



"The EMS 12 Lead – 101"

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

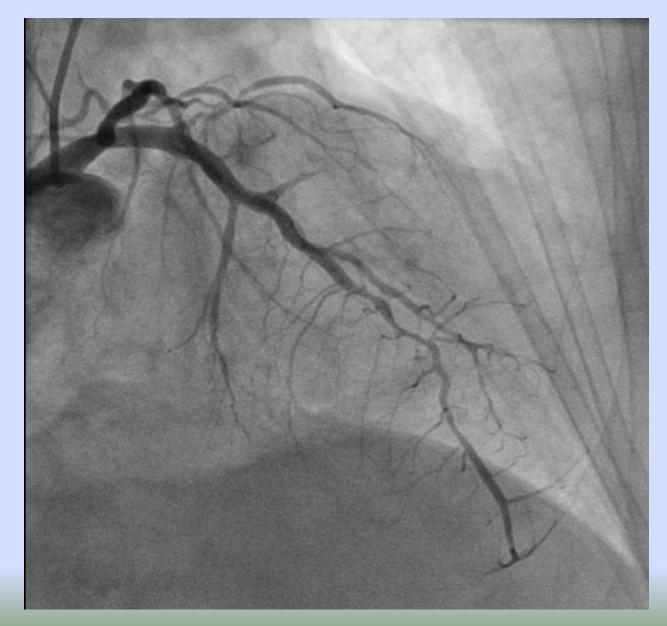


Welcome !





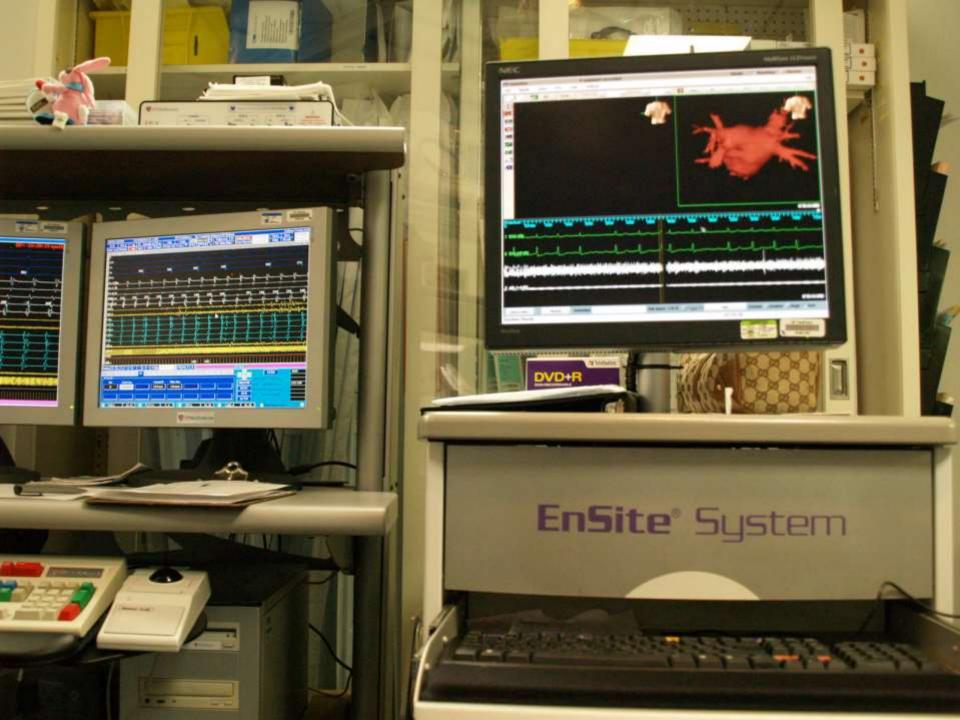
Cardiac Cath Lab



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: "<u>12 Lead ECG Interpretation in Acute</u> <u>Coronary Syndrome with Case Studies from the</u> <u>Cardiac Cath Lab</u>," 2010, TriGen publishing / Ingram Books
- Author of: "<u>STEMI Assistant</u>," 2014, TriGen publishing / Ingram Books
- Florida Nursing CE Provider # 50-12998
- 12 Lead ECG Instructor, 1994-present (multiple hospitals, USF College of Medicine 1994)
- Website: <u>www.ECGtraining.org</u>

In the CARDIAC CATHETERIZATION LAB, we read our patients' 12 Lead ECGs and then evaluate their commary interies and semicolar function during angiography. Stated at plan English, we republy beam how to correlate 12 lead ECG findings with what's ready going on inside our patients' hours. Secure ECGs from this perspective adds a new dimension to understanding the complex pathophysiologies of cardiovascular disease.

This book prepares you to:

- INTERPRET 12 Load ECGs.
- ASSIMLATE DATA derived from the 12 Lead ECG into a comprehensive patient evaluation process
 designed to maximize diagnostic accuracy, while taking into consideration the 12 Lead ECGs inherent
 LACK of SENSITIVATY 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 property ou to ANTICIPATE the FAILURE OF CRITICAL CARRIAGE STRUCTURES – othen 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 patients and mode their 12 load ECGs:

- Fellows in Envergency, Cardiciagy, and Family Medicine
- Medical Residents
- Veturan Physicians wanting a post review in ACS patient evaluation
- Physician Assistants and Marso Practitioners
- Energency Department Notes
- Coronary Care Unit and Cardiac Televerty Narona
- Walk-in Clinic Physicilans and Marines
- Paramentics

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

Joseph P. Ornato, MD, FACP, FACC, FACEP

- Professor and Chairman, Department of Emergency Medicine
- Medical College of Virginia/Virginia Commonwealth University
- Medical Director, Richmond Ambulance Authority,
- Richmond, Virginia

"This book integrates academic ECG principles with relat-world clinical practice by incorporation of well chosen cath lab case studies into its curriculum. This combination left readers are patients and their ECGs through the eyes of an expense col cath lab Instructionalist, and provides a balanced approach to patient evaluation that compressions for the IICGs interent lack of sensitivity and specificity. I highly recommend this book for all Emergency Medicine and Cardiology Fellows. For experienced clinicians, it's a support newlew."

Humberto Coto, MD, FACP, FACC

 Chief of Interventional Cardiology St. Joseph's Hospital Tampe, Florida



THE CATH LAB SERIES presents

LEAD

ECG

INTERPRETATION

3

ACUTE

STUDIES

븅

CATH

AB

.

12 LEAD ECG

INTERPRETATION

ACUTE

CORONARY

with CASE STUDIES from the

SYNDROME

CARDIAC CATHETERIZATION LAB

WAYNE W RUPPERT

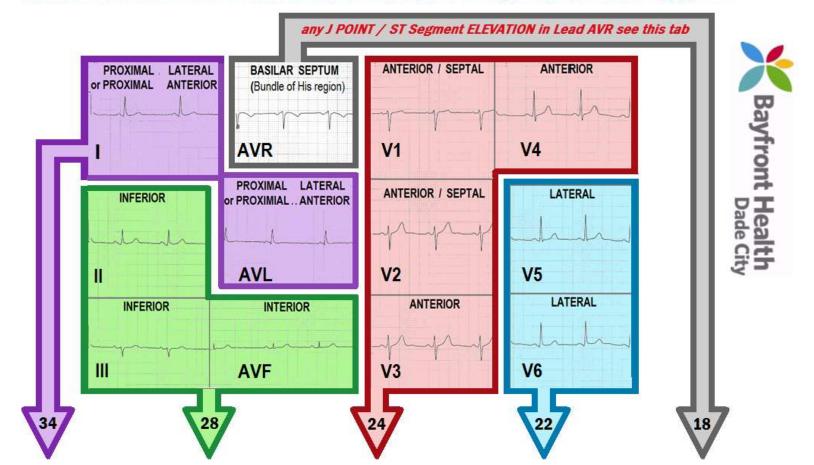
www.TriGenPress.com www.ECGtraining.org BarnesandNoble.com Amazon.com



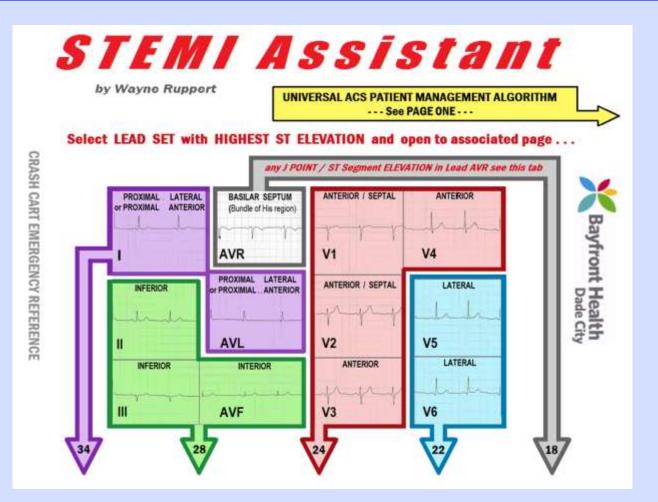
by Wayne Ruppert

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

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



www.TriGenPress.com www.ECGtraining.org BarnesandNoble.com Amazon.com **STEIL ASSISTANC:** an Emergency Crash Cart Interactive Reference Manual - free Download



STEMI Assistant – Information Video

"<u>STEMI Assistant</u>" Editorial Board:

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

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

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

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

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

Copyright 2010, 2011, 2015, 2019

All cardiovascular subject-related images, graphics and diagrams were created by the author, Wayne Ruppert, and have been taken from his two published textbooks, "STEMI Assistant" and "12 Lead ECG Interpretation in ACS with Case Studies from the Cardiac Cath Lab," are Copyright protected, and may not be removed from this PowerPoint presentation. This presentation may not be used as part of a profitgenerating program without prior written consent from the author.

Wayneruppert@aol.com



American College of Cardiology 20th 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

www.practicalclinicalskills.com www.skillstat.com/tools/ecg-simulator www.ECGtraining.org Download Today's Presentation To go: <u>www.ECGtraining.org</u> from the MENU BAR at left side of screen, select "Downloads PDF," and then select:

"EMS 12 Lead 101"

WWW.ECGTRAINING.ORG HELPFUL PDF DOWNLOADS

HOME

12 LEAD ECG IN ACS

STEMI ASSISTANT

ACCREDITATION

WORKSHOPS

ECG ID OF SADS

WORKSHOP OBJECTIVES

TEXTBOOKS

PHYSICIAN REVIEWS

BIO OF WAYNE RUPPERT

TESTIMONIALS

DOWNLOADS - PDF

HELPFUL INFORMATION

CONTACT US

All materials featured on this page are copyright protected. This content is offered for INDIVIDUAL USE by Medica manner and/or printed for sale or distribution without prior written consent of the author. EXCEPTION: Physicians owned hospitals and all EMS agencies who routinely serve CHS hospitals may download, reproduce and distribute
Download EMS 12 Lead 101 - 2019
Download BHSR 2019 Basic ECG with Obtaining STAT 12 Lead

Download Hands-Only CPR and AED Course

Download The Lifesaving ECG Part 1

Download The Lifesaving ECG Part 2

Download Advanced 12 Lead ECG in ACS and SADS Key West 2018

Download Basic ECG - Key West 2018

Download 12 Lead ECG - 8 hour class - Part 1 - 2018

Download 12 Lead ECG - 8 hour class - Part II - 2018

Download Advanced Physicians ACLS 2018 SRRMC

Download ACC 20th Congress Serial 12 Lead ECG Course

Download A SHORT Course in LONG QT Syndrome

Download ACC 20th Congress - Continuous ST Segment Monitoring Course

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

Download ACC 20th Congress - Serial 12 Lead ECG Interpretation Part 2

Download Sudden Cardiac Death Prevention - ACC / SCPC 19th Congress

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



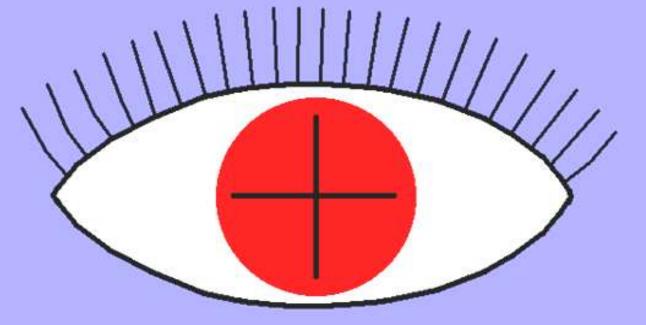
THE ECG MACHINE

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

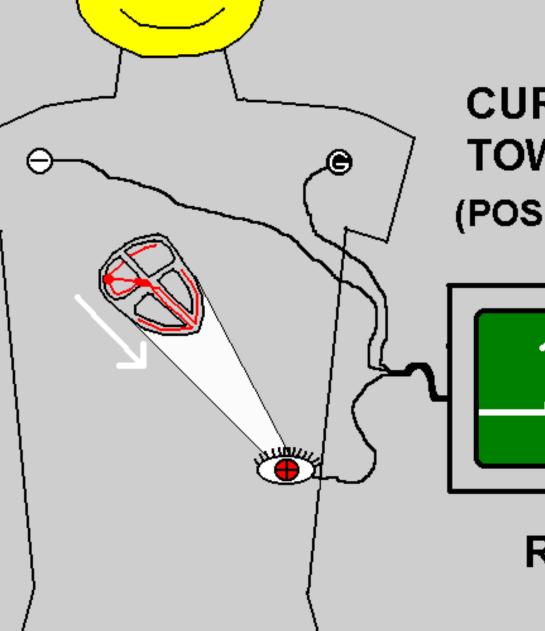
G

- LEADS I, II, III, and V1, V2, V3, V4, V5, V6
 - 1 POSITIVE ELECTRODE -
 - 1 NEGATIVE ELECTRODE -
 - **1 GROUND ELECTRODE**
- LEADS AVR, AVL, and AVF
 - **1 POSITIVE ELECTRODE**
 - **2 NEGATIVE ELECTRODES**
 - 1 GROUND ELECTRODE

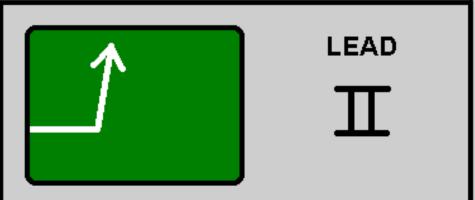
IS THE "EYE" . . .



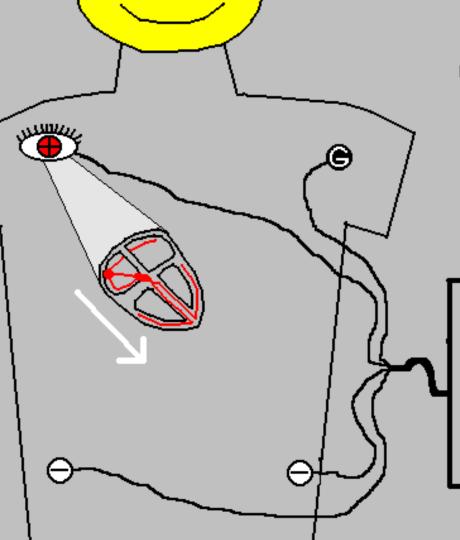
THE POSITIVE ELECTRODE



CURRENT MOVING TOWARD THE EYE (POSITIVE ELECTRODE)



RECORDS AN "UPWARD" DEFLECTION

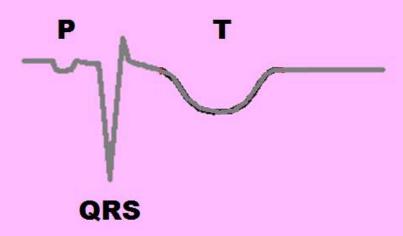


CURRENT MOVING AWAY FROM THE EYE (POSITIVE ELECTRODE)

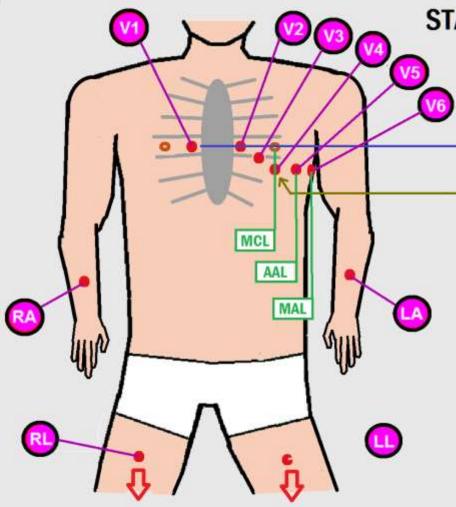


RECORDS A "DOWNWARD" DEFLECTION





EVERYTHING IS "UPSIDE-DOWN"



STANDARD LEAD PLACEMENT ---12 LEAD ECG

4 th INTERCOSTAL SPACE

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

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

• 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 property infants and

Kligfield et al Standardization and Interpretation of the ECG, Part I

AHA/ACC/HRS Scientific Statement

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

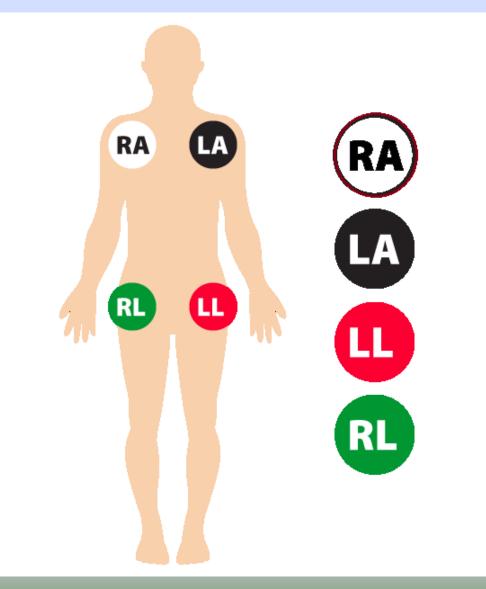
Recommendations

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.

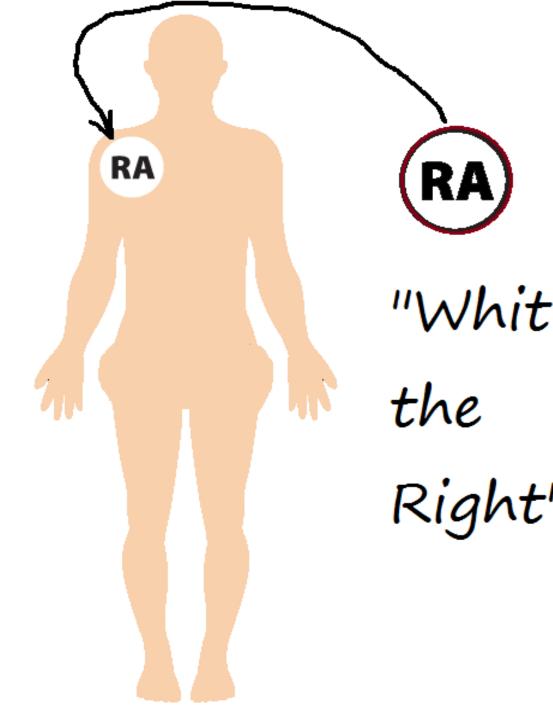
- Limb leads should be on the limbs.
- When emergency circumstances dictate that limb leads be placed on patient's torso, the words "LIMB LEADS ON PATIENT'S TORSO" should be noted on the ECG.
 - However in the field, it may not be possible or practical to put limb leads on limbs!

Limb leads – field ECG



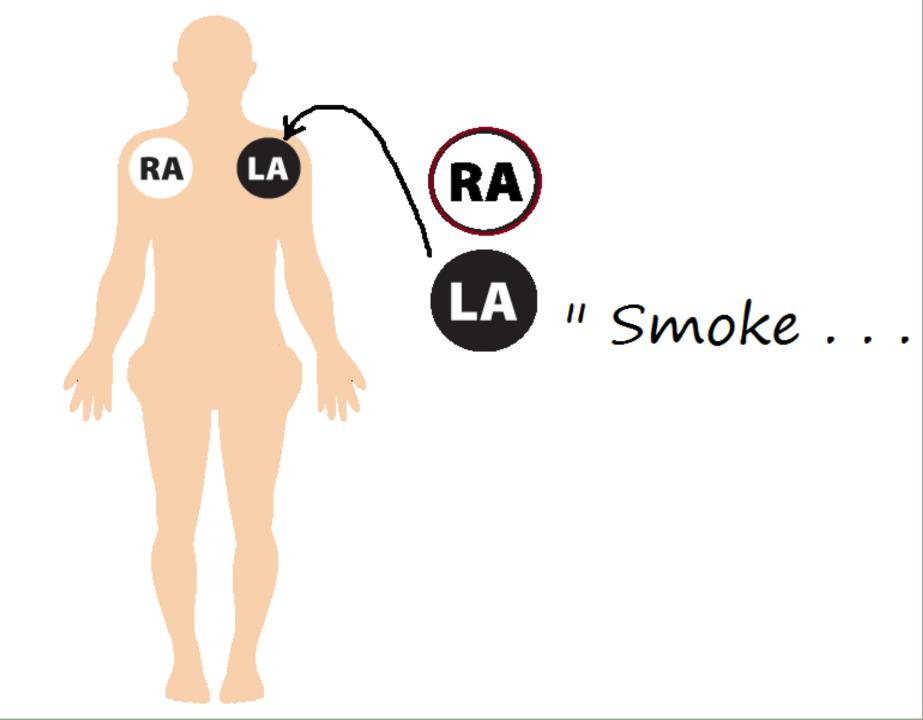
Remember this . . .

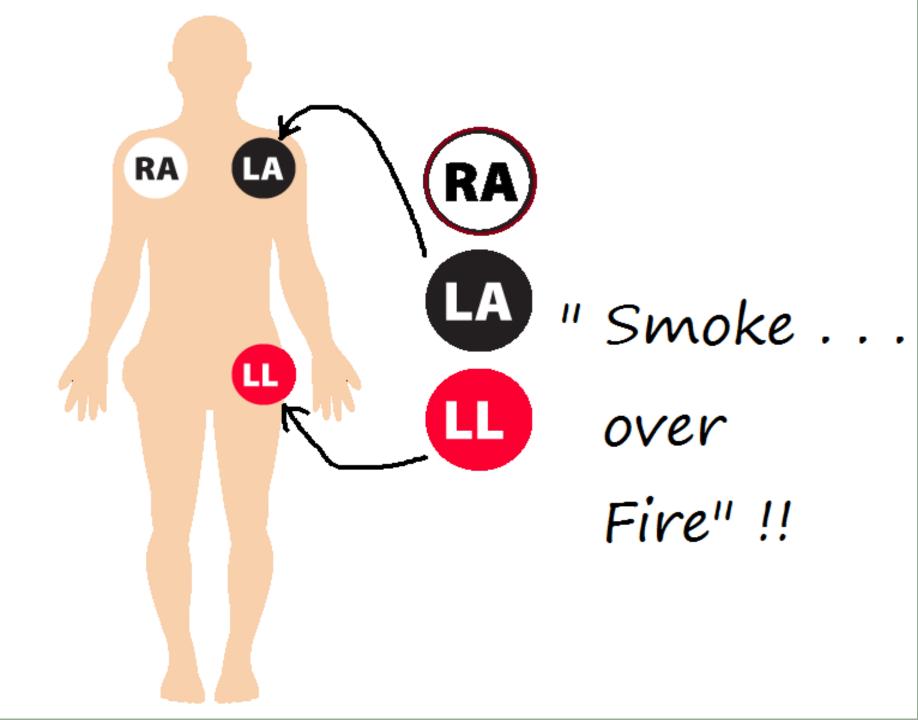
- White to the Right
- Smoke over Fire
- Green Grass below the White Clouds

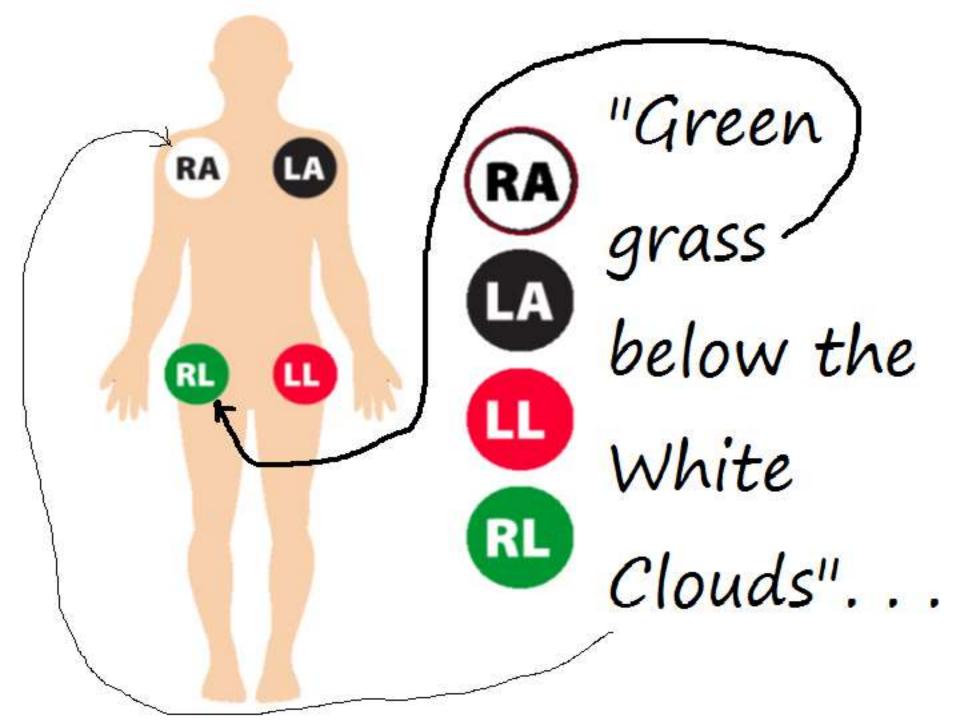


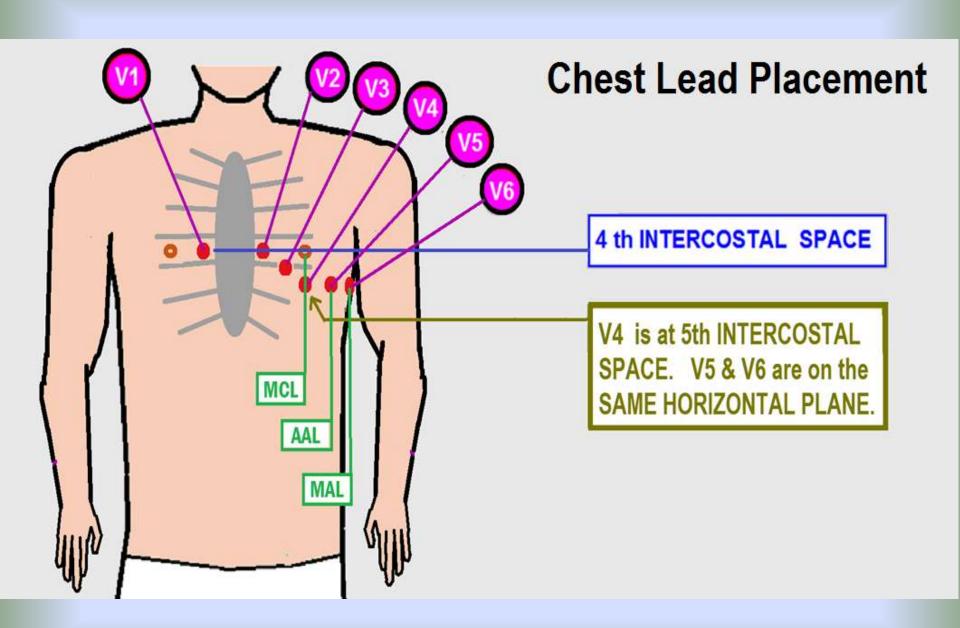
"White to

Right"









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

1.1

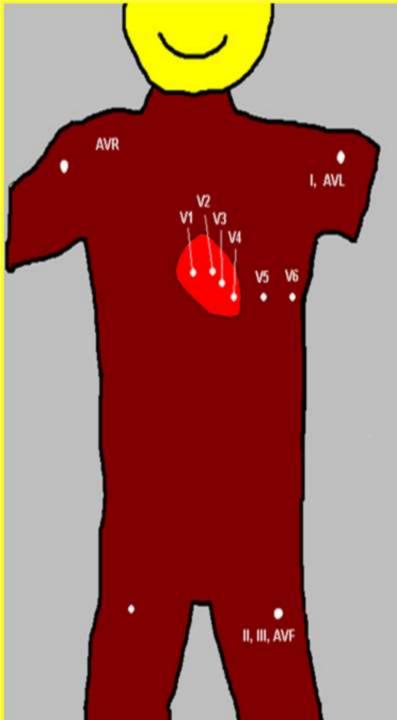
the often profound alterations in waveforms that can result from precordial electrode misplacement.^{85,86} A common error is superior misplacement of V_1 and V_2 in the second or third intercostal space. This can result in reduction of initial R-wave amplitude in these leads, approximating 0.1 mV per 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

Kligfield et al Standardization and Interpretation of the ECG, Part I

Obtaining the 12 Lead ECG, etc

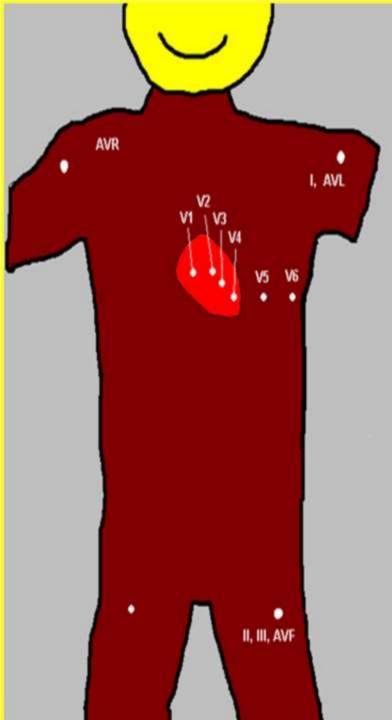
- Patient should lay as flat as possible
- If you see too much "artifact" on the ECG, you may have to coach patient to "relax all of your muscles"...
 - Arms
 - Legs
 - Chest
 - Abdomen, etc.

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



AREAS VIEWED by 12 LEAD ECG

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



AREAS VIEWED by 12 LEAD ECG

AVR	BASILAR SEPTAL	
AVL, I		

V1, V2 ANTERIOR

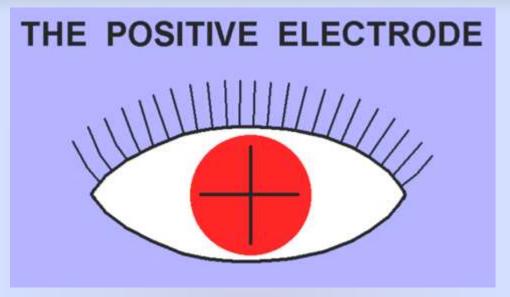
SEPTAL

POSTERIOR (recip.)

V3, V4	ANTERIOR

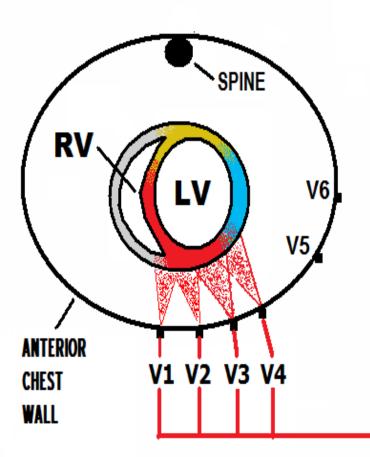
V5, V6 LATERAL

II, III, AVF INFERIOR

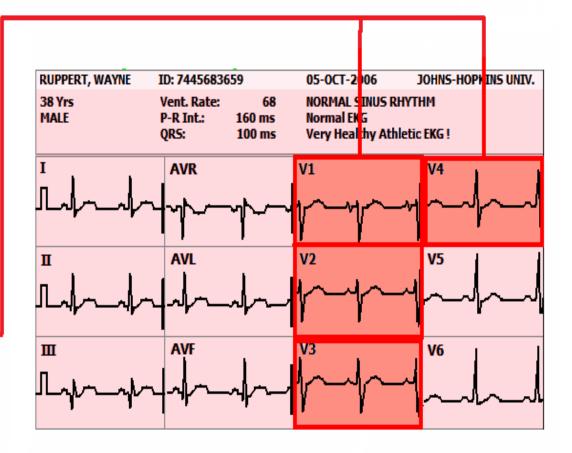


What each of the 12 Leads "sees," in more detail . . .

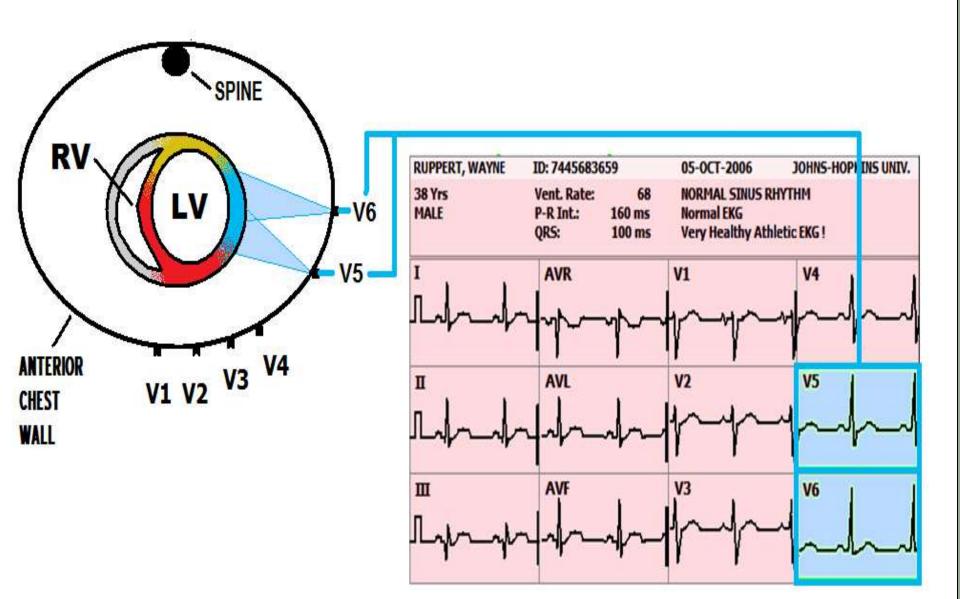
V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL of the LEFT VENTRICLE

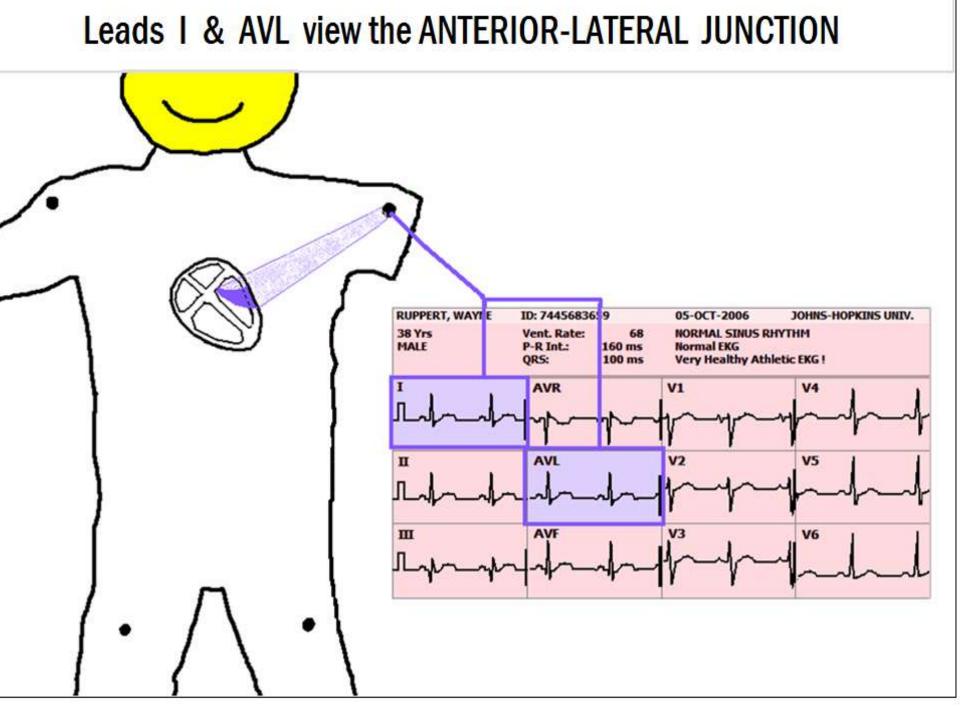


V1, V2 - ANTERIOR / SEPTAL V3, V4 - ANTERIOR

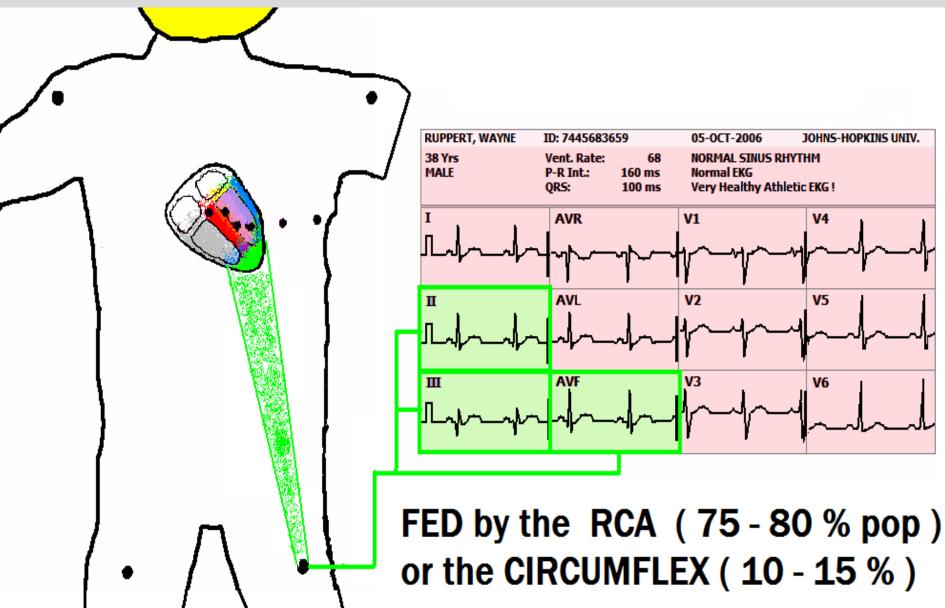


V5 - V6 VIEW THE LATERAL WALL of the LEFT VENTRICLE

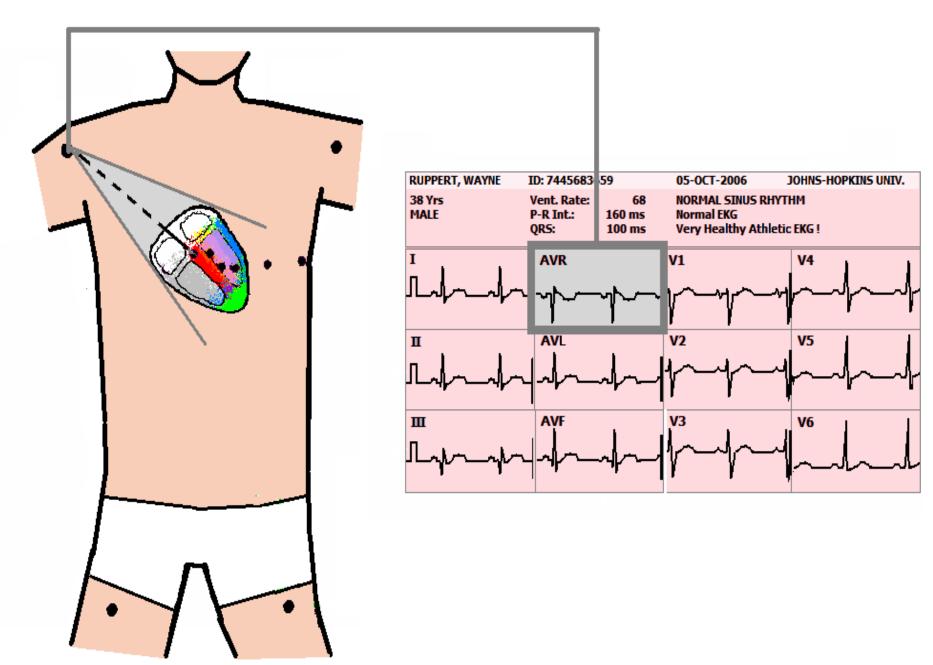


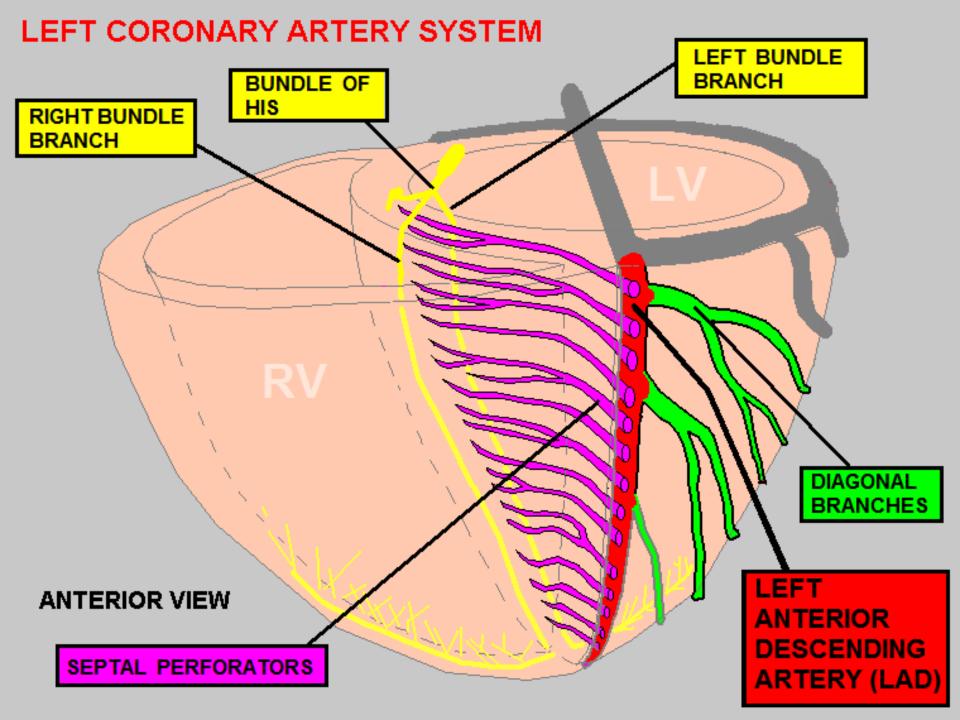


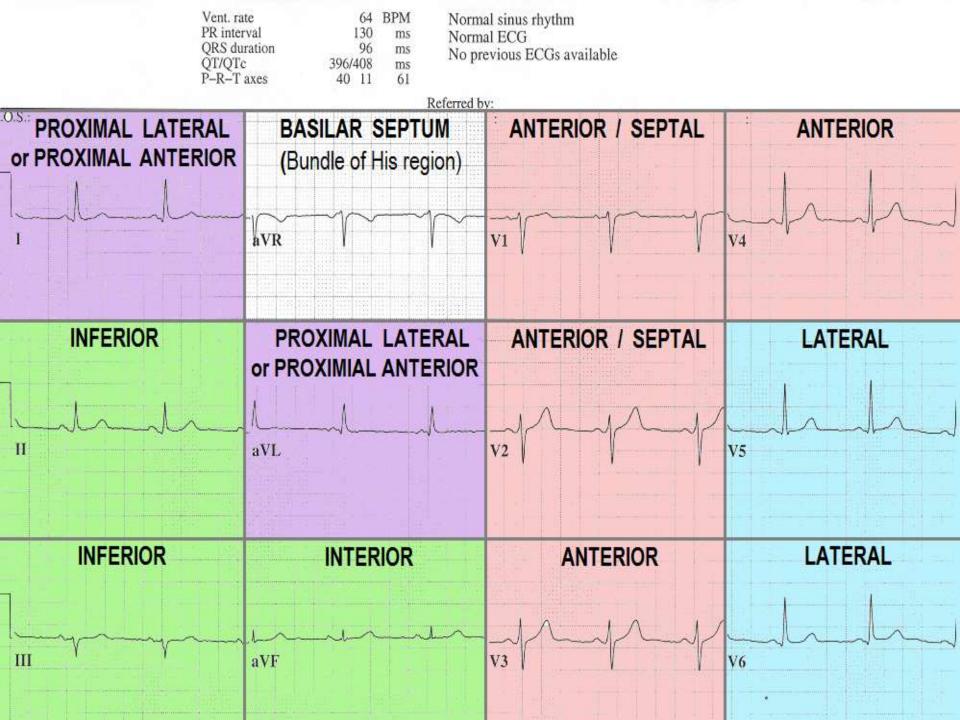
LEADS II, III, and aVF VIEW INFERIOR WALL of the LEFT VENTRICLE



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

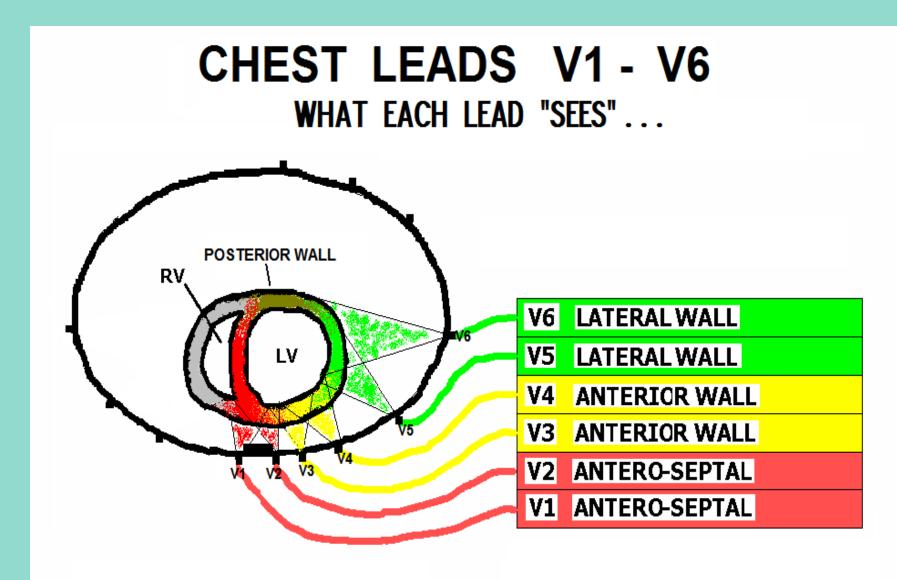




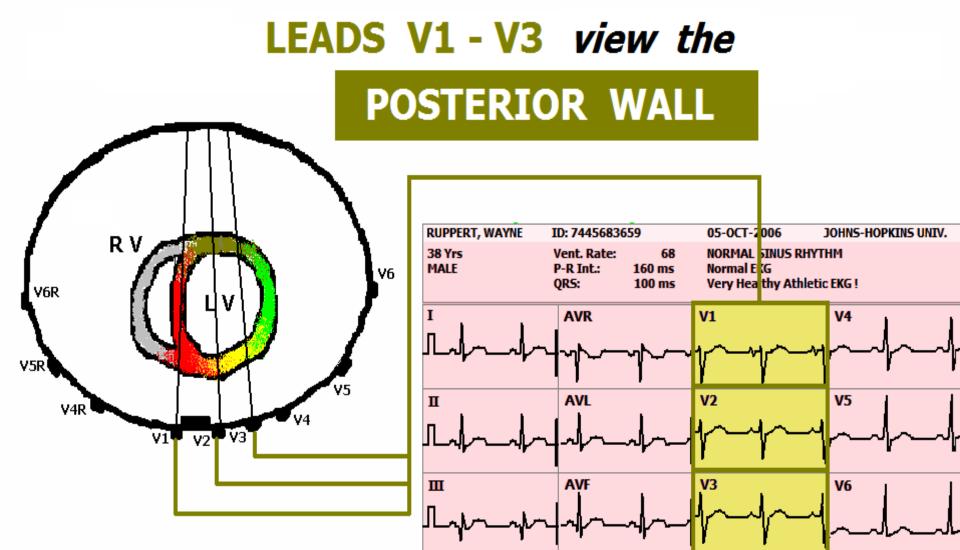


A 12 Lead ECG sees MOST of the Heart, but it has some BLIND SPOTS !!

