12 LEAD ECG INTERPRETATION in

ACUTE CORONARY SYNDROME

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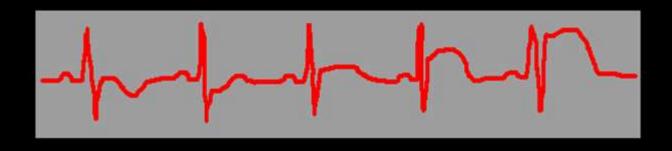
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PROGRAM CONTENTS SESSION TWO

THE ACUTE CORONARY SYNDROMES



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



IDENTIFY and MANAGE ACUTE CORONARY SYNDROMES:



NON-STEMI

UNSTABLE ANGINA

CP-LOW RISK

PROBLEM: Each year in the United States, it is estimated between 68,000 and 136,000 people with ACS are *misdiagnosed* and *discharged* from our nation's emergency departments, resulting in needless *morbidity* and *mortality*. The emotional loss to grieving family members is incalculable. The negative financial impact to families who lose their primary income providers, as well as losses incurred by health care providers in legal expenses is also significant.

"The ACS Scorecard"

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!

PATIENT EVALUATION

- INITIAL APPROACH (SHOCK SURVEY)
- ABCs
- CHIEF COMPLAINT
- SECONDARY EVALUATION
 - RAPID, FOCUSED ASSESSMENT
 - PAIN / PRESSURE / BREATHING / SYMPTOMS ?

SHOCK ASSESSMENT



SHOCK =

INADEQUTE TISSUE
PERFUSION

- STARTS THE INSTANT YOU SEE PATIENT
- ENDS WHEN YOU REACH THE PATIENT'S SIDE

SHOCK ASSESSMENT

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

NORMAL

STATUS: 6 SHOCK 6

FAIL the SHOCK SURVEY?

THE ROOT CAUSE...

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

PASSED SHOCK SURVEY:

. Move on to "RULING OUT ACS" By conducting the . . .

INITIAL EVALUATION:

- 1. ASSESSMENT
- 2. RISK STRATIFICATION
- 3. ECG
- 4. CARDIAC MARKERS

ON-GOING E.D. EVALUATION

- -REPEAT EKGs
- -REPEAT CARDIAC MARKERS

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RULE OUT ACUTE CORONARY SYNDROME PHASE 2: PERFORM RAPID, TARGETED ASSESSMENT. DOES **PATIENT COMPLAIN OF:** TYPICAL ACS SYMPTOMS ? 3 ATYPICAL ACS SYMPTOMS? AUSCULTATE LUNG and HEART SOUNDS 5 YES NO OBTAIN and EVALUATE CONDUCT APPROPRIATE 12 LEAD EKG DIAGNOSTIC WORK-UP 10 NON-11 8 ST 12 NON-ST DIAGNOSTIC Depression **ELEVATION** SPECIFIC ST NORMAL and/or T EKG: or T WAVE in 2 or more **EKG** WAVE OLD LBBB, changes that LEADS -- or inversion: **PACEMAKER** may indicate **OBTAIN 18** RHYTHM. NEW or **ISCHEMIA** LEAD EKG -WIDE QRS presumably ANY ST 9 w/ LBBB **NEW LBBB ELEVATION? PATTERN** YES NO IMPLEMENT 13A INSTITUTIONAL **ELEVATED** ACUTE MI CARDIAC **OBTAIN PROTOCOLS MARKERS?** and **EVALUATE** SERIAL EKGs REPEAT 13B CARDIAC **MARKERS** as IMPLEMENT per AHA or INSTITUTIONAL INSTITUTIONAL NSTEMI **PROTOCOLS PROTOCOLS** DETERMINE RISK STRATIFICATION SCORE CONSIDER ADDITIONAL DIAGNOSTIC TESTING, eg STRESS TESTING, ECHO, CARDIAC CATHETERIZATION **RULE OUT OTHER LETHAL CARDIAC CONDITIONS** PHASE 3:

RULE OUT LIFE-THREATENING CONDITIONS

PHASE 1:

"The ACS Scorecard"

$ \overline{\mathbf{V}} $	PRESENTING SYMPTOMS
	RISK FACTOR PROFILE
	ECG ABNORMALITIES
	CARDIAC MARKERS

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

CHIEF COMPLAINT

KEY WORDS:

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

SHORTNESS BREATH

DIZZINESS / LIGHTHEADEDNESS

ETC. ETC. ETC.



TYPICAL SYPTOMS of ACUTE CORNARY SYNDROME:

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

INFARCTION

- - - "Classic Symptoms" - - -



QUICK ASSESSMENT "SHORT FORM"

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

stable angina

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



unstable angina

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

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



ATYPICAL SYMPTOMS of ACS

???

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

Stroke (previous history of)

Heart failure (previous history of)

Race (non-white)

Elderly (age 75+)

Women

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) Fatigue

Indigestion Abdominal pain

Nausea Cold sweats

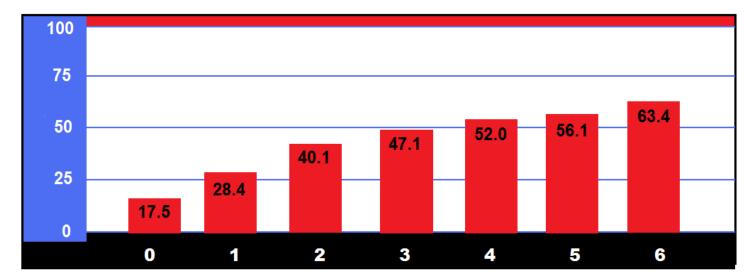
Dizziness Elevated heart rate

Syncope Dsypnea

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Effect of Having Multiple Risk Factors for AMI Without Chest Pain

% of PATIENTS with ACUTE MI PRESENTING TO THE EMERGENCY DEPARTMENT WITHOUT CHEST PAIN



NUMBER OF RISK FACTORS PRESENT

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

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

WOMEN'S MAJOR SYMPTOMS PRIOR TO THEIR HEART ATTACK:

۰	UNUSUAL FATIGUE	71 %
•	SLEEP DISTURBANCE	48 %
•	SOB	42 %
٠	INDIGESTION	39 %
۰	ANXIETY	36 %

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

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

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



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

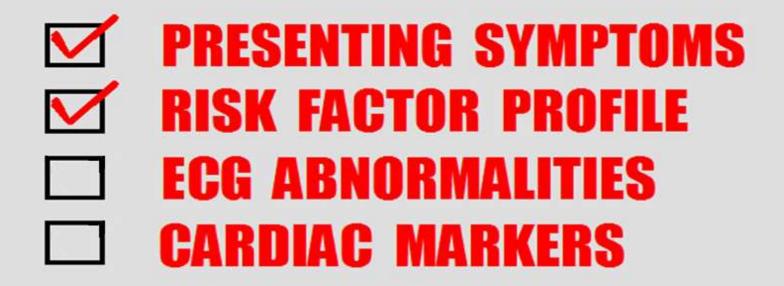
Circulation, 2003:108;2619-2623

"The ACS Scorecard"

$ \overline{\mathbf{V}} $	PRESENTING SYMPTOMS
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RISK FACTORS

for the development of

CORONARY ARTERY DISEASE:

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

CASE STUDY: IMPORTANCE of RISK FACTORS

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

62 y/o MALE presents to cardiologist's office with intermittent ACS symptoms (chest heaviness, dyspnea). - Pt. DOES NOT correlate symptoms with exertion.

RISK FACTOR PROFILE:

- FAMILY HISTORY both parents + CAD before age 65
- PREVIOUS CIGARETTE SMOKER 20+ yrs., quit 15 years ago
- ★ HIGH CHOLESTEROL Dx 5 yrs ago, taking STATIN med since.
- **DIABETES** Controlled with diet and oral meds.

PHYSICAL EXAM: Patient supine on exam table, skin warm, dry, color NL Patient is asymptomatic, all systems WNL

VITAL SIGNS: BP 153/88, P 80, R 16, SAO2 99%

DIAGNOSTIC TESTING: EKG NORMAL, EXERCISE STRESS TEST PASSED.

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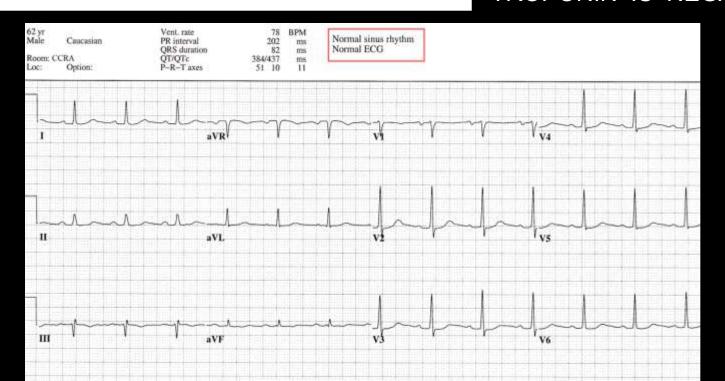
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TROPONIN IS NEGATIVE.



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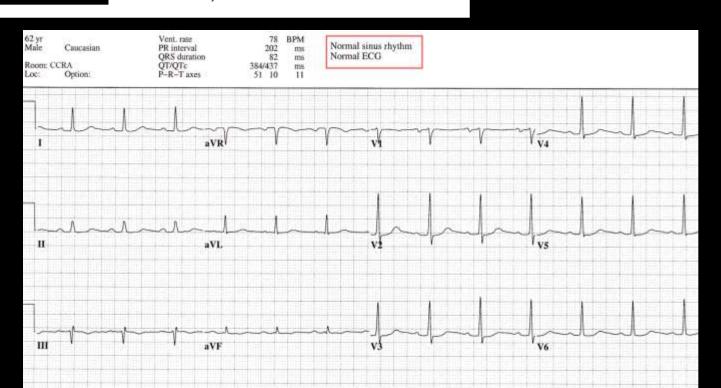
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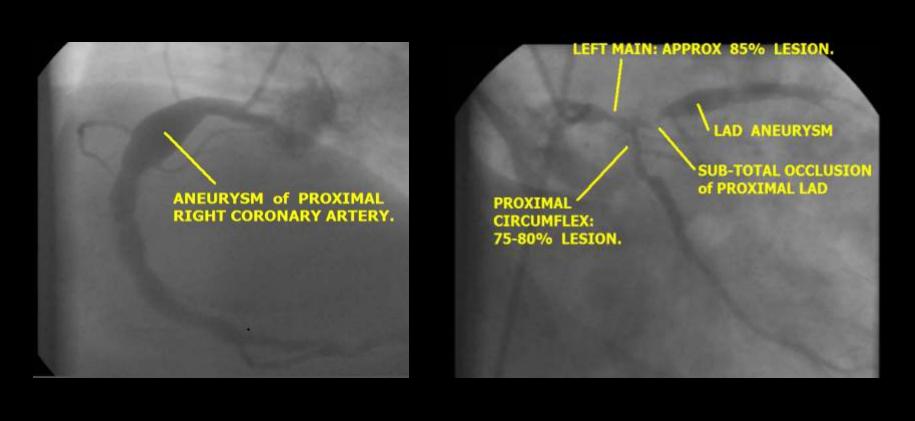
DIAGNOSTIC TESTING: EKG NORMAL, EXERCISE STRESS TEST PASSED.

"The ACS Scorecard"

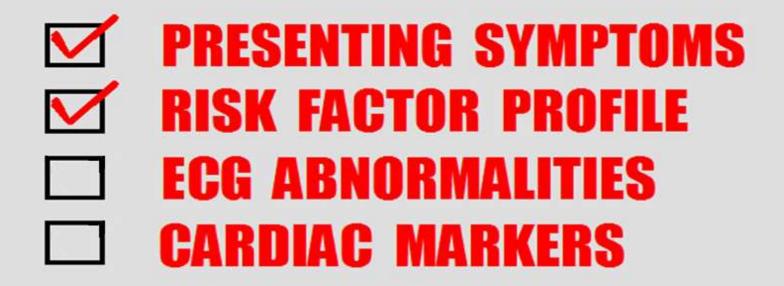
- ✓ PRESENTING SYMPTOMS
 ✓ RISK FACTOR PROFILE
 ☐ ECG ABNORMALITIES
- ☐ CARDIAC MARKERS

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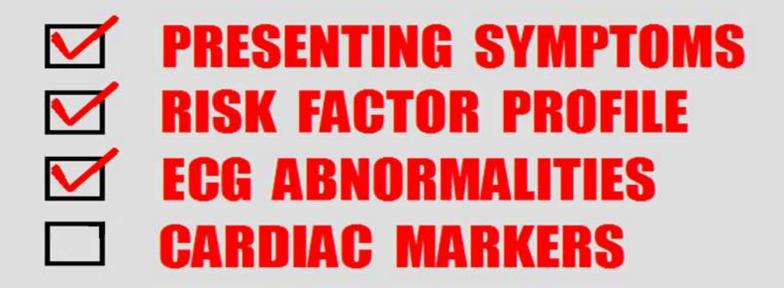


"The ACS Scorecard"



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ECG EVALUATION for ACS:

STEP 1: EVALUATE WIDTH of QRS

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IF THE QRS IS TOO WIDE.....

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( GREATER THAN 120 ms )
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. . . . IS the QRS morphology:

LEFT BUNDLE BRANCH BLOCK

- OR -

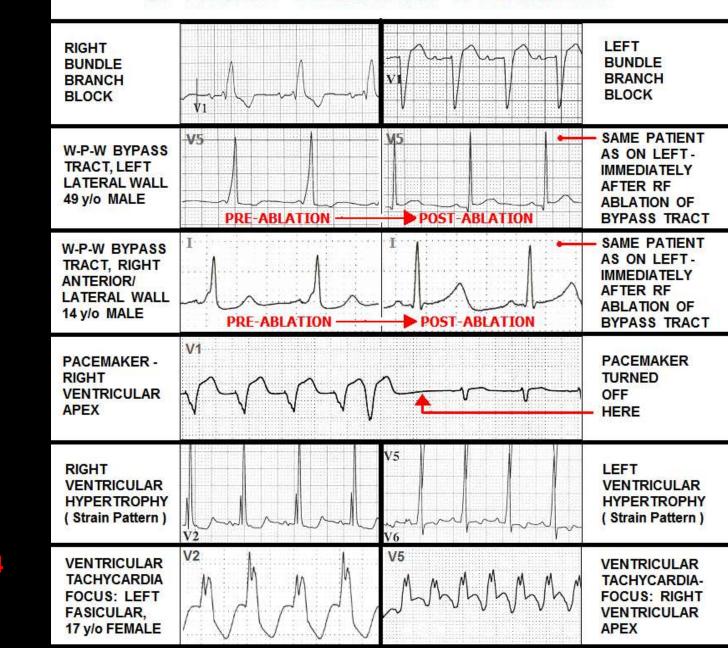
RIGHT BUNDLE BRANCH BLOCK ?????

WIDE QRS COMPLEXES ALTER THE -J POINTS -ST SEGMENTS -T WAVES

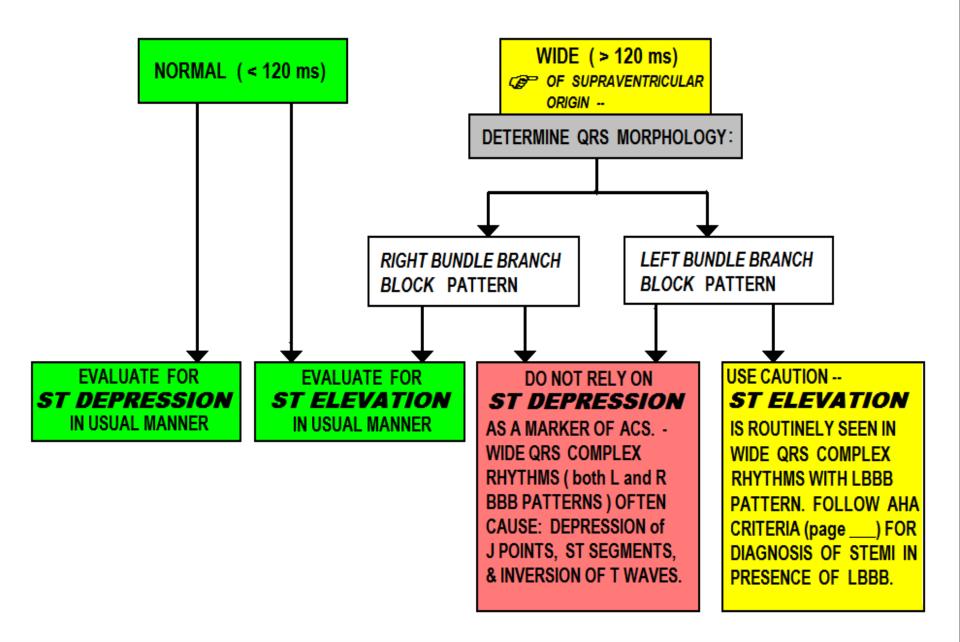
Of the ECG . . .

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ONDITIONS WHICH ALTER THE ECG MARKERS of ACUTE CORONARY SYNDROME



STEP 1 - EVALUATE WIDTH OF QRS:



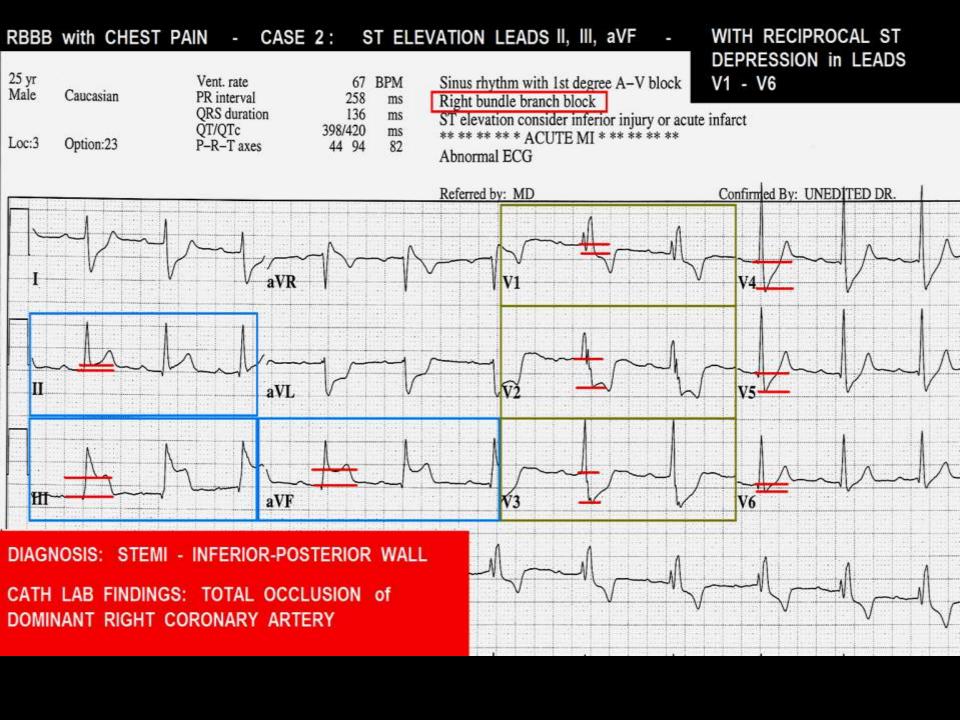
INFARCTION

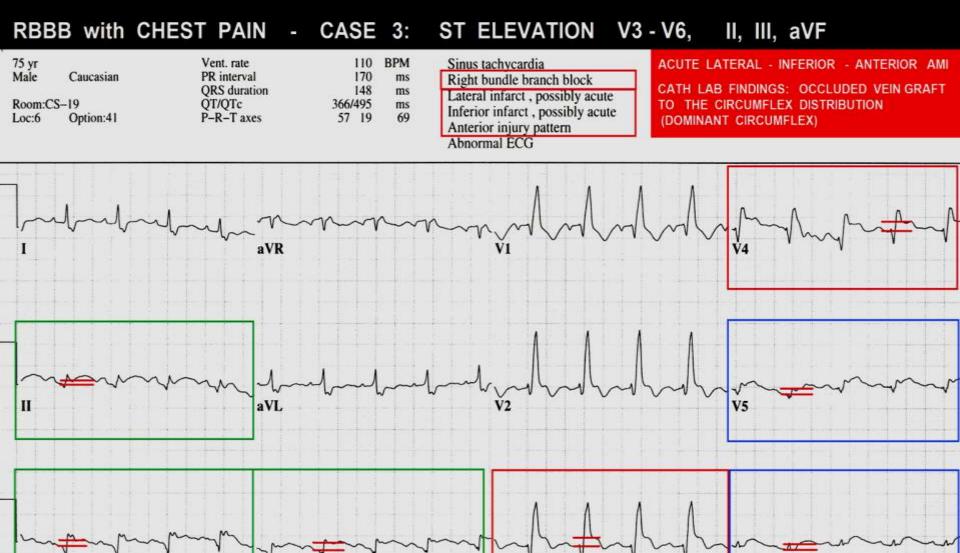
A.H.A. ACLS GUIDELINES 2000 / 2006

PATIENTS with RIGHT BUNDLE BRANCH BLOCK --



use J-POINTS and S-T SEGMENTS in the *usual* manner to screen for ACUTE MI





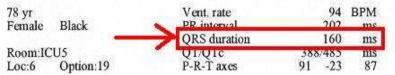
A.H.A. ACLS GUIDELINES

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

for diagnosis of STEMI

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

consider diagnosis as STEMI until proven otherwise.



Normal sinus rhythm with occasional Premature ventricular complexes

Left bundle branch block Abnormal ECG - Normal arteries

- Normal LV Function

No hypertrophy

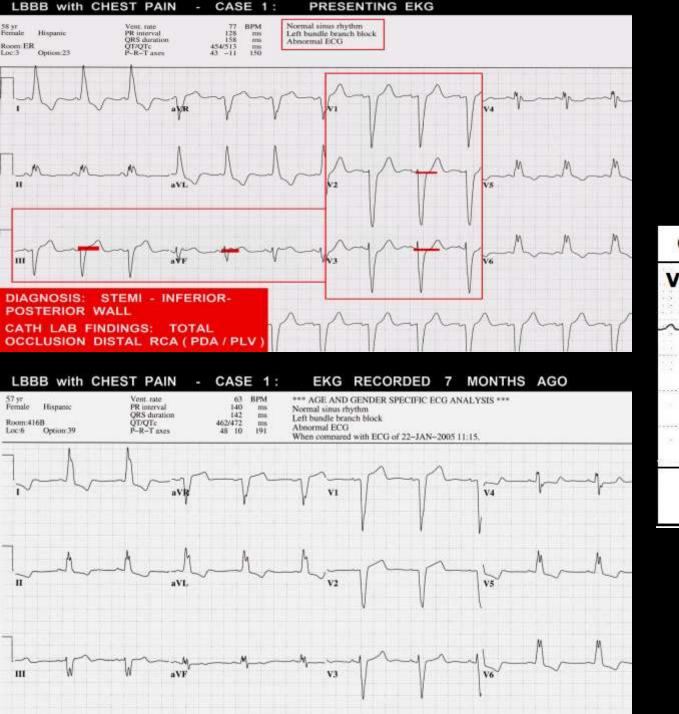






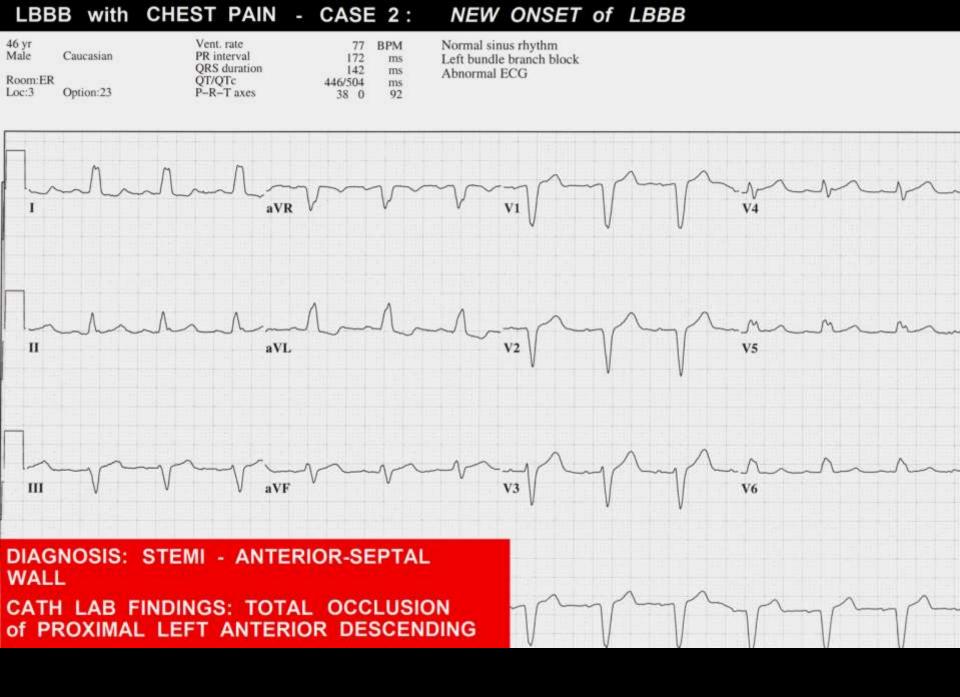
HELPFUL INDICATORS FOR ECG DIAGNOSIS OF STEMI in the presence of LBBB:

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





CHANGE



IF THE QRS COMPLEXES ON THE EKG ARE OF NORMAL WIDTH (<120 ms):

STEP 2 - EVALUATE the EKG for ACS

THE EKG MARKERS USED FOR DETERMINING THE PRESENCE OF ACUTE CORONARY SYNDROME INCLUDE:

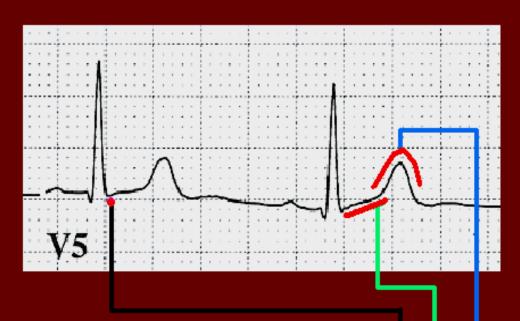
- J POINTS
- ST SEGMENTS
- T WAVES

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

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NORMAL ST - T WAVES

- WHEN QRS WIDTH IS NORMAL (< 120 ms)



ASSESS:

- J POINT: ISOELECTRIC (or < 1 mm dev.)

- ST SEG: SLIGHT, POSITIVE INCLINATION

- T WAVE: UPRIGHT, POSITIVE -



in EVERY LEAD EXCEPT aVR !!

ALL KINDS of WEIRD

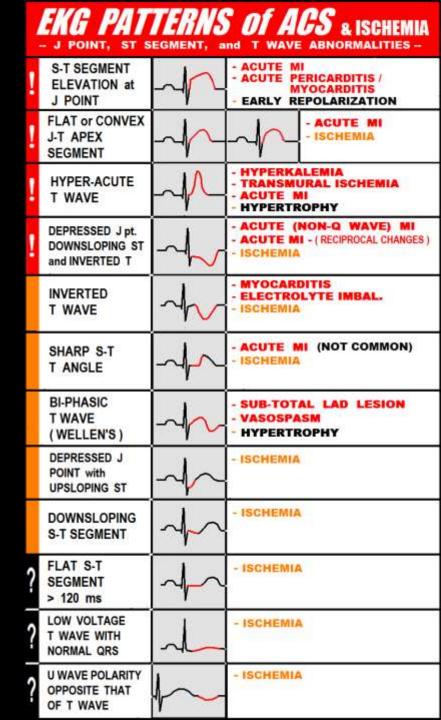
ST SEGMENT and T WAVE

VARIATIONS

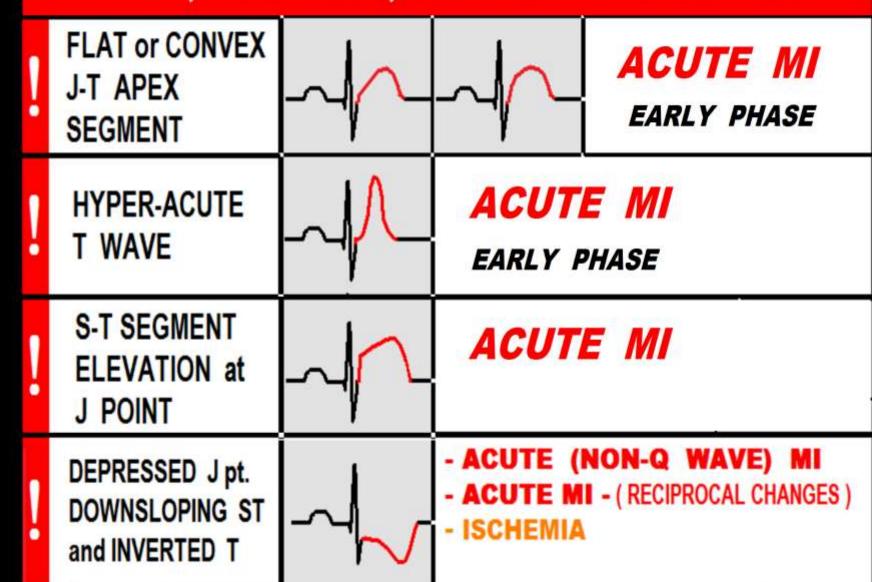
ALL CAN SPELL

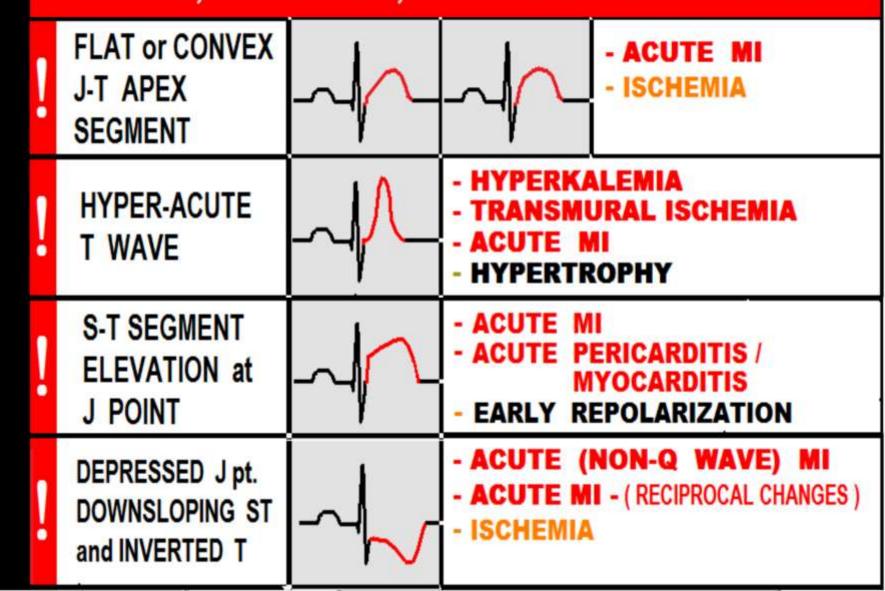
T-R-O-U-B-L-E.

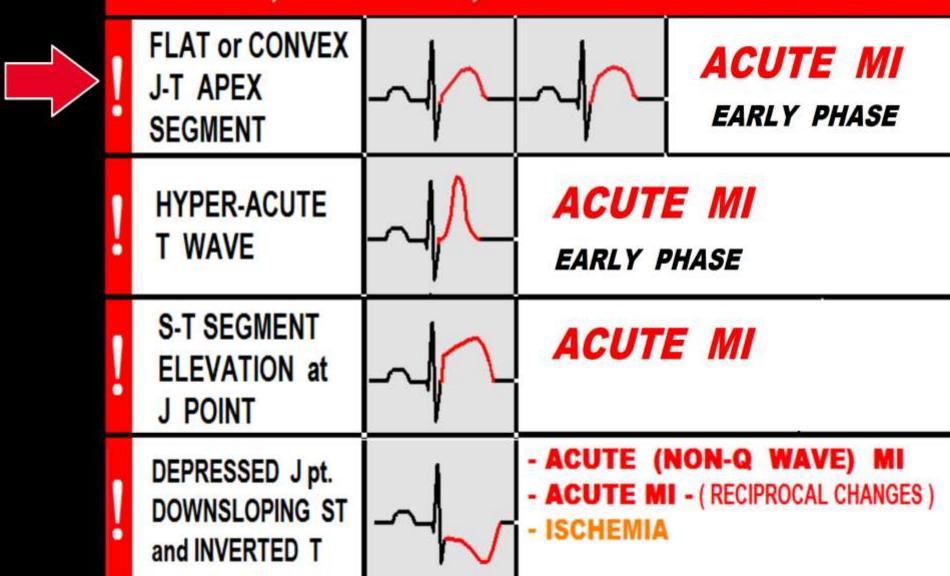
"IF IT'S NOT NORMAL, it's ABNORMAL!

