



Bayfront Health Hospitals

Bayfront Health Seven Rivers, Crystal River, FL



Bayfront Health Brooksville, Brooksville, FL



Seven Rivers Freestanding ED, Citrus Hills, FL



Bayfront Health Spring Hill, Spring Hill, FL



The Lifesaving 12 Lead ECG: Part 1

Wayne W Ruppert, CVT, CCCC, NREMT-P
Regional Cardiovascular Coordinator
Chest Pain Center, Heart Failure and
Therapeutic Hypothermia Programs



Welcome !



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

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

Source of Curriculum:

- Case Studies from Cardiac Catheterization and Electrophysiology Labs, 1996 – Present

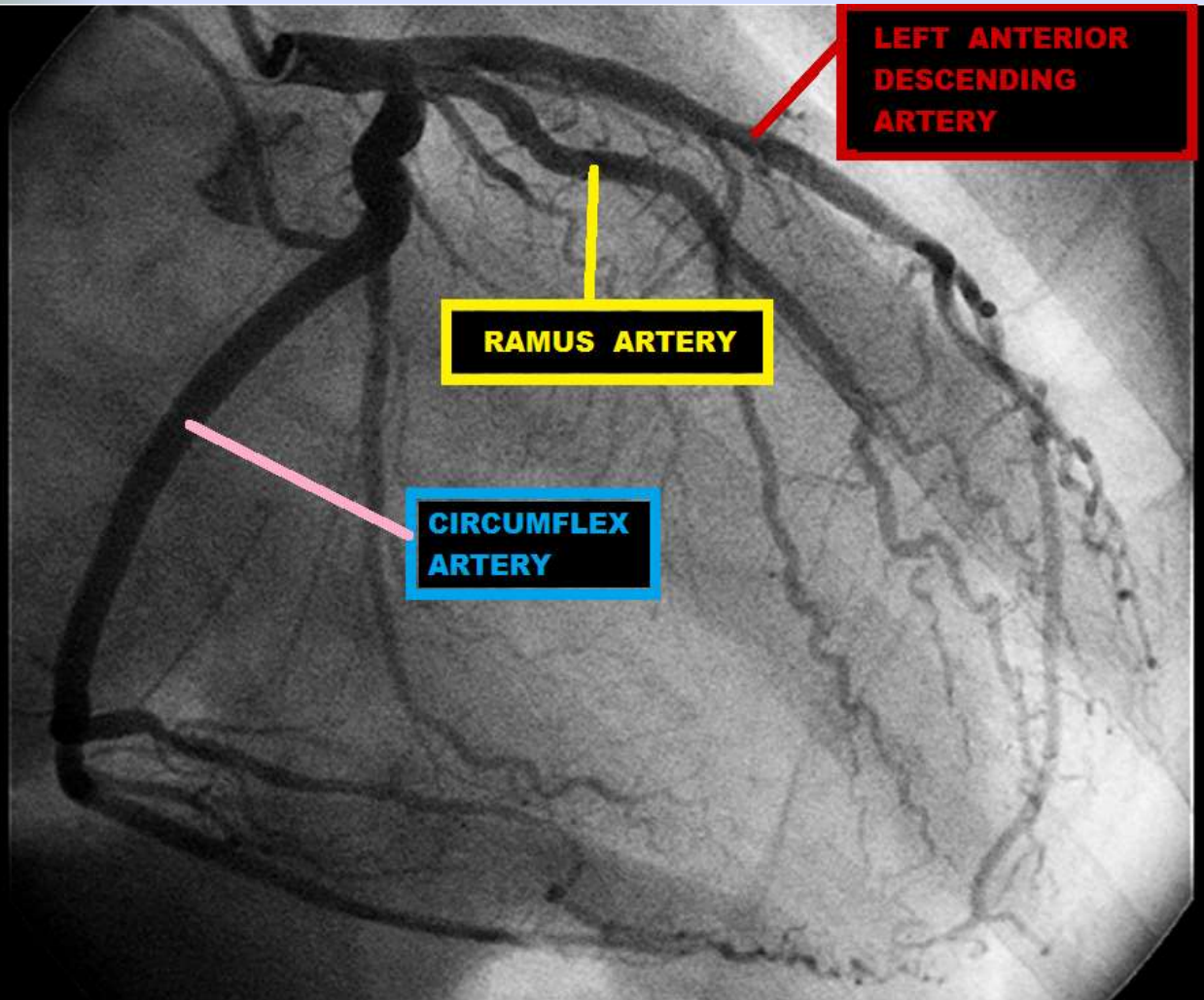
13,000 – 15,000 EP and Cath Lab cases between 1996 - Today



Wayne Ruppert and Dr. James Irwin, St Joseph's Hospital, Tampa, 7/29/2004

7 . 29 06 : 55

Cardiac Cath Lab Advantage:



Correlation
of ECG leads
with
SPECIFIC
cardiac
anatomic
structures.

Electrophysiology Lab Case Studies



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



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

EP Lab Advantage:



Correlation
of ECG
derived
diagnosis
with true
intra-cardiac
electrogram
acquired
diagnosis.

Source of Curriculum:

- Case Studies from Cardiac Catheterization and Electrophysiology Labs, 1996 – Present
- Current Evidence-based Research
 - Journal of the American College of Cardiology (JACC)
 - American Heart Association (AHA) Circulation
 - ACC/AHA Guidelines
 - New England Journal of Medicine

Source of Curriculum:

- Case Studies from Cardiac Catheterization and Electrophysiology Labs, 1996 – Present
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 - Journal of the American College of Cardiology (JACC)
 - American Heart Association (AHA) Circulation
 - ACC/AHA Guidelines
 - New England Journal of Medicine
- Two peer reviewed, published textbooks

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' hearts.* Seeing ECGs from this perspective adds a new dimension to understanding the complex pathophysiologies of cardiovascular 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 diagnostic 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 CARDIAC 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 reads their 12 lead ECGs:

- Fellows in Emergency, Cardiology, and Family Medicine
- Medical Residents
- Veteran Physicians wanting a good review in ACS patient evaluation
- Physician Assistants and Nurse Practitioners
- Emergency Department Nurses
- Coronary Care Unit and Cardiac Telemetry Nurses
- Walk-in Clinic Physicians and Nurses
- Paramedics

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

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

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

"This book integrates academic ECG principles with 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



12 LEAD ECG INTERPRETATION IN ACUTE CORONARY SYNDROME with CASE STUDIES from the CATH LAB -- WAYNE RUPPERT

THE CATH LAB SERIES presents,

12 LEAD ECG INTERPRETATION IN

ACUTE CORONARY SYNDROME

with CASE STUDIES from the

CARDIAC CATHETERIZATION LAB

WAYNE W RUPPERT

www.TriGenPress.com
www.ECGtraining.org

BarnesandNoble.com
Amazon.com

TEXTBOOK REVIEWED BY:

Joseph P. Ornato, MD, FACP, FACEP, FACC, Professor and Chairman, Department of Emergency Medicine, Medical College of Virginia-Virginia Commonwealth University

Humberto Coto, MD, FACP, FACC, Chief of Cardiology, St. Joseph's Hospital

Matthew Glover, MD, FACP, FACC, Interventional Cardiologist, St. Joseph's Hospital

Xavier Prida, MD, FACP, FACC, Interventional Cardiologist, St. Joseph's Hospital

Charles Sand, MD, FACP, FACEP, Emergency Department Physician, St. Joseph's Hospital

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

STEMI Assistant

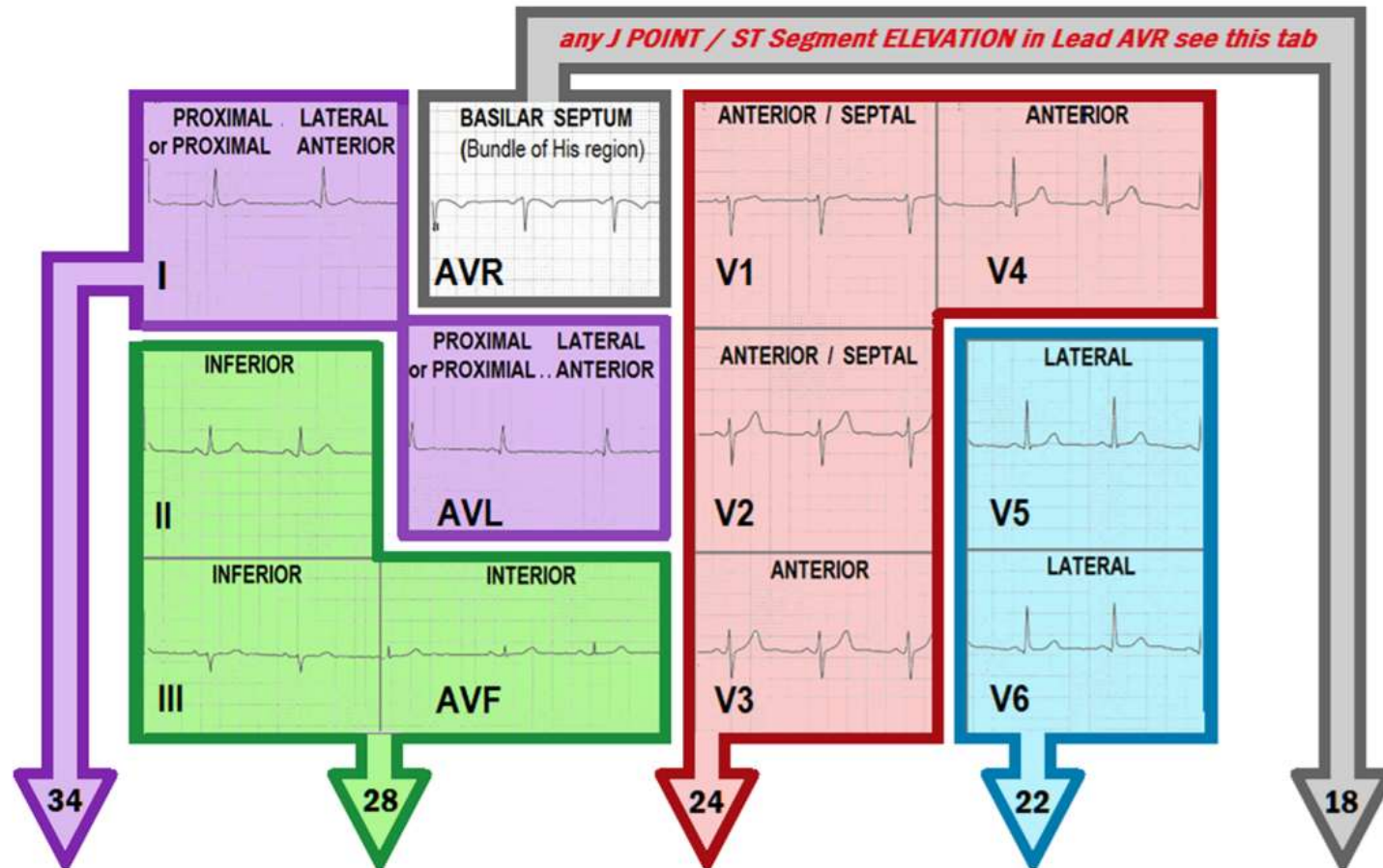
by Wayne Ruppert

UNIVERSAL ACS PATIENT MANAGEMENT ALGORITHM

--- See PAGE ONE ---

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

CRASH CART EMERGENCY REFERENCE




Bayfront Health
Dade City

TEXTBOOK REVIEWED BY:

Barbra Backus, MD, PhD Inventor of “The HEART Score,” University Medical Center, Utrecht, Netherlands

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

Anna Ek, AACC, BSN, RN Accreditation Review Specialist, The American College of Cardiology

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

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

STEMI Assistant

[Tutorial Video](#)

[Free download – electronic copy \(PDF file\)](#)

Copyright 2010, 2015, 2018

All cardiovascular subject-related images, graphics and diagrams in this PowerPoint 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](#),” which are Copyright protected. No content may be removed from this PowerPoint presentation, nor may this presentation or any component thereof be used without written consent from the author.

Wayne.ruppert@bayfronthealth.com

COURSE PRE-REQUISITE:

You should already have Basic Single-Lead ECG Rhythm Strip Interpretation Skills.

THIS COURSE IS NOT A BASIC ECG RHYTHMS COURSE. If you are not already reasonably comfortable with interpreting and understanding basic ECG dysrhythmias (i.e.: heart blocks, A-Fib, V-Tach, etc.) we DO NOT recommend that you attend this workshop; instead we recommend our “Basic ECG Rhythms Workshop.”

The Lifesaving 12 Lead ECG Course:

Is a condensed curriculum focused on acute conditions which are associated with a high degree of morbidity and mortality:

The Lifesaving 12 Lead ECG Course:

Is a condensed curriculum focused on acute conditions which are associated with a high degree of morbidity and mortality:

1. Acute Coronary Syndromes

- STEMI (pre-infarction, acute & evolving / old MI)
- NSTEMI
- Unstable Angina
- Low Risk Chest Pain

The Lifesaving 12 Lead ECG Course:

Is a condensed curriculum focused on acute conditions which are associated with a high degree of morbidity and mortality:

2. Sudden Cardiac Death Syndromes

- Long QT Syndrome (Congenital & Drug Induced)
- Brugada Syndrome
- Cardiomyopathy (Hypertrophic and other)
- Arrhythmogenic Right Ventricular Dysplasia
- Wolff-Parkinson-White Syndrome
- Catecholnergic Polymorphic Ventricular Tachy.

SUGGESTION *for optimal learning.....*

To get the most from this class:

- Do not try to write down or memorize every point.

SUGGESTION for optimal learning.....

To get the most from this class:

- Do not try to write down or memorize every point.
- **DOWNLOAD** this PowerPoint in its entirety – review and study it at you own pace.

SUGGESTION *for optimal learning.....*

To get the most from this class:

- Do not try to write down or memorize every point.
- DOWNLOAD this PowerPoint in its entirety – review and study it at your own pace.
- For now Simply LISTEN to everything that is said. If it “makes sense,” then you’re learning.

SUGGESTION *for optimal learning.....*

To get the most from this class:

- Do not try to write down or memorize every point.
- DOWNLOAD this PowerPoint in its entirety – review and study it at your own pace.
- For now Simply LISTEN to everything that is said. If it “makes sense,” then you’re learning.
- In other words, *“just go along for the ride.”*



The Lifesaving 12 Lead ECG Course:

Session 1 (morning session) Contents:

- **Introduction and The ECG in Perspective**
- **Risk Stratification: The HEART Score**
- **Essential Cardiac A & P**
 - Cellular (depolarization / repolarization)
 - Structural
- **Heart Sounds and Valvular Function**

The Lifesaving 12 Lead ECG Course:

Session 1 Contents, continued:

- **Bypass Tract Pathophysiology**
- **ECG Principles**
- **Coronary Artery Anatomy and Correlation with the 12 Lead ECG**
- **Waveforms and Intervals**
- **Bundle Branch Blocks**
- **Axis Deviation and Rotation**

The Lifesaving 12 Lead ECG Course:

Session 2 (afternoon session) Contents:

- **Sudden Cardiac Death Syndromes**
 - Long QT
 - Hypertrophic Cardiomyopathy
 - Arrhythmogenic Right Ventricular Cardiomyopathy
 - Brugada Syndrome
- **Application of The HEART Score**
- **Acute Coronary Syndromes**
With Cath Lab Case Studies

Helpful Web Resources:

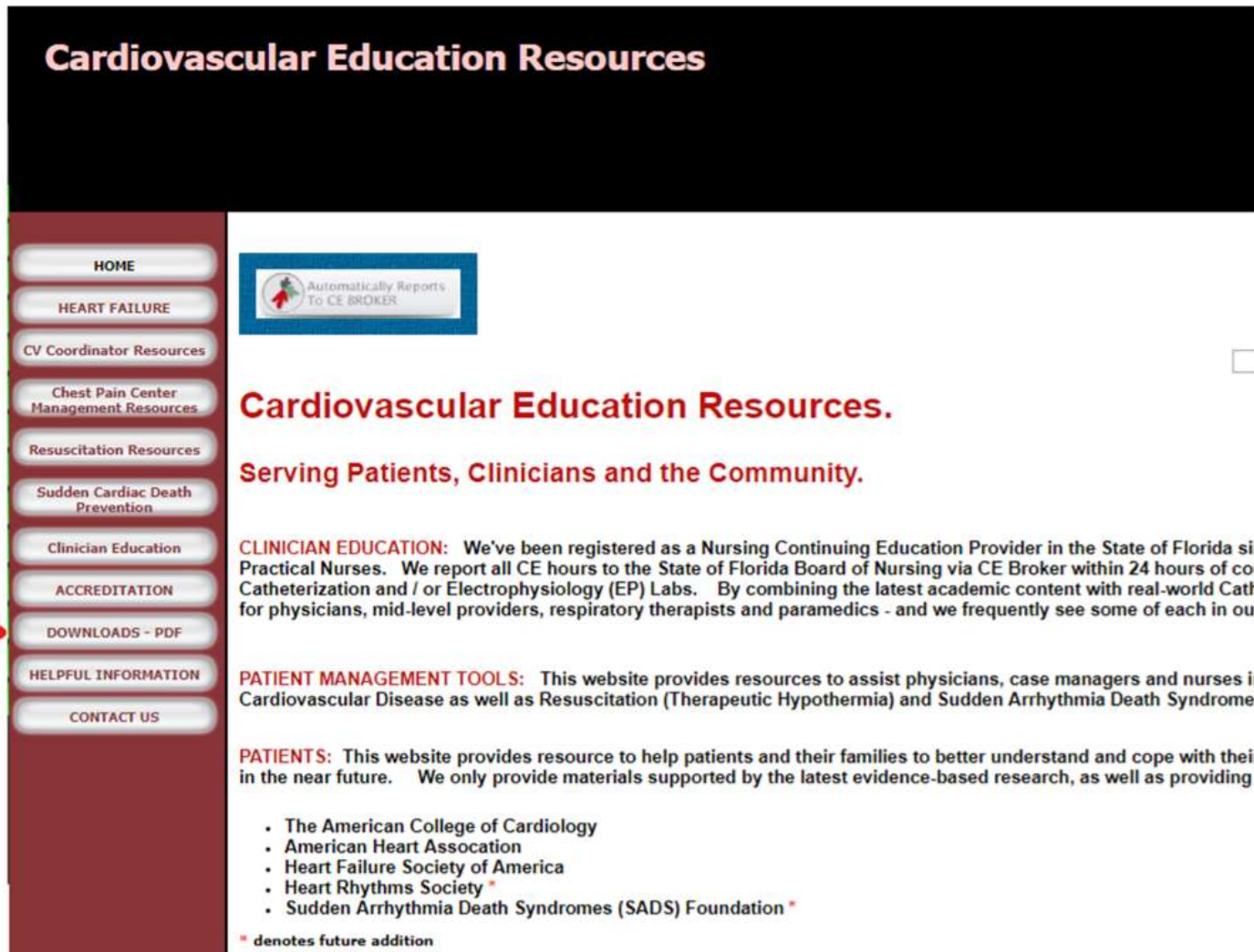
www.practicalclinicalskills.com

www.skillstat.com/tools/ecg-simulator

www.ECGtraining.org

1. Go to: www.ECGtraining.org

2. Select "Downloads PDF" from menu bar



Cardiovascular Education Resources

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Automatically Reports To CE BROKER

Cardiovascular Education Resources.

Serving Patients, Clinicians and the Community.

CLINICIAN EDUCATION: We've been registered as a Nursing Continuing Education Provider in the State of Florida for Practical Nurses. We report all CE hours to the State of Florida Board of Nursing via CE Broker within 24 hours of completion. Catheterization and / or Electrophysiology (EP) Labs. By combining the latest academic content with real-world Cath for physicians, mid-level providers, respiratory therapists and paramedics - and we frequently see some of each in our

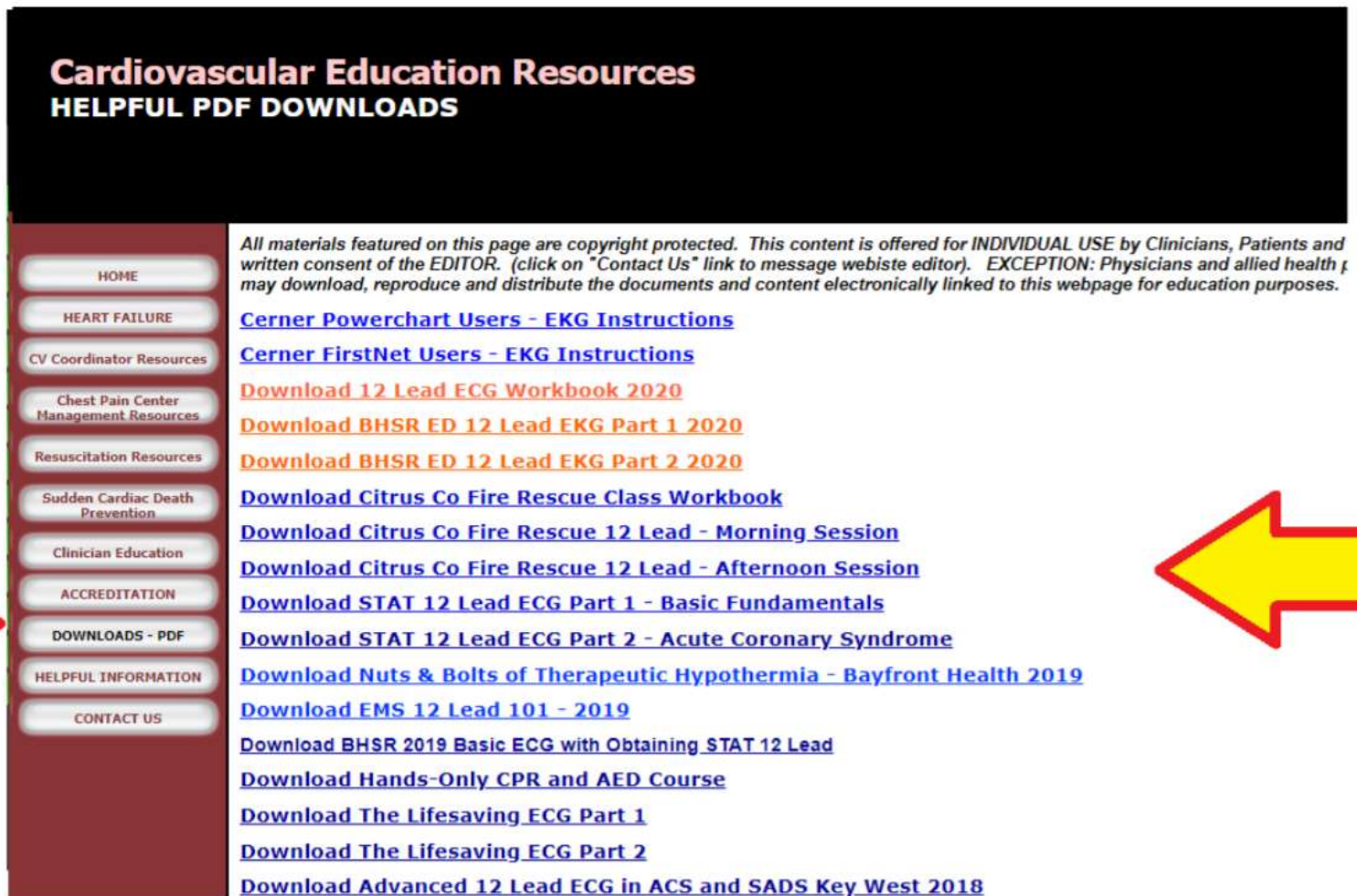
PATIENT MANAGEMENT TOOLS: This website provides resources to assist physicians, case managers and nurses in Cardiovascular Disease as well as Resuscitation (Therapeutic Hypothermia) and Sudden Arrhythmia Death Syndromes

PATIENTS: This website provides resource to help patients and their families to better understand and cope with their in the near future. We only provide materials supported by the latest evidence-based research, as well as providing I

- The American College of Cardiology
- American Heart Association
- Heart Failure Society of America
- Heart Rhythms Society *
- Sudden Arrhythmia Death Syndromes (SADS) Foundation *

* denotes future addition

1. Go to: www.ECGtraining.org
2. Select "Downloads PDF" from menu bar
3. Select your courses



Cardiovascular Education Resources
HELPFUL PDF DOWNLOADS

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[Download Hands-Only CPR and AED Course](#)
[Download The Lifesaving ECG Part 1](#)
[Download The Lifesaving ECG Part 2](#)
[Download Advanced 12 Lead ECG in ACS and SADS Key West 2018](#)

Then Select:

[The Lifesaving 12 Lead ECG - Part 1](#)

[The Lifesaving 12 Lead ECG - Part 2](#)

The EKG in PERSPECTIVE

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

AND . . .

***Sometimes,
ECGs
LIE to us !***

***ECGs and USED CAR SALESMEN
often have MUCH in common !***



The EKG in PERSPECTIVE

PROBLEMS WITH EKGs . . .

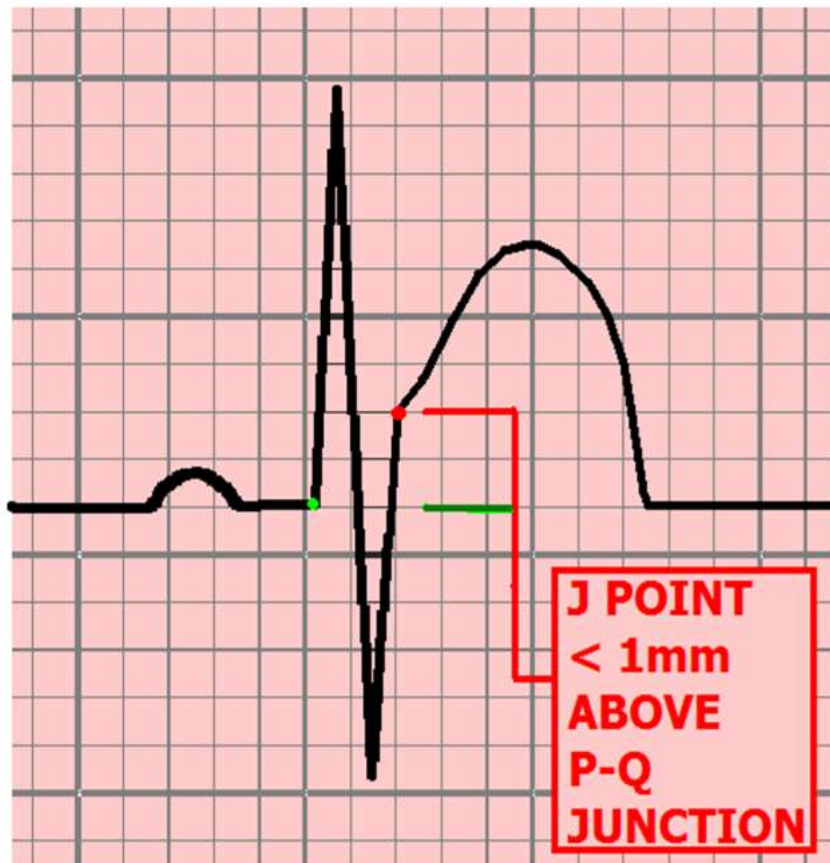
↓ **SENSITIVITY**
(FALSE NEGATIVES)

↓ **SPECIFICITY**
(FALSE POSITIVES)

AND . . .

PROBLEMS WITH SPECIFICITY . . .

S-T SEGMENT ELEVATION - COMMON ETIOLOGIES:



CONDITION:

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

1North (06)

Rate 83 . SINUS RHYTHM.....normal P axis, V-rate 50- 99
PR 152 . RIGHT BUNDLE BRANCH BLOCK.....QRSd>120, terminal axis(90,270)
QRS 122 . ANTEROLATERAL INFARCT, ACUTE.....Q >35ms, ST >0.20mV, V2-V6
QT 412
QTc 485

--AXIS--

P 59

QRS 14

T 33

12 Lead; Standard Placement

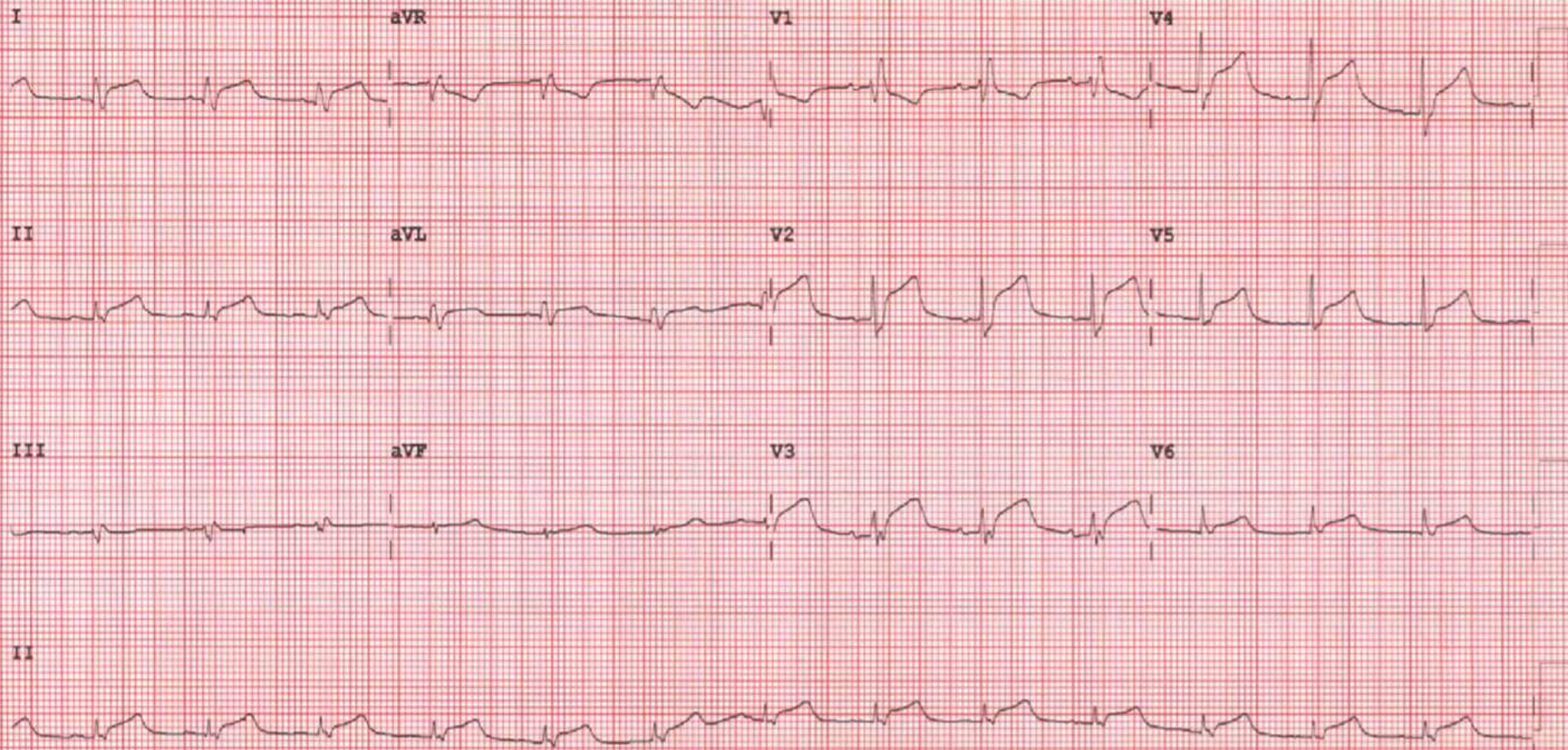
- ABNORMAL ECG -

>>> Acute MI <<<

Requested by:

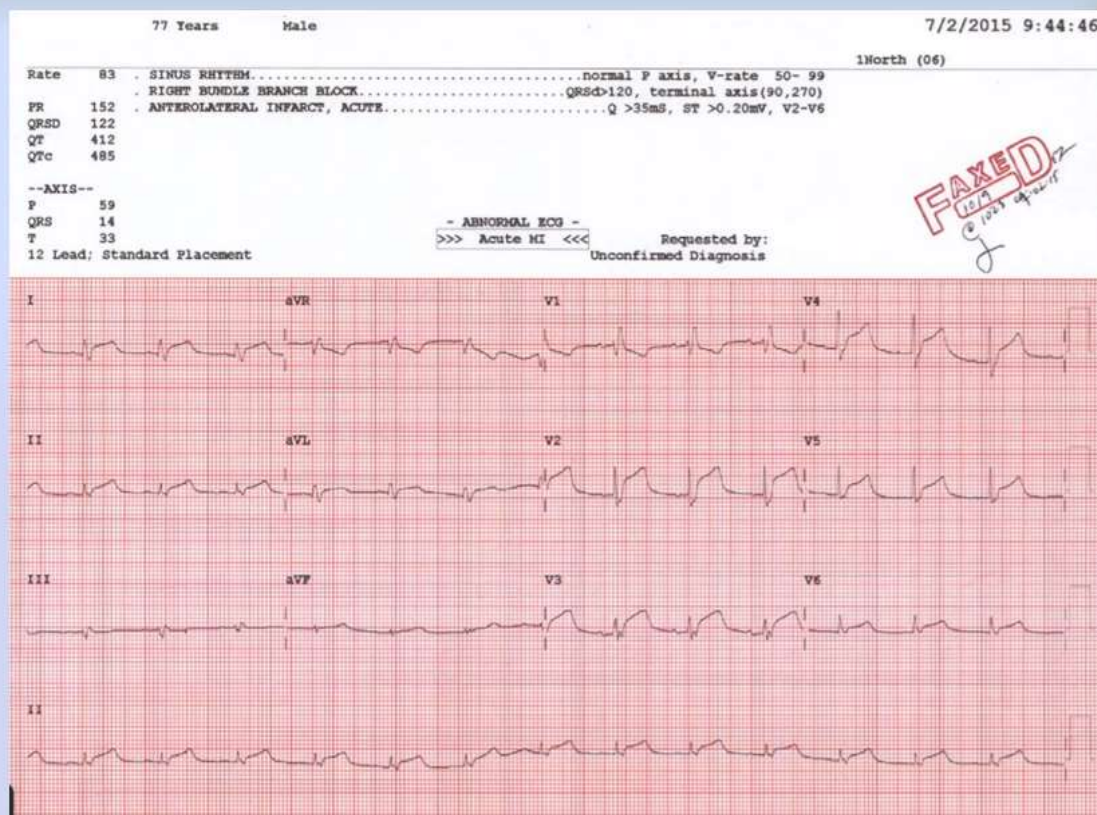
Unconfirmed Diagnosis

FAXED
10/19
@ 1023 07:02:15
J



Patient:

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



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



EKGs in PERSPECTIVE, con't:



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



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



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



PRE-TEST EKG.
PATIENT STANDING,
- ASYMPTOMATIC.

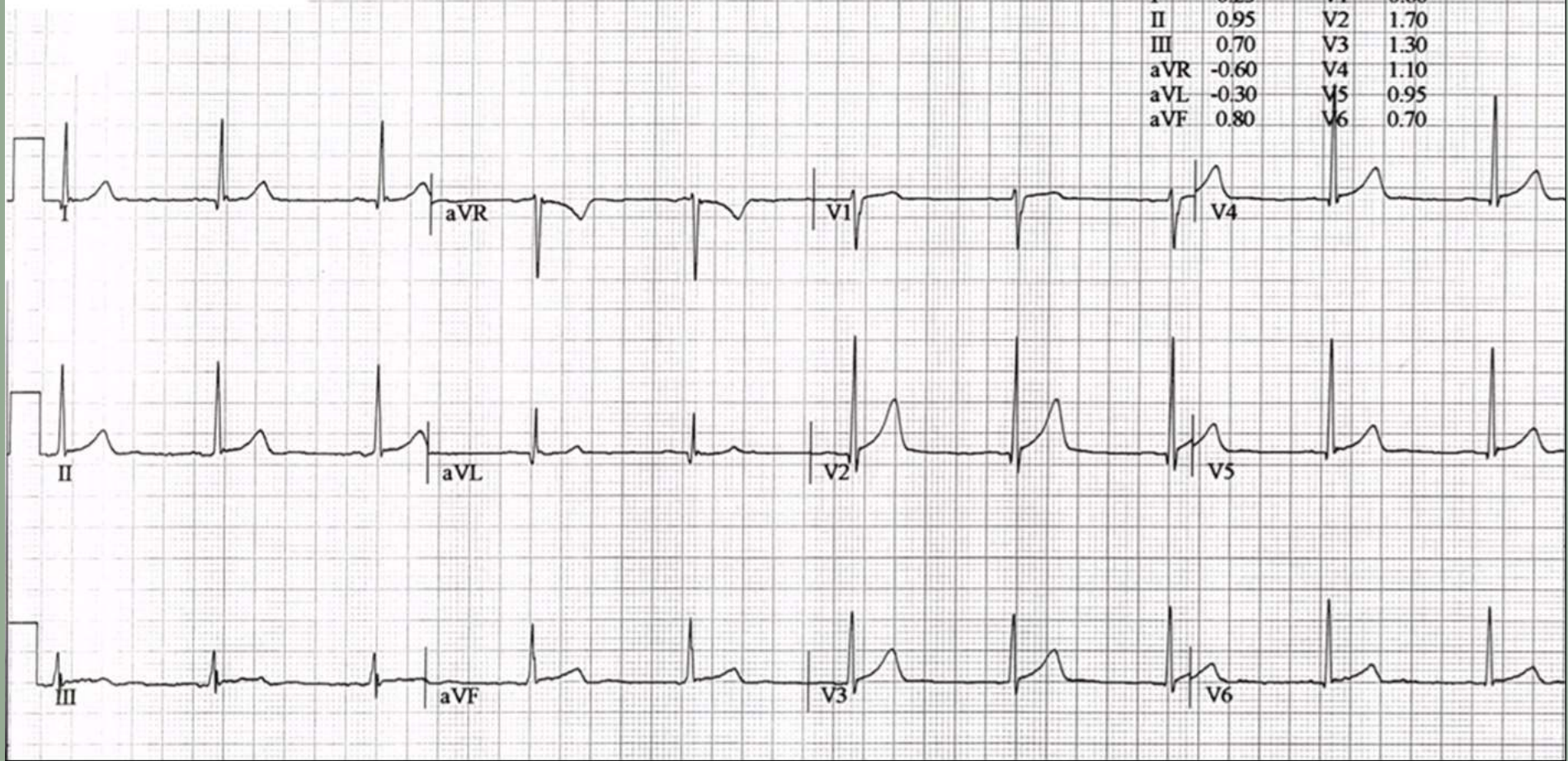
58 bpm
00:56 118/68 mmHg

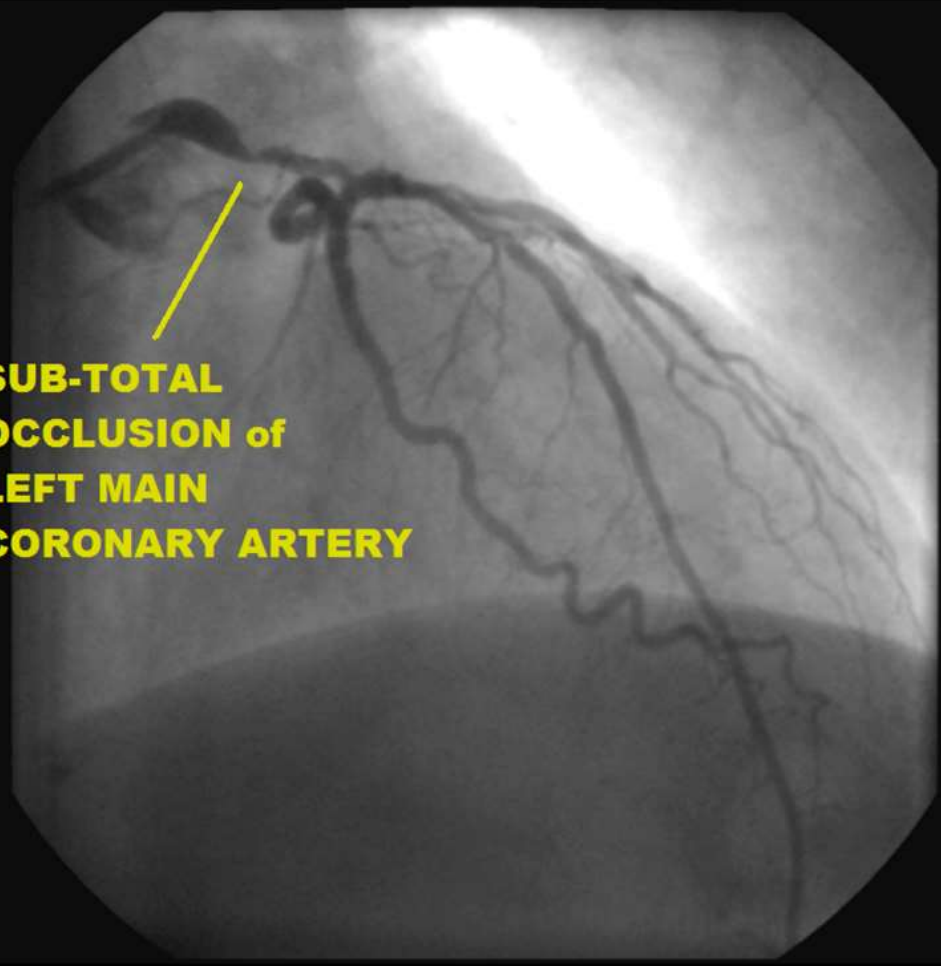
PRETEST
STANDING
00:58

BRUCE
0.0 mph
0.0 %

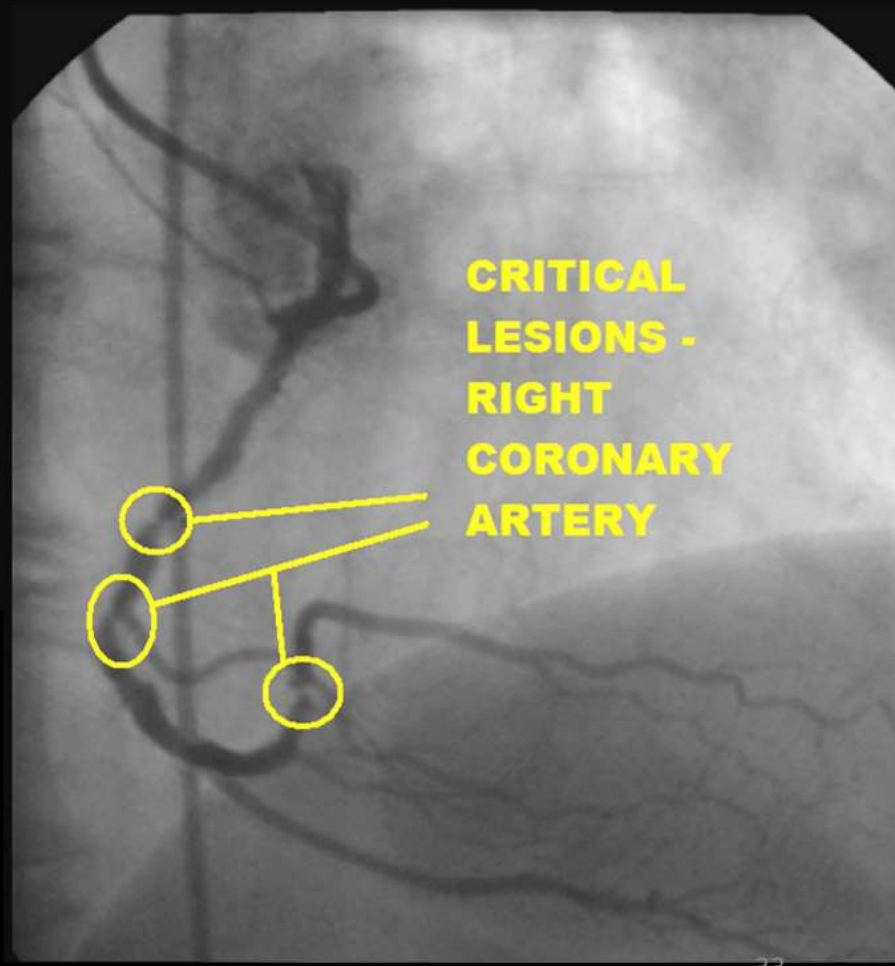
Measured at 60ms Post J (10mm/mV)
Auto Points

Lead	ST(mm)	Lead	ST(mm)
I	0.25	V1	0.60
II	0.95	V2	1.70
III	0.70	V3	1.30
aVR	-0.60	V4	1.10
aVL	-0.30	V5	0.95
aVF	0.80	V6	0.70





**SUB-TOTAL
OCCLUSION of
LEFT MAIN
CORONARY ARTERY**



**CRITICAL
LESIONS -
RIGHT
CORONARY
ARTERY**

*“From time to time,
the EKG – derived
diagnosis will be
TOTALLY INCORRECT.”*

**Despite the ECG's problematic
issues with**

**Lack of Sensitivity
&**

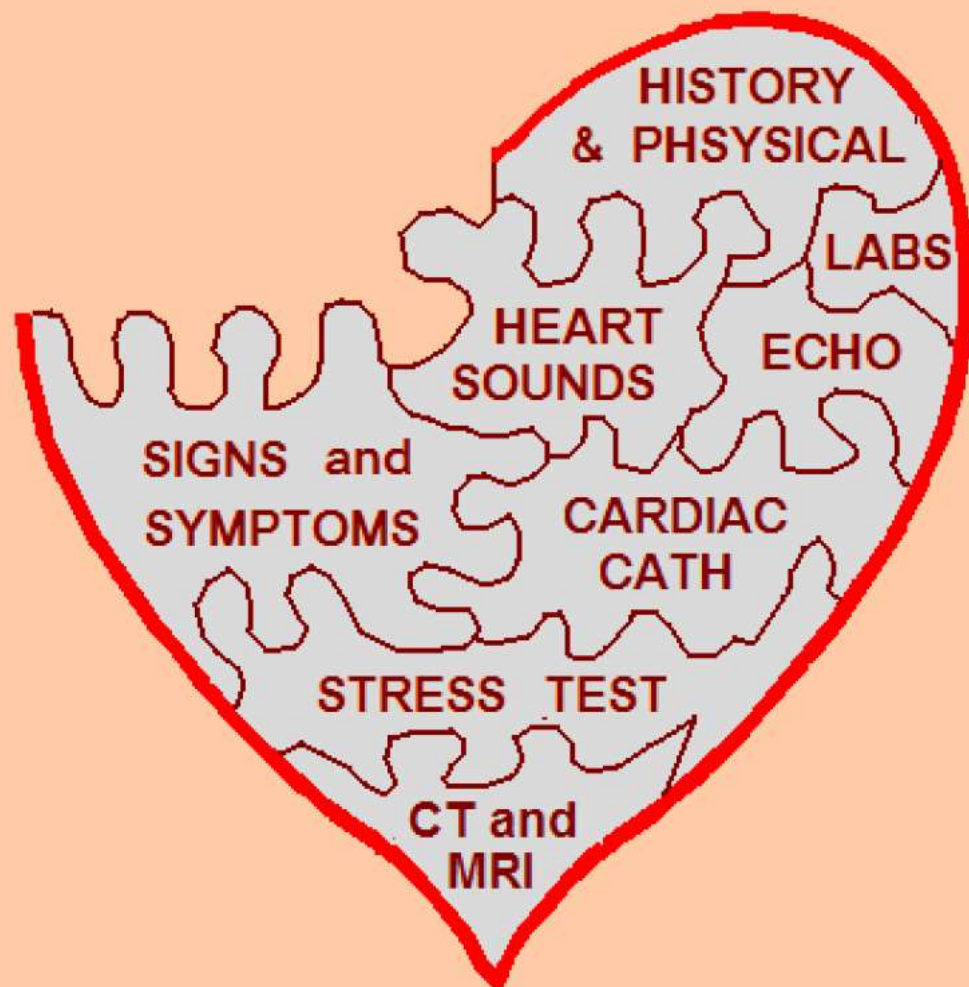
Lack of Specificity,

***The 12 Lead ECG remains
one of our QUICKEST, most cost-
efficient front-line Triage Tools
that we have today.***

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



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

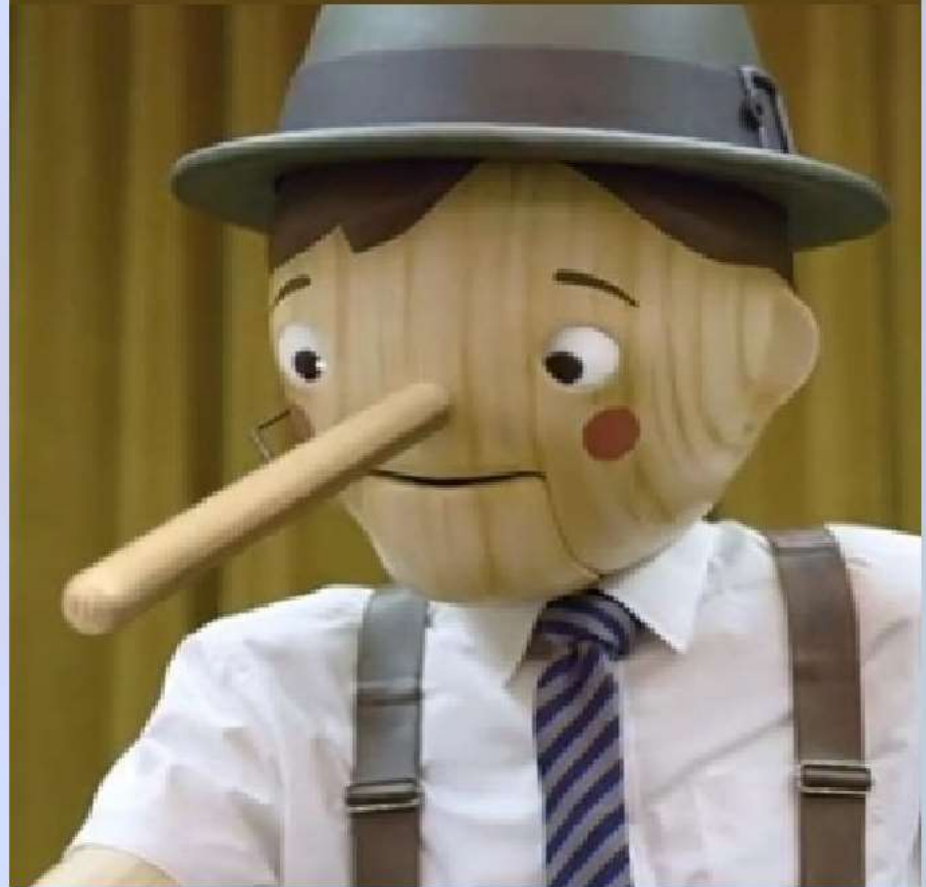




So how do we know when the ECG is telling us the truth ???