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



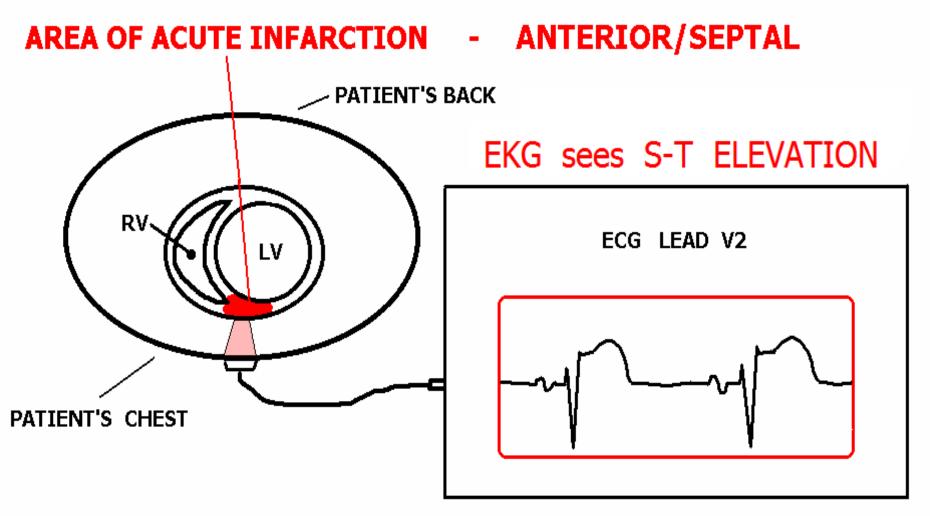
Regarding the **POSTERIOR WALL**, the Standard 12 Lead will give you some "good clues" that there's a PROBLEM by displaying what we call "RECIPROCAL CHANGES" kind of like seeing "reflected changes" in a mirror.....



via RECIPROCAL CHANGES.

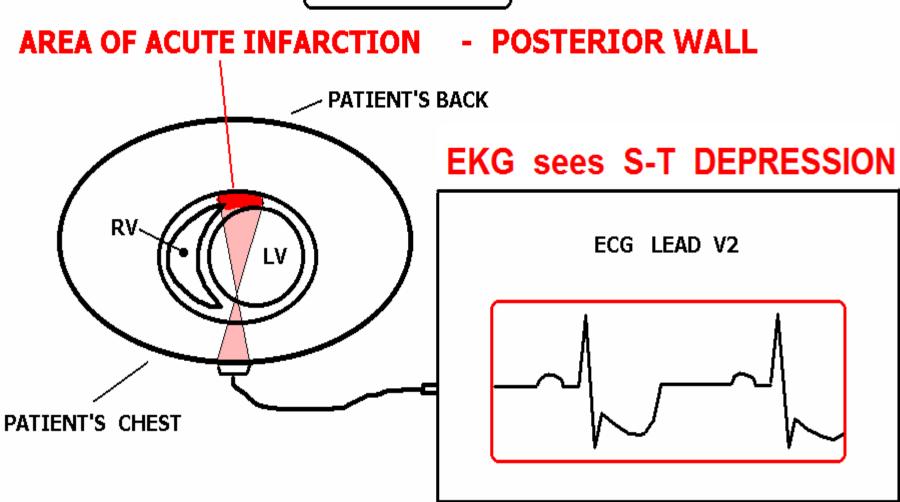
HOW EKG VIEWS INDICATIVE CHANGES





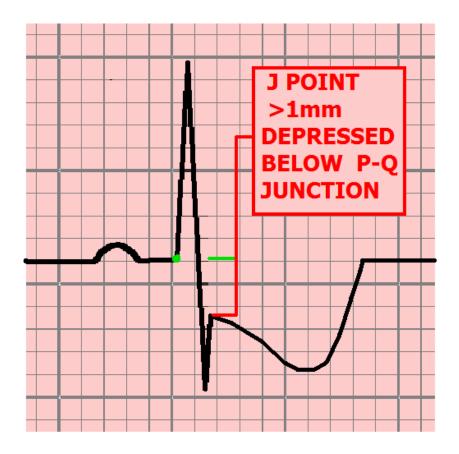
HOW EKG VIEWS RECIPROCAL CHANGES

EXAMPLE:



ST DEPRESSION can be from any other these conditions:

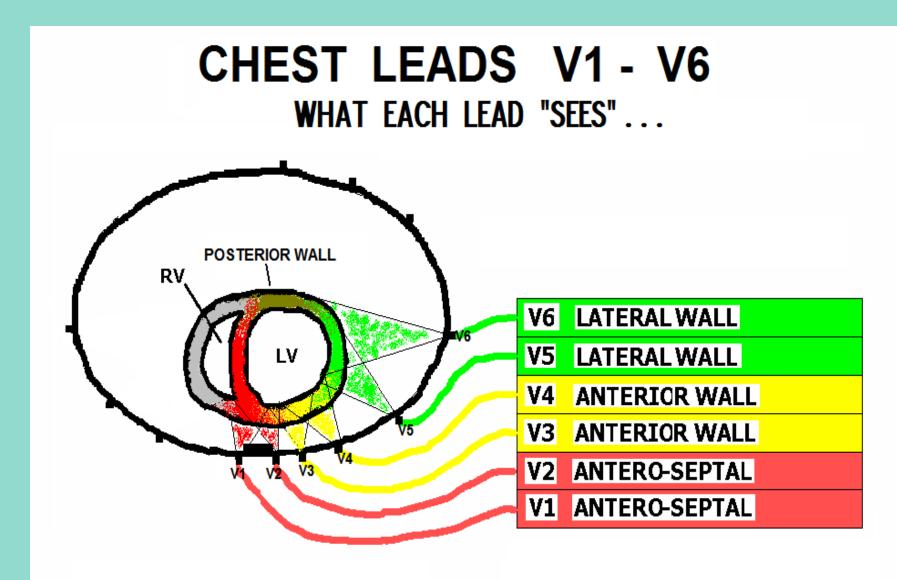
S-T SEGMENT DEPRESSION - COMMON ETIOLOGIES:



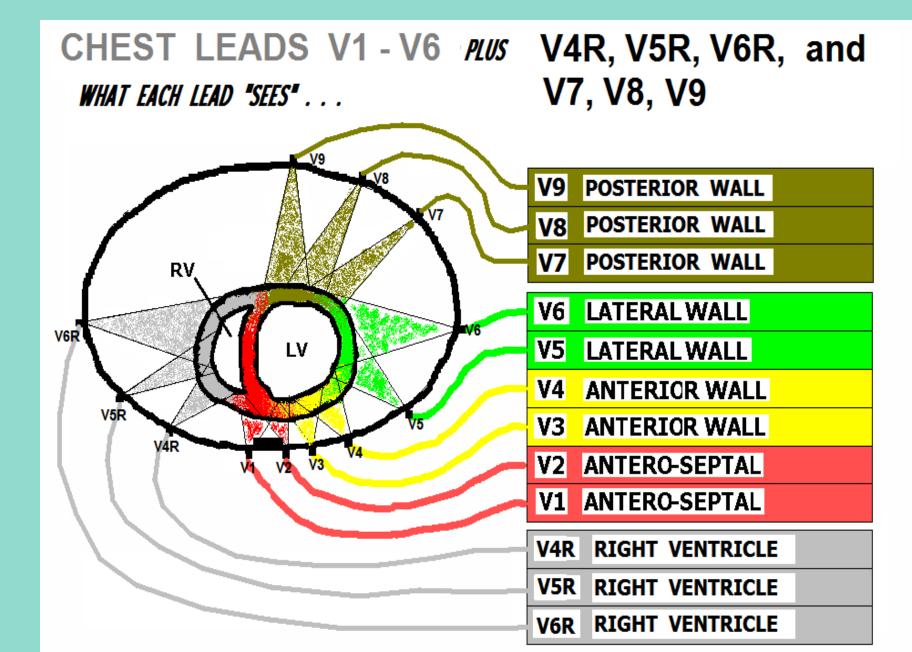
CONDITION:

- 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

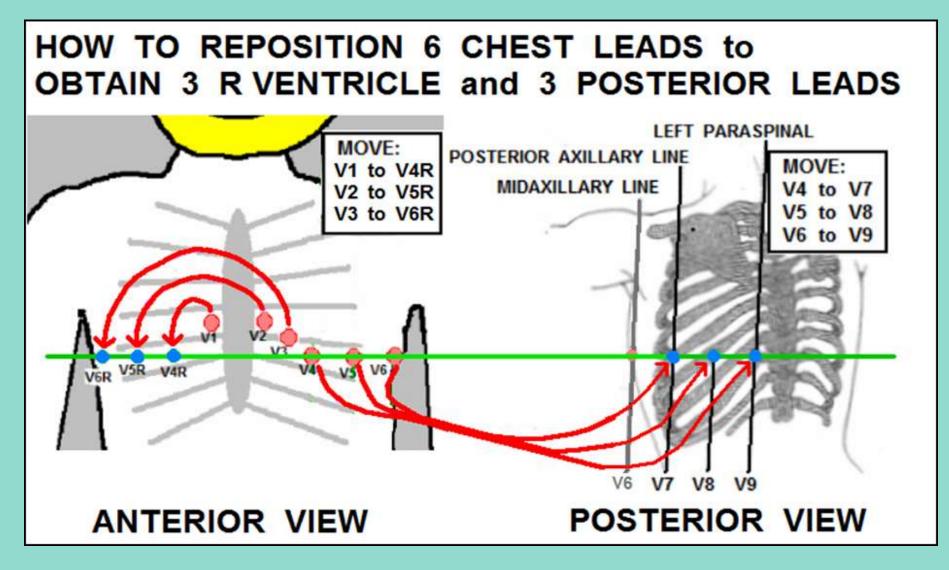
THE 12 LEAD ECG HAS TWO MAJOR BLIND SPOTS ...



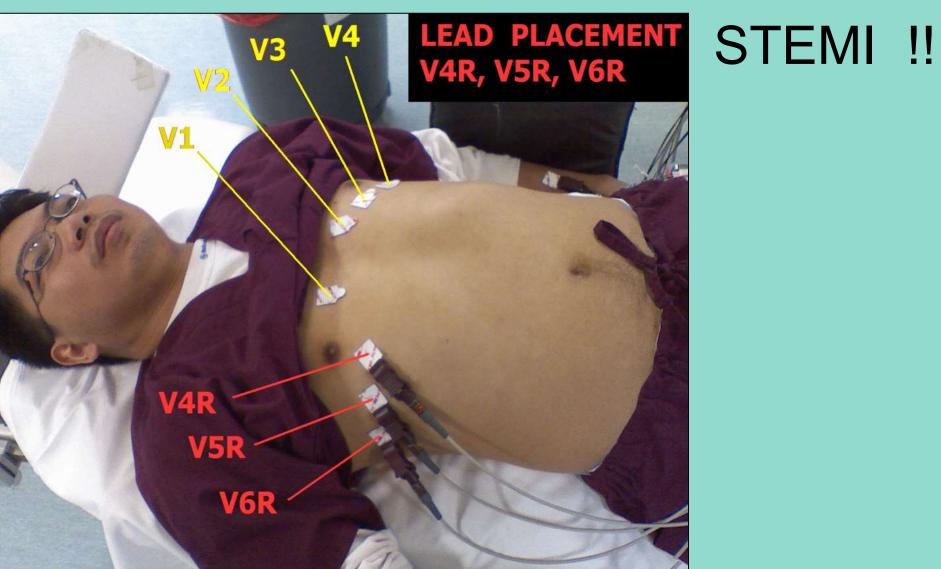
THE 18 LEAD ECG COVERS THE ENTIRE HEART ...



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

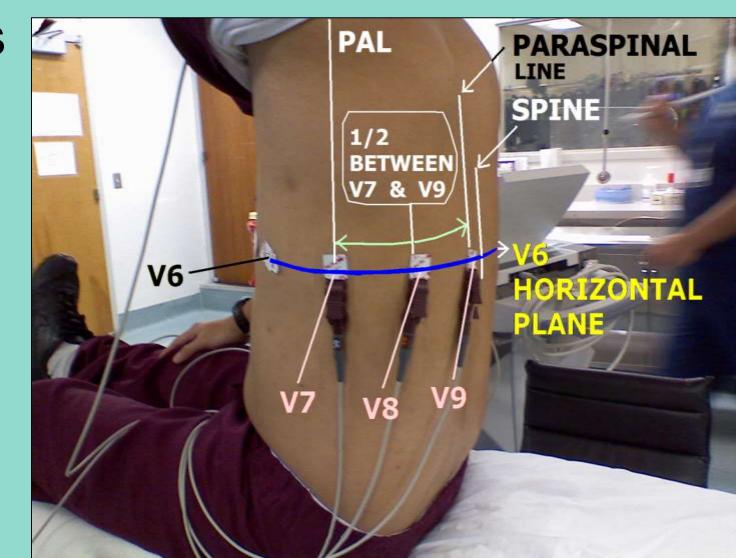


RIGHT SIDED ECG is indicated whenever you see INFERIOR WALL

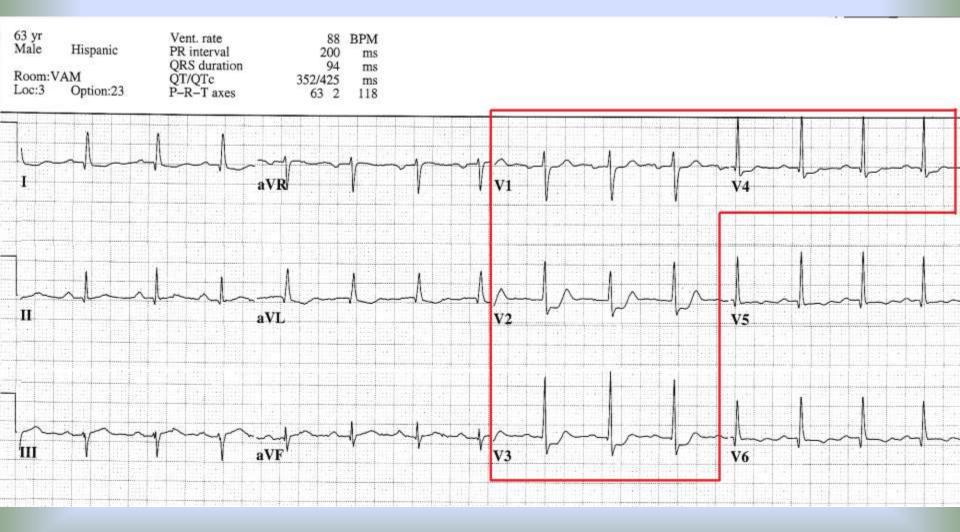


POSTERIOR ECG is indicated whenever you see ST DEPRESSION

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:

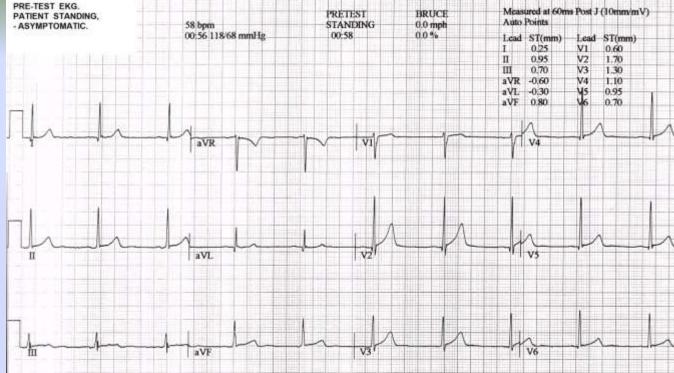
SENSITIVITY (FALSE NEGATIVES)

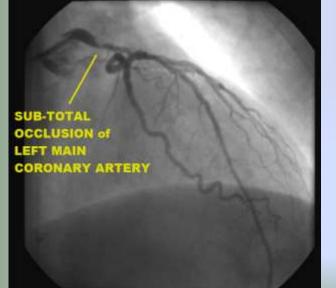
SPECIFICITY
(FALSE POSITIVES)

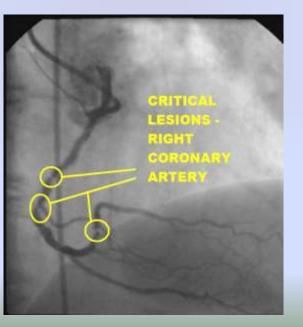
PROBLEMS WITH SENSITIVITY

NORMAL ECG.

But

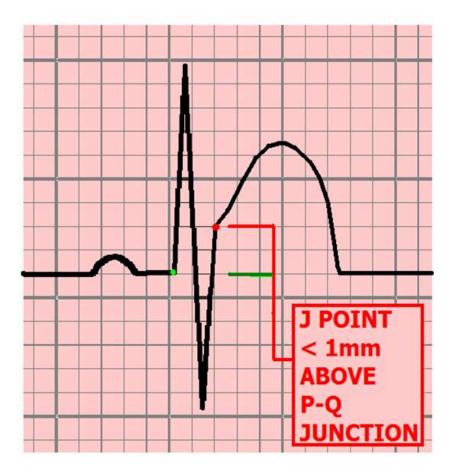






LETHAL TRIPLE VESSEL DISEASE

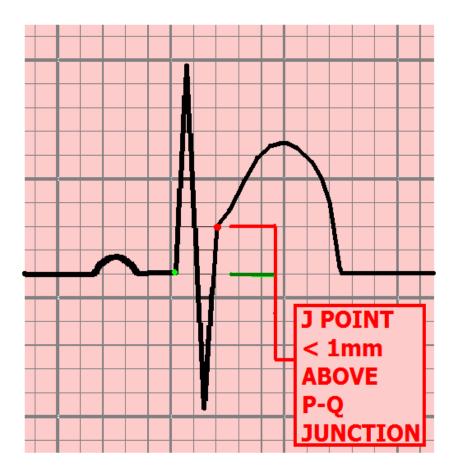
S-T SEGMENT ELEVATION - COMMON ETIOLOGIES:



CONDITION:

• ACUTE INFARCTION (STEMI)

S-T SEGMENT ELEVATION - COMMON ETIOLOGIES:

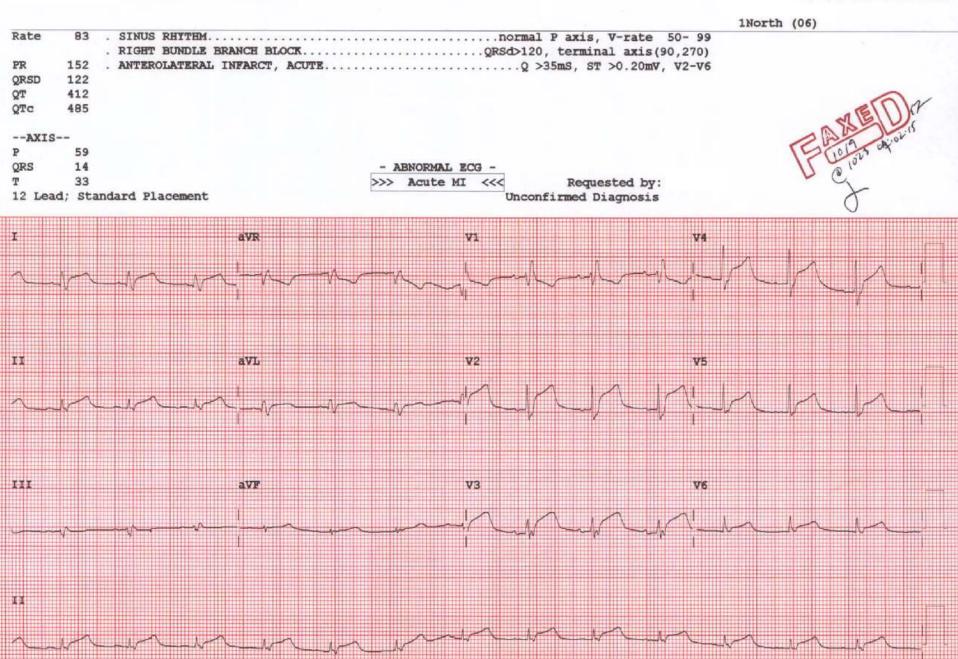


CONDITION:

- 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.

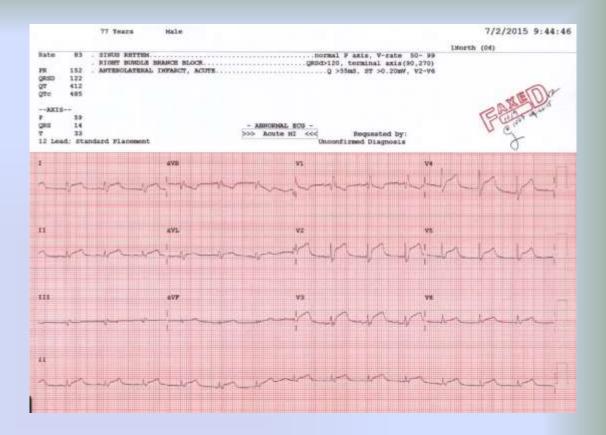
77 Years Male

7/2/2015 9:44:46



Patient:

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



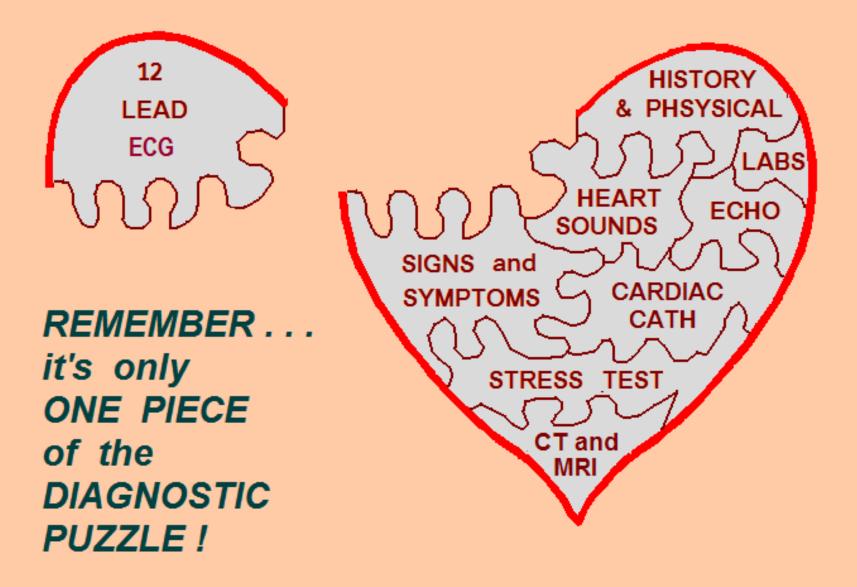
EARLY REPOLARIZATION. This degree of ST Elevation in early repolarization is VERY RARE: The only such ECG I have seen in approximately 13,000 cardiac catheterizations.

Despite the ECG's problematic issues with Lack of Sensitivity 8 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



 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!!

The ECG . . .



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

The ECG . . .



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 is 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

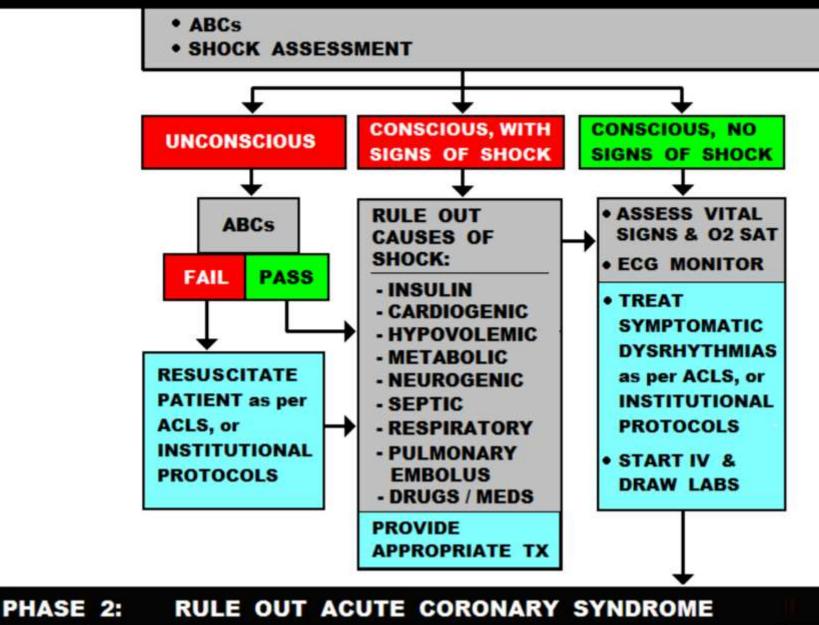
SHOCK ASSESSMENT

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
STATUS:	SHOCK SK	NORMAL

FAIL the SHOCK SURVEY?

RAPIDLY FIND AND TREAT
THE ROOT CAUSE ...

PHASE 1: RULE OUT LIFE-THREATENING CONDITIONS



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 is 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.



<u>TYPICAL SYPTOMS of</u> <u>Acute Cornary Syndrome:</u>

✓ CHEST PAIN - DESCRIBED AS ...

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

SHORTNESS OF BREATH

- MAY or MAY NOT BE PRESENT

NAUSEA / VOMITING

- MAY or MAY NOT BE PRESENT

INFARCTION

- - - "Classic Symptoms" - - -

QUICK ASSESSMENT "SHORT FORM"

SUBSTERNAL CHEST PAIN (HAVE PATIENT POINT TO WORST PAIN)

- DESCRIBED AS "DULL PAIN," "PRESSURE," or "HEAVINESS"
- DOES NOT CHANGE WITH DEEP BREATH

stable angina

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



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

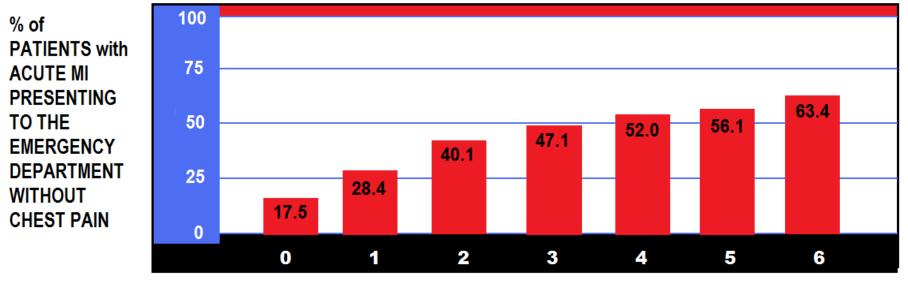
Common atypical complaints associated with AMI without chest pain include:

Malaise (weakness) Indigestion Nausea Dizziness Syncope

Fatigue Abdominal pain Cold sweats Elevated heart rate Dsypnea

BOOK PAGE: 70

Effect of Having Multiple Risk Factors for AMI Without Chest Pain



NUMBER OF RISK FACTORS PRESENT

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

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

WOMEN'S MAJOR SYMPTOMS PRIOR TO THEIR HEART ATTACK:

- UNUSUAL FATIGUE 71 %
- SLEEP DISTURBANCE 48 %
- • SOB
 42 %

 • INDIGESTION
 39 %

 • ANXIETY
 36 %
- ANXIETY 36 %

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

WOMEN'S MAJOR SYMPTOMS DURING THEIR HEART ATTACK:

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

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

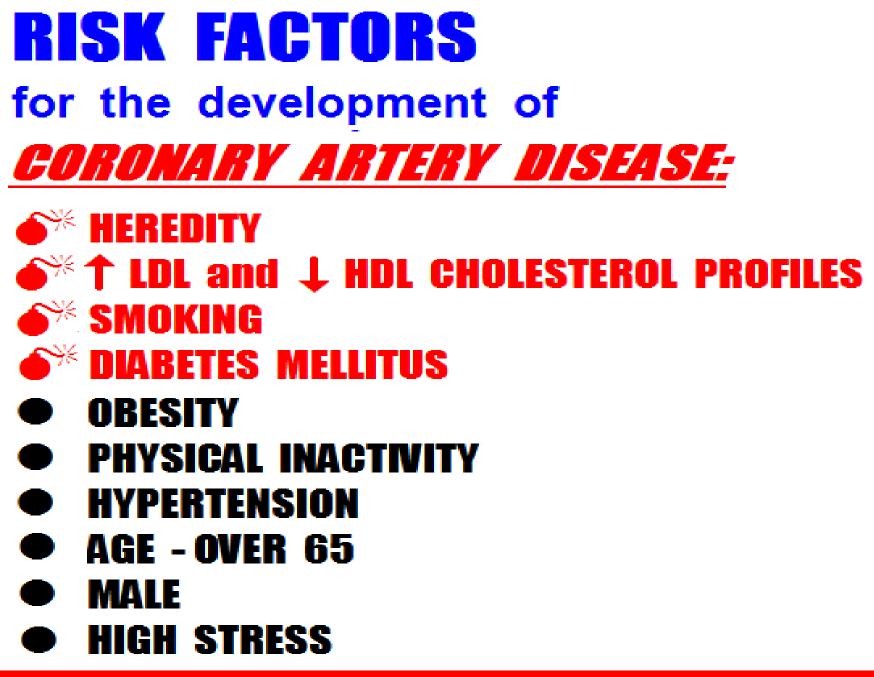
PRESENTING SYMPTOMS RISK FACTOR PROFILE ECG ABNORMALITIES CARDIAC MARKERS

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 is present</u>.... steps must be AGGRESSIVELY TAKEN to definitively RULE OUT the PRESENCE 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 present.... steps must be AGGRESSIVELY TAKEN to definitively RULE OUT the PRESENCE of ACS!



per the AMERICAN HEART ASSOCIATION



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	
	> 1 and < 3x normal limit	1	
	≤ 1x normal limit	0	
		Total	

*Risk factors for atherosclerotic disease:

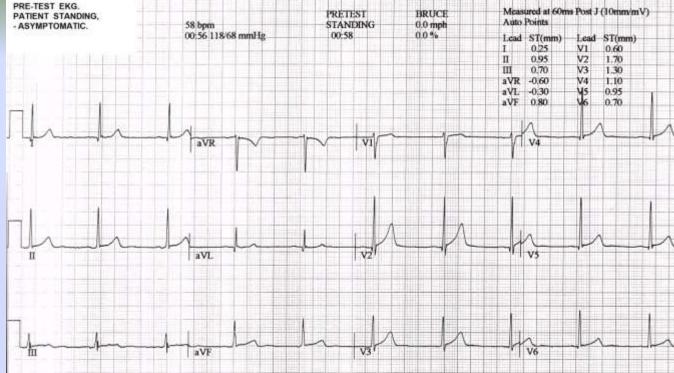
Hypercholesterolemia Hypertension Diabetes Mellitus Cigarette smoking Positive family history Obesity

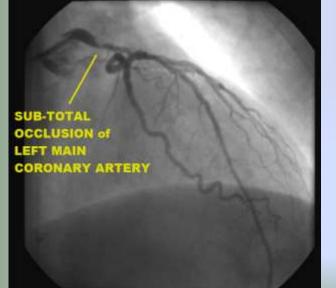
Here is the HEART Score! Let's see what the score is for our patient with the NORMAL ECGwho had **Critical Triple Vessel Disease and** needed STAT **Bypass Surgery!**

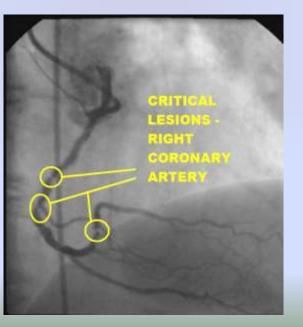
PROBLEMS WITH SENSITIVITY

NORMAL ECG.

But







LETHAL TRIPLE VESSEL DISEASE



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

*Risk factors for atherosclerotic disease:

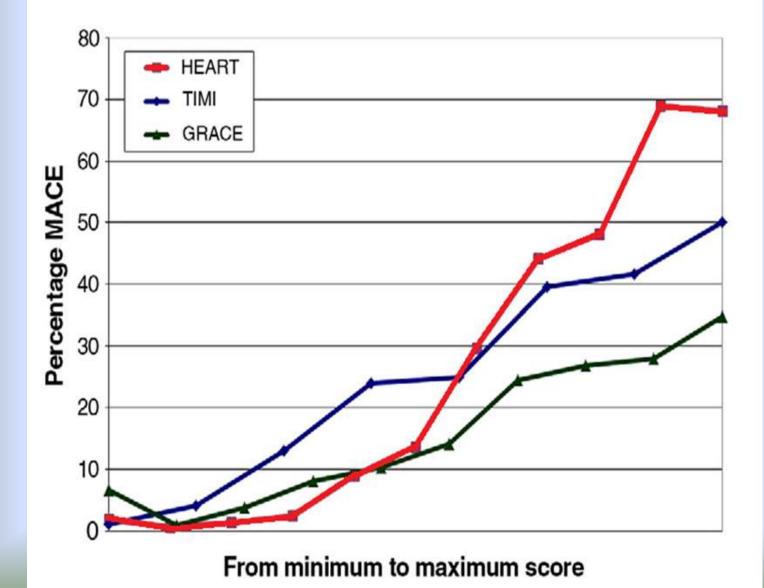
Hypercholesterolemia Hypertension Diabetes Mellitus

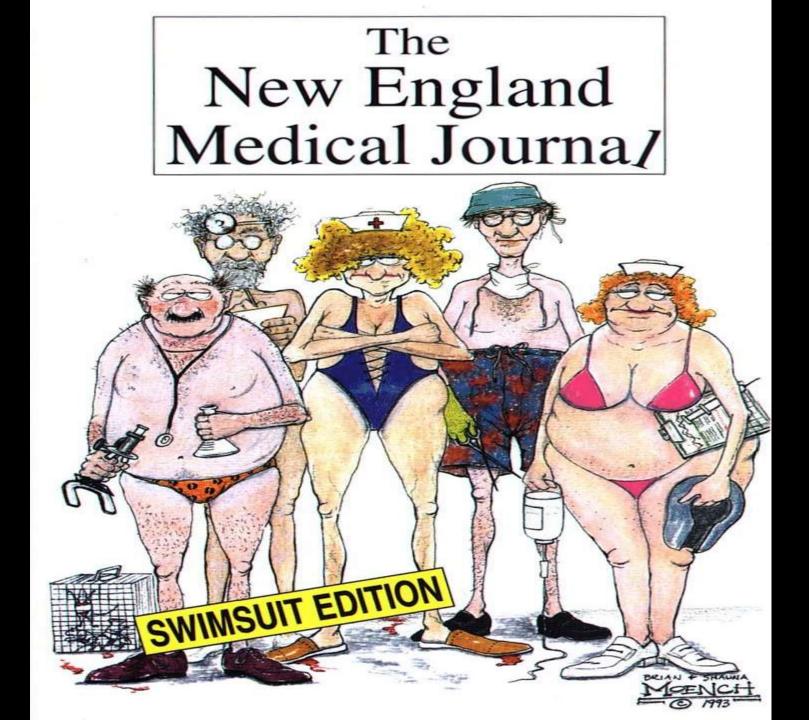
Cigarette smoking Positive family history Obesity

- **H** = chest pain = 2
- $\mathbf{E} = \mathbf{E}\mathbf{C}\mathbf{G}$ normal = 0
- **A** = 63 = 1
 - $\mathbf{R} = 3$ risk factors = 2
- \mathbf{T} = Trop. NL = 0

HEART Score: = 5

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





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

1. SYMPTOMS START DURING PHYSICAL EXERTION.

2.

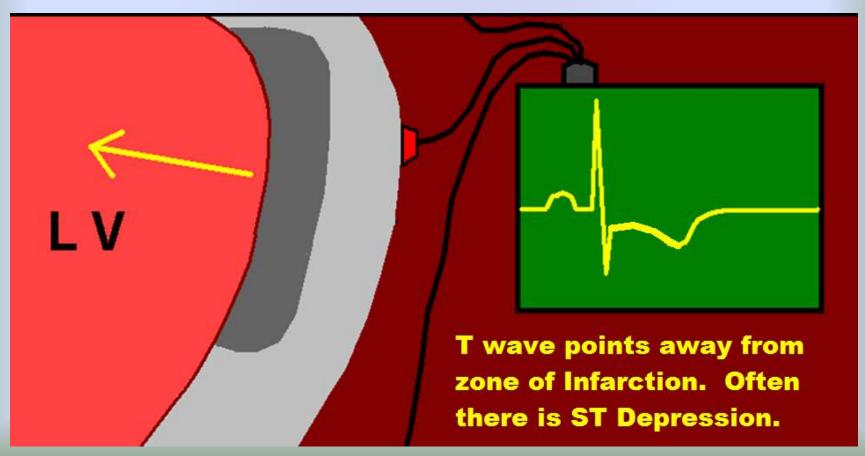
SYMPTOMS ARE



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:

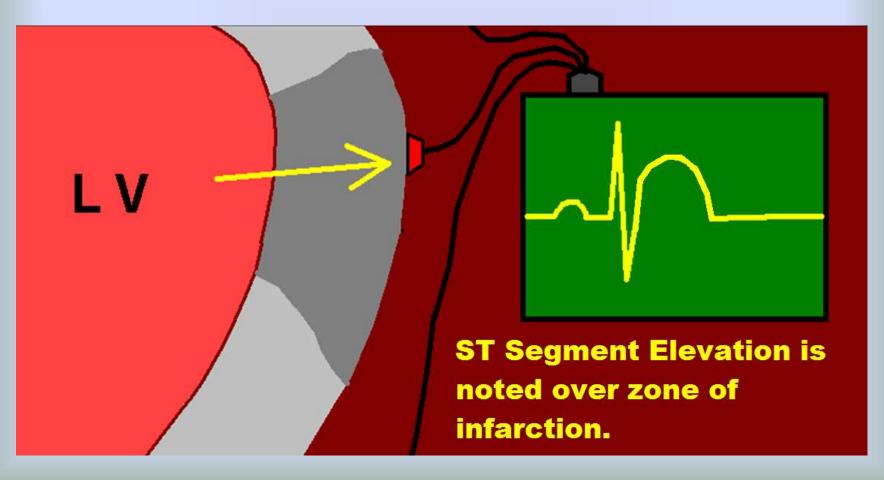
- ST Depression
- Other ST Segment changes
- Inverted T wave
- THE ECG MAY BE TOTALLY NORMAL.

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

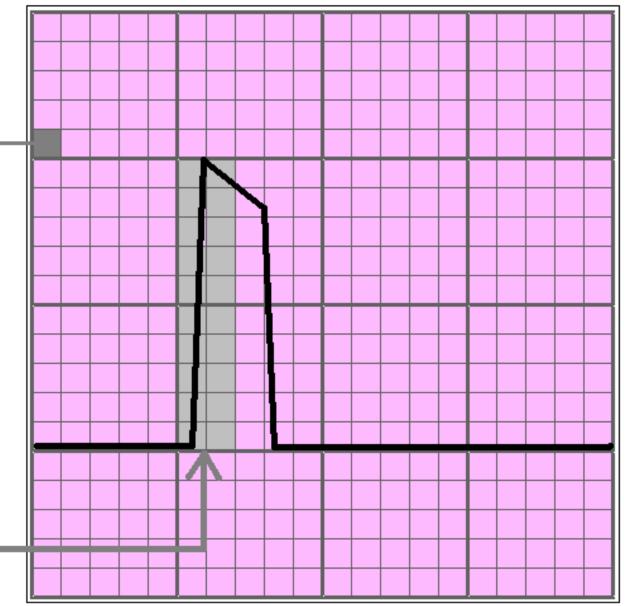
EACH 1mm BOX = .04 SECONDS, or 40 MILLISECONDS (40 ms)

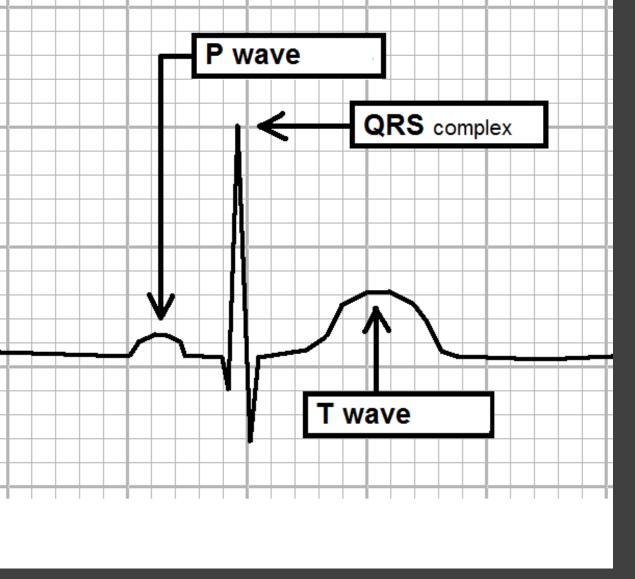
5 SMALL BOXES = .20 SECONDS, or 200 MILLISECONDS (200 ms)

-										
L ITS										
ED G										
)										_
<										
os							 			 _
i = or										
DS				 			 		 	 _

ECG PAPER - THE VERTICAL AXIS:

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





P WAVE = ATRIAL DEPOLARIZATION

QRS COMPLEX =

VENTRICULAR DEPOLARIZATION (contracting)

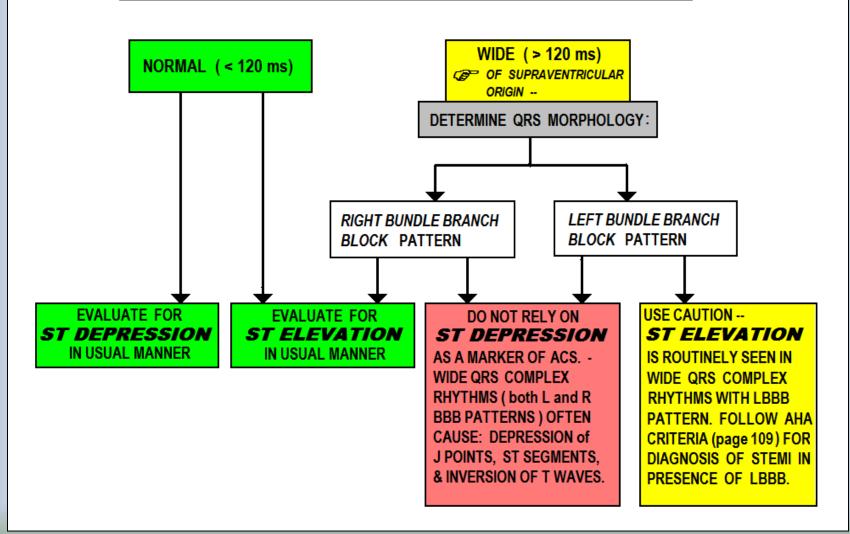
T WAVE = VENTRICULAR REPOLARIZATION (recharging) Q: To evaluate the patient for ischemia or infarction, what part of the ECG do we look at? Q: To evaluate the patient for ischemia or infarction, what part of the ECG do we look at?

..... in each lead !

