



### The INTEGRATED **ECG**

#### **Bravera Health Seven Rivers**



#### **Bravera Citrus Hills**

**Bravera Health Brooksville** 



#### **Bravera Health Spring Hill**



Wayne W Ruppert, CVT, CCCC, NREMT-P Regional Director Cardiovascular Accreditations

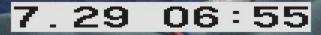


Welcome!

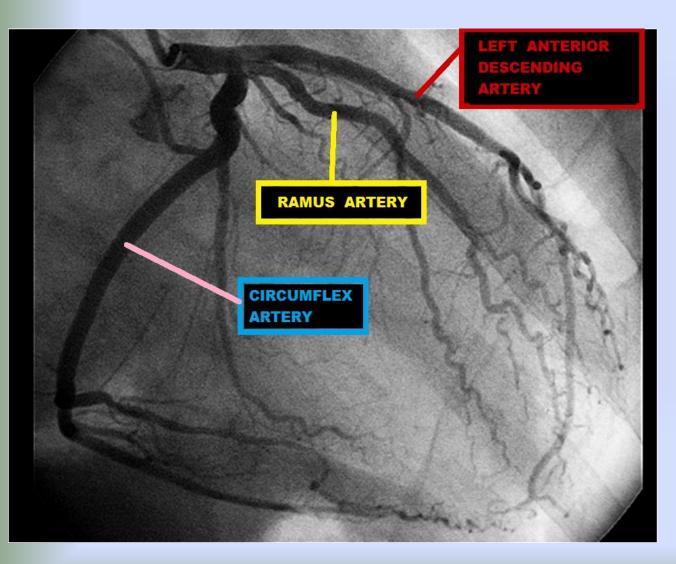


#### 13,000 – 15,000 EP and Cath Lab cases between 1996 - Today

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 Pacemapping during EP study at the St Joseph's Hospital Heart Institute, Pediatric Electrophysiology Program, Tampa, FL in 2004

### EP Lab Advantage:



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

### Wayne Ruppert – Bio:

- 1978 1996 EMT-Paramedic
- 1996 2012 Interventional Cardiovascular Technologist Cardiac Cath Lab and Electrophysiology Labs
- 2012 Present Cardiovascular Programs Director / Coordinator

### Wayne Ruppert - Bio:

- Cardiovascular Coordinator 2012-present (coordinated 7 successful accreditations)
- Interventional Cardiovascular / Electrophysiology Technologist, 1995-Present. (Approx 13,000 patients)
- Author of: "<u>12 Lead ECG Interpretation in Acute</u> <u>Coronary Syndrome with Case Studies from the Cardiac</u> <u>Cath Lab</u>," 2010, TriGen publishing / Ingram Books
- Author of: "<u>STEMI Assistant</u>," 2014, TriGen publishing / Ingram Books
- Florida Nursing CE Provider # 50-12998
- 12 Lead ECG Instructor, 1994-present (multiple hospitals, USF College of Medicine 1994)
- ACLS Instructor: 1982 2022
- Website: <u>www.ECGtraining.org</u>

### Source of Curriculum:

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

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- 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



#### THE CATH LAB SERIES presents ....

12 LEAD

ECG

INTERPRETATION

5

ACUTE

CORONARY

SYNDROME

with

CASE

STUDIES

from

the

CATH

LAB

:

WAYNE

RUPPER'



#### with CASE STUDIES from the

SYNDROME

ACUTE

CORONARY -

#### CARDIAC CATHETERIZATION LAB

WAYNE W RUPPERT

#### www.TriGenPress.com www.ECGtraining.org

#### BarnesandNoble.com Amazon.com

# **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

<u>Charles Sand, MD, FACP, FACEP</u>, Emergency Department Physician, St. Joseph's Hospital

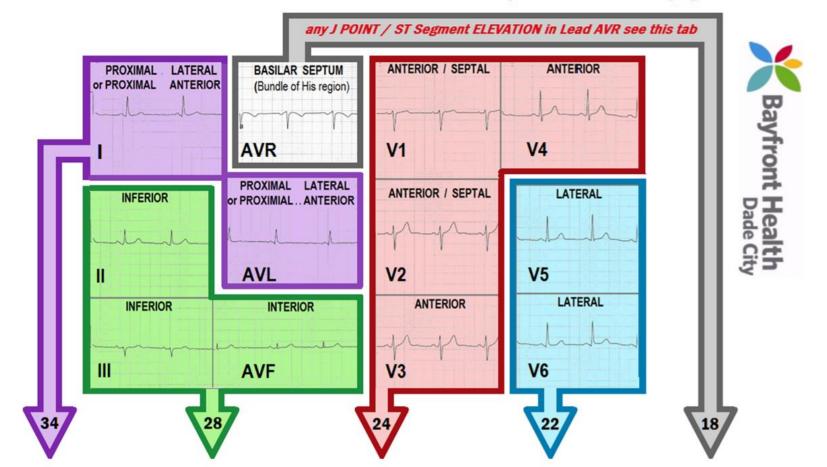
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by Wayne Ruppert

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

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



www.TriGenPress.com www.ECGtraining.org BarnesandNoble.com Amazon.com

# **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

<u>Anna Ek, AACC, BSN, RN</u> Accreditation Review Specialist, The American College of Cardiology

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

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**Tutorial Video** 

#### Free download – electronic copy (PDF file)

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All cardiovascular subject-related images, graphics and diagrams in this PowerPoint were created by the author, Wayne Ruppert, and have been taken from his two published textbooks, "STEMI Assistant" and "12 Lead ECG Interpretation in ACS with Case Studies from the Cardiac Cath Lab," which are Copyright protected. No content may be removed from this PowerPoint presentation, nor may this presentation or any component thereof be used without written consent from the author.

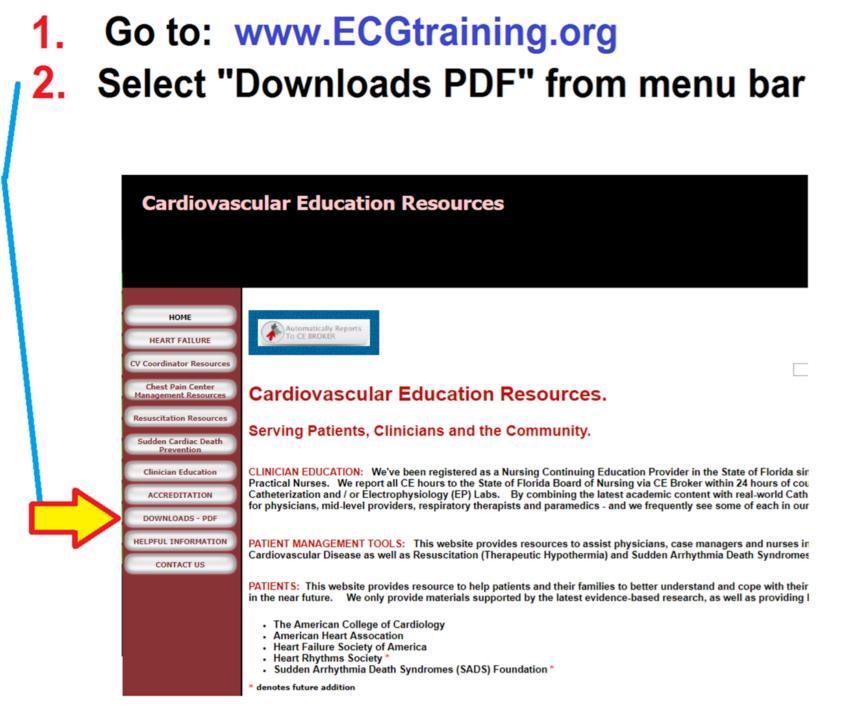
Wayne.ruppert@bayfronthealth.com

### Helpful Web Resources:

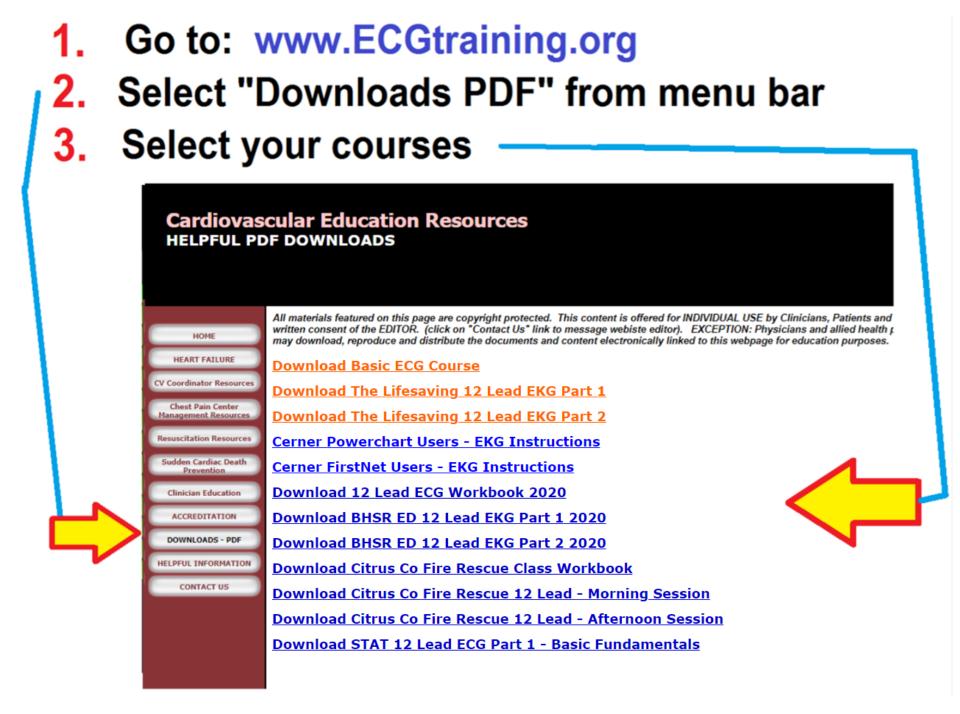
www.practicalclinicalskills.com

www.skillstat.com/tools/ecg-simulator

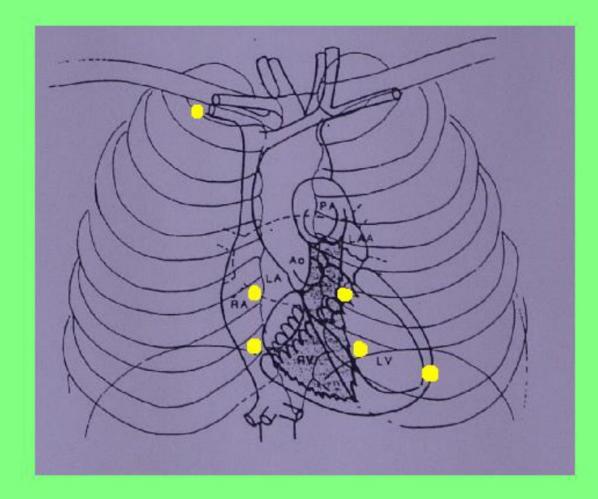
www.ECGtraining.org



**\_** \_ \_ \_ \_









- NormalHeartSounds
- Murmurs
   systolic
  - diastolic
- Friction Rubs



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

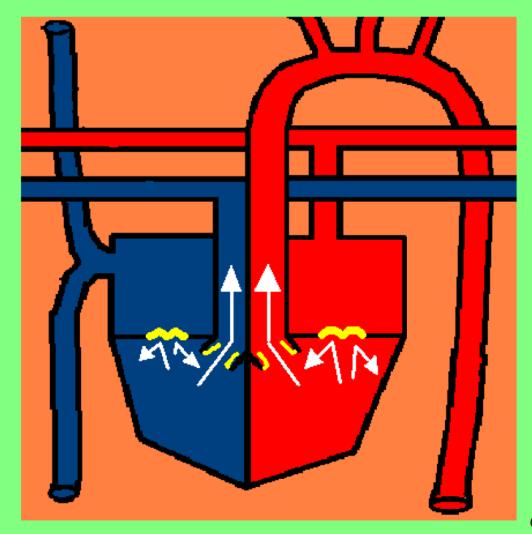
HEART SOUNDS ARE GENERATED BY THE SOUND OF THE HEART VALVES <u>CLOSING</u>.

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

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

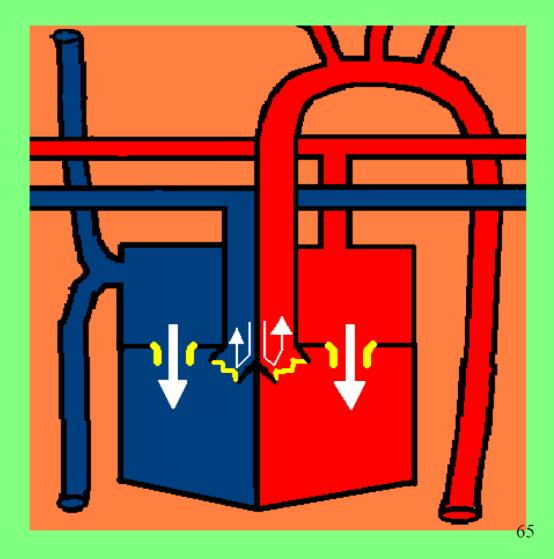
S-1 BEGINNING OF SYSTOLE.

SOUND OF THE MITRAL AND TRICUSPID VALVES CLOSING.



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.

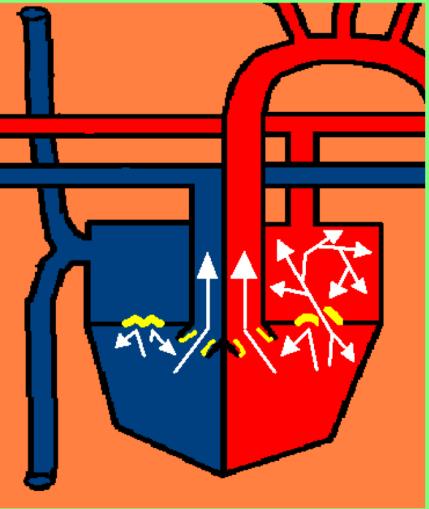
S-1 MURMUR SOUNDS LIKE:

"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.



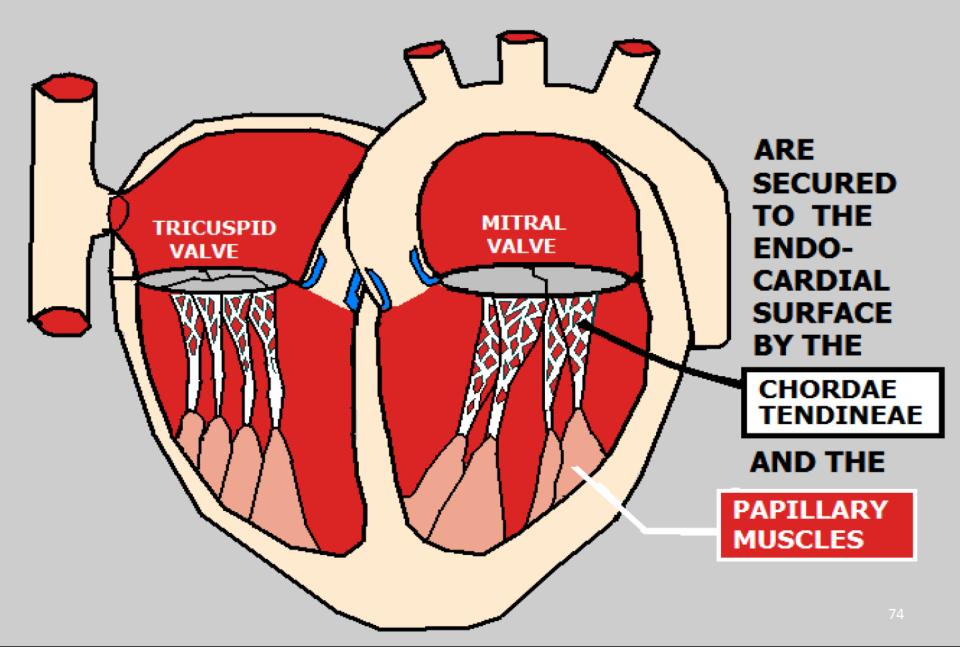
ACUTE MITRAL VALVE RUPTURE USUALLY OCCURS 7-10 DAYS POST EXTENSIVE MI (e.g.: INFERIOR POSTERIOR LATERAL MI). **ACUTE Mitral Valve REGURGITATION** can be caused by **EXTENSIVE "Multi-Site"** Myocardial Infarction and Necrosis – which results in PAPILLARY MUSCLE **NECROSIS** and **PAPILLARY MUSCLE TEAR.** 

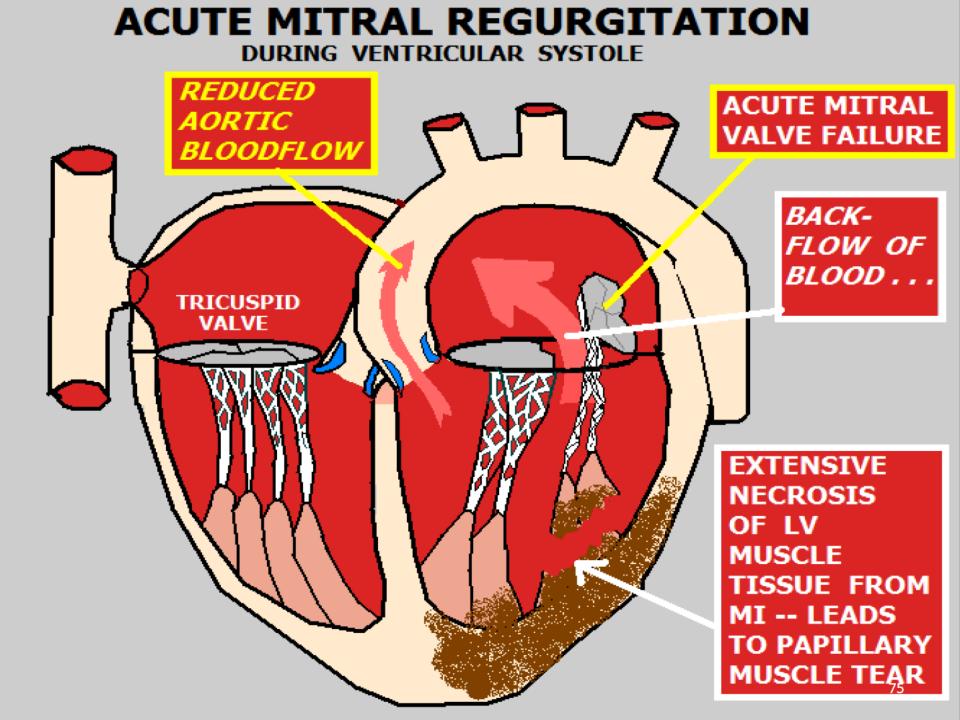
Papillary muscles are attached to "multiple surfaces" . . . .

# 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**





### 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.



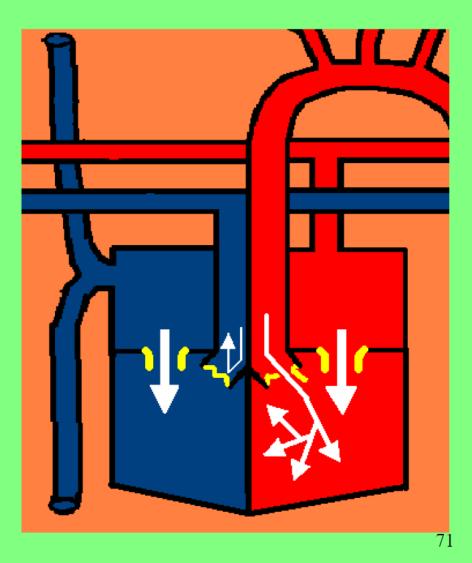
#### S-2 MURMUR SOUNDS LIKE:

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

#### CAUSE OF DIASTOLIC (S2) MURMUR

DAMAGE TO
 AORTIC and/or
 PULMONIC
 VALVE(s)

CAUSES REGURGITATION



# Access University of Washington Department of Medicine

Heart Sound Simulator

## PATIENT'S HEMODYNAMIC STATUS

+

**SYMPTOMS** 

+ ECG

#### HEMODYNAMIC STATUS

- ABCs
- Shock

#### • SYMPTOMS

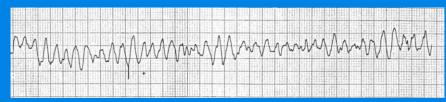
- Chest Pain / Pressure
- Other ACS Symptoms
- ECG
  - 12 Lead
  - Single Lead "rhythm strip"

• HEMODYNAMIC STATUS

- ABCs (Airway open? + Breathing? + Pulse?)

#### Start CPR

Apply ECG – determine rhythm- shockable?
 – SHOCKABLE: V-fib / V-tach / Torsades







Defib 120-200 BiPhasic

- IV Access
- Advanced Airway Defib 120-200 or HIGHER
- Epinephrine 1mg IV Defib 120-200 or HIGHER
- Amiodarone 300mg OR Lidocaine 1.0 -1.5 mg/kg
   Defib 120-200 or HIGHER
- Epinephrine 1mg IV Defib 120-200 or HIGHER
- CONTINUE as per ACLS....

#### Start CPR

Apply ECG – determine rhythm- shockable?
 – SHOCKABLE: V-fib / V-tach / Torsades

Torsades de Pointes . . . the QRS pattern resembles a "TWISTED RIBBON" . . . .





**CONSIDER using Lidocaine** in place of Amiodarone due to the increased possibility of QT PROLONGATION . . . Defib 120-200 BiPhasic

- IV Access
- Advanced Airway Defib 120-200 or HIGHER
- Epinephrine 1mg IV Defib 120-200 or HIGHER
- <del>Amiodarone 300mg -</del> OR -
- Lidocaine 1.0-1.5 mg/kg
- Defib 120-200 or HIGHER
- Epinephrine 1mg IV Defib 120-200 or HIGHER
- CONTINUE as per ACLS....

#### Start CPR

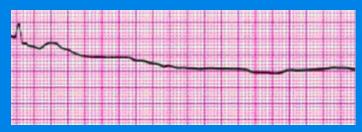
Apply ECG – determine rhythm- shockable?
 – NOT SHOCKABLE: Agonal Rhythm / Asystole / PEA



- Continue CPR
- IV/IO Access
- Advanced Airway
- Epinephrine 1mg IV
- Rule out reversible causes
- <u>CONTINUE as per ACLS</u>....

#### Start CPR

Apply ECG – determine rhythm- shockable?
 – NOT SHOCKABLE: Agonal Rhythm / Asystole / PEA



if the patient HAS A PULSE with AGONAL COMPLEXES . . . . IMMEDIATELY BEGIN TRANSCUTANEOUS PACING you will probably save the patient's life !

- Continue CPR
- IV/IO Access
- Advanced Airway
- Epinephrine 1mg IV
- Rule out reversible causes
- CONTINUE as per ACLS....

- HEMODYNAMIC STATUS
  - ABCs
  - Shock Assessment

# SHOCK ASSESSMENT



### SHOCK = INADEQUTE TISSUE PERFUSION

- STARTS THE INSTANT YOU SEE PATIENT

- ENDS WHEN YOU REACH THE PATIENT'S SIDE

# SHOCK ASSESSMENT

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

**SHOCK** is THE CORRIDOR TO DEATH

# SHOCK – FIND CAUSE . . .

- HYPOVOLEMIC (internal or external bleeding)
- OBSTRUCTIVE (PE / tamponade)
- PSYCHOGENIC (sudden fear self-correcting)
- NEUROLOGICAL (spinal injury)
- INSULIN (hypoglycemia)
- SEPTIC (systemic infection)
- CARDIOGENIC (abnormal heart rate or contractility)

### Actions at the Scene:

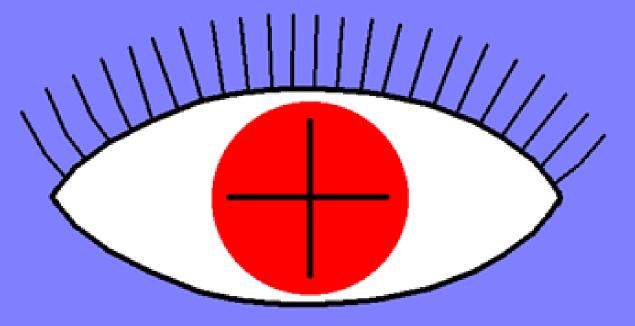
- Vital signs
- ECG
- Verbal history
- O2 (if indicated)
- IV (if indicated)

# THE EKG MACHINE

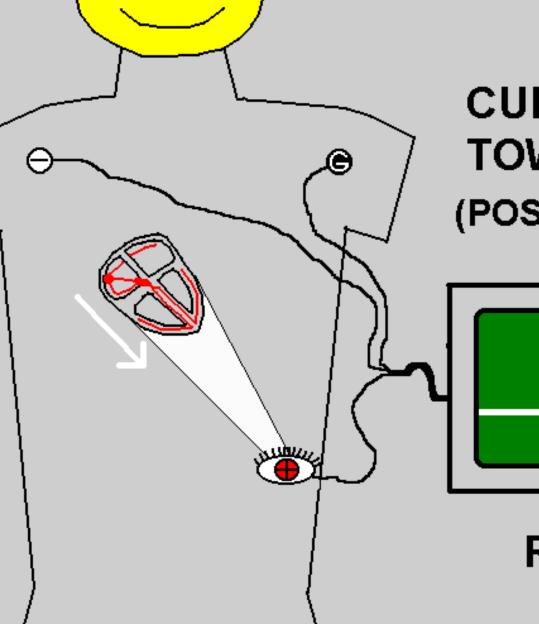
STANDARD 12 LEADS - USES 10 WIRES (6 CHEST and 4 LIMB)

- I, II, III, and V1, V2, V3, V4, V5, V6 EACH CONSIST OF:

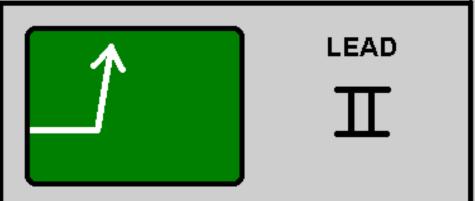
### THE POSITIVE ELECTRODE



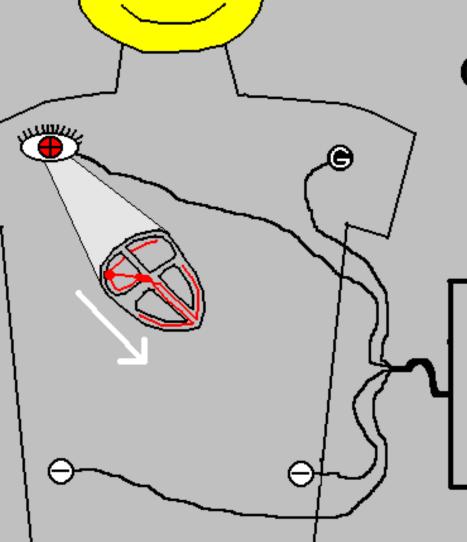
#### IS THE "EYE" . . .



#### CURRENT MOVING TOWARD THE EYE (POSITIVE ELECTRODE)



#### RECORDS AN "UPWARD" DEFLECTION



#### CURRENT MOVING AWAY FROM THE EYE (POSITIVE ELECTRODE)



RECORDS A "DOWNWARD" DEFLECTION

• Heart Rate:

-Should be between 50-150

• Heart Rate:

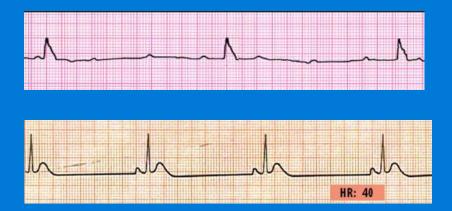
-Should be between 50-150

DECREASED CARDIAC OUTPUT may be present when heart rate is:

- LESS THAN 50

- GREATER THAN 150

- Heart Rate
  - TOO SLOW (less than 50) with signs of shock:
  - SPEED UP THE HEART RATE
  - (follow ACLS and Protocols)

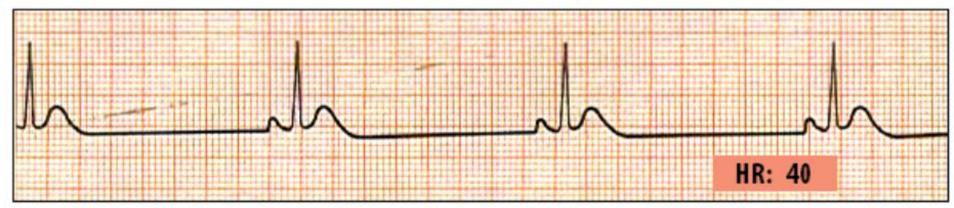


- **Usual treatment:**
- Atropine
- Pacemaker

### **Bradycardias & Heart Block**

- There are several ECG Rhythms seen with "BRADYCARDIA"
- While you should be able to distinguish each rhythm, what is MORE IMPORTANT is that you simply "identify when the heart rate being TOO SLOW is causing the patient to be symptomatic (SHOCK) . . . . and that you. . .
- KNOW how to treat it.

#### THIS RHYTHM IS: SINUS BRADYCARDIA



#### WE MUST CONSIDER UNDERLYING CAUSES:

INCREASED VAGAL TONE

HYPOTHERMIA \_\_\_\_\_

ORGANOPHOSPHATE POISONING  $\longrightarrow$ ATHLETIC METABOLISM  $\longrightarrow$ (excellent health!)

#### AND TREAT THEM:

ATROPINE

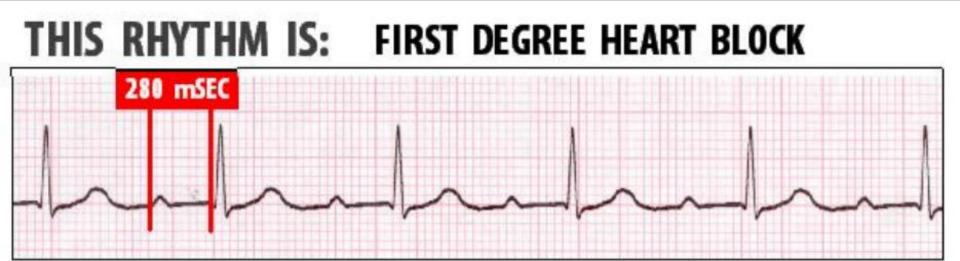
CARDIAC CATH - PTCA / STENT THROMBOLYTICS

CORRECT ELECTROLYTES

WARM PATIENT

ATROPINE

**COMPLIMENT PATIENT!** 



MAIN IDENTIFICATION CHARACTERISTIC(S): P - R INTERVAL TOO LONG -(GREATER THAN 200 mSEC.)

RATE	NORMAL
RHYTHM	REGULAR
P-R INTERVAL	> 200 mSEC
P: QRS RATIO	1:1
QRS INTERVAL	NORMAL

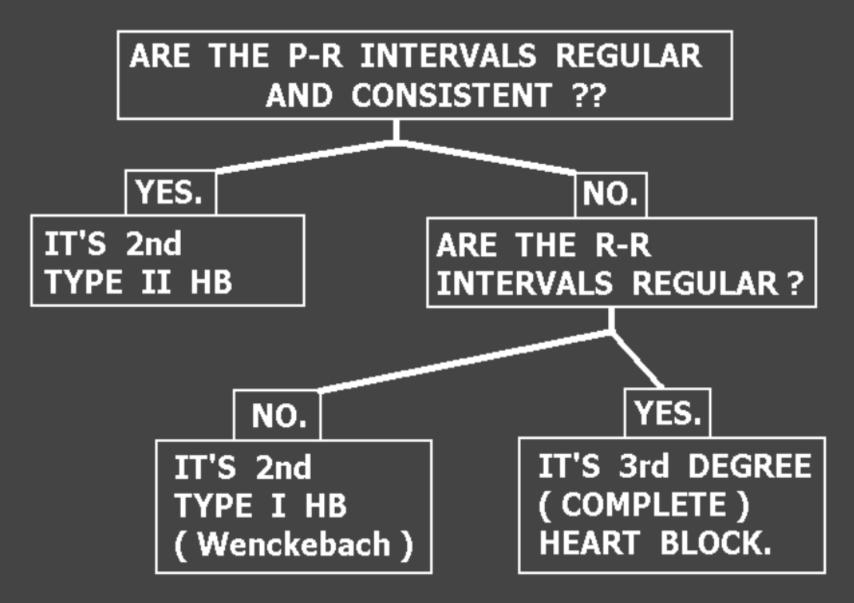
# **P:QRS RATIO** IF GREATER THAN 1:1

THINK:

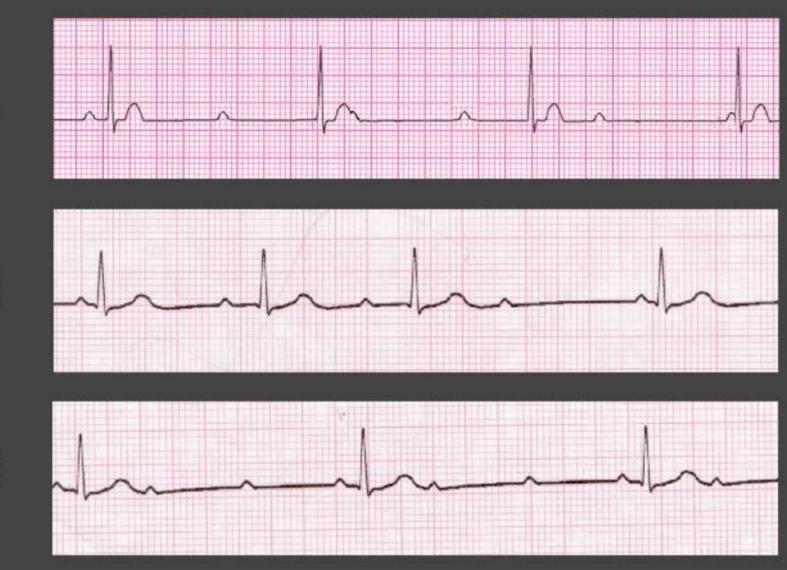
# - 2° HEART BLOCK (TYPE 1 or 2) - 3° HEART BLOCK



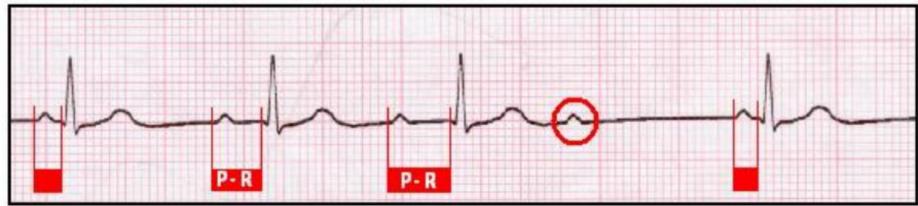
#### MORE P-WAVES THAN QRS COMPLEXES PRESENT.



#### LET'S TEST THE PROCEDURE . . .



### THIS RHYTHM IS: 2nd <sup>0</sup> TYPE I HB (Wenckebach)

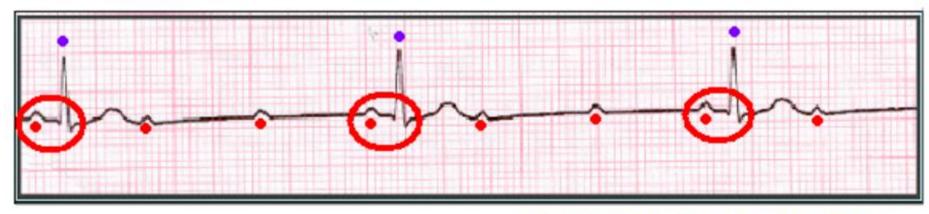


MAIN IDENTIFICATION CHARACTERISTIC(S): P-R INTERVAL GETS PROGRESSIVELY LONGER UNTIL IT DROPS A QRS -- THEN CYCLE REPEATS

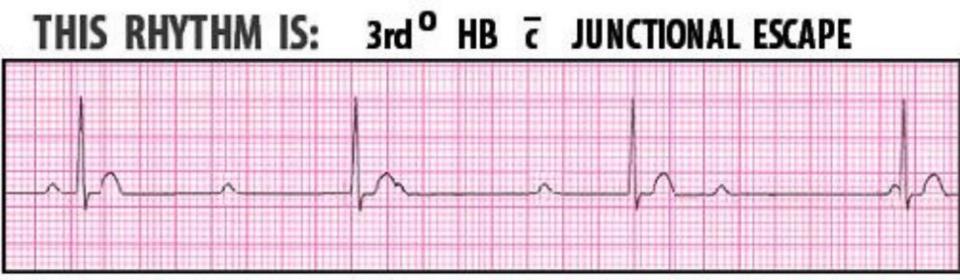
RATE ------RHYTHM ------P-R INTERVAL -----P: QRS RATIO ------QRS INTERVAL -----

NORMAL or BRADYCARDIC REGULARLY IRREGULAR VARIES (regularly irregular) VAIRES (usually 1:1 and 2:1) NORMAL

#### THIS RHYTHM IS: 2nd <sup>o</sup> TYPE II HEART BLOCK



MAIN IDENTIFICATION CHARACTERISTIC(S): MORE THAN ONE P WAVE FOR EACH QRS -- BUT EVERY QRS HAS A NORMAL, CONSISTENT P - R INTERVAL



#### MAIN IDENTIFICATION CHARACTERISTIC(S): P - R INTERVAL INCOSISTENT, P - P INTERVALS REGULAR, R - R INTERVALS REGULAR -- NO RELATIONSHIP BETWEEN P WAVES AND QRS COMPLEXES.

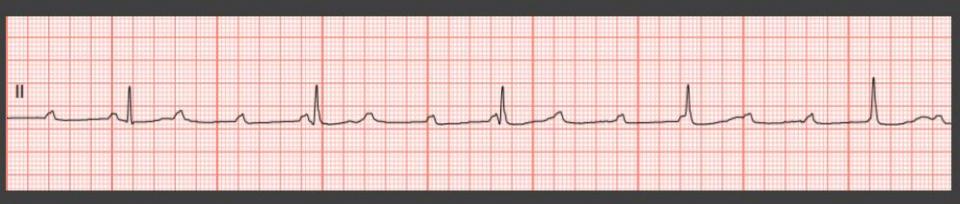
RATE	USUALLY BRADYCARDIC (40 - 60 JUNCTIONAL RATE)
RHYTHM	REGULAR
P-R INTERVAL	INCONSISTENT (irregularly irregular)
P: QRS RATIO	VARIES - USUALLY > 2:1
QRS INTERVAL	NORMAL ( < 120 ms ) UNLESS PT HAS BUNDLE BRANCH BLOCK

### THIS RHYTHM IS: 3rd<sup>O</sup> HB & IDIOVENTRICULAR ESCAPE



MAIN IDENTIFICATION CHARACTERISTIC(S): P - R INTERVALS INCONSISTENT P - P INTERVALS REGULAR, R - R INTERVALS REGULAR. NO RELATIONSHIP BETWEEN P WAVES AND QRS COMPLEXES. QRS COMPLEXES are WIDER THAN 120ms, AND OF SLOW VENTRICULAR RATE (usually < 40)

RATE	USUALLY BRADYCARDIC ( < 40 VENTRICULAR RATE)
RHYTHM	REGULAR
P-R INTERVAL	INCONSISTENT (irregularly irregular)
P: QRS RATIO	VARIES - USUALLY > 2:1
QRS INTERVAL	WIDER THAN 120 ms



???

#### THIS RHYTHM IS: JUNCTIONAL RHYTHM



MAIN IDENTIFICATION CHARACTERISTIC(S): P WAVES ABSENT, or LOCATED JUST AFTER QRS (in S-T seg) or JUST BEFORE QRS (short P-R). WHEN P wave RATE ------- 40-60 RHYTHM ------ 40-60 REGULAR P-R INTERVAL ----- ABSENT or SHORT P: QRS RATIO ----- 1:1

QRS INTERVAL ----- NORMAL

- Heart Rate
  - TOO FAST (greater than 150) with signs of shock:
  - SLOW the heart rate
    (follow ACLS and Protocols)

hhandhhandhhandhha



Usual treatment: - Synchronized Cardioversion

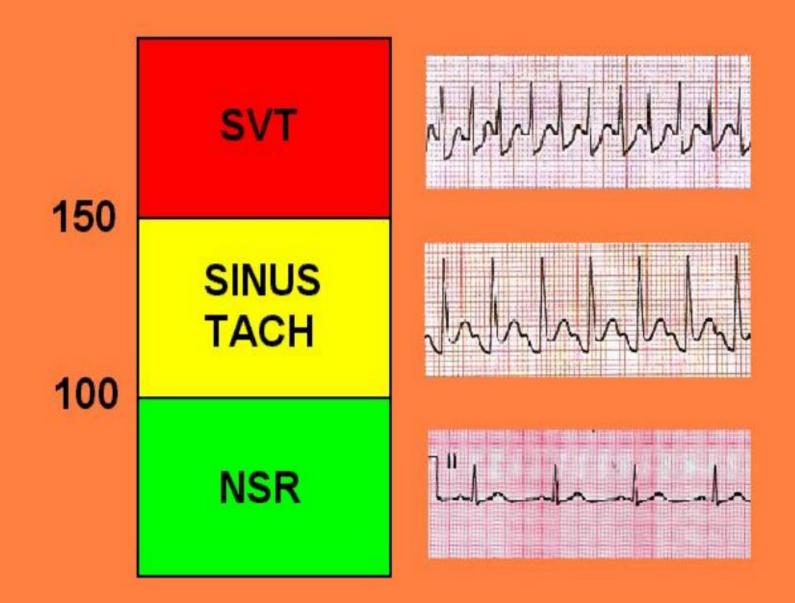
### Tachycardias

- Is the patient STABLE or UNSTABLE?
- QRS narrow or wide ???
  - Narrow = "not greater than 120 ms" (3 mm)
  - Wide = "greater than 120 ms (3 mm)

# ALL UNSTABLE TACHYCARDIAS:

- SYNCHRONIZED CARDIOVERSION
  - As per agency PROTOCOL and/or ACLS
  - NARROW tachycardias = less initial energy
  - WIDE QRS tachycardias AND A-fib RVR = higher initial energy

## ACLS TACHYCARDIA GUIDELINES



## THIS RHYTHM IS: SINUS TACHYCARDIA



MAIN IDENTIFICATION CHARACTERISTIC(S): SINUS RHYTHM, RATE HIGHER THAN 100. (ACLS guidelines: heart rate 100 - 150 )

RATE	100 - 150 ( can be > 150 )
RHYTHM	REGULAR
P-R INTERVAL	NORMAL (120 - 200 ms)
P: QRS RATIO	1:1
QRS INTERVAL	NORMAL (< 120 ms), (unless Bundle Branch Block present)

## THIS RHYTHM IS: SINUS TACHYCARDIA



#### WE MUST CONSIDER UNDERLYING CAUSES :

# ANXIETY/FEAR

# AND TREAT THEM :

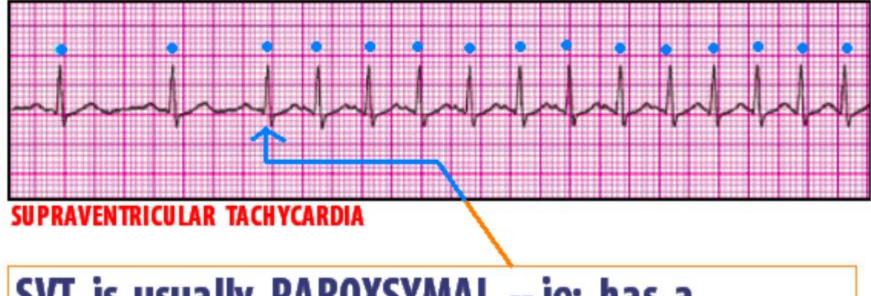
### $\rightarrow$ CALM PATIENT

HYPOVOLEMIA DEHYDRATION BLOOD LOSS

#### 

→ FLUID S
→ STOP BLEEDING
→ CONSIDER MEDICAL Tx
→ IDENTIFY & Tx DISORDER

### RHTHYM CLUES . . . .



# SVT is usually PAROXSYMAL -- ie: has a SUDDEN ONSET.

SINUS TACHYCARDIA usually has a "ramp - up " and "ramp - down " period -- a gradual change in HEART RATE.

