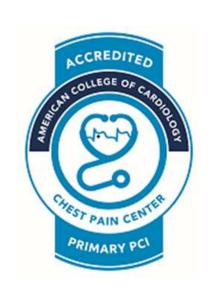
# Bayfront Health Seven Rivers





#### "The EMS 12 Lead - 101"

Wayne W Ruppert, CVT, CCCC, NREMT-P Interventional Cardiovascular Technologist Cardiovascular Coordinator Bayfront Health Seven Rivers

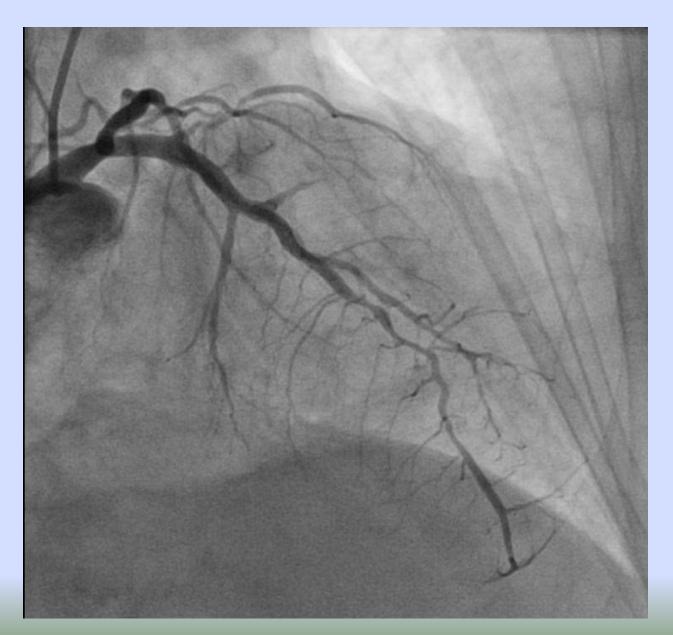


#### Welcome!





### Cardiac Cath Lab



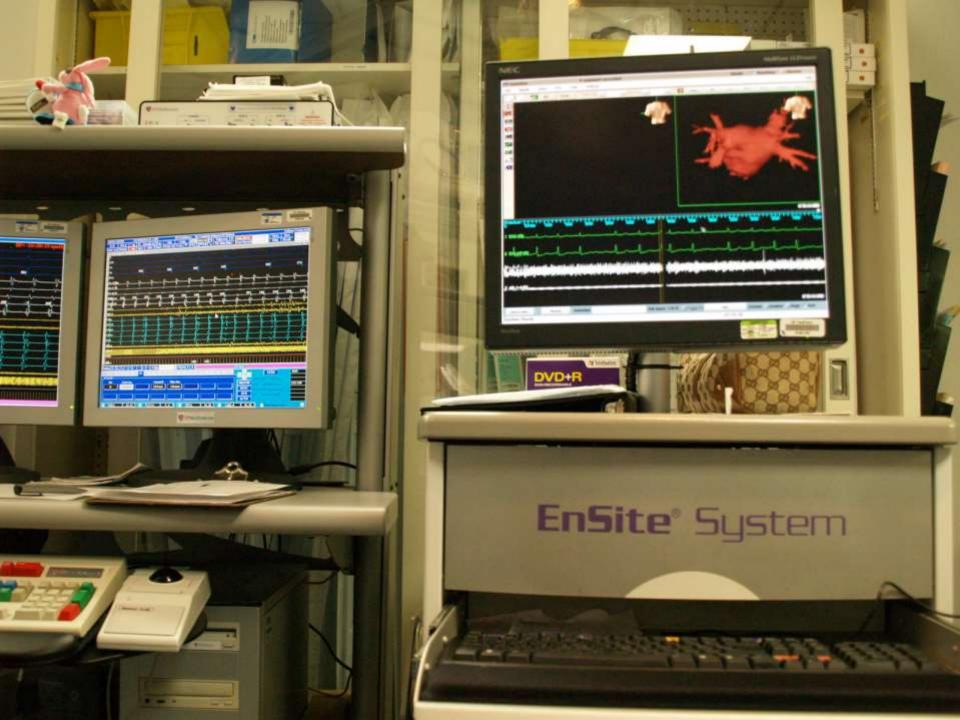
### **Electrophysiology Lab**





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



#### Wayne Ruppert bio:

- Cardiovascular Coordinator 2012-present (coordinated 5 successful accreditations)
- Interventional Cardiovascular / Electrophysiology Technologist, 1995-Present.
- Author of: "12 Lead ECG Interpretation in Acute Coronary Syndrome with Case Studies from the Cardiac Cath Lab," 2010, TriGen publishing / Ingram Books
- Author of: "STEMI Assistant," 2014, TriGen publishing / Ingram Books
- Florida Nursing CE Provider # 50-12998
- 12 Lead ECG Instructor, 1994-present (multiple hospitals, USF College of Medicine 1994)
- Website: www.ECGtraining.org

In the CARDIAC CATHETERIZATION LAB, we read our patients. 12 Lead ECGs and then evaluate their coonsary unterest and vertricular function during argiography. Stated in plan English, we republif learn how to correlate 12 least ECG findings with what's really gobig on inside our partents' hourts. 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.
- ASSMILATE 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 enherent LACK of SENSTITUTY 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 STEM, this knowledge propers you to ARTICIPATE the FALLURG 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 researce for every medical professional who evaluates adjusts and reads their 12 lead ECGs:

- Fellows in Energency, Cardiology, and Family Medicine
- Medical Residents
- Veteran Physicians weeting a good review in ACS patient evaluation
- . Physician Assistants and Murse Practitioners
- \* Energency Department Norses
- Coronary Care Unit and Cardiac Telepostry Warnes
- Wall-In Clinic Physicians and Marine
- Parametra

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

#### Joseph F. 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 hall 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 Follows. For experienced clinicisms, it's a superir review."

Humberto Coto, MD, FACP, FACC

- Chief of Interventional Cardiology

St. Joseph's Hospital Tampa, Florida



LEAD

CATH

LAB

THE CATH LAB SERIES presents. 2 LEAD ECG INTERPRETATION ACUTE CORONARY SYNDROME with CASE STUDIES from the CARDIAC CATHETERIZATION LAB WAYNE W RUPPERT

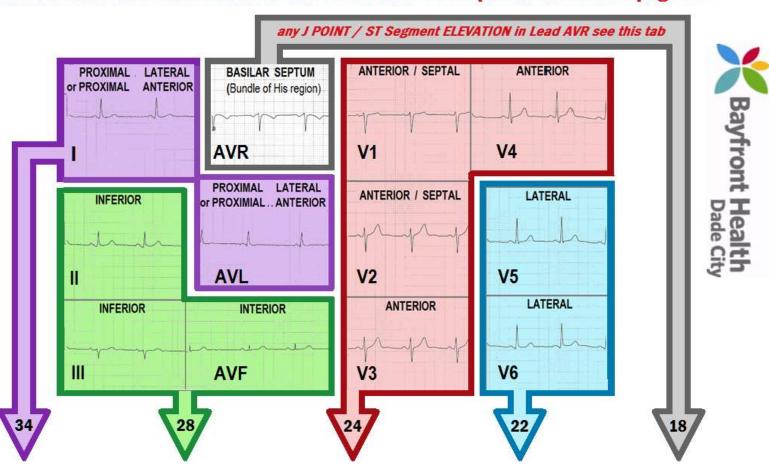
<u>www.TriGenPress.com</u> www.ECGtraining.org <u>BarnesandNoble.com</u> Amazon.com

# STEMI Assistant

by Wayne Ruppert

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

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



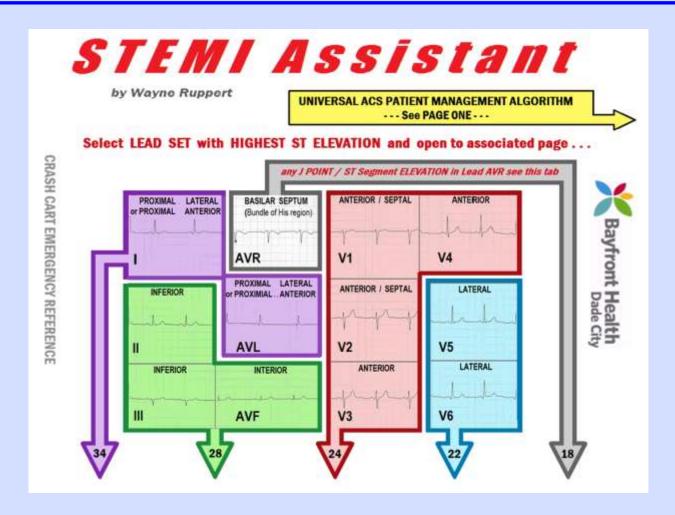
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**STEMI Assistant – Information Video** 

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Wayneruppert@aol.com



# American College of Cardiology 20<sup>th</sup> Congress 2017

Red Rock Resort, Las Vegas October 25 & 26, 2017

# Advanced Telemetry & 12 Lead ECG Workshop – Part 1

**Observation Medicine Tract** 

By: Wayne W Ruppert, CVT, CCCC, NREMT-P

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HOME

12 LEAD ECG IN ACS

STEMI ASSISTANT

ACCREDITATION

WORKSHOPS

ECG ID OF SADS

WORKSHOP OBJECTIVES

TEXTBOOKS

PHYSICIAN REVIEWS

BIO OF WAYNE RUPPERT

TESTIMONIALS

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**Download Basic ECG - Key West 2018** 

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**Download Advanced Physicians ACLS 2018 SRRMC** 

Download ACC 20th Congress Serial 12 Lead ECG Course

**Download A SHORT Course in LONG QT Syndrome** 

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Download ACC 20th Congress - Serial 12 Lead ECG Interpretation Part 1

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#### Single Lead ECG – vs – 12 Leads

- Single lead "rhythm strips" are obtained to see what rhythm the patient is currently in (e.g.: NSR, atrial fibrillation, heart blocks, etc).
- 12 Lead ECG is used to see if any part of the heart is not getting adequate blood supply (i.e.: ischemia, infarction). Plus, it aids in the diagnosis of many other conditions......

#### ....Other 12 Lead ECG Diagnoses:

- Bundle Branch Blocks\* and Fascicular Blocks
- Necrosis (old MI, evolving MI\*)
- V-Tach –vs– SVT with aberrancy
- Hypertrophy (enlargement of 1 or more chambers)
- Electrolyte imbalances
- Sudden Arrhythmia Death Syndromes:
  - Brugada, Long QT\*, ARVD, CPVT, HCM,
- Wolff-Parkinson-White Syndrome
- Acute Myocarditis / Pericarditis
- And many more

\* Covered in this course

#### EMS 12 Lead ECG



# OBTAINING THE 12 LEAD ECG

#### 10 wires . . .

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



#### THE ECG MACHINE

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

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

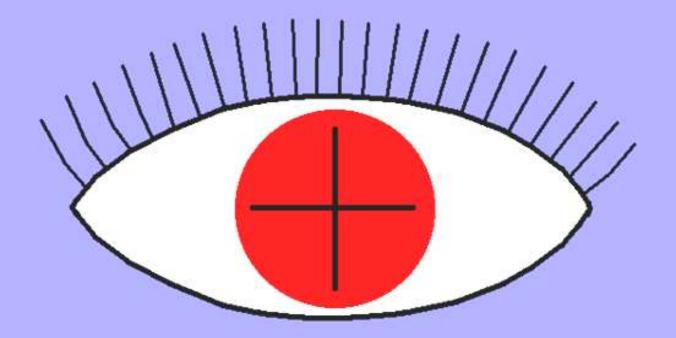


- LEADS AVR, AVL, and AVF
  - 1 POSITIVE ELECTRODE

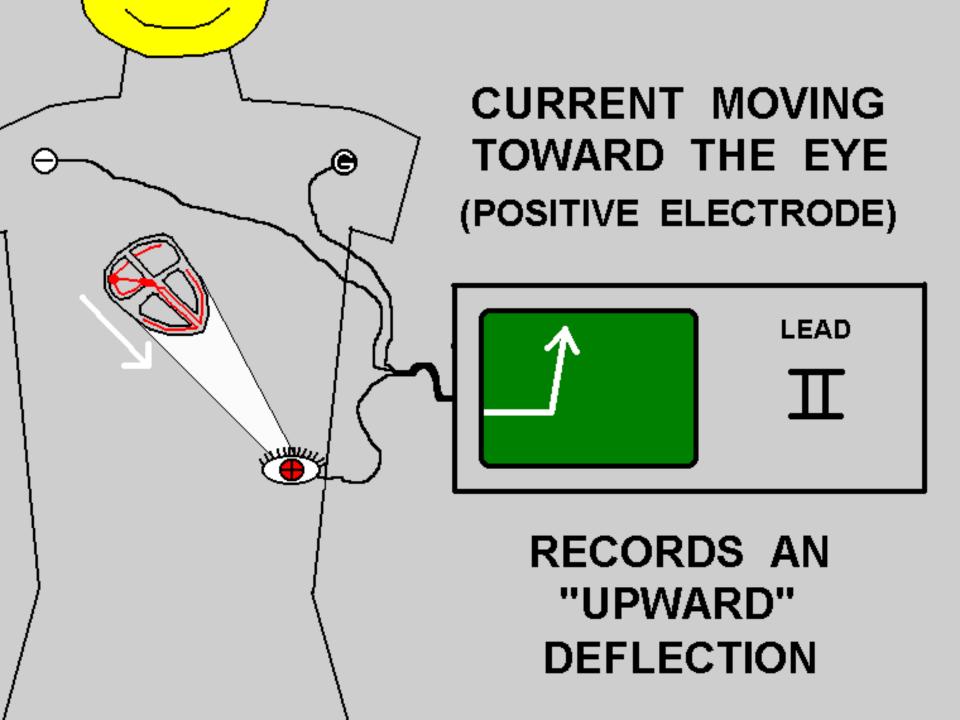
    2 NEGATIVE ELECTRODES

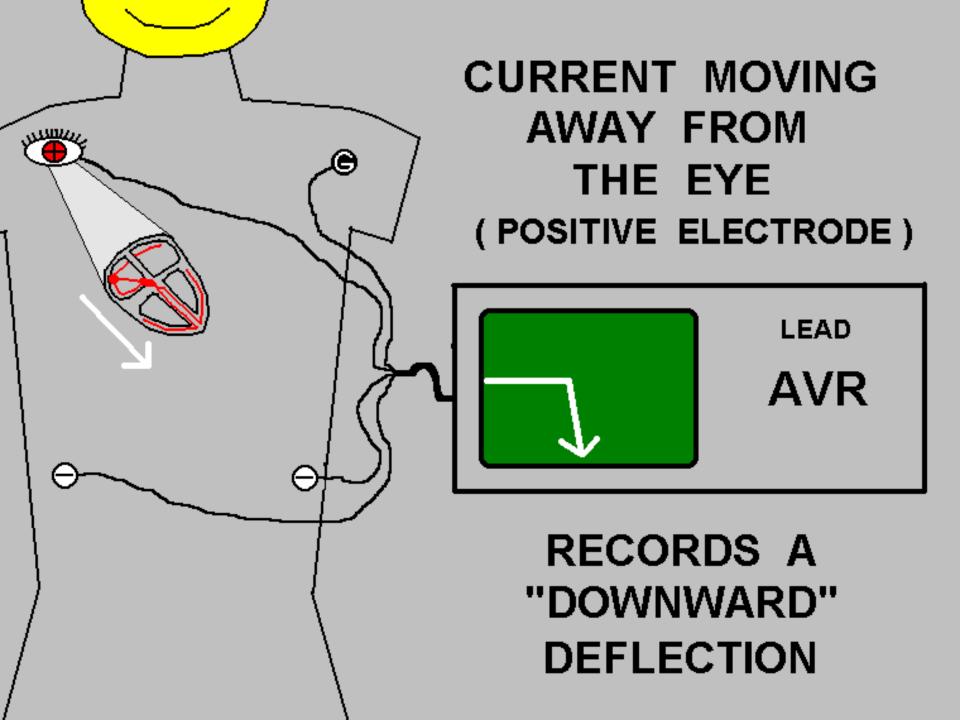
    1 GROUND ELECTRODE

#### THE POSITIVE ELECTRODE

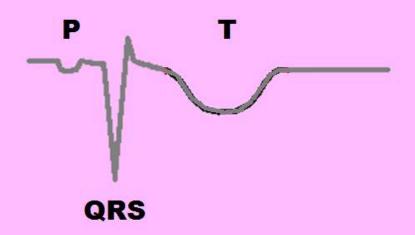


IS THE "EYE" . . .

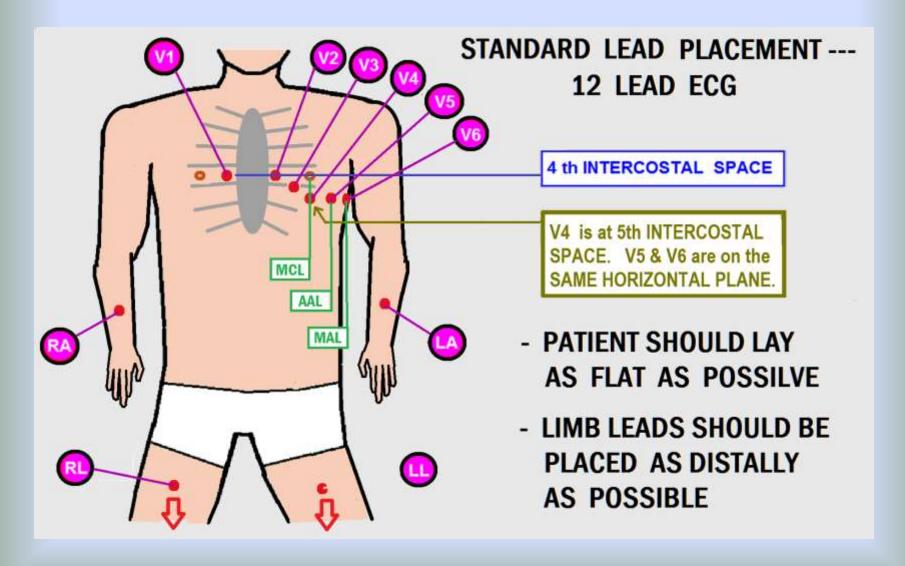




#### **LEAD AVR**



EVERYTHING
IS
"UPSIDE-DOWN"



Limb leads should be on the limbs.

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

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

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

#### Recommendations for the Standardization and Interpretation of the Electrocardiogram

Part I: The Electrocardiogram and Its Technology

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

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

# Recommendations for the Standardization and Interpretation of the Electrocardiogram

Part I: The Electrocardiogram and Its Technology

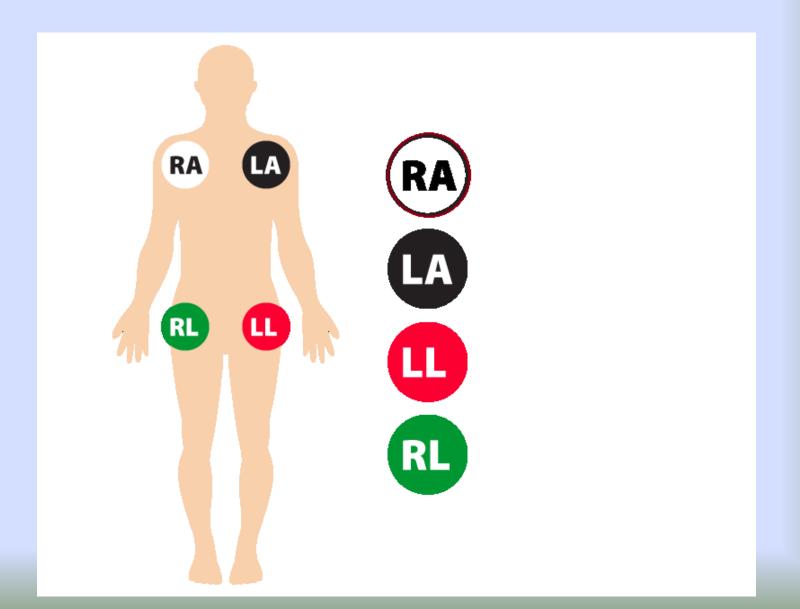
#### Recommendations

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

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

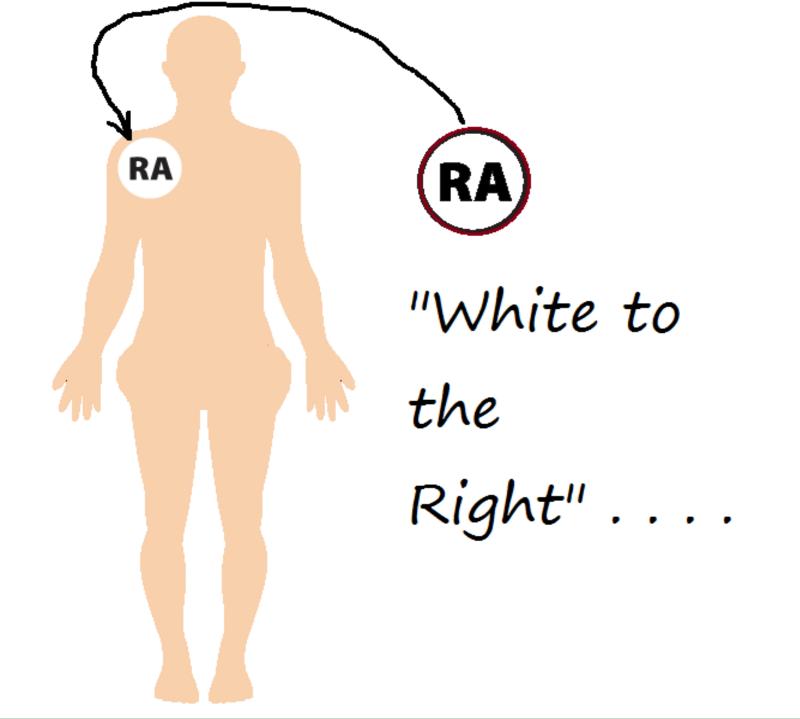
- Limb leads should be on the limbs.
- When emergency circumstances dictate that limb leads be placed on patient's torso, the words "LIMB LEADS ON PATIENT'S TORSO" should be noted on the ECG.
  - However in the field, it may not be possible or practical to put limb leads on limbs!

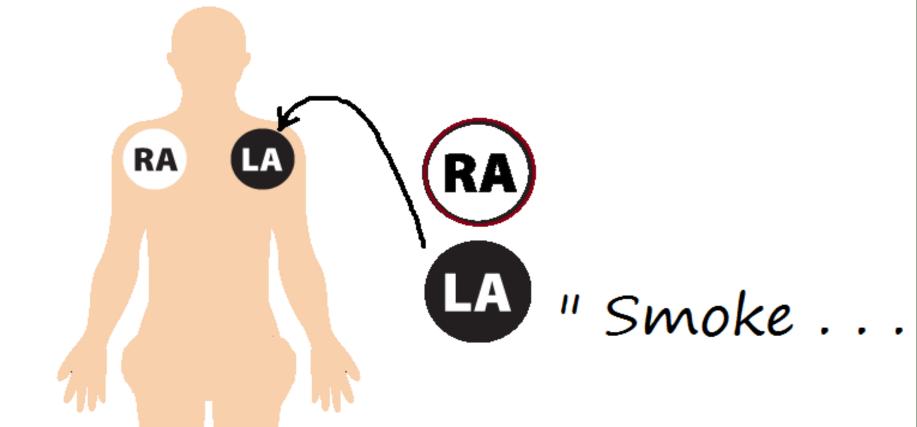
#### Limb leads – field ECG

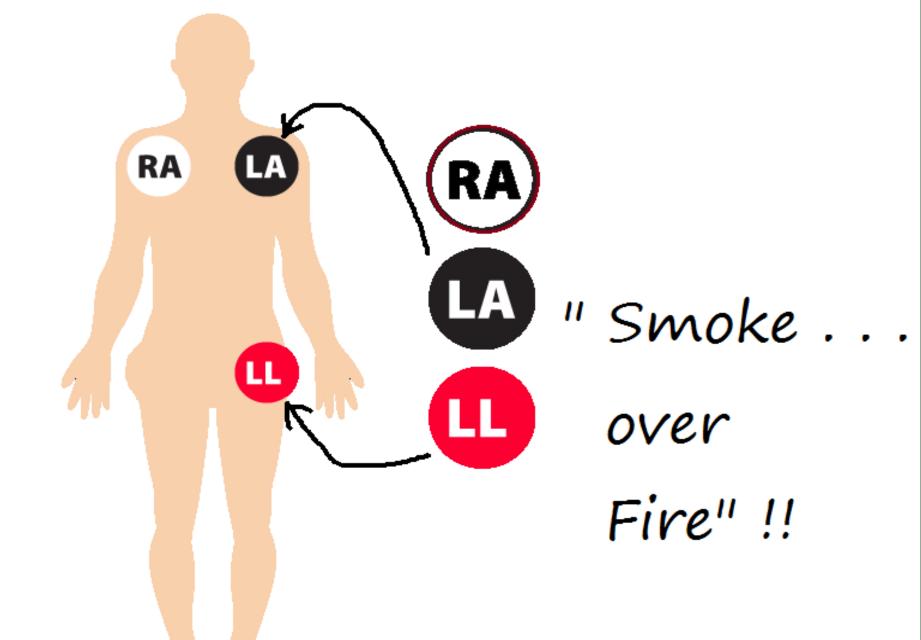


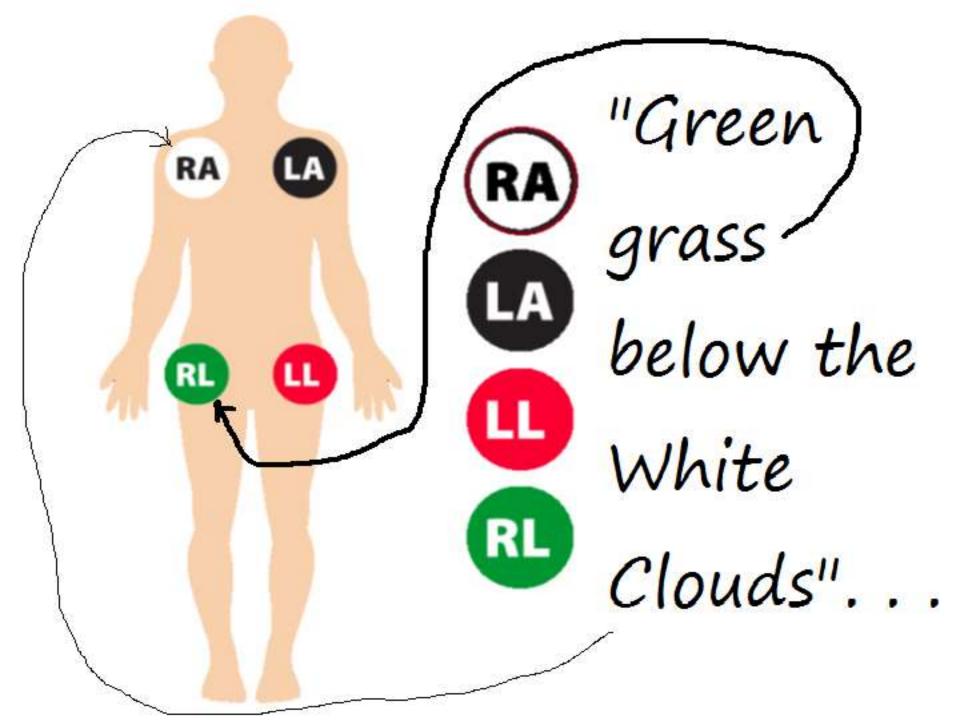
#### Remember this . . .

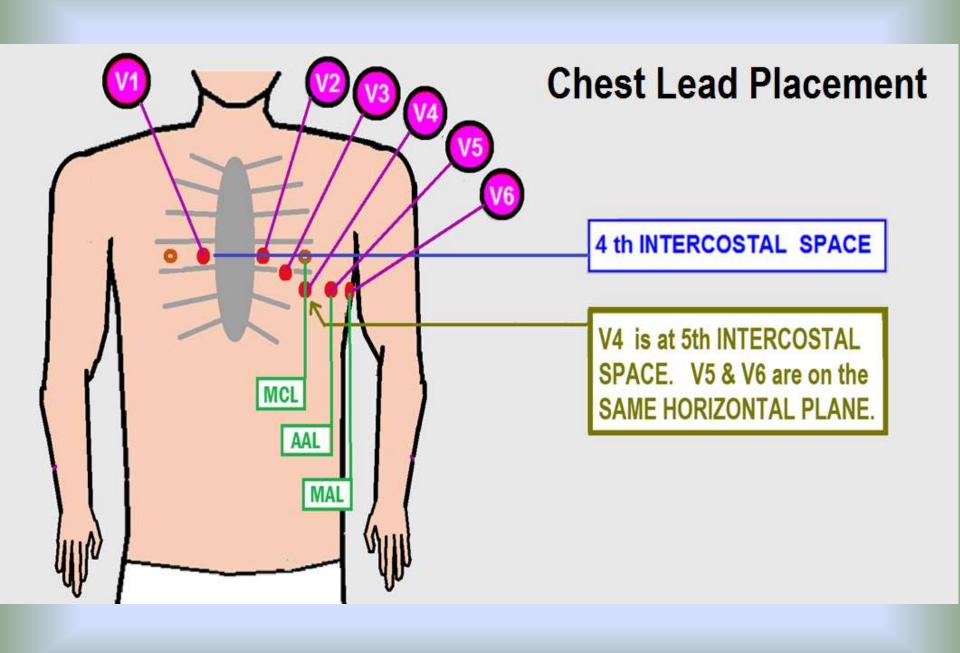
- White to the Right
- Smoke over Fire
- Green Grass below the White Clouds











#### 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.

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

#### Recommendations for the Standardization and Interpretation of the Electrocardiogram

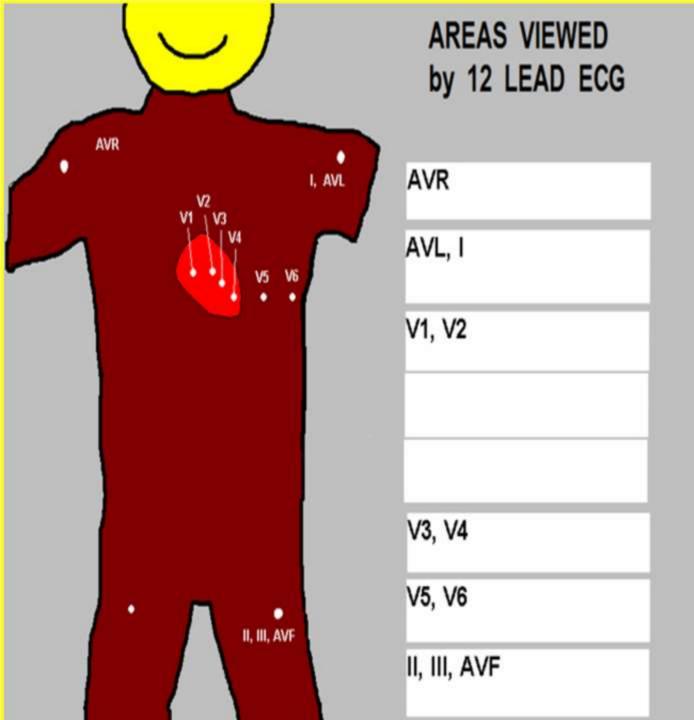
Part I: The Electrocardiogram and Its Technology

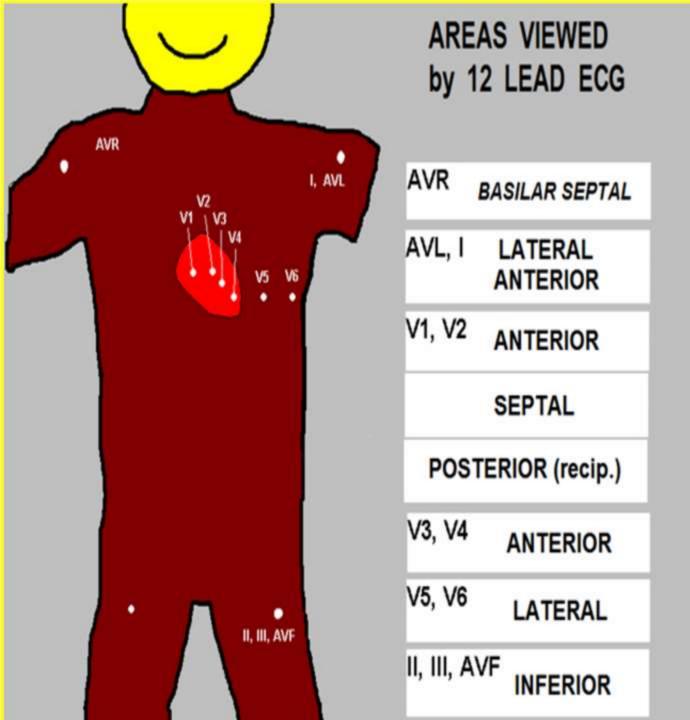
the often profound alterations in waveforms that can result from precordial electrode misplacement. 85,86 A common error is superior misplacement of  $V_1$  and  $V_2$  in the second or third intercostal space. This can result in reduction of initial R-wave amplitude in these leads, approximating 0.1 mV per interspace, which can cause poor R-wave progression or erroneous signs of anterior infarction.87 Superior displacement of the  $V_1$  and  $V_2$  electrodes will often result in rSr' complexes with T-wave inversion, resembling the complex in lead aVR. It also has been shown that in patients with low diaphragm position, as in obstructive pulmonary disease, 88,89

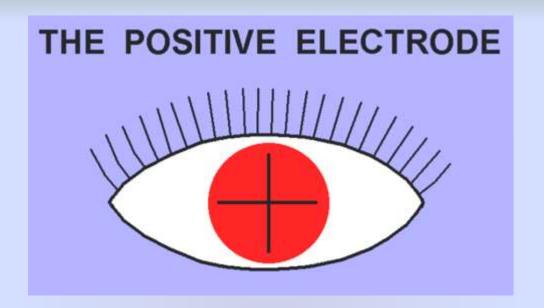
#### Obtaining the 12 Lead ECG, etc

- Patient should lay as flat as possible
- If you see too much "artifact" on the ECG, you may have to coach patient to "relax all of your muscles"...
  - Arms
  - Legs
  - Chest
  - Abdomen, etc.

To help you understand what part of the heart each lead of a 12 Lead ECG "sees" . . . .



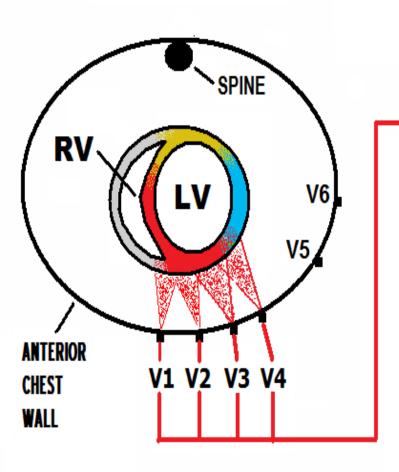




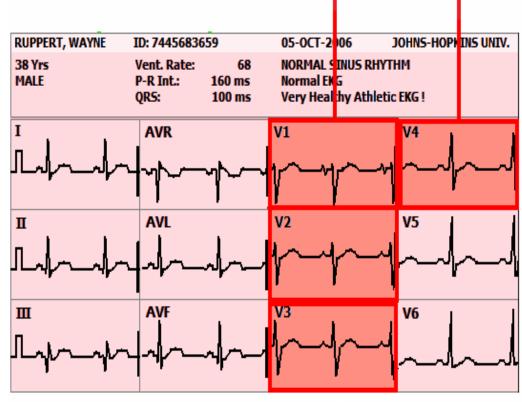
## What each of the 12 Leads "sees," in more detail . . . .

#### V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL

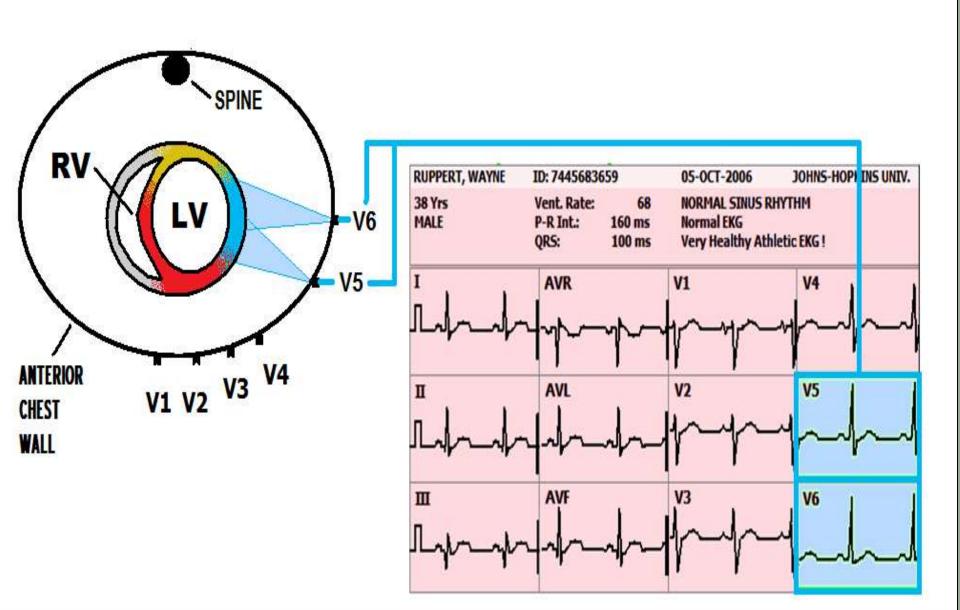
of the LEFT VENTRICLE



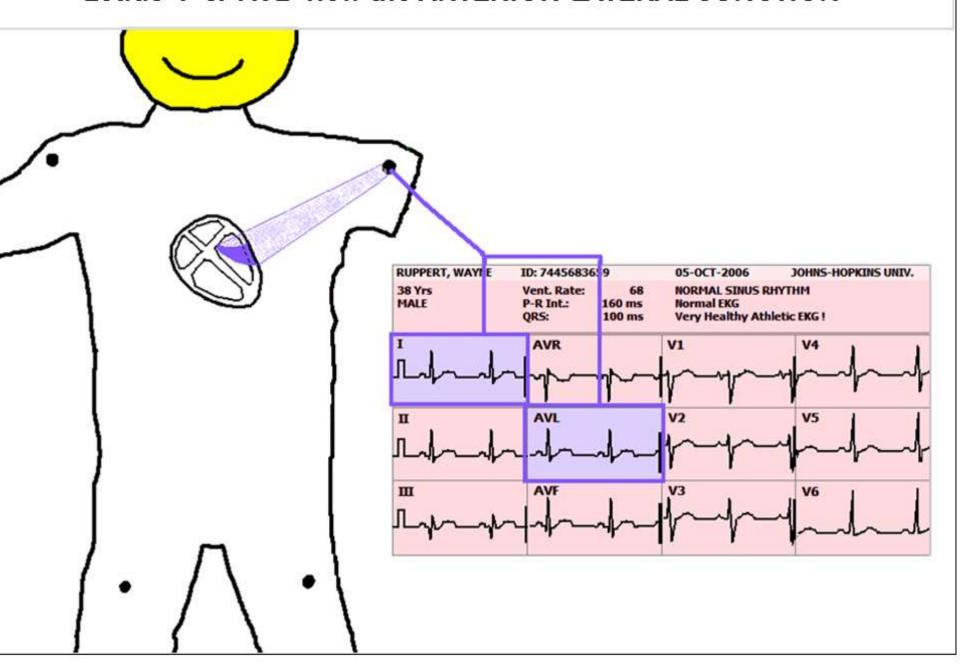
V1, V2 - ANTERIOR / SEPTAL V3, V4 - ANTERIOR



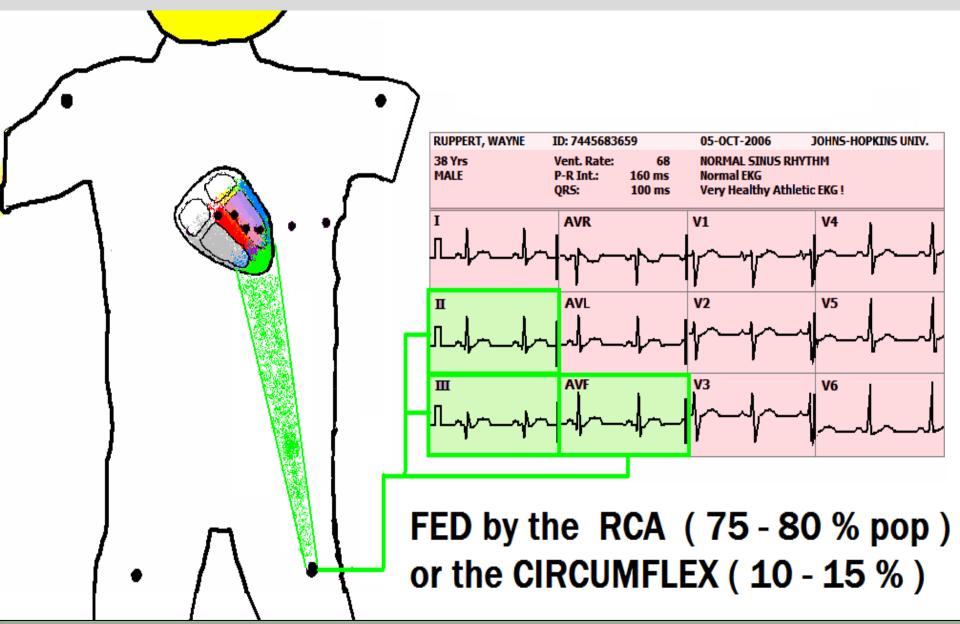
### V5 - V6 VIEW THE LATERAL WALL of the LEFT VENTRICLE



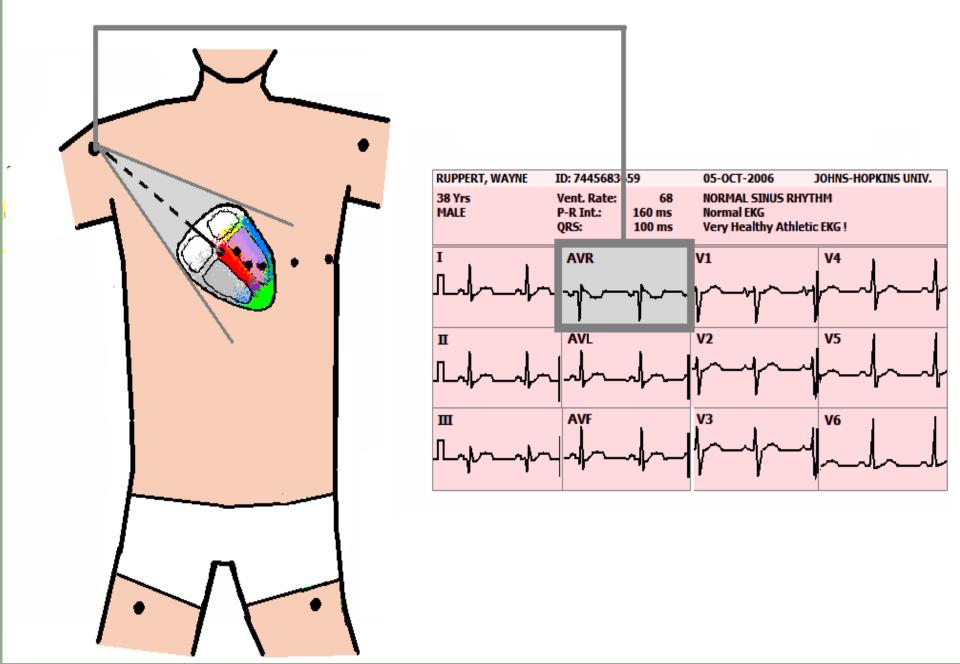
#### Leads I & AVL view the ANTERIOR-LATERAL JUNCTION

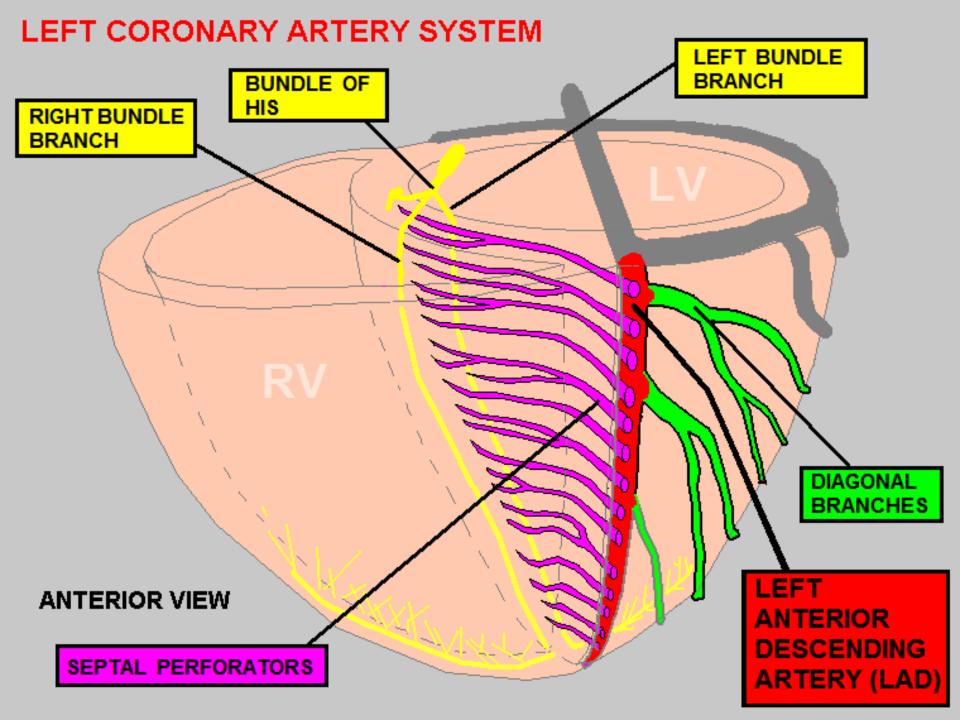


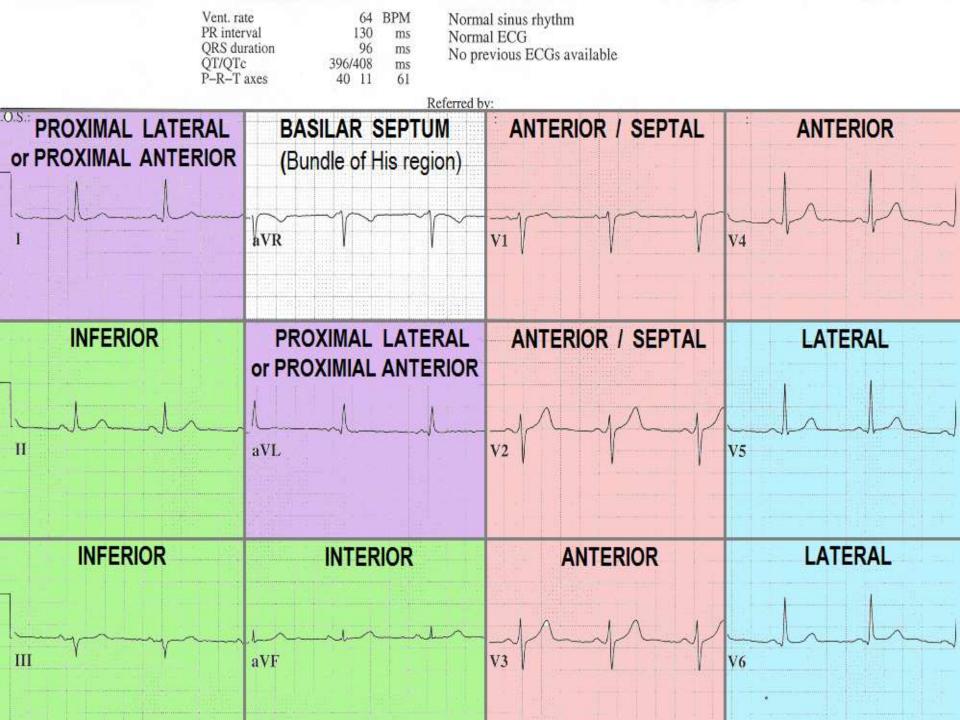
### LEADS II, III, and aVF VIEW INFERIOR WALL of the LEFT VENTRICLE



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





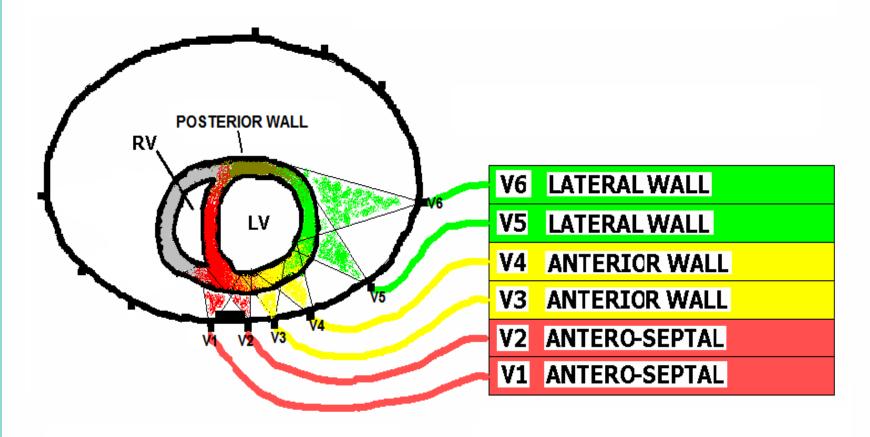


## A 12 Lead ECG sees MOST of the Heart, but it has some BLIND SPOTS!! . . . .

#### THE 12 LEAD ECG HAS TWO MAJOR BLIND SPOTS...

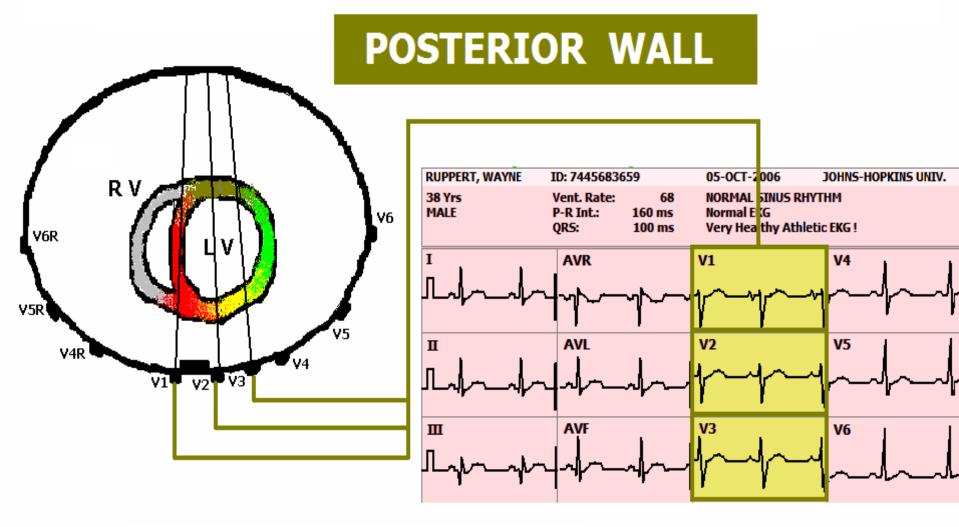
#### CHEST LEADS V1 - V6

WHAT EACH LEAD "SEES" . . .



