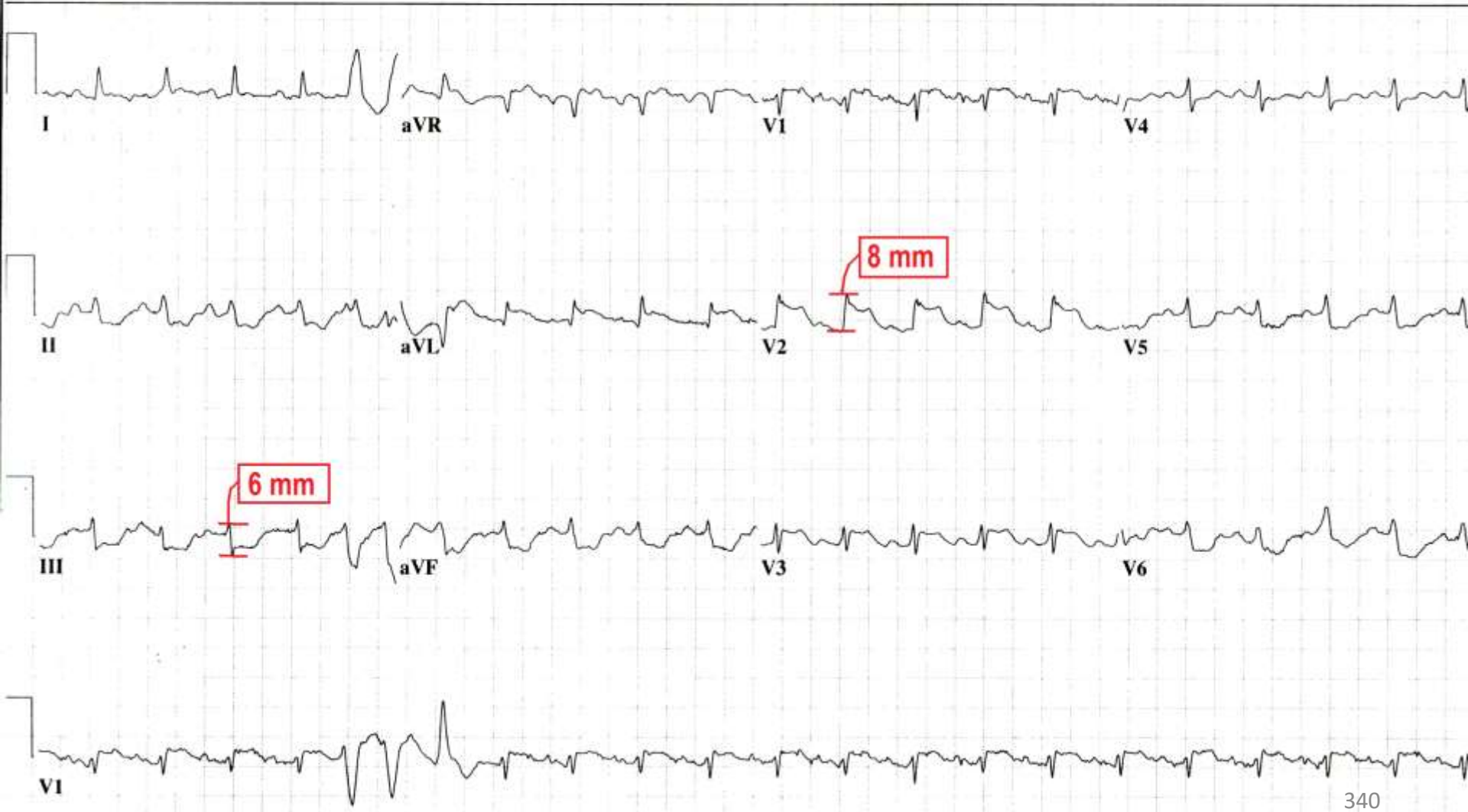


33 yr
Female Black

Room:ATL
Loc:3 Option:23

| | | |
|---|---|---|
| Vent. rate | 132 | BPM |
| PR interval | 154 | ms |
| QRS duration | 76 | ms |
| QT/QTc | 282/417 | ms |
| P-R-T axes | 51 17 | -80 |

"**unedited copy: report is computer generated only, without physician interpretation".
*** Age and gender specific ECG analysis ***
Sinus tachycardia with occasional , and consecutive
Premature ventricular complexes
Low voltage QRS
ST elevation consider anterolateral injury or acute infarct
*** ** ** ** * ACUTE MI ** ** ** **
Abnormal ECG
No previous ECGs available



# • Q WAVES •

**Normal Q Waves**

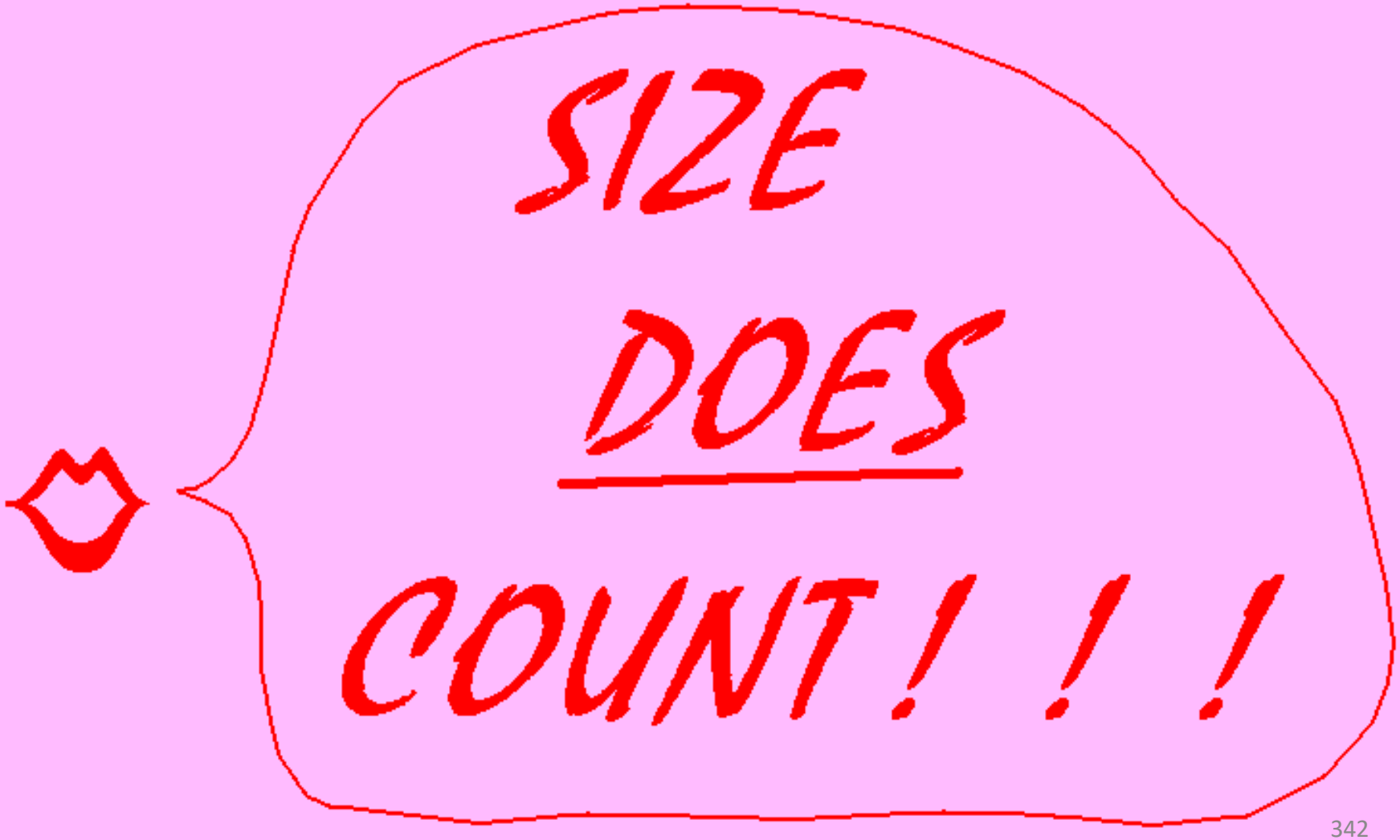
caused by depolarization of the intraventricular septum

**Abnormal Q Waves -**

caused by:

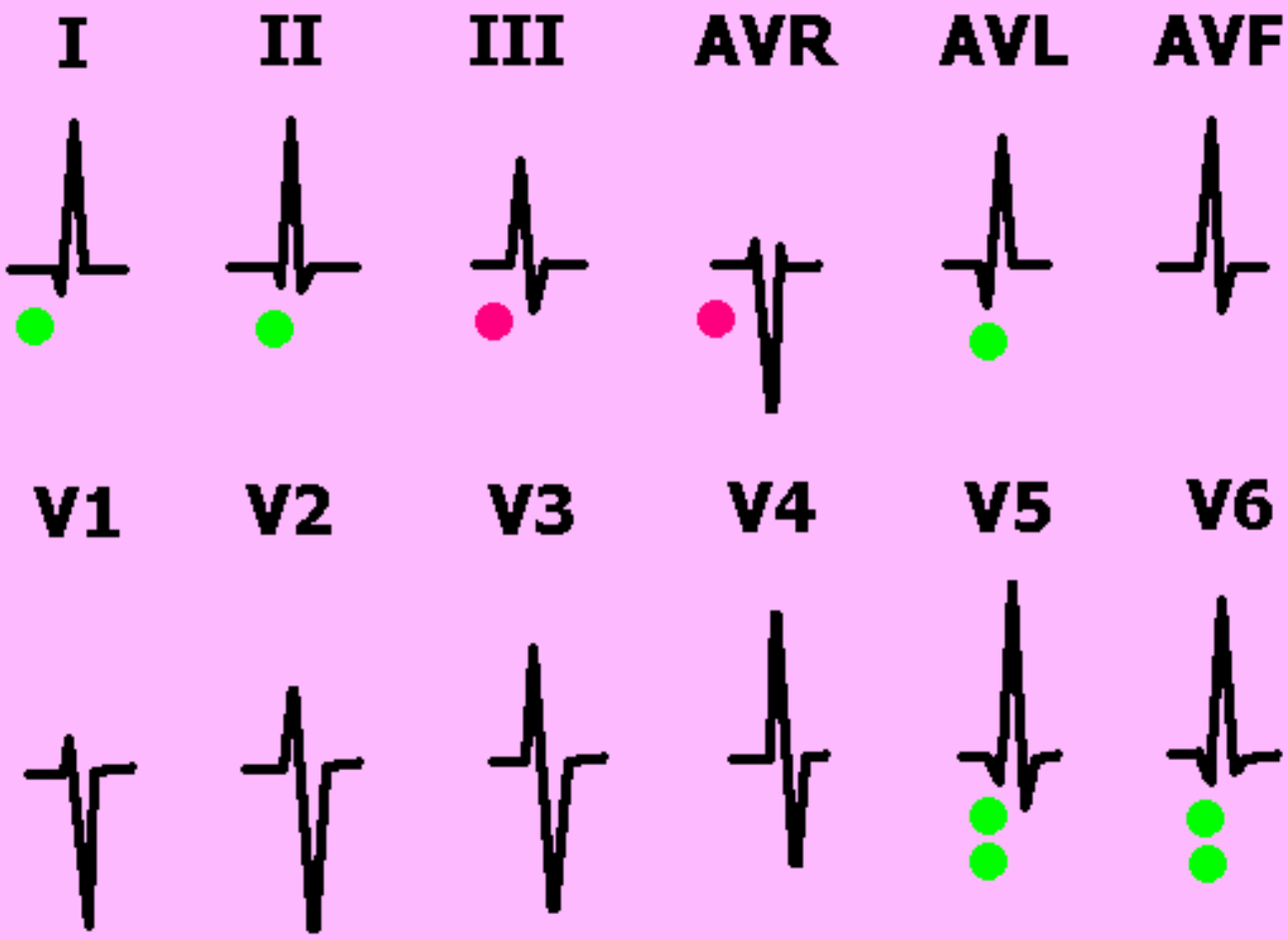
- necrosis ( old infarction )
- hypertrophy

# • Q WAVES •



# LEADS WHERE Q WAVES ARE NORMAL

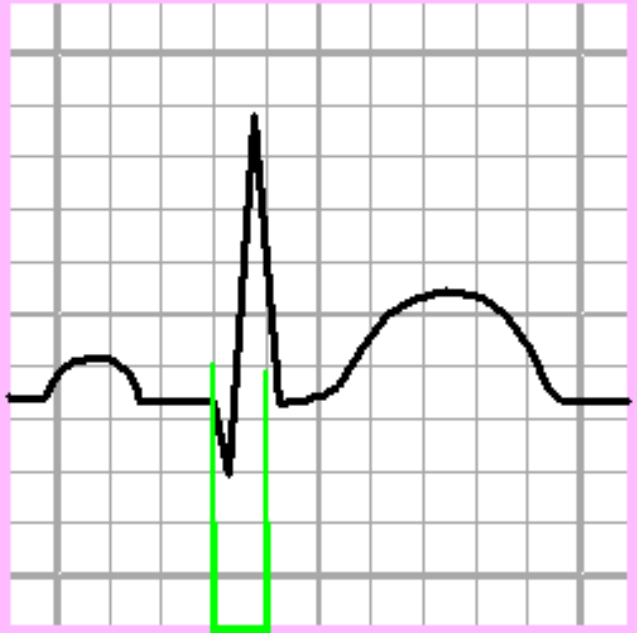
- Normal Q WAVES caused by SEPTAL DEPOLARIZATION




| | | |
|---|---|---|
| ● Q WAVES NORMAL AND FREQUENTLY SEEN | ● ● Q WAVES EXPECTED | ● Q WAVES, IF PRESENT, CAN NORMALLY BE ANY SIZE |

# THE QRS COMPLEX

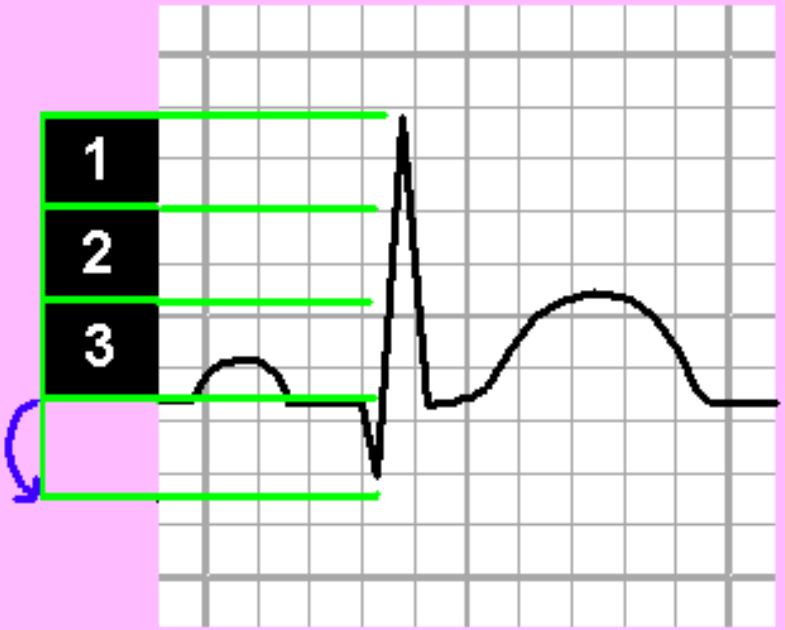
## GENERAL RULES FOR NORMAL Q WAVES - WIDTH



**LESS THAN .40 ( 1 mm ) WIDE**

# THE QRS COMPLEX

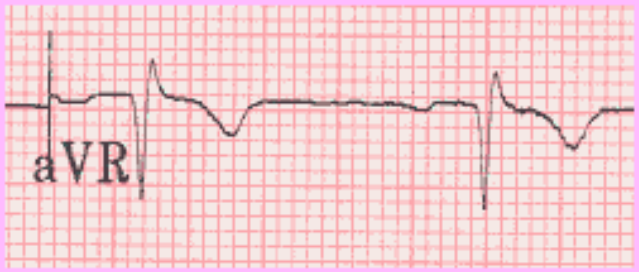
## GENERAL RULES FOR NORMAL Q WAVES - HEIGHT



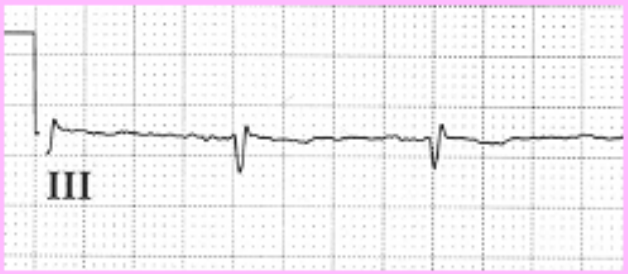

☞ LESS THAN 1/3 THE HEIGHT OF THE R WAVE

# THE QRS COMPLEX

## NORMAL Q WAVES
## EXCEPTIONS TO THE RULES




LEAD AVR



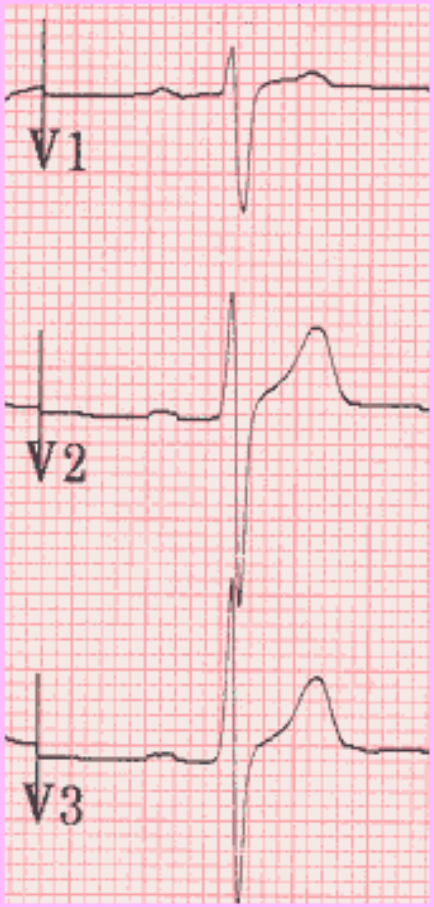

LEAD Ⅲ

☞ THE Q WAVE CAN BE ANY SIZE

# THE QRS COMPLEX

## NORMAL Q WAVES
## EXCEPTIONS TO THE RULES

☞ THERE SHOULD BE NO Q WAVES PRESENT IN LEADS: V1
V2
V3



# THE QRS COMPLEX

## Q WAVE RULES - SUMMARY:

- Q WAVES SHOULD BE LESS THAN .40 WIDE ( 1 mm )

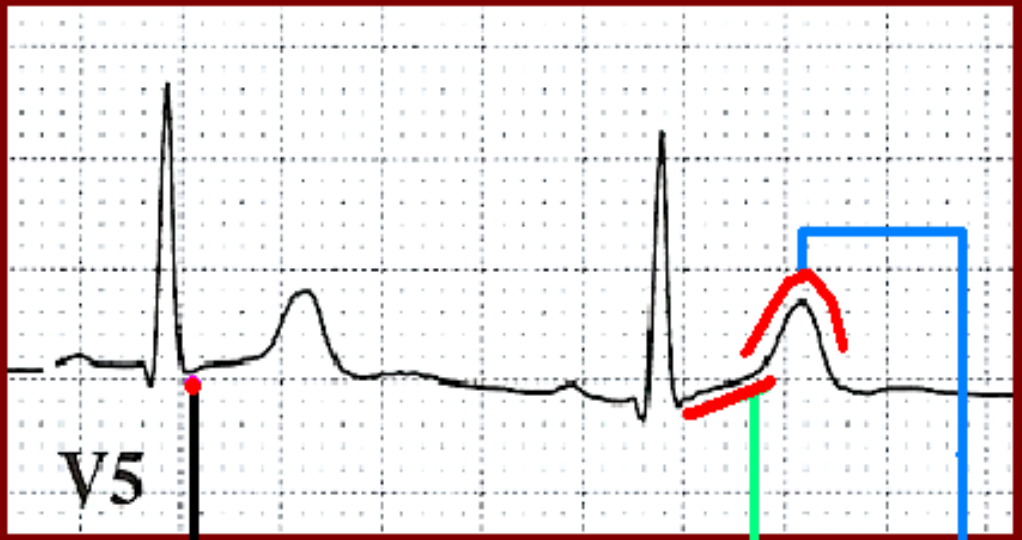
- Q WAVES SHOULD BE LESS THAN 1/3 THE HEIGHT OF THE R WAVE

- Q WAVES CAN BE ANY SIZE IN LEADS III and AVR

- THERE SHOULD BE NO Q WAVES IN LEADS V1, V2, or V3

# NORMAL ST - T WAVES

- WHEN QRS WIDTH IS NORMAL ( < 120 ms )

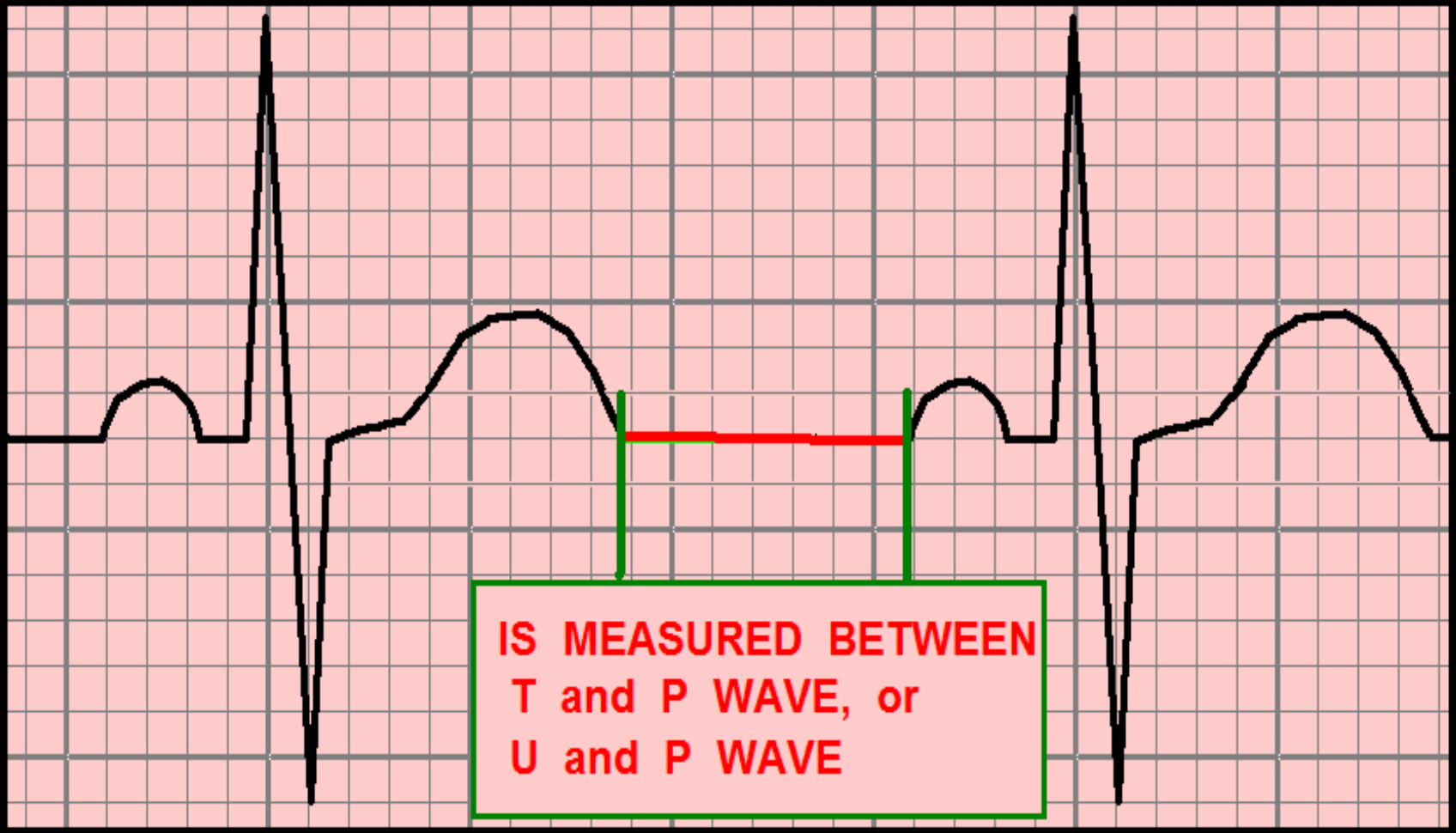



## *ASSESS:*

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

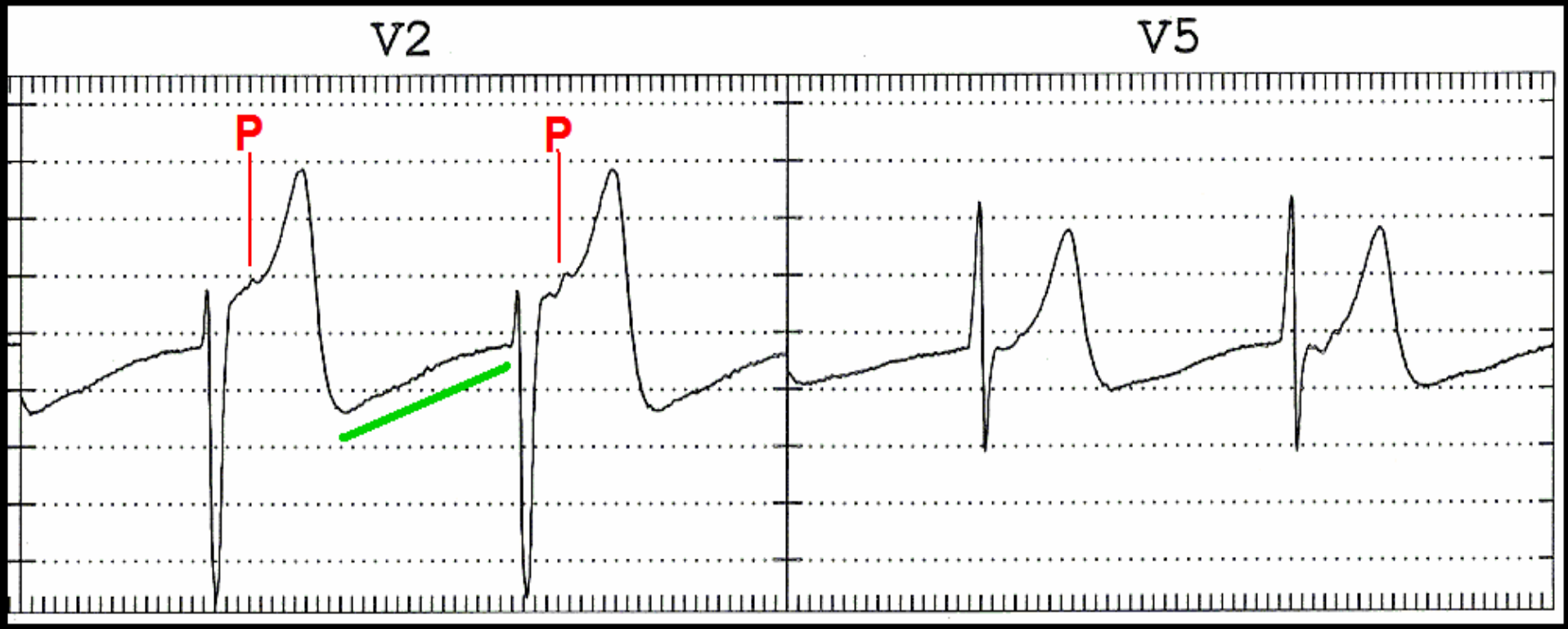
☞ *in EVERY LEAD EXCEPT aVR ! !*

# THE ISOELECTRIC LINE



# THE ISOELECTRIC LINE

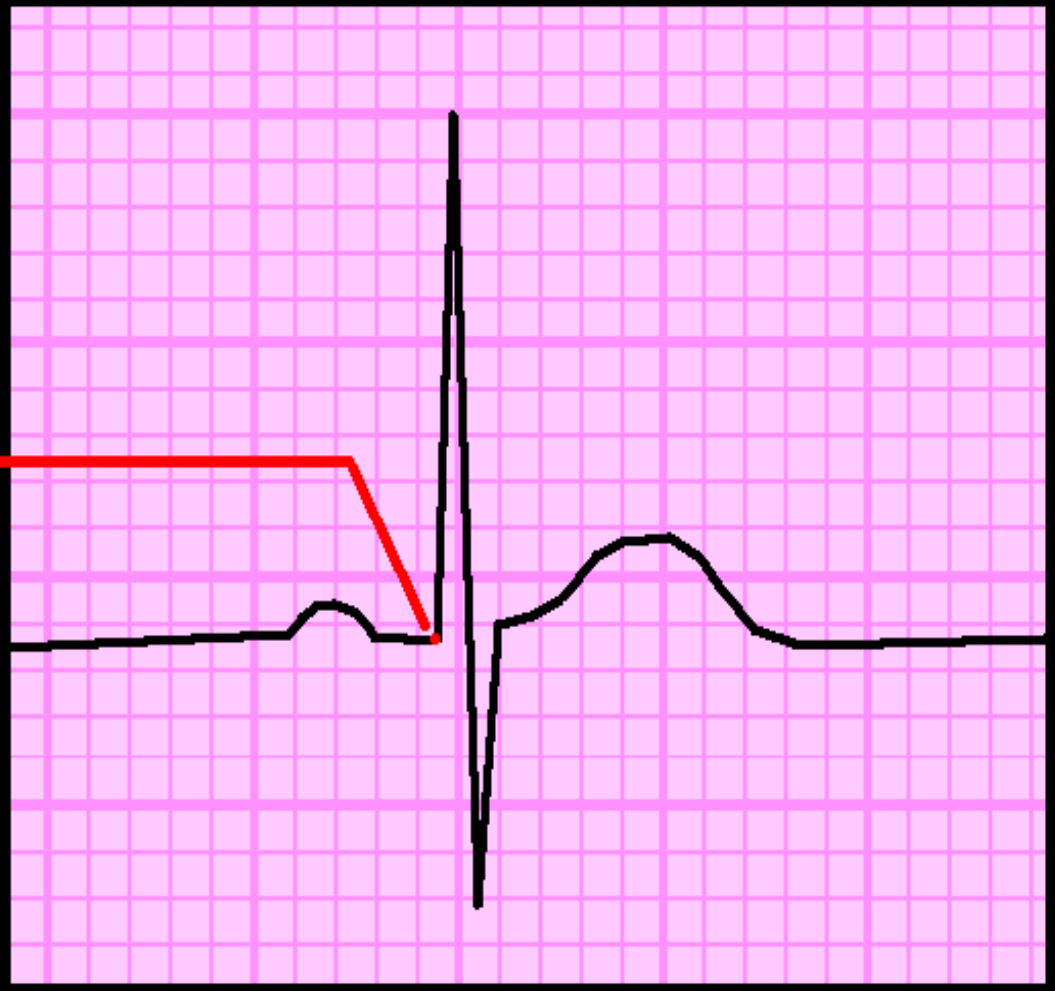
EKG from 13 y/o girl in ACCELERATED JUNCTIONAL RHYTHM. note: upsloping T-P interval, and P buried in T waves.



# THE P-Q JUNCTION

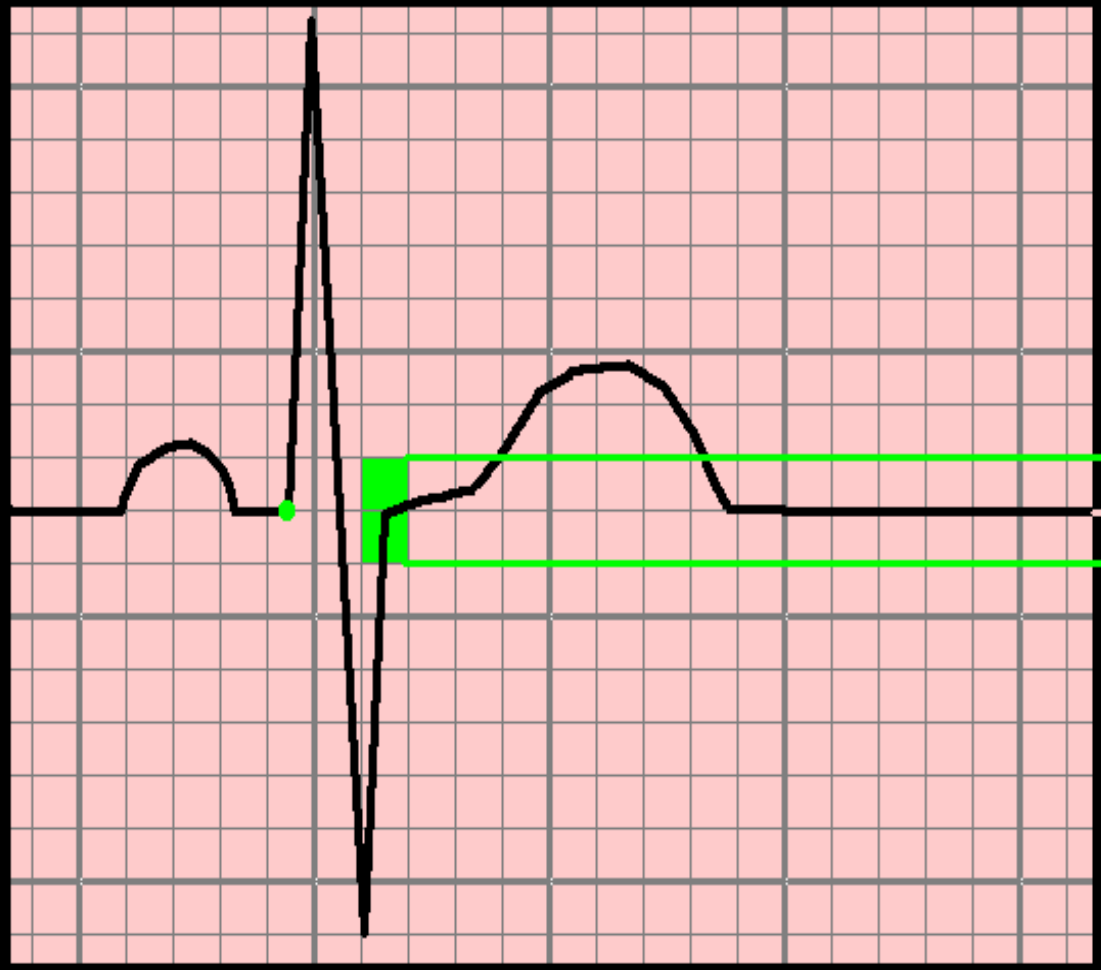
. . . is the POINT where the P-R SEGMENT ends and the QRS COMPLEX BEGINS.

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

# THE J POINT SHOULD BE . .

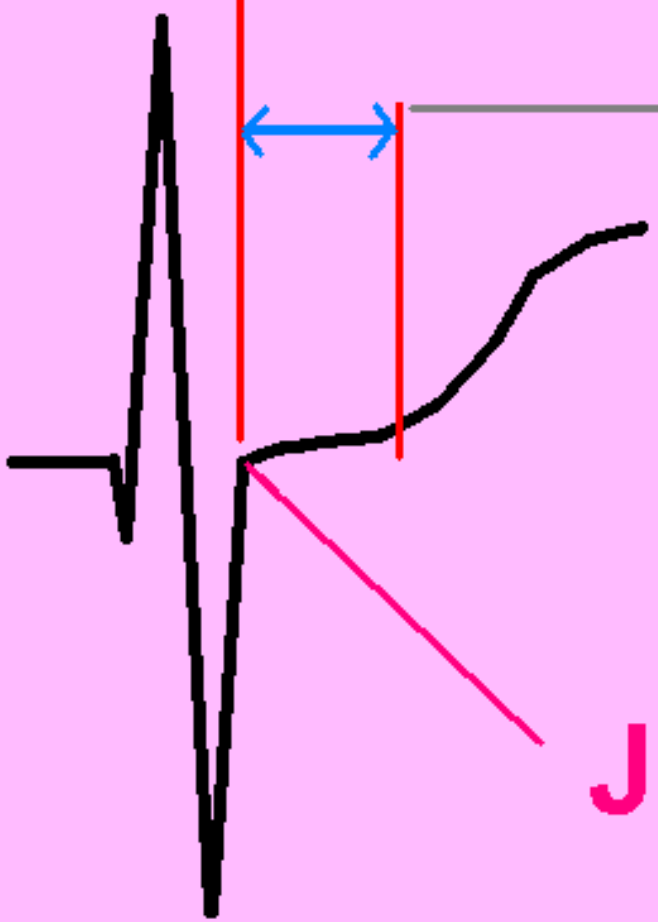


WITHIN
1 mm
ABOVE

OR

BELOW
THE
P-Q
JUNCTION

# THE S-T SEGMENT

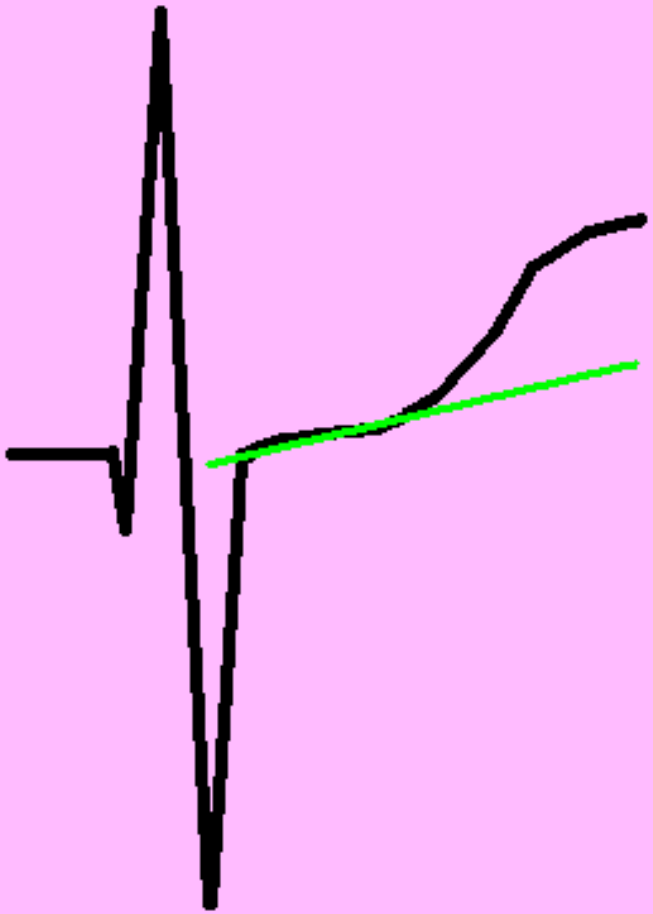


BEGINS AT J POINT

ENDS AT BEGINNING OF T WAVE

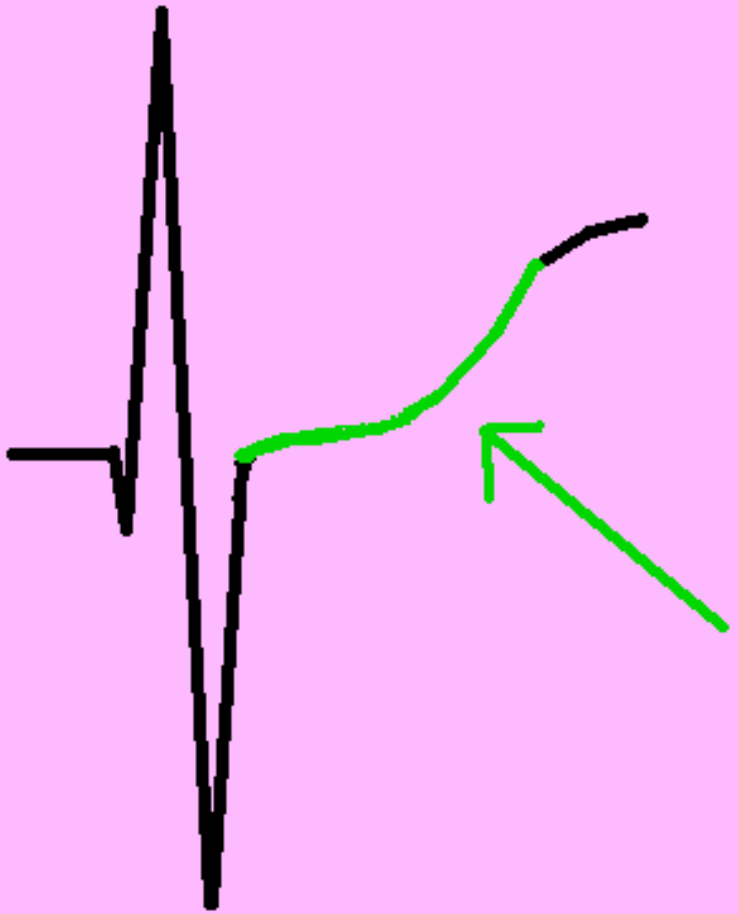
J POINT

# THE S-T SEGMENT



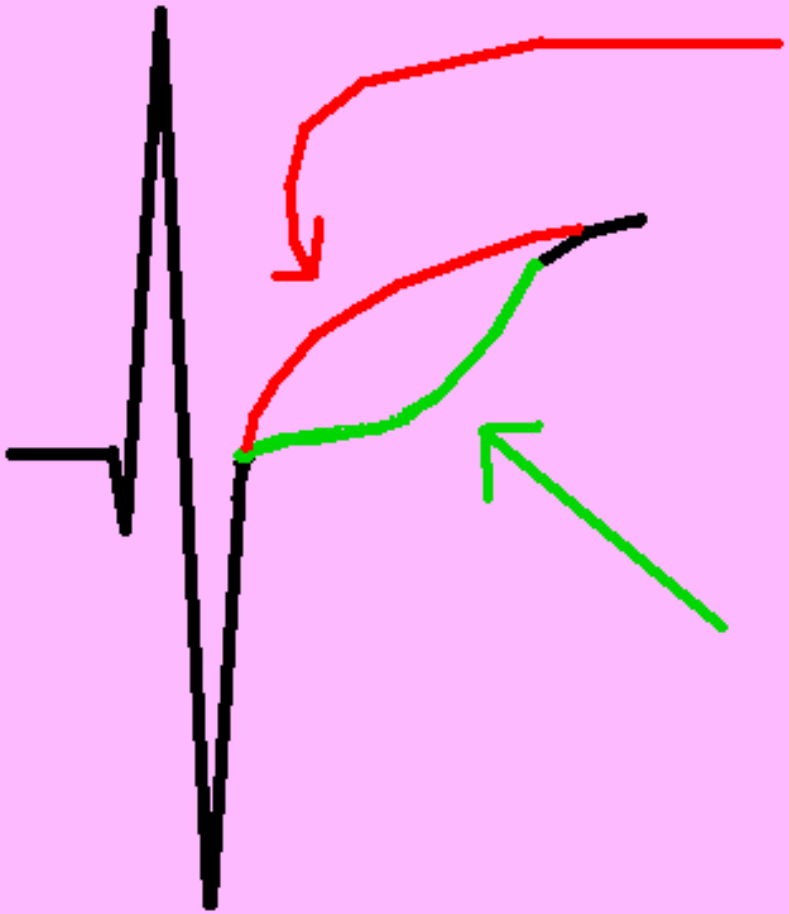
SHOULD HAVE
A "SLIGHT POSITIVE"
INCLINATION

# THE S-T SEGMENT



SHOULD BE "CONCAVE" IN SHAPE . . .

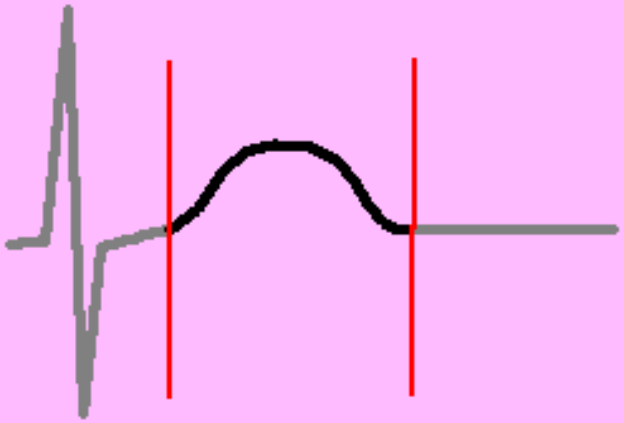
# THE S-T SEGMENT



AS OPPOSED TO "CONVEX" IN SHAPE

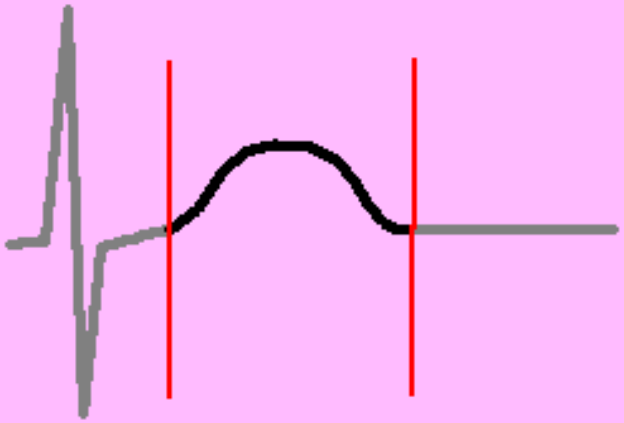
SHOULD BE "CONCAVE" IN SHAPE . . .

# THE T WAVE



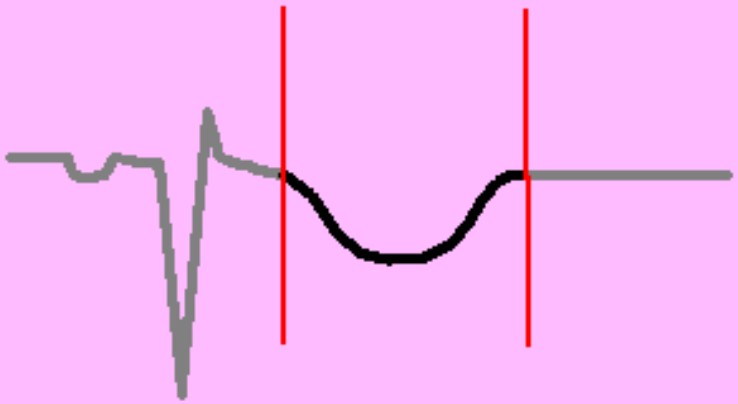
- SHOULD BE A "NICE," ROUNDED, CONVEX SHAPE
- SHOULD BE SYMMETRICAL

# THE T WAVE



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

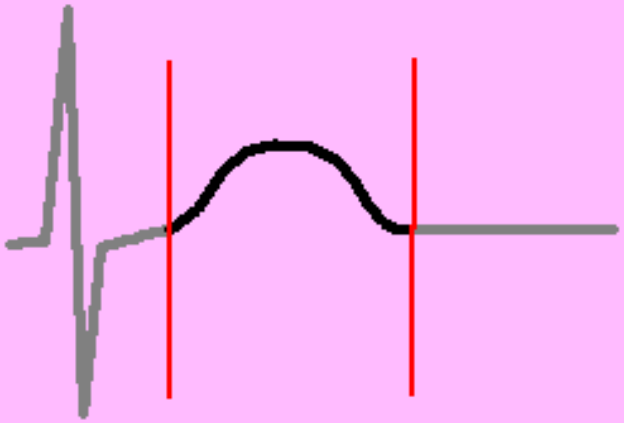
# THE T WAVE



## LEAD AVR

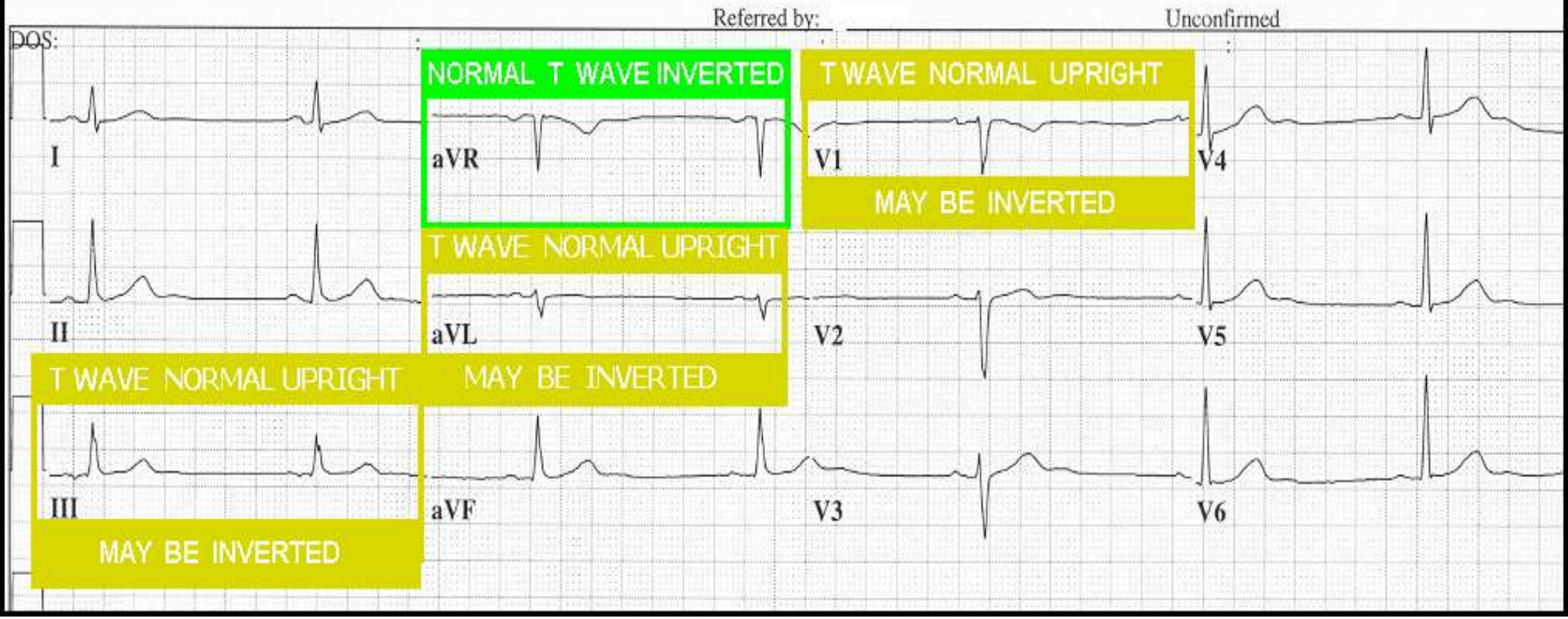
- REMEMBER, IN LEAD AVR
*EVERYTHING*
IS
"UPSIDE-DOWN"

# THE T WAVE



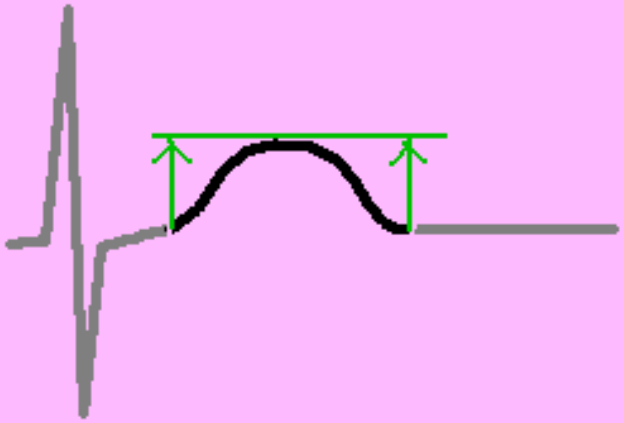
- SHOULD BE A "NICE," ROUNDED, CONVEX SHAPE
- SHOULD BE SYMMETRICAL
- SHOULD BE UPRIGHT IN ALL LEADS, EXCEPT AVR
- MAY BE INVERTED IN LEADS I, III, and V1

# Leads where the T WAVE may be INVERTED:




An inverted T wave in TWO OR MORE CONTIGUOUS LEADS = potential problem ( ischemia )

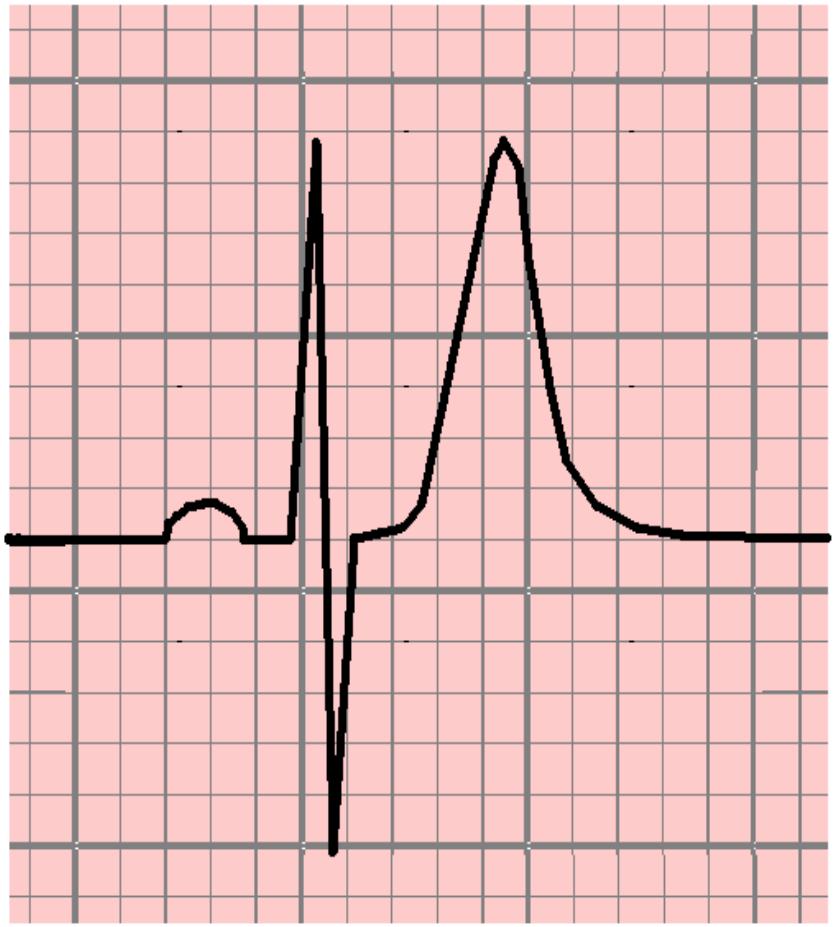
# THE T WAVE



## 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.

# HYPER-ACUTE T WAVES - COMMON ETIOLOGIES:

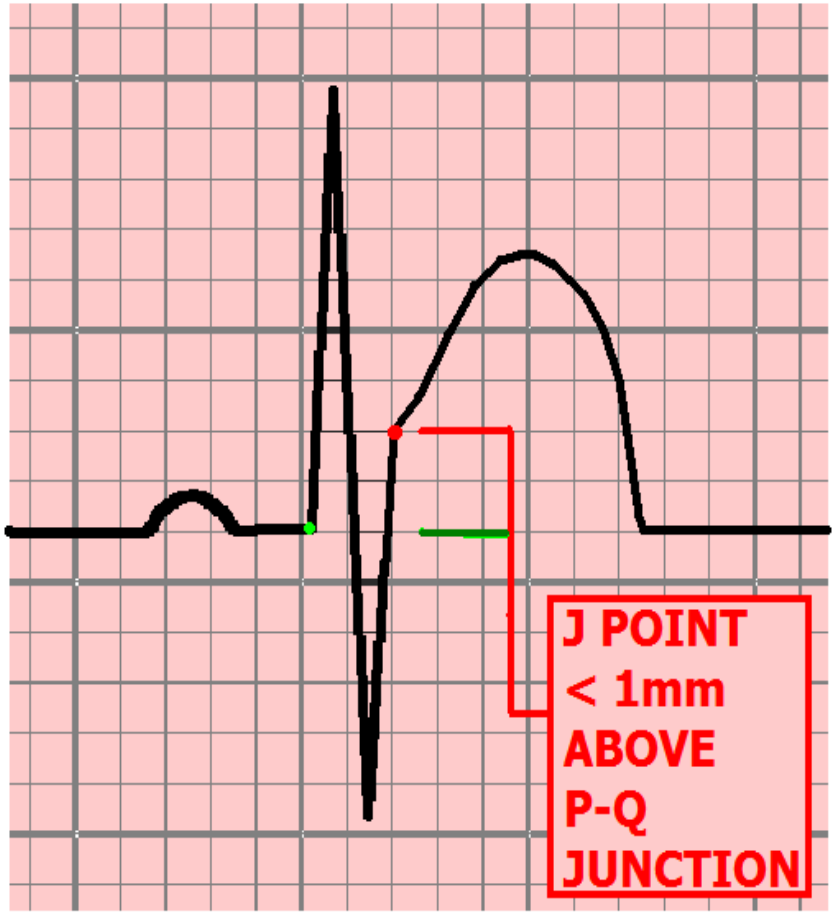


CONDITION:

- HYPERKALEMIA
- ACUTE MI
- TRANSMURAL ISCHEMIA
- HYPERTROPHY

*MORE INFORMATION ON HYPERACUTE T WAVES COMING UP SOON . . .*

# S-T SEGMENT ELEVATION - COMMON ETIOLOGIES:




CONDITION:

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

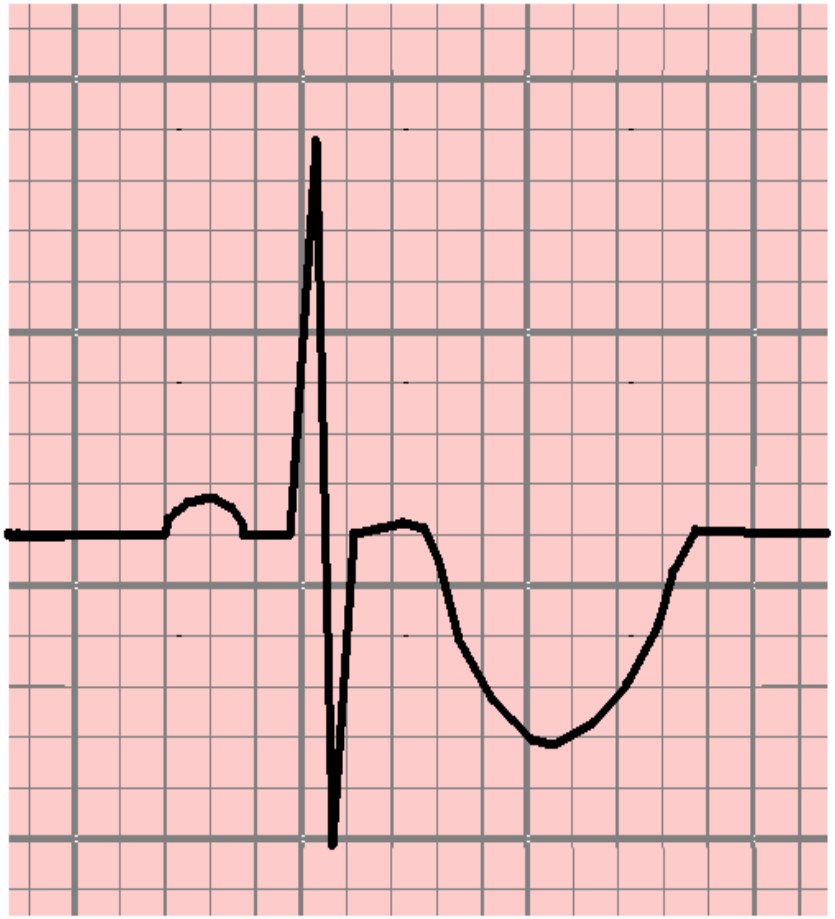
ON THE NEXT PAGE IN YOUR BOOK ARE SOME EXAMPLES OF THE ABOVE CONDITIONS

# S-T SEGMENT DEPRESSION - COMMON ETIOLOGIES:


CONDITION:

- RECIPROCAL CHANGES of ACUTE MI
- NON-Q WAVE M.I. ( NON-STEMI )
- ISCHEMIA
- POSITIVE STRESS TEST
- VENTRICULAR HYPERTROPHY (STRAIN PATTERN)
- WOLFF-PARKINSON-WHITE
- OLD MI ( NECROSIS vs. ISCHEMIA )
- DIGITALIS
- R. BUNDLE BRANCH BLOCK

# T WAVE INVERSION - COMMON ETIOLOGIES:



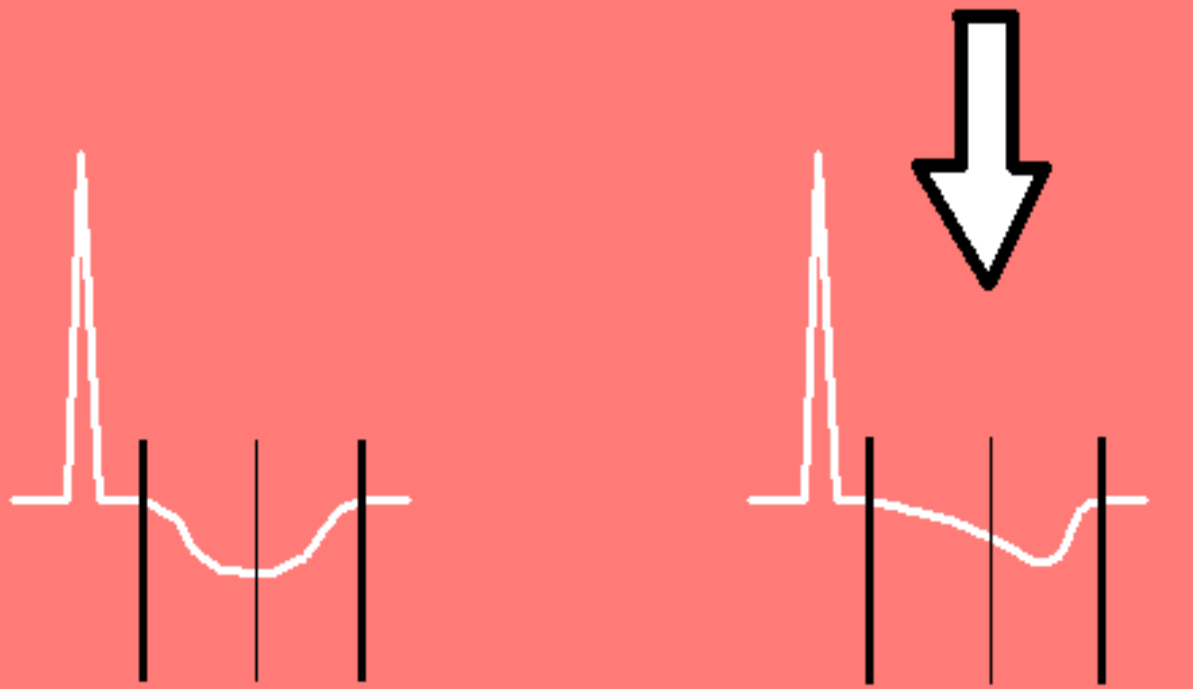
CONDITION:

- MYOCARDITIS
- ELECTROLYTE IMBALANCE
- ISCHEMIA
- POSITIVE STRESS TEST
- CEREBRAL DISORDER
- MITRAL VALVE PROLAPSE
- VENTRICULAR HYPERTROPHY
- WOLFF-PARKINSON-WHITE
- HYPERVENTILATION
- CARDIOACTIVE DRUGS
- OLD MI ( NECROSIS vs. ISCHEMIA )
- DIGITALIS
- R. BUNDLE BRANCH BLOCK
- NO OBVIOUS CAUSE

# CHAMBER ENLARGEMENT

## VENTRICULAR STRAIN PATTERNS

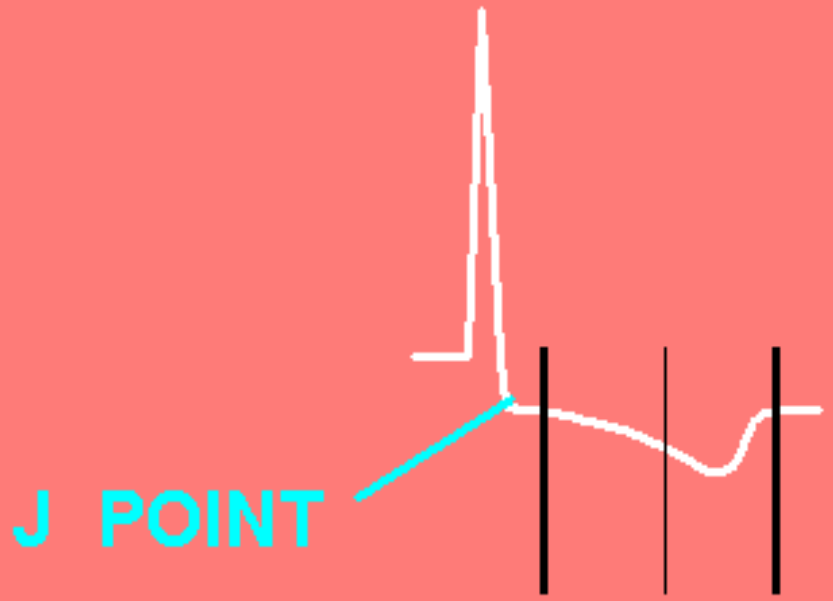
☞ T-WAVES ARE INVERTED and ASYMMETRICAL



# CHAMBER ENLARGEMENT

## VENTRICULAR STRAIN PATTERNS

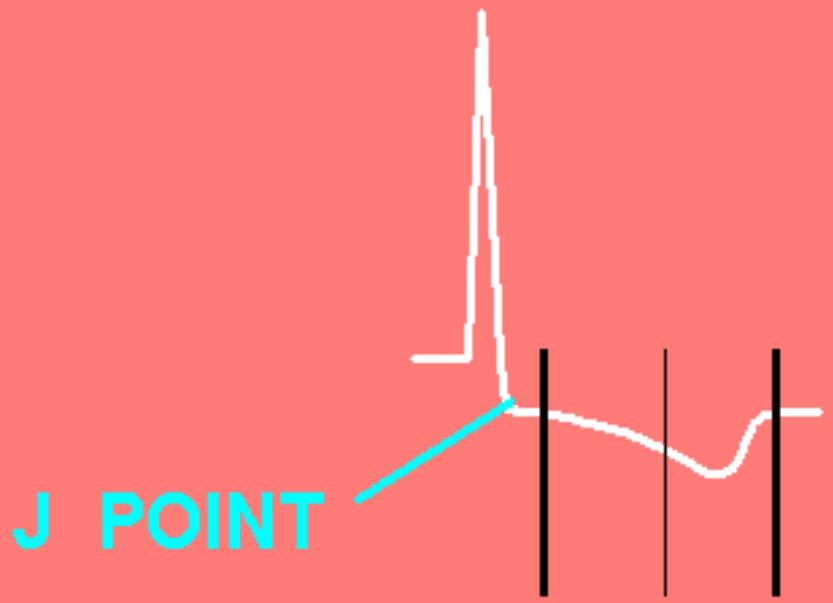
- T WAVES ARE INVERTED AND ASMMETRICAL
- THERE MAY BE S-T SEGMENT DEPRESSION



# CHAMBER ENLARGEMENT

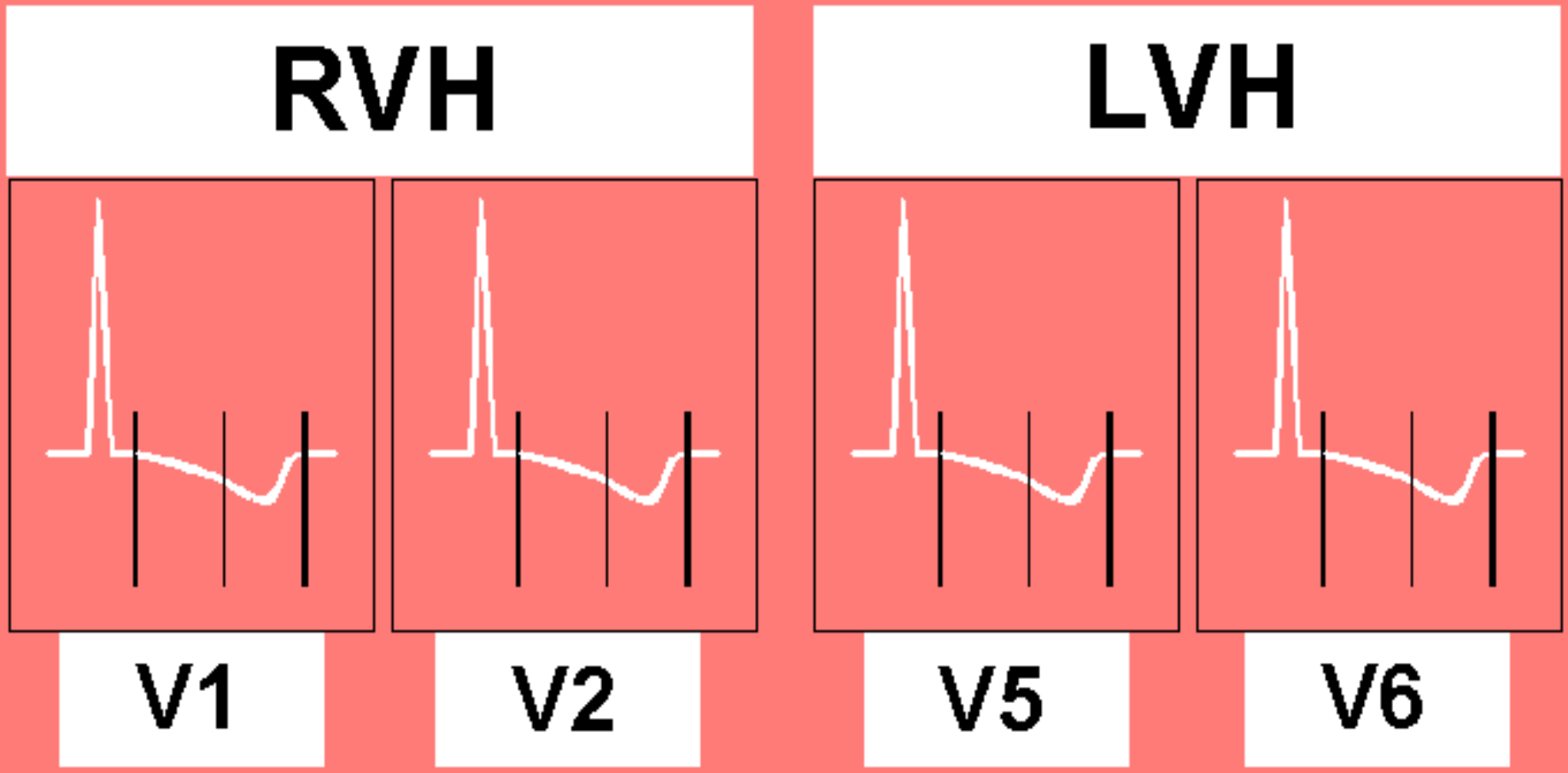
## VENTRICULAR STRAIN PATTERNS

STRAIN PATTERNS ARE INDICATIVE OF SYSTOLIC OVERLOAD -- THE VENTRICLES HAVING TO OVERCOME GREAT FORCE TO EXPEL BLOOD.



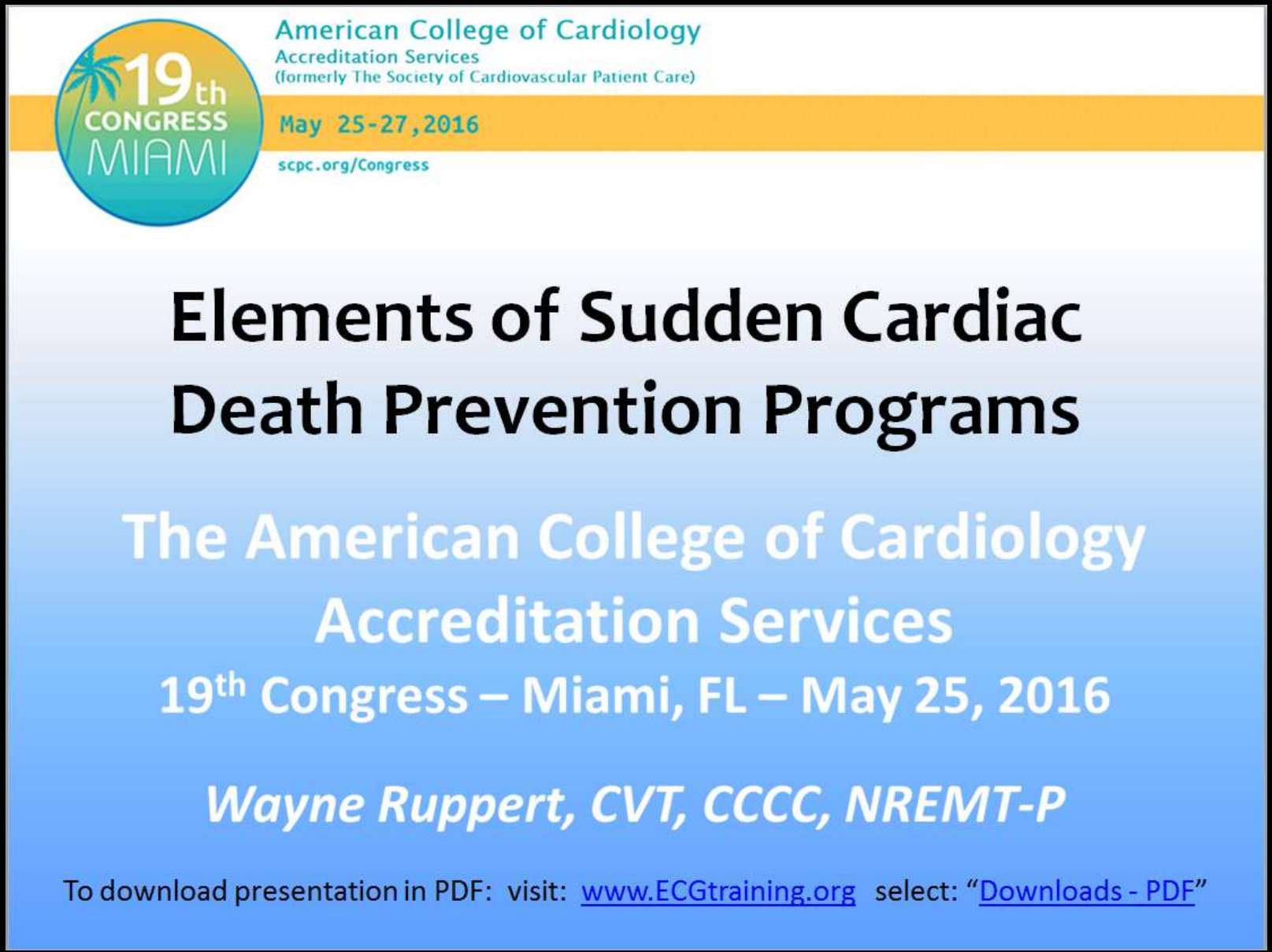
# CHAMBER ENLARGEMENT

## VENTRICULAR STRAIN PATTERNS



MOM and DAD at Lee's Diner, York, PA 2006

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



Brief, focused ECG excerpts from the presentation given by Wayne Ruppert at the "19th Congress," American College of Cardiology Accreditation Services" national conference, on
MAY 25, 2016
Miami, FL …….

# Prevalence
# SADS Foundation Stats:

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

# The SADS Conditions:

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

# Estimated SADS Prevalence in US Population:

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

# Prevalence

## Sudden Deaths in Young Competitive Athletes

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

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

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

**Most ACS Cardiac Arrest Patients are over age 30.**

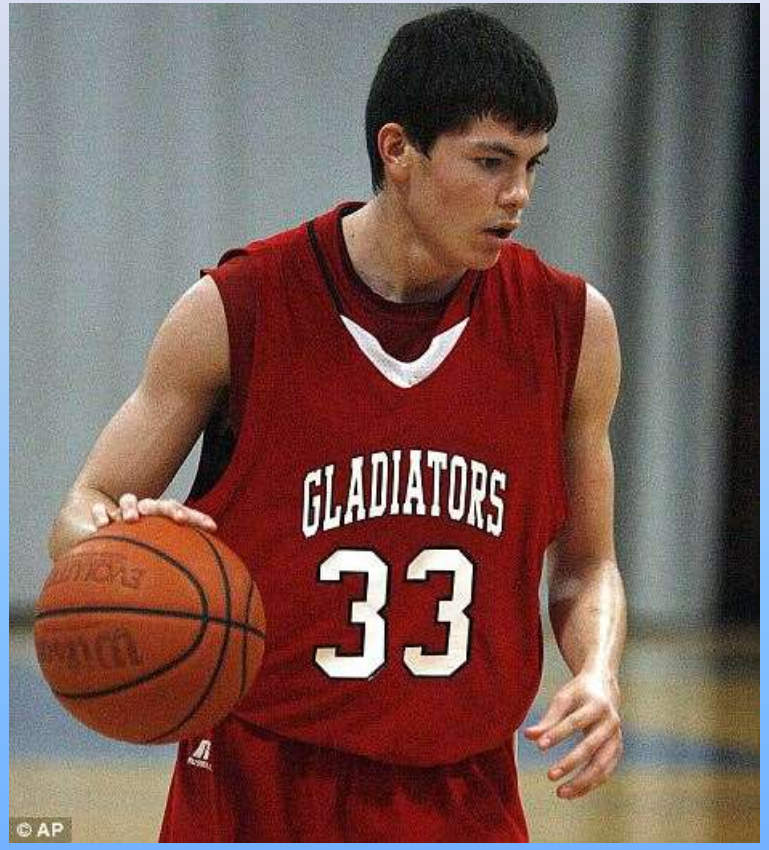
***Meet the typical Cardiac Arrest patients affected by SADS . . . .***

# High School Athlete Dies After Collapsing 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



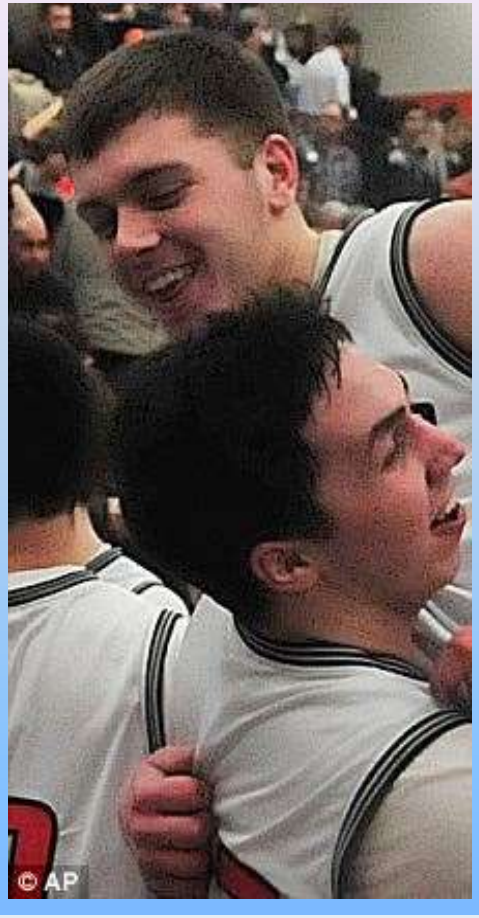

By DAILY MAIL REPORTER
UPDATED: 12:03 EST, 14 March 2011

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."***

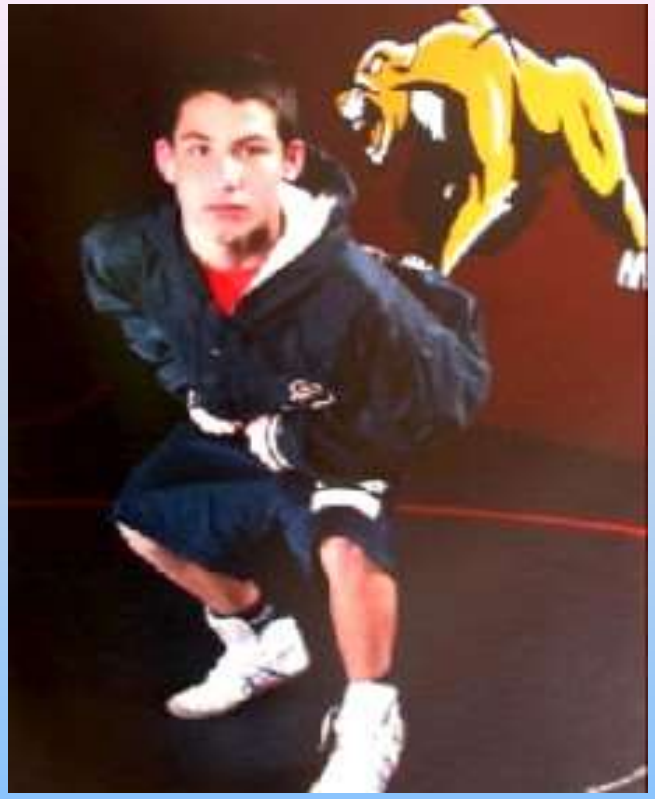

**High school quarterback Reggie Garrett threw his second touchdown pass of the night, walked off the field, and collapsed from sudden cardiac arrest. He died in the ambulance on the way to the hospital in West Orange, Texas.**

**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 21**
**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.

# Athletes like Christian Eriksen can return after cardiac arrest, say experts

Kaya Burgess, Science Reporter

Saturday July 31 2021, 12.01am BST, The Times

Christian Eriksen gives a thumbs-up after his successful treatment following his collapse at the Euros
DFA VIA REUTERS

Athletes can "almost always" return to competitive sport after being diagnosed with a potentially deadly genetic heart condition and can even play again after suffering a cardiac arrest, scientists have found.

The research could be good news for Christian Eriksen, the Danish footballer who had to be resuscitated after his heart stopped during Denmark's Euro 2020 match with Finland last month.

## . . . . And on a more personal note:

**This slide added April 27, 2016:**

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

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

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

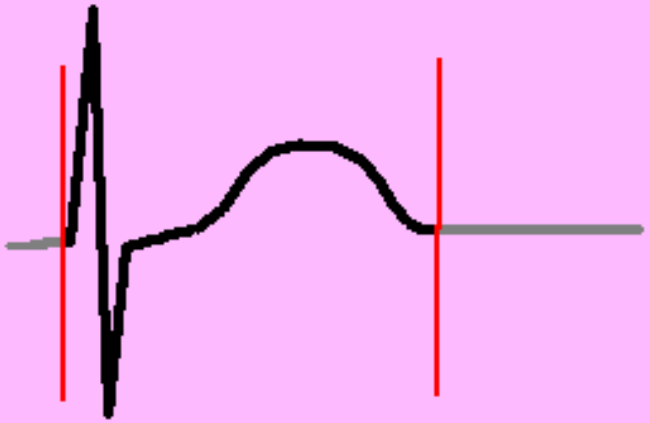
**Entry 5/2/2016: I was advised that the cause of cardiac arrest was Hypertrophic Cardiomyopathy.**

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

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

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

# THE Q-T INTERVAL



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

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

# THE *QTc INTERVAL

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

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

*Annals of Internal Medicine, 1988 109:905.*

# Determining the QTc

## Manual calculation:

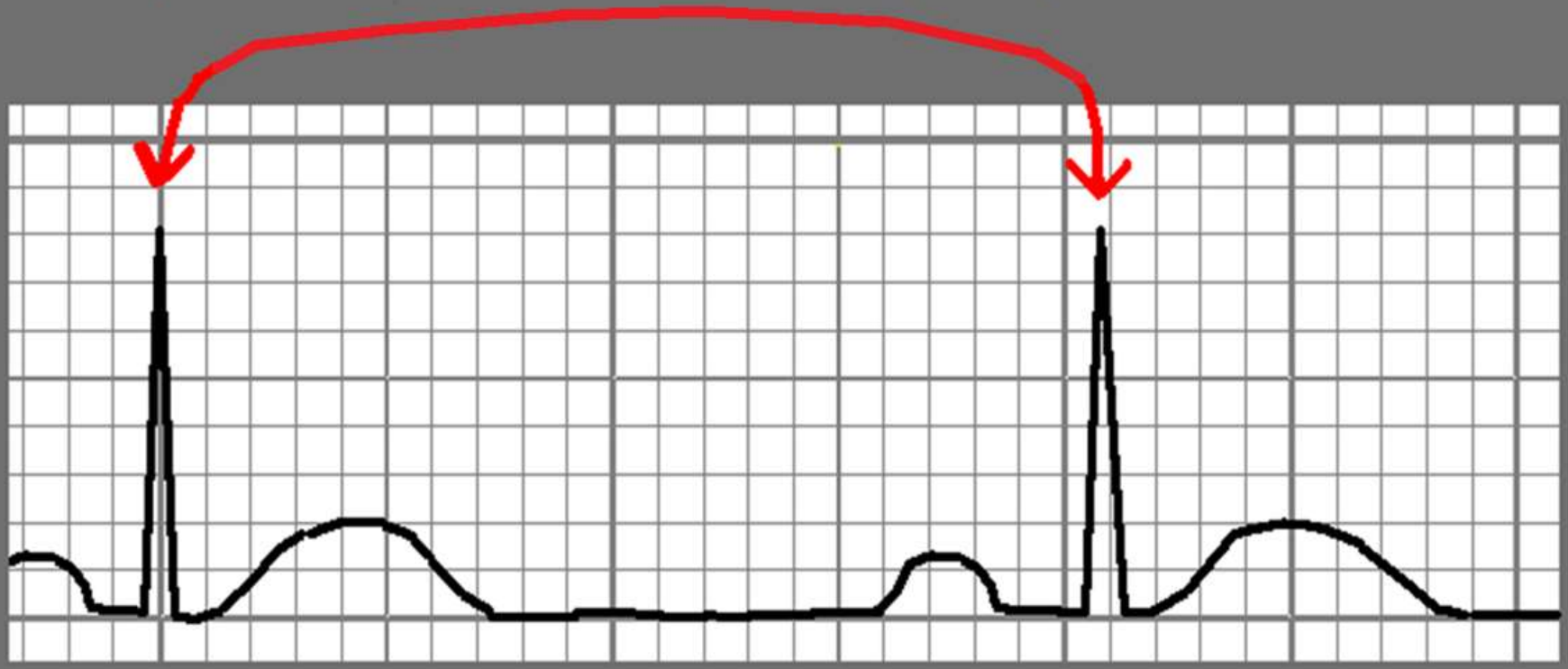
**QT CORRECTION FORMULAS:**

| | |
|---|---|
| Bazett's | $QTc=QT/\sqrt{RR}$ |
| Fredericia | $QTc=QT/(RR)1/3$ |
| Framingham | $QTc=QT+0.154(1-RR)$ |
| Rautaharju | $QTp=656/(1+HR/100)$ |

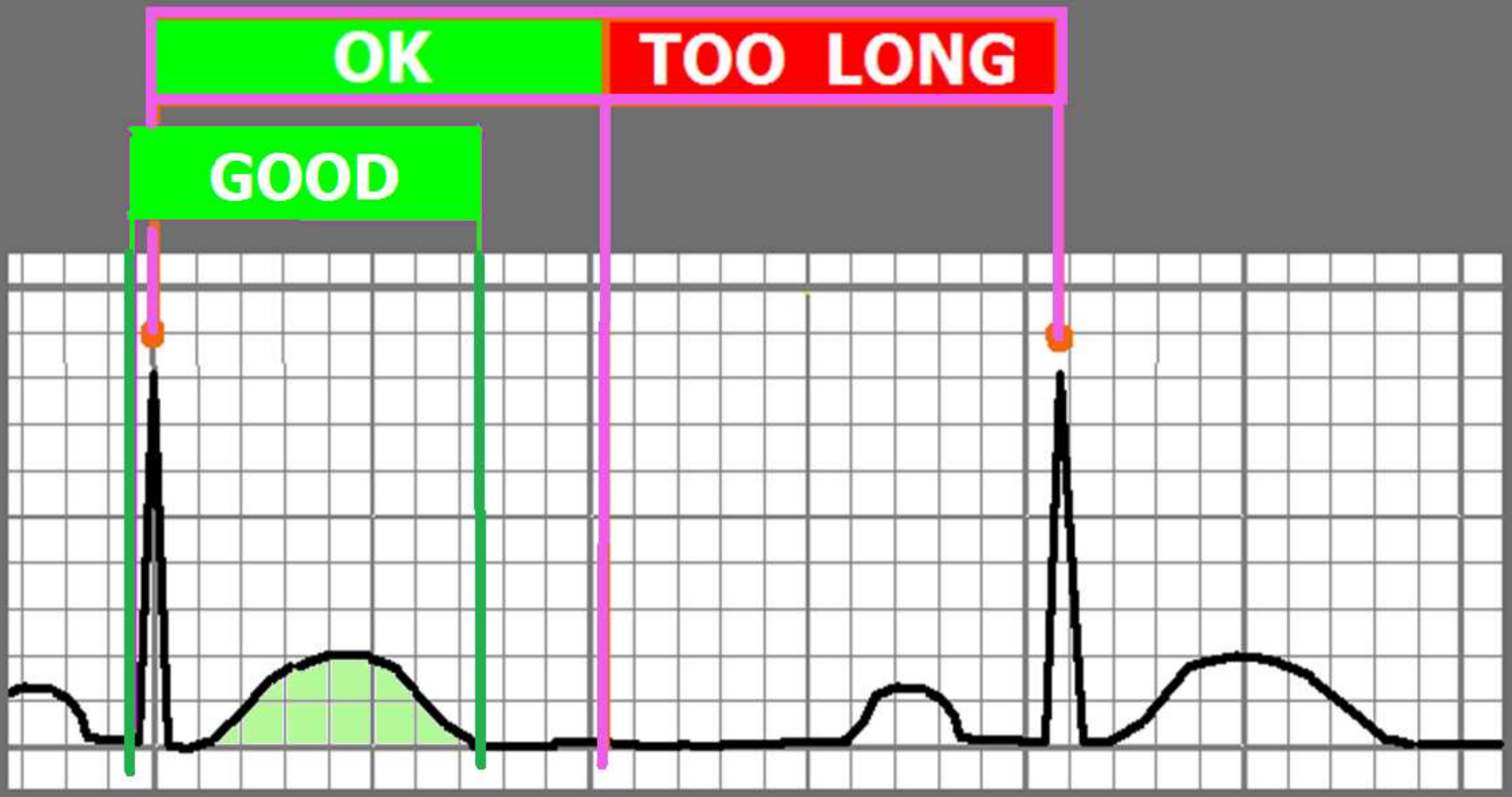
# DETERMINING Q-T INTERVAL LIMITS

## THE "QUICK PEEK" METHOD

- Relatively accurate method to quickly identify patients with abnormal QT Intervals.
  - Applies to patients with normal heart rates (60-100) and narrow QRS ( QRSd <120ms )



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



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

# Determining the QT / QTc

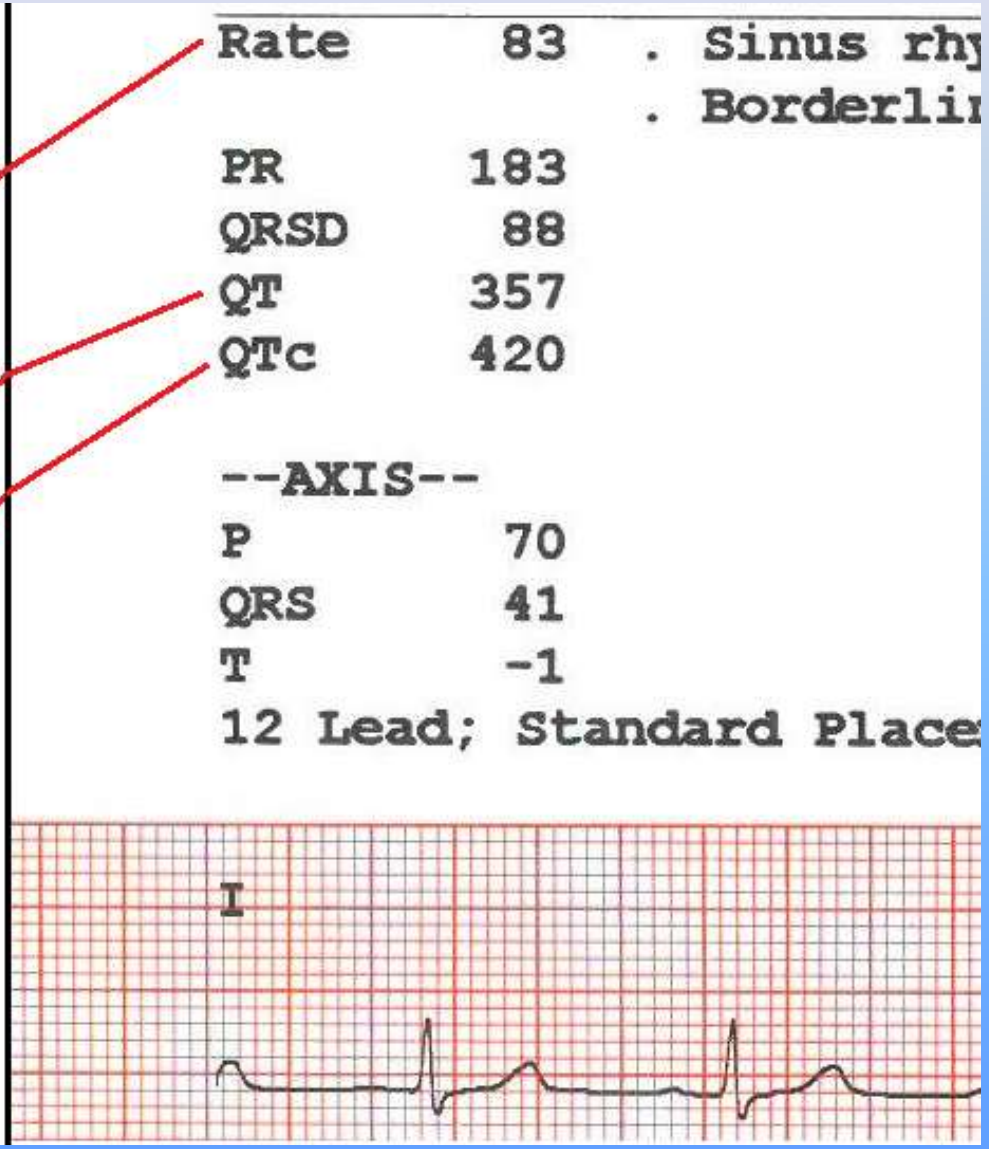
## Method 1 – 12 Lead ECG Report:

Standard 12 Lead ECG printout . . .

