12 LEAD ECG INTERPRETATION in

ACUTE CORONARY SYNDROME

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

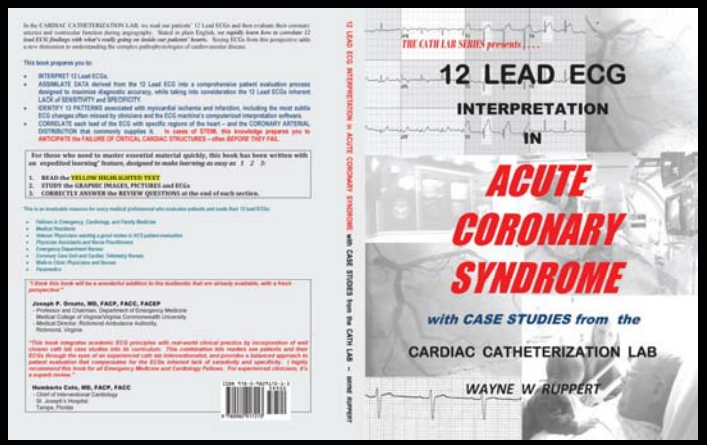
- Didactic Materials:

"Practical Electrocardiography" by Galen Wagner, MD and Henry J. L. Marriott, MD
"Practical Electrophysiology" by Richard Fogoros, MD

70+ current Medical Journal article citations: primary sources NEJM, JACC, JAMA, AHA Circulation, + others

CASE STUDIES from St. Joseph's Hospital CARDIAC CATH LAB 1997 – TODAY

PowerPoint presentation converted to TEXTBOOK in 2010.



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TEXTBOOK REVIEWED BY:

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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 . . .

The EKG in PERSPECTIVE

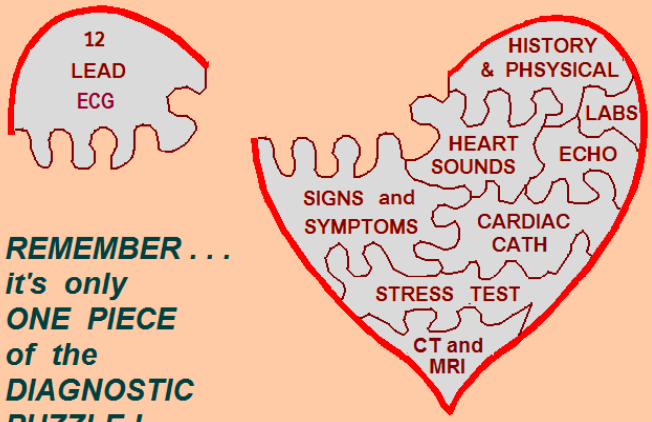
PROBLEMS WITH EKGs . . .

↓ SENSITIVITY
(FALSE NEGATIVES)

↓ SPECIFICITY
(FALSE POSITIVES)

AND . . .

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



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

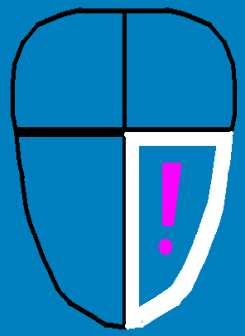
THE CHAMBER MOST IMPORTANT TO KEEPING THE PATIENT ALIVE

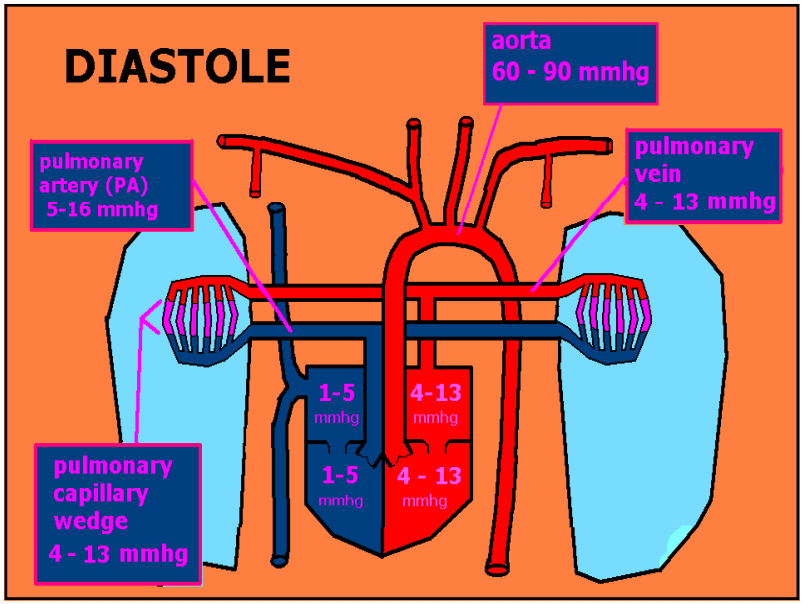
(and the ONLY one you can't live without)

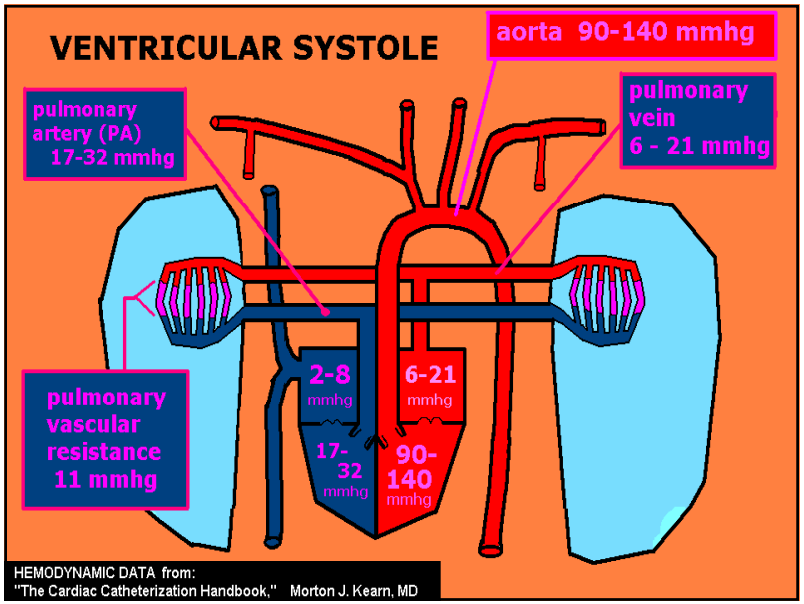
IS THE

LEFT VENTRICLE

WHICH WE WILL REFER TO AS **THE PUMP**





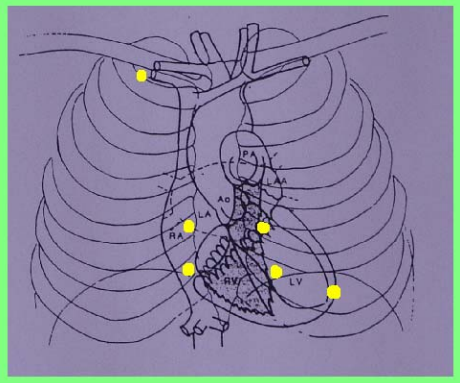


VERY BASIC HEART SOUNDS ASSESSMENT

ABNORMAL EKG CHANGES THAT MAY PRESENT WITH ABNORMAL HEART SOUNDS:

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

HEART SOUNDS ASSESSMENT



VERY BASIC HEART SOUNDS ASSESSMENT

- Normal Heart Sounds
- Murmurs
  - systolic
  - diastolic
- Friction Rubs

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

HEART SOUNDS ASSESSMENT

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

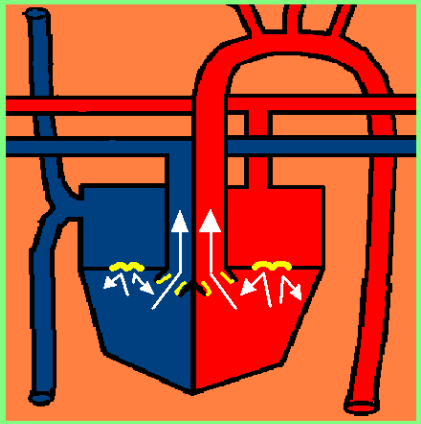
THERE ARE TWO NORMAL HEART SOUNDS, KNOWN AS: S-1 and S-2

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

HEART SOUNDS ASSESSMENT

S-1 BEGINNING OF SYSTOLE.

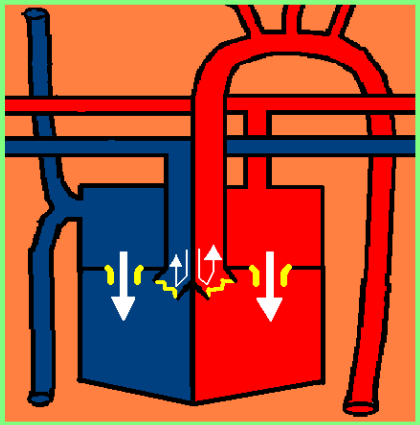
SOUND OF THE MITRAL AND TRICUSPID VALVES CLOSING.



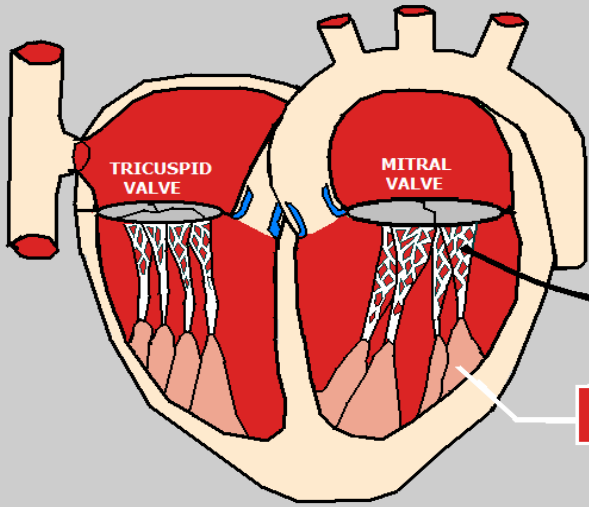
HEART SOUNDS ASSESSMENT

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

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



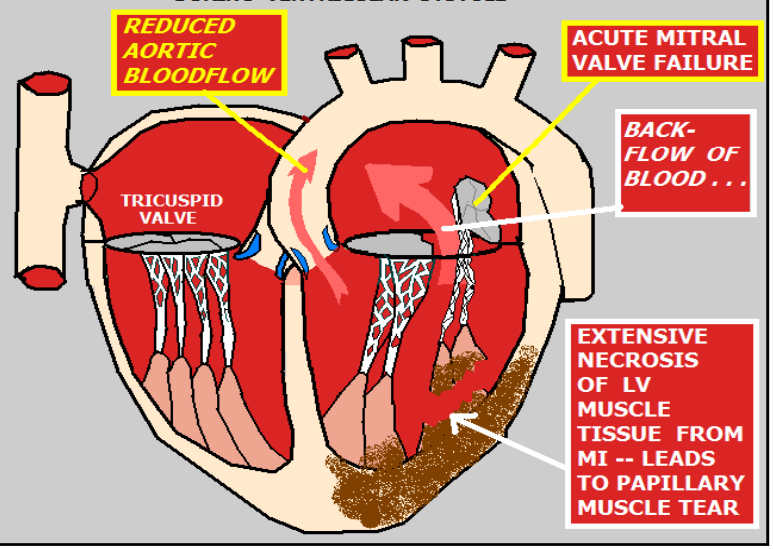
ATRIO-VENTRICULAR VALVES



ARE SECURED TO THE ENDO-CARDIAL SURFACE BY THE CHORDAE TENDINEAE AND THE PAPILLARY MUSCLES

ACUTE MITRAL REGURGITATION

DURING VENTRICULAR SYSTOLE



BASIC HEART SOUNDS ASSESSMENT

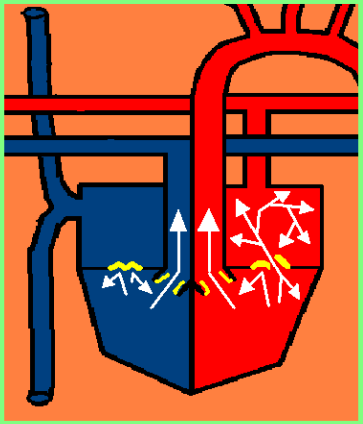
MURMUR = "SWOOSH" SOUND CAUSED BY THE SOUND OF TURBULENCE.

S-1 MURMUR SOUNDS LIKE:

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

CAUSE OF SYSTOLIC (S 1) MURMUR

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

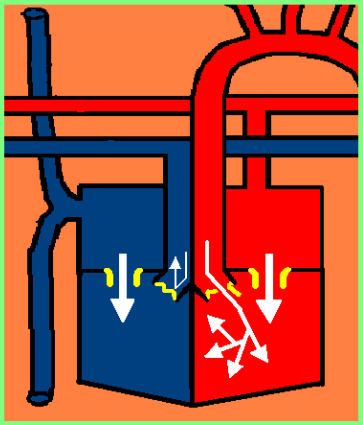


- MOST SYSTOLIC MURMURS CAUSED BY MITRAL VALVE FAILURE.
- ACUTE MITRAL VALVE REGURGITATION IS A POTENTIALLY LETHAL COMPLICATION OF ACUTE / RECENT EXTENSIVE TRANSMURAL MI

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

CAUSE OF DIASTOLIC (S 2) MURMUR

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



BASIC HEART SOUNDS ASSESSMENT

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

S-2 MURMUR SOUNDS LIKE:

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

- AORTIC VALVE FAILURE MOST COMMON CAUSE OF S-2 MURMUR
- DUE TO THE HIGHER PRESSURES OF THE LEFT SIDE OF THE HEART

BASIC HEART SOUNDS ASSESSMENT

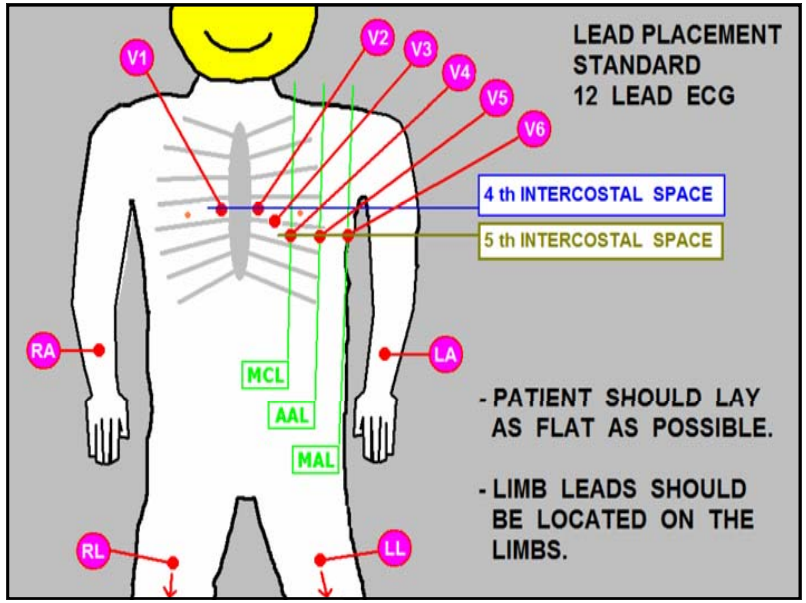
FRICTION RUB

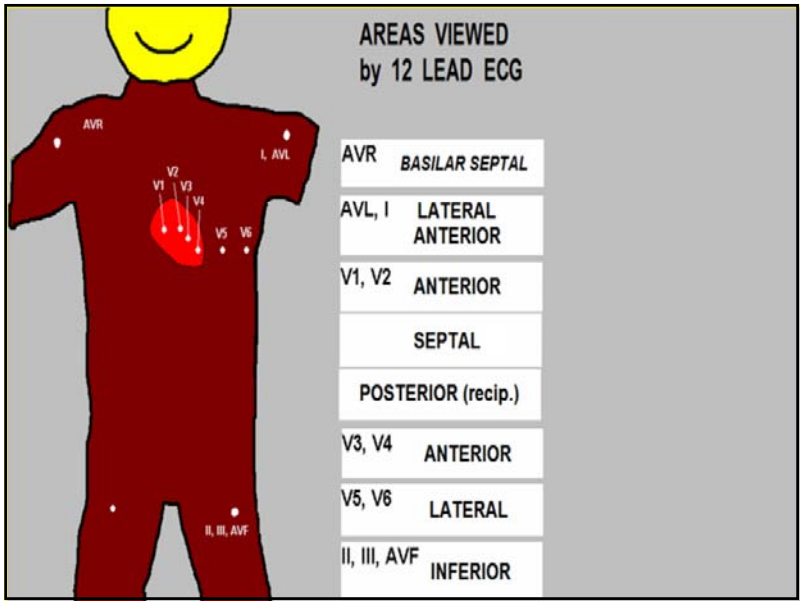
- ASSOCIATED WITH PERICARDITIS
- SOUNDS LIKE THE GENTLE RUBBING OF SANDPAPER
- HAS 3 COMPONENTS: SYSTOLIC, EARLY, and LATE DIASTOLIC

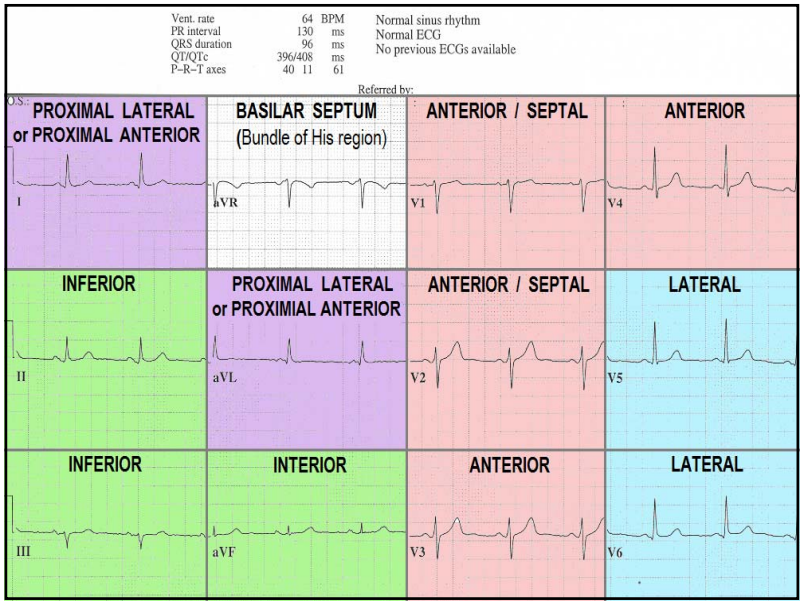
BASIC HEART SOUNDS ASSESSMENT

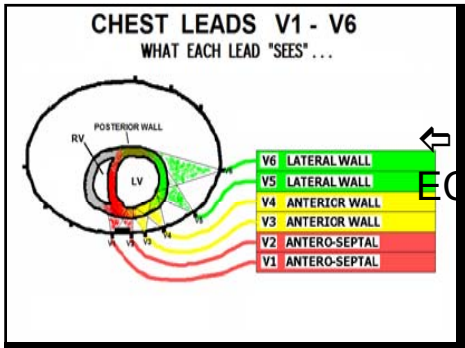
FRICTION RUB

- IS PRESENT IN MOST ACUTE TRANSMURAL MI PATIENTS
- MAY BE PRESENT WITHIN HOURS AFTER ONSET
- IS TRANSIENT -- MAY LAST FOR A FEW DAYS





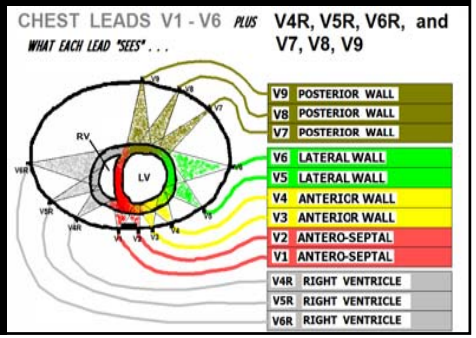




The standard 12 Lead ECG has TWO significant BLIND SPOTS:

- RIGHT VENTRICLE
- POSTERIOR WALL of Left Ventricle

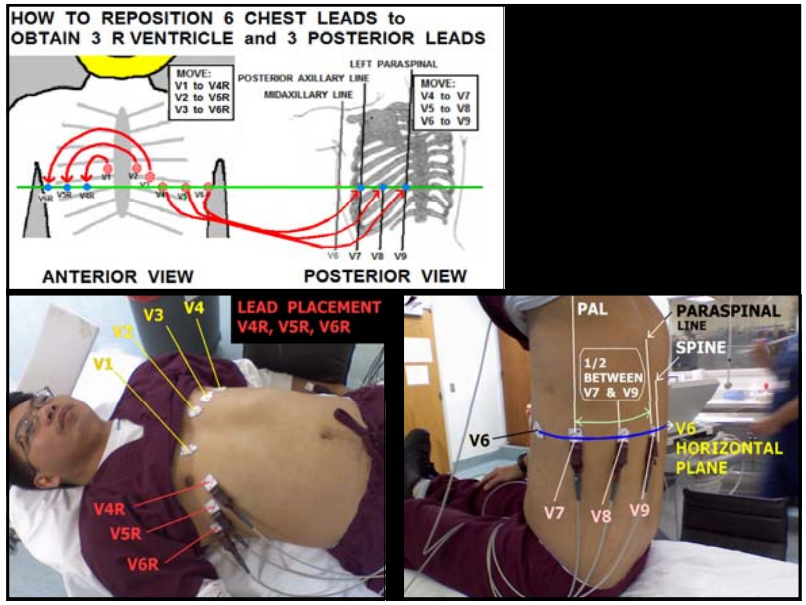
To SEE the RIGHT VENTRICLE and the POSTERIOR WALL, you must do an 18 LEAD ECG !



To do this with a STANDARD 12 Lead ECG machine,

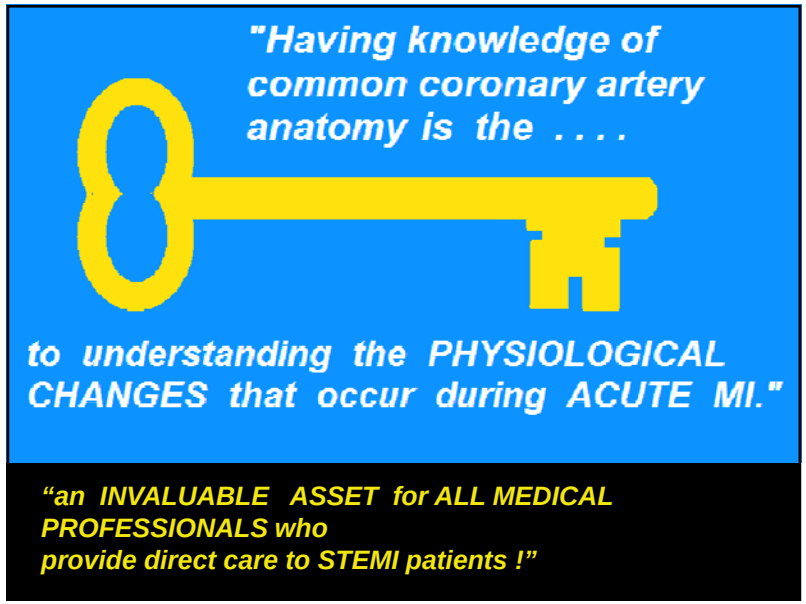
FIRST, do you 12 Lead ECG in the traditional manner,

and then you will REPOSITION the CHEST LEADS like this . . . .



INDICATIONS for an 18 LEAD ECG include whenever you see:

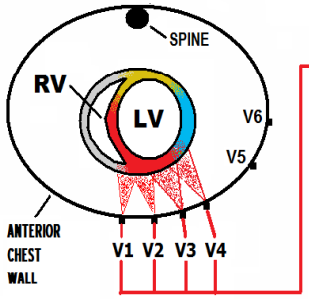
- INFERIOR WALL STEMI
- ST DEPRESSION in any of the ANTERIOR LEADS (V1 – V4)



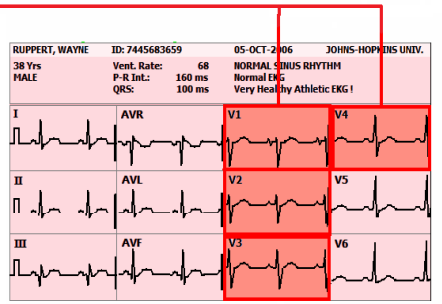
INTERPRET THE EKG, THEN:

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

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

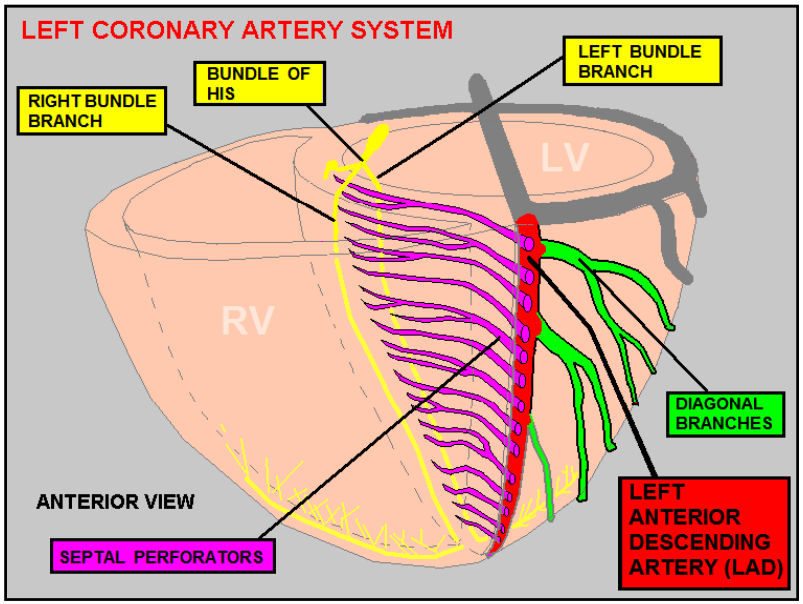


V1, V2 - ANTERIOR / SEPTAL
V3, V4 - ANTERIOR



Which Coronary Artery typically Supplies the ANTERIOR WALL ?