Regarding the **POSTERIOR WALL**, the Standard 12 Lead will give you some "good clues" that there's a PROBLEM by displaying what we call "RECIPROCAL CHANGES"....kind of like seeing "reflected changes" in a mirror.....

#### **LEADS V1 - V3** view the

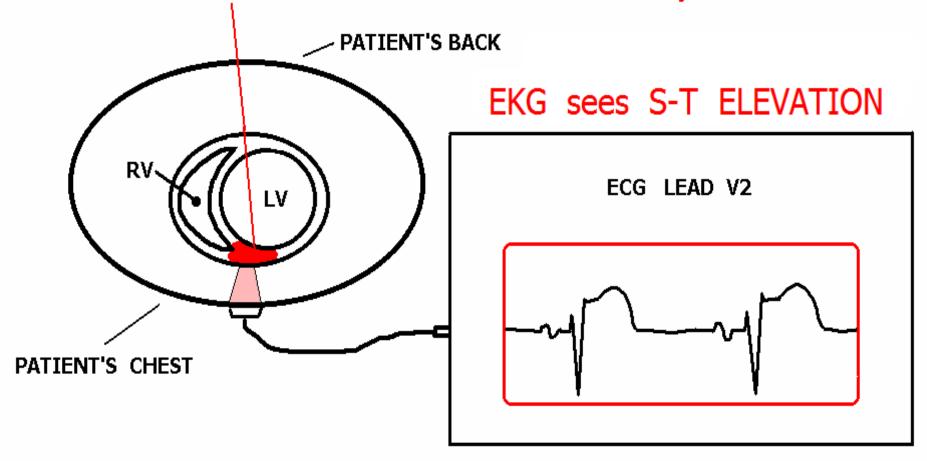


via RECIPROCAL CHANGES.

#### HOW EKG VIEWS INDICATIVE CHANGES

**EXAMPLE:** 

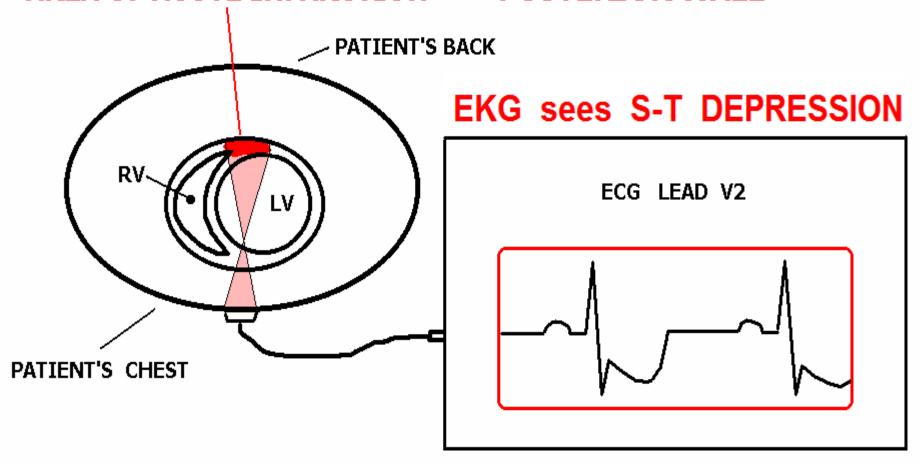
AREA OF ACUTE INFARCTION - ANTERIOR/SEPTAL



#### **HOW EKG VIEWS RECIPROCAL CHANGES**

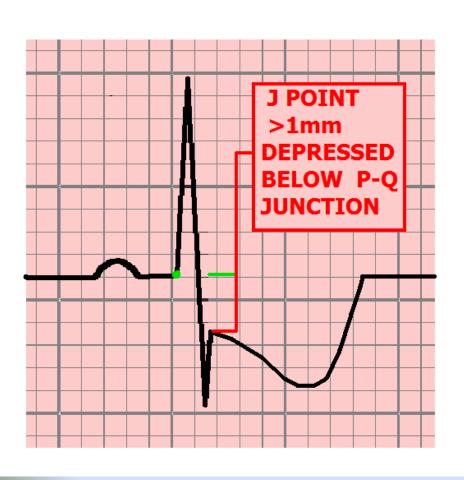
**EXAMPLE:** 

AREA OF ACUTE INFARCTION - POSTERIOR WALL



#### ST DEPRESSION can be from any other these conditions:

#### S-T SEGMENT DEPRESSION - COMMON ETIOLOGIES:



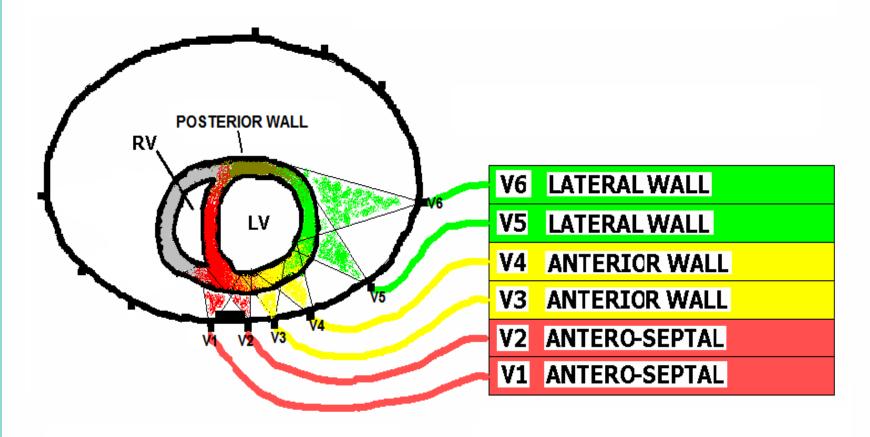
#### CONDITION:

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

#### THE 12 LEAD ECG HAS TWO MAJOR BLIND SPOTS...

#### CHEST LEADS V1 - V6

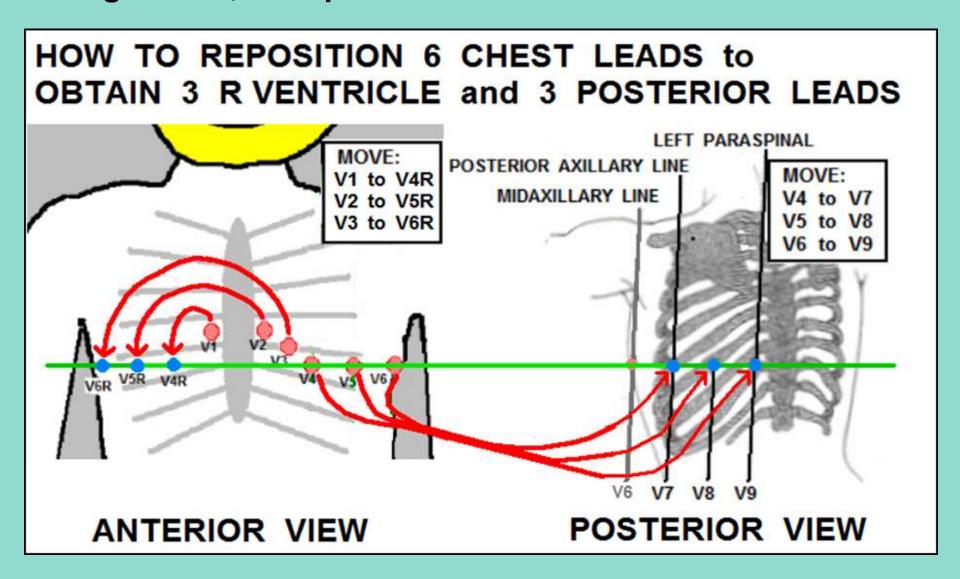
WHAT EACH LEAD "SEES" . . .



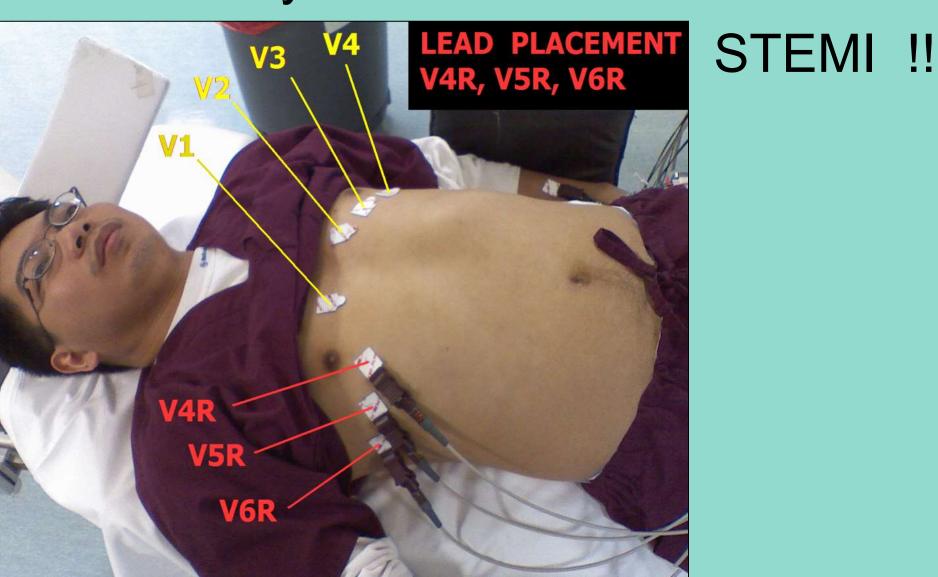
#### THE 18 LEAD ECG COVERS THE ENTIRE HEART...

CHEST LEADS V1 - V6 PLUS V4R, V5R, V6R, and V7, V8, V9 WHAT EACH LEAD "SEES" . . . POSTERIOR WALL POSTERIOR WALL POSTERIOR WALL RV V6 LATERAL WALL V6R LV V5 LATERAL WALL V4 ANTERIOR WALL V3 ANTERIOR WALL V4R V2 ANTERO-SEPTAL V1 ANTERO-SEPTAL V4R RIGHT VENTRICLE V5R RIGHT VENTRICLE V6R RIGHT VENTRICLE

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

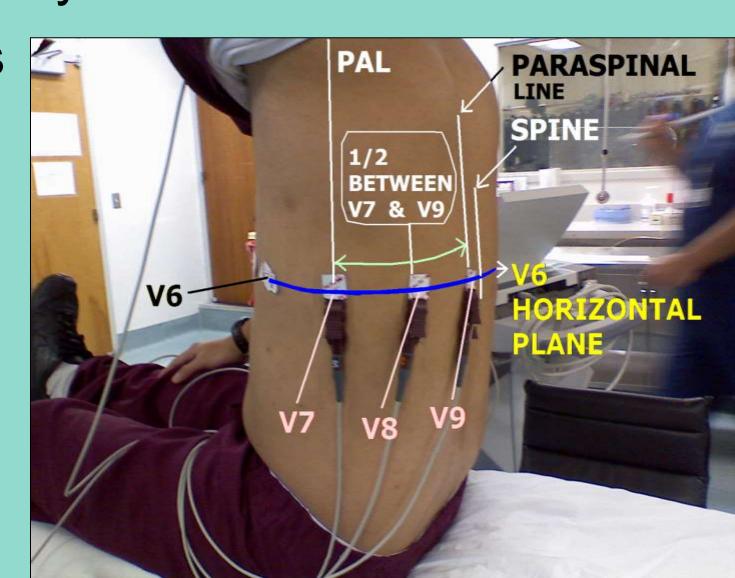


#### RIGHT SIDED ECG is indicated whenever you see INFERIOR WALL

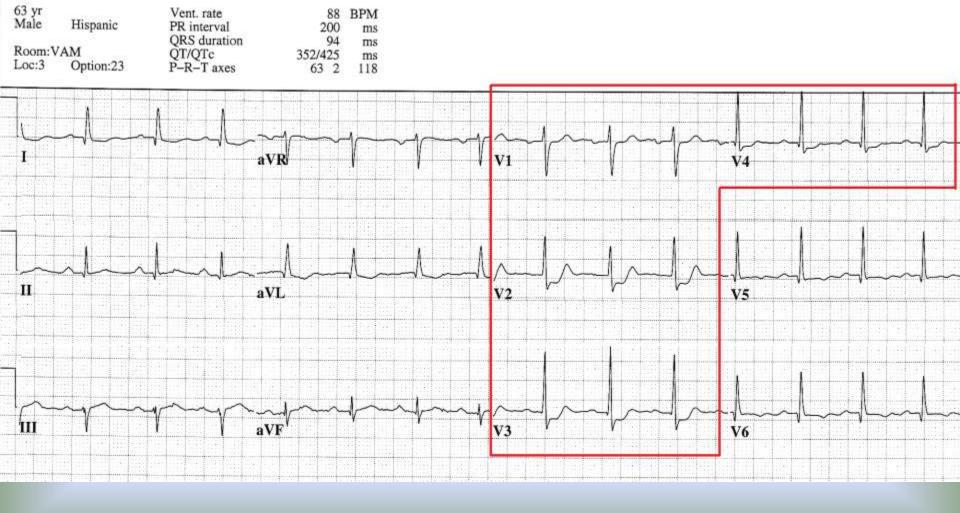


### POSTERIOR ECG is indicated whenever you see ST DEPRESSION

In Leads V1-V4



#### ST Depression in V1 – V4 . . . .



## INDICATIONS for 18 Lead ECG include:

- INFERIOR WALL MI
  - Right sided ECG
- ST Depression V1-V4
  - Posterior ECG

## Practical application of 18 Lead ECGs is presented in the CASE STUDIES section of this curriculum.

Before we go any farther, you should know . . .

# Sometimes, ECGs LIE to us!

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



#### THE ECG in PERSPECTIVE:

**PROBLEMS** with ECG:

- ↓ SENSITIVITY

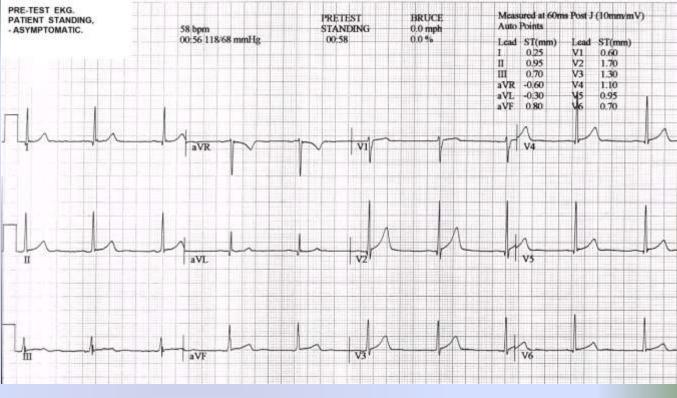
  ( FALSE NEGATIVES )
- ↓ SPECIFICITY

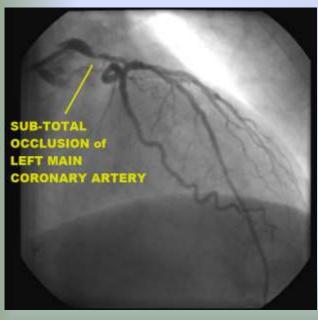
  ( FALSE POSITIVES )

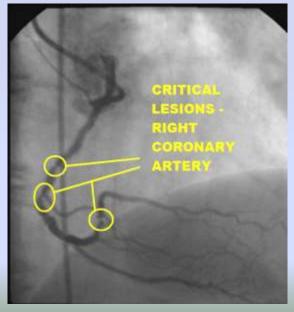
## PROBLEMS WITH SENSITIVITY . . .

## NORMAL ECG.

But . . . . .



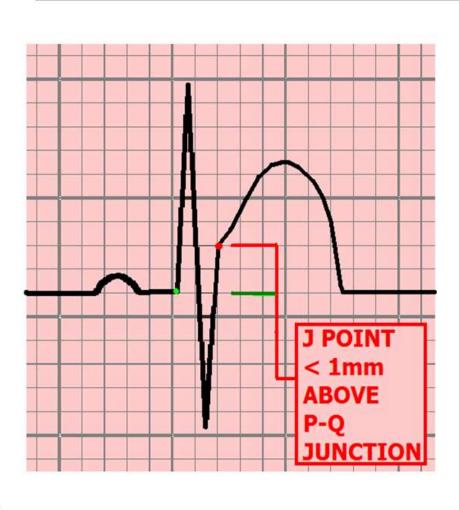




LETHAL
TRIPLE
VESSEL
DISEASE

#### PROBLEMS WITH SPECIFICITY . . .

#### S-T SEGMENT ELEVATION - COMMON ETIOLOGIES:

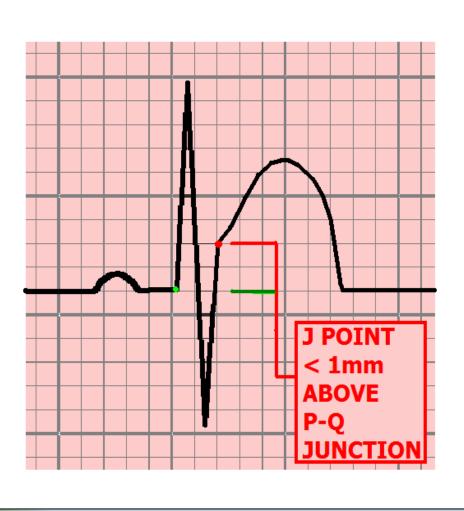


#### CONDITION:

ACUTE INFARCTION (STEMI)

#### PROBLEMS WITH SPECIFICITY . . .

#### S-T SEGMENT ELEVATION - COMMON ETIOLOGIES:



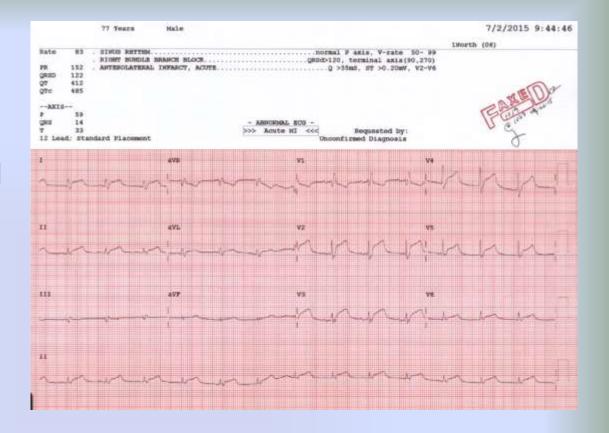
#### CONDITION:

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

#### **Patient:**

- Asymptomatic
- Troponin normal
- Cardiac Cath

   angiography =
   no obstructive
   CAD."
- Discharge diagnosis:



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

Despite the ECG's problematic issues with Lack of Sensitivity &

Lack of Specificity,

The 12 Lead ECG remains

one of our QUICKEST, most costefficient front-line Triage Tools
that we have today.

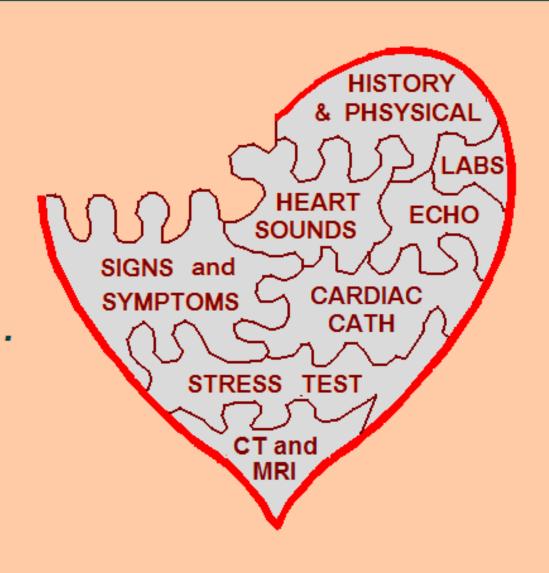


So how do we know when the ECG is telling us the truth ???

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



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



 In the hospital we utilize ACS Risk **Stratification tools** - such as The HEART Score. We also have Troponins, Echo, CT and Cardiac Cath. In the field, you have far fewer resources!!



- What do you have in the field?
  - Symptoms
  - Risk Factors
  - ECG
  - Physical Exam



### The QUADRAD of ACS

PRESENTING SYMPTOMS
RISK FACTOR PROFILE
ECG ABNORMALITIES
CARDIAC MARKERS

A <u>POSITIVE</u> finding in <u>TWO</u> or MORE of the above categories indicates it is <u>EXTREMELY</u> <u>LIKELY</u> that <u>ACS</u> is <u>present</u>... steps must be AGGRESSIVELY TAKEN to definitively RULE OUT the PRESENCE of ACS!

# You arrive at a patient's residence. The scene is safe, and you're led into the living room by the patient's daughter . . .

The instant you see the patient, you assess for:

- CAB (pulse, breathing) the patient is awake, to you know he has a pulse and you see him breathing
- 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

NORMAL

STATUS: 6 SHOCK 6

## FAIL the SHOCK SURVEY?

RAPIDLY FIND AND TREAT
THE ROOT CAUSE...

#### PHASE 1: **RULE OUT LIFE-THREATENING CONDITIONS** • ABCs SHOCK ASSESSMENT CONSCIOUS, WITH CONSCIOUS, NO UNCONSCIOUS SIGNS OF SHOCK SIGNS OF SHOCK RULE OUT ASSESS VITAL ABCS SIGNS & O2 SAT CAUSES OF SHOCK: ECG MONITOR FAIL PASS - INSULIN TREAT - CARDIOGENIC SYMPTOMATIC - HYPOVOLEMIC DYSRHYTHMIAS - METABOLIC RESUSCITATE as per ACLS, or - NEUROGENIC PATIENT as per INSTITUTIONAL - SEPTIC ACLS, or - RESPIRATORY **PROTOCOLS** INSTITUTIONAL - PULMONARY START IV & PROTOCOLS **EMBOLUS** DRAW LABS - DRUGS / MEDS PROVIDE APPROPRIATE TX

### The QUADRAD of ACS

$\square$	PRESENTING SYMPTOMS
	RISK FACTOR PROFILE
	ECG ABNORMALITIES
	CARDIAC MARKERS

A <u>POSITIVE</u> finding in <u>TWO</u> or MORE of the above categories indicates it is <u>EXTREMELY</u> <u>LIKELY</u> that <u>ACS</u> is <u>present</u>... steps must be AGGRESSIVELY TAKEN to definitively RULE OUT the PRESENCE of ACS!

#### CHIEF COMPLAINT

#### **KEY WORDS:**

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

**SHORTNESS BREATH** 

**DIZZINESS / LIGHTHEADEDNESS** 

ETC. ETC. ETC.



## TYPICAL SYPTOMS of ACUTE CORNARY SYNDROME:

- $\checkmark$
- CHEST PAIN DESCRIBED AS ...
- "HEAVINESS, PRESSURE, DULL PAIN, TIGHTNESS"
- CENTERED IN CHEST, SUBSTERNAL
- MAY RADIATE TO SHOULDERS, JAW, NECK, LEFT or RIGHT ARM
- NOT EFFECTED by:
  - MOVEMENT
  - POSITION
  - DEEP INSPIRATION
- $\checkmark$ 
  - SHORTNESS OF BREATH
    - MAY or MAY NOT BE PRESENT
- **√** 
  - NAUSEA / VOMITING
    - MAY or MAY NOT BE PRESENT

## INFARCTION

- - - "Classic Symptoms" - - -



QUICK ASSESSMENT "SHORT FORM"

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

#### stable angina

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



#### unstable angina

- 1. SYMPTOMS MAY START AT ANY TIME, EVEN DURING REST
- 2. SYMPTOMS ARE <u>NEW</u>, <u>DIFFERENT</u>, or <u>WORSE</u> THAN PREVIOUS EPISODES

BEWARE of the patient with "INTERMITTENT CHEST PAIN"....



## ATYPICAL SYMPTOMS of ACS

???

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

Stroke (previous history of)

Heart failure (previous history of)

Race (non-white)

Elderly (age 75+)

Women

**D**iabetes 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) Fatigue

Indigestion Abdominal pain

Nausea Cold sweats

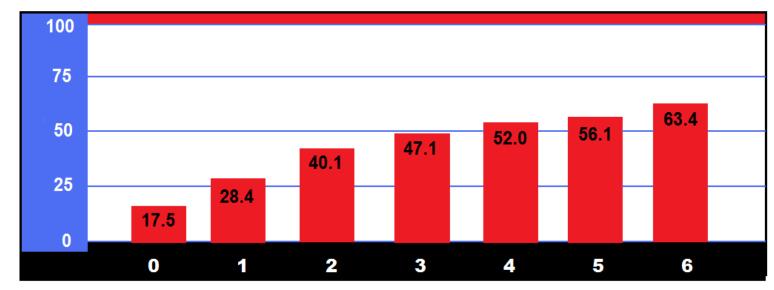
Dizziness Elevated heart rate

Syncope Dsypnea

**BOOK PAGE: 70** 

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

% of
PATIENTS with
ACUTE MI
PRESENTING
TO THE
EMERGENCY
DEPARTMENT
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

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

<ul> <li>UNUSUAL FATIGUE</li> </ul>	71 %
<ul> <li>SLEEP DISTURBANCE</li> </ul>	48 %
• SOB	42 %
<ul> <li>INDIGESTION</li> </ul>	39 %
<ul><li>ANXIETY</li></ul>	36 %

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

#### WOMEN'S MAJOR SYMPTOMS **DURING THEIR HEART ATTACK:**

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



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

Circulation, 2003:108;2619-2623

#### Physical Exam – Clues of MI:

- Skin may be PALE, CLAMMY
- SWEATING! (Diaphoresis)
- Clutching /Rubbing chest
- BP can be high, normal or low
- Anxiety / "look of impending doom."

### The QUADRAD of ACS



A <u>POSITIVE</u> finding in <u>TWO</u> or MORE of the above categories indicates it is <u>EXTREMELY</u> <u>LIKELY</u> that <u>ACS</u> is <u>present</u>.... steps must be AGGRESSIVELY TAKEN to definitively RULE OUT the PRESENCE of ACS!

#### The QUADRAD of ACS

PRESENTING SYMPTOMS
RISK FACTOR PROFILE
CG ABNORMALITIES
CARDIAC MARKERS

A <u>POSITIVE</u> finding in <u>TWO</u> or MORE of the above categories indicates it is <u>EXTREMELY</u> <u>LIKELY</u> that <u>ACS</u> is <u>present</u>.... steps must be AGGRESSIVELY TAKEN to definitively RULE OUT the PRESENCE of ACS!

## **RISK FACTORS**

for the development of

#### CORONARY ARTERY DISEASE:

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



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

#### \*Risk factors for atherosclerotic disease:

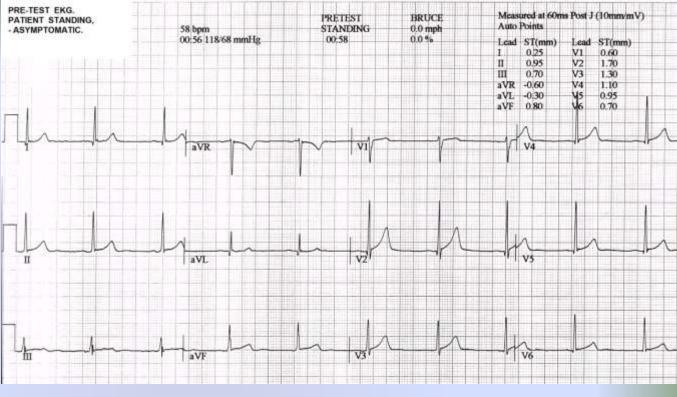
Hypercholesterolemia Hypertension Diabetes Mellitus Cigarette smoking Positive family history Obesity

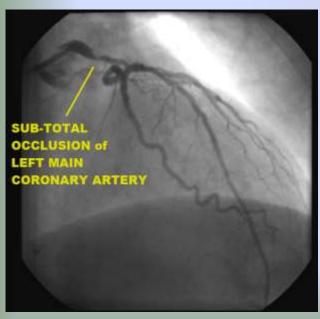
Here is the HEART Score! Let's see what the score is for our patient with the NORMAL ECG ....who had **Critical Triple** Vessel Disease and needed STAT Bypass Surgery! ....

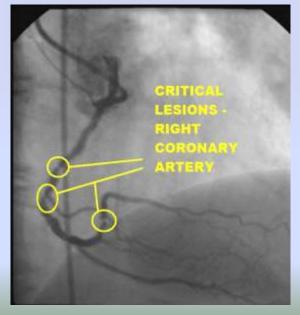
## PROBLEMS WITH SENSITIVITY . . .

## NORMAL ECG.

But . . . . .







LETHAL
TRIPLE
VESSEL
DISEASE



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

\*Risk factors for atherosclerotic disease:

Hypercholesterolemia Hypertension Diabetes Mellitus

Cigarette smoking Positive family history Obesity  $\mathbf{H}$  = chest pain = 2

 $\mathbf{E} = \mathbf{ECG} \text{ normal} = \mathbf{0}$ 

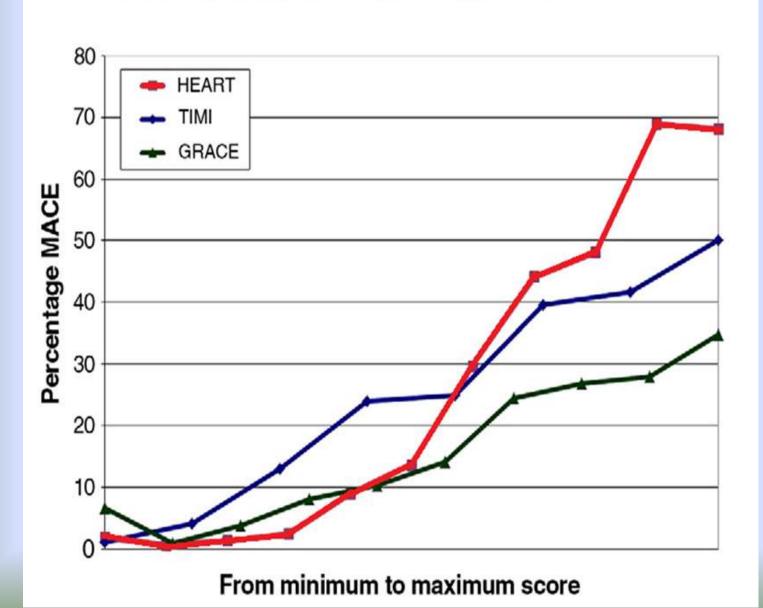
A = 63 = 1

 $\mathbf{R} = 3 \text{ risk factors} = 2$ 

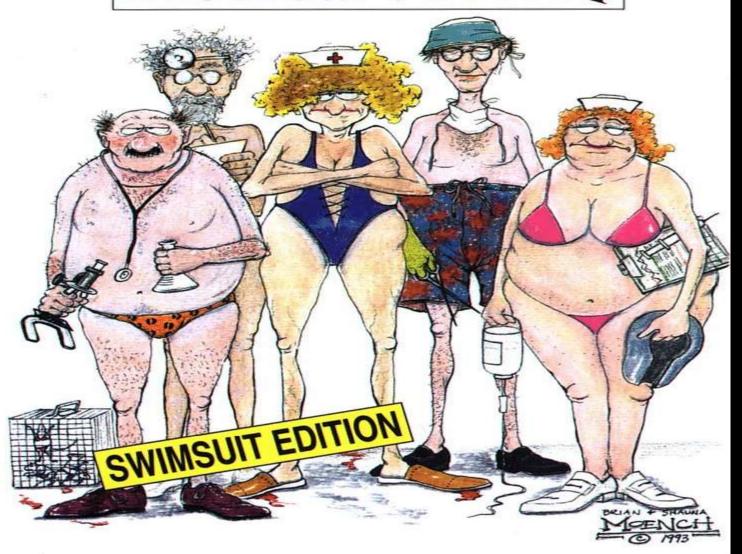
T = Trop. NL = 0

**HEART Score:** = 5

## Comparison of PREDICTIVE RELIABILITY of the HEART SCORE to the Modified TIMI and GRACE ACS Risk Scores:



# The New England Medical Journa/



#### Some Basic Vocabulary:

- Ischemia = Inadequate blood supply to cells, but cells are still getting blood. Cellular Oxygen Demand is HIGHER than the Oxygen Supply.
- Infarction = blood supply to cells has been cut off. Cells are no longer receiving oxygen or glucose. Cells survive by consuming available glycogen reserves, convert to ANAEROBIC metabolism. Unless blood supply is restored, cells die when glycogen reserves are depleted.

#### Some Basic Vocabulary:

- Acute Coronary Syndrome (ACS) is made up of the following cardiac conditions:
  - Unstable Angina
  - Non-ST Segment Elevation Myocardial Infarction (NSTEMI)
  - ST Segment Elevation Myocardial Infarction (STEMI)
- Low Risk Chest Pain

#### **Unstable Angina**

#### stable angina

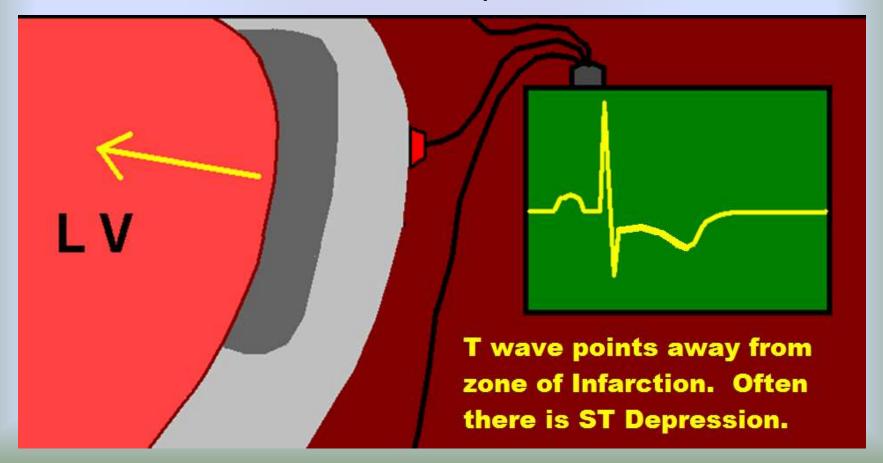
- SYMPTOMS START DURING PHYSICAL EXERTION.
- 2. SYMPTOMS ARE "PREDICTABLE"

VS.

#### unstable angina

- 1. SYMPTOMS MAY START AT ANY TIME, EVEN DURING REST
- 2. SYMPTOMS ARE <u>NEW</u>, <u>DIFFERENT</u>, or <u>WORSE</u> THAN PREVIOUS EPISODES

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



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

The 12 Lead ECG may show:

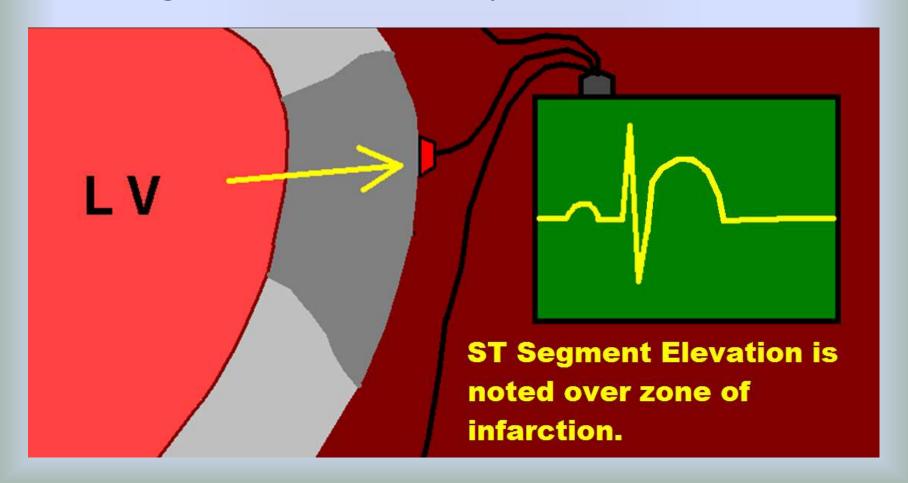
- ST Depression
- Other ST Segment changes
- Inverted T wave
- THE ECG MAY BE TOTALLY NORMAL.

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

This is a "Partial Wall Thickness" MI, heart cells are dying, and the Troponin becomes detectable in the patient's bloodstream. Usually "less severe" than a STEMI, patient needs blood thinners and to get to the cath lab in 24-48 hours.

#### **STEMI**

ST Segment Elevation Myocardial Infarction.



ST Segment Elevation Myocardial Infarction. ("full-wall thickness," Transmural event)

This is a life-threatening emergency. Part of the patient's heart is dying. Blood flow must be restored within 90 minutes or less in order to preserve heart muscle. Based on the region of the heart affected, critical and often lethal complications may rapidly develop.

# A quick review of some very basic ECG concepts:

- When you turn on the ECG machine, it defaults to normal values.....
  - Records at 25mm/ second (horizontal axis)
  - Amplitude (vertical axis) is set so "1 mv = 10 small (1mm) boxes."

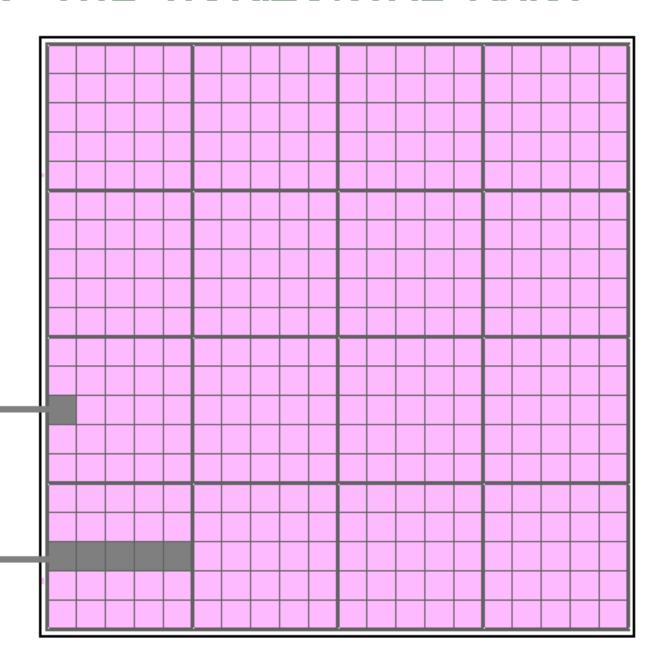
#### **ECG PAPER - THE HORIZONTAL AXIS:**

THE HORIZONTAL AXIS REPRESENTS TIME...

STANDARD SPEED FOR RECORDING ADULT EKGs = 25 mm / SECOND

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

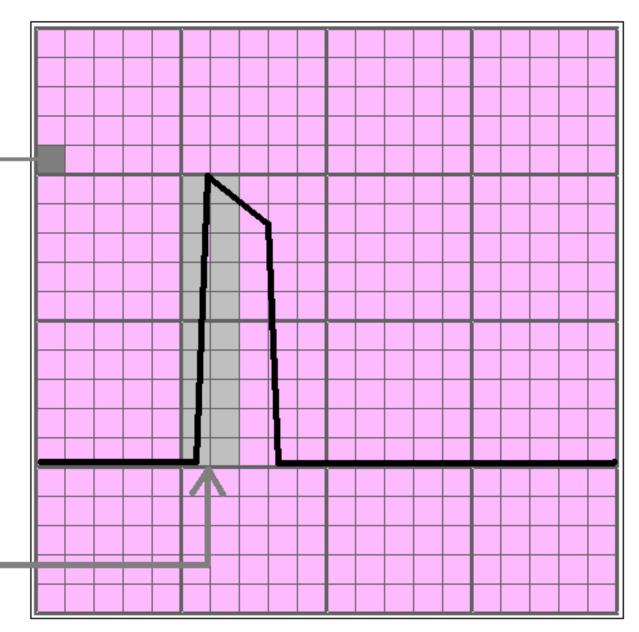
5 SMALL BOXES = .20 SECONDS, or 200 MILLISECONDS (200 ms)

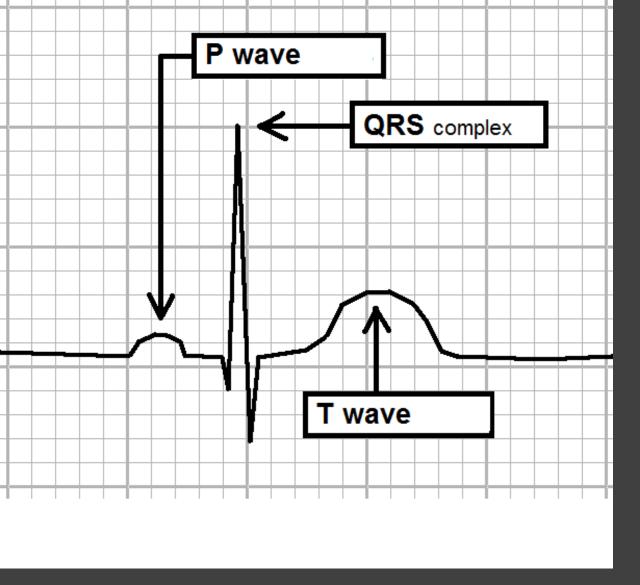


#### **ECG PAPER - THE VERTICAL AXIS:**



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





P WAVE =
ATRIAL
DEPOLARIZATION

QRS COMPLEX =
VENTRICULAR
DEPOLARIZATION
(contracting)

T WAVE =
VENTRICULAR
REPOLARIZATION
(recharging)

Q: To evaluate the patient for ischemia or infarction, what part of the ECG do we look at?

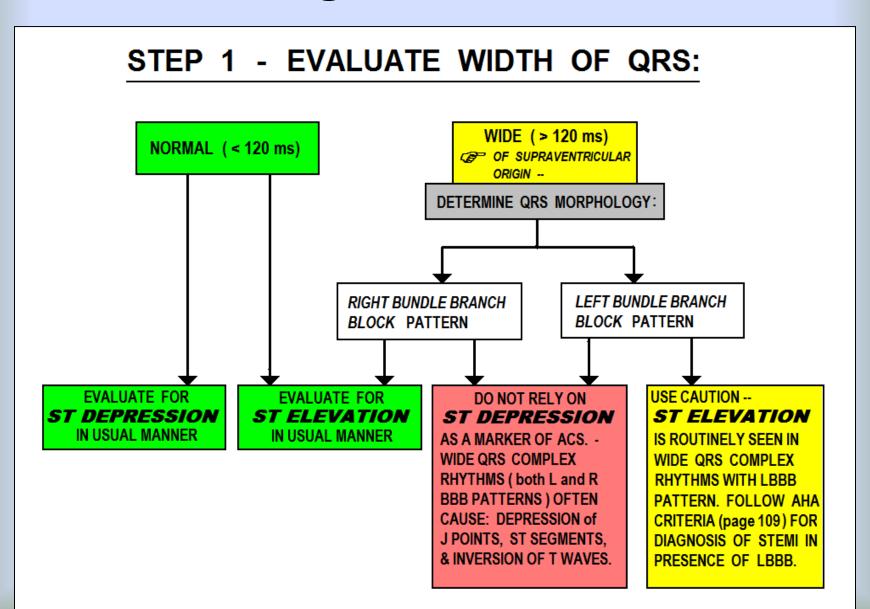
Q: To evaluate the patient for ischemia or infarction, what part of the ECG do we look at?

#### A: We evaluate the

- J Points
- ST Segments &
- T Waves

.....in each lead!

#### 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

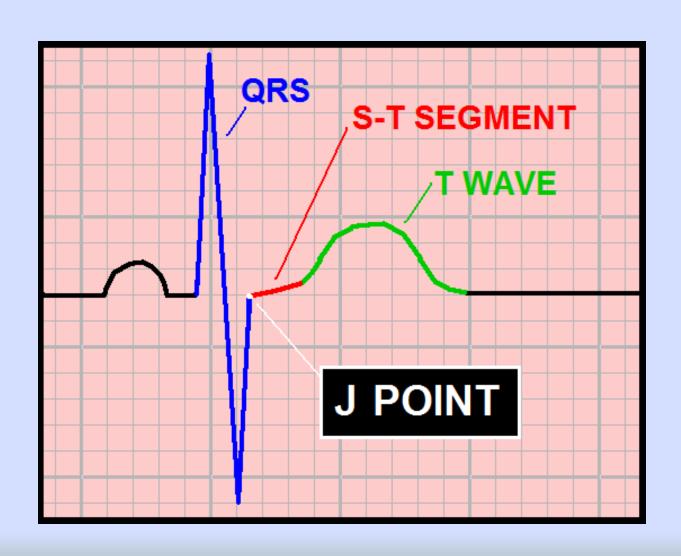
CAREFULLY SCRUTINIZE THESE MARKERS IN EVERY LEAD OF THE 12 LEAD EKG, TO DETERMINE IF THEY ARE NORMAL or ABNORMAL.

Q: Why is QRS width an issue when we look at J Points, ST Segments and T Waves??

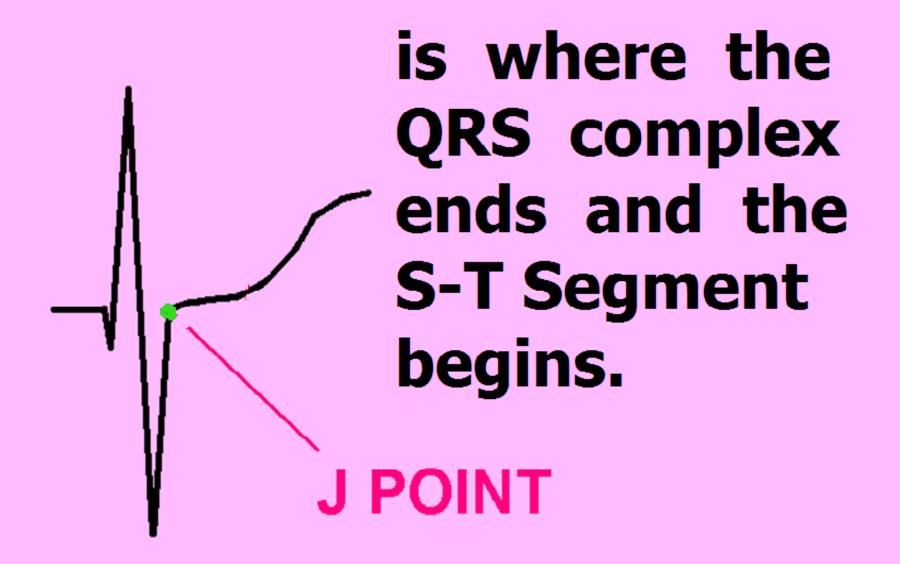
Q: Why is QRS width an issue when we look at J Points, ST Segments and T Waves??

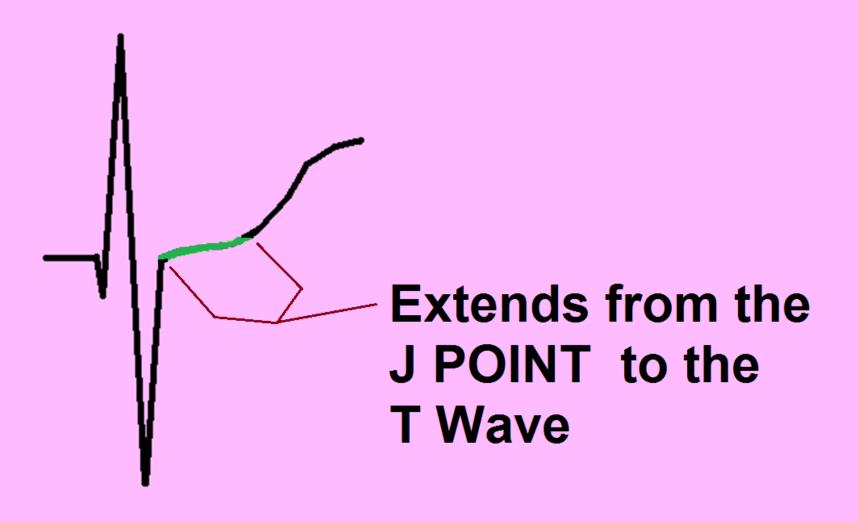
A: When the QRS is abnormally wide (> 120ms), it ALTERS the J Points, ST Segements and T Waves.

