12 LEAD ECG INTERPRETATION in

Acute Coronary Syndromes & Sudden Arrhythmia Death Syndromes (ACS & SADS)

Key West 2018

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Electrophysiology Lab Case Studies

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

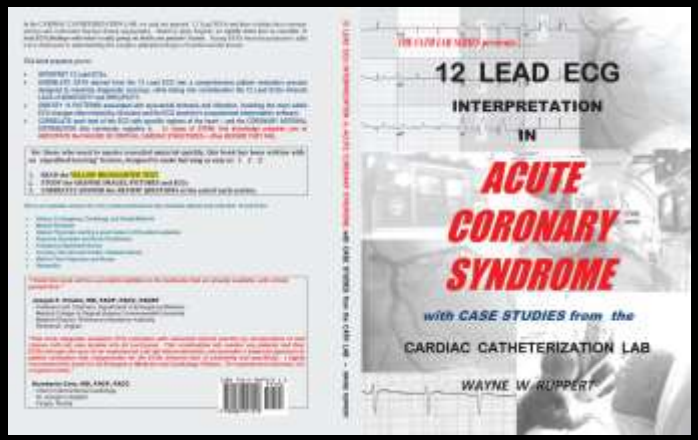
Author Wayne Ruppert conducting Pace-mapping during EP study at the St Joseph's Hospital Heart Institute, Pediatric Electrophysiology Program, Tampa, FL in 2004

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

Observation Medicine ECG Course

BASIS:

- **Current ACC/AHA Guidelines and Recommendations**
- **Multiple additional recent Evidence-Based Publications**
- **ECGs from case files of the author, Wayne Ruppert**
- **Graphic art / images from published textbooks authored by Wayne Ruppert**



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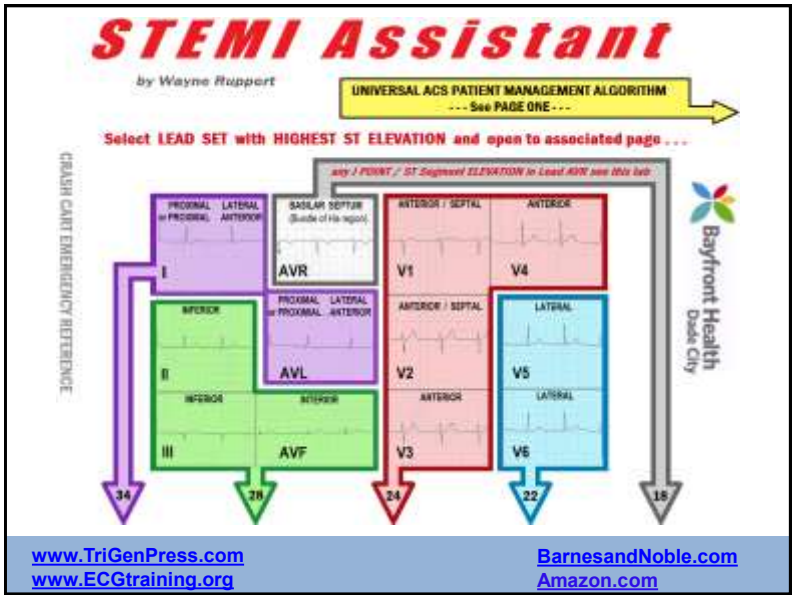


www.ECGtraining.org
www.practicalclinicalskills.com

PROGRAM CONTENTS

SESSION TWO

THE ACUTE CORONARY SYNDROMES

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

+ [illegible]

The EKG in PERSPECTIVE

1. Much development in the 1950s and 60s, and at that time, EKGs were the primary diagnostic tool.
2. Today we have better diagnostic tools (e.g. ECHO, CARDIAC CATH, EP STUDIES) that sometimes conflict with traditional EKG-made diagnoses.
3. Some EKG findings are more accurate and reliable than others .

AND . . .

Sometimes,
ECGs
LIE to us !

ECGs and USED CAR SALESMEN often have MUCH in common !

The EKG in PERSPECTIVE

PROBLEMS WITH EKGs . . .

↓ SENSITIVITY

(FALSE NEGATIVES)

↓ SPECIFICITY

(FALSE POSITIVES)

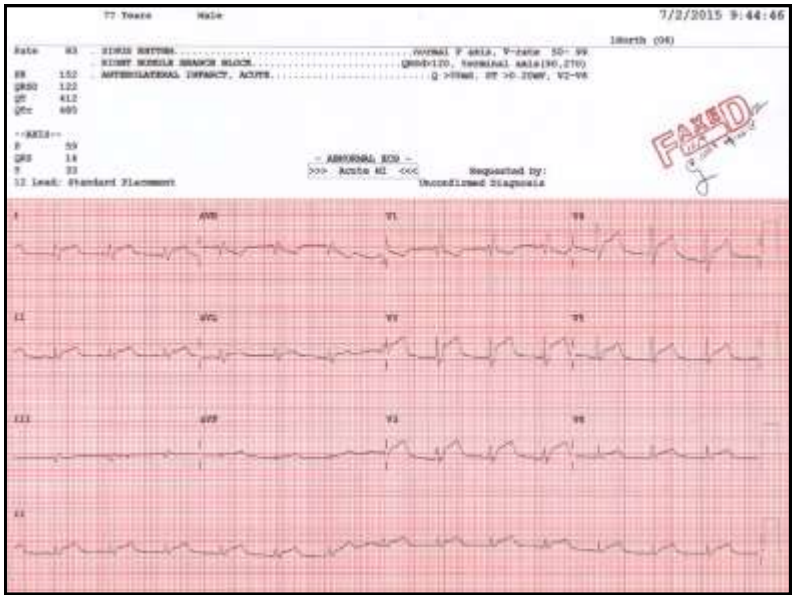
AND . . .

PROBLEMS WITH SPECIFICITY . . .

S-T SEGMENT ELEVATION - COMMON ETIOLOGIES:

CONDITION:

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



Patient:

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

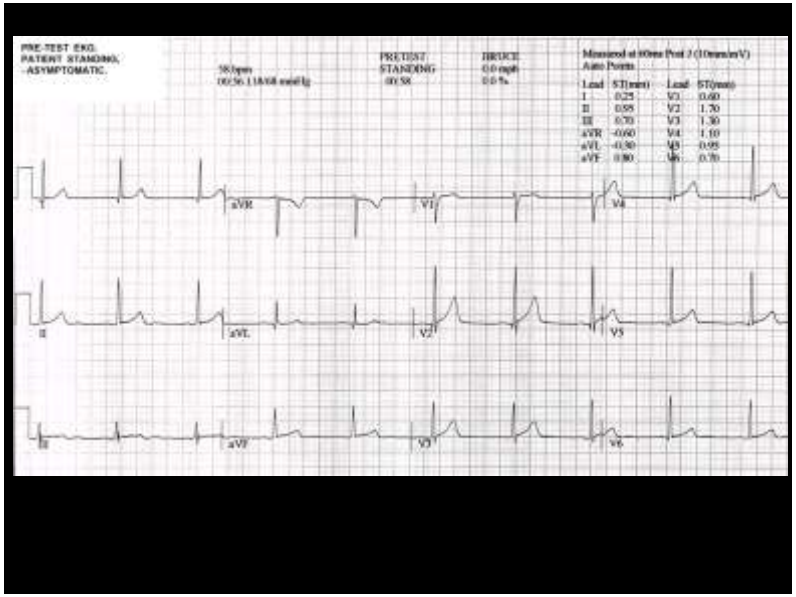
EARLY REPOLARIZATION. This degree of ST Elevation in early repolarization is VERY RARE: The only such ECG I have seen in approximately 13,000 cardiac catheterizations.

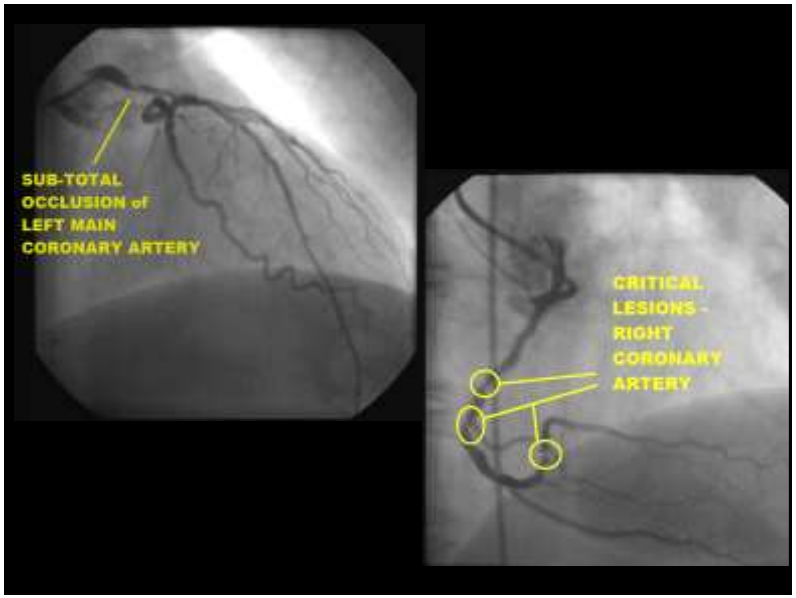
EKGs in PERSPECTIVE, con't:

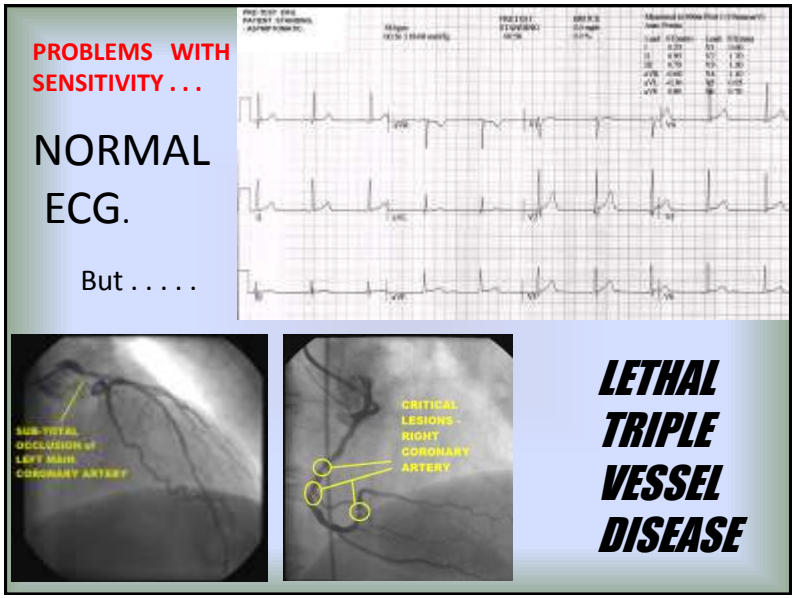
One of the MOST MISLEADING scenarios of all is when the EKG APPEARS PERFECTLY NORMAL . . .

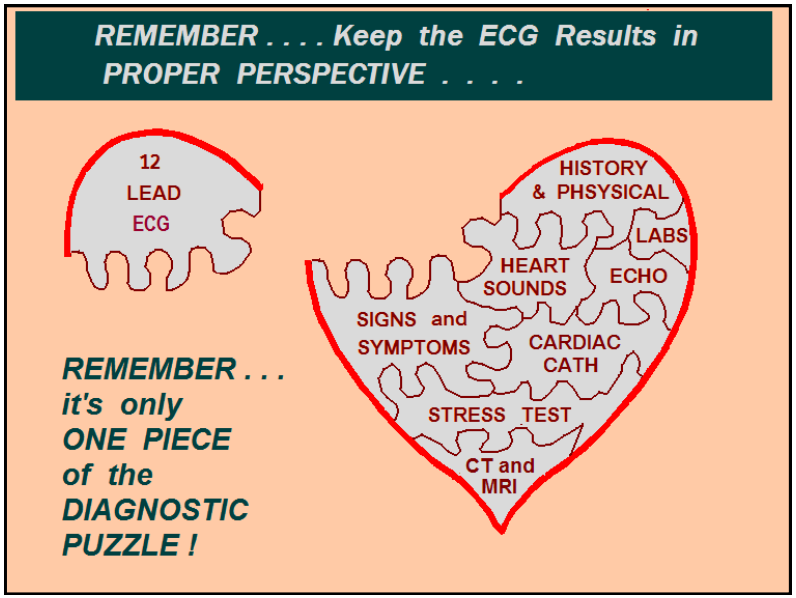
. . . but MASKS serious, LIFE - THREATENING CONDITIONS.

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




SUB-TOTAL
OCCLUSION of
LEFT MAIN
CORONARY ARTERY
CRITICAL
LESIONS -
RIGHT
CORONARY
ARTERY


PROBLEMS WITH SENSITIVITY . . .
NORMAL ECG.
But . . . . .
LETHAL TRIPLE VESSEL DISEASE



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

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

HEART

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

*Risk factors for atherosclerotic disease:

Hypercholesterolemia, Cigarette smoking, Hypertension, Positive family history, Diabetes Mellitus, Obesity

RISK FACTORS

for the development of

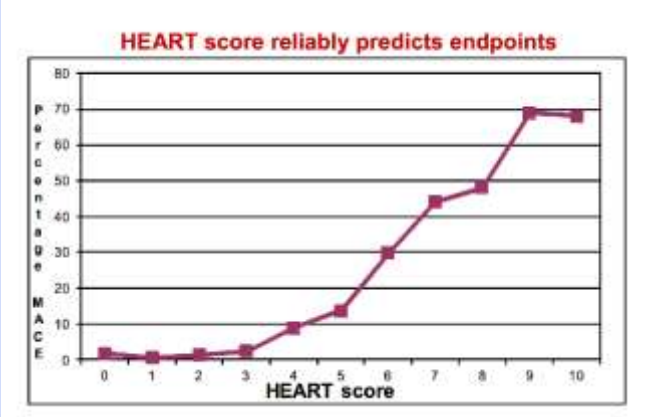
CORONARY ARTERY DISEASE:

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

per the AMERICAN HEART ASSOCIATION

Score	% pts	MACE/n	MACE	Death	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, CAG

Heart Score Reliability



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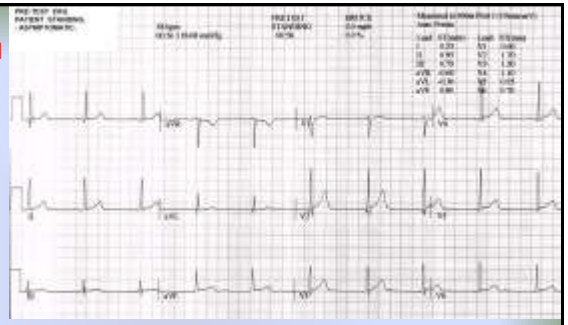

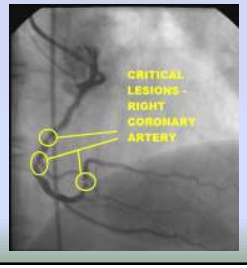

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		Total	

*Risk factors for atherosclerotic disease:

Hypercholesterolemia, Cigarette smoking, Hypertension, Positive family history, Diabetes Mellitus, Obesity

H = chest pain = 2

E = ECG normal = 0

A = 63 = 1

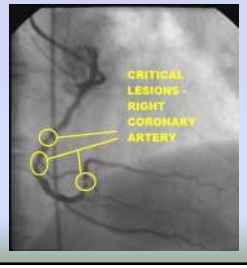
R = 3 risk fctors = 2

T = Trop. NL = 0

HEART Score: = 5

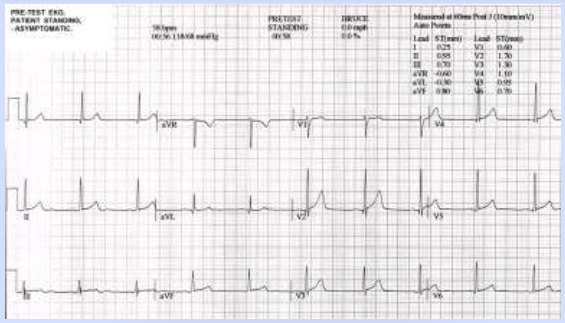
HEART SCORE:

5



Outcome:

Successful Emergency Bypass Surgery



2 patients with the above ECG.
- Patient 1 HEART Score of “0”
- Patient 2 HEART Score of “7”

Should they get the same care ??

Symptoms

TYPICAL SYPTOMS of *ACUTE CORNARY SYNDROME:*

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

INFARCTION

- - - "Classic Symptoms" - - -

QUICK ASSESSMENT "SHORT FORM"

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

stable angina		unstable angina
1. SYMPTOMS START DURING PHYSICAL EXERTION.	VS.	1. SYMPTOMS MAY START AT ANY TIME, EVEN DURING REST
2. SYMPTOMS ARE "PREDICTABLE"		2. SYMPTOMS ARE *NEW*, *DIFFERENT*, or *WORSE* 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:

- **S**troke (previous history of)
- **H**eart failure (previous history of)
- **R**ace (non-white)
- **E**lderly (age 75+)
- **W**omen
- **D**iabetes mellitus

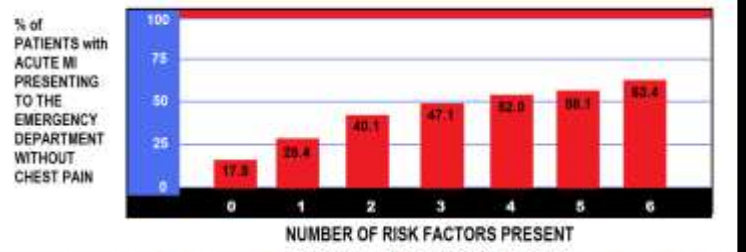
* The information listed in the table to the immediate left resulted from a study conducted by John G. Canto, MD, MSPH, et. al., of the University of Alabama. The study consisted of 434,877 patients diagnosed with AMI between 1994 and 1998 in 1,674 US hospitals. Study results were published in the Journal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

Common atypical complaints associated with AMI without chest pain include:

Malaise (weakness)	**F**atigue
Indigestion	**A**bdominal pain
Nausea	**C**old sweats
Dizziness	**E**levated heart rate
Syncope	**D**sypnea

BOOK PAGE: 70

Effect of Having Multiple Risk Factors for AMI Without Chest Pain



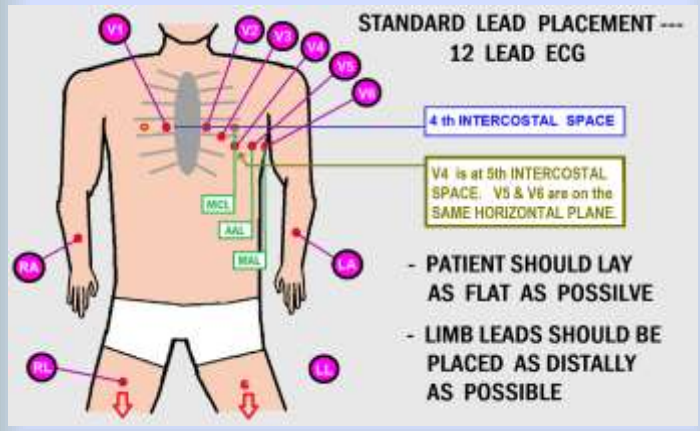
RISK FACTORS INCLUDE: Stroke (previous), Heart failure (previous), Race (non-white), Elderly (age 75+), Women, Diabetes

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

OBTAINING THE 12 LEAD ECG

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

Obtaining the 12 Lead ECG



Obtaining the 12 Lead ECG

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

Obtaining the 12 Lead ECG

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

Obtaining the 12 Lead ECG

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

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

AHA/ACC/HRS Scientific Statement

Recommendations for the Standardization and Interpretation of the Electrocardiogram

Part I: The Electrocardiogram and Its Technology

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

AHA/ACC/HRS Scientific Statement

Recommendations for the Standardization and Interpretation of the Electrocardiogram

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Recommendations

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

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

Leads V1 & V2 on 12 Lead ECG:

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

AHA/ACC/HRS Scientific Statement

Recommendations for the Standardization and Interpretation of the Electrocardiogram

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

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

Initial 12 Lead ECG

- **Obtain and interpret within 10 minutes of patient presentation**

Initial 12 Lead ECG

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- **Interpreted by physician / advanced practitioner**

Initial 12 Lead ECG

- Obtain and interpret within 10 minutes of patient presentation
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- **Determines presence of STEMI and/or other imminent life-threatening condition**

Initial 12 Lead ECG

- Obtain and interpret within 10 minutes of patient presentation
- Interpreted by physician / advanced practitioner
- Determines presence of STEMI and/or other imminent life-threatening condition
- **Should be compared to any previously recorded ECGs in the patient's medical records**

Initial 12 Lead ECG, continued:

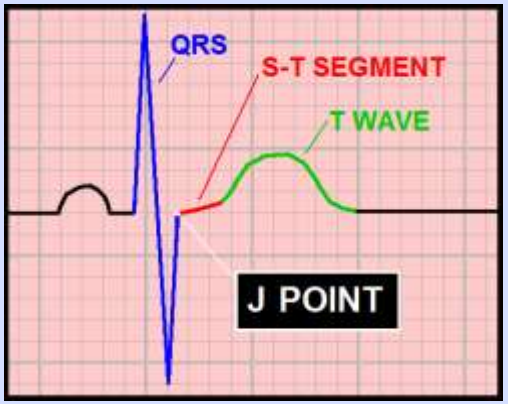
- **Additional Serial ECGs should be compared to the BASELINE ECG for determining the presence of Dynamic J Point, ST-Segment and T Wave Changes**

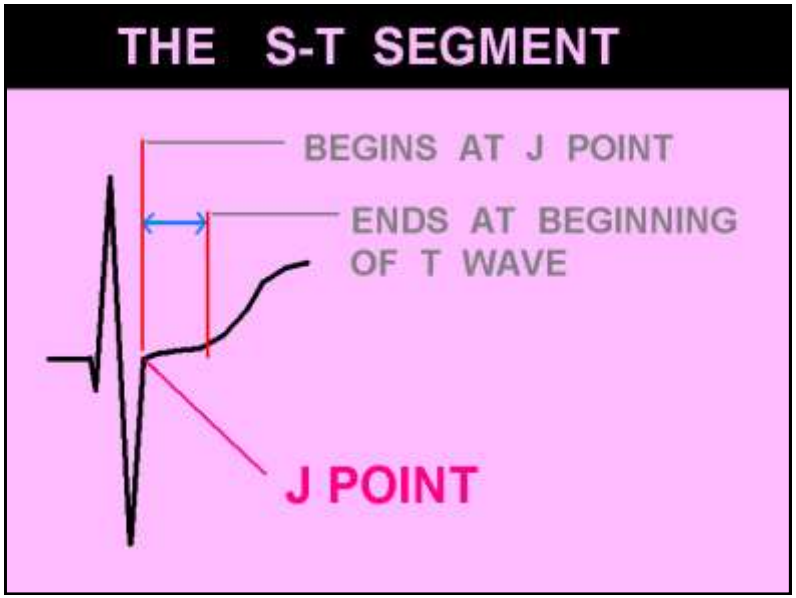
Initial 12 Lead ECG, continued:

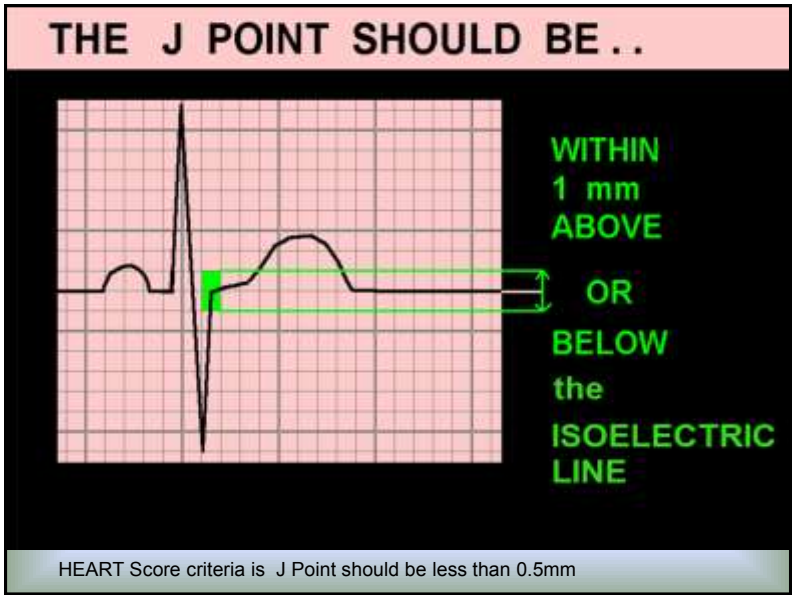
- Additional Serial ECGs should be compared to the BASELINE ECG for determining the presence of Dynamic J Point, ST-Segment and T Wave Changes
- **Serves as "footprint" for determining ECG lead(s) to be used during Continuous ECG Monitoring**
  - **Ischemia**
  - **QT interval**

Normal ECG – No ACS Indicators

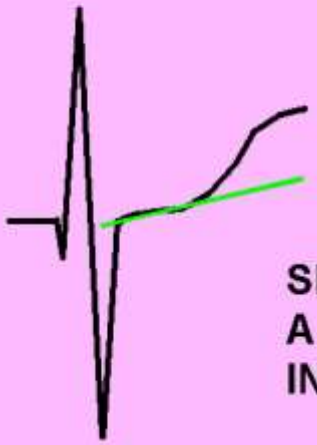
Defining NORMAL – QRS <120ms:




THE S-T SEGMENT
BEGINS AT J POINT
ENDS AT BEGINNING OF T WAVE
J POINT

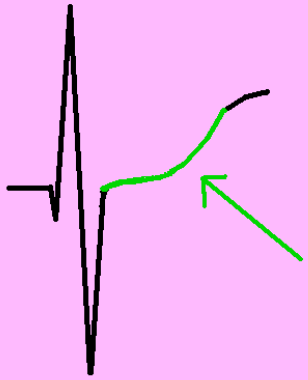

THE J POINT SHOULD BE . .
WITHIN 1 mm ABOVE
OR
BELOW
the
ISOELECTRIC LINE
HEART Score criteria is J Point should be less than 0.5mm

THE S-T SEGMENT



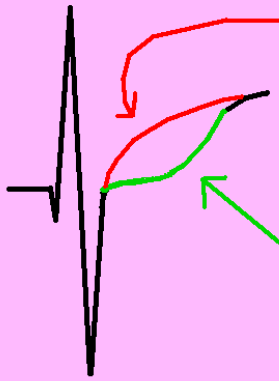
SHOULD HAVE
A "SLIGHT POSITIVE"
INCLINATION

THE S-T SEGMENT



SHOULD BE
"CONCAVE" IN
SHAPE . . .

THE S-T SEGMENT



AS OPPOSED TO "CONVEX" IN SHAPE

SHOULD BE "CONCAVE" IN SHAPE . . .

Evaluating the ECG for ACS:

A TWO-STEP process:

Evaluating the ECG for ACS:

A TWO-STEP process:

STEP 1: Evaluate QRS Width

Evaluating the ECG for ACS:

A TWO-STEP process:

STEP 1: Evaluate QRS Width

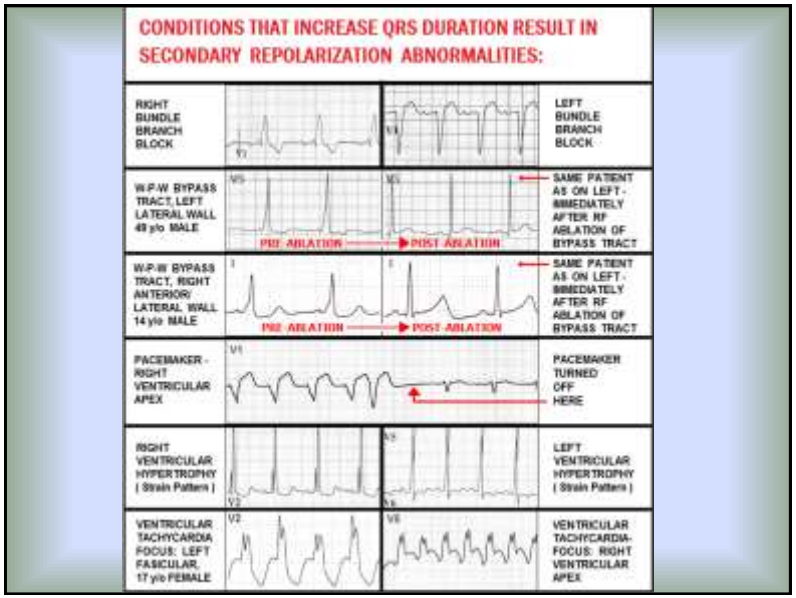
STEP 2: Evaluate J Points, ST-Segment and T waves in EVERY Lead

STEP 1 – evaluate QRS width:

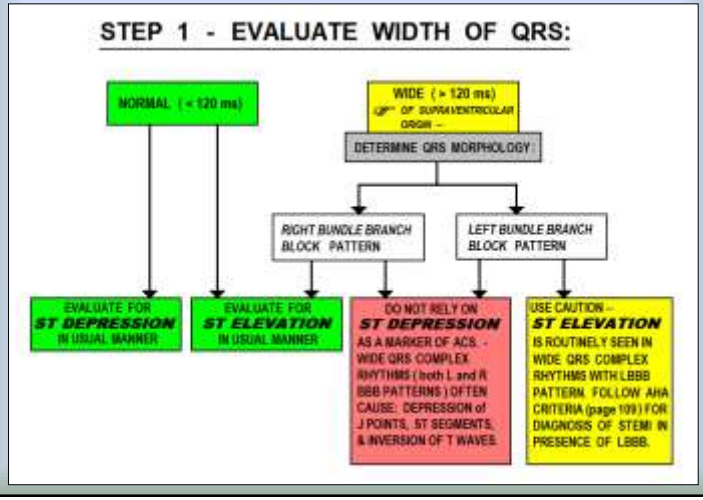
- **QRS is ABNORMALLY WIDE (>120 ms),**
  - **indicates DEPOLARIZATION ABNORMALITY (e.g. "bundle branch block, Wolff-Parkinson-White Syndrome, etc).**

STEP 1 – evaluate QRS width:

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



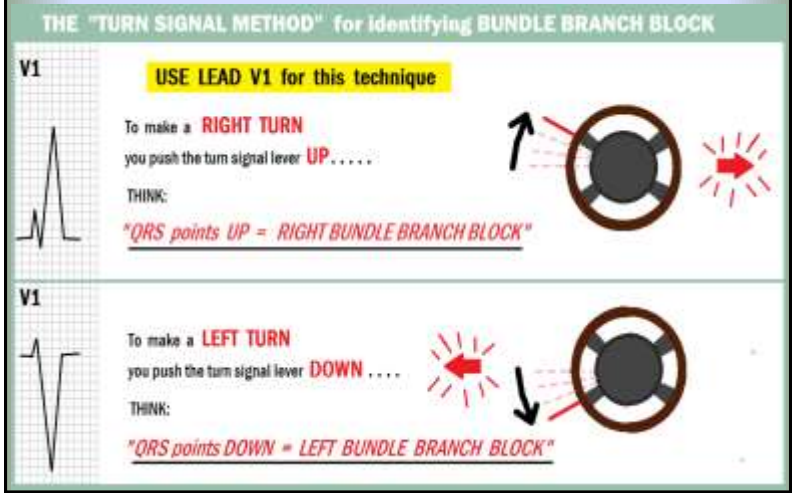
Evaluating the ECG for ACS:



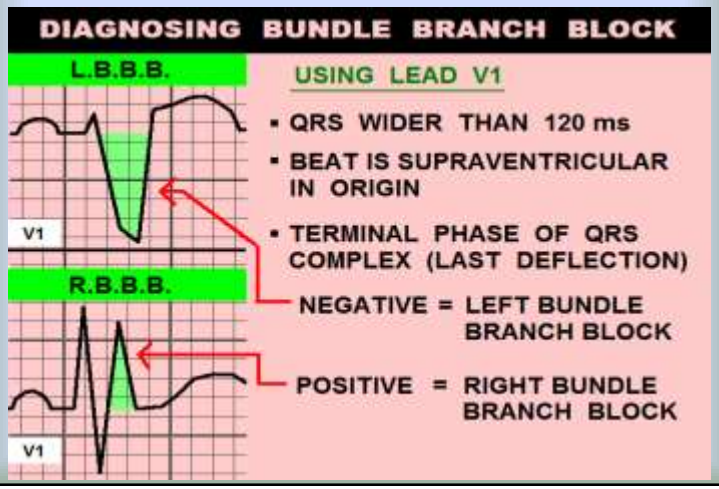
Wide QRS present:
QRSd > 120ms

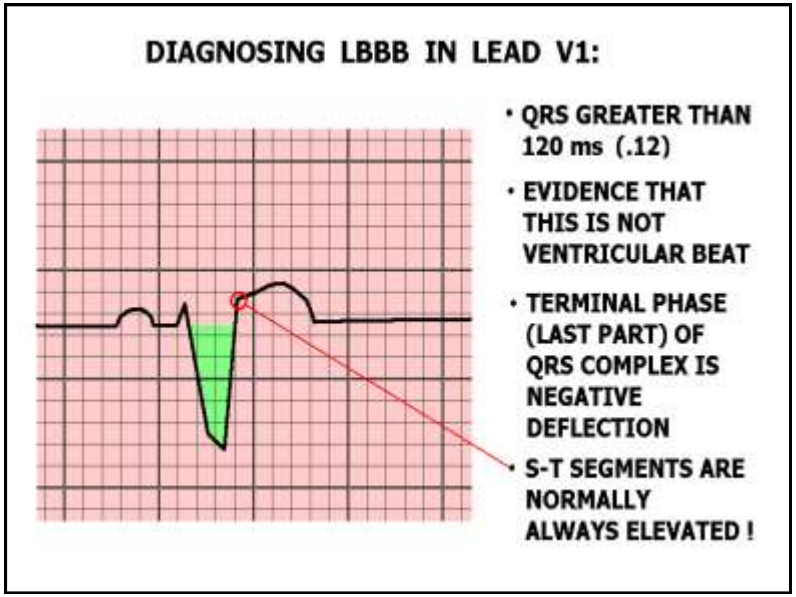
- **Determine RIGHT vs. LEFT Bundle Branch Block Pattern**

Simple "Turn Signal Method" . . .