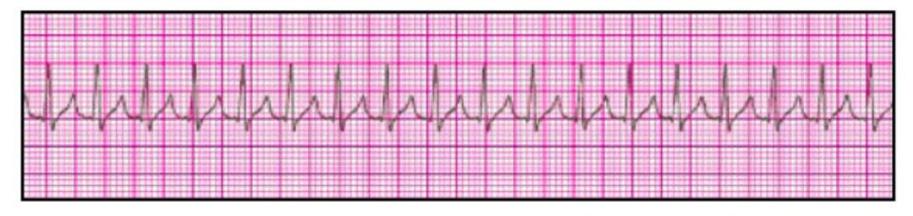
### THIS RHYTHM IS: SUPRAVENTRICULAR TACHYCARDIA (SVT)



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
P: QRS RATIO	1:1	TO P WAVE BURIED IN T WAVES
QRS INTERVAL	NORMAL	

## 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."

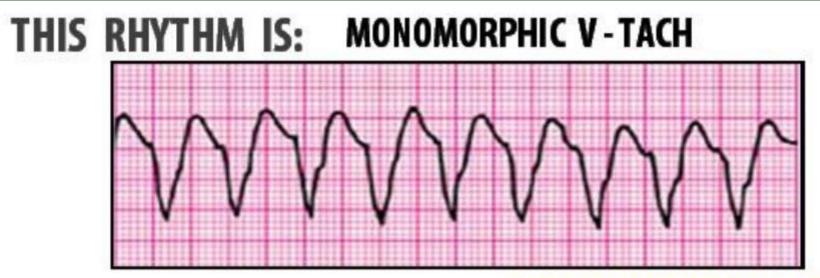
#### **TREATMENT / INTERVENTIONS :**





#### MAIN IDENTIFICATION CHARACTERISTIC(S):

RATE -----RHYTHM ------P-R INTERVAL -----P: QRS RATIO ------QRS INTERVAL -----



MAIN IDENTIFICATION CHARACTERISTIC(S): WIDE QRS COMPLEXES (> 120 ms) HR USUALLY BETWEEN 150 - 200; ALL QRS COMPLEXES APPEAR SAME IN SHAPE and DEFELCTION; IF P WAVES SEEN, DISASSOTIATED w/ QRS

RATE	> 100 (usually 150 - 200)
RHYTHM	REGULAR
P-R INTERVAL	N/A
P: QRS RATIO	N/A
QRS INTERVAL	> 120 ms

## V-Tach

- NO PULSE Follow Protocols / ACLS for "V-Fib / V-Tach"
- PULSE but UNSTABLE Synchronized Cardioversion
- STABLE Give MEDS as per Protocols / ACLS

### THIS RHYTHM IS: POLYMORPHIC V - TACH



#### MAIN IDENTIFICATION CHARACTERISTIC(S): WIDE QRS COMPLEXES, MULTIPLE SHAPES AND FORMS, POSITVE AND NEGATIVE DEFLECTIONS, APPEARS TO ROTATE BETWEEN NEGATIVE AND POSITIVE (TWISTING OF POINTS)

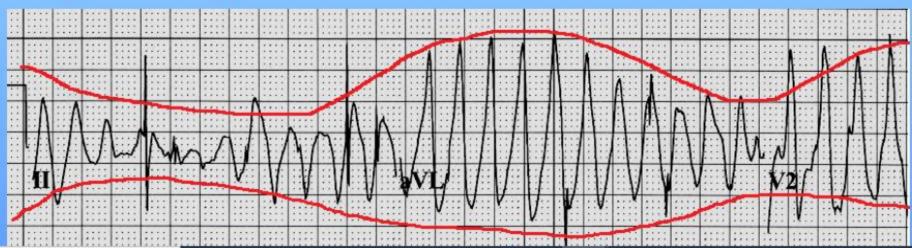
RATE	200-300
RHYTHM	VARIES
P-R INTERVAL	N/A
P: QRS RATIO	N/A
QRS INTERVAL	VARIES

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

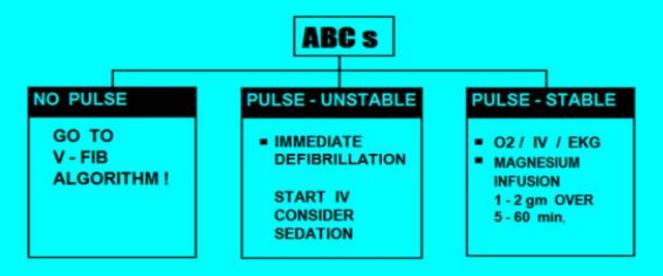
resembles . . . .



## a piece of Twisted Ribbon !







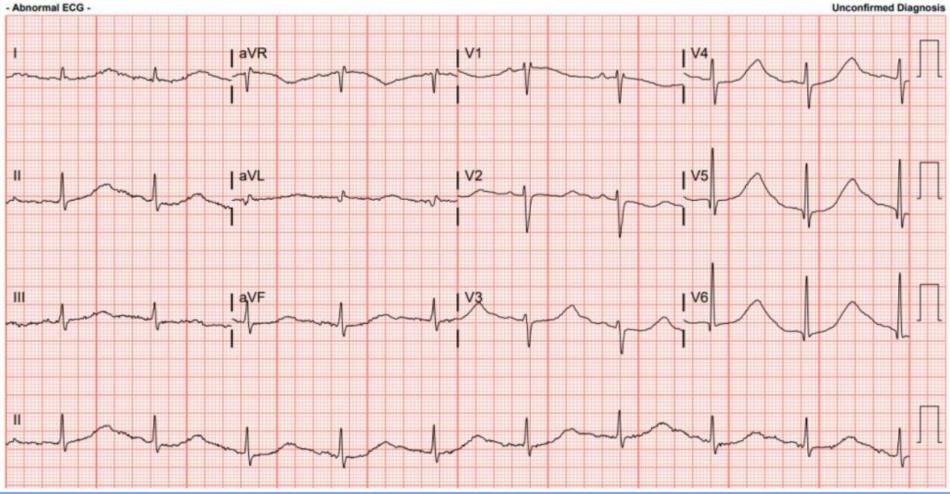
DO NOT give PROCAINAMIDE, AMIODARONE, or SOTALOL to patients with TORSADES or POLYMORPHIC VT !!!

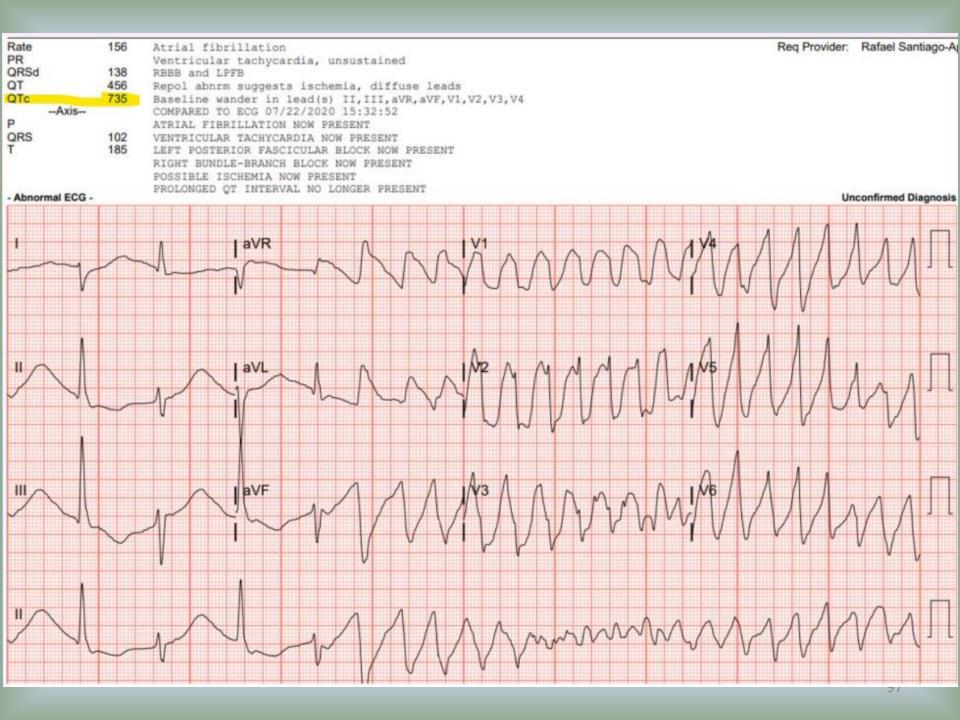
# QTc Values:

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		

Reg Provider: Rafael Santiago-Ap

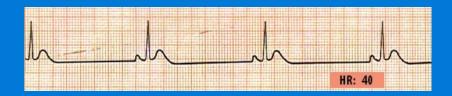






- Heart Rate TOO SLOW or TOO FAST
  - Wide QRS
  - Narrow QRS









• Heart Rate:

-Should be between 50-150

- Heart Rate:
  - -Should be between 50-150
- Decreased Contractility: – STEMI / Acute Coronary Syndrome (vascular)



- Heart Rate:
  - -Should be between 50-150
- Decreased Contractility:
   STEMI / Acute Coronary Syndrome (vascular)
   Myocarditis (muscle dysfunction)

- Heart Rate:
  - -Should be between 50-150
- Decreased Contractility:

   STEMI / Acute Coronary Syndrome (vascular)
   Myocarditis (muscle dysfunction)
   Often mimics STEMI on the ECG. Often
   "challenging" for advanced practitioners to diagnose.

# Integrated ECG:

- HEMODYNAMIC STATUS
  - ABCs
  - Shock
- SYMPTOMS
  - Chest Pain / Pressure

# Integrated ECG:

- HEMODYNAMIC STATUS
  - ABCs
  - Shock
- SYMPTOMS
  - Chest Pain / Pressure = STAT 12 LEAD ECG !!!

(within 10 minutes) !!

# CHIEF COMPLAINT

#### **KEY WORDS:**

#### "CHEST: PAIN / HEAVINESS / PRESSURE/ FUNNY FEELING IN," etc.

#### SHORTNESS BREATH

**DIZZINESS / LIGHTHEADEDNESS** 

ETC. ETC. ETC.

SYMTOMS OF MYOCARDIAL INFARCTION:

#### 1. CHEST PAIN:

- Substernal can radiate to neck, shoulders, jaw, L or R arm
- Pain described as "Dull Pain" or "Pressure" or "Heaviness" but can be sharp
- Usually NOT effected by DEEP INSPIRATION, POSITION, or MOVEMENT

SYMTOMS OF MYOCARDIAL INFARCTION:

 CHEST PAIN
 SHORTNESS OF BREATH May or may not be present.

SYMTOMS OF MYOCARDIAL INFARCTION:

- 1. CHEST PAIN
- 2. SHORTNESS OF BREATH
- 3. NAUSEA
  - May or may not be present

SYMTOMS OF MYOCARDIAL INFARCTION:

- 1. CHEST PAIN
- 2. SHORTNESS OF BREATH
- 3. NAUSEA
- 4. COLD, CLAMMY, PALE SKIN and other signs of hypoperfusion may be present

# - - - "Classic Symptoms" - - -

### **QUICK ASSESSMENT "SHORT FORM**"

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

# Integrated ECG:

### HEMODYNAMIC STATUS

- ABCs
- Shock
- SYMPTOMS
  - Chest Pain / Pressure
  - Other ACS Symptoms

# Integrated ECG:

- HEMODYNAMIC STATUS
  - ABCs
  - Shock
- SYMPTOMS
  - Chest Pain / Pressure
  - Other ACS Symptoms = STAT 12 LEAD ECG !!!
    - (within 10 minutes)!!

# **ATYPICAL SYMPTOMS of ACS**

???

Acute MI patients who present without chest pain<sup>\*</sup> are SHREWD:

Stroke (previous history of) Heart failure (previous history of) Race (non-white) Elderly (age 751) Women Diabetes mellitus \* The information listed in the table to the immediate left resulted from a study conducted by John G. Canto, MD, MSPH, et. al., of the University of Alabama. The study consisted of 434,877 patients diagnosed with AMI between 1994 and 1998 in 1,674 US hospitals. Study results were published in the Journal of the American Medical Association (JAMA) on June 28, 2000, Vol. 283, No. 24, pages 3223-3229

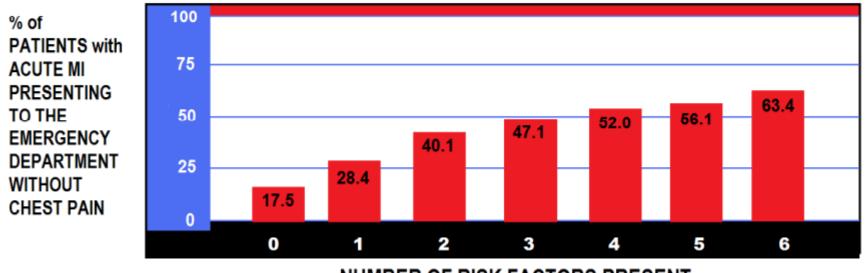
#### Common atypical complaints associated with AMI without chest pain include:

Malaise (weakness) Indigestion Nausea Dizziness Syncope

Fatigue Abdominal pain Cold sweats Elevated heart rate Dsypnea

#### BOOK PAGE: 70

#### Effect of Having Multiple Risk Factors for AMI Without Chest Pain



NUMBER OF RISK FACTORS PRESENT

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

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

# Integrated ECG:

### HEMODYNAMIC STATUS

- ABCs
- Shock

#### SYMPTOMS

- Chest Pain / Pressure
- Other ACS Symptoms
- ECG
  - 12 Lead
  - Single Lead "rhythm strip"

## Actions at the Scene

• If patient has ANY symptoms of ACS, get a

# STAT 12 Lead ECG

## EMS 12 Lead ECG

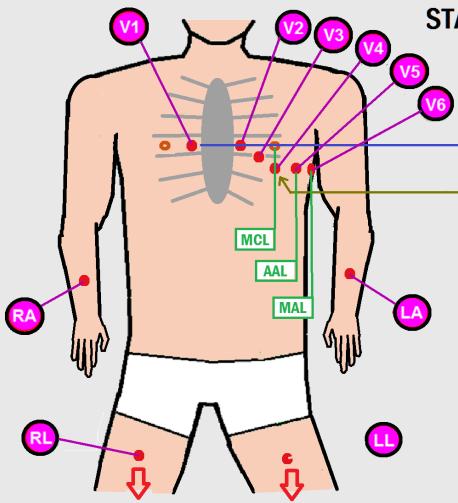


## 10 wires . . .

- 4 limb leads
- 6 chest ("V") leads



# **Obtaining the 12 Lead ECG**



#### STANDARD LEAD PLACEMENT ---12 LEAD ECG

4 th INTERCOSTAL SPACE

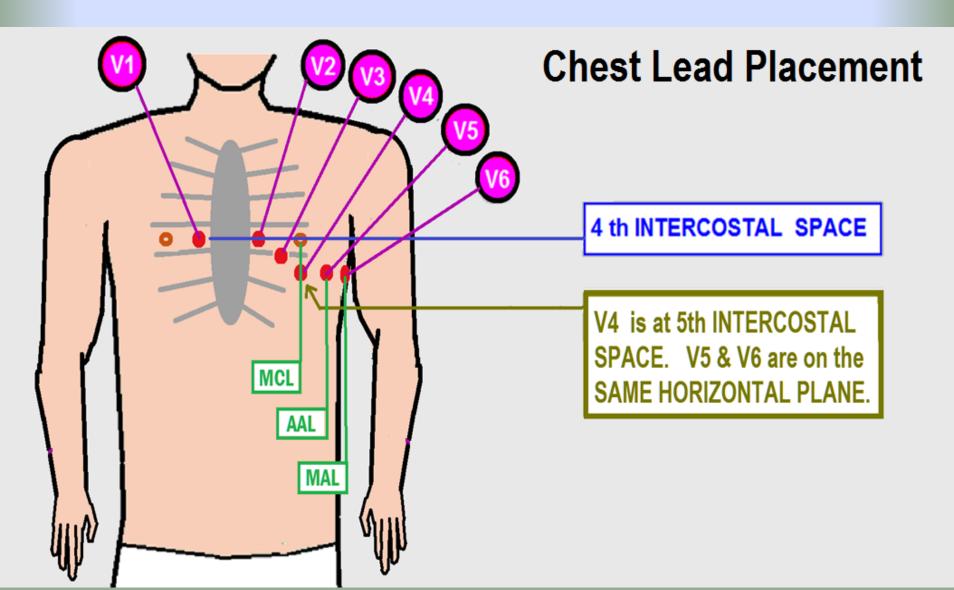
V4 is at 5th INTERCOSTAL SPACE. V5 & V6 are on the SAME HORIZONTAL PLANE.

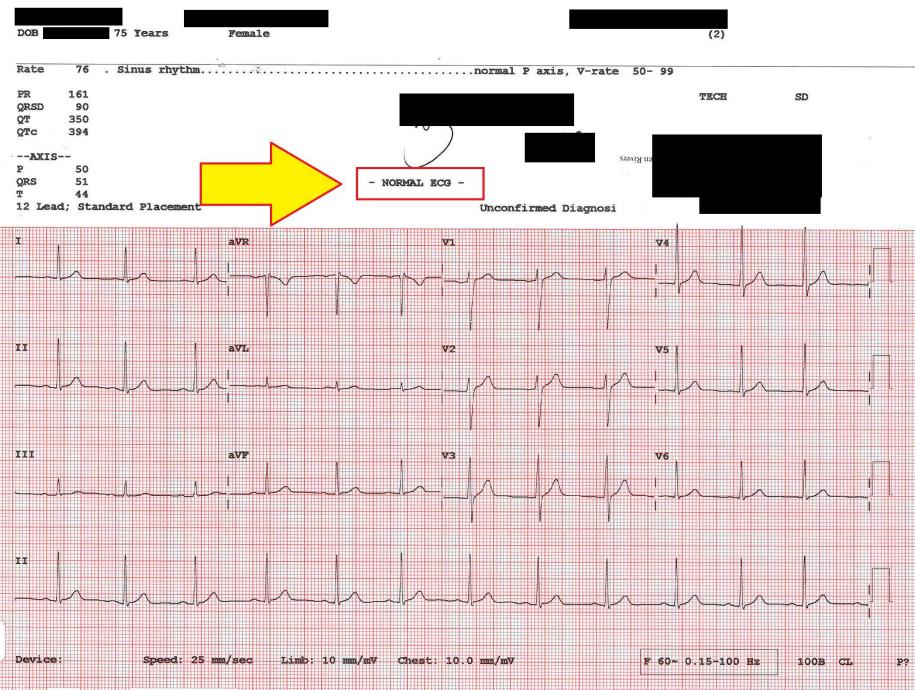
- PATIENT SHOULD LAY AS FLAT AS POSSILVE
- LIMB LEADS SHOULD BE PLACED AS DISTALLY AS POSSIBLE

# 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:

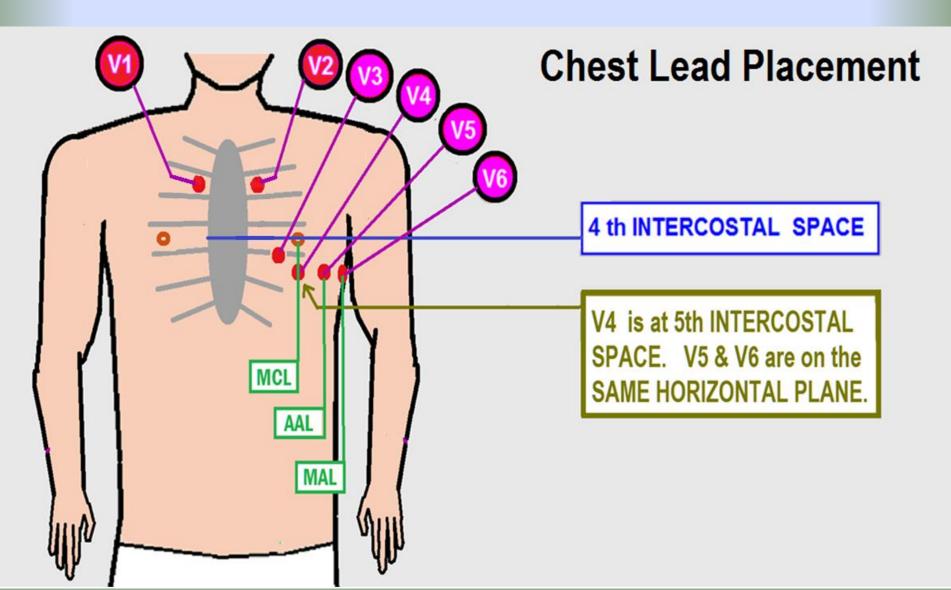


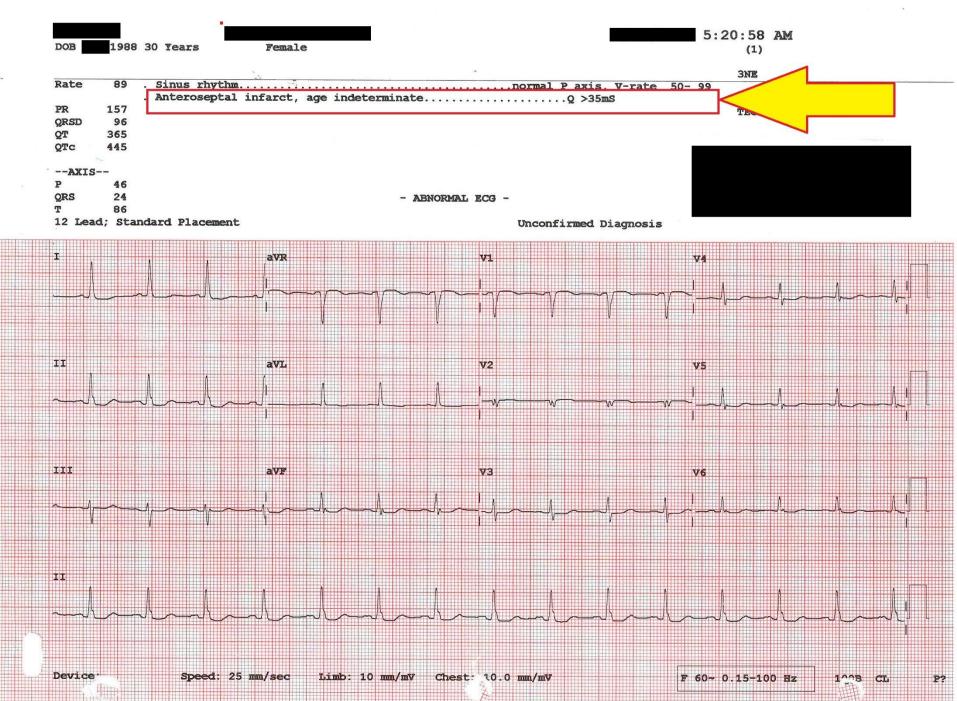


COVIDIEN Kendall

HEF 30768678

## **INCORRECT** Lead placement:





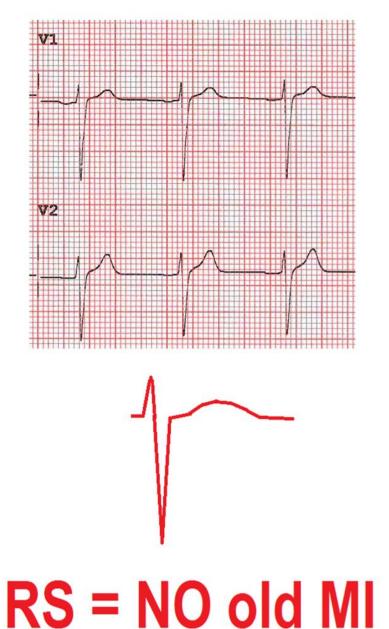
#### **AHA/ACC/HRS Scientific Statement**

#### **Recommendations for the Standardization and Interpretation of the Electrocardiogram** Part I: The Electrocardiogram and Its Technology

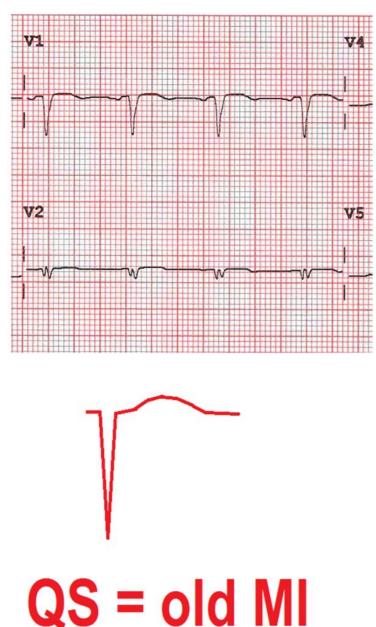
1.1

the often profound alterations in waveforms that can result from precordial electrode misplacement.<sup>85,86</sup> 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.<sup>87</sup> 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,<sup>88,89</sup>

#### **Correct Lead Placement**

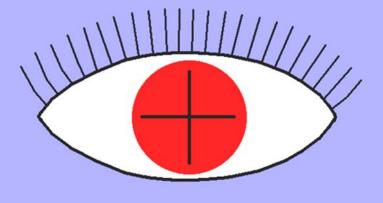


#### **Incorrect Lead Placement**

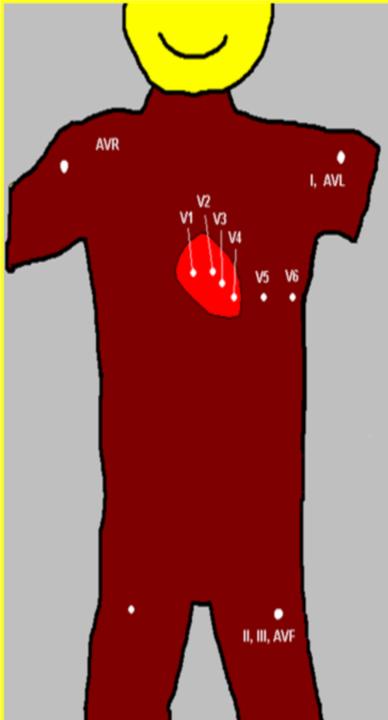


# What part of the HEART does each lead SEE ?

#### THE POSITIVE ELECTRODE



IS THE "EYE" . . .



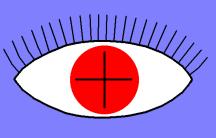
### AREAS VIEWED by 12 LEAD ECG



V5, V6

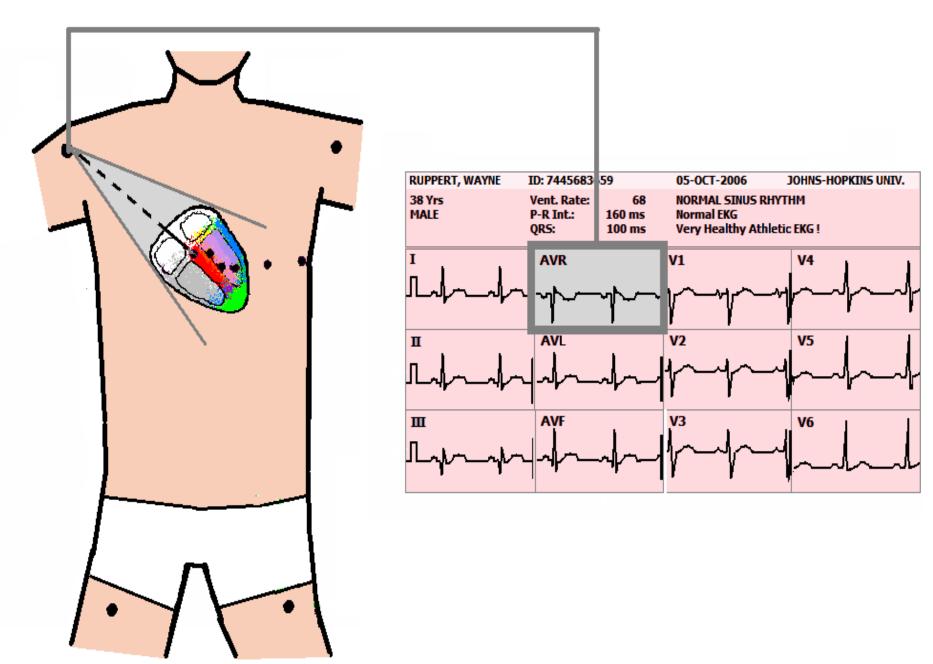
II, III, AVF

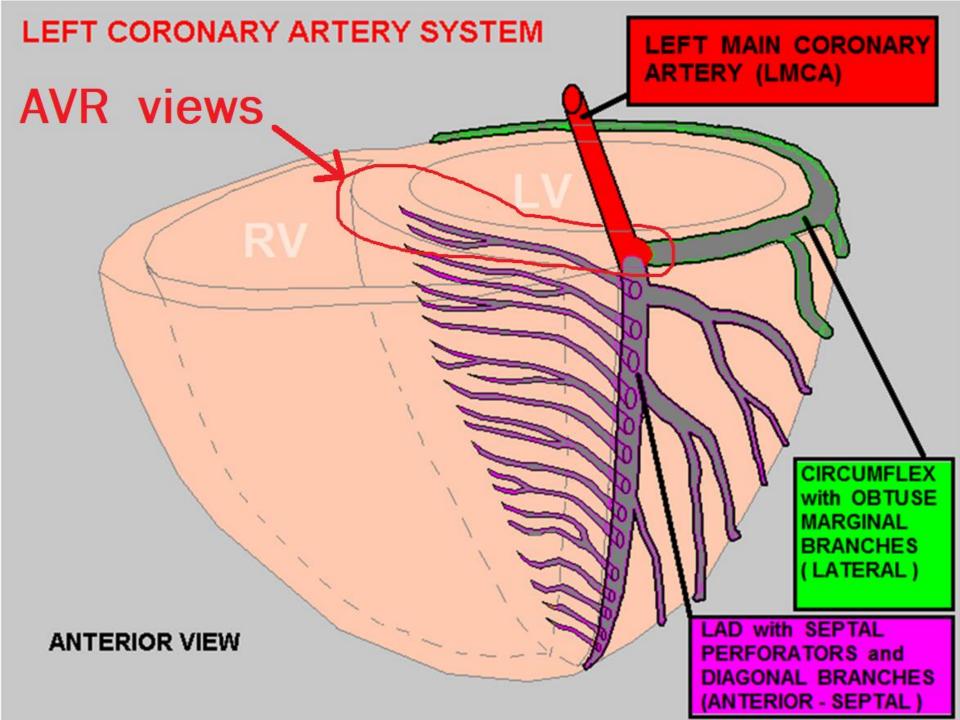
THE POSITIVE ELECTRODE



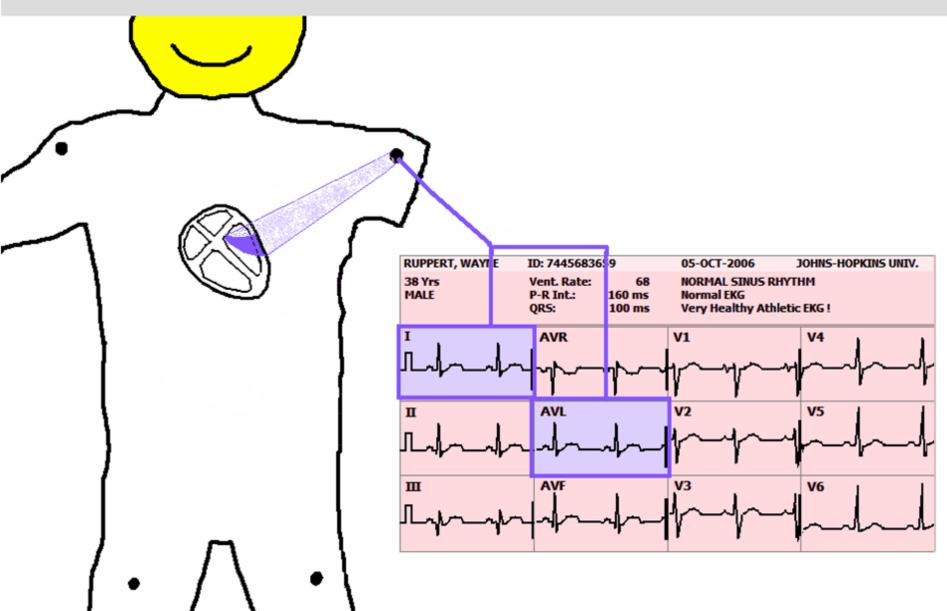
IS THE "EYE" . . .

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

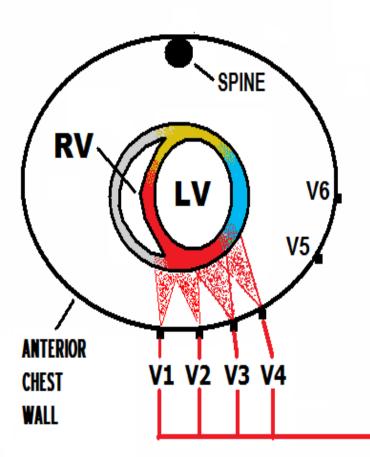




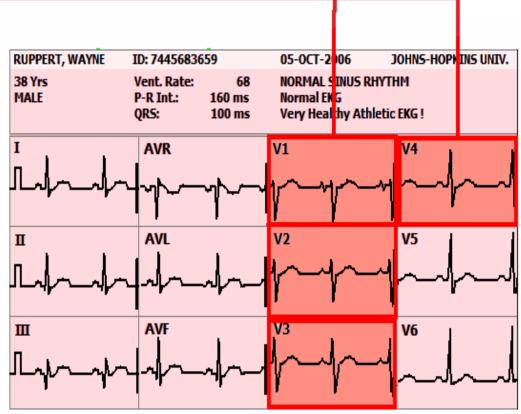
## LEADS I and aVL VIEW the LATERAL - ANTERIOR WALL

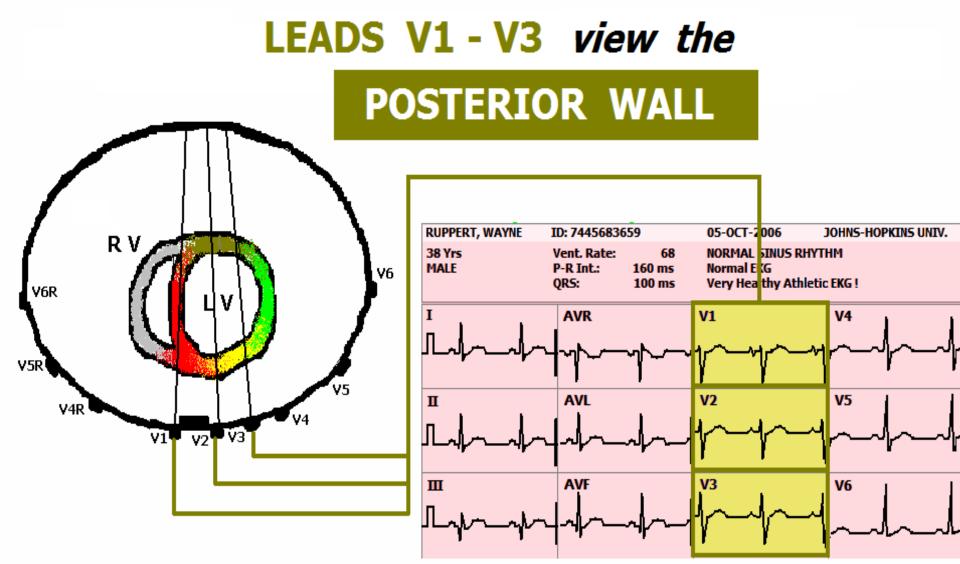


## V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL of the LEFT VENTRICLE



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

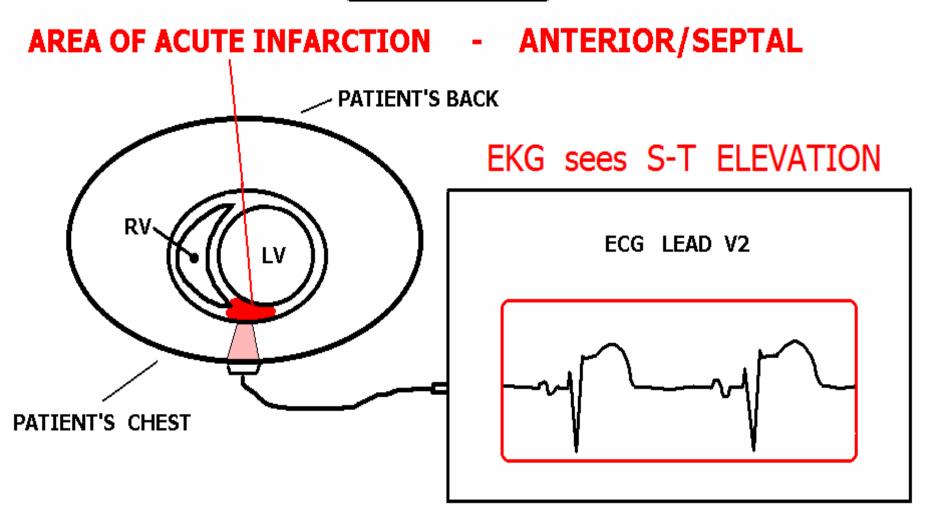




#### via RECIPROCAL CHANGES.

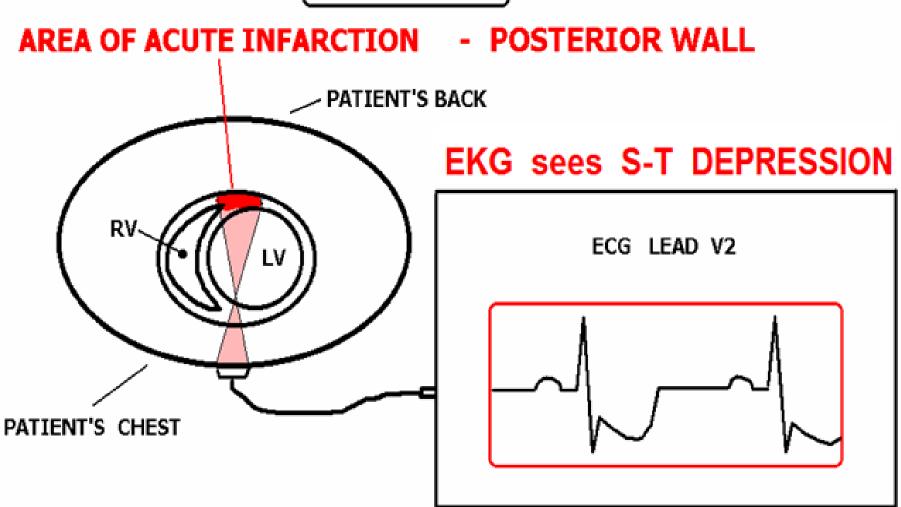
#### HOW EKG VIEWS INDICATIVE CHANGES

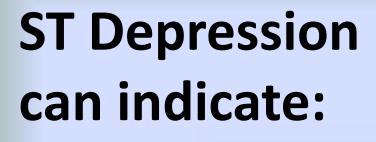
## EXAMPLE:

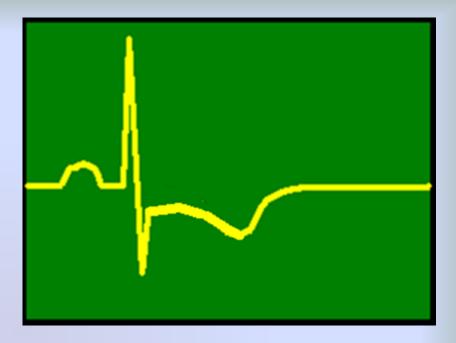


#### HOW EKG VIEWS RECIPROCAL CHANGES

### EXAMPLE:

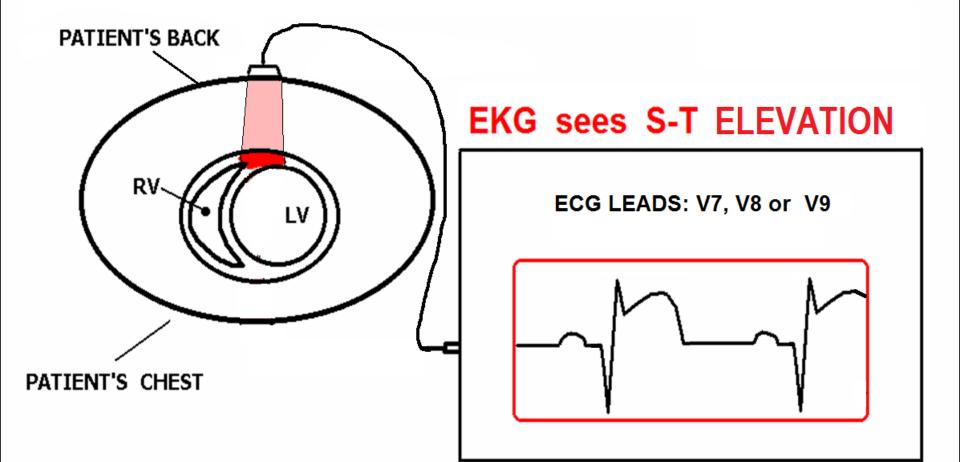




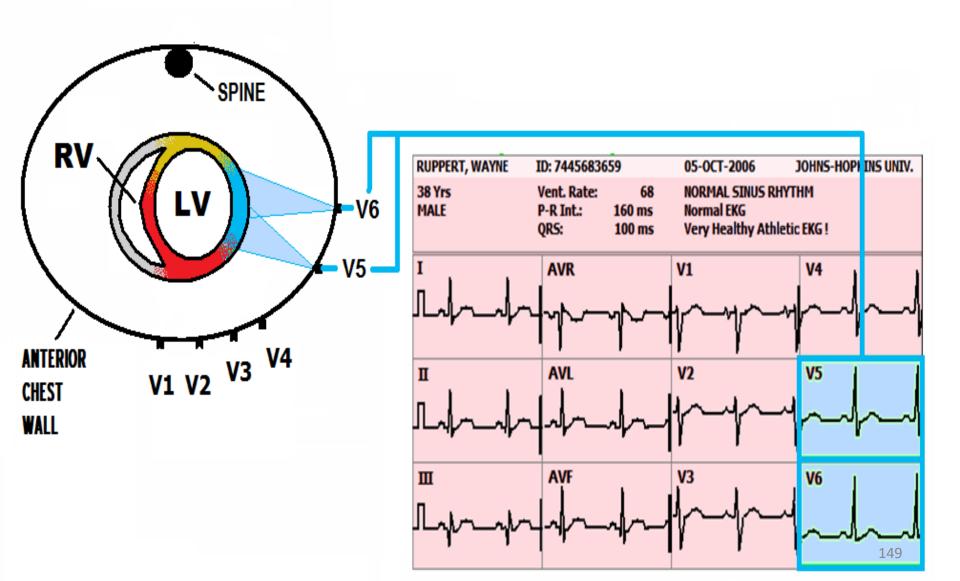


- ISCHEMIA
- "Partial-wall thickness" MI (NSTEMI)
- STEMI (in the opposite side of the heart)
- Other things (like RBBB, certain medications, etc).

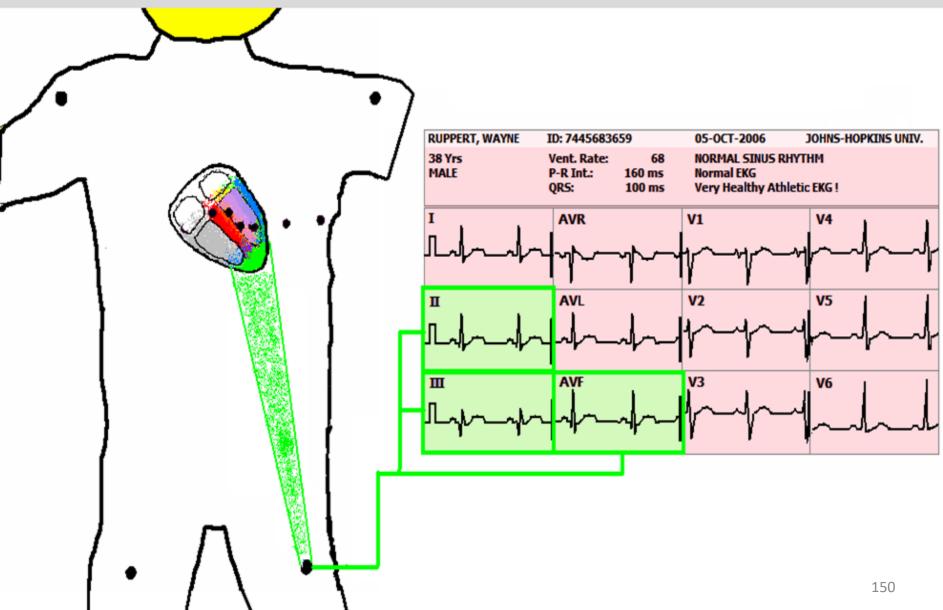
### If we put ECG leads on the BACK of a PATIENT who is having an ACUTE POSTERIOR WALL MI....

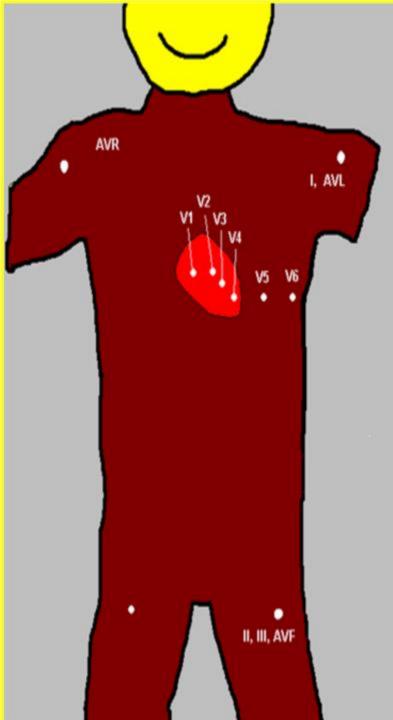


## V5 - V6 VIEW THE LATERAL WALL of the LEFT VENTRICLE



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





#### AREAS VIEWED by 12 LEAD ECG

AVR	BASILAR SEPTAL	
A) // I		

AVL, I LATERAL ANTERIOR

V1, V2 ANTERIOR

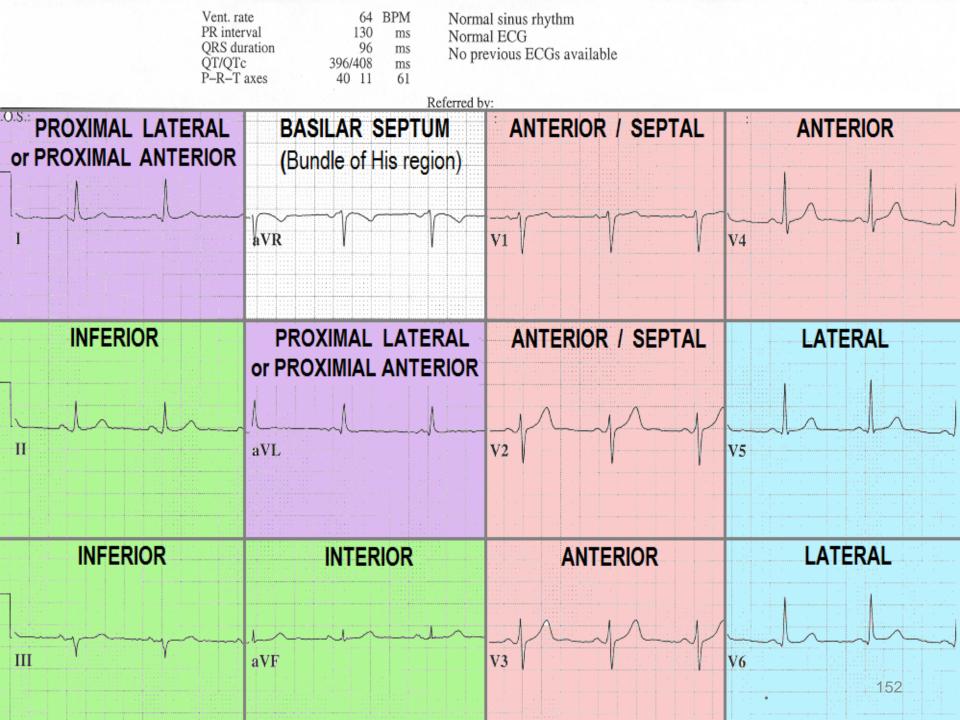
SEPTAL

POSTERIOR (recip.)