Heart Rate = 83

QT Interval = 357

QTc = 420

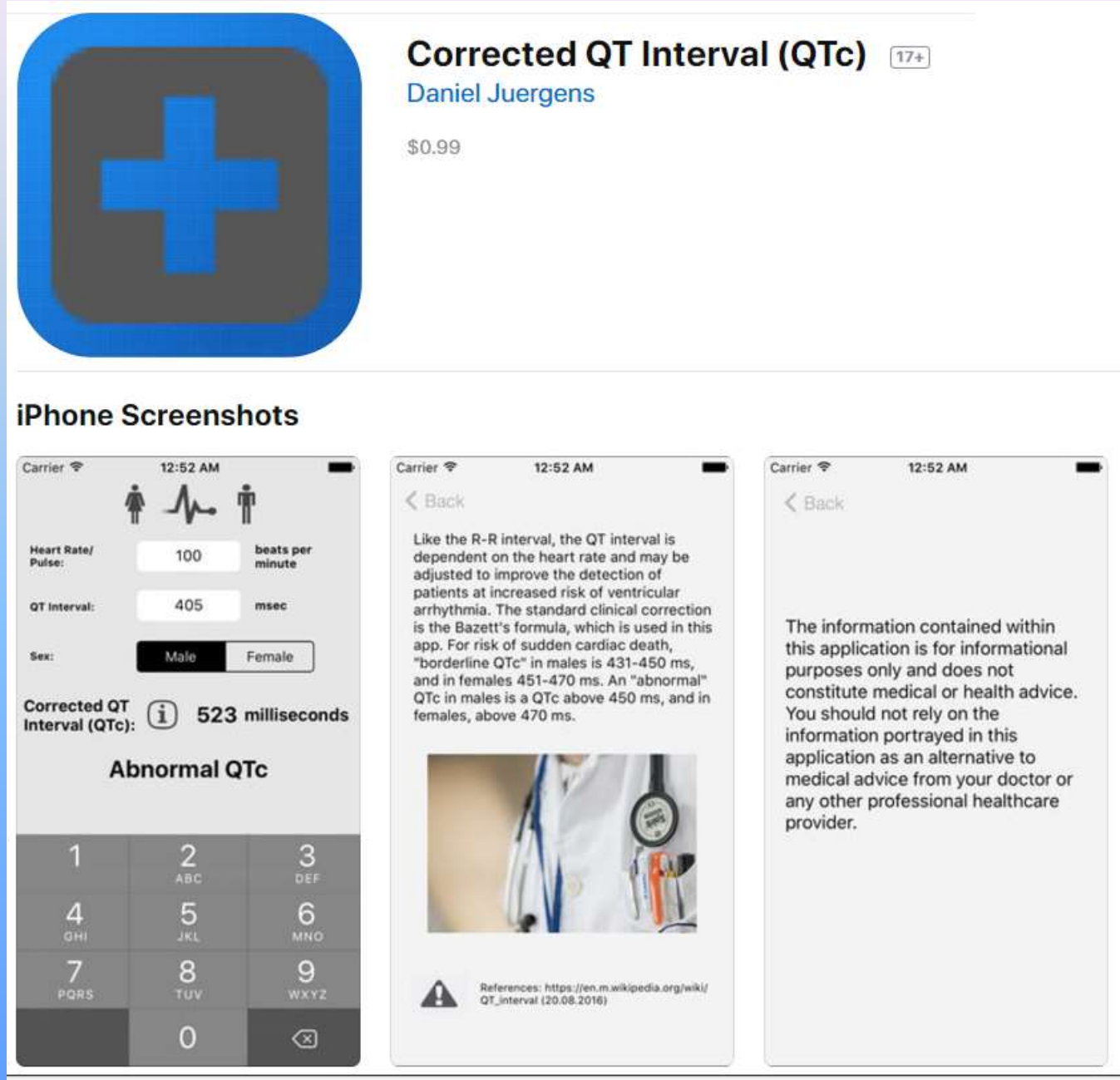


# Determining the QTc

## Method 4, Use a Smartphone App:

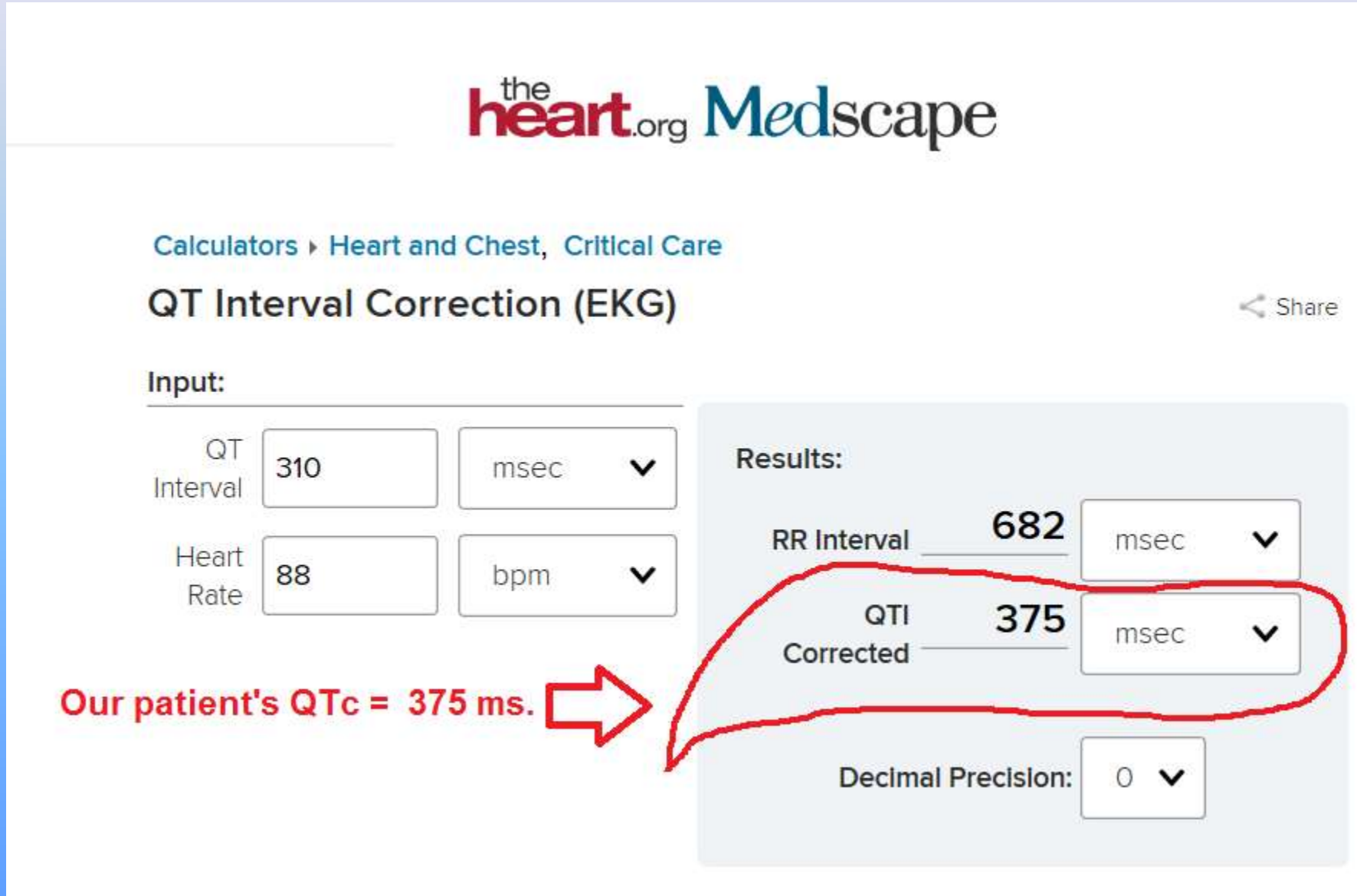
- **iPhone**
  - https://itunes.apple.com/us/app/corrected-qt-interval-qtc/id1146177765?mt=8
- **Android**
  - https://play.google.com/store/apps/details?id=com.medsam.qtccalculator&hl=en

# “There’s an APP for that!”



# Determining the QTc

## Method 3, Use a Web-based App:

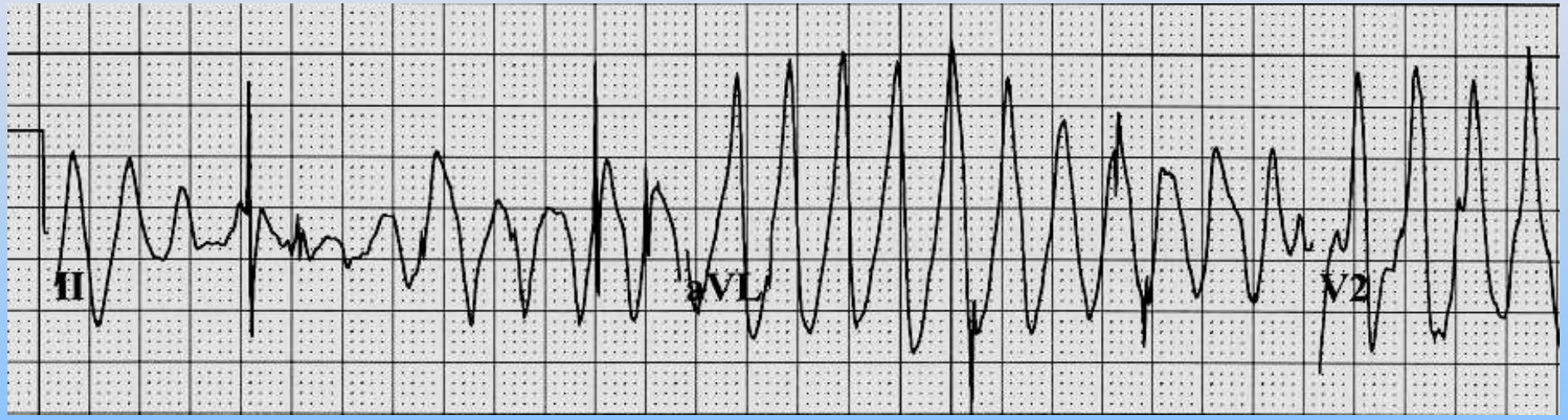


# QTc Values:

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

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

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



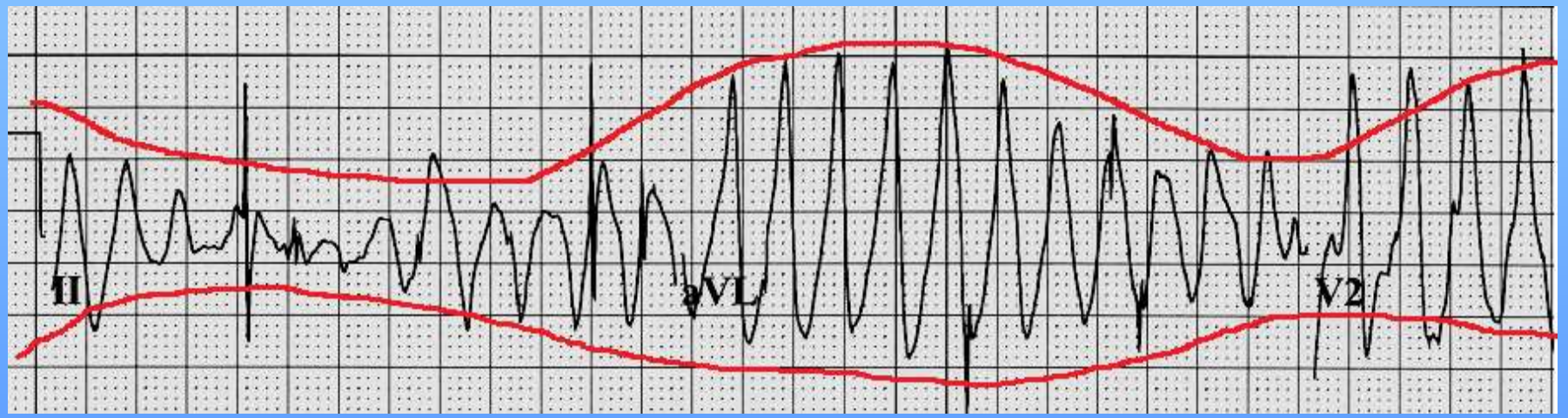

**Torsades de Pointes (TdP) – HEMODYNAMICS:**

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

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

**resembles . . . . .**

***a piece of Twisted Ribbon !***

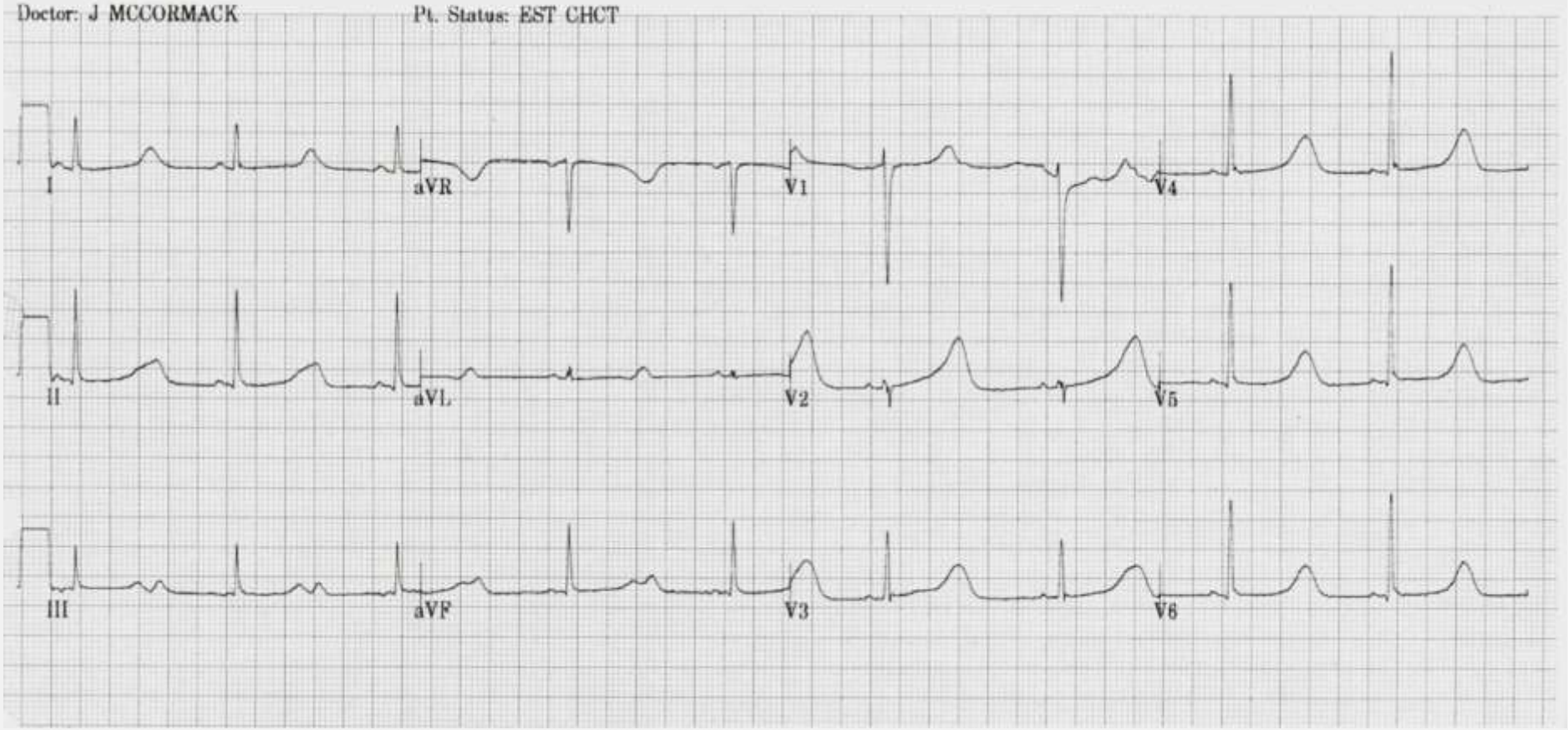


**22 y/o FEMALE**

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

PEDIATRIC CARDIOLOGY ASSOCIATES

**Chief Complaint: "Grand-Mal Seizures"**
**…. *With NO postictal phase!***




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


Cardiology in the Young

## Article contents



# The Role of Genetic Testing In Paediatric Syndromes of Sudden Death: State Of The Art and Future Considerations

Published online by Cambridge University Press: **01 November 2009**

Jorge McCormack

Show author details



## Abstract

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*Cardiology in the Young* (2009), **19(Suppl. 2)**, 54–65
doi:10.1017/S1047951109991636



*Original Article*

## The Role of Genetic Testing In Paediatric Syndromes of Sudden Death: State Of The Art and Future Considerations

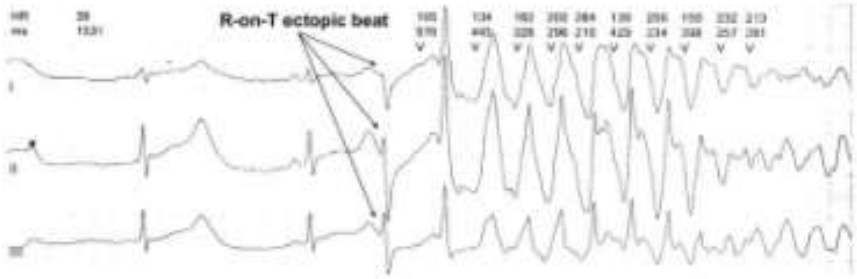
Jorge McCormack, MD, FACC

*The Congenital Heart Institute of Florida (CHIF), Division of Pediatric Cardiology, All Children's Hospital and Children's Hospital of Tampa, Pediatric Cardiology Associates/Pediatrix Medical Group, Saint Petersburg and Tampa, Florida, United States of America*

# Long QT Syndromes and Torsade de Pointes

Gan-Xin Yan

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




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

Figure 1:

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

**GENETICALLY ACQUIRED LONG QT SYNDROMES:**
ECG PATTERNS of 3 MOST COMMON VARIATIONS:

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

## Etiology of Long QT Syndromes:

**Congenital** (14 known subtypes)

Genetic mutation results in abnormalities of cellular ion channels

**Acquired**

- Drug Induced
- Metabolic/electrolyte induced
- Very low energy diets / anorexia
- CNS & Autonomic nervous system disorders

**Miscellaneous**

- Coronary Artery Disease
- Mitral Valve Prolapse

# PROLONGED Q-T INTERVAL

THINK:

- CHECK K+ AND MAG LEVELS
- POSSIBILITY OF TORSADES

# PROLONGED Q-T INTERVAL

THINK:

- CHECK K+ AND MAG LEVELS
- POSSIBILITY OF TORSADES

***- QUESTION MEDS THAT PROLONG Q-T***

# QT Prolongation -- *STAT Intervention:*

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

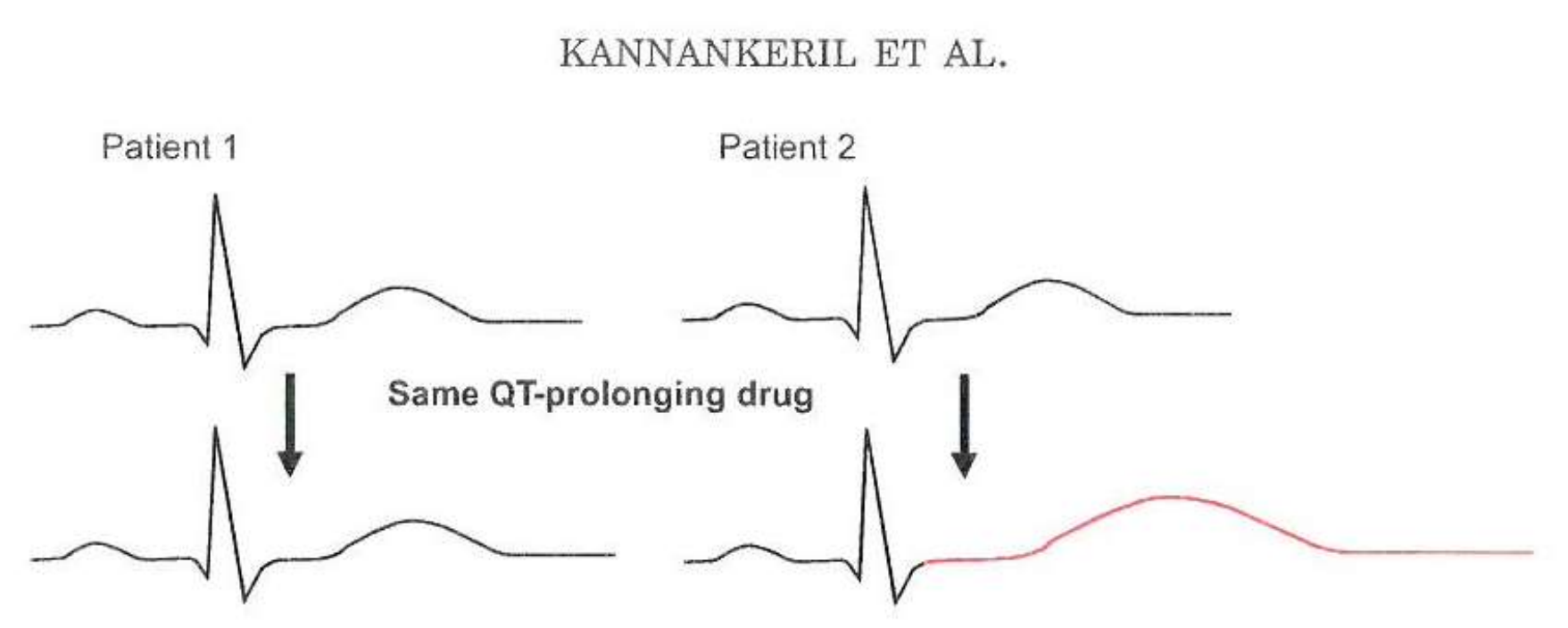
***Commonly used QT prolonging meds include:***

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

***and MANY more!***

PATIENT 1: NORMAL

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




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

Policy, Procedure and Protocol

Bayfront Health Dade City

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

1. PURPOSE:

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

2. POLICY:

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

Click here to download QTc Interval Monitoring Policy

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

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

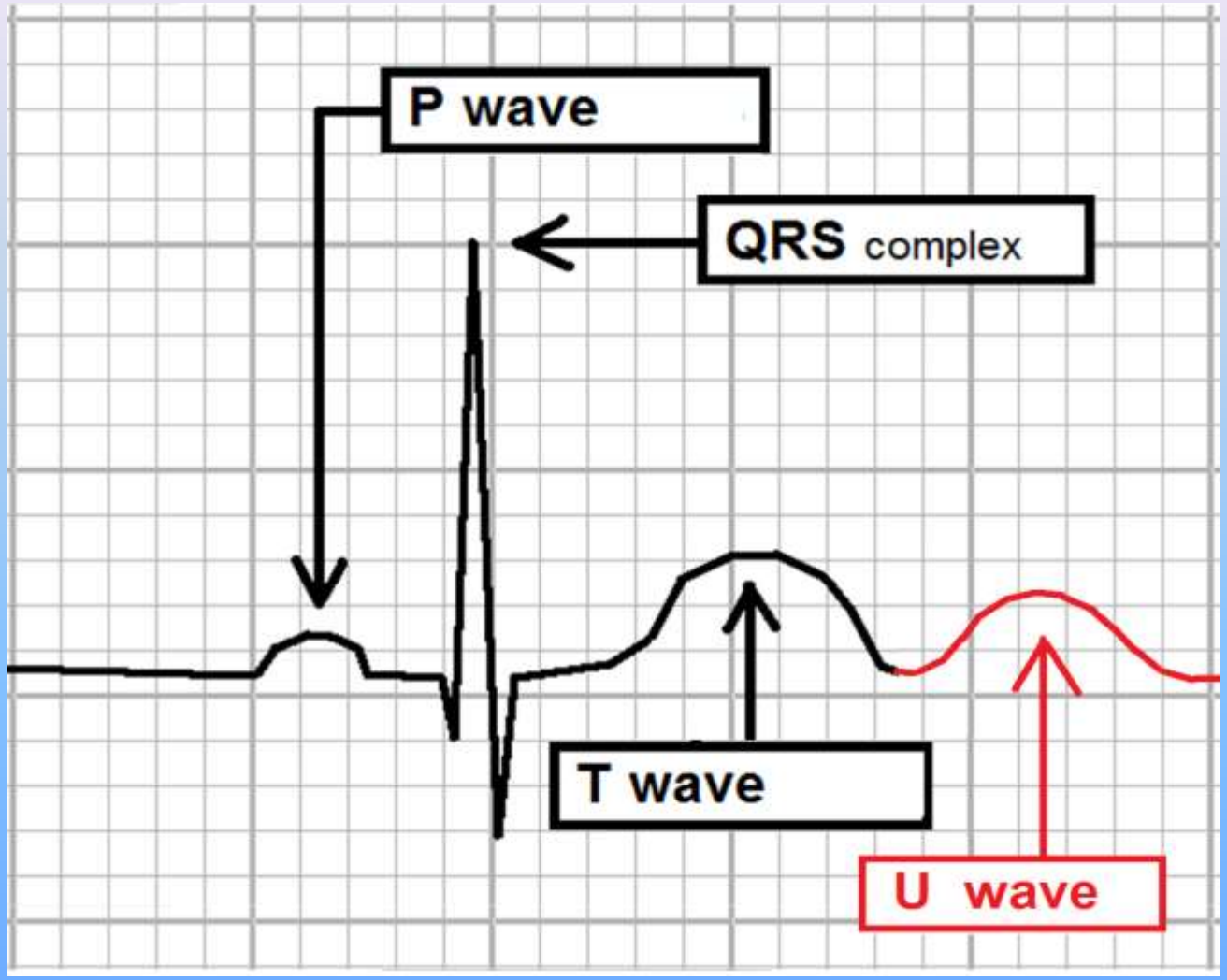
Click for link to hospital model policy & procedure for: "QT Prolonging Medications; QT interval monitoring"

# U Waves

**Occasionally an extra wave is noted after each T wave.**

**It typically resembles**

***"a secondary T wave."***




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

# U Waves . . .

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

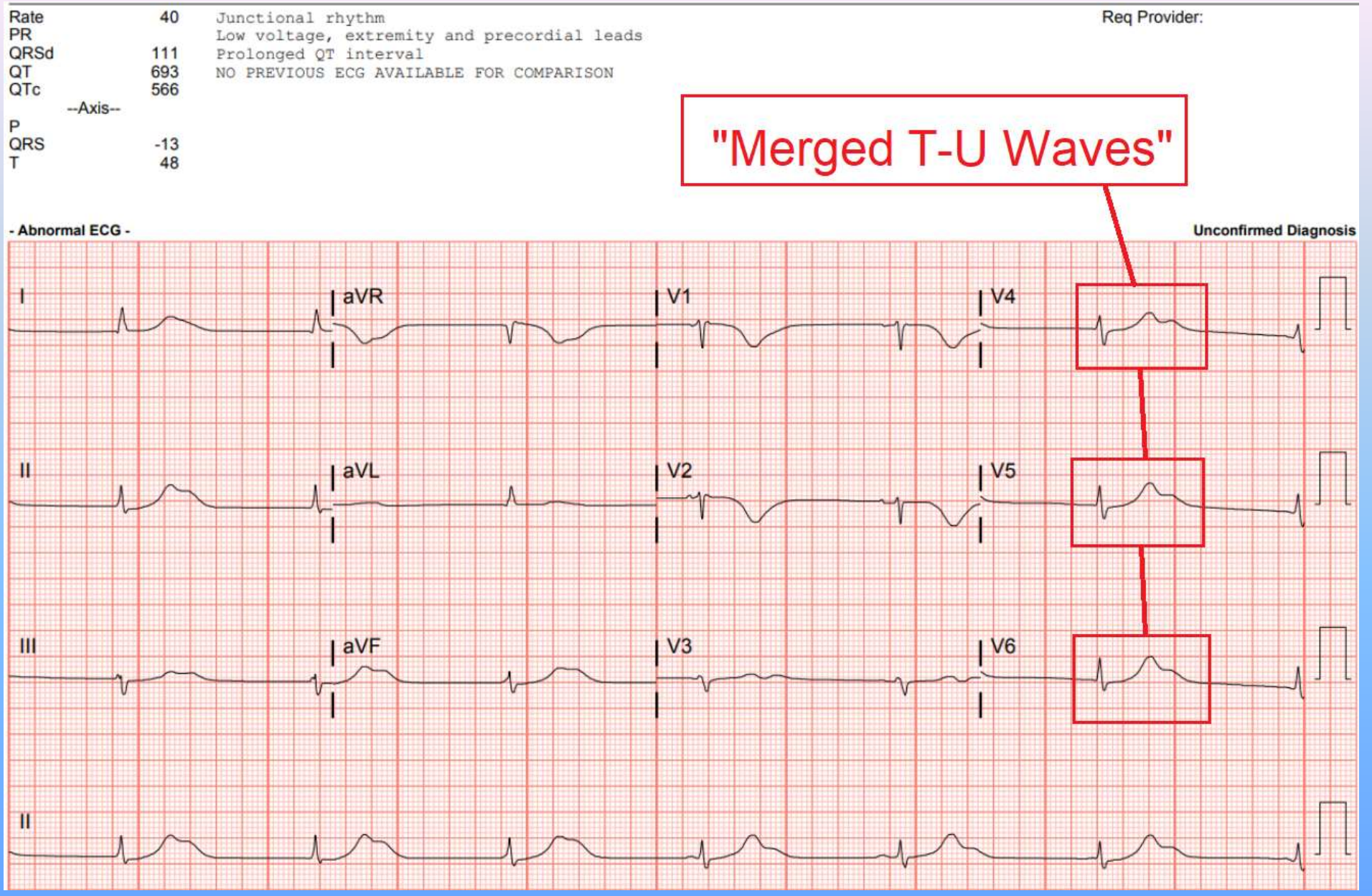
**** These are also causes of QT interval prolongation.***

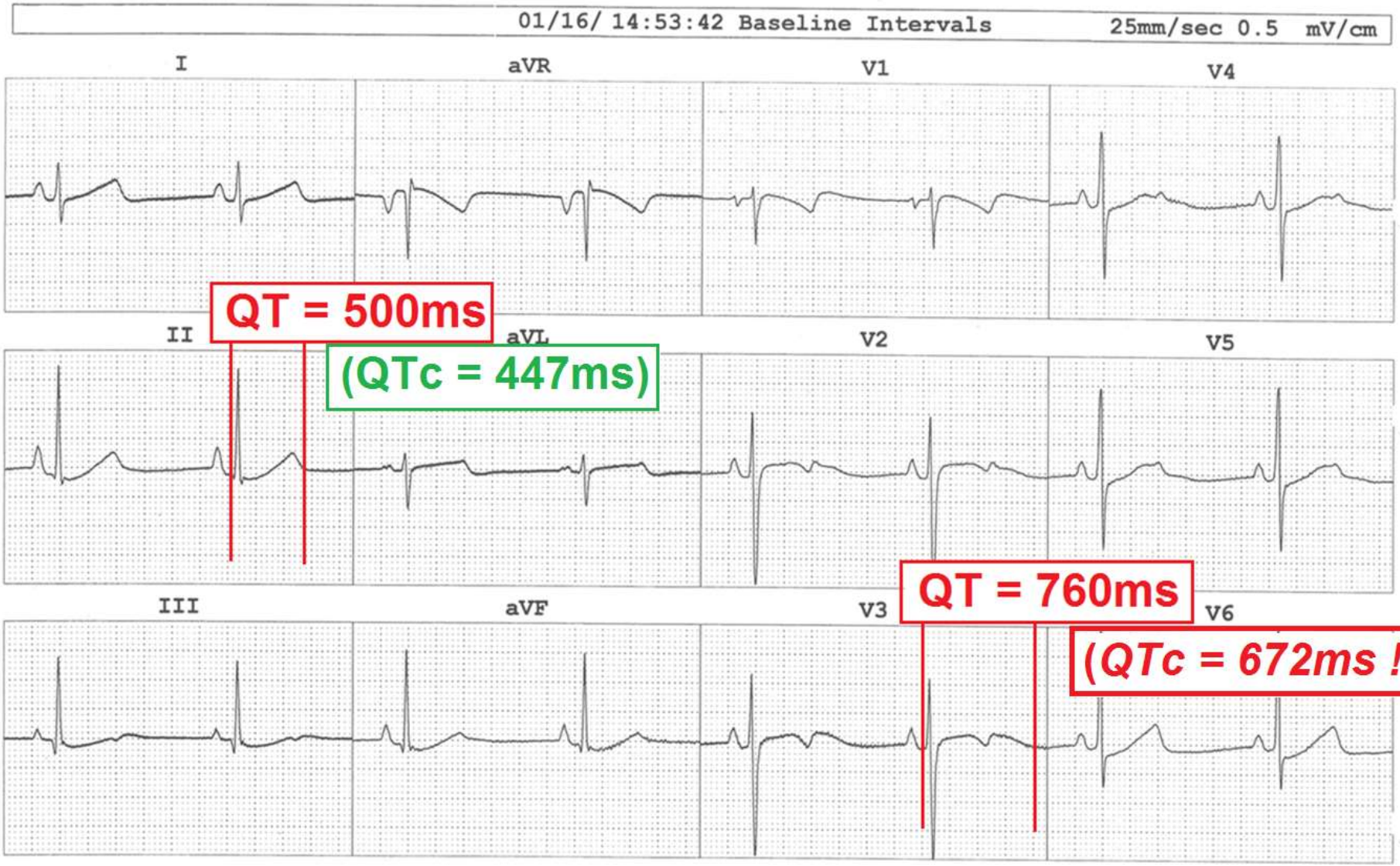
# Abnormal U Waves

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

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

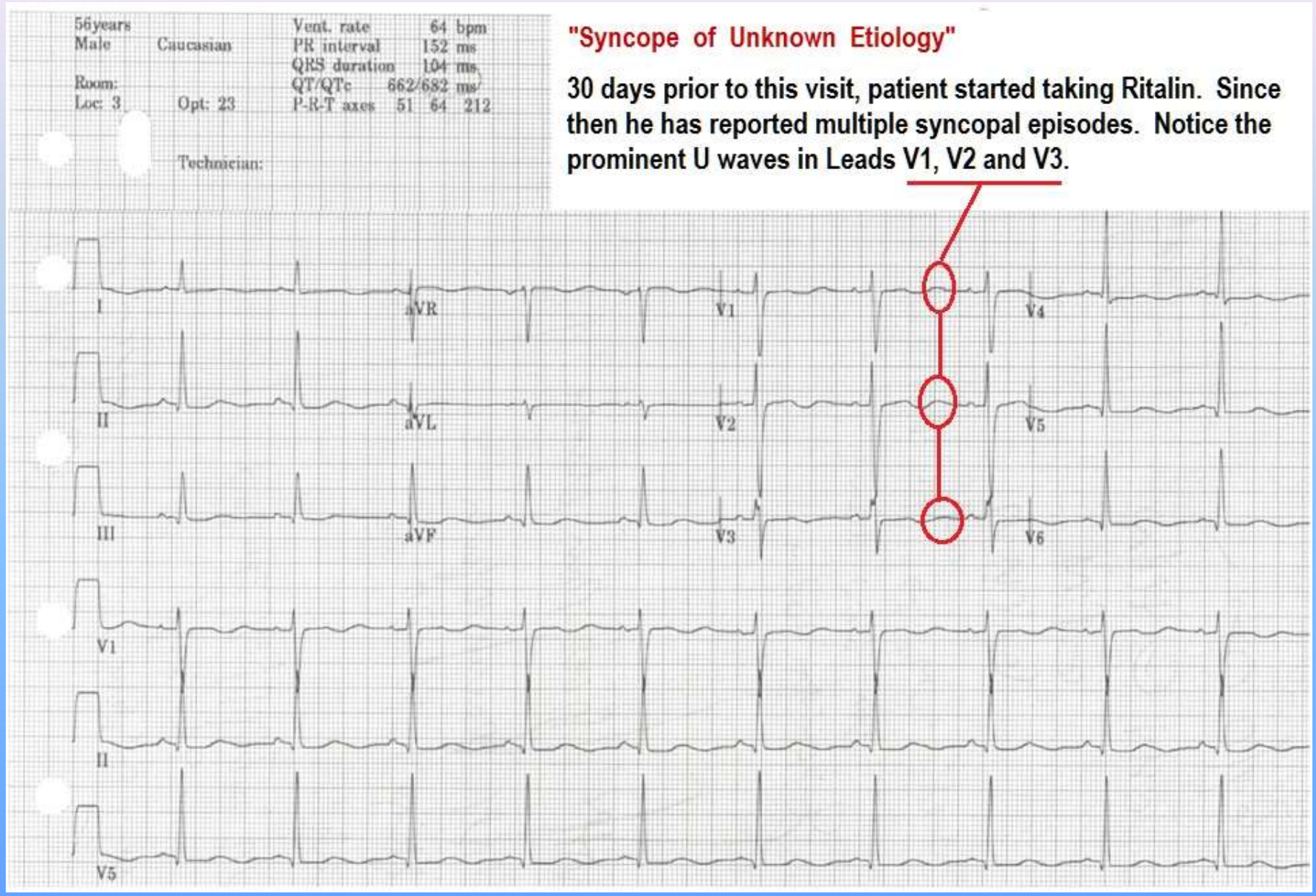
**EVIDENCE SOURCE:**
**ACC/AHA/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram Part IV: The ST Segment, T and U Waves, and the QT Interval.**


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

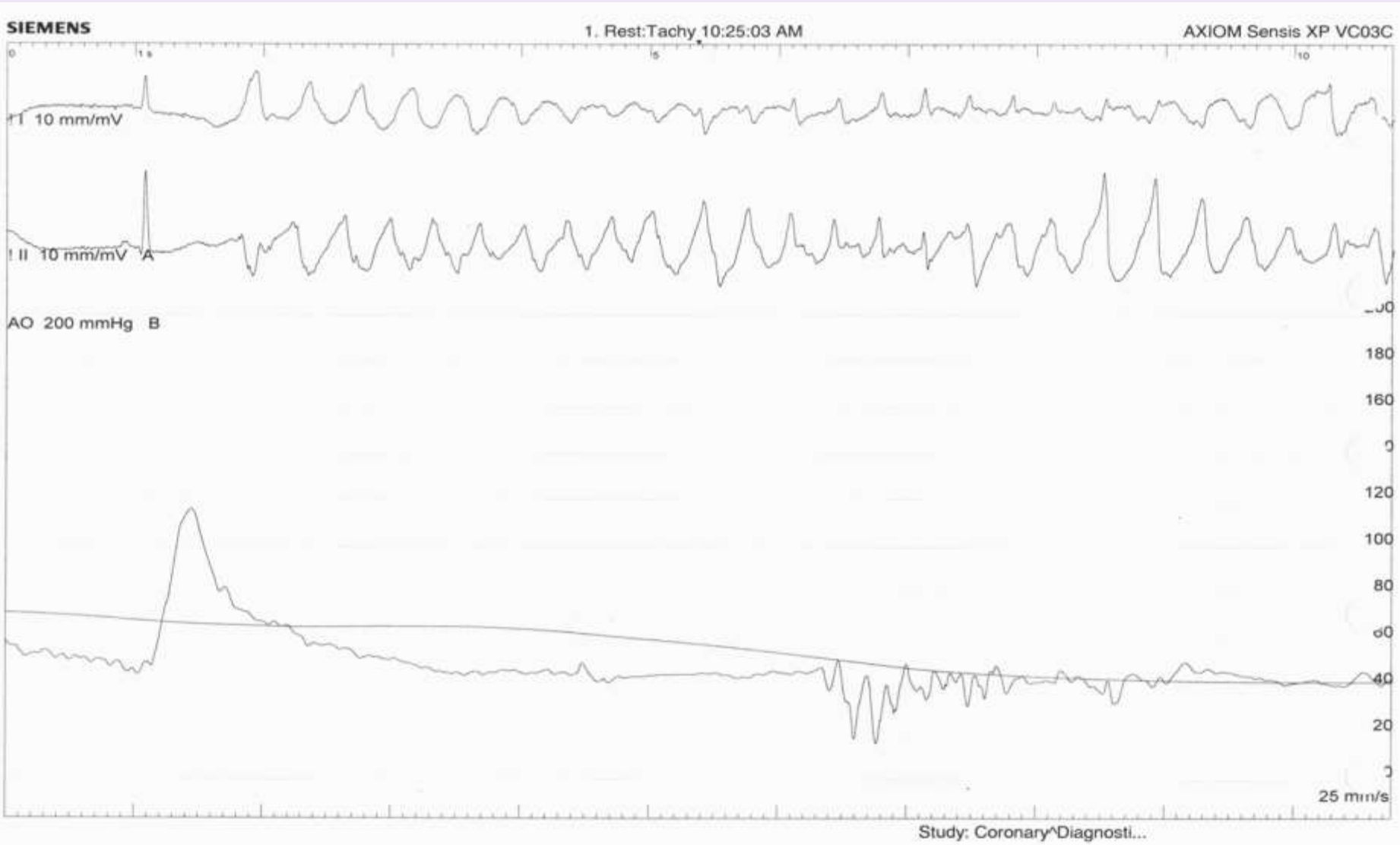



**This ECG illustrates the degree of variation that can be noted between different leads on the 12 Lead ECG. ALWAY measure the QT Interval in the lead with the GREATEST value.**

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

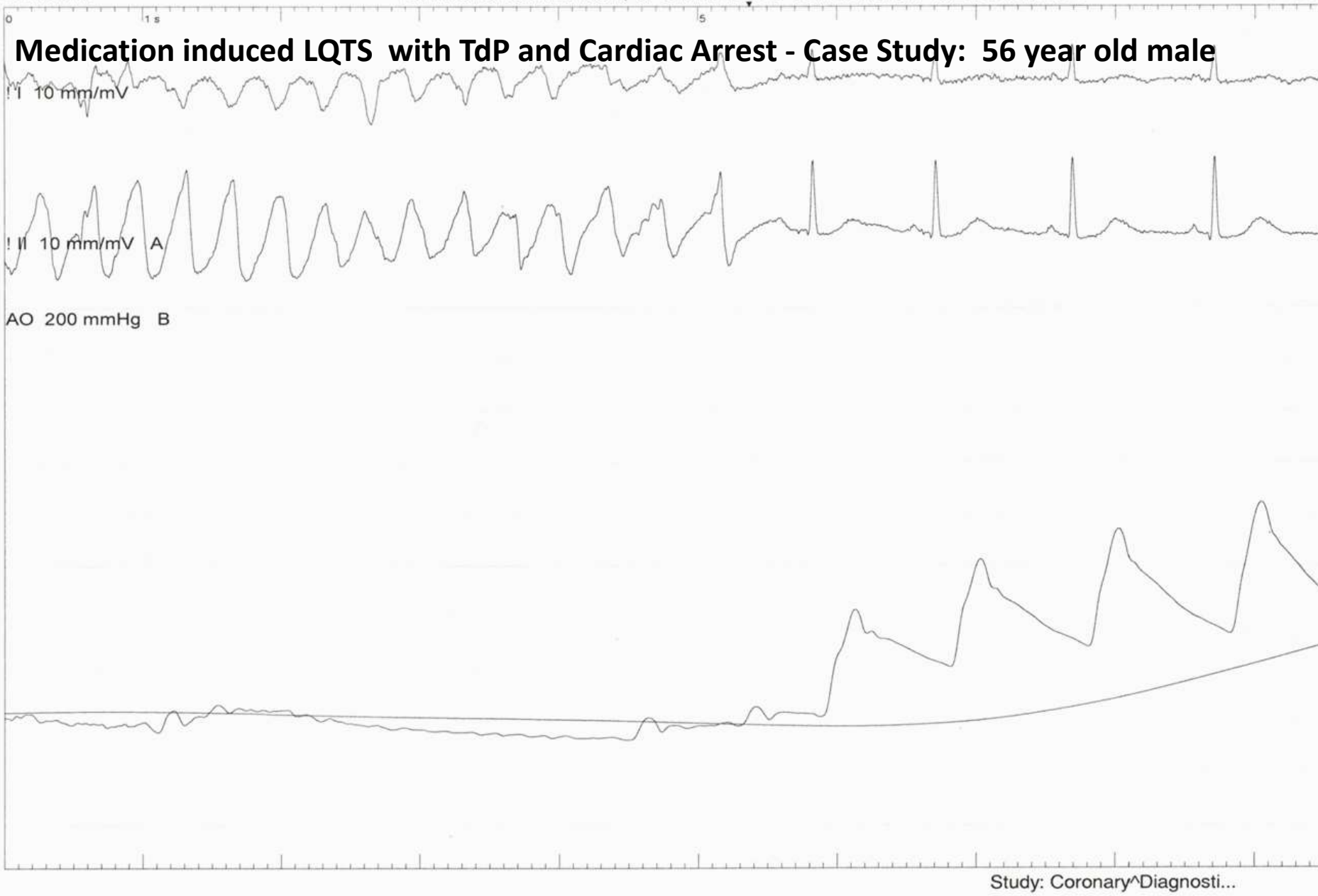


## Medication induced LQTS with TdP and Cardiac Arrest - Case Study: 56 year old male



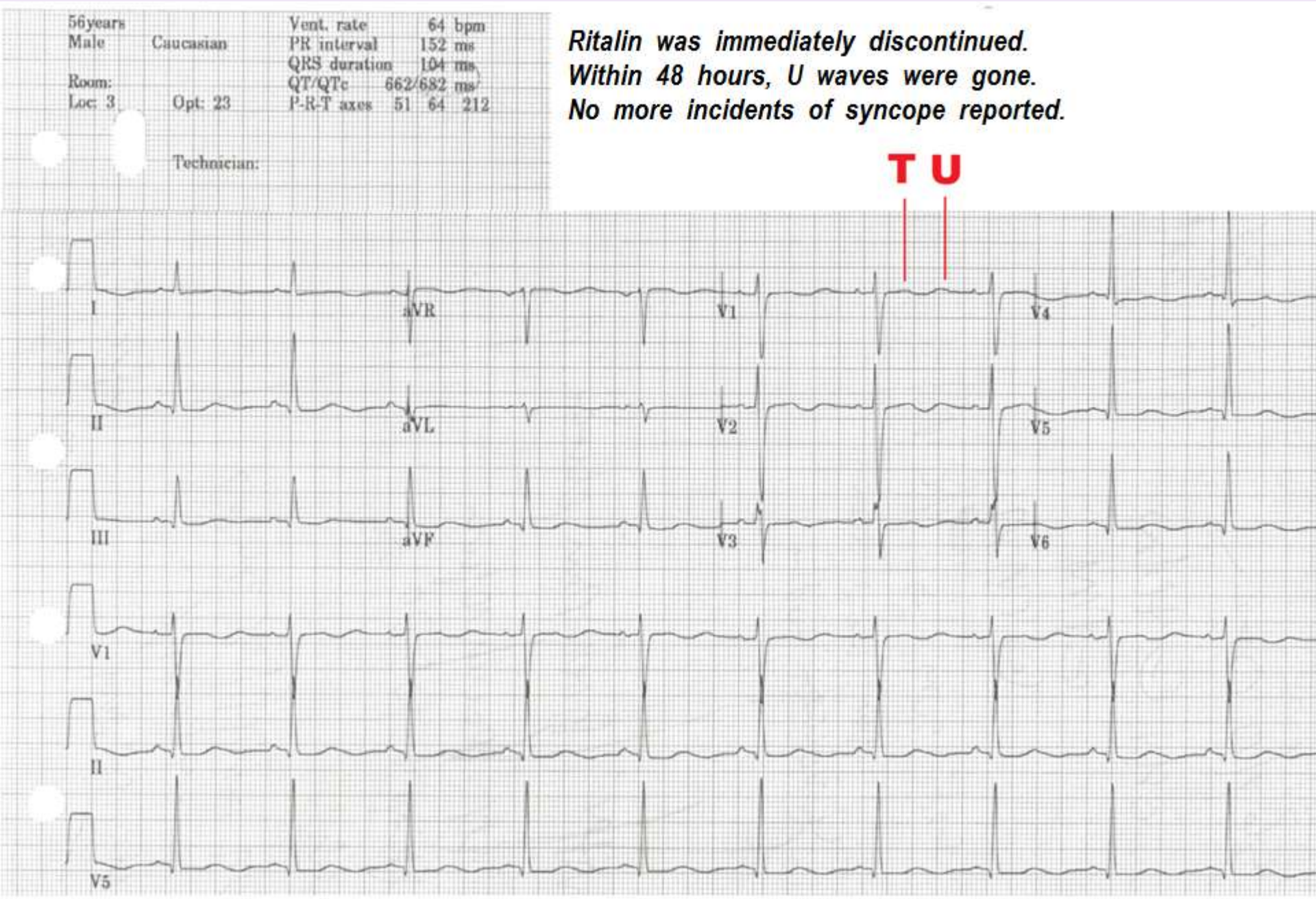

**Run of Torsades de Pointes occurred during Cardiac Catheterization . . .**

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




**Torsades de Pointes self-terminates just before aborted Defibrillation**

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



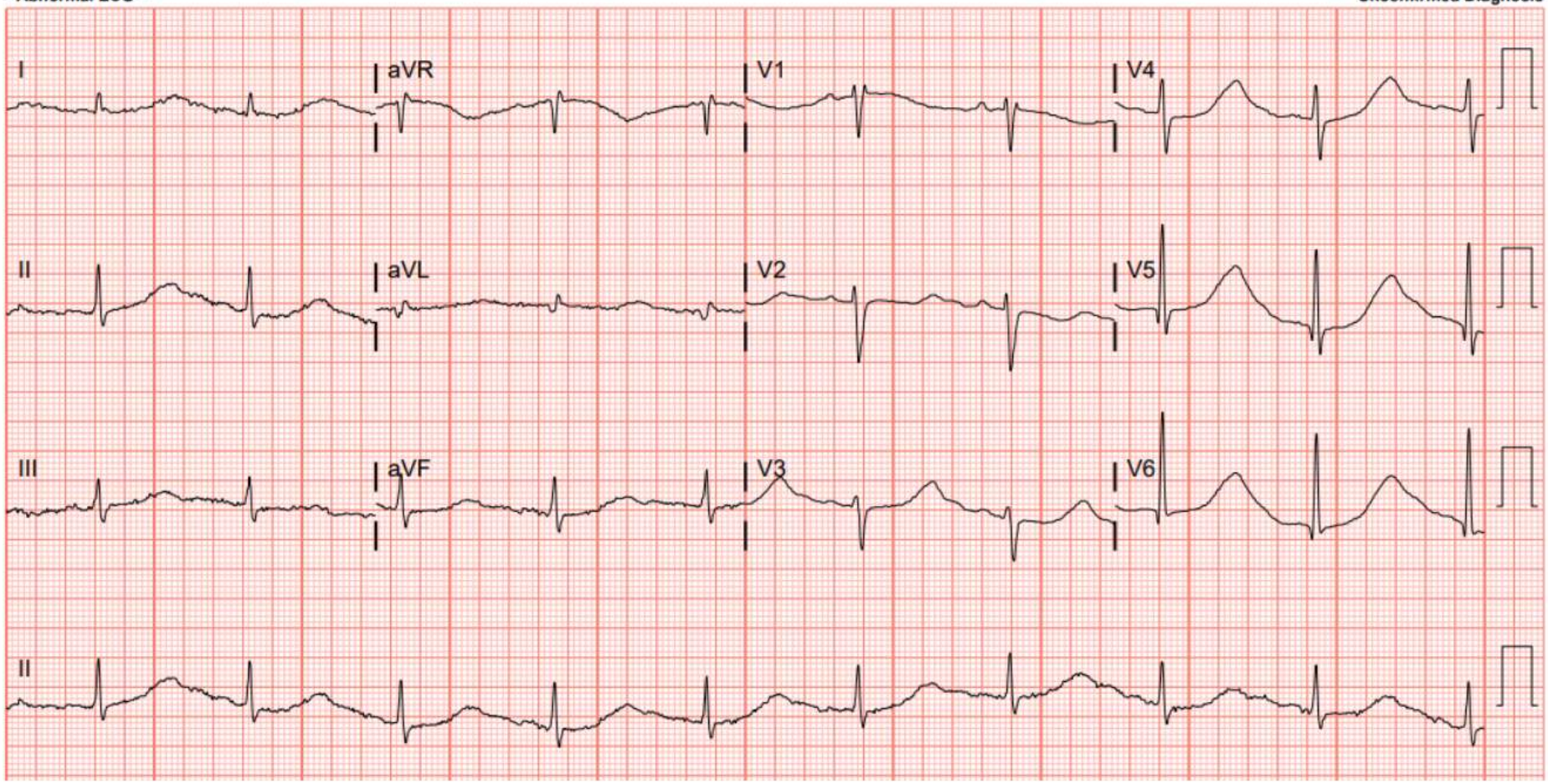
| | |
|---|---|
| Rate | 58 |
| PR | 185 |
| QRSd | 126 |
| QT | 668 |
| QTc | 657 |
| --Axis-- | |
| P | 107 |
| QRS | 61 |
| T | 45 |

Sinus rhythm
IVCD, consider atypical RBBB
Baseline wander in lead(s) V2,V3,V4,V6
COMPARED TO ECG 07/22/2020 16:56:59
SINUS RHYTHM NOW PRESENT

Req Provider: Rafael Santiago-Aponte

- Abnormal ECG -

Unconfirmed Diagnosis

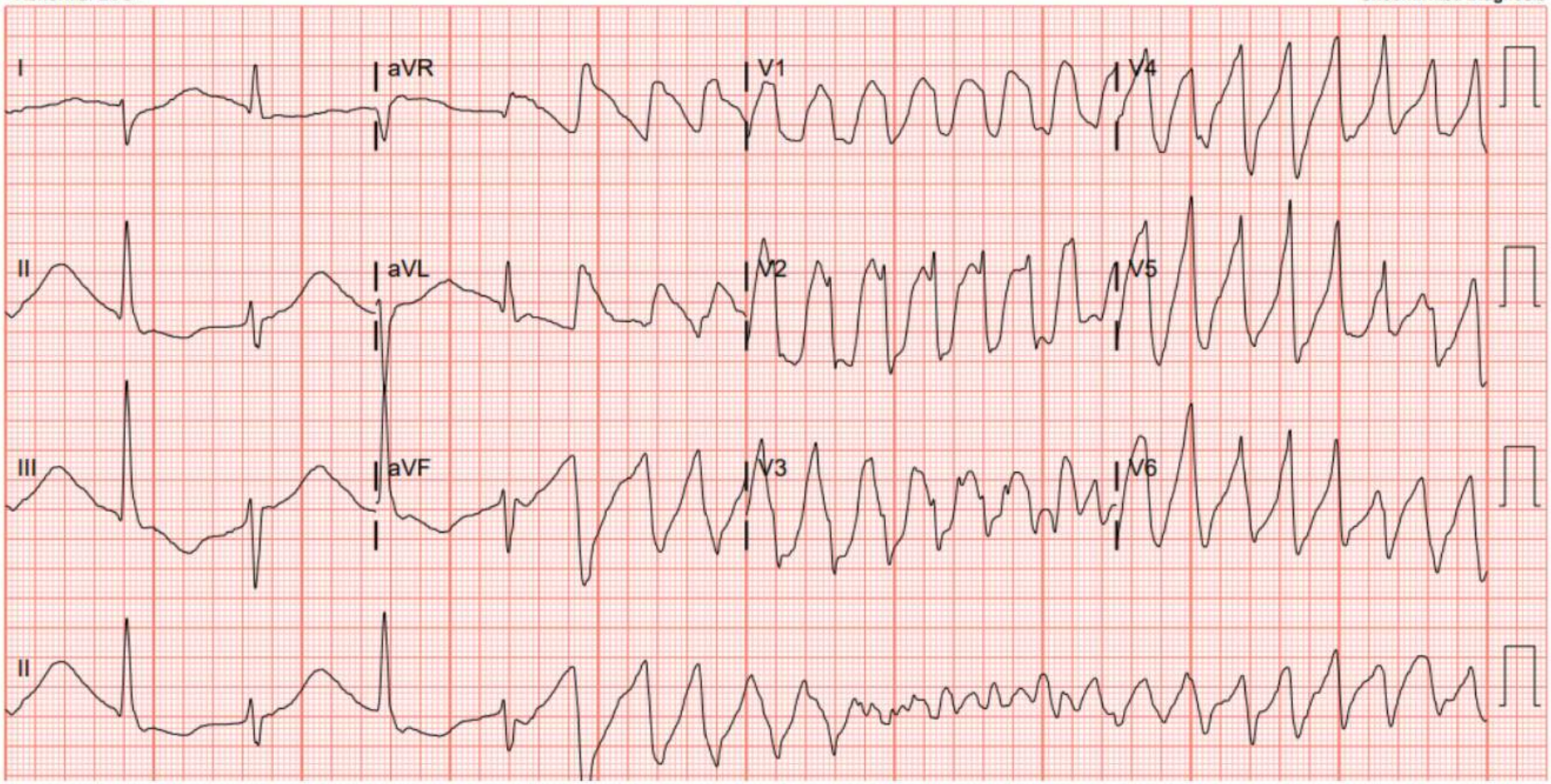


| | | |
|---|---|---|
| Rate | 156 | Atrial fibrillation |
| PR | | Ventricular tachycardia, unsustained |
| QRSd | 138 | RBBB and LPFB |
| QT | 456 | Repol abnrm suggests ischemia, diffuse leads |
| QTc | 735 | Baseline wander in lead(s) II,III,aVR,aVF,V1,V2,V3,V4 |
| --Axis-- | | COMPARED TO ECG 07/22/2020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

Req Provider: Rafael Santiago-Aponte

**- Abnormal ECG -**

**Unconfirmed Diagnosis**



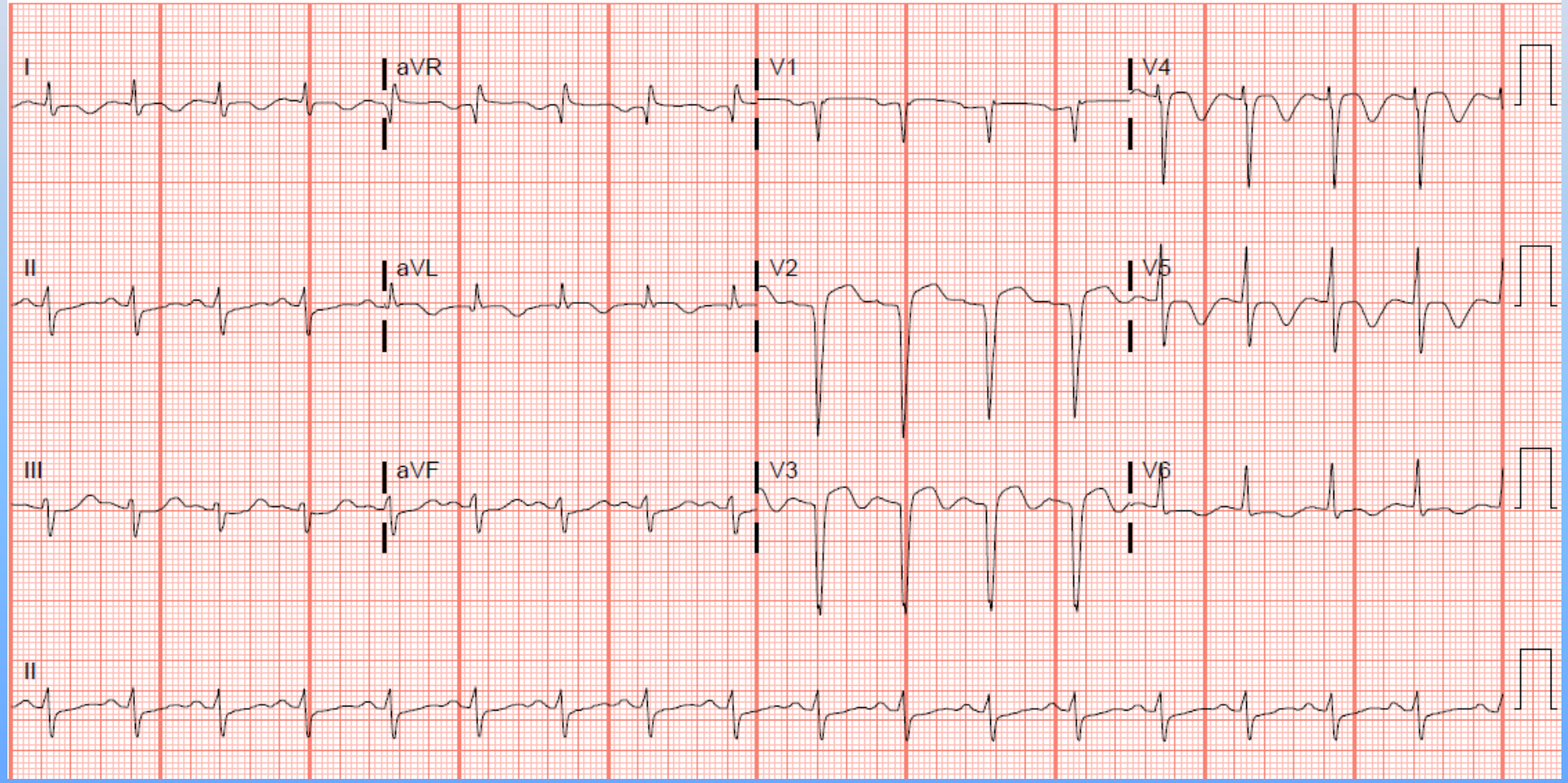
| | |
|---|---|
| Rate | 104 |
| PR | 96 |
| QRSd | 110 |
| QT | 443 |
| QTc | 583 |
| --Axis-- | |
| P | -90 |
| QRS | -84 |
| T | 149 |

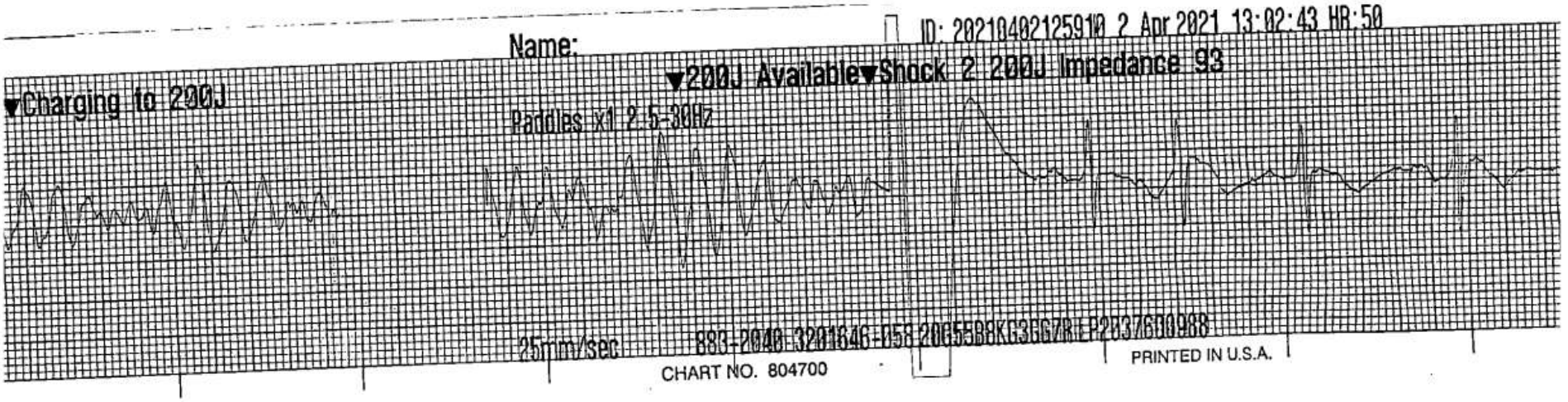
Sinus or ectopic atrial tachycardia
Consider right atrial enlargement
LAD, consider left anterior fascicular block
Anterior infarct, acute (LAD)
Prolonged QT interval
COMPARED TO ECG 03/23/2021 19:46:56
NO SIGNIFICANT CHANGES

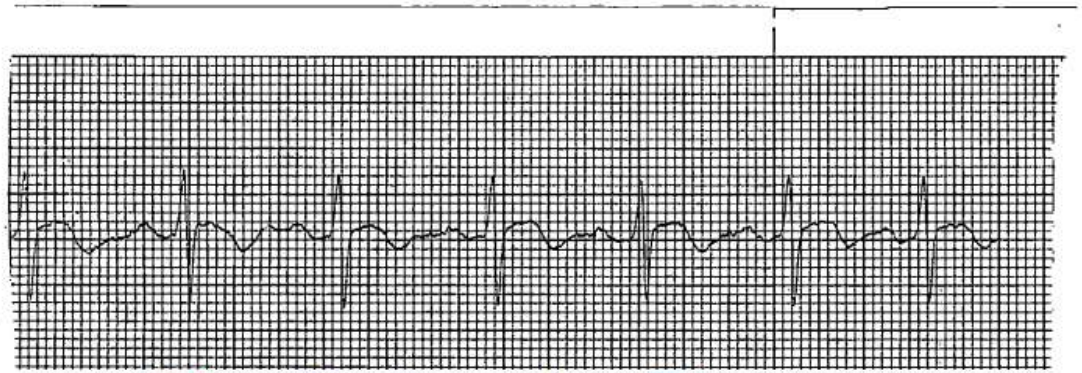
Req Provider: Joshua Dietzer

- Abnormal ECG -

Unconfirmed Diagnosis




Name:
ID: 20210402125910 2 Apr 2021 13:02:43 HR:50
▼Charging to 200J
▼200J Available▼Shock 2 200J Impedance 93
Paddles x1 2.5-30Hz
25mm/sec
CHART NO. 804700
PRINTED IN U.S.A.

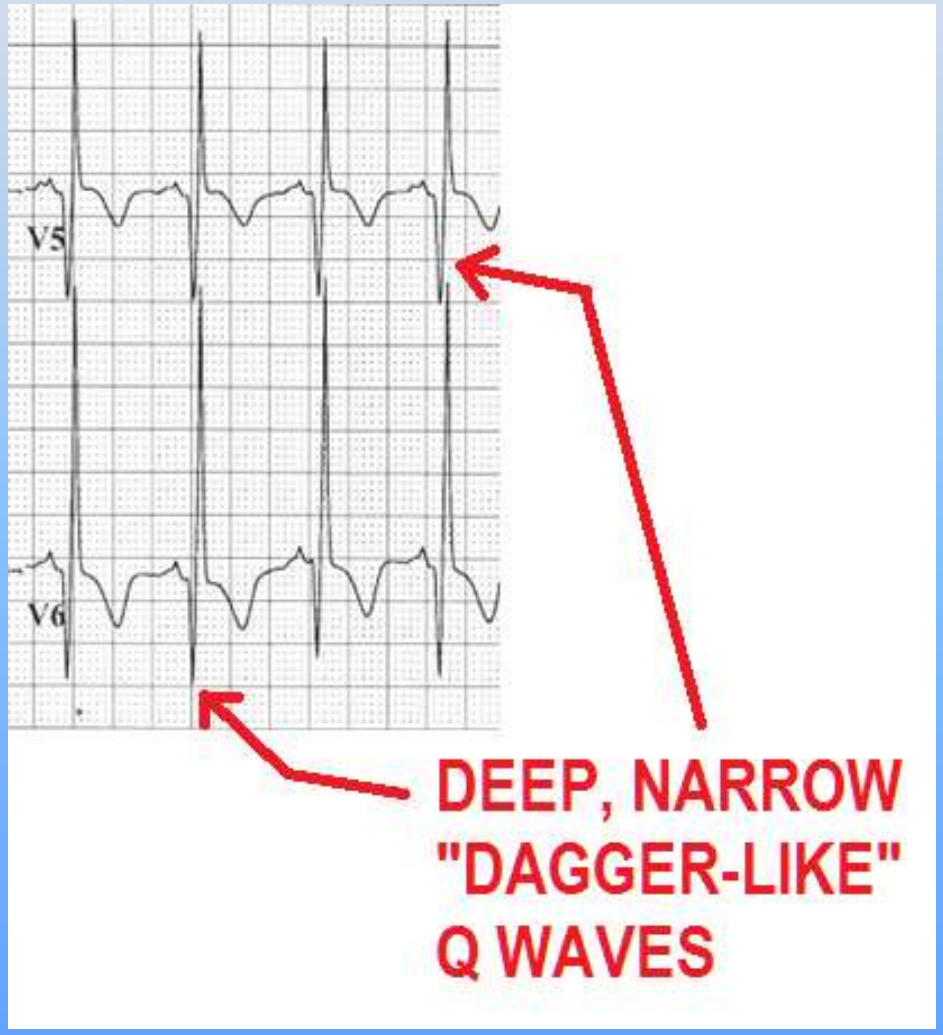


Page 1 of 1

# ECG Indicators: Hypertrophic Cardiomyopathy

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

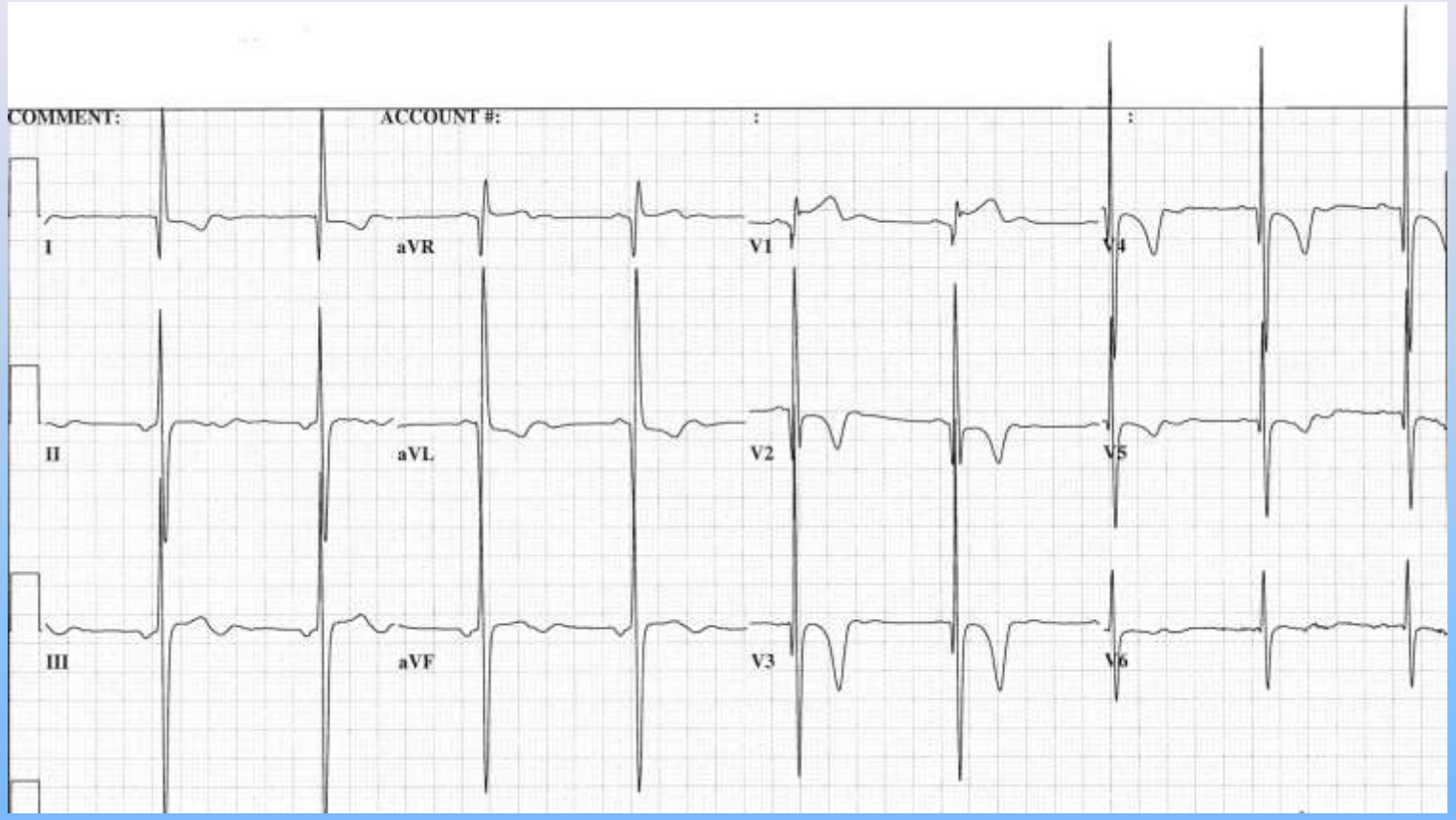
# ECG Indicators:
# Hypertrophic Cardiomyopathy



# 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

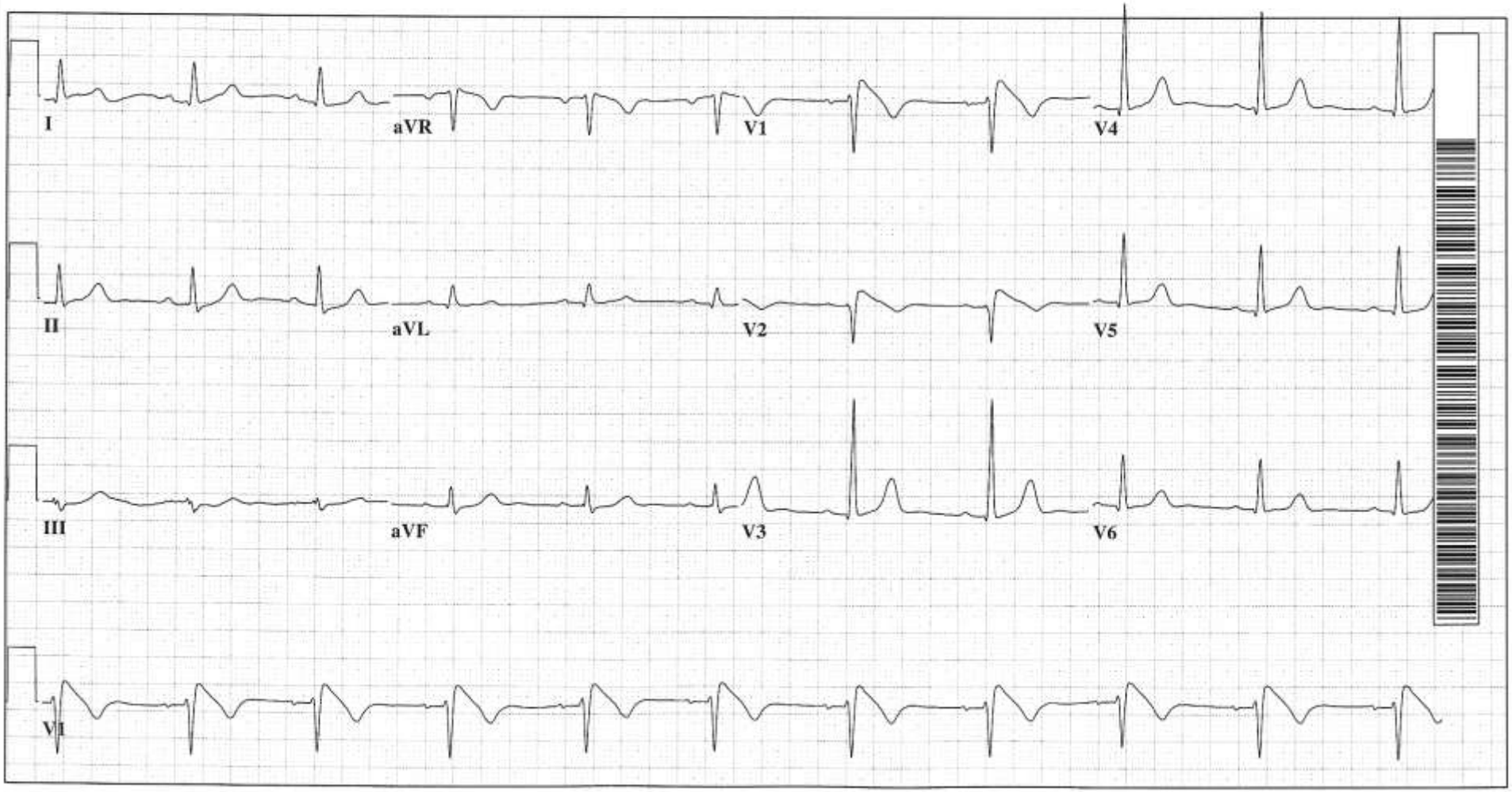
Room:C4A
Loc:3 Option:23

| | | |
|---|---|---|
| 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

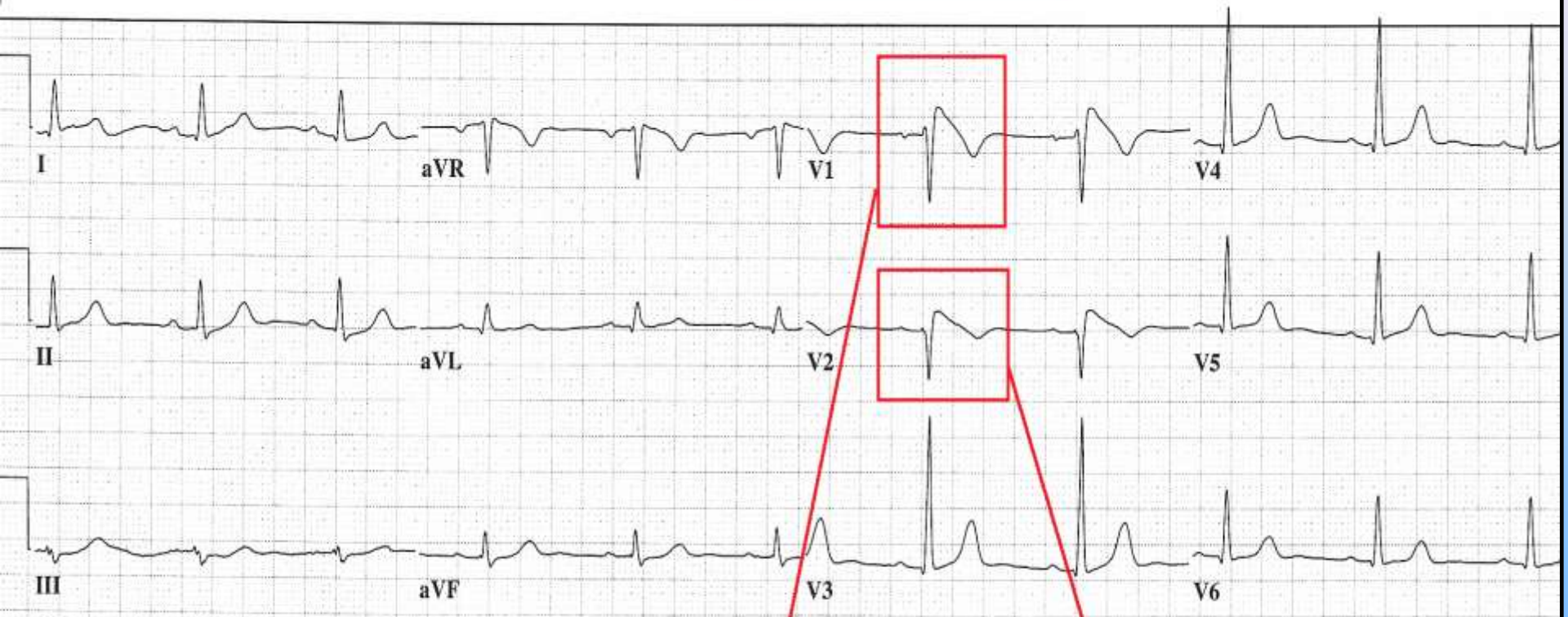
EID:46 EDT:

37 yr
Female Caucasian

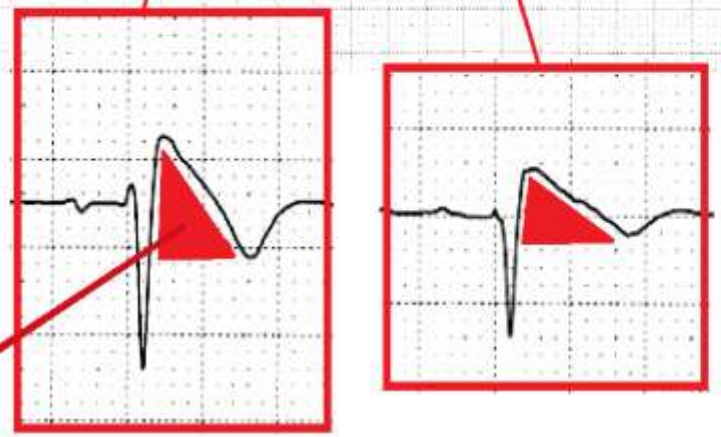
| | | |
|---|---|---|
| Vent. rate | 62 | BPM |
| PR interval | 180 | ms |
| QRS duration | 88 | ms |
| QT/QTc | 418/424 | ms |
| P–R–T axes | 37 22 | 47 |

Normal sinus rhythm
Normal ECG
No previous ECGs available

NOTE COMPUTER INTERPRETATION !



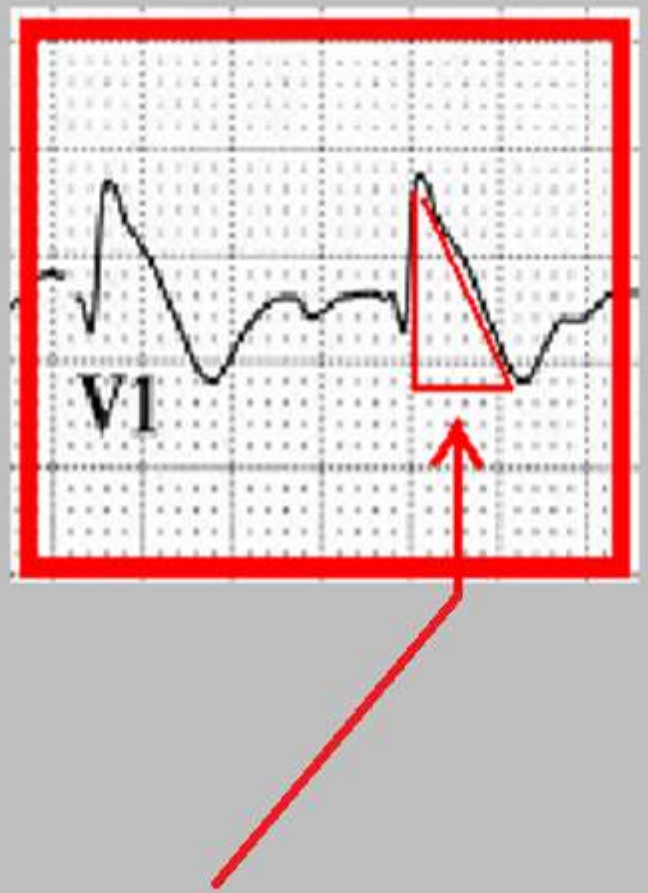

THIS PATIENT EXHIBITS A "CLASSIC" TYPE I BRUGADA SYNDROME ECG PATTERN:
- ELEVATED J POINTS IN V1, V2
- DOWNSLOPING "COVED" ST SEGMENT
- INVERTED T WAVE.



***NEVER FORGET THE "TRIANGULAR" SHAPE !***

# BRUGADA SYNDROME

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



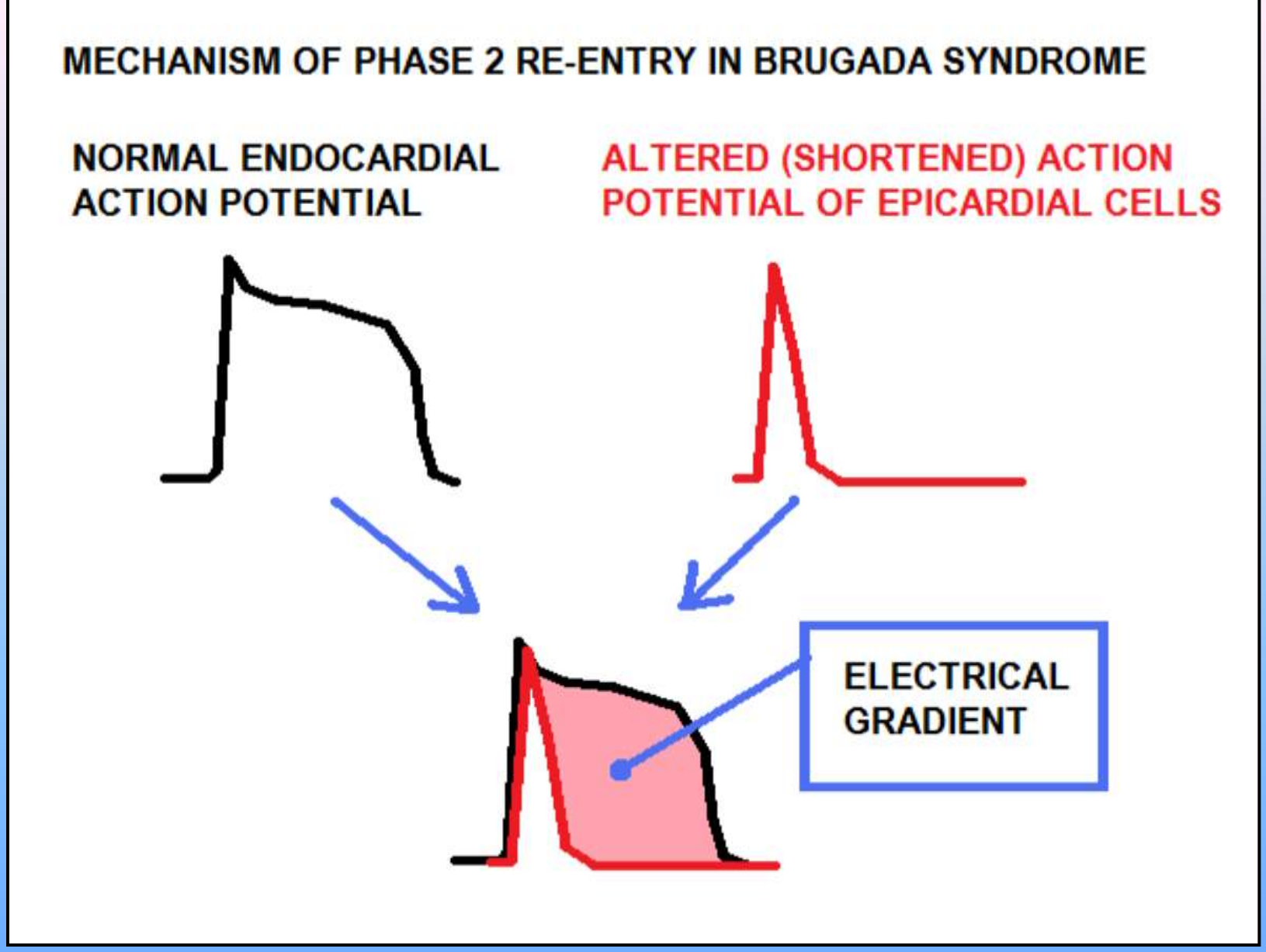
# PATTERNS of S-T ELEVATION :

## BEWARE of the

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

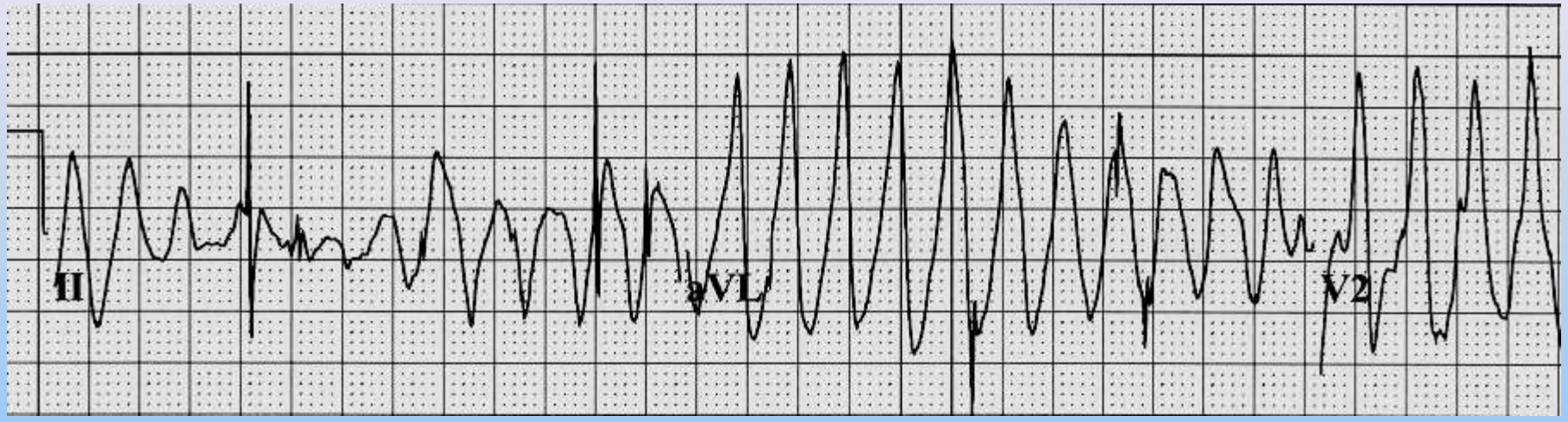
THINK - -

## BRUGADA SYNDROME




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

# Brugada / Long QT Syndromes cause:




**Torsades de Pointes:**

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

# TREATMENT OF TORSADES de POINTES per AHA ACLS 2015:

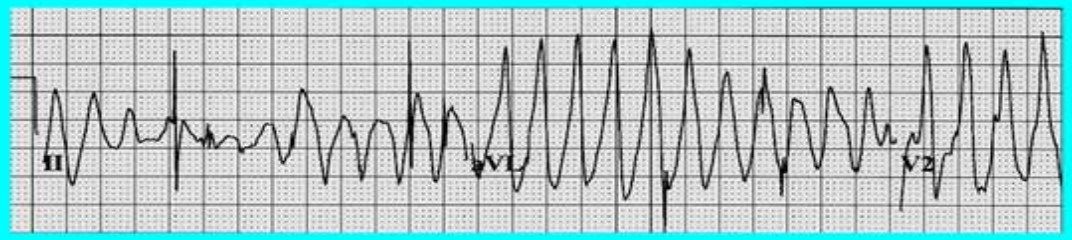
-TRANSIENT: MAGNESIUM SULFATE 1 – 2 gm IV infusion over 5 – 60 minutes.

-PERSISTENT, PATIENT UNSTABLE: DEFIBRILLATION

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

# WIDE COMPLEX TACHYCARDIA
## TORSADES de POINTES

( QRS > 120 ms )




**ABC s**

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

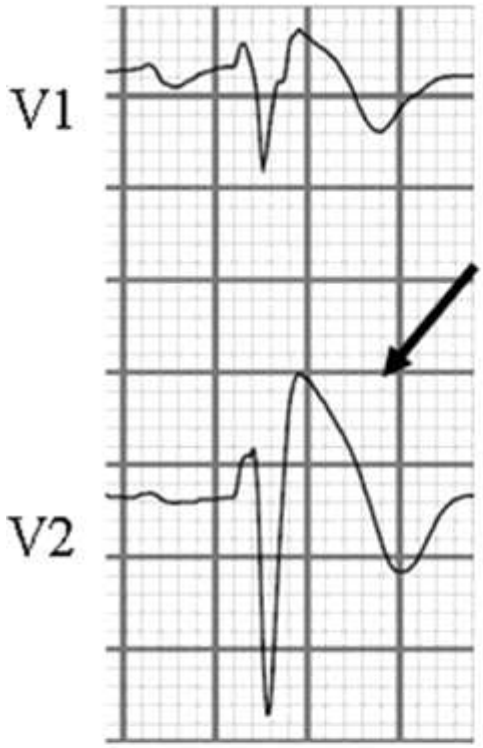
***DO NOT give PROCAINAMIDE, AMIODARONE, or SOTALOL to patients with TORSADES or POLYMORPHIC VT !!!***

OTHER CONSIDERATIONS:

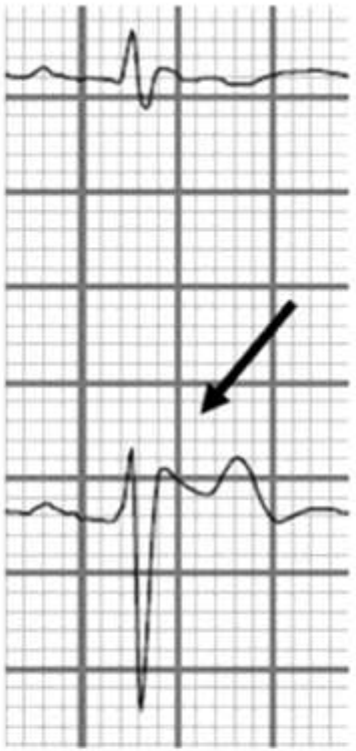
- EVALUATE BASELINE ECG RHYTHM FOR PRONGED Q-T INVERVAL.
- EVALUATE PATIENT'S MEDS FOR Q-T PROLONGING DRUGS
  . . . if PATIENT HAS BEEN RECEIVING ANY Q-T PROLONGING DRUGS, IMMEDIATELY DISCONTINUE AND CONTACT PHYSICIAN STAT.
- EVALUATE PATIENT HISTORY FOR PREVIOUS EVENTS OF "SYNCOPE OF UNKOWN ETIOLOGY"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

REPORT ANY ABNORMAL FINDINGS TO PHYSICIAN.

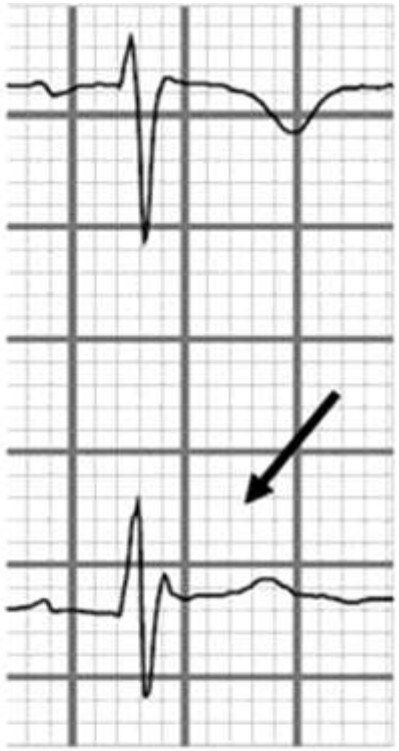
# ECG abnormality diagnostic or suspected of Brugada syndrome.




**Type 1:**
**Coved type**
**ST-segment elevation**



**Type 2:**
**saddle-back type**
**ST-segment elevation**



**Type 3:**
**Saddle-back type**
**"ST-segment elevation"**

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

American Heart Association.