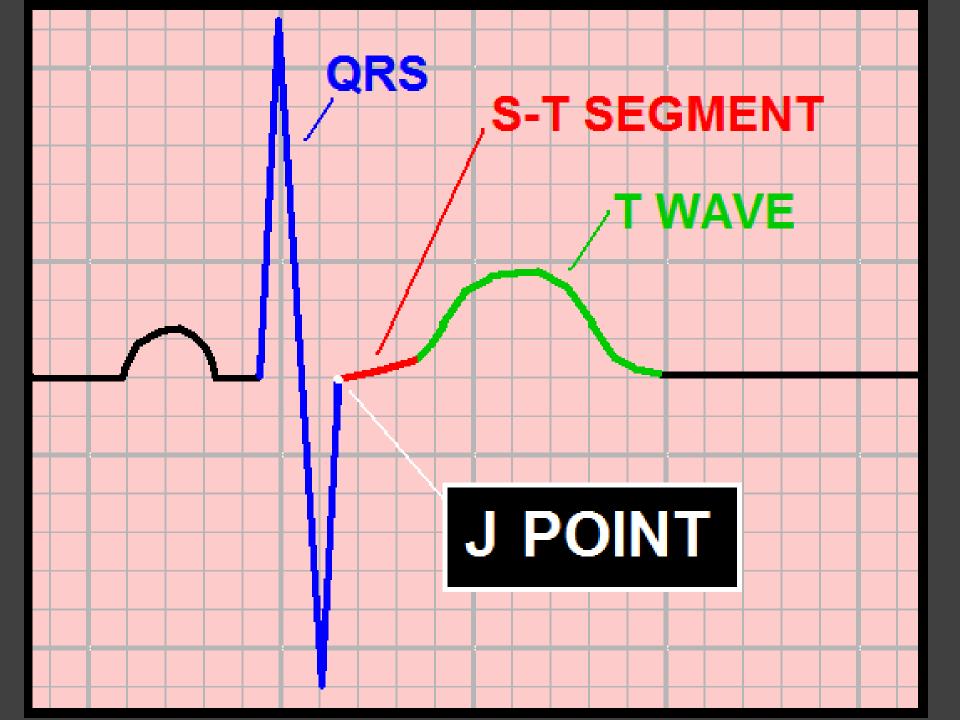
#### Defining NORMAL – QRS <120ms:



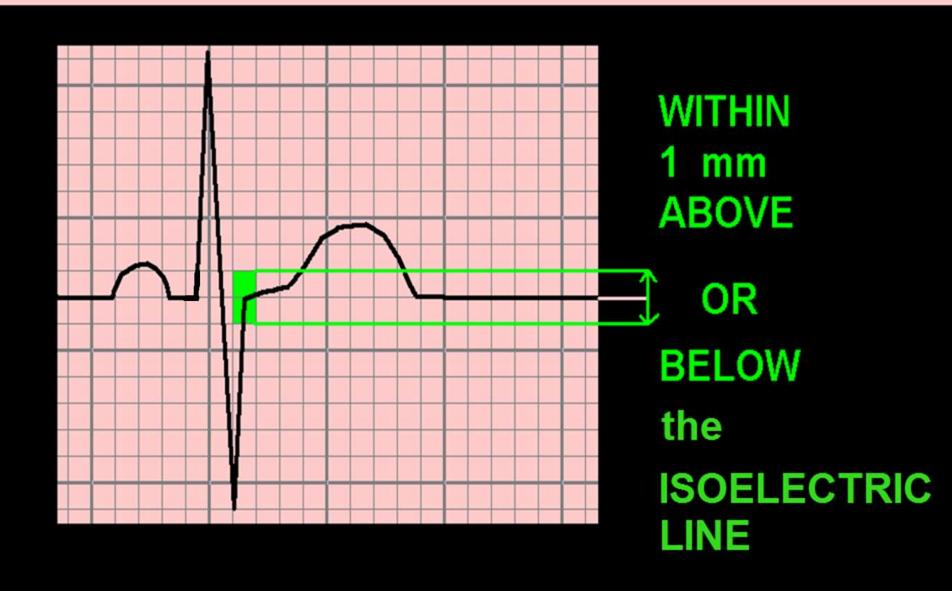
### THE J POINT



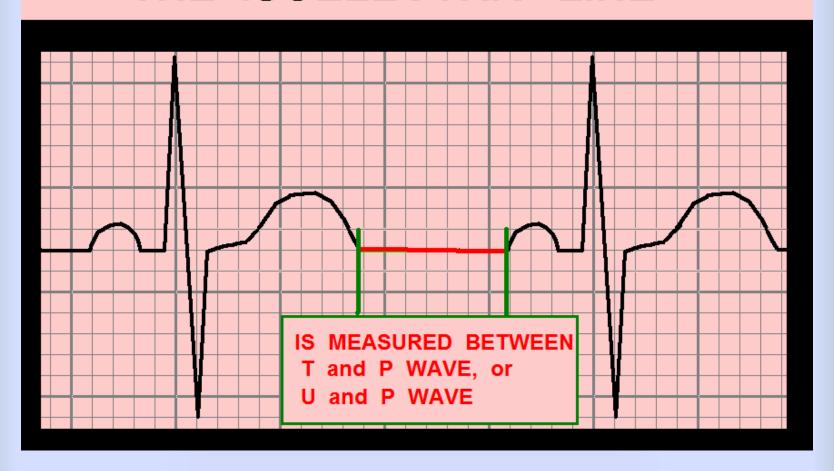




#### THE J POINT SHOULD BE ...

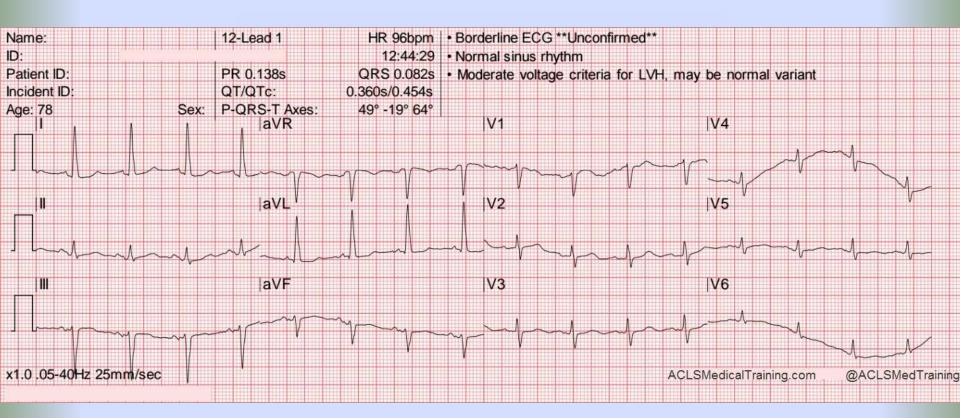


#### THE ISOELECTRIC LINE



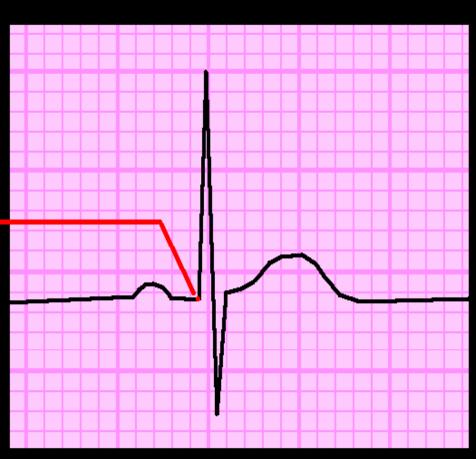
. . .the "flat line" between ECG complexes, when there is no detectable electrical activity . . .

# The Isoelectric Line - it's not always isoelectric!



#### THE P-Q JUNCTION

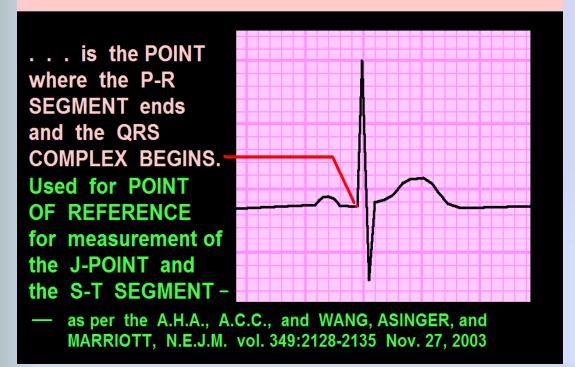
. . . is the POINT where the P-R SEGMENT ends and the QRS COMPLEX BEGINS. **Used for POINT** OF REFERENCE for measurement of the J-POINT and the S-T SEGMENT -



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

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



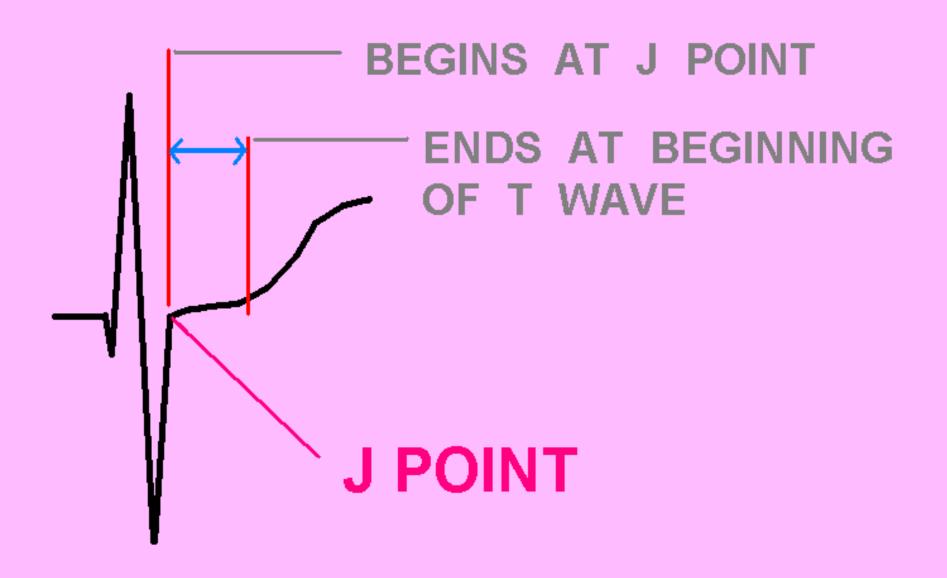


not isoelectric!"

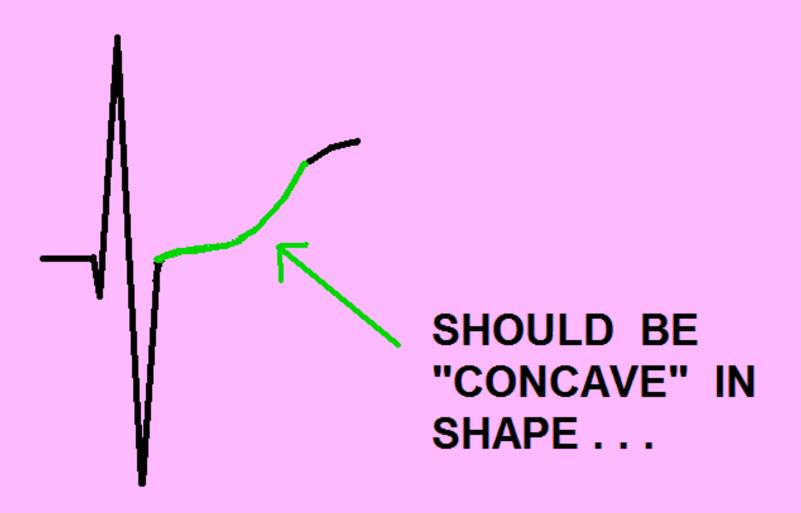
#### **Defining NORMAL:**

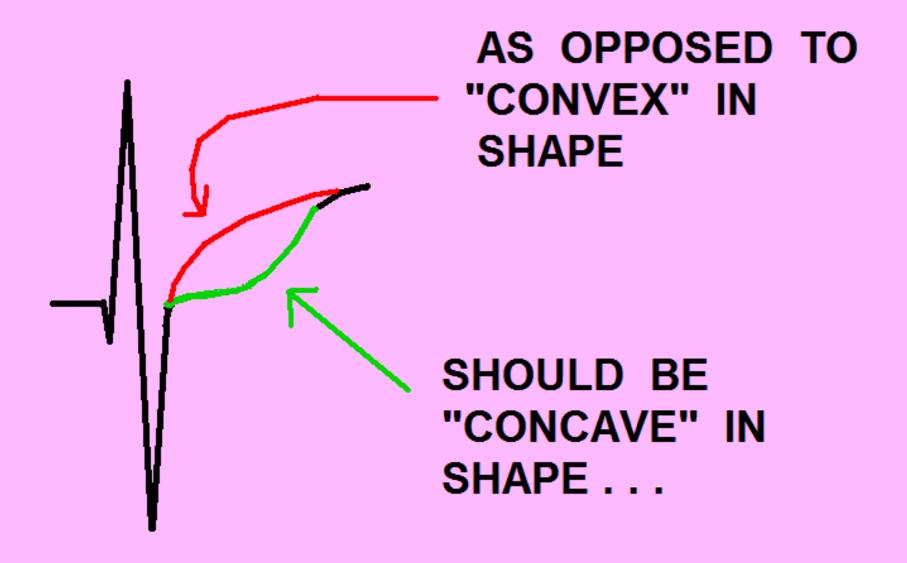
#### THE J POINT SHOULD BE ...



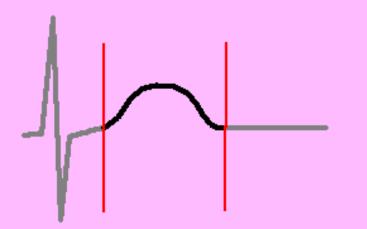








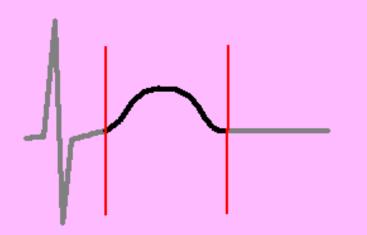
#### THE T WAVE



SHOULD BE
 A "NICE,"
 ROUNDED,
 CONVEX SHAPE

SHOULD BE SYMMETRICAL

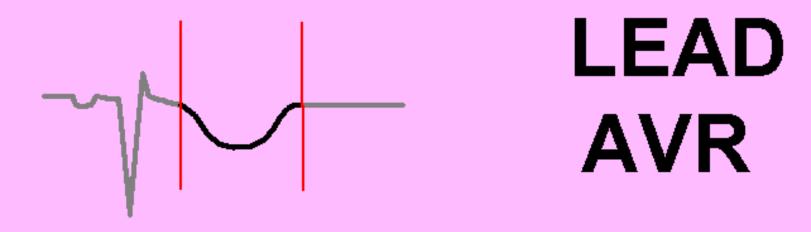
#### THE T WAVE



SHOULD BE
 A "NICE,"
 ROUNDED,
 CONVEX SHAPE

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

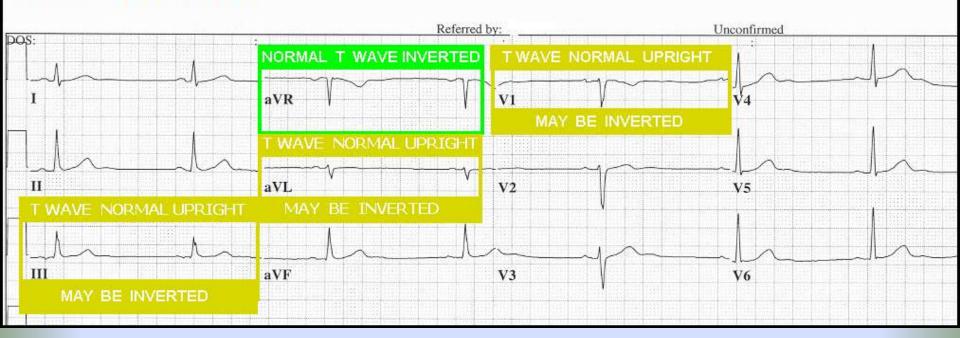
#### THE T WAVE



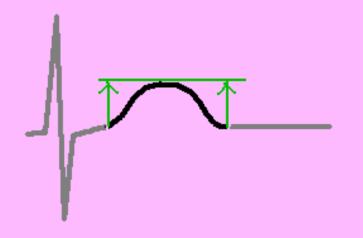
REMEMBER, IN LEAD AVR
 EVERYTHING
 IS
 "UPSIDE-DOWN"

## Normal Variants: T Wave Inversion

# Leads where the T WAVE may be INVERTED:



## THE T WAVE



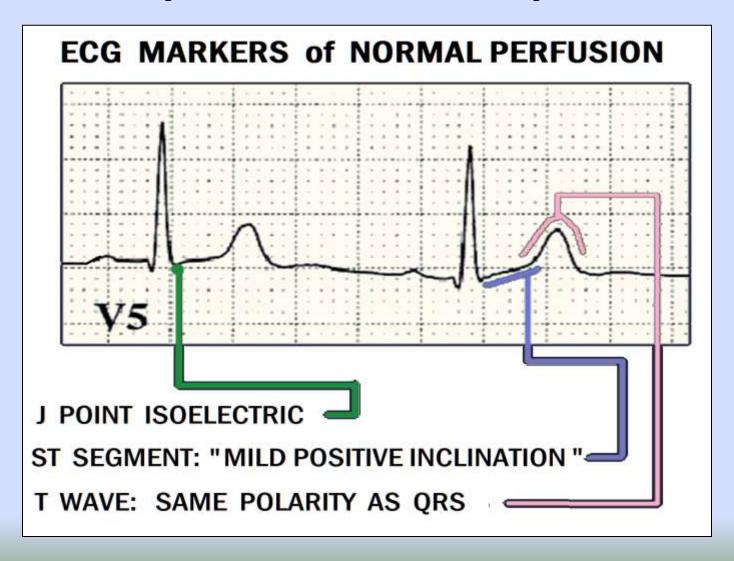
# AMPLITUDE GUIDELINES:

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

# The next slide shows an ECG waveform with *normal* J Points, ST Segments and T waves.....

# THINK OF THIS AS YOUR "MEASURING STICK" of what NORMAL is !!!

# Patients with normal QRS duration (QRS < 120 ms):



Q: If the previous slide showed what normal J Points, ST Segments and T waves look like, what is ABNORMAL?

Q: If the previous slide showed what normal J Points, ST Segments and T waves look like, what is ABNORMAL?

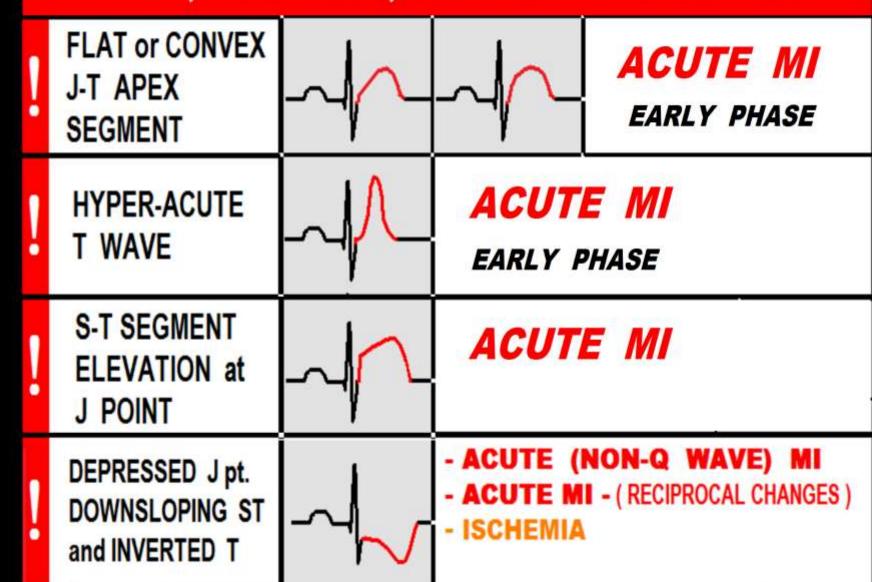
A: EVERYTHING ELSE !!!

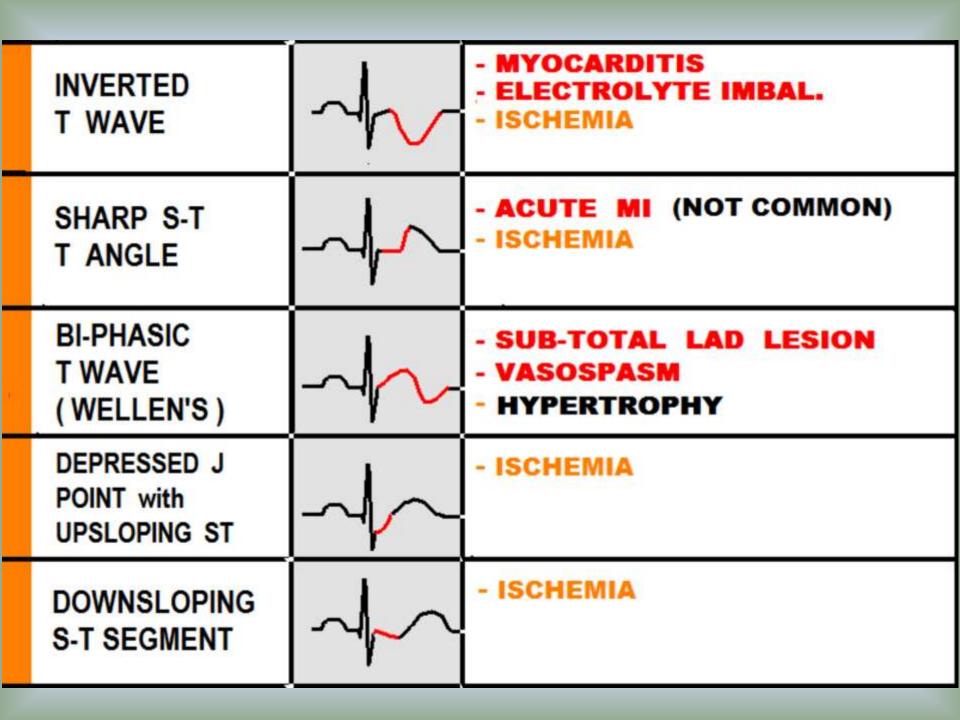
## **Up Next:**

**ECG Indicators of ABNORMAL PERFUSION** (possible ischemia / infarction) in Patients with Normal Width QRS Complexes (QRS duration < 120 ms)

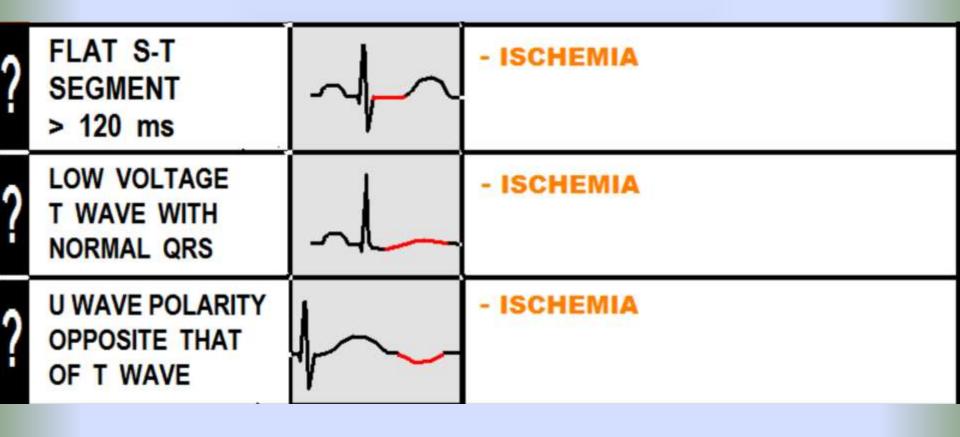
# PATTERNS of ACS & ISCHEMIA

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





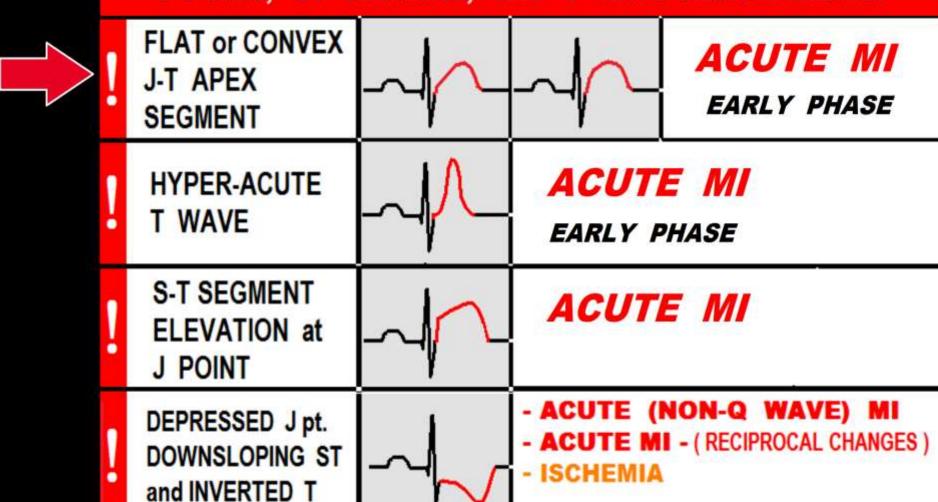
# Some less common, less reliable possible indicators of ACS:

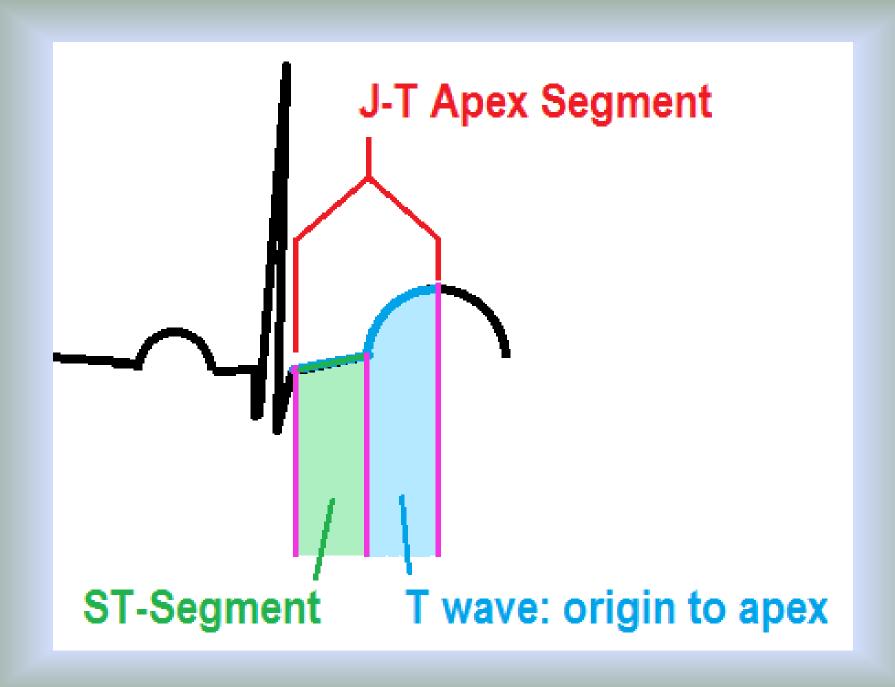


#### LET'S START HERE . . . .

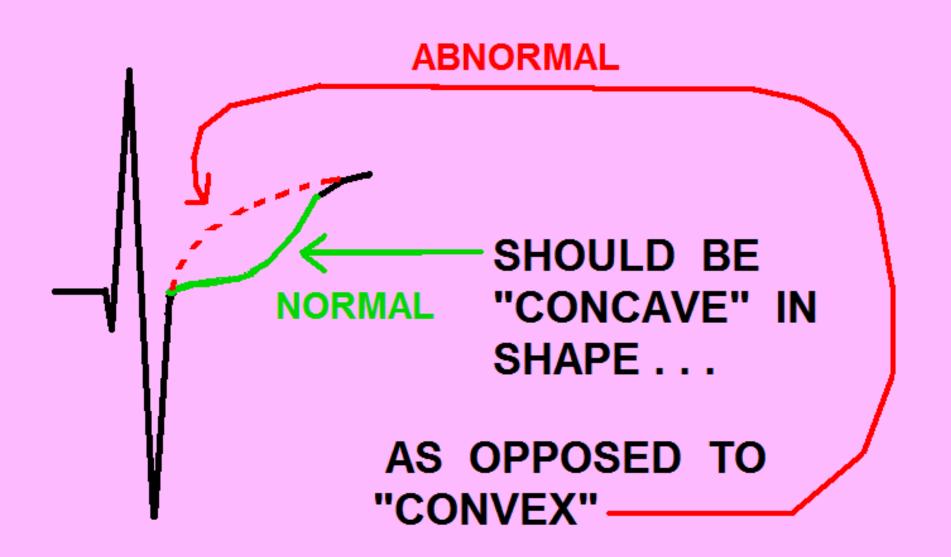
## PATTERNS of ACS & ISCHEMIA

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

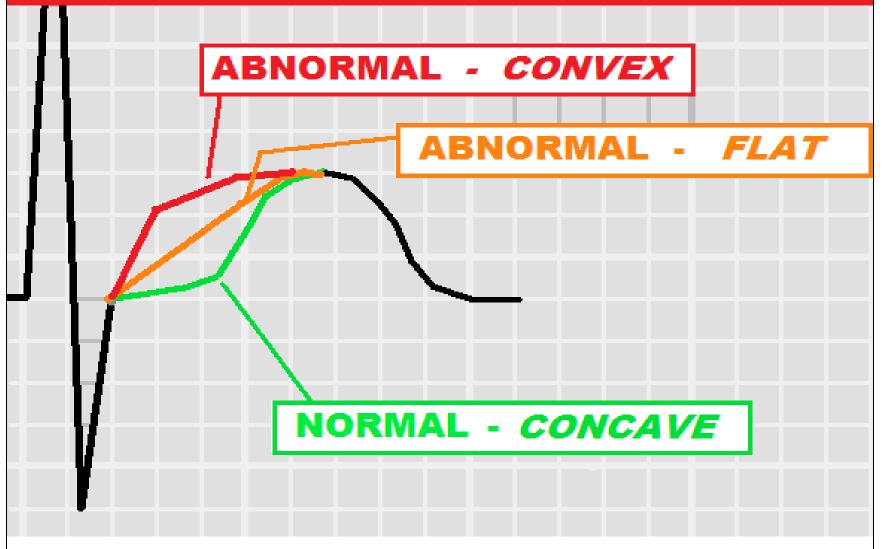




# THE S-T SEGMENT



#### J-T APEX SEGMENT VARIATIONS



#### PATTERNS of EARLY INFARCTION

-- FLAT and CONVEX J-T APEX SEGMENTS

#### 

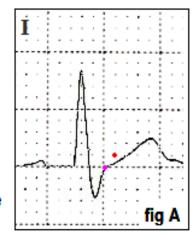
From:
AMERICAN HEART ASSOCIATION
ACLS 2005 REVISIONS

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

CONCAVE appearance.

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

Both figures were recorded from a 54 year old male while resting (figure A), and during



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

J POINT

"JPOINT plus 40 ms"

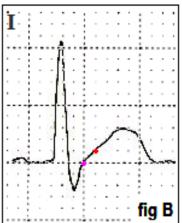
shows ST ELEVATION > 1 mm

INFARCTION -EARLY PHASE

**NORMAL** 

ST SEGMENT

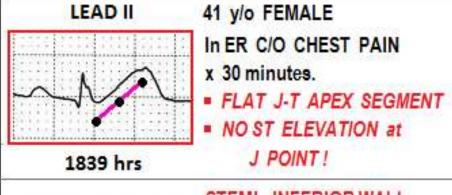
**PATTERN** 

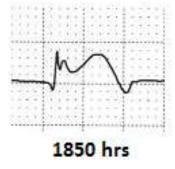


assumes a CONVEX shape.
When measured 40 ms
beyond the J POINT, the ST
segment is elevated > 1 mm.
This phenonemon is seen
routinely in the cath lab
prior to the occurance of ST
ELEVATION at the J POINT
during PTCA and STENTING.

PTCA of the Left Anterior Descending artery (figure B).

# J POINT END of ST SEGMENT T WAVE APEX FLAT J-T APEX SEGMENT CONSIDER EARLY PHASE of ACUTE MI



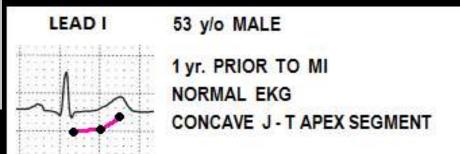


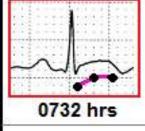
#### STEMI - INFERIOR WALL

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

 CATH LAB FINDINGS:
 TOTAL OCCLUSION of the RIGHT CORONARY ARTERY

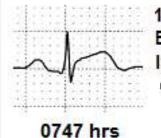
# J POINT END of ST SEGMENT T WAVE APEX CONVEX J-T APEX SEGMENT CONSIDER EARLY PHASE of ACUTE MI!





### STEMI LATERAL WALL • CONVEX J-T APEX SEGMENT

- MINIMAL ST ELEVATION
- at J POINT



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

#### CASE STUDY: ABNORMAL J-T APEX SEGMENTS

#### CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

#### **RISK FACTOR PROFILE:**

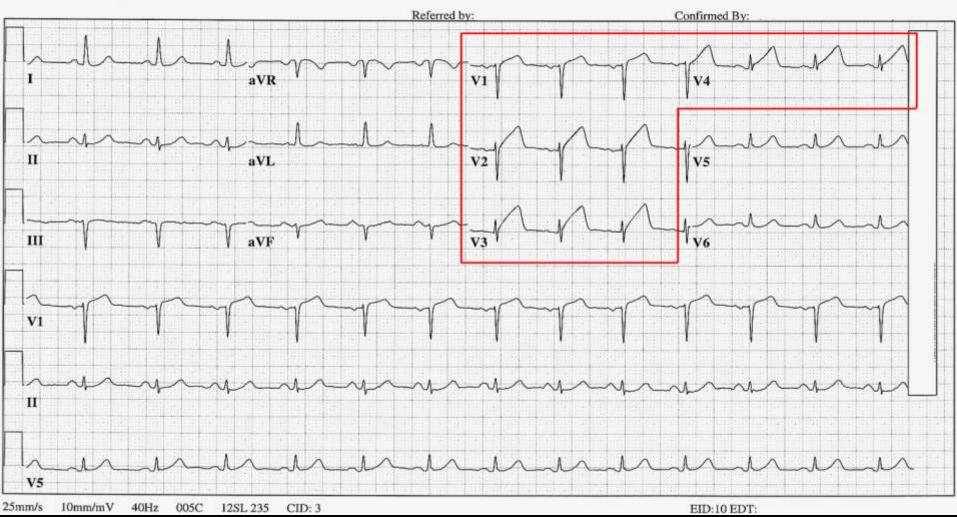
- FAMILY HISTORY father died of MI at age 62
- ☑ PREVIOUS CIGARETTE SMOKER quit 15 years ago.
- ✓ CHOLESTEROL DOES NOT KNOW; "never had it checked."
- ✓ OBESITY

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

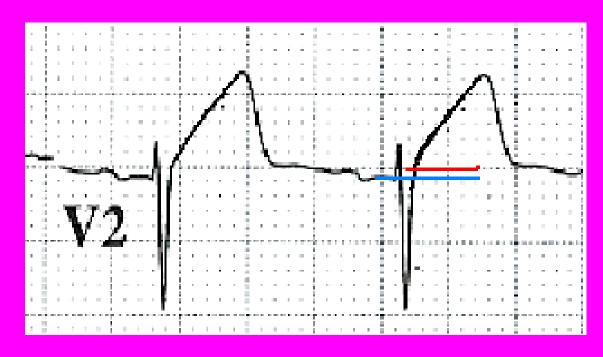
VITAL SIGNS: BP 142/94, P 80, R 20, SAO2 98%

LABS: JUST OBTAINED, RESULTS NOT AVAILABLE YET.

56 yr Vent. rate 80 **BPM** \*\*UNEDITED COPY - REPORT IS COMPUTER GENERATED ONLY, WITHOUT Male Caucasian PR interval 154 PHYSICIAN INTERPRETATION QRS duration 78 ms Normal sinus rhythm Room: A9 QT/QTc 380/438 ms Normal ECG Loc:3 Option:23 P-R-T axes 51 -24 38 No previous ECGs available Technician: W Ruppert



#### measurement of S-T elevation



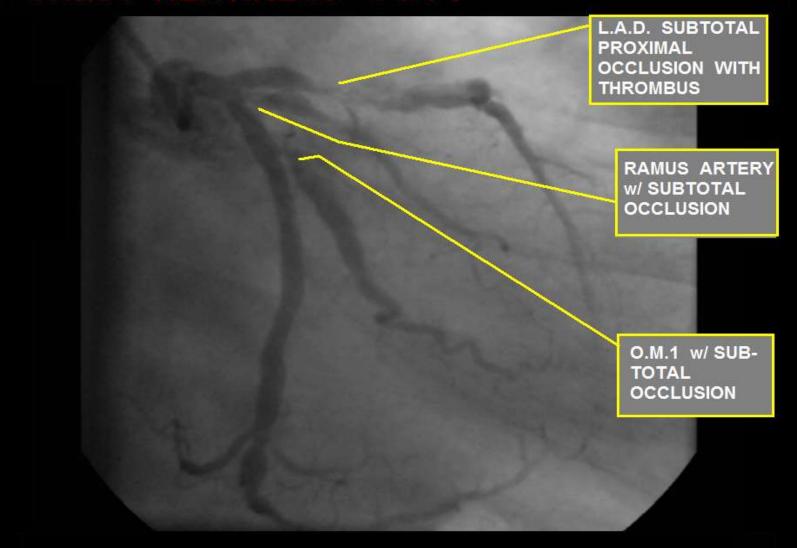
S-T elevation at J point = 0.5 mm

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



S-T elevation at J point = 0.5 mmS-T elevation at J + .04 = 2.0 mm

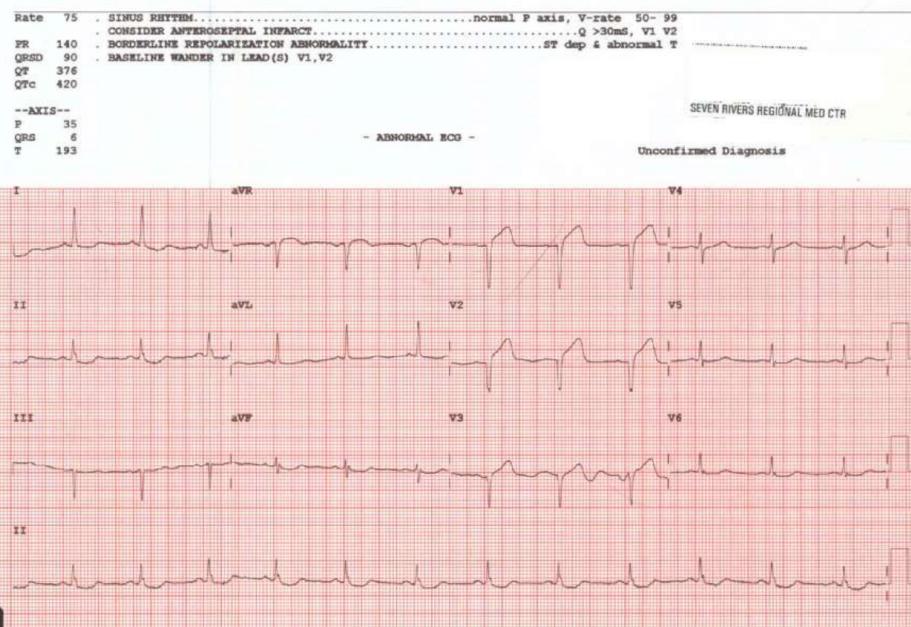
# CASE STUDY: 56 y/o male with INTERMITTENT "CHEST HEAVINESS" . . . .



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

# ECG Patterns associated with "EARLY PHASE MI:"

- J-T Apex abnormalities
- Dynamic ST-T Wave
   Changes on Serial ECGs



Chest: 10.0 mm/mV

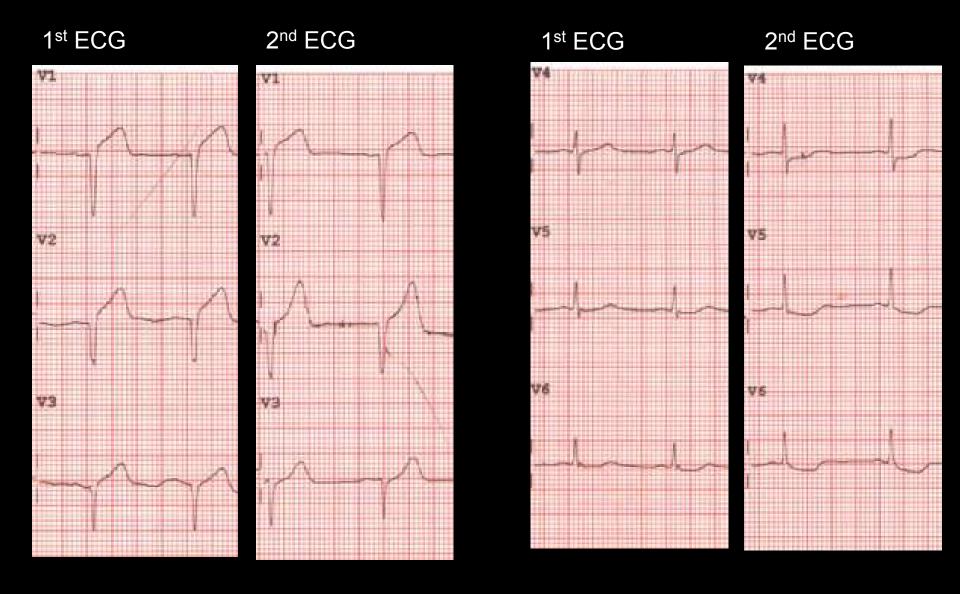
F 60- 0.15-100 Hz

PHOSON

Limb: 10 mm/mV

Dev:

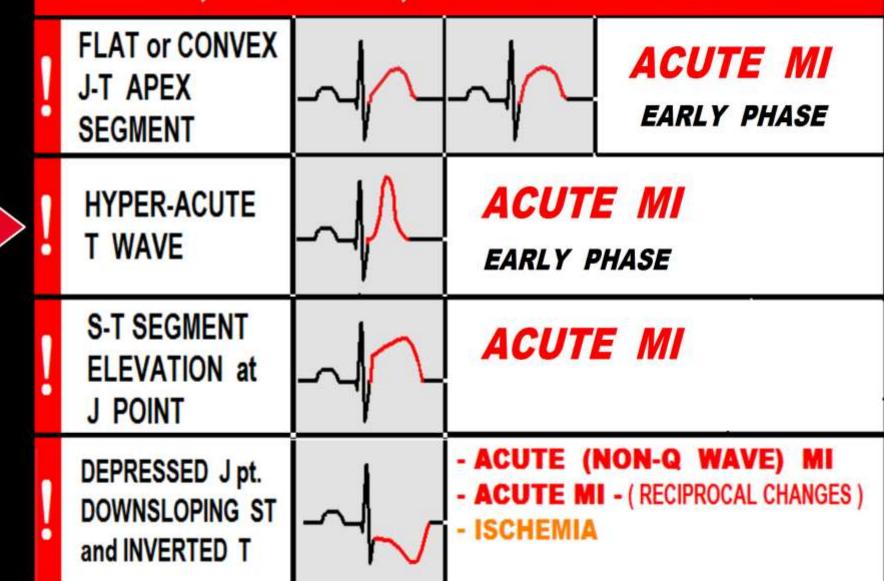
# 3. Dynmamic ST-T Wave Changes in Serial ECGs. Recorded at SRRMC



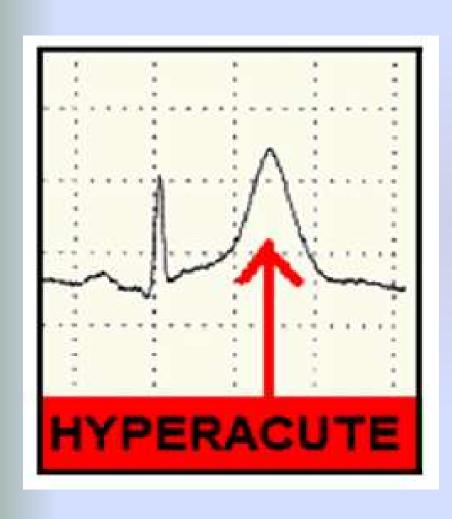
# Acute In-Stent Thrombus Proximal LAD

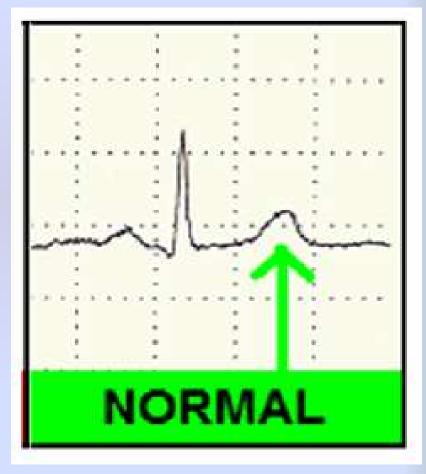
# PATTERNS of ACS & ISCHEMIA

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

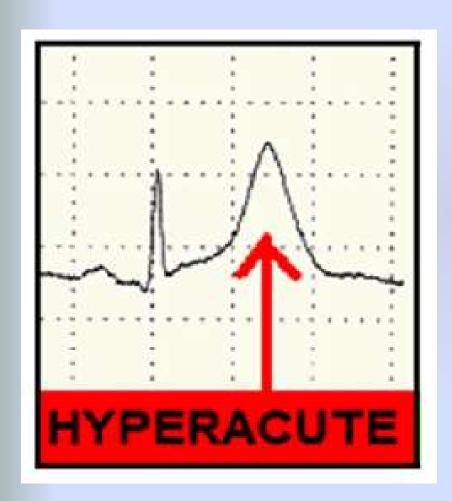


## T waves should not be HYPERACUTE



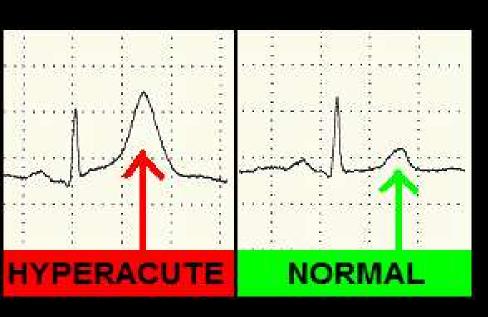


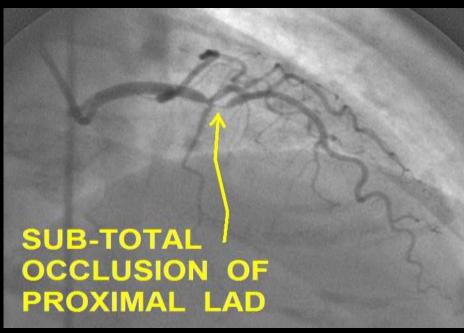
## HYPERACUTE T Waves may indicate:



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

# HYPERACUTE T WAVES





ECG waveforms obtained just before (hyperacute) and just after (normal) the critical blockage was stented in this patient's Proximal Left Anterior Descending (LAD) artery.

## Helpful Clue: Hyper-Acute T Waves

 GLOBAL Hyper-acute T Waves (in leads viewing multiple myocardial regions / arterial distributions) favors HYPERKALEMIA 55years Female

Caucasian

Room:

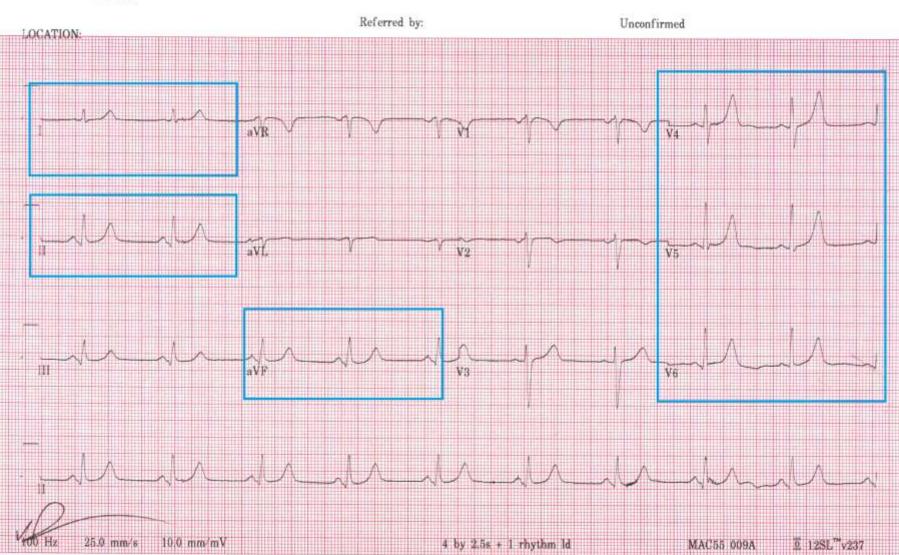
Vent. rate 57 bpm PR interval 150 ms QRS duration 102 ms QT/QTc 472/459 ms 76 70 58

Sinus bradyc Possible Left atrial enlargement Borderline ECG

ER ATTENDING REVIEW

K + = 6.7

Technician: Test ind:



## Helpful Clue: Hyper-Acute T Waves

- GLOBAL Hyper-acute T Waves (in leads viewing multiple myocardial regions / arterial distributions) favors HYPERKALEMIA
- Hyper-acute T Wave noted in ONE ARTERIAL DISTRIBUTION (Anterior / Lateral / Inferior) favors TRANSMURAL ISCHEMIA / Early Phase Acute MI

#### CASE STUDY: HYPERACUTE T WAVES

#### CHIEF COMPLAINT and SIGNIFICANT HISTORY:

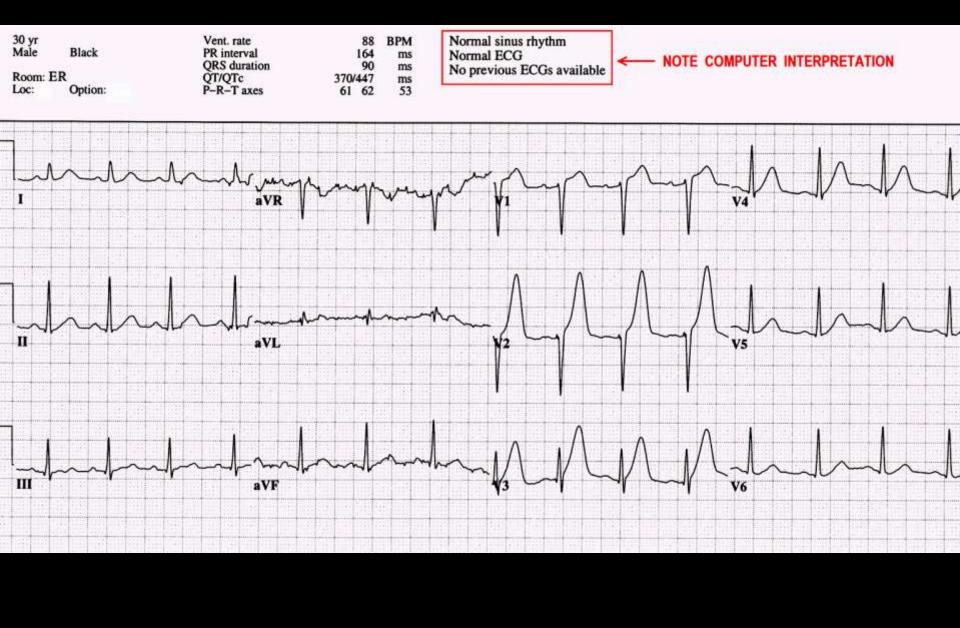
30 y/o male presents to ER via EMS, c/o sudden onset of dull chest pain x 40 min. Pain level varies, not effected by position, movement or deep inspiration. No associated symptoms.