V3, V4	ANTERIOR

V5, V6 LATERAL

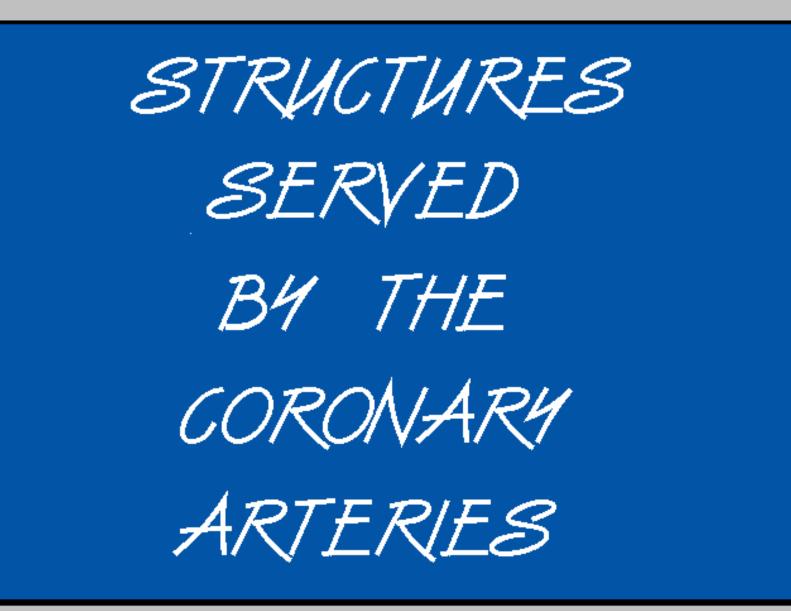
II, III, AVF INFERIOR

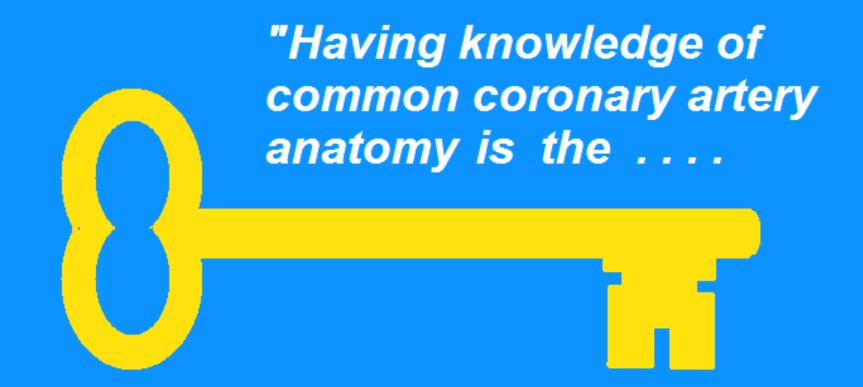


THE CORONARY









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

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

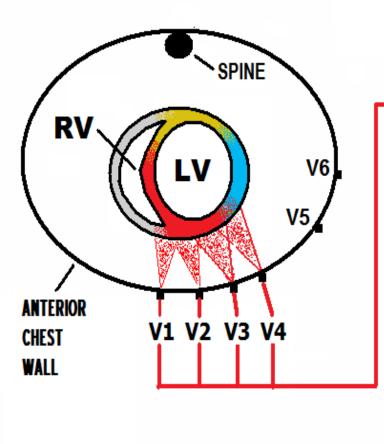
# The 12 Lead ECG becomes your "erystal ball !!"



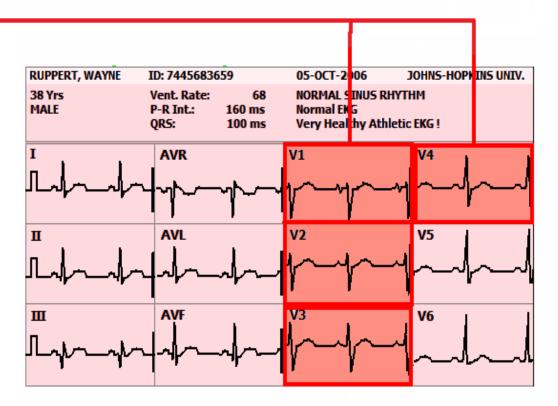
## **INTERPRET THE EKG, THEN:**

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!

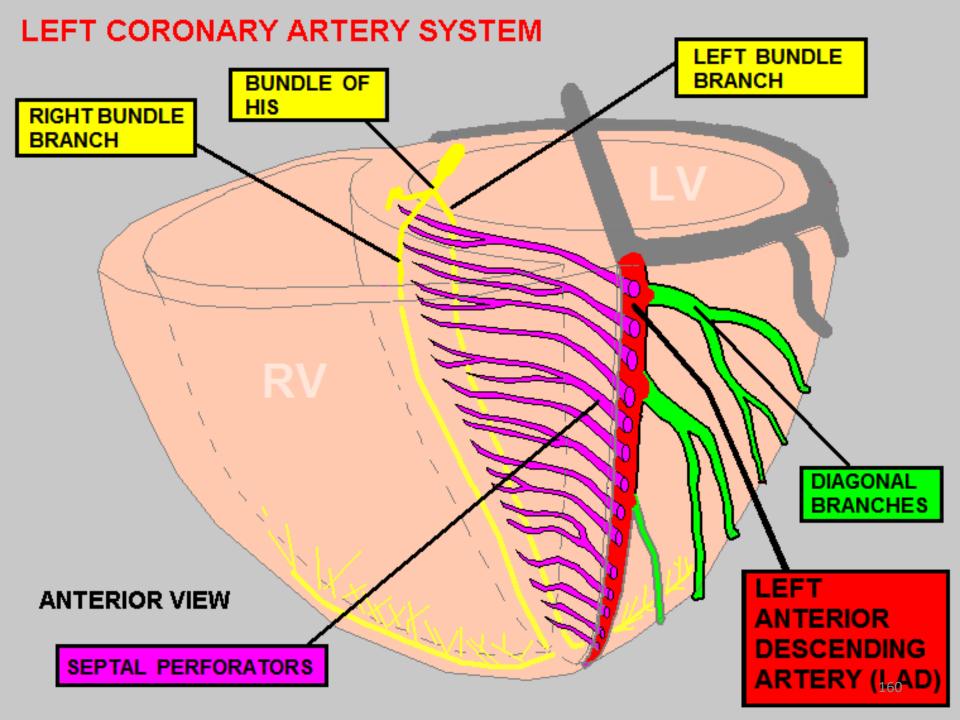
### V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL of the LEFT VENTRICLE

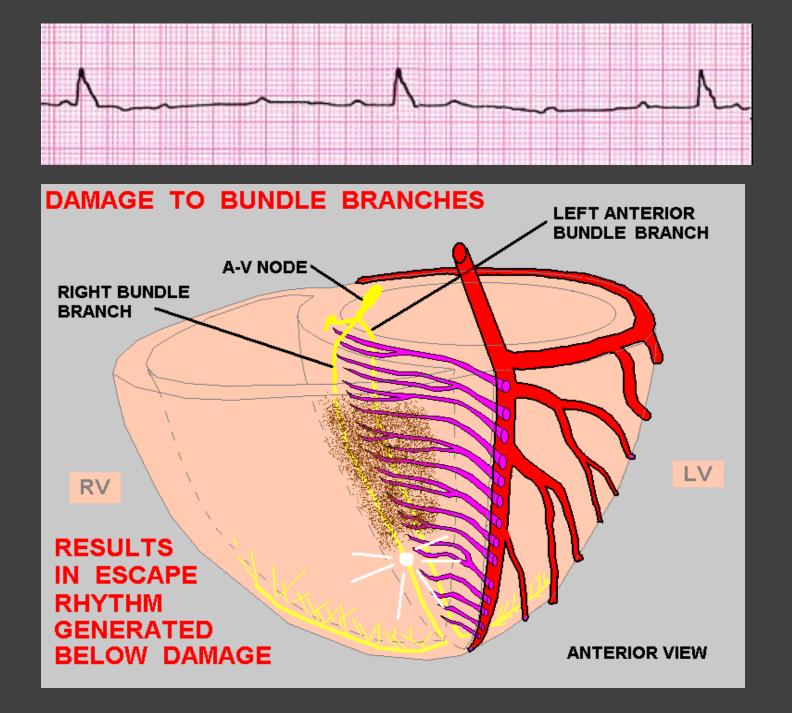


V1, V2 - ANTERIOR / SEPTAL V3, V4 - ANTERIOR



Which Coronary Artery typically Supplies the ANTERIOR WALL? 159

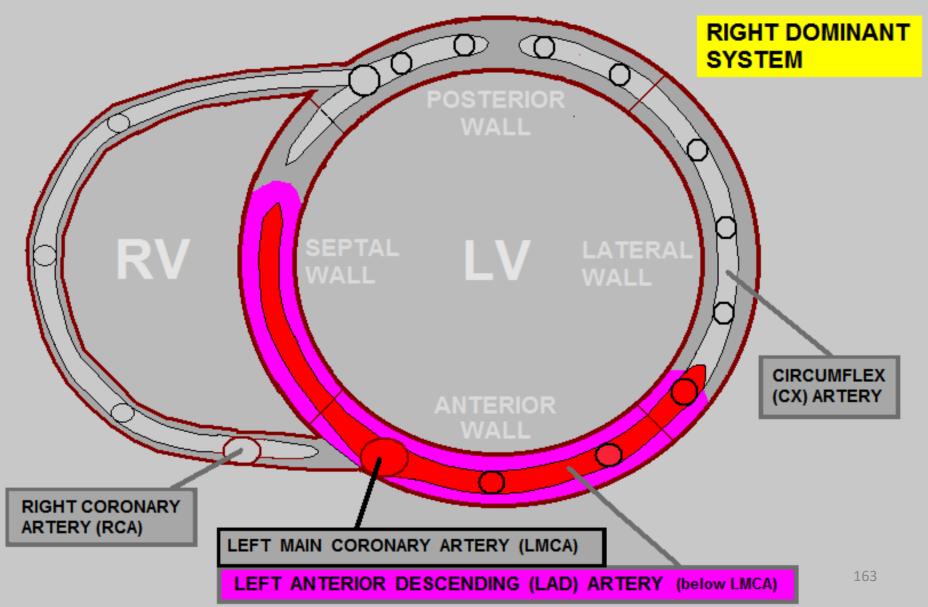




cutaway view of the

#### LEFT ANTERIOR DESCENDING ARTERY (LAD)

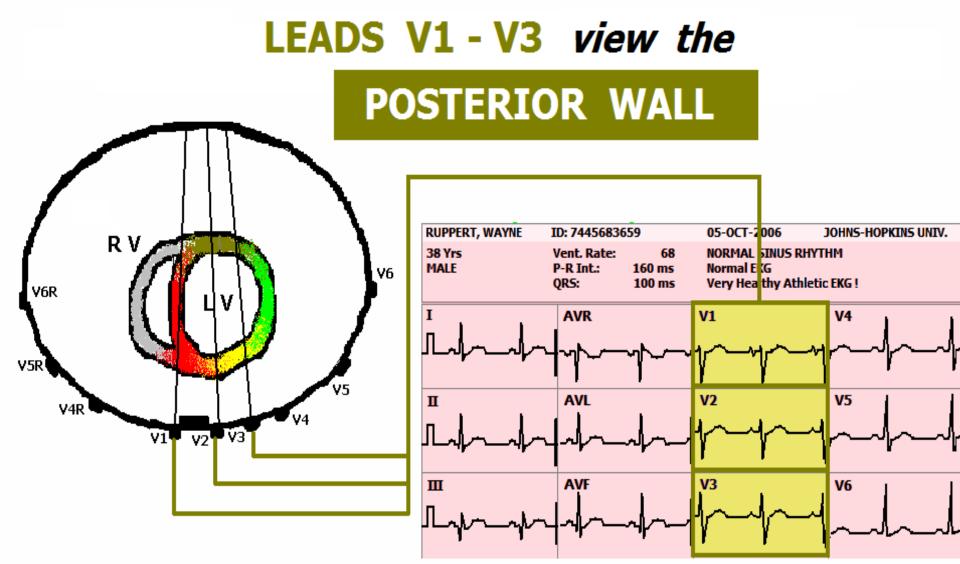
#### SUPPLIES APPROX. 35 - 45% of the LV MUSCLE MASS





# LEFT ANTERIOR DESCENDING ARTERY (LAD)

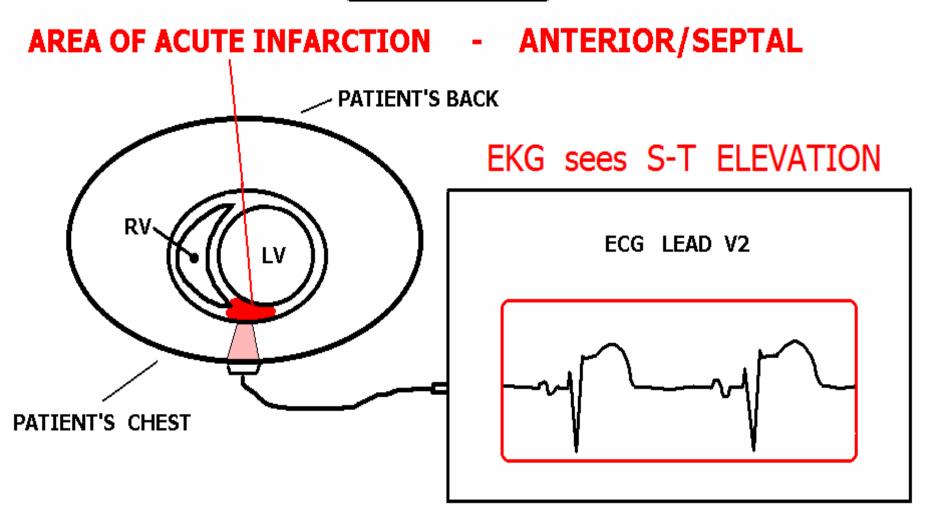
BUNDLE OF HIS
BUNDLE BRANCHES ()
35 - 45 % OF LV MUSCLE MASS
ANTERIOR WALL
SEPTAL WALL (anterior 2/3)



## via RECIPROCAL CHANGES.

## HOW EKG VIEWS INDICATIVE CHANGES

## EXAMPLE:



# ST Depression in Leads V1 – V4:



Direct view of ISCHEMIA (anterior wall)

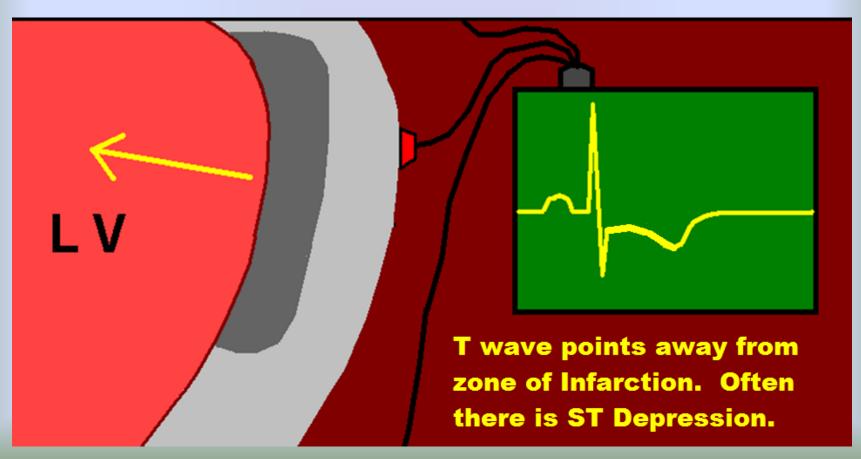




- Direct view of ISCHEMIA (anterior wall)
- Direct view of NSTEMI (anterior wall)

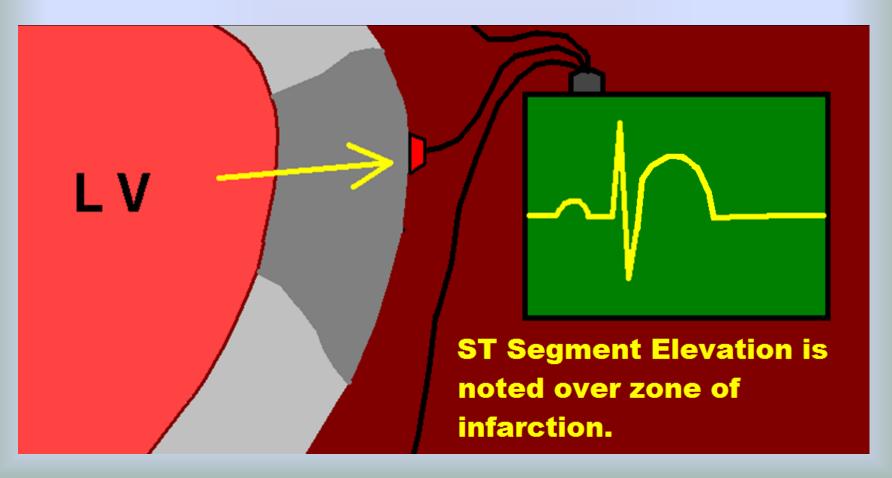
# Non-STEMI (NSTEMI)

Non-ST Segment Elevation Myocardial Infarction. "sub-endocardial MI" . . . "partial wall thickness"



# STEMI

ST Segment Elevation Myocardial Infarction.



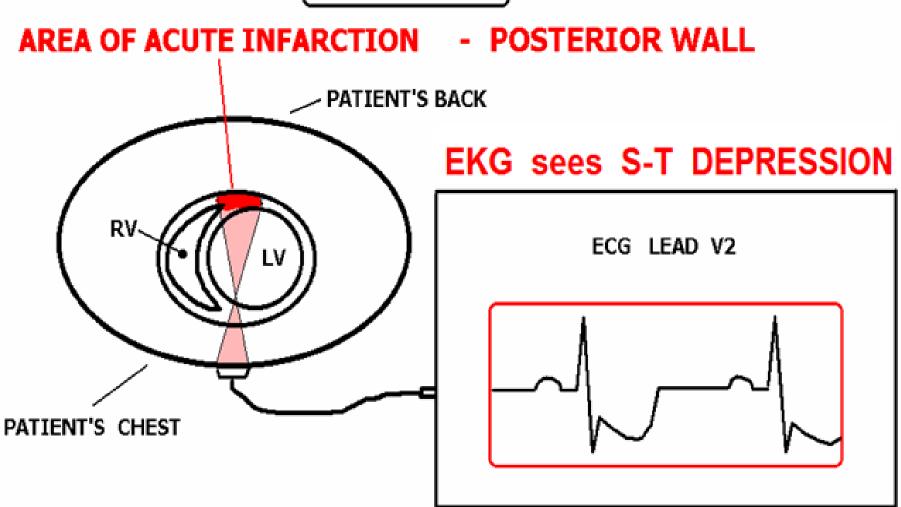




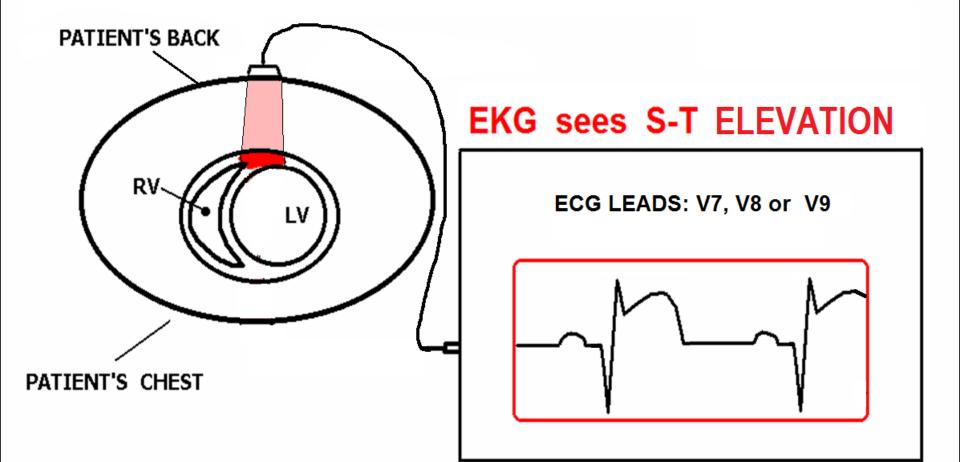
- Direct view of ISCHEMIA (anterior wall)
- Direct view of NSTEMI (anterior wall)
- Reciprocal view of STEMI (opposite side of heart - posterior wall)

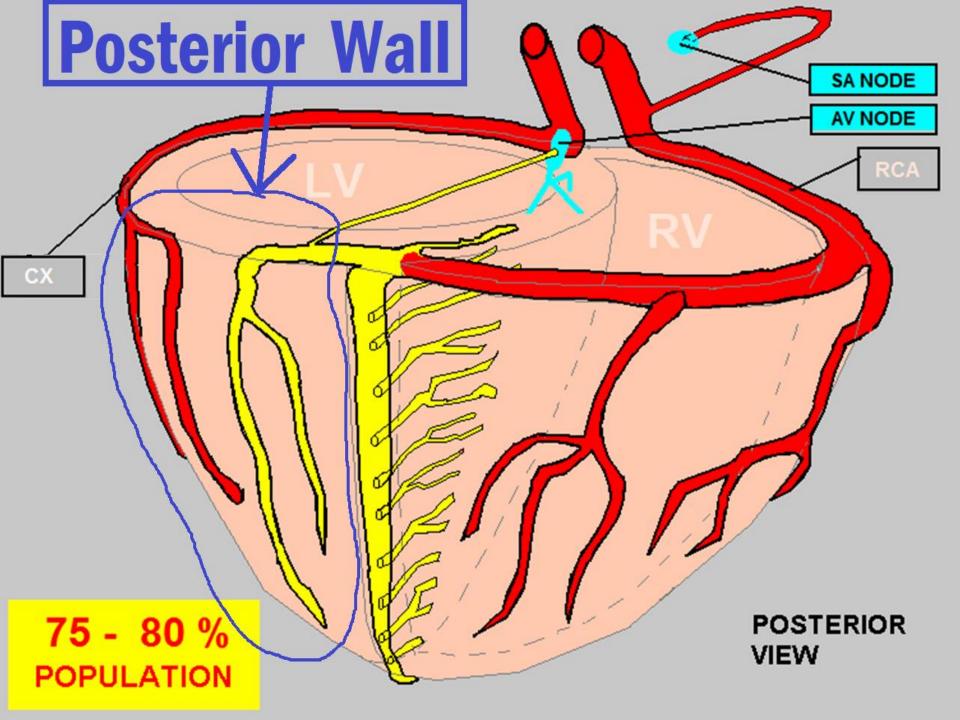
## HOW EKG VIEWS RECIPROCAL CHANGES

## EXAMPLE:

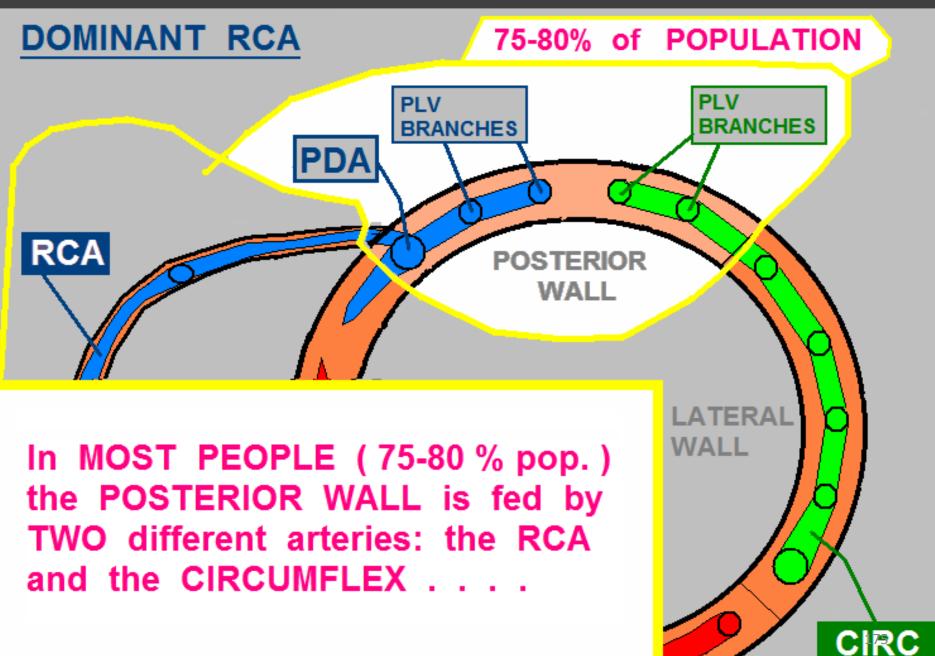


## If we put ECG leads on the BACK of a PATIENT who is having an ACUTE POSTERIOR WALL MI....

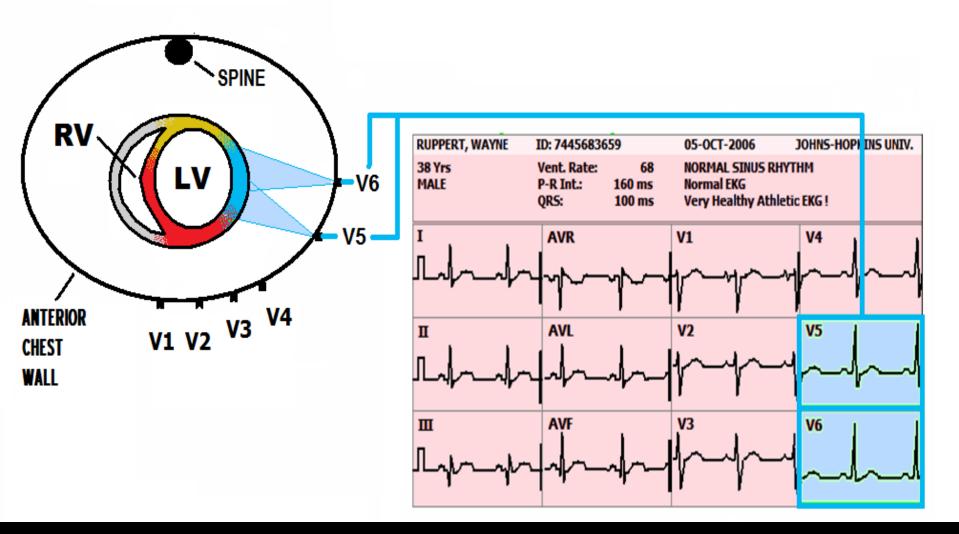




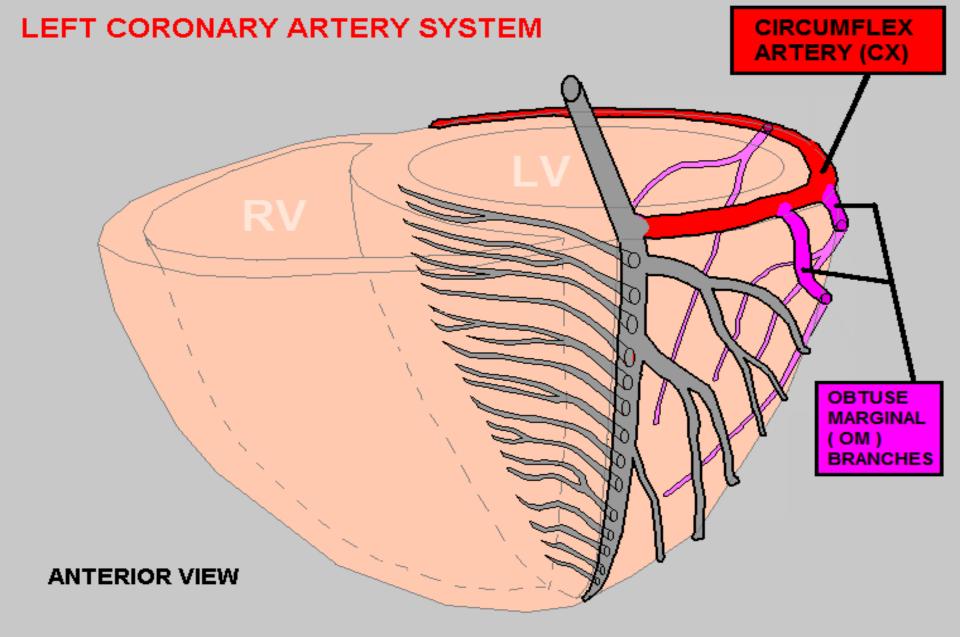
### POSTERIOR WALL BLOOD SUPPLY



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



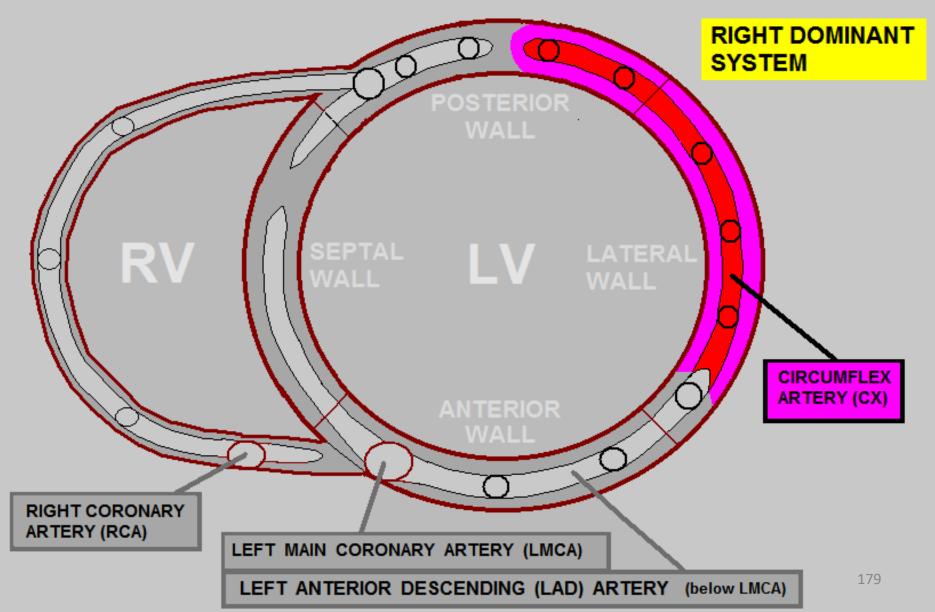
Which Coronary Artery typically Supplies the LATERAL WALL? 176

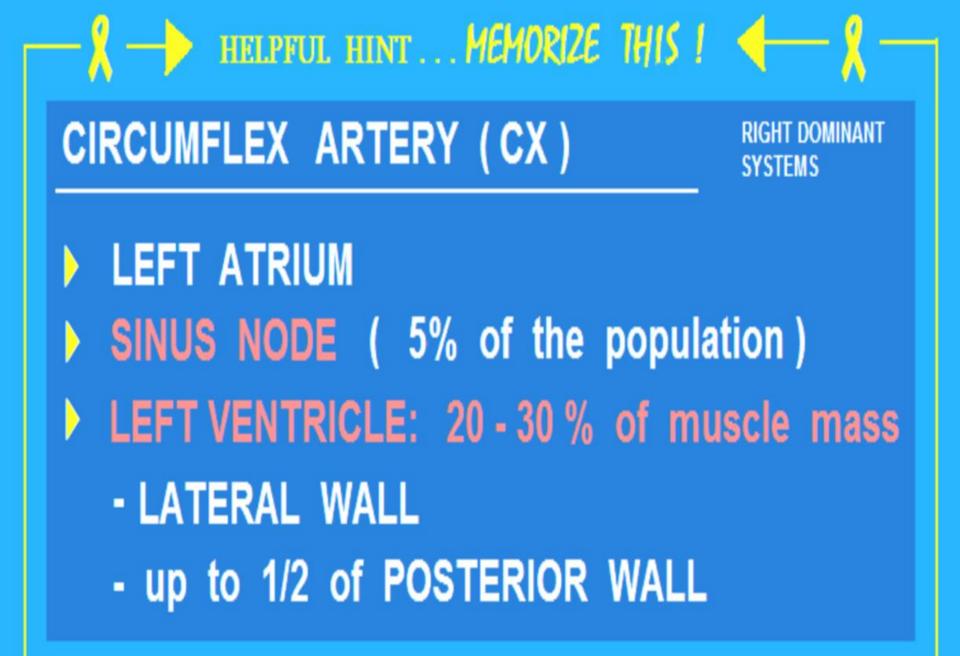


cutaway view of the

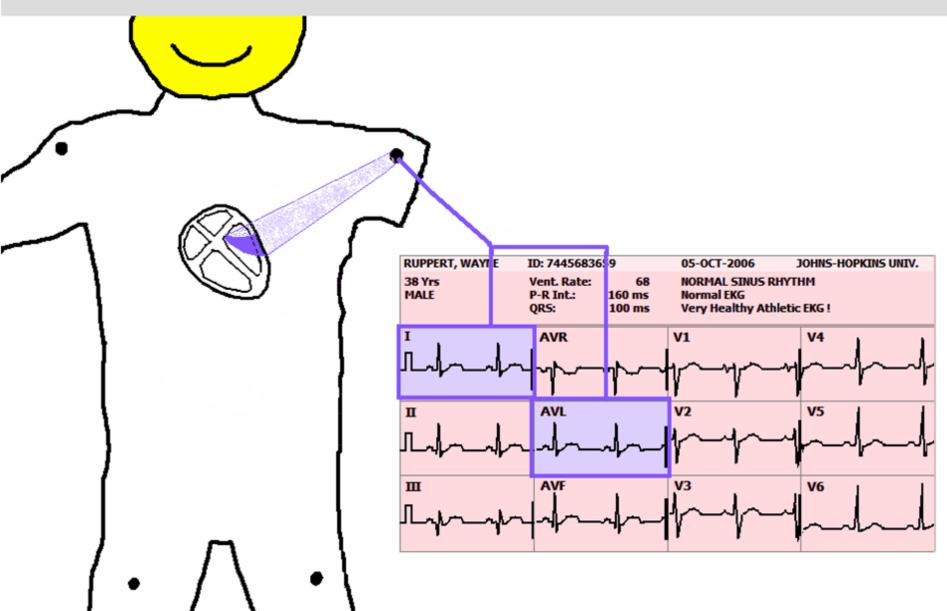
### CIRCUMFLEX ARTERY (CX) DISTRIBUTION

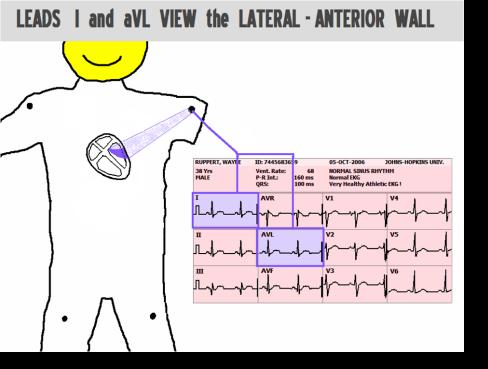
#### SUPPLIES 20 - 30 % of the LV MUSCLE MASS



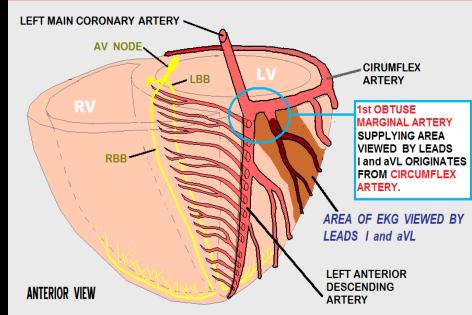


# LEADS I and aVL VIEW the LATERAL - ANTERIOR WALL

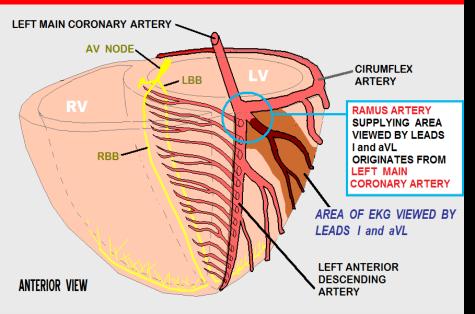




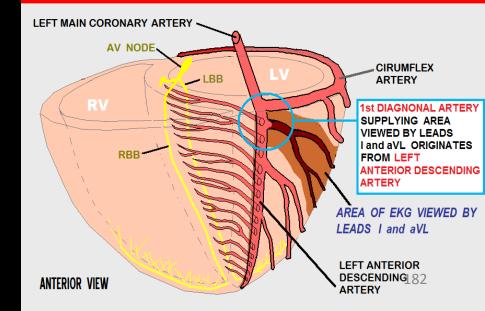
#### OCCLUSION of OBTUSE MARGINAL ARTERY



#### OCCLUSION of RAMUS ARTERY



#### OCCLUSION of DIAGONAL ARTERY



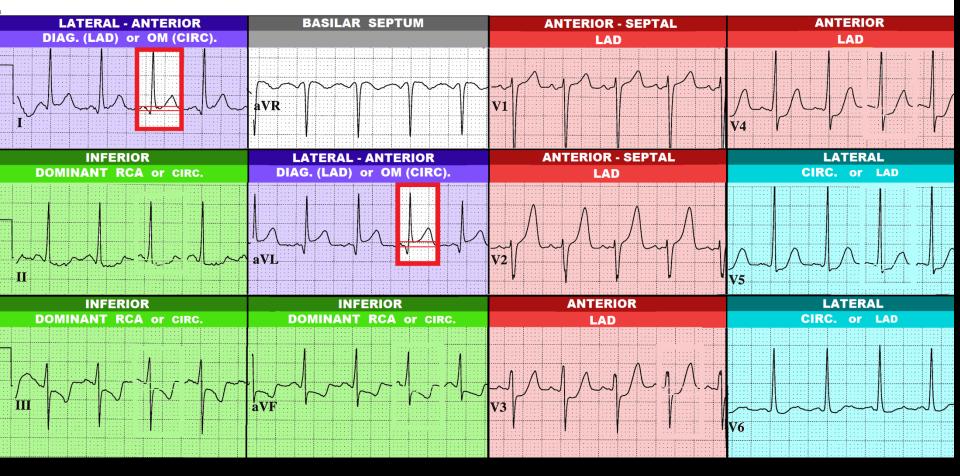
46 yr	Vent. rate	109	BPM
Female	PR interval	132	ms
	QRS duration	82	ms
Room:ER	QT/QTc	346/465	ms
	P–R–T axes	60 11	-32

Sinus tachycardia

Left ventricular hypertrophy with repolarization abnormality

ST elevation consider lateral injury or acute infarct

\*\*\* \*\* \*\* \*\* \* ACUTE MI \*\* \*\* \*\*

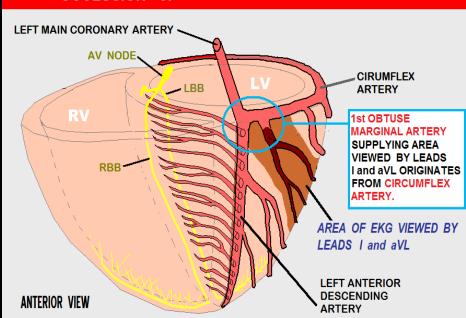


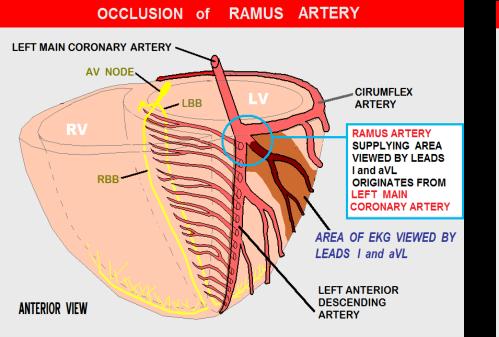
## ST Segment elevation ONLY in Leads I and aVL

ST SEGMENT ELEVATION

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

- RAMUS BRANCH
- 1<sup>st</sup> DIAGONAL off of LAD
- 1<sup>st</sup> OBTUSE MARGINAL off of CIRCUMFLEX





#### LEFT MAIN CORONARY ARTERY AV NODE UV CIRUMFLEX ARTERY SUPPLYING AREA VIEWED BY LEADS I and aVL ORIGINATES FROM LEFT ANTERIOR DESCENDING ARTERY

ANTERIOR VIEW

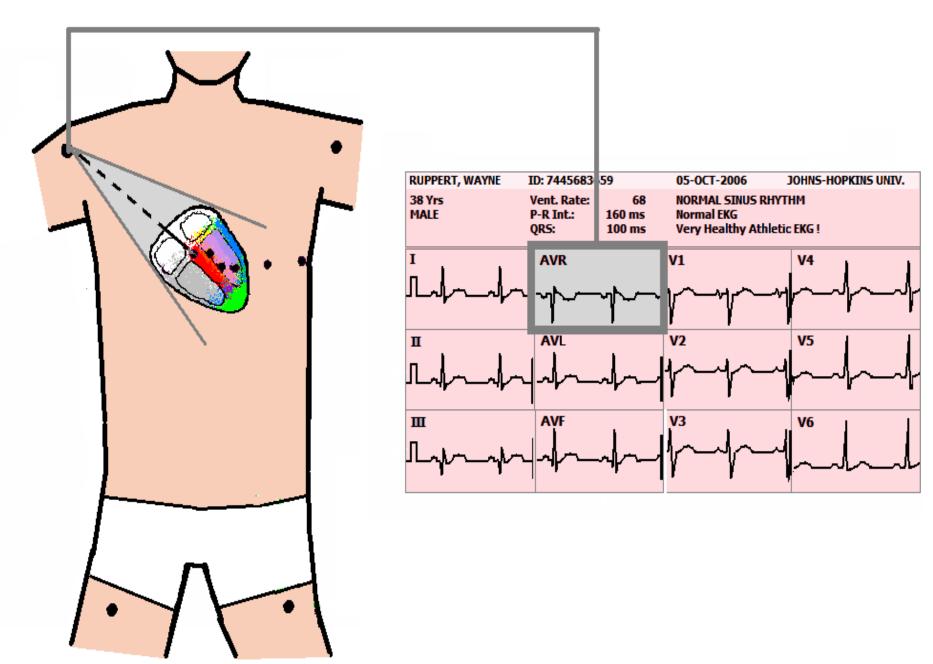
OCCLUSION of DIAGONAL ARTERY

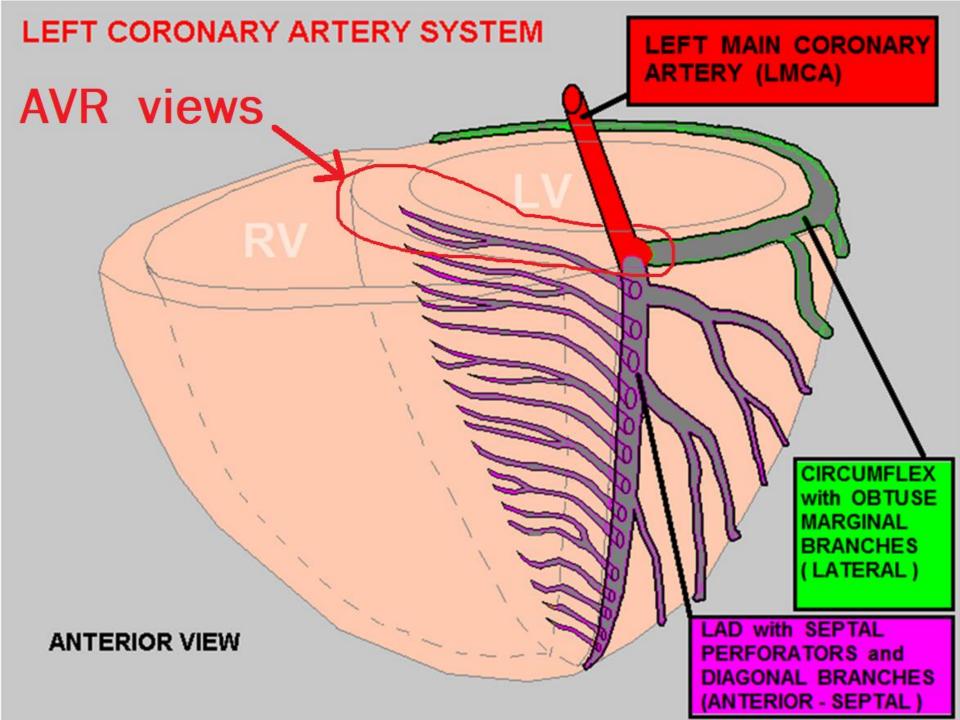
LEADS I and aVL

LEFT ANTERIOR DESCENDING 84 ARTERY

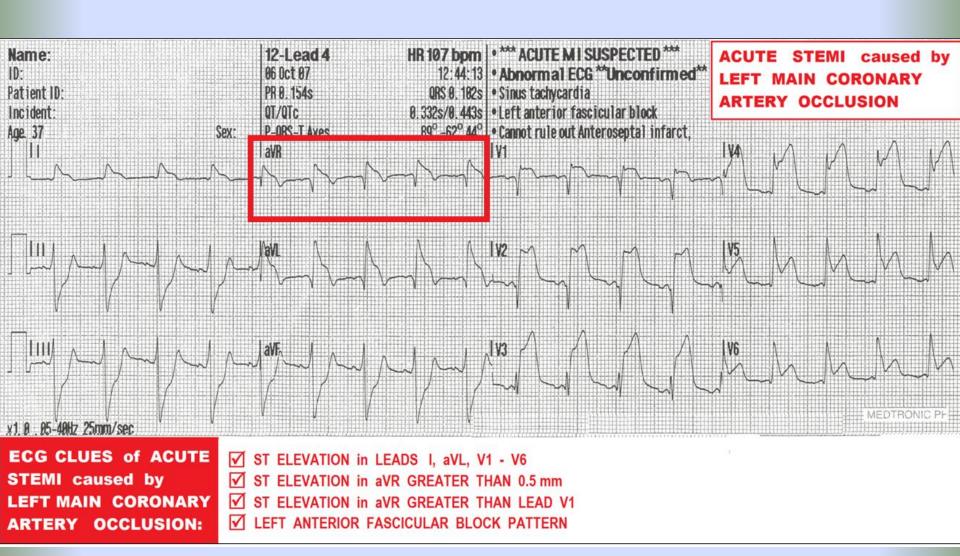
#### OCCLUSION of OBTUSE MARGINAL ARTERY

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

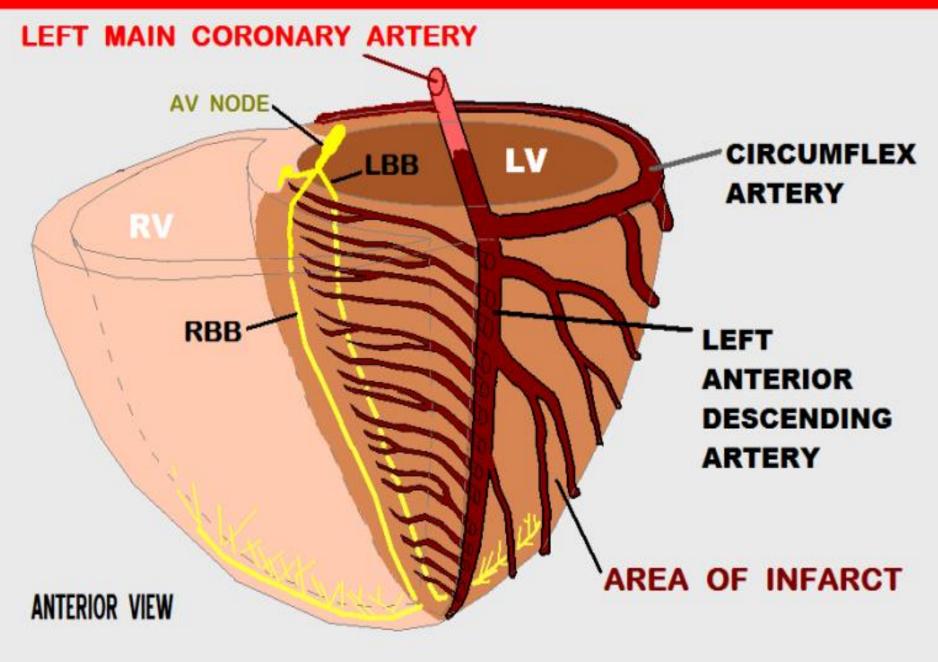




When LEAD AVR shows ST Elevation: **STEMI:** consider occlusion of the Left Main Coronary Artery.



