

WAYNE W RUPPERT, CVT, CCCC, NREMT-P

Cardiovascular Coordinator & Emergency Manager
Bayfront Health Seven Rivers
Crystal River, FL

Interventional Cardiovascular & Electrophysiology Lab Technologist

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# PRE-REQUISITES:

#### **BASIC ECG INTERPRETATION SKILLS:**

- This is NOT a Basic ECC Rhythms course.
- If you're in this class, you should already know your basic rhythms.

#### **AM Session CONTENTS:**

- Introduction and The ECG in Perspective
- Essential Cardiac A & P
  - Cellular (nerve, muscle, connective)
  - Structural (chambers / valves / Fibrous Skeleton)
- Heart Sounds
  - Acute Mitral Regurgitation

#### **AM Session CONTENTS, continued:**

- Wolff-Parkinson-White Syndrome
- ECG Principles
- Coronary Artery Anatomy and Correlation with the 12 Lead ECG
- Waveforms and Intervals
- Bundle Branch Blocks
- Axis Deviation and Rotation

#### **PM Session CONTENTS:**

- Sudden Cardiac Death Syndromes
  - Long QT
  - Hypertrophic Cardiomyopathy
  - Arrythmogenic Right Ventricular Cardiomyopathy
  - Brugada Syndrome
- Acute Coronary Syndromes
   With Cath Lab Case Studies

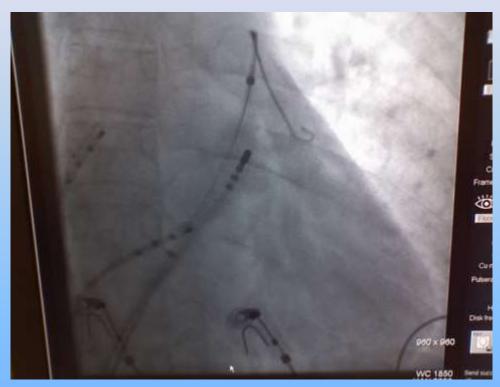
# WHO are U ???

# Wayne Ruppert - Bio:

- Cardiovascular Coordinator 2012-present (coordinated 4 successful accreditations)
- Interventional Cardiovascular / Electrophysiology
   Technologist, 1995-Present. (Approx 13,000 patients)
- Author of: "12 Lead ECG Interpretation in Acute Coronary Syndrome with Case Studies from the Cardiac Cath Lab," 2010, TriGen publishing / Ingram Books
- Author of: "STEMI Assistant," 2014, TriGen publishing / Ingram Books
- Florida Nursing CE Provider # 50-12998
- 12 Lead ECG Instructor, 1994-present (multiple hospitals, USF College of Medicine 1994)
- Website: www.ECGtraining.org



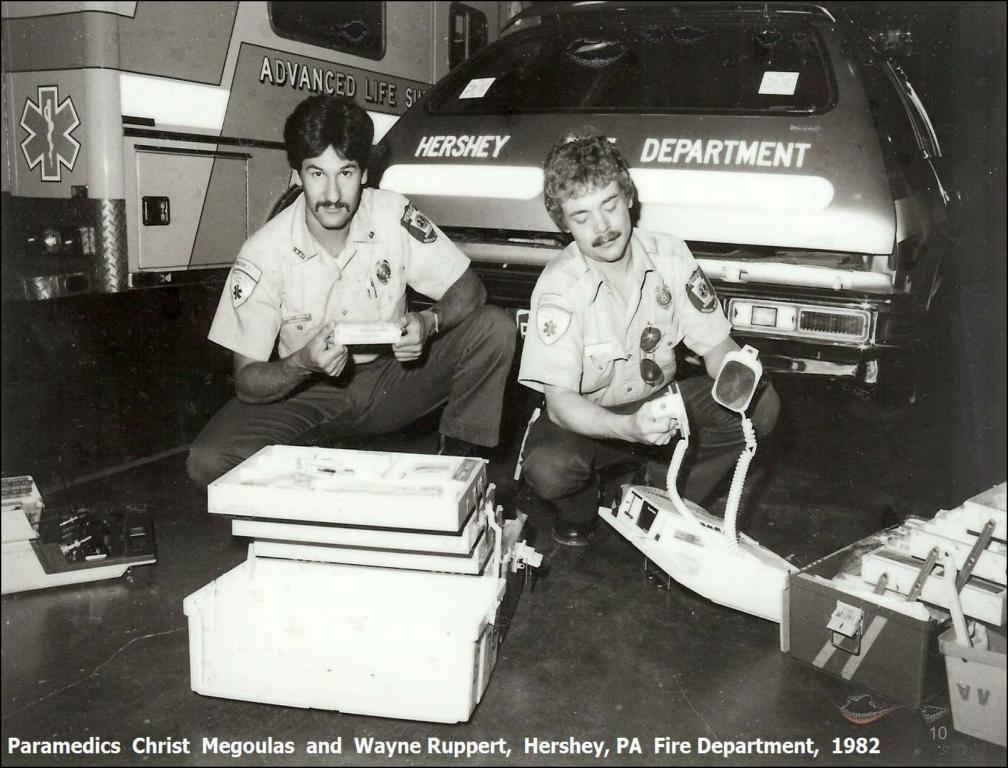
# **Electrophysiology Lab Case Studies**





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 Pacemapping during EP study at the St Joseph's Hospital Heart Institute, Pediatric Electrophysiology Program, Tampa, FL in 2004



In the CARDIAC CATHETERIZATION LAB, we read our patients' 12 Lead ECGs and then evaluate their coronary arteries and ventricular function during angiography. Stated in plain English, we rapidly learn how to correlate 12 lead ECG findings with what's really going on inside our patients' bearts. Seeing ECGs from this perspective adds a new dimension to understanding the complex pathophysiologies of cardiov accular disease.

#### This book prepares you to:

- INTERPRET 12 Lead ECGs.
- ASSIMILATE DATA derived from the 12 Lead ECG into a comprehensive patient evaluation process designed to maximize disapnostic accuracy, while taking into consideration the 12 Lead ECGs inherent LACK of SENSITIVITY and SPECIFICITY.
- IDENTIFY 13 PATTERNS associated with myocardial ischemia and infarction, including the most subtle ECG changes often missed by clinicians and the ECG machine's computerized interpretation software.
- CORRELATE each lead of the ECG with specific regions of the heart and the CORONARY ARTERIAL
  DISTRIBUTION that commonly supplies it. In cases of STEMI, this knowledge prepares you to
  ANTICIPATE the FAILURE OF CRITICAL CARDIAG STRUCTURES often BEFORE THEY FAIL.

For those who need to master essential material quickly, this book has been written with an expedited learning feature, designed to make learning as easy as 1 - 2 - 3:

- 1. READ the YELLOW HIGHLIGHTED TEXT
- 2. STUDY the GRAPHIC IMAGES, PICTURES and ECGs
- 3. CORRECTLY ANSWER the REVIEW QUESTIONS at the end of each section.

This is an invaluable resource for every medical professional who evaluates patients and much their 12 lead ECOs:

- Follows in Emergency, Cardiology, and Family Medicine
- Medical Residents
- Veterar Physicians wenting a good review in ACS patient evaluation
- Physician Assistants and Marse Practitioners
- Environncy Department Nurses
- \* Coronary Care Unit and Cardiac Telemetry Murses
- Work-in Clinic Physicians and Norses
- · Paramedics

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

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

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

Richmond Virginia

"This book integrates academic ECG principles with real-world clinical practice by incorporation of well chosen cath lab case studies into its curriculum. This combination lets readers see patients and their ECGs through the eyes of an experienced cath lab interventionalist, and provides a balanced approach to patient evaluation that compensates for the ECGs inherent lack of sensitivity and specificity. I highly recommend this book for all Emergency Medicine and Cardiology Fellows. For experienced clinicians, it's a superb review."

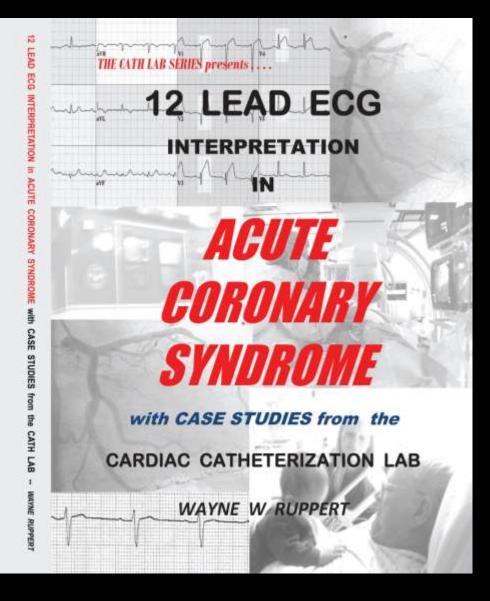
#### Humberto Coto, MD, FACP, FACC

- Chief of Interventional Cardiology

St. Joseph's Hospital

Tampa, Florida



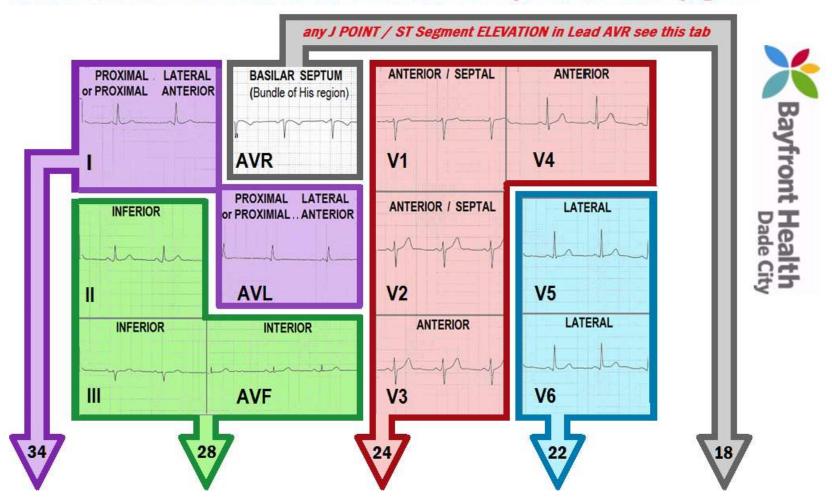


<u>www.TriGenPress.com</u> www.ECGtraining.org <u>BarnesandNoble.com</u> <u>Amazon.com</u> CRASH CART EMERGENCY REFERENCE

by Wayne Ruppert

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

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



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

Wayne.ruppert@bayfronthealth.com

#### **BASIS:**

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

# www.ECGtraining.org www.practicalclinicalskills.com

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12 LEAD ECG IN ACS

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

<u>Download Initial Stabilization of the Atrial Fib Patient - SCPC 19th Congress</u>

**Download Continuous ST-Segment Monitoring Policy** 

Download QTc Monitoring Policy for Patients on QT Prolonging Meds

<u>Download QT Monitoring Protocol - Patients on QT Prolonging Meds - 2018</u>

Download A-Fib / Flutter ER Physician's Order Set - BHDC

Download A-Fib / Flutter Flowchart Emerg Care BHDC

Download Team Driven Performance Improvement - SCPC 19th Congress

**Download TDPI in Ambulance Industry Journal** 

**Download TJC Sentinel Event Alert - Disruptive Physicians** 

**Download ACLS 2015 Algorithm Cheat Sheets** 

Download 2015 ACLS Algorithms with ECG examples

Download Basic ECG Course Handout - 4 slides per page



#### Then Select:

The Lifesaving 12 Lead ECG - Part 1
The Lifesaving 12 Lead ECG - Part 2

#### The EKG in PERSPECTIVE

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



# 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 )
```

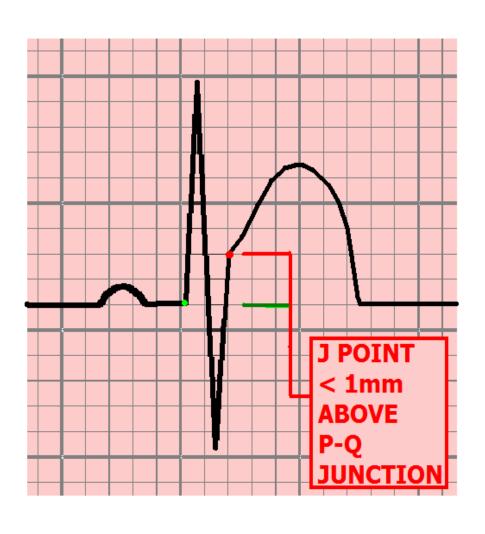
**↓** SPECIFICITY

( FALSE POSITIVES )

AND . . .

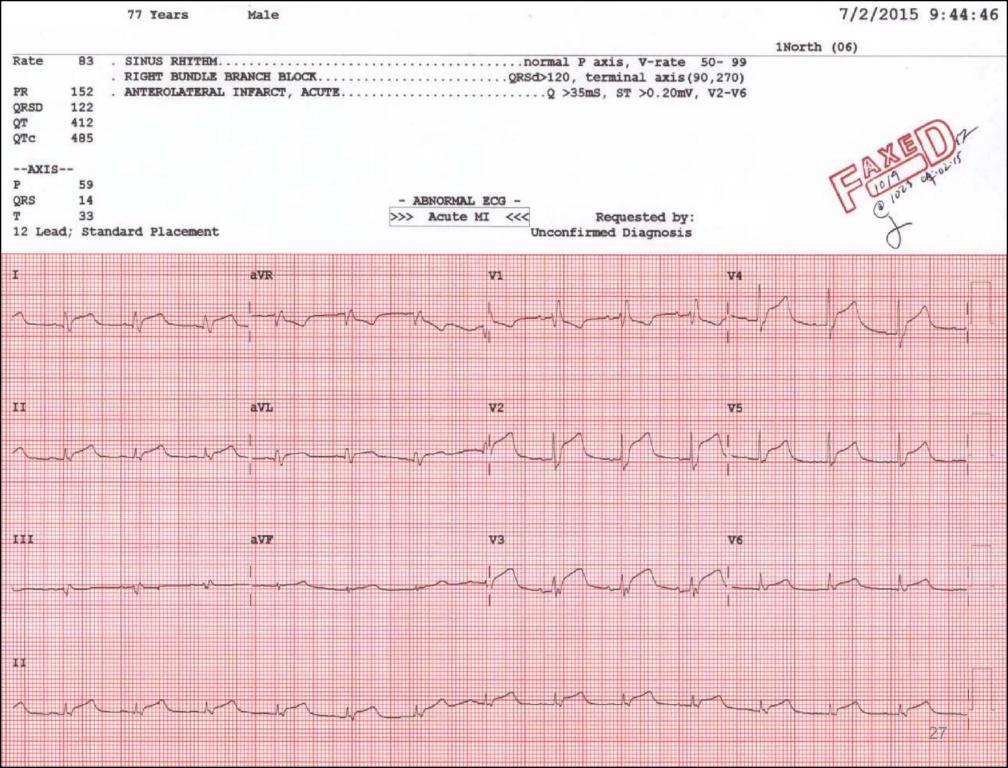
#### PROBLEMS WITH SPECIFICITY . . .

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



#### CONDITION:

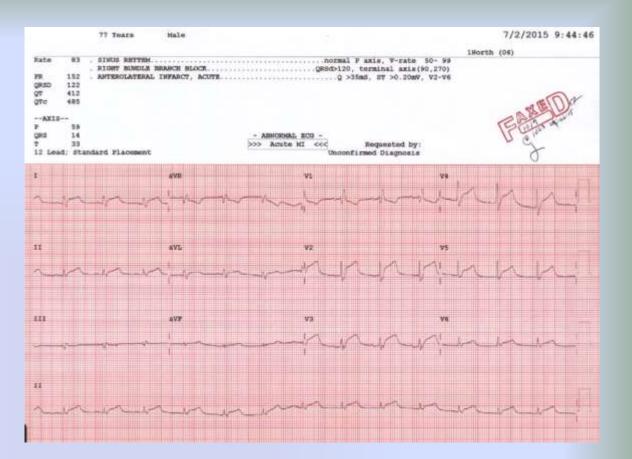
- ACUTE INFARCTION
- HYPERKALEMIA
- BRUGADA SYNDROME
- PULMONARY EMBOLUS
- INTRACRANIAL BLEED
- MYOCARDITIS / PERICARDITIS
- L. VENT. HYPERTROPHY
- PRINZMETAL'S ANGINA
- L. BUNDLE BRANCH BLOCK
- PACED RHYTHM
- EARLY REPOLARIZATION & "MALE PATTERN" S-T ELEV.



#### **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





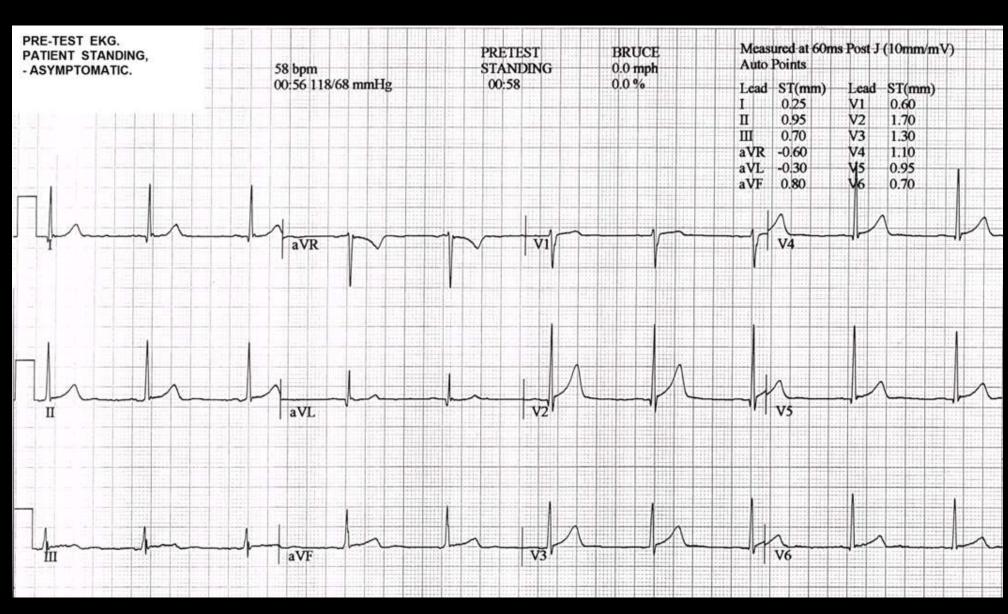


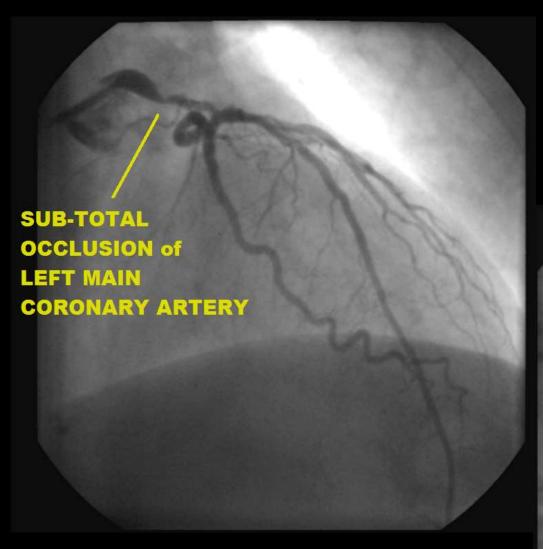
. . . but MASKS serious, LIFE - THREATENING CONDITIONS.

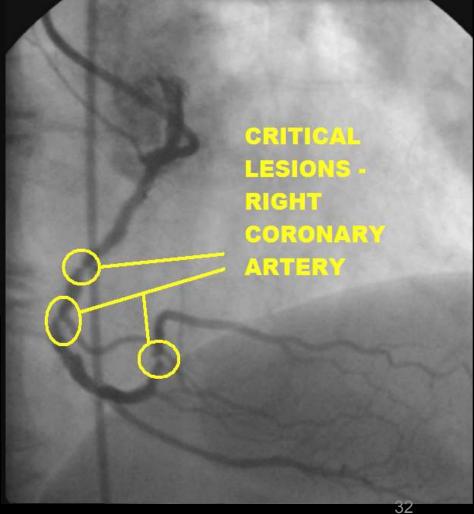


that is why <u>YOU</u> must do a THOROUGH PATIENT EVALUATION . . . and have a HIGH INDEX OF SUSPICION!!!





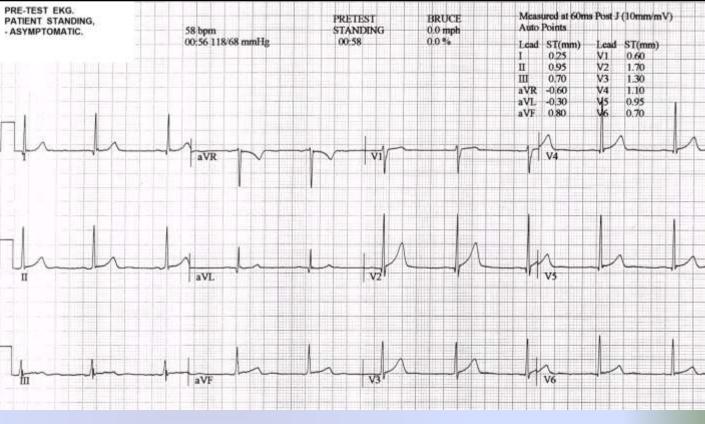


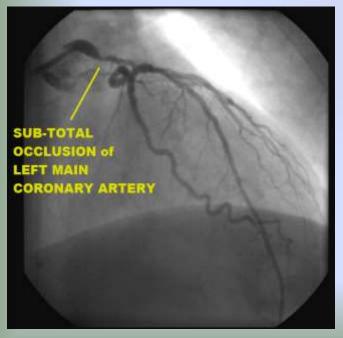


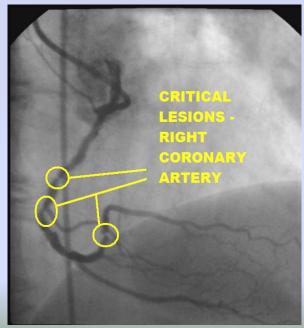
# PROBLEMS WITH SENSITIVITY . . .

# NORMAL ECG.

But . . . . .





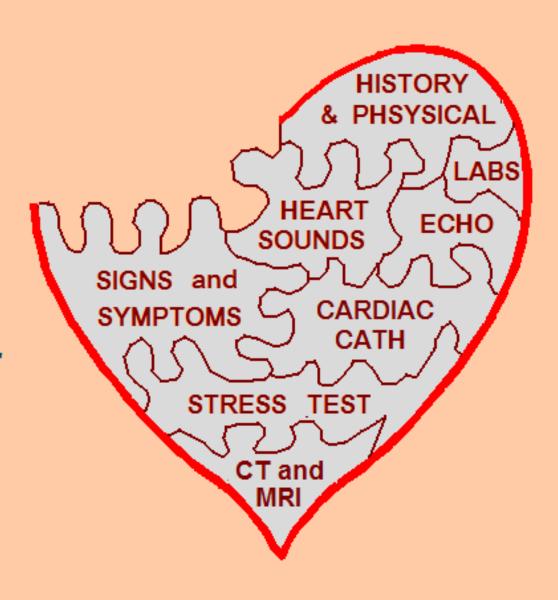


LETHAL
TRIPLE
VESSEL
DISEASE

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



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



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

Lack of Specificity,

The 12 Lead ECG remains

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

 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.





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

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

Hypercholesterolemia
Hypertension
Diabetes Mellitus

Cigarette smoking Positive family history Obesity

## C-Statistic scores achieved in this study:

**HEART: 0.83** 

TIMI: 0.75

**GRACE: 0.70** 

## **C-Statistic interpretation:**

A score of "1.00" would mean the score predicts outcome with 100% perfection. A score of 0.50 is the same as a "50/50 coin toss." A score of LESS THAN 0.50 means that the score predicts the opposite outcome.

### US HEART Score Validation

 1,070 observation unit patients at Wake Forest

Out performed clinician gestalt!

Mahler et. al, Crit Path Cardiol, 2011

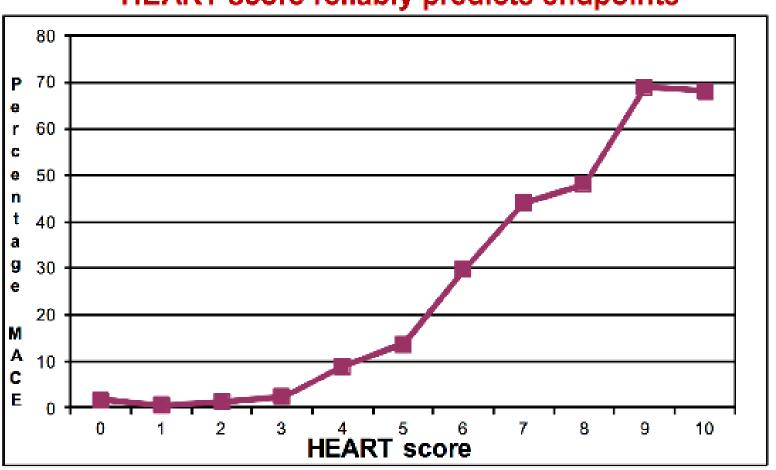
Mahler et. al, Int J Cardiol, 2013

# The HEART Score:

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**

#### **HEART** score reliably predicts endpoints

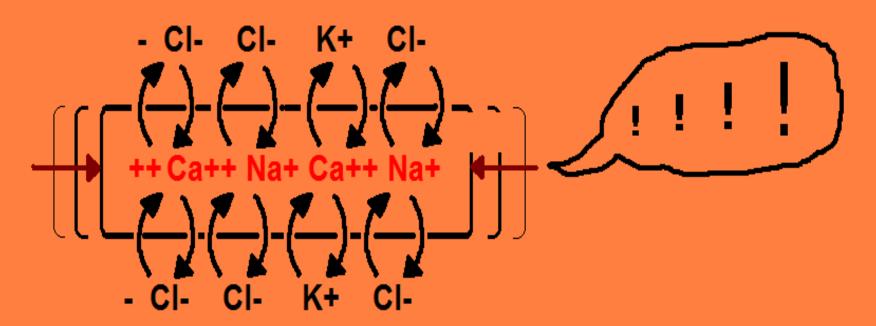


# Cardiac A & P

CARDIAC CELLS AT REST have POSITIVE charged IONS on the OUTSIDE of the cell membrane, and NEGATIVE charged IONS on the INSIDE



... when the IONS shift ... that is, the POSITIVE IONS that were on the outside TRADE PLACES with the NEGATIVE IONS that were on the INSIDE ....



... THE CELL CONTRACTS!

# THIS (OF COURSE) IS KNOW AS ...

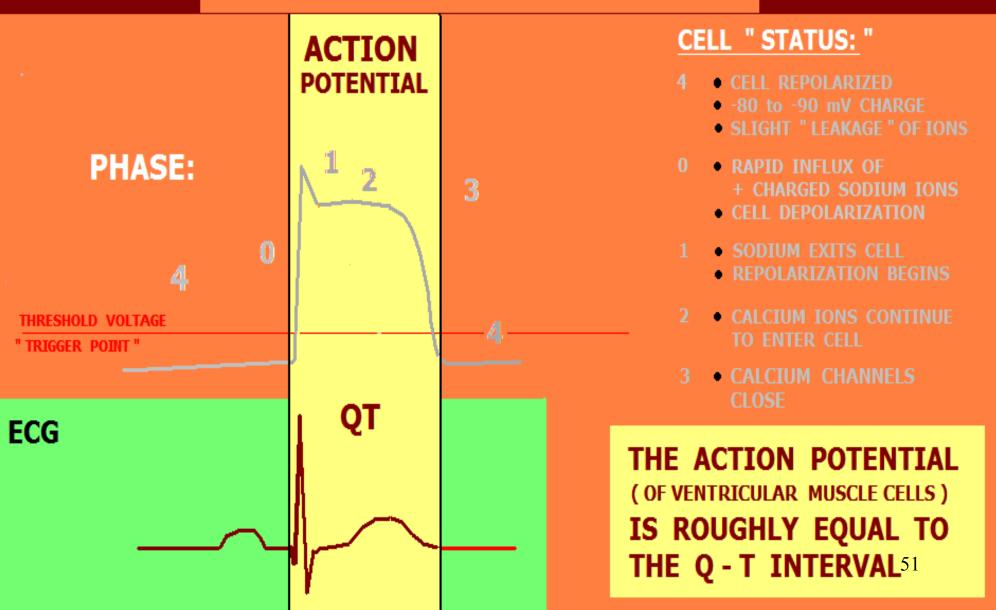
# **DEPOLARIZATION**

WHEN EVERYTHING IS WORKING PROPERLY, THE WAVE OF DEPOLARIZING CELLS CAUSES THE HEART TO CONTRACT, AND PUMP BLOOD TO THE LUNGS AND THE SYSTEMIC CIRCULATION

AFTER DEPOLARIZATION, THE CELLS RELAX.

THE IONS RETURN TO THEIR ORIGINAL POSITIONS -THIS PROCESS IS KNOWN AS **REPOLARIZATION** 

#### VENTRICULAR MUSCLE CELL ACTION POTENTIAL

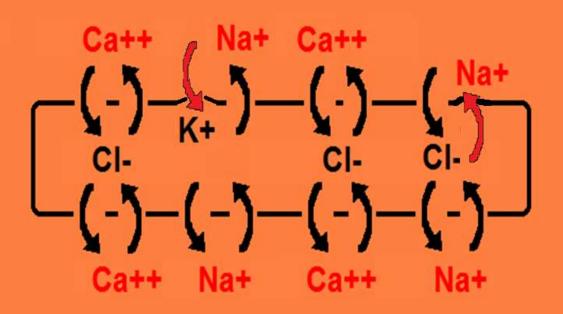


When ION CHANNELS are MALFORMED, the abnormal channel shape may DELAY the transfer of IONS....

.... this can DELAY REPOLARIZATION, which will show on the ECG as "QT Prolongation"

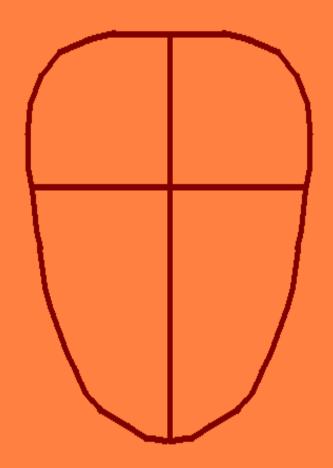
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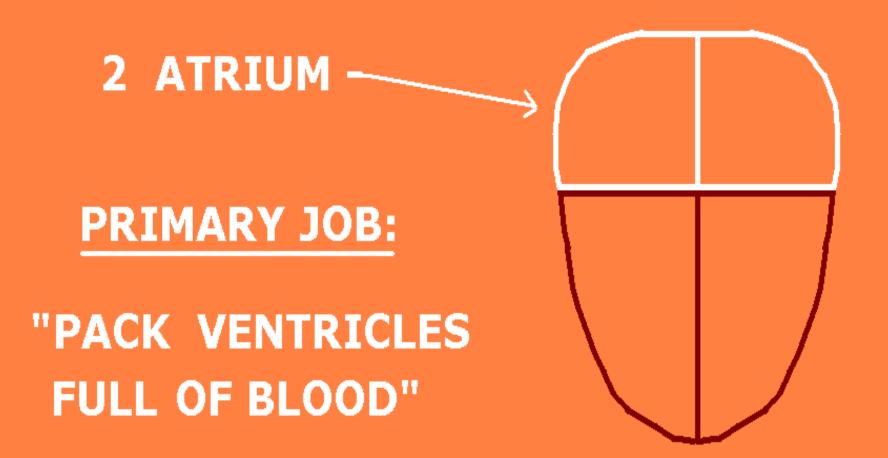


to LETHAL
DYSRHYTHMIAS
such as
Torsades de
Pointes,
CARDIAC ARREST
and SUDDEN
DEATH.

## FOUR CHAMBERED PUMP



## FOUR CHAMBERED PUMP...

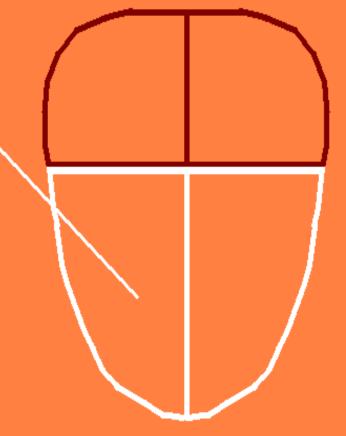


## FOUR CHAMBERED PUMP...

2 VENTRICLES

PRIMARY JOB:

"PUMP BLOOD TO THE LUNGS AND THE REST OF THE BODY"



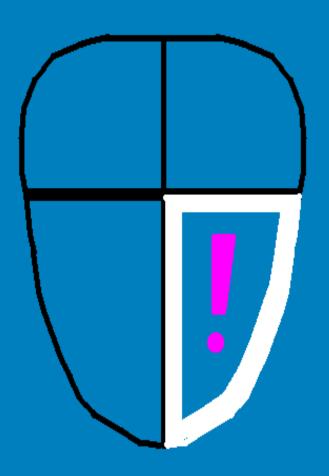
# THE CHAMBER MOST IMPORTANT TO KEEPING THE PATIENT ALIVE

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

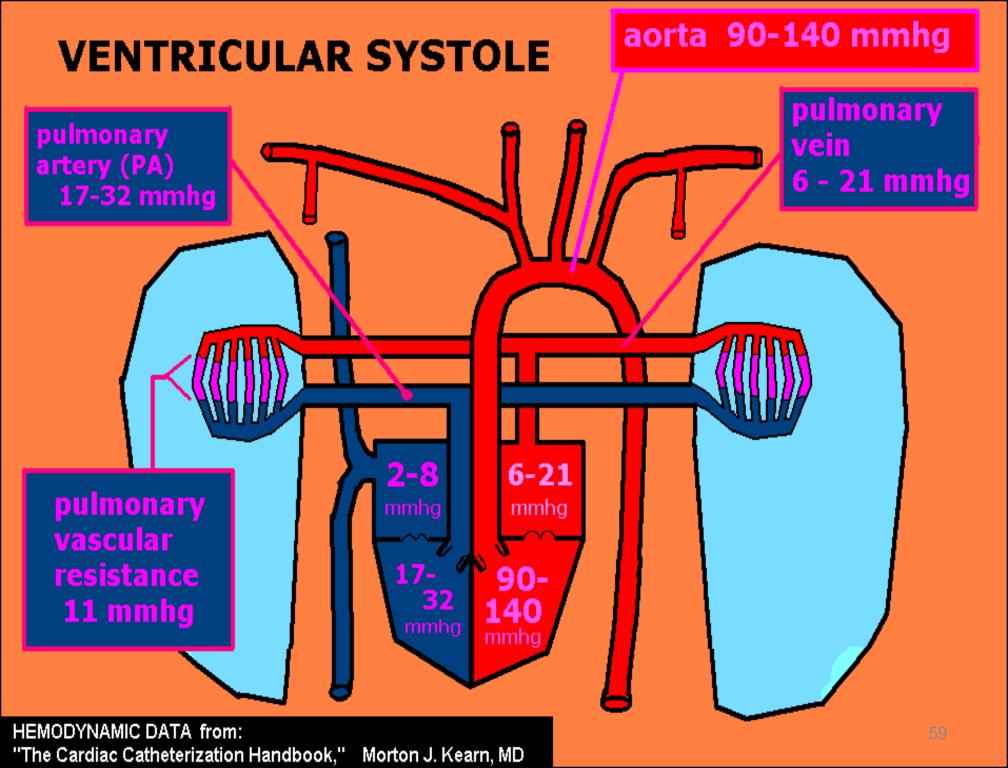
IS THE

LEFT VENTRICLE

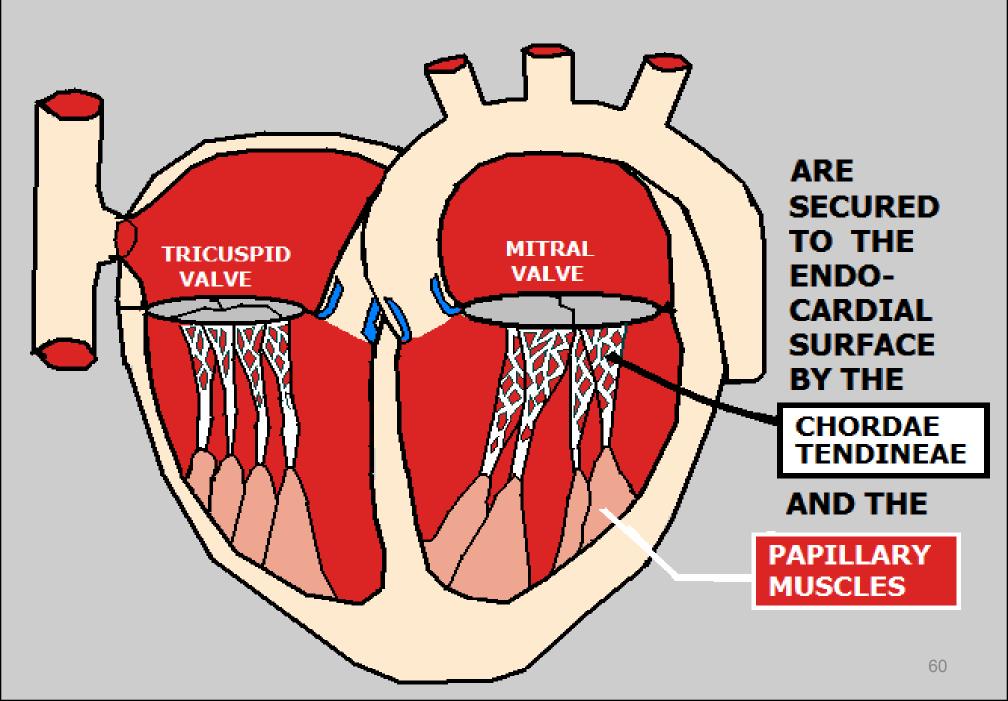
WHICH WE WILL REFER TO AS THE PUMP



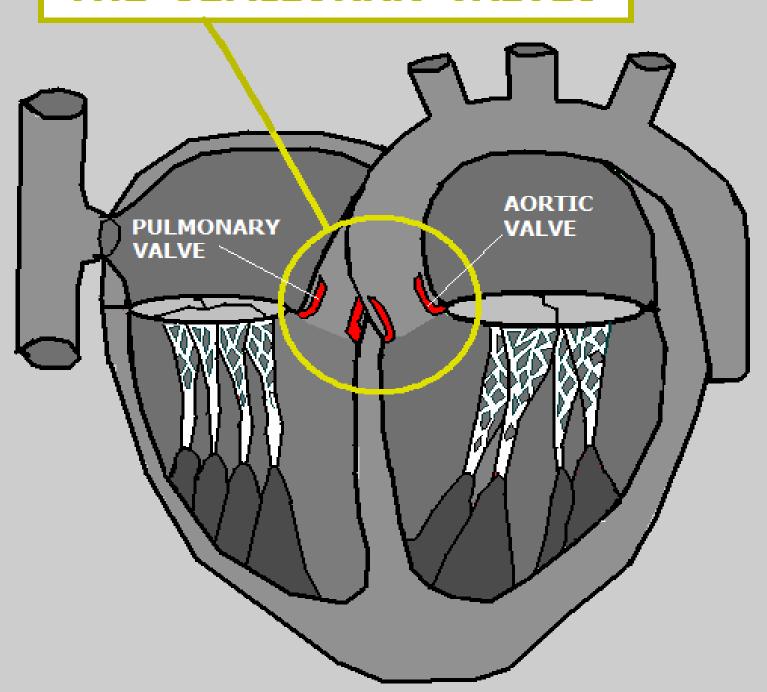
## aorta DIASTOLE 60 - 90 mmhg pulmonary pulmonary vein artery (PA) 4 - 13 mmhg 5-16 mmhg 4-13 1-5 mmhg pulmonary 4 - 13 capillary mmhg wedge 4 - 13 mmhg



## ATRIO-VENTRICULAR VALVES



## THE SEMILUNAR VALVES





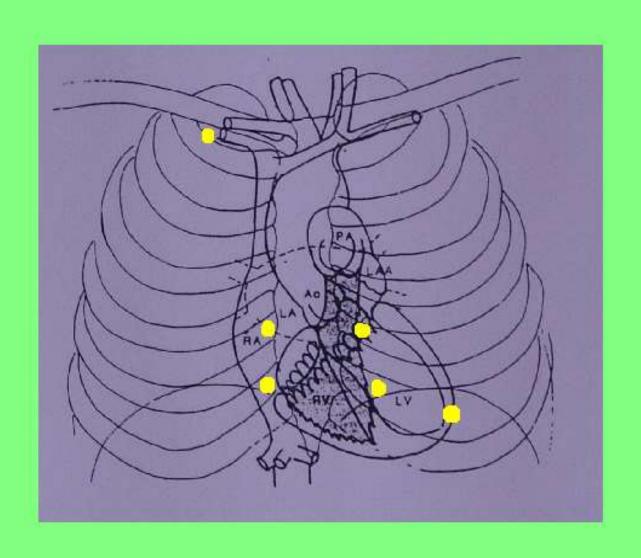
## BASIC HEART SOUNDS ASSESSMENT

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

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









## BASIC HEART SOUNDS ASSESSMENT

- NormalHeartSounds
- Murmurs
  - systolic
  - diastolic
- FrictionRubs



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

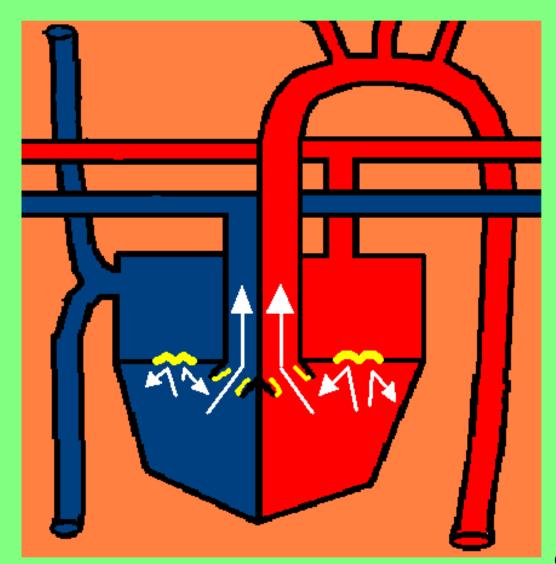
HEART SOUNDS ARE GENERATED BY THE SOUND OF THE HEART VALVES <u>CLOSING</u>.

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

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

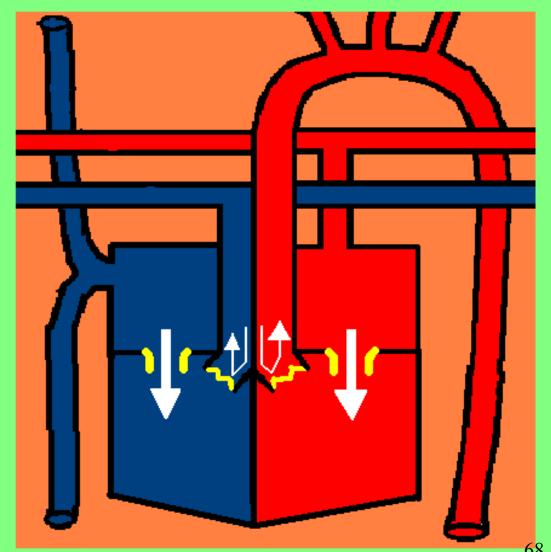
S-1 BEGINNING OF SYSTOLE.

SOUND OF THE MITRAL AND TRICUSPID VALVES CLOSING.



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

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



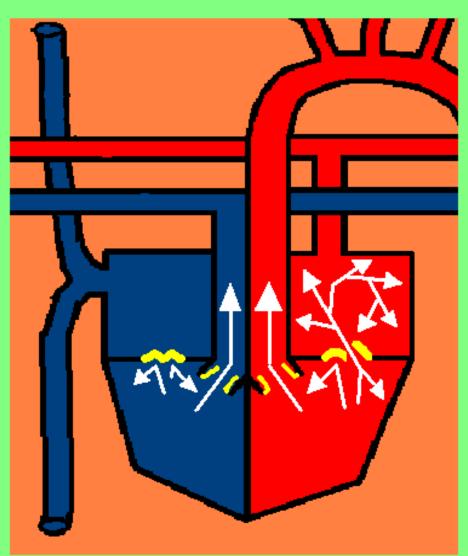


# VERY BASIC HEART SOUNDS ASSESSMENT

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

# CAUSE OF SYSTOLIC (S 1) MURMUR

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



## BASIC HEART SOUNDS ASSESSMENT

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







MOST SYSTOLIC MURMURS CAUSED BY MITRAL VALVE FAILURE.

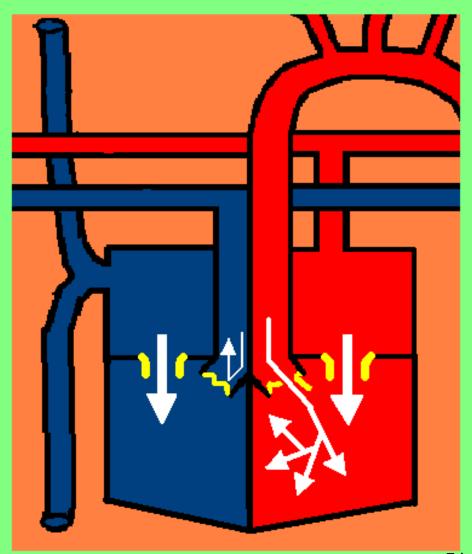
ACUTE MITRAL VALVE
REGURGITATION IS A
POTENTIALLY LETHAL
COMPLICATION OF
ACUTE / RECENT
EXTENSIVE TRANSMURAL MI

MOST SYSTOLIC MURMURS CAUSED BY MITRAL VALVE FAILURE.

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

# CAUSE OF DIASTOLIC (S2) MURMUR

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



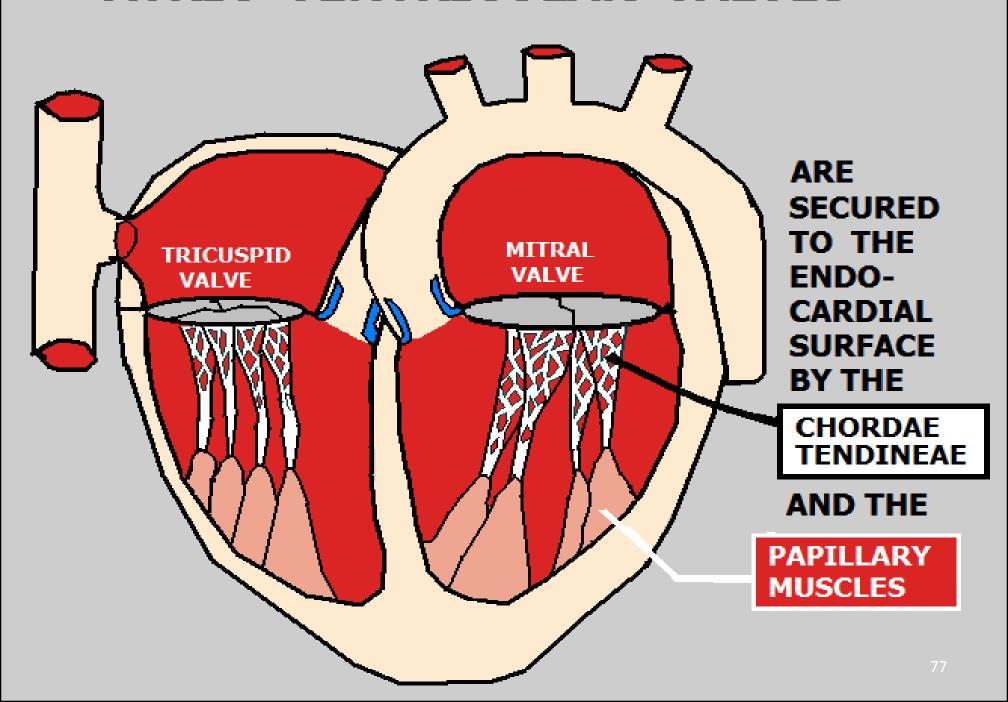
Chronic Valvular REGURGITATION (Leaky Valve) leads to elevated heart chamber pressures and HYPERTROPHY.

Chronic Valvular STENOSIS ("Creaky" Valve) leads to Cardiac Muscle STRAIN and HYPERTROPHY.

**ACUTE Mitral Valve** REGURGITATION can be caused by **EXTENSIVE** "Multi-Site" Myocardial Infarction and Necrosis – which results in PAPILLARY MUSCLE **NECROSIS** and **PAPILLARY** MUSCLE TEAR.

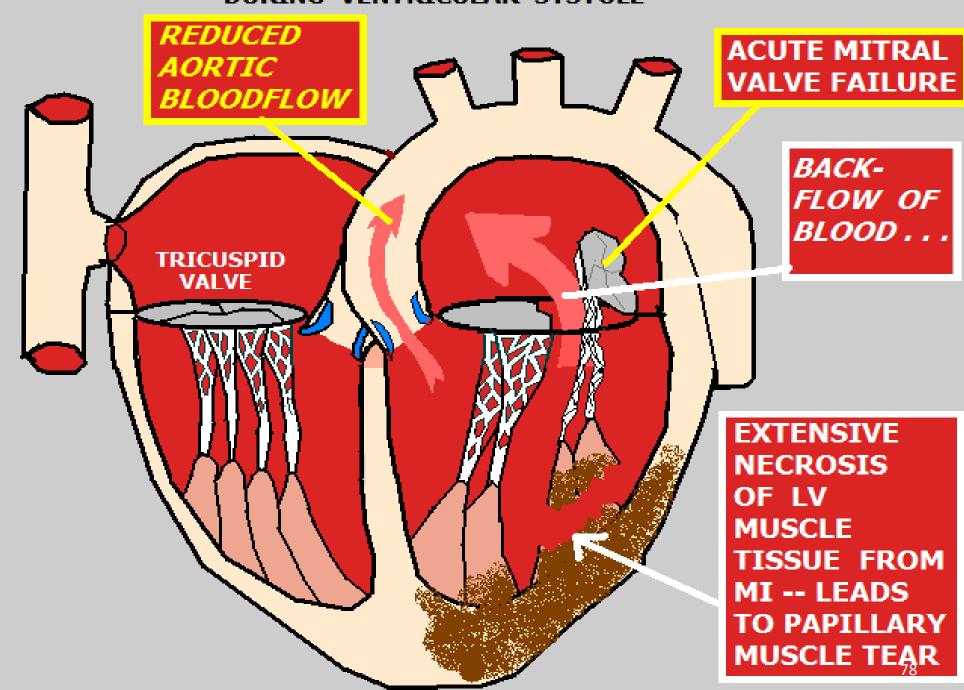
Papillary muscles are attached to "multiple surfaces" . . . . .

## ATRIO-VENTRICULAR VALVES



### **ACUTE MITRAL REGURGITATION**

DURING VENTRICULAR SYSTOLE



# Symptoms of Acute Mitral Regurgitation::

- SHOCK
- PROFOUND HYPTENSION
- PINK, FROTHY SPUTUM, PULMONARY EDEMA
- SYSTOLIC (S1) MURMUR