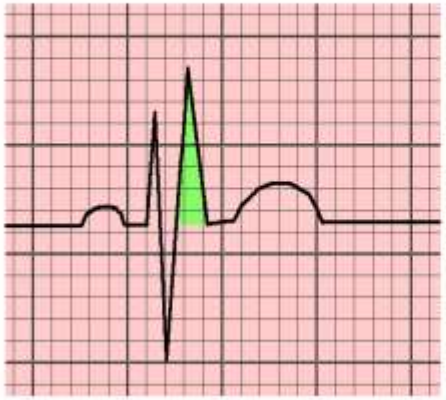


"Terminal Phase of QRS Method". . .





DIAGNOSING RBBB IN LEAD V1:



- WIDER THAN 120 ms (.12)

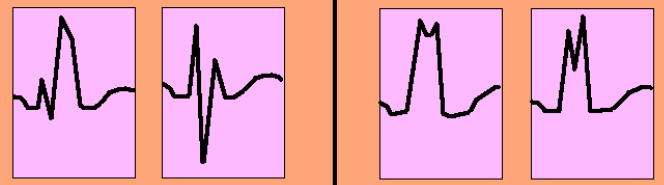
(or 3 little boxes)

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

DIAGNOSING BUNDLE BRANCH BLOCK

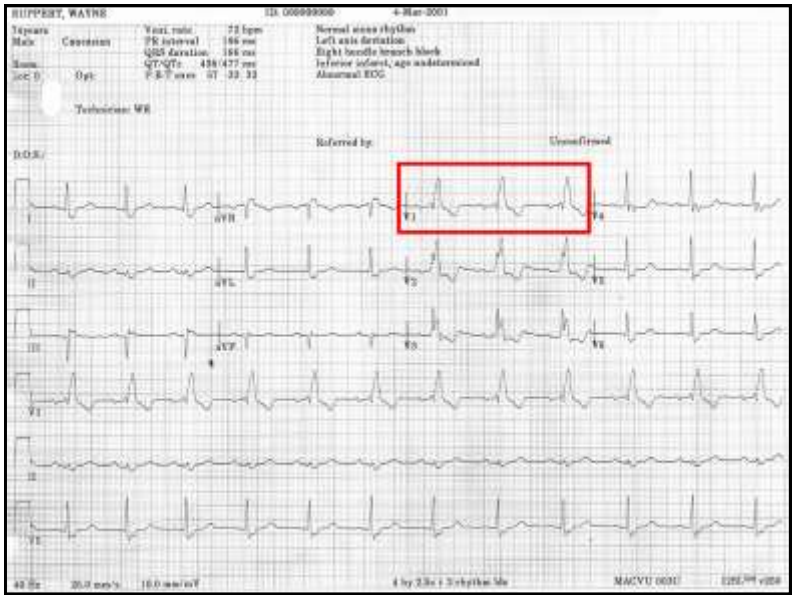
USING LEADS V1, V2, and V5, V6:

LOCATING RsR' or RR' COMPLEXES:



RIGHT BUNDLE BRANCH BLOCK

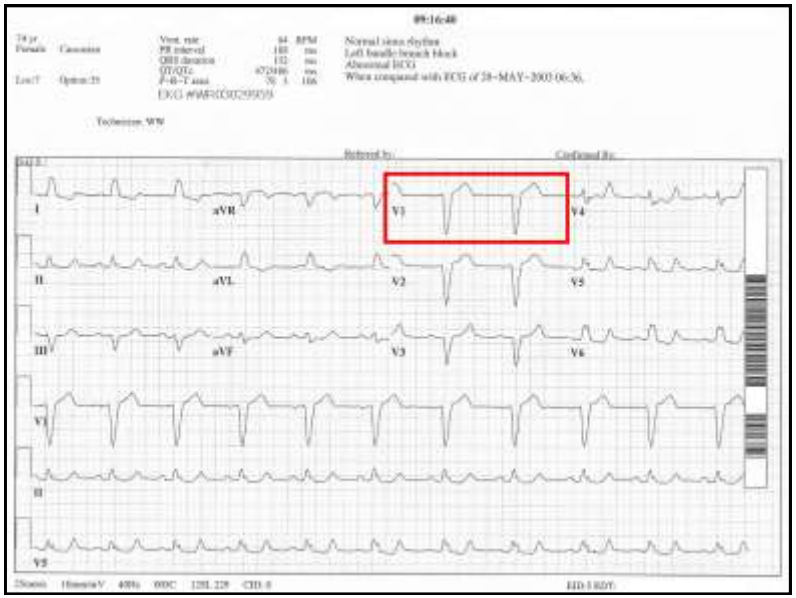
LEFT BUNDLE BRANCH BLOCK

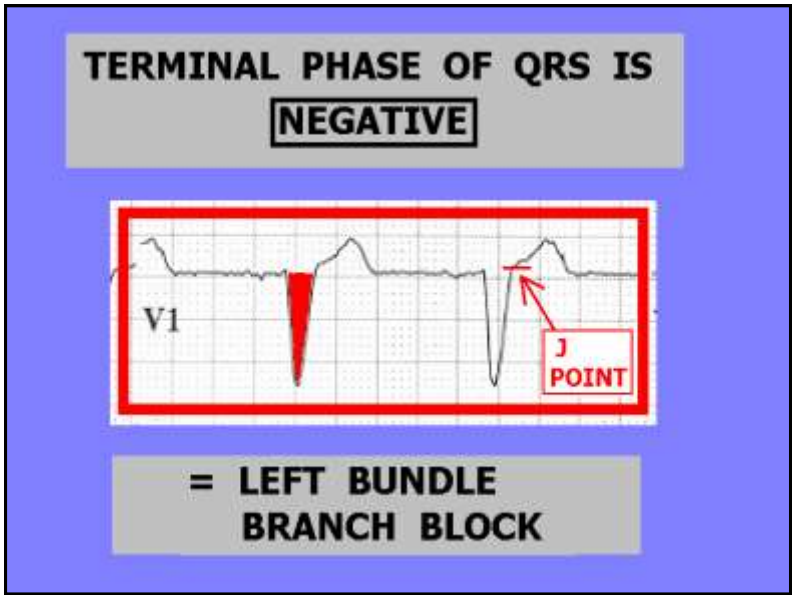


TERMINAL PHASE OF QRS IS

POSITIVE

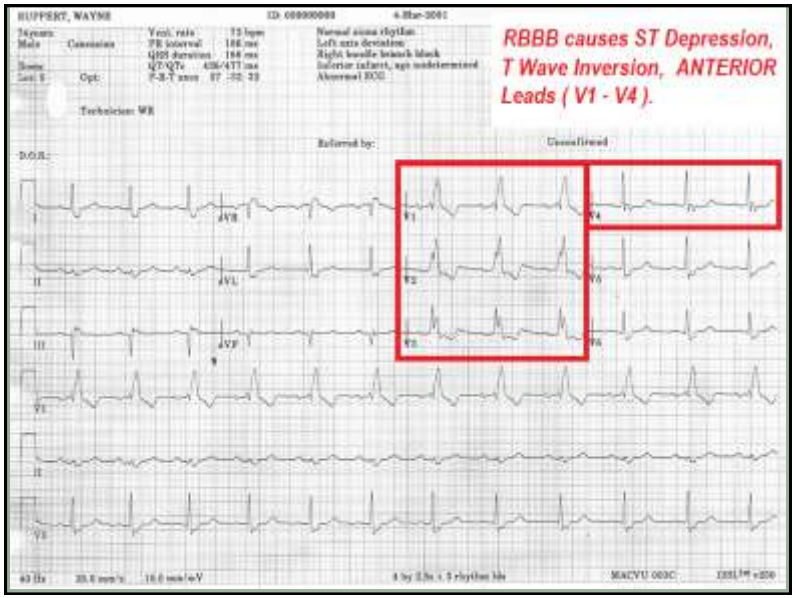
= RIGHT BUNDLE BRANCH BLOCK


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


TERMINAL PHASE OF QRS IS
NEGATIVE
V1
J
POINT
= LEFT BUNDLE
BRANCH BLOCK

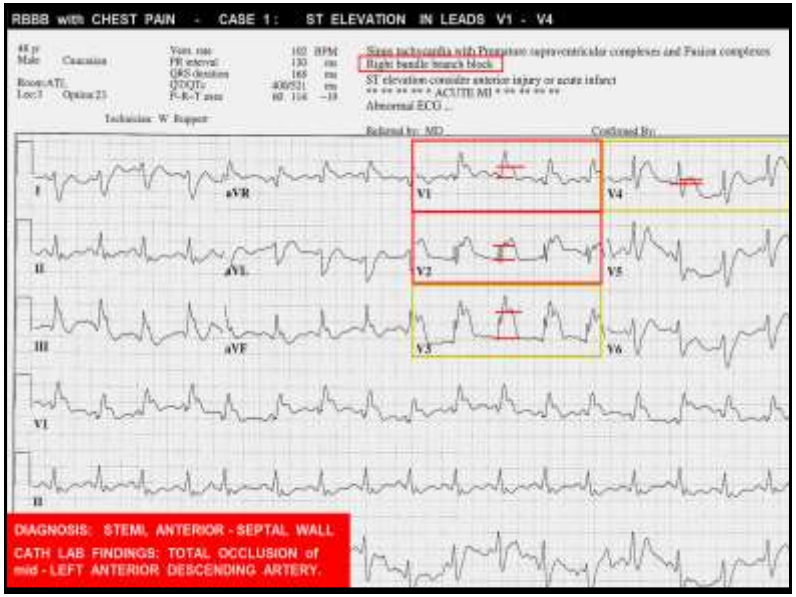
Wide QRS present:
(QRSd > 120ms)

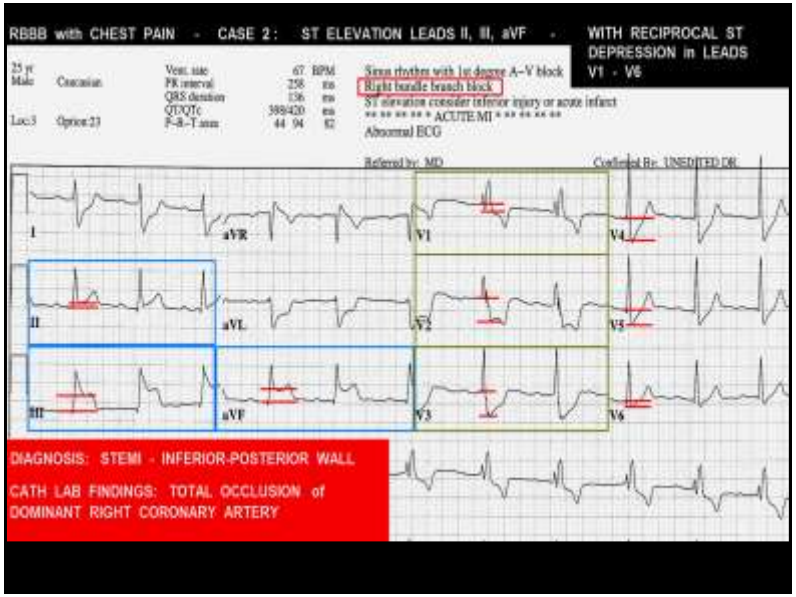
- **When RIGHT Bundle Branch Block pattern is present:**
  - **Precordial Leads typically demonstrate ST Depression and T wave Inversion**

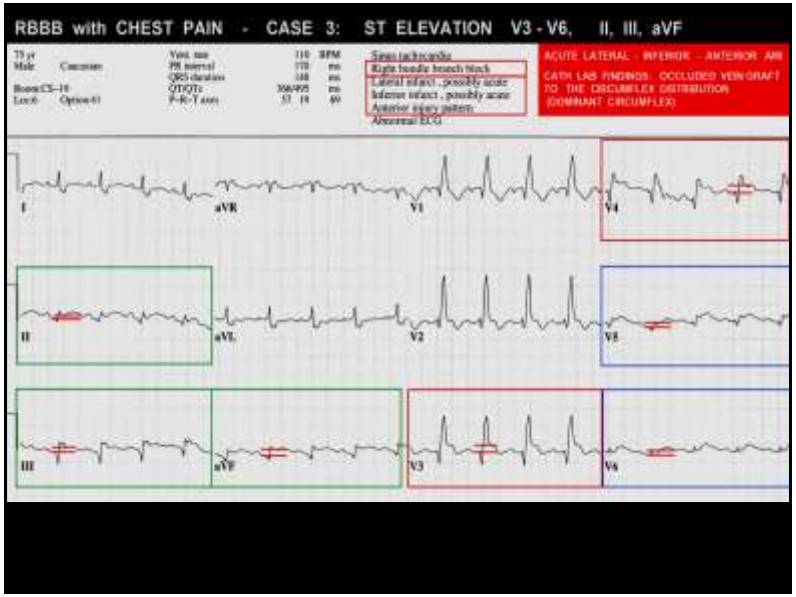


Wide QRS present:
(QRSd > 120ms)

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




RBBB with CHEST PAIN - CASE 2: ST ELEVATION LEADS II, III, aVF - WITH RECIPROCAL ST DEPRESSION in LEADS V1 - V6
Sinus rhythm with 1st degree A-V block
Right bundle branch block
ST elevation consider inferior injury or acute infarct
** ** ** ** * ACUTE MI * ** ** ** **
Abnormal ECG
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
DIAGNOSIS: STEMI - INFERIOR-POSTERIOR WALL
CATH LAB FINDINGS: TOTAL OCCLUSION of DOMINANT RIGHT CORONARY ARTERY


RBBB with CHEST PAIN - CASE 3: ST ELEVATION V3 - V6, II, III, aVF
Sinus tachycardia
Right bundle branch block
Abnormal ECG
ACUTE LATERAL - INFERIOR - ANTERIOR MI
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

Wide QRS present:

(QRSd > 120ms)

- **When LBBB QRS pattern is present:**

Wide QRS present:

(QRSd > 120ms)

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

Wide QRS present:

(QRSd > 120ms)

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

Wide QRS present:

(QRSd > 120ms)

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

Diagnosis of STEMI with LBBB pattern:

2013 ACC/AHA Guideline for Management of STEMI

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

Diagnosis of STEMI with LBBB pattern:

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

Diagnosis of STEMI with LBBB pattern:

2013 ACC/AHA Guideline for Management of STEMI

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

Diagnosis of STEMI with LBBB pattern:

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

A.H.A. ACLS GUIDELINES

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

   for diagnosis of STEMI

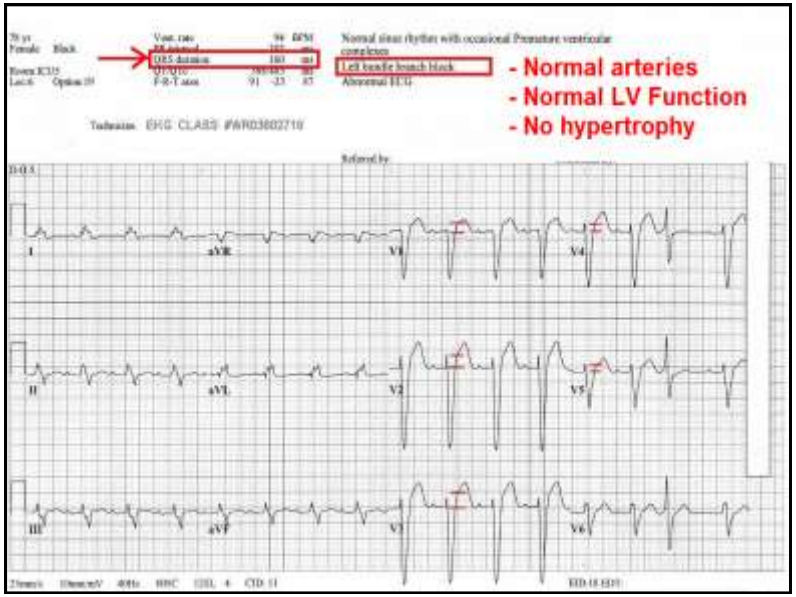
2. If patient has:

   a) previously NORMAL ECGs (no LBBB)

   -- or --

   b) no old ECGs available for comparison

   consider diagnosis as STEMI until proven otherwise.

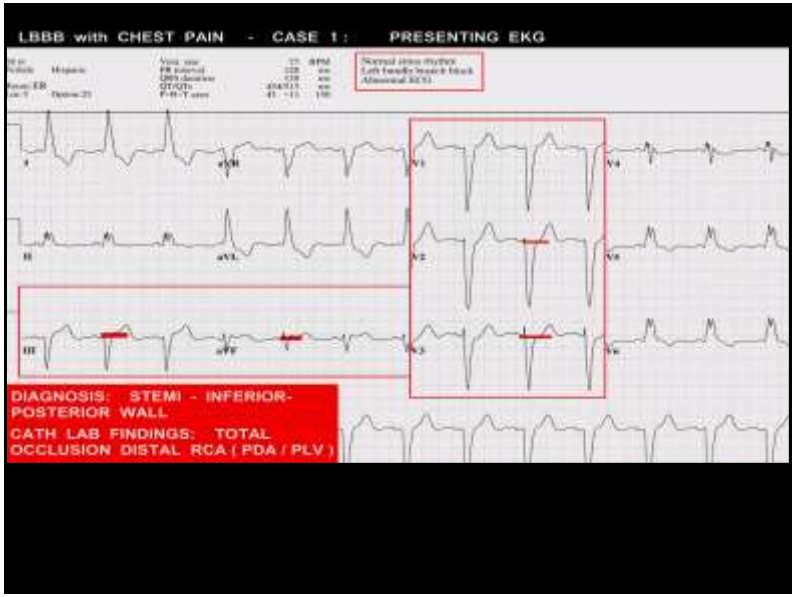


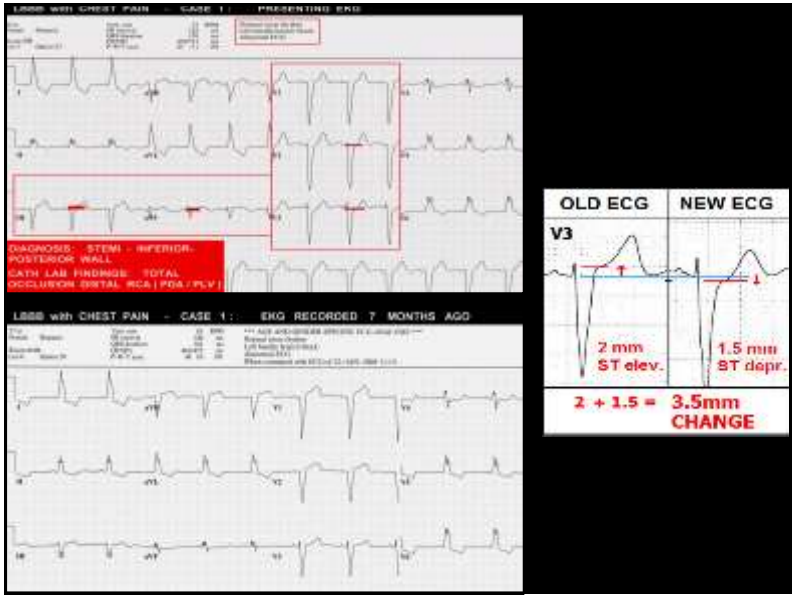
HELPFUL INDICATORS FOR ECG DIAGNOSIS OF STEMI in the presence of LBBB:

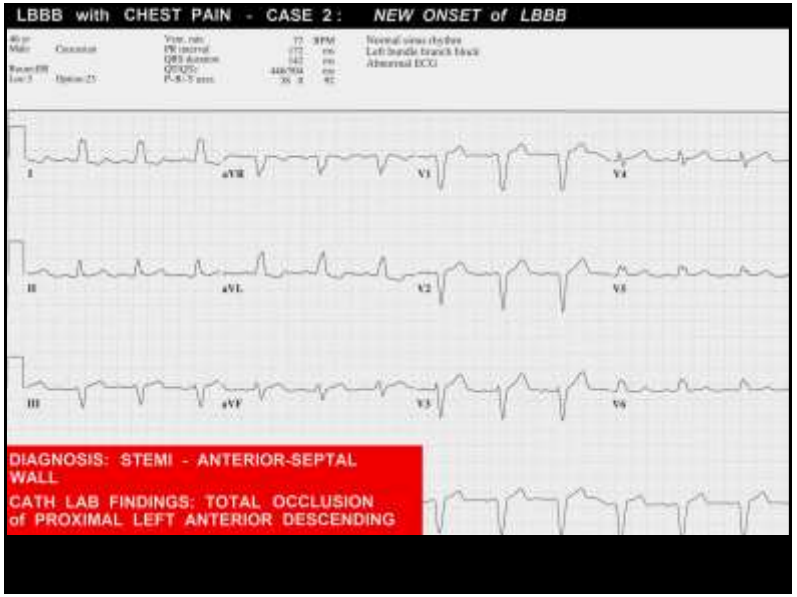
- ST ELEVATION > 5 mm
- COMPARE J POINT, ST SEGMENTS and T WAVES of previous ECG with LBBB to NEW ECG.
- CONVEX ST SEGMENT = poss. MI
  CONCAVE ST SEGMENT = normal
- CONCORDANT ST changes (1 mm or > ST DEPRESSION V1 - V3 or ST ELEVATION LEADS II, III, AVF)
- ST ELEVATION in LEADS II, III, and/or AVF

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

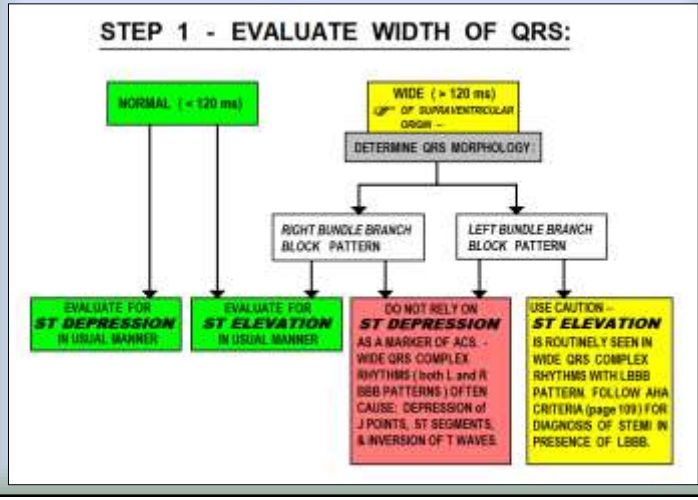
"Electrocardiographic Diagnosis of Evolving Acute Myocardial Infarction in the Presence of Left Bundle-Branch Block" Birnbaum et al, N Engl J Med 1996; 334:481-487


LBBB with CHEST PAIN - CASE 1 : PRESENTING EKG
DIAGNOSIS: STEMI - INFERIOR-POSTERIOR WALL
CATH LAB FINDINGS: TOTAL OCCLUSION DISTAL RCA (PDA / PLV)


LBBB with CHEST PAIN - CASE 1 : EKG RECORDED 7 MONTHS AGO
OLD ECG
NEW ECG
V3
2 mm ST elev.
1.5 mm ST depr.
2 + 1.5 = 3.5mm CHANGE



Evaluating the ECG for ACS:



Evaluating the ECG for ACS:

Patients with Normal Width QRS (QRSd < 120ms)

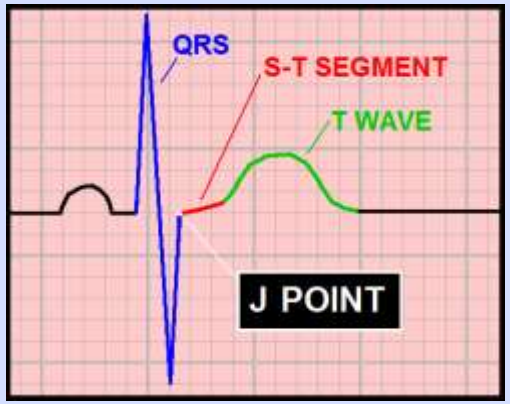
STEP 2 - EVALUATE the EKG for ACS

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

- J POINTS
- ST SEGMENTS
- T WAVES

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

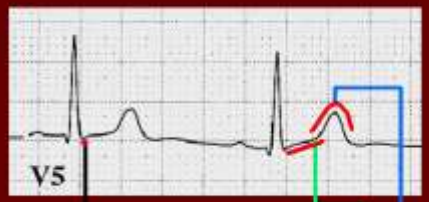
Defining NORMAL – QRS <120ms:



When QRS duration is NORMAL (< 120 ms):

NORMAL ST - T WAVES

- WHEN QRS WIDTH IS NORMAL (< 120 ms)



ASSESS:

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

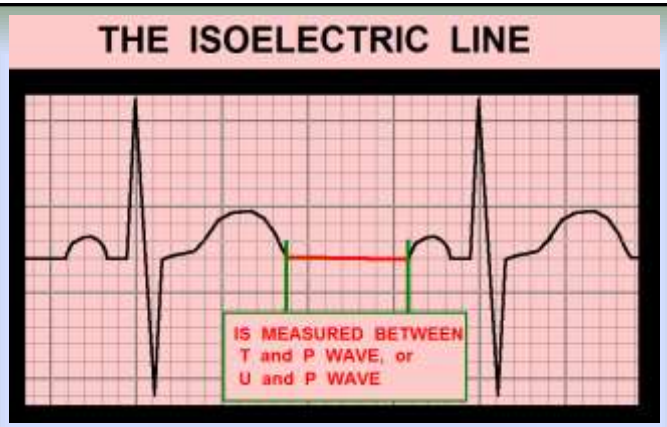
in EVERY LEAD EXCEPT aVR ! !

THE J POINT SHOULD BE . .

WITHIN 1 mm ABOVE

OR

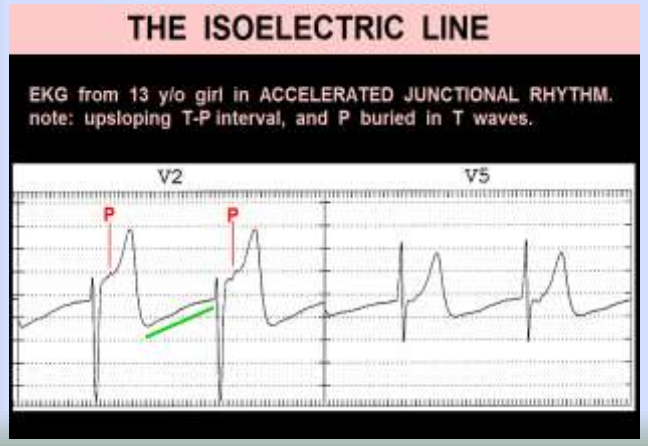
BELOW the ISOELECTRIC LINE



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

The Isoelectric Line -

it's not always isoelectric !



THE P-Q JUNCTION

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

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

Use the P-Q junction as a reference point for measuring the J Point and ST-Segment when "iso-electric line is not iso-electric !

THE P-Q JUNCTION

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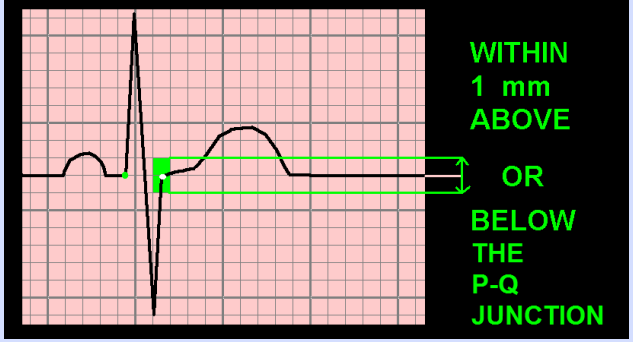
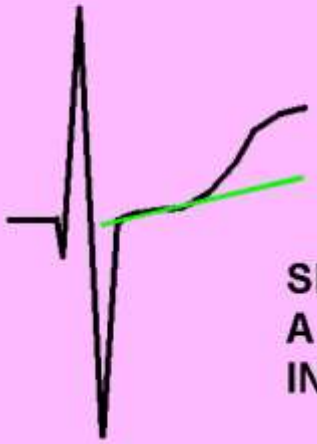
— as per the A.H.A., A.C.C., and WANG, ASINGER, and MARRIOTT, N.E.J.M. vol. 349:2128-2135 Nov. 27, 2003

Defining NORMAL:

THE J POINT SHOULD BE . .