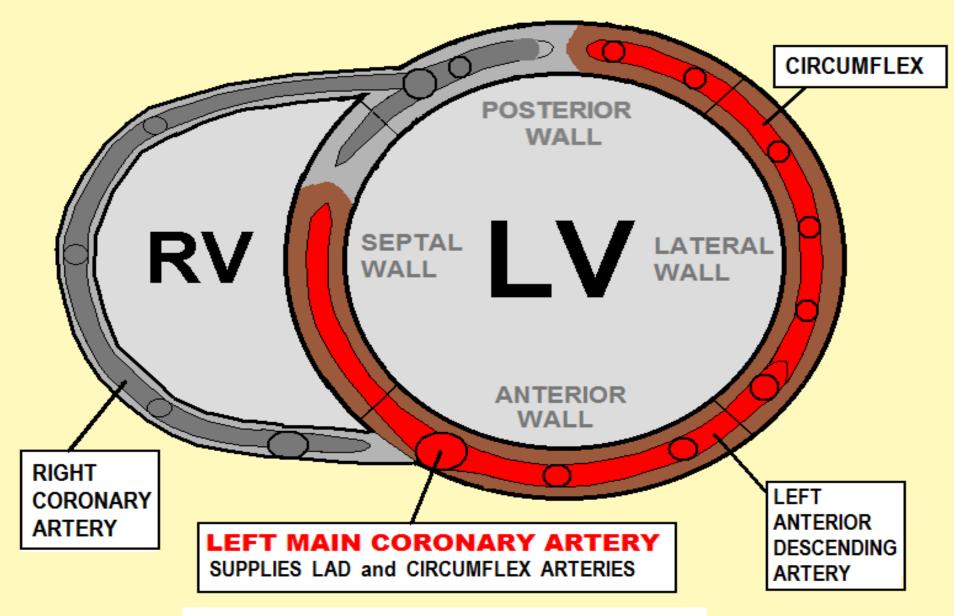
### **OCCLUSION** of the LEFT MAIN CORONARY ARTERY



## TOTAL OCCLUSION of the LEFT MAIN CORONARY ARTERY

## The LEFT MAIN CORONARY ARTERY

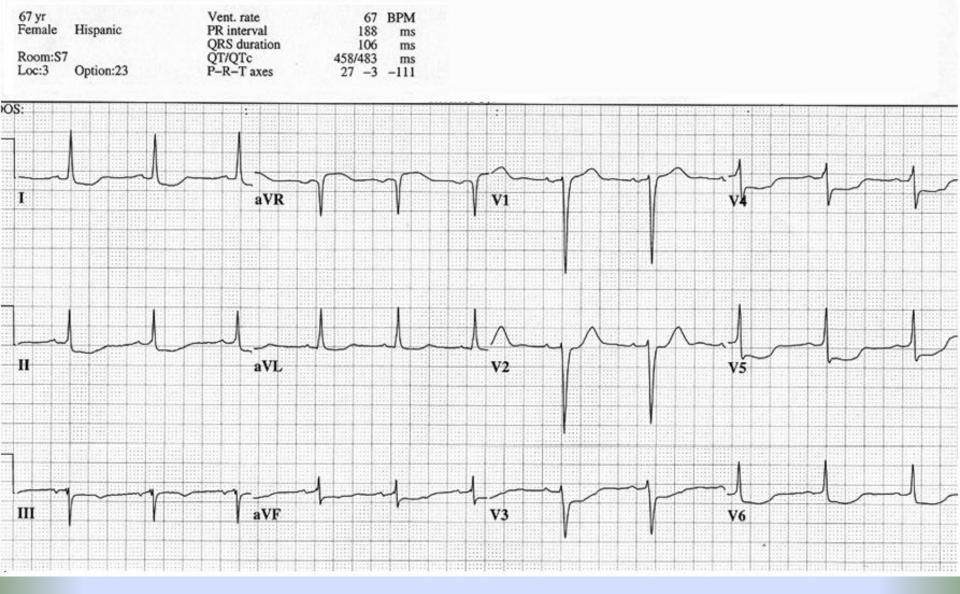
### SUPPLIES 75 - 100 % of the LEFT VENTRICULAR MUSCLE MASS



When LEAD AVR shows ST Elevation: **STEMI:** consider occlusion of the Left Main Coronary Artery.

When LEAD AVR shows ST **Elevation**: **NSTEMI** and **Unstable Angina** consider LMCA **Occlusion – or TRIPLE VESSEL DISEASE** 

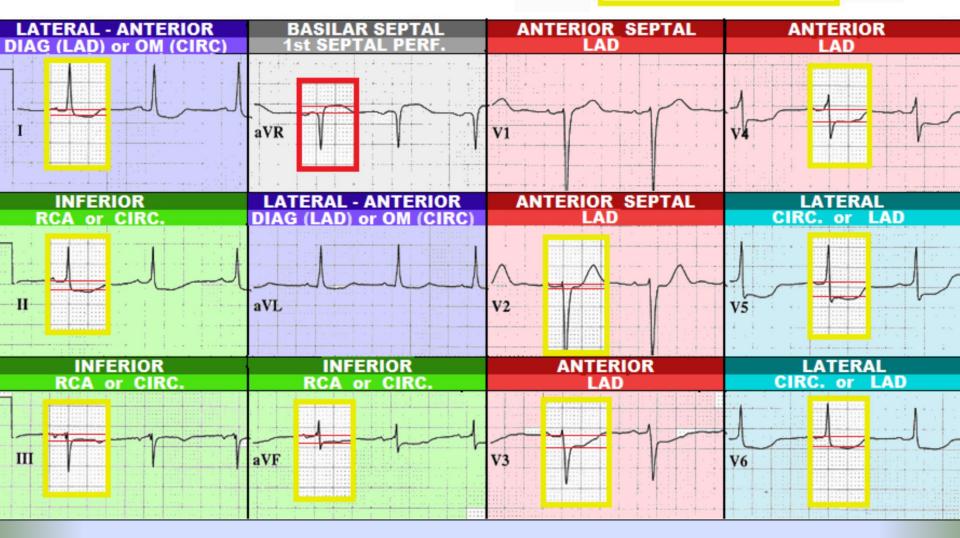
In patients without STEMI, ST **Elevation in AVR, when seen** with global indications of ischemia (ST Depression in 8 leads or more), is indicative of advanced multi-vessel disease or significant Left Main **Coronary Artery stenosis** 



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

#### ST SEGMENT ELEVATION

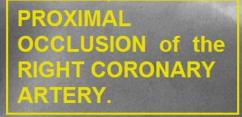
#### ST SEGMENT DEPRESSION



# **GLOBAL ISCHEMIA**

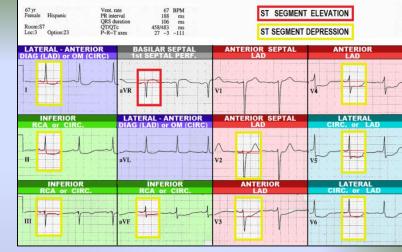
- ST Elevation Lead aVR
- ST Depression in 8 or more other Leads
- Indicates either SUB-TOTALLY OCCLUDED LEFT MAIN CORONARY ARTERY – or – TRIPLE VESSEL DISEASE.
- MOST PATIENTS WITH THIS ECG PRESENTATION REQUIRE OPEN HEART SURGERY.

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



RIGHT CORONARY ARTERY filling retrograde via COLLATERAL ARTERIES.

COLLATERAL CIRCULATION from SEPTAL PERFORATORS to RCA DISTRIBUTION.

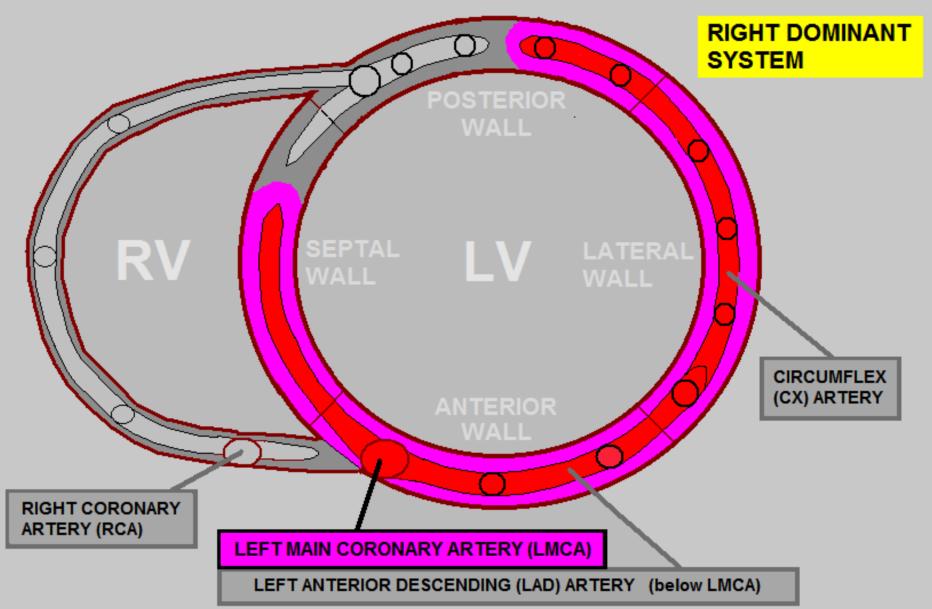


## SUB-TOTAL OCCLUSION IF CIRCUMFLEX ARTERY.

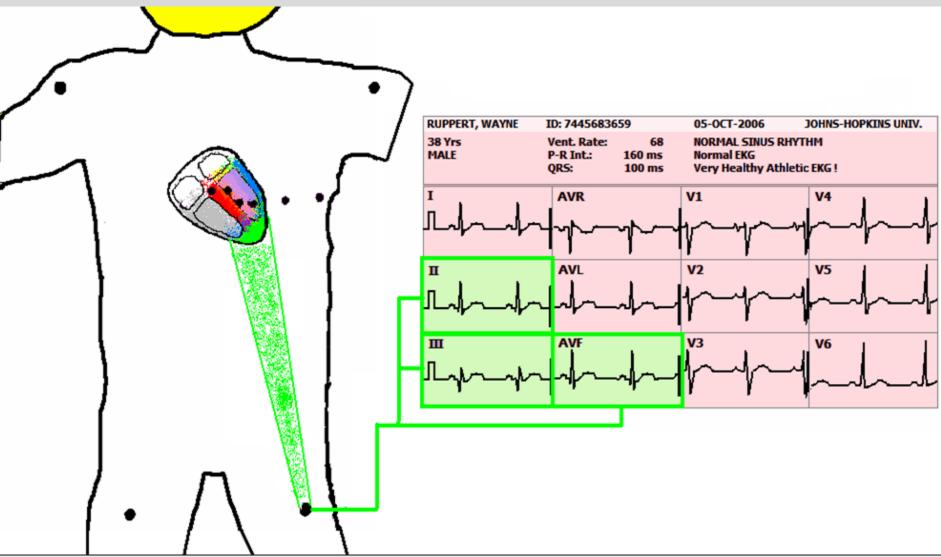
cutaway view of the

### LEFT MAIN CORONARY ARTERY (LMCA)

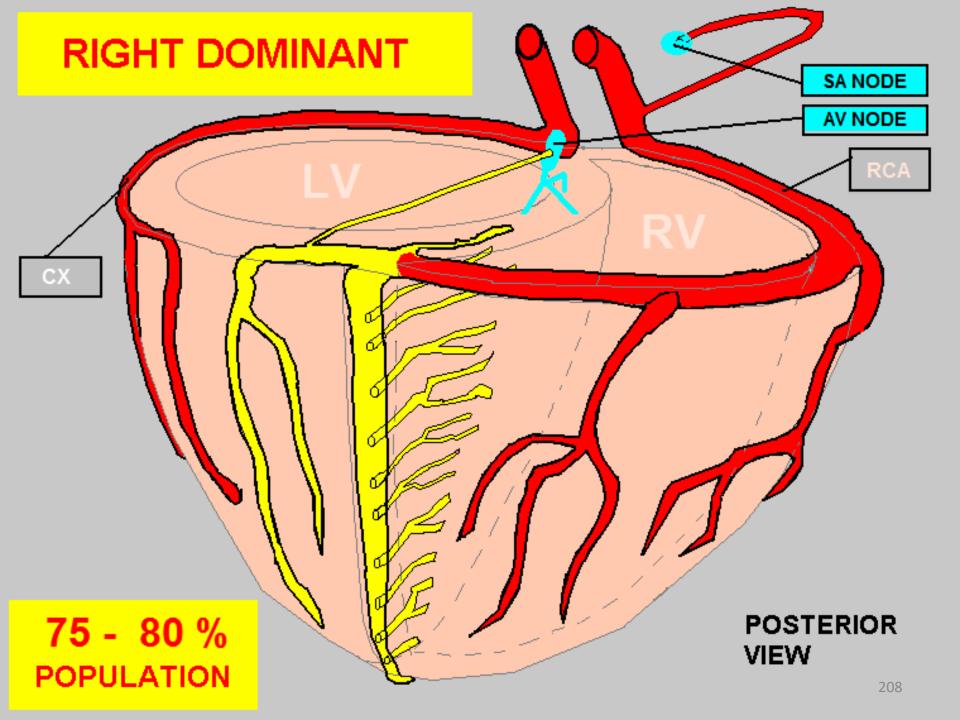
### **GP** SUPPLIES APPROXIMATELY 75% OF LV MUSCLE MASS



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

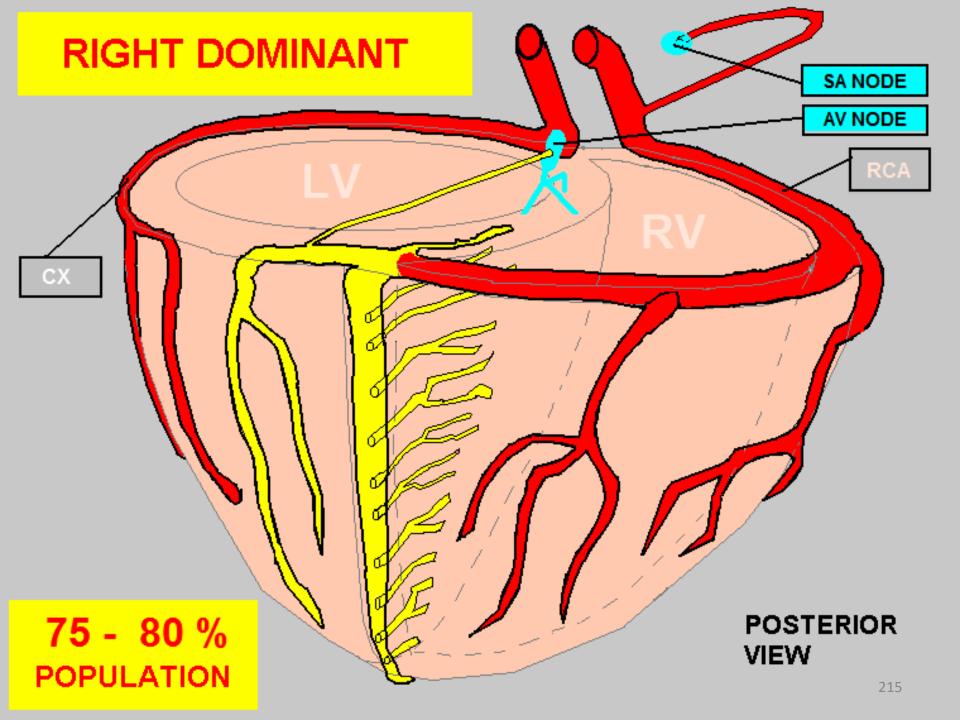


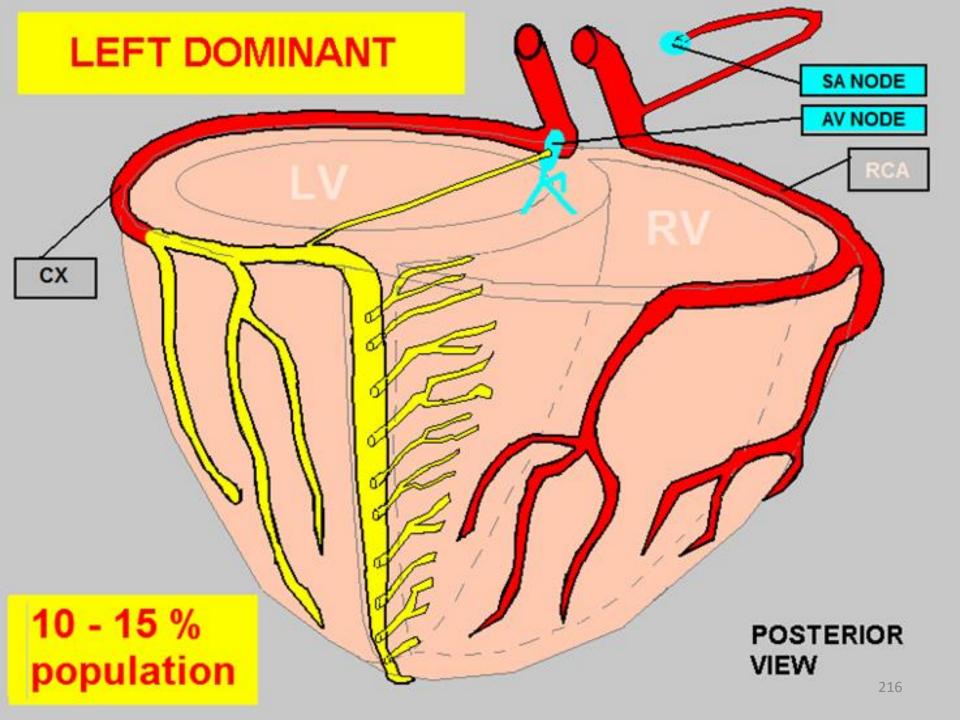
Which CORONARY ARTERY usually supplies the INFERIOR WALLo?

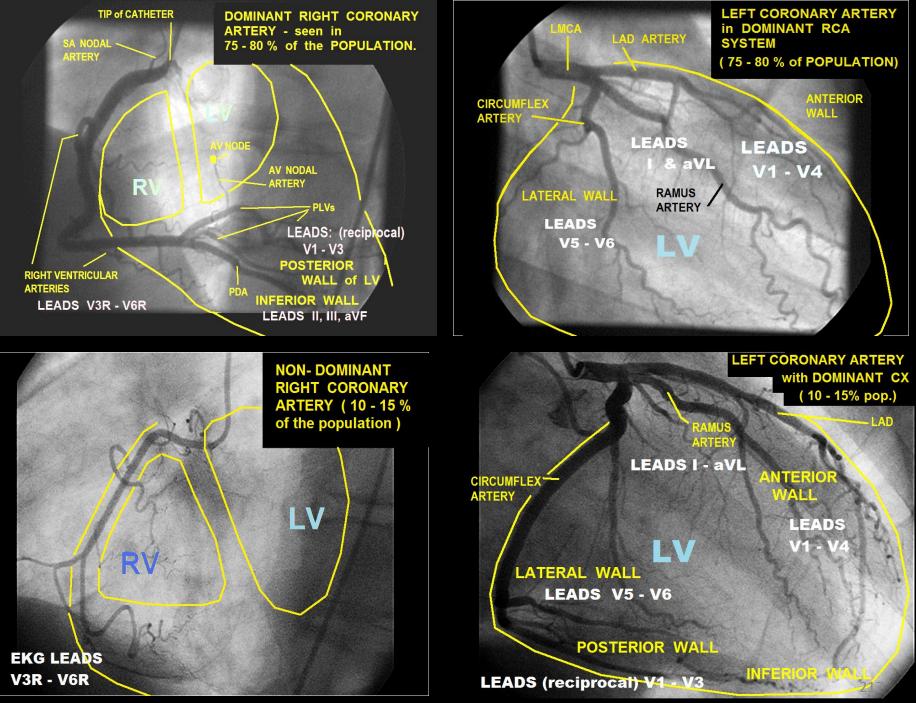


– 🕺 — 🔶 HELPFUL HINT ... MEMORIZE THIS I 🛛 🔶 🕺 **RIGHT CORONARY ARTERY (RCA) RIGHT DOMINANT** SYSTEMS RIGHT ATRIUM SINUS NODE (55% of the population) RIGHT VENTRICLE - 100 % of muscle mass LEFT VENTRICLE: 15 - 25 % of muscle mass - INFERIOR WALL - approx. 1/2 of POSTERIOR WALL AV NODE

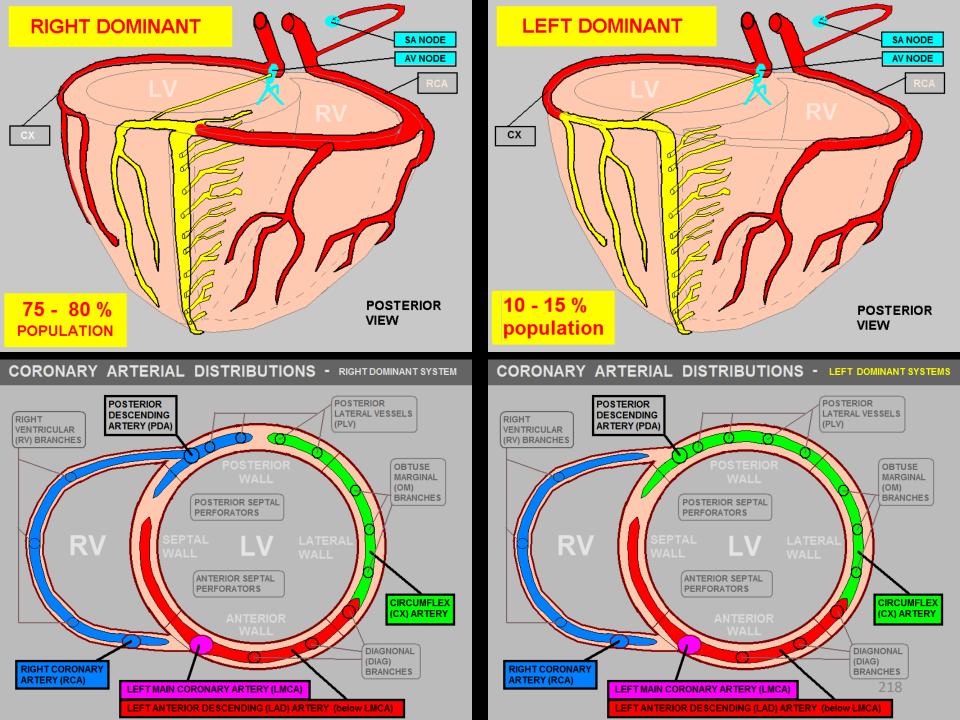
DOMINANT RIGHT CORONARY ARTERY - Most common arterial anatomy (75-80% of population) So if the Right Coronary Artery Is DOMINANT in 75 – 80% of the POPULATION, what accounts for the Other 20 – 25% ??





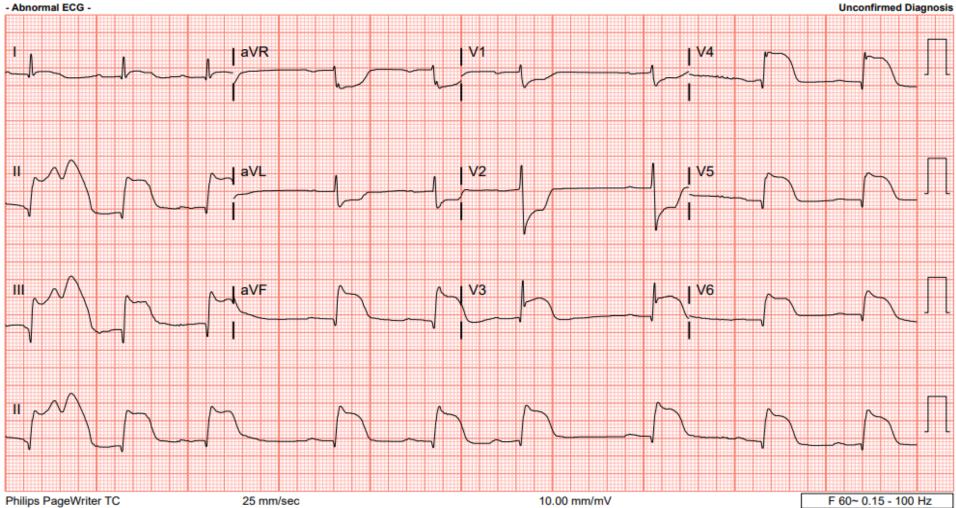


LEADS II, III, aVF



Pat ID RX DX		01/20/2021 07:46:46 08/17/1955 65 yrs	Account #	Bayfront Health Dept Room Tech	Seven Rivers ED ED EDWR mg
Rate PR QRSd QT QTc Av P QRS T	54 329 139 437 415 xis -83 80 77	<ul> <li>Atrial premature complex</li> <li>Prolonged PR interval</li> <li>Nonspecific intraventricular</li> <li>Inferoposterior infarct, acute</li> <li>Anterolateral infarct, acute</li> <li>Baseline wander in lead(s) V.</li> <li>NO PREVIOUS ECG AVAILABLE FOR</li> </ul>	conduction delay te (LCx) 3,V4	Req Provider:	Xandus Chen

#### - Abnormal ECG -

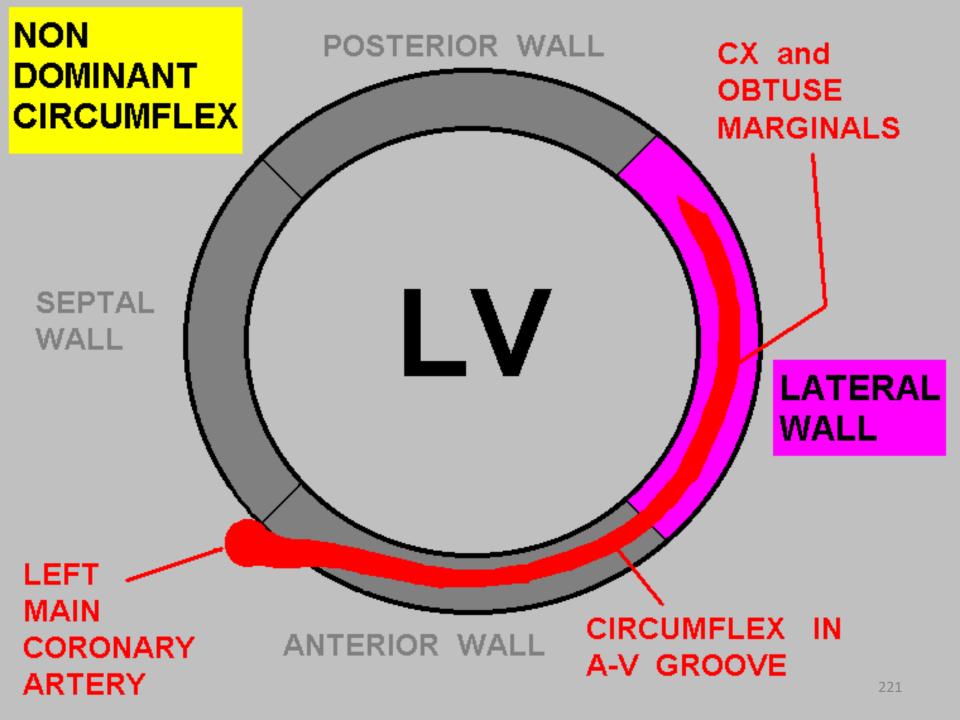


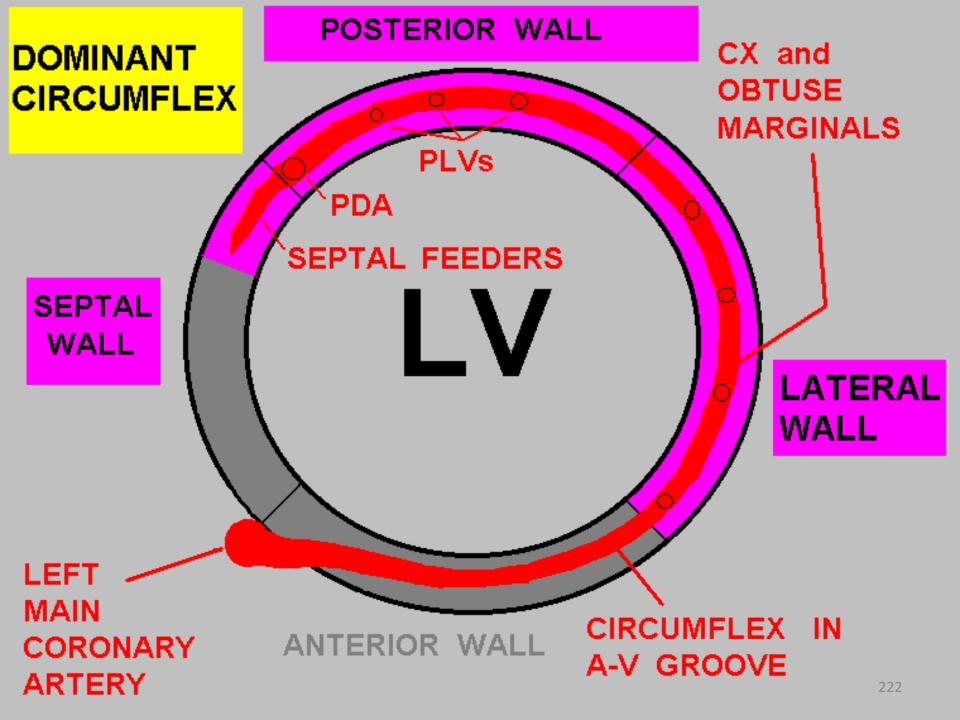
# CIRCUMFLEX ARTERY (CX)

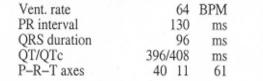
# - NON-DOMINANT CX: CX = 15-30% OF LV MASS

### - DOMINANT CX:

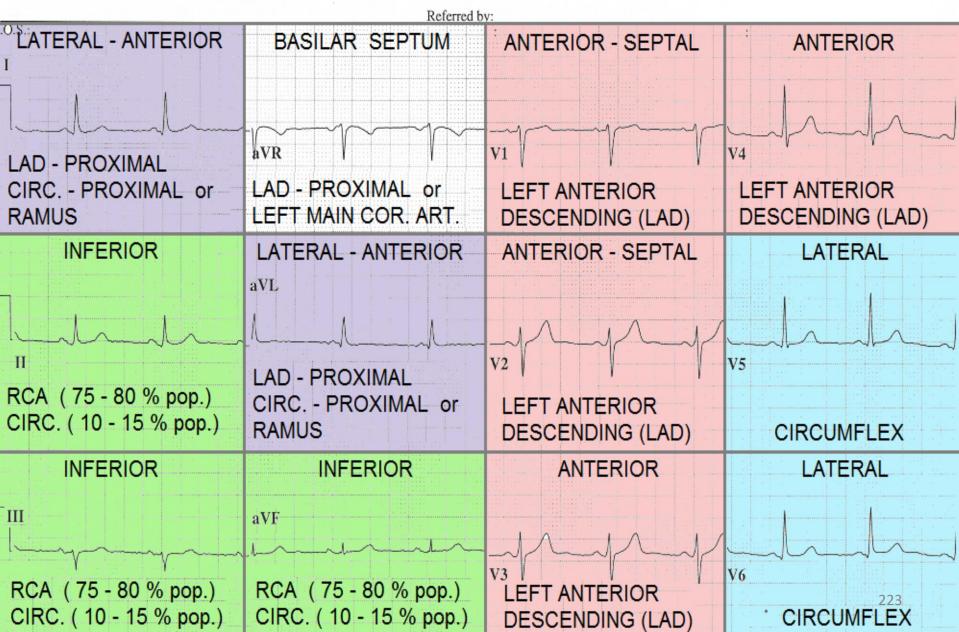
# CX = 15 - 30% OF LV MASS + PDA = 15 - 25% OF LV MASS TOTAL 30 - 55% OF LV MASS







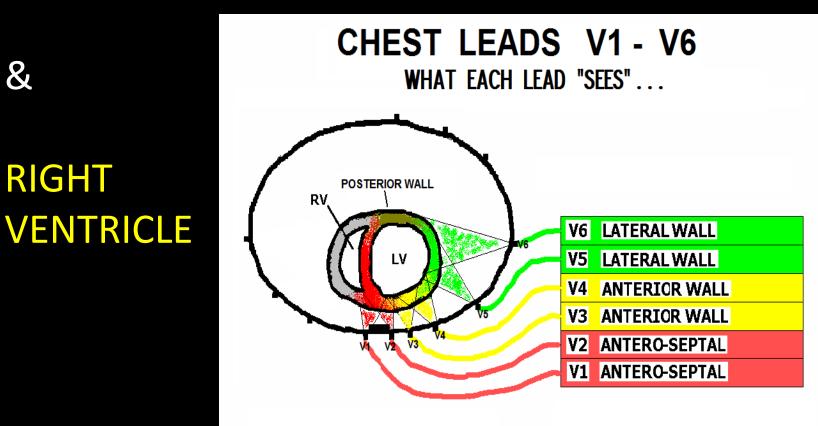
Normal sinus rhythm Normal ECG No previous ECGs available



# The 12 Lead ECG Has TWO major BLIND SPOTS . . . . **The POSTERIOR WALL**

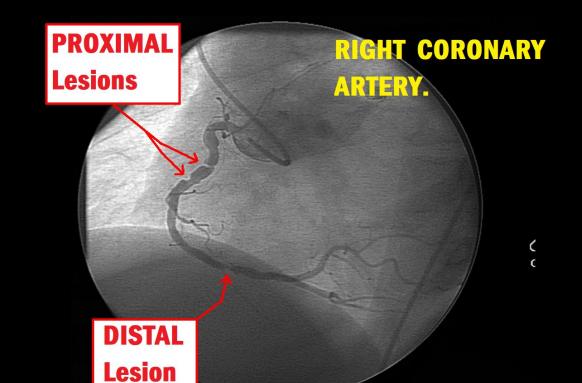
&

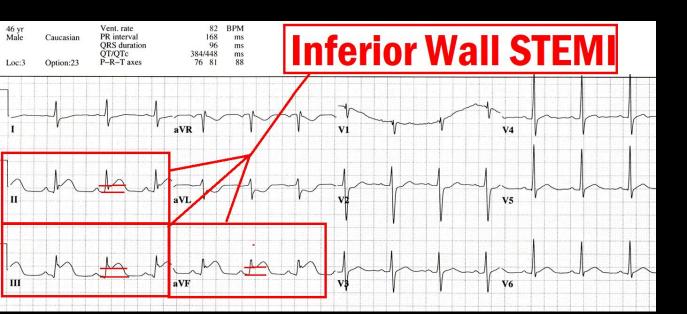
RIGHT



# 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.



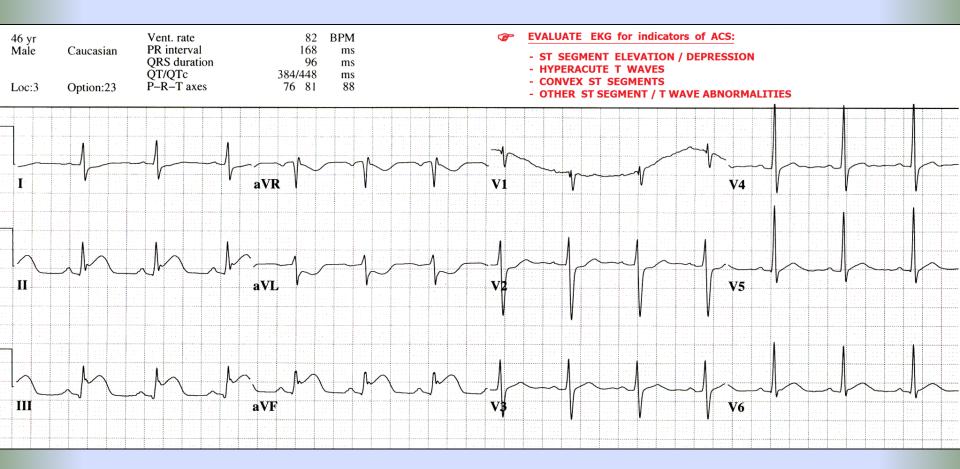


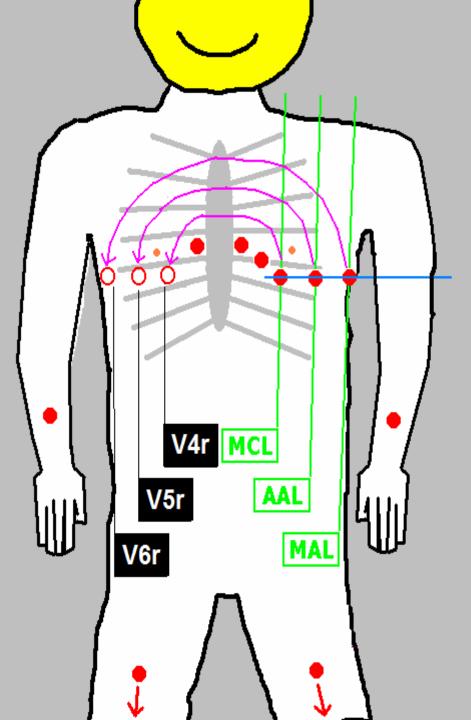
226

# To see the RIGHT VENTRICLE ...

# ... such as in cases of INFERIOR WALL M.I.

# Provide State of a State of a



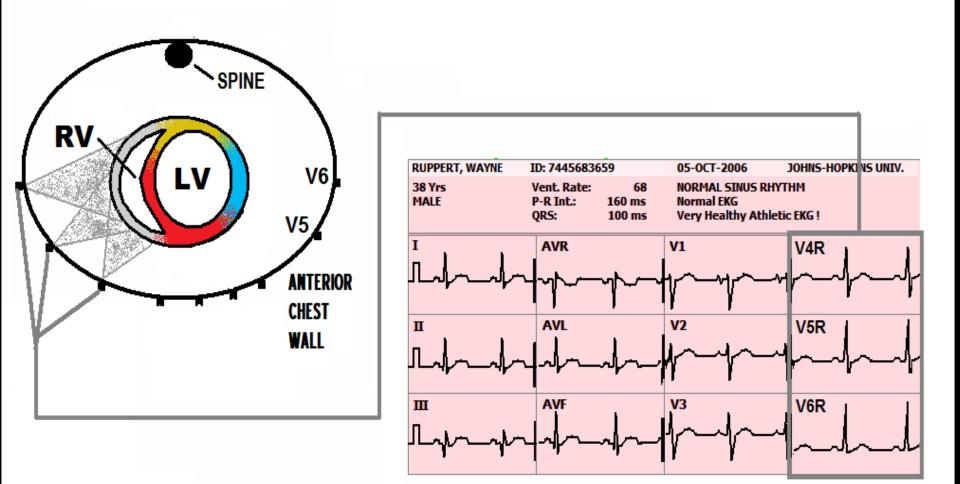


#### To do a RIGHT - SIDED EKG . .

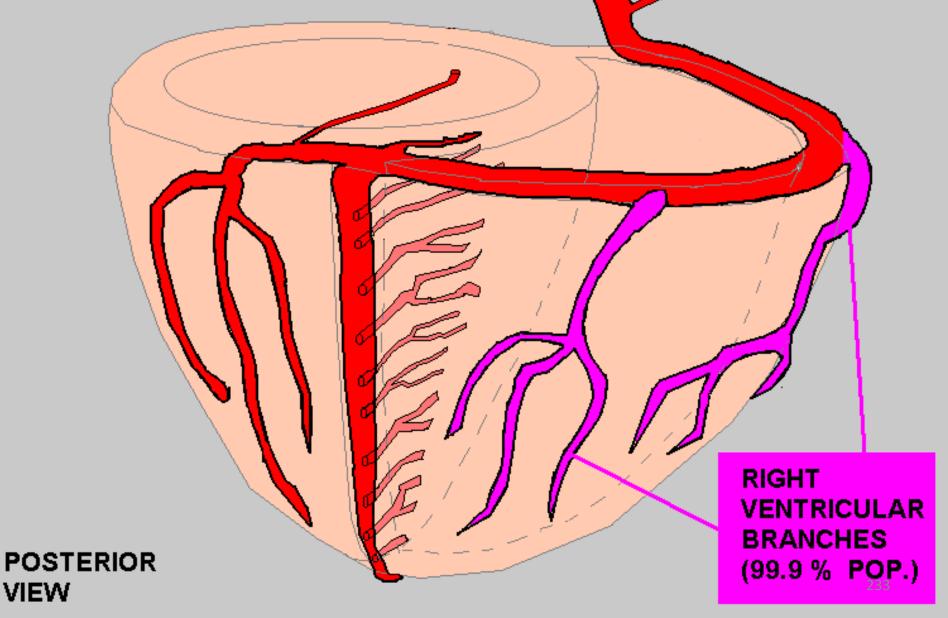
MOVE leads V4, V5, and V6

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

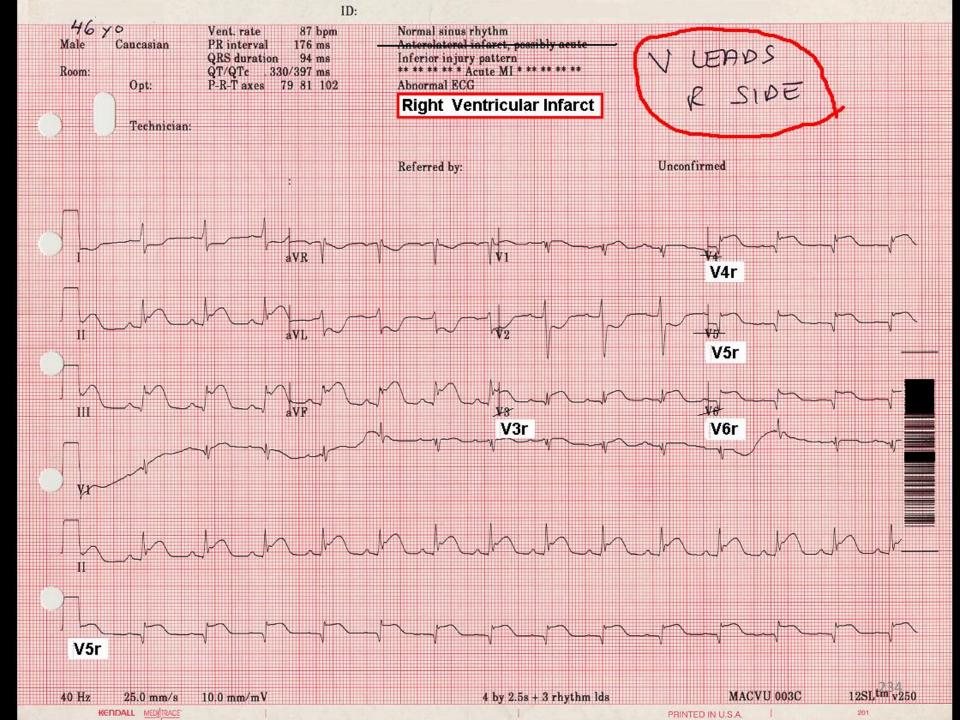
# V4R - V6R VIEW THE RIGHT VENTRICLE







SA NODAL



# 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.

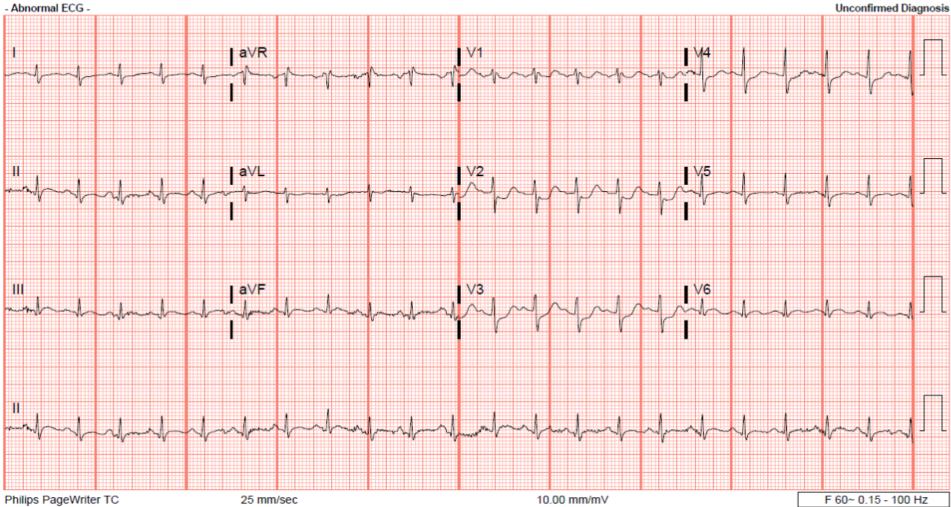
# Whenever you see **STDEPRESSION** in Leads V1 - V4

# you must do a **POSTERIOR LEAD ECG** (V7 - V9)

# to see if you Patient is having a POSTERIOR WALL STEM

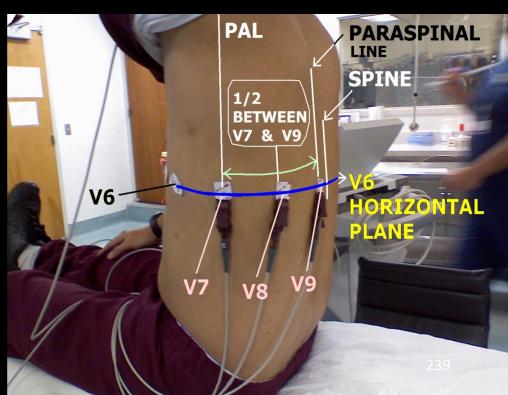
Pat ID RX			2019 22:07:54 46 yrs	Caucasian Female Account #	Dept Room	Seven Rivers ED ED
DX					Tech	LDC
Rate		131	Sinus tachycardia		Req Provider:	CHARLES NOLES
PR		128	Probable inferior infarct, old		-	
QRSd		92	Posterior infarct, acute (LCx)			
QT		317	ST depression V1-V3, suggest record	ding posterior leads		
QTc		468	NO PREVIOUS ECG AVAILABLE FOR COMP.	ARISON		
-	-Axis					
P		65				
QRS		83				
Т		132				

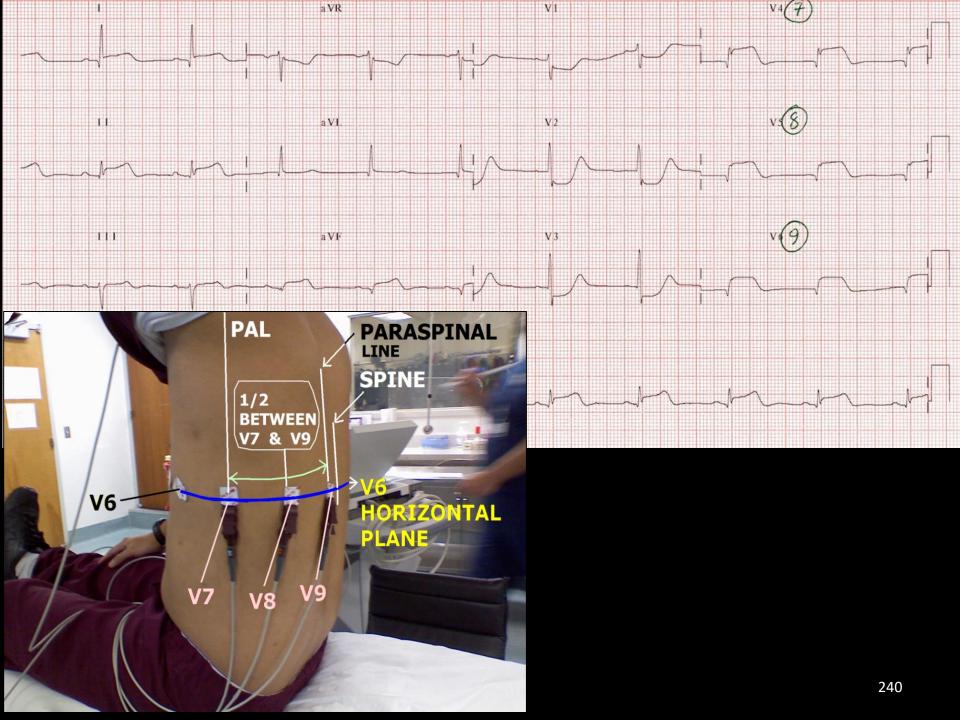
#### - Abnormal ECG -



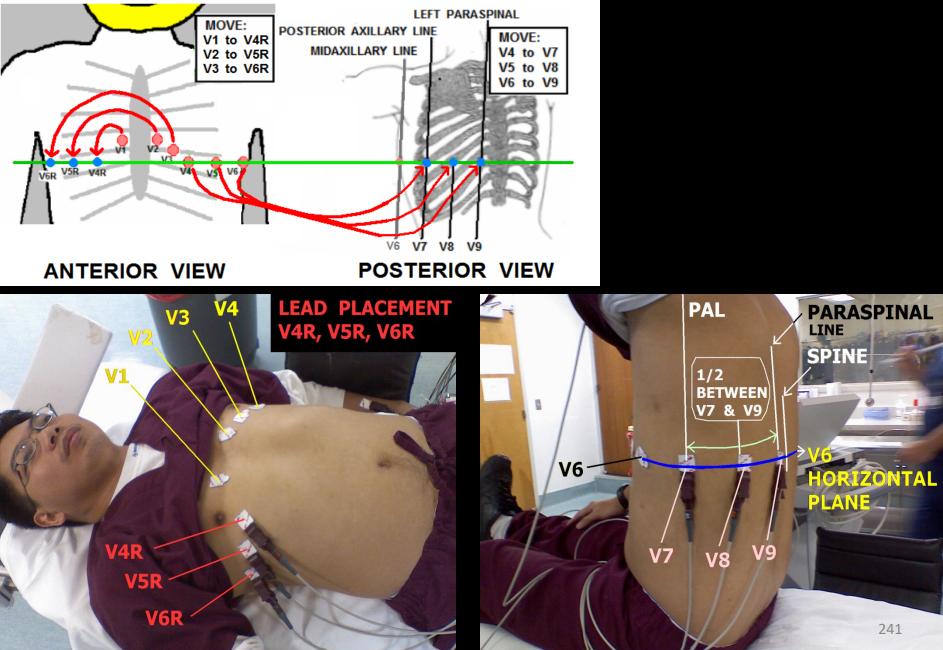
Whenever your patient's ECG exhibits ST DEPRESSION in any of the ANTERIOR LEADS (V1-V4), CONSIDER the possibility of POSTERIOR WALL STEMI !!