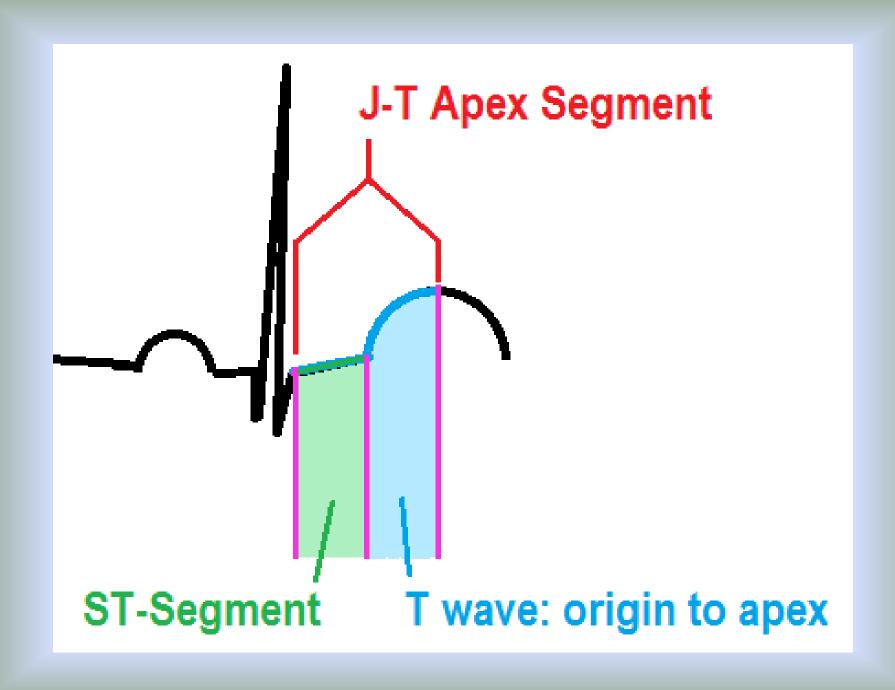


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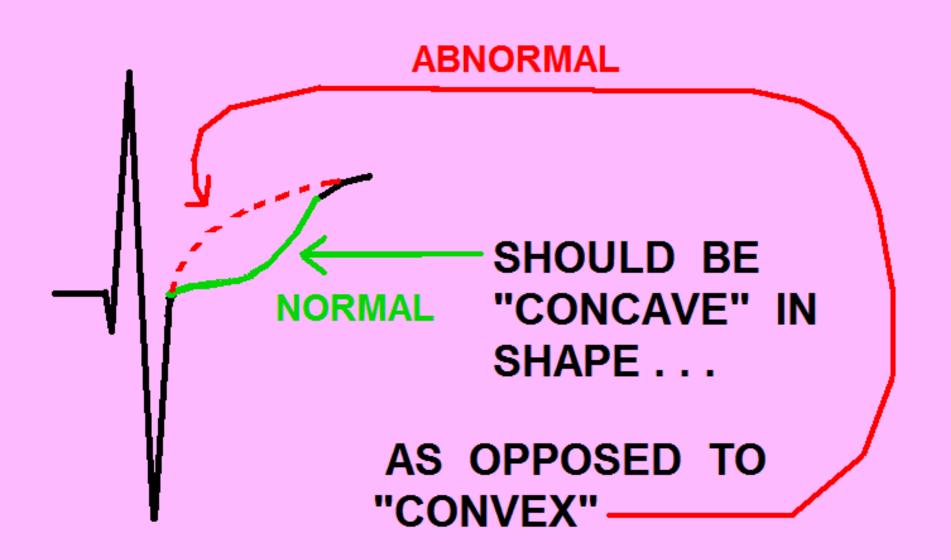








THE S-T SEGMENT



From:

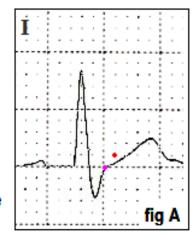
AMERICAN HEART ASSOCIATION ACLS 2005 REVISIONS

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

CONCAVE appearance.

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

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



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

J POINT

"JPOINT plus 40 ms"

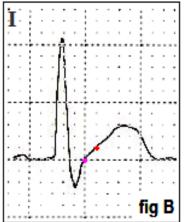
shows ST ELEVATION > 1 mm

INFARCTION -EARLY PHASE

NORMAL

ST SEGMENT

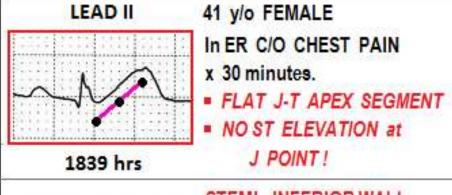
PATTERN

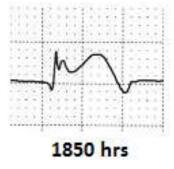


assumes a CONVEX shape.
When measured 40 ms
beyond the J POINT, the ST
segment is elevated > 1 mm.
This phenonemon is seen
routinely in the cath lab
prior to the occurance of ST
ELEVATION at the J POINT
during PTCA and STENTING.

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

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



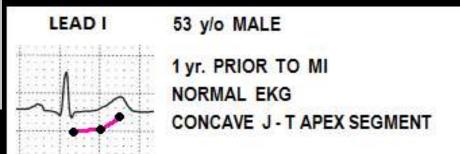


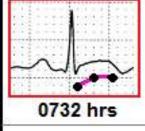
STEMI - INFERIOR WALL

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

 CATH LAB FINDINGS:
 TOTAL OCCLUSION of the RIGHT CORONARY ARTERY

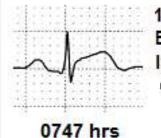
J POINT END of ST SEGMENT T WAVE APEX CONVEX J-T APEX SEGMENT CONSIDER EARLY PHASE of ACUTE MI!





STEMI LATERAL WALL • CONVEX J-T APEX SEGMENT

- MINIMAL ST ELEVATION
- at J POINT



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

CASE STUDY: ABNORMAL J-T APEX SEGMENTS

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

RISK FACTOR PROFILE:

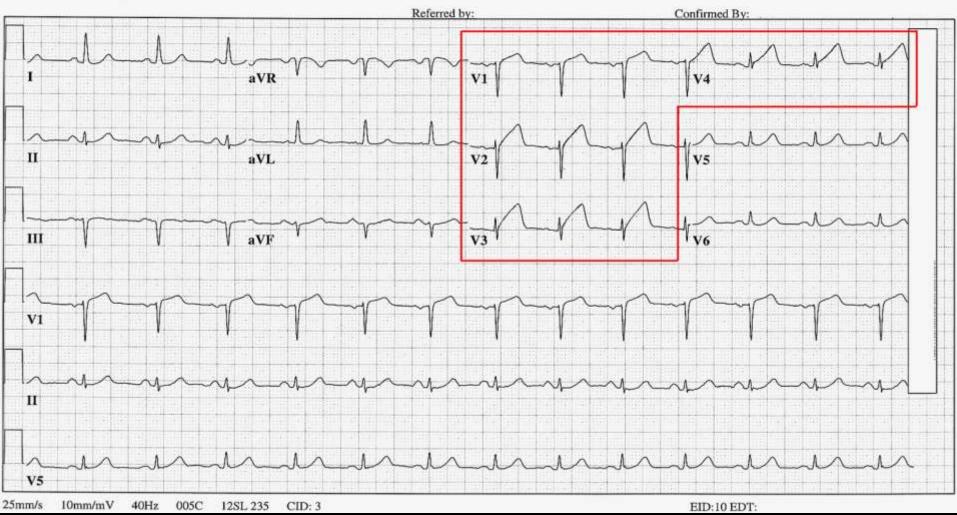
- FAMILY HISTORY father died of MI at age 62
- ☑ PREVIOUS CIGARETTE SMOKER quit 15 years ago.
- ☑ CHOLESTEROL DOES NOT KNOW; "never had it checked."
- ✓ OBESITY

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

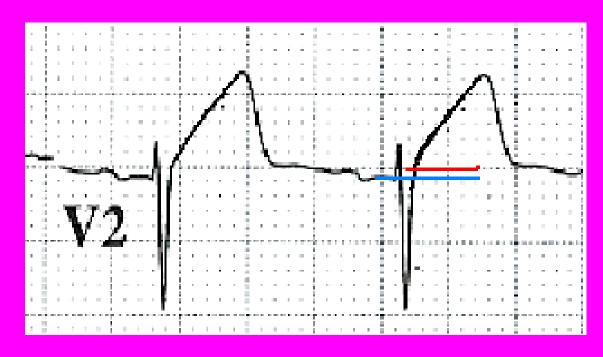
VITAL SIGNS: BP 142/94, P 80, R 20, SAO2 98%

LABS: JUST OBTAINED, RESULTS NOT AVAILABLE YET.

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



measurement of S-T elevation



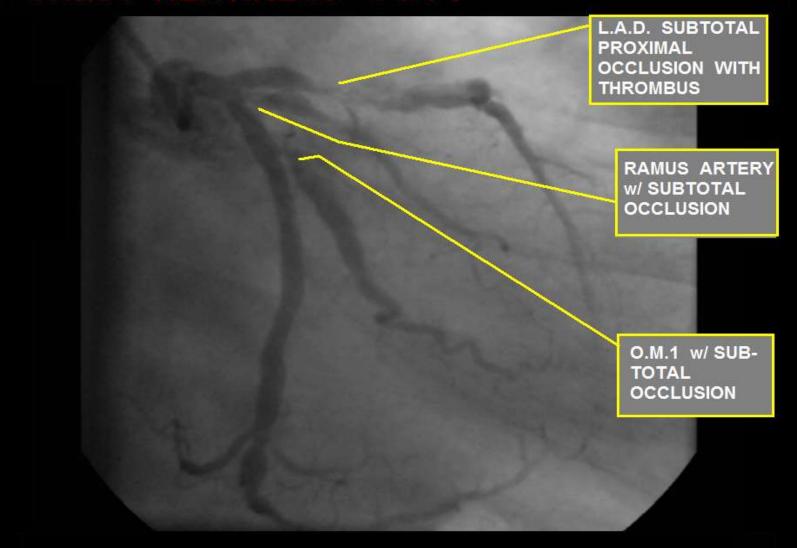
S-T elevation at J point = 0.5 mm

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



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

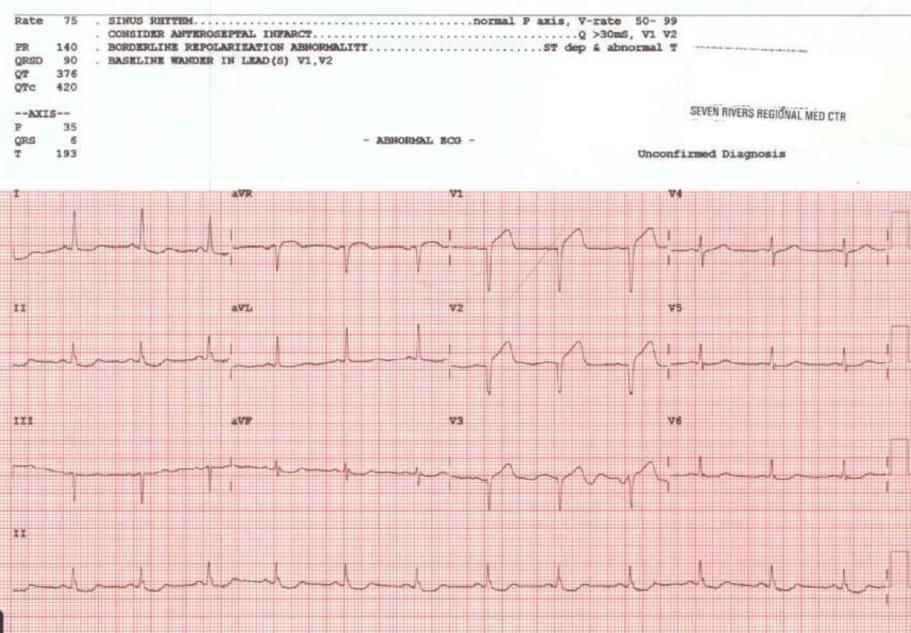
CASE STUDY: 56 y/o male with INTERMITTENT "CHEST HEAVINESS"



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

ECG Patterns associated with "EARLY PHASE MI:"

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



Chest: 10.0 mm/mV

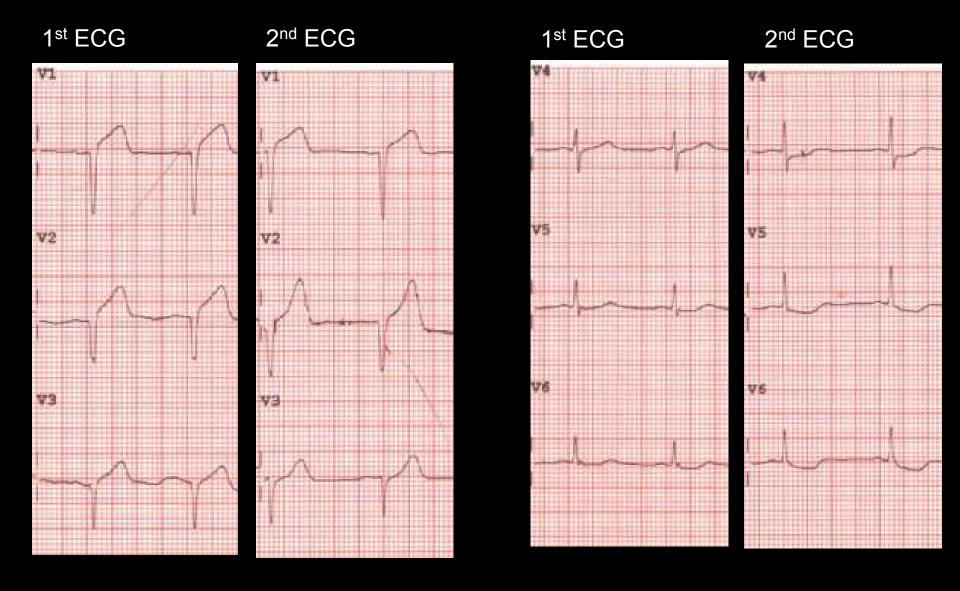
F 60- 0.15-100 Hz

PHOSON

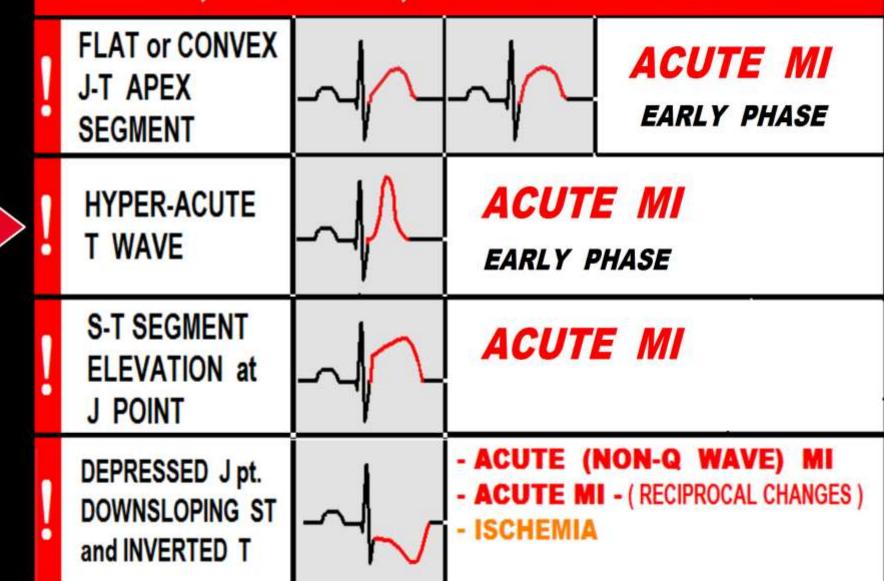
Limb: 10 mm/mV

Dev:

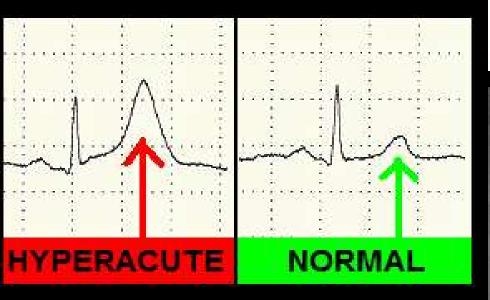
3. Dynmamic ST-T Wave Changes in Serial ECGs. Recorded at SRRMC



Acute In-Stent Thrombus Proximal LAD

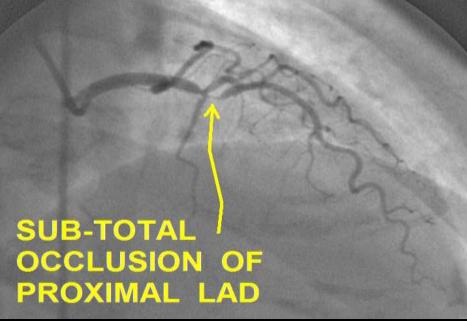


HYPERACUTE T WAVES



BOOK PAGE: 88

HYPER-ACUTE T WAVES - COMMON ETIOLOGIES: CONDITION: SEE PAGE(S): HYPERKALEMIA — xx - xx ACUTE MI — xx - xx TRANS-MURAL ISCHEMIA — xx - xx HYPERTROPHY — xx - xx



Helpful Clue: Hyper-Acute T Waves

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

Vent. rate PR interval

Room:

57 bpm 150 ms QRS duration 102 ms QT/QTc 472/459 ms 76 70 58

Sinus bradyc Possible Left atrial enlargement Borderline ECG

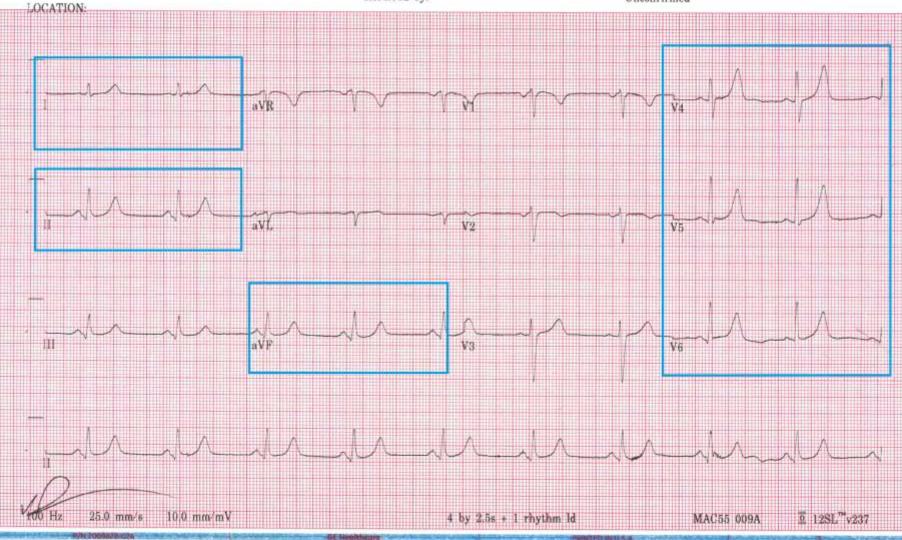
ER ATTENDING REVIEW

K + = 6.7

Technician: Test ind:



Unconfirmed



Helpful Clue: Hyper-Acute T Waves

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

CASE STUDY: HYPERACUTE T WAVES

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

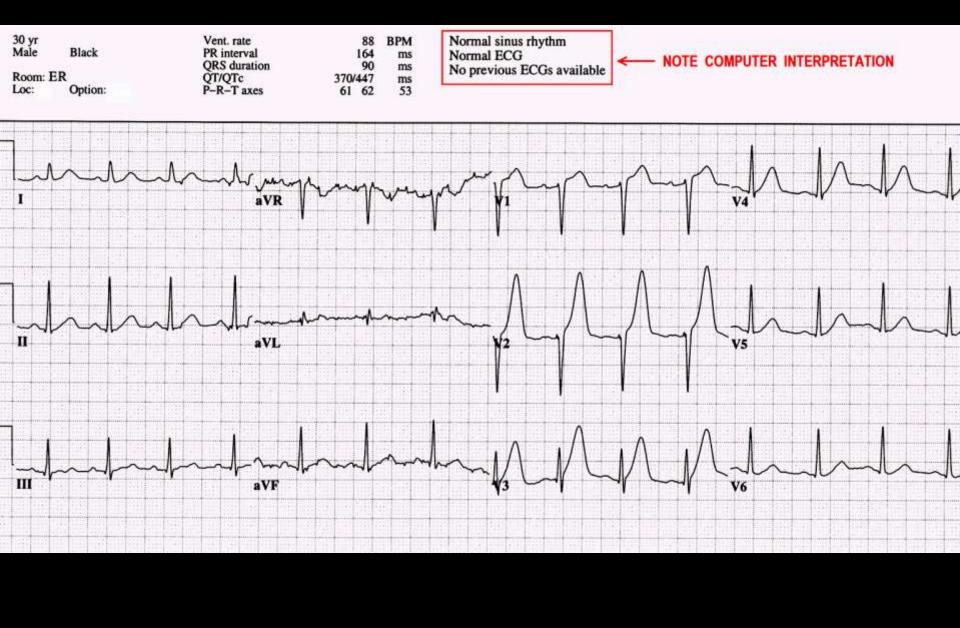
30 y/o male presents to ER via EMS, c/o sudden onset of dull chest pain x 40 min. Pain level varies, not effected by position, movement or deep inspiration. No associated symptoms.

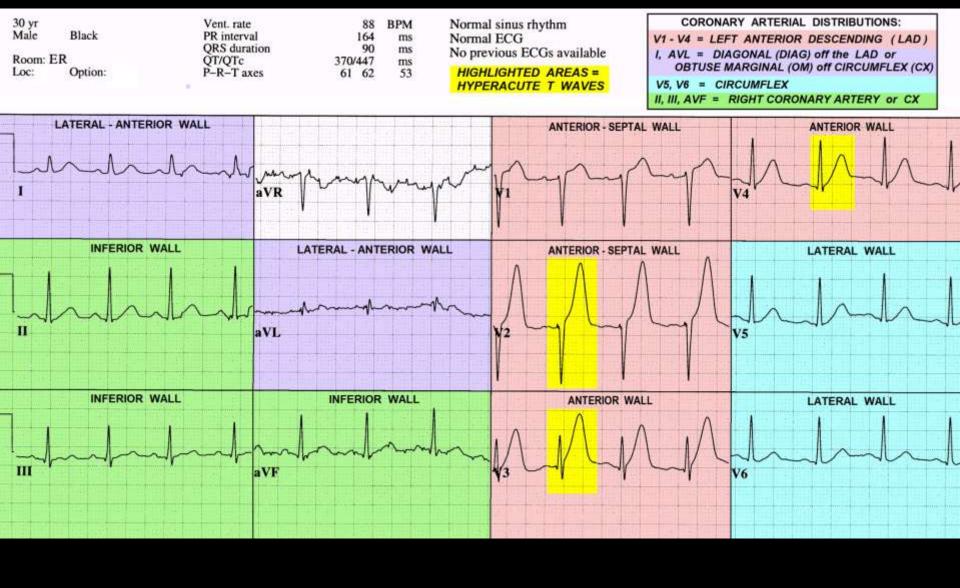
RISK FACTOR PROFILE: NONE. CHOLESTEROL UNKNOWN.

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

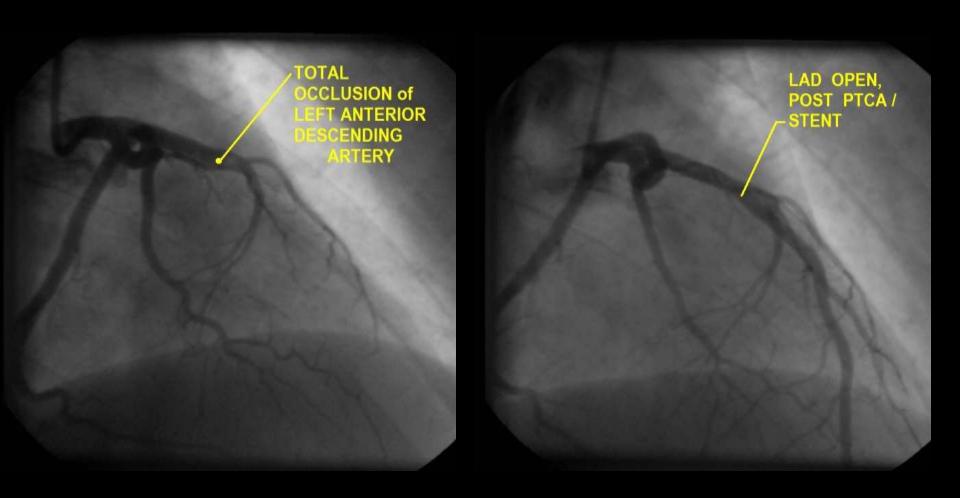
VITAL SIGNS: BP 136/88 P 90 R 20 SAO2 98%

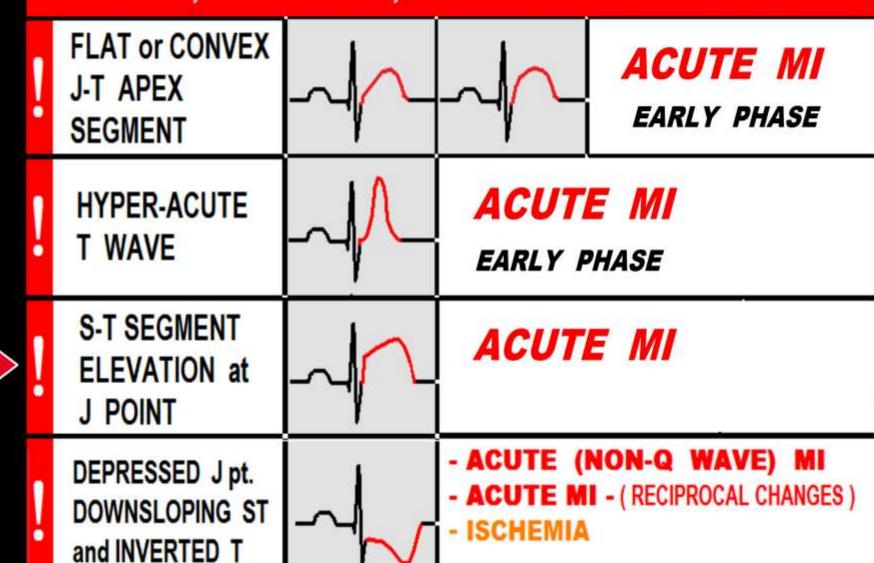
DIAGNOSTIC TESTING: 1st TROPONIN I - ultra: <0.07





Cath Lab findings:





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

*LEADS V2 and V3:

MALES AGE 40 and up ---- 2.0 mm

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

FEMALES ----- 1.5 mm

ALL OTHER LEADS: 1.0 mm or more,

in TWO or more

CONTIGUOUS LEADS

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

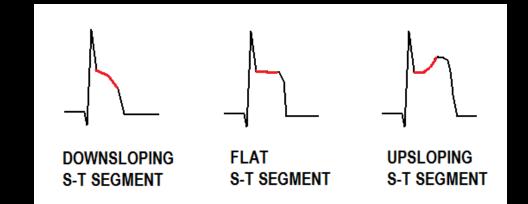
ST SEGMENT ELEVATION:

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



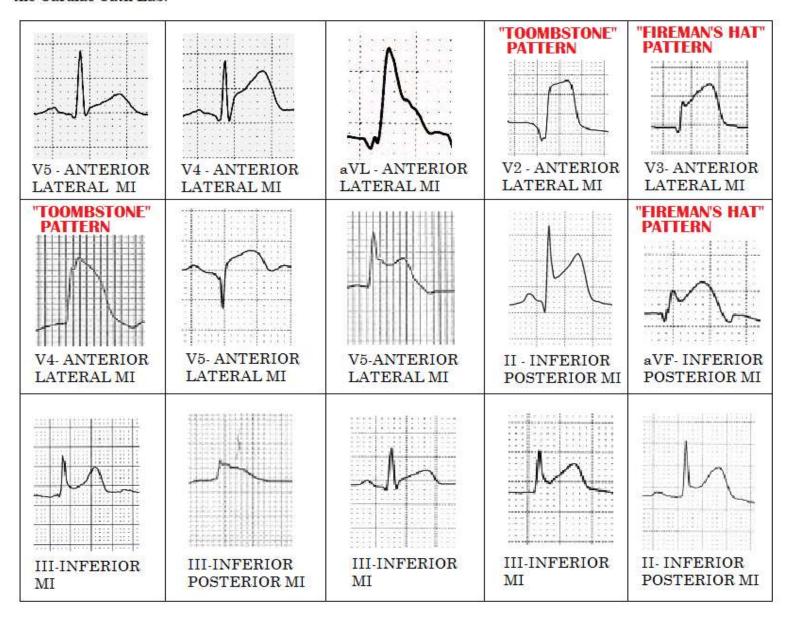
IN THIS CASE, a normal response to balloon occlusion of the RIGHT CORONARY ARTERY during PTCA in the CARDIAC CATH LAB

3 COMMON PATTERNS of ST SEGMENT ELEVATION From ACUTE MI:



ST SEGMENT ELEVATION in ACUTE MI:

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



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

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

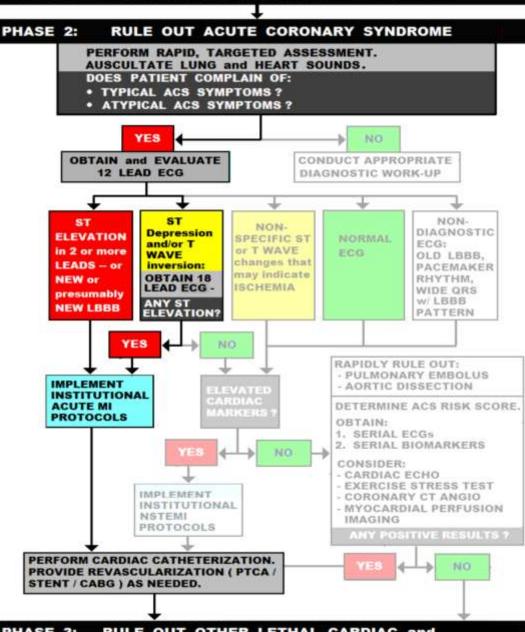
However the *lack of Reciprocal S-T Depression* DOES NOT rule out AMI.

ACUTE MI

COMPLICATIONS TO ANTICIPATE FOR ALL MI PATIENTS:

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

STEMI GASE STUDIES



RULE OUT LIFE-THREATENING CONDITIONS

PHASE 1:

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

CASE STUDY 1 - STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

RISK FACTOR PROFILE:

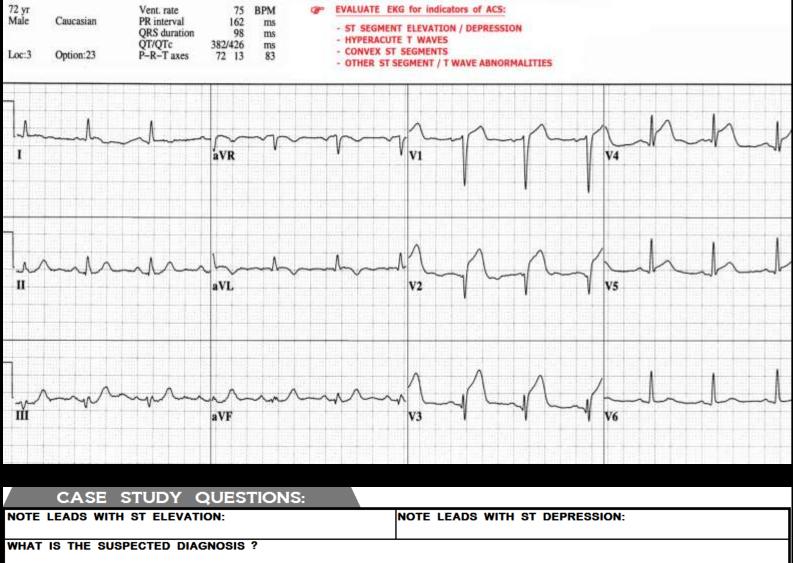
- FAMILY HISTORY father died of MI at age 77
- FORMER CIGARETTE SMOKER smoked for 30 year quit 27 years ago
- ► DIABETES oral meds and diet controlled
- HIGH CHOLESTEROL controlled with STATIN meds
- AGE: OVER 65

PHYSICAL EXAM: Patient calm, alert, oriented X 4, skin cool, dry, pale.

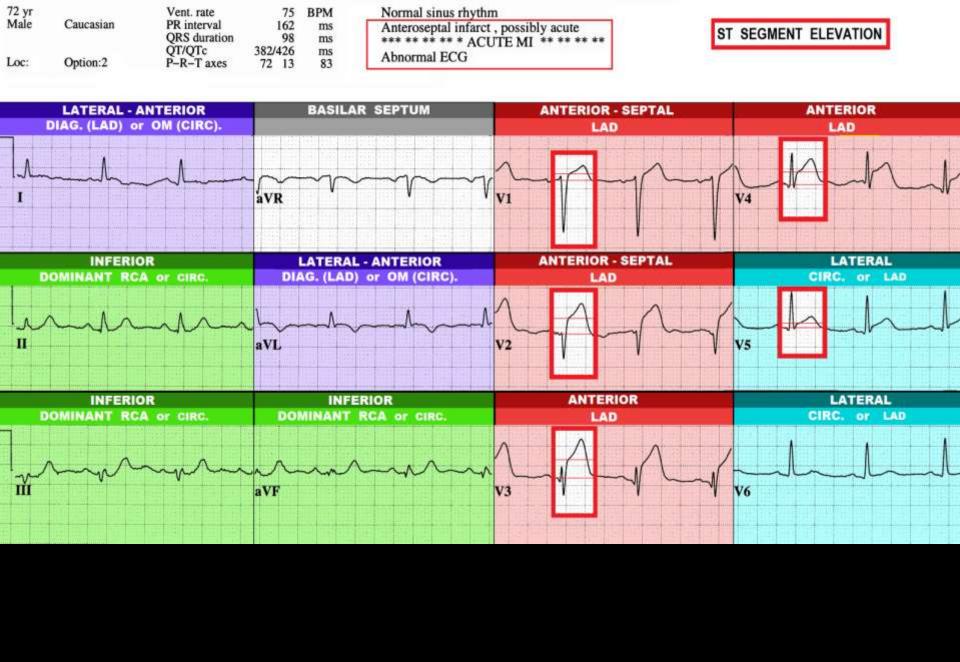
No JVD, Lungs clear bilaterally. Heart sounds normal S1, S2. No peripheral edema.