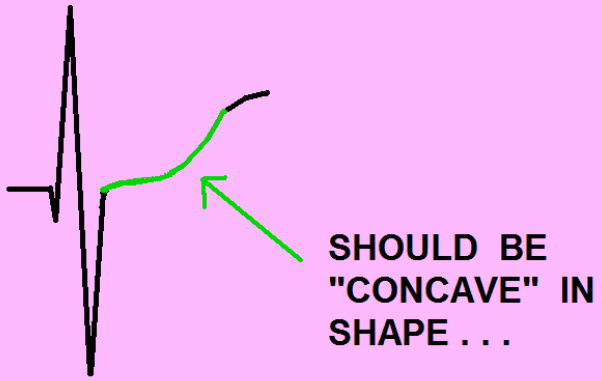


THE S-T SEGMENT

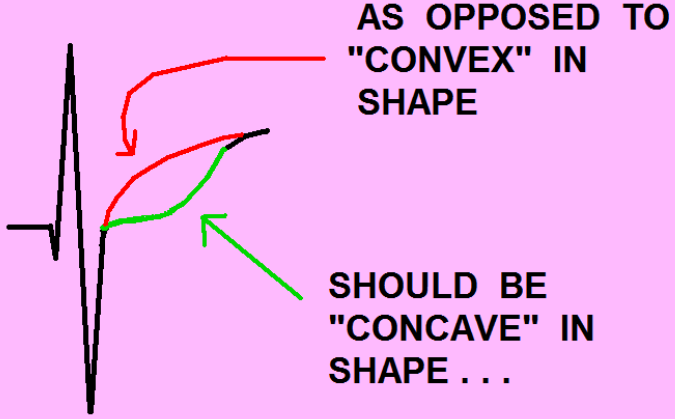


SHOULD HAVE
A "SLIGHT POSITIVE"
INCLINATION

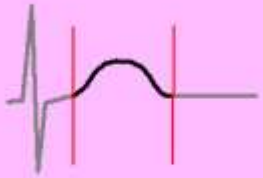
THE S-T SEGMENT



THE S-T SEGMENT

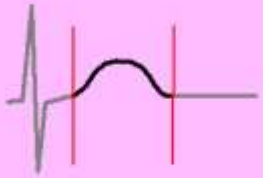


THE T WAVE



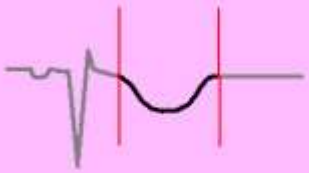
- SHOULD BE A "NICE," ROUNDED, CONVEX SHAPE
- SHOULD BE SYMMETRICAL

THE T WAVE



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

THE T WAVE

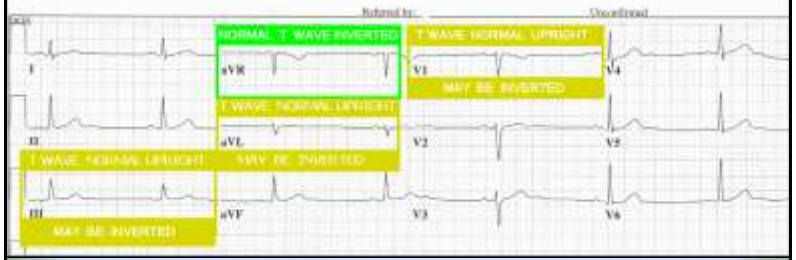


LEAD AVR

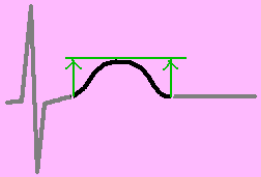
- REMEMBER, IN LEAD AVR *EVERYTHING* IS "UPSIDE-DOWN"

Normal Variants: *T Wave Inversion*

Leads where the T WAVE may be INVERTED:



THE T WAVE



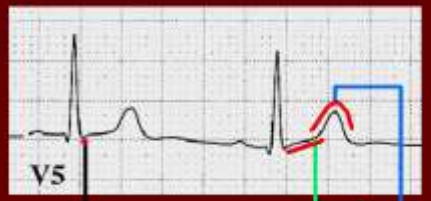
AMPLITUDE GUIDELINES:

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

When QRS duration is NORMAL (< 120 ms):

NORMAL ST - T WAVES

- WHEN QRS WIDTH IS NORMAL (< 120 ms)



ASSESS:

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

☞ *in EVERY LEAD EXCEPT aVR ! !*

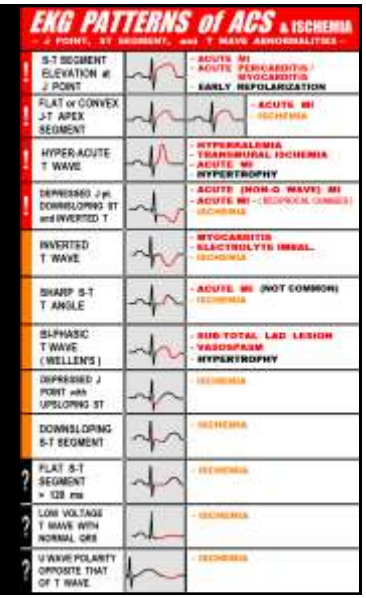
ECG Indicators of ACS in Patients with *Normal Width* QRS Complexes (QRS duration < 120 ms)

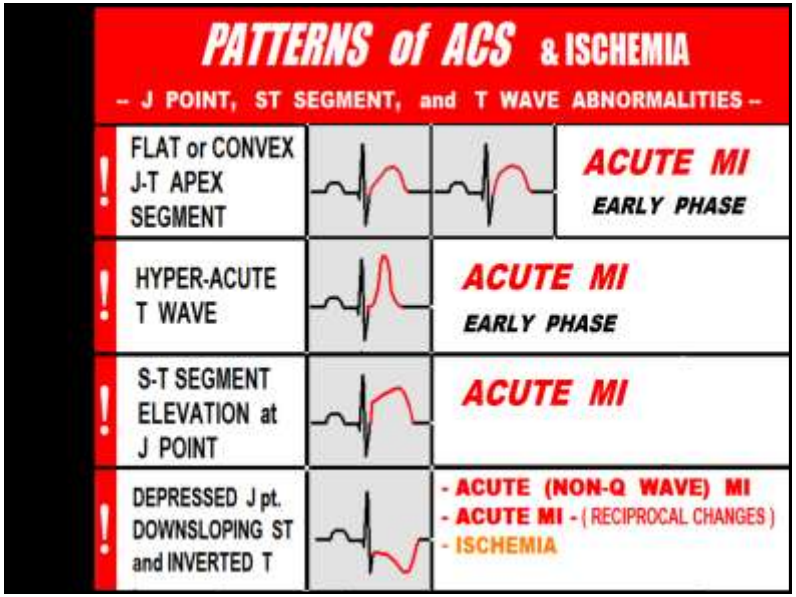
Multiple patterns of ABNORMAL:
- J Point
- ST-Segment
- T Wave

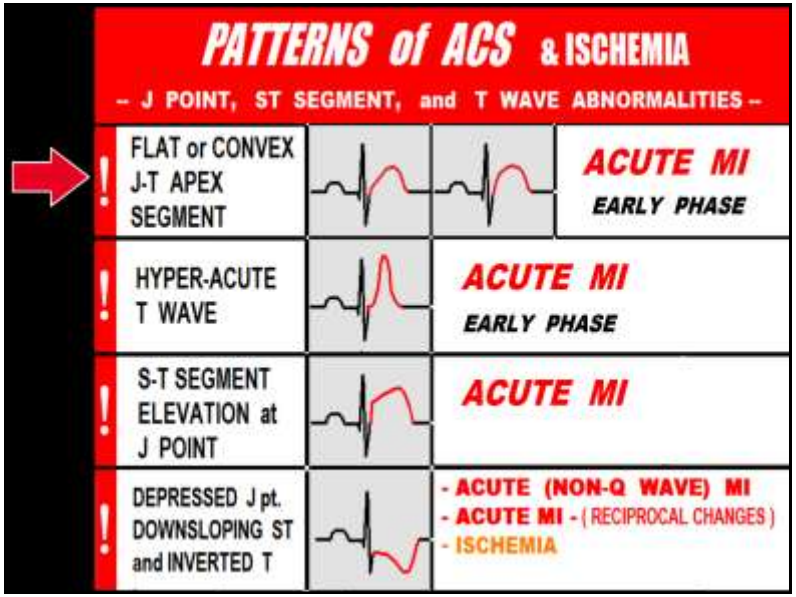
configurations may indicate ACS.

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

BOOK PAGE: 83




PATTERNS of ACS & ISCHEMIA
– J POINT, ST SEGMENT, and T WAVE ABNORMALITIES –
FLAT or CONVEX J-T APEX SEGMENT
ACUTE MI
EARLY PHASE
HYPER-ACUTE T WAVE
ACUTE MI
EARLY PHASE
S-T SEGMENT ELEVATION at J POINT
ACUTE MI
DEPRESSED J pt. DOWNSLOPING ST and INVERTED T
- ACUTE (NON-Q WAVE) MI
- ACUTE MI - (RECIPROCAL CHANGES)
- ISCHEMIA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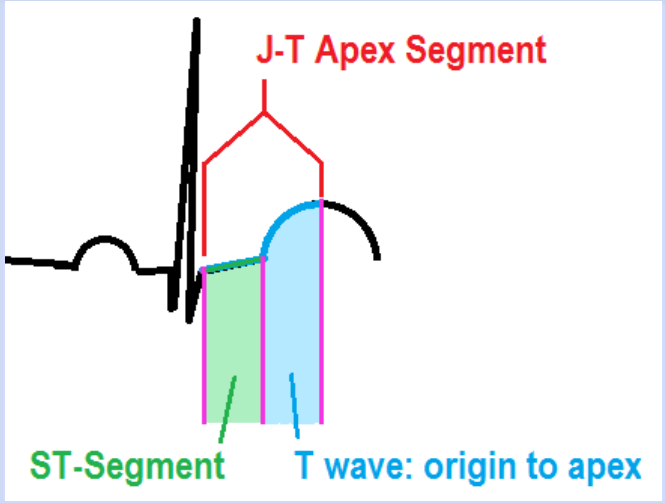
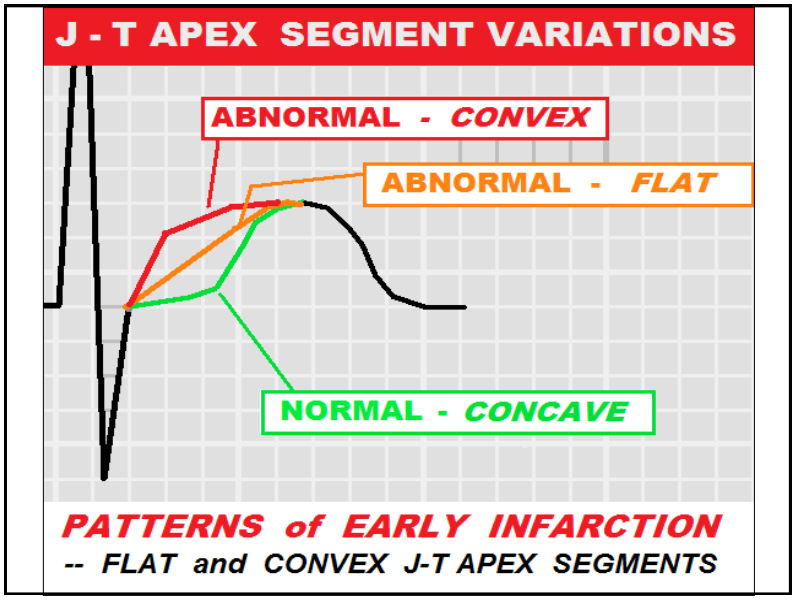
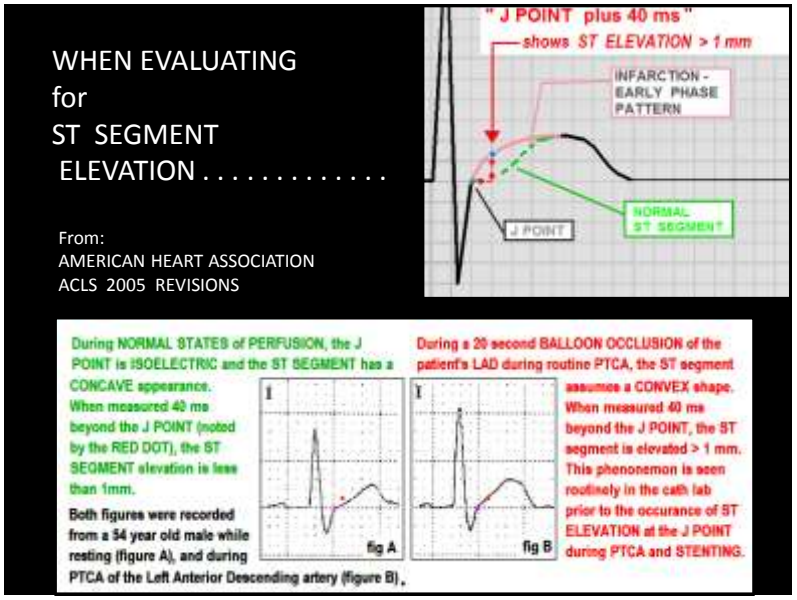
DEPRESSED J pt. DOWNSLOPING ST and INVERTED T
- ACUTE (NON-Q WAVE) MI
- ACUTE MI - (RECIPROCAL CHANGES)
- ISCHEMIA

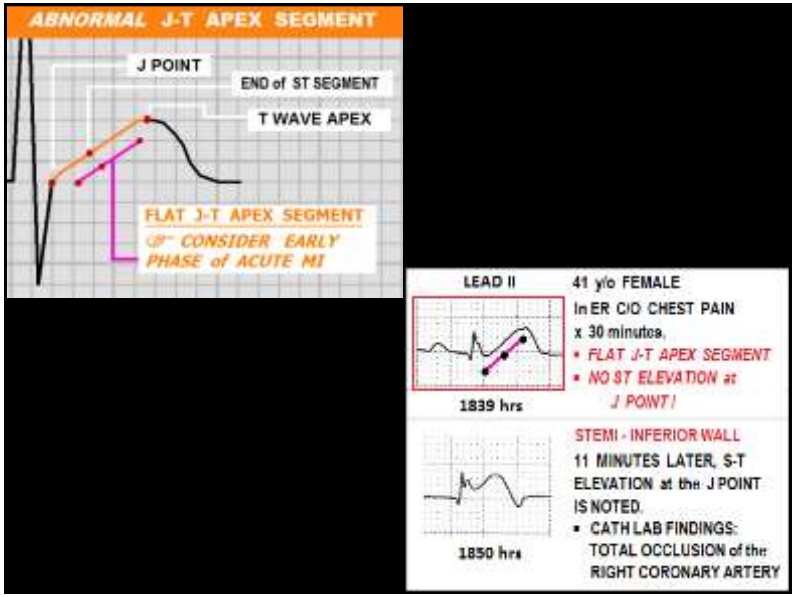
ECG Patterns associated with "EARLY PHASE MI:"

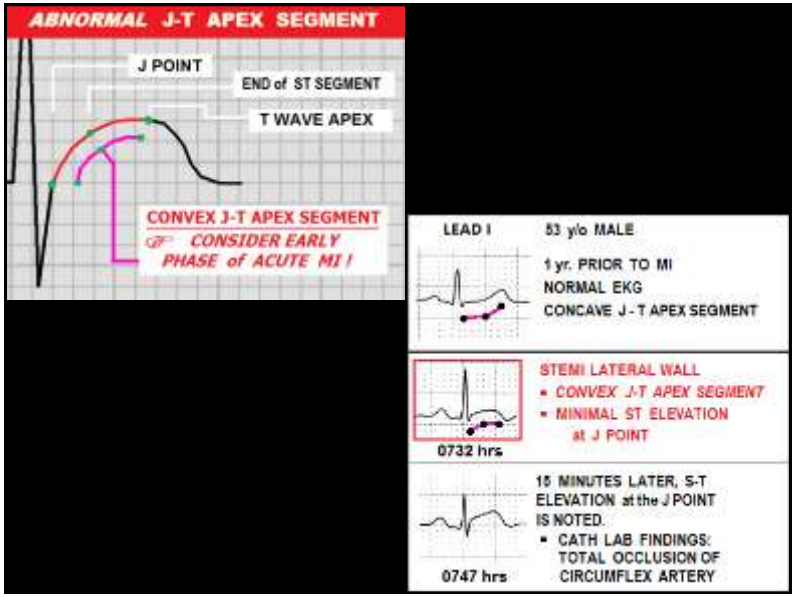
- ***J-T Apex abnormalities***
- ***Hyper-Acute T Waves***
- ***ST-T Wave Changes***




J - T APEX SEGMENT VARIATIONS
ABNORMAL - CONVEX
ABNORMAL - FLAT
NORMAL - CONCAVE
PATTERNS of EARLY INFARCTION
-- FLAT and CONVEX J-T APEX SEGMENTS


WHEN EVALUATING
for
ST SEGMENT
ELEVATION . . . . . .
From:
AMERICAN HEART ASSOCIATION
ACLS 2005 REVISIONS
" J POINT plus 40 ms "
shows ST ELEVATION > 1 mm
INFARCTION - EARLY PHASE PATTERN
J POINT
NORMAL ST SEGMENT
During NORMAL STATES of PERFUSION, the J POINT is ISOELECTRIC and the ST SEGMENT has a CONCAVE appearance. When measured 40 ms beyond the J POINT (noted by the RED DOT), the ST SEGMENT elevation is less than 1mm.
Both figures were recorded from a 54 year old male while resting (figure A), and during PTCA of the Left Anterior Descending artery (figure B).
fig A
fig B
During a 20 second BALLOON OCCLUSION of the patient's LAD during routine PTCA, the ST segment assumes a CONVEX shape. When measured 40 ms beyond the J POINT, the ST segment is elevated > 1 mm. This phenonemon is seen routinely in the cath lab prior to the occurance of ST ELEVATION at the J POINT during PTCA and STENTING.


ABNORMAL J-T APEX SEGMENT
J POINT
END of ST SEGMENT
T WAVE APEX
FLAT J-T APEX SEGMENT
CONSIDER EARLY PHASE of ACUTE MI
LEAD II
1839 hrs
41 y/o FEMALE
In ER C/O CHEST PAIN x 30 minutes.
• FLAT J-T APEX SEGMENT
• NO ST ELEVATION at J POINT!
1850 hrs
STEMI - INFERIOR WALL
11 MINUTES LATER, S-T ELEVATION at the J POINT IS NOTED.
• CATH LAB FINDINGS: TOTAL OCCLUSION of the RIGHT CORONARY ARTERY


ABNORMAL J-T APEX SEGMENT
J POINT
END of ST SEGMENT
T WAVE APEX
CONVEX J-T APEX SEGMENT
CONSIDER EARLY PHASE of ACUTE MI!
LEAD I
53 y/o MALE
1 yr. PRIOR TO MI
NORMAL EKG
CONCAVE J-T APEX SEGMENT
0732 hrs
STEMI LATERAL WALL
• CONVEX J-T APEX SEGMENT
• MINIMAL ST ELEVATION at J POINT
0747 hrs
15 MINUTES LATER, S-T ELEVATION at the J POINT IS NOTED.
• CATH LAB FINDINGS: TOTAL OCCLUSION OF CIRCUMFLEX ARTERY

CASE STUDY: ABNORMAL J-T APEX SEGMENTS

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

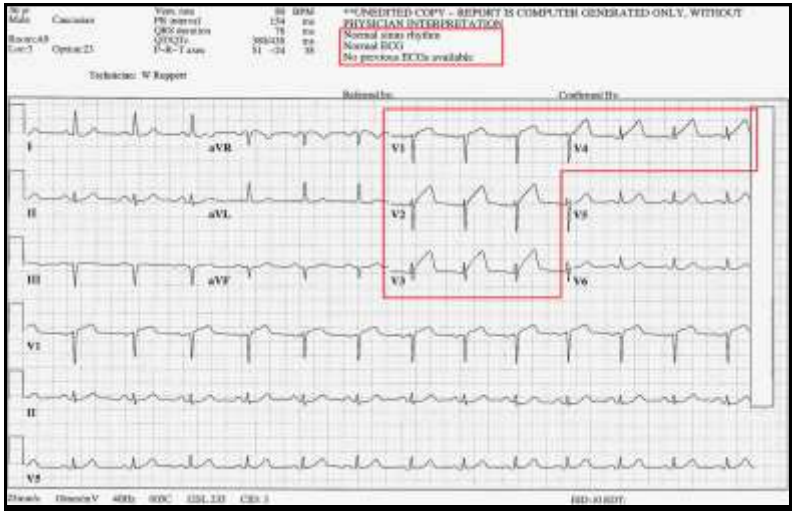
RISK FACTOR PROFILE:

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

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

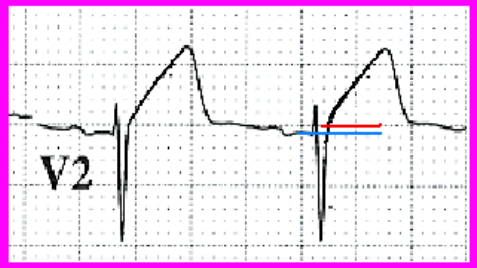
VITAL SIGNS: BP 142/94, P 80, R 20, SAO2 98%

LABS: JUST OBTAINED, RESULTS NOT AVAILABLE YET.



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

measurement of S-T elevation



S-T elevation at J point = 0.5 mm

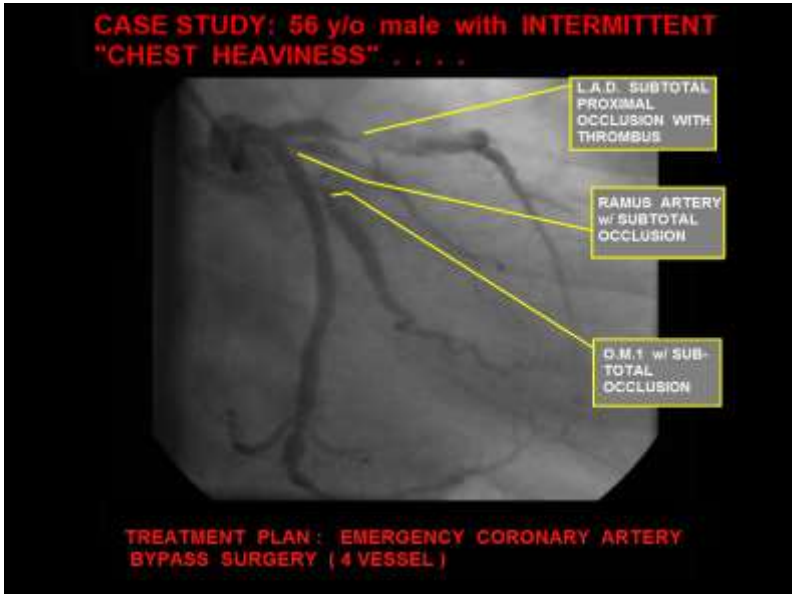
ACUTE MI = S-T elev. > 1.0 mm

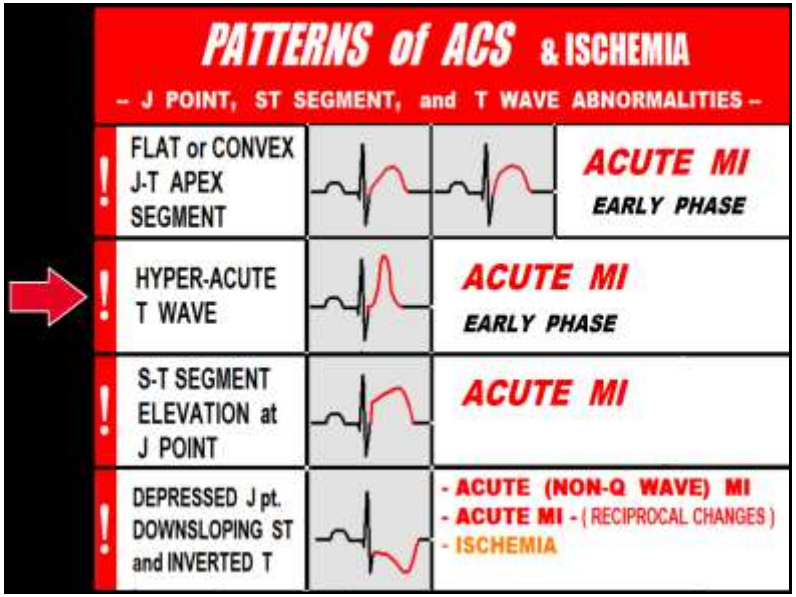
measurement of S-T elevation
by "J point + .04" method

S-T elevation at J point = 0.5 mm

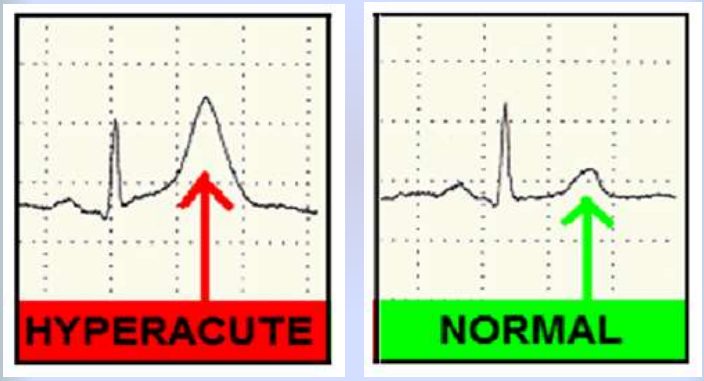
S-T elevation at J + .04 = 2.0 mm

ACUTE MI = S-T elev. > 1.0 mm

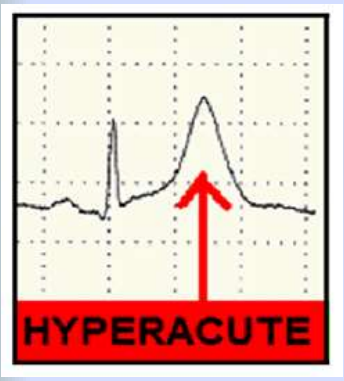

CASE STUDY: 56 y/o male with INTERMITTENT "CHEST HEAVINESS" . . . .
L.A.D. SUBTOTAL PROXIMAL OCCLUSION WITH THROMBUS
RAMUS ARTERY w/ SUBTOTAL OCCLUSION
O.M.1 w/ SUB-TOTAL OCCLUSION
TREATMENT PLAN : EMERGENCY CORONARY ARTERY BYPASS SURGERY (4 VESSEL)


PATTERNS of ACS & ISCHEMIA
– J POINT, ST SEGMENT, and T WAVE ABNORMALITIES –
FLAT or CONVEX J-T APEX SEGMENT
ACUTE MI
EARLY PHASE
HYPER-ACUTE T WAVE
ACUTE MI
EARLY PHASE
S-T SEGMENT ELEVATION at J POINT
ACUTE MI
DEPRESSED J pt. DOWNSLOPING ST and INVERTED T
- ACUTE (NON-Q WAVE) MI
- ACUTE MI - (RECIPROCAL CHANGES)
- ISCHEMIA

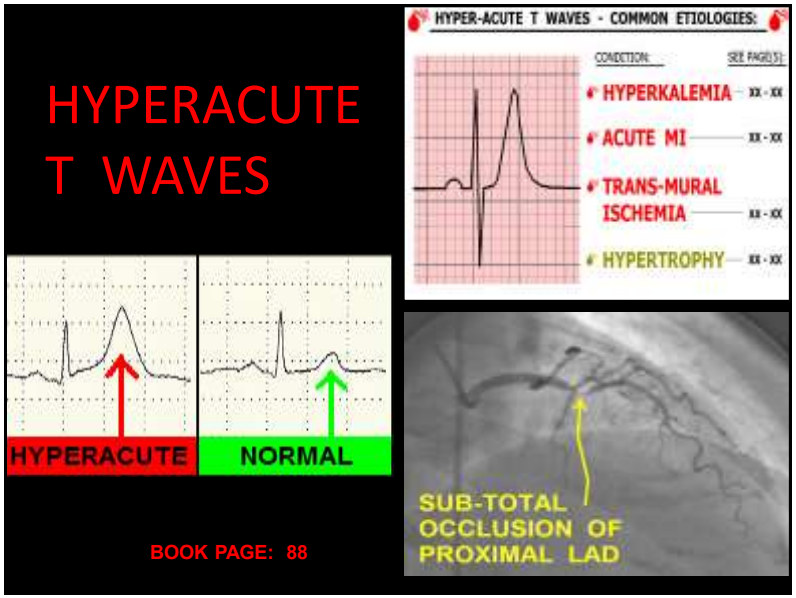
T waves should not be HYPERACUTE



HYPERACUTE T Waves may indicate:

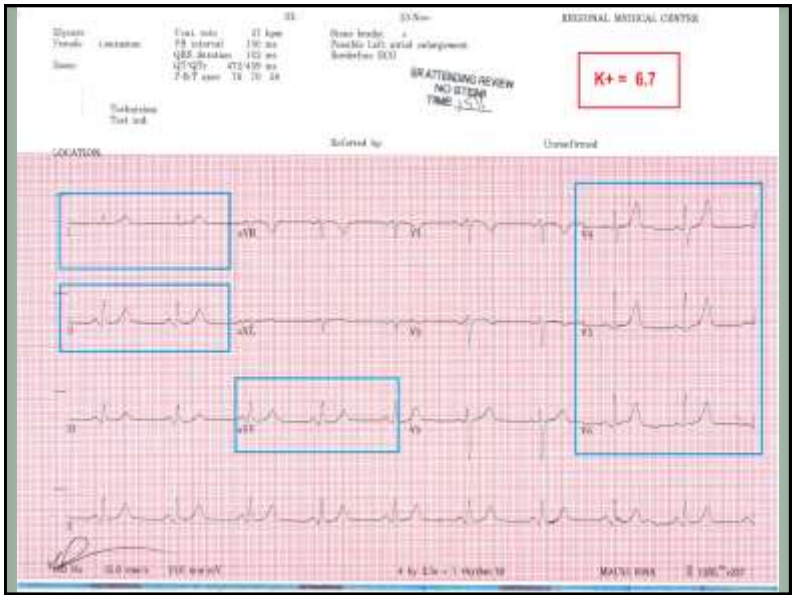


- **Early phase Acute MI**
- **Transmural ischemia** (usually seen in one region of the ECG)
- **Hyperkalemia** (seen globally across ECG)
- **Hypertrophy**



Helpful Clue: Hyper-Acute T Waves

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



Helpful Clue: Hyper-Acute T Waves

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

CASE STUDY: HYPERACUTE T WAVES

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

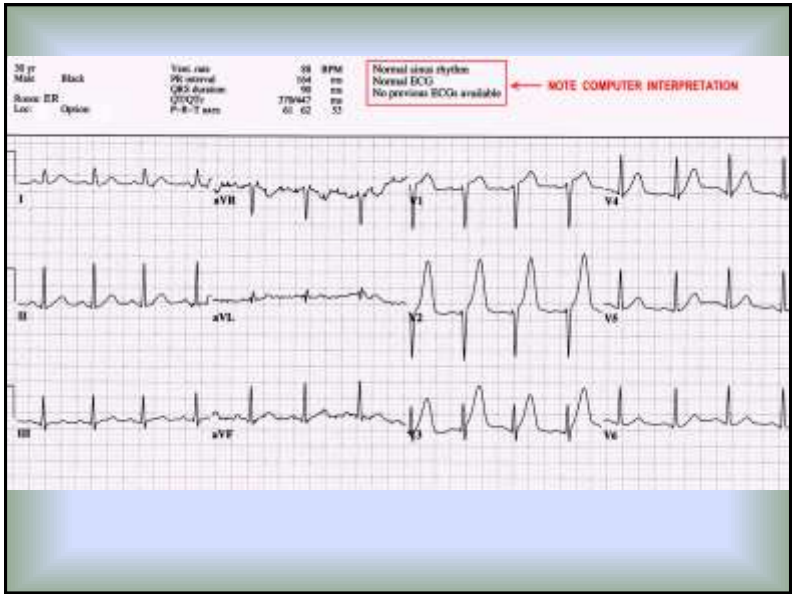
30 y/o male presents to ER via EMS, c/o sudden onset of dull chest pain x 40 min. Pain level varies, not effected by position, movement or deep inspiration. No associated symptoms.