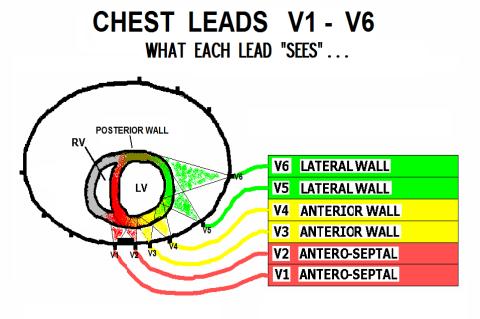
... To DIGANOSE Posterior Wall STEMI, we should see LEADS V7 – V9 !!





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

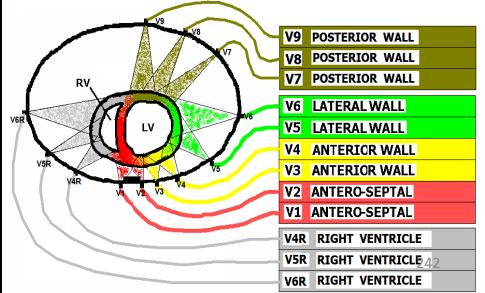


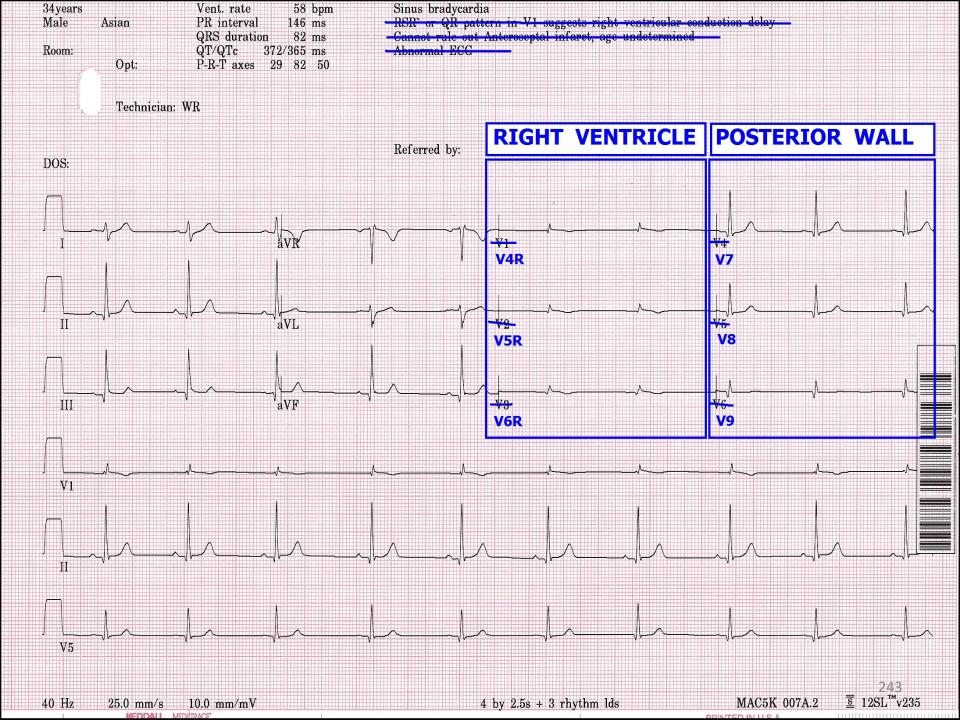


### ⇐ The 12 Lead ECG

### The 18 Lead ECG $\Rightarrow$





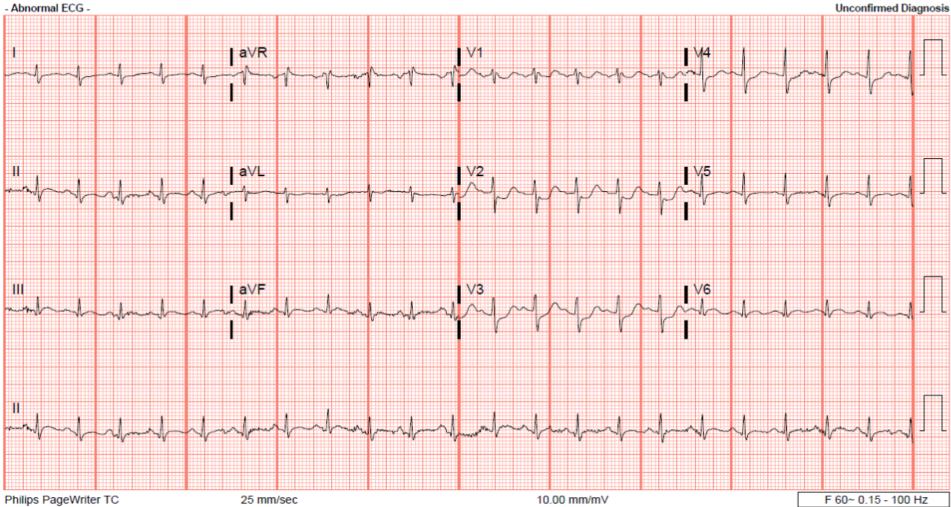


# POSTERIOR WALL MI usually accompanies INFERIOR and/or LATERAL WALL MI !!!

POSTERIOR WALL MI usually accompanies **INFERIOR** and/or LATERAL WALL MI !!! . . . On rare occasions, we see isolated cases of POSTERIOR WALL MI

Pat ID RX			2019 22:07:54 46 yrs	Caucasian Female Account #	Dept Room	Seven Rivers ED ED
DX					Tech	LDC
Rate		131	Sinus tachycardia		Req Provider:	CHARLES NOLES
PR		128	Probable inferior infarct, old		-	
QRSd		92	Posterior infarct, acute (LCx)			
QT		317	ST depression V1-V3, suggest record	ding posterior leads		
QTc		468	NO PREVIOUS ECG AVAILABLE FOR COMP.	ARISON		
-	-Axis					
P		65				
QRS		83				
Т		132				

#### - Abnormal ECG -





"ROAD TO FOREVER," Rt 385, Oklahoma panhandle, 1994

## Your patient has chest pain.....

### Your patient has chest pain.....

You obtain a STAT 12 Lead ECG......

# **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

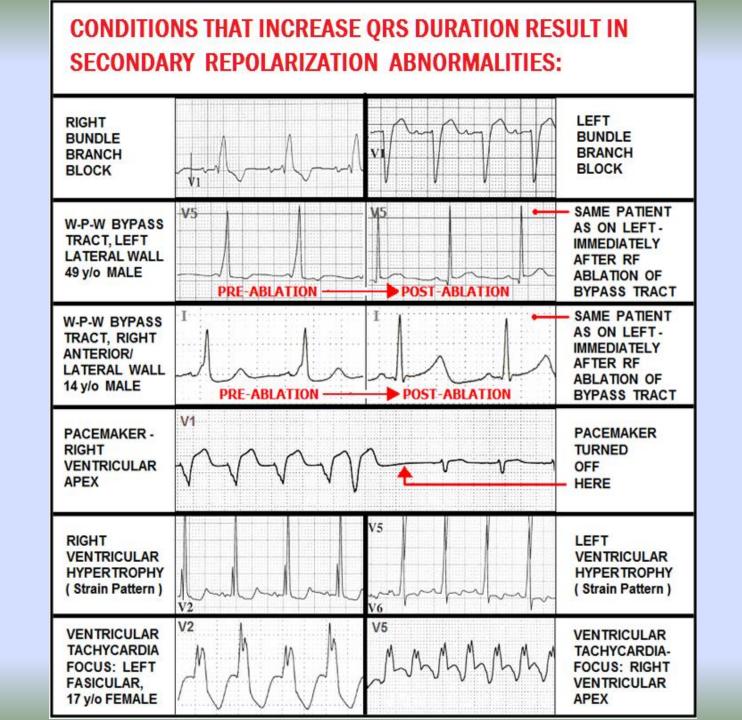
### STEP 1 – evaluate QRS width:

 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.



 Determine RIGHT vs. LEFT Bundle Branch Block Pattern

### Simple "Turn Signal Method" . . .

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

#### **USE LEAD V1 for this technique**

To make a **RIGHT TURN** 

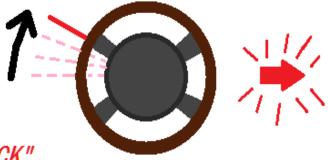
you push the turn signal lever **UP**....

THINK:

**V1** 

**V1** 

"QRS points UP = RIGHT BUNDLE BRANCH BLOCK"



To make a **LEFT TURN** you push the turn signal lever **DOWN** ....

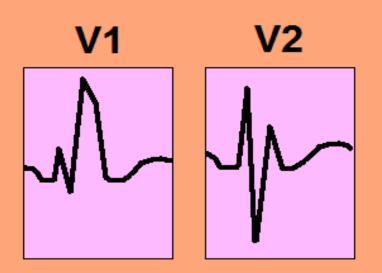
THINK:



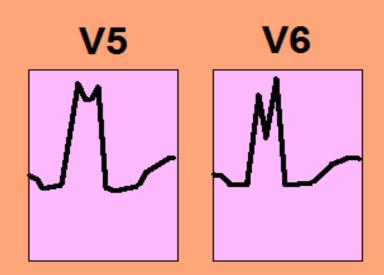
#### DIAGNOSING BUNDLE BRANCH BLOCK

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

LOCATING RsR' or RR' COMPLEXES:



#### RIGHT BUNDLE BRANCH BLOCK

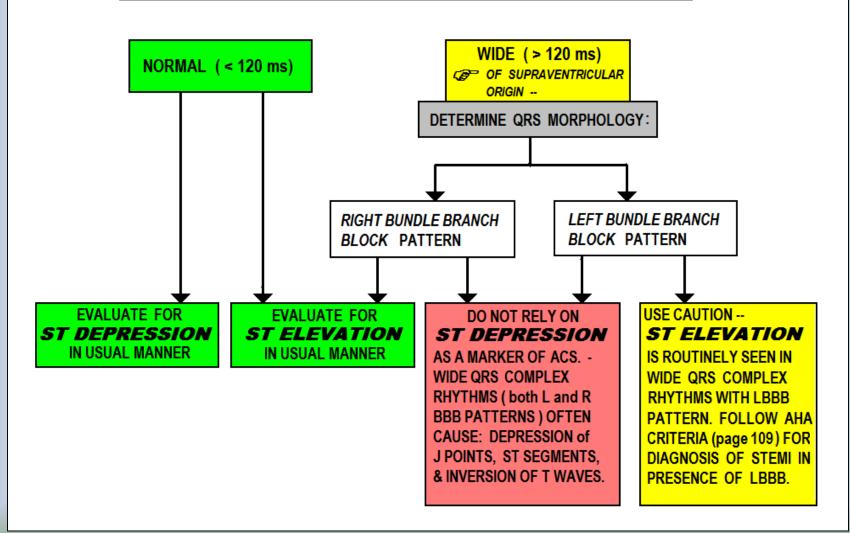


#### LEFT BUNDLE BRANCH BLOCK

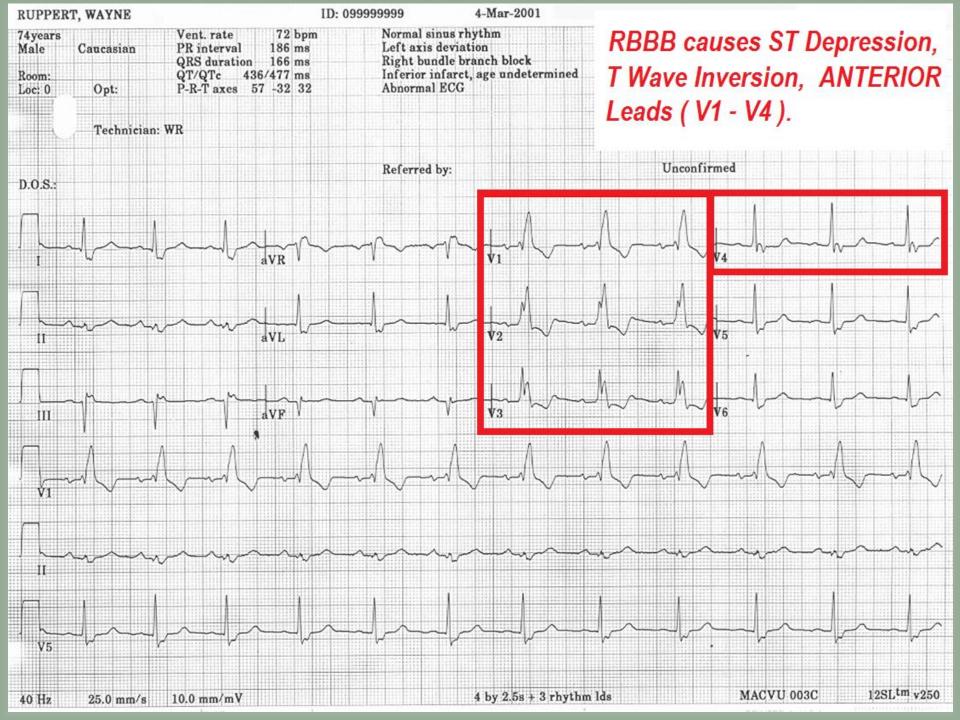
From: "Rapid Interpretation of ECGs" by Dale Dubin, MD

### Evaluating the ECG for ACS:



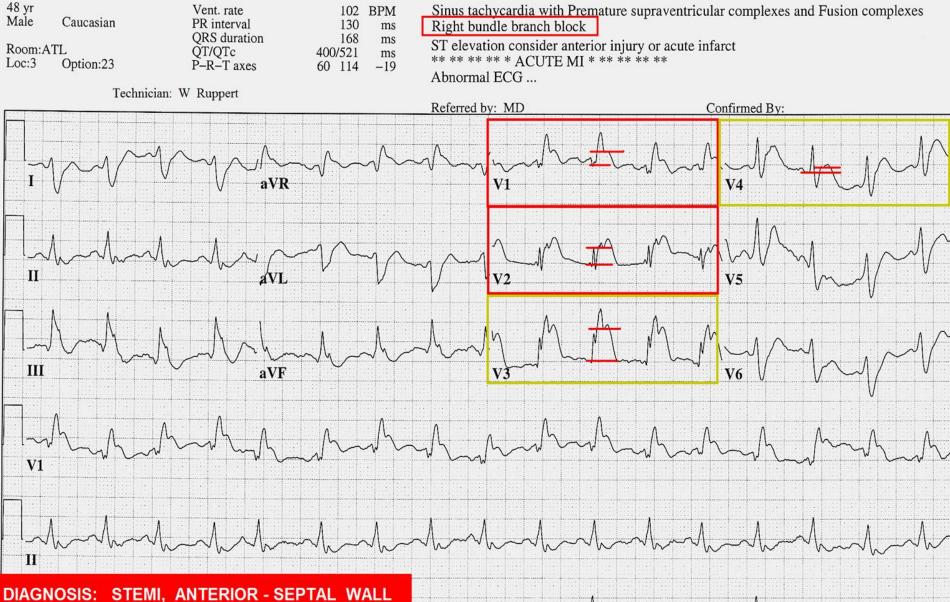


- When RIGHT Bundle Branch Block pattern is present:
  - Precordial Leads typically demonstrate ST
     Depression and T wave Inversion

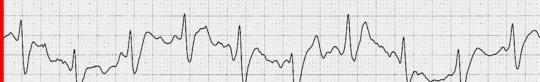


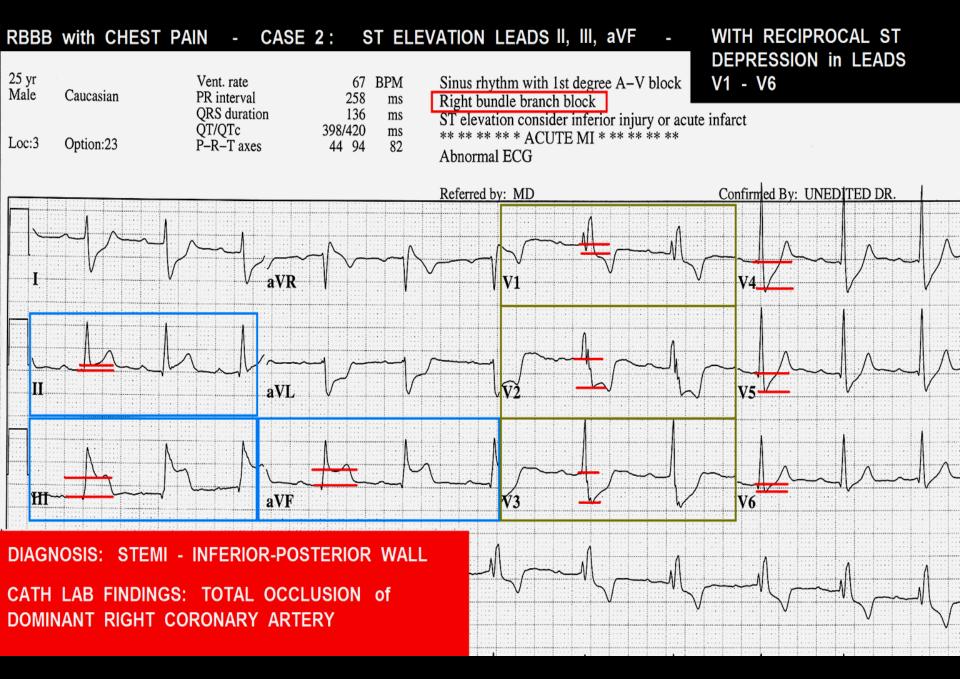
- When RIGHT Bundle Branch Block pattern is present:
  - Precordial Leads typically demonstrate ST
     Depression and T wave Inversion
  - DOES NOT MASK STEMI; when ST Elevation is noted, CONSIDER STEMI ! !

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

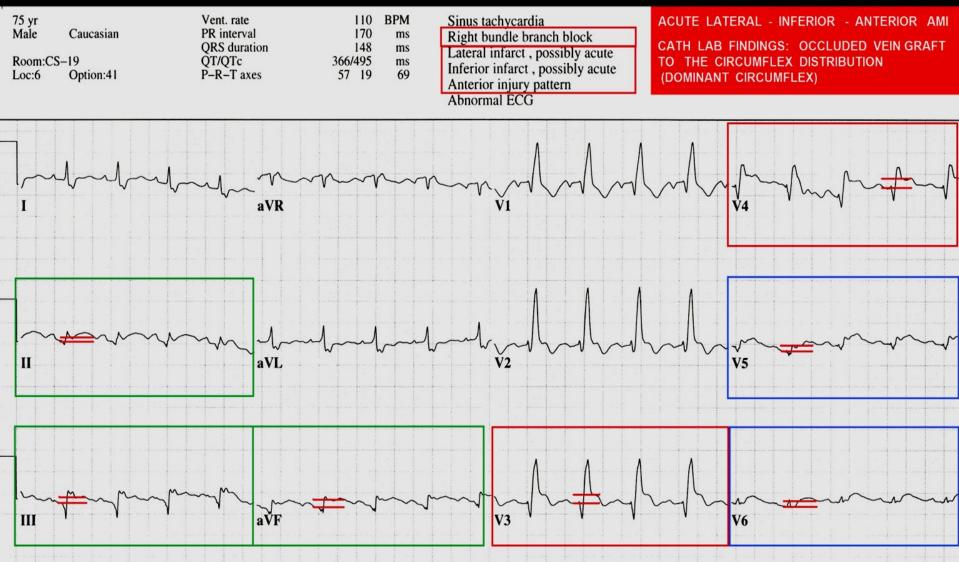


CATH LAB FINDINGS: TOTAL OCCLUSION of mid - LEFT ANTERIOR DESCENDING ARTERY.





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



When LBBB QRS pattern is present:

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

- 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)

- 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).

**2013 ACC/AHA Guideline for Management of STEMI** 

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

**2013 ACC/AHA Guideline for Management of STEMI** 

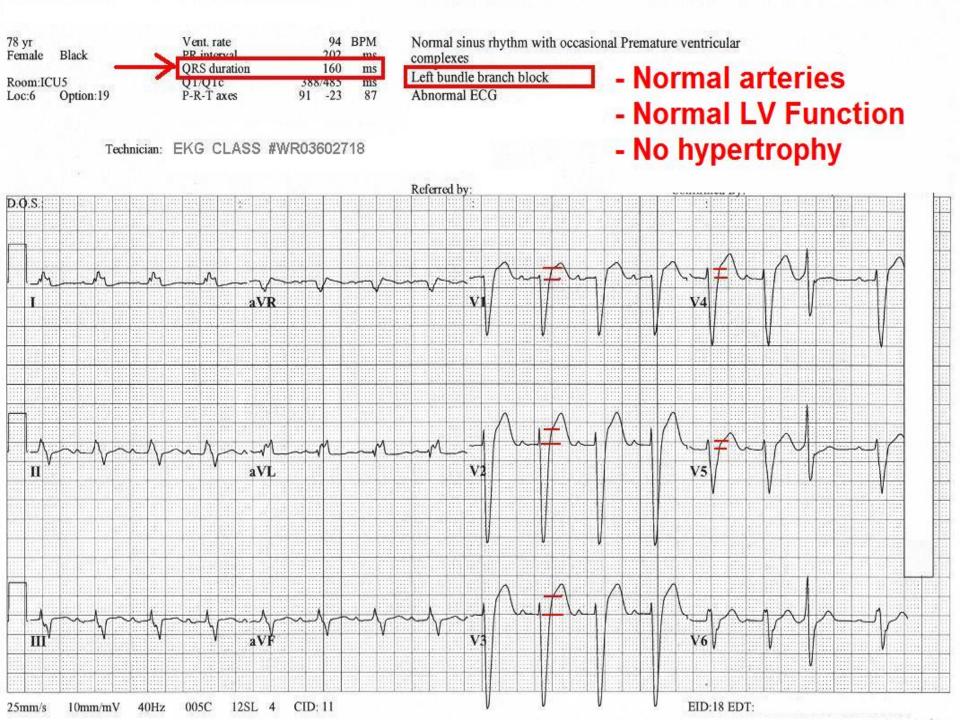
- ST Elevation of 0.1mv (1mm) or more in leads with Positive Deflection QRS complexes
- ST Elevation of 0.5mv (5mm) or more in leads with Negative Deflection QRS complexes

**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

**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





#### 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,

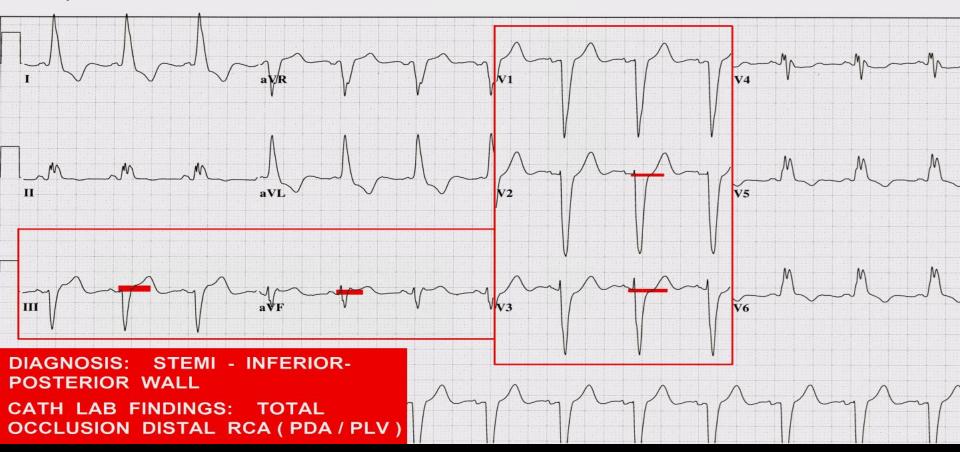
The J Point elevation can exceed 0.5 mv (5mm) above the iso-electric line in patients without ACS.

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

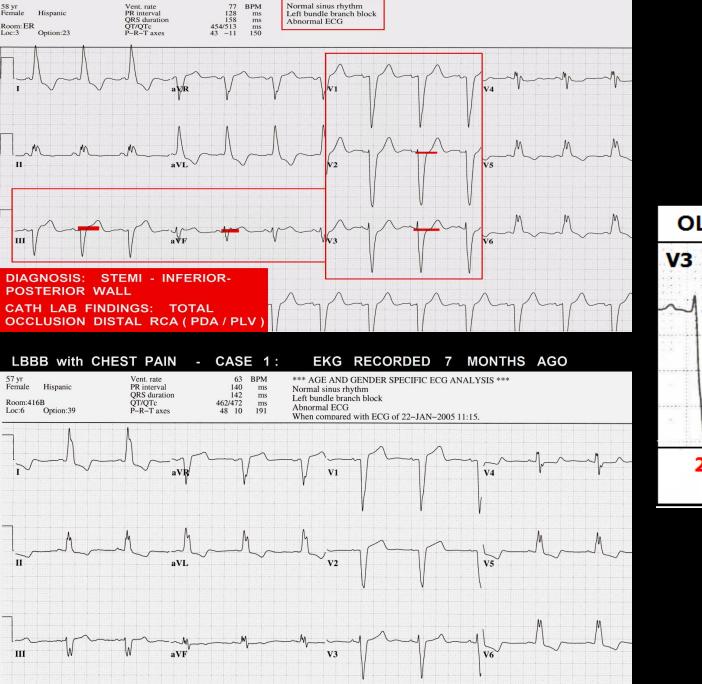


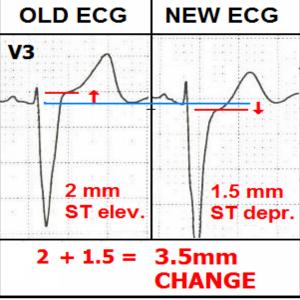
Vent. rate PR interval QRS duration QT/QTc P-R-T axes

77 BPM 128 ms 158 ms 454/513 ms 43 -11 150 Normal sinus rhythm Left bundle branch block Abnormal ECG



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

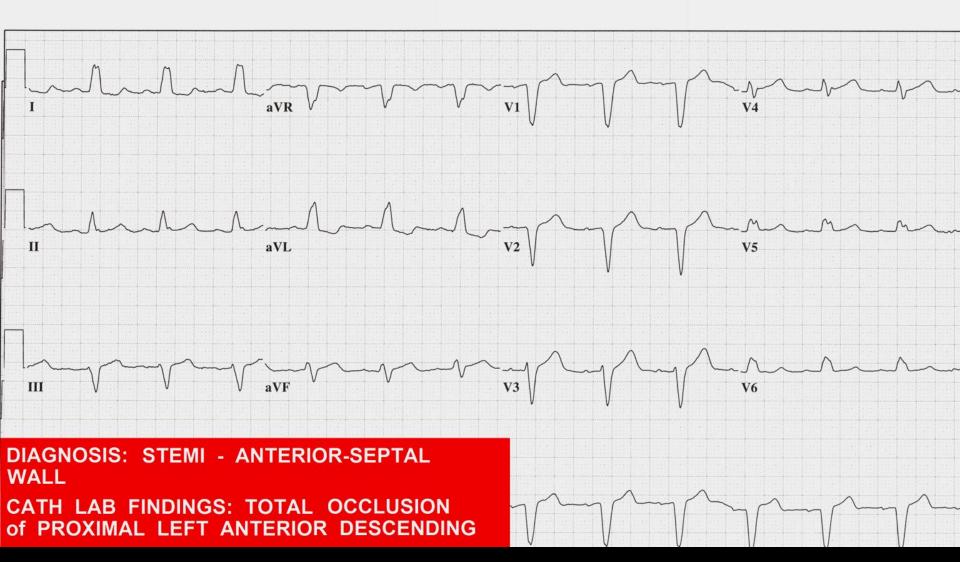




#### LBBB with CHEST PAIN - CASE 2: NEW ONSET of LBBB

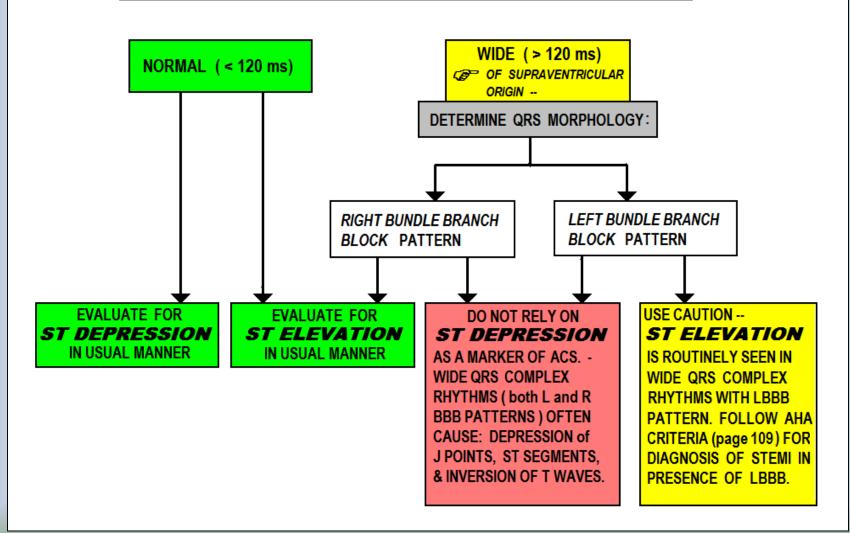
46 yr		Vent. rate	77	BPM
Male	Caucasian	PR interval	172	ms
		QRS duration	142	ms
Room:ER		QT/QTc	446/504	ms
Loc:3	Option:23	P-R-T axes	38 0	92

Normal sinus rhythm Left bundle branch block Abnormal ECG



### Evaluating the ECG for ACS:





Evaluating the ECG for ACS: Patients with Normal Width QRS (QRSd < 120ms)

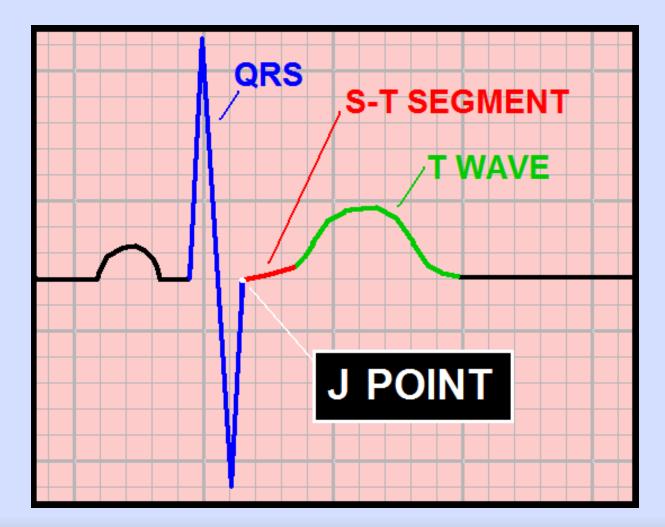
#### **STEP 2 - EVALUATE the EKG for ACS**

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:**



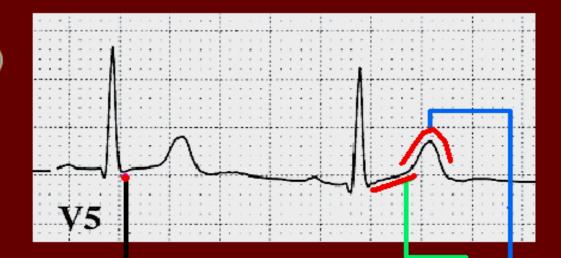
#### When QRS duration is NORMAL ( < 120 ms):

## NORMAL ST - T WAVES

in EVERY LEAD EXCEPT aVR !!

- WHEN QRS WIDTH IS NORMAL (< 120 ms)

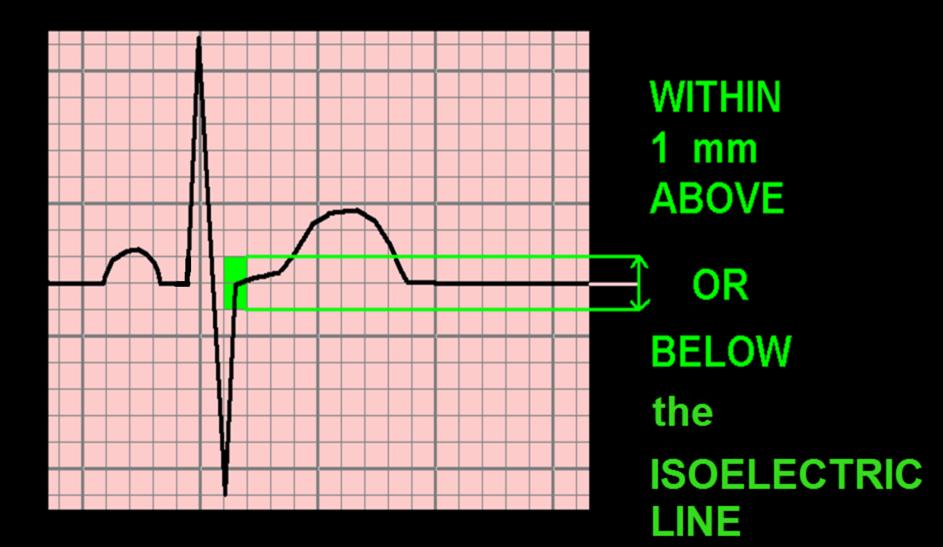
#### ASSESS:



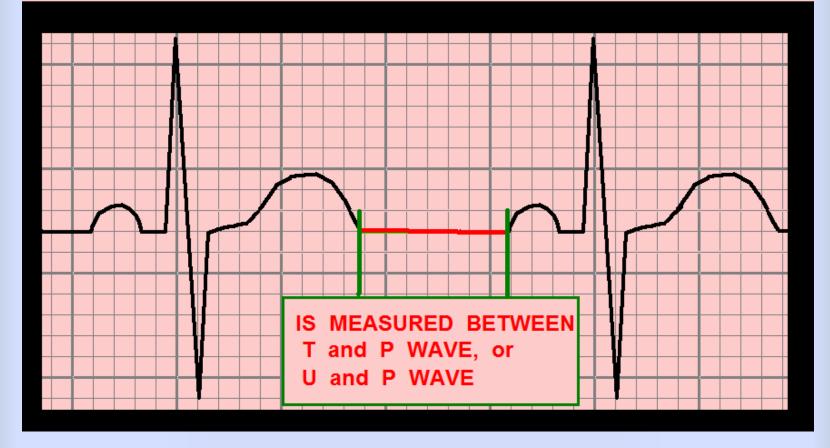
- J POINT: ISOELECTRIC (or < 1 mm dev.)
- ST SEG: SLIGHT, POSITIVE INCLINATION -

- T WAVE: UPRIGHT, POSITIVE -

### THE J POINT SHOULD BE ..



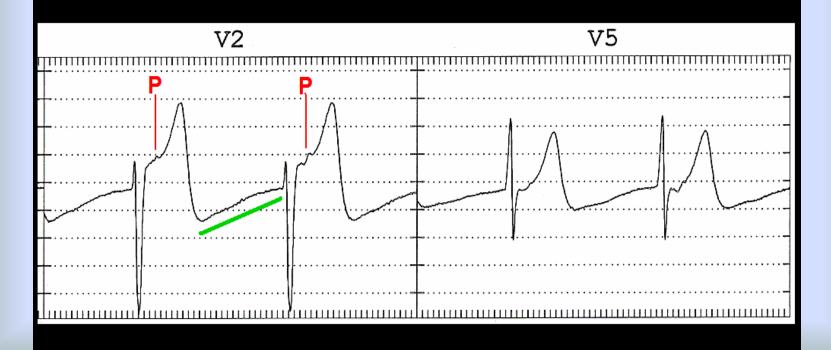
#### THE ISOELECTRIC LINE



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

## The Isoelectric Line it's not always isoelectric ! THE ISOELECTRIC LINE

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



## THE P-Q JUNCTION

. . . is the POINT where the P-R SEGMENT ends and the QRS COMPLEX BEGINS. **Used for POINT** OF REFERENCE for measurement of the J-POINT and the S-T SEGMENT -

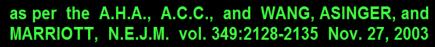


- as per the A.H.A., A.C.C., and WANG, ASINGER, and MARRIOTT, N.E.J.M. vol. 349:2128-2135 Nov. 27, 2003

## Use the P-Q junction as a reference point for measuring the J Point and ST-Segment when "iso-electric line is

### THE P-Q JUNCTION

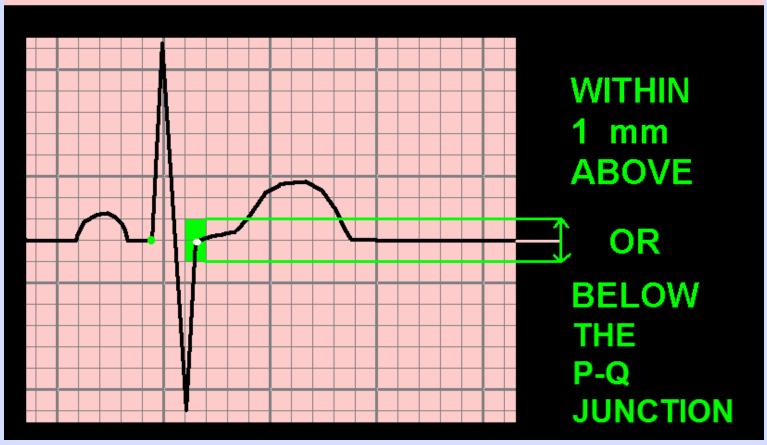
... is the POINT where the P-R SEGMENT ends and the QRS COMPLEX BEGINS. Used for POINT OF REFERENCE for measurement of the J-POINT and the S-T SEGMENT –



## not iso-electric !

## **Defining NORMAL:**

## THE J POINT SHOULD BE..



# 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

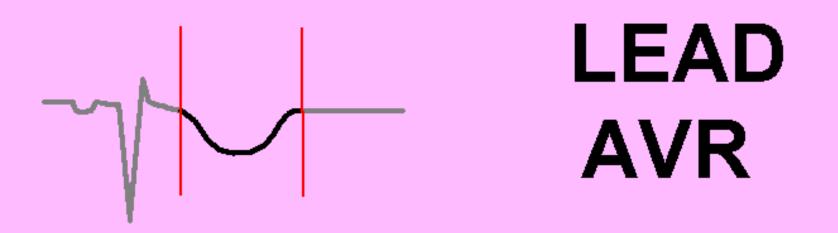
### SHOULD BE "CONCAVE" IN SHAPE ...



SHOULD BE SYMMETRICAL



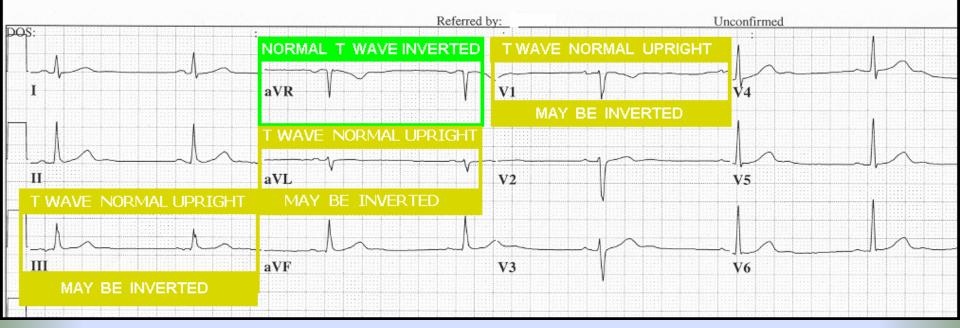
- SHOULD BE SYMMETRICAL
- SHOULD BE UPRIGHT IN ALL LEADS, EXCEPT AVR



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

## Normal Variants: *T Wave Inversion*

# Leads where the T WAVE may be INVERTED:





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

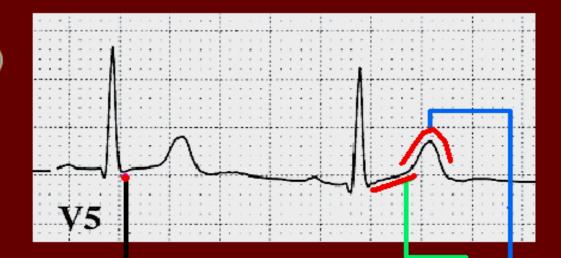
### When QRS duration is NORMAL ( < 120 ms):

# NORMAL ST - T WAVES

in EVERY LEAD EXCEPT aVR !!