```
"SWOOSH – DUB.....SWOOSH – DUB
.....SWOOSH – DUB"
```

## BASIC HEART SOUNDS ASSESSMENT

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



#### S-2 MURMUR SOUNDS LIKE:

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

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

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

## BASIC HEART SOUNDS ASSESSMENT

## FRICTION RUB

- ASSOCIATED WITH PERICARDITIS
- SOUNDS LIKE THE GENTLE RUBBING OF SANDPAPER



HAS 3 COMPONENTS: SYSTOLIC, EARLY, and LATE DIASTOLIC

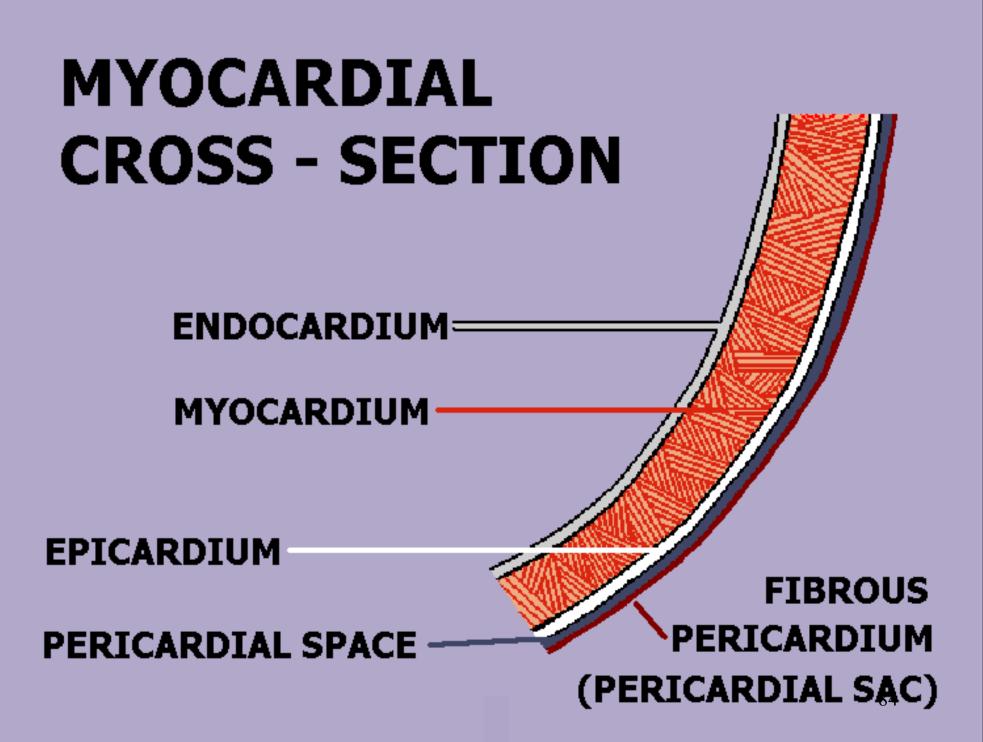
## BASIC HEART SOUNDS ASSESSMENT

## FRICTION RUB

□ IS PRESENT IN MOST ACUTE TRANSMURAL MI PATIENTS



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



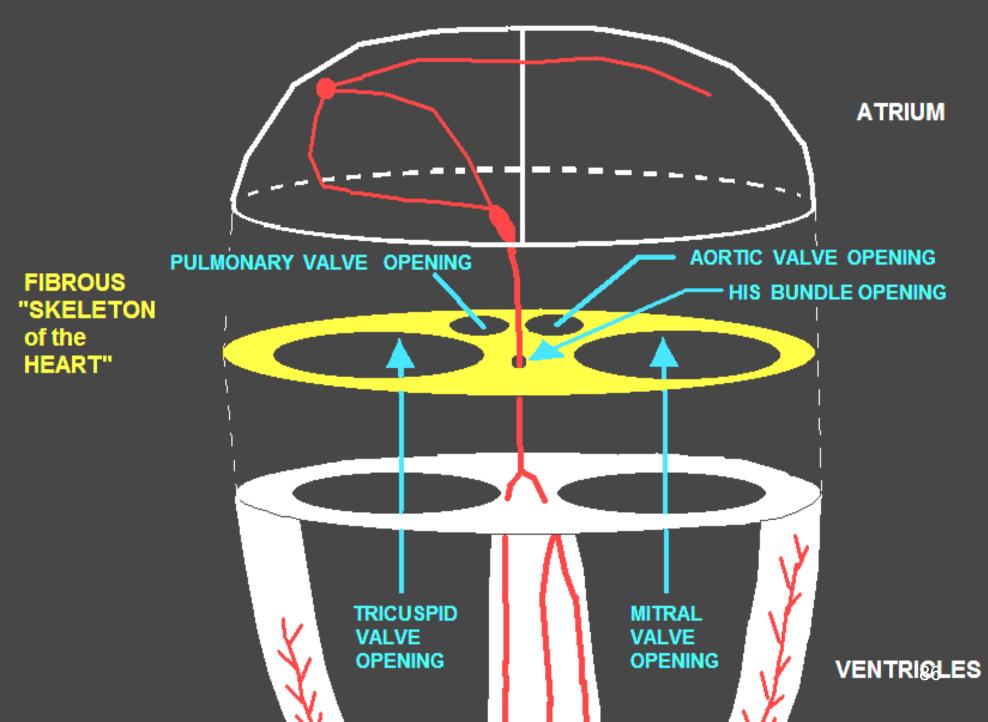
# NORMAL AMOUNT OF

FLUID IN

PERICARDIAL SPACE =

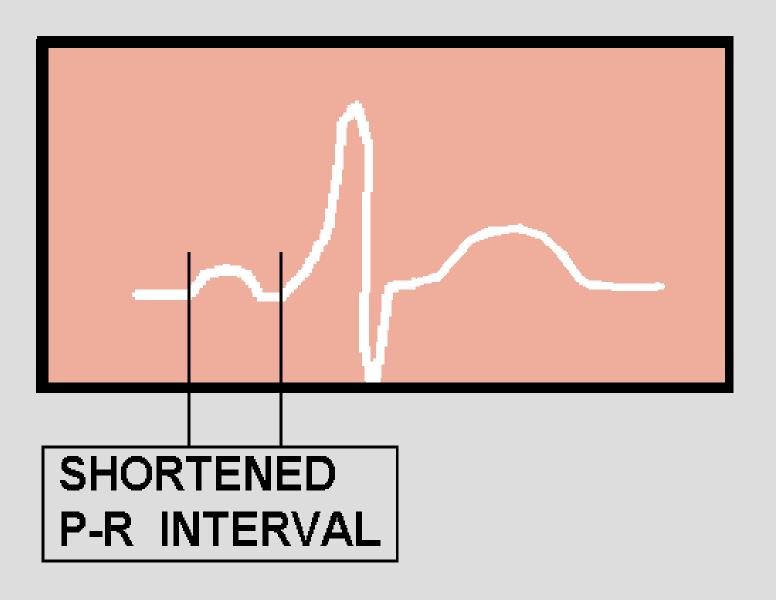
20 - 50 cc

### THE "SKELETON OF THE HEART"



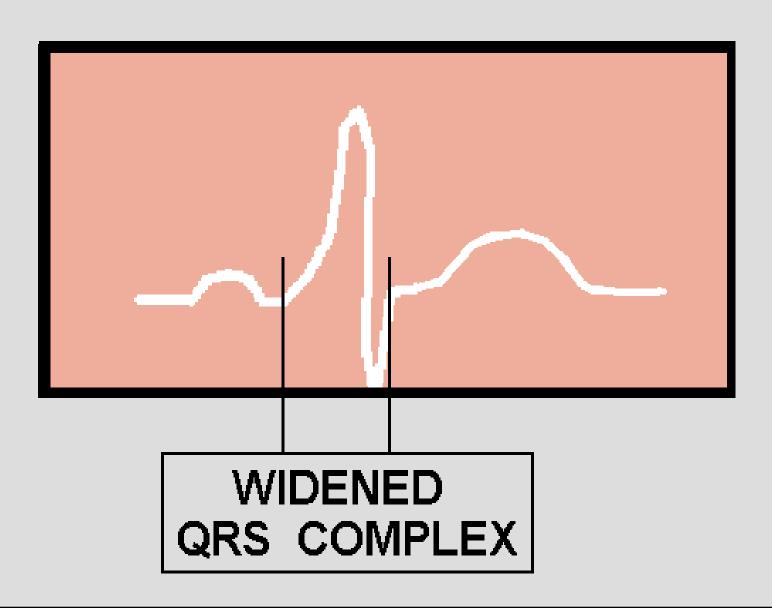
# **WOLFF-PARKINSON-WHITE**

## **EKG CHARACTERISTICS**



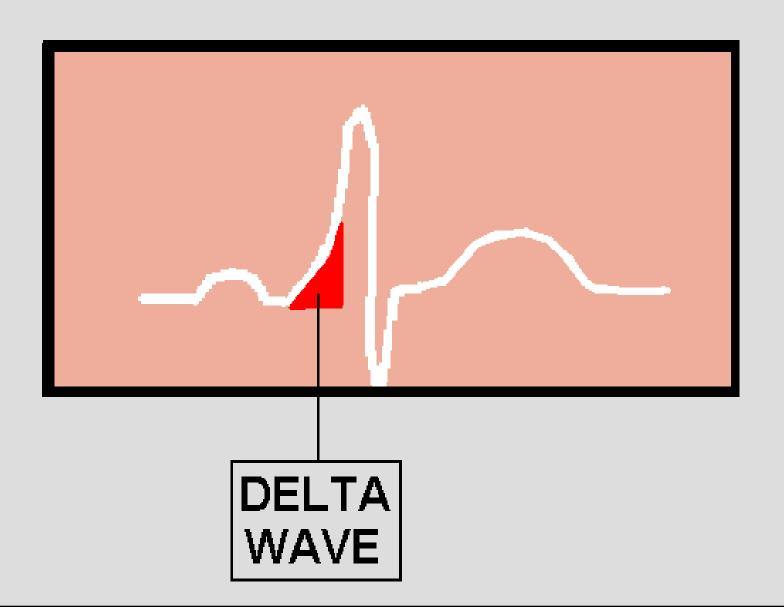
# **WOLFF-PARKINSON-WHITE**

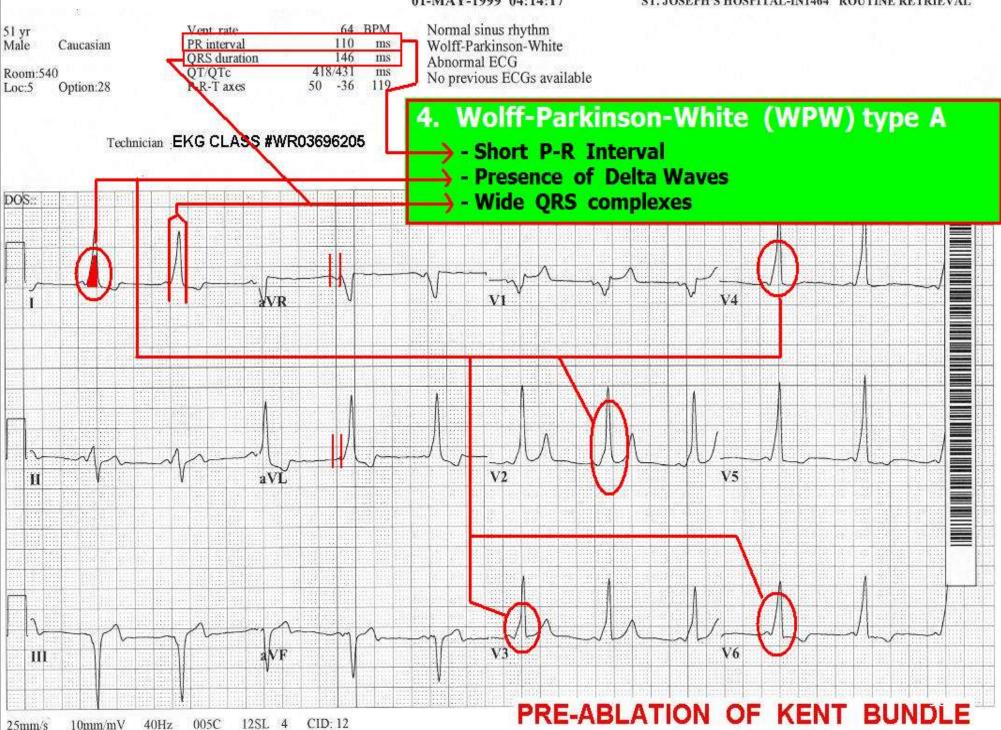
## EKG CHARACTERISTICS

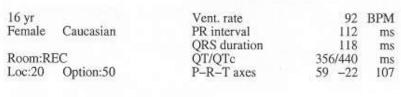


# **WOLFF-PARKINSON-WHITE**

## EKG CHARACTERISTICS

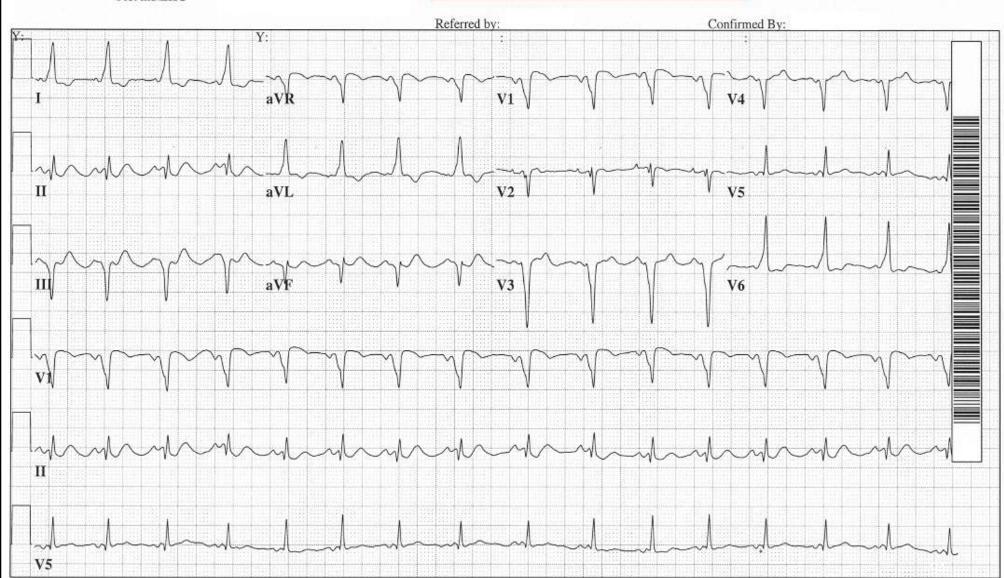






History: Unknown EKG CLASS #WR030100 Technician: DP 60783 Test ind:EKG

Normal sinus rhythm with sinus arrhythmia Left atrial enlargement Anterior infarct, age undetermined Inferior infarct, age undetermined ST & T wave abnormality, consider lateral ischemia Wolff-Parkinson-White Abnormal ECG No previous ECGs available



16 yr Vent. rate 92 BPM Female Caucasian PR interval 112 ms **ORS** duration 118 ms Room:REC QT/QTc 356/440 ms Loc:20 Option:50 P-R-T axes 59 -22 107

> History: Unknown EKG CLASS #WR030100 Technician: DP 60783 Test ind:EKG

Normal sinus rhythm with sinus arrhythmia

Left atrial enlargement

Anterior infaret, age undetermined Inferior infarct, age undetermined

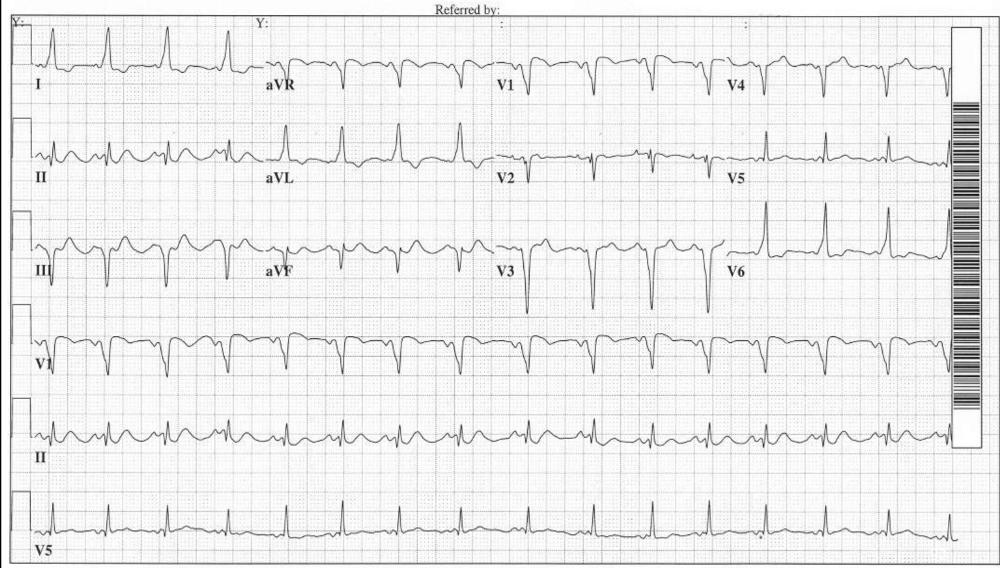
ST & T wave abnormality, consider lateral ischemia

Wolff-Parkinson-White

Abnormal ECG

No previous ECGs available

WOLFF-PARKINSON-WHITE TYPE B



## **Patients with Wolff-Parkinson-White:**

- Typically Pediatric / Young Adult
- May not know they have it
- May experience episodes of "palpitations" or "Very Fast Heartbeat."

The W-P-W often CAUSES A-fib, and the patient CALLS 911 or PRESENTS TO THE ED like this.....

# 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%

#### ST. JOSEPH'S HOSPITAL-

 37 yr
 Vent. rate
 180 BPM

 Male
 Caucasian
 PR interval
 \* ms

 QRS duration
 148 ms

 Room:OP
 QT/QTc
 284/491 ms

 Loc:8
 Option:16
 P-R-T axes
 \* -77 103

WIDE QRS TACHYCARDIA – POSSIBLE VT Right bundle branch block PATTERN Abnormal ECG

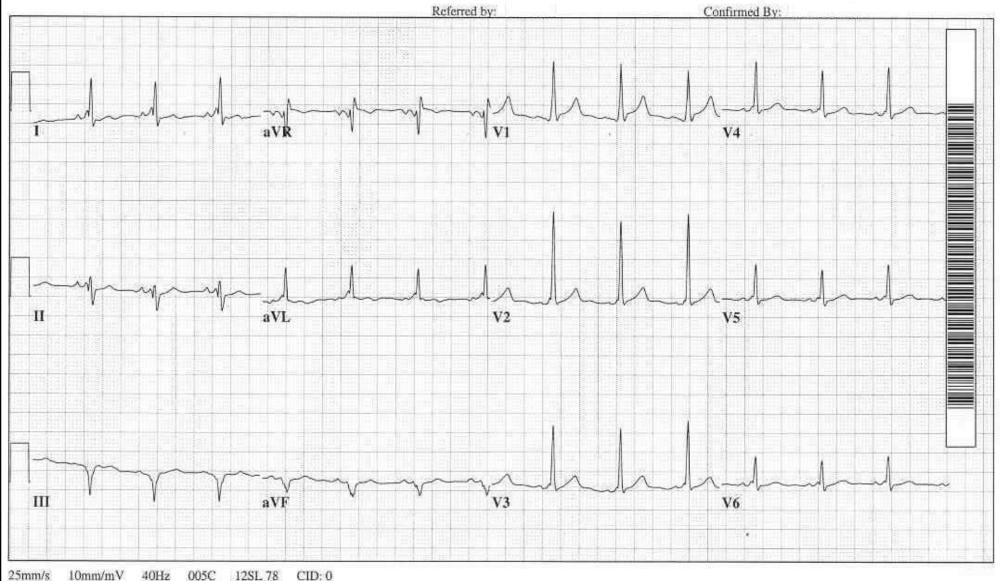


Physician correctly identified Atrial Fibrillation with Rapid Ventricular Response.

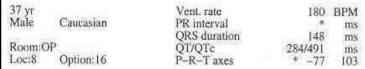
However did NOT identify the Wolff-Parkinson-White component.

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

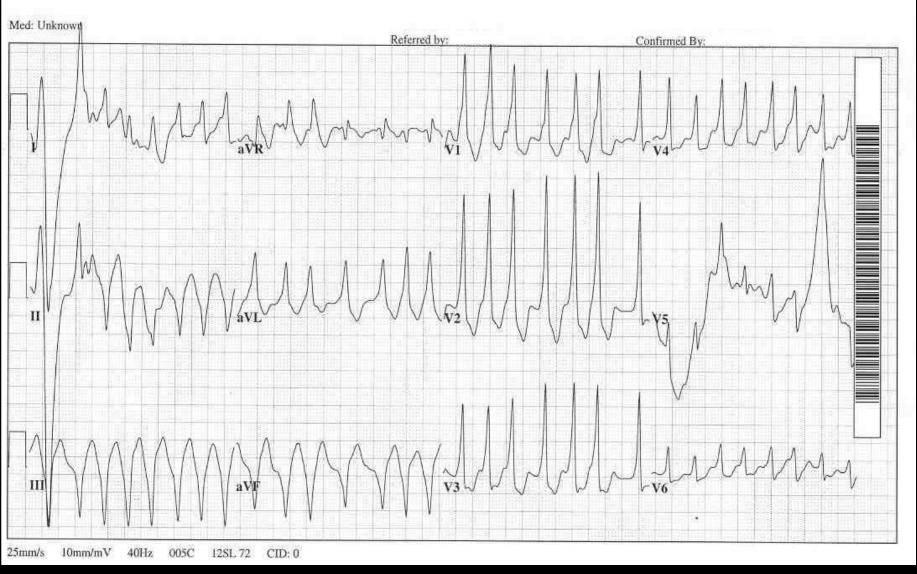
37 yr Male Vent. rate 82 BPM PR interval QRS duration QT/QTe P-R-T axes Caucasian 132 ms 128 ms Room:OP 392/458 77 -44 ms Loc:8 Option:19 154 Normal sinus rhythm Ventricular pre-excitation, WPW pattern type A Abnormal ECG



#### ST. JOSEPH'S HOSPITAL-



WIDE QRS TACHYCARDIA – POSSIBLE VT Right bundle branch block PATTERN Abnormal ECG



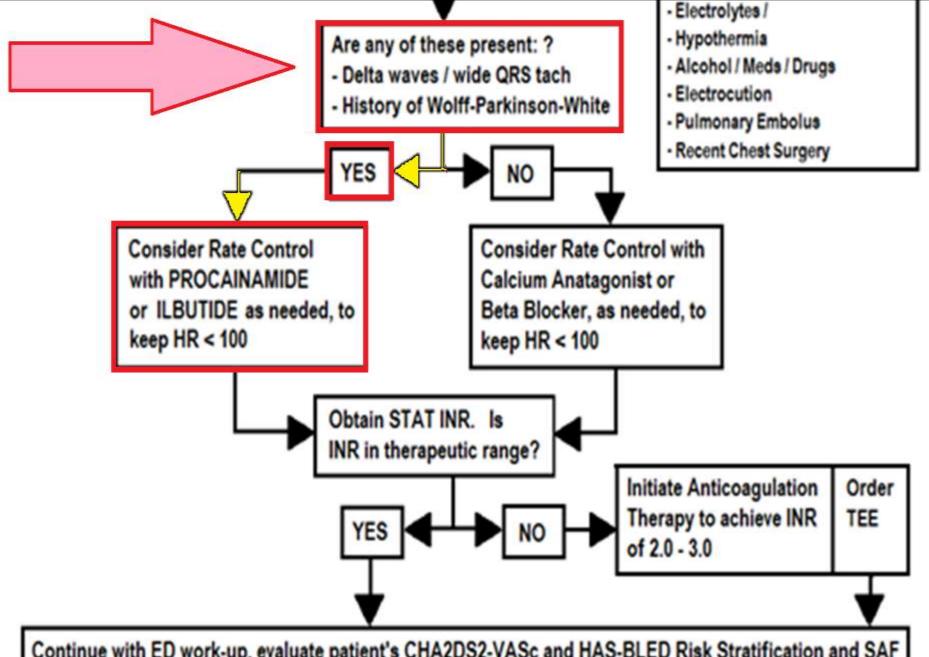
-NOTE IRREGULARITY OF RHYTHM - (SUGGESTIVE of A-FIB) -LOOK FOR DELTA WAVES



#### CHARACTERISTICS of W-P-W with Afib & RVR:

- WIDE COMPLEX TACHYCARDIA
- IRREGULARLY IRREGULAR R R INTERVALS !!





Continue with ED work-up, evaluate patient's CHA2DS2-VASc and HAS-BLED Risk Stratification and SAF Scores, consider consultation with Hospitalist and Electrophysiologist, consider appropriate disposition:

Admission to ICU / CPCU / Telemetry / Observation / Discharge

## WIDE COMPLEX TACHYCARDIA

(QRS > 120 ms)

**MONOPHASIC** 

**ABC** s

#### NO PULSE

GO TO V - FIB ALGORITHM!

#### **PULSE - UNSTABLE**

- IMMEDIATE SYNC. CARDIOVERSION:
  - 100 j biphasic
  - consider sedation
- INCREASE joules
- MEDS:
  - -PROCAINAMIDE
  - -AMIODARONE

#### **PULSE - STABLE**

- O2, IV-IO, EKG
- MEDS:
- ADENOSINE 6-12 (only if REGULAR)
- PROCAINAMIDE (20-50mg/min)
- AMIODARONE (150 over 10min + 1mg/ min INFUSION

## WIDE COMPLEX TACHYCARDIA

(QRS > 120 ms)

MONOPHASIC

**ABC** s

#### NO PULSE

GO TO V - FIB ALGORITHM!

#### **PULSE - UNSTABLE**

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  - -AMIODARONE

#### **PULSE - STABLE**

- O2, IV-IO, EKG
- MEDS:
- ADENOSINE 0-12
- PROCAINAMIDE (20-50mg/min)
- MUUNANUNE
  - (150 a 10min +