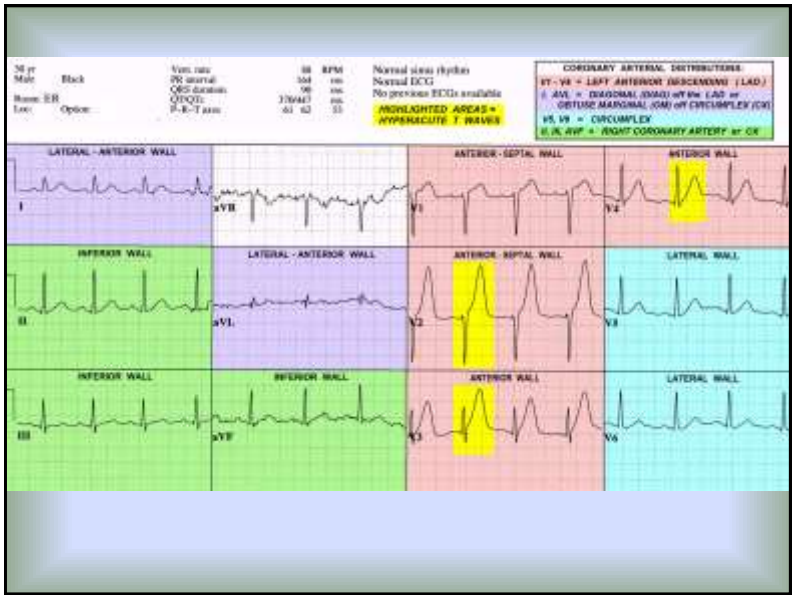
RISK FACTOR PROFILE: NONE. CHOLESTEROL UNKNOWN.

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

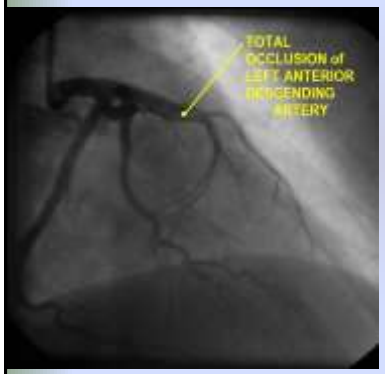
VITAL SIGNS: BP 136/88 P 90 R 20 SAO2 98%

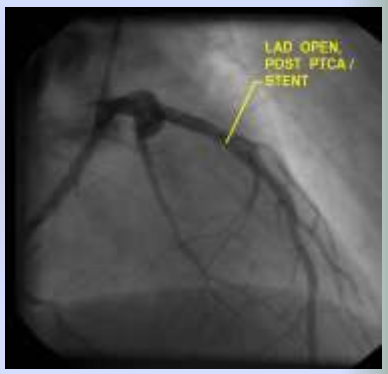
DIAGNOSTIC TESTING: 1st TROPONIN I - ultra: <0.07





Cath Lab findings:





Dynamic ST-T Wave Changes:

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

Dynamic ST-T Wave Changes:

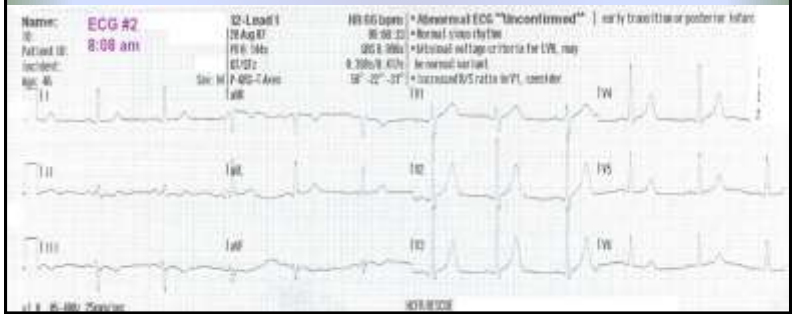
- Other than HEART RATE related variations (which affect intervals), ***J Points, ST-Segments and T Waves SHOULD NOT CHANGE.***
- **When changes to J Points, ST-Segments and/or T waves are NOTED, consider EVOLVING MYOCARDIAL ISCHEMIA and/or EARLY PHASE MI, until proven otherwise.**

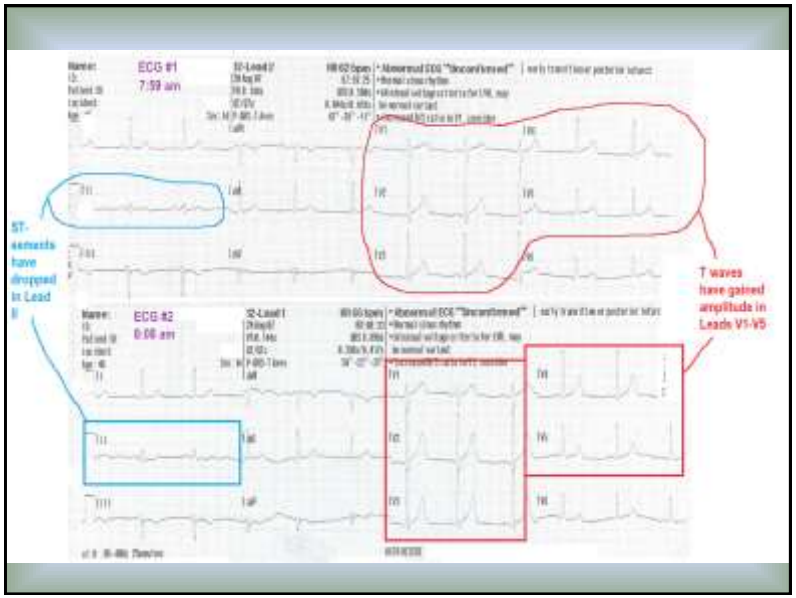
46 year old male

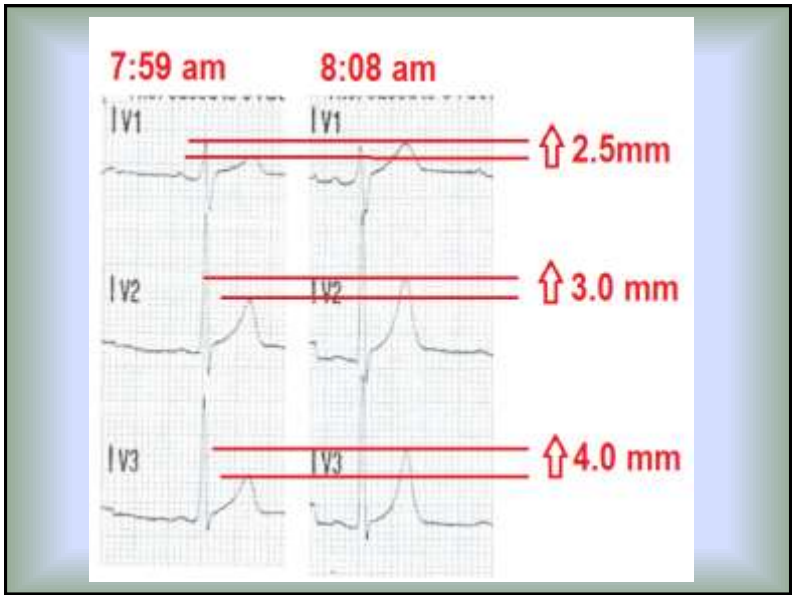
- Exertional dyspnea X “several weeks”
- Intermittent chest pressure X last 3 hours. Currently pain free.

46 year old male: ECG 1

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



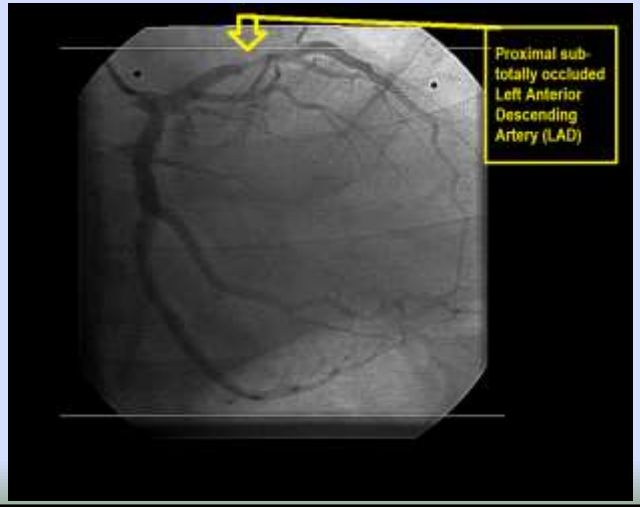

ECG #1
7:59 am
ECG #2
8:08 am
ST-segments have dropped in Lead II
T waves have gained amplitude in Leads V1-V5


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

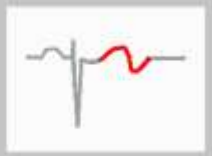
ST-Segment Depression

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

Cath Lab Angiography:



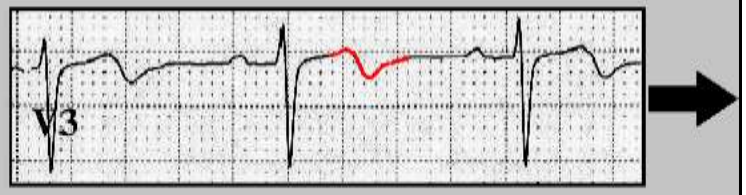
ISCHEMIA



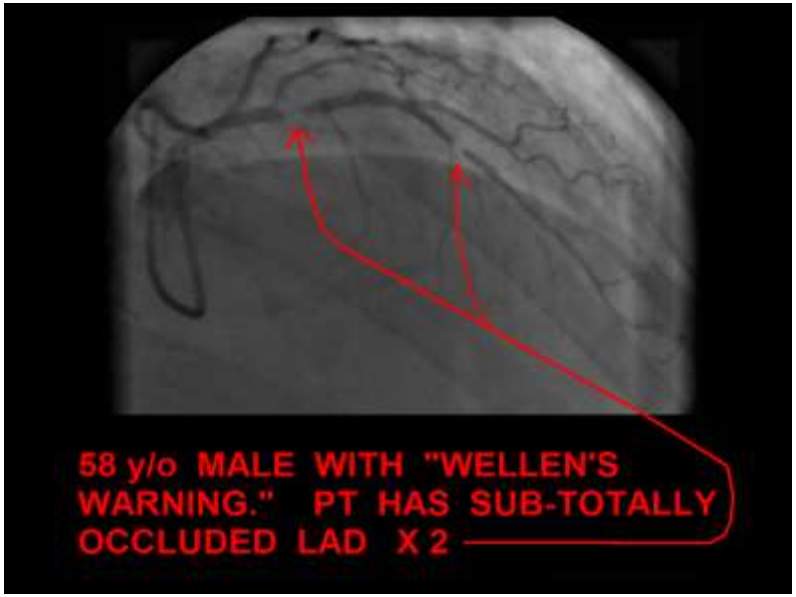
BI-PHASIC T WAVE

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

BI-PHASIC T WAVES



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



Classic "Wellen's Syndrome:"

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

Wellen's Syndrome ETIOLOGY:

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

Wellen's Syndrome EPIDEMIOLOGY & PROGNOSIS:

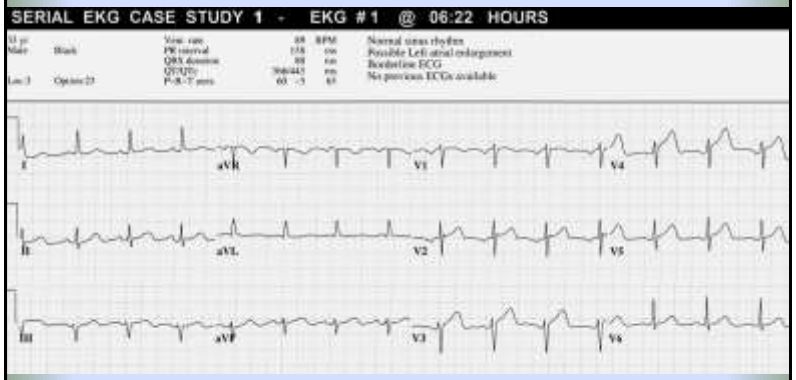
- Present in 14-18% of patients admitted with unstable angina
- 75% patients not treated developed extensive Anterior MI within 3 weeks.
- ***Median Average time from presentation to Acute Myocardial Infarction – 8 days***

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

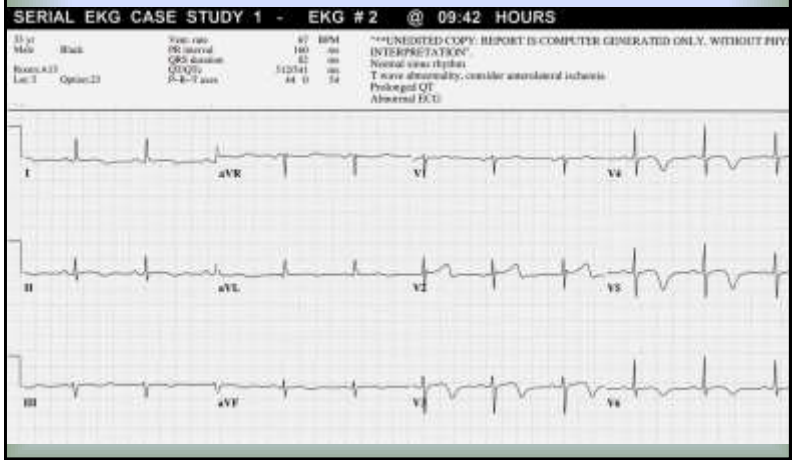
Wellen's Syndrome Case Study

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

Wellen's Syndrome Case Study



Wellen's Syndrome Case Study

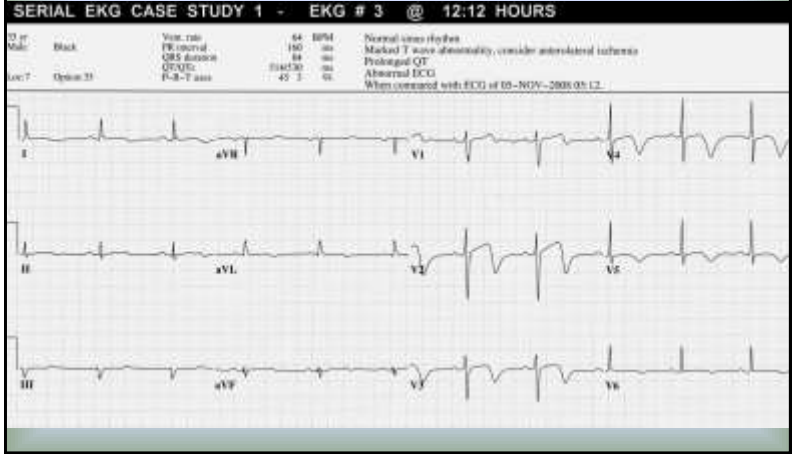


DYNAMIC ST-T Wave Changes ARE PRESENT !!

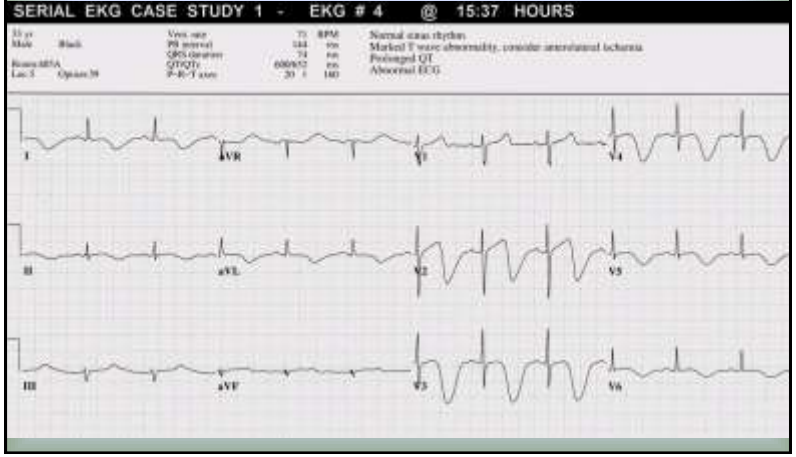
NOW

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

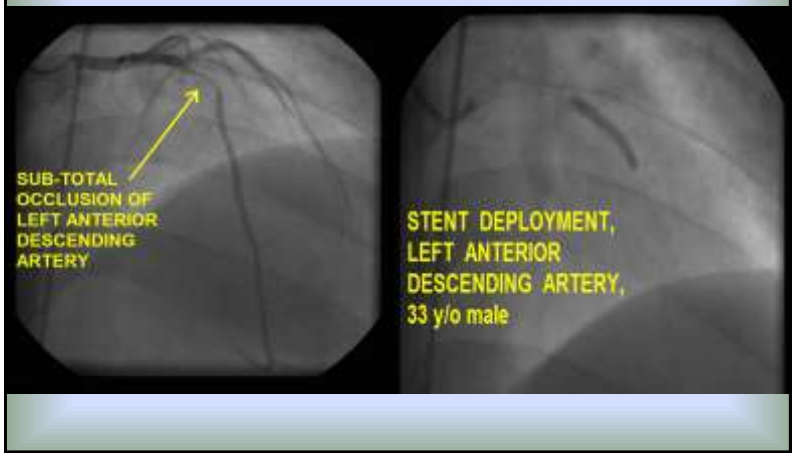
Wellen's Syndrome Case Study



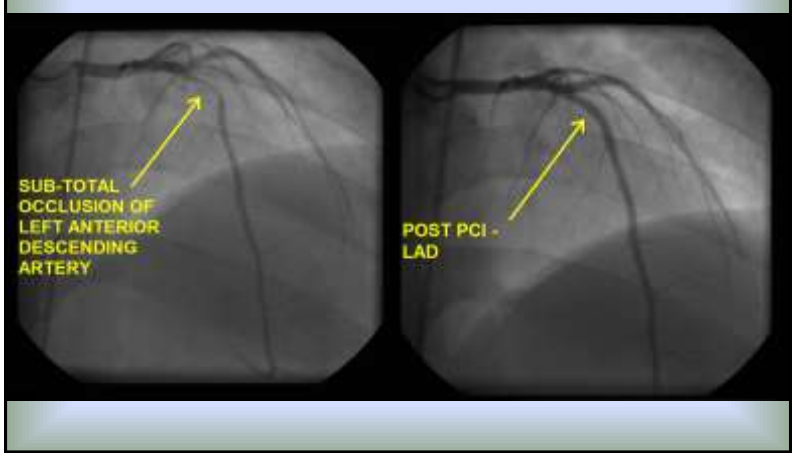
Wellen's Syndrome Case Study



Wellen's Syndrome Case Study

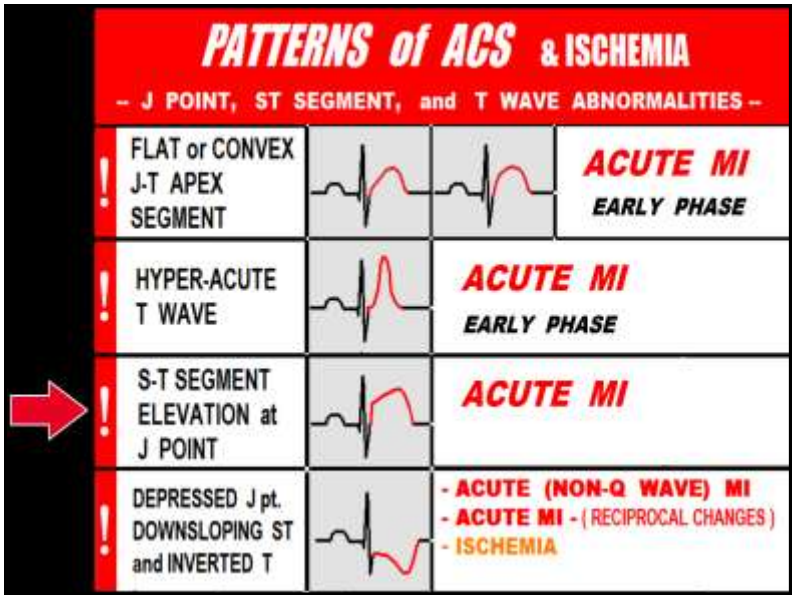


Wellen's Syndrome Case Study



Additional Resources:

- Wellen's Syndrome, NEJM case study



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

Recommendations

1. For men 40 years of age and older, the threshold value for abnormal J-point elevation should be 0.2 mV (2 mm) in leads V_2 and V_3 and 0.1 mV (1 mm) in all other leads.
2. For men less than 40 years of age, the threshold values for abnormal J-point elevation in leads V_2 and V_3 should be 0.25 mV (2.5 mm).
3. For women, the threshold value for abnormal J-point elevation should be 0.15 mV (1.5 mm) in leads V_2 and V_3 and greater than 0.1 mV (1 mm) in all other leads.
4. For men and women, the threshold for abnormal J-point elevation in V_3R and V_4R should be 0.05 mV (0.5 mm), except for males less than 30 years of age, for whom 0.1 mV (1 mm) is more appropriate.
5. For men and women, the threshold value for abnormal J-point elevation in V_5 through V_6 should be 0.05 mV (0.5 mm).
6. For men and women of all ages, the threshold value for abnormal J-point depression should be −0.05 mV (−0.5 mm) in leads V_2 and V_3 and −0.1 mV (−1 mm) in all other leads.

ST SEGMENT ELEVATION:

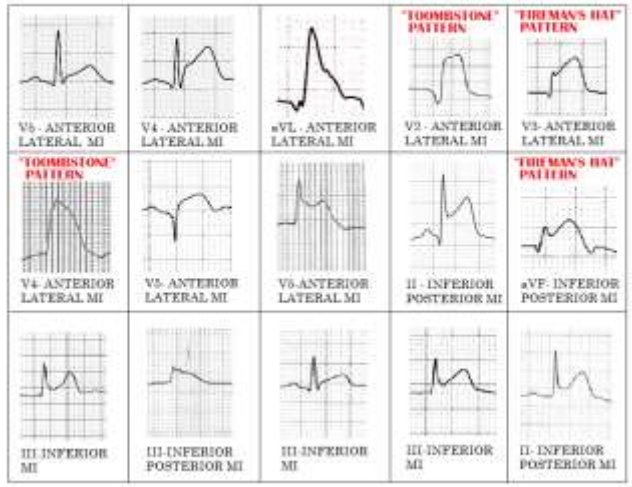
S-T SEGMENTS ELEVATE WITHIN SECONDS OF CORONARY ARTERY OCCLUSION:

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

3 COMMON PATTERNS of ST SEGMENT ELEVATION From ACUTE MI:

ST SEGMENT ELEVATION in ACUTE MI:

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



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

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

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

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

ACUTE MI

COMPLICATIONS TO ANTICIPATE FOR ALL MI PATIENTS :

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

STEMI

- Correlation of ECG Leads with Coronary Arterial Anatomy and the STRUCTURES SERVED by the OCCLUDED ARTERY . . . .

STEMI

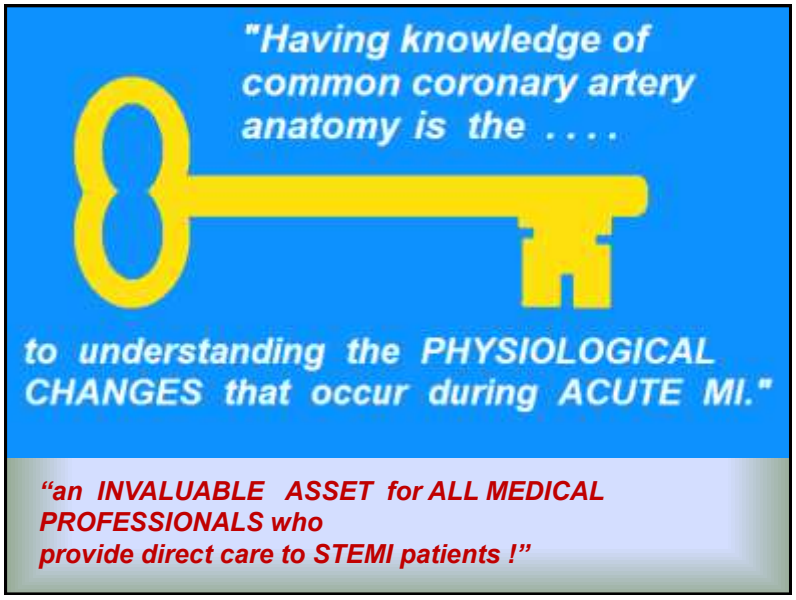
- Correlation of ECG Leads with Coronary Arterial Anatomy and the STRUCTURES SERVED by the OCCLUDED ARTERY . . . .

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

STEMI

- **Correlation of ECG Leads with Coronary Arterial Anatomy and the STRUCTURES SERVED by the OCCLUDED ARTERY . . . .**

. . . . . Will serve as a "crystal ball," allowing you to ANTICIPATE complications of STEMI . . . . . BEFORE they occur !!



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

INTERPRET THE EKG, THEN:

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

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

CASE STUDY 1 - STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

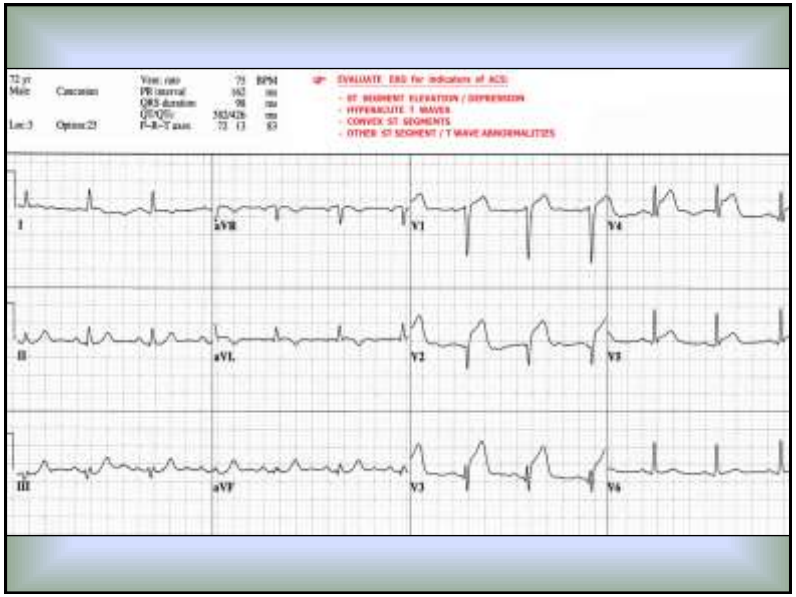
RISK FACTOR PROFILE:

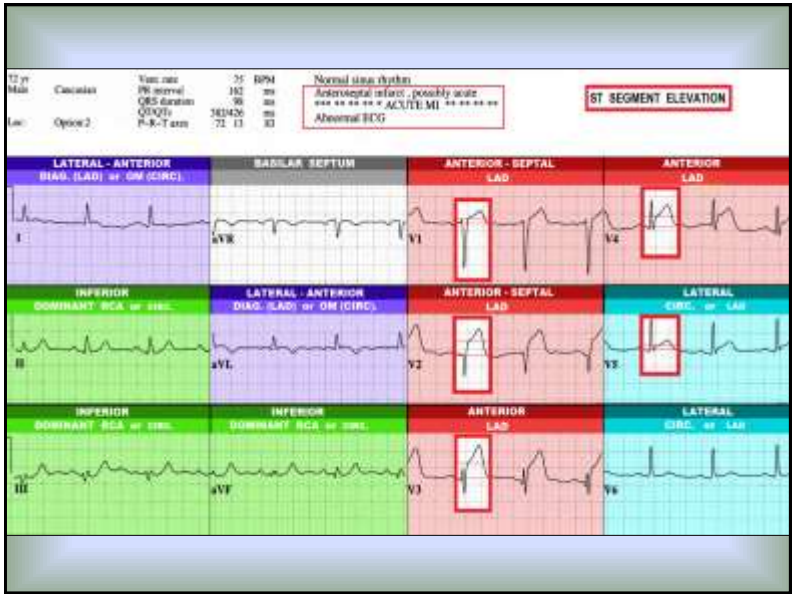
- FAMILY HISTORY - father died of MI at age 77
- FORMER CIGARETTE SMOKER - smoked for 30 year - quit 27 years ago
- DIABETES - oral meds and diet controlled
- HIGH CHOLESTEROL - controlled with STATIN meds
- AGE: OVER 65

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

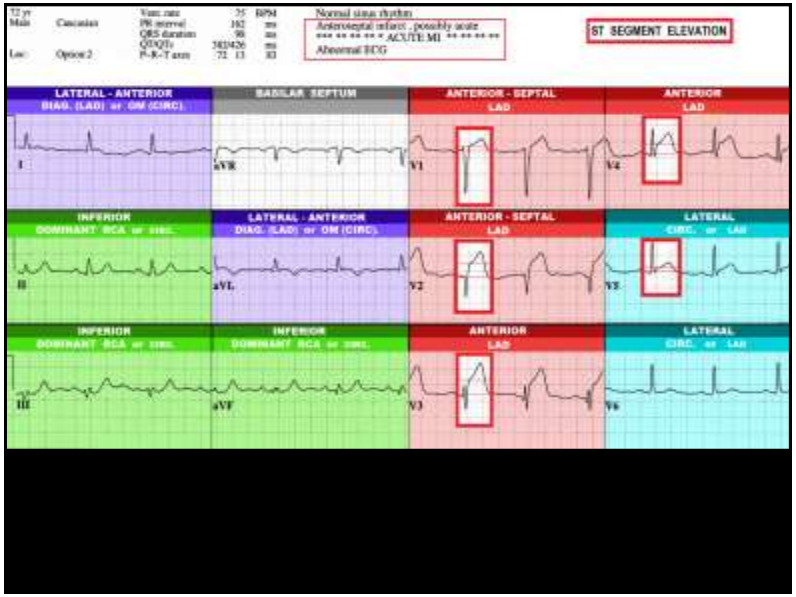
VITAL SIGNS: BP: 100/64, P: 75, R: 20, SAO2: 94%