- ***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.***

The ECG . . .



HEART

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

***Risk factors for atherosclerotic disease:**

Hypercholesterolemia	Cigarette smoking
Hypertension	Positive family history
Diabetes Mellitus	Obesity

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



HEART Pathway 12+

Chest pain. Risk-stratified.

[Impathiq](#)

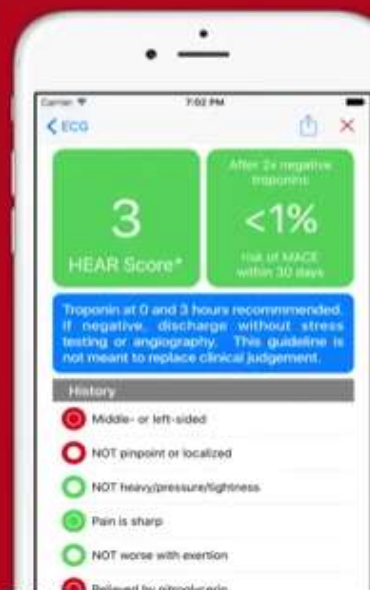
Designed for iPhone

★★★★★ 4.5 • 13 Ratings

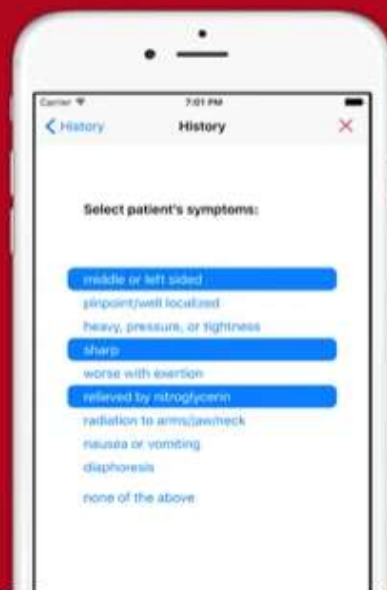
Free

iPhone Screenshots

Use a validated cardiac risk score to avoid unnecessary testing



The HEART Pathway uses history, ECG, and other key risk factors



The HEART Pathway can be done in less than 30 seconds at bedside



The HEART Pathway has been shown to save \$200 per chest pain patient

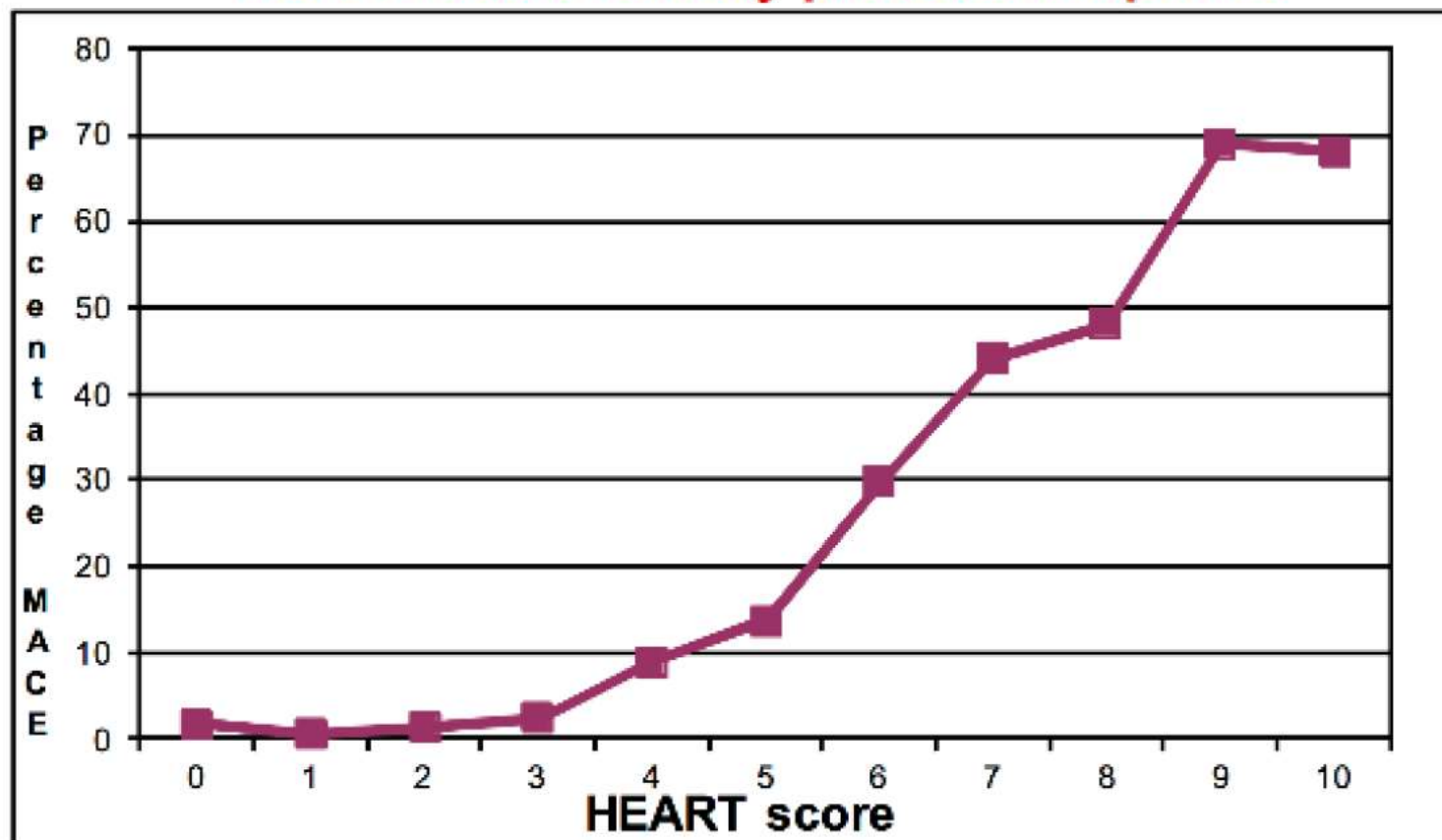


The HEART Score

Score	Common Diagnosis:	Disposition:
0-3	Low Risk Chest Pain	Early Discharge with referral
4-6	Low Risk Chest Pain Unstable Angina	Observation Unit or Admission Tele
7-10	Unstable Angina NSTEMI STEMI	Tele Admission ICU Admission STAT Cath Lab

Heart Score Reliability

HEART score reliably predicts endpoints



CAUSES OF EKG ABNORMALITIES

PROBLEMS WITH THE HEART'S

- BLOOD SUPPLY
- ELECTRICAL SYSTEM
- PHYSICAL STRUCTURE
- CELLULAR FUNCTION

CAUSES OF EKG ABNORMALITIES

PROBLEMS WITH THE HEART'S

- BLOOD SUPPLY

- ISCHEMIA
- INFARCTION
- NECROSIS

- ELECTRICAL SYSTEM

- PHYSICAL STRUCTURE

- CELLULAR FUNCTION

CAUSES OF EKG ABNORMALITIES

PROBLEMS WITH THE HEART'S

- BLOOD SUPPLY
- ELECTRICAL SYSTEM
 - AUTOMATICITY
 - REENTRY
 - PRE-EXCITATION
- PHYSICAL STRUCTURE
- CELLULAR FUNCTION

CAUSES OF EKG ABNORMALITIES

PROBLEMS WITH THE HEART'S

- BLOOD SUPPLY
- ELECTRICAL SYSTEM
- PHYSICAL STRUCTURE
 - CONGENITAL DEFECTS
 - HYPERTROPHY
 - VALVULAR DYSFUNCTION
- CELLULAR FUNCTION

CAUSES OF EKG ABNORMALITIES

PROBLEMS WITH THE HEART'S

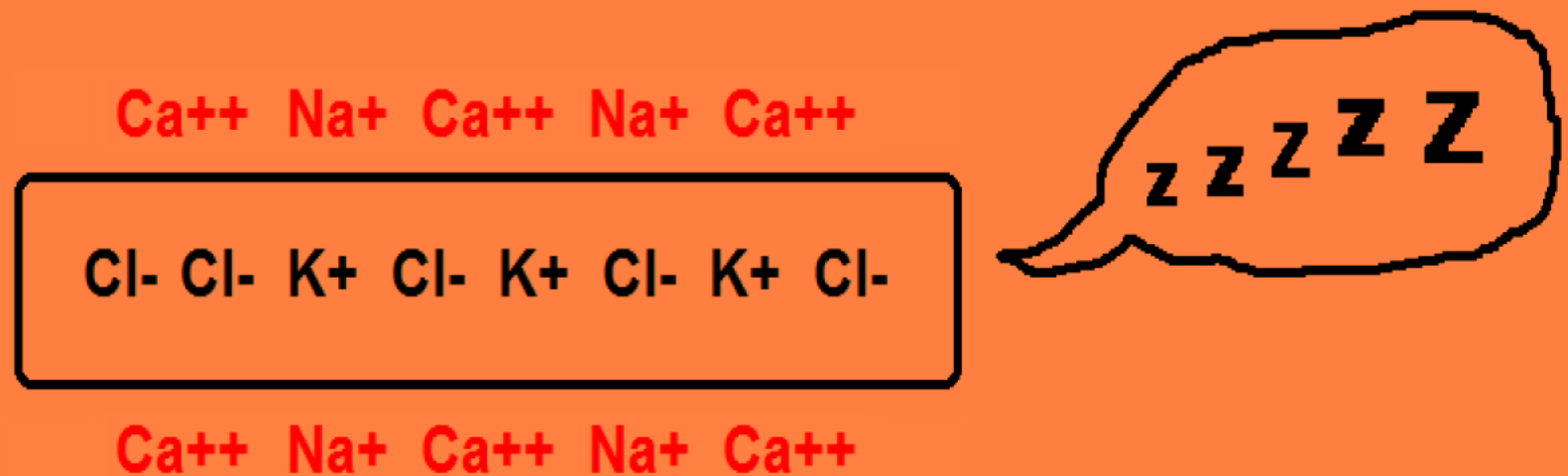
- BLOOD SUPPLY
- ELECTRICAL SYSTEM
- PHYSICAL STRUCTURE
- CELLULAR FUNCTION
 - ELECTROLYTES
 - MEDICATIONS / DRUGS
 - BODY TEMPERATURE
 - PH

Cardiac A & P



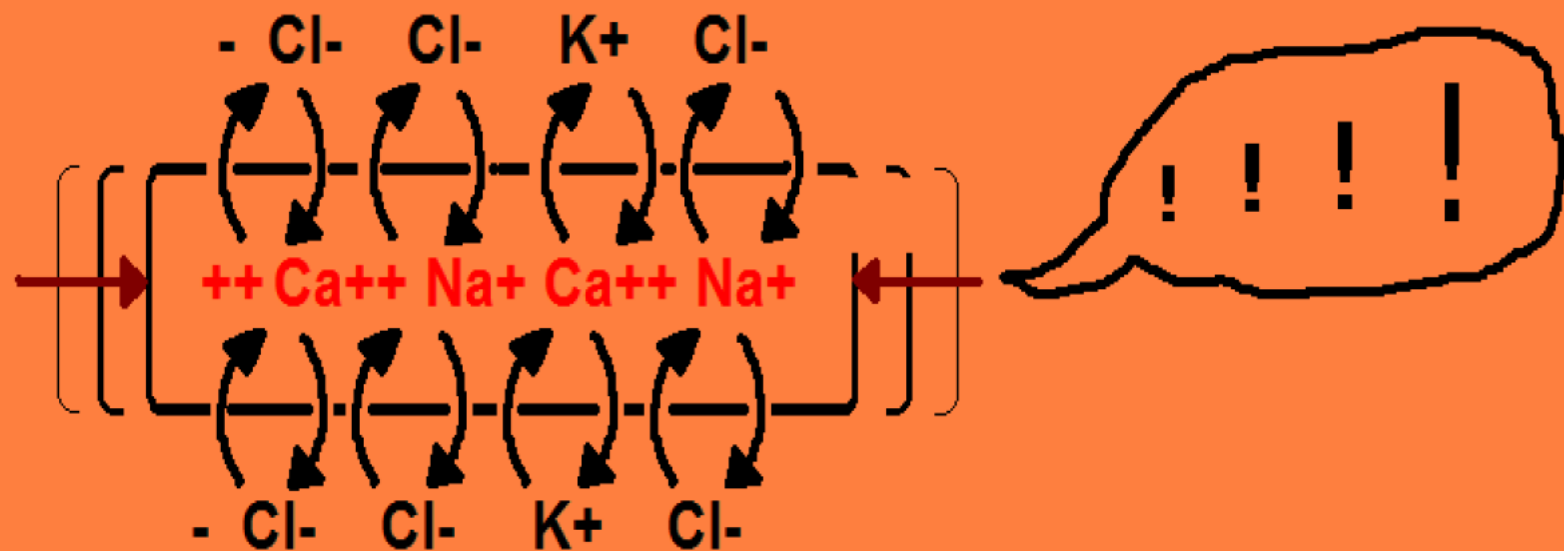
CARDIAC ANATOMY and PHYSIOLOGY "101"

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



CARDIAC ANATOMY and PHYSIOLOGY "101"

... 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!**

CARDIAC ANATOMY and PHYSIOLOGY "101"

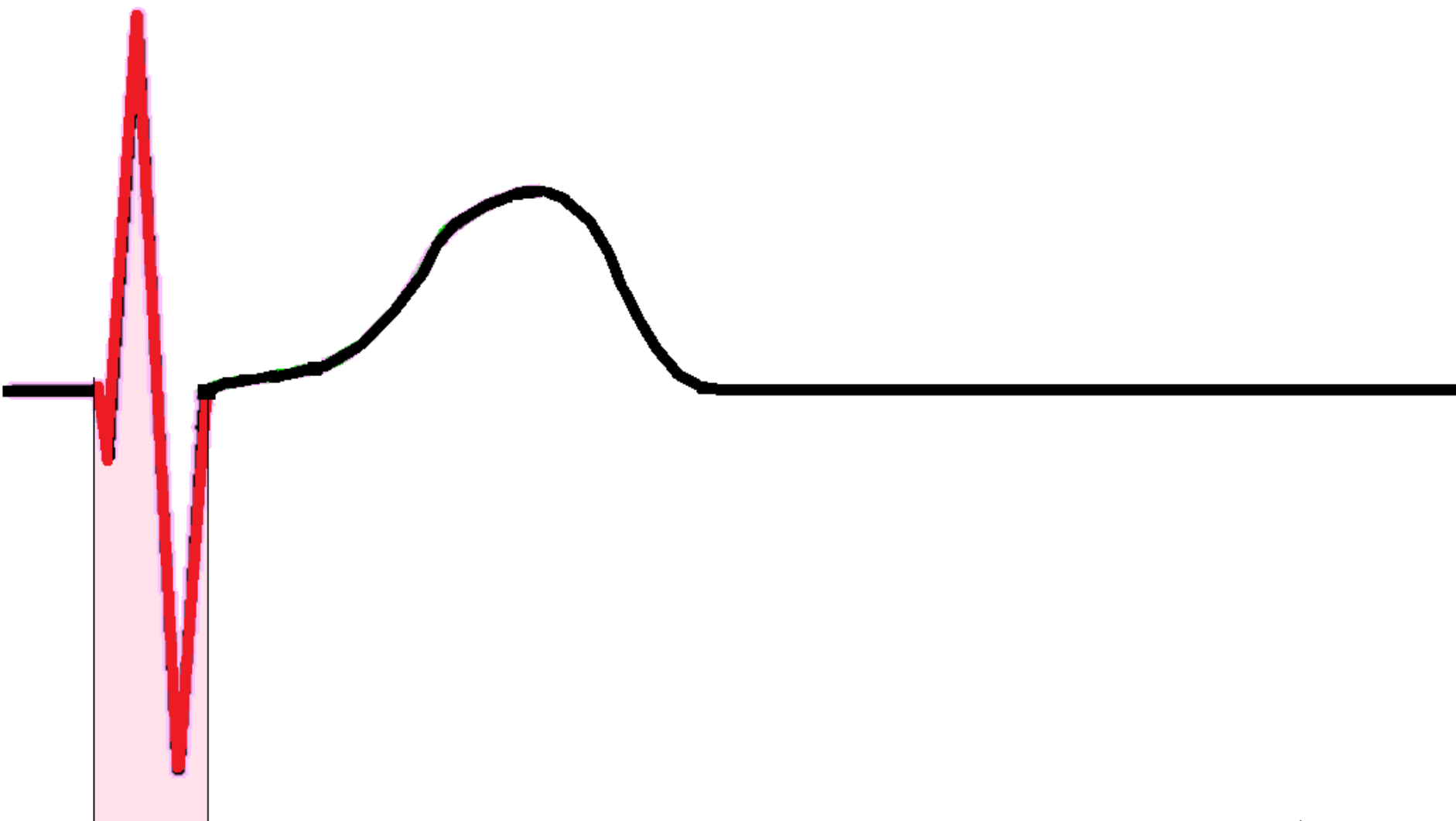
THIS (OF COURSE) IS KNOWN AS . . .

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

Ventricular Depolarization:

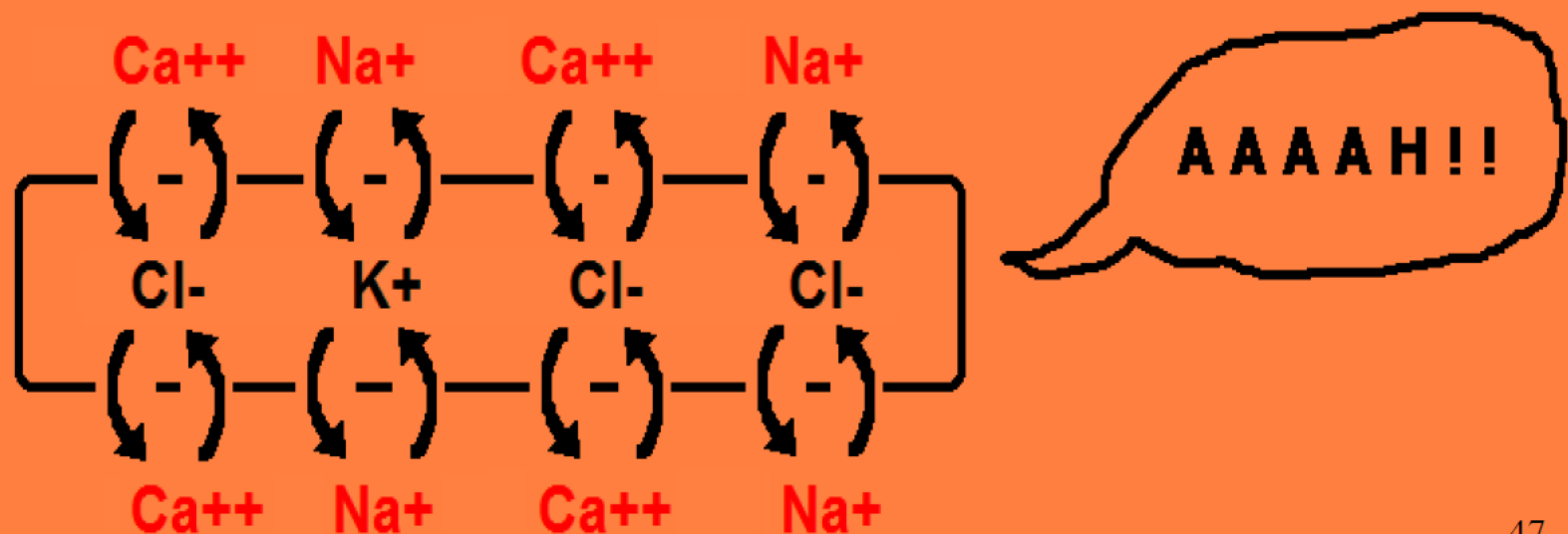
- Is represented by the **QRS Complex**



QRS Complex = Ventricular Depolarization

CARDIAC ANATOMY and PHYSIOLOGY "101"

AFTER DEPOLARIZATION, THE CELLS RELAX.
THE IONS RETURN TO THEIR ORIGINAL POSITIONS --
THIS PROCESS IS KNOWN AS **REPOLARIZATION**

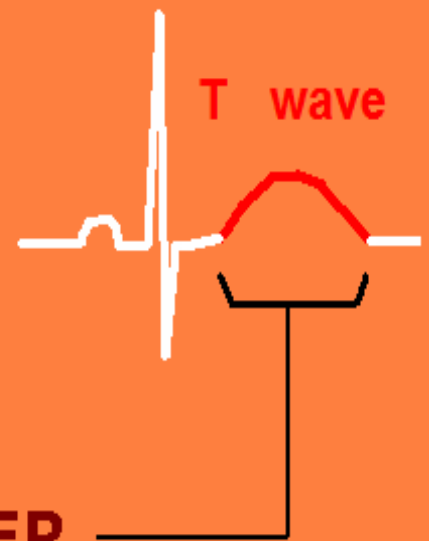


CARDIAC ANATOMY and PHYSIOLOGY "101"



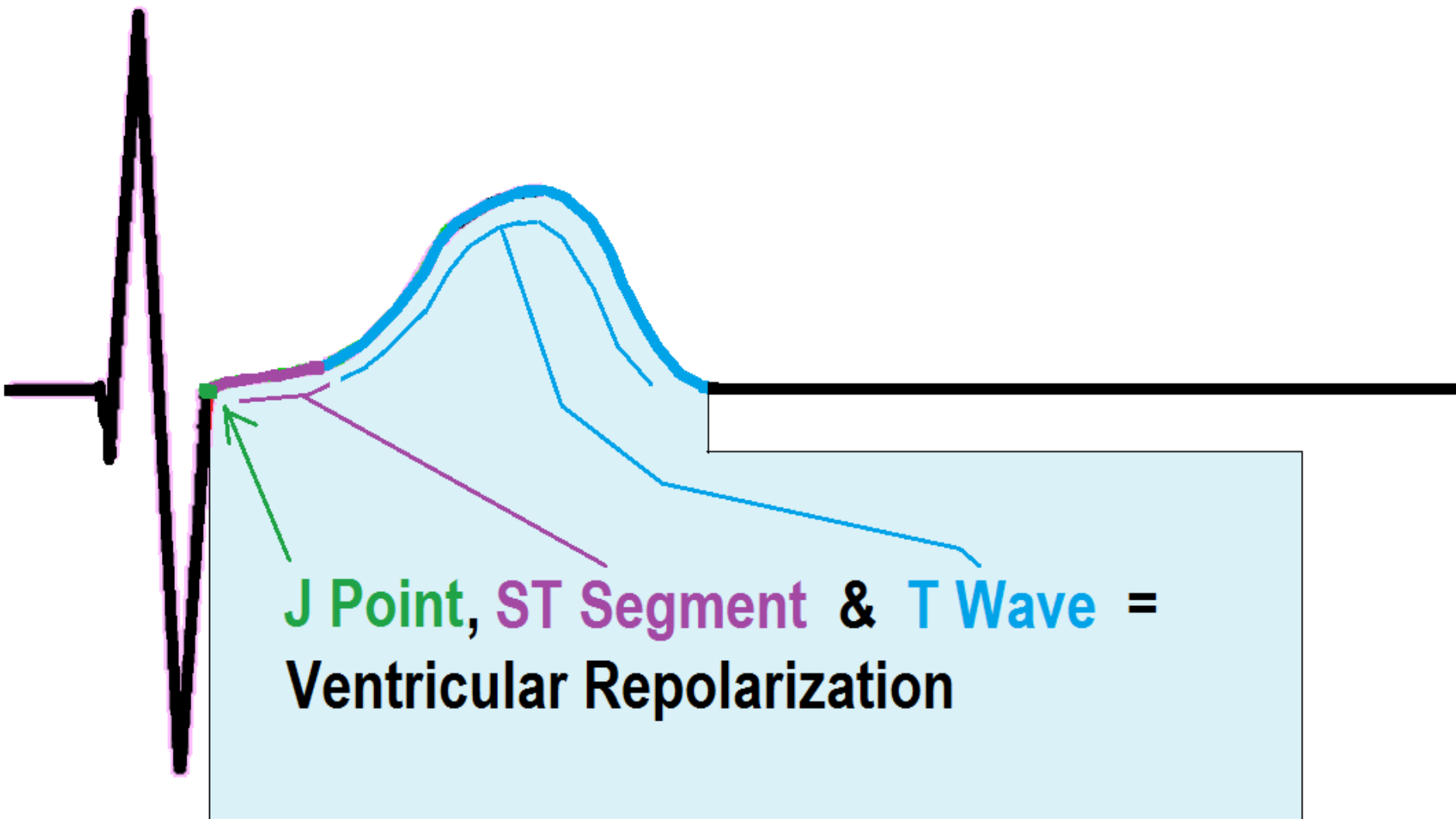
IMPORTANT CONCEPT:

ALL MYOCARDIAL CELLS **DO NOT**
REPOLARIZE AT THE EXACT
SAME MOMENT --
LIKE DEPOLARIZATION,
THE PROCESS OF REPOLARIZATION
OCCURS IN A "WAVE-LIKE" MANNER

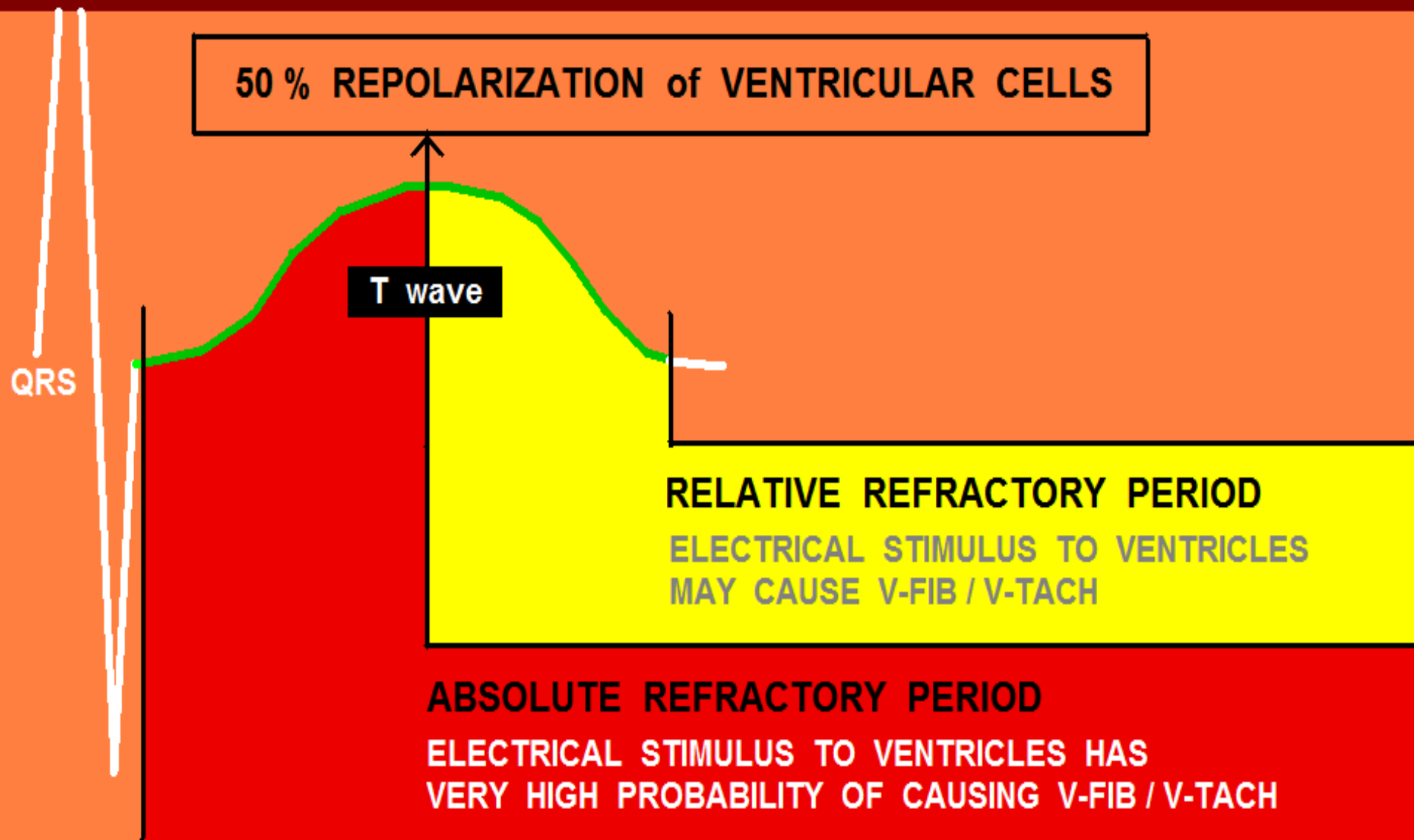


Repolarization on the ECG:

- Is represented by the:
 - **J Point**
 - **ST Segment**
 - **T Wave**



CARDIAC ANATOMY and PHYSIOLOGY "101"



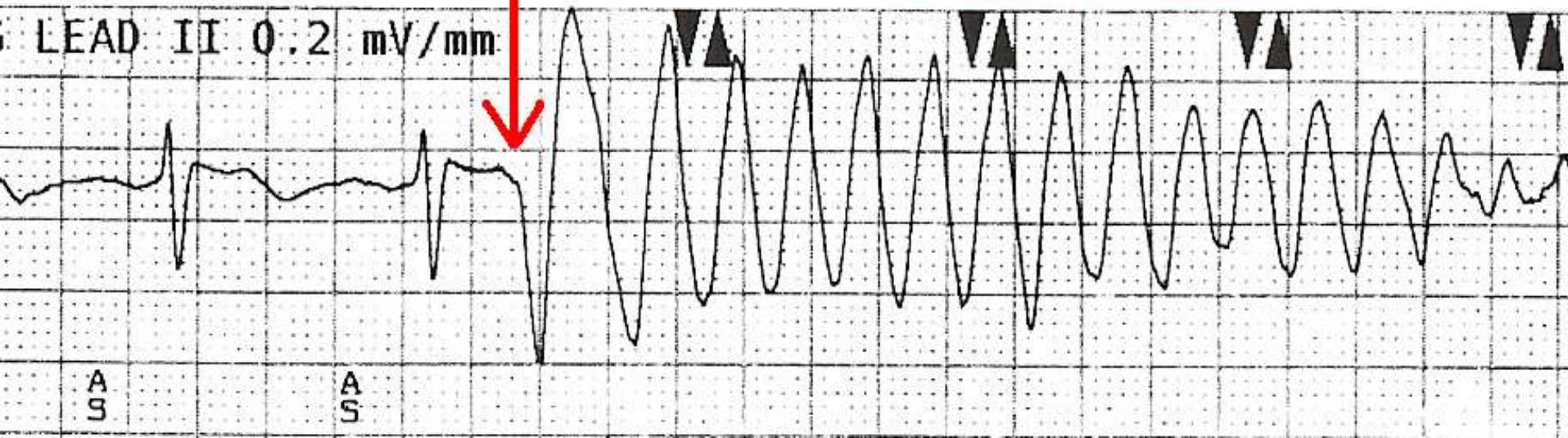
ROUTINE TEST OF ICD

ELECTRICAL IMPULSE
ADMINISTERED DURING ABSOLUTE
REFRACTORY PERIOD -- INDUCES
VENTRICULAR FIBRILLATION

08-Sep-2006 18:01:47

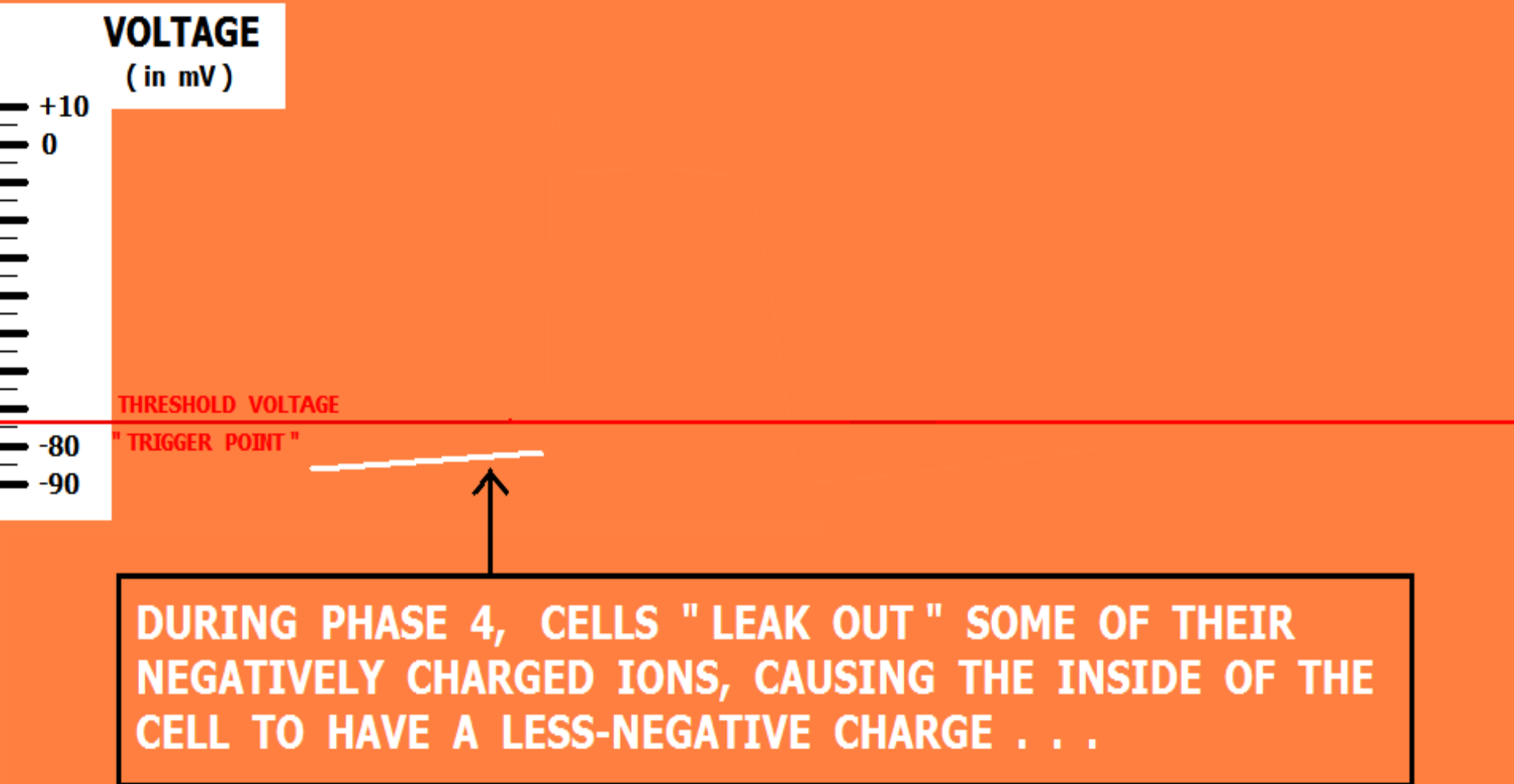
Test Started

SPECIAL THANKS TO:
Ray Heinley
Medtronic Corporation
for this contribution



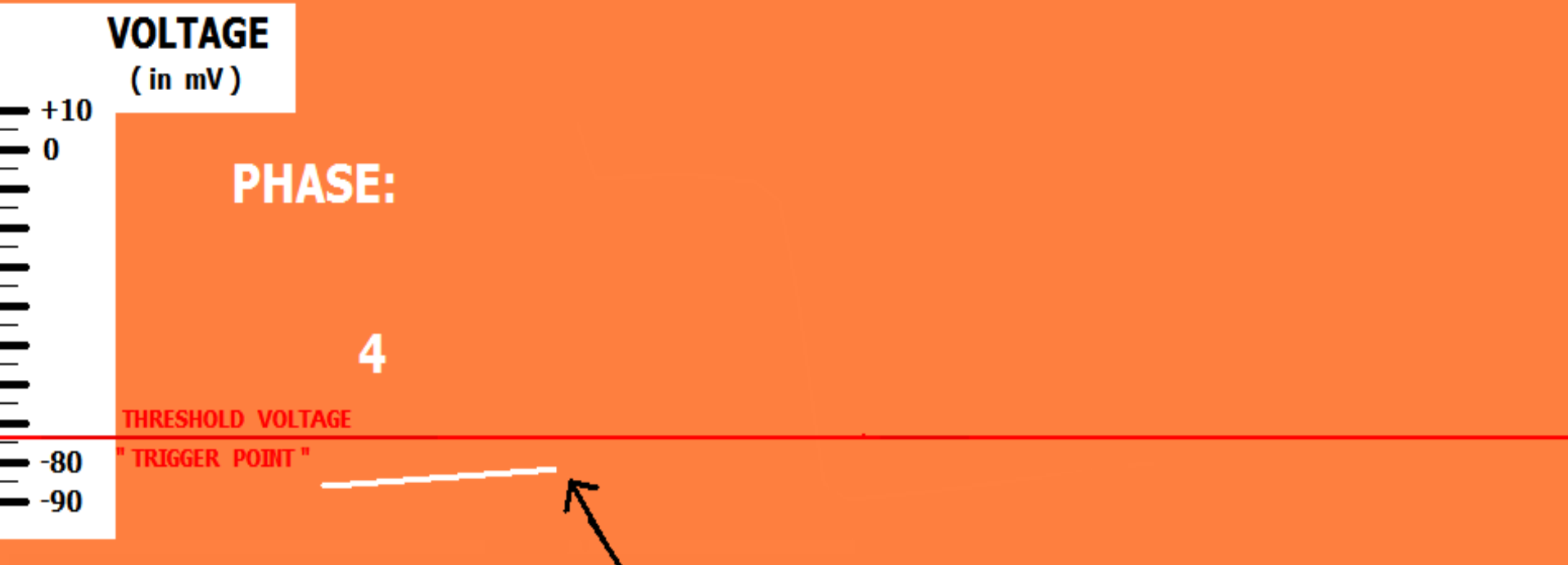
CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL



CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL



ONE OF TWO EVENTS WILL CAUSE THE CELL TO DEPOLARIZE:

CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL



ONE OF TWO EVENTS WILL CAUSE THE CELL TO DEPOLARIZE:

1. A NEIGHBORING CELL DEPOLARIZES, TRIGGERING A "CHAIN REACTION"

CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL

CELL " STATUS: "

- 4: • CELL COMPLETELY
REPOLARIZED
- -80 to -90 mV CHARGE
 - SLIGHT "LEAKAGE" OF IONS

PHASE:

4

THRESHOLD VOLTAGE

" TRIGGER POINT "



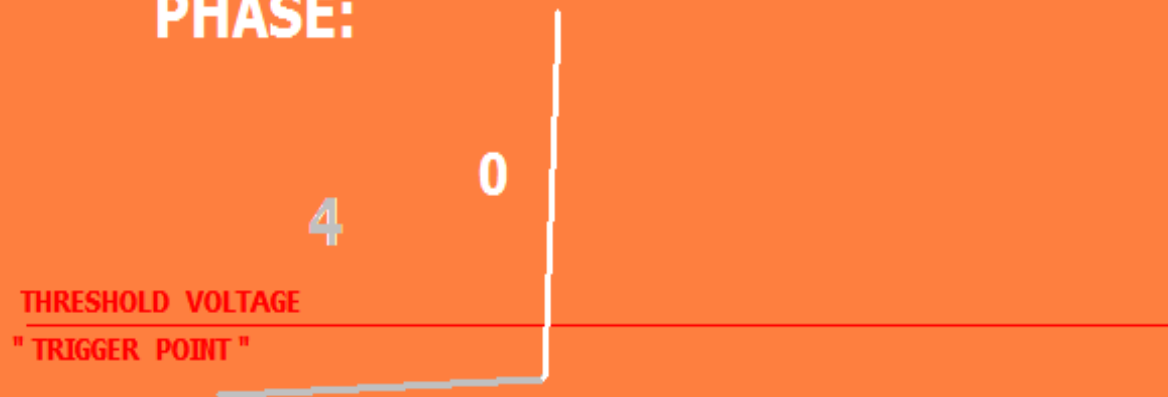
ECG



CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL

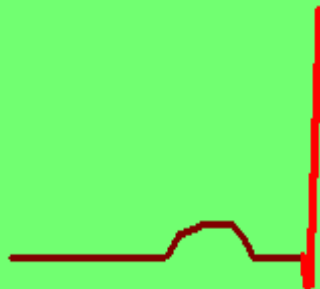
PHASE:



CELL "STATUS: "

- 4
 - CELL REPOLARIZED
 - -80 to -90 mV CHARGE
 - SLIGHT "LEAKAGE" OF IONS
- 0:
 - IN TYPICAL MUSCLE CELLS, PHASE 0 INITIATED BY:
CELLS "PUSHED OVER TRIGGER POINT" BY:
 - PACEMAKER CELLS
 - NEIGHBOR MUSCLE CELL DEPOLARIZATION
 - RAPID INFLUX OF POSITIVELY CHARGED SODIUM IONS via "FAST CHANNELS"
 - CELL DEPOLARIZATION

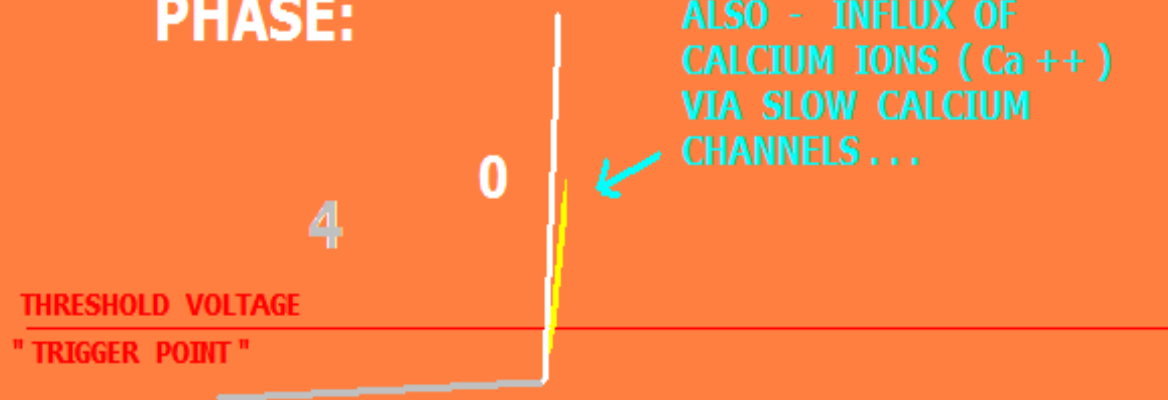
ECG



CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL

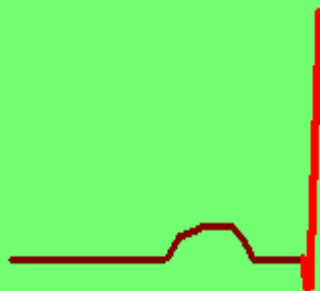
PHASE:



CELL "STATUS: "

- 4
 - CELL REPOLARIZED
 - -80 to -90 mV CHARGE
 - SLIGHT "LEAKAGE" OF IONS
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 - IN TYPICAL MUSCLE CELLS, PHASE 0 INITIATED BY:
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 - PACEMAKER CELLS
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 - CELL DEPOLARIZATION

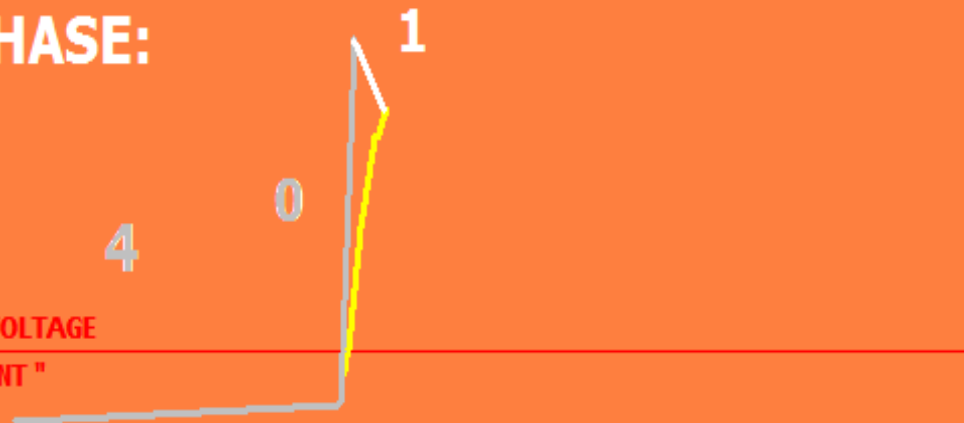
ECG



CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL

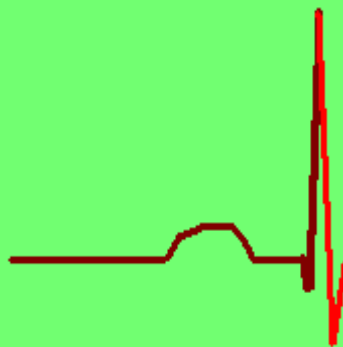
PHASE:



CELL "STATUS: "

- 4
 - CELL REPOLARIZED
 - -80 to -90 mV CHARGE
 - SLIGHT "LEAKAGE" OF IONS
- 0
 - RAPID INFLUX OF + CHARGED SODIUM IONS
 - CELL DEPOLARIZATION
- 1:
 - SODIUM IONS BEGIN TO EXIT THE CELL
 - THIS BEGINS THE REPOLARIZATION PROCESS

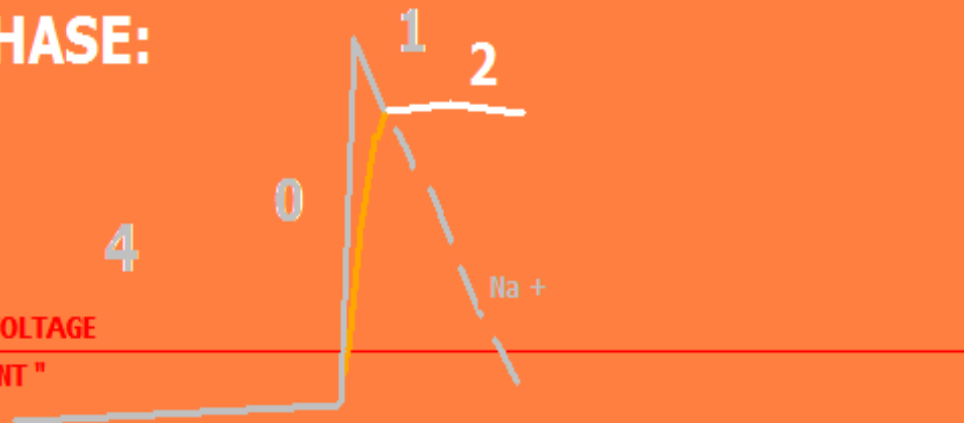
ECG



CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL

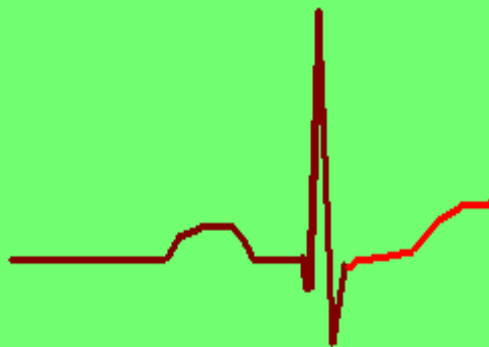
PHASE:



CELL "STATUS: "

- 4 • CELL REPOLARIZED
- -80 to -90 mV CHARGE
- SLIGHT "LEAKAGE" OF IONS
- 0 • RAPID INFLUX OF + CHARGED SODIUM IONS
- CELL DEPOLARIZATION
- 1 • SODIUM EXITS CELL
- REPOLARIZATION BEGINS
- 2 • CALCIUM IONS ARE COMPLETING THEIR "SLOW ENTRY" OF CARDIAC CELLS, PROLONGING THE ACTION POTENTIAL

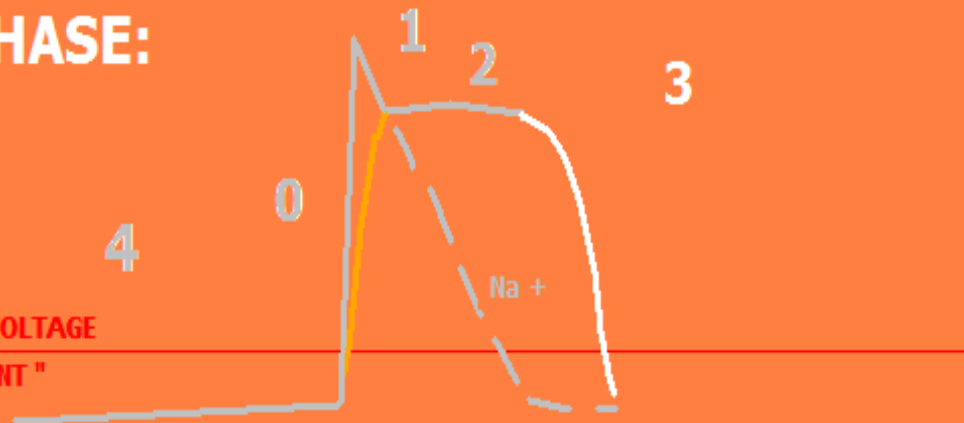
ECG



CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL

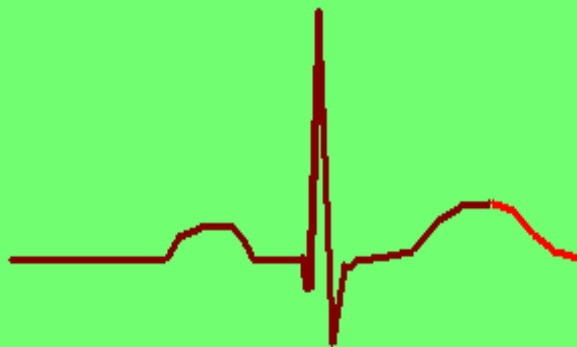
PHASE:



CELL "STATUS:"

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 - -80 to -90 mV CHARGE
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 - CELL DEPOLARIZATION
- 1 • SODIUM EXITS CELL
 - REPOLARIZATION BEGINS
- 2 • CALCIUM IONS CONTINUE TO ENTER CELL
- 3 • CALCIUM CHANNELS CLOSE

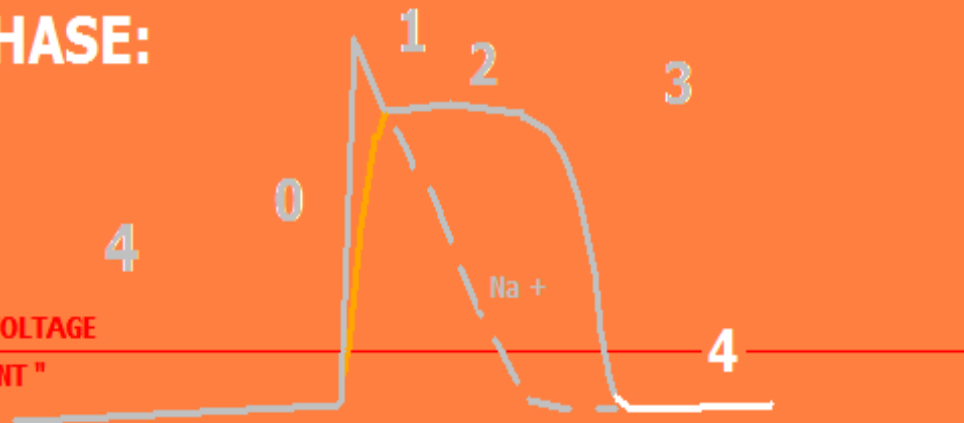
ECG



CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL

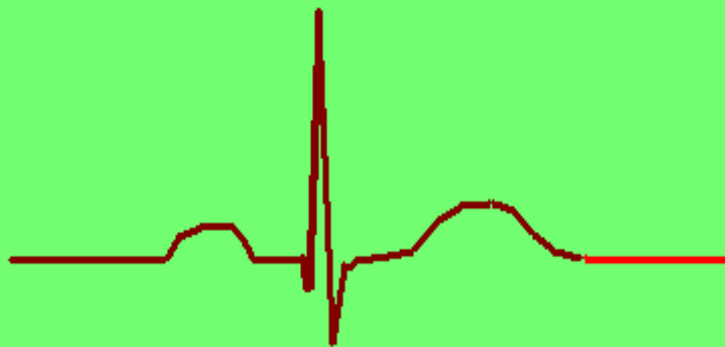
PHASE:



CELL "STATUS: "

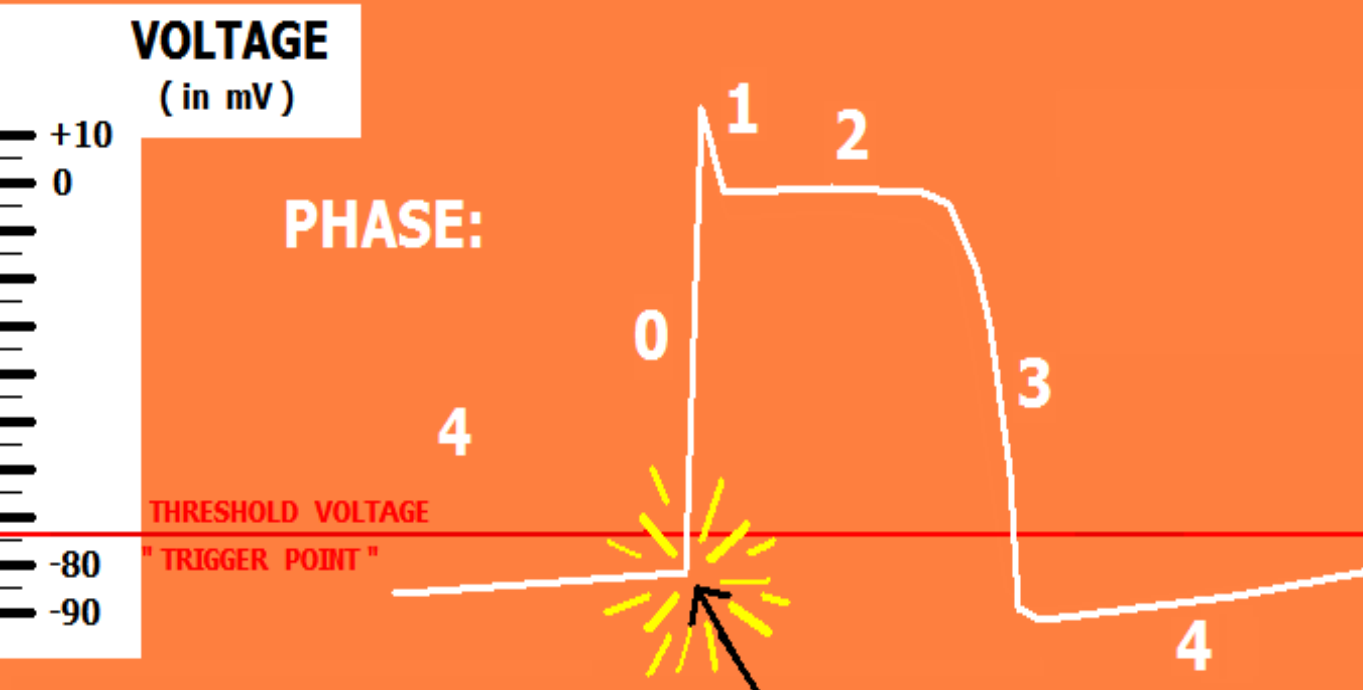
- 4 • CELL REPOLARIZED
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+ CHARGED SODIUM IONS
• CELL DEPOLARIZATION
 - 1 • SODIUM EXITS CELL
• REPOLARIZATION BEGINS
 - 2 • CALCIUM IONS CONTINUE
TO ENTER CELL
 - 3 • CALCIUM CHANNELS
CLOSE
- ONE CARDIAC CYCLE
(HEART BEAT) IS NOW
COMPLETE.

ECG



CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL

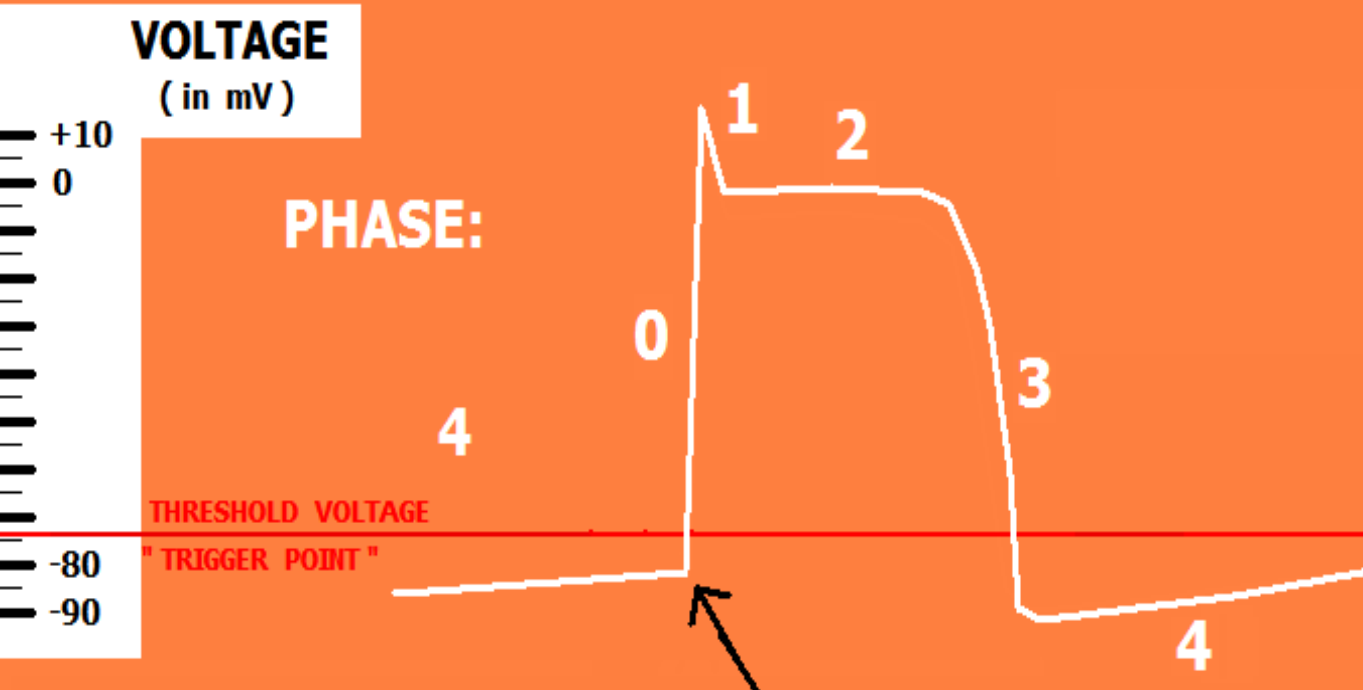


ONE OF TWO EVENTS WILL CAUSE THE CELL TO DEPOLARIZE:

1. A NEIGHBORING CELL DEPOLARIZES, TRIGGERING A "CHAIN REACTION" CAUSING EACH OF ITS NEIGHBORING CELLS TO DEPOLARIZE

CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL

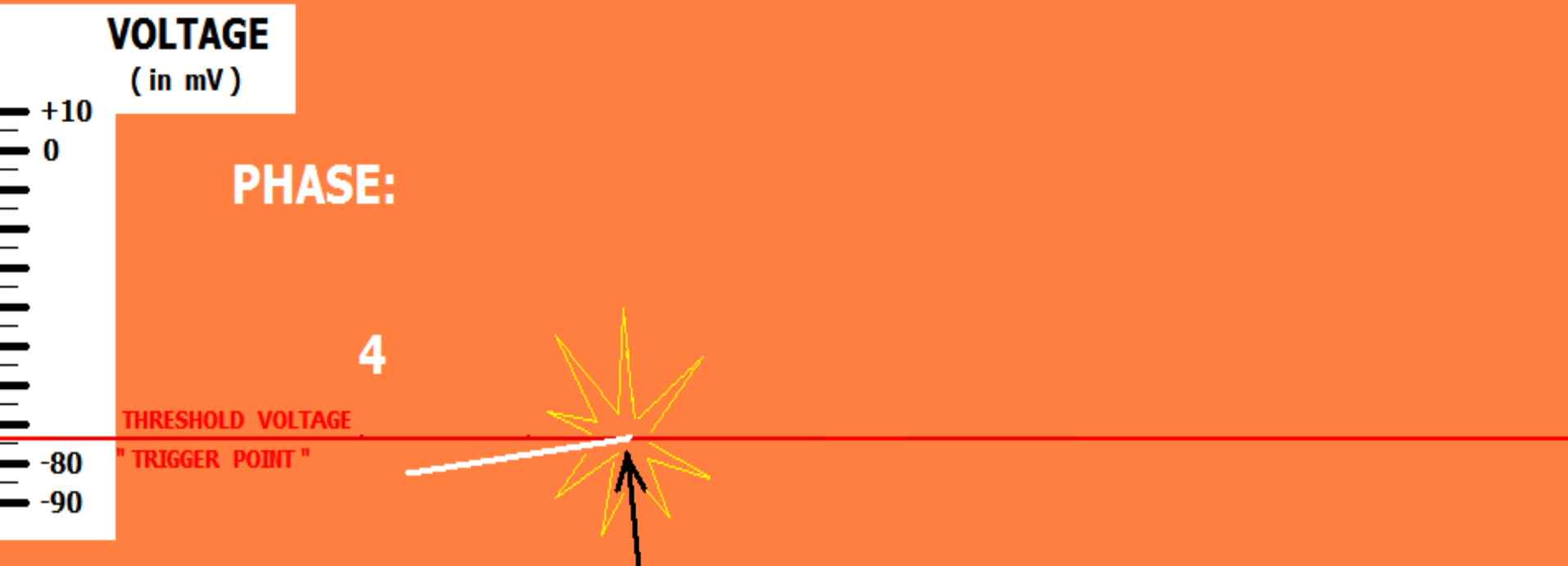


ONE OF TWO EVENTS WILL CAUSE THE CELL TO DEPOLARIZE:

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CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL

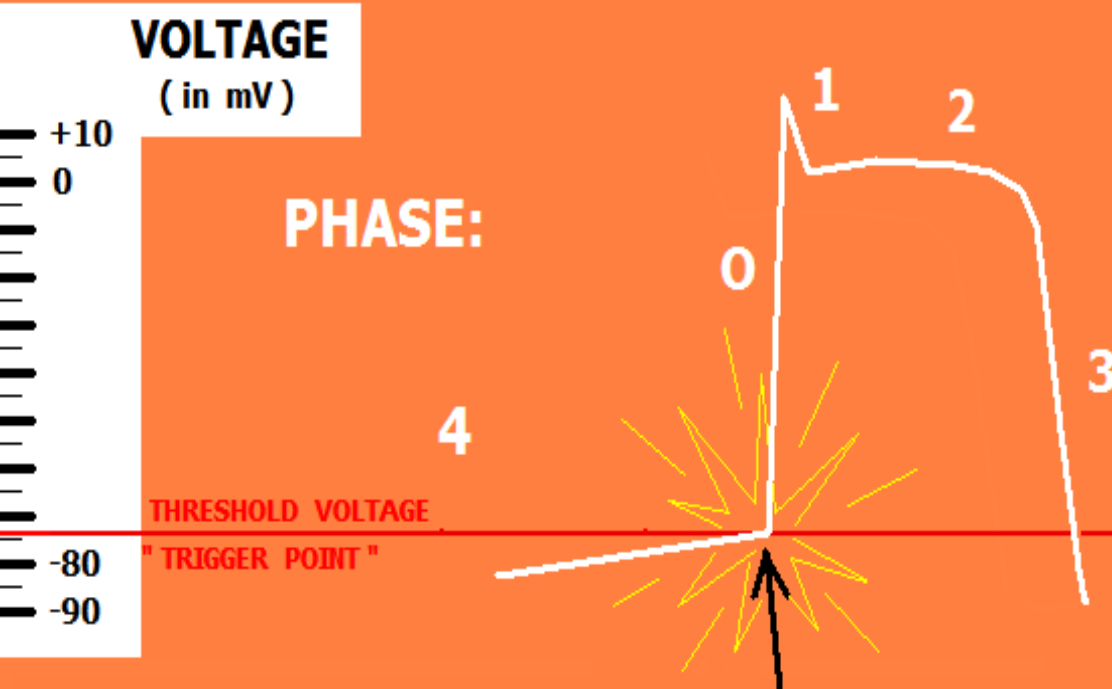


ONE OF TWO EVENTS WILL CAUSE THE CELL TO DEPOLARIZE:

2. THE CELL WILL "LEAK" ENOUGH OF IT'S OWN IONS TO CAUSE IT TO REACH THE **THRESHOLD VOLTAGE**, (a.k.a the **"TRIGGER POINT"**)

CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL

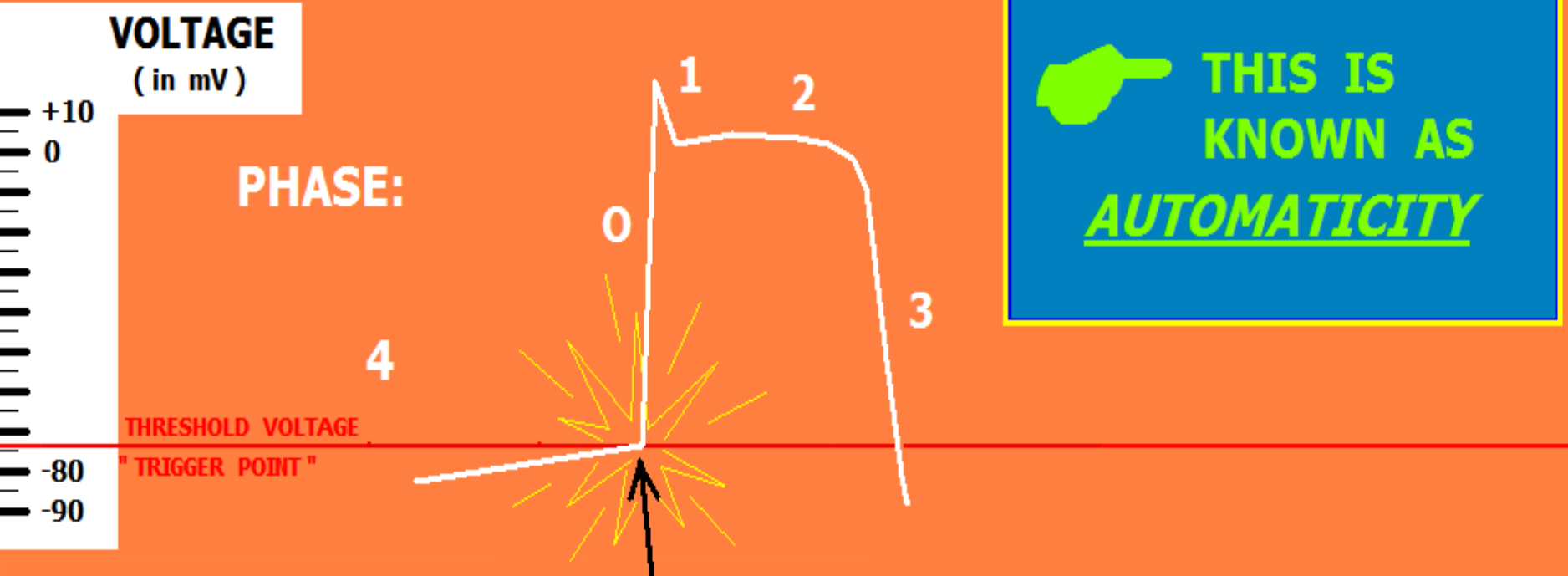


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CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL



ONE OF TWO EVENTS WILL CAUSE THE CELL TO DEPOLARIZE:

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CARDIAC ANATOMY and PHYSIOLOGY "101"

COMPONENTS OF ACTION POTENTIAL WAVEFORM

CARDIAC MUSCLE CELLS COMBINE



FAST SODIUM CHANNELS AND SLOW CALCIUM CHANNELS

CARDIAC ANATOMY and PHYSIOLOGY "101"

COMPONENTS OF ACTION POTENTIAL WAVEFORM

CARDIAC MUSCLE CELLS COMBINE

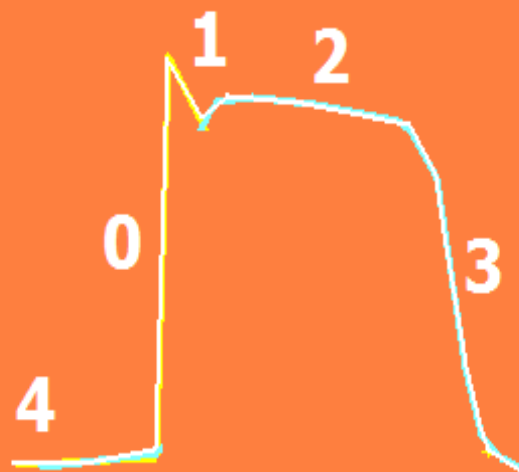


FAST SODIUM CHANNELS AND SLOW CALCIUM CHANNELS

CARDIAC ANATOMY and PHYSIOLOGY "101"

COMPONENTS OF ACTION POTENTIAL WAVEFORM

CARDIAC MUSCLE CELLS COMBINE



FAST SODIUM CHANNELS AND SLOW CALCIUM CHANNELS

. . . to get that familiar "action potential" shape !

CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL

PHASE:

**ACTION
POTENTIAL**

CELL " STATUS: "

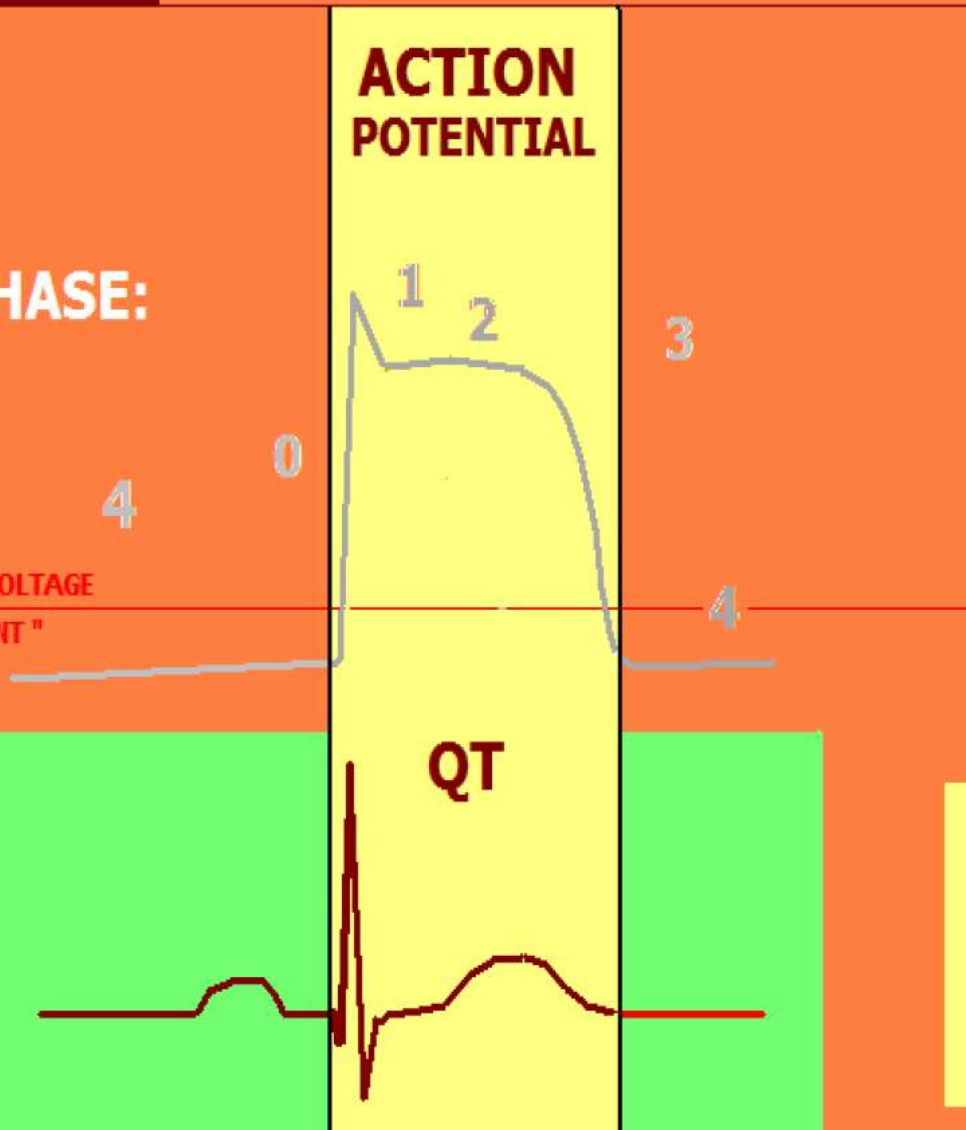
- 4 • CELL REPOLARIZED
 - -80 to -90 mV CHARGE
 - SLIGHT " LEAKAGE " OF IONS
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 - CELL DEPOLARIZATION
- 1 • SODIUM EXITS CELL
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- 3 • CALCIUM CHANNELS CLOSE

THRESHOLD VOLTAGE
" TRIGGER POINT "

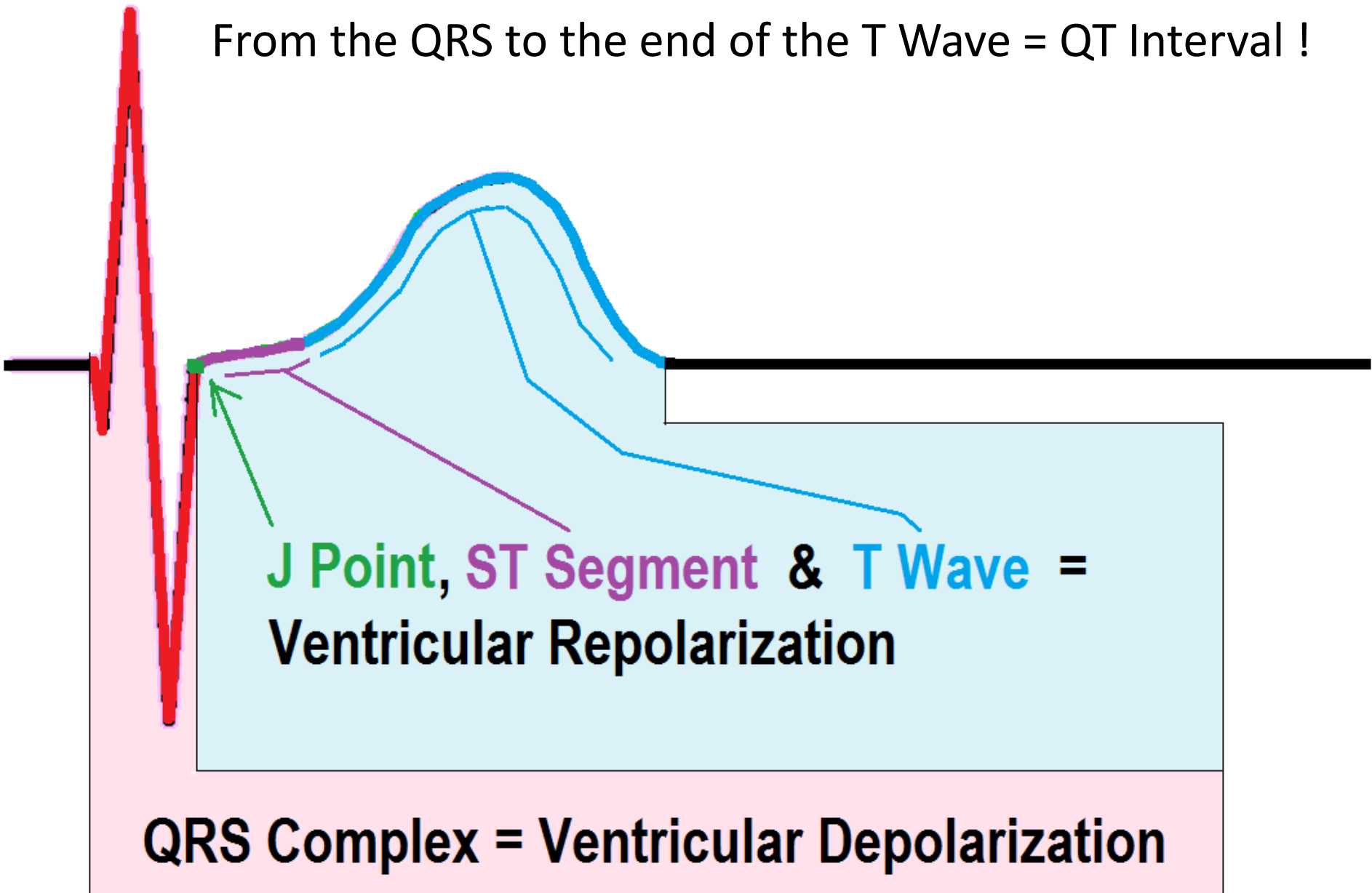
QT

**THE ACTION POTENTIAL
(OF VENTRICULAR MUSCLE CELLS)
IS ROUGHLY EQUAL TO
THE Q - T INTERVAL⁴⁸**

ECG



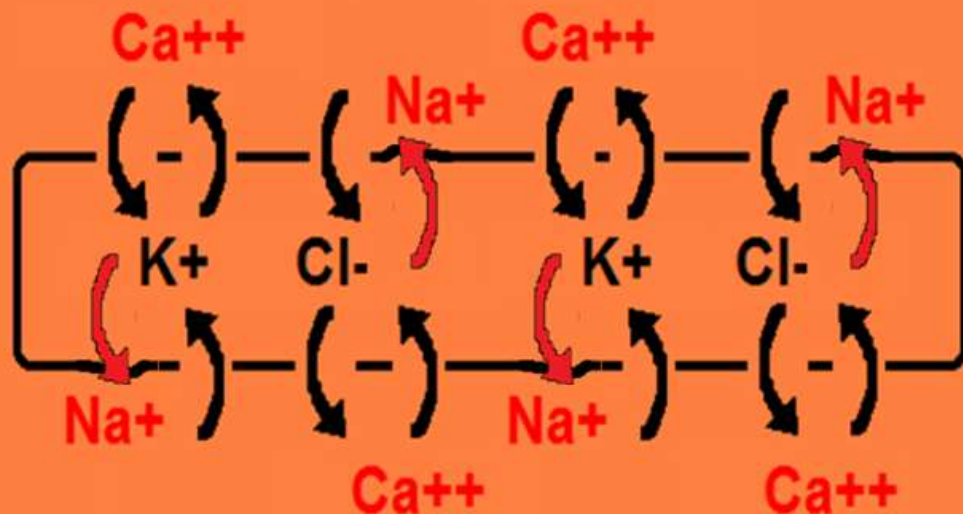
From the QRS to the end of the T Wave = QT Interval !



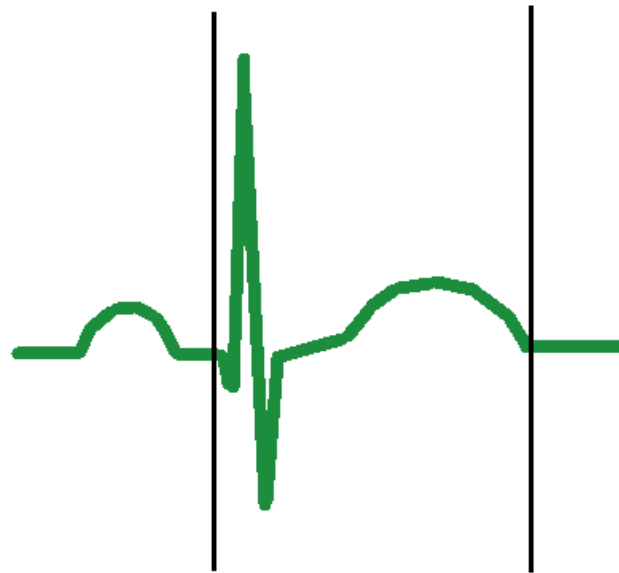
CARDIAC ANATOMY and PHYSIOLOGY "101"

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

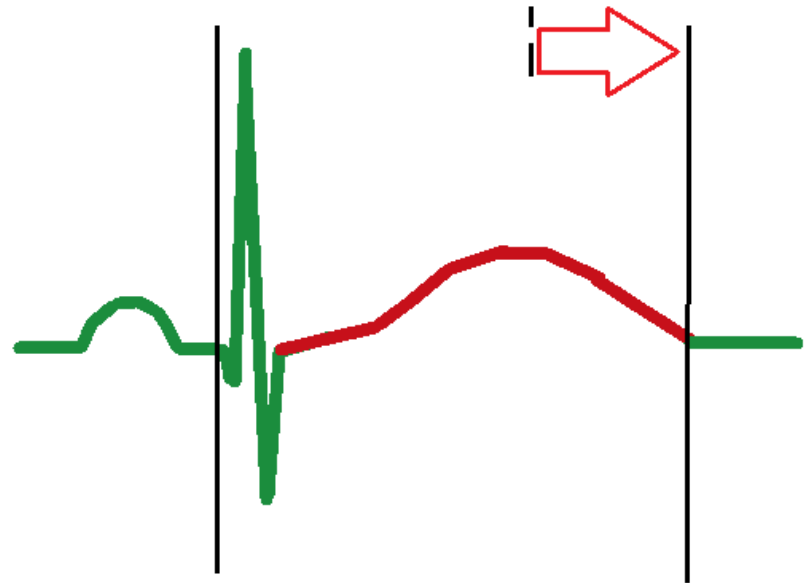
. . . . this can DELAY REPOLARIZATION, which will show on the ECG as "QT Prolongation"



Normal
QT Interval



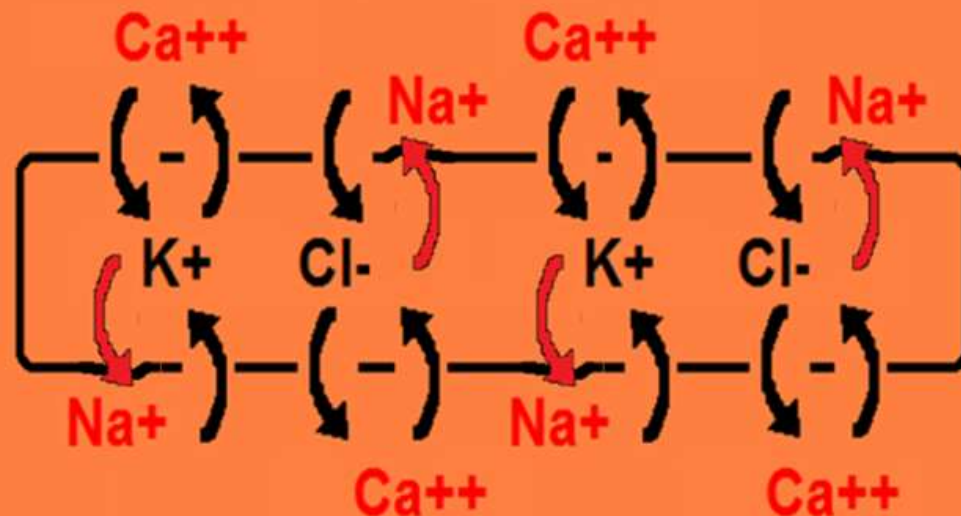
ABNORMAL
(prolonged)
QT Interval



CARDIAC ANATOMY and PHYSIOLOGY "101"

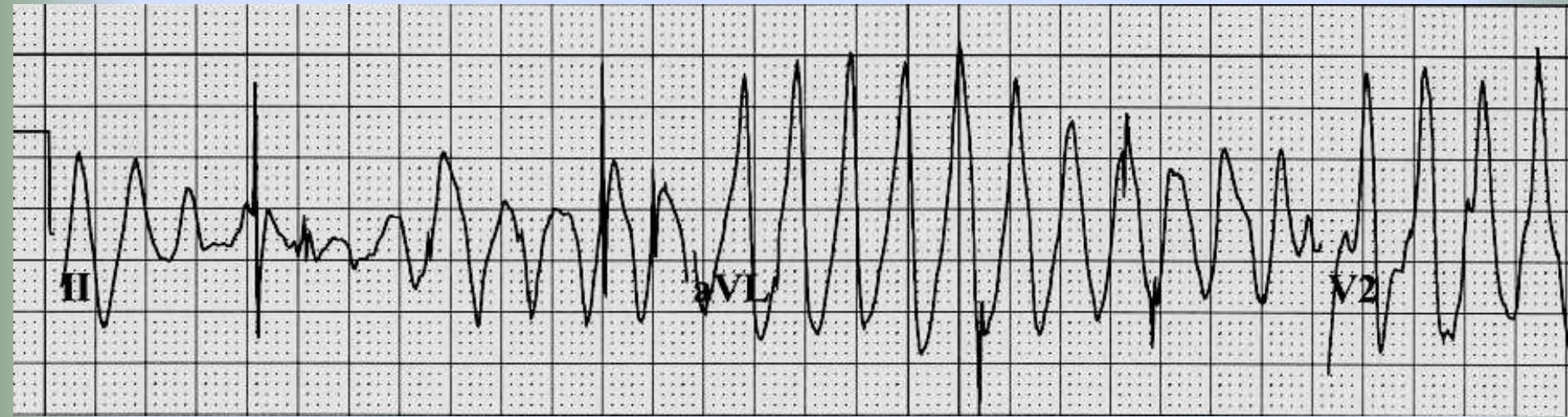
When ION CHANNELS are MALFORMED, the abnormal channel shape may DELAY the transfer of IONS

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



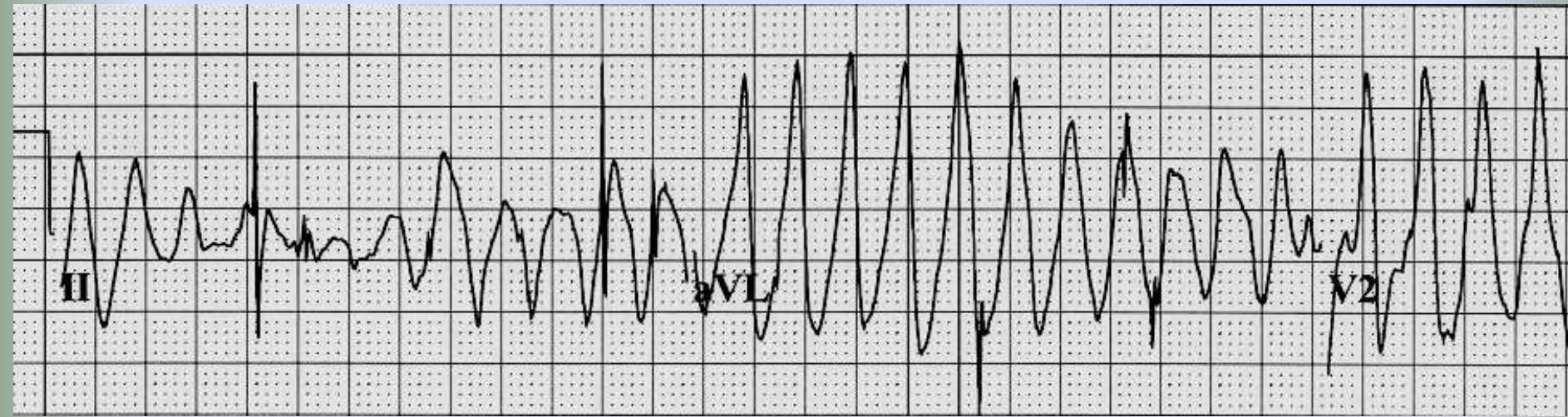
**which can lead
to Torsades ...
Cardiac
Arrest ... and
SUDDEN DEATH.**

Torsades de Pointes (TdP)



- Common cause: $QTc > 600$ ms
- Patients typically have little to no cardiac output when in this rhythm
- TdP may self-terminate or deteriorate into **VENTRICULAR FIBRILLATION**

Torsades de Pointes (TdP)

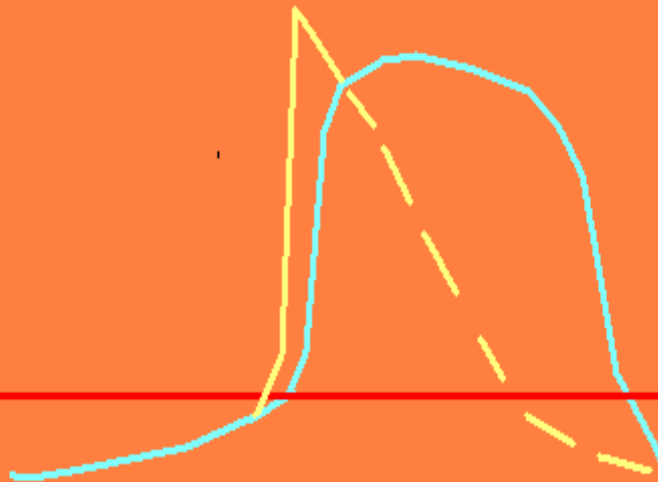


- **Common cause: $QTc > 600$ ms**
- Patients typically have little to no cardiac output when in this rhythm
- TdP may self-terminate or deteriorate into **VENTRICULAR FIBRILLATION**

CARDIAC ANATOMY and PHYSIOLOGY "101"

COMPONENTS OF ACTION POTENTIAL WAVEFORM

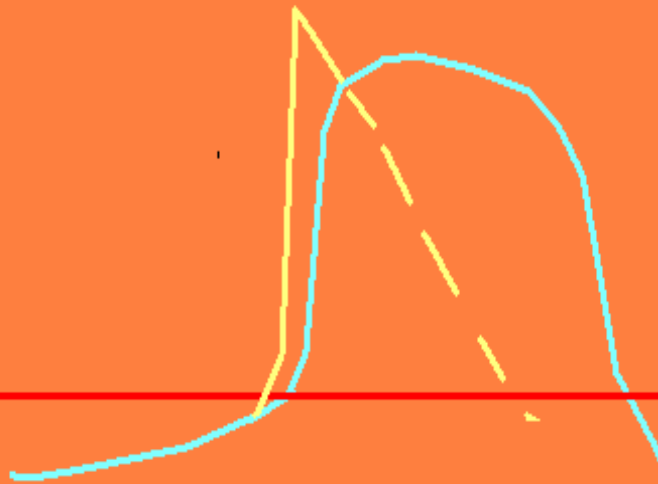
CARDIAC PACEMAKER CELLS (SINUS NODE and A-V NODE)



CARDIAC ANATOMY and PHYSIOLOGY "101"

COMPONENTS OF ACTION POTENTIAL WAVEFORM

CARDIAC PACEMAKER CELLS (SINUS NODE and A-V NODE)

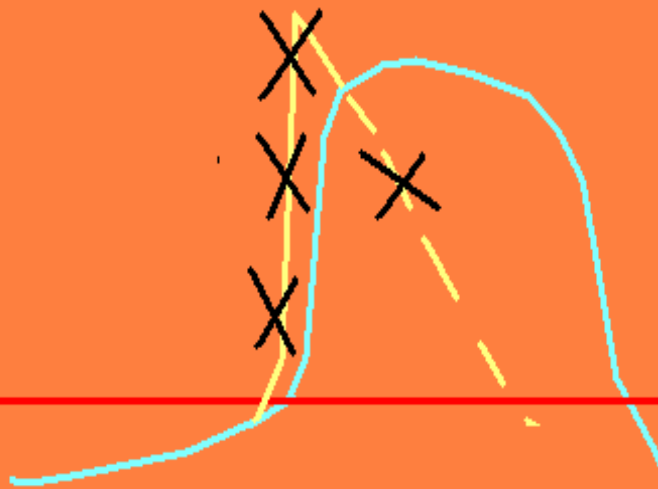


DO NOT HAVE FAST SODIUM CHANNELS...