  - img/ min INTUSION

NO AV NODAL BLOCKERS [ e.g. ADENOSINE, CALCIUM CHANNEL BLOCKERSI FOR WILLE BOWERS THE BUILDING THAT BOULD BE ATRIAL EIBRILLATION Pre-Excitation (W-P-W)



"ROAD TO FOREVER," Rt 385, Oklahoma panhandle, 1999

# OBTAINING THE 12 LEAD ECG

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

Limb leads should be on the limbs.

- 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.

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

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

#### AHA/ACC/HRS Scientific Statement

# Recommendations for the Standardization and Interpretation of the Electrocardiogram

Part I: The Electrocardiogram and Its Technology

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

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

# Recommendations for the Standardization and Interpretation of the Electrocardiogram

Part I: The Electrocardiogram and Its Technology

#### Recommendations

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

## Leads V1 & V2 on 12 Lead ECG:

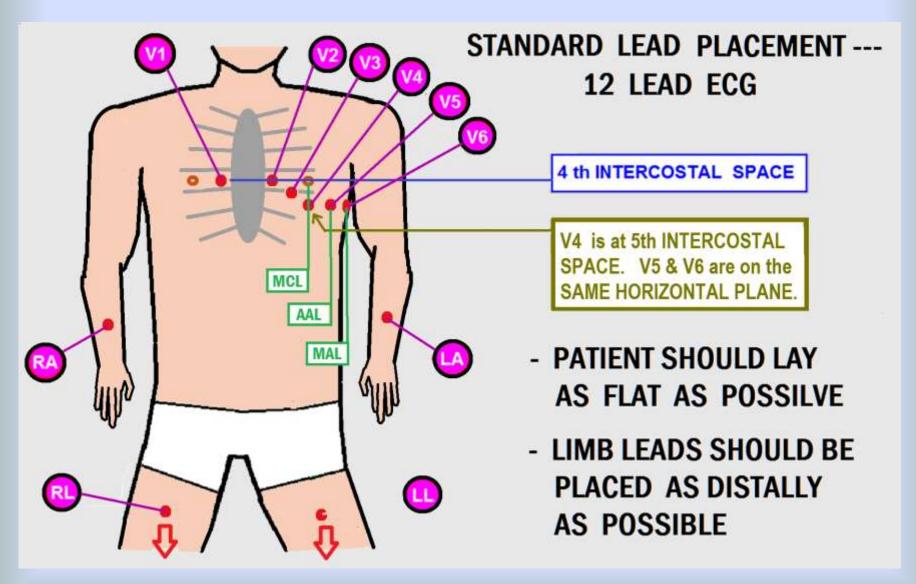
- Proper lead placement of precordial Leads
   V1 and V2 are 4th intercostal space on opposite sides of the sternum.
- Incorrect placement of Leads V1 and V2 will result in: reduction of R wave amplitude (resulting in poor R wave progression) leading to misdiagnosis of previous anterior / septal infarction.

#### AHA/ACC/HRS Scientific Statement

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

Part I: The Electrocardiogram and Its Technology

the often profound alterations in waveforms that can result from precordial electrode misplacement.85,86 A common error is superior misplacement of  $V_1$  and  $V_2$  in the second or third intercostal space. This can result in reduction of initial R-wave amplitude in these leads, approximating 0.1 mV per interspace, which can cause poor R-wave progression or erroneous signs of anterior infarction.87 Superior displacement of the V<sub>1</sub> and V<sub>2</sub> 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



## THE ECG MACHINE

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

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

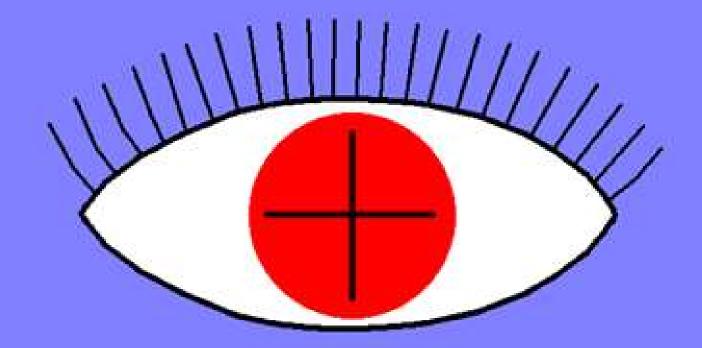


- LEADS AVR, AVL, and AVF
  - 1 POSITIVE ELECTRODE

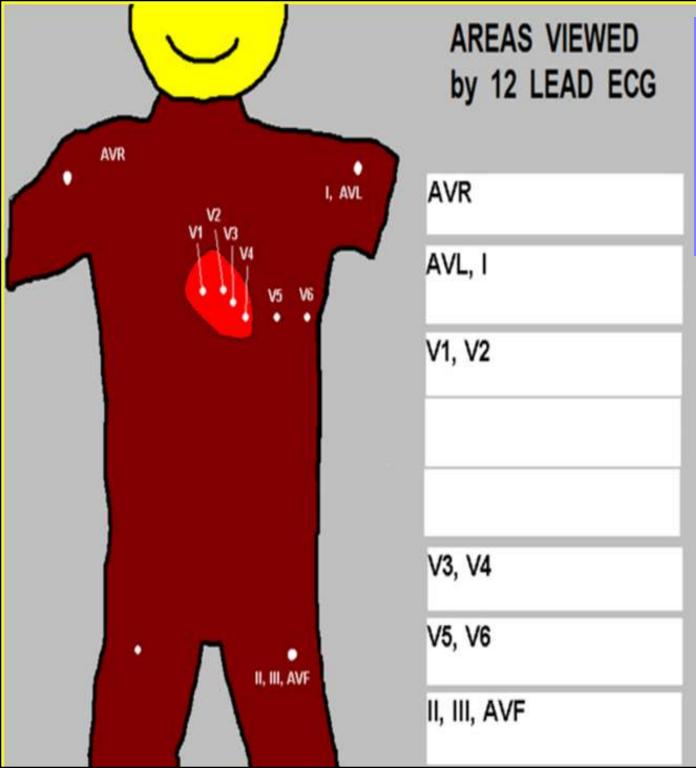
    2 NEGATIVE ELECTRODES

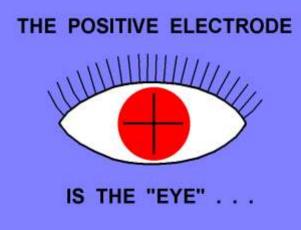
    1 GROUND ELECTRODE

# THE POSITIVE ELECTRODE

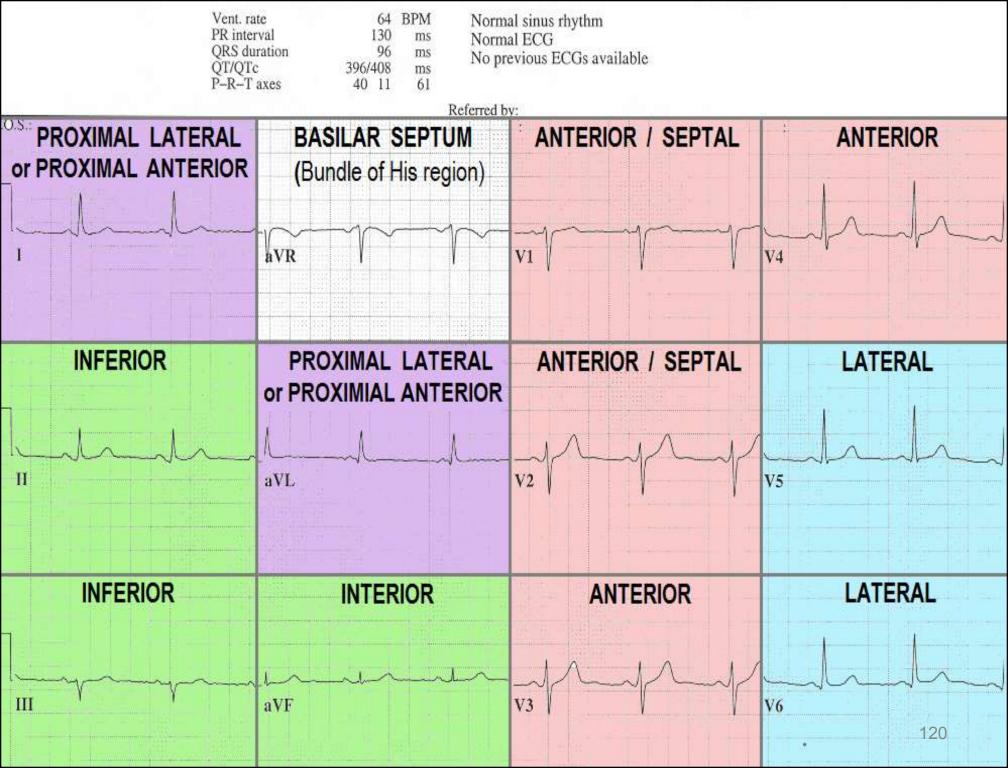


IS THE "EYE" .





#### AREAS VIEWED by 12 LEAD ECG AVR AVR I, AVL **BASILAR SEPTAL** AI AS AVL, I LATERAL V5 ANTERIOR **V6** V1, V2 **ANTERIOR** SEPTAL POSTERIOR (recip.) V3, V4 ANTERIOR V5, V6 LATERAL II, III, AVF II, III, AVF INFERIOR

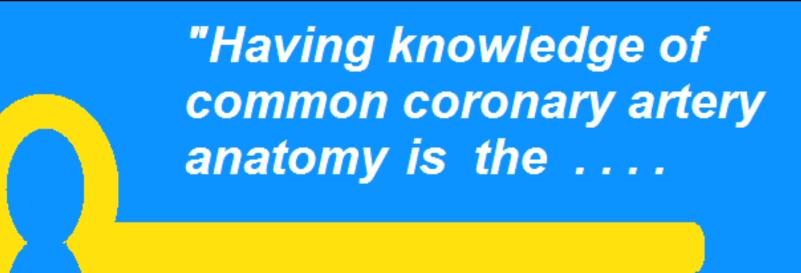


# THE CORONARY



ARTERIES

STRUCTURES SERVED BY THE CORONARY ARTERIES



to understanding the PHYSIOLOGICAL CHANGES that occur during ACUTE MI."

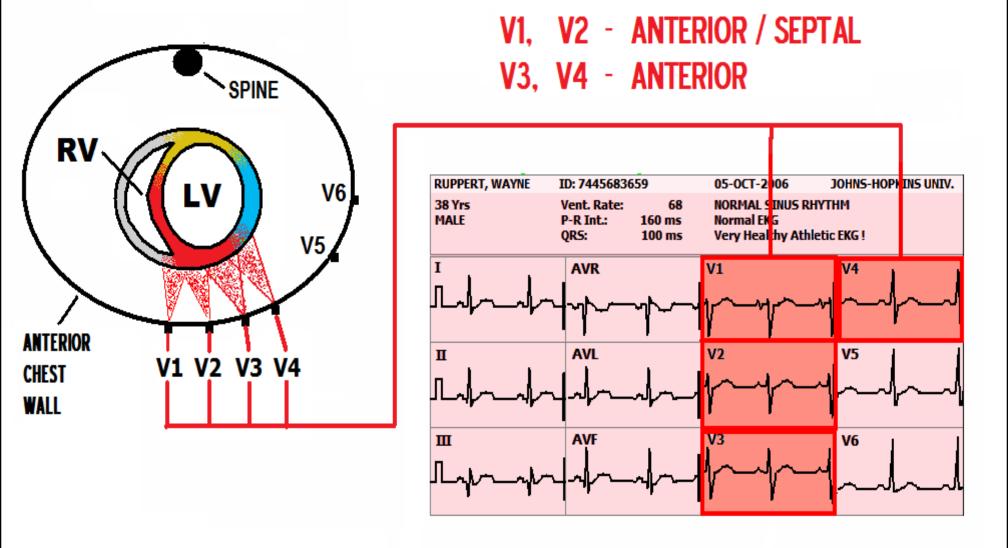
"INVALUABLE ASSET for ALL MEDICAL PROFESSIONALS who provide direct care to STEMI patients!"

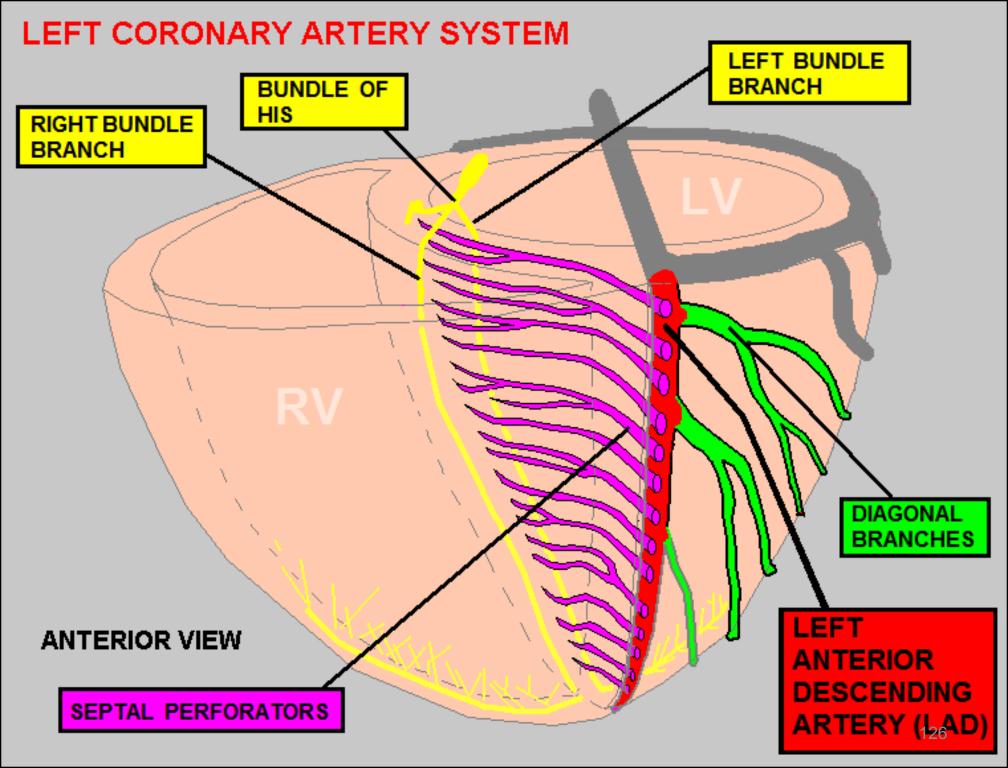
# INTERPRET THE EKG, THEN:

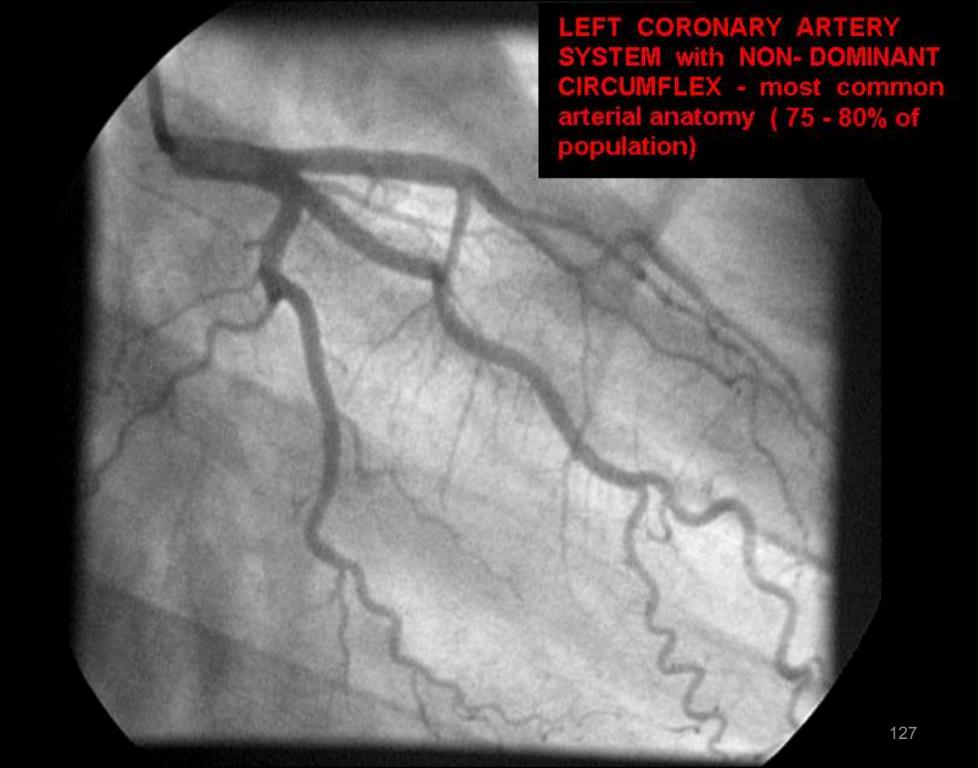
- IDENTIFY THE AREA OF THE HEART WITH A PROBLEM...
- RECALL THE ARTERY WHICH SERVES THAT REGION...
- RECALL OTHER STRUCTURES
  SERVED BY THAT ARTERY...
- ANTICIPATE FAILURE OF THOSE STRUCTURES...
- 1NTERVENE APPROPRIATELY!

# V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL

of the LEFT VENTRICLE









# – 🎗 — 🕨 HELPFUL HINT ... MEMORIZE THIS ! 🔫 — 🤾 —

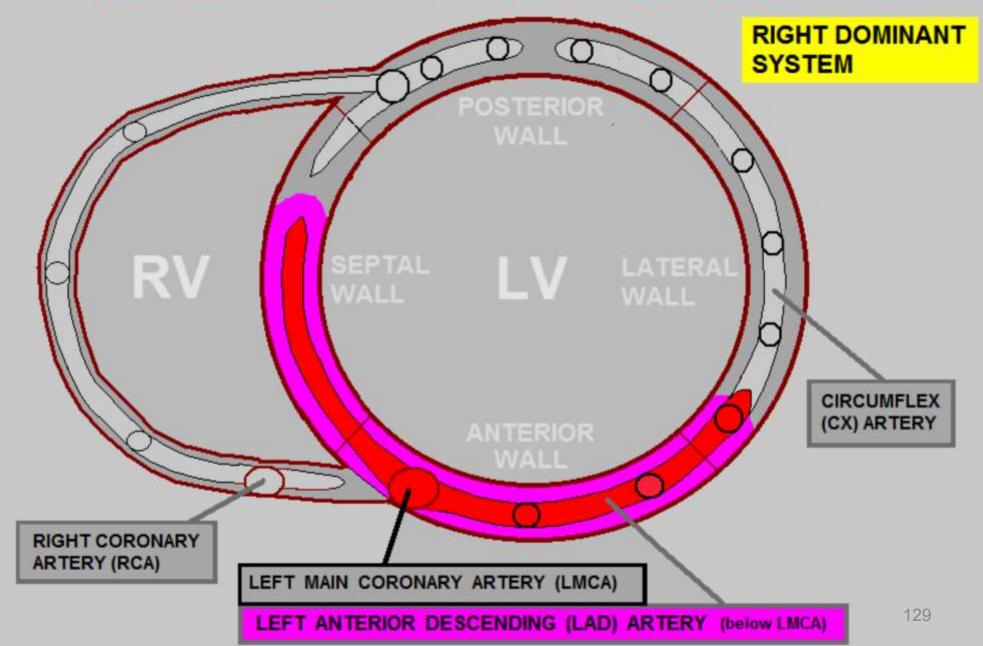


# LEFT ANTERIOR DESCENDING ARTERY (LAD)

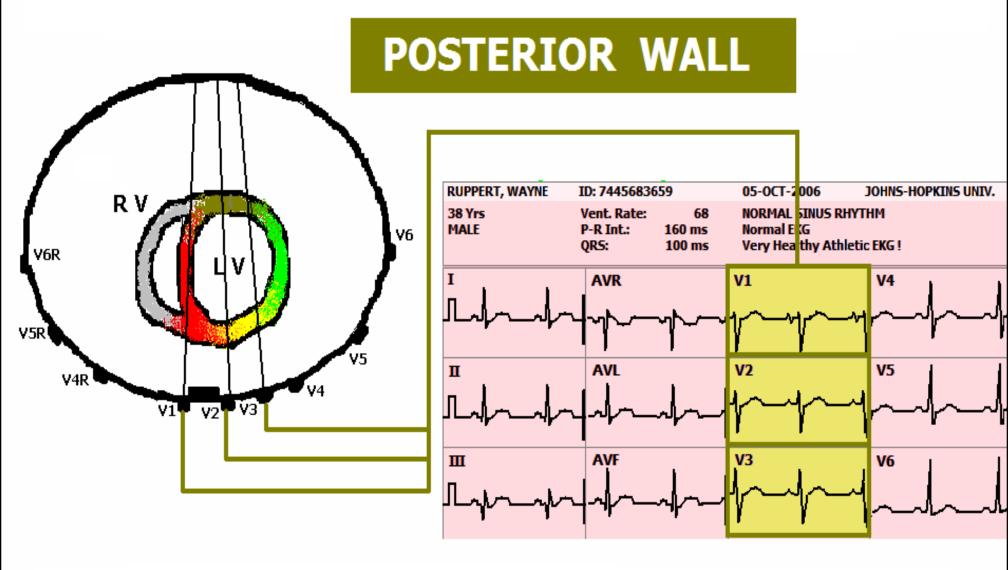
- **BUNDLE OF HIS**
- **BUNDLE BRANCHES ( )**
- > 35 45 % OF LV MUSCLE MASS
  - ANTERIOR WALL
  - SEPTAL WALL (anterior 2/3)

#### LEFT ANTERIOR DESCENDING ARTERY (LAD)





# **LEADS V1 - V3** view the

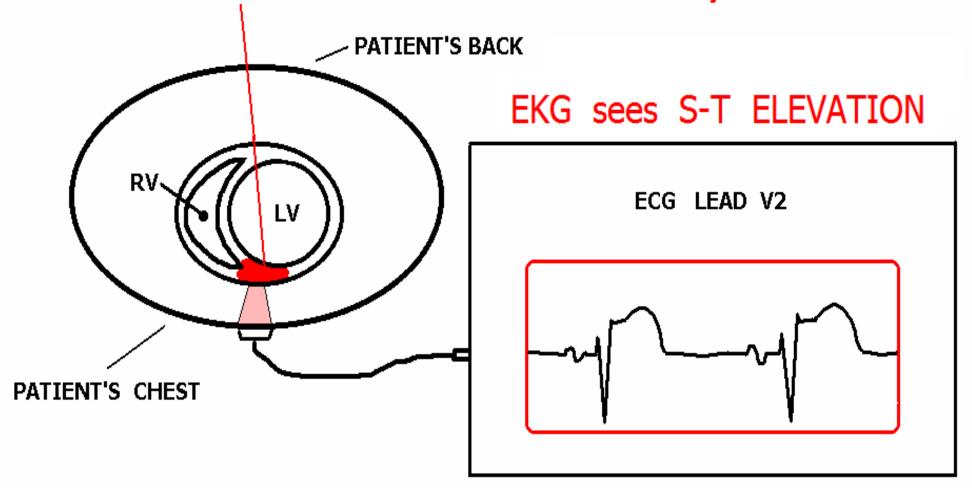


# via RECIPROCAL CHANGES.

#### **HOW EKG VIEWS INDICATIVE CHANGES**

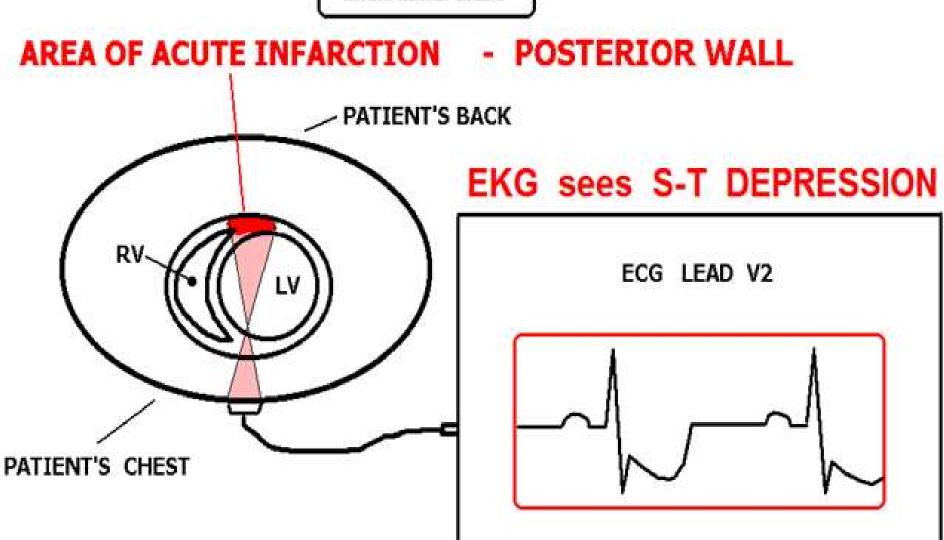
**EXAMPLE:** 

## AREA OF ACUTE INFARCTION - ANTERIOR/SEPTAL

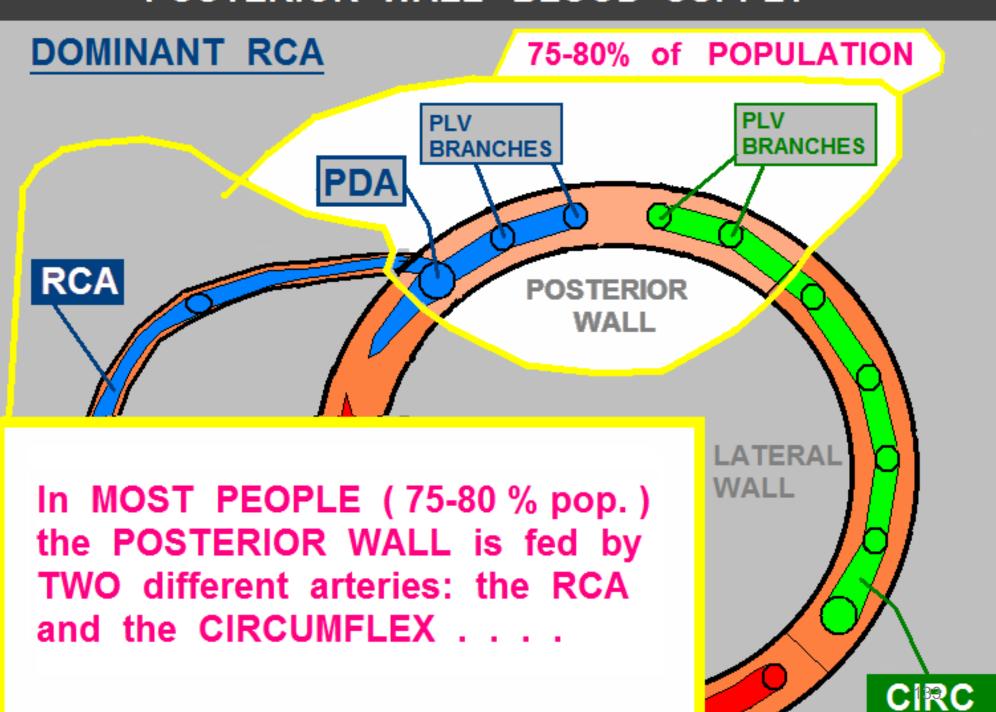


### **HOW EKG VIEWS RECIPROCAL CHANGES**

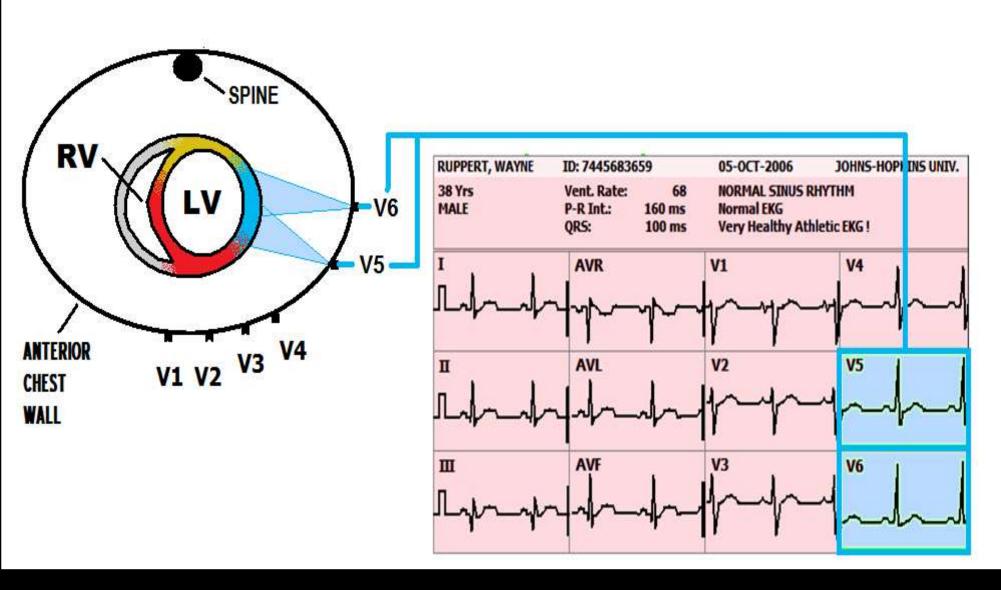
**EXAMPLE:** 

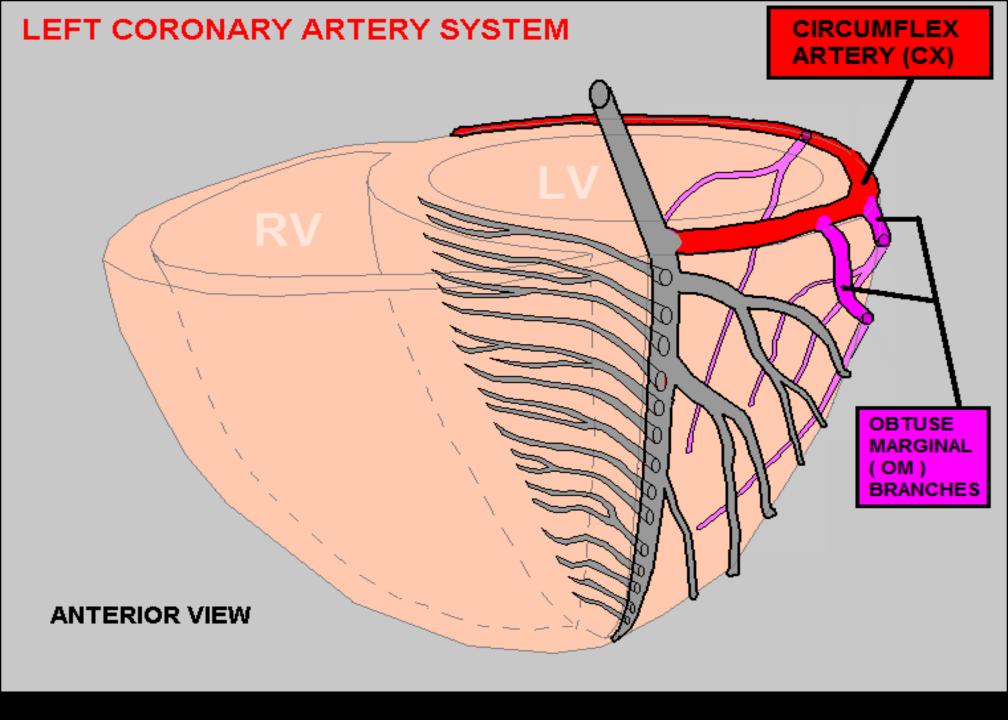


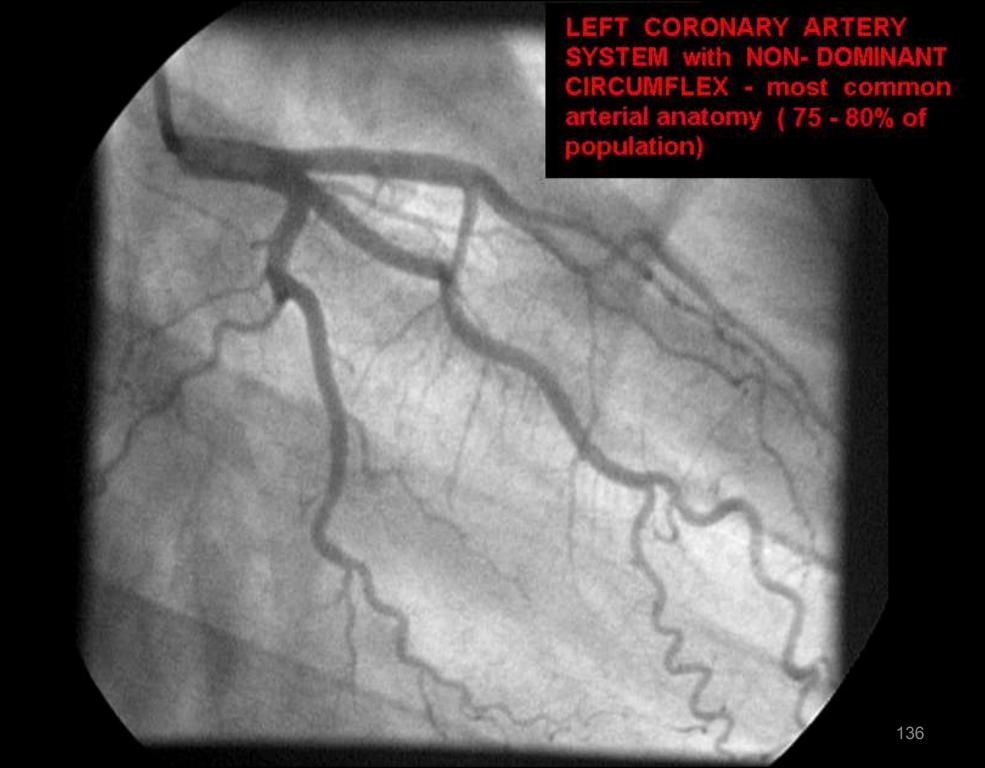
#### POSTERIOR WALL BLOOD SUPPLY



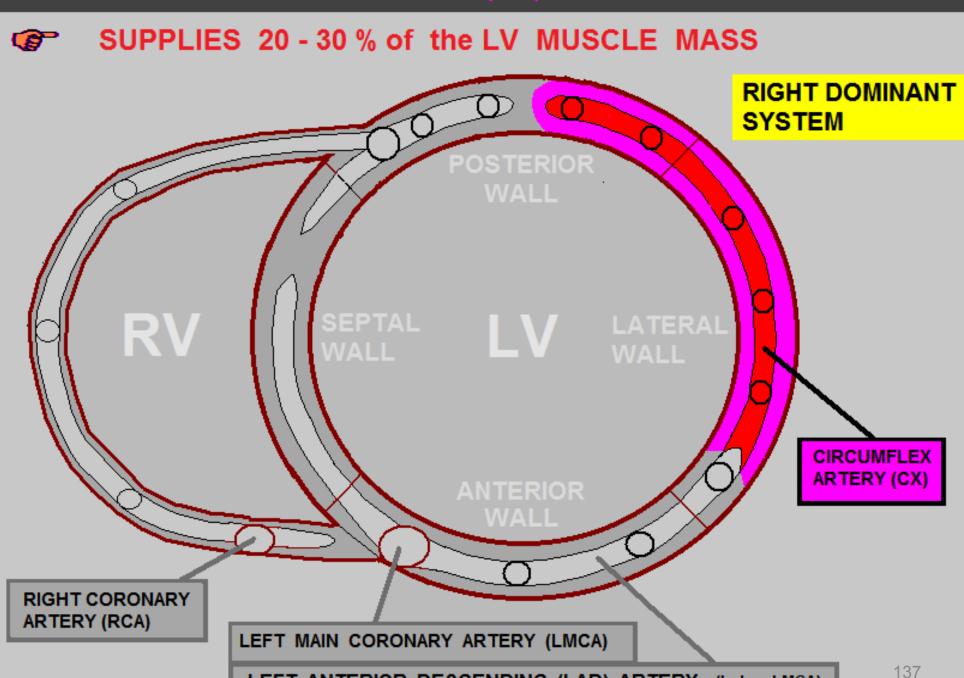
# V5 - V6 VIEW THE LATERAL WALL of the LEFT VENTRICLE







#### CIRCUMFLEX ARTERY (CX) DISTRIBUTION



LEFT ANTERIOR DESCENDING (LAD) ARTERY (below LMCA)



# – 🎗 — → HELPFUL HINT ... MEMORIZE THIS! 🔙 — 🤾 —



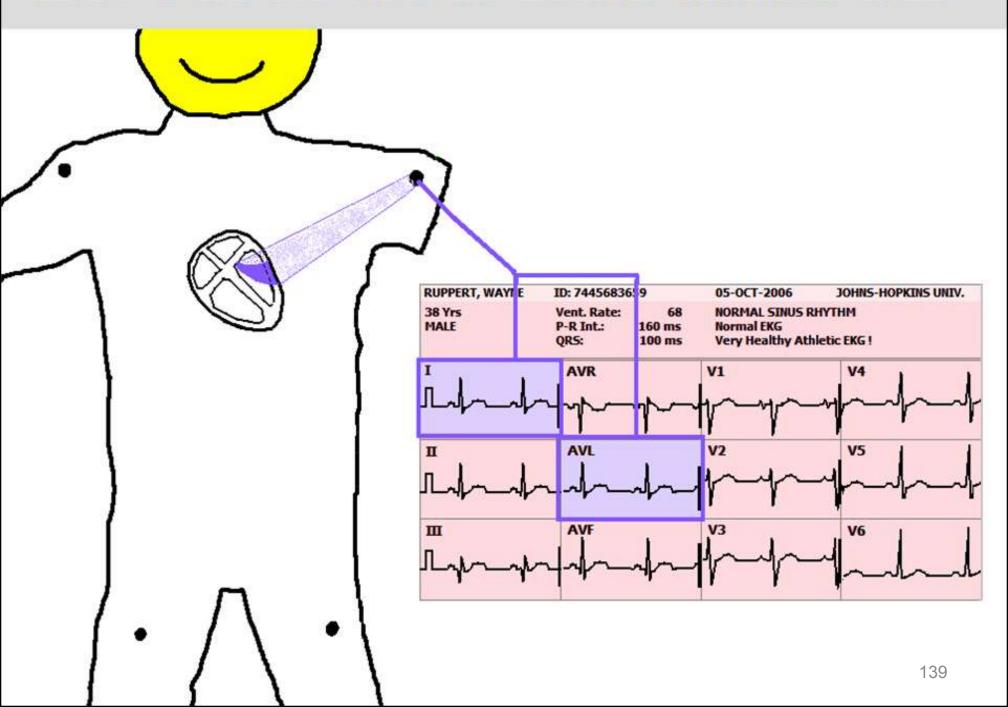


# CIRCUMFLEX ARTERY (CX)

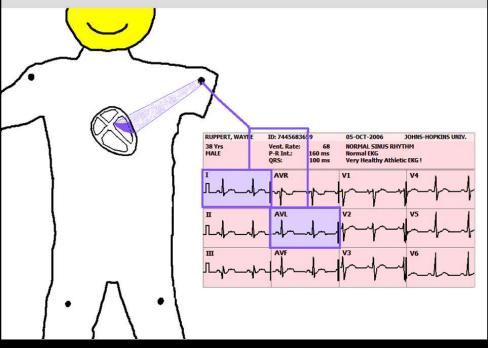
SYSTEMS