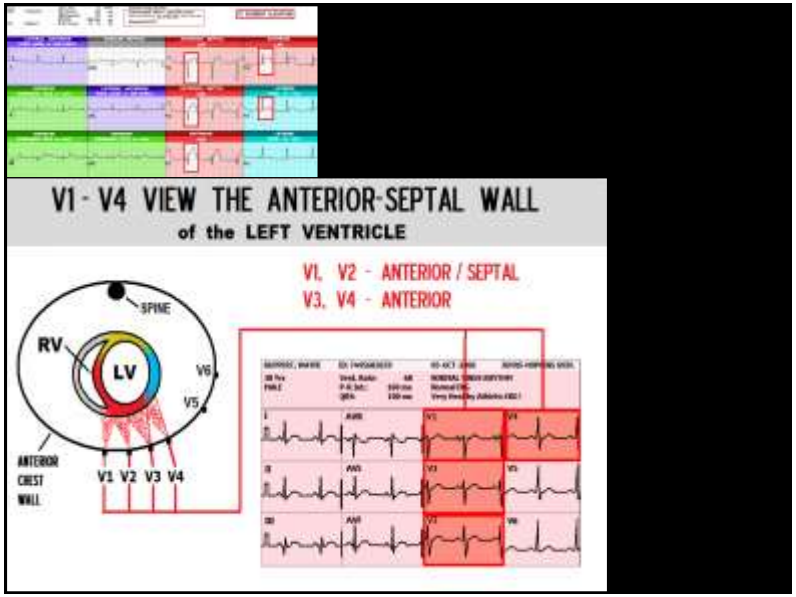
LABS: FIRST TROPONIN: 6.4

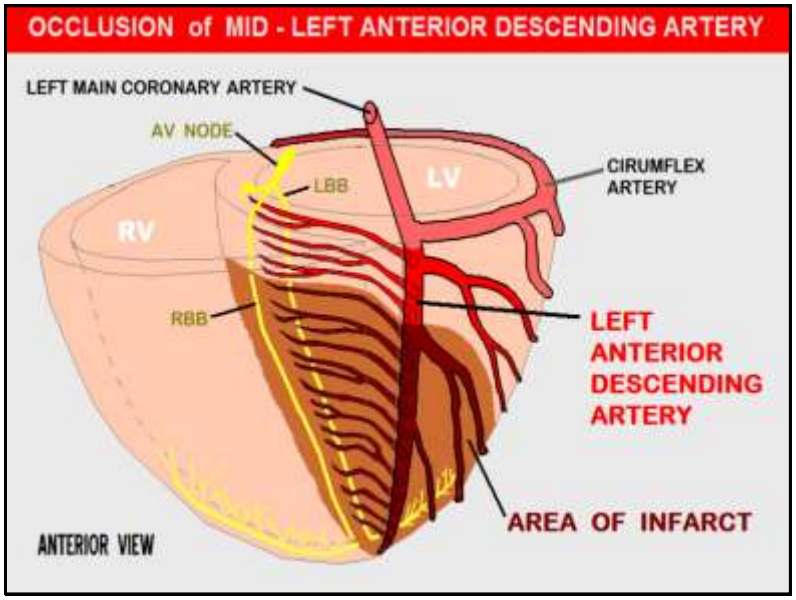


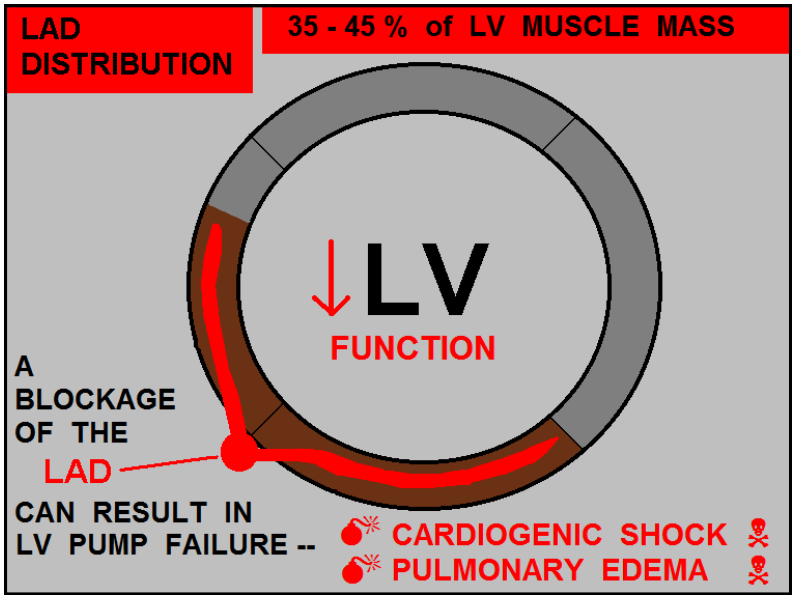


Note: There is NO Reciprocal ST Depression on this STEMI ECG !


ST SEGMENT ELEVATION
LATERAL - ANTERIOR
DIAG. (LAD) or OM (CIRC).
BASILAR SEPTUM
ANTERIOR - SEPTAL
LAD
ANTERIOR
LAD
I
aVR
V1
V4
INFERIOR
DOMINANT RCA or CIRC.
LATERAL - ANTERIOR
DIAG. (LAD) or OM (CIRC).
ANTERIOR - SEPTAL
LAD
LATERAL
CIRC. or LAD
II
aVL
V2
V5
INFERIOR
DOMINANT RCA or CIRC.
INFERIOR
DOMINANT RCA or CIRC.
ANTERIOR
LAD
LATERAL
CIRC. or LAD
III
aVF
V3
V6


V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL
of the LEFT VENTRICLE
V1, V2 - ANTERIOR / SEPTAL
V3, V4 - ANTERIOR
SPINE
RV
LV
V6
V5
ANTERIOR CHEST WALL
V1 V2 V3 V4

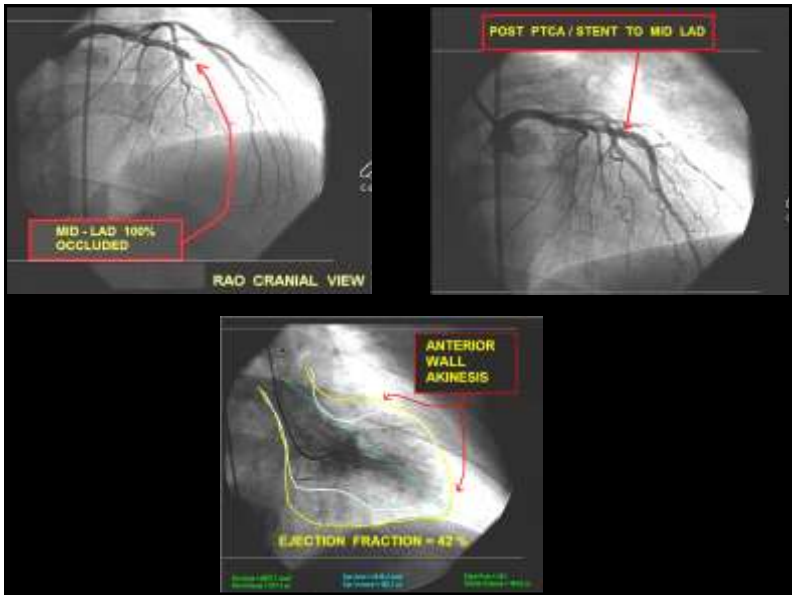

OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY
LEFT MAIN CORONARY ARTERY
AV NODE
LBB
LV
CIRUMFLEX ARTERY
RV
RBB
LEFT ANTERIOR DESCENDING ARTERY
AREA OF INFARCT
ANTERIOR VIEW


LAD DISTRIBUTION
35 - 45 % of LV MUSCLE MASS
↓LV
FUNCTION
A BLOCKAGE OF THE
LAD
CAN RESULT IN
LV PUMP FAILURE --
CARDIOGENIC SHOCK
PULMONARY EDEMA

LEFT ANTERIOR DESCENDING ARTERY (LAD)

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

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



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.

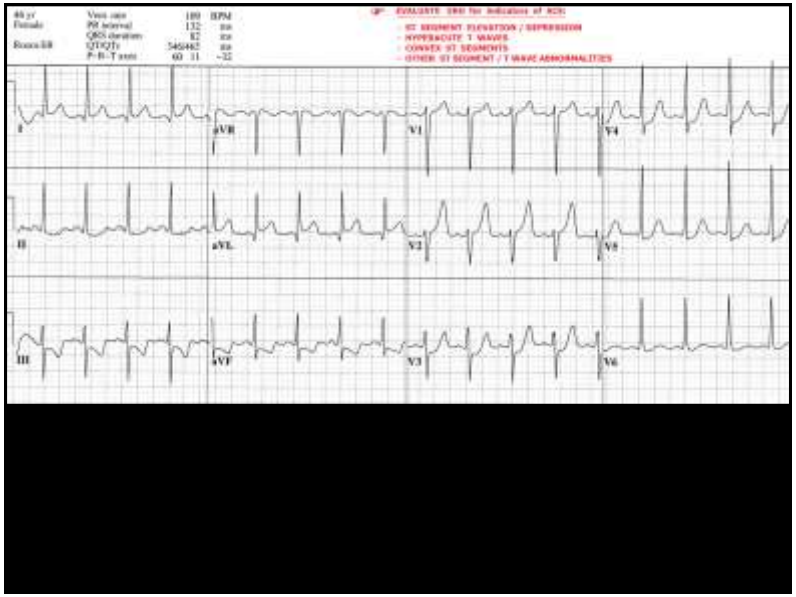
RISK FACTOR PROFILE:

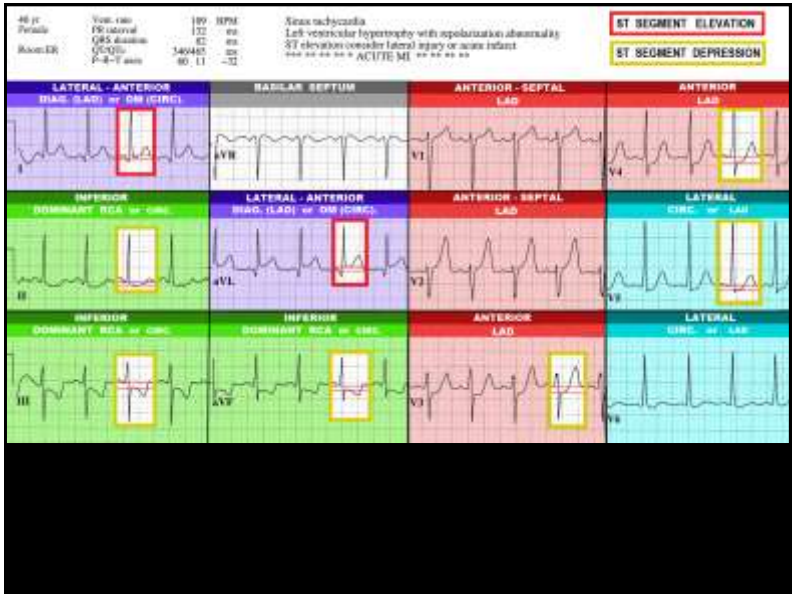
- FAMILY HISTORY - father died of CAD, older brother had CABG, age 39
- DIABETES - diet controlled
- HYPERTENSION

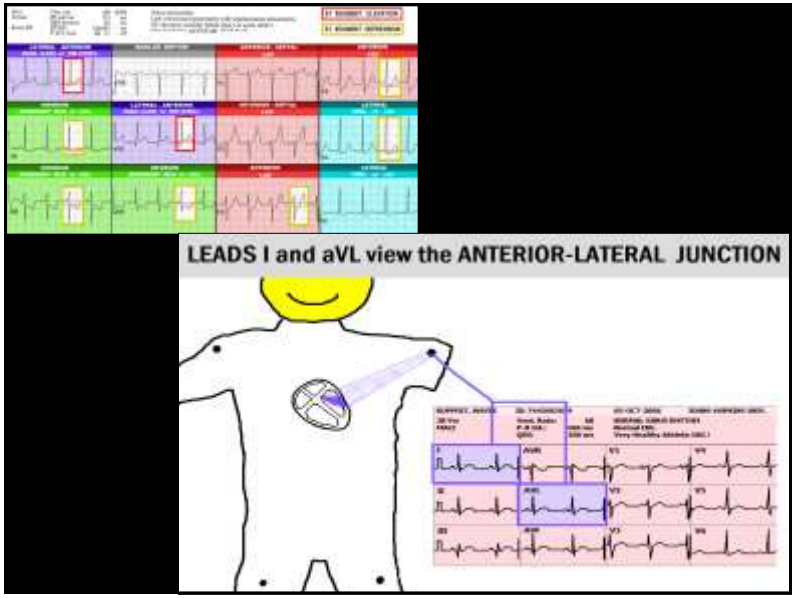
PHYSICAL EXAM: Pt. CAOx4, anxious, SKIN cold, clammy, diaphoretic. No JVD. Lungs: clear, bilaterally. Heart Sounds: Normal S1, S2.

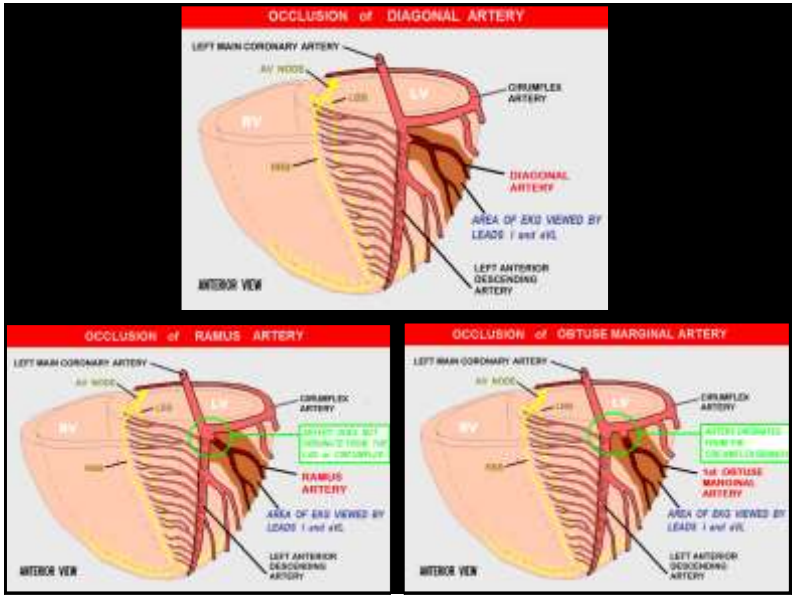
VITAL SIGNS: BP: 168/98, P: 110, R: 24, SAO2: 97% on O2 4 LPM via nasal canula

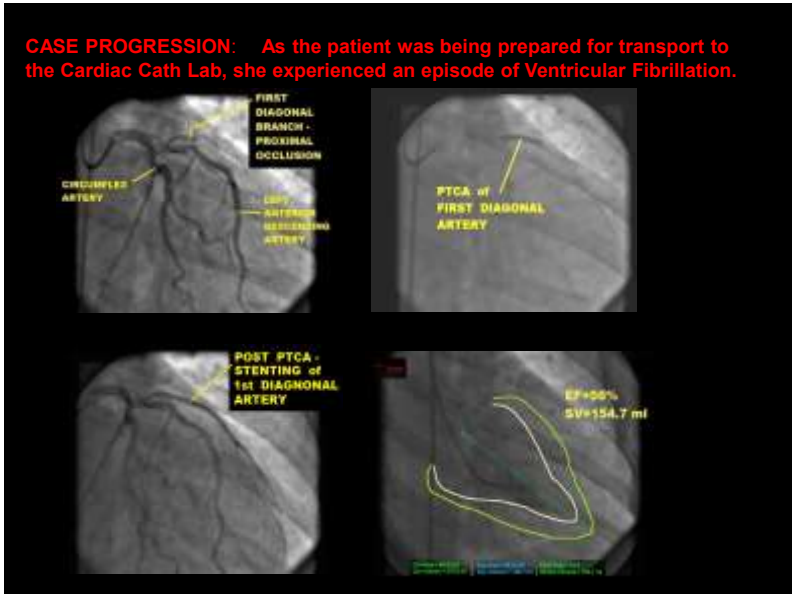
LABS: TROPONIN ultra = 2.8




ST SEGMENT ELEVATION
ST SEGMENT DEPRESSION
LATERAL - ANTERIOR
BASILAR SEPTUM
ANTERIOR - SEPTAL
LAD
ANTERIOR
LAD
INFERIOR
LATERAL - ANTERIOR
ANTERIOR - SEPTAL
LAD
LATERAL
INFERIOR
INFERIOR
ANTERIOR
LAD
LATERAL


LEADS I and aVL view the ANTERIOR-LATERAL JUNCTION


OCCLUSION of DIAGONAL ARTERY
LEFT MAIN CORONARY ARTERY
AV NODE
CIRCUMFLEX ARTERY
RV
LV
DIAGONAL ARTERY
AREA OF EKG VIEWED BY LEADS I and aVL
LEFT ANTERIOR DESCENDING ARTERY
ANTERIOR VIEW
OCCLUSION of RAMUS ARTERY
RAMUS ARTERY
OCCLUSION of OBTUSE MARGINAL ARTERY
1st OBTUSE MARGINAL ARTERY


CASE PROGRESSION: As the patient was being prepared for transport to the Cardiac Cath Lab, she experienced an episode of Ventricular Fibrillation.
FIRST DIAGONAL BRANCH - PROXIMAL OCCLUSION
CIRCUMFLEX ARTERY
PTCA of FIRST DIAGONAL ARTERY
POST PTCA - STENTING of 1st DIAGNONAL ARTERY
EF=58%
SV=154.7 ml

CASE STUDY 3: STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

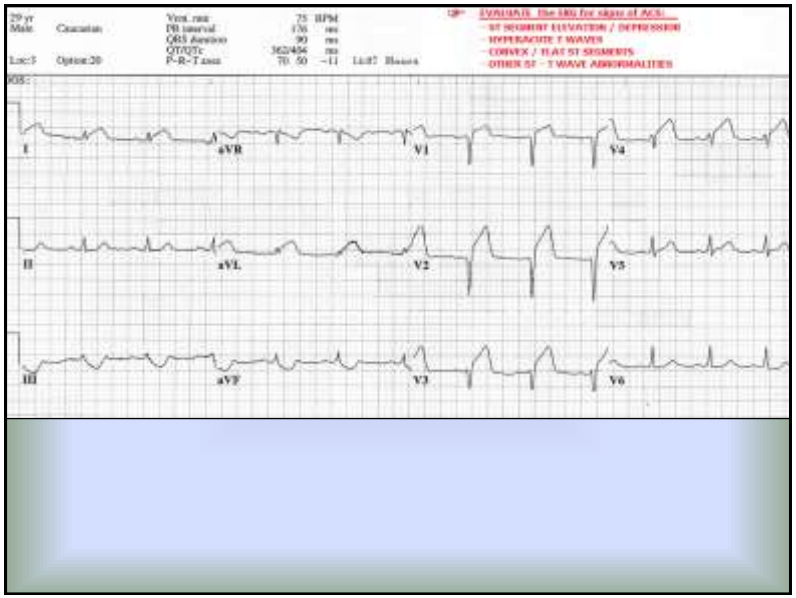
RISK FACTOR PROFILE:

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

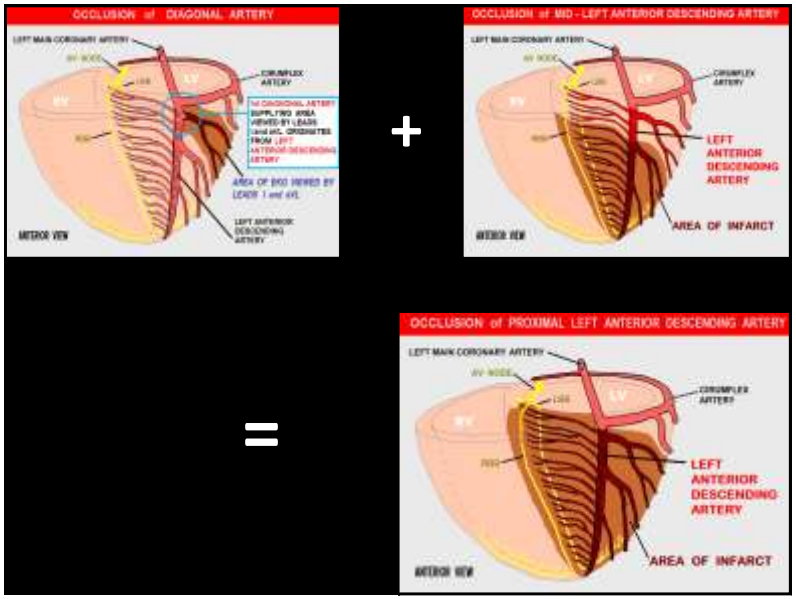
PHYSICAL EXAM: Patient alert, oriented X 4, skin cool, dry, pale. Patient restless. No JVD, Lungs clear bilaterally. Heart sounds normal S1, S2. No peripheral edema.

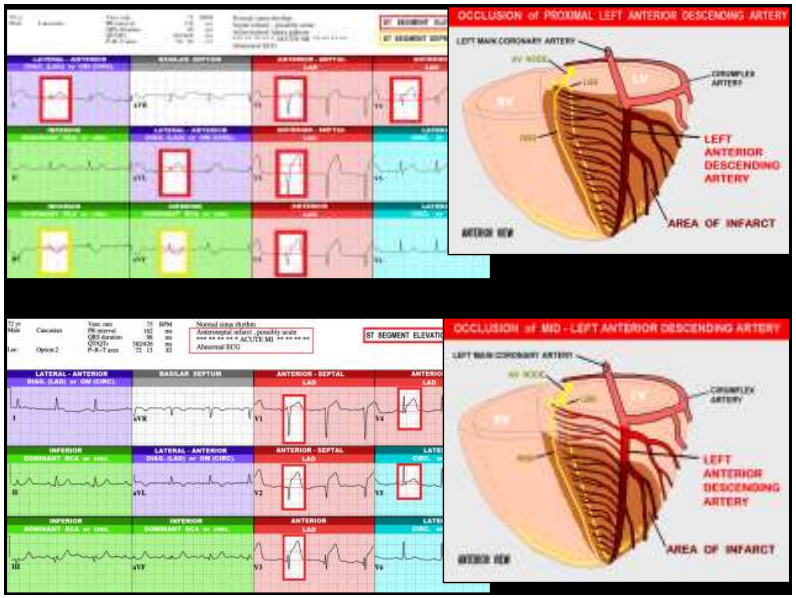
VITAL SIGNS: BP: 104/78, P: 76, R: 20, SAO2: 96%

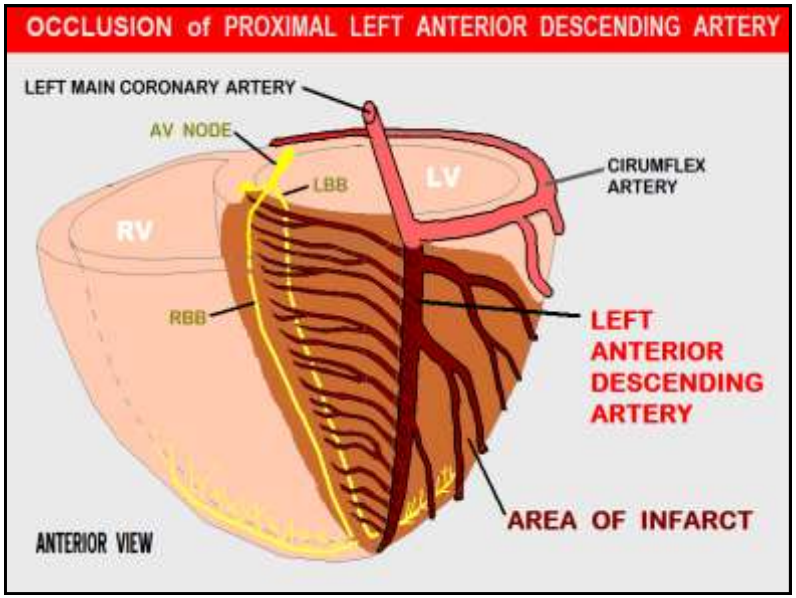
LABS: INITIAL CARDIAC MARKERS - NEGATIVE



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


OCCLUSION of DIAGONAL ARTERY
LEFT MAIN CORONARY ARTERY
AV NODE
LV
RV
CIRCUMFLEX ARTERY
AREA OF ECG VIEWED BY LEADS I and aVL
LEFT ANTERIOR DESCENDING ARTERY
ANTERIOR VIEW
+
OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY
LEFT MAIN CORONARY ARTERY
AV NODE
LV
RV
CIRCUMFLEX ARTERY
LEFT ANTERIOR DESCENDING ARTERY
AREA OF INFARCT
ANTERIOR VIEW
=
OCCLUSION of PROXIMAL LEFT ANTERIOR DESCENDING ARTERY
LEFT MAIN CORONARY ARTERY
AV NODE
LV
RV
CIRCUMFLEX ARTERY
LEFT ANTERIOR DESCENDING ARTERY
AREA OF INFARCT
ANTERIOR VIEW

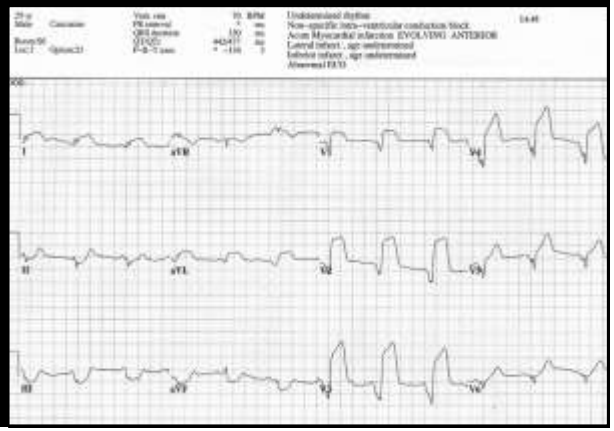

OCCLUSION of PROXIMAL LEFT ANTERIOR DESCENDING ARTERY
LEFT MAIN CORONARY ARTERY
AV NODE
LV
RV
CIRCUMFLEX ARTERY
LEFT ANTERIOR DESCENDING ARTERY
AREA OF INFARCT
ANTERIOR VIEW
ST SEGMENT ELEVATION
OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY
LEFT MAIN CORONARY ARTERY
AV NODE
LV
RV
CIRCUMFLEX ARTERY
LEFT ANTERIOR DESCENDING ARTERY
AREA OF INFARCT
ANTERIOR VIEW



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

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

-WHAT THERAPEUTIC INTERVENTIONS SHOULD BE IMPLMENTED AT THIS POINT ?



CASE STUDY 4: CRITICAL DECISIONS SCENARIO

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

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

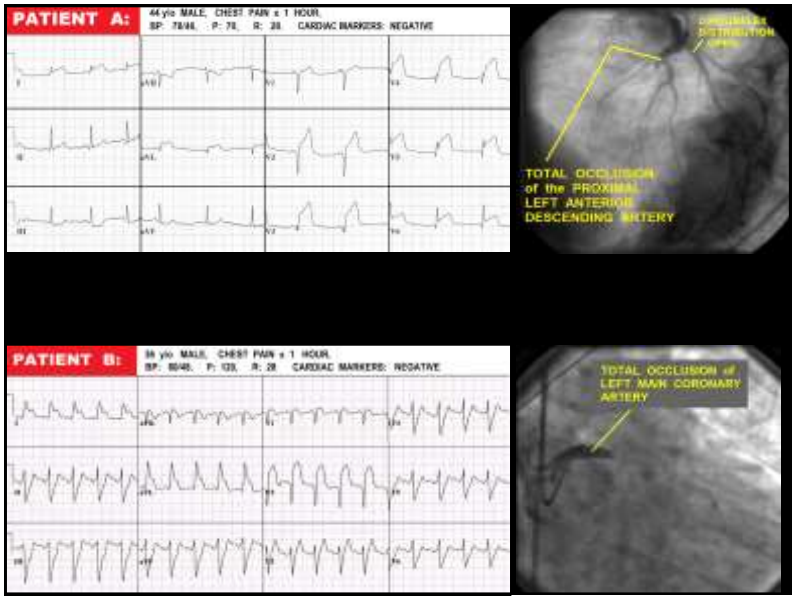
PATIENT A: 44 y/o MALE, CHEST PAIN x 1 HOUR, BP: 78/44, P: 78, R: 28 CARDIAC MARKERS: NEGATIVE

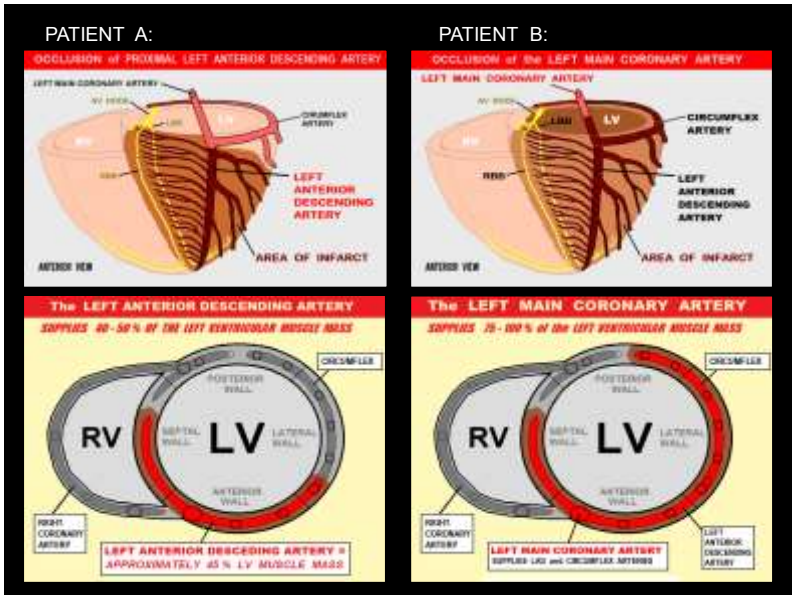
PATIENT B: 36 y/o MALE, CHEST PAIN x 1 HOUR, BP: 90/60, P: 120, R: 28 CARDIAC MARKERS: NEGATIVE

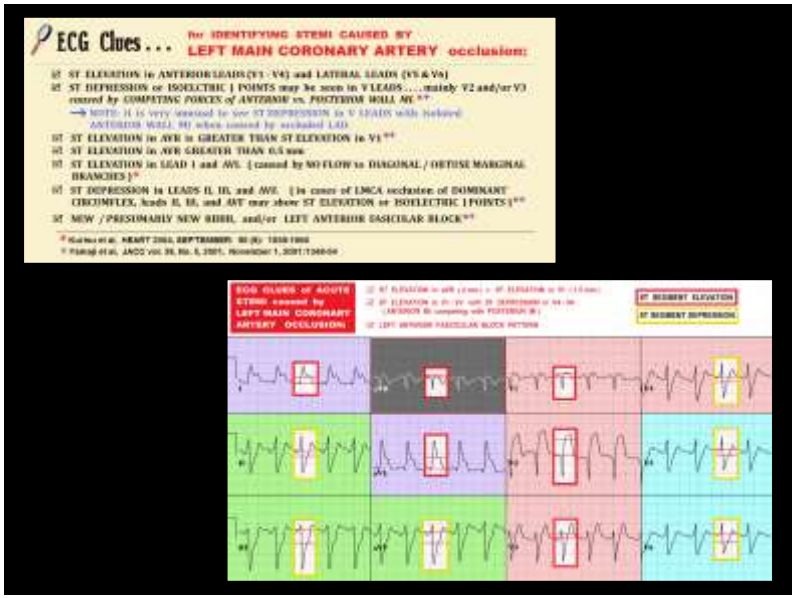
WHO SHOULD GO TO THE CATH LAB FIRST ?