CARDIAC ANATOMY and PHYSIOLOGY "101"

COMPONENTS OF ACTION POTENTIAL WAVEFORM

CARDIAC PACEMAKER CELLS (SINUS NODE and A-V NODE)



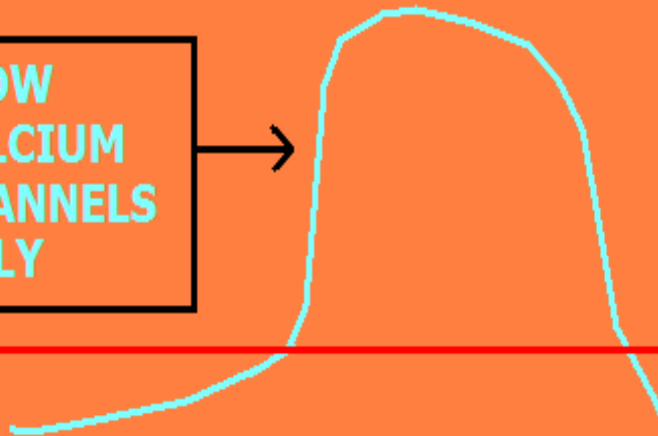
DO NOT HAVE FAST SODIUM CHANNELS...

CARDIAC ANATOMY and PHYSIOLOGY "101"

COMPONENTS OF ACTION POTENTIAL WAVEFORM

CARDIAC PACEMAKER CELLS (SINUS NODE and A-V NODE)

**SLOW
CALCIUM
CHANNELS
ONLY**

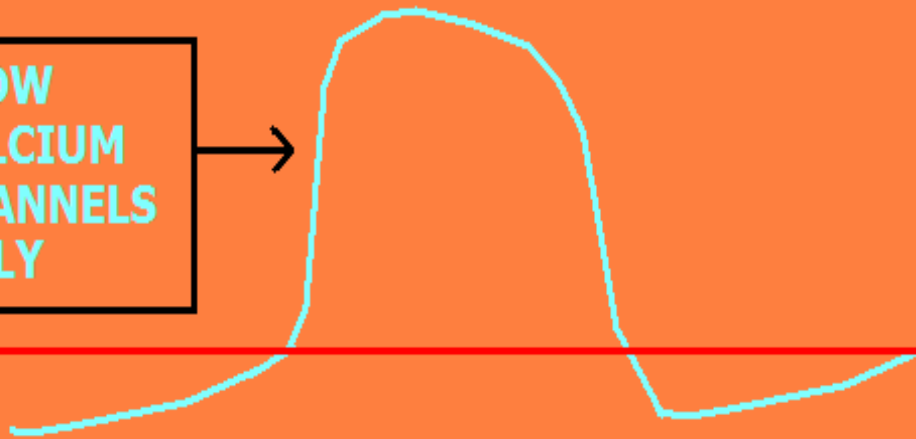


CARDIAC ANATOMY and PHYSIOLOGY "101"

COMPONENTS OF ACTION POTENTIAL WAVEFORM

CARDIAC PACEMAKER CELLS (SINUS NODE and A-V NODE)

SLOW
CALCIUM
CHANNELS
ONLY



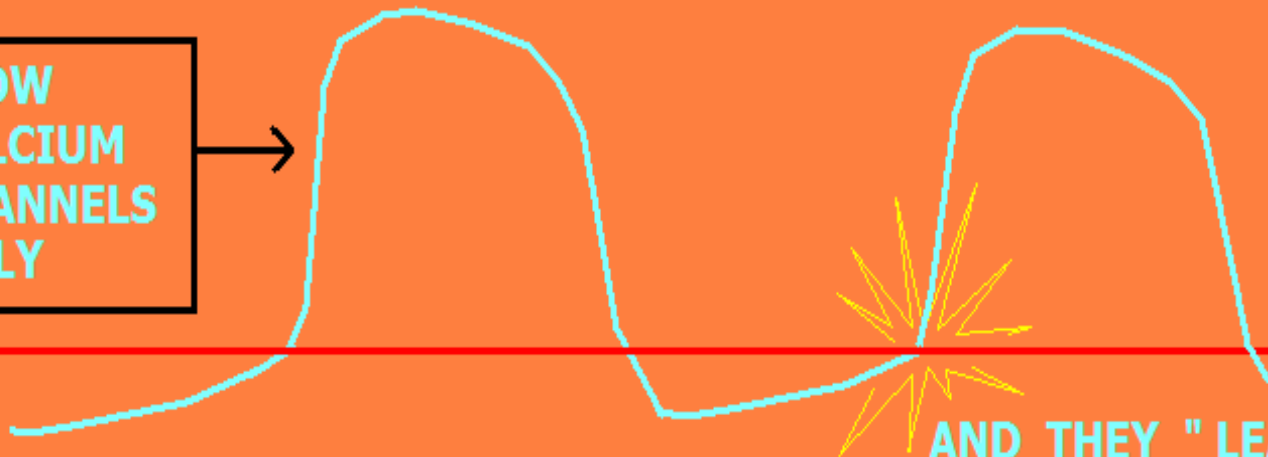
AND THEY "LEAK OUT"
IONS AT A FASTER RATE
THAN MUSCLE CELLS

CARDIAC ANATOMY and PHYSIOLOGY "101"

COMPONENTS OF ACTION POTENTIAL WAVEFORM

CARDIAC PACEMAKER CELLS (SINUS NODE and A-V NODE)

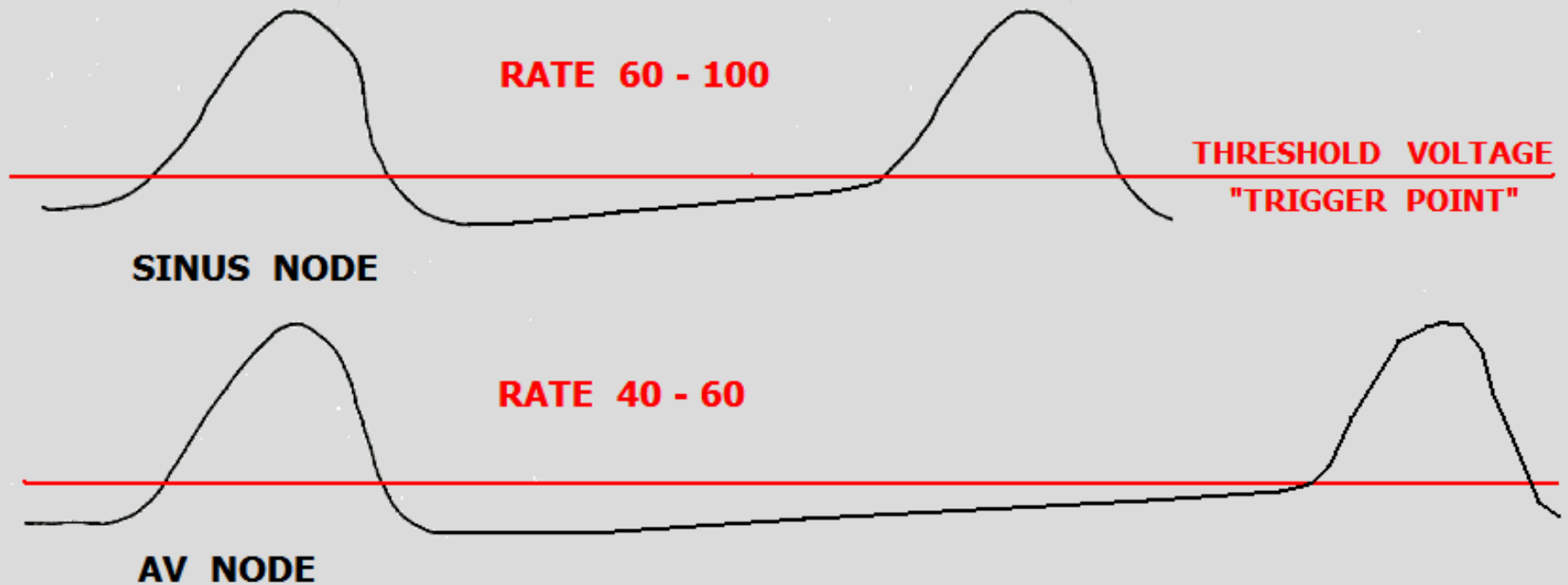
**SLOW
CALCIUM
CHANNELS
ONLY**



**AND THEY "LEAK OUT"
IONS AT A FASTER RATE
THAN MUSCLE CELLS -- FAST
ENOUGH TO HIT THE
VOLTAGE THRESHOLD and
DEPOLARIZE THEMSELVES !**

DIFFERENCES IN ACTION POTENTIAL IN DIFFERENT TYPES OF HEART CELLS

CARDIAC PACEMAKER CELLS



**DIFFERENCES IN "LEAKAGE RATES" OF IONS DURING PHASE 4
DETERMINE THE CELL'S "INHERENT FIRING RATES"**

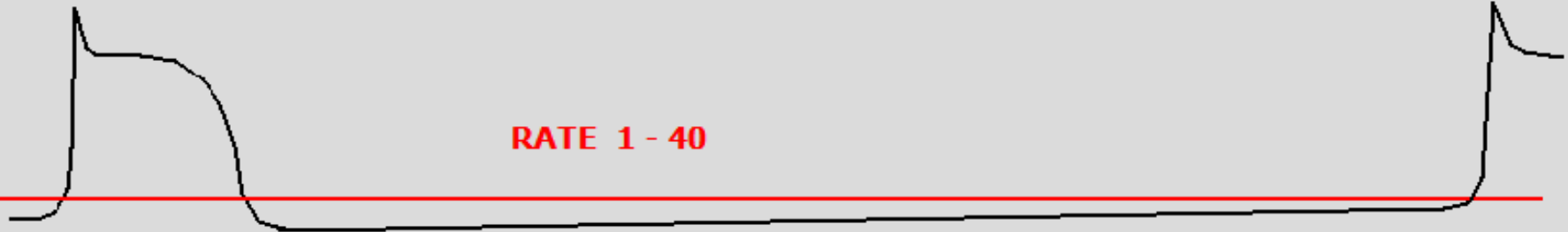
DIFFERENCES IN ACTION POTENTIAL IN DIFFERENT TYPES OF HEART CELLS

MUSCLE and PURKINJE FIBER ACTION POTENTIALS



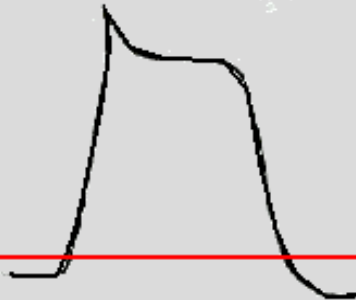
USUALLY DO NOT SELF-DEPOLARIZE UNLESS
ABNORMAL CHANGE IN AUTOMATICITY

ATRIAL MUSCLE



RATE 1 - 40

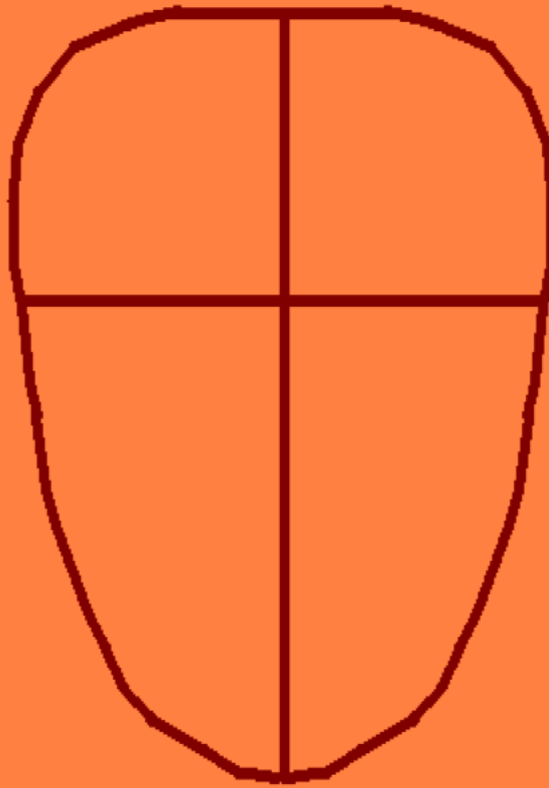
PURKINJE FIBER



USUALLY DO NOT SELF-DEPOLARIZE UNLESS
ABNORMAL CHANGE IN AUTOMATICITY

VENTRICULAR MUSCLE

FOUR CHAMBERED PUMP



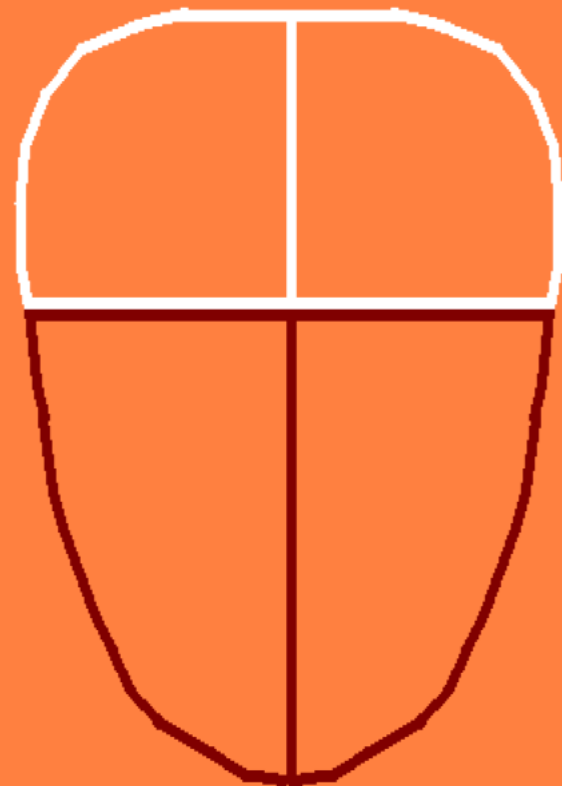
FOUR CHAMBERED PUMP . . .

2 ATRIUM



PRIMARY JOB:

**"PACK VENTRICLES
FULL OF BLOOD"**

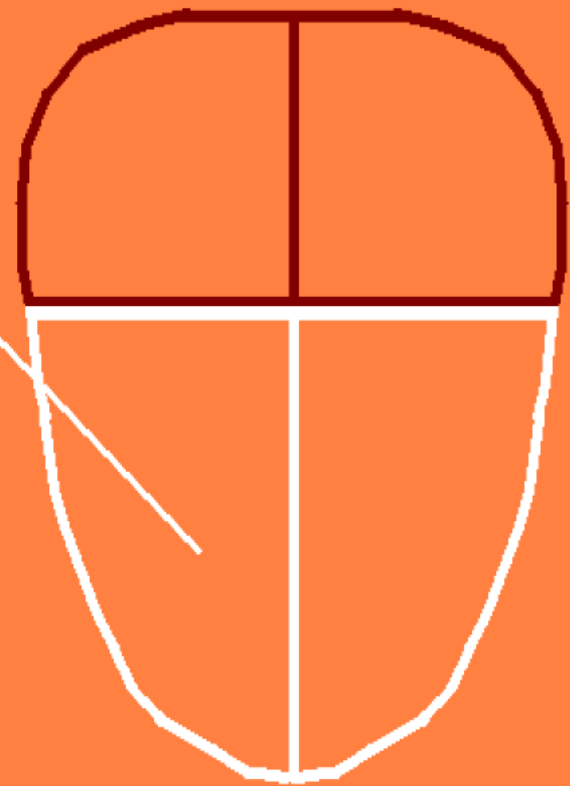


FOUR CHAMBERED PUMP . . .

2 VENTRICLES

PRIMARY JOB:

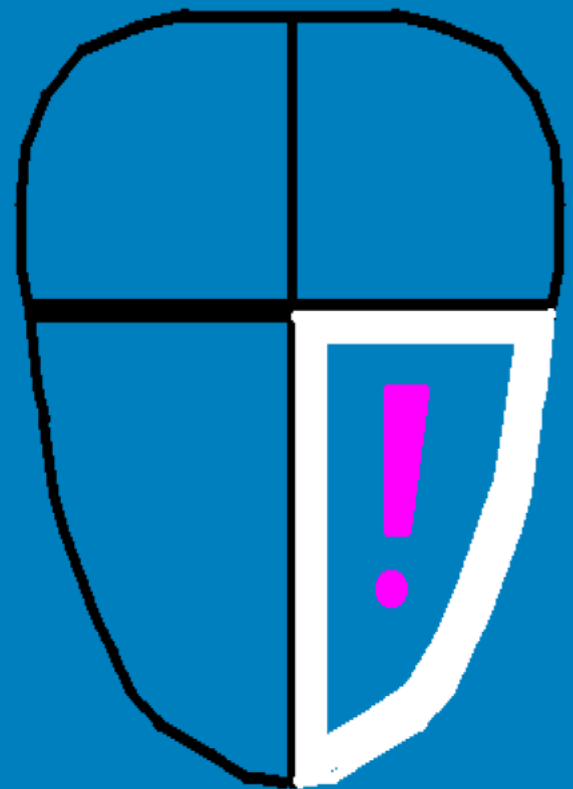
"PUMP BLOOD TO THE
LUNGS AND THE
REST OF THE BODY"



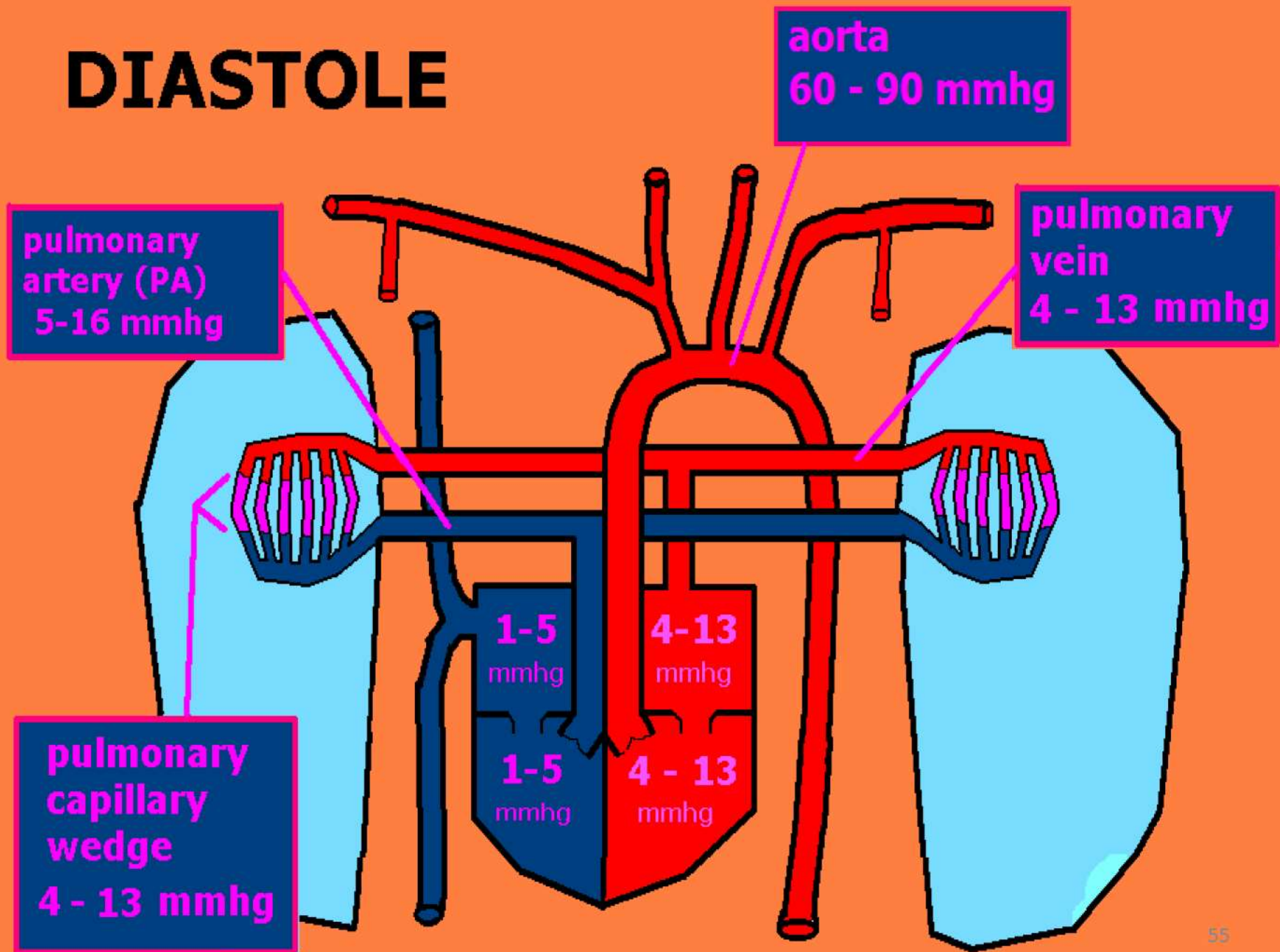
THE CHAMBER MOST IMPORTANT
TO KEEPING THE PATIENT ALIVE

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

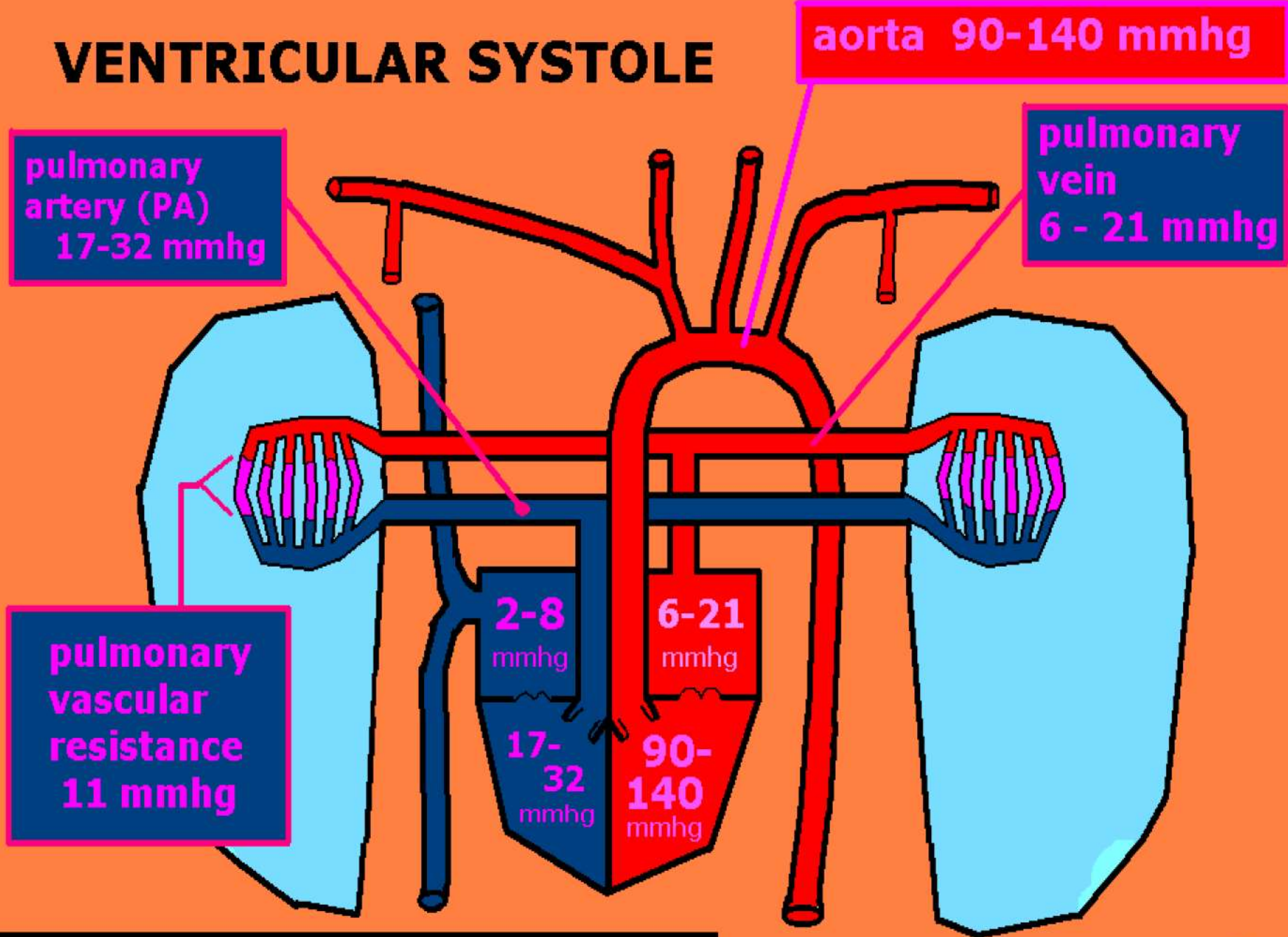
IS THE
LEFT VENTRICLE
WHICH WE WILL REFER
TO AS THE PUMP



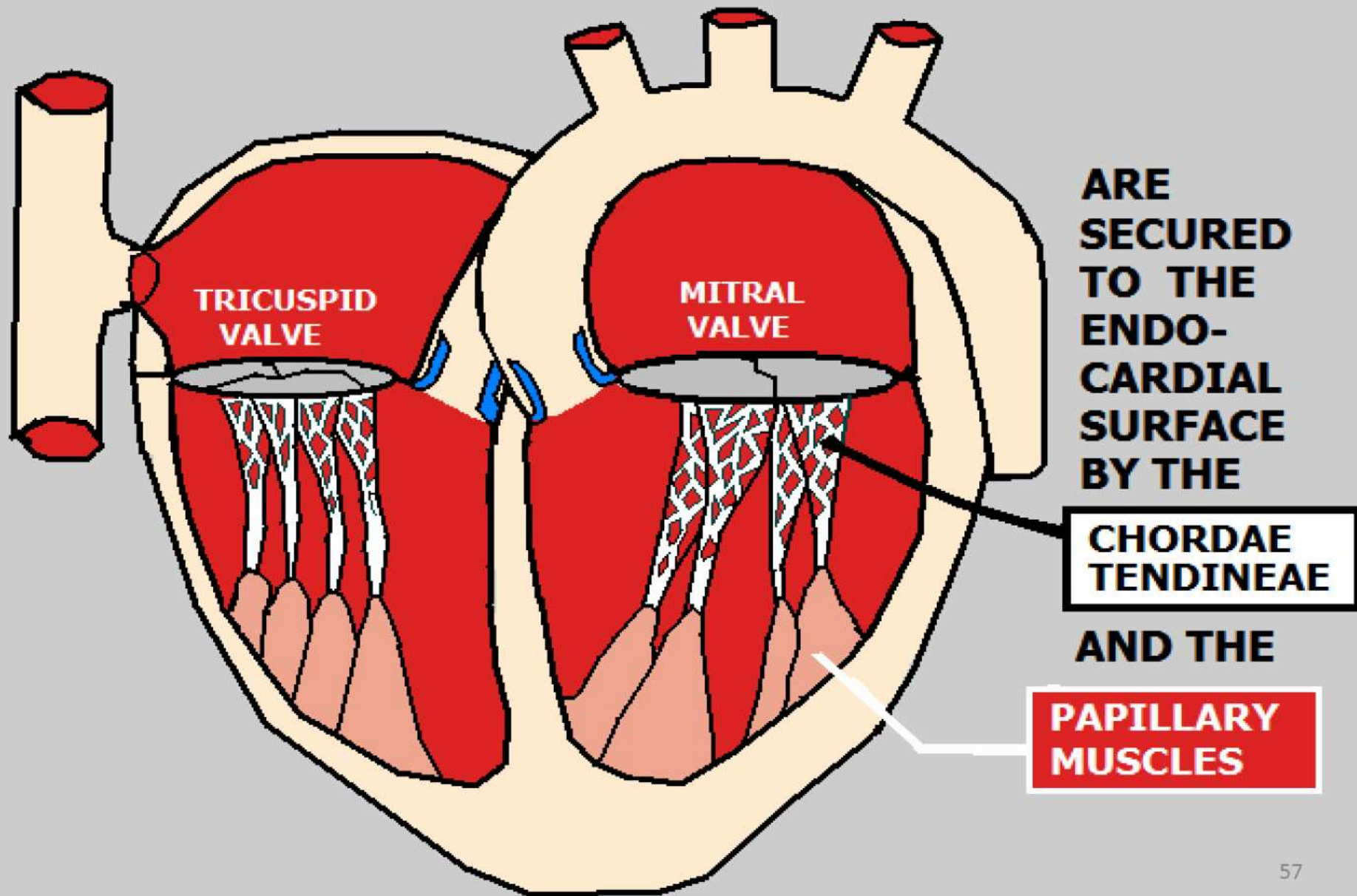
DIASTOLE



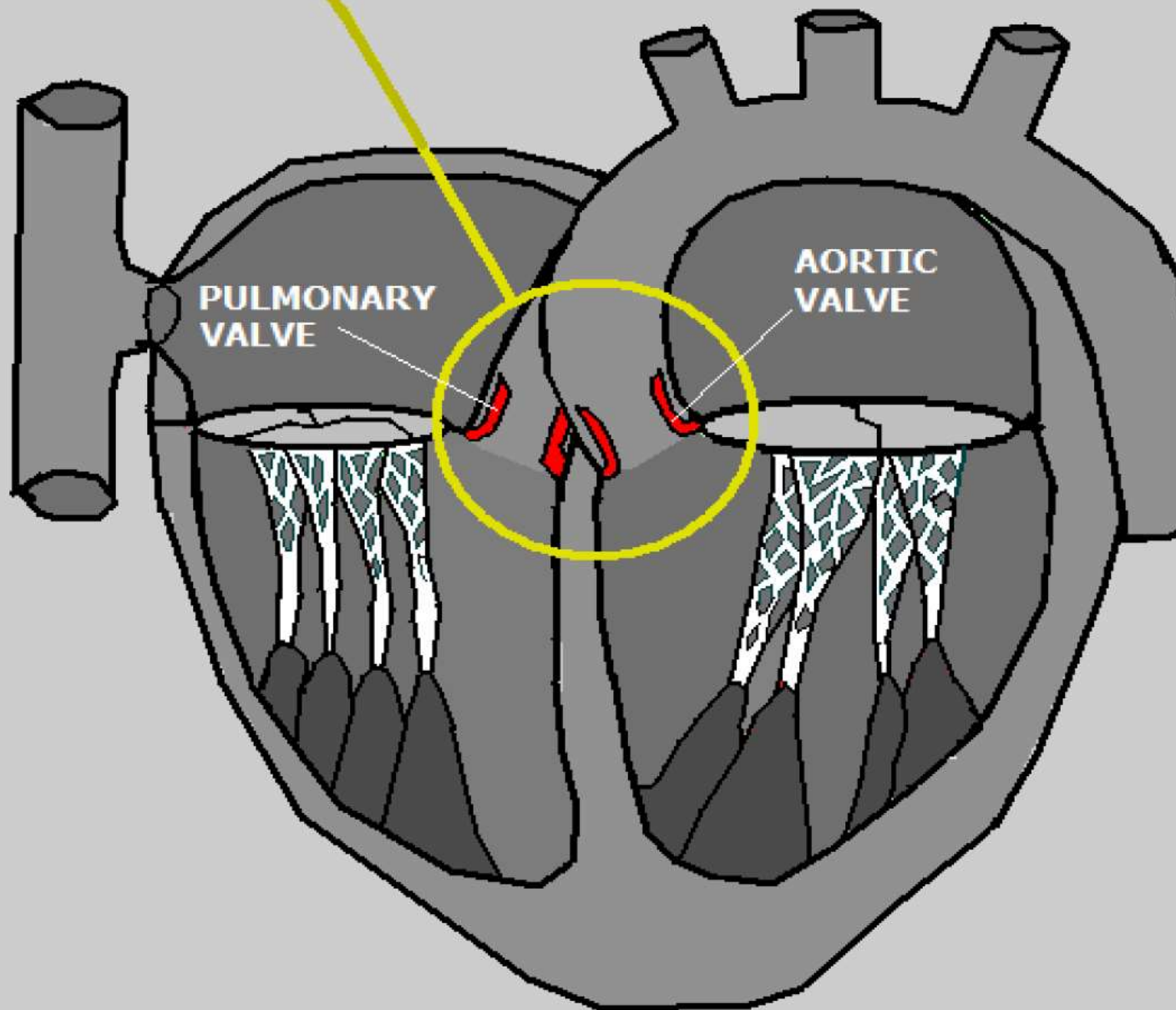
VENTRICULAR SYSTOLE



ATRIO-VENTRICULAR VALVES



THE SEMILUNAR VALVES



VERY

BASIC HEART SOUNDS ASSESSMENT

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

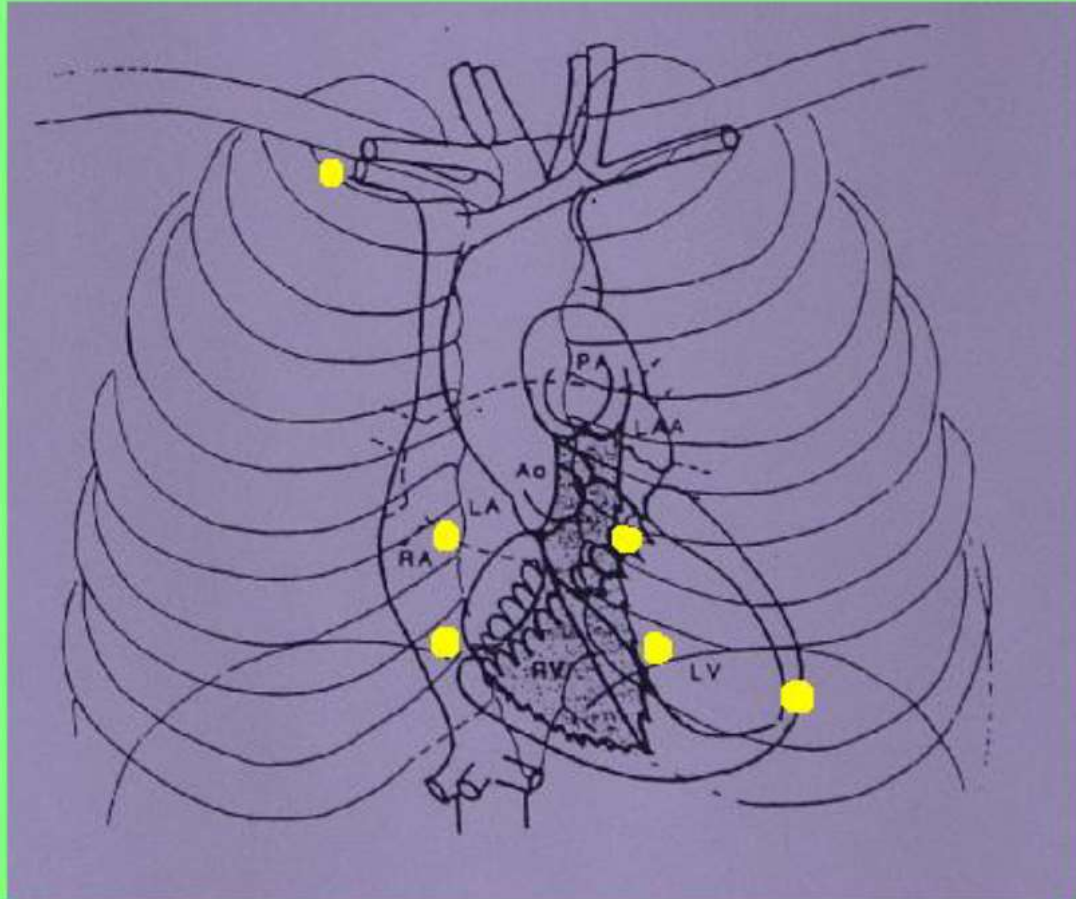
- ACUTE MI
- CHAMBER HYPERTROPHY
- RECENT MI (NECROSIS)
- PERICARDITIS



HEART SOUNDS ASSESSMENT



HEART SOUNDS ASSESSMENT



VERY BASIC HEART SOUNDS ASSESSMENT

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



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

HEART SOUNDS ASSESSMENT

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

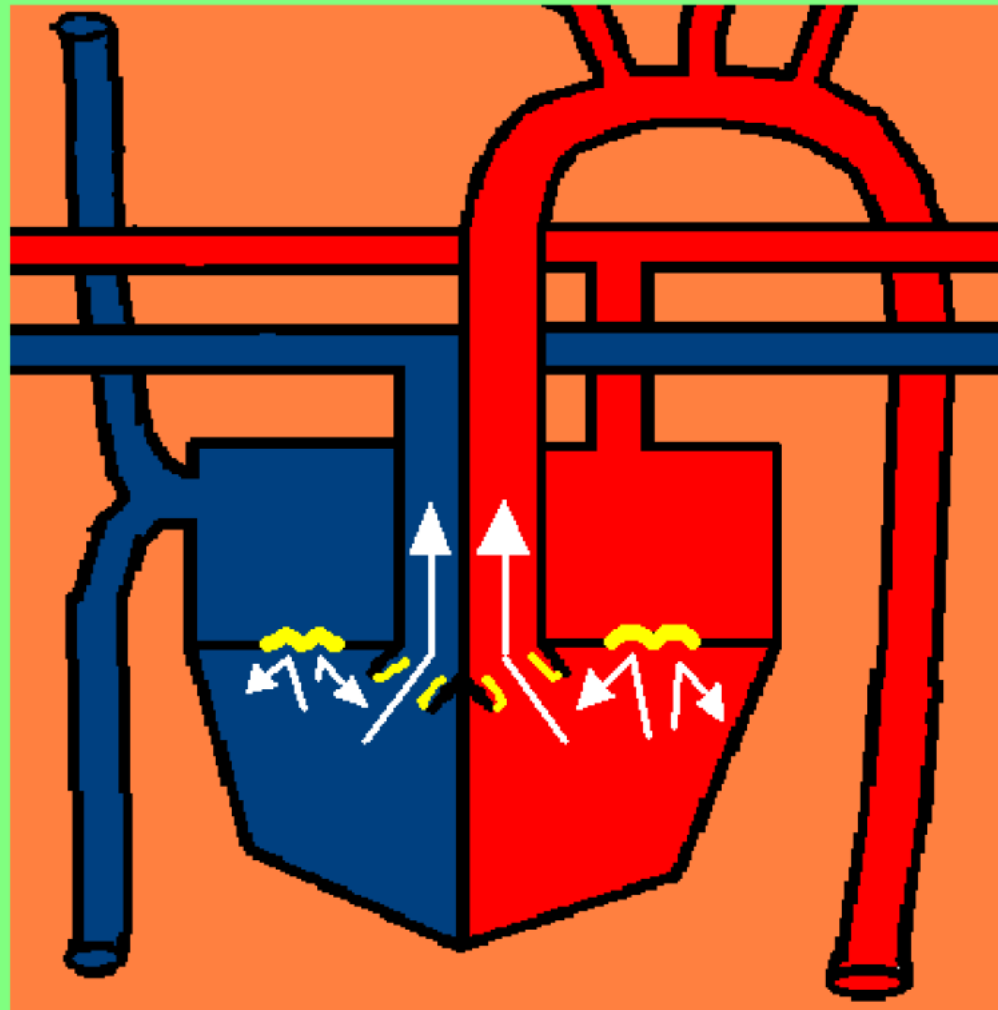
THERE ARE TWO NORMAL HEART SOUNDS,
KNOWN AS: S-1 and S-2

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

HEART SOUNDS ASSESSMENT

S-1
BEGINNING
OF
SYSTOLE.

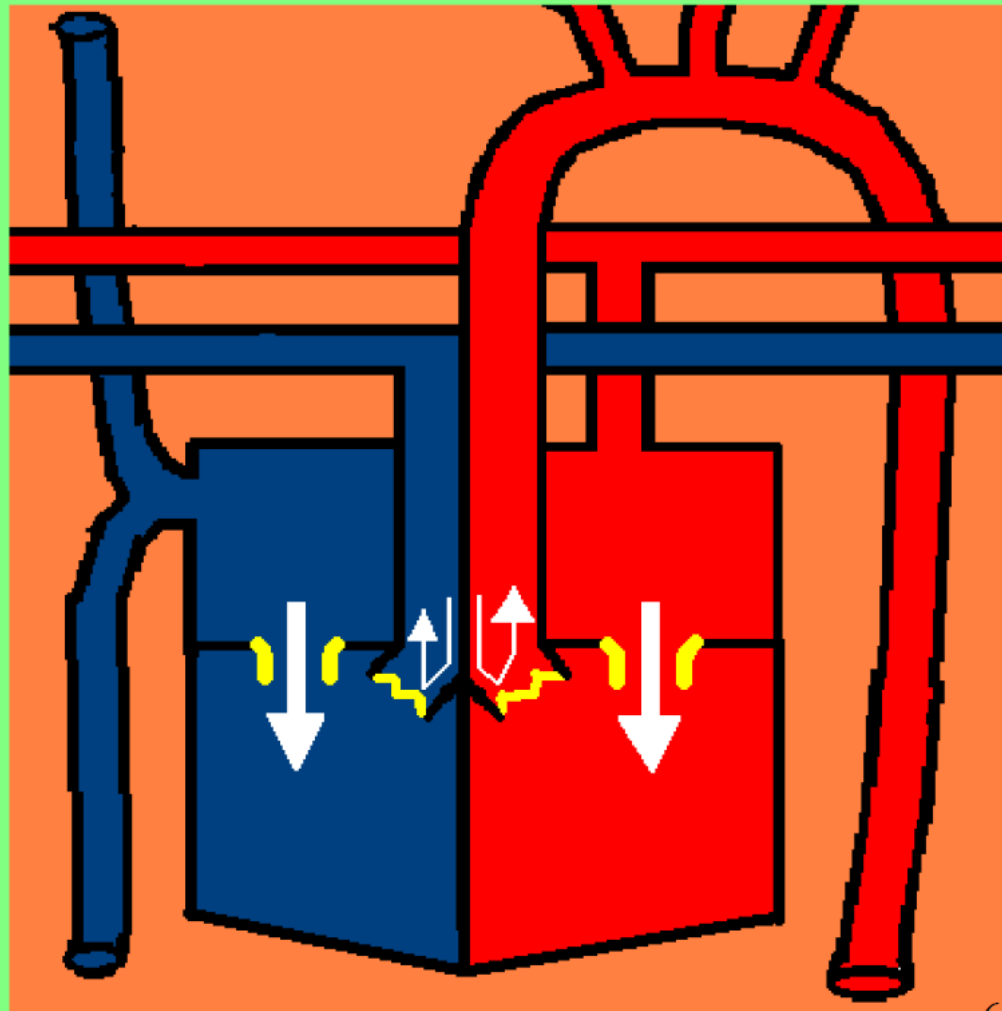
SOUND OF
THE
MITRAL
AND
TRICUSPID
VALVES
CLOSING.