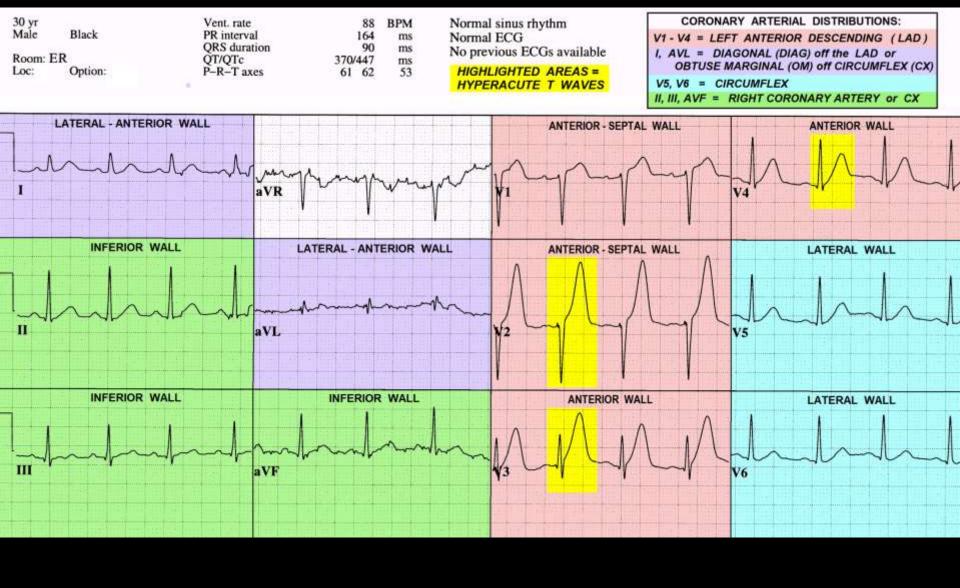
RISK FACTOR PROFILE: NONE. CHOLESTEROL UNKNOWN.

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

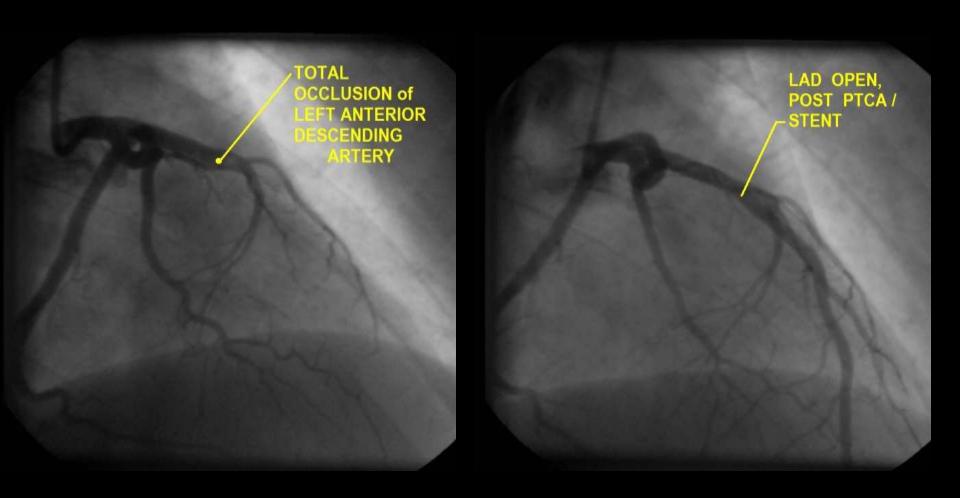
VITAL SIGNS: BP 136/88 P 90 R 20 SAO2 98%

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





### Cath Lab findings:



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

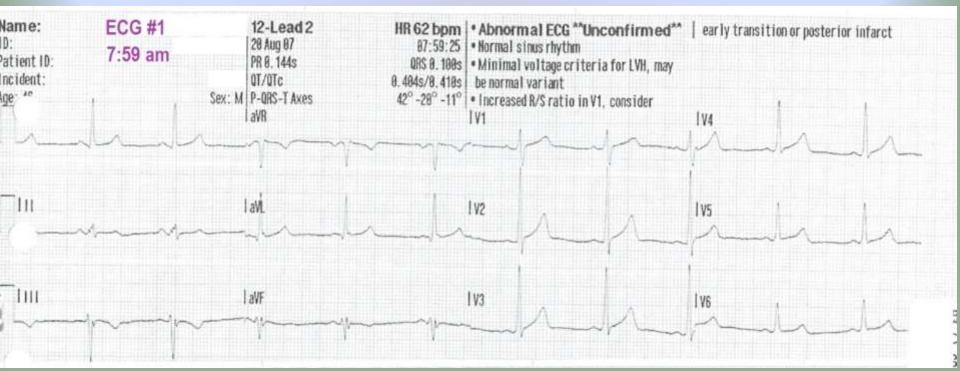
 Other than HEART RATE related variations (which affect intervals), J Points, ST-Segments and T Waves SHOULD NOT CHANGE.

#### **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.

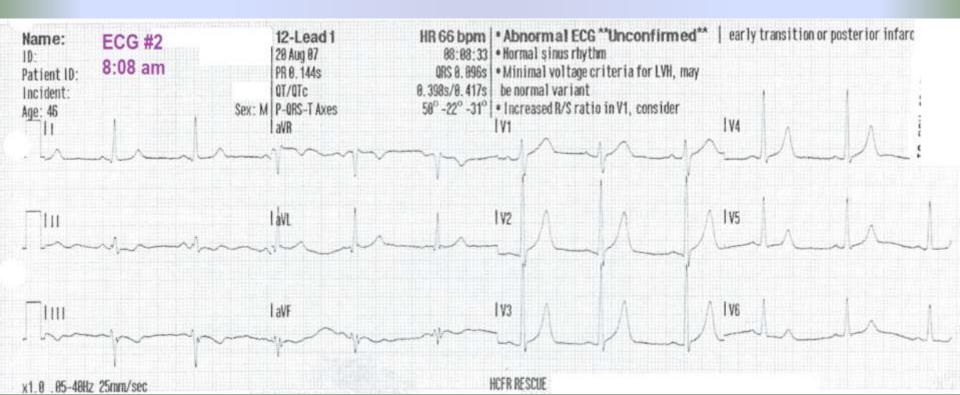
#### 46 year old male

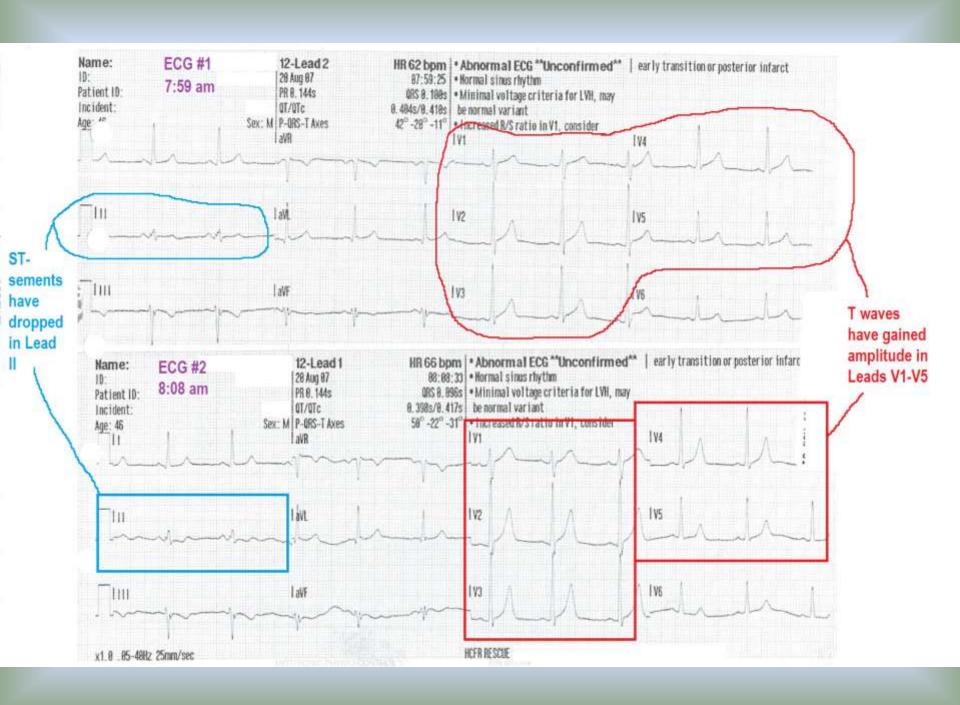
- Exertional dyspnea X "several weeks"
- Intermittent chest pressure X last 3 hours.
   Currently pain free.

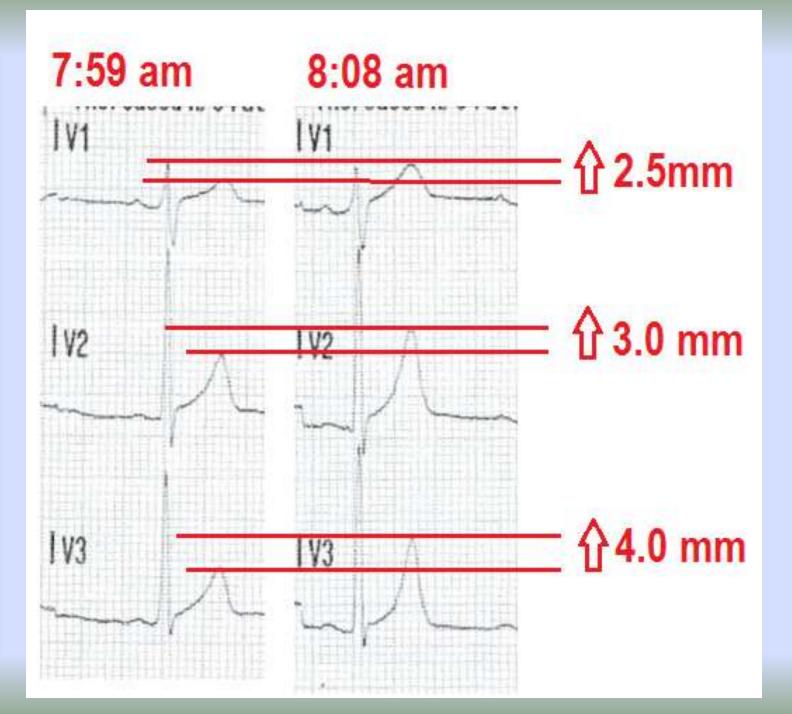


#### 46 year old male: ECG 1

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







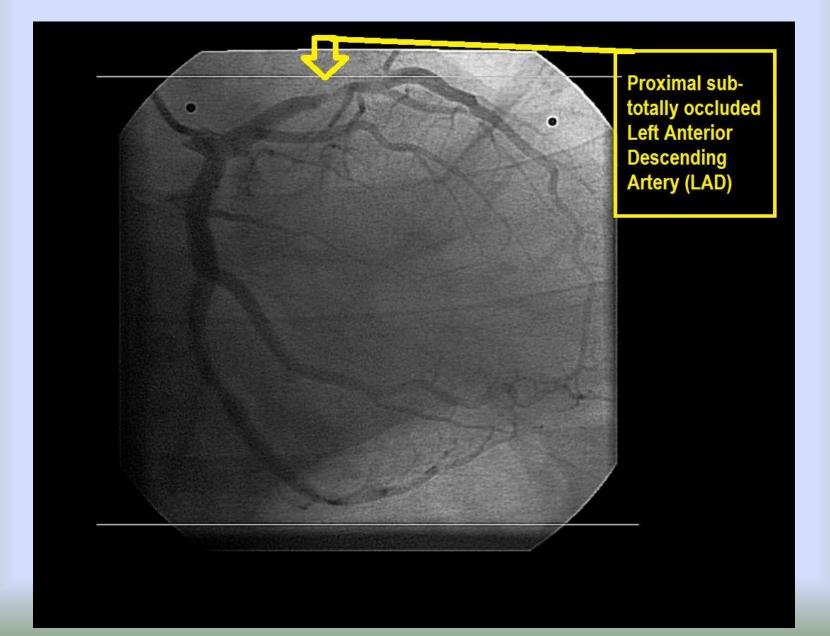
#### ST-Segment Depression

7:59 am

8:08 am



#### Cath Lab Angiography:

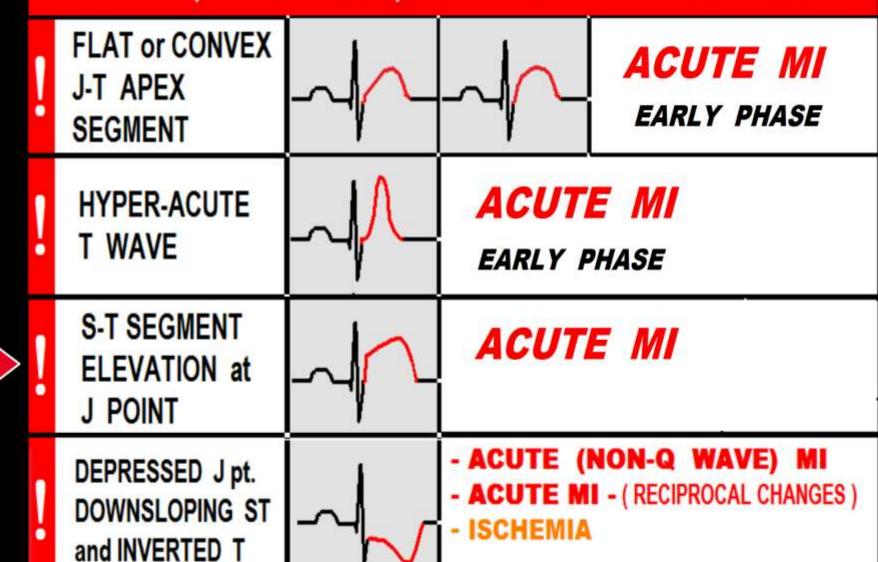




"NOWHERE", NEW MEXICO, 1994

#### PATTERNS of ACS & ISCHEMIA

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



## ECG CRITERIA for DIAGNOSIS of STEMI: (ST ELEVATION @ J POINT)

#### \*LEADS V2 and V3:

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

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

FEMALES ----- 1.5 mm

**ALL OTHER LEADS:** 1.0 mm or more,

in TWO or more

**CONTIGUOUS LEADS** 

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

#### ST SEGMENT ELEVATION:

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



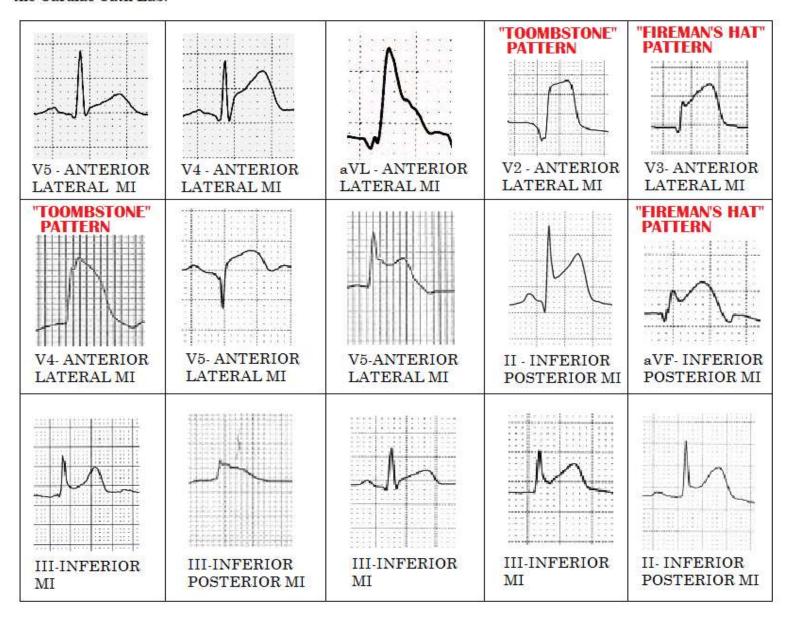
IN THIS CASE, a normal response to balloon occlusion of the RIGHT CORONARY ARTERY during PTCA in the CARDIAC CATH LAB

3 COMMON PATTERNS of ST SEGMENT ELEVATION From ACUTE MI:



#### ST SEGMENT ELEVATION in ACUTE MI:

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

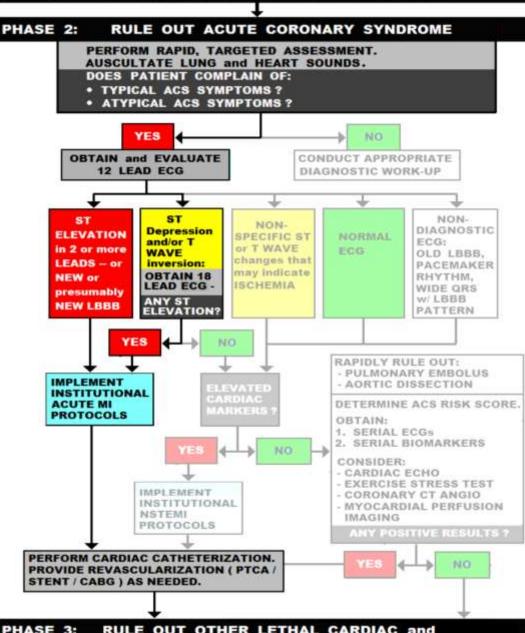


Reciprocal S-T Segment Depression *may* or *may not* be present during AMI.

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

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

### STEMI GASE STUDIES



**RULE OUT LIFE-THREATENING CONDITIONS** 

PHASE 1:

PHASE 3: RULE OUT OTHER LETHAL CARDIAC and NON-CARDIAC CONDITIONS.

### ACUTE MI

## COMPLICATIONS TO ANTICIPATE FOR ALL MI PATIENTS:

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



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

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 "crystal ball!"



#### INTERPRET THE EKG, THEN:

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

## 

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

#### CASE STUDY 1 - STEMI

#### CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

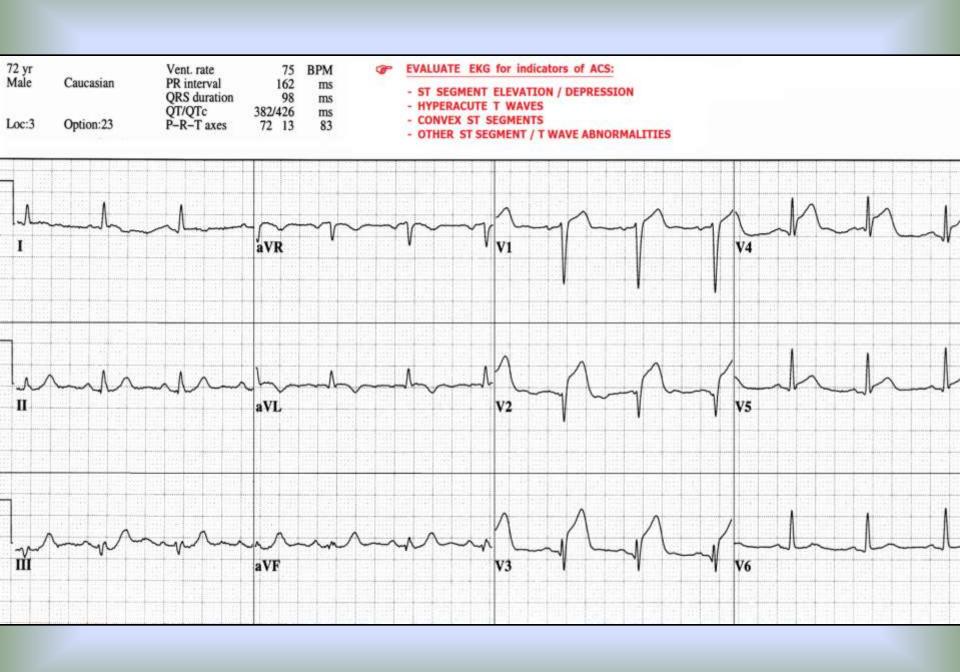
#### RISK FACTOR PROFILE:

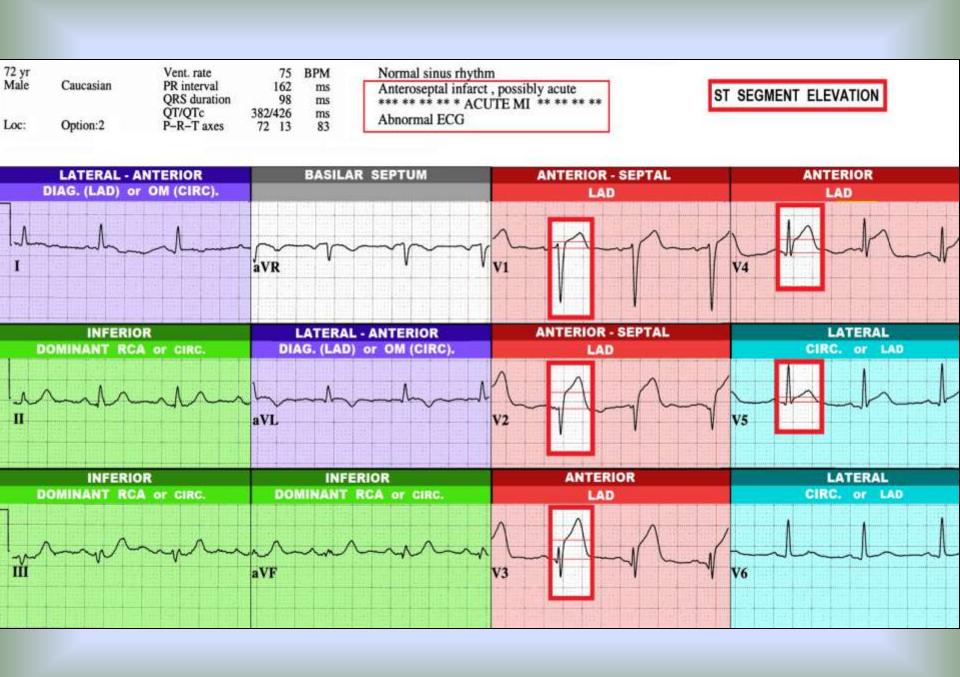
- FAMILY HISTORY father died of MI at age 77
- FORMER CIGARETTE SMOKER smoked for 30 year quit 27 years ago
- **► DIABETES** oral meds and diet controlled
- HIGH CHOLESTEROL controlled with STATIN meds
- AGE: OVER 65
- PHYSICAL EXAM: Patient calm, alert, oriented X 4, skin cool, dry, pale.

No JVD, Lungs clear bilaterally. Heart sounds normal S1, S2. No peripheral edema.

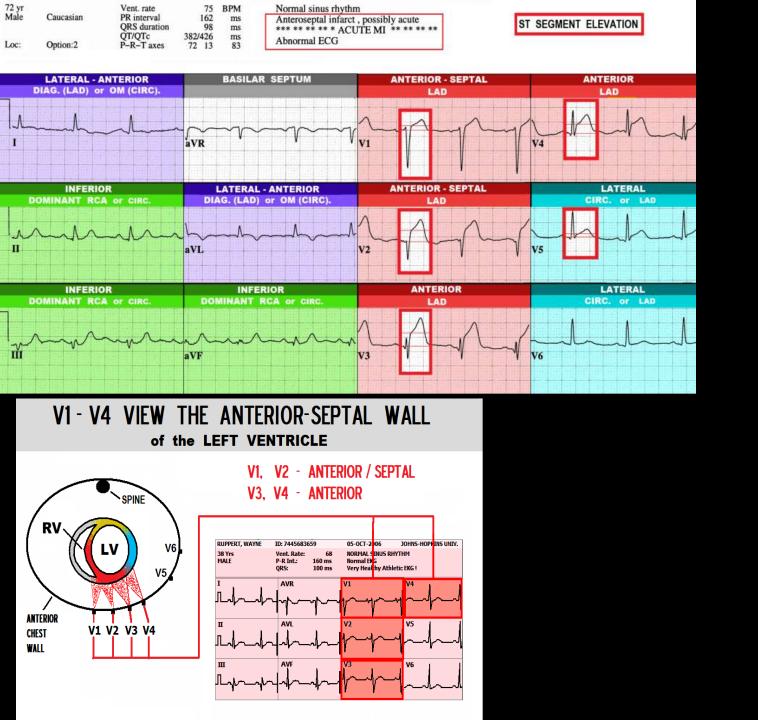
VITAL SIGNS: BP: 100/64, P: 75, R: 20, SAO2: 94%

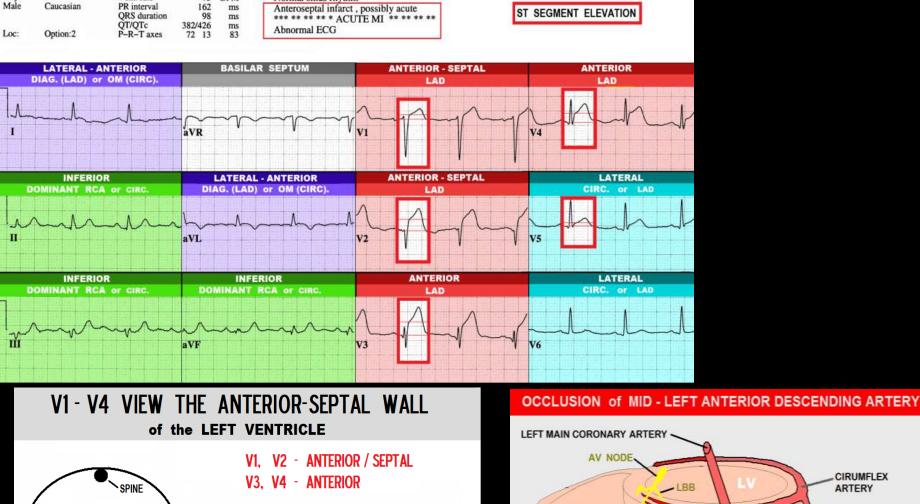
LABS: FIRST TROPONIN: 6.4





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





05-0CT-2 06

Normal FK

NORMAL SINUS RHYTHM

Very Heal thy Athletic EKG!

JOHNS-HOPKINS UNIV.

Normal sinus rhythm

Vent. rate

PR interval

ORS duration

Caucasian

75 **BPM** 

98

ms

RUPPERT, WAYNE

MALE

V6

V1 V2 V3 V4

ANTERIOR

CHEST WALL

ID: 7445683659

160 ms

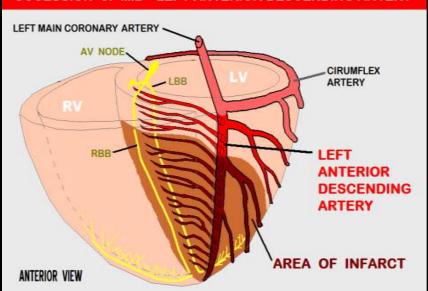
Vent. Rate:

P-R Int.:

AVR

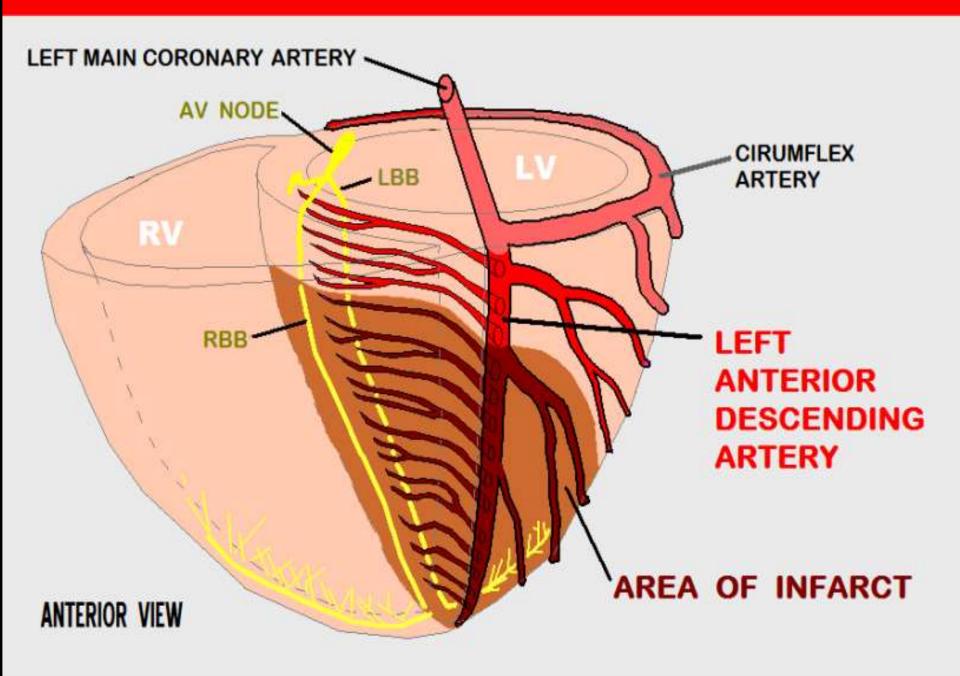
QRS:

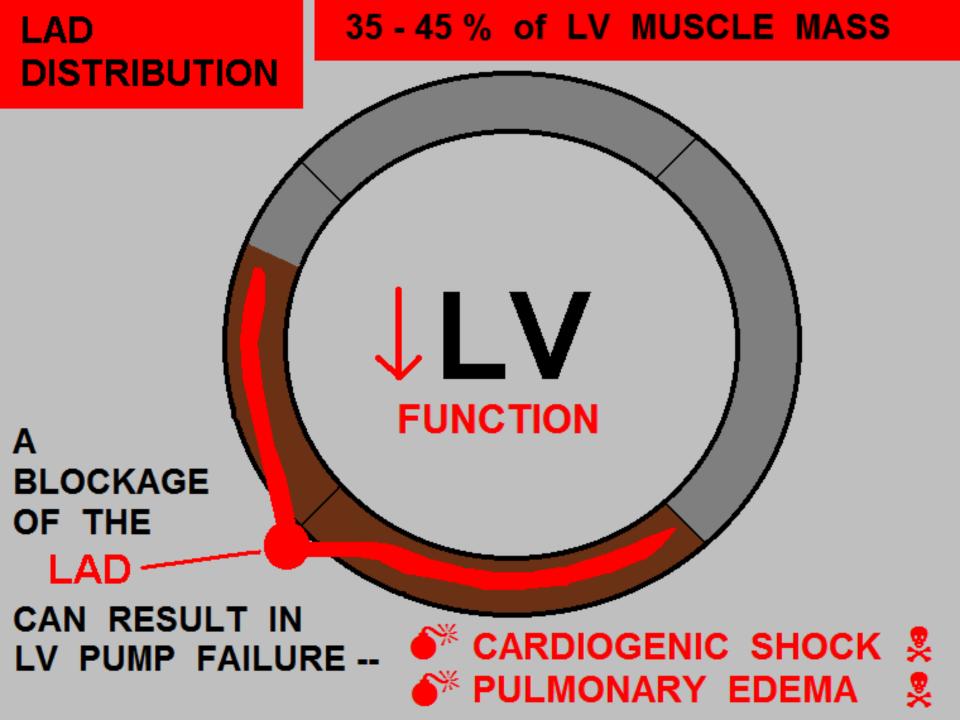
162



ST SEGMENT ELEVATION

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







## LEFT ANTERIOR DESCENDING ARTERY (LAD)

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

## ANTICIPATED COMPLICATIONS of ANTERIOR-SEPTAL WALL STEMI & POSSIBLE INDICATED INTERVENTIONS: - CARDIAC ARREST - CARDIAC DYSRHYTHMIAS (VT / VF) ACLS (antiarrhythmics)

- INTRA-AORTIC BALLOON PUMP

(use caution with fluid challenges

(use caution with dieuretics due to

pump failure and hypotension)

due to PULMONARY EDEMA)

- CPAP

- ET INTUBATION

TRANSCUTANEOUS or

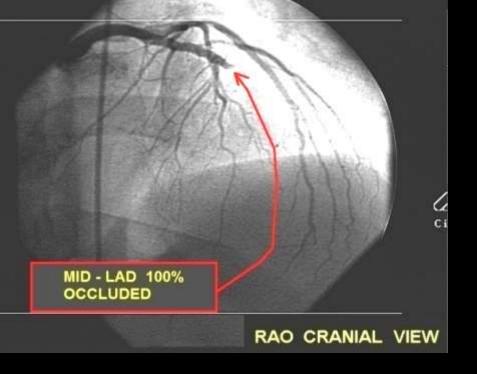
TRANSVENOUS PACING

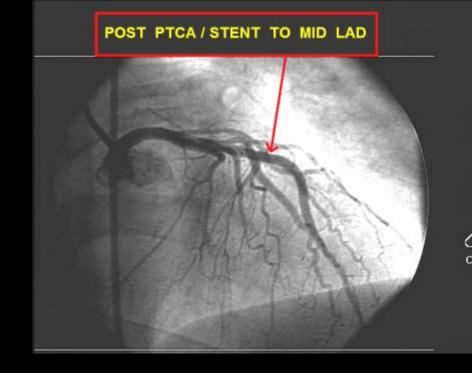
- CARDIAC DYSRHYTHMIAS (VT / VF)
- PUMP FAILURE with
CARDIOGENIC SHOCK
- DOPAMINE / DOBUTAMINE / LEVOPHED

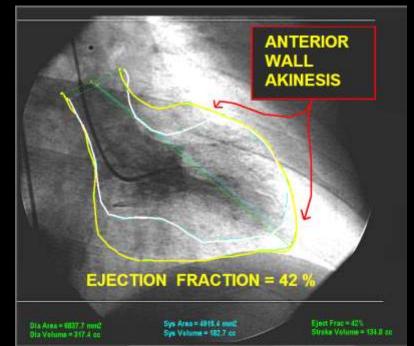
- PULMONARY EDEMA

- 3rd DEGREE HEART BLOCK - NOT

**RESPONSIVE TO ATROPINE** 







#### 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:

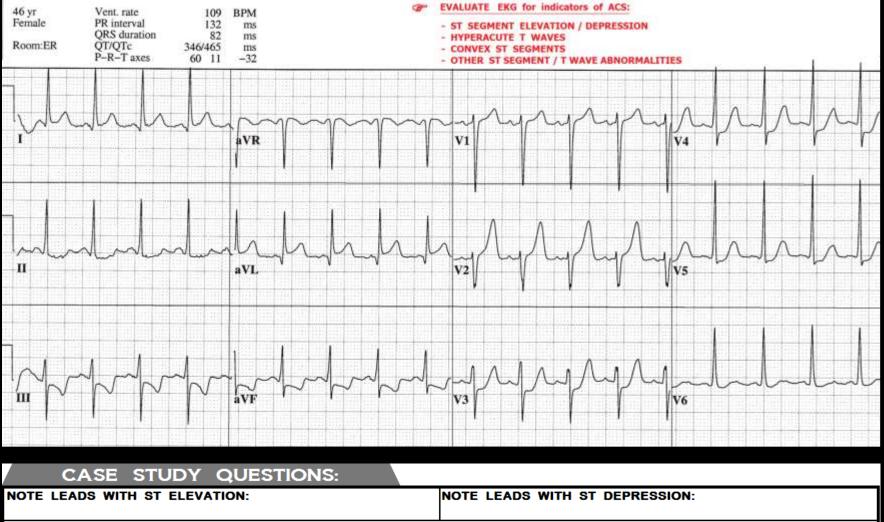
- FAMILY HISTORY father died of CAD, older brother had CABG, age 39
- DIABETES diet controlled
- HYPERTENSION

PHYSICAL EXAM: Pt. CAOx4, anxious, SKIN cold, clammy, diaphoretic. No JVD.

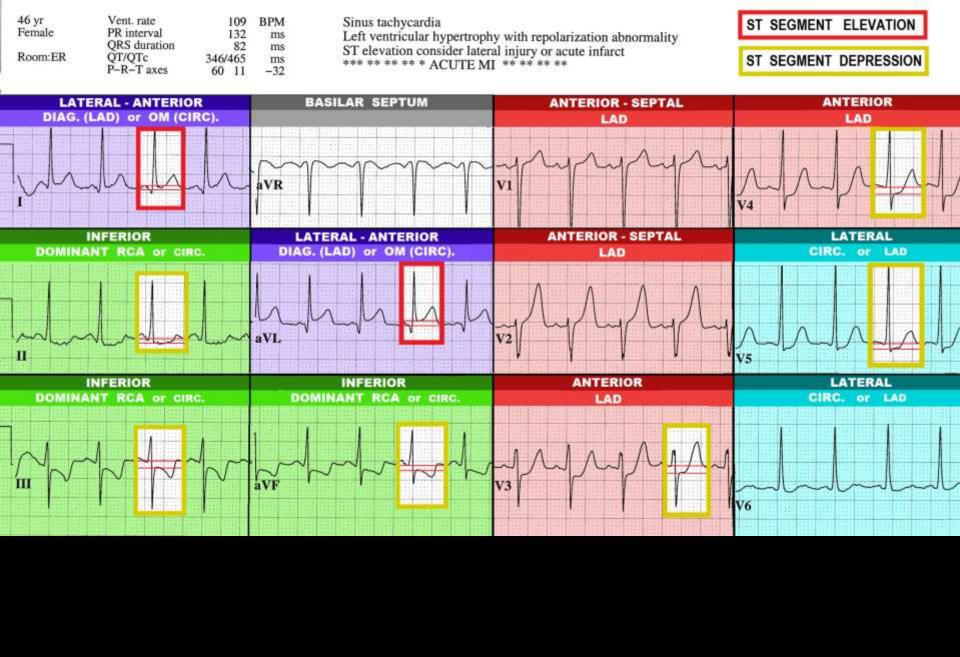
Lungs: clear, bilaterally. Heart Sounds: Normal S1, S2.

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

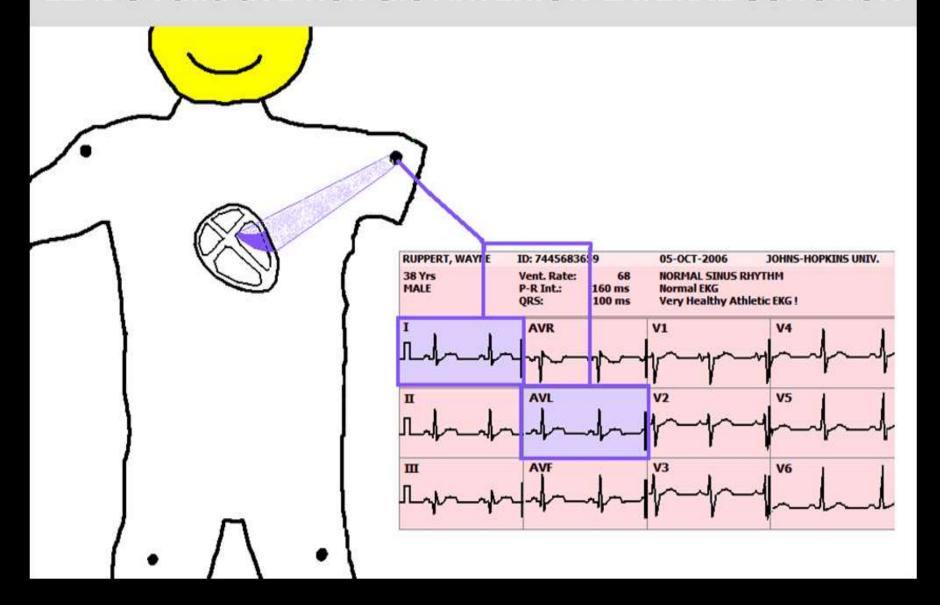
LABS: TROPONIN ultra = 2.8

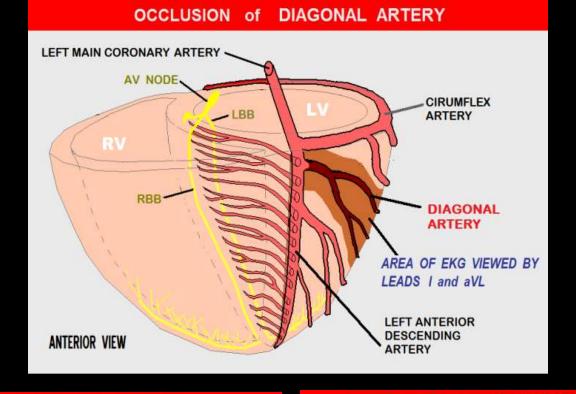


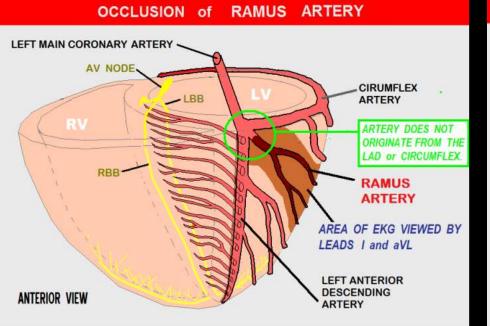
# NOTE LEADS WITH ST ELEVATION: WHAT IS THE SUSPECTED DIAGNOSIS ? WHAT IS THE "CULPRIT ARTERY" -- if applicable? LIST ANY CRITICAL STRUCTURES COMPROMISED: LIST ANY POTENTIAL COMPLICATIONS:

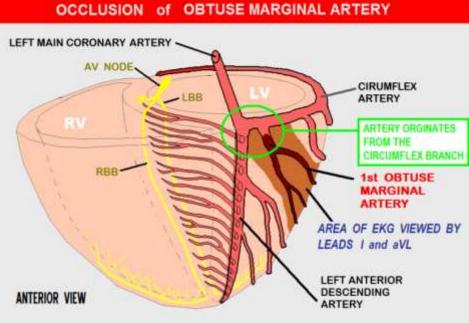


### LEADS I and aVL view the ANTERIOR-LATERAL JUNCTION

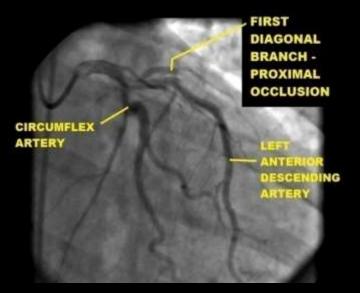


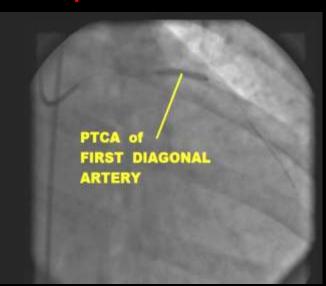




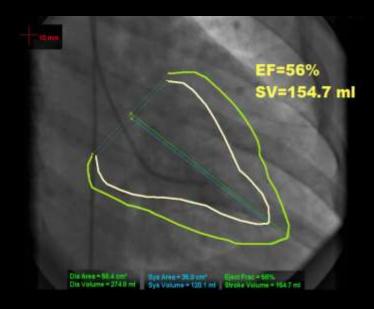


CASE PROGRESSION: As the patient was being prepared for transport to the Cardiac Cath Lab, she experienced an episode of Ventricular Fibrillation.