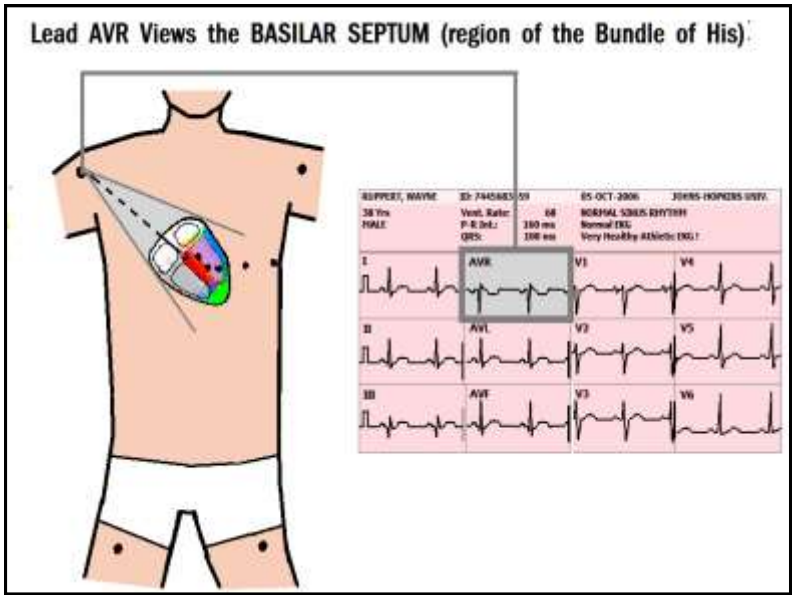
And . . . .

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

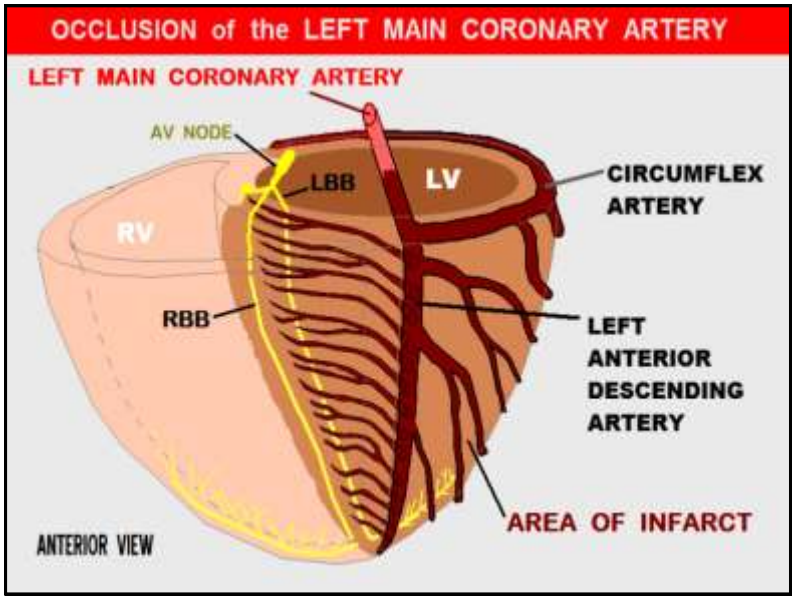

PATIENT A:
44 y/o MALE, CHEST PAIN x 1 HOUR.
BP: 78/46, P: 70, R: 20 CARDIAC MARKERS: NEGATIVE
TOTAL OCCLUSION of the PROXIMAL LEFT ANTERIOR DESCENDING ARTERY
PATIENT B:
TOTAL OCCLUSION of LEFT MAIN CORONARY ARTERY

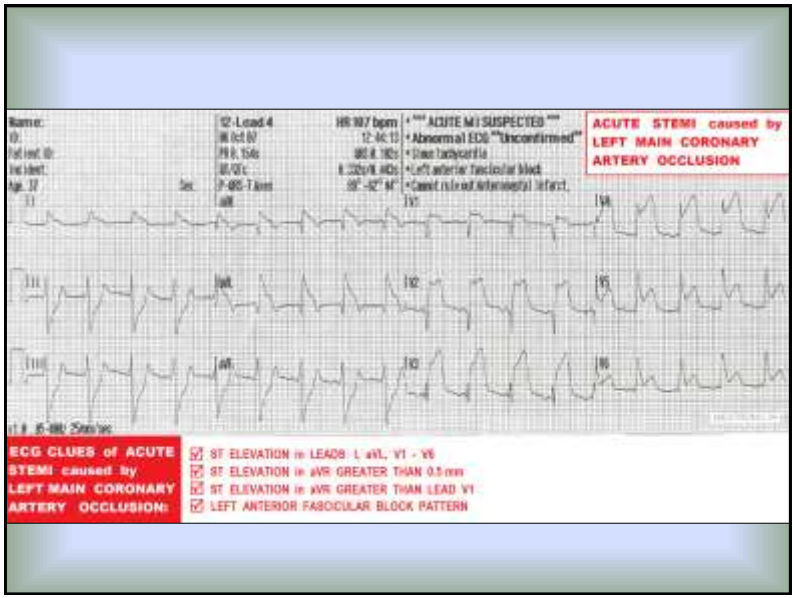

PATIENT A:
PATIENT B:
OCCLUSION of PROXIMAL LEFT ANTERIOR DESCENDING ARTERY
OCCLUSION of the LEFT MAIN CORONARY ARTERY
LEFT MAIN CORONARY ARTERY
CIRCUMFLEX ARTERY
LEFT ANTERIOR DESCENDING ARTERY
AREA OF INFARCT
The LEFT ANTERIOR DESCENDING ARTERY
The LEFT MAIN CORONARY ARTERY
RV
LV
LEFT ANTERIOR DESCENDING ARTERY = APPROXIMATELY 45 % LV MUSCLE MASS
LEFT MAIN CORONARY ARTERY

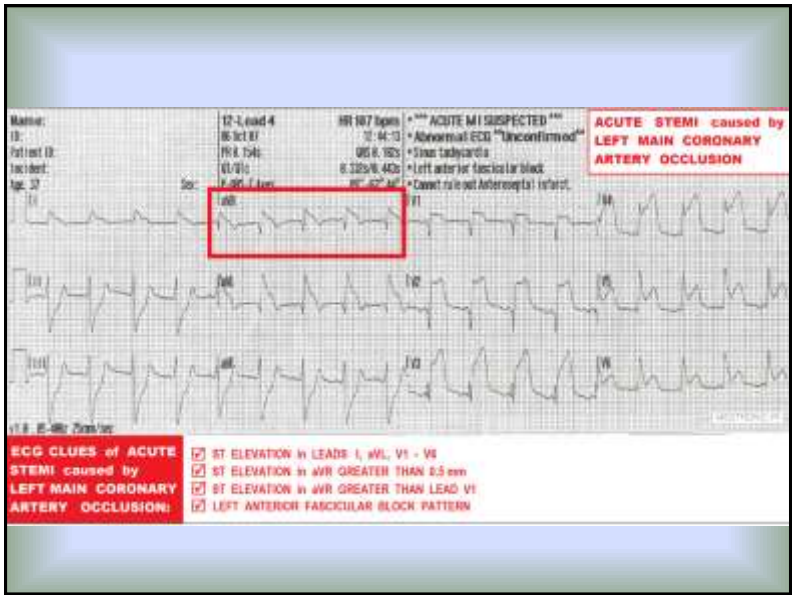

ECG Clues . . .
for IDENTIFYING STEMI CAUSED BY
LEFT MAIN CORONARY ARTERY occlusion:
ST ELEVATION in ANTERIOR LEADS (V1 - V4) and LATERAL LEADS (V5 & V6)
ST DEPRESSION or ISOELECTRIC (POINTS may be seen in V LEADS . . . . mainly V2 and/or V3
caused by COMPETING FORCES of ANTERIOR vs. POSTERIOR WALL MI.**
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)**
ST NEW / PRESUMABLY NEW RBBB, and/or LEFT ANTERIOR FASCICULAR BLOCK**
ECG CLUES OF ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION
ST SEGMENT ELEVATION
ST SEGMENT DEPRESSION

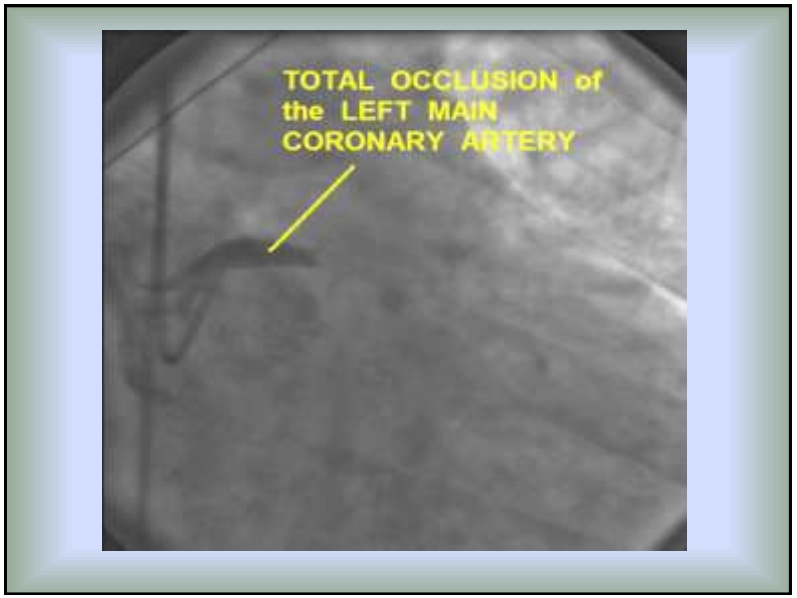

Lead AVR Views the BASILAR SEPTUM (region of the Bundle of His)
AVR
V1
V4
AVL
V2
V5
AVF
V3
V6

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




ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION
ECG CLUES of ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION:
ST ELEVATION in LEADS I, aVL, V1 - V6
ST ELEVATION in aVR GREATER THAN 0.5 mm
ST ELEVATION in aVR GREATER THAN LEAD V1
LEFT ANTERIOR FASCICULAR BLOCK PATTERN


ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION
ECG CLUES of ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION:
ST ELEVATION in LEADS I, aVL, V1 - V6
ST ELEVATION in aVR GREATER THAN 0.5 mm
ST ELEVATION in aVR GREATER THAN LEAD V1
LEFT ANTERIOR FASCICULAR BLOCK PATTERN



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.*

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

- ☑ ST ELEVATION in ANTERIOR LEADS (V1 - V4) and LATERAL LEADS (V5 & V6)
- ☑ ST DEPRESSION or ISOELCTRIC] 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

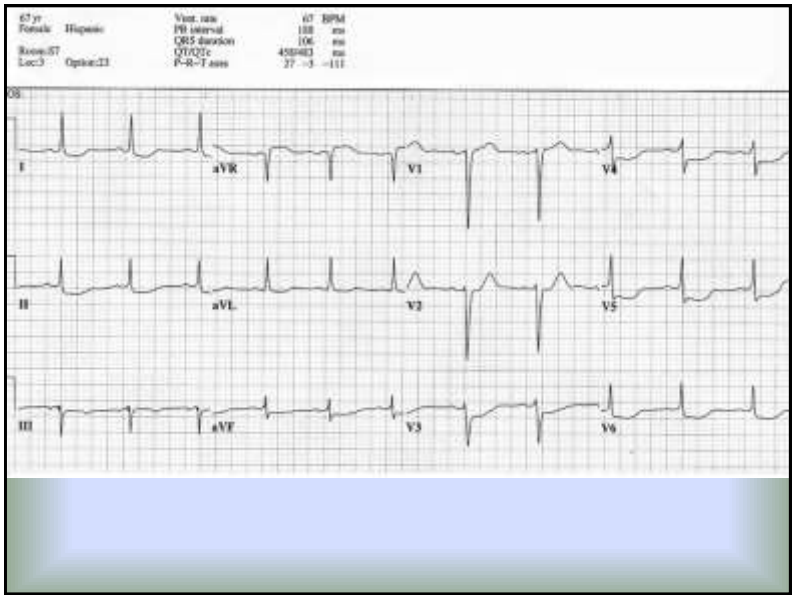
Yamaji et al, JACC vol 38, No 5, 2001: 1348-54

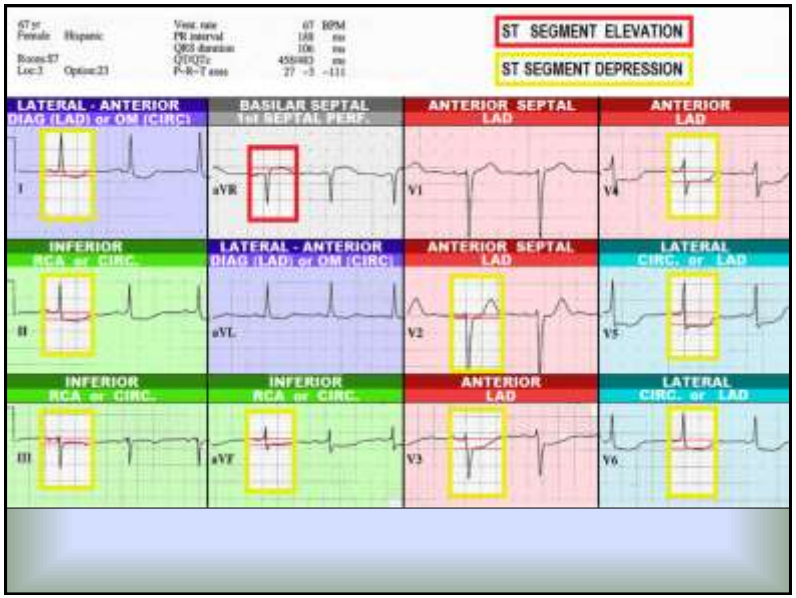
Electrocardiogram patterns in acute left main occlusion: J Electrocardiol. 2008 Nov-Dec;41(6):626-9.

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

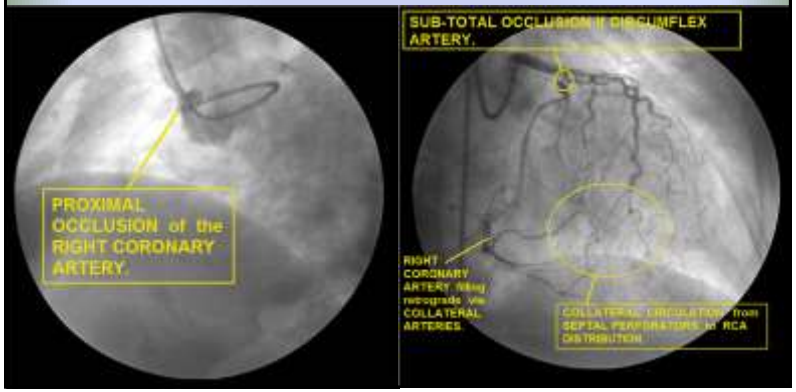
"In patients with:
- **Angina at rest**
- **ST Elevation in AVR and ST Depression in 8 or more ECG leads (global ischemia), it is reported with a *75% predictive accuracy* of 3-vessel or left main coronary artery stenosis" . . .**

- Wagner et al, **2009 ACC/AHA Standardization and Interpretation of the ECG, Part VI, ACS**.


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


ST SEGMENT ELEVATION
ST SEGMENT DEPRESSION
LATERAL - ANTERIOR
DIAG (LAD) or OM (CIRC)
BASILAR SEPTAL
1st SEPTAL PERF.
ANTERIOR SEPTAL
LAD
ANTERIOR
LAD
INFERIOR
RCA or CIRC.
LATERAL - ANTERIOR
DIAG (LAD) or OM (CIRC)
ANTERIOR SEPTAL
LAD
LATERAL
CIRC. or LAD
INFERIOR
RCA or CIRC.
INFERIOR
RCA or CIRC.
ANTERIOR
LAD
LATERAL
CIRC. or LAD

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



ANTICIPATED COMPLICATIONS of GLOBAL ISCHEMIA with POSSIBLE NSTEMI -- INTERVENTIONS to be CONSIDERED:	
Patients with CHEST PAIN at REST and this ECG presentation have a 75% incidence of severe LMCA STENOSIS and/or TRIPLE - VESSEL DISEASE -- in such cases Coronary Artery Bypass Surgery (CABG) is frequently indicated.	PREHOSPITAL: if patient has no hospital preference consider transport to Chest Pain Center WITH Open Heart Surgery capabilities IF nearby. HOSPITAL: consider use of SHORT-ACTING intravenous GP IIb/IIIa receptor agonists
- ACTIVE CHEST PAIN	ACUTE CHEST PAIN PROTOCOL
- ISCHEMIA - CONSIDER DYSRHYTHMIAS	ACLS PROTOCOL
- INCREASED PROBABILITY of IMMINENT MYOCARDIAL INFARCTION	1. AGGRESSIVE SERIAL TROPONIN and SERIAL ECG PROTOCOLS (2014 AHA /ACC / NSTE-ACS Guidelines) 2. Positive TROPONIN: consider STAT / early Cardiac Catheterization

Excerpt from ***STEMI Assistant***

CASE STUDY 7 - STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

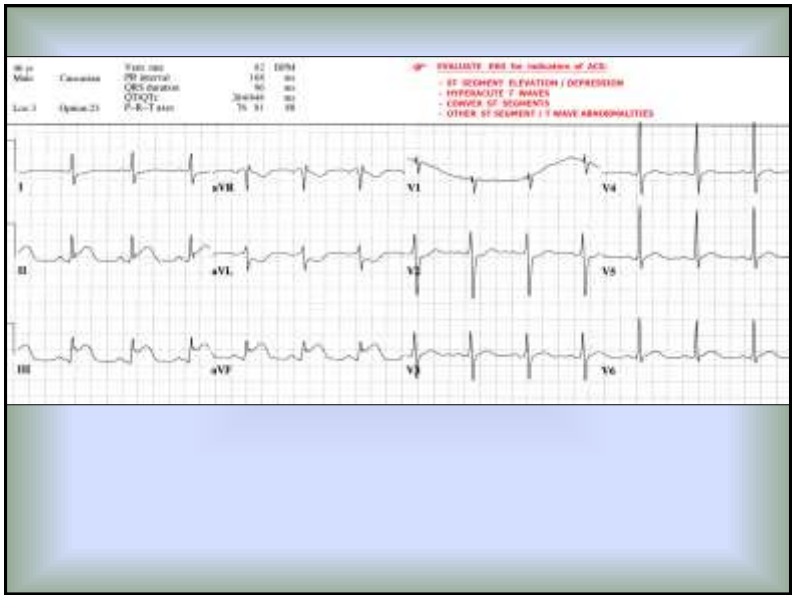
RISK FACTOR PROFILE:

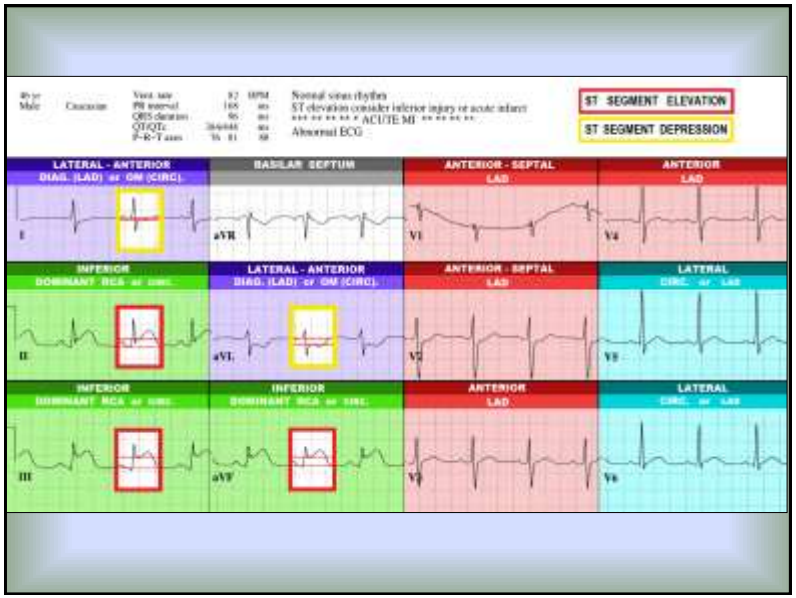
- CURRENT CIGARTTE SMOKER x 18 YEARS
- HYPERTENSION
- HIGH LDL CHOLESTEROL

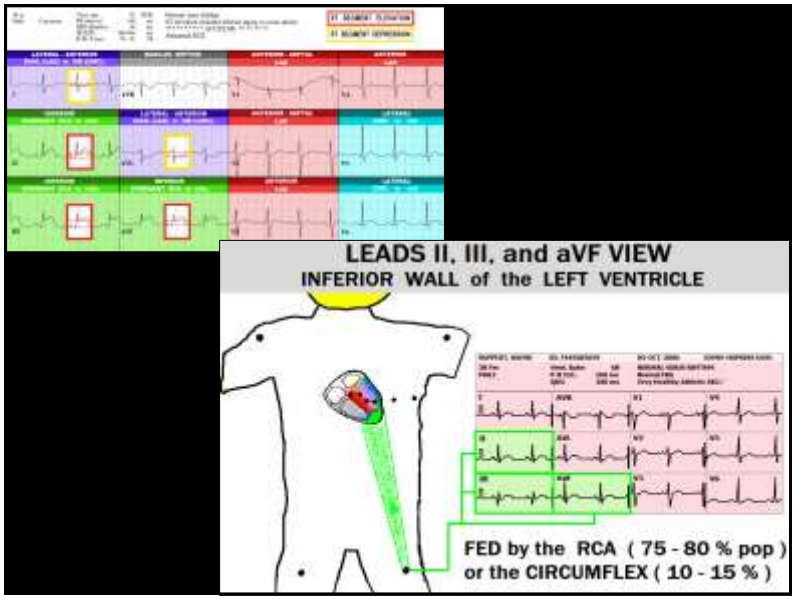
PHYSICAL EXAM: Patient is alert & oriented x 4, skin warm, dry, color normal. Non-anxious Lungs clear, normal S1, S2. No JVD, No ankle edema.

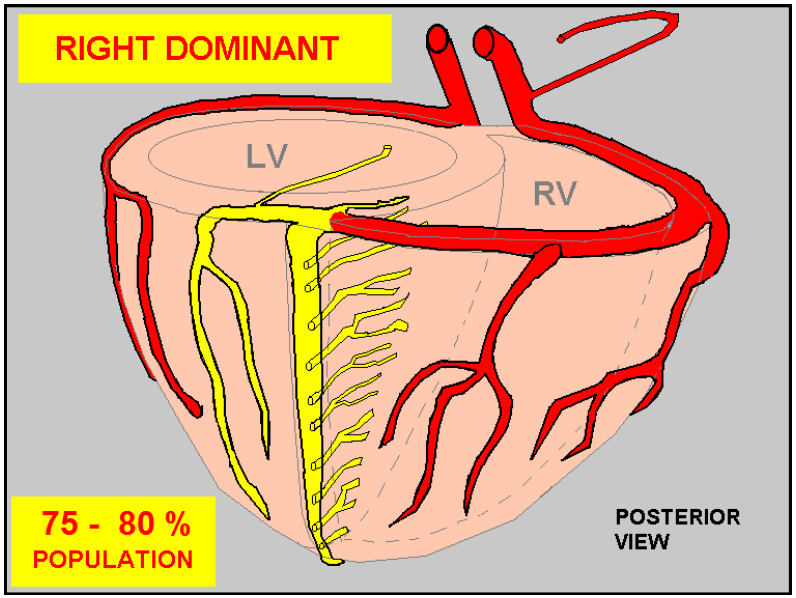
VITAL SIGNS: BP: 136/88 P: 88 R: 20 SAO2: 100% on 4 LPM O2

LABS: TROPONIN: < .04




ST SEGMENT ELEVATION
ST SEGMENT DEPRESSION
LATERAL - ANTERIOR
DIAG. (LAD) or OM (CIRC).
BASILAR SEPTUM
ANTERIOR - SEPTAL
LAD
ANTERIOR
LAD
I
aVR
V1
V4
INFERIOR
DOMINANT RCA or CIRC.
LATERAL - ANTERIOR
DIAG. (LAD) or OM (CIRC).
ANTERIOR - SEPTAL
LAD
LATERAL
CIRC. or LAD
II
aVL
V2
V5
INFERIOR
DOMINANT RCA or CIRC.
INFERIOR
DOMINANT RCA or CIRC.
ANTERIOR
LAD
LATERAL
CIRC. or LAD
III
aVF
V3
V6


LEADS II, III, and aVF VIEW
INFERIOR WALL of the LEFT VENTRICLE
FED by the RCA (75 - 80 % pop)
or the CIRCUMFLEX (10 - 15 %)

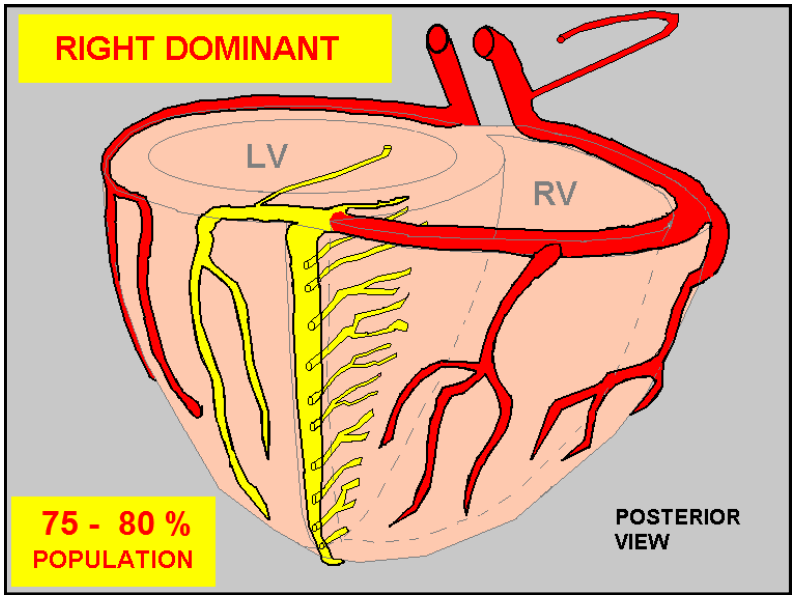


HELPFUL HINT . . . MEMORIZE THIS !

RIGHT CORONARY ARTERY (RCA)

RIGHT DOMINANT SYSTEMS

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



A standard

12 LEAD EKG

Does NOT show the

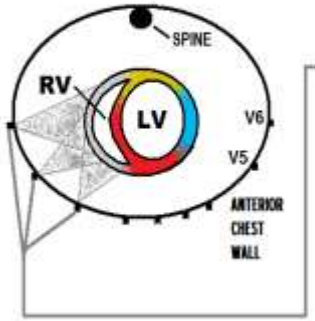
RIGHT VENTRICLE

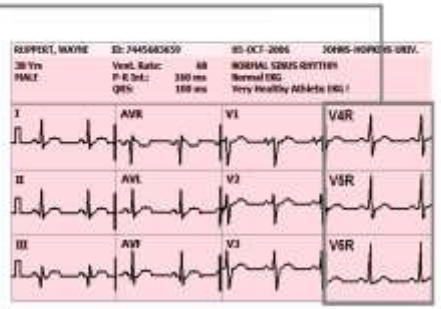
To see the
RIGHT VENTRICLE . . .

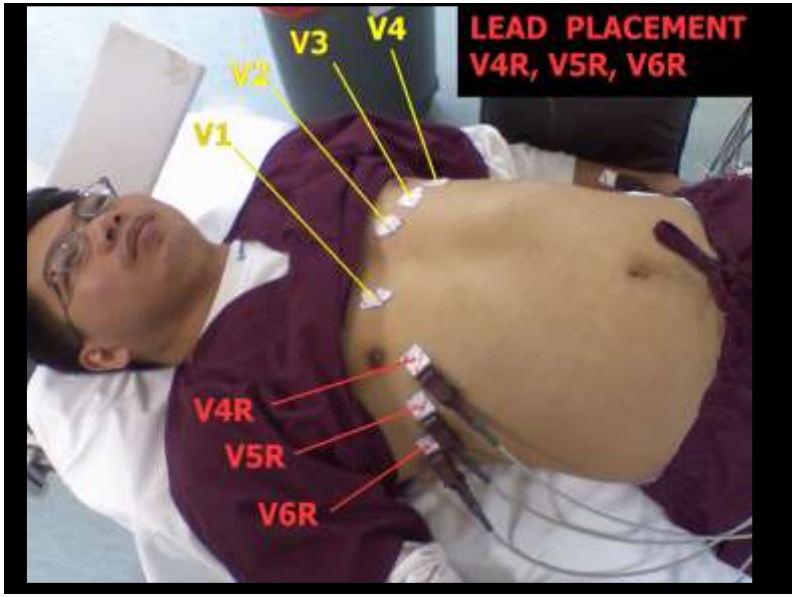
. . . such as in cases of
INFERIOR WALL M.I.

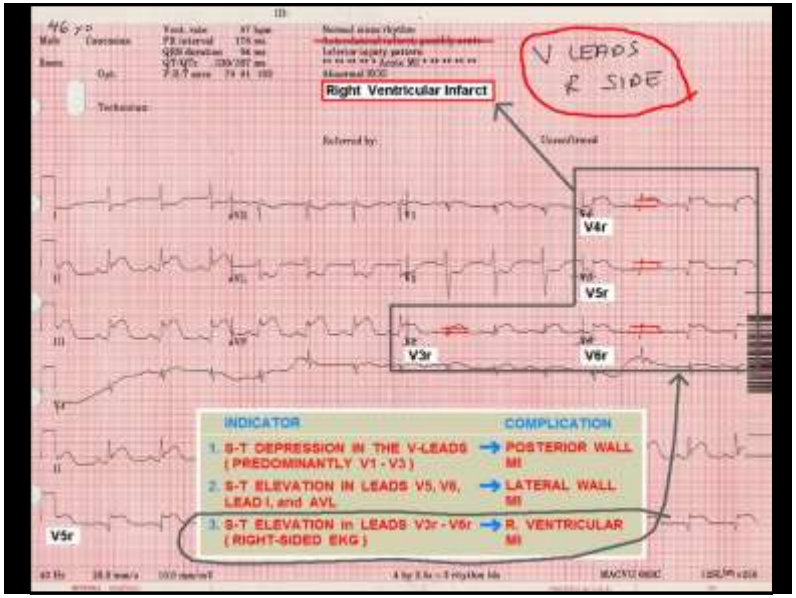
☞ You must do a
RIGHT - SIDED EKG !!