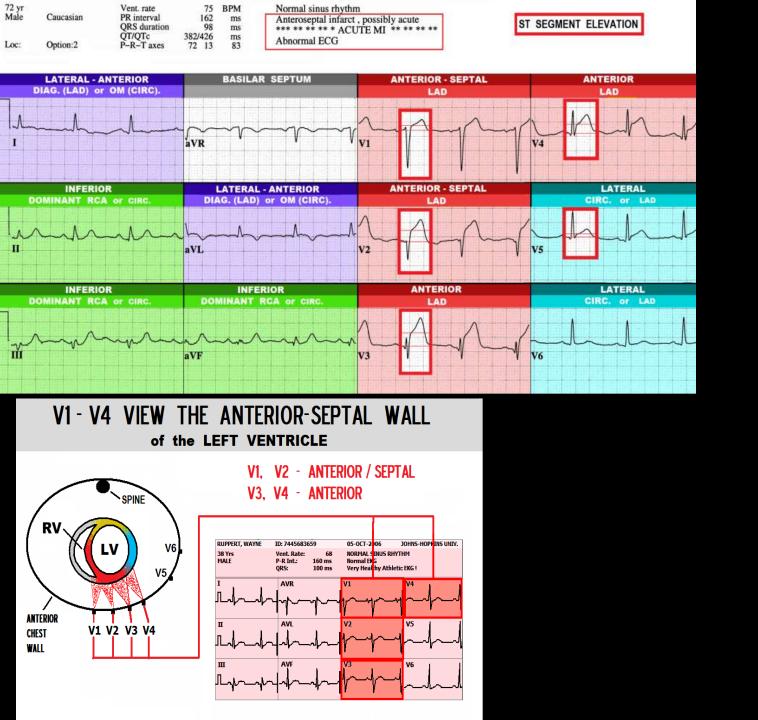
VITAL SIGNS: BP: 100/64, P: 75, R: 20, SAO2: 94%

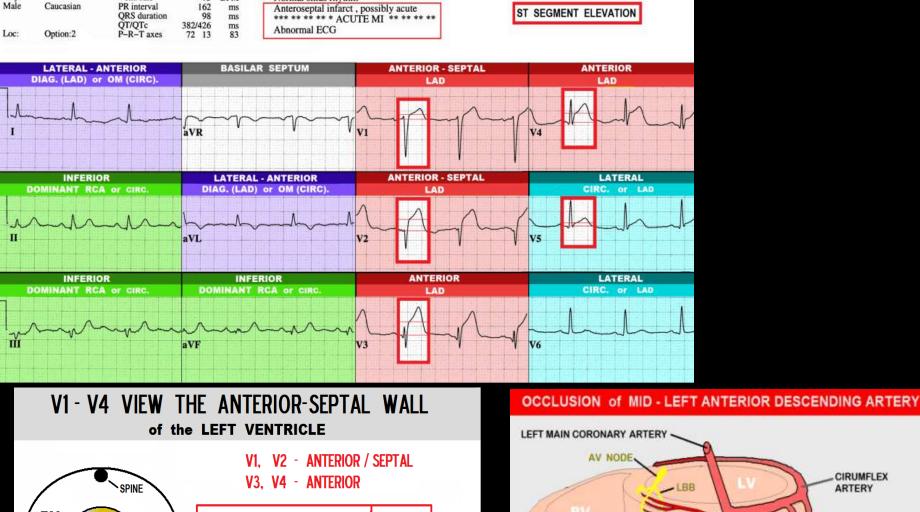
LABS: FIRST TROPONIN: 6.4



NOTE 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:







05-0CT-2 06

Normal FK Very Heal hy Athletic EKG!

NORMAL SINUS RHYTHM

JOHNS-HOPKINS UNIV.

Normal sinus rhythm

Vent. rate

PR interval

ORS duration

Caucasian

75 **BPM**

ms

RUPPERT, WAYNE

MALE

V6

V1 V2 V3 V4

ANTERIOR

CHEST WALL

ID: 7445683659

160 ms

Vent. Rate:

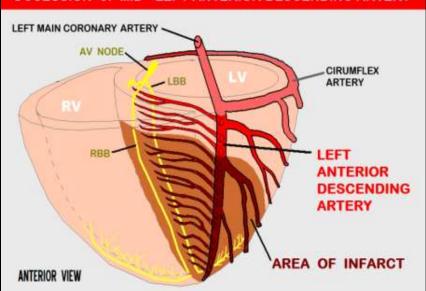
P-R Int.:

AVR

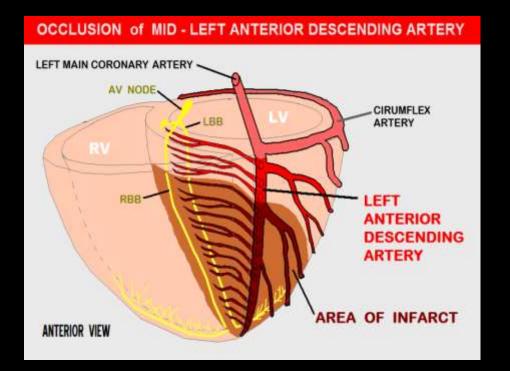
QRS:

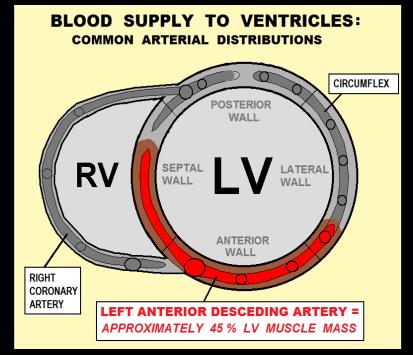
162

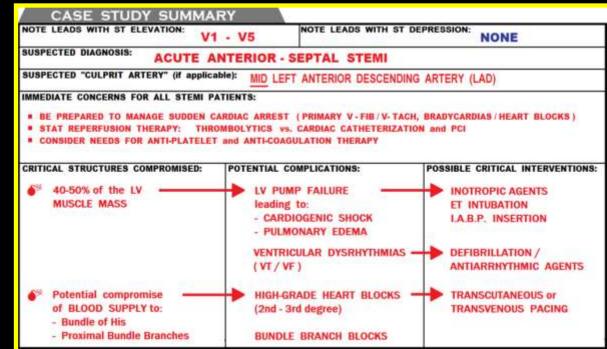
98

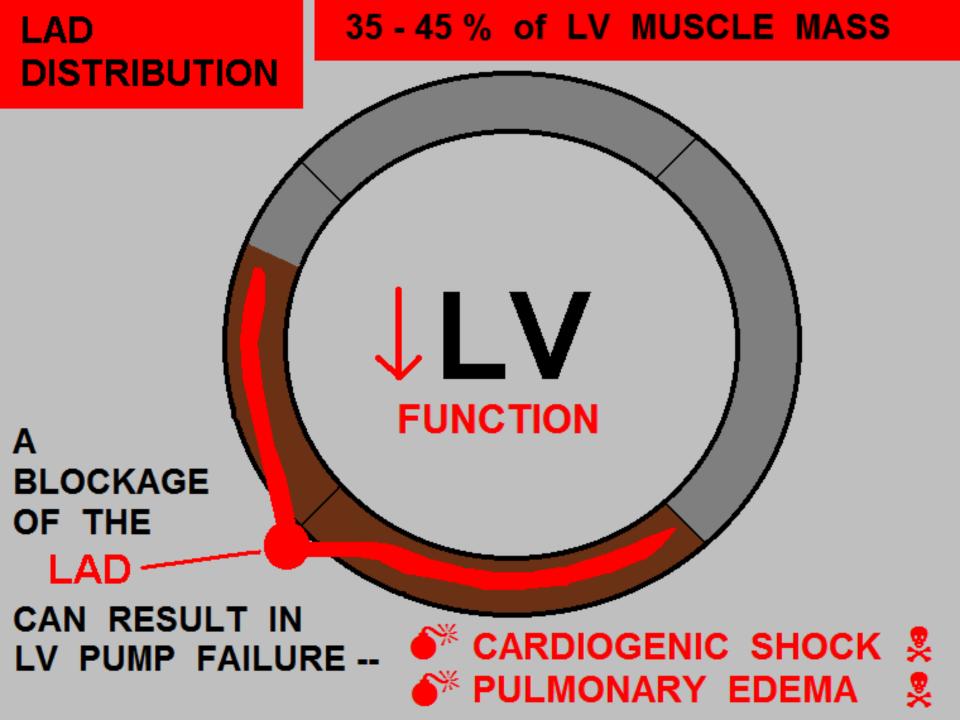


ST SEGMENT ELEVATION





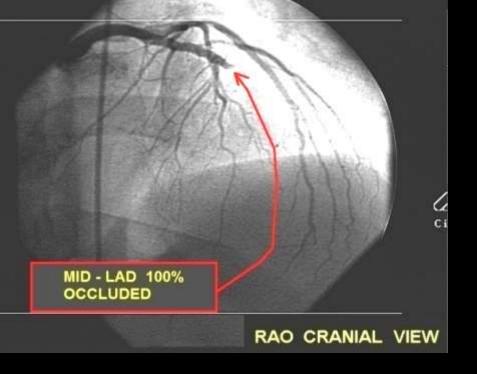


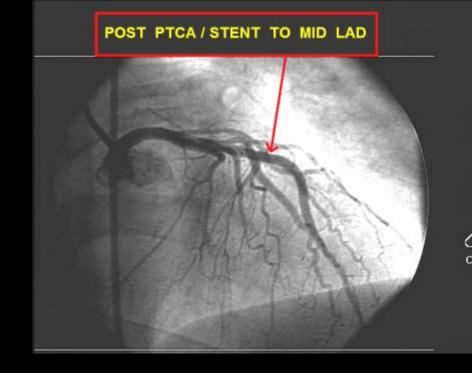


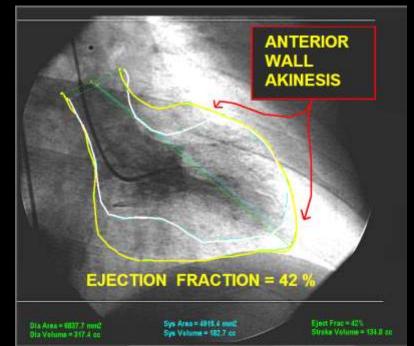


LEFT ANTERIOR DESCENDING ARTERY (LAD)

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







CASE STUDY 2: STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

RISK FACTOR PROFILE:

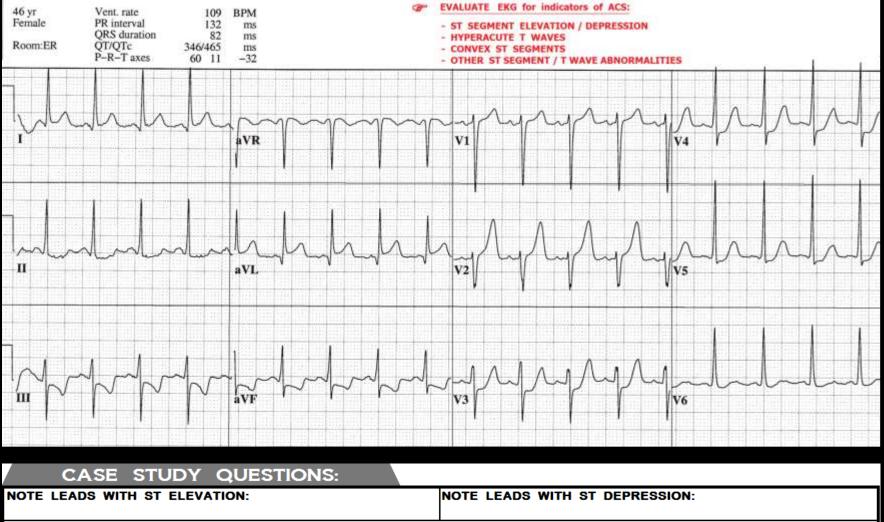
- FAMILY HISTORY father died of CAD, older brother had CABG, age 39
- DIABETES diet controlled
- HYPERTENSION

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

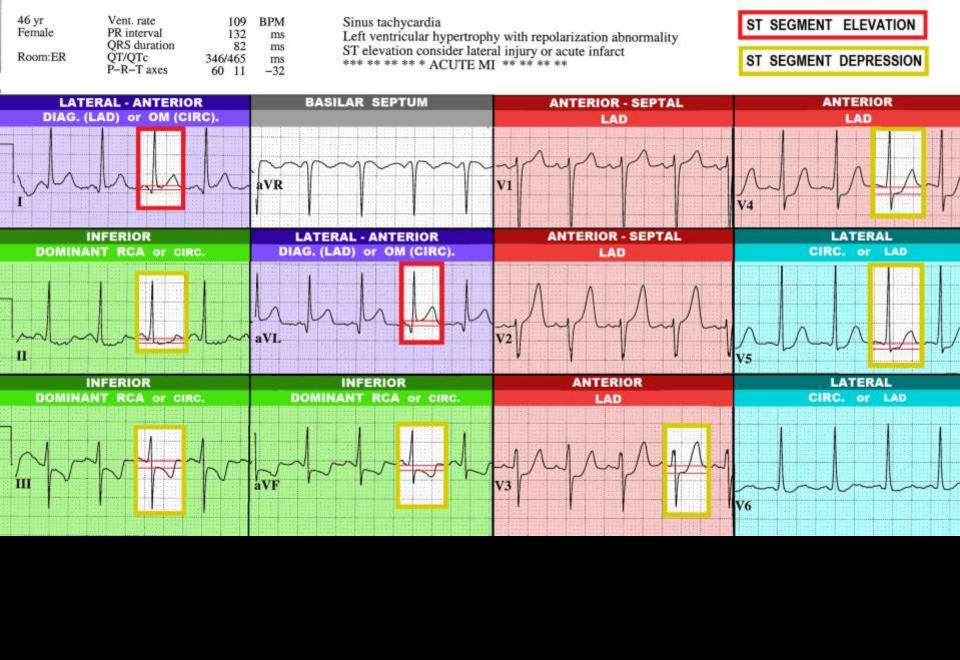
Lungs: clear, bilaterally. Heart Sounds: Normal S1, S2.

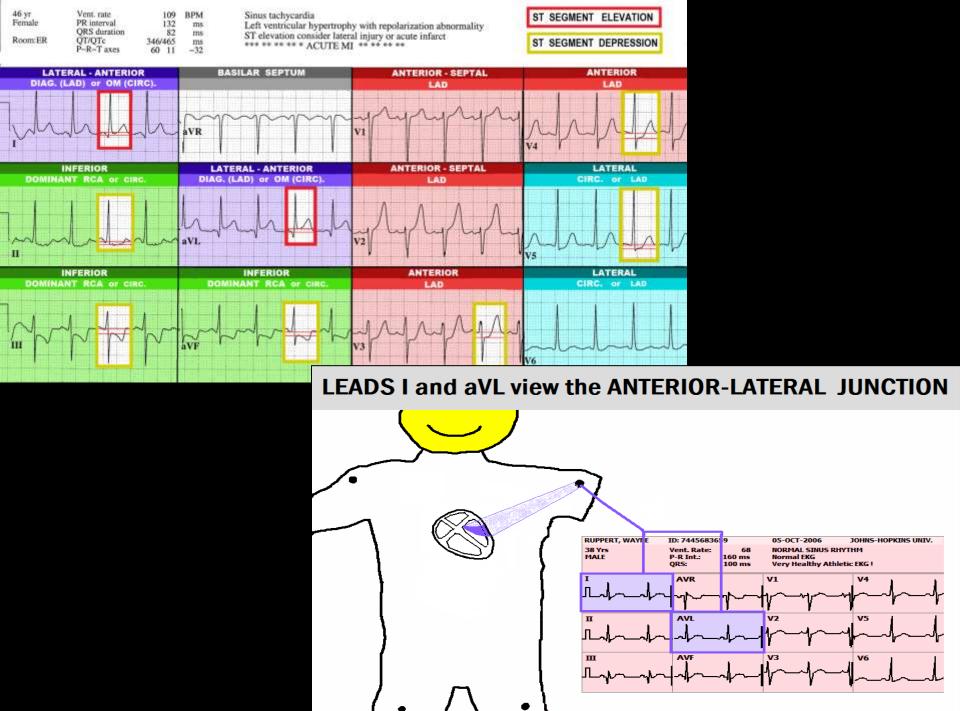
VITAL SIGNS: BP: 168/98, P: 110, R: 24, SAO2: 97% on O2 4 LPM via nasal canula

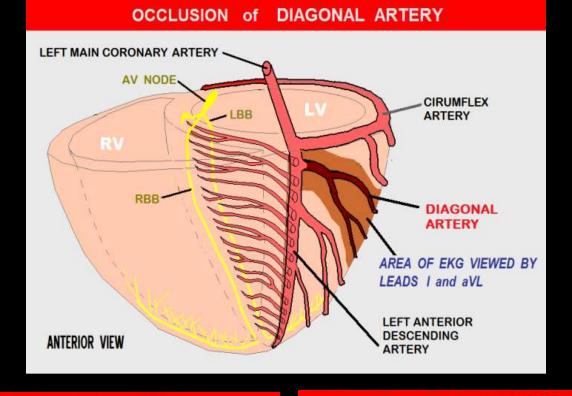
LABS: TROPONIN ultra = 2.8

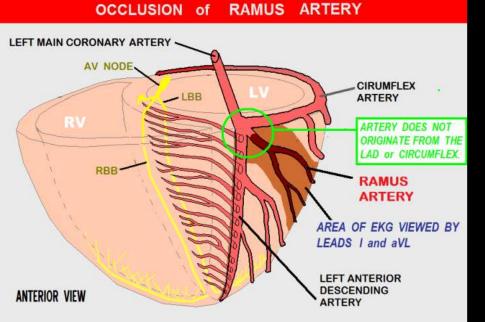


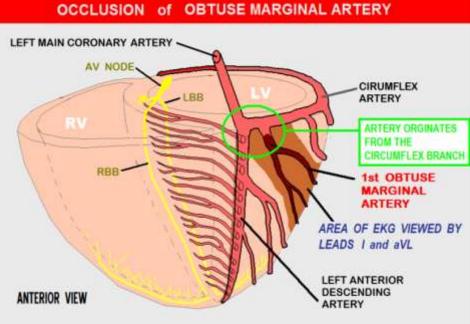
NOTE LEADS WITH ST ELEVATION: WHAT IS THE SUSPECTED DIAGNOSIS ? WHAT IS THE "CULPRIT ARTERY" -- if applicable? LIST ANY CRITICAL STRUCTURES COMPROMISED: LIST ANY POTENTIAL COMPLICATIONS:



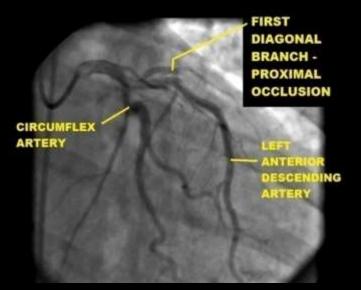


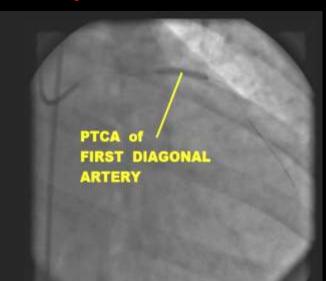






CASE PROGRESSION: As the patient was being prepared for transport to the Cardiac Cath Lab, she experienced an episode of Ventricular Fibrillation.









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

CASE STUDY 3: STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

RISK FACTOR PROFILE:

FAMILY HISTORY - father died of MI age 46

CURRENT CIGARETTE SMOKER

■ "MILD" HYPERTENSION - untreated

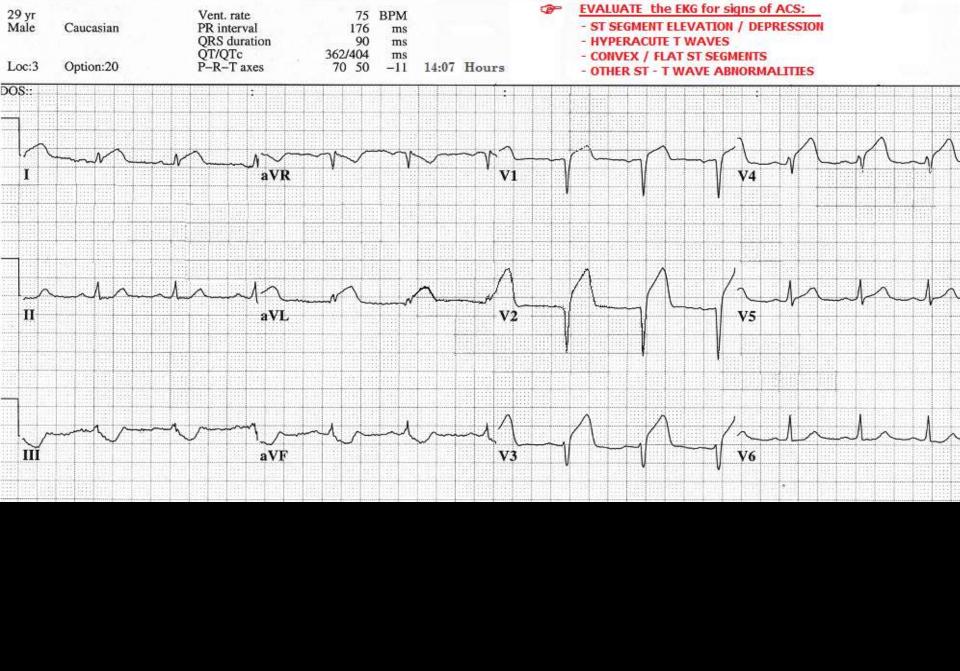
☑ CHOLESTEROL - unknown - "never had it checked."

PHYSICAL EXAM: Patient alert, oriented X 4, skin cool, dry, pale. Patient restless.

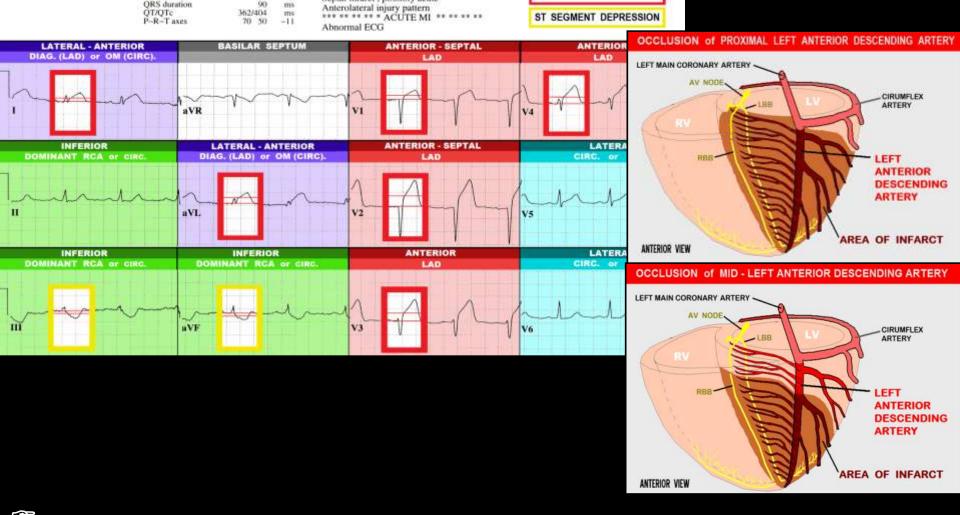
No JVD, Lungs clear bilaterally. Heart sounds normal S1, S2. No peripheral edema.

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

LAES: INITIAL CARDIAC MARKERS - NEGATIVE







SEGMENT ELEVATION

- THERE ARE TWO IMPORTANT CLUES that the patient's BLOCKAGE is in the *PROXIMAL* LEFT ANTERIOR DESCENDING ARTERY:
- 1. When ST elevation is noted in leads I and aVL in cases of ANTERIOR WALL STEMI, it is a good indicator that the FIRST DIAGONAL BRANCH is included in the zone of infarction.
- RECIPROCAL ST DEPRESSION in the INFERIOR LEADS (II, III, and/or aVF) is an indication that the LAD is blocked proximal to the FIRST DIAGONAL BRANCH.^[1]

75

176

Normal sinus rhythm

Septal infarct, possibly acute

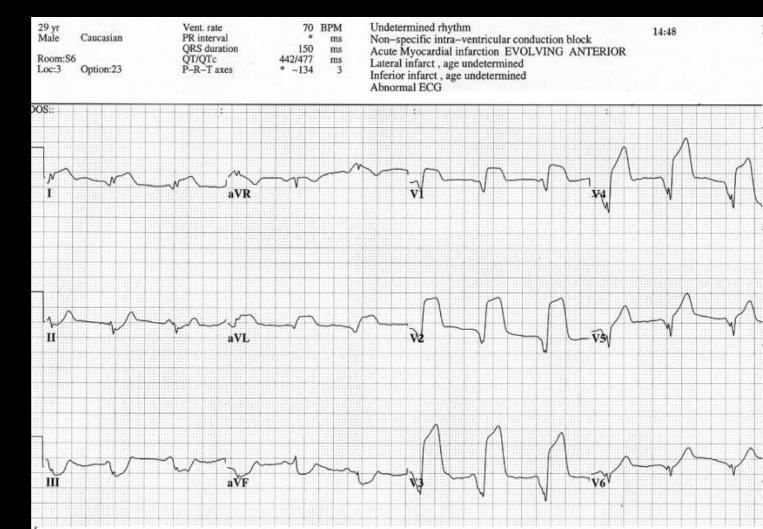
Vent. rate

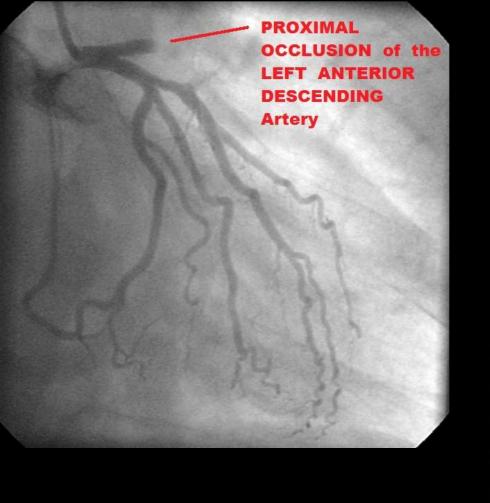
PR interval

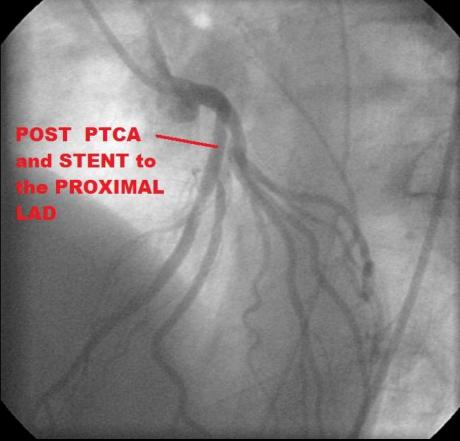
Caucasian

WHILE WAITING FOR THE RETEVASE TO WORK, THE PATIENT BEGAN VOMITING. SKIN BECAME ASHEN & DIAPHORETIC. REPEAT BP = 50/30.

-WHAT THERAPEUTIC INTERVENTIONS SHOULD BE IMPLMENTED AT THIS POINT?







CASE STUDY SUMMARY ST ELEVATION: V1 - V5, I, aVL ST DEPRESSION: III, aVF SUSPECTED DIAGNOSIS: **ACUTE ANTERIOR - SEPTAL STEMI** SUSPECTED "CULPRIT ARTERY" (if applicable): PROXIMAL LEFT ANTERIOR DESCENDING ARTERY (LAD) 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: POSSIBLE CRITICAL INTERVENTIONS: POTENTIAL COMPLICATIONS: 40-50% of the LV LV PUMP FAILURE INOTROPIC AGENTS MUSCLE MASS leading to: **ET INTUBATION** - CARDIOGENIC SHOCK I.A.B.P. INSERTION PULMONARY EDEMA **DEFIBRILLATION** / VENTRICULAR DYSRHYTHMIAS (VT/VF) ANTIARRHYTHMIC AGENTS Potential compromise HIGH-GRADE HEART BLOCKS TRANSCUTANEOUS or of BLOOD SUPPLY to: (2nd - 3rd degree) TRANSVENOUS PACING Bundle of His

PLUS: EXTENSION OF THROMBUS LOAD INTO THE LEFT MAIN CORONARY ARTERY....