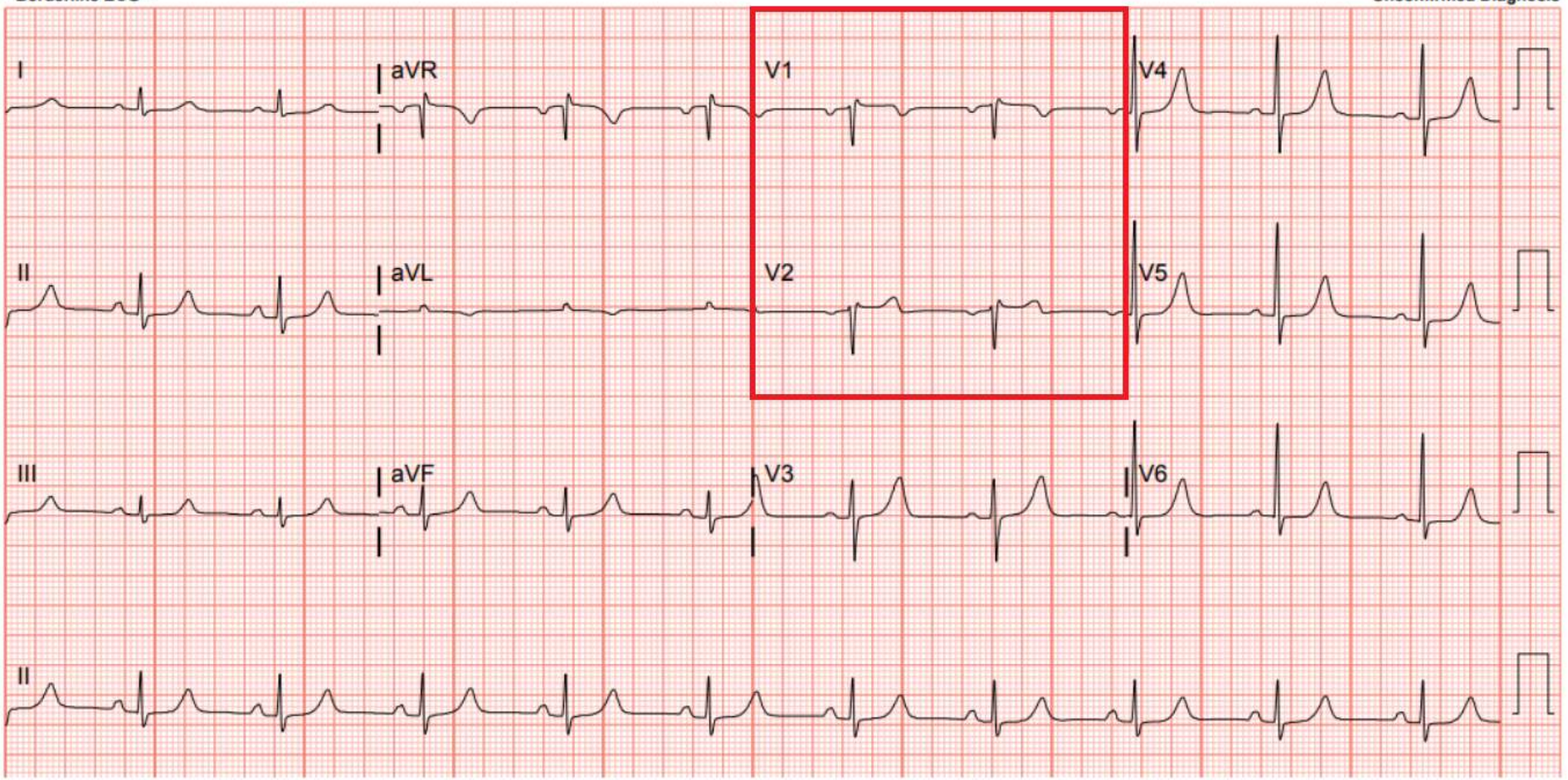
| | | |
|---|---|---|
| Rate | 63 | Sinus rhythm |
| PR | 168 | Probable left atrial enlargement |
| QRSd | 85 | RSR' in V1 or V2, right VCD or RVH |
| QT | 440 | COMPARED TO ECG 09/27/2019 02:43:44 |
| QTc | 451 | RIGHT VENTRICULAR HYPERTROPHY NOW PRESENT |
| --Axis-- | | |
| P | 66 | |
| QRS | 27 | |
| T | 67 | |

Req Provider: R POTU

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

- Borderline ECG -

Unconfirmed Diagnosis



12/23/1962 56 yrs | Female | Account # [redacted] | Dept ED | Room ED07 | Tech jk

RX
DX

| | |
|---|---|
| Rate | 69 |
| PR | 161 |
| QRSd | 109 |
| QT | 397 |
| QTc | 426 |
| --Axis-- | |
| P | 8 |
| QRS | -3 |
| T | 58 |

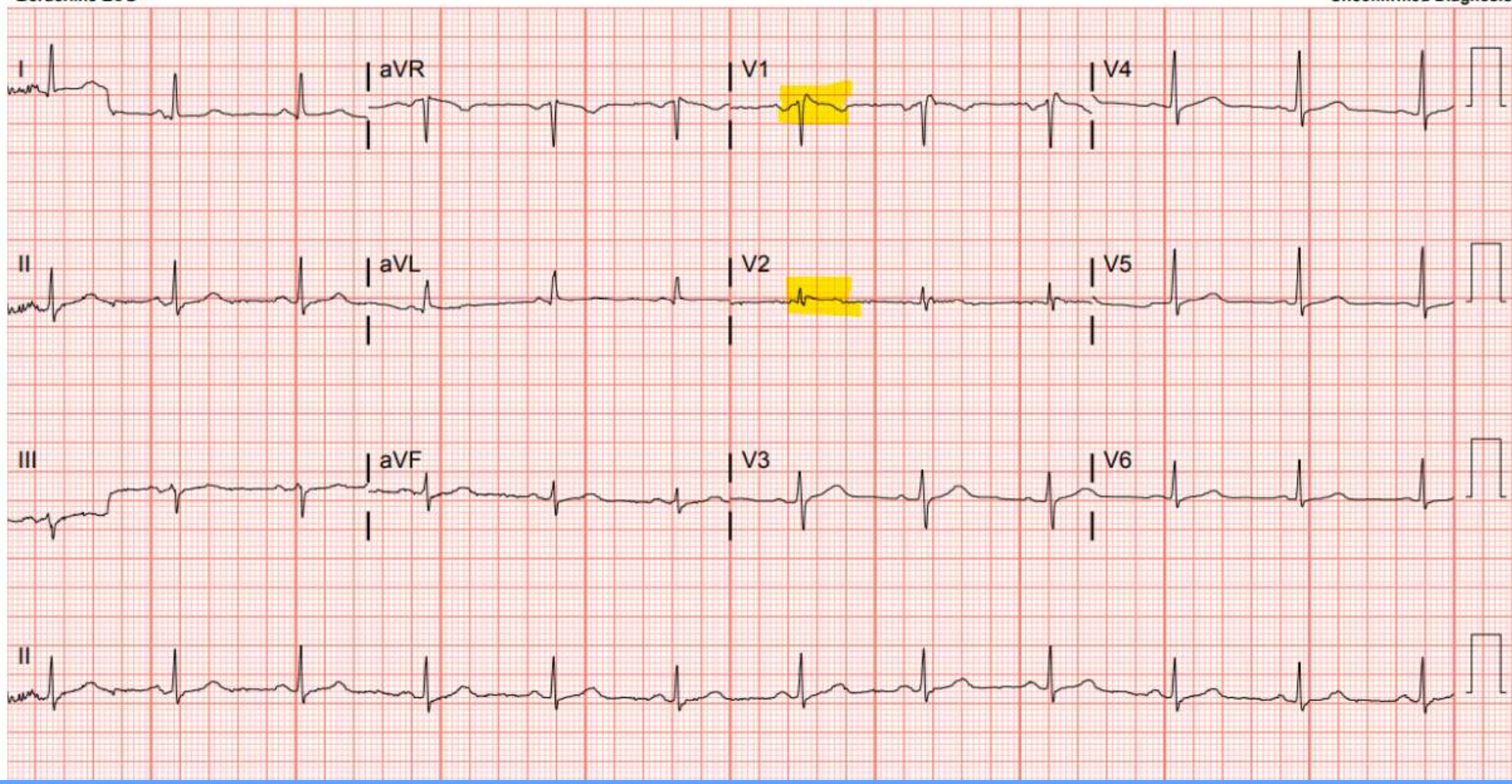
Sinus rhythm
Probable left atrial enlargement
RSR' in V1 or V2, right VCD or RVH
NO PREVIOUS ECG AVAILABLE FOR COMPARISON

Req Provider: MODINAT BALOG

## Brugada Type 3

- Borderline ECG -

Unconfirmed Diagnosis



Pat ID [redacted]
10/04/2019 10:56:31
[redacted] 56 yrs
[redacted] Female
Account #
Bayfront Health Seven Rivers ED
RX
DX
Dept
Room
Tech tb
Req Provider:

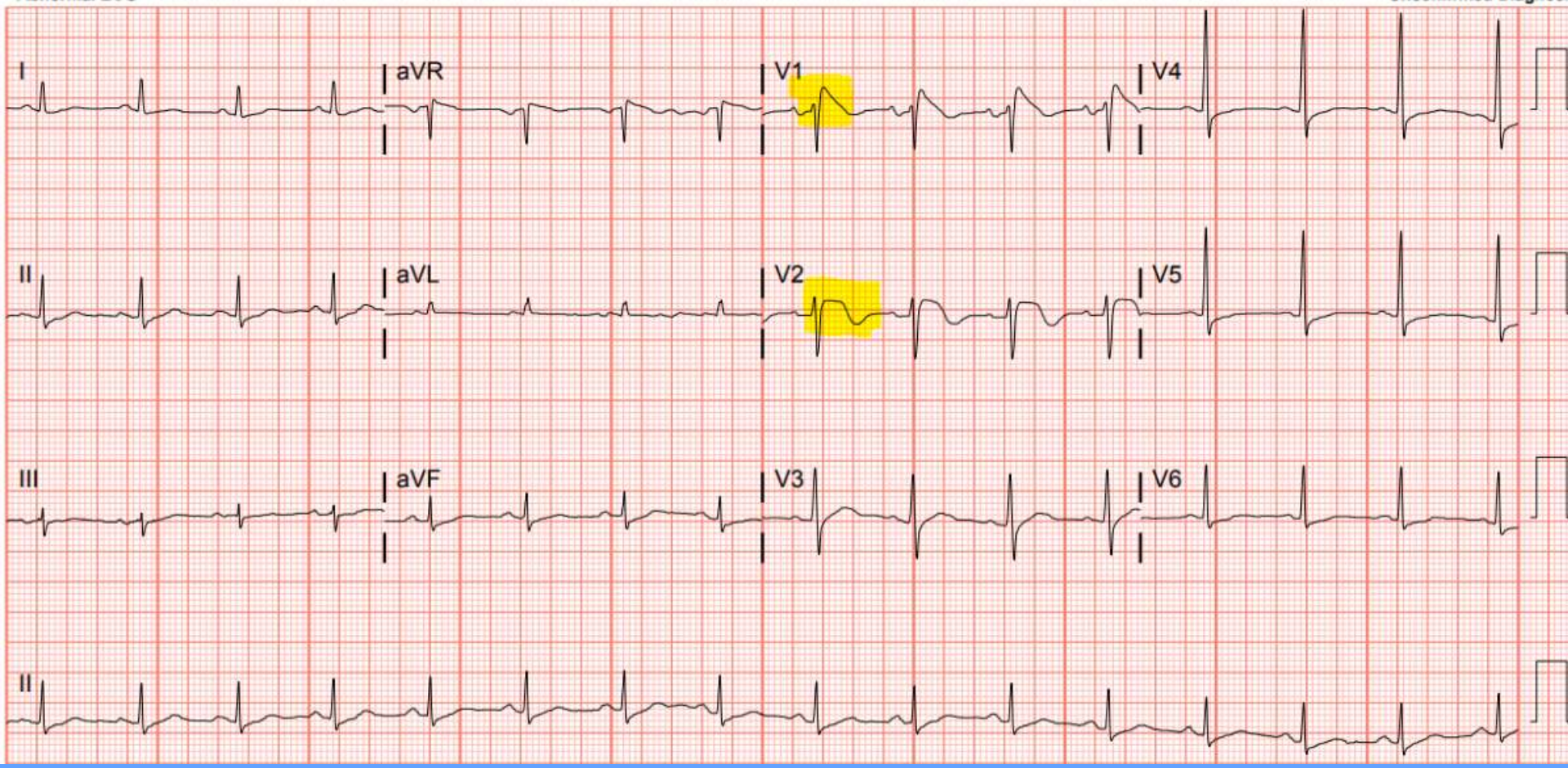
| | | |
|---|---|---|
| Rate | 93 | Sinus rhythm |
| PR | 150 | Probable left atrial enlargement |
| QRSd | 66 | Anteroseptal infarct, acute |
| QT | 419 | Prolonged QT interval |
| QTc | 522 | Baseline wander in lead(s) II,III,aVR,aVL,aVF |
| --Axis-- | | COMPARED TO ECG 10/04/2019 10:50:56 |
| P |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

**Patient developed fever 102.6 degrees.**

**Brugada Type 1 pattern now present.**

- Abnormal ECG -

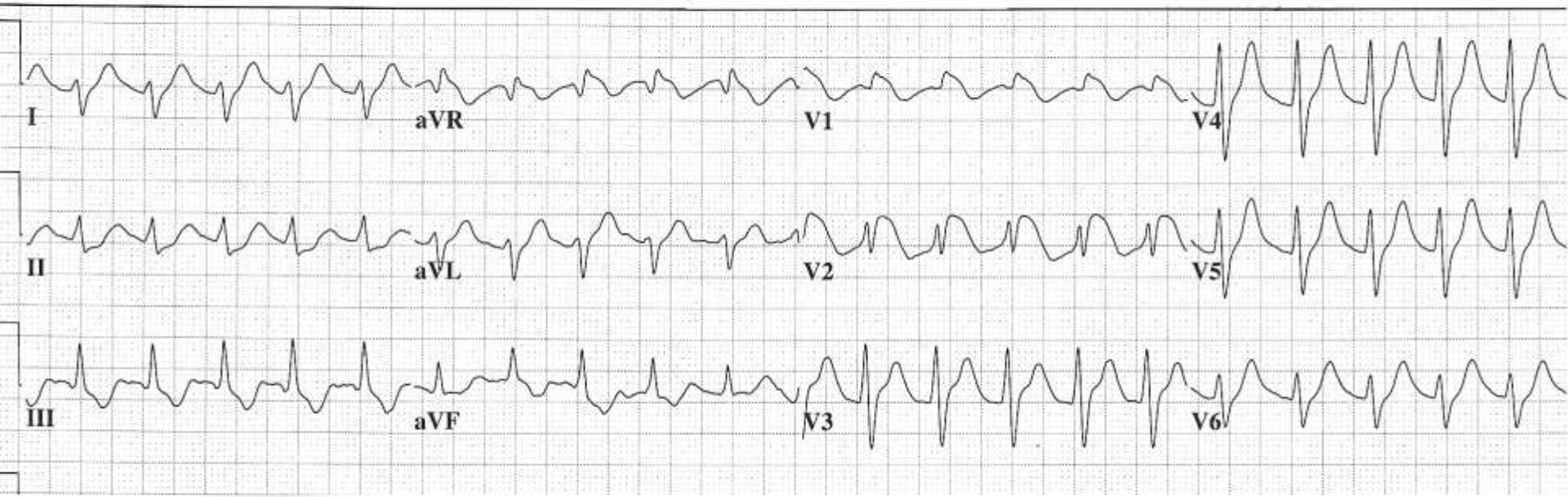
Unconfirmed Diagnosi



**33 y/o FEMALE**

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

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




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

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

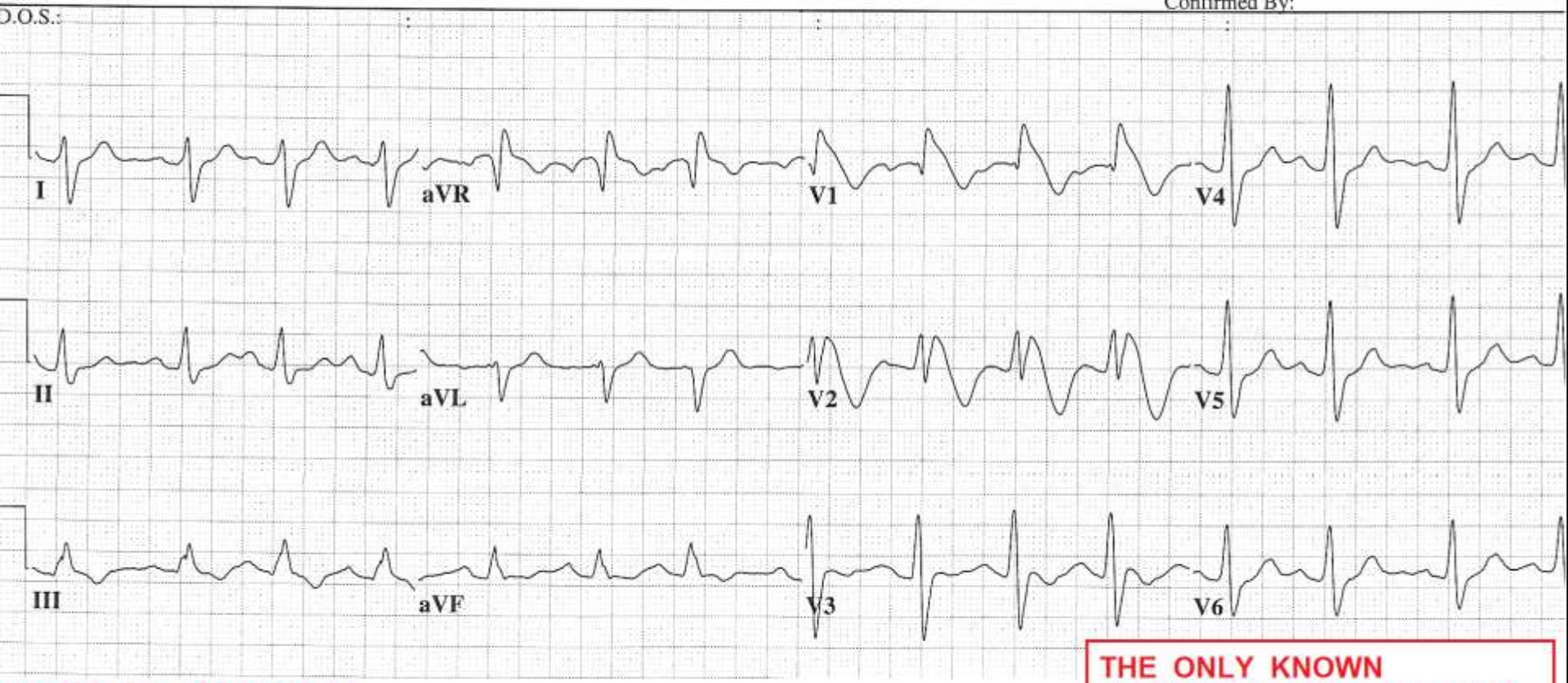
**VISIT: www.BRUGADA.org FOR MORE INFORMATION.**

# 42 y/o FEMALE

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

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

Confirmed By:



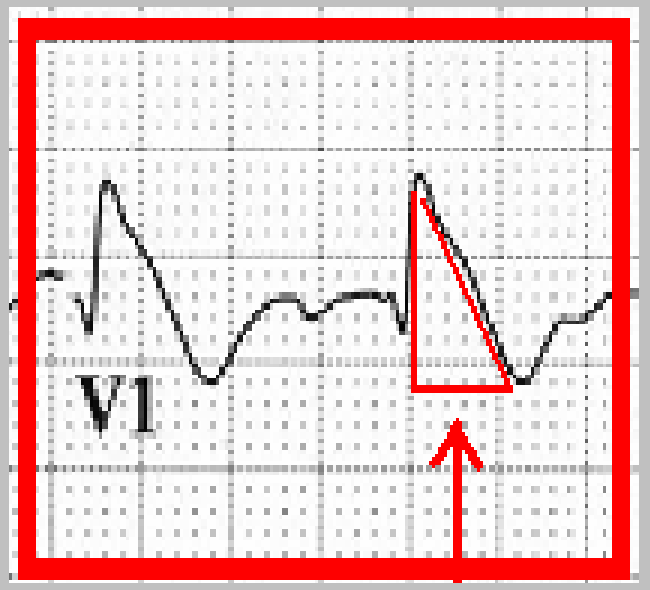

**BRUGADA SYNDROME.**

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

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

# BRUGADA SYNDROME

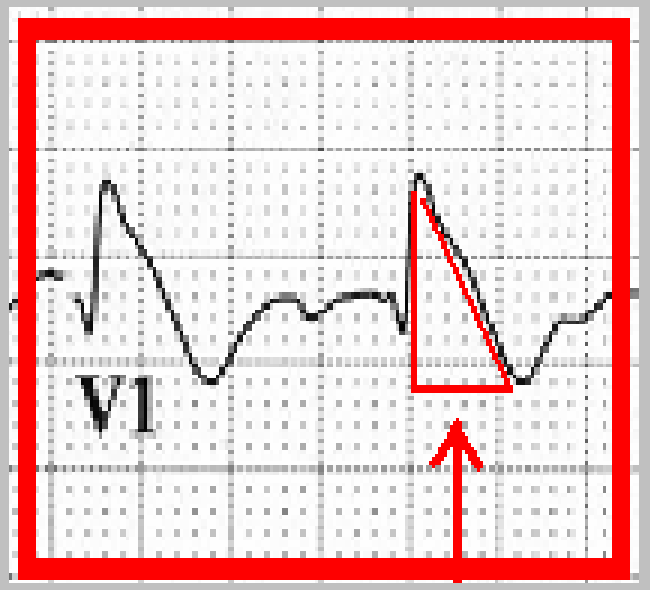
- GENETIC DISORDER - GENE SCN5A, which encodes CARDIAC SODIUM CHANNELS.
- CAUSES EARLY RIGHT VENTRICULAR SUB-EPICARDIAL REPOLARIZATION




- CAUSES RUNS OF TORSADES de POINTES, and SUDDEN DEATH from TORSADES and V-FIB.
- IS BELIEVED TO CAUSE 4 - 12 % of ALL SUDDEN DEATHS, and 50 % of ALL CARDIAC DEATHS where pt. has a STRUCTUALLY NORMAL HEART.

# BRUGADA SYNDROME

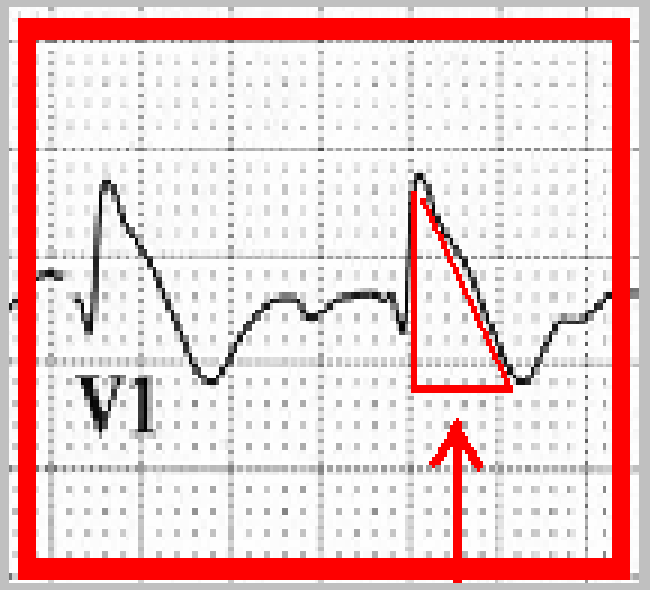
- SEVERAL VARIATIONS of this disorder are known to exist.
- CONCEALED and NON-CONCEALED.
- The NON-CONCEALED version HAS THE V1-V3 abnormality VISIBLE at all times.




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

# BRUGADA SYNDROME

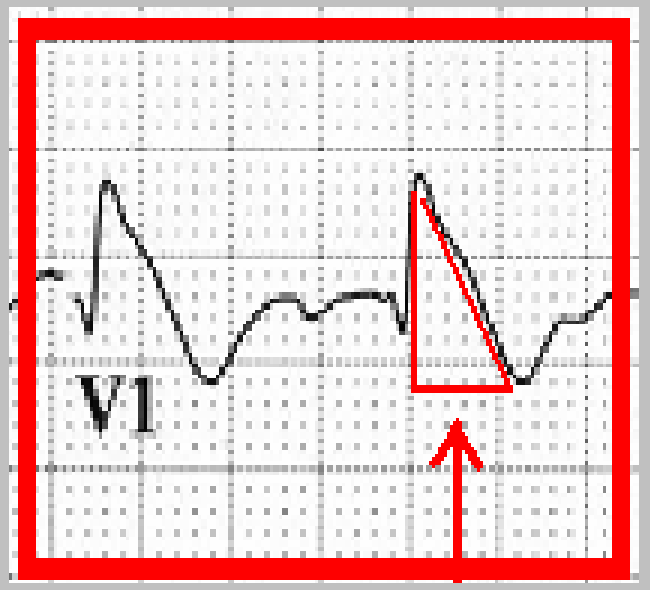
- YOUNG MALES of SOUTHEAST ASIAN DESCENT are in HIGH RISK GROUP, however this disorder affects ANY RACE or GENDER.




- BRUGADA SYNDROME is HEREDITARY.
- SUSPECT BRUGADA SYNDROME in patients with FAMILY HISTORY of BRUGADA / SUDDEN DEATH, and/or TORSADES.

# BRUGADA SYNDROME - TESTING

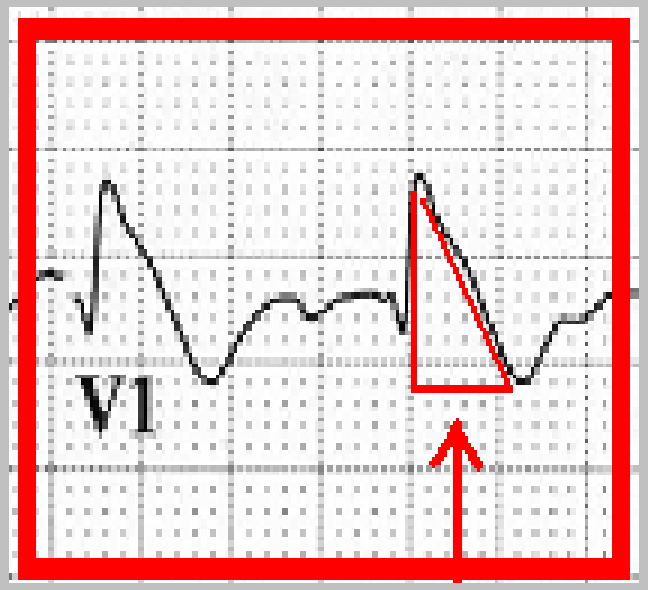
- For CONCEALED cases, a drug study of AJMALINE, FLECAINIDE, or PROCAINAMIDE can UNMASK the "tell-tale" TRIANGULAR COMPLEXES of V1 and V2.




- IN EP STUDIES, a PROLONGED H-V INTERVAL may be observed.
- GENETIC TESTING is performed by THE RAMON A. BRUGADA FOUNDATION.

# BRUGADA SYNDROME - TREATMENT

ICD implantation is the only known effective treatment to date.



www.BRUGADA.org

# Arrhythmogenic Right Ventricular Dysplasia

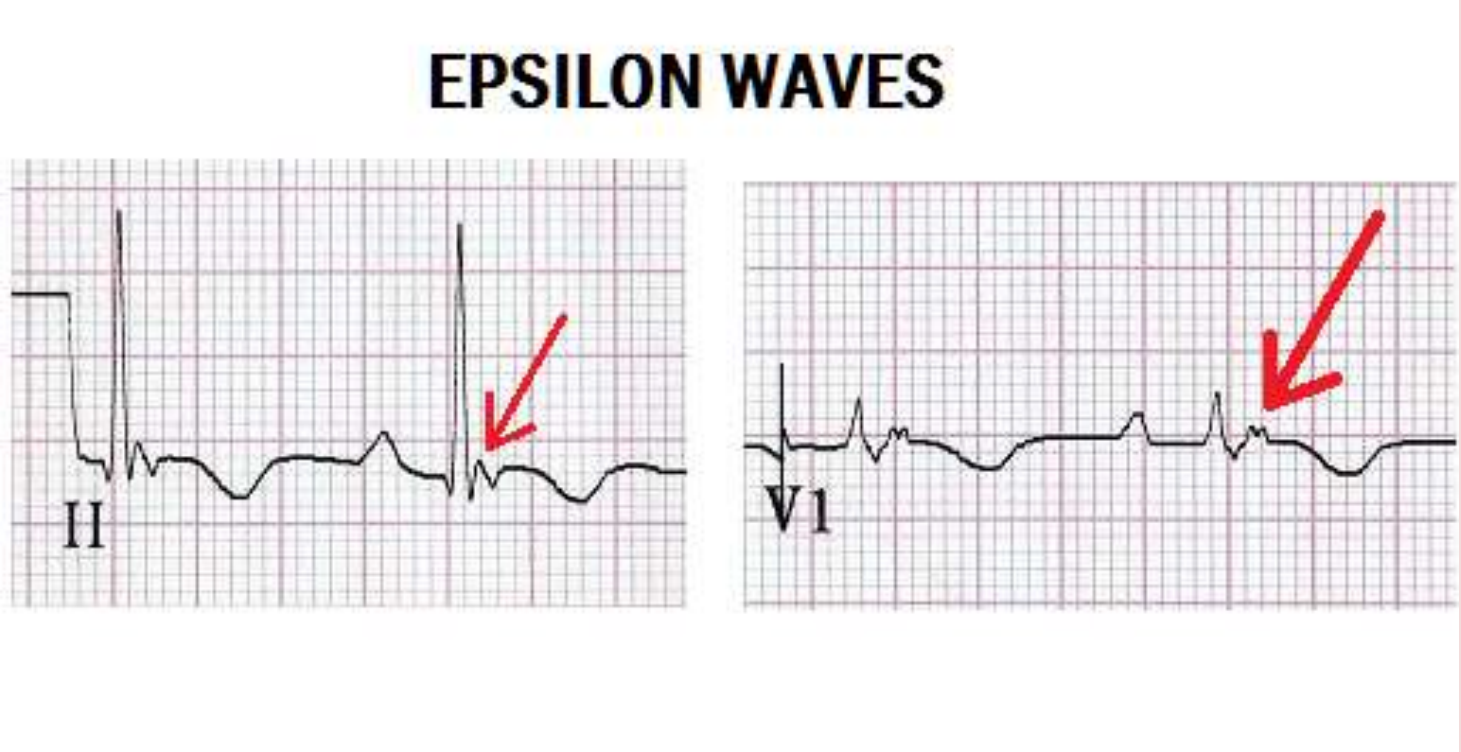
- **A genetically acquired myocardial disease associated with paroxysmal ventricular arrhythmias and sudden cardiac death.**
- **Characterized pathologically by fibro-fatty replacement of the right ventricular myocardium.**
- **The second most common cause of sudden cardiac death in young people (after HOCM), causing *up to 20% of sudden cardiac deaths in patients < 35 yrs of age*.**
- **Typically inherited as an autosomal dominant trait, with variable penetrance and expression (there is an autosomal recessive form called Naxos Disease, which is associated with woolly hair and skin changes).**
- **More common in men than women (3:1) and in people of Italian or Greek descent.**
- **Estimated to affect approximately 1 in 5,000 people overall.**

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

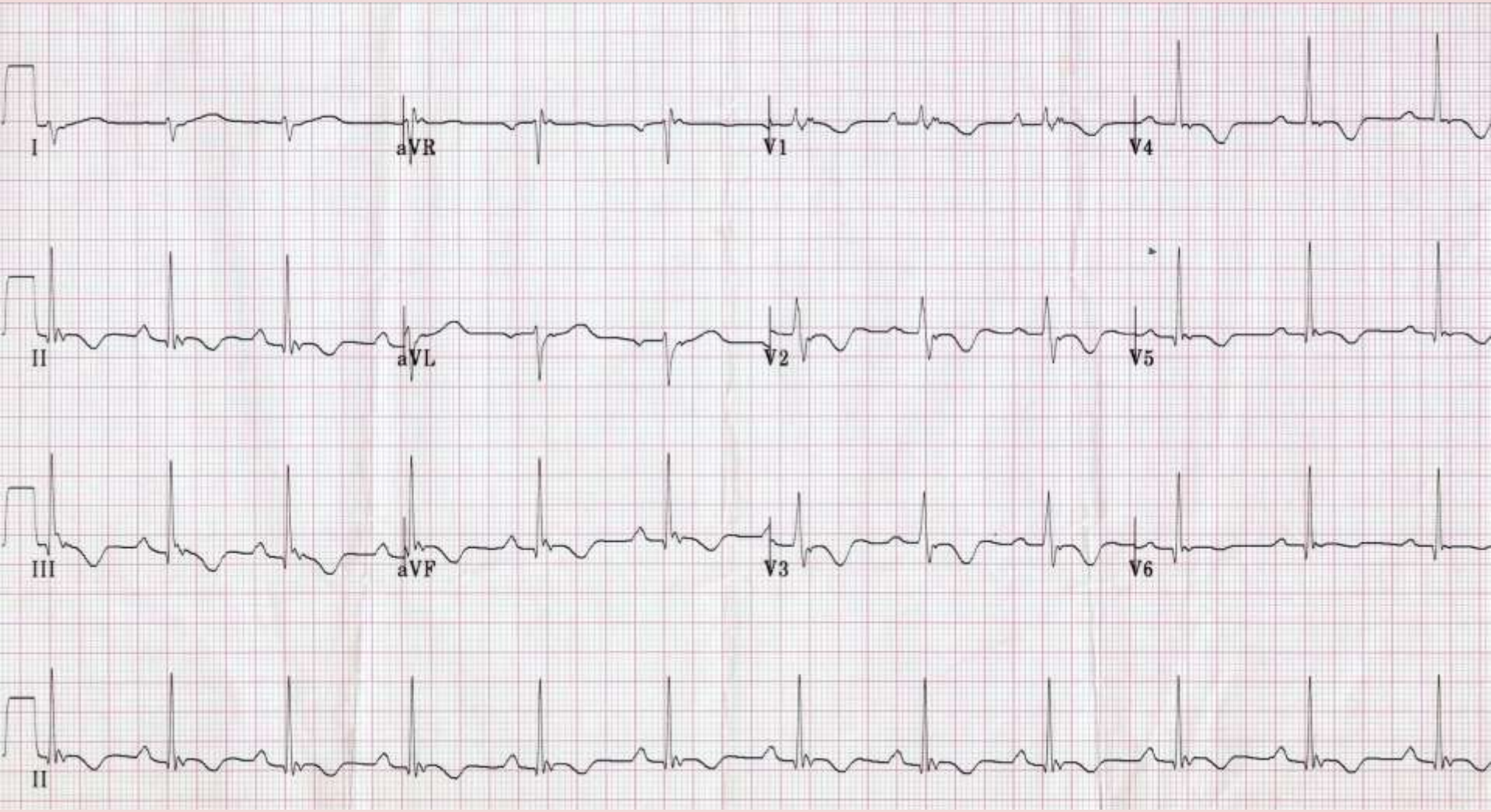
## Arrhythmogenic Right Ventricular (RV) Cardiomyopathy and/or Dysplasia:

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

# ARVD – 12 Lead ECG Indicators

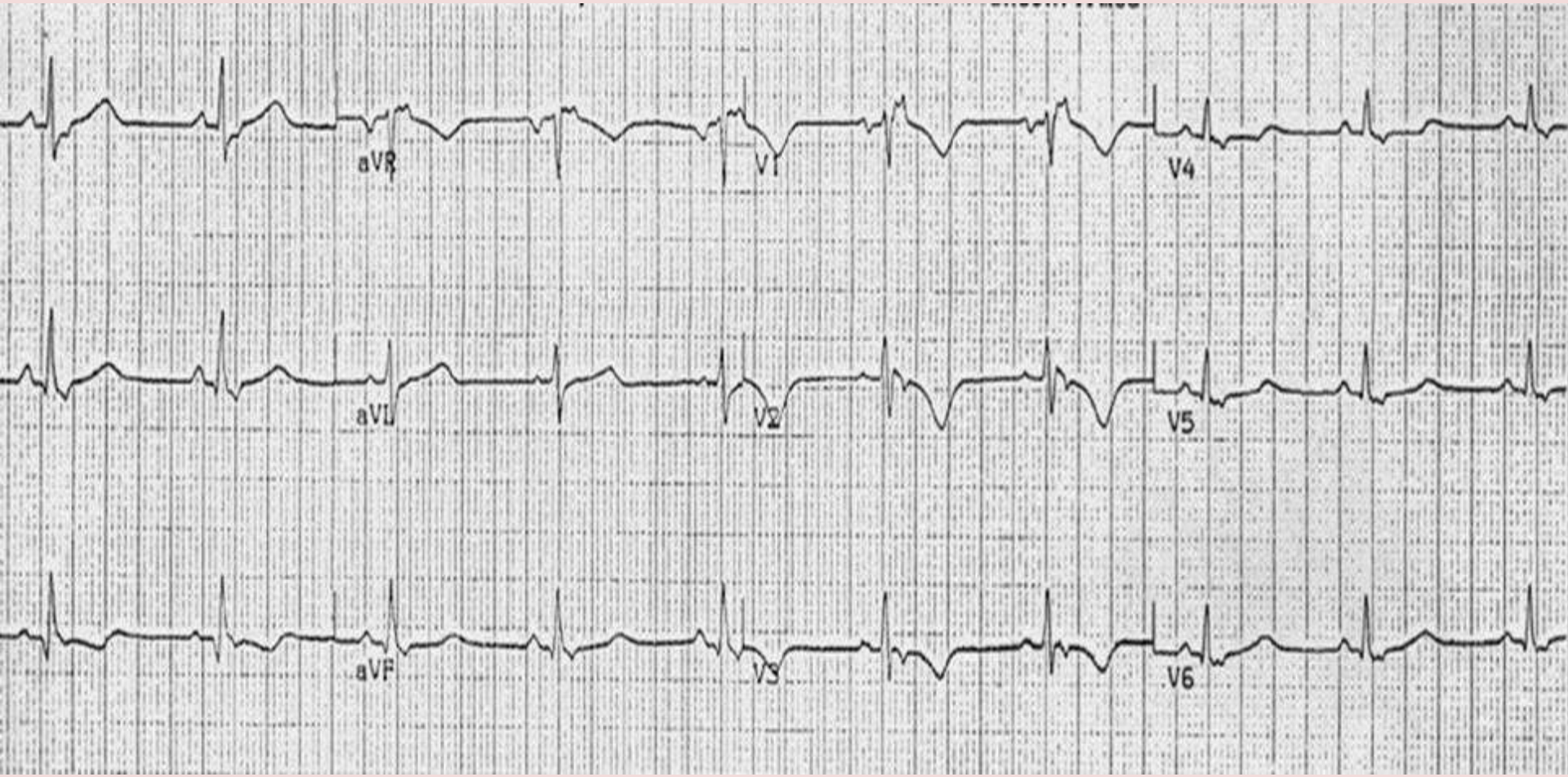


# ARVD ECG 1

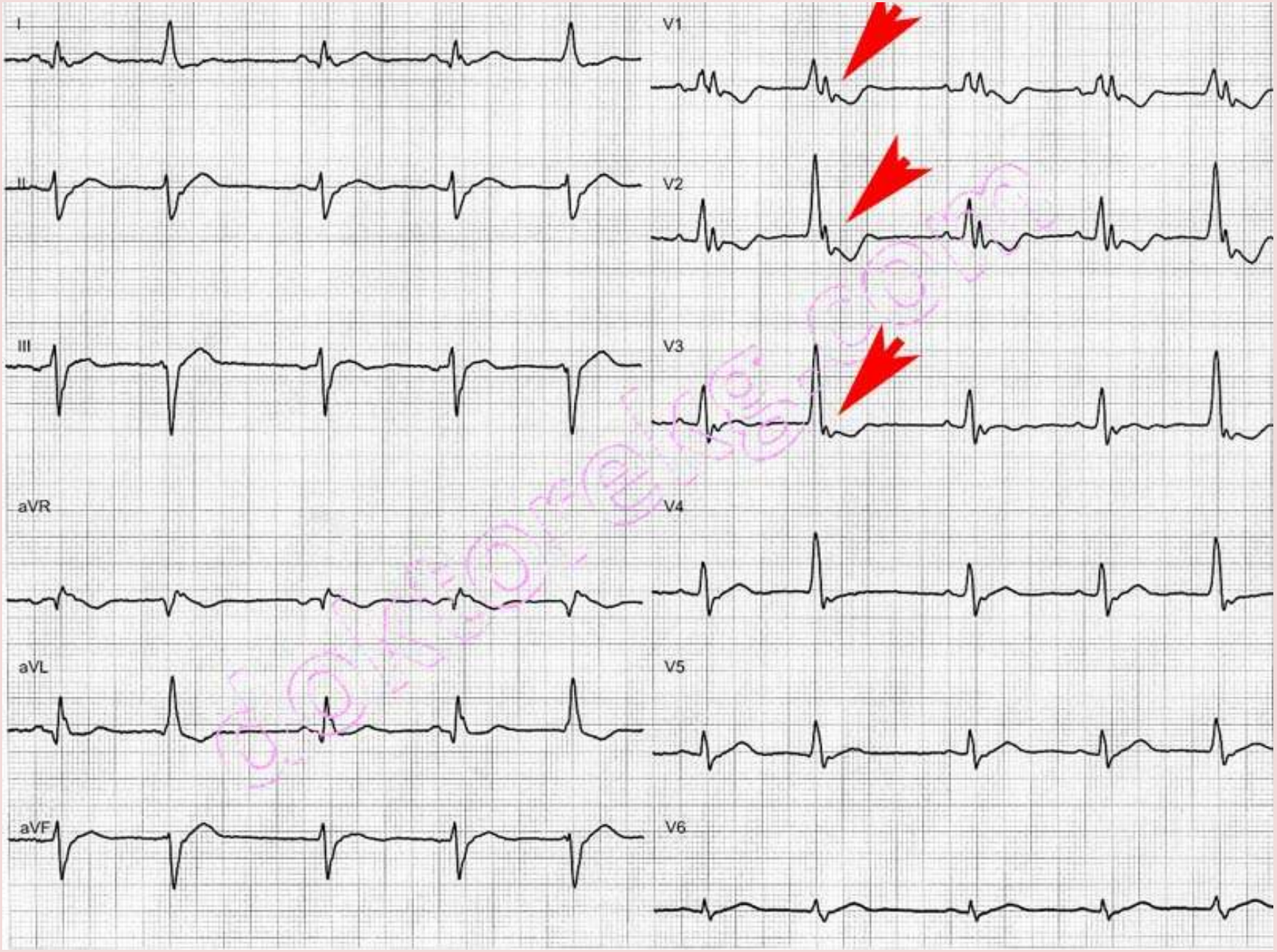



1. **“Incomplete RBBB” Pattern**
2. **V1, V2 Rs pattern**
3. **Inverted T waves, symmetrical, - Global**
4. **Epsilon’s waves**

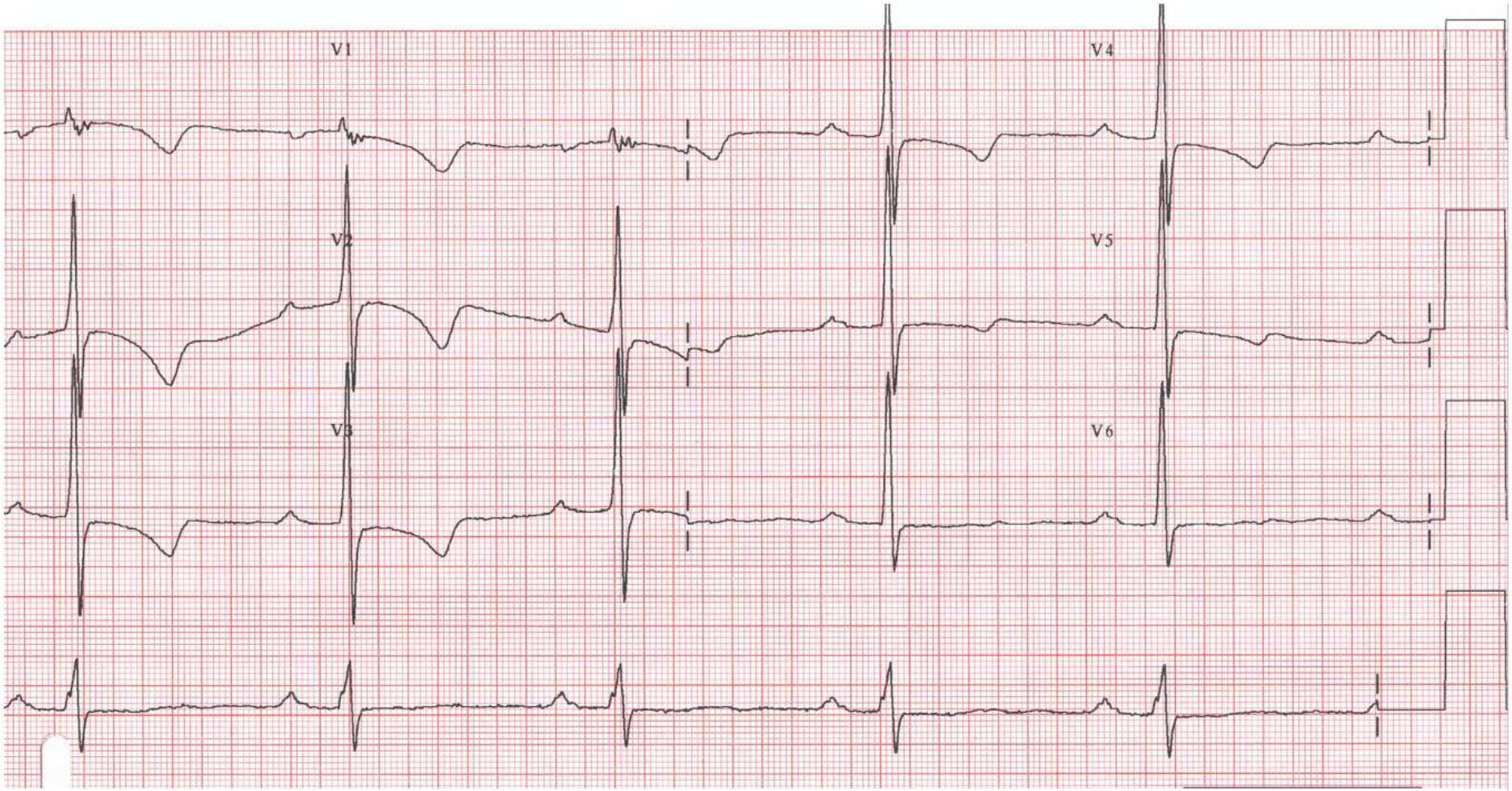
# ARVD ECG 2



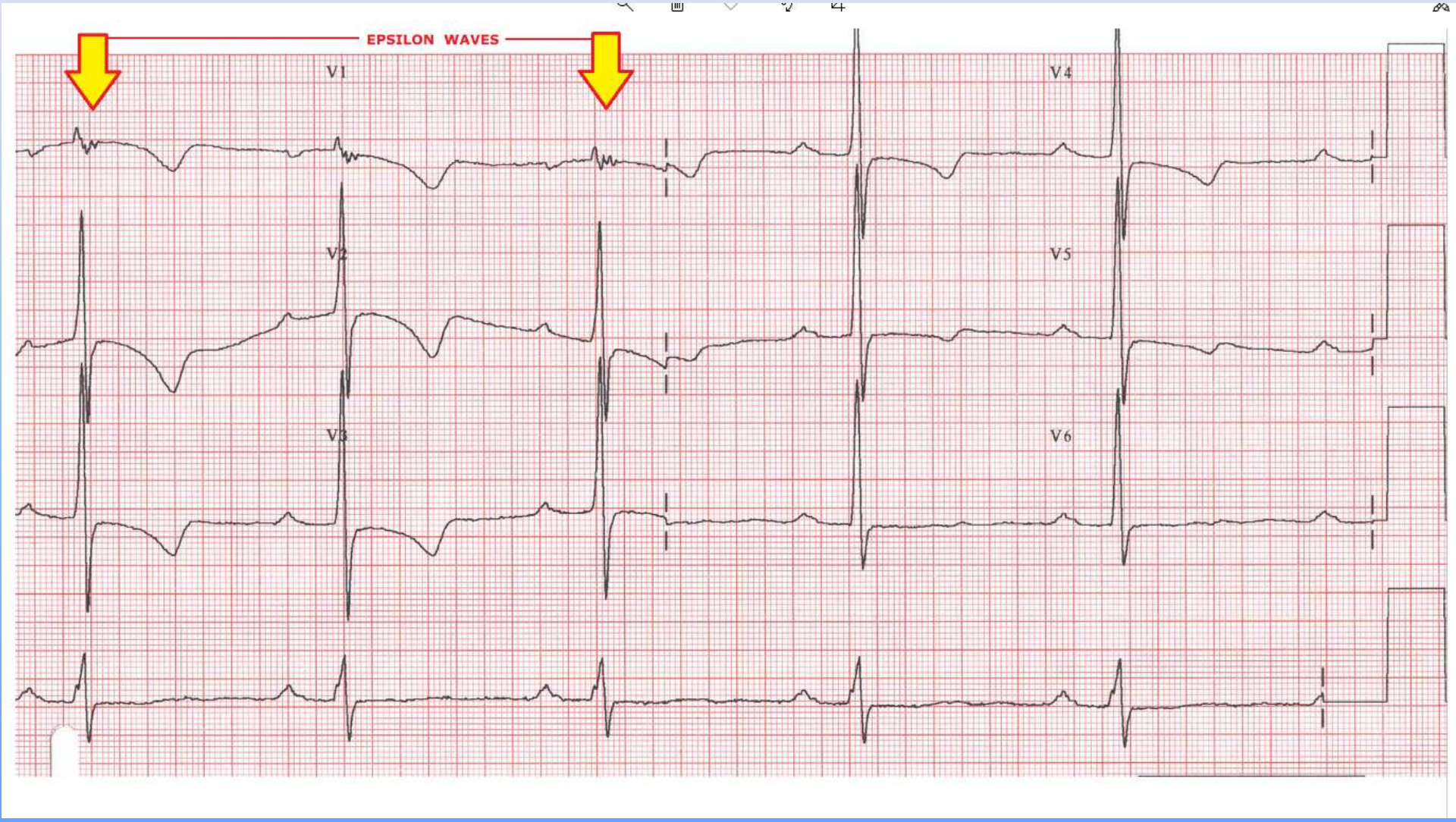

1. “Incomplete RBBB” Pattern
2. V1, V2 Rs pattern
3. Inverted T waves, symmetrical, - Global
4. Epsilon’s waves

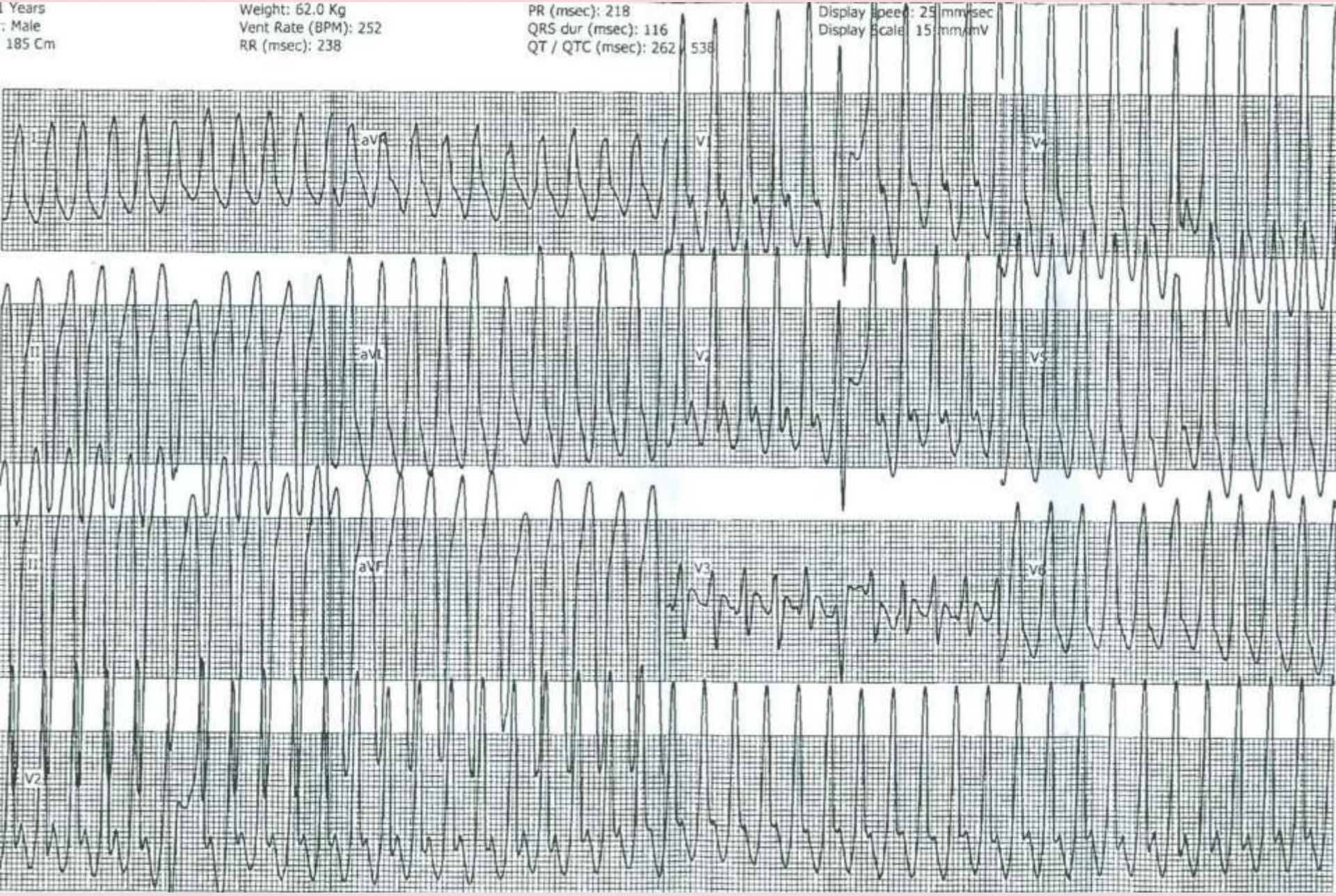

I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

# Would you spot the Epsilon's Waves?

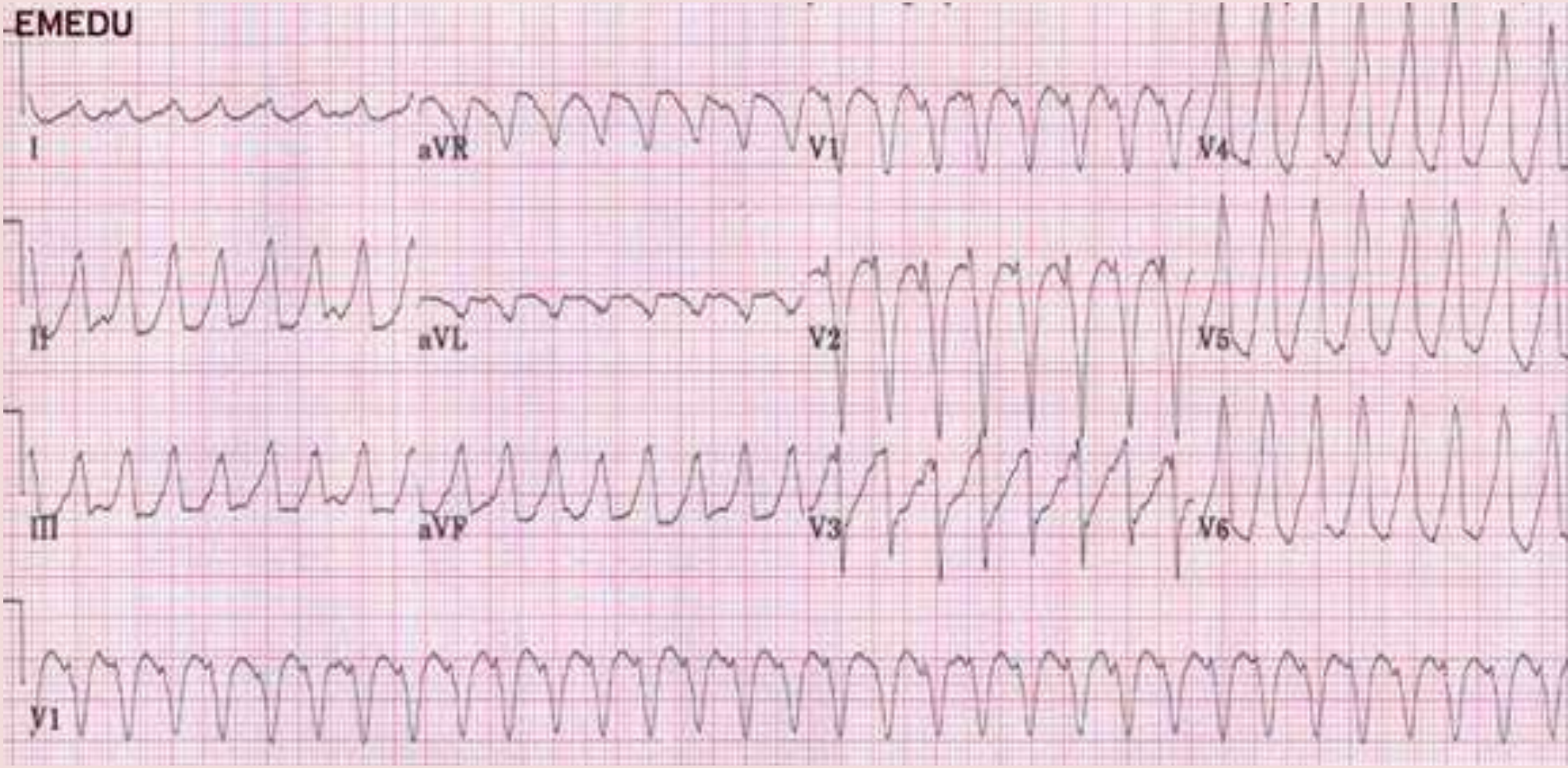


# BHSR Patient – Epsilon's Waves




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

# ARVD INDUCED VT



# Evidence Based Reference Sources

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

# Evidence Based Reference Sources, cont'

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

# Other Reference Sources:

www.JACC.org

http://circ.ahajournals.org/


www.SADS.org

# The New England Medical Journal

# THE ACUTE CORONARY SYNDROMES

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

# 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: Evaluate QRS Width**

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

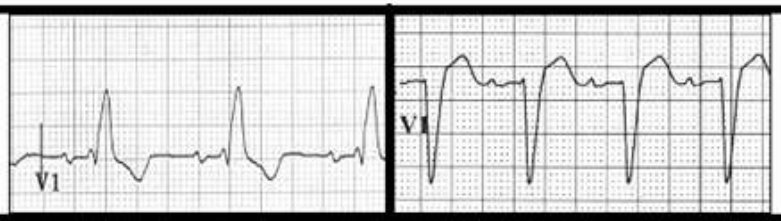
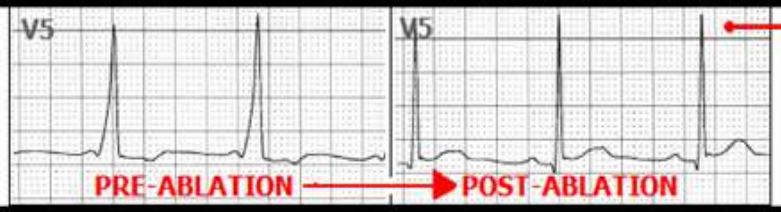
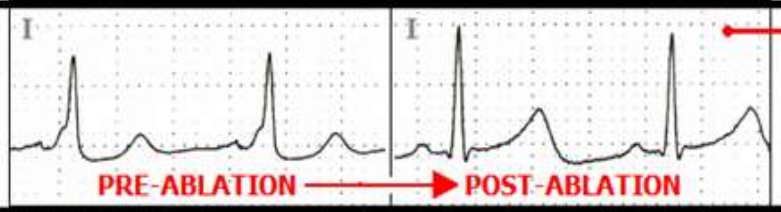
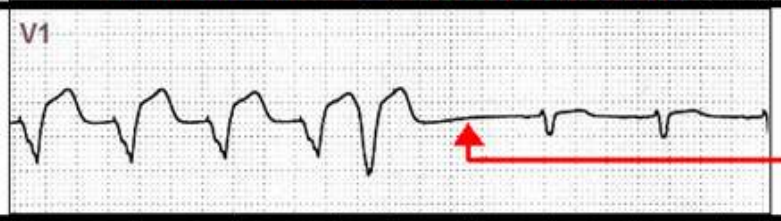
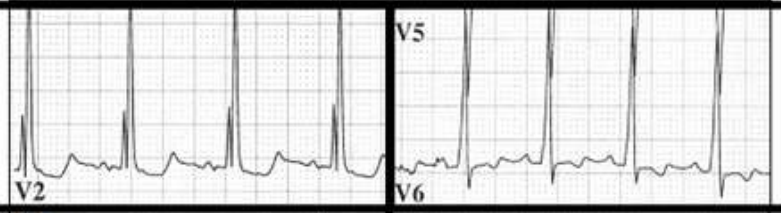
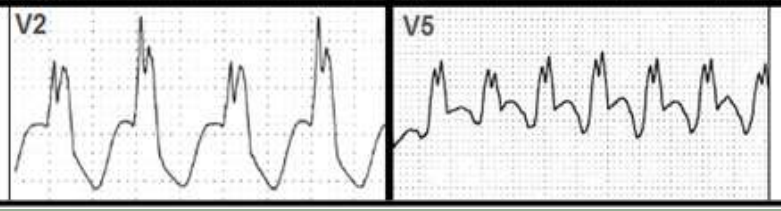
# STEP 1 – evaluate QRS width:

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

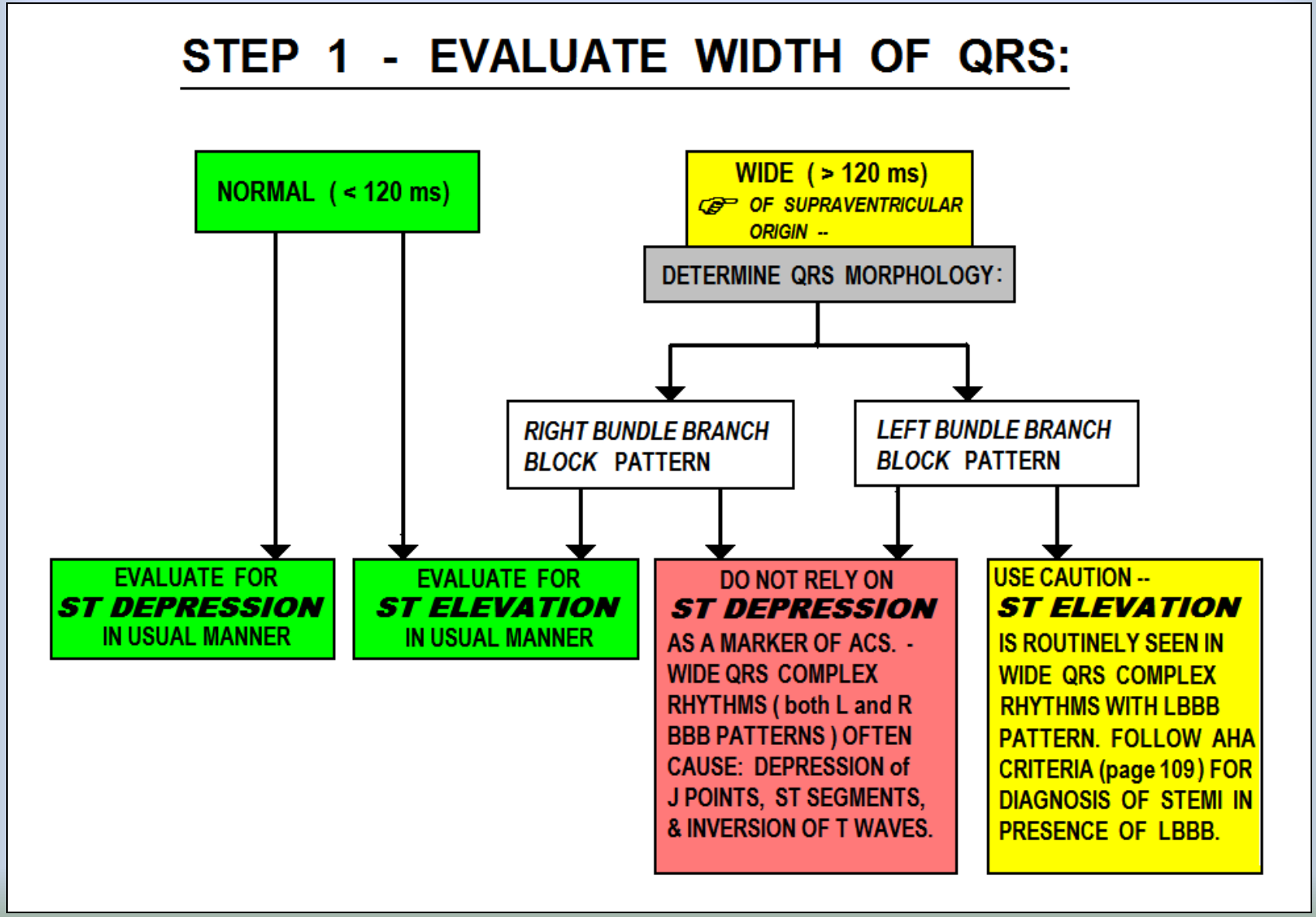
# STEP 1 – evaluate QRS width:

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

# CONDITIONS THAT INCREASE QRS DURATION RESULT IN SECONDARY REPOLARIZATION ABNORMALITIES:

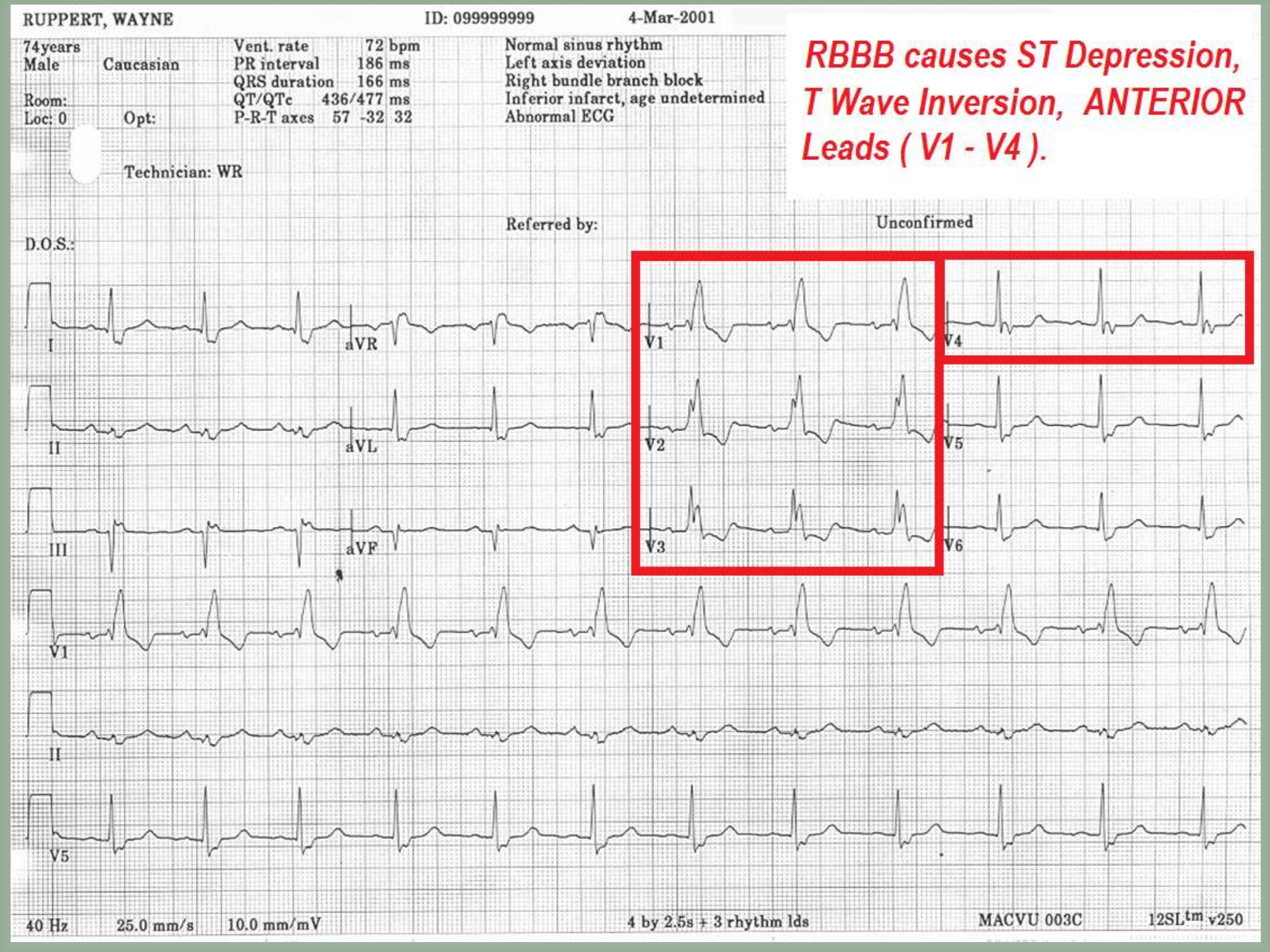
| | | |
|---|---|---|
| RIGHT BUNDLE BRANCH BLOCK |   | LEFT BUNDLE BRANCH BLOCK |
| W-P-W BYPASS TRACT, LEFT LATERAL WALL 49 y/o MALE |   | SAME PATIENT AS ON LEFT - IMMEDIATELY AFTER RF ABLATION OF BYPASS TRACT |
| W-P-W BYPASS TRACT, RIGHT ANTERIOR/ LATERAL WALL 14 y/o MALE |   | SAME PATIENT AS ON 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:



# Wide QRS present:
# (QRSd > 120ms)

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


RUPPERT, WAYNE
ID: 099999999
4-Mar-2001
74years
Male
Caucasian
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
Room:
Loc: 0
Opt:
Technician: WR
Referred by:
Unconfirmed
D.O.S.:
RBBB causes ST Depression, T Wave Inversion, ANTERIOR Leads ( V1 - V4 ).
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
12SL tm 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

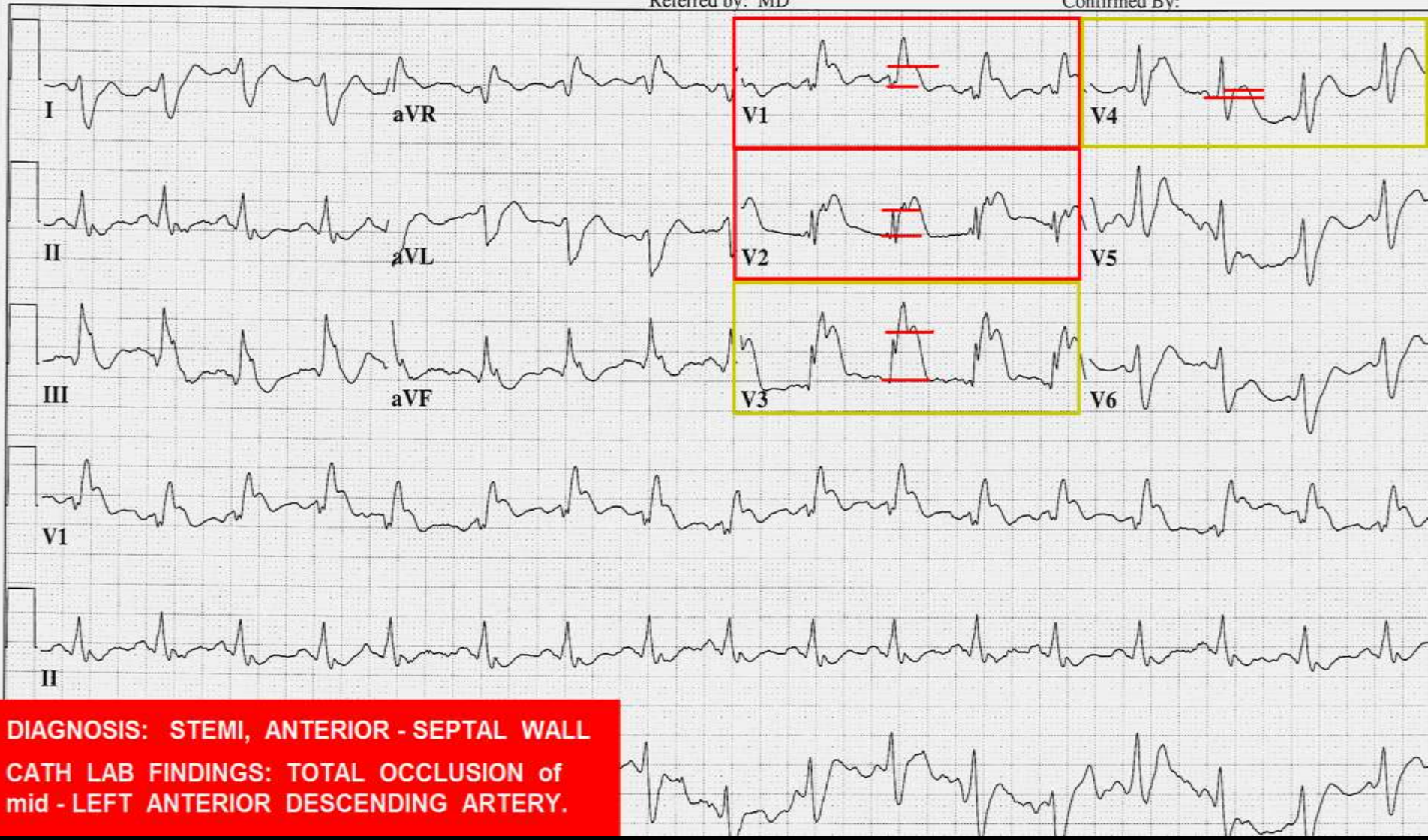
| | | |
|---|---|---|
| Vent. rate | 102 | BPM |
| PR interval | 130 | ms |
| QRS duration | 168 | ms |
| QT/QTc | 400/521 | ms |
| P–R–T axes | 60 114 | –19 |

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

Technician: W Ruppert

Referred by: MD

Confirmed By:




**DIAGNOSIS: STEMI, ANTERIOR - SEPTAL WALL**

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

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

25 yr
Male Caucasian

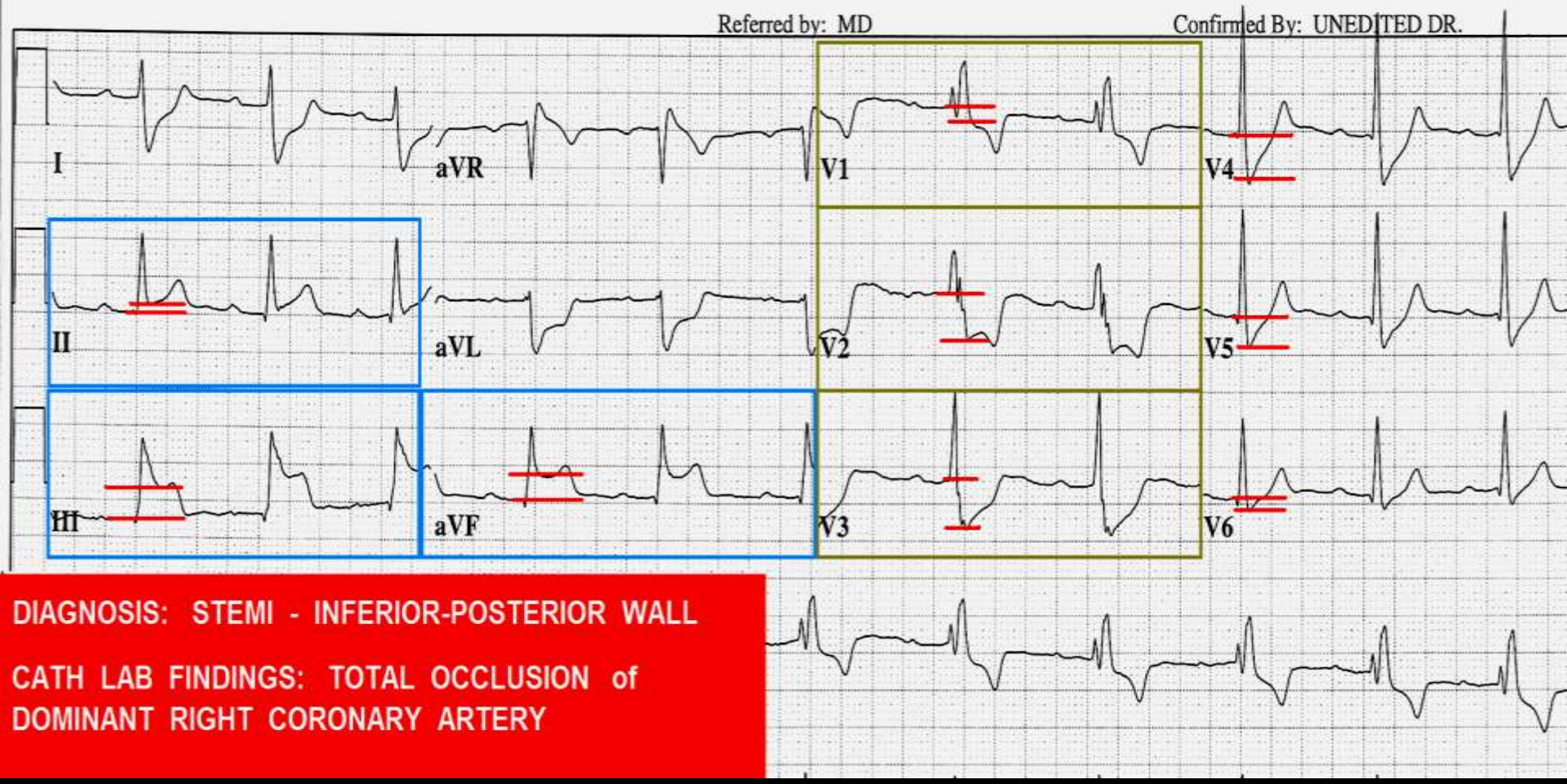
Loc:3 Option:23

| | | |
|---|---|---|
| Vent. rate | 67 | BPM |
| PR interval | 258 | ms |
| QRS duration | 136 | ms |
| QT/QTc | 398/420 | ms |
| P-R-T axes | 44 94 | 82 |

Sinus rhythm with 1st degree A-V block
Right bundle branch block
ST elevation consider inferior injury or acute infarct
** ** ** ** * ACUTE MI * ** ** ** **
Abnormal ECG

Referred by: MD

Confirmed By: UNEDITED DR.




**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

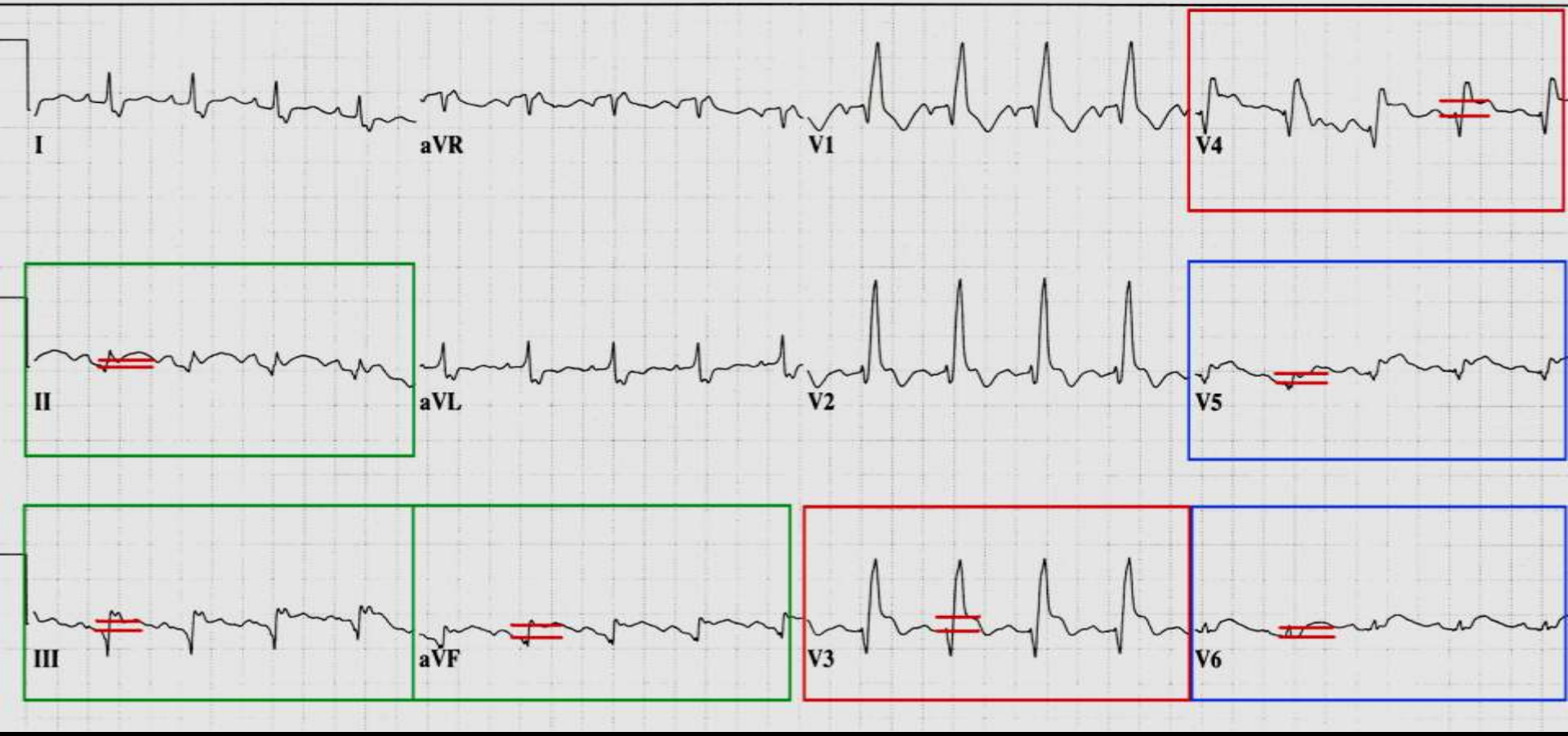
Room:CS–19
Loc:6 Option:41

| | | |
|---|---|---|
| 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***

# Diagnosis of STEMI with LBBB pattern:

## 2013 ACC/AHA Guideline for Management of STEMI

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

# Diagnosis of STEMI with LBBB pattern:

## 2013 ACC/AHA Guideline for Management of STEMI

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

# Diagnosis of STEMI with LBBB pattern:

## 2013 ACC/AHA Guideline for Management of STEMI

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

78 yr
Female Black

Room:ICU5
Loc:6 Option:19

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

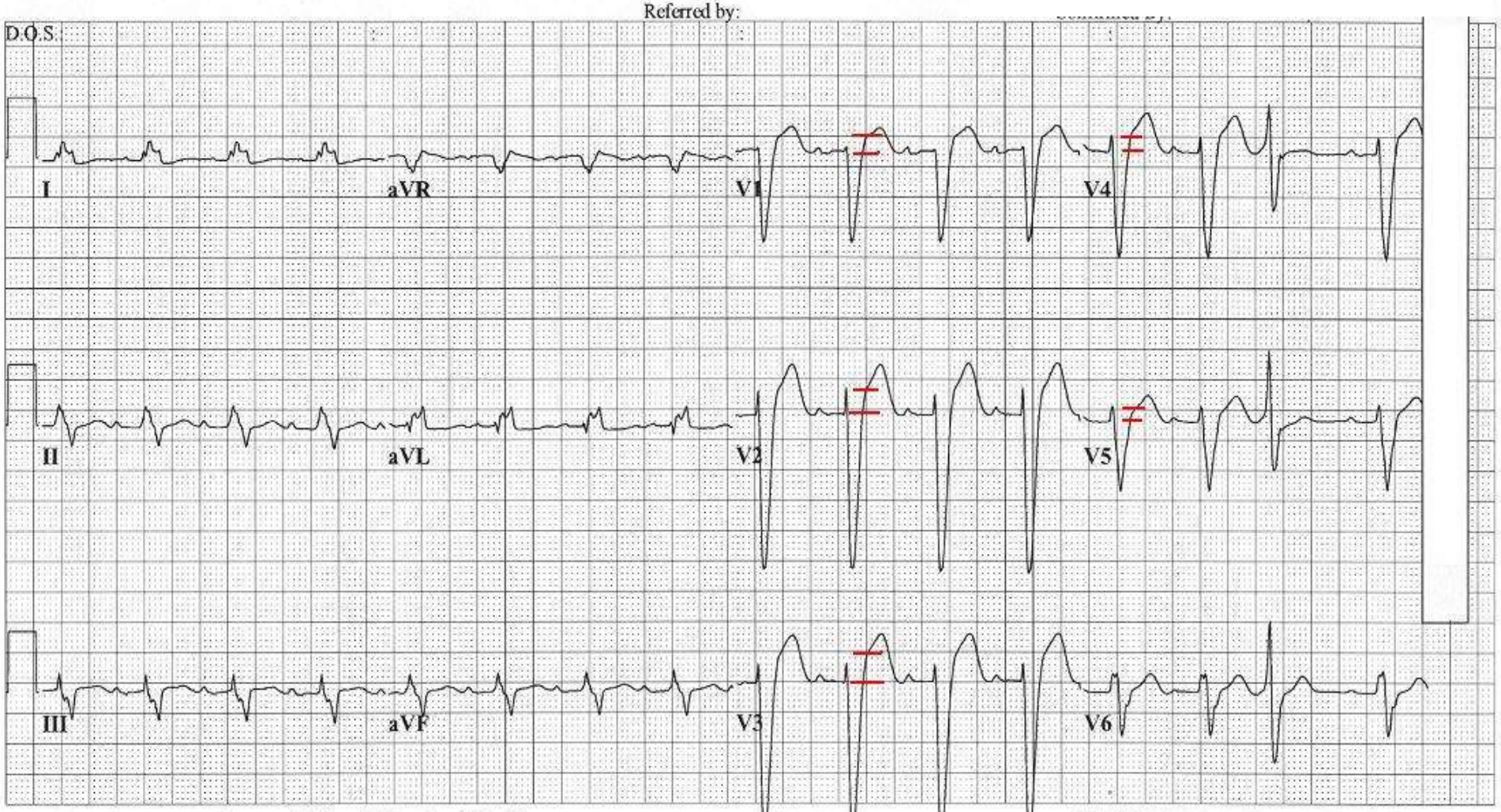
Normal sinus rhythm with occasional Premature ventricular complexes
Left bundle branch block
Abnormal ECG

- Normal arteries
- Normal LV Function
- No hypertrophy

Technician: EKG CLASS #WR03602718

Referred by:

D.O.S.




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**

***Be advised that in patients with***

**Left Bundle Branch Block**

Combined with

**Ventricular Hypertrophy,**

***The J Point elevation can exceed 0.5 mv (5mm) above the iso-electric line in patients without ACS.***

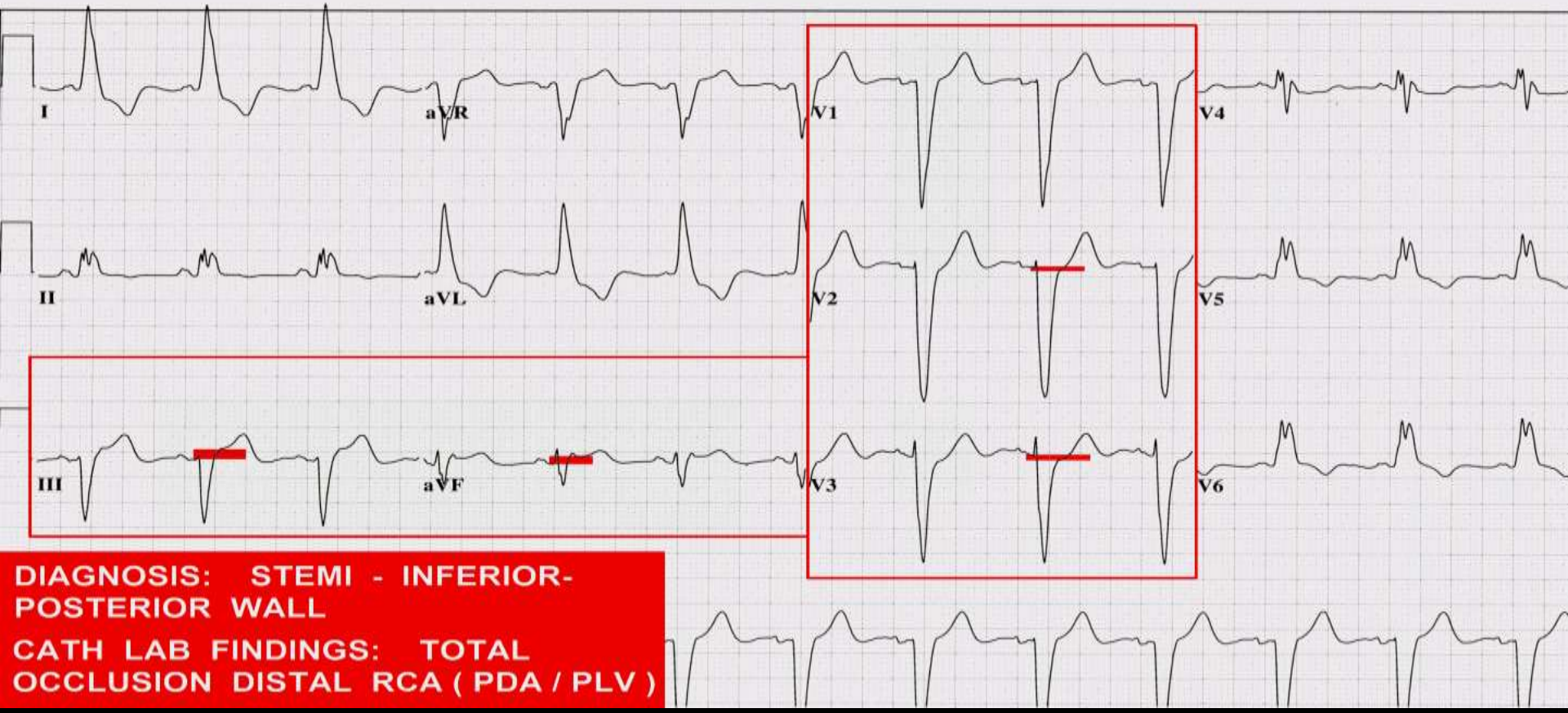
# LBBB with CHEST PAIN - CASE 1 : PRESENTING EKG

58 yr
Female Hispanic

Room: ER
Loc:3 Option:23

| | | |
|---|---|---|
| Vent. rate | 77 | BPM |
| PR interval | 128 | ms |
| QRS duration | 158 | ms |
| QT/QTc | 454/513 | ms |
| P-R-T axes | 43 -11 | 150 |

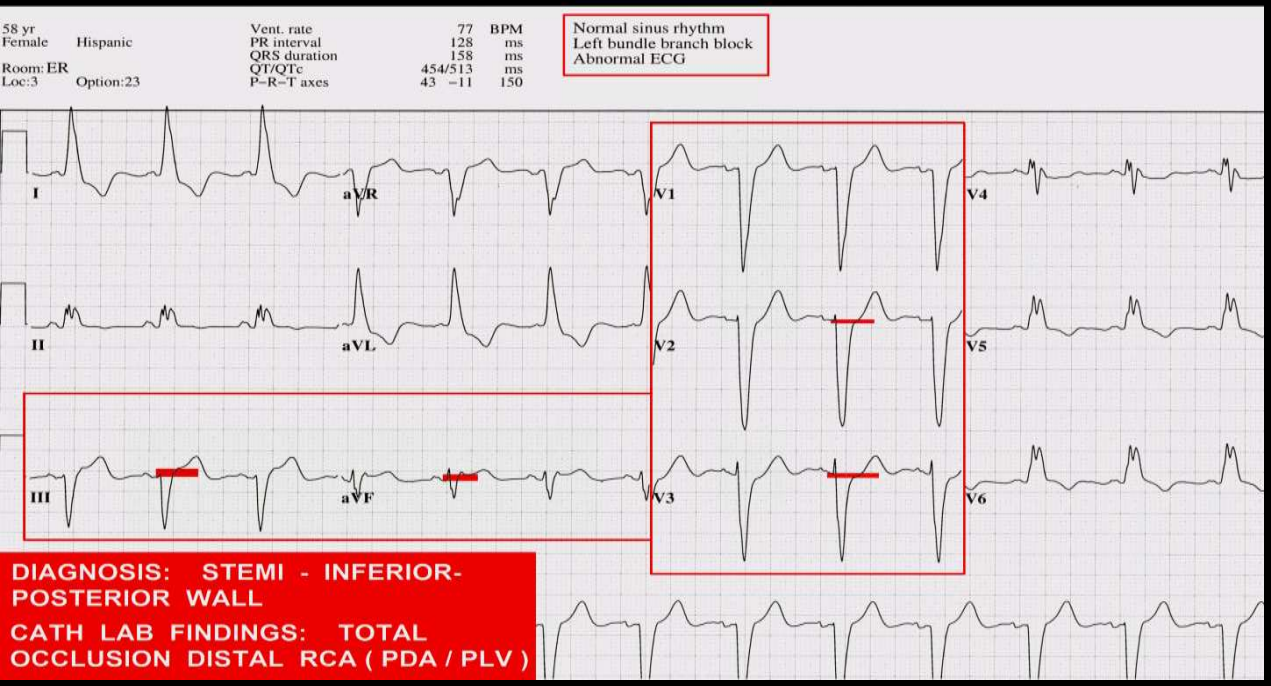
Normal sinus rhythm
Left bundle branch block
Abnormal ECG




**DIAGNOSIS: STEMI - INFERIOR-POSTERIOR WALL**

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

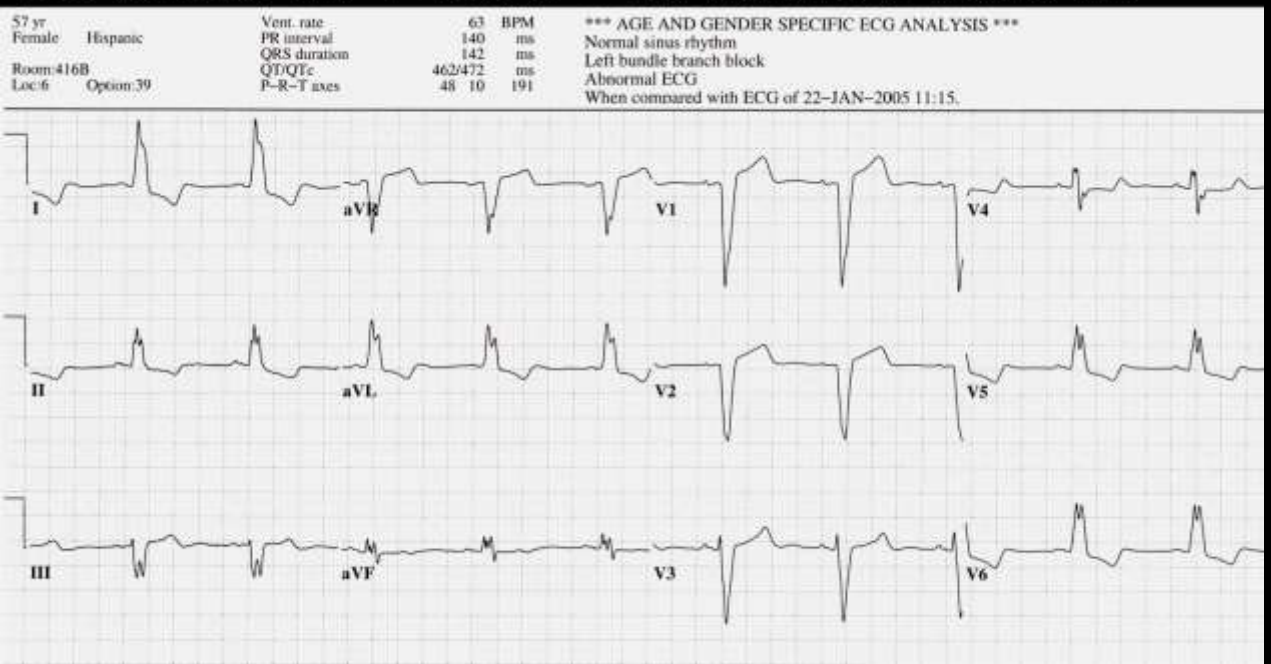
## LBBB with CHEST PAIN - CASE 1: PRESENTING EKG

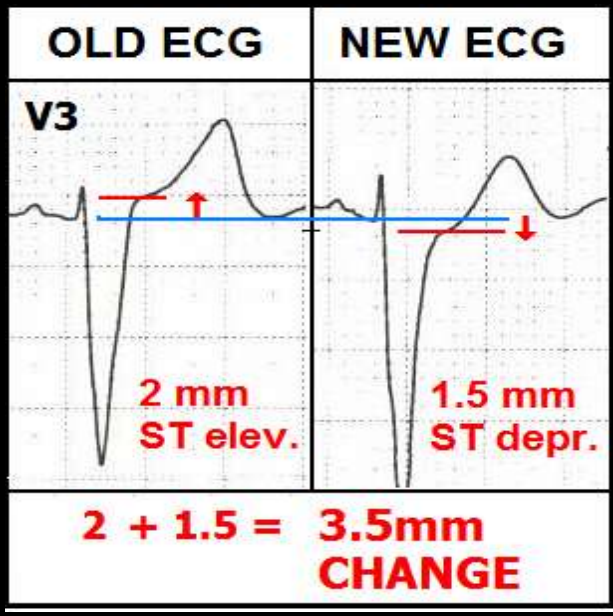



DIAGNOSIS: STEMI - INFERIOR-POSTERIOR WALL

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

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



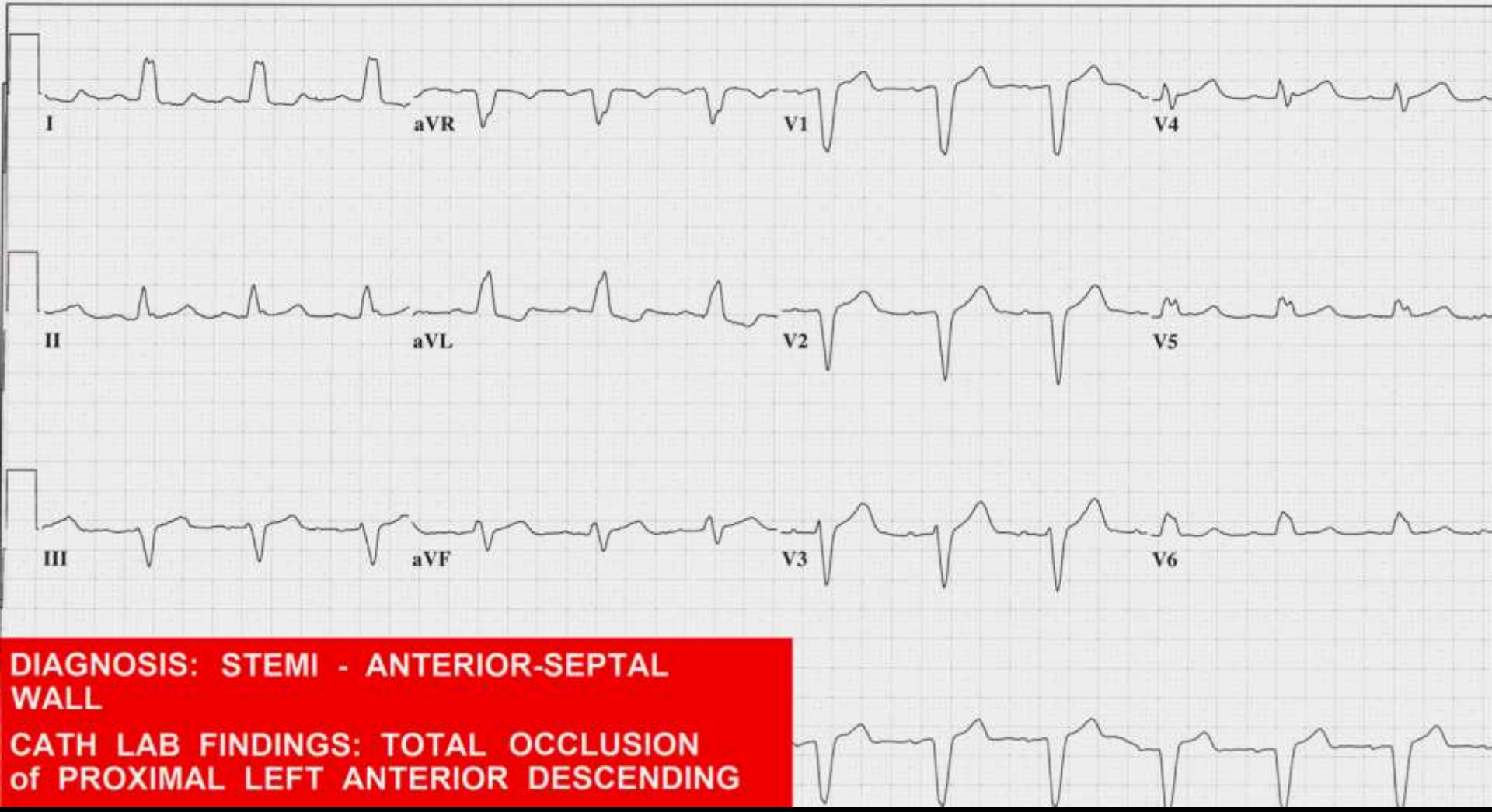
# LBBB with CHEST PAIN - CASE 2: *NEW ONSET of LBBB*

46 yr
Male Caucasian

Room:ER
Loc:3 Option:23

| | | |
|---|---|---|
| Vent. rate | 77 | BPM |
| PR interval | 172 | ms |
| QRS duration | 142 | ms |
| QT/QTc | 446/504 | ms |
| P-R-T axes | 38 0 | 92 |

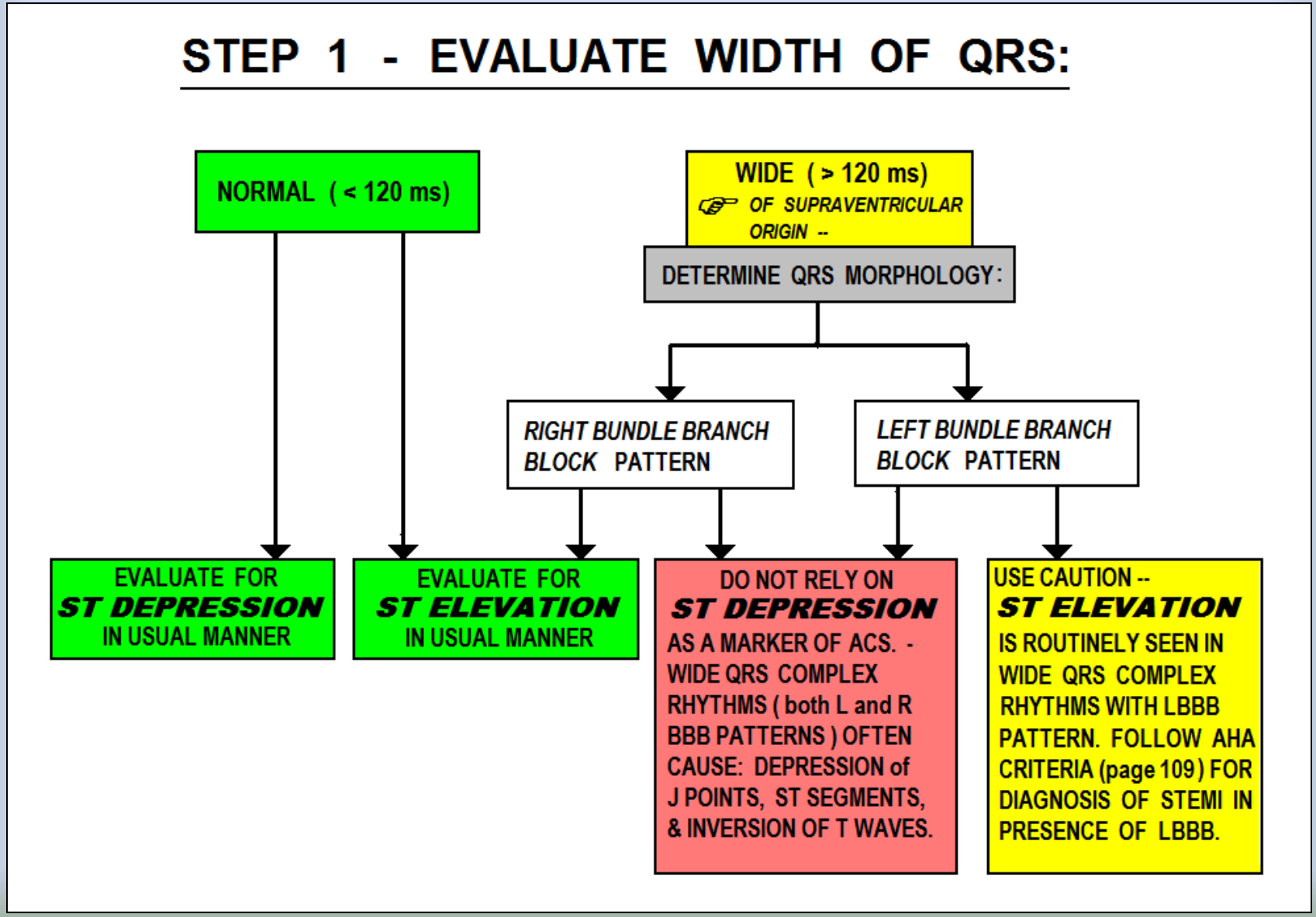
Normal sinus rhythm
Left bundle branch block
Abnormal ECG




**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)*

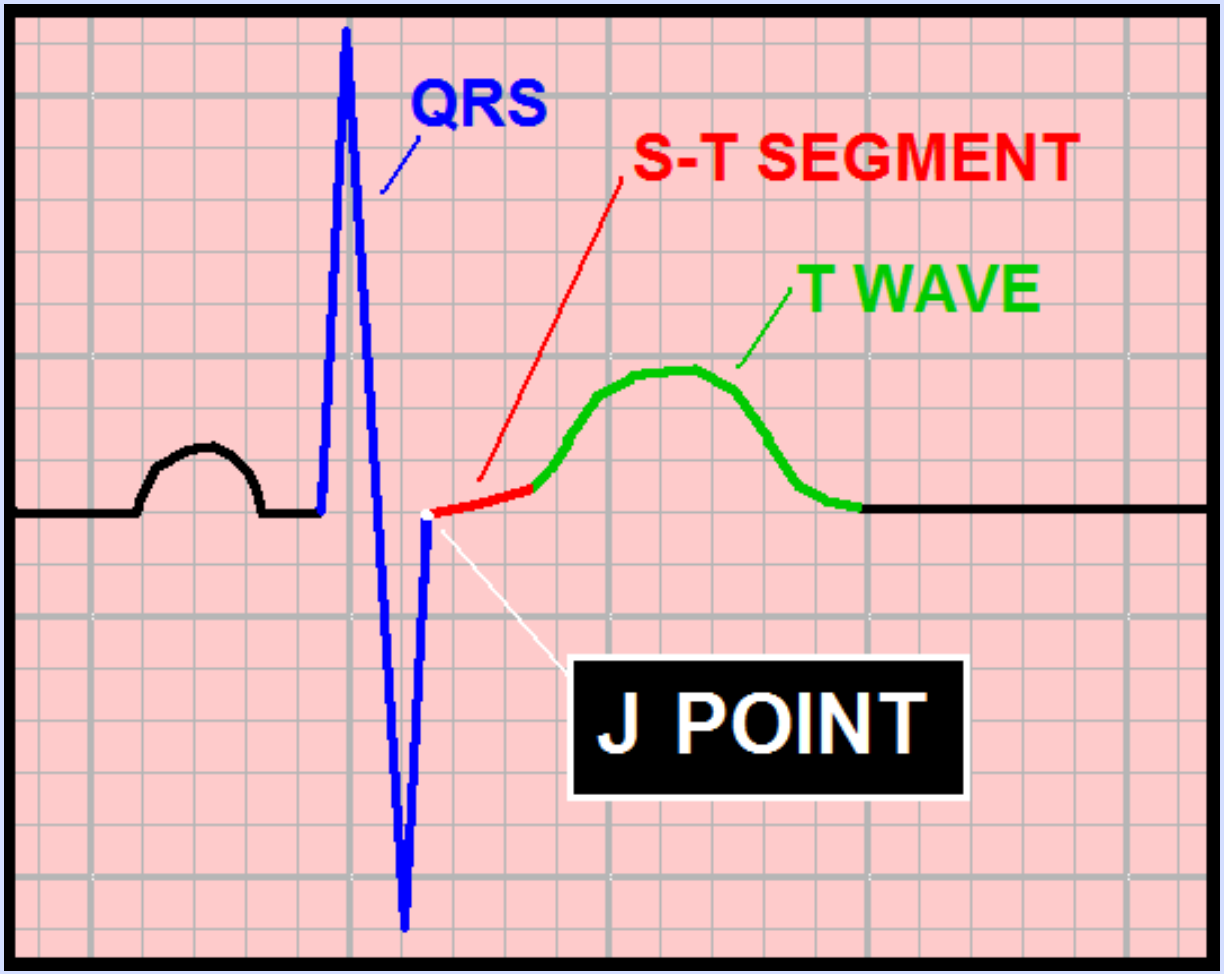
### STEP 2 - EVALUATE the EKG for ACS

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*.