- WHEN QRS WIDTH IS NORMAL (< 120 ms)

## ASSESS:



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

- T WAVE: UPRIGHT, POSITIVE -

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

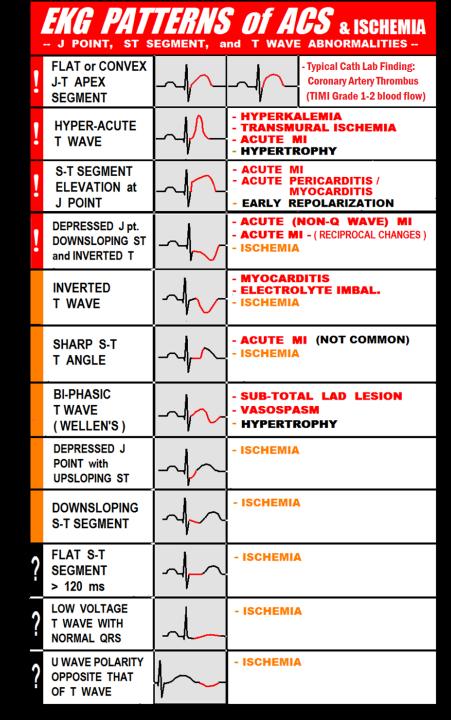
# Multiple patterns of ABNORMAL:

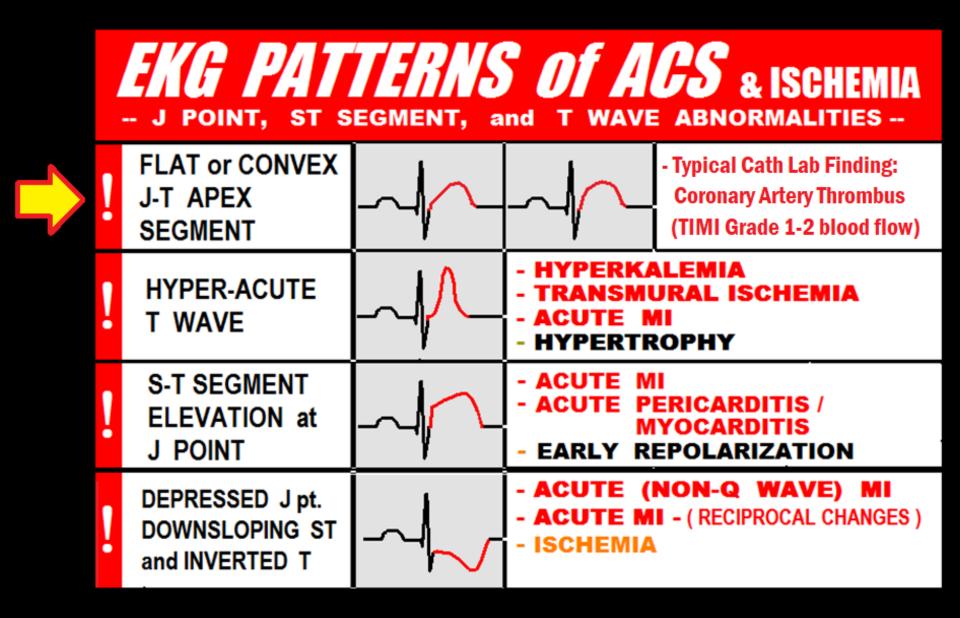
- J Point
- ST-Segment
- T Wave

# configurations may indicate ACS.

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

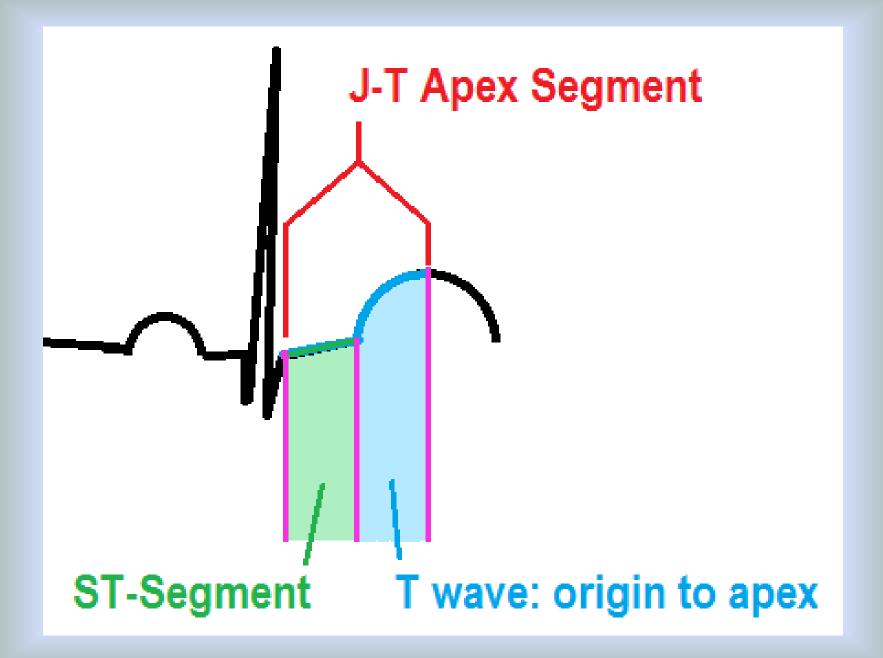
BOOK PAGE: 83

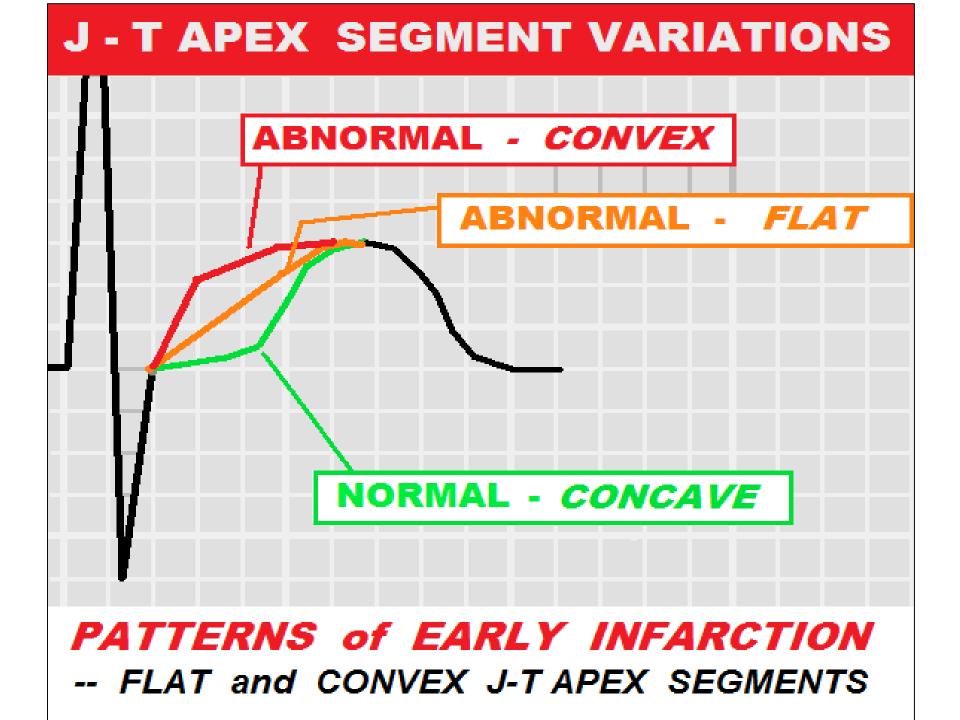




# **ECG Patterns associated with "EARLY PHASE MI:"**

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





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

From: AMERICAN HEART ASSOCIATION ACLS 2005 REVISIONS "J POINT plus 40 ms" shows ST ELEVATION > 1 mm INFARCTION -EARLY PHASE PATTERN J POINT NORMAL ST SEGMENT

During NORMAL STATES of PERFUSION, the J POINT is ISOELECTRIC and the ST SEGMENT has a

CONCAVE appearance. When measured 40 ms beyond the J POINT (noted by the RED DOT), the ST SEGMENT elevation is less than 1mm.

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

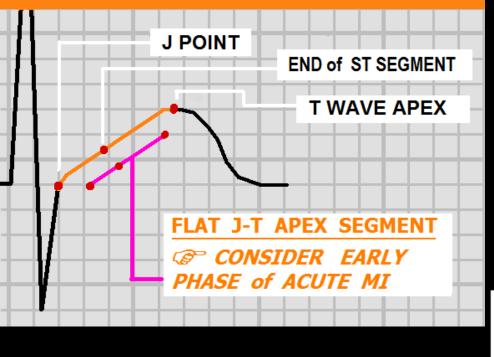
I fig A

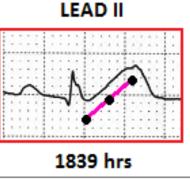
During a 20 second BALLOON OCCLUSION of the patient's LAD during routine PTCA, the ST segment

fig B

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.

#### **ABNORMAL J-T APEX SEGMENT**



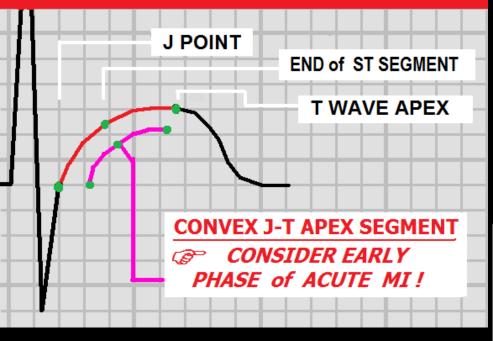


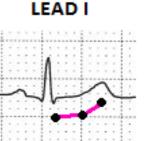
- 41 y/o FEMALE In ER C/O CHEST PAIN x 30 minutes. *FLAT J-T APEX SEGMENT*
- NO ST ELEVATION at J POINT !



- 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

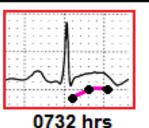
### **ABNORMAL J-T APEX SEGMENT**





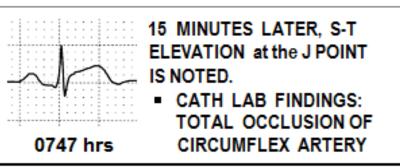
#### 53 y/o MALE

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



#### STEMI LATERAL WALL

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



### 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%

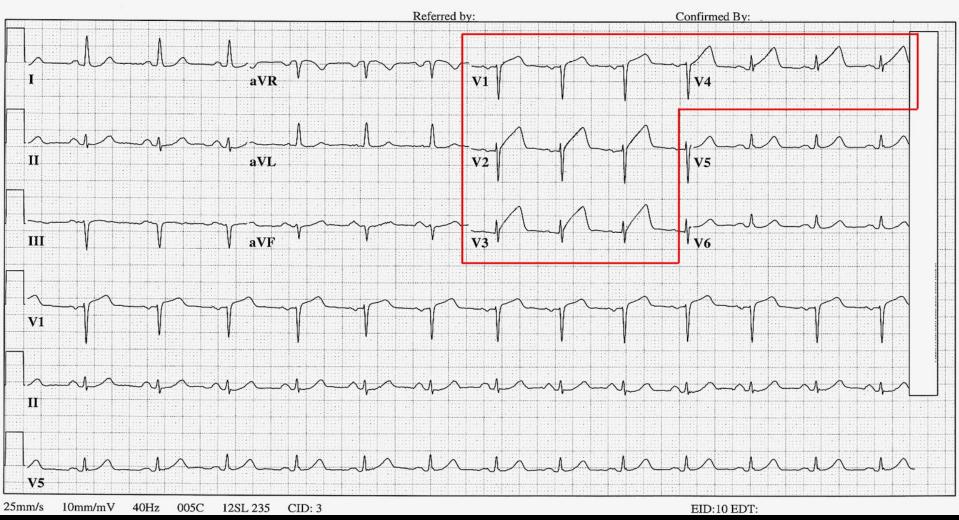
### LABS: JUST OBTAINED, RESULTS NOT AVAILABLE YET.

56 yr		Vent. rate	80	BPM
Male	Caucasian	PR interval	154	ms
		QRS duration	78	ms
Room:A9		QT/QTc	380/438	ms
Loc:3	Option:23	P-R-T axes	51 -24	38

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

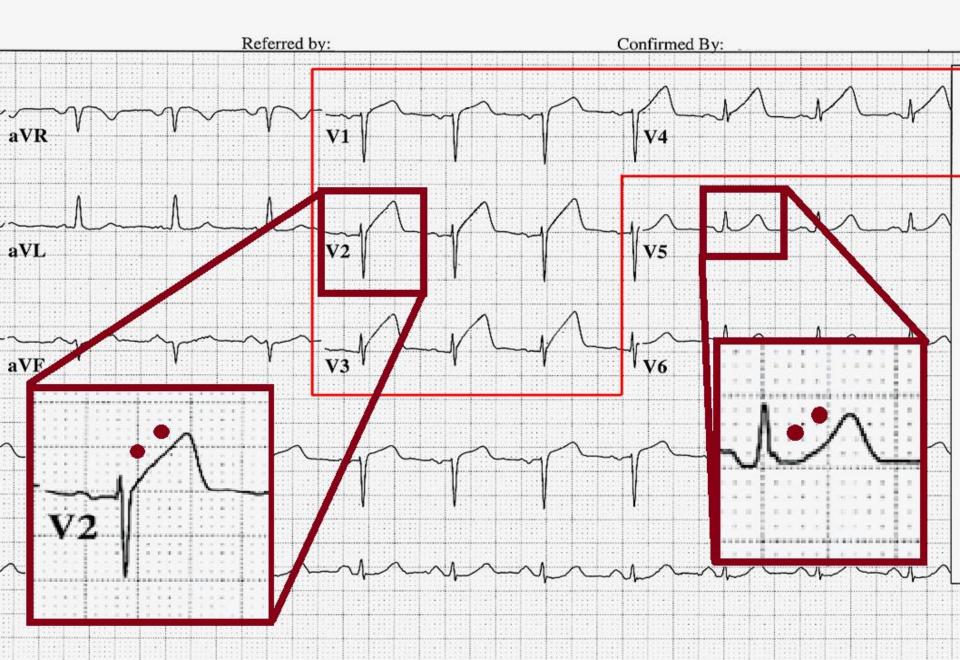
Normal sinus rhythm Normal ECG No previous ECGs available

Technician: W Ruppert

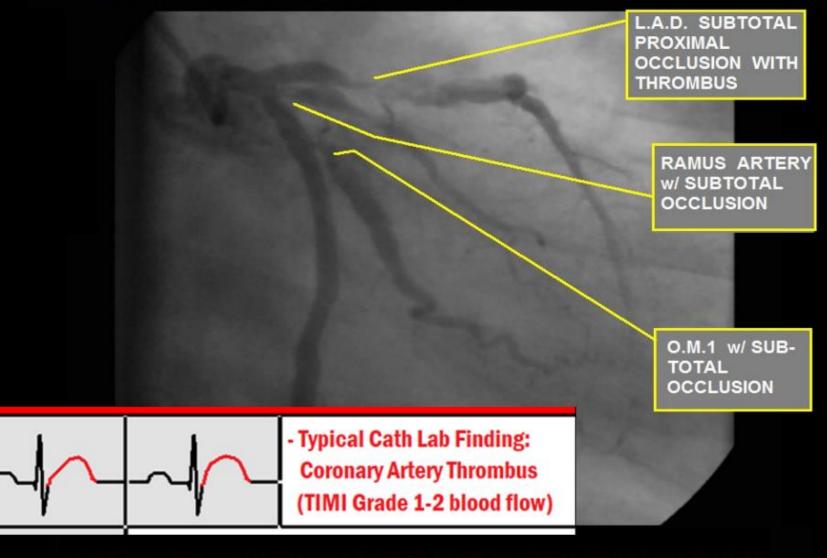


ECG COMPUTER DOES NOT NOTICE THE CONVEX J-T APEX SEGMENTS !

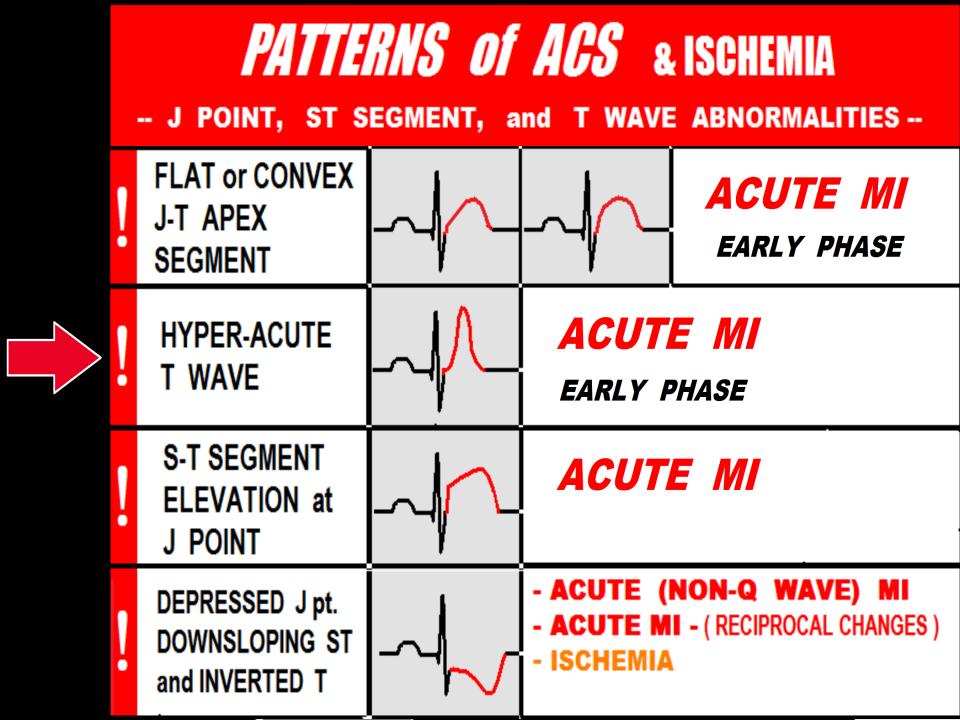
380/438 ms 51 -24 38	Normal ECG	
51 -24 58	No previous ECGs available	



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



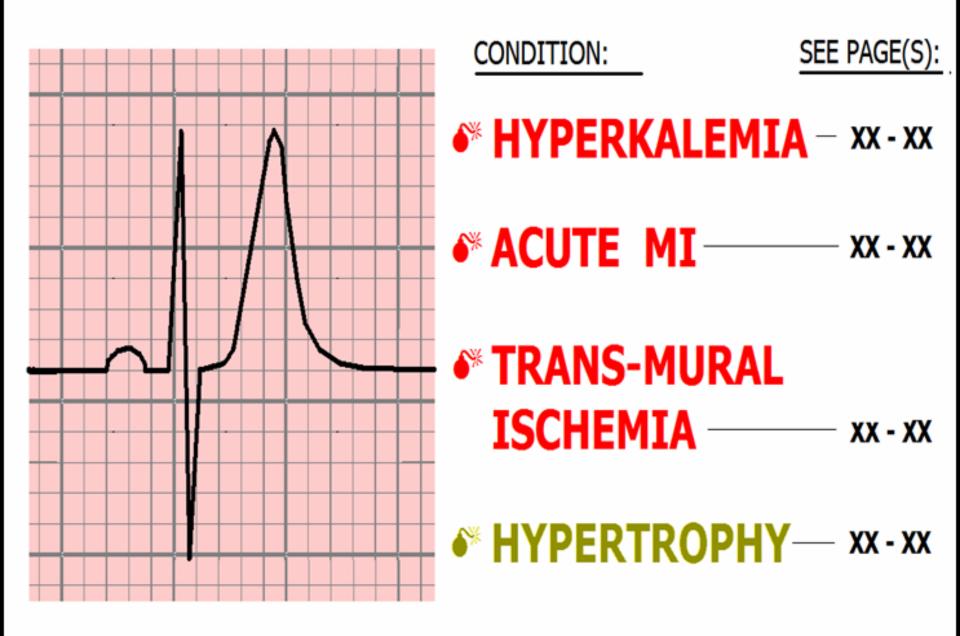
TREATMENT PLAN : EMERGENCY CORONARY ARTERY BYPASS SURGERY (4 VESSEL)



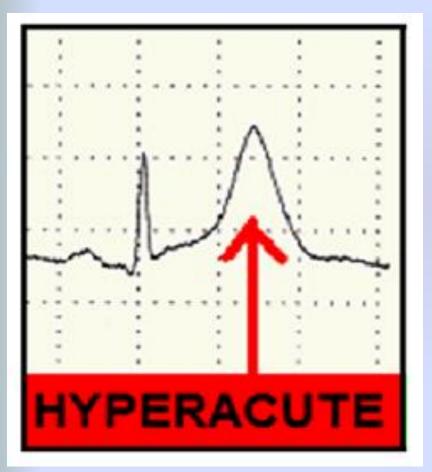


## **W** HYPER-ACUTE T WAVES - COMMON ETIOLOGIES:



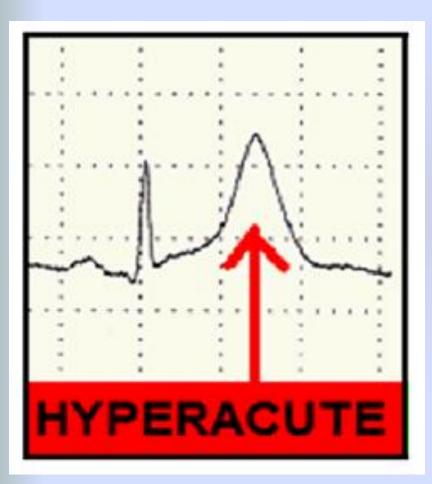


## T waves should not be HYPERACUTE



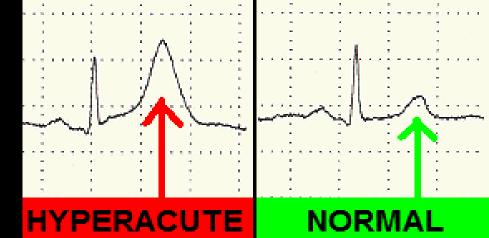


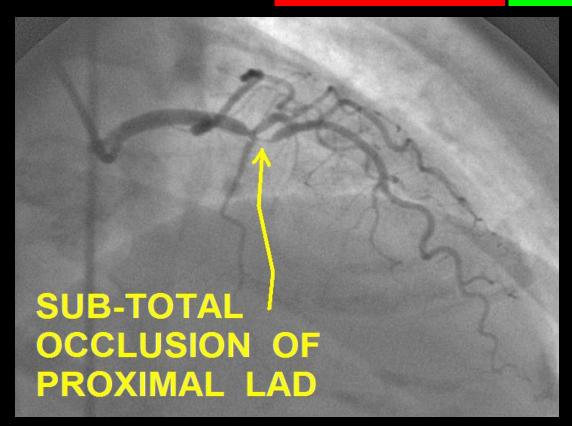
## HYPERACUTE T Waves may indicate:



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

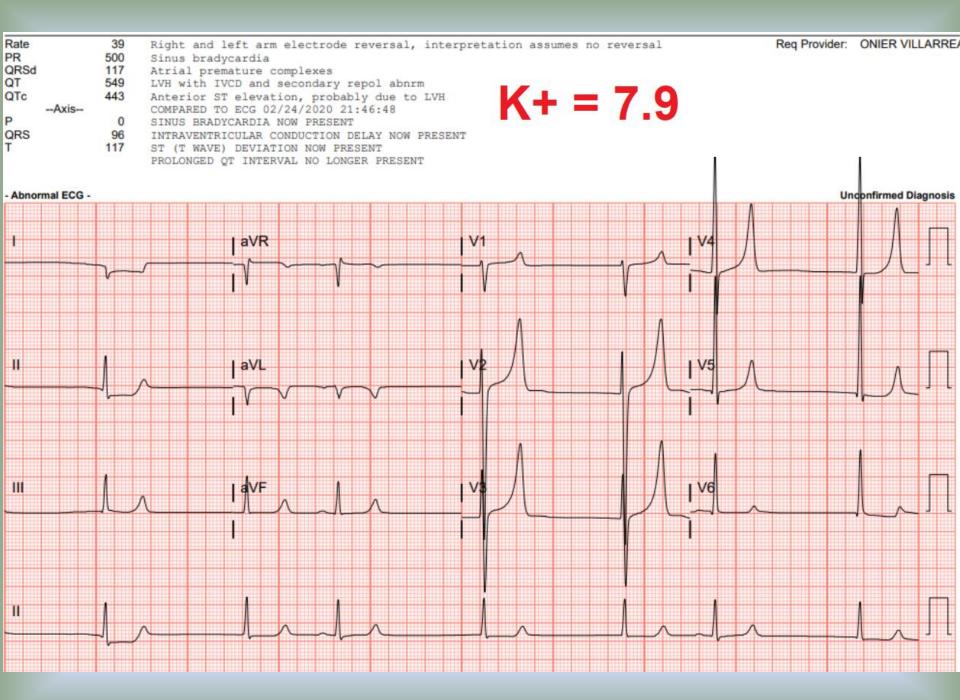
# HYPERACUTE T WAVES

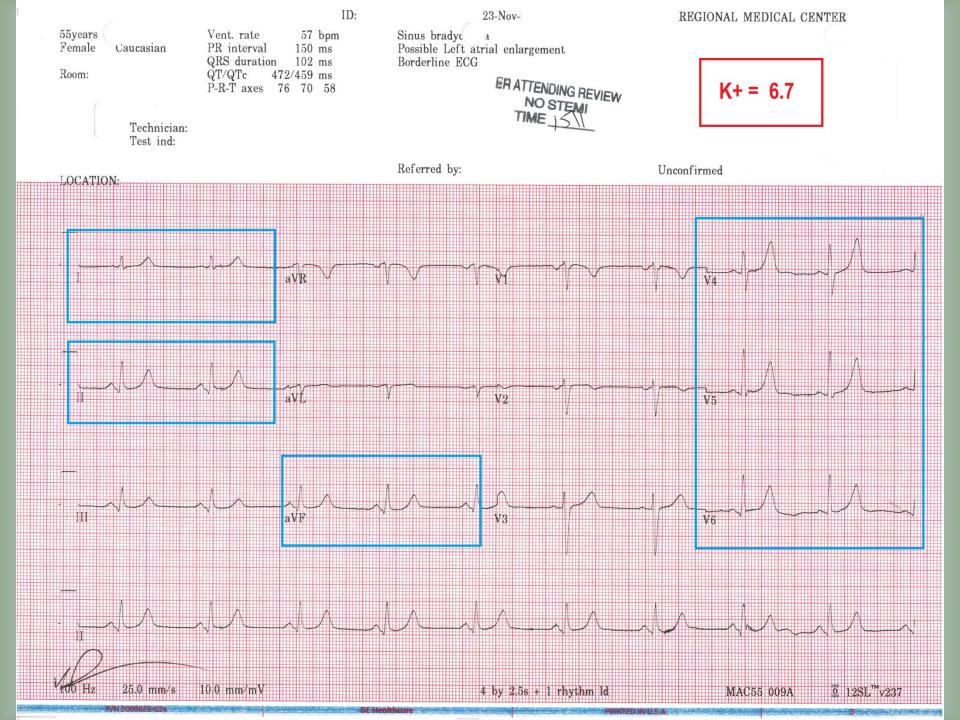




## Helpful Clue: Hyper-Acute T Waves

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





## 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

### CHIEF COMPLAINT and SIGNIFICANT HISTORY:

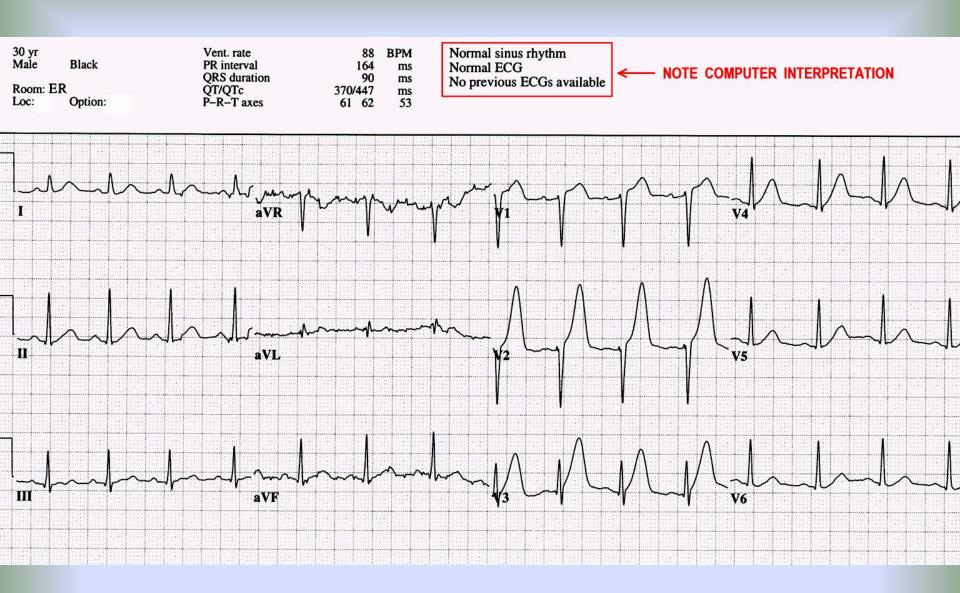
30 y/o male presents to ER via EMS, c/o sudden onset of dull chest pain x 40 min. Pain level varies, not effected by position, movement or deep inspiration. No associated symptoms.

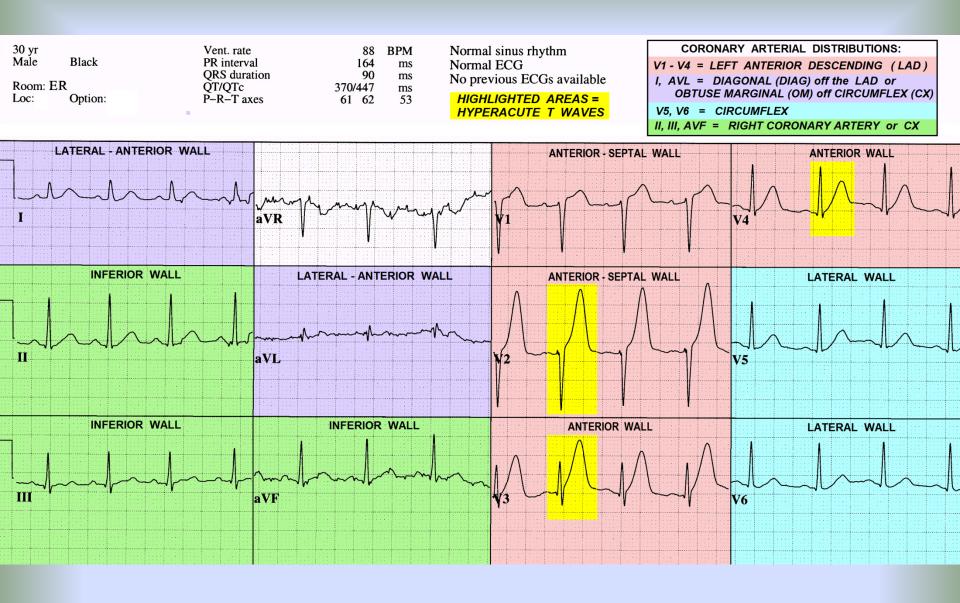
### RISK FACTOR PROFILE: NONE. CHOLESTEROL UNKNOWN.

**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

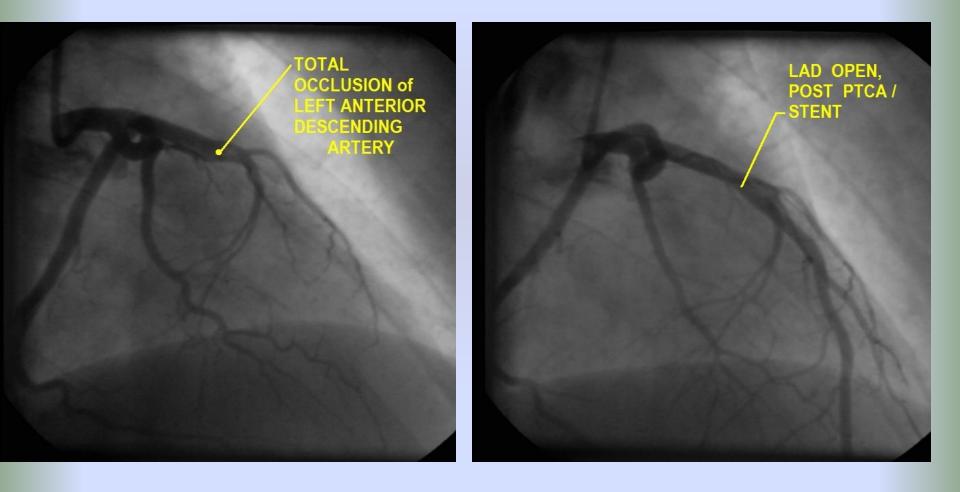
VITAL SIGNS: BP 136/88 P 90 R 20 SAO2 98%

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





## **Cath Lab findings:**



# **Dynamic ST-T Wave Changes:**

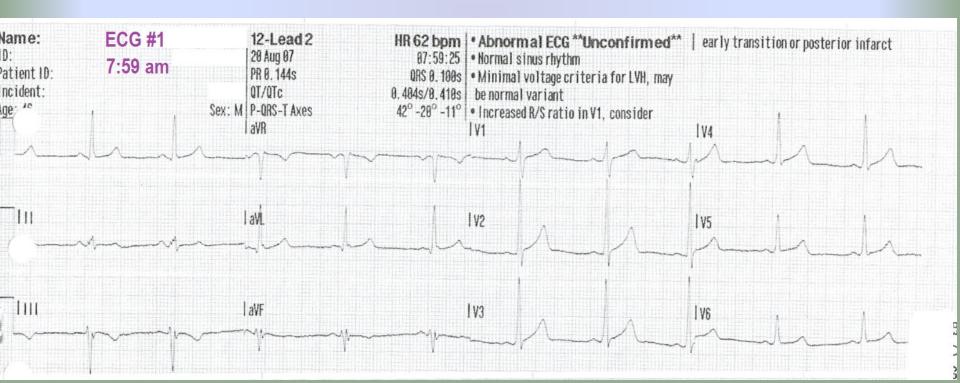
 Other than HEART RATE related variations (which affect intervals), *J Points, ST-Segments and T Waves SHOULD NOT CHANGE.*

# **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.

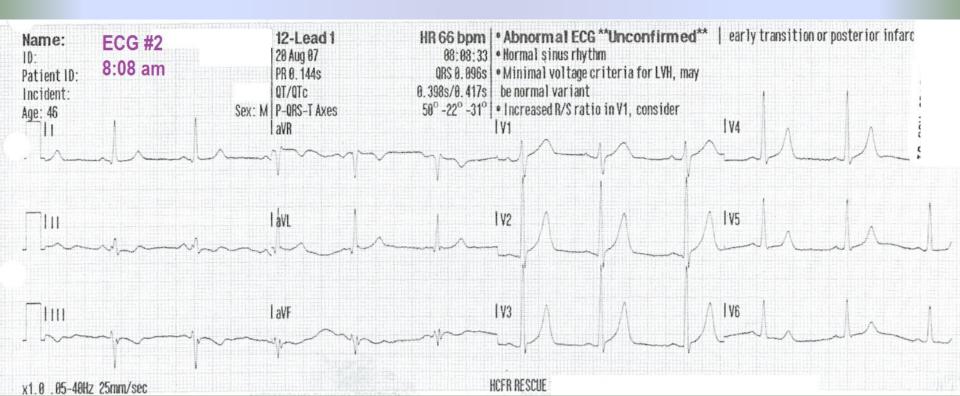
# 46 year old male

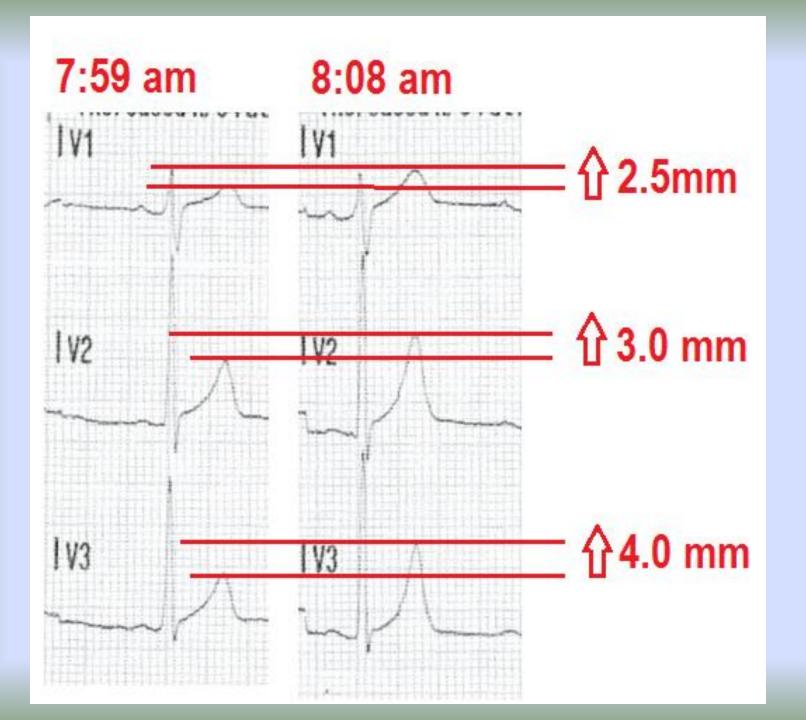
- Exertional dyspnea X "several weeks"
- Intermittent chest pressure X last 3 hours. Currently pain free.



# 46 year old male: ECG 1

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



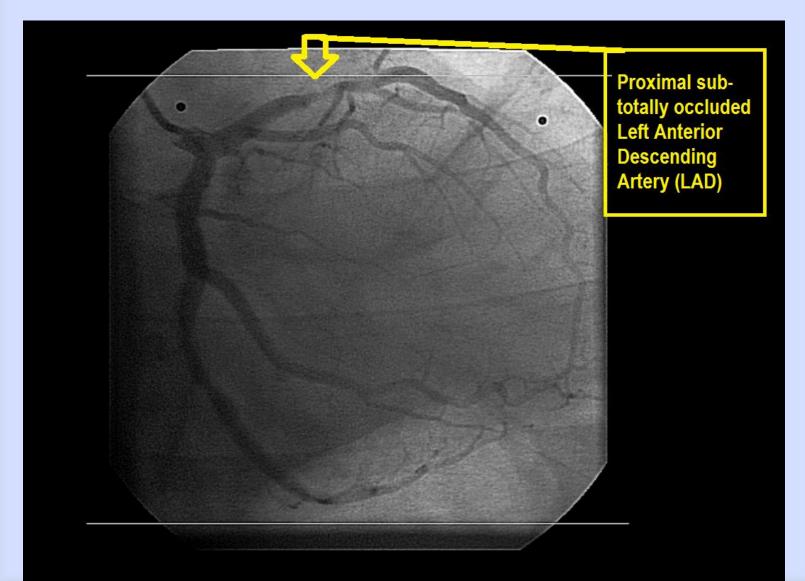


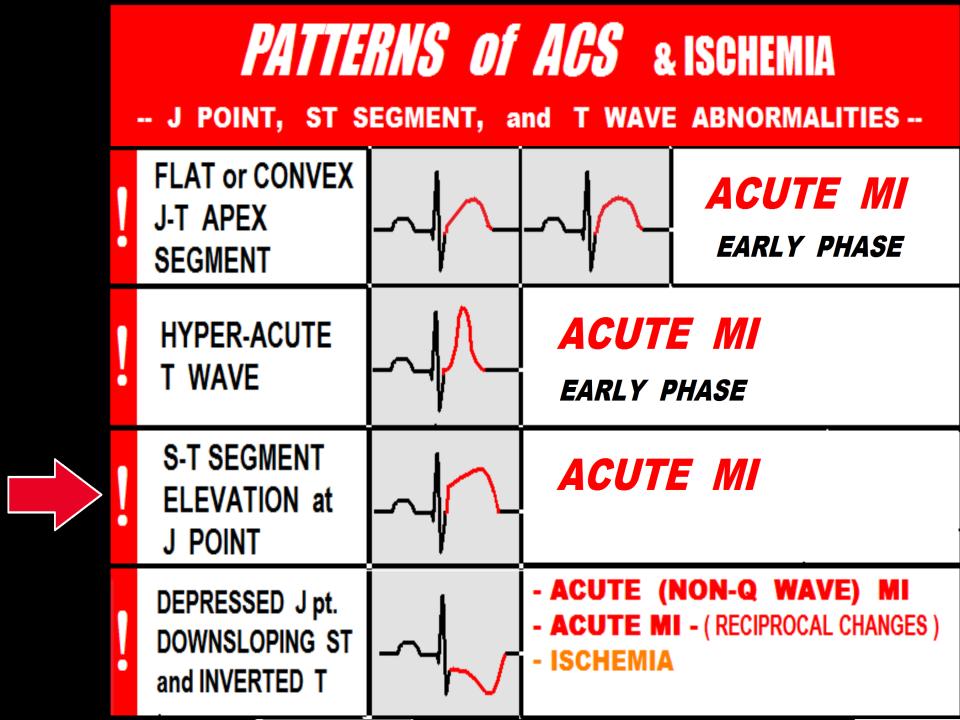
## **ST-Segment Depression**

### **7:59** am **8:08** am



# Cath Lab Angiography:





## **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,

1.0 mm or more, in TWO or more CONTIGUOUS LEADS

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

## **STEMI Criteria for 18 Lead ECGs:**

Right-Sided Chest Leads (V3R – V6R): <u>0.5</u> mm

Posterior Chest Leads (V7 – V9): <u>0.5</u> mm

\* P. Rautaharju et al, "<u>Standardization and Interpretation</u> 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

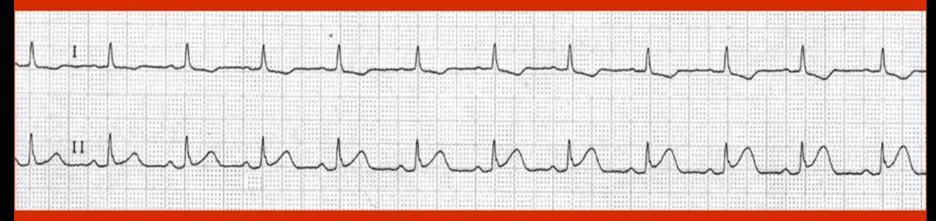
### Acute Ischemia and Infarction," Galen Wagner, et al

### 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<sub>2</sub> and V<sub>3</sub> 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<sub>2</sub> and V<sub>3</sub> and -0.1 mV (-1 mm) in all other leads.

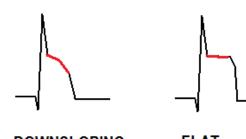
# **ST SEGMENT ELEVATION:**

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

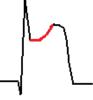


IN THIS CASE, a normal response to balloon occlusion of the RIGHT CORONARY ARTERY during PTCA in the CARDIAC CATH LAB

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







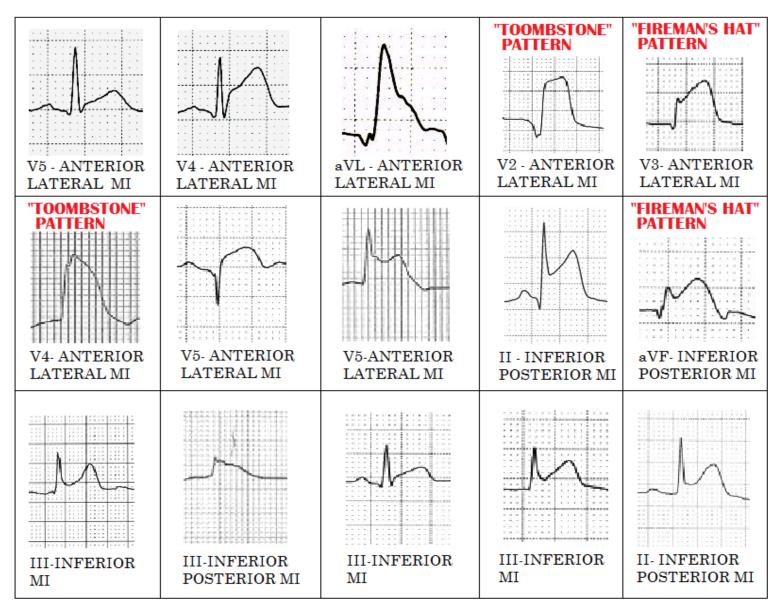
DOWNSLOPING S-T SEGMENT

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:



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

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

The presence of S-T Depression on an EKG which exhibits significant S-T elevation is a fairly reliable indicator that STEMI is the diagnosis. Reciprocal S-T Segment Depression may or may not be present during STEMI.

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

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

# ACUTE MI

# **COMPLICATIONS TO ANTICIPATE FOR ALL MI PATIENTS :**





## **FAILURE OF STRUCTURE(S) SERVED BY THE BLOCKED ARTERY**

Lancaster County, Pennsylvania Winter, 2002

tentine.