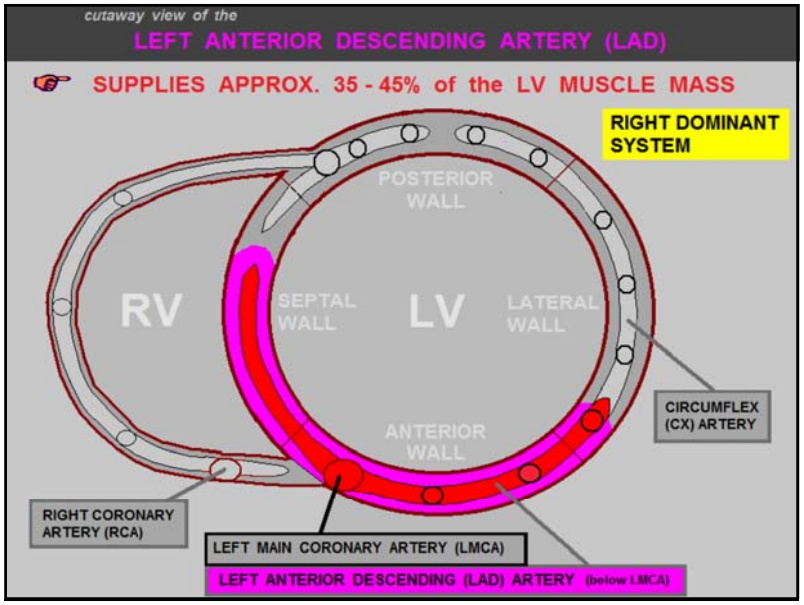
LEFT CORONARY ARTERY SYSTEM

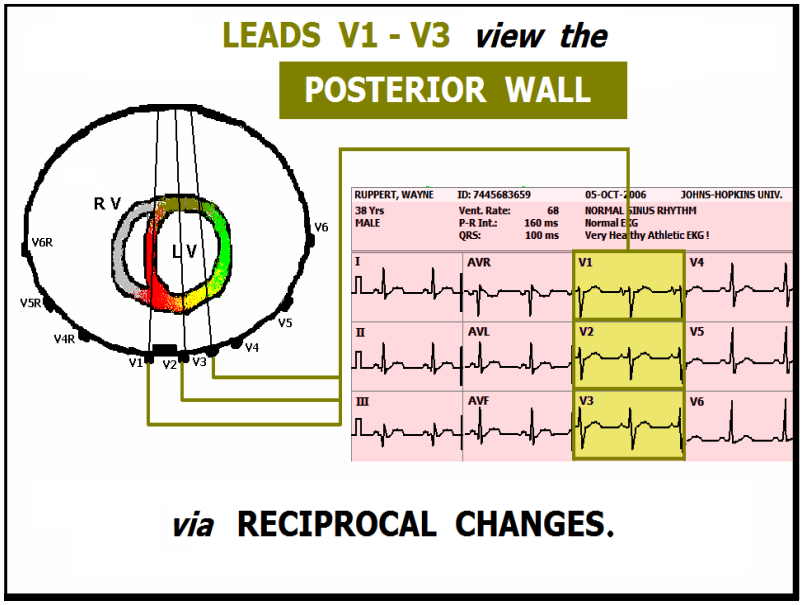


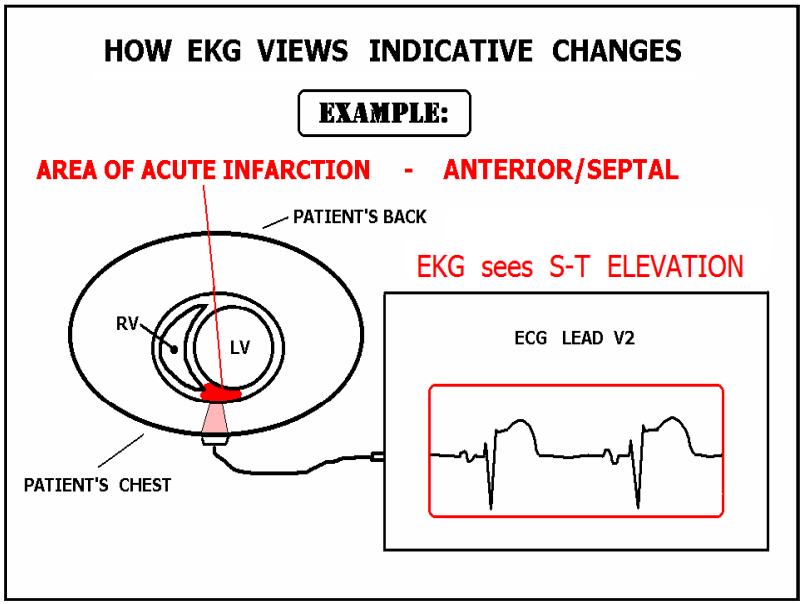
HELPFUL HINT . . . *MEMORIZE THIS !*

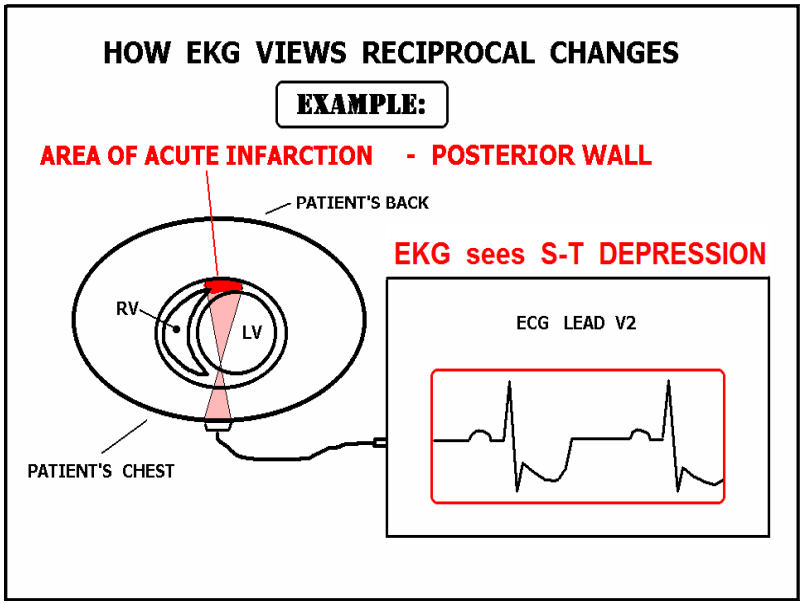
LEFT ANTERIOR DESCENDING ARTERY (LAD)

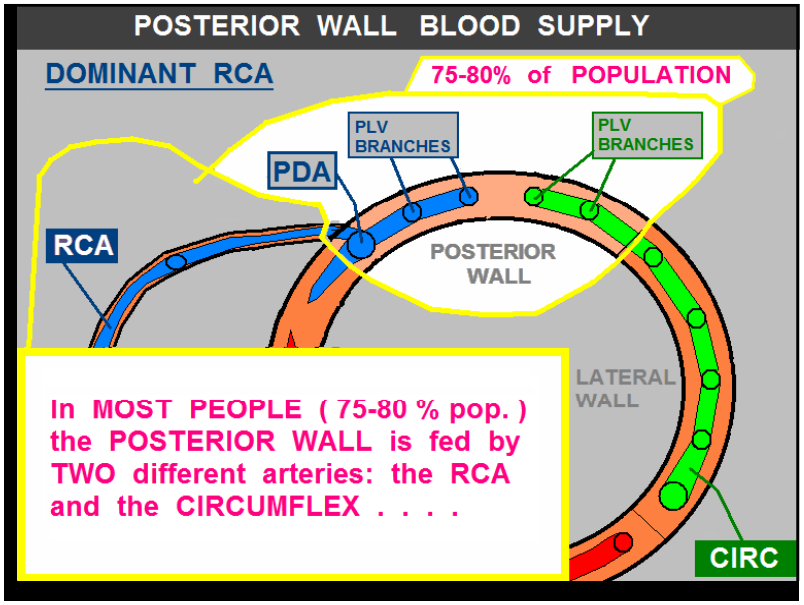
- BUNDLE OF HIS
- BUNDLE BRANCHES ( )
- 35 - 45 % OF LV MUSCLE MASS
  - ANTERIOR WALL
  - SEPTAL WALL (anterior 2/3)


cutaway view of the
LEFT ANTERIOR DESCENDING ARTERY (LAD)
SUPPLIES APPROX. 35 - 45% of the LV MUSCLE MASS
RIGHT DOMINANT SYSTEM
POSTERIOR WALL
RV
SEPTAL WALL
LV
LATERAL WALL
ANTERIOR WALL
CIRCUMFLEX (CX) ARTERY
RIGHT CORONARY ARTERY (RCA)
LEFT MAIN CORONARY ARTERY (LMCA)
LEFT ANTERIOR DESCENDING (LAD) ARTERY (below LMCA)


LEADS V1 - V3 view the
POSTERIOR WALL
R V
LV
V6
V6R
V5R
V4R
V5
V4
V1
V2
V3
RUPPERT, WAYNE
ID: 7445683659
05-OCT-2006
JOHNS-HOPKINS UNIV.
38 Yrs
MALE
Vent. Rate: 68
P-R Int.: 160 ms
QRS: 100 ms
NORMAL SINUS RHYTHM
Normal ECG
Very Healthy Athletic EKG !
I
AVR
V1
V4
II
AVL
V2
V5
III
AVF
V3
V6
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 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



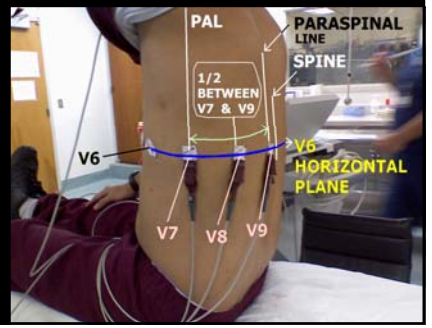
POSTERIOR WALL MI usually accompanies INFERIOR and/or LATERAL WALL MI !!!

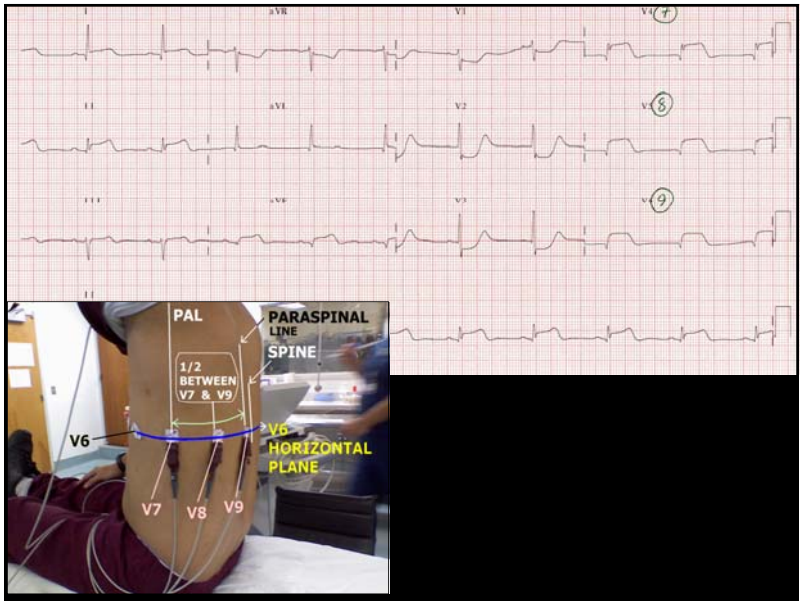
POSTERIOR WALL MI usually accompanies INFERIOR and/or LATERAL WALL MI !!!

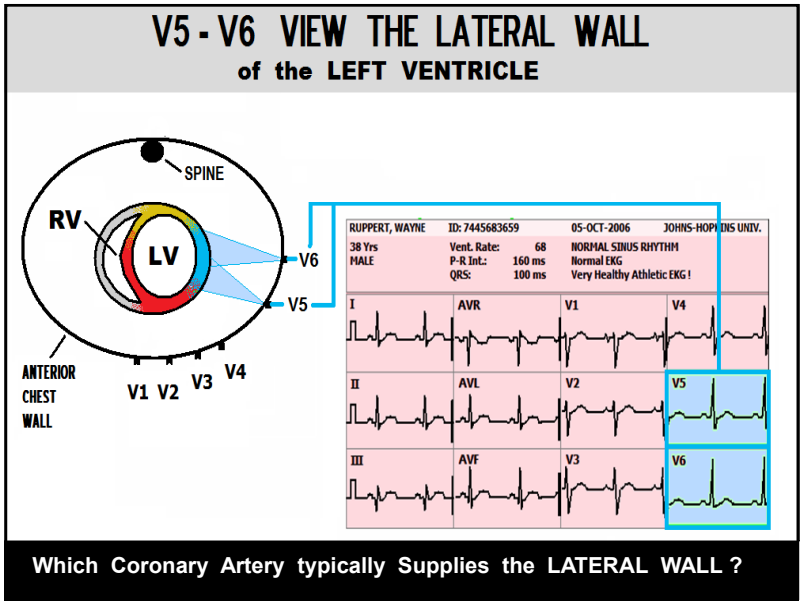
. . . . On rare occasions, we see isolated cases of POSTERIOR WALL MI

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

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




PAL
PARASPINAL LINE
SPINE
1/2 BETWEEN V7 & V9
V6
V6 HORIZONTAL PLANE
V7
V8
V9


V5 - V6 VIEW THE LATERAL WALL
of the LEFT VENTRICLE
SPINE
RV
LV
V6
V5
ANTERIOR CHEST WALL
V1 V2
V3
V4
RUPPERT, WAYNE
ID: 7445683659
05-OCT-2006
JOHNS-HOPKINS UNIV.
38 Yrs
MALE
Vent. Rate: 68
P-R Int.: 160 ms
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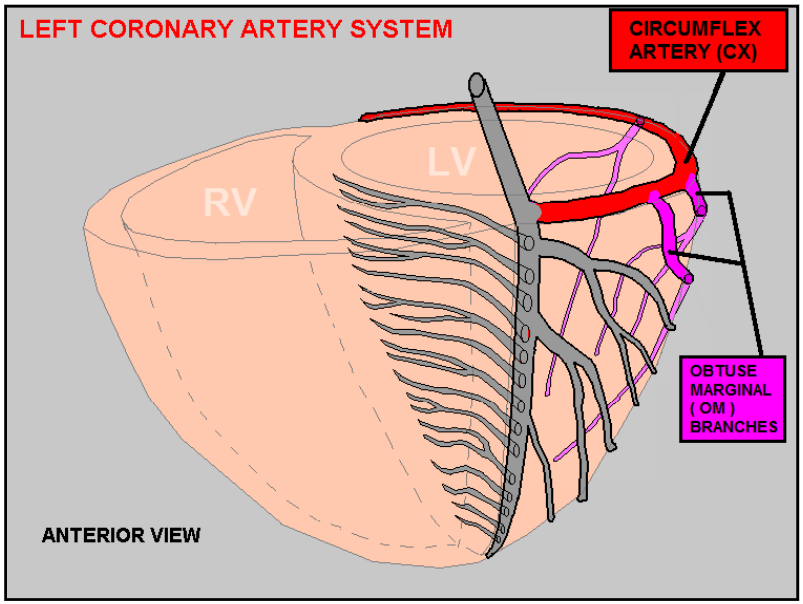
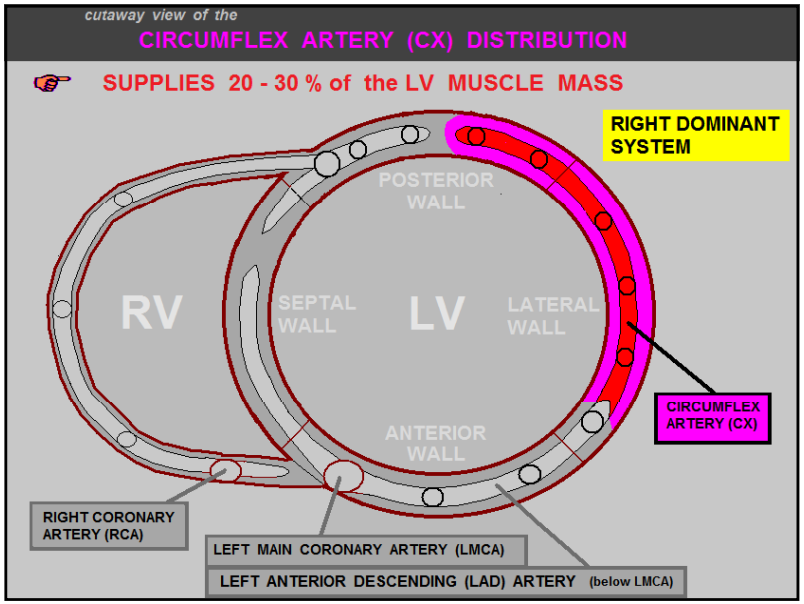
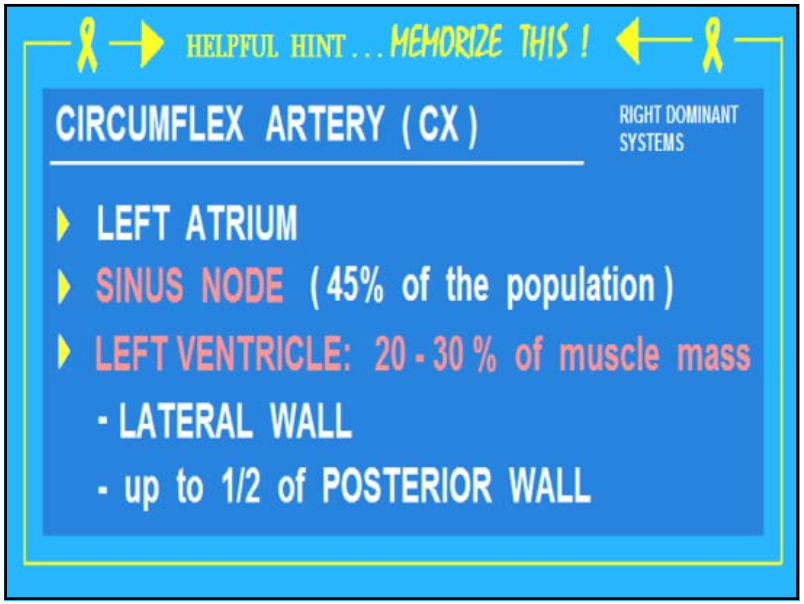
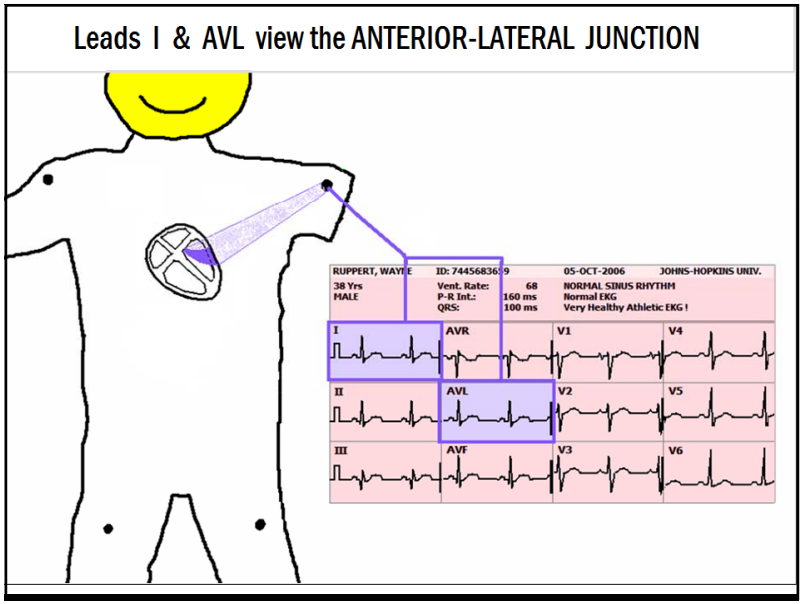
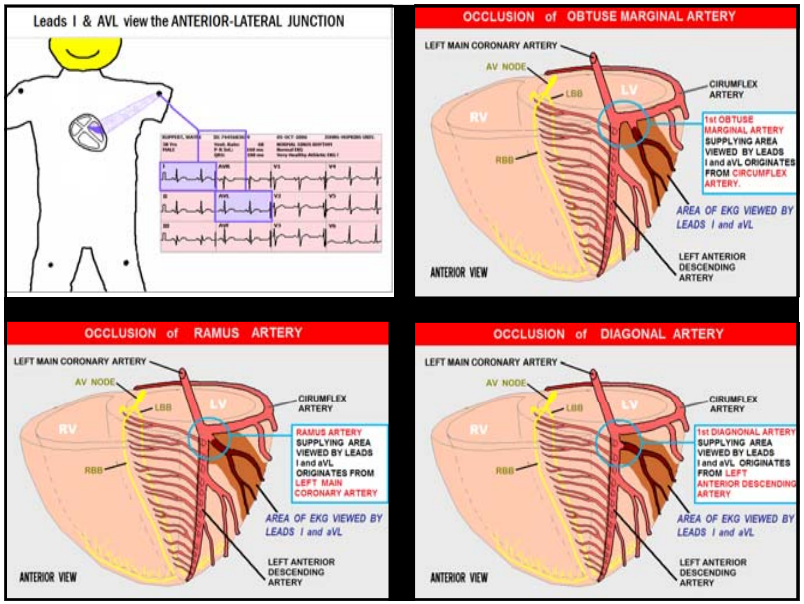
NORMAL SINUS RHYTHM
Normal EKG
Very Healthy Athletic EKG !
I
AVR
V1
V4
II
AVL
V2
V5
III
AVF
V3
V6
Which Coronary Artery typically Supplies the LATERAL WALL ?

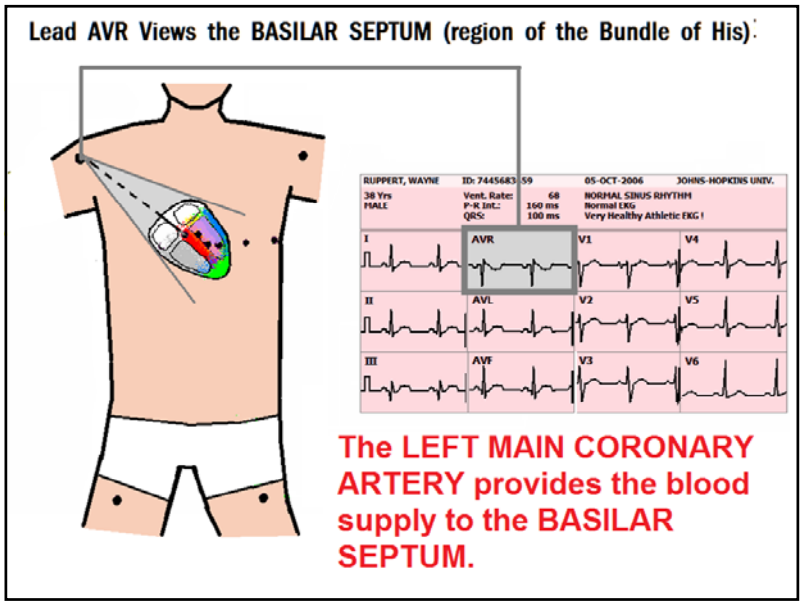

LEFT CORONARY ARTERY SYSTEM
CIRCUMFLEX ARTERY (CX)
RV
LV
OBTUSE MARGINAL (OM) BRANCHES
ANTERIOR VIEW

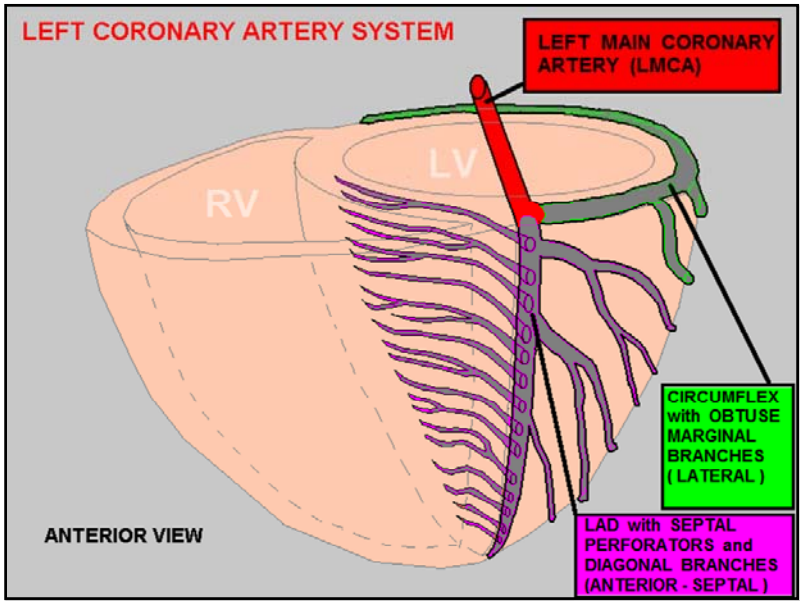

cutaway view of the
CIRCUMFLEX ARTERY (CX) DISTRIBUTION
SUPPLIES 20 - 30 % of the LV MUSCLE MASS
RIGHT DOMINANT SYSTEM
POSTERIOR WALL
RV
SEPTAL WALL
LV
LATERAL WALL
ANTERIOR WALL
CIRCUMFLEX ARTERY (CX)
RIGHT CORONARY ARTERY (RCA)
LEFT MAIN CORONARY ARTERY (LMCA)
LEFT ANTERIOR DESCENDING (LAD) ARTERY (below LMCA)


HELPFUL HINT . . . MEMORIZE THIS !
CIRCUMFLEX ARTERY (CX)
RIGHT DOMINANT SYSTEMS
LEFT ATRIUM
SINUS NODE (45% of the population)
LEFT VENTRICLE: 20 - 30 % of muscle mass
- LATERAL WALL
- up to 1/2 of POSTERIOR WALL


Leads I & AVL view the ANTERIOR-LATERAL JUNCTION
I
AVR
V1
V4
II
AVL
V2
V5
III
AVF
V3
V6

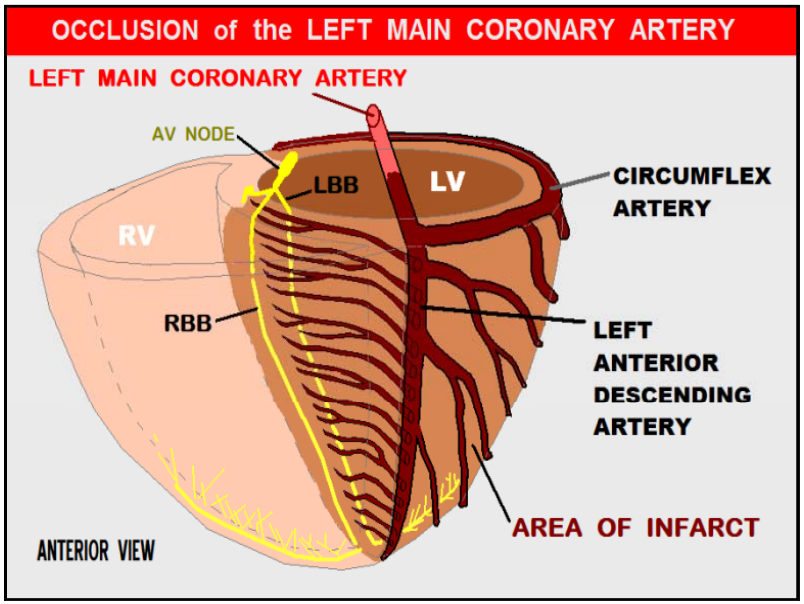

Leads I & AVL view the ANTERIOR-LATERAL JUNCTION
OCCLUSION of OBTUSE MARGINAL ARTERY
LEFT MAIN CORONARY ARTERY
AV NODE
LBB
RV
LV
RBB
CIRUMFLEX ARTERY
1st OBTUSE MARGINAL ARTERY SUPPLYING AREA VIEWED BY LEADS I and aVL ORIGINATES FROM CIRCUMFLEX ARTERY.
AREA OF EKG VIEWED BY LEADS I and aVL
ANTERIOR VIEW
LEFT ANTERIOR DESCENDING ARTERY
OCCLUSION of RAMUS ARTERY
RAMUS ARTERY SUPPLYING AREA VIEWED BY LEADS I and aVL ORIGINATES FROM LEFT MAIN CORONARY ARTERY
OCCLUSION of DIAGONAL ARTERY
1st DIAGONAL ARTERY SUPPLYING AREA VIEWED BY LEADS I and aVL ORIGINATES FROM LEFT ANTERIOR DESCENDING ARTERY

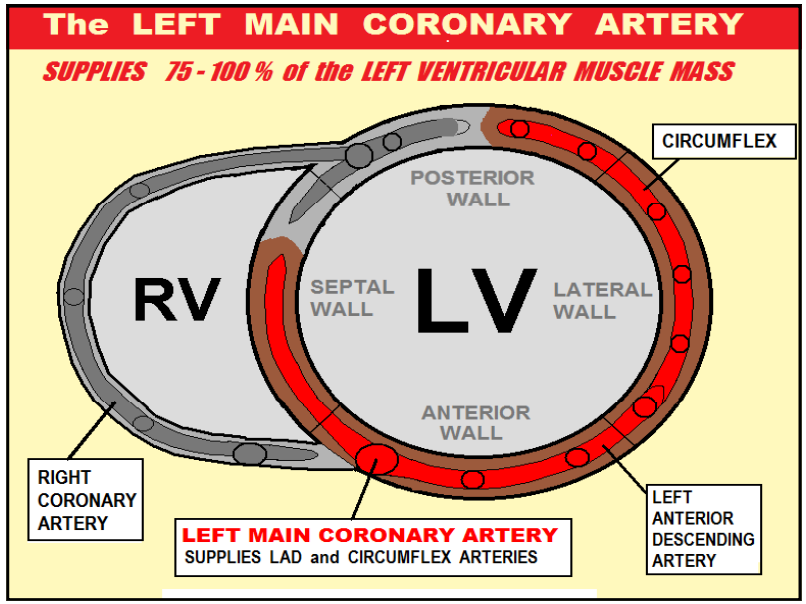

Lead AVR Views the BASILAR SEPTUM (region of the Bundle of His)
I
AVR
V1
V4
II
AVL
V2
V5
III
AVF
V3
V6
The LEFT MAIN CORONARY ARTERY provides the blood supply to the BASILAR SEPTUM.



When LEAD AVR shows ST Elevation:

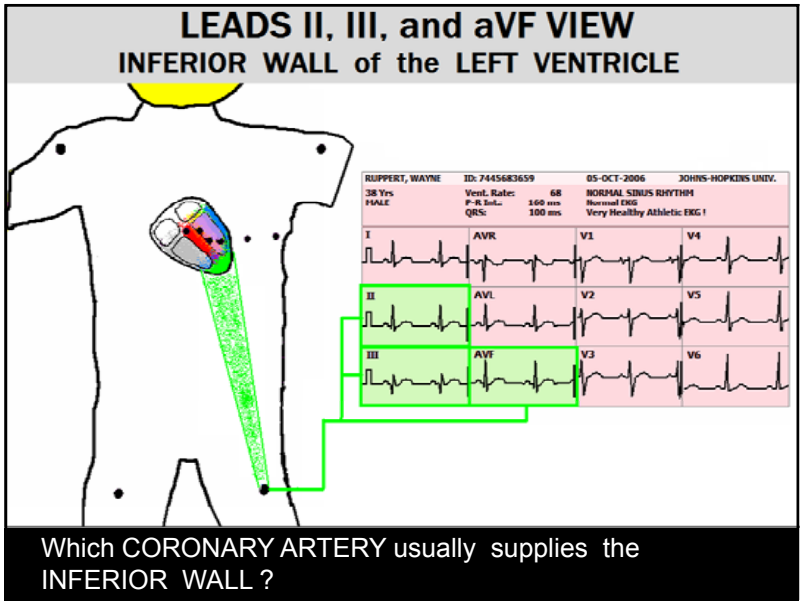
- **STEMI:** consider occlusion of the Left Main Coronary Artery.