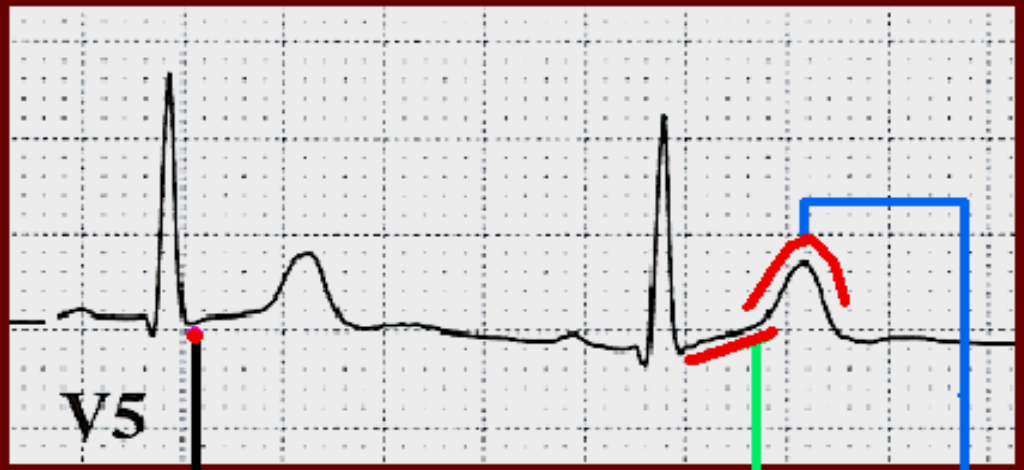
# Defining NORMAL – QRS <120ms:



## When QRS duration is NORMAL ( < 120 ms ):

# NORMAL ST - T WAVES

- WHEN QRS WIDTH IS NORMAL ( < 120 ms )

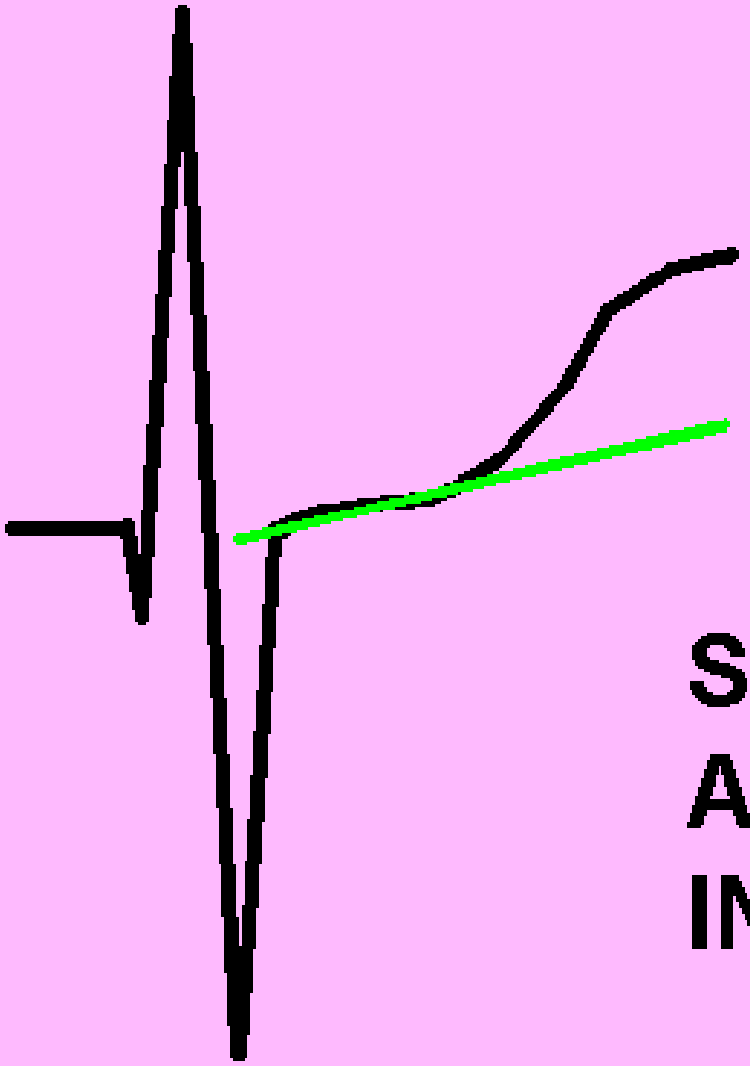



## *ASSESS:*

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

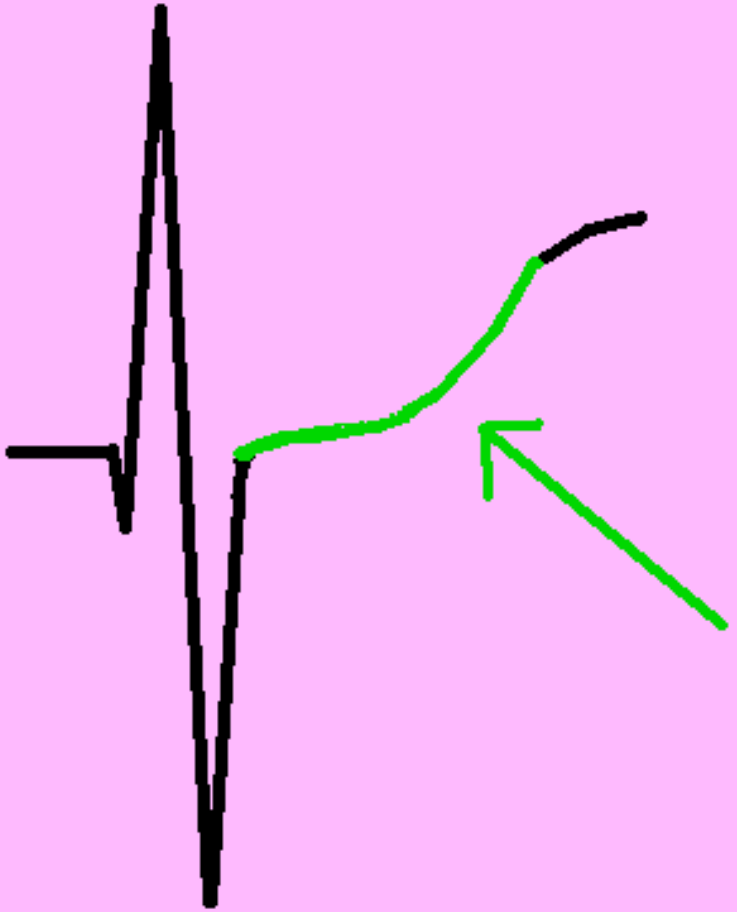
☞ *in EVERY LEAD EXCEPT aVR ! !*

# THE S-T SEGMENT



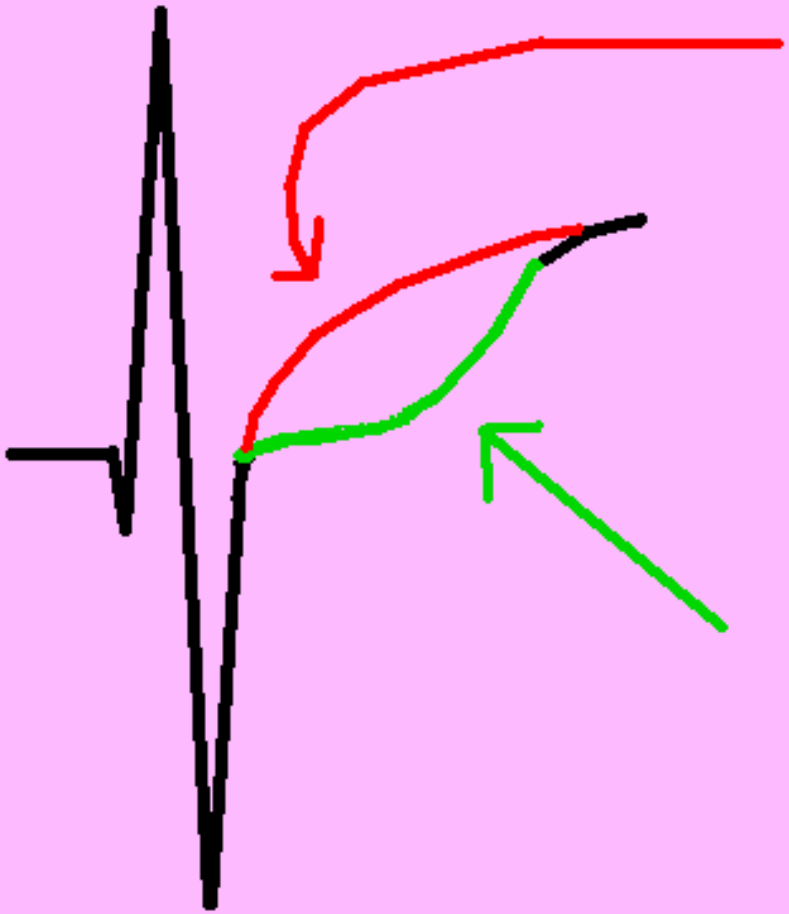
SHOULD HAVE
A "SLIGHT POSITIVE"
INCLINATION

# THE S-T SEGMENT



SHOULD BE "CONCAVE" IN SHAPE . . .

# THE S-T SEGMENT



AS OPPOSED TO "CONVEX" IN SHAPE

SHOULD BE "CONCAVE" IN SHAPE . . .

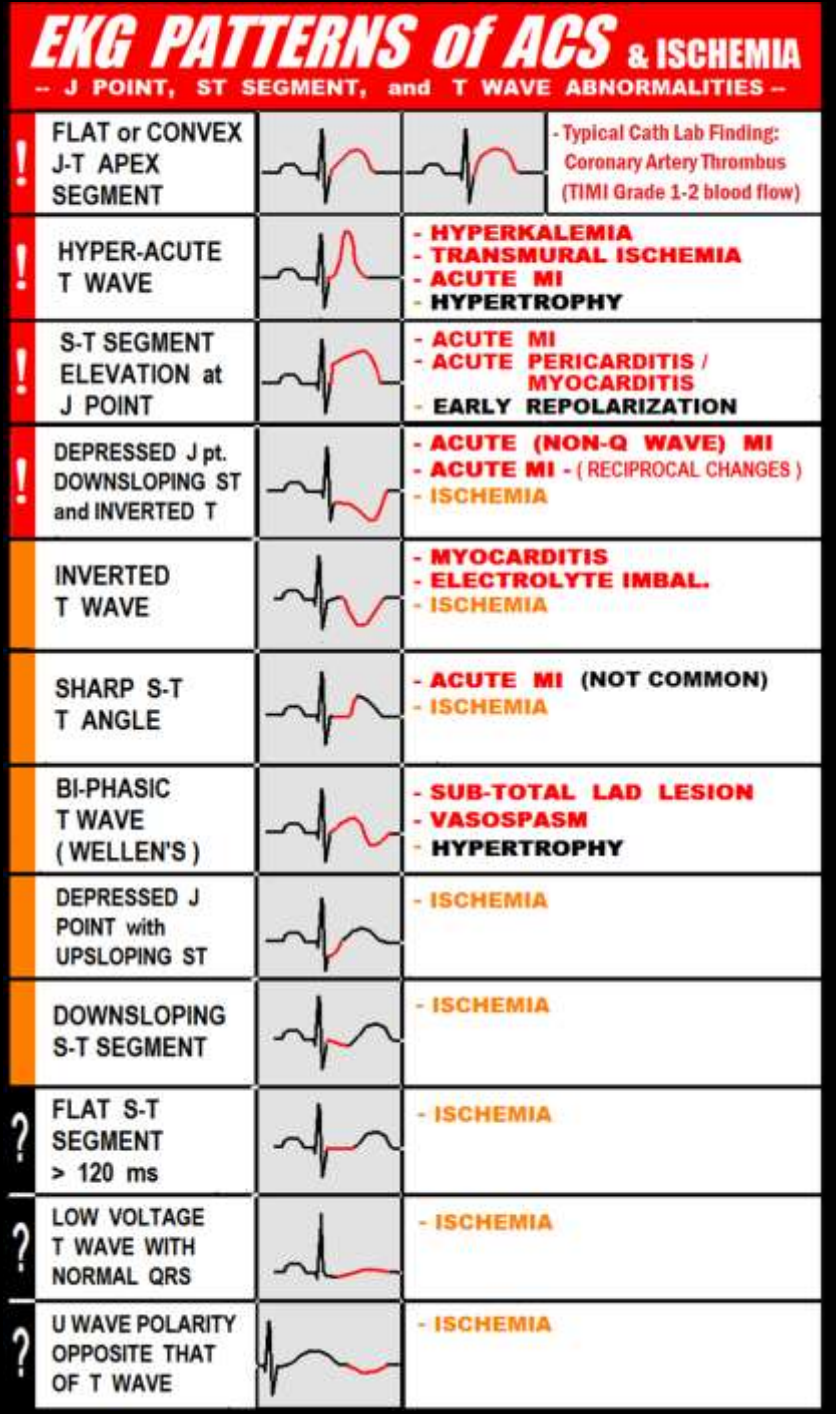
Multiple patterns of **ABNORMAL:**

- **J Point**
- **ST-Segment**
- **T Wave**

configurations **may indicate ACS.**

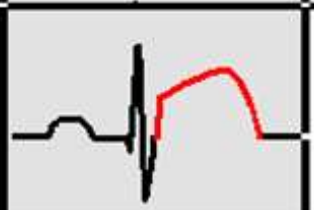
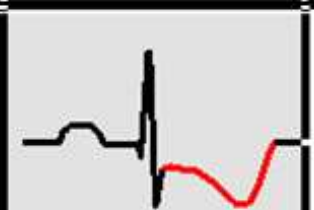
Remember, "IF IT'S NOT **NORMAL**, it's ***ABNORMAL !***"

**BOOK PAGE: 83**



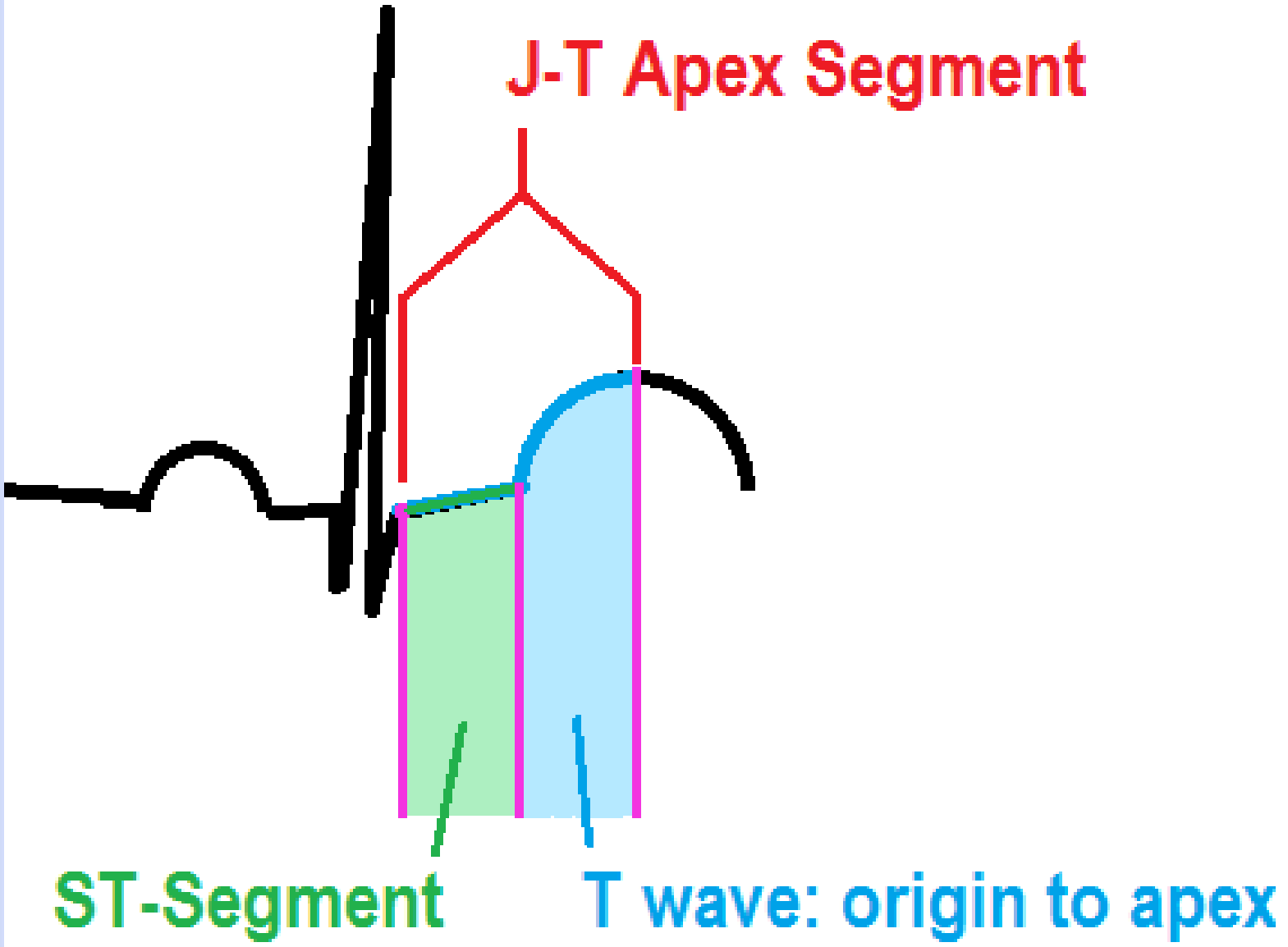
# EKG PATTERNS of ACS & ISCHEMIA

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

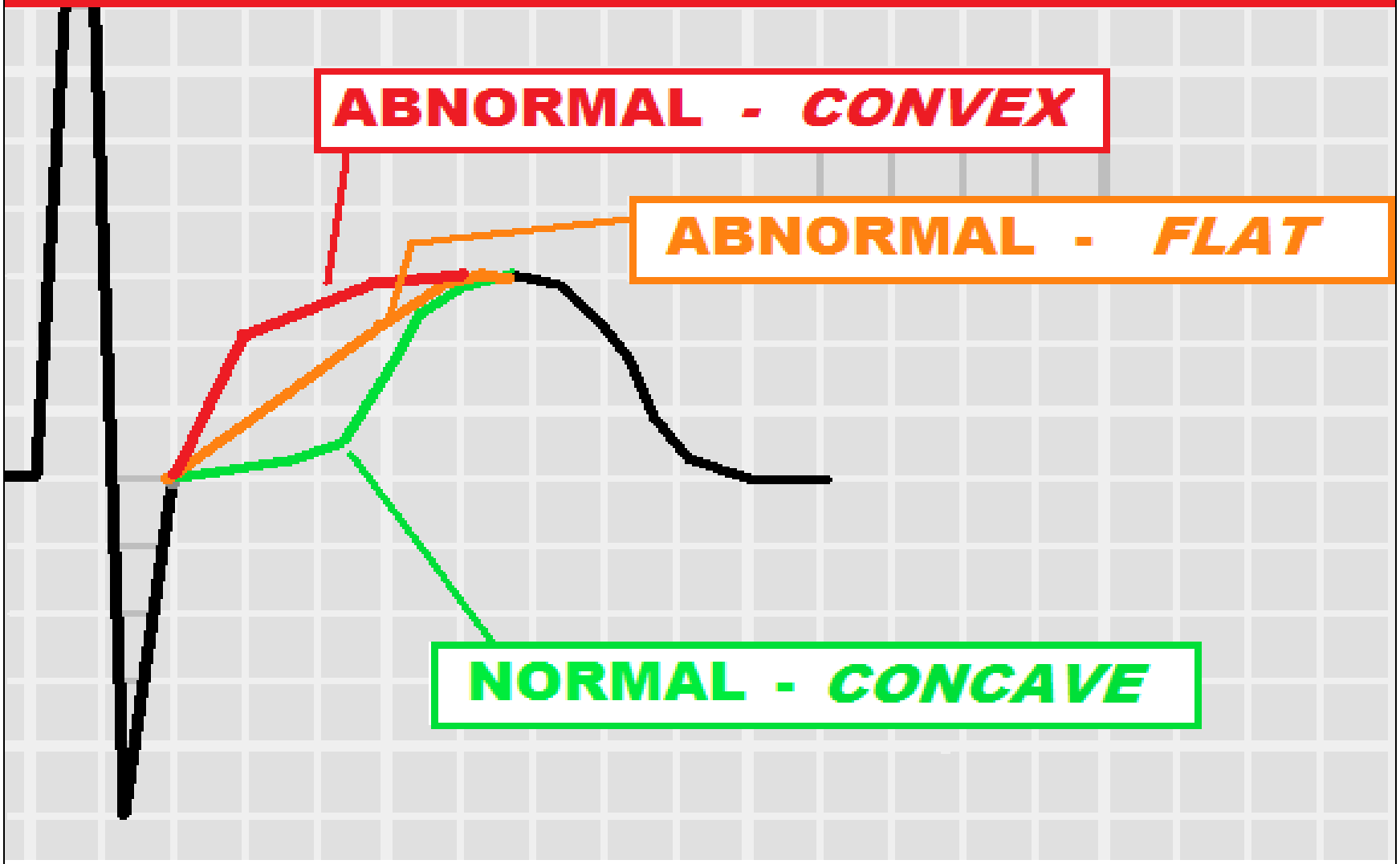
| | | | |
|---|---|---|---|
| ! | FLAT or CONVEX J-T APEX SEGMENT |  | - Typical Cath Lab Finding: Coronary Artery Thrombus (TIMI Grade 1-2 blood flow) |
| ! | HYPER-ACUTE T WAVE |  | - HYPERKALEMIA<br>- TRANSMURAL ISCHEMIA<br>- ACUTE MI<br>- HYPERTROPHY |
| ! | S-T SEGMENT ELEVATION at J POINT |  |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

# *ECG Patterns associated with "EARLY PHASE MI:"*

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


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

# J - T APEX SEGMENT VARIATIONS

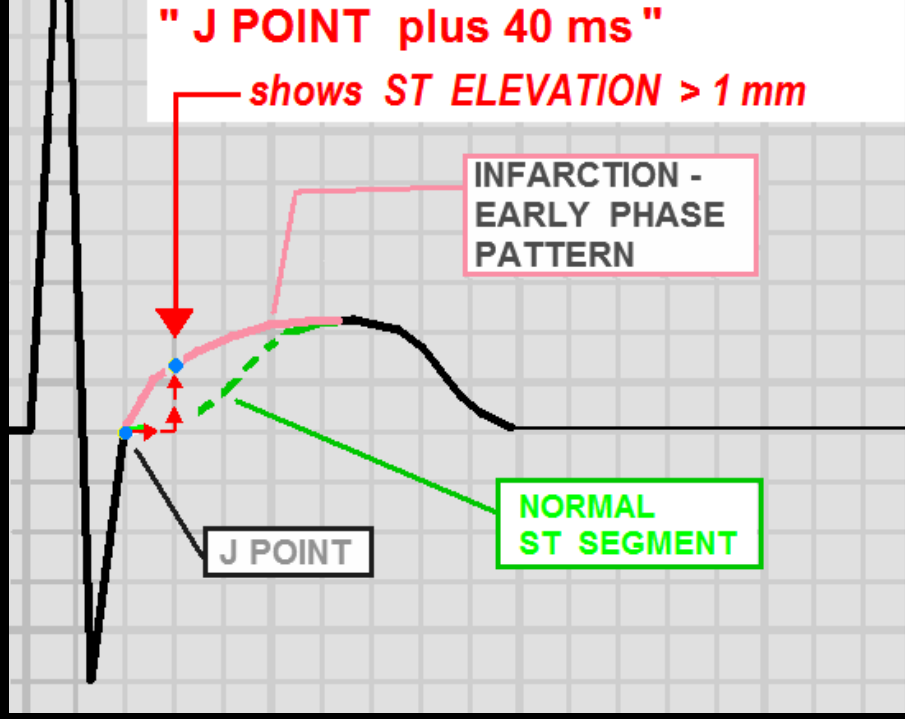



## PATTERNS of EARLY INFARCTION

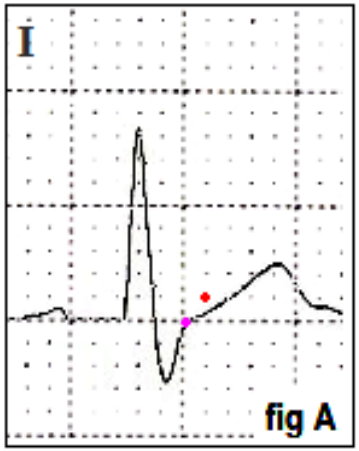
-- FLAT and CONVEX J-T APEX SEGMENTS

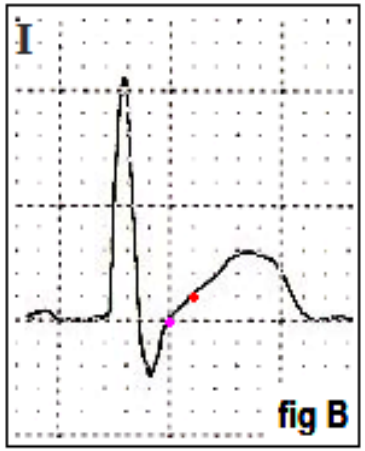
# WHEN EVALUATING for ST SEGMENT ELEVATION . . . . . .

From:
AMERICAN HEART ASSOCIATION
ACLS 2005 REVISIONS




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

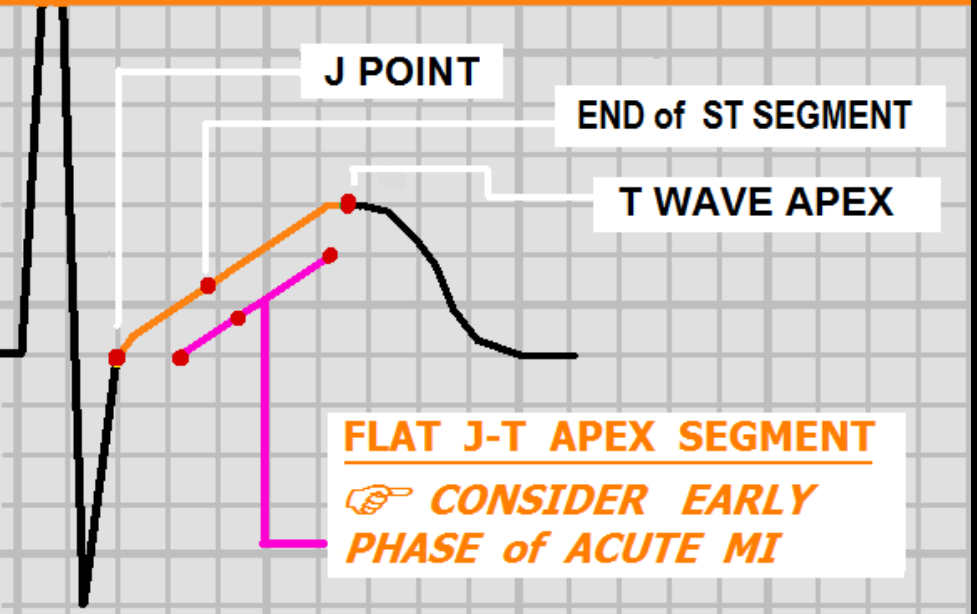


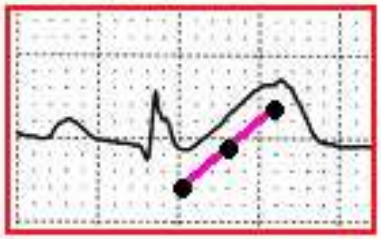

**During a 20 second BALLOON OCCLUSION of the patient's LAD during routine PTCA, the ST segment assumes a CONVEX shape. When measured 40 ms beyond the J POINT, the ST segment is elevated > 1 mm. This phenonemon is seen routinely in the cath lab prior to the occurance of ST ELEVATION at the J POINT during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

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

# ABNORMAL J-T APEX SEGMENT




LEAD II

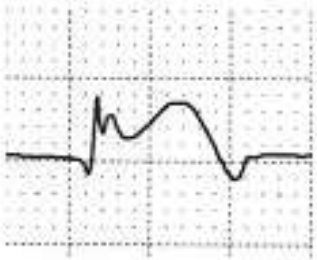


1839 hrs

41 y/o FEMALE

In ER C/O CHEST PAIN x 30 minutes.

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



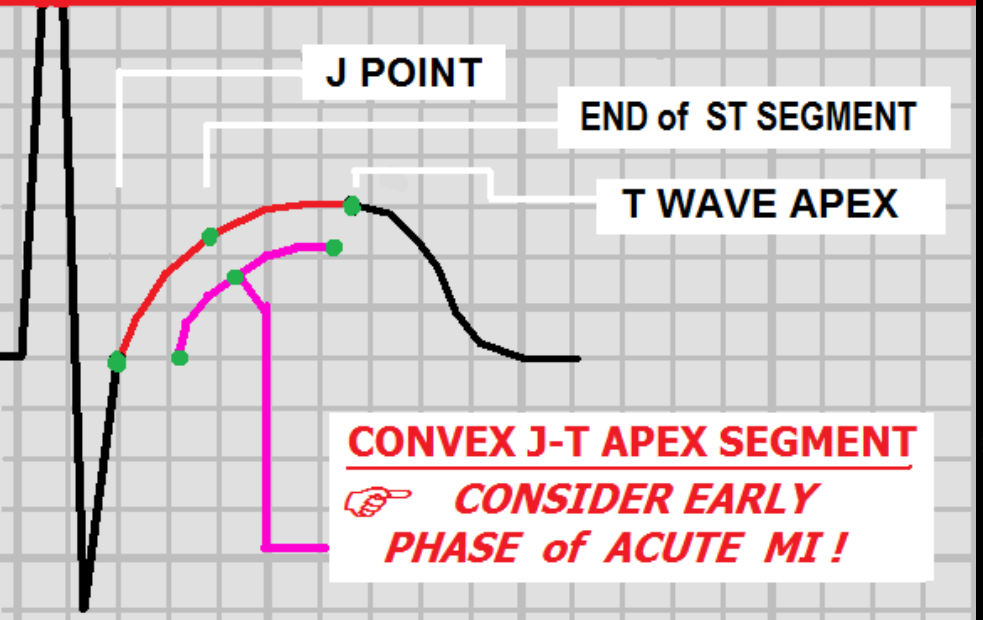
1850 hrs

STEMI - INFERIOR WALL

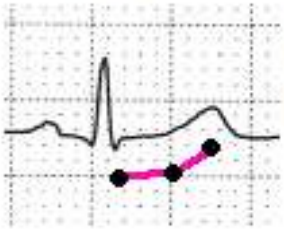
11 MINUTES LATER, S-T ELEVATION at the J POINT IS NOTED.

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

# ABNORMAL J-T APEX SEGMENT

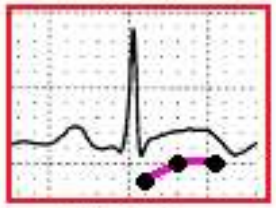



LEAD I



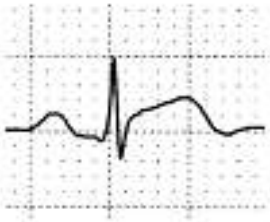
53 y/o MALE

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



0732 hrs

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



0747 hrs

15 MINUTES LATER, S-T ELEVATION at the J POINT IS NOTED.
- CATH LAB FINDINGS: TOTAL OCCLUSION OF CIRCUMFLEX ARTERY

# CASE STUDY: ABNORMAL J-T APEX SEGMENTS

## CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

## RISK FACTOR PROFILE:

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

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

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

**LABS:** JUST OBTAINED, RESULTS NOT AVAILABLE YET.

56 yr
Male Caucasian

Room:A9
Loc:3 Option:23

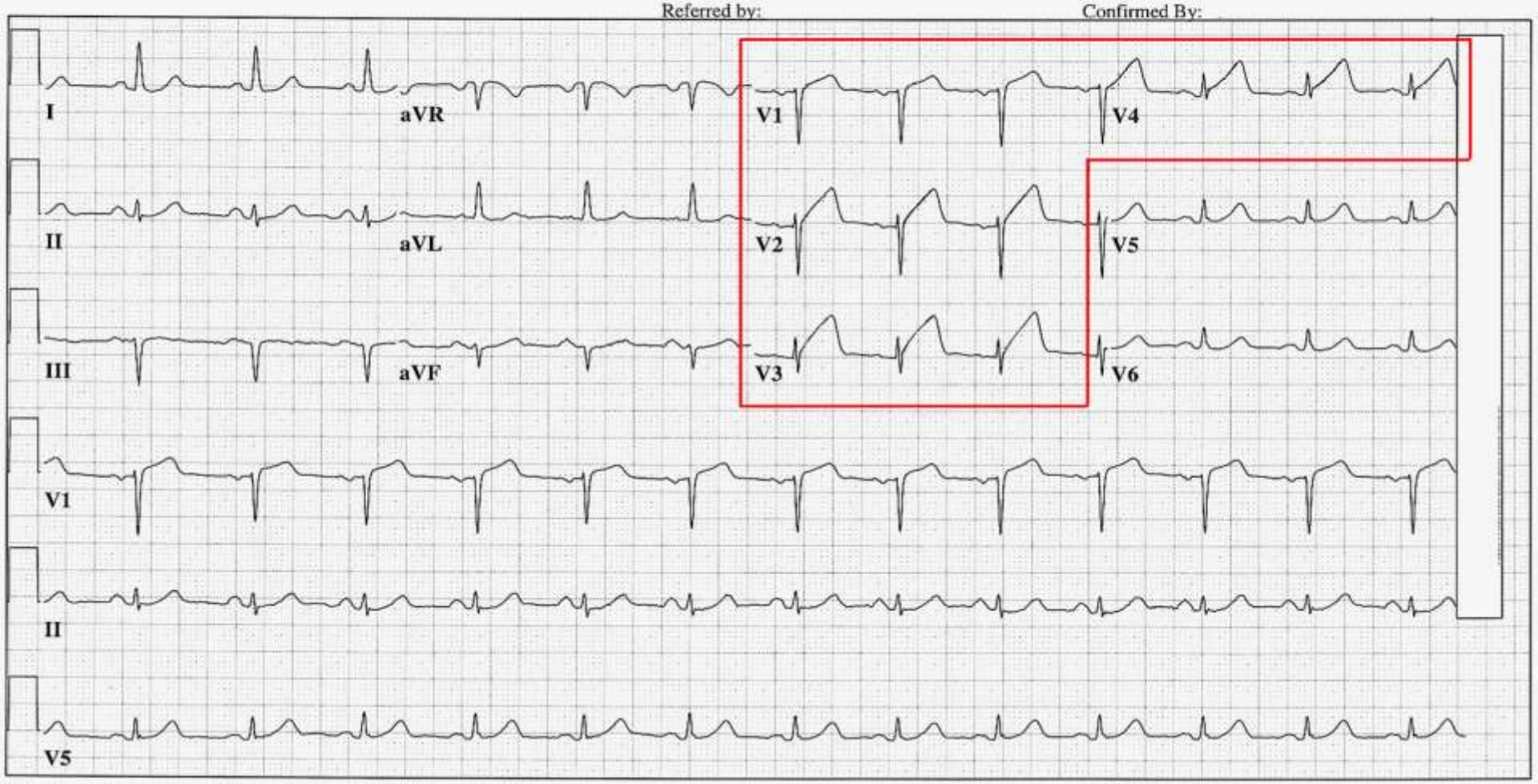
| | | |
|---|---|---|
| Vent. rate | 80 | BPM |
| PR interval | 154 | ms |
| QRS duration | 78 | ms |
| QT/QTc | 380/438 | ms |
| P–R–T axes | 51 –24 | 38 |

**UNEDITED COPY – REPORT IS COMPUTER GENERATED ONLY, WITHOUT PHYSICIAN INTERPRETATION

Normal sinus rhythm
Normal ECG
No previous ECGs available

Technician: W Ruppert

Referred by: Confirmed By:




25mm/s 10mm/mV 40Hz 005C 12SL 235 CID: 3 EID:10 EDT:

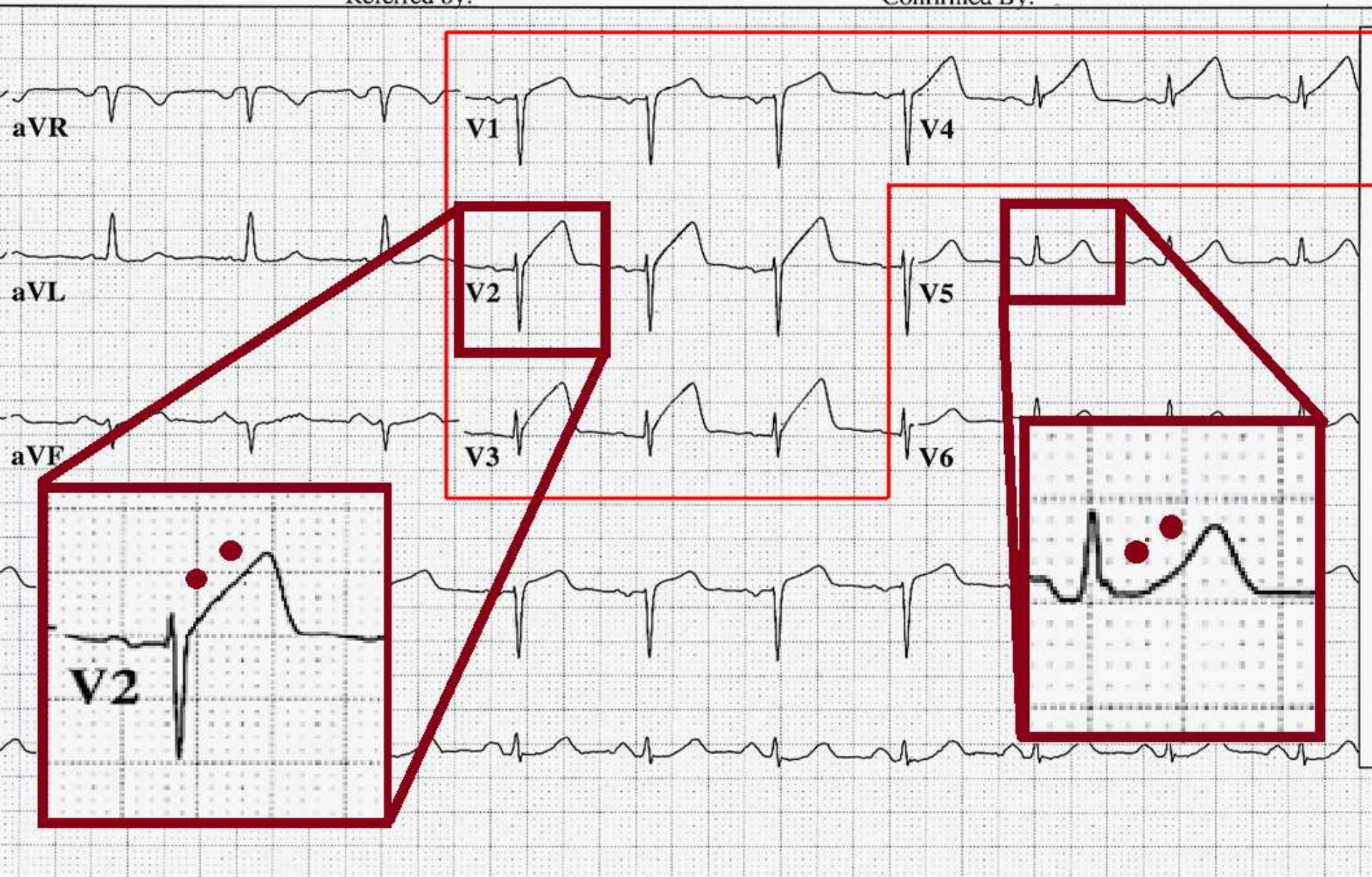
*ECG COMPUTER DOES NOT NOTICE THE CONVEX J-T APEX SEGMENTS !*

380/438 ms
51 –24 38

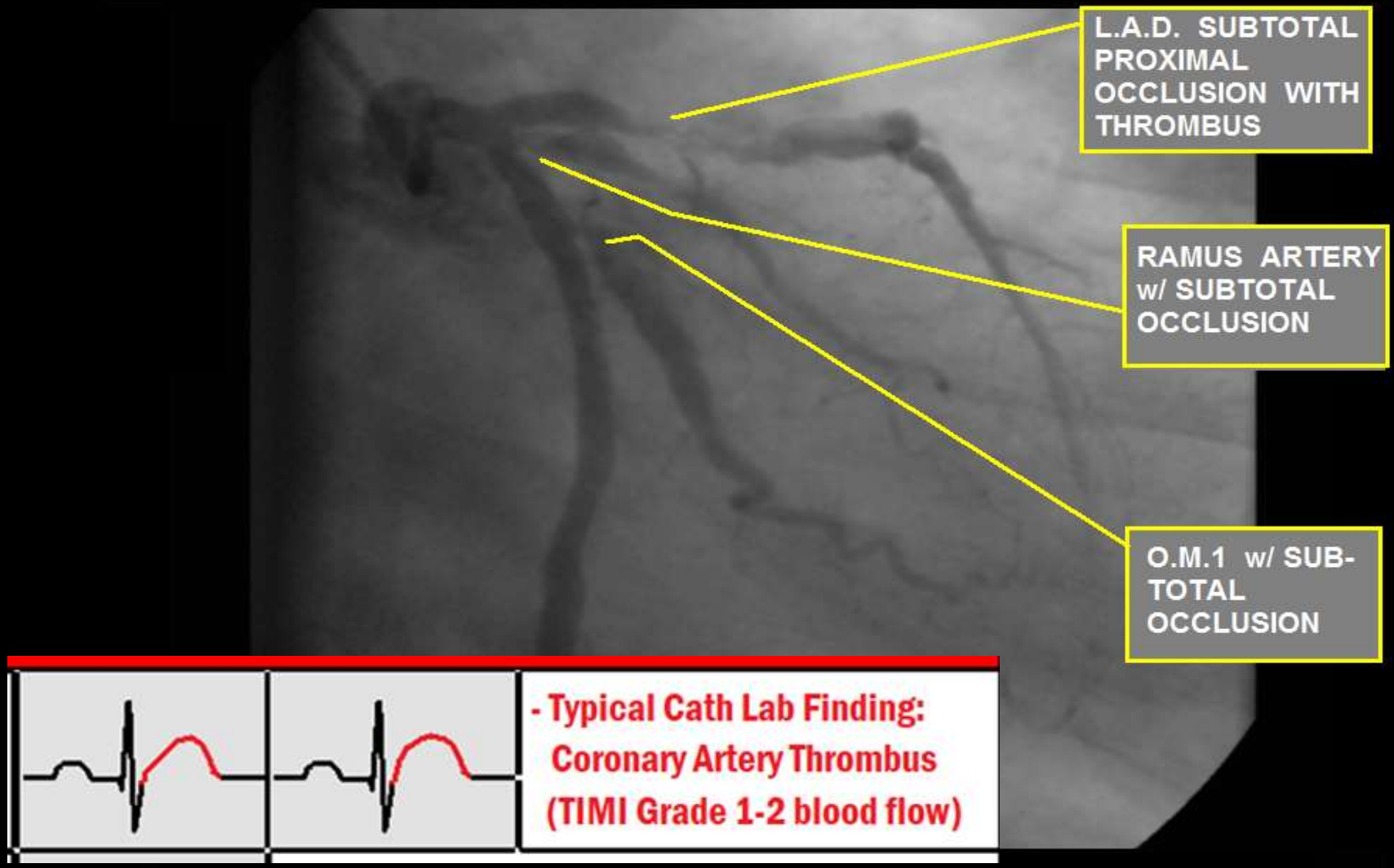
Normal sinus rhythm
Normal ECG
No previous ECGs available

Referred by:

Confirmed By:



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



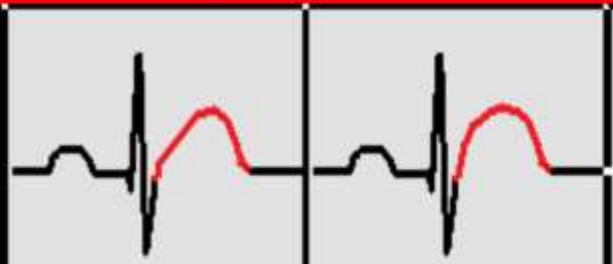
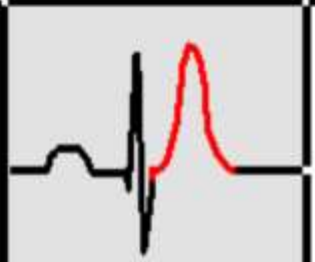
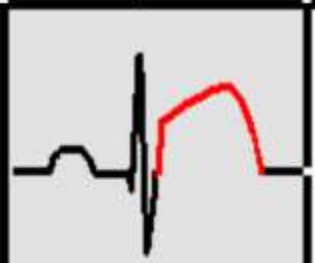
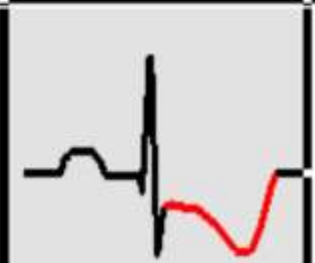



- Typical Cath Lab Finding:
  Coronary Artery Thrombus
  (TIMI Grade 1-2 blood flow)

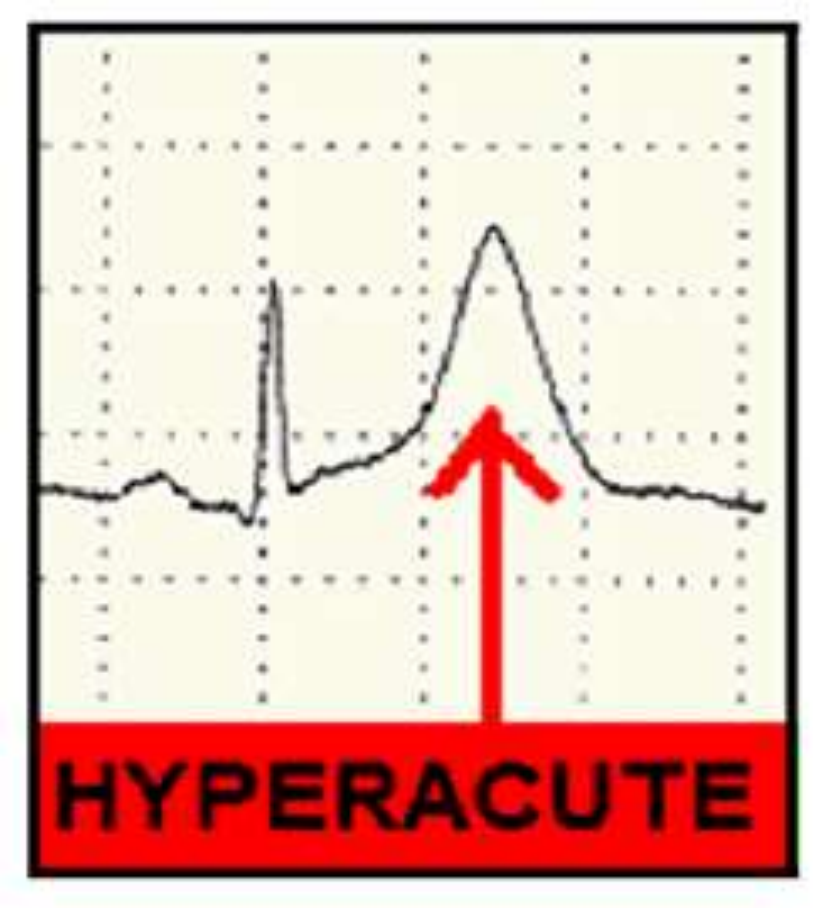
TREATMENT PLAN : EMERGENCY CORONARY ARTERY BYPASS SURGERY ( 4 VESSEL )

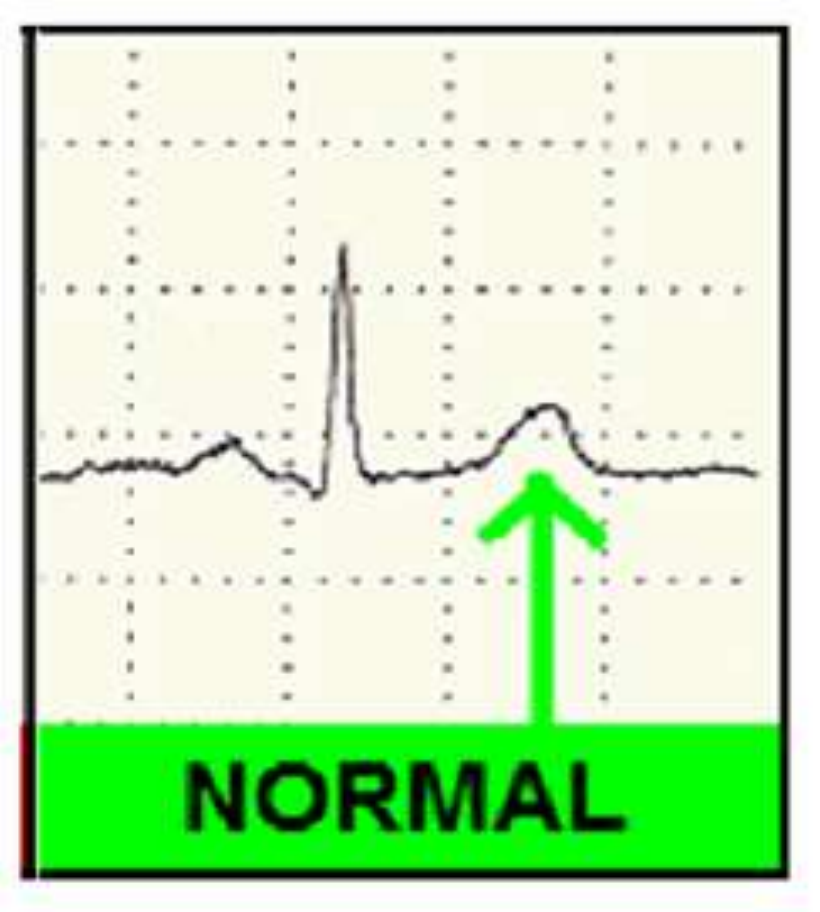
# PATTERNS of ACS & ISCHEMIA

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

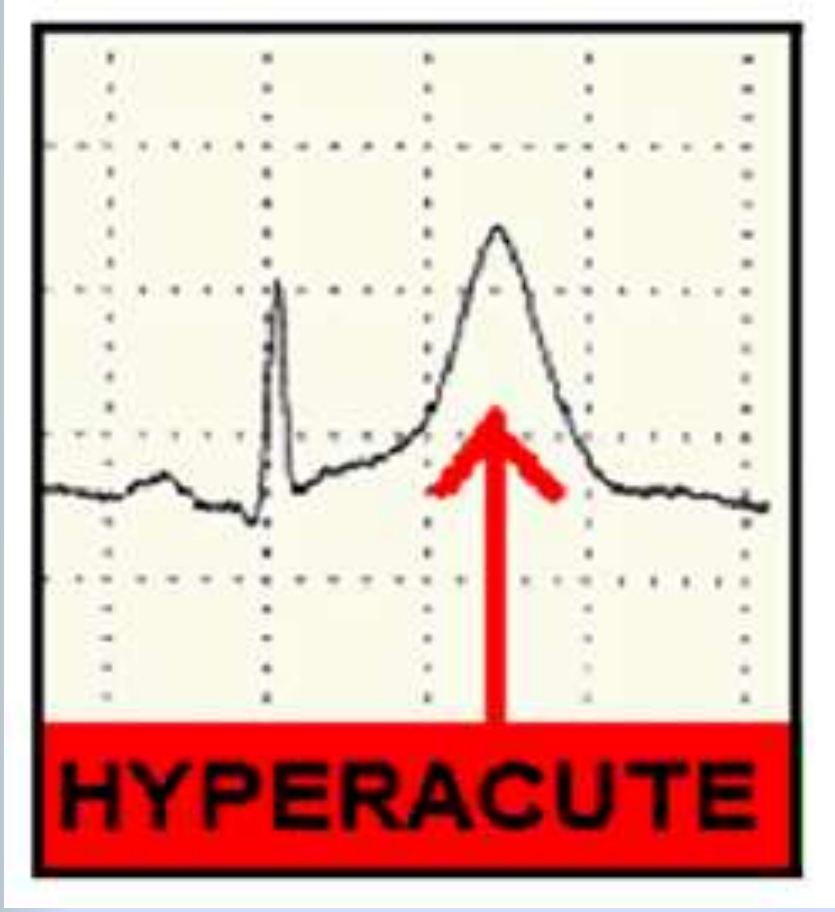
| | | | |
|---|---|---|---|
| ! | FLAT or CONVEX J-T APEX SEGMENT |  | **ACUTE MI** EARLY PHASE |
| ! | HYPER-ACUTE T WAVE |  | **ACUTE MI** EARLY PHASE |
| ! | S-T SEGMENT ELEVATION at J POINT |  | **ACUTE MI** |
| ! | DEPRESSED J pt. DOWNSLOPING ST and INVERTED T |  | - **ACUTE (NON-Q WAVE) MI**<br>-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

# T waves should not be HYPERACUTE



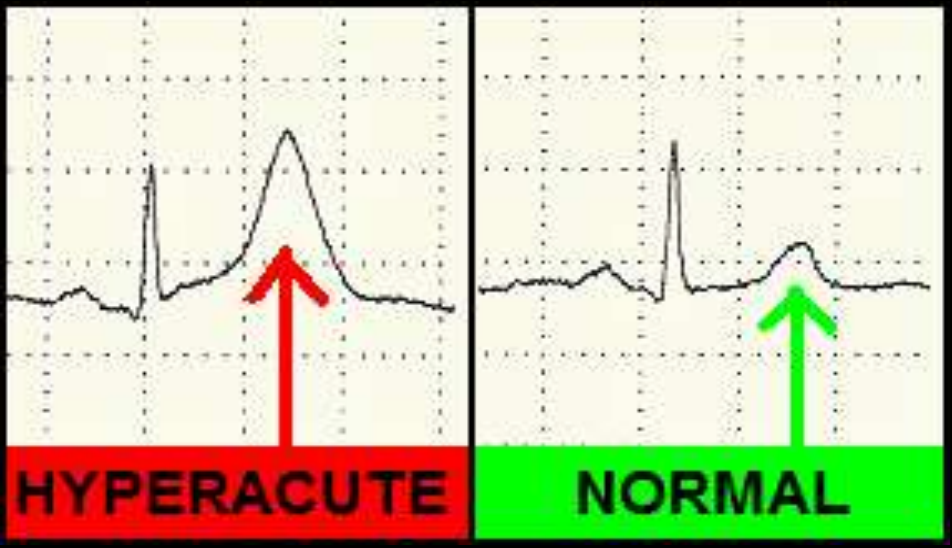



# HYPERACUTE T Waves may indicate:



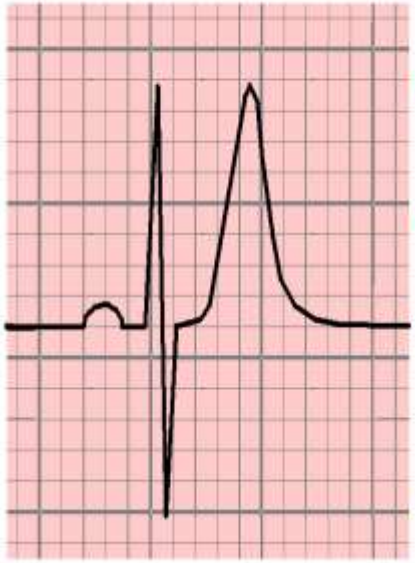

- **Early phase Acute MI**
- **Transmural ischemia** (usually seen in one region of the ECG)
- **Hyperkalemia** (seen globally across ECG)
- **Hypertrophy**

# HYPERACUTE T WAVES

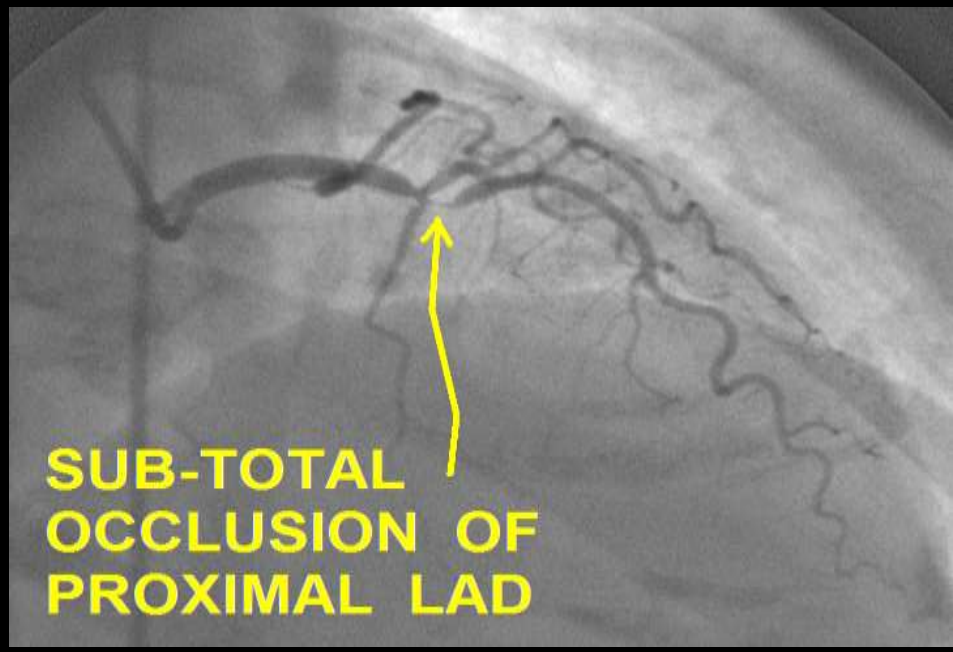



BOOK PAGE: 88

## HYPER-ACUTE T WAVES - COMMON ETIOLOGIES:



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



# Helpful Clue: Hyper-Acute T Waves

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

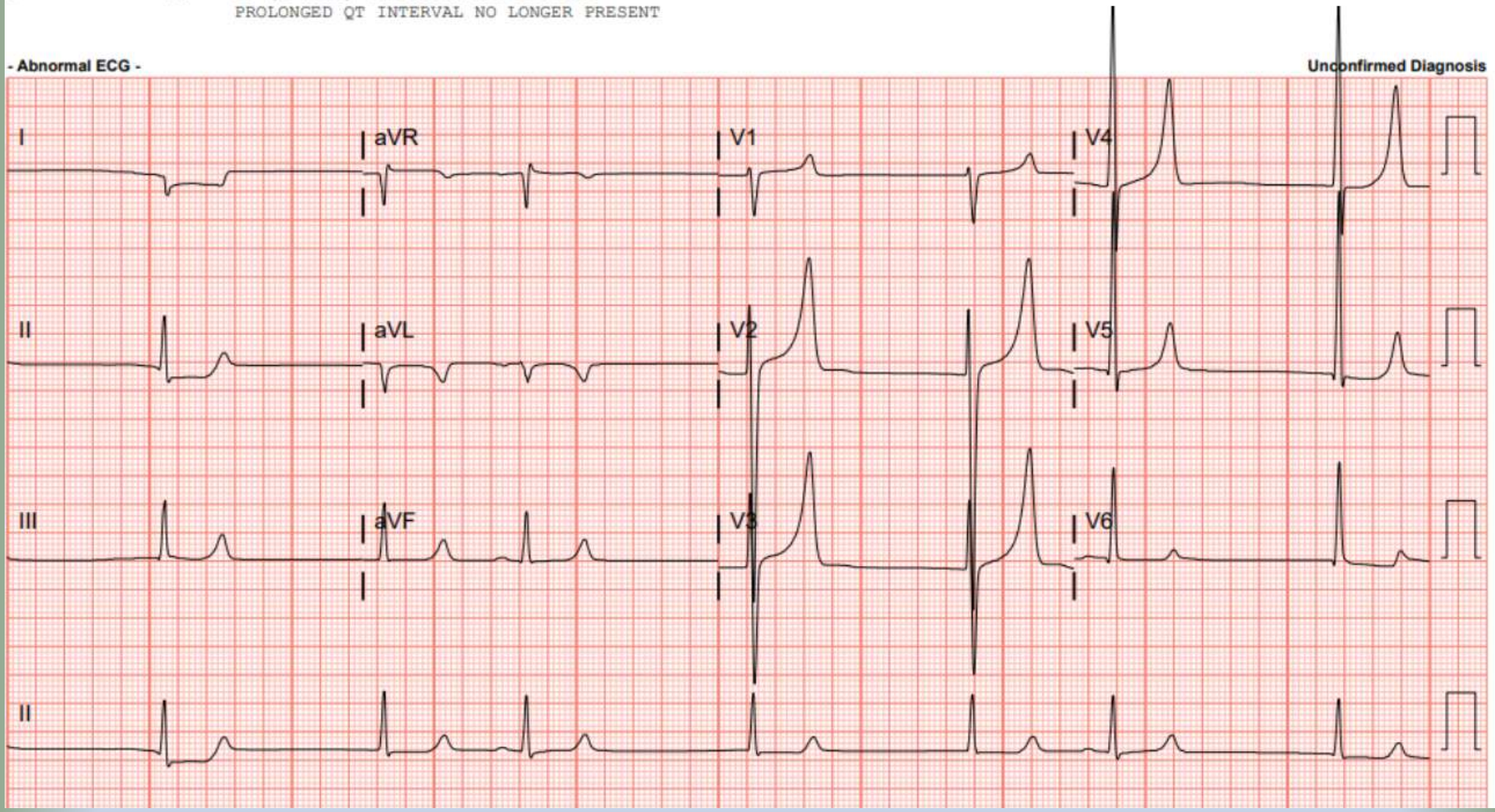
| | | |
|---|---|---|
| Rate | 39 | Right and left arm electrode reversal, interpretation assumes no reversal |
| PR | 500 | Sinus bradycardia |
| QRSd | 117 | Atrial premature complexes |
| QT | 549 | LVH with IVCD and secondary repol abnrm |
| QTc | 443 | Anterior ST elevation, probably due to LVH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

Req Provider: ONIER VILLARREA

**K+ = 7.9**

- Abnormal ECG -

Unconfirmed Diagnosis



ID:

23-Nov-

REGIONAL MEDICAL CENTER

55years
Female Caucasian

Room:

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

Sinus bradyc a
Possible Left atrial enlargement
Borderline ECG

ER ATTENDING REVIEW
NO STEMI
TIME 1511

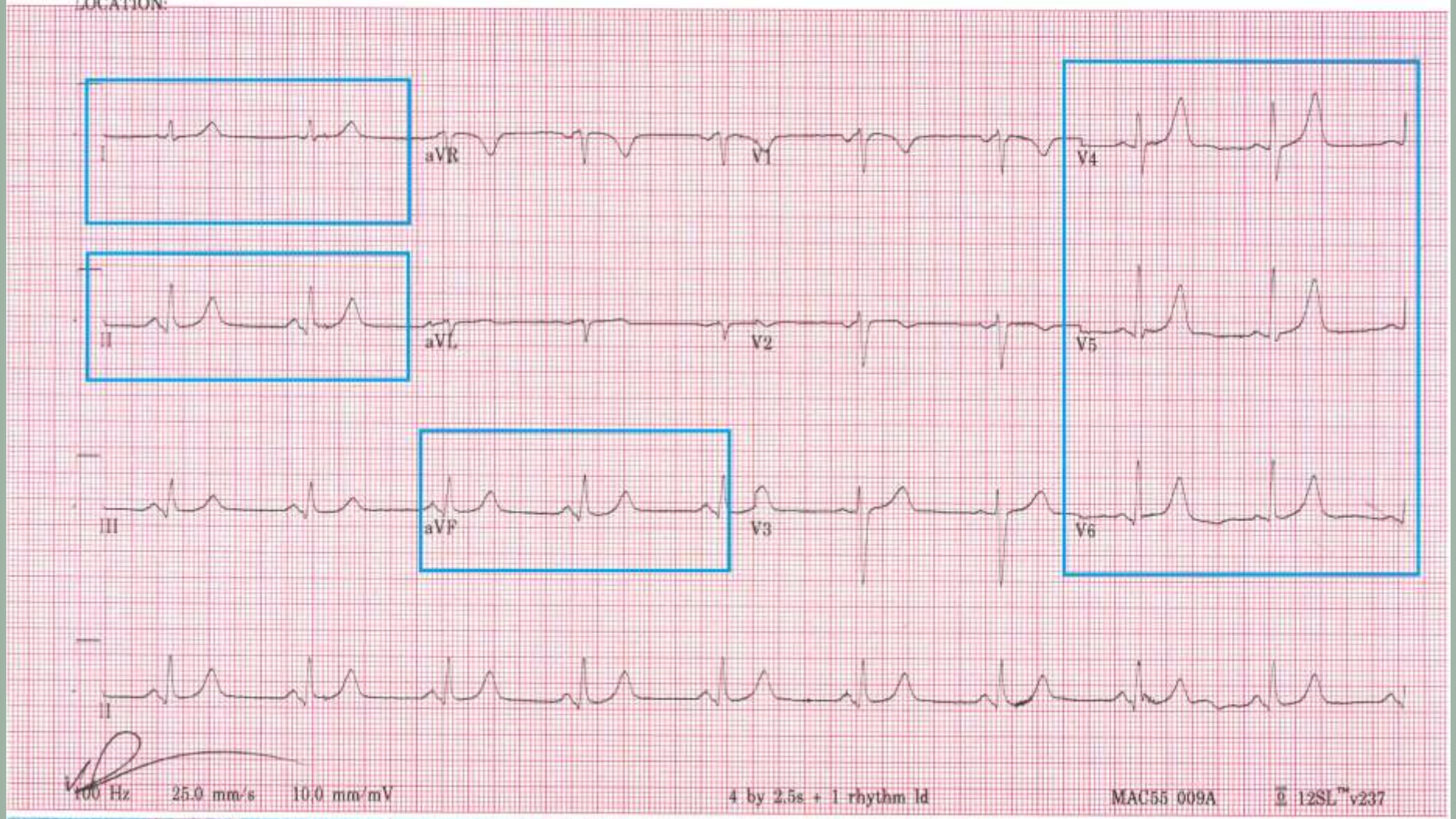
K+ = 6.7

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

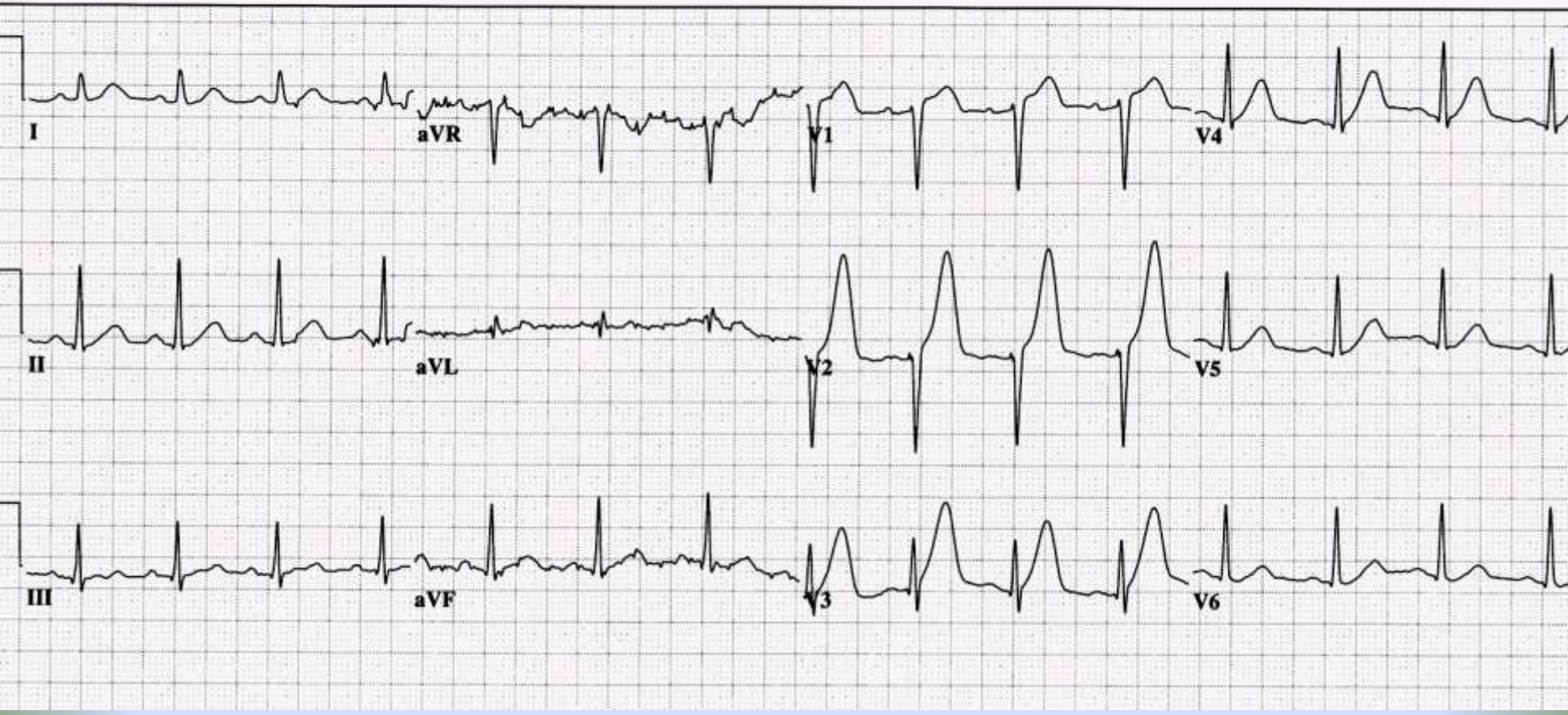
30 yr
Male Black

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:

| | | |
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Normal sinus rhythm
Normal ECG
No previous ECGs available

***HIGHLIGHTED AREAS = HYPERACUTE T WAVES***

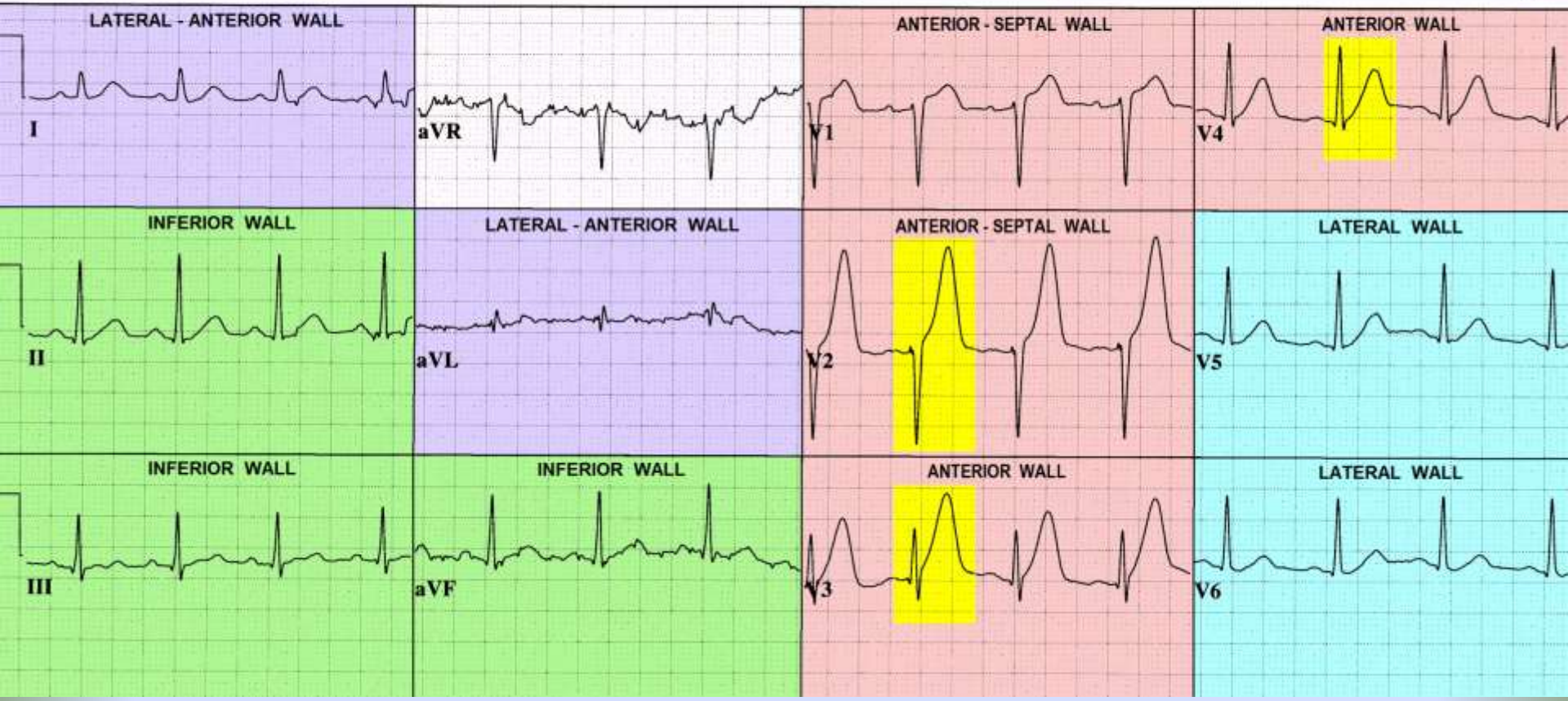
**CORONARY ARTERIAL DISTRIBUTIONS:**

***V1 - V4 = LEFT ANTERIOR DESCENDING ( LAD )***

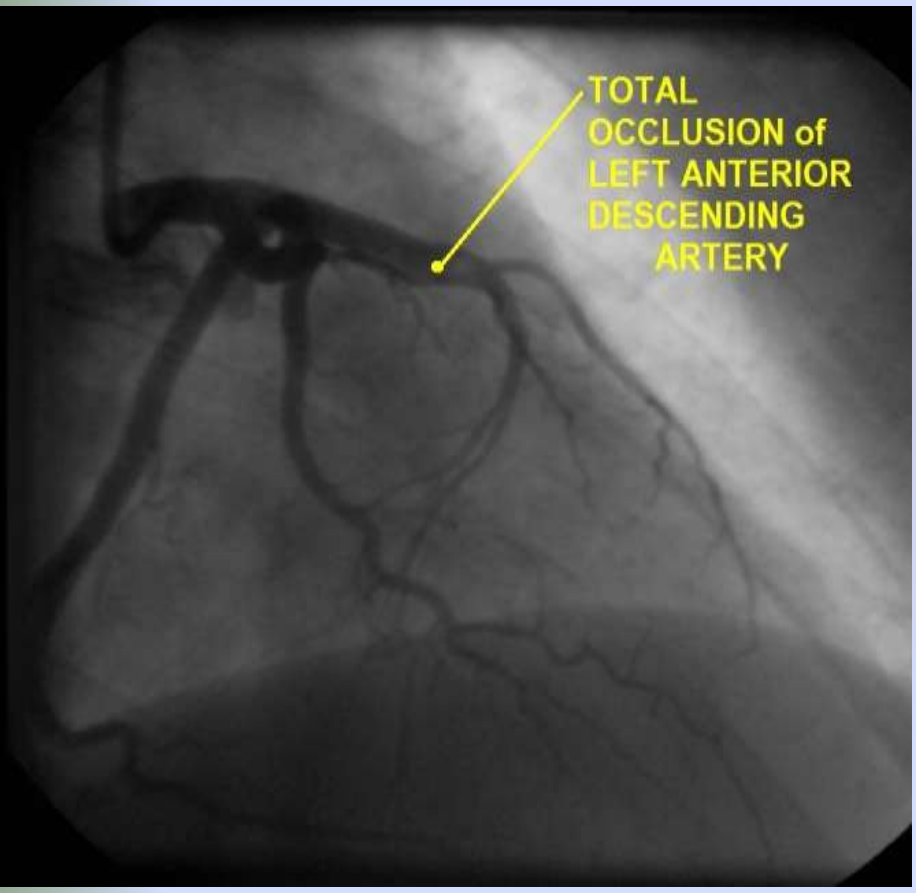
***I, AVL = DIAGONAL (DIAG) off the LAD or OBTUSE MARGINAL (OM) off CIRCUMFLEX (CX)***

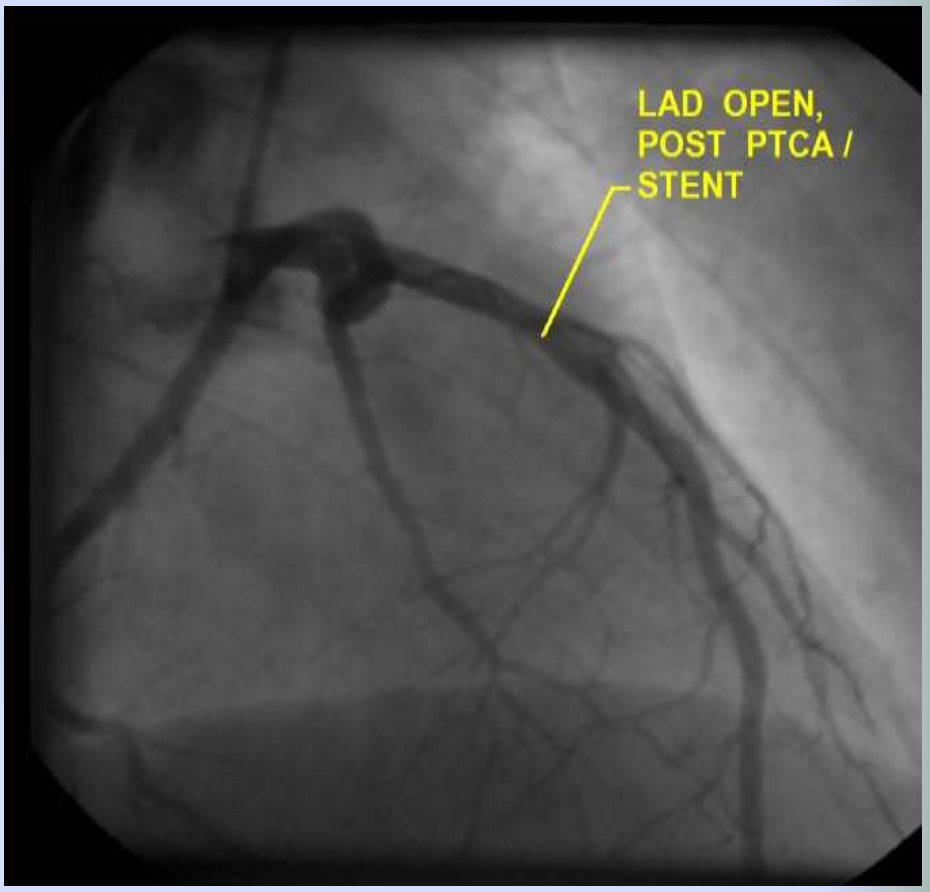
***V5, V6 = CIRCUMFLEX***

***II, III, AVF = RIGHT CORONARY ARTERY or CX***



# Cath Lab findings:



# Dynamic ST-T Wave Changes:

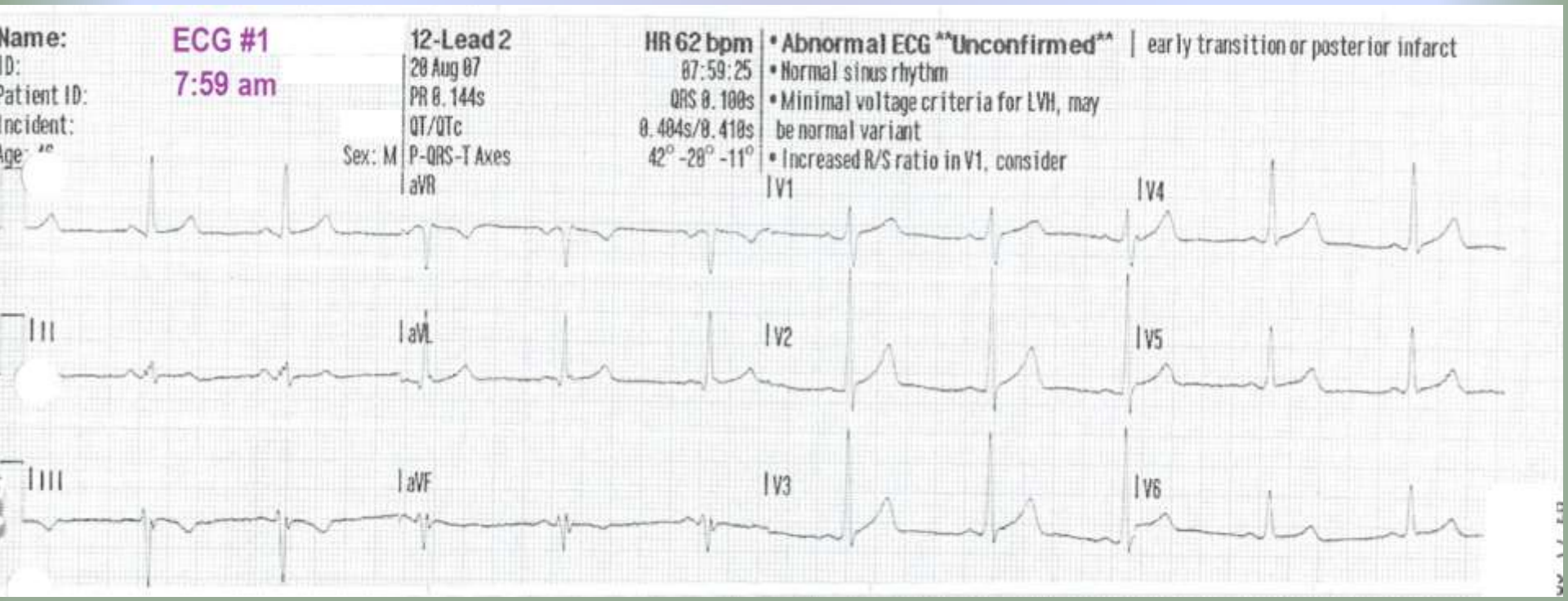
- Other than HEART RATE related variations (which affect intervals), ***J Points, ST-Segments and T Waves SHOULD NOT CHANGE.***

# Dynamic ST-T Wave Changes:

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

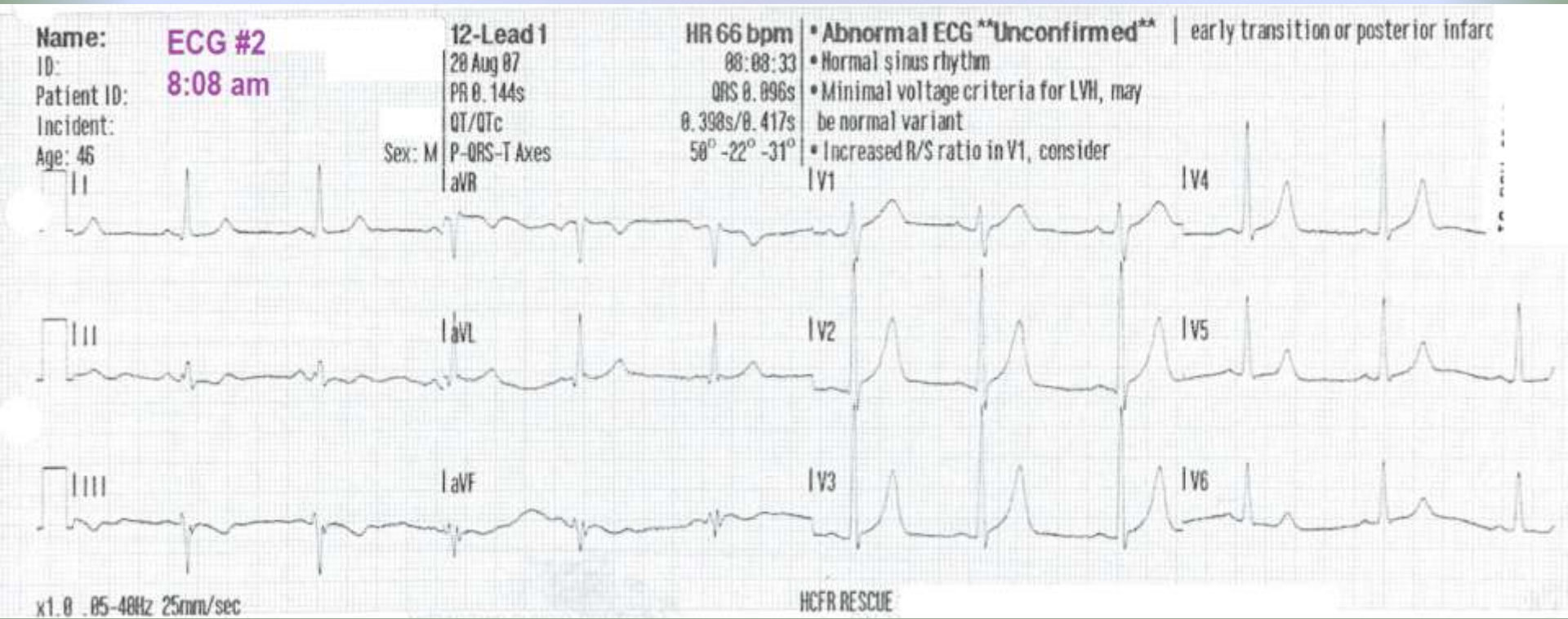
# 46 year old male

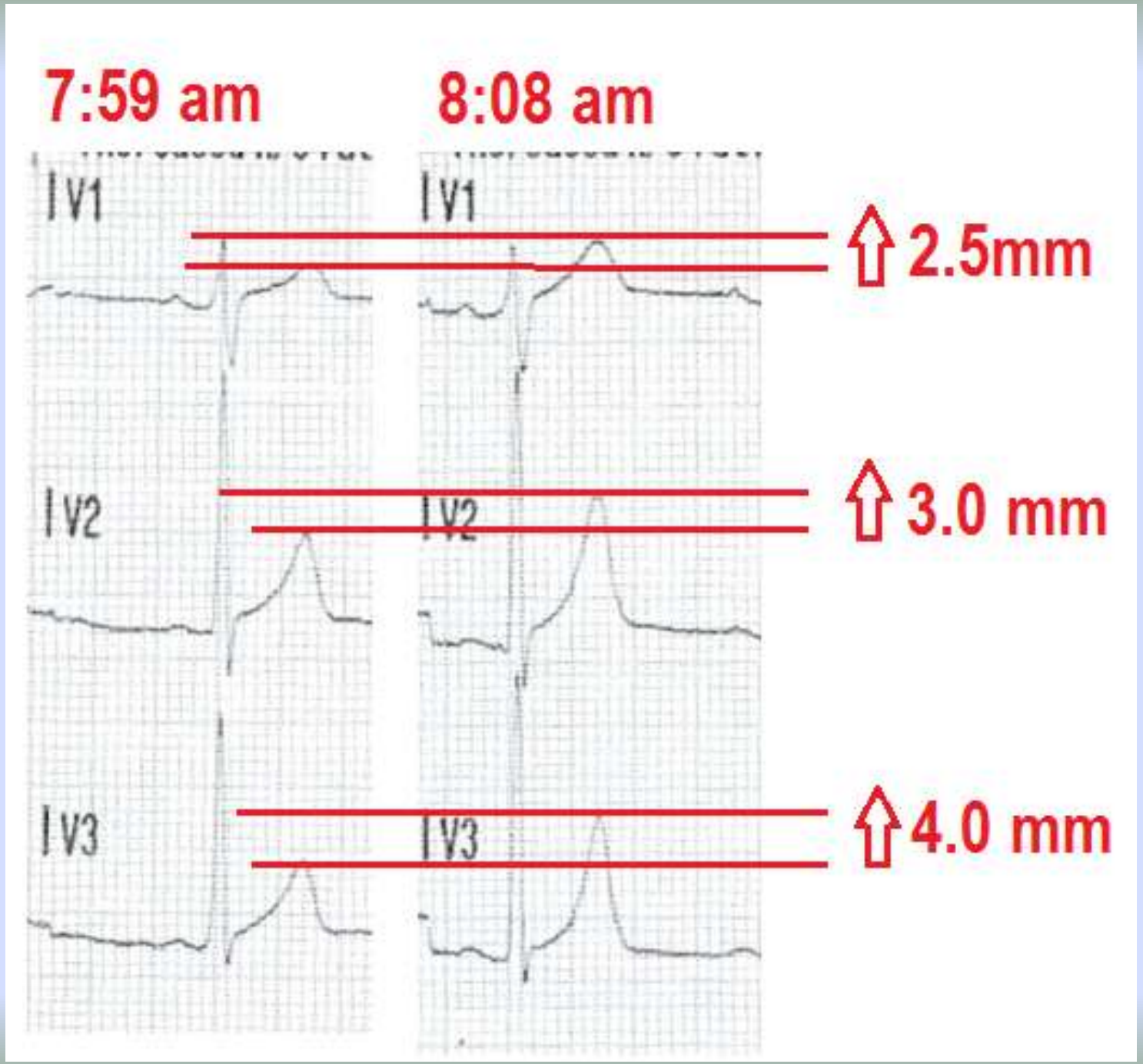
- Exertional dyspnea  X “several weeks”
- Intermittent chest pressure  X  last 3 hours. Currently pain free.