HEART SOUNDS ASSESSMENT

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

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



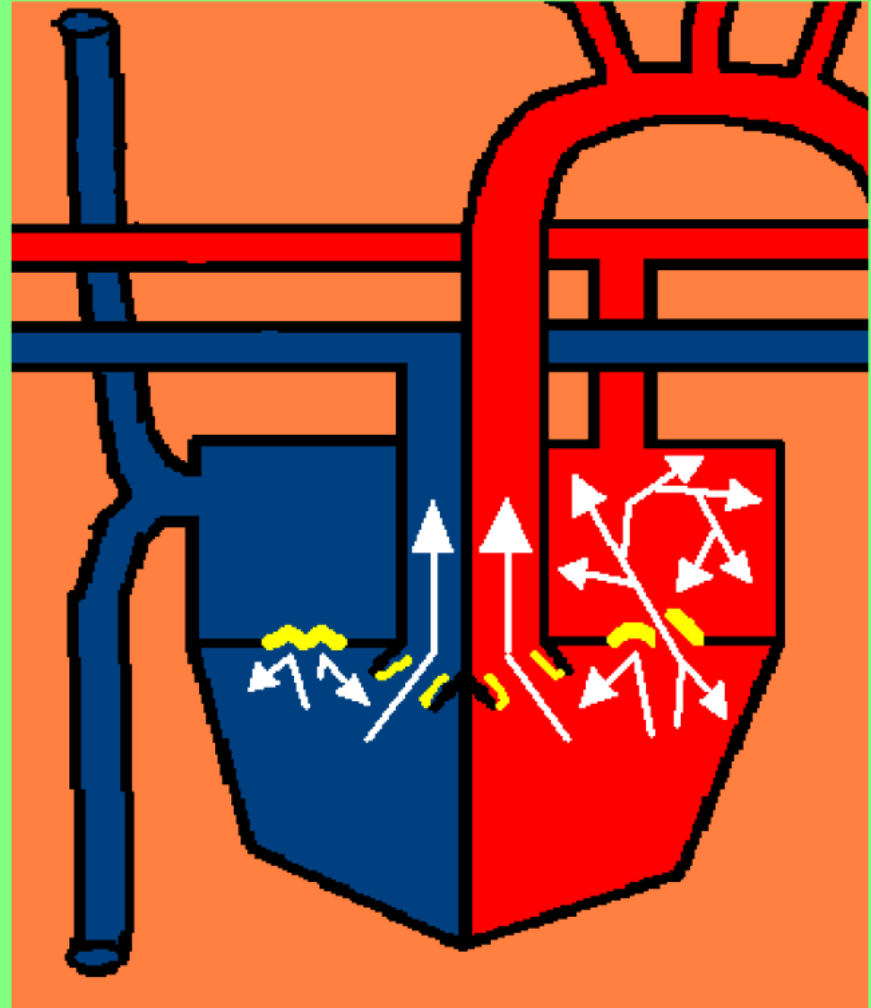
VERY

BASIC HEART SOUNDS ASSESSMENT

ABNORMAL SOUND	SUSPECTED EKG CHANGES
MURMURS - SYSTOLIC - DIASTOLIC	<ul style="list-style-type: none">- ACUTE MI- CHAMBER HYPERTROPHY- NECROSIS - RECENT EXTENSIVE MI (7-10 days)
FRICTION RUB	<ul style="list-style-type: none">- ACUTE MI- RECENT MI (NECROSIS)- PERICARDITIS

CAUSE OF SYSTOLIC (S 1) MURMUR

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



BASIC HEART SOUNDS ASSESSMENT

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

S-1 MURMUR SOUNDS LIKE:

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



**❑ MOST SYSTOLIC MURMURS
CAUSED BY MITRAL VALVE
FAILURE.**

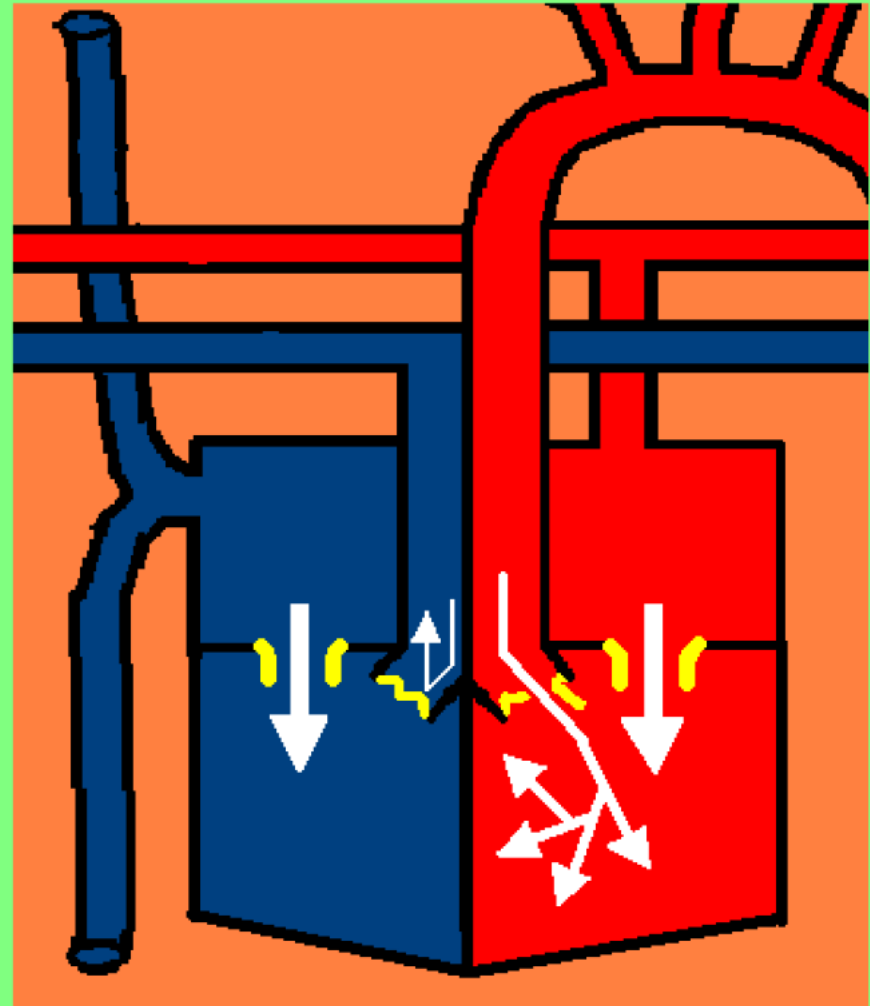


**ACUTE MITRAL VALVE
REGURGITATION IS A
POTENTIALLY LETHAL
COMPLICATION OF
ACUTE / RECENT
EXTENSIVE TRANSMURAL MI**

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

CAUSE OF DIASTOLIC (S_2) MURMUR

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



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

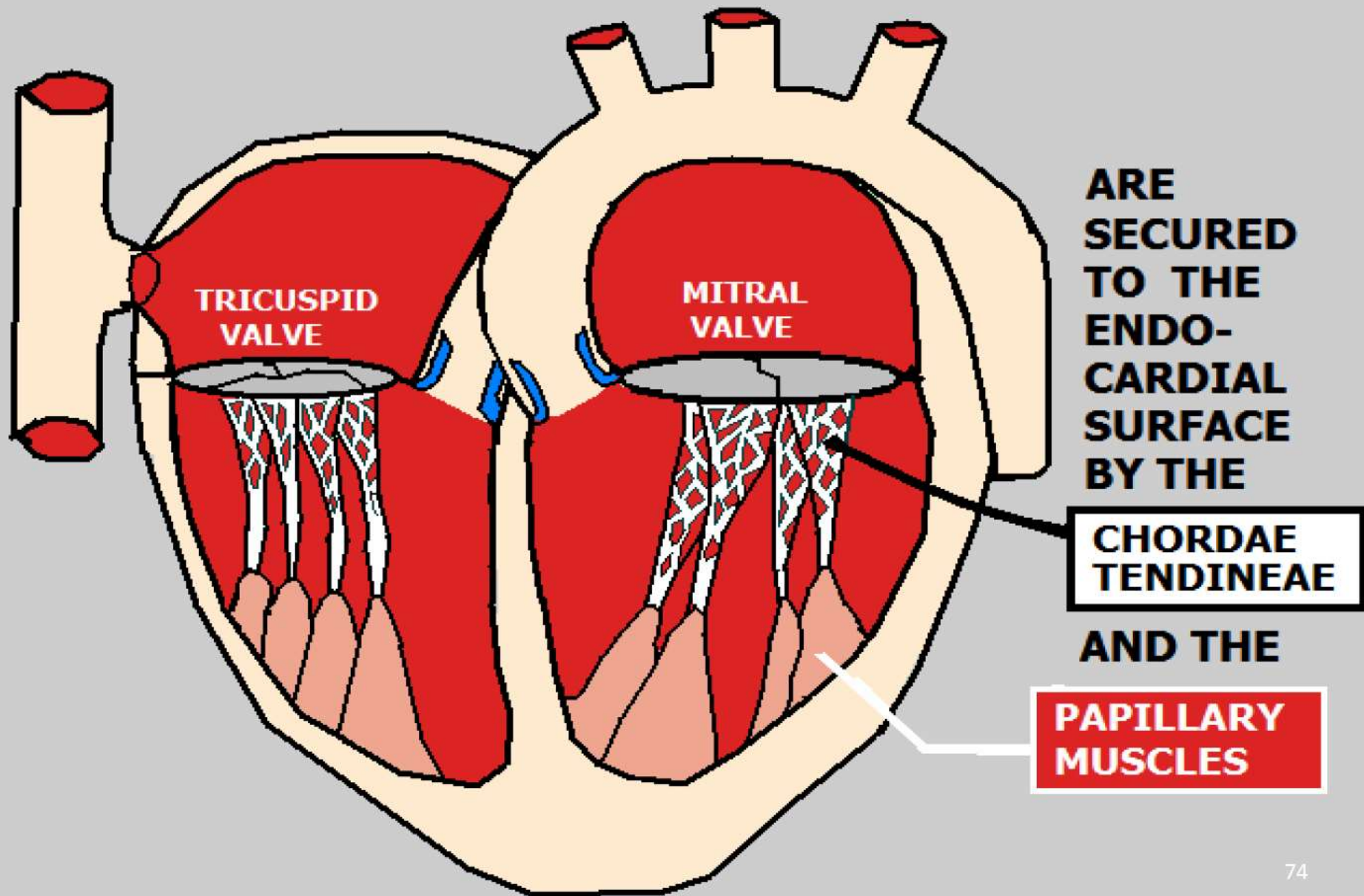
Chronic **Valvular STENOSIS** (“Creaky”
Valve) leads to Cardiac Muscle STRAIN
and HYPERTROPHY.

BOTH conditions, if untreated,
eventually leads to **HEART FAILURE.**

**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.**

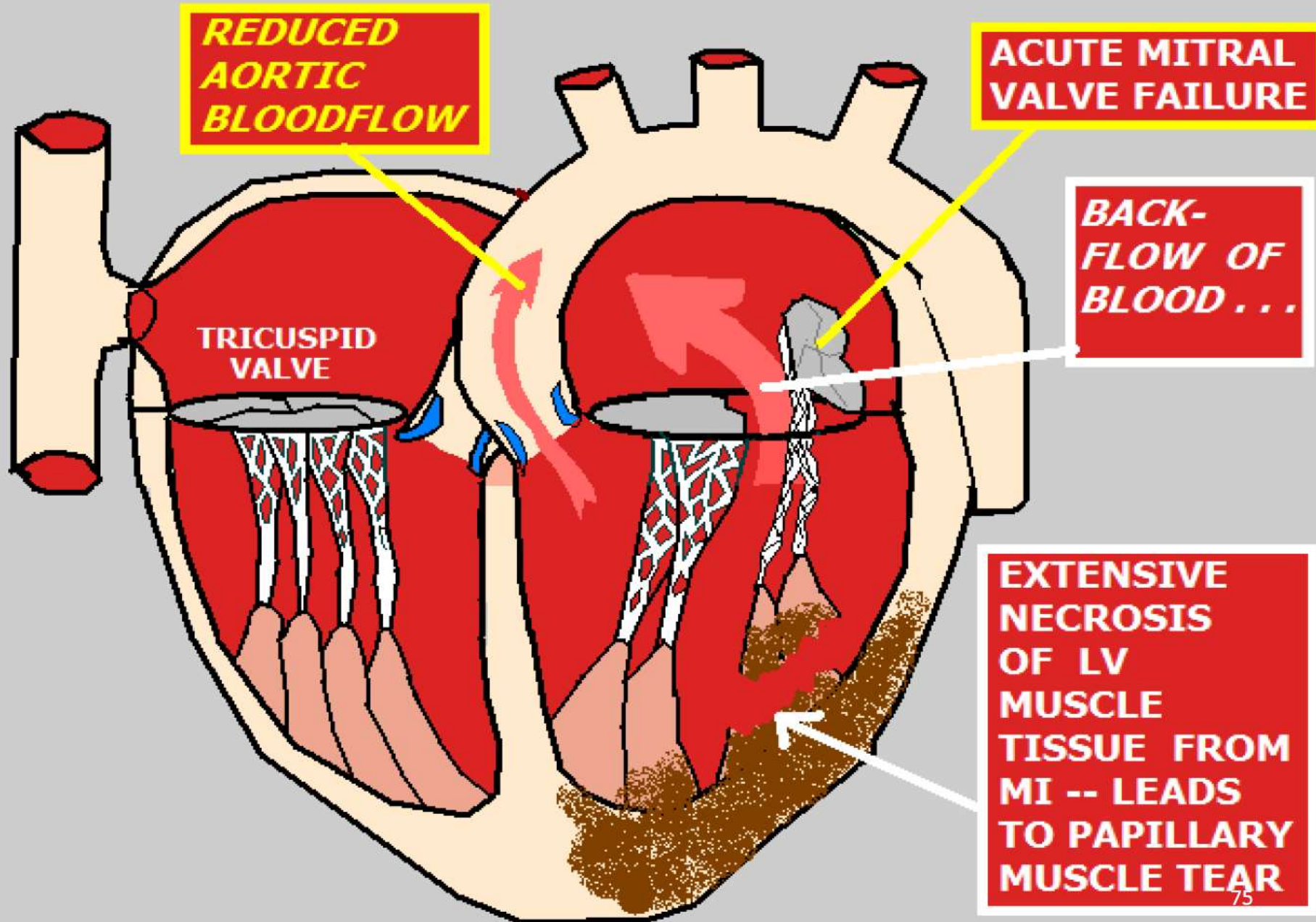
**Papillary muscles are attached to
“multiple surfaces”**

ATRIO-VENTRICULAR VALVES



ACUTE MITRAL REGURGITATION

DURING VENTRICULAR SYSTOLE



Symptoms of Acute Mitral Regurgitation

- SHOCK
- PROFOUND HYPOTENSION
- 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 . . . "**

Heart Sounds: S₃

- S₃ sounds like: “kenTUCky . . . kenTUCky”
- Caused by: increased atrial pressure.
- S₃ is associated with: **Heart Failure, Dilated Cardiomyopathy.**

Heart Sounds: S4

- S4 sounds like: “TENnessee. . . TENnessee”
- Caused by: stiffened left ventricle.
- S4 is associated with: **Hypertension, Aortic Stenosis, Ischemic or Hypertrophic Cardiomyopathy.**

Access
University of Washington
Department of Medicine

Heart Sound Simulator

BASIC HEART SOUNDS ASSESSMENT

FRICTION RUB

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



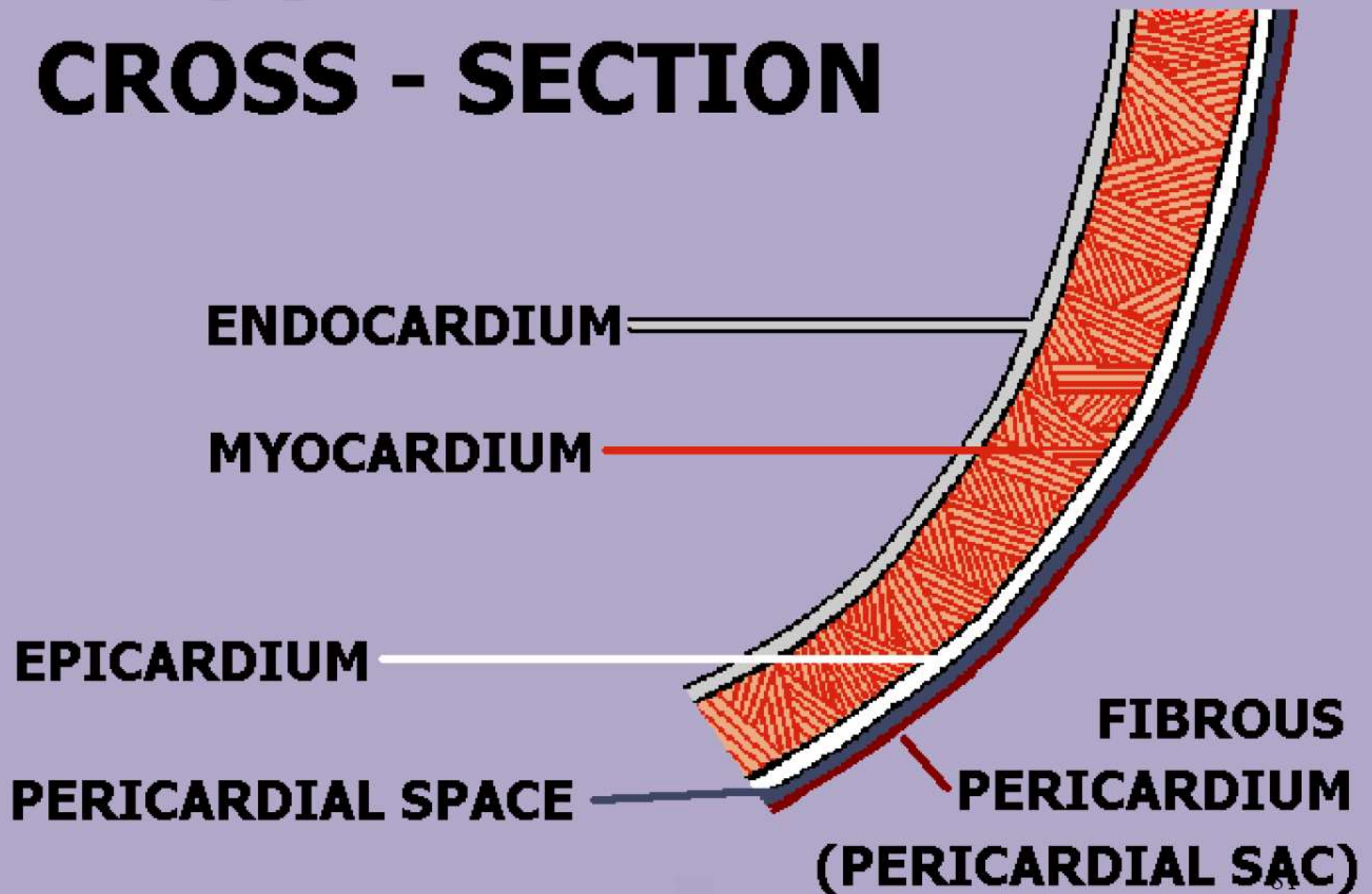
BASIC HEART SOUNDS ASSESSMENT

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



MYOCARDIAL CROSS - SECTION



**NORMAL AMOUNT OF
FLUID IN
PERICARDIAL SPACE =
20 - 50 cc**

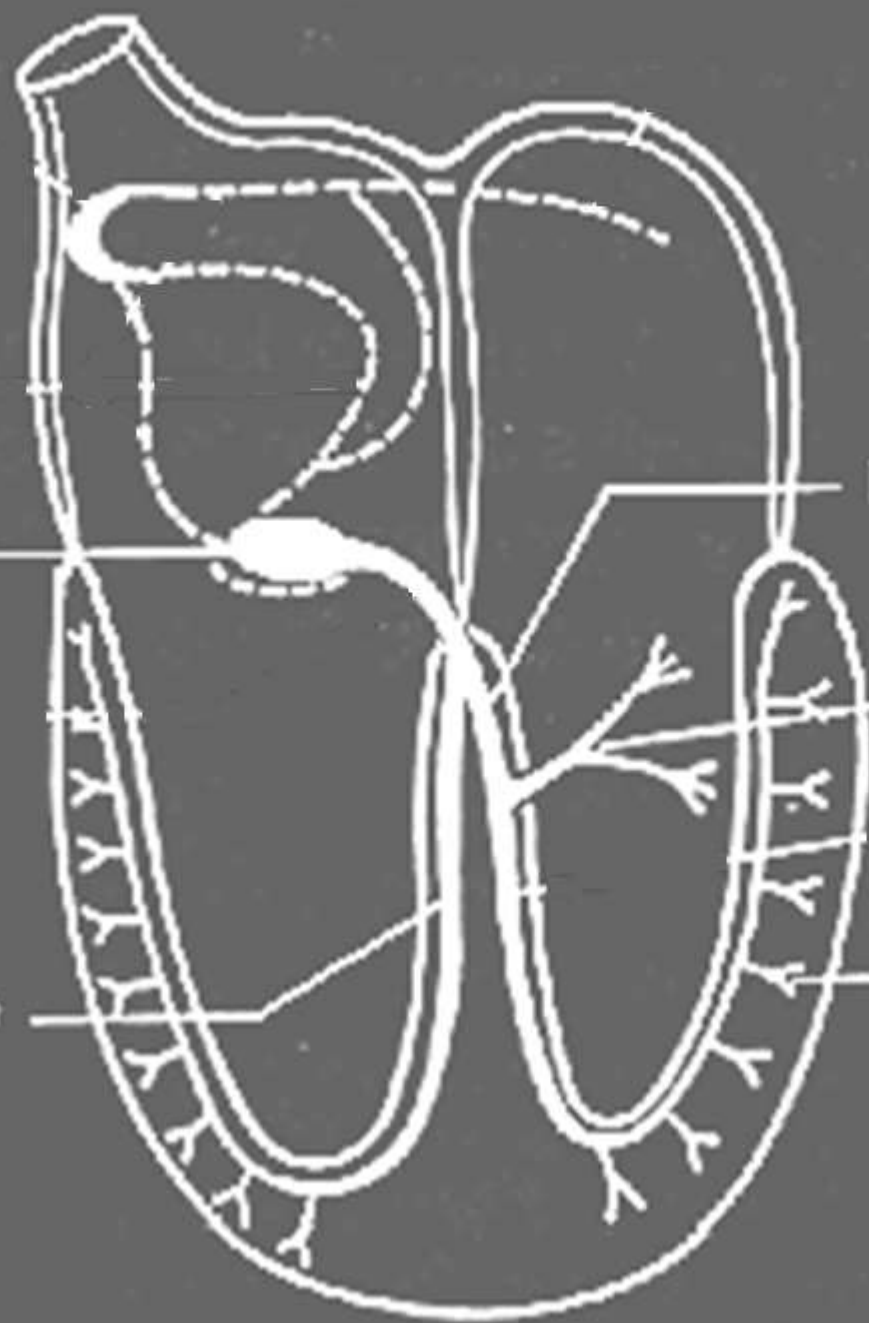
Sinus node

AV node

Right bundle
branch

Left bundle
branch

Purkinje fibers



Sinus node

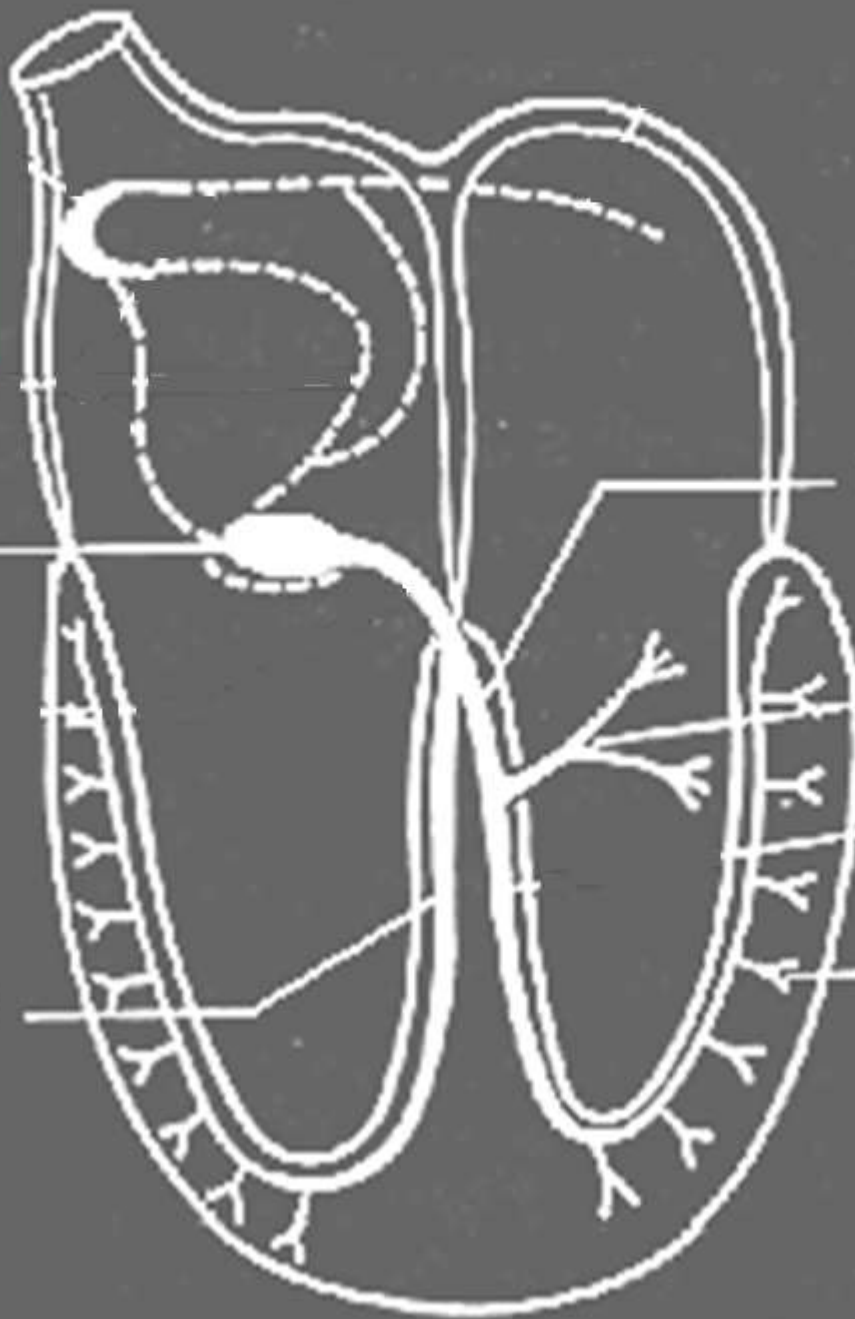
60 - 100
beats / min.

AV node

Left bundle
branch

Right bundle
branch

Purkinje fibers



~~Sinus node~~

AV node

40 - 60
beats / min.

Right bundle
branch

Left bundle
branch

Purkinje fibers



~~Sinus node~~

~~AV node~~

Left bundle
branch

Right bundle
branch

Purkinje fibers

Pacemaker site in the
Ventricles:
20 - 40 beats / min



ATRIAL DEPOLARIZATION

— P WAVE
IS RECORDED
ON EKG

LEAD II

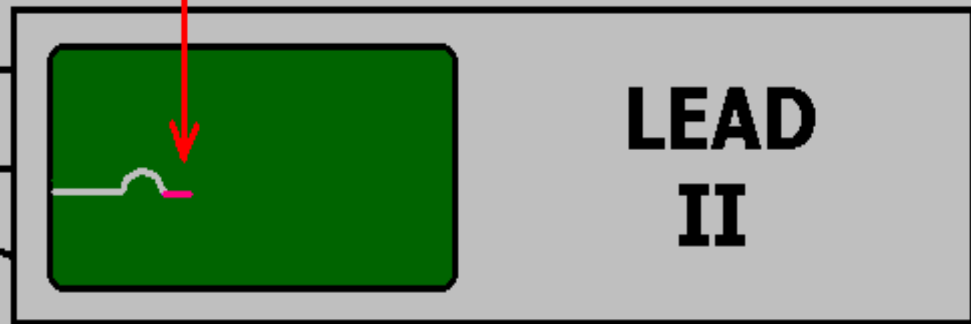
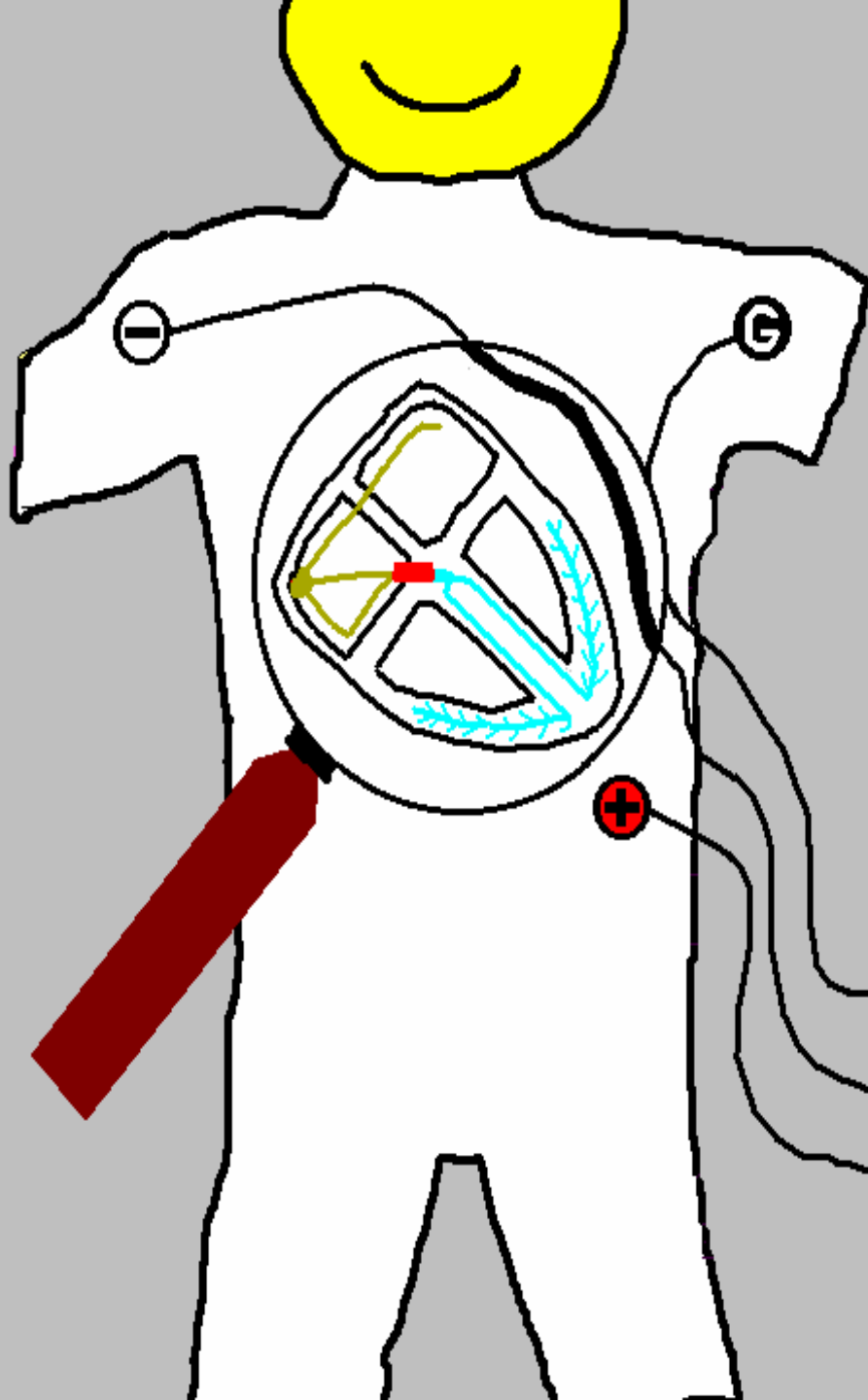
THE P-R SEGMENT

ELECTRICAL ACTIVITY
DURING P-R SEGMENT:

- Depolarization wave in A-V node
- Atrial Repolarization

.10 SECOND
ISOELECTRIC PAUSE

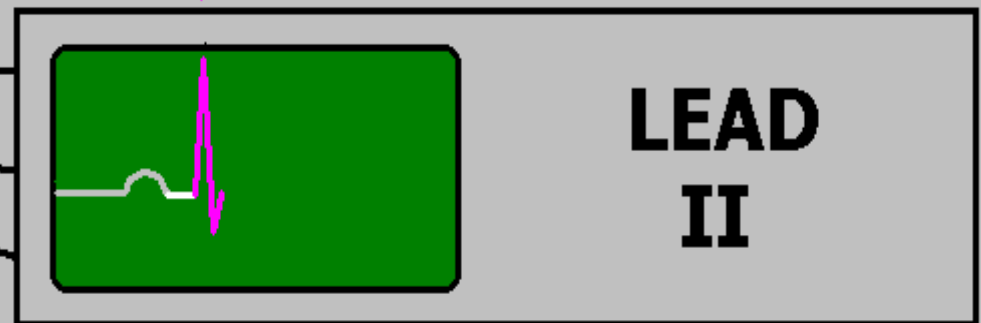
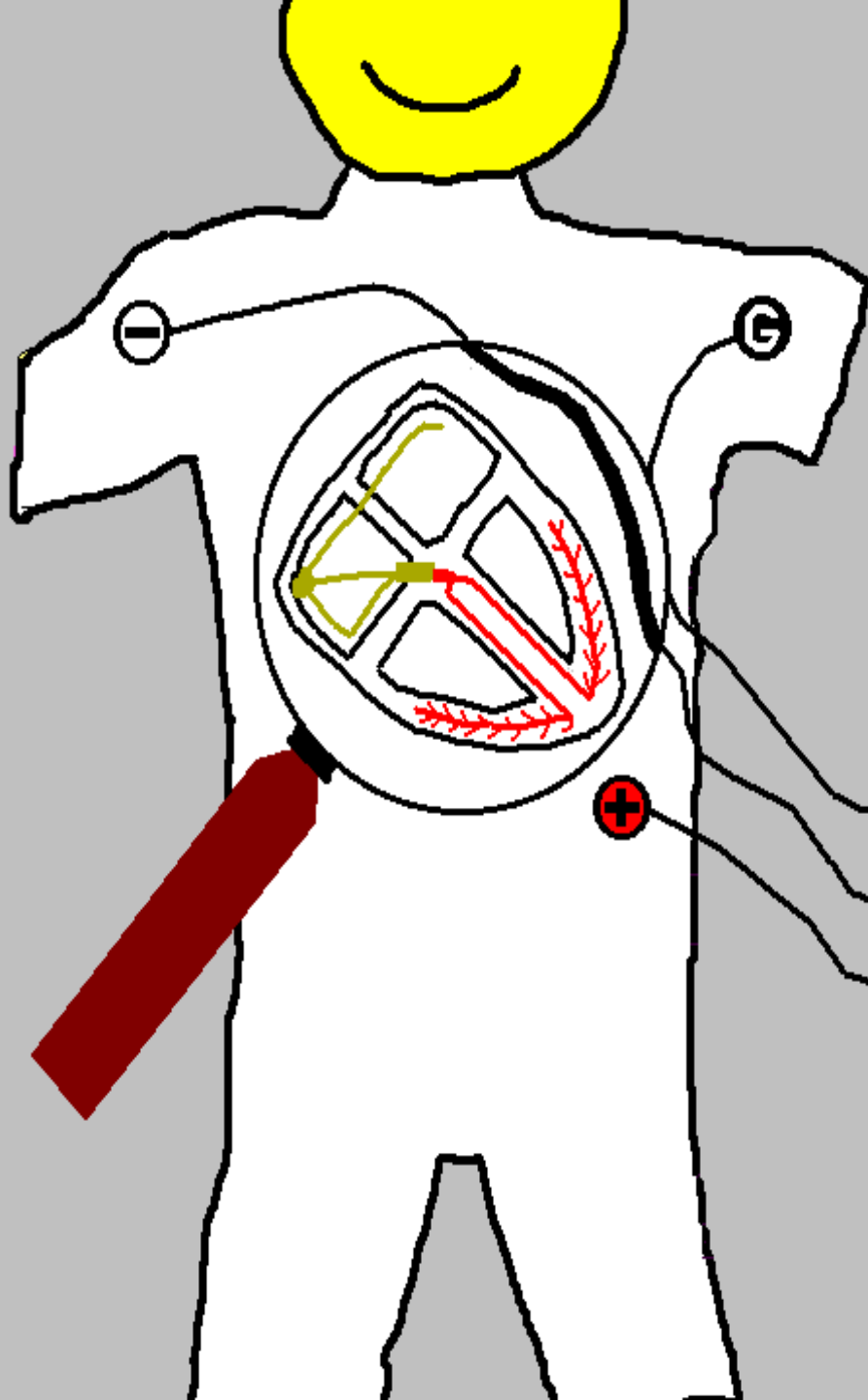
**LEAD
II**



VENTRICULAR DEPOLARIZATION

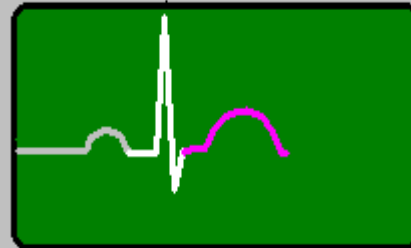
QRS COMPLEX
IS RECORDED
ON EKG

LEAD
II



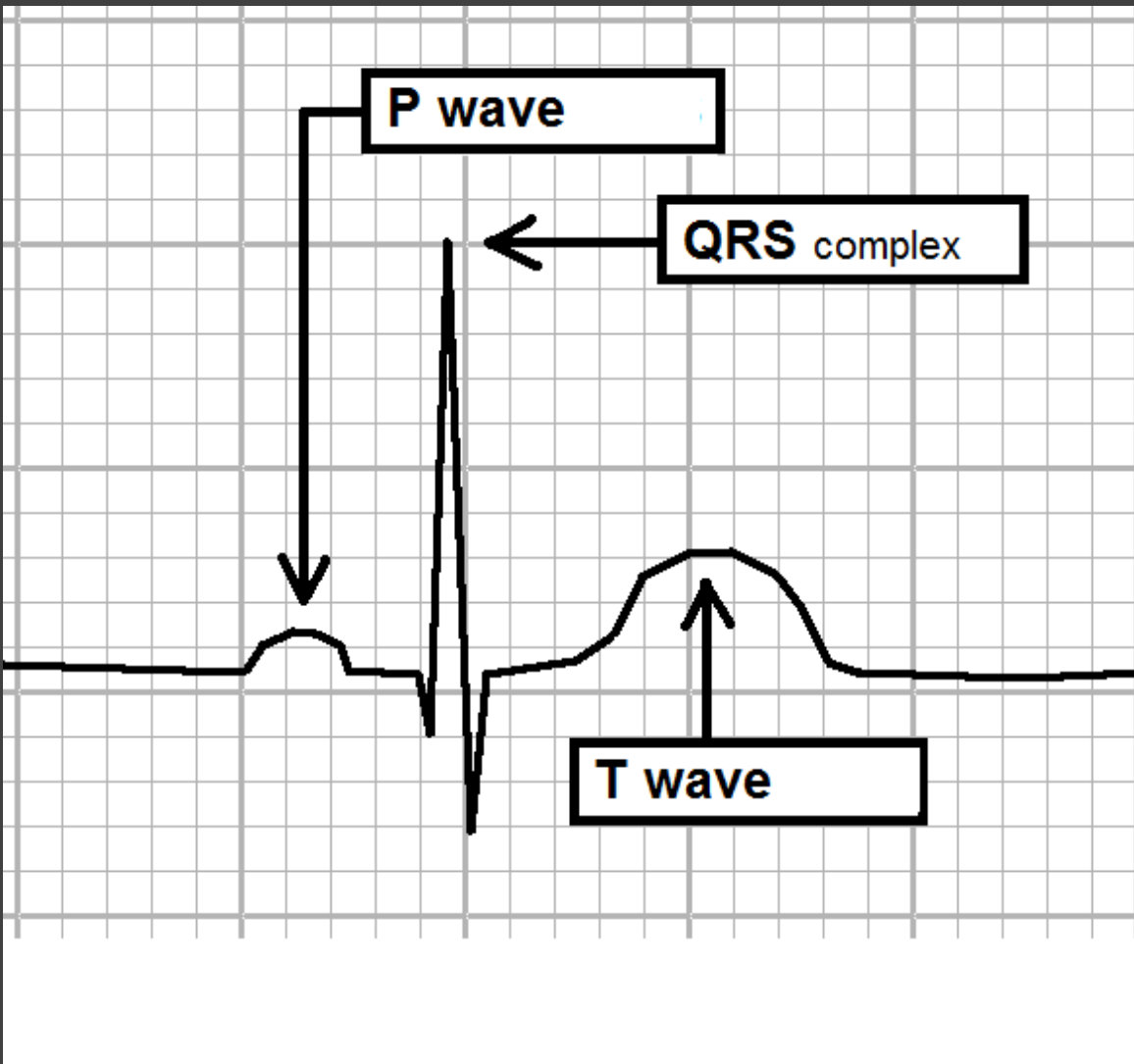
VENTRICULAR REPOLARIZATION

WRITES A "T"
WAVE ON THE
ECG



**LEAD
II**

REVIEW of NORMAL ECG Waveforms:



P WAVE =
ATRIAL DEPOLARIZATION

QRS COMPLEX =
VENTRICULAR
DEPOLARIZATION
(contracting)

T WAVE =
VENTRICULAR
REPOLARIZATION
(recharging)



OLD BARN, SHREWSBURY, PA — 2001

EMS 12 Lead ECG



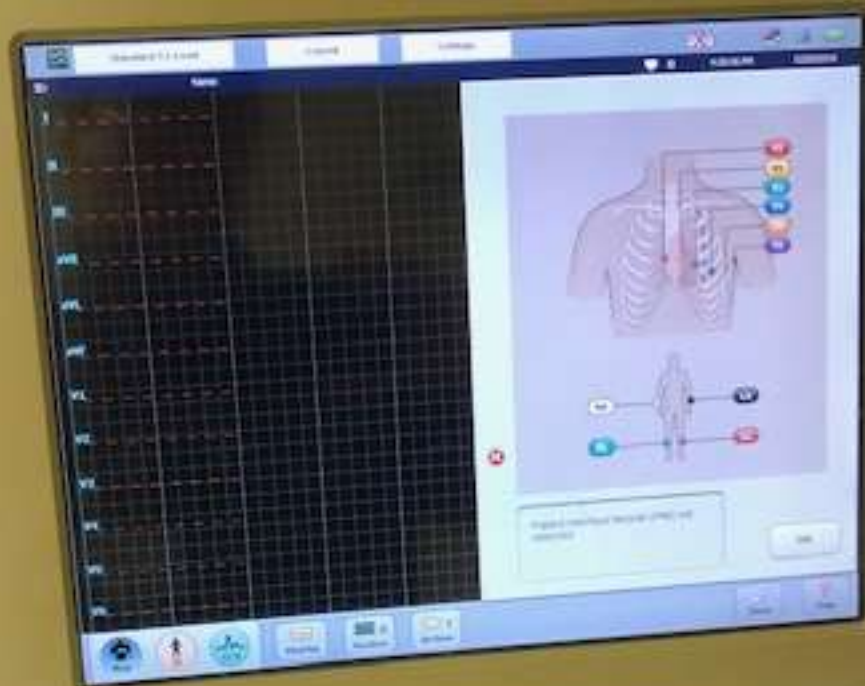
10 wires . . .

- 4 limb leads
- 6 chest (“V”) leads





PHILIPS



IT IS IMPORTANT TO
ensure that the ECG leads are
attached to the patient's chest
before all other monitoring equipment
is attached to the patient.
When ECG is completed, remove the
leads from the patient.

If you don't do all of the above
steps, the ECG will NOT attach
to the patient's CHART in Carevue
and the doctor won't see it!

Philips TC2



OBTAINING THE 12 LEAD ECG

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

Obtaining the 12 Lead ECG

- Limb leads should be on the limbs.