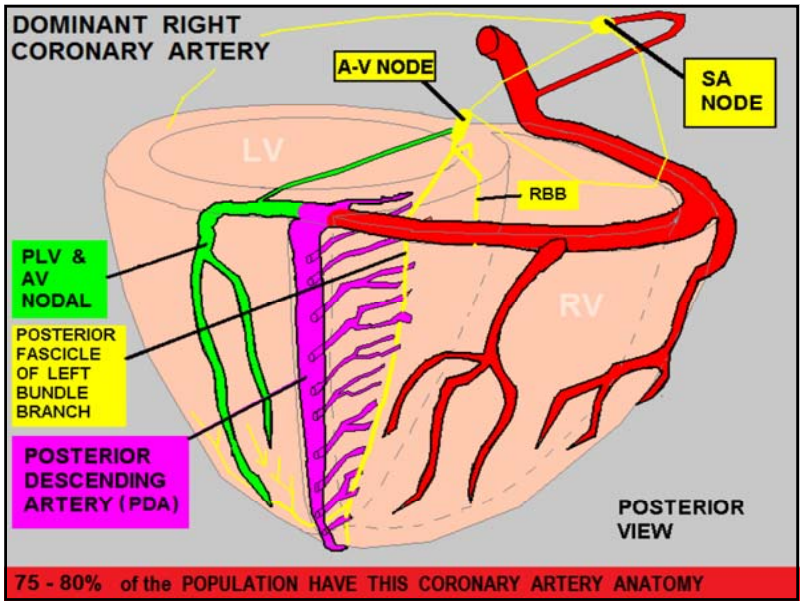


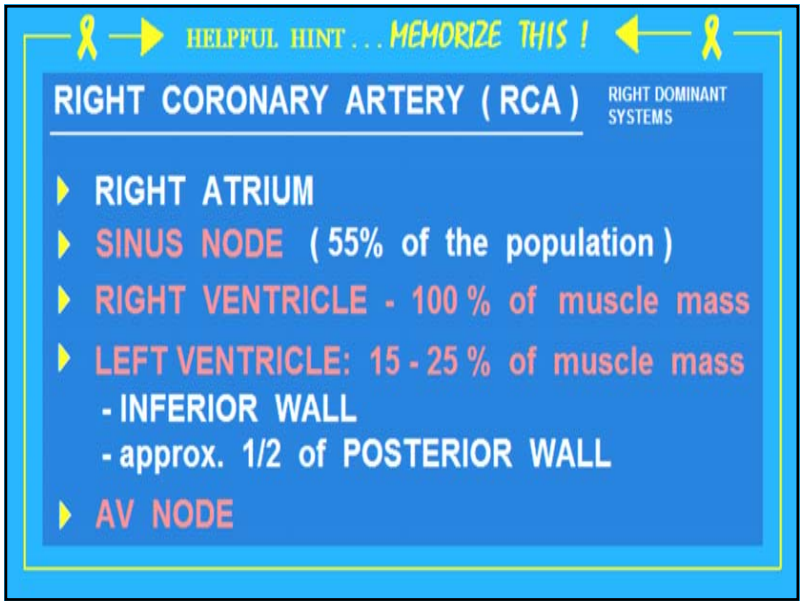


When LEAD AVR shows ST Elevation:

- **STEMI:** consider occlusion of the Left Main Coronary Artery.
- **NSTEMI** and **Unstable Angina** consider LMCA Occlusion – or TRIPLE VESSEL DISEASE







Because the RCA feeds the INFERIOR WALL in 75-80% of the population, AND it also feeds the RIGHT VENTRICLE, in every case of INFERIOR WALL MI, there is a high probability that RIGHT VENTRICULAR MI is ALSO present ! ! ! . . . . . .

IN ***EVERY*** CASE of

INFERIOR WALL STEMI

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

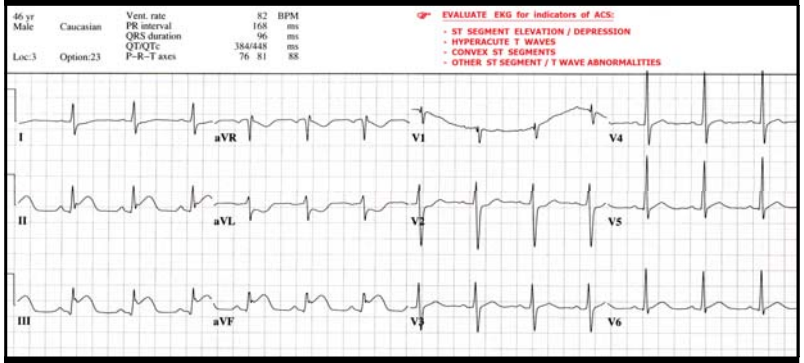
- NITROGLYCERIN
- Dieuretics

NITROGLYCERIN is a CLASS III CONTRINDICATION in RIGHT VENTRICULAR MI ! !*

It *WILL* precipitate PROFOUND HYPOTENSION !

*** A.H.A. ACLS 2010**

So when you see INFERIOR WALL STEMI . . . . .



A standard

12 LEAD EKG

Does NOT show the

RIGHT VENTRICLE

To see the

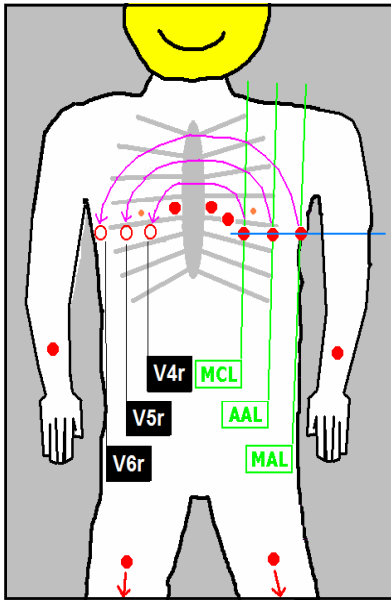
RIGHT VENTRICLE . . .

. . . such as in cases of

INFERIOR WALL M.I.

☞ You must do a

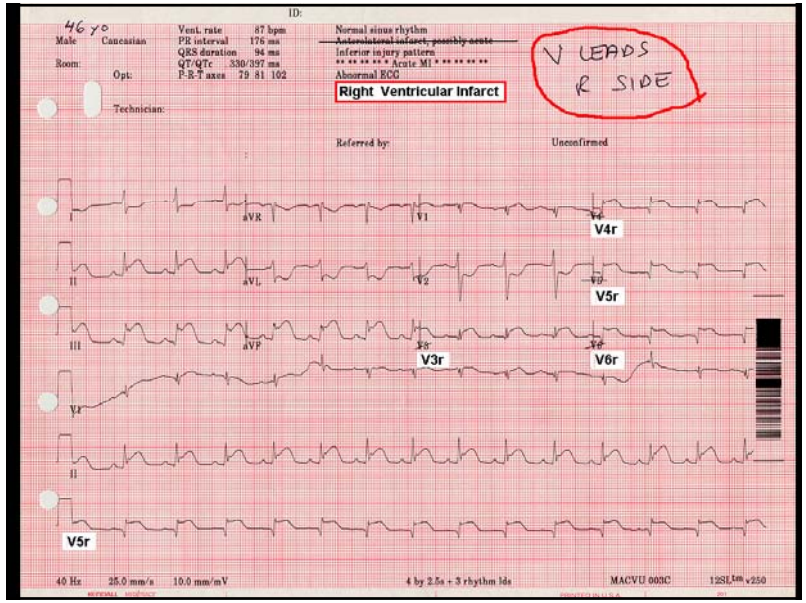
RIGHT - SIDED EKG ! !

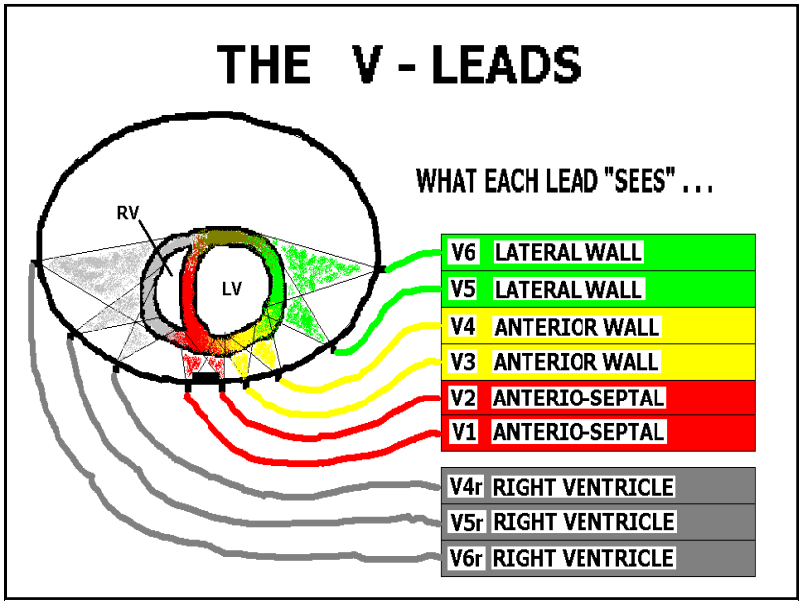


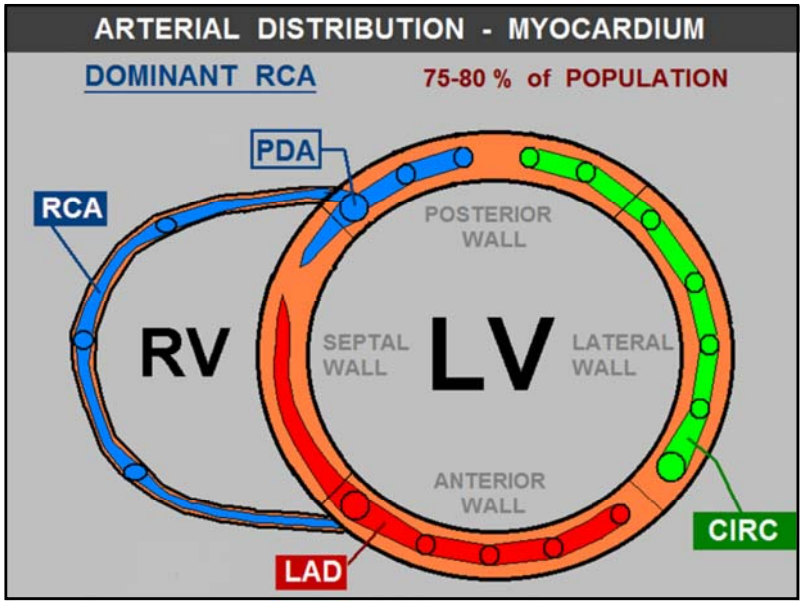
To do a
RIGHT - SIDED EKG . .

MOVE leads
V4, V5, and V6

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







INFERIOR WALL STEMI --- summary:

75-80% caused by RCA OCCLUSION, *think:*
- minimal LV Pump failure
- Right Ventricular MI -- EXTREME sensitivity to NITRATES :

10-15% caused by CIRCUMFLEX OCCLUSION, *think:*
- PROFOUND PUMP FAILURE
- CARDIOGENIC SHOCK
- PULMONARY EDEMA
- CARDIAC ARREST

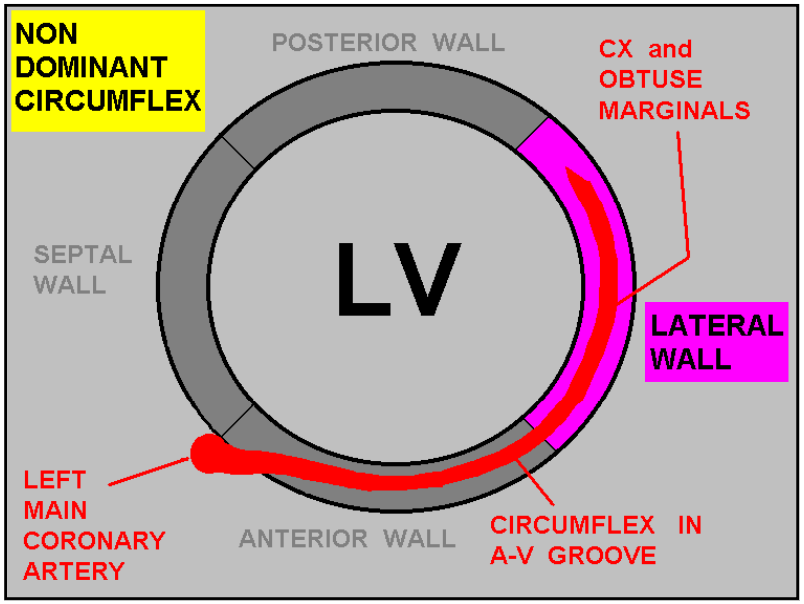
CIRCUMFLEX ARTERY (CX)

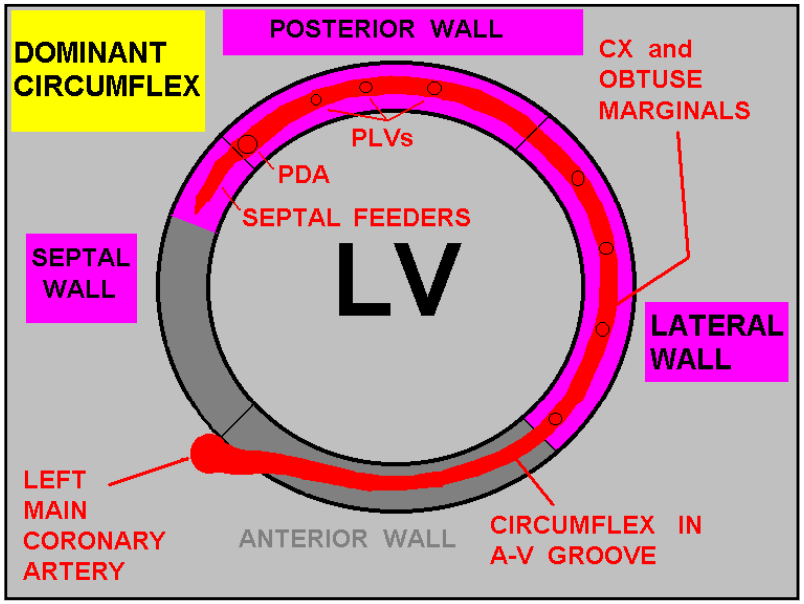
- NON-DOMINANT CX:

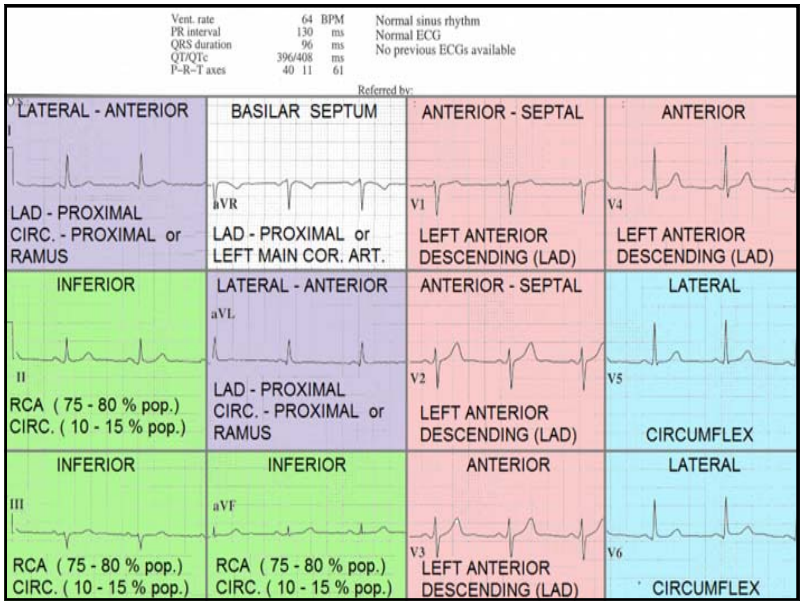
CX = 15 - 30% OF LV MASS

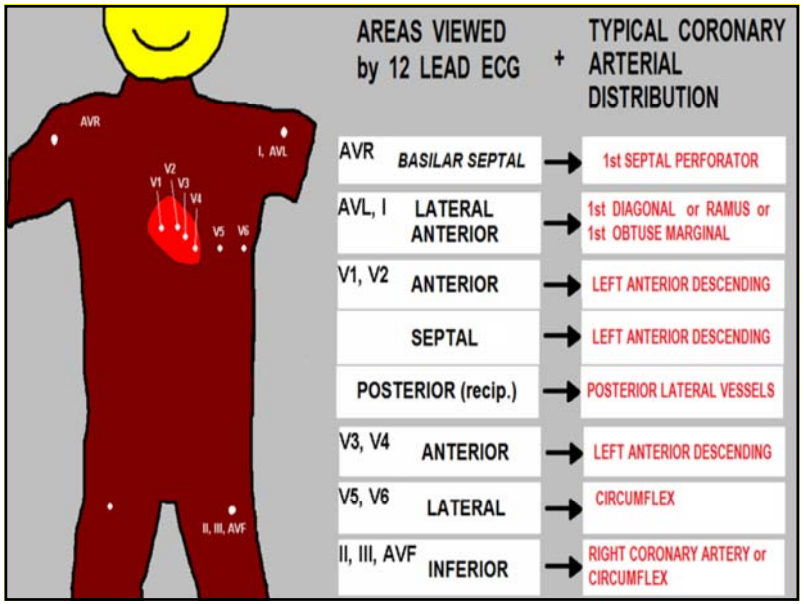
- DOMINANT CX:

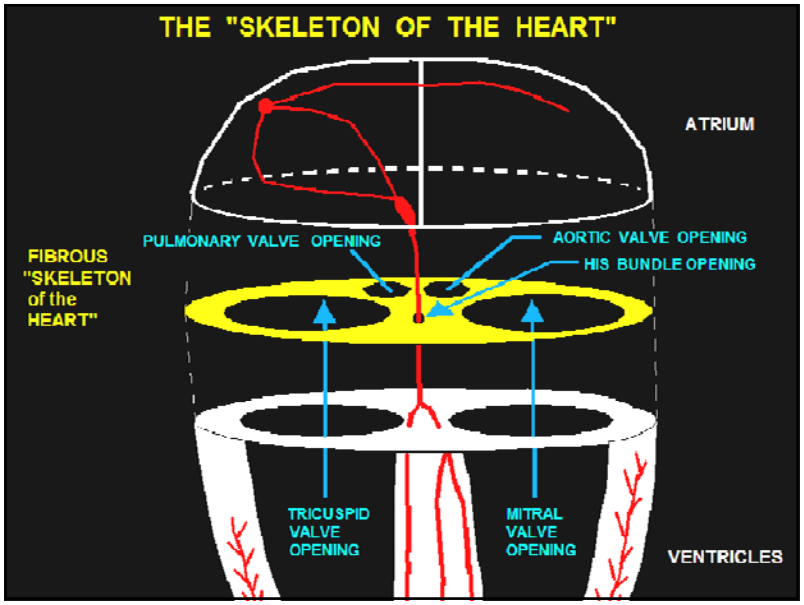
CX = 15 - 30% OF LV MASS
+ PDA = 15 - 25% OF LV MASS
TOTAL 30 - 55% OF LV MASS

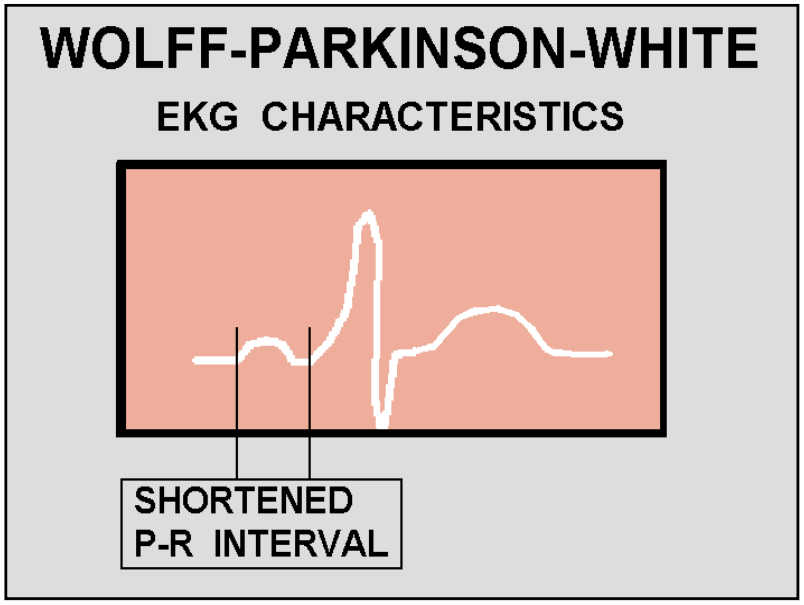

NON DOMINANT CIRCUMFLEX
POSTERIOR WALL
CX and OBTUSE MARGINALS
SEPTAL WALL
LV
LATERAL WALL
LEFT MAIN CORONARY ARTERY
ANTERIOR WALL
CIRCUMFLEX IN A-V GROOVE

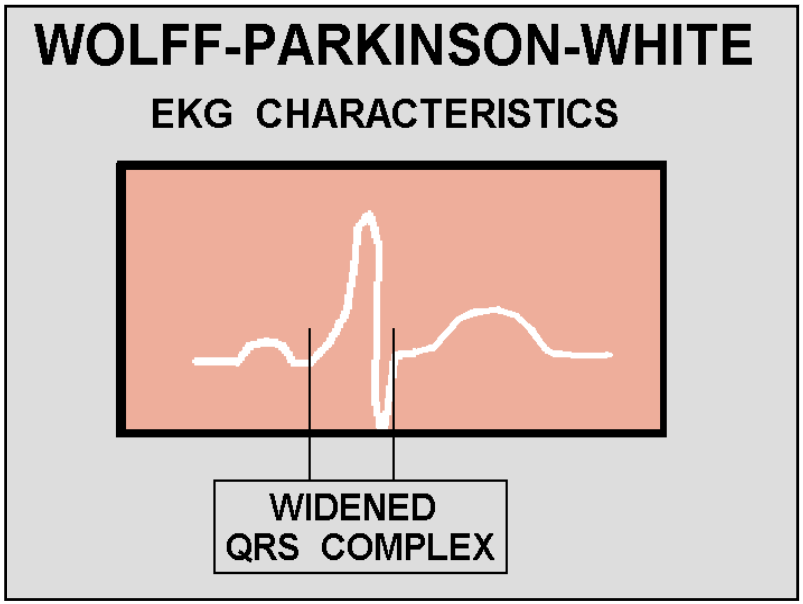

DOMINANT CIRCUMFLEX
POSTERIOR WALL
CX and OBTUSE MARGINALS
PLVs
PDA
SEPTAL FEEDERS
SEPTAL WALL
LV
LATERAL WALL
LEFT MAIN CORONARY ARTERY
ANTERIOR WALL
CIRCUMFLEX IN A-V GROOVE

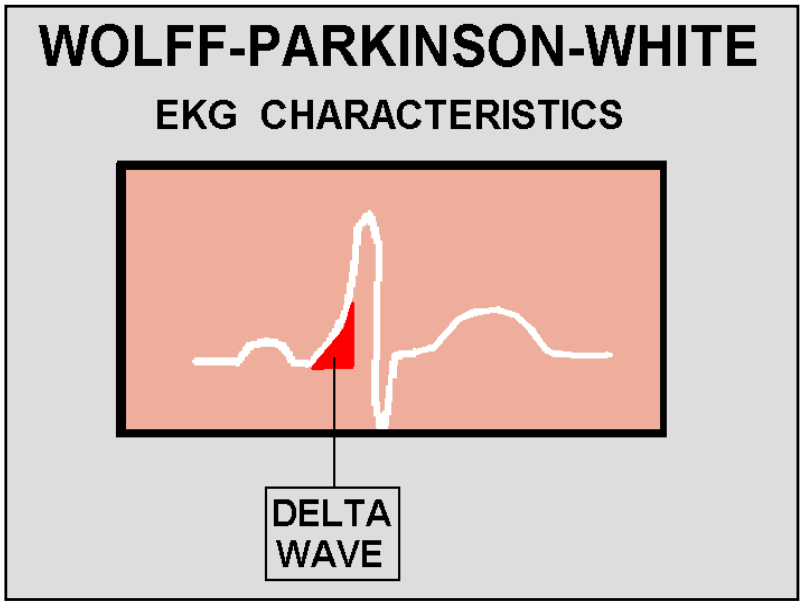

Vent. rate 64 BPM
PR interval 130 ms
QRS duration 96 ms
QT/QTc 396/408 ms
P-R-T axes 40 11 61
Normal sinus rhythm
Normal ECG
No previous ECGs available
Referred by:
LATERAL - ANTERIOR
LAD - PROXIMAL CIRC. - PROXIMAL or RAMUS
BASILAR SEPTUM
LAD - PROXIMAL or LEFT MAIN COR. ART.
ANTERIOR - SEPTAL
LEFT ANTERIOR DESCENDING (LAD)
ANTERIOR
LEFT ANTERIOR DESCENDING (LAD)
INFERIOR
RCA (75 - 80 % pop.) CIRC. (10 - 15 % pop.)
LATERAL - ANTERIOR
LAD - PROXIMAL CIRC. - PROXIMAL or RAMUS
ANTERIOR - SEPTAL
LEFT ANTERIOR DESCENDING (LAD)
LATERAL
CIRCUMFLEX
INFERIOR
RCA (75 - 80 % pop.) CIRC. (10 - 15 % pop.)
INFERIOR
RCA (75 - 80 % pop.) CIRC. (10 - 15 % pop.)
ANTERIOR
LEFT ANTERIOR DESCENDING (LAD)
LATERAL
CIRCUMFLEX

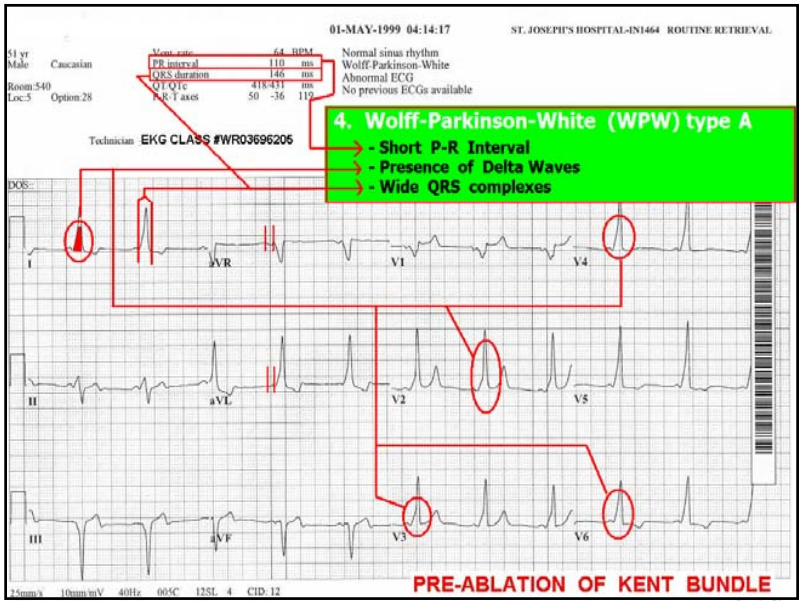

AREAS VIEWED by 12 LEAD ECG
+
TYPICAL CORONARY ARTERIAL DISTRIBUTION
AVR
I, AVL
V1 V2 V3 V4 V5 V6
II, III, AVF
AVR BASILAR SEPTAL → 1st SEPTAL PERFORATOR
AVL, I LATERAL ANTERIOR → 1st DIAGONAL or RAMUS or 1st OBTUSE MARGINAL
V1, V2 ANTERIOR → LEFT ANTERIOR DESCENDING
SEPTAL → LEFT ANTERIOR DESCENDING
POSTERIOR (recip.) → POSTERIOR LATERAL VESSELS
V3, V4 ANTERIOR → LEFT ANTERIOR DESCENDING
V5, V6 LATERAL → CIRCUMFLEX
II, III, AVF INFERIOR → RIGHT CORONARY ARTERY or CIRCUMFLEX

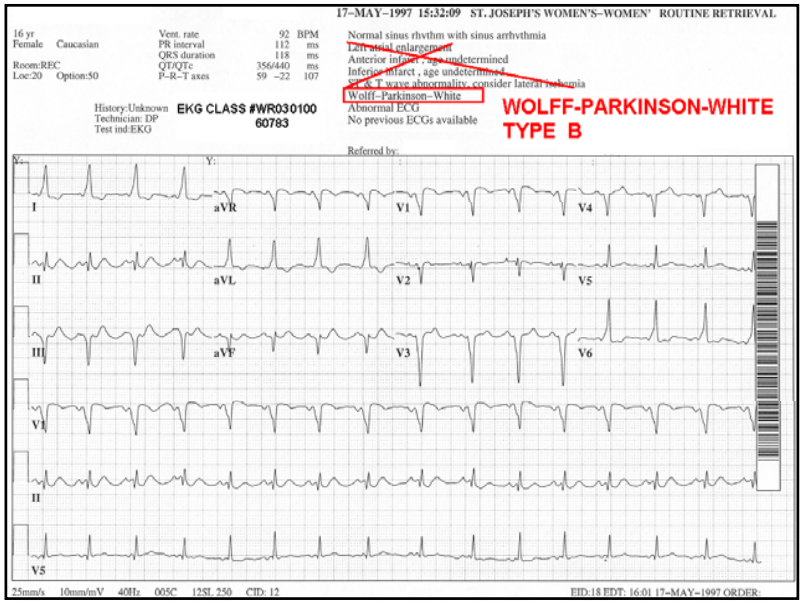

THE "SKELETON OF THE HEART"
ATRIUM
PULMONARY VALVE OPENING
AORTIC VALVE OPENING
HIS BUNDLE OPENING
FIBROUS "SKELETON of the HEART"
TRICUSPID VALVE OPENING
MITRAL VALVE OPENING
VENTRICLES