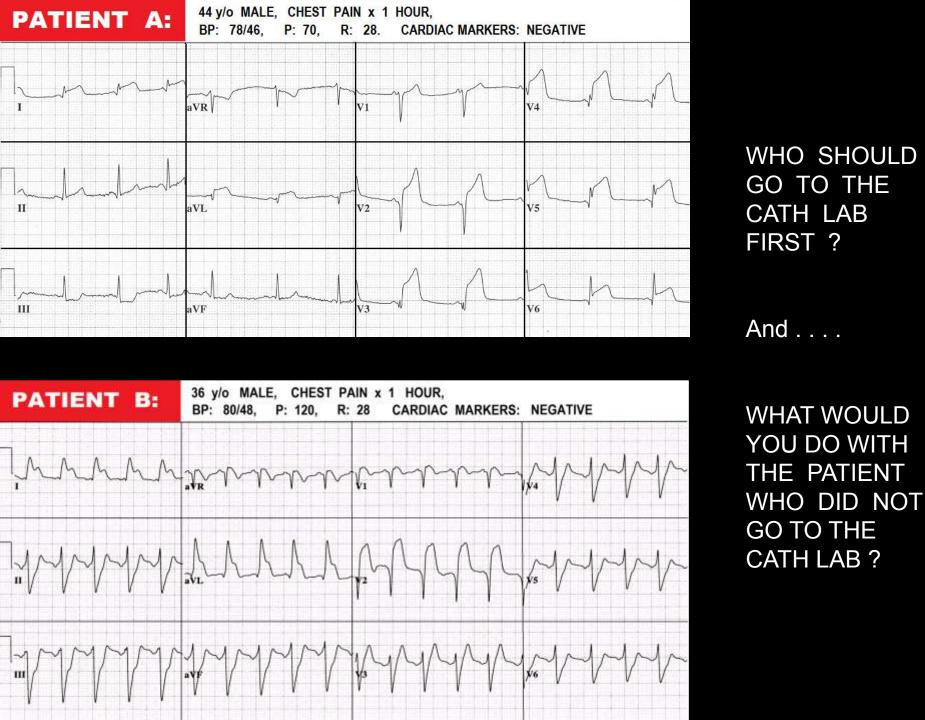
BUNDLE BRANCH BLOCKS

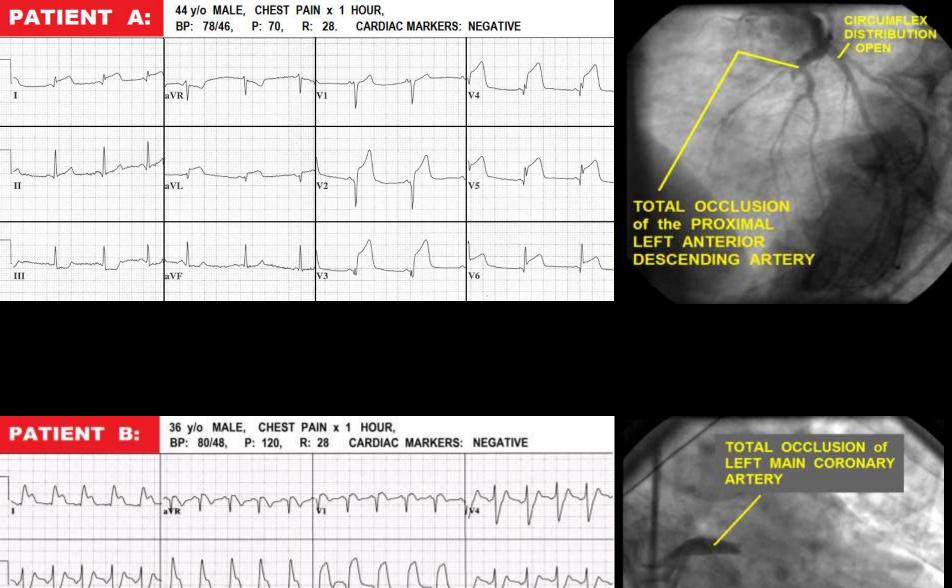
Proximal Bundle Branches

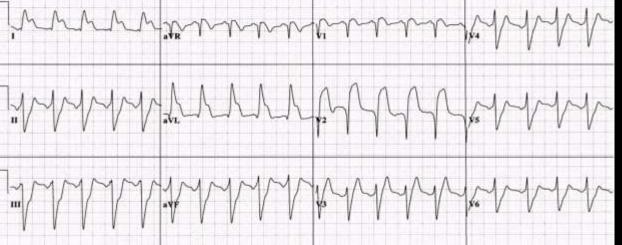
CASE STUDY 4: CRITICAL DECISIONS SCENARIO

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

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





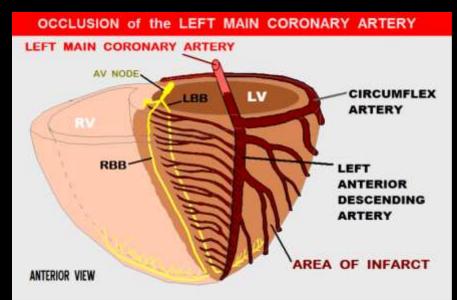


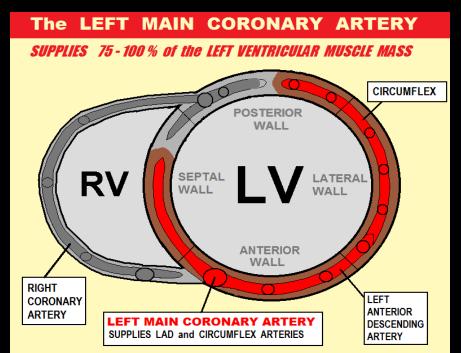
PATIENT A:

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

SUPPLIES 40-50% OF THE LEFT VENTRICULAR MUSCIE MASS RIGHT CORONARY ARTERY LEFT ANTERIOR DESCEDING ARTERY = APPROXIMATELY 45 % LV MUSCLE MASS

PATIENT B:





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

- ☑ ST DEPRESSION or ISOELCTRIC J POINTS may be seen in V LEADS.... mainly V2 and/or V3 caused by COMPETING FORCES of ANTERIOR vs. POSTERIOR WALL MI.**

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

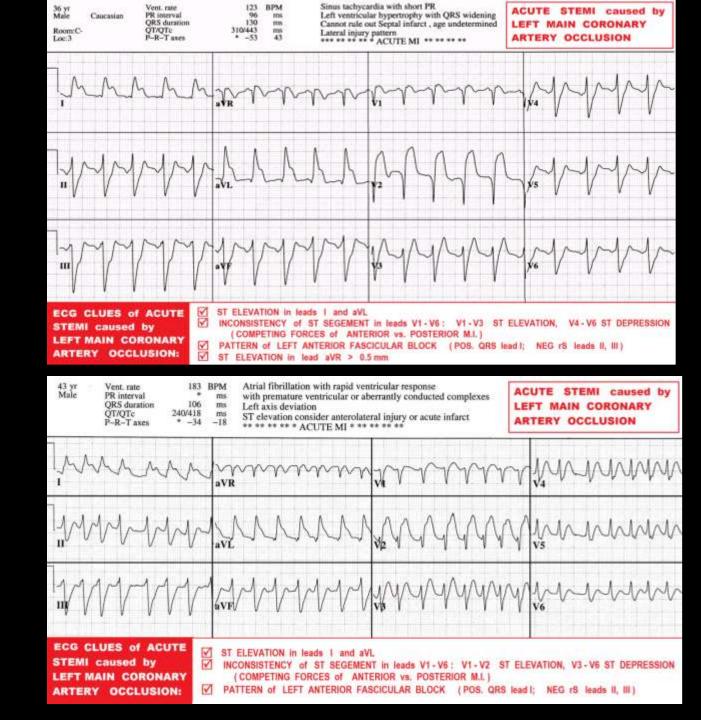
ECG CLUES of ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION:

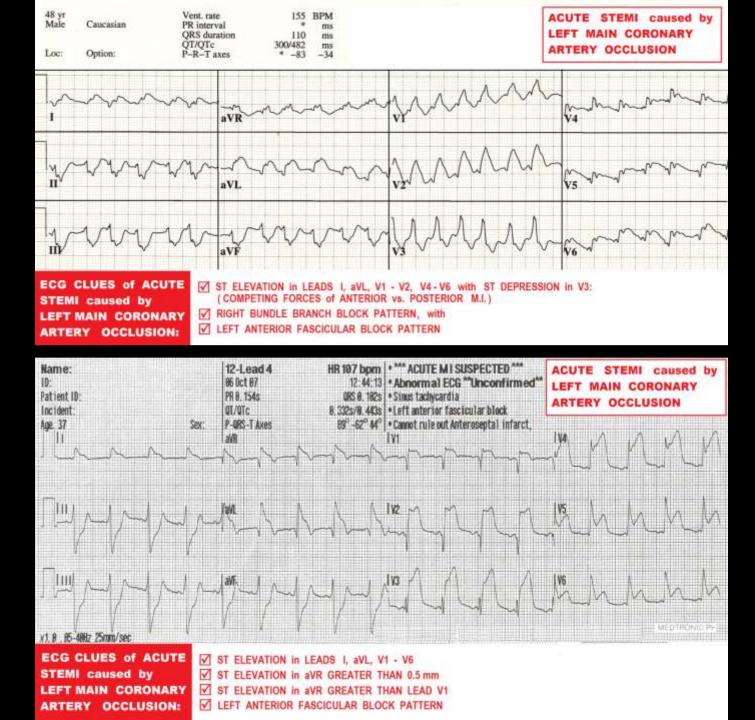
- ST ELEVATION in aVR (2 mm) > ST ELEVATION in V1 (1.5 mm)
- ST ELEVATION in V1 V3 with ST DEPRESSION in V4 V6 (ANTERIOR MI competing with POSTERIOR MI)
- LEFT ANTERIOR FASCICULAR BLOCK PATTERN

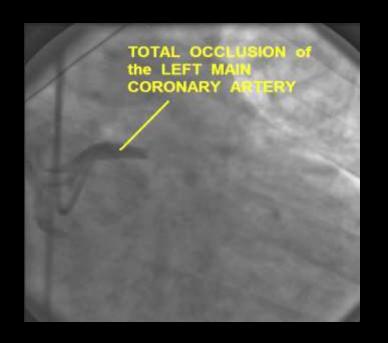
ST SEGMENT ELEVATION

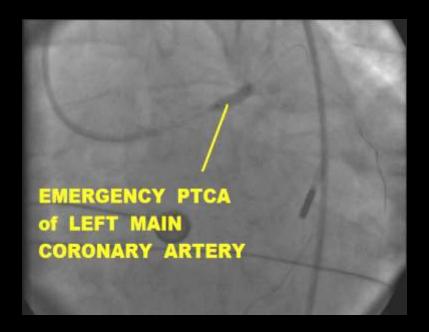
ST SEGMENT DEPRESSION



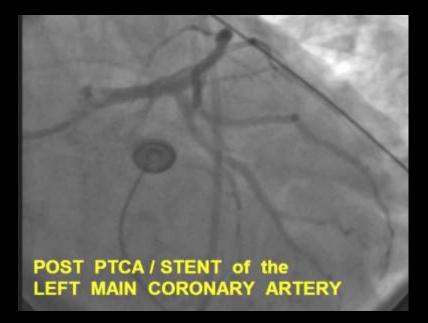








Despite the dismal mortality rate associated with STEMI from total LMCA occlusion, this patient survived and was later discharged. His EF is estimated at approximately 30%. He received an ICD, and is currently stable.



CASE STUDY 4: CRITICAL DECISIONS SCENARIO

CONCLUSIONS:

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

CATHETERIZATION for EMERGENCY PCI?

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

and the Interventional Cardiologist correctly identified the EKG patterns

of LMCA occlusion.

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

TO BE SENT TO THE CATH LAB FIRST?

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

THROMBOLYTIC THERAPY would achieve the FASTEST ROUTE to REPERFUSION --

-- by at least 60 minutes.

CASE STUDY 7 - STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

RISK FACTOR PROFILE:

6 CURRENT CIGARTTE SMOKER x 18 YEARS

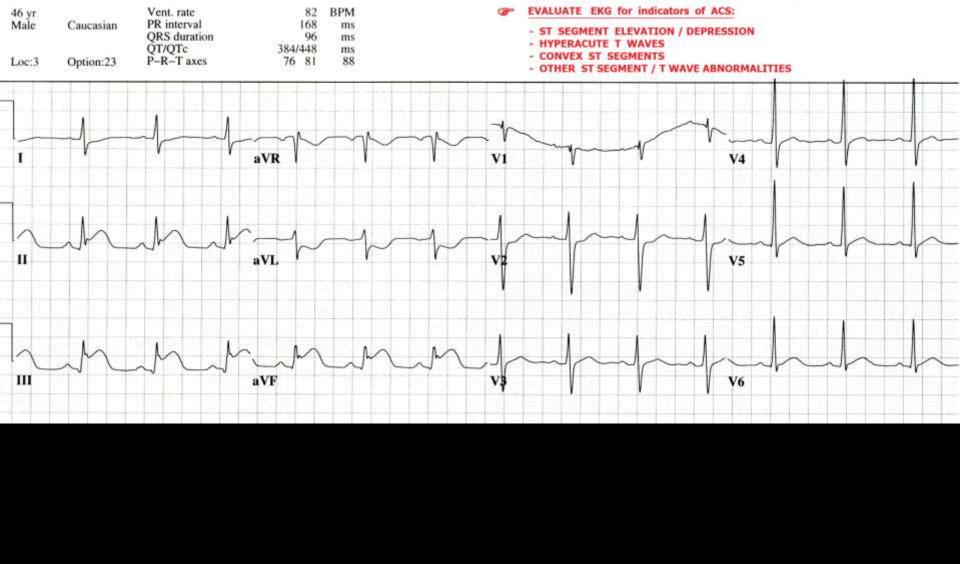
6* HYPERTENSION

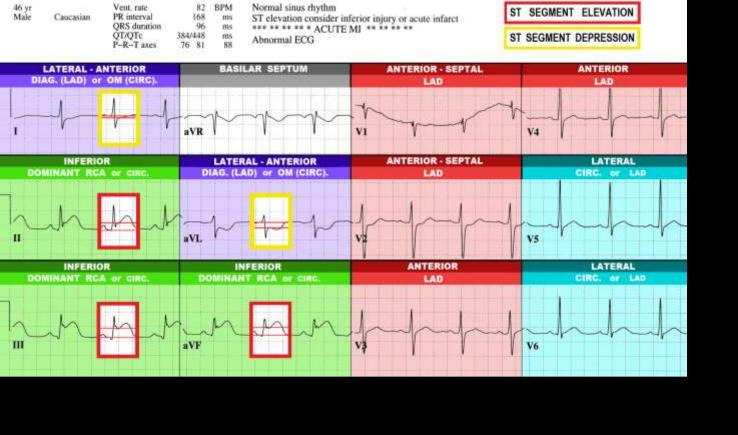
●* HIGH LDL CHOLESTEROL

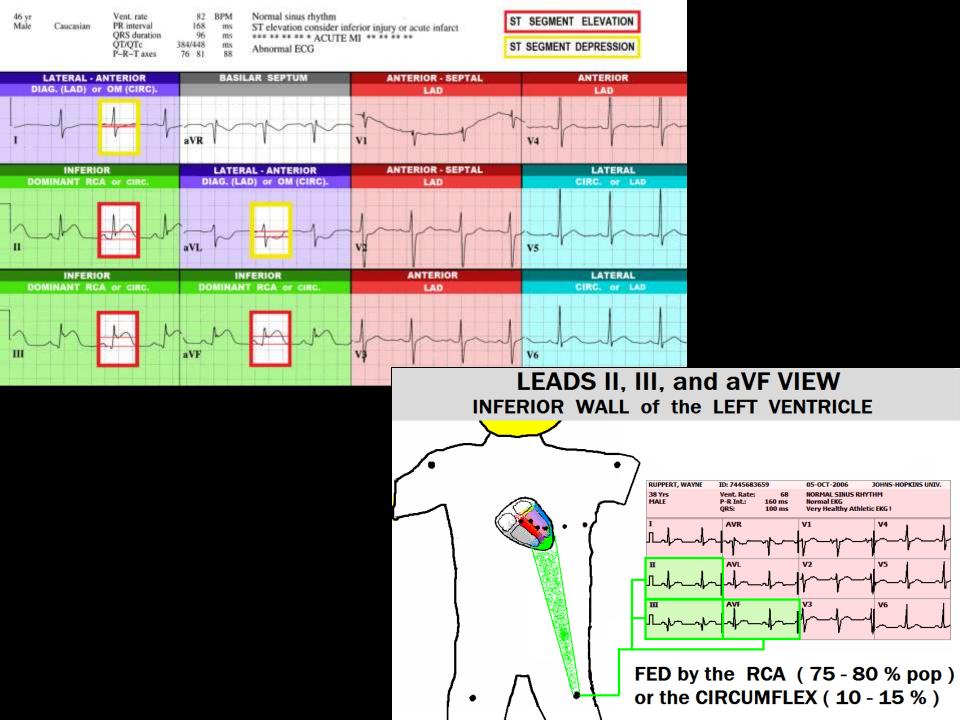
PHYSICAL EXAM: Patient is alert & oriented x 4, skin warm, dry, color normal. Non-anxious Lungs clear, normal S1, S2. No JVD, No ankle edema.

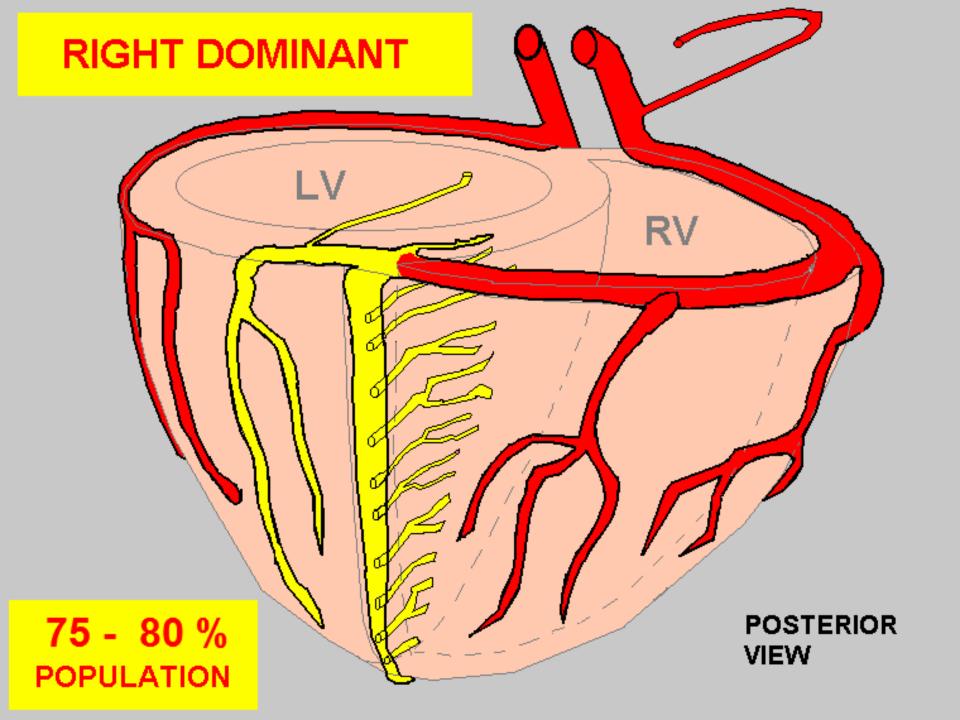
VITAL SIGNS: BP: 136/88 P: 88 R: 20 SAO2: 100% on 4 LPM O2

LABS: TROPONIN: < .04

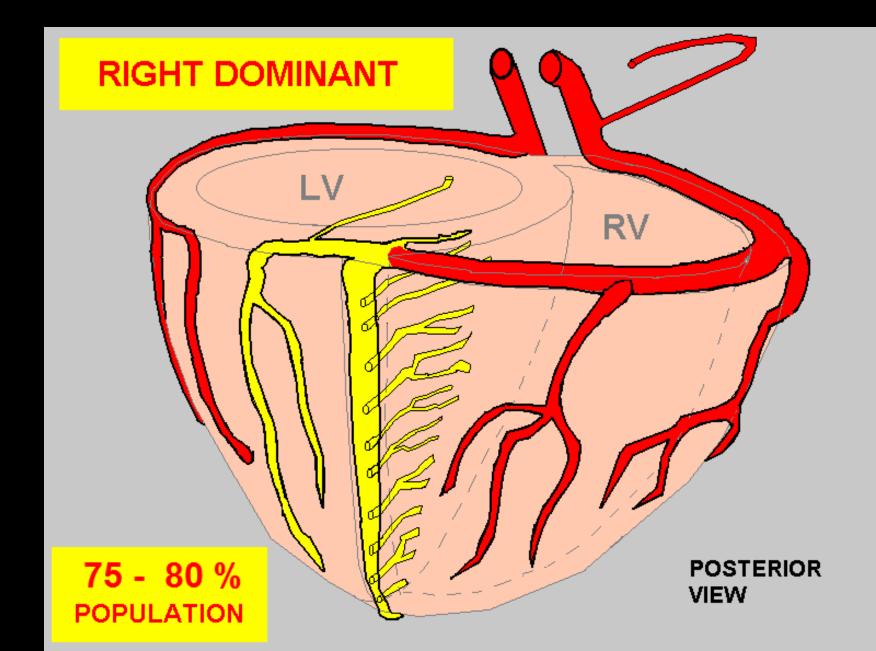








TEST QUESTION #27



The MANY FACES of INFERIOR MI ...

INFERIOR WALL MIs can range from being "MILD" (many are) to being MORE DEADLY than a "typical" ANTERIOR WALL MI...

UNDERLYING VASCULATURE THAT SERVES THE INFERIOR WALL CAN VARY GREATLY

The MANY FACES of INFERIOR MI ...

□ ► INFERIOR
 □ ► □ INFERIOR-RV
 □ ► □ INFERIOR-POSTERIOR
 □ ► □ INFERIOR-POSTERIOR
 □ ► □ INFERIOR-RV
 -POSTERIOR

INFERIOR - LATERAL

INFERIOR - LATERAL

POSTERIOR - RV

- POSTERIOR

. . . . and more !!

2 6661

S 242424

The MANY FACES of INFERIOR MI ...

INFERIOR - LATERAL
- POSTERIOR
INFERIOR - LATERAL
- POSTERIOR - RV

* BASED ON AUTHOR'S PERSONAL OBSERVATIONS

*FREQUENCY OF OCCURRENCE

INFERIOR

INFERIOR-RV

INFERIOR-RV

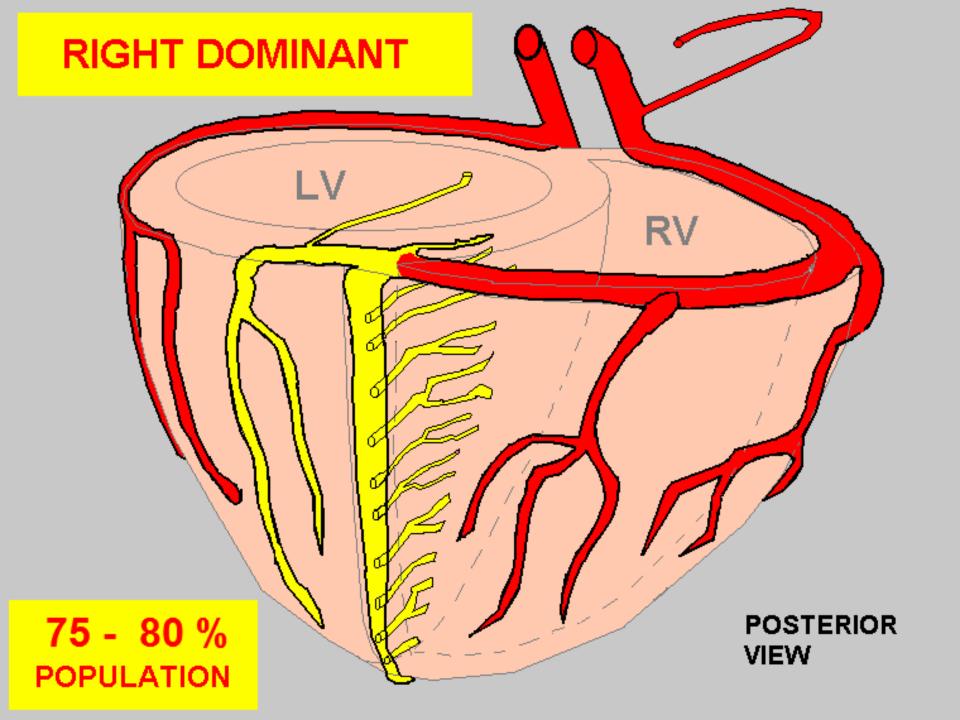
-POSTERIOR

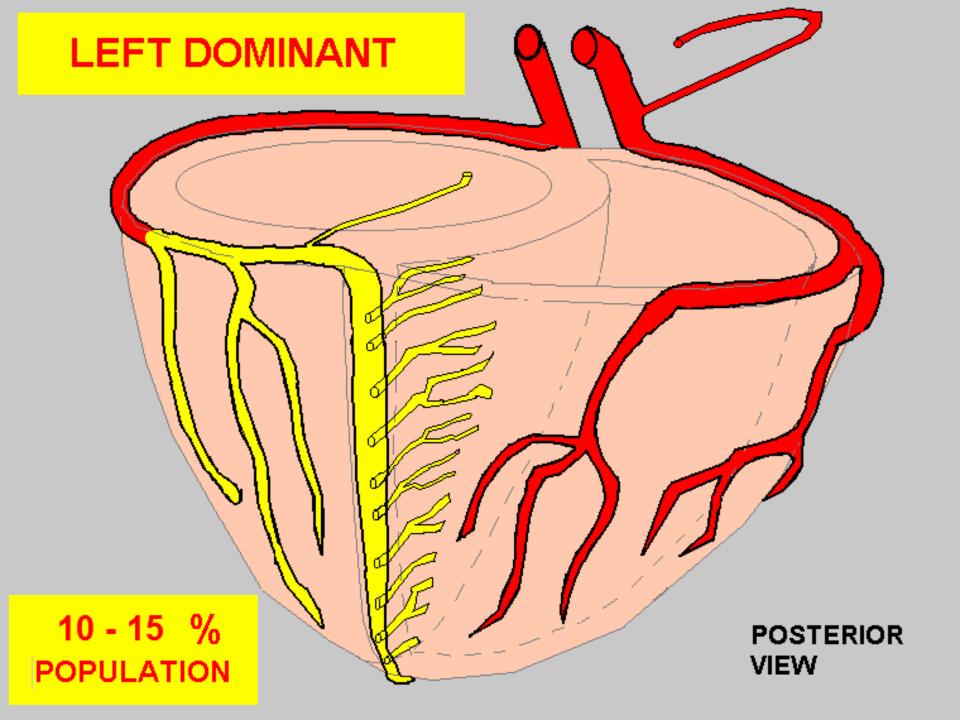
INFERIOR-POSTERIOR

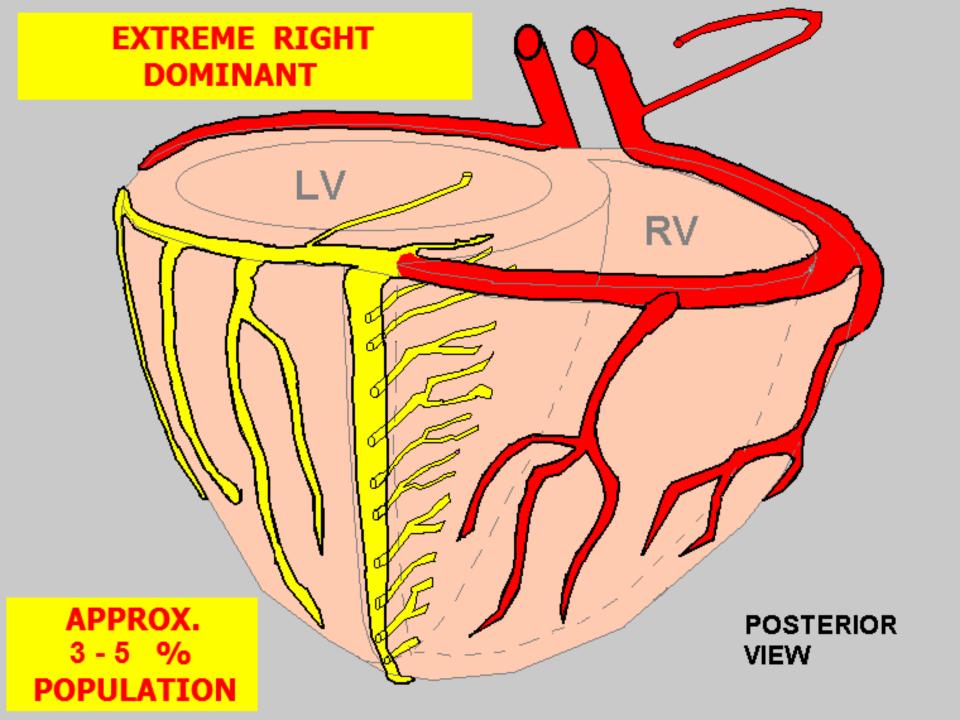
COMMON

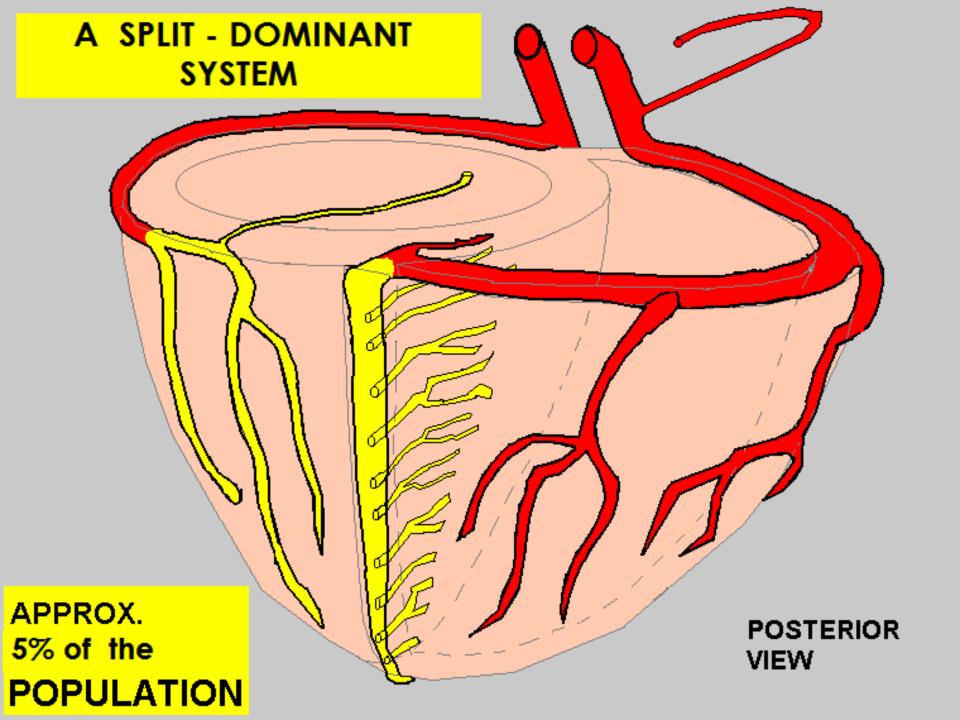
COMMON

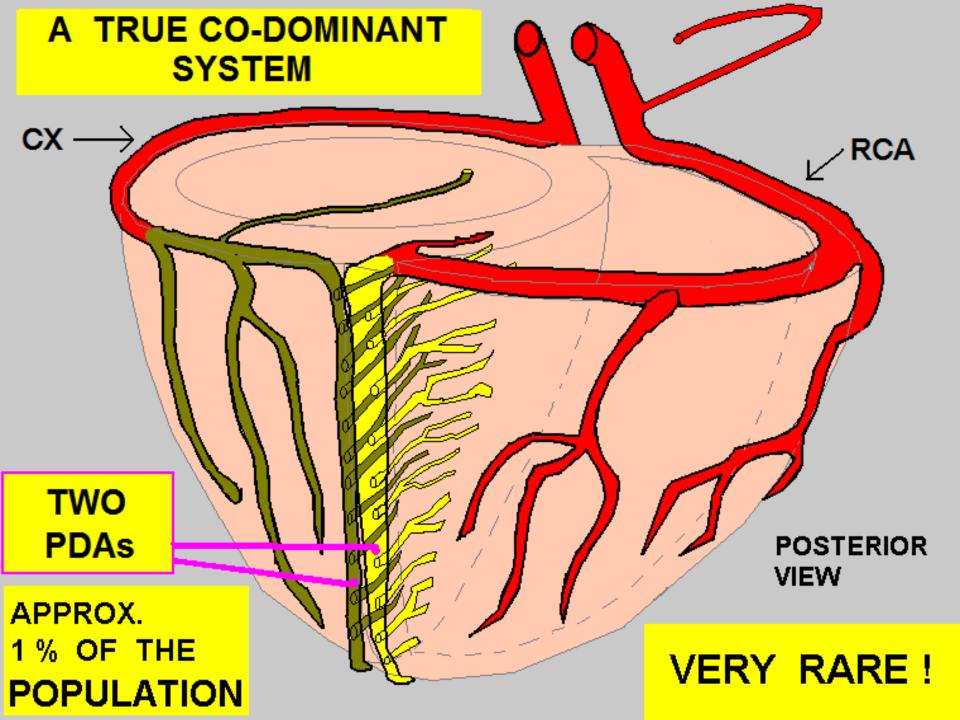
LESS COMMON











The MANY FACES of INFERIOR MI ...

INSTEAD OF PERFORMING THE

3 STEPS METHOD

YOU CAN OBTAIN AN

18 LEAD EKG.

IT WILL PROVIDE YOU WITH MORE DATA, AND HAS BETTER SPECIFICITY....

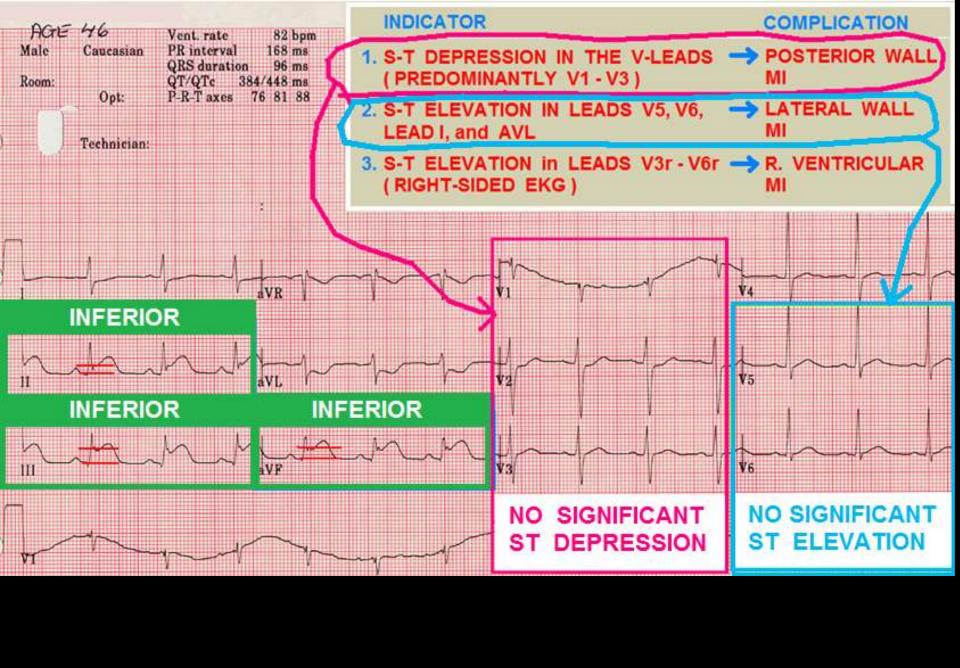
The MANY FACES of INFERIOR MI ...