Obtaining the 12 Lead ECG

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

Obtaining the 12 Lead ECG

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

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

AHA/ACC/HRS Scientific Statement

Recommendations for the Standardization and Interpretation of the Electrocardiogram

Part I: The Electrocardiogram and Its Technology

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

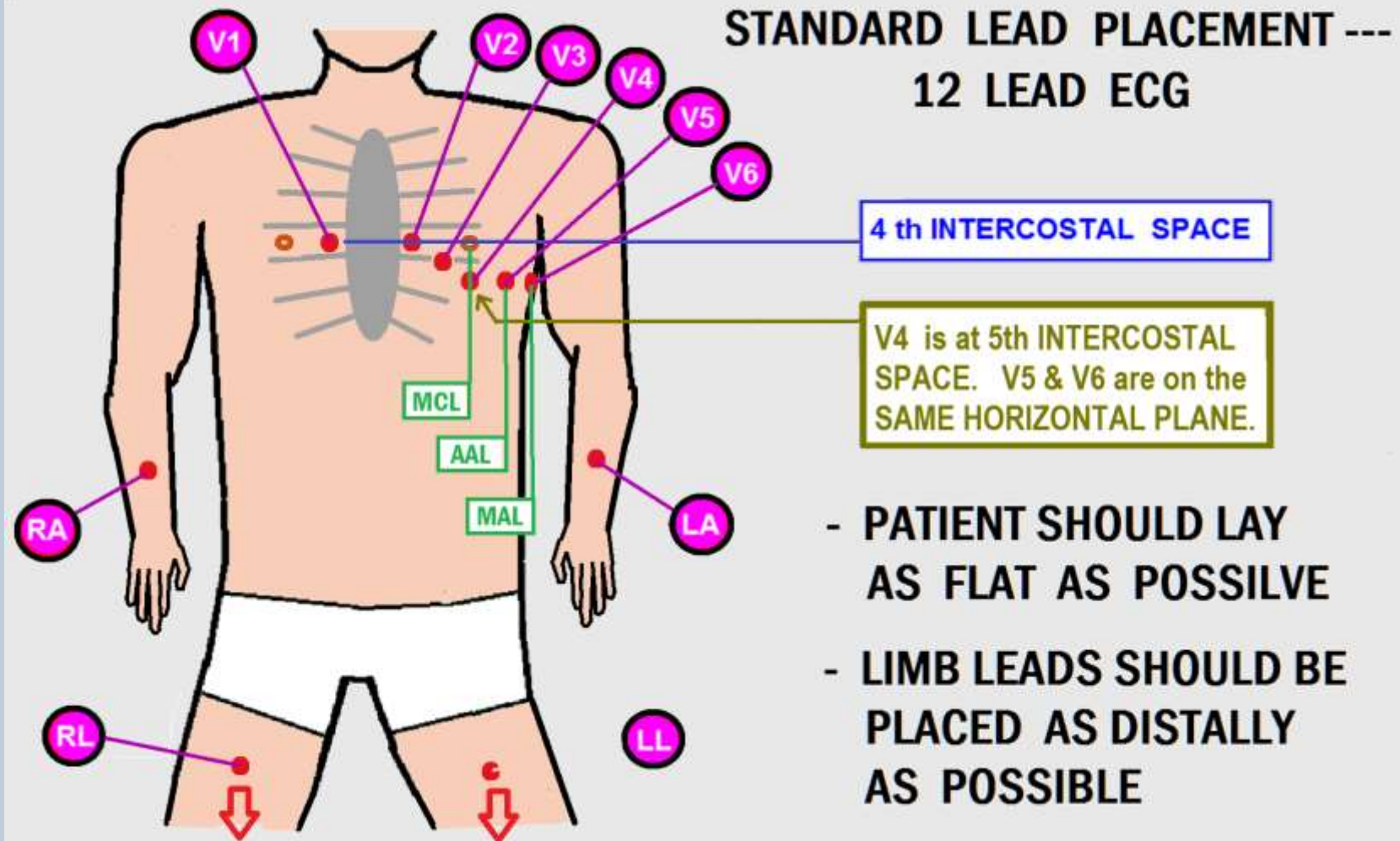
AHA/ACC/HRS Scientific Statement

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

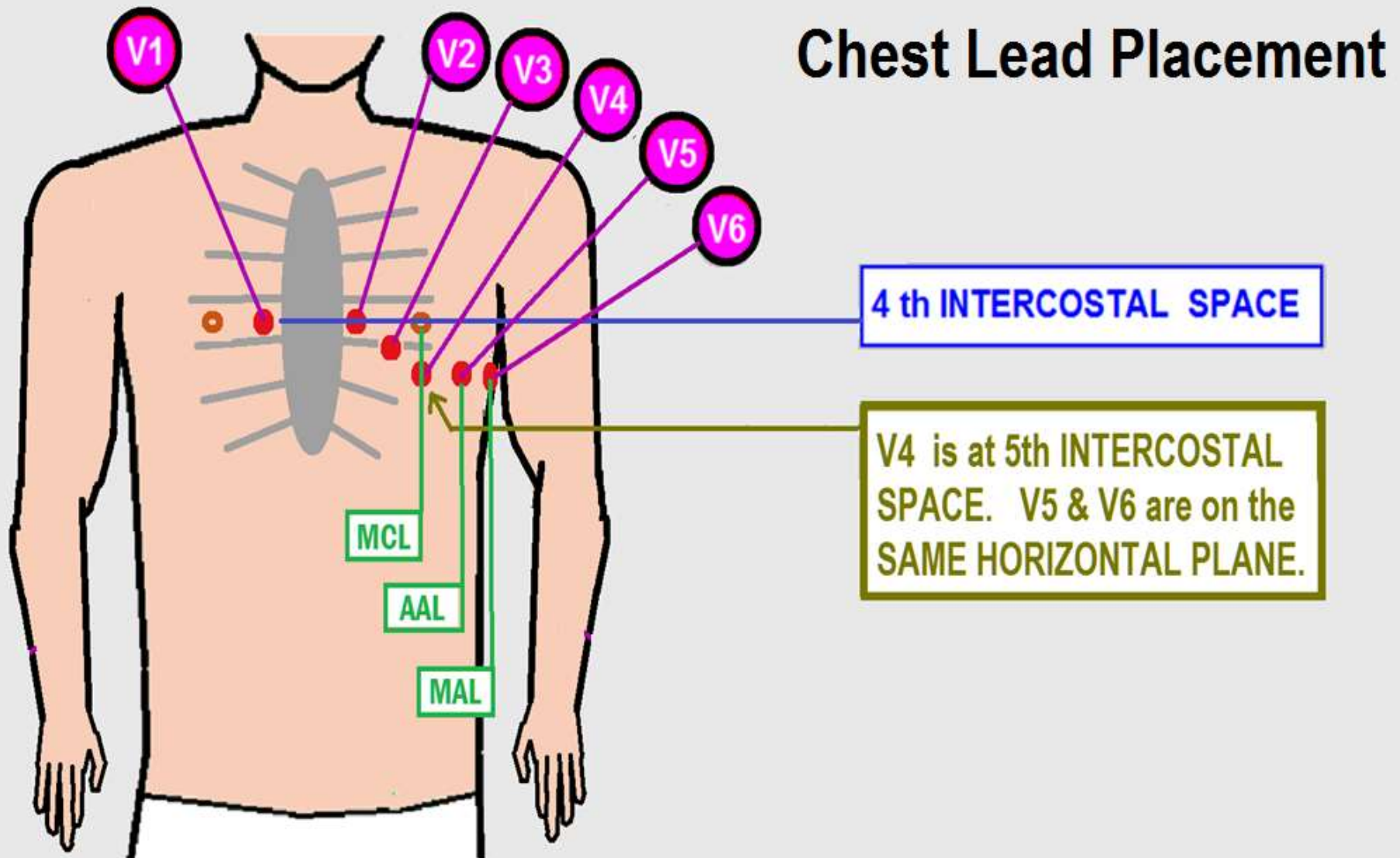
Obtaining the 12 Lead ECG



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.**

CORRECT Lead placement:



DOB [REDACTED] 75 Years

Female

(2)

Rate 76 Sinus rhythm.....normal P axis, V-rate 50- 99

PR 161
QRSD 90
QT 350
QTc 394

TECH SD

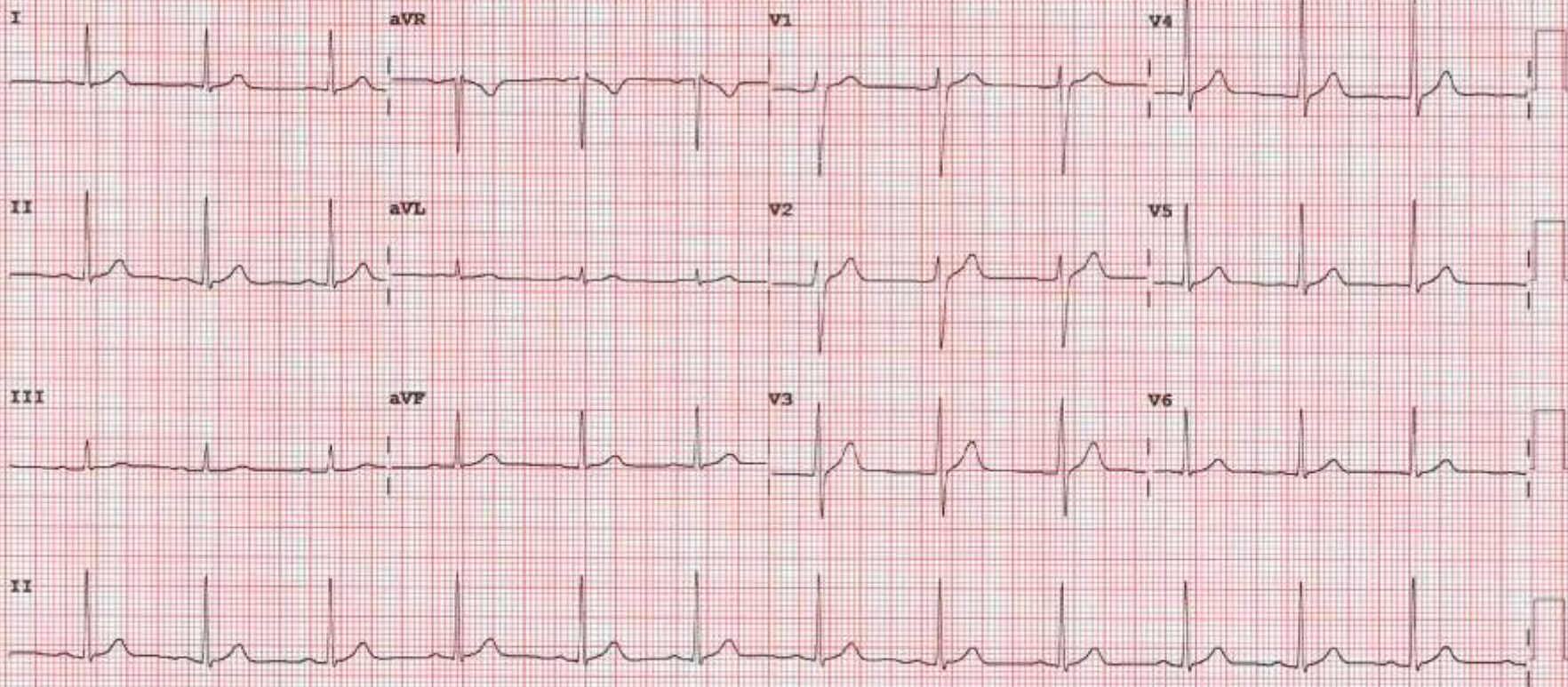
--AXIS--

P 50
QRS 51
T 44

12 Lead; Standard Placement

- NORMAL ECG -

Unconfirmed Diagnosis



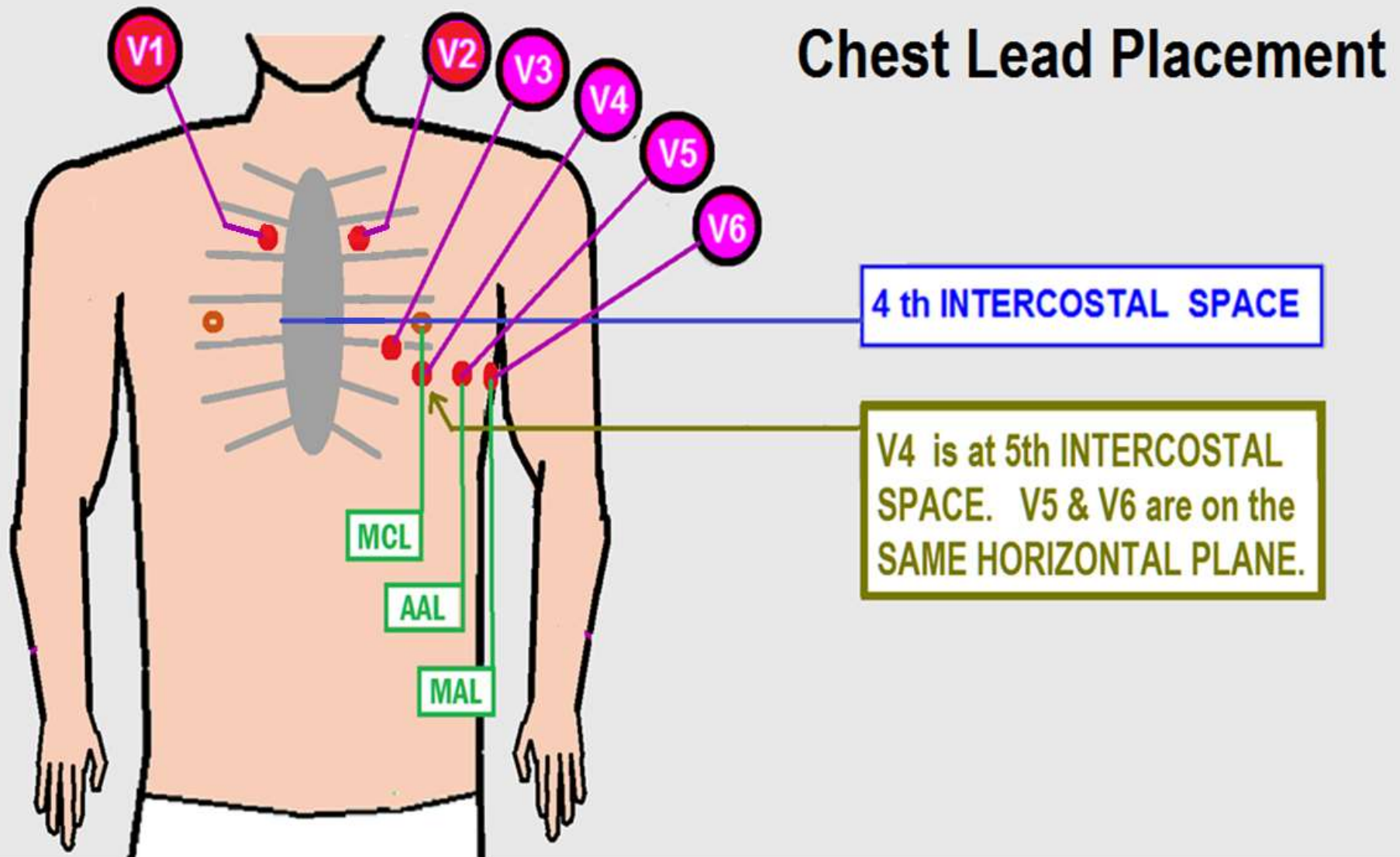
Device: Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10.0 mm/mV

F 60- 0.15-100 Hz

100B CL

P?

INCORRECT Lead placement:



DOB [REDACTED] 1988 30 Years

Female

5:20:58 AM

(1)

Rate 89 Sinus rhythm.....normal P axis V-rate 50- 99
Anteroseptal infarct, age indeterminate.....Q >35ms

PR 157
QRSD 96
QT 365
QTc 445

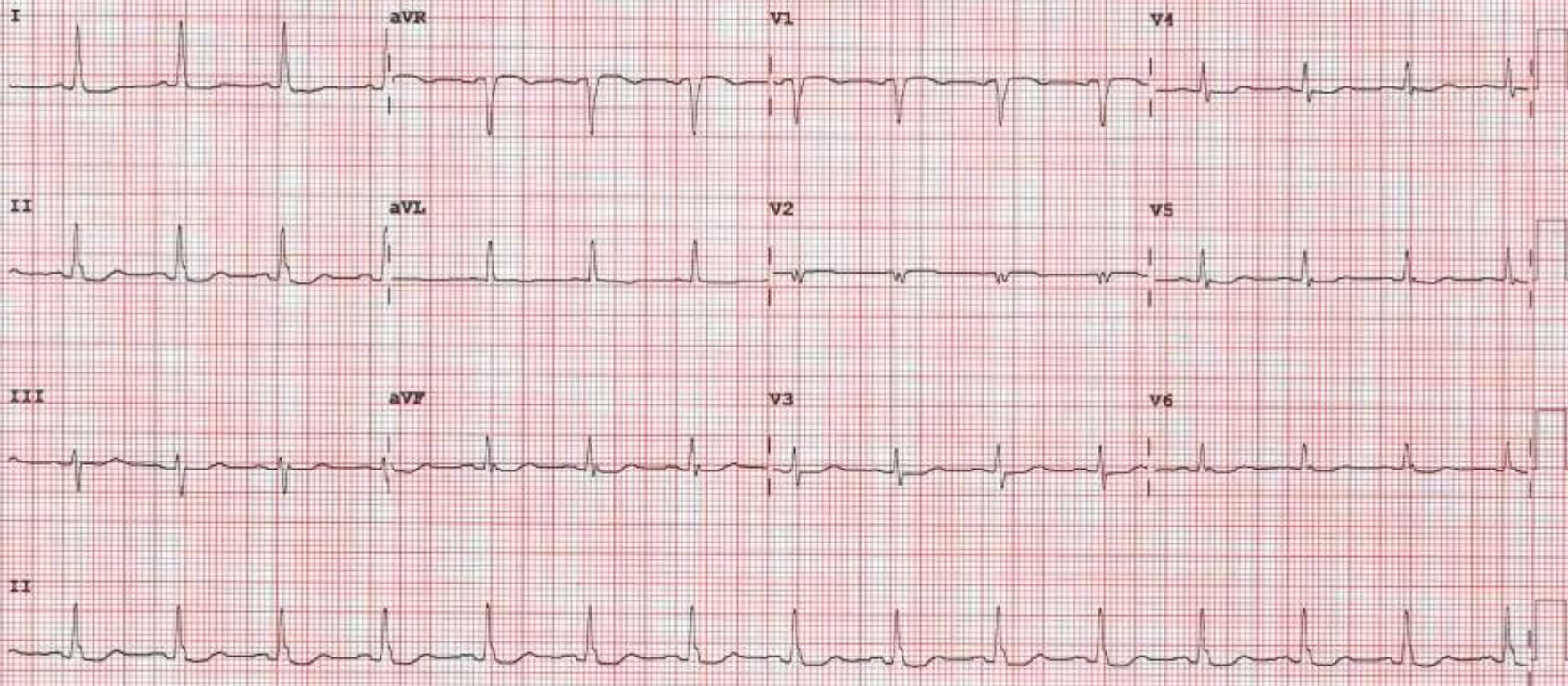
--AXIS--

P 46
QRS 24
T 86

12 Lead; Standard Placement

- ABNORMAL ECG -

Unconfirmed Diagnosis



Device

Speed: 25 mm/sec

Limb: 10 mm/mV

Chest: 10.0 mm/mV

F 60~ 0.15-100 Hz

1^1^8 CL

P?

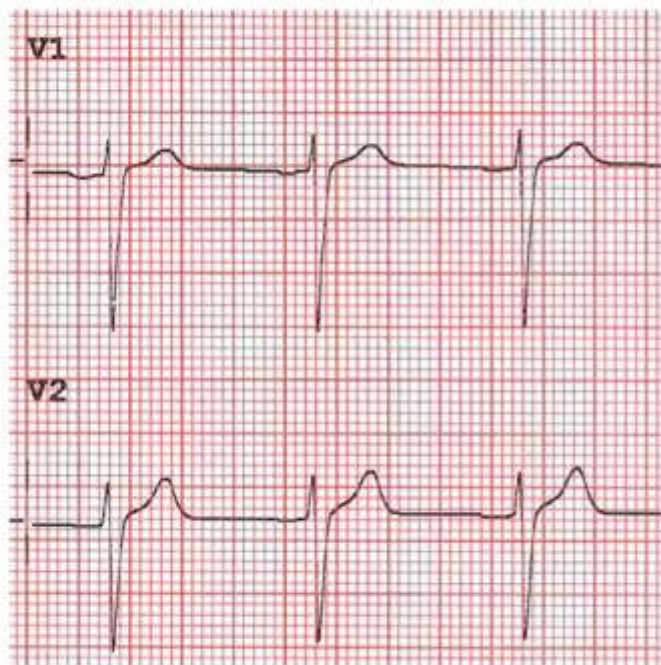
AHA/ACC/HRS Scientific Statement

Recommendations for the Standardization and Interpretation of the Electrocardiogram

Part I: The Electrocardiogram and Its Technology

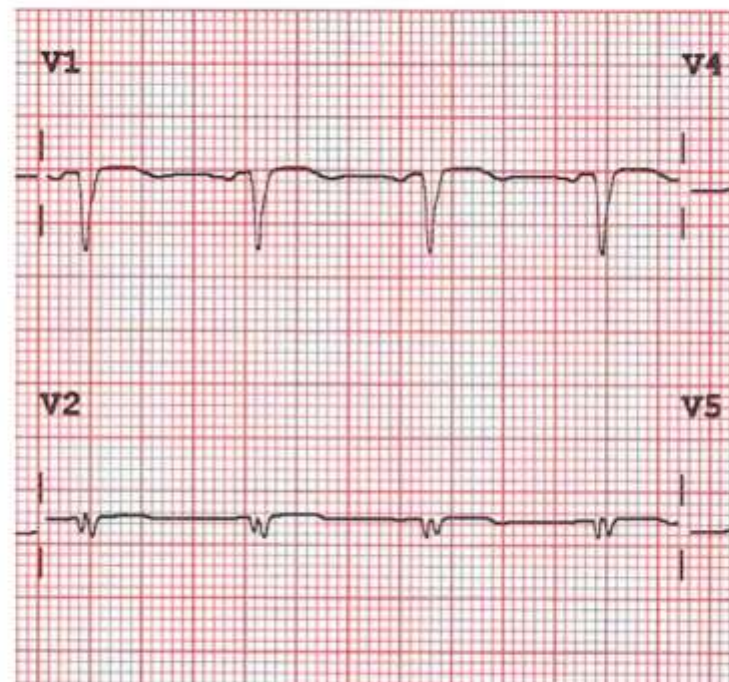
the often profound alterations in waveforms that can result from precordial electrode misplacement.^{85,86} A common error is superior misplacement of V_1 and V_2 in the second or third intercostal space. This can result in reduction of initial R-wave amplitude in these leads, approximating 0.1 mV per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

Correct Lead Placement



RS = NO old MI

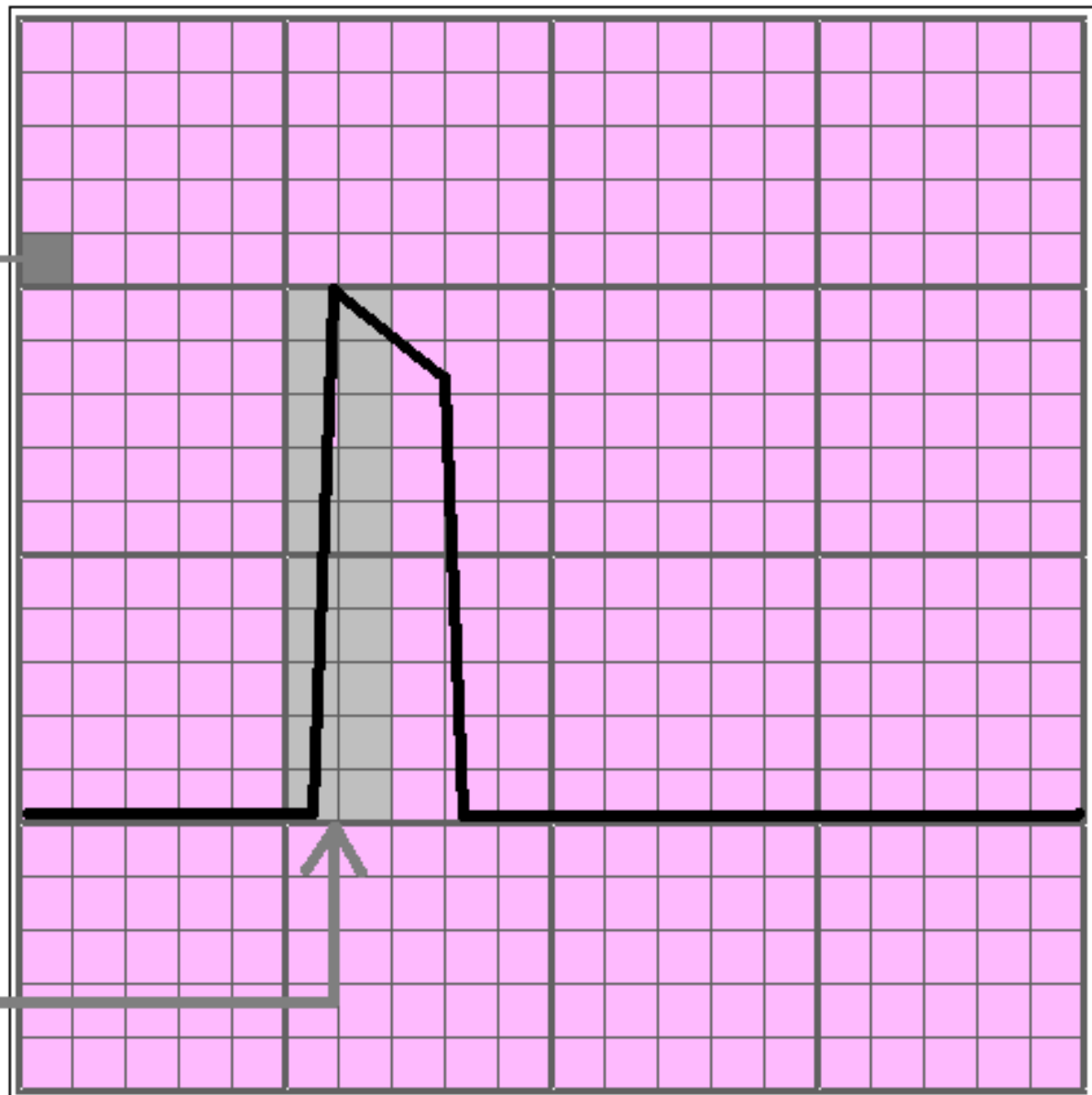
Incorrect Lead Placement



QS = old MI

ECG PAPER - THE VERTICAL AXIS:

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



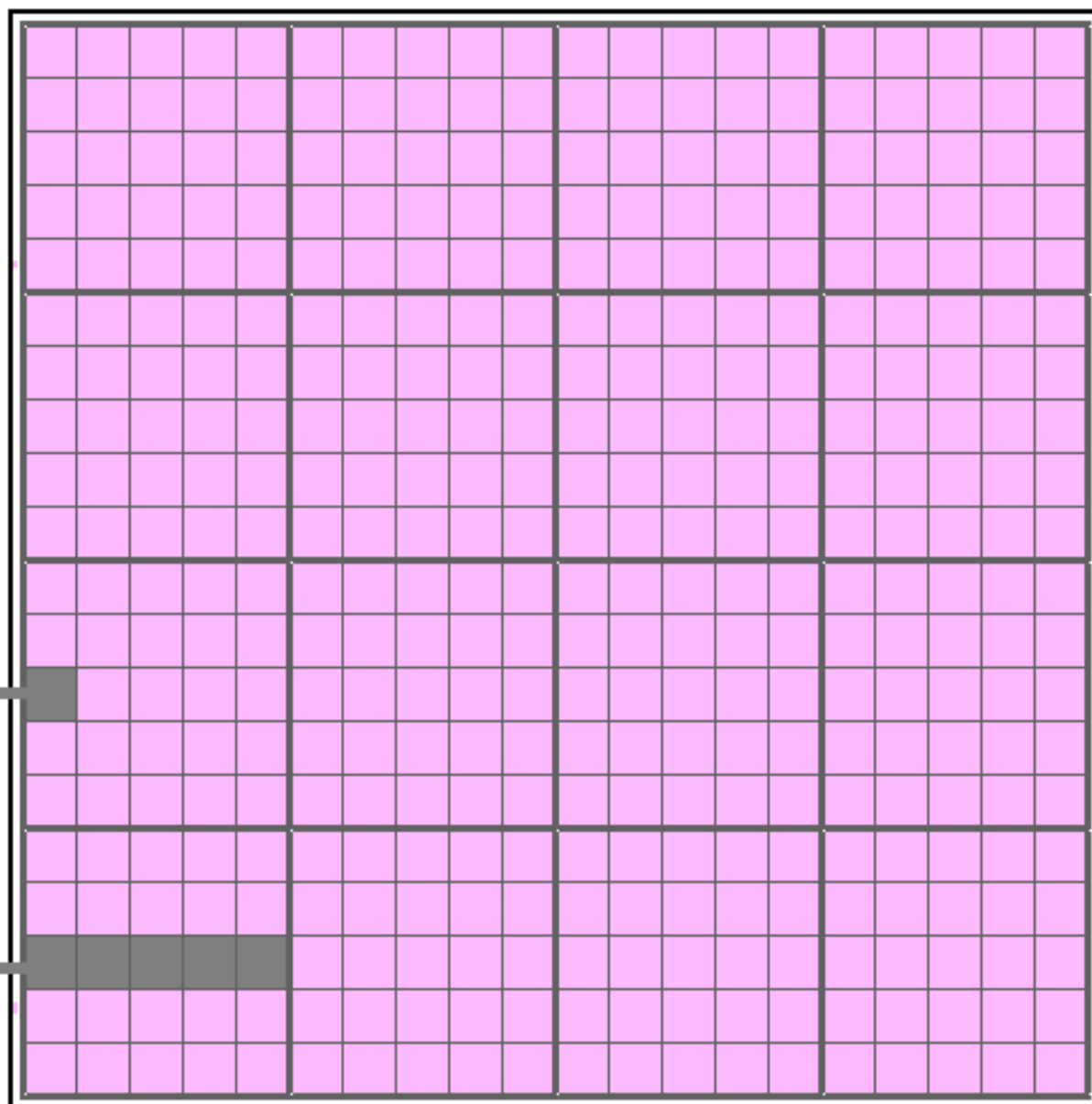
ECG PAPER - THE HORIZONTAL AXIS:

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)



40years

Male Caucasian

Vent. rate 65 bpm

PR interval 192 ms

QRS duration 104 ms

QT/QTc 362/376 ms

P-R-T axes 39 0 23

Normal sinus rhythm

Normal ECG

NORMAL 12 LEAD ECG**6 LIMB LEADS - view the vertical axis****6 PRECORDIAL LEADS - view the horizontal axis****← 3 SECONDS →**
D.O.S.: T851

Referred by:

Reviewed by:

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

40 Hz 25.0 mm/s 10.0 mm/mV

1 or more CONTINUOUS RHYTHM STRIPS12SLtm v250

PRINTED IN U.S.A.

DOB [REDACTED] 75 Years

Rate 76 Sinus rhythm.

PR 161

QRSD 90

QT 350

QTc 394

---AXIS---

P 50

QRS 51

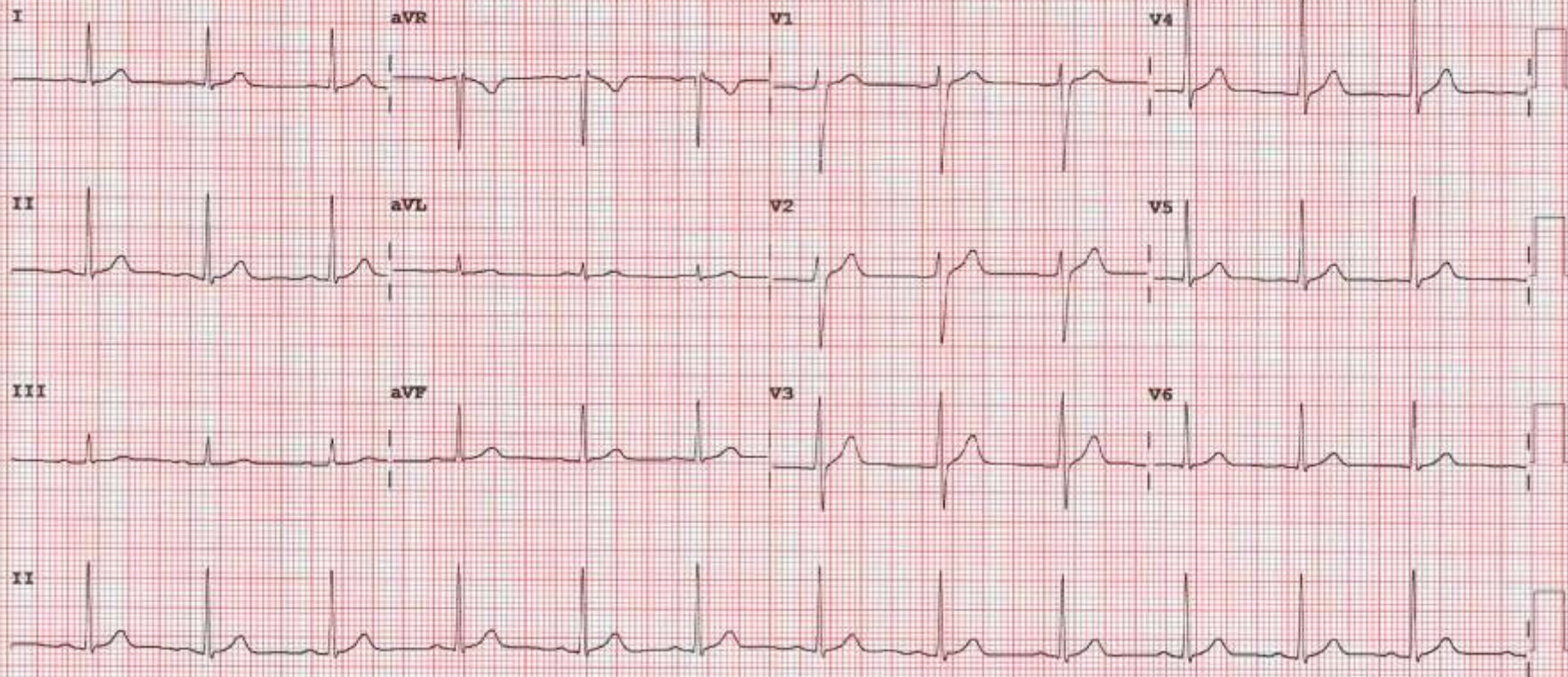
T 44

12 Lead; Standard Placement

Evaluate this EKG for each of the “Normal” criteria seen on the NEXT 2 SLIDES

- NORMAL ECG -

Unconfirmed Diagnosis



Device:

Speed: 25 mm/sec

Limb: 10 mm/mV

Chest: 10.0 mm/mV

F 60- 0.15-100 Hz

100B CL

P?

The Normal 12 Lead EKG

- NSR (rate 60-100, regular rhythm)
- P Waves upright all leads except aVR
- P Waves inverted lead aVR, possibly V1
- QRS upright Leads I, II, III, aVL, aVF, V5, V6
- QRS inverted Leads aVR, V1, V2
- QRS biphasic: Leads V3, V4
- P wave size: up to 2mm tall, 2.5mm long
- QRS height Limb Leads: 5-15mm tall
- QRS height V Leads 10-15mm tall
- QRS width: not to exceed 3mm (120 ms)
- Overall QRS Amplitude: not greater than 30mm

The Normal 12 Lead EKG

- T waves – Upright all Leads except aVR
- T wave – Inverted in Lead aVR
- (everything is inverted in lead aVR)
- T wave MAY be inverted (as a normal variant) in Leads III and aVL.
- Overall QRS Amplitude: not greater than 30mm

MEASURING THE "OVERALL QRS AMPLITUDE"

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

Referred by:

Confirmed By:

TALLEST R WAVE is in LEAD V4 = 11 mm

DEEPEST S WAVE is in LEAD V2 = 8 mm

OVERALL QRS AMPLITUDE = 19 mm

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 ECG MACHINE

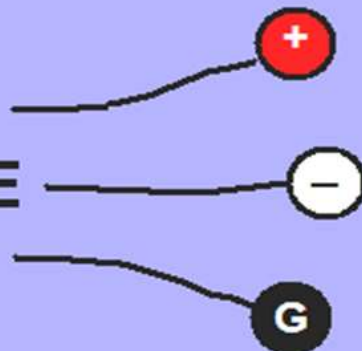
STANDARD 12 LEADS - USES 10 WIRES
(6 CHEST and 4 LIMB)

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

1 POSITIVE ELECTRODE

1 NEGATIVE ELECTRODE

1 GROUND ELECTRODE

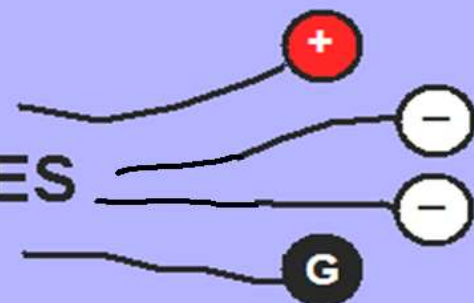


- LEADS AVR, AVL, and AVF

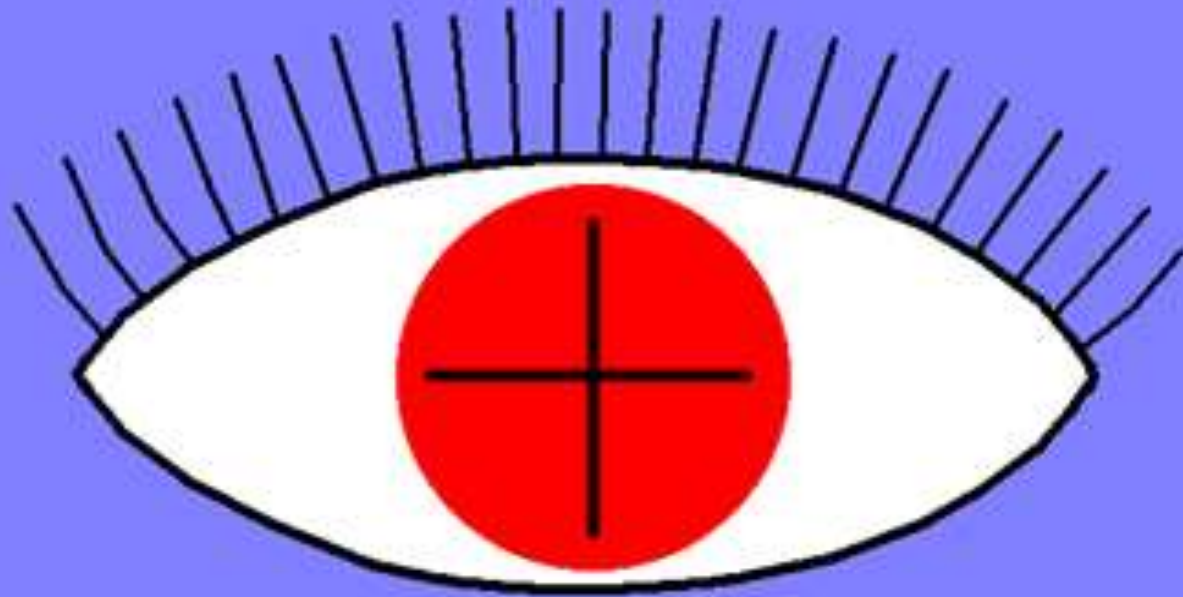
1 POSITIVE ELECTRODE

2 NEGATIVE ELECTRODES

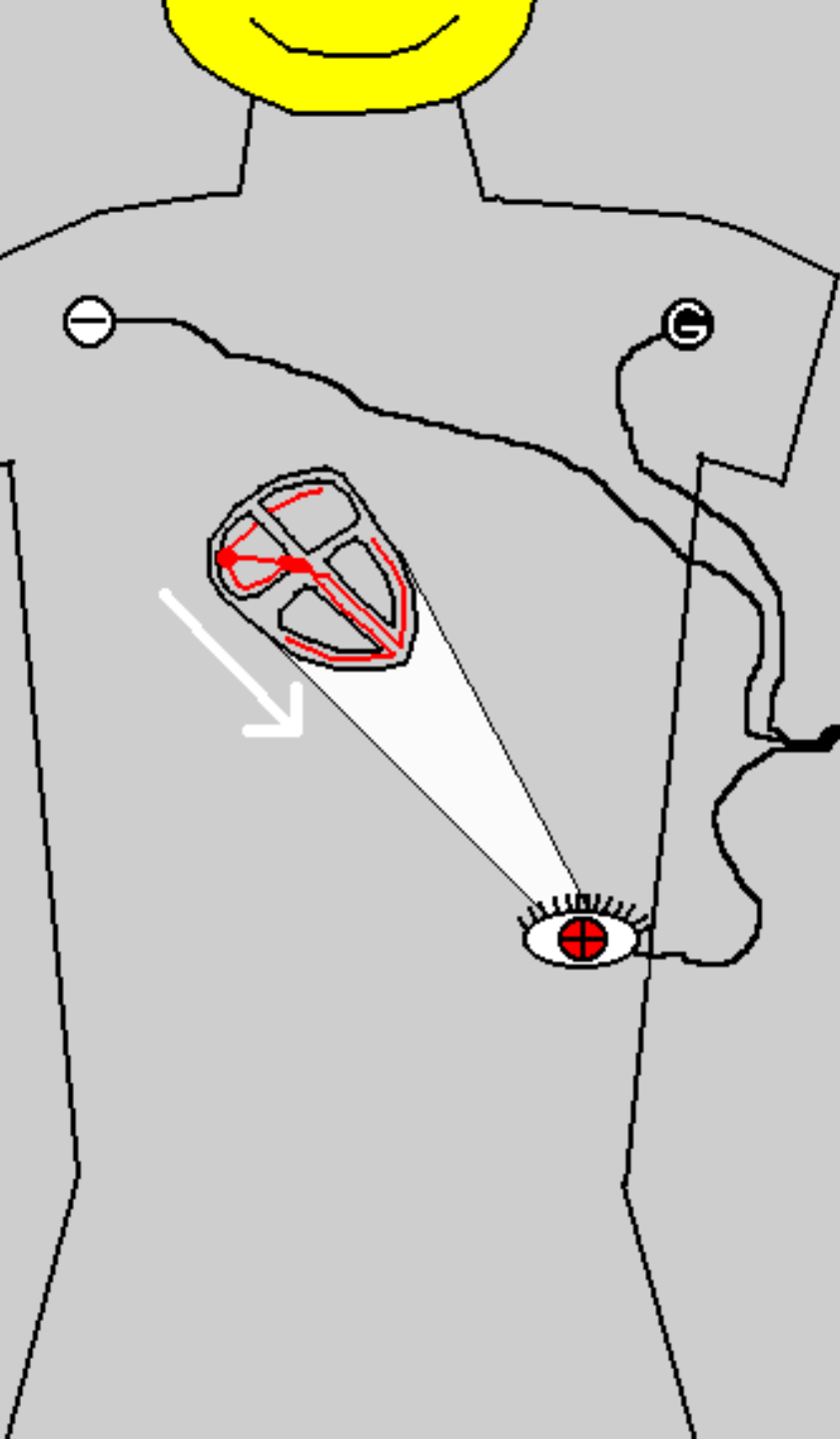
1 GROUND ELECTRODE



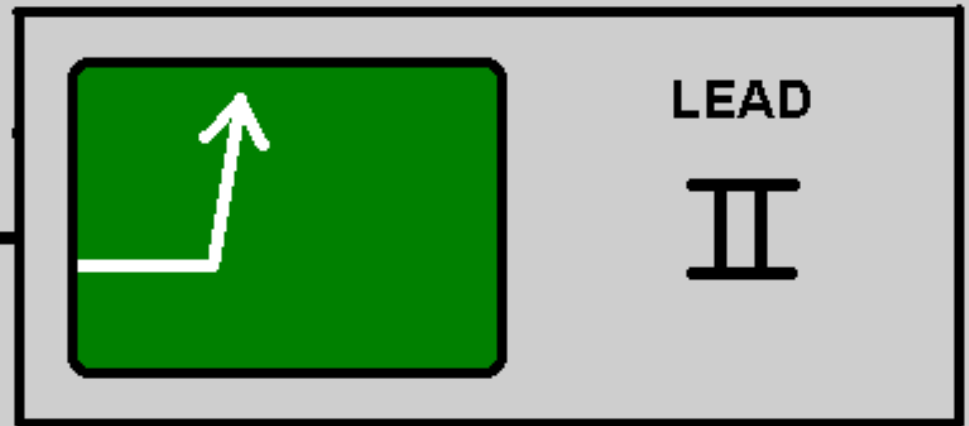
THE POSITIVE ELECTRODE



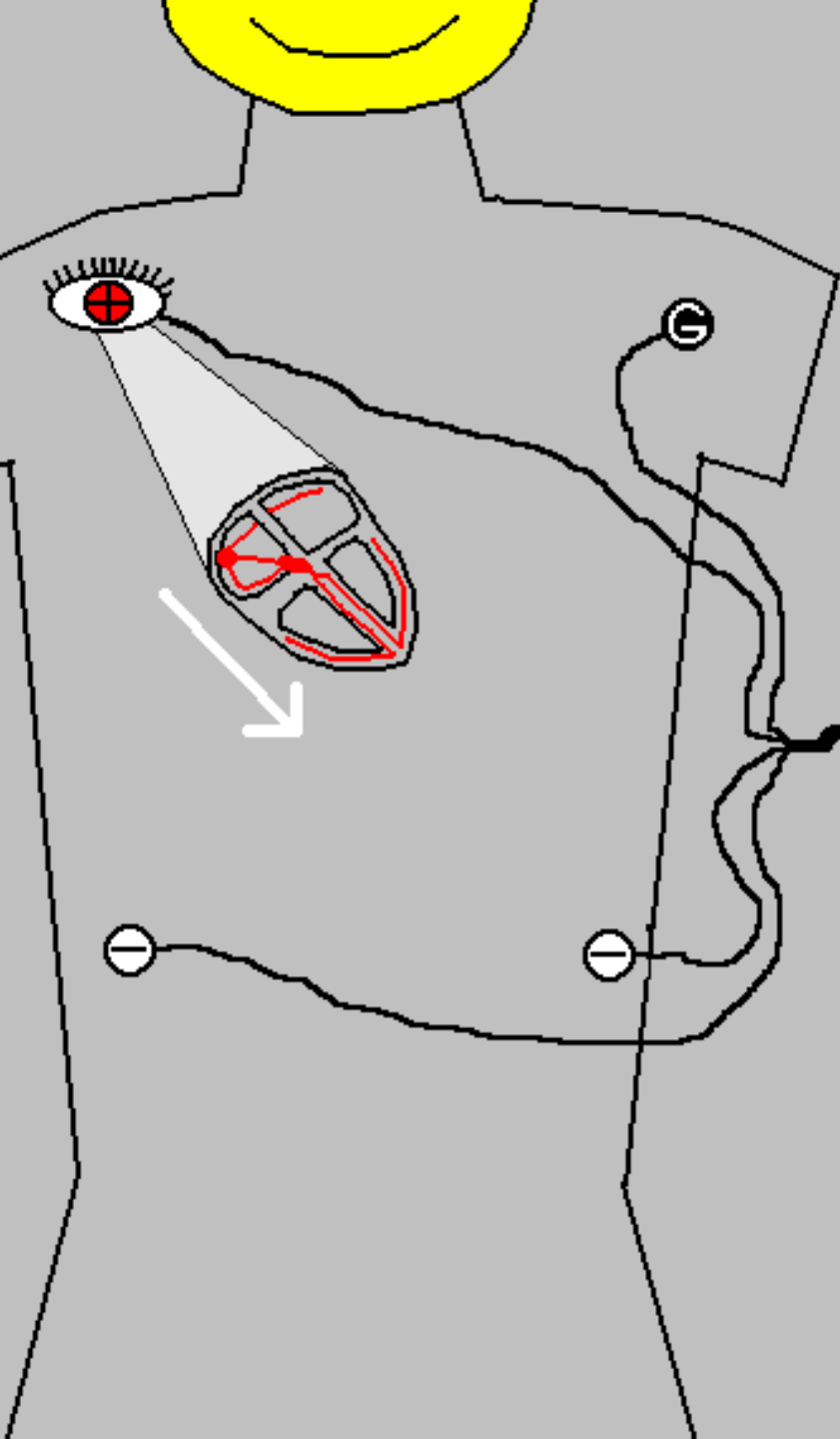
IS THE "EYE" . . .