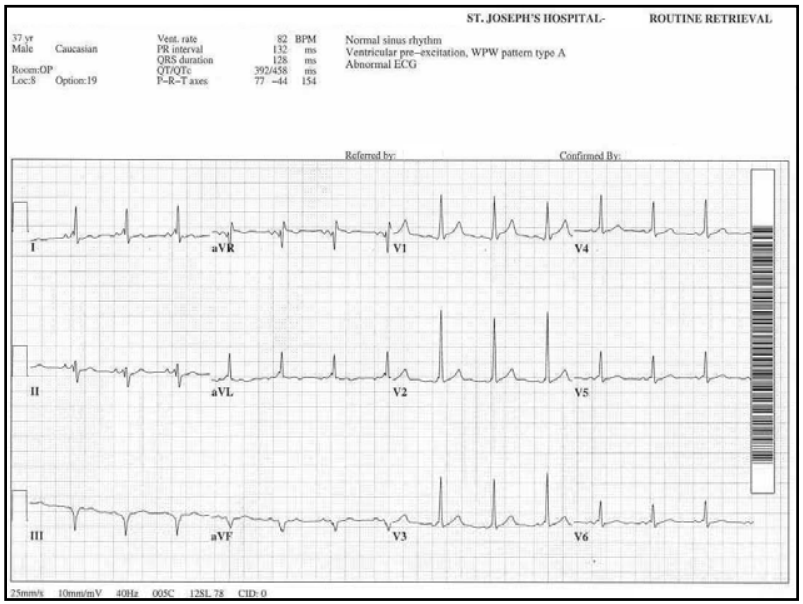

WOLFF-PARKINSON-WHITE
EKG CHARACTERISTICS
SHORTENED P-R INTERVAL


WOLFF-PARKINSON-WHITE
EKG CHARACTERISTICS
WIDENED QRS COMPLEX


WOLFF-PARKINSON-WHITE
EKG CHARACTERISTICS
DELTA WAVE







37 y/o male

Chief Complaint: Lightheadedness, Palpitations, Shortness of Breath

HPI: Sudden onset of above symptoms approx. 1 hour ago

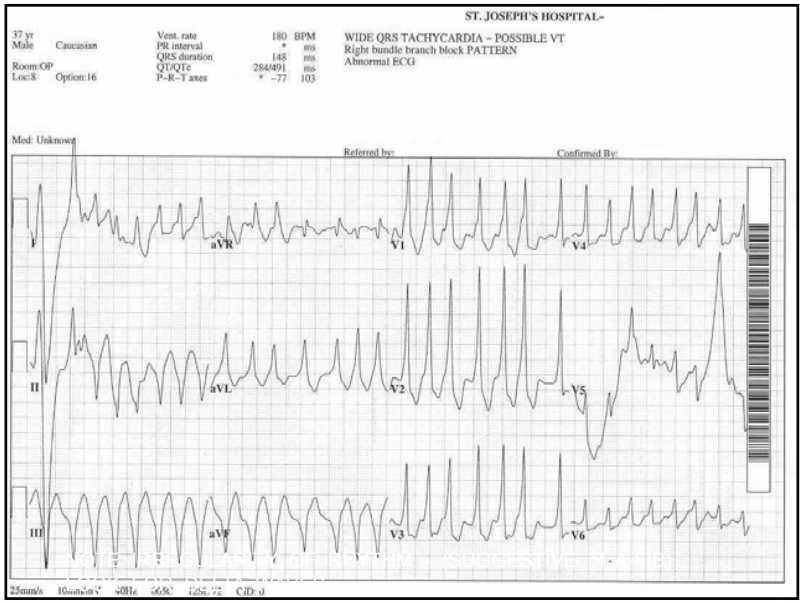
PMH: HTN (non-compliant)

37 y/o male

PE: Alert, oriented, restless, cool, pale, dry skin. PERL, No JVD, Lungs clear. Abd soft non tender, Extremities: WNL, no edema

Meds: None, NKDA

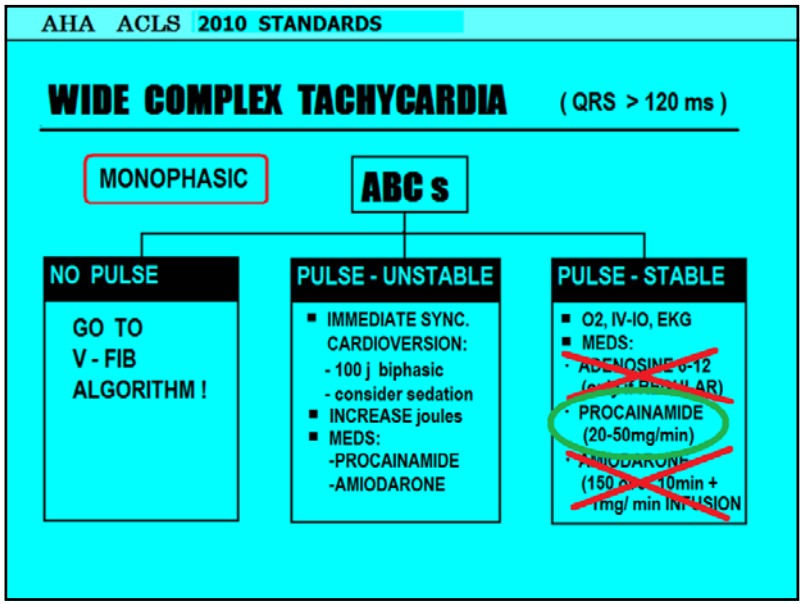
VS: BP 106/50, P 180, R 26, SAO2 93%

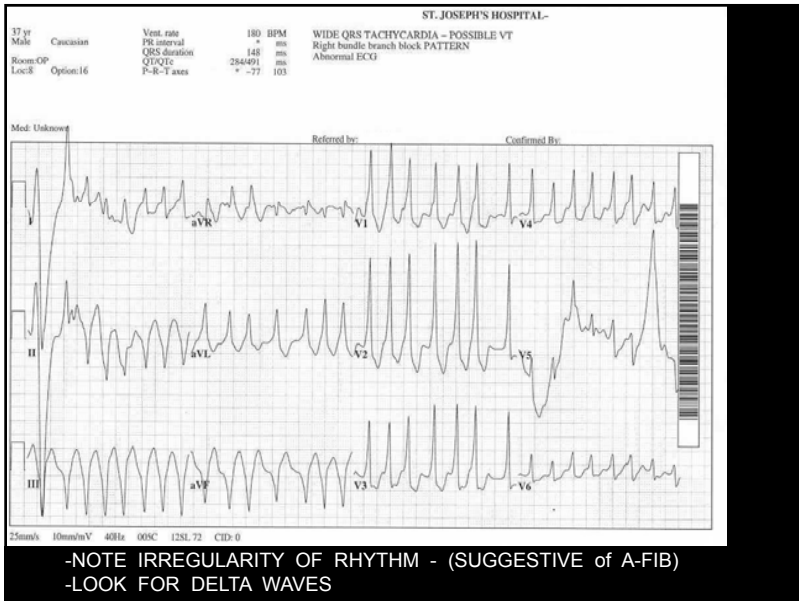


Physician correctly identified Atrial Fibrillation with Rapid Ventricular Response.

However did NOT identify the Wolff-Parkinson-White component.

Patient was given Diltiazem – promptly converted to - ***VENTRICULAR FIBRILLATION.***





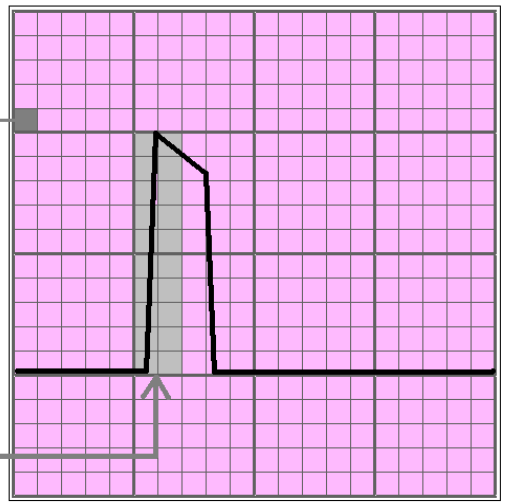
-NOTE IRREGULARITY OF RHYTHM - (SUGGESTIVE of A-FIB)
-LOOK FOR DELTA WAVES

CHARACTERISTICS of W-P-W with Afib & RVR:
- **WIDE COMPLEX TACHYCARDIA**
- **IRREGULARLY IRREGULAR R – R INTERVALS !!**

NO AV NODAL BLOCKERS (e.g. ADENOSINE, CALCIUM CHANNEL BLOCKERS) FOR WIDE COMPLEX TACHYCARDIAS THAT COULD BE ATRIAL FIBRILLATION with Pre-Excitation (W-P-W)

ECG PAPER - THE VERTICAL AXIS:

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



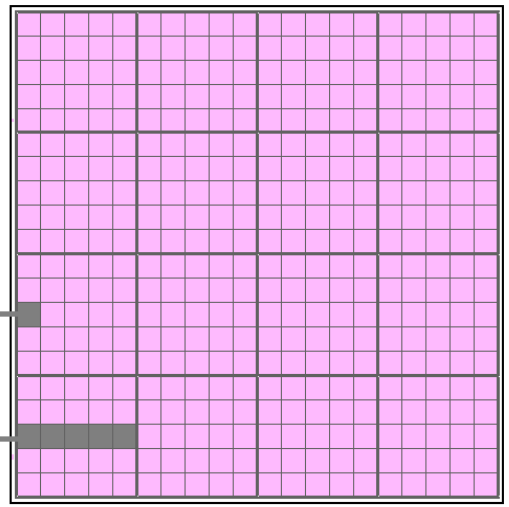
ECG PAPER - THE HORIZONTAL AXIS:

THE HORIZONTAL AXIS REPRESENTS TIME . . .

STANDARD SPEED FOR RECORDING ADULT EKGs = 25 mm / SECOND

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

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

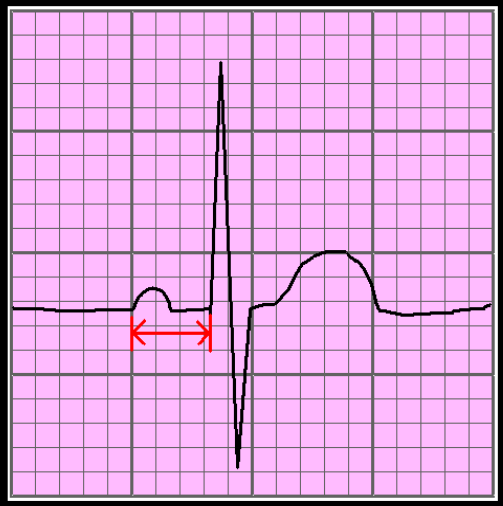


NORMAL P-R INTERVAL

.12 - .20 SEC

or

120 - 200 mSEC



P - R INTERVAL TOO SHORT . . .

LESS THAN 120 mSEC

THINK:

- ECTOPIC ATRIAL ACTIVITY
- PRE-EXCITATION (WPW)
- JUNCTIONAL (nearly on top of QRS, possibly inverted)

P - R INTERVAL TOO LONG

GREATER THAN 200 mSEC

THINK:

- HEART BLOCK

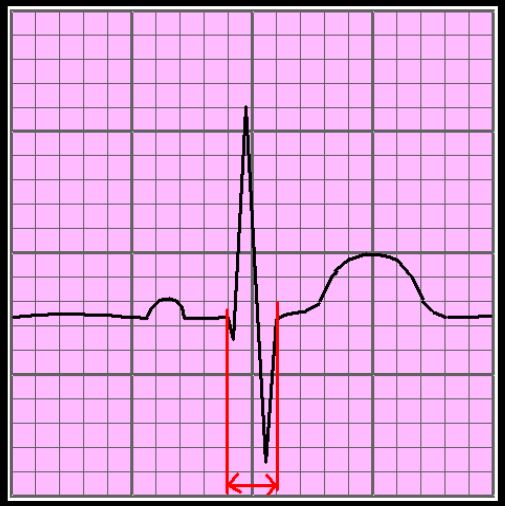
QRS INTERVAL

LESS THAN

.12

OR

120 mSEC



QRS COMPLEX TOO WIDE

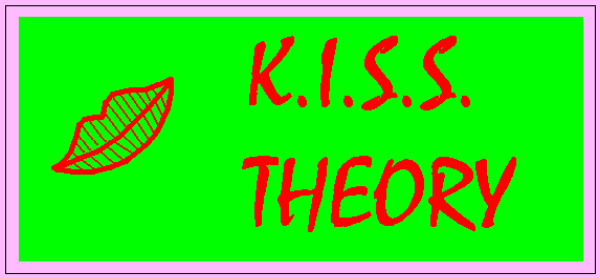
WIDER THAN 120 mSEC

THINK:

- BUNDLE BRANCH BLOCK
- VENTRICULAR COMPEX (ES)
- PACED RHYTHM
- L VENTRICULAR HYPERTROPHY
- ELECTROLYTE IMBAL. (↑K+ ↓Ca++)
- DELTA WAVE (PRE-EXCITATION)

THE QRS COMPLEX

DIAGNOSING
BUNDLE BRANCH BLOCK



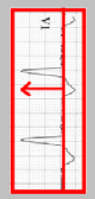
DIAGNOSING BUNDLE BRANCH BLOCKS in LEAD V1

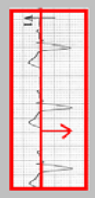
*** The "York Hospital" Method:**

1. ***ROTATE*** your rhythm strip (of LEAD V1) CLOCKWISE

2. ***DIAGNOSE*** -- The side of the ISOELECTRIC LINE that the bulk of the QRS complex rests -- is the side of the heart with the BUNDLE BRANCH BLOCK !

LBBB !

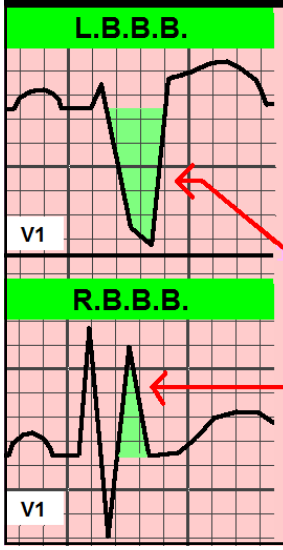




RBBB !

* I do not know whom is responsible for developing this "fine pearl" of cardiology wisdom, but the first time I ever heard of it -- after teaching 12 Lead EKG for several years -- was while teaching EKG at York Hospital in York, Pennsylvania. I promised the room full of nurses -- who were laughing, by this time -- that I would credit them for enlightening me on this rather useful and usually accurate technique !

DIAGNOSING BUNDLE BRANCH BLOCK



USING LEAD V1

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

NEGATIVE = LEFT BUNDLE BRANCH BLOCK

POSITIVE = RIGHT BUNDLE BRANCH BLOCK

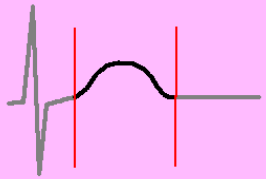
SOME CAUSES OF RIGHT BUNDLE BRANCH BLOCK (RBBB)

- CONGENITAL VARIATION (IN HEALTHY HEART)
- CONDUCTION SYSTEM DISEASE
- OLD ANT./SEPTAL MI (NECROSIS TO RBB)
- PREVIOUS C.A.B.G. (RBB CUT DURING SURGERY)
- SEVERE R.V.H.
- ACUTE PULMONARY EMBOLUS
- BRUGADA SYNDROME

SOME CAUSES OF LEFT BUNDLE BRANCH BLOCK (LBBB)

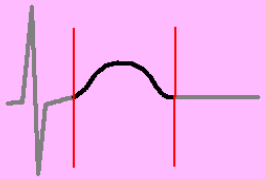
- CONDUCTION SYSTEM DISEASE
- OLD ANT./SEPTAL MI (NECROSIS TO LBB)
- CARDIOMYOPATHY
- SEVERE L.V.H.
- ACUTE MYOCARDITIS

THE T WAVE



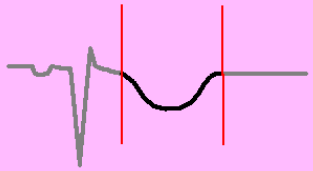
- SHOULD BE A "NICE," ROUNDED, CONVEX SHAPE
- SHOULD BE SYMMETRICAL

THE T WAVE



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

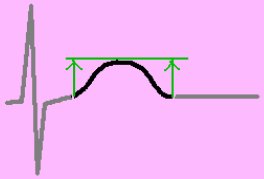
THE T WAVE



LEAD AVR

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

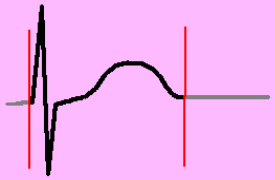
THE T WAVE



AMPLITUDE GUIDELINES:

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

THE Q - T INTERVAL



- BEGINNING OF QRS COMPLEX TO THE END OF THE T WAVE
- NORMAL VALUES VARY BASED ON HEART RATE
- SEVERAL WAYS TO DETERMINE NORMAL LIMITS

THE *QTc INTERVAL

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

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

Annals of Internal Medicine, 1988 109:905.

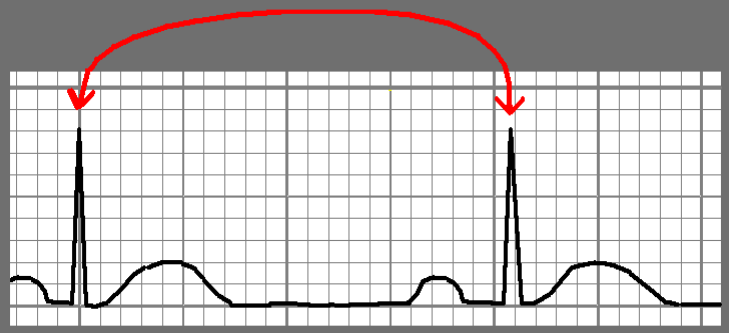
QT CORRECTION FORMULAS:

Bazett's	QTc=QT/√ RR
Fredericia	QTc=QT/(RR)1/3
Framingham	QTc=QT+0.154(1-RR)
Rautaharju	QTc=656/(1+HR/100)

DETERMINING Q-T INTERVAL LIMITS

THE "QUICK PEEK" METHOD

(for Heart Rates 60 - 100)

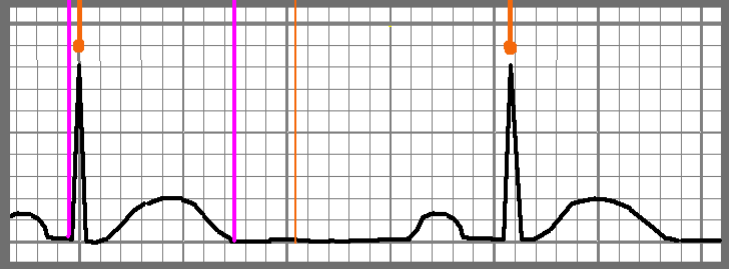


The Q - T Interval should be LESS THAN 1/2 the

R - R Interval

OK | TOO LONG

GOOD



ECG Indicators of Long QT Syndrome:

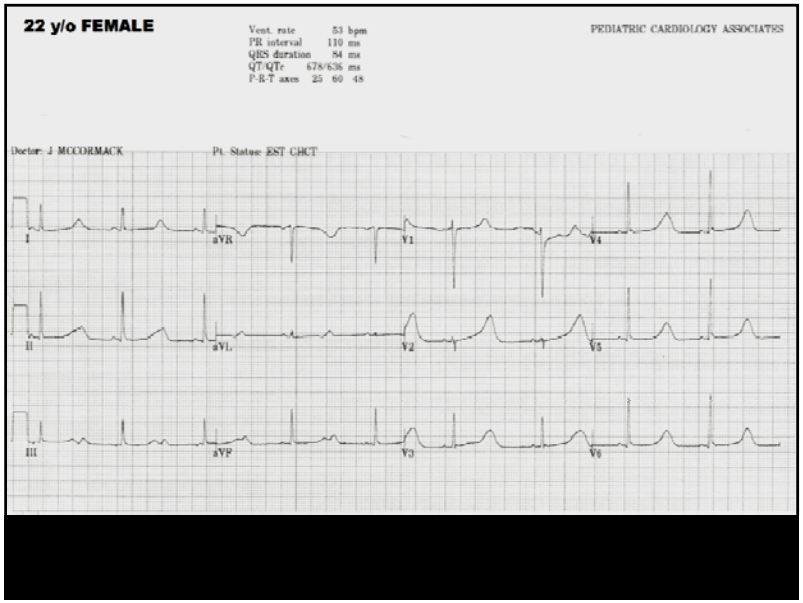
- **QTc 460ms or longer in females***
- **QTc 450ms or longer in males***
- **T wave alterans**
- **U waves >100% of the T wave**
- **U waves merged with T waves**
- **U waves >0.1mv (1mm on standard calibrated ECG)**

*P. Rautaharju, et al, "Standardization and Interpretation of the ECG, Part IV"
JACC2009;53, no. 11:982-991

☞ **WHEN LQTS IS SUSPECTED, TAKE THE FOLLOWING PRECAUTIONS . . . .**

You get dispatched to a grocery store for "seizures."

You find a 22 year old female alert and oriented to person, place and time. Witnesses describe a brief grand-mal seizure, then woke up and was alert. She has a history of seizures, and is on Cerebyx.



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

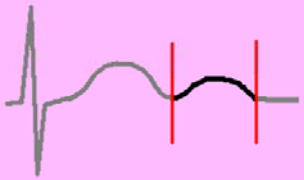
If patient has a PROLONGED Q-T INTERVAL, AVOID DRUGS THAT LENGTHEN THE Q-T.

Such drugs include:

-Amiodarone	-Ritalin
-Procainamide	-Benadryl
-Levaquin	-Haloperidol
-Erythromycin	-Thorazine
-Norpace	-Propulcid
-Tequin	..... AND MANY MORE .....

www.torsades.org , & www.azcert.org

THE U WAVE

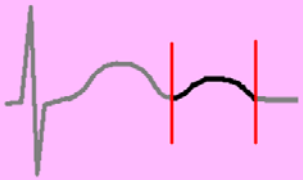


- SEEN INFREQUENTLY

U WAVES PANIC VALUES (CONSIDERED INDICATOR OF LONG QT SYNDROME) WHEN:

- 100% or more SIZE OF T WAVE
- MERGED WITH T WAVE
- MORE THAN 1mm IN HEIGHT

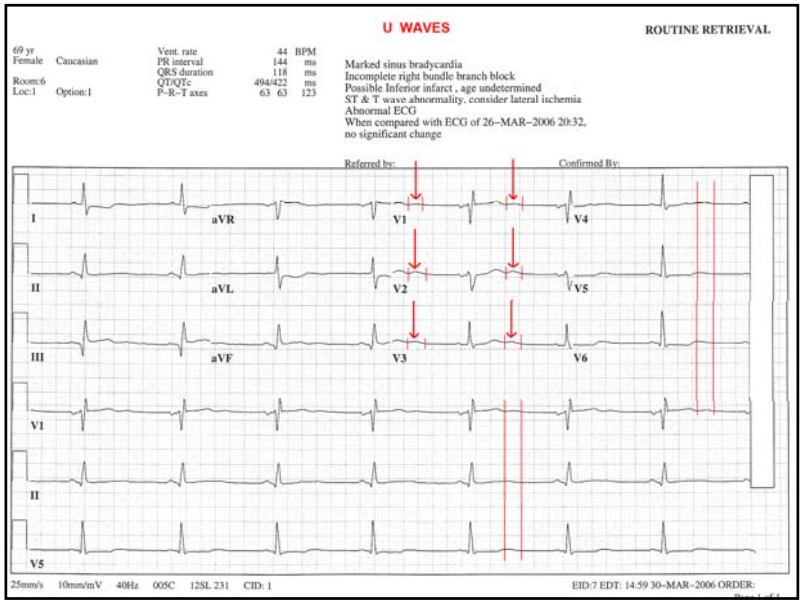
THE U WAVE



- SEEN INFREQUENTLY

- MOST VISIBLE IN LEADS V2 & V3

- OFTEN NOT PRESENT IN LEADS II, III, AVF



When ECG Indicators of Long QT Synrome are present:

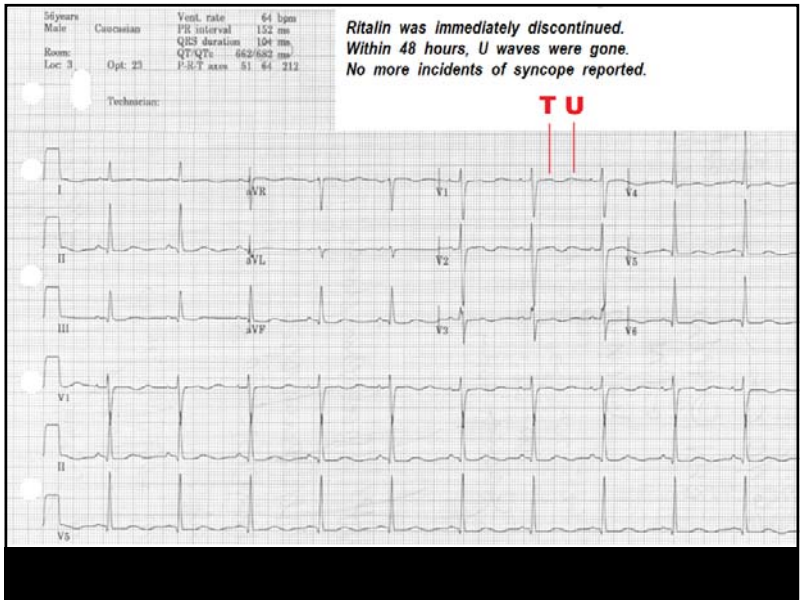
- Obtain a thorough patient history, to rule out incidence of syncope and family history of sudden death/ near sudden death.
- Evaluate patient's meds list for meds that prolong the QT Interval.
- Rule out hypothermia
- Rule out CVA
- Evaluate the patient's electrolyte levels, and
- MONITOR PATIENT'S ECG FOR RUNS OF TORSADES
- Consider "expert consult" (electrophysiologist) to rule out LQTS

Q: What is the ideal medication to treat Torsades?

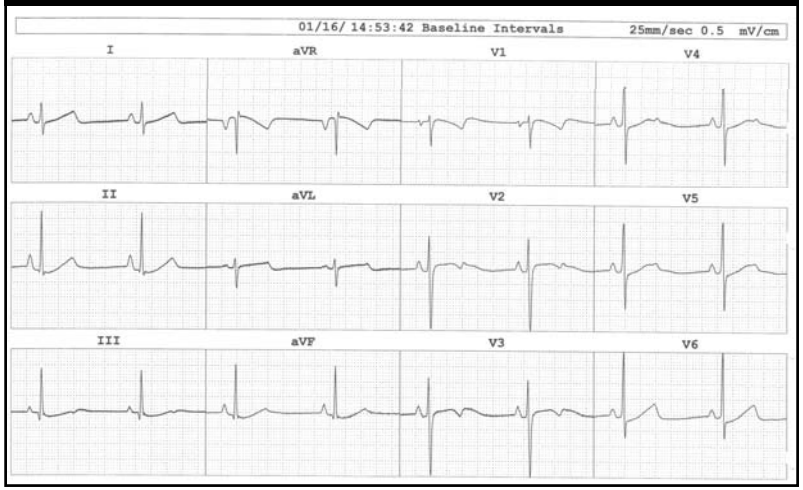
A: Magnesium Sulfate, 1 – 2 grams over 5 – 60 minutes (AHA ACLS)

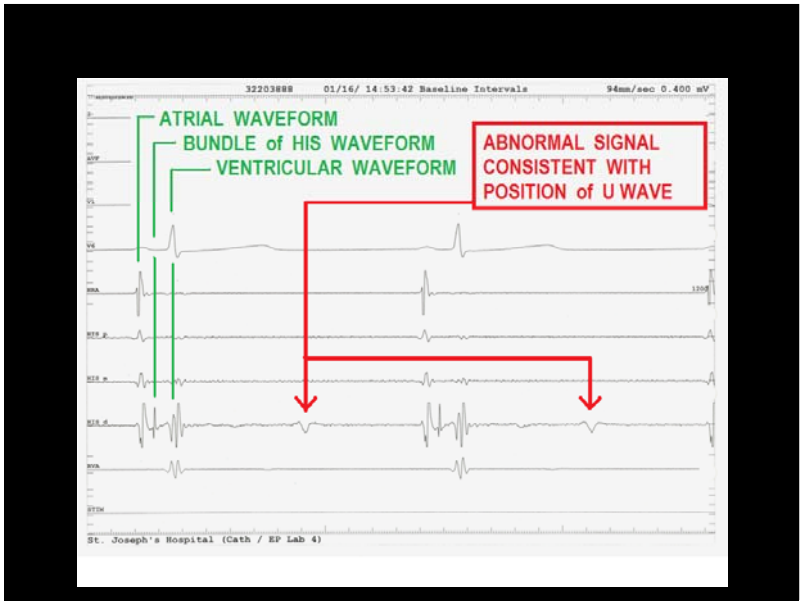
HERNANDO COUNTY FIRE RESCUE PROTOCOL:

Torsades
Mag Sulphate (9.21)
2 gm IV over 10 minuntes



15 year old male , suffered sudden cardiac arrest. Successful out-of-hospital resuscitation with CPR / AED. His ECG is shown below:

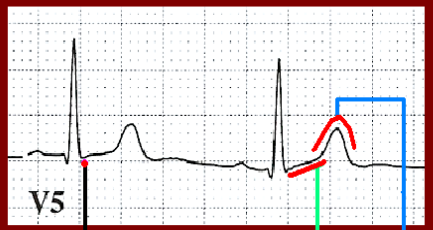




ABSOLUTELY NO DRUGS THAT PROLONG THE Q-T INTERVAL ! !

NORMAL ST - T WAVES

- WHEN QRS WIDTH IS NORMAL (< 120 ms)

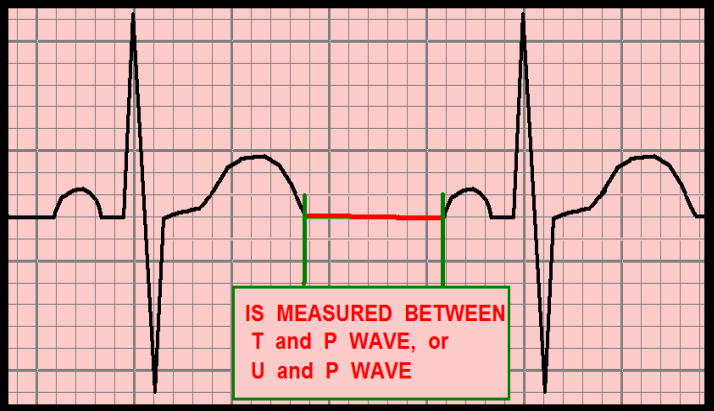


ASSESS:

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

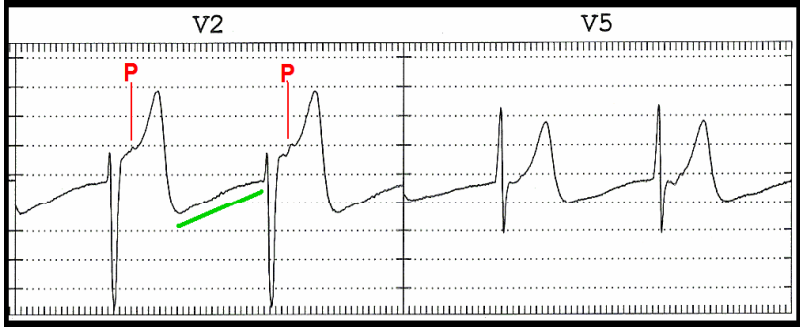
☞ *in EVERY LEAD EXCEPT aVR ! !*

THE ISOELECTRIC LINE



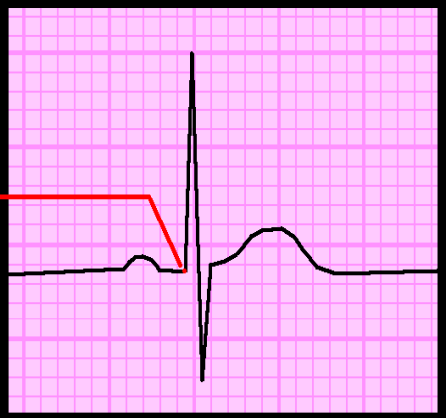
THE ISOELECTRIC LINE

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



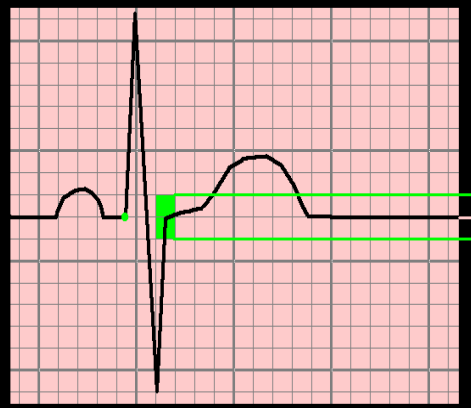
THE P-Q JUNCTION

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



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

THE J POINT SHOULD BE . .