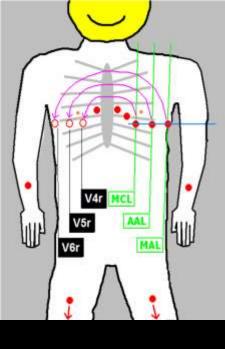
WHEN YOU OBSERVE AN INFERIOR WALL MI (S-T ELVATION LEADS II, III, and AVF)... ALWAYS LOOK FOR THE FOLLOWING INDICATORS TO ASSESS THE EXTENT OF THE MI:

INDICATOR

COMPLICATION

- 1. S-T DEPRESSION IN THE V-LEADS -> POSTERIOR WALL (PREDOMINANTLY V1 V3) MI
- 2. S-T ELEVATION IN LEADS V5, V6, -> LATERAL WALL LEAD I, and AVL
- 3. S-T ELEVATION in LEADS V3r V6r → R. VENTRICULAR (RIGHT-SIDED EKG) MI



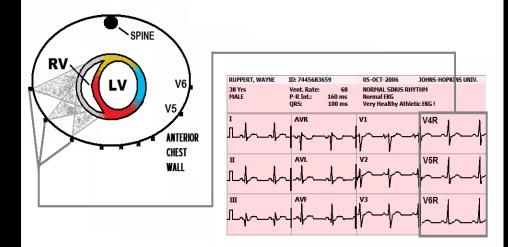


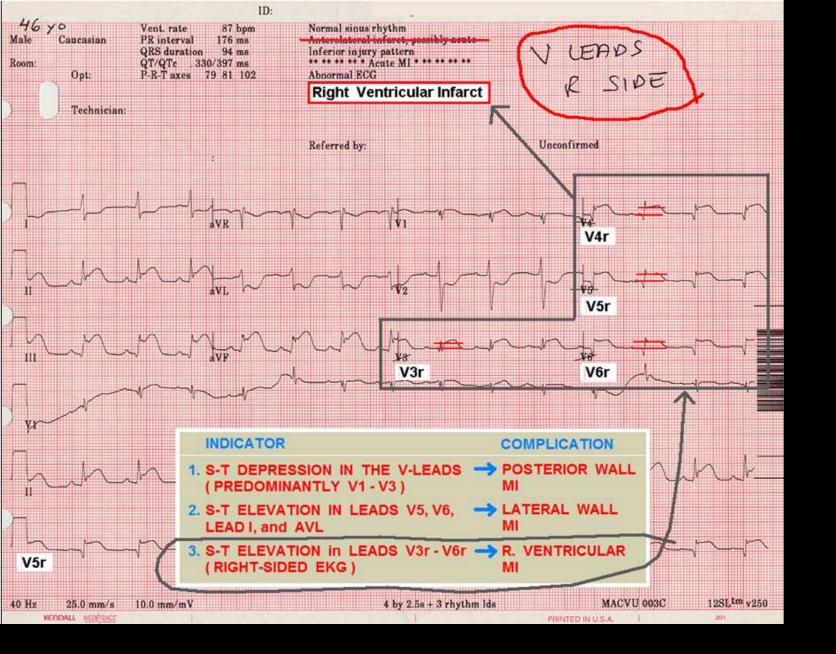
To do a RIGHT - SIDED EKG . .

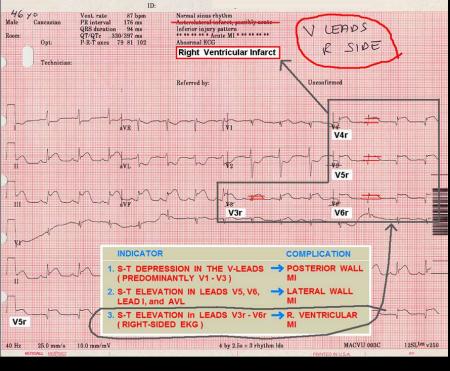
MOVE leads V4, V5, and V6

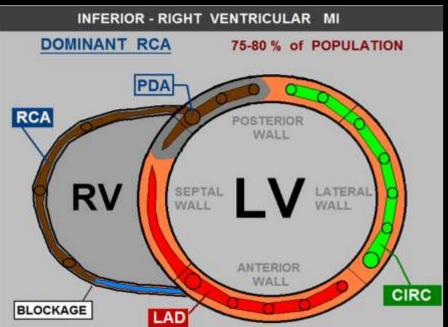
to the corresponding placement on the RIGHT SIDE of patient's chest . . .

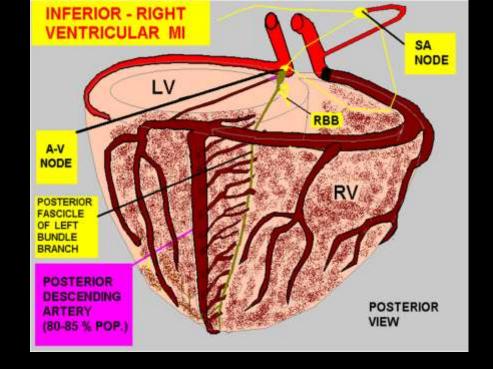
V4R - V6R VIEW THE RIGHT VENTRICLE

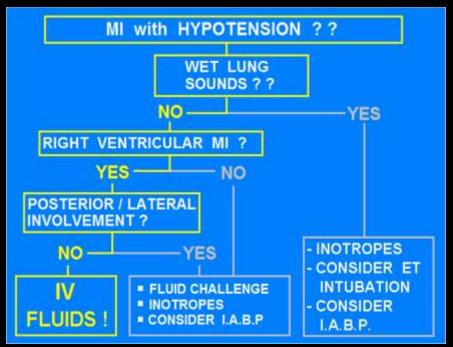


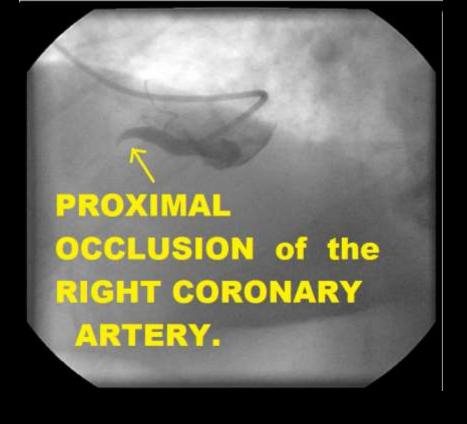


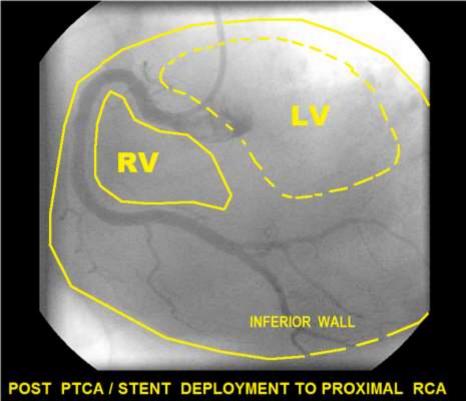












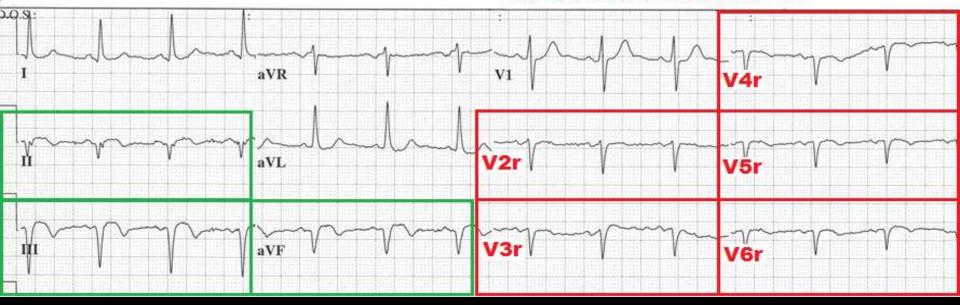
CAS	SE STUDY SUMMAR	RY \					
ST ELEVAT	ON: II, III, aVF , V4R -	V6R	ST DEPRESSION:	aVL			
SUSPECTED DIAGNOSIS: ACUTE INFERIOR - RIGHT VENTRICULAR WALL MI							
SUSPECTED "CULPRIT ARTERY" (if applicable):							
RIGHT CORONARY ARTERY - DOMINANT							
IMMEDIATE CONCERNS FOR ALL STEMI PATIENTS:							
	PARED TO MANAGE SUDDEN CA	the second of Assessed to the San San		COLUMN TO SERVICE STATE OF THE PROPERTY OF THE	The state of the s		
 STAT REPERFUSION THERAPY: THROMBOLYTICS vs. CARDIAC CATHETERIZATION and PCI CONSIDER NEEDS FOR ANTI-PLATELET and ANTI-COAGULATION THERAPY 							
CRITICAL S	TRUCTURES COMPROMISED:	POTENTIAL CO	MPLICATIONS:	Poss	IBLE CRITICAL INTERVENTIONS:		
6 ³⁶ 15-29 MAS	5% OF THE LV MUSCLE -		OSSIBILITY OF FAILURE.		FLUID CHALLENGE NOTROPIC AGENTS		
	OF THE RIGHT		S AND OPIATES	-	FLUID BOLUSES		
	S NODE ARTERY ————————————————————————————————————	BRADYCA ASYSTOL			ATROPINE TRANSCUTANEOUS PACING		
BY D	ODAL ARTERY SUPPLIED — OMINANT ARTERY (RCA or IN MOST PATIENTS	- 1 DEGRE	REE type I, II		ATROPINE TRANSCUTANEOUS PACING		

64 yr 79 BPM Vent. rate Male PR interval Caucasian 136 ms QRS durationms QT/QTc 350/401 ms Loc:3 Option:23 P-R-T axes 42 -41

ECG LEADS PLACED ON RIGHT CHEST WALL.

ECG INDICATORS of EVOLVING INFERIOR - RIGHT VENTRICULAR MYOCARDIAL INFARCTION:

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



CASE STUDY 9 - STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

RISK FACTOR PROFILE:

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

PULSE:

HIGH LDL CHOLESTEROL

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

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

LABS: TROPONIN: < .04

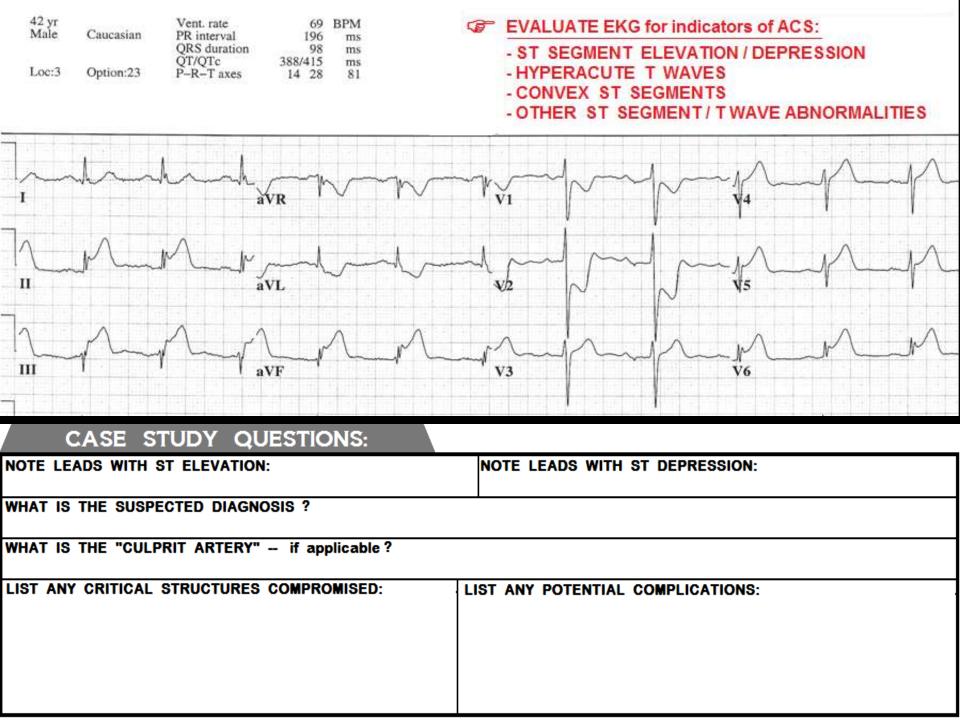
SHOCK ASSESSMENT

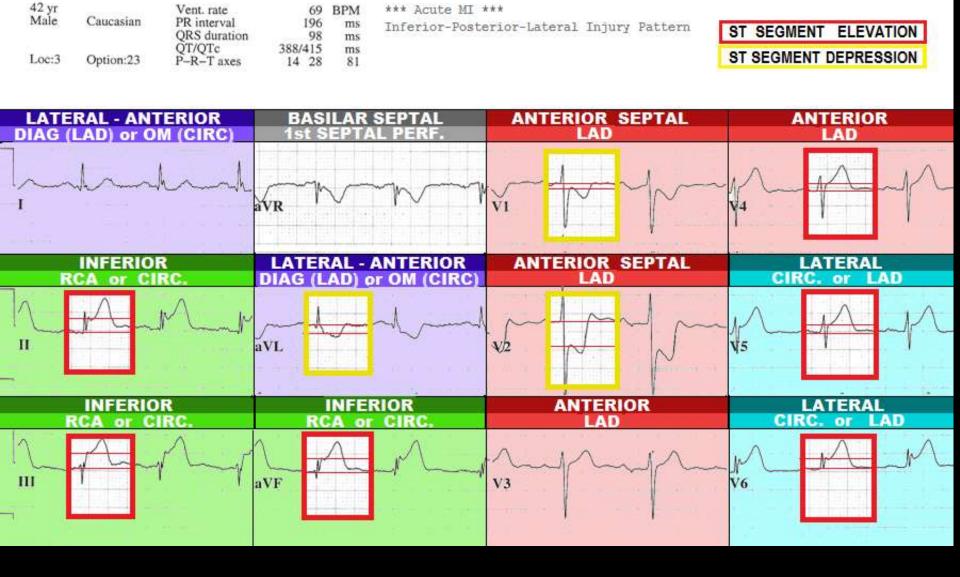
LOC:	ANXIOUS	AWAKE
	RESTLESS	ALERT &
	LETHARGIC	ORIENTED
	UNCONSCIOUS	A production of the second of the second
	UNCONSCIOUS	

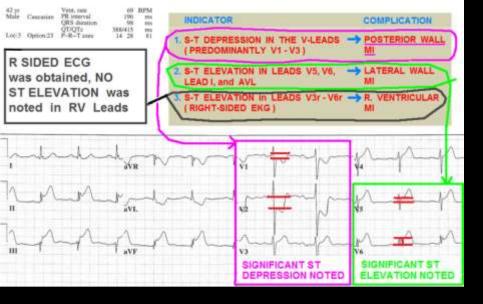
BREATHING: TACHYPNEA NORMAL

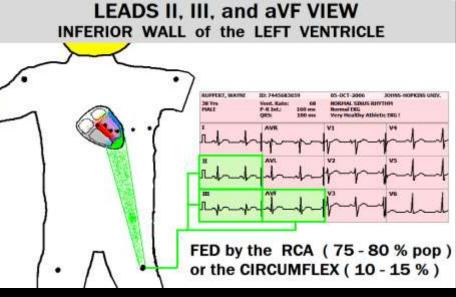
WEAK / THREADY STRONG
TOO FAST or SLOW

STATUS: SHOCK NORMAL



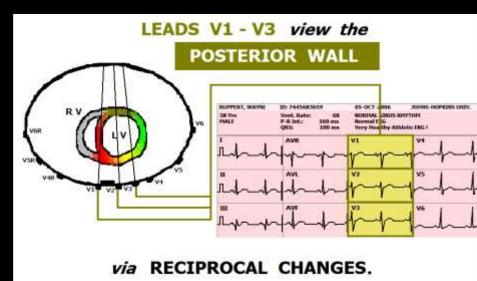


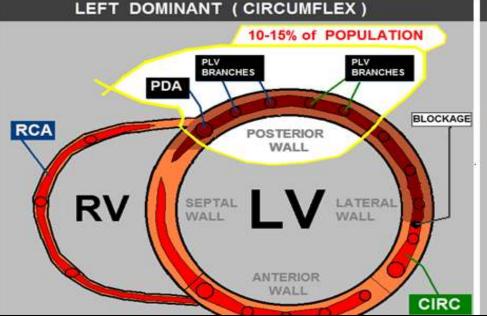


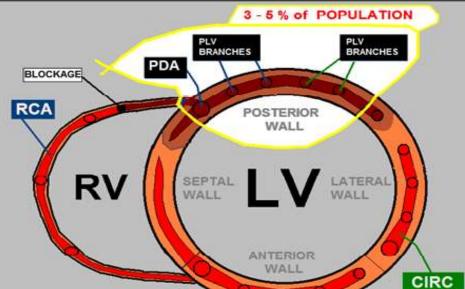


V5 - V6 VIEW THE LATERAL WALL of the LEFT VENTRICLE RUPPERT, WAYNE ID: 7445603659 U5-OCT-2006 NONIS-HOPERS UNIV. 30 Yes Vend. Blate: 58 HORMAL SDUIS-RIPPTHIP HOLE P-R Int.: 160 ons (Prop. 100 ons (Pro

ANTERIOR CHEST WALL



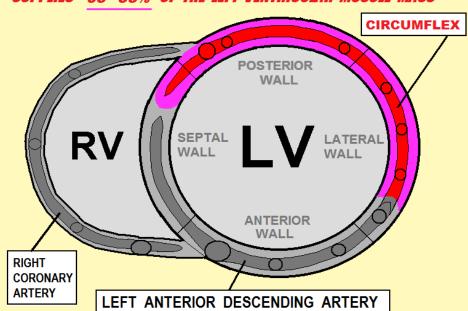




"EXTREME RIGHT DOMINANT" RCA

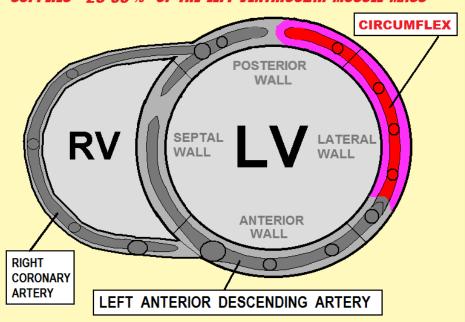
The DOMINANT CIRCUMFLEX ARTERY...

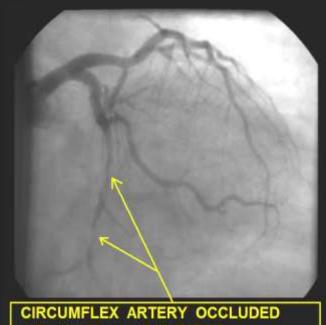
SUPPLIES 35 - 55% OF THE LEFT VENTRICULAR MUSCLE MASS



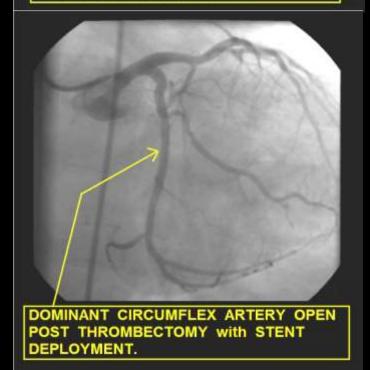
The NON - DOMINANT CIRCUMFLEX ARTERY

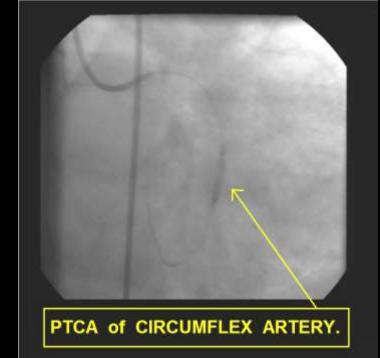
SUPPLIES 25-30 % OF THE LEFT VENTRICULAR MUSCLE MASS

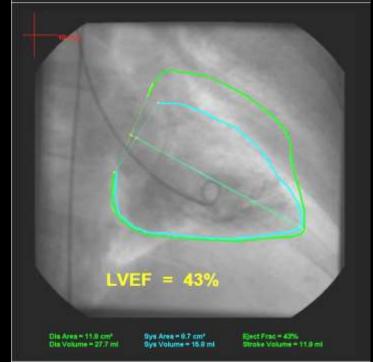




CIRCUMFLEX ARTERY OCCLUDED with significant THROMBUS.







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

ACUTE PAPILLARY MUSCLE -

REGURGITATION (7 - 10 DAYS)

TEAR and MITRAL VALVE

INOTROPIC AGENTS

EMERGENCY SURGERY

DIEURETICS

SIGNIFICANT AMOUNT of .

INSERTION to BASE of LV

PAPILLARY MUSCLE

CASE STUDY 10 - STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

48 y/o FEMALE arrives via EMS, c/o "EXCRUCIATING HEAVINESS" in center of chest, X 1 hour. She also c/o nausea (vomited several times). Per EMS, she experienced 4 episodes of V-Fib, was defibrillated successfully each time. Amiodarone drip is running @ 1mg/min.

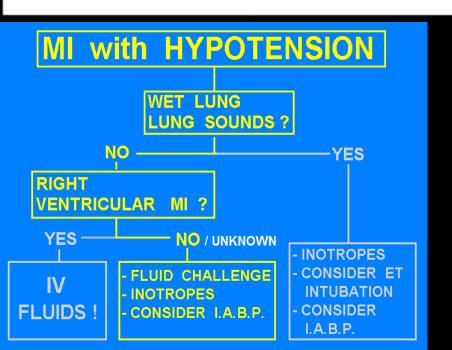
RISK FACTOR PROFILE:

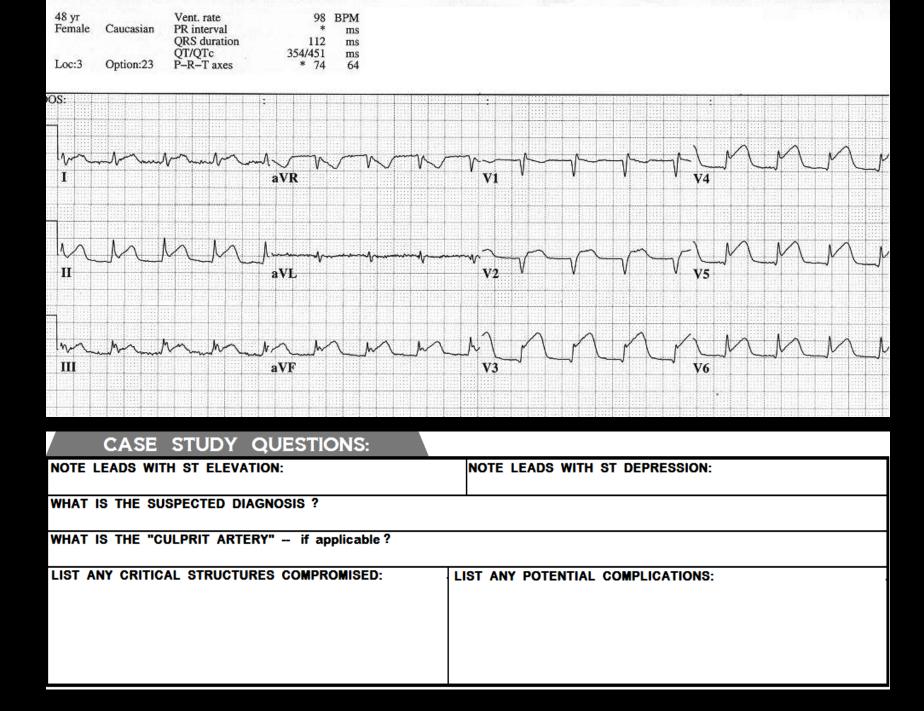
- CIGARETTE SMOKER
- HYPERTENSION
- BROTHER HAD AMI AT AGE 44

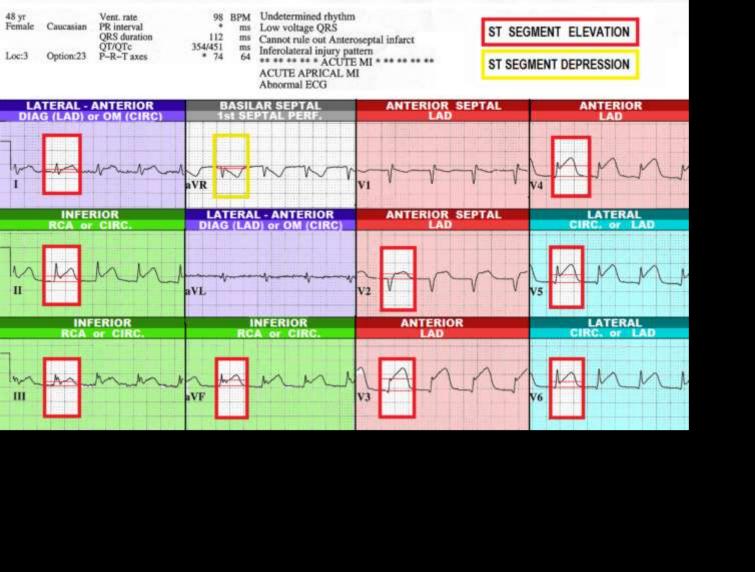
PHYSICAL EXAM: CAO x 4, anxious, SKIN cool, pale, diaphoretic. Lung sounds: clear Heart Sounds normal S1, S2,

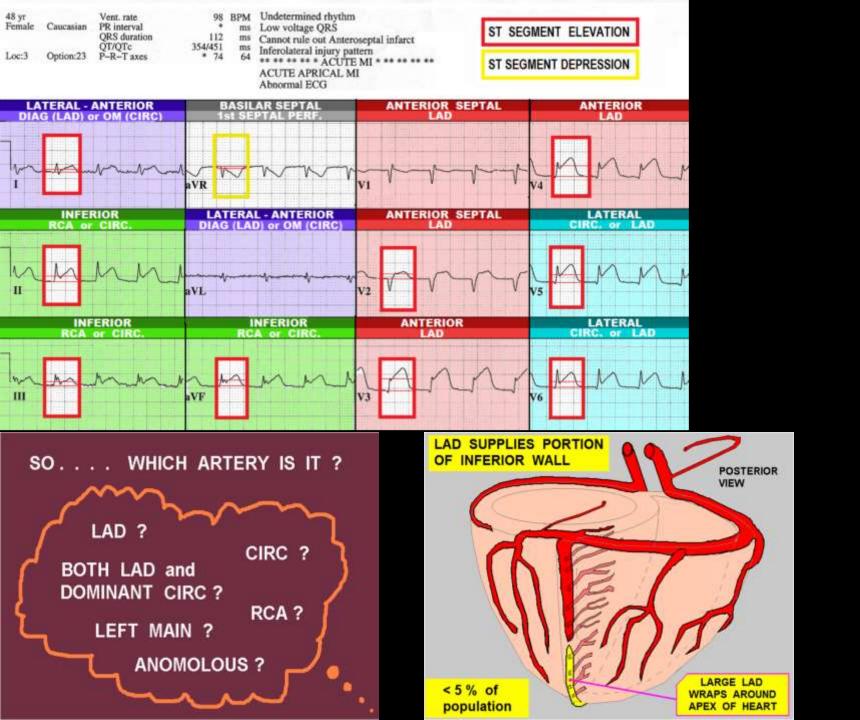
VITAL SIGNS: BP: 78/56 P: 100 R: 28 SAO2: 94% on 4 LPM O2

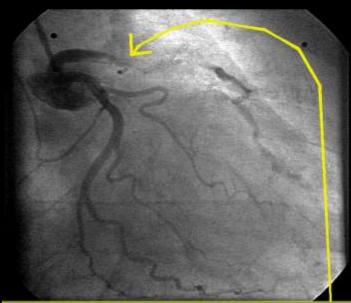
LABS: TROPONIN: < .04



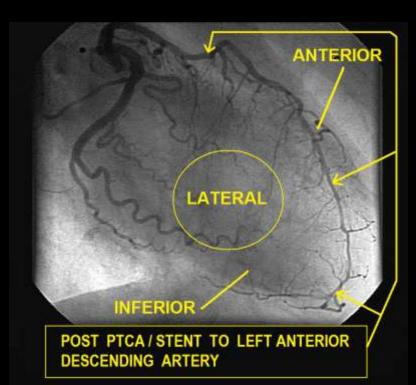


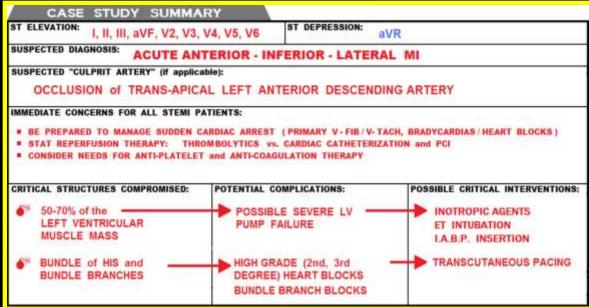






PRE-INTERVENTION IMAGE. PROXIMAL OCCLUSION of the LEFT ANTERIOR DESCENDING ARTERY.





EVOLVING STEMI:

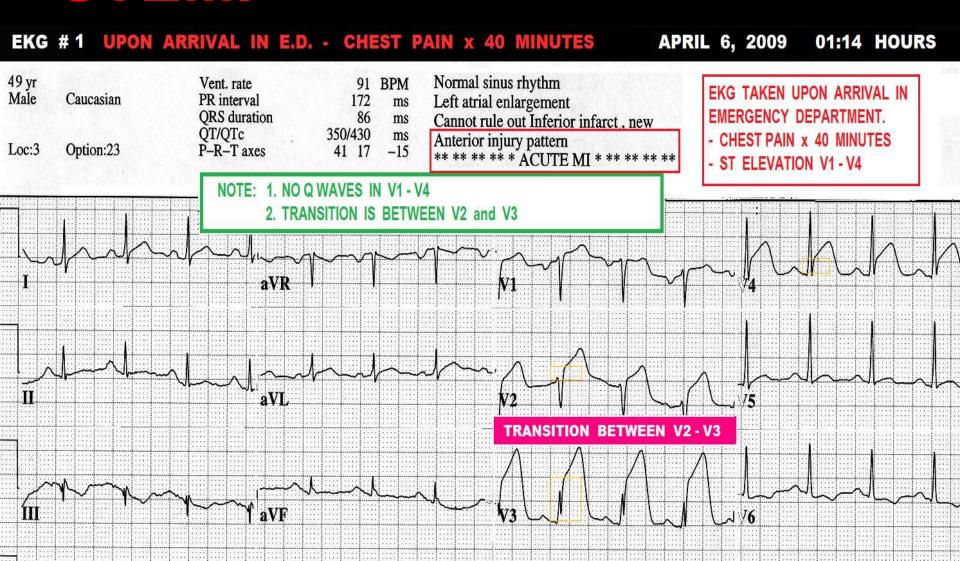
- -ST SEGMENTS DROP
- **-Q WAVES FORM**
- -R WAVE PROGRESSION CHANGES

IN PRECORDIAL LEADS.

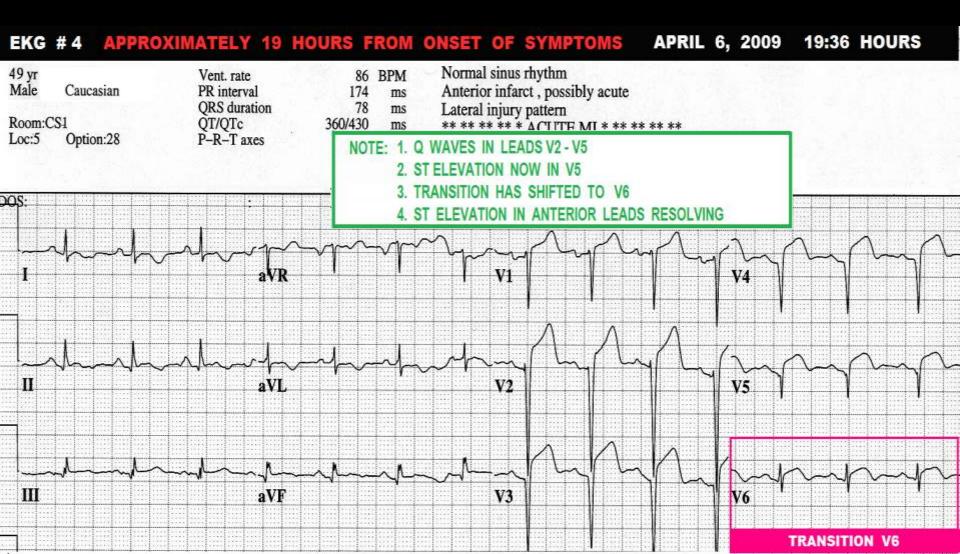
Q WAVE RULES - SUMMARY:

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

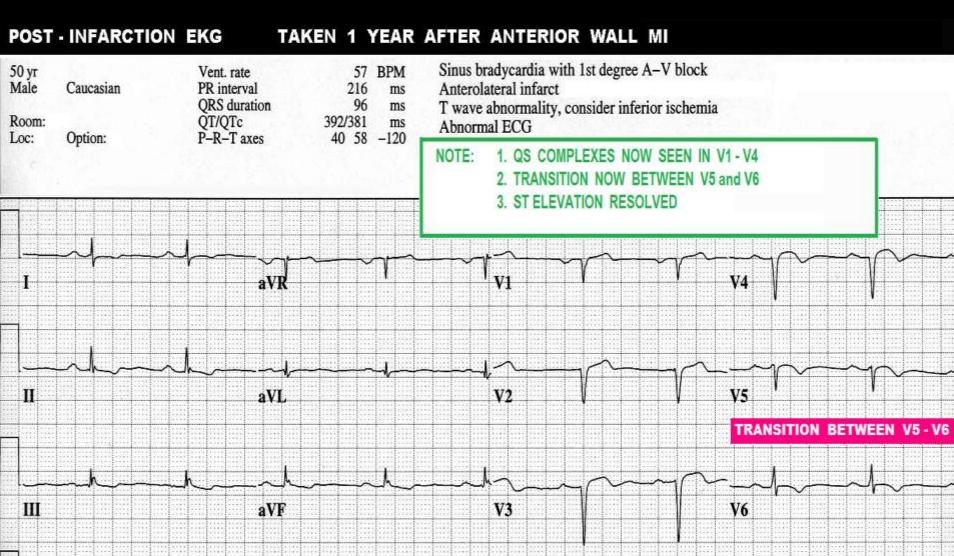
ACUTE ANTERIOR WALL STEMI



EVOLVING ANTERIOR WALL STEMI



FULLY EVOLVED ANTERIOR WALL MI





"NOWHERE", NEW MEXICO, 1994

BRUGADA SYNDROME and Other Inforction Mimies

CASE STUDY 18 -- BRUGADA SYNDROME

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

37 y/o FEMALE patient arrives via EMS after being involved in a low speed motor vehicle accident. Per EMS crew, patient was the driver and sole occupant of a car that struck a tree. Patient does not recall accident. Upon further questioning, patient admits to other episodes of syncope and near-syncope. Patient denies feeling any chest pain / pressure or shortness of breath. She states she "felt great" today, until just before the the accident, when she "suddenly felt lightheaded and must have blacked-out."