**CURRENT MOVING
TOWARD THE EYE
(POSITIVE ELECTRODE)**



**RECORDS AN
"UPWARD"
DEFLECTION**



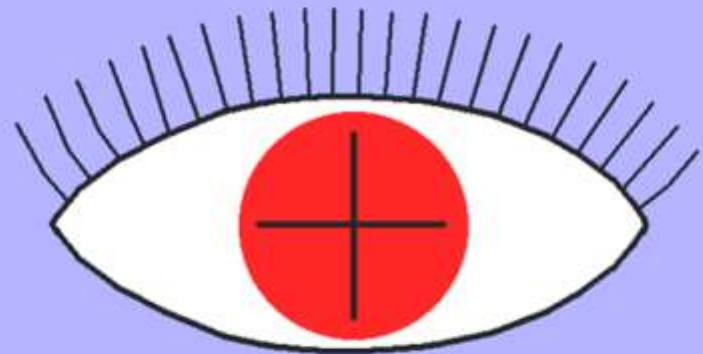
**CURRENT MOVING
AWAY FROM
THE EYE
(POSITIVE ELECTRODE)**



**RECORDS A
"DOWNWARD"
DEFLECTION**

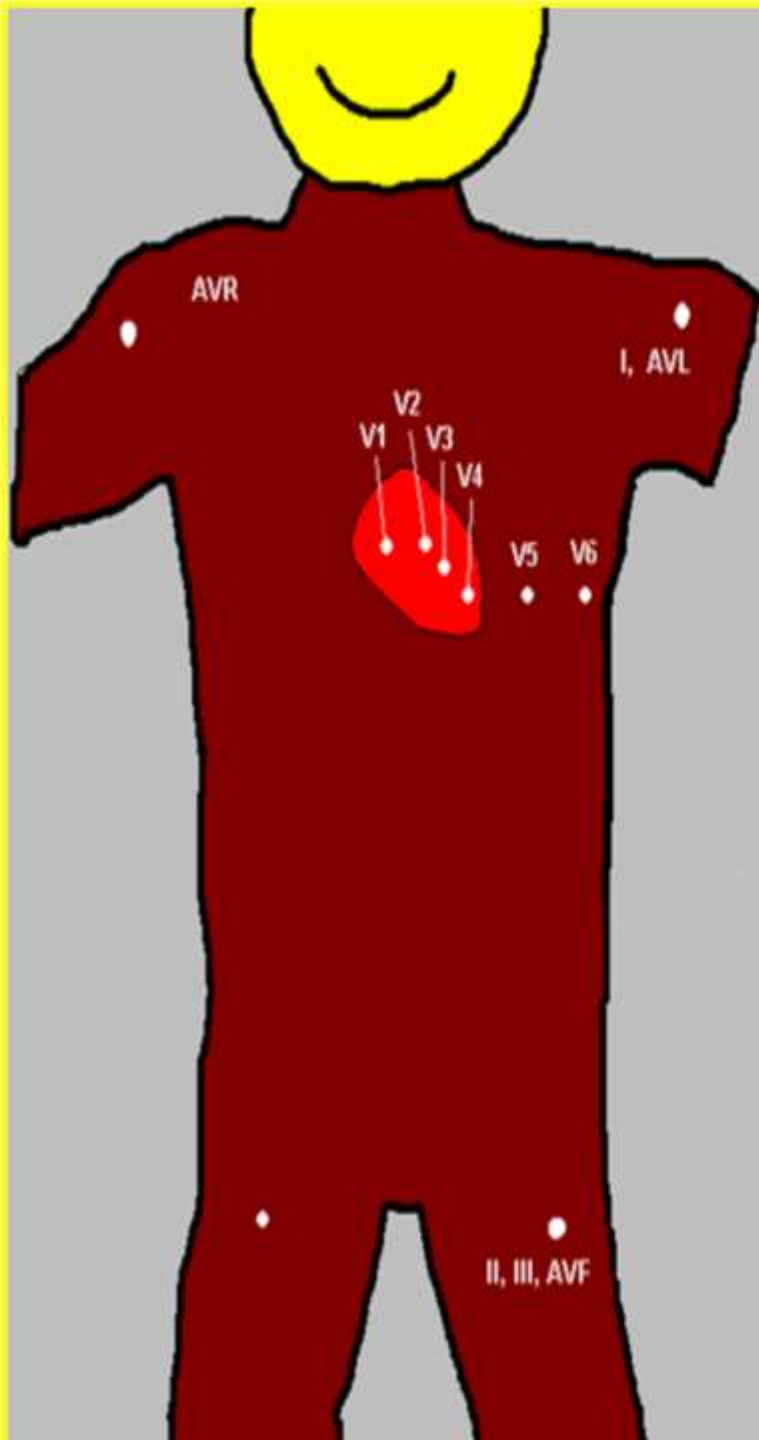
***What part of the HEART
does each lead SEE ?***

THE POSITIVE ELECTRODE



IS THE "EYE" . . .

Imagine a body made of clear glass, with only a HEART inside. We dip this body in liquid chocolate, and then scratch holes in each spot where we normally place the ECG leads



AREAS VIEWED by 12 LEAD ECG

AVR

AVL, I

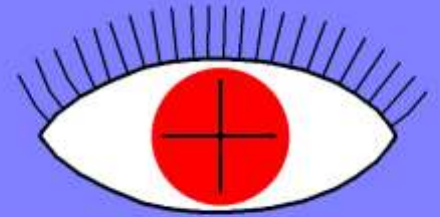
V1, V2

V3, V4

V5, V6

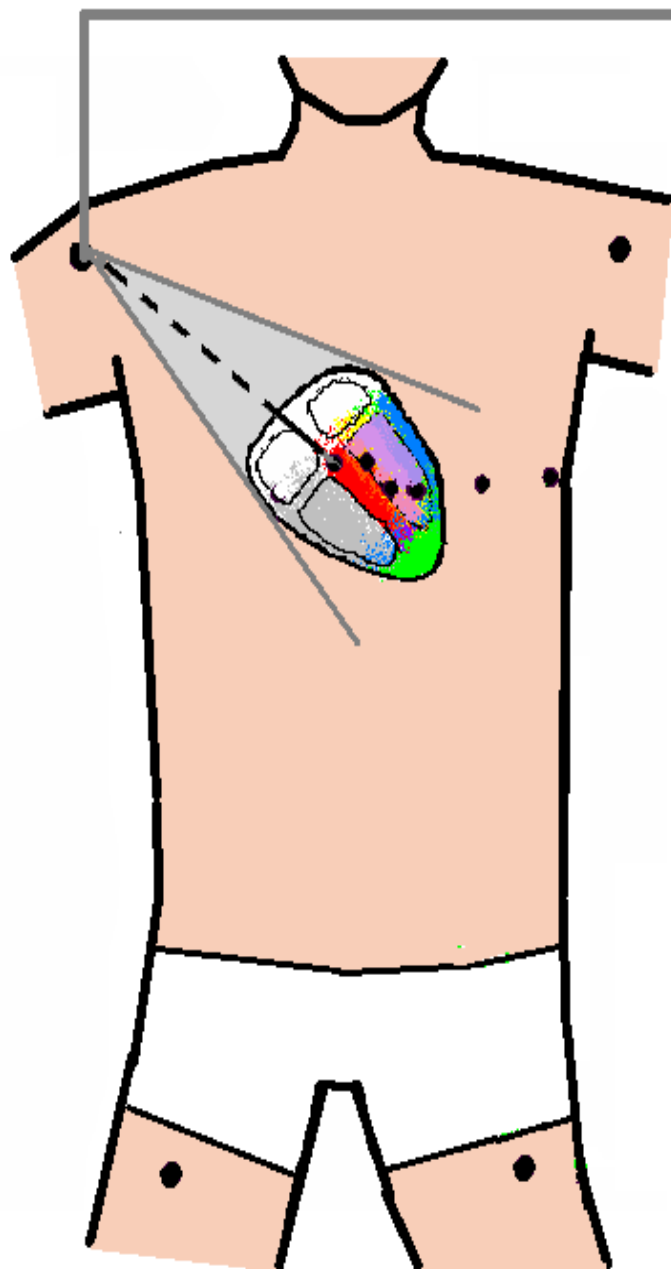
II, III, AVF

THE POSITIVE ELECTRODE



IS THE "EYE" . . .

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

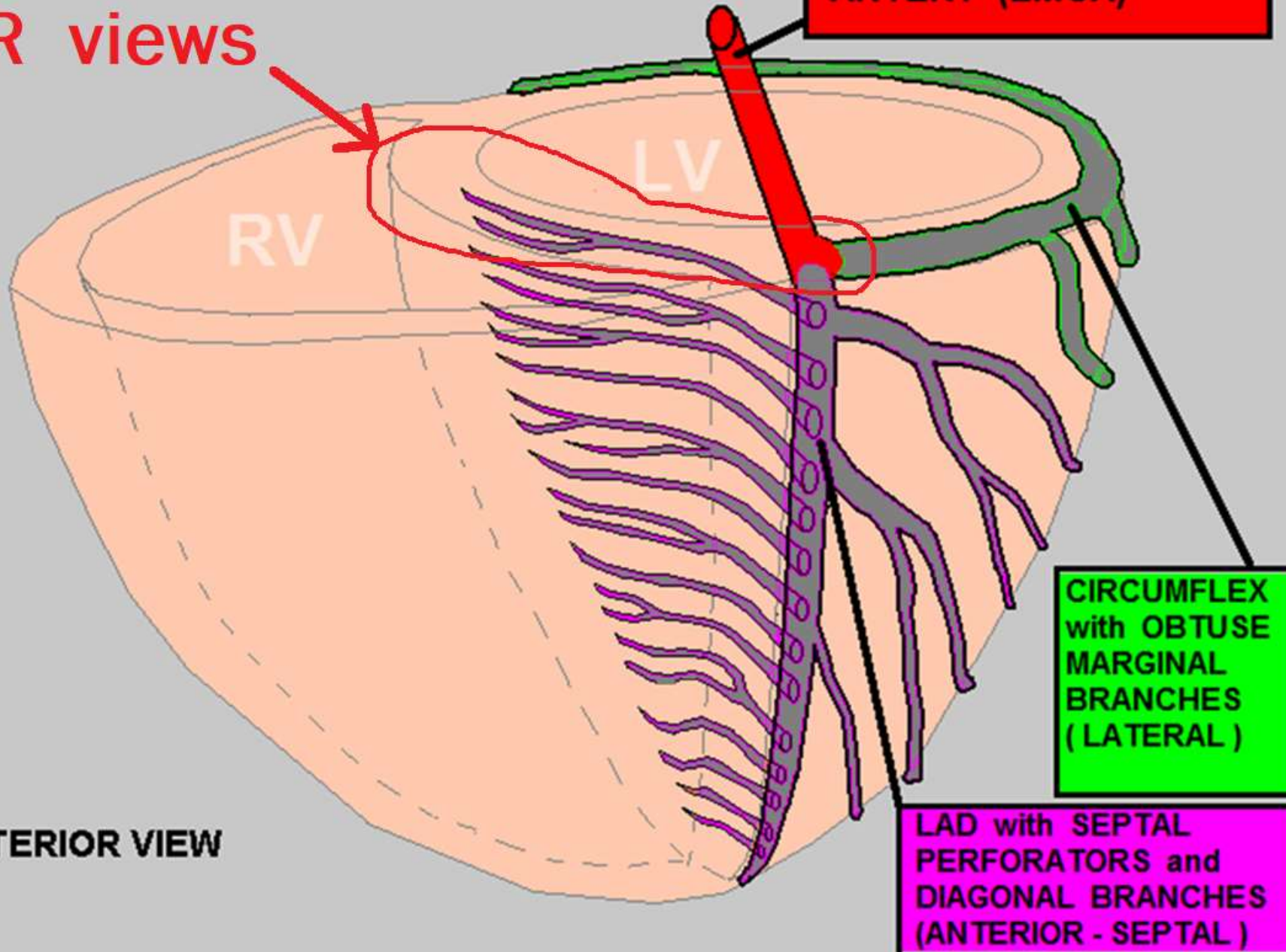


RUPPERT, WAYNE		ID: 7445683	59	05-OCT-2006	JOHNS-HOPKINS UNIV.	
38 Yrs	Vent. Rate:	68	NORMAL SINUS RHYTHM			
MALE	P-R Int.:	160 ms	Normal EKG			
	QRS:	100 ms	Very Healthy Athletic EKG !			
I	AVR	V1	V4			
II	AVL	V2	V5			
III	AVF	V3	V6			

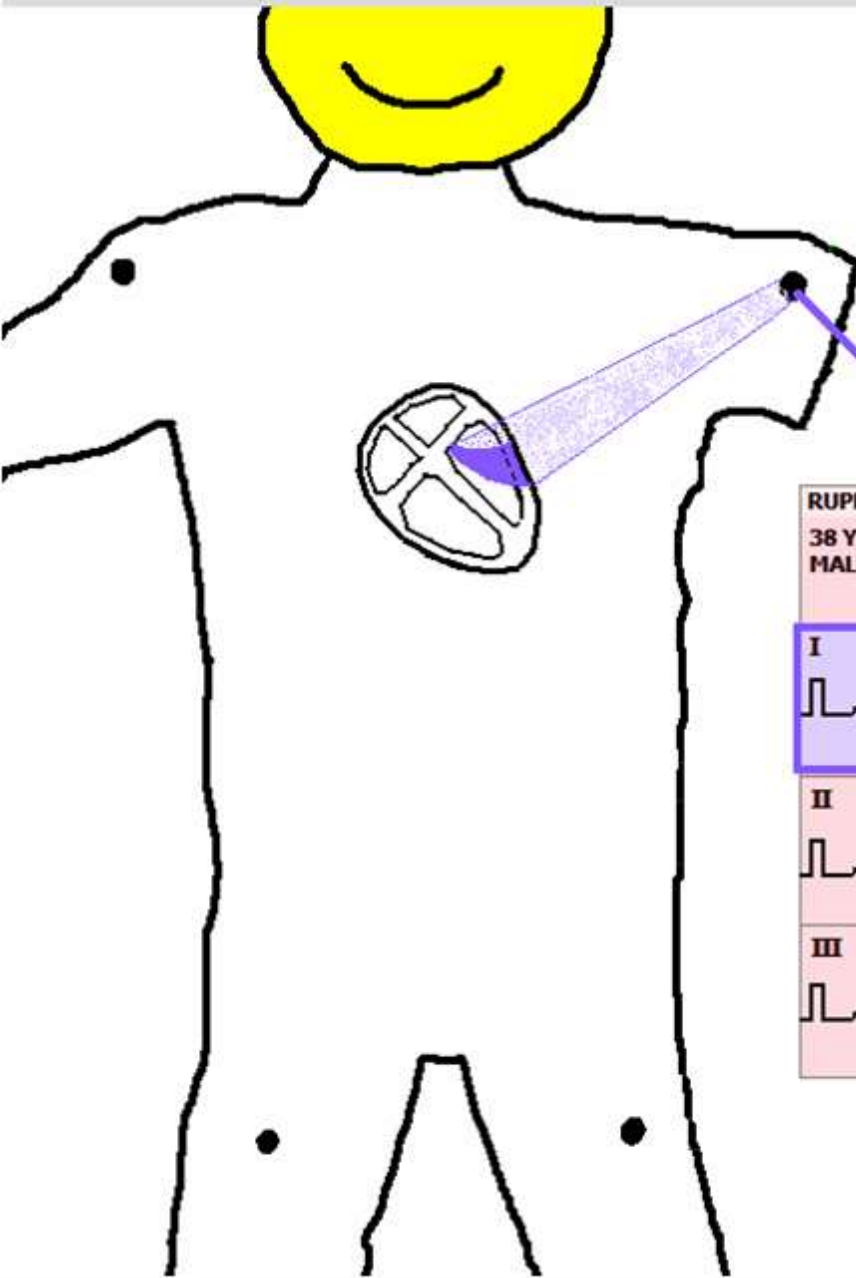
LEFT CORONARY ARTERY SYSTEM

AVR views

ANTERIOR VIEW

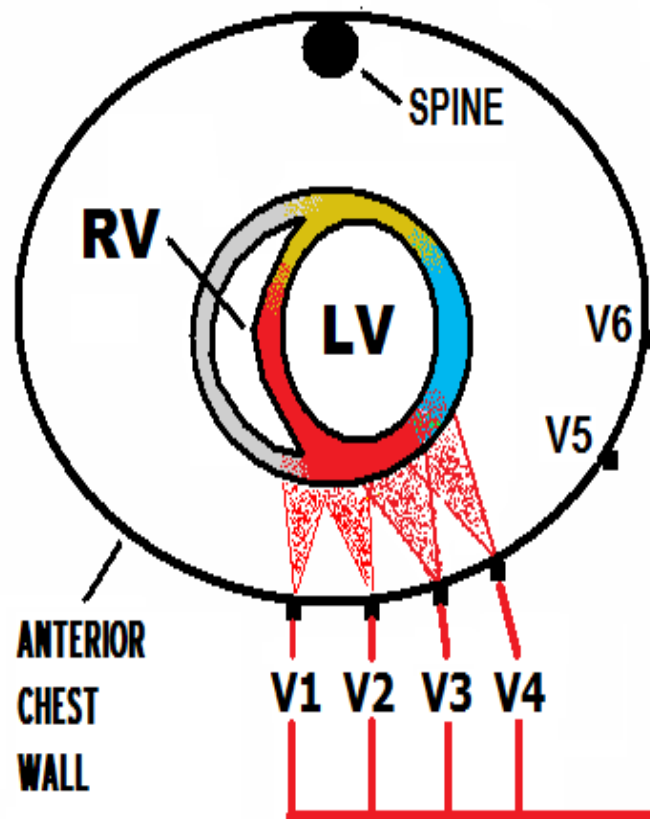


LEADS I and aVL VIEW the LATERAL - ANTERIOR WALL

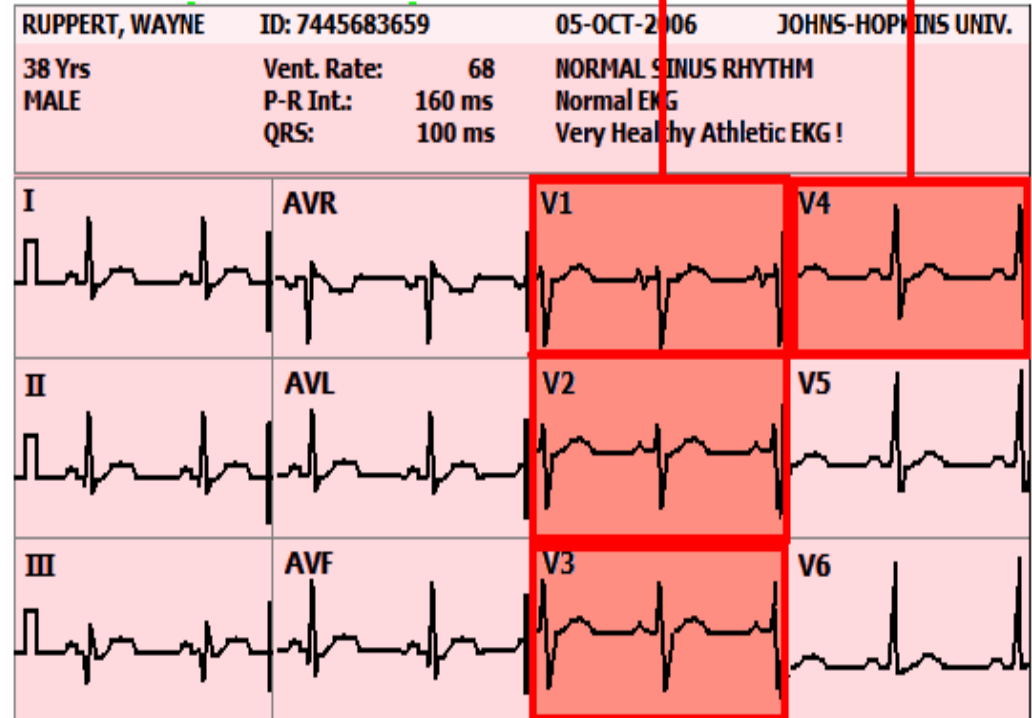


RUPPERT, WAYNE		ID: 744568369	05-OCT-2006	JOHNS-HOPKINS UNIV.
38 Yrs		Vent. Rate: 68	NORMAL SINUS RHYTHM	
MALE		P-R Int: 160 ms	Normal EKG	
		QRS: 100 ms	Very Healthy Athletic EKG !	
I	AVR	V1	V4	
II	AVL	V2	V5	
III	AVF	V3	V6	

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

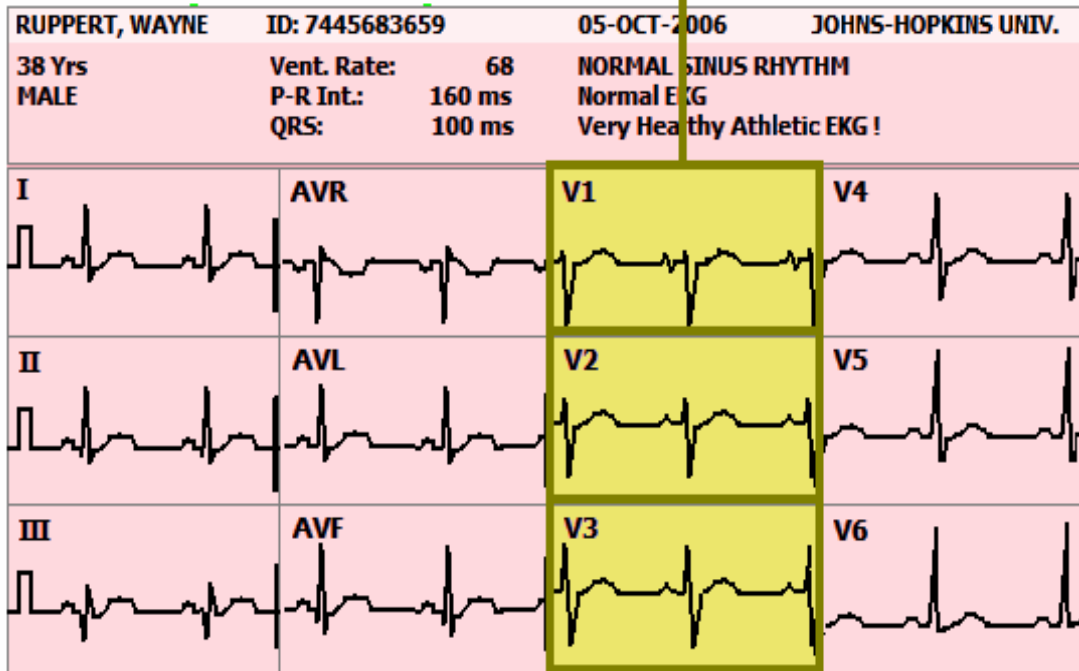
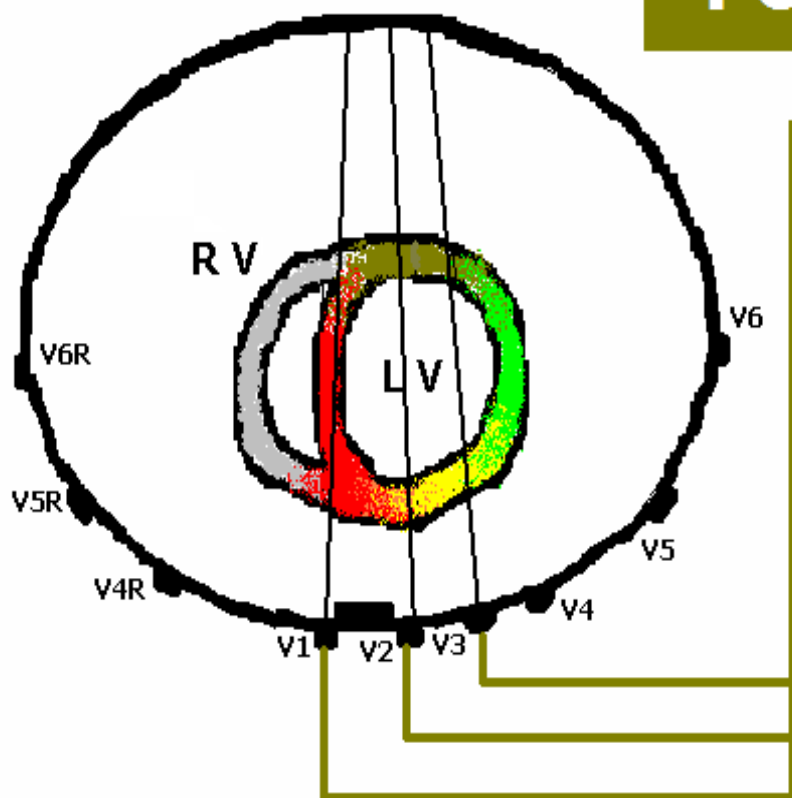


V1, V2 - ANTERIOR / SEPTAL
V3, V4 - ANTERIOR



LEADS V1 - V3 *view the*

POSTERIOR WALL

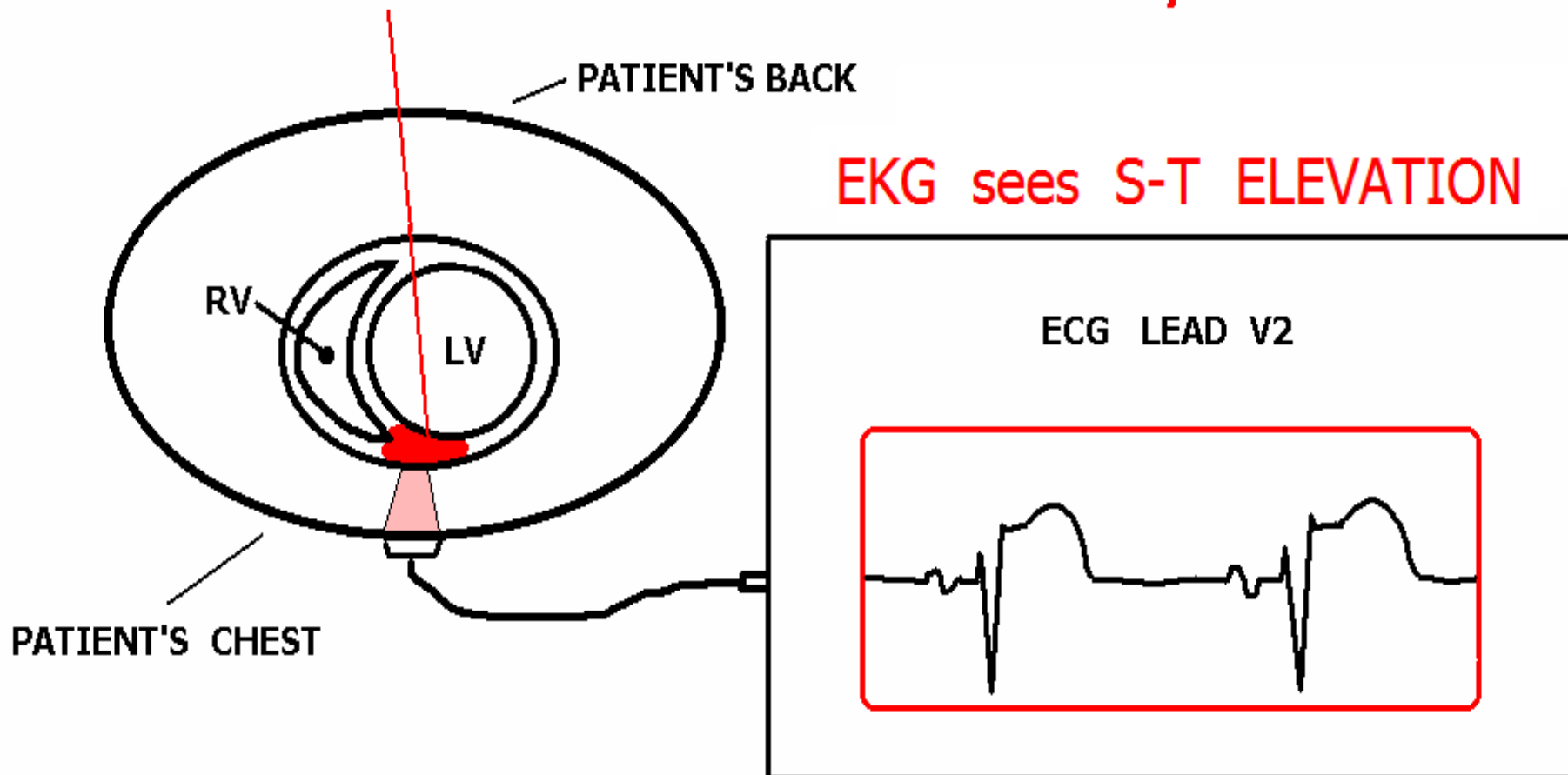


via **RECIPROCAL CHANGES.**

HOW EKG VIEWS INDICATIVE CHANGES

EXAMPLE:

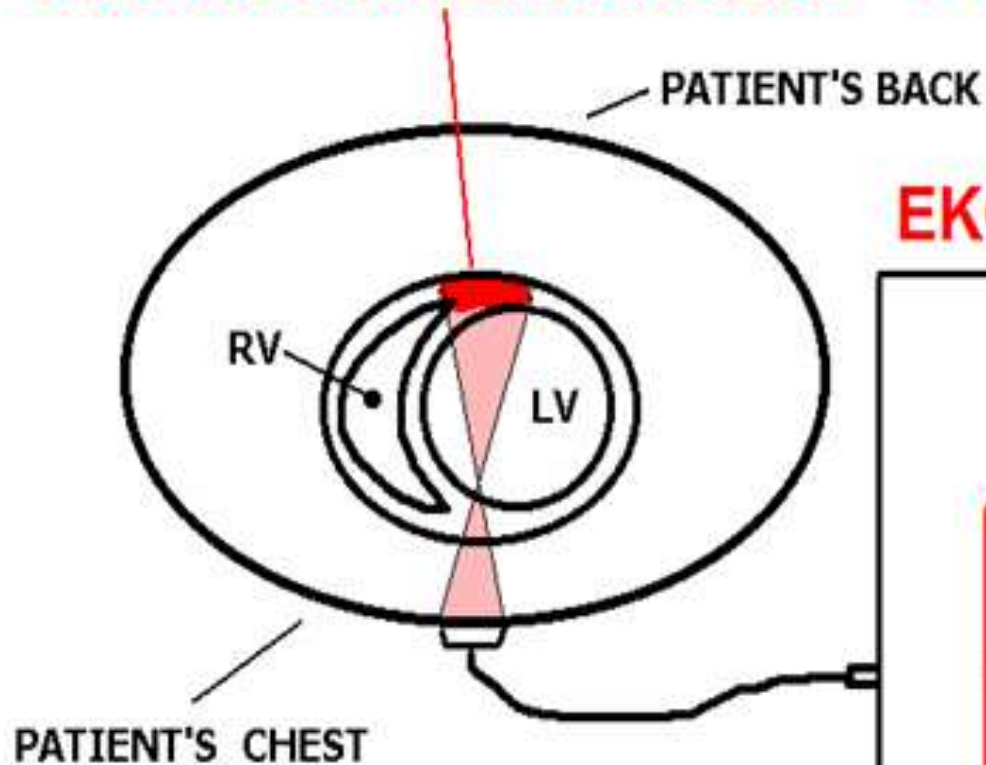
AREA OF ACUTE INFARCTION - ANTERIOR/SEPTAL



HOW EKG VIEWS RECIPROCAL CHANGES

EXAMPLE:

AREA OF ACUTE INFARCTION - POSTERIOR WALL

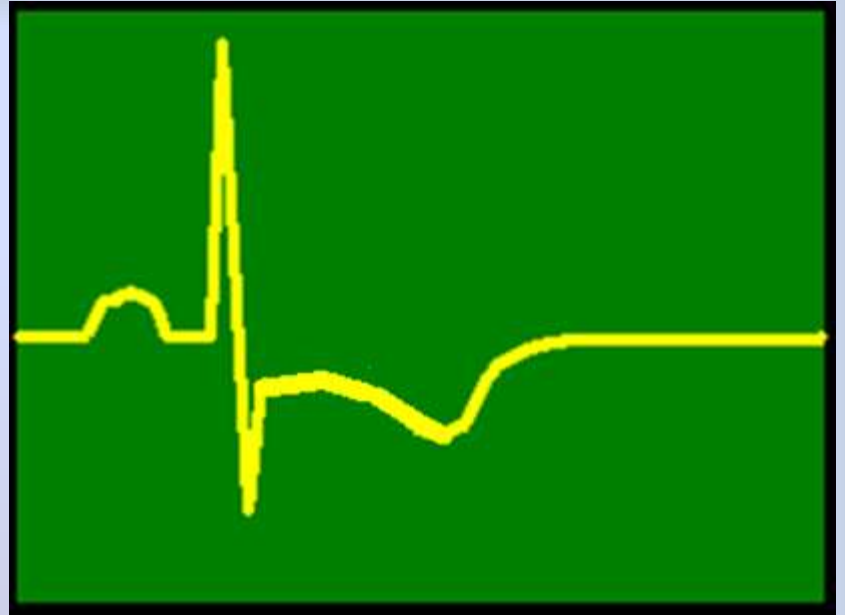


EKG sees S-T DEPRESSION

ECG LEAD V2

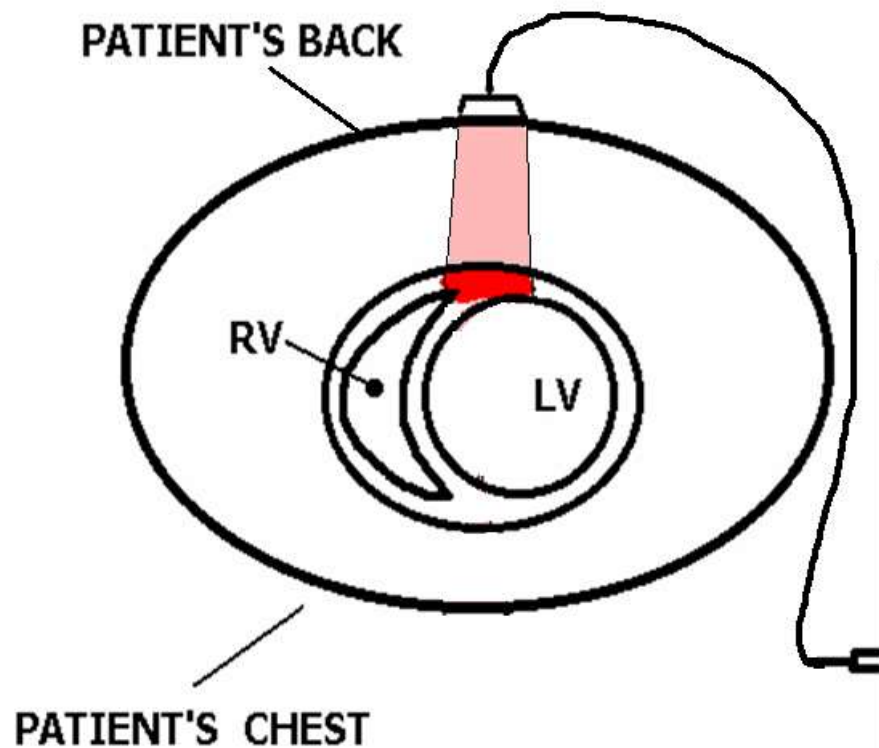


ST Depression can indicate:



- ISCHEMIA
- “Partial-wall thickness” MI (NSTEMI)
- STEMI (in the opposite side of the heart)
- Other things (like RBBB, certain medications, etc).

**If we put ECG leads on the BACK
of a PATIENT who is having an
ACUTE POSTERIOR WALL MI**

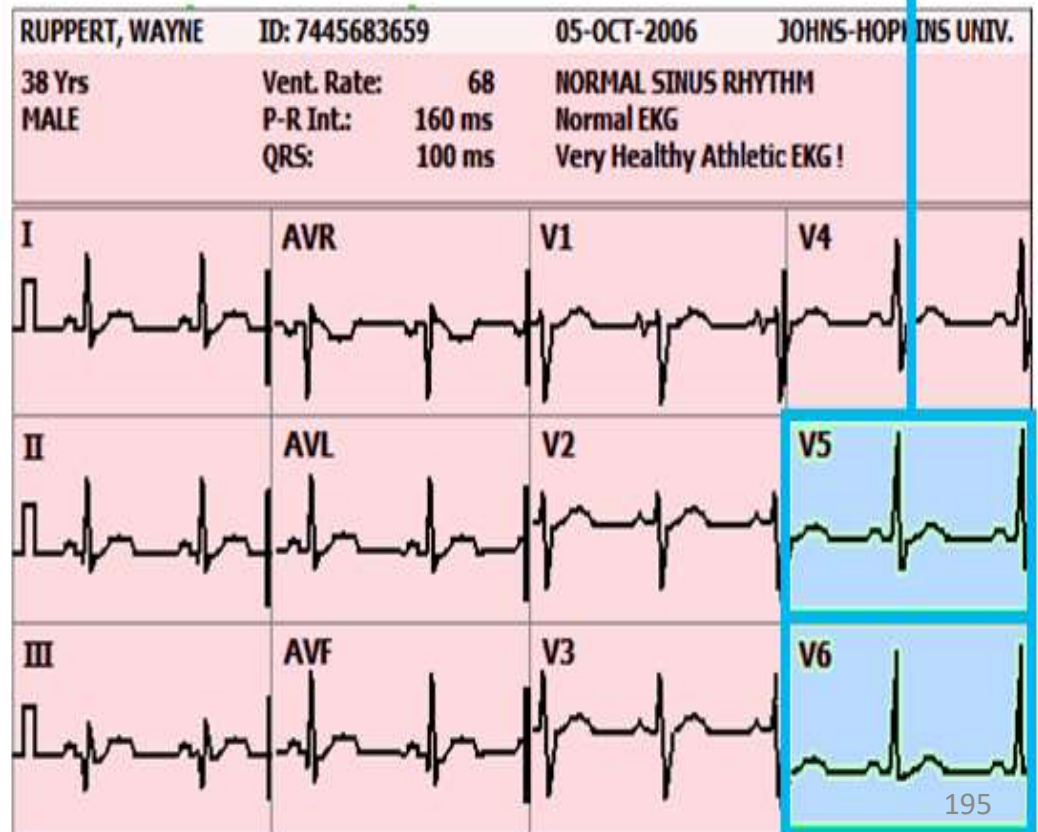
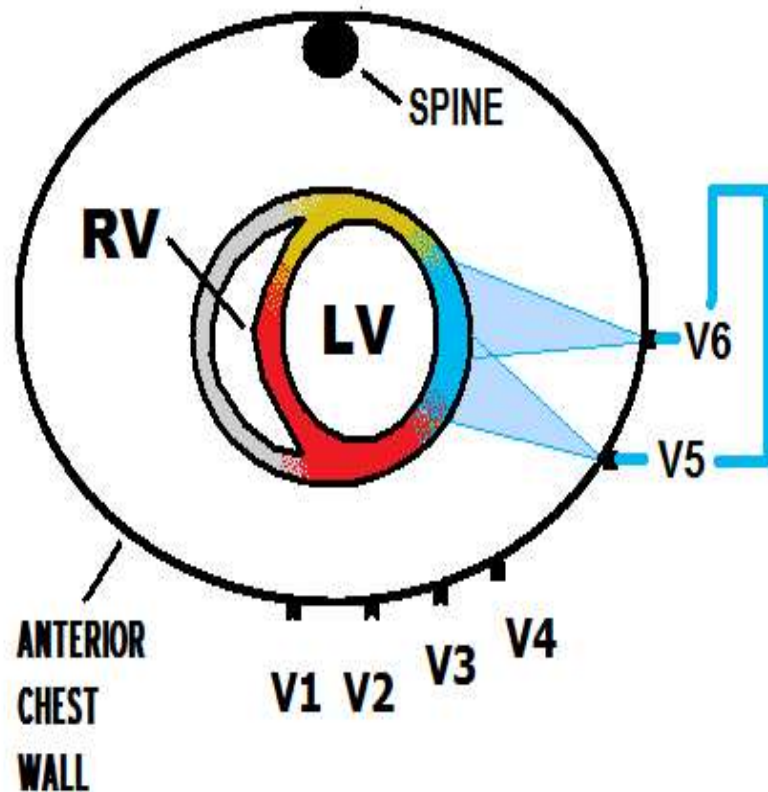


EKG sees S-T ELEVATION

ECG LEADS: V7, V8 or V9

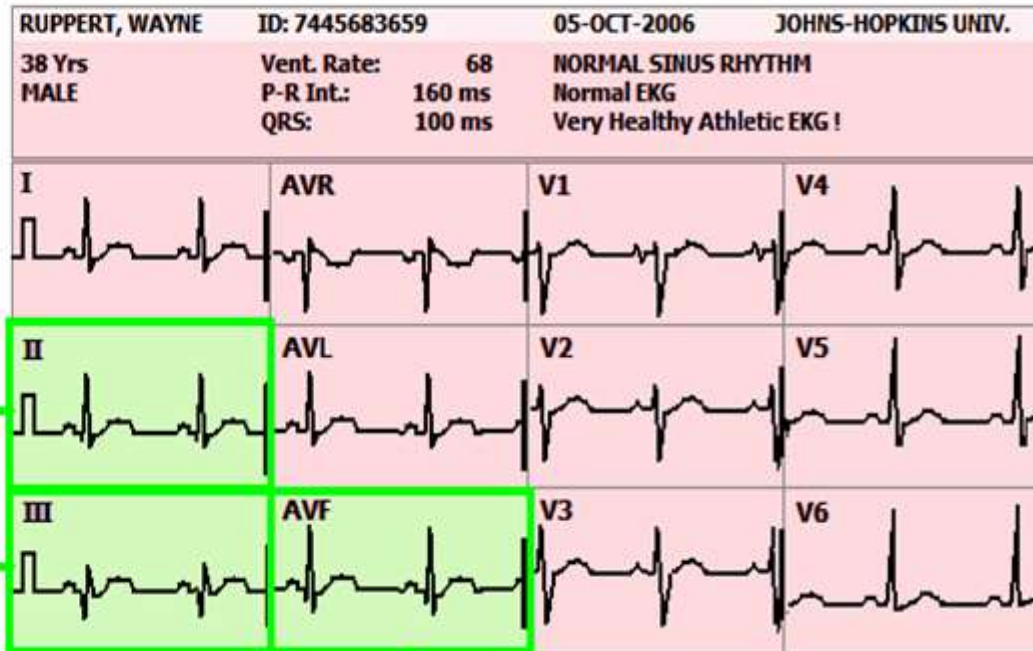


V5 - V6 VIEW THE LATERAL WALL of the LEFT VENTRICLE



LEADS II, III, and aVF VIEW

INFERIOR WALL of the LEFT VENTRICLE



AREAS VIEWED by 12 LEAD ECG



AVR	<i>BASILAR SEPTAL</i>
-----	-----------------------

AVL, I	LATERAL ANTERIOR
--------	---------------------

V1, V2	ANTERIOR
--------	----------

SEPTAL

POSTERIOR (recip.)

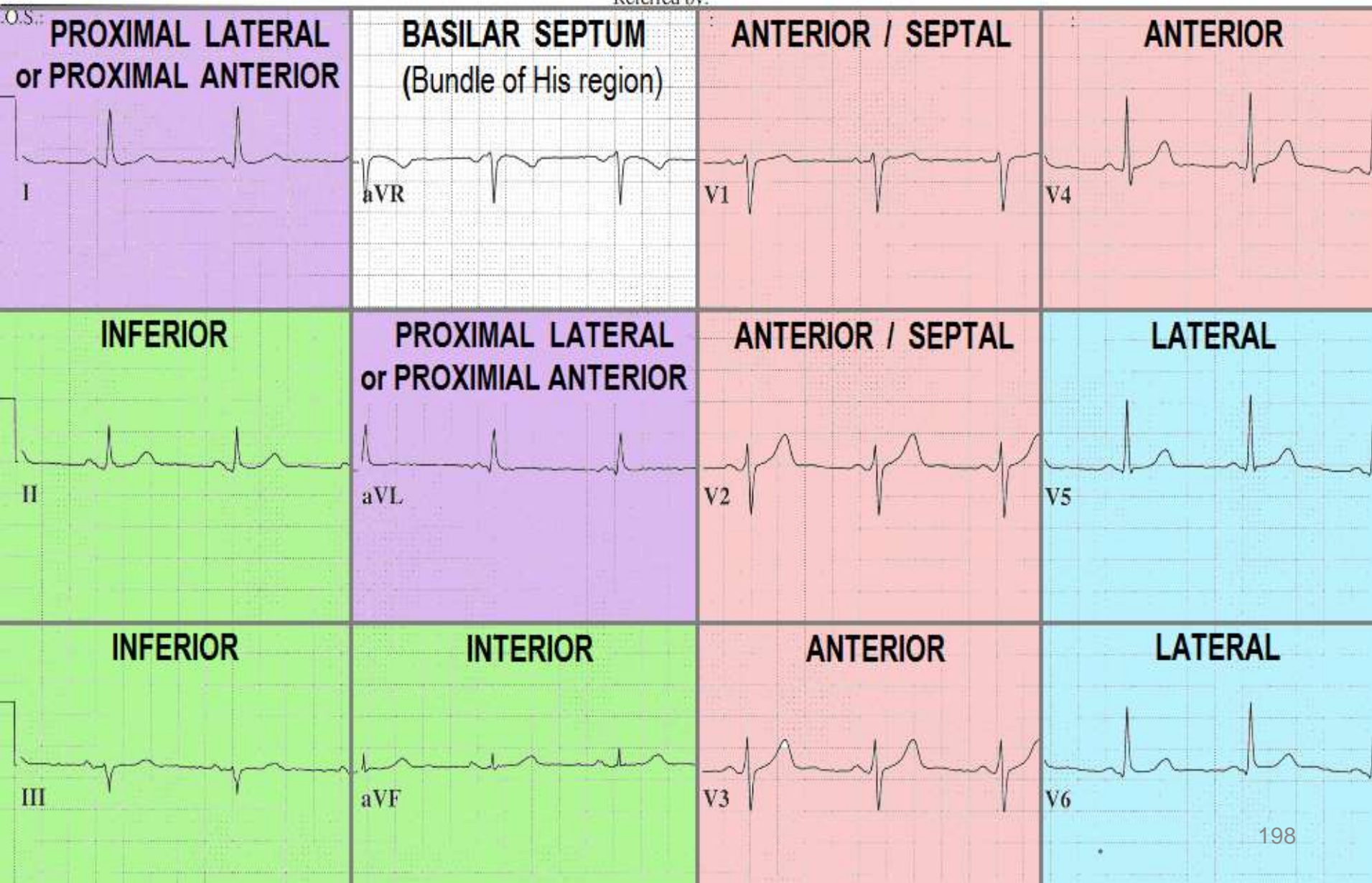
V3, V4	ANTERIOR
--------	----------

V5, V6	LATERAL
--------	---------

II, III, AVF	INFERIOR
--------------	----------

Vent. rate	64	BPM	Normal sinus rhythm
PR interval	130	ms	Normal ECG
QRS duration	96	ms	No previous ECGs available
QT/QTc	396/408	ms	
P-R-T axes	40 11 61		

Referred by:



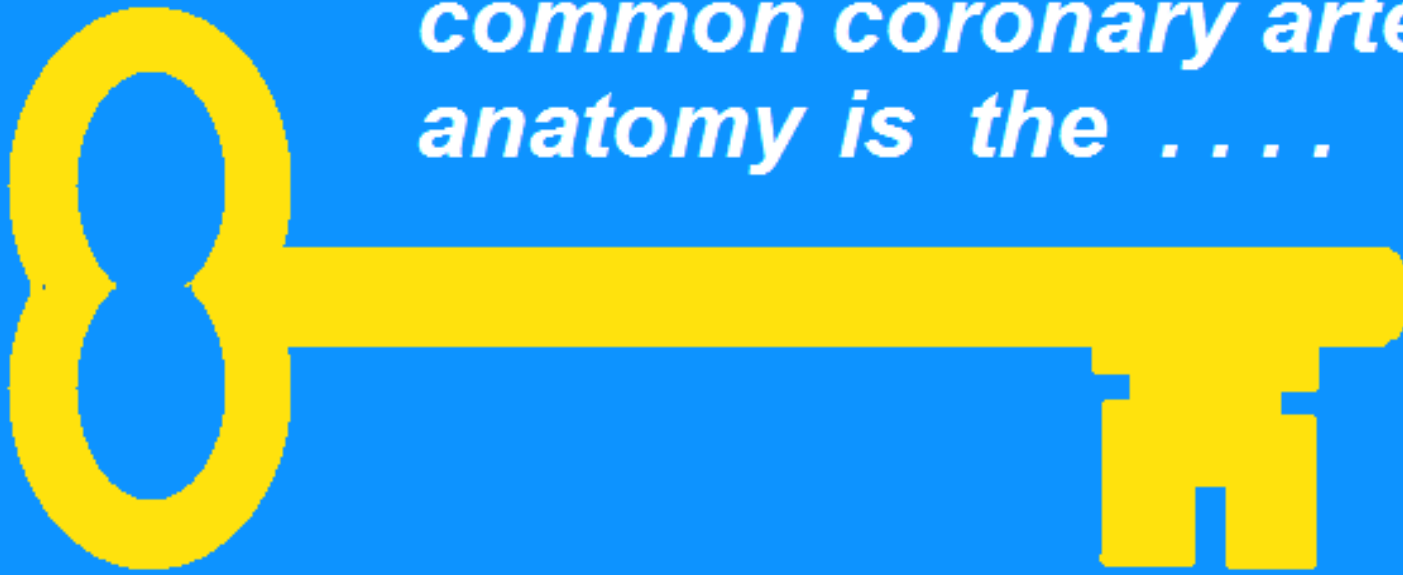
THE CORONARY



ARTERIES

*STRUCTURES
SERVED
BY THE
CORONARY
ARTERIES*

*"Having knowledge of
common coronary artery
anatomy is the*



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

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

The 12 Lead ECG becomes your “crystal ball !!”