- 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 and aVL VIEW the LATERAL - ANTERIOR WALL



#### LEADS I and aVL VIEW the LATERAL - ANTERIOR WALL



#### LEFT MAIN CORONARY ARTERY AV NODE 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 LEFT ANTERIOR DESCENDING ANTERIOR VIEW ARTERY

OCCLUSION of OBTUSE MARGINAL ARTERY

# RV NODE RAMUS ARTERY RAMUS ARTERY SUPPLYING AREA VIEWED BY LEADS I and aVL ORIGINATES FROM LEFT MAIN CORONARY ARTERY AREA OF EKG VIEWED BY LEADS I and aVL

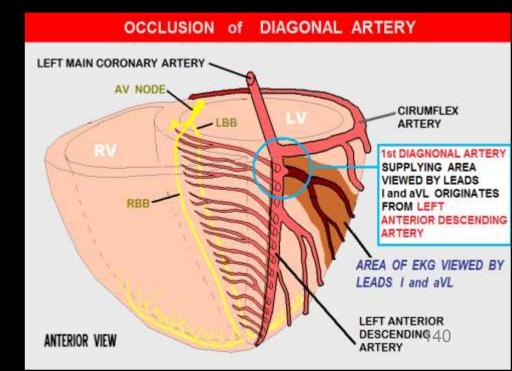
ANTERIOR VIEW

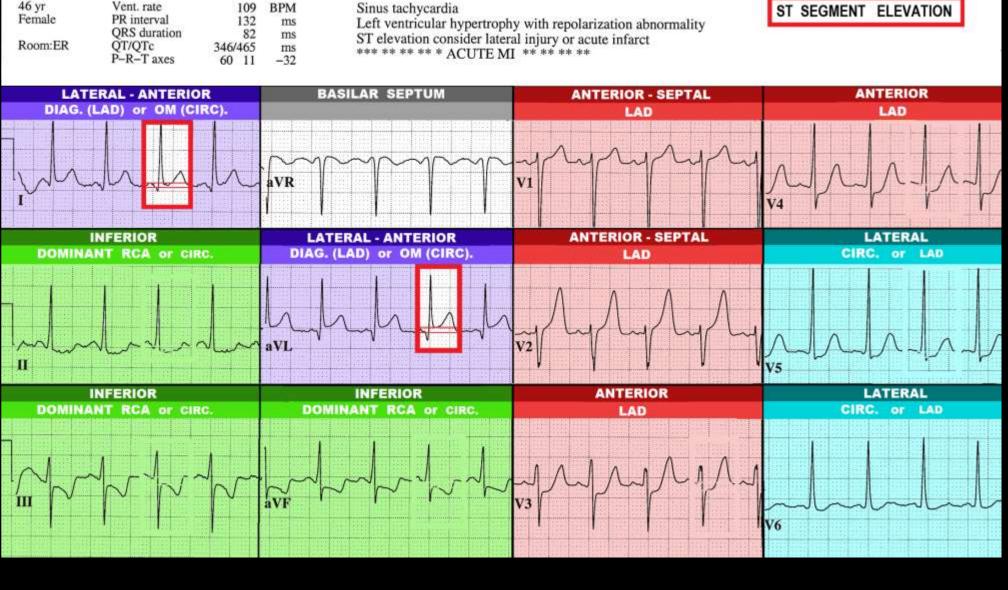
LEFT ANTERIOR

DESCENDING

ARTERY

OCCLUSION of RAMUS ARTERY





# ST Segment elevation ONLY in Leads I and aVL

46 yr

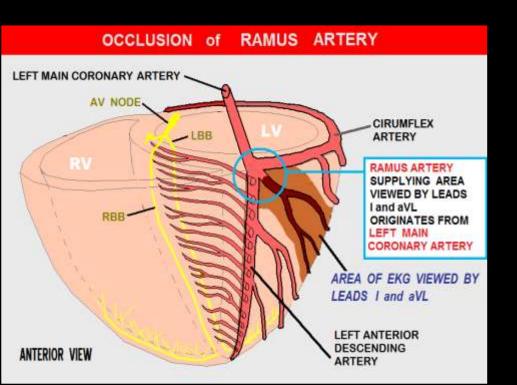
Vent. rate

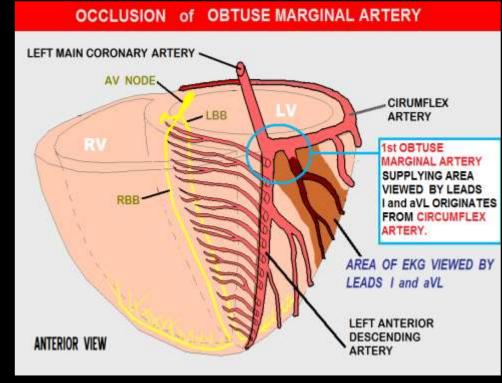
**BPM** 

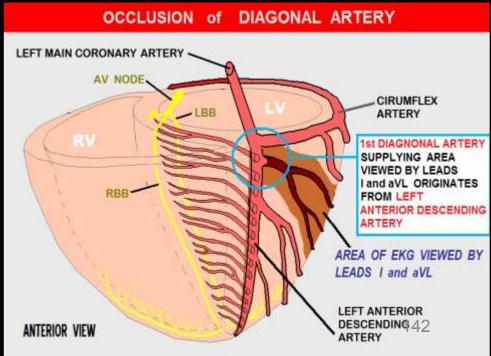
Sinus tachycardia

Usually indicates the "Culprit Artery" is most likely One of the following:

- RAMUS BRANCH
- 1st DIAGONAL off of LAD
- 1st OBTUSE MARGINAL off of CIRCUMFLEX



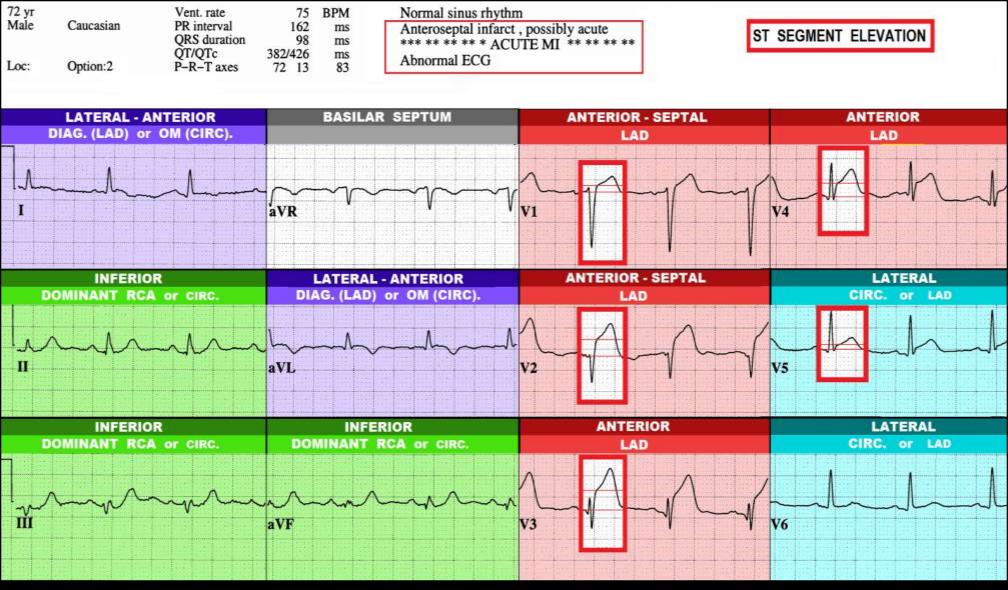




# Here's why we care: Think of Leads I and aVL as

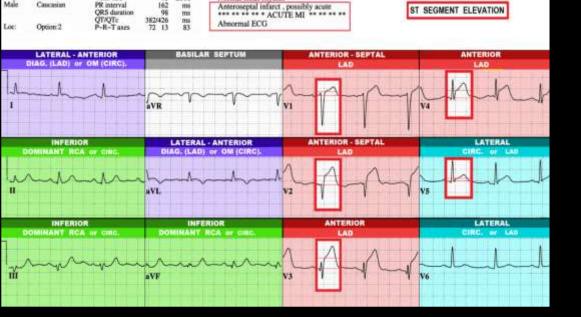


"THE POWERBALL"....



If you patient's ECG shows

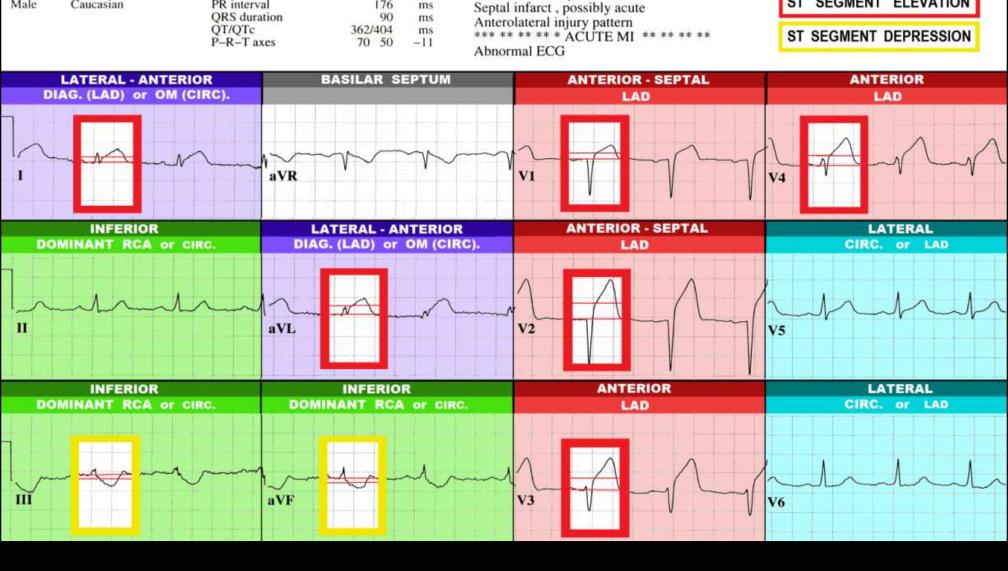
ST Elevation in Leads V1 – V4 . . . .



If your patient's ECG shows
ST Elevation in Leads V1 – V4...

The obstruction is usually located at The MID – LAD level.

## OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY LEFT MAIN CORONARY ARTERY -AV NODE CIRUMFLEX ARTERY RBB LEFT **ANTERIOR** DESCENDING ARTERY AREA OF INFARCT ANTERIOR VIEW



Normal sinus rhythm

29 yr

Male

Caucasian

Vent. rate

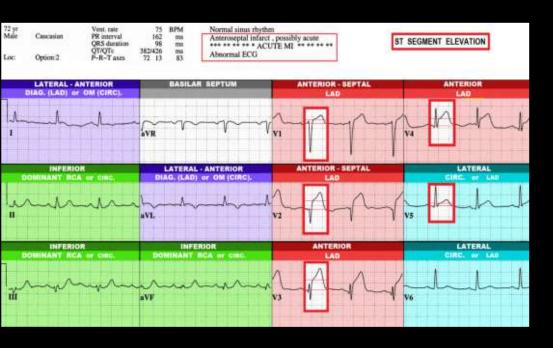
PR interval

176

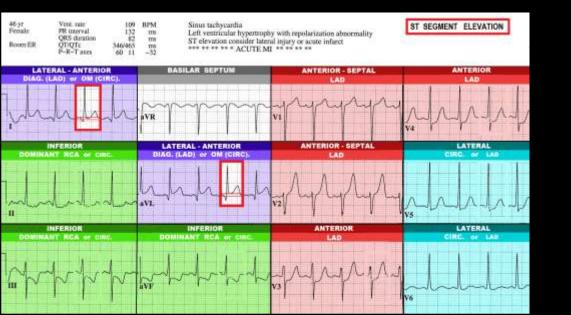
This patient's ECG shows ST ↑ in V1 – V4 AND Leads I and aVL...

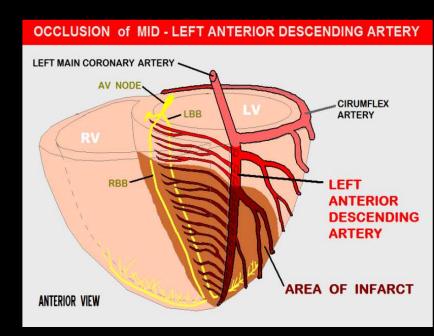
SEGMENT ELEVATION

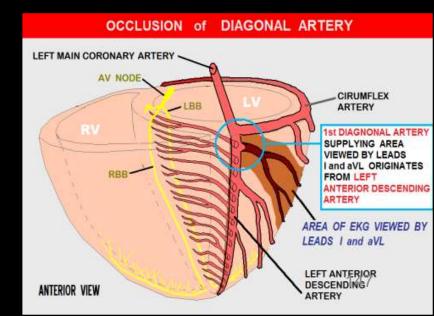
#### That means WE ADD THIS:



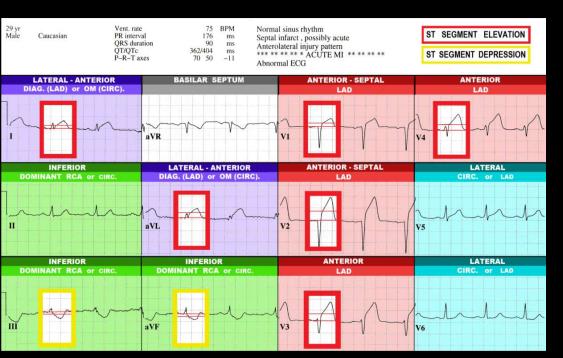
#### TO THIS:

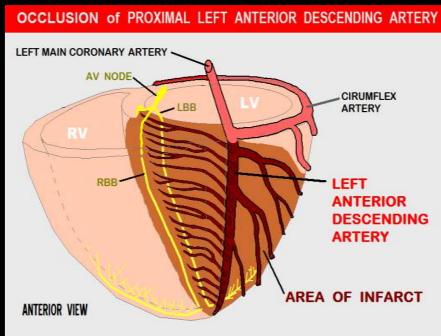






# AND WE GET THIS ....

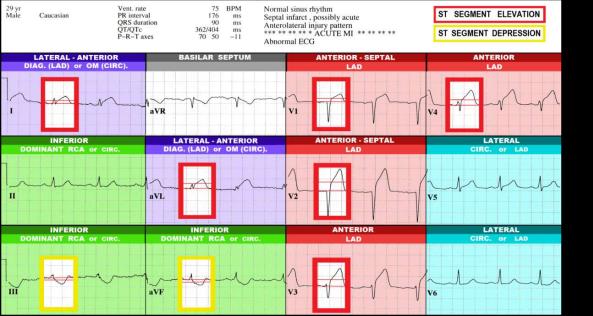




# Our patient just hit the POWERBALL!

Use of the Electrocardiogram in Acute Myocardial Infarction,"

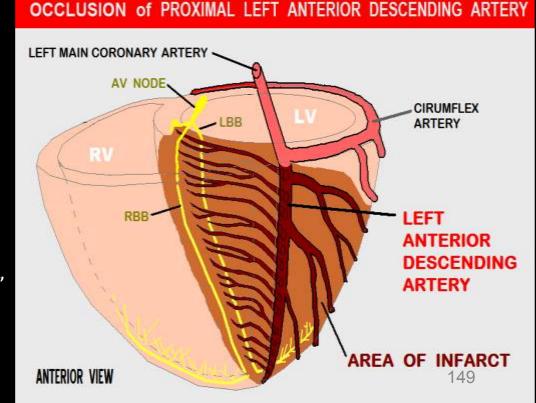
Zimetbaum, et al, NEJM 348:933-940

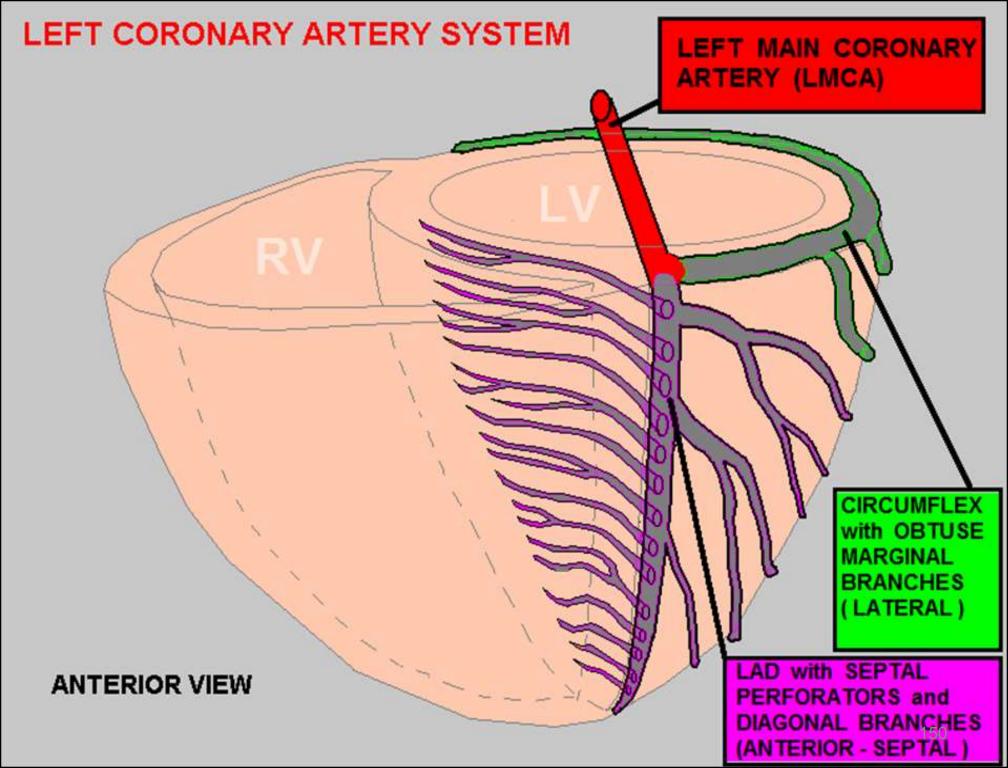


If you patient's ECG shows
ST Elevation in Leads V1 – V4 & I and aVL...

The obstruction is usually located at in the PROXIMAL LAD, above the level of the 1st Diagonal Branch!!

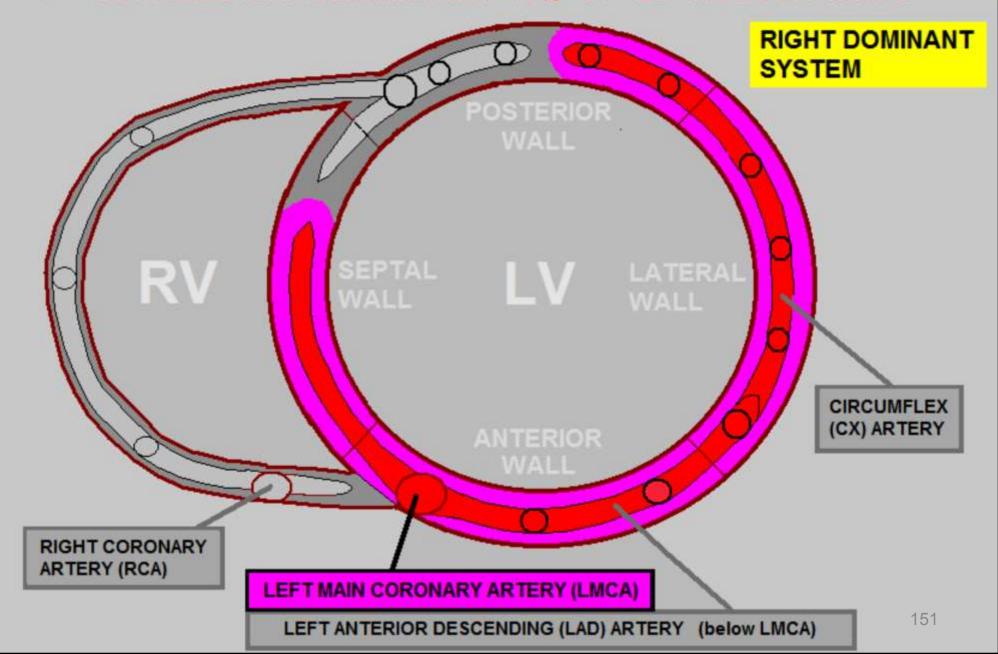
Use of the Electrocardiogram in Acute Myocardial Infarction," Zimetbaum, et al, NEJM 348:933-940



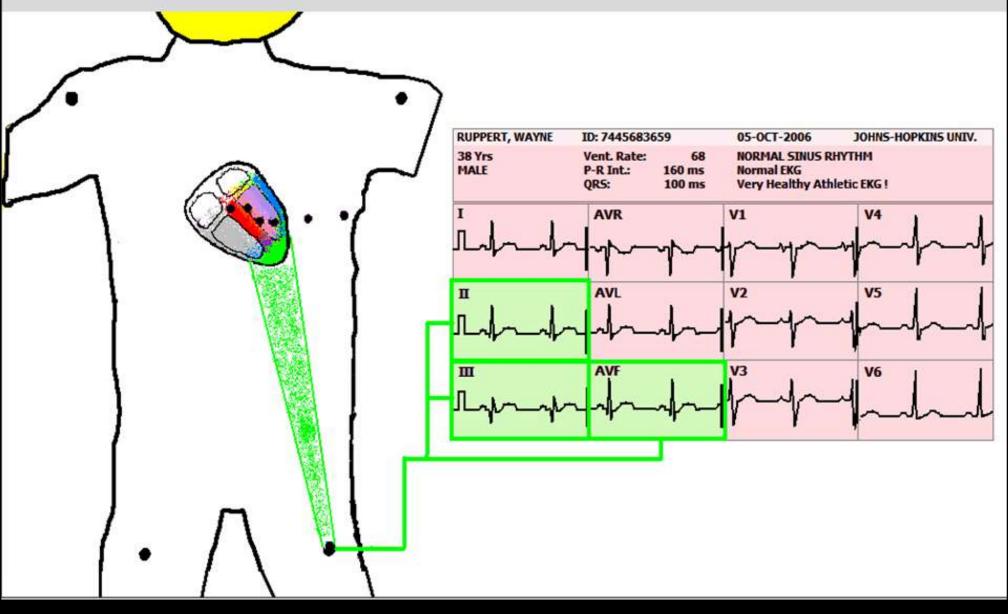


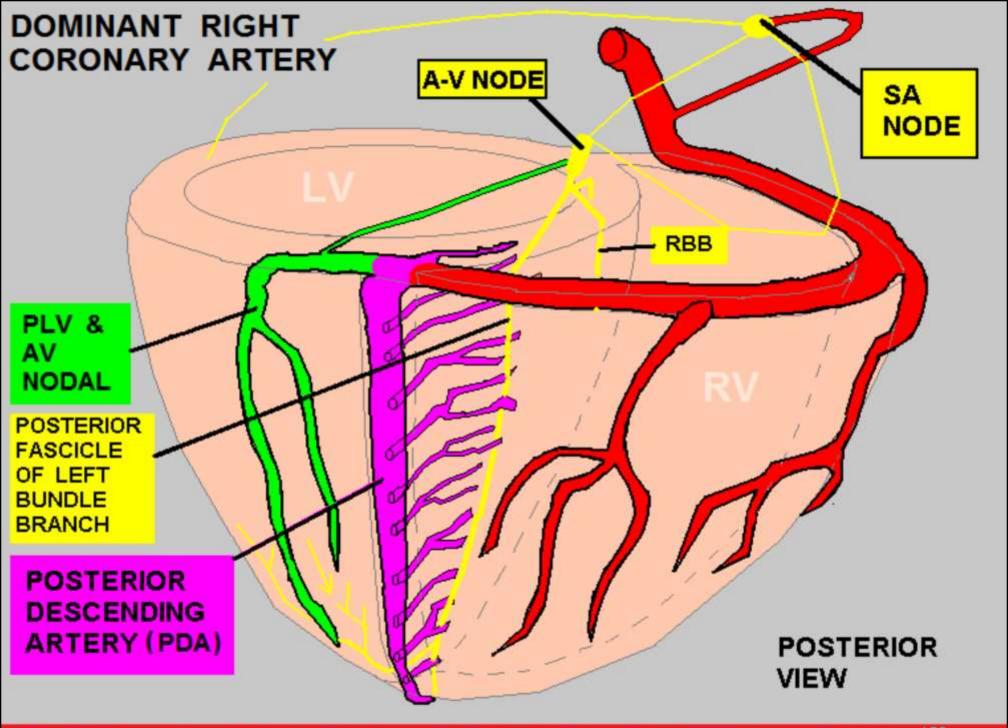
## LEFT MAIN CORONARY ARTERY (LMCA)

#### SUPPLIES APPROXIMATELY 75% OF LV MUSCLE MASS



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







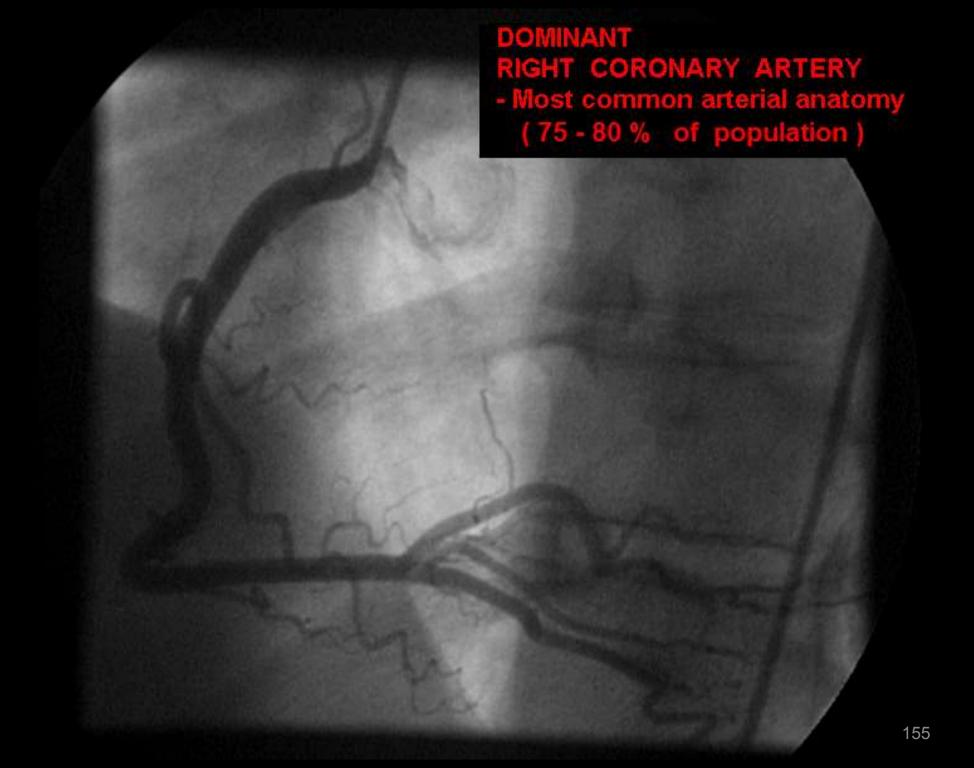
### HELPFUL HINT ... HEMORIZE THIS!



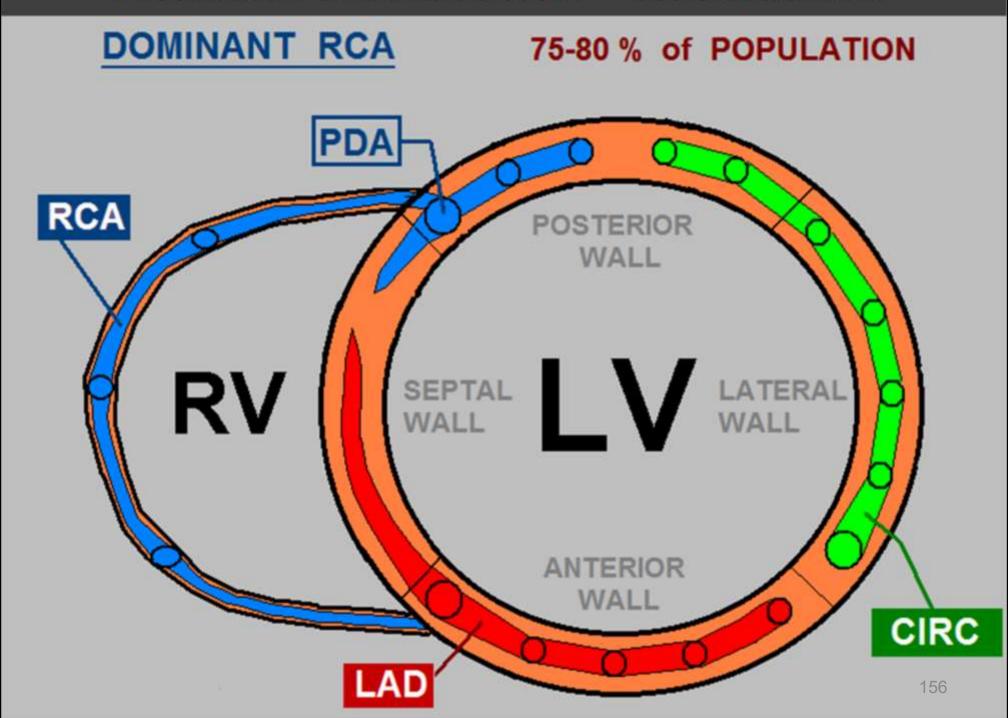
### RIGHT CORONARY ARTERY (RCA)

SYSTEMS

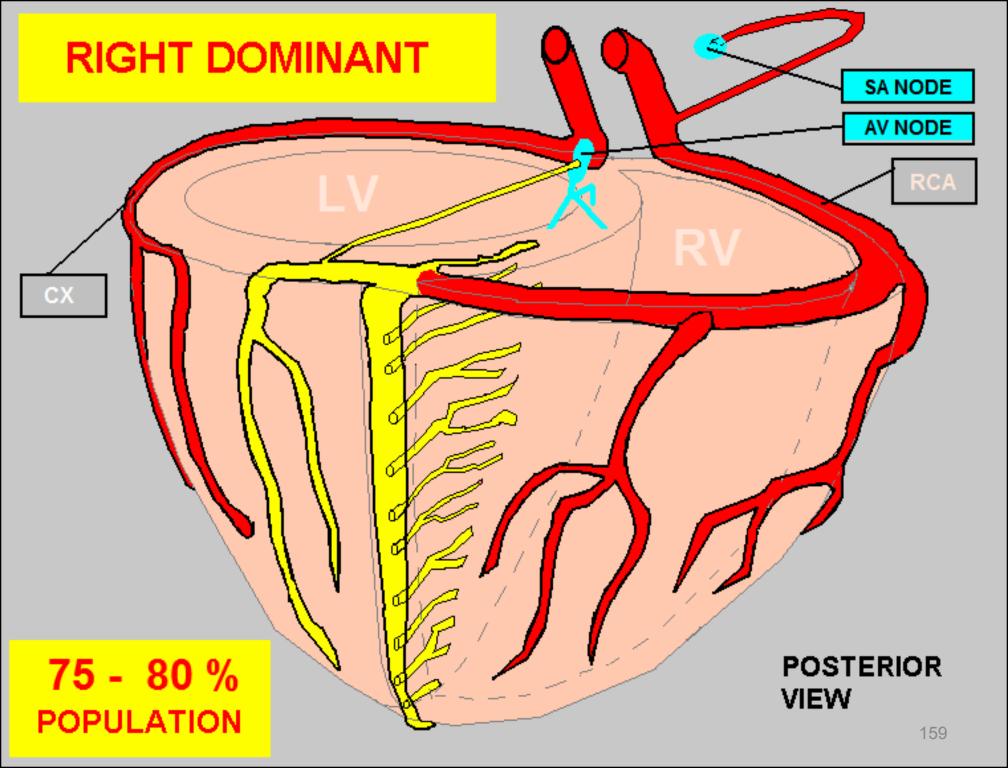
- RIGHT ATRIUM
- **SINUS NODE** (55% of the population)
- RIGHT VENTRICLE 100 % of muscle mass
- ▶ LEFT VENTRICLE: 15 25 % of muscle mass
  - INFERIOR WALL
  - approx. 1/2 of POSTERIOR WALL
- AV NODE

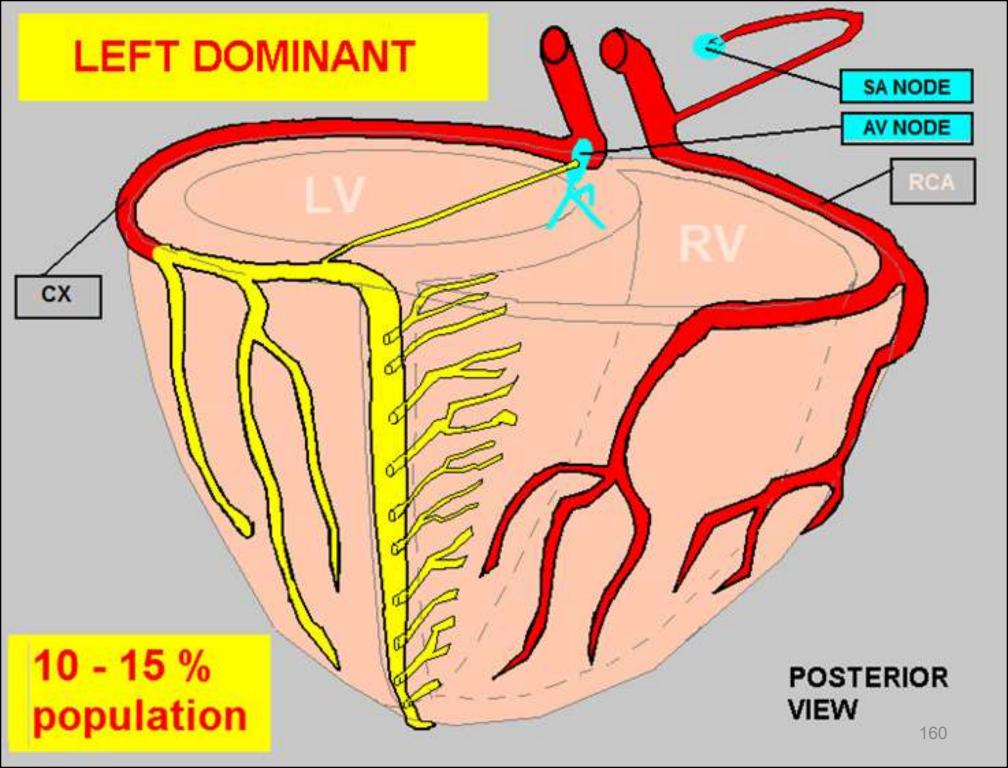


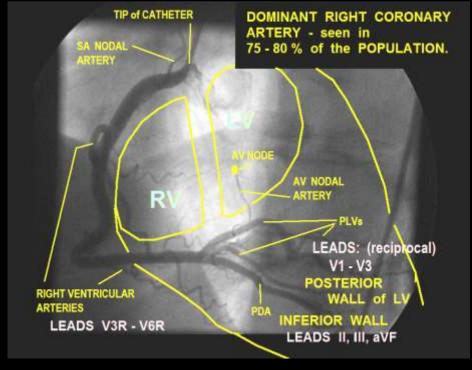
#### ARTERIAL DISTRIBUTION - MYOCARDIUM

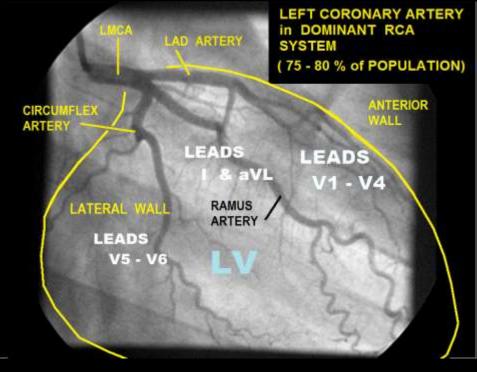


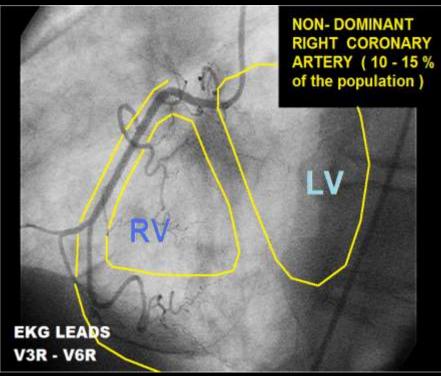
So if the Right Coronary Artery Is DOMINANT in 75 – 80% of the POPULATION, what accounts for the Other 20 – 25% ??

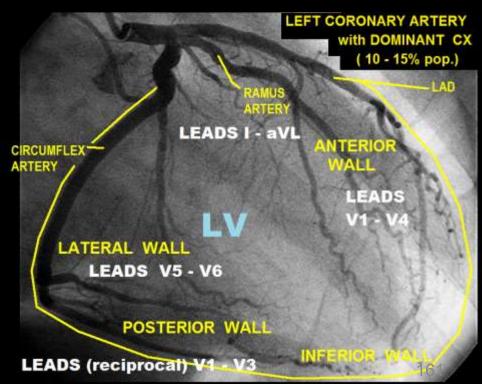




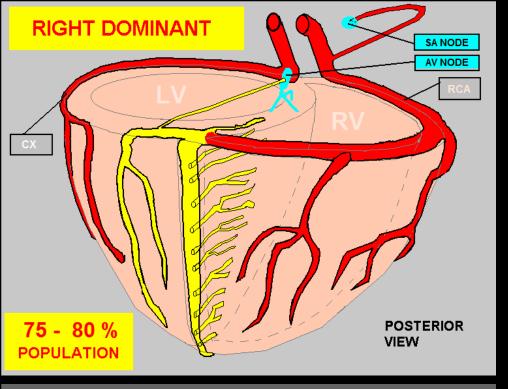


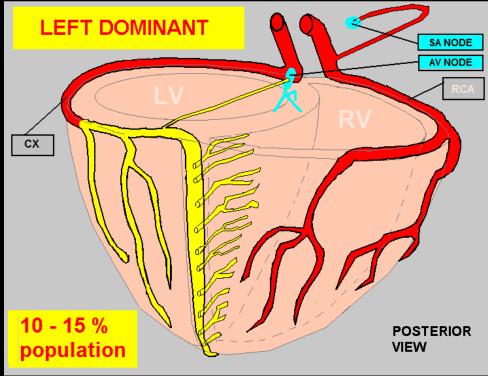


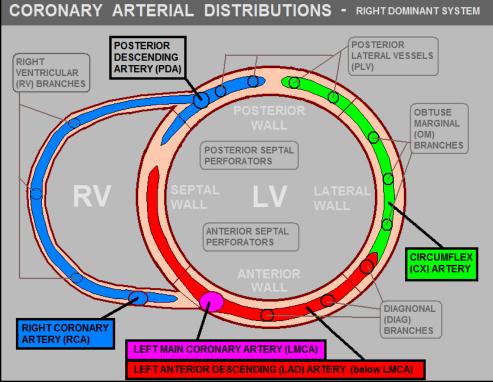


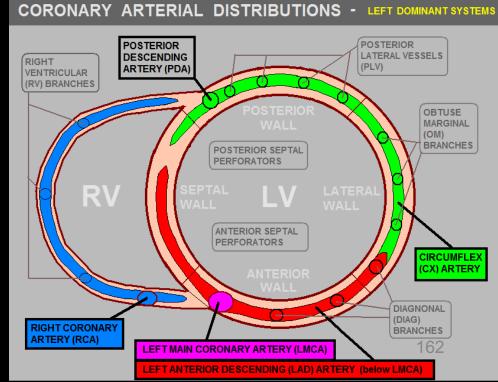


LEADS II, III, aVF









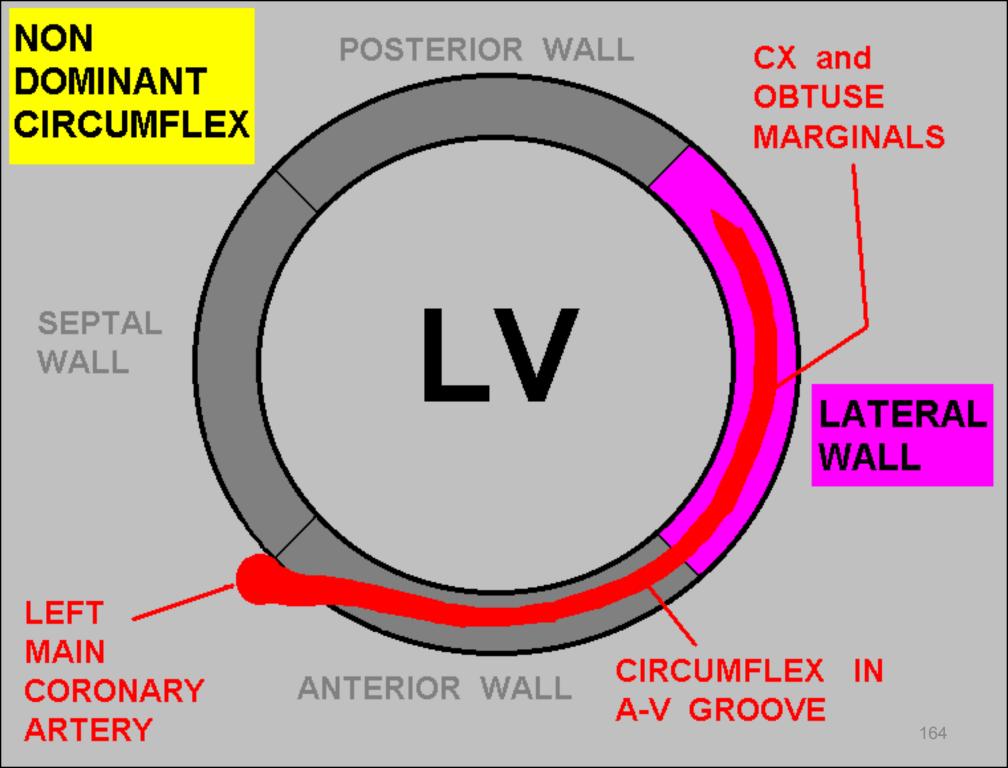
### CIRCUMFLEX ARTERY (CX)

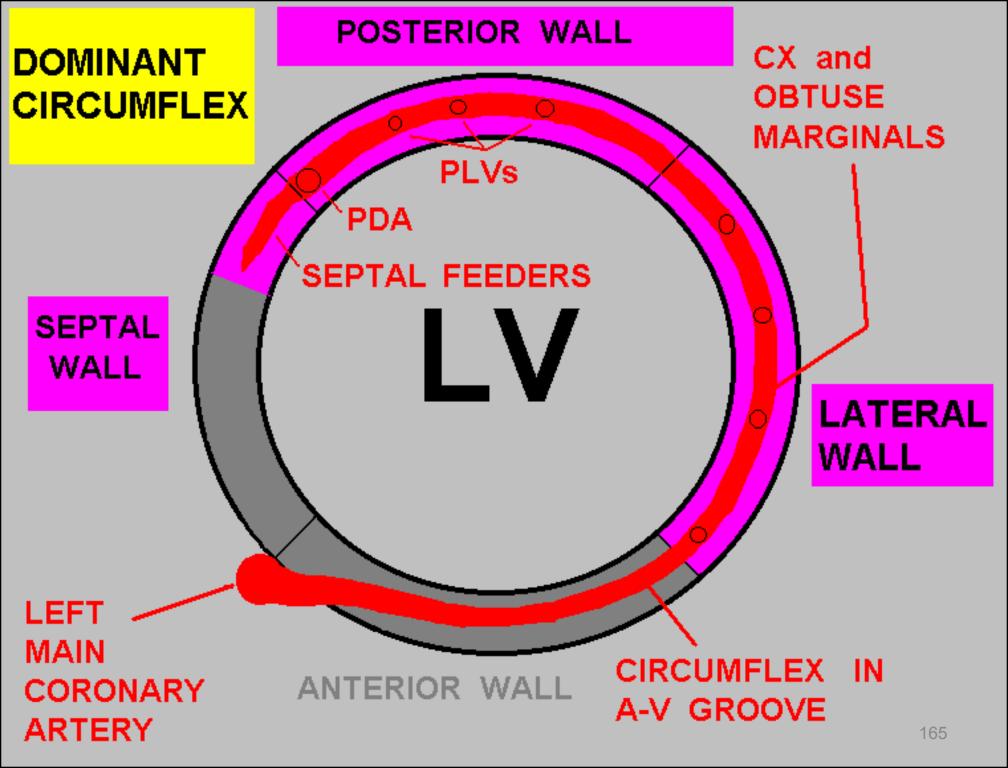
- NON-DOMINANT CX:

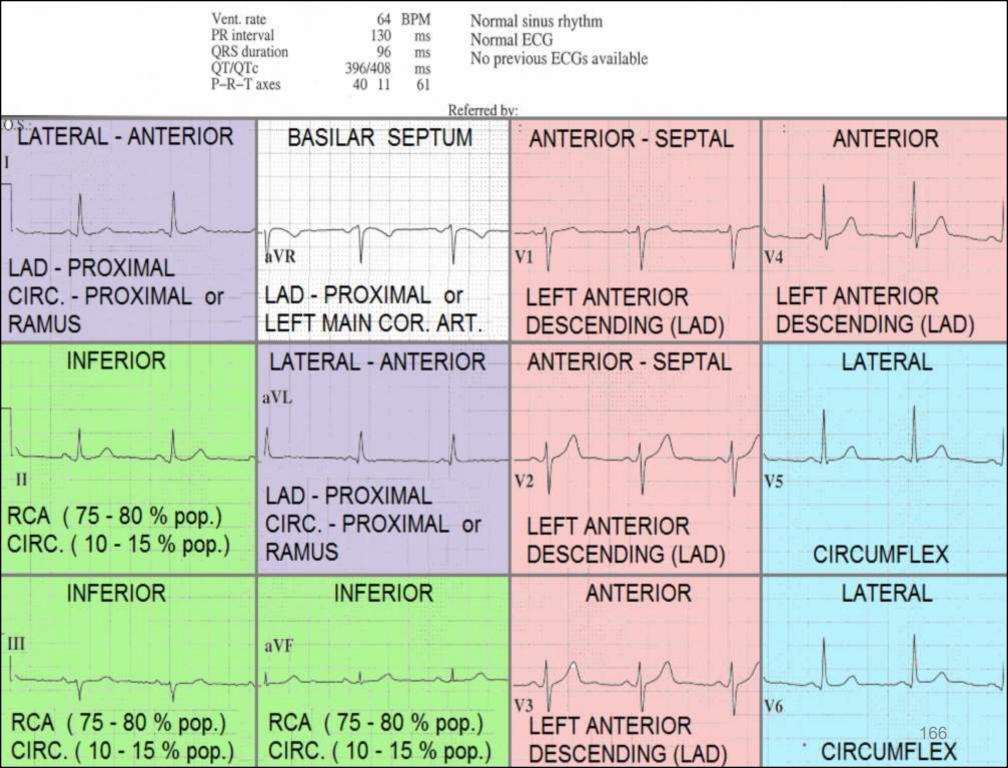
CX = 15 - 30% OF LV MASS

- DOMINANT CX:

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







### A standard

## 12 LEAD EKG

Does NOT show the

### RIGHT VENTRICLE

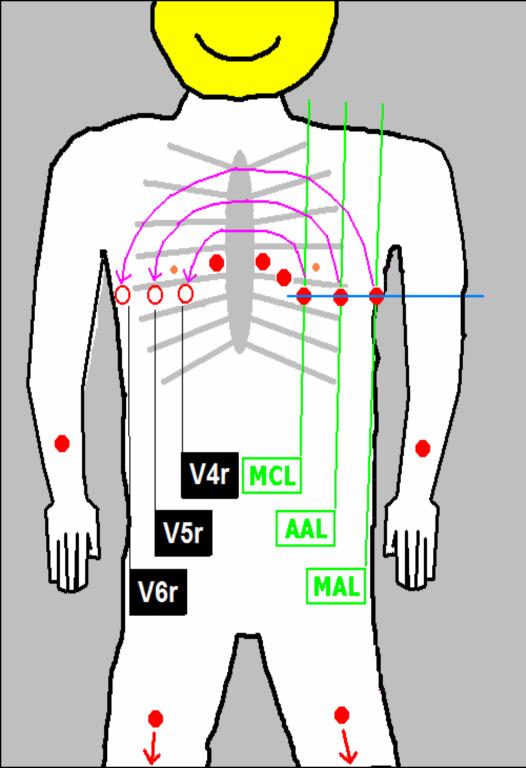
# To see the RIGHT VENTRICLE ...

... such as in cases of

# INFERIOR WALL M.I.

You must do a

RIGHT - SIDED EKG!!

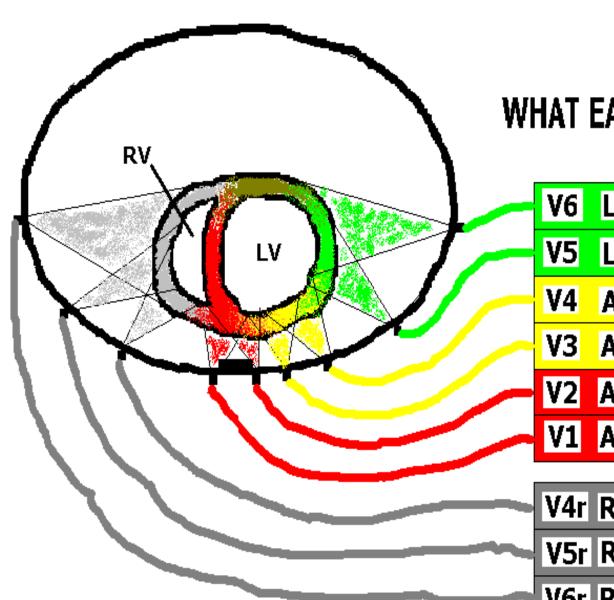


To do a RIGHT - SIDED EKG . .

MOVE leads V4, V5, and V6

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

### THE V-LEADS



WHAT EACH LEAD "SEES" . . .

V6 LATERAL WALL

V5 LATERAL WALL

V4 ANTERIOR WALL

V3 ANTERIOR WALL

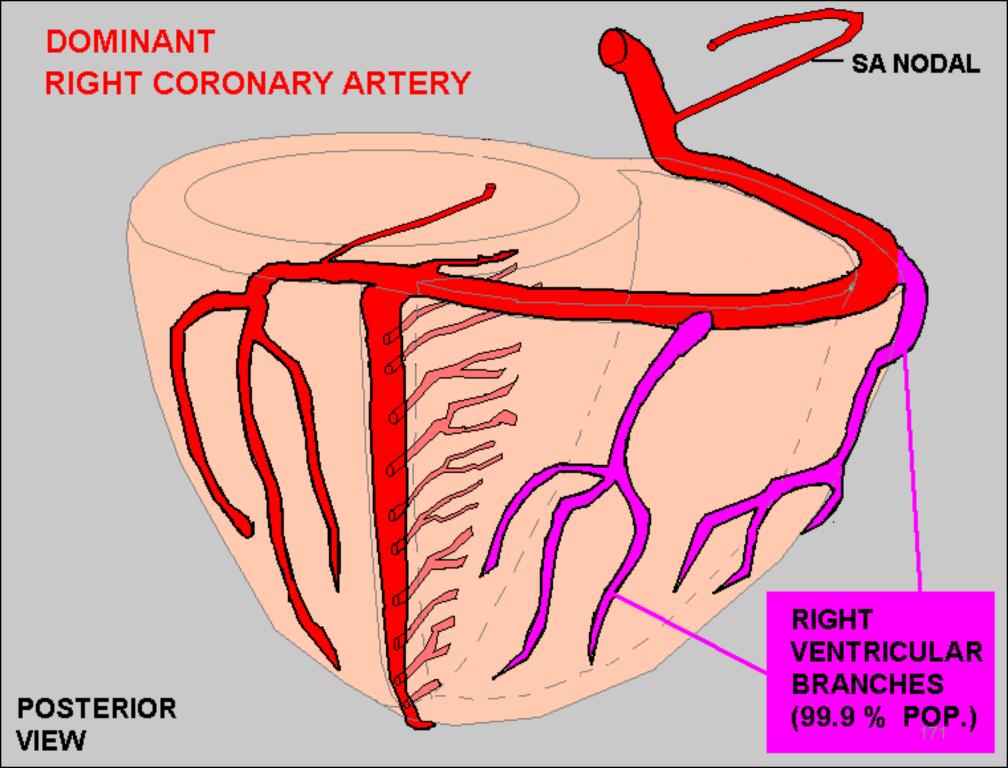
V2 ANTERIO-SEPTAL