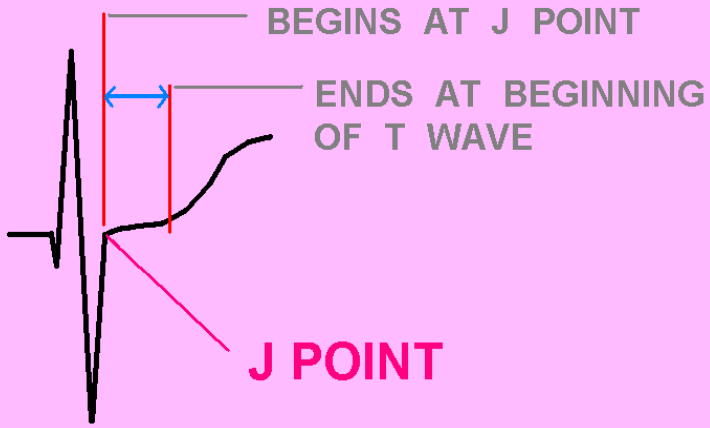


WITHIN 1 mm ABOVE

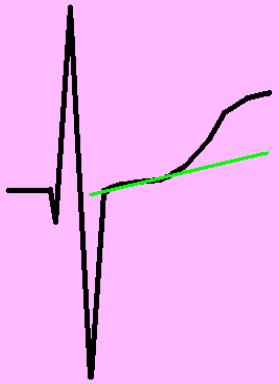
OR

BELOW THE P-Q JUNCTION

THE S-T SEGMENT

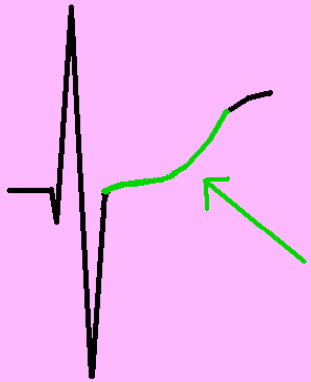


THE S-T SEGMENT



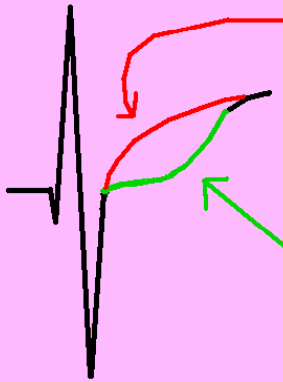
SHOULD HAVE
A "SLIGHT POSITIVE"
INCLINATION

THE S-T SEGMENT



SHOULD BE
"CONCAVE" IN
SHAPE . . .

THE S-T SEGMENT



AS OPPOSED TO
"CONVEX" IN
SHAPE

SHOULD BE
"CONCAVE" IN
SHAPE . . .

CHANGES ASSOCIATED WITH CELLULAR PERFUSION INVOLVING THE:

- QRS
- J point
- ST Segment
- T wave

CARDIAC CELL PERFUSION STATES

NORMAL STATE OF PERFUSION

ARTERIAL BLOCKAGES → NONE SIGNIFICANT
CELLULAR OXYGENATION → NORMAL
CELLULAR METABOLISM → AEROBIC
CELLULAR FUNCTION → NORMAL CONTRACTION

EKG: J POINT ISOELECTRIC, ST SEG "SLIGHT, POSTIVE INCLINATION, T WAVE POSITIVE, UPRIGHT.

ISCHEMIA

ARTERIAL BLOCKAGES → PARTIAL OBSTRUCTION
CELLULAR OXYGENATION → INSUFFICIENT
CELLULAR METABOLISM → AEROBIC
CELLULAR FUNCTION → REDUCED CONTRACTION
PATIENT SYMPTOMS → *POSSIBLE*, WITH EXERTION

EKG: J POINT DEPRESSED, ST SEGMENT VARIES, T WAVE VARIES

INFARCTION

ARTERIAL BLOCKAGES → TOTAL OBSTRUCTION
CELLULAR OXYGENATION → NONE
CELLULAR METABOLISM → ANAEROBIC *CELL BEGINS TO BURN GLYCOGEN RESERVES*
CELLULAR FUNCTION → STOPS CONTRACTING
PATIENT SYMPTOMS → *TYPICAL or ATYPICAL ACS Sx*

EKG - INDICATIVE: J POINT ELEVATES, ST SEGMENT CONVEX, T WAVE POSITIVE, MAY ENLARGE
EKG - RECIPROCAL: J POINT DEPRESSES, ST SEGMENT DOWNSLOPING, T WAVE INVERTED

NECROSIS

ARTERIAL BLOCKAGES → TOTAL OBSTRUCTION
CELLULAR OXYGENATION → NONE
CELLULAR METABOLISM → *CELL DIES WHEN GLYCOGEN RESERVES DEPLETED.*
CELLULAR FUNCTION → NONE. CELL DEAD.
PATIENT SYMPTOMS → *POSS. HYPOTENSION, DEATH*

EKG - INDICATIVE: J POINTS, ST SEGMENTS NORMALIZE; ABNORMAL Q WAVES FORM
EKG - RECIPROCAL: J POINTS, ST SEGMENTS NORMALIZE; ABNORMAL TALL R WAVES FORM

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**

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

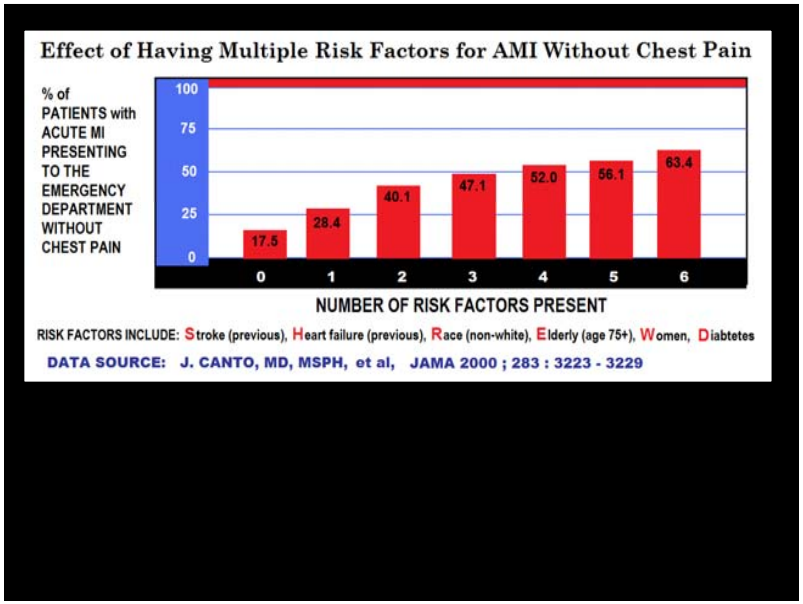
Stroke (previous history of)
Heart failure (previous history of)
Race (non-white)
Elderly (age 75+)
Women
Diabetes mellitus

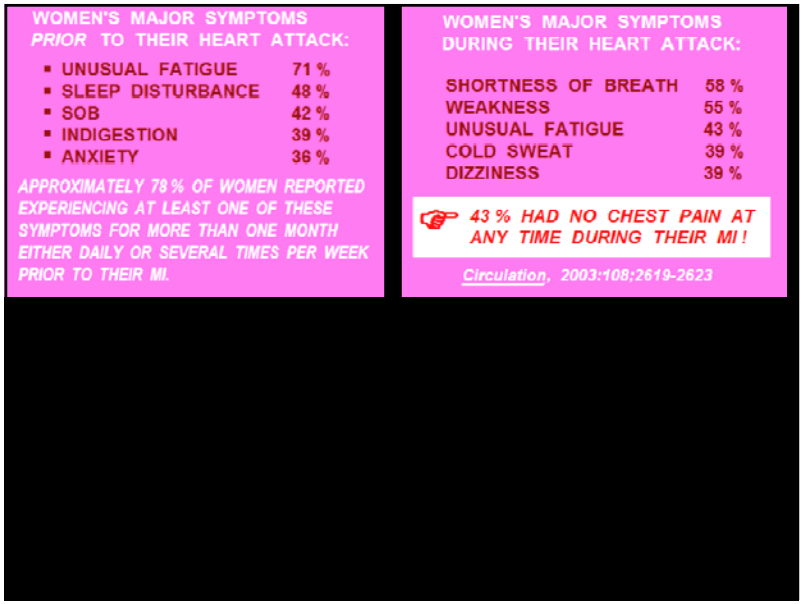
* The information listed in the table to the immediate left resulted from a study conducted by John G. Canto, MD, MSPH, et. al., of the University of Alabama. The study consisted of 434,877 patients diagnosed with AMI between 1994 and 1998 in 1,674 US hospitals. Study results were published in the Journal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

Common atypical complaints associated with AMI without chest pain include:

Malaise (weakness)
Indigestion
Nausea
Dizziness
Syncope
Fatigue
Abdominal pain
Cold sweats
Elevated heart rate
Dsyspnea

BOOK PAGE: 70





ECG EVALUATION for ACS:

STEP 1: EVALUATE *WIDTH of QRS*

BOOK PAGE: 73

IF THE QRS IS TOO WIDE . . . . . .

(GREATER THAN 120 ms)

. . . . IS the QRS morphology:

LEFT BUNDLE BRANCH BLOCK

- OR -

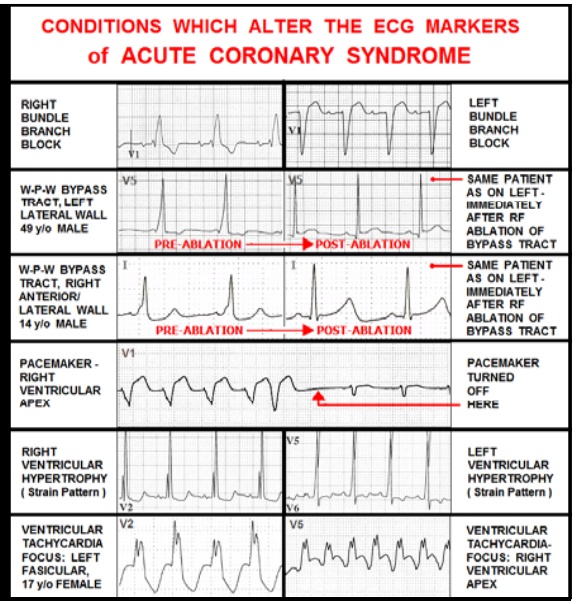
RIGHT BUNDLE BRANCH BLOCK

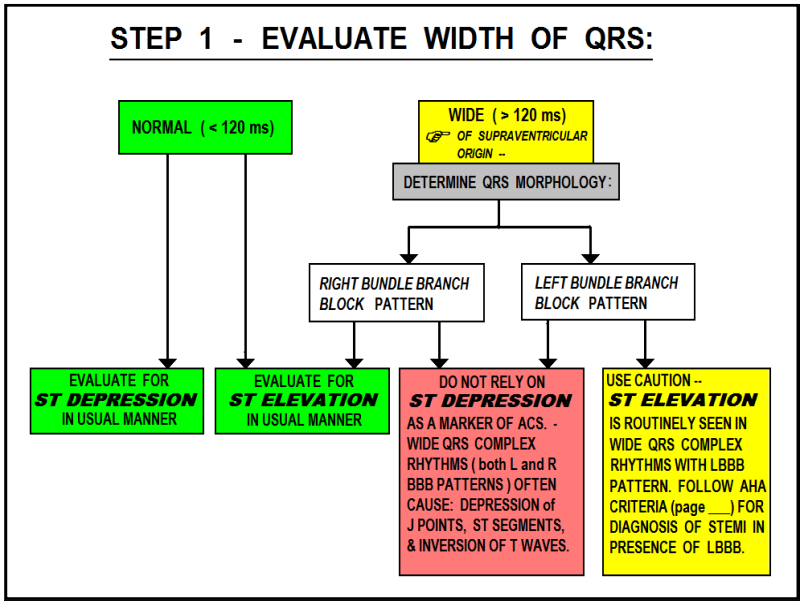
?????

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

Of the ECG . . .

BOOK PAGE: 74





INFARCTION

A.H.A. ACLS GUIDELINES 2000 / 2006

PATIENTS with RIGHT BUNDLE BRANCH BLOCK --

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

A.H.A. ACLS GUIDELINES

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.

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

STEP 2 - EVALUATE the EKG for ACS

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

- J POINTS
- ST SEGMENTS
- T WAVES

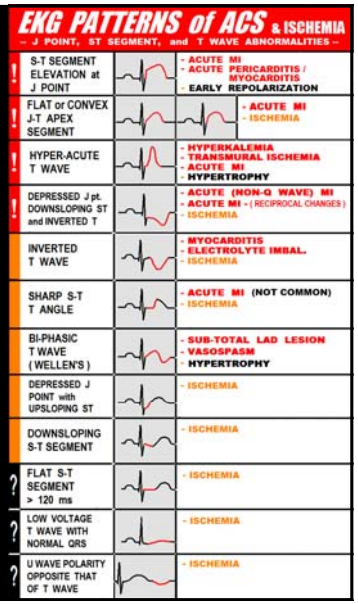
CAREFULLY SCRUTINIZE THESE MARKERS IN EVERY LEAD OF THE 12 LEAD EKG, TO DETERMINE IF THEY ARE *NORMAL* or *ABNORMAL*.

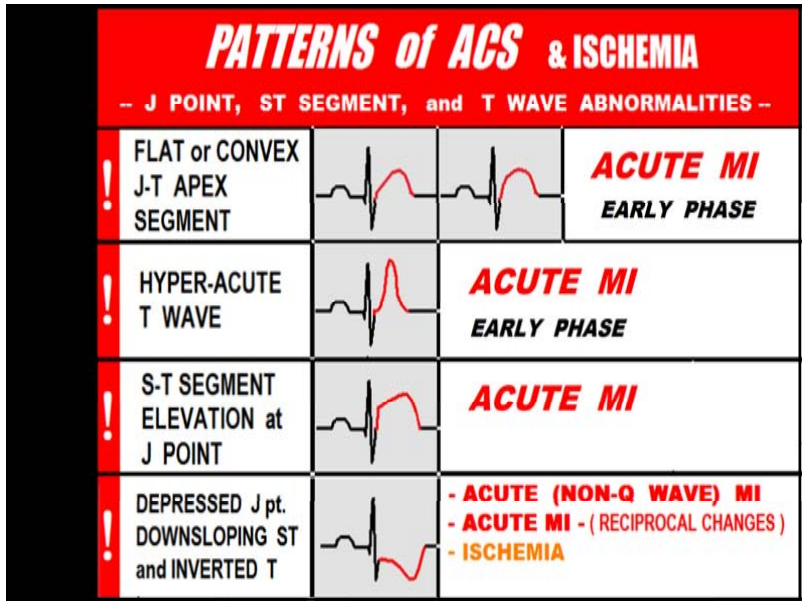
BOOK PAGE: 80

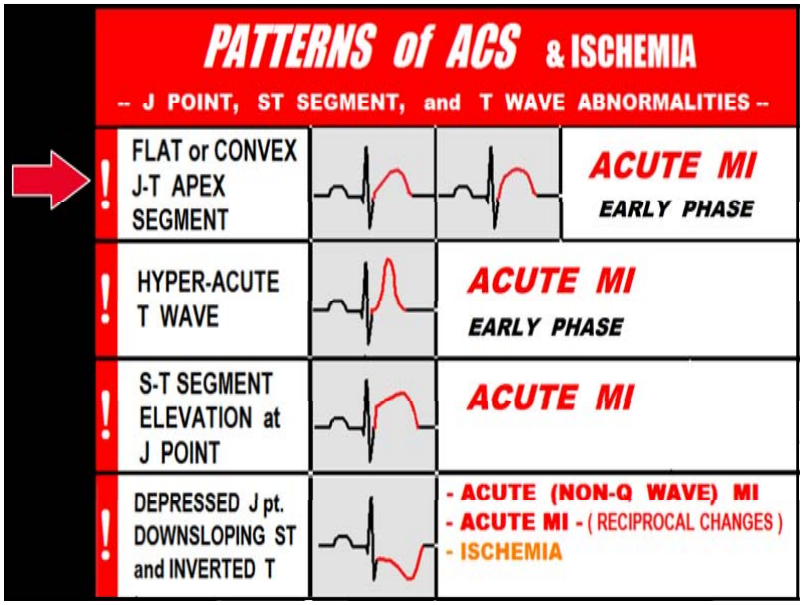
ALL KINDS of *WEIRD* ST SEGMENT and T WAVE VARIATIONS . . . . ALL CAN SPELL T-R-O-U-B-L-E.

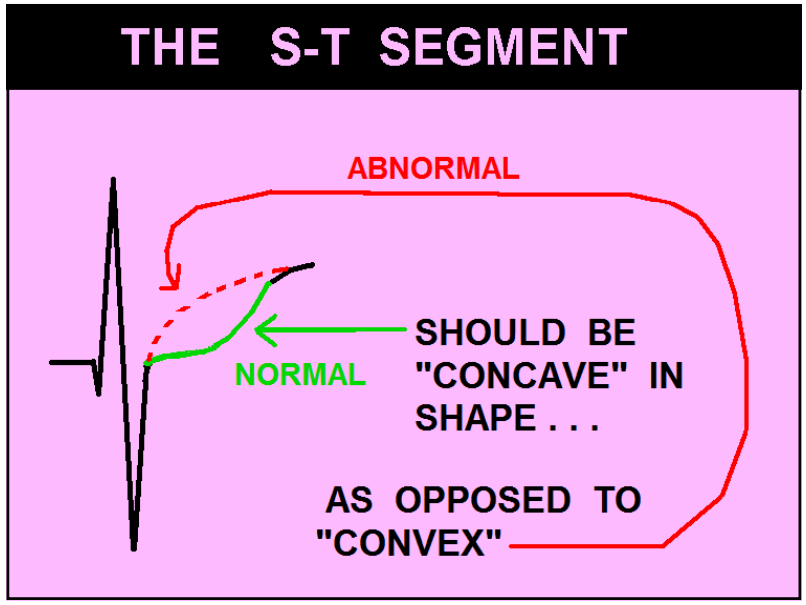
"IF IT'S NOT NORMAL, it's *ABNORMAL !*

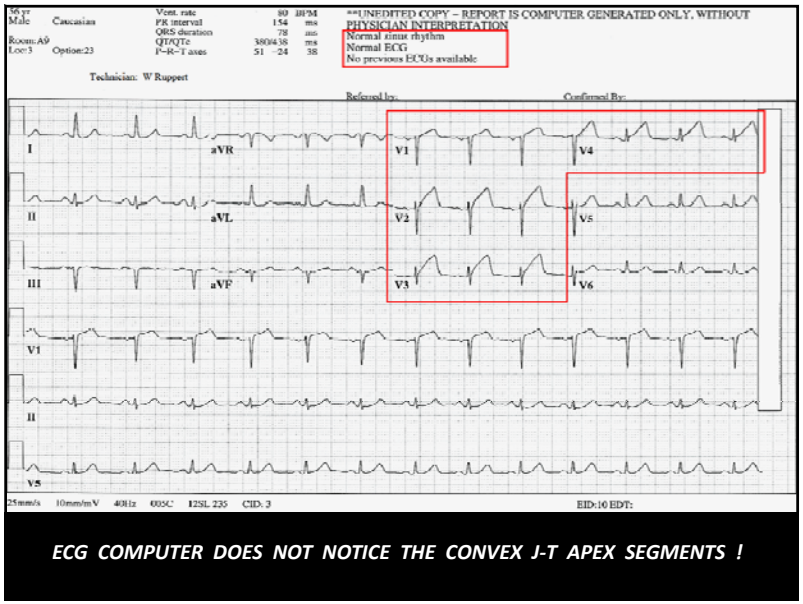
BOOK PAGE: 83

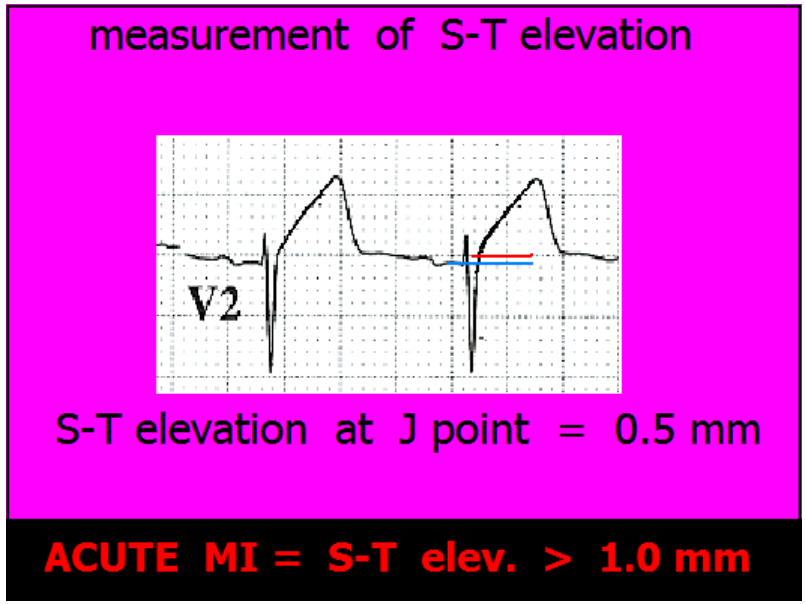





PATTERNS of ACS & ISCHEMIA
-- J POINT, ST SEGMENT, and T WAVE ABNORMALITIES --
FLAT or CONVEX J-T APEX SEGMENT
ACUTE MI
EARLY PHASE
HYPER-ACUTE T WAVE
ACUTE MI
EARLY PHASE
S-T SEGMENT ELEVATION at J POINT
ACUTE MI
DEPRESSED J pt. DOWNSLOPING ST and INVERTED T
- ACUTE (NON-Q WAVE) MI
- ACUTE MI - (RECIPROCAL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


THE S-T SEGMENT
ABNORMAL
NORMAL
SHOULD BE "CONCAVE" IN SHAPE . . .
AS OPPOSED TO "CONVEX"


UNEDITED COPY – REPORT IS COMPUTER GENERATED ONLY, WITHOUT PHYSICIAN INTERPRETATION
Normal sinus rhythm
Normal ECG
No previous ECGs available
Technician: W Ruppert
ECG COMPUTER DOES NOT NOTICE THE CONVEX J-T APEX SEGMENTS !


measurement of S-T elevation
V2
S-T elevation at J point = 0.5 mm
ACUTE MI = S-T elev. > 1.0 mm

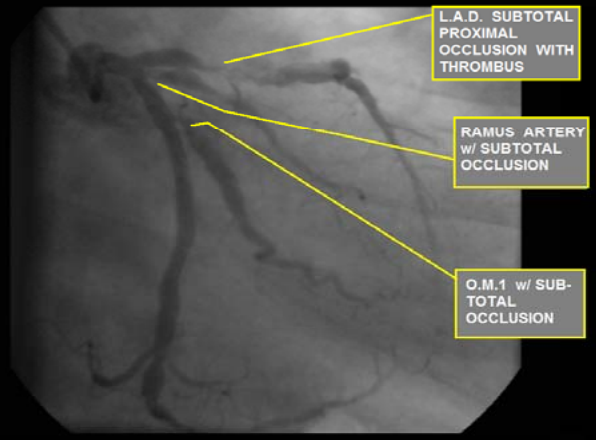
measurement of S-T elevation by "J point + .04" method

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" . . . .



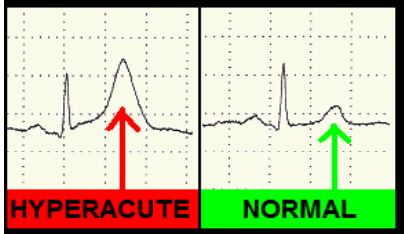
TREATMENT PLAN : EMERGENCY CORONARY ARTERY BYPASS SURGERY (4 VESSEL)

PATTERNS of ACS & ISCHEMIA

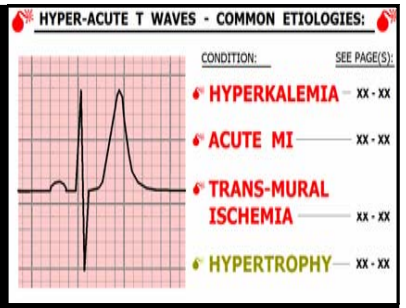
-- J POINT, ST SEGMENT, and T WAVE ABNORMALITIES --

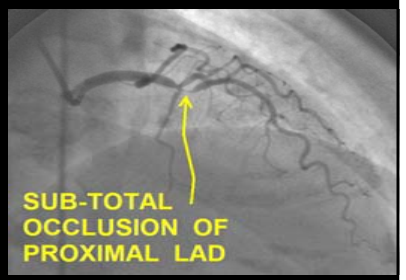
!	FLAT or CONVEX J-T APEX SEGMENT	**ACUTE MI** EARLY PHASE
!	HYPER-ACUTE T WAVE	**ACUTE MI** EARLY PHASE
!	S-T SEGMENT ELEVATION at J POINT	**ACUTE MI**
!	DEPRESSED J pt. DOWNSLOPING ST and INVERTED T	- ACUTE (NON-Q WAVE) MI - ACUTE MI - (RECIPROCAL CHANGES) - ISCHEMIA

HYPERACUTE T WAVES



BOOK PAGE: 88





CASE STUDY: HYPERACUTE T WAVES

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

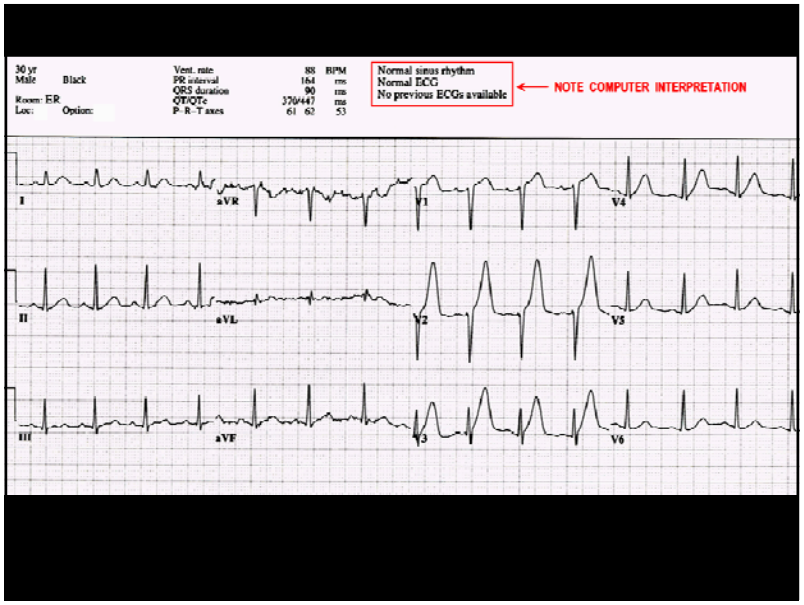
30 y/o male presents to ER via EMS, c/o sudden onset of dull chest pain x 40 min. Pain level varies, not effected by position, movement or deep inspiration. No associated symptoms.

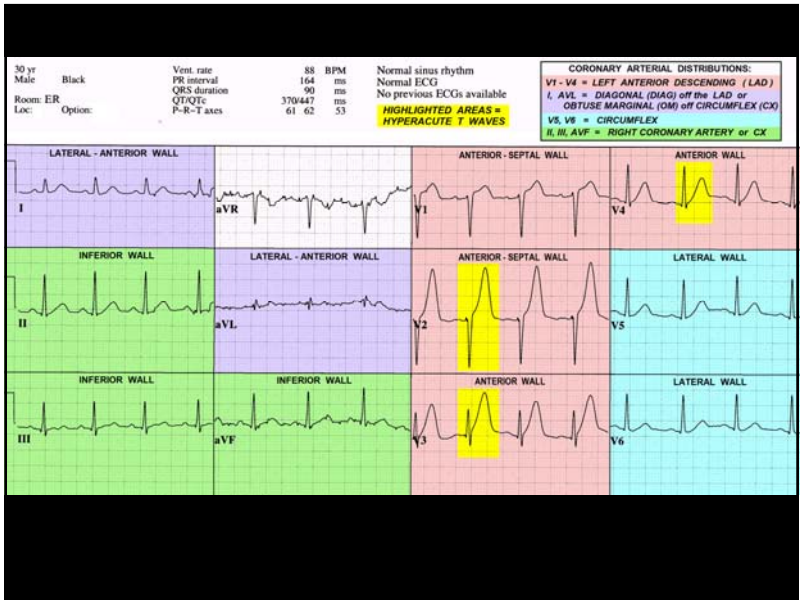
RISK FACTOR PROFILE: NONE. CHOLESTEROL UNKNOWN.