CASE STUDY SUMMARY		
ST ELEVATION: I, aVL	ST DEPRESSION:	II, III, aVF, V3 - V5
SUSPECTED DIAGNOSIS: ACUTE LA	TERAL WALL M.I.	
SUSPECTED "CULPRIT ARTERY" (if applica	able):	
USUALLY ONE OF THE SMALLER SIE	DE-BRANCH ARTERIES:	
1. DIAGONAL ARTERY. (This is a	side-branch artery off of the LEF	T ANTERIOR DESCENDING (LAD) artery.
2. OBTUSE MARGINAL ARTERY. (Th		
3. RAMUS ARTERY.	White the second of the second of the second	
IMMEDIATE CONCERNS FOR ALL STEMI P	ATIENTS:	
■ BE PREPARED TO MANAGE SUDDEN C	ARDIAC ARREST (PRIMARY V-FIB/	/- TACH, BRADYCARDIAS / HEART BLOCKS )
STAT REPERFUSION THERAPY: THR		RIZATION and PCI
CONSIDER NEEDS FOR ANTI-PLATELET	and ANTI-COAGULATION THERAPY	
CRITICAL STRUCTURES COMPROMISED:	POTENTIAL COMPLICATIONS:	POSSIBLE CRITICAL INTERVENTIONS:
6 <sup>34</sup> 15-30% of the LV −	POSSIBLE MODERATE	INOTROPIC AGENTS
MUSCLE MASS	LV PUMP FAILURE	ET INTUBATION
a destruction of the state of t	Et Tom Paleone	I.A.B.P. INSERTION

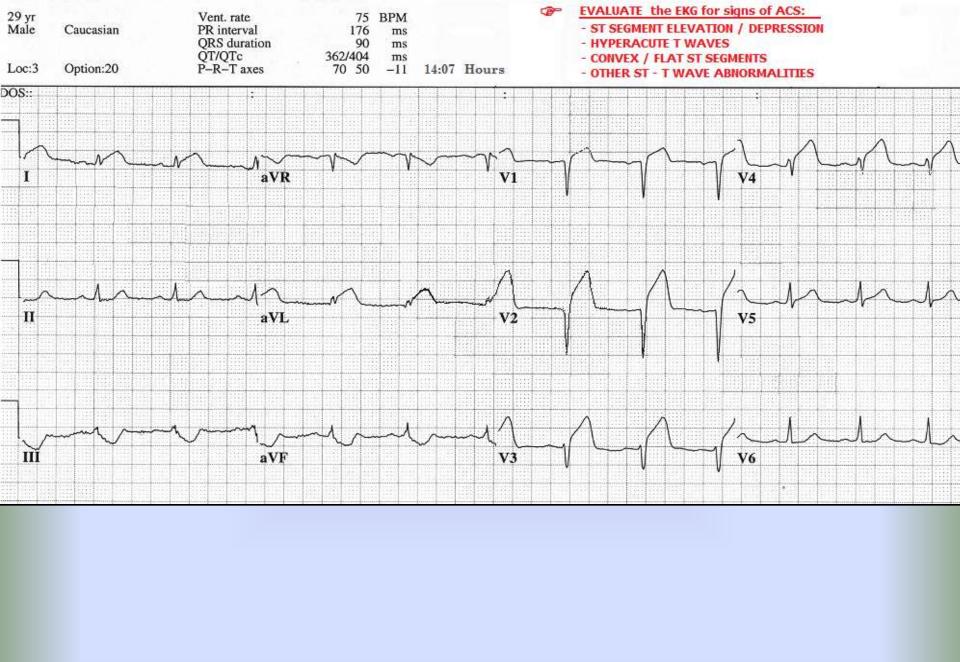
### CASE STUDY 3: STEMI

### CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

### RISK FACTOR PROFILE:

- FAMILY HISTORY father died of MI age 46
- CURRENT CIGARETTE SMOKER
- "MILD" HYPERTENSION untreated
- ☑ CHOLESTEROL unknown "never had it checked."
- PHYSICAL EXAM: Patient alert, oriented X 4, skin cool, dry, pale. Patient restless. No JVD, Lungs clear bilaterally. Heart sounds normal S1, S2. No peripheral edema.
- VITAL SIGNS: BP: 104/78, P: 76, R: 20, SAO2: 96%
- LABS: INITIAL CARDIAC MARKERS NEGATIVE



29 yr Male

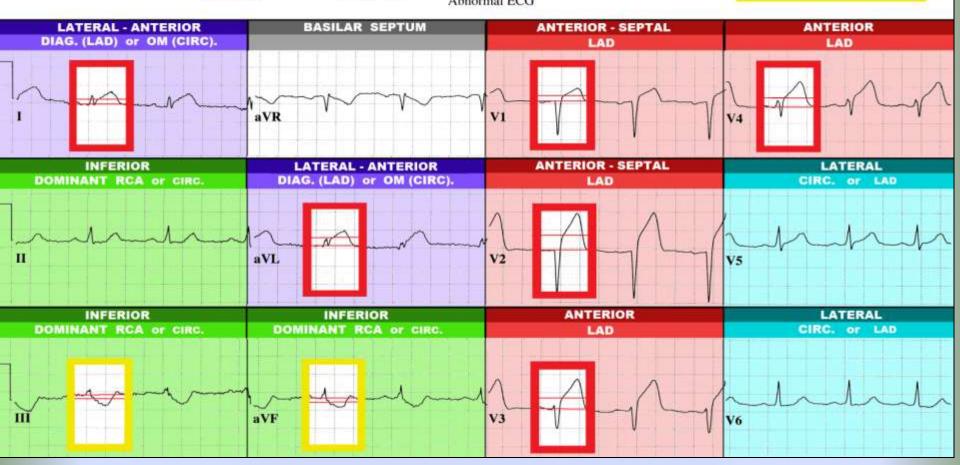
Caucasian

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

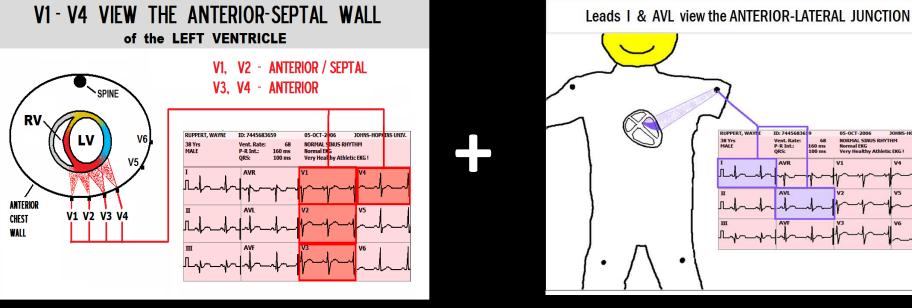
75 BPM 176 ms 90 ms 362/404 ms 70 50 -11 Normal sinus rhythm
Septal infarct, possibly acute
Anterolateral injury pattern
\*\*\* \*\* \*\* \*\* ACUTE MI \*\* \*\* \*\*
Abnormal ECG

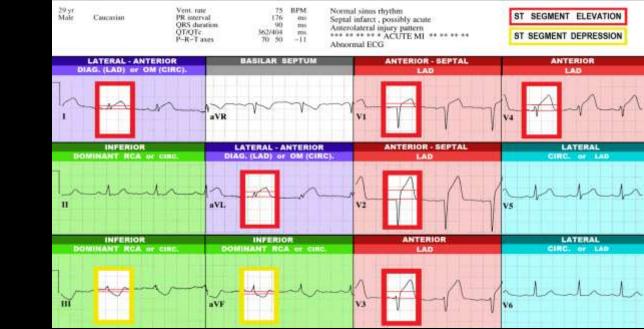
ST SEGMENT ELEVATION

ST SEGMENT DEPRESSION

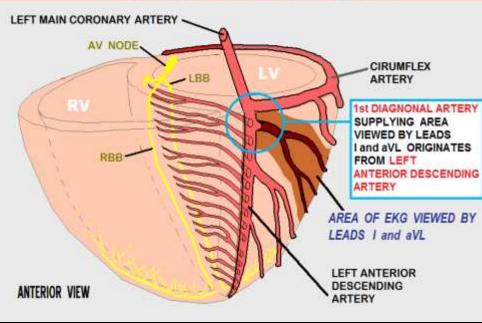


- Reciprocal ST Depression is NOW PRESENT
- Additional ST Elevation is present in Leads I, AVL



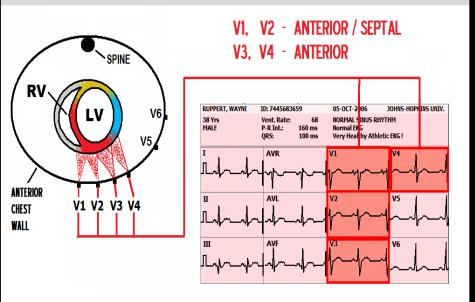


# Leads I & AVL view the ANTERIOR-LATERAL JUNCTION | INITIPITAT, WARTE | 10: 744568360 | 05-0ct - 2006 | 20:80c - 1009K016 UREV. | | World. Baris | 160 erg |

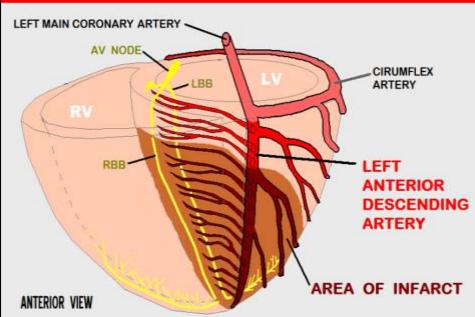


OCCLUSION of DIAGONAL ARTERY

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



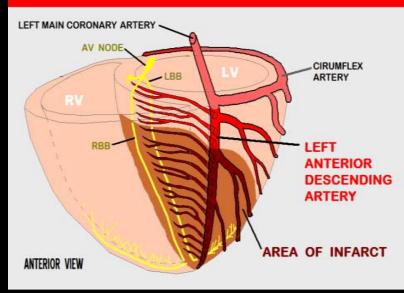
#### OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY



# CIRUMFLEX ARTERY SUPPLYING AREA VIEWED BY LEADS I and aVL ORIGINATES FROM LEFT

ANTERIOR VIEW







ANTERIOR DESCENDING

AREA OF EKG VIEWED BY LEADS I and aVL

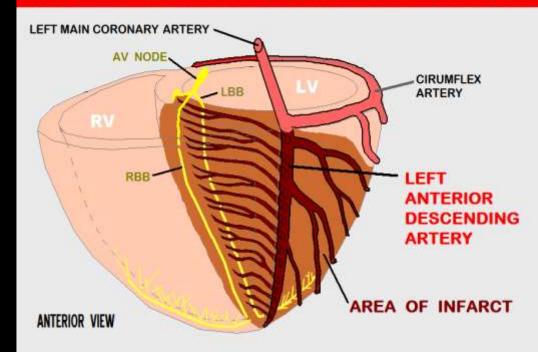
ARTERY

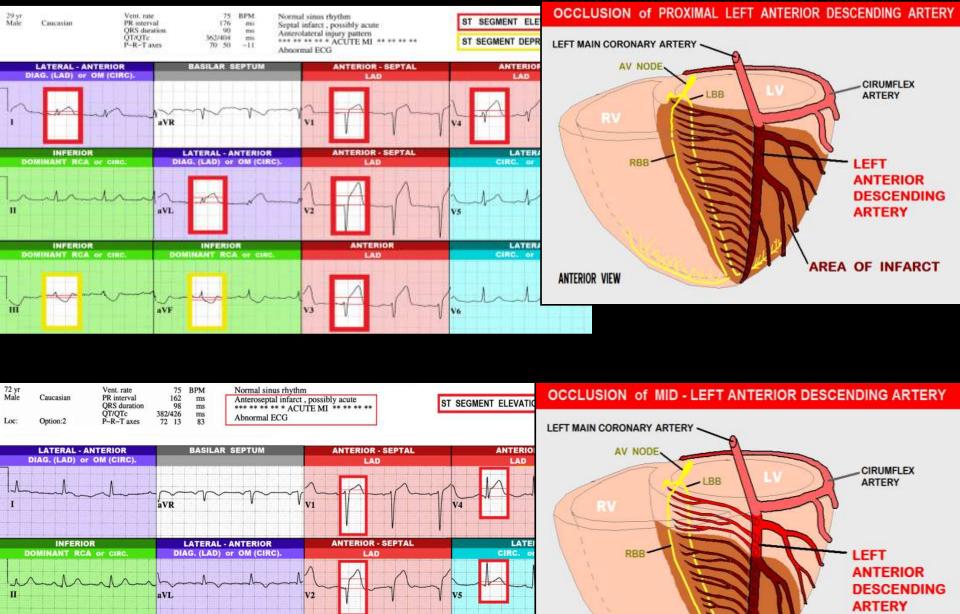
LEFT ANTERIOR

DESCENDING

ARTERY

#### OCCLUSION of PROXIMAL LEFT ANTERIOR DESCENDING ARTERY





LATE

ANTERIOR VIEW

AREA OF INFARCT

CIRC.

INFERIOR

DOMINANT RCA or CIRC.

aVF

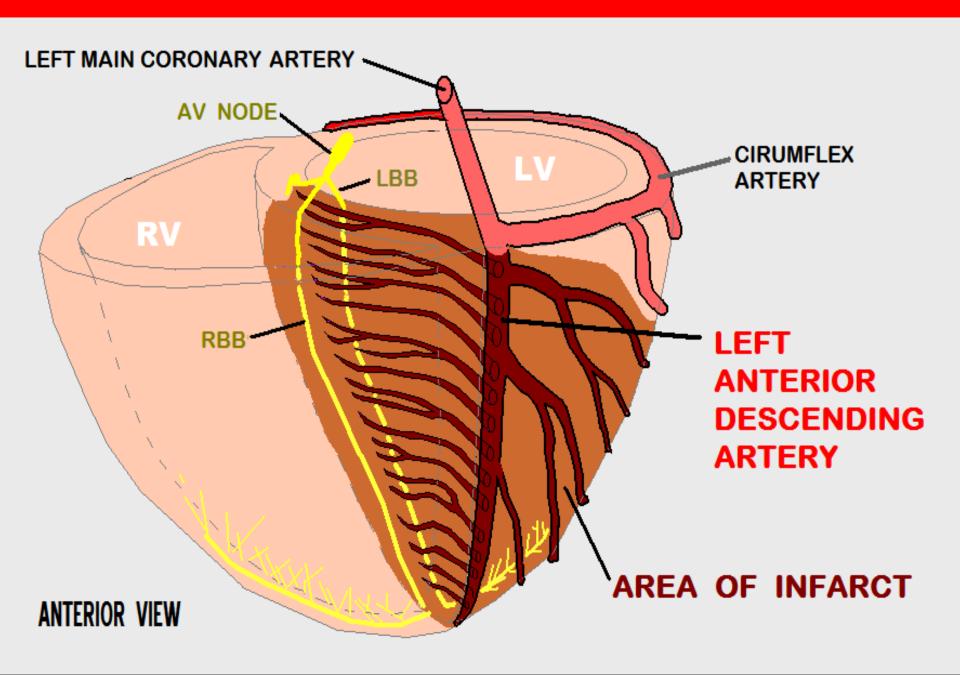
ANTERIOR

LAD

INFERIOR

DOMINANT RCA or CIRC

### OCCLUSION of PROXIMAL LEFT ANTERIOR DESCENDING ARTERY



# ANTICIPATED COMPLICATIONS of ANTERIOR-SEPTAL WALL STEMI & POSSIBLE INDICATED INTERVENTIONS: - CARDIAC ARREST BCLS / ACLS

- INTRA-AORTIC BALLOON PUMP

(use caution with fluid challenges

TRANSCUTANEOUS or

TRANSVENOUS PACING

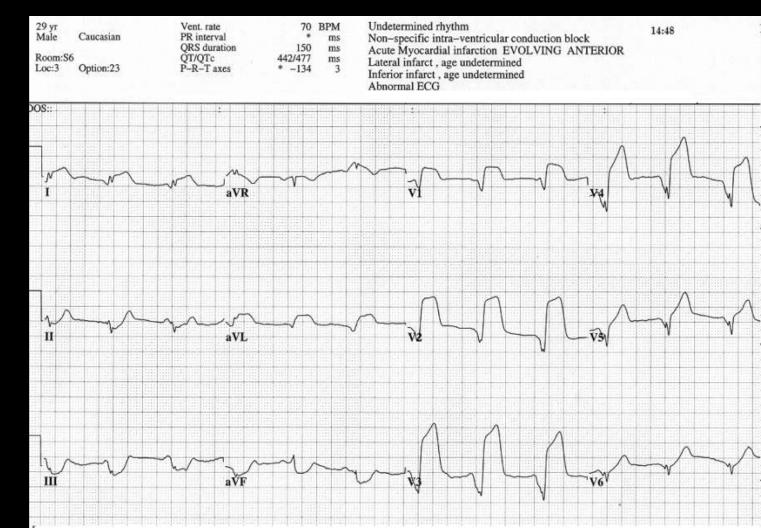
- CARDIAC DYSRHYTHMIAS (VT / VF)
- PUMP FAILURE with
CARDIOGENIC SHOCK
- DOPAMINE / DOBUTAMINE / LEVOPHED

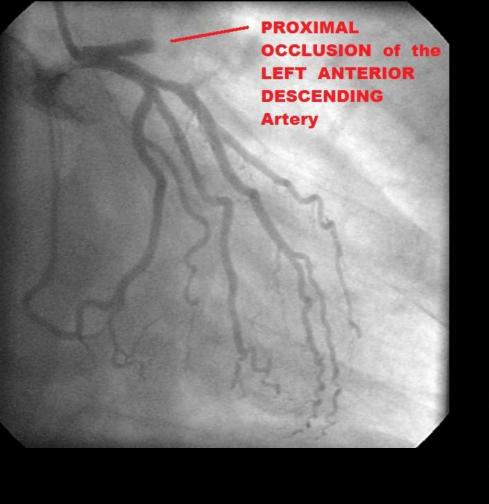
- PULMONARY EDEMA

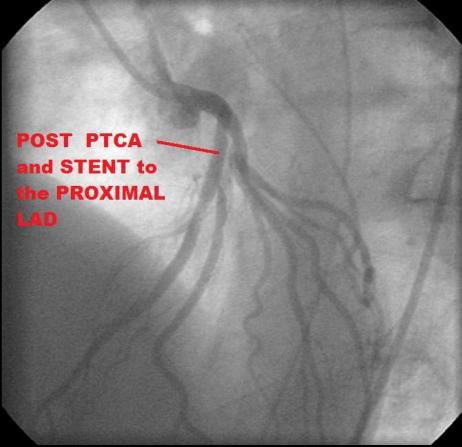
- CPAP
- ET INTUBATION
(use caution with dieuretics due to pump failure and hypotension)

- 3rd DEGREE HEART BLOCK - NOT RESPONSIVE TO ATROPINE 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?



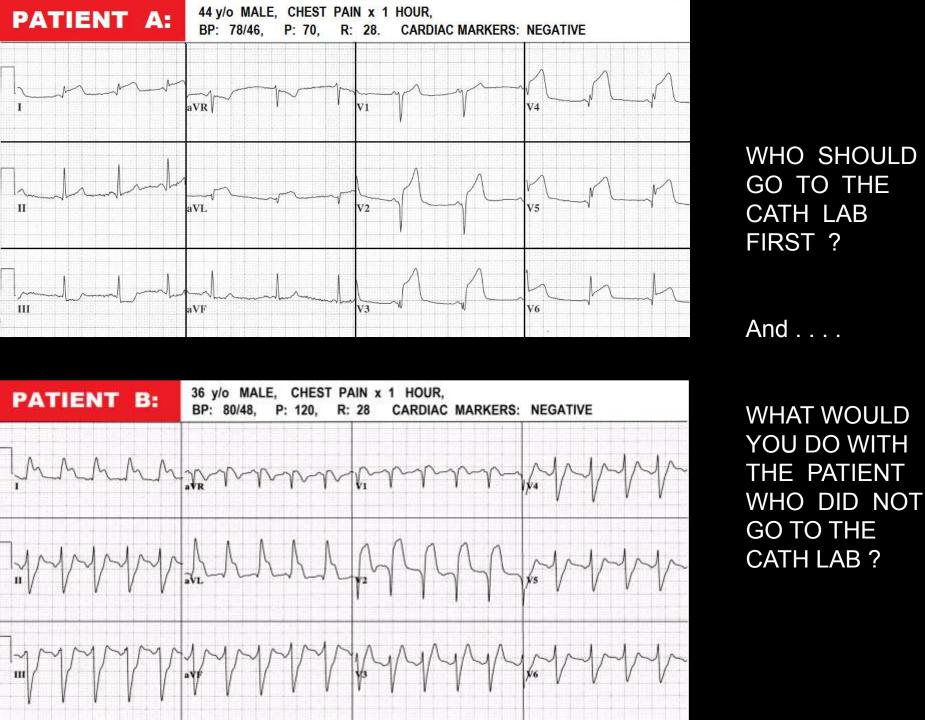


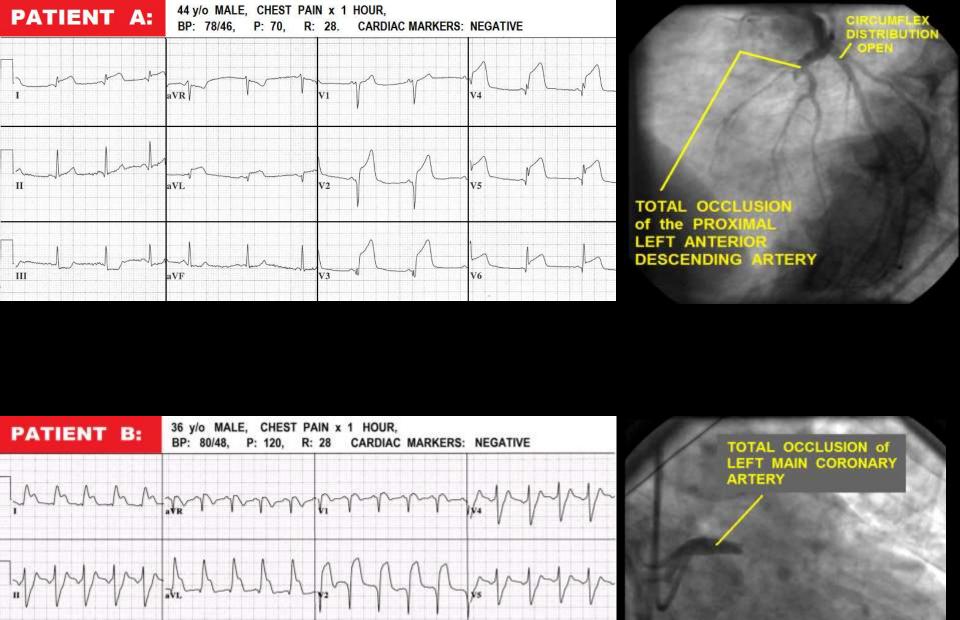


### CASE STUDY 4: CRITICAL DECISIONS SCENARIO

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

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





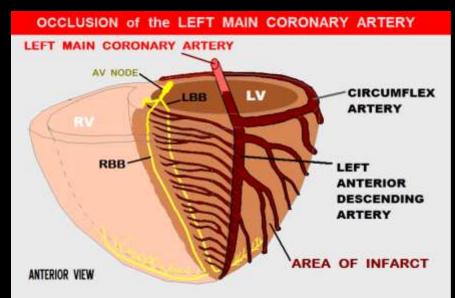
MANAMAN MANAMA

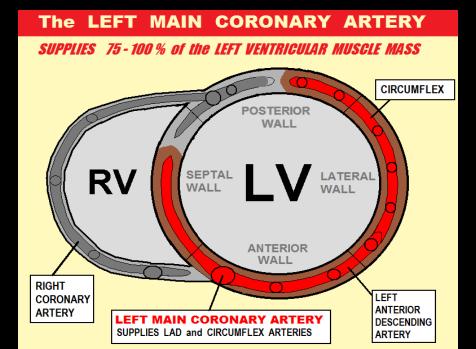
### PATIENT A:

# LEFT MAIN CORONARY ARTERY AV NODE AV NODE LEFT ANTERIOR DESCENDING ARTERY LEFT ANTERIOR DESCENDING ARTERY AREA OF INFARCT

# RIGHT CORONARY ARTERY LEFT ANTERIOR DESCEDING ARTERY ANTERIOR WALL ANTERIOR WA

#### PATIENT B:





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

- ☑ 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 [ 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

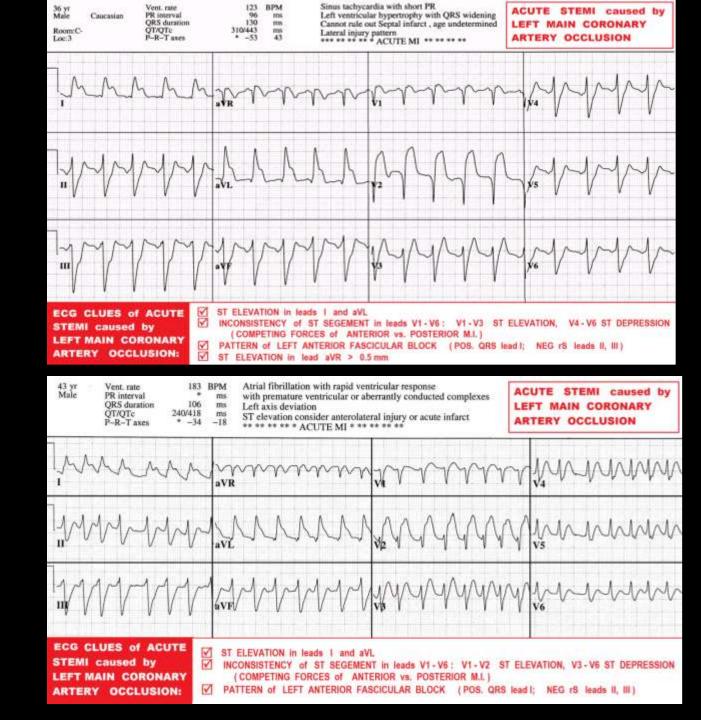
ECG CLUES of ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION:

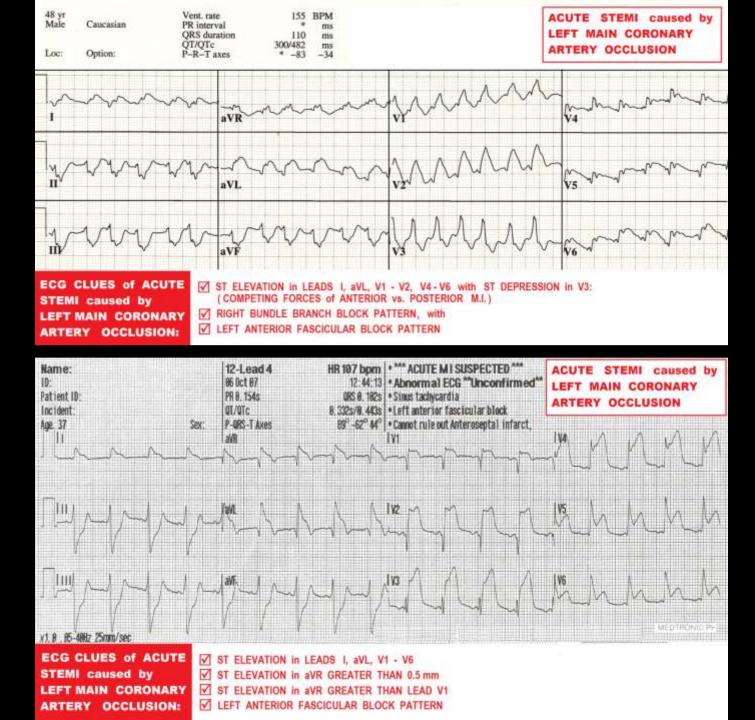
- ST ELEVATION in aVR (2 mm) > ST ELEVATION in V1 (1.5 mm)
- ST ELEVATION in V1 V3 with ST DEPRESSION in V4 V6 (ANTERIOR MI competing with POSTERIOR MI)
- LEFT ANTERIOR FASCICULAR BLOCK PATTERN

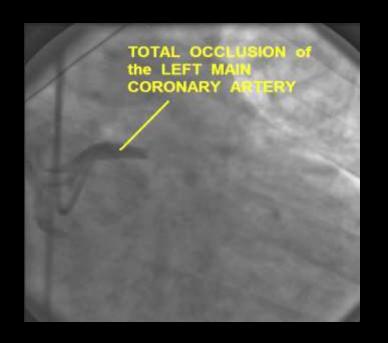
ST SEGMENT ELEVATION

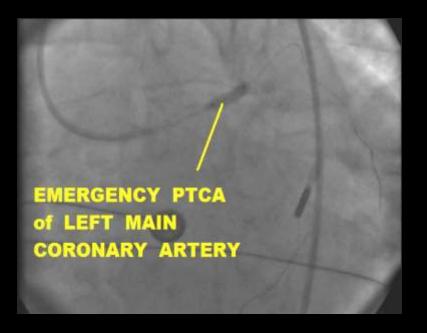
ST SEGMENT DEPRESSION



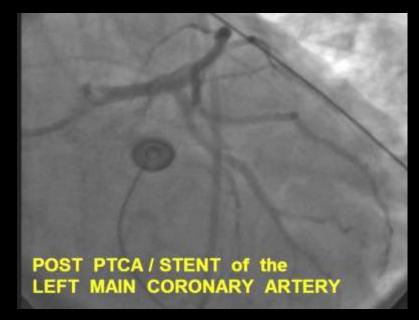








Despite the dismal mortality rate associated with STEMI from total LMCA occlusion, this patient survived and was later discharged. His EF is estimated at approximately 30%. He received an ICD, and is currently stable.



### CASE STUDY 4: CRITICAL DECISIONS SCENARIO

CONCLUSIONS:

QUESTION 1: WHICH PATIENT SHOULD BE TAKEN FIRST FOR IMMEDIATE CARDIAC

CATHETERIZATION for EMERGENCY PCI?

ANSWER: PATIENT B was taken emergently to the Cardiac Cath Lab - both the ED physician

and the Interventional Cardiologist correctly identified the EKG patterns

of LMCA occlusion.

QUESTION 2: WHAT COURSE OF ACTION SHOULD BE TAKEN WITH THE PATIENT NOT CHOSEN

TO BE SENT TO THE CATH LAB FIRST?

ANSWER: PATIENT A received thrombolytic therapy in the ED. It was determined that

THROMBOLYTIC THERAPY would achieve the FASTEST ROUTE to REPERFUSION --

-- by at least 60 minutes.

### 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.

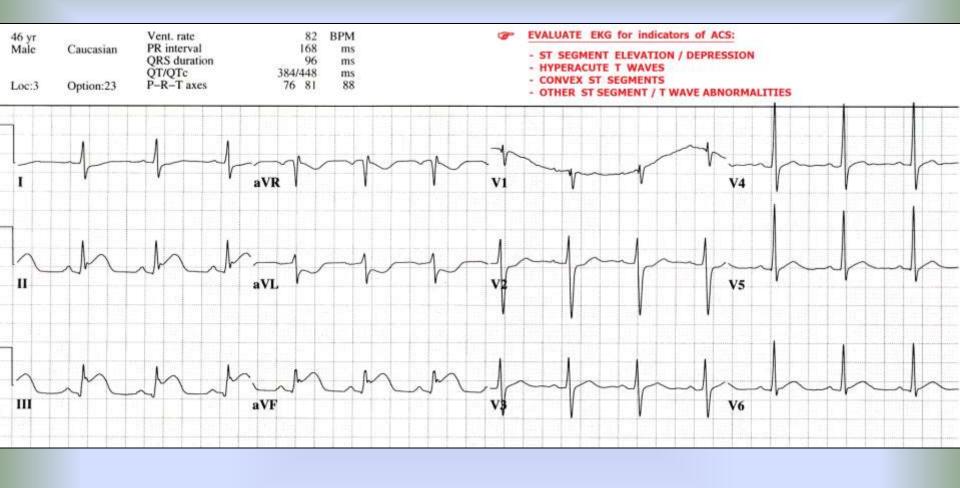
### RISK FACTOR PROFILE:

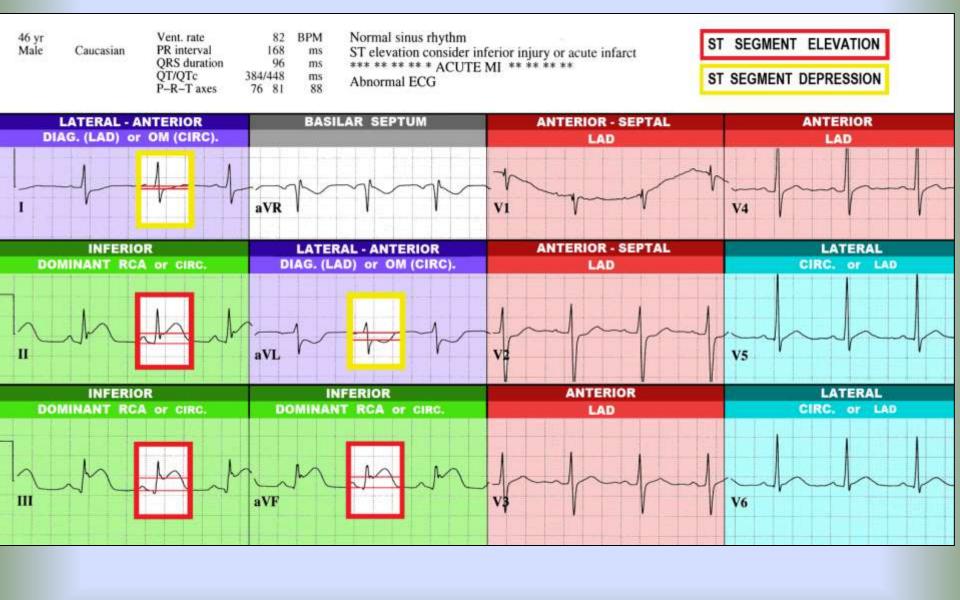
- **●\*\* CURRENT CIGARTTE SMOKER x 18 YEARS**
- **HYPERTENSION**
- MHIGH LDL CHOLESTEROL

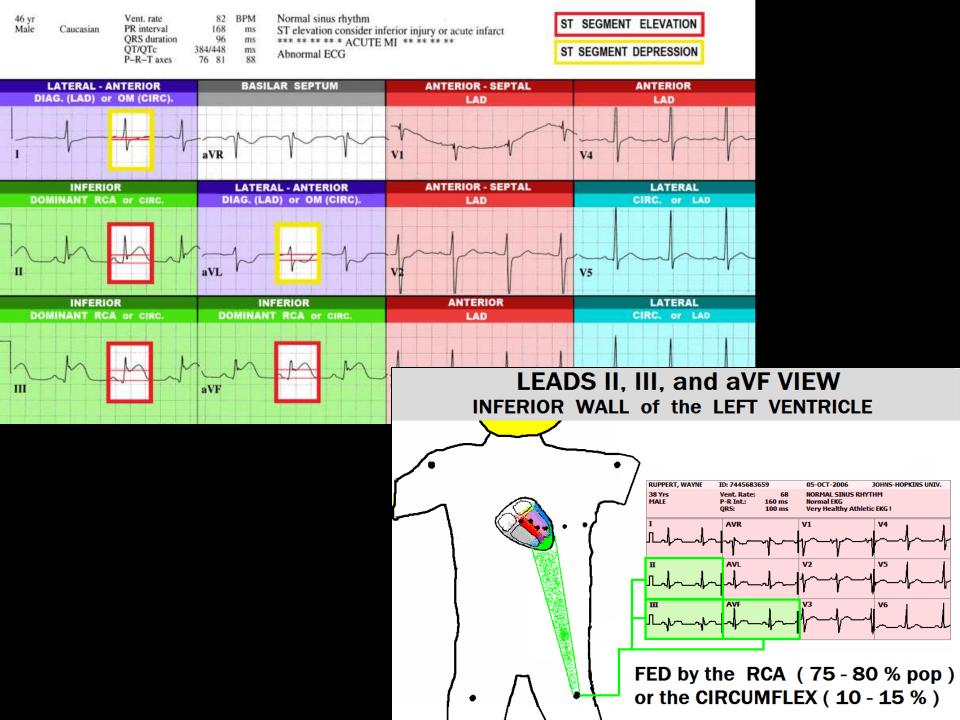
PHYSICAL EXAM: Patient is alert & oriented x 4, skin warm, dry, color normal. Non-anxious Lungs clear, normal S1, S2. No JVD, No ankle edema.

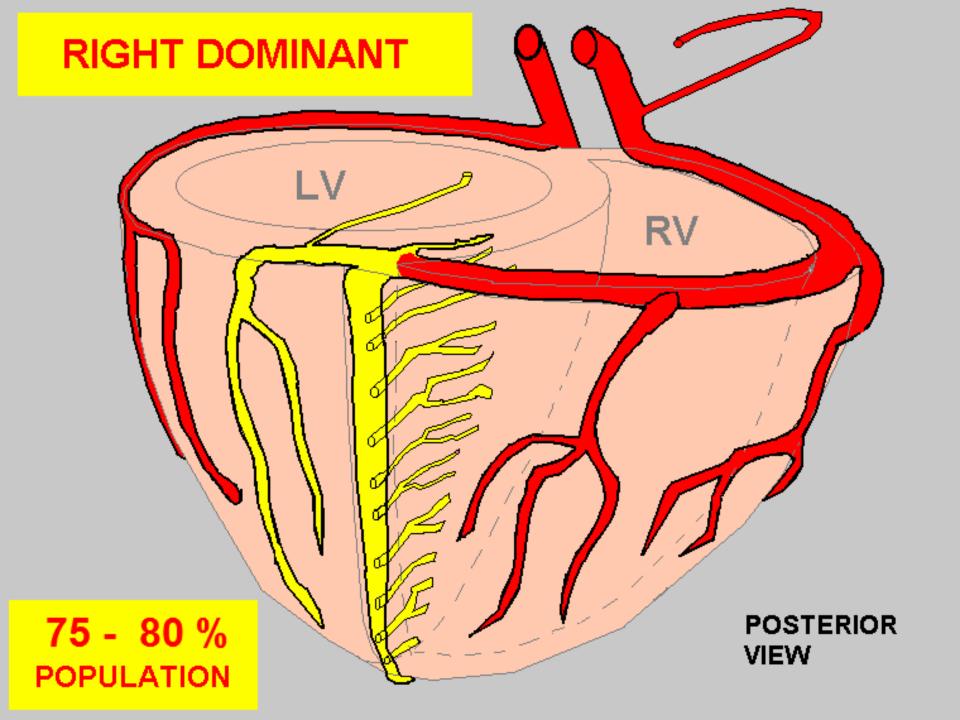
VITAL SIGNS: BP: 136/88 P: 88 R: 20 SAO2: 100% on 4 LPM O2

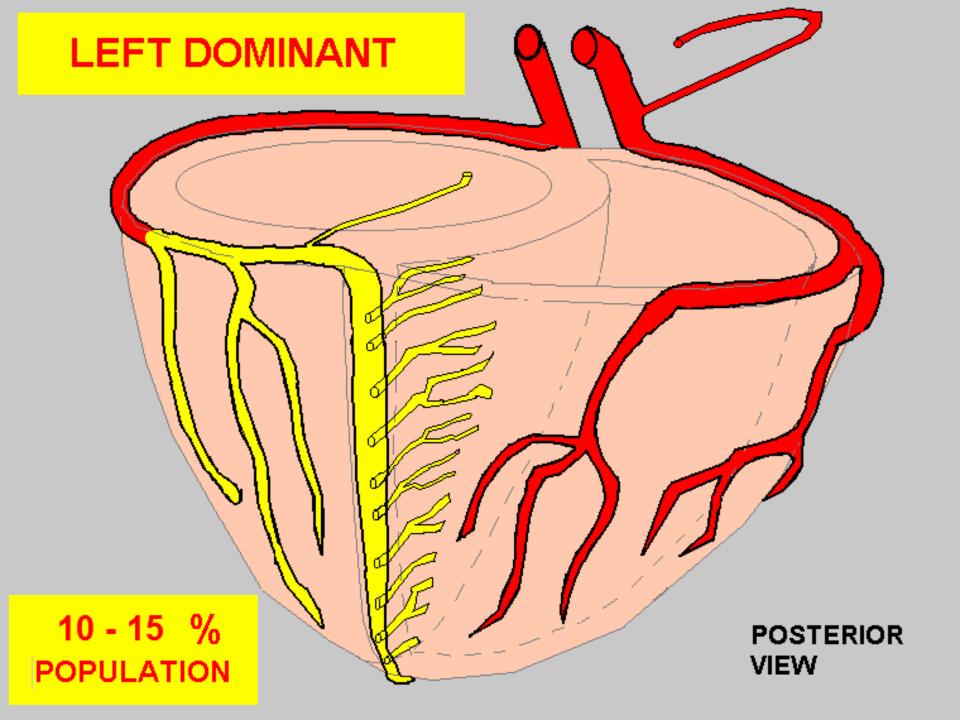
LABS: TROPONIN: < .04















### 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

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

- CARDIAC ARREST BCLS / ACLS

**ACLS** (antiarrhythmics) - CARDIAC DYSRHYTHMIAS (VT / VF)

- SINUS BRADYCARDIA (follow ACLS and/or UNIT protocols)

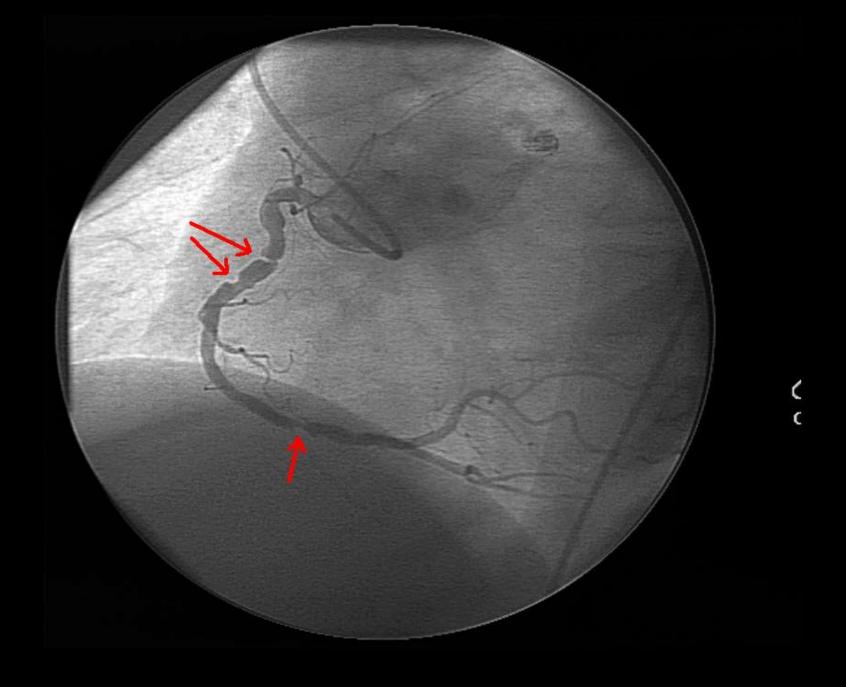
ATROPINE 0.5mg, REPEAT as needed UP TO 3mg. - HEART BLOCKS (1st, 2nd & 3rd Degree HB) ATROPINE 0.5mg, REPEAT as needed UP TO 3mg, Transcutaneous Pacing, (follow ACLS and/or UNIT protocols) the Right Ventricle.

- The standard 12 Lead ECG does NOT view - RIGHT VENTRICULAR MYOCARDIAL INFARCTION - You must do a RIGHT-SIDED ECG to see if RV MI is present. - Do NOT give any Inferior Wall STEMI patient NITRATES or DIURETICS until RV MI has been RULED OUT.

- POSTERIOR WALL MI presents on the 12 Lead ECG as ST DEPRESSION in Leads V1 -

ON THIS FCG.

- POSTERIOR WALL INFARCTION V3. - POSTERIOR WALL MI is NOT PRESENT



### A standard

## 12 LEAD EKG

Does NOT show the

# RIGHT VENTRICLE

# To see the RIGHT VENTRICLE ...

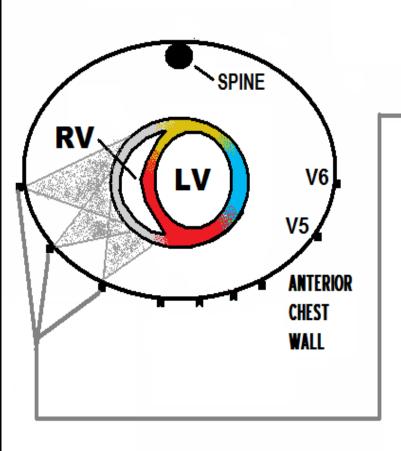
... such as in cases of

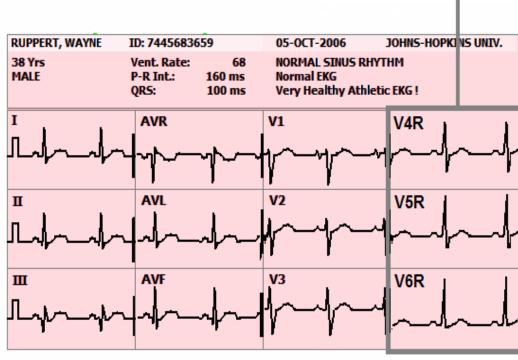
# INFERIOR WALL M.I.

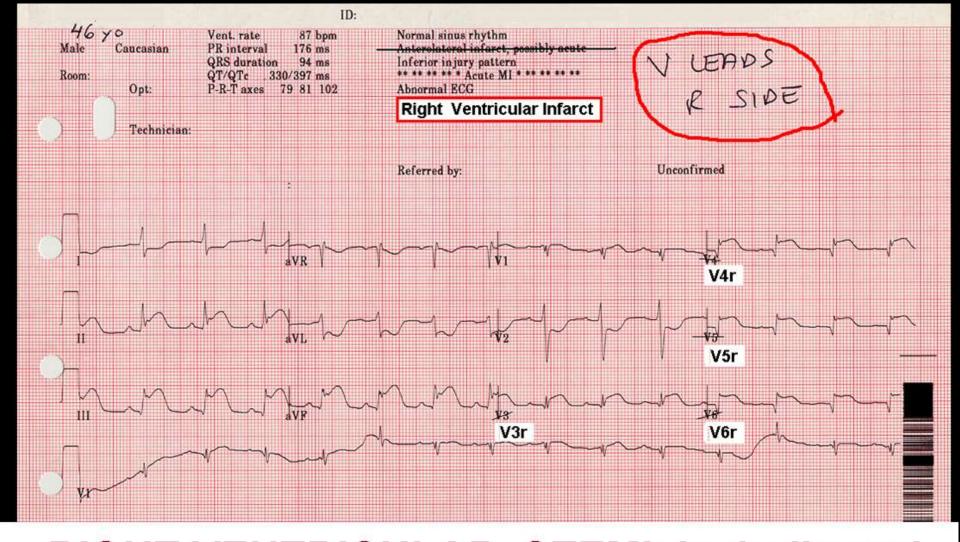
You must do a

RIGHT - SIDED EKG!!

# V4R - V6R VIEW THE RIGHT VENTRICLE







RIGHT VENTRICULAR STEMI is indicated when ST Segment Elevation of 0.5mv is present.

## IN EVERY CASE of

# INFERIOR WALL STEMI

You must first *RULE OUT*RIGHT VENTRICULAR MI

BEFORE giving any:

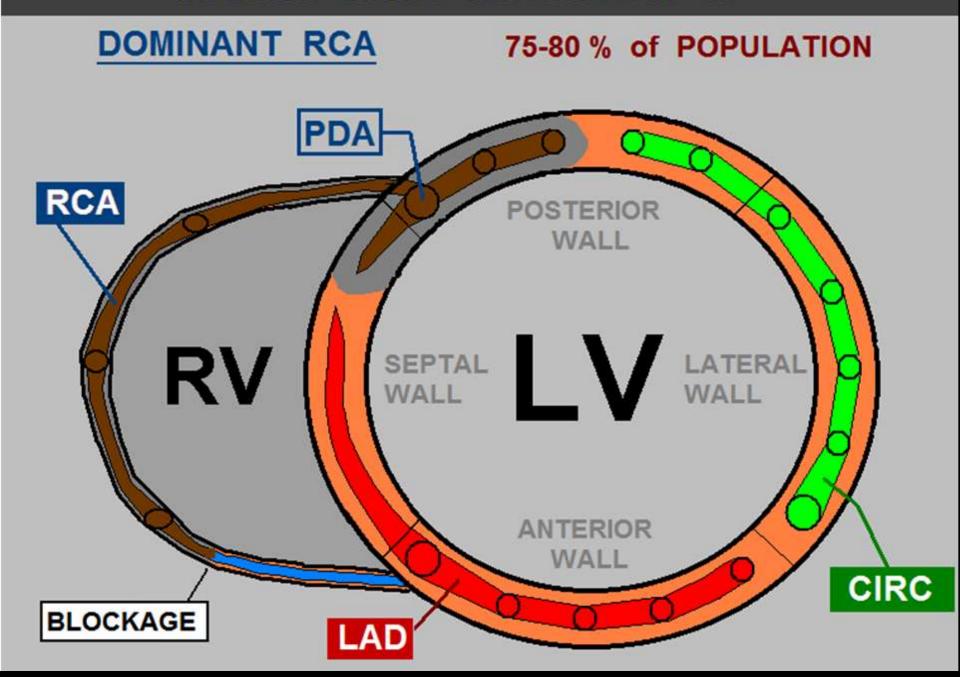
- NITROGLYCERIN
- Diuretics

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

# They precipitate SEVERE HYPOTENSION

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

#### INFERIOR - RIGHT VENTRICULAR MI



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

- CARDIAC ARREST	BCLS / ACLS
- CARDIAC DYSRHYTHMIAS (VT / VF)	ACLS (antiarrhythmics)

ATROPINE 0.5mg, REPEAT as needed UP TO 3mg.

ATROPINE 0.5mg, REPEAT as needed UP TO 3mg, Transcutaneous Pacing, (follow ACLS and/or UNIT

(follow ACLS and/or UNIT protocols)

- NITRATES and DIURETICS are CONTRA-

- TREAT HYPOTENSION WITH FLUIDS. (It is Not uncommon to give 500-2000ml of

- POSTERIOR WALL MI presents on the 12

- POSTERIOR WALL MI is NOT PRESENT

Lead ECG as ST DEPRESSION in Leads V1 -

NORMAL SALINE to stabilize BP.

protocols)

**V3**.

ON THIS ECG.

INDICATED.