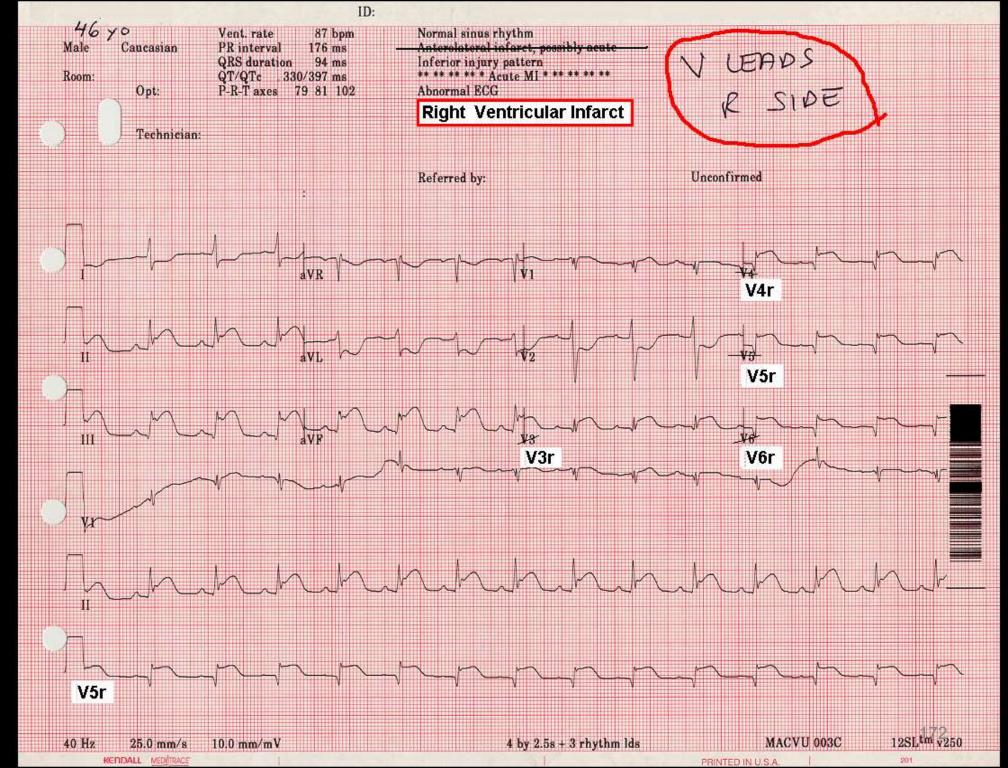
V1 ANTERIO-SEPTAL

V4r RIGHT VENTRICLE

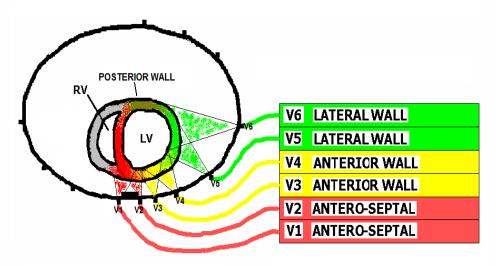
V5r RIGHT VENTRICLE

V6r RIGHT VENTRICLE



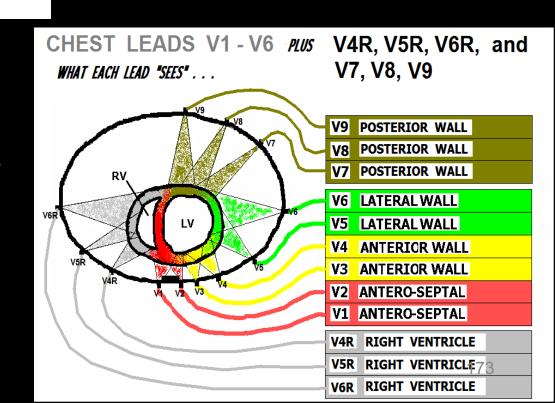


### CHEST LEADS V1 - V6 WHAT EACH LEAD "SEES"...

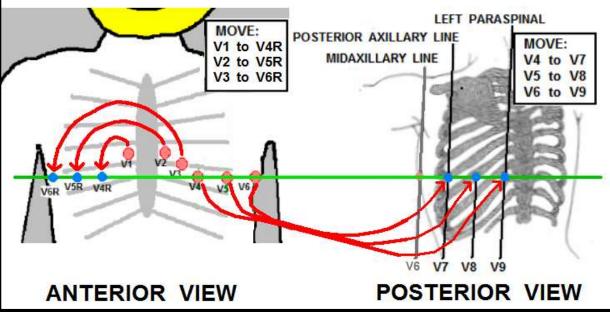


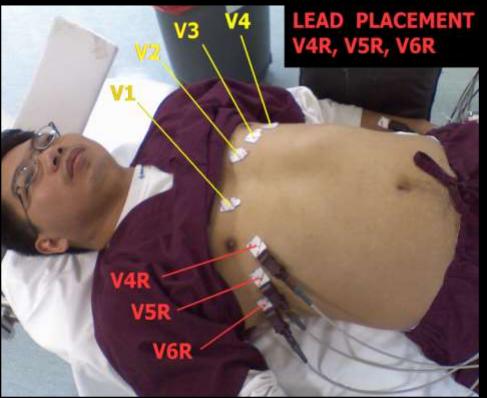
← The 12 Lead ECG

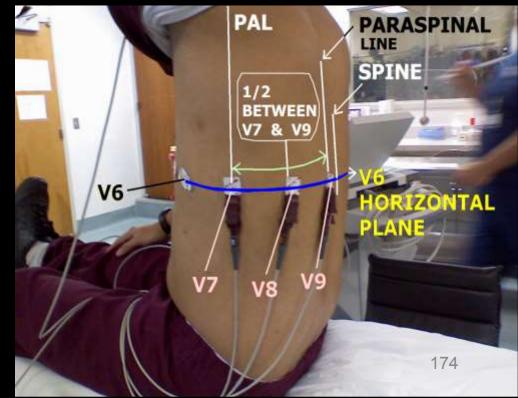
### The 18 Lead ECG ⇒

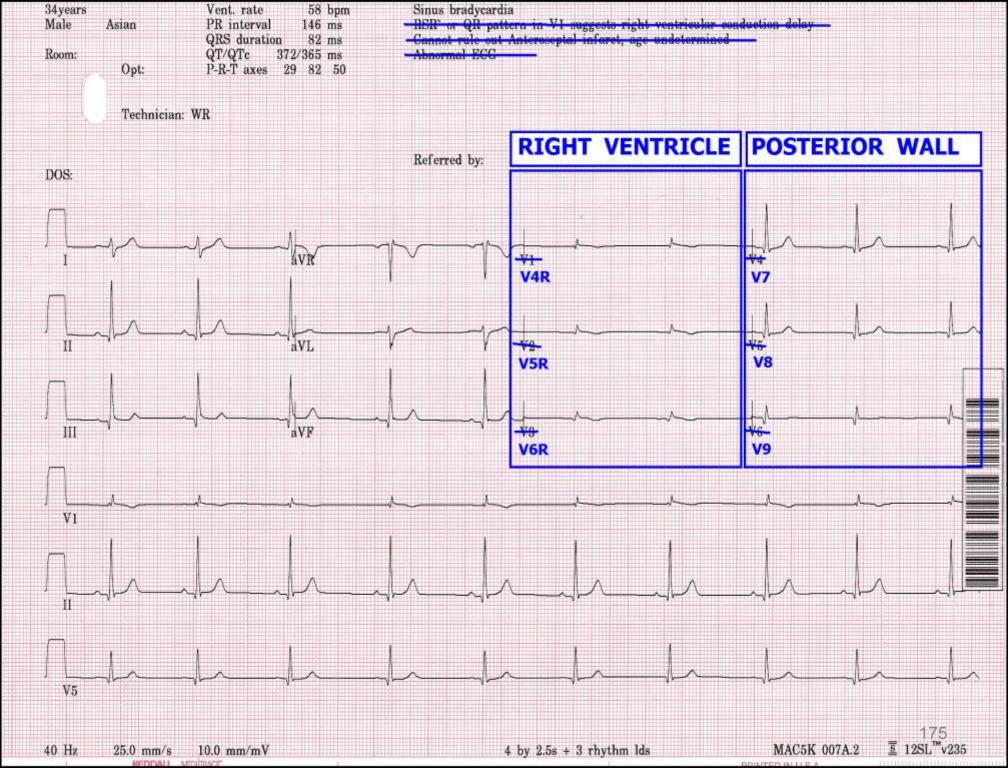


### HOW TO REPOSITION 6 CHEST LEADS to OBTAIN 3 R VENTRICLE and 3 POSTERIOR LEADS







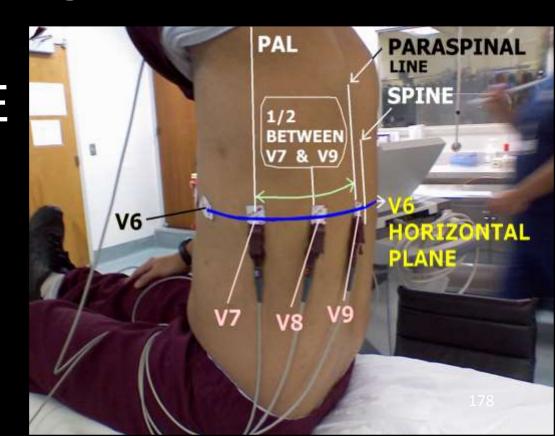


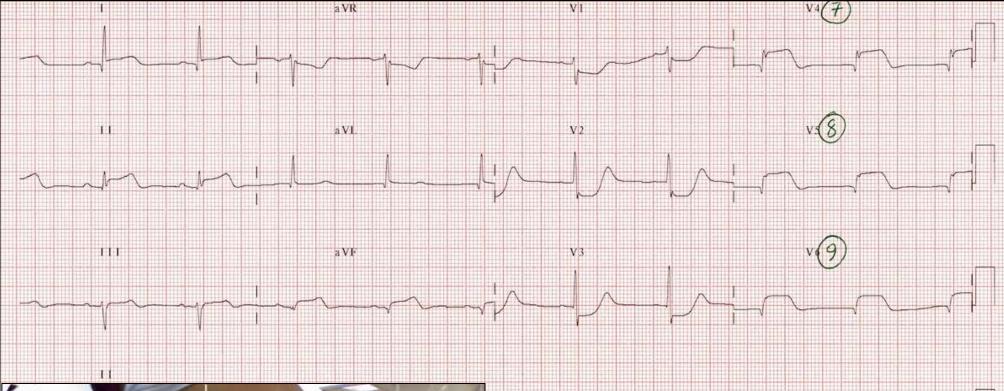
# POSTERIOR WALL MI usually accompanies INFERIOR and/or LATERAL WALL MI!!!

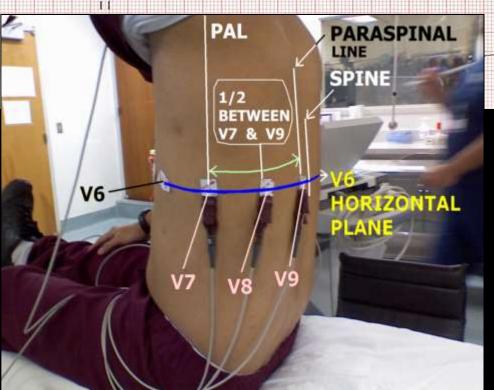
POSTERIOR WALL MI usually accompanies INFERIOR and/or LATERAL WALL MI !!! ... On rare occasions, we see isolated cases of POSTERIOR WALL MI

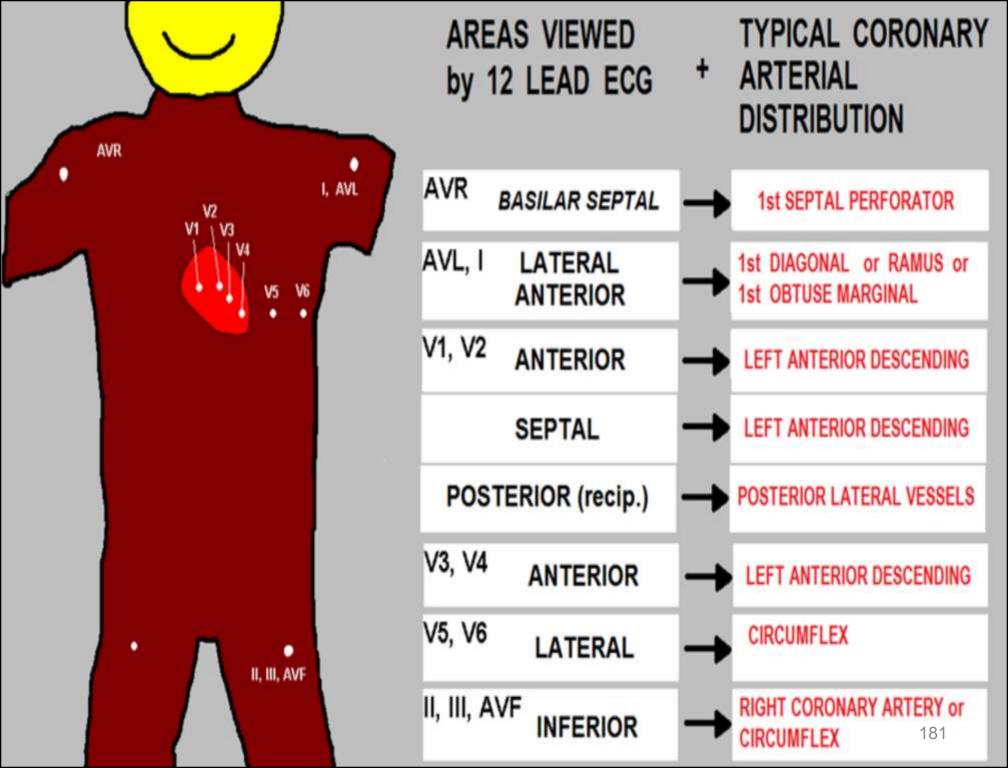
Whenever your patient's ECG exhibits ST DEPRESSION in any of the ANTERIOR LEADS (V1-V4), CONSIDER the possibility of POSTERIOR WALL STEMI!!

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



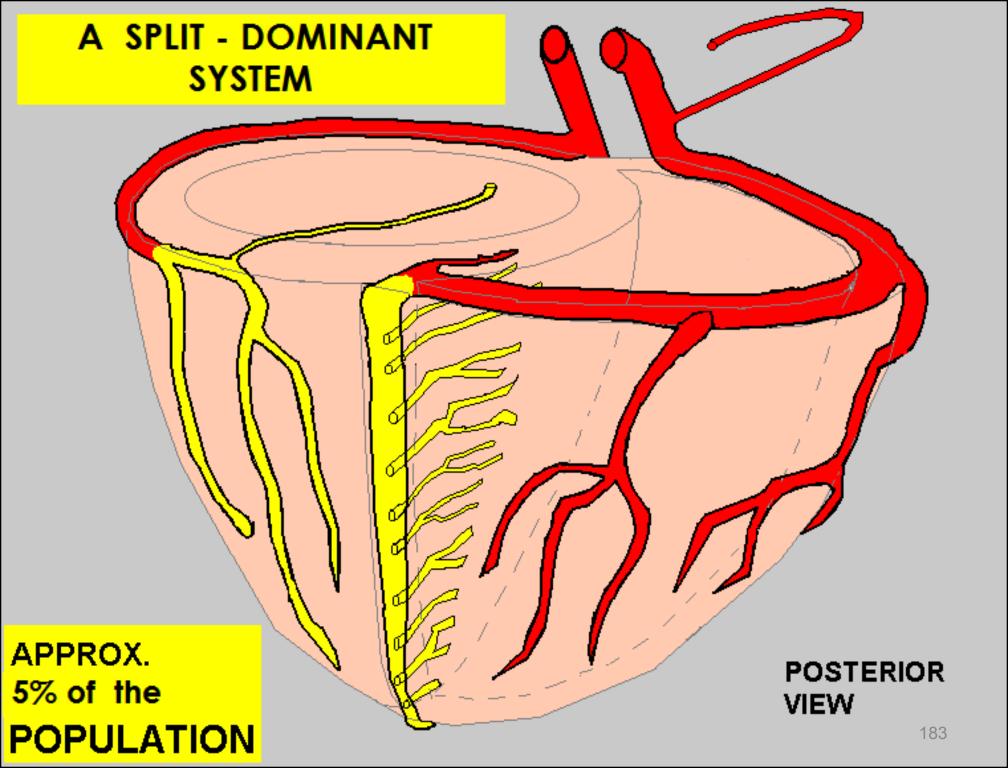






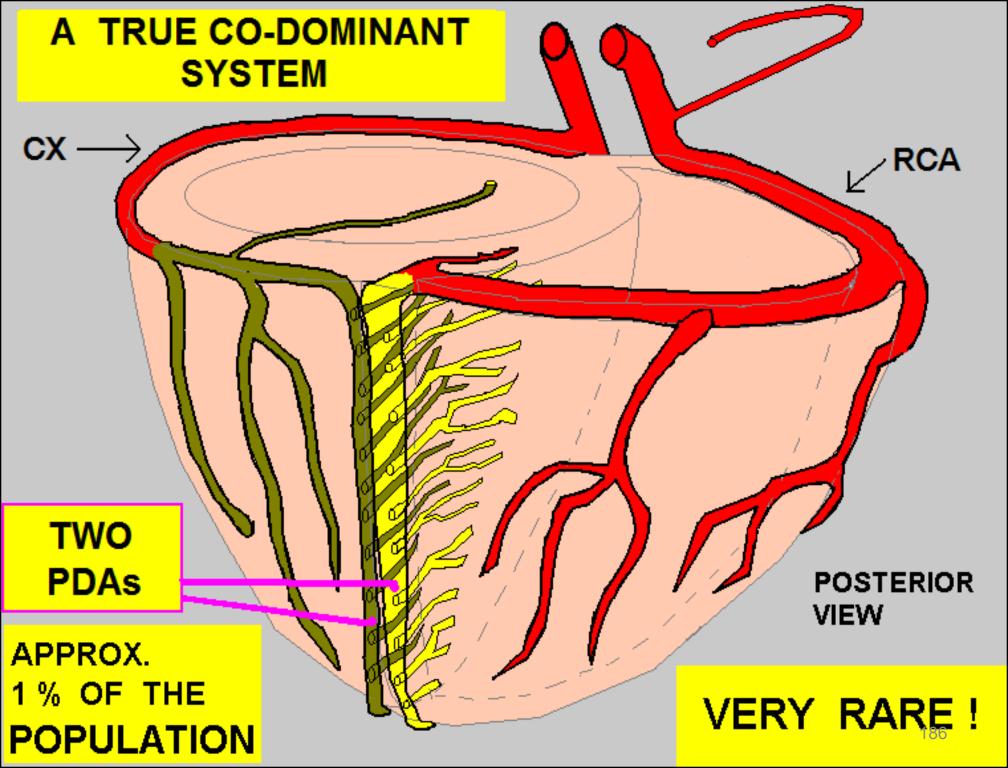
RIGHT DOMINANT and LEFT DOMINANT systems account for approximately 90 % of the population....

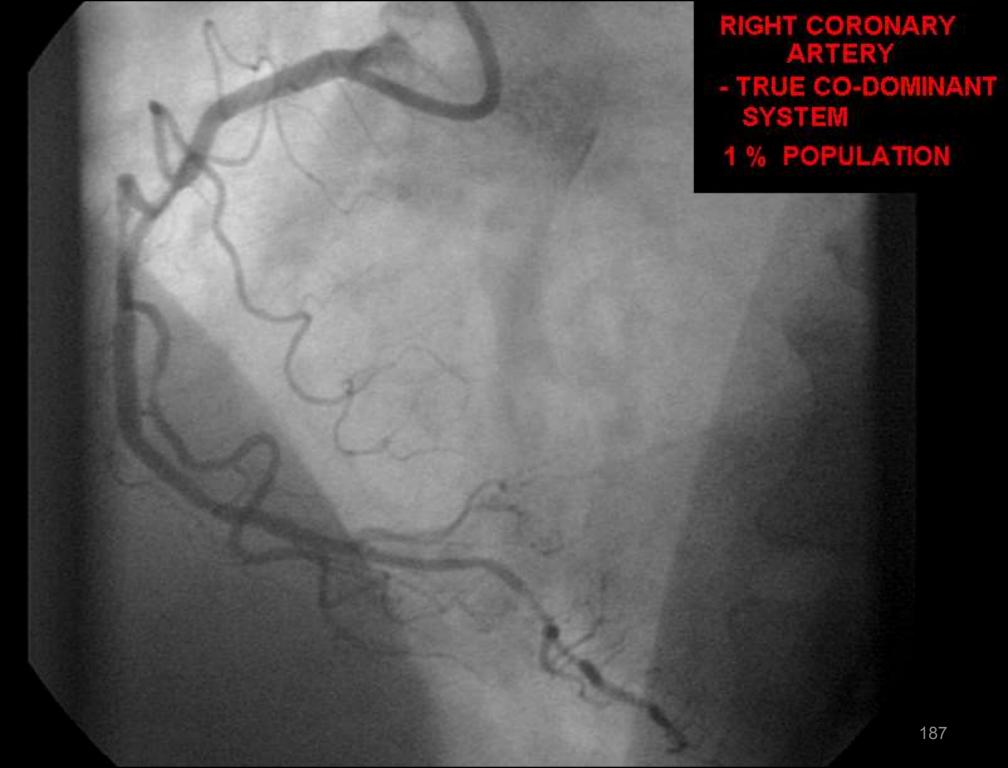
COMING UP ... some LESS COMMON variations that comprise the remaining 10%...

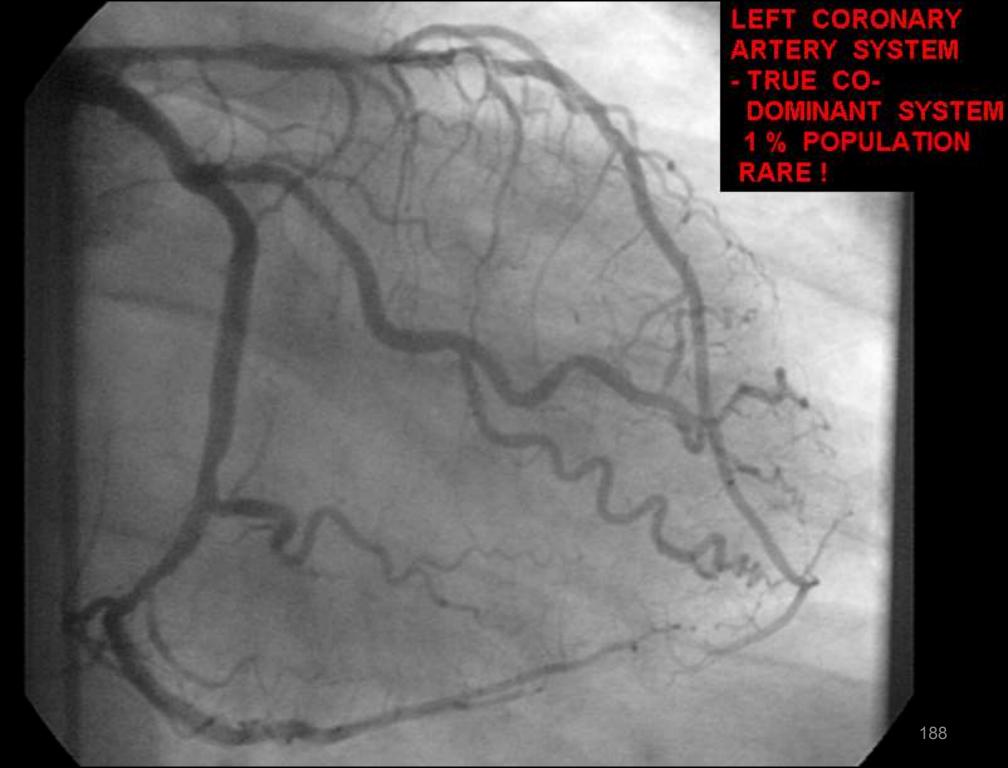


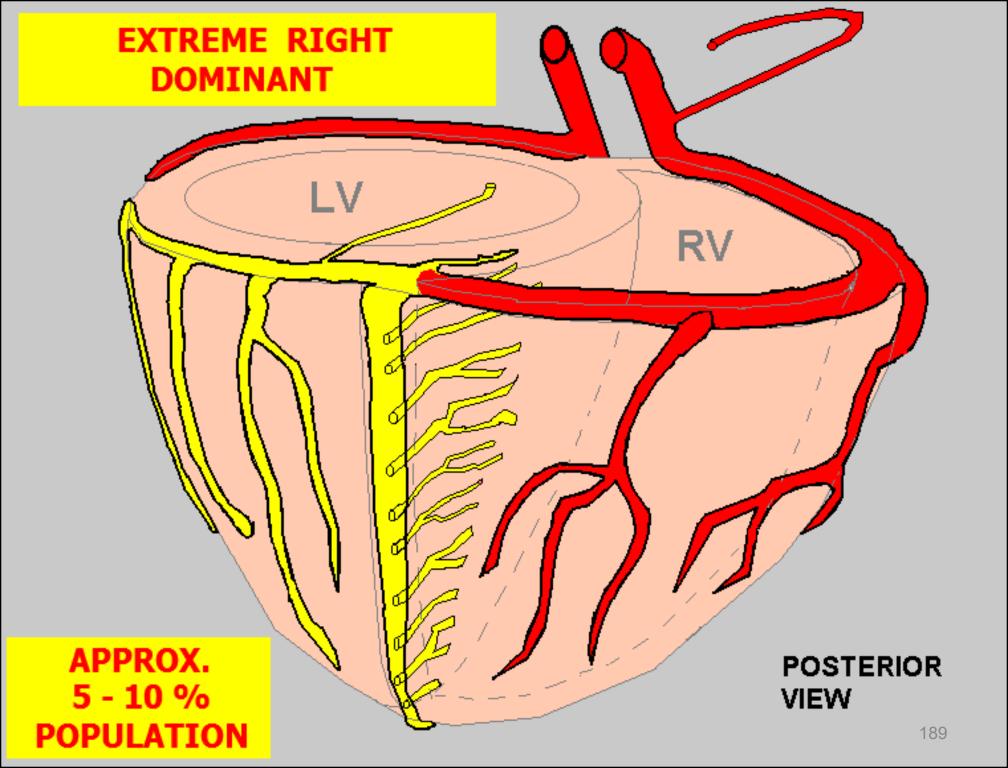


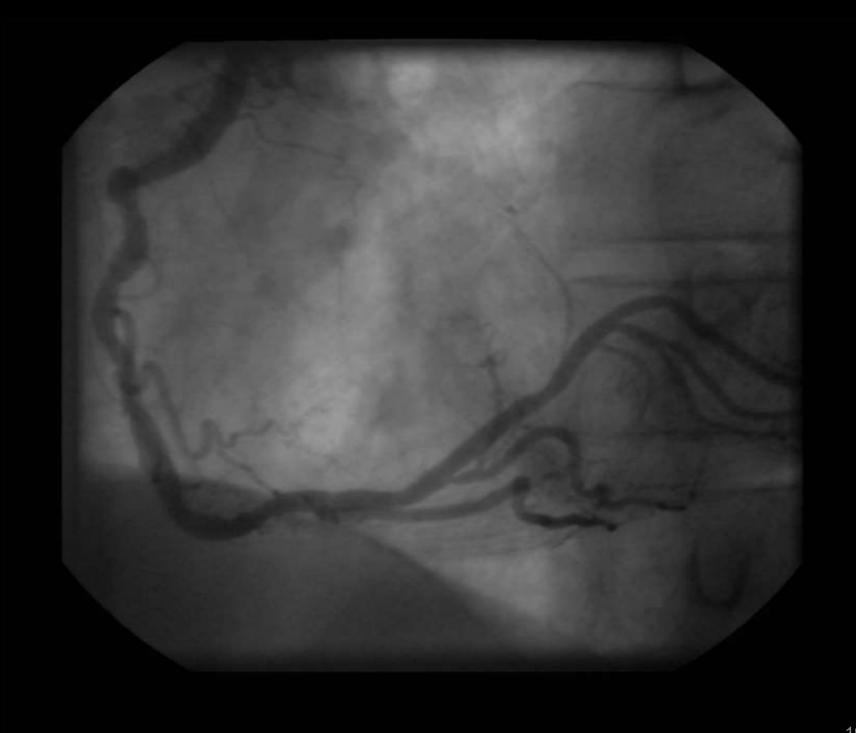




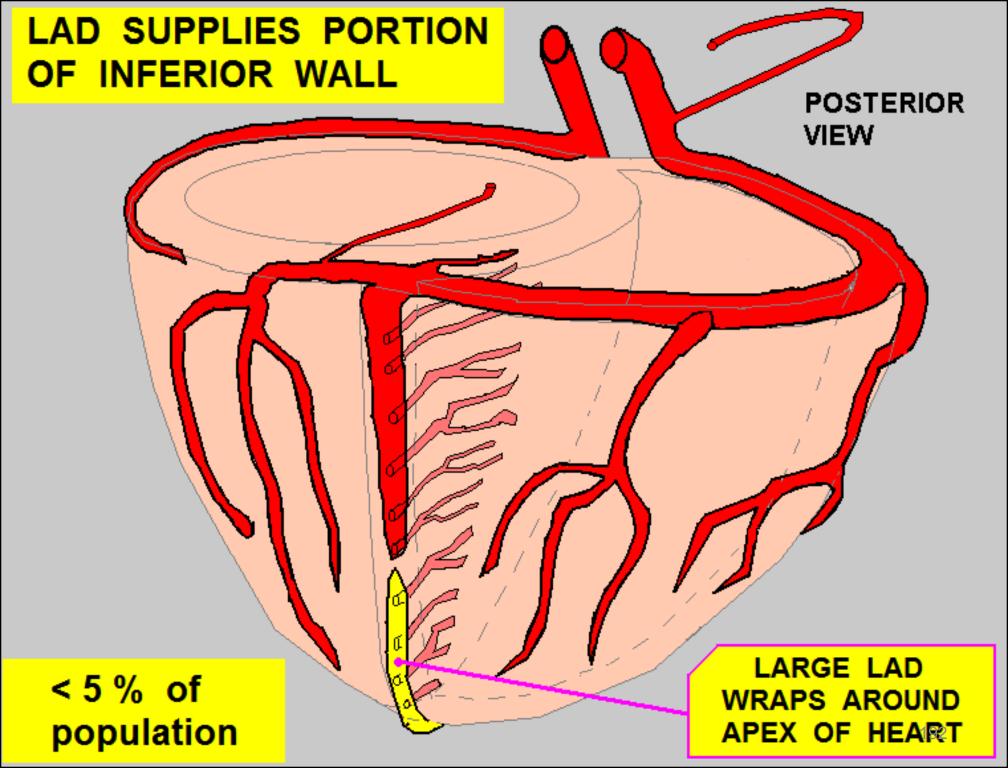






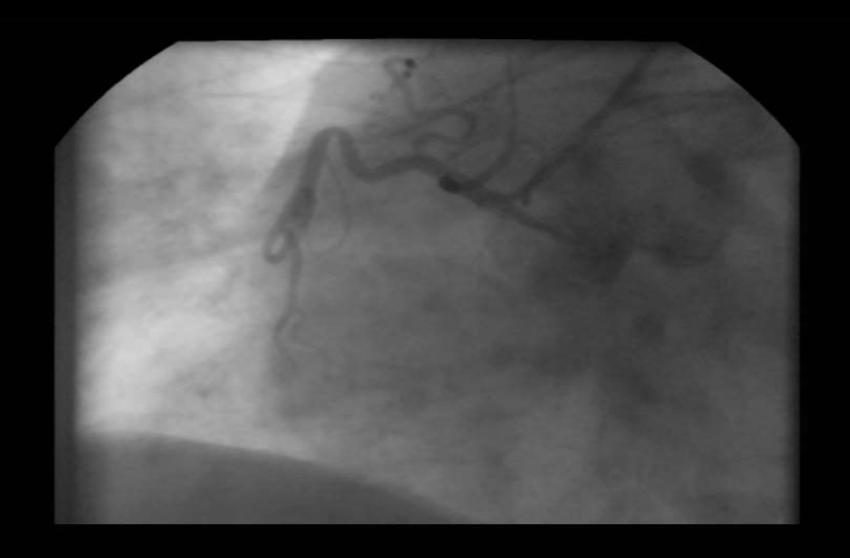




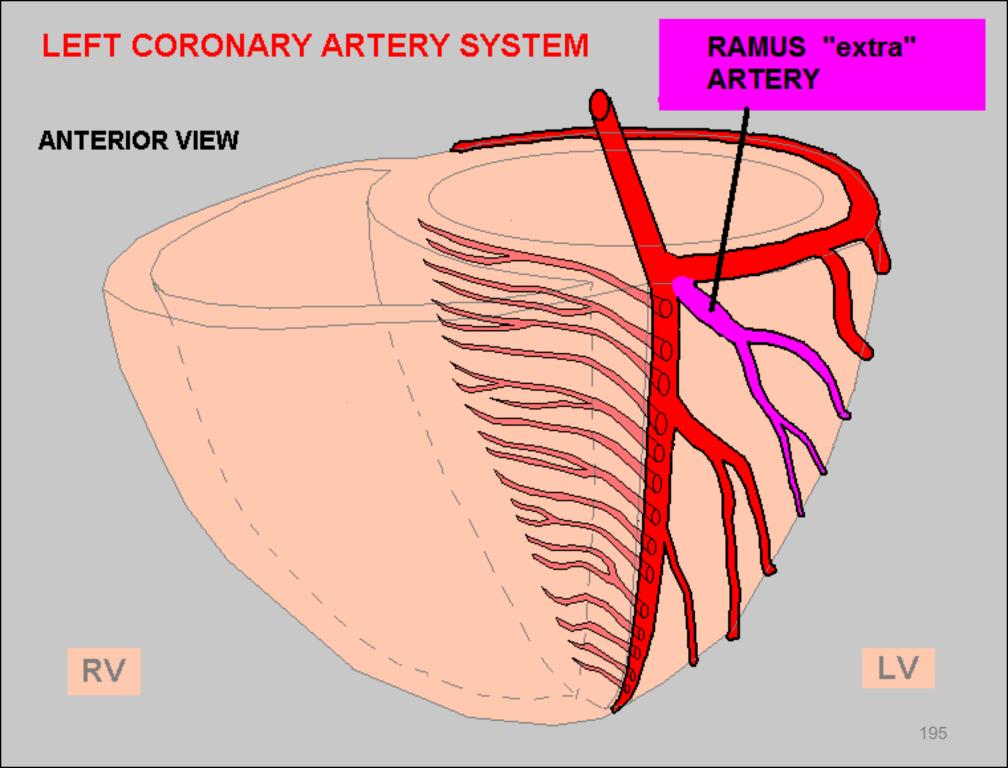


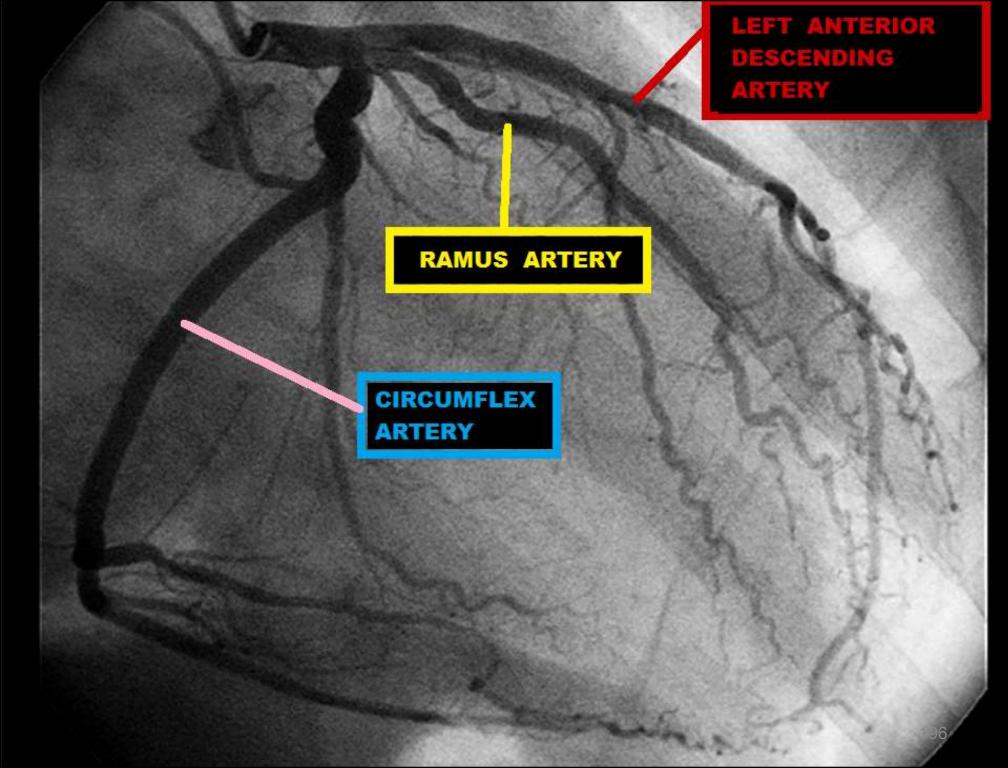
# ANTERIOR VIEW

LEFT ANTERIOR DESCENDING artery wraps around apex of heart and supplies INFERIOR WALL



SMALL, NON-DOMINANT RCA





# CORODARY

ARTERY

ADOMALITIES

# CIRCUMFLEX

Originales From the

RCA



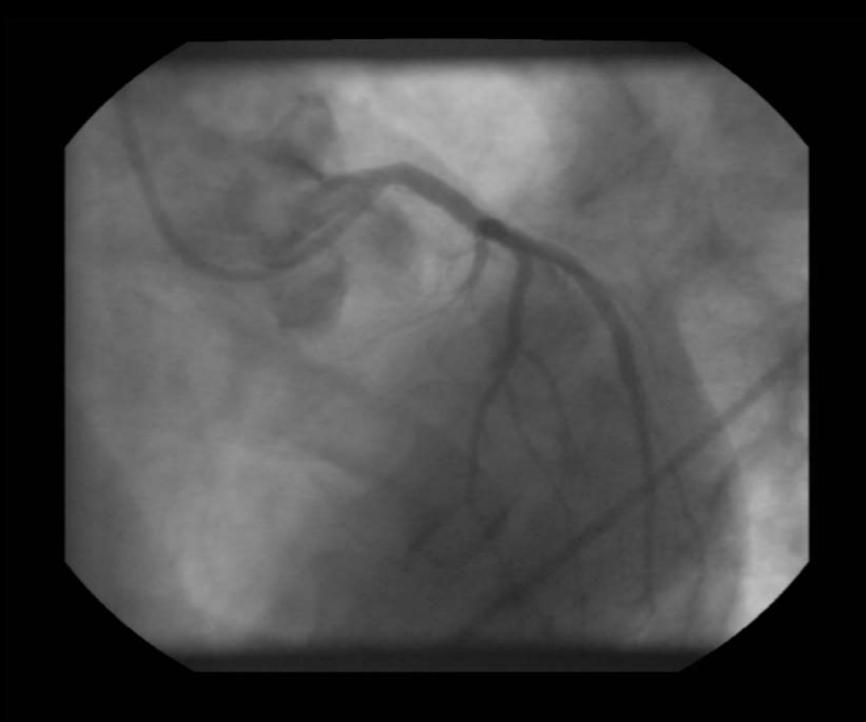


LAD

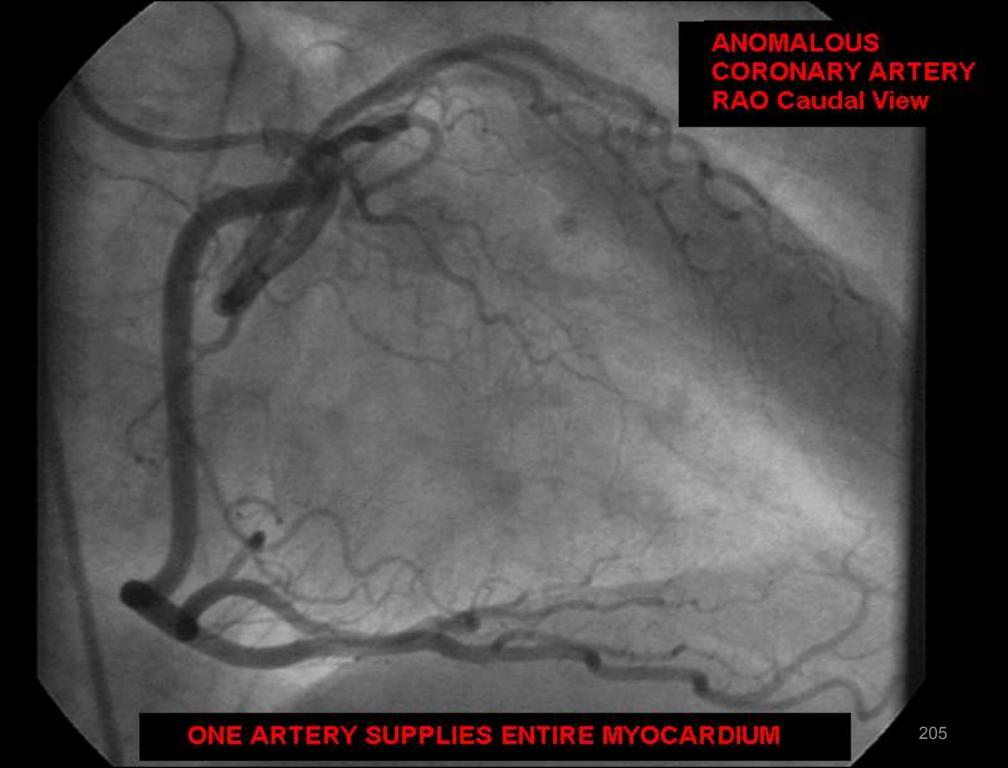
originates from

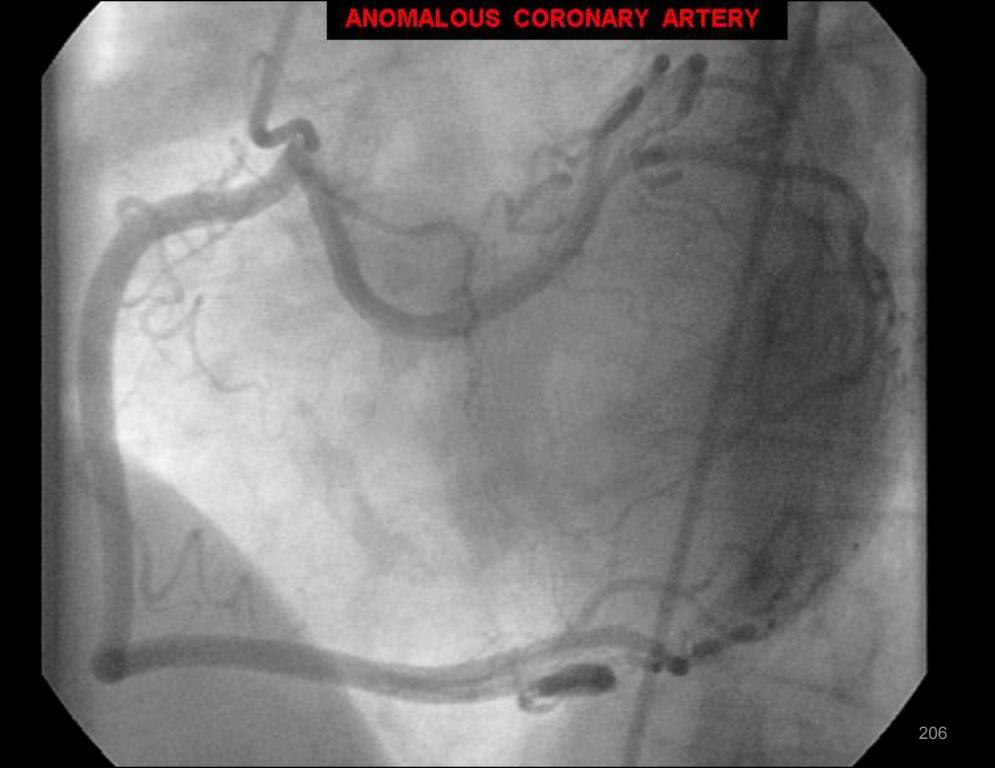
RCA





SINGLE ARTERY SUPPLIES EDTRE HEART







# SIGNIFICANT INCREASE OF SUDDEN DEATH

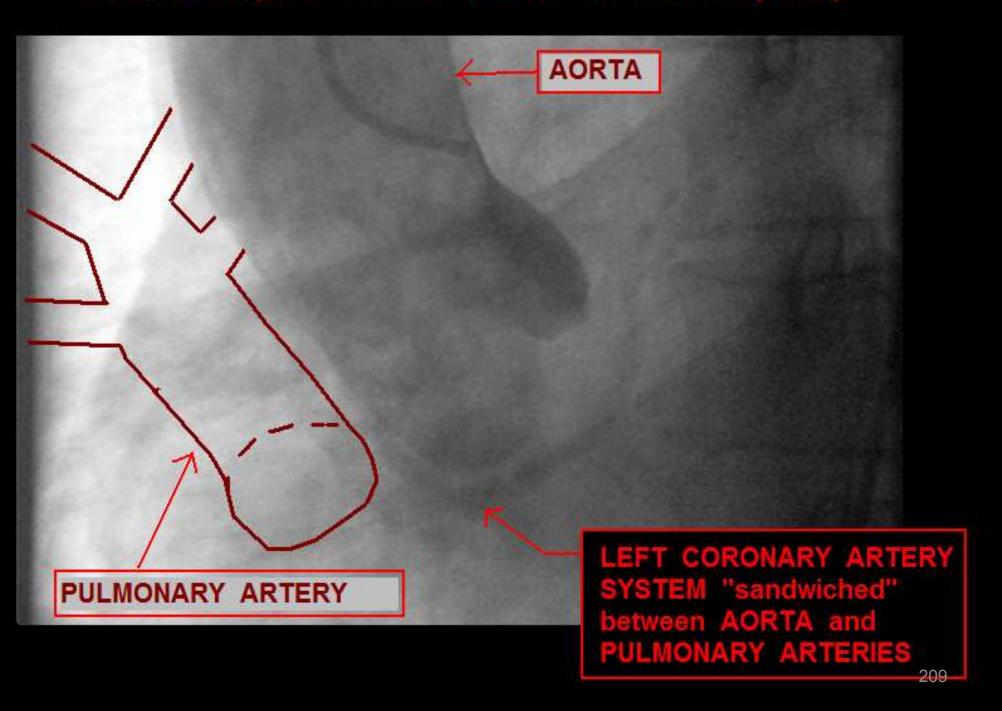
for people with anomalous coronary artery

- due to constriction of the circumflex branch as it wraps around AORTA
- this condition can be corrected surgically.

#### Aortic Root Injection of Patient With Anomalous Coronary Artery



#### Aortic Root Injection of Patient With Anomalous Coronary Artery





#### The Cardiac Electrical System

#### SINUS NODE

- INHERENT RATE 60 100
- BLOOD SUPPLY:
  - INFERIOR WALL (RCA) or
  - LATERAL WALL (CIRC.)

#### **AV NODE**

- INHERENT RATE 40 60
- BLOOD SUPPLY:
  - INFERIOR WALL (RCA or CIRC.)

#### BUNDLE OF HIS

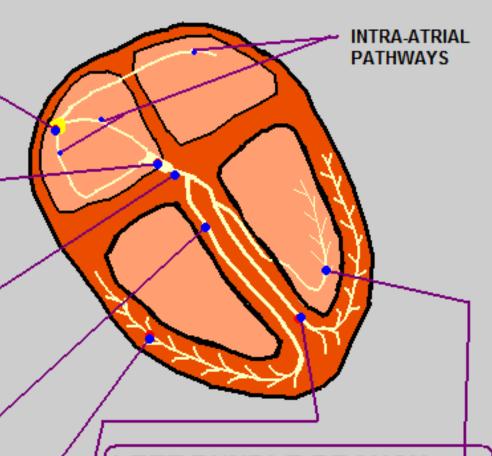
- INHERENT RATE 40 60
- BLOOD SUPPLY:
  - ANTERIOR WALL (LAD)

#### RIGHT BUNDLE BRANCH

- BLOOD SUPPLY:
  - ANTERIOR WALL (LAD)

#### PURKINJE FIBERS

INHERENT RATE 1-40



#### LEFT BUNDLE BRANCH

- ANTERIOR FASCICLE
- BLOOD SUPPLY:
  - ANTERIOR WALL (LAD)

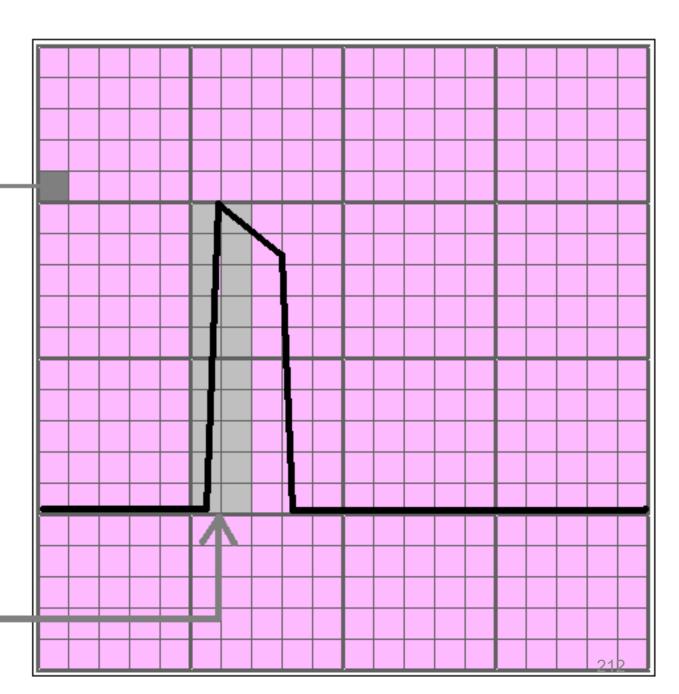
#### POSTERIOR FASCICLE

- BLOOD SUPPLY:
  - INFERIOR WALL (RCA or GIRC)

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



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



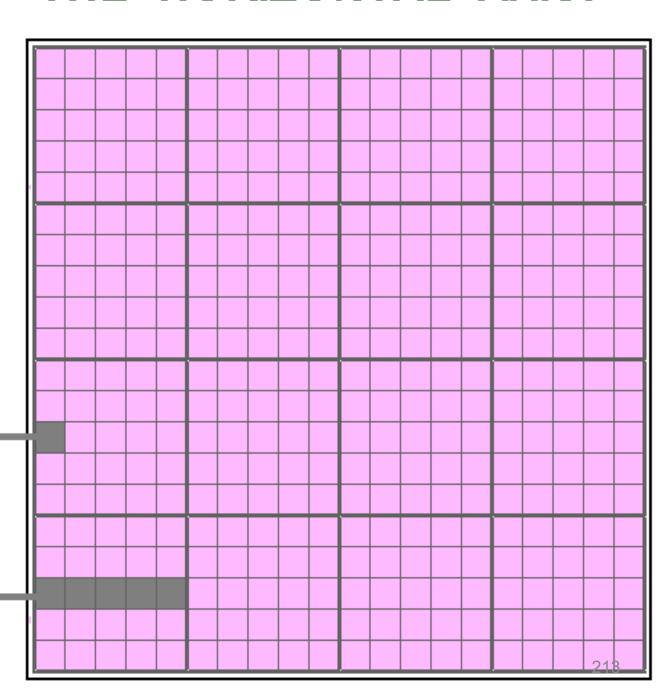
#### ECG PAPER - THE HORIZONTAL AXIS:

THE HORIZONTAL AXIS REPRESENTS TIME...

STANDARD SPEED FOR RECORDING ADULT EKGs = 25 mm / SECOND

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

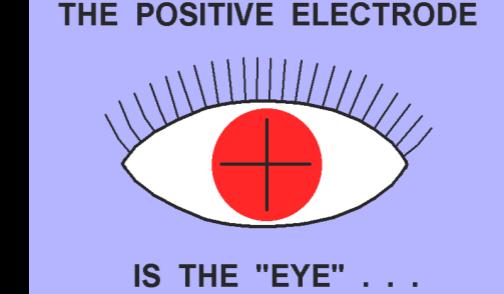
5 SMALL BOXES = .20 SECONDS, or 200 MILLISECONDS (200 ms)

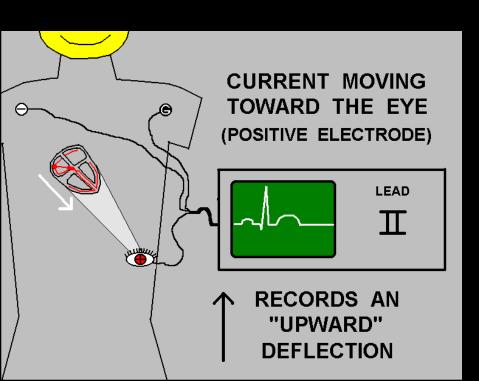


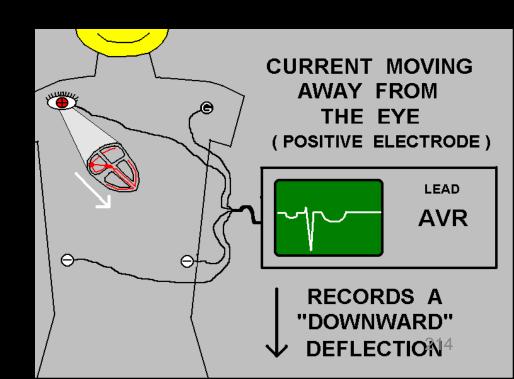
#### THE ECG MACHINE

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

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

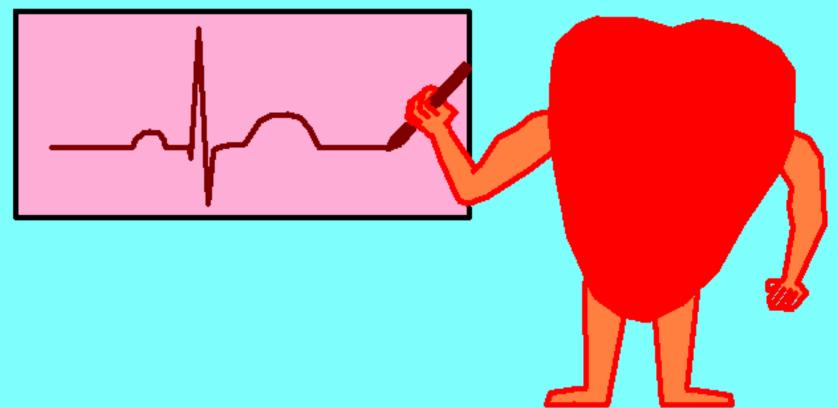




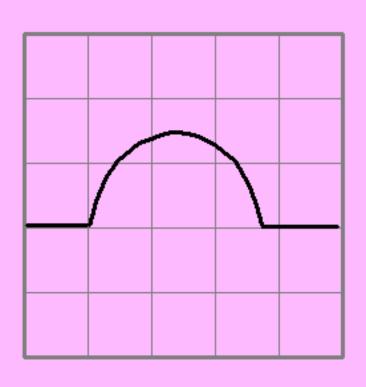


# PUTTING IT ALL ON PAPER...

WAVEFORMS and INTERVALS . . .



- SHOULD BE UPRIGHT, CONVEX-SHAPED DOME IN ALL LEADS EXCEPT AVR and V1
- SHOULD BE LESS
   THAN .2 mv
   (2 mm) HIGH



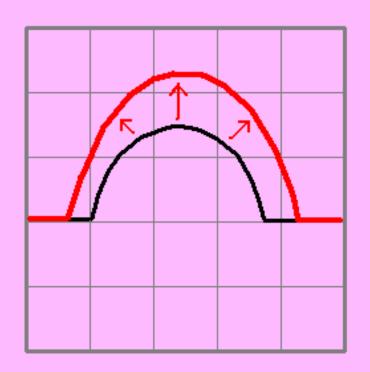
SHOULD BE LESS
 THAN 100 ms (2.5mm) LONG

When the P WAVE

is

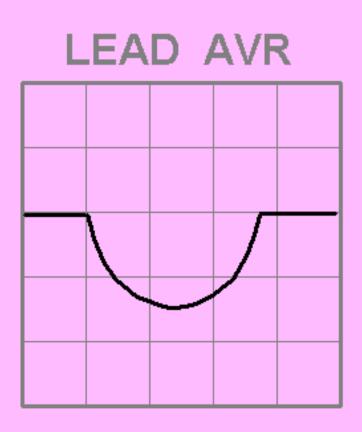
TOO LARGE

We think of



# ATRIAL HYPERTROPHY

- SHOULD BE INVERTED IN LEAD AVR



#### IN LEAD V1 MAY BE:

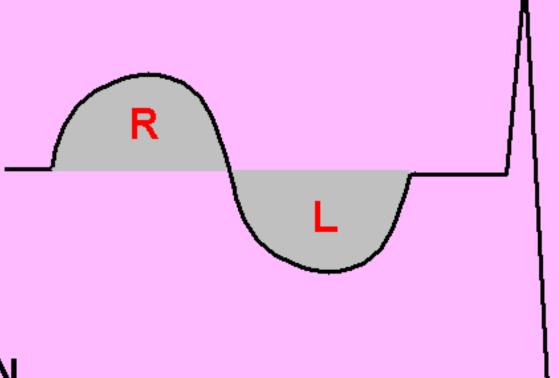
POSITIVE

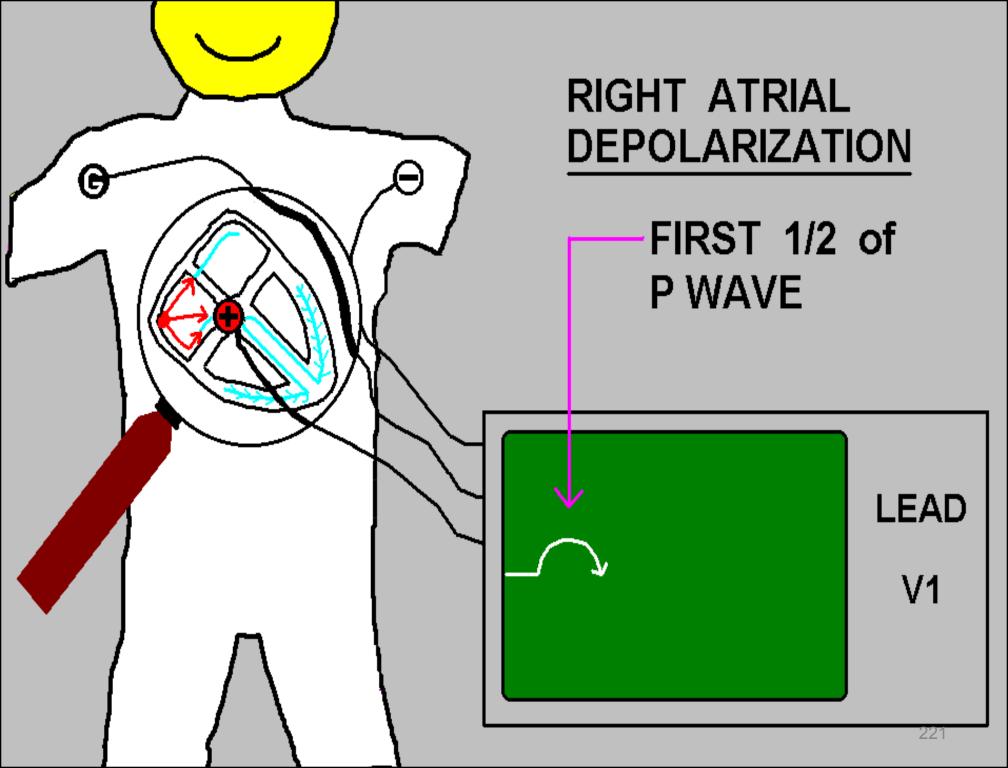


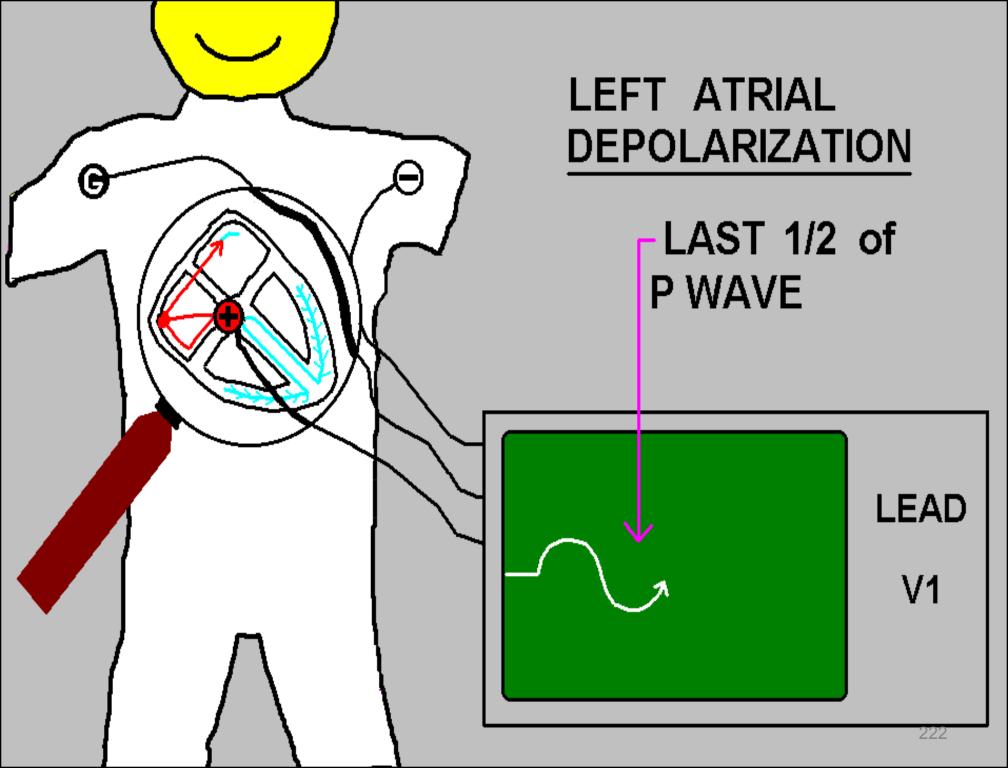
OR BI-PHASIC

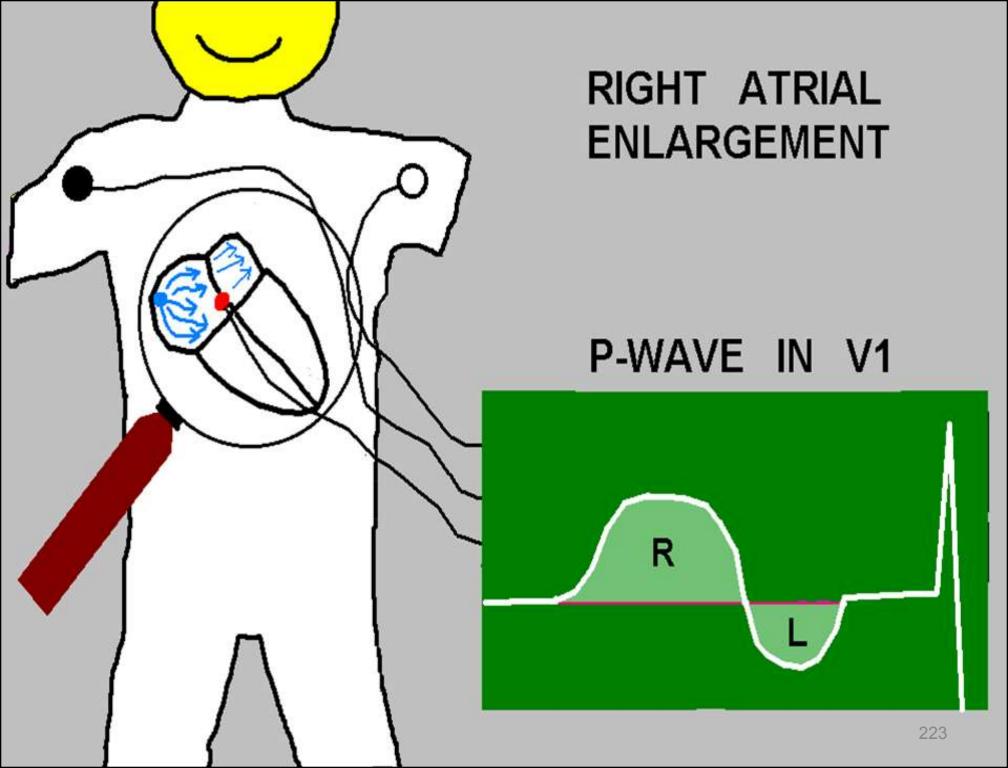


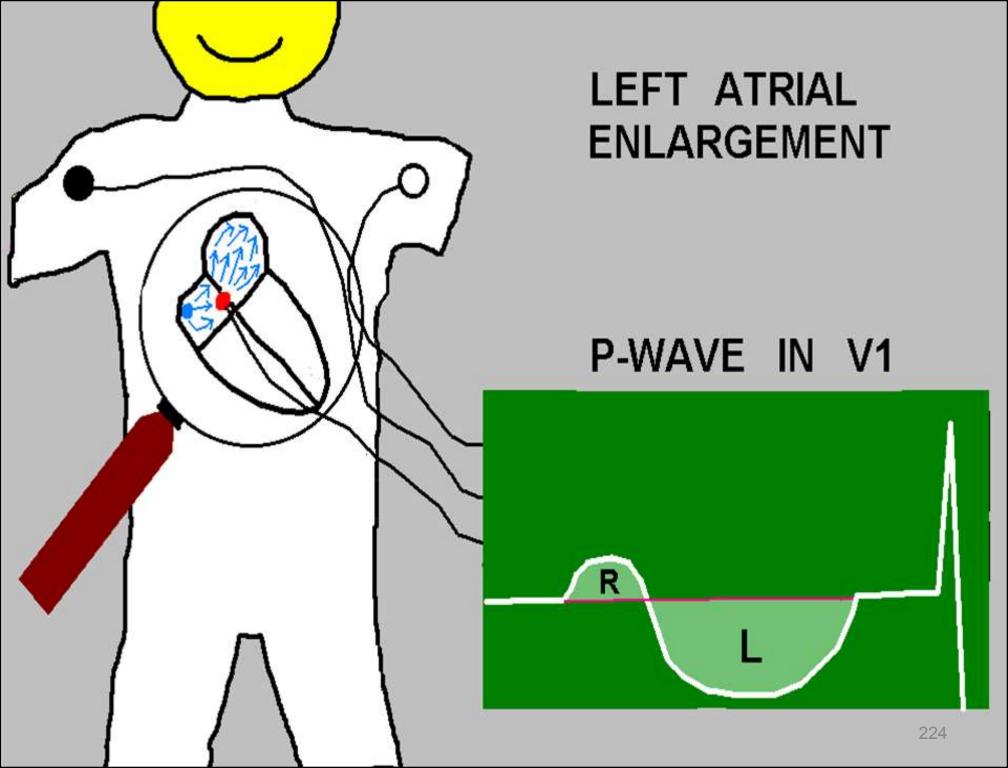
WHEN THE P
 WAVE IS BI PHASIC IN V1, IT DISPLAYS
 BOTH R and
 L ATRIAL
 DEPOLARIZATION