# 46 year old male: ECG 1

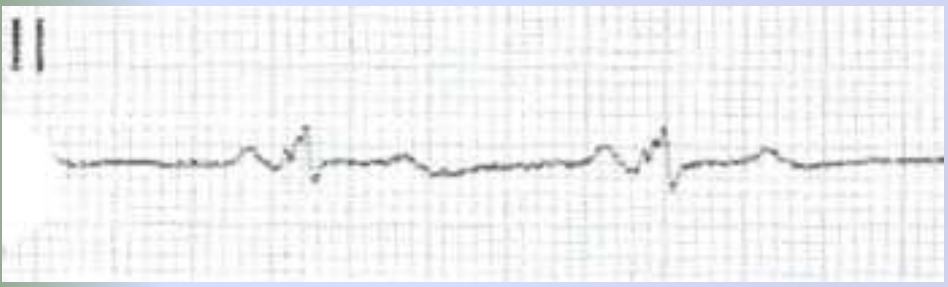
- Chest pressure has returned, “5” on 1-10 scale. 2nd ECG obtained due to “change in symptoms”:



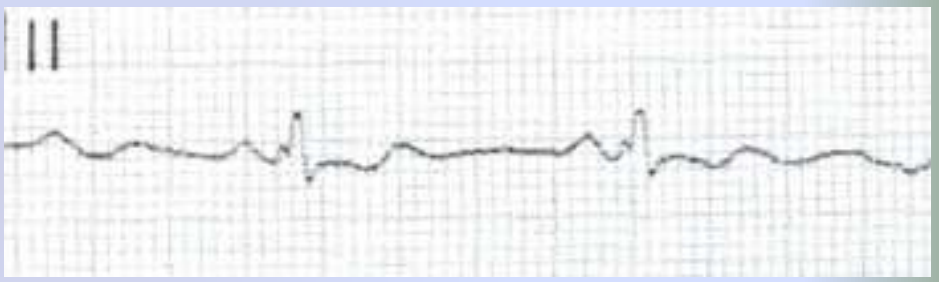

7:59 am
8:08 am
V1
V1
2.5mm
V2
V2
3.0 mm
V3
V3
4.0 mm

# ST-Segment Depression

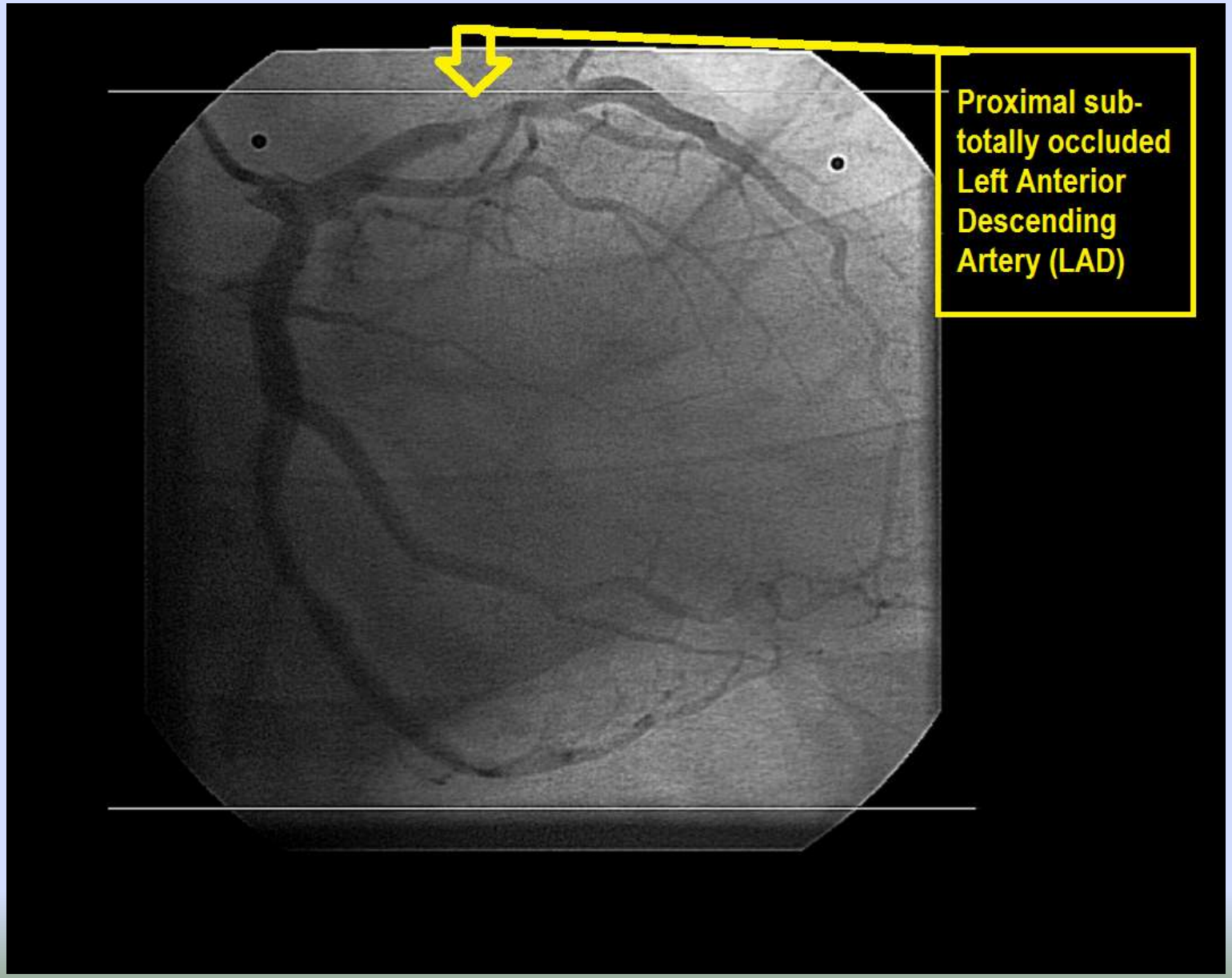
**7:59 am**



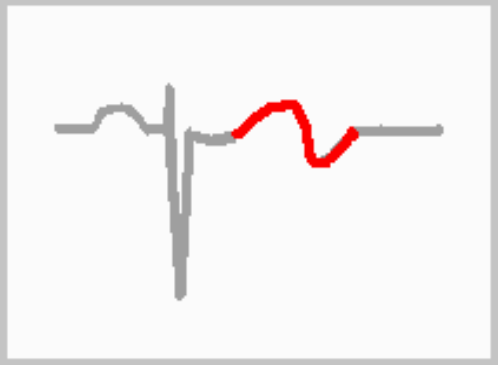

**8:08 am**



# Cath Lab Angiography:



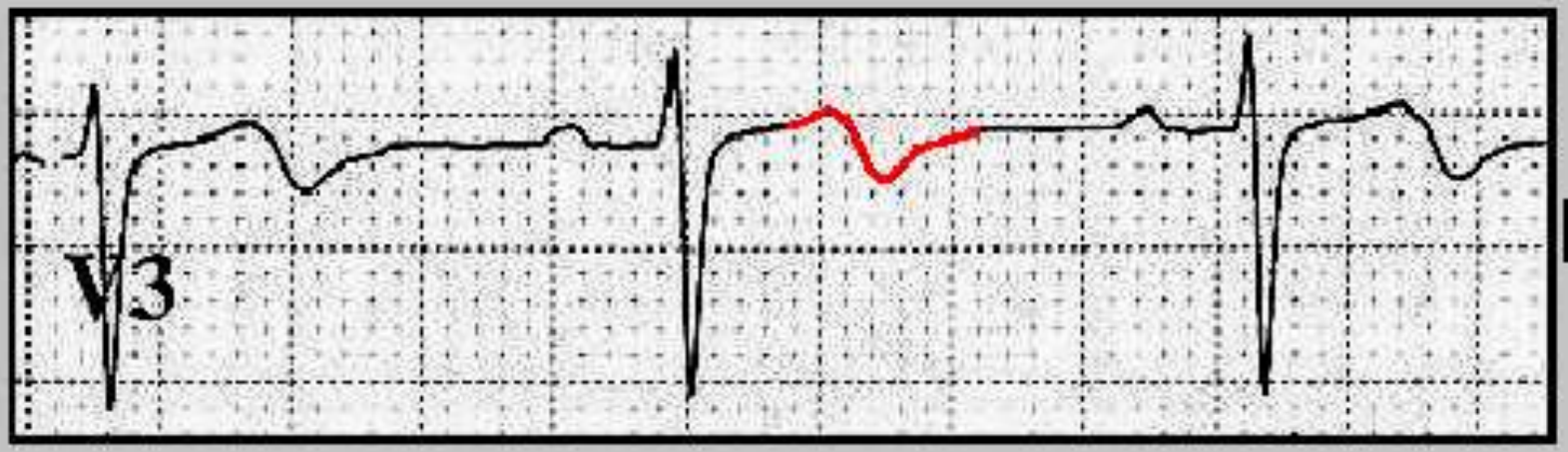
# ISCHEMIA



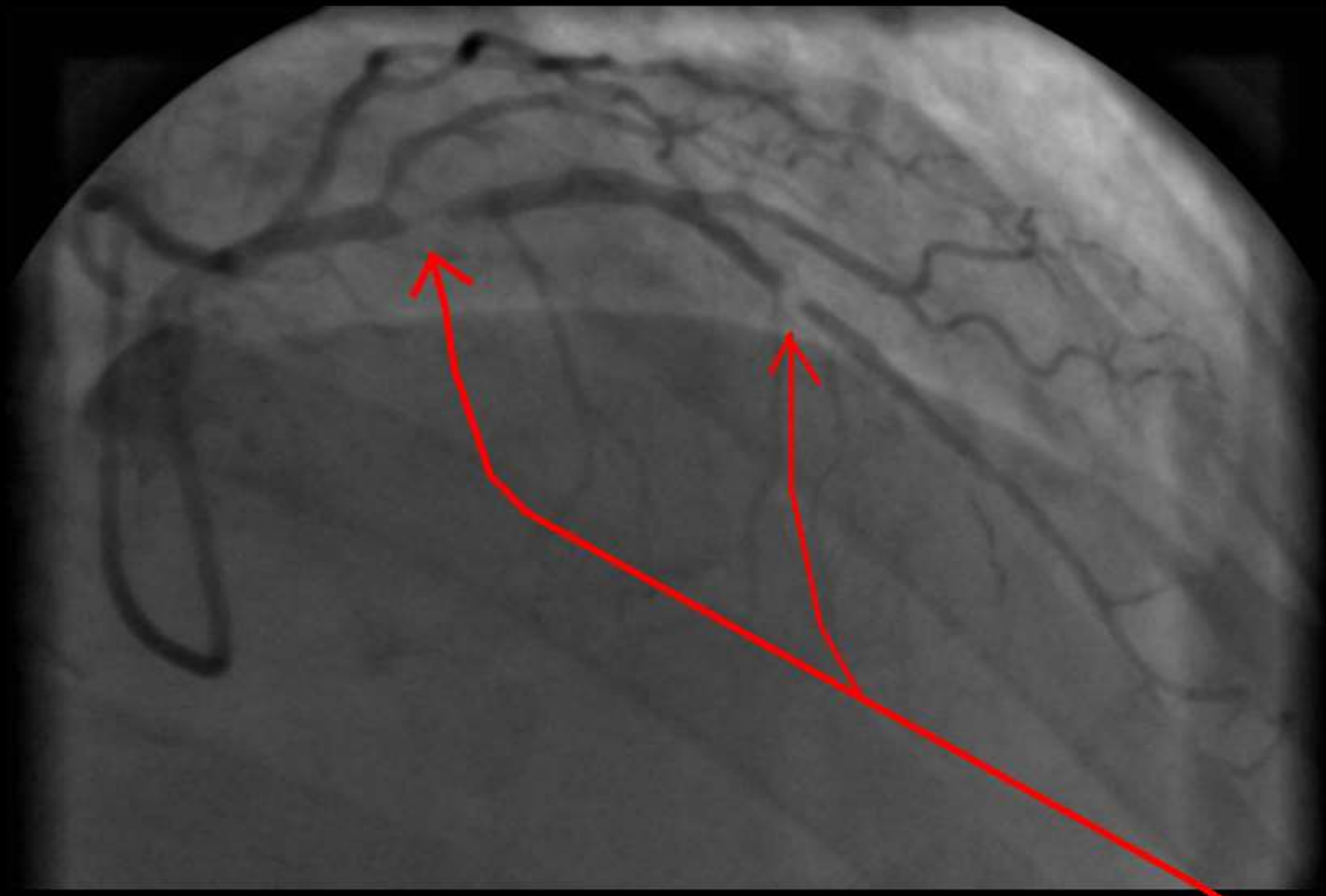
## *BI-PHASIC T WAVE*

- 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:

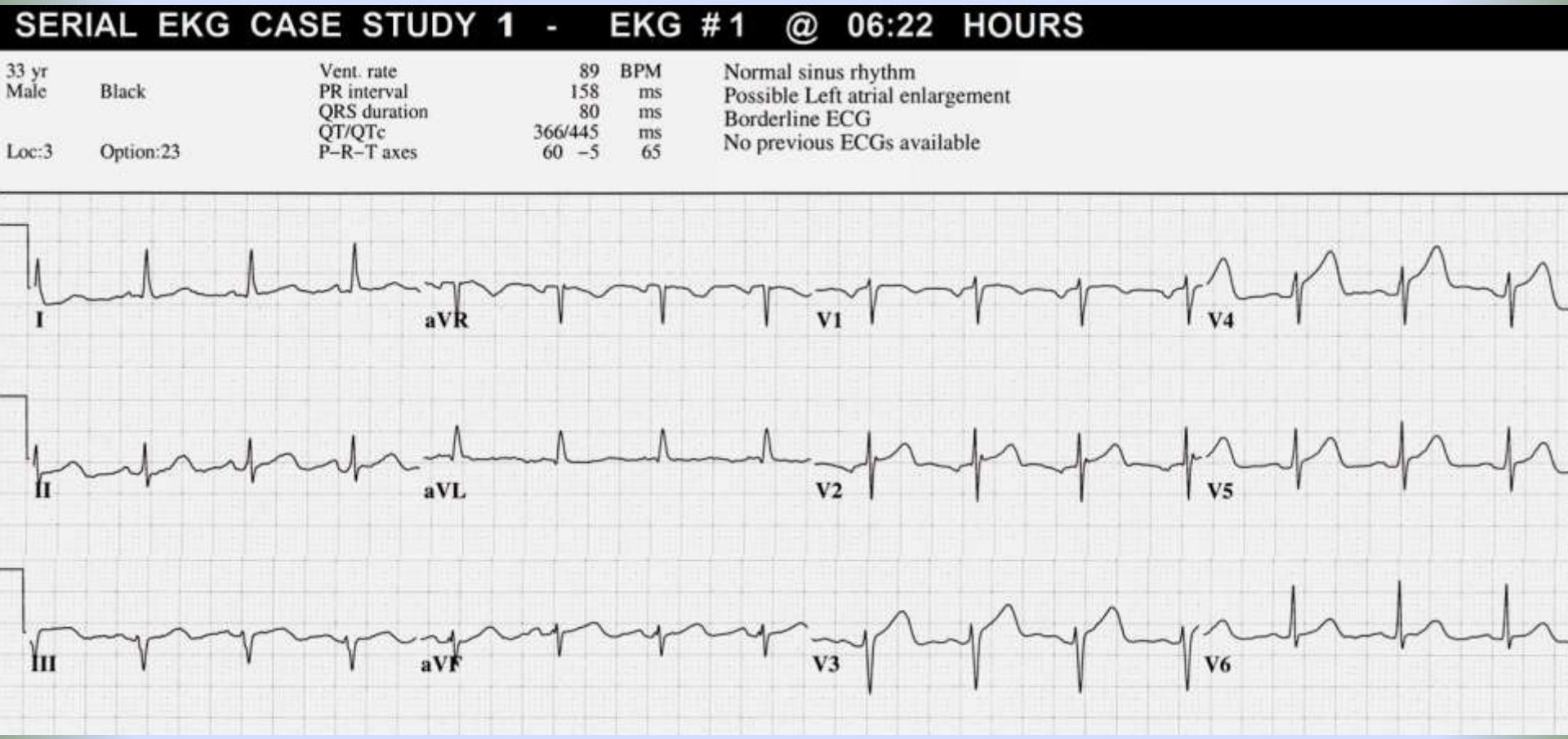
- **Present in 14-18% of patients admitted with unstable angina**
- **75% patients not treated developed extensive Anterior MI within 3 weeks.**
- ***Median Average time from presentation to Acute Myocardial Infarction – 8 days***

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

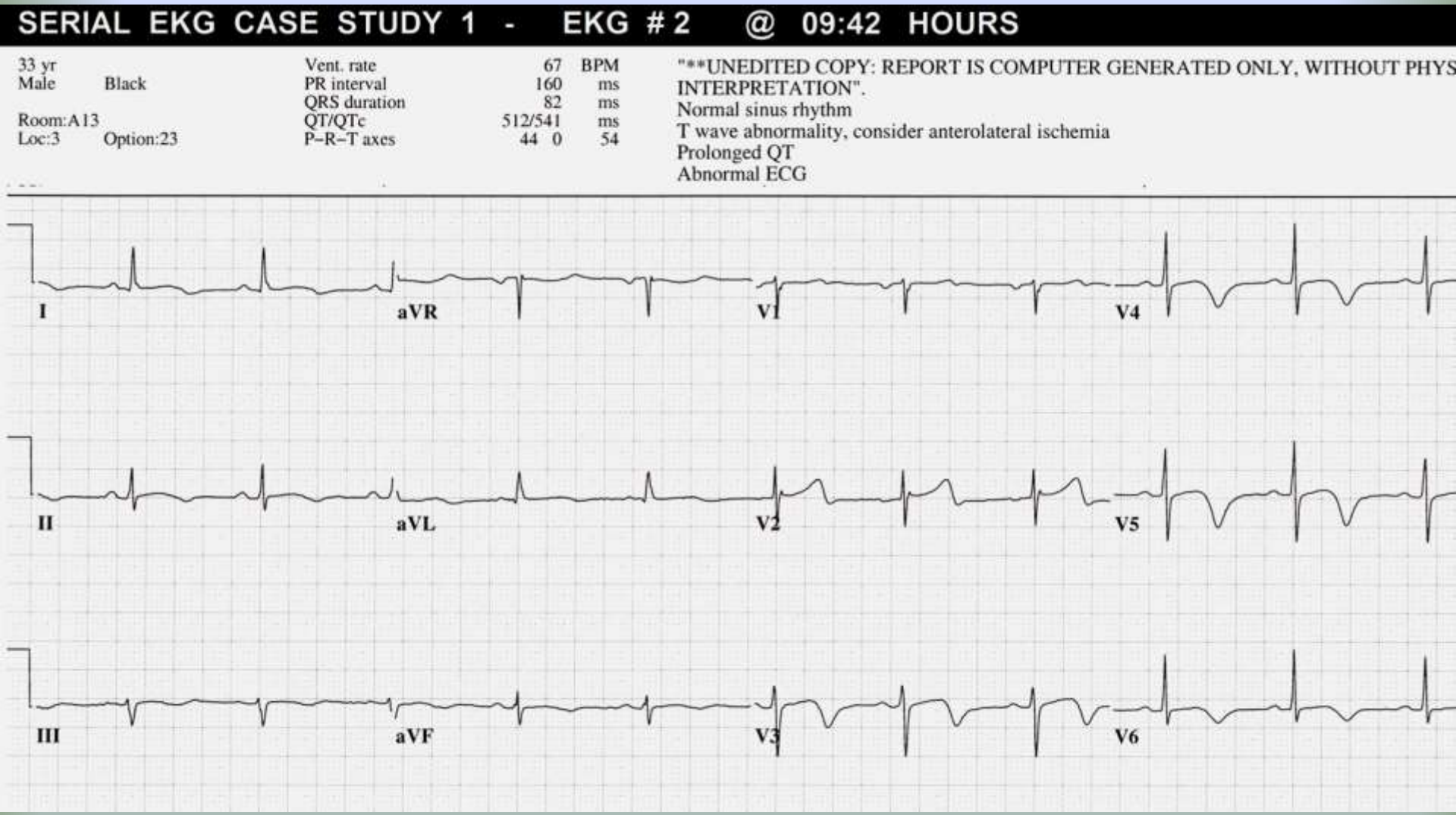
# Wellen's Syndrome Case Study

- 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

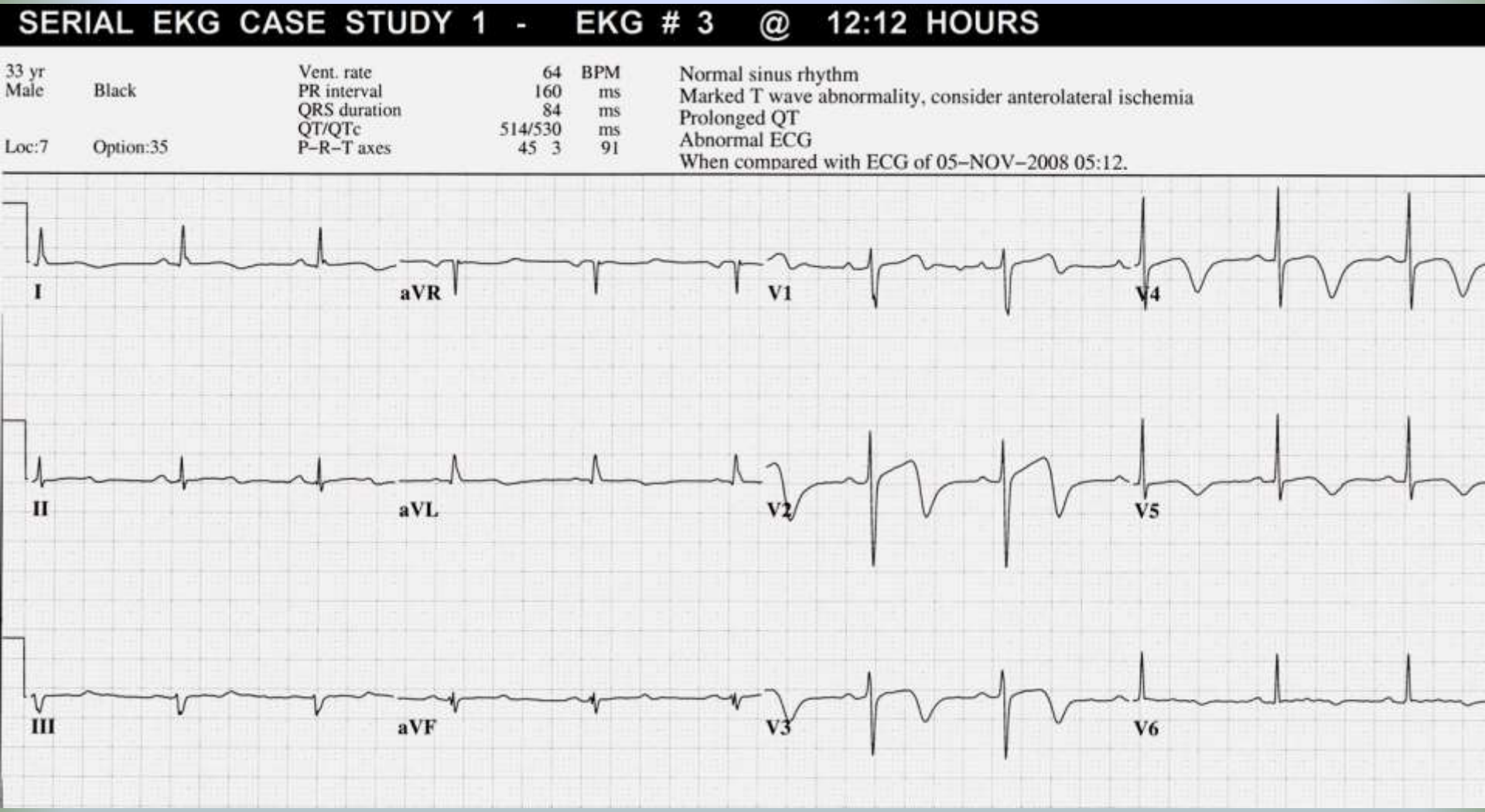


***DYNAMIC ST-T Wave Changes ARE PRESENT !!***

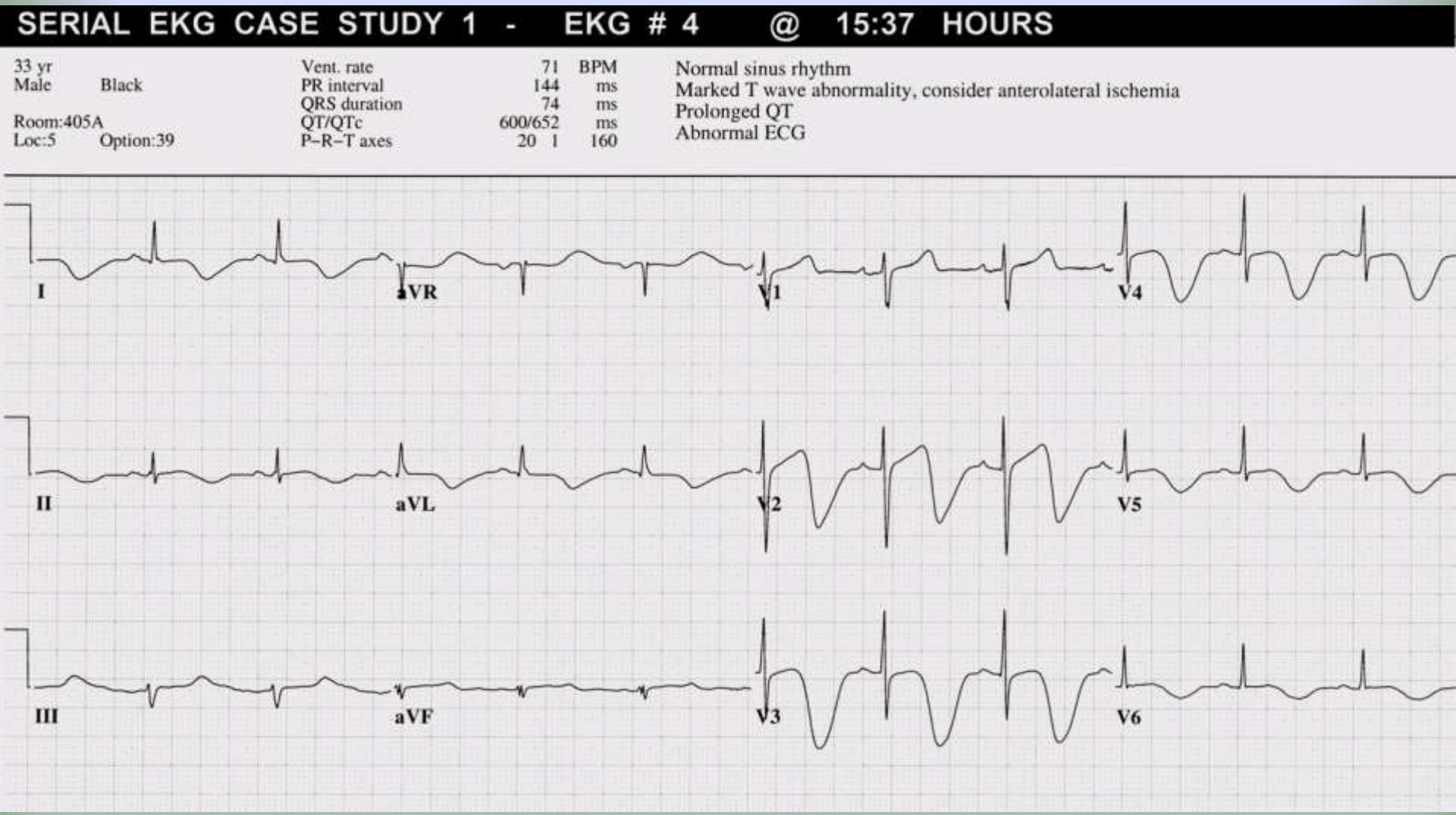
# NOW

***is the time for the STAT CALL to the CARDIOLOGIST !!!!***

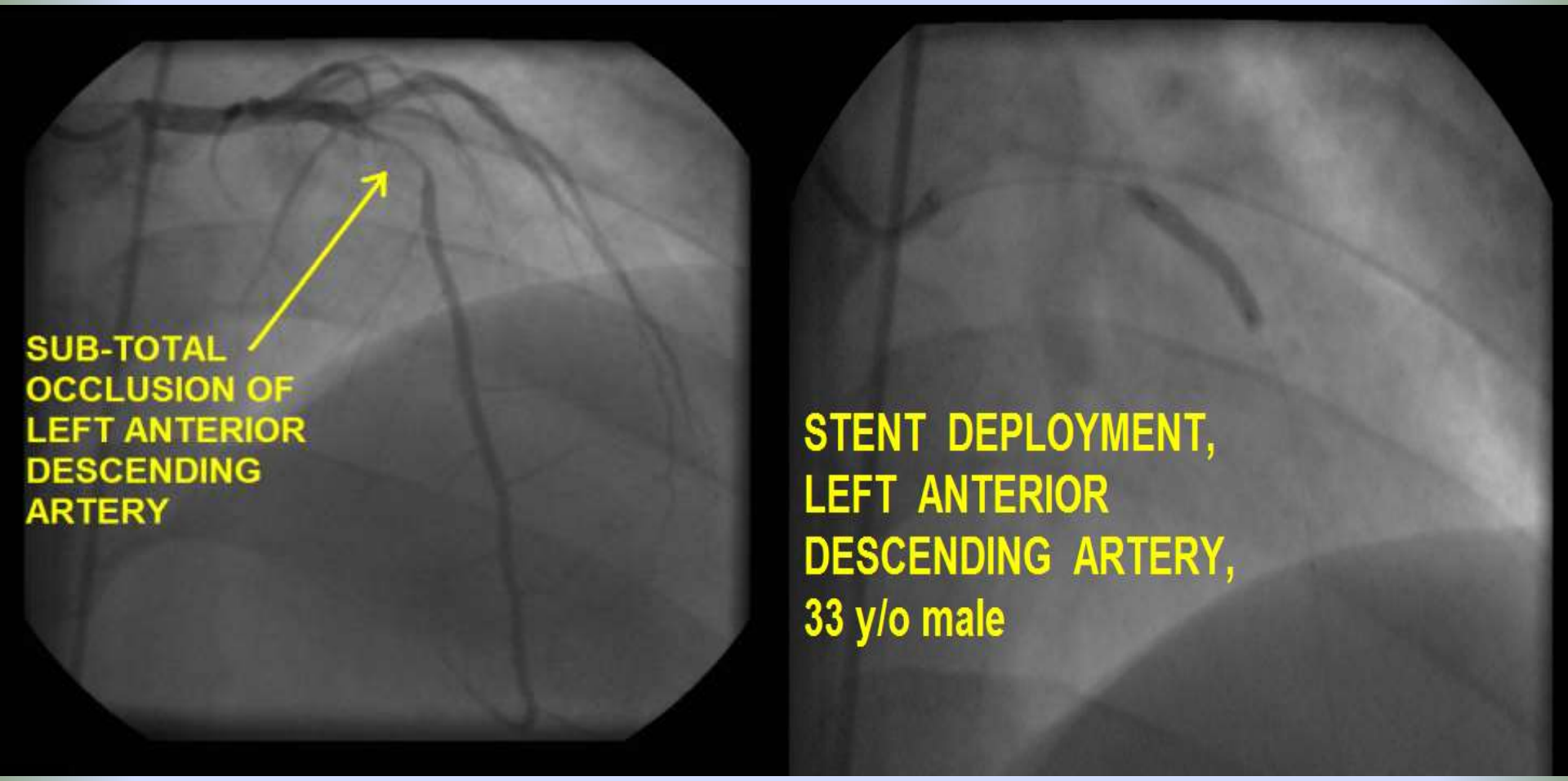
# Wellen's Syndrome Case Study



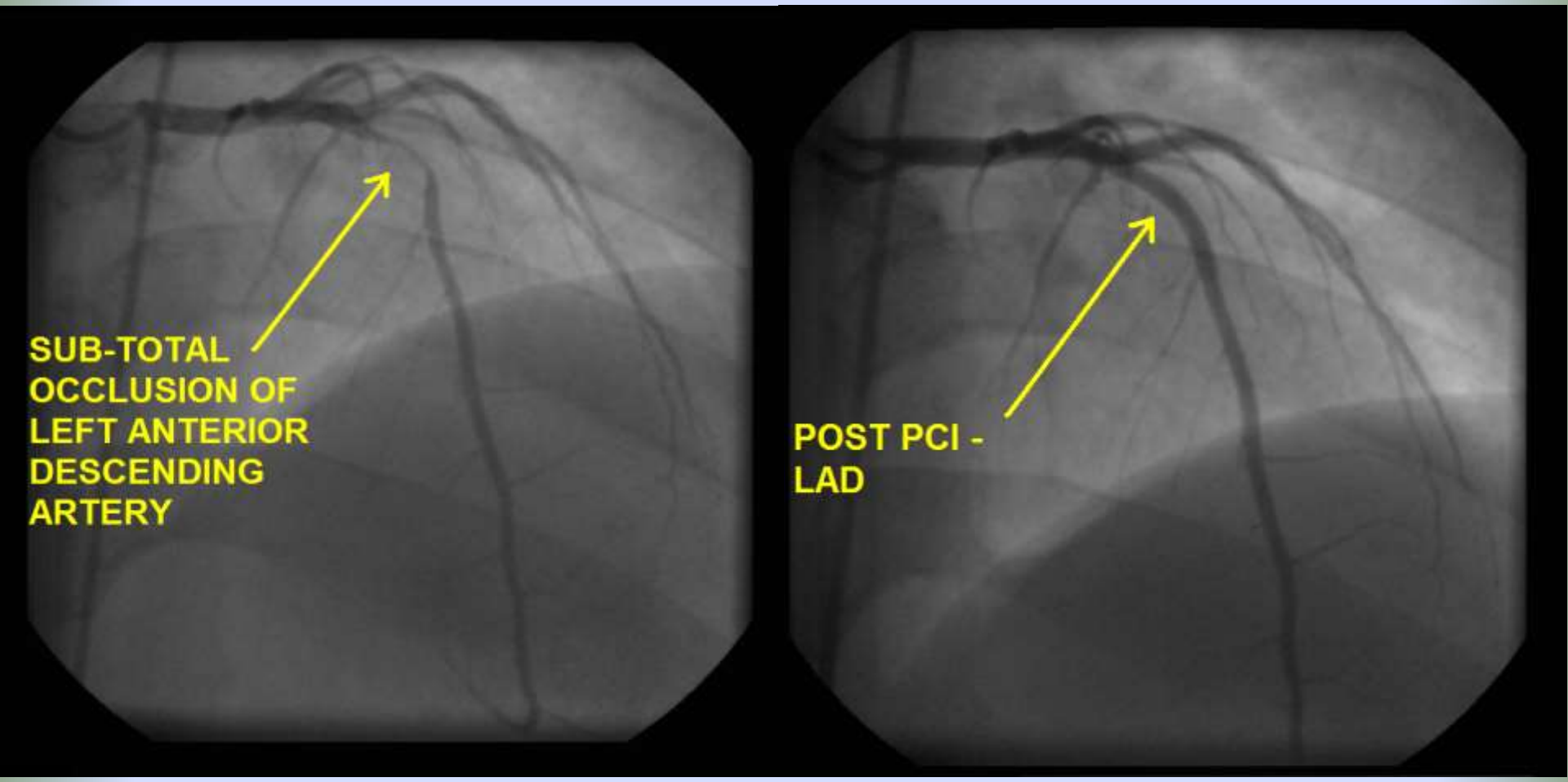
# Wellen's Syndrome Case Study



# Wellen's Syndrome Case Study



# Wellen's Syndrome Case Study

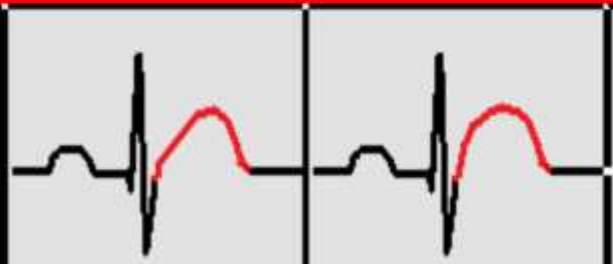
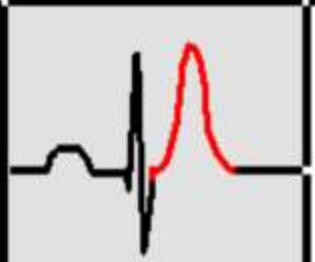
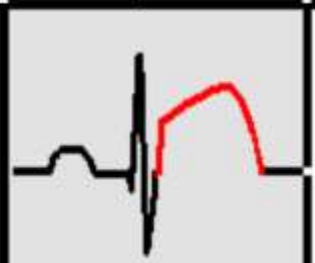
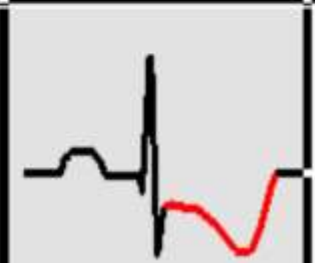


# Additional Resources:

- Wellen's Syndrome, NEJM case study

# 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**<br>- **ACUTE MI** - ( RECIPROCAL CHANGES )<br>- **ISCHEMIA** |

# ECG CRITERIA for DIAGNOSIS of STEMI:

# (ST ELEVATION @ J POINT)

## *LEADS V2 and V3:

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

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

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

**ALL OTHER LEADS:** 1.0 mm or more,
in TWO or more
CONTIGUOUS LEADS

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

# STEMI Criteria for 18 Lead ECGs:

***Right-Sided Chest Leads***

***(V3R – V6R): 0.5 mm***

***Posterior Chest Leads***

***(V7 – V9): 0.5 mm***

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

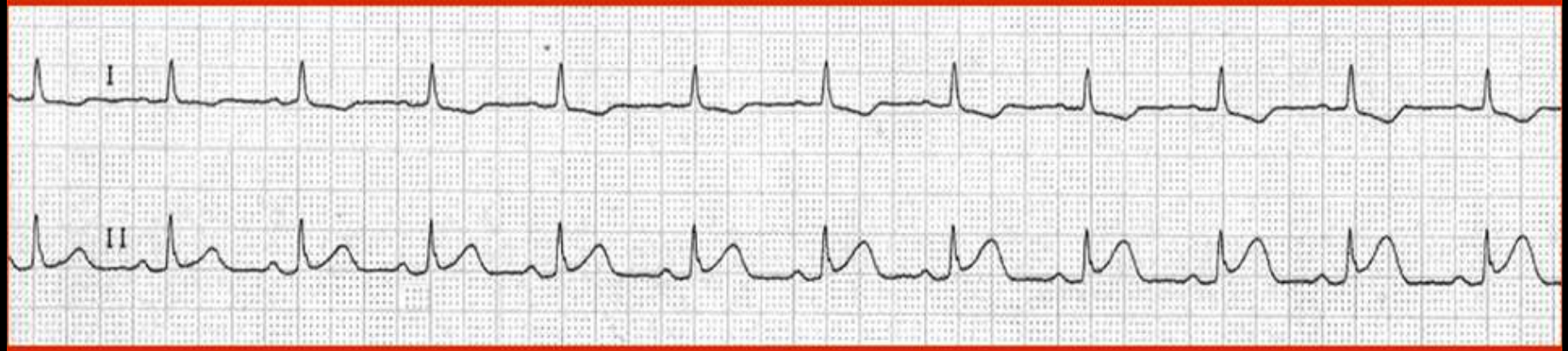
# Abnormal ST Elevation Criteria: ACC/AHA 2009 "Standardization and Interpretation of the ECG, Part VI Acute Ischemia and Infarction," Galen Wagner, et al

**Recommendations**

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

# ST SEGMENT ELEVATION:

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




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

**3 COMMON PATTERNS of ST SEGMENT ELEVATION From ACUTE MI:**

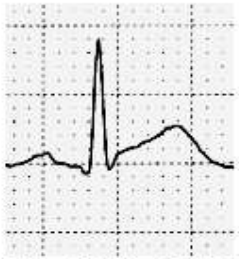
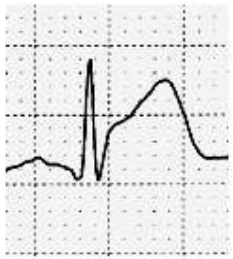
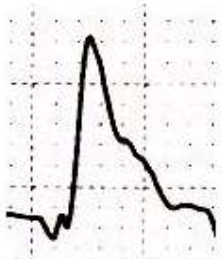
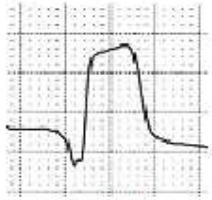
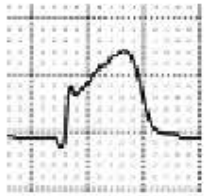
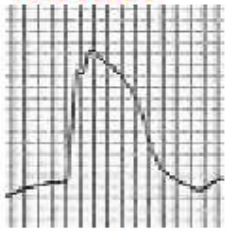
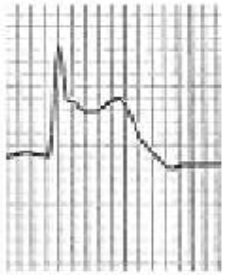
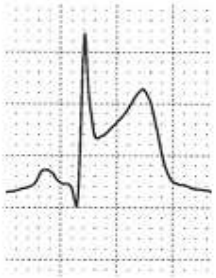
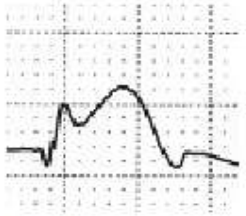
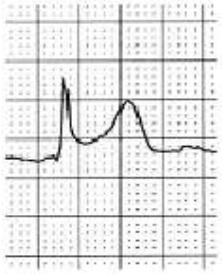
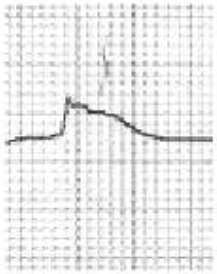
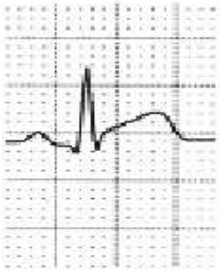
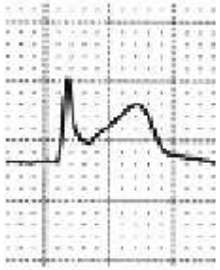
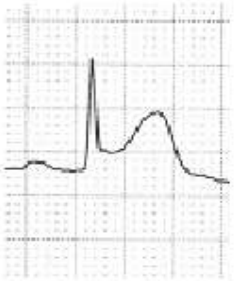
DOWNSLOPING S-T SEGMENT

FLAT S-T SEGMENT

UPSLOPING S-T SEGMENT

# 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


“NOWHERE”, NEW MEXICO,  1994

# 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 . . . .**

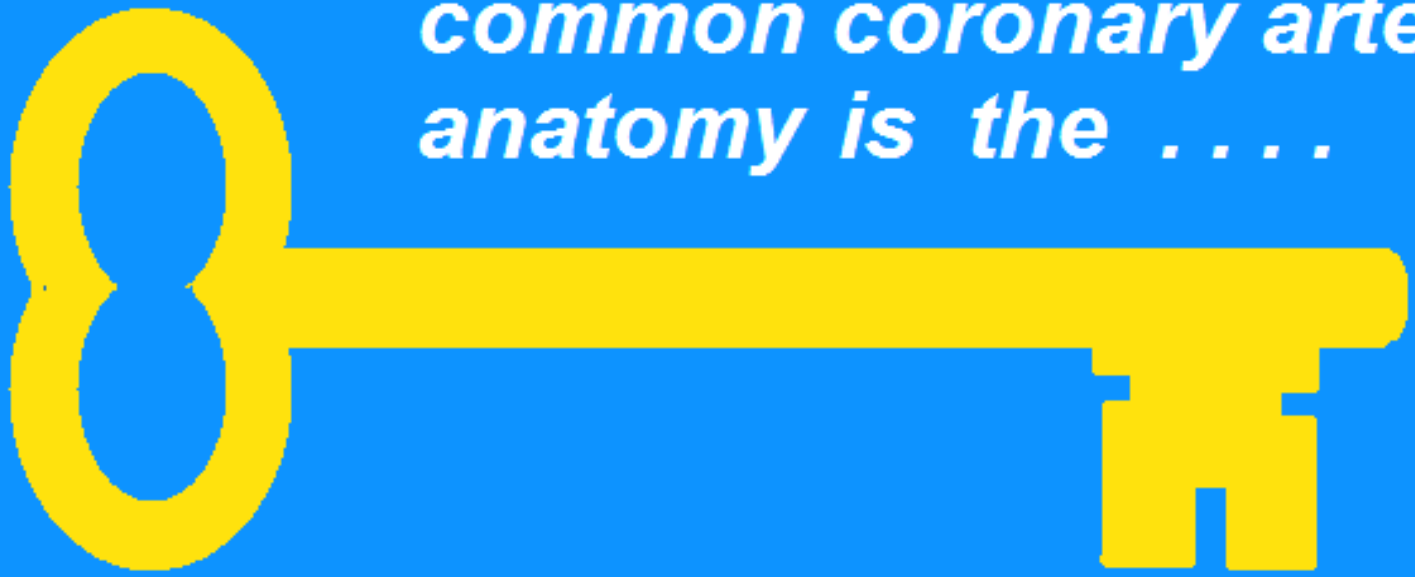
**. . . . . Will serve as a "crystal ball," allowing you to ANTICIPATE complications of STEMI . . . .**

# 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.

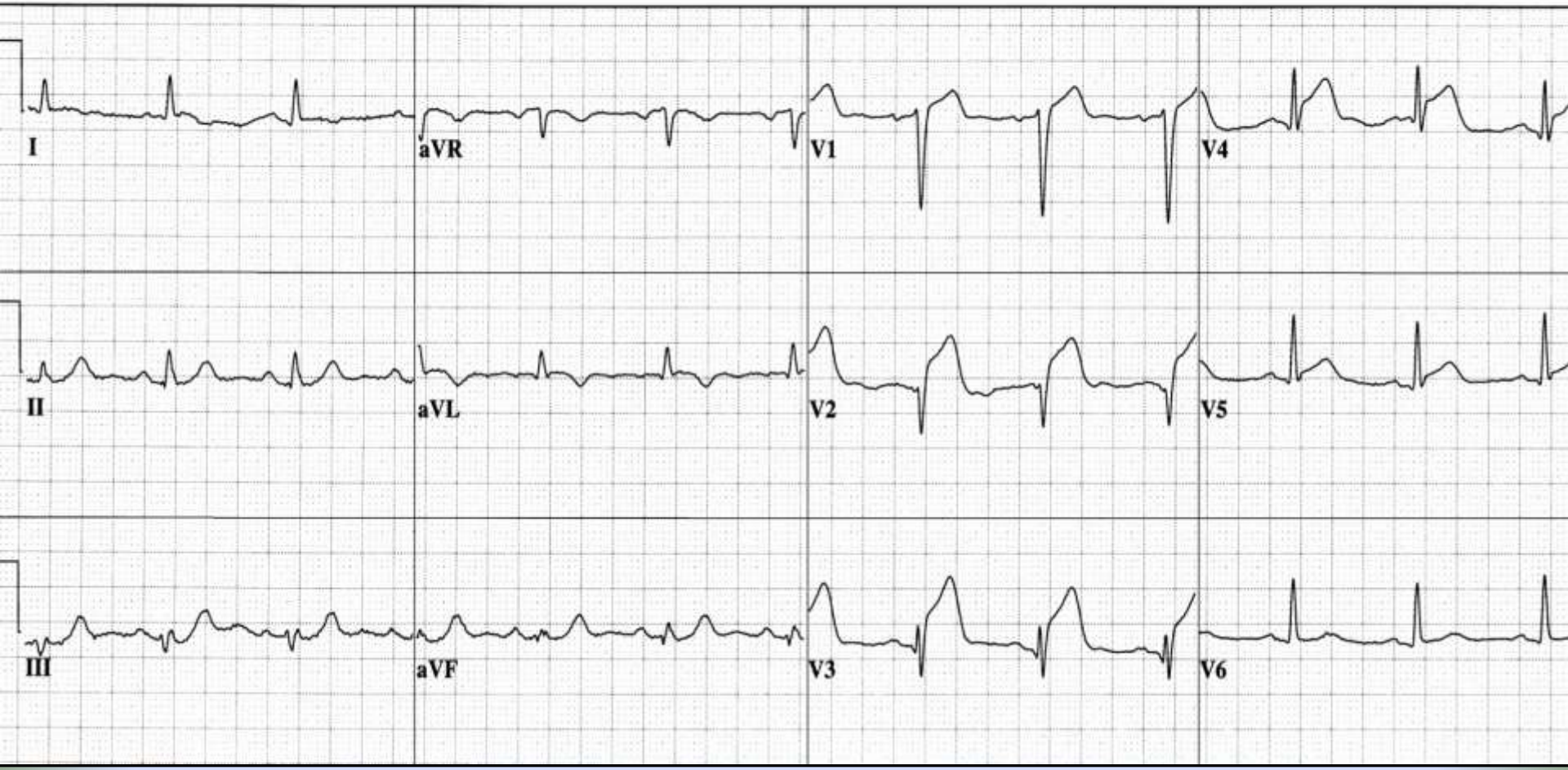
**VITAL SIGNS:** BP: 100/64, P: 75, R: 20, SAO2: 94%

**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



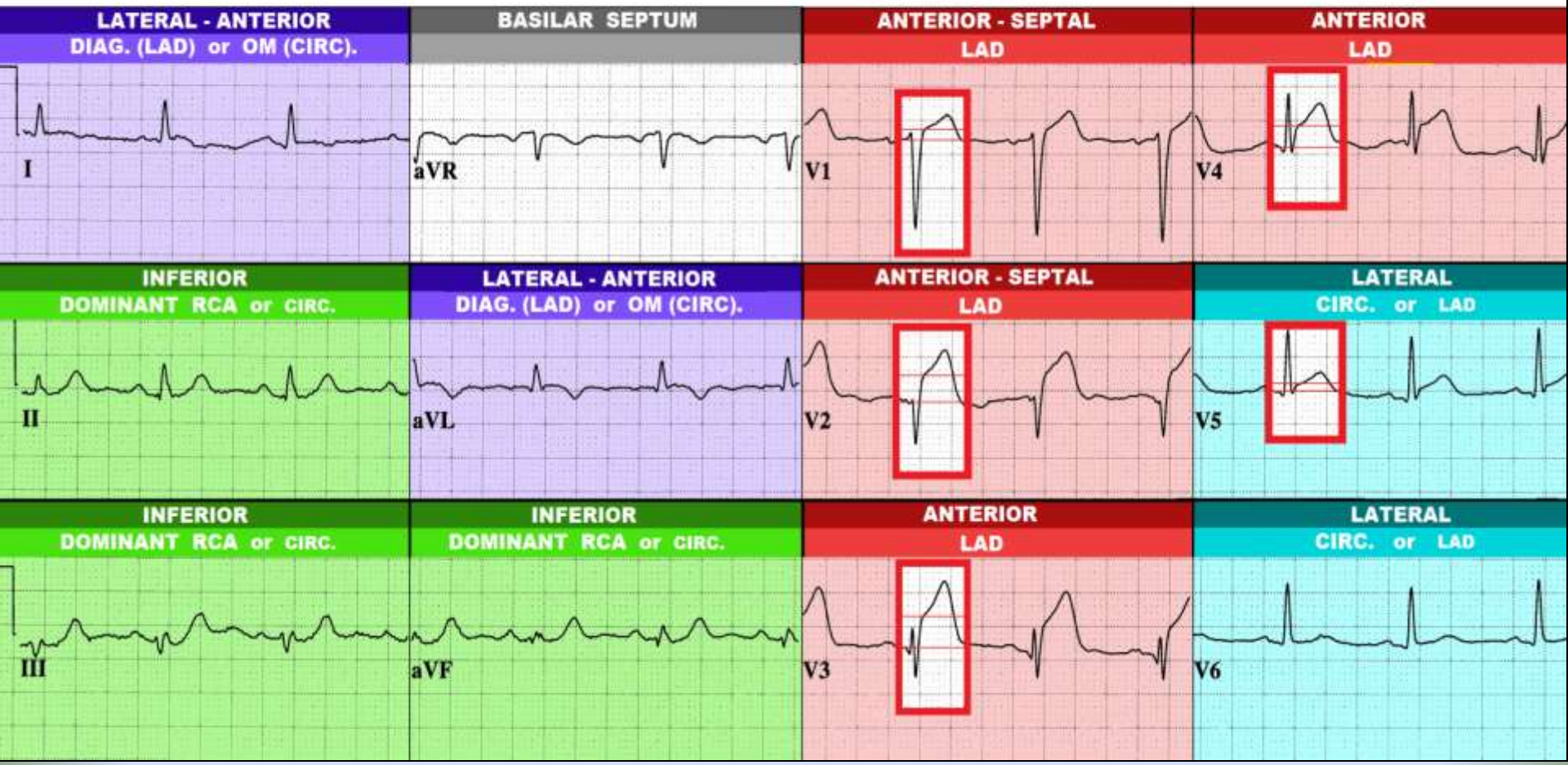
72 yr
Male Caucasian

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 !**

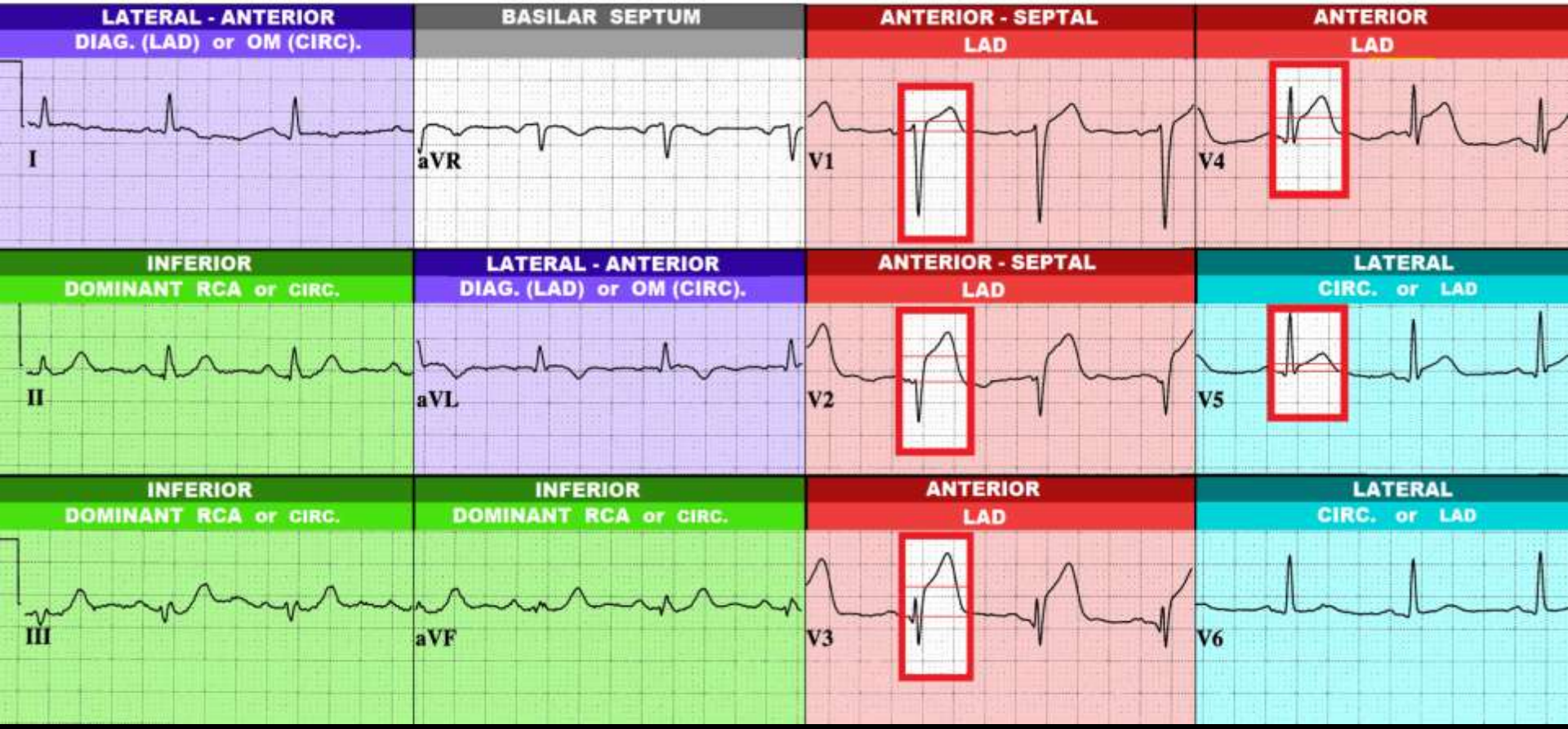
72 yr
Male Caucasian

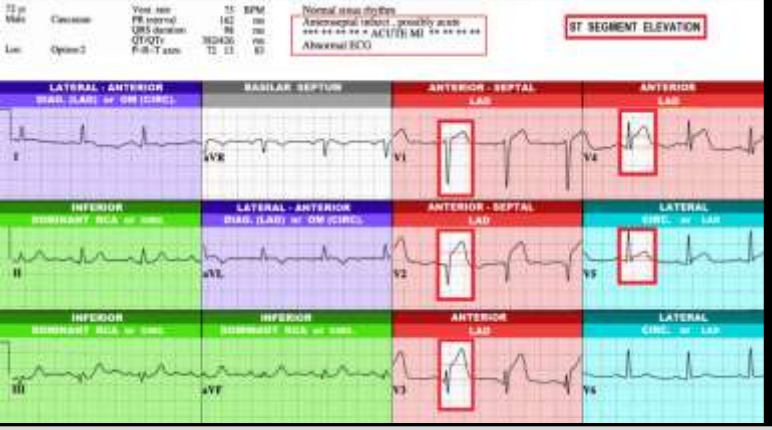
Loc: Option:2

| | | |
|---|---|---|
| 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

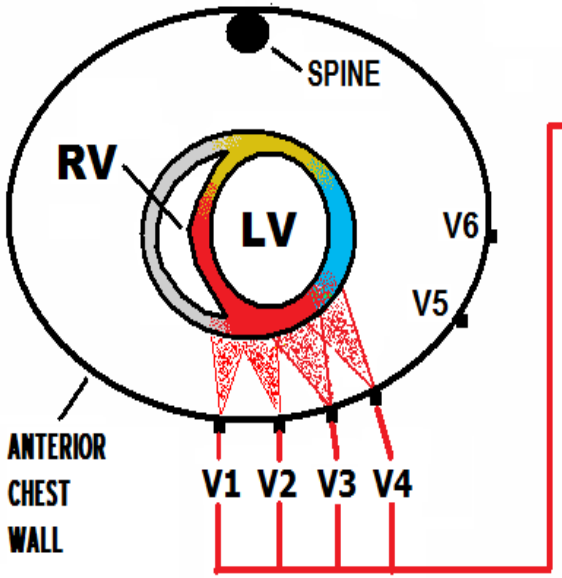
ST SEGMENT ELEVATION





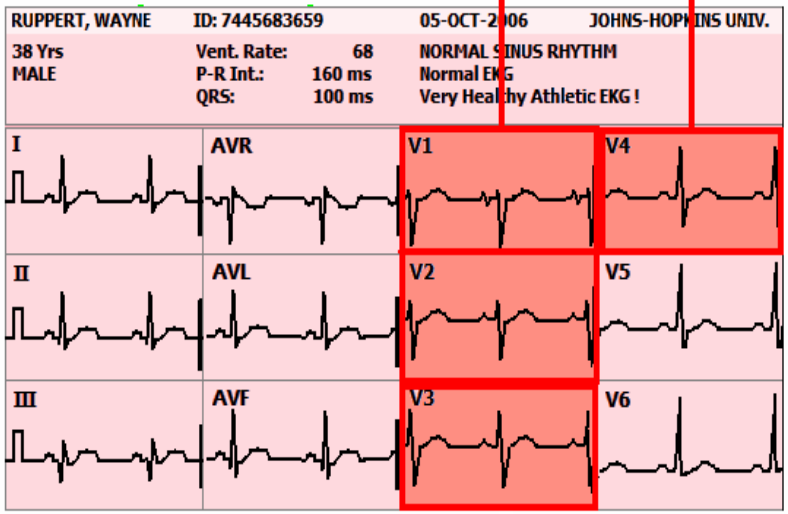

# V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL

## of the LEFT VENTRICLE

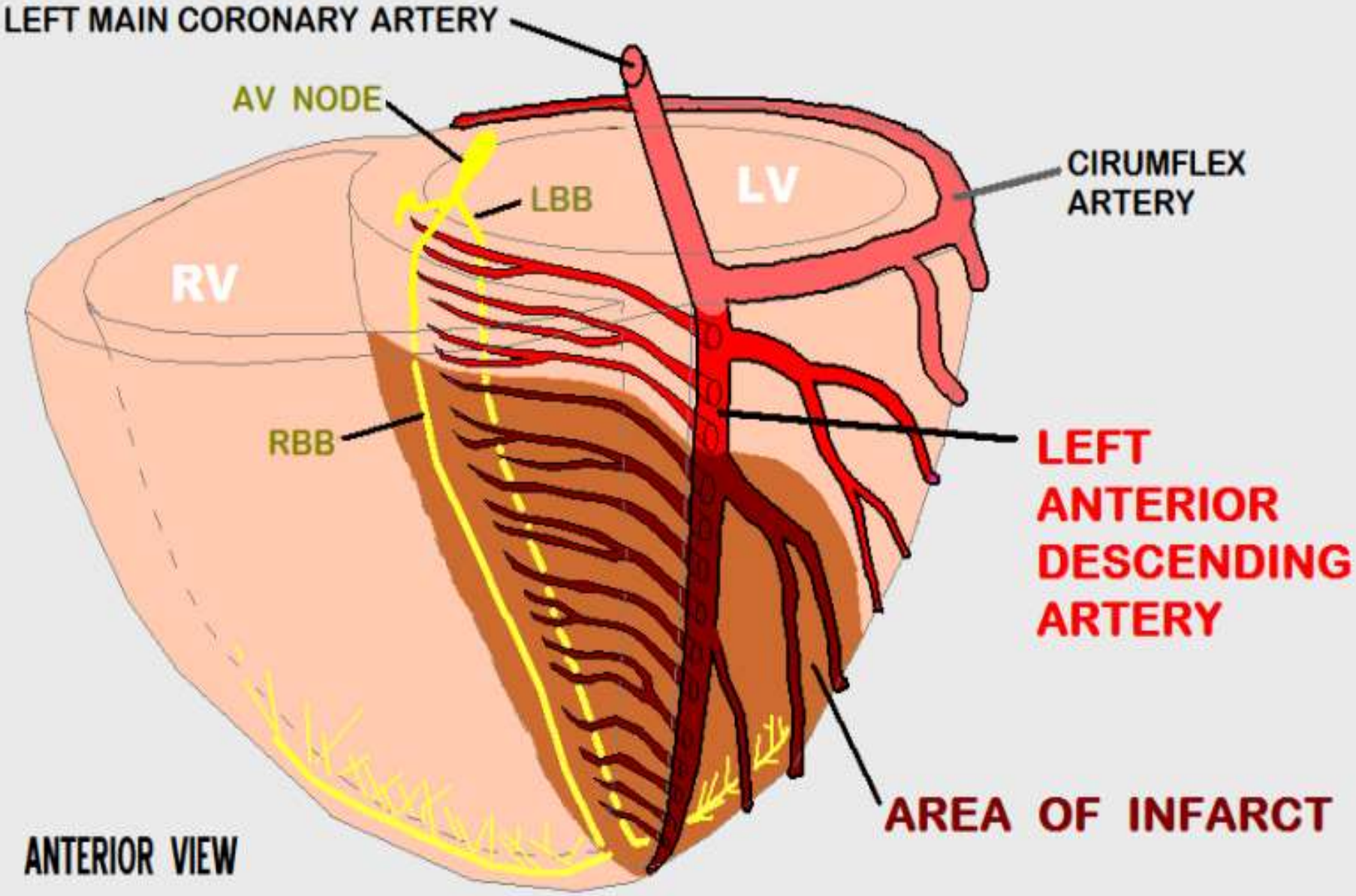



V1, V2 - ANTERIOR / SEPTAL

V3, V4 - ANTERIOR

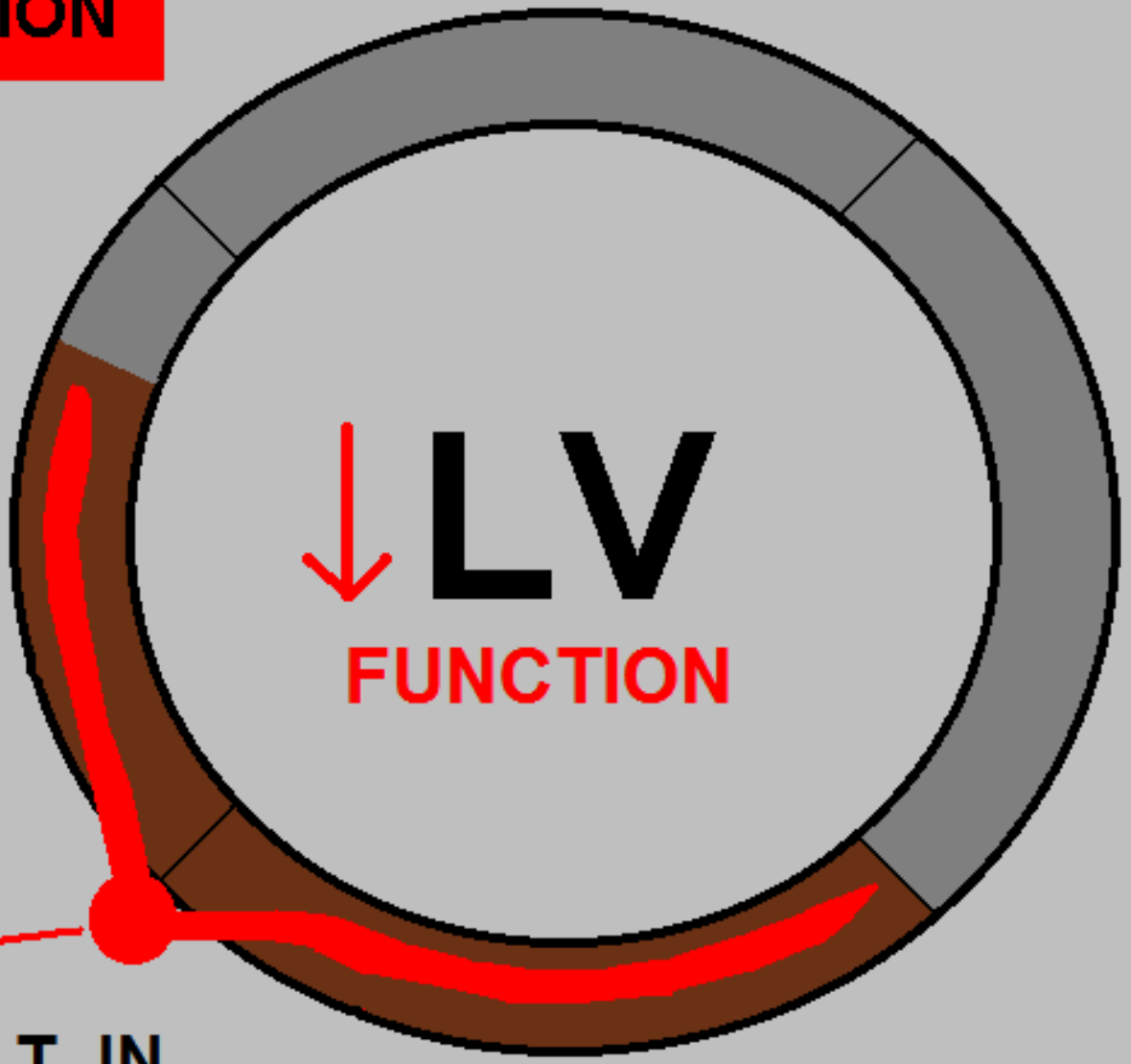


# OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY



# LAD DISTRIBUTION

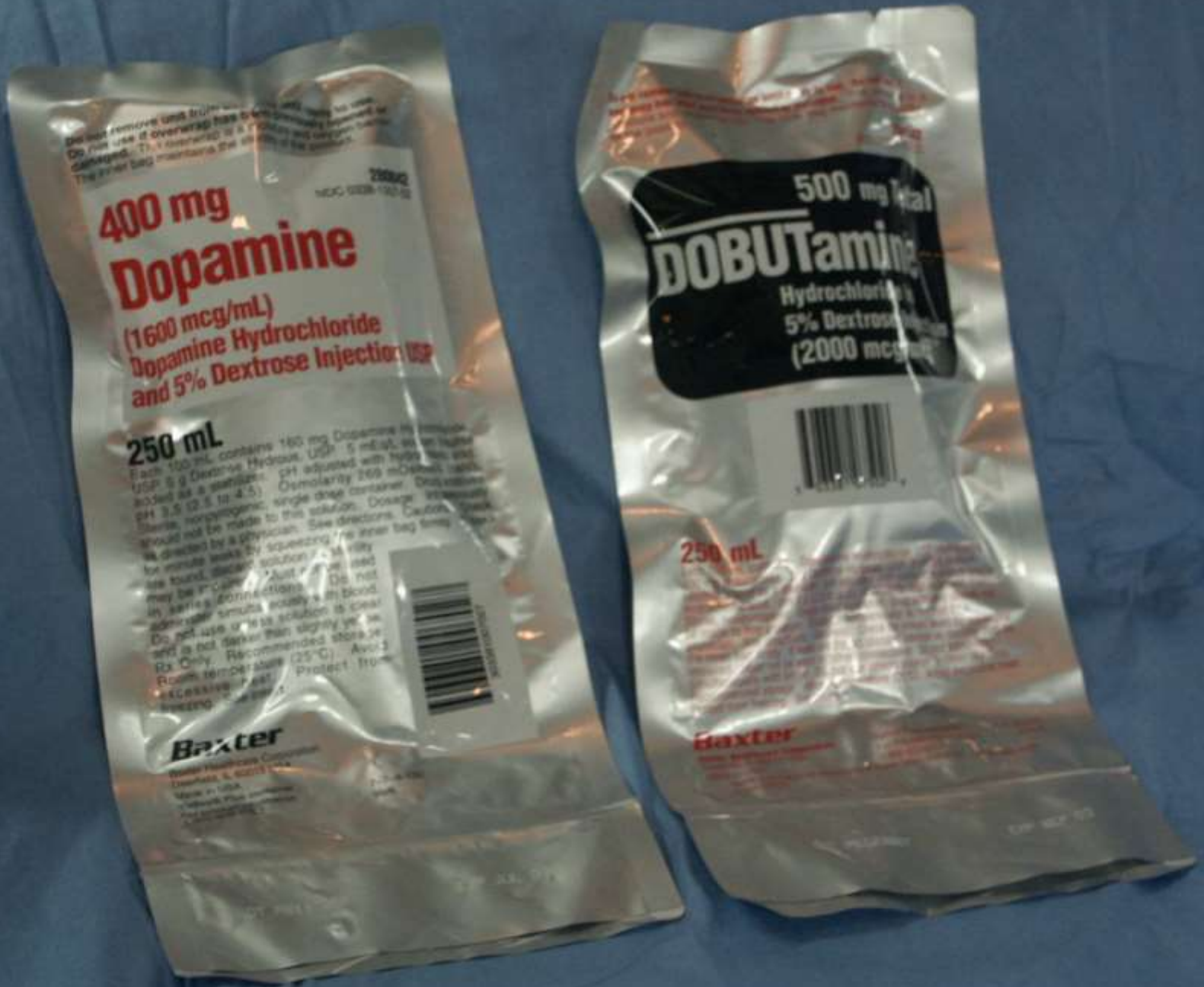
## 35 - 45 % of LV MUSCLE MASS




A BLOCKAGE OF THE LAD

CAN RESULT IN LV PUMP FAILURE --

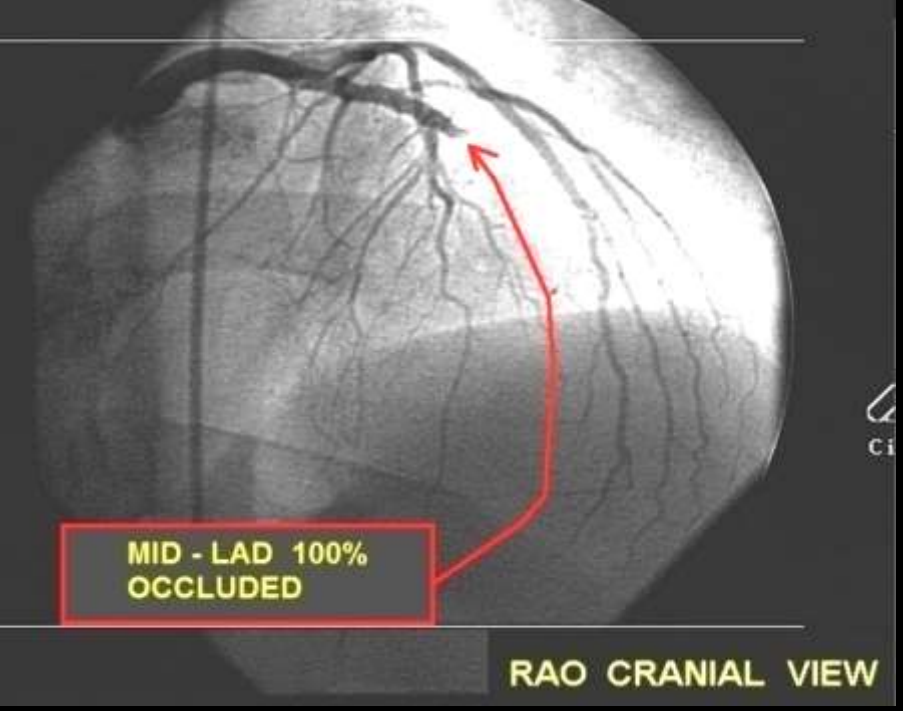
- CARDIOGENIC SHOCK
- PULMONARY EDEMA

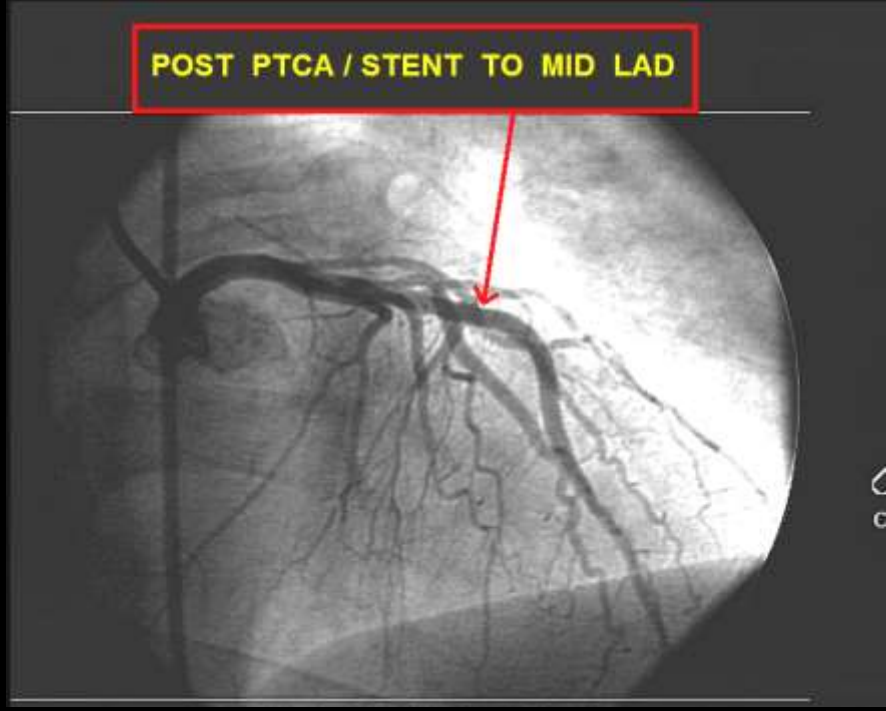

400 mg
Dopamine
(1600 mcg/mL)
Dopamine Hydrochloride
and 5% Dextrose Injection USP
250 mL
Baxter
500 mg Total
DOBUTamine
Hydrochloride in
5% Dextrose
250 mL
Baxter

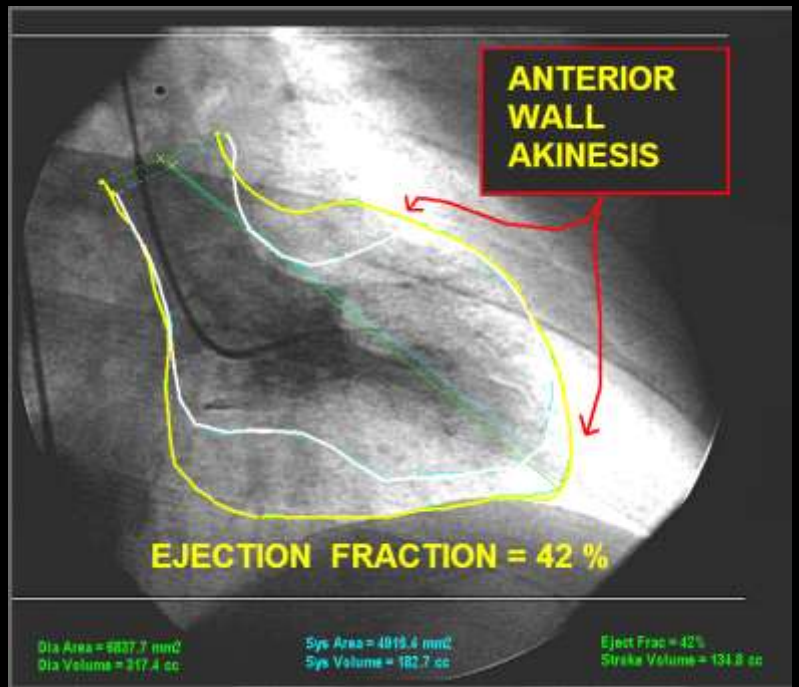
# LEFT ANTERIOR DESCENDING ARTERY ( LAD )

- ANTERIOR WALL OF LEFT VENTRICLE
- 35 - 45 % OF LEFT VENTRICLE MUSCLE MASS
- SEPTUM, ANTERIOR 2/3
- BUNDLE BRANCHES
- ANTERIOR-MEDIAL PAPILLARY MUSCLE

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

# 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.

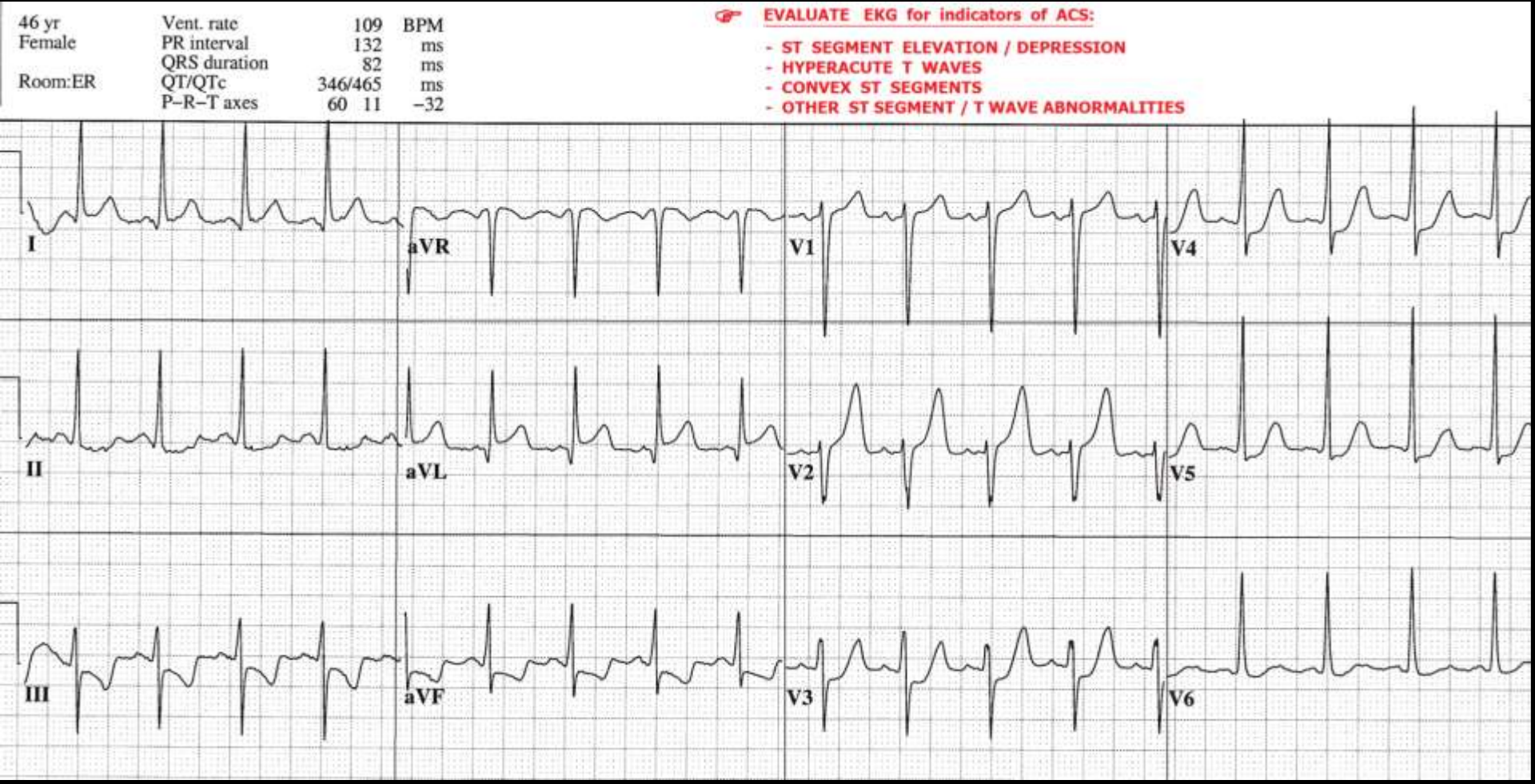
## RISK FACTOR PROFILE:

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

46 yr
Female

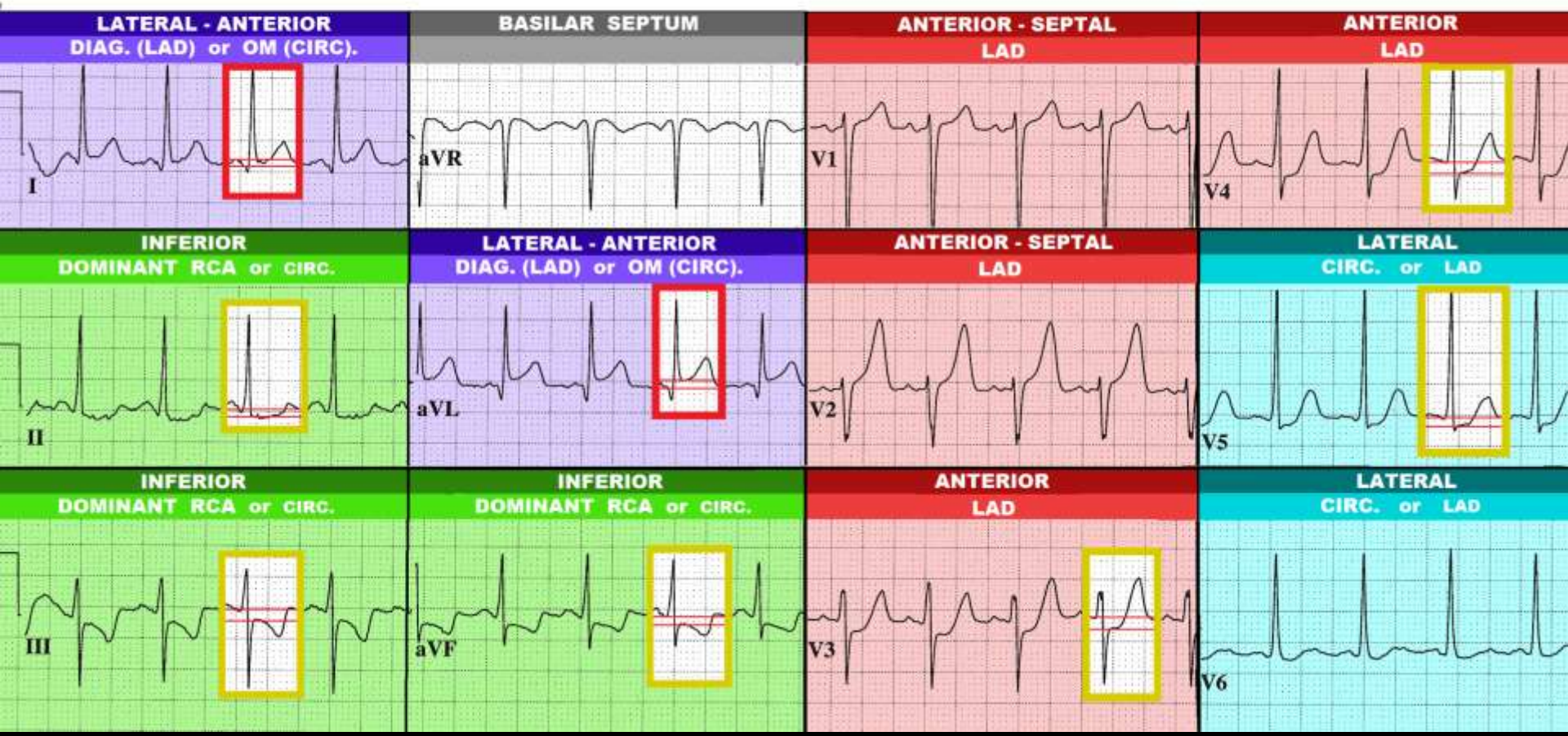
Room:ER

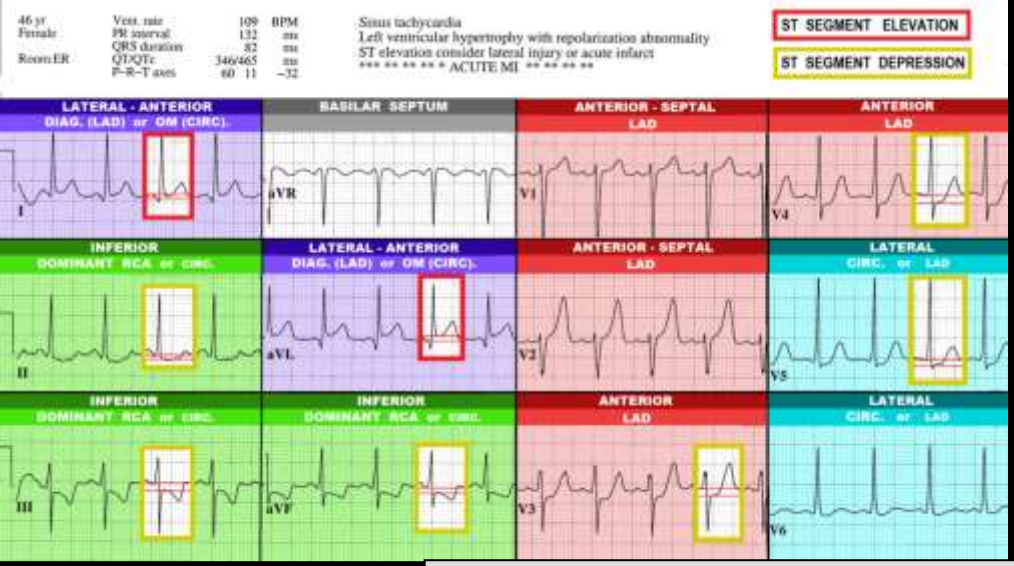
| | | |
|---|---|---|
| Vent. rate | 109 | BPM |
| PR interval | 132 | ms |
| QRS duration | 82 | ms |
| QT/QTc | 346/465 | ms |
| P–R–T axes | 60 11 | –32 |

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

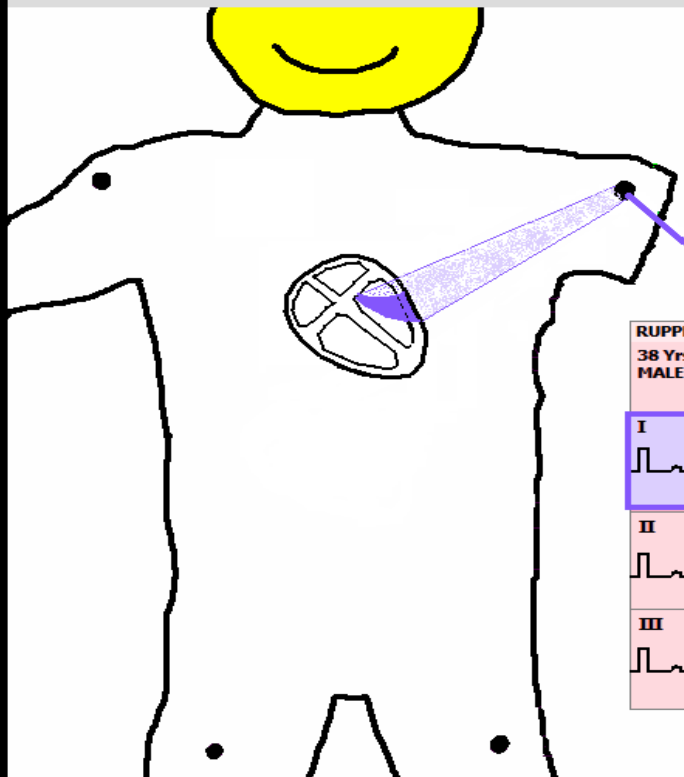
ST SEGMENT ELEVATION

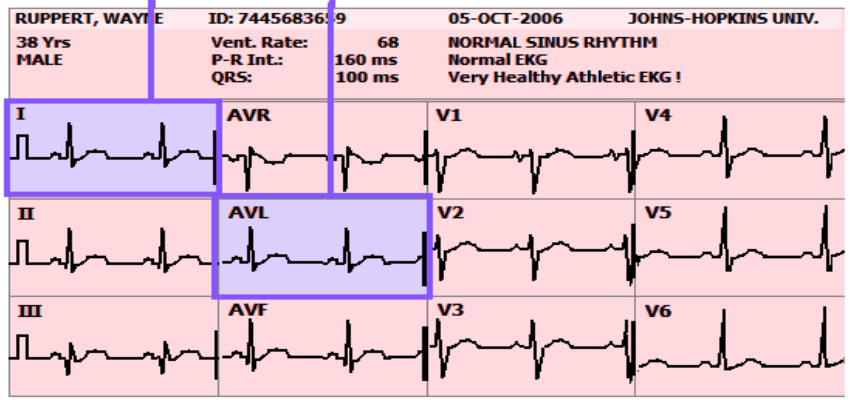
ST SEGMENT DEPRESSION



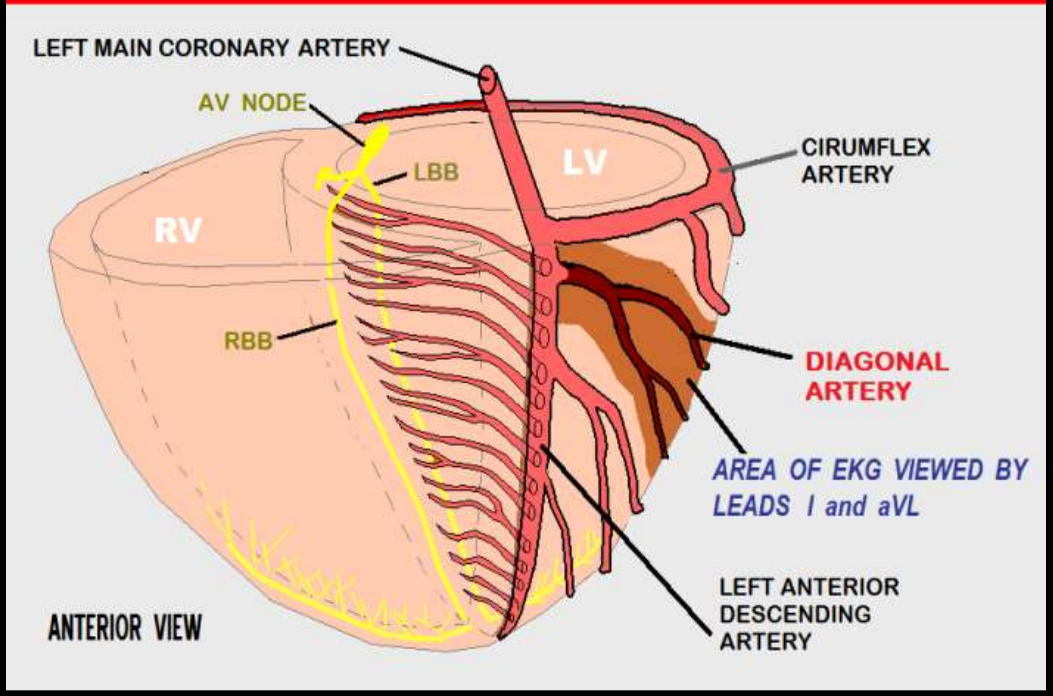



# LEADS I and aVL view the ANTERIOR-LATERAL JUNCTION

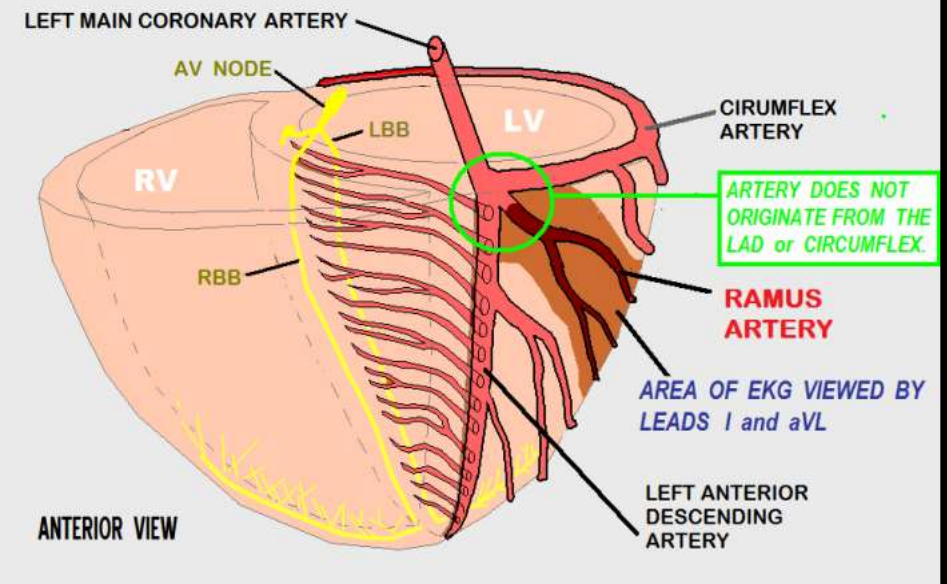




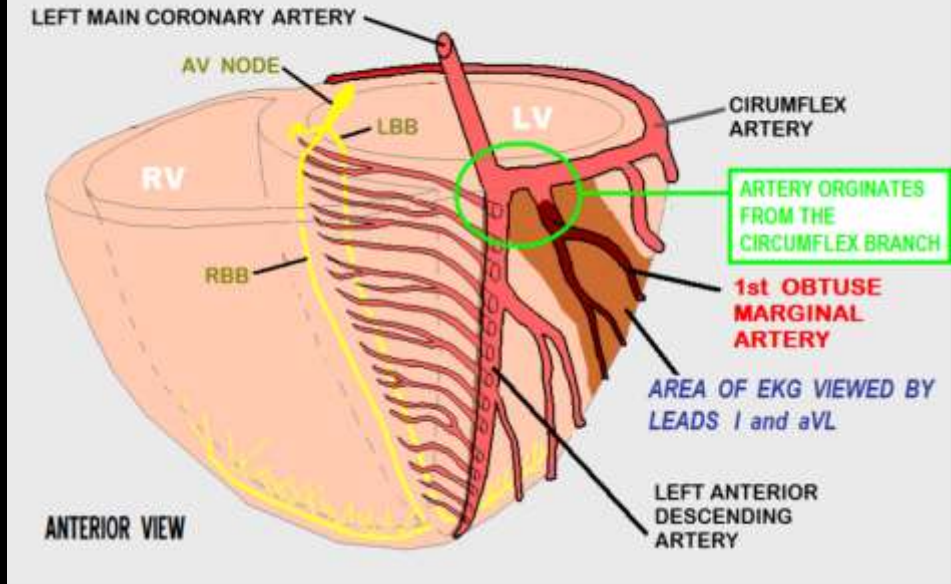
## OCCLUSION of DIAGONAL ARTERY



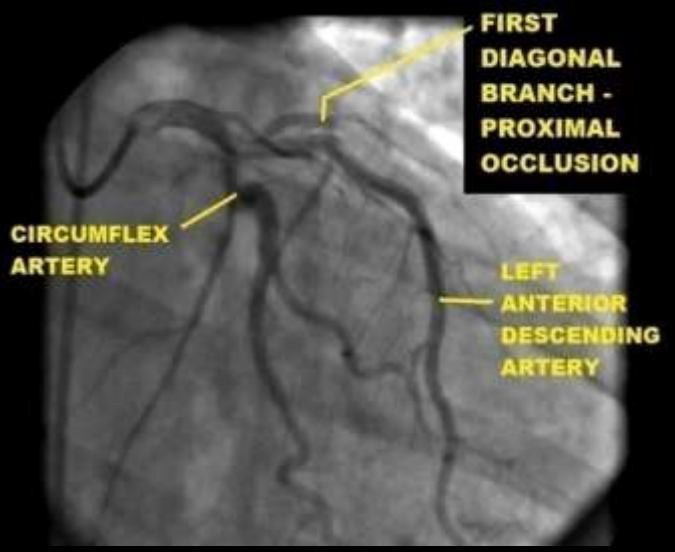

## OCCLUSION of RAMUS ARTERY

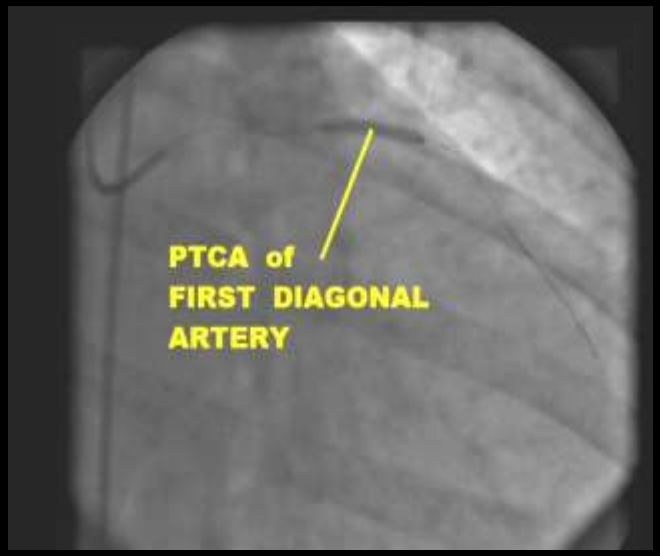


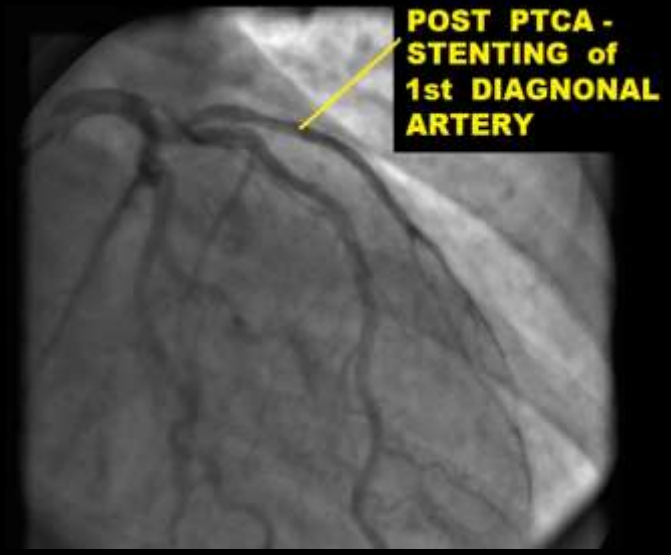

## OCCLUSION of OBTUSE MARGINAL ARTERY

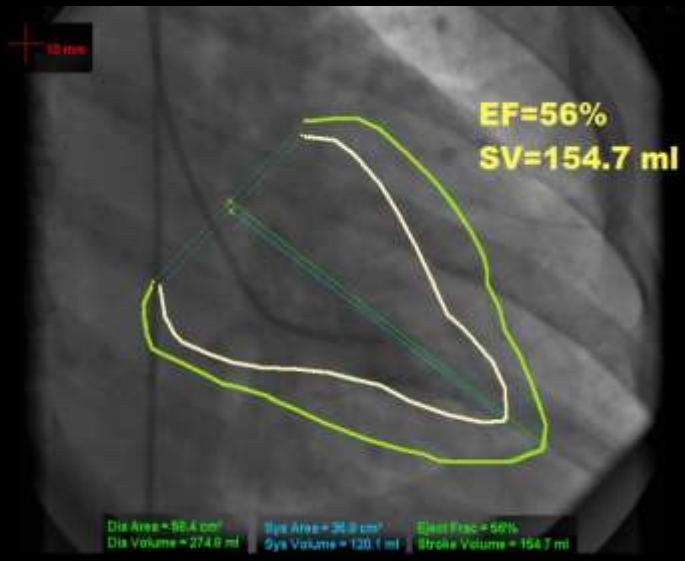


**CASE PROGRESSION: As the patient was being prepared for transport to the Cardiac Cath Lab, she experienced an episode of Ventricular Fibrillation.**



[redacted]

11111[redacted]11
Born 1/[redacted]1941 77 Years

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

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

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

--AXIS--
P 56
QRS -51
T -7

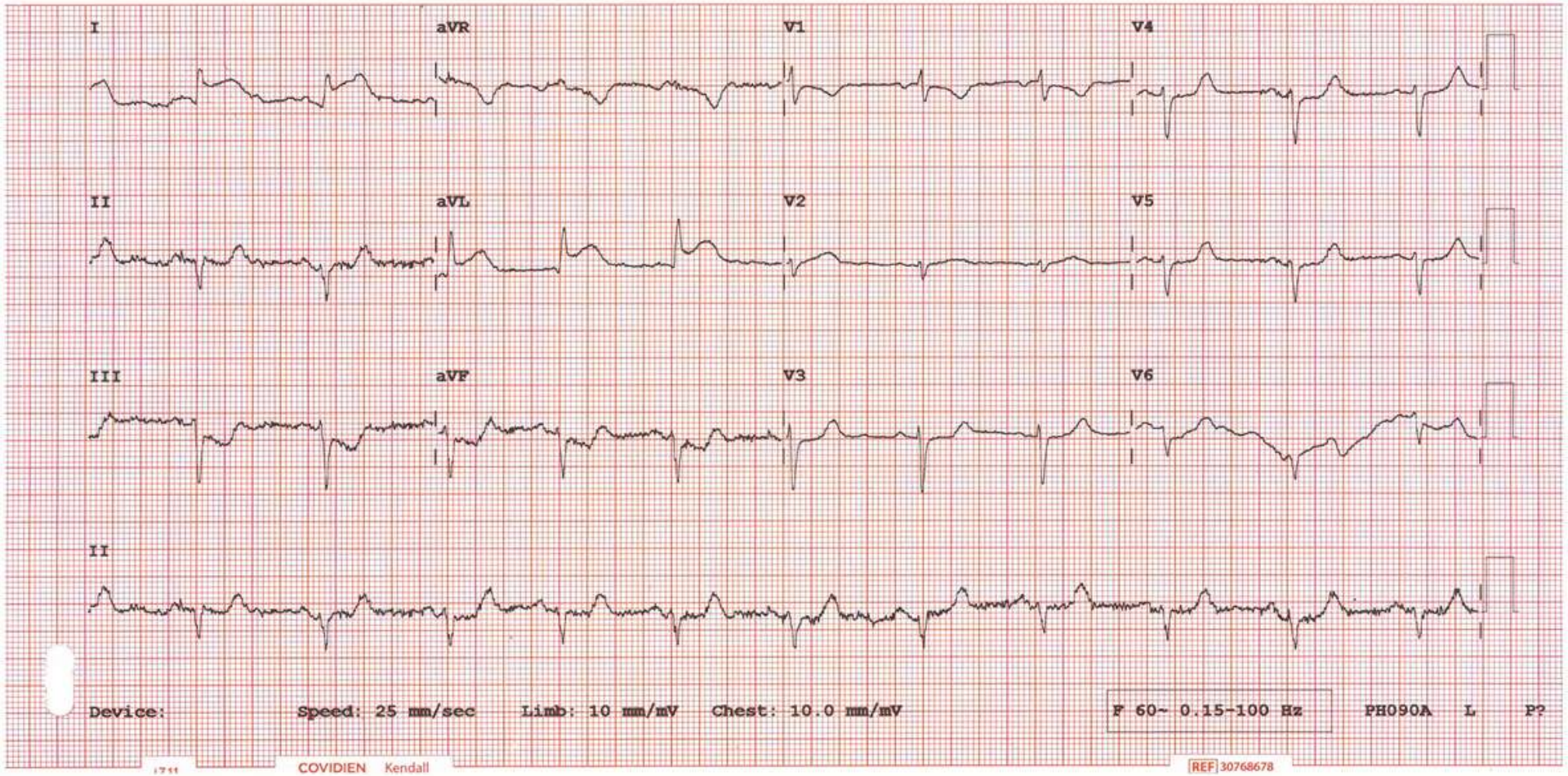
Room: er11

Physician [redacted]
Date
Time 1331
STEMI YES NO

- ABNORMAL ECG -

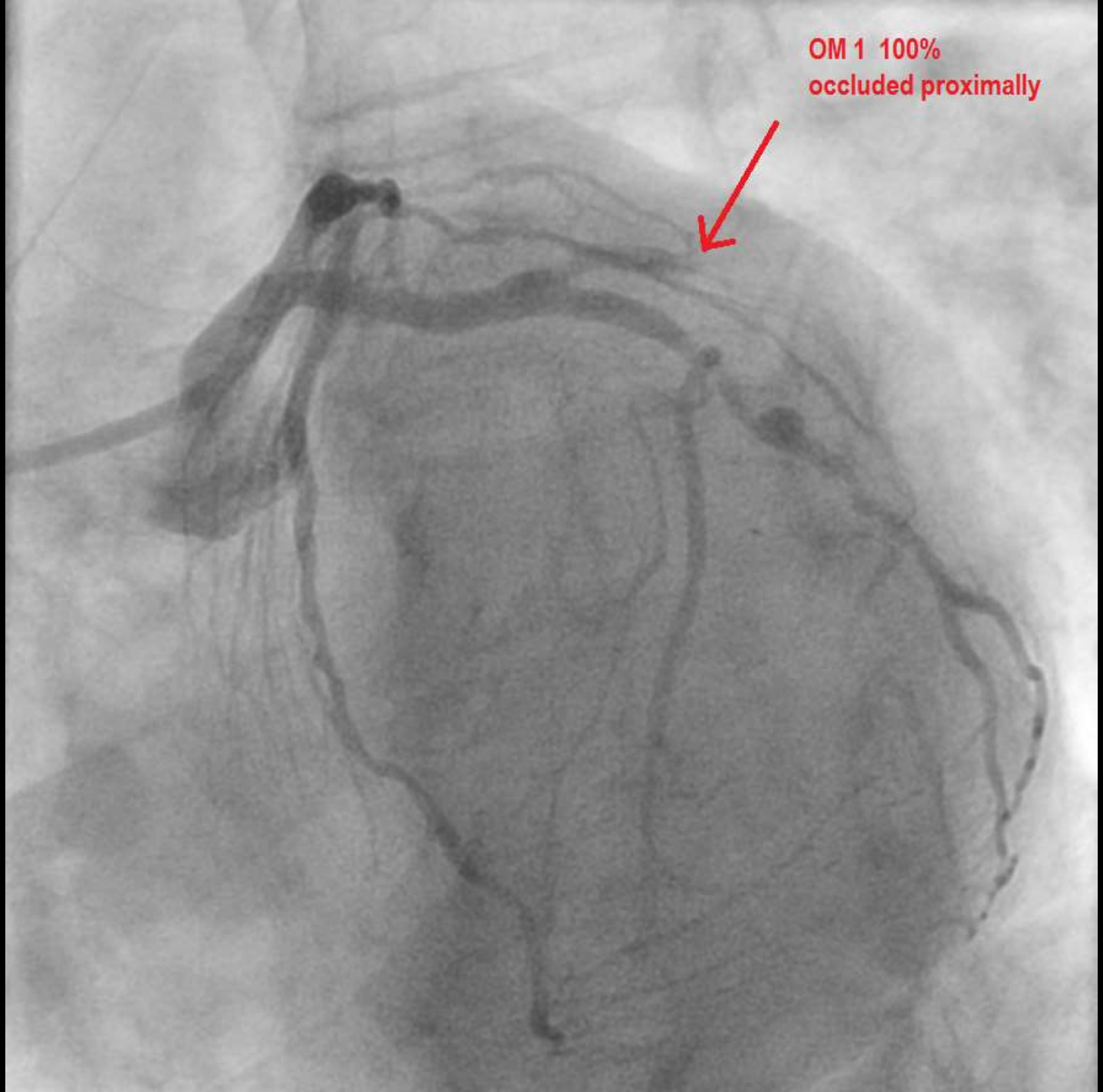
12 Lead; Standard Placement

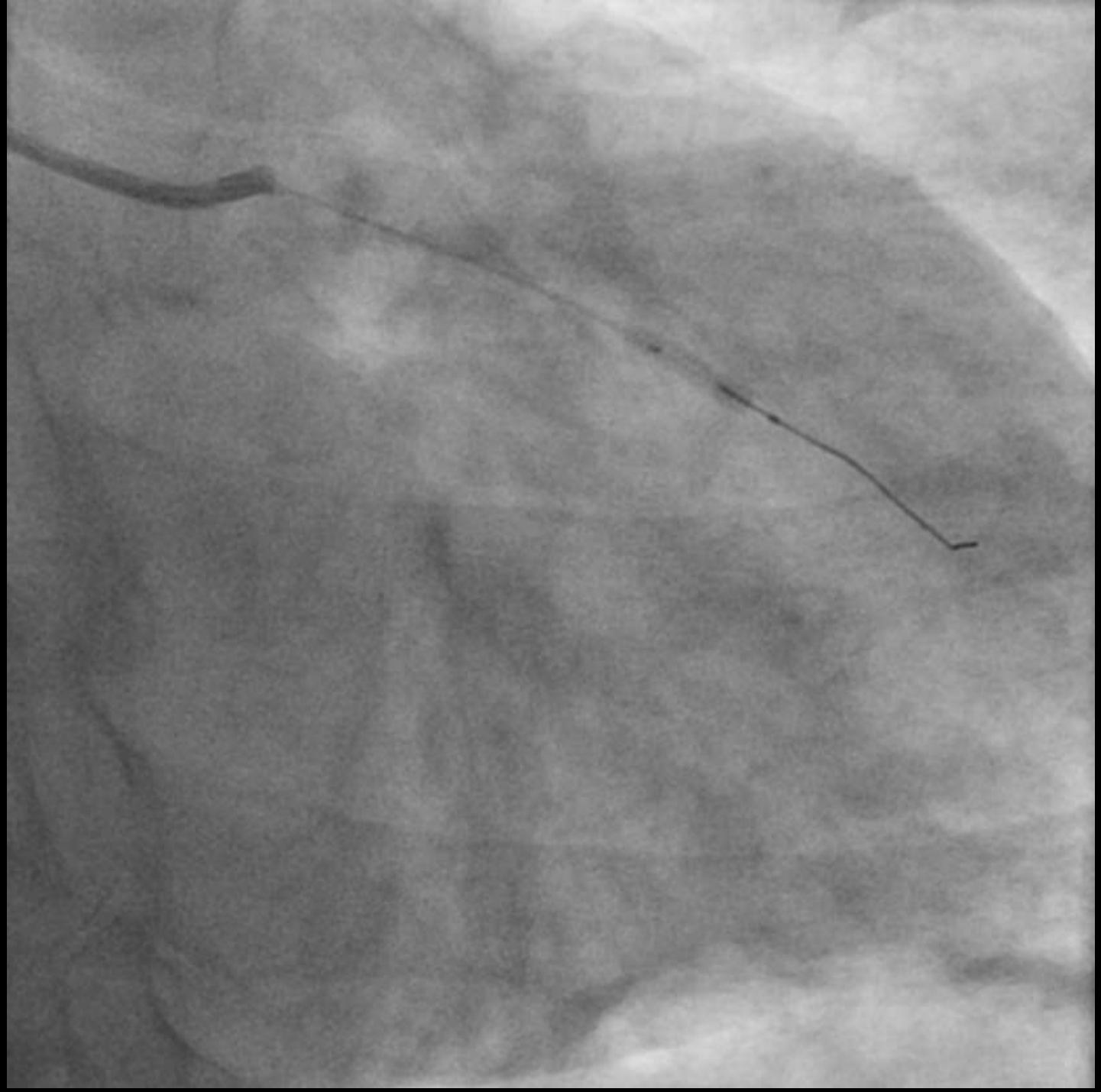
Unconfirmed Diagnosis

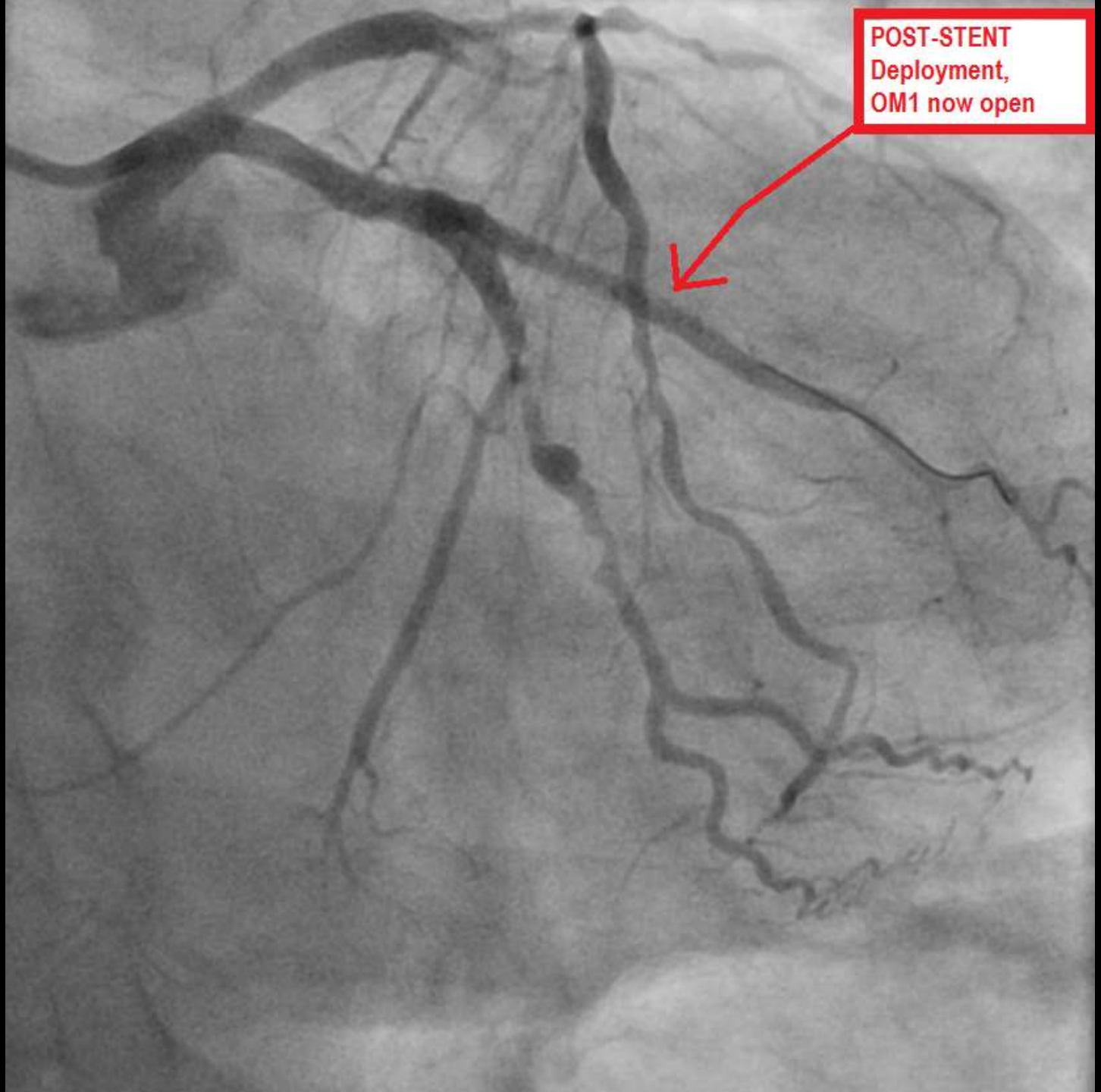



Device: Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10.0 mm/mV F 60~ 0.15-100 Hz PH090A L P?