- A: We evaluate the
- J Points
- ST Segments &
- T Waves

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:

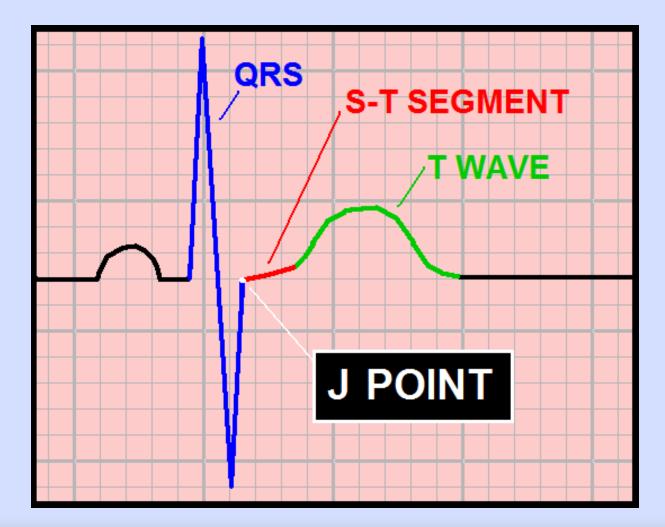
- J POINTS
- ST SEGMENTS
- T WAVES

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:

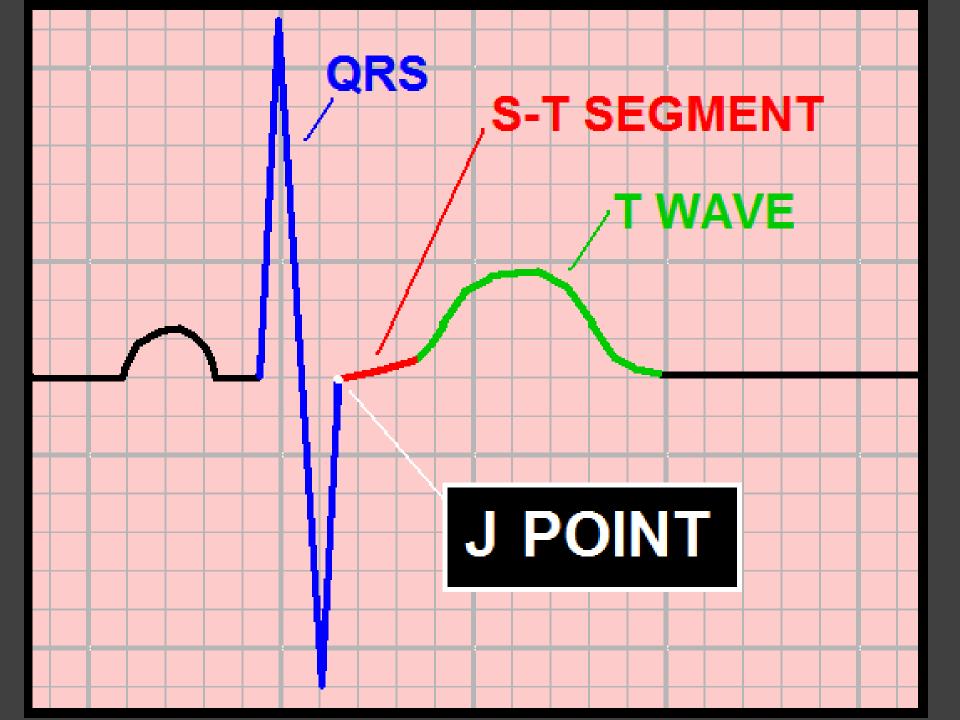


THE J POINT

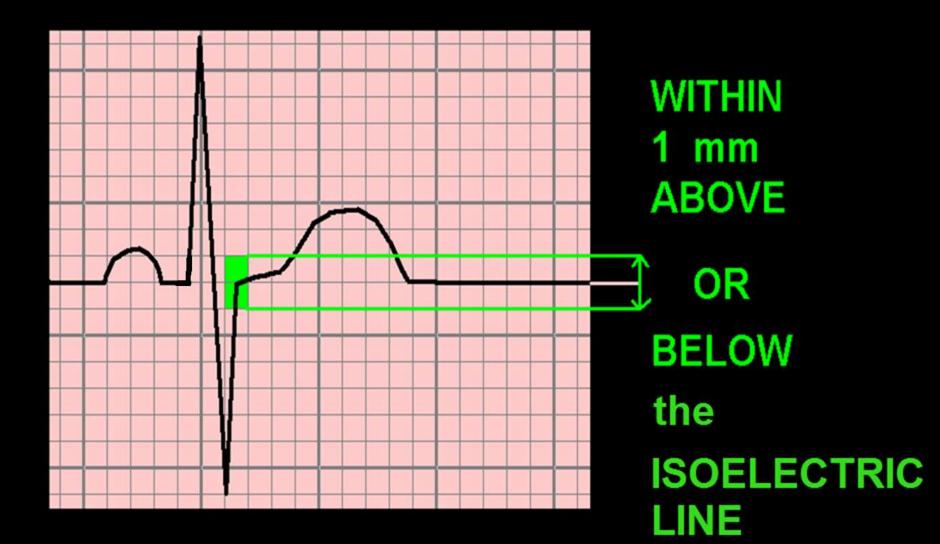
is where the **QRS** complex ends and the S-T Segment begins.

J POINT

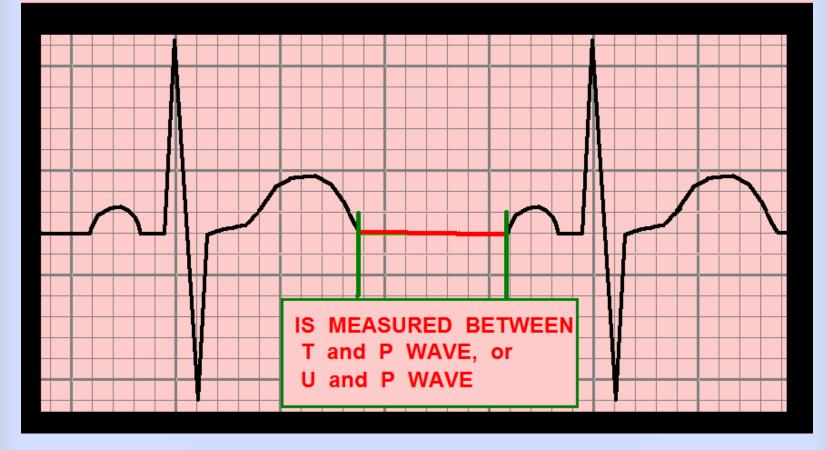
Extends from the J POINT to the T Wave



THE J POINT SHOULD BE ..

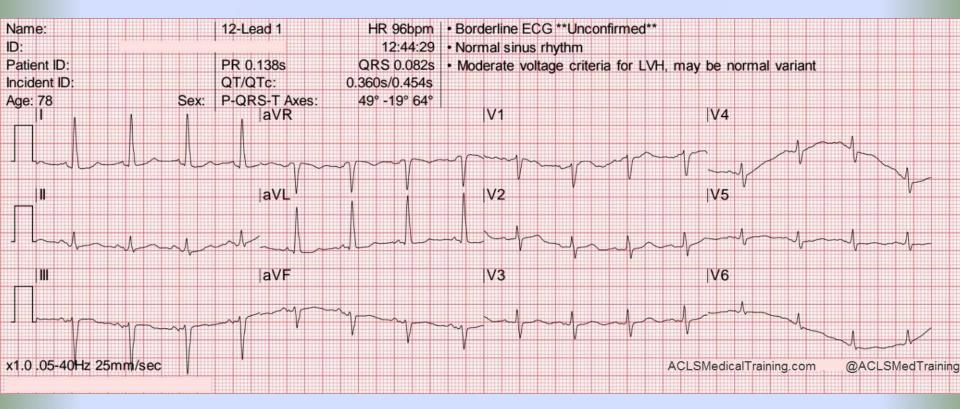


THE ISOELECTRIC LINE



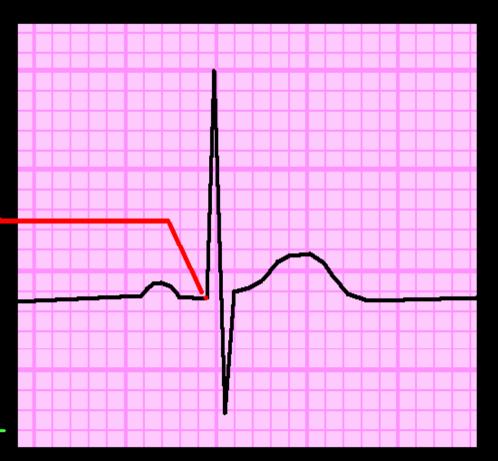
. . .the "flat line" between ECG complexes, when there is no detectable electrical activity . . .

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

THE P-Q JUNCTION

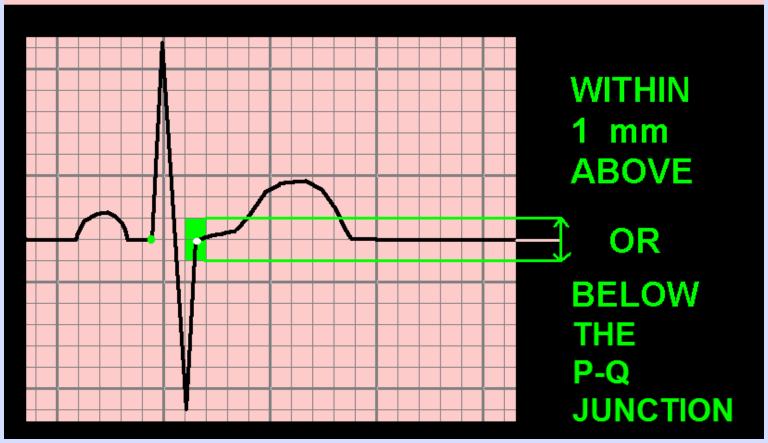
... is the POINT where the P-R SEGMENT ends and the QRS COMPLEX BEGINS. Used for POINT OF REFERENCE for measurement of the J-POINT and the S-T SEGMENT –

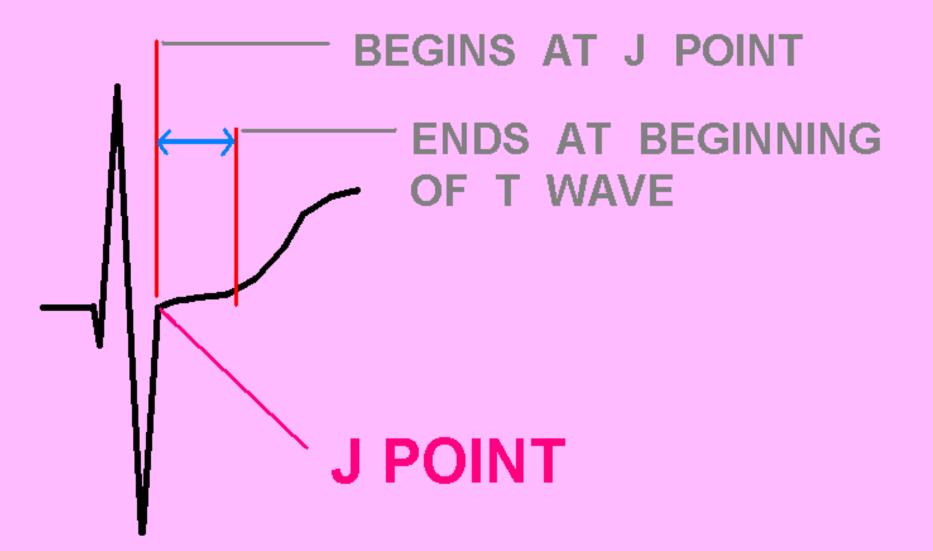


 as per the A.H.A., A.C.C., and WANG, ASINGER, and MARRIOTT, N.E.J.M. vol. 349:2128-2135 Nov. 27, 2003 not isoelectric !"

Defining NORMAL:

THE J POINT SHOULD BE..





SHOULD HAVE A "SLIGHT POSITIVE" INCLINATION

SHOULD BE "CONCAVE" IN SHAPE . . .

AS OPPOSED TO "CONVEX" IN SHAPE

SHOULD BE "CONCAVE" IN SHAPE . . .

THE T WAVE



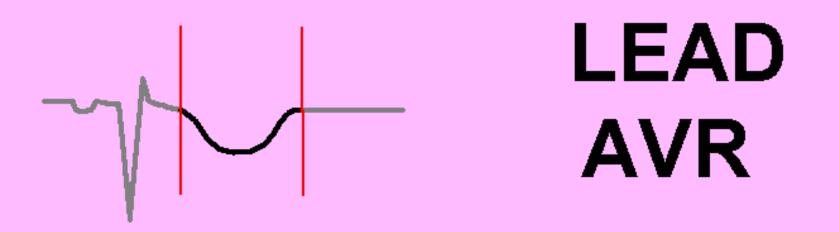
SHOULD BE SYMMETRICAL

THE T WAVE



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

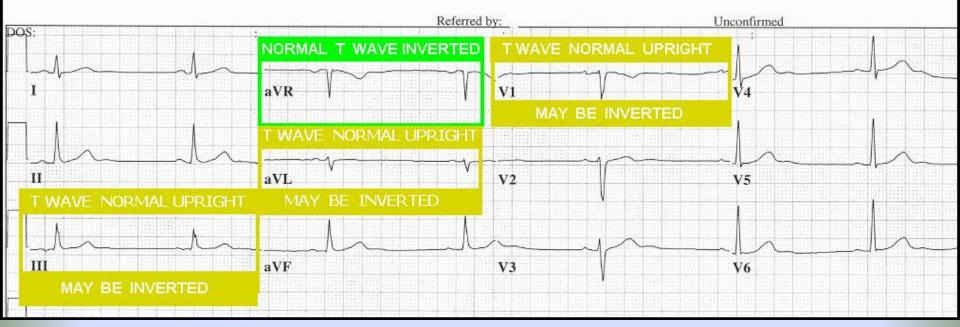
THE T WAVE



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

Normal Variants: *T Wave Inversion*

Leads where the T WAVE may be INVERTED:



THE T WAVE

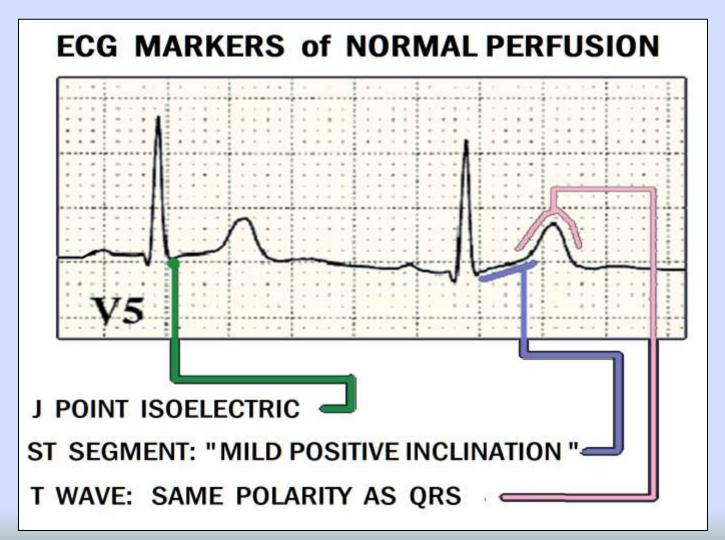


- IN THE LIMB LEADS, SHOULD BE LESS THAN 1.0 mv (10 mm)
- IN THE PRECORDIAL LEADS, SHOULD BE LESS THAN 0.5 mv (5 mm)
- SHOULD NOT BE TALLER THAN R WAVE IN 2 OR MORE LEADS.

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) :

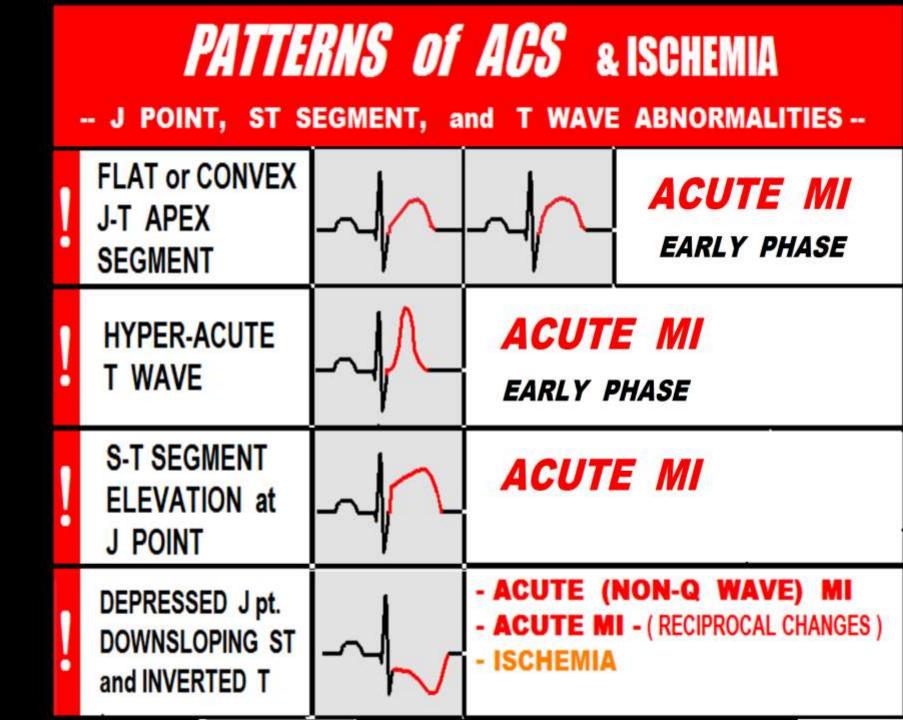


Q: If the previous slide showed what *normal* J Points, ST Segments and T waves look like, what is ABNORMAL ? Q: If the previous slide showed what *normal* J Points, ST Segments and T waves look like, what is ABNORMAL ?

A: EVERYTHING ELSE !!!



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

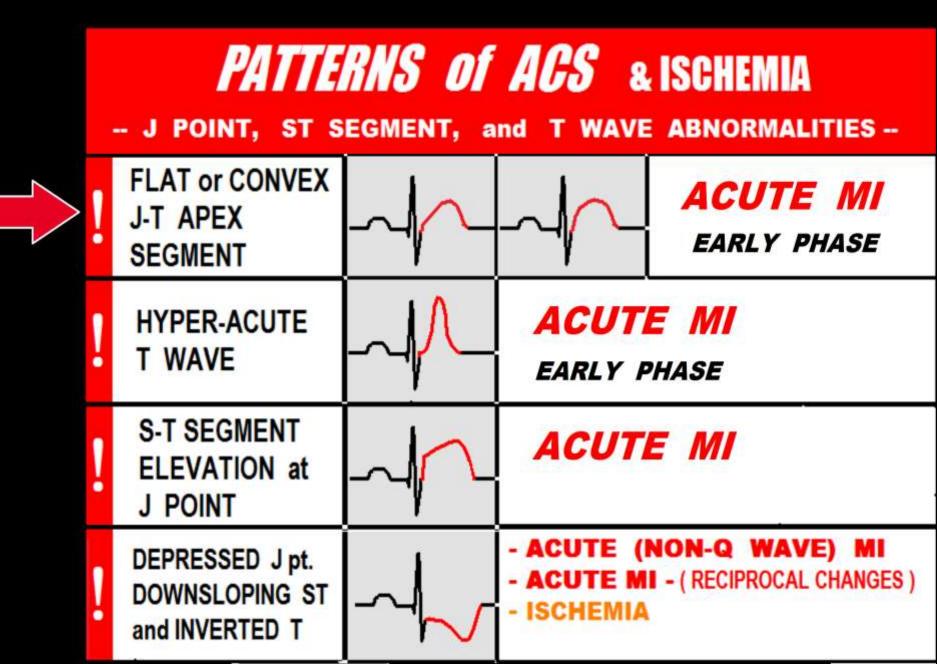


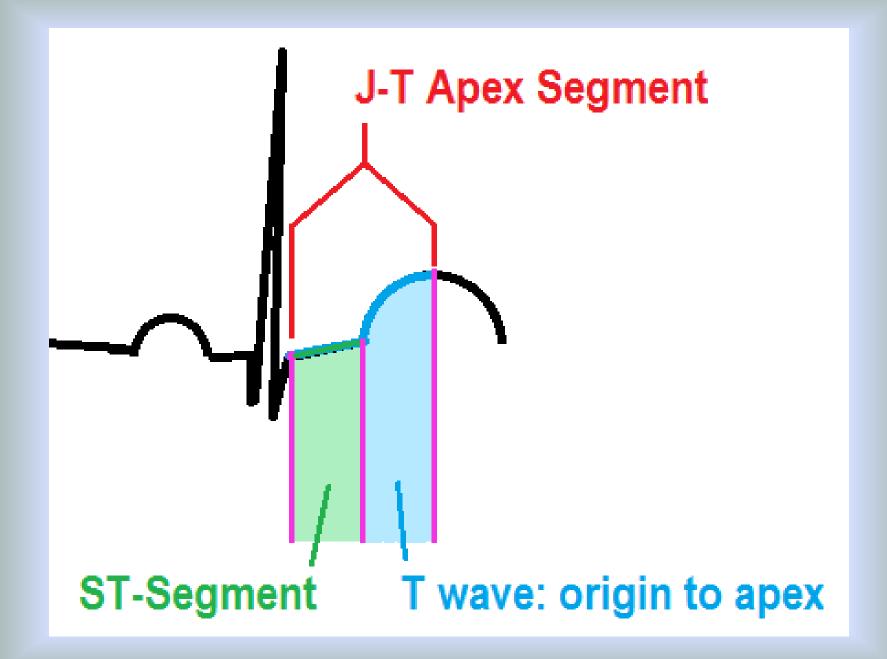
INVERTED T WAVE	$\sim \sim$	- MYOCARDITIS - ELECTROLYTE IMBAL. - ISCHEMIA
SHARP S-T T ANGLE	~ <u></u> /~	- ACUTE MI (NOT COMMON) - ISCHEMIA
BI-PHASIC T WAVE (WELLEN'S)	-~~	- SUB-TOTAL LAD LESION - VASOSPASM - HYPERTROPHY
DEPRESSED J POINT with UPSLOPING ST	~/~	- ISCHEMIA
DOWNSLOPING S-T SEGMENT	$\sim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

Some less common, less reliable possible indicators of ACS:

?	FLAT S-T SEGMENT > 120 ms	$\sim \downarrow \sim$	- ISCHEMIA
?	LOW VOLTAGE T WAVE WITH NORMAL QRS	~	- ISCHEMIA
?	U WAVE POLARITY OPPOSITE THAT OF T WAVE	$\downarrow \sim$	- ISCHEMIA

LET'S START HERE





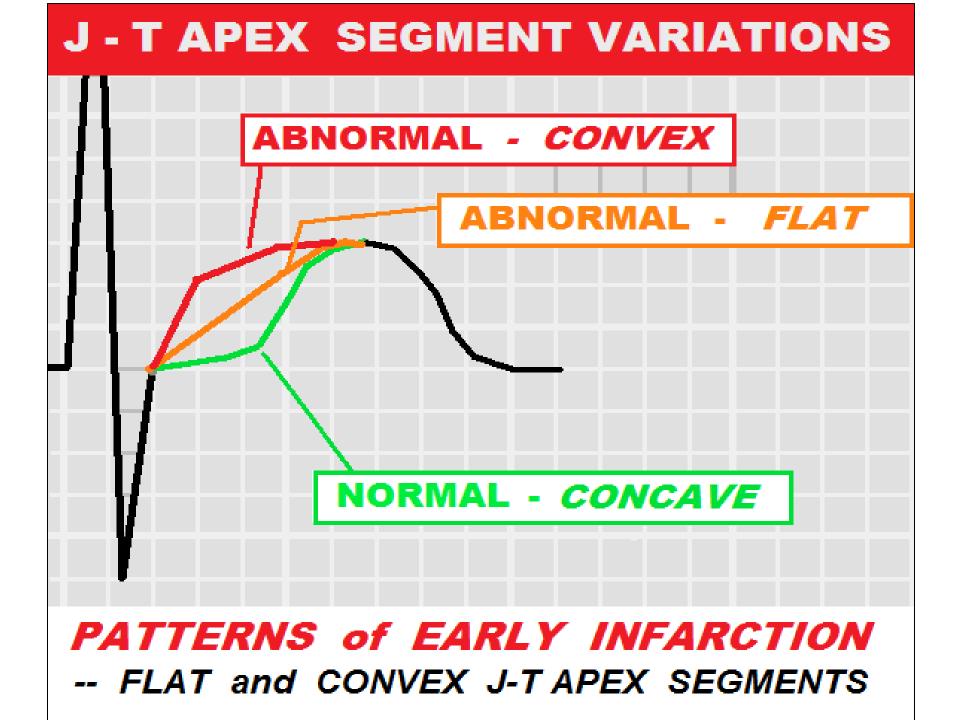
THE S-T SEGMENT

NORMAL

ABNORMAL

SHOULD BE "CONCAVE" IN SHAPE . . .

AS OPPOSED TO "CONVEX"



WHEN EVALUATING for ST SEGMENT ELEVATION

From: AMERICAN HEART ASSOCIATION ACLS 2005 REVISIONS

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

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

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

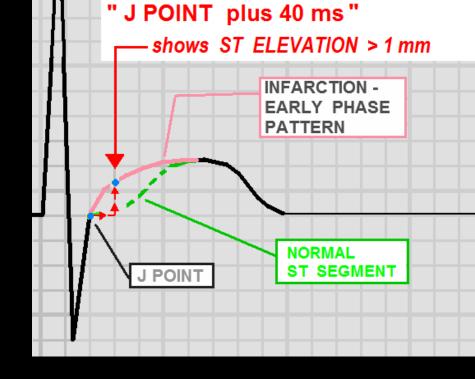
PTCA of the Left Anterior Descending artery (figure B).

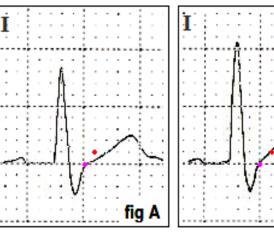


assumes a CONVEX shape. When measured 40 ms beyond the J POINT, the ST segment is elevated > 1 mm. This phenonemon is seen routinely in the cath lab prior to the occurance of ST ELEVATION at the J POINT during PTCA and STENTING.

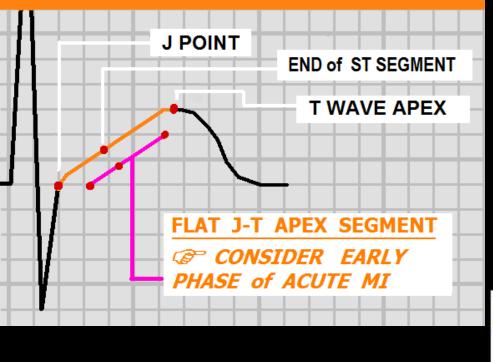


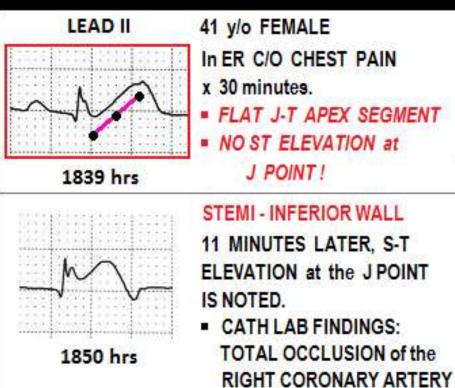
fig B



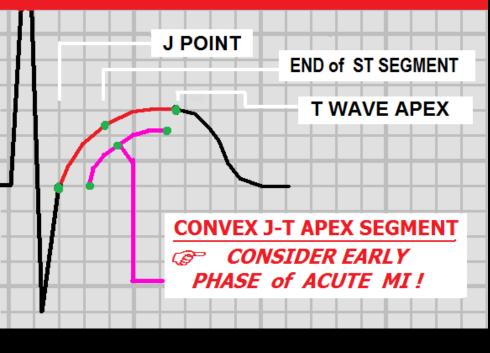


ABNORMAL J-T APEX SEGMENT



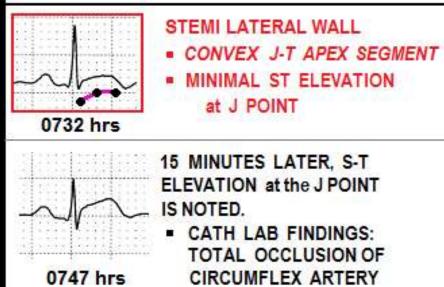


ABNORMAL J-T APEX SEGMENT





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



CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

RISK FACTOR PROFILE:

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

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

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

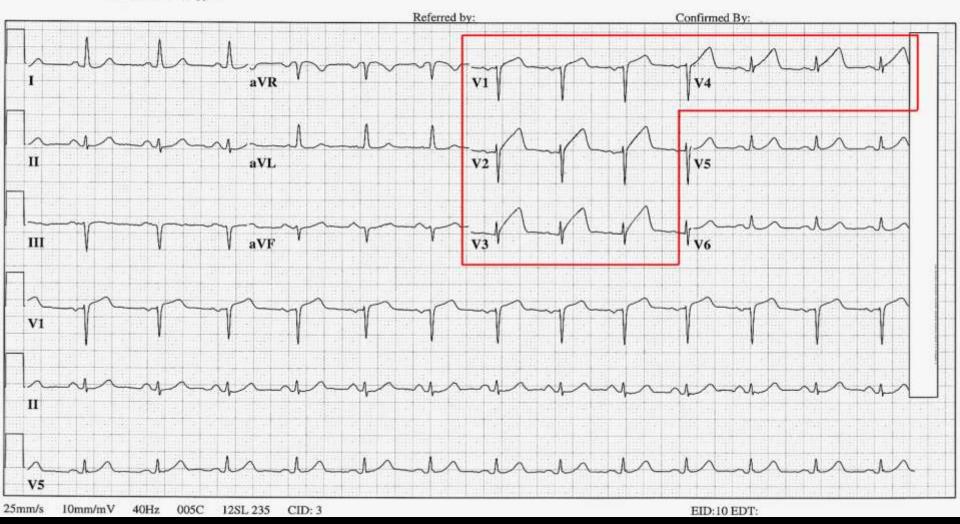
LABS: JUST OBTAINED, RESULTS NOT AVAILABLE YET.

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

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

Normal sinus rhythm Normal ECG No previous ECGs available

Technician: W Ruppert



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

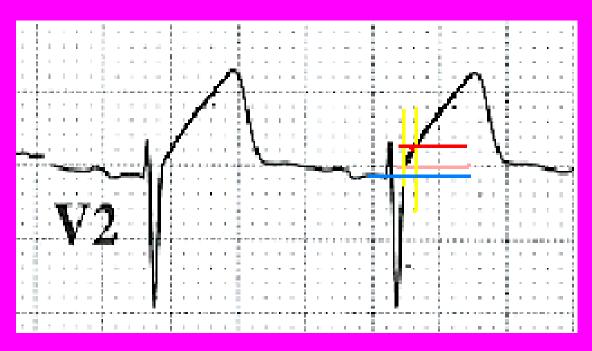
measurement of S-T elevation



S-T elevation at J point = 0.5 mm

ACUTE MI = S-T elev. > 1.0 mm

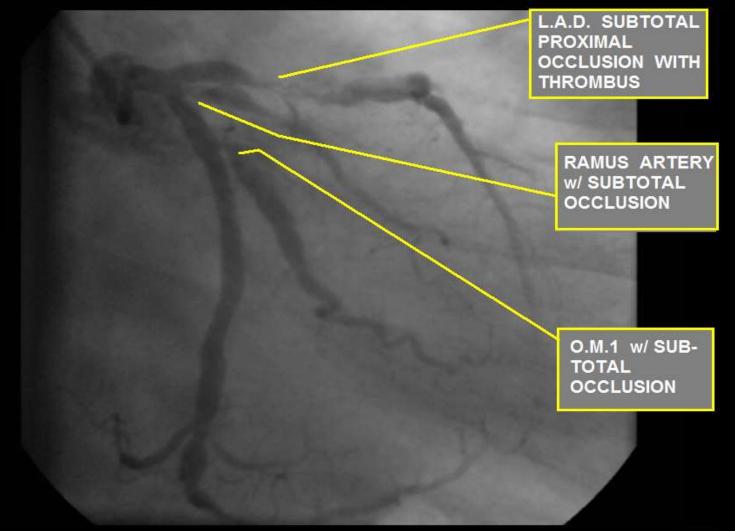
measurement of S-T elevation by "J point + .04" method



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

ACUTE MI = S-T elev. > 1.0 mm

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



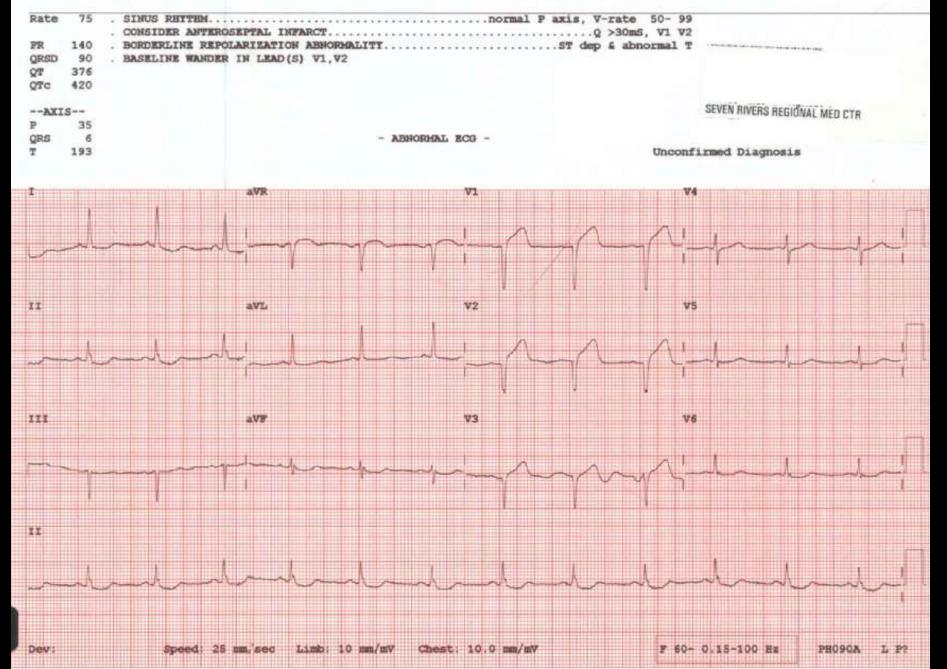
TREATMENT PLAN : EMERGENCY CORONARY ARTERY BYPASS SURGERY (4 VESSEL)

ECG Patterns associated with "EARLY PHASE MI:"

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



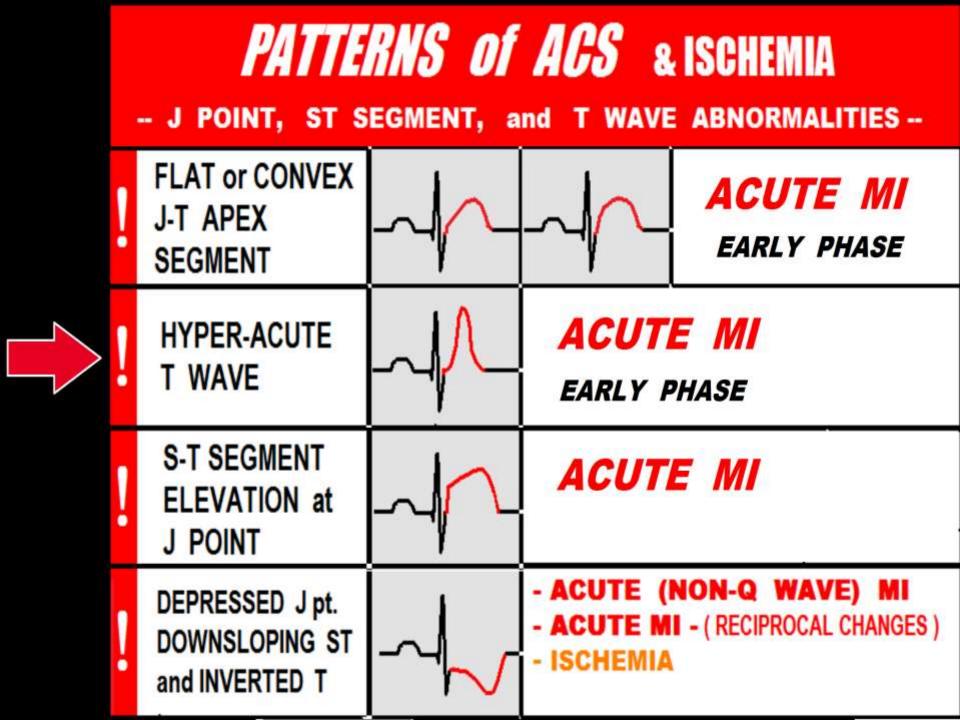




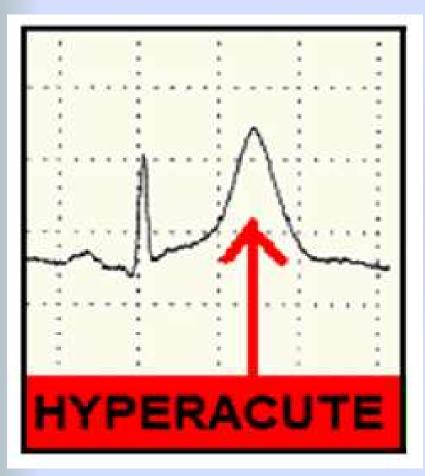
3. Dynmamic ST-T Wave Changes in Serial ECGs. Recorded at SRRMC

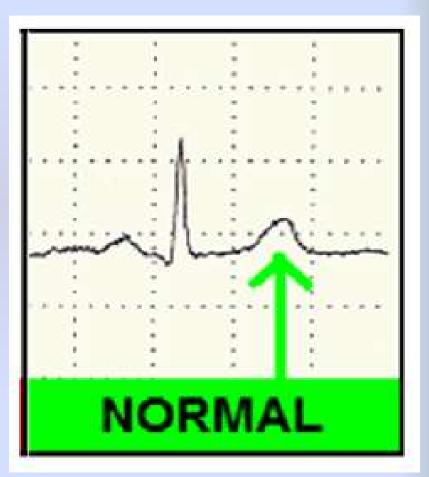


Acute In-Stent Thrombus Proximal LAD

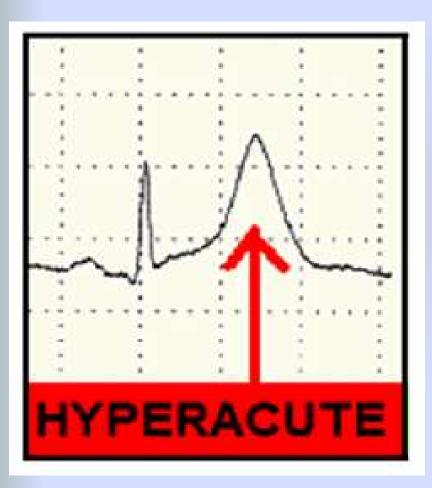


T waves should not be HYPERACUTE



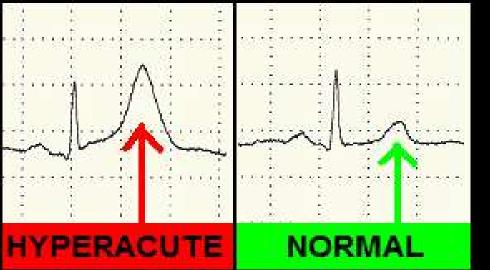


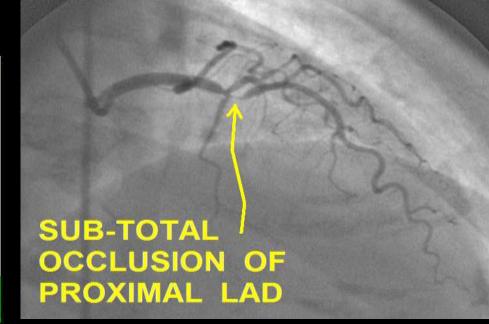
HYPERACUTE T Waves may indicate:



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

HYPERACUTE T WAVES

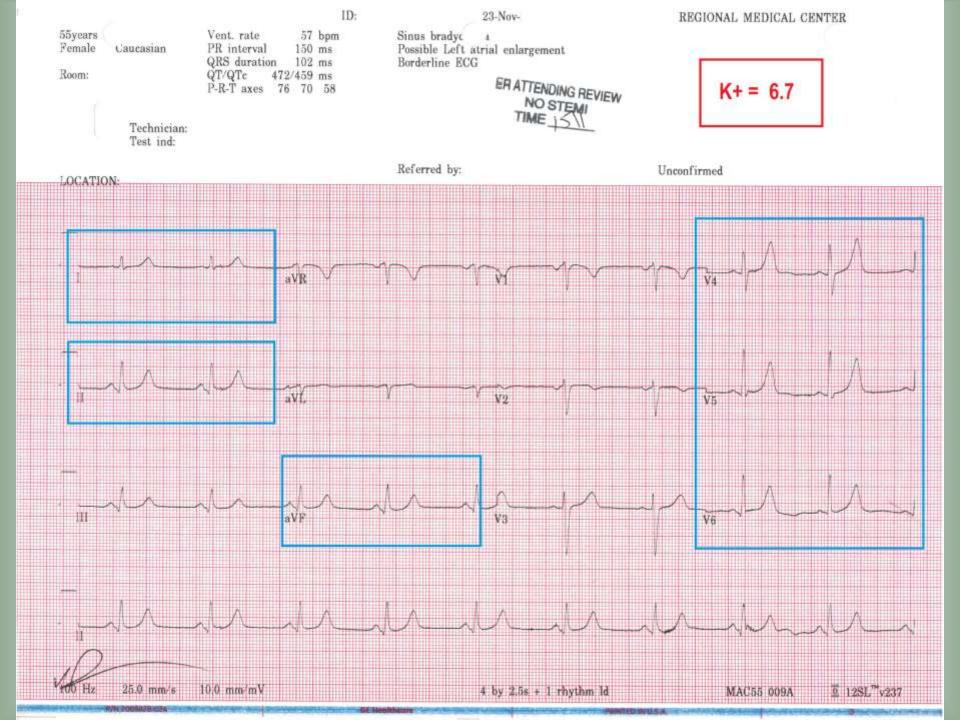




ECG waveforms obtained just before (hyperacute) and just after (normal) the critical blockage was stented in this patient's Proximal Left Anterior Descending (LAD) artery.

Helpful Clue: Hyper-Acute T Waves

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



Helpful Clue: Hyper-Acute T Waves

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

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

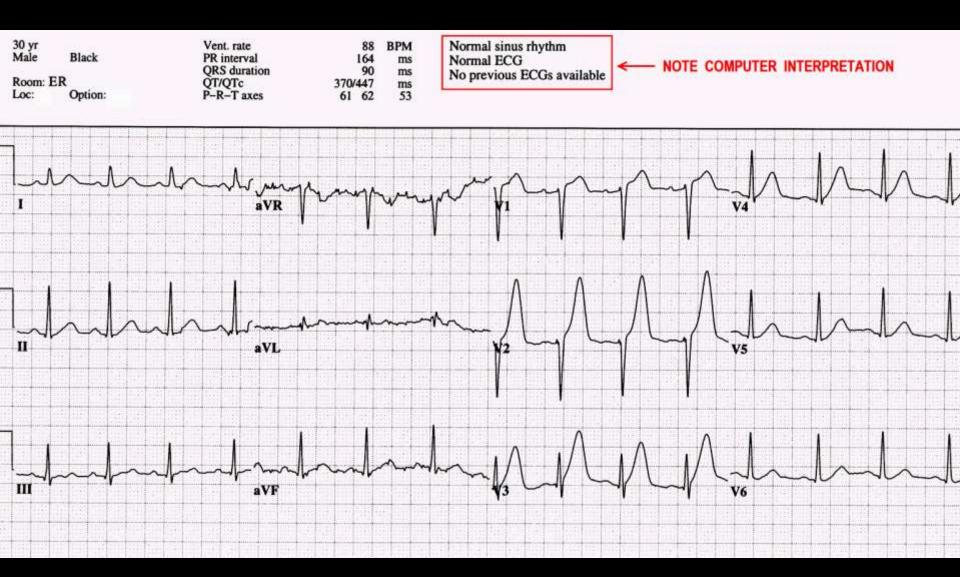
30 y/o male presents to ER via EMS, c/o sudden onset of dull chest pain x 40 min. Pain level varies, not effected by position, movement or deep inspiration. No associated symptoms.

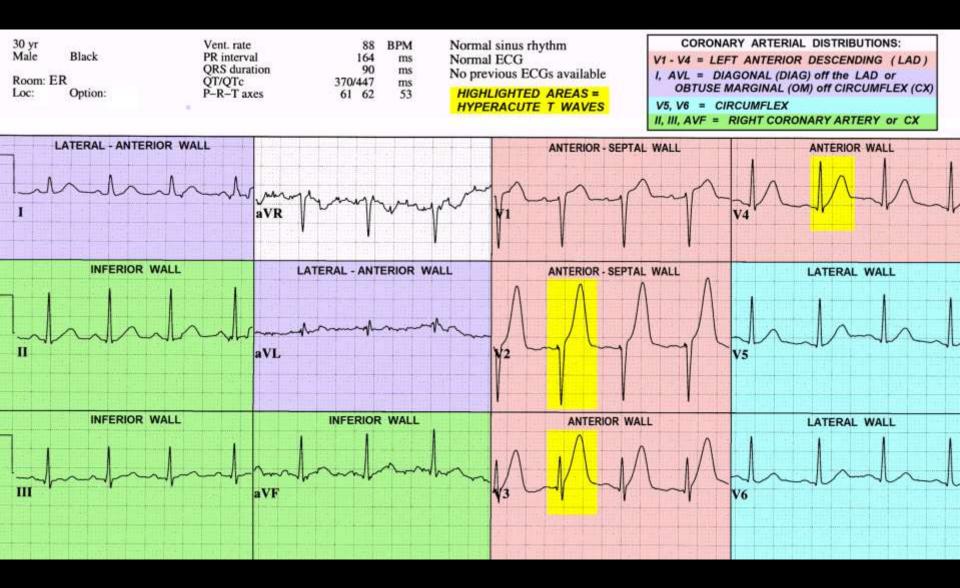
RISK FACTOR PROFILE: NONE. CHOLESTEROL UNKNOWN.