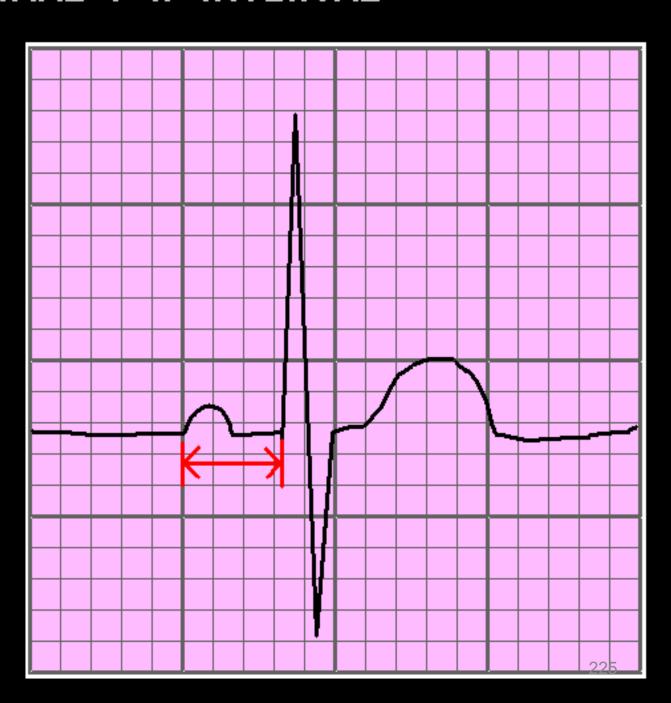
### NORMAL P-R INTERVAL

.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

#### THE P-R SEGMENT

SHOULD

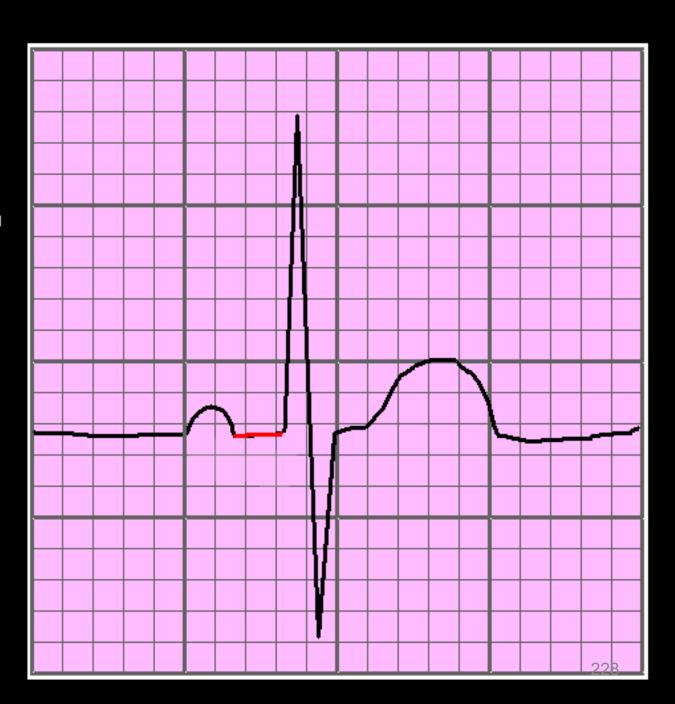
RETURN TO

THE

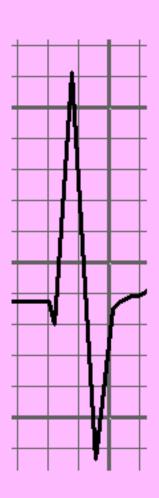
ISO-

**ELECTRIC** 

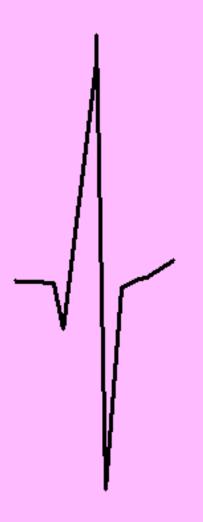
LINE.



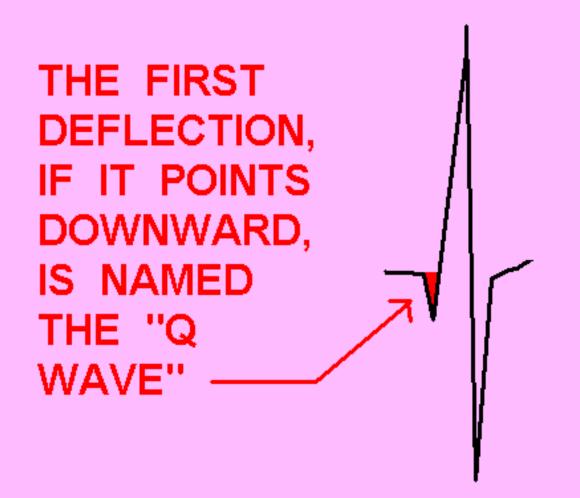
- MAY BE POSITIVE, NEGATIVE, OR BI- PHASIC, BASED ON THE LEAD VIEWED
- TOTAL WIDTH SHOULD BE LESS THAN 120 ms / or .12



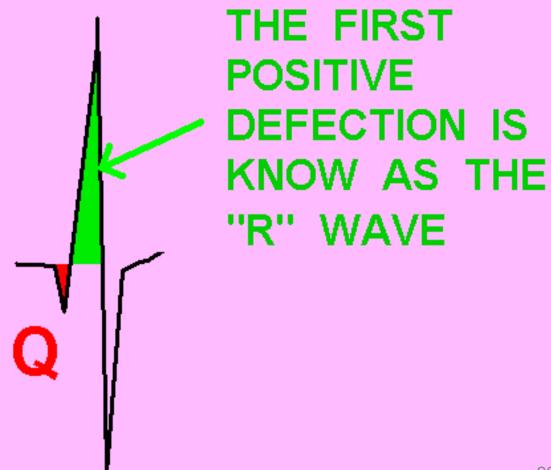
THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . .



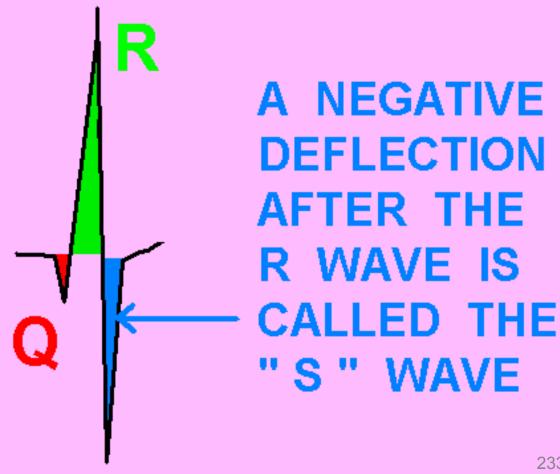
THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .



THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .

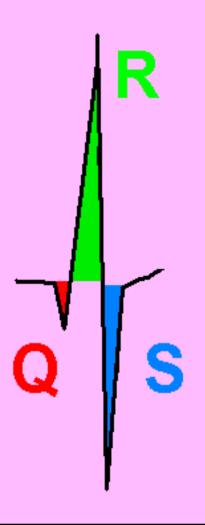


THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . .



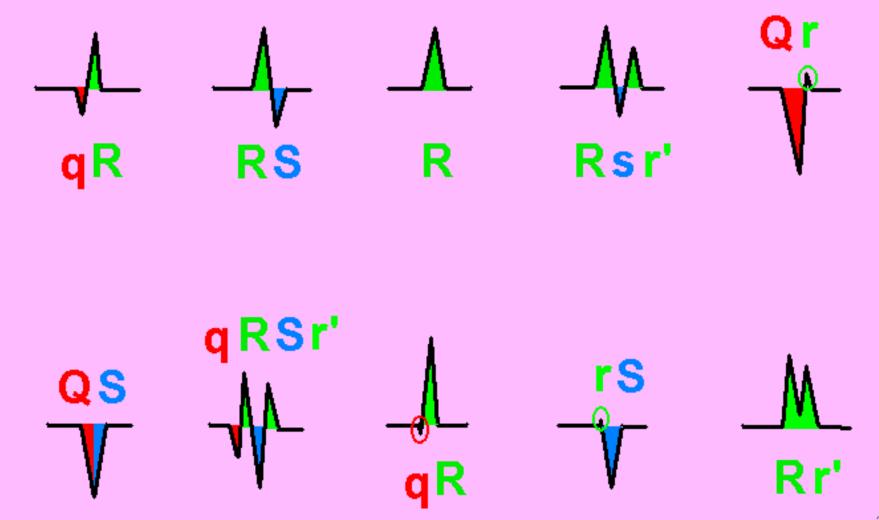
THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .

AND IS
THE <u>ONLY</u>
TRUE
"QRS"
COMPLEX



SOME OF THE OTHER VARIATIONS INCLUDE...

#### WHAT ARE THESE COMPLEXES ??



#### **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)

# Wide QRS present: QRSd > 120ms

 Determine RIGHT vs. LEFT Bundle Branch Block Pattern

## Simple "Turn Signal Method" . . .

THE "TURN SIGNAL METHOD" for identifying BUNDLE BRANCH BLOCK

٧1

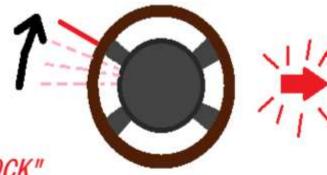
USE LEAD V1 for this technique

To make a RIGHT TURN

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

THINK:

"QRS points UP = RIGHT BUNDLE BRANCH BLOCK"





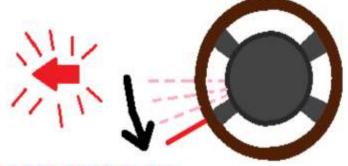
٧1



To make a LEFT TURN

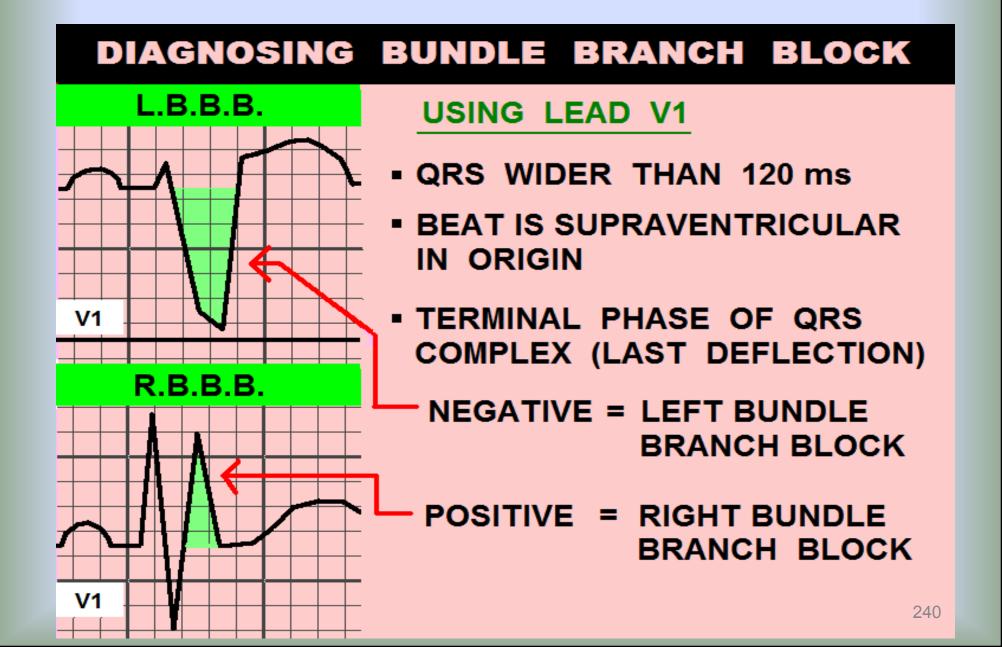
you push the turn signal lever DOWN . . . .

THINK:

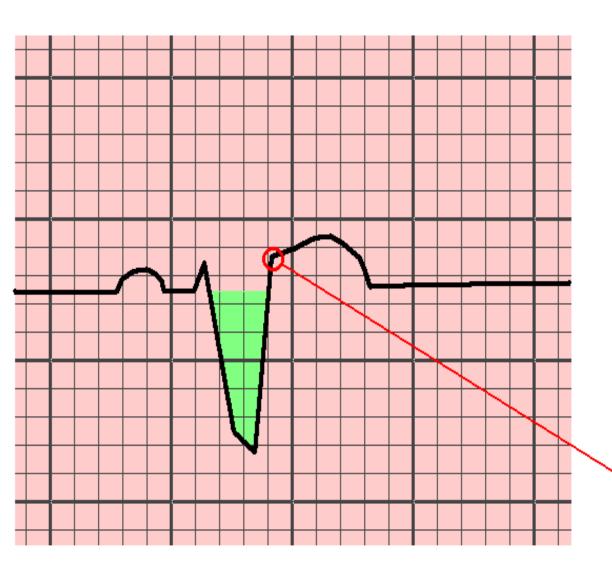


"QRS points DOWN = LEFT BUNDLE BRANCH BLOCK"

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

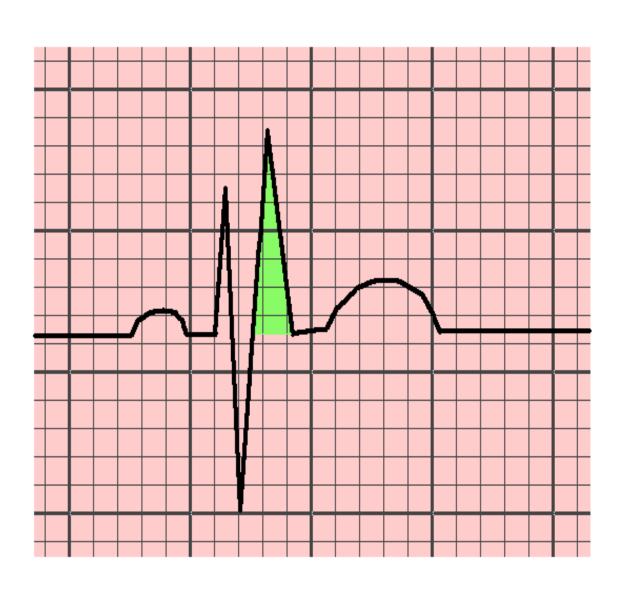


#### **DIAGNOSING LBBB IN LEAD V1:**



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

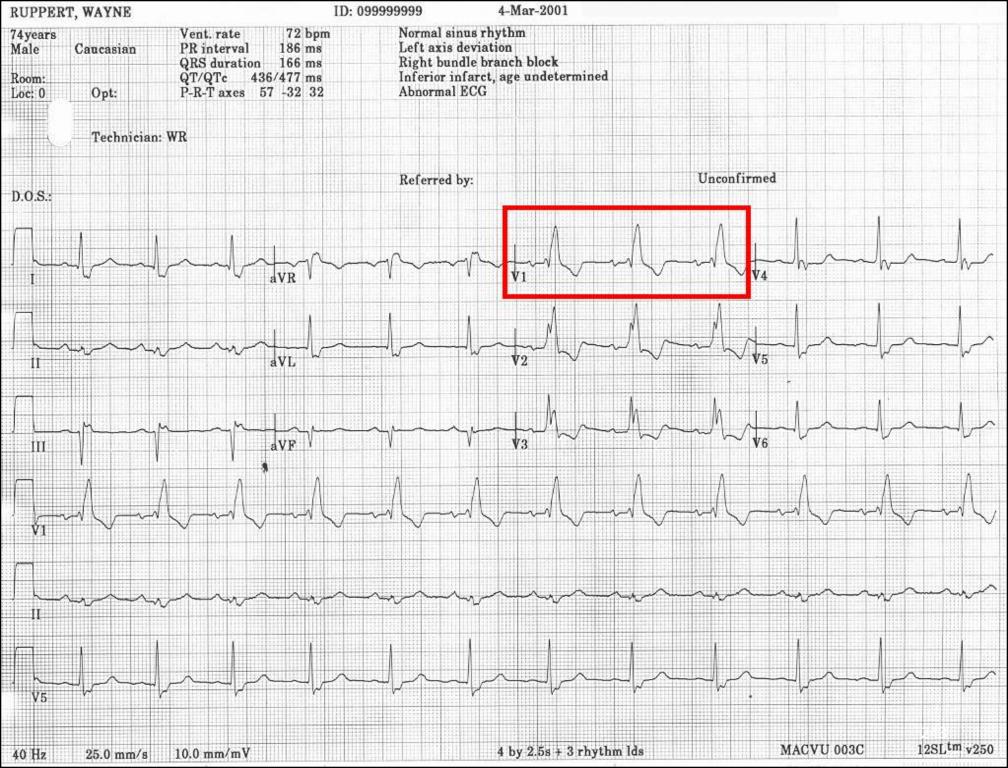
#### **DIAGNOSING RBBB IN LEAD V1:**



• WIDER THAN 120 ms (.12)

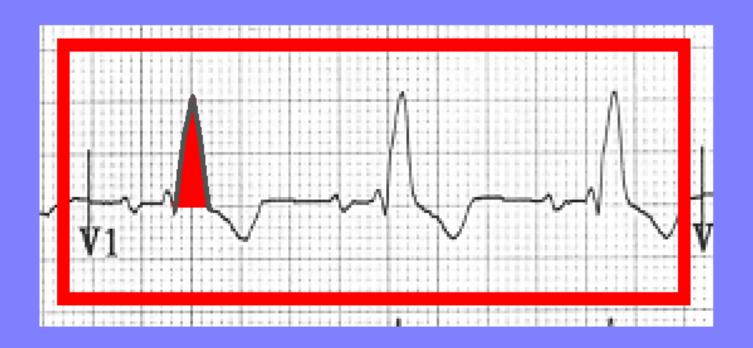
(or 3 little boxes)

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

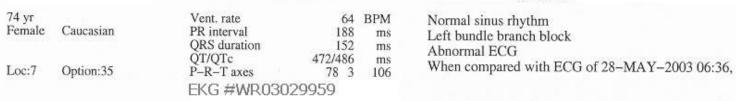


#### TERMINAL PHASE OF QRS IS

#### **POSITIVE**



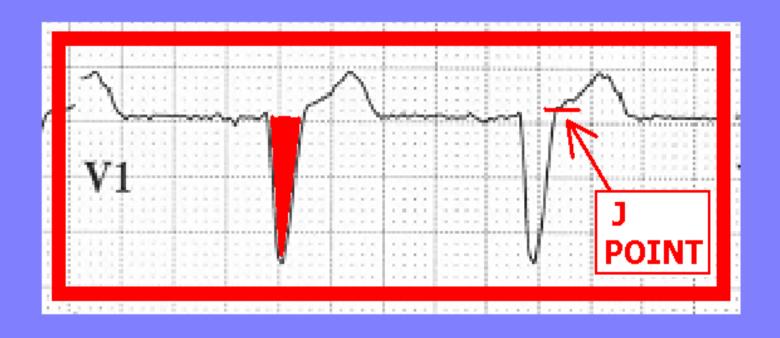
= RIGHT BUNDLE BRANCH BLOCK



Technician: WW



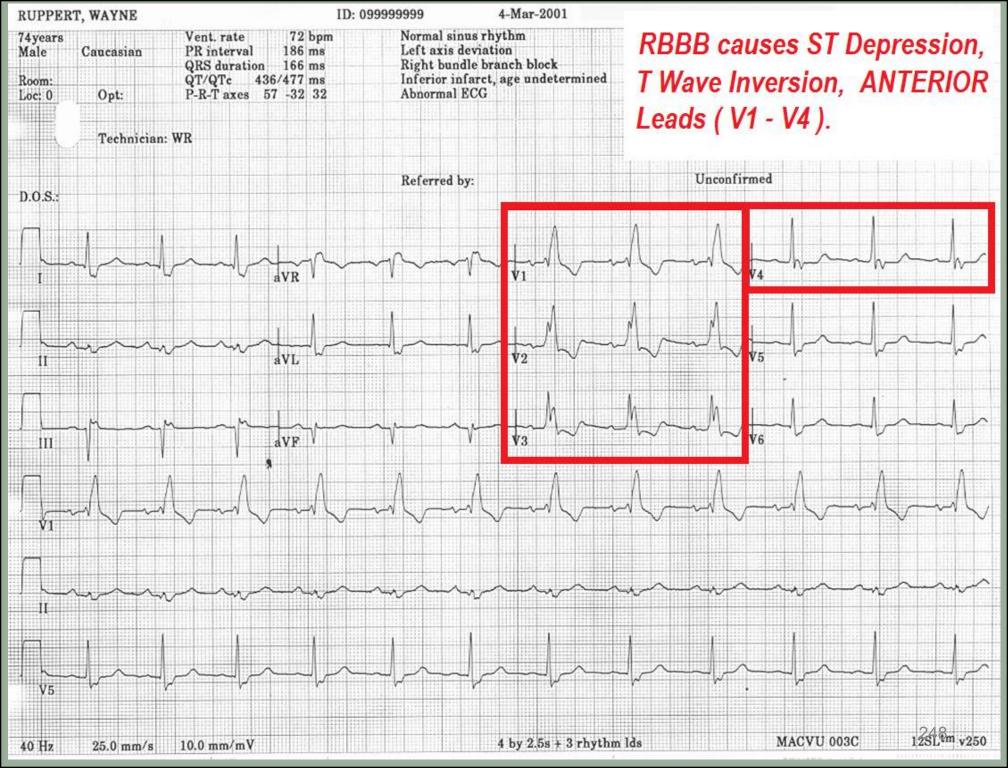
# TERMINAL PHASE OF QRS IS NEGATIVE



= LEFT BUNDLE
BRANCH BLOCK

## Wide QRS present: (QRSd > 120ms)

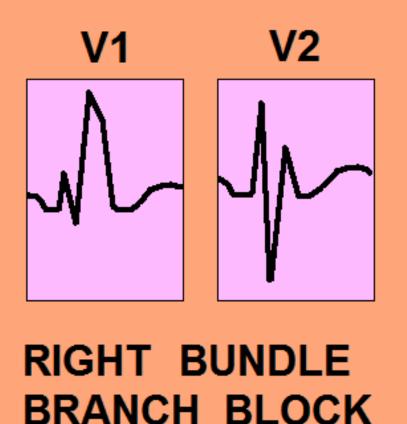
- When RIGHT Bundle Branch Block pattern is present:
  - Precordial Leads typically demonstrate ST
     Depression and T wave Inversion

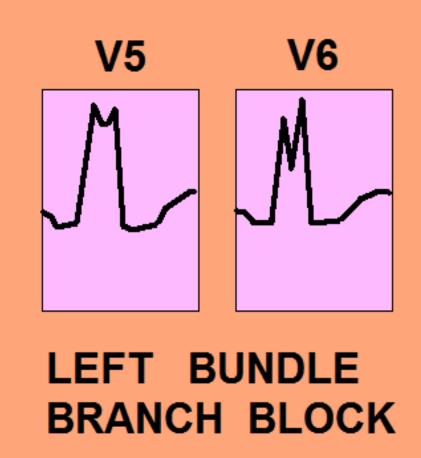


#### DIAGNOSING BUNDLE BRANCH BLOCK

USING LEADS V1, V2, and V5, V6:

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





#### **QRS HEIGHT**

is a reflection of the QRS AMPLITUDE.

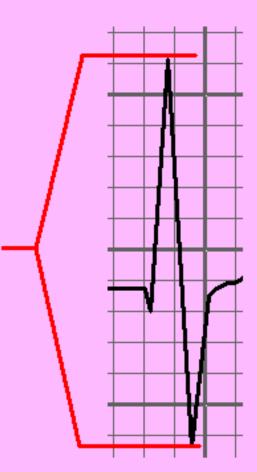
The NORMAL QRS AMPLITUDE varies from one lead to another...



#### **QRS AMPLITUDE**

#### is influenced by:

- age
- physical fitness
- body size
- conduction system disorders
- chamber hypertrophy



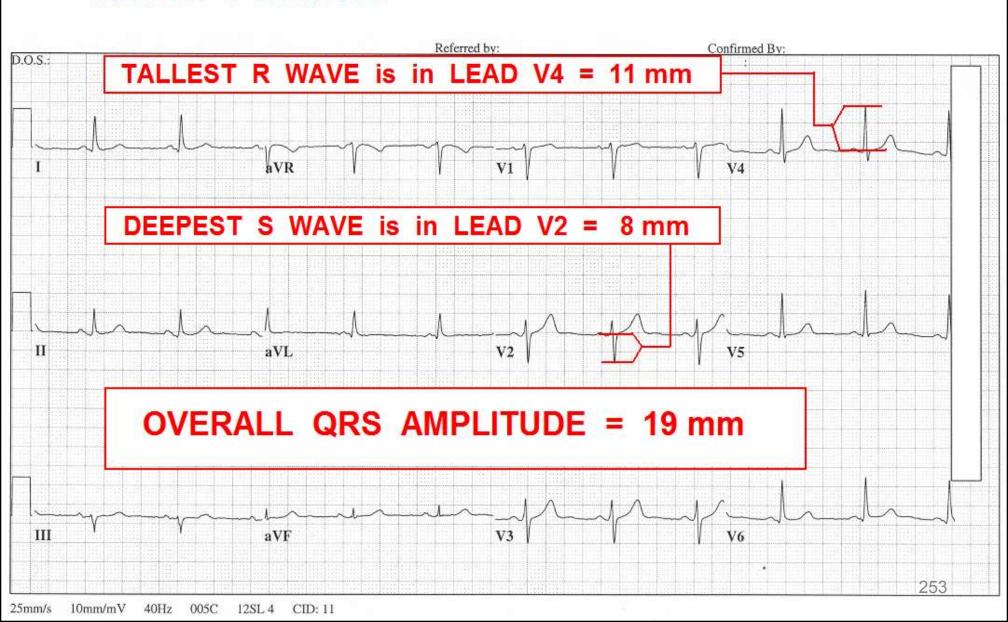
#### **QRS AMPLITUDE**

is measured by finding the TALLEST POSITIVE DEFLECTION (R WAVE) and the DEEPEST **NEGATIVE DEFLECTION** (SWAVE) on the 12 LEAD EKG and ADDING THE VALUES TOGETHER



#### MEASURING THE "OVERALL QRS AMPLITUDE"

Add the SIZE of the TALLEST R WAVE to the SIZE of the DEEPEST S WAVE....

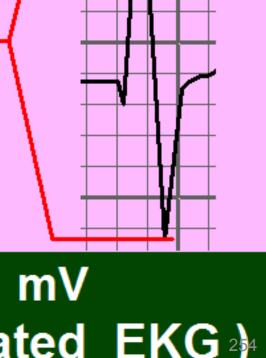


#### **QRS AMPLITUDE**

MAXIMUM NORMAL VALUES are difficult to define due to differences in PATIENT AGE, BODY - SIZE, and FITNESS.

HOWEVER A GENERAL VALUE GUIDELINE IS: 3.0 mV

(30 mm on normally calibrated EKG



#### **OVERALL QRS AMPLITUDE TOO HIGH:**

(GREATER THAN 3.0 mV / 30 mm)

#### THINK:

VENTRICULAR HYPERTROPHY

### **Hypertrophy "Cheats":**

- WHEN QRS COMPLEX(ES) "SPEAR"
   OUTSIDE OF THEIR SPACE.
- WHEN QRS COMPLEXES SPEAR THROUGH OTHER LEADS!

 Vent. rate
 90 BPM

 PR interval
 136 ms

 QRS duration
 94 ms

 QT/QTc
 378/462 ms

 P-R-T axes
 77 123 58

Normal sinus rhythm
Right atrial enlargement
Right axis deviation
Incomplete right bundle branch block, plus right ventricular hypertrophy
NORMAL SINUS INFERIOR LATERAL CHANGES
Abnormal ECG

#### EKG CLASS #WRO3616941

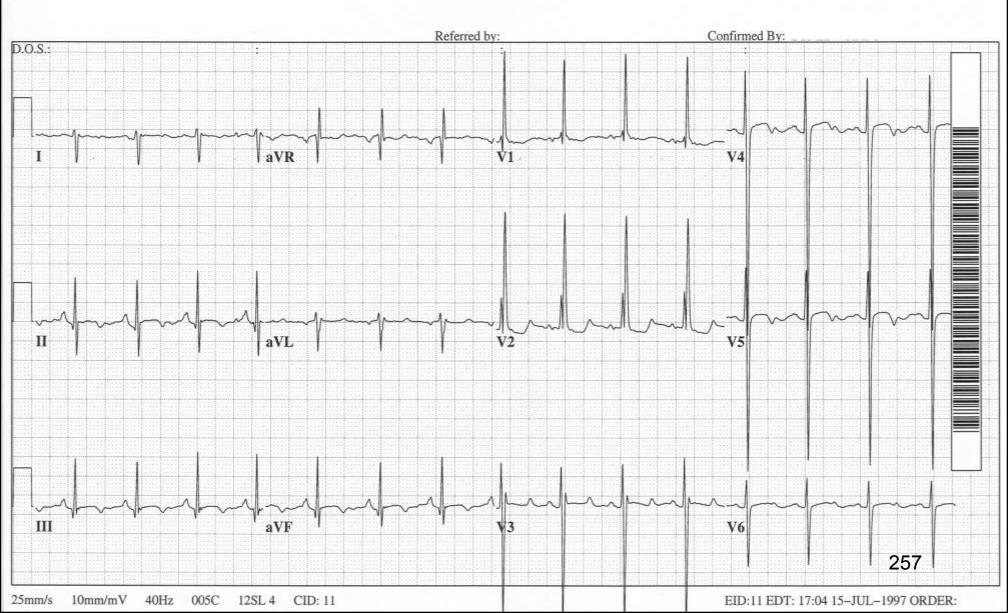
17 yr Male

Loc:3

Room:ER

Black

Option:16



Normal sinus rhythm

Left atrial enlargement

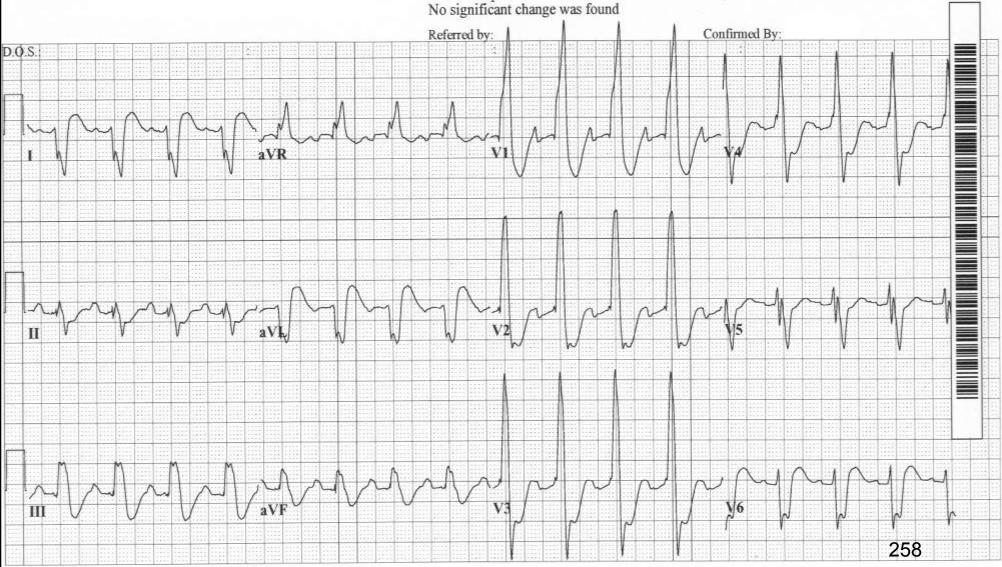
100 BPM 53 yr Vent. rate 198 Male PR interval Caucasian ms 186 QRS duration ms Room: ER S3 QT/QTc 380/490 ms P-R-T axes 79 163 -20Loc:3 Option:18