-



"NOWHERE", NEW MEXICO, 1994

### 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.

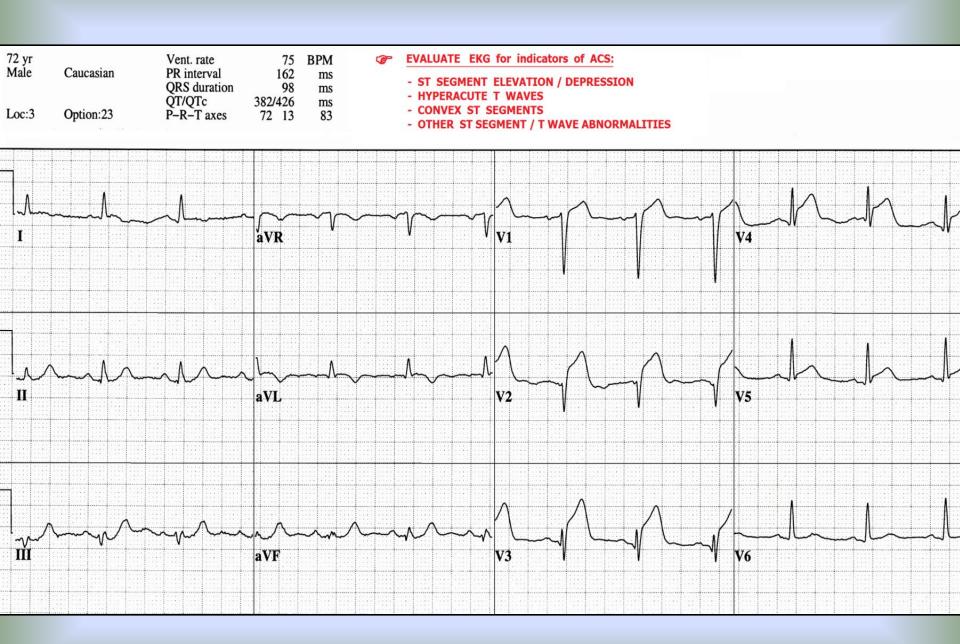
### RISK FACTOR PROFILE:

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

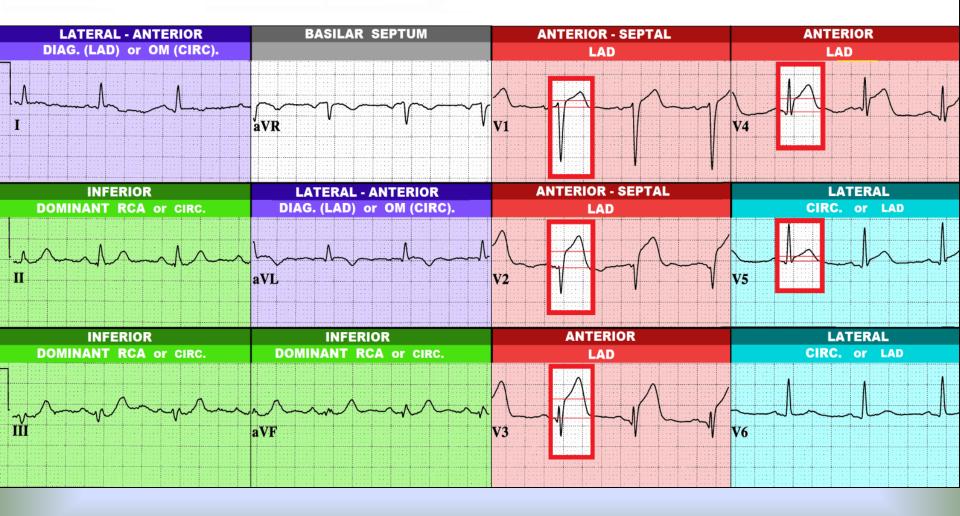
PHYSICAL EXAM: Patient calm, alert, oriented X 4, skin cool, dry, pale. No JVD, Lungs clear bilaterally. Heart sounds normal S1, S2. No peripheral edema.

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

LABS: FIRST TROPONIN: 6.4



72 yr	<b>a</b> .	Vent. rate	75	BPM	Normal sinus rhythm	
Male	Caucasian	PR interval	162	ms	Anteroseptal infarct, possibly acute	ST SEGMENT ELEVATION
l		QRS duration	98	ms	*** ** ** ** * ACUTE MI ** ** ** **	ST SEGMENT ELEVATION
Loc:	Option:2	QT/QTc P–R–T axes	382/426 72 13	ms 83	Abnormal ECG	

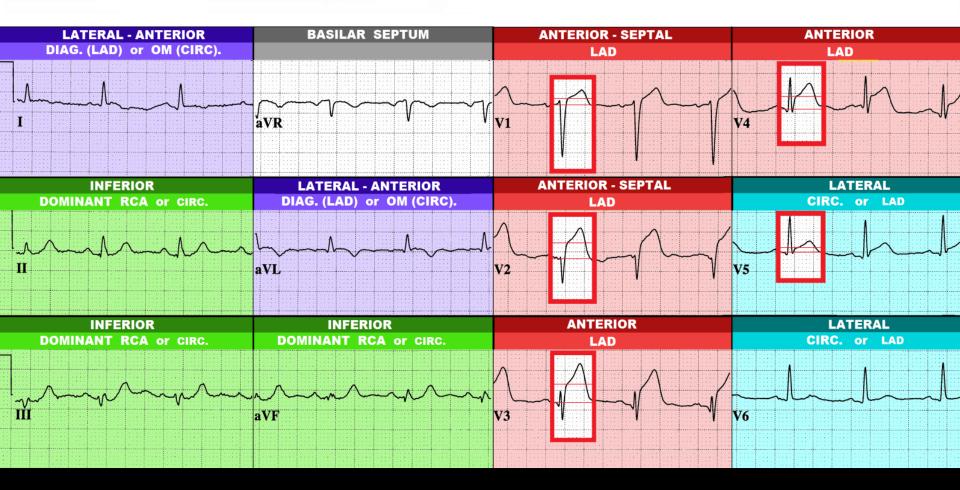


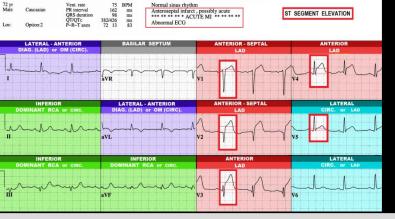
# Note: There is NO Reciprocal ST Depression on this STEMI ECG !

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

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

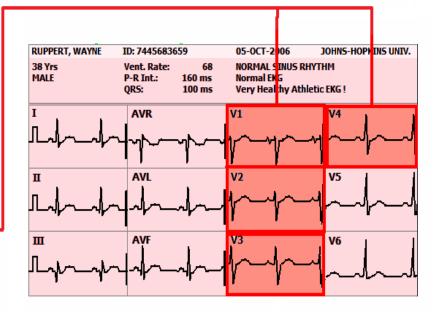
#### ST SEGMENT ELEVATION



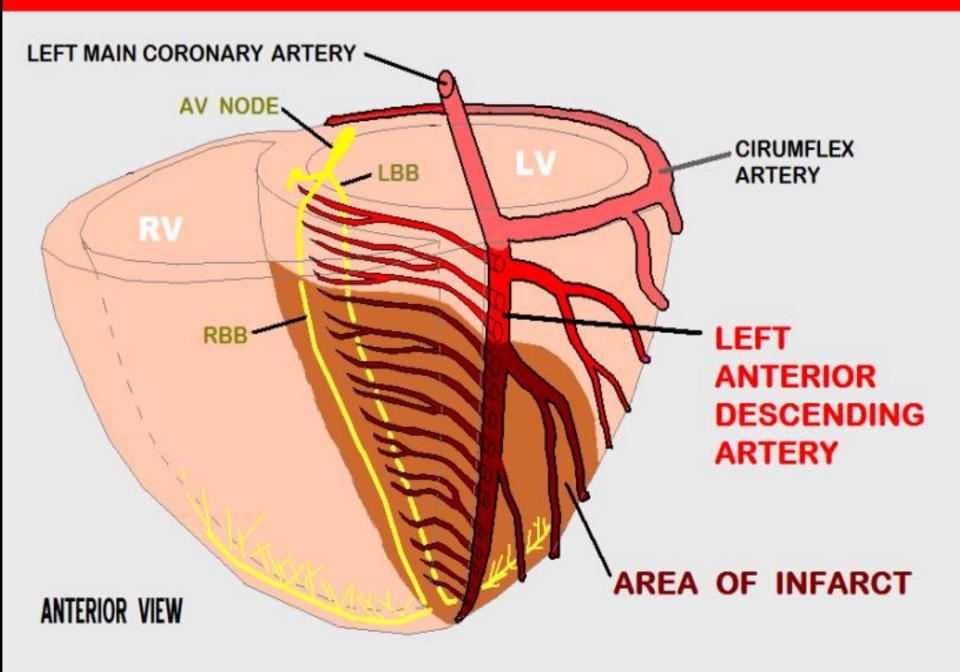


### V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL of the LEFT VENTRICLE

RV LV V6 V5 ANTERIOR CHEST V1 V2 V3 V4 WALL V1, V2 - ANTERIOR / SEPTAL V3, V4 - ANTERIOR



### **OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY**



## LAD DISTRIBUTION

### 35-45% of LV MUSCLE MASS

2

**FUNCTION** Α **BLOCKAGE** OF THE LAD CAN RESULT IN \* CARDIOGENIC SHOCK LV PUMP FAILURE --**PULMONARY EDEMA** 

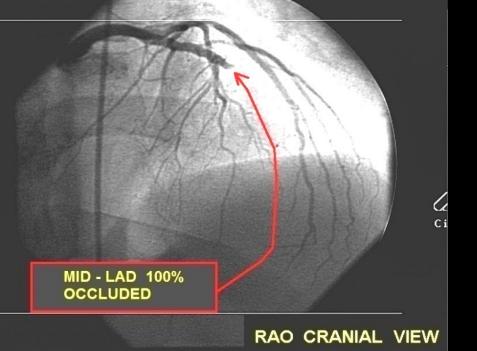


# LEFT ANTERIOR DESCENDING ARTERY (LAD)

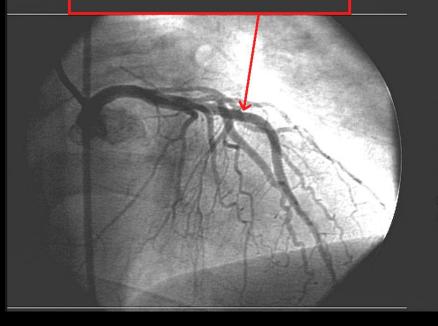
- ANTERIOR WALL OF LEFT VENTRICLE
- 35-45% OF LEFT VENTRICLE MUSCLE MASS
  - SEPTUM, ANTERIOR 2/3
  - **BUNDLE BRANCHES** 
    - ANTERIOR-MEDIAL PAPILLARY MUSCLE

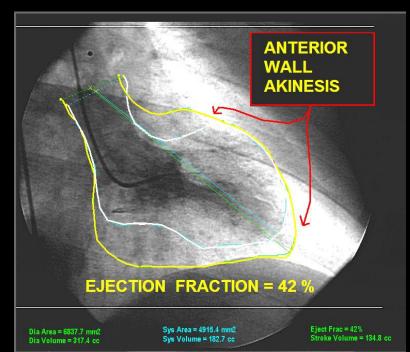
AN	NTICIPATED COMPLICATIONS of A	ANTERIOR-SEPTAL WALL
ST	TEMI	
&	POSSIBLE INDICATED INTERVEN	NTIONS:

- CARDIAC ARREST	BCLS / ACLS
- CARDIAC DYSRHYTHMIAS	ACLS (antiarrhythmics)
(VT / VF)	
- PUMP FAILURE with	INOTROPE THERAPY:
CARDIOGENIC SHOCK	-DOPAMINE / DOBUTAMINE /
	LEVOPHED
	- INTRA-AORTIC BALLOON
	PUMP
	(use caution with fluid
	challenges due to
	PULMONARY EDEMA)
- PULMONARY EDEMA	- CPAP
	- ET INTUBATION
	(use caution with dieuretics
	due to pump failure and
	hypotension)



#### POST PTCA/STENT TO MID LAD





### CASE STUDY 2: STEMI

### CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

### RISK FACTOR PROFILE:

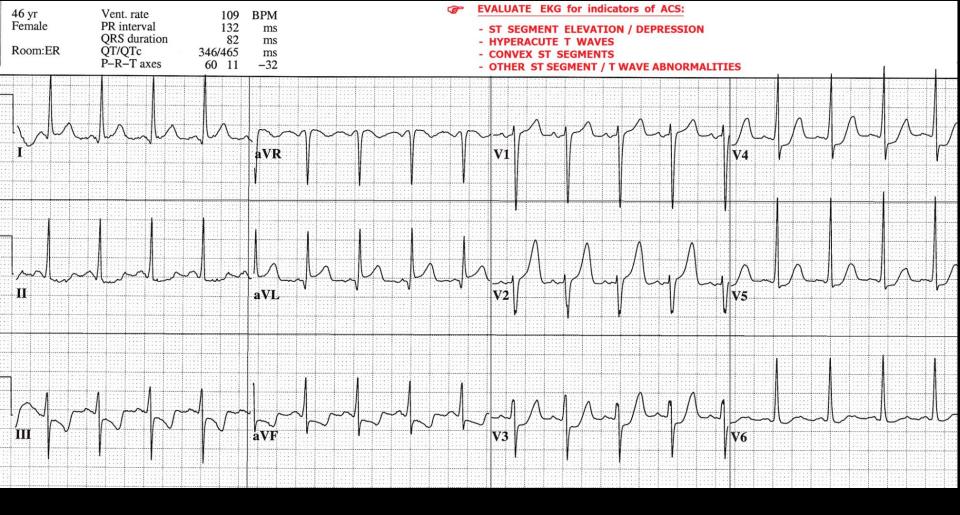


**FAMILY HISTORY** - father died of CAD, older brother had CABG, age 39 DIABETES - diet controlled HYPERTENSION

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

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

LABS: TROPONIN ultra = 2.8



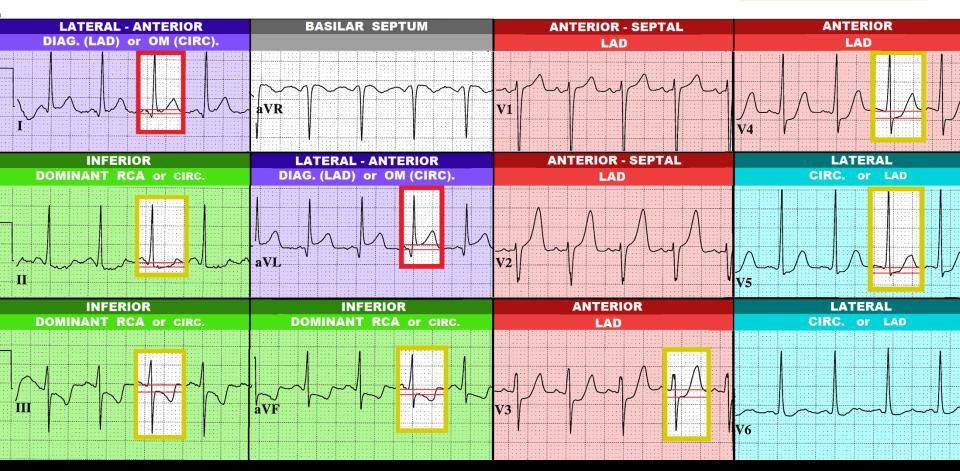
46 yr	Vent. rate	109	BPM
Female	PR interval	132	ms
	QRS duration	82	ms
Room:ER	QT/QTc	346/465	ms
	P–R–T axes	60 11	-32

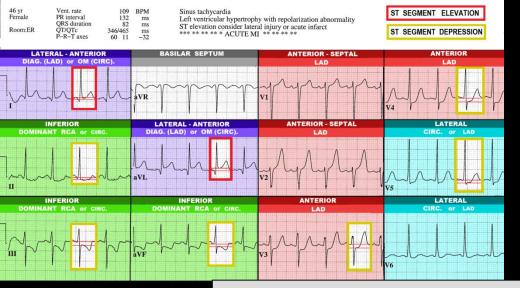
Sinus tachycardia

Left ventricular hypertrophy with repolarization abnormality

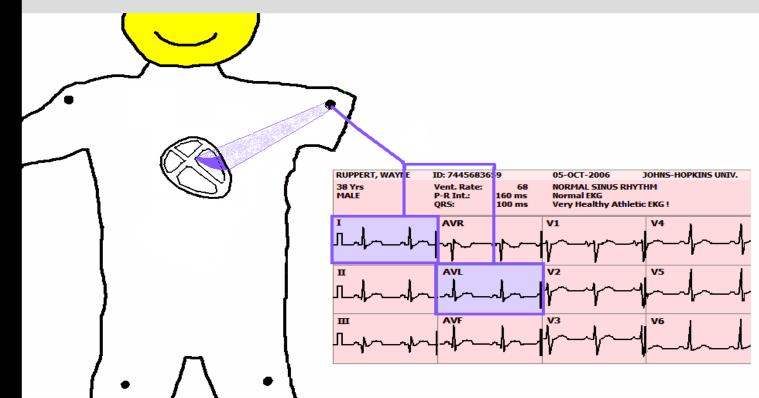
ST elevation consider lateral injury or acute infarct \*\*\* \*\* \*\* ACUTE MI \*\* \*\* \*\* ST SEGMENT ELEVATION

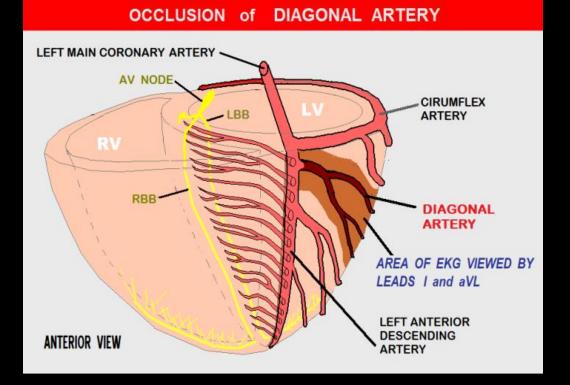
ST SEGMENT DEPRESSION



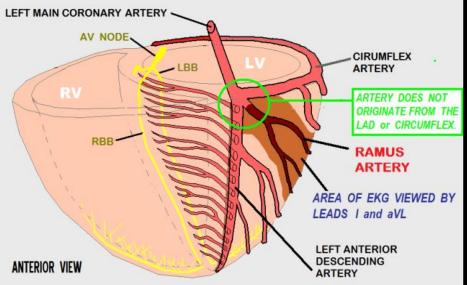


## **LEADS I and aVL view the ANTERIOR-LATERAL JUNCTION**

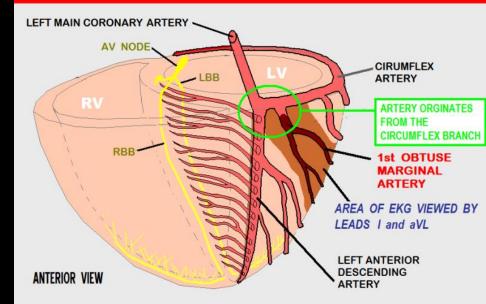




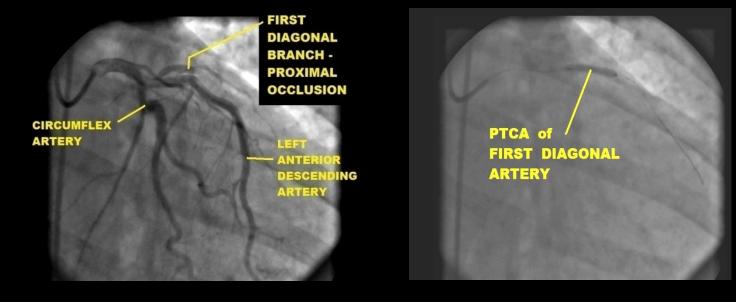
# OCCLUSION of RAMUS ARTERY

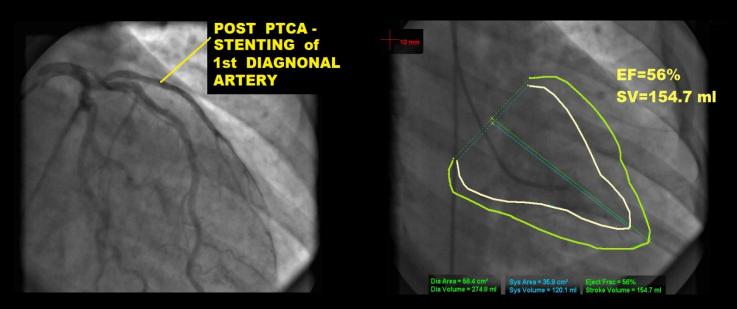


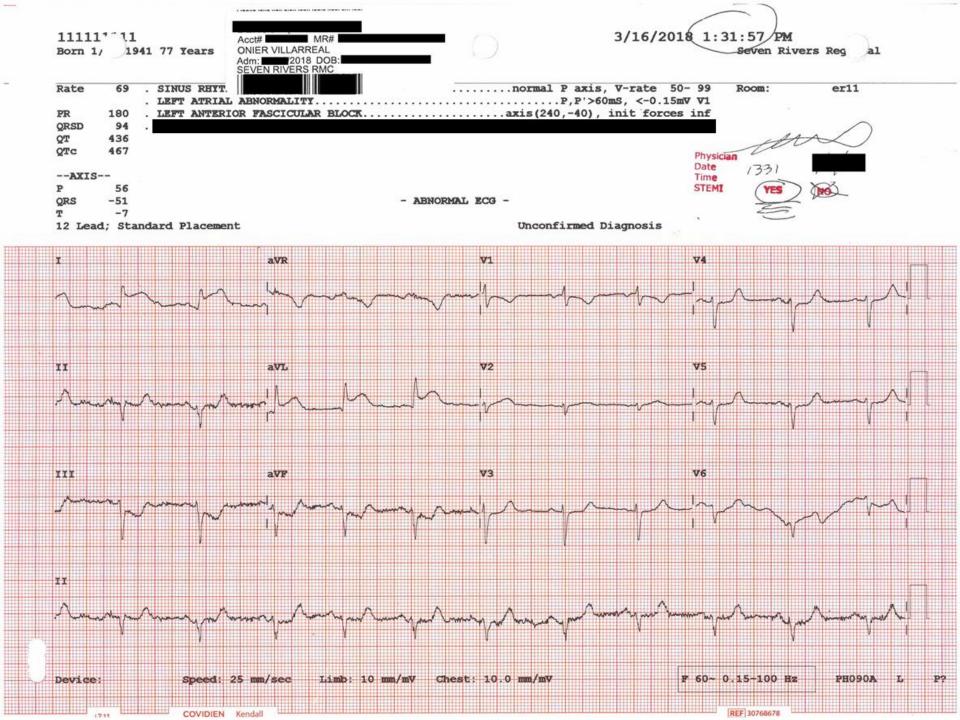
#### OCCLUSION of OBTUSE MARGINAL ARTERY



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

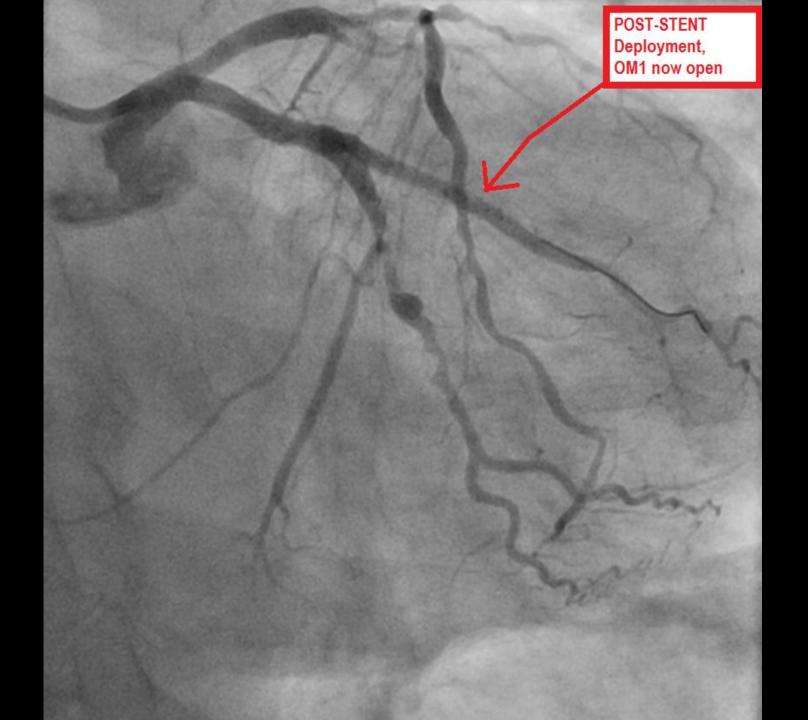












# CASE STUDY 3: STEM

## 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.

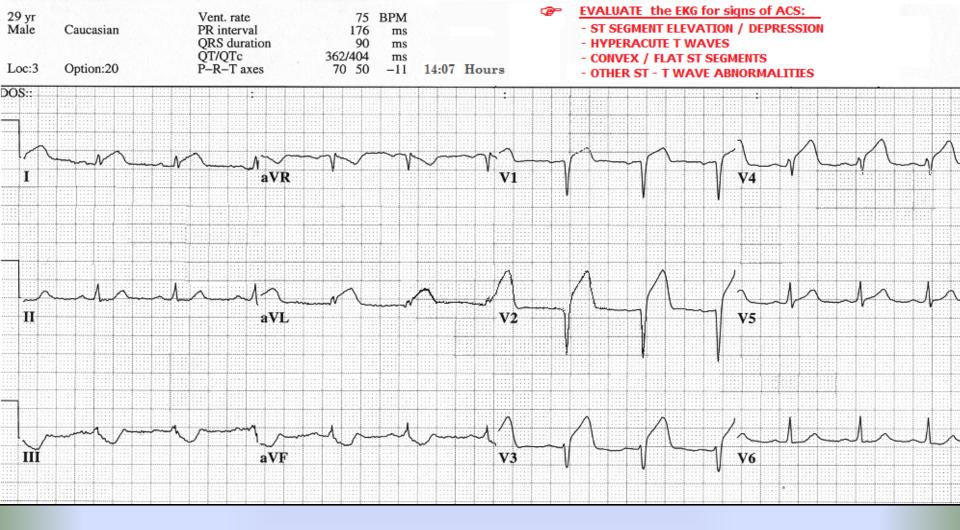
## **RISK FACTOR PROFILE:**

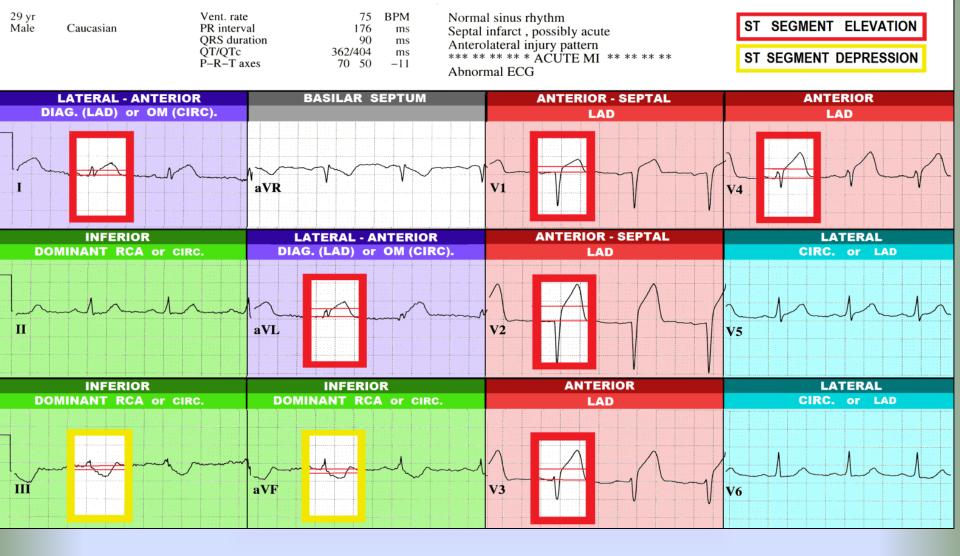
- FAMILY HISTORY father died of MI age 46
- CURRENT CIGARETTE SMOKER
- "MILD" HYPERTENSION untreated
- CHOLESTEROL unknown "never had it checked."

**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.

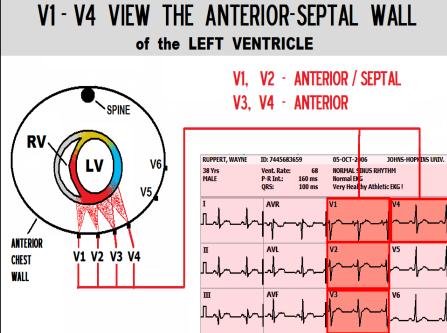
VITAL SIGNS: BP: 104/78, P: 76, R: 20, SAO2: 96%

LABS: INITIAL CARDIAC MARKERS - NEGATIVE

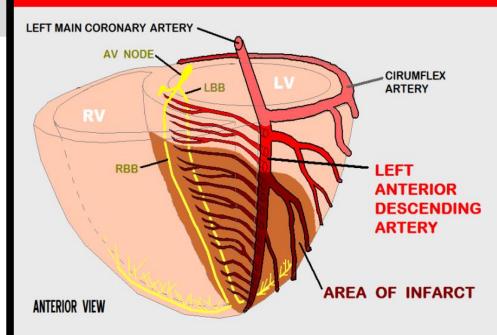


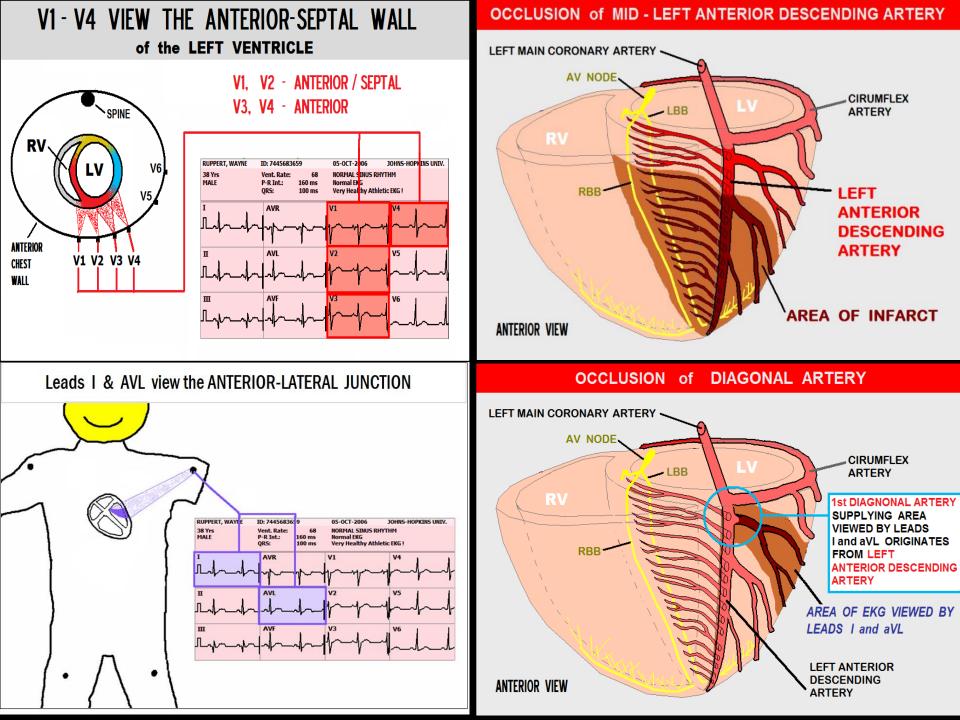


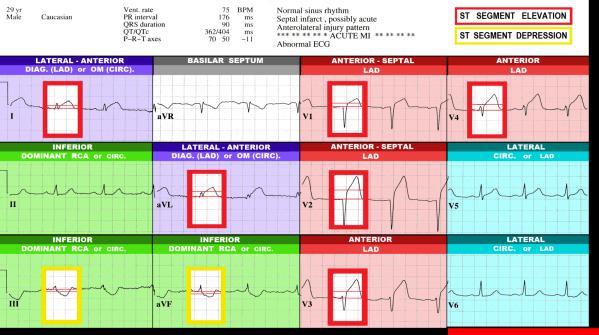
# Reciprocal ST Depression is NOW PRESENT Additional ST Elevation is present in Leads I, AVL



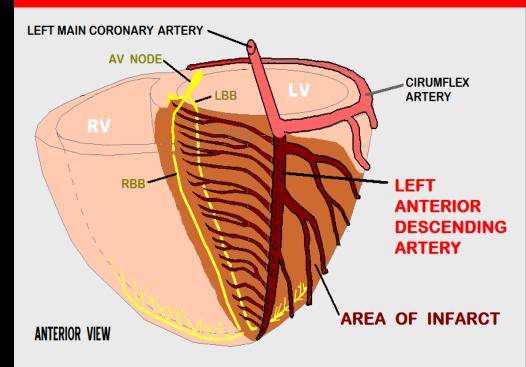
#### OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY



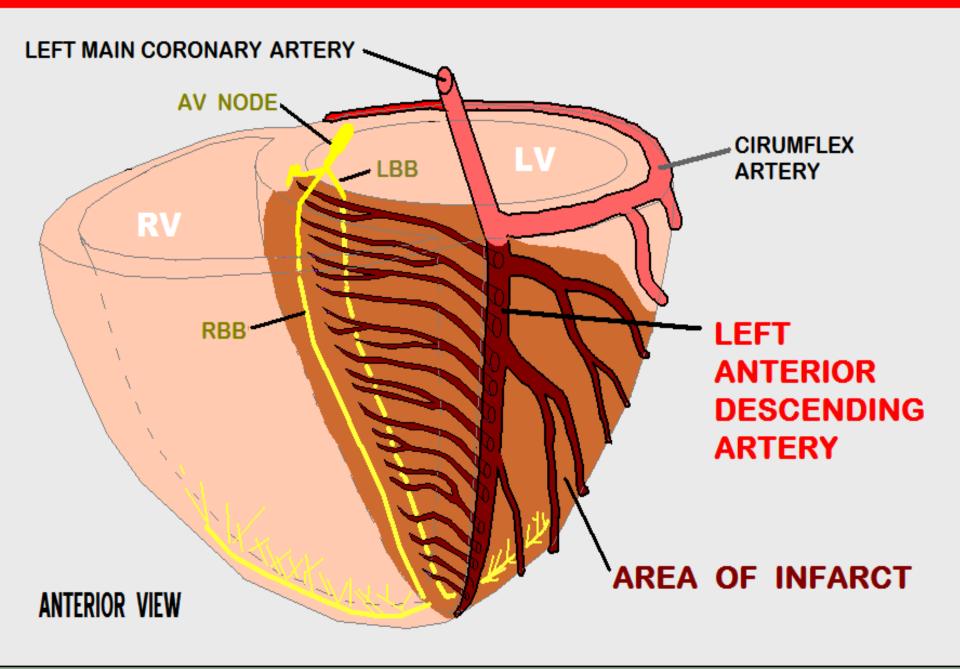




#### OCCLUSION of PROXIMAL LEFT ANTERIOR DESCENDING ARTERY



# **OCCLUSION** of PROXIMAL LEFT ANTERIOR DESCENDING ARTERY

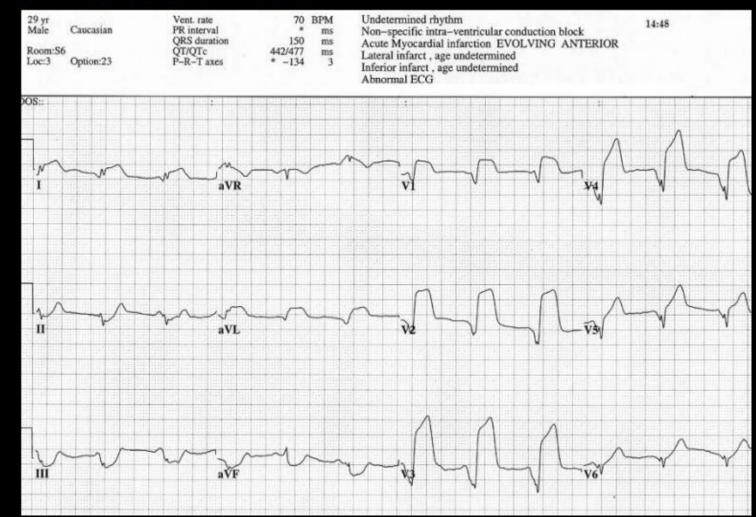


ANTICIPATED COMPLICATIONS of ANTERIOR-SEPTAL WALL
STEMI

& POSSIBLE INDICATED INTERVENTIONS:

& PUSSIBLE INDICATED INTERVENTIONS.			
- CARDIAC ARREST	BCLS / ACLS		
- CARDIAC DYSRHYTHMIAS	ACLS (antiarrhythmics)		
(VT / VF)			
- PUMP FAILURE with	INOTROPE THERAPY:		
CARDIOGENIC SHOCK	-DOPAMINE / DOBUTAMINE /		
	LEVOPHED		
	- INTRA-AORTIC BALLOON		
	PUMP		
	(use caution with fluid		
	challenges due to		
	PULMONARY EDEMA)		
- PULMONARY EDEMA	- CPAP		
	- ET INTUBATION		
	(use caution with dieuretics		
	due to pump failure and		
	hypotension)		

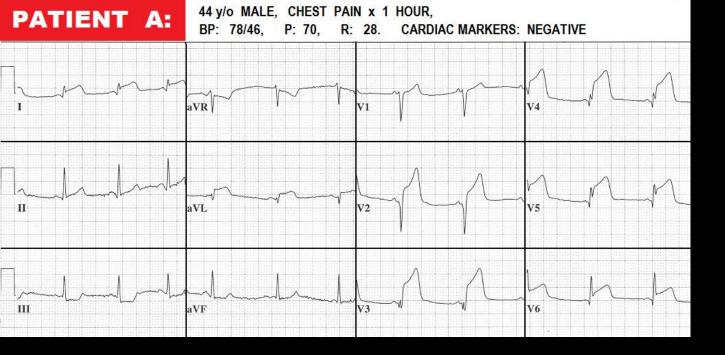
# WHILE AWAITING THE CATH TEAM, THE PATIENT BEGAN VOMITING. SKIN BECAME ASHEN & DIAPHORETIC. REPEAT BP = 50/30. -WHAT THERAPEUTIC INTERVENTIONS SHOULD BE IMPLMENTED AT THIS POINT ?

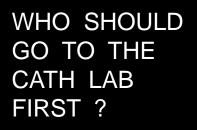


PROXIMAL OCCLUSION of the LEFT ANTERIOR DESCENDING Artery



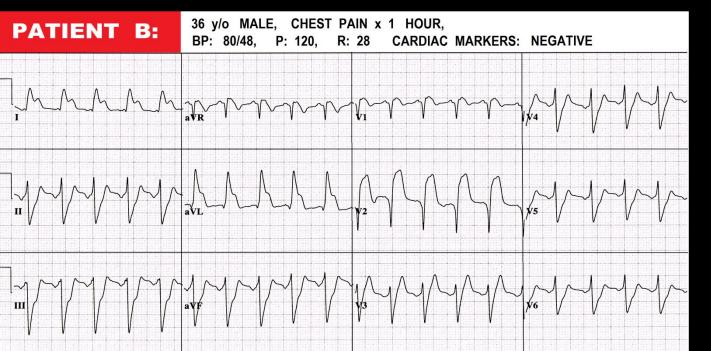
POST PTCA \_\_\_\_\_ and STENT to the PROXIMAL LAD

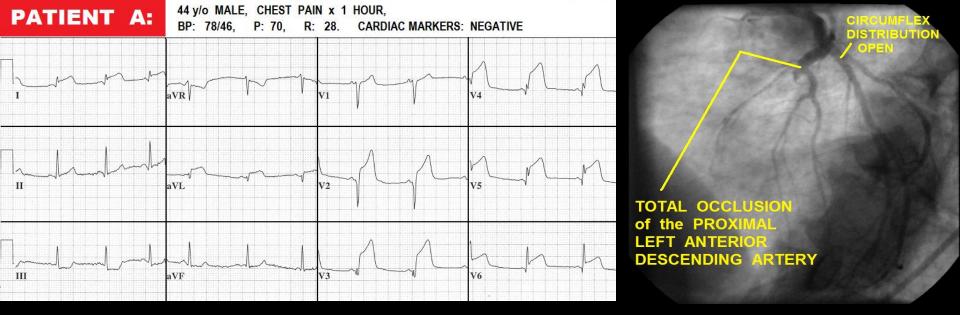


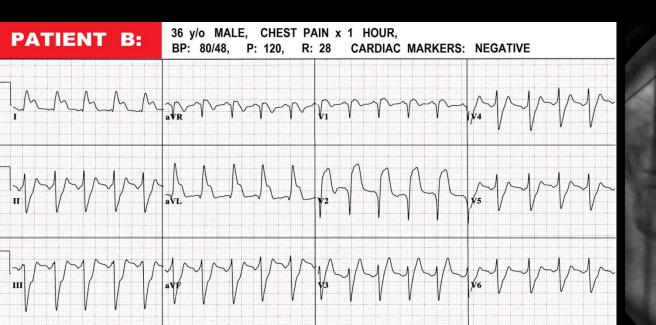


And . . . .

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



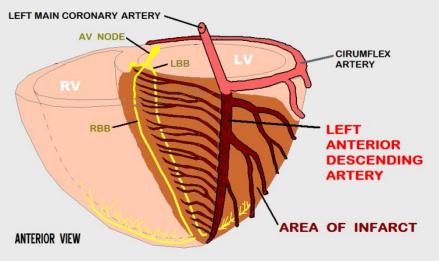




TOTAL OCCLUSION of LEFT MAIN CORONARY ARTERY

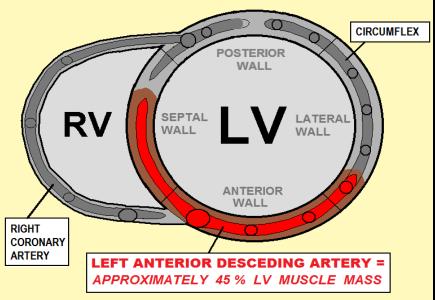
# PATIENT A:

#### OCCLUSION of PROXIMAL LEFT ANTERIOR DESCENDING ARTERY

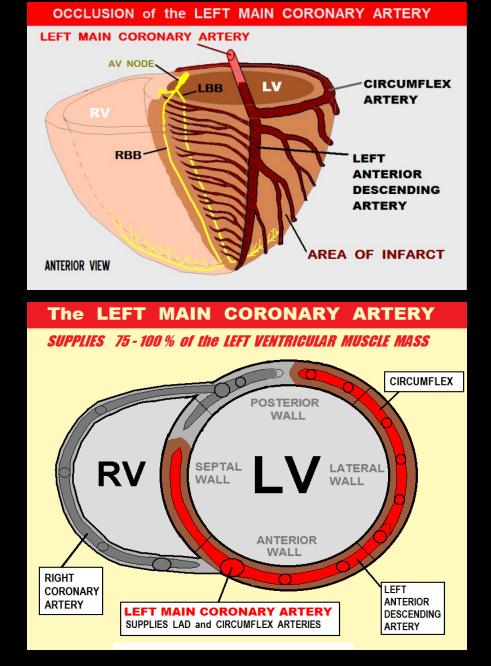


The LEFT ANTERIOR DESCENDING ARTERY

SUPPLIES 40 - 50 % OF THE LEFT VENTRICULAR MUSCLE MASS

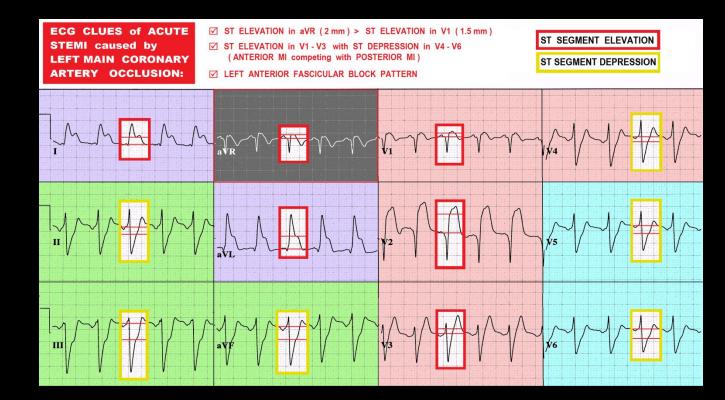


# PATIENT B:

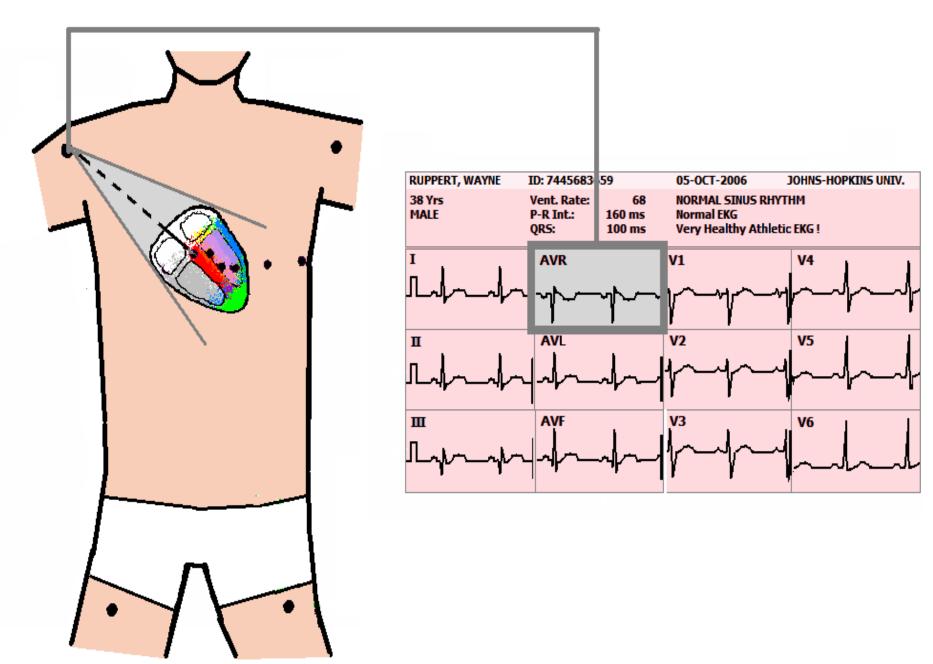


# **PECG Clues...** for identifying stemi caused by LEFT MAIN CORONARY ARTERY occlusion:

- ☑ ST ELEVATION in ANTERIOR LEADS (V1 V4) and LATERAL LEADS (V5 & V6)
- ☑ ST DEPRESSION or ISOELCTRIC J POINTS may be seen in V LEADS....mainly V2 and/or V3 caused by COMPETING FORCES of ANTERIOR vs. POSTERIOR WALL MI.\*+
  - → NOTE: it is very unusual to see ST DEPRESSION in V LEADS with isolated ANTERIOR WALL MI when caused by occluded LAD.
- ☑ ST ELEVATION in AVR is GREATER THAN ST ELEVATION in V1\*\*
- ☑ ST ELEVATION in AVR GREATER THAN 0.5 mm
- ☑ ST ELEVATION in LEAD I and AVL (caused by NO FLOW to DIAGONAL / OBTUSE MARGINAL BRANCHES)\*
- ☑ ST DEPRESSION in LEADS II, III, and AVF. (in cases of LMCA occlusion of DOMINANT CIRCUMFLEX, leads II, III, and AVF may show ST ELEVATION or ISOELECTRIC J POINTS)\*+
- ☑ NEW / PRESUMABLY NEW RBBB, and/or LEFT ANTERIOR FASICULAR BLOCK\*+
- \* Kurisu et al, HEART 2004, SEPTEMBER: 90 (9): 1059-1060
- + Yamaji et al, JACC vol. 38, No. 5, 2001, November 1, 2001:1348-54

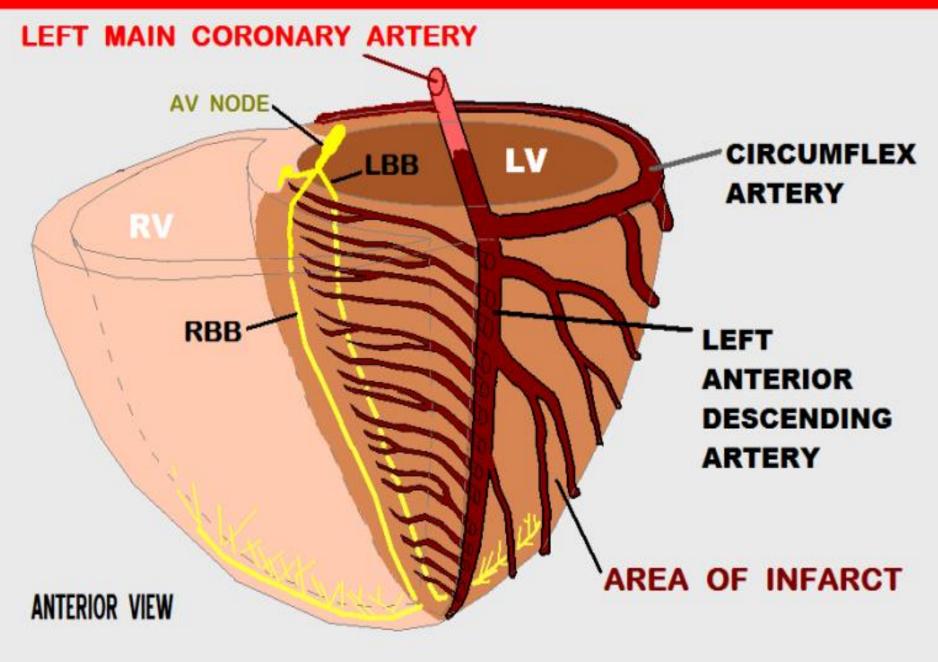


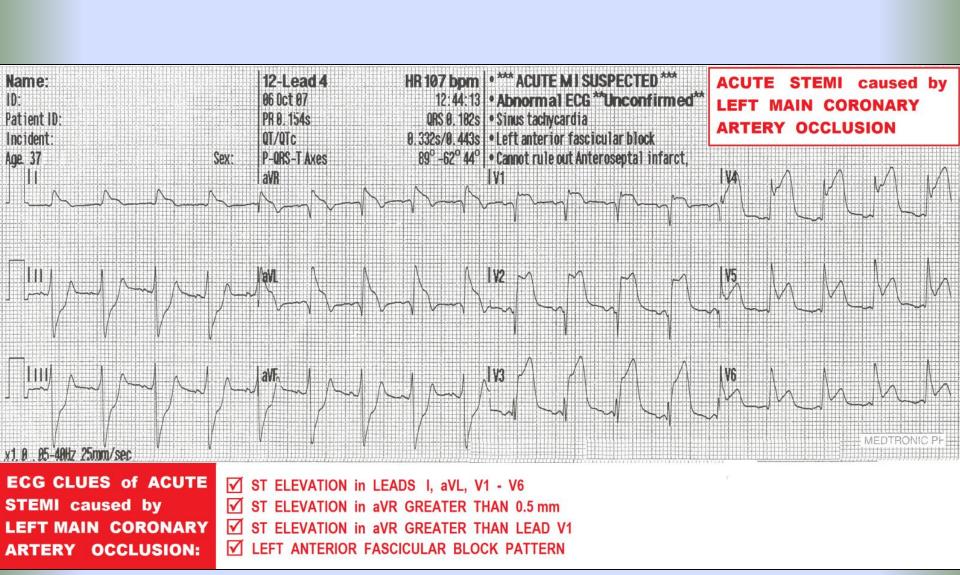
Lead AVR Views the BASILAR SEPTUM (region of the Bundle of His)

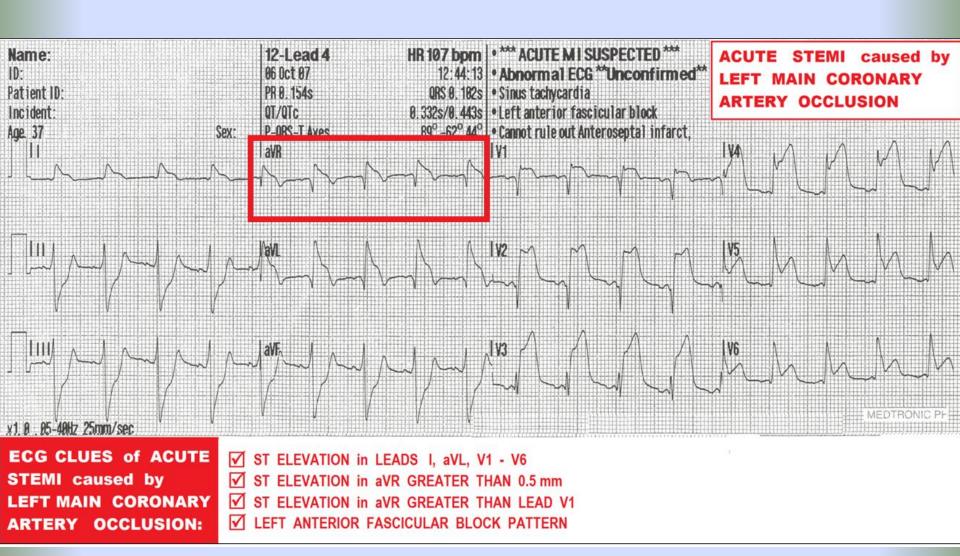


In STEMI with ST-Segment Elevation in Lead AVR, This is indicative of Left Main Coronary Artery Occlusion . . .