- SINUS BRADYCARDIA

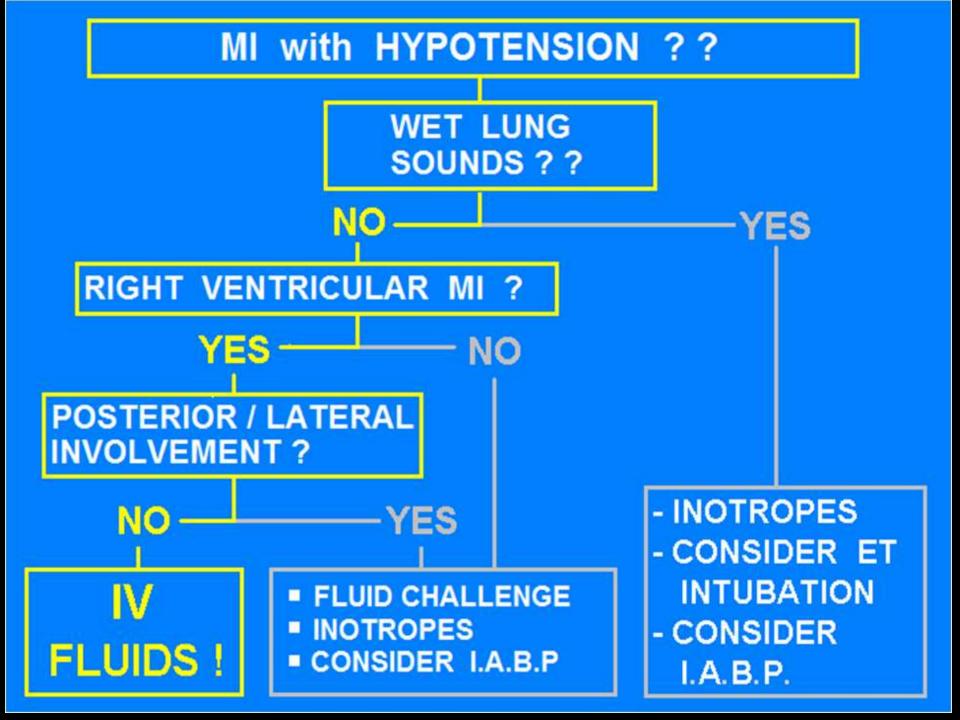
**INFARCTION** 

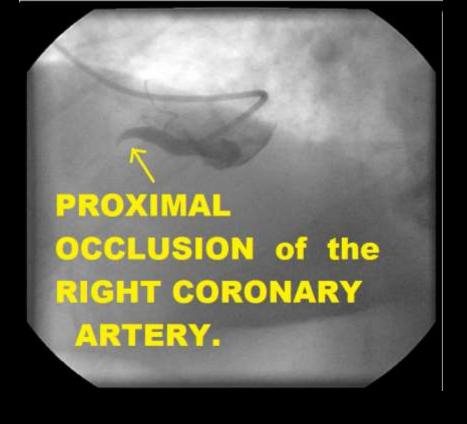
- HEART BLOCKS (1st, 2nd & 3rd Degree HB)

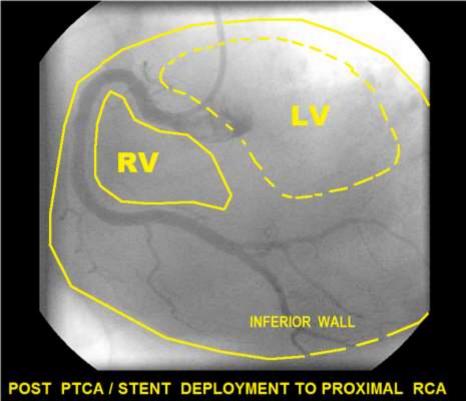
- RIGHT VENTRICULAR MYOCARDIAL

- POSTERIOR WALL INFARCTION

# If this patient becomes HYPOTENSIVE . . . . .







#### CASE STUDY 9 - STEMI

#### CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

#### RISK FACTOR PROFILE:

- **CIGARETTE SMOKER**
- **HYPERTENSION**

PULSE:

HIGH LDL CHOLESTEROL

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

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

LABS: TROPONIN: < .04

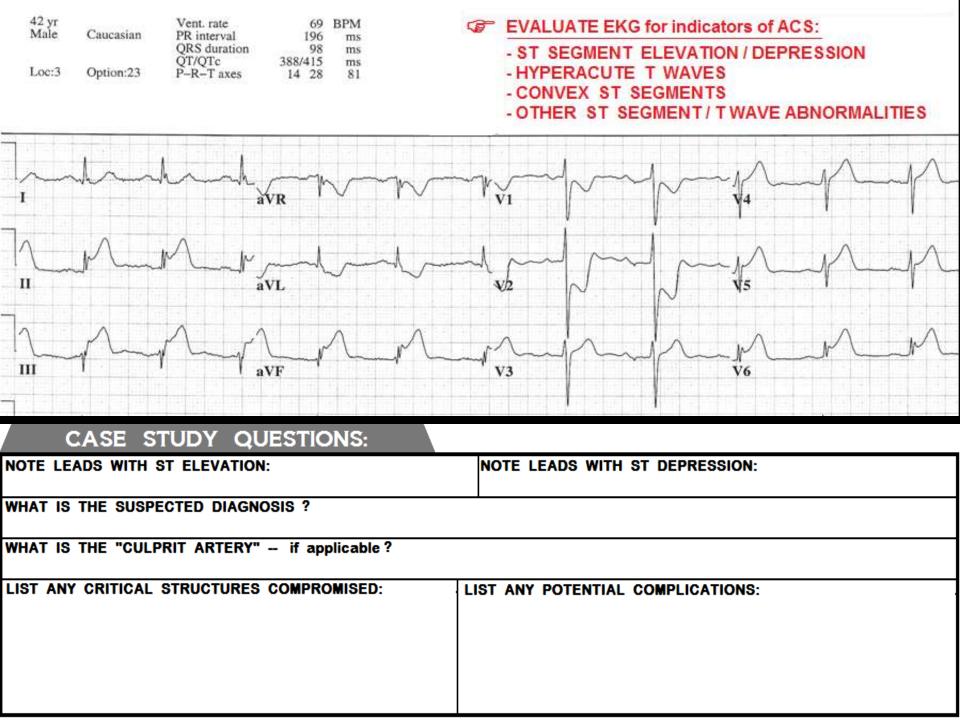
#### SHOCK ASSESSMENT

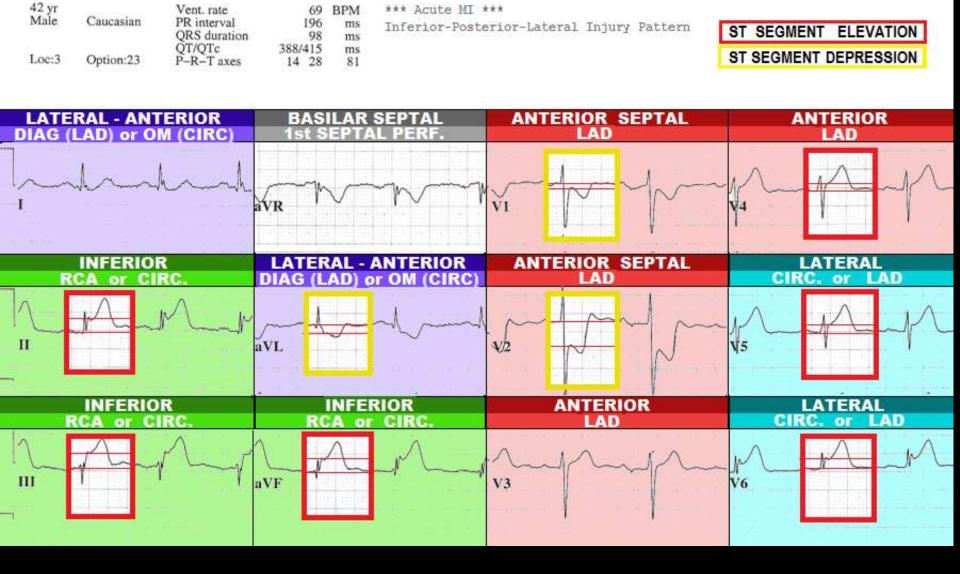
LOC:	ANXIOUS	AWAKE
	RESTLESS	ALERT &
	LETHARGIC	ORIENTED
	UNCONSCIOUS	A production of the second of the second
	UNCONSCIOUS	Control of the Control of the Control

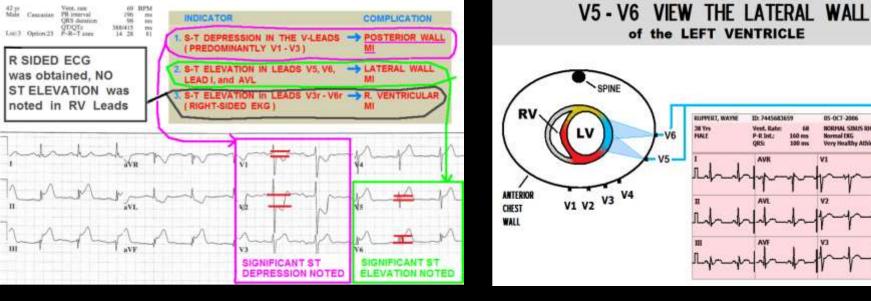
BREATHING: TACHYPNEA NORMAL

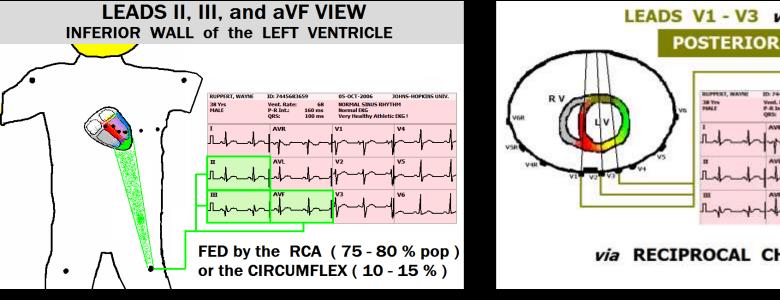
WEAK / THREADY STRONG
TOO FAST or SLOW

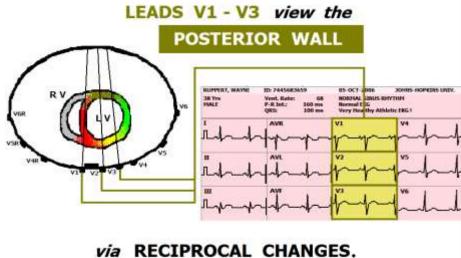
STATUS: SHOCK NORMAL





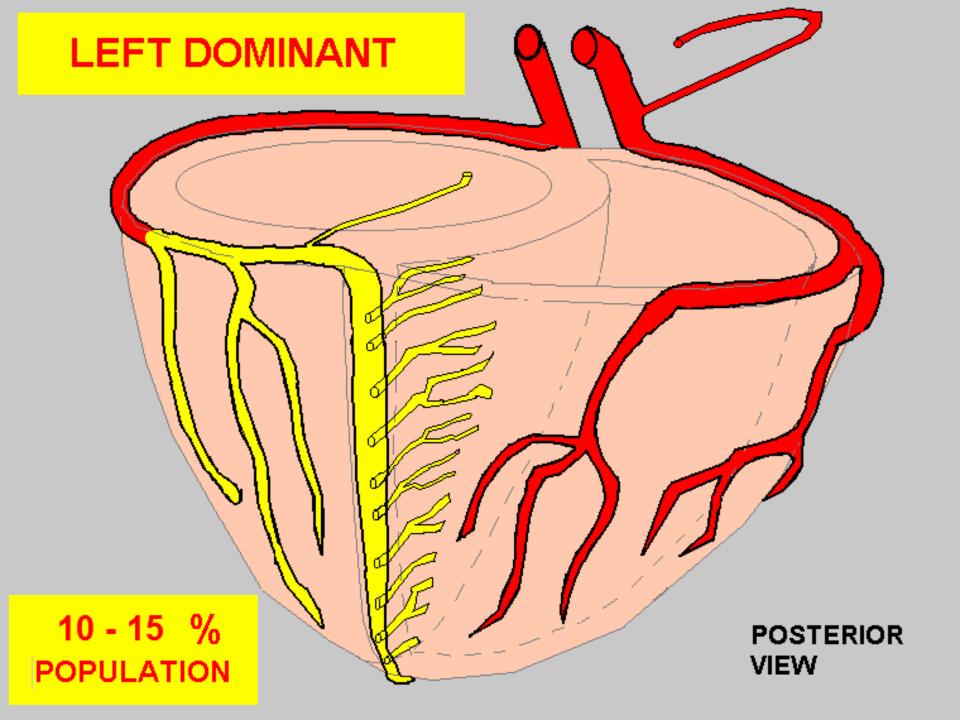


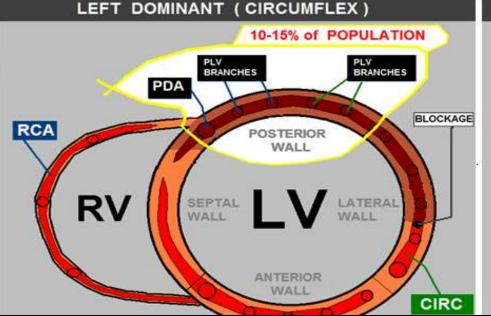


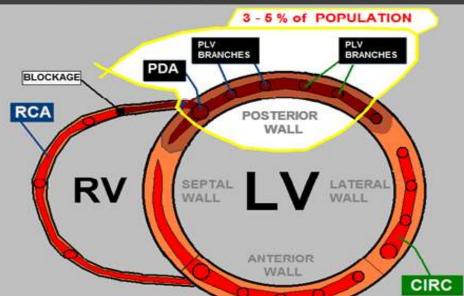


HORMAL SINUS RHYTHIN

Normal EKG Very Healthy Attrictic HIG I



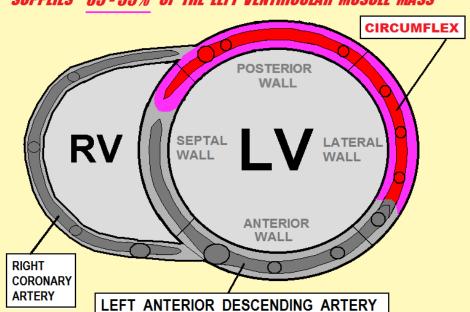




"EXTREME RIGHT DOMINANT" RCA

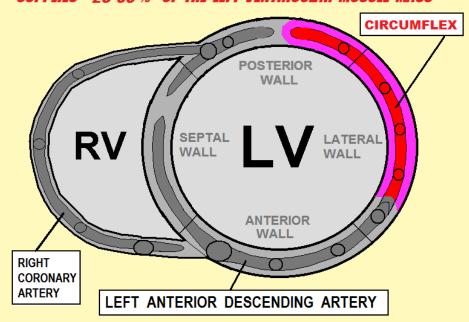
#### The DOMINANT CIRCUMFLEX ARTERY...

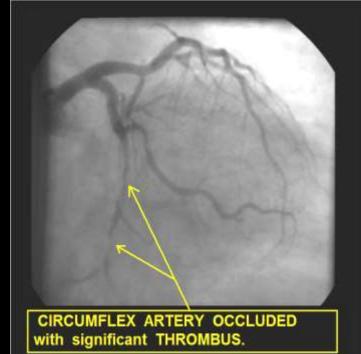
SUPPLIES 35 - 55% OF THE LEFT VENTRICULAR MUSCLE MASS

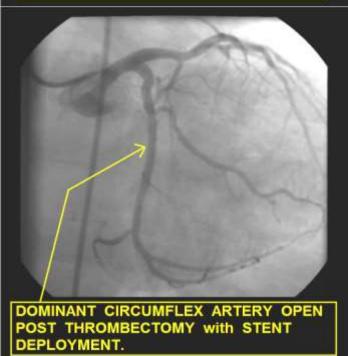


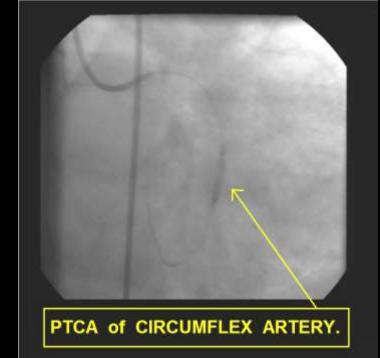
#### The NON - DOMINANT CIRCUMFLEX ARTERY

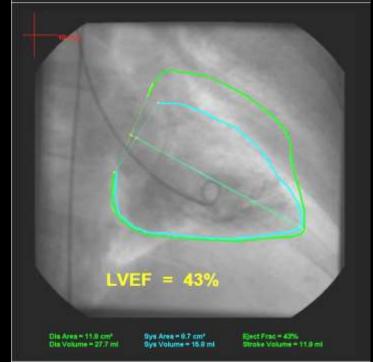
SUPPLIES 25-30 % OF THE LEFT VENTRICULAR MUSCLE MASS











#### CASE STUDY SUMMARY ST ELEVATION: ST DEPRESSION: II, III, aVF, V5, V6 V1 - V3. POSSIBLY I and aVL SUSPECTED DIAGNOSIS: **ACUTE INFERIOR - POSTERIOR - LATERAL MI** SUSPECTED "CULPRIT ARTERY" (if applicable): OCCLUSION of DOMINANT CIRCUMFLEX ARTERY IMMEDIATE CONCERNS FOR ALL STEMI PATIENTS: ■ BE PREPARED TO MANAGE SUDDEN CARDIAC ARREST (PRIMARY V-FIB/V-TACH, BRADYCARDIAS/HEART BLOCKS) STAT REPERFUSION THERAPY: THROMBOLYTICS vs. CARDIAC CATHETERIZATION and PCI CONSIDER NEEDS FOR ANTI-PLATELET and ANTI-COAGULATION THERAPY CRITICAL STRUCTURES COMPROMISED: POTENTIAL COMPLICATIONS: POSSIBLE CRITICAL INTERVENTIONS: 30 - 55% of LV INOTROPIC AGENTS POSSIBLE SEVERE LV MUSCLE MASS PUMP FAILURE **ET INTUBATION** I.A.B.P. INSERTION SINUS BRADYCARDIA / SINUS ----ATROPINE SA NODE ARREST TRANSCUTANEOUS PACING ATROPINE AV NODE HEART BLOCKS TRANSCUTANEOUS PACING

ACUTE PAPILLARY MUSCLE -

REGURGITATION (7 - 10 DAYS)

TEAR and MITRAL VALVE

INOTROPIC AGENTS

**EMERGENCY SURGERY** 

DIEURETICS

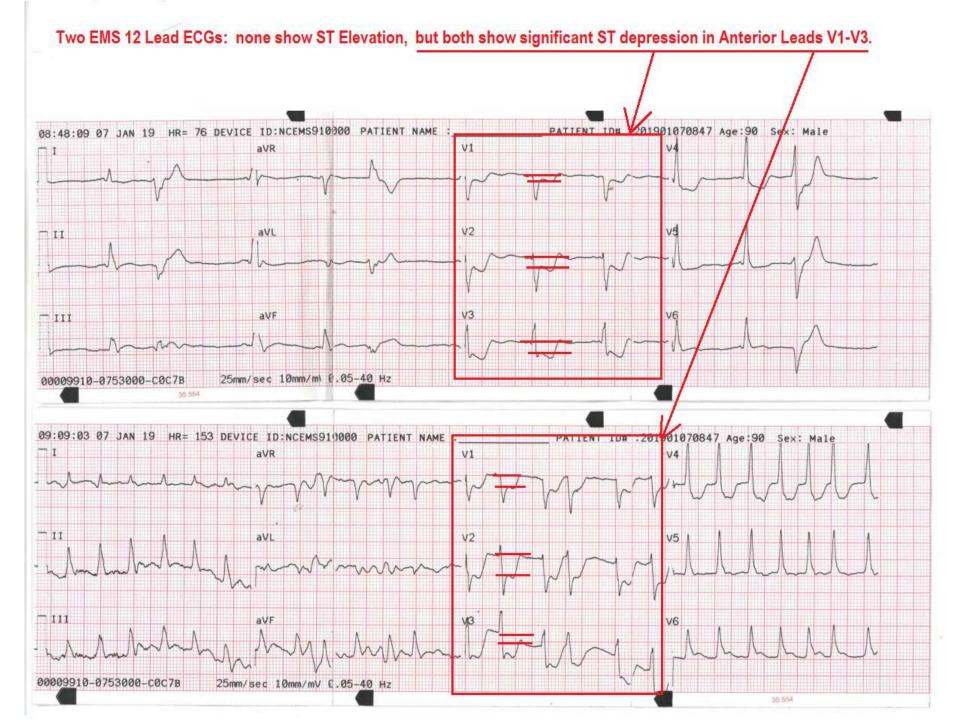
SIGNIFICANT AMOUNT of .

INSERTION to BASE of LV

PAPILLARY MUSCLE

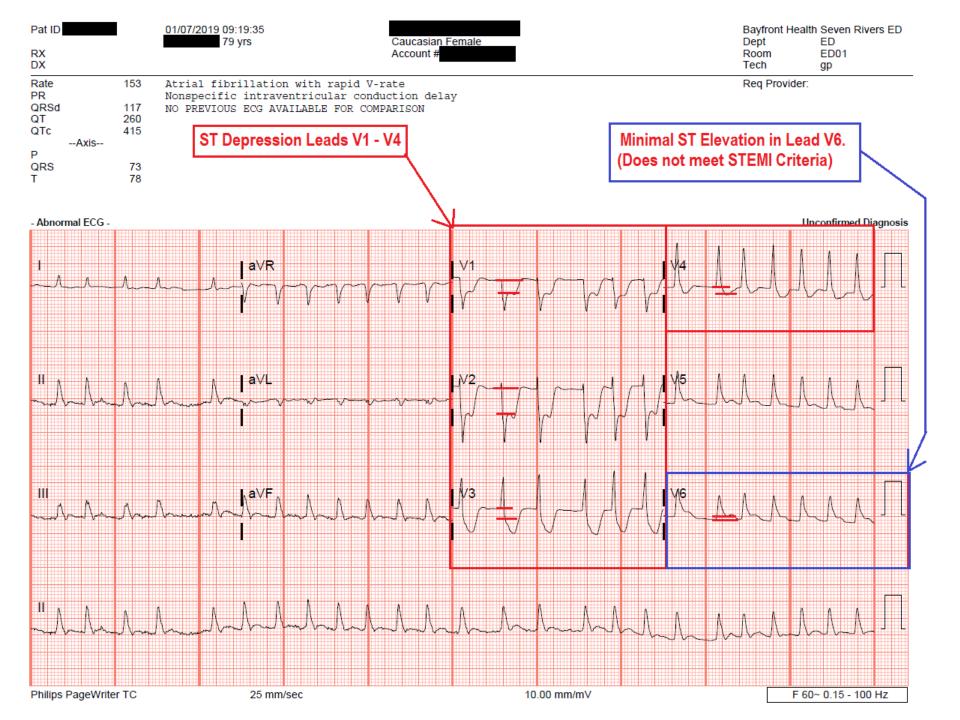
# Case Study- January 2019

- 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.

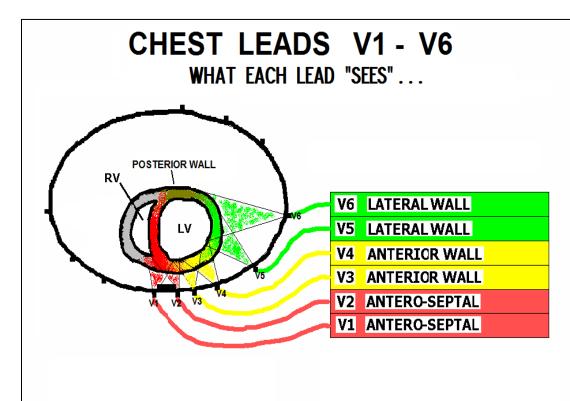


# Causes of ST Depression V1-V4

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

#### Posterior Wall STEMI....

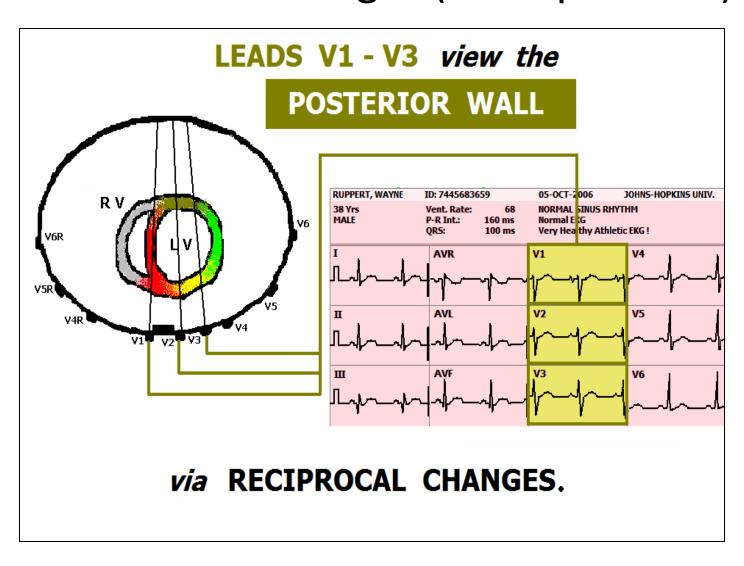
 Does not show ST elevation on standard 12 lead ECG because NONE of the 12 leads view the Posterior Wall directly....



#### Posterior Wall STEMI....

- Often shows NO ST Elevation on the standard 12 Lead ECG.
- Will show up on standard 12 Lead ECG as "ST Depression" (Reciprocal) in Leads V1-V3 (sometimes V4-V6, too).

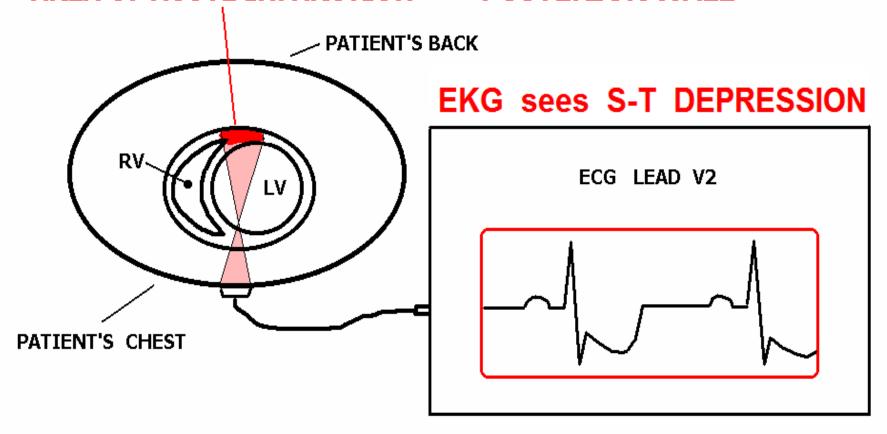
# V1-V3 see the Posterior Wall ONLY through RECIPROCAL changes (ST Depression)



#### **HOW EKG VIEWS RECIPROCAL CHANGES**

**EXAMPLE:** 

AREA OF ACUTE INFARCTION - POSTERIOR WALL



## Posterior Wall STEMI....

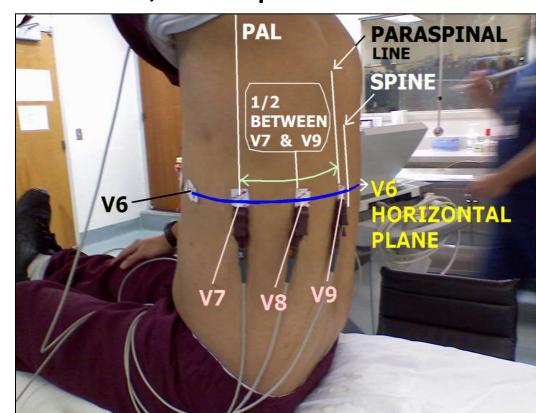
 To see ST Elevation from a Posterior Wall STEMI, you must place ECG leads on the patient's back...

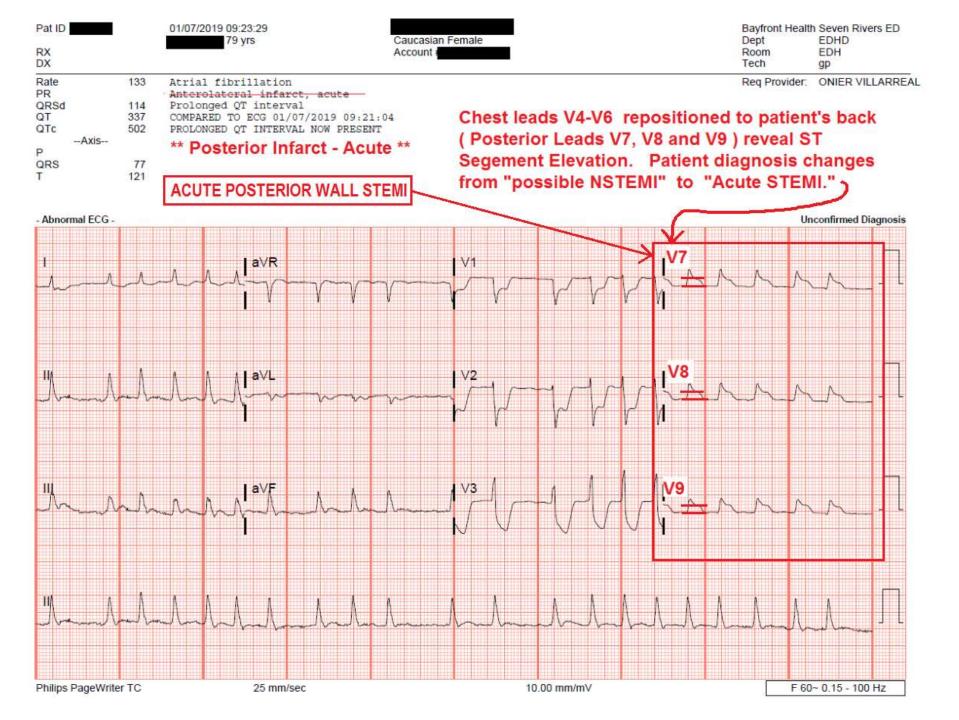
## Continued Exam in the ED....

 Upon noting ST Depression in Anterior Leads V1-V4, ED Paramedic Gary Polizzi place three leads on the patient's back. Gary used the lead wires for V4, V5 and V6, with placement

as shown here:

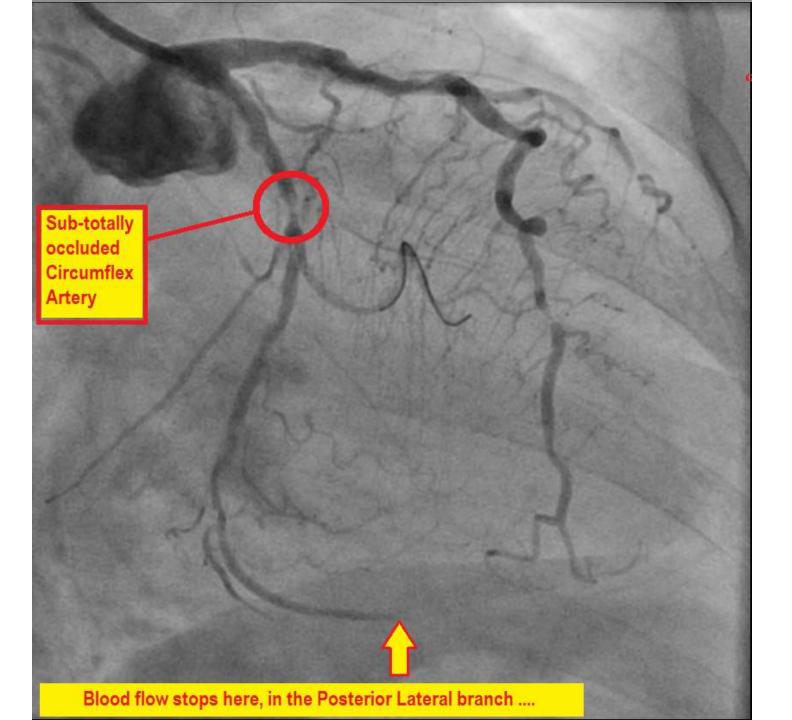
 The "Posterior Lead ECG" is seen on the next slide.....

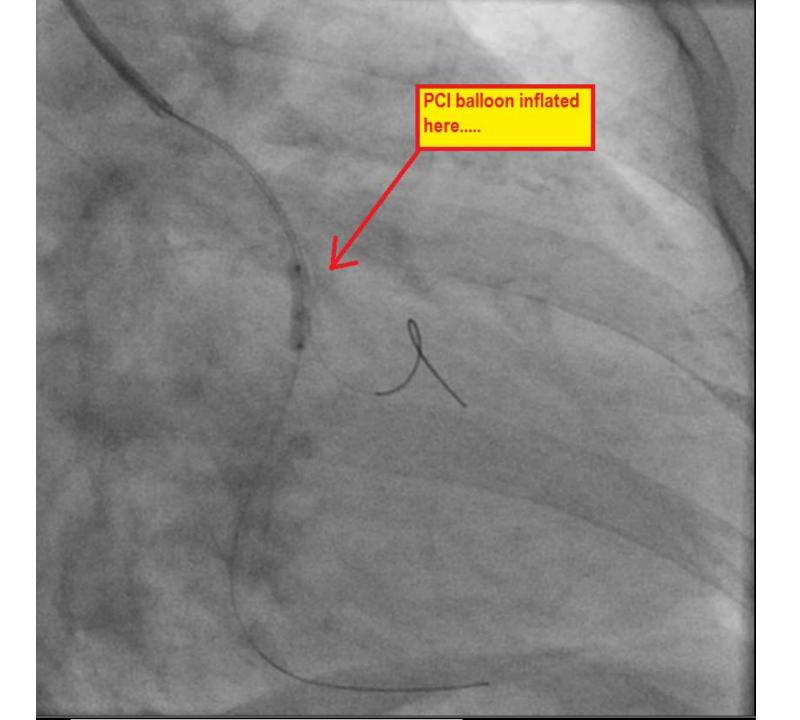


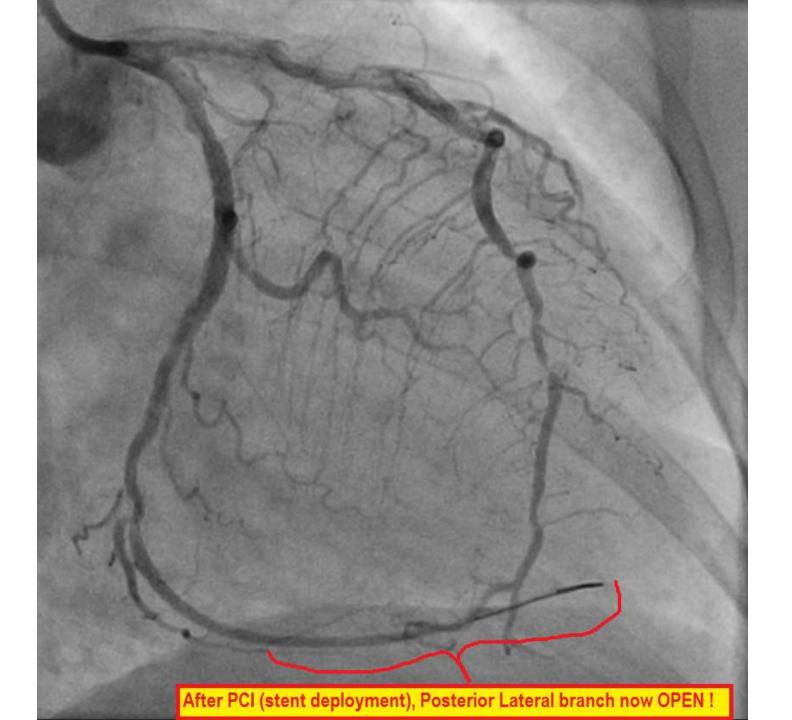


#### STEMI Alert!

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......

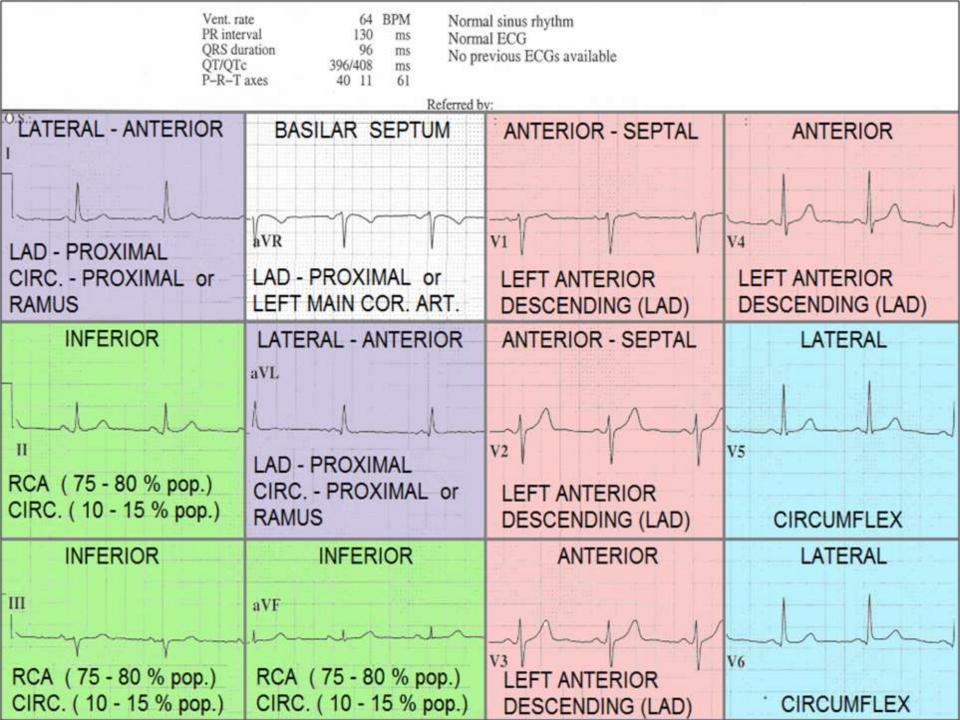






#### **SUMMARY**

- 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!



# Evaluating the ECG for ACS:

#### STEP 1 - EVALUATE WIDTH OF QRS: WIDE ( > 120 ms) NORMAL (< 120 ms) OF SUPRAVENTRICULAR ORIGIN --DETERMINE QRS MORPHOLOGY: LEFT BUNDLE BRANCH RIGHT BUNDLE BRANCH **BLOCK PATTERN BLOCK PATTERN EVALUATE FOR USE CAUTION --**EVALUATE FOR DO NOT RELY ON ST ELEVATION ST DEPRESSION ST ELEVATION ST DEPRESSION IN USUAL MANNER IN USUAL MANNER AS A MARKER OF ACS. -IS ROUTINELY SEEN IN WIDE QRS COMPLEX WIDE QRS COMPLEX RHYTHMS (both L and R RHYTHMS WITH LBBB **BBB PATTERNS) OFTEN** PATTERN. FOLLOW AHA CAUSE: DEPRESSION of CRITERIA (page 109) FOR J POINTS. ST SEGMENTS. DIAGNOSIS OF STEMI IN & INVERSION OF T WAVES. PRESENCE OF LBBB.

# Wide QRS present: QRSd > 120ms

 Determine RIGHT vs. LEFT Bundle Branch Block Pattern

# Simple "Turn Signal Method" . . .

THE "TURN SIGNAL METHOD" for identifying BUNDLE BRANCH BLOCK

V1

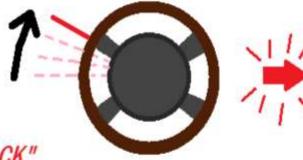
**USE LEAD V1 for this technique** 

To make a RIGHT TURN

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

THINK:

"QRS points UP = RIGHT BUNDLE BRANCH BLOCK"



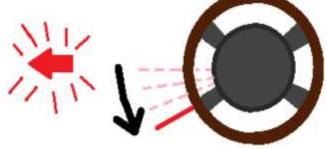
٧1



To make a LEFT TURN

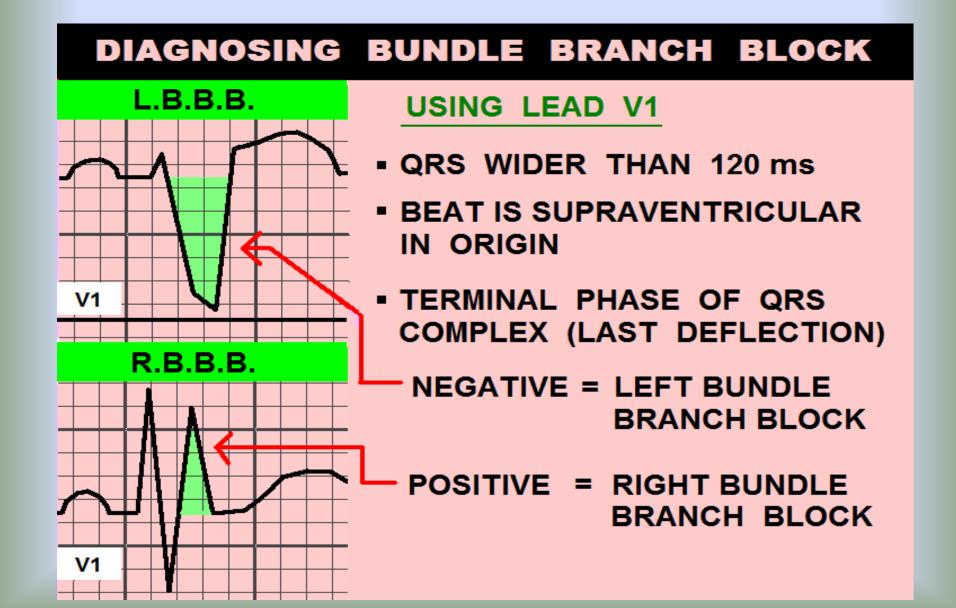
you push the turn signal lever DOWN . . . .

THINK:

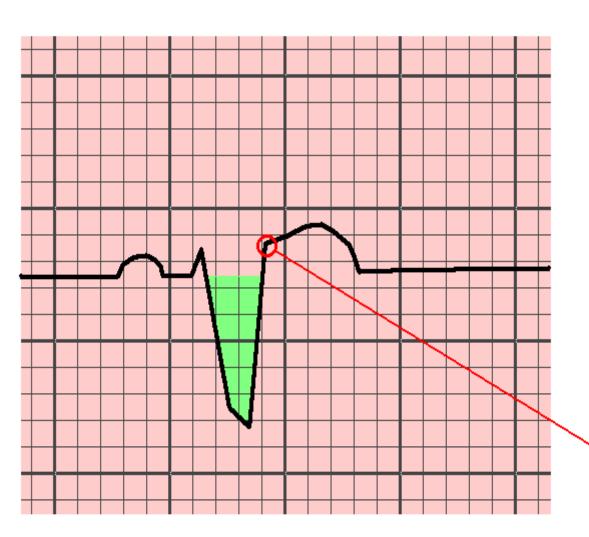


"QRS points DOWN = LEFT BUNDLE BRANCH BLOCK"

#### "Terminal Phase of QRS Method"...

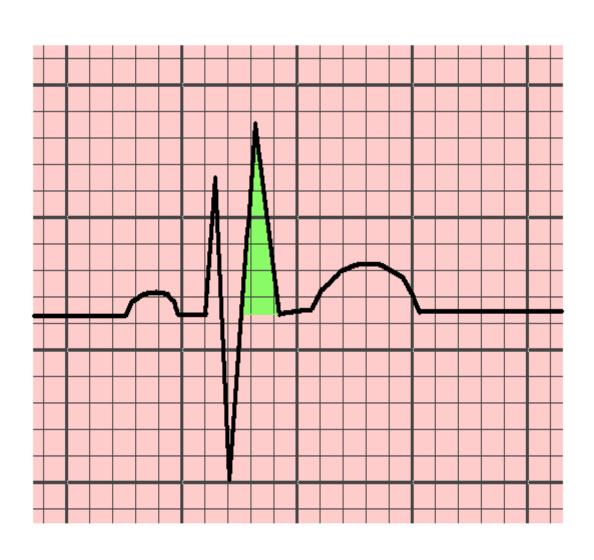


#### DIAGNOSING LBBB IN LEAD V1:



- QRS GREATER THAN 120 ms (.12)
- EVIDENCE THAT THIS IS NOT VENTRICULAR BEAT
- TERMINAL PHASE (LAST PART) OF QRS COMPLEX IS NEGATIVE DEFLECTION
- · S-T SEGMENTS ARE NORMALLY ALWAYS ELEVATED!

#### **DIAGNOSING RBBB IN LEAD V1:**



WIDER THAN
 120 ms (.12)

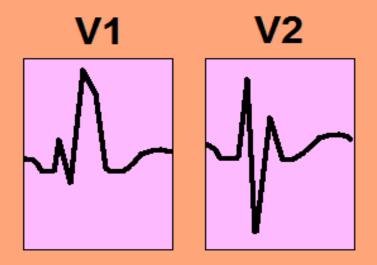
(or 3 little boxes)

 TERMINAL PHASE (LAST PART) OF QRS COMPLEX IS POSITIVE DEFLECTION

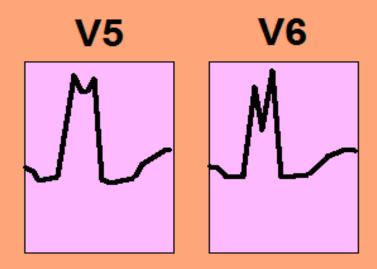
#### DIAGNOSING BUNDLE BRANCH BLOCK

USING LEADS V1, V2, and V5, V6:

#### LOCATING RsR' or RR' COMPLEXES:

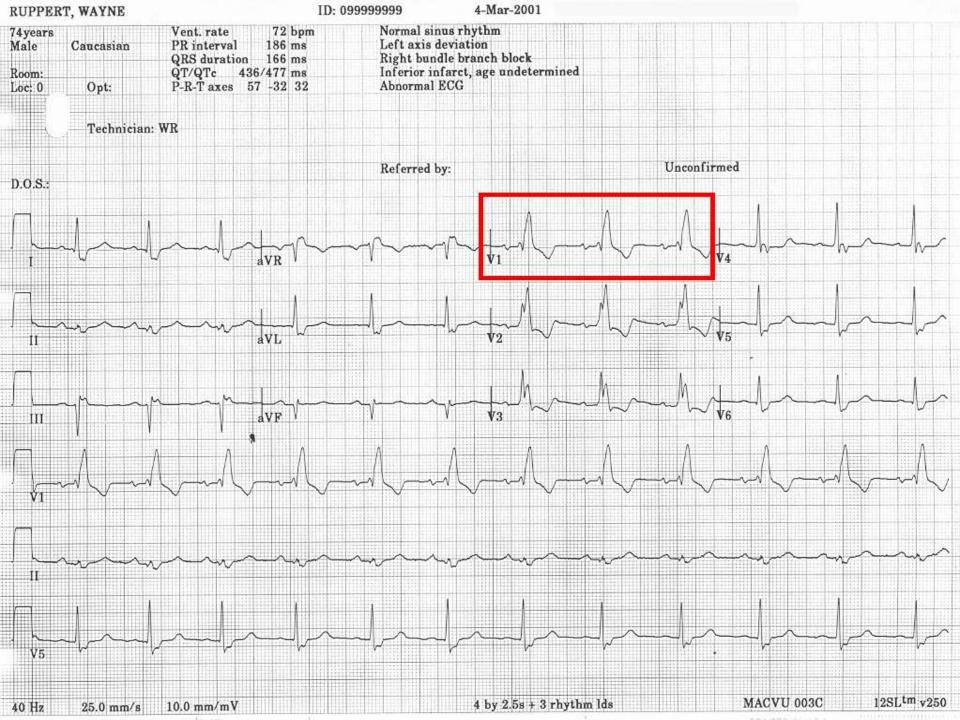


RIGHT BUNDLE BRANCH BLOCK



LEFT BUNDLE BRANCH BLOCK

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



# TERMINAL PHASE OF QRS IS POSITIVE

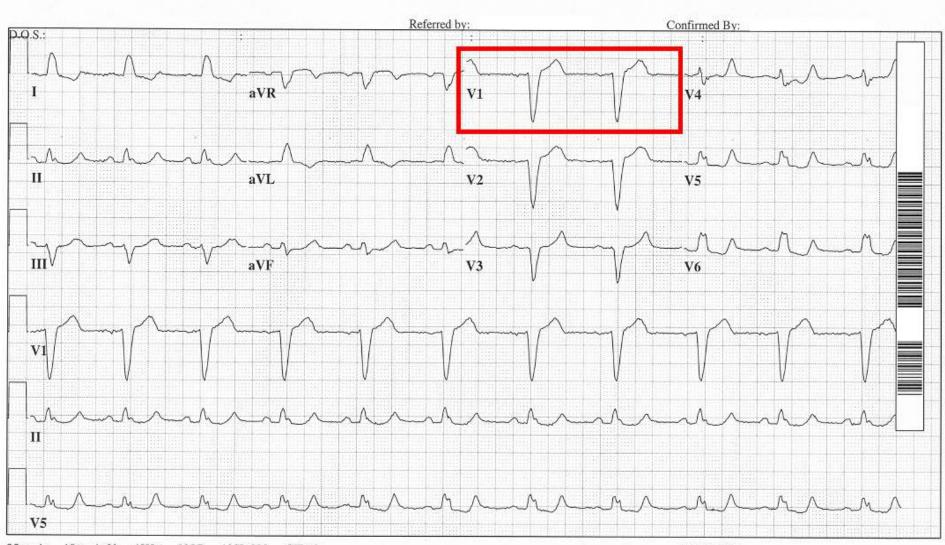


= RIGHT BUNDLE BRANCH BLOCK 74 yr Female Vent. rate 64 BPM Caucasian PR interval 188 ms QRS duration 152 ms QT/QTc 472/486 ms Loc:7 Option:35 P-R-T axes 78 3 106 EKG #WR03029959

Normal sinus rhythm Left bundle branch block Abnormal ECG

When compared with ECG of 28–MAY–2003 06:36,

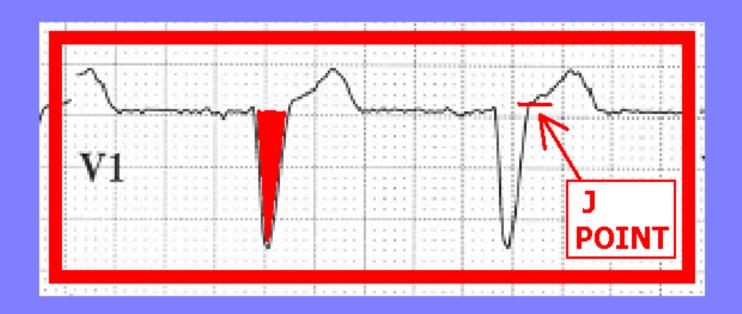
Technician: WW



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

EID:5 EDT:

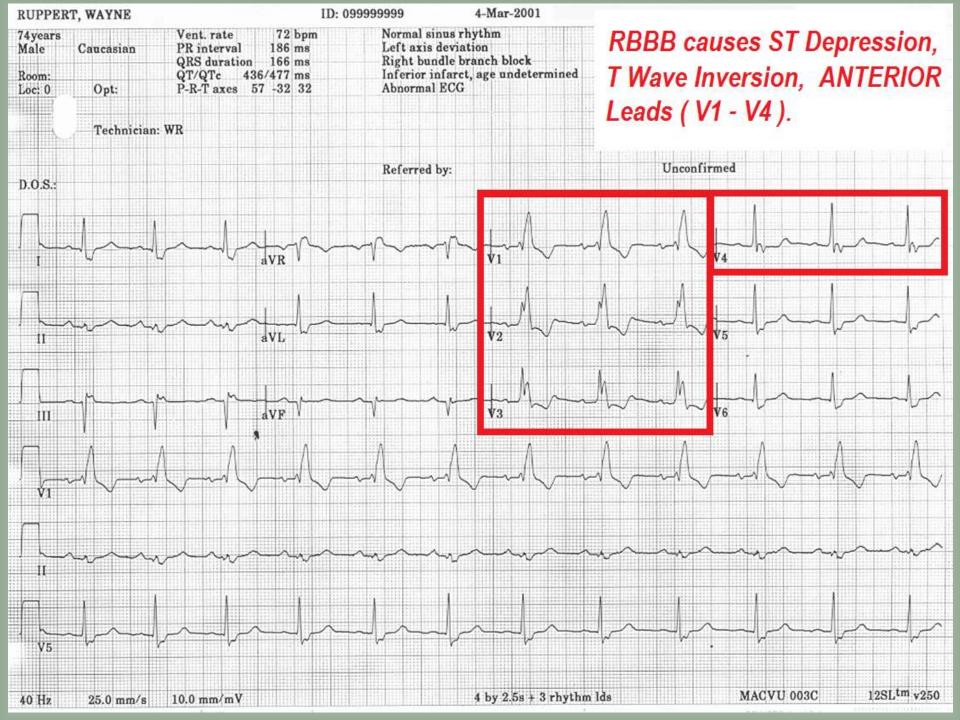
# TERMINAL PHASE OF QRS IS NEGATIVE



= LEFT BUNDLE
BRANCH BLOCK

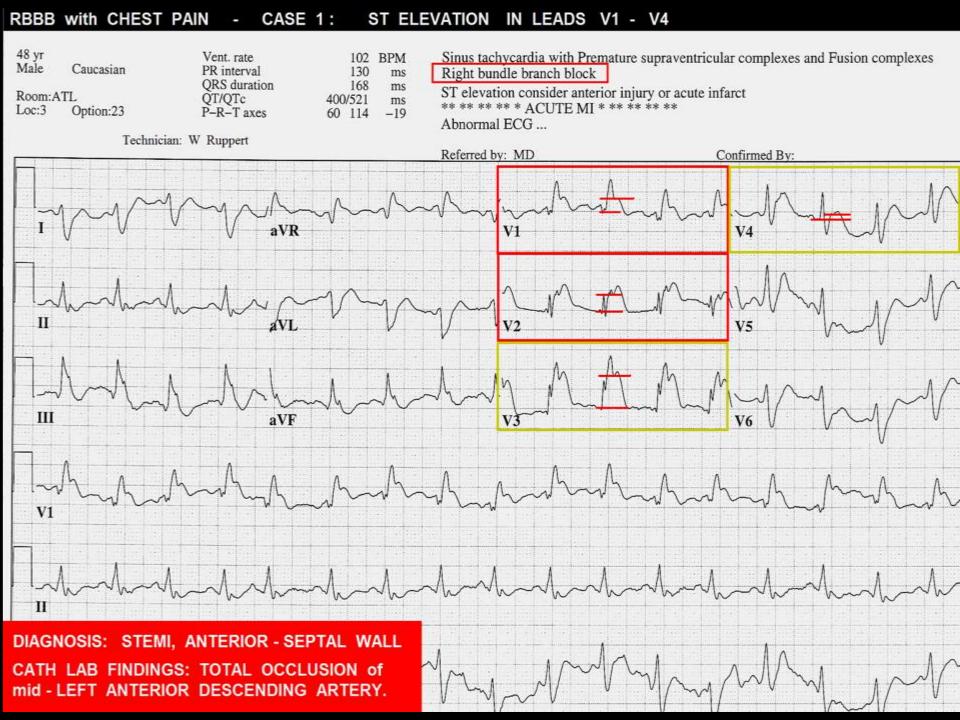
# Wide QRS present: (QRSd > 120ms)

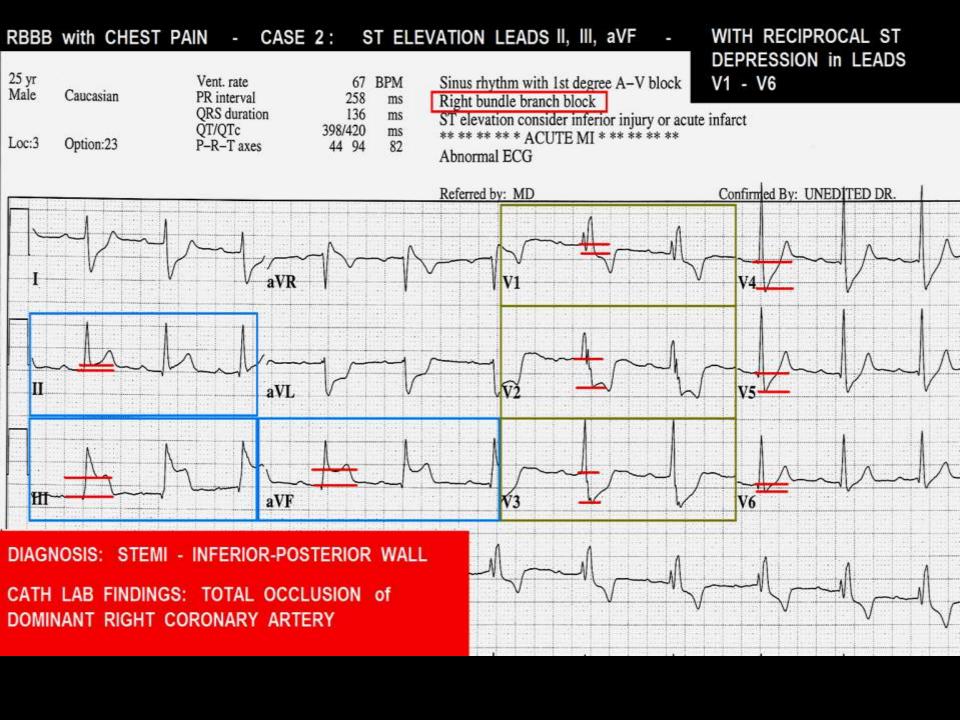
- When RIGHT Bundle Branch Block pattern is present:
  - Precordial Leads typically demonstrate ST
     Depression and T wave Inversion

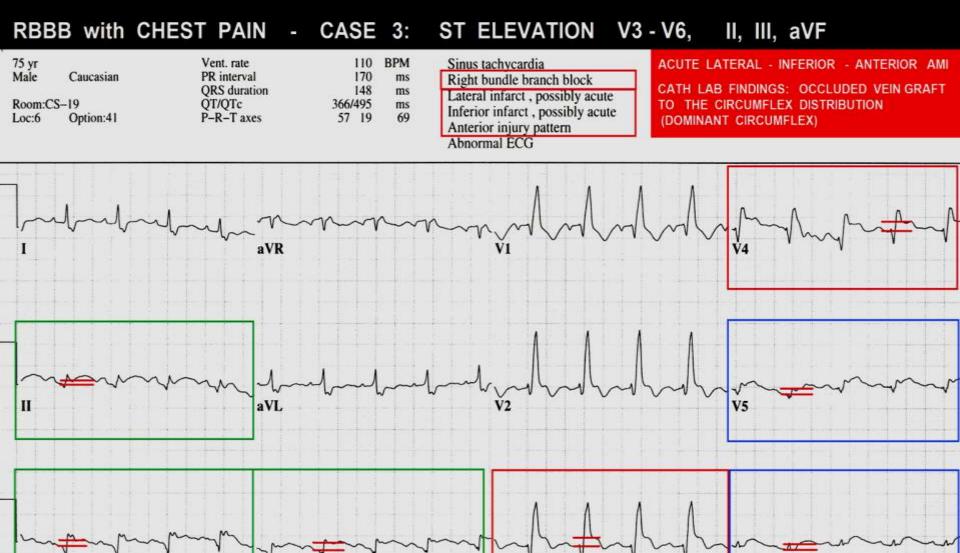


# Wide QRS present: (QRSd > 120ms)

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







(QRSd > 120ms)

When LBBB QRS pattern is present:

(QRSd > 120ms)

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

(QRSd > 120ms)

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

(QRSd > 120ms)

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

**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

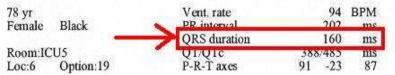
#### A.H.A. ACLS GUIDELINES

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

for diagnosis of STEMI

- 2. If patient has:
  - a) previously NORMAL ECGs (no LBBB)
    -- or --
  - b) no old ECGs available for comparison

consider diagnosis as STEMI until proven otherwise.