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

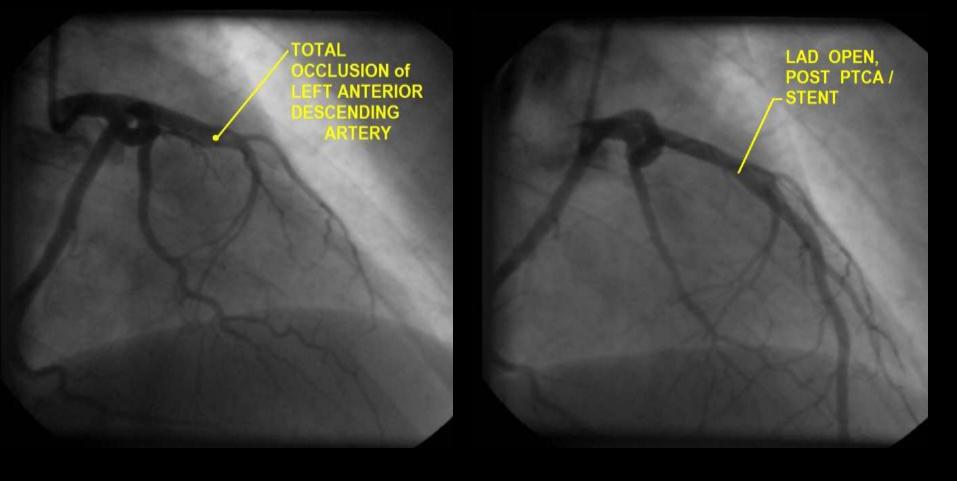
VITAL SIGNS: BP 136/88 P 90 R 20 SA02 98%

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





Cath Lab findings:



Dynamic ST-T Wave Changes:

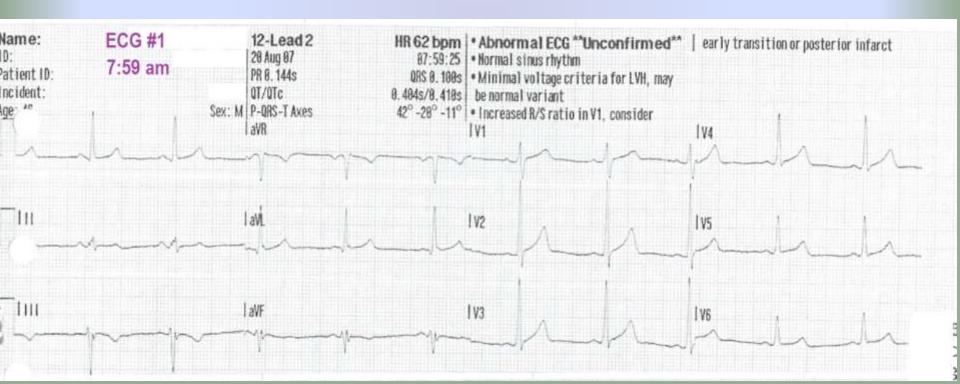
 Other than HEART RATE related variations (which affect intervals), *J Points, ST-Segments and T Waves SHOULD NOT CHANGE.*

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.

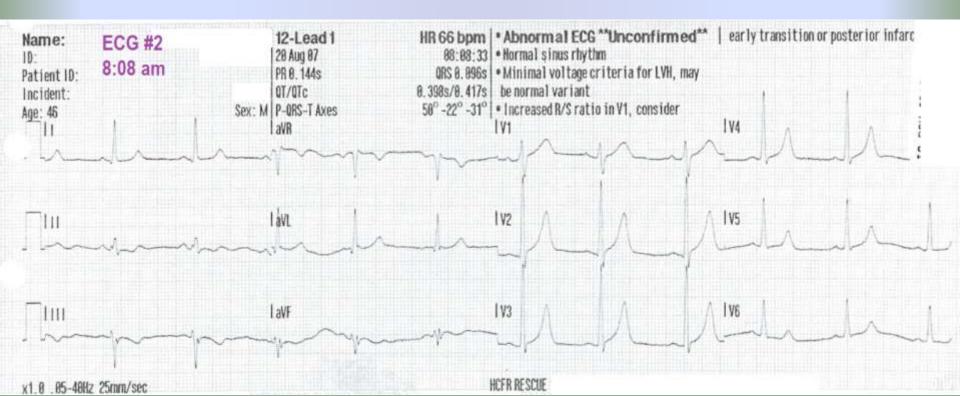
46 year old male

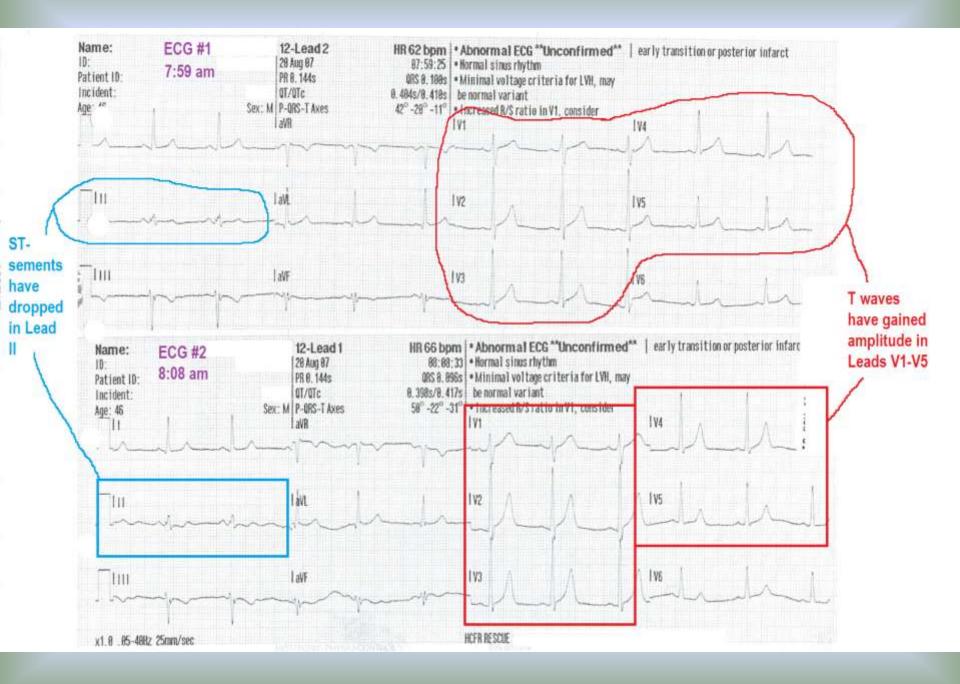
- Exertional dyspnea X "several weeks"
- Intermittent chest pressure X last 3 hours. Currently pain free.

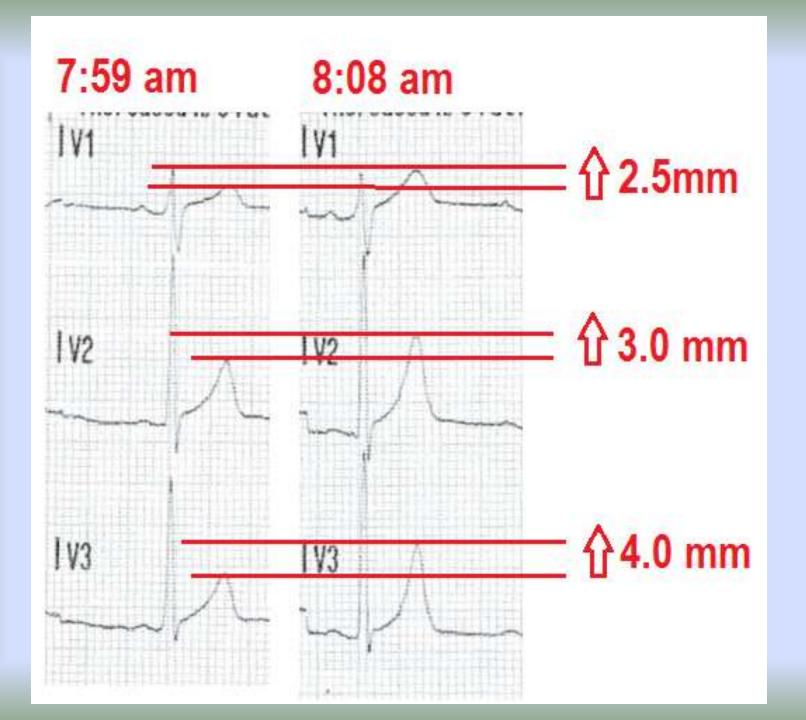


46 year old male: ECG 1

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





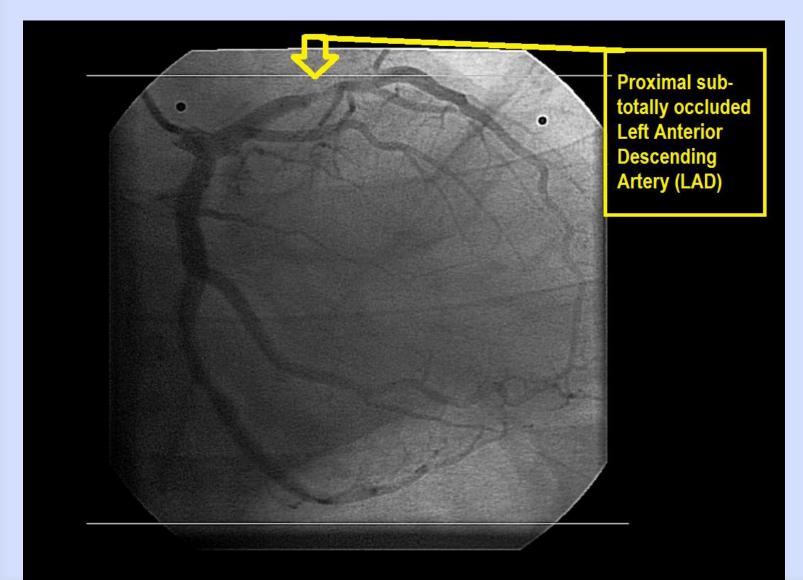


ST-Segment Depression

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

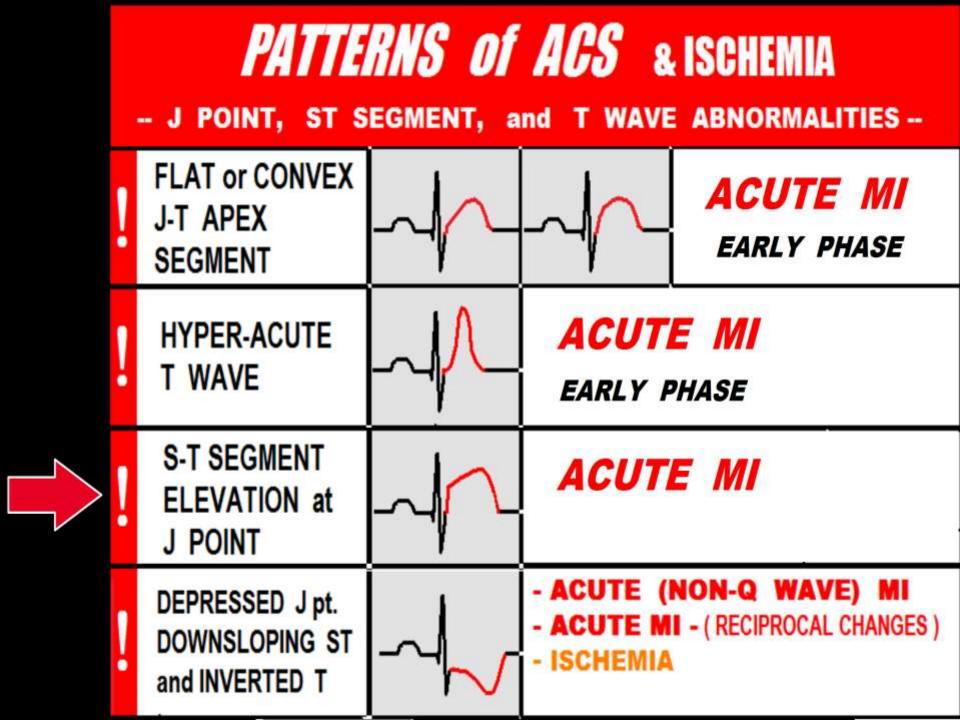


Cath Lab Angiography:





"NOWHERE", NEW MEXICO, 1994



ECG CRITERIA for DIAGNOSIS of STEMI:

(ST ELEVATION @ J POINT)

*LEADS V2 and V3:

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

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

FEMALES ----- 1.5 mm

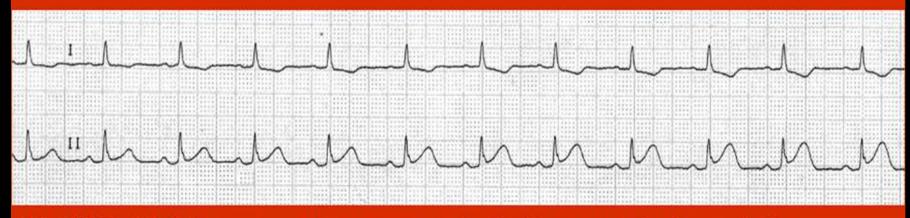
ALL OTHER LEADS: 1.0 mm or more,

1.0 mm or more, in TWO or more CONTIGUOUS LEADS

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

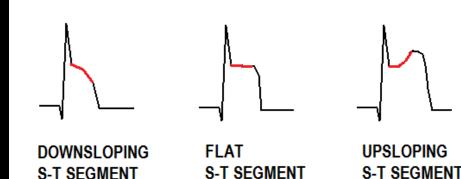
ST SEGMENT ELEVATION:

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



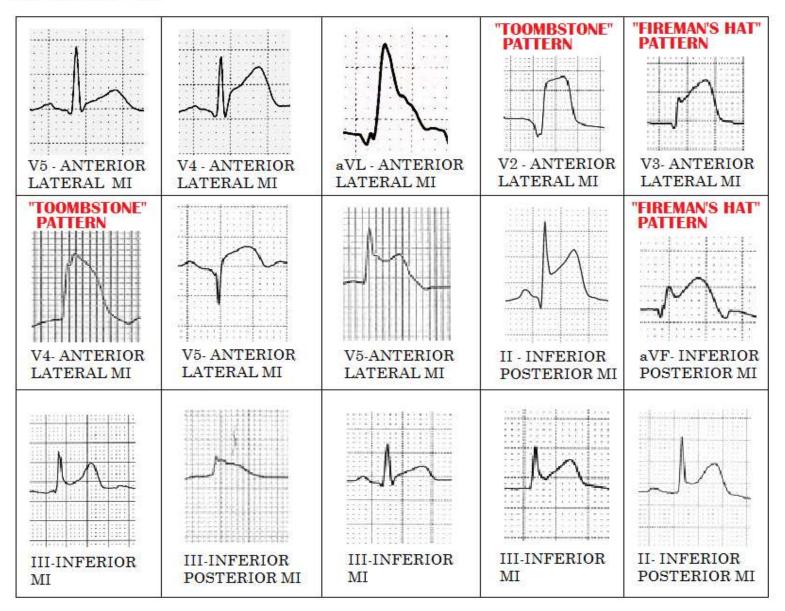
IN THIS CASE, a normal response to balloon occlusion of the RIGHT CORONARY ARTERY during PTCA in the CARDIAC CATH LAB

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:



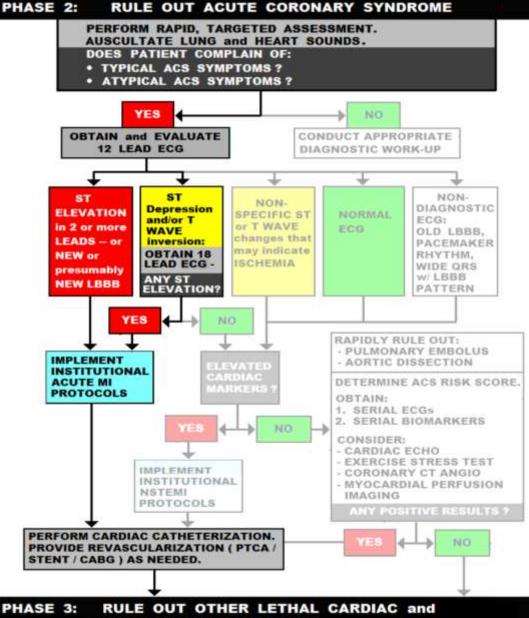
Reciprocal S-T Segment Depression *may* or *may not* be present during AMI.

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.

PHASE 1: RULE OUT LIFE-THREATENING CONDITIONS





NON-CARDIAC CONDITIONS.

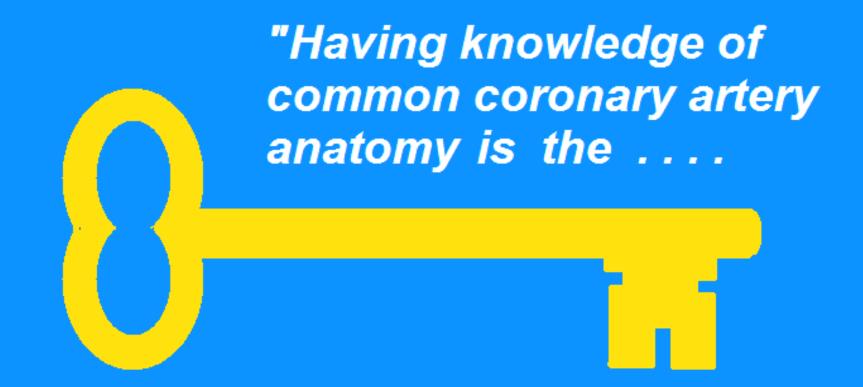
ACUTE MI

COMPLICATIONS TO ANTICIPATE FOR ALL MI PATIENTS :





FAILURE OF STRUCTURE(S) SERVED BY THE BLOCKED ARTERY



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 ... ANTICIPATE FAILURE OF THOSE STRUCTURES . . . • INTERVENE APPROPRIATELY!



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

CASE STUDY 1 - STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

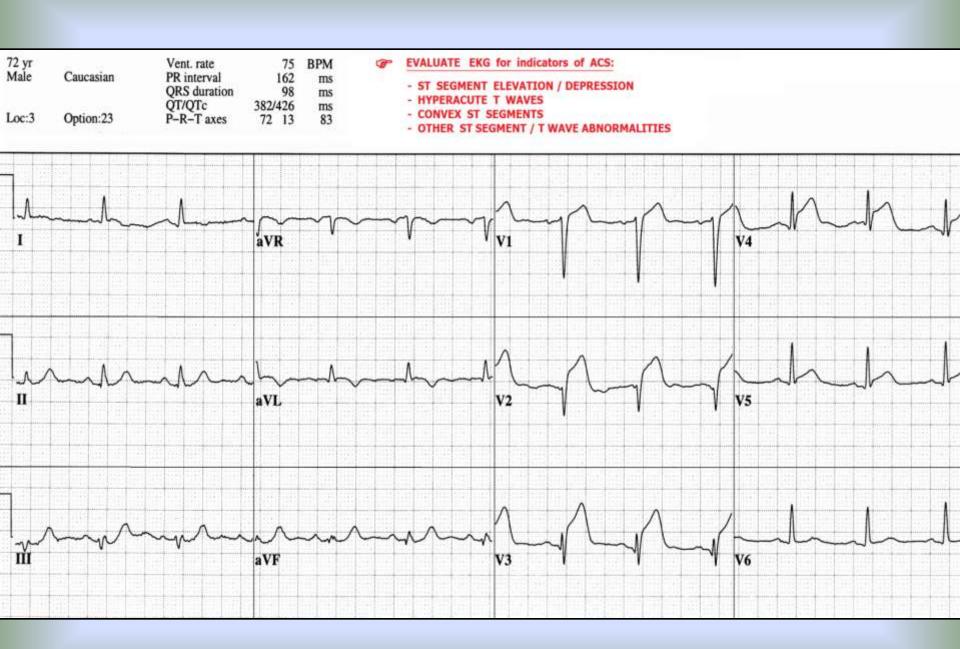
RISK FACTOR PROFILE:

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

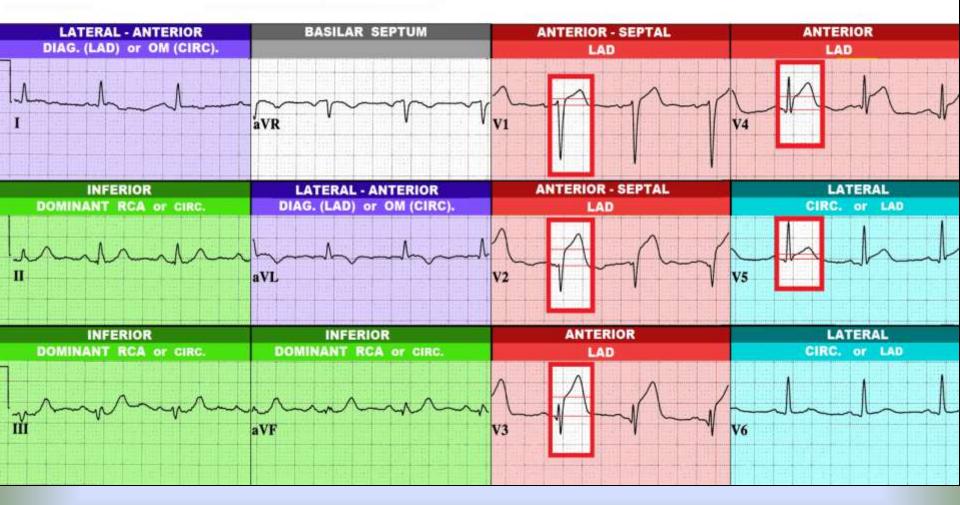
PHYSICAL EXAM: Patient calm, alert, oriented X 4, skin cool, dry, pale. No JVD, Lungs clear bilaterally. Heart sounds normal S1, S2. No peripheral edema.

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

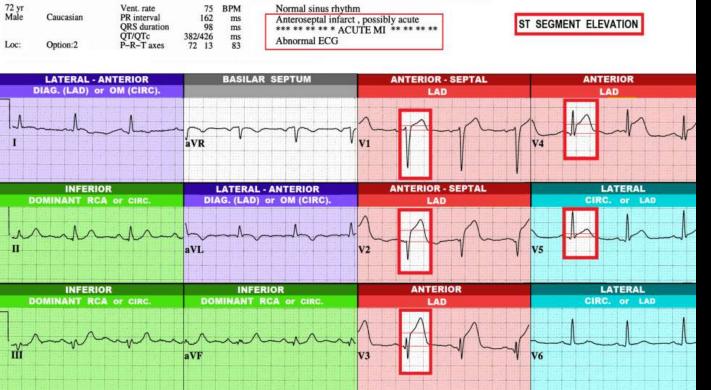
LABS: FIRST TROPONIN: 6.4



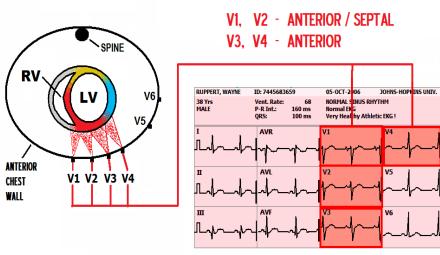
72 yr Male	Caucasian	Vent. rate PR interval ORS duration	75 162 98	BPM ms ms	Normal sinus rhythm Anteroseptal infarct, possibly acute *** ** ** ** ACUTE MI ** ** **	ST SEGMENT ELEVATION
Loc:	Option:2	QT/QTc P-R-T axes	382/426 72 13	ms 83	Abnormal ECG	

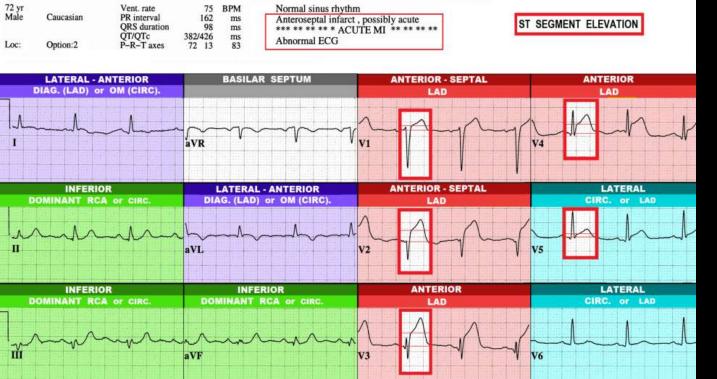


Note: There is NO Reciprocal ST Depression on this STEMI ECG !

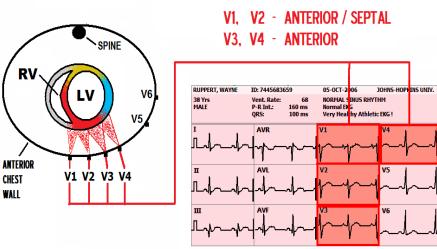


V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL of the LEFT VENTRICLE

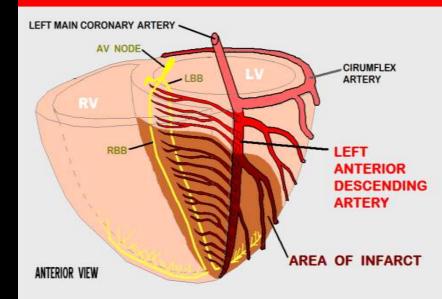




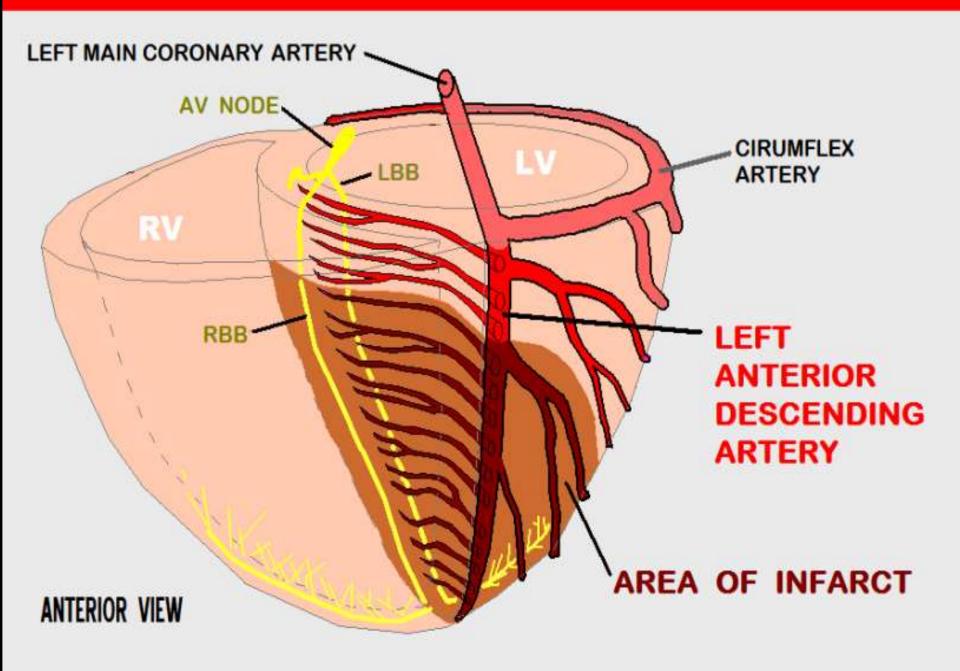
V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL of the LEFT VENTRICLE



OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY



OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY



LAD DISTRIBUTION

35-45% of LV MUSCLE MASS

9

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

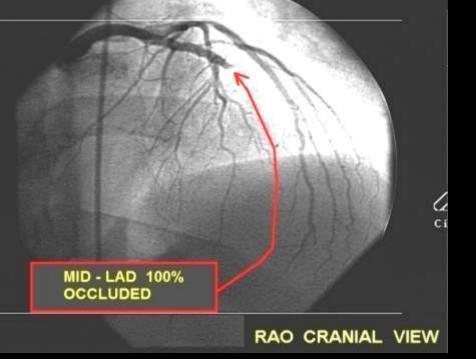


LEFT ANTERIOR DESCENDING ARTERY (LAD)

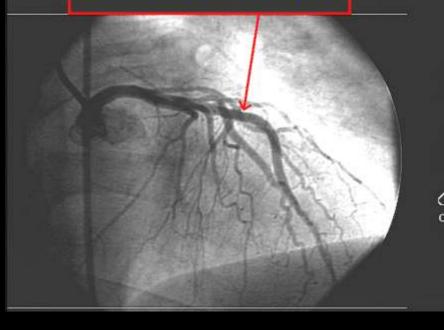
- ANTERIOR WALL OF LEFT VENTRICLE
- 35-45% OF LEFT VENTRICLE MUSCLE MASS
 - SEPTUM, ANTERIOR 2/3
 - **BUNDLE BRANCHES**
 - ANTERIOR-MEDIAL PAPILLARY MUSCLE

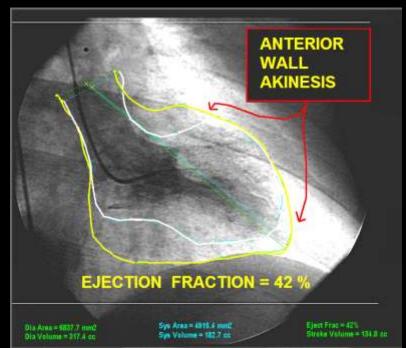
ANTICIPATED COMPLICATIONS of ANTERIOR-SEPTAL WALL STEMI & POSSIBLE INDICATED INTERVENTIONS:

- CARDIAC ARREST	BCLS / ACLS
- CARDIAC DYSRHYTHMIAS (VT / VF)	ACLS (antiarrhythmics)
- PUMP FAILURE with	INOTROPE THERAPY:
CARDIOGENIC SHOCK	-DOPAMINE / DOBUTAMINE /
	LEVOPHED
	- INTRA-AORTIC BALLOON PUMP
	(use caution with fluid challenges
	due to PULMONARY EDEMA)
- PULMONARY EDEMA	- CPAP
	- CPAP - ET INTUBATION
	(use caution with dieuretics due to
	pump failure and hypotension)
- 3rd DEGREE HEART BLOCK - NOT	TRANSCUTANEOUS or
RESPONSIVE TO ATROPINE	TRANSVENOUS PACING



POST PTCA / STENT TO MID LAD





CASE STUDY 2: STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

RISK FACTOR PROFILE:

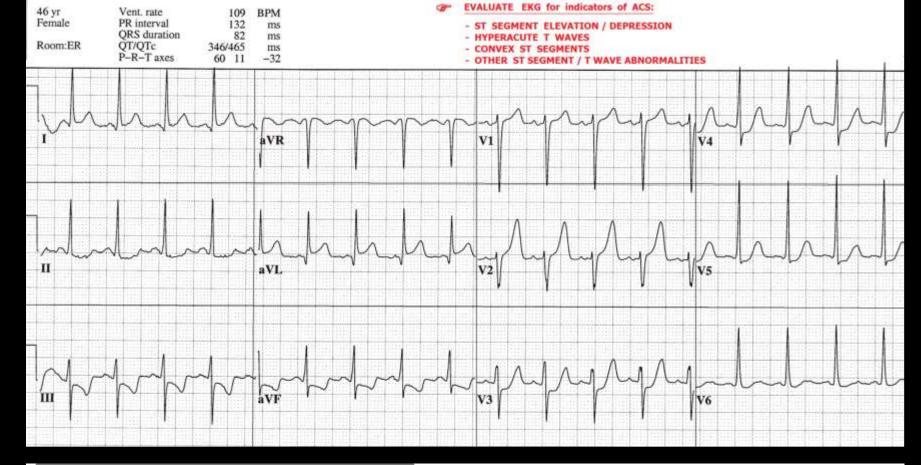


FAMILY HISTORY - father died of CAD, older brother had CABG, age 39 DIABETES - diet controlled HYPERTENSION

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

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

LABS: TROPONIN ultra = 2.8



CASE STUDY QUESTIONS:

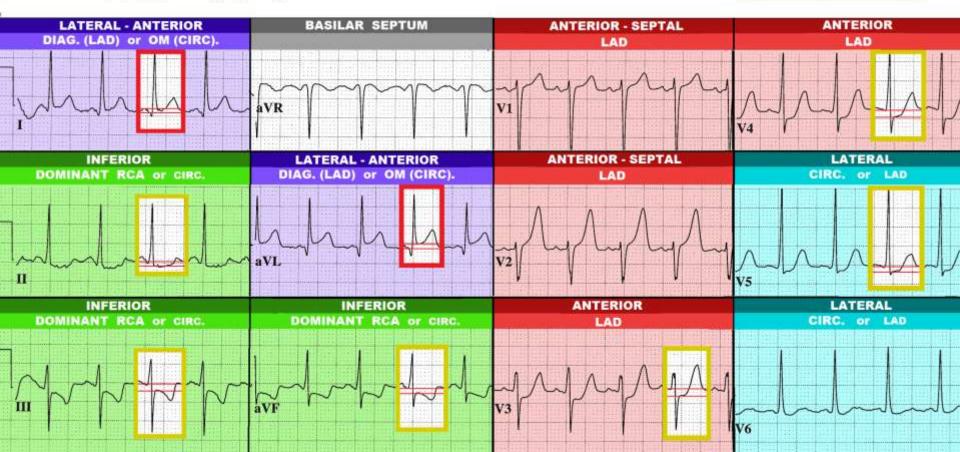
NOTE LEADS WITH ST ELEVATION:	NOTE LEADS WITH ST DEPRESSION:
WHAT IS THE SUSPECTED DIAGNOSIS ?	
WHAT IS THE "CULPRIT ARTERY" if applicable?	
LIST ANY CRITICAL STRUCTURES COMPROMISED:	LIST ANY POTENTIAL COMPLICATIONS:

46 yr	Vent. rate	109	BPM
Female	PR interval	132	ms
250-270-02277	QRS duration	82	ms
Room:ER	QT/QTc	346/465	ms
	P-R-T axes	60 11	-32

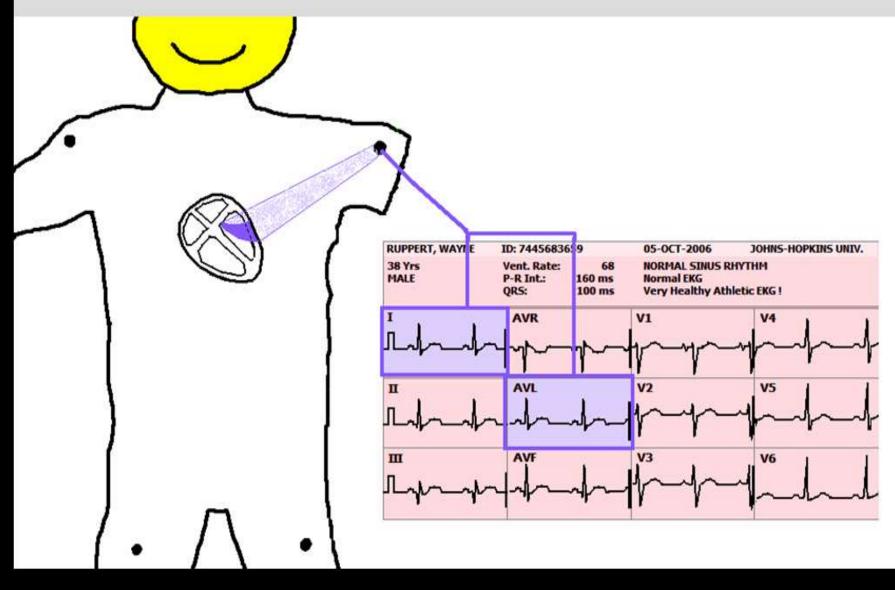
Sinus tachycardia Left ventricular hypertrophy with repolarization abnormality ST elevation consider lateral injury or acute infarct

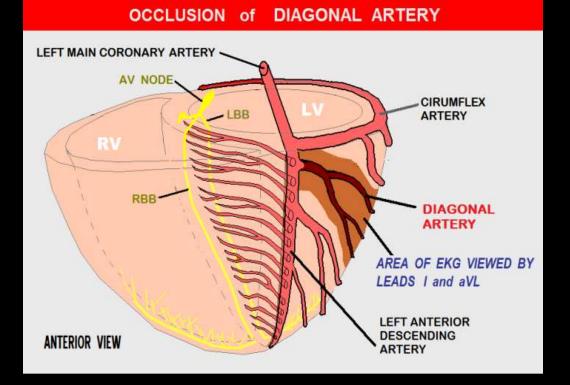
*** ** ** ** * ACUTE MI ** ** ** **



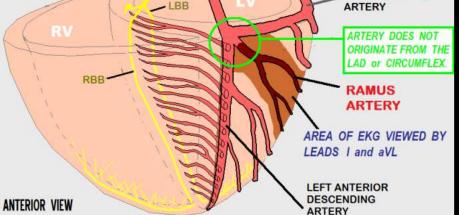


LEADS I and aVL view the ANTERIOR-LATERAL JUNCTION

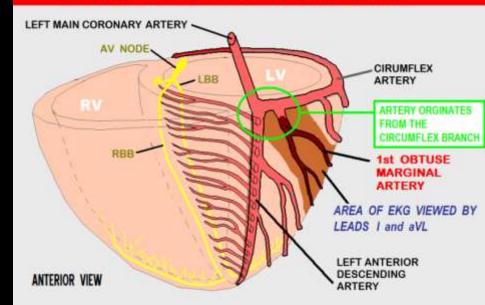




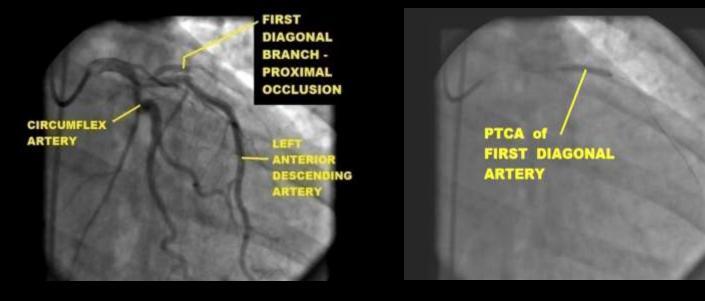
OCCLUSION of RAMUS ARTERY

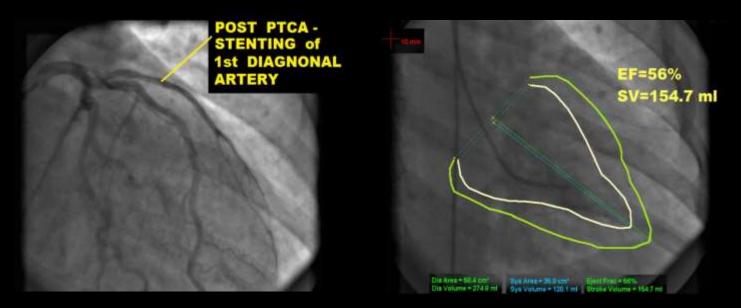


OCCLUSION of OBTUSE MARGINAL ARTERY



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





CASE STUDY SUMMARY			
ST ELEVATION: I, aVL	ST DEPRESSION:	II, III, aVF, V3 - V5	
SUSPECTED DIAGNOSIS: ACUTE LATERAL WALL M.I.			
SUSPECTED "CULPRIT ARTERY" (if applicable):			
USUALLY ONE OF THE SMALLER SIDE-BRAM	NCH ARTERIES:		
1. DIAGONAL ARTERY. (This is a side-branch artery off of the LEFT ANTERIOR DESCENDING (LAD) artery.			
2. OBTUSE MARGINAL ARTERY. (This is a side-branch artery off of the CIRCUMFLEX artery)			
3. RAMUS 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: POTE	NTIAL COMPLICATIONS:	POSSIBLE CRITICAL INTERVENTIONS:	
	POSSIBLE MODERATE	INOTROPIC AGENTS	
MUSCLE MASS	LV PUMP FAILURE	ET INTUBATION I.A.B.P. INSERTION	

CASE STUDY 3: STEM

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

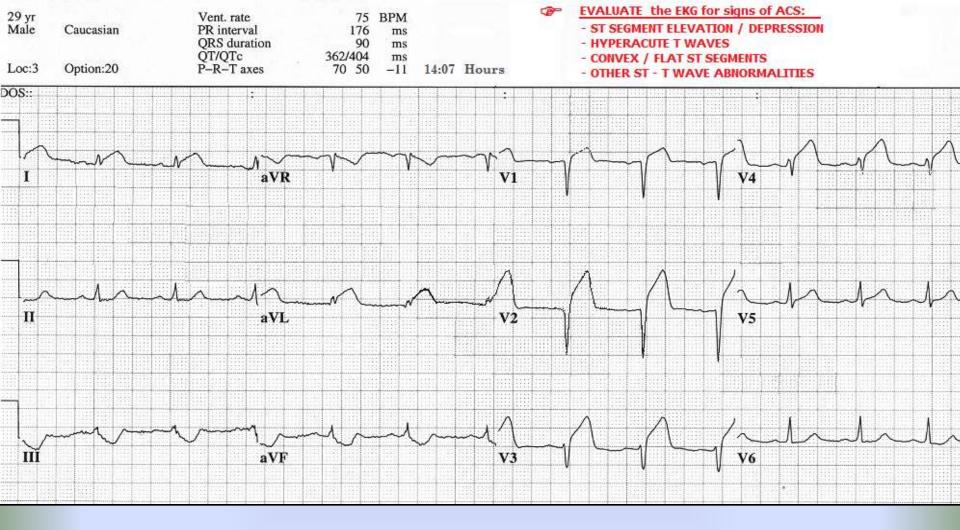
RISK FACTOR PROFILE:

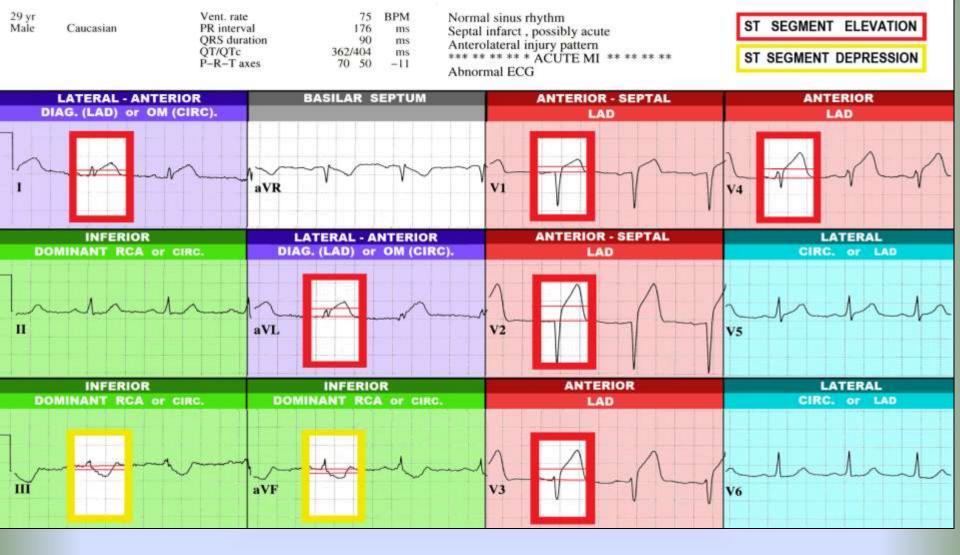
- FAMILY HISTORY father died of MI age 46
- CURRENT CIGARETTE SMOKER
- "MILD" HYPERTENSION untreated
- CHOLESTEROL unknown "never had it checked."