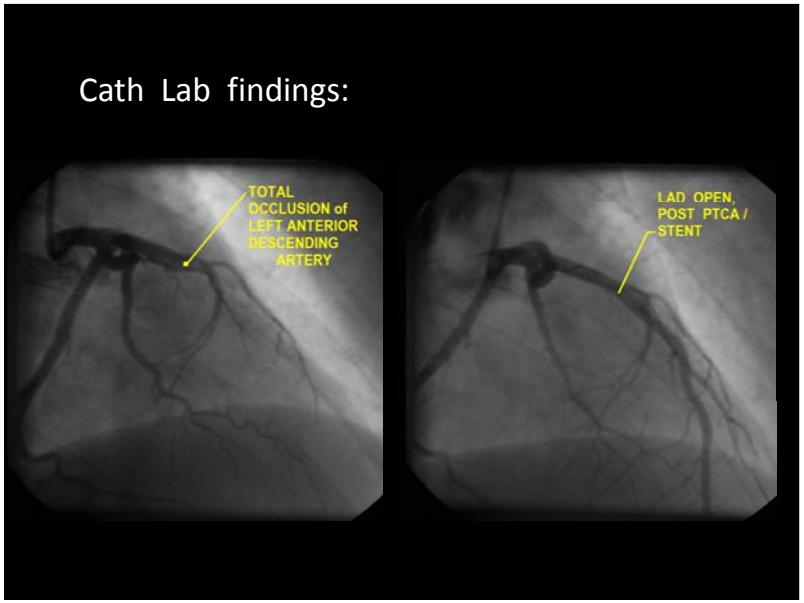
PHYSICAL EXAM: Patient is supine on exam table, CAO x 4, anxious, restless, skin pale, cool, dry. Patient c/o chest pressure, "7" on 1 - 10 scale, uneffected by position, movement, deep inspiration. Lungs clear. HS: NL S1, S2, no rubs, murmurs, gallops

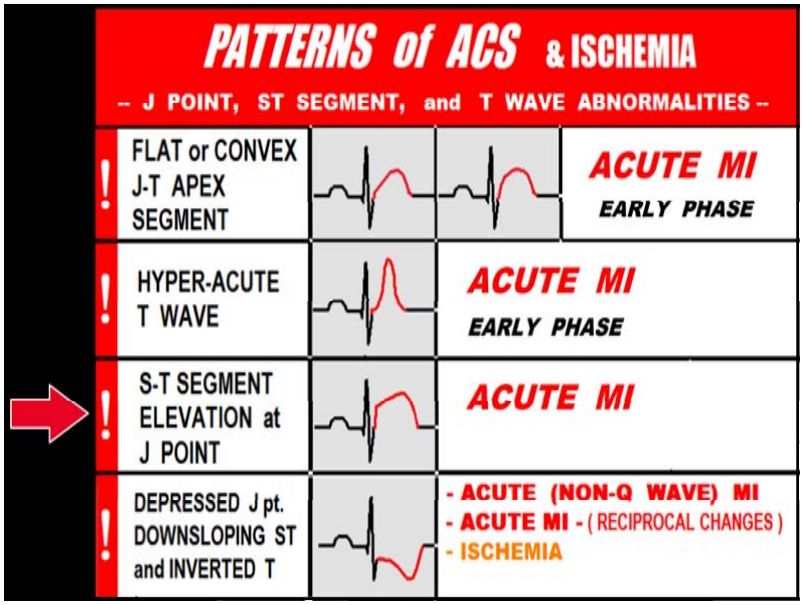
VITAL SIGNS: BP 136/88 P 90 R 20 SAO2 98%

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









ECG CRITERIA for DIAGNOSIS of STEMI:

(ST ELEVATION @ J POINT)

***LEADS V2 and V3:**

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

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

FEMALES ---------- 1.5 mm

ALL OTHER LEADS: 1.0 mm or more, in TWO or more CONTIGUOUS LEADS

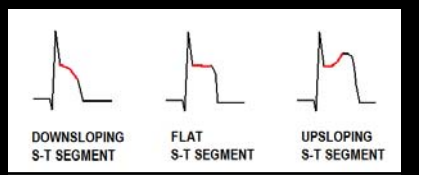
* **P. Rautaharju et al, "Standardization and Interpretation of the ECG," JACC 2009;(53)No.11:982-991**

ST SEGMENT ELEVATION:

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

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

3 COMMON PATTERNS of ST SEGMENT ELEVATION From ACUTE MI:



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

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

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

ACUTE MI

COMPLICATIONS TO ANTICIPATE FOR ALL MI PATIENTS :

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

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.

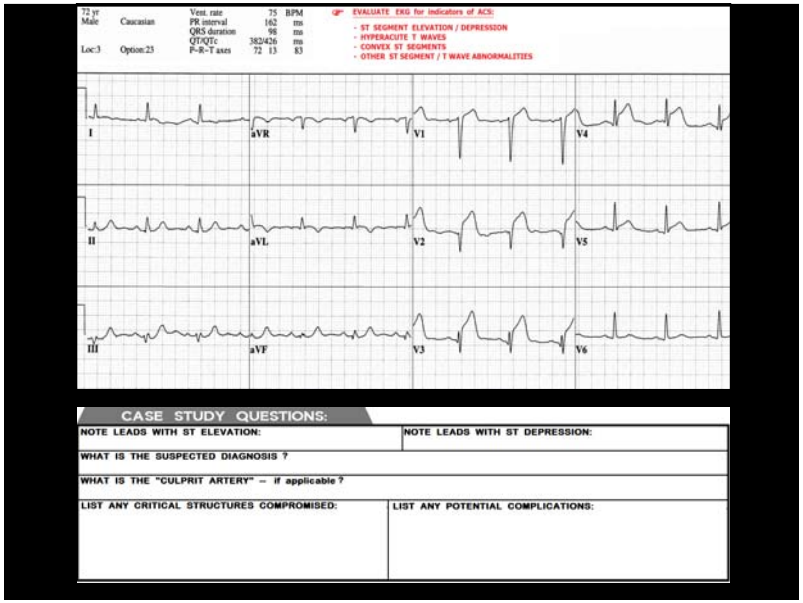
RISK FACTOR PROFILE:

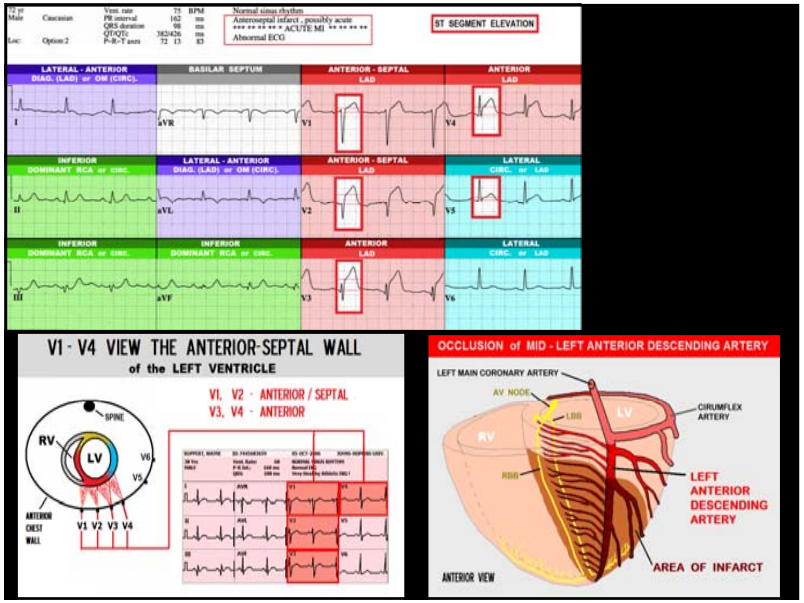
- FAMILY HISTORY - father died of MI at age 77
- FORMER CIGARETTE SMOKER - smoked for 30 year - quit 27 years ago
- DIABETES - oral meds and diet controlled
- HIGH CHOLESTEROL - controlled with STATIN meds
- AGE: OVER 65

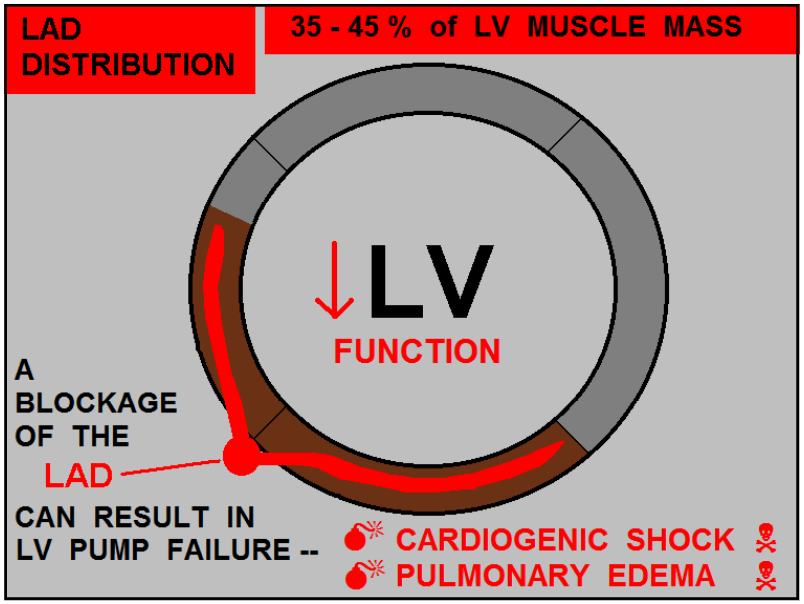
PHYSICAL EXAM: Patient calm, alert, oriented X 4, skin cool, dry, pale. No JVD, Lungs clear bilaterally. Heart sounds normal S1, S2. No peripheral edema.

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

LABS: FIRST TROPONIN: 6.4





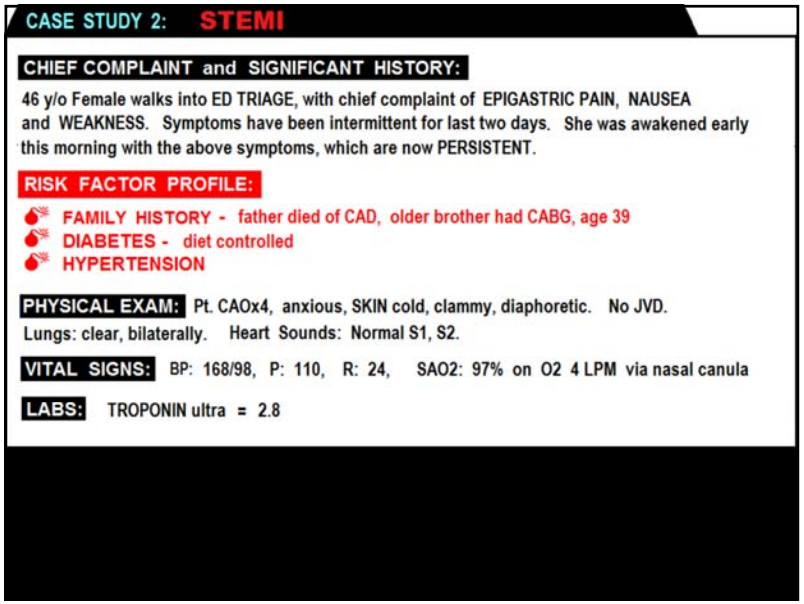


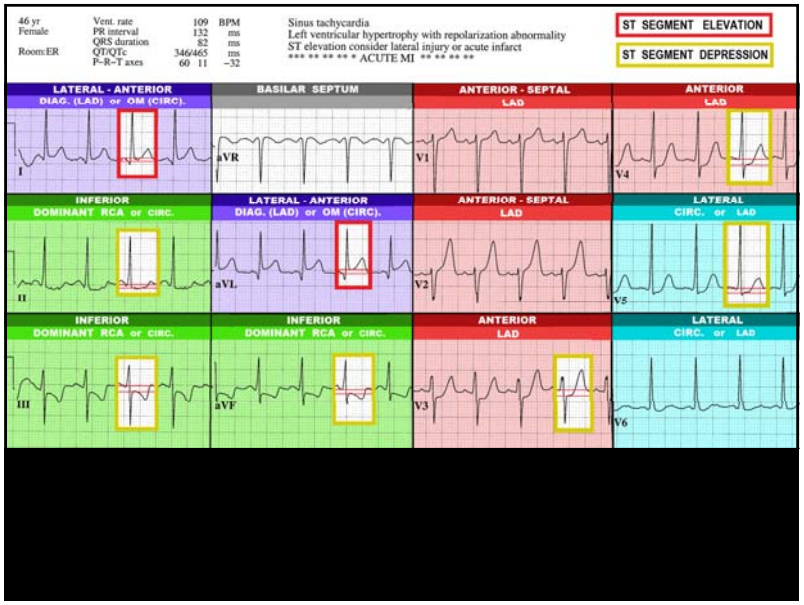
HCFD PROTOCOL – CARDIOGENIC SHOCK:

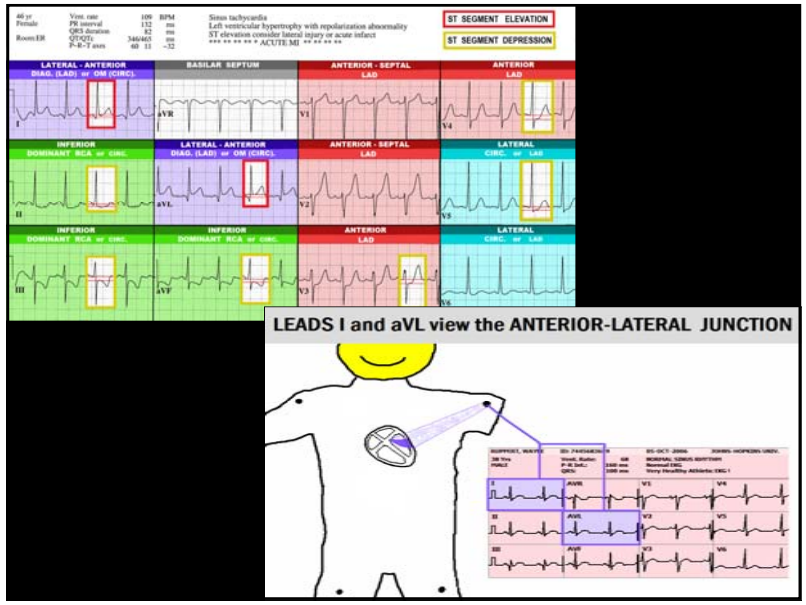
Dopamine drip: (400 mg/250cc/D5W or premix bag) start 5 mcg/kg/min and titrate to achieve a blood pressure of 100 systolic. Max of 20 mcg/kg/min

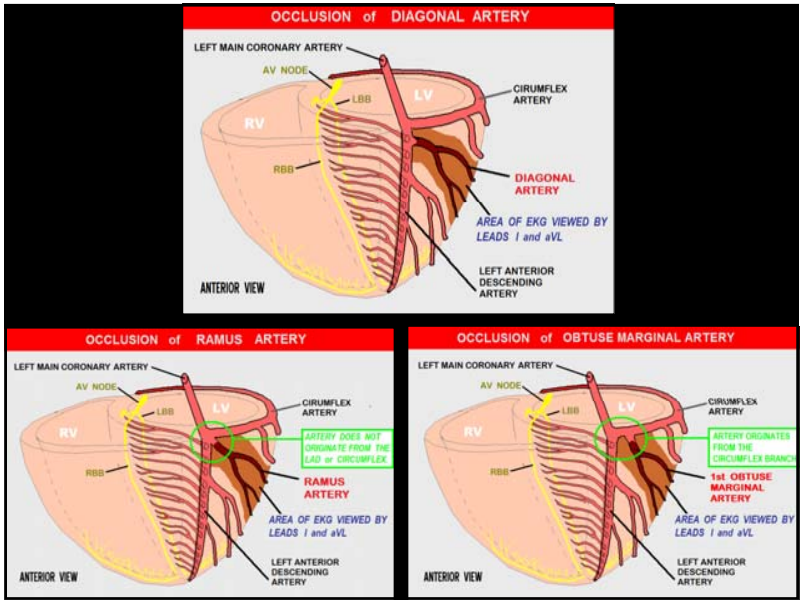
LEFT ANTERIOR DESCENDING ARTERY (LAD)

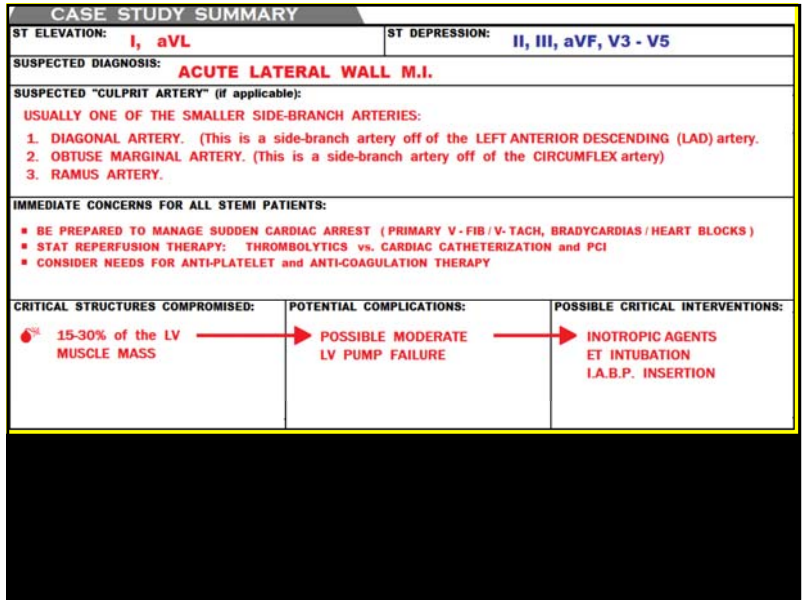
- ANTERIOR WALL OF LEFT VENTRICLE
- 35 - 45 % OF LEFT VENTRICLE MUSCLE MASS
- SEPTUM, ANTERIOR 2/3
- BUNDLE BRANCHES
- ANTERIOR-MEDIAL PAPILLARY MUSCLE




46 yr
Female
Room:ER
Vent. rate 109 BPM
PR interval 132 ms
QRS duration 82 ms
QT/QTc 346/465 ms
P-R-T axes 60 11 -32
Sinus tachycardia
Left ventricular hypertrophy with repolarization abnormality
ST elevation consider lateral injury or acute infarct
*** ** ** ** * ACUTE MI ** ** ** **
ST SEGMENT ELEVATION
ST SEGMENT DEPRESSION
LATERAL - ANTERIOR
DIAG. (LAD) or OM (CIRC).
BASILAR SEPTUM
ANTERIOR - SEPTAL
LAD
ANTERIOR
LAD
INFERIOR
DOMINANT RCA or CIRC.
LATERAL - ANTERIOR
DIAG. (LAD) or OM (CIRC).
ANTERIOR - SEPTAL
LAD
LATERAL
CIRC. or LAD
INFERIOR
DOMINANT RCA or CIRC.
INFERIOR
DOMINANT RCA or CIRC.
ANTERIOR
LAD
LATERAL
CIRC. or LAD
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6


ST SEGMENT ELEVATION
ST SEGMENT DEPRESSION
LEADS I and aVL view the ANTERIOR-LATERAL JUNCTION


OCCLUSION of DIAGONAL ARTERY
LEFT MAIN CORONARY ARTERY
AV NODE
LBB
LV
RV
RBB
CIRUMFLEX ARTERY
DIAGONAL ARTERY
AREA OF EKG VIEWED BY LEADS I and aVL
LEFT ANTERIOR DESCENDING ARTERY
ANTERIOR VIEW
OCCLUSION of RAMUS ARTERY
ARTERY DOES NOT ORIGINATE FROM THE LAD or CIRCUMFLEX
RAMUS ARTERY
OCCLUSION of OBTUSE MARGINAL ARTERY
ARTERY ORIGINATES FROM THE CIRCUMFLEX BRANCH
1st OBTUSE MARGINAL ARTERY


CASE STUDY SUMMARY
ST ELEVATION: I, aVL
ST DEPRESSION: II, III, aVF, V3 - V5
SUSPECTED DIAGNOSIS: ACUTE LATERAL WALL M.I.
SUSPECTED "CULPRIT ARTERY" (if applicable):
USUALLY ONE OF THE SMALLER SIDE-BRANCH ARTERIES:
1. DIAGONAL ARTERY. (This is a side-branch artery off of the LEFT ANTERIOR DESCENDING (LAD) artery.
2. OBTUSE MARGINAL ARTERY. (This is a side-branch artery off of the CIRCUMFLEX artery)
3. RAMUS ARTERY.
IMMEDIATE CONCERNS FOR ALL STEMI PATIENTS:
BE PREPARED TO MANAGE SUDDEN CARDIAC ARREST (PRIMARY V - FIB / V- TACH, BRADYCARDIAS / HEART BLOCKS)
STAT REPERFUSION THERAPY: THROMBOLYTICS vs. CARDIAC CATHETERIZATION and PCI
CONSIDER NEEDS FOR ANTI-PLATELET and ANTI-COAGULATION THERAPY
CRITICAL STRUCTURES COMPROMISED:
15-30% of the LV MUSCLE MASS
POTENTIAL COMPLICATIONS:
POSSIBLE MODERATE LV PUMP FAILURE
POSSIBLE CRITICAL INTERVENTIONS:
INOTROPIC AGENTS
ET INTUBATION
I.A.B.P. INSERTION

CASE STUDY 3: STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

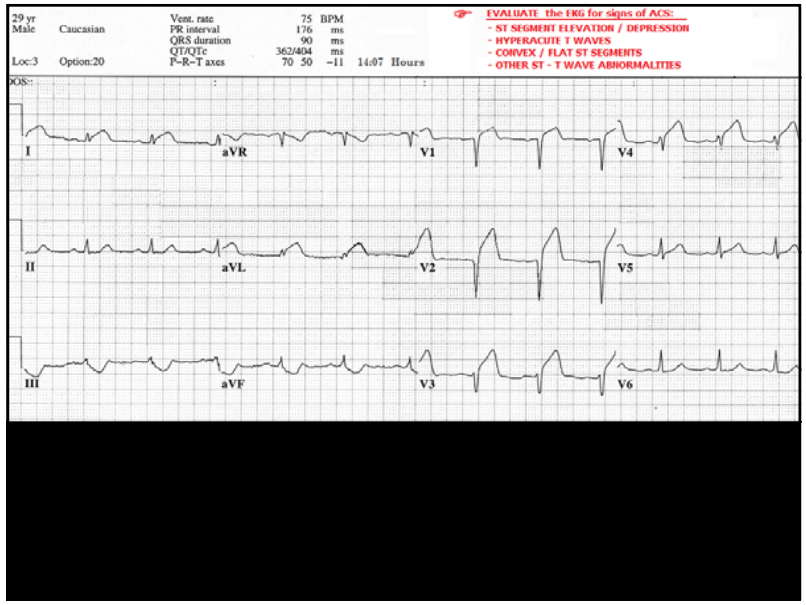
RISK FACTOR PROFILE:

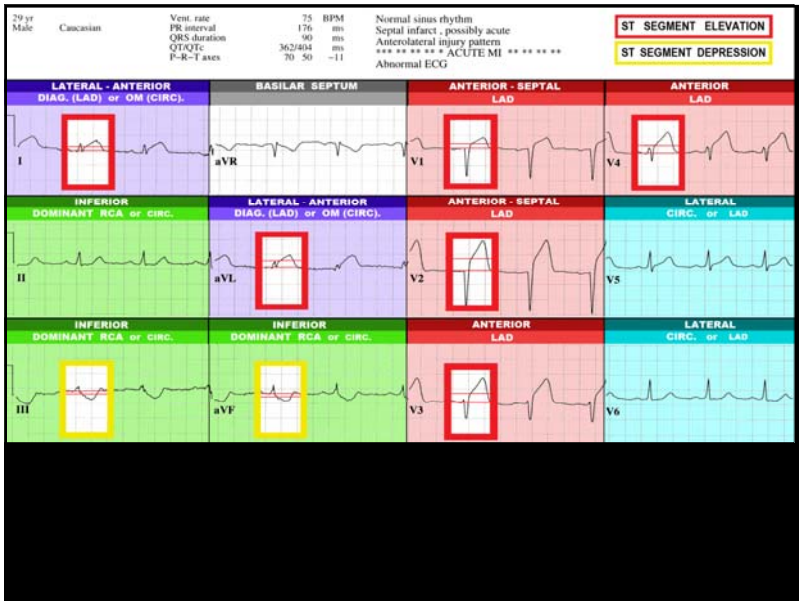
- FAMILY HISTORY - father died of MI age 46
- CURRENT CIGARETTE SMOKER
- "MILD" HYPERTENSION - untreated
- ☑ CHOLESTEROL - unknown - "never had it checked."

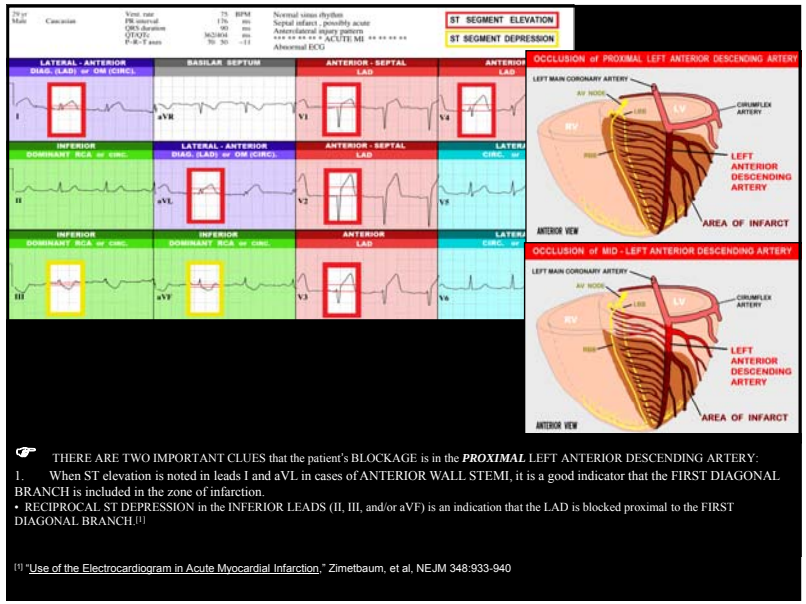
PHYSICAL EXAM: Patient alert, oriented X 4, skin cool, dry, pale. Patient restless. No JVD, Lungs clear bilaterally. Heart sounds normal S1, S2. No peripheral edema.

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

LABS: INITIAL CARDIAC MARKERS - NEGATIVE







☞ THERE ARE TWO IMPORTANT CLUES that the patient's BLOCKAGE is in the ***PROXIMAL*** LEFT ANTERIOR DESCENDING ARTERY:

1. When ST elevation is noted in leads I and aVL in cases of ANTERIOR WALL STEMI, it is a good indicator that the FIRST DIAGONAL BRANCH is included in the zone of infarction.
• RECIPROCAL ST DEPRESSION in the INFERIOR LEADS (II, III, and/or aVF) is an indication that the LAD is blocked proximal to the FIRST DIAGONAL BRANCH.[1]

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

CASE STUDY SUMMARY

ST ELEVATION: V1 - V5, I, aVL	ST DEPRESSION: III, aVF
SUSPECTED DIAGNOSIS: ACUTE ANTERIOR - SEPTAL STEMI	
SUSPECTED "CULPRIT ARTERY" (if applicable): PROXIMAL LEFT ANTERIOR DESCENDING ARTERY (LAD)	

IMMEDIATE CONCERNS FOR ALL STEMI PATIENTS:

- BE PREPARED TO MANAGE SUDDEN CARDIAC ARREST (PRIMARY V - FIB / V- TACH, BRADYCARDIAS / HEART BLOCKS)
- STAT REPERFUSION THERAPY: THROMBOLYTICS vs. CARDIAC CATHETERIZATION and PCI
- CONSIDER NEEDS FOR ANTI-PLATELET and ANTI-COAGULATION THERAPY

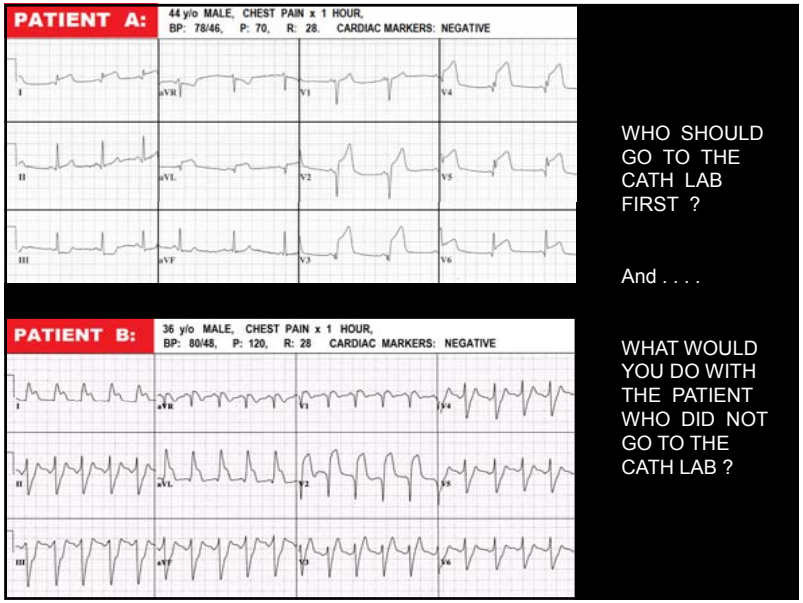
CRITICAL STRUCTURES COMPROMISED:	POTENTIAL COMPLICATIONS:	POSSIBLE CRITICAL INTERVENTIONS:
40-50% of the LV MUSCLE MASS	LV PUMP FAILURE leading to: - CARDIOGENIC SHOCK - PULMONARY EDEMA	INOTROPIC AGENTS ET INTUBATION I.A.B.P. INSERTION
	VENTRICULAR DYSRHYTHMIAS (VT / VF)	DEFIBRILLATION / ANTIARRHYTHMIC AGENTS
Potential compromise of BLOOD SUPPLY to: - Bundle of His - Proximal Bundle Branches	HIGH-GRADE HEART BLOCKS (2nd - 3rd degree) BUNDLE BRANCH BLOCKS	TRANSCUTANEOUS or TRANSVENOUS PACING

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

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.