COVIDIEN Kendall REF 30768678


OM 1 100%
occluded proximally




POST-STENT
Deployment,
OM1 now open

# CASE STUDY 3: STEMI

**CHIEF COMPLAINT and SIGNIFICANT HISTORY:**

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

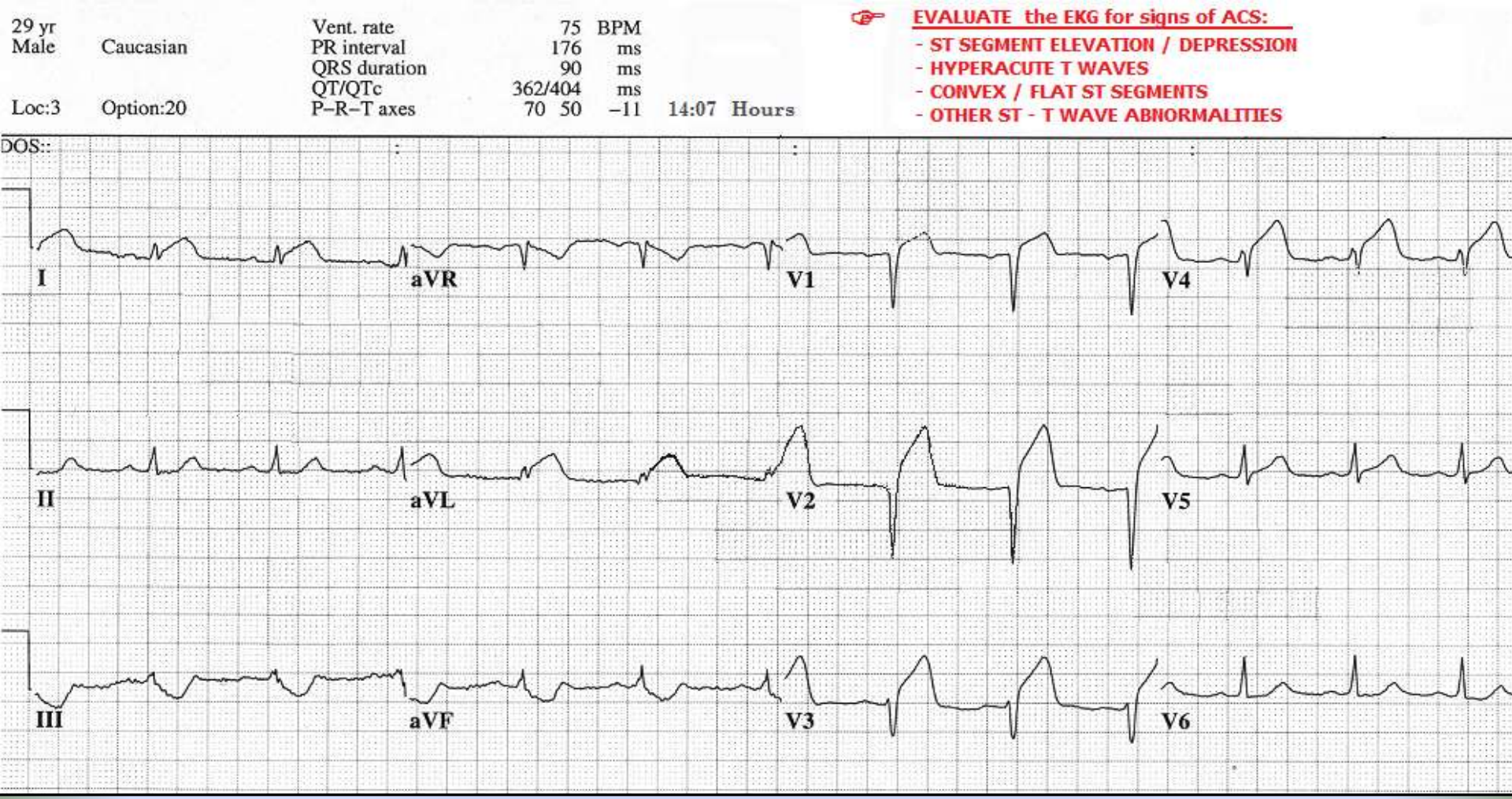
**RISK FACTOR PROFILE:**

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

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

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

**LABS:** INITIAL CARDIAC MARKERS - NEGATIVE


29 yr
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
DOS::
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

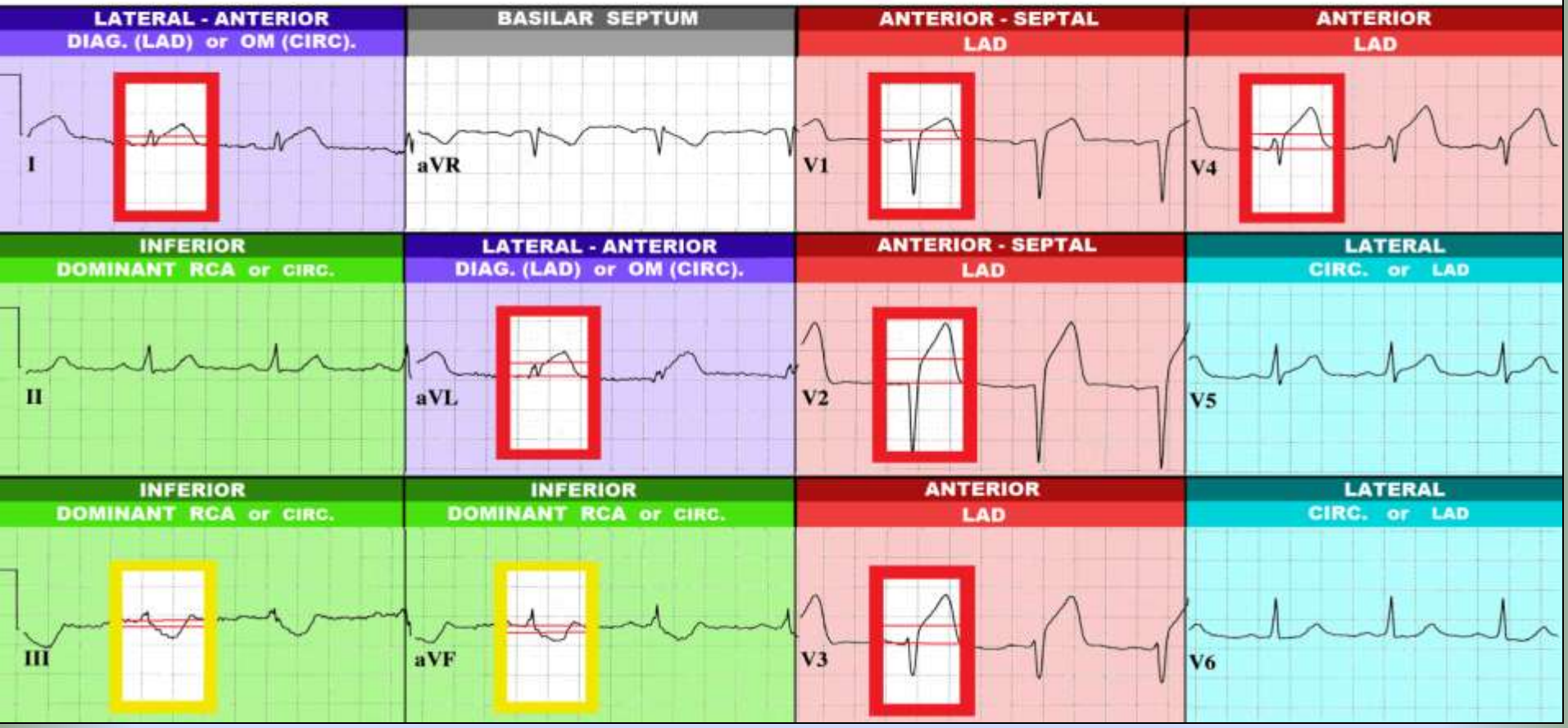
29 yr
Male
Caucasian

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

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

ST SEGMENT ELEVATION

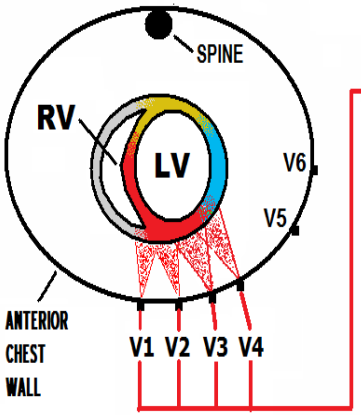
ST SEGMENT DEPRESSION



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

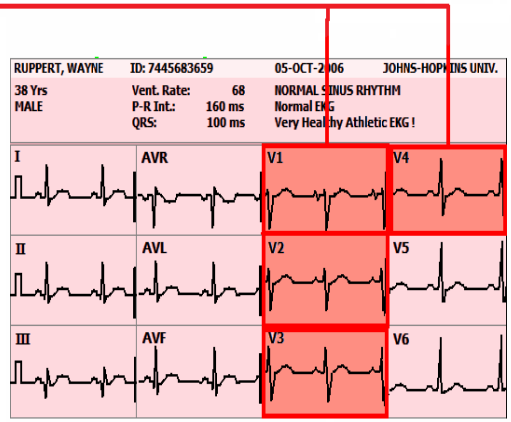
# V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL

## of the LEFT VENTRICLE

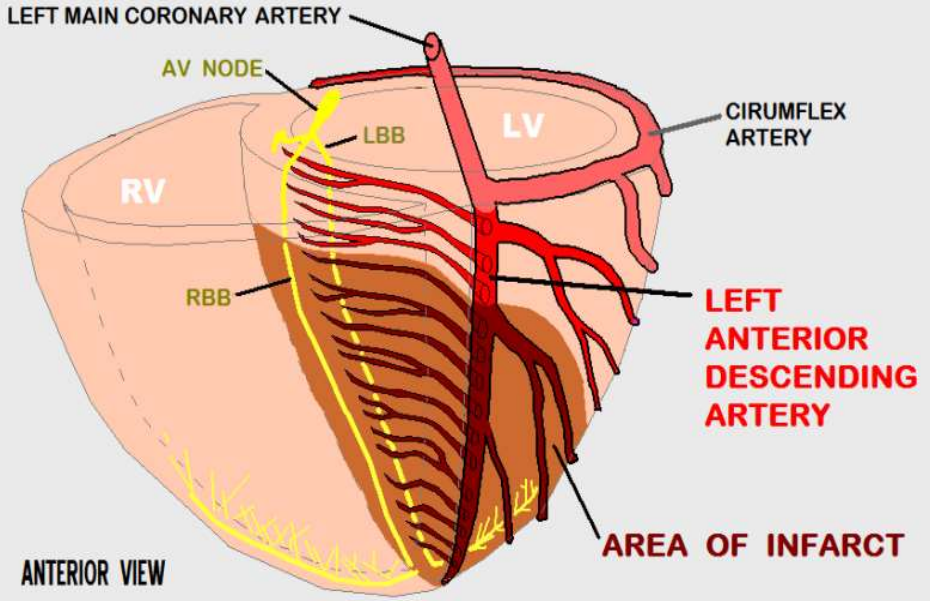



V1, V2 - ANTERIOR / SEPTAL

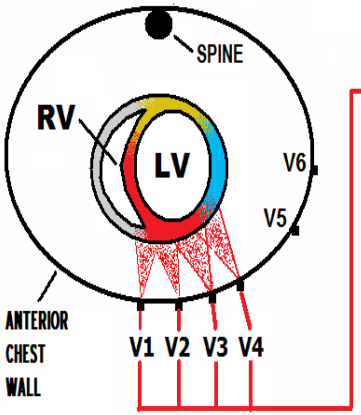
V3, V4 - ANTERIOR



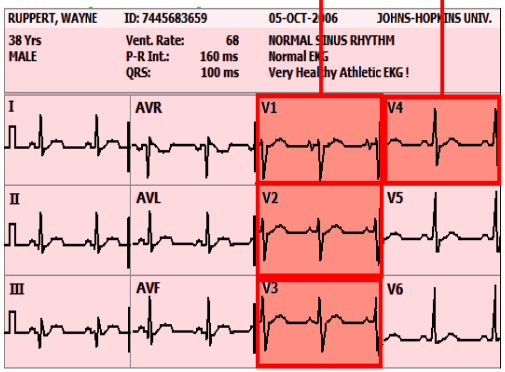

# OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY



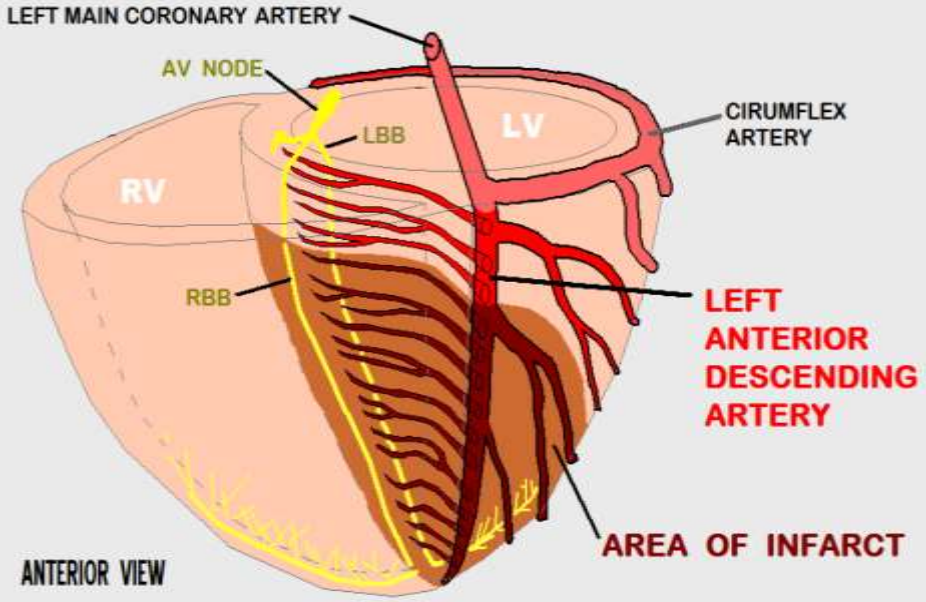
# V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL
## of the LEFT VENTRICLE



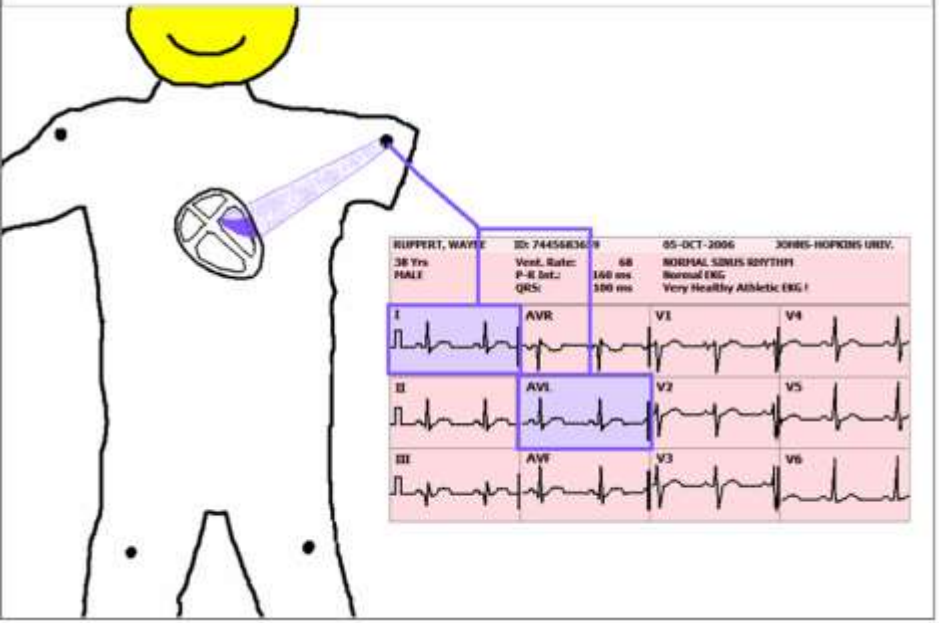

V1, V2 - ANTERIOR / SEPTAL
V3, V4 - ANTERIOR



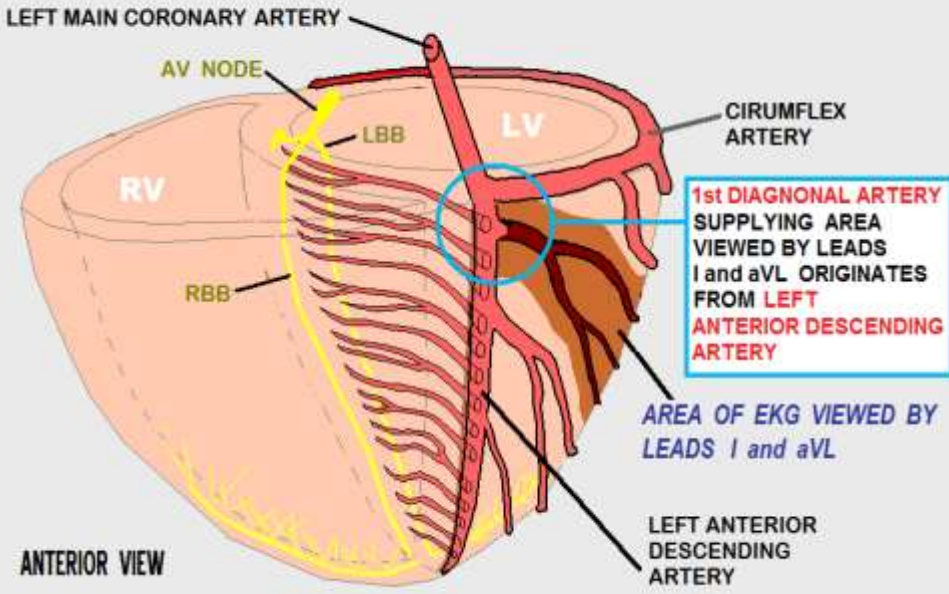

# OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY

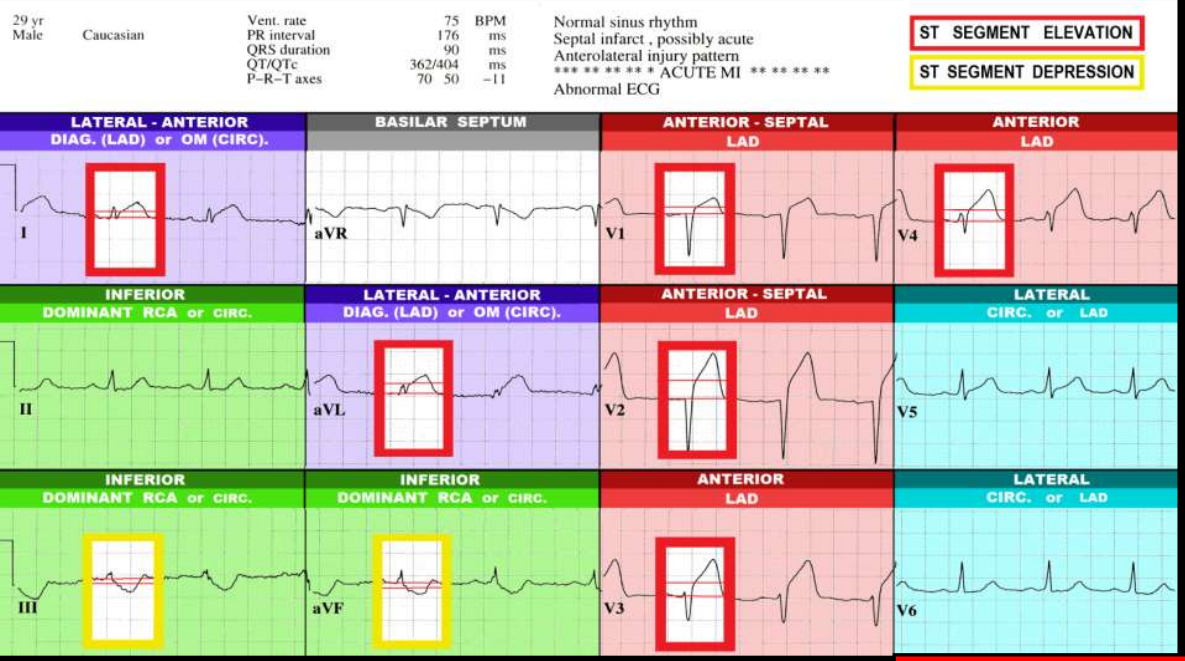


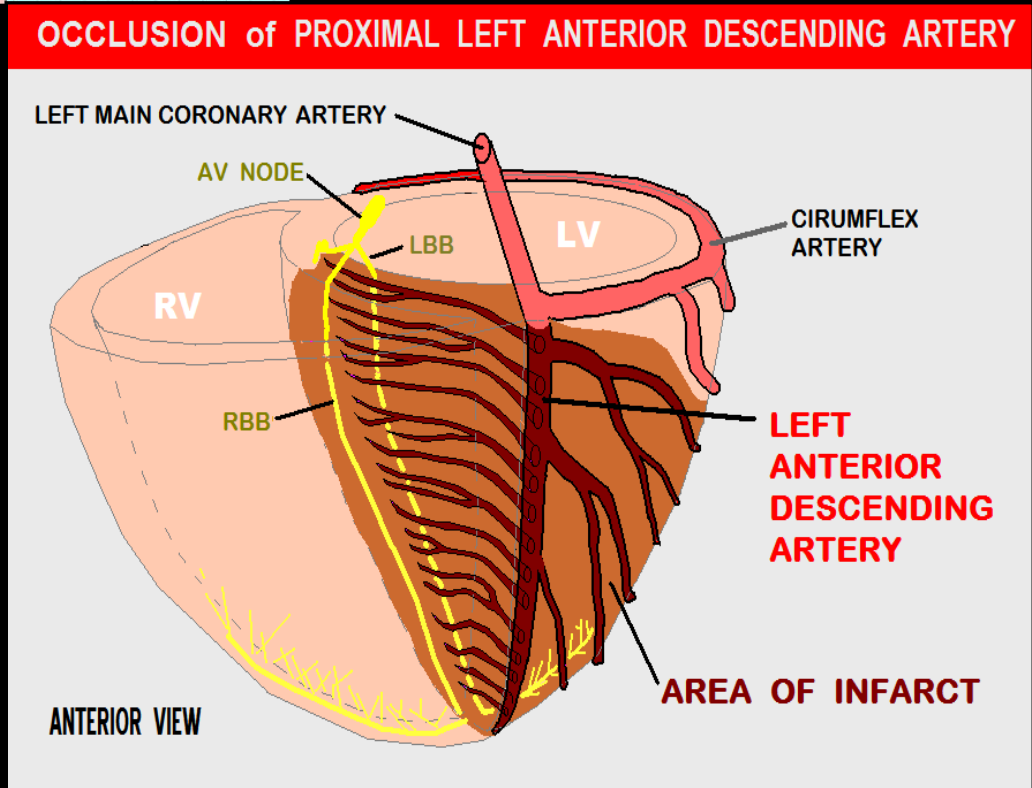

# Leads I & AVL view the ANTERIOR-LATERAL JUNCTION



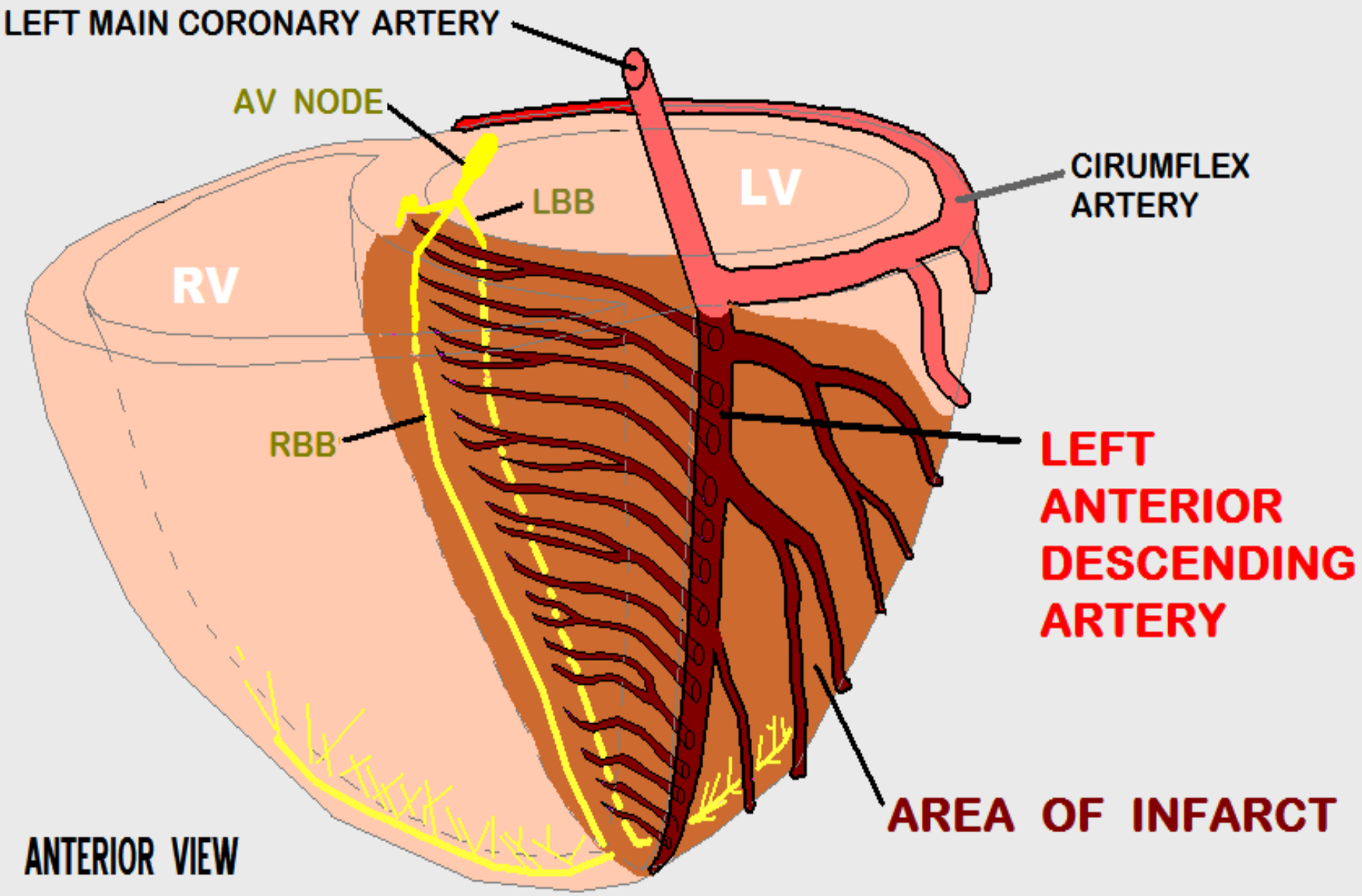

# OCCLUSION of DIAGONAL ARTERY




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


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

# OCCLUSION of PROXIMAL LEFT ANTERIOR DESCENDING ARTERY

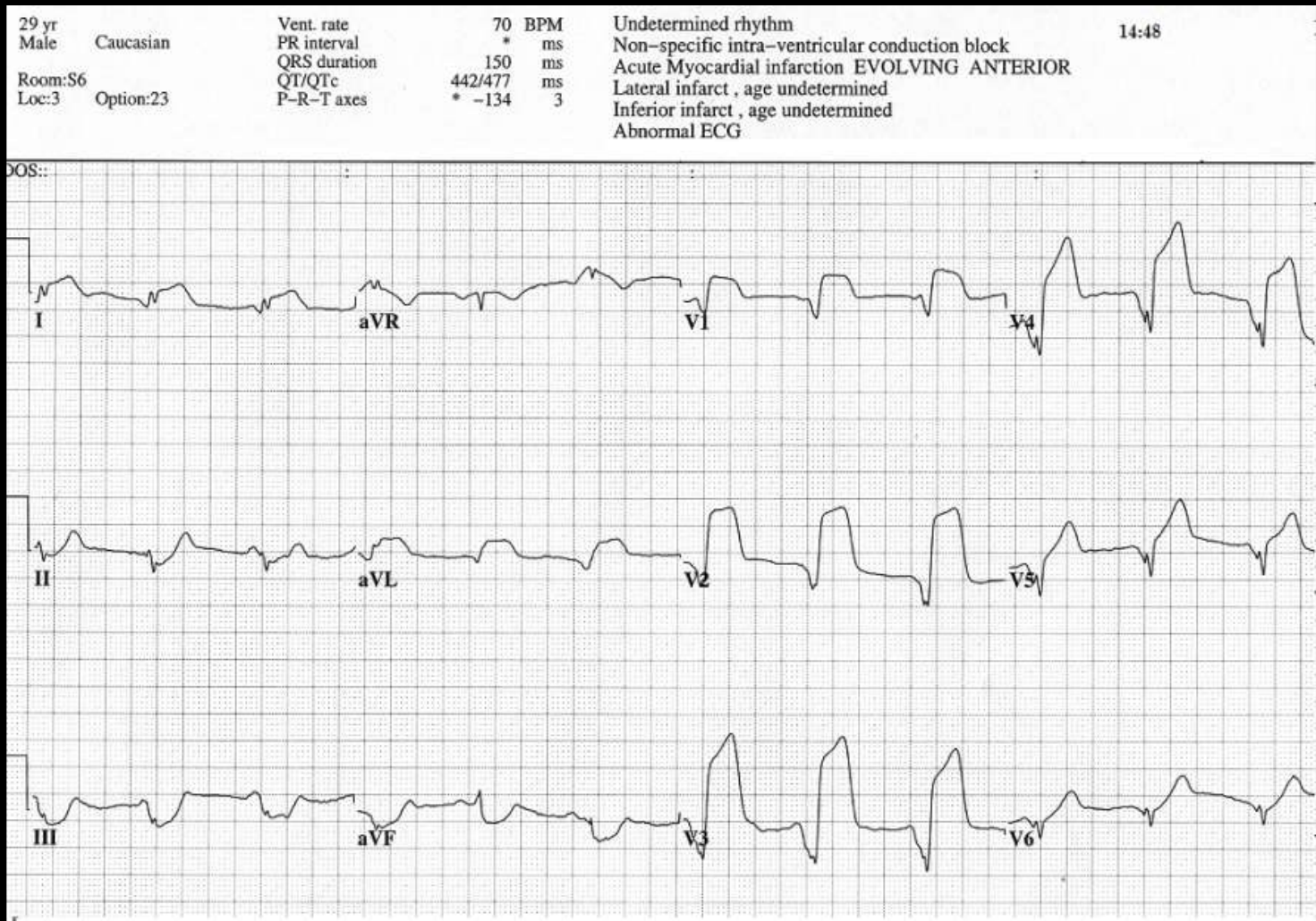


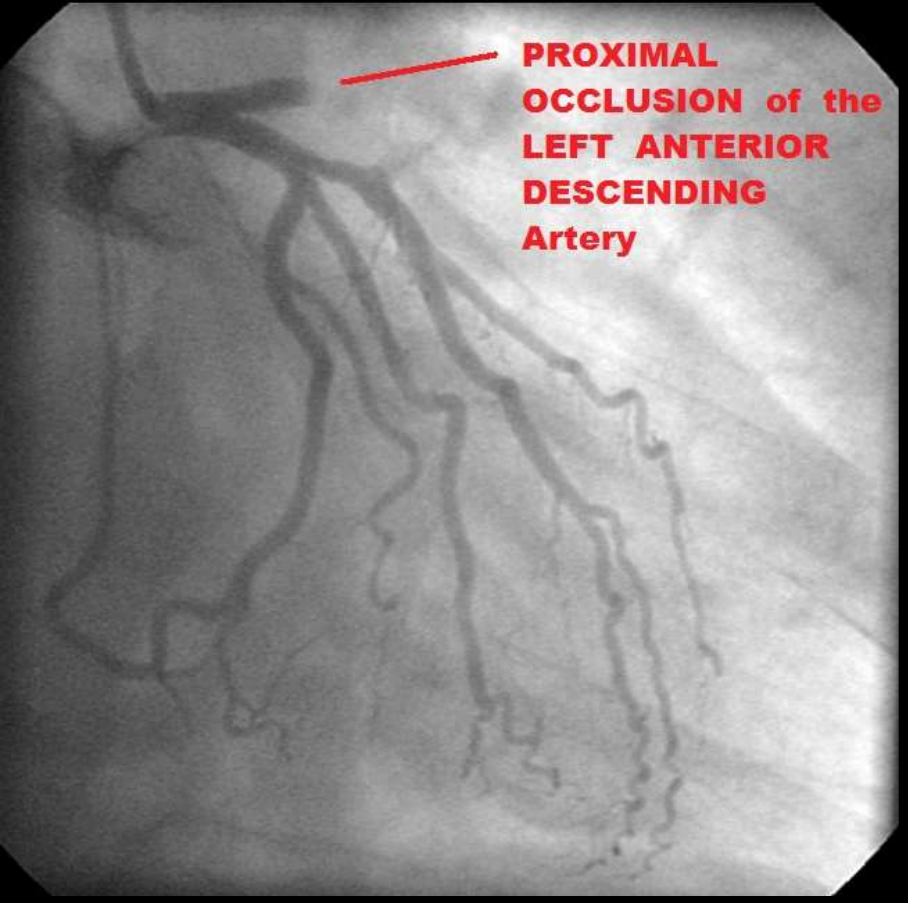
| ANTICIPATED COMPLICATIONS of ANTERIOR-SEPTAL WALL STEMI & POSSIBLE INDICATED INTERVENTIONS: | |
|---|---|
| - CARDIAC ARREST | BCLS / ACLS |
| - CARDIAC DYSRHYTHMIAS (VT / VF) | ACLS (antiarrhythmics) |
| - PUMP FAILURE with CARDIOGENIC SHOCK | INOTROPE THERAPY:<br>-DOPAMINE / DOBUTAMINE / LEVOPHED<br>- INTRA-AORTIC BALLOON PUMP<br>(use caution with fluid challenges due to PULMONARY EDEMA) |
| - PULMONARY EDEMA | - CPAP<br>- ET INTUBATION<br>(use caution with dieuretics due to pump failure and hypotension) |
| - 3rd DEGREE HEART BLOCK - NOT RESPONSIVE TO ATROPINE | TRANSCUTANEOUS or TRANSVENOUS PACING |

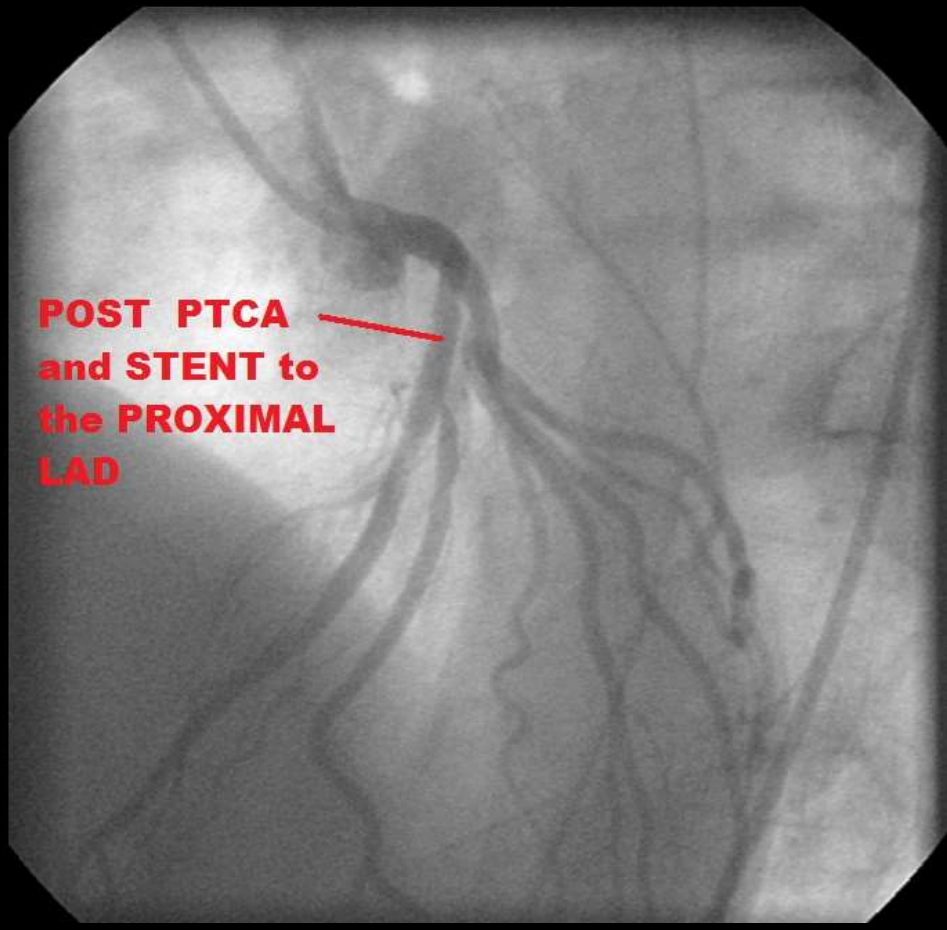
WHILE AWAITING THE CATH LAB TEAM, THE PATIENT BEGAN VOMITING. SKIN BECAME ASHEN & DIAPHORETIC. REPEAT BP = 50/30.

WHILE AWAITING THE CATH LAB 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

**The patient was discharged a few days later, with a referral to Cardiac Rehab.**

# EVOLVING STEMI:

-ST SEGMENTS DROP

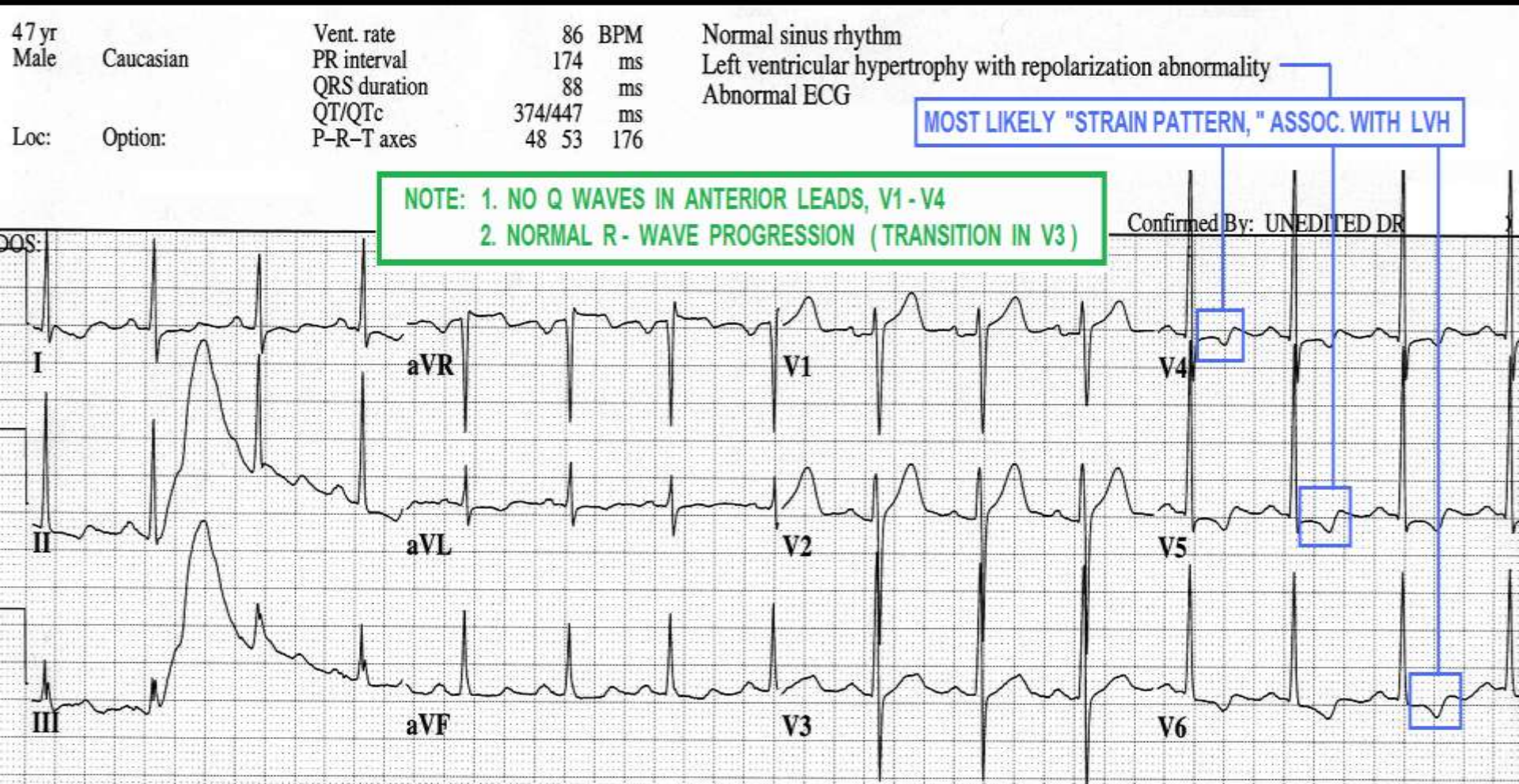
-Q WAVES FORM

-R WAVE PROGRESSION CHANGES IN PRECORDIAL LEADS.

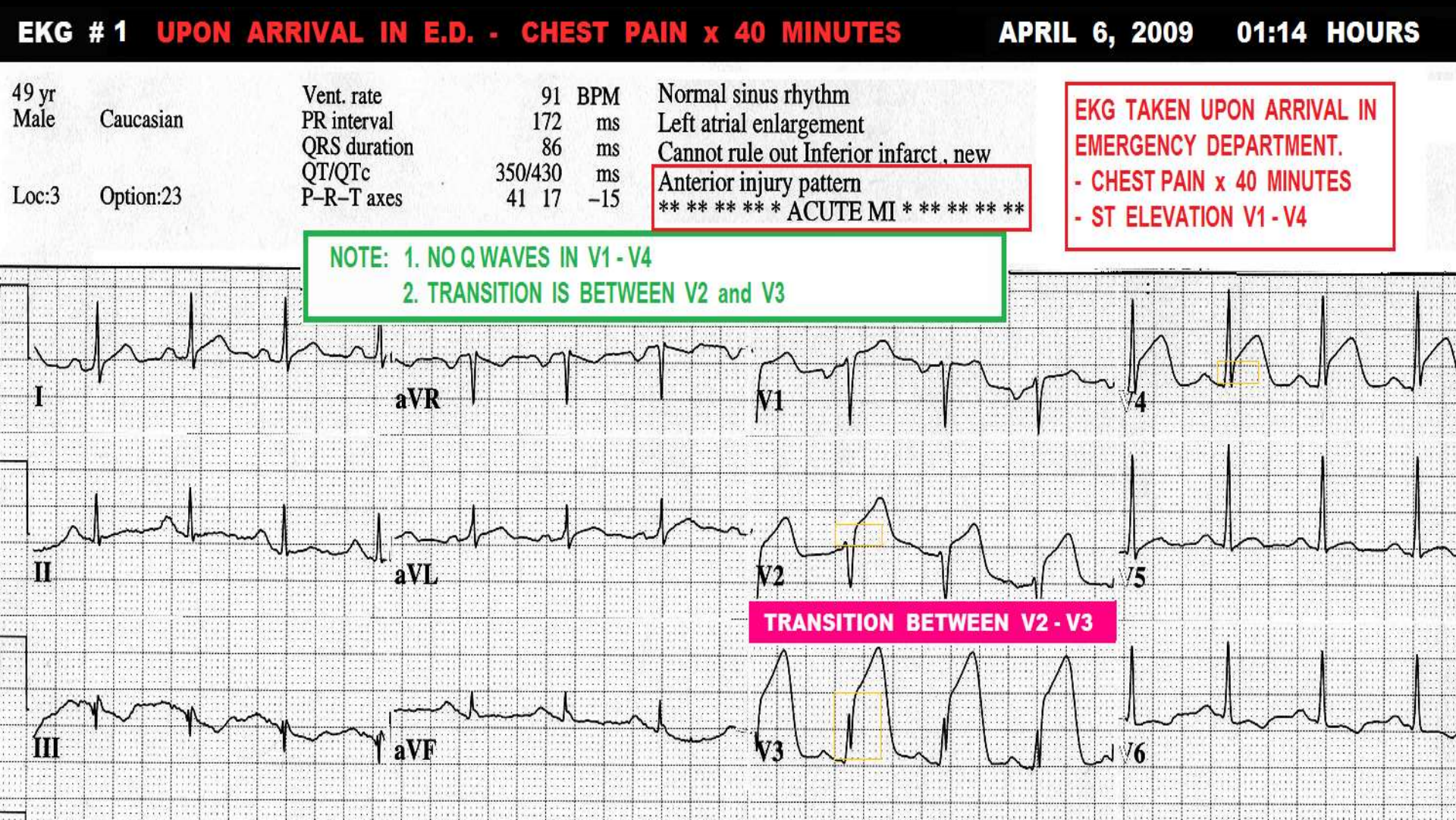
## Q WAVE RULES - SUMMARY:

- Q WAVES SHOULD BE LESS THAN .40 WIDE ( 1 mm )
- Q WAVES SHOULD BE LESS THAN 1/3 THE HEIGHT OF THE R WAVE
- Q WAVES CAN BE ANY SIZE IN LEADS III and AVR
- THERE SHOULD BE NO Q WAVES IN LEADS V1, V2, or V3

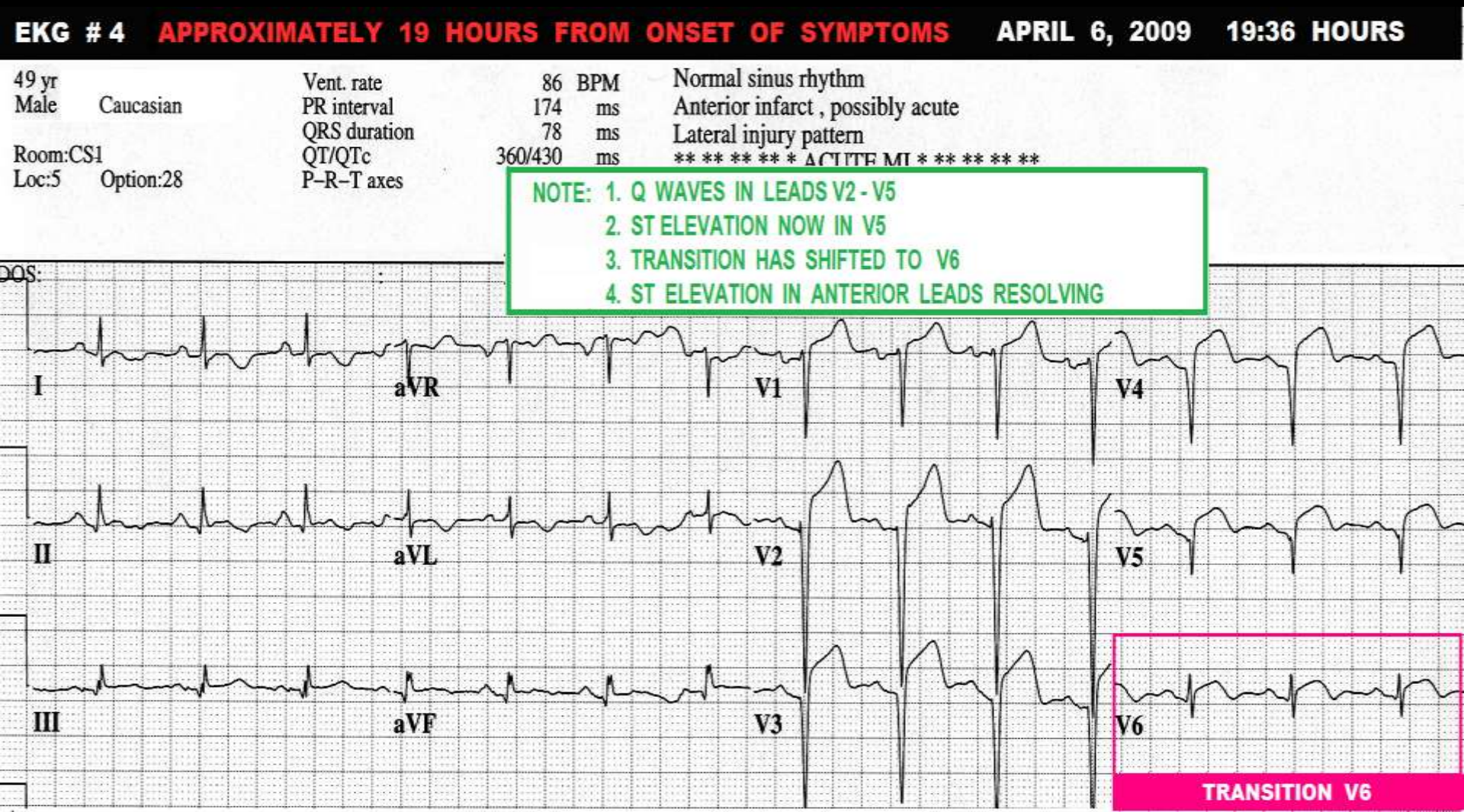
# PRE-INFARCTION ECG



# ACUTE ANTERIOR WALL STEMI

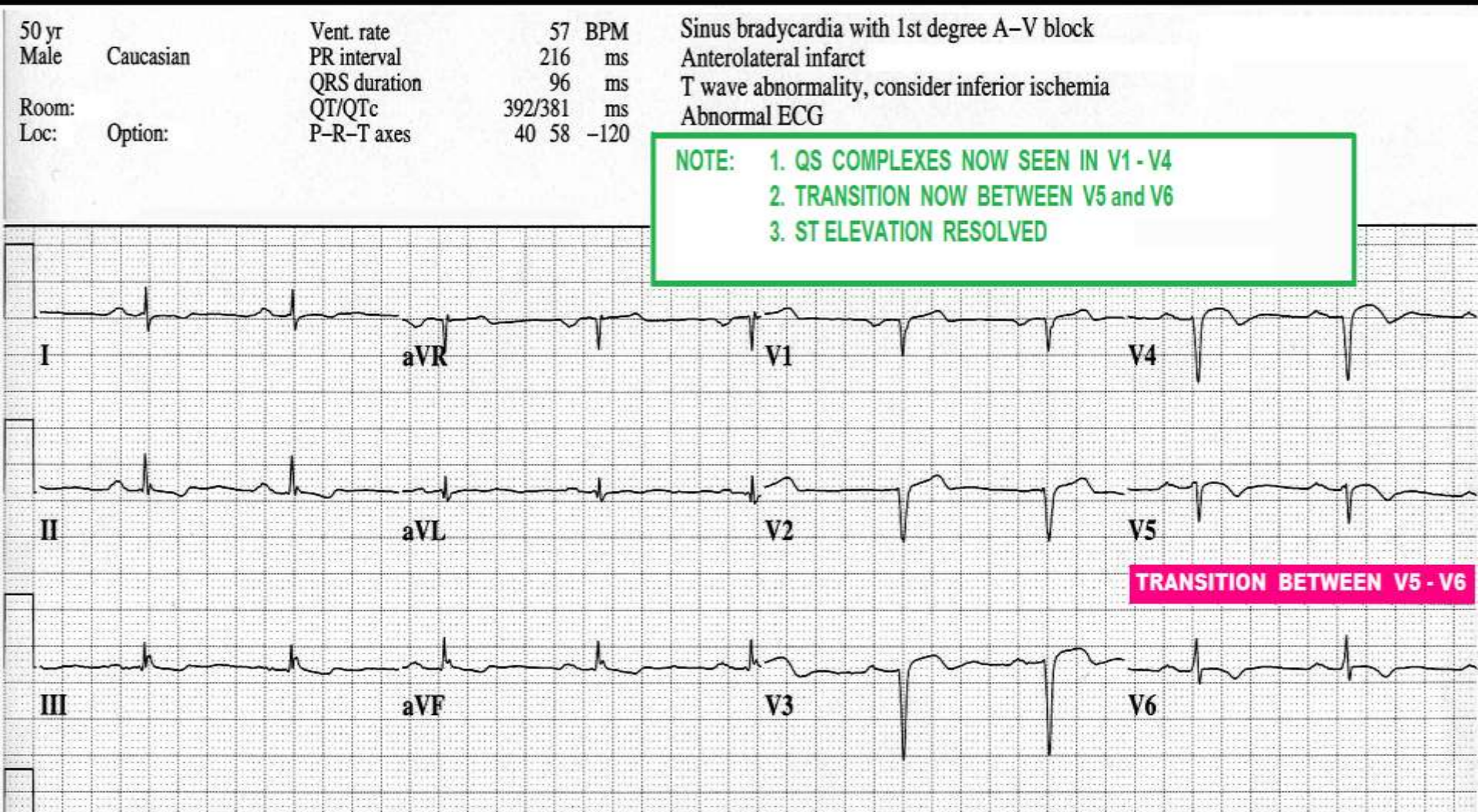


# EVOLVING ANTERIOR WALL STEMI



# FULLY EVOLVED ANTERIOR WALL MI

**POST - INFARCTION EKG TAKEN 1 YEAR AFTER ANTERIOR WALL MI**



26–NOV–1999 02:03:00 ST. JOSEPH'S HOSPITAL–CCU ROUTINE RETRIEVAL

29 yr
Male

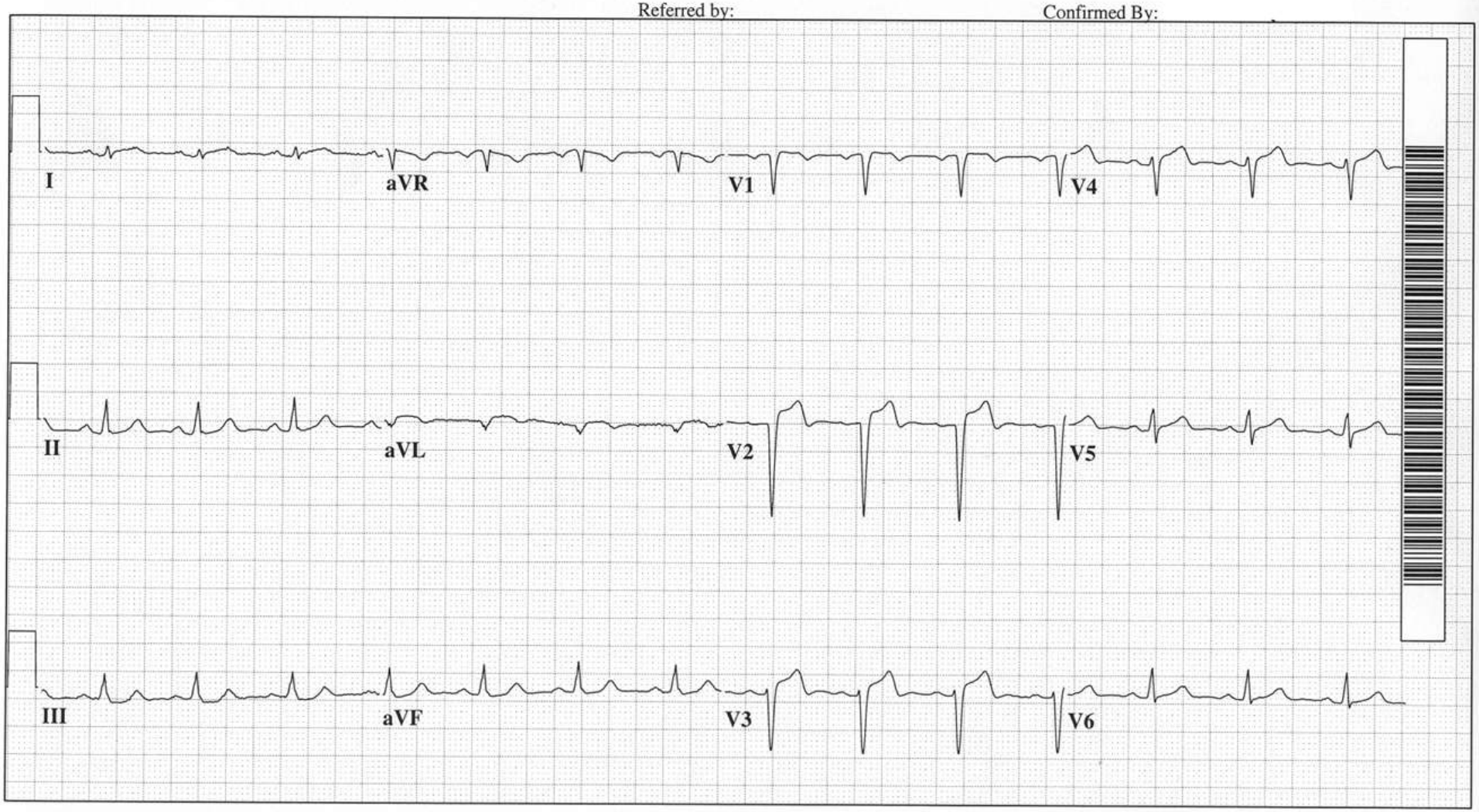
Loc:1 Option:1

| | |
|---|---|
| Vent. rate | 85 BPM |
| PR interval | 156 ms |
| QRS duration | 88 ms |
| QT/QTc | 340/404 ms |
| P–R–T axes | 60 79 49 |

**WHAT IS THE DIAGNOSIS BY EKG ?**

**EKG CLASS #WR03694519**

Referred by: Confirmed By:




25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 1

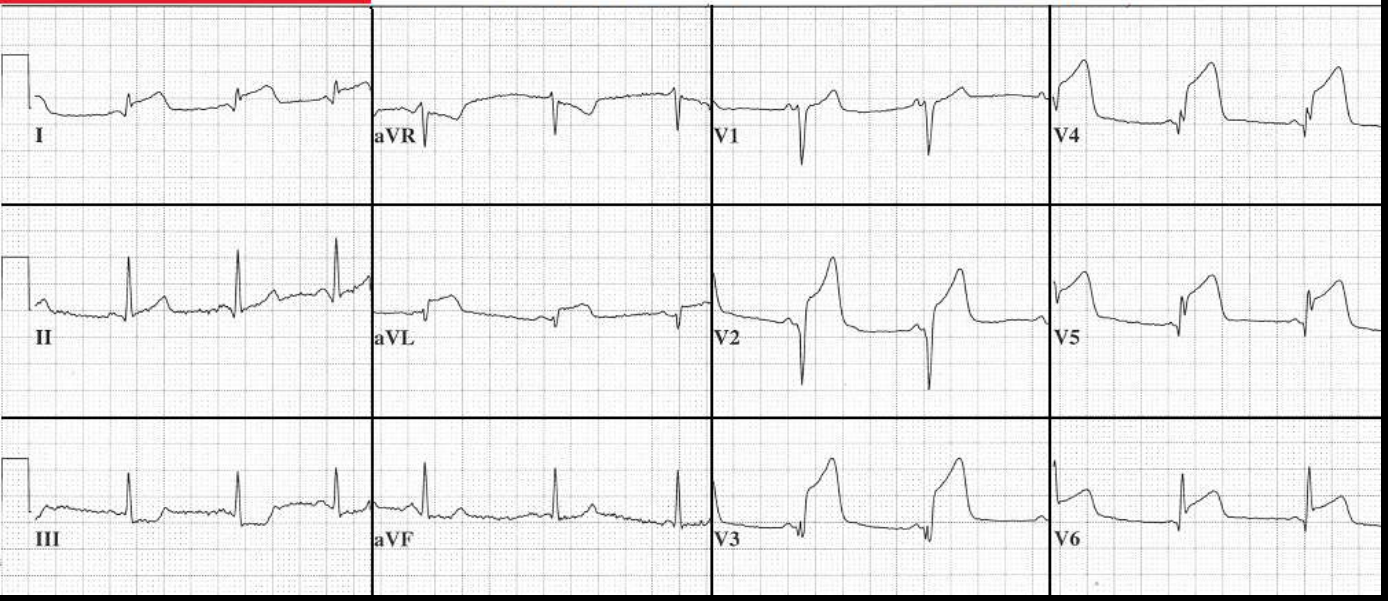
EID:13 EDT: 18:01 30–NOV–1999 ORDER:

## 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.

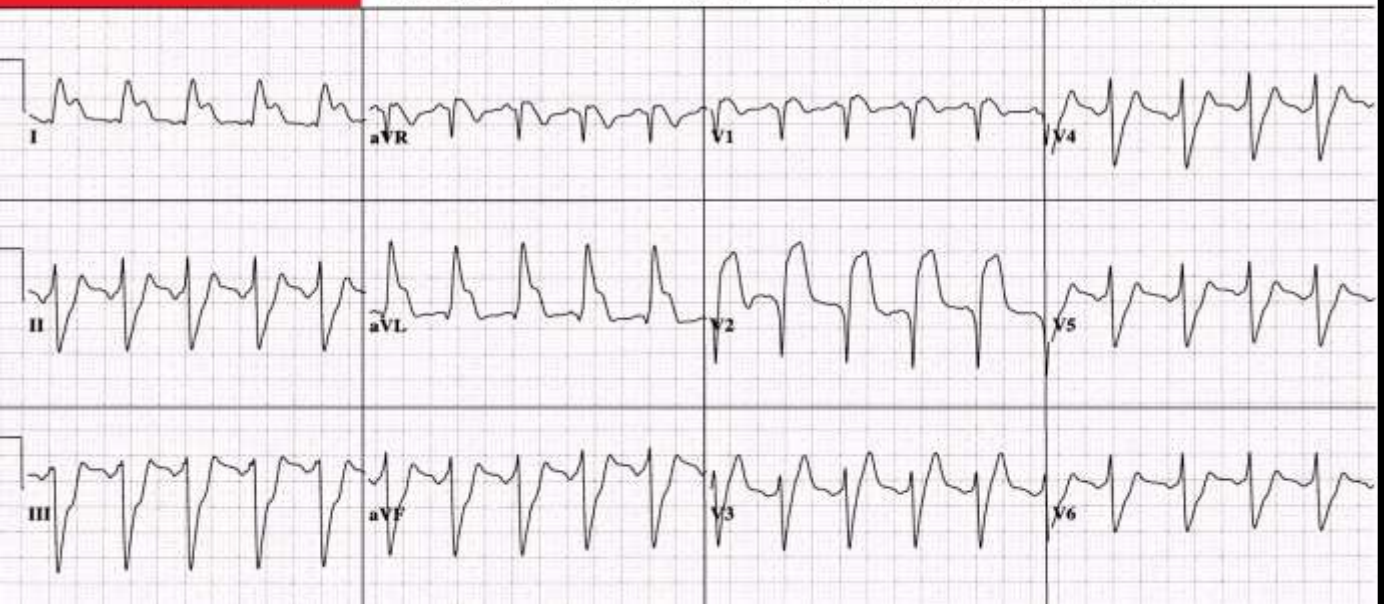
**PATIENT A:** 44 y/o MALE, CHEST PAIN x 1 HOUR,
BP: 78/46, P: 70, R: 28. CARDIAC MARKERS: NEGATIVE




WHO SHOULD GO TO THE CATH LAB FIRST ?

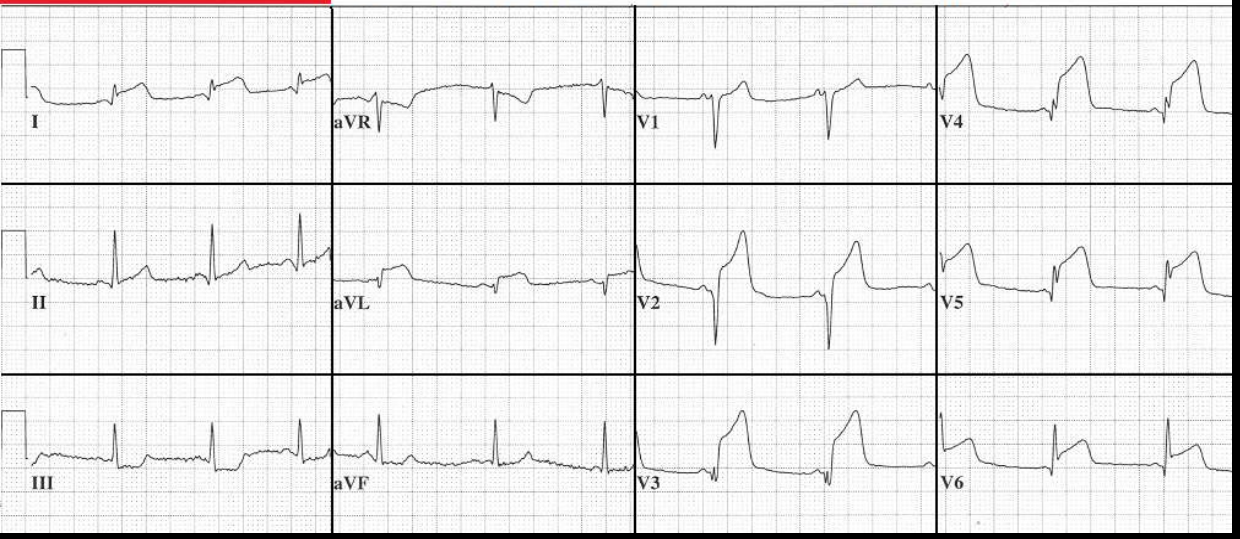
And . . . .

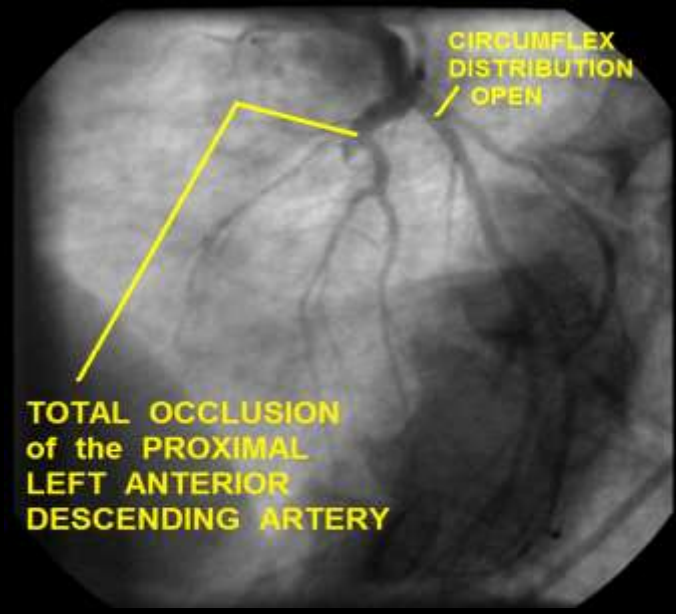
**PATIENT B:** 36 y/o MALE, CHEST PAIN x 1 HOUR,
BP: 80/48, P: 120, R: 28 CARDIAC MARKERS: NEGATIVE



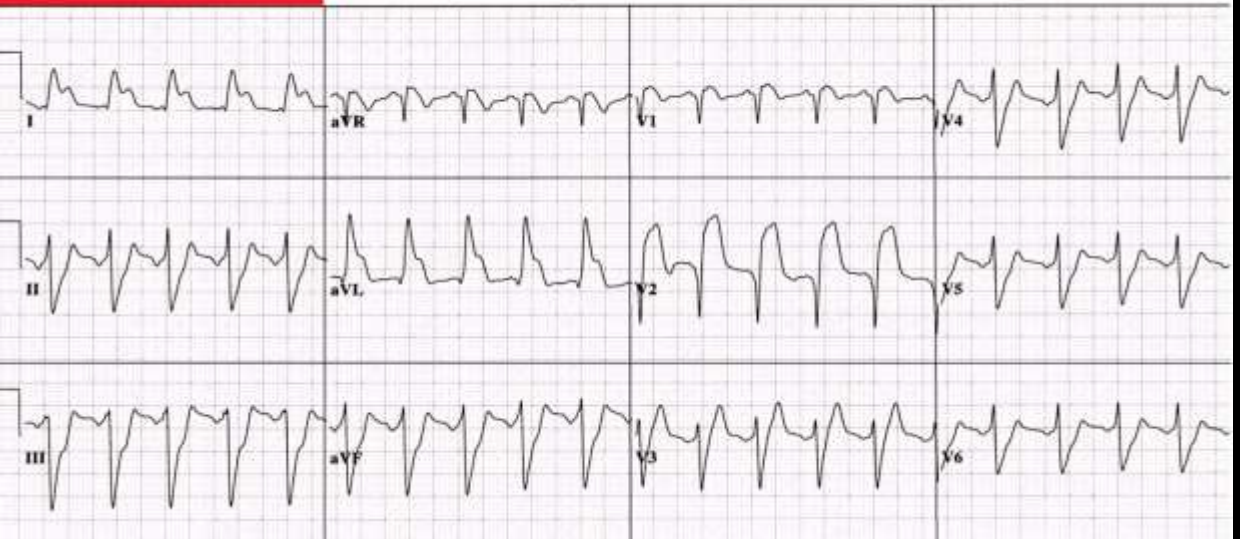

WHAT WOULD YOU DO WITH THE PATIENT WHO DID NOT GO TO THE CATH LAB ?

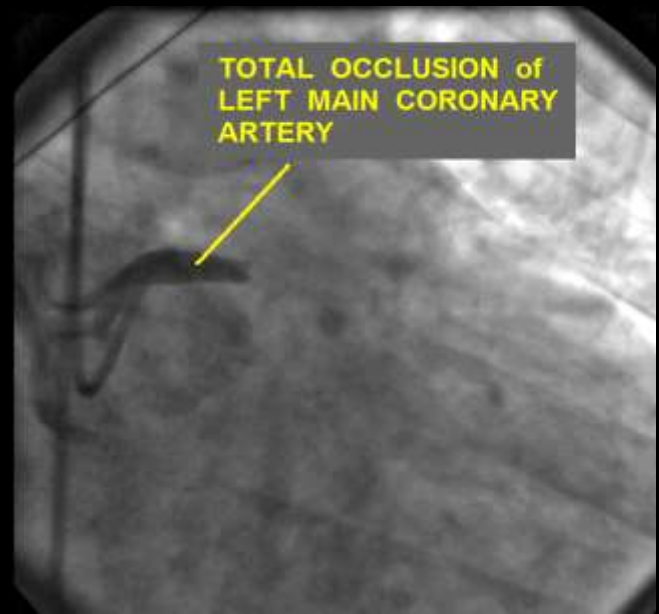
**PATIENT A:** 44 y/o MALE, CHEST PAIN x 1 HOUR,
BP: 78/46, P: 70, R: 28. CARDIAC MARKERS: NEGATIVE



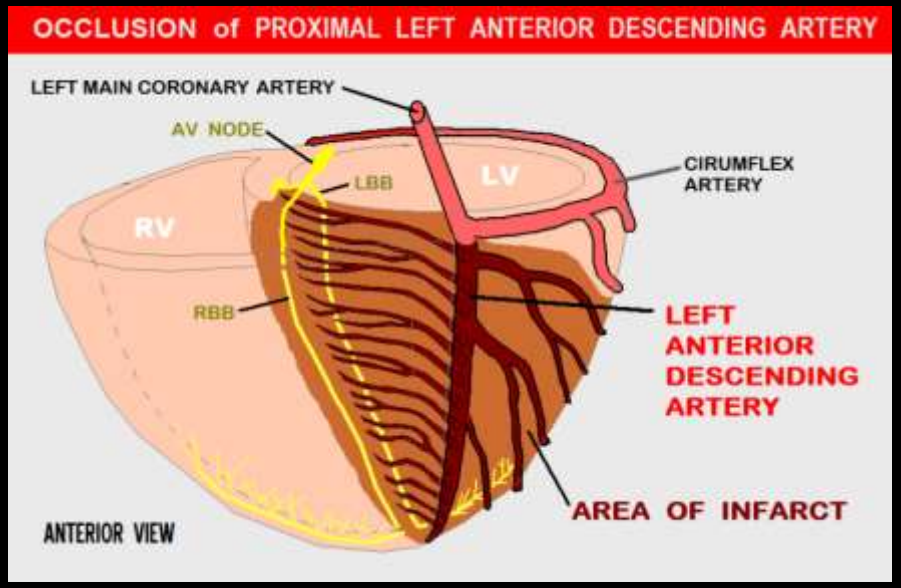




**PATIENT B:** 36 y/o MALE, CHEST PAIN x 1 HOUR,
BP: 80/48, P: 120, R: 28 CARDIAC MARKERS: NEGATIVE

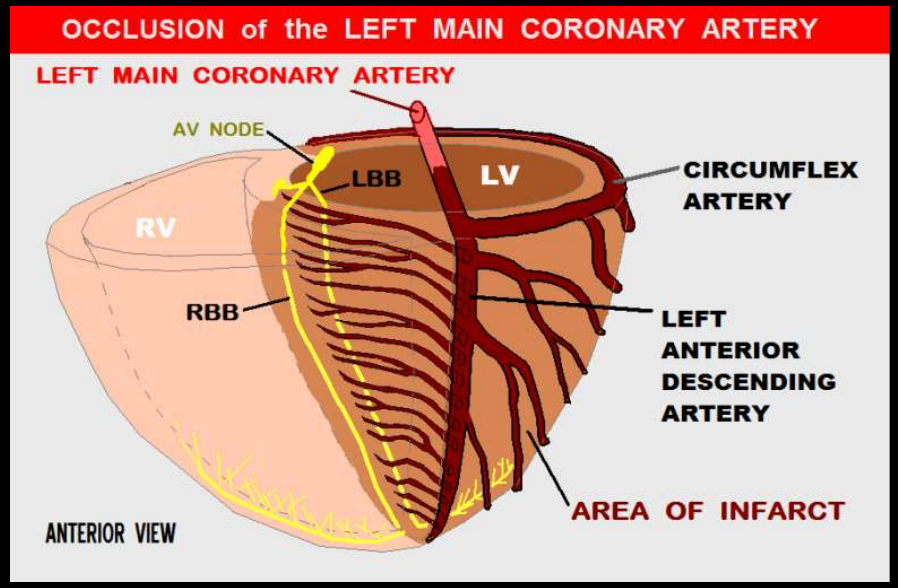


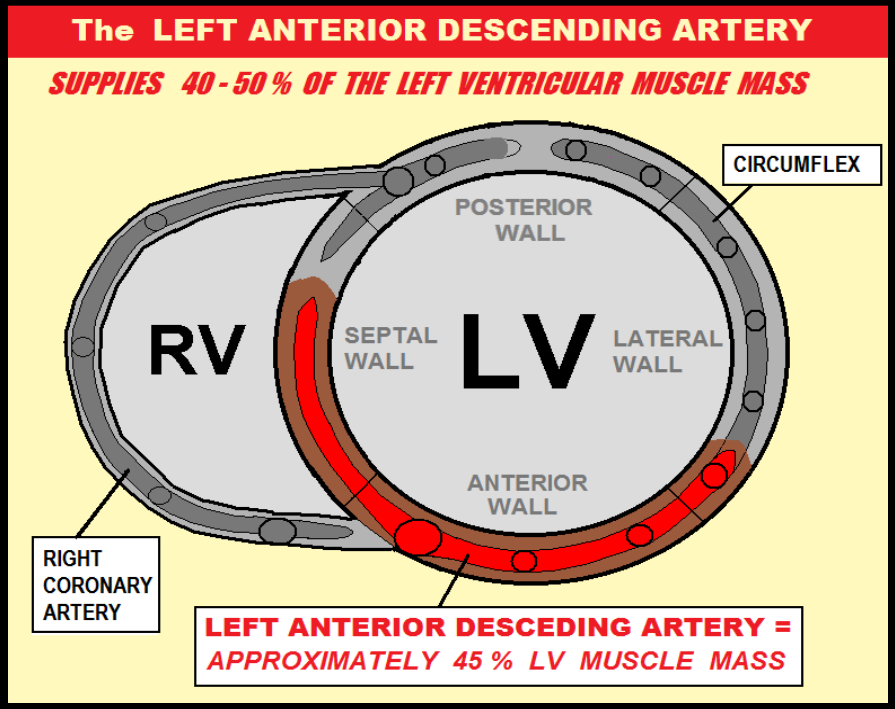



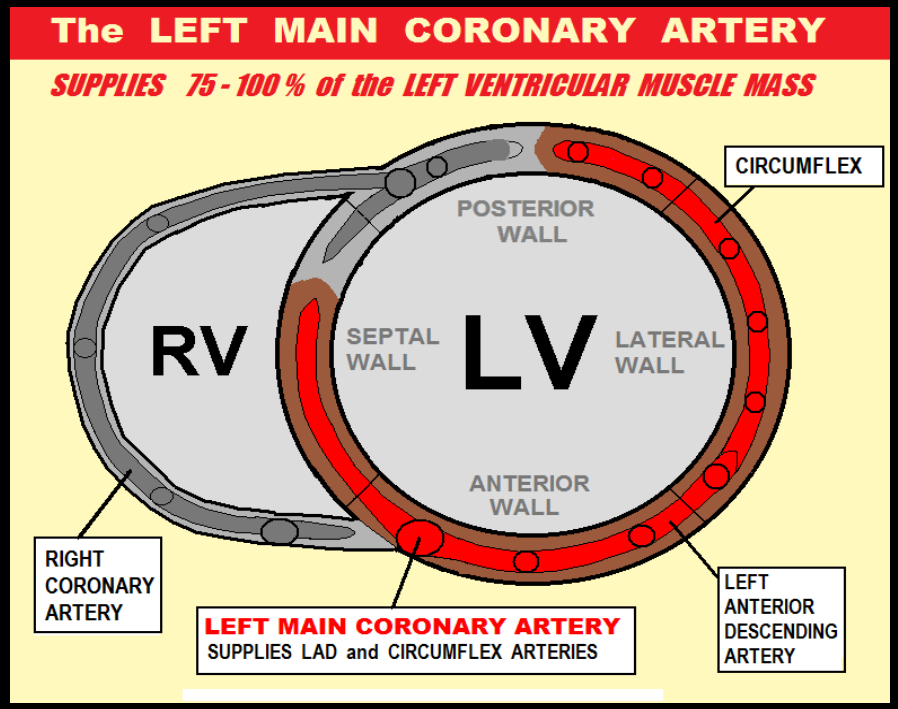
PATIENT A:




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 *+

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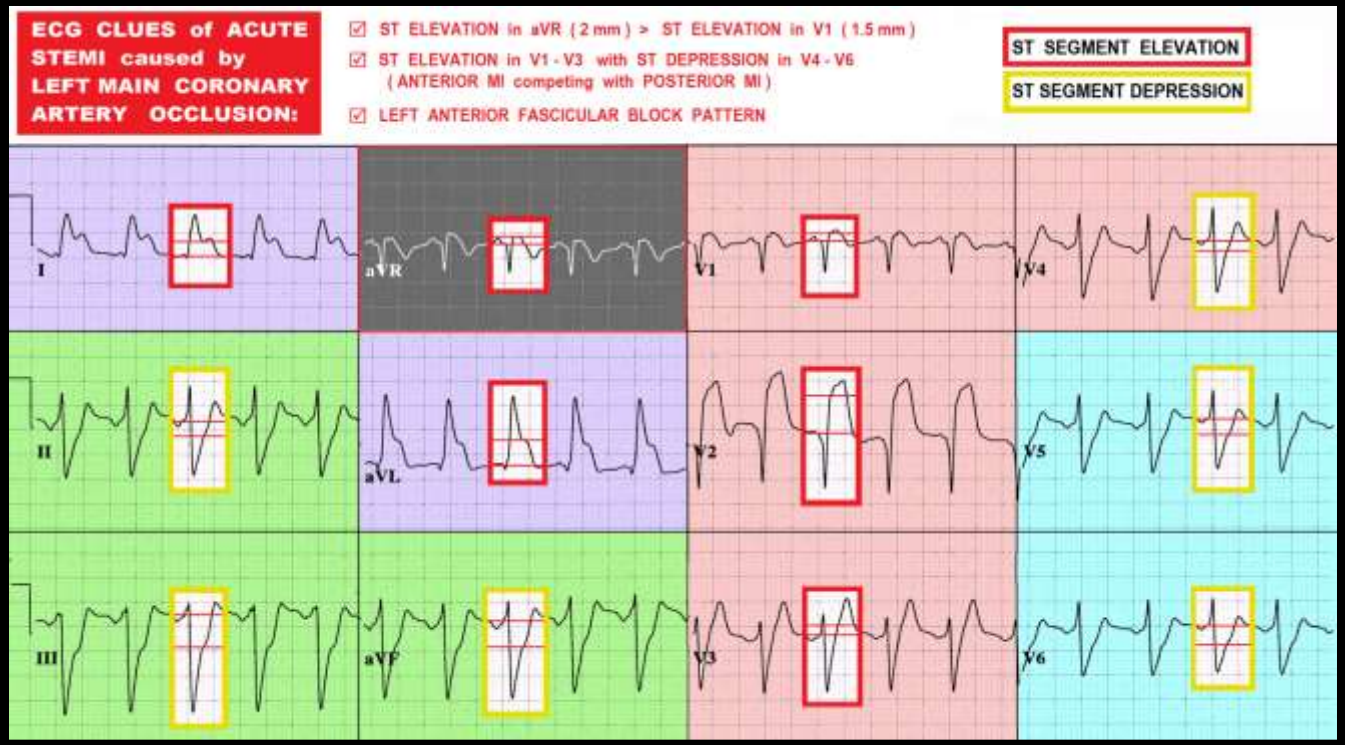
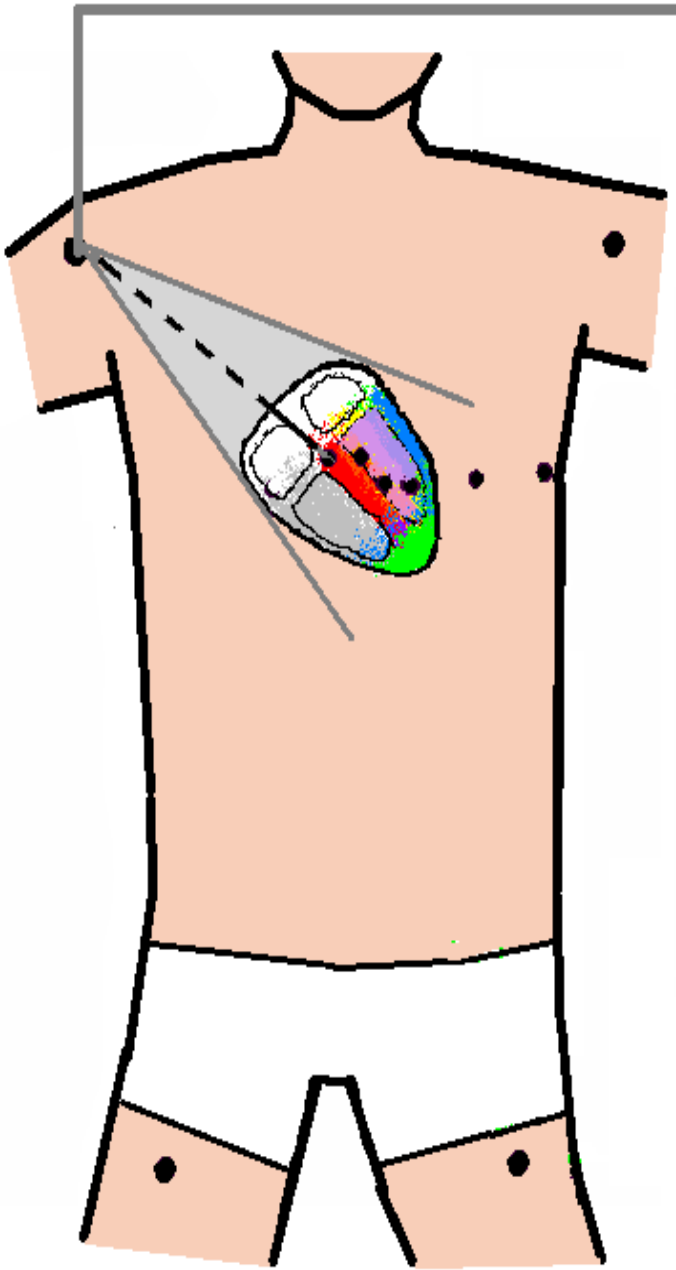
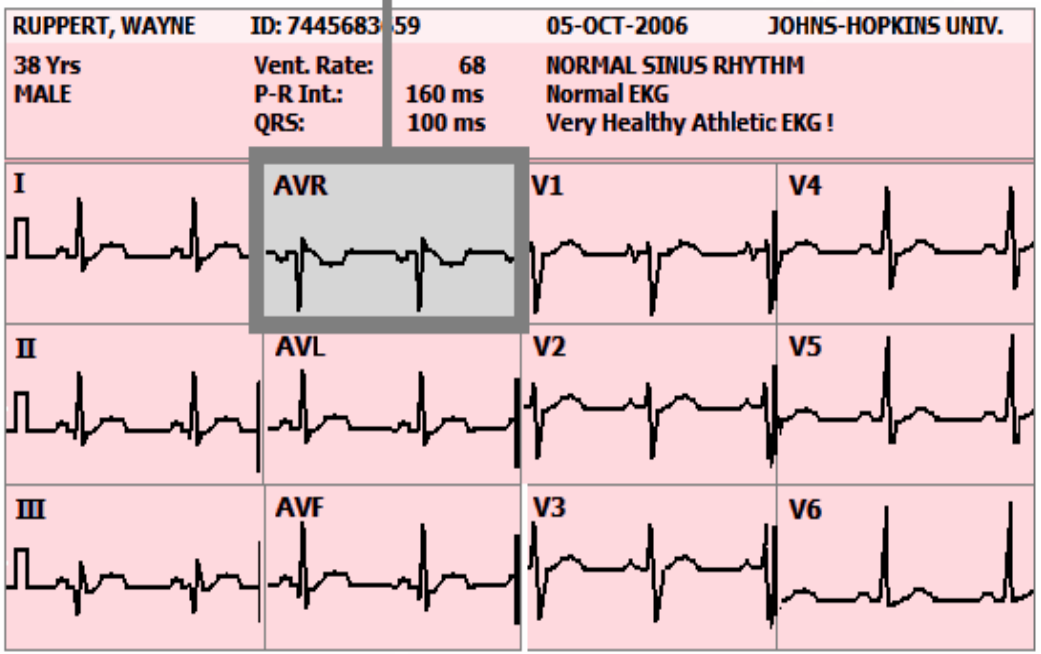
* Kurisu et al, HEART 2004, SEPTEMBER: 90 (9): 1059-1060

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

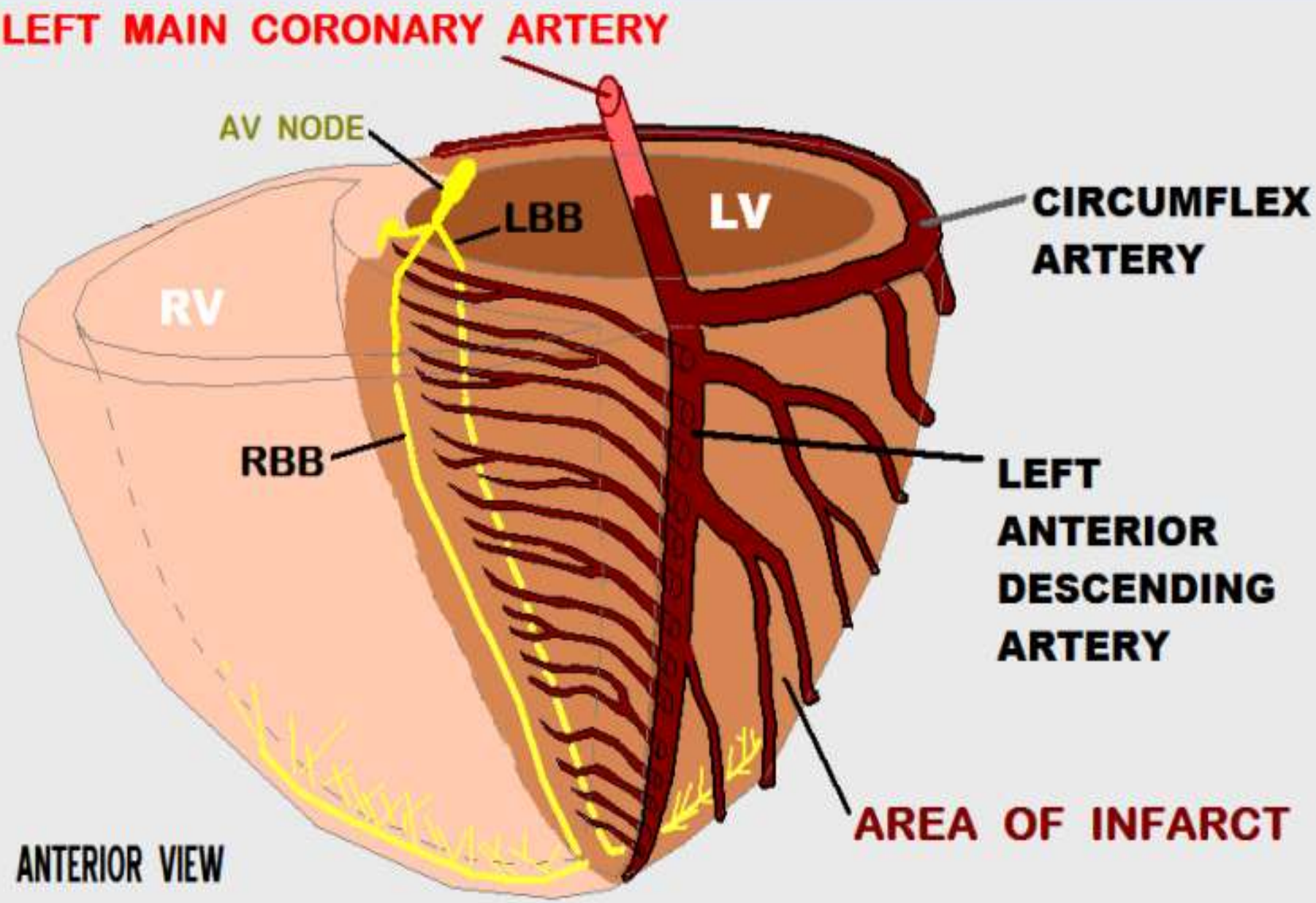
# Lead AVR Views the BASILAR SEPTUM (region of the Bundle of His)

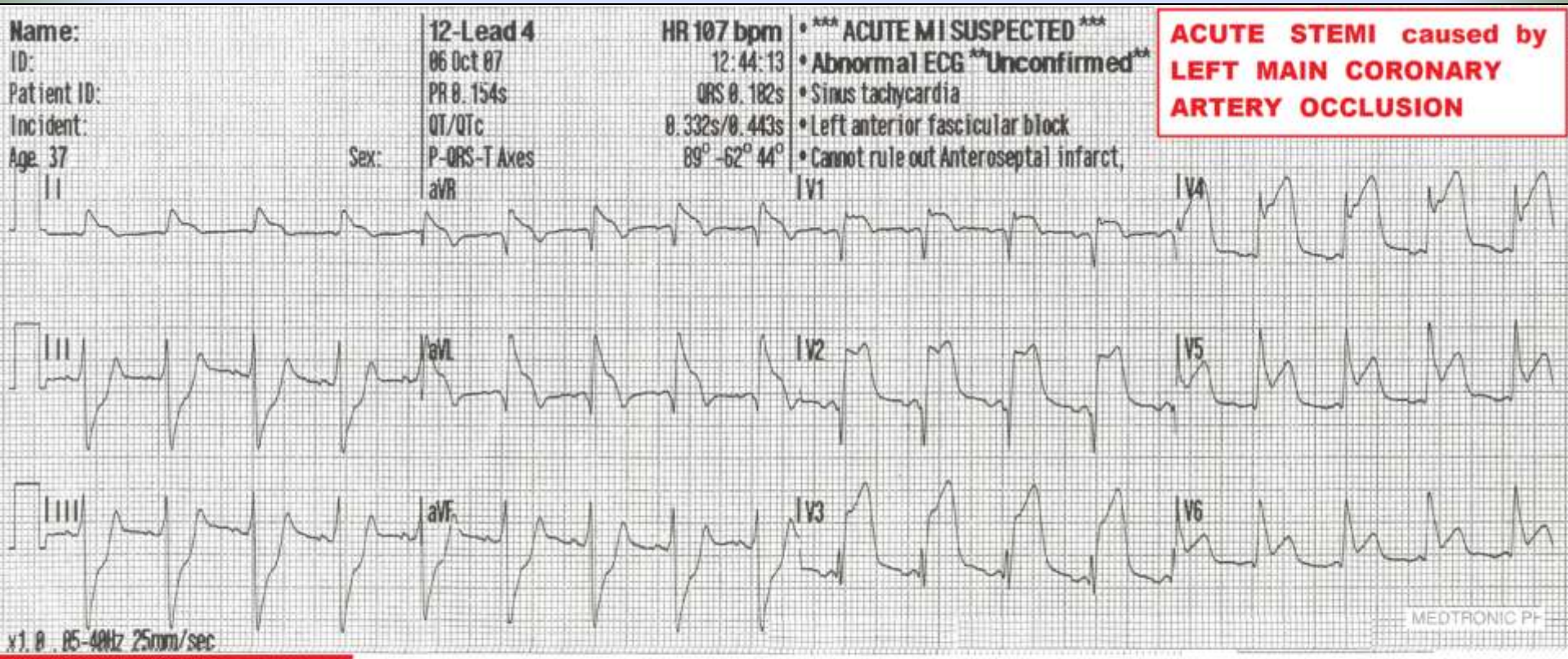




In STEMI with ST-Segment Elevation in Lead AVR,
This is indicative of
Left Main Coronary Artery Occlusion . . .