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

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

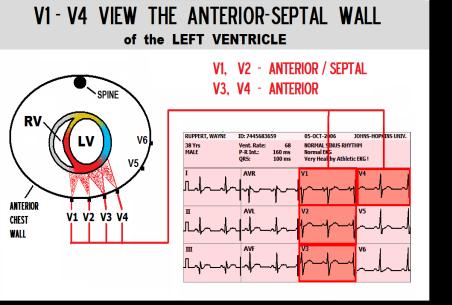
LABS: INITIAL CARDIAC MARKERS - NEGATIVE

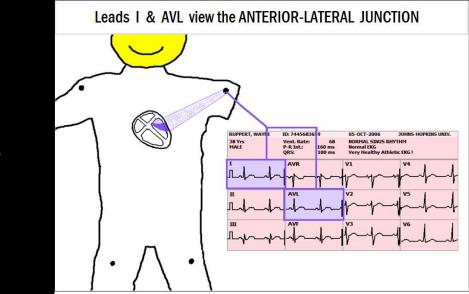




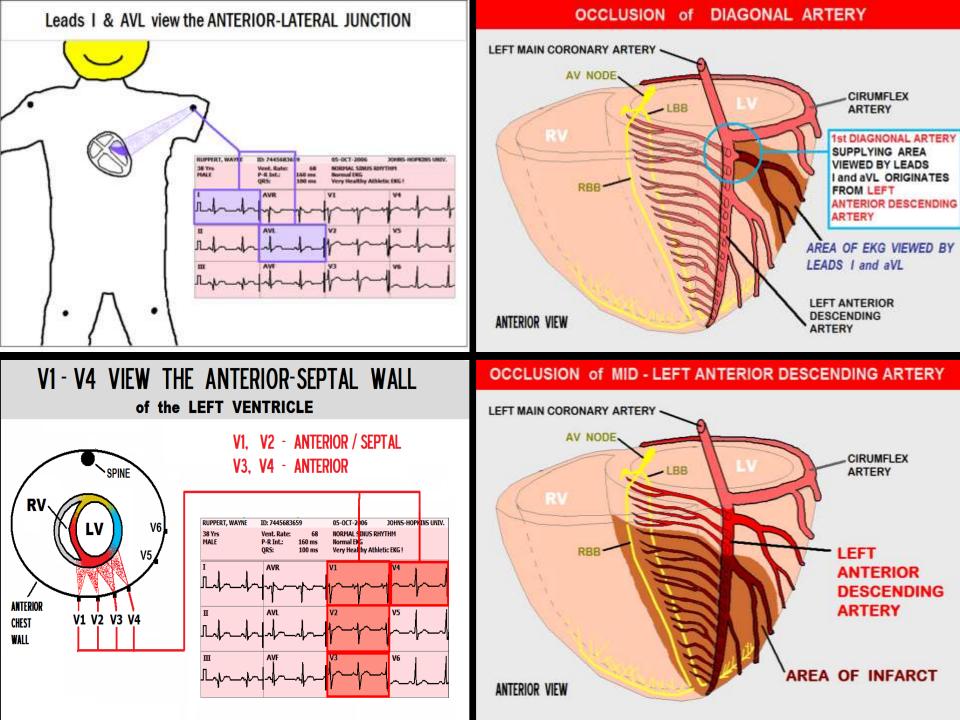
Reciprocal ST Depression is NOW PRESENT Additional ST Elevation is

present in Leads I, AVL

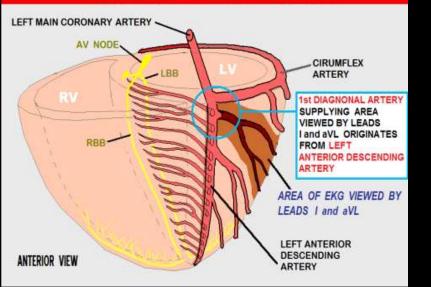




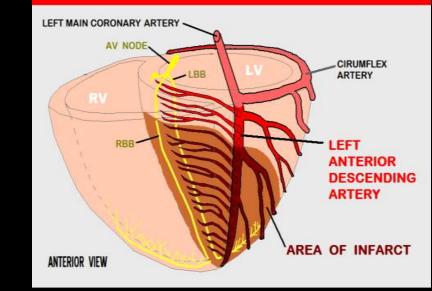




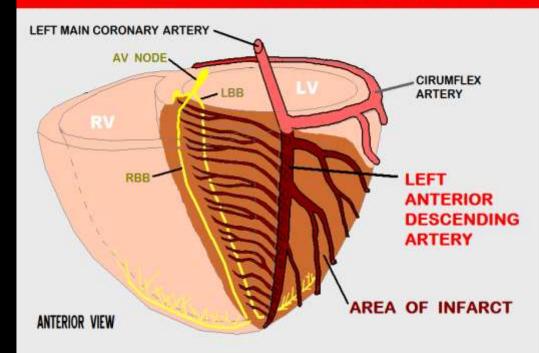
OCCLUSION of DIAGONAL ARTERY

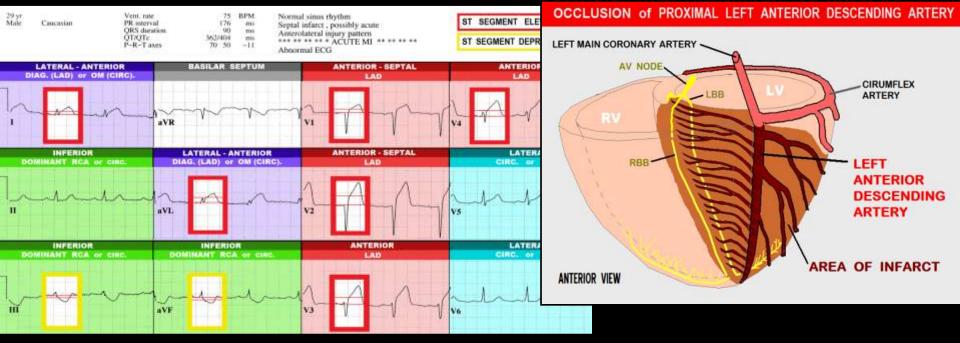


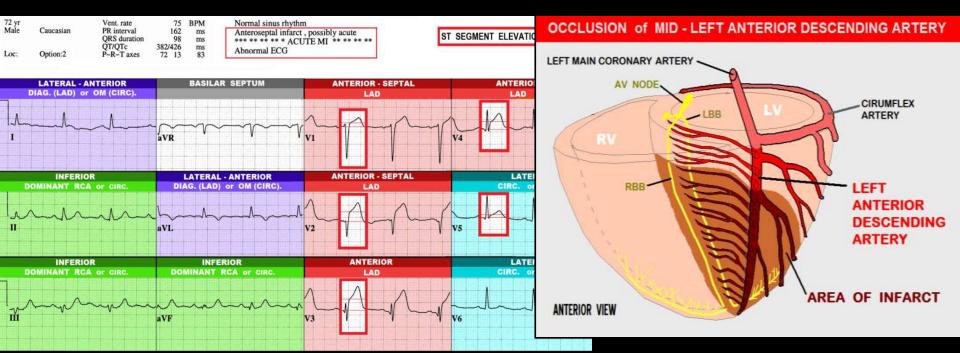
OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY



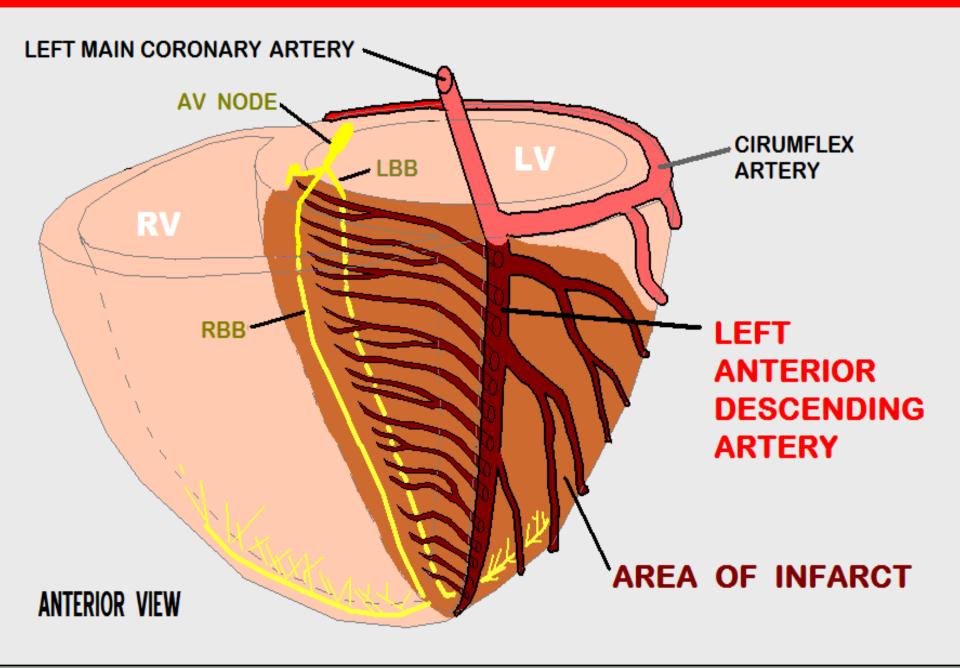
OCCLUSION of PROXIMAL LEFT ANTERIOR DESCENDING ARTERY







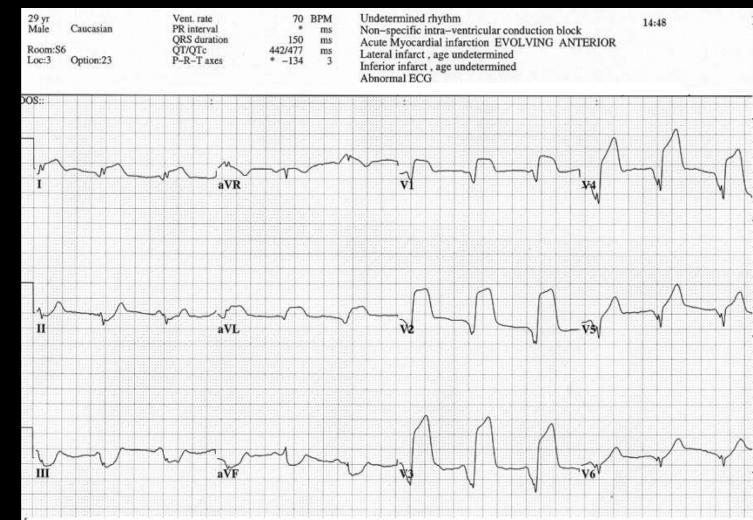
OCCLUSION of PROXIMAL LEFT ANTERIOR DESCENDING ARTERY



ANTICIPATED COMPLICATIONS of ANTERIOR-SEPTAL WALL STEMI & POSSIBLE INDICATED INTERVENTIONS:

- CARDIAC ARREST	BCLS / ACLS
- CARDIAC DYSRHYTHMIAS (VT / VF)	ACLS (antiarrhythmics)
- PUMP FAILURE with	INOTROPE THERAPY:
CARDIOGENIC SHOCK	-DOPAMINE / DOBUTAMINE /
	LEVOPHED
	- INTRA-AORTIC BALLOON PUMP
	(use caution with fluid challenges
	due to PULMONARY EDEMA)
- PULMONARY EDEMA	- CPAP
	- ET INTUBATION
	(use caution with dieuretics due to
	pump failure and hypotension)
- 3rd DEGREE HEART BLOCK - NOT	TRANSCUTANEOUS or
RESPONSIVE TO ATROPINE	TRANSVENOUS PACING

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



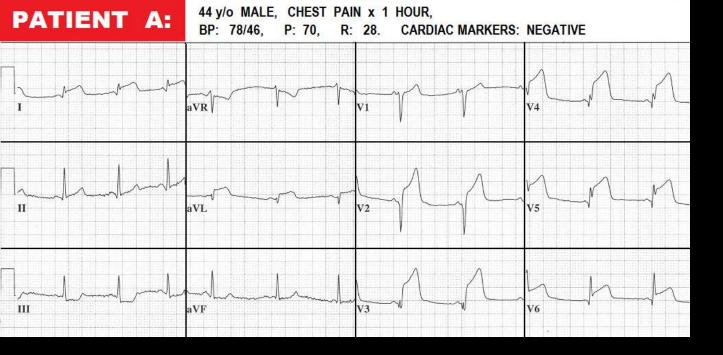
PROXIMAL OCCLUSION of the LEFT ANTERIOR DESCENDING Artery

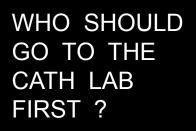
> POST PTCA _____ and STENT to the PROXIMAL LAD

CASE STUDY 4: CRITICAL DECISIONS SCENARIO

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

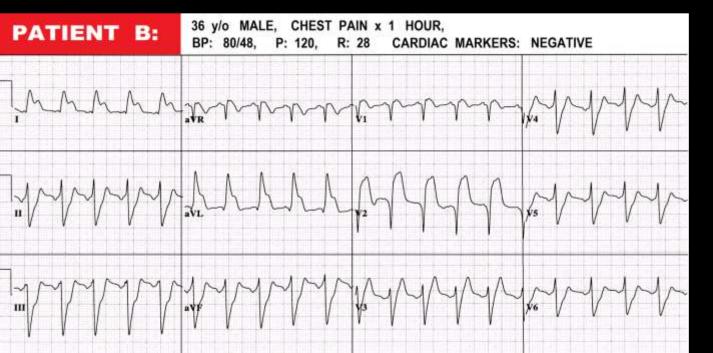
You are the ranking medical officer on duty in the ED when two acute STEMI patients arrive, ten minutes apart. The Cath Lab has one lab open, and can take ONE patient immediately. Both patients duration of symptoms and state of hemodynamic stability are similar.

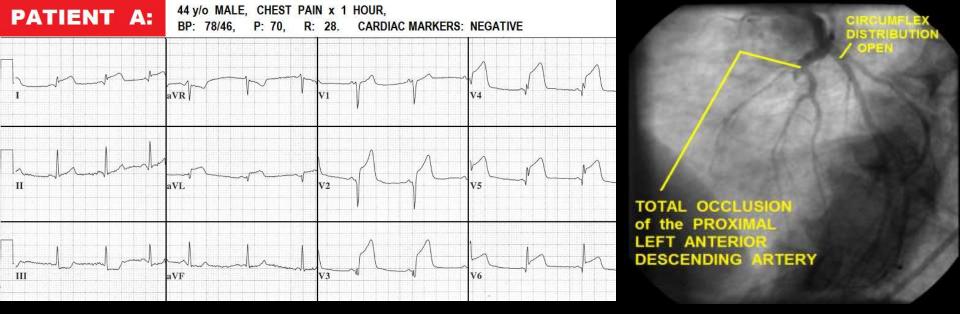


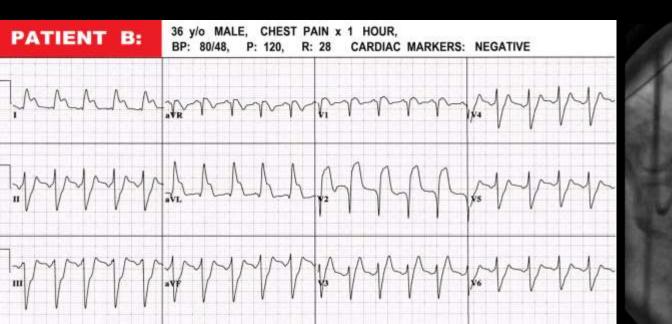


And

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

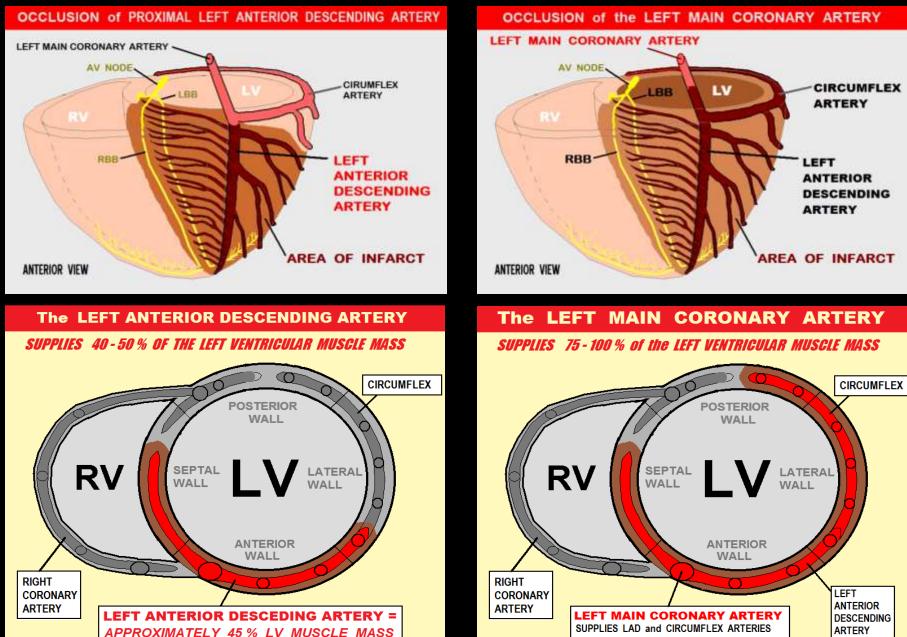






TOTAL OCCLUSION of LEFT MAIN CORONARY ARTERY

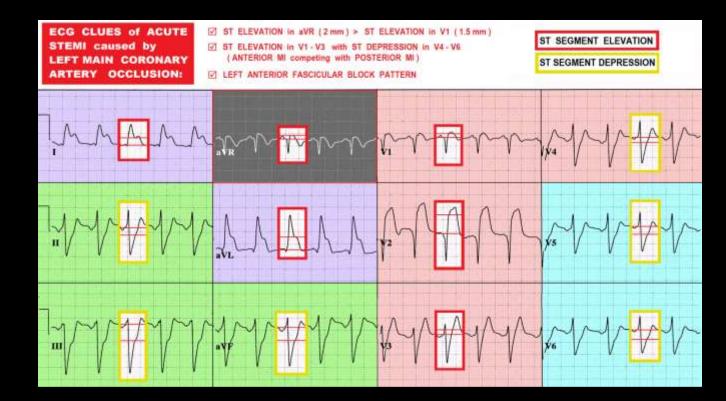
PATIENT A:



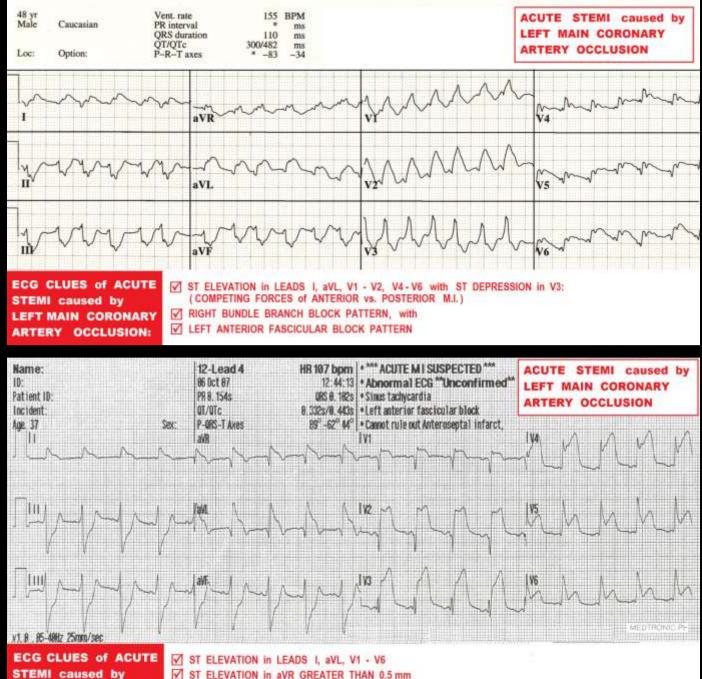
PATIENT B:

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

- ST ELEVATION in ANTERIOR LEADS (V1 V4) and LATERAL LEADS (V5 & V6)
- ST DEPRESSION or ISOELCTRIC J POINTS may be seen in V LEADS..., mainly V2 and/or V3 caused by COMPETING FORCES of ANTERIOR vs. POSTERIOR WALL ML**
 - → 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

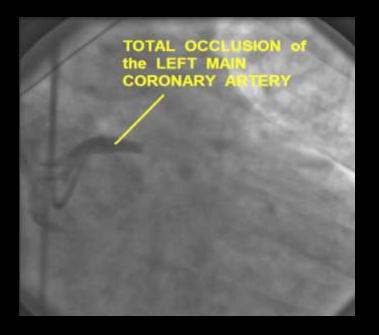


36 yr Male Vent. rate 123 **BPM** Sinus tachycardia with short PR ACUTE STEMI caused by Left ventricular hypertrophy with QRS widening Caucasian PR interval 96 105 QRS duration 130 Cannot rule out Septal infarct , age undetermined ITB LEFT MAIN CORONARY 310/443 Room:C-QT/QTc 43 Lateral injury pattern ACUTE MI Loc:3 P-R-T aves * -53 ARTERY OCCLUSION AVR 14 1 ш \$6 ECG CLUES of ACUTE M ST ELEVATION in leads 1 and aVL \mathbf{V} INCONSISTENCY of ST SEGEMENT in leads V1 · V6 : V1 · V3 ST ELEVATION, V4 - V6 ST DEPRESSION STEMI caused by (COMPETING FORCES of ANTERIOR vs. POSTERIOR M.I.) LEFT MAIN CORONARY \checkmark PATTERN of LEFT ANTERIOR FASCICULAR BLOCK (POS. QRS lead I; NEG rS leads II, III) ARTERY OCCLUSION: \checkmark ST ELEVATION in lead aVR > 0.5 mm Atrial fibrillation with rapid ventricular response 43 yr Male 183 BPM Vent, rate ACUTE STEMI caused by PR interval ٠ with premature ventricular or aberrantly conducted complexes ms 106 **ORS** duration ma LEFT MAIN CORONARY Left axis deviation QT/QTc 240/418 mes ST elevation consider anterolateral injury or acute infarct **ARTERY OCCLUSION** P-R-T axes * -34 -18 ** ** ** ** * ACUTE MI * ** ** ** ** WW why which ECG CLUES of ACUTE \checkmark ST ELEVATION in leads | and aVL STEMI caused by INCONSISTENCY of ST SEGEMENT in leads V1-V6: V1-V2 ST ELEVATION, V3-V6 ST DEPRESSION LEFT MAIN CORONARY (COMPETING FORCES of ANTERIOR vs. POSTERIOR M.I.) PATTERN of LEFT ANTERIOR FASCICULAR BLOCK (POS. QRS lead I; NEG rS leads II, III) ARTERY OCCLUSION:

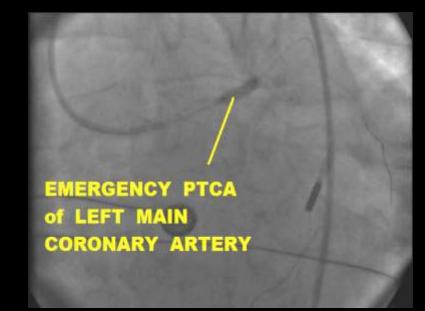


STEMI caused by S LEFT MAIN CORONARY S ARTERY OCCLUSION: S

✓ ST ELEVATION IN LEADS 1, aVL, V1 - V6
✓ ST ELEVATION IN aVR GREATER THAN 0.5 mm
✓ ST ELEVATION IN aVR GREATER THAN LEAD V1
✓ LEFT ANTERIOR FASCICULAR BLOCK PATTERN



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

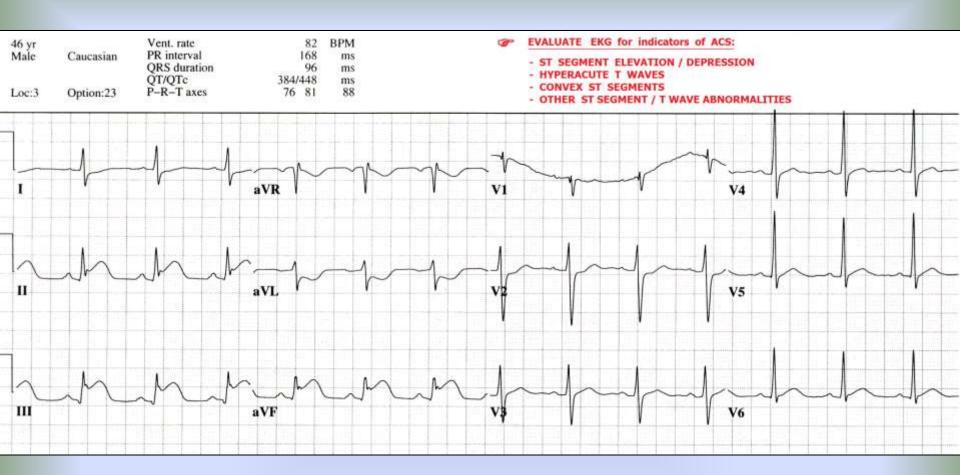
CHIEF COMPLAINT and SIGNIFICANT HISTORY:

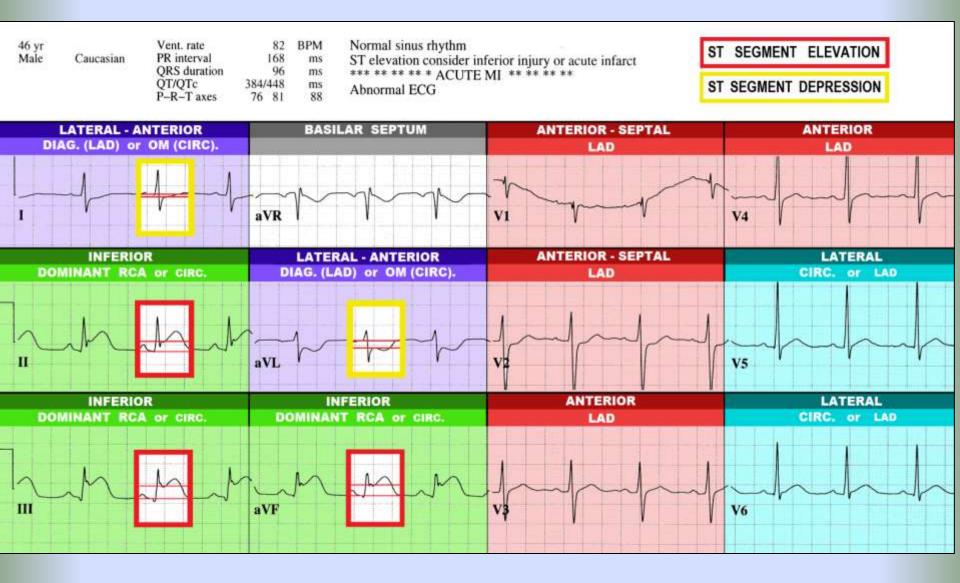
46 yr. old MALE arrives in ER, C/O SUDDEN ONSET OF CHEST PRESSURE 45 MINUTES AGO. PAIN IS CONSTANT, PRESSURE-LIKE, AND NOT EFFECTED BY POSITION, MOVEMENT or DEEP INSPIRATION. ALSO C/O D.I.B.

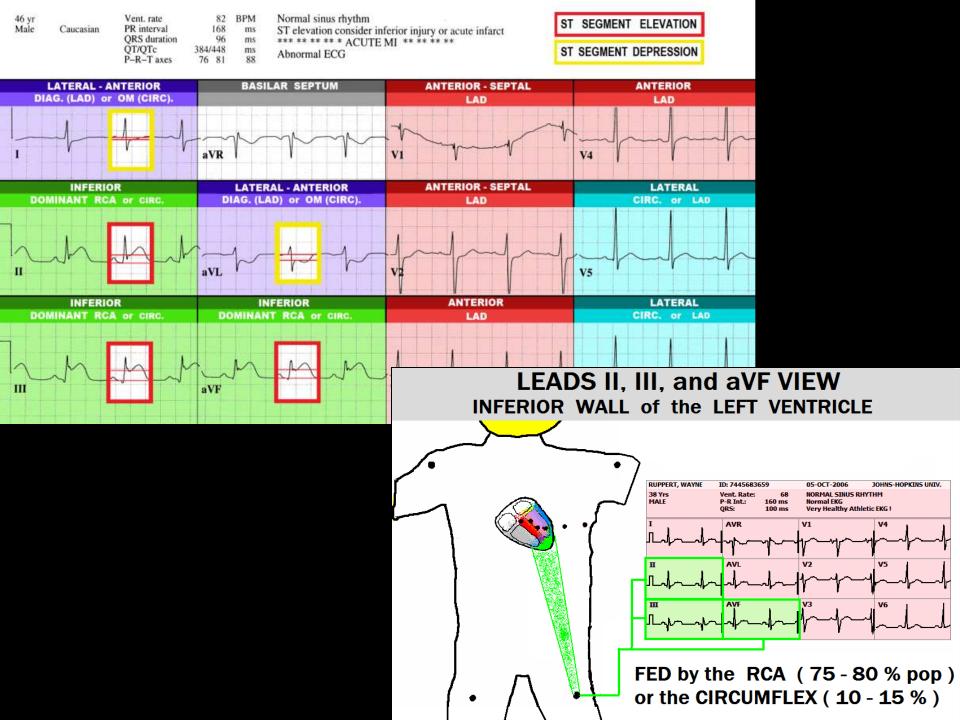
RISK FACTOR PROFILE:

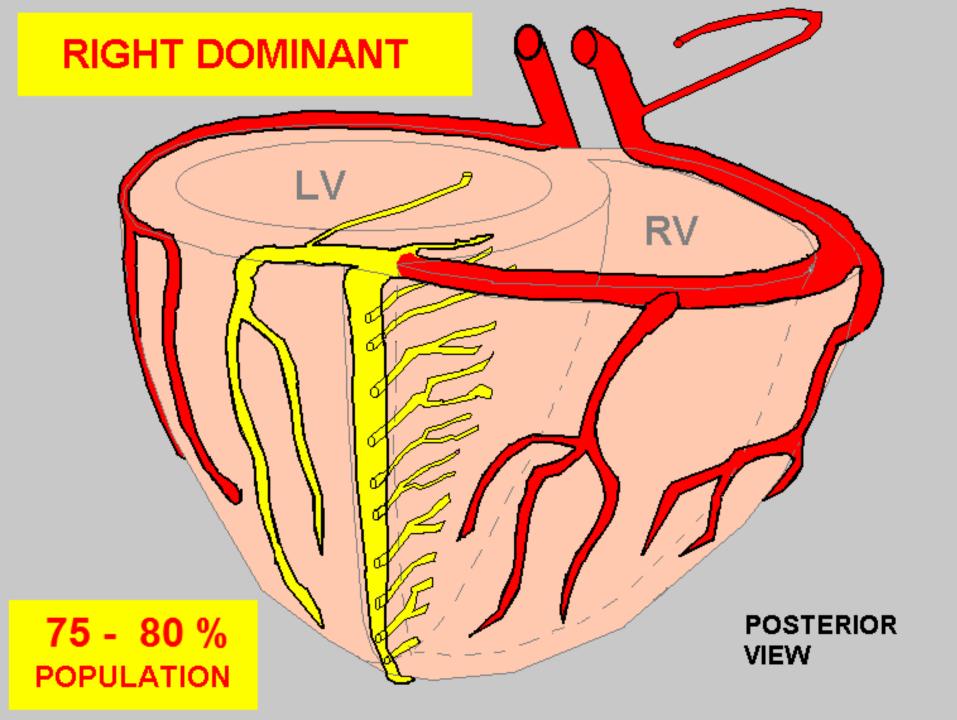


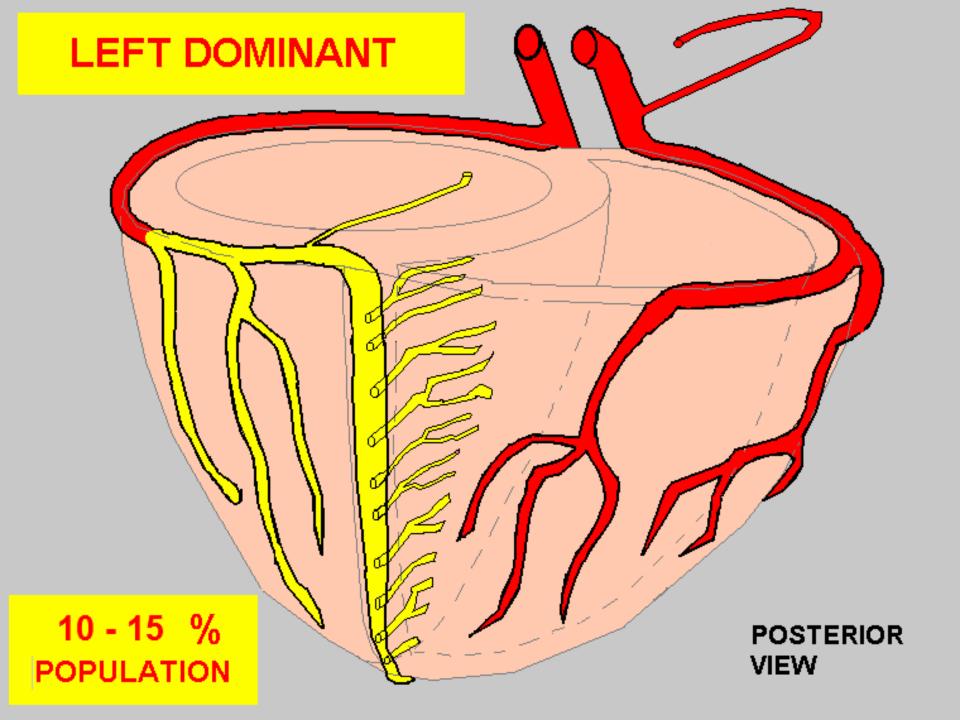
LABS: TROPONIN: < .04







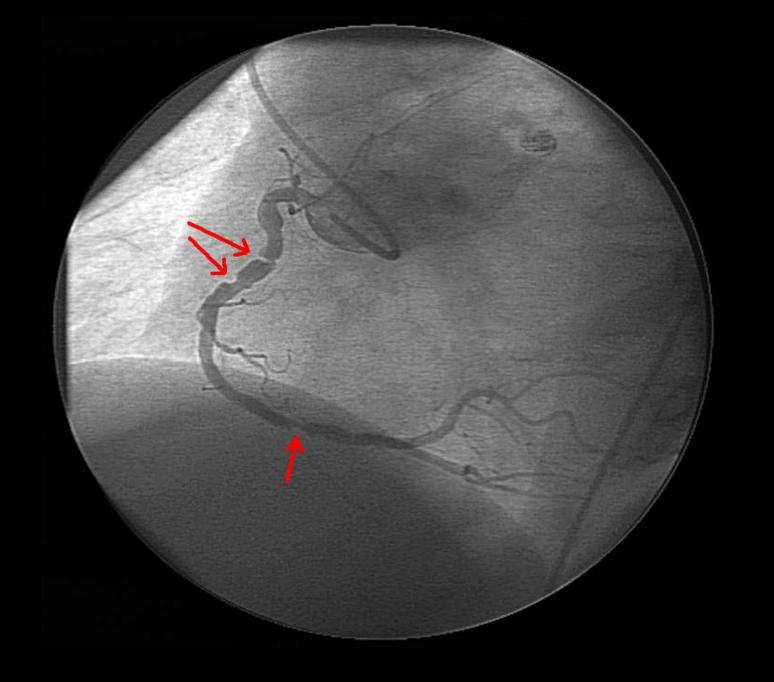




- 🎗 —> HELPFUL HINT ... MEMORIZE THIS ! 🔶 **RIGHT CORONARY ARTERY (RCA)** HT DOMINANT SYSTEMS RIGHT ATRIUM SINUS NODE (55% of the population) RIGHT VENTRICLE - 100 % of muscle mass LEFT VENTRICLE: 15 - 25 % of muscle mass - INFERIOR WALL - approx. 1/2 of POSTERIOR WALL AV NODE

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

- CARDIAC ARREST	BCLS / ACLS
- CARDIAC DYSRHYTHMIAS (VT / VF)	ACLS (antiarrhythmics)
- SINUS BRADYCARDIA	ATROPINE 0.5mg, REPEAT as needed UP TO 3mg. (follow ACLS and/or UNIT protocols)
- HEART BLOCKS (1st, 2nd & 3rd Degree HB)	ATROPINE 0.5mg, REPEAT as needed UP TO 3mg, Transcutaneous Pacing, (follow ACLS and/or UNIT protocols)
- RIGHT VENTRICULAR MYOCARDIAL INFARCTION	 The standard 12 Lead ECG does NOT view the Right Ventricle. You must do a RIGHT-SIDED ECG to see if RV MI is present. Do NOT give any Inferior Wall STEMI patient NITRATES or DIURETICS until RV MI has been RULED OUT.
- POSTERIOR WALL INFARCTION	 POSTERIOR WALL MI presents on the 12 Lead ECG as ST DEPRESSION in Leads V1 - V3. POSTERIOR WALL MI is NOT PRESENT ON THIS ECG.



۲ د

A standard

12 LEAD EKG

Does NOT show the

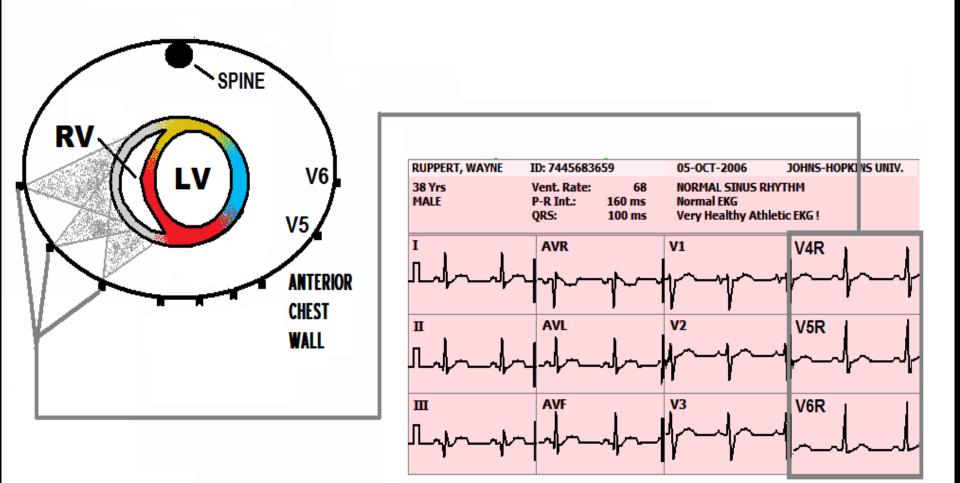
RIGHT VENTRICLE

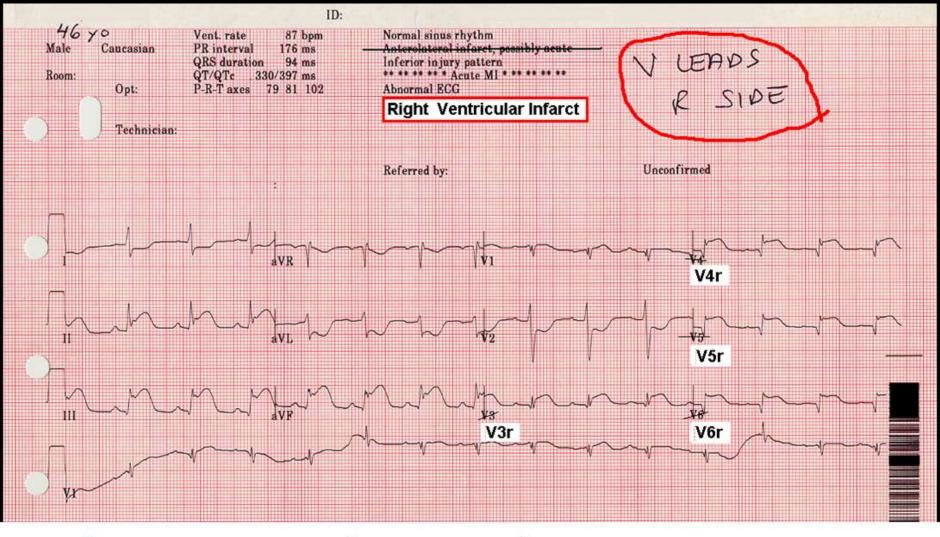
To see the RIGHT VENTRICLE ...

... 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

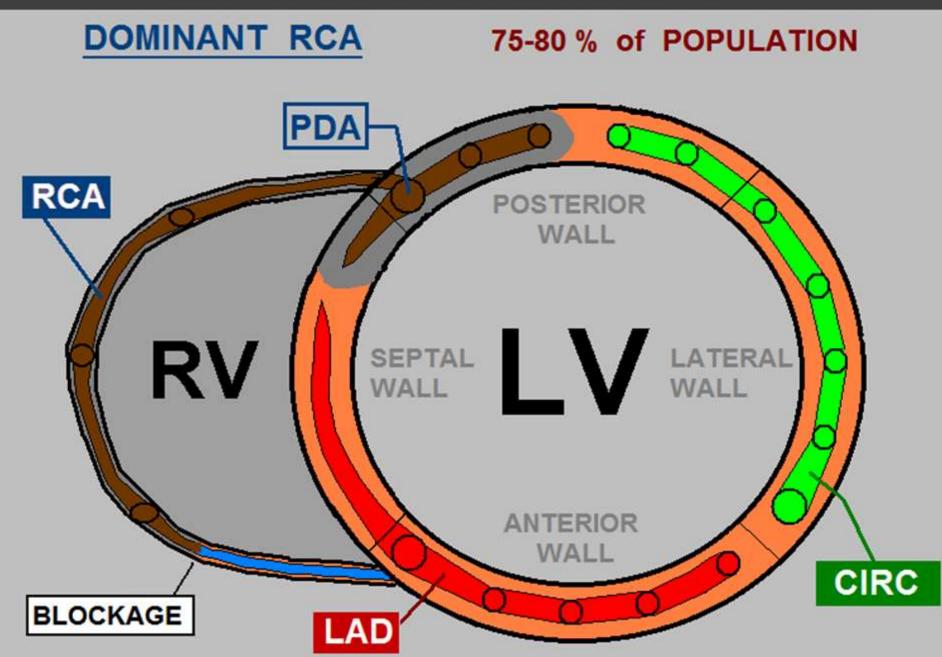
You must first *RULE OUT* **RIGHT VENTRICULAR MI BEFORE** giving any:

- NITROGLYCERIN
- Diuretics

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

* A.H.A. ACLS 2010 / 2015

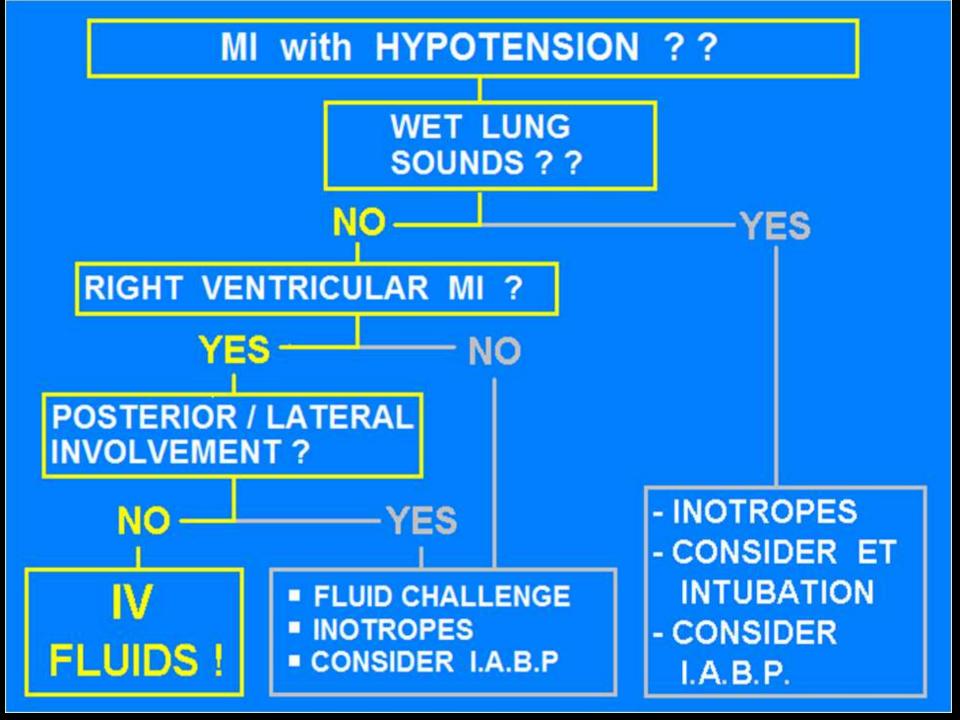
INFERIOR - RIGHT VENTRICULAR MI

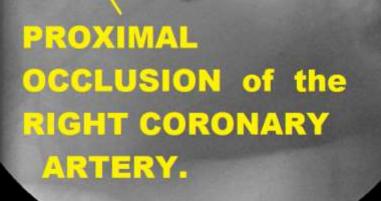


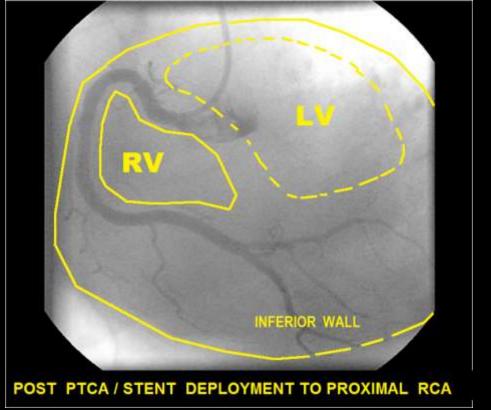
ANTICIPATED COMPLICATIONS of INFERIOR - RIGHT VENRICULAR WALL STEMI secondary to PROXIMAL RCA Occlusion & POSSIBLE INDICATED INTERVENTIONS:

- CARDIAC ARREST	BCLS / ACLS
- CARDIAC DYSRHYTHMIAS (VT / VF)	ACLS (antiarrhythmics)
- SINUS BRADYCARDIA	ATROPINE 0.5mg, REPEAT as needed UP TO 3mg. (follow ACLS and/or UNIT protocols)
- HEART BLOCKS (1st, 2nd & 3rd Degree HB)	ATROPINE 0.5mg, REPEAT as needed UP TO 3mg, Transcutaneous Pacing, (follow ACLS and/or UNIT protocols)
- RIGHT VENTRICULAR MYOCARDIAL INFARCTION	 NITRATES and DIURETICS are CONTRA- INDICATED. TREAT HYPOTENSION WITH FLUIDS. (It is Not uncommon to give 500-2000ml of NORMAL SALINE to stabilize BP.
- POSTERIOR WALL INFARCTION	 POSTERIOR WALL MI presents on the 12 Lead ECG as ST DEPRESSION in Leads V1 - V3. POSTERIOR WALL MI is NOT PRESENT ON THIS ECG.