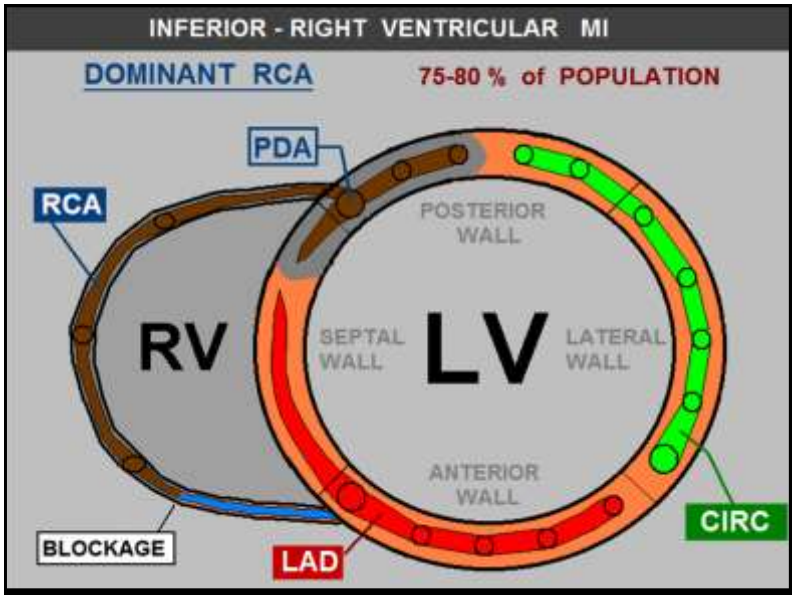
V4R - V6R VIEW THE RIGHT VENTRICLE





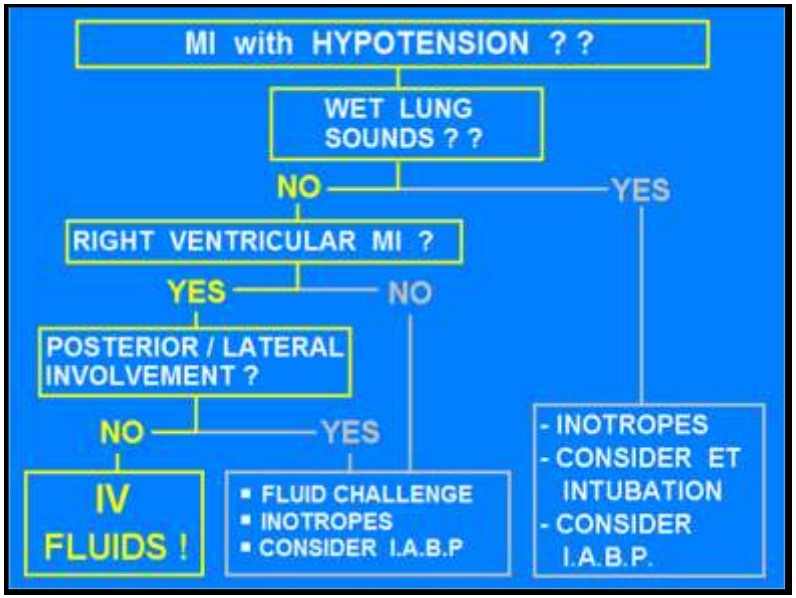

LEAD PLACEMENT
V4R, V5R, V6R
V1
V2
V3
V4
V4R
V5R
V6R


Right Ventricular Infarct
V LEADS
R SIDE
V4r
V5r
V3r
V6r
INDICATOR
COMPLICATION
1. S-T DEPRESSION IN THE V-LEADS (PREDOMINANTLY V1 - V3) → POSTERIOR WALL MI
2. S-T ELEVATION IN LEADS V5, V6, LEAD I, and AVL → LATERAL WALL MI
3. S-T ELEVATION in LEADS V3r - V6r → R. VENTRICULAR MI
(RIGHT-SIDED EKG)



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

If this patient becomes HYPOTENSIVE . . . . .



IN ***EVERY*** CASE of

INFERIOR WALL STEMI

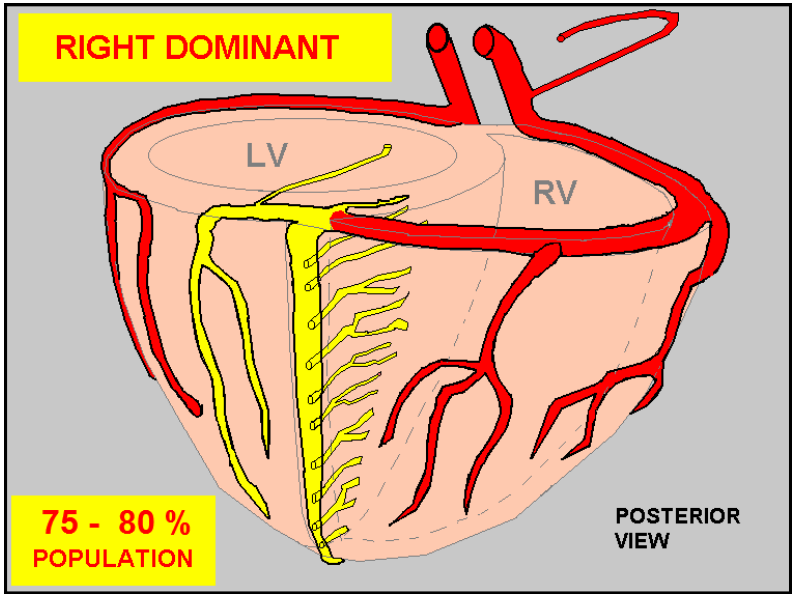
You must first *RULE OUT*
RIGHT VENTRICULAR MI
BEFORE giving any:

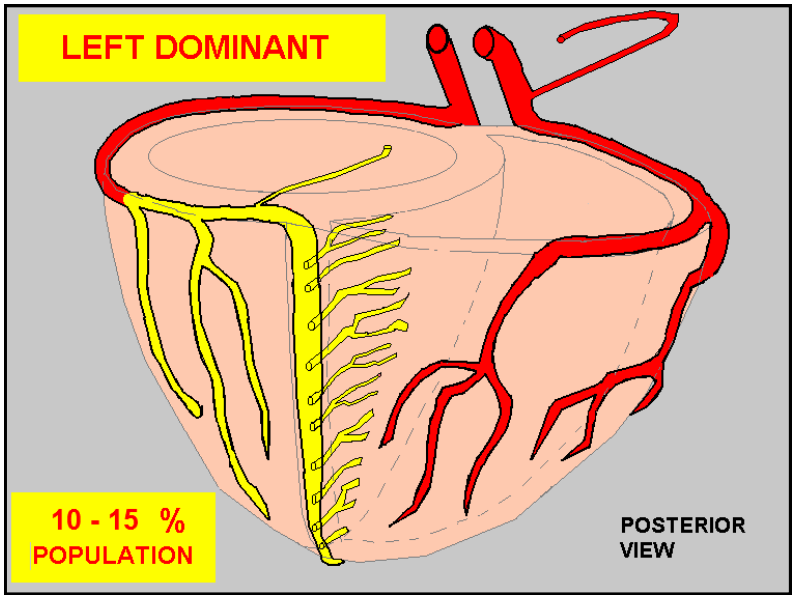
- NITROGLYCERIN
- Diuretics

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

They precipitate SEVERE HYPOTENSION

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





CASE STUDY 9 - **STEMI**

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

RISK FACTOR PROFILE:

- CIGARETTE SMOKER
- HYPERTENSION
- HIGH LDL CHOLESTEROL

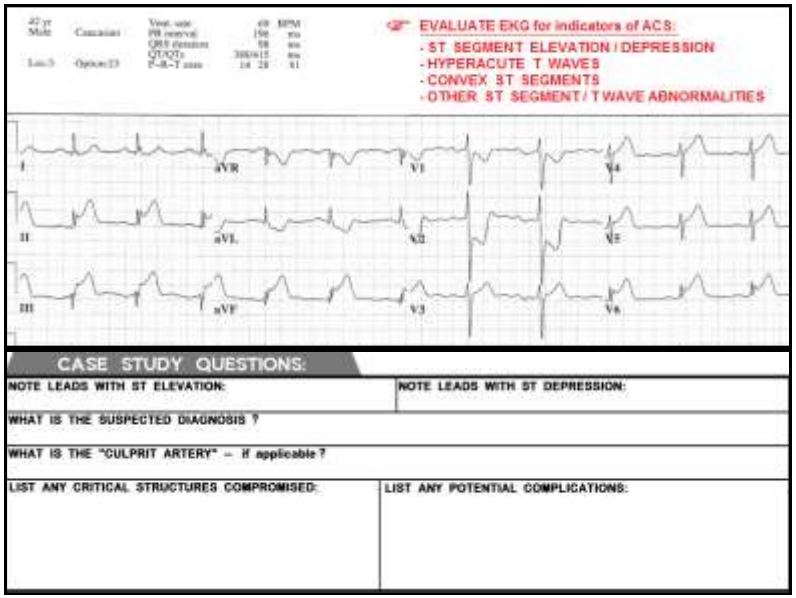
PHYSICAL EXAM: Patient is alert & oriented x 4, ANXIOUS, with COOL, PALE, DIAPHORETIC SKIN. C/O NAUSEA, and is VOMITING. LUNG SOUNDS: COARSE CRACKLES, BASES, bilaterally

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

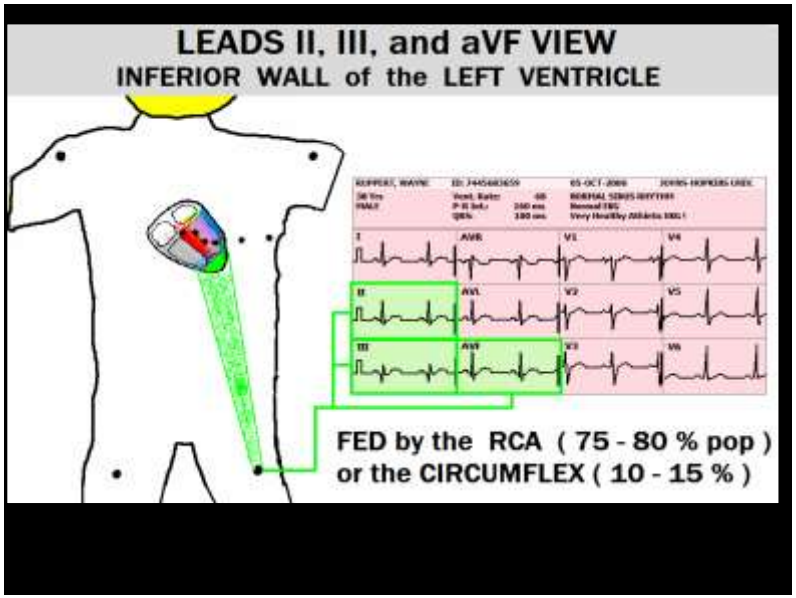
LABS: TROPONIN: < .04

SHOCK ASSESSMENT

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




ST SEGMENT ELEVATION
ST SEGMENT DEPRESSION
LATERAL - ANTERIOR
DIAG (LAD) or OM (CIRC)
BASILAR SEPTAL
tht SEPTAL PERF.
ANTERIOR SEPTAL
LAD
ANTERIOR
LAD
INFERIOR
RCA or CIRC.
LATERAL - ANTERIOR
DIAG (LAD) or OM (CIRC)
ANTERIOR SEPTAL
LAD
LATERAL
CIRC. or LAD
INFERIOR
RCA or CIRC.
INFERIOR
RCA or CIRC.
ANTERIOR
LAD
LATERAL
CIRC. or LAD


LEADS II, III, and aVF VIEW
INFERIOR WALL of the LEFT VENTRICLE
FED by the RCA (75 - 80 % pop)
or the CIRCUMFLEX (10 - 15 %)


ST SEGMENT ELEVATION
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LATERAL - ANTERIOR
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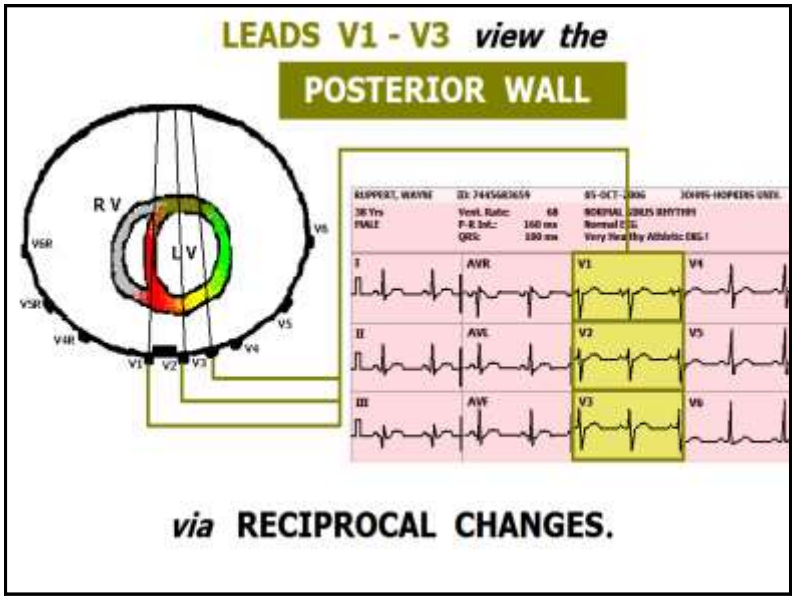
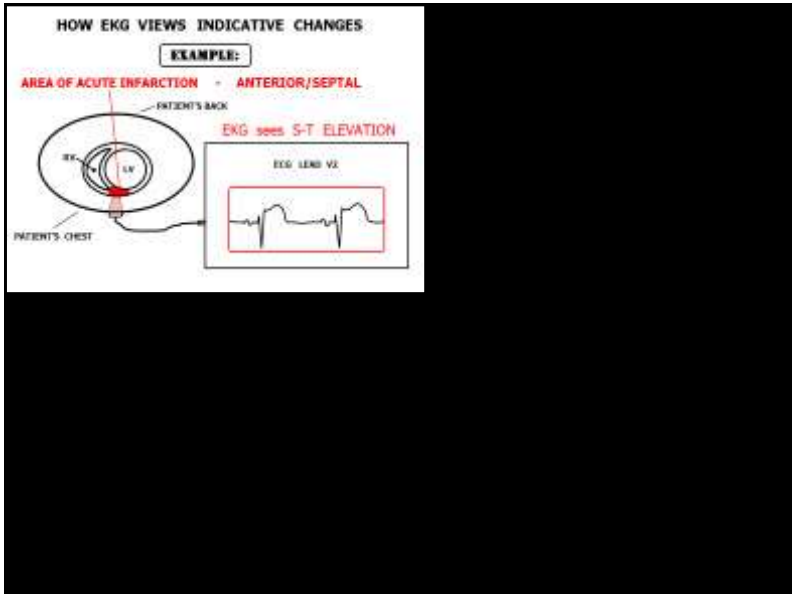
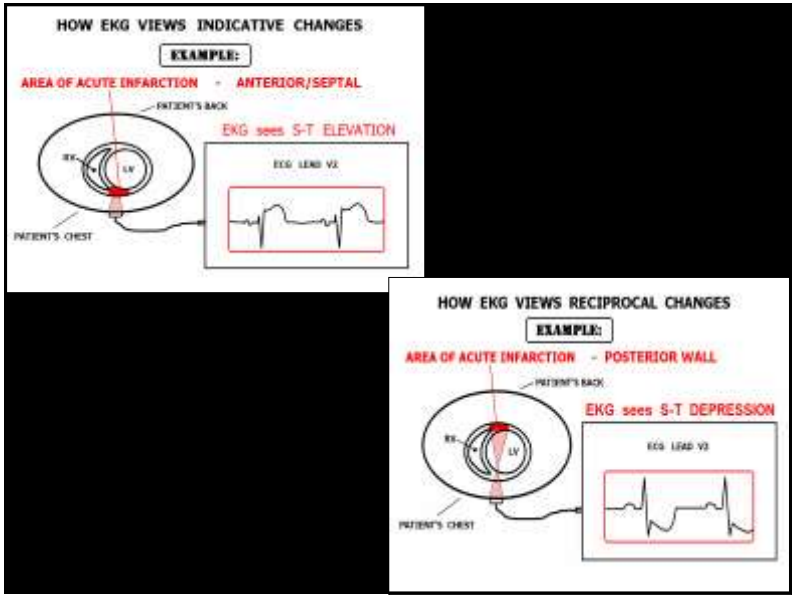
ANTERIOR
LAD
LATERAL
CIRC. or LAD


LEADS V1 - V3 view the
POSTERIOR WALL
R V
L V
via RECIPROCAL CHANGES.


HOW EKG VIEWS INDICATIVE CHANGES
EXAMPLE:
AREA OF ACUTE INFARCTION - ANTERIOR/SEPTAL
PATIENT'S BACK
EKG sees S-T ELEVATION
RV
LV
ECG LEAD V2
PATIENT'S CHEST


HOW EKG VIEWS INDICATIVE CHANGES
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RV
LV
ECG LEAD V2
PATIENT'S CHEST
HOW EKG VIEWS RECIPROCAL CHANGES
EXAMPLE:
AREA OF ACUTE INFARCTION - POSTERIOR WALL
PATIENT'S BACK
EKG sees S-T DEPRESSION
RV
LV
ECG LEAD V2
PATIENT'S CHEST


ST SEGMENT ELEVATION
ST SEGMENT DEPRESSION
LATERAL - ANTERIOR
DIAG (LAD) or OM (CIRC)
BASILAR SEPTAL
INF SEPTAL PERF.
ANTERIOR SEPTAL
LAD
ANTERIOR
LAD
INFERIOR
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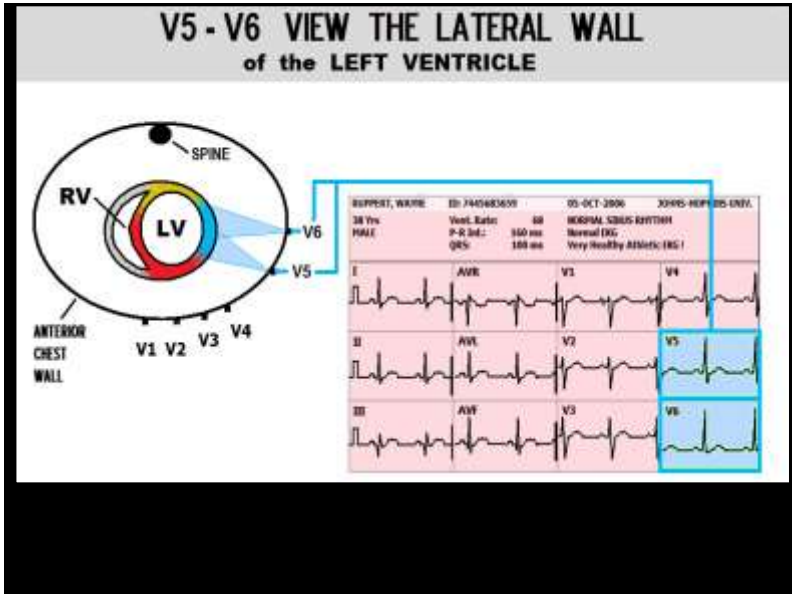
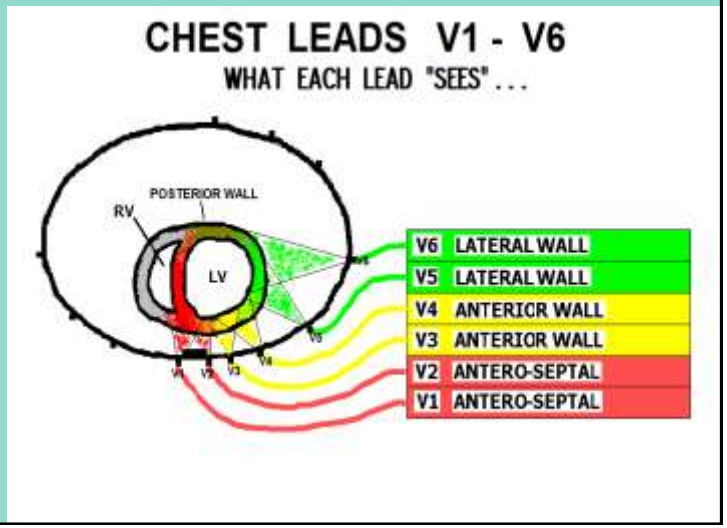
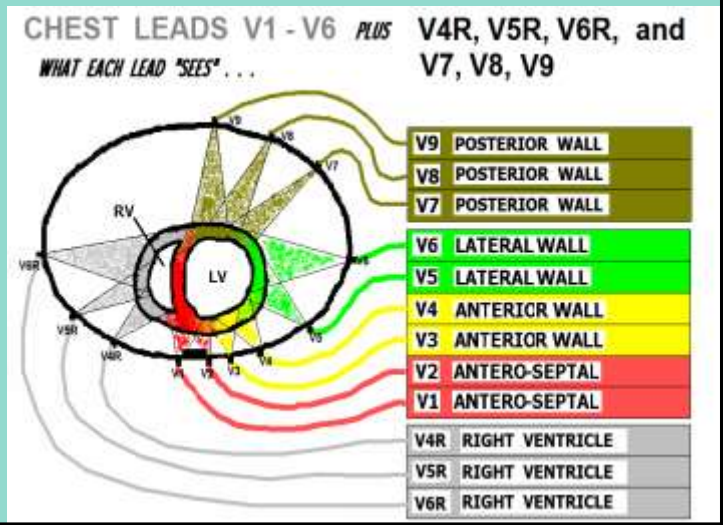
LATERAL - ANTERIOR
DIAG (LAD) or OM (CIRC)
ANTERIOR SEPTAL
LAD
LATERAL
CIRC. or LAD
INFERIOR
RCA or CIRC.
INFERIOR
RCA or CIRC.
ANTERIOR
LAD
LATERAL
CIRC. or LAD


V5 - V6 VIEW THE LATERAL WALL
of the LEFT VENTRICLE
SPINE
RV
LV
V6
V5
ANTERIOR
CHEST
WALL
V1 V2 V3 V4

THE 12 LEAD ECG HAS TWO MAJOR BLIND SPOTS . .



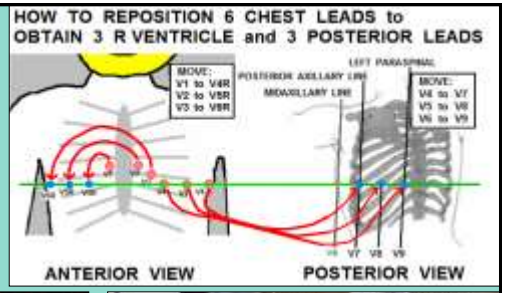
THE 18 LEAD ECG COVERS THE ENTIRE HEART . .

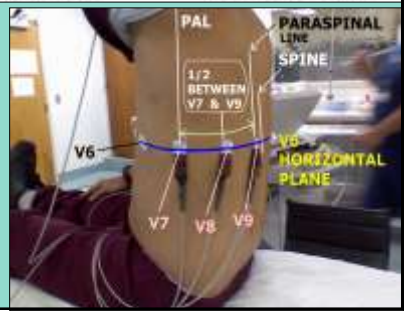


INDICATIONS for 18 Lead ECG include:

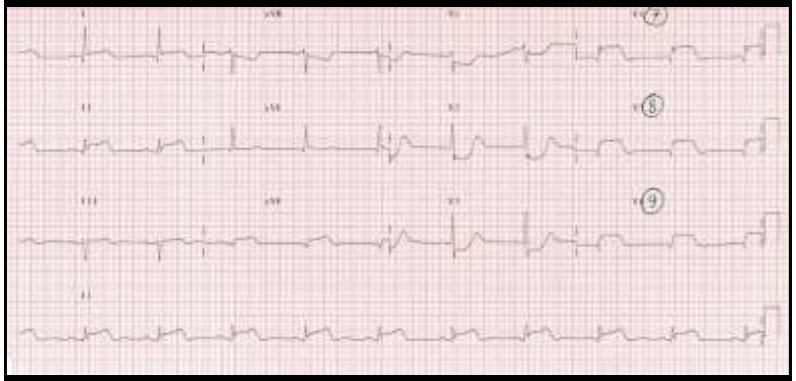
- **INFERIOR WALL MI**
- **ST Depression in LEADS V1-V4**

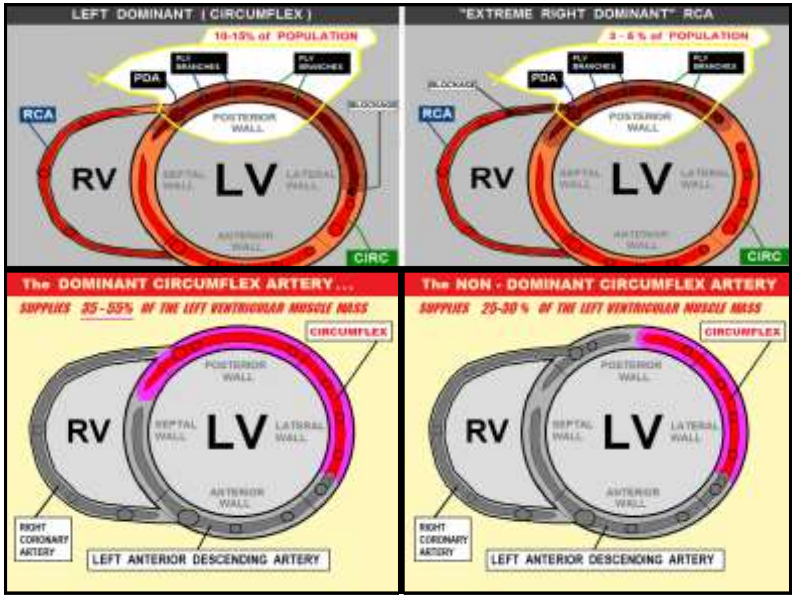
To do 18 Lead ECG with 12 Lead machine – after you obtain 12 Lead, reposition CHEST LEADS to this configuration, then print !



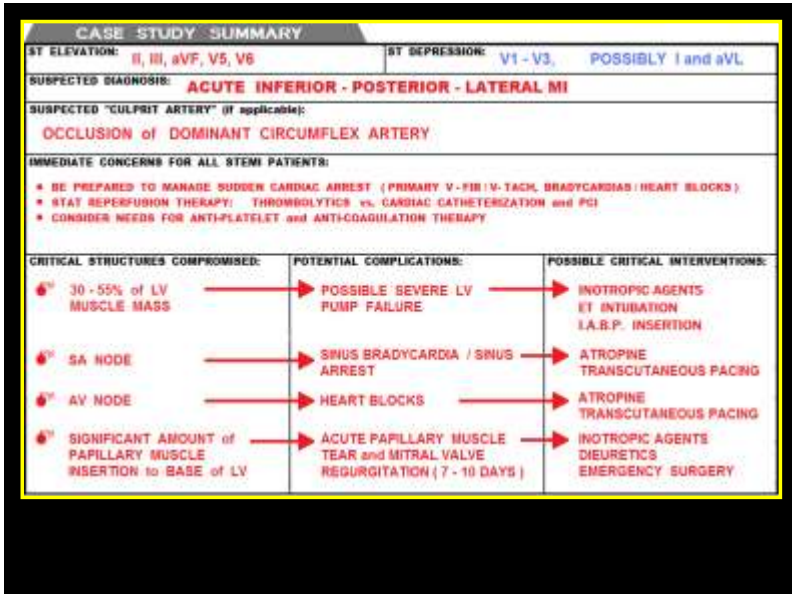


Posterior wall STEMI – ST Elevation
V7 – V9



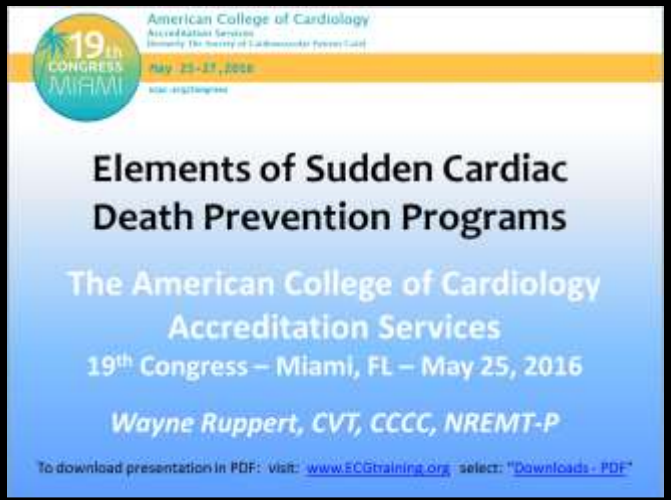



CIRCUMFLEX ARTERY OCCLUDED with significant THROMBUS.
PTCA of CIRCUMFLEX ARTERY.
DOMINANT CIRCUMFLEX ARTERY OPEN POST THROMBECTOMY with STENT DEPLOYMENT.
LVEF = 43%


CASE STUDY SUMMARY
ST ELEVATION: II, III, aVF, V5, V6
ST DEPRESSION: 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 MUSCLE MASS
POSSIBLE SEVERE LV PUMP FAILURE
INOTROPIC AGENTS
ET INTUBATION
I.A.B.P. INSERTION
SA NODE
SINUS BRADYCARDIA / SINUS ARREST
ATROPINE
TRANSCUTANEOUS PACING
AV NODE
HEART BLOCKS
ATROPINE
TRANSCUTANEOUS PACING
SIGNIFICANT AMOUNT of PAPILLARY MUSCLE INSERTION to BASE of LV
ACUTE PAPILLARY MUSCLE TEAR and MITRAL VALVE REGURGITATION (7 - 10 DAYS)
INOTROPIC AGENTS
DIURETICS
EMERGENCY SURGERY

"NOWHERE", NEW MEXICO, 1994

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



Brief, focused ECG excerpts from the "19th Congress, American College of Cardiology Accreditation Services" national conference, Miami, 2016…….