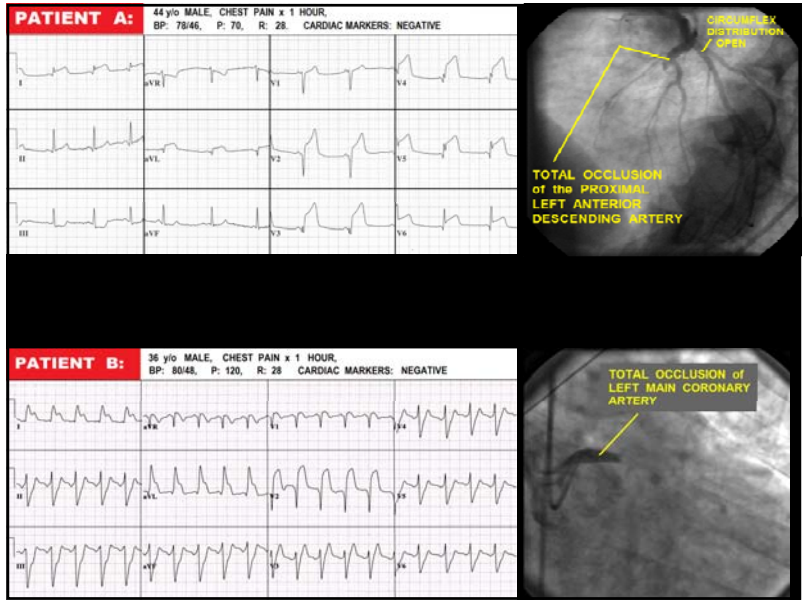
You are the ranking medical officer on duty in the ED when two acute STEMI patients arrive, ten minutes apart. The Cath Lab has one lab open, and can take ONE patient immediately. Both patients duration of symptoms and state of hemodynamic stability are similar.

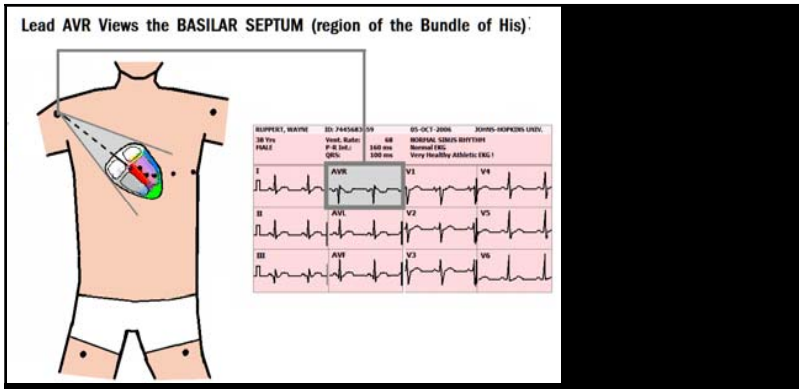


WHO SHOULD GO TO THE CATH LAB FIRST ?

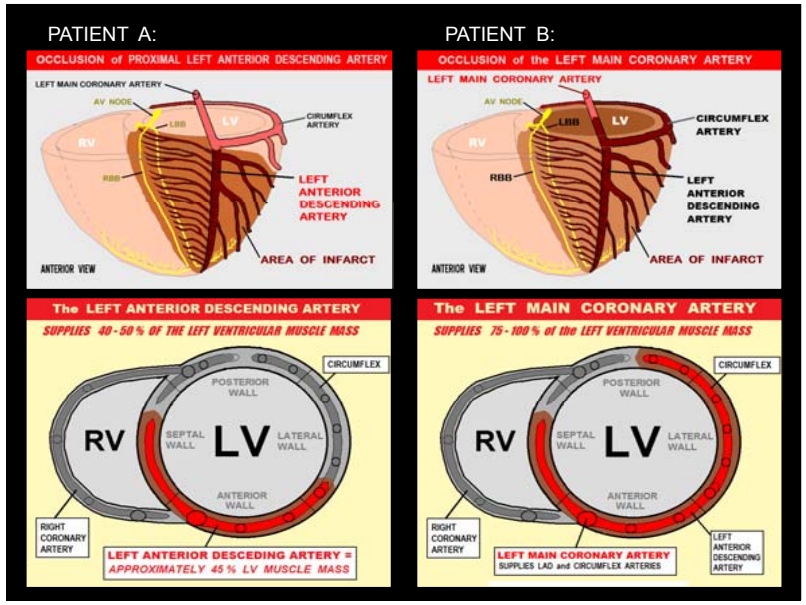
And . . . .

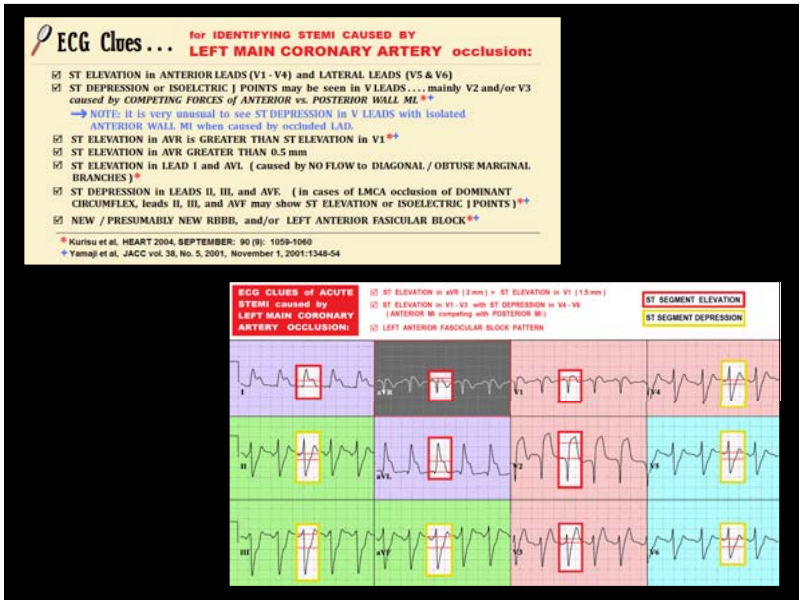
WHAT WOULD YOU DO WITH THE PATIENT WHO DID NOT GO TO THE CATH LAB ?

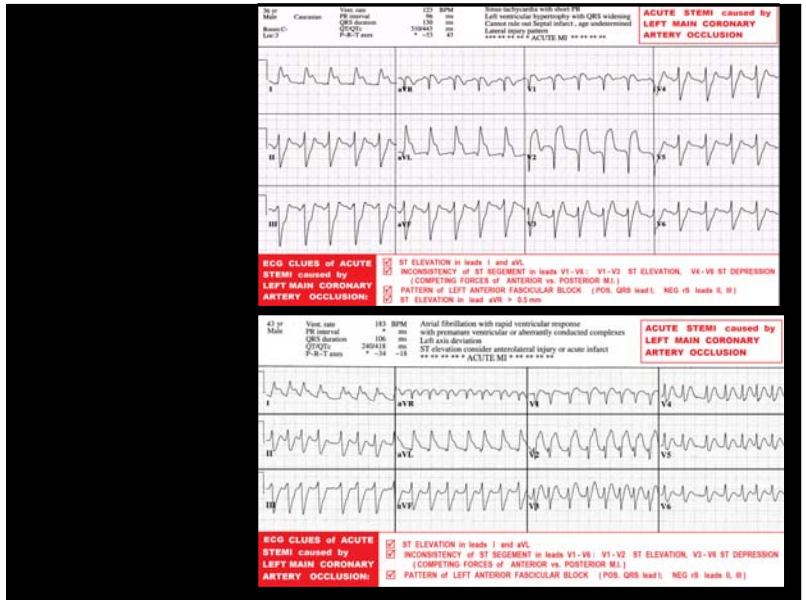


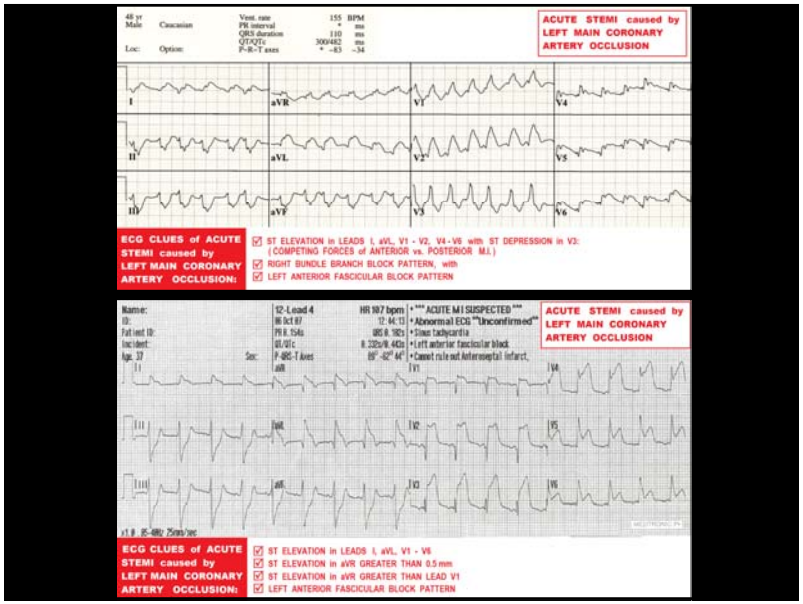


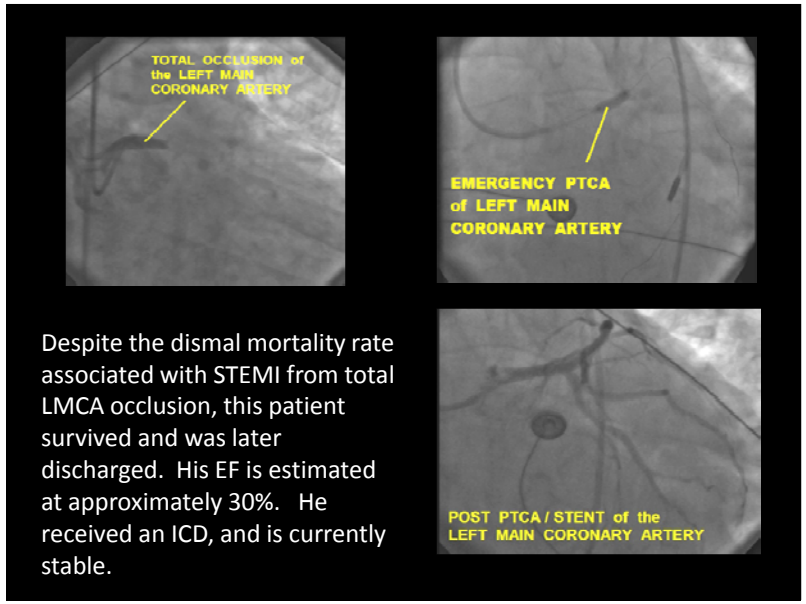
ST ELEVATION in Lead AVR
With STEMI . . . . .
THINK *"LEFT MAIN CORONARY ARTERY OCCLUSION !"*



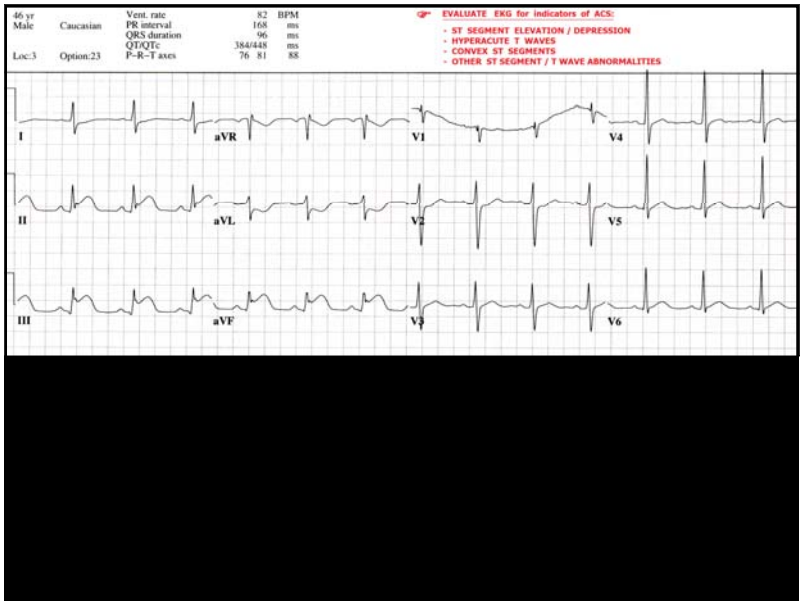


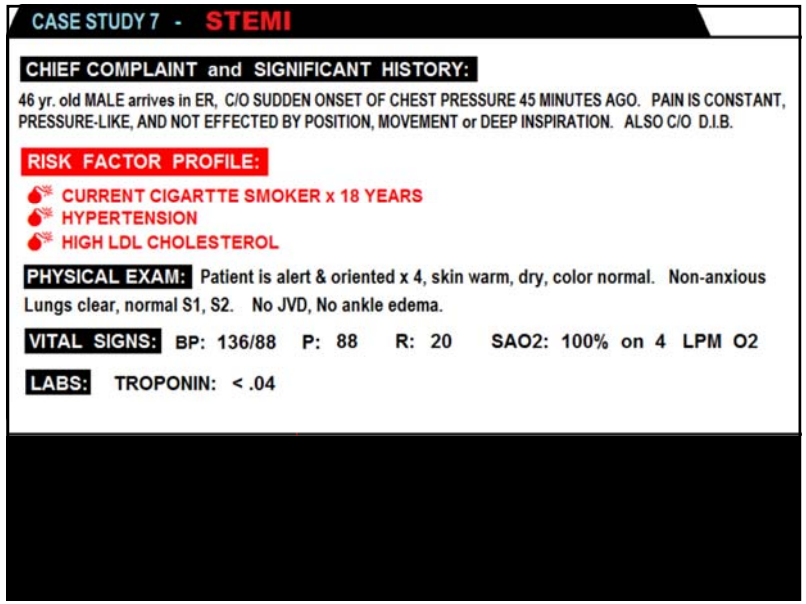


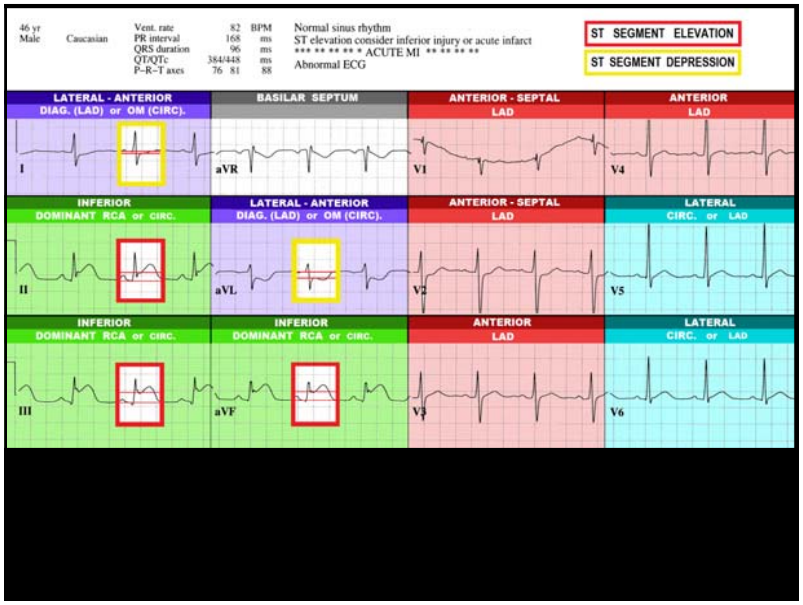


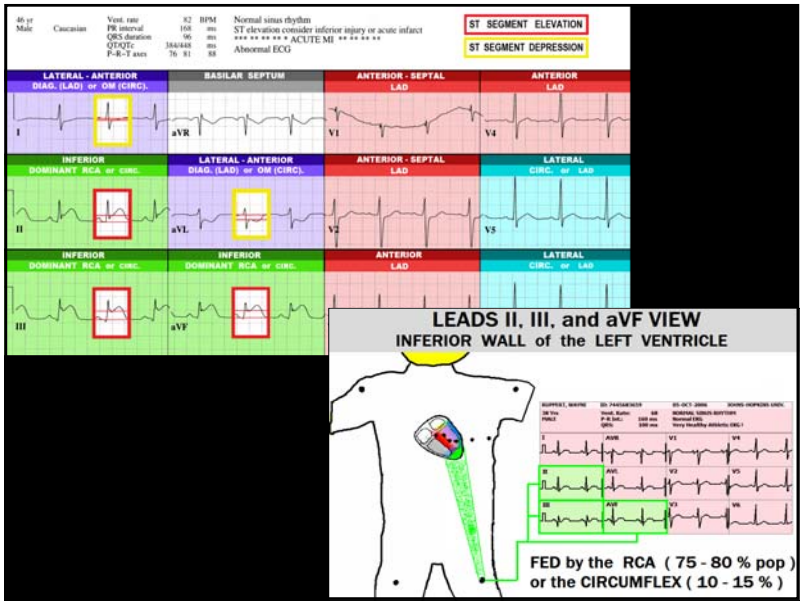


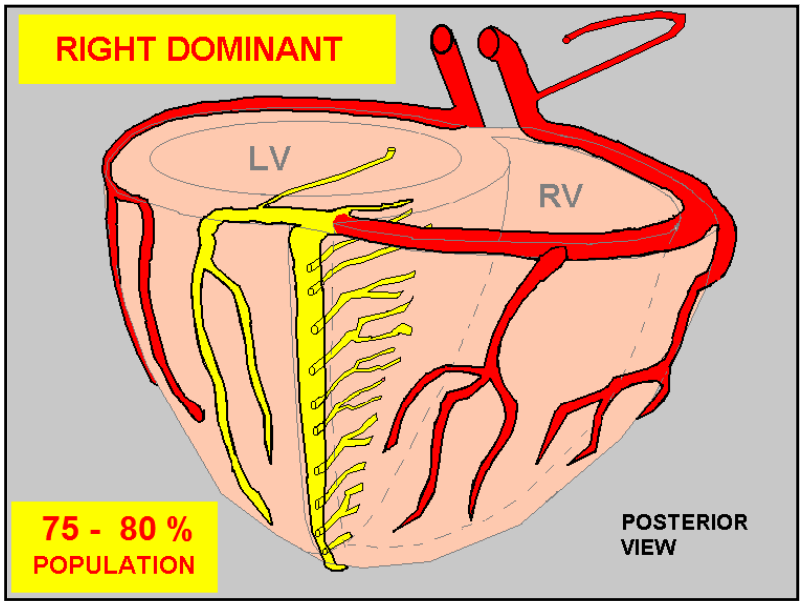
Despite the dismal mortality rate associated with STEMI from total LMCA occlusion, this patient survived and was later discharged. His EF is estimated at approximately 30%. He received an ICD, and is currently stable.











INFERIOR WALL MI:

ALWAYS RULE OUT

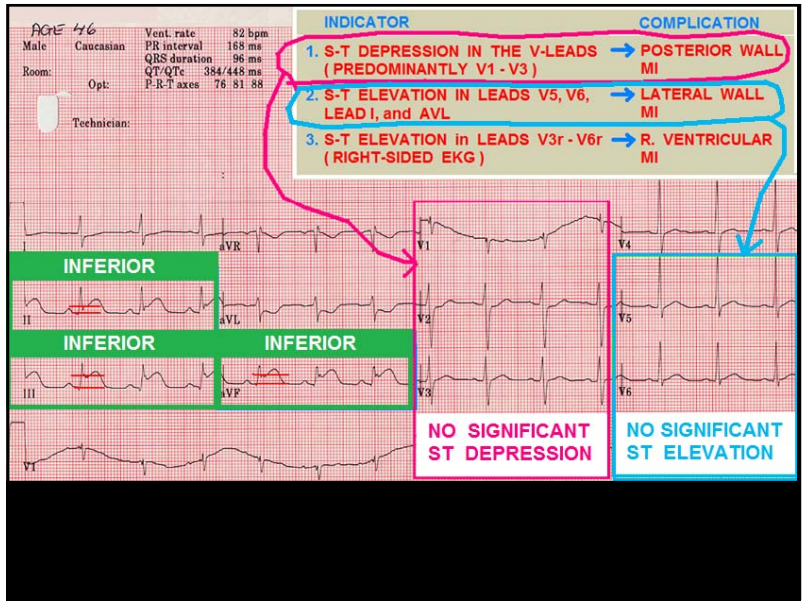
RIGHT VENTRICULAR MI

BEFORE
GIVING ANY NITRATES !!

The MANY FACES of INFERIOR MI ...

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

INDICATOR		COMPLICATION
1. S-T DEPRESSION IN THE V-LEADS (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 (RIGHT-SIDED EKG)	→	R. VENTRICULAR MI

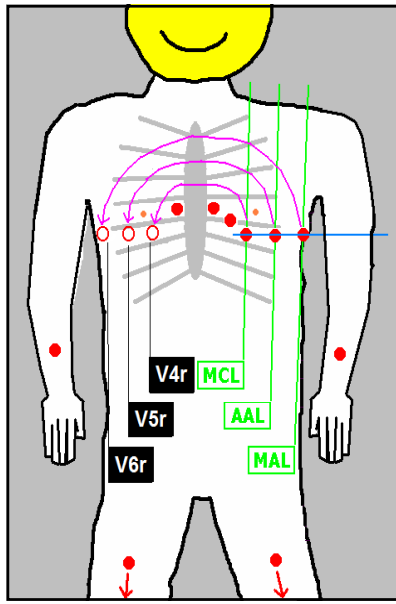


A standard

12 LEAD EKG

Does NOT show the

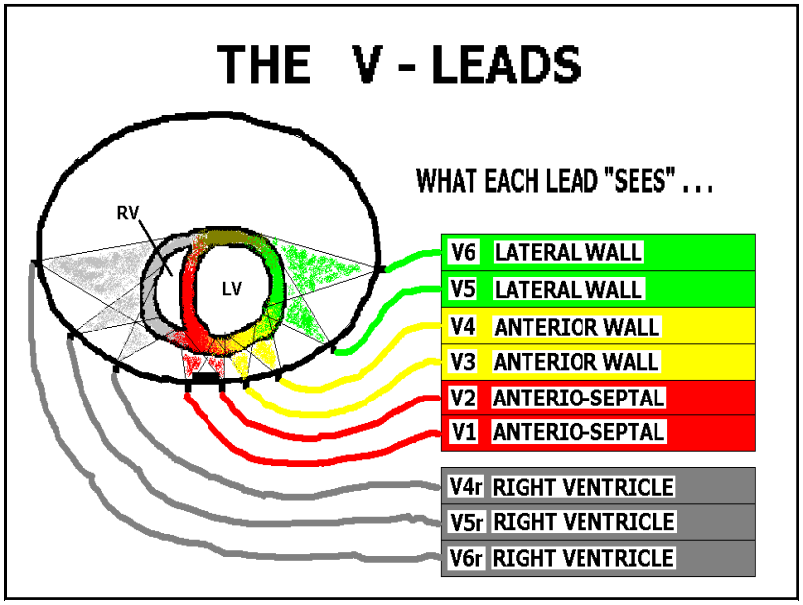
RIGHT VENTRICLE

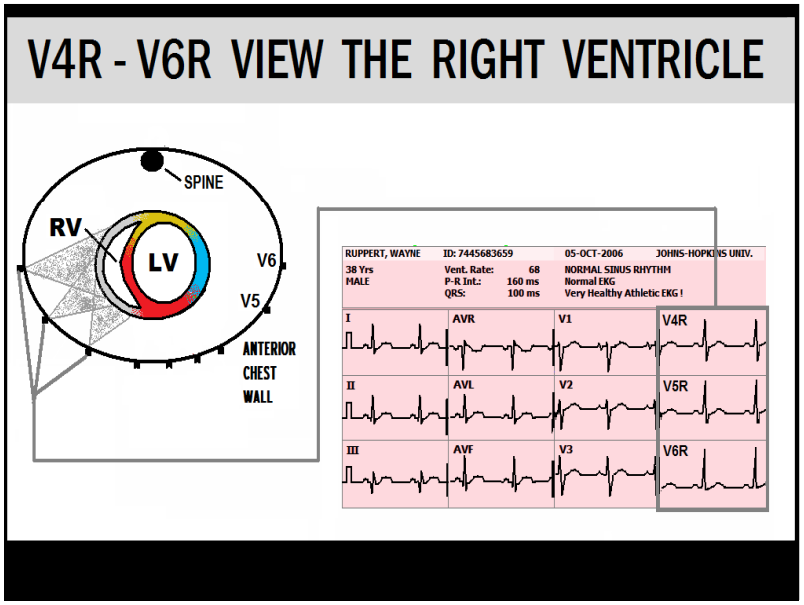


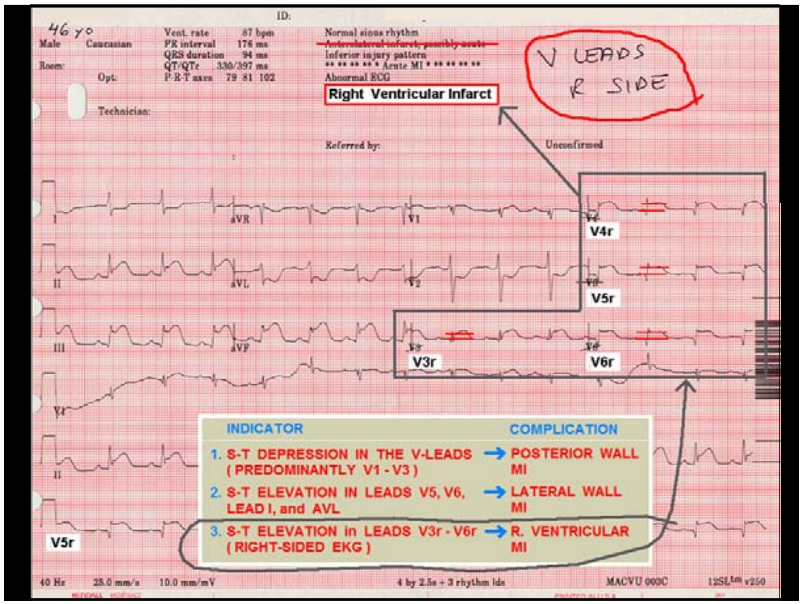
To do a RIGHT - SIDED EKG . .