RISK FACTOR PROFILE:

FAMILY HISTORY: MATERNAL AUNT DIED AT AGE 31, UNEXPECTEDLY. WAS RULED AS A "HEART ATTACK." THERE WAS NO PRIOR KNOWN HISTORY OF CAD.

PHYSICAL EXAM: Pt. CAO x 3, skin warm, dry, color normal. Abrasions /contusions on face (airbag deployment). Patient appears to be in excellent physical condition, states she exercises several times per week (aerobics, weight training, swimming).

VITAL SIGNS: BP: 112/66, P: , R: 20, SAO2: 100% on room air.

LABS: TROPONIN: < .04 BMP and CBC: all values within normal limits.

IS THERE ANYTHING ABNORMAL WITH THIS EKG?

37 yr Female Vent. rate 62 BPM Caucasian PR interval 180 QRS duration 88 Room:C4A QT/QTc P-R-T axes 418/424 Option:23 Loc:3 37 22

Normal sinus rhythm Normal ECG No previous ECGs available

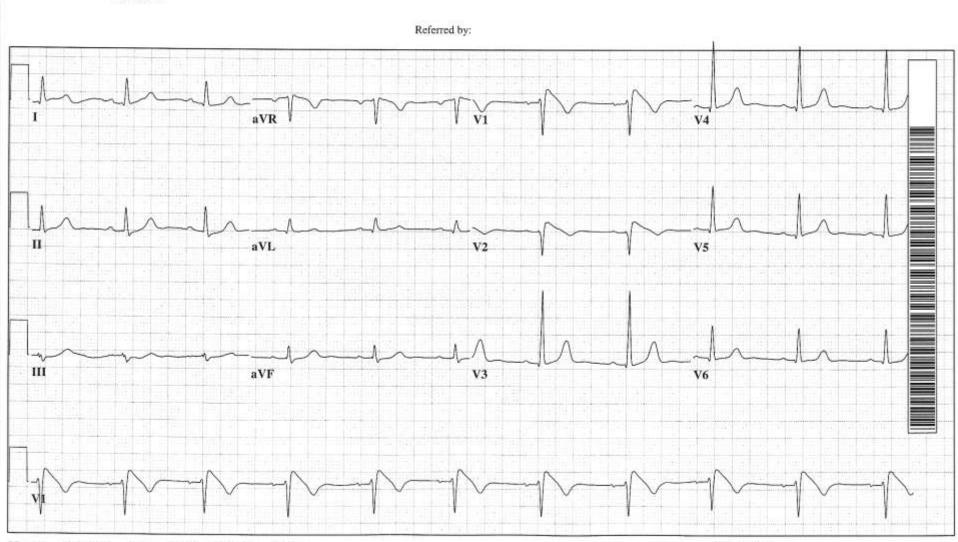
ms

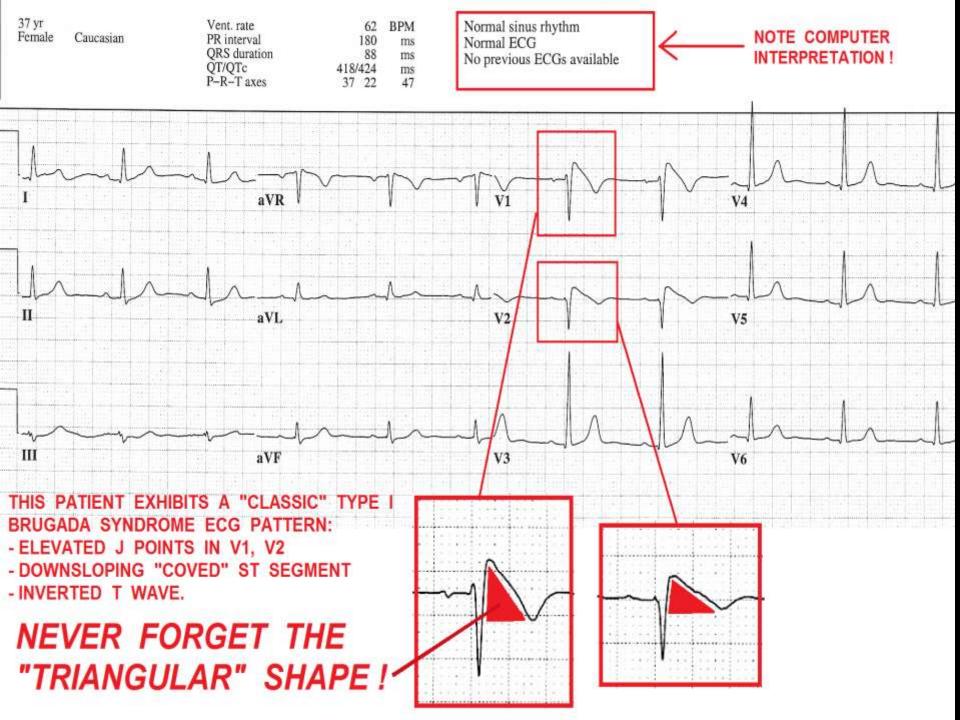
ms

ms

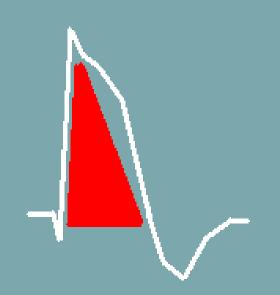
47

Technician:





PATTERNS of S-T ELEVATION:



BEWARE of the

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

THINK - -



BRUGADA SYNDROME

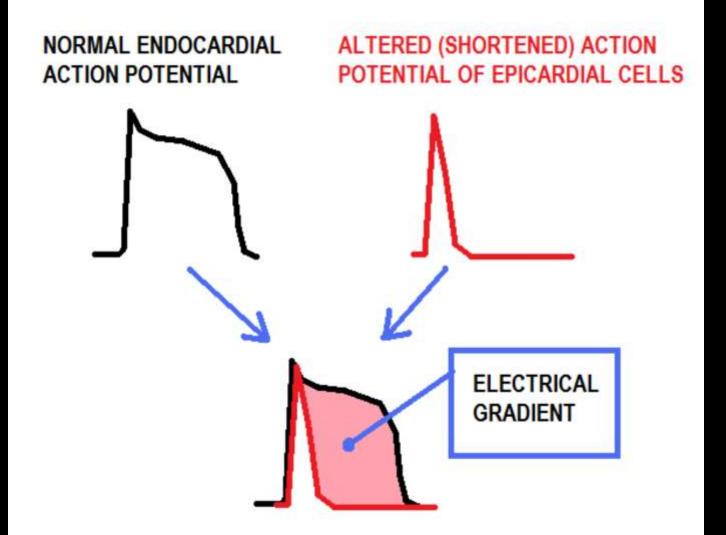


- 1. RBBB PATTERN
- 2. S-T ELEVATION V1, V2, possibly V3
- 3. ATYPICAL "TRIANGLE" SHAPED S-T SEGMENT

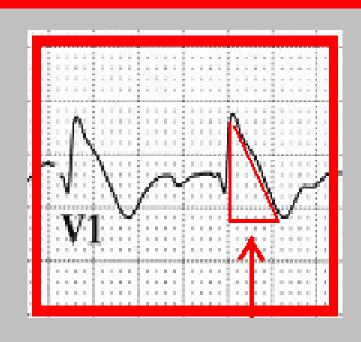


- 4. USUALLY EFFECTS YOUNG, HEALTHY PEOPLE
- 5. CAUSES SUDDEN DEATH by TORSADES

MECHANISM OF PHASE 2 RE-ENTRY IN BRUGADA SYNDROME

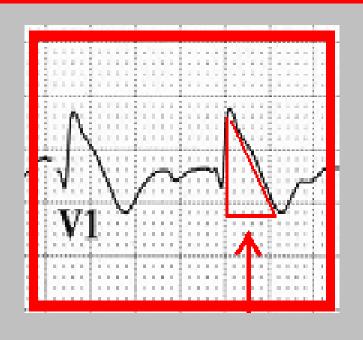


- GENETIC DISORDER GENE SCN5A, which encodes
 CARDIAC SODIUM CHANNELS.
- CAUSES EARLY RIGHT VENTRICULAR SUB-EPICARDIAL REPOLARIZATION



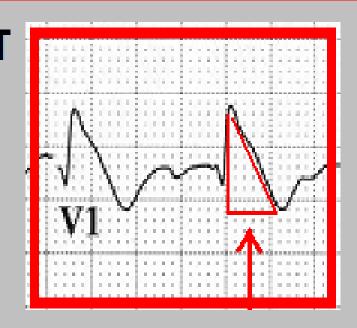
- CAUSES RUNS OF TORSADES de POINTES, and SUDDEN DEATH from TORSADES and V-FIB.
- IS BELIEVED TO CAUSE 4 12 % of ALL SUDDEN DEATHS, and 50 % of ALL CARDIAC DEATHS where pt. has a STRUCTUALLY NORMAL HEART.

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



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

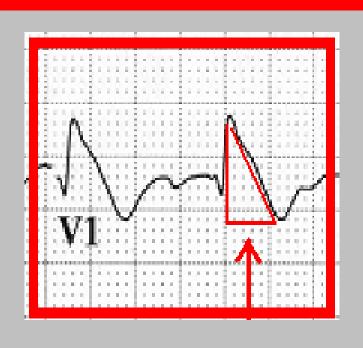
- YOUNG MALES of SOUTHEAST ASIAN DESCENT are in HIGH RISK GROUP, however this disorder affects ANY RACE or GENDER.



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

BRUGADA SYNDROME - TESTING

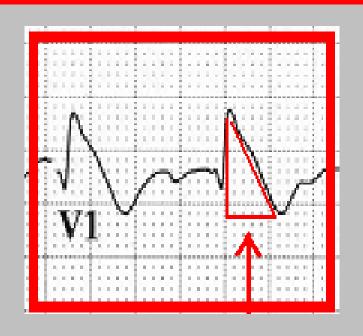
- For CONCEALED cases, a drug study of AJMALINE, FLECAINIDE, or PROCAINAMIDE can UNMASK the "tell-tale" TRIANGULAR COMPLEXES of V1 and V2.



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

BRUGADA SYNDROME - TREATMENT

ICD implantation is the only known effective treatment to date.



www.BRUGADA.org



 Vent. rate
 129
 BPM

 PR interval
 * ms

 QRS duration
 112 ms

 QT/QTc
 398/583 ms

 P-R-T axes
 * 121 -2

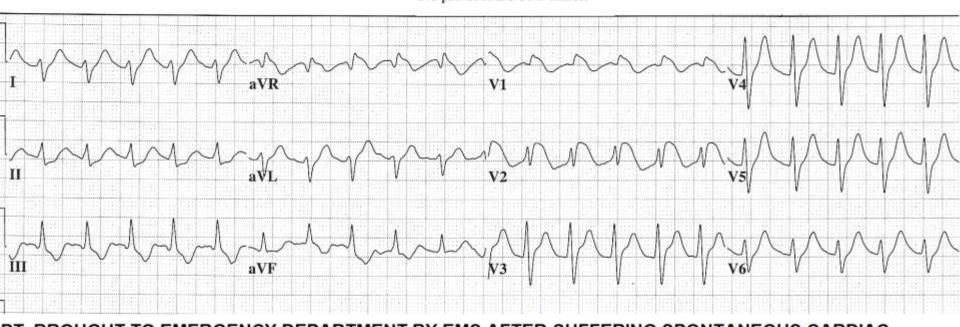
Undetermined rhythm

Incomplete right bundle branch block Right ventricular hypertrophy

ST elevation consider anterior injury or acute infarct

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

Abnormal ECG No previous ECGs available



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

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

VISIT: www.BRUGADA.org FOR MORE INFORMATION.

42 y/o FEMALE

 Vent. rate
 86
 BPM

 PR interval
 200
 ms

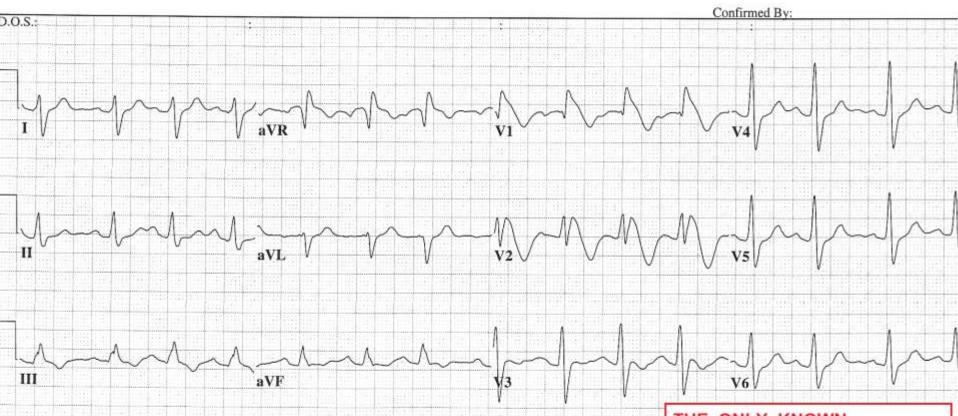
 QRS duration
 148
 ms

 QT/QTc
 414/495
 ms

 P-R-T axes
 64
 114
 17

Normal sinus rhythm with sinus arrhythmia Right bundle branch block ST elevation consider anterior injury or acute infarct ** ** ** * ACUTE MI * ** ** **

Abnormal ECG No previous ECGs available



BRUGADA SYNDROME.

PATIENT HAD HISTORY of SYNCOPE of UNKNOWN ETIOLOGY. FAMILY HISTORY of SUDDEN DEATH of YOUNG, HEALTHY ADULTS.

VISIT: www.BRUGADA.org FOR MORE INFORMATION!

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

41 y/O I LIVIALI	41	y/o	FEMALE
------------------	----	-----	--------

 Vent. rate
 137
 BPM

 PR interval
 116
 ms

 QRS duration
 162
 ms

 QT/QTc
 308/465
 ms

 P-R-T axes
 69
 50
 58

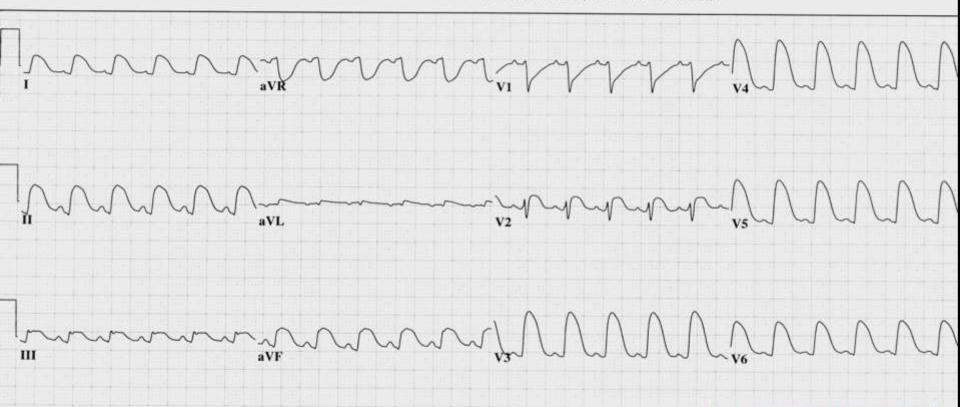
Sinus tachycardia

Non-specific intra-ventricular conduction block Abnormal ECG

When compared with ECG of 05-MAR-2008 13:35,

QRS duration has increased

ST elevation now present in Inferior leads ST elevation now present in Anterior leads

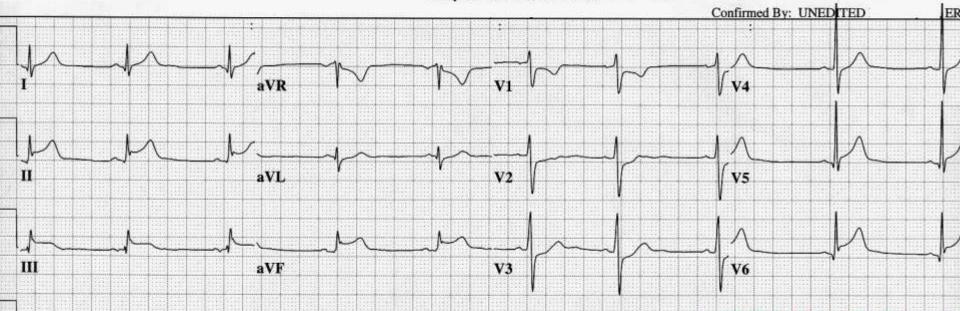


ACUTE BACTERIAL MYOCARDITIS.
INTENSE FLU-LIKE SYMPTOMS x 4-5 DAYS.
SUDDEN ONSET OF SUBSTERNAL CHEST
PRESSURE with SHORTNESS OF BREATH

EJECTION FRACTION BY ECHOCARDIOGRAM = 10%

23 y/o MALE: Vent. rate 56 BPM **UNEDITED COPY - REPORT IS COMPUTER GENERATED ONLY, WITHOUT 128 PR interval ms PHYSICIAN INTERPRETATION ORS duration -96 ms Sinus bradycardia with sinus arrhythmia QT/QTc 410/395 ms ST elevation consider inferolateral injury or acute infarct
** ** ** * ACUTE MI * ** ** ** P-R-T axes 23 66 47

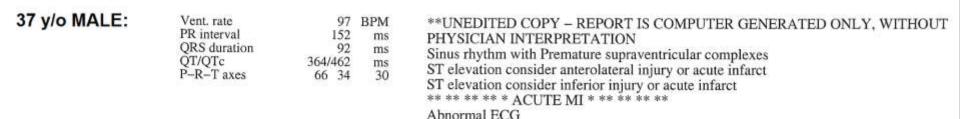
> Abnormal ECG No previous ECGs available

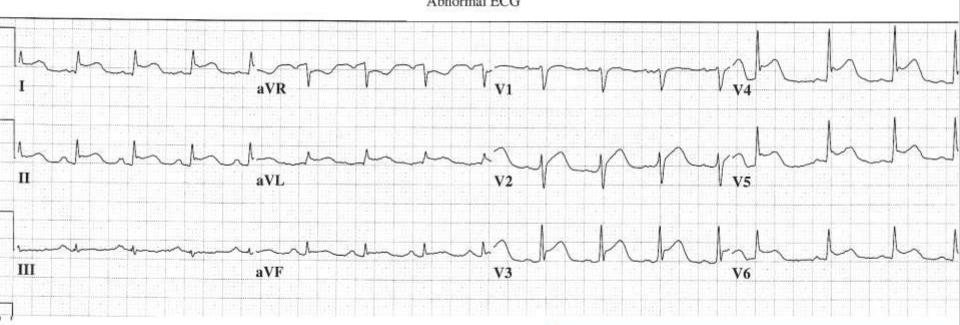


ACUTE PERICARDITIS.

SHARP SUBSTERNAL CHEST PAIN x 1 DAY, HAD VIRAL SYMPTOMS with MOUTH ULCERS x 3 DAYS. CHEST PAIN INCREASES WITH DEEP INSPIRATION.

TESTED POSITIVE FOR COXSACKIE A and B VIRUS

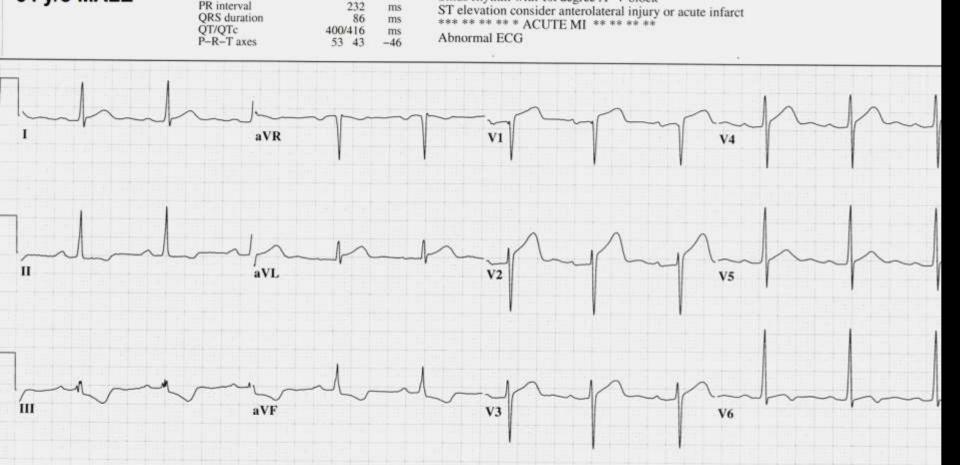




ACUTE PERICARDITIS.

SHARP CHEST PAIN x 1 DAY, PROGRESSIVELY INCREASED PAIN, INCREASES WITH DEEP INSPIRATION. RECENT HISTORY OF VIRAL SYMPTOMS.

NOTE: THIS PATIENT EXPERIENCED SUDDEN VENTRICULAR FIBRILLATION in the EMERGENCY DEPARTMENT, WAS DEFIBRILLATED x 1 WITH RETURN OF SINUS RHYTHM. RECOVERED FULLY.



Sinus rhythm with 1st degree A-V block

BPM

65

64 y/o MALE

Vent. rate

PR interval

Dx: EARLY REPOLARIZATION. PT. ASYMPTOMATIC HAD 7 EKGs SPANNING 11 YEARS WITH IDENTICAL FINDINGS. NOTE U WAVES V3 - V5.

54 y/o FE	M	А	ᄔ
-----------	---	---	---

 Vent. rate
 82
 BPM

 PR interval
 * ms

 QRS duration
 132 ms

 QT/QTc
 360/420 ms

 P-R-T axes
 159 31 21

*** AGE AND GENDER SPECIFIC ECG ANALYSIS ***

Unusual P axis, possible ectopic atrial rhythm with complete heart block and

Wide QRS rhythm

Non-specific intra-ventricular conduction block ST elevation consider inferior injury or acute infarct ST elevation consider anterior injury or acute infarct *** ** ** ** ACUTE MI ** ** ** ** ...

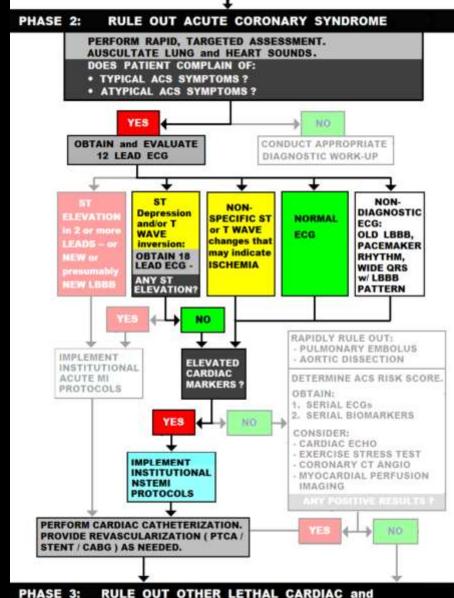


HYPERKALEMIA - K+ 8.6

Pt. FOUND UNRESPONSIVE BY FRIENDS. NO PRECIPITATING COMPLAINTS. Dx: ACUTE RENAL FAILURE.

Pt. EXPERIENCED CARDIAC ARREST, SUCCESSFUL RESUSCITATION with NaHCO3 100 mEq, CALCIUM CHLORIDE 1.0 gram, INSULIN 10 units, and DEXTROSE 25 gm. IV. DISCHARGED 11 DAYS LATER.





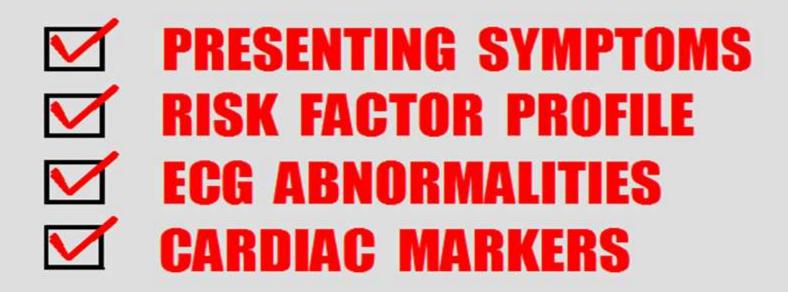
RULE OUT LIFE-THREATENING CONDITIONS

PHASE 1:

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

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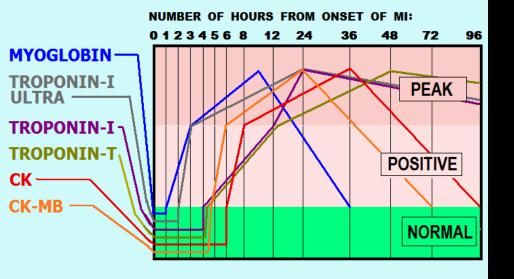
"The ACS Scorecard"



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

CARDIAC MARKERS

RISE - PEAK - NORMALIZE TIME APPROXIMATIONS



CARDIAC MARKERS

RISE - PEAK - NORMALIZE TIME APPROXIMATIONS

NUMBER OF HOURS FROM ONSET OF MI:

	RISES (POSITIVE)	- PEAKS	RETUR	
MYOGLOBIN	1 - 3	8 - 10	24 - 36	
TROPONIN-I ULTRA ———	2 - 3	10 - 24	5 - 10	days
TROPONIN-I	4 - 12	10 - 24	5 - 10	days
TROPONIN-T —	4 - 12	12 - 48	5 - 15	days
CK	6 - 8	24 - 36	3 - 4	days
СК-МВ ———	4 - 6	10 - 24	3	days

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HEART score for chest pain patients

<u>H</u> istory	Highly suspicious	2
(Anamnesis)	Moderately suspicious	1
	Slightly suspicious	0
<u>E</u> CG	Significant ST-deviation	2
	Non-specific repolarisation disturbance / LBBB / PM	1
	Normal	0
<u>A</u> ge	≥ 65 years	2
	45 – 65 years	1
	≤ 45 years	0
Risk factors	≥ 3 risk factors <i>or</i> history of atherosclerotic disease	2
	1 or 2 risk factors	1
	No risk factors known	0
<u>T</u> roponin	≥ 3x normal limit	2
	1-3x normal limit	1
	≤ normal limit	0
		Total

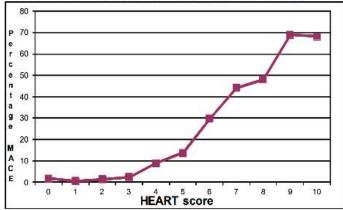
Risk factors for atherosclerotic disease:

Hypercholesterolemia Cigarette smoking

Hypertension Positive family history

Diabetes Mellitus Obesity (BMI>30)

HEART score reliably predicts endpoints



HEART	~ % pts	MACE/n	MACE	Death	Proposed Policy
0-3	32%	38/1993	1.9%	0.05%	Discharge
4-6	51%	413/3136	13%	1.3%	Observation, risk management
7-10	17%	518/1045	50%	2.8%	Observation, treatment, CAS

*MACE = Major Adverse Cardiac Event = Myocardial Infarction, PCI/CABG, all-cause death. Based on N=6174

Literature:

1. Chest pain in the emergency room: value of the HEART score.

Six AJ, Backus BE, Kelder JC. Neth Heart J. 2008;16:191-6.

2. Chest pain in the emergency room: a multicenter validation of the HEART Score.

Chest pain in the emergency room: a multicenter validation of the HEART Score Backus BE, Six AJ, Kelder JC, et al. Crit Pathways in Cardiol. 2010;9:164-9.

A prospective validation of the HEART score for chest pain patients at the emergency department.
 Backus BE, Six AJ, Kelder JC, et al. Int J Cardiol. 2013;168:2153-8.

 The HEART score for the assessment of patients with chest pain in the emergency department Six AJ, Cullen L, Backus BE, et al. Crit Pathways in Cardiol 2013;12:121–126.

Impact of using the HEART score in chest pain patients at the emergency department: a stepped wedge, cluster randomized trial. Poldervaart JM, et al. Annals of Internal Medicine. 2017. Epub ahead of print

Questions and comments:

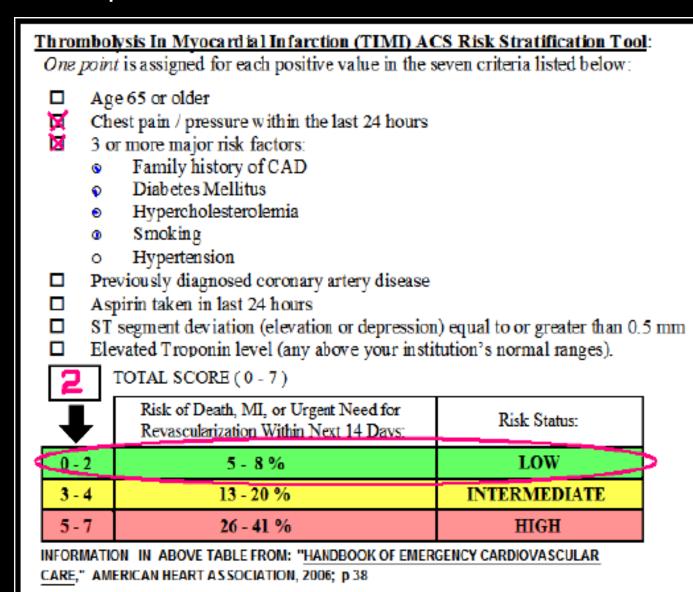
Barbra Backus backus@heartscore.nl
Jacob Six six@heartscore.nl
Judith Poldervaart poldervaart@heartscore.nl

www.heartscore.nl

HEART Score

-VS-

TIMI Score



CASE STUDY: IMPORTANCE of RISK FACTORS

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

62 y/o MALE presents to cardiologist's office with intermittent ACS symptoms (chest heaviness, dyspnea). - Pt. DOES NOT correlate symptoms with exertion.

RISK FACTOR PROFILE:

- **●** FAMILY HISTORY both parents + CAD before age 65**
- PREVIOUS CIGARETTE SMOKER 20+ yrs., quit 15 years ago
- HIGH CHOLESTEROL Dx 5 yrs ago, taking STATIN med since.
- **DIABETES** Controlled with diet and oral meds.

PHYSICAL EXAM: Patient supine on exam table, skin warm, dry, color NL Patient is asymptomatic, all systems WNL

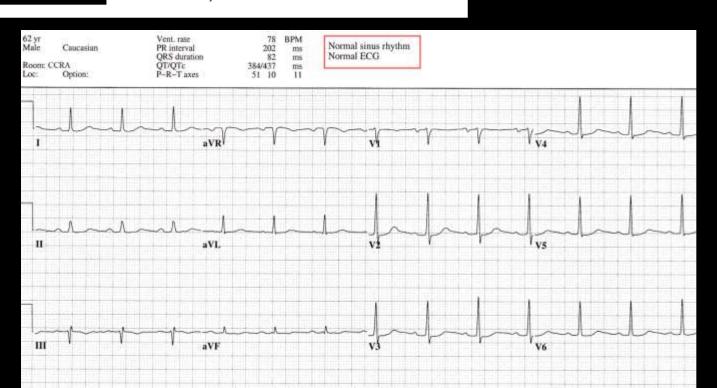
VITAL SIGNS: BP 153/88, P 80, R 16, SAO2 99%

DIAGNOSTIC TESTING: EKG NORMAL, EXERCISE STRESS TEST PASSED.

"The ACS Scorecard"

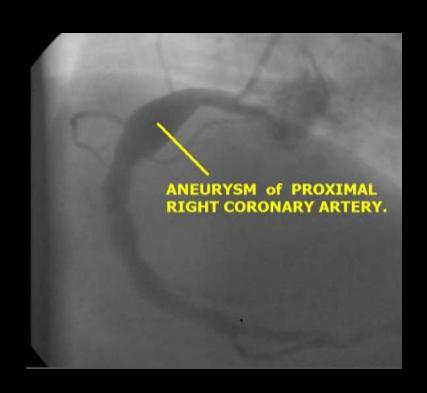
- ✓ PRESENTING SYMPTOMS
 ✓ RISK FACTOR PROFILE
 ☐ ECG ABNORMALITIES
- CARDIAC MARKERS

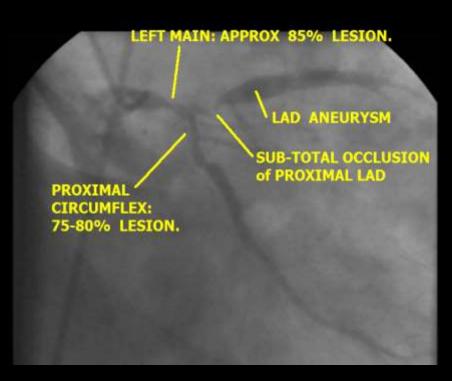
A POSITIVE finding in TWO or MORE of the above categories indicates it is EXTREMELY LIKELY that ACS is present.... steps must be AGGRESSIVELY TAKEN to definitively RULE OUT the PRESENCE of ACS!



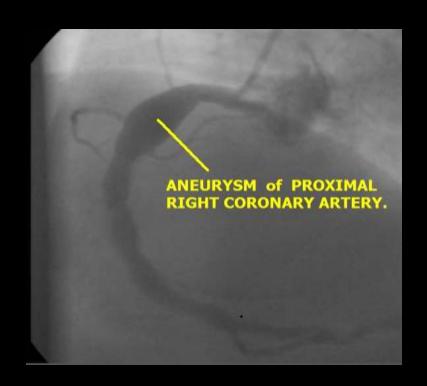
The Interventional Cardiologist was very suspicious of the man's Risk Factors and Symptoms, and convinced the man to consent to a Cardiac Catheterization......