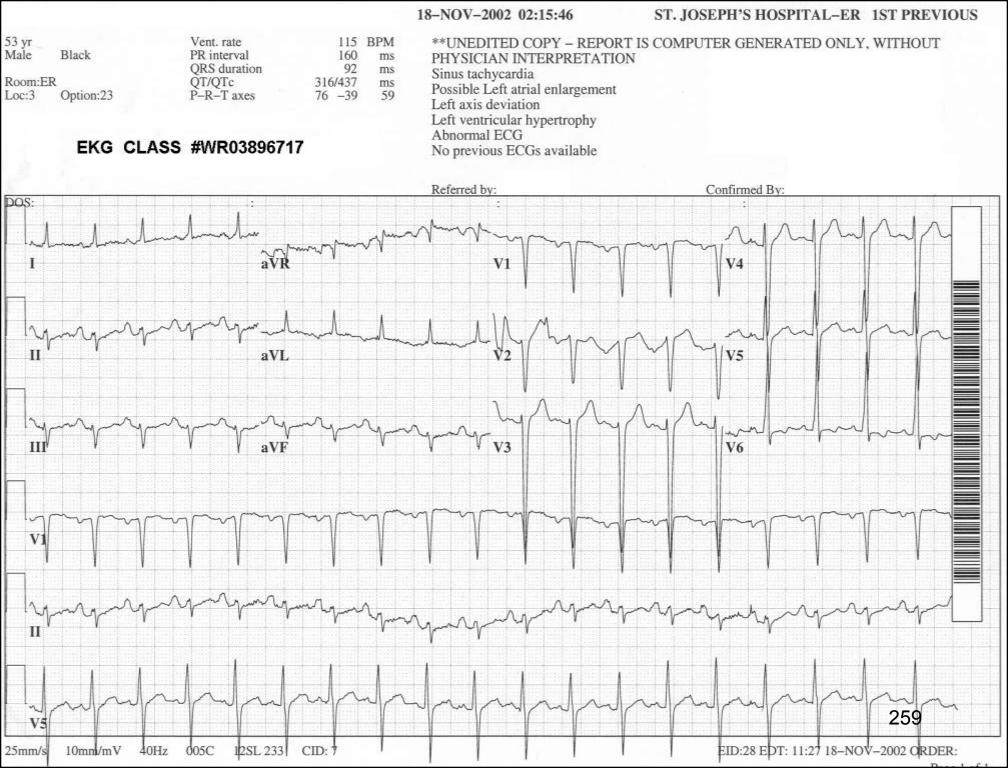
EKG CLASS #WR03028722

Right bundle branch block, plus right ventricular hypertrophy Left posterior fascicular block
\*\*\* Bifascicular block \*\*\* NONSPECIFIC ST CHANGES

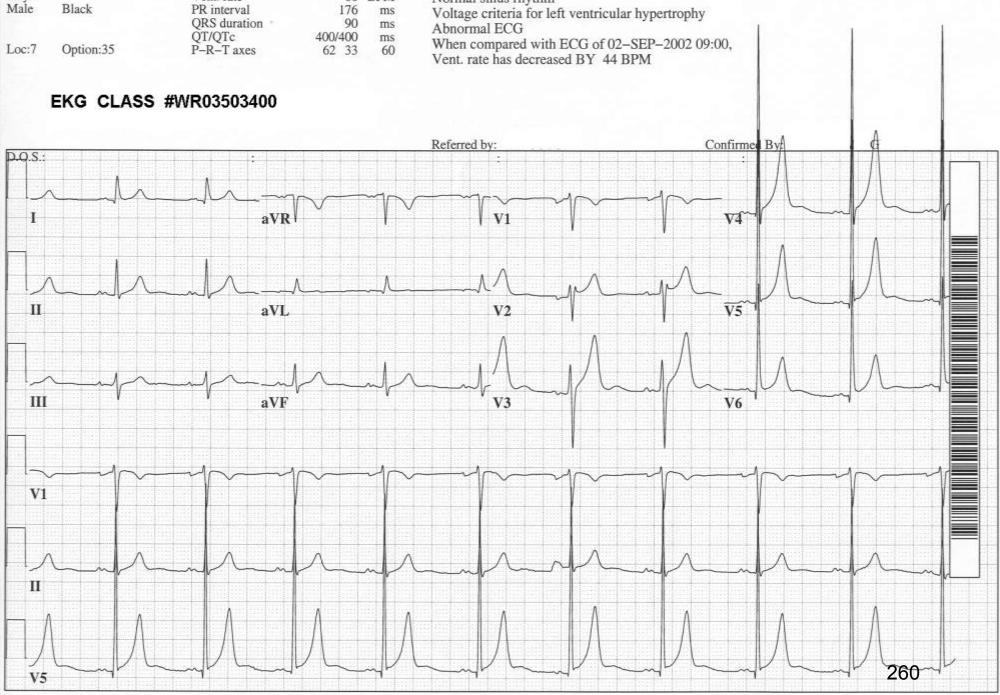
Abnormal ECG

When compared with ECG of 21-APR-1996 11:44,





#### 10-APR-2003 11:49:36 ST. JOSEPH'S HOSPITAL-CCR ROUTINE RETRIEVAL 60 BPM Normal sinus rhythm Voltage criteria for left ventricular hypertrophy ms ms Abnormal ECG ms When compared with ECG of 02-SEP-2002 09:00, 60 Vent. rate has decreased BY 44 BPM



Vent. rate

61 yr

#### **QRS AMPLITUDE**

#### CRITERIA FOR MINIMUM AMPLITUDE:

Abnormally LOW QRS VOLTAGE occurs when the OVERALL QRS is:

≤ 0.5 mV IN ANY LIMB LEAD

— and —

≤ 1.0 mV IN ANY PRECORDIAL LEAD

#### **OVERALL QRS AMPLITUDE TOO LOW:**

(VERTICAL QRS SIZE)

THINK (in absence of obvious OBESITY):



MYOCARDITIS /
CONSTRICTIVE PERICARDITIS



**EFFUSIONS / TAMPONADE** 



COPD c HYPERINFLATION



**AMYLOIDOSIS** 

(abnormal protein accumulation in organs)



SCLERODERMA

(abnormal hardening of skin)



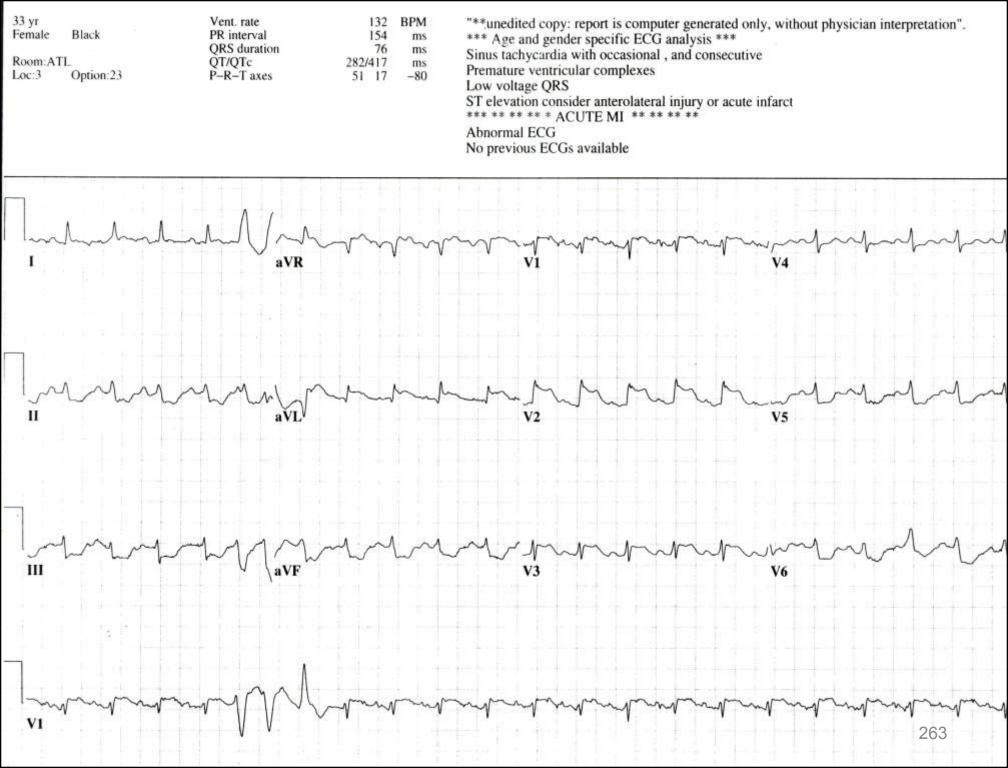
**HEMACHROMOTOSIS** 

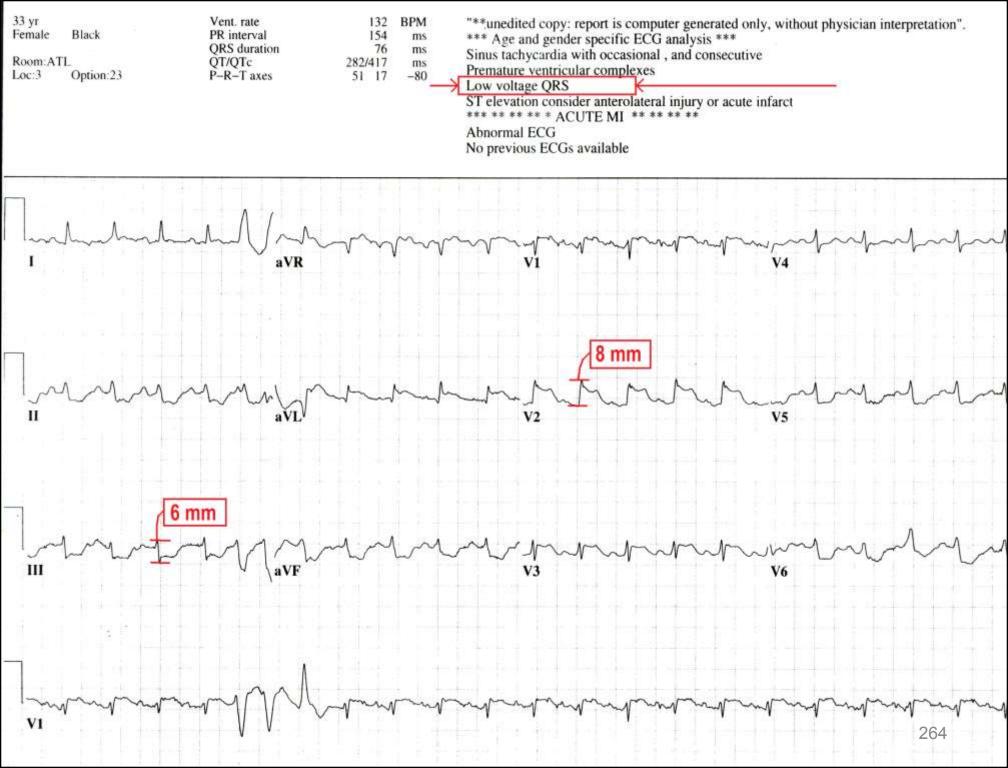
(excessive iron buildup in blood /organs)



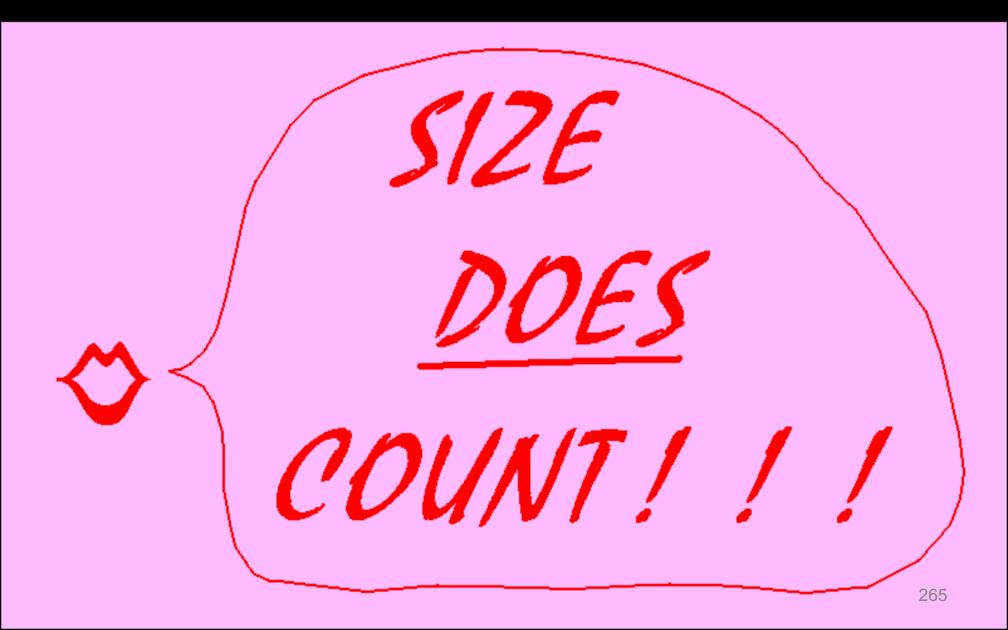
MYXEDEMA

(thyroid disorder)



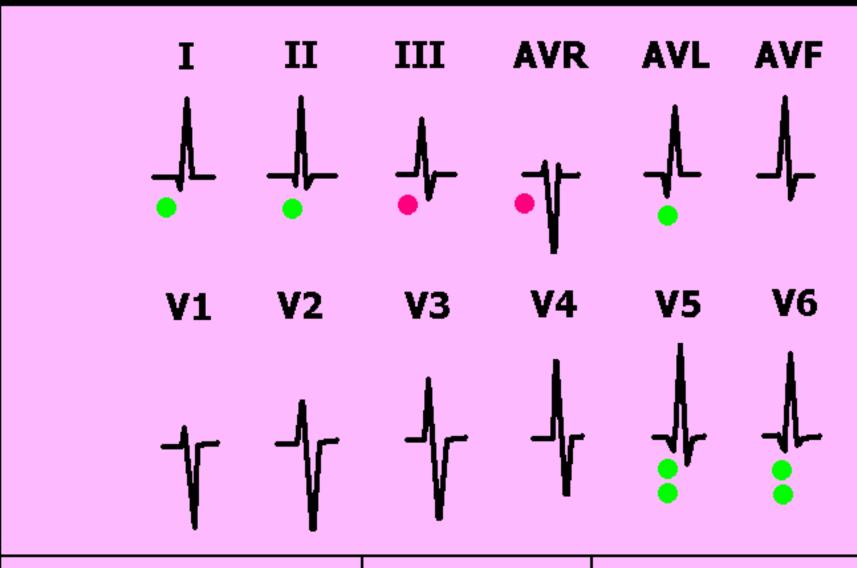


## Q WAVES •

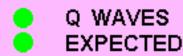


#### LEADS WHERE Q WAVES ARE NORMAL

- Normal Q WAVES caused by SEPTAL DEPOLARIZATION



 Q WAVES NORMAL AND FREQUENTLY SEEN



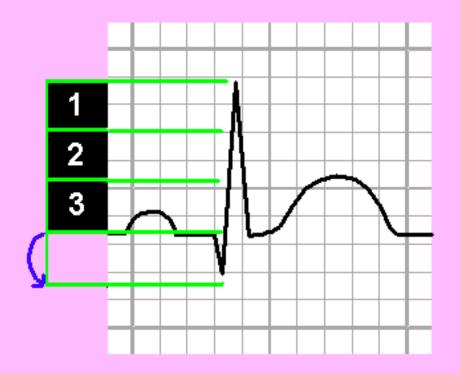
Q WAVES, IF PRESENT, CAN NORMALLY BE ANY SIZE<sup>66</sup>

# GENERAL RULES FOR NORMAL Q WAVES - WIDTH



LESS THAN .40 (1 mm) WIDE

#### GENERAL RULES FOR NORMAL Q WAVES - HEIGHT





LESS THAN 1/3 THE HEIGHT OF THE R WAVE

# NORMAL Q WAVES EXCEPTIONS TO THE RULES



LEAD AVR



LEAD III



THE Q WAVE CAN BE ANY SIZE

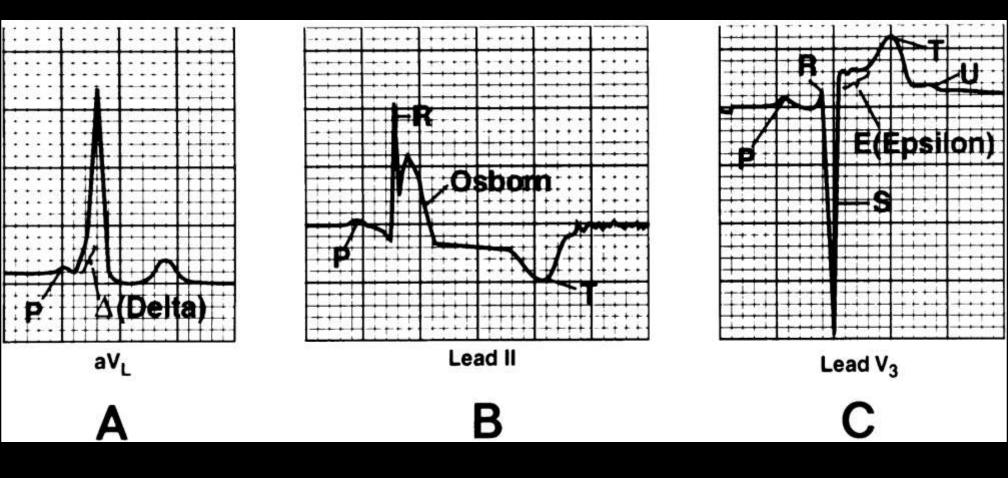
# NORMAL Q WAVES EXCEPTIONS TO THE RULES

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



#### **Q WAVE RULES - SUMMARY:**

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



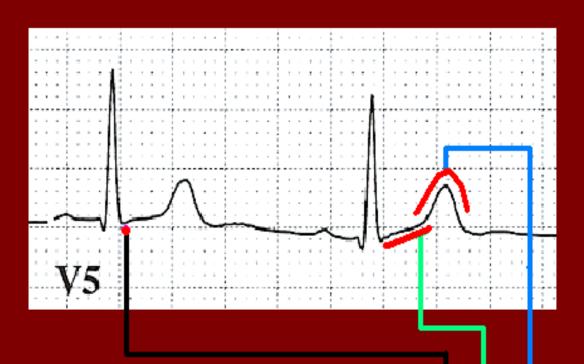
- A. Delta wave, seen in Wolff-Parkinson-White Syndrome
- B. Osborn's wave, seen in HYPOTHERMIA
- C. Epsilon's wave, seen in Right Ventricular Dysplasia





## NORMAL ST - T WAVES

- WHEN QRS WIDTH IS NORMAL (< 120 ms)



#### ASSESS:

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

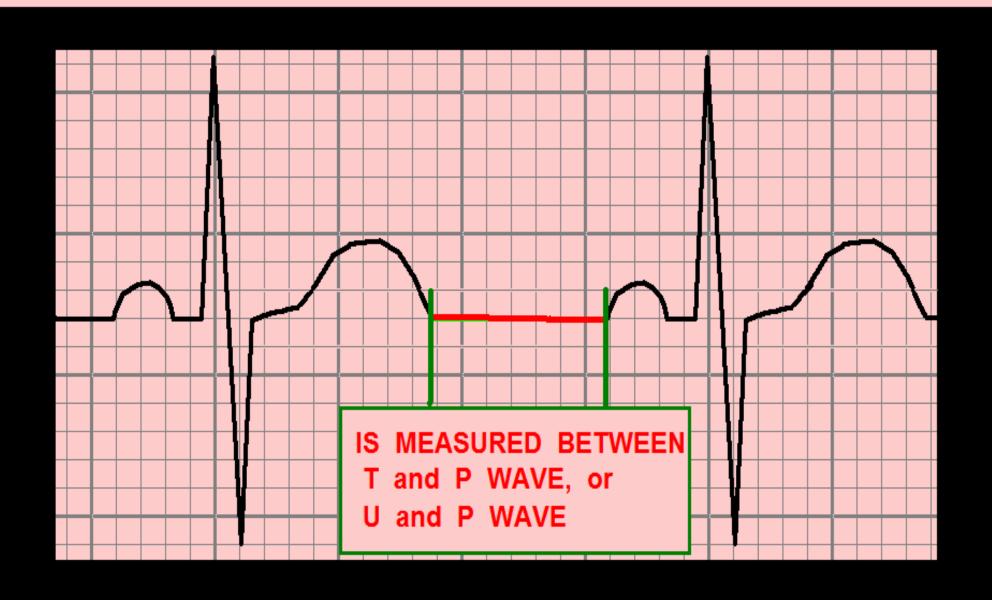
- ST SEG: SLIGHT, POSITIVE INCLINATION -

- T WAVE: UPRIGHT, POSITIVE -



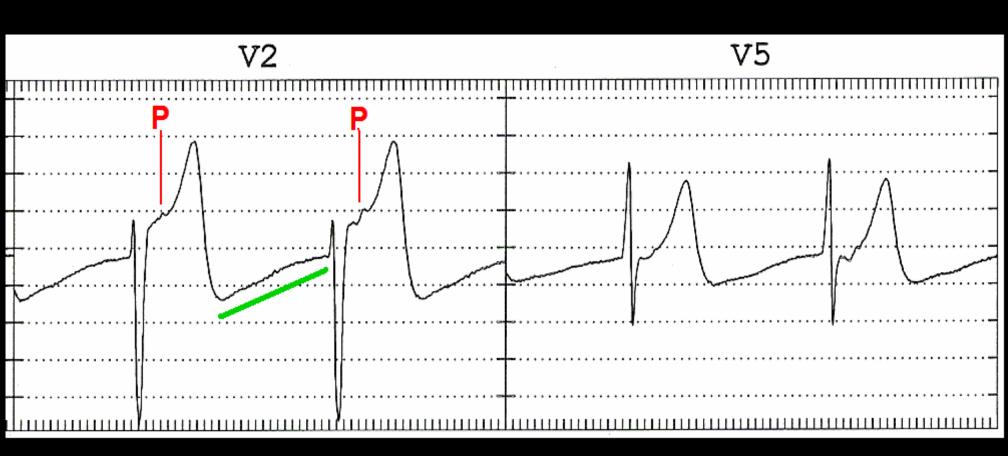
in EVERY LEAD EXCEPT aVR !!

### 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.



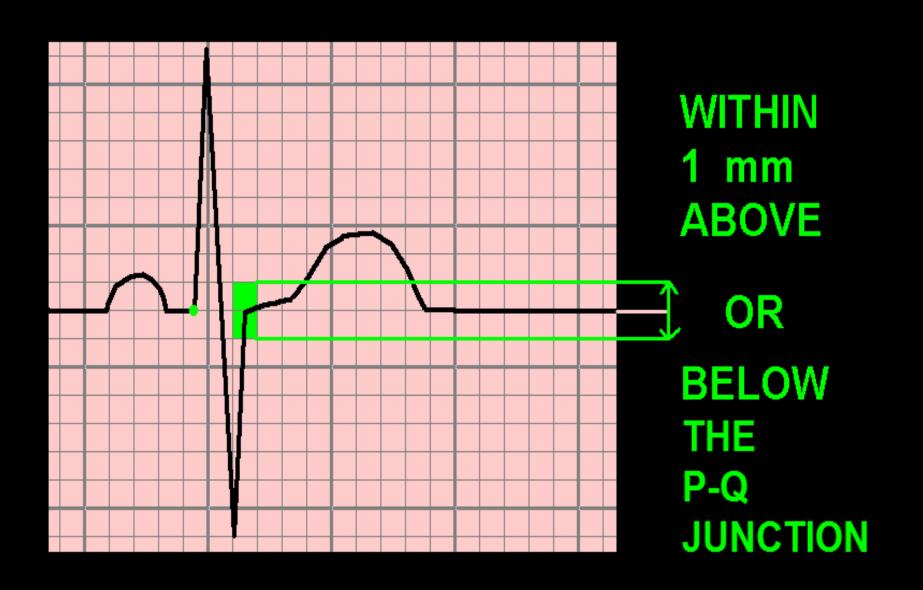
### THE P-Q JUNCTION

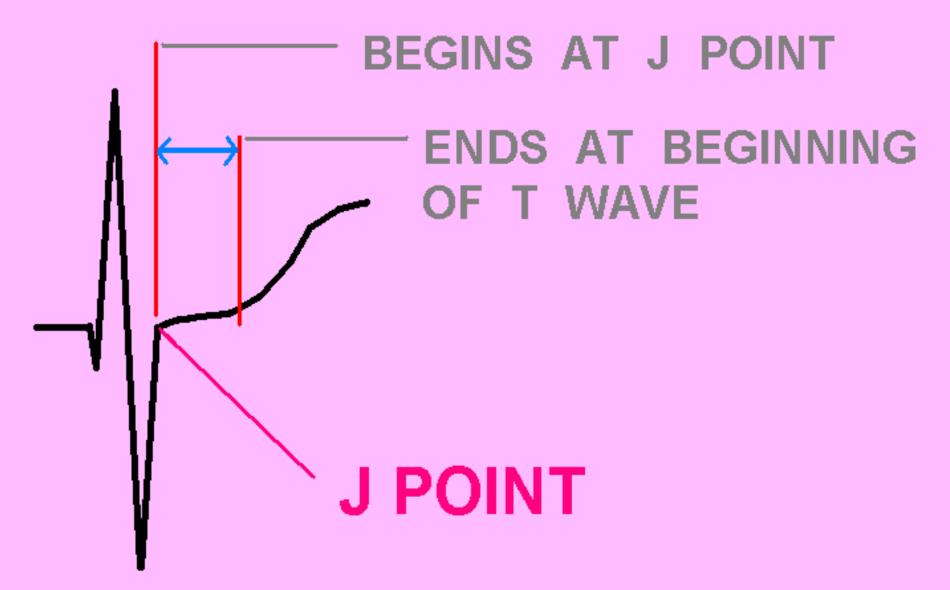
. . . is the POINT where the P-R SEGMENT ends and the QRS COMPLEX BEGINS. **Used for POINT** OF REFERENCE for measurement of the J-POINT and the S-T SEGMENT -

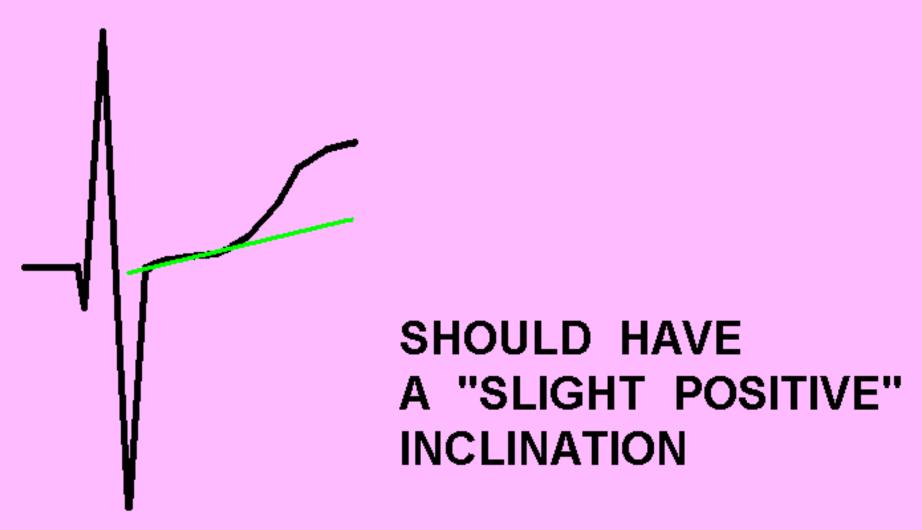


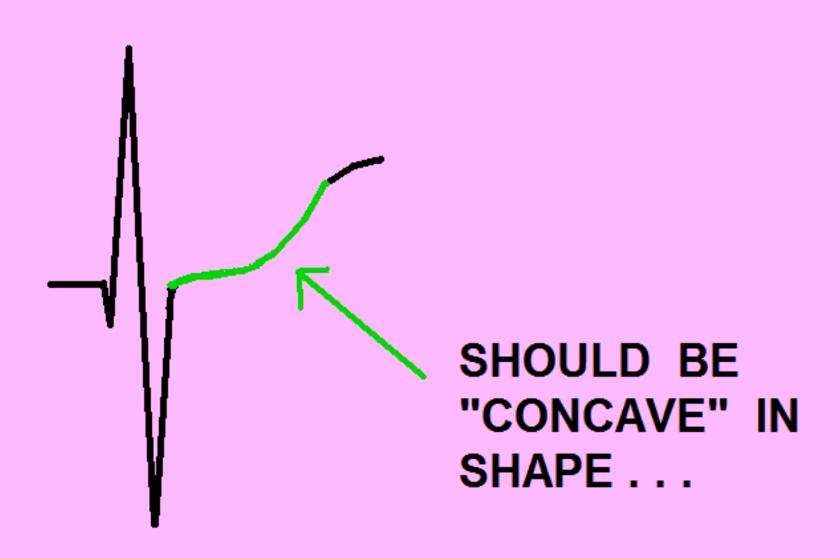
 as per the A.H.A., A.C.C., and WANG, ASINGER, and MARRIOTT, N.E.J.M. vol. 349:2128-2135 Nov. 27, 2003

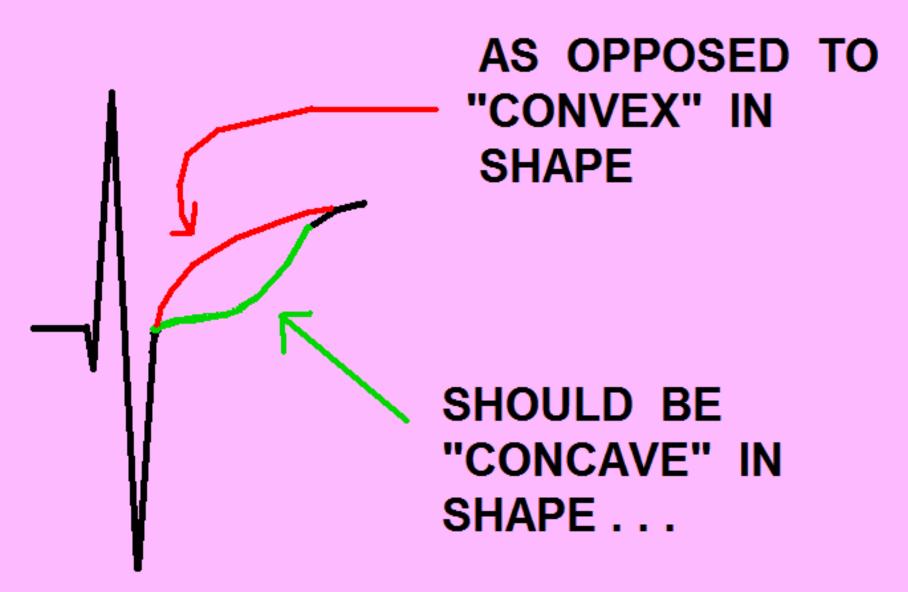
#### THE J POINT SHOULD BE ...

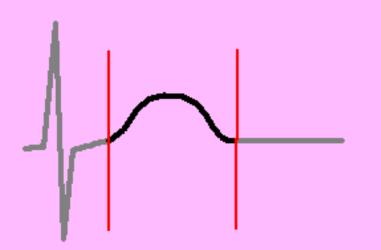






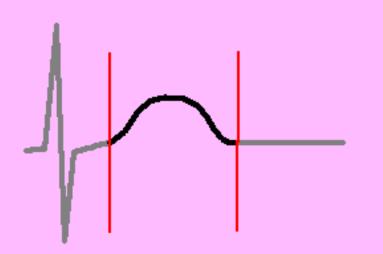






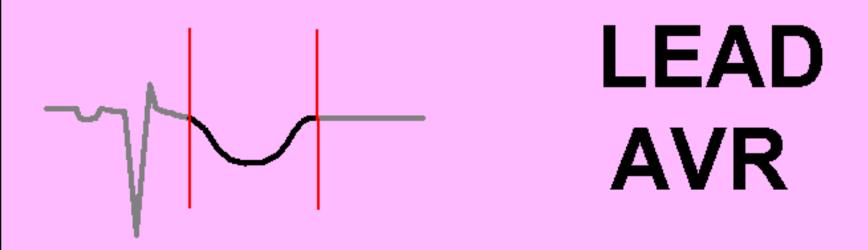
SHOULD BE
 A "NICE,"
 ROUNDED,
 CONVEX SHAPE

SHOULD BE SYMMETRICAL

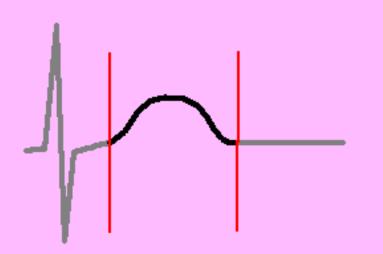


SHOULD BE
 A "NICE,"
 ROUNDED,
 CONVEX SHAPE

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



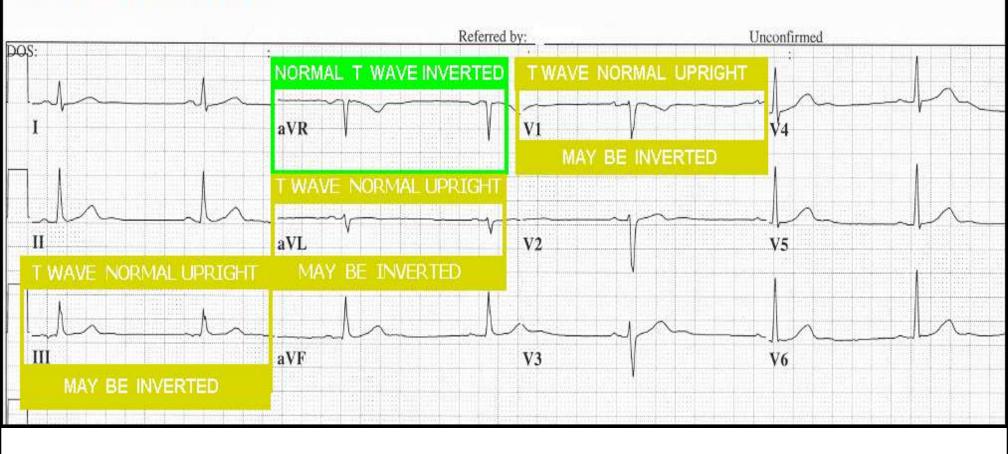
REMEMBER, IN LEAD AVR
 EVERYTHING
 IS
 "UPSIDE-DOWN"



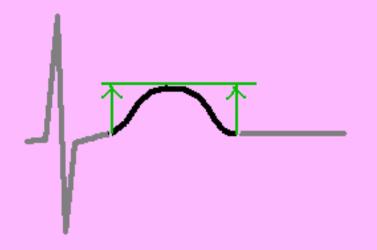
SHOULD BE
 A "NICE,"
 ROUNDED,
 CONVEX SHAPE

- SHOULD BE SYMMETRICAL
- SHOULD BE UPRIGHT IN ALL LEADS, EXCEPT AVR
- MAY BE INVERTED IN LEADS I, III, and V1

# Leads where the T WAVE may be INVERTED:



An inverted T wave in TWO OR MORE CONTIGUOUS LEADS = potential problem (ischemia)



# 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.



#### **MACOUTE T WAVES - COMMON ETIOLOGIES:**





CONDITION:

**\* HYPERKALEMIA** 

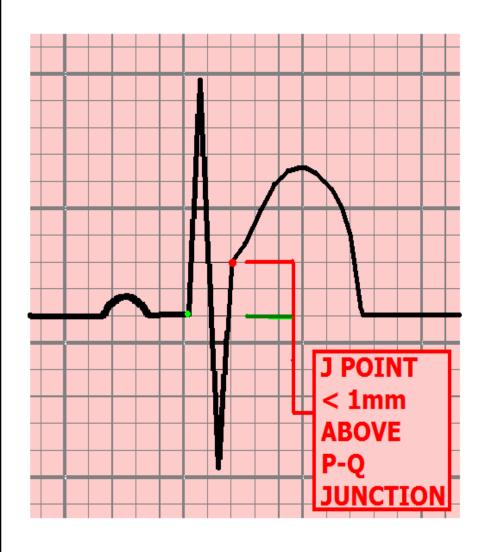
**ACUTE MI** 

**◆\* TRANSMURAL ISCHEMIA** 

**\* HYPERTROPHY** 

#### S-T SEGMENT ELEVATION

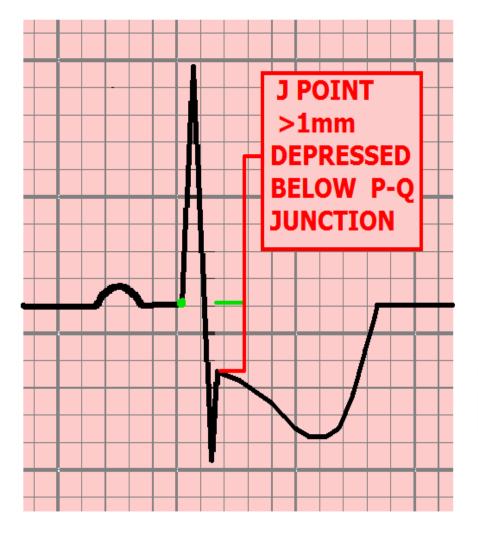
#### - COMMON ETIOLOGIES:



#### 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.

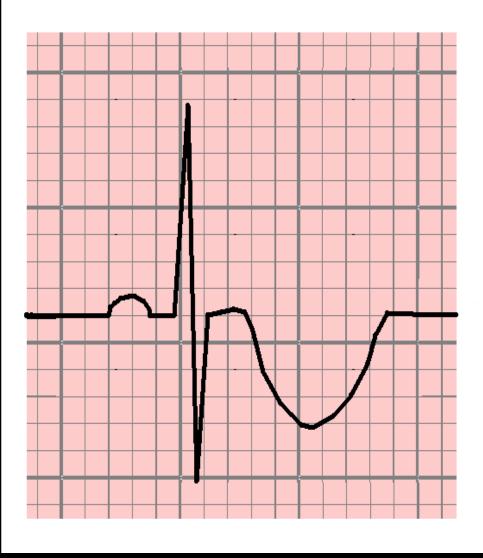
#### S-T SEGMENT DEPRESSION - COMMON ETIOLOGIES:



#### CONDITION:

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

#### T WAVE INVERSION - COMMON ETIOLOGIES:



#### CONDITION:

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

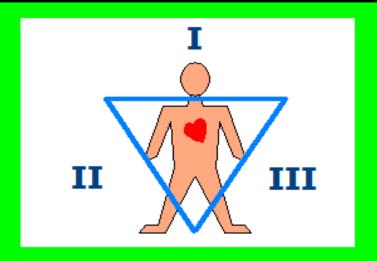