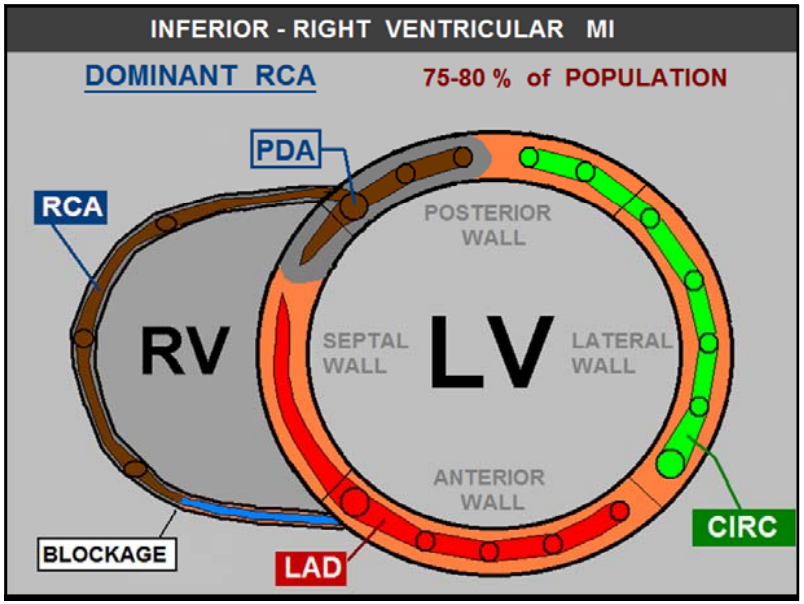
MOVE leads V4, V5, and V6

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

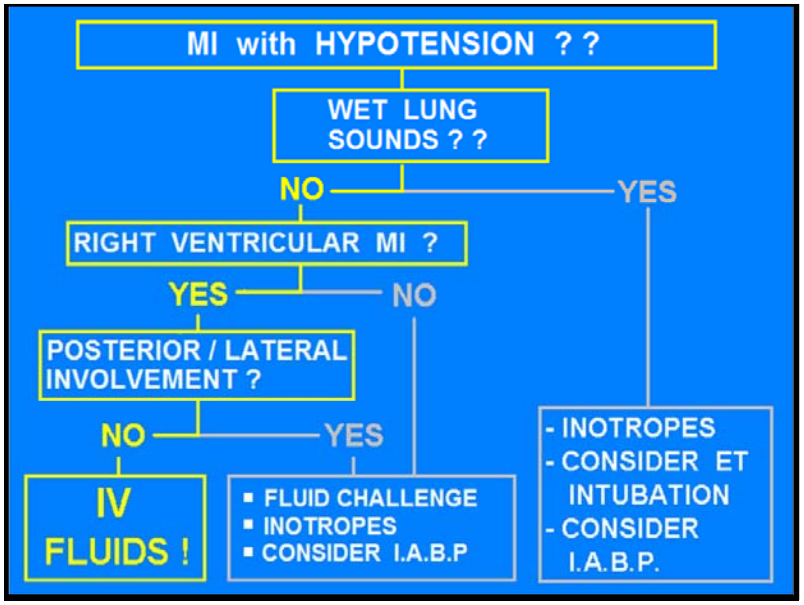

THE V - LEADS
WHAT EACH LEAD "SEES" . . .
RV
LV
V6 LATERAL WALL
V5 LATERAL WALL
V4 ANTERIOR WALL
V3 ANTERIOR WALL
V2 ANTERIO-SEPTAL
V1 ANTERIO-SEPTAL
V4r RIGHT VENTRICLE
V5r RIGHT VENTRICLE
V6r RIGHT VENTRICLE

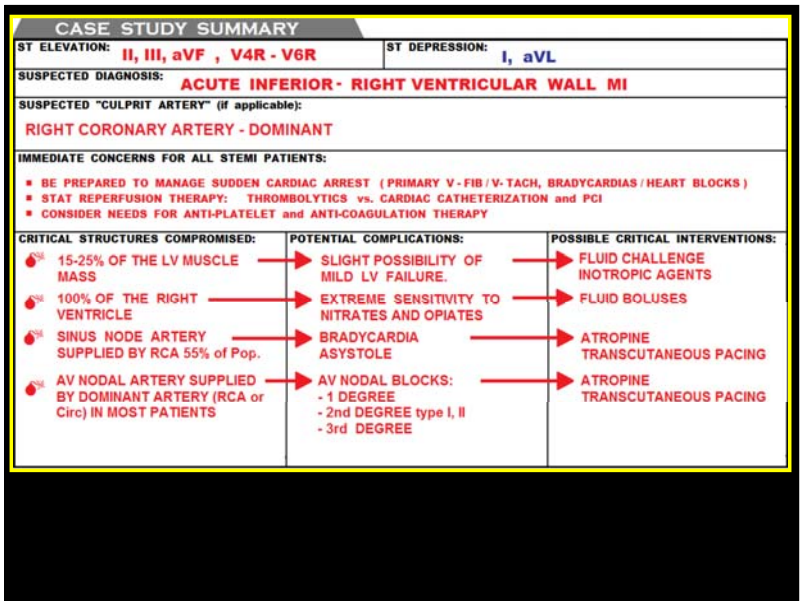

V4R - V6R VIEW THE RIGHT VENTRICLE
SPINE
RV
LV
V6
V5
ANTERIOR CHEST WALL
RUPPERT, WAYNE
ID: 7445683659
05-OCT-2006
JOHNS-HOPKINS UNIV.
38 Yrs
MALE
Vent. Rate: 68
P-R Int.: 160 ms
QRS: 100 ms
NORMAL SINUS RHYTHM
Normal EKG
Very Healthy Athletic EKG !
I
AVR
V1
V4R
II
AVL
V2
V5R
III
AVF
V3
V6R


46 yo
Male Caucasian
Vent. rate 87 bpm
PR interval 176 ms
QRS duration 94 ms
QT/QTc 330/397 ms
P-R-T axes 79 81 102
Normal sinus rhythm
Inferior injury pattern
Abnormal ECG
Right Ventricular Infarct
V LEADS R SIDE
Unconfirmed
V4r
V5r
V3r
V6r
V5r
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 (RIGHT-SIDED EKG) → R. VENTRICULAR MI


INFERIOR - RIGHT VENTRICULAR MI
DOMINANT RCA
75-80 % of POPULATION
PDA
RCA
POSTERIOR WALL
RV
SEPTAL WALL
LV
LATERAL WALL
ANTERIOR WALL
CIRC
BLOCKAGE
LAD

If this patient becomes HYPOTENSIVE . . . . .





CASE STUDY 9 - STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

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

RISK FACTOR PROFILE:

- CIGARETTE SMOKER
- HYPERTENSION
- HIGH LDL CHOLESTEROL

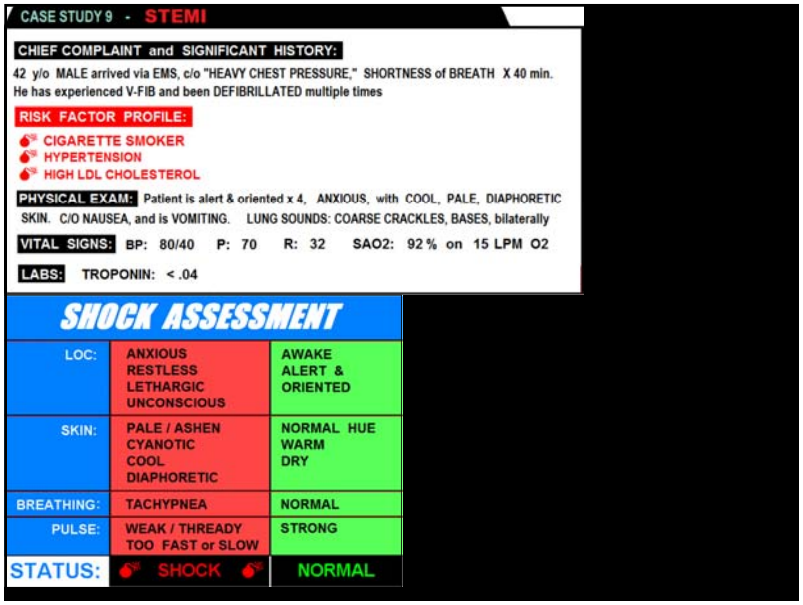
PHYSICAL EXAM: Patient is alert & oriented x 4, ANXIOUS, with COOL, PALE, DIAPHORETIC SKIN. C/O NAUSEA, and is VOMITING. LUNG SOUNDS: COARSE CRACKLES, BASES, bilaterally

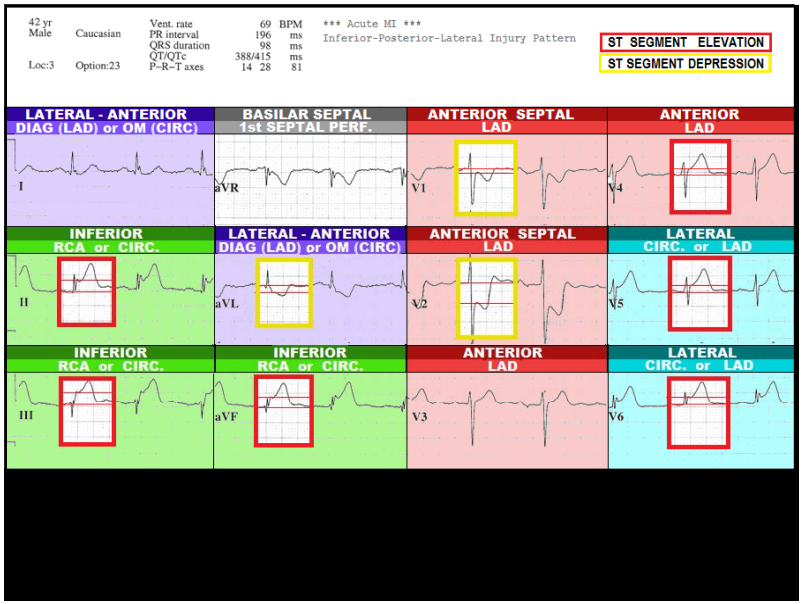
VITAL SIGNS: BP: 80/40 P: 70 R: 32 SAO2: 92 % on 15 LPM O2

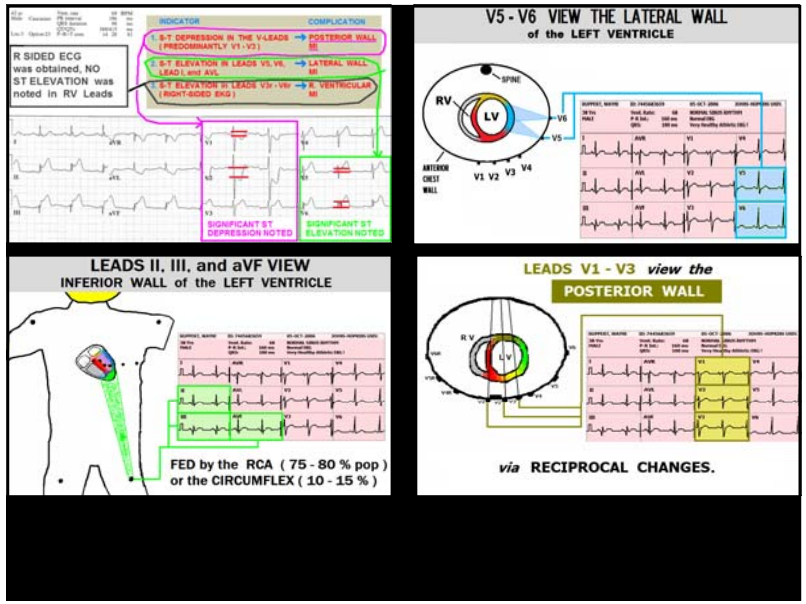
LABS: TROPONIN: < .04

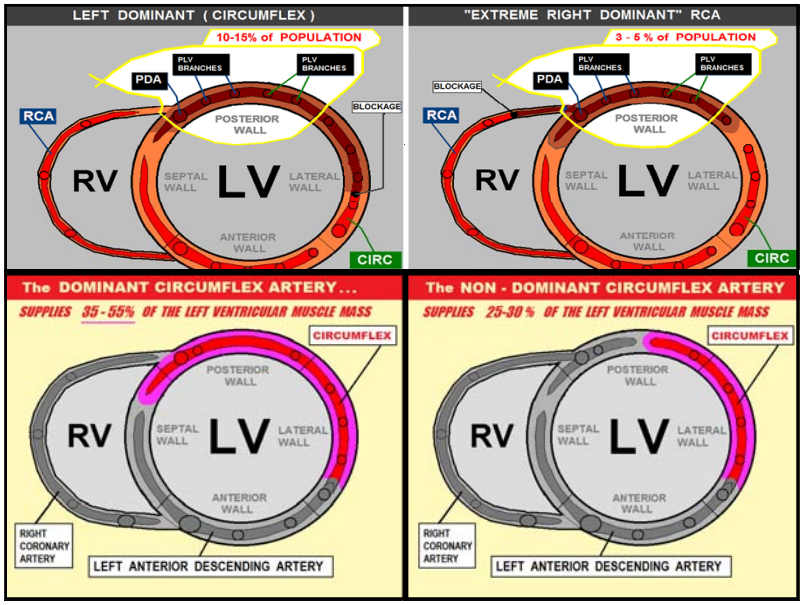
SHOCK ASSESSMENT

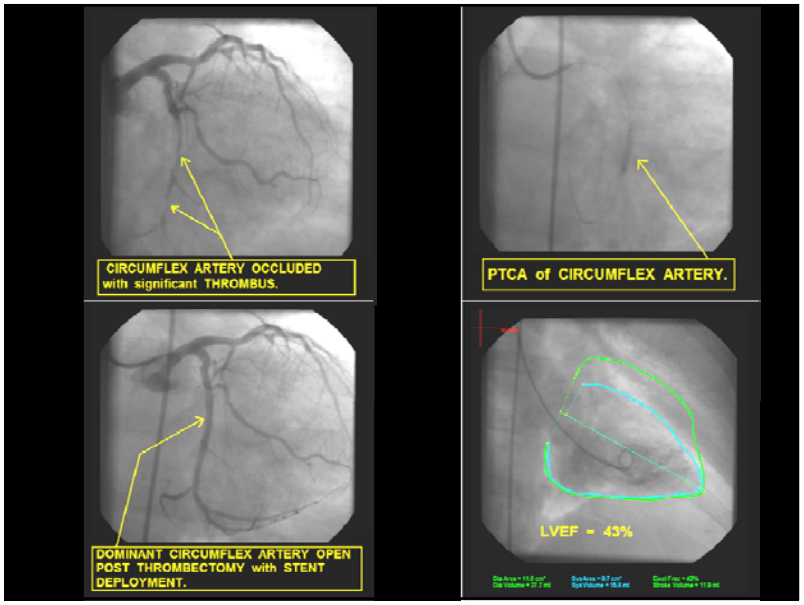
LOC:	ANXIOUS RESTLESS LETHARGIC UNCONSCIOUS	AWAKE ALERT & ORIENTED
SKIN:	PALE / ASHEN CYANOTIC COOL DIAPHORETIC	NORMAL HUE WARM DRY
BREATHING:	TACHYPNEA	NORMAL
PULSE:	WEAK / THREADY TOO FAST or SLOW	STRONG
STATUS:	SHOCK	NORMAL

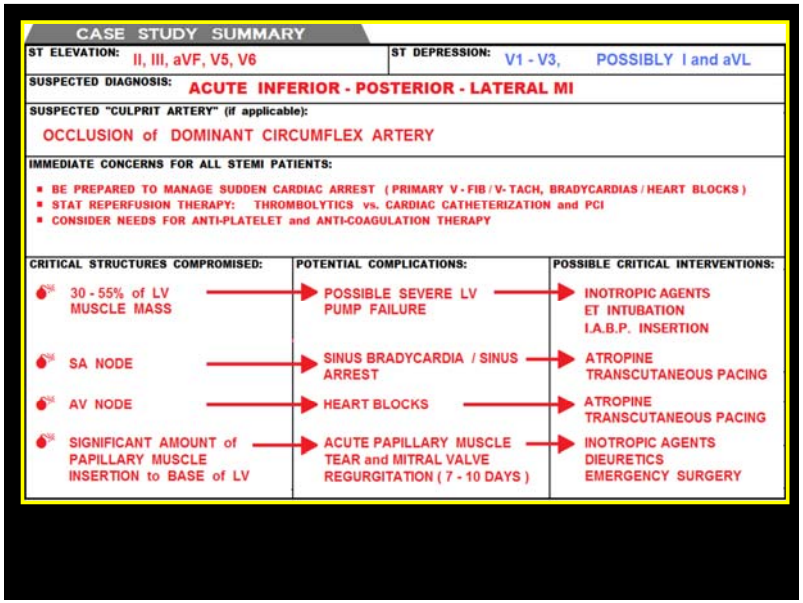


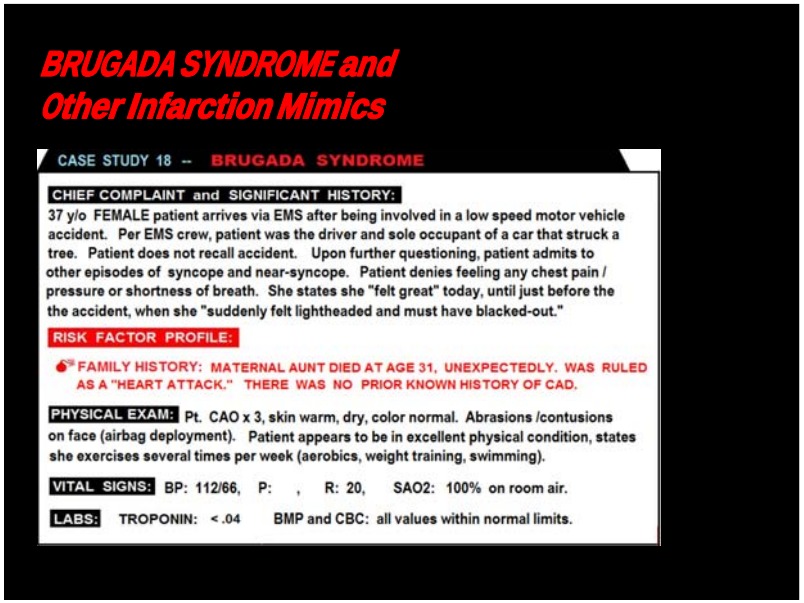


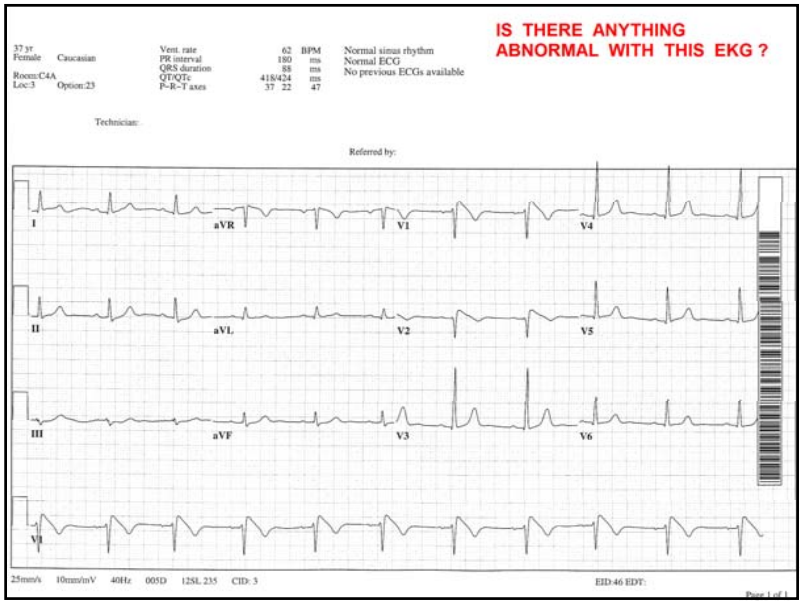


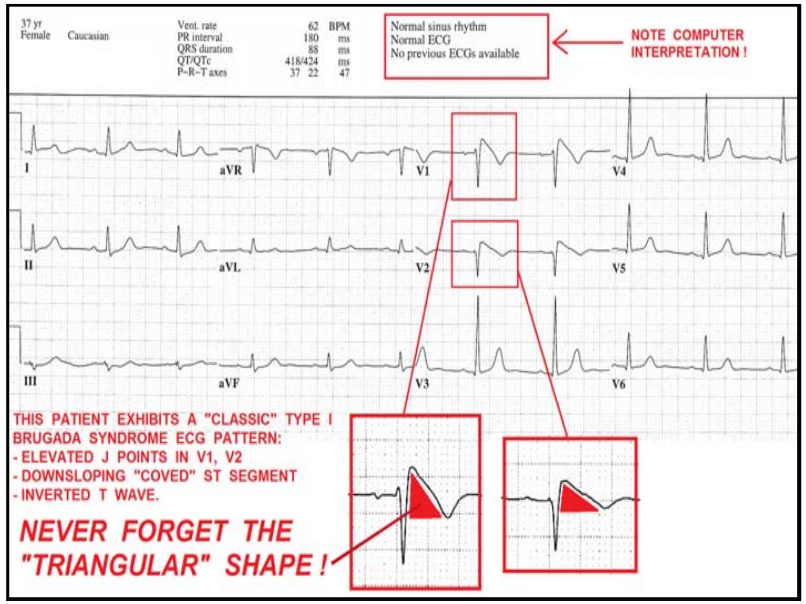

LEFT DOMINANT (CIRCUMFLEX)
10-15% of POPULATION
PLV BRANCHES
PLV BRANCHES
PDA
RCA
BLOCKAGE
POSTERIOR WALL
RV
SEPTAL WALL
LV
LATERAL WALL
ANTERIOR WALL
CIRC
"EXTREME RIGHT DOMINANT" RCA
3 - 5 % of POPULATION
BLOCKAGE
The DOMINANT CIRCUMFLEX ARTERY...
SUPPLIES 35 - 55% OF THE LEFT VENTRICULAR MUSCLE MASS
CIRCUMFLEX
RIGHT CORONARY ARTERY
LEFT ANTERIOR DESCENDING ARTERY
The NON - DOMINANT CIRCUMFLEX ARTERY
SUPPLIES 25-30 % OF THE LEFT VENTRICULAR MUSCLE MASS

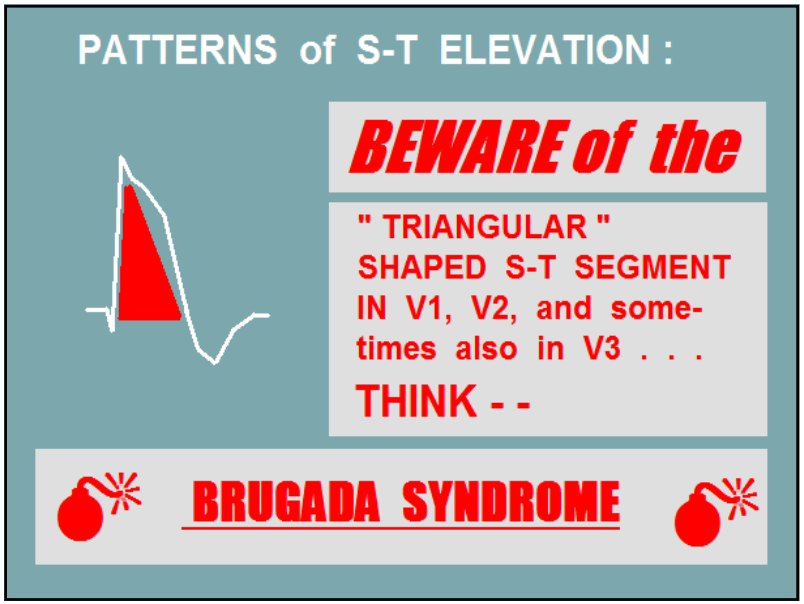

CIRCUMFLEX ARTERY OCCLUDED with significant THROMBUS.
PTCA of CIRCUMFLEX ARTERY.
DOMINANT CIRCUMFLEX ARTERY OPEN POST THROMBECTOMY with STENT DEPLOYMENT.
LVEF = 43%

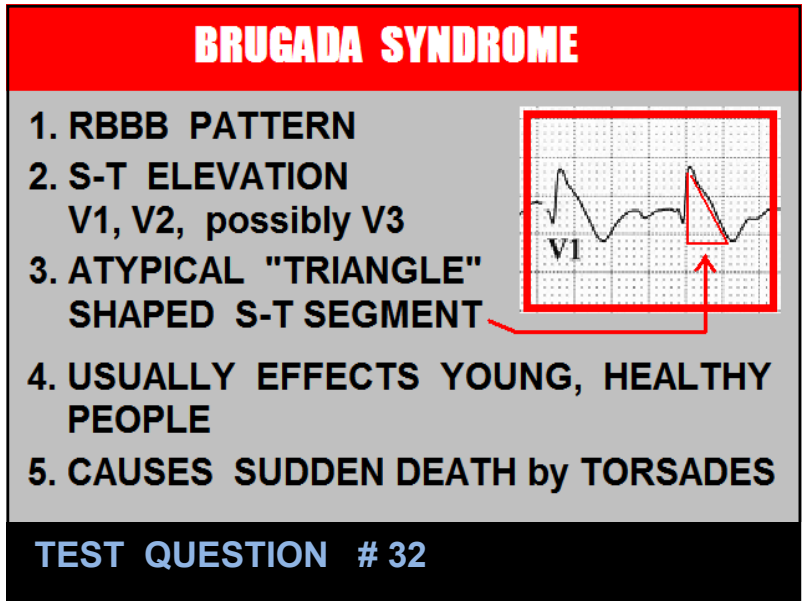

CASE STUDY SUMMARY
ST ELEVATION: II, III, aVF, V5, V6
ST DEPRESSION: V1 - V3, POSSIBLY I and aVL
SUSPECTED DIAGNOSIS: ACUTE INFERIOR - POSTERIOR - LATERAL MI
SUSPECTED "CULPRIT ARTERY" (if applicable):
OCCLUSION of DOMINANT CIRCUMFLEX ARTERY
IMMEDIATE CONCERNS FOR ALL STEMI PATIENTS:
BE PREPARED TO MANAGE 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 DIEURETICS EMERGENCY SURGERY

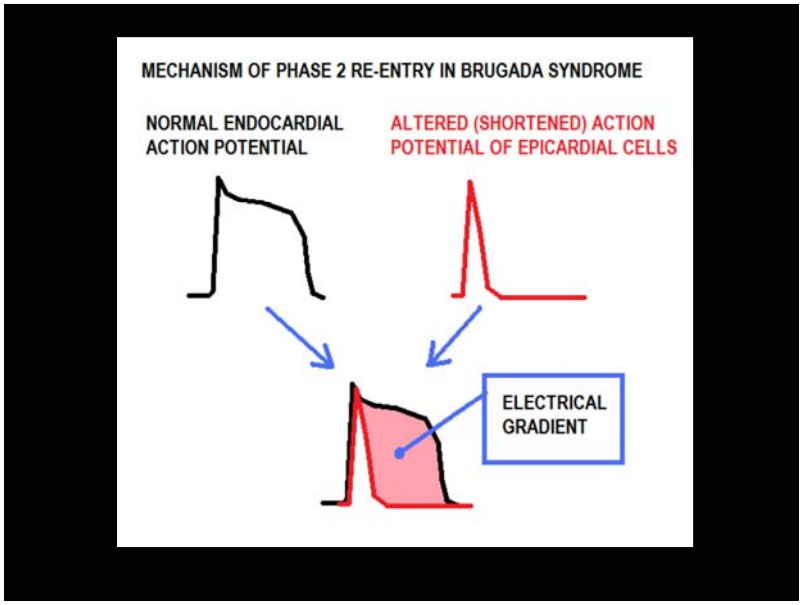

BRUGADA SYNDROME and Other Infarction Mimics
CASE STUDY 18 -- BRUGADA SYNDROME
CHIEF COMPLAINT and SIGNIFICANT HISTORY:
37 y/o FEMALE patient arrives via EMS after being involved in a low speed motor vehicle accident. Per EMS crew, patient was the driver and sole occupant of a car that struck a tree. Patient does not recall accident. Upon further questioning, patient admits to other episodes of syncope and near-syncope. Patient denies feeling any chest pain / pressure or shortness of breath. She states she "felt great" today, until just before the the accident, when she "suddenly felt lightheaded and must have blacked-out."
RISK FACTOR PROFILE:
FAMILY HISTORY: MATERNAL AUNT DIED AT AGE 31, UNEXPECTEDLY. WAS RULED AS A "HEART ATTACK." THERE WAS NO PRIOR KNOWN HISTORY OF CAD.
PHYSICAL EXAM: Pt. CAO x 3, skin warm, dry, color normal. Abrasions /contusions on face (airbag deployment). Patient appears to be in excellent physical condition, states she exercises several times per week (aerobics, weight training, swimming).
VITAL SIGNS: BP: 112/66, P: , R: 20, SAO2: 100% on room air.
LABS: TROPONIN: < .04 BMP and CBC: all values within normal limits.


IS THERE ANYTHING ABNORMAL WITH THIS EKG ?
37 yr Female Caucasian
Vent. rate 62 BPM
PR interval 180 ms
QRS duration 88 ms
QT/QTc 418/424 ms
P-R-T axes 37 22 47
Normal sinus rhythm
Normal ECG
No previous ECGs available
Technician:
Referred by:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1

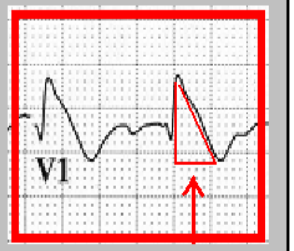

37 yr Female Caucasian
Vent. rate 62 BPM
PR interval 180 ms
QRS duration 88 ms
QT/QTc 418/424 ms
P-R-T axes 37 22 47
Normal sinus rhythm
Normal ECG
No previous ECGs available
NOTE COMPUTER INTERPRETATION !
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
THIS PATIENT EXHIBITS A "CLASSIC" TYPE I BRUGADA SYNDROME 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 some-times also in V3 . . .
THINK - -
BRUGADA SYNDROME


BRUGADA SYNDROME
1. RBBB PATTERN
2. S-T ELEVATION V1, V2, possibly V3
3. ATYPICAL "TRIANGLE" SHAPED S-T SEGMENT
V1
4. USUALLY EFFECTS YOUNG, HEALTHY PEOPLE
5. CAUSES SUDDEN DEATH by TORSADES
TEST QUESTION # 32

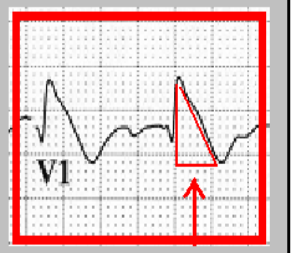


BRUGADA SYNDROME



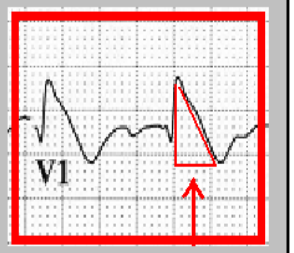
- GENETIC DISORDER - GENE SCN5A, which encodes CARDIAC SODIUM CHANNELS.
- CAUSES EARLY RIGHT VENTRICULAR SUB-EPICARDIAL REPOLARIZATION
- CAUSES RUNS OF TORSADES de POINTES, and SUDDEN DEATH from TORSADES and V-FIB.
- IS BELIEVED TO CAUSE 4 - 12 % of ALL SUDDEN DEATHS, and 50 % of ALL CARDIAC DEATHS where pt. has a STRUCTUALLY NORMAL HEART.

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.

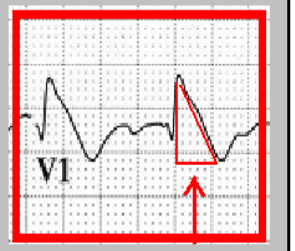
BRUGADA SYNDROME



- YOUNG MALES of SOUTHEAST ASIAN DESCENT are in HIGH RISK GROUP, however this disorder affects ANY RACE or GENDER.
- BRUGADA SYNDROME is HEREDITARY.
- SUSPECT BRUGADA SYNDROME in patients with FAMILY HISTORY of BRUGADA / SUDDEN DEATH, and/or TORSADES.

BRUGADA SYNDROME - TESTING

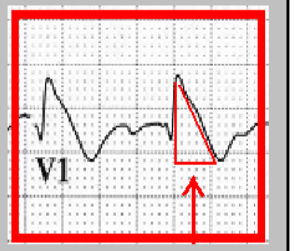
- For CONCEALED cases, a drug study of AJMALINE, FLECAINIDE, or PROCAINAMIDE can UNMASK the "tell-tale" TRIANGULAR COMPLEXES of V1 and V2.



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

BRUGADA SYNDROME - TREATMENT

ICD implantation is the only known effective treatment to date.



www.BRUGADA.org

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