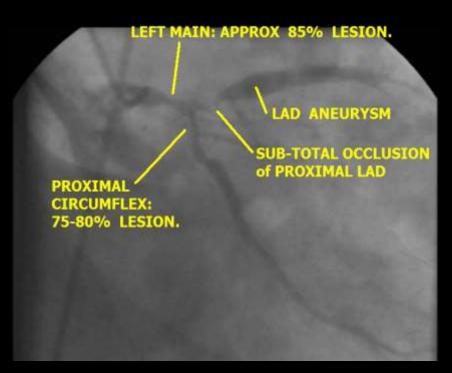
The Interventional Cardiologist was very suspicious of the man's Risk Factors and Symptoms, and convinced the man to consent to a Cardiac Catheterization......





The Interventional Cardiologist was very suspicious of the man's Risk Factors and Symptoms, and convinced the man to consent to a Cardiac Catheterization......





It's a good thing the Doctor didn't include the TIMI Score in his clinical decision-making. The patient was directly for emergency bypass surgery.

CASE STUDY 11 - NSTEMI ATYPICAL EKG

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

42 y/o MALE in ED c/o INTERMITTENT SUBSTERNAL CHEST PAIN x 9 HOURS, "8" on 1-10 scale, pain does not radiate, not effected by position/deep inspiration. Denies DIB.

Pt. given NTG 0.4mg SL without releif of CHEST PAIN.

RISK FACTOR PROFILE:

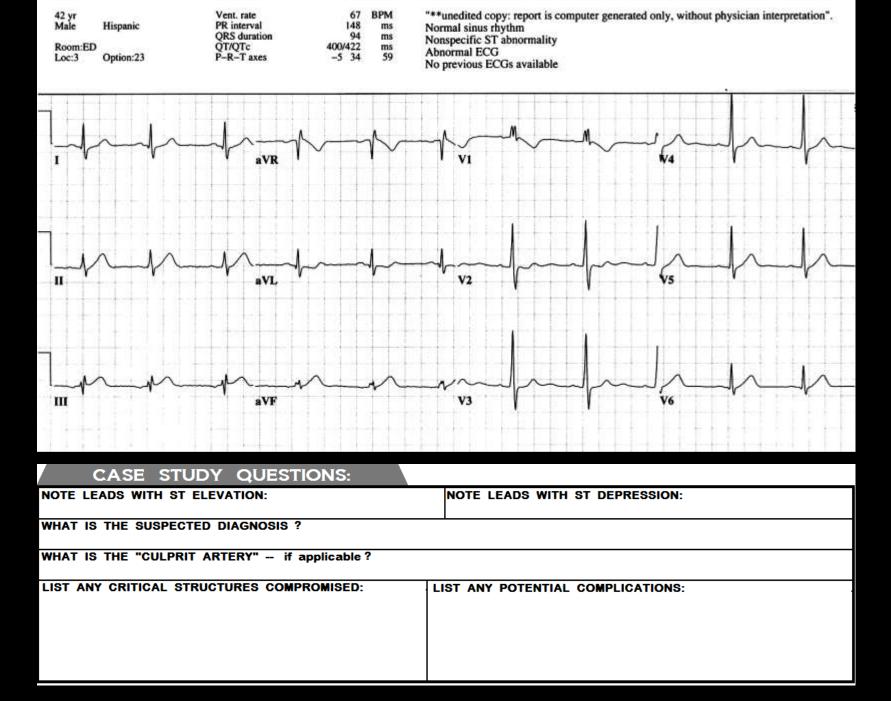
●* ELEVATED LDL CHOLESTEROL, LOW HDL CHOLESTEROL

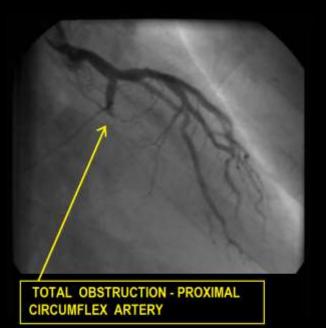
✔ PATIENT DENIES SMOKING, FAMILY HISTORY, HYPERTENSION

PHYSICAL EXAM: CAOx4, SKIN WARM, DRY, COLOR NORMAL, NON-ANXIOUS, LUNGS CLEAR, HEART SOUNDS NORMAL S1, S2, NO JVD, NO ANKLE EDEMA

VITAL SIGNS: BP: 122/76 P: 86 R: 16 SAO2: 98% on 2 LPM O2

LABS: TROPONIN: >500 CK: 4,410 CK MB: 224.1 CK INDEX: 5.1

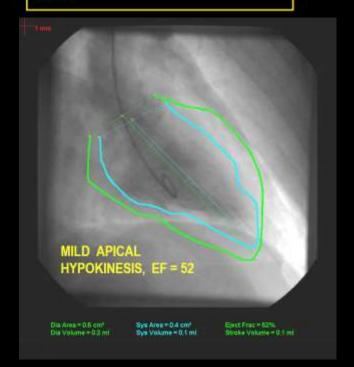








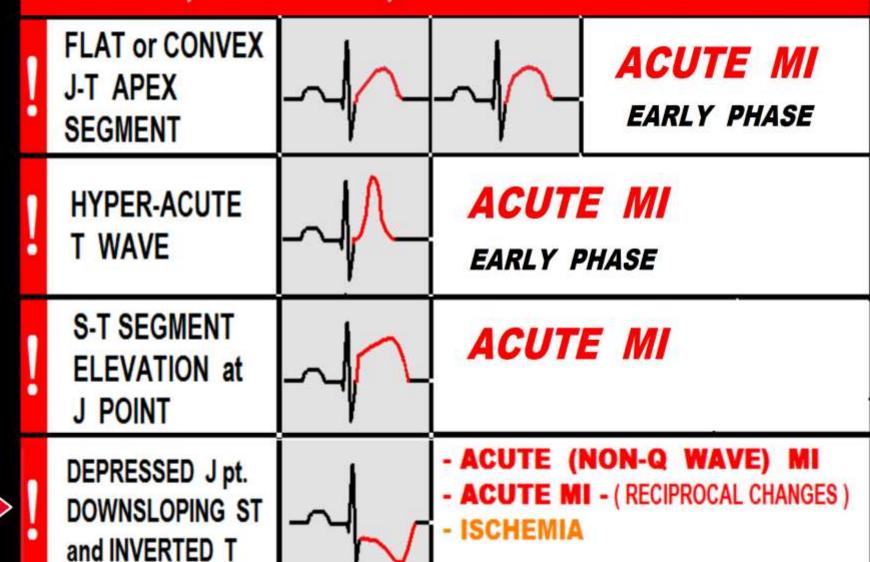
DOMINANT RIGHT CORONARY ARTERY OPEN



CASE STUDY SUMMARY			
ST ELEVATION:	ST DEPRESSION: NONE.		
SUSPECTED DIAGNOSIS: ACUTE NSTEMI - BASED ON SYMPTOMS & ELEVATED MARKERS			
SUSPECTED "CULPRIT ARTERY" (if applicable): UNABLE TO DETERMINE BASED ON 12 LEAD EKG PRESENTATION.			
IMMEDIATE CONCERNS FOR ALL ACUTE MI PATIENTS:			
BE PREPARED TO MANAGE SUDDEN CARDIAC ARREST (PRIMARY V-FIB/V-TACH, BRADYCARDIAS/HEART BLOCKS) STAT REPERFUSION THERAPY: THROMBOLYTICS vs. CARDIAC CATHETERIZATION and PCI CONSIDER NEEDS FOR ANTI-PLATELET and ANTI-COAGULATION THERAPY CRITICAL STRUCTURES COMPROMISED: POTENTIAL COMPLICATIONS: POSSIBLE CRITICAL INTERVENTIONS:			
240	AND CONTRACTOR AND CO		
MUSCLE MASS POSSIBLE MODERATE	SMALL FLUID CHALLENGE INOTROPIC AGENTS		
45% of POPULATION HAS SINUS BRASINUS NODE SUPPLIED BY CIRCUMFLEX ARTERY	ADYCARDIA ATROPINE TRANSCUTANEOUS PACING		

PATTERNS of ACS & ISCHEMIA

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



CASE STUDY 12 - NSTEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

63 y/o MALE in ED complaining of continuous PRESSURE in both SHOULDERS, with radiation down the RIGHT ARM to the elbow x approx. 6 hours. He took Motrin 800mg. without relief. Also c/o intermittent NAUSEA. He DENIES CHEST PRESSURE / DISCOMFORT and DIB.

RISK FACTOR PROFILE:

● ELEVATED TRIGLYCERIDES and LOW HDL

✓ SMOKER FOR 30+ YEARS, QUIT 6 YEARS AGO

PHYSICAL EXAM: CAOx4, SKIN WARM, DRY, COLOR PALE. PUPILS PERLA, NO JVD,

LUNGS CLEAR, HEART SOUNDS NORMAL S1, S2. NO ANKLE EDEMA

VITAL SIGNS: BP: 106/50 P: 90 R: 20 SAO2: 96% on 4 LPM O2

LABS: TROPONIN: 66.3 CK: 187 CK MB: 4.2

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) Fatigue

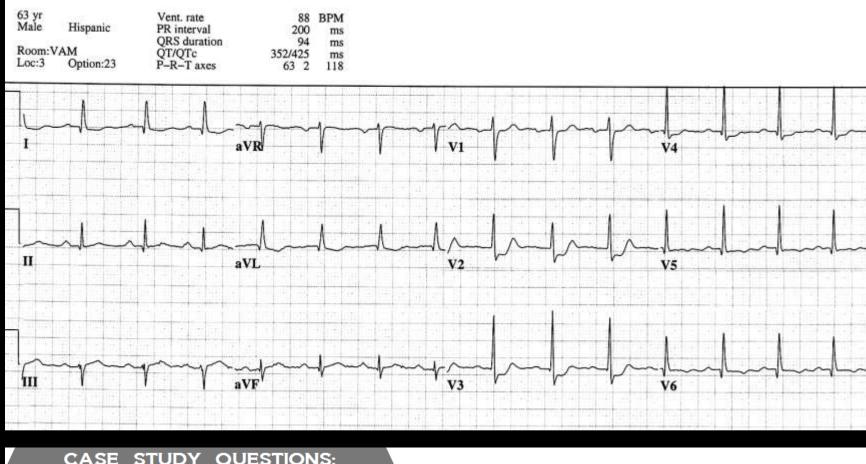
Indigestion Abdominal pain

Nausea Cold sweats

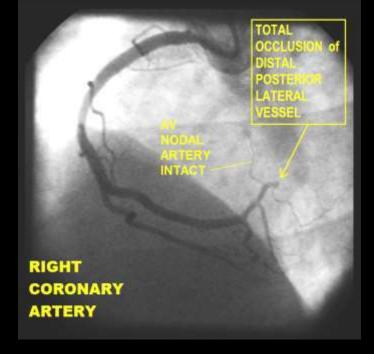
Dizziness Elevated heart rate

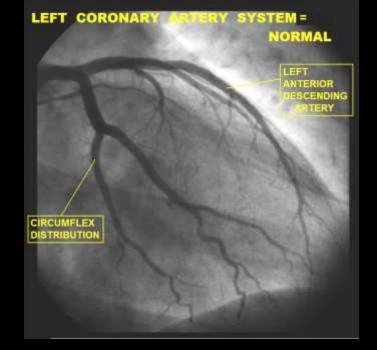
Syncope Dsypnea

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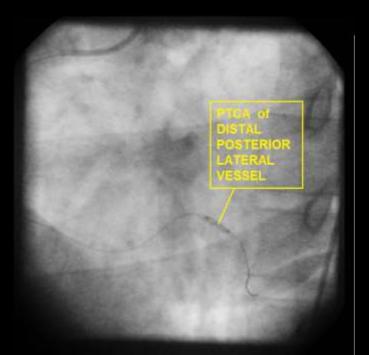


CASE STUDY QUESTIONS:	
NOTE LEADS WITH ST ELEVATION:	NOTE LEADS WITH ST DEPRESSION:
WHAT IS THE SUSPECTED DIAGNOSIS ?	
WHAT IS THE "CULPRIT ARTERY" if applicable?	
LIST ANY CRITICAL STRUCTURES COMPROMISED:	LIST ANY POTENTIAL COMPLICATIONS:

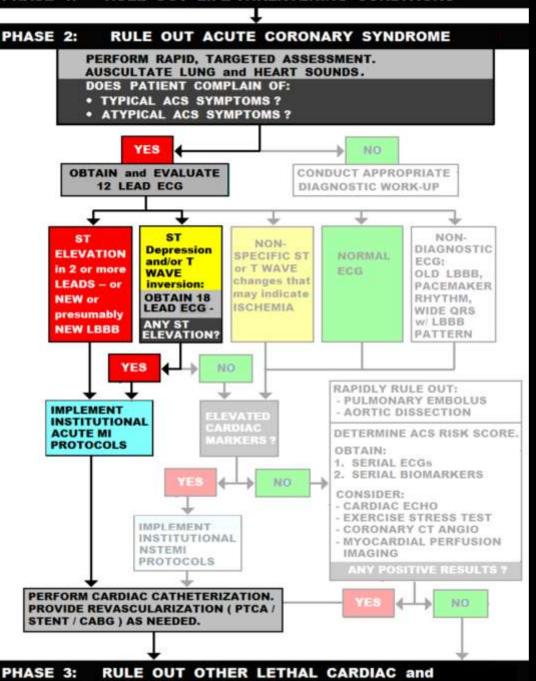








REMEMBER ...IT'S POSSIBLE TO HAVE A STEMI.. WHEN THERE'S NO ST ELEV. ON THE 12 LEAD!!



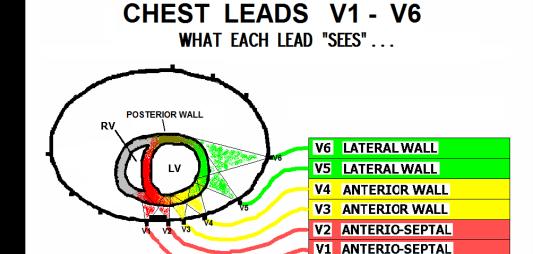
RULE OUT LIFE-THREATENING CONDITIONS

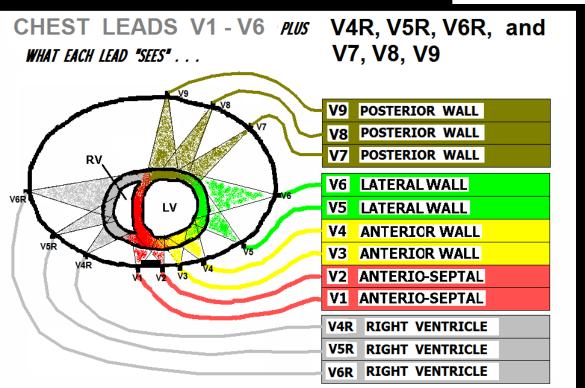
PHASE 1:

PHASE 3: **NON-CARDIAC CONDITIONS.**

THE 12 LEAD EKG HAS TWO MAJOR "BLIND SPOTS:"

- POSTERIOR WALL
- R VENTRICLE



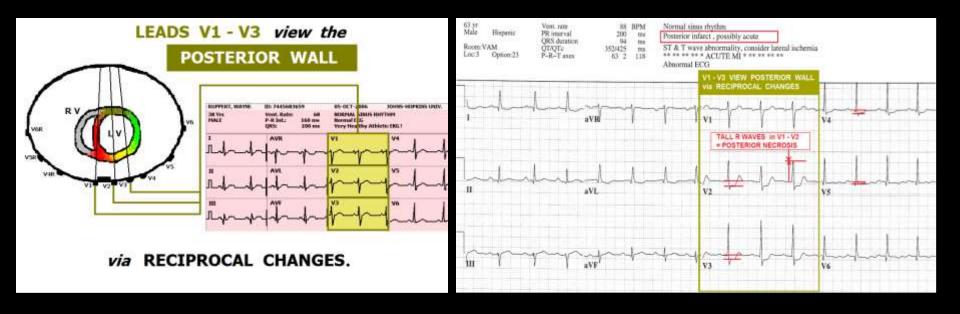


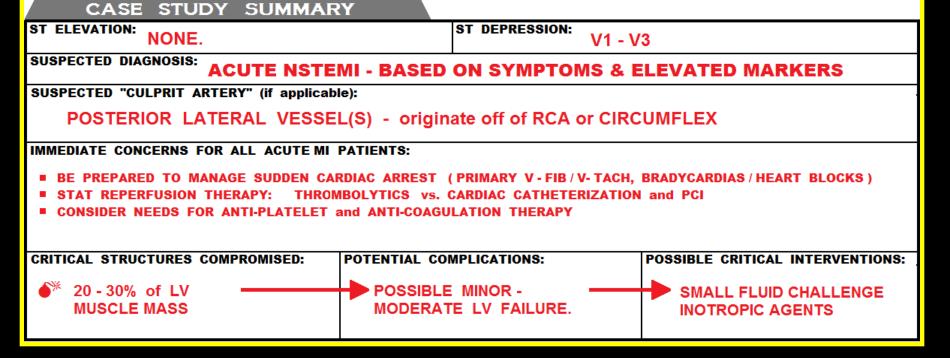
THE 18 LEAD EKG
ADDS COVERAGE
OF THE
-POSTERIOR WALL
-R VENTRICLE

INDICATIONS FOR OBTAINING AN 18 LEAD EKG:

1. INFERIOR WALL MIS (ST ELEV II, III, aVF)

2. SUSPECTED POSTERIOR WALL MI (ST DEPR V1, V2, and/or V3, V4)





CASE STUDY 13 - NSTEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

67 y/o FEMALE presents to ED with intermittent exertional CHEST PRESSURE x 1 day. Pt. DENIES shortness of breath, nausea. CHEST PRESSURE does not radiate.

RISK FACTOR PROFILE:

CIGARETTE SMOKER x 40 YEARS

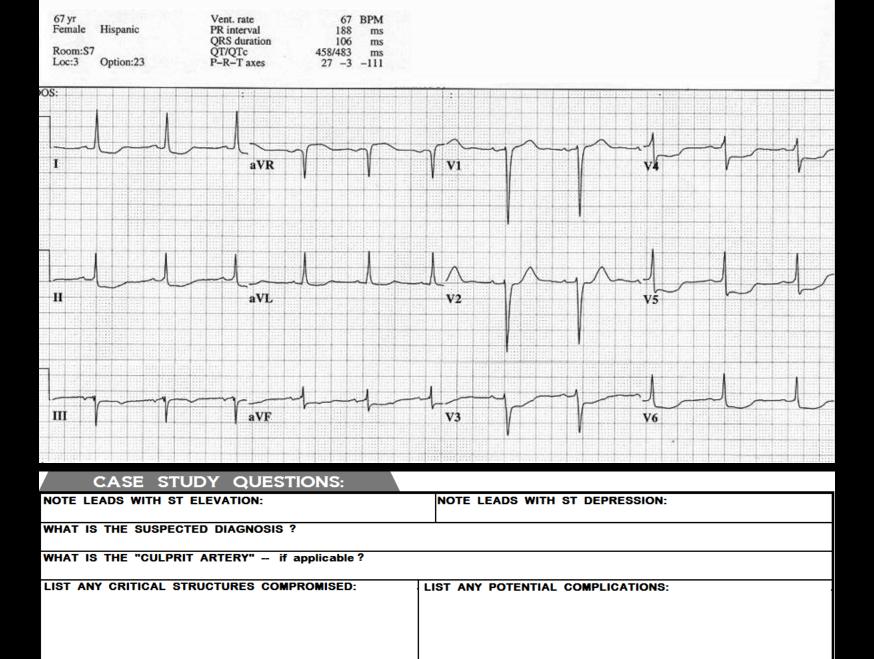
HYPERTENSION

▲ AGE >65

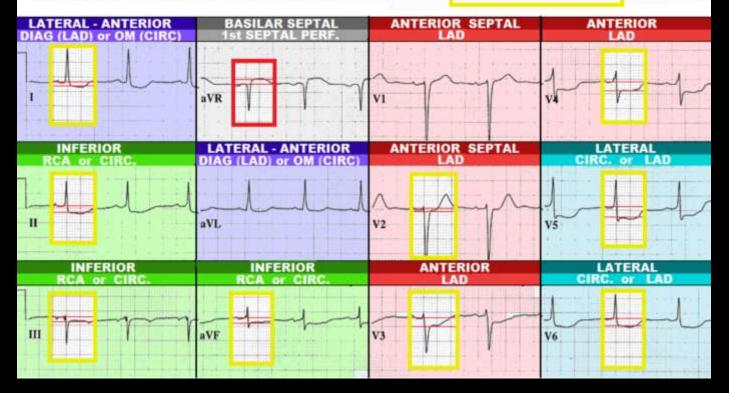
PHYSICAL EXAM: Pt. CAO x 4 in NAD, SKIN WARM, DRY, COLOR NORMAL. PUPILS PERLA, NO JVD LUNGS = DECREASED, CRACKLES IN BASES. HEART SOUNDS NORMAL S1, S2, NO ANKLE EDEMA,

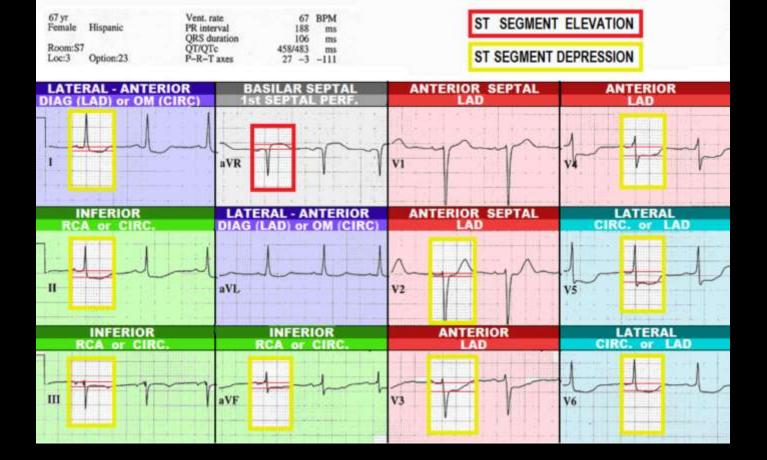
VITAL SIGNS: BP: 133/88 P: 68 R: 20 SAO2: 95% on 2 LPM O2

LAES: TROPONIN: 4.8 CK: 525 CK MB: 29



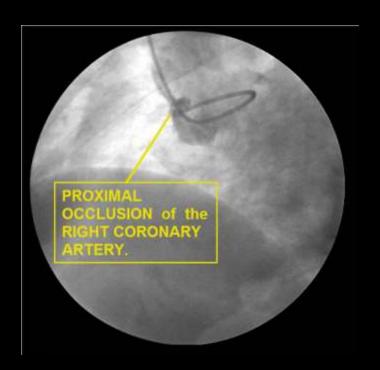
ST SEGMENT ELEVATION
ST SEGMENT DEPRESSION

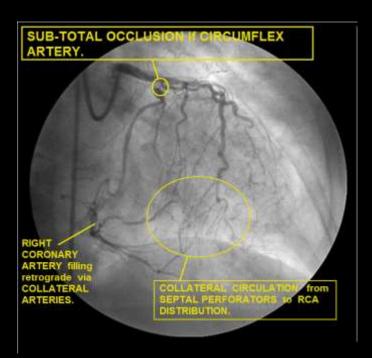


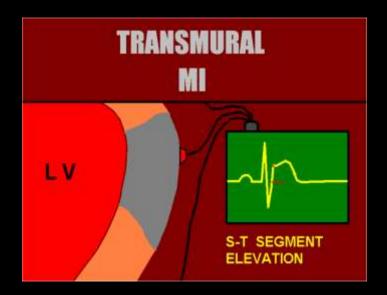


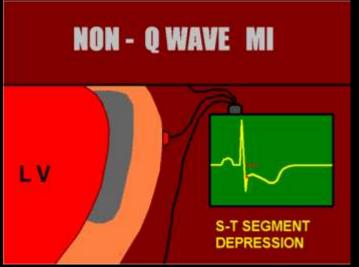
- LEAD aVR sometimes referred to as "the forgotten 12th lead" can be a source of valuable information. In this case study, lead aVR is the only lead with ST elevation.
- In cases of myocardial ischemia and NSTEMI, ST segment elevation of lead aVR has been associated with a high incidence of triple vessel disease, [1] which is true in this case study.
- In cases of anterior wall STEMI, elevation of lead aVR indicates the patient's lesion is proximal to the origin of the first septal perforator. [2]
- When the ST elevation of lead aVR is higher than that of V1, it is considered an indicator that the left main coronary artery is obstructed.^[3] Please review Case Study 4 (p 183), STEMI, and involving occlusion of Left Main Coronary Artery.

While reviewing ECGs for inclusion in this curriculum, we noticed the correlation between J point elevation in lead aVR and the incidence of severe multivessel disease.











CASE STUDY 14 - NSTEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

45 y/o FEMALE c/o CHEST PAIN, SHORTNESS of BREATH and WEAKNESS x "SEVERAL DAYS." She states she has been under "a great amount of stress" in the past month, and recently started taking diet pills containing EPHEDRA.

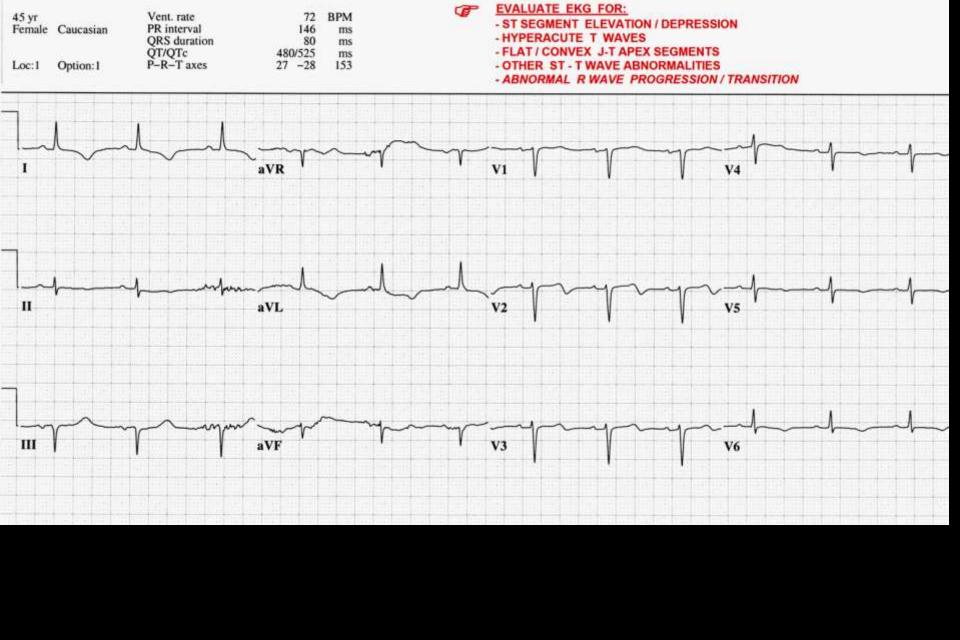
RISK FACTOR PROFILE:

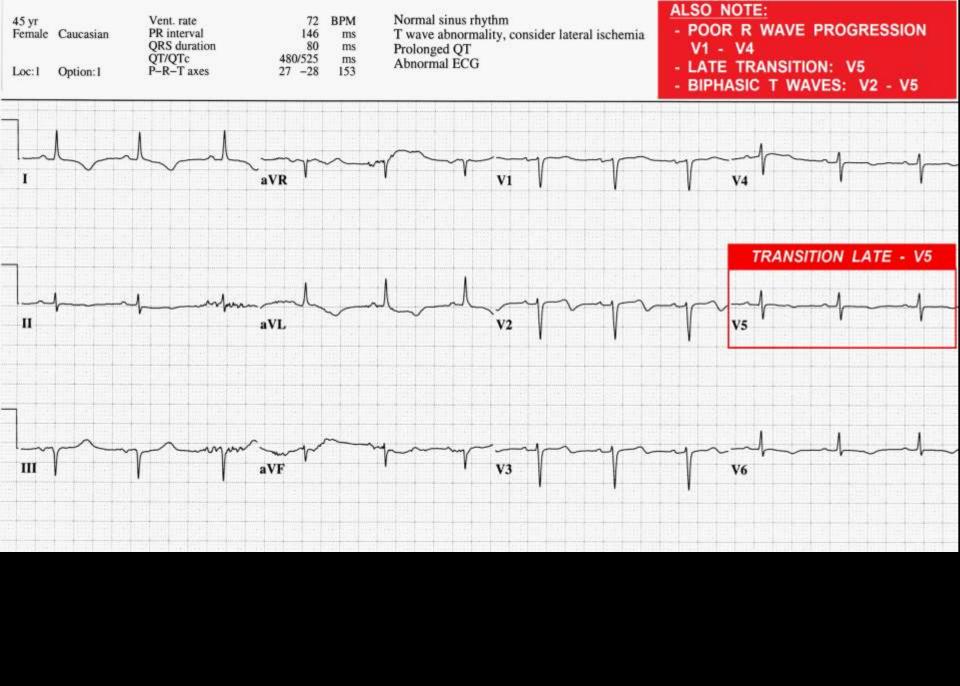


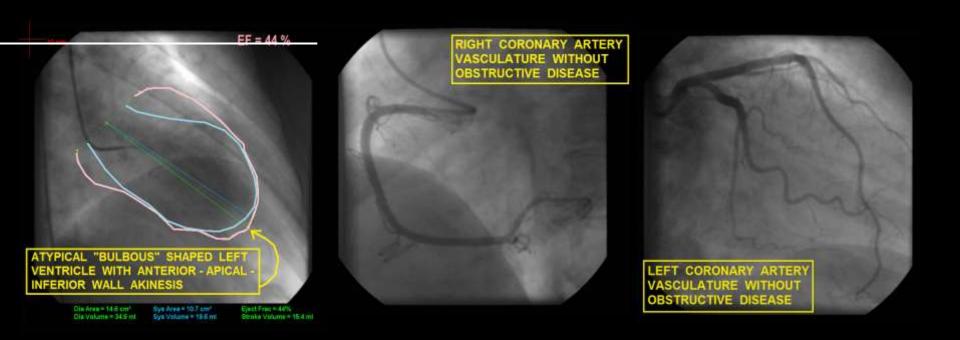
PHYSICAL EXAM: Pt. CAO X 4, skin warm, dry, color normal. Lung sounds clear, HS Normal S1, S2. No JVD, No ankle edema.

VITAL SIGNS: BP: 106/66 P: 80 R: 24 SAO2: 95 % on 2 LPM O2

LABS: TROPONIN: 135







Apical ballooning syndrome, also known as "broken heart syndrome" and "acute stress induced cardiomyopathy," may account for up to 2% of all incidents of acute myocardial infarction.^[1] This condition is uncommon but often life-threatening. ABS can be provoked by extreme emotional distress and adrenergic substances (including ephedrine alkaloids).

UNSTABLE ANGINA CASE STUDIES

stable angina

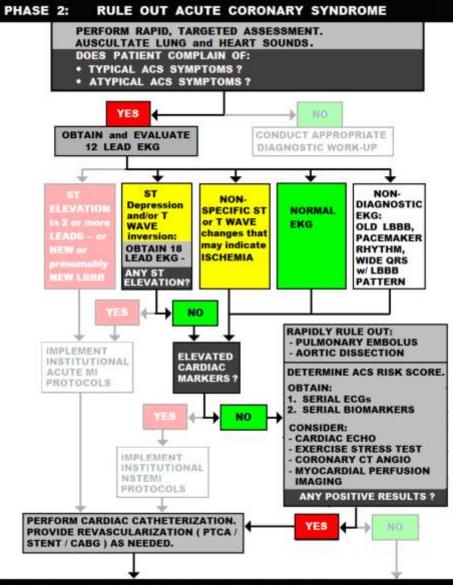
- SYMPTOMS START DURING PHYSICAL EXERTION.
- SYMPTOMS ARE "PREDICTABLE"



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

PHASE 1: RULE OUT LIFE-THREATENING CONDITIONS



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

ACUTE CHEST PAIN PROTOCOL

DATE:		
	•	
	Obtain STAT 12 Lead ECG; Repeat every 30 minutes for persistent or worsening pain.	
	 IF ST segment elevation is noted on ECG, immediately notify the House Nursing Supervisor of possible Code STEMI patient. 	
	 Notify *physician that STAT 12 Lead ECG is available online in PACS system for STAT interpretation if PACS is not available, FAX a copy of each ECG to physician 	
	Obtain STAT Vital signs, including patient's level of pain. Repeat every 15 minutes X 4	
	If SAO2 <94%, administer Oxygen, 2 - 4 Litres/minute via nasal canula, titrate to keep SAO2 >94%	
	Follow ACLS Protocols	
	Initiate IV NS @KVO rate, preferably with 20g catheter or larger.	
	Administer Aspirin - chew four 81mg baby aspirin (or one 325 mg adult tablet, if baby ASA not avail)	
	✓ HOLD if sildenafil citrate (Viagra) or vardenafil (Levitra) has been taken within 24 hours	
	HOLD if tadalafil (Cialis) has been taken within 48 hours	
	✓ HOLD and notify MD if SBP less than 90 mmHg	
	Morphine Sulfate 2mg IV PRN for chest pain, may repeat every 5 minutes up to Max of 10 doses (20mg total) in 4 hours	
	, ,	
	Notify physician of any abnormal ECG findings, ECG changes or abnormal Troponin values.	