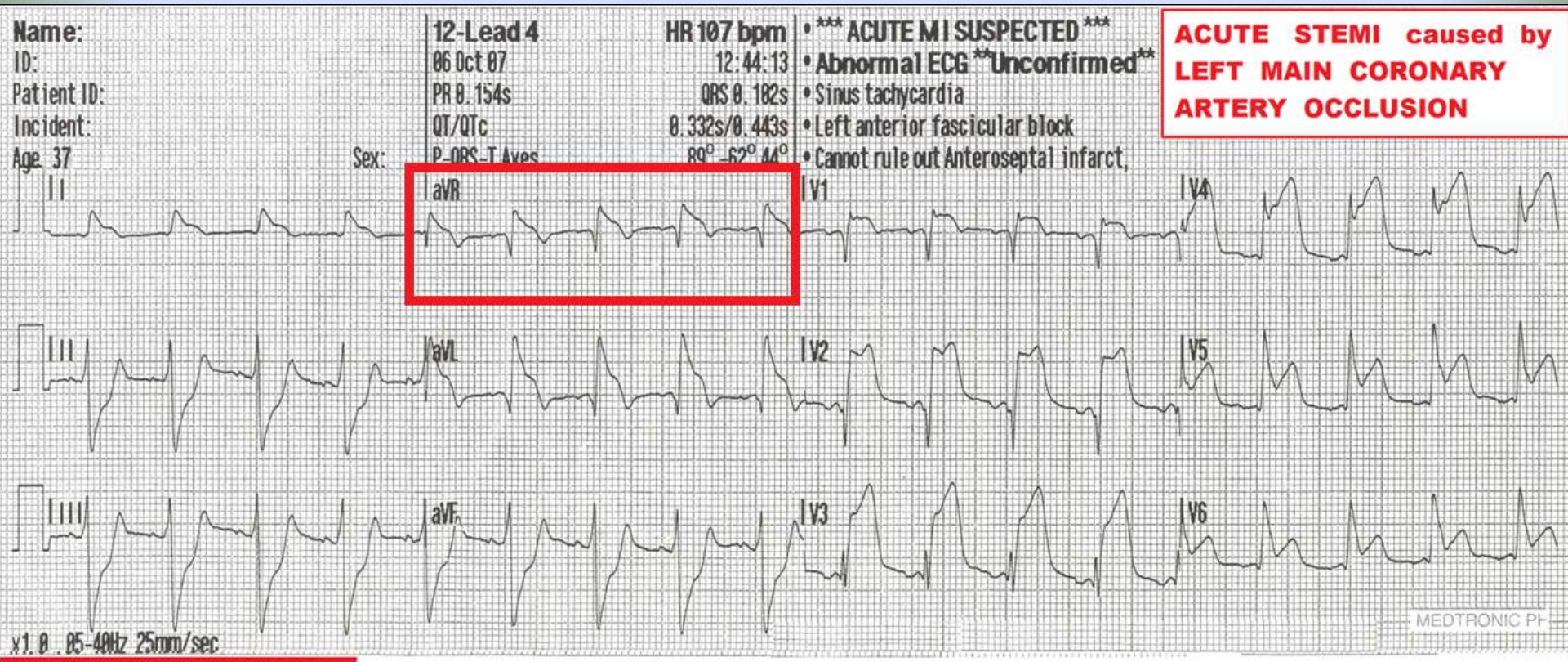
# OCCLUSION of the LEFT MAIN CORONARY ARTERY






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

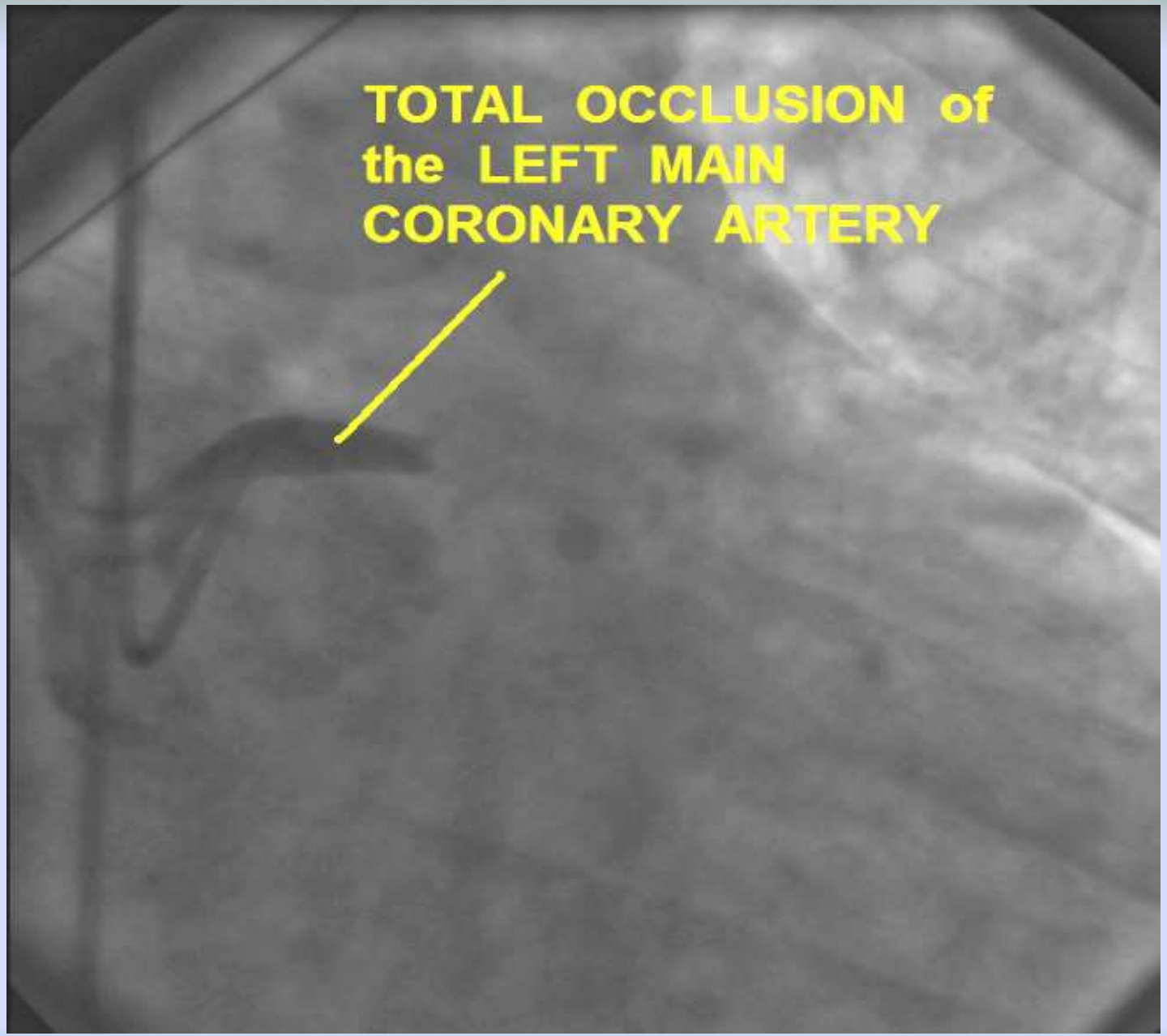
- ☑ ST ELEVATION in LEADS I, aVL, V1 - V6
- ☑ ST ELEVATION in aVR GREATER THAN 0.5 mm
- ☑ ST ELEVATION in aVR GREATER THAN LEAD V1
- ☑ LEFT ANTERIOR FASCICULAR BLOCK PATTERN

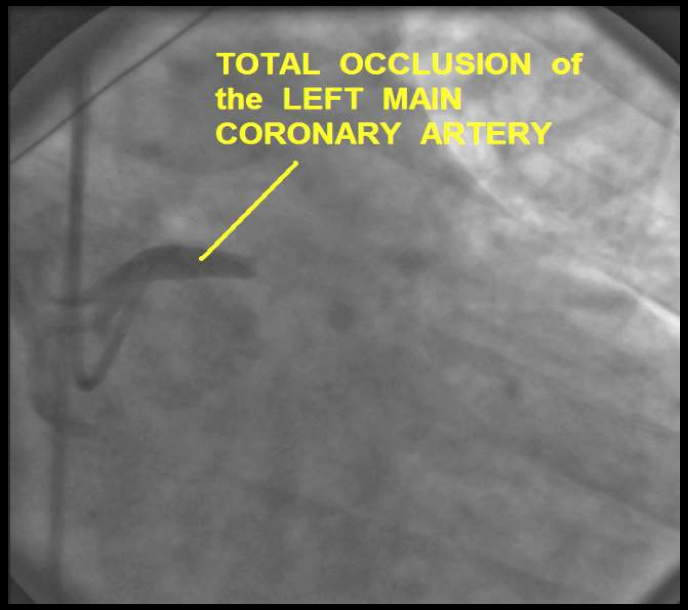


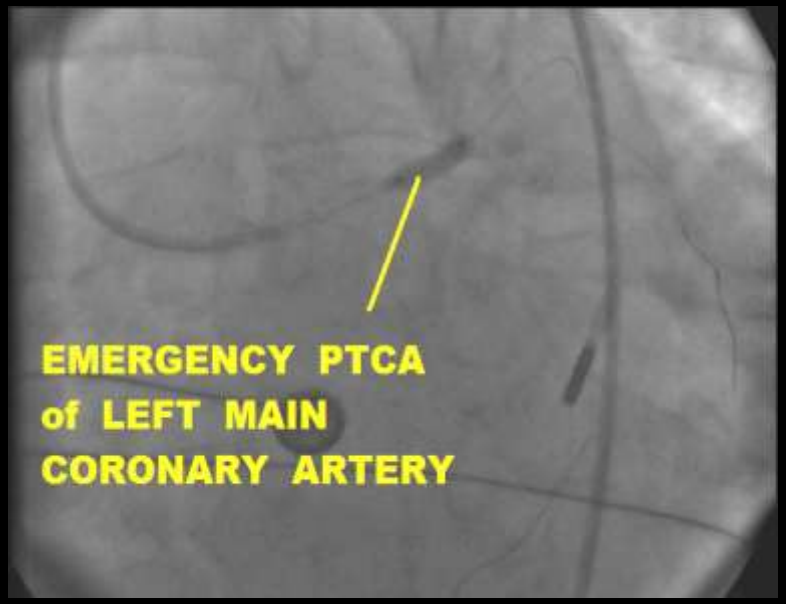

**ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION**

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

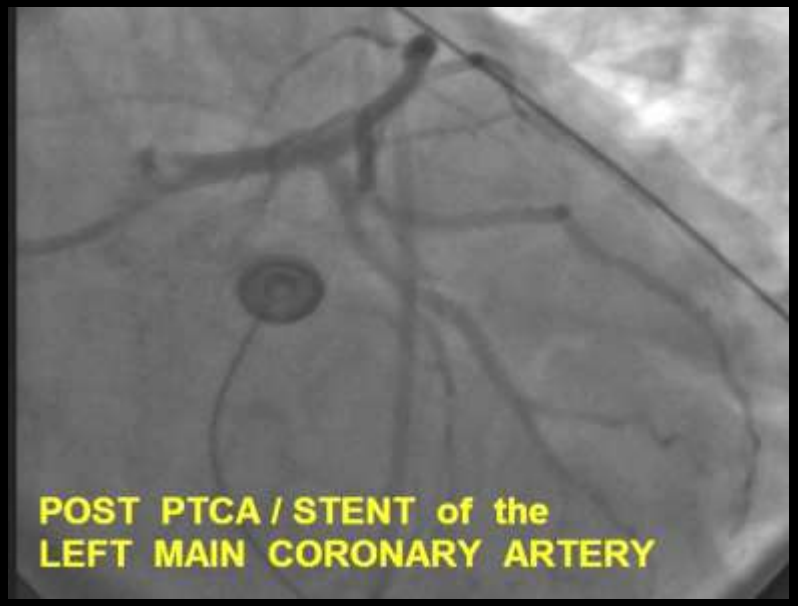
- ☑ ST ELEVATION in LEADS I, aVL, V1 - V6
- ☑ ST ELEVATION in aVR GREATER THAN 0.5 mm
- ☑ ST ELEVATION in aVR GREATER THAN LEAD V1
- ☑ LEFT ANTERIOR FASCICULAR BLOCK PATTERN


TOTAL OCCLUSION of
the LEFT MAIN
CORONARY ARTERY




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



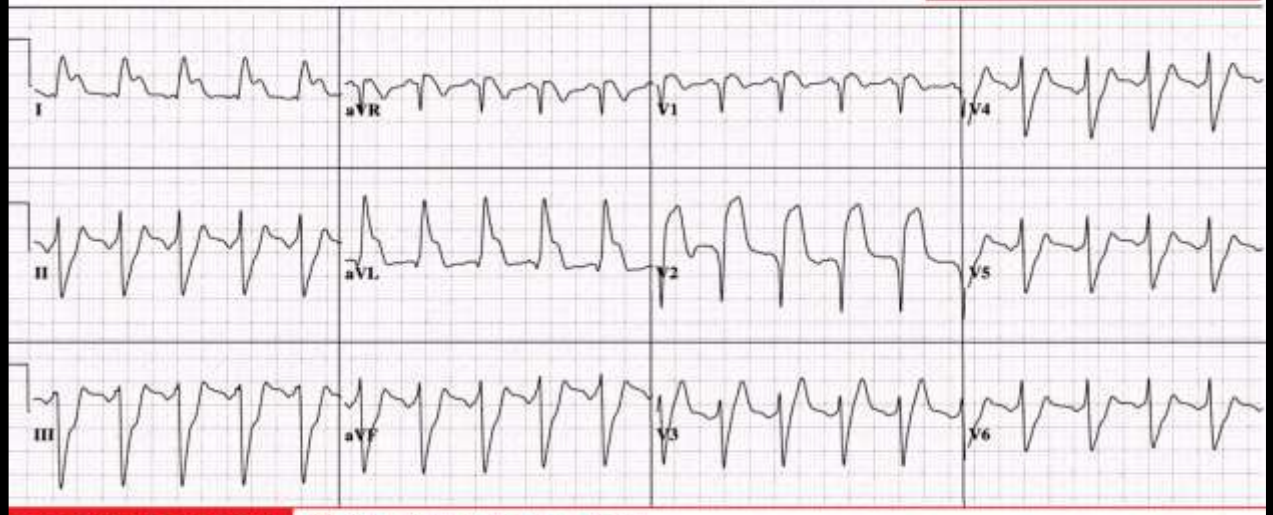
36 yr Male Caucasian

Room:C-
Loc:3

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

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

**ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION**




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

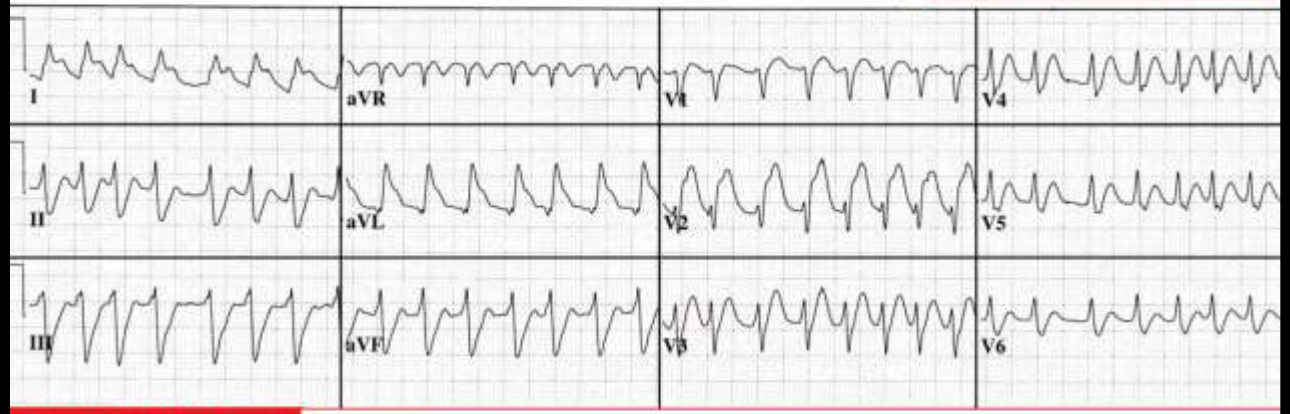
- ☑ ST ELEVATION in leads I and aVL
- ☑ INCONSISTENCY of ST SEGEMENT in leads V1 - V6 : V1 - V3 ST ELEVATION, V4 - V6 ST DEPRESSION (COMPETING FORCES of ANTERIOR vs. POSTERIOR M.I.)
- ☑ PATTERN of LEFT ANTERIOR FASCICULAR BLOCK (POS. QRS lead I; NEG rS leads II, III)
- ☑ ST ELEVATION in lead aVR > 0.5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

43 yr Male

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

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

**ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION**




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

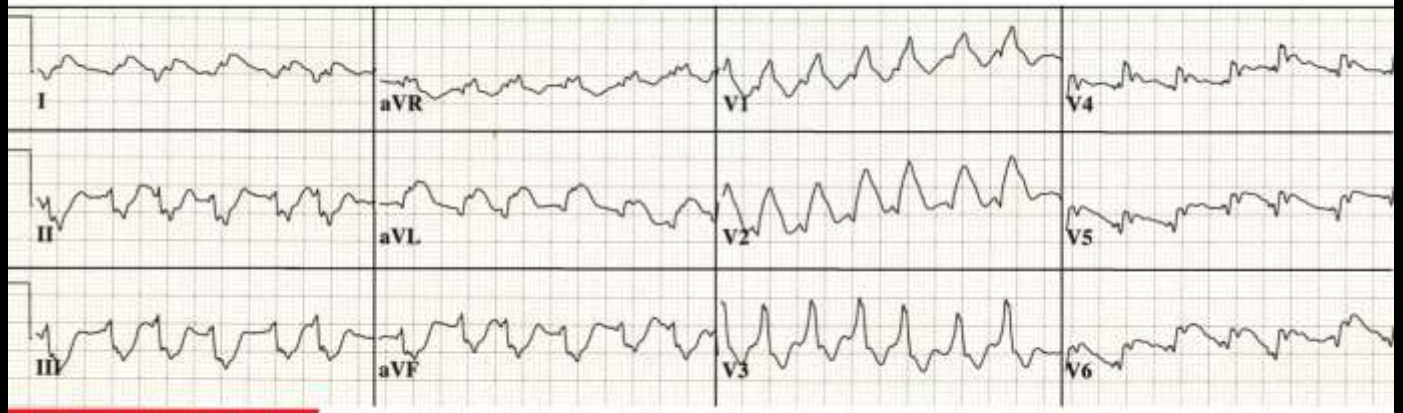
- ☑ ST ELEVATION in leads I and aVL
- ☑ INCONSISTENCY of ST SEGEMENT in leads V1 - V6 : V1 - V2 ST ELEVATION, V3 - V6 ST DEPRESSION (COMPETING FORCES of ANTERIOR vs. POSTERIOR M.I.)
- ☑ PATTERN of LEFT ANTERIOR FASCICULAR BLOCK (POS. QRS lead I; NEG rS leads II, III)

48 yr
Male Caucasian

Loc: Option:

| | | |
|---|---|---|
| Vent. rate | 155 | BPM |
| PR interval | * | ms |
| QRS duration | 110 | ms |
| QT/QTc | 300/482 | ms |
| P-R-T axes | * -83 | -34 |

**ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION**



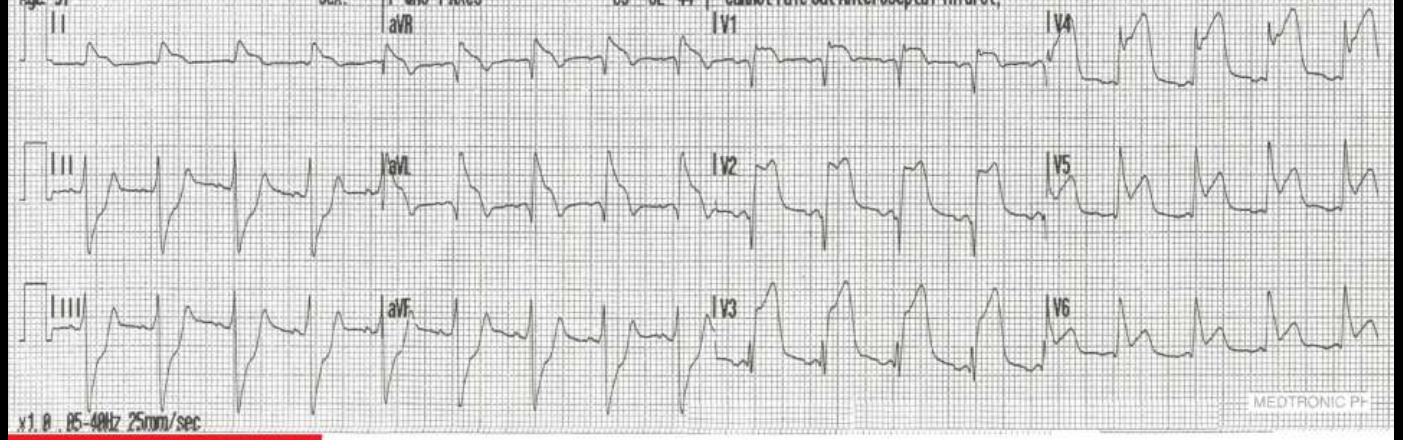

**ECG CLUES of ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION:**

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

Name:
ID:
Patient ID:
Incident:
Age: 37 Sex:

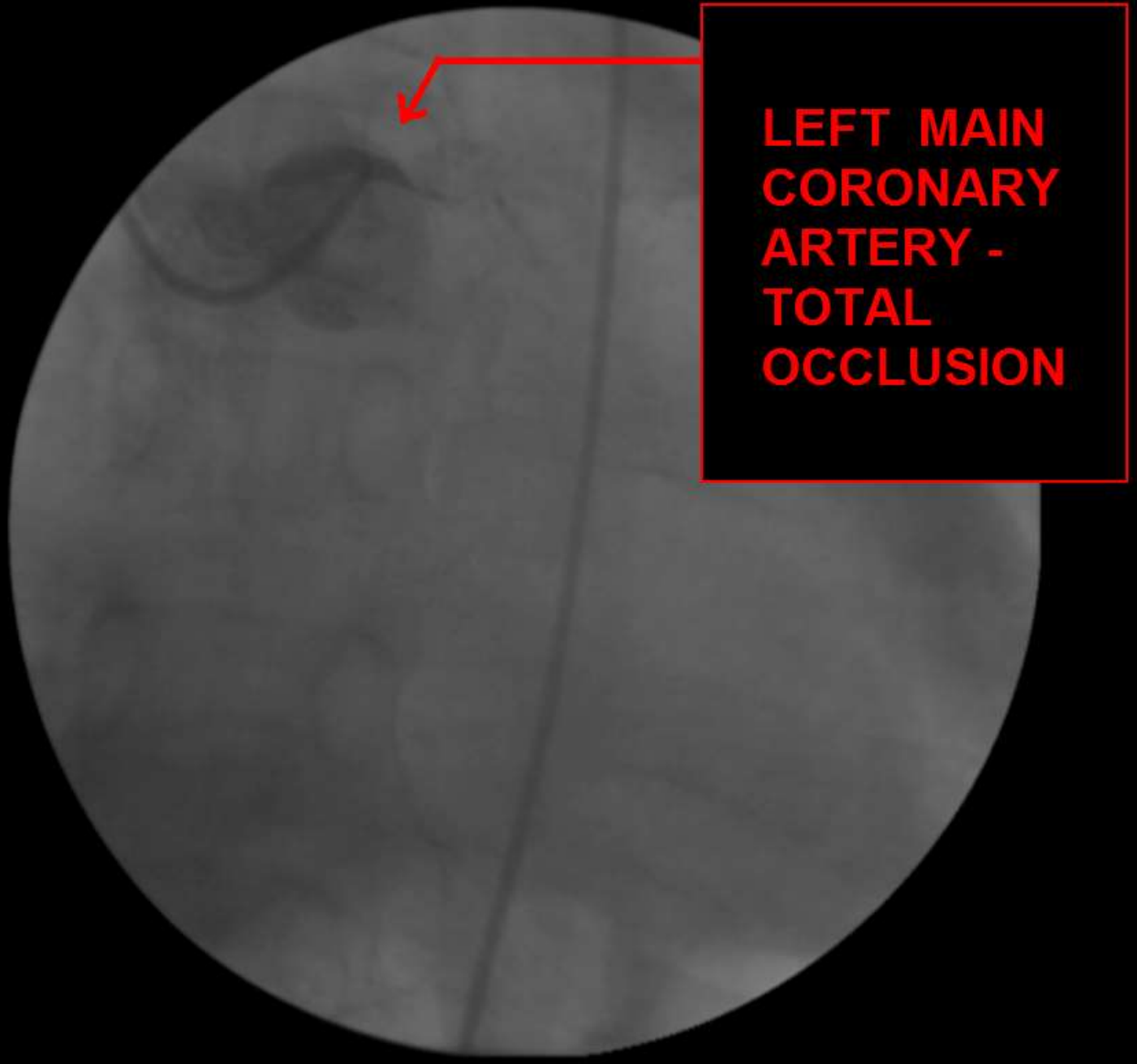
| | | |
|---|---|---|
| 12-Lead 4 | HR 107 bpm | *** ACUTE MI SUSPECTED *** |
| 06 Oct 07 | 12:44:13 | • Abnormal ECG **Unconfirmed** |
| PR 0.154s | QRS 0.182s | • Sinus tachycardia |
| QT/QTc | 0.332s/0.443s | • Left anterior fascicular block |
| P-QRS-T Axes | 89° -62° 44° | • Cannot rule out Anteroseptal infarct, |

**ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION**




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

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


LEFT MAIN
CORONARY
ARTERY -
TOTAL
OCCLUSION

# CASE STUDY 4: CRITICAL DECISIONS SCENARIO

CONCLUSIONS:

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

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

---

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

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

# 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


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

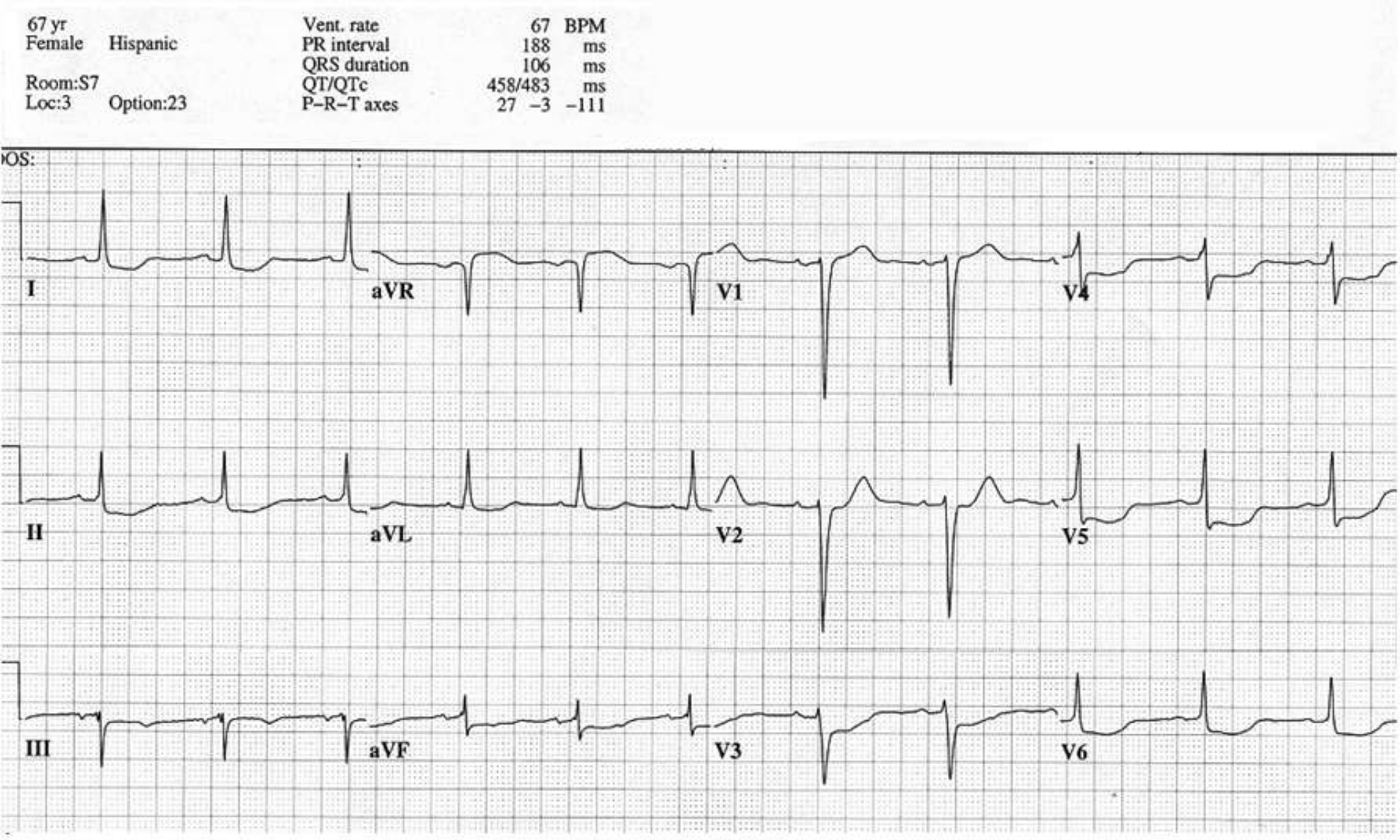
Electrocardiogram patterns in acute left main occlusion: J Electrocardiol. 2008 Nov-Dec;41(6):626-9.

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

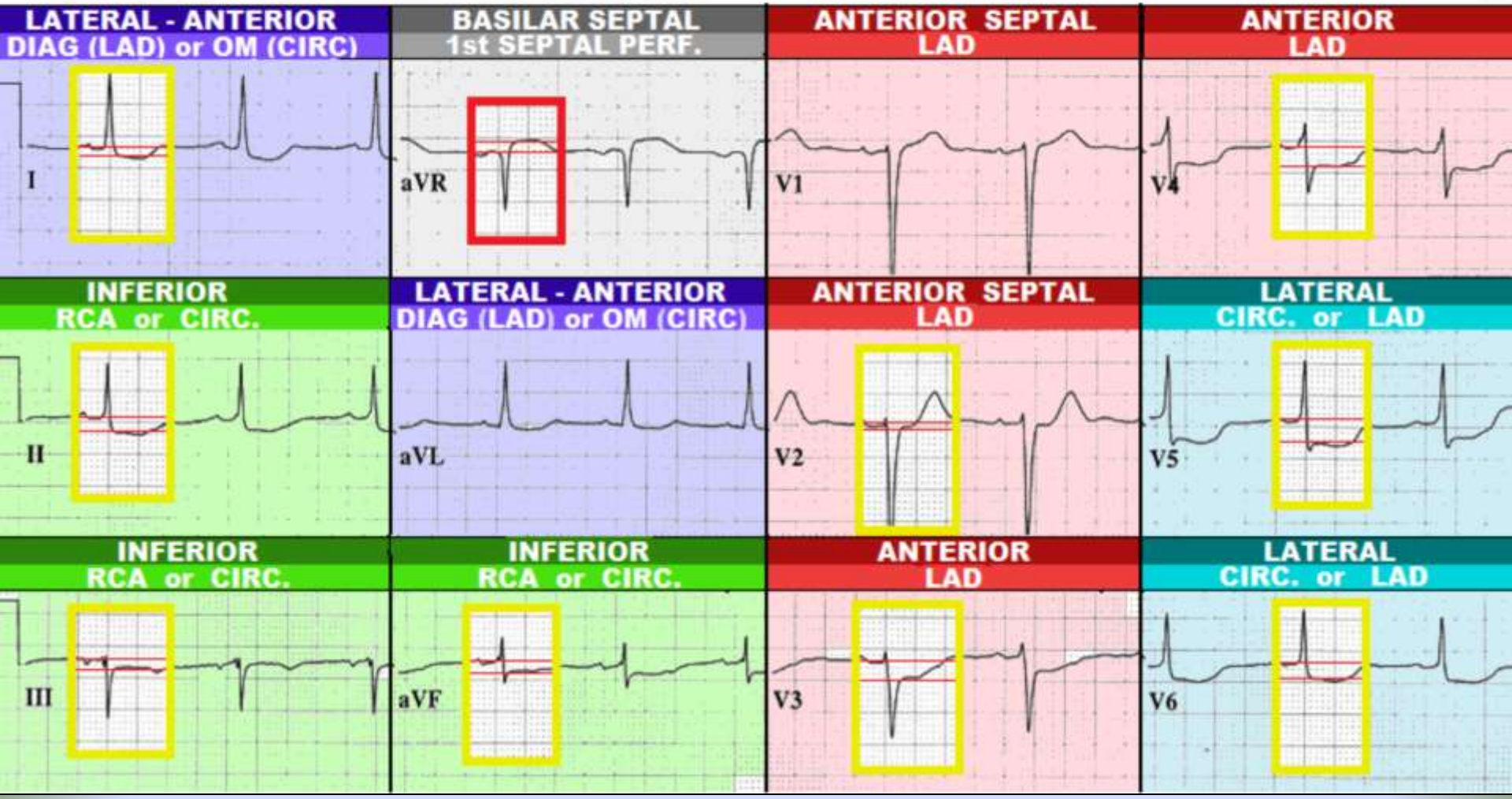
67 yr
Female Hispanic

Room:S7
Loc:3 Option:23

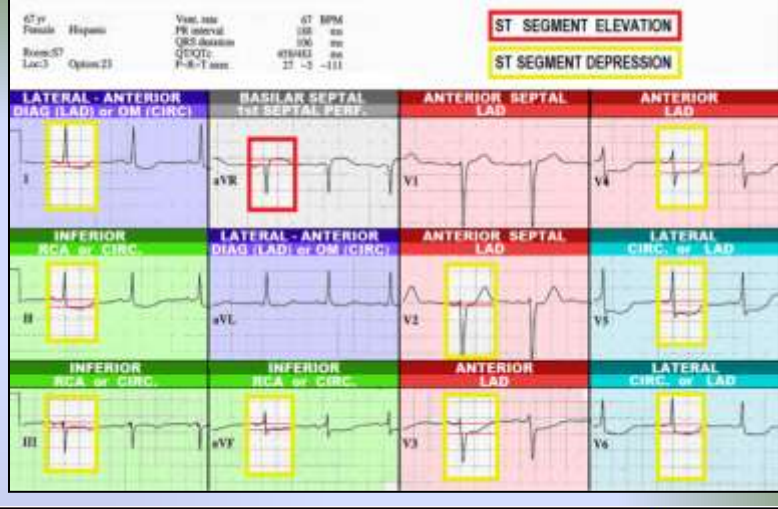
| | | |
|---|---|---|
| Vent. rate | 67 | BPM |
| PR interval | 188 | ms |
| QRS duration | 106 | ms |
| QT/QTc | 458/483 | ms |
| P-R-T axes | 27 -3 | -111 |

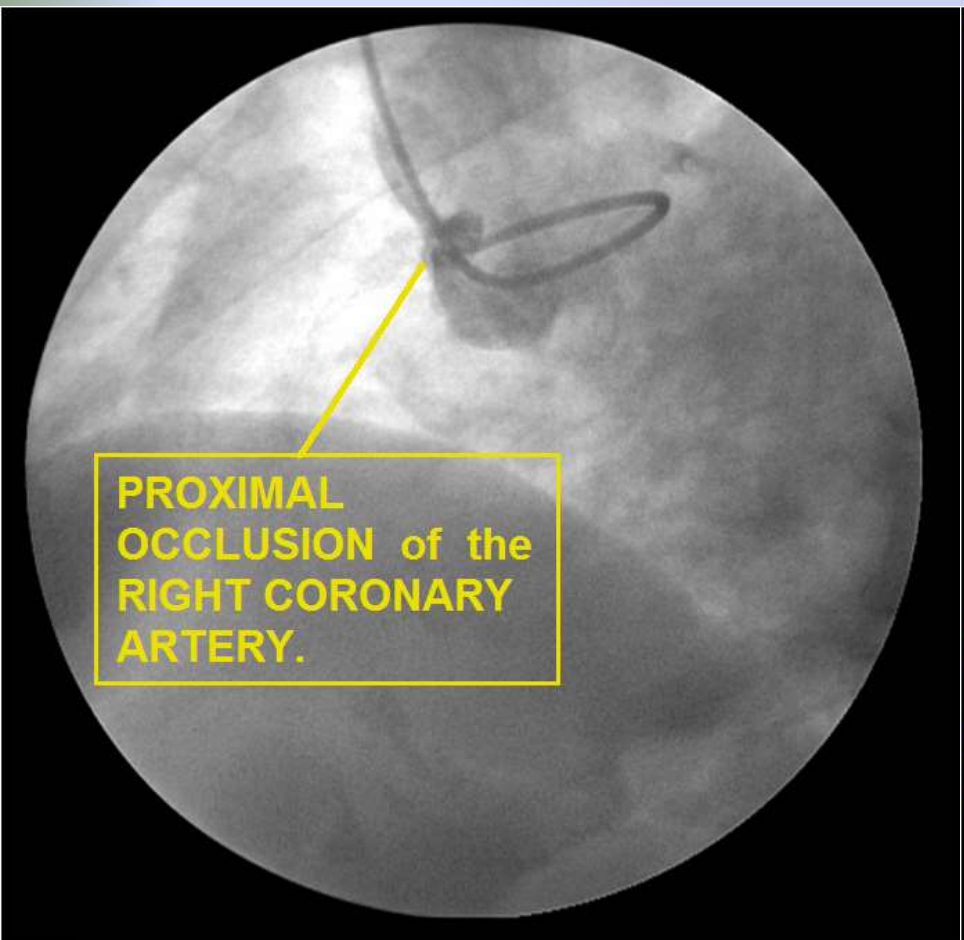
**ST SEGMENT ELEVATION**

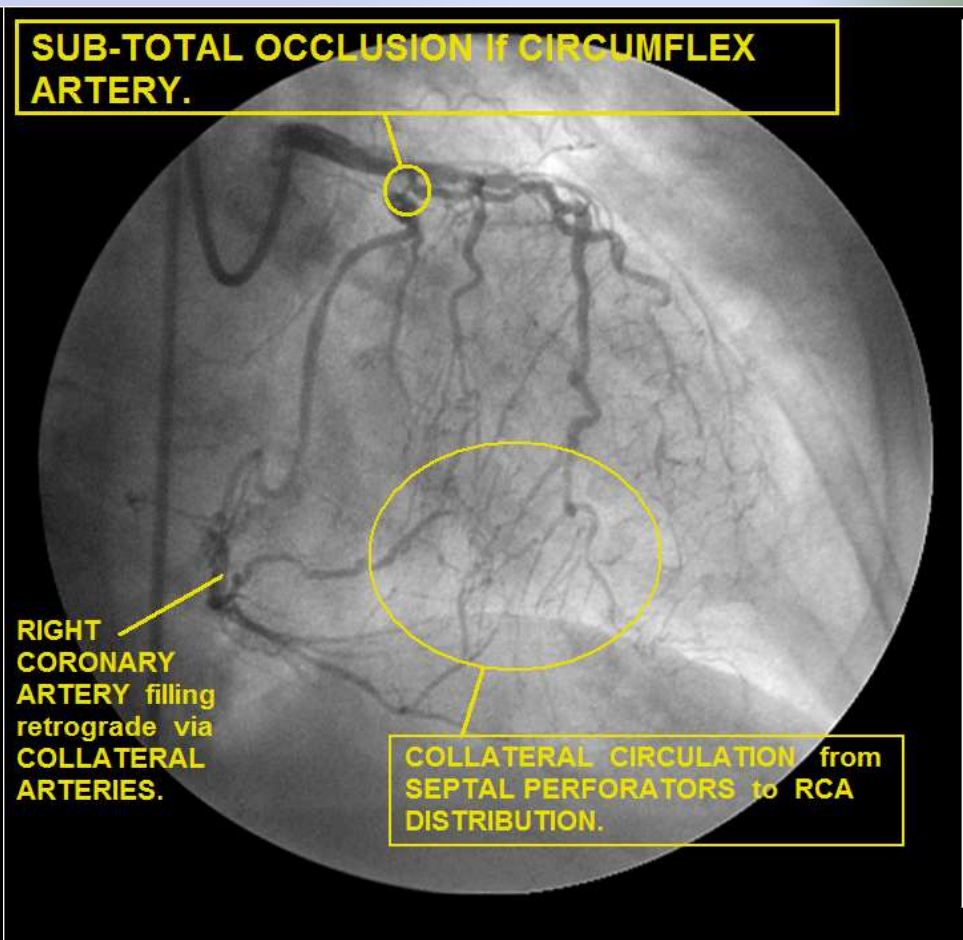
**ST SEGMENT DEPRESSION**



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



Pat ID [redacted]

[redacted]/2021 18:13:30
63 yrs

[redacted]
Male
Account # [redacted]

RX
DX

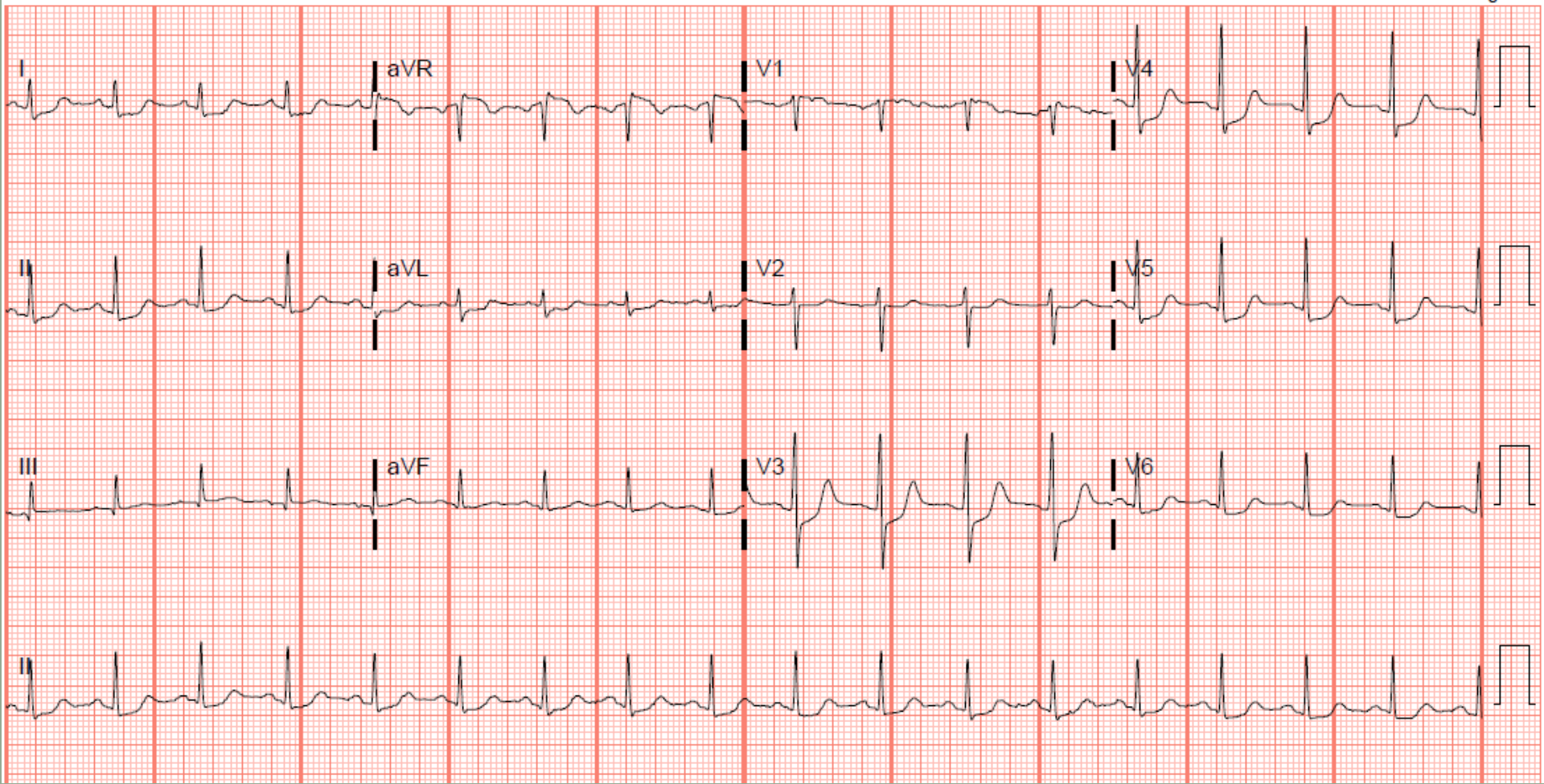
Bayfront Health Seven Rivers ED
Dept ED
Room ED08
Tech ao

Req Provider:

| | | |
|---|---|---|
| Rate | 104 | Sinus tachycardia |
| PR | 134 | RSR' in V1 or V2, right VCD or RVH |
| QRSd | 90 | Repol abnrm, severe global ischemia (LM/MVD) |
| QT | 337 | COMPARED TO ECG 01/07/2021 18:06:15 |
| QTc | 444 | SINUS TACHYCARDIA NOW PRESENT |
| --Axis-- | | RIGHT VENTRICULAR HYPERTROPHY NOW PRESENT |
| P | 40 | POSSIBLE ISCHEMIA NOW PRESENT |
| QRS | 66 | |
| T | 50 | |

- Abnormal ECG -

Unconfirmed Diagnosis



Operative Findings:
Coronary circulation findings:
- Right dominant coronary system
- Left main distal 80%
- Left anterior descending ostial 80% and mid 90% lesion with D1 40% lesiojn ostial
- Circumflex ostial 80% and mid 40%
- OM1 and OM2 with 70% lesions
- Right coronary artery ostial 70% with normal RPDA and RPLA

IMPRESSIONS:
- LM and significant coronary artery disease.
- Normal left ventricular function.
- Normal left ventricular end-diastolic pressure.

RECOMMENDATIONS:
- please transfer the pt to CMH for CABG the team was contacted ( Dr Kim and Dr Hoang)
- restart the iv heparin in 6 hrs
- resume the ASA and statin
- Rest of therapy per cardiovascular team.
- Routine post-cardiac catheterization care and per protocol access site management.

| 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.<br><br>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)<br>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.

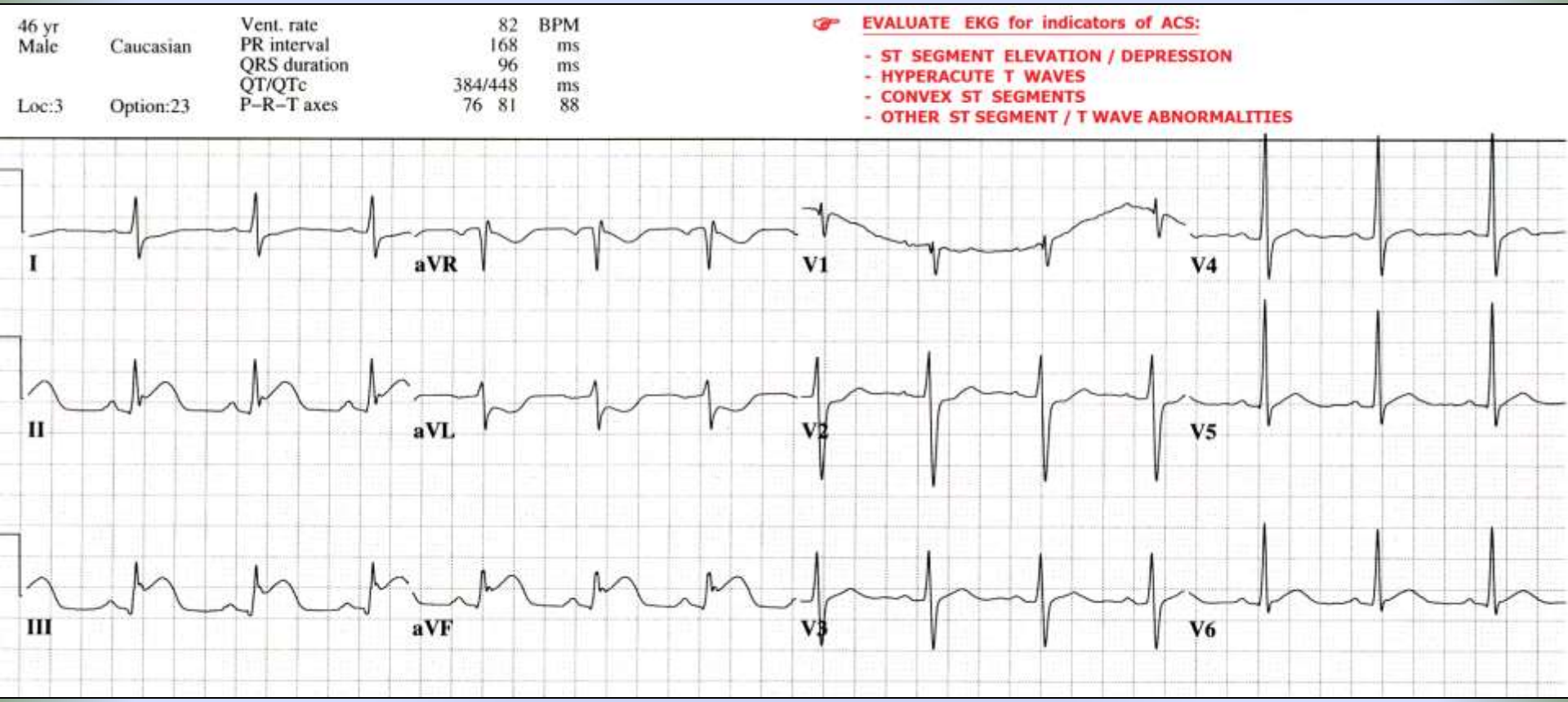
## RISK FACTOR PROFILE:

- 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
Male
Caucasian
Loc:3
Option:23
Vent. rate 82 BPM
PR interval 168 ms
QRS duration 96 ms
QT/QTc 384/448 ms
P–R–T axes 76 81 88
EVALUATE EKG for indicators of ACS:
- ST SEGMENT ELEVATION / DEPRESSION
- HYPERACUTE T WAVES
- CONVEX ST SEGMENTS
- OTHER ST SEGMENT / T WAVE ABNORMALITIES
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

46 yr
Male

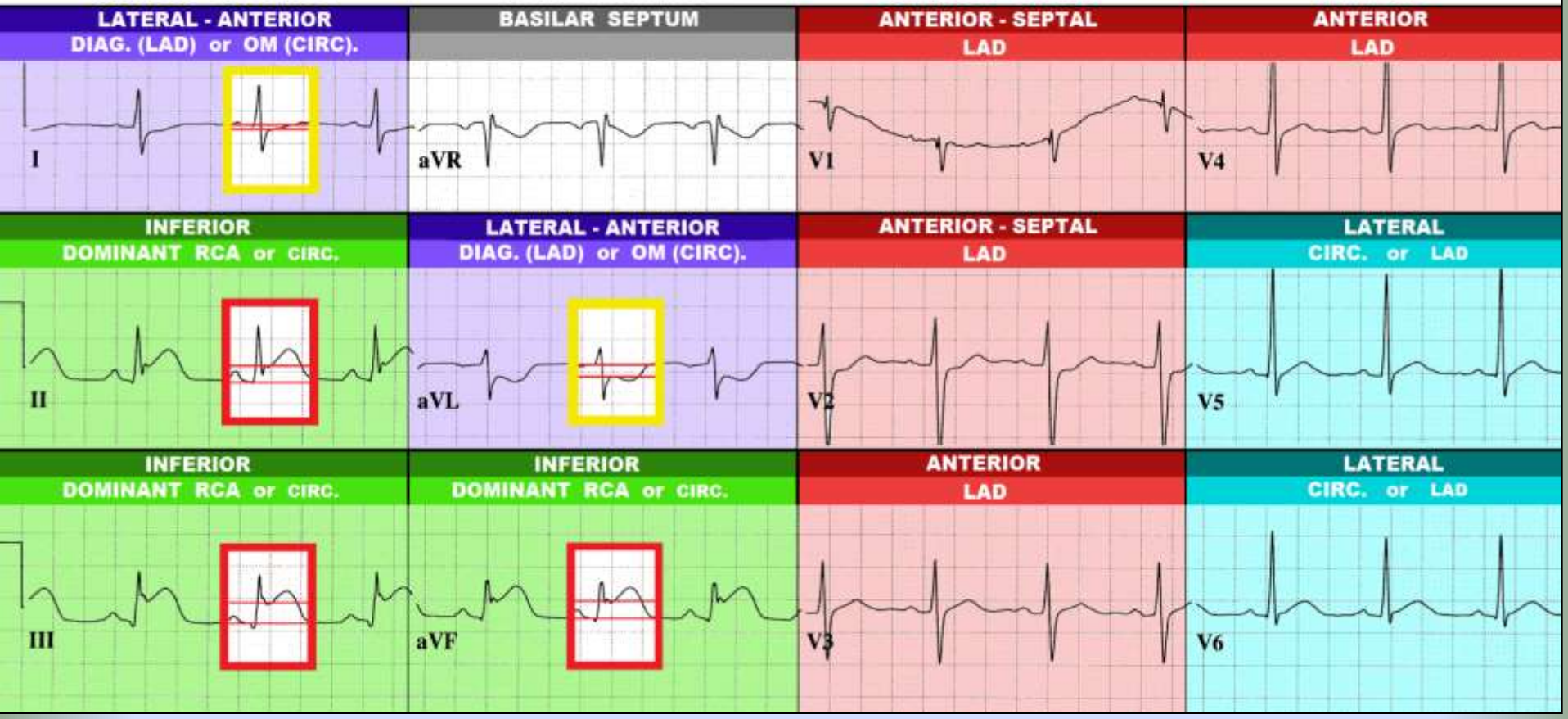
Caucasian

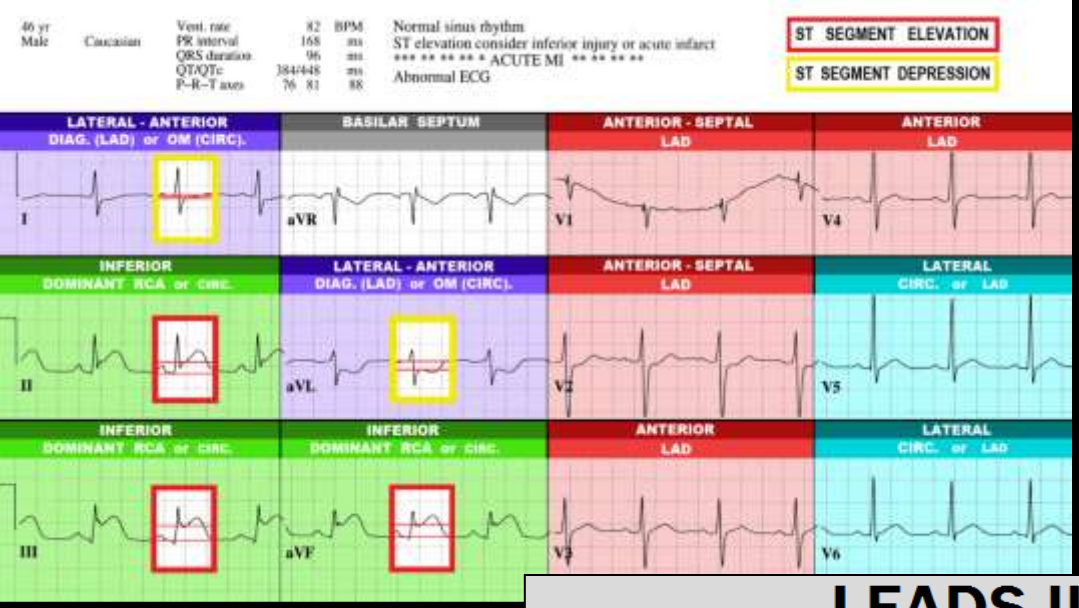
| | | |
|---|---|---|
| Vent. rate | 82 | BPM |
| PR interval | 168 | ms |
| QRS duration | 96 | ms |
| QT/QTc | 384/448 | ms |
| P–R–T axes | 76 81 | 88 |

Normal sinus rhythm
ST elevation consider inferior injury or acute infarct
*** ** ** ** * ACUTE MI ** ** ** **
Abnormal ECG

ST SEGMENT ELEVATION

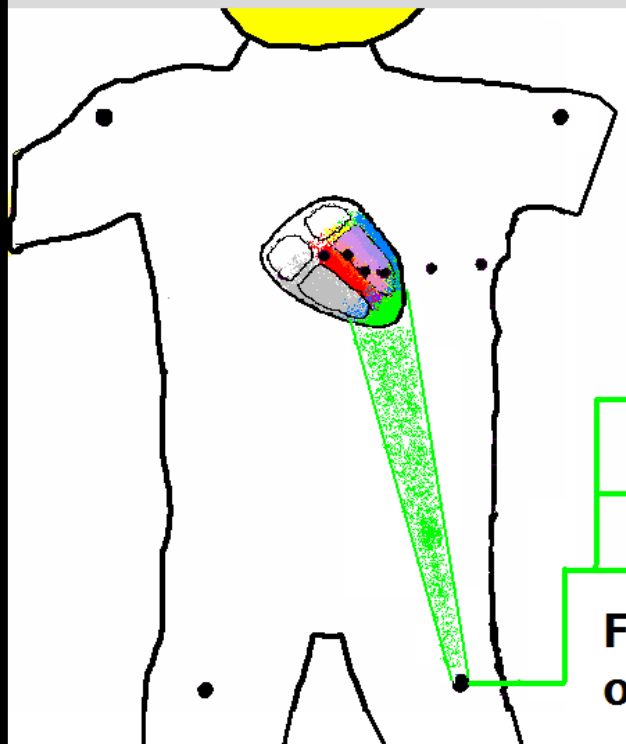
ST SEGMENT DEPRESSION

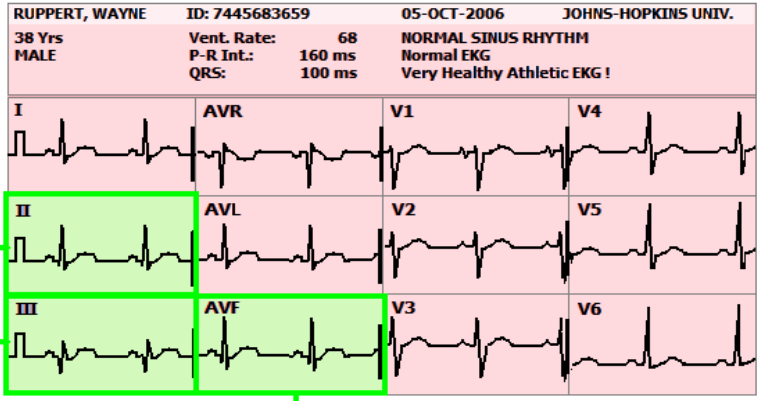





# LEADS II, III, and aVF VIEW

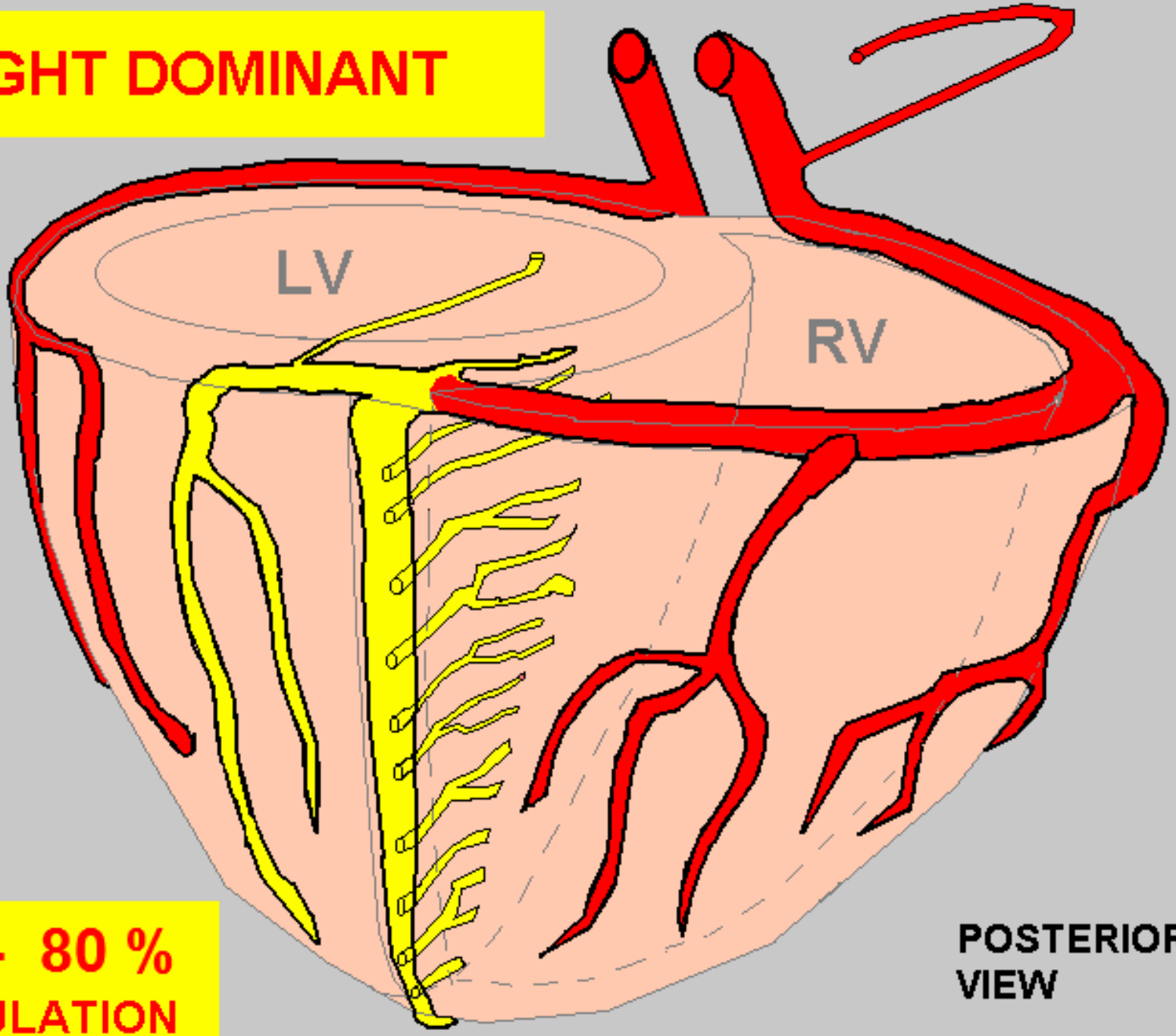
## INFERIOR WALL of the LEFT VENTRICLE






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

# RIGHT DOMINANT




**75 - 80 %**
**POPULATION**

**POSTERIOR VIEW**

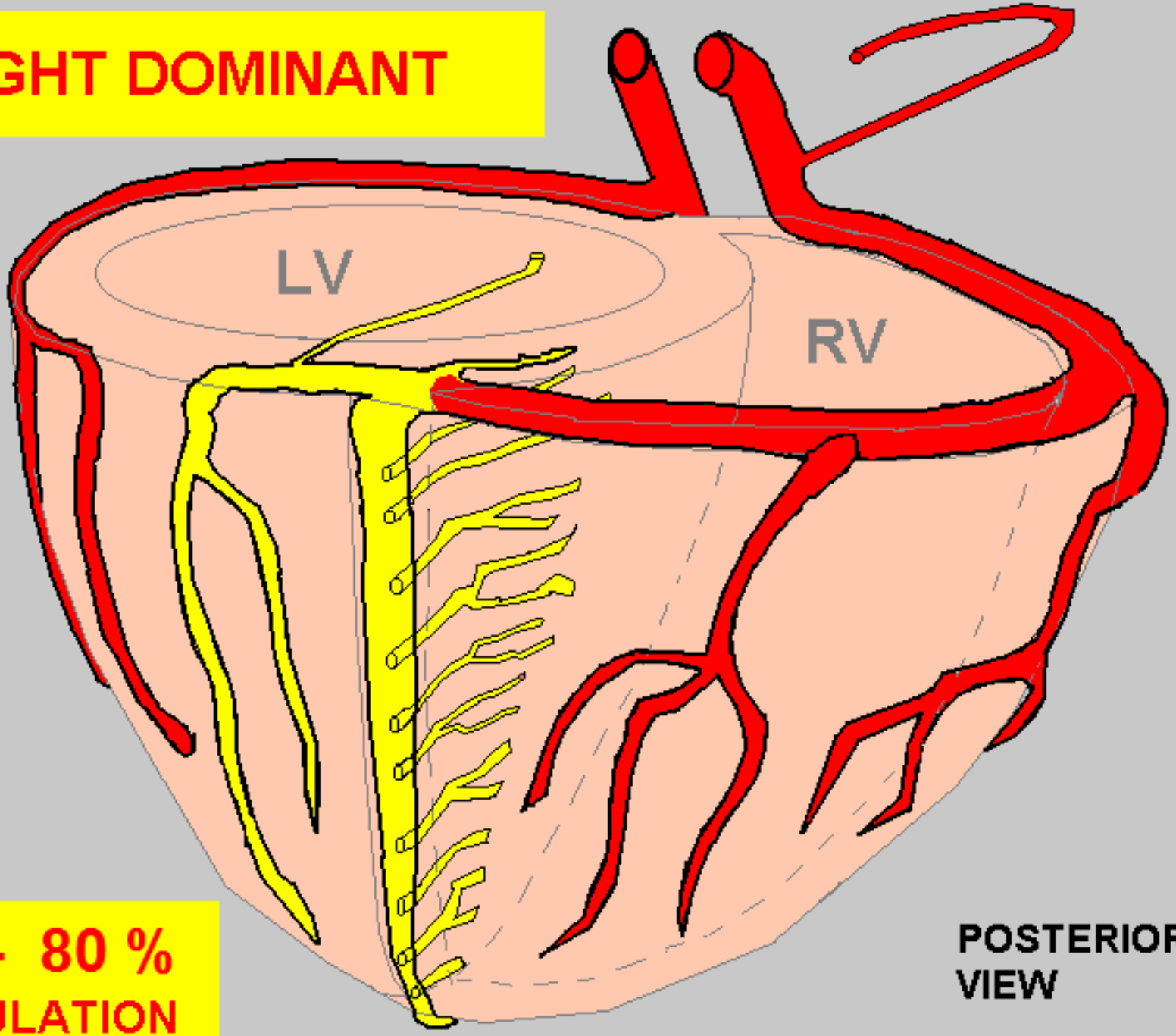
HELPFUL HINT . . . MEMORIZE THIS !

# RIGHT CORONARY ARTERY ( RCA )

RIGHT DOMINANT SYSTEMS

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

# RIGHT DOMINANT




**75 - 80 %**
**POPULATION**

**POSTERIOR VIEW**

A standard

# 12 LEAD EKG

Does NOT show the

# RIGHT VENTRICLE

To see the

RIGHT VENTRICLE . . .

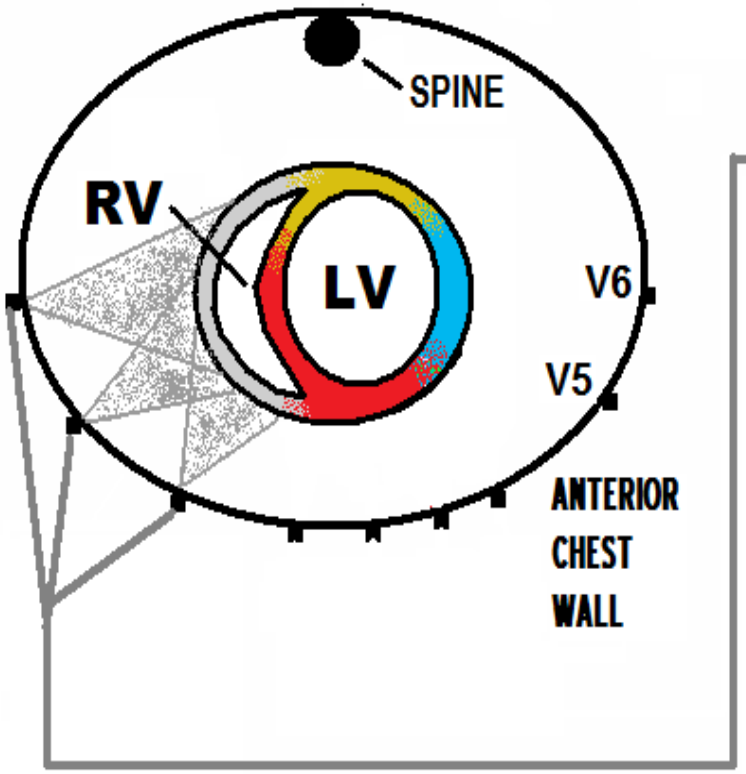
. . . such as in cases of

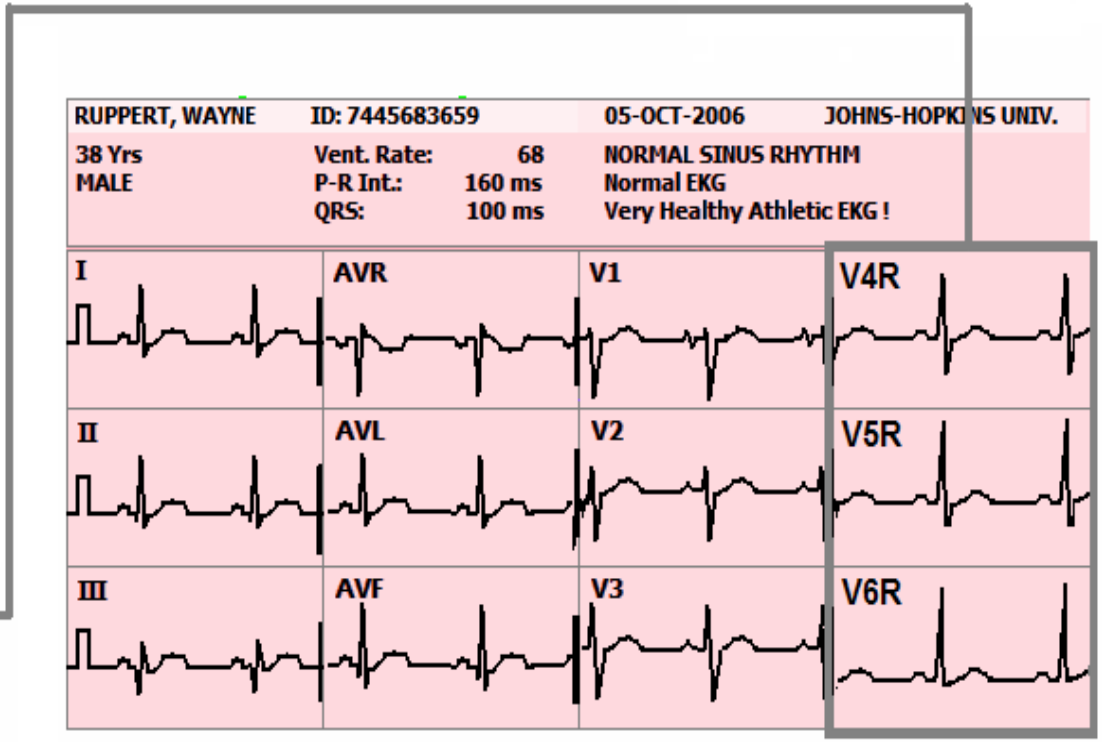
INFERIOR WALL M.I.

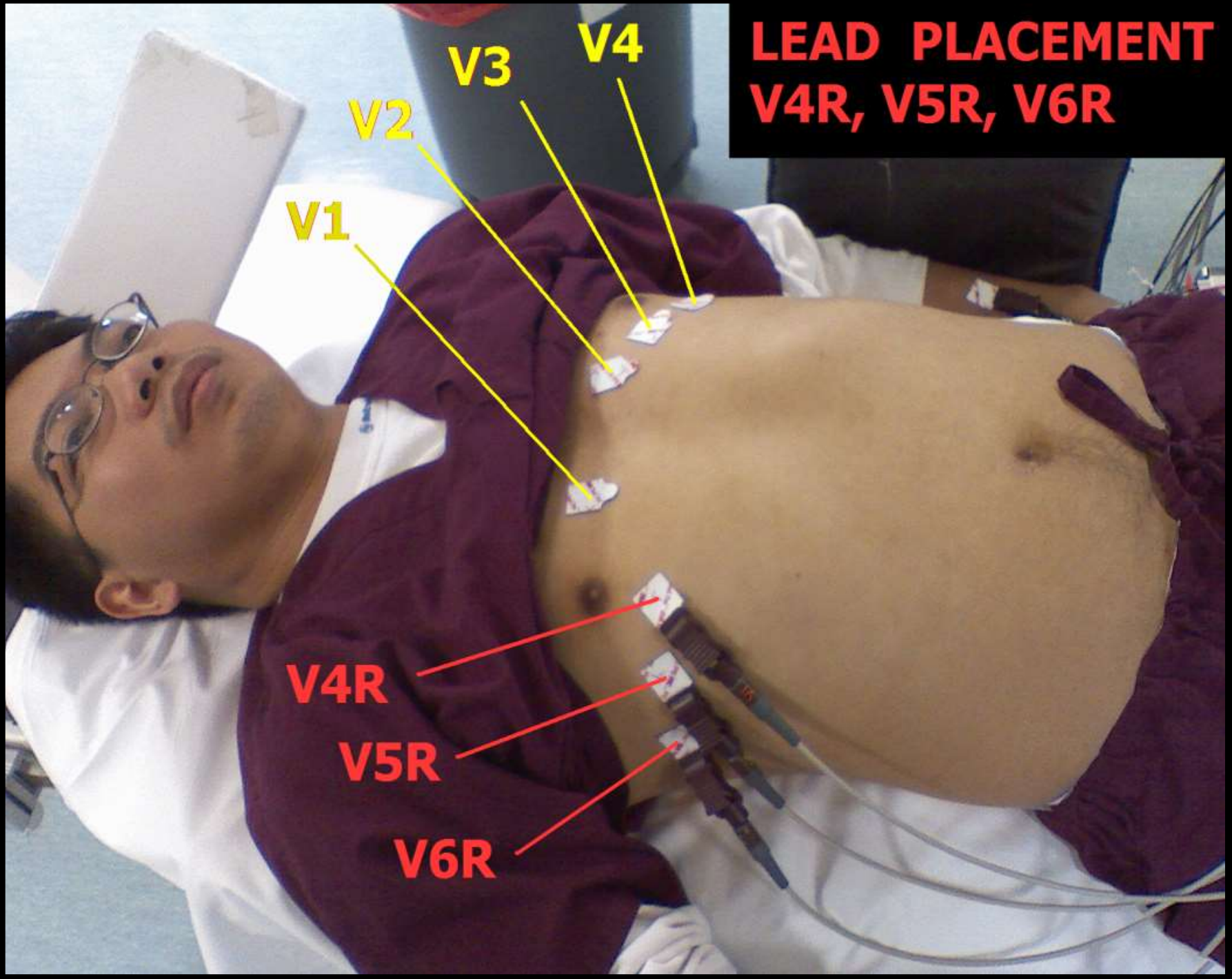
☞ You must do a

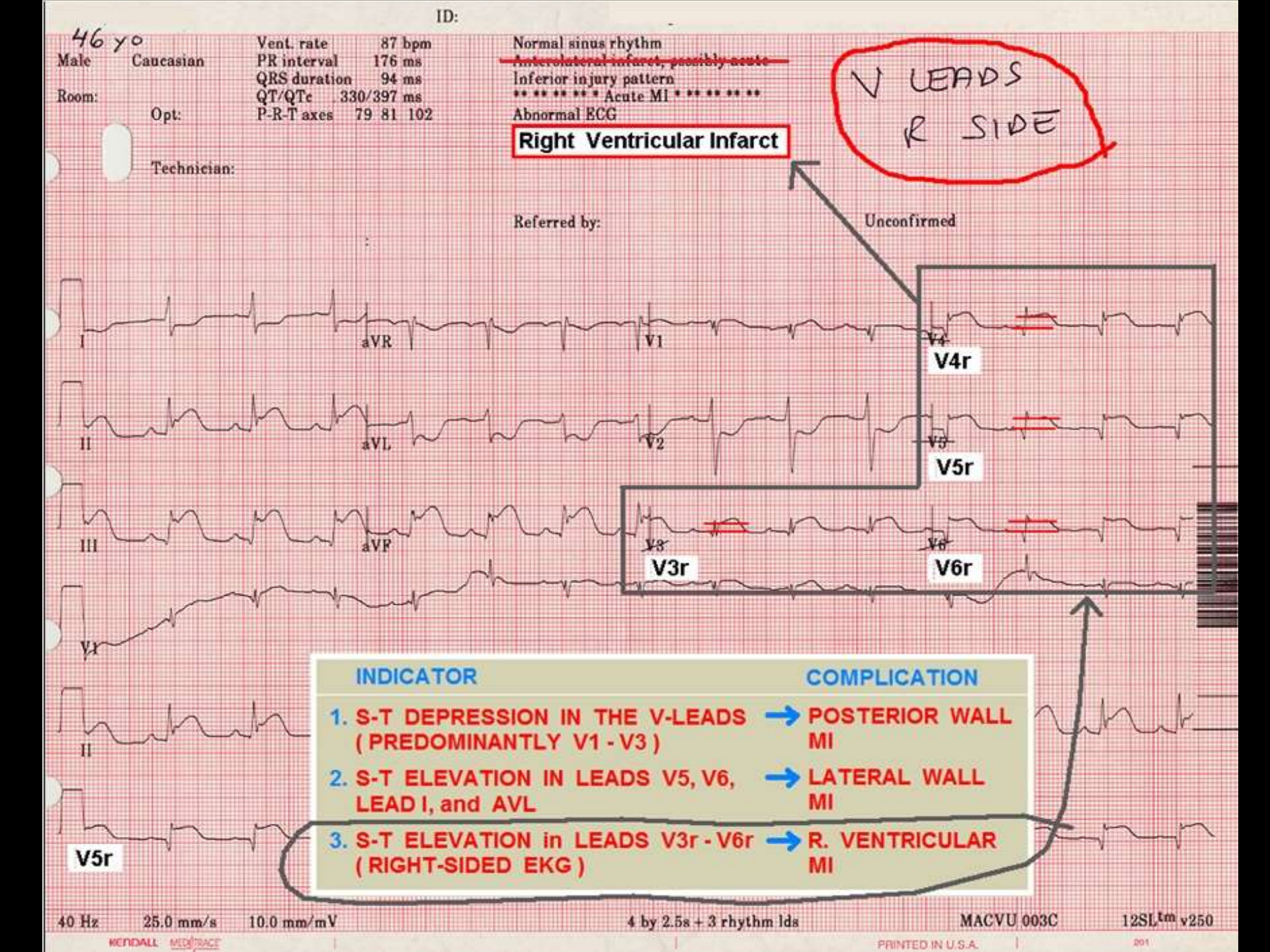
RIGHT - SIDED EKG ! !

# V4R - V6R VIEW THE RIGHT VENTRICLE



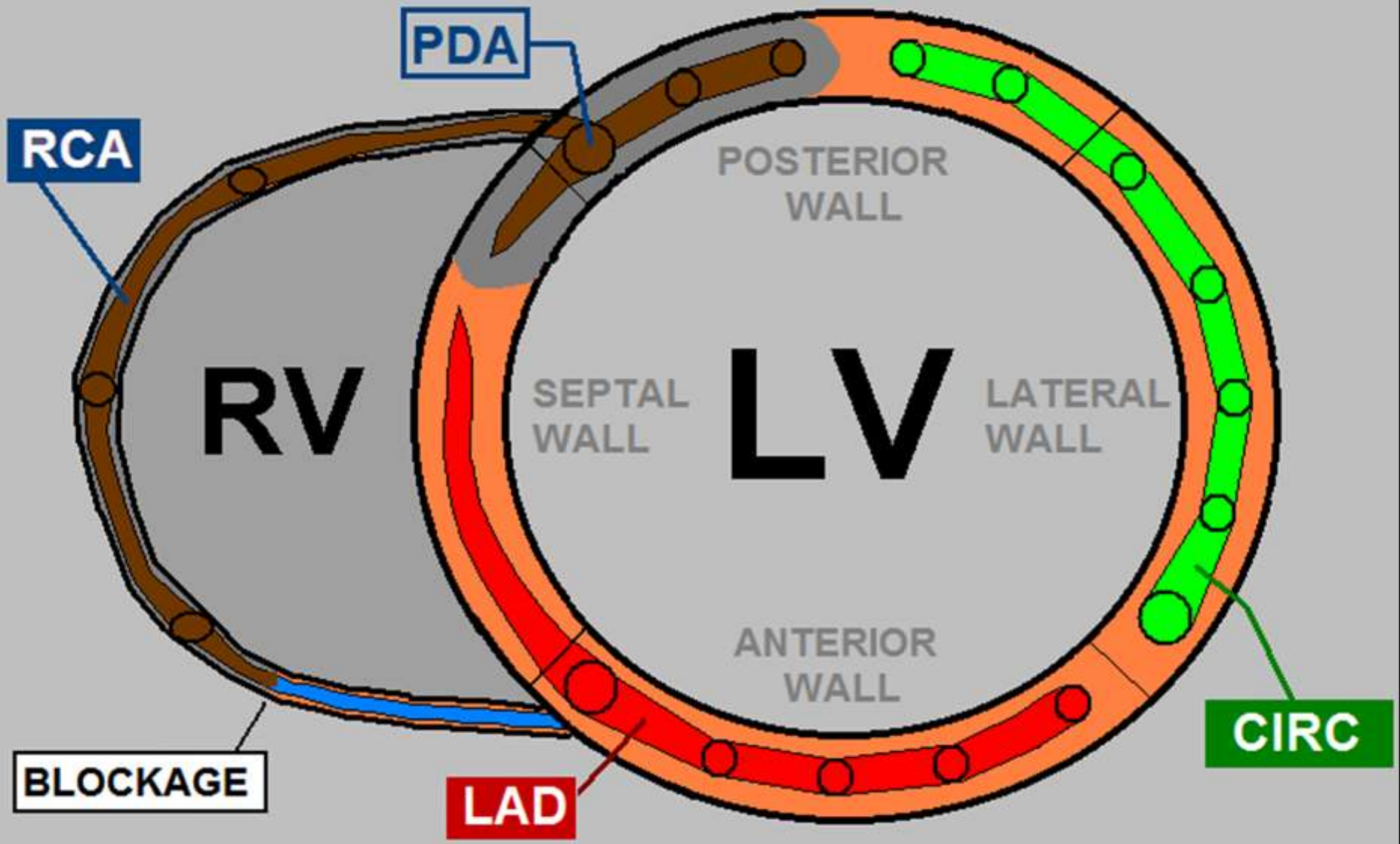

LEAD PLACEMENT
V4R, V5R, V6R
V1
V2
V3
V4
V4R
V5R
V6R


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
Inferior injury pattern
** ** ** ** * Acute MI * ** ** ** **
Abnormal ECG
Right Ventricular Infarct
V LEADS
R SIDE
Referred by:
Unconfirmed
I
aVR
V1
V4
V4r
II
aVL
V2
V5
V5r
III
aVF
V3
V3r
V6
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
12SL tm v250
KENDALL MEDITRACE
PRINTED IN U.S.A.

# INFERIOR - RIGHT VENTRICULAR MI

## DOMINANT RCA

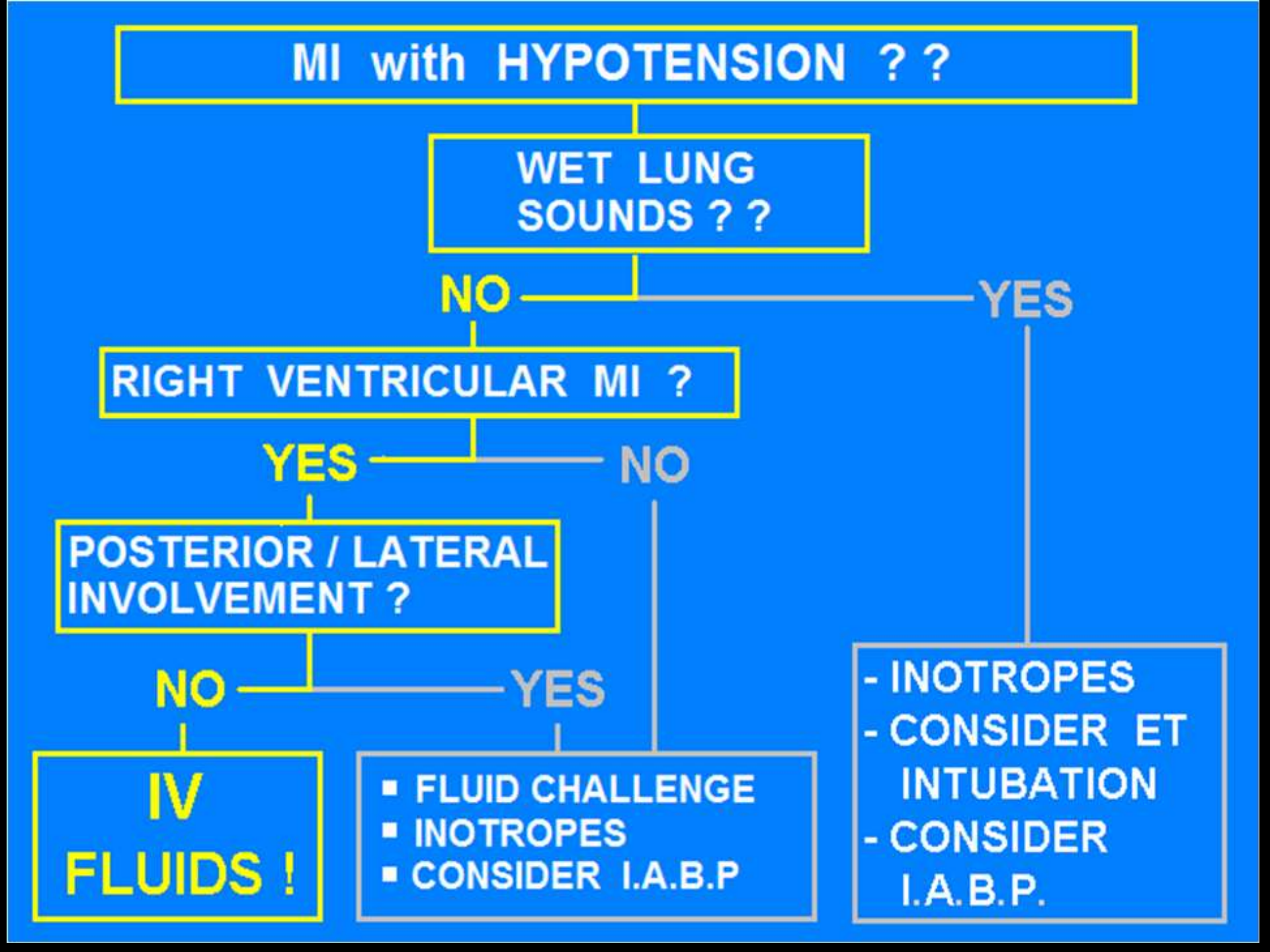
75-80 % of POPULATION

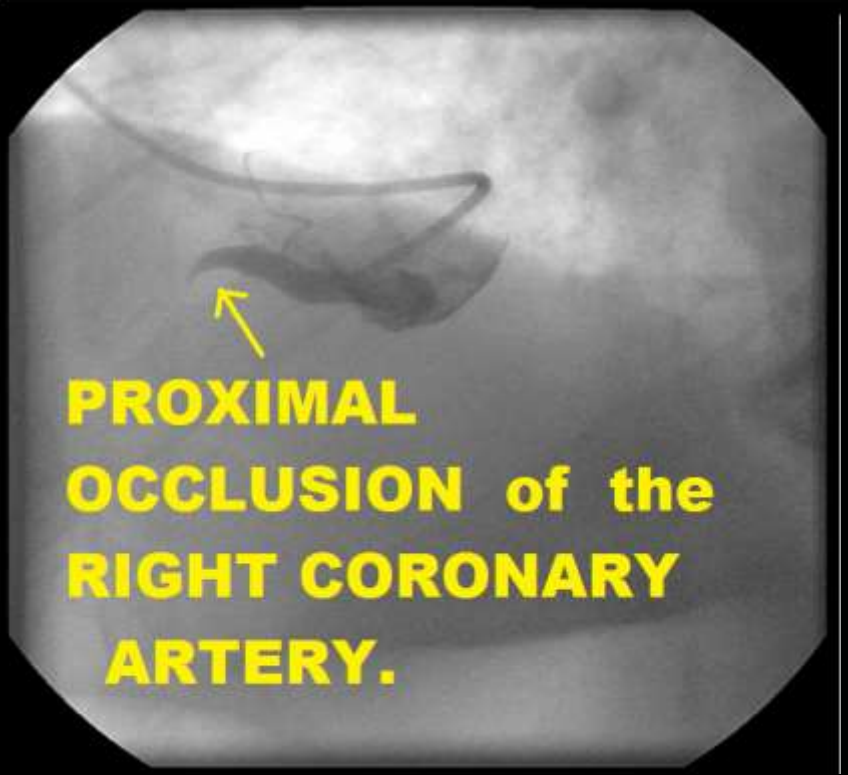


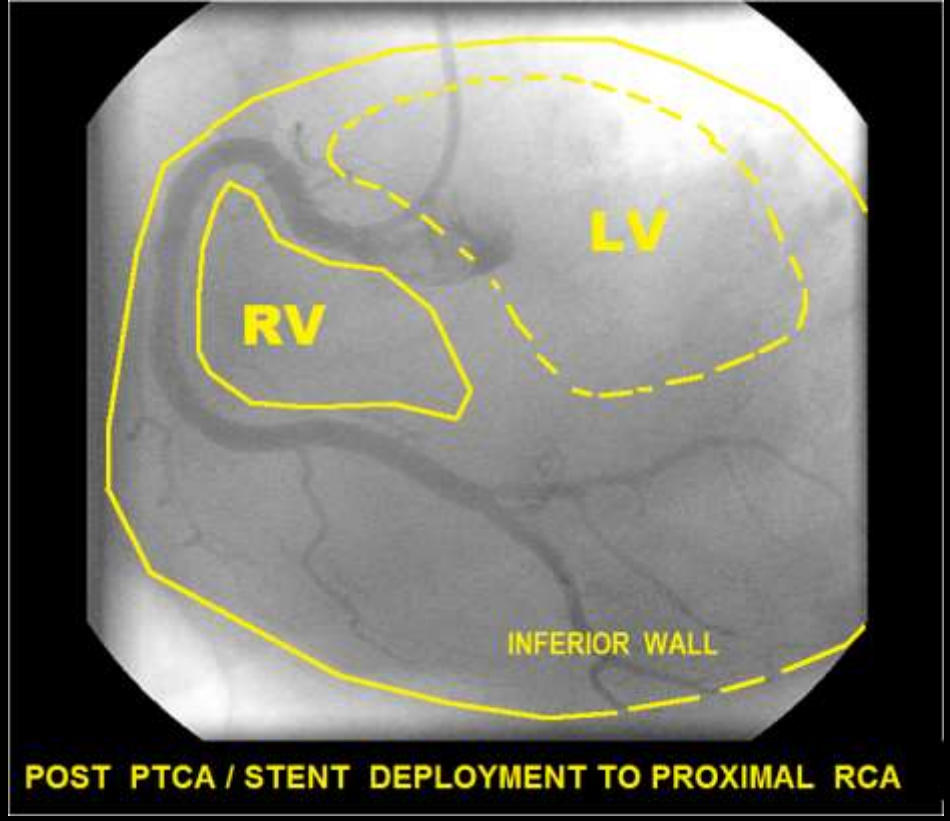
# ANTICIPATED COMPLICATIONS of INFERIOR WALL STEMI secondary to RCA Occlusion & POSSIBLE INDICATED INTERVENTIONS:

| | |
|---|---|
| - 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.<br>- You must do a RIGHT-SIDED ECG to see if RV MI is present.<br>- 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.<br>- 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**

# Evolving MI

# INFARCTION


AS MYOCARDIAL CELLS BECOME NECROTIC ---

IN THE LIMB LEADS:

- Q WAVES BEGIN TO DEVELOP
- S-T SEGMENTS BEGIN TO RETURN TO THE ISO-ELECTRIC LINE . . . .

23-JUL-2002 18:50:42 ST. JOSEPH'S HOSPITAL-ER ROUTINE RETRIEVAL

41 yr
Female Caucasian

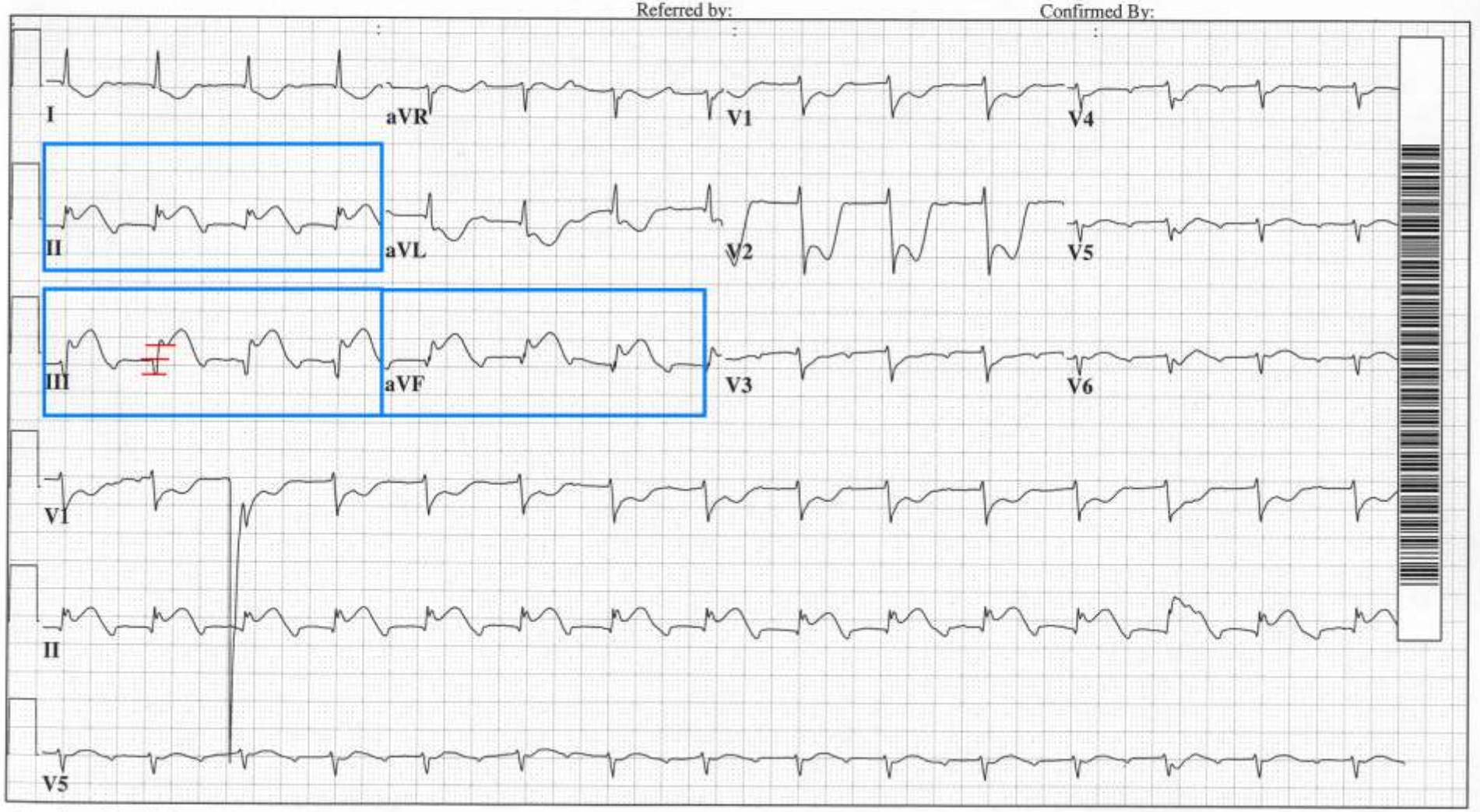
Room:ATL
Loc:3 Option:23

| | | |
|---|---|---|
| Vent. rate | 88 | BPM |
| PR interval | 308 | ms |
| QRS duration | 80 | ms |
| QT/QTc | 332/401 | ms |
| P-R-T axes | -108 33 | 112 |

**EKG CLASS #WR03882294**

**UNEDITED COPY - REPORT IS COMPUTER GENERATED ONLY, WITHOUT PHYSICIAN INTERPRETATION
Demand pacemaker; interpretation is based on intrinsic rhythm
Unusual P axis, possible ectopic atrial rhythm with 1st degree A-V block with occasional Premature ventricular complexes
Anterolateral infarct , age undetermined
Inferior injury pattern
** ** ** ** * ACUTE MI * ** ** ** **
Abnormal ECG ...