Normal sinus rhythm with occasional Premature ventricular complexes

Left bundle branch block Abnormal ECG - Normal arteries

- Normal LV Function

No hypertrophy







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

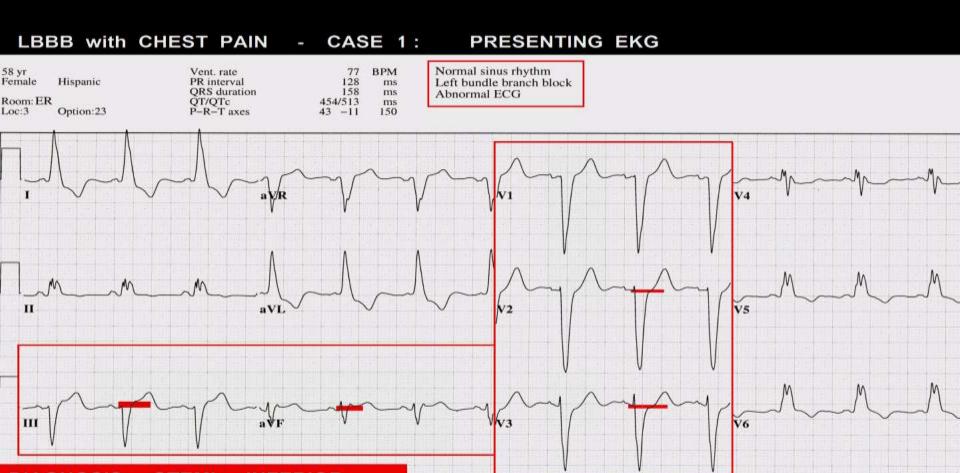
- ST ELEVATION > 5 mm
- COMPARE J POINT, ST SEGMENTS and T WAVES of previous ECG with LBBB to NEW ECG.
- CONVEX ST SEGMENT = poss. MI
  CONCAVE ST SEGMENT = normal
- CONCORDANT ST changes (1 mm or > ST DEPRESSION V1 - V3 or ST ELEVATION LEADS II, III, AVF)
- ST ELEVATION in LEADS II, III, and/or AVF

"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.

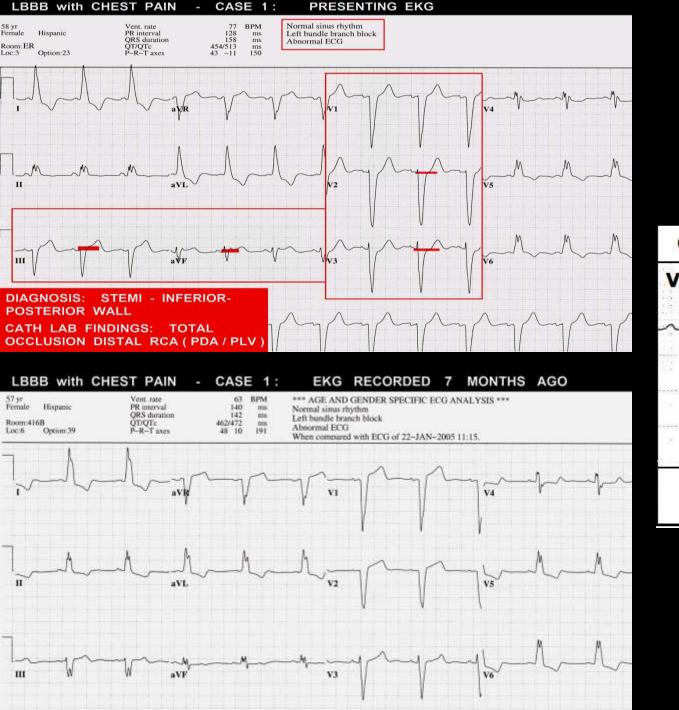


DIAGNOSIS: STEMI - INFERIOR-

POSTERIOR WALL

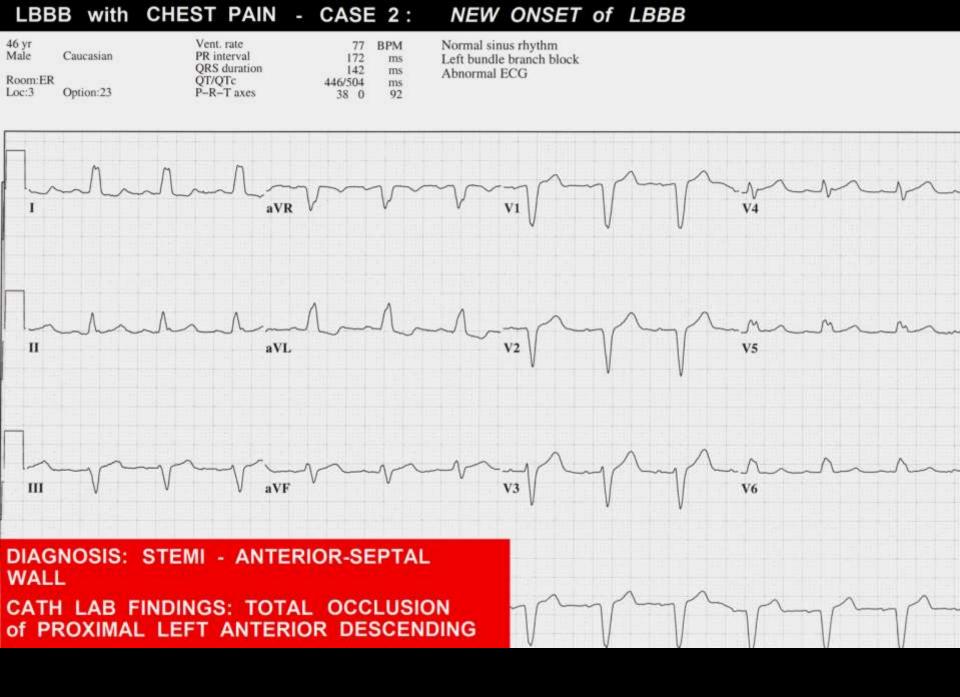
CATH LAB FINDINGS: TOTAL

OCCLUSION DISTAL RCA (PDA / PLV)



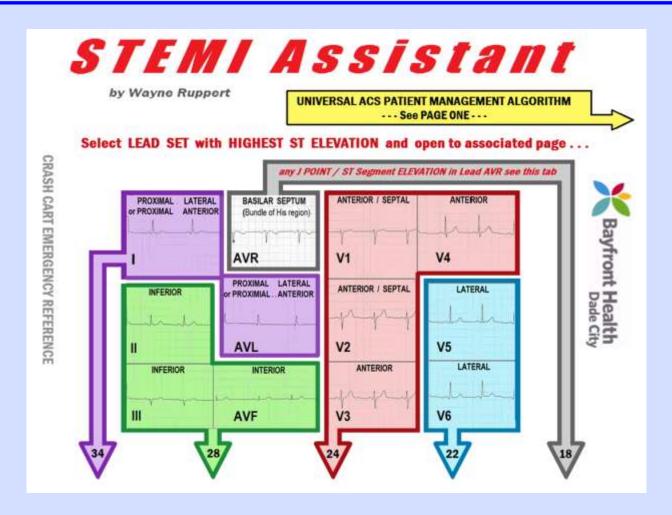


CHANGE



# STELL SS SEATE an Emergency Crash

#### Cart Interactive Reference Manual - free Download



**STEMI Assistant – Information Video** 

#### Helpful STEMI ECG Resources

[1] "Use of the Electrocardiogram in Acute Myocardial Infarction," Zimetbaum, et al, NEJM 348:933-940

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

<u>ECG in STEMI – excellent powerpoint – quick reference, in-depth material</u>

#### Helpful STEMI ECG Resources

**Download Non-ED STEMI Protocol - example** 

**Download STEMI Alert ED Physicians Order Set** 

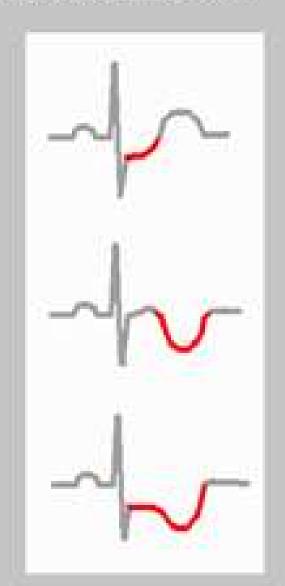
# **ISCHEMIA**

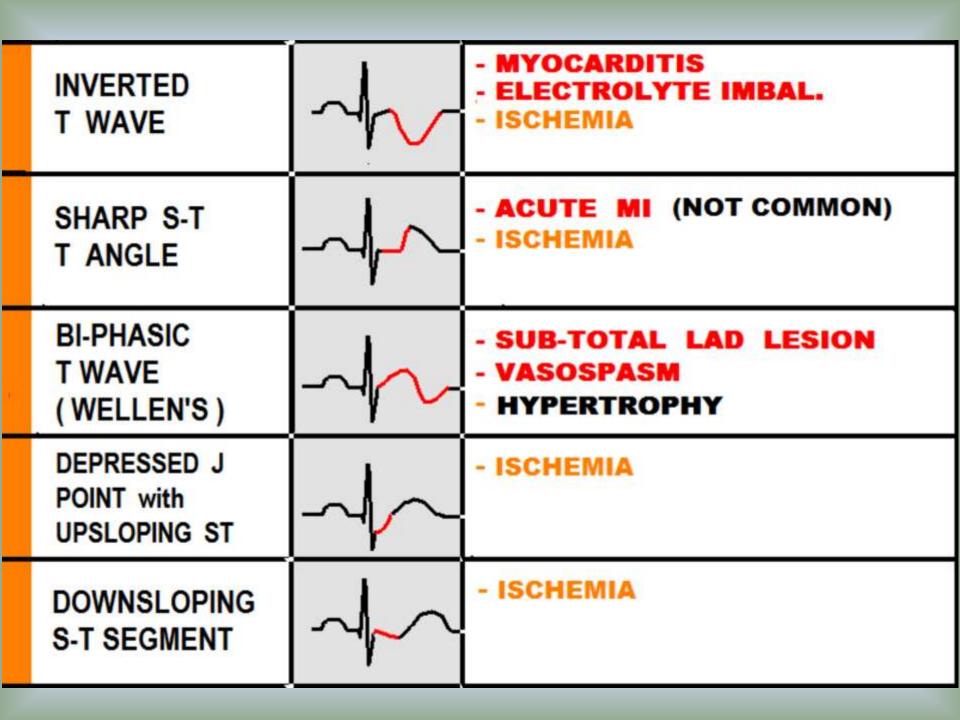
#### **HELPFUL PATTERNS . . .**

J POINT DEPRESSION (>1 mm)

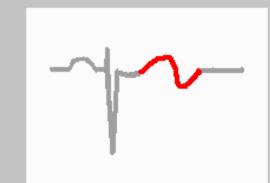
INVERTED T WAVES

- J POINT DEPRESSION
- + INVERTED T WAVES





#### **ISCHEMIA**



#### BI-PHASIC T WAVE

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

## **BI-PHASIC T WAVES**



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



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

#### Classic "Wellen's Syndrome:"

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

#### Wellen's Syndrome ETIOLOGY:

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

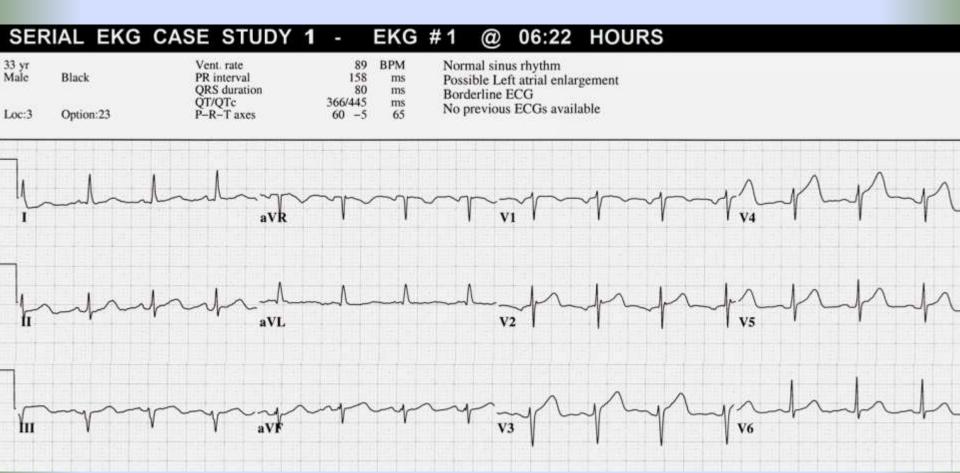
## Wellen's Syndrome EPIDEMIOLOGY & PROGNOSIS:

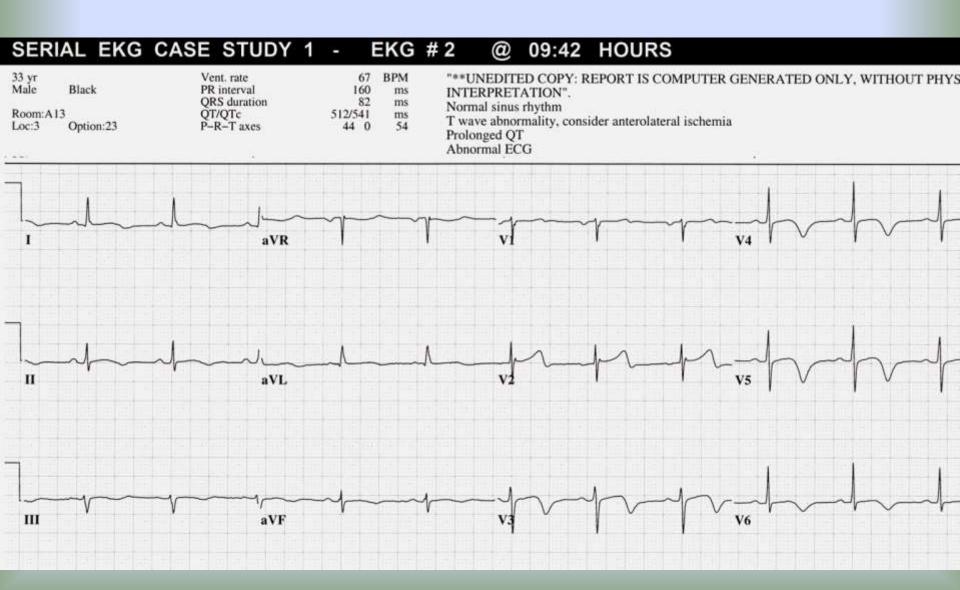
- Present in 14-18% of patients admitted with unstable angina
- 75% patients not treated developed extensive Anterior MI within 3 weeks.
- Median Average time from presentation to Acute Myocardial Infarction – 8 days

Sources: <u>H Wellens et. Al, Am Heart J 1982;</u>

v103(4) 730-736

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





# DYNAMIC ST-T Wave Changes ARE PRESENT!!

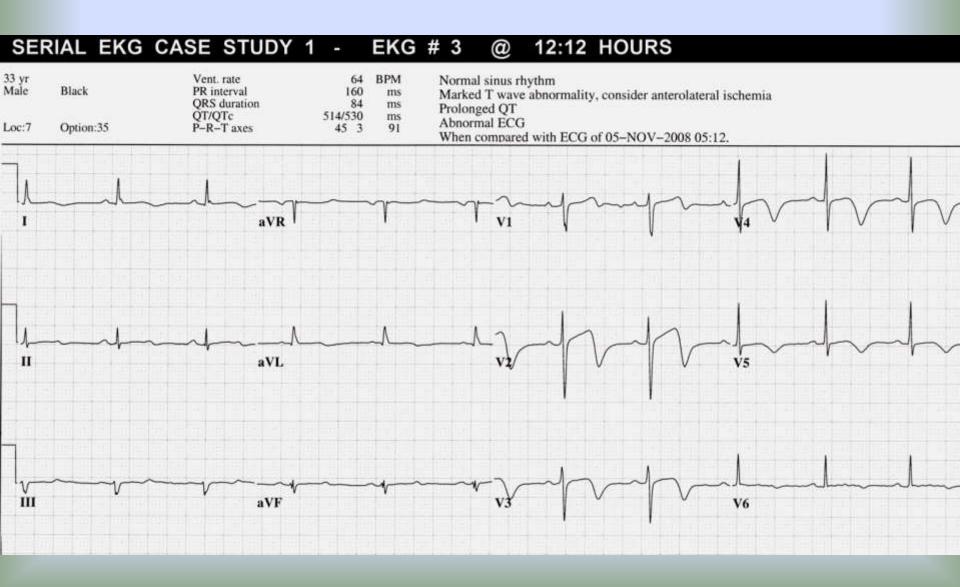
## NOW

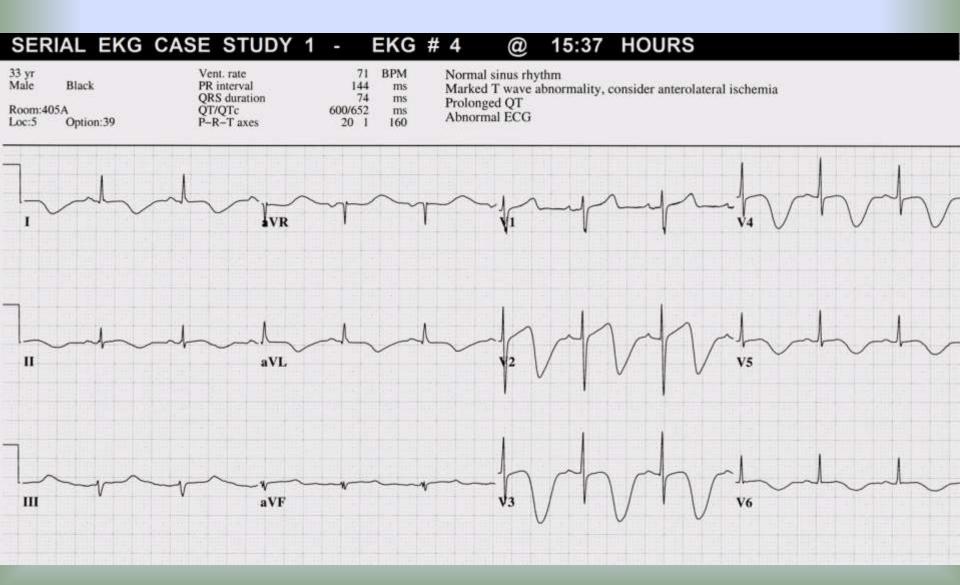
is the time for the

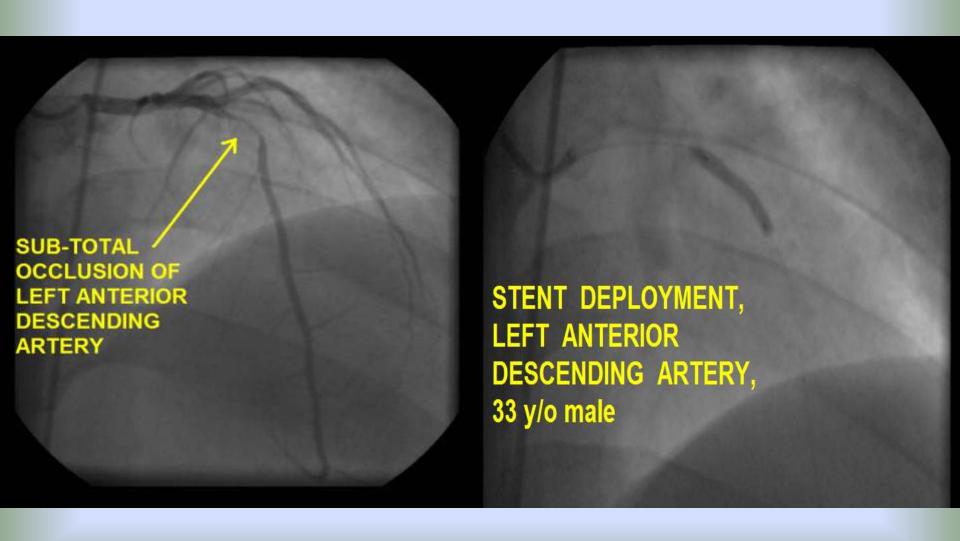
STAT CALL

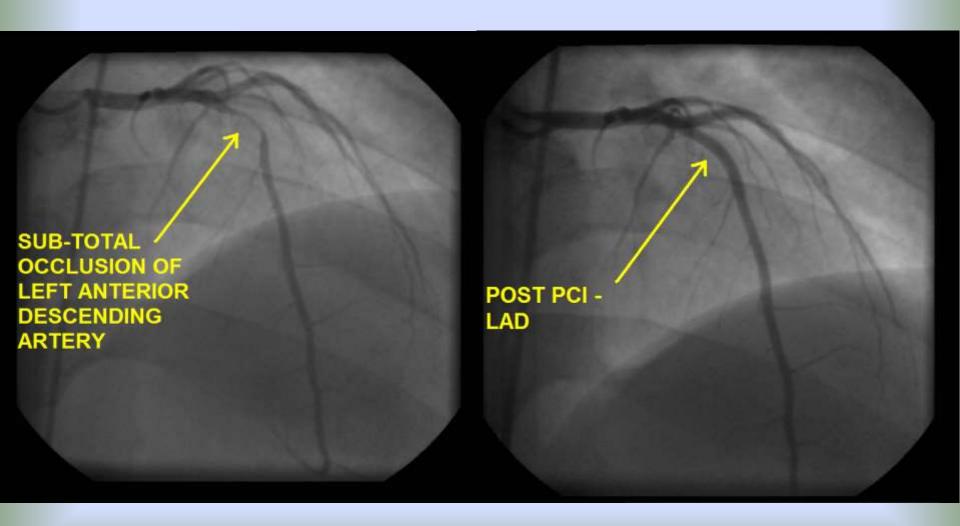
to the

CARDIOLOGIST !!!!





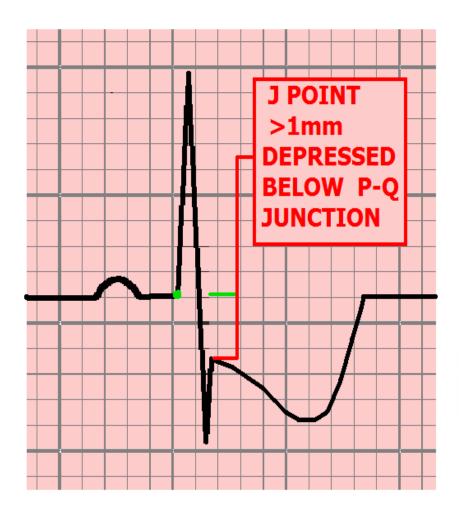




#### Additional Resources:

Wellen's Syndrome, NEJM case study

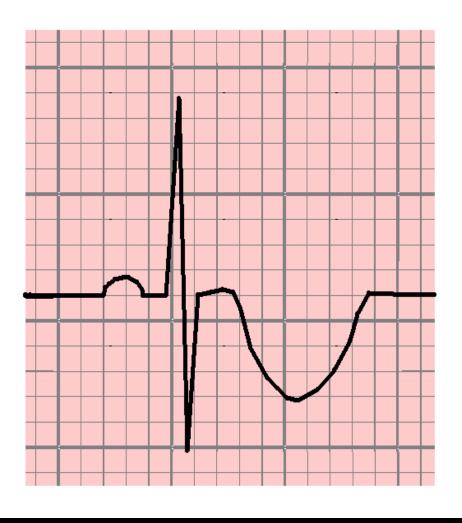
#### S-T SEGMENT DEPRESSION - COMMON ETIOLOGIES:



#### CONDITION:

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

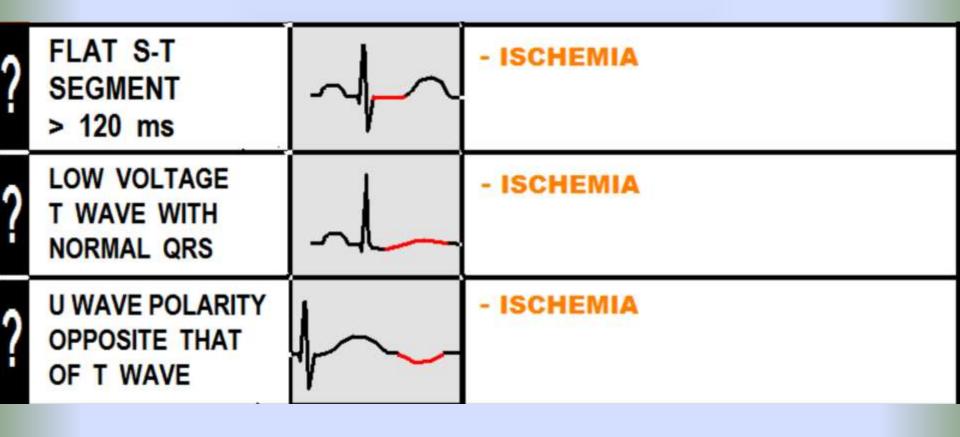
#### T WAVE INVERSION - COMMON ETIOLOGIES:



#### CONDITION:

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

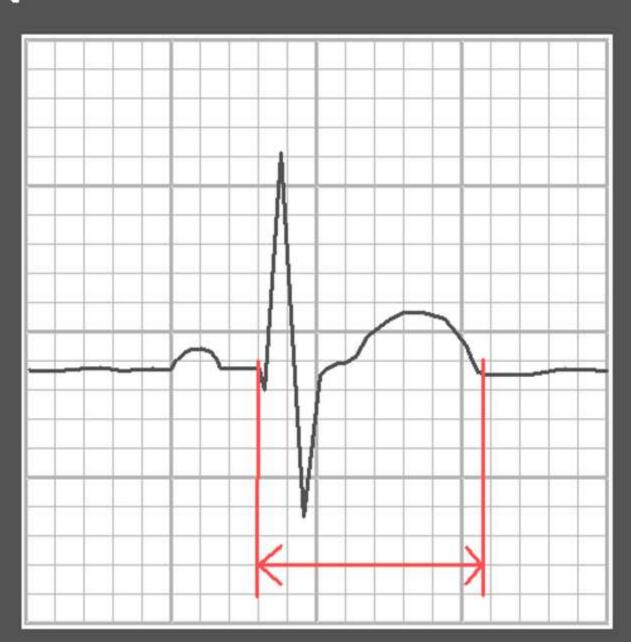
# Some less common, less reliable possible indicators of ACS:



#### Long QT Syndrome

#### Q - T INTERVAL

- VARIES BASED
ON HEART RATE
AND SEX



#### THE \*QTc INTERVAL

\*QTc = Q-T interval,

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

Annals of Internal Medicine, 1988 109:905.

#### Determining the QTc

Manual calculation:

#### **QT CORRECTION FORMULAS:**

Bazett's QTc=QT/√RR

Fredericia QTc=QT/(RR)1/3

Framingham QTc=QT+0.154(1-RR)

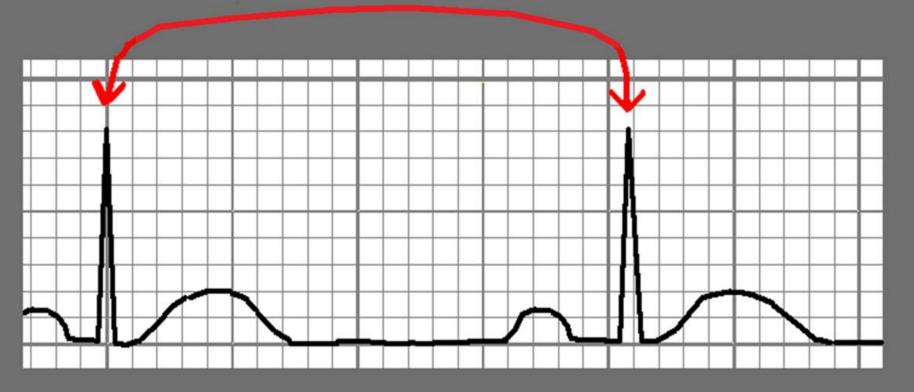
Rautaharju QTp=656/(1+HR/100)

#### DETERMINING Q-T INTERVAL LIMITS THE "QUICK PEEK" METHOD

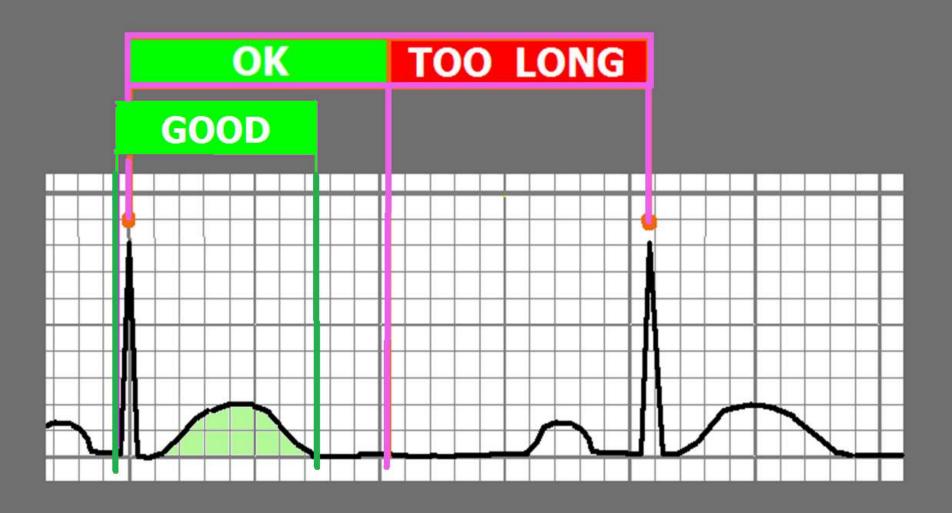


Relatively accurate method to quickly identify patients with abnormal QT Intervals.

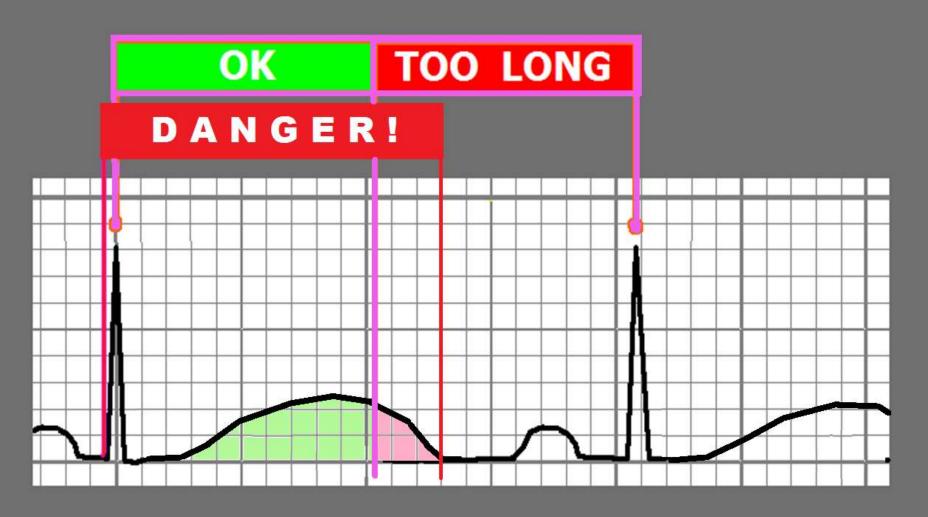
- Applies to patients with normal heart rates (60-100) and narrow QRS (QRSd <120ms)



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

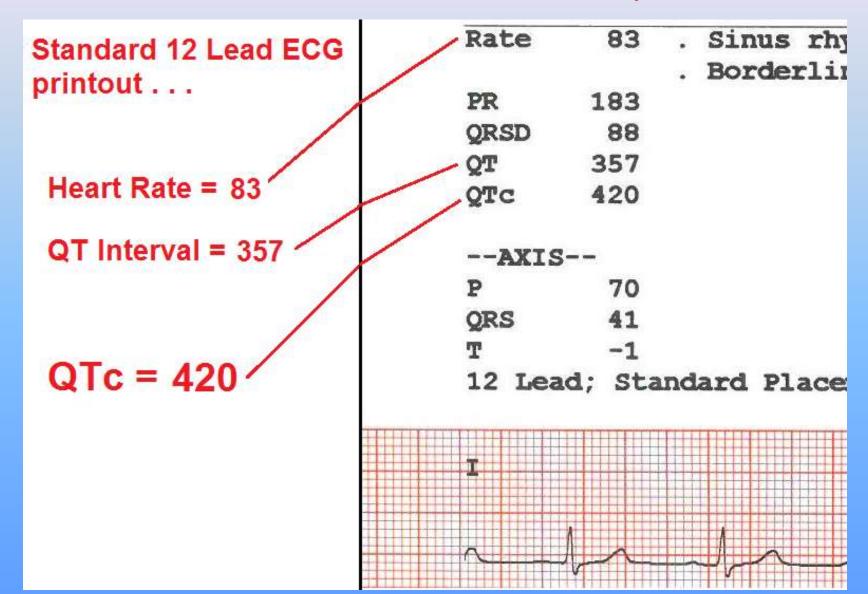


# The Q-T Interval should be LESS THAN 1/2 the R-R Interval



#### Determining the QT / QTc

Method 1 – 12 Lead ECG Report:



#### Determining the QTc

Method 4, Use a Smartphone App:

#### iPhone

- https://itunes.apple.com/us/app/corrected-qtinterval-qtc/id1146177765?mt=8

#### Android

 https://play.google.com/store/apps/details?id=co m.medsam.qtccalculator&hl=en

# "There's an APP for that!"



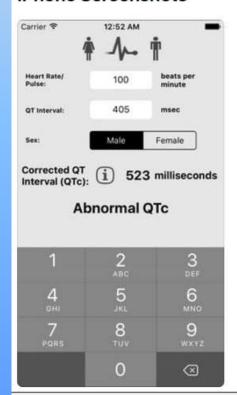
#### Corrected QT Interval (QTc) 17+

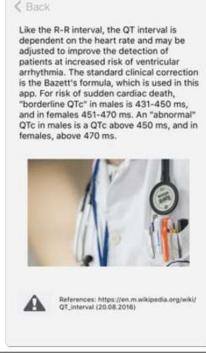
Daniel Juergens

\$0.99

Carrier ?

#### iPhone Screenshots





12:52 AM

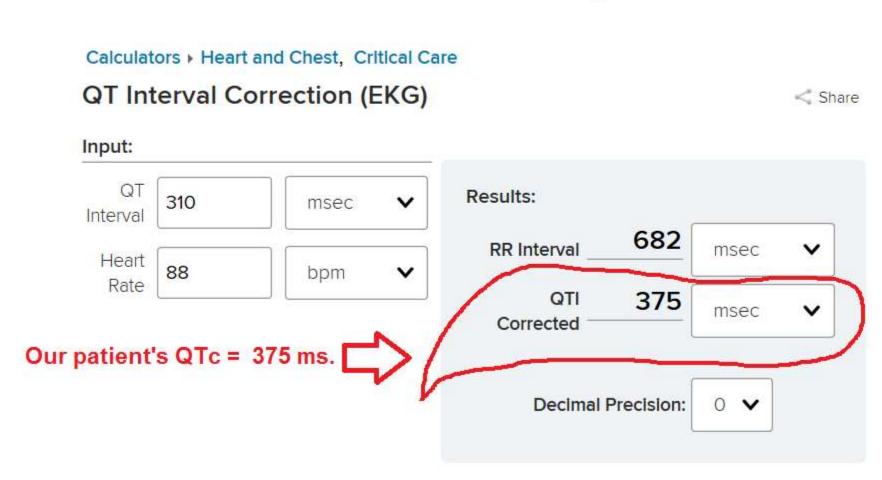


The information contained within this application is for informational purposes only and does not constitute medical or health advice. You should not rely on the information portrayed in this application as an alternative to medical advice from your doctor or any other professional healthcare provider.

#### Determining the QTc

Method 3, Use a Web-based App:





#### **QTc Values:**

Too Short: < 390 ms

**Normal** 

-Males: 390 - 450 ms

-Females: 390 - 460 ms

**Borderline High** 

-Males: 450 - 500 ms

-Females: 460 - 500 ms

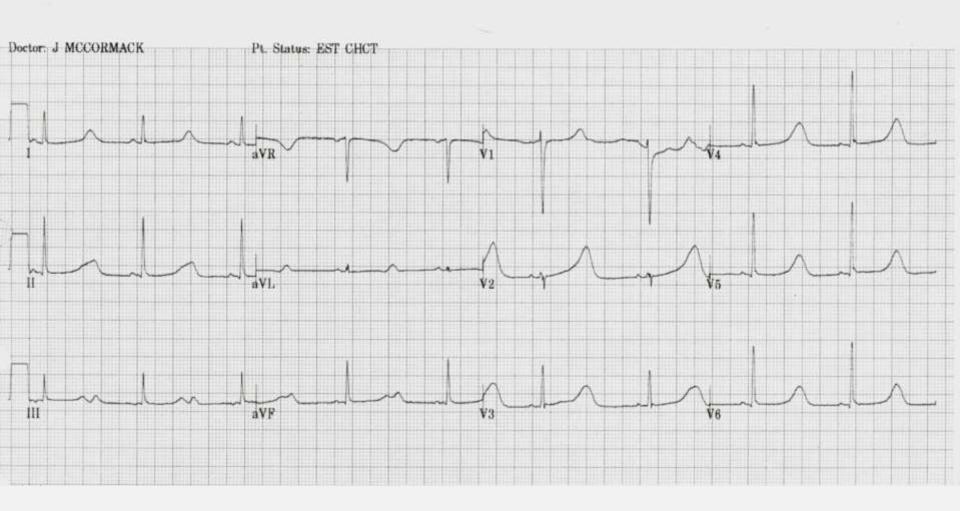
High (All Genders): 500 - 600 ms

**Critical High** 

(associated with TdP): 600 + ms

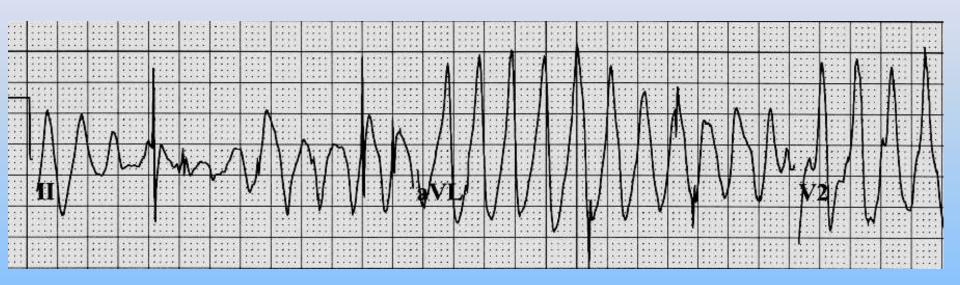
SOURCE: "ACC/AHA/HRS Recommendations for Standardization and Interpretation of the ECG, Part IV: The ST Segment, T and U Waves, and the QT Interval" Rautaharju et al 2009

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



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

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



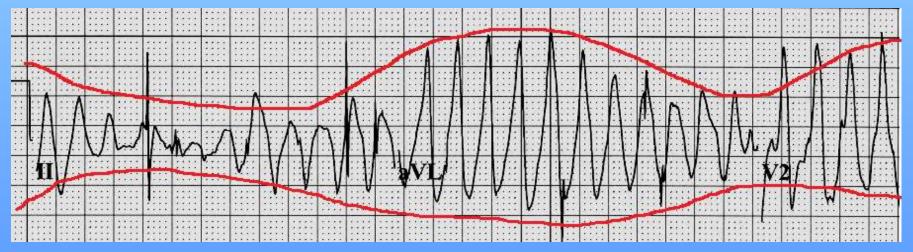
#### Torsades de Pointes (TdP) – HEMODYNAMICS:

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

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



#### a piece of Twisted Ribbon!



#### Evolving MI & "Old MI"

#### • Q WAVES •

Normal Q Waves

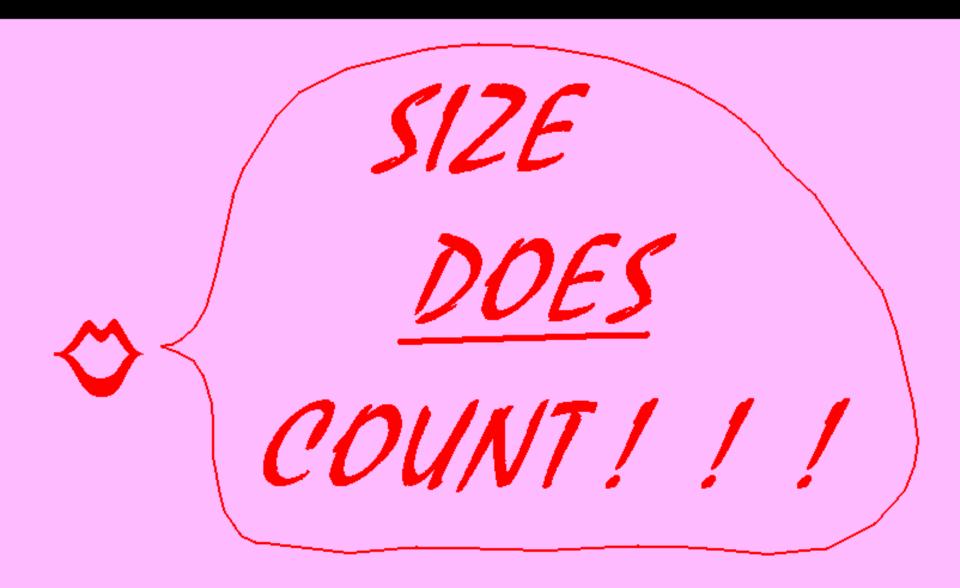
caused by depolarization of the intraventricular septum

Abnormal Q Waves -

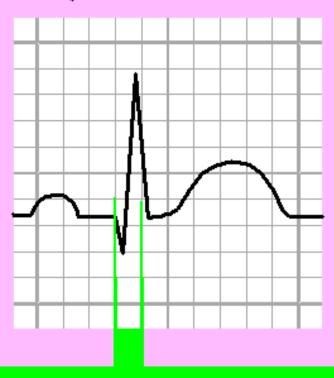
#### caused by:

- necrosis (old infarction)
- hypertrophy

## Q WAVES •

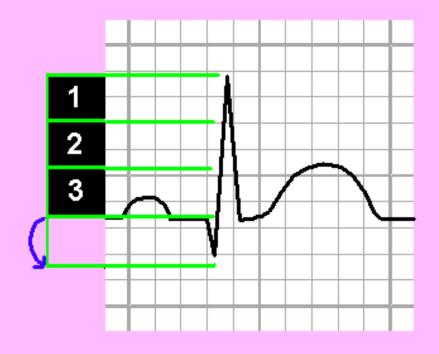


GENERAL RULES FOR NORMAL Q WAVES - WIDTH



LESS THAN .40 (1 mm) WIDE

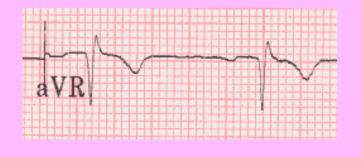
GENERAL RULES FOR NORMAL Q WAVES - HEIGHT



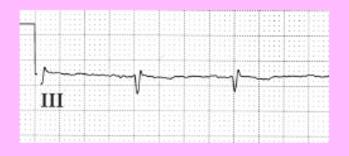


LESS THAN 1/3 THE HEIGHT OF THE R WAVE

## NORMAL Q WAVES EXCEPTIONS TO THE RULES



LEAD AVR



LEAD III



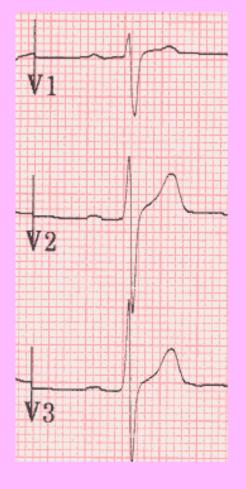
THE Q WAVE CAN BE ANY SIZE

## NORMAL Q WAVES EXCEPTIONS TO THE RULES

V2

V3

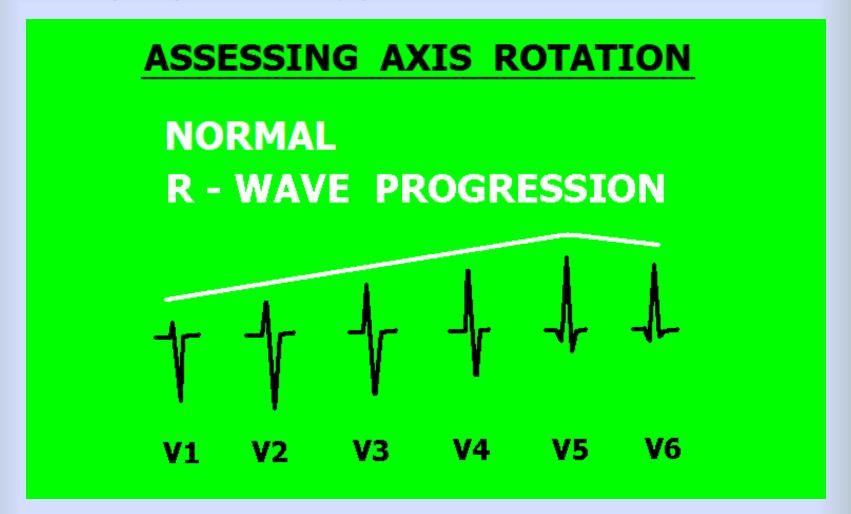
THERE
SHOULD BE NO Q
WAVES PRESENT
IN LEADS: V1



#### Q WAVE RULES - SUMMARY:

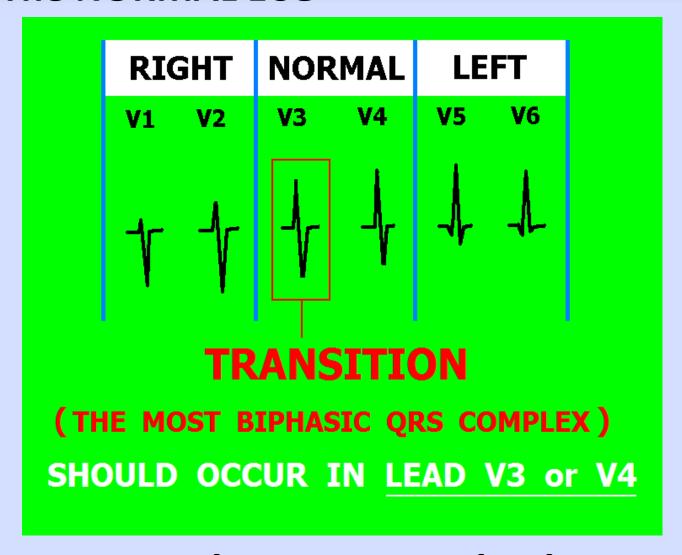
- Q WAVES SHOULD BE LESS THAN .40 WIDE (1 mm)
- Q WAVES SHOULD BE LESS THAN
   1/3 THE HEIGHT OF THE R WAVE
- Q WAVES CAN BE ANY SIZE IN LEADS III and AVR
- THERE SHOULD BE NO Q WAVES IN LEADS V1, V2, or V3

#### The NORMAL ECG



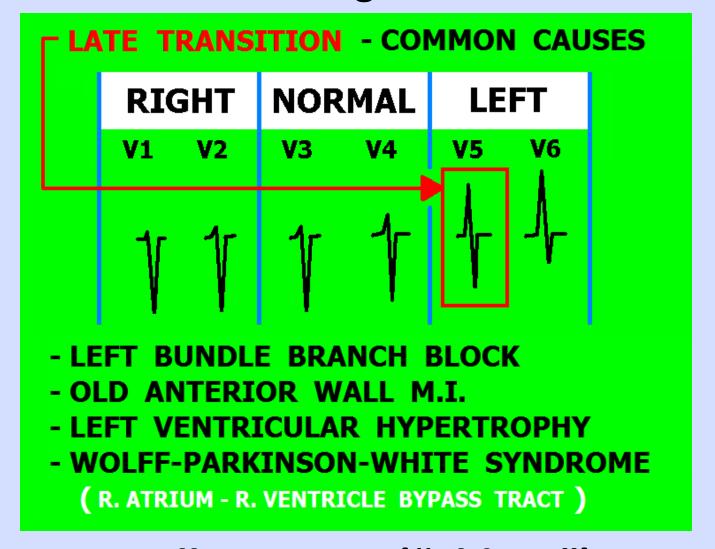
R wave amplitude (size) gradually increases from V1 through V6 . . . .