If this patient becomes HYPOTENSIVE







CASE STUDY 9 - STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

RISK FACTOR PROFILE:

- CIGARETTE SMOKER
- HYPERTENSION
- HIGH LDL CHOLESTEROL

PHYSICAL EXAM: Patient is alert & oriented x 4, ANXIOUS, with COOL, PALE, DIAPHORETIC SKIN. C/O NAUSEA, and is VOMITING. LUNG SOUNDS: COARSE CRACKLES, BASES, bilaterally VITAL SIGNS: BP: 80/40 P: 70 R: 32 SAO2: 92% on 15 LPM O2

LABS: TROPONIN: < .04

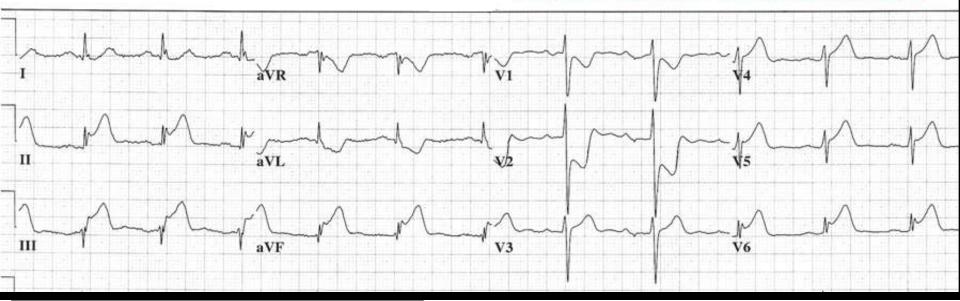
SHOCK ASSESSMENT

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

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

C EVALUATE EKG for indicators of ACS:

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



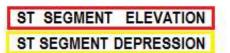
CASE STUDY QUESTIONS:

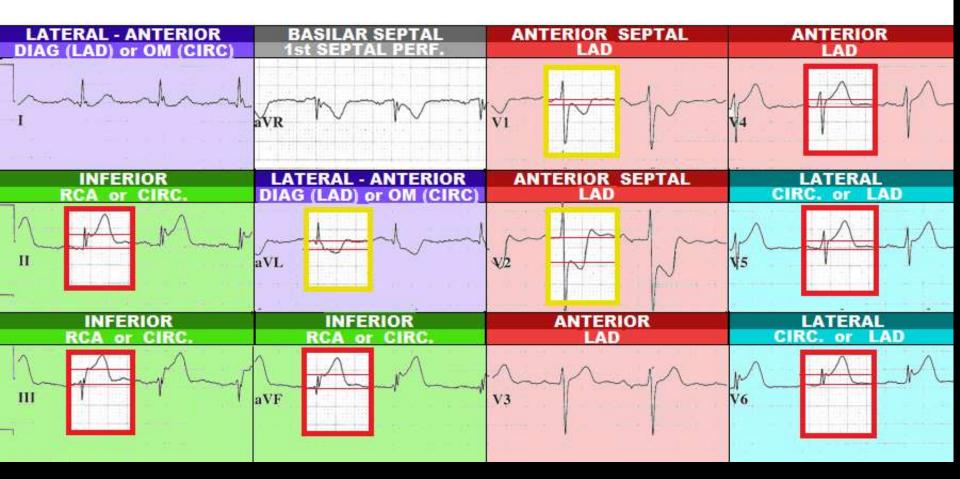
NOTE LEADS WITH ST ELEVATION:	NOTE LEADS WITH ST DEPRESSION:
WHAT IS THE SUSPECTED DIAGNOSIS ?	
WHAT IS THE "CULPRIT ARTERY" if applicable ?	
LIST ANY CRITICAL STRUCTURES COMPROMISED:	LIST ANY POTENTIAL COMPLICATIONS:

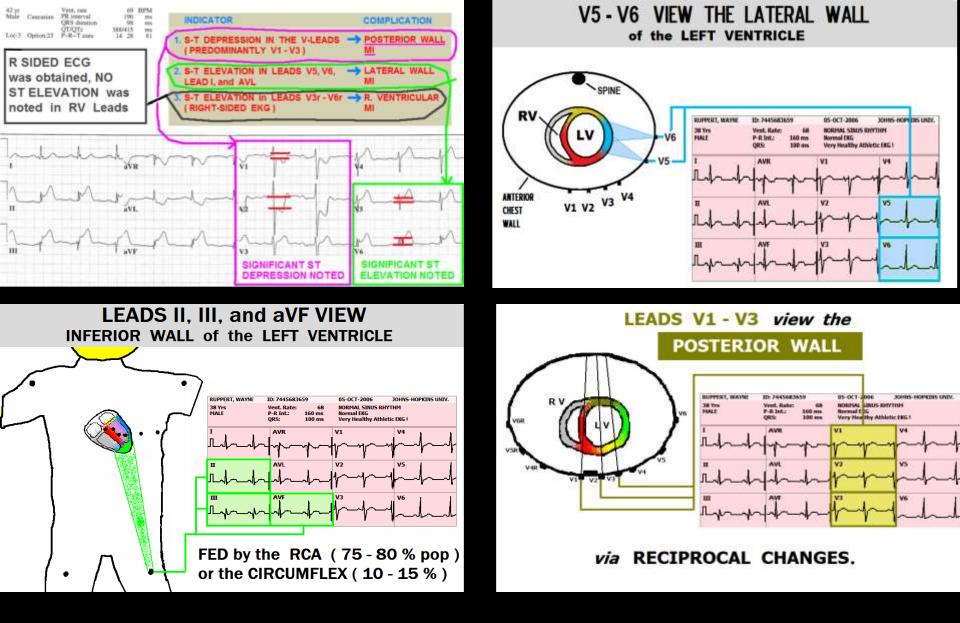
42 yr		Vent. rate	69	BPM
Male	Caucasian	PR interval	196	ms
		QRS duration	98	ms
G		QT/QTc	388/415	ms
Loc:3	Option:23	P-R-T axes	14 28	81

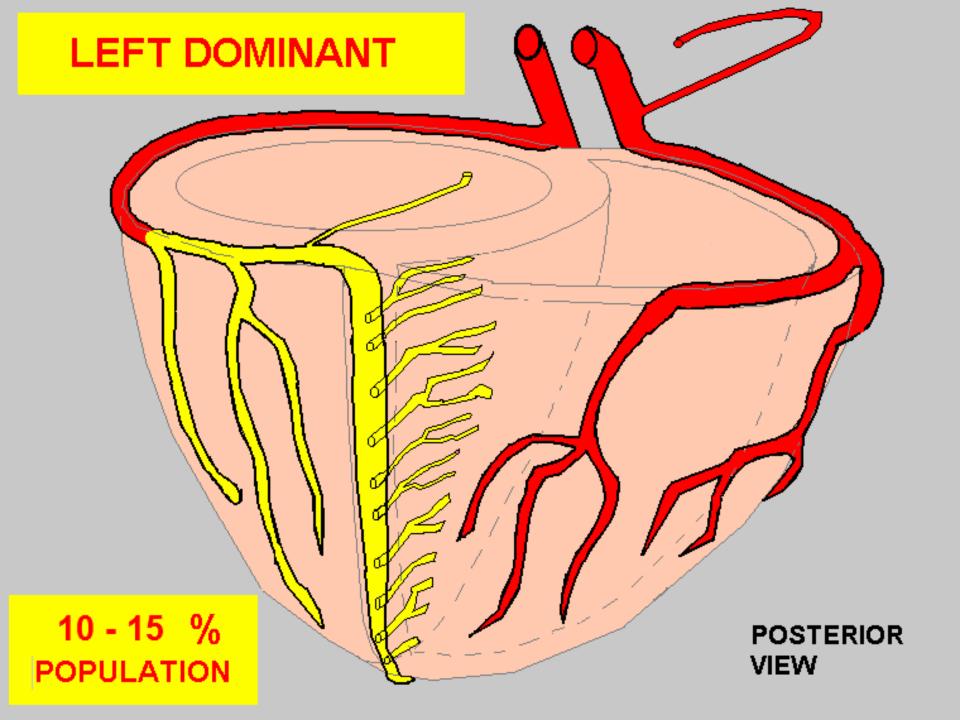
*** Acute MI ***

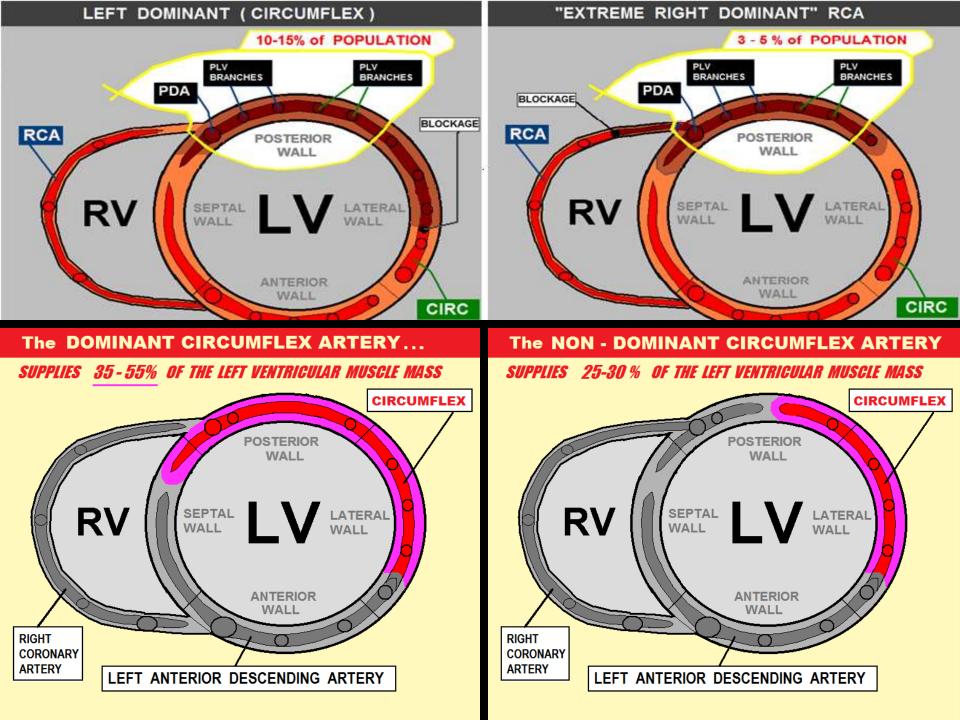
Inferior-Posterior-Lateral Injury Pattern

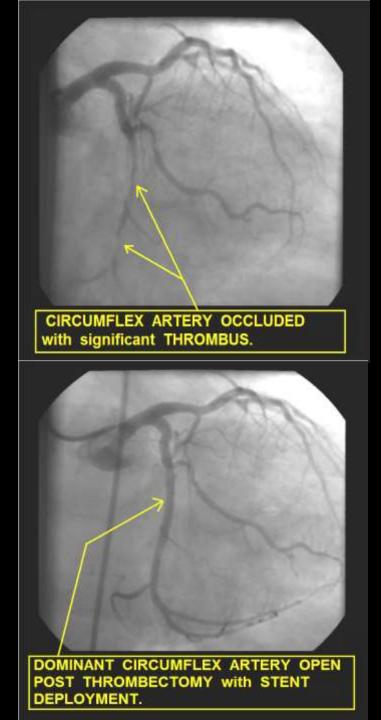


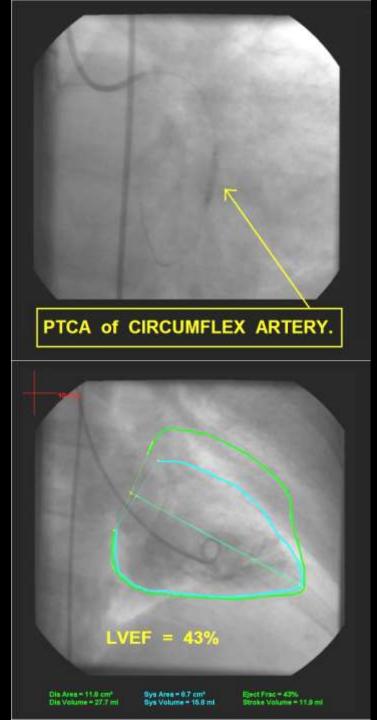


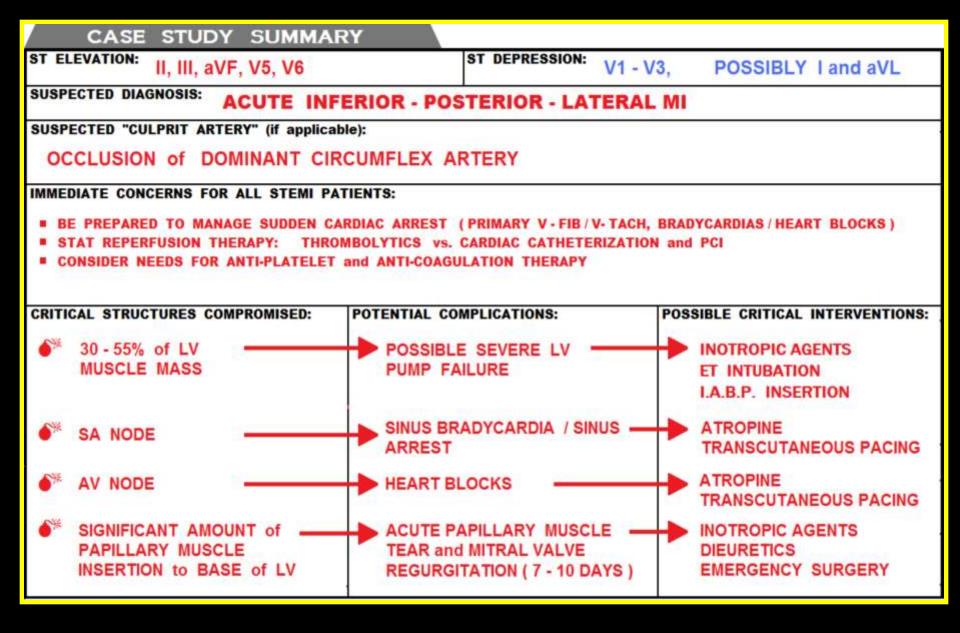






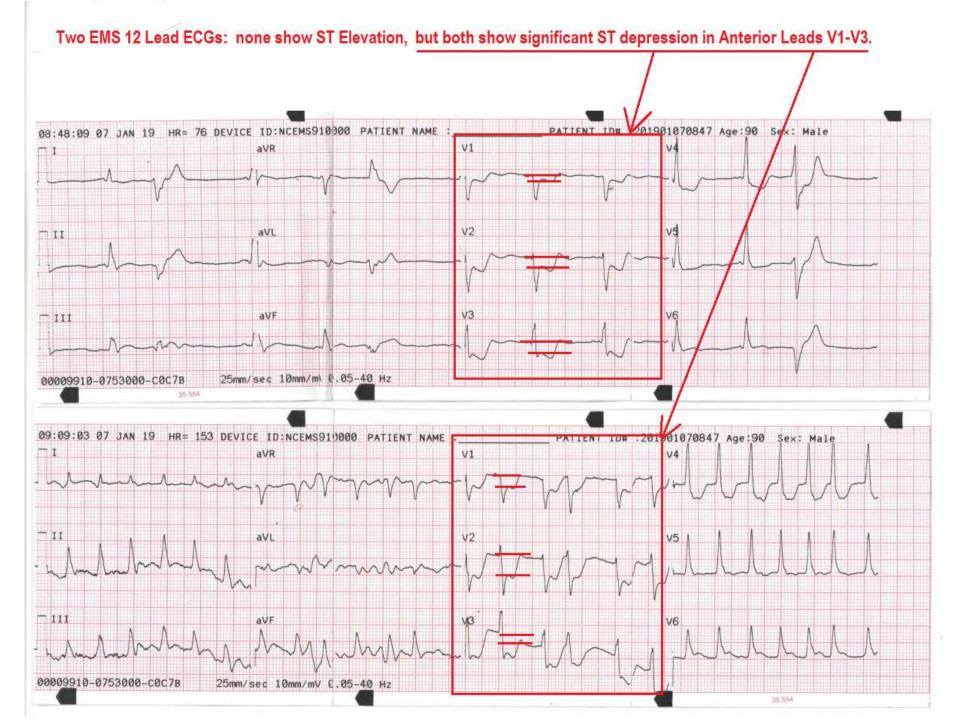






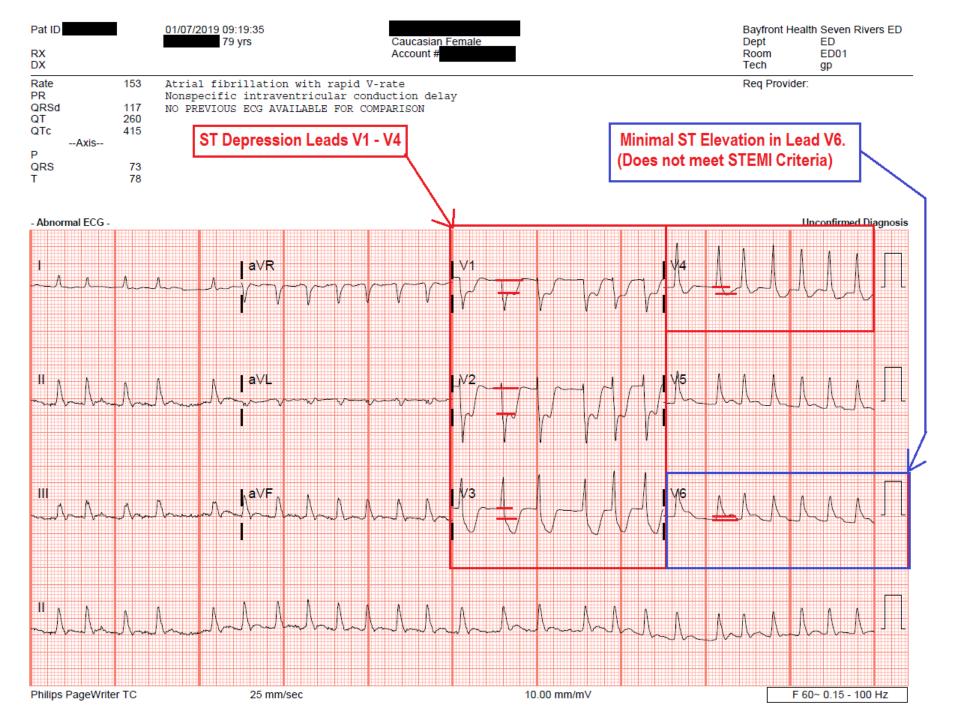
Case Study-January 2019

- 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.

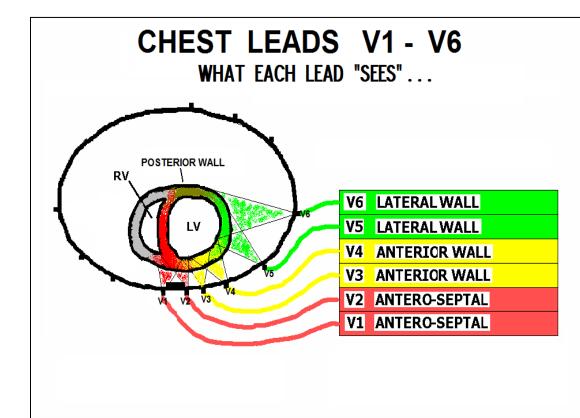


Causes of ST Depression V1-V4

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

Posterior Wall STEMI....

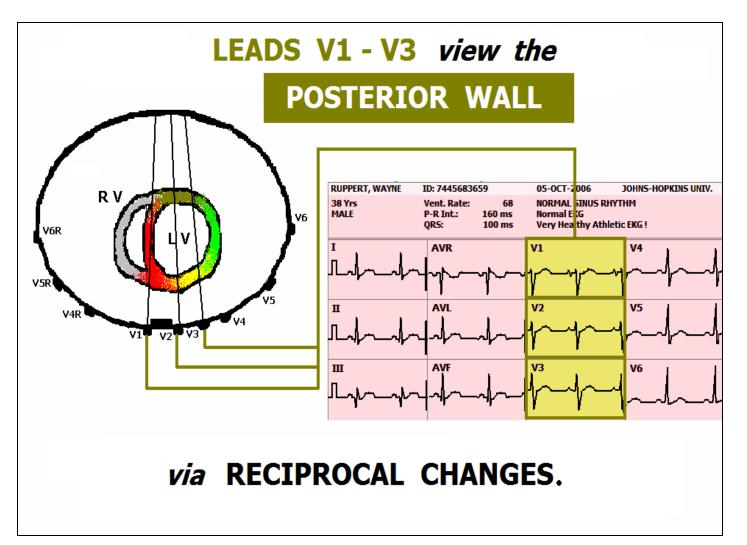
 Does not show ST elevation on standard 12 lead ECG because NONE of the 12 leads view the Posterior Wall directly....

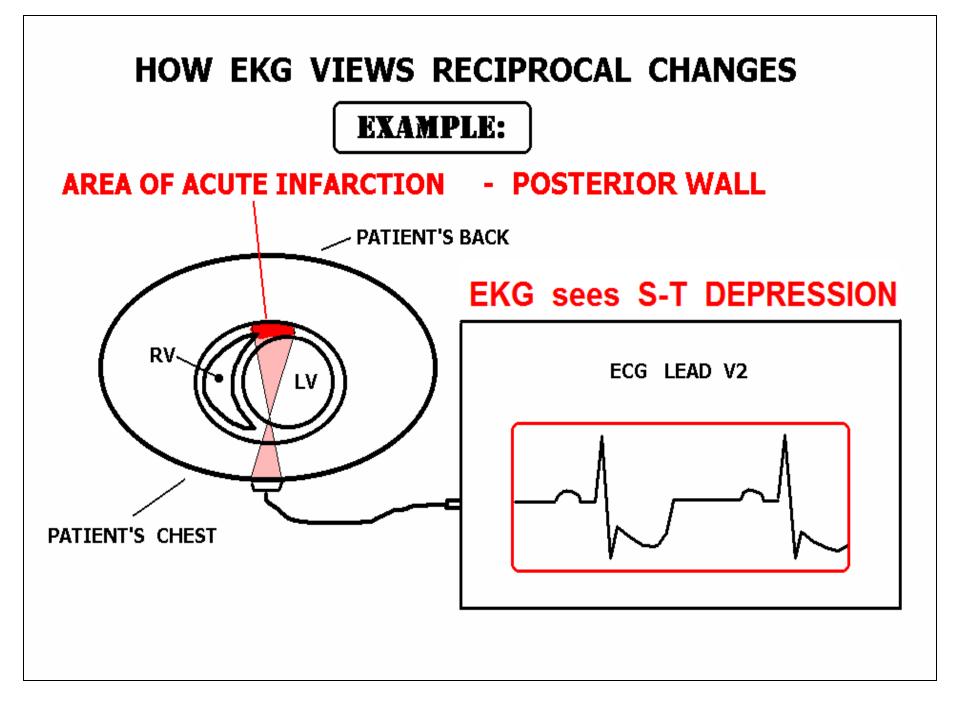


Posterior Wall STEMI....

- 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)



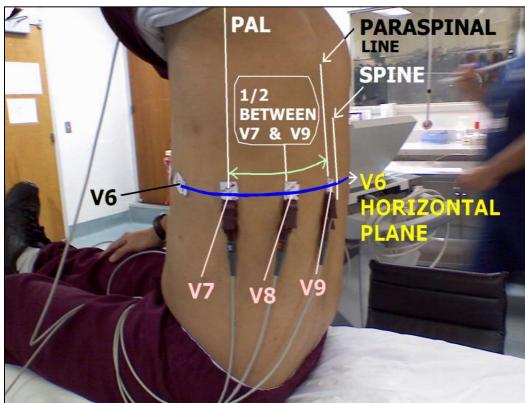


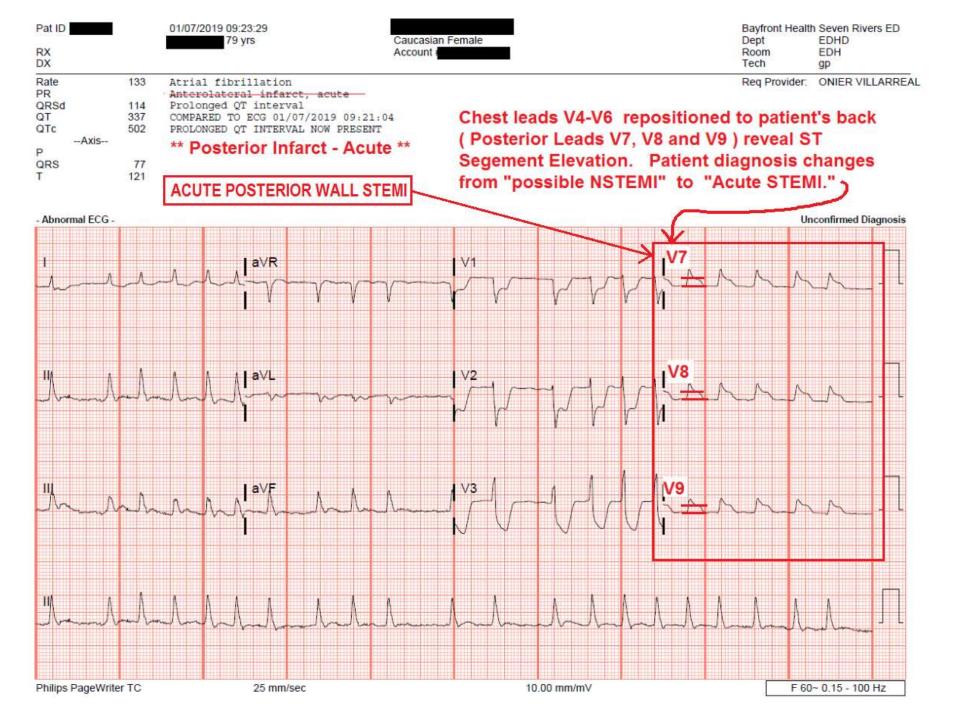
Posterior Wall STEMI....

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

Continued Exam in the ED....

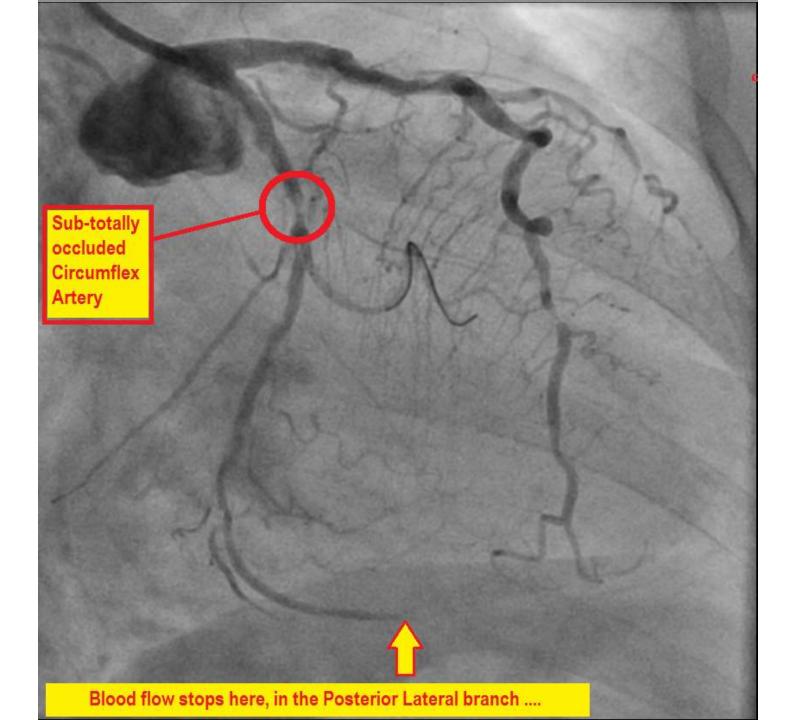
- Upon noting ST Depression in Anterior Leads V1-V4, ED Paramedic Gary Polizzi place three leads on the patient's back. Gary used the lead wires for V4, V5 and V6, with placement as shown here:
- The "Posterior Lead ECG" is seen on the next slide.....

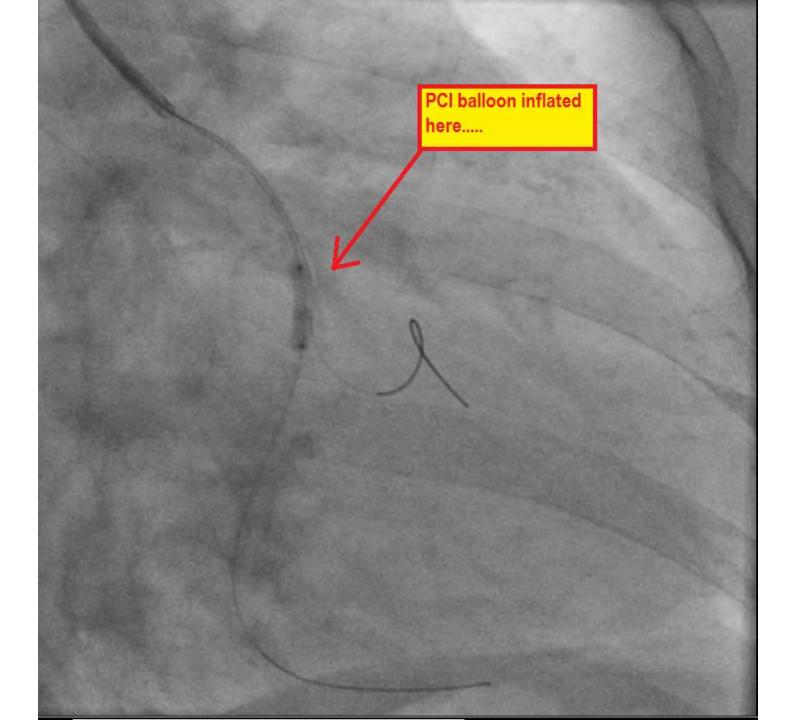


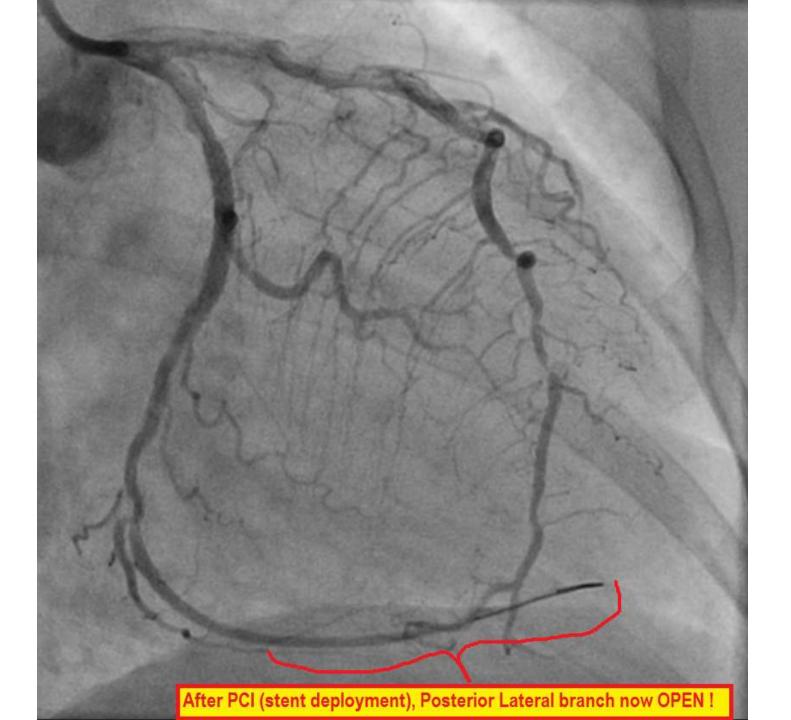


STEMI Alert !

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......

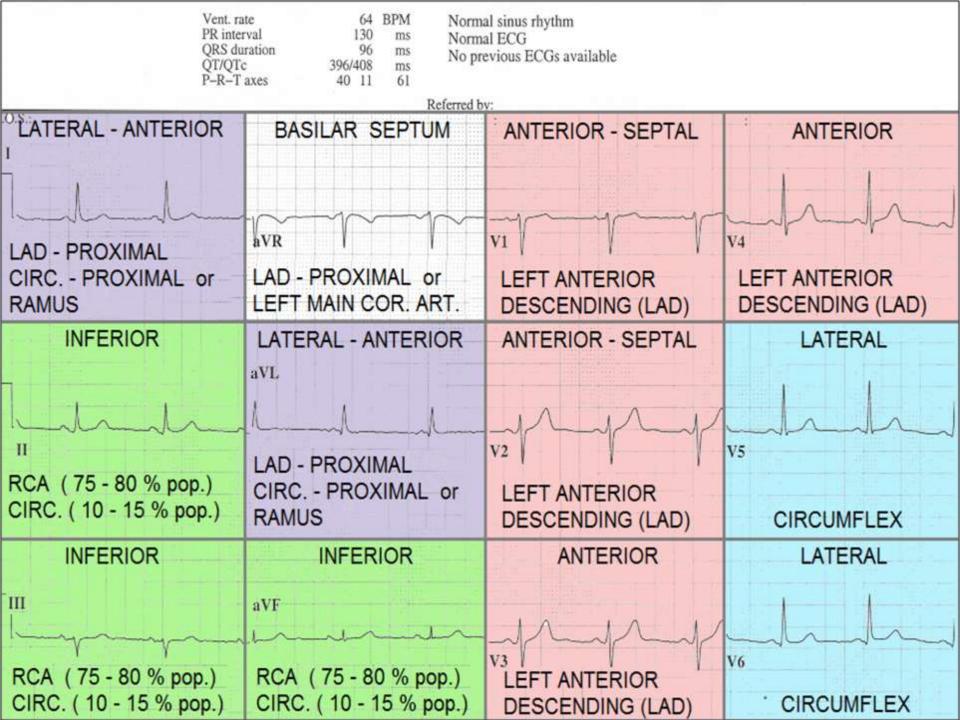






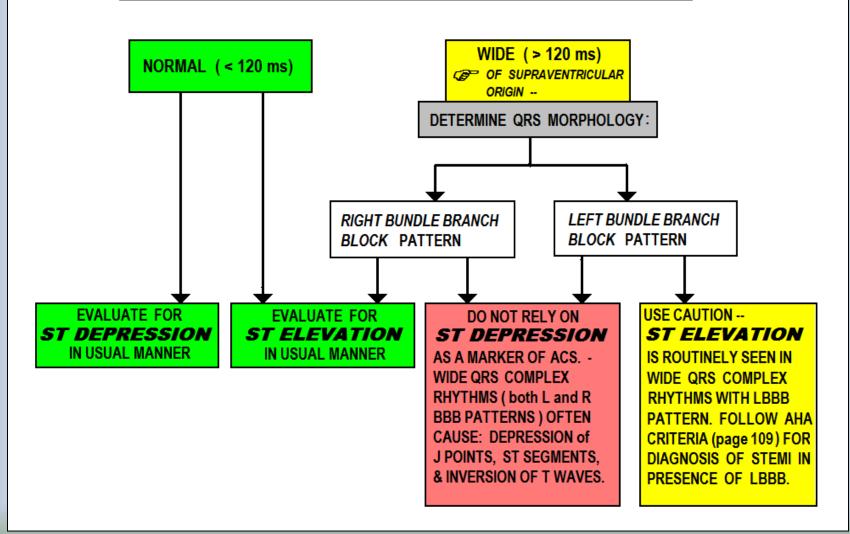
SUMMARY

- 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:





 Determine RIGHT vs. LEFT Bundle Branch Block Pattern

Simple "Turn Signal Method" . . .

THE "TURN SIGNAL METHOD" for identifying BUNDLE BRANCH BLOCK

USE LEAD V1 for this technique

To make a **RIGHT TURN**

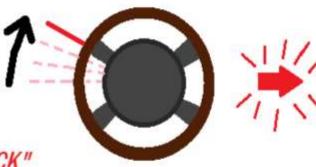
you push the turn signal lever UP.....

THINK:

V1

V1

"QRS points UP = RIGHT BUNDLE BRANCH BLOCK"



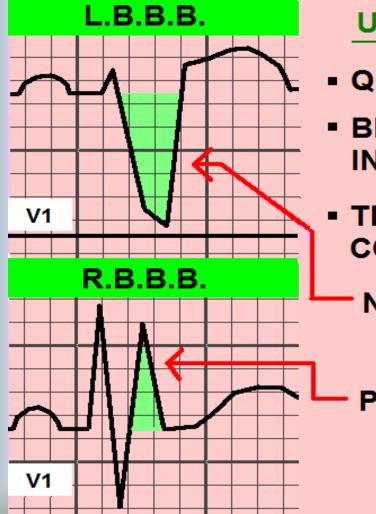
To make a **LEFT TURN** you push the turn signal lever **DOWN**

THINK:

"QRS points DOWN = LEFT BUNDLE BRANCH BLOCK"

"Terminal Phase of QRS Method"...

DIAGNOSING BUNDLE BRANCH BLOCK



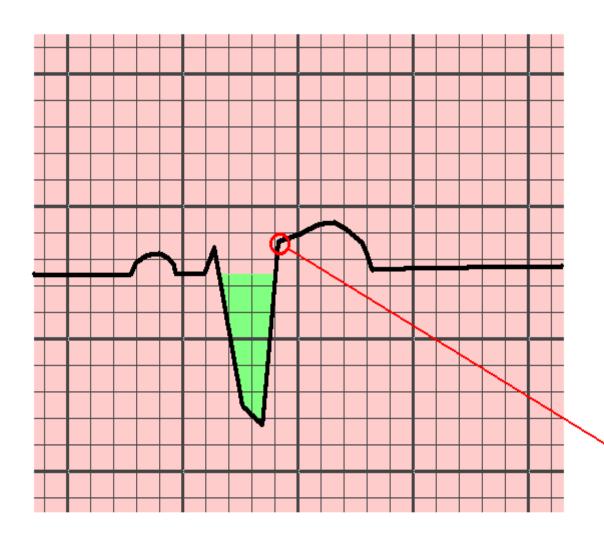
USING LEAD V1

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

NEGATIVE = LEFT BUNDLE BRANCH BLOCK

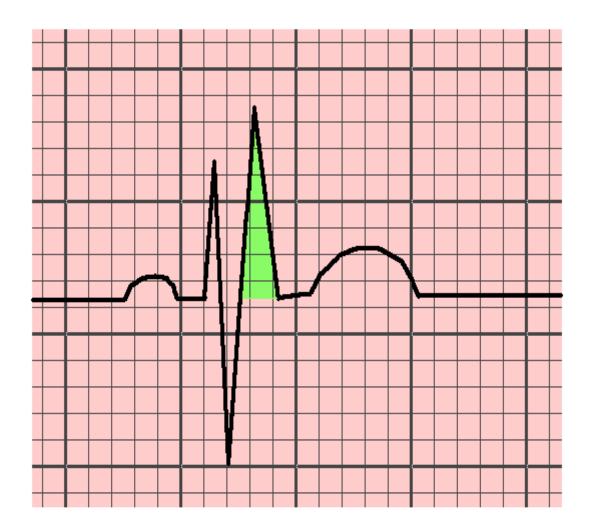
- POSITIVE = RIGHT BUNDLE BRANCH BLOCK

DIAGNOSING 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:

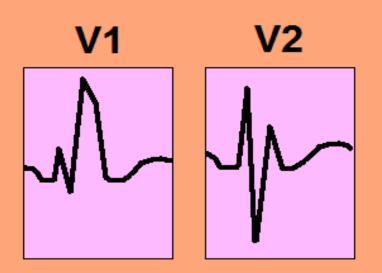


- WIDER THAN 120 ms (.12)
- (or 3 little boxes)
- TERMINAL PHASE (LAST PART) OF QRS COMPLEX IS POSITIVE DEFLECTION

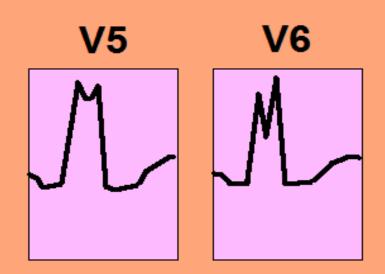
DIAGNOSING BUNDLE BRANCH BLOCK

USING LEADS V1, V2, and V5, V6:

LOCATING RsR' or RR' COMPLEXES:

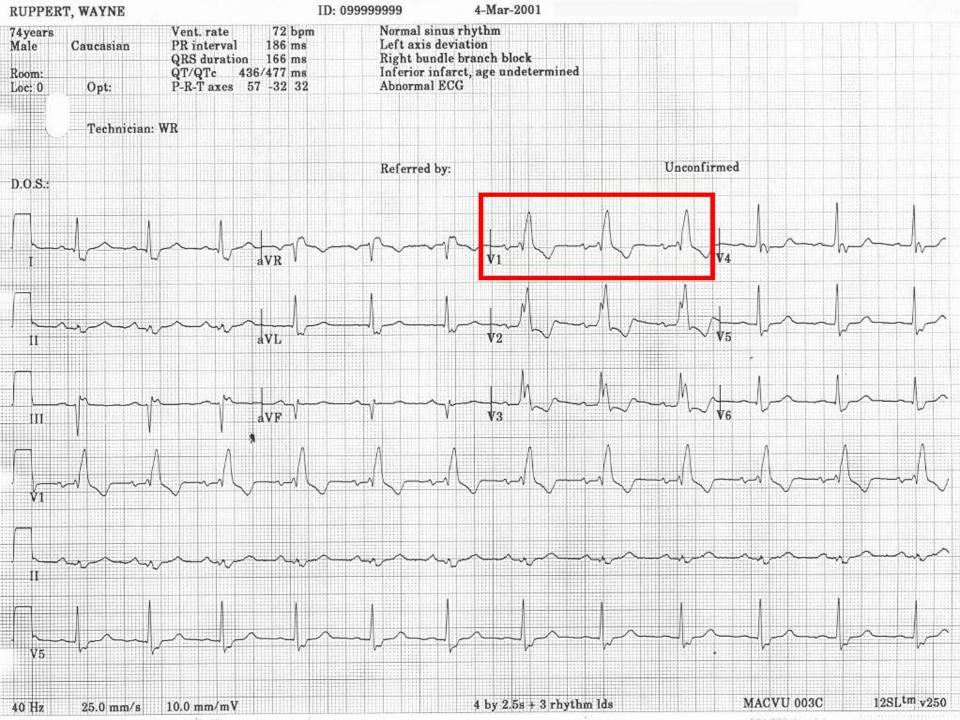


RIGHT BUNDLE BRANCH BLOCK

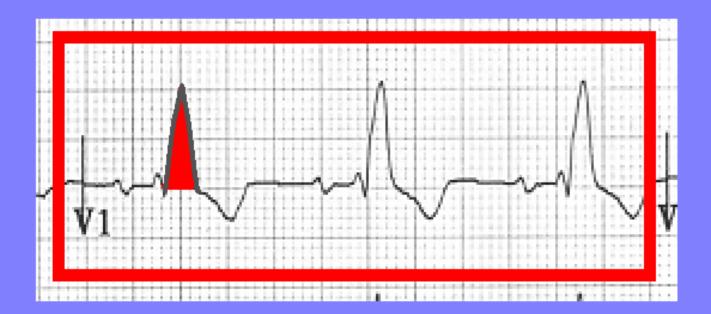


LEFT BUNDLE BRANCH BLOCK

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



TERMINAL PHASE OF QRS IS POSITIVE



= RIGHT BUNDLE BRANCH BLOCK

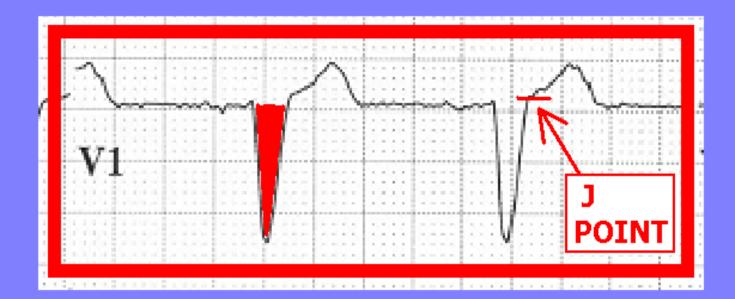
09:16:40

74 yr Female	Caucasian	Vent. rate PR interval QRS duration	64 188 152	BPM ms ms	Normal sinus rhythm Left bundle branch block Abnormal ECG When compared with ECG of 28–MAY–2003 06:36,
Loc:7	Option:35	QT/QTc P-R-T axes EKG #WR030	472/486 78 3)29959	ms 106	

Technician: WW

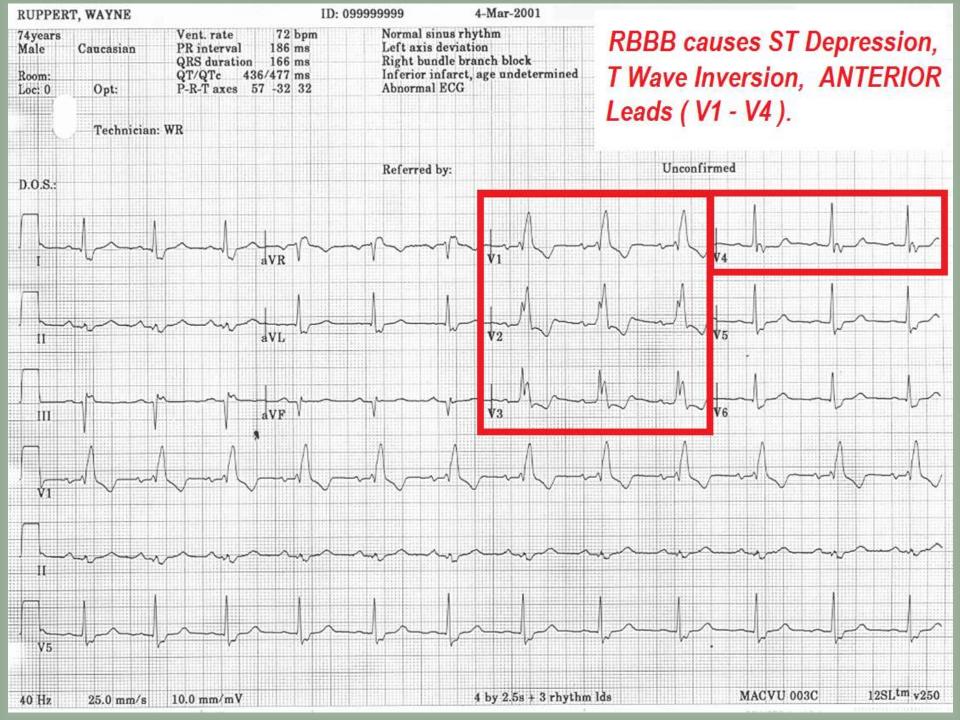


TERMINAL PHASE OF QRS IS **NEGATIVE**



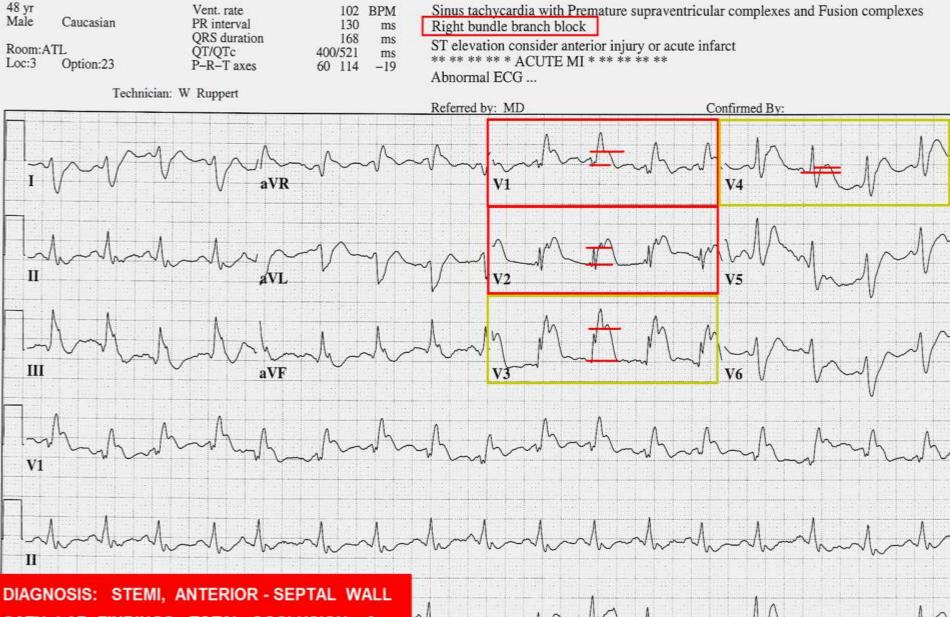
= LEFT BUNDLE BRANCH BLOCK

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



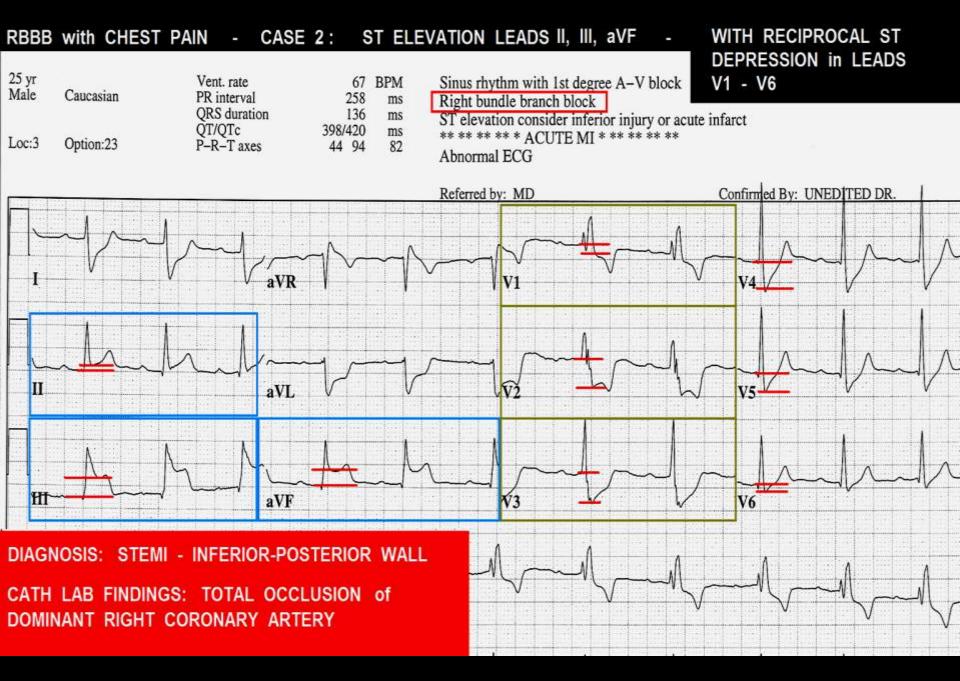
- When RIGHT Bundle Branch Block pattern is present:
 - Precordial Leads typically demonstrate ST
 Depression and T wave Inversion
 - DOES NOT MASK STEMI; when ST Elevation is noted, CONSIDER STEMI ! !