Referred by: Confirmed By:




25mm/s 10mm/mV 40Hz 005C 12SL 250 CID: 11

EID:13 EDT: 20:29 23-JUL-2002 ORDER:

23–JUL–2002 19:00:54 ST. JOSEPH'S HOSPITAL–ER ROUTINE RETRIEVAL

41 yr
Female Caucasian

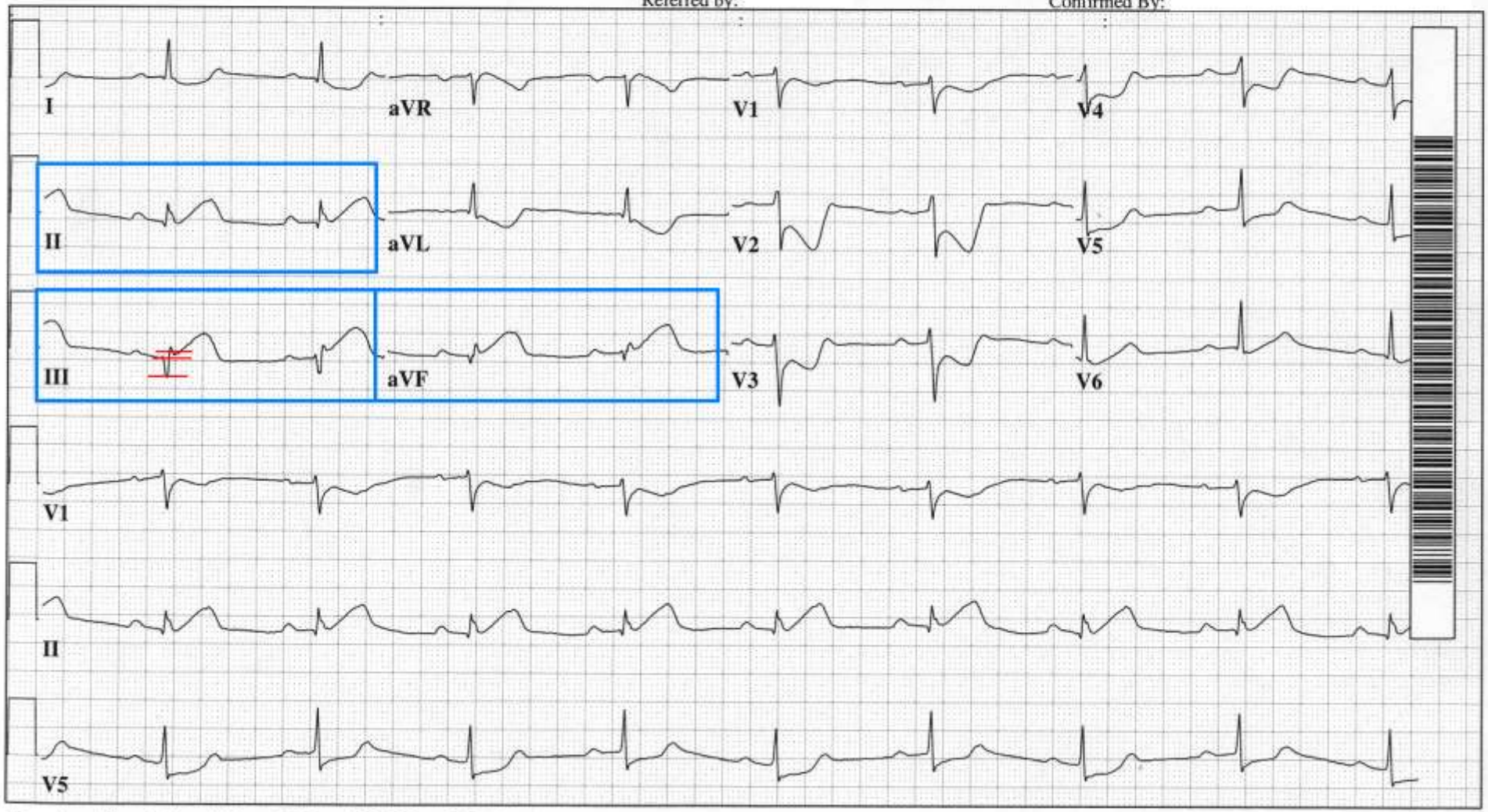
Room:ATL
Loc:3 Option:23

| | | |
|---|---|---|
| Vent. rate | 53 | BPM |
| PR interval | 236 | ms |
| QRS duration | 84 | ms |
| QT/QTc | 458/429 | ms |
| P–R–T axes | 60 14 | 94 |

**EKG CLASS #WR03882294**

**UNEDITED COPY – REPORT IS COMPUTER GENERATED ONLY, WITHOUT PHYSICIAN INTERPRETATION
Sinus bradycardia with 1st degree A–V block
Inferior–posterior infarct , possibly acute
ST & T wave abnormality, consider lateral ischemia
** ** ** ** * ACUTE MI * ** ** ** **
Abnormal ECG
When compared with ECG of 23–JUL–2002 18:50,
MANUAL COMPARISON REQUIRED, DATA IS UNCONFIRMED

Referred by: Confirmed By:




25mm/s 10mm/mV 40Hz 005C 12SL 250 CID: 11

EID:13 EDT: 20:29 23–JUL–2002 ORDER:

23-JUL-2002 22:17:35 ST. JOSEPH'S HOSPITAL-CCU ROUTINE RETRIEVAL

41 yr
Female Caucasian

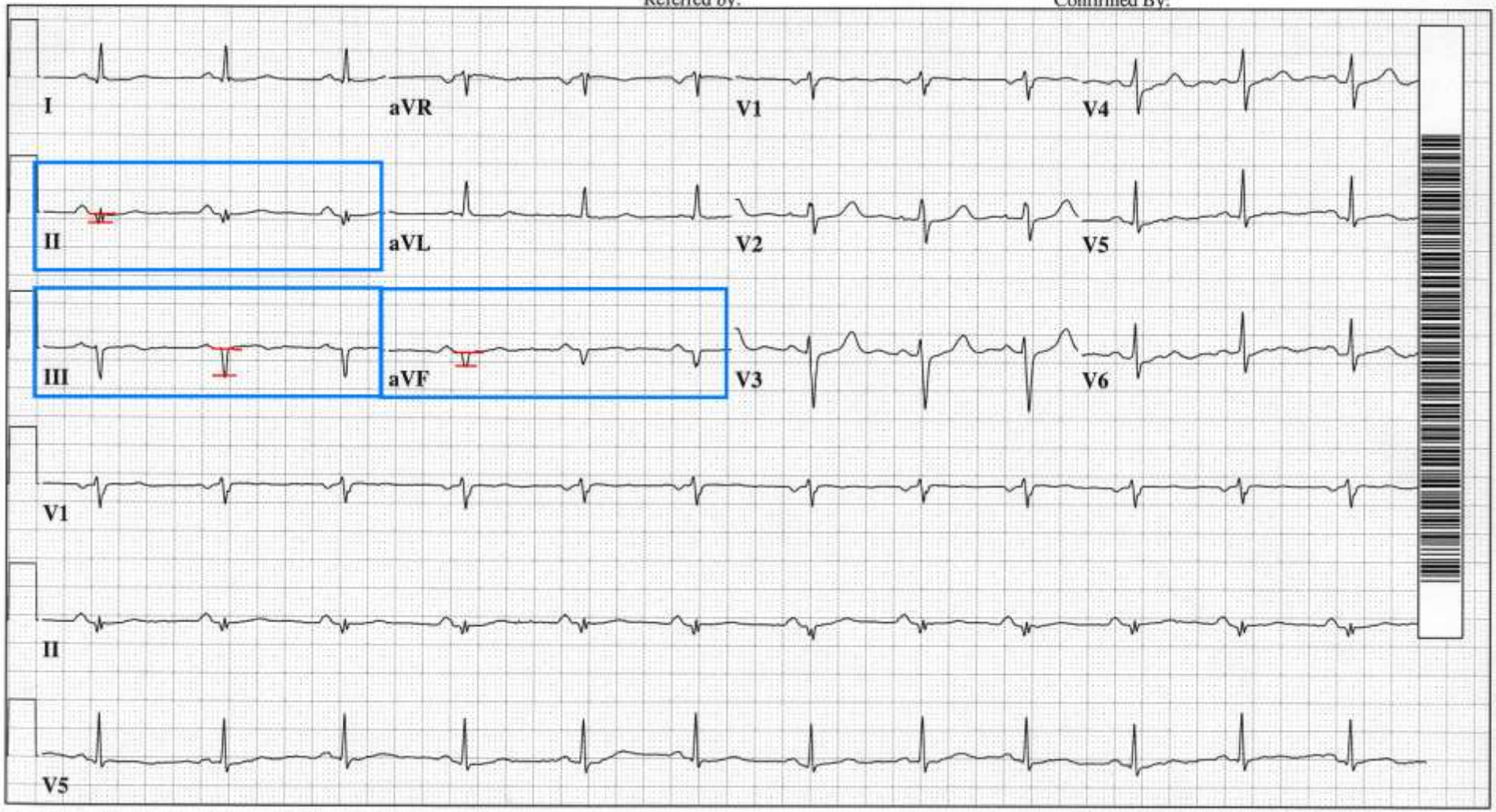
Room:CCU
Loc:1 Option:1

| | | |
|---|---|---|
| Vent. rate | 73 | BPM |
| PR interval | 150 | ms |
| QRS duration | 88 | ms |
| QT/QTc | 402/442 | ms |
| P-R-T axes | 58 -31 | 51 |

Normal sinus rhythm
Left axis deviation
Inferior infarct (cited on or before 23-JUL-2002)
Abnormal ECG
When compared with ECG of 23-JUL-2002 19:00,
PR interval has decreased
QRS axis Shifted left
Serial changes of evolving Inferior infarct Present

**EKG CLASS #WR03882294**

Referred by: Confirmed By:




25mm/s 10mm/mV 40Hz 005C 12SL 231 CID: 1

EID:11 EDT: 10:47 25-JUL-2002 ORDER:

# CHANGES ASSOCIATED WITH CELLULAR PERFUSION INVOLVING THE:

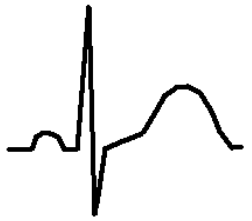
- QRS
- J POINT
- ST SEGMENT
- T WAVE

CARDIAC CELL PERFUSION STATES

## NORMAL STATE OF PERFUSION

ARTERIAL BLOCKAGES → NONE SIGNIFICANT
CELLULAR OXYGENATION → NORMAL
CELLULAR METABOLISM → AEROBIC
CELLULAR FUNCTION → NORMAL CONTRACTION

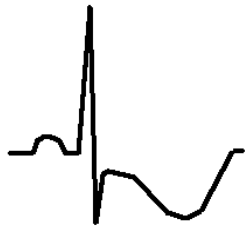
EKG: J POINT ISOELECTRIC, ST SEG "SLIGHT, POSTIVE INCLINATION, T WAVE POSITIVE, UPRIGHT.



## ISCHEMIA

ARTERIAL BLOCKAGES → PARTIAL OBSTRUCTION
CELLULAR OXYGENATION → INSUFFICIENT
CELLULAR METABOLISM → AEROBIC
CELLULAR FUNCTION → REDUCED CONTRACTION
PATIENT SYMPTOMS → *POSSIBLE*, WITH EXERTION

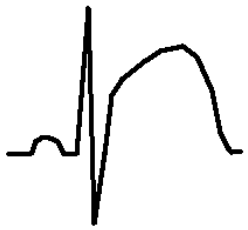
EKG: J POINT DEPRESSED, ST SEGMENT VARIES, T WAVE VARIES



## INFARCTION

ARTERIAL BLOCKAGES → TOTAL OBSTRUCTION
CELLULAR OXYGENATION → NONE
CELLULAR METABOLISM → ANAEROBIC *CELL BEGINS TO BURN GLYCOGEN RESERVES*
CELLULAR FUNCTION → STOPS CONTRACTING
PATIENT SYMPTOMS → *TYPICAL or ATYPICAL ACS Sx*

EKG - INDICATIVE: J POINT ELEVATES, ST SEGMENT CONVEX, T WAVE POSITIVE, MAY ENLARGE
EKG - RECIPROCAL: J POINT DEPRESSES, ST SEGMENT DOWNSLOPING, T WAVE INVERTED



## NECROSIS

ARTERIAL BLOCKAGES → TOTAL OBSTRUCTION
CELLULAR OXYGENATION → NONE
CELLULAR METABOLISM → *CELL DIES WHEN GLYCOGEN RESERVES DEPLETED.*
CELLULAR FUNCTION → NONE. CELL DEAD.
PATIENT SYMPTOMS → *POSS. HYPOTENSION, DEATH*

EKG - INDICATIVE: J POINTS, ST SEGMENTS NORMALIZE; ABNORMAL Q WAVES FORM
EKG - RECIPROCAL: J POINTS, ST SEGMENTS NORMALIZE; ABNORMAL TALL R WAVES FORM

**When a patient has an INFERIOR WALL STEMI With RIGHT VENTRICULAR involvement . . . .**

**If reperfusion is DELAYED, and NECROSIS forms . . . .**

**Do SIGNIFICANT Q WAVES form in the RIGHT VENTRICULAR LEADS ? ? ?**

64 yr Male Caucasian

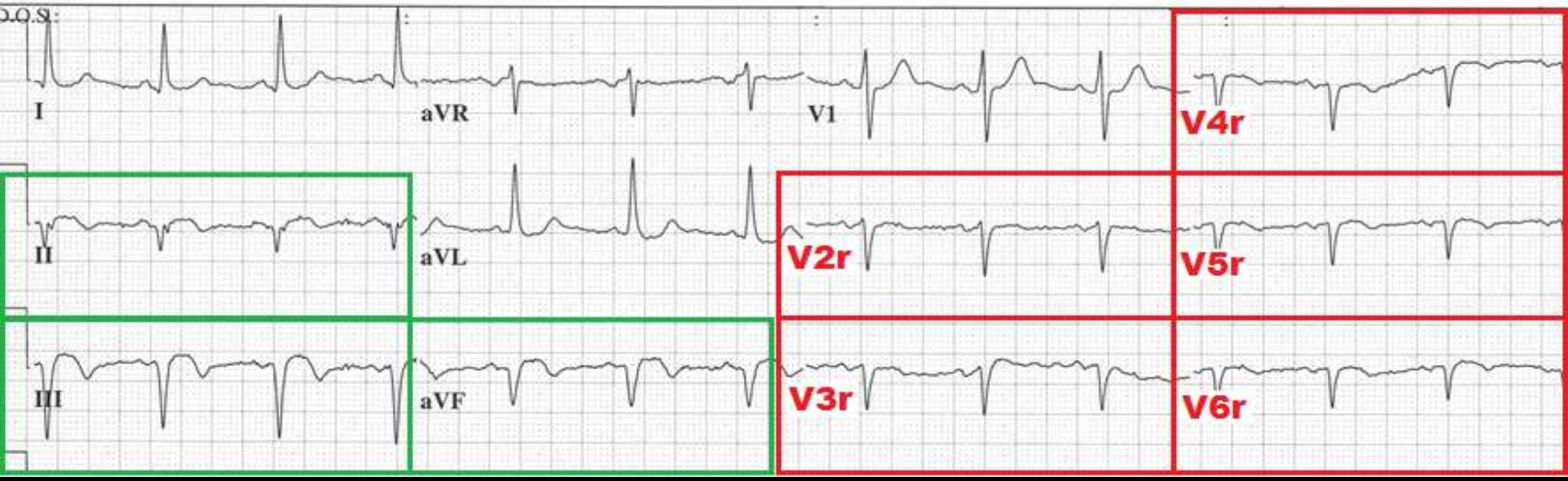
Loc:3 Option:23

| | | |
|---|---|---|
| Vent. rate | 79 | BPM |
| PR interval | 136 | ms |
| QRS duration | 92 | ms |
| QT/QTc | 350/401 | ms |
| P–R–T axes | 42 –41 | –3 |

**ECG LEADS PLACED ON RIGHT CHEST WALL.**

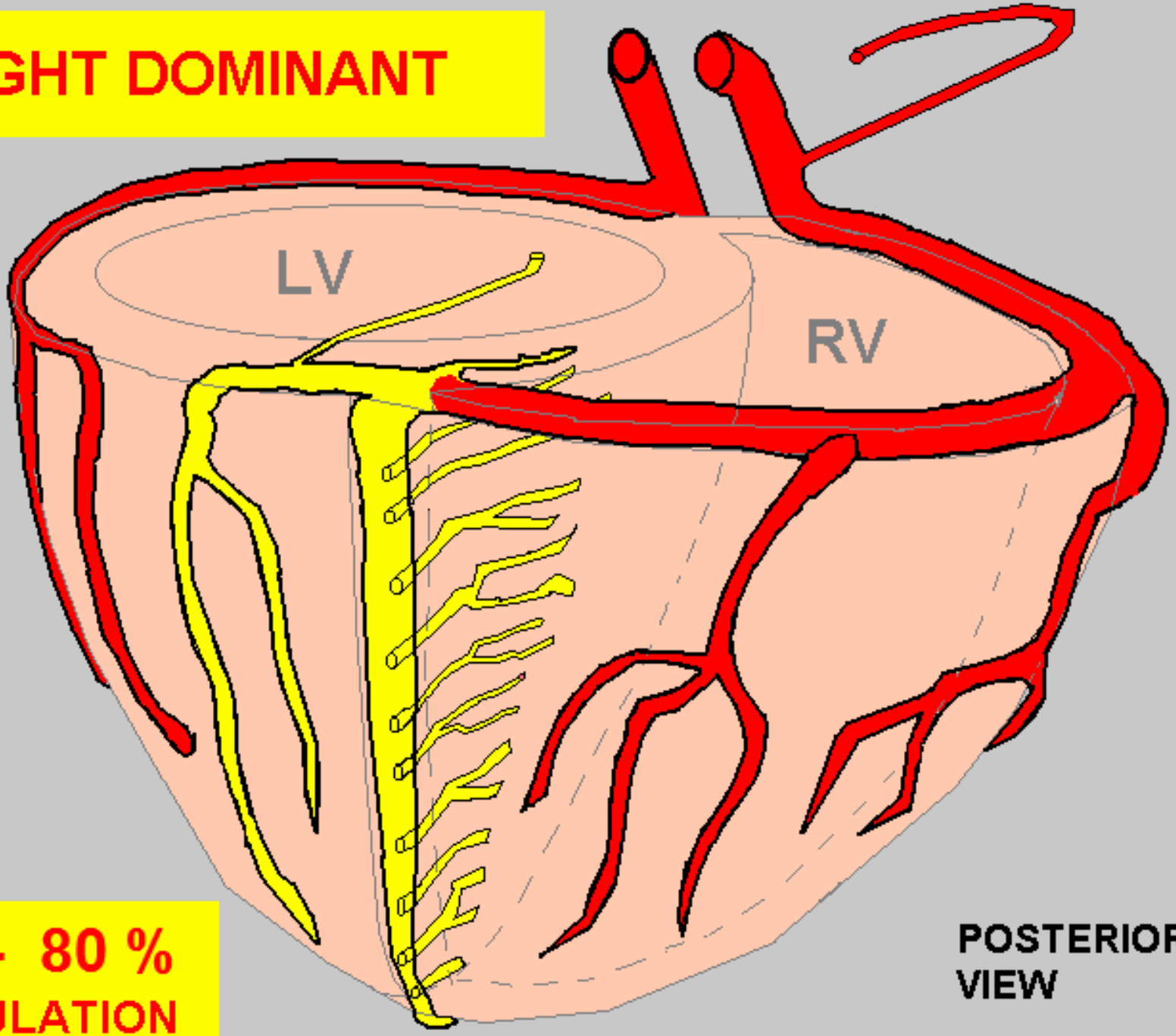
## ECG INDICATORS of EVOLVING INFERIOR - RIGHT VENTRICULAR MYOCARDIAL INFARCTION:

- QS COMPLEXES LEADS II, III, aVF
- QS COMPLEXES LEADS V2r - V6r




# YES !

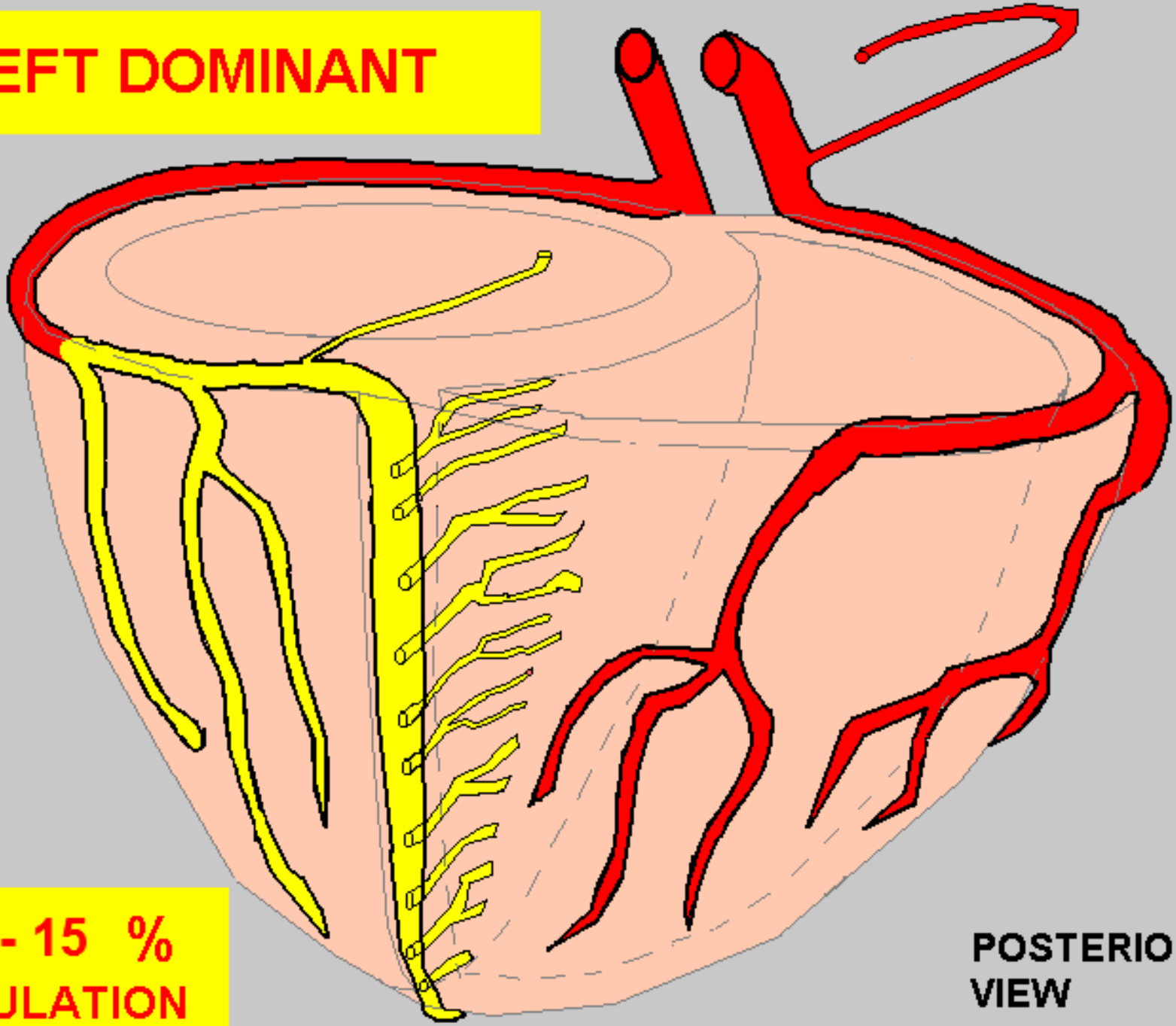
# RIGHT DOMINANT




**75 - 80 %**
**POPULATION**

**POSTERIOR VIEW**

# LEFT DOMINANT



10 - 15 %
POPULATION

POSTERIOR
VIEW

# 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

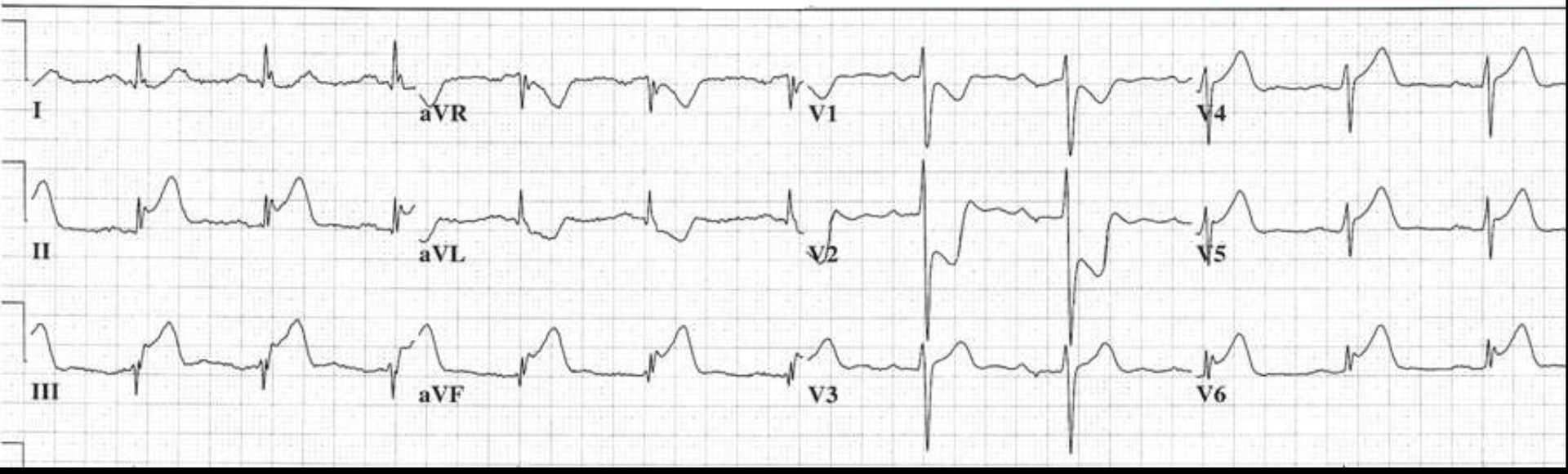
# SHOCK ASSESSMENT

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

| | | | | |
|---|---|---|---|---|
| 42 yr | | Vent. rate | 69 | BPM |
| Male | Caucasian | 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:** |

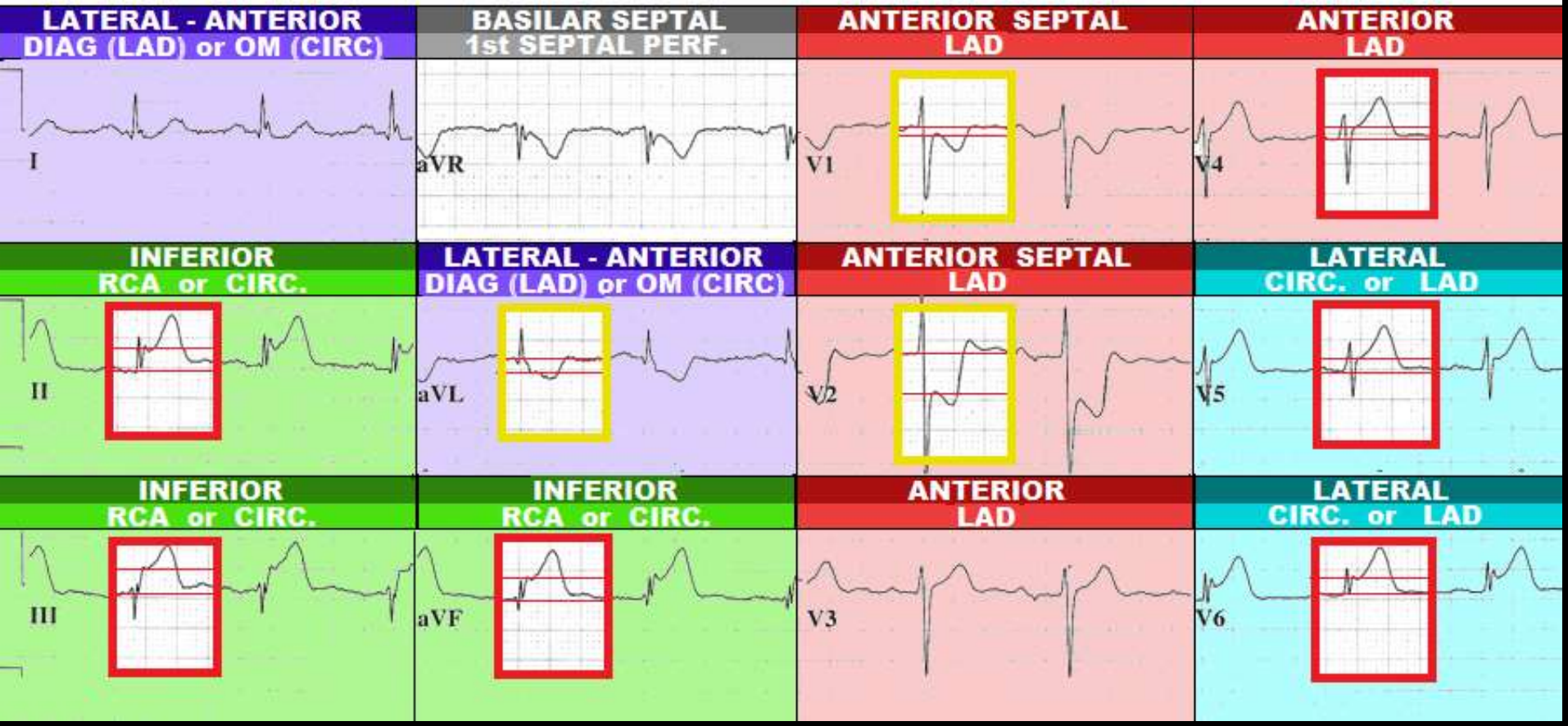
42 yr
Male
Caucasian

Loc:3
Option:23

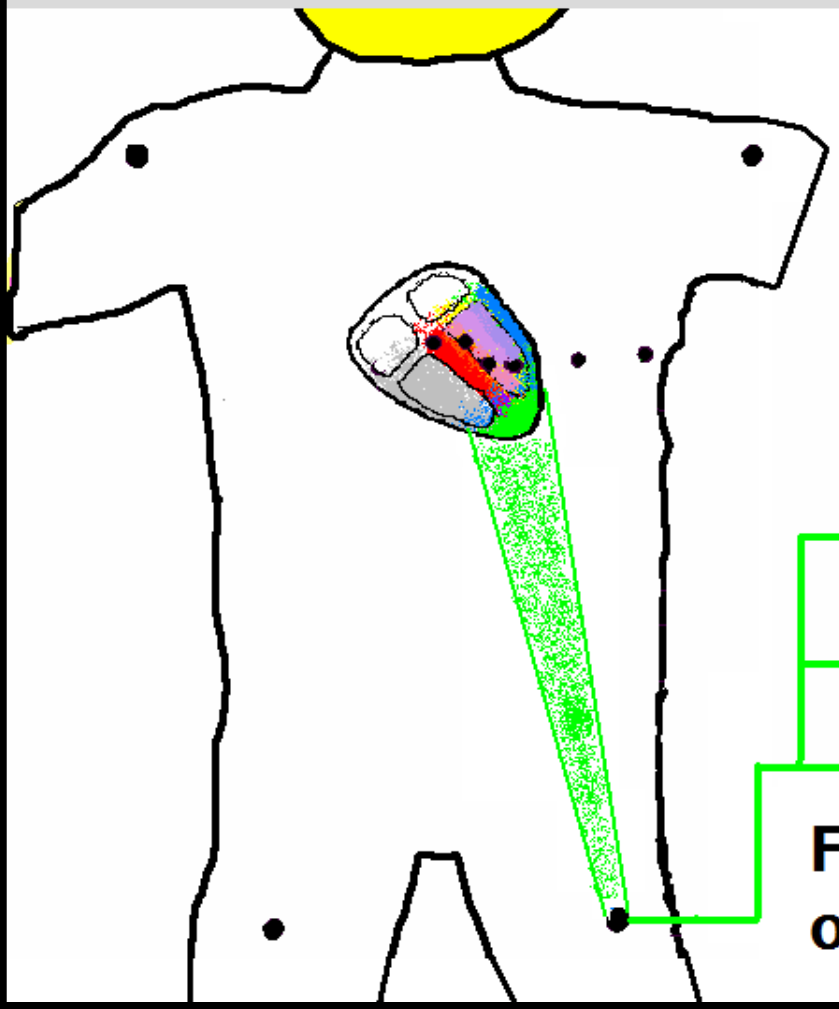
| | | |
|---|---|---|
| Vent. rate | 69 | BPM |
| PR interval | 196 | ms |
| QRS duration | 98 | ms |
| QT/QTc | 388/415 | ms |
| P-R-T axes | 14 28 | 81 |

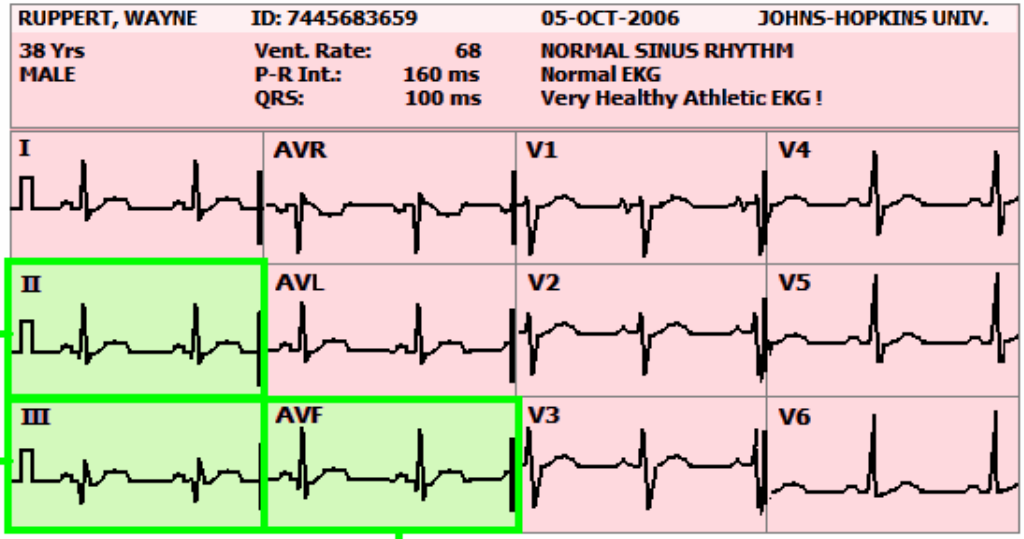
*** Acute MI ***
Inferior-Posterior-Lateral Injury Pattern

ST SEGMENT ELEVATION
ST SEGMENT DEPRESSION



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






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

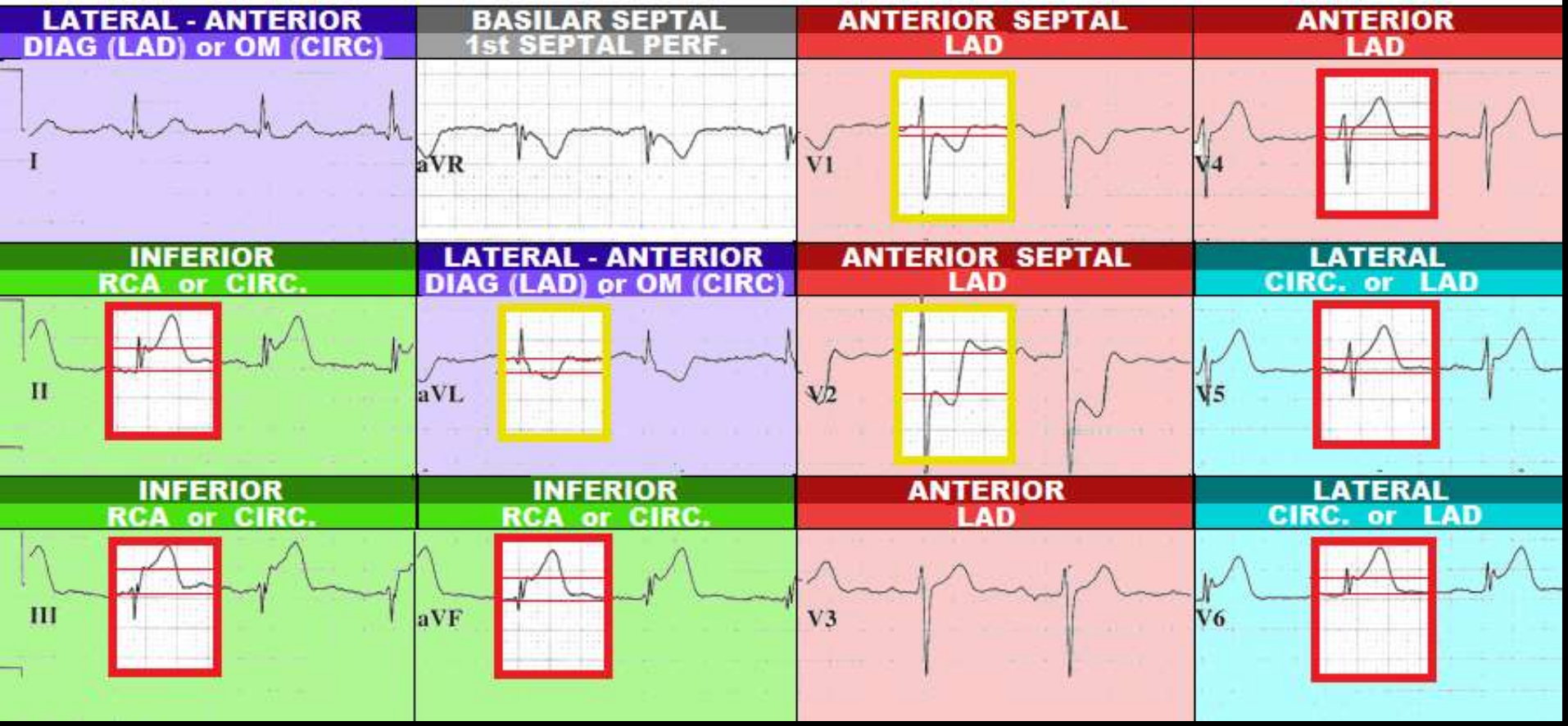
42 yr
Male
Caucasian

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 |

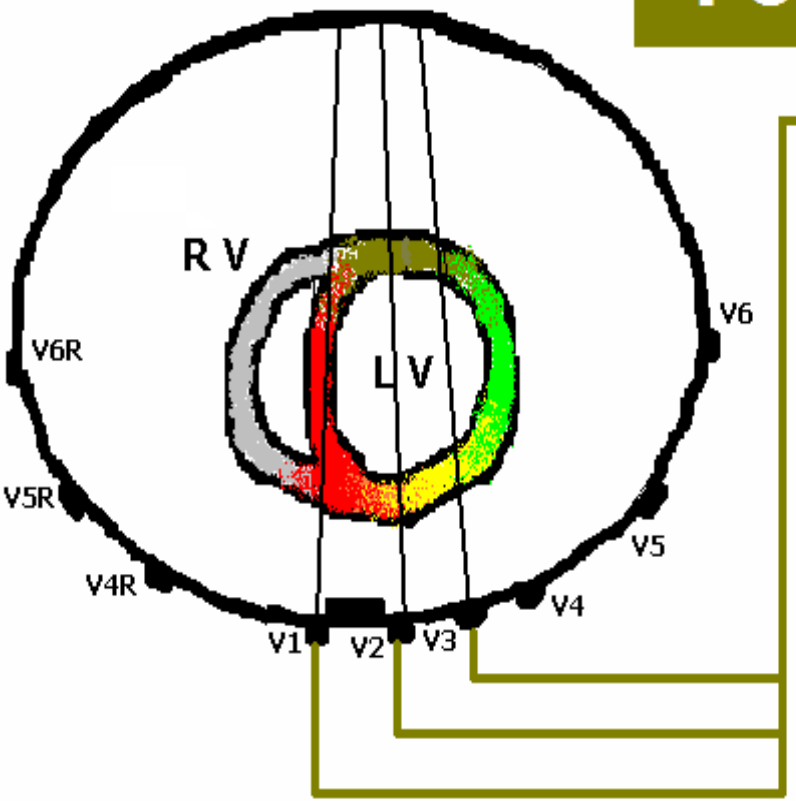
*** Acute MI ***
Inferior-Posterior-Lateral Injury Pattern

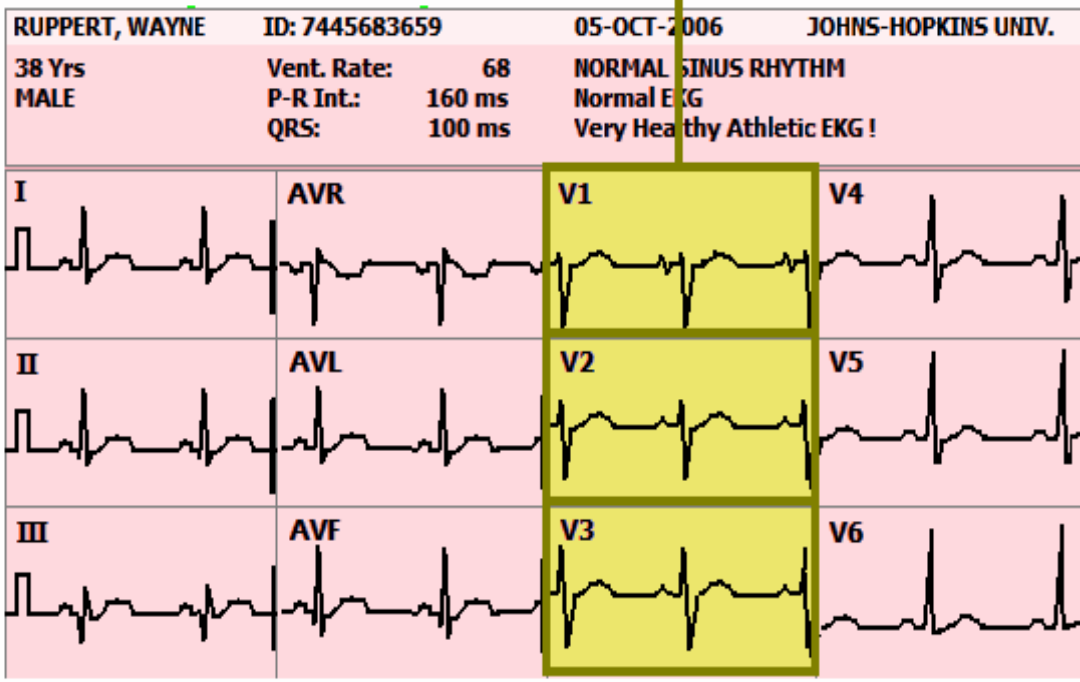
ST SEGMENT ELEVATION
ST SEGMENT DEPRESSION



# LEADS V1 - V3 *view the*

## POSTERIOR WALL



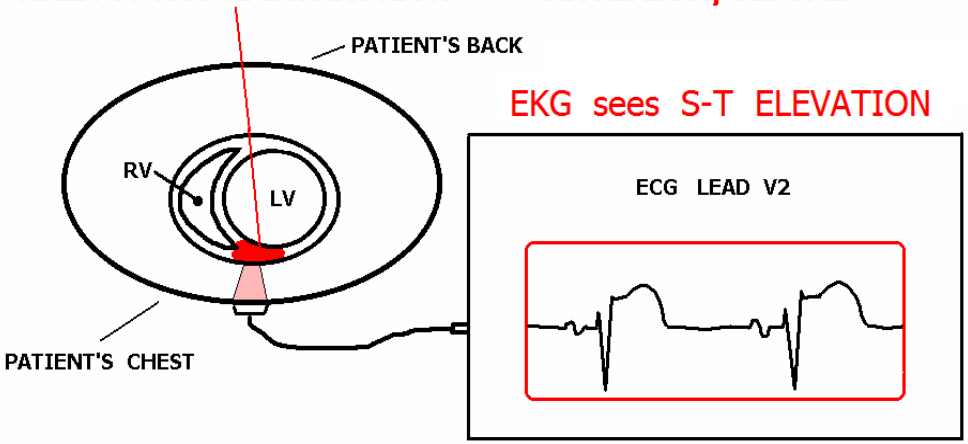




## *via* RECIPROCAL CHANGES.

# HOW EKG VIEWS INDICATIVE CHANGES

**EXAMPLE:**

**AREA OF ACUTE INFARCTION - ANTERIOR/SEPTAL**

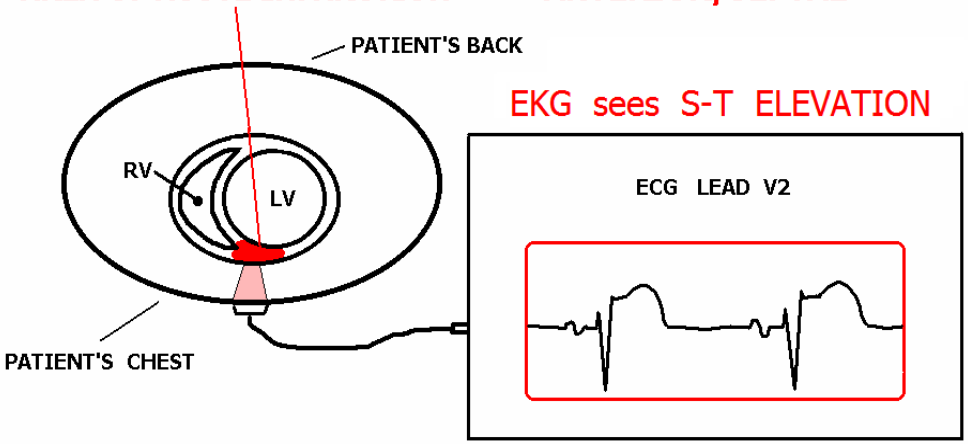



EKG sees S-T ELEVATION

## HOW EKG VIEWS INDICATIVE CHANGES

**EXAMPLE:**

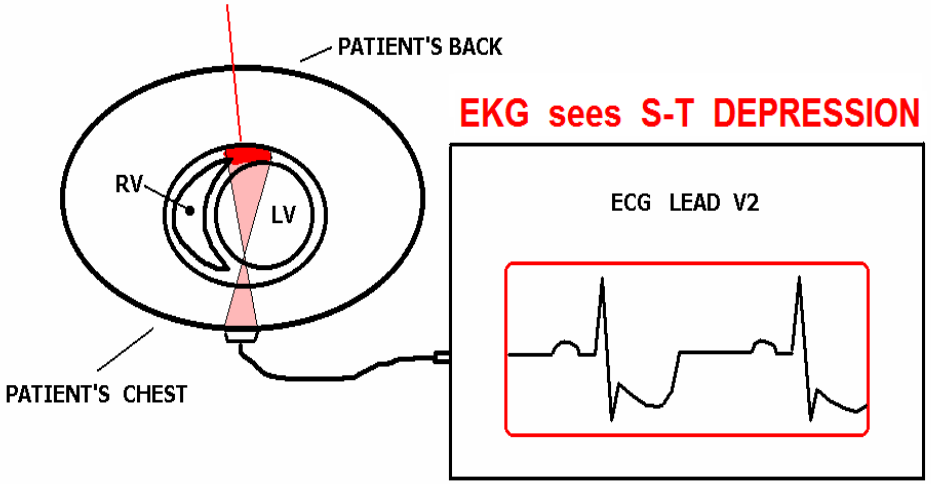
**AREA OF ACUTE INFARCTION - ANTERIOR/SEPTAL**




## HOW EKG VIEWS RECIPROCAL CHANGES

**EXAMPLE:**

**AREA OF ACUTE INFARCTION - POSTERIOR WALL**



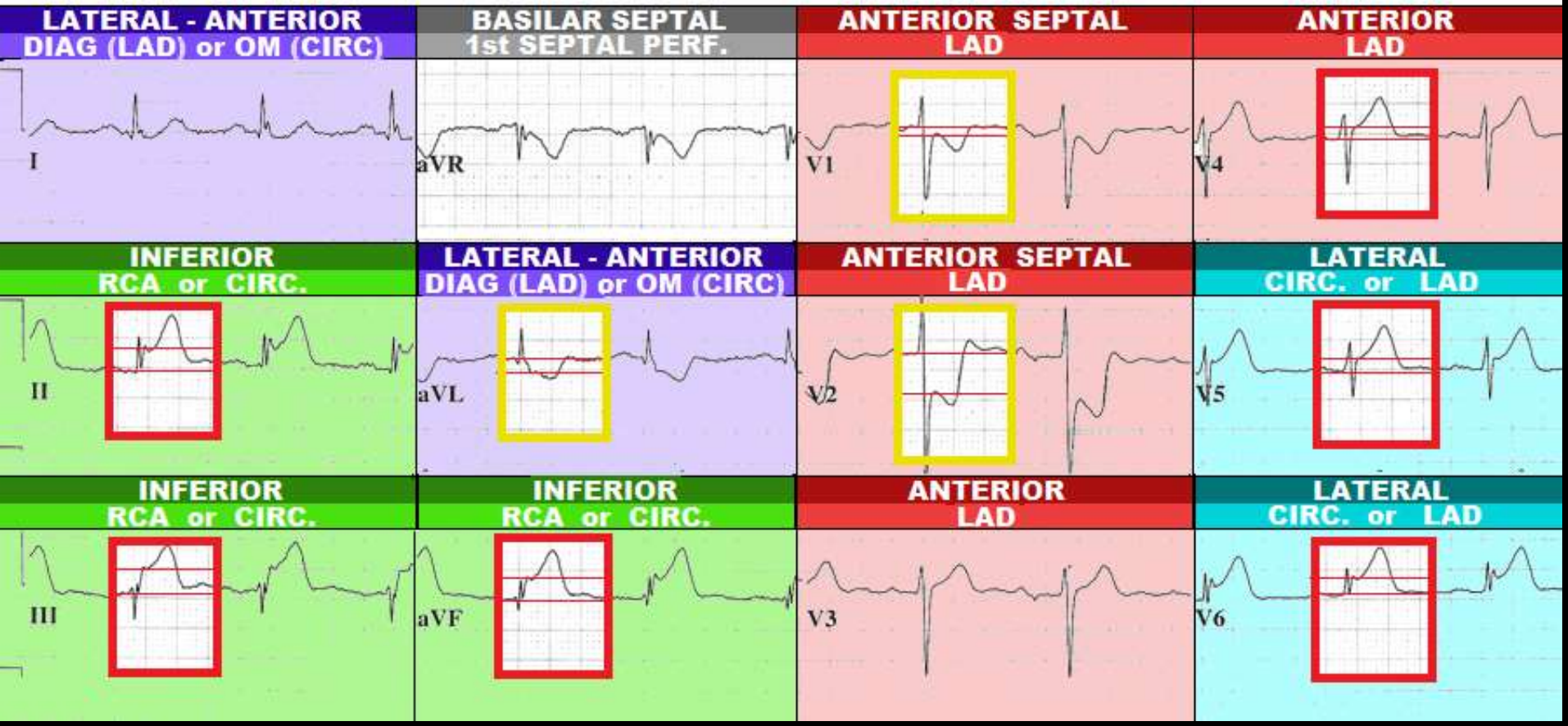
42 yr
Male
Caucasian

Loc:3
Option:23

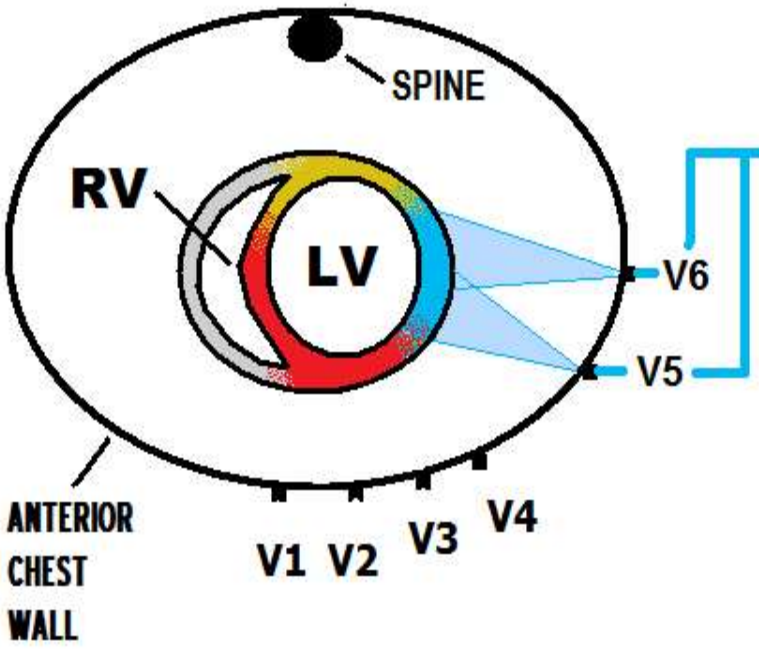
| | | |
|---|---|---|
| Vent. rate | 69 | BPM |
| PR interval | 196 | ms |
| QRS duration | 98 | ms |
| QT/QTc | 388/415 | ms |
| P-R-T axes | 14 28 | 81 |

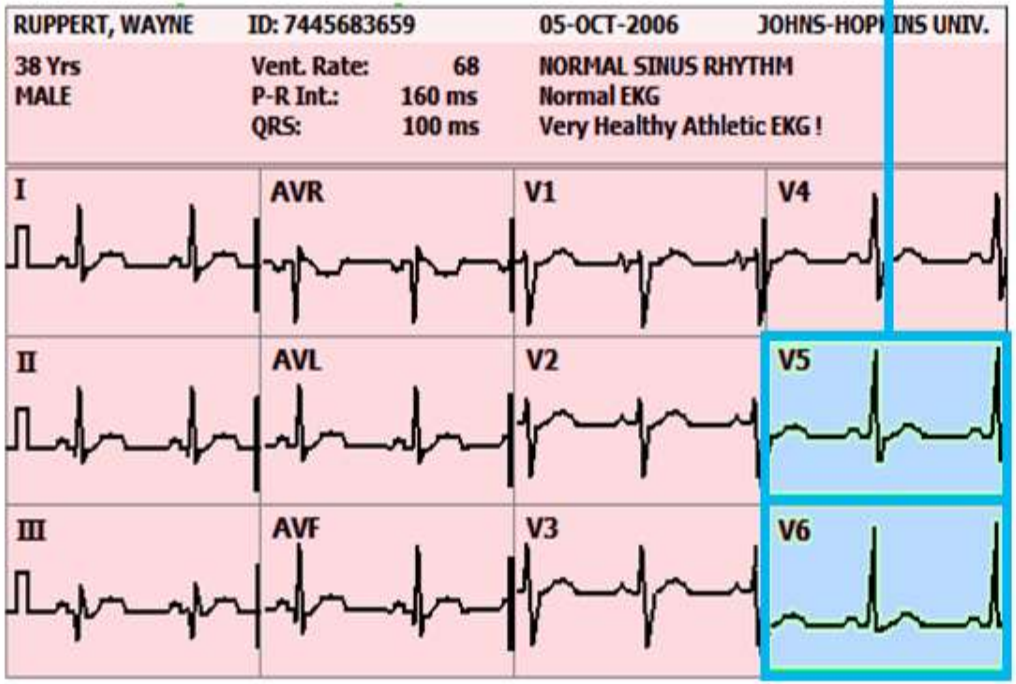
*** Acute MI ***
Inferior-Posterior-Lateral Injury Pattern

ST SEGMENT ELEVATION
ST SEGMENT DEPRESSION



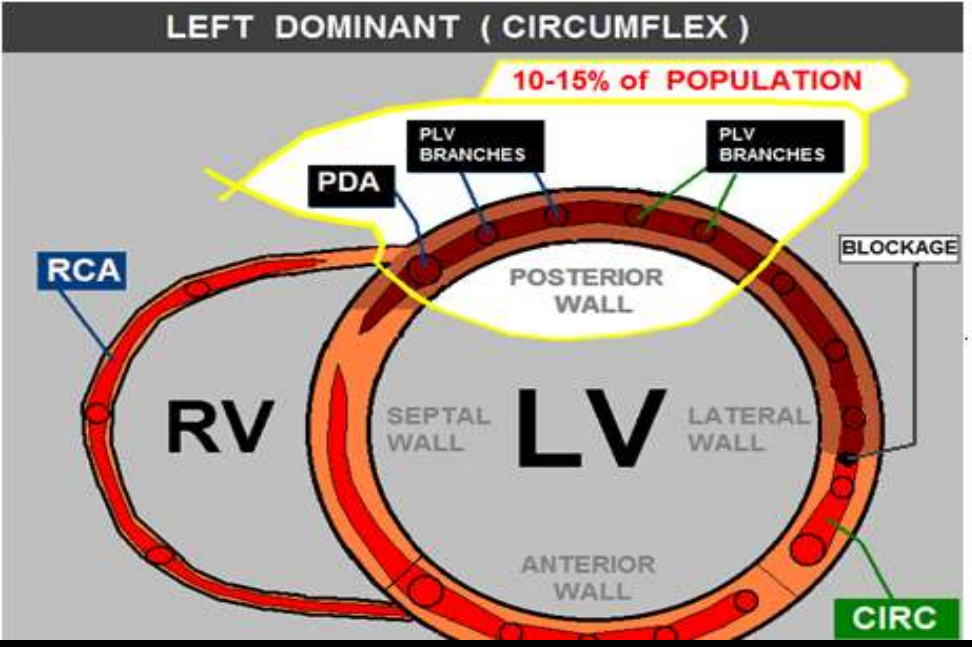
# V5 - V6 VIEW THE LATERAL WALL
## of the LEFT VENTRICLE

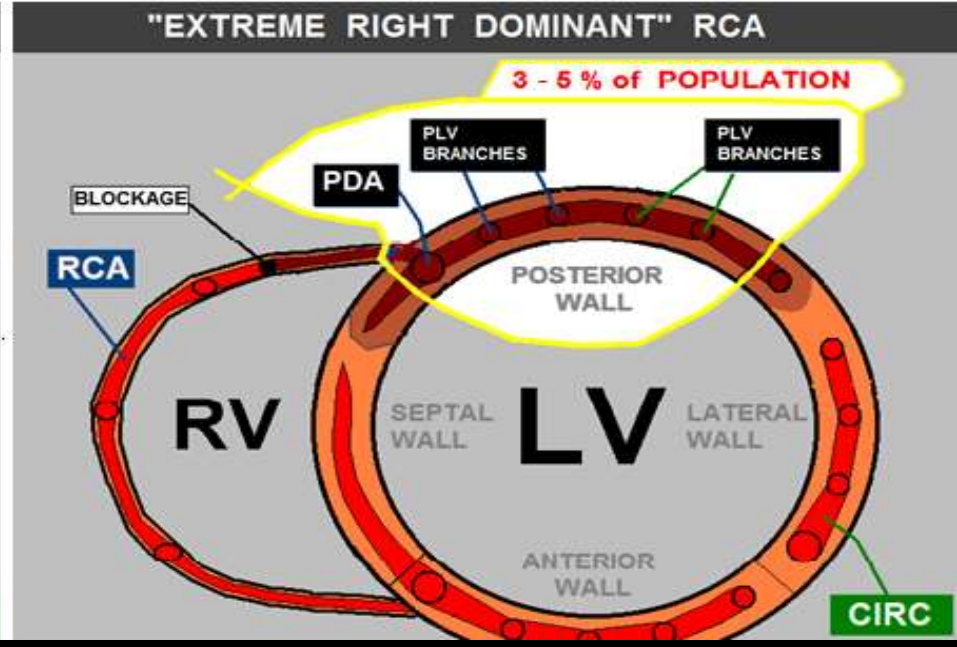


# INDICATIONS for 18 Lead ECG include:

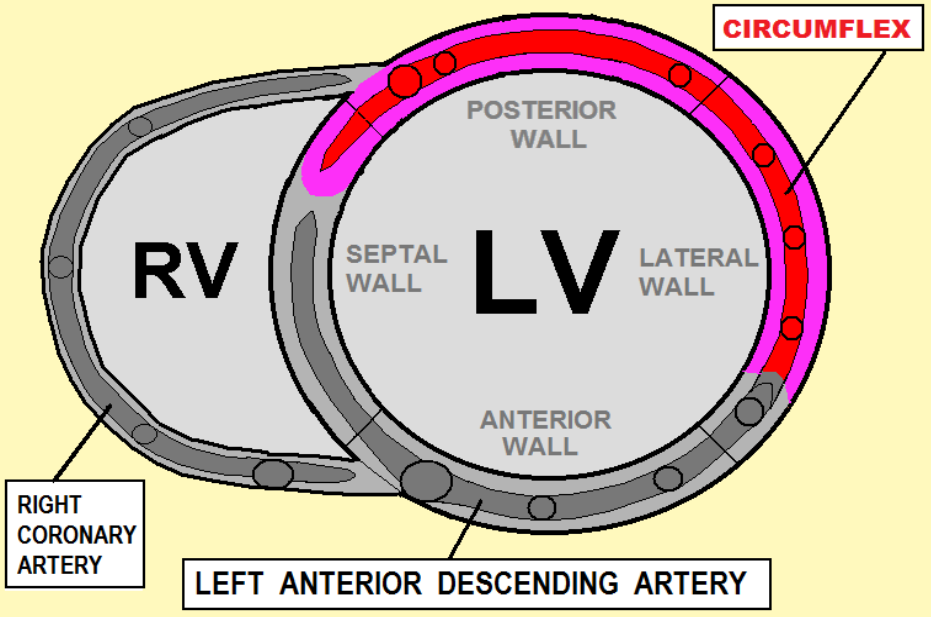
- INFERIOR WALL MI
- ST Depression in LEADS V1-V4



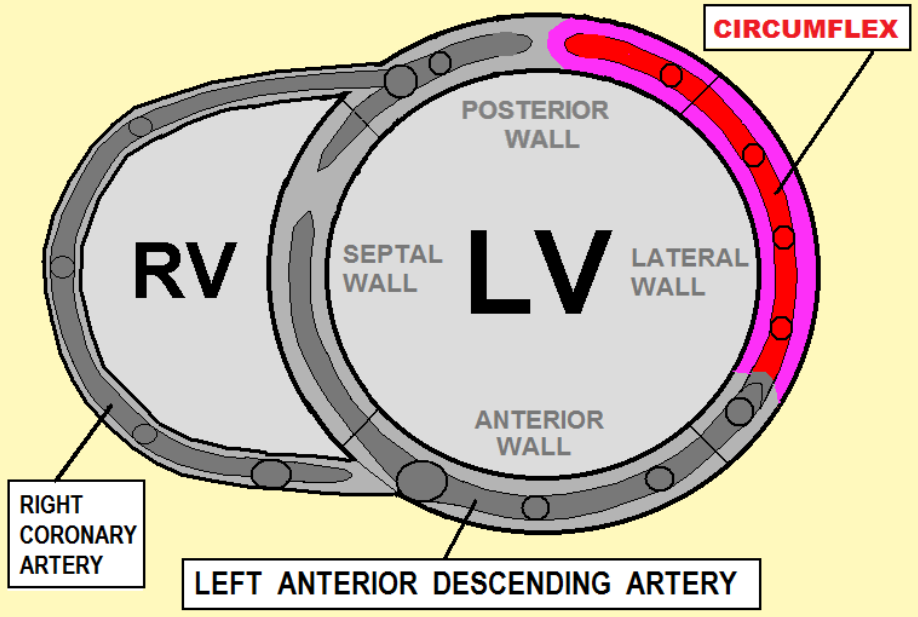

## The DOMINANT CIRCUMFLEX ARTERY . . .

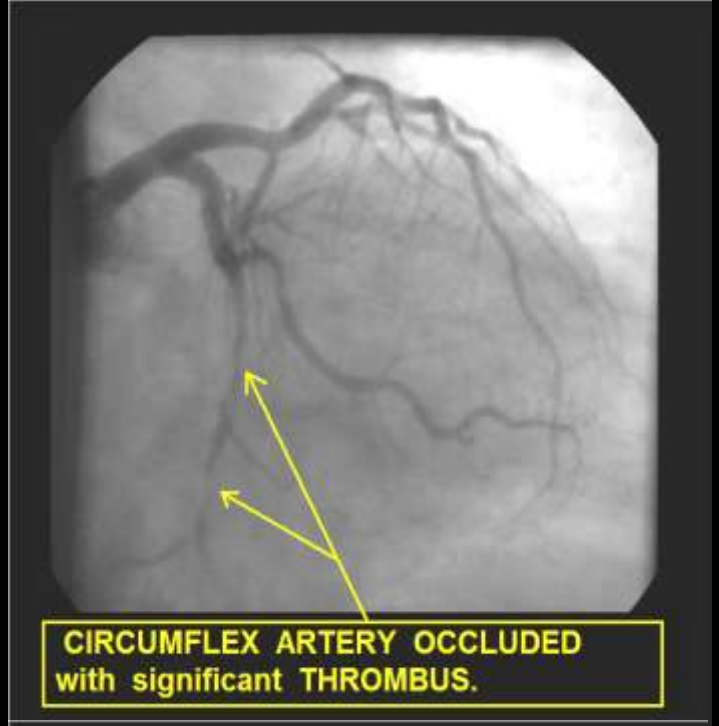
*SUPPLIES 35 - 55% OF THE LEFT VENTRICULAR MUSCLE MASS*

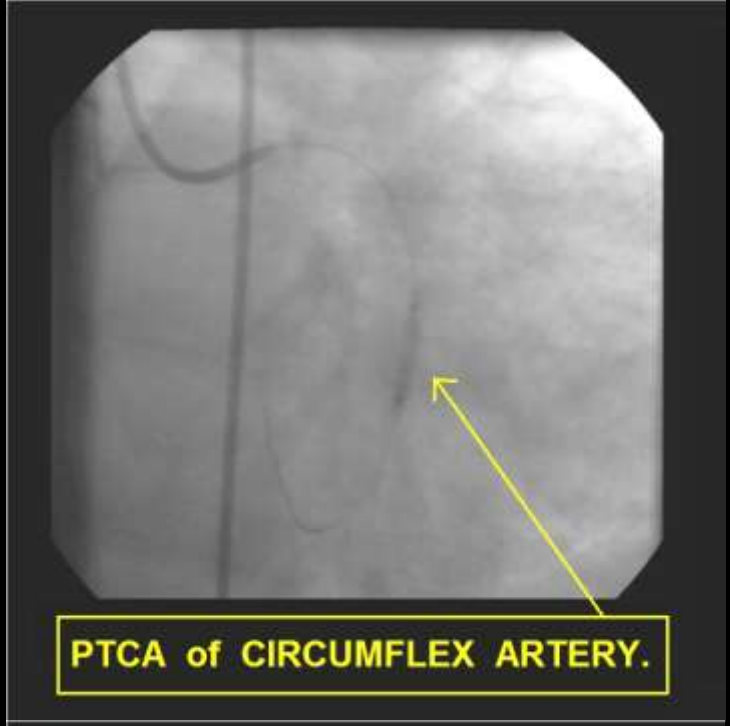


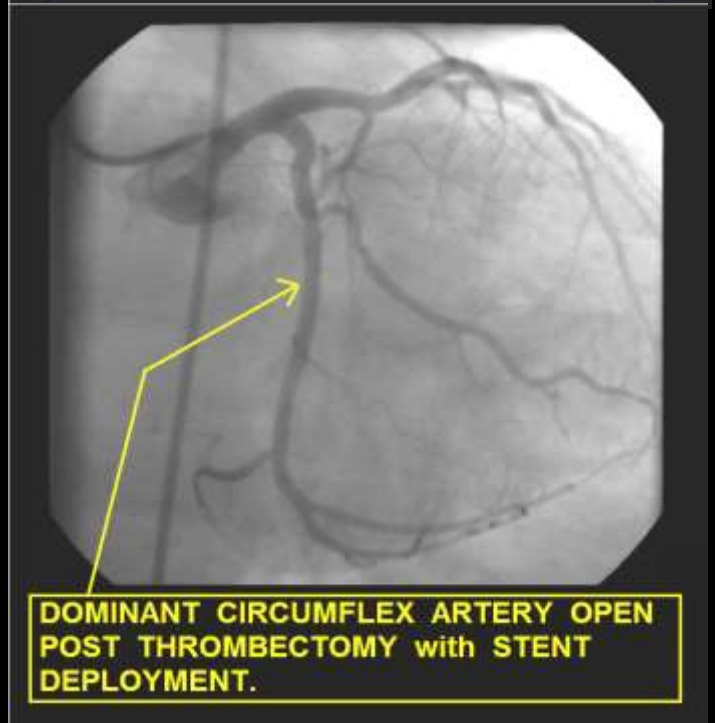

## The NON - DOMINANT CIRCUMFLEX ARTERY

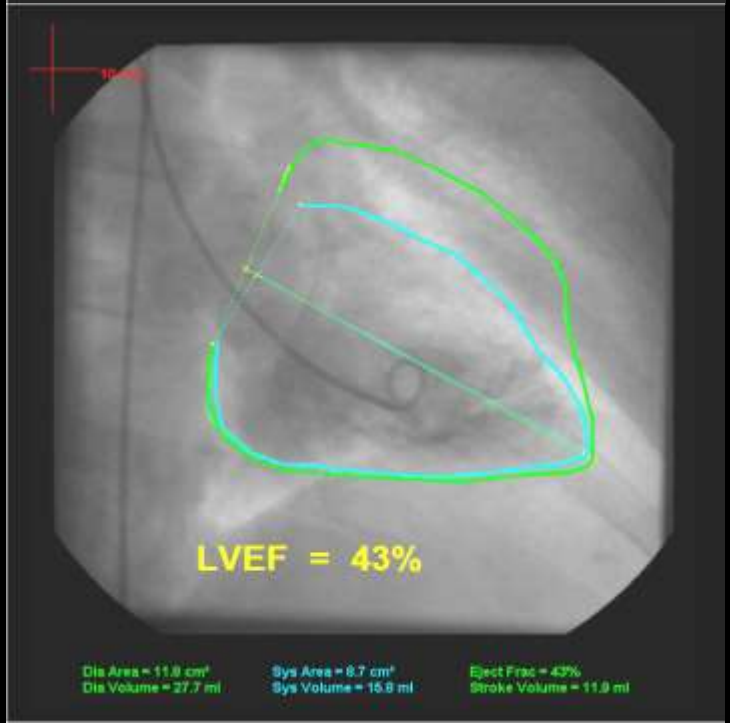
*SUPPLIES 25-30 % OF THE LEFT VENTRICULAR MUSCLE MASS*




CIRCUMFLEX ARTERY OCCLUDED
with significant THROMBUS.


PTCA of CIRCUMFLEX ARTERY.

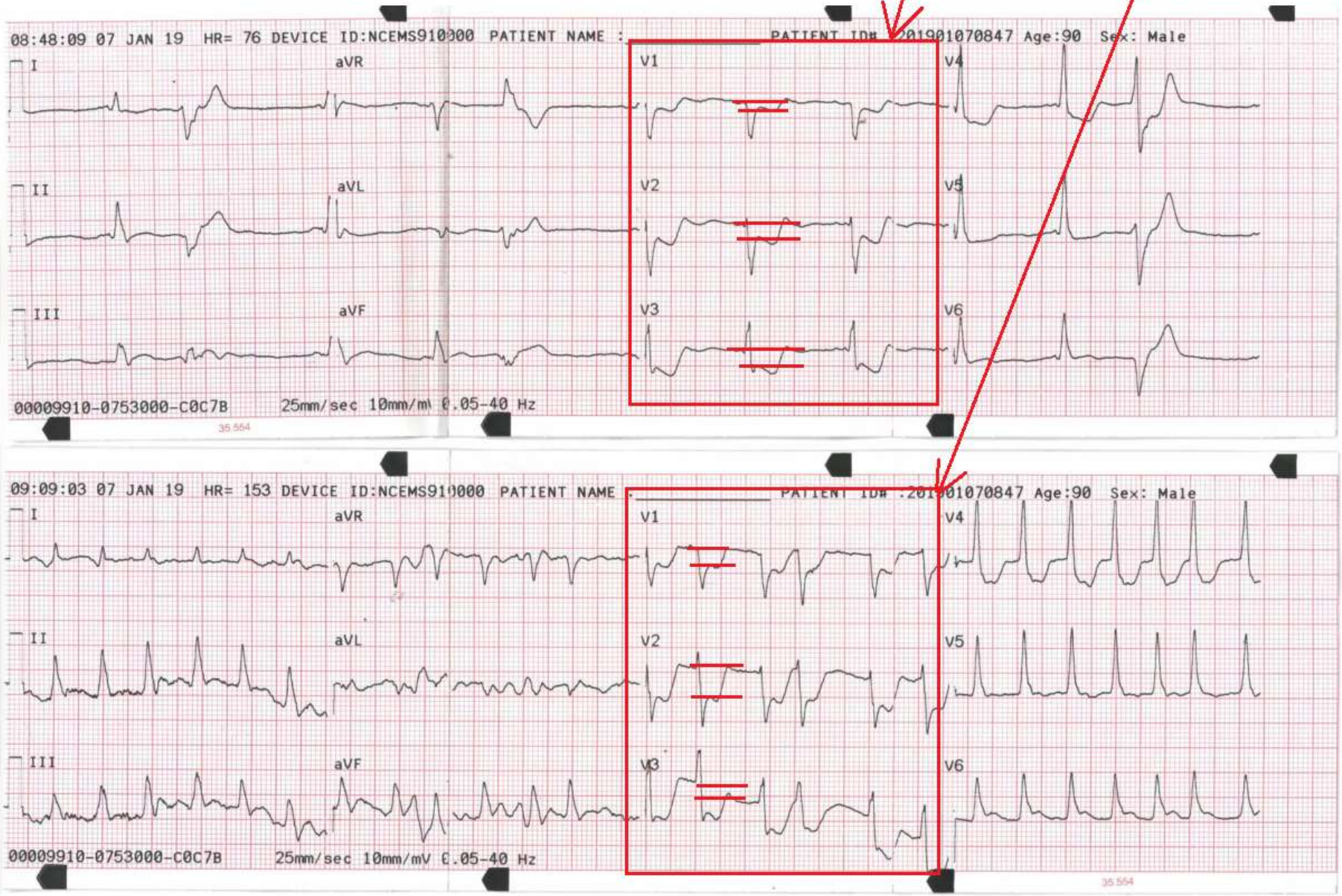

DOMINANT CIRCUMFLEX ARTERY OPEN
POST THROMBECTOMY with STENT
DEPLOYMENT.


LVEF = 43%
Dia Area = 11.8 cm²
Dia Volume = 27.7 ml
Sys Area = 8.7 cm²
Sys Volume = 15.8 ml
Eject Frac = 43%
Stroke Volume = 11.9 ml

# Case Study- January 2019

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

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



# Initial Exam in ED

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

Pat ID [redacted]

01/07/2019 09:19:35
[redacted] 79 yrs

[redacted]
Caucasian Female
Account # [redacted]

Bayfront Health Seven Rivers ED
Dept ED
Room ED01
Tech gp

RX
DX

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

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

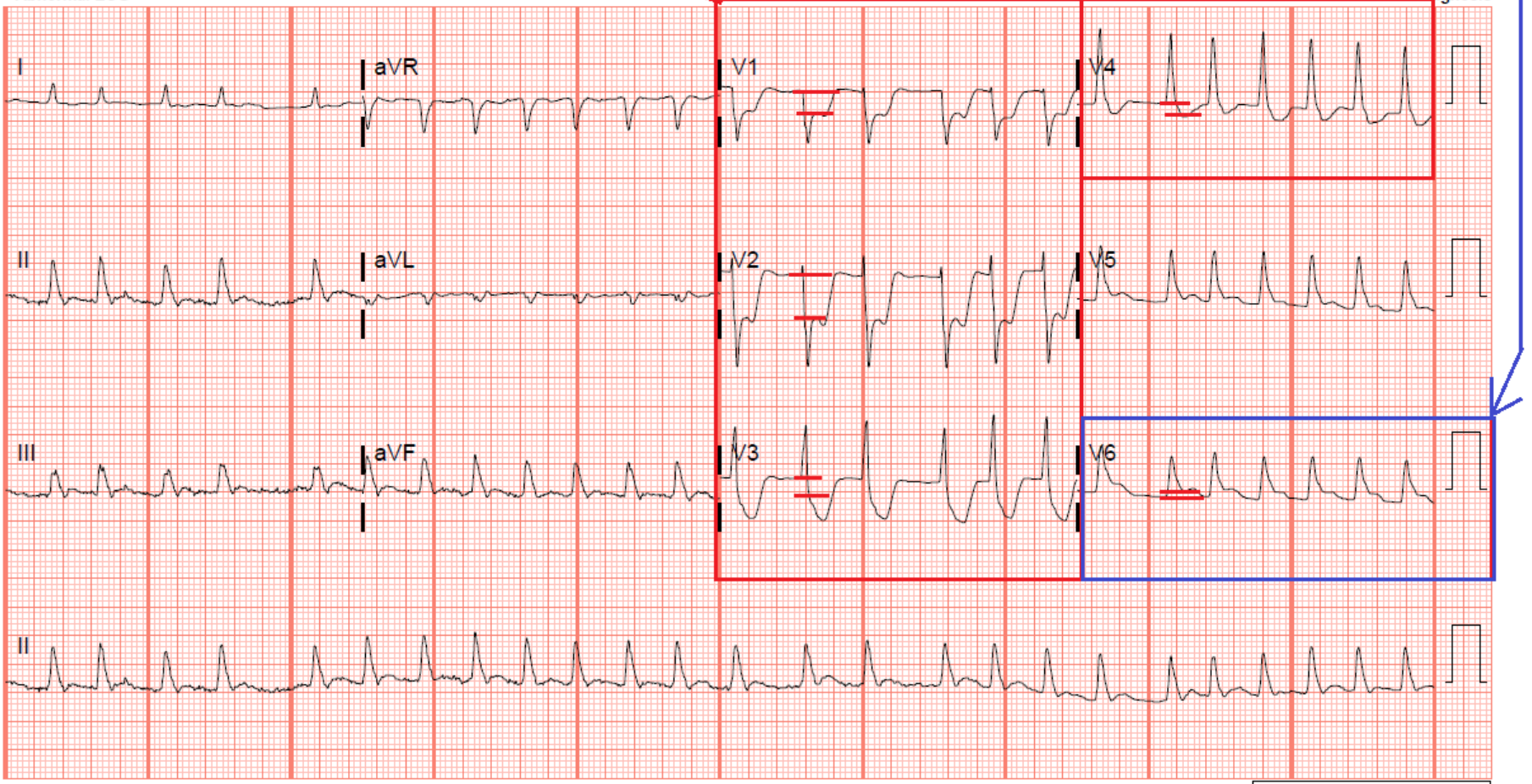
Req Provider:

**ST Depression Leads V1 - V4**

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

- Abnormal ECG -

Unconfirmed Diagnosis




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

# Causes of ST Depression V1-V4

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

Pat ID [redacted]

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

[redacted]
Caucasian Female
Account [redacted]

Bayfront Health Seven Rivers ED
Dept EDHD
Room EDH
Tech gp

RX
DX

Req Provider: ONIER VILLARREAL

| | | |
|---|---|---|
| Rate | 133 | Atrial fibrillation |
| PR | | ~~Anterolateral infarct, acute~~ |
| QRSd | 114 | Prolonged QT interval |
| QT | 337 | COMPARED TO ECG 01/07/2019 09:21:04 |
| QTc | 502 | PROLONGED QT INTERVAL NOW PRESENT |
| --Axis-- | | |
| P | | |
| QRS | 77 | |
| T | 121 | |

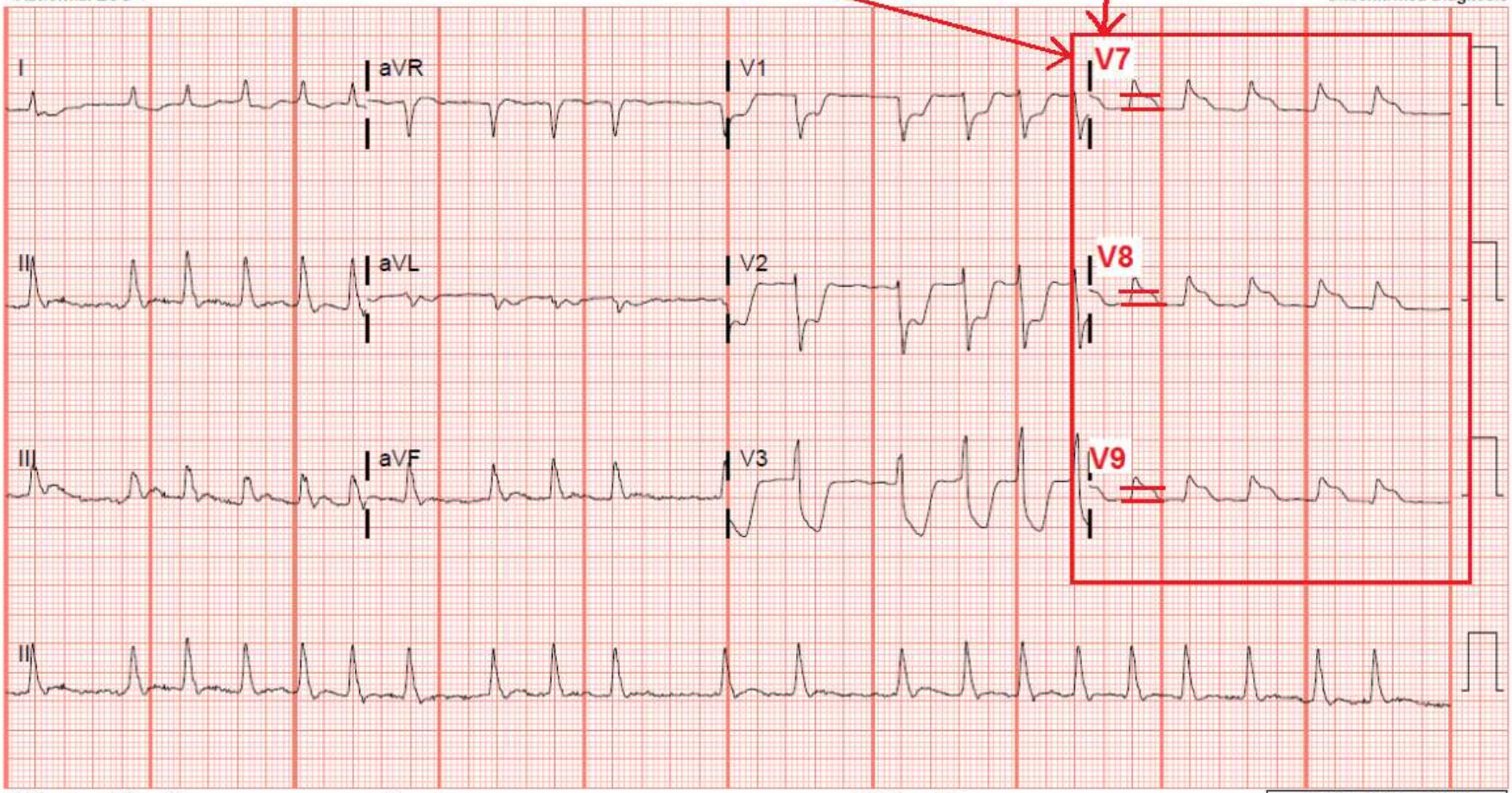
**** Posterior Infarct - Acute ****

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

**ACUTE POSTERIOR WALL STEMI**

- Abnormal ECG -

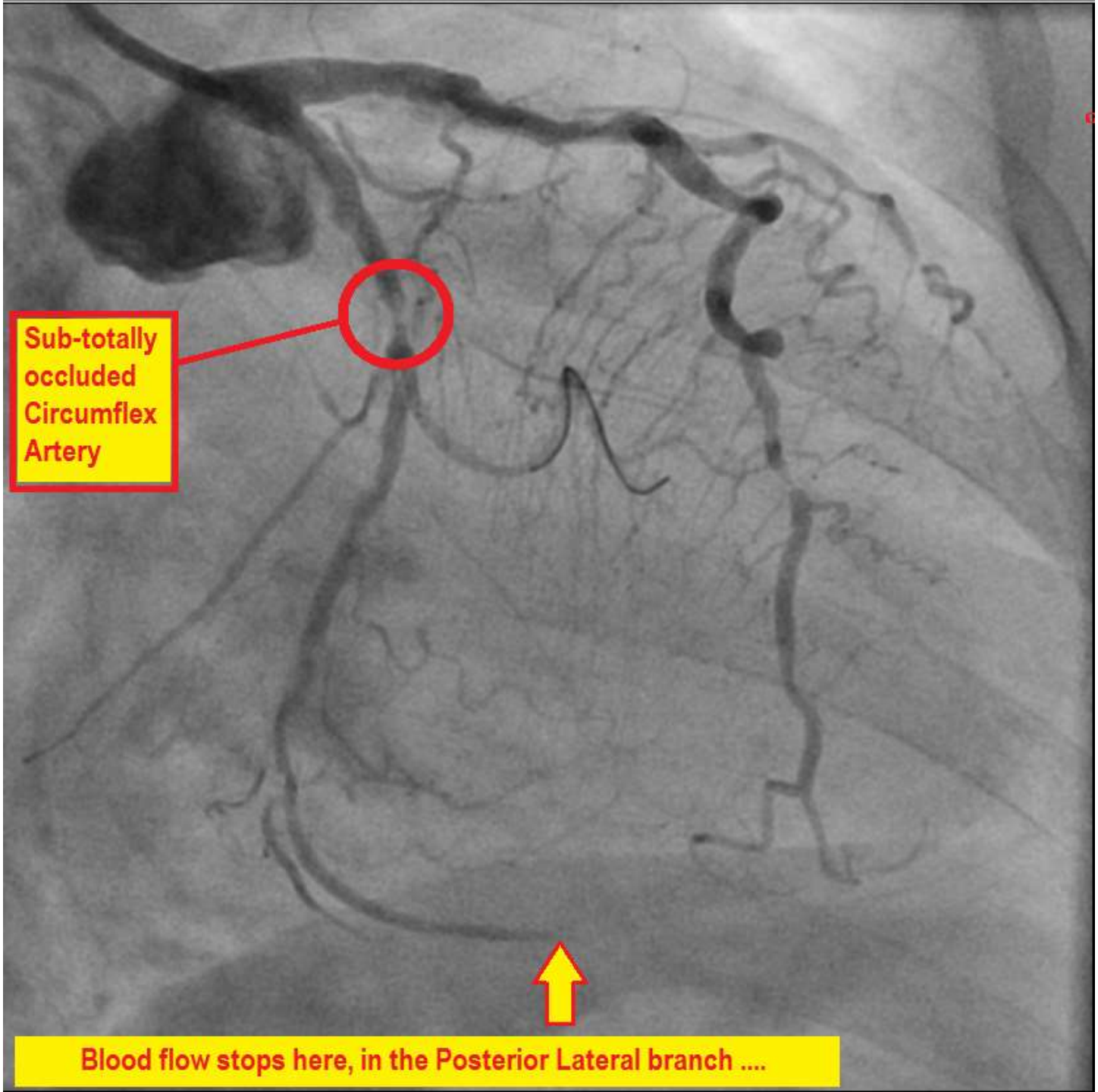
Unconfirmed Diagnosis

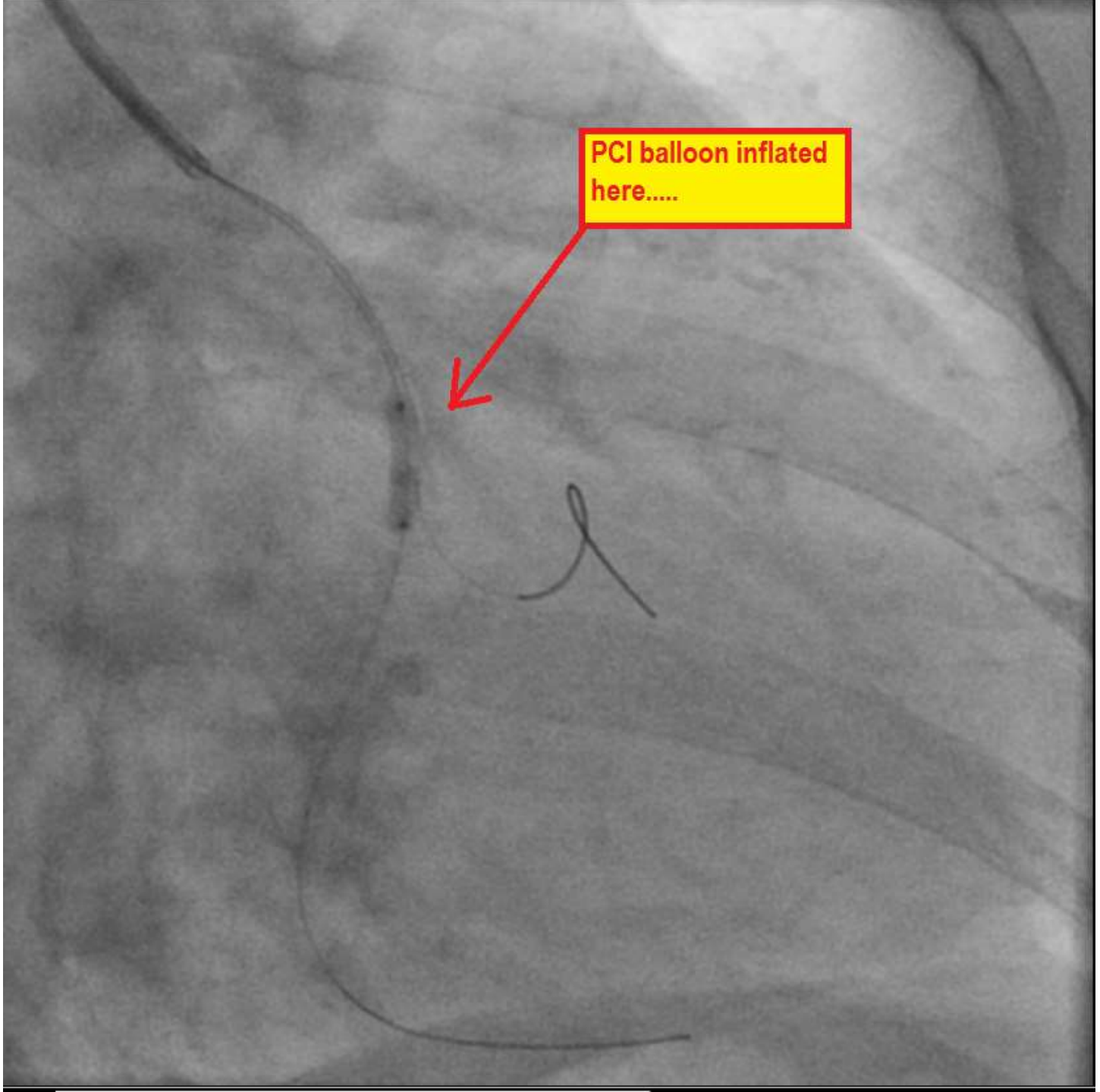


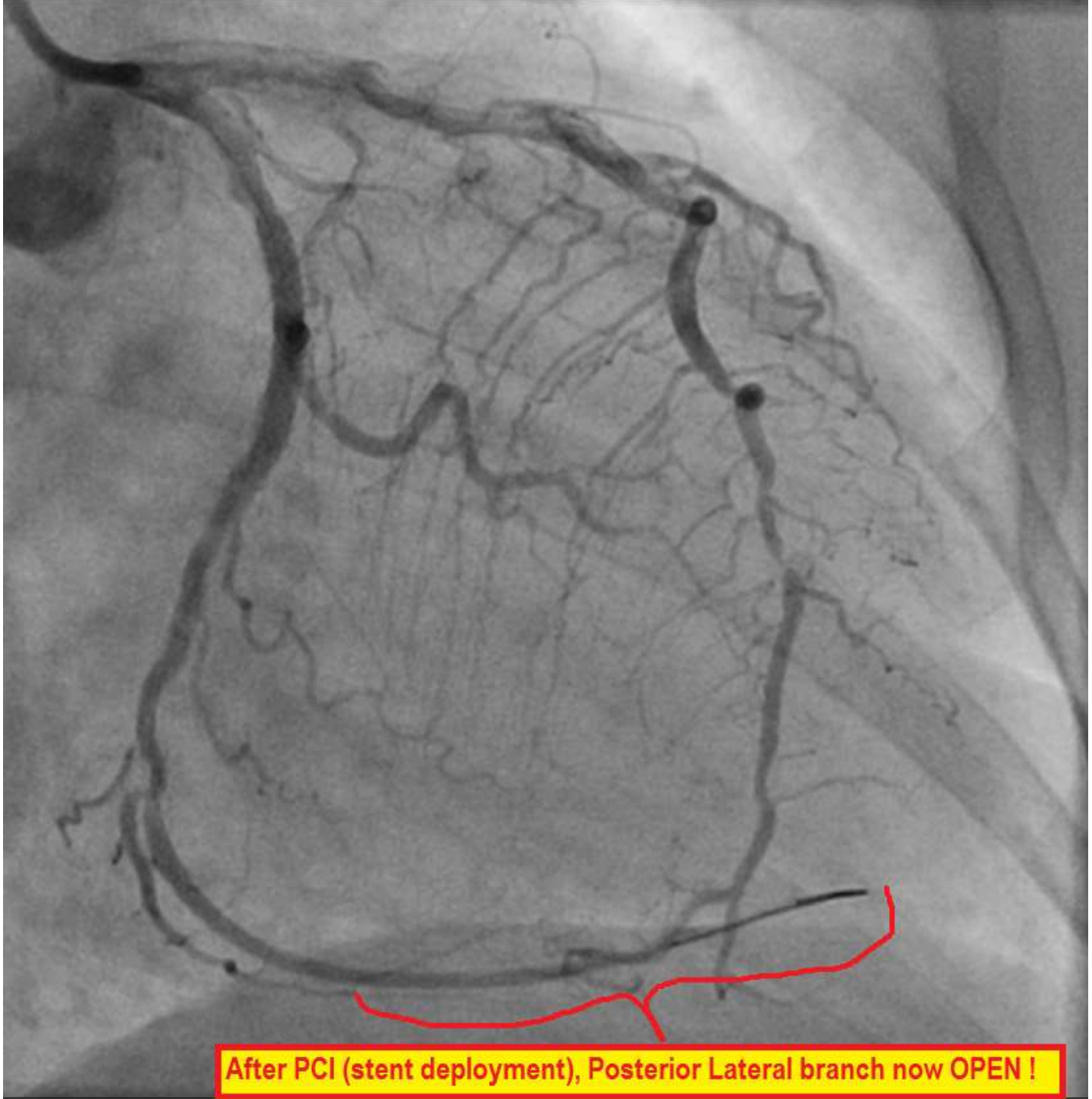

Philips PageWriter TC | 25 mm/sec | 10.00 mm/mV | F 60~ 0.15 - 100 Hz

# STEMI Alert !

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


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


PCI balloon inflated here.....


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

# SUMMARY

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

# OLD POSTERIOR MI - features

# INFARCTION


AS MYOCARDIAL CELLS BECOME NECROTIC ---

IN THE V LEADS:

POSTERIOR WALL MI

- S-T SEGMENTS return to normal
- TALL R-WAVES FORM V1, V2, V3
- R-WAVE PROGRESSION becomes EARLY

21-MAR-1997 15:37:12 ST. JOSEPH'S HOSPITAL-PAT ROUTINE RETRIEVAL

56 yr
Male Caucasian

Room:SGC
Loc:2 Option:13

| | | |
|---|---|---|
| Vent. rate | 64 | BPM |
| PR interval | 130 | ms |
| QRS duration | 84 | ms |
| QT/QTc | 398/410 | ms |
| P-R-T axes | 69 -17 | -97 |

Normal sinus rhythm
Inferior-posterior infarct , age undetermined
Abnormal ECG
No previous ECGs available

- SIGNIFICANT Q WAVES LEADS II, III, AVF

- TRANSITION V1 -- EARLY

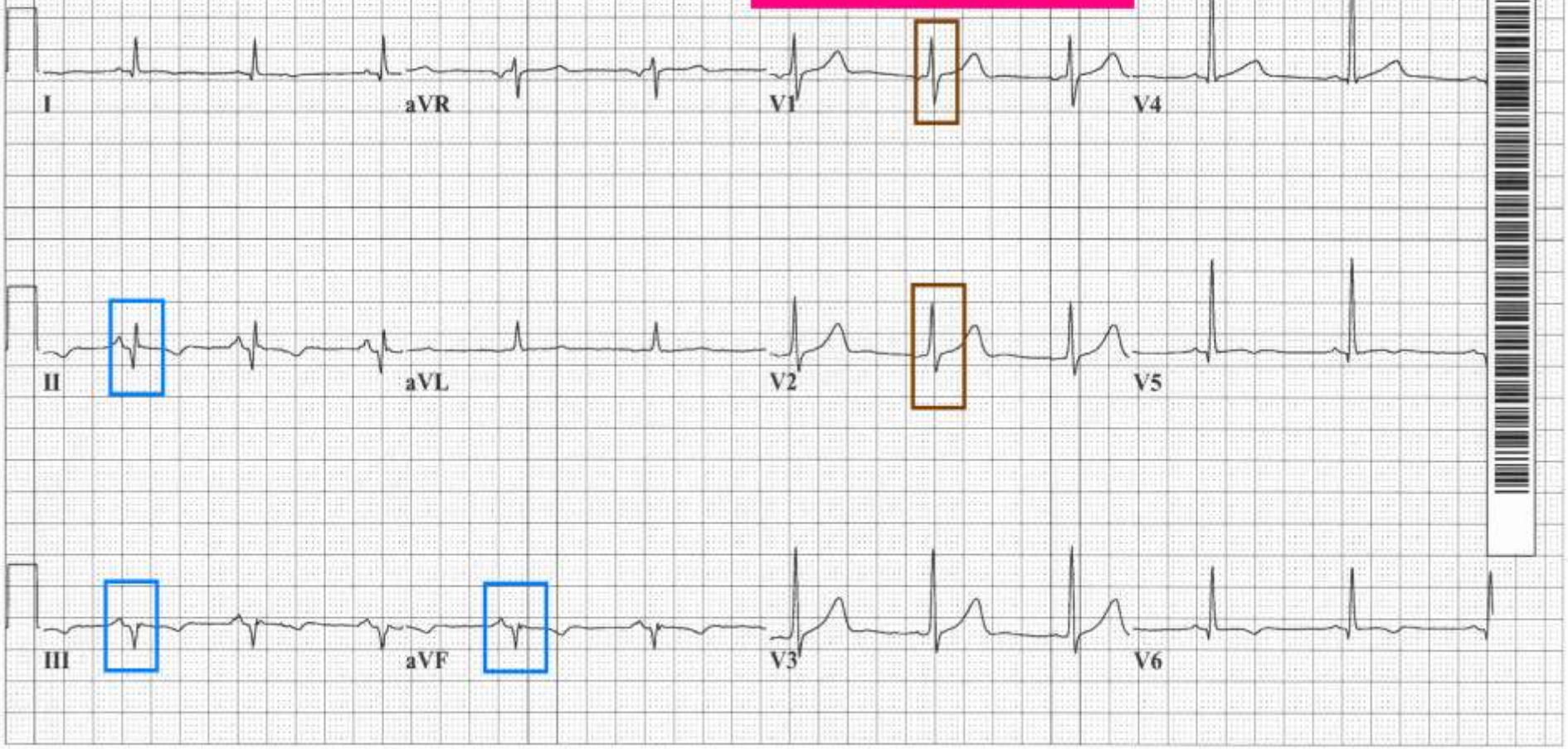
**EKG CLASS #WR03601840**

Referred by:

Confirmed By:

D.O.S.:3-27

- TRANSITION HERE -




25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 8

EID:17 EDT: 08:45 22-MAR-1997 ORDER:

Page 1 of 1

***YOU MADE IT !!!***

**Any**

**???**

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