#### The NORMAL ECG



In V3 or V4, the QRS complex becomes Biphasic.

"Poor R Wave Progression" . . . .



Anterior Wall necrosis ("old MI") is a common cause of "Poor R Wave Progression".

### **EVOLVING STEMI:**

- -ST SEGMENTS DROP
- **-Q WAVES FORM**
- -R WAVE PROGRESSION CHANGES

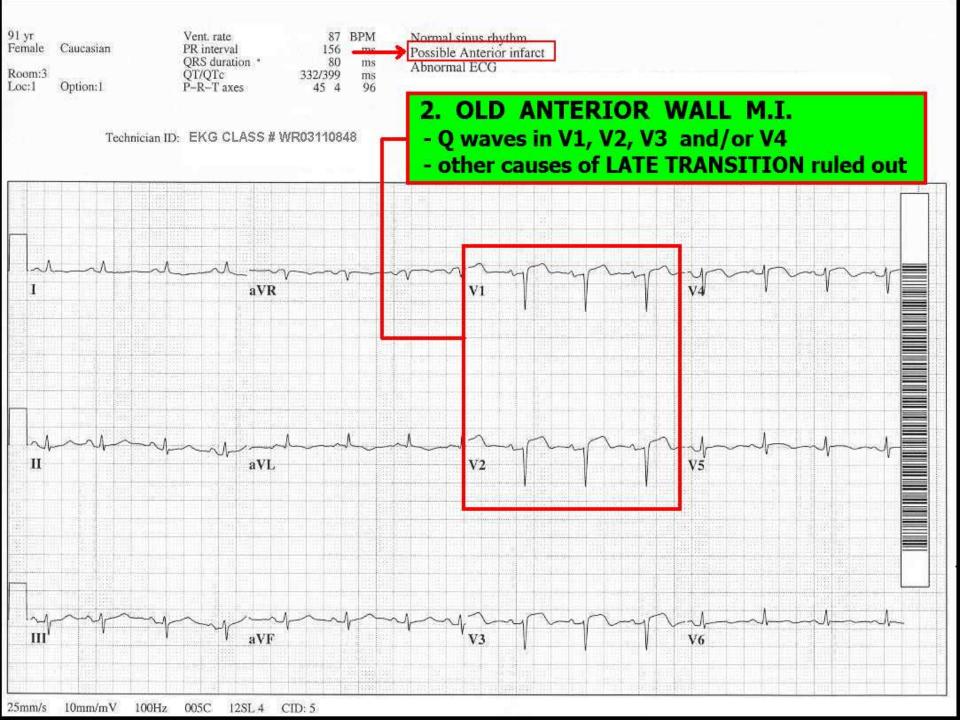
IN PRECORDIAL LEADS.

#### Q WAVE RULES - SUMMARY:

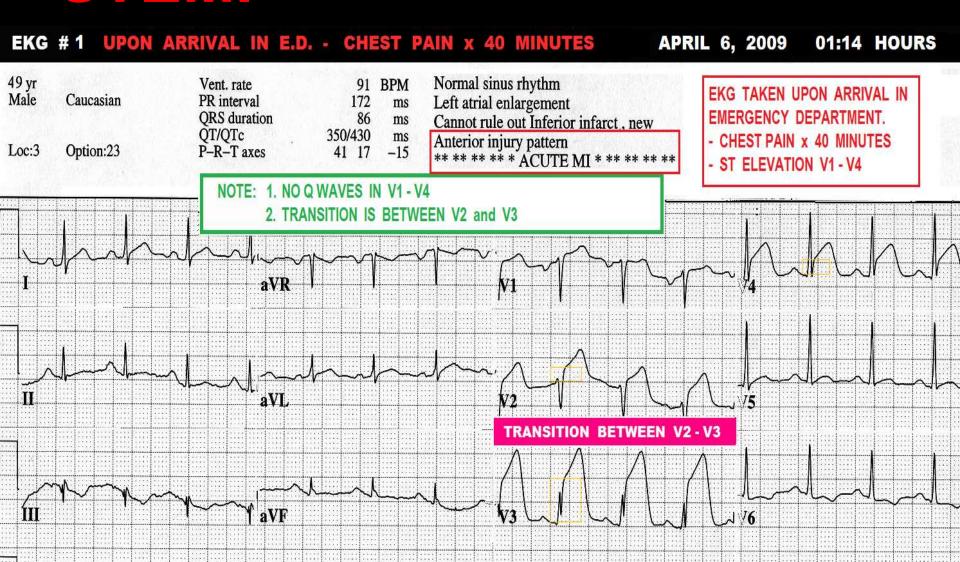
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- THERE SHOULD BE NO Q WAVES IN LEADS V1, V2, or V3

## **EVOLVING STEMI:**

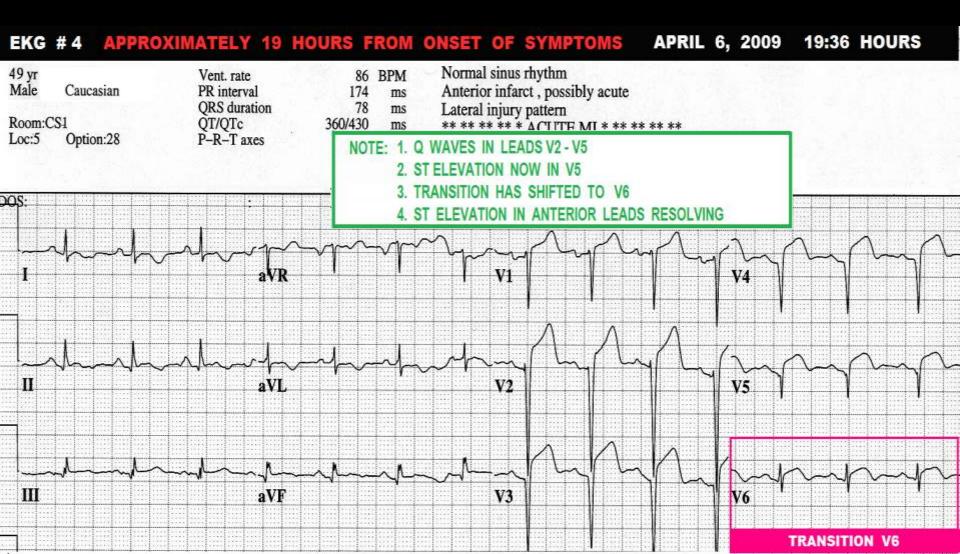
- -ST SEGMENTS DROP
- **-Q WAVES FORM**
- -R WAVE PROGRESSION CHANGES IN PRECORDIAL LEADS.



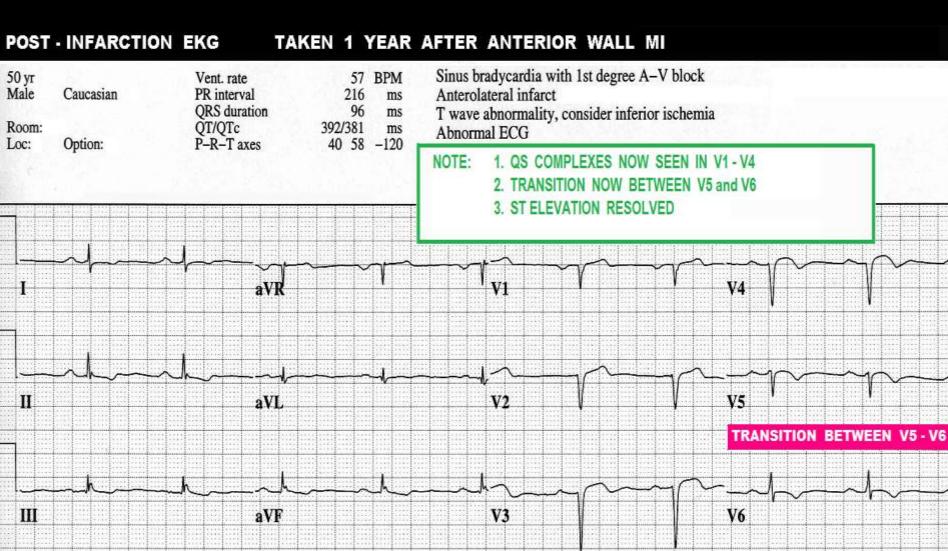
# ACUTE ANTERIOR WALL STEMI



## EVOLVING ANTERIOR WALL STEMI



# FULLY EVOLVED ANTERIOR WALL MI



### Additional Materials:

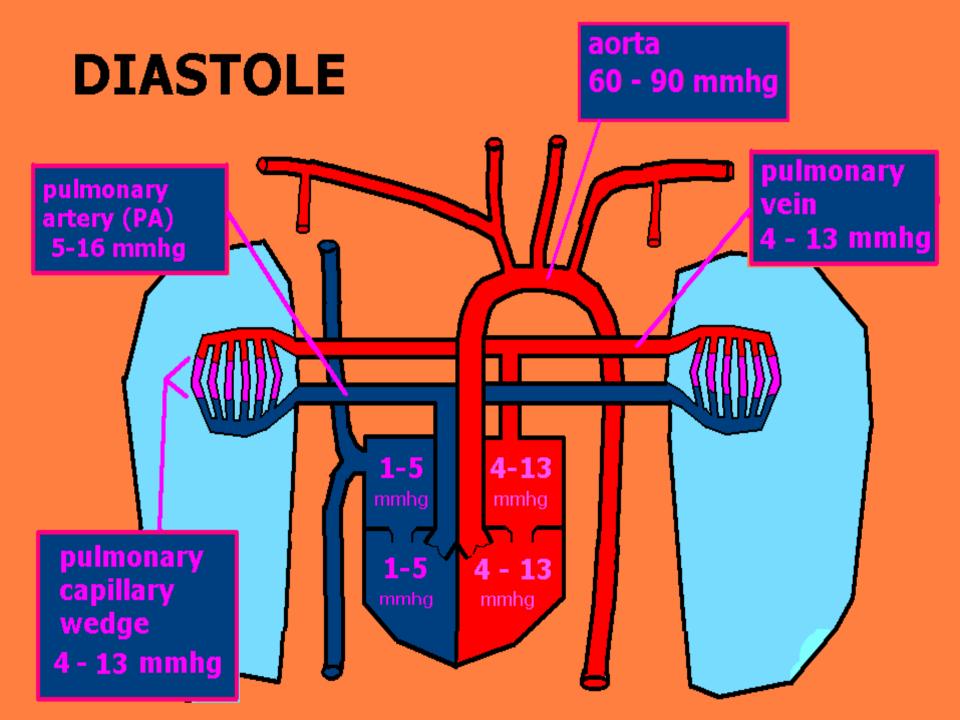


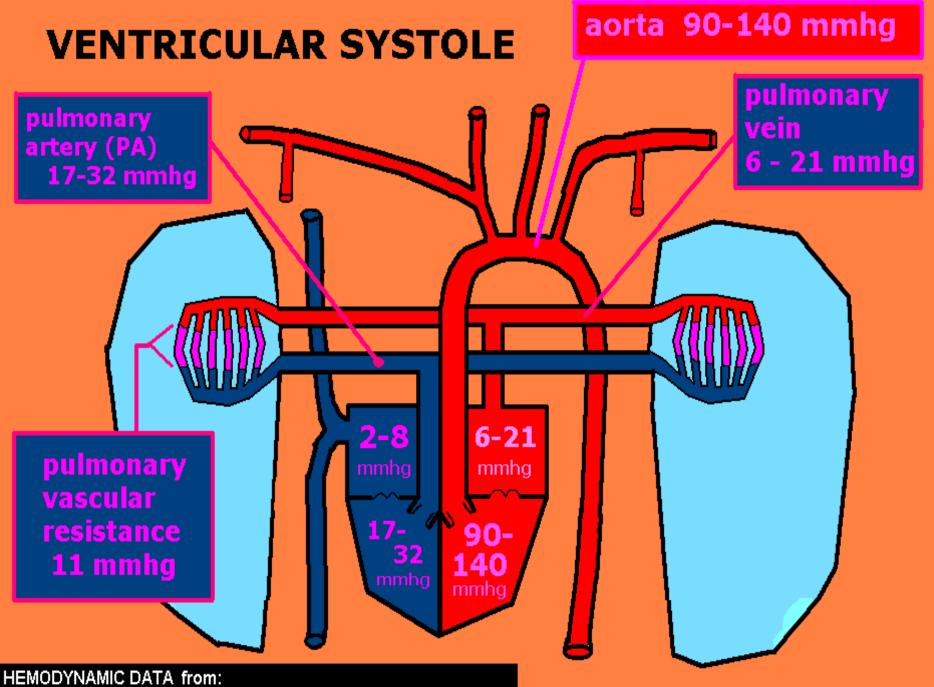
### BASIC HEART SOUNDS ASSESSMENT

## ABNORMAL EKG CHANGES THAT MAY PRESENT WITH ABNORMAL HEART SOUNDS:

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





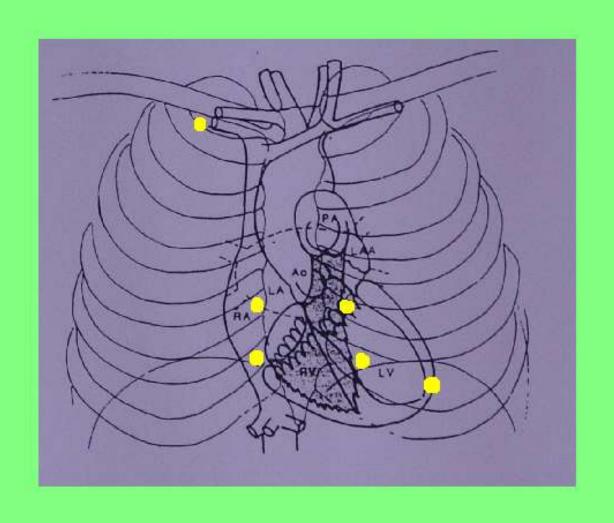


"The Cardiac Catheterization Handbook," Morton J. Kearn, MD

## HEART SOUNDS ASSESSMENT



## HEART SOUNDS ASSESSMENT



#### VERY

#### BASIC HEART SOUNDS ASSESSMENT

- NormalHeartSounds
- Murmurs
  - systolic
  - diastolic
- FrictionRubs



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

## HEART SOUNDS ASSESSMENT

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

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

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



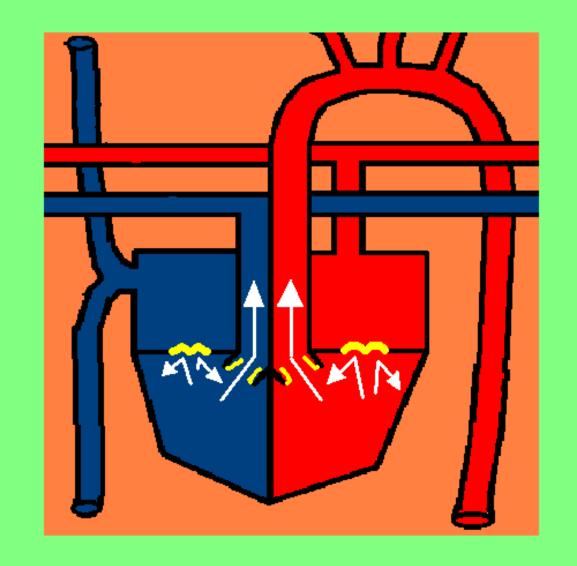
## VERY BASIC HEART SOUNDS ASSESSMENT

TE MI MBER HYPERTROPHY ROSIS - RECENT
ROSIS - RECENT
ISIVE MI (7-10 days)
TE MI
ENT MI (NECROSIS)
CARDITIS
E

## HEART SOUNDS ASSESSMENT

S-1 BEGINNING OF SYSTOLE.

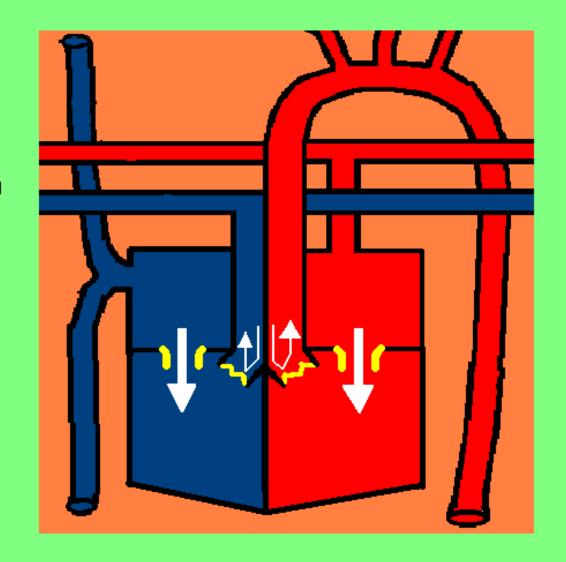
SOUND OF THE MITRAL AND TRICUSPID VALVES CLOSING.



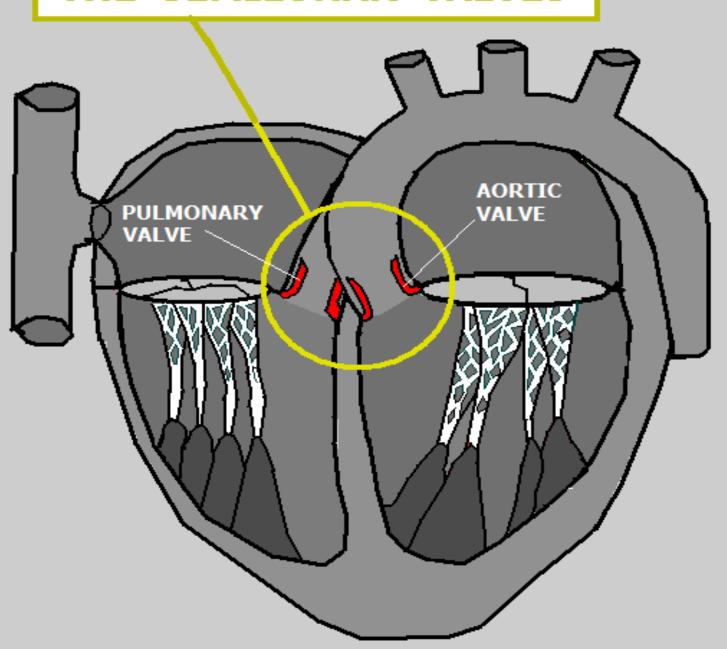
## HEART SOUNDS ASSESSMENT

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

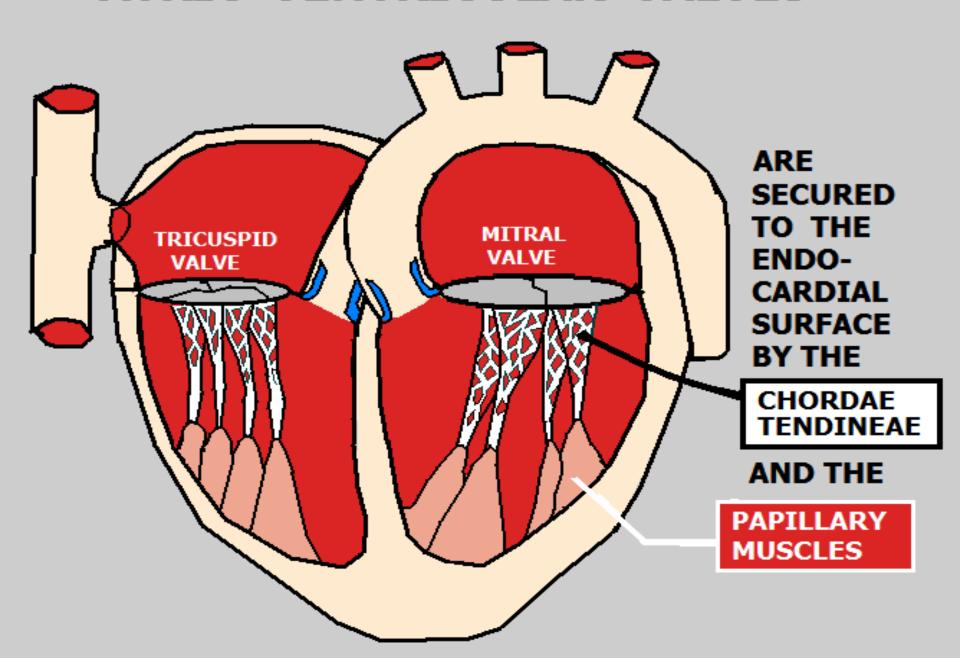
IT IS THE SOUND OF THE AORTIC AND PULMONARY VALVES CLOSING.



#### THE SEMILUNAR VALVES

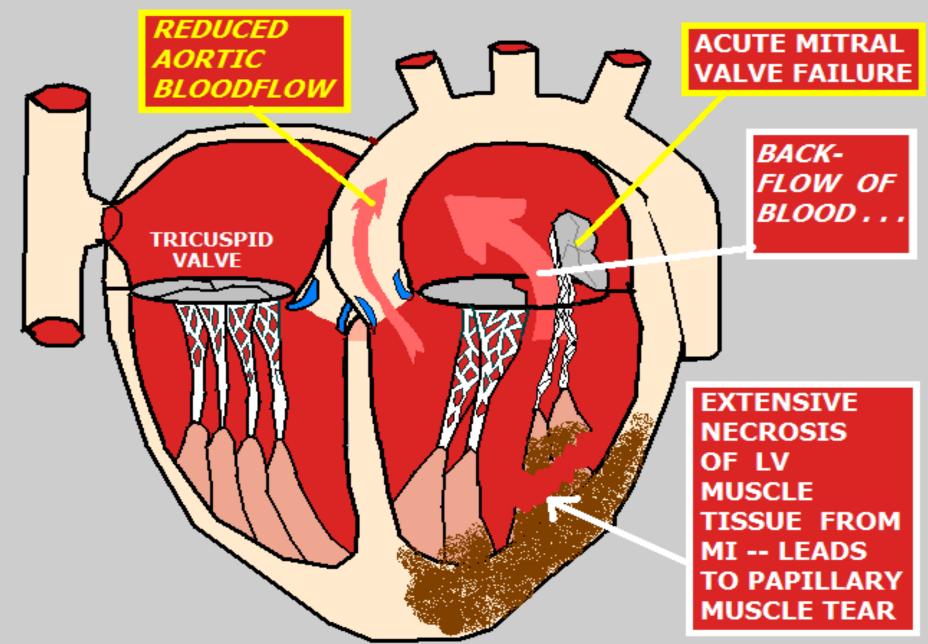


#### ATRIO-VENTRICULAR VALVES



#### ACUTE MITRAL REGURGITATION

**DURING VENTRICULAR SYSTOLE** 



#### BASIC HEART SOUNDS ASSESSMENT

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

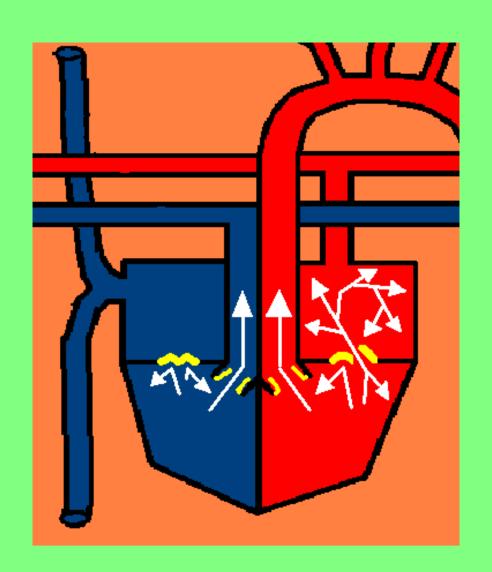
S-1 MURMUR SOUNDS LIKE:

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



### CAUSE OF SYSTOLIC (S 1) MURMUR

- DAMAGE TO
   MITRAL and/or
   TRICUSPID
   VALVE(s)
- CAUSESREGURGITATION

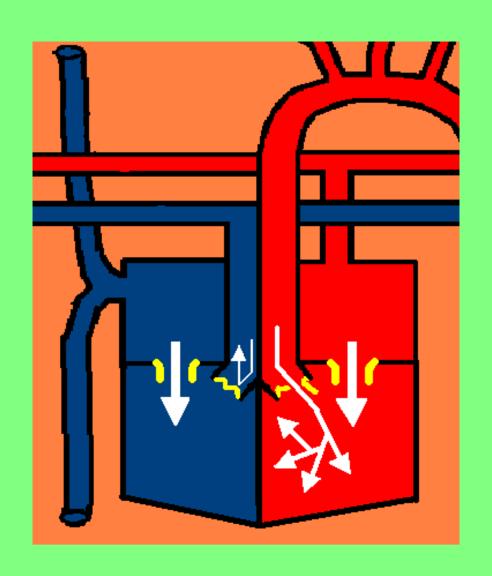


MOST SYSTOLIC MURMURS CAUSED BY MITRAL VALVE FAILURE.

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

### CAUSE OF DIASTOLIC (S2) MURMUR

- DAMAGE TO
   AORTIC and/or
   PULMONIC
   VALVE(s)
- CAUSESREGURGITATION



#### 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 . . . . LUB-
```

■ AORTIC VALVE FAILURE MOST COMMON CAUSE OF S-2 MURMUR

DUE TO THE HIGHER PRESSURES OF THE LEFT SIDE OF THE HEART

#### BASIC HEART SOUNDS ASSESSMENT

#### FRICTION RUB

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



#### BASIC HEART SOUNDS ASSESSMENT

#### FRICTION RUB

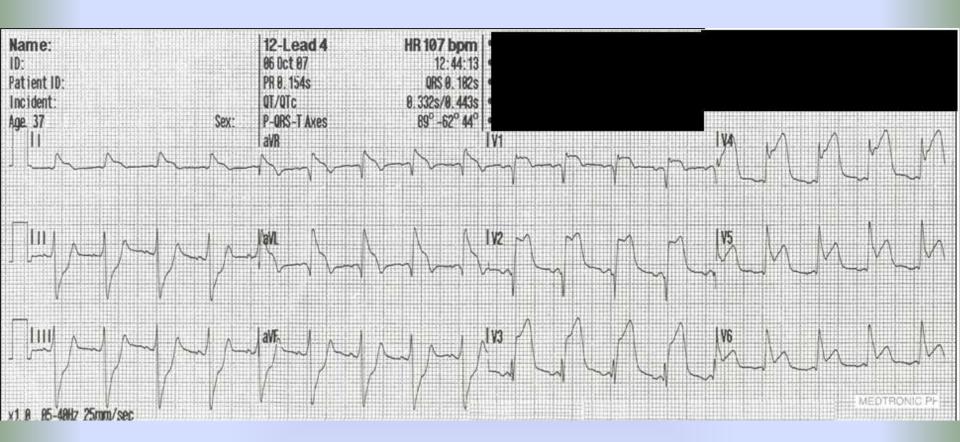
☐ IS PRESENT IN MOST ACUTE TRANSMURAL MI PATIENTS



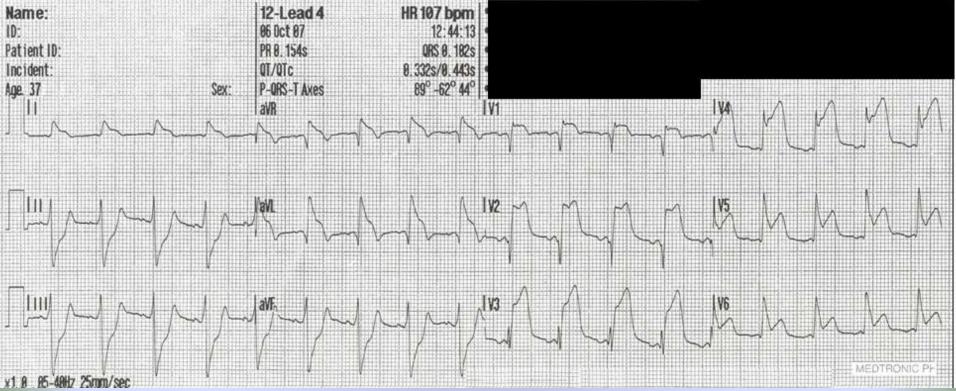
- MAY BE PRESENT
  WITHIN HOURS AFTER ONSET
- IS TRANSIENT -- MAY LAST FOR A FEW DAYS

### Practice ECGs . . .

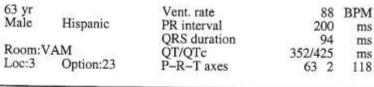
#### Let's review . . . .

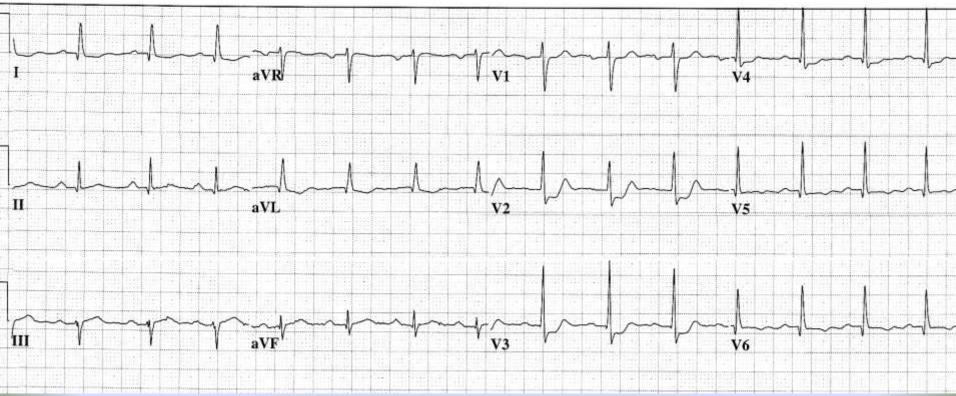


- 1. ECG abnormality(ies)?
- 2. Possible diagnosis?
- 3. Action / Intervention?

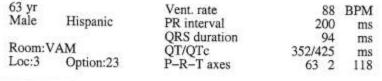


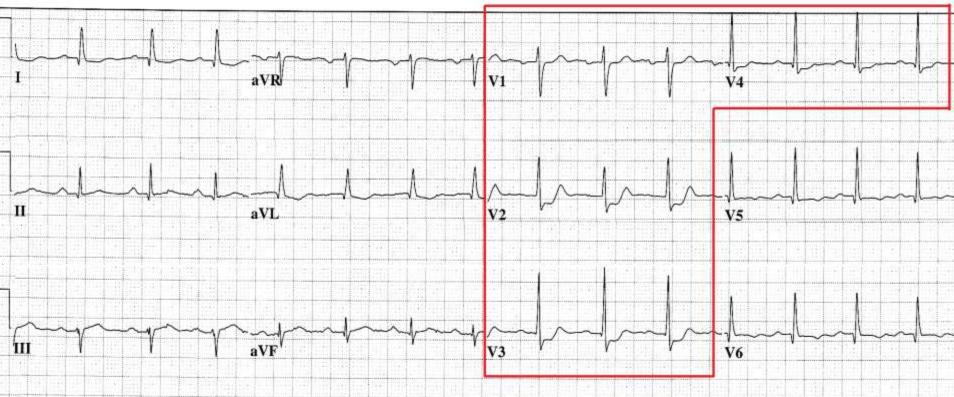
- 1. ECG abnormality(ies)? ST Elevation Leads I, AVR AVL, V1, V2, V3, V4, V5 & V6. ST Depression II, III and AVF
- 2. Possible diagnosis? Acute Anterolateral Wall STEMI secondary to Left Main Coronary Artery occlusion (widow-maker MI).
- 3. Action / Intervention? STAT CATH LAB vs STAT Thrombolytics. Prepare for Cardiac Arrest



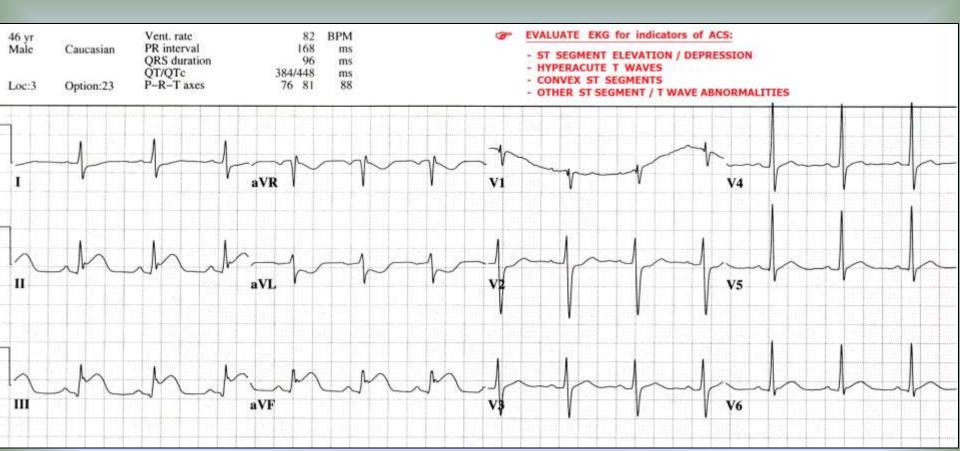


- 1. ECG abnormality(ies)?
- 2. Possible diagnosis?
- 3. Action / Intervention?

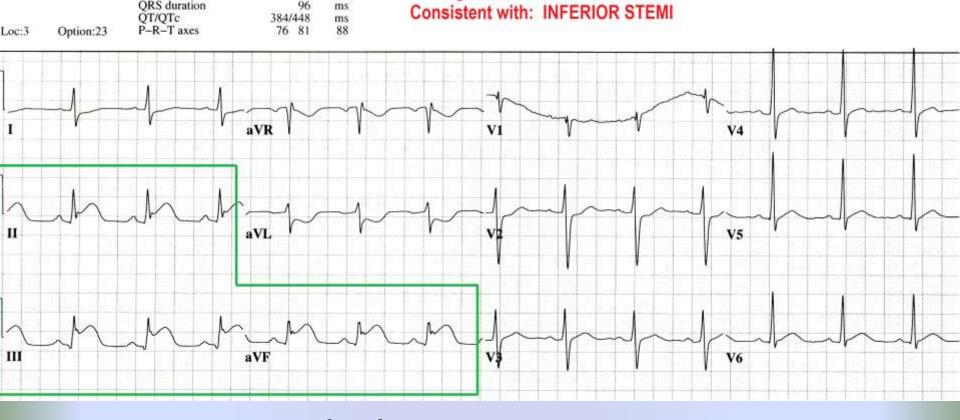




- 1. ECG abnormality(ies)? ST Depression V1-V4
- 2. Possible diagnosis? Anterior ischemia vs. Posterior wall STEMI
- 3. Action / Intervention? Posterior ECG (V7-V9)



- 1. ECG abnormality(ies)?
- 2. Possible diagnosis?
- 3. Action / Intervention?



ST-Segment Elevation in Leads II, III and AVF

- 1. ECG abnormality(ies)? ST Elevation, Leads II, III & AVF
- 2. Possible diagnosis? Inferior Wall STEMI

**BPM** 

46 yr

Male

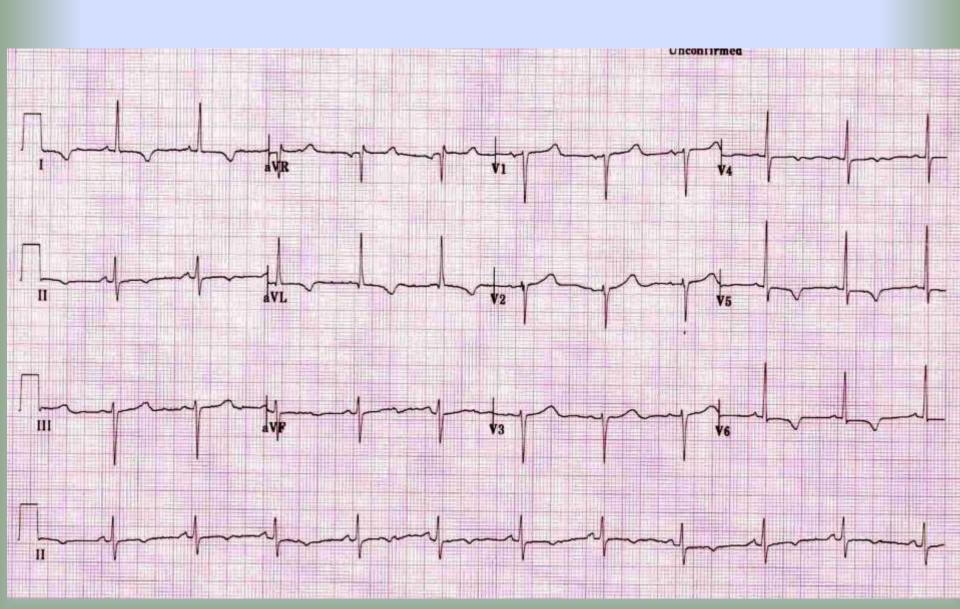
Caucasian

Vent. rate

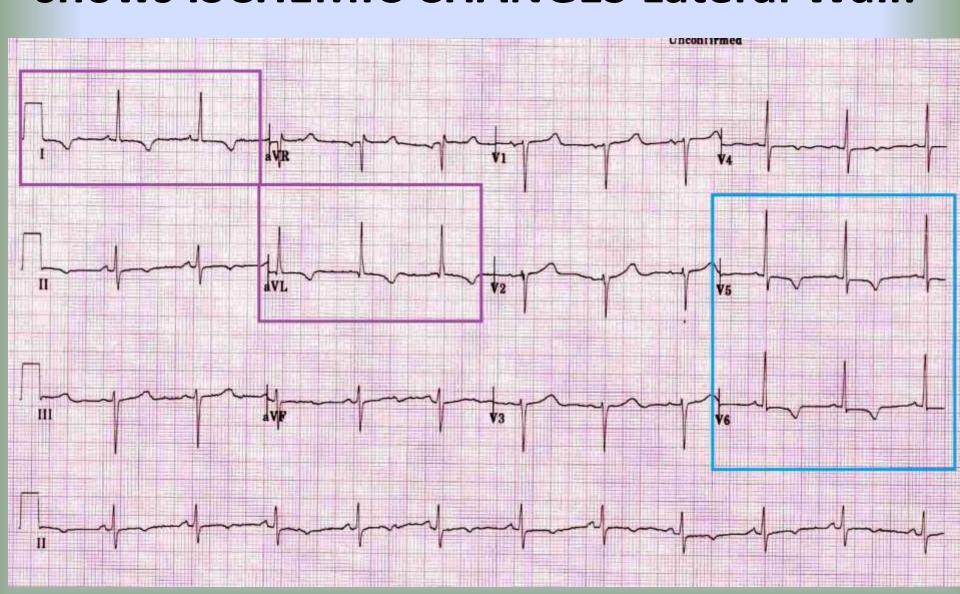
PR interval

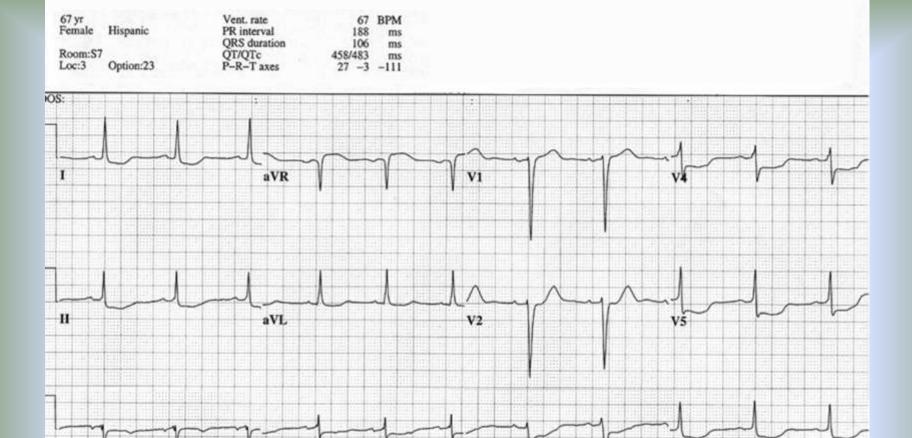
3. Action / Intervention? 1. Do R-sided ECG, prepare for Atropine administration, external pacing, cardiac arrest, STAT cath lab visit!

## What leads show signs of possible ACS?



## 12 Lead ECG shows ISCHEMIC CHANGES Lateral Wall:

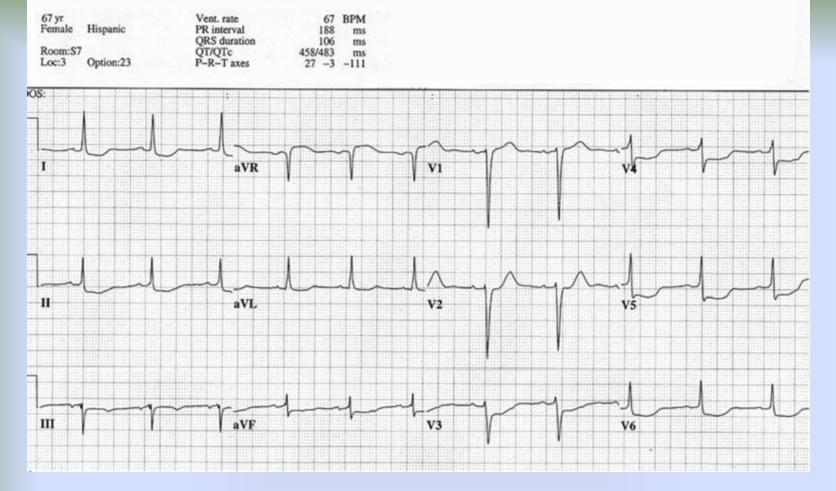




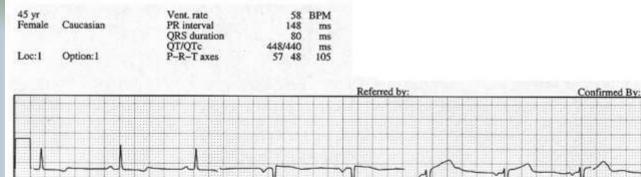
V3

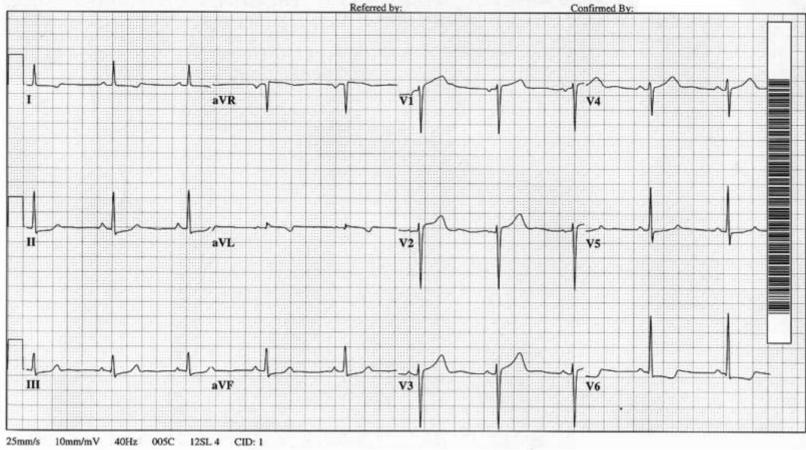
V6

- 1. ECG abnormality(ies)?
- 2. Possible diagnosis?
- 3. Action / Intervention?

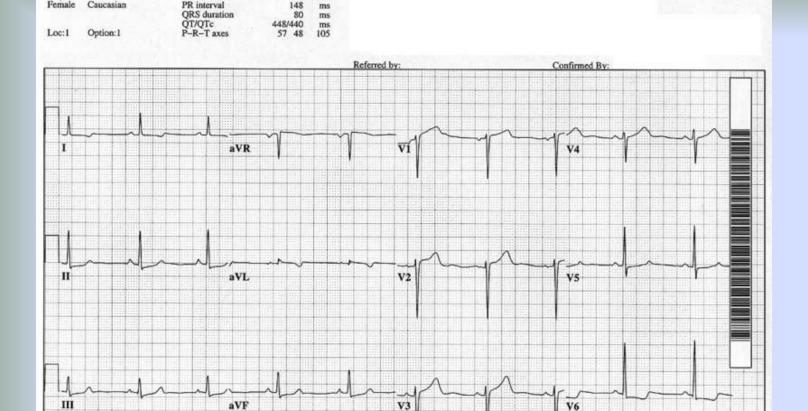


- 1. ECG abnormality(ies)? ST Elevation Lead AVR, Global ST Depression (I, II, III, AVL, AVF, V2, V3, V4, V5, V6)
- 2. Possible diagnosis? possible LMCA or 3x vessel disease.
- 3. Action / Intervention? Troponins, Continuous ST monitoring, cath lab visit STAT or ASAP (based on sympt.)





- 1. ECG abnormality(ies)?
- 2. Possible diagnosis?
- 3. Action / Intervention?



- 1. ECG abnormality(ies)? Inferior (II, III, AVF) ST Depr (ischemia?), I & AVL T wave inversion, V5 ST Depr
- 2. Possible diagnosis? Inferior / Lateral ischemia
- 3. Action / Intervention? Serial ECGs / Troponins, additional diagnostic testing, cath lab

# Your thoughts, ideas, comments and feedback are welcome...

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Cell: 813-230-4747



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