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

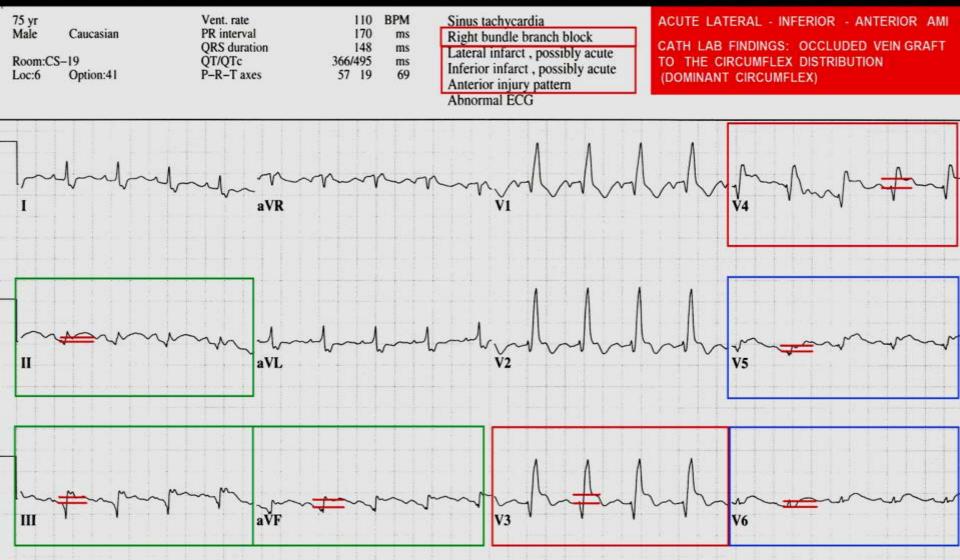


CATH LAB FINDINGS: TOTAL OCCLUSION of mid - LEFT ANTERIOR DESCENDING ARTERY.





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



When LBBB QRS pattern is present:

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

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

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

2013 ACC/AHA Guideline for Management of STEMI

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

2013 ACC/AHA Guideline for Management of STEMI

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

2013 ACC/AHA Guideline for Management of STEMI

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

2013 ACC/AHA Guideline for Management of STEMI

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

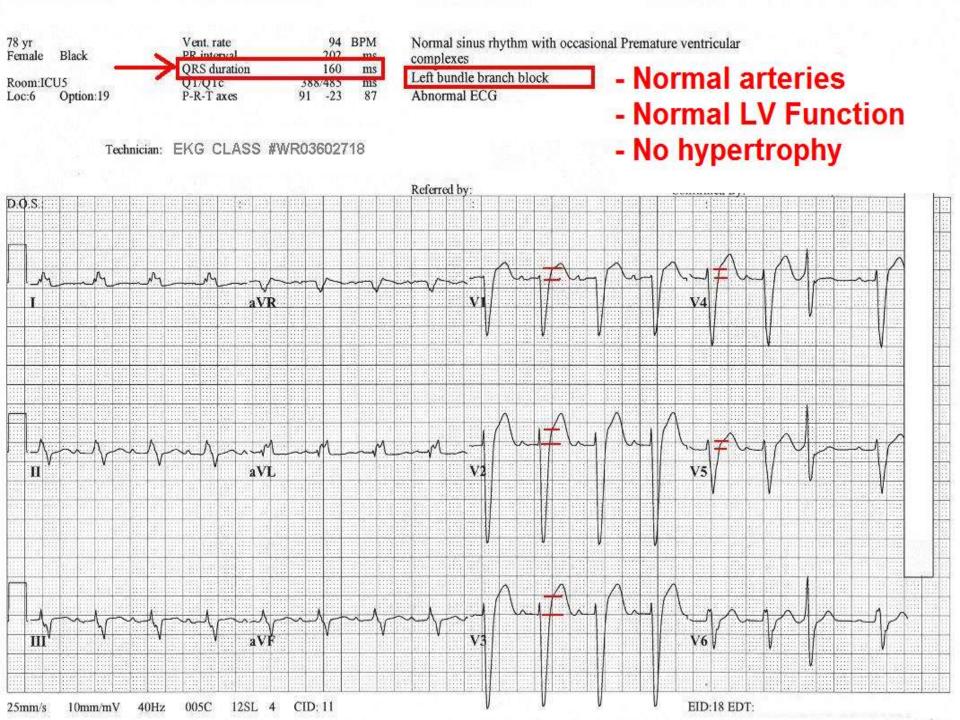
A.H.A. ACLS GUIDELINES

- 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.





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

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

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

"Electrocardiographic Diagnosis of Evolving Acute Myocardial Infarction in the Presence of Left Bundle-Branch Block" Birnbaum et al, N Engl J Med 1996; 334:481-487 Be advised that in patients with

Left Bundle Branch Block Combined with Ventricular Hypertrophy,

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

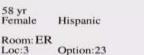
LBBB with CHEST PAIN - CASE 1: PRESENTING EKG

BPM

ms

ms

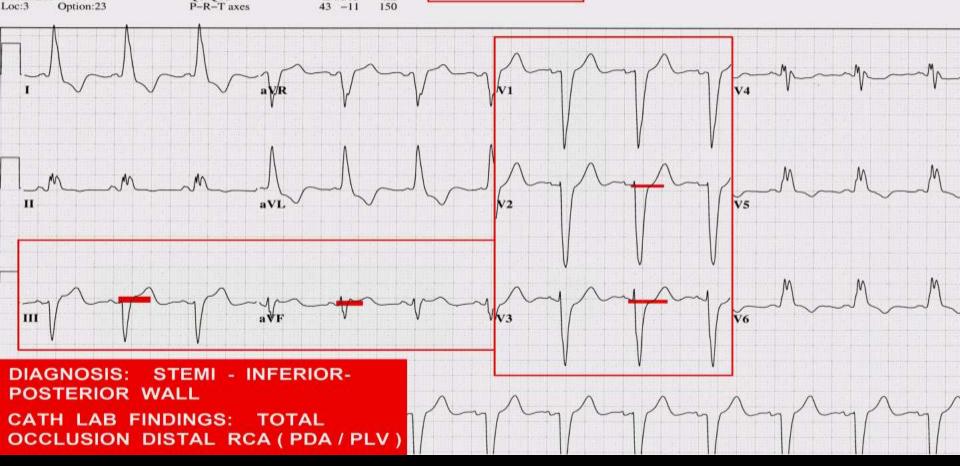
ms

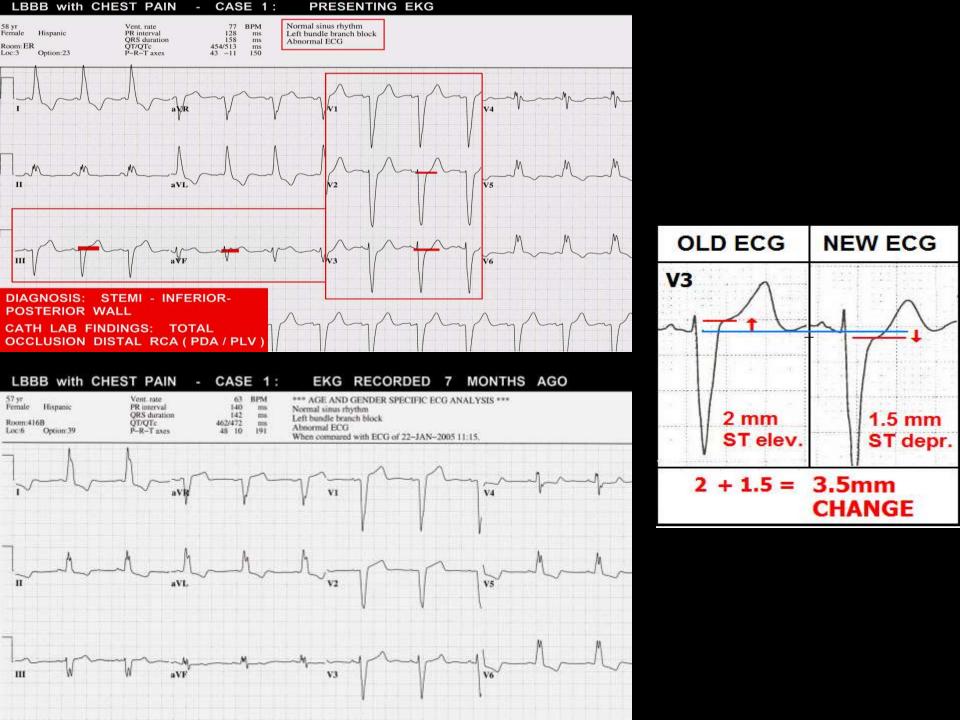


Vent. rate PR interval QRS duration QT/QTc 77 128 158 454/513 43 -11

Normal sinus rhythm Left bundle branch block

Abnormal ECG





LBBB with CHEST PAIN NEW ONSET of LBBB CASE 2:

77

172

142

38 0

BPM

ms

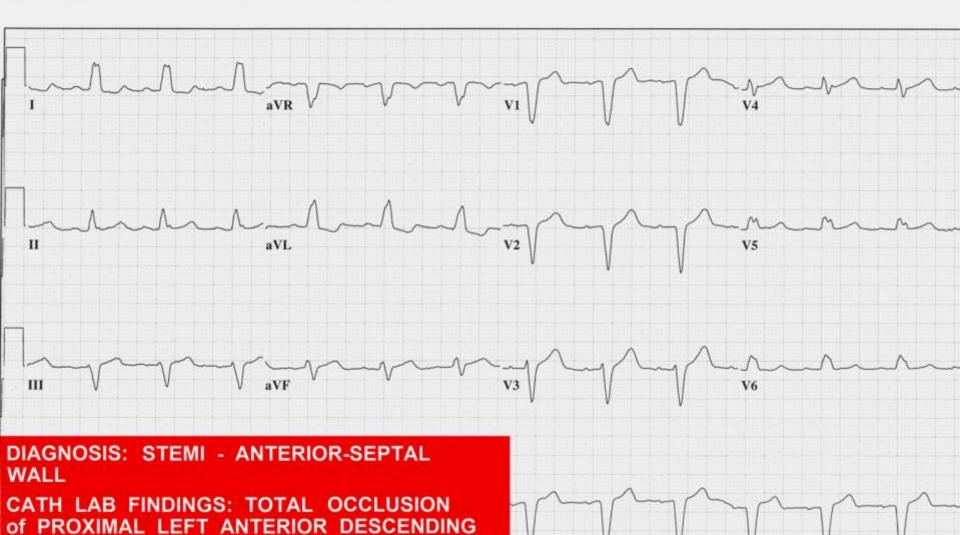
ms

ms

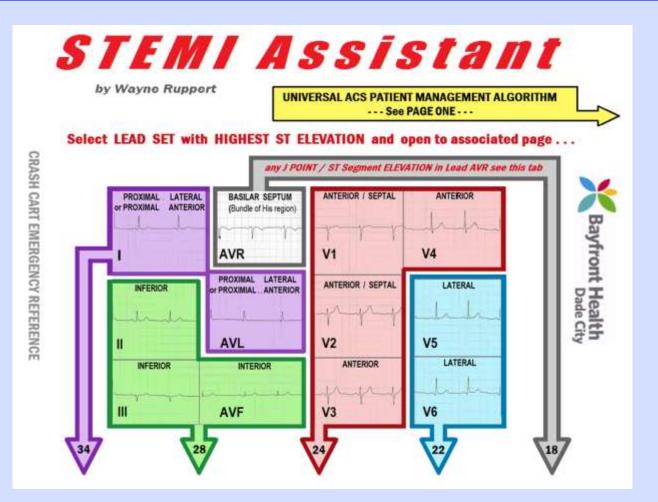
92



Normal sinus rhythm Left bundle branch block Abnormal ECG



STEIL ASSISTANC: an Emergency Crash Cart Interactive Reference Manual - free Download



STEMI Assistant – Information Video

Helpful STEMI ECG Resources

^[1] <u>"Use of the Electrocardiogram in Acute Myocardial</u> 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

ECG in STEMI – excellent powerpoint – quick reference, in-depth material

Helpful STEMI ECG Resources

Download Non-ED STEMI Protocol - example

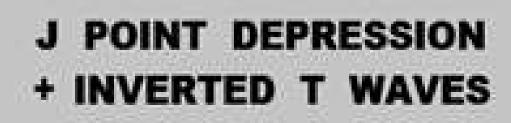
Download STEMI Alert ED Physicians Order Set

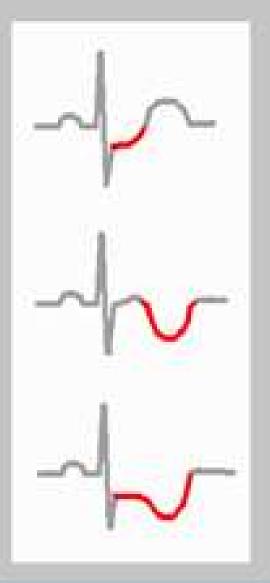




J POINT DEPRESSION (>1 mm)

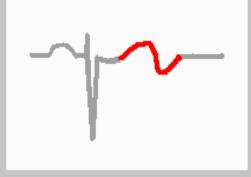
INVERTED T WAVES





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





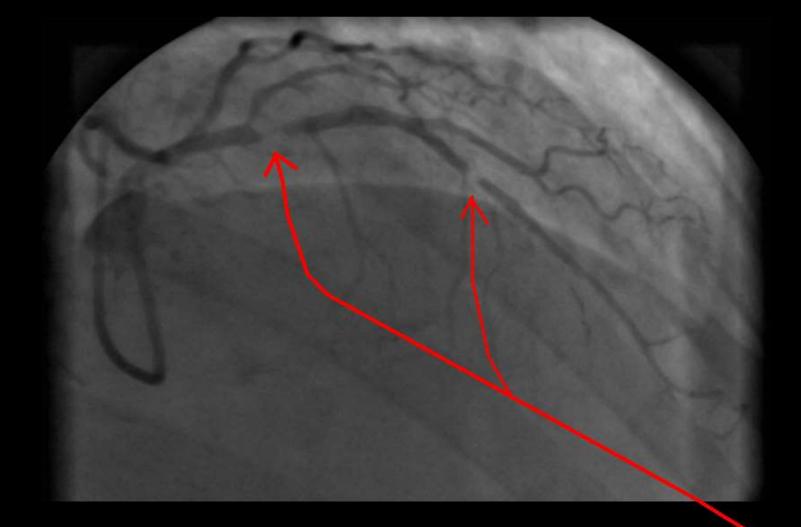
BI-PHASIC T WAVE

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

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



BI-PHASIC T WAVES



58 y/o MALE WITH "WELLEN'S WARNING." PT HAS SUB-TOTALLY OCCLUDED LAD X2

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

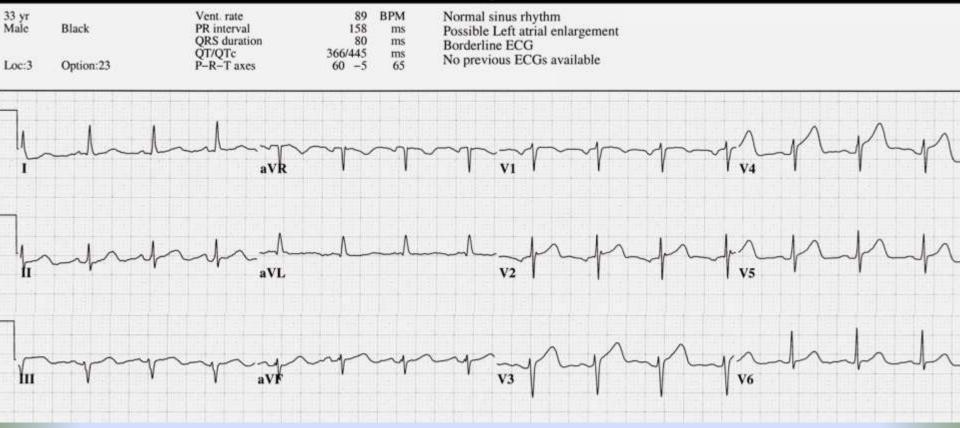
Wellen's Syndrome EPIDEMIOLOGY & PROGNOSIS:

- 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

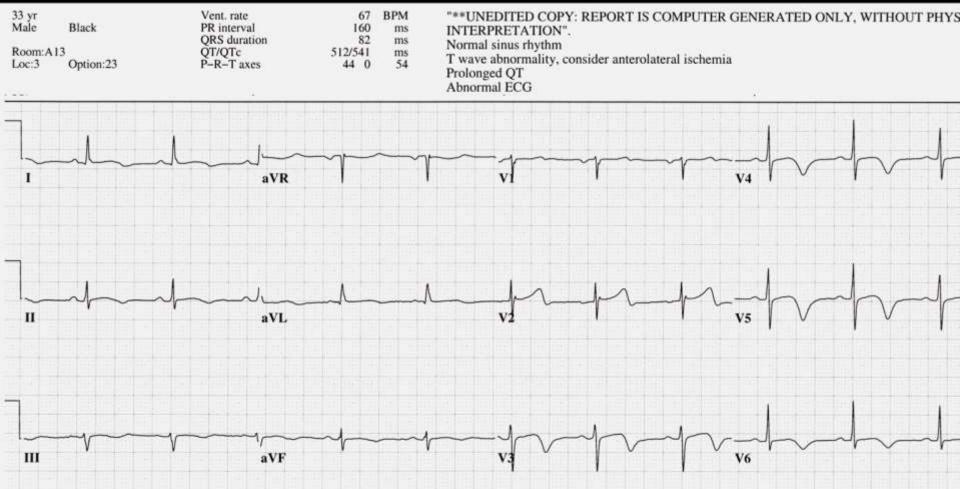
Sources: <u>H Wellens et. Al, Am Heart J 1982;</u> v103(4) 730-736

- 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.

SERIAL EKG CASE STUDY 1 - EKG #1 @ 06:22 HOURS



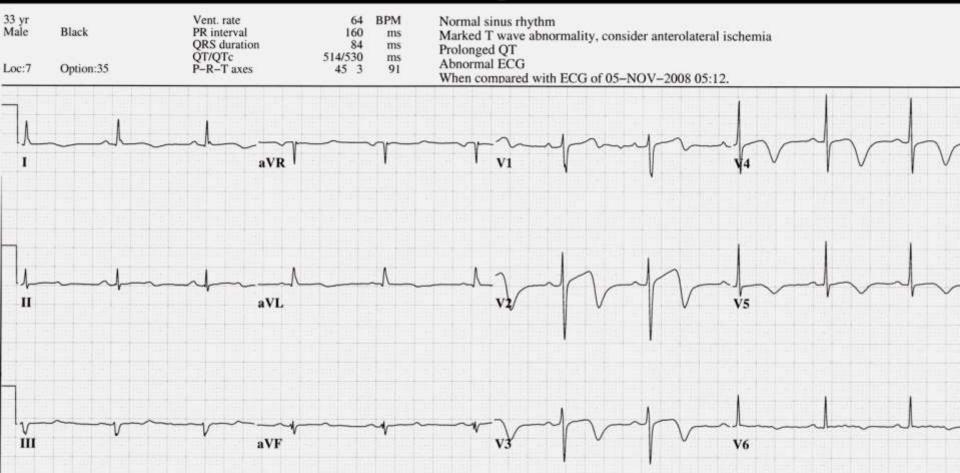
SERIAL EKG CASE STUDY 1 - EKG #2 @ 09:42 HOURS



DYNAMIC ST-T Wave Changes ARE PRESENT !!

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

SERIAL EKG CASE STUDY 1 - EKG # 3 @ 12:12 HOURS



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

600/652

20 1

71

144

74

BPM

ms

ms

ms

160

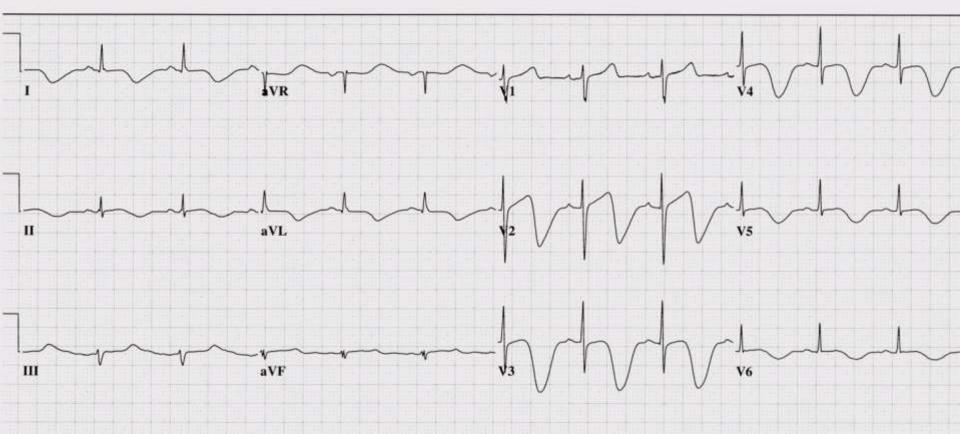


Vent. rate PR interval QRS duration QT/QTc P-R-T axes Normal sinus rhythm Marked T wave abnorm

Marked T wave abnormality, consider anterolateral ischemia

Prolonged QT

Abnormal ECG



SUB-TOTAL OCCLUSION OF LEFT ANTERIOR DESCENDING ARTERY

STENT DEPLOYMENT, LEFT ANTERIOR DESCENDING ARTERY, 33 y/o male

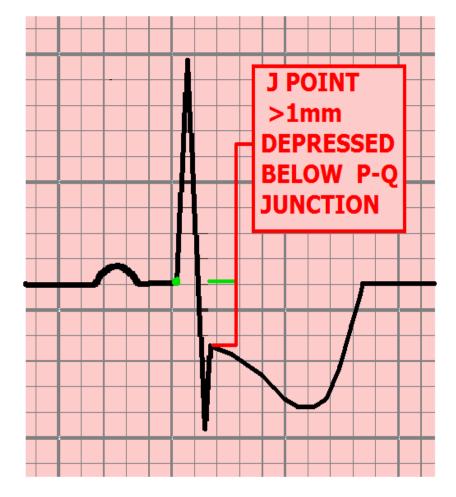
SUB-TOTAL OCCLUSION OF LEFT ANTERIOR DESCENDING ARTERY

POST PCI -LAD

Additional Resources:

Wellen's Syndrome, NEJM case study

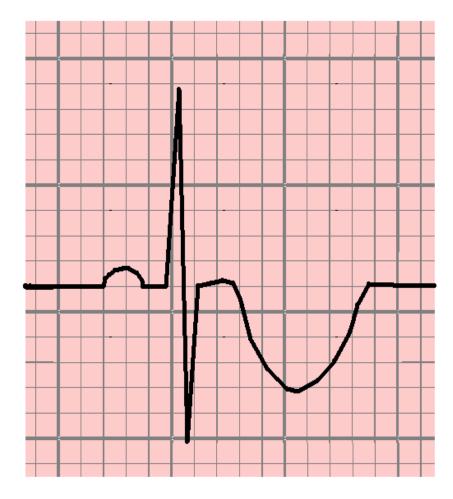
S-T SEGMENT DEPRESSION - COMMON ETIOLOGIES:



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

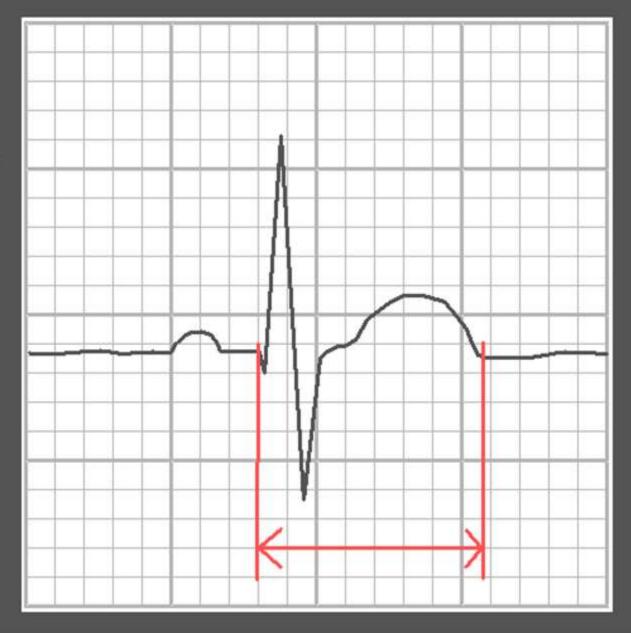
Some less common, less reliable possible indicators of ACS:

?	FLAT S-T SEGMENT > 120 ms	$\sim \downarrow \sim$	- ISCHEMIA
?	LOW VOLTAGE T WAVE WITH NORMAL QRS	~	- ISCHEMIA
?	U WAVE POLARITY OPPOSITE THAT OF T WAVE	$\downarrow \sim$	- ISCHEMIA

Long QT Syndrome

Q - T INTERVAL

- VARIES BASED ON HEART RATE AND SEX



THE *QTC INTERVAL

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

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.

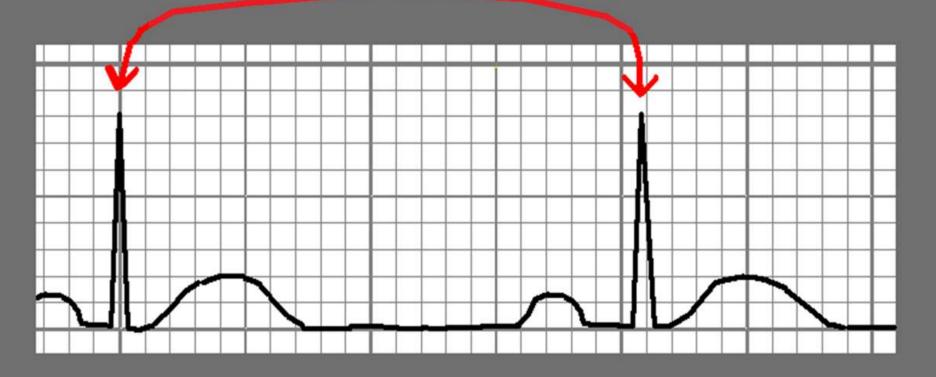
Determining the QTc Manual calculation:

QT CORRECTION FORMULAS:

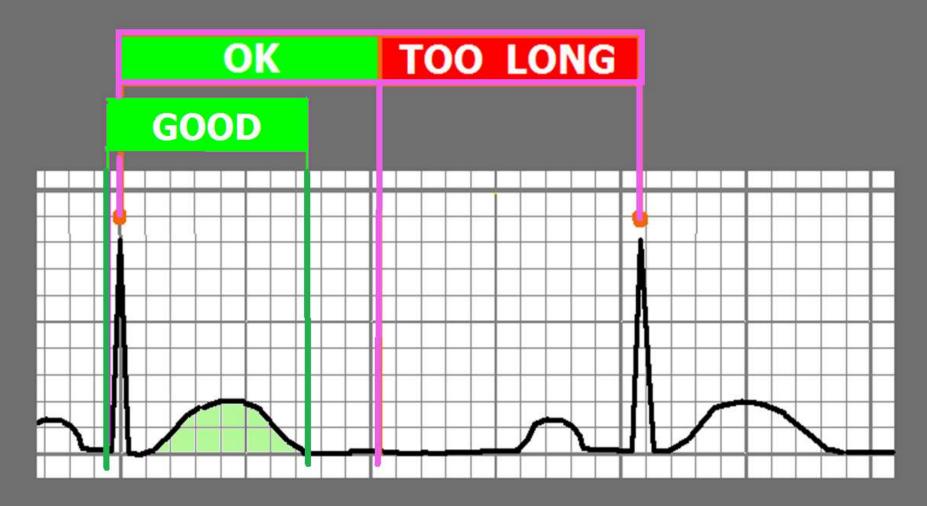
Bazett's Fredericia Framingham Rautaharju QTc=QT/ \sqrt{RR} QTc=QT/(RR)1/3 QTc=QT+0.154(1-RR) QTp=656/(1+HR/100)

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

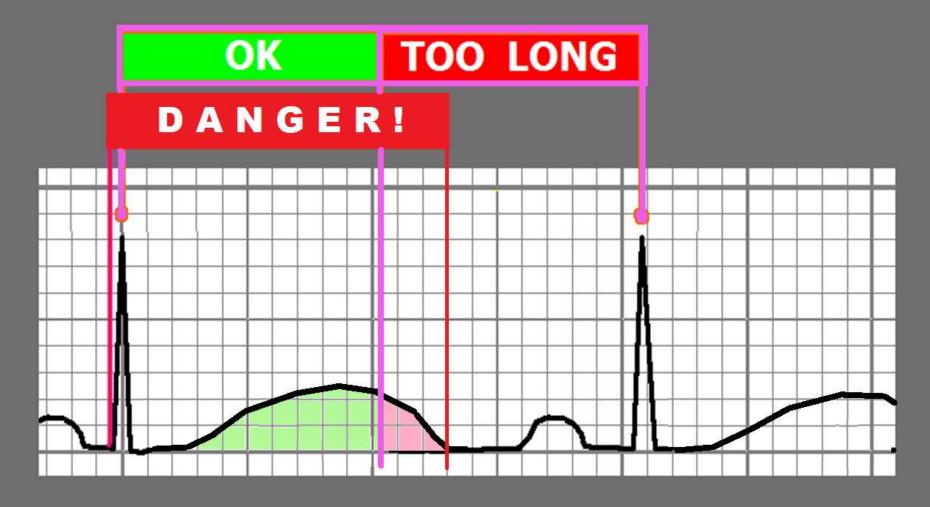
Relatively accurate method to quickly identify patients with abnormal QT Intervals.
 Applies to patients with normal heart rates (60-100) and narrow QRS (QRSd <120ms)



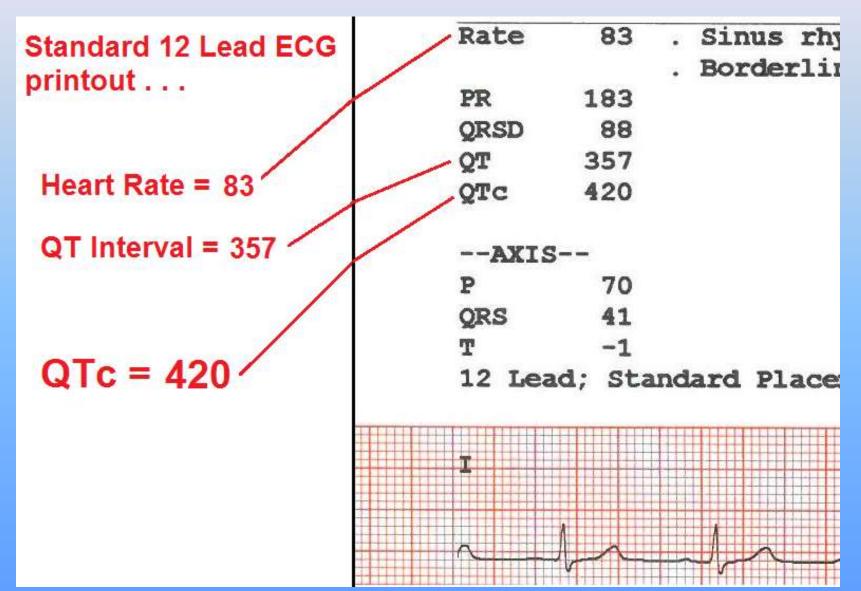
The Q - T Interval should be LESS THAN 1/2 the R - R Interval



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

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



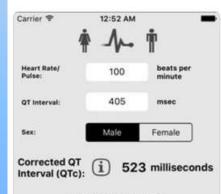
Corrected QT Interval (QTc) 17+

Daniel Juergens

\$0.99

"There's an APP for that!"

iPhone Screenshots



Abnormal QTc

1	2 ^**	3 DEF
<u>4</u>	5	6
вні	JKL	MNO
7	8	9
PORS	TUV	wxyz
	0	8

Carrier 😤 12:52 AM

< Back

Like the R-R interval, the QT interval is dependent on the heart rate and may be adjusted to improve the detection of patients at increased risk of ventricular arrhythmia. The standard clinical correction is the Bazett's formula, which is used in this app. For risk of sudden cardiac death, "borderline QTc" in males is 431-450 ms, and in females 451-470 ms, An "abnormal" QTc in males is a QTc above 450 ms, and in females, above 470 ms.



QT_interval (20.08.2016)



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:

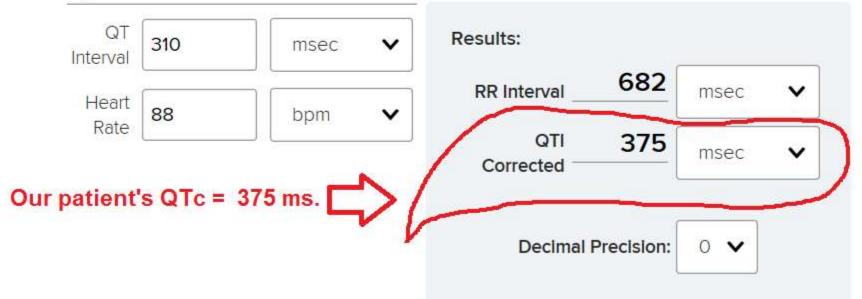


Calculators > Heart and Chest, Critical Care

QT Interval Correction (EKG)

< Share

Input:



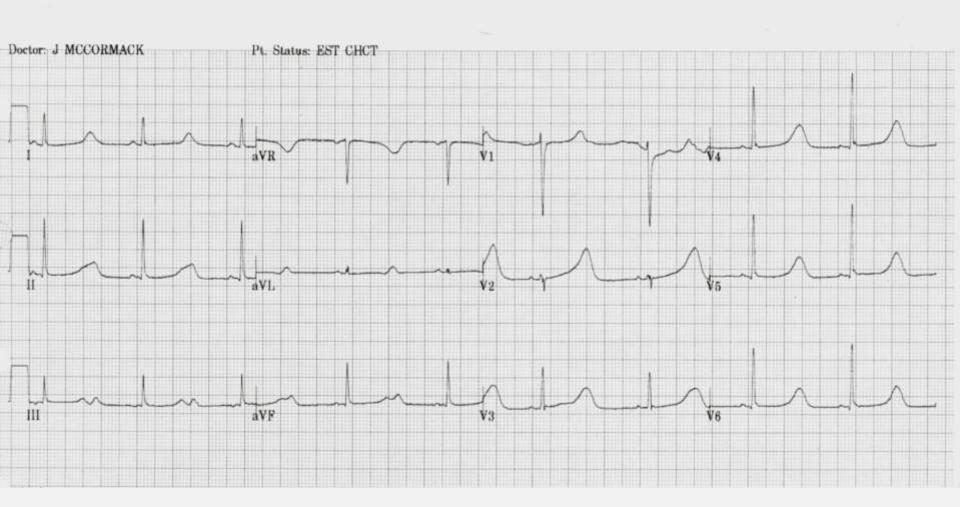
QTc Values:

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

22 y/o FEMALE

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



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:

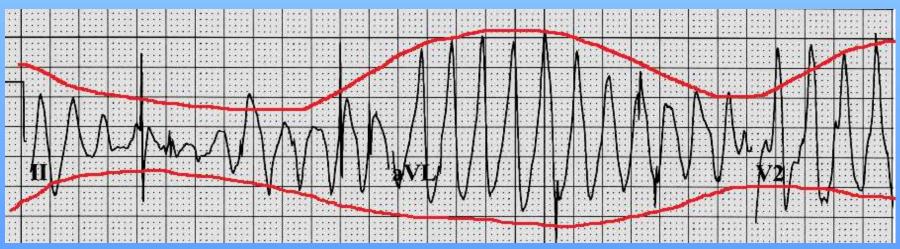
- Decreased to NO Cardiac Output
- Often patient PULSELESS during episode
- Patients often report SYNCOPE when TdP self-terminates.
- May DETERIORATE into VENTRICULAR FIBRILLATION and CARDIAC ARREST. ("Sudden Death")

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

resembles



a piece of Twisted Ribbon !



Evolving MI & "Old MI"

• Q WAVES •



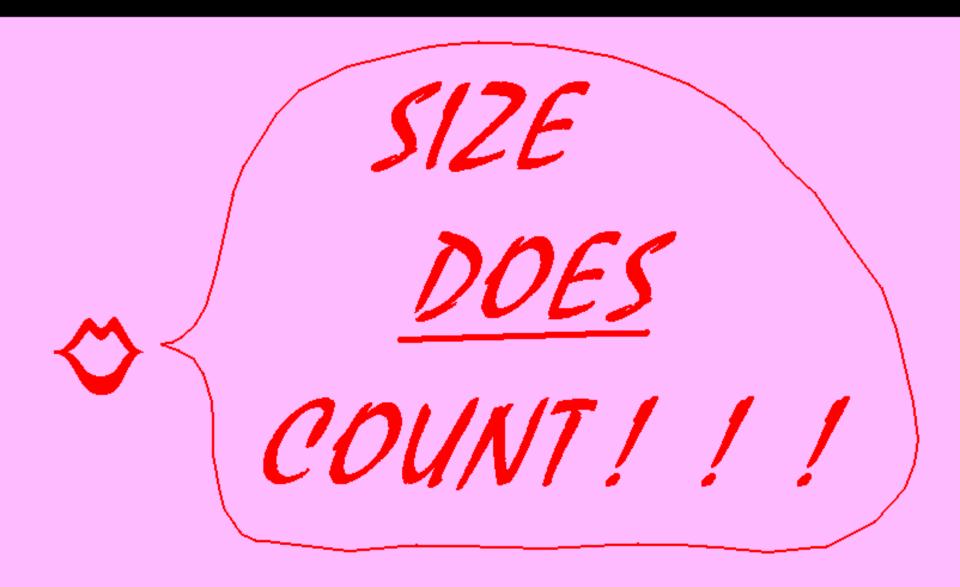
caused by depolarization of the intraventricular septum



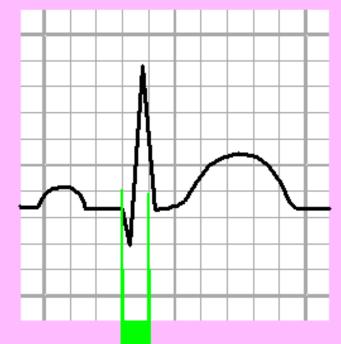
caused by:

- necrosis (old infarction)
- hypertrophy

• Q WAVES •

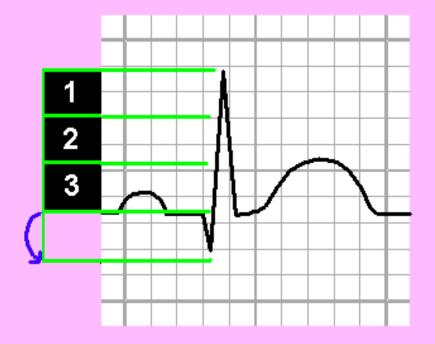


GENERAL RULES FOR NORMAL Q WAVES - WIDTH



LESS THAN .40 (1 mm) WIDE

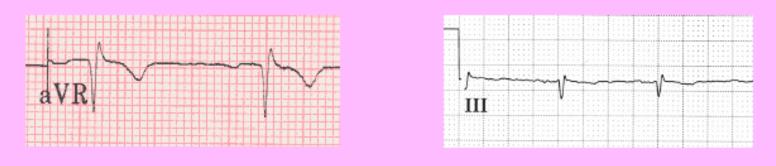
GENERAL RULES FOR NORMAL Q WAVES - HEIGHT





LESS THAN 1/3 THE HEIGHT OF THE R WAVE

NORMAL Q WAVES EXCEPTIONS TO THE RULES

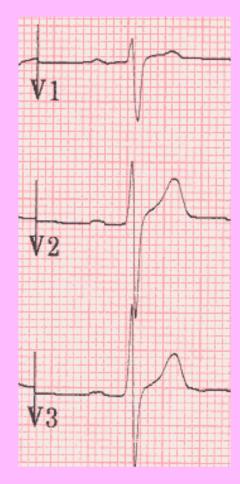


LEAD AVR LEAD III

THE Q WAVE CAN BE ANY SIZE

NORMAL Q WAVES EXCEPTIONS TO THE RULES

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



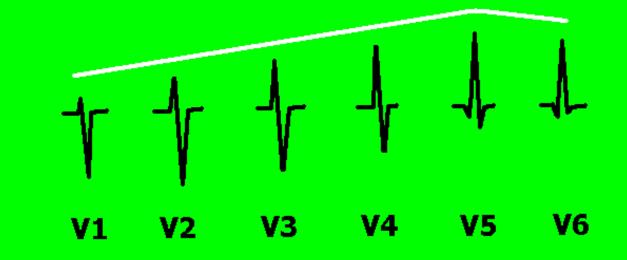
Q WAVE RULES - SUMMARY:

- 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

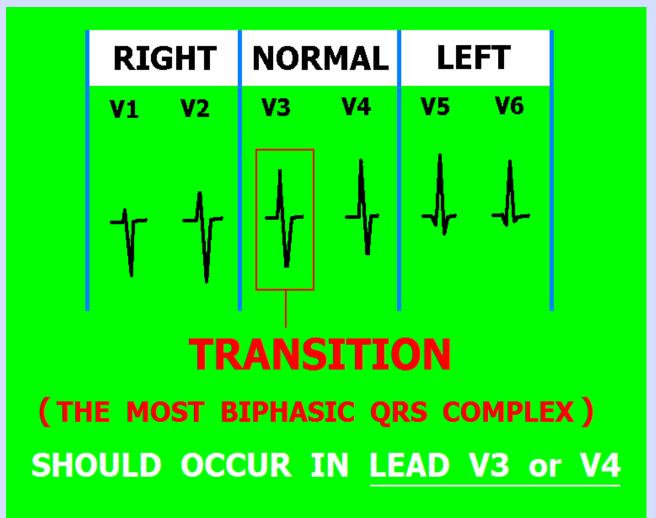
ASSESSING AXIS ROTATION

NORMAL R - WAVE PROGRESSION



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

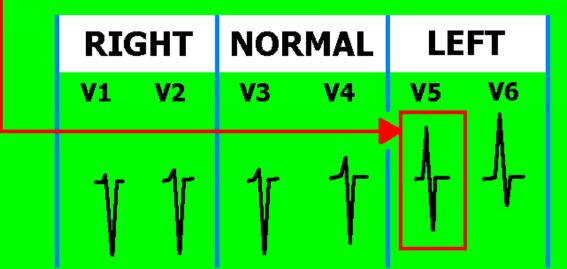
The NORMAL ECG



In V3 or V4, the QRS complex becomes Biphasic.