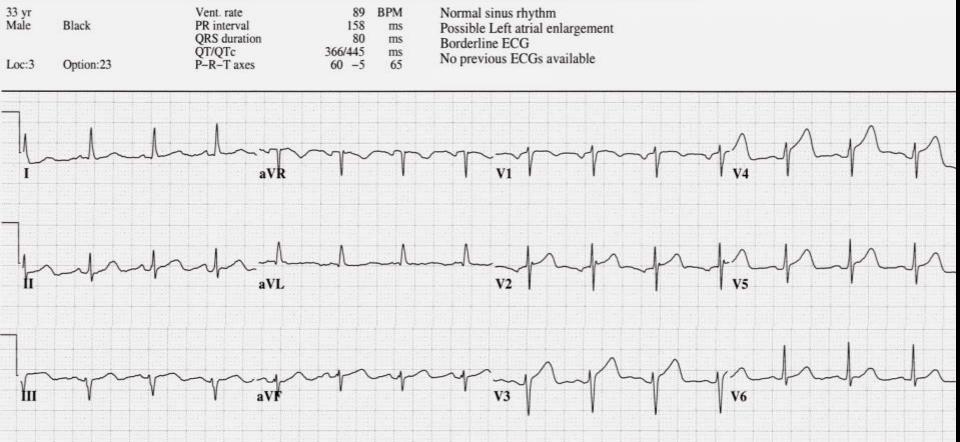
^{*} If patient is currently under the services of Cardiology, the "physician" is the Cardiologist.

If patient is not being followed by Cardiology, then "physician" refers to Attending Physician.

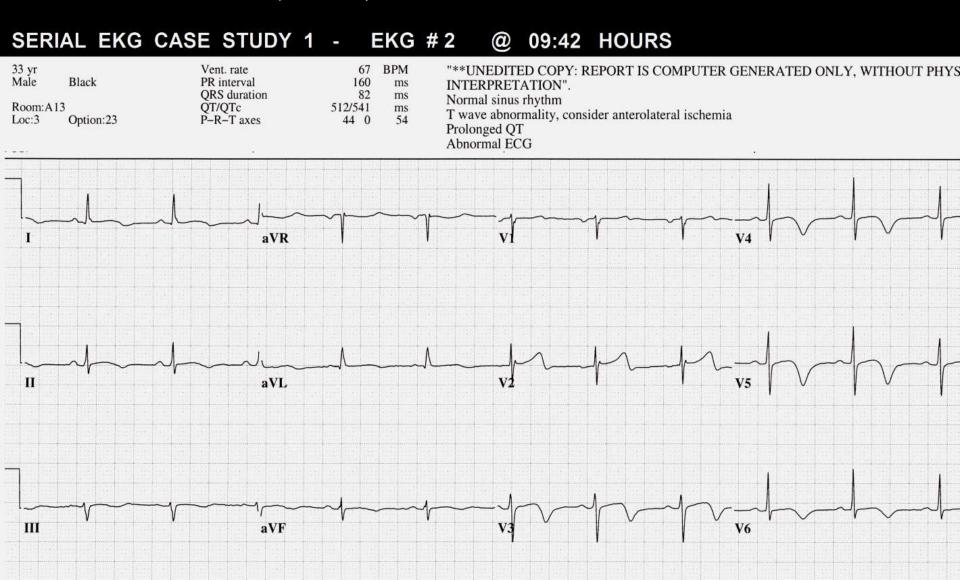
CASE STUDY: SERIAL ECGs.

33 y/o MALE, C/O "COUGHING WITH CHEST PAIN." ST ELEVATION BELIEVED TO BE "EARLY REPOLARIZATION." A VETERAN ED PHYSICIAN DISCERNED THAT THE PATIENT'S CHEST PAIN STARTED BEFORE THE COUGHING, AND ORDERED SERIAL ECGs.

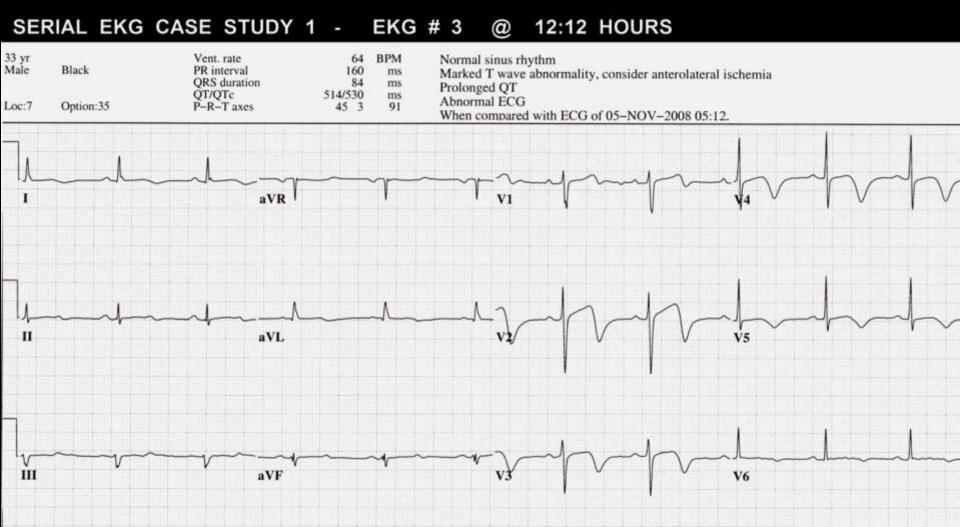
SERIAL EKG CASE STUDY 1 - EKG #1 @ 06:22 HOURS



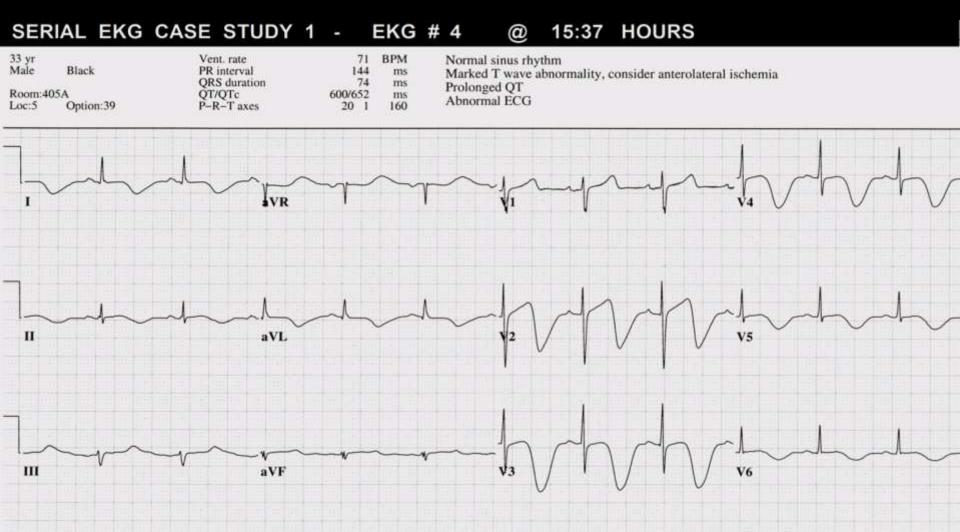
APPROX. 3 hrs LATER: BI-PHASIC T WAVES V2, V3; INVERTED Ts V4 – V6

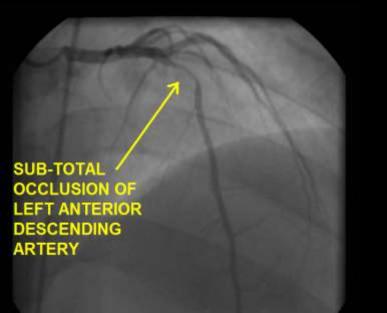


APPROX 6 hrs AFTER 1ST ECG: BIPHASIC Ts V1, V2; INVERTED T WAVES V3 - V6



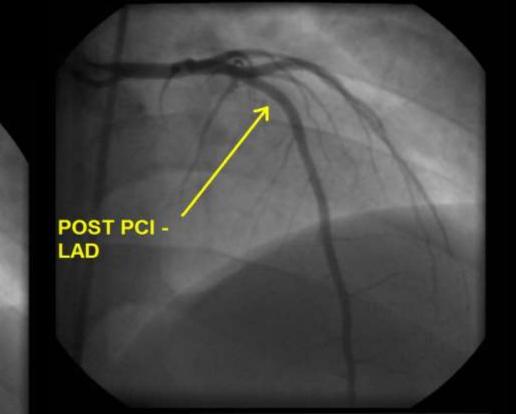
APPROX 9 hrs AFTER 1ST ECG: BIPHASIC Ts V1, V2; INVERTED T WAVES V3 - V6



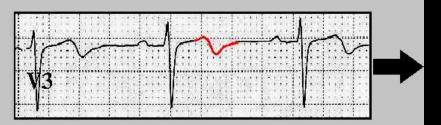


PATIENT TAKEN TO THE CARDIAC CATH LAB, WHERE A SUB-TOTALLY OCCLUDED PROXIMAL L.A.D. WAS DISCOVERED (left). BOTTOM LEFT: PTCA/STENT TO L.A.D. BOTTOM RIGHT: POST STENT TO L.A.D.

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



BI-PHASIC T WAVES



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



Classic "Wellen's Syndrome:"

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

Wellen's Syndrome ETIOLOGY:

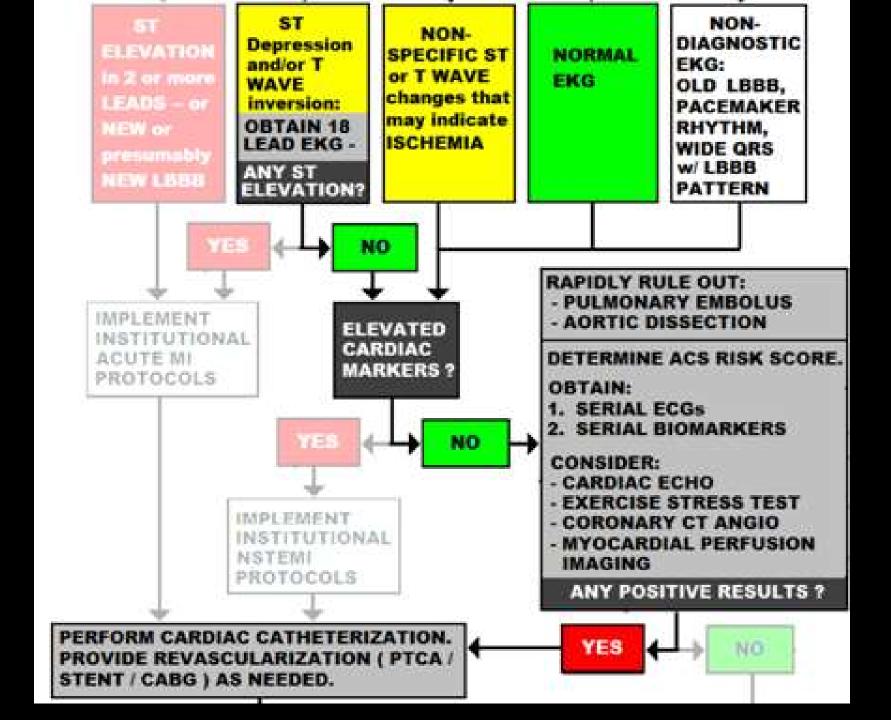
- Critical Lesion, Proximal LAD
- Coronary Artery Vasospasm
- Cocaine use (vasospasm)
- Increased myocardial oxygen demand
- Generalized Hypoxia / anemia / low H&H

Wellen's Syndrome EPIDEMIOLOGY & PROGNOSIS:

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

Sources: H Wellens et. Al, Am Heart J 1982;

v103(4) 730-736



CASE STUDY: RELEVANCE OF STRESS TESTING

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

71 y/o male presents to the cardiologist's office, c/o EXERTIONAL SUBSTERNAL CHEST PRESSURE and DIZZINESS. PMHx of Hypertension, AV Nodal Reentrant Tachycardia and vertigo.

RISK FACTOR PROFILE:

FAMILY HISTORY - both parents.

☑ PREVIOUS CIGARETTE SMOKER

CHOLESTEROL - unknown.

AGE - OVER 65

✓ HYPERTENSION

PHYSICAL EXAM: Patient alert, oriented x 3, skin warm, dry, color normal, carotids 2+ bilaterally, no bruits, Lungs clear, HS S1, S2 normal, no murmurs/gallops/rubs. Extremities: good distal pulses, no edema.

VITAL SIGNS: BP: 118/60, P: 74, R: 16, SAO2: 98%

LABS: CARDIAC MARKERS NEGATIVE. BMP, CBC: WNL.

DIANOSTIC EVALUATIONS:

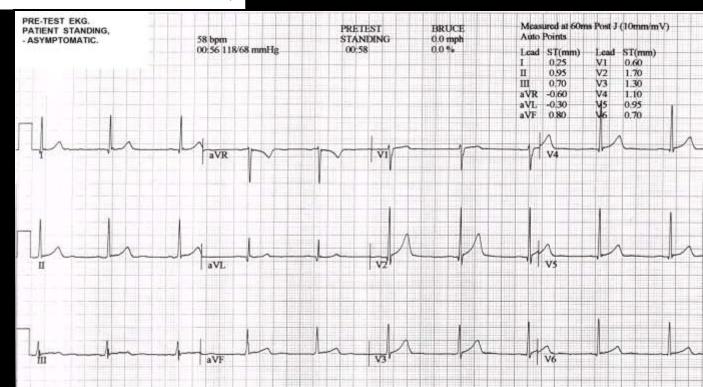
☞ 2-D/M-MODE DOPPLER ECHOCARDIOGRAM: NORMAL

(LV size, Normal LV function, trace of mitral and tricuspid regurgitation).

MYOCARDIAL PERFUSION STUDY: NORMAL.

(LVEF = 60%, STRESS and REST TOMOGRAPHIC PERFUSION IMAGES = NORMAL).

VALUE OF STRESS TESTING...



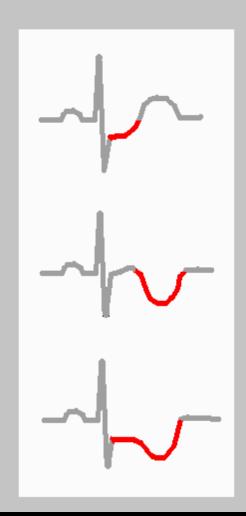
ISCHEMIA

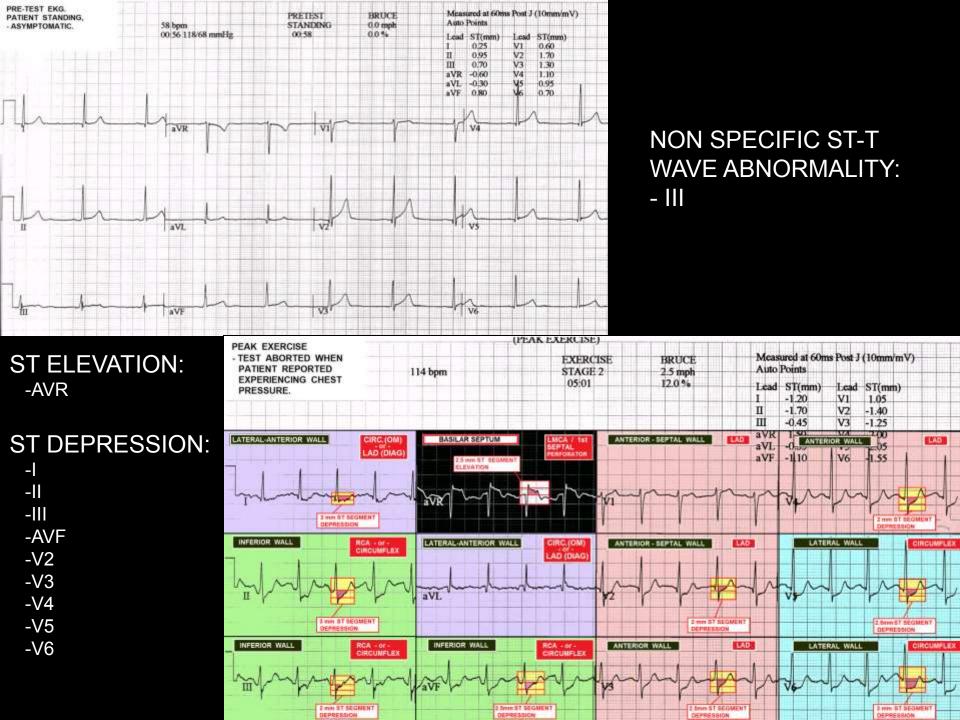
HELPFUL PATTERNS . . .

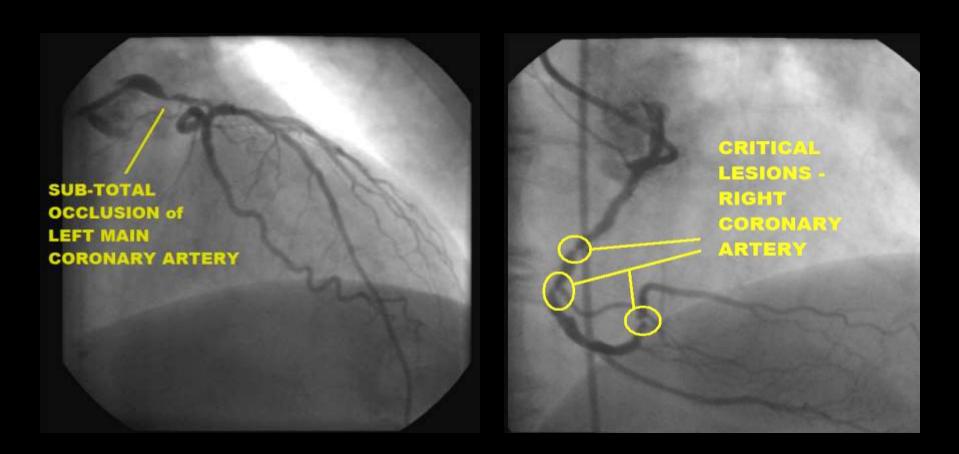
J POINT DEPRESSION (> 1 mm)

INVERTED T WAVES

J POINT DEPRESSION + INVERTED T WAVES









CASE STUDY 15 - UNSTABLE ANGINA

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

42 y/o FEMALE c/o INTERMITTENT CHEST PRESSURE which has been WORSENING during the past week. Also c/o mild DIB. Symptoms previously provoked by exertion, now comes on at rest.

RISK FACTOR PROFILE:

HYPERTENSION

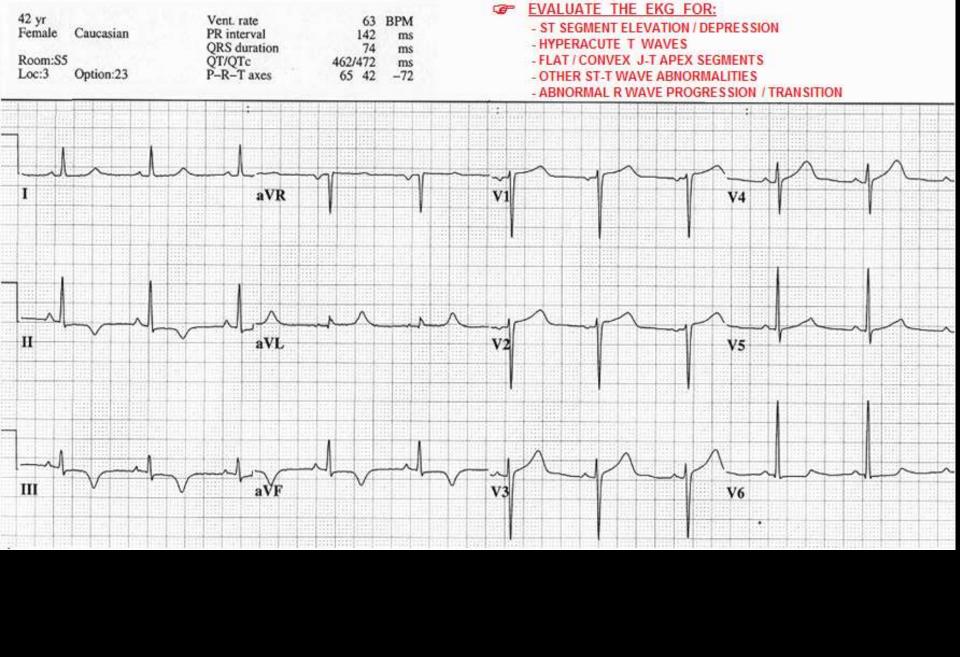
●*CIGARETTE SMOKER x 15 YEARS

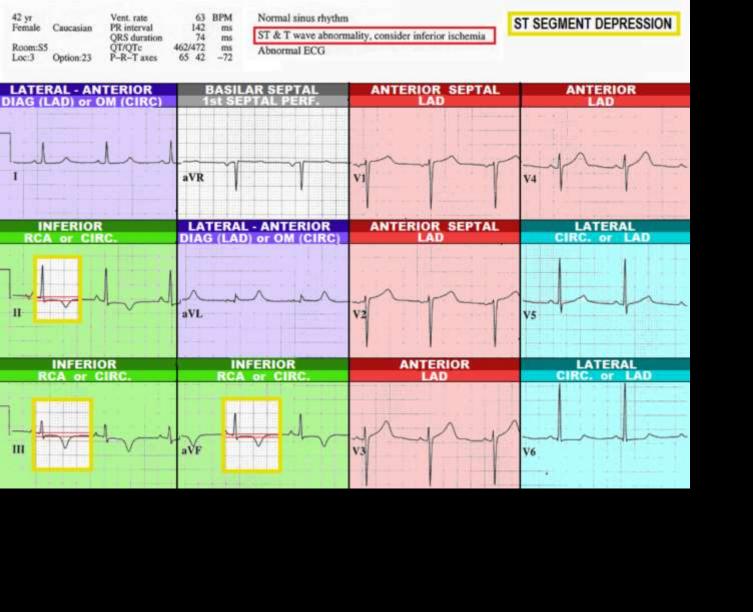
FAMILY HISTORY - FATHER Dx WITH CAD, HAD CABG AT 52

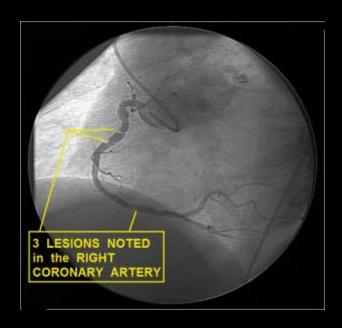
PHYSICAL EXAM: Pt. ASYMPTOMATIC at time of exam. SKIN WARM, DRY, COLOR NORMAL, PERLA, LUNGS= CLEAR, HS NORMAL S1, S2, NO ANKLE EDEMA.

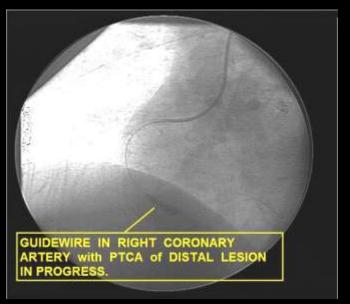
VITAL SIGNS: BP: 148/92 P: 64 R: 20 SAO2: 97 % on 2 LPM O2

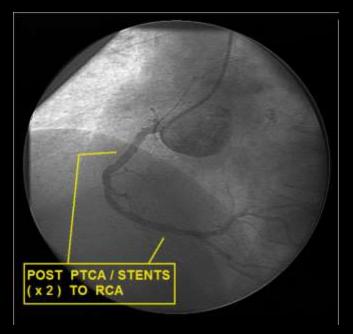
LABS: TROPONIN: <.04

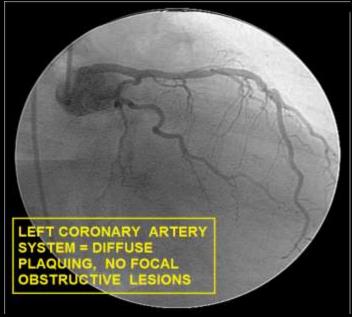












CASE STUDY 16 - UNSTABLE ANGINA

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

38 y/o MALE presents with sensation of exertional CHEST and NECK PAIN, described as "burning." Patient states symptoms also occur when he is under emotional duress. Symptoms have been occurring intermittently for approx. 2-3 weeks.

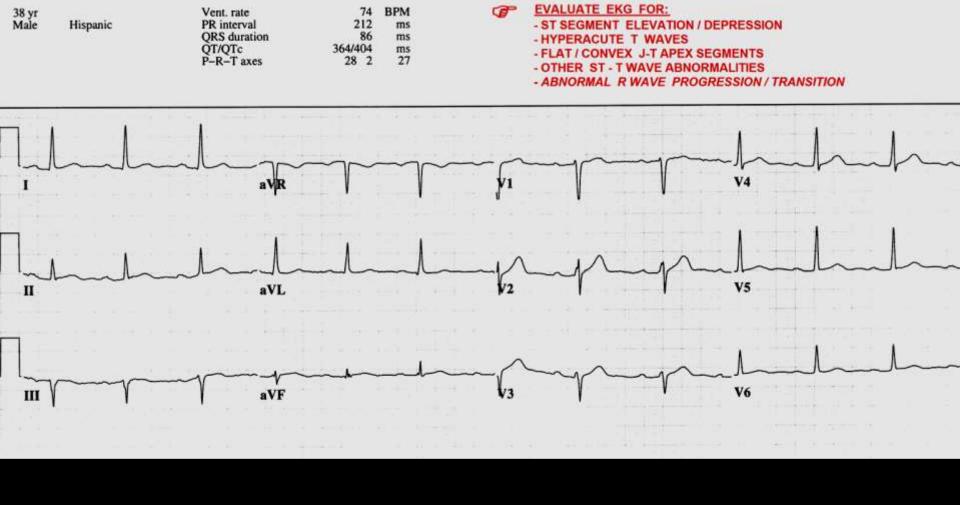
RISK FACTOR PROFILE:

HYPERTENSION
DIABETES x 5 YEARS

PHYSICAL EXAM: Pt. ASYMPTOMATIC at time of exam. SKIN WARM, DRY, COLOR NORMAL, PERLA, LUNGS= CLEAR, HS NORMAL S1, S2, NO ANKLE EDEMA.

VITAL SIGNS: BP: 144/92 P: 78 R: 16 SAO2: 100% on room air

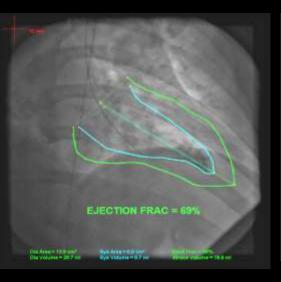
LABS: TROPONIN: <.04

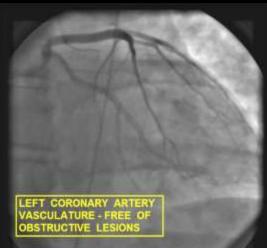


74

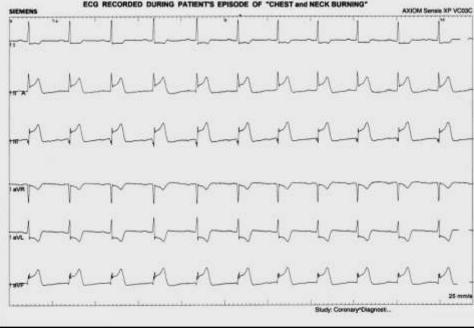
BPM

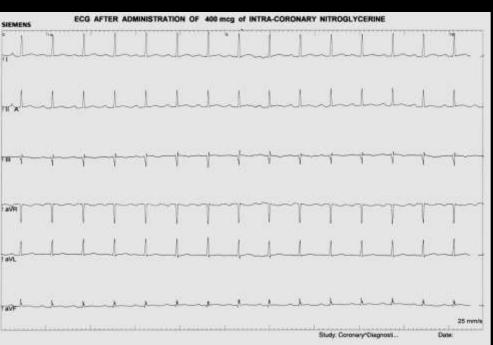
EVALUATE EKG FOR:















Prinzmetal or variant angina is caused by focal coronary artery vasospasm, and was first described by Myron Prinzmetal in 1959 as a syndrome of episodic chest pain that comes on at rest with ST segment elevation. Prinzmetal angina is classified as unstable angina due to its unpredictability, and has been associated with myocardial infarction, ventricular dysrhythmias and cardiac arrest. The primary mechanism of vasospasm is hypercontraction of vascular smooth muscle cells. Variant angina is not an indicator of CAD; many patients are free of atherosclerotic plaque. Some factors known to provoke coronary artery vasospasm include: vasoconstrictor medications, stimulants such as cocaine, ephedrine and amphetamines, emotional duress, exposure to cold and alcohol withdraw.

Typical Prinzmetal's variant angina occurs at rest, in the early hours of the morning. The pain is often described as severe chest tightness or pressure. Variant angina is usually treated with and responds well to calcium channel blockers and nitrates.^[3]

^[1] Prinzmetal et al, *Am J Med*. 1959;27:375-388.

^[2] National Institutes of Health, Library of Medicine, www.NIH.gov

^[3] National Institutes of Health, Library of Medicine, www.NIH.gov

CASE STUDY 17 - UNSTABLE ANGINA

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

45 y/o MALE c/o EXERTIONAL CHEST PRESSURE x past 2 months, getting worse. In last week, CHEST PRESSURE has come on at rest. DYSPNEA sometimes present. Pain is relieved when patient rests, however now takes longer than 20 minutes to subside.

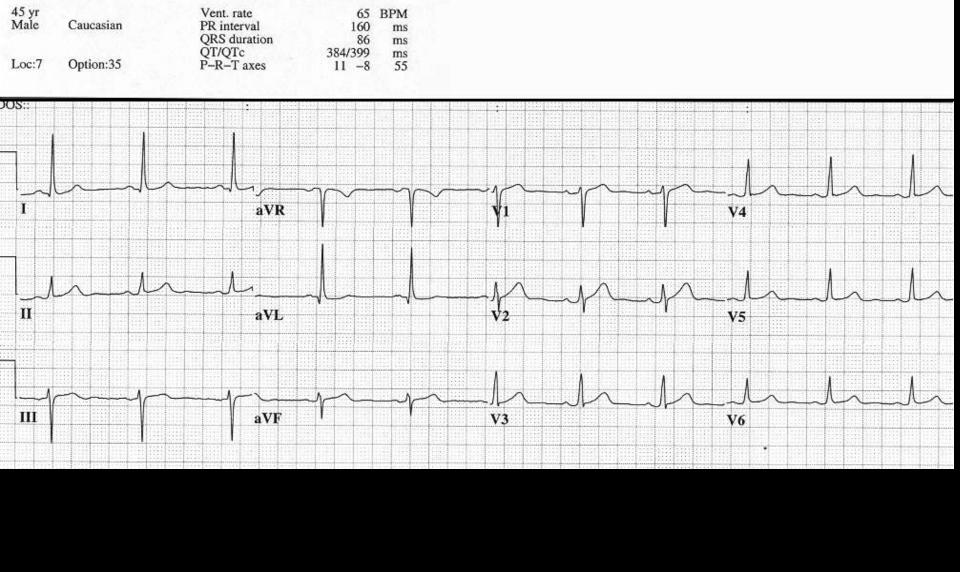
RISK FACTOR PROFILE:

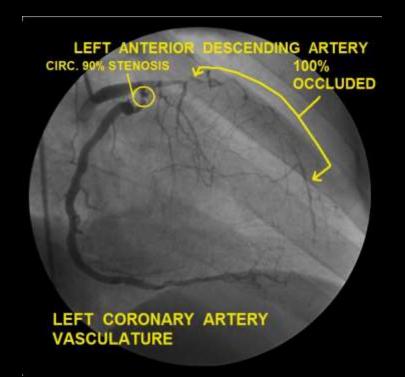
- FAMILY HISTORY: father died of AMI age 50, brother had CABG age 44
- **™** CIGARETTE SMOKER x 20 YEARS
- **●*HYPERTENSION**
- **●**ELEVATED LDL, TRIGLYCERIDES, LOW HDL CHOLESTEROL**

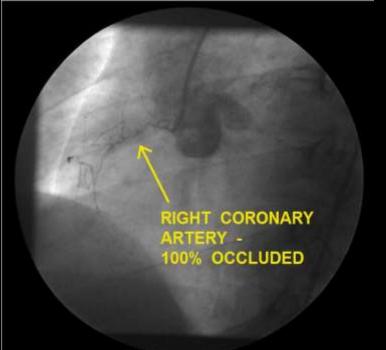
PHYSICAL EXAM: Pt. asymptomatic at time of exam, skin warm, dry, color normal, pupils PERLA, no JVD, lungs = clear, heart sounds normal S1, S2. Abd. soft, non-tender, No ankle edema

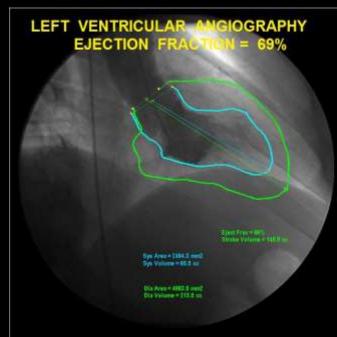
VITAL SIGNS: BP: 177/96 P: 64 R: 16 SAO2: 99 % on room air

LABS: TROPONIN: <.04









QUESTIONS???





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