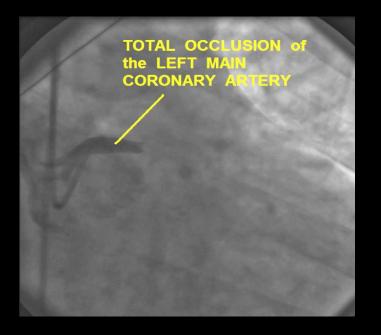
# **OCCLUSION** of the LEFT MAIN CORONARY ARTERY



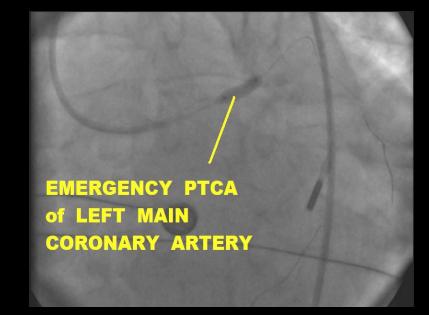


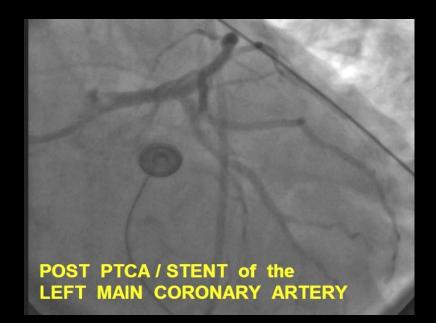


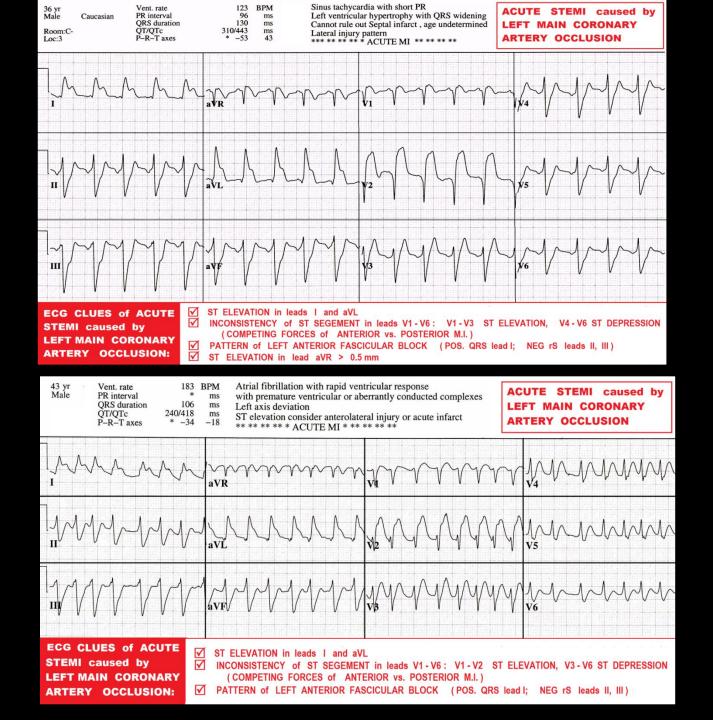
# TOTAL OCCLUSION of the LEFT MAIN CORONARY ARTERY

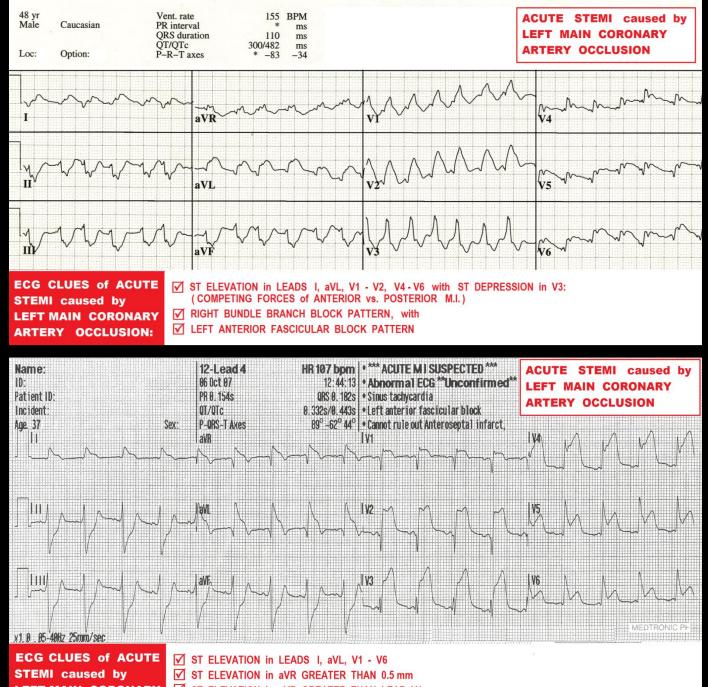


Despite the dismal mortality rate associated with STEMI from total LMCA occlusion, this patient survived and was later discharged. His EF is estimated at approximately 30%. He received an ICD, and is currently stable.









**LEFT MAIN CORONARY** ST ELEVATION in aVR GREATER THAN LEAD V1 **ARTERY OCCLUSION:** □ LEFT ANTERIOR FASCICULAR BLOCK PATTERN

LEFT MAIN CORONARY ARTERY -TOTAL OCCLUSION

# CASE STUDY 4: CRITICAL DECISIONS SCENARIO

### CONCLUSIONS:

- QUESTION 1: WHICH PATIENT SHOULD BE TAKEN FIRST FOR IMMEDIATE CARDIAC CATHETERIZATION for EMERGENCY PCI ?
- ANSWER: PATIENT B was taken emergently to the Cardiac Cath Lab both the ED physician and the Interventional Cardiologist correctly identified the EKG patterns of LMCA occlusion.
- QUESTION 2: WHAT COURSE OF ACTION SHOULD BE TAKEN WITH THE PATIENT NOT CHOSEN TO BE SENT TO THE CATH LAB FIRST?
- ANSWER: PATIENT A received thrombolytic therapy in the ED. It was determined that THROMBOLYTIC THERAPY would achieve the FASTEST ROUTE to REPERFUSION ---- by at least 60 minutes.

# CASE STUDY 7 - STEMI

## CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

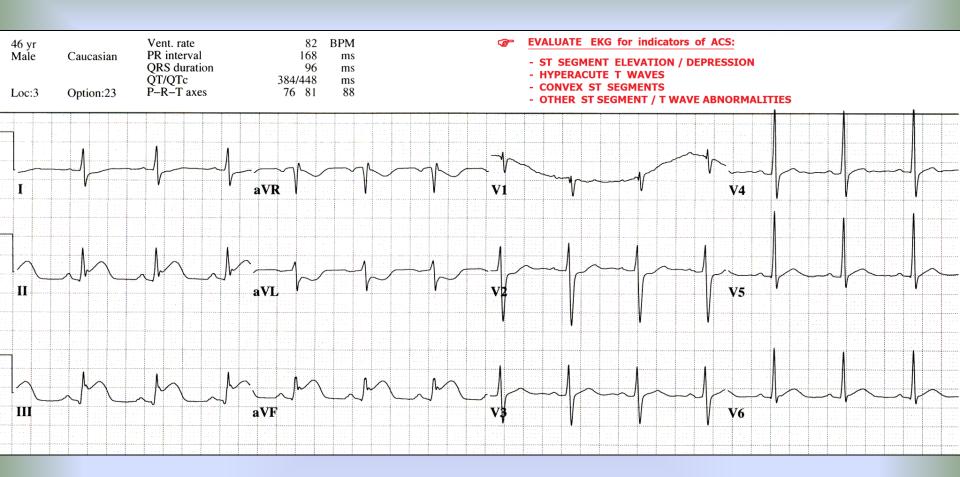
## RISK FACTOR PROFILE:

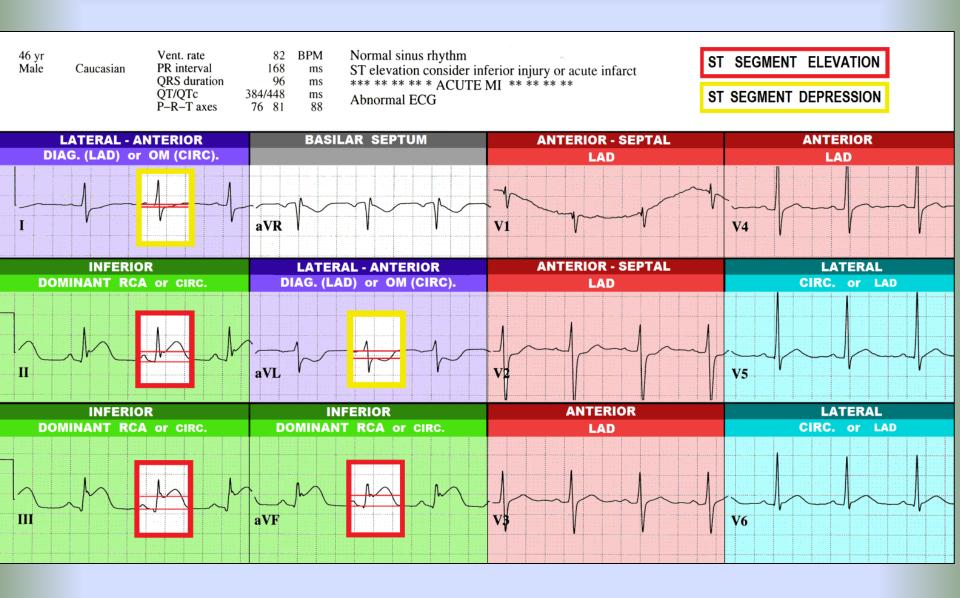
- CURRENT CIGARTTE SMOKER x 18 YEARS
- HYPERTENSION
- HIGH LDL CHOLESTEROL

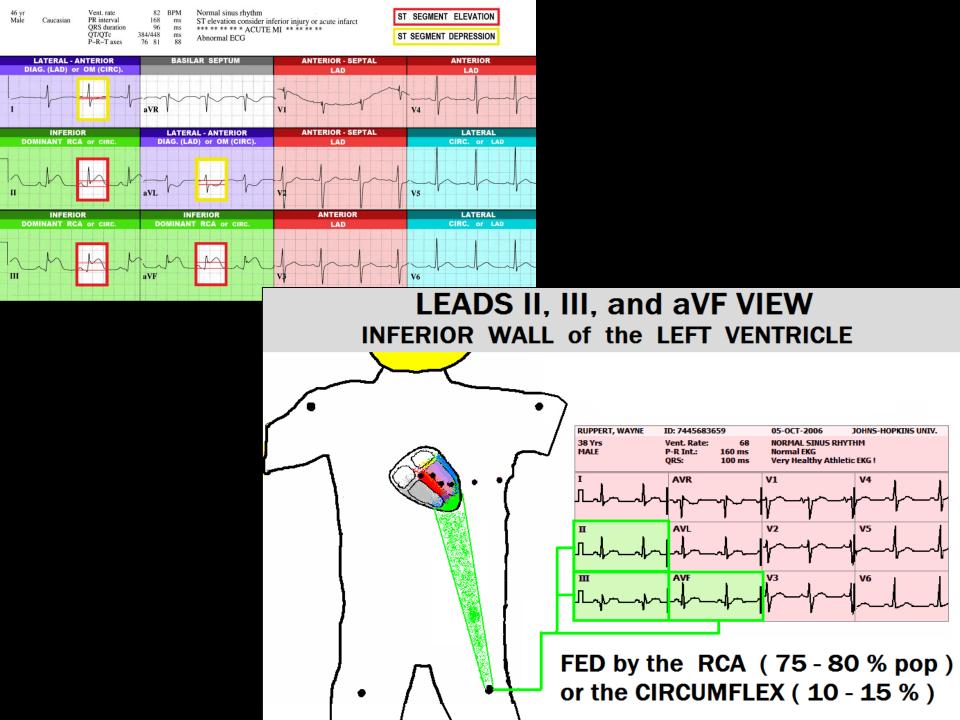
PHYSICAL EXAM: Patient is alert & oriented x 4, skin warm, dry, color normal. Non-anxious Lungs clear, normal S1, S2. No JVD, No ankle edema.

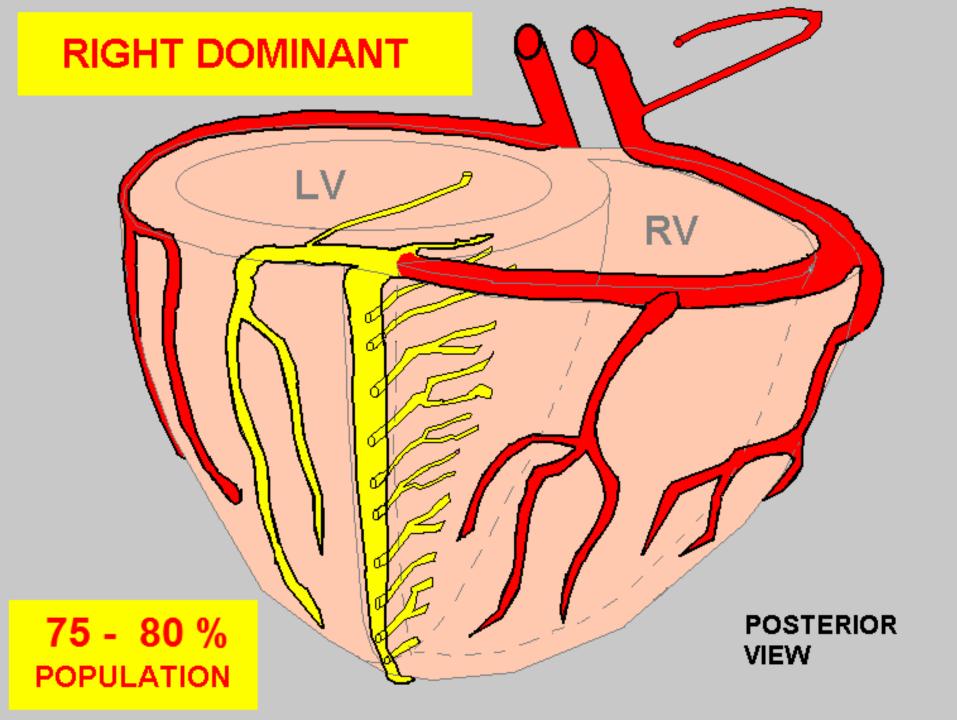
VITAL SIGNS: BP: 136/88 P: 88 R: 20 SAO2: 100% on 4 LPM O2

LABS: TROPONIN: < .04



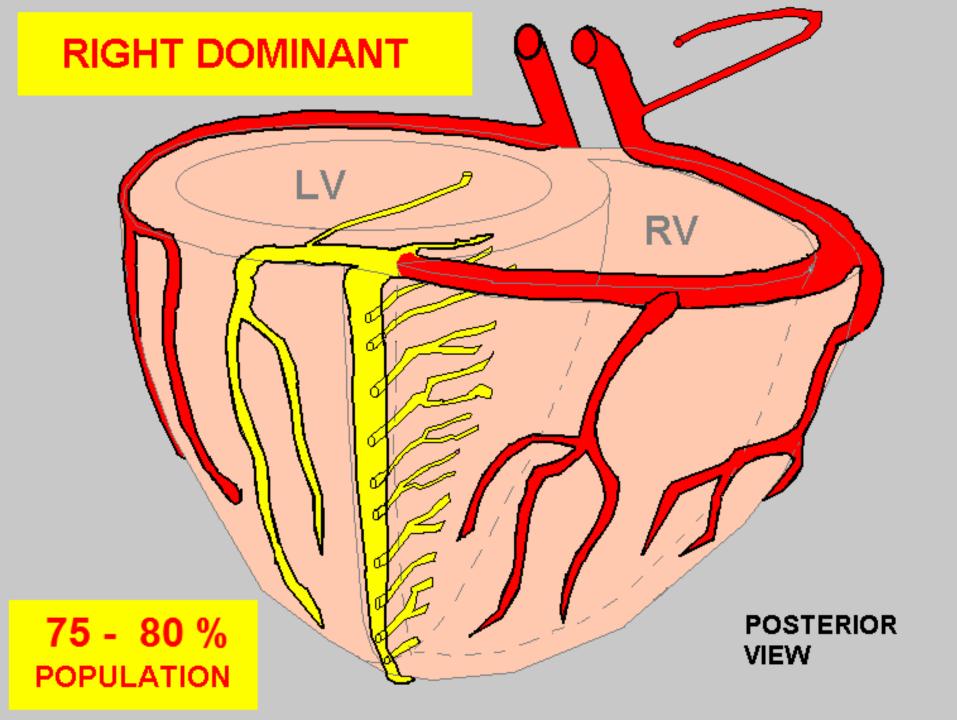






**RIGHT CORONARY ARTERY (RCA) RIGHT DOMINANT** SYSTEMS RIGHT ATRIUM SINUS NODE (55% of the population) RIGHT VENTRICLE - 100 % of muscle mass LEFT VENTRICLE: 15 - 25 % of muscle mass - INFERIOR WALL - approx. 1/2 of POSTERIOR WALL AV NODE

– 🕺 — 🔶 HELPFUL HINT .... MEMORIZE THIS I 🛛 🔶 🥂



### A standard

## **12 LEAD EKG**

### Does NOT show the

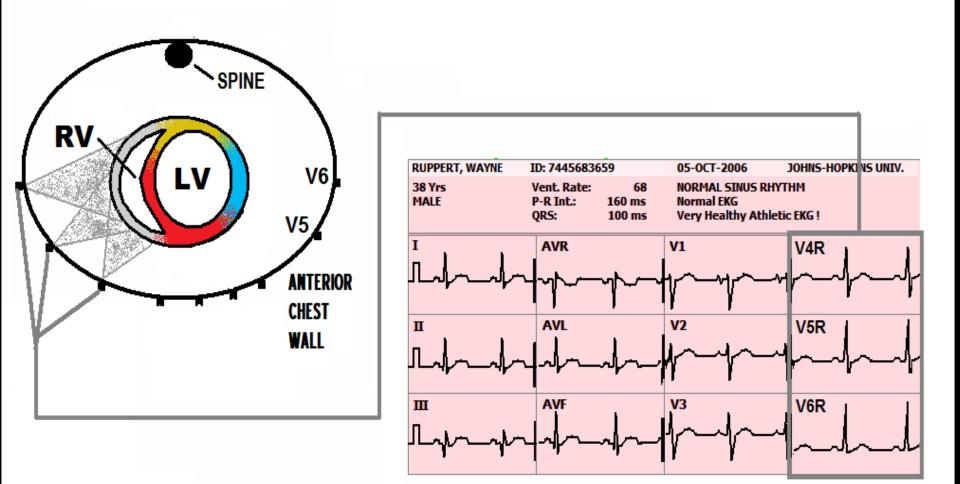
## **RIGHT VENTRICLE**

# To see the RIGHT VENTRICLE ...

# INFERIOR WALL M.I.

# @ You must do a RIGHT - SIDED EKG!!

### V4R - V6R VIEW THE RIGHT VENTRICLE



### LEAD PLACEMENT V4R, V5R, V6R

V4R V5R

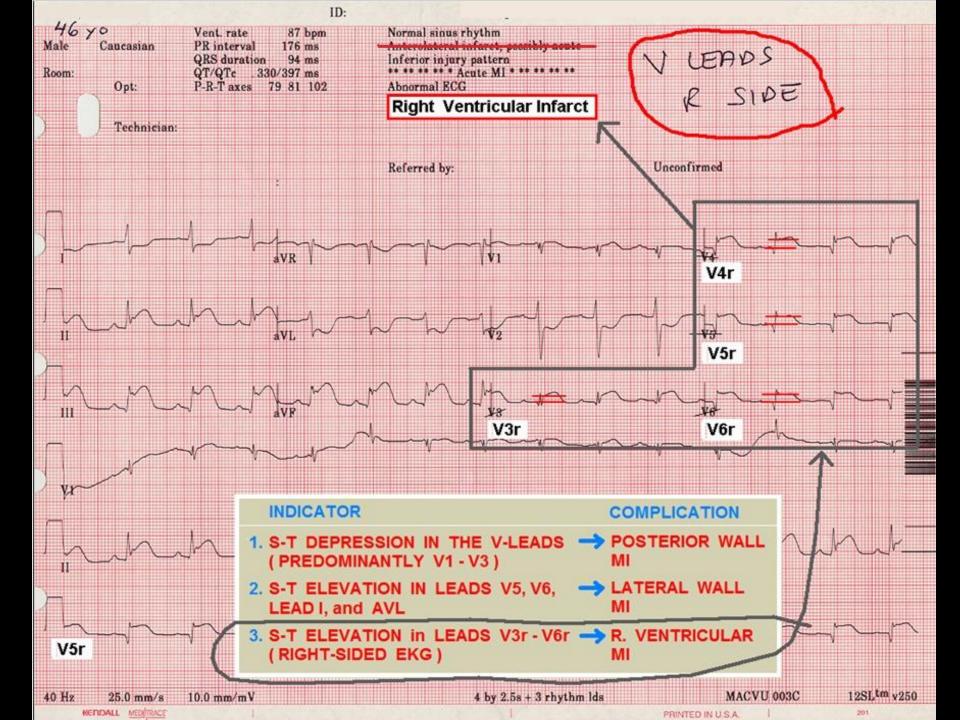
V6R

**V4** 

V3

V2

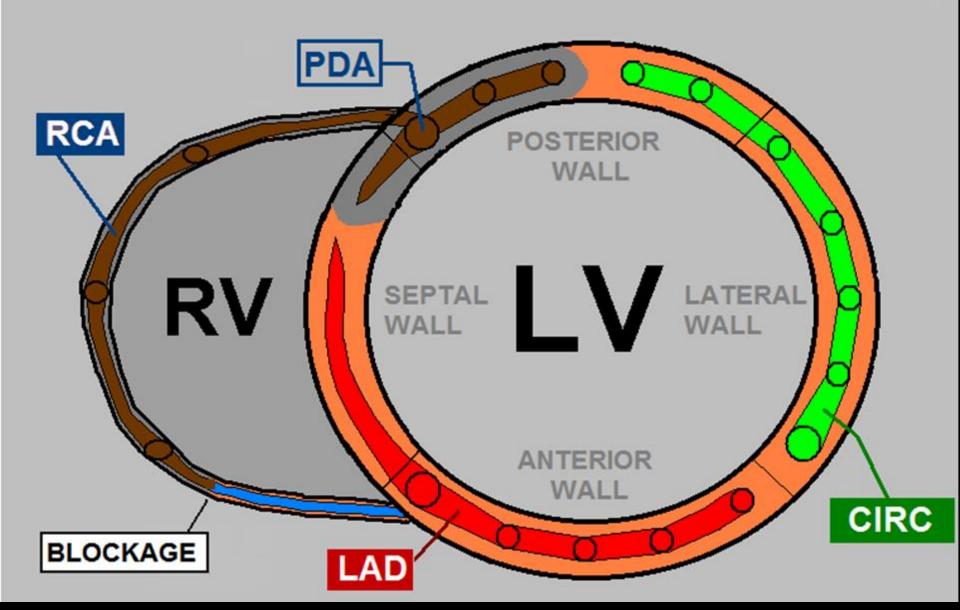
**V1** 



#### **INFERIOR - RIGHT VENTRICULAR MI**

### DOMINANT RCA

### 75-80 % of POPULATION

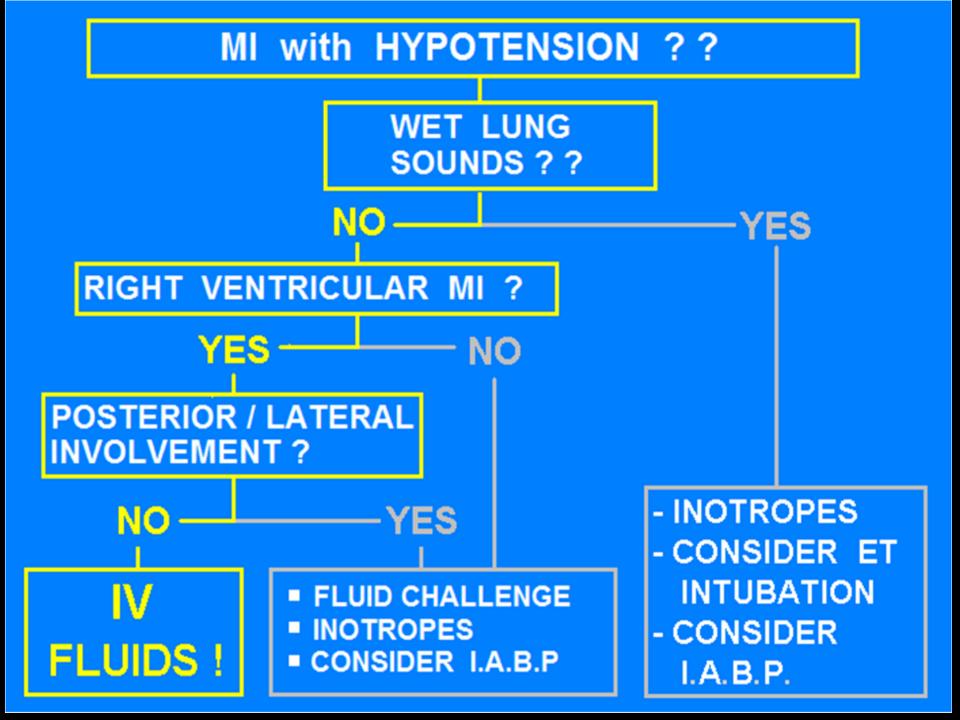


### ANTICIPATED COMPLICATIONS of INFERIOR WALL STEMI secondary to

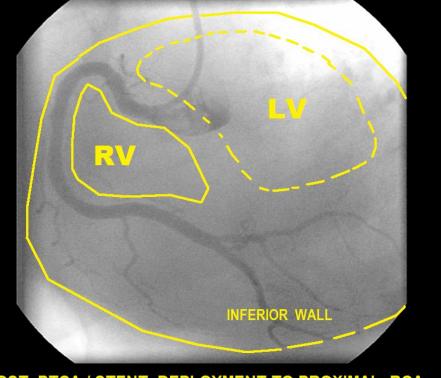
RCA Occlusion & POSSIBLE INDICATED INTERVENTIONS:

- 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	ATROPINE 0.5mg, REPEAT as needed
HB)	UP TO 3mg, Transcutaneous Pacing,
	(follow ACLS and/or UNIT protocols)
- RIGHT VENTRICULAR MYOCARDIAL	- The standard 12 Lead ECG does NOT
INFARCTION	view
	the Right Ventricle.
	- You must do a RIGHT-SIDED ECG to
	see if RV
	MI is present.
	- Do NOT give any Inferior Wall STEMI
	patient
	NITRATES or DIURETICS until RV MI
	has boon RUILED OUT
	been RULED OUT.

# If this patient becomes HYPOTENSIVE ....



### PROXIMAL OCCLUSION of the RIGHT CORONARY ARTERY.



POST PTCA / STENT DEPLOYMENT TO PROXIMAL RCA

### IN EVERY CASE of

### **INFERIOR WALL STEMI**

You must first *RULE OUT* **RIGHT VENTRICULAR MI BEFORE** giving any:

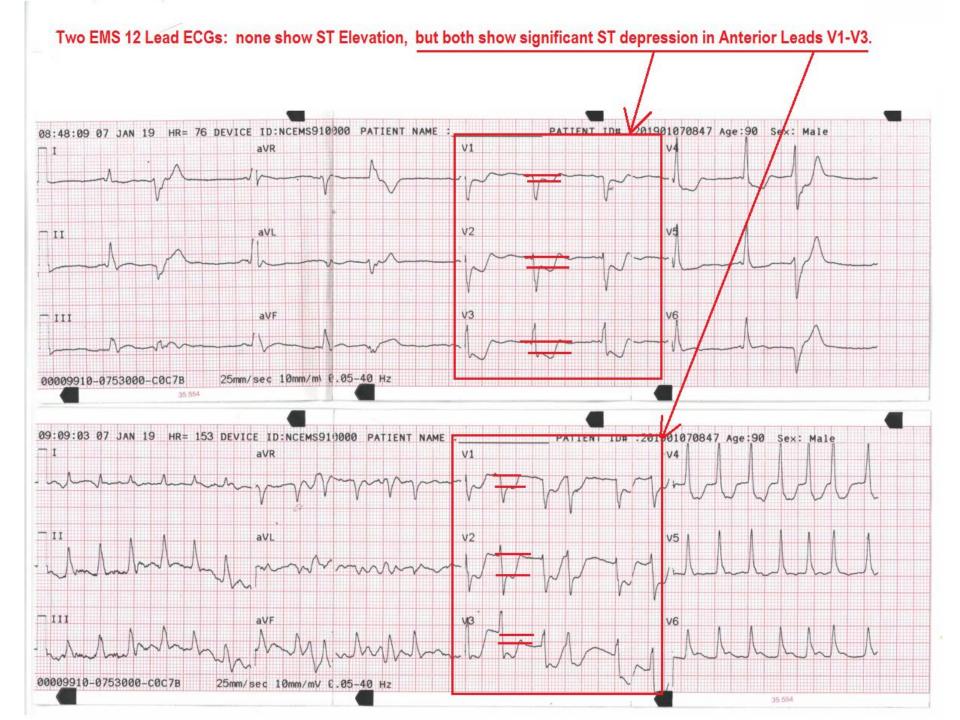
- NITROGLYCERIN
- Diuretics

## **Nitroglycerin & Diuretics** are **CLASS III CONTRINDICATED** in **RIGHT VENTRICULAR MI !!**\* **They precipitate SEVERE HYPOTENSION**

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

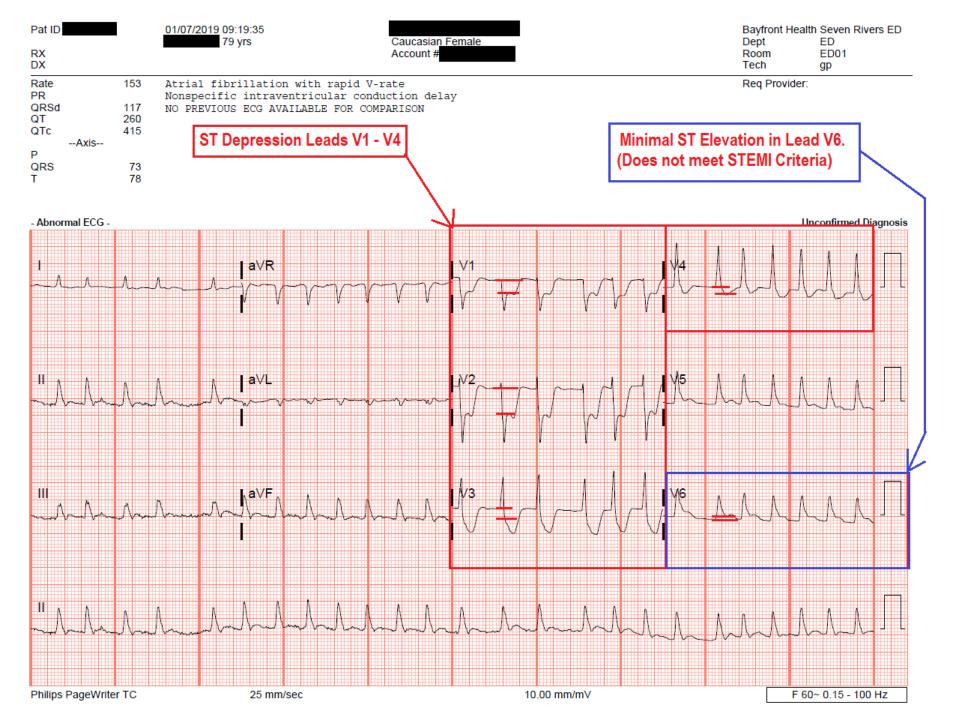
### Case Study-January 2019

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



### Initial Exam in ED

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

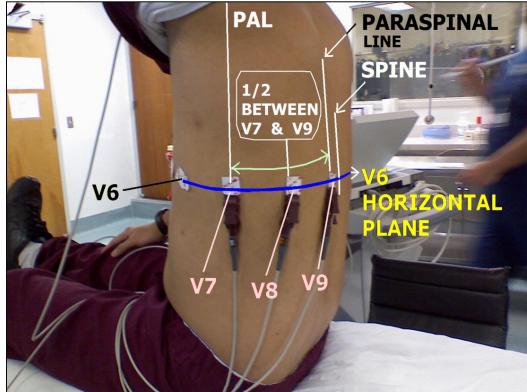


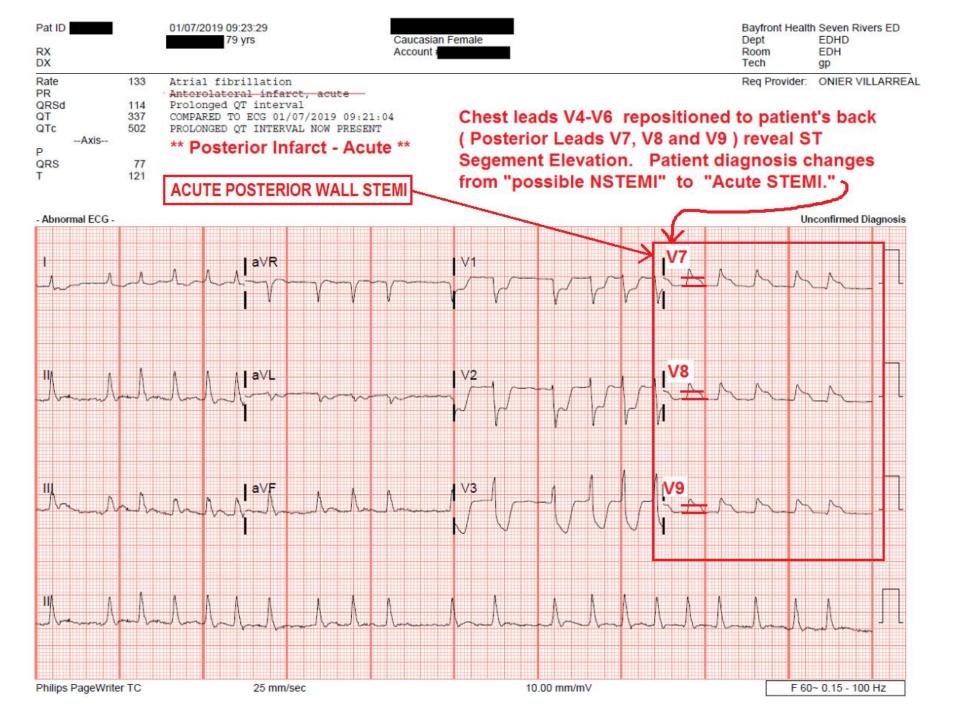
### Causes of ST Depression V1-V4

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

### Continued Exam in the ED....

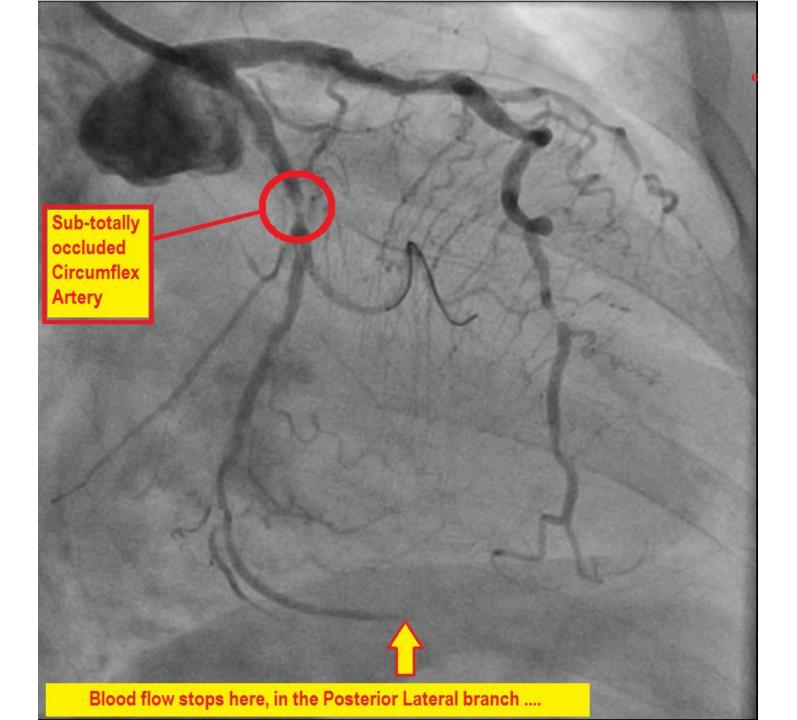
- Upon noting ST Depression in Anterior Leads, 3 leads were placed on the patient's back. The lead wires for V4, V5 and V6, were repositioned, as shown here:
- The "Posterior Lead ECG" is seen on the next slide.....

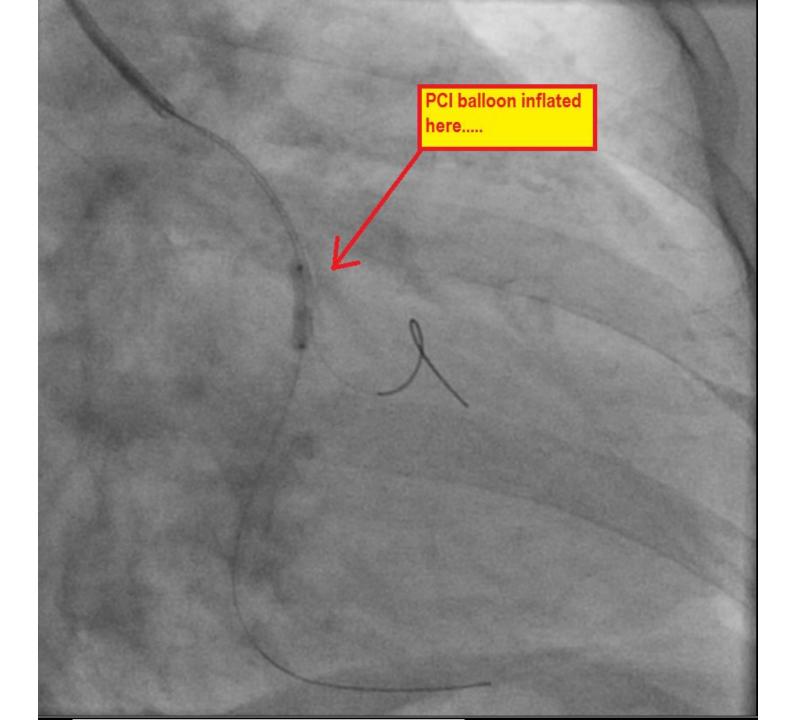


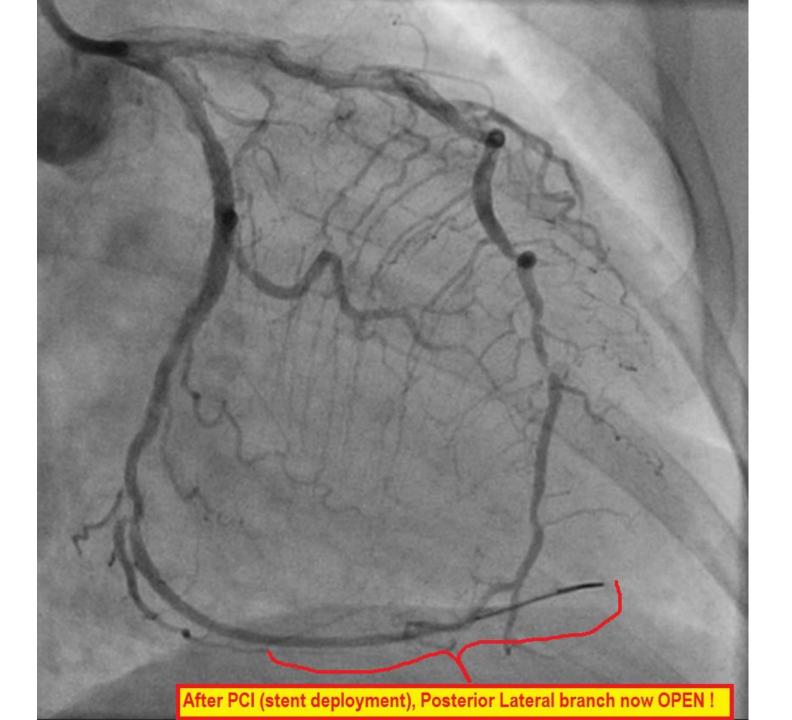


### STEMI Alert !

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......







### 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.