#### **OPTIONAL CURRICULUM:**



# EVALUATE THE AXIS IN BOTH PLANES

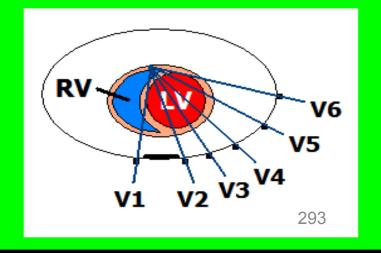
- VERTICAL

" AXIS DEVIATION "



- HORIZONTAL

" AXIS ROTATION "



#### **AXIS DEVIATION**

LEAD I

LEAD AVF

NORMAL

人

L

**LEFT** 

ℷ

γ

**RIGHT** 

 ${\sf V}$ 

人

**FAR RIGHT** 

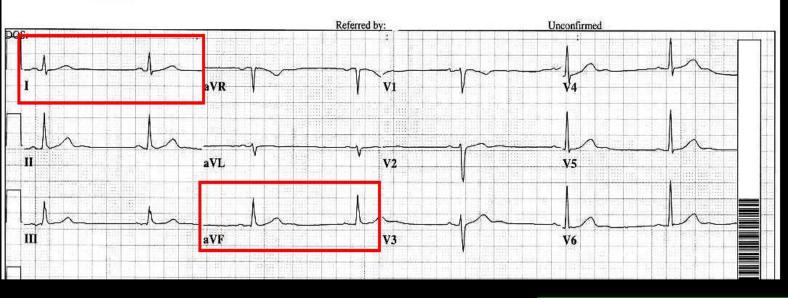
abla

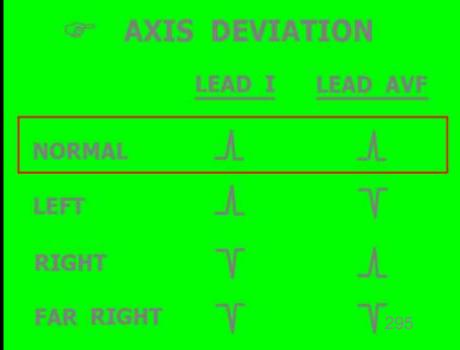
 ${\sf V}$ 

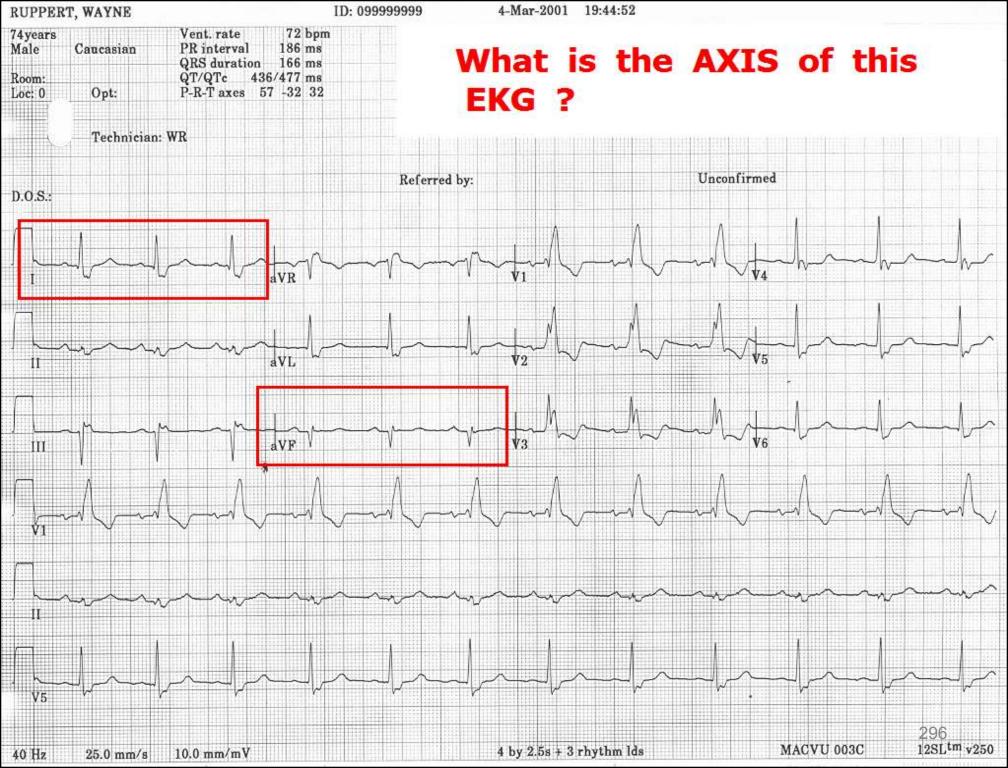
66 yr Male Vent. rate 41 BPM Caucasian PR interval 192 QRS duration QT/QTc P-R-T axes 94 ms 526/433 Room:401A ms 38 70 58 Loc:6 Option:16

#### **NORMAL AXIS**

Technician:









#### AXIS DEVIATION

#### COMMON CONDITIONS WHICH MAY CAUSE

## **LEFT AXIS DEVIATION:**

- LEFT BUNDLE BRANCH BLOCK
- **PACEMAKER**
- **◆** C.O.P.D.
- LEFT VENTRICULAR HYPERTROPHY
- **OLD INFERIOR WALL MI**
- HYPERKALEMIA
- LEFT ANTERIOR FASCICULAR BLOCK
- **₩OLFF-PARKINSON-WHITE (types A & B)**

81 yr Female Hispanic

Hispanic

Room:303A Loc:6 Option:11 
 Vent. rate
 82
 BPM

 PR interval
 128
 ms

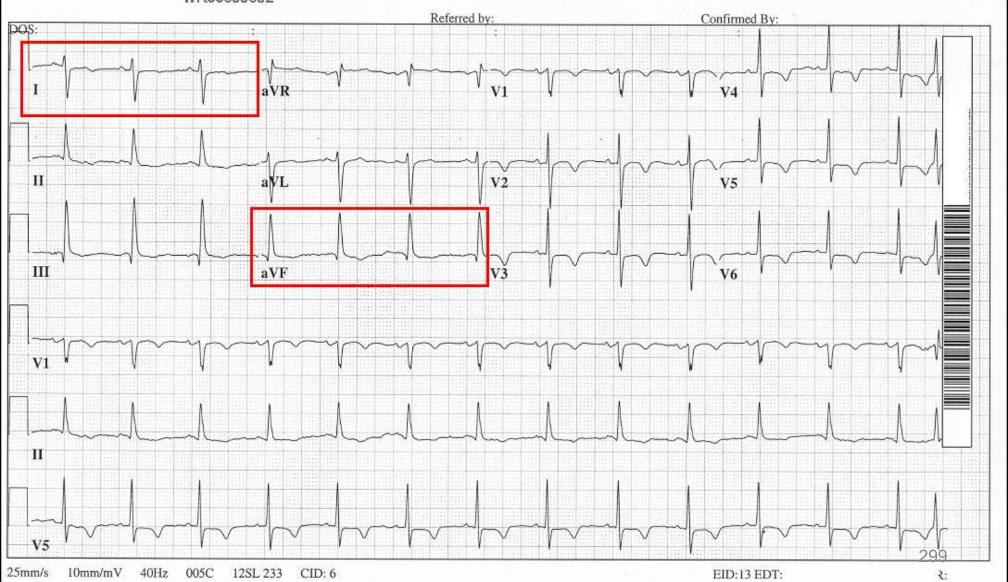
 QRS duration
 86
 ms

 QT/QTc
 392/457
 ms

 P-R-T axes
 38
 112
 -142

## What is the AXIS of this EKG?

Technician: EKG CLASS CODE WR03899892





#### AXIS DEVIATION

### COMMON CONDITIONS WHICH MAY CAUSE RIGHT AXIS DEVIATION:

- NORMAL FOR PEDS & TALL, THIN ADULTS
- RIGHT VENTRICULAR HYPERTROPHY
- OLD LATERAL WALL MI
- LEFT POSTERIOR FASICULAR BLOCK
- PULMONARY EMBOLUS
- DEXTROCARDIA
- **◆** C.O.P.D.
- ATRIAL / VENTRICULAR SEPTAL DEFECTS

Caucasian Male

Room:5 Loc:1

92 BPM \*

ACCELERATED IDIOVENTRICULAR RHYTHM

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

172

ms

ms

61

EKG CLASS CODE #WR03611255



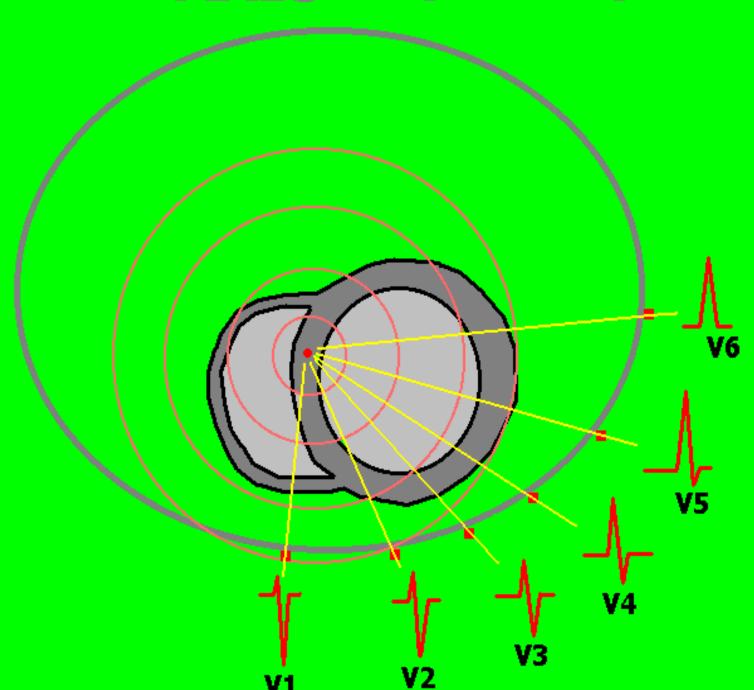


#### AXIS DEVIATION

#### COMMON CONDITIONS WHICH MAY CAUSE

## (NO-MAN'S LAND AXIS) FAR RIGHT AXIS DEVIATION:

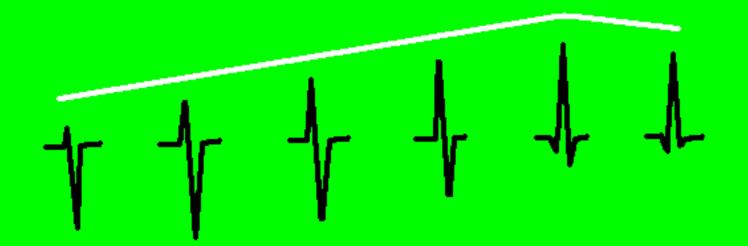
- LEAD TRANSPOSITION
- PACEMAKER RHYTHMS
- **VENTRICULAR RHYTHMS**
- **◆** C.O.P.D.
- HYPERKALEMIA



#### ASSESSING AXIS ROTATION:

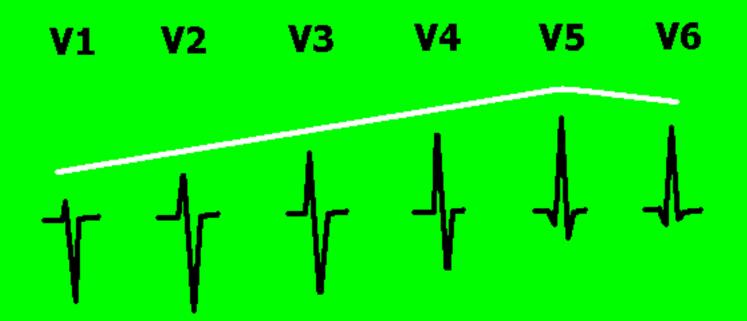
V1 V2 V3 V4 V5 V6

#### R - WAVE PROGRESSION



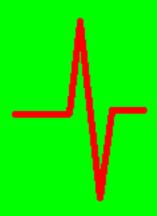
### 2. IDENTIFICATION OF TRANSITION

#### ASSESSING AXIS ROTATION:

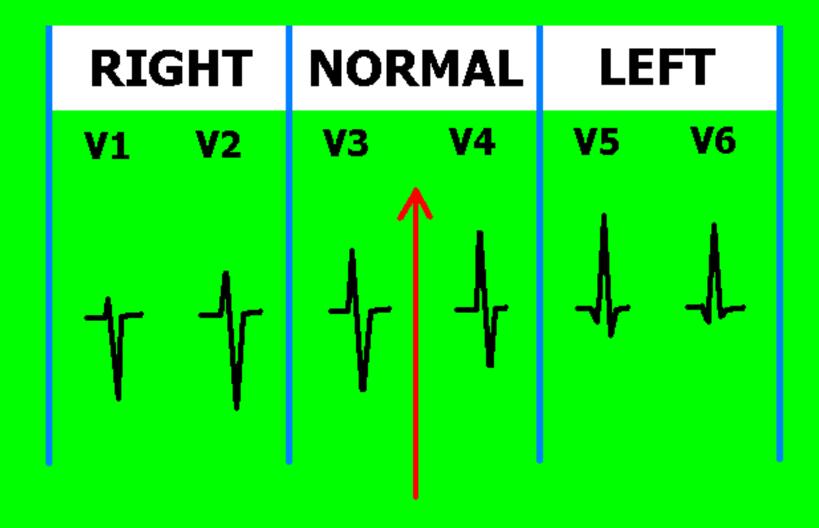


3. RECALL COMMON PATTERNS of ABNORMAL R-WAVE PROGRESSION to help you build your list of POSSIBLE DIAGNOSES.

### AXIS ROTATION TRANSITION



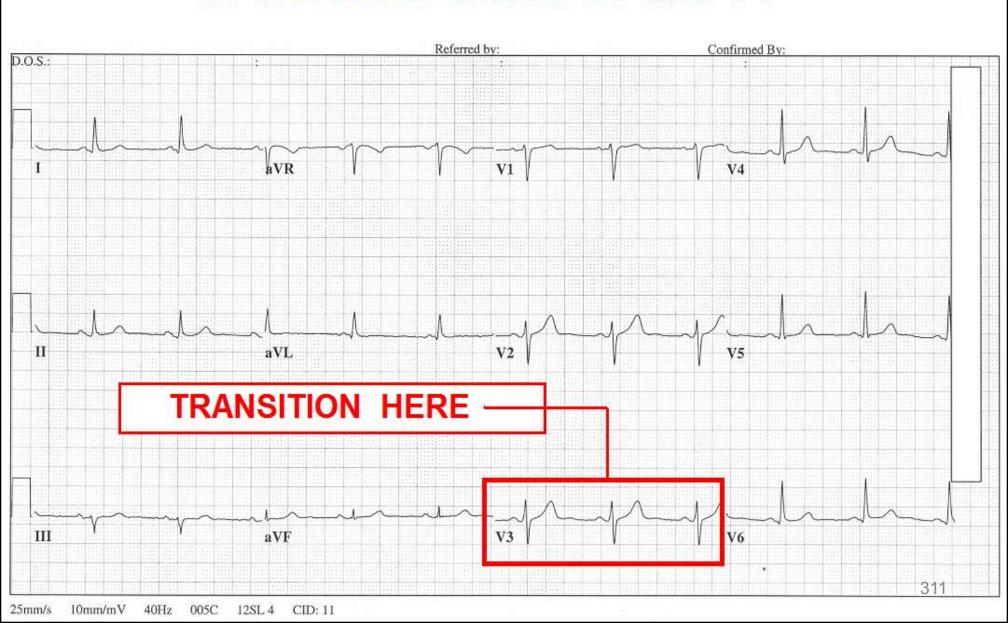
# OCCURS IN THE LEAD WHERE THE QRS IS THE MOST BIPHASIC

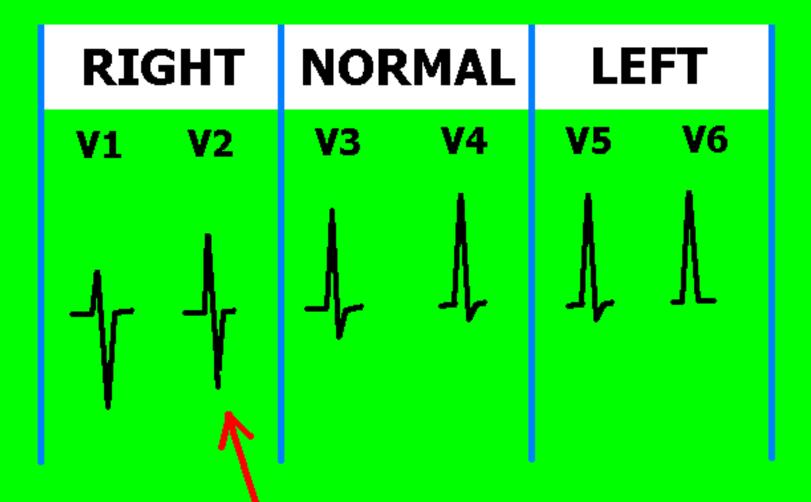


TRANSITION SHOULD OCCUR IN LEADS V3 or V4

#### NORMAL TRANSITION

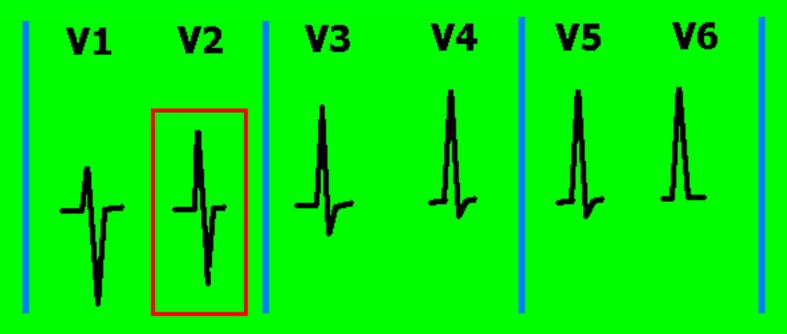
#### IS BETWEEN LEADS V3 and V4





\ "EARLY TRANSITION"
"SHIFTED TO THE RIGHT"

#### \*COMMON CAUSES of EARLY TRANSITION



- 1. Right Bundle Branch Block
- 2. Right Ventricular Hypertrophy
- 3. Old Posterior Wall MI
- 4. Wolff-Parkinson-White (type A)

LEFT - SIDED PATHWAY - FROM MARRIOTT'S "Practical Electrocardiography - 10th Edition," 2000

### COMMON CAUSES OF EARLY TRANSITION ....SOME HELPFUL CLUES:

#### 1. Right Bundle Branch Block (RBBB)

- QRS wider than 120ms
- Supraventricular rhythm (normal P : QRS relationship)
- RSR' or RR' ("notching") in V1, V2, and/or V3

#### 2. Right Ventricular Hypertrophy (RVH)

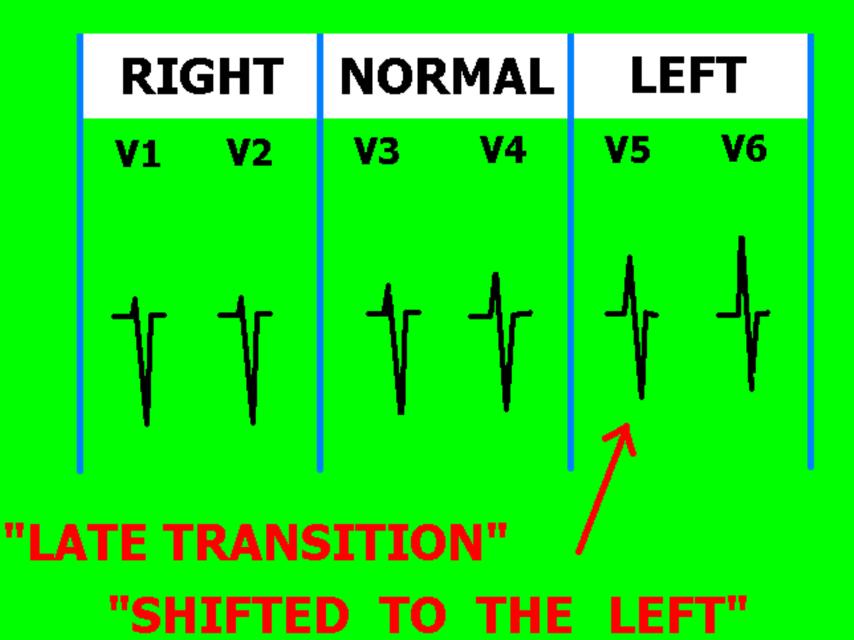
- Corresponding Right Atrial Hypertrophy (RAH)
- Right Axis Deviation (RAD)
- QRS in LEAD I more NEGATIVE than POSITIVE ( R<S )

#### 3. Old Posterior Wall MI

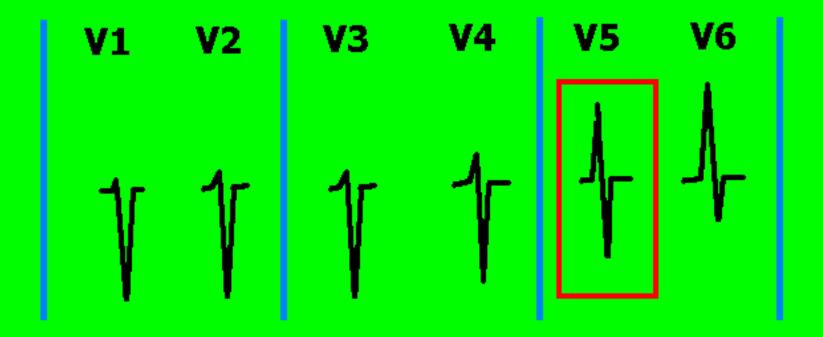
- Usually accompanied by OLD INFERIOR WALL MI
- Does NOT abnormally widen the QRS complex

#### 4. Wolff-Parkinson-White (WPW) type A

- Short P-R Interval
- Presence of Delta Waves
- Wide QRS complexes



#### **COMMON CAUSES of LATE TRANSITION**



- 1. Old Anterior Wall M.I.
- 2. Left Bundle Branch Block
- 3. Left Ventricular Hypertrophy
- 4. Wolff-Parkinson-White (type B)

RIGHT-SIDED PATHWAY - FROM MARRIOTT'S "Practical Electrocardiography - 10th Edition," 2000

#### COMMON CAUSES OF LATE TRANSITION

.... WITH SOME COMMON HELPFUL CLUES:

#### 1. Old Anterior MI

- Q Waves in V1, V2, and /or V3
- Other causes of LATE TRANSITION ruled out
- 2. Left Bundle Branch Bock (LBBB)
  - Supraventricular Rhythm
  - QRS wider than 120 ms ( .12 sec )
  - RsR' or RR' ("notching") in V5 and/or V6
- 3. Left Ventricular Hypertrophy (LVH)
  - Corresponding Left Atrial Hypertrophy (LAH)
  - T wave Strain Pattern V5 / V6
  - Intrinsicoid Deflection in V5 / V6 > 45 ms
  - V1 S wave + V5 or V6 R wave > 35 mm
  - R or S wave in any LIMB LEAD > 2.0 mV (20 mm)
- 4. Wolff-Parkinson-White (Type B)
  - Presence of DELTA waves
  - Short P-R Interval (< 120 ms)</li>
  - Wide QRS ( > 120 ms )