"Poor R Wave Progression"

LATE TRANSITION - COMMON CAUSES



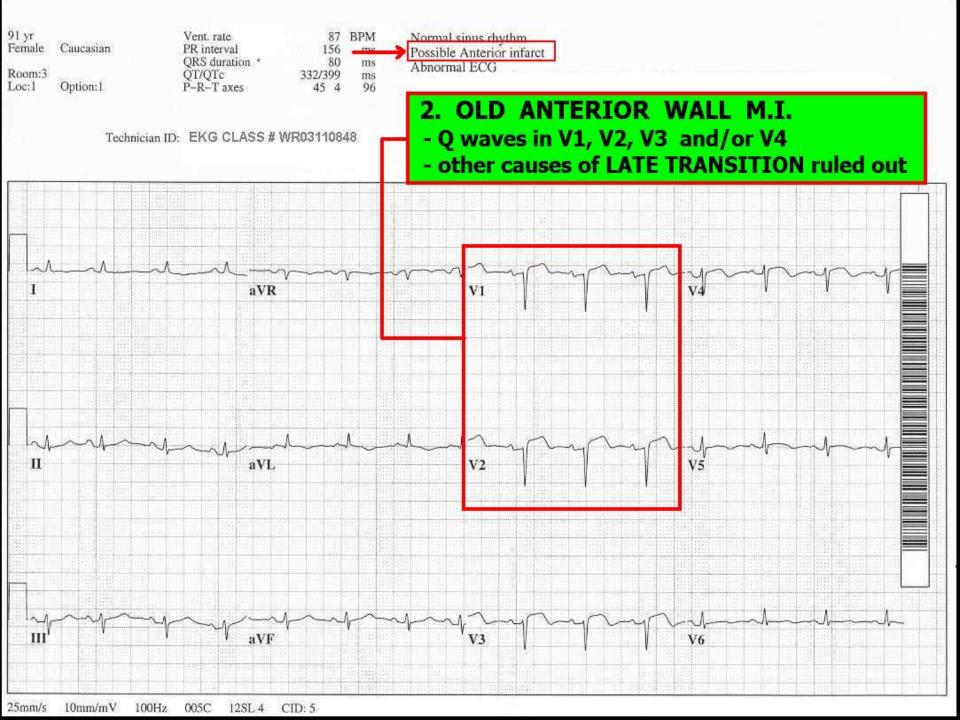
- LEFT BUNDLE BRANCH BLOCK
- OLD ANTERIOR WALL M.I.
- LEFT VENTRICULAR HYPERTROPHY
- WOLFF-PARKINSON-WHITE SYNDROME (R. ATRIUM - R. VENTRICLE BYPASS TRACT)

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

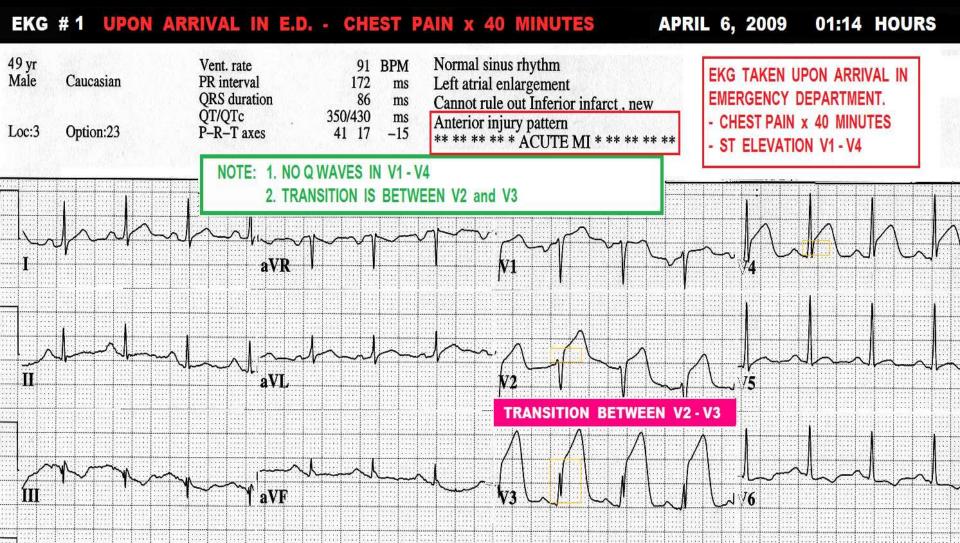
EVOLVING STEMI: -ST SEGMENTS DROP -Q WAVES FORM -R WAVE PROGRESSION CHANGES IN PRECORDIAL Q WAVE RULES - SUMMARY: LEADS. - Q WAVES SHOULD BE LESS THAN .40 WIDE (1 mm)

- 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

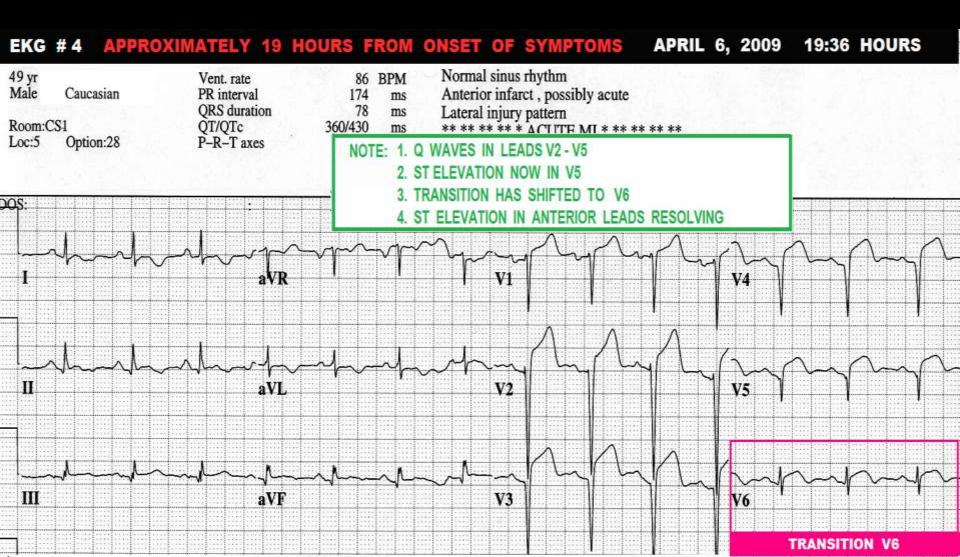
EVOLVING STEMI: -ST SEGMENTS DROP -Q WAVES FORM -R WAVE PROGRESSION CHANGES **IN PRECORDIAL** LEADS.



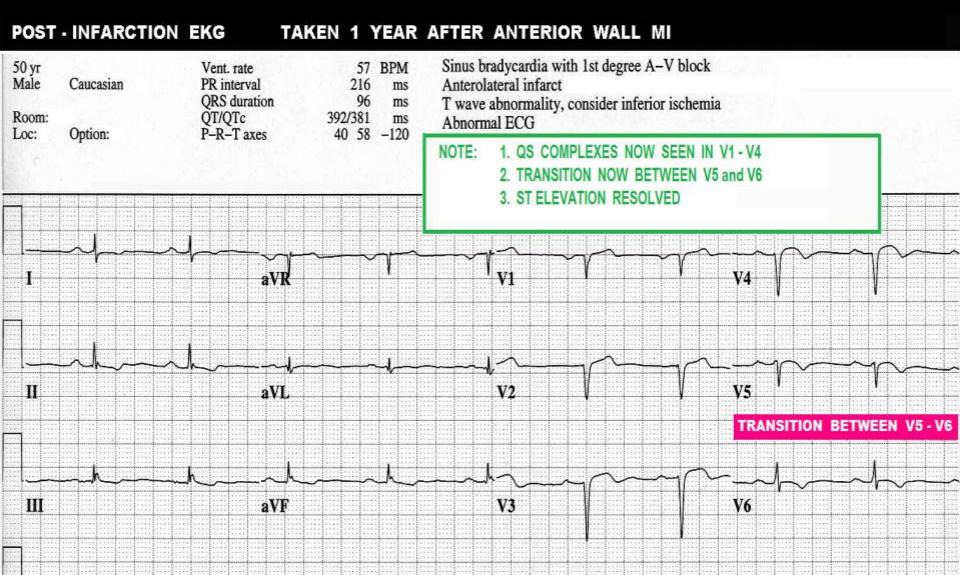
ACUTE ANTERIOR WALL STEMI



EVOLVING ANTERIOR WALL STEMI



FULLY EVOLVED ANTERIOR WALL MI



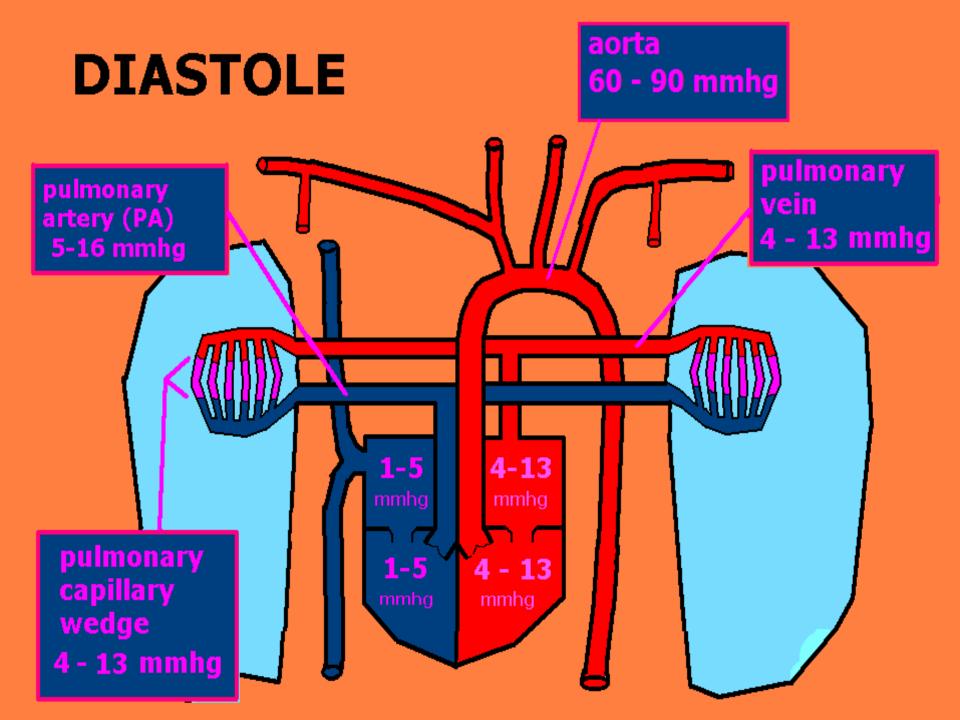
Additional Materials:

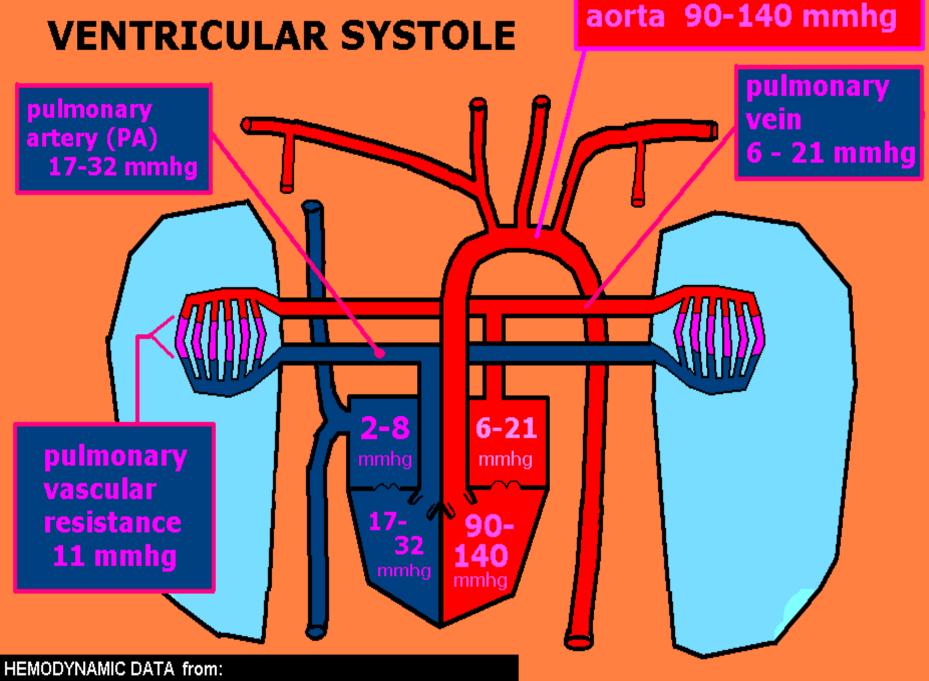


ABNORMAL EKG CHANGES THAT MAY PRESENT WITH ABNORMAL HEART SOUNDS:

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



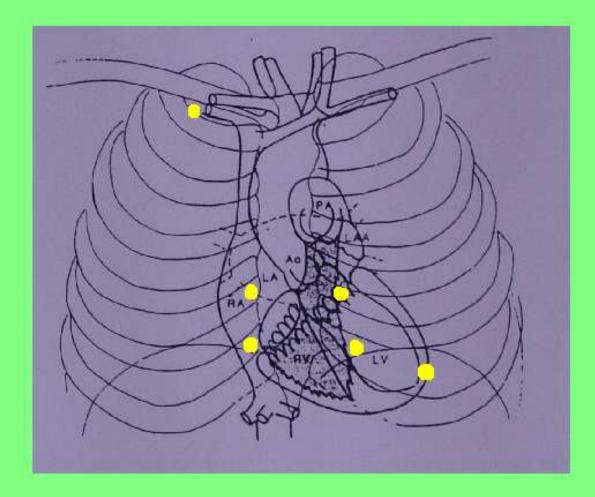




"The Cardiac Catheterization Handbook,"

Morton J. Kearn, MD







- NormalHeartSounds
- Murmurs
 - systolic
 - diastolic
- Friction Rubs



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

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

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

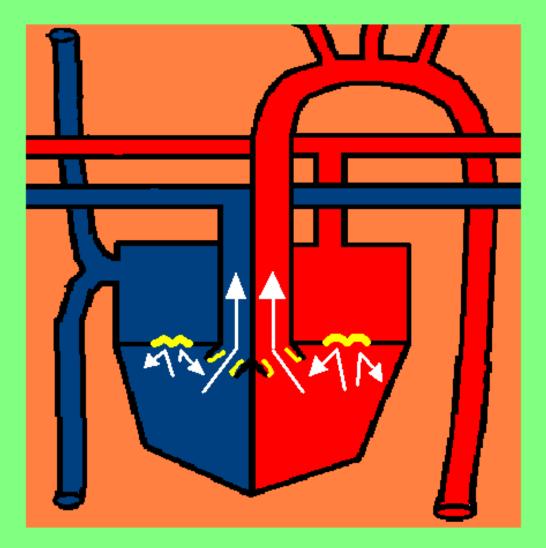
WE OFTEN DESCRIBE THESE HEART SOUNDS AS "LUB - DUP"



ABNORMAL SOUND	SUSPECTED EKG CHANGES
MURMURS	- ACUTE MI
- SYSTOLIC	- CHAMBER HYPERTROPHY
- DIASTOLIC	- NECROSIS - RECENT
	EXTNSIVE MI (7-10 days)
FRICTION RUB	- ACUTE MI
	- RECENT MI (NECROSIS)
	- PERICARDITIS

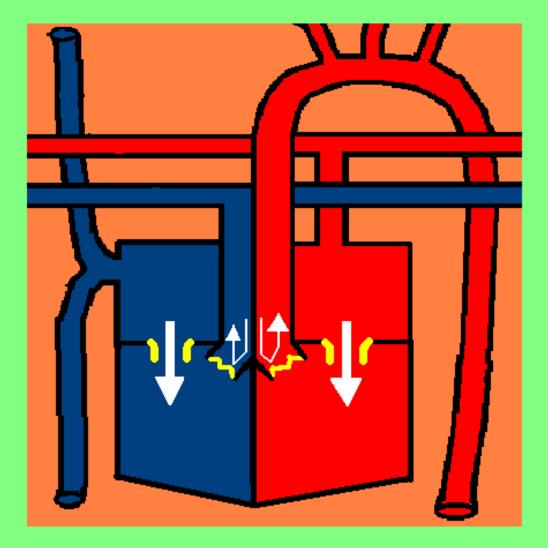
S-1 BEGINNING OF SYSTOLE.

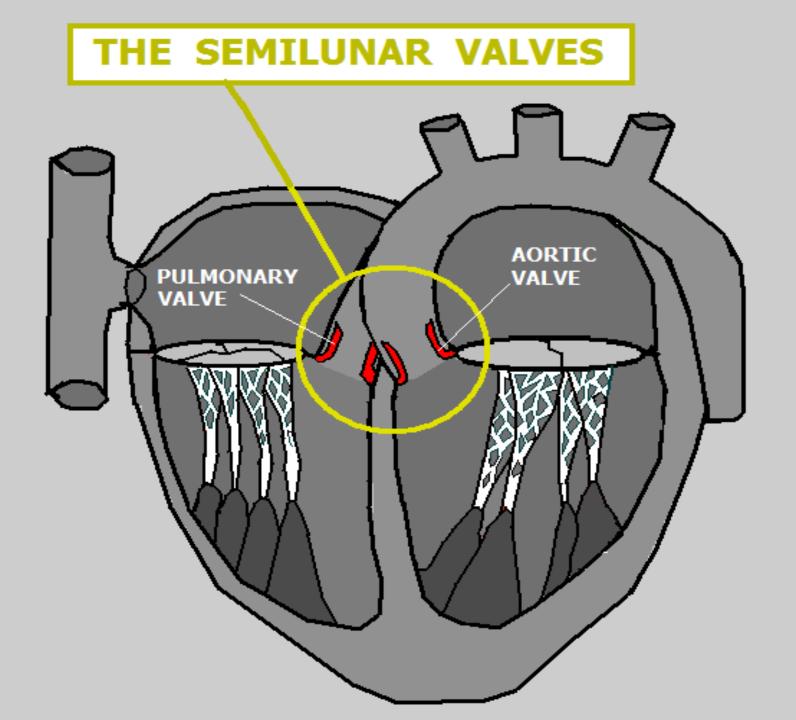
SOUND OF THE MITRAL AND TRICUSPID VALVES CLOSING.



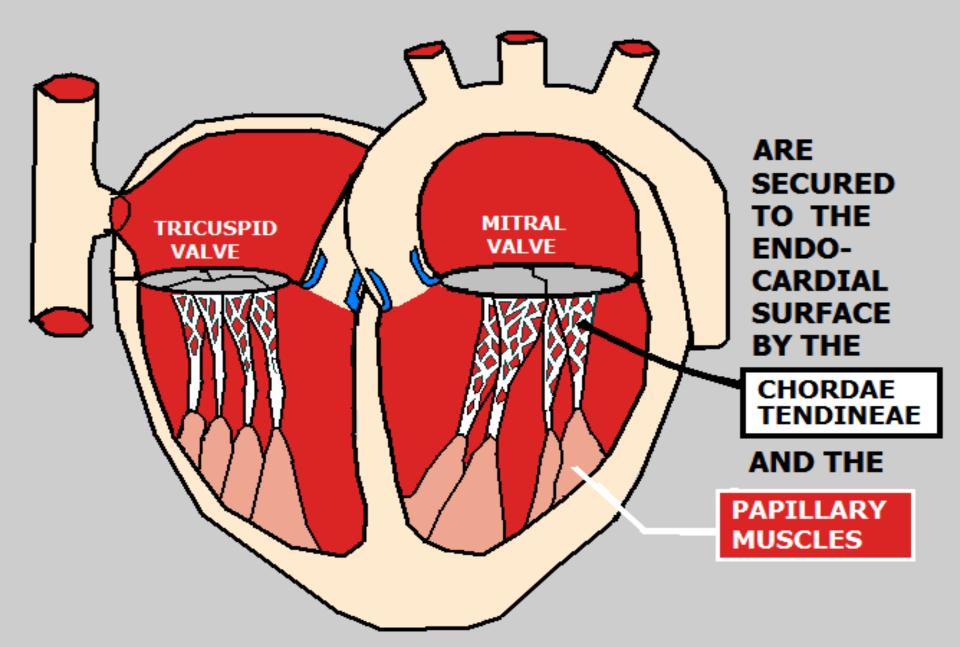
S-2 OCCURS AT THE END OF SYSTOLE (THE BEGINNING OF DIASTOLE).

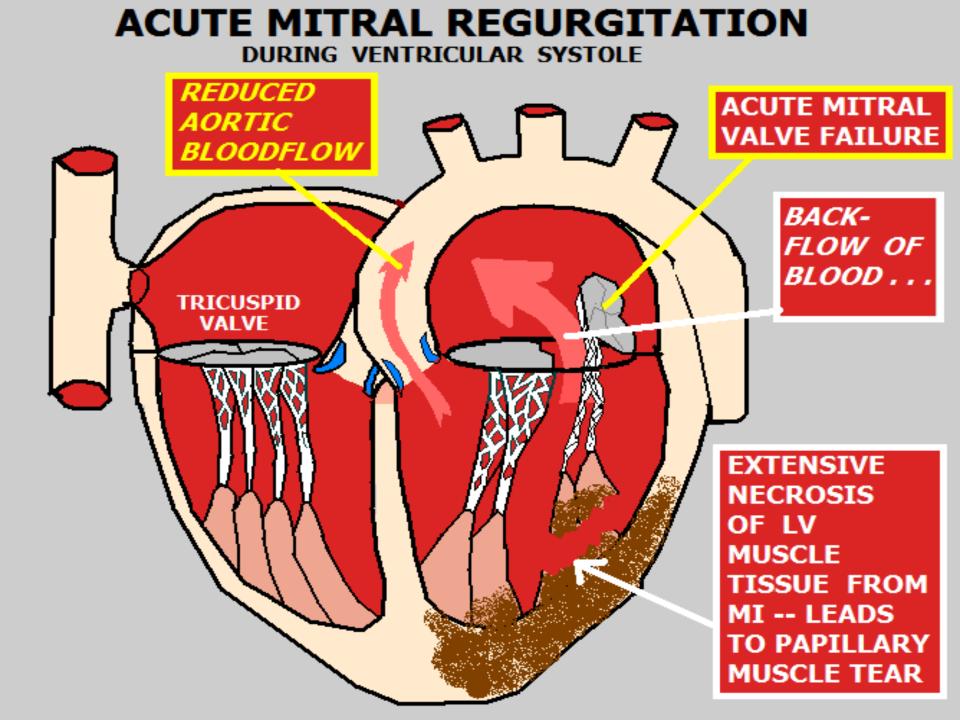
IT IS THE SOUND OF THE AORTIC AND PULMONARY VALVES CLOSING.





ATRIO-VENTRICULAR VALVES





BASIC HEART SOUNDS ASSESSMENT

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

S-1 MURMUR SOUNDS LIKE:

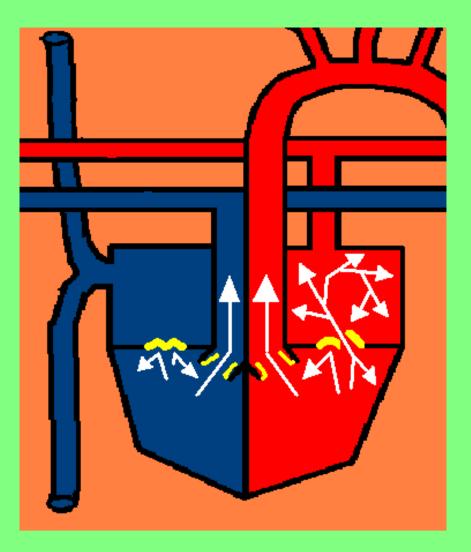


"SWOOSH-DUB . . . SWOOSH-DUB . . . SWOOSH-DUB SWOOSH-DUB . . . "

CAUSE OF SYSTOLIC (S 1) MURMUR

DAMAGE TO MITRAL and/or TRICUSPID VALVE(s)

CAUSES REGURGITATION



MOST SYSTOLIC MURMURS CAUSED BY MITRAL VALVE FAILURE.

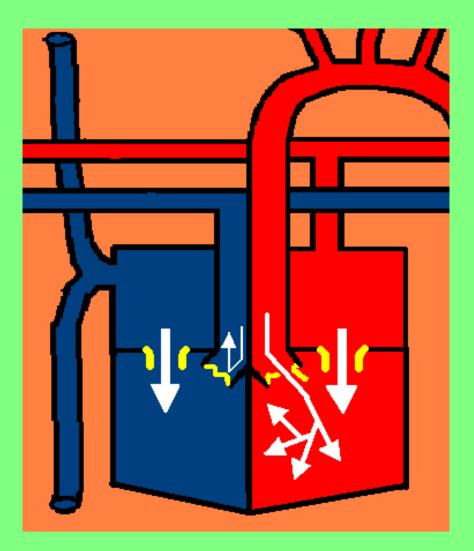
 ACUTE MITRAL VALVE REGURGITATION IS A POTENTIALLY LETHAL COMPLICATION OF ACUTE / RECENT EXTENSIVE TRANSMURAL MI

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

CAUSE OF DIASTOLIC (S2) MURMUR

DAMAGE TO
 AORTIC and/or
 PULMONIC
 VALVE(s)

CAUSES REGURGITATION



BASIC HEART SOUNDS ASSESSMENT

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



S-2 MURMUR SOUNDS LIKE:

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

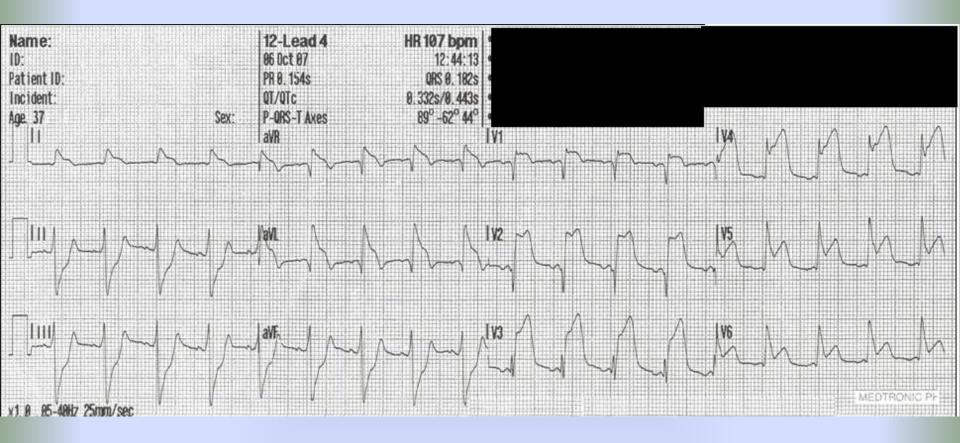
IS PRESENT IN MOST ACUTE TRANSMURAL MI PATIENTS



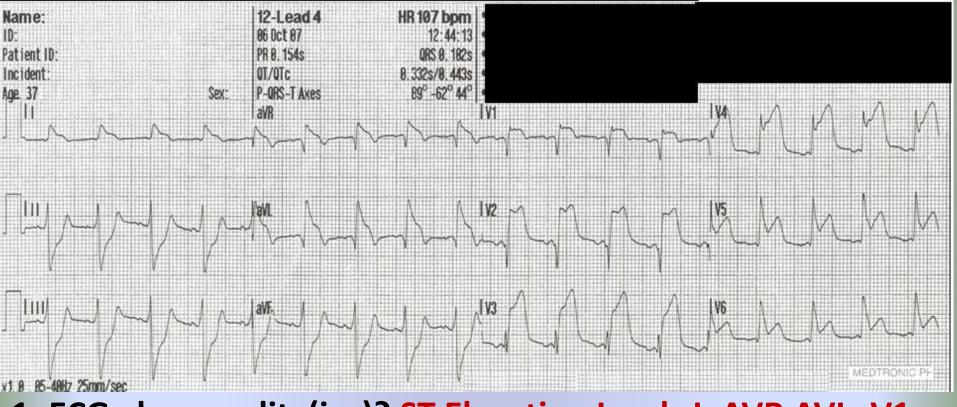
- MAY BE PRESENT WITHIN HOURS AFTER ONSET
- IS TRANSIENT -- MAY LAST FOR A FEW DAYS

Practice ECGs . . .

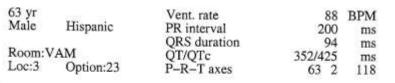
Let's review

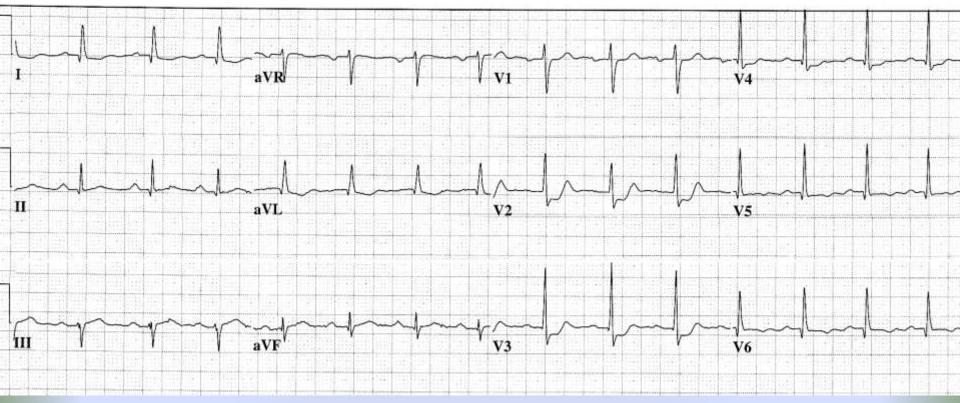


- 1. ECG abnormality(ies)?
- 2. Possible diagnosis?
- **3.** Action / Intervention?

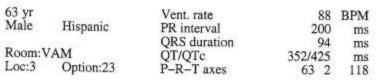


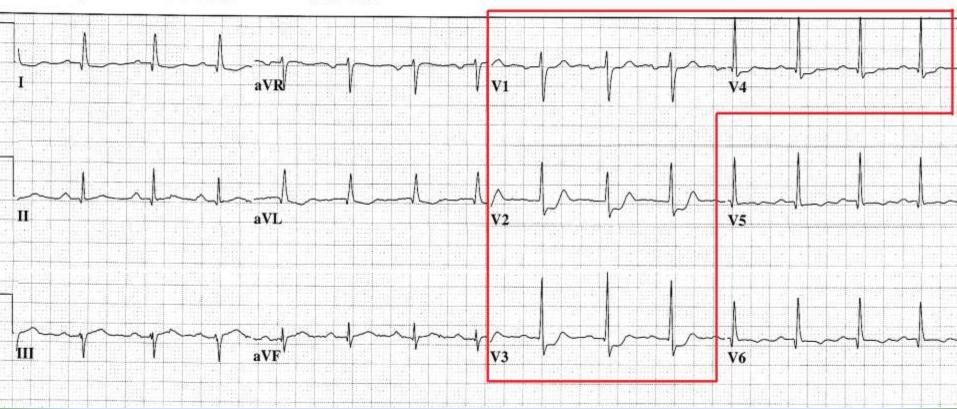
- ECG abnormality(ies)? ST Elevation Leads I, AVR AVL, V1, V2, V3, V4, V5 & V6. ST Depression II, III and AVF
 Possible diagnosis? Acute Anterolateral Wall STEMI secondary to Left Main Coronary Artery occlusion (widowmaker MI).
- 3. Action / Intervention? STAT CATH LAB vs STAT Thrombolytics. Prepare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



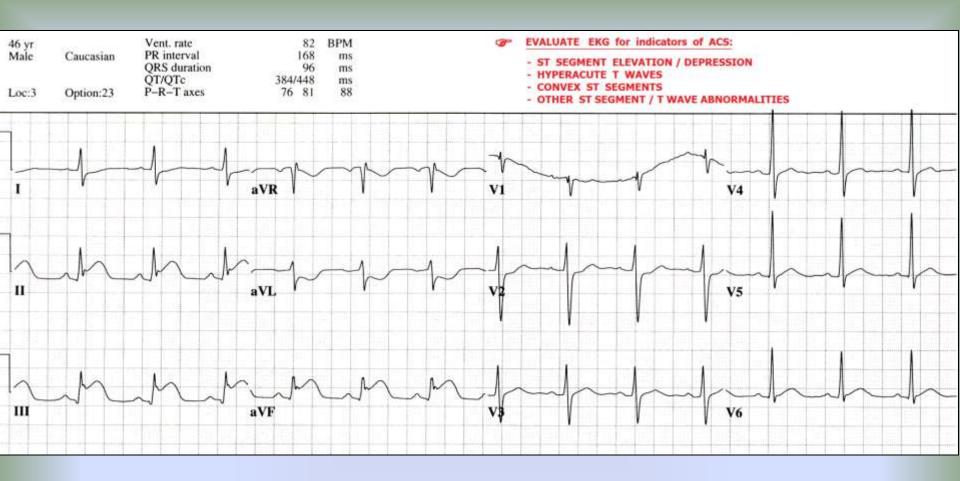


- 1. ECG abnormality(ies)?
- 2. Possible diagnosis?
- **3.** Action / Intervention?

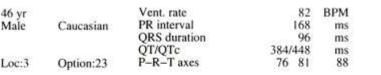




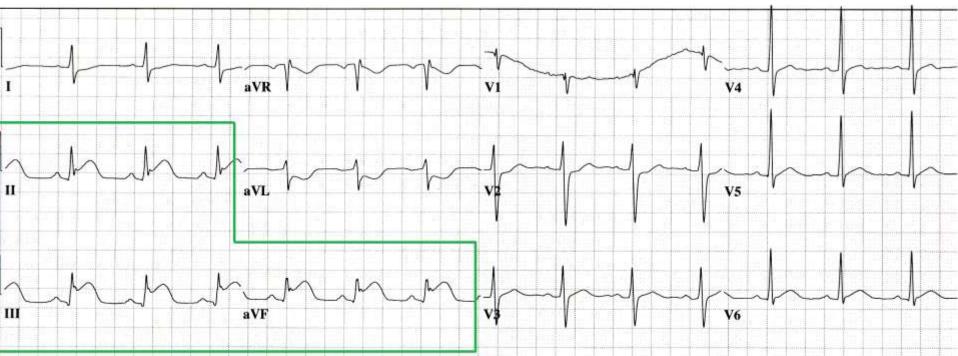
- ECG abnormality(ies)? ST Depression V1-V4
 Possible diagnosis? Anterior ischemia vs. Posterior wall STEMI
- 3. Action / Intervention? Posterior ECG (V7-V9)



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

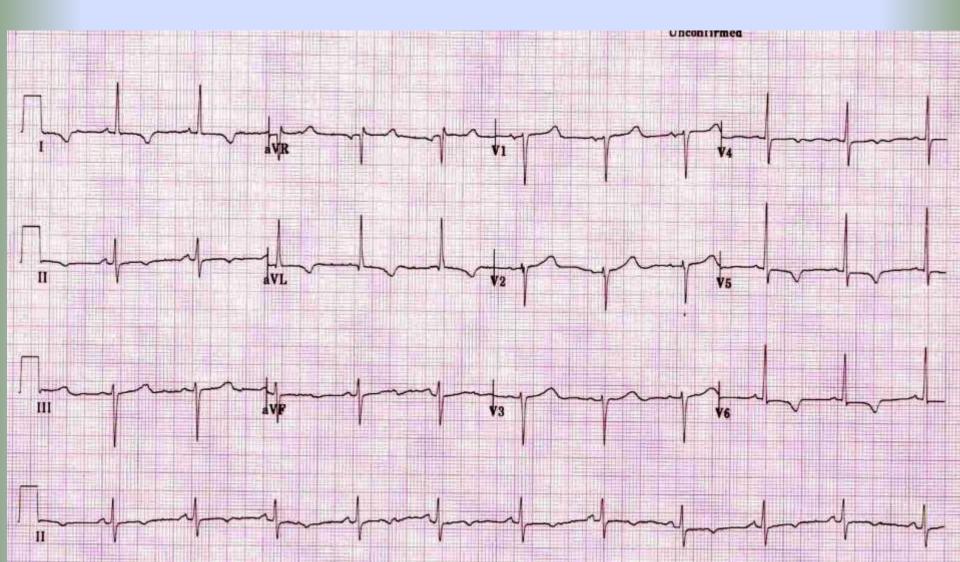


ST-Segment Elevation in Leads II, III and AVF Consistent with: INFERIOR STEMI

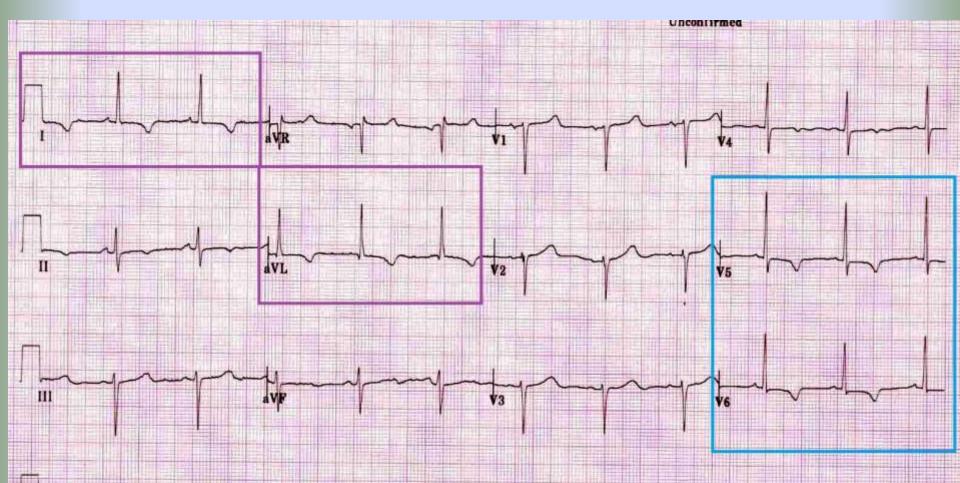


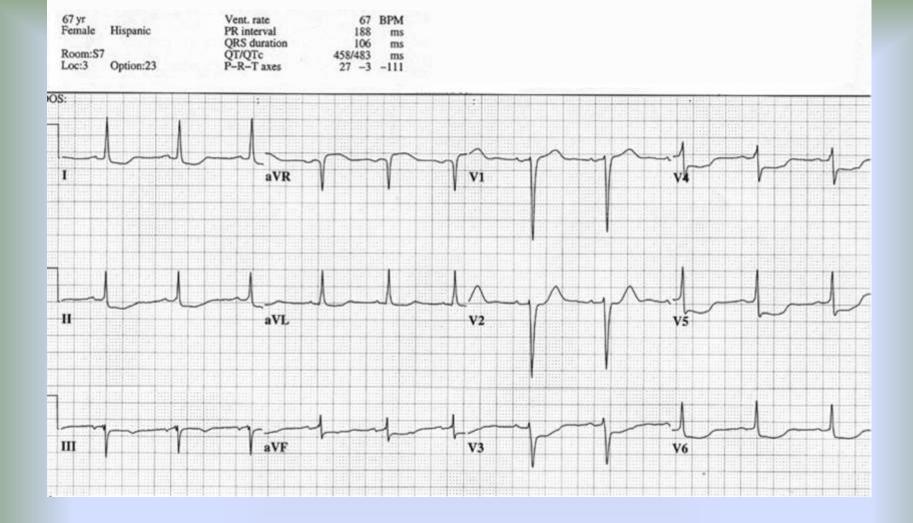
ECG abnormality(ies)? ST Elevation, Leads II,III & AVF
 Possible diagnosis? Inferior Wall STEMI
 Action / Intervention? 1. Do R-sided ECG, prepare for
 Atropine administration, external pacing, cardiac arrest,
 STAT cath lab visit !

What leads show signs of possible ACS?

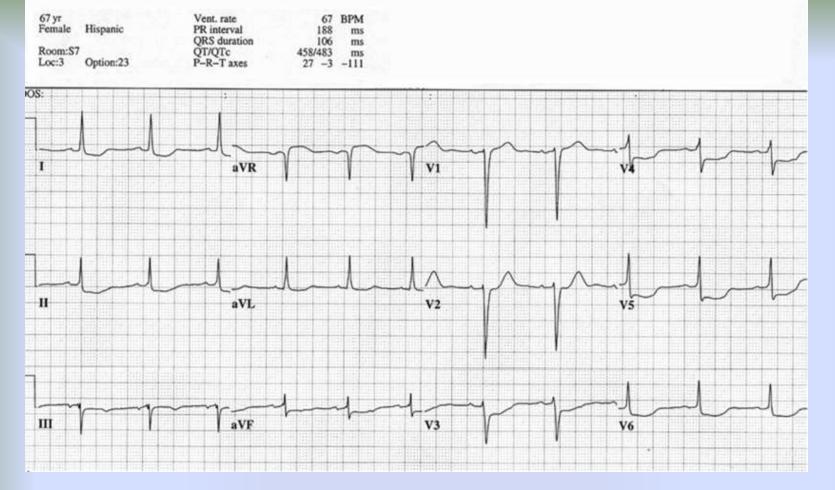


12 Lead ECG shows ISCHEMIC CHANGES Lateral Wall:

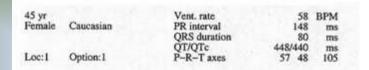


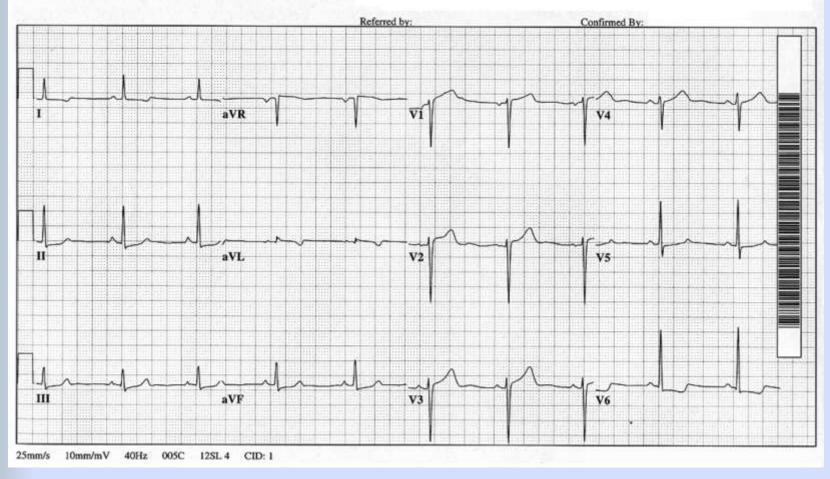


- 1. ECG abnormality(ies)?
- 2. Possible diagnosis?
- **3.** Action / Intervention?

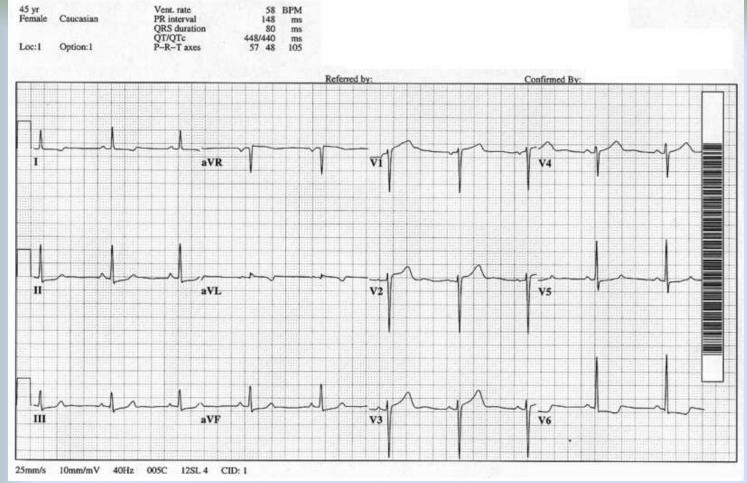


 ECG abnormality(ies)? ST Elevation Lead AVR, Global ST Depression (I, II, III, AVL, AVF, V2, V3, V4, V5, V6)
 Possible diagnosis? possible LMCA or 3x vessel disease.
 Action / Intervention? Troponins, Continuous ST monitoring, cath lab visit STAT or ASAP (based on sympt.)





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



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

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

Author's correspondence information:

Wayne W Ruppert

Wayneruppert@bayfronthealth.com

Office: 352-795-8558

Cell: 813-230-4747



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