Prevalence
SADS Foundation Stats:

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

The SADS Conditions:

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

Estimated SADS Prevalence in US Population:

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

Prevalence

Sudden Deaths in Young Competitive Athletes

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

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

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

Prevalence

Adverse Drug Reactions: Torsades de Pointes secondary to QT prolonging medications:

- Occur in and out of hospital
- Underreported
- Medical community undereducated
- 7,000 in-hospital ADRs / year (all cause)
- Major issue with pharmaceutical industry, many drugs removed from market due to high incidence of TdP and TdP associated mortality

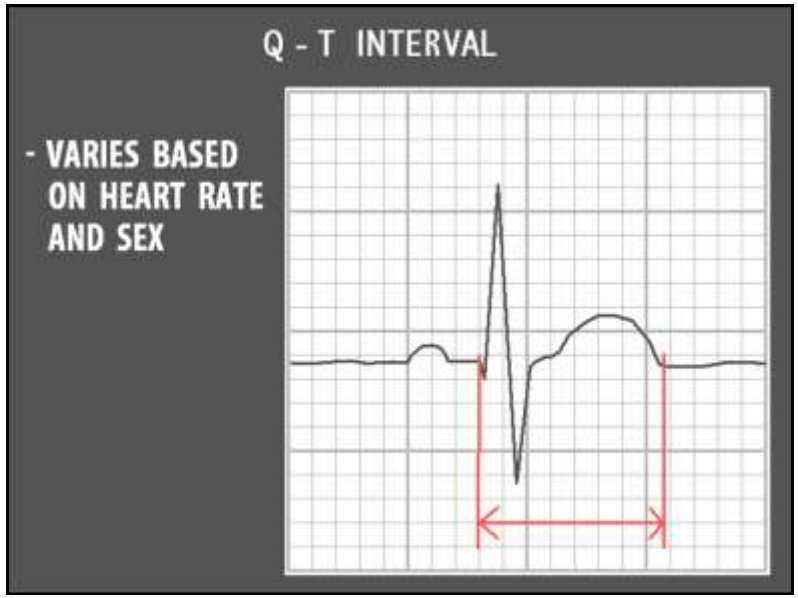
Compared to sudden death from CAD, SADS mortality prevalence is low, HOWEVER . . . .

- Nearly EVERY SADS death is a NEEDLESS TRAGEDY that could have been AVOIDED with appropriate screening and management.
- Many SADS victims are infants, children and young adults who are otherwise healthy.
- Sudden death is often the first symptom of SADS
- Diagnosed and managed properly, SADS patients can live long, productive and happy lives

☞ **Leave the detailed ECG diagnosis to the cardiologist.**

☞ **Leave the detailed ECG diagnosis to the cardiologist.**

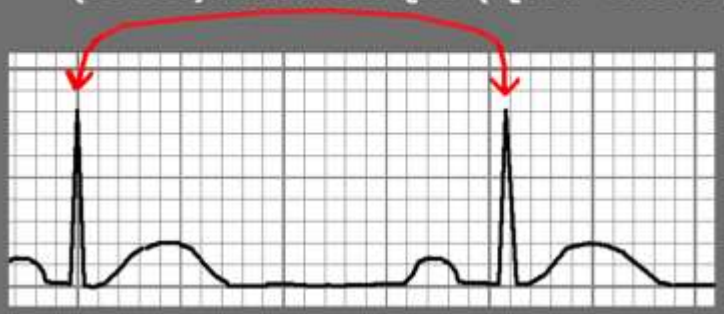
However every critical care nurse, paramedic or other professional who reads an ECG should be aware of some important clues . . .



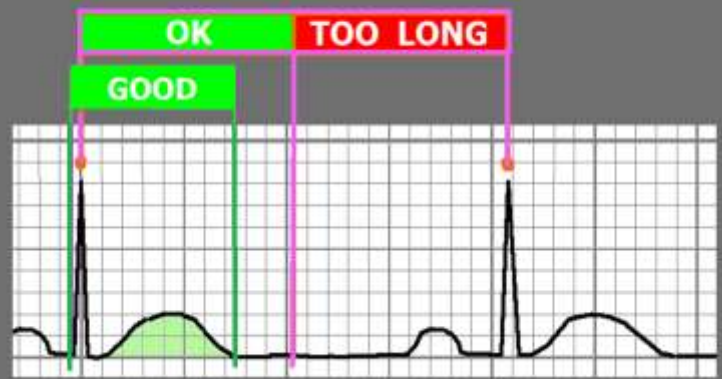
DETERMINING Q-T INTERVAL LIMITS

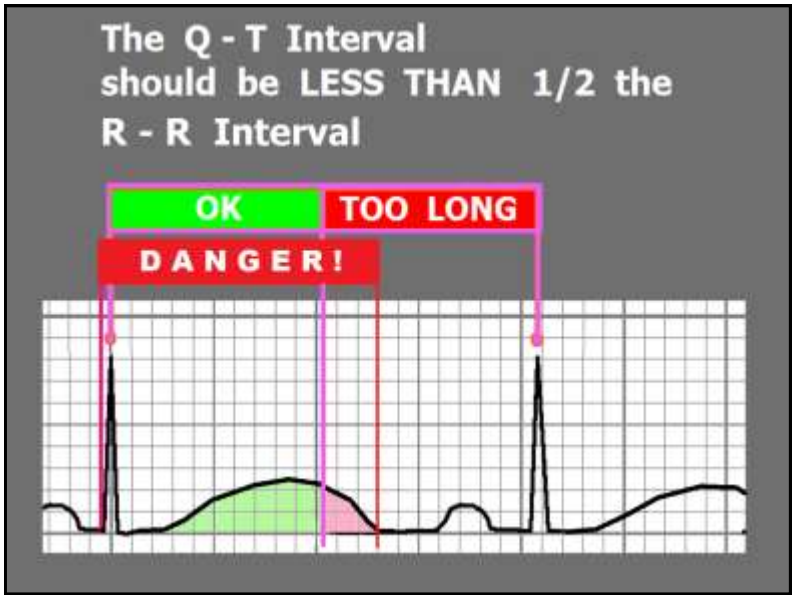
THE "QUICK PEEK" METHOD

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



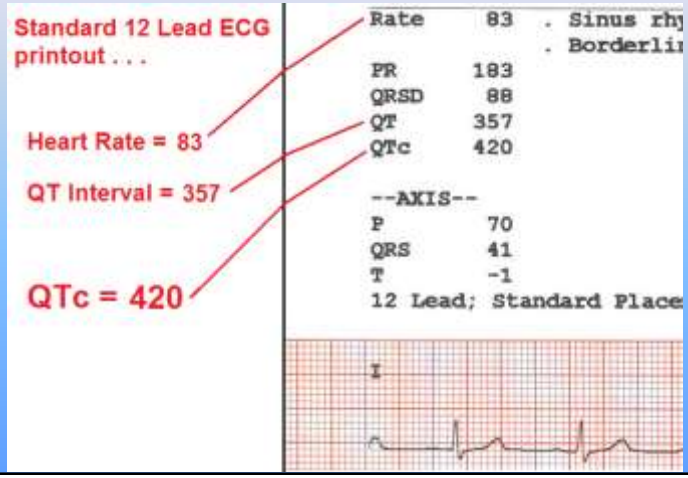
The Q - T Interval should be LESS THAN 1/2 the R - R Interval

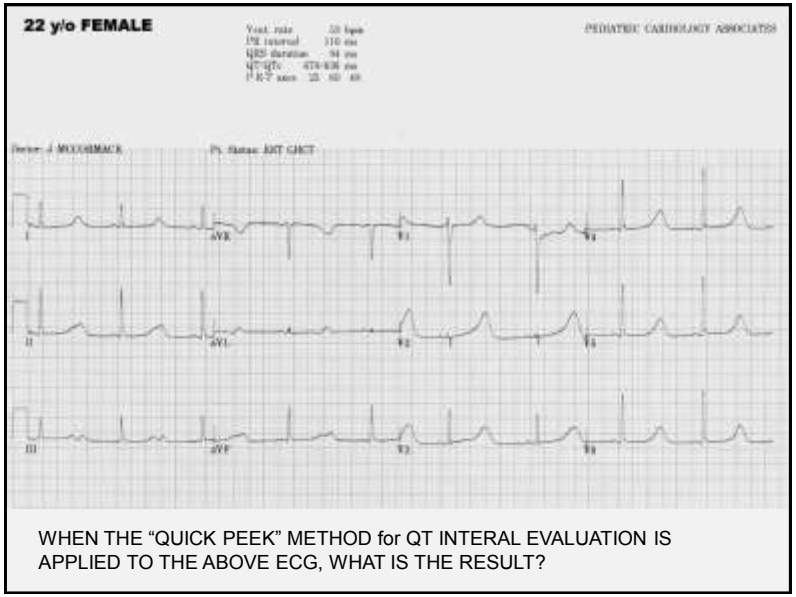




Determining the QT / QTc

Method 1 – 12 Lead ECG Report:





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

QTc Values:

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

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

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

Torsades de Pointes (TdP) – HEMODYNAMICS:

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

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

a piece of Twisted Ribbon !

Etiology of Long QT Syndromes:

Congenital (14 known subtypes)

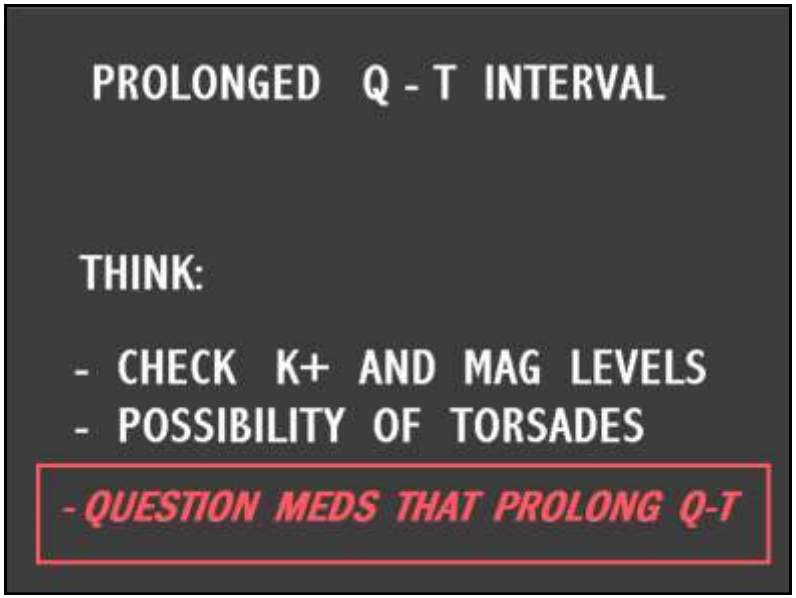
Genetic mutation results in abnormalities of cellular ion channels

Acquired

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

Miscellaneous

- Coronary Artery Disease
- Mitral Valve Prolapse



QT Prolongation -- *STAT Intervention:*

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

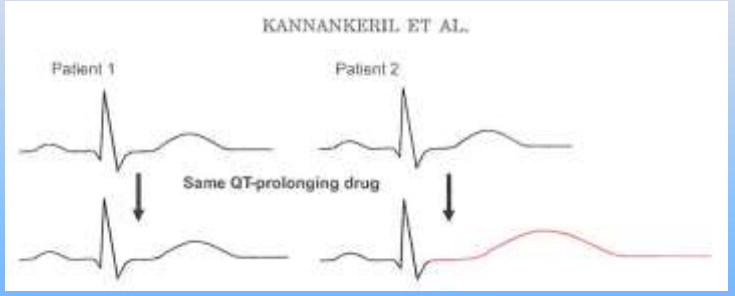
Commonly used QT prolonging meds include:

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

and MANY more!

PATIENT 1: NORMAL

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

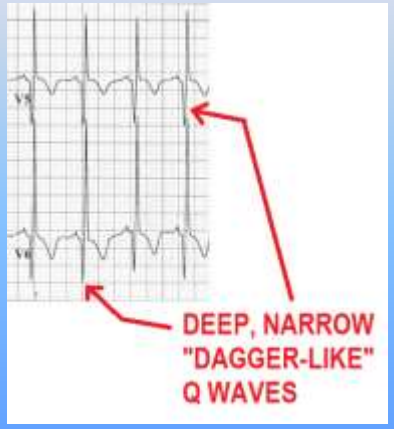


Click here for link to paper by Kannankeril et al (2010 Pharmacological Reviews) that describes genetic susceptibility described above.

ECG Indicators: Hypertrophic Cardiomyopathy

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

ECG Indicators: Hypertrophic Cardiomyopathy



ECG Indicators: Hypertrophic Cardiomyopathy

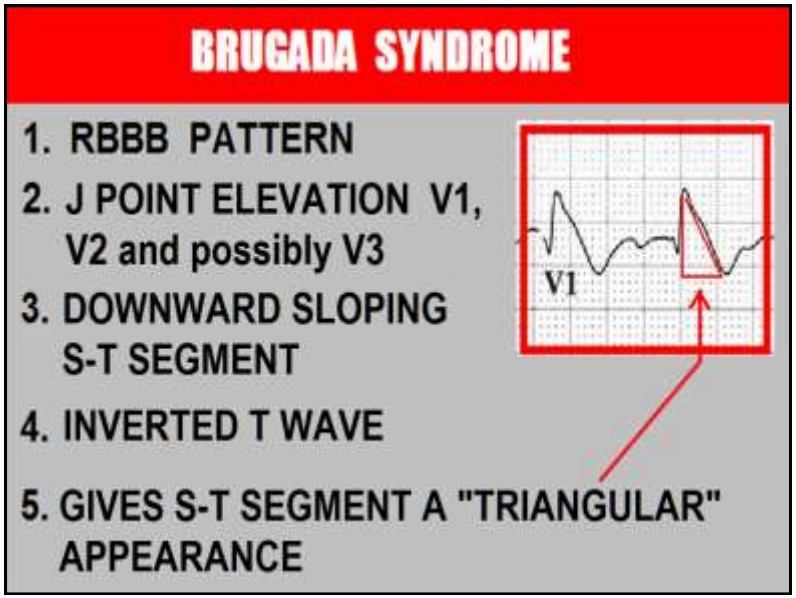
- ECG may be normal
- Deep, narrow (dagger-like) Q waves
- Inverted T waves in multiple regions
- Left Ventricular and possibly Left Atrial Hypertrophy

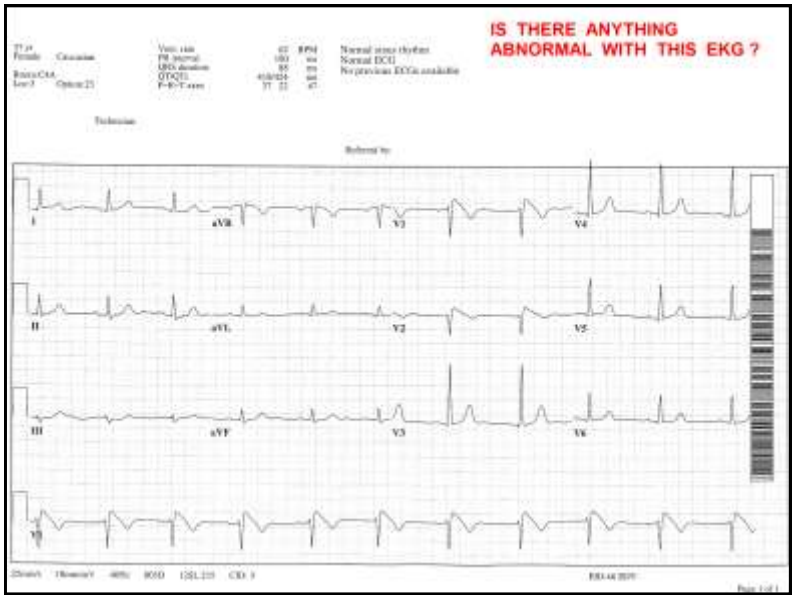
Hypertrophic Cardiomyopathy (HCM)

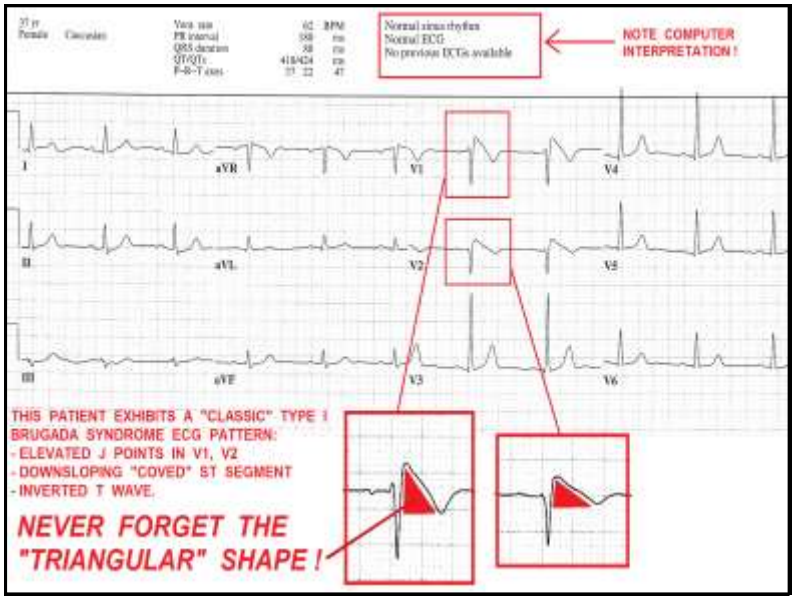
12 Lead ECG Traits:

- **QRS Height -- exceeds normal size, "spearing through QRS" in other leads**
- **Inverted T waves appear in multiple regions (ANTERIOR, LATERAL)**
- **BiPHASIC T waves in Inferior Leads.**
- **T WAVES are SYMMETRICAL .**

ECG Indicators:
Brugada Syndrome




IS THERE ANYTHING ABNORMAL WITH THIS EKG ?


Normal sinus rhythm
Normal ECG
No previous ECGs available
NOTE COMPUTER INTERPRETATION !
THIS PATIENT EXHIBITS A "CLASSIC" TYPE I BRUGADA SYNDROME ECG PATTERN:
- ELEVATED J POINTS IN V1, V2
- DOWNSLOPING "COVED" ST SEGMENT
- INVERTED T WAVE.
NEVER FORGET THE "TRIANGULAR" SHAPE !

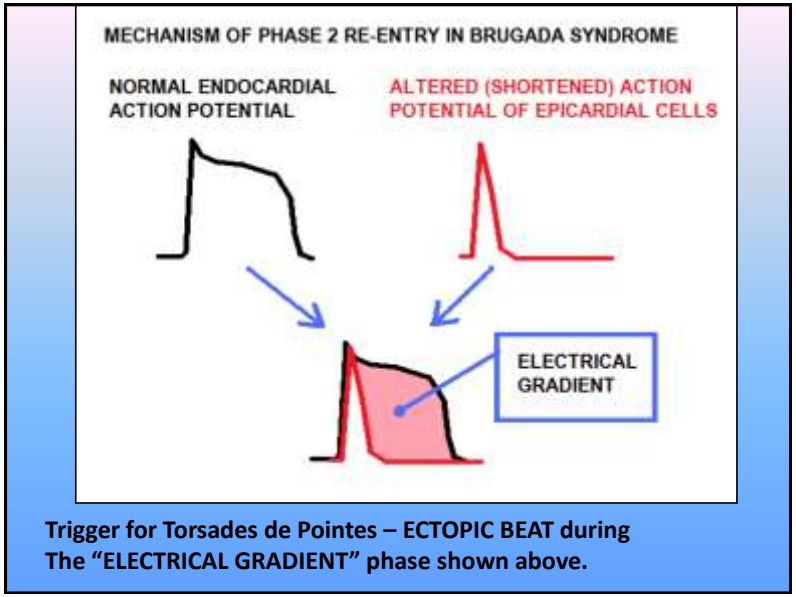
PATTERNS of S-T ELEVATION :

BEWARE of the

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

THINK - -

BRUGADA SYNDROME

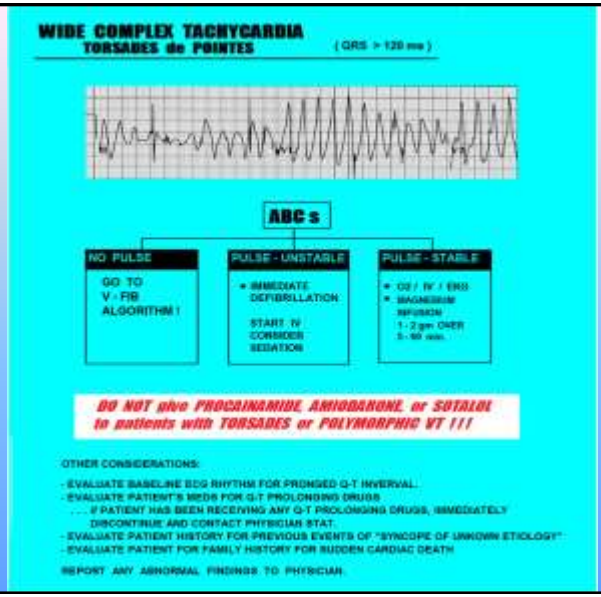


Trigger for Torsades de Pointes – ECTOPIC BEAT during The "ELECTRICAL GRADIENT" phase shown above.

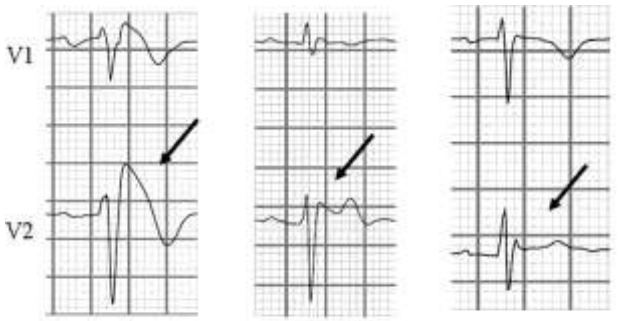
Brugada / Long QT Syndromes cause:

Torsades de Pointes:

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



ECG abnormality diagnostic or suspected of Brugada syndrome.



Type 1: Coved type ST-segment elevation

Type 2: saddle-back type ST-segment elevation

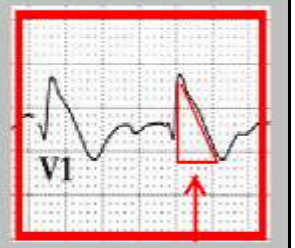
Type 3: Saddle-back type "ST-segment elevation"

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

American Heart Association

BRUGADA SYNDROME

- SEVERAL VARIATIONS of this disorder are known to exist.
- CONCEALED and NON-CONCEALED.
- The NON-CONCEALED version HAS THE V1-V3 abnormality VISIBLE at all times.
- The CONCEALED version - pt. has a NORMAL EKG at most times - a DRUG STUDY, an EP STUDY, and / or GENETIC TESTING must be done to rule out or confirm diagnosis.



Arrhythmogenic Right Ventricular Dysplasia

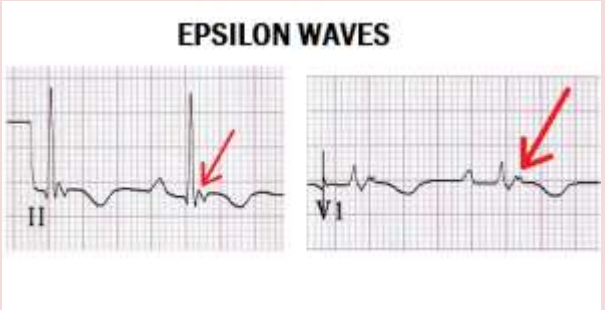
- A genetically acquired myocardial disease associated with paroxysmal ventricular arrhythmias and sudden cardiac death.
- Characterized pathologically by fibro-fatty replacement of the right ventricular myocardium.
- The second most common cause of sudden cardiac death in young people (after HOCM),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

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

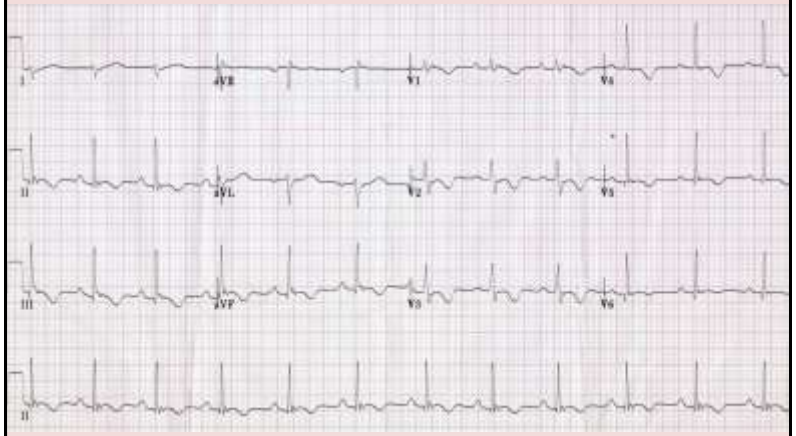
Arrhythmogenic Right Ventricular (RV) Cardiomyopathy and/or Dysplasia:

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

ARVD – 12 Lead ECG Indicators

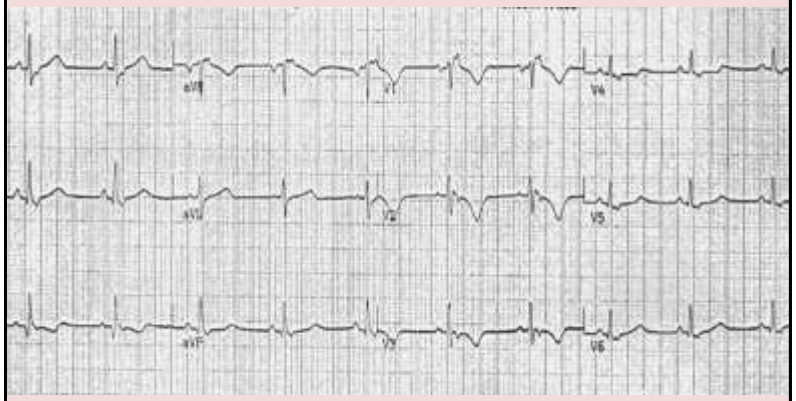


ARVD ECG 1



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

ARVD ECG 2



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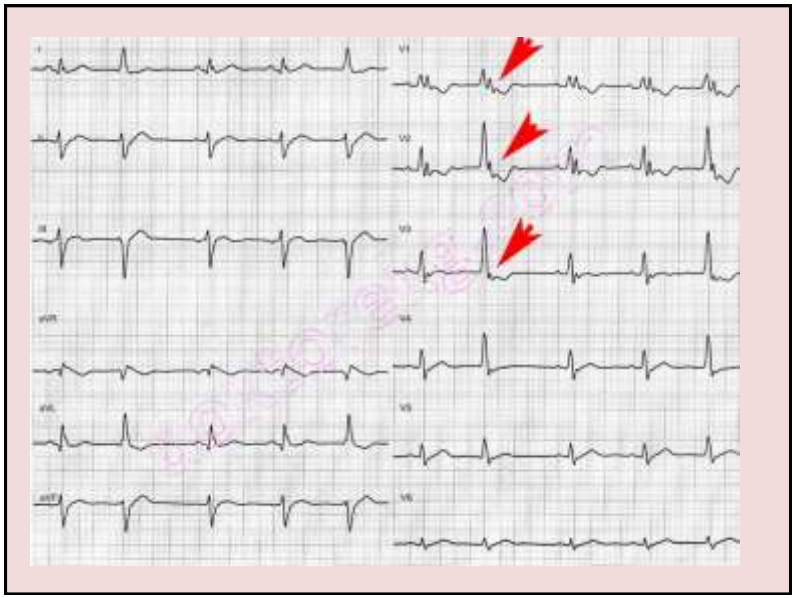
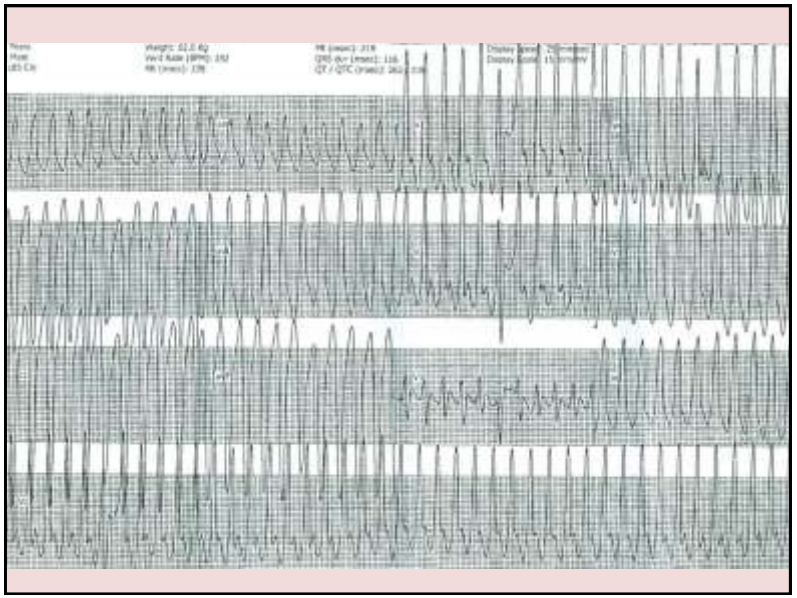
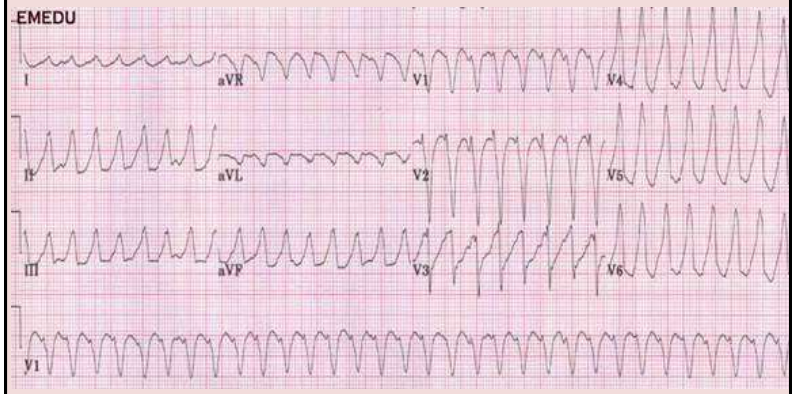
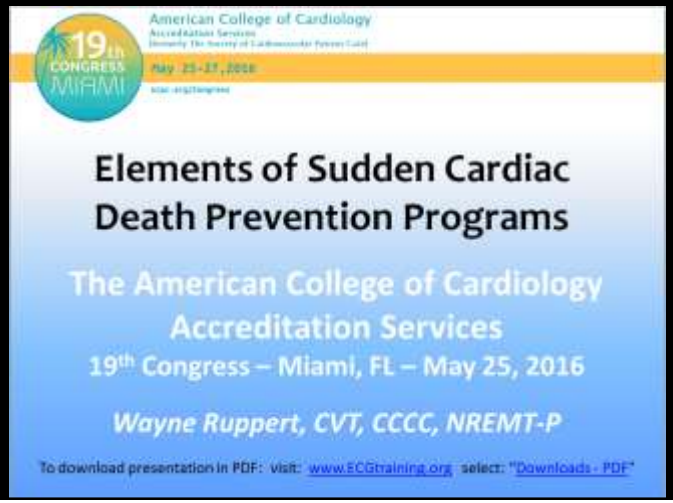




ARVD INDUCED VT



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



Evidence Based Reference Sources

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

Evidence Based Reference Sources, cont'

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

Other Reference Sources:

www.JACC.org

http://circ.ahajournals.org/

www.SADS.org

QUESTIONS ? ? ?

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

JEREMY, ME, and DAD -- at LEE'S DINER, YORK, PA 2003