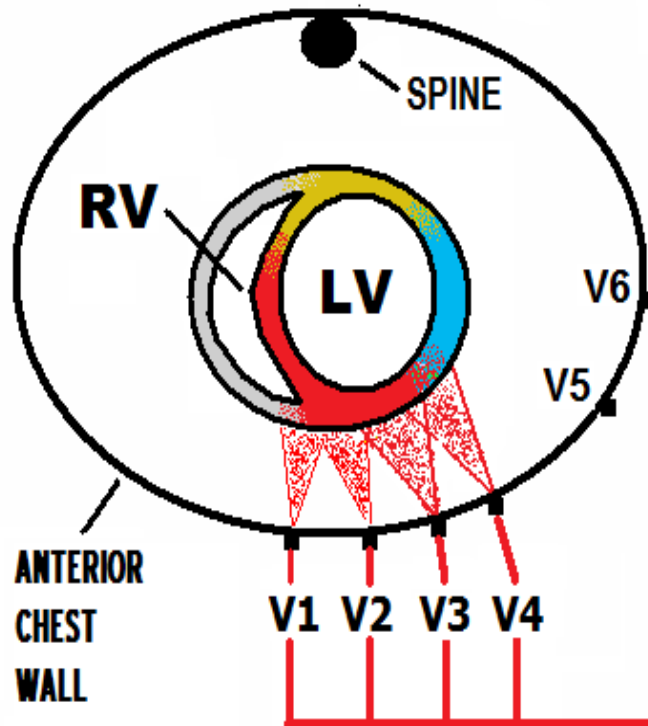


INTERPRET THE EKG, THEN:

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

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

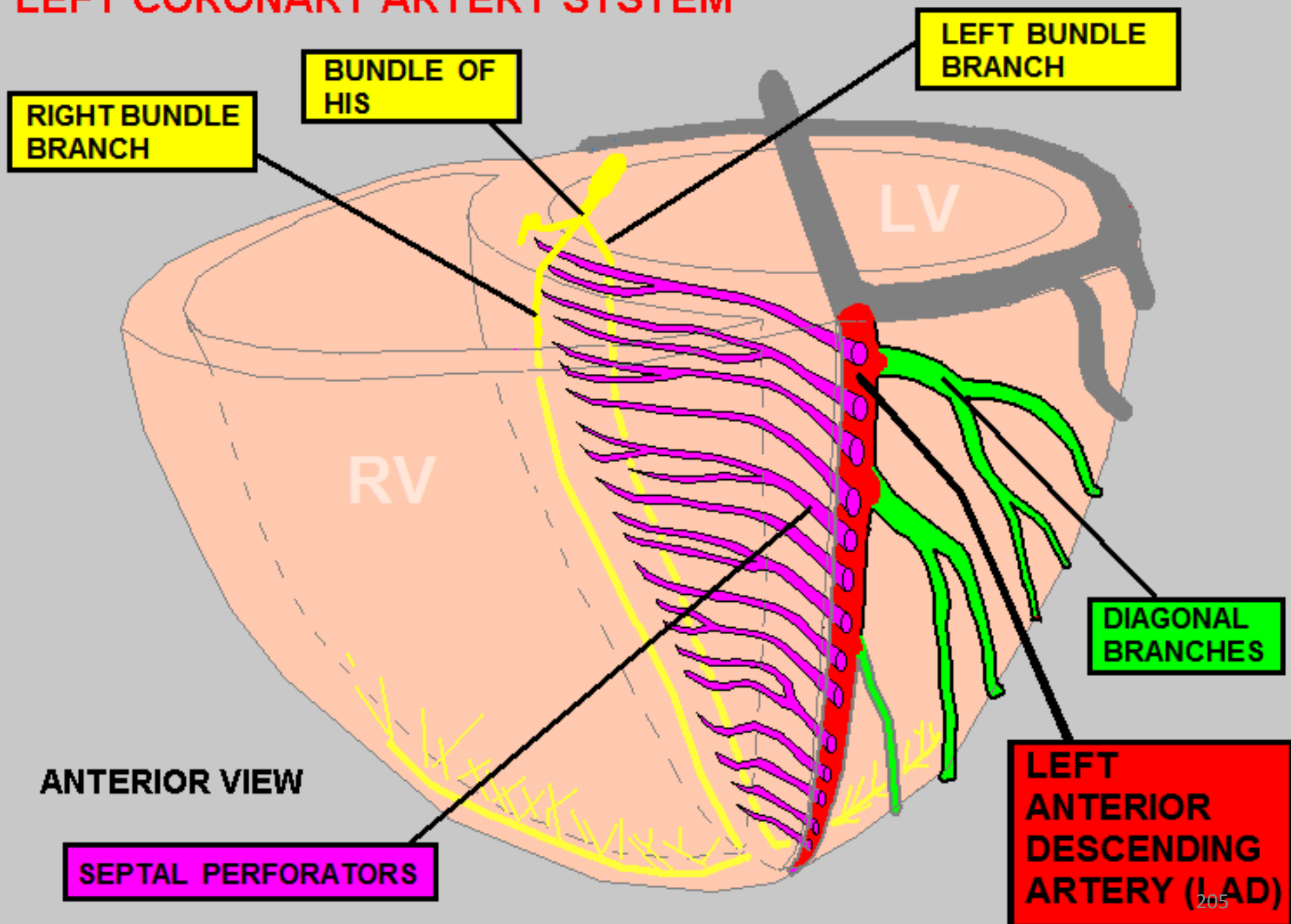
V1, V2 - ANTERIOR / SEPTAL
V3, V4 - ANTERIOR



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

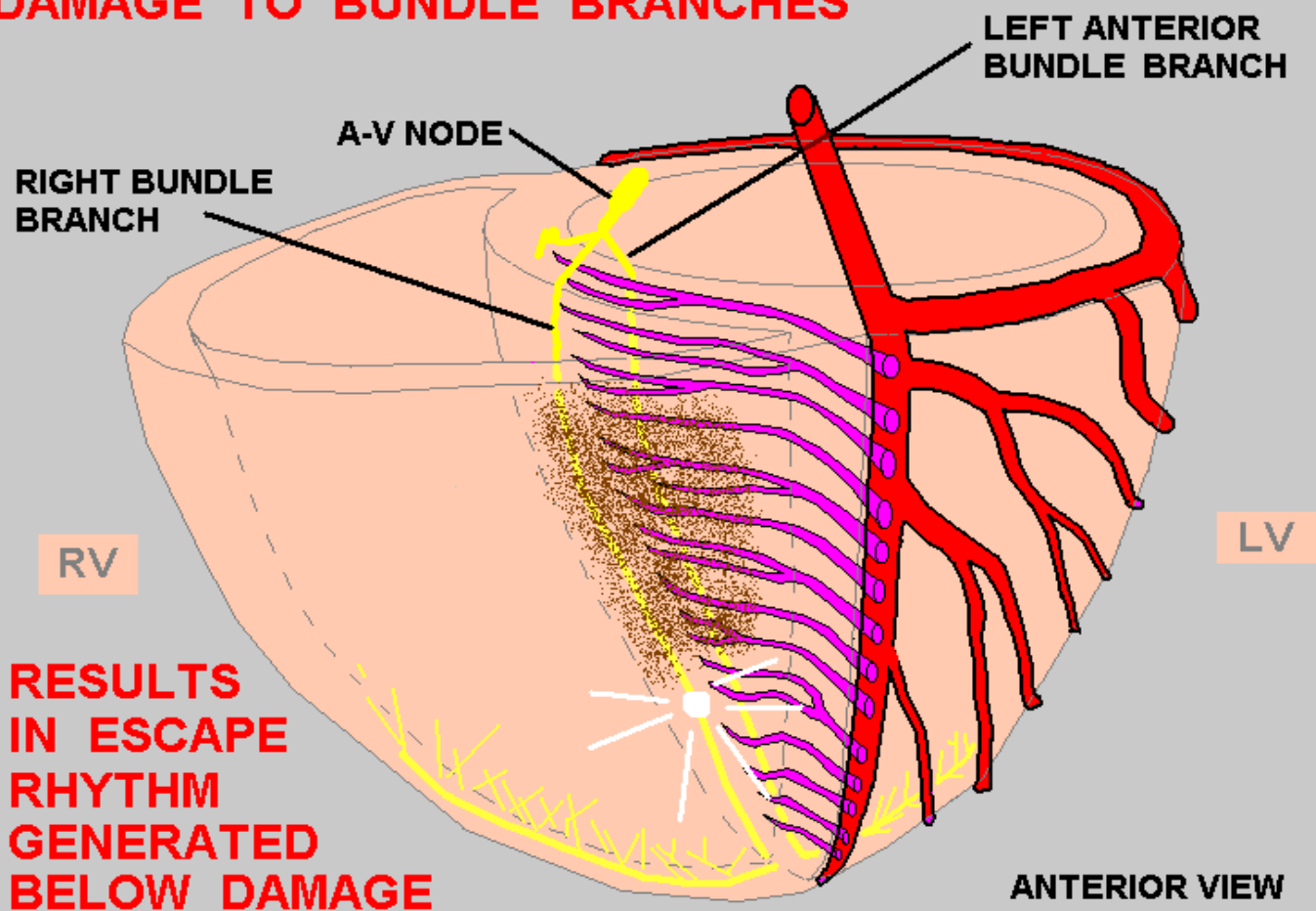
Which Coronary Artery typically Supplies the ANTERIOR WALL ? 204

LEFT CORONARY ARTERY SYSTEM





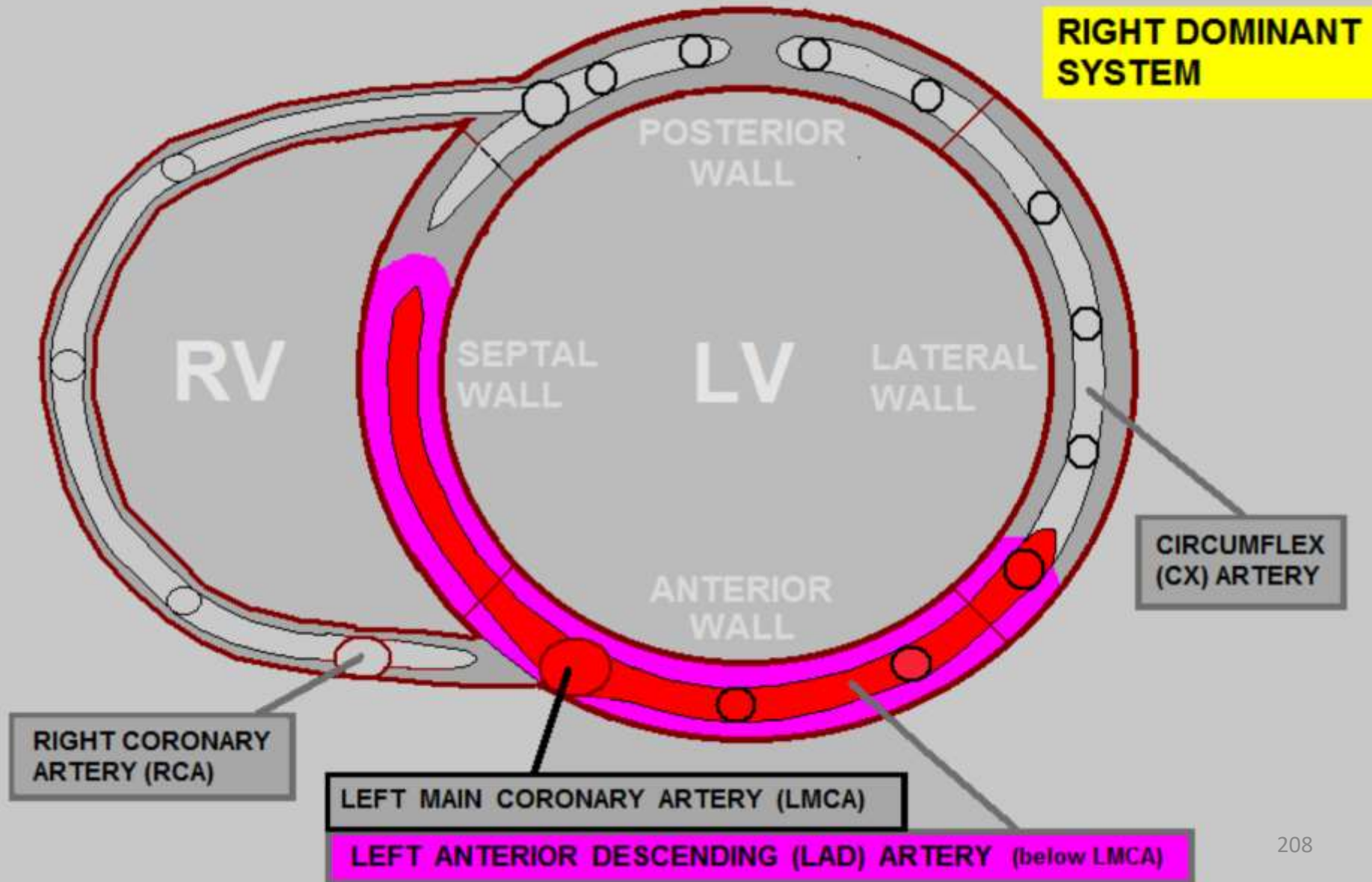
DAMAGE TO BUNDLE BRANCHES

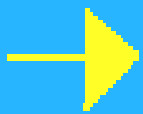


LEFT ANTERIOR DESCENDING ARTERY (LAD)



SUPPLIES APPROX. 35 - 45% of the LV MUSCLE MASS





HELPFUL HINT... *MEMORIZE THIS!*

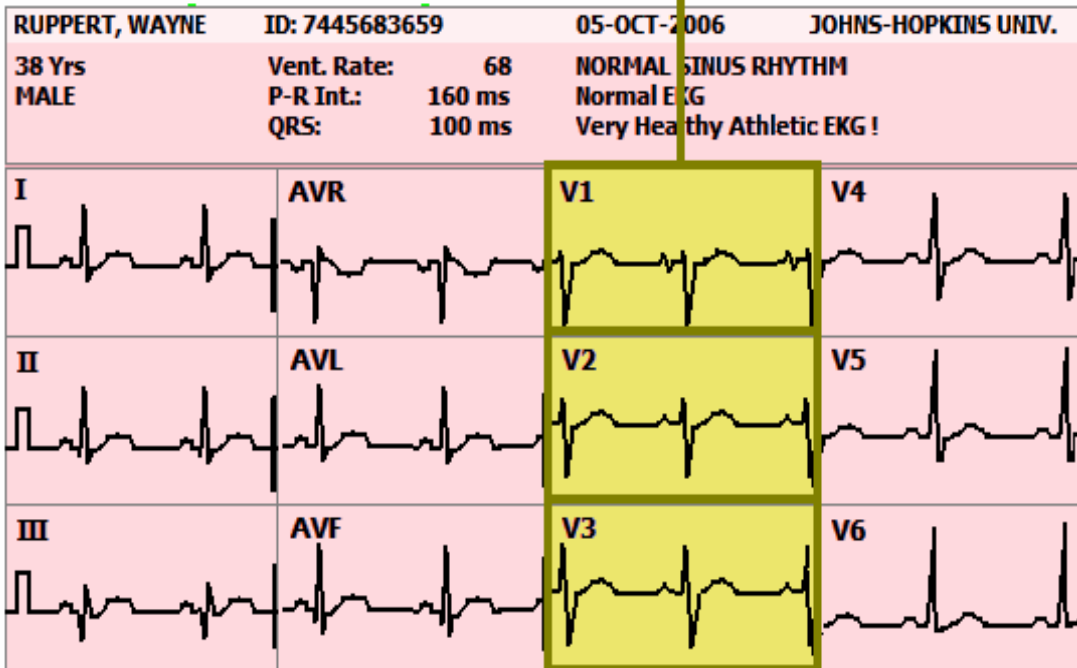
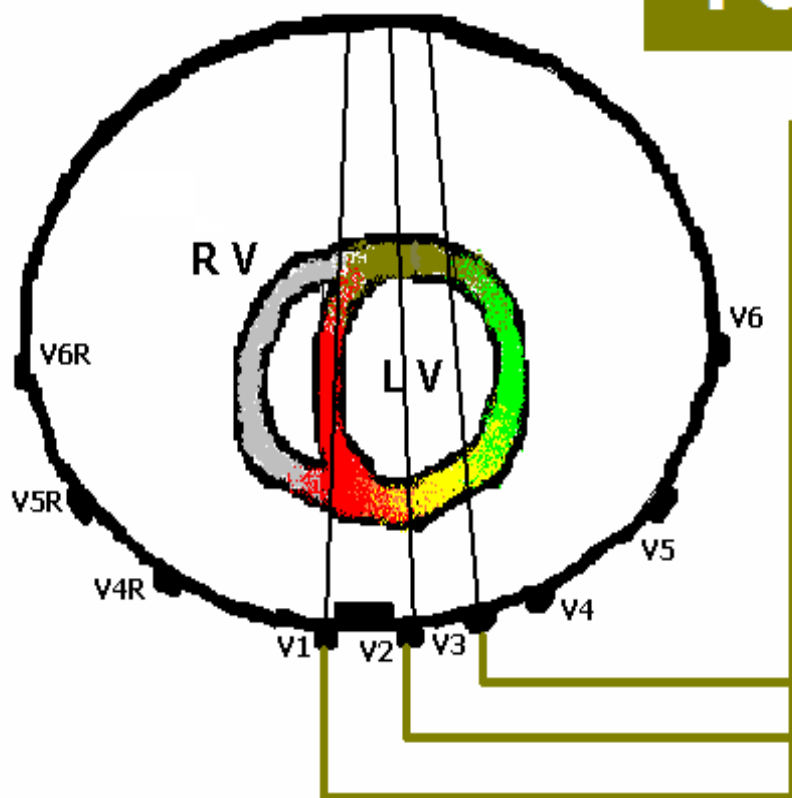


LEFT ANTERIOR DESCENDING ARTERY (LAD)

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

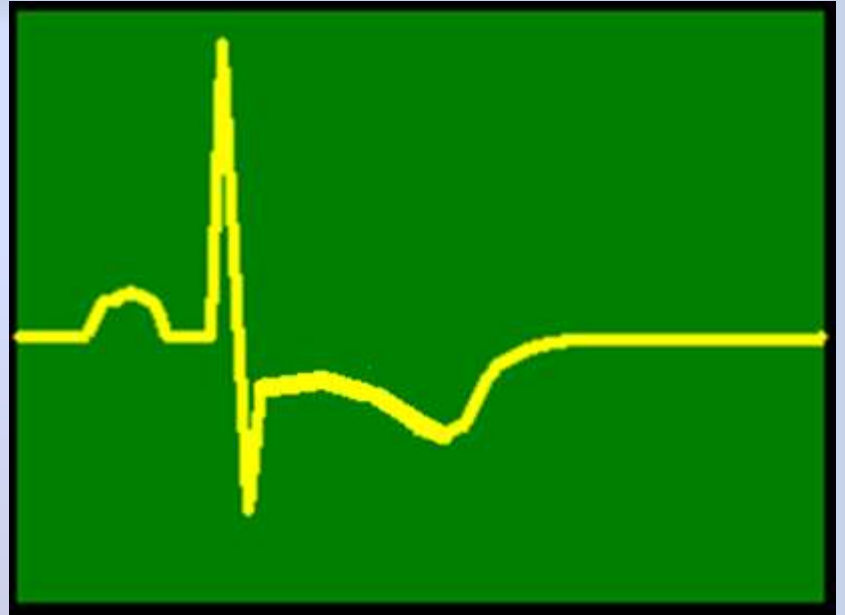
LEADS V1 - V3 *view the*

POSTERIOR WALL



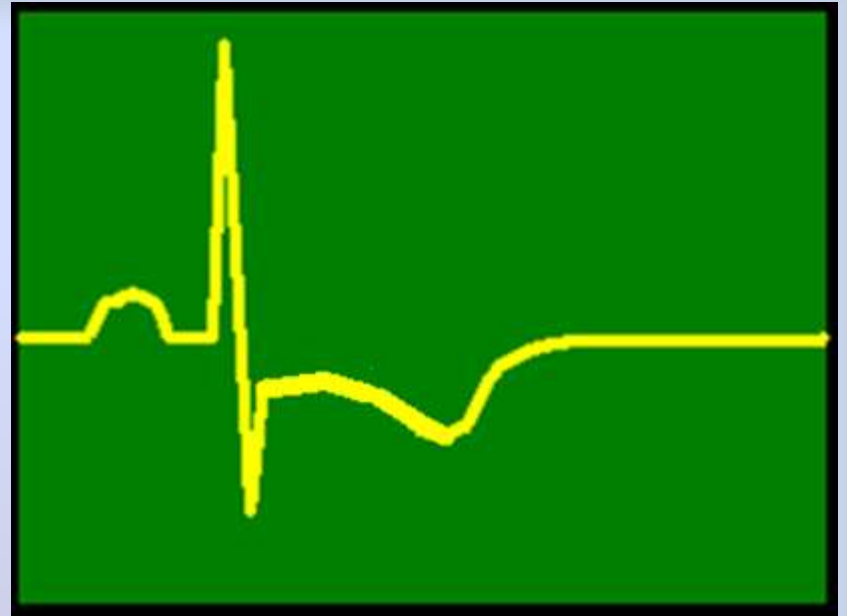
via **RECIPROCAL CHANGES.**

ST Depression in Leads V1 – V4:



- **Direct view of ISCHEMIA (anterior wall)**

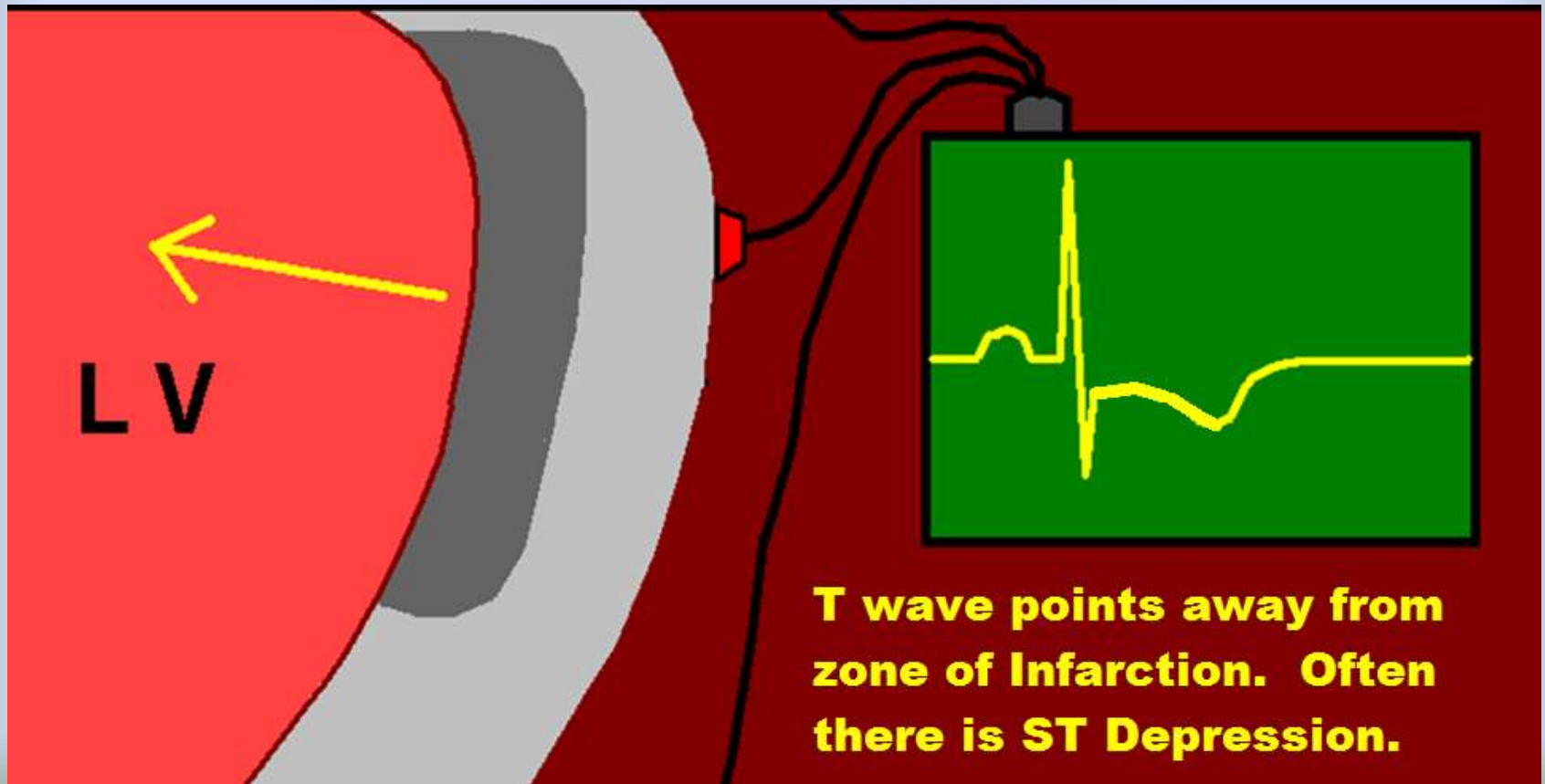
ST Depression in Leads V1 – V4:



- Direct view of ISCHEMIA (anterior wall)
- Direct view of NSTEMI (anterior wall)

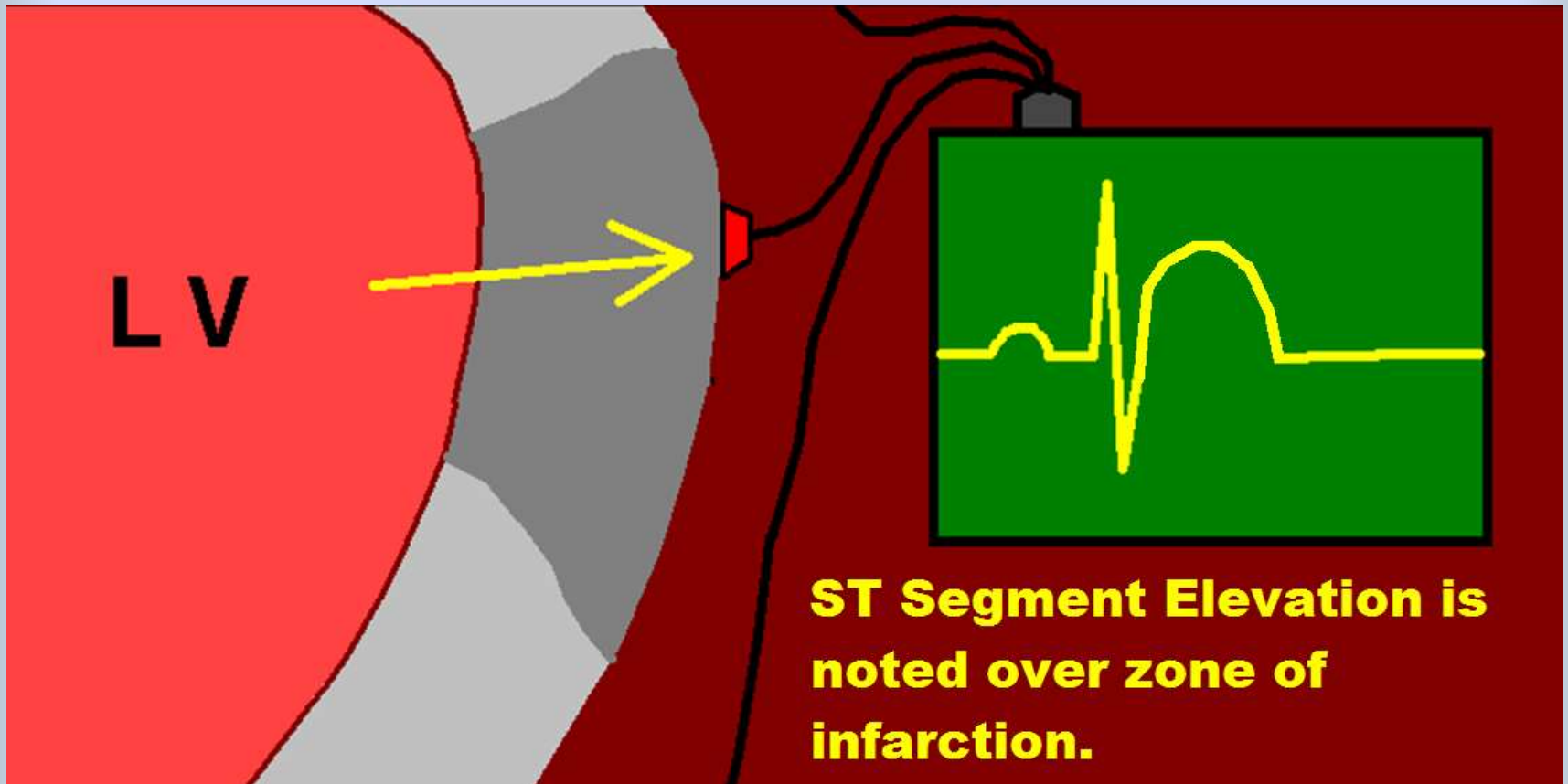
Non-STEMI (NSTEMI)

Non-ST Segment Elevation Myocardial Infarction.
“sub-endocardial MI” . . . “partial wall thickness”

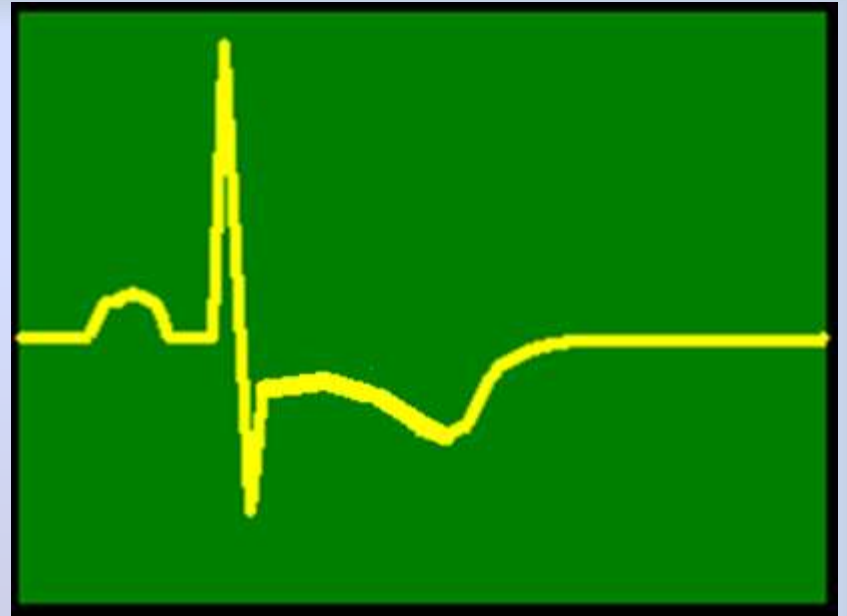


STEMI

- ST Segment Elevation Myocardial Infarction.



ST Depression in Leads V1 – V4:

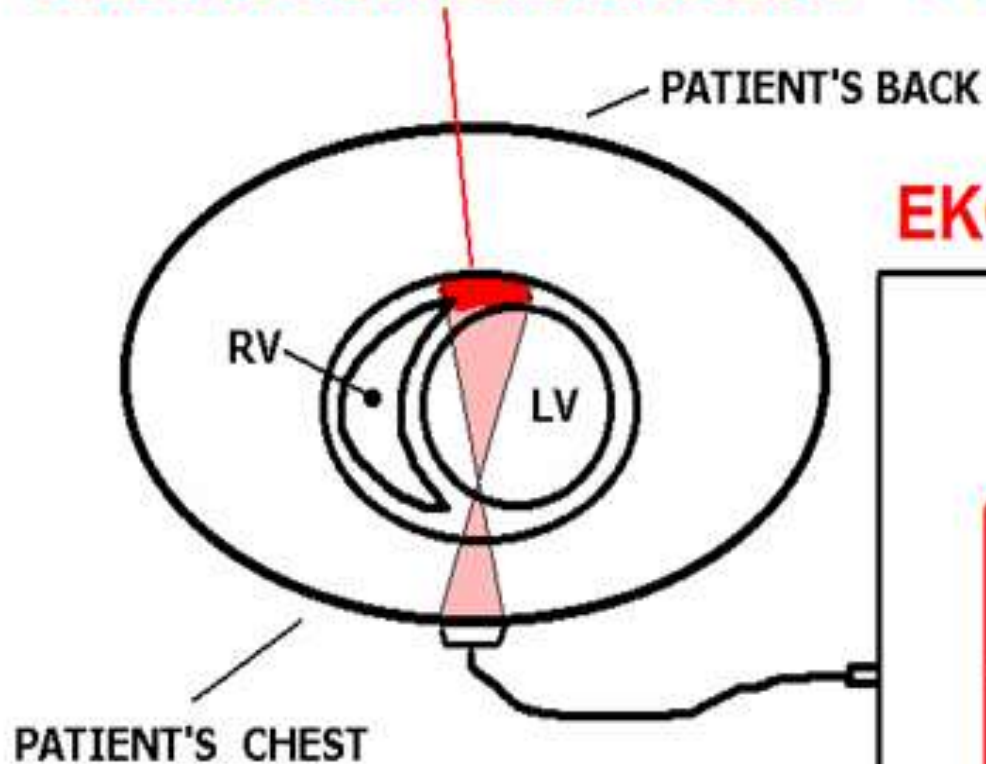


- Direct view of ISCHEMIA (anterior wall)
- Direct view of NSTEMI (anterior wall)
- Reciprocal view of STEMI (opposite side of heart - posterior wall)

HOW EKG VIEWS RECIPROCAL CHANGES

EXAMPLE:

AREA OF ACUTE INFARCTION - POSTERIOR WALL

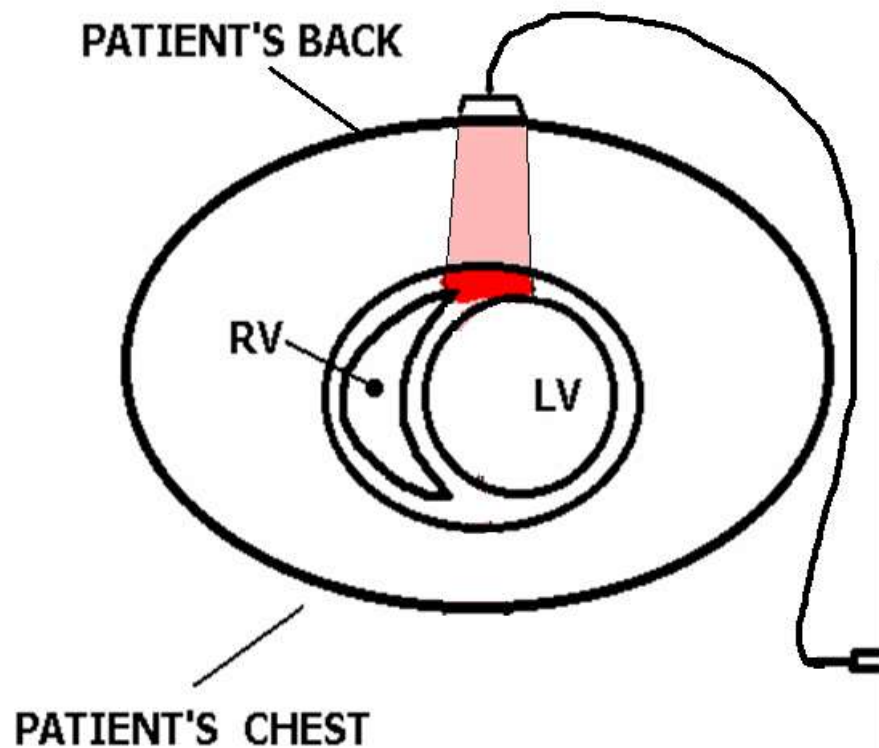


EKG sees S-T DEPRESSION

ECG LEAD V2



If we put ECG leads on the BACK of a PATIENT who is having an **ACUTE POSTERIOR WALL MI**

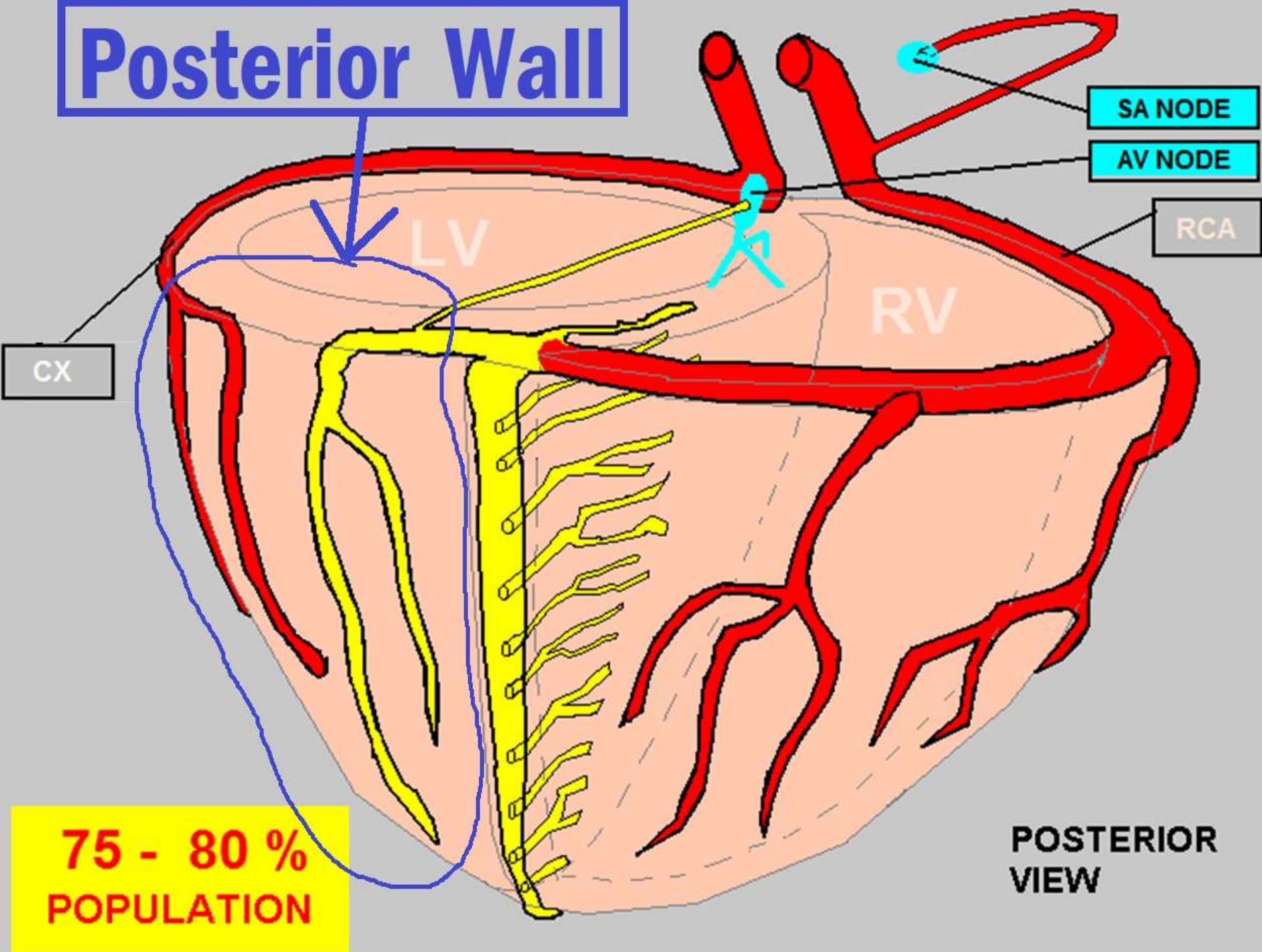


EKG sees S-T ELEVATION

ECG LEADS: V7, V8 or V9



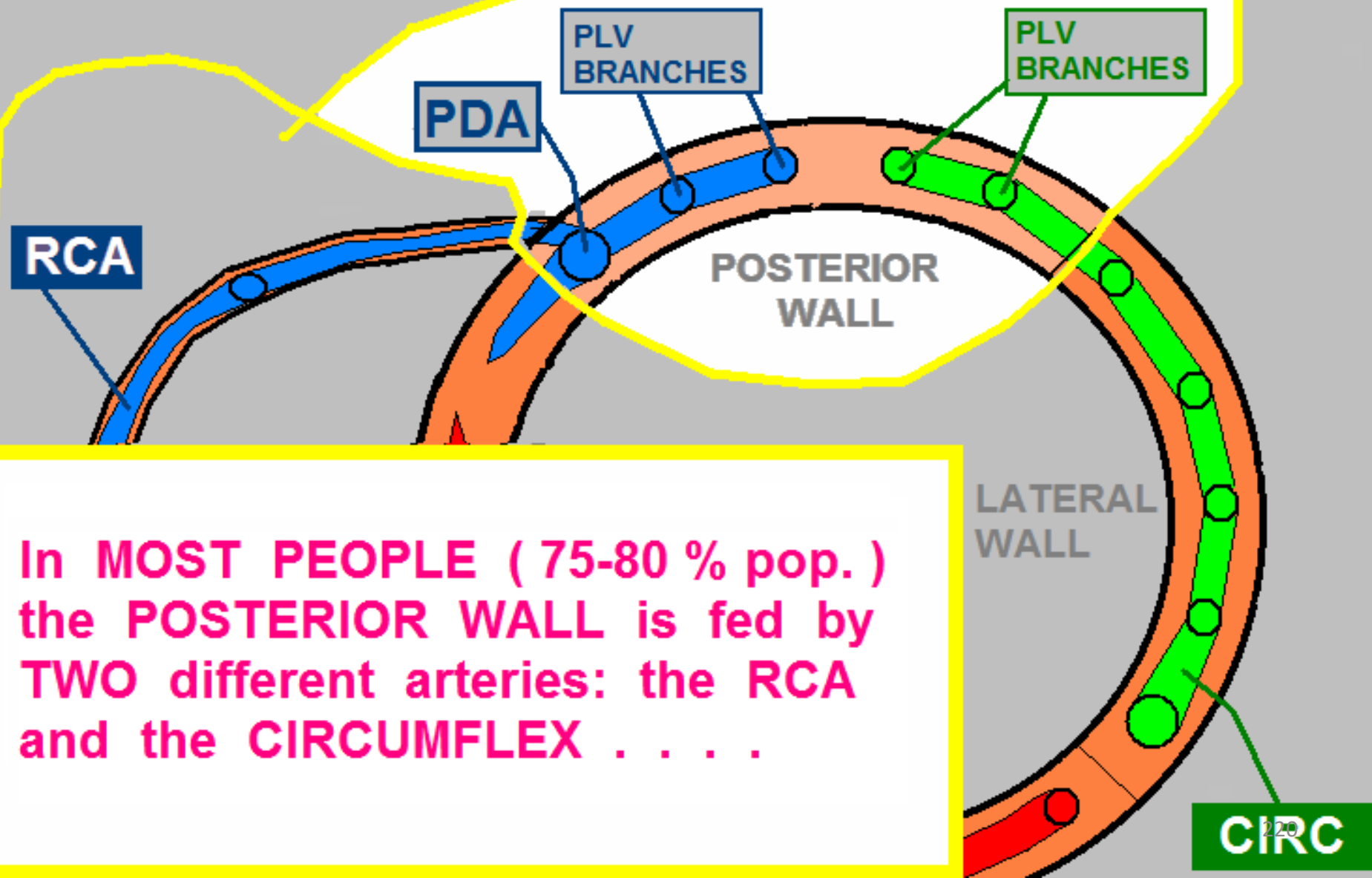
Posterior Wall



POSTERIOR WALL BLOOD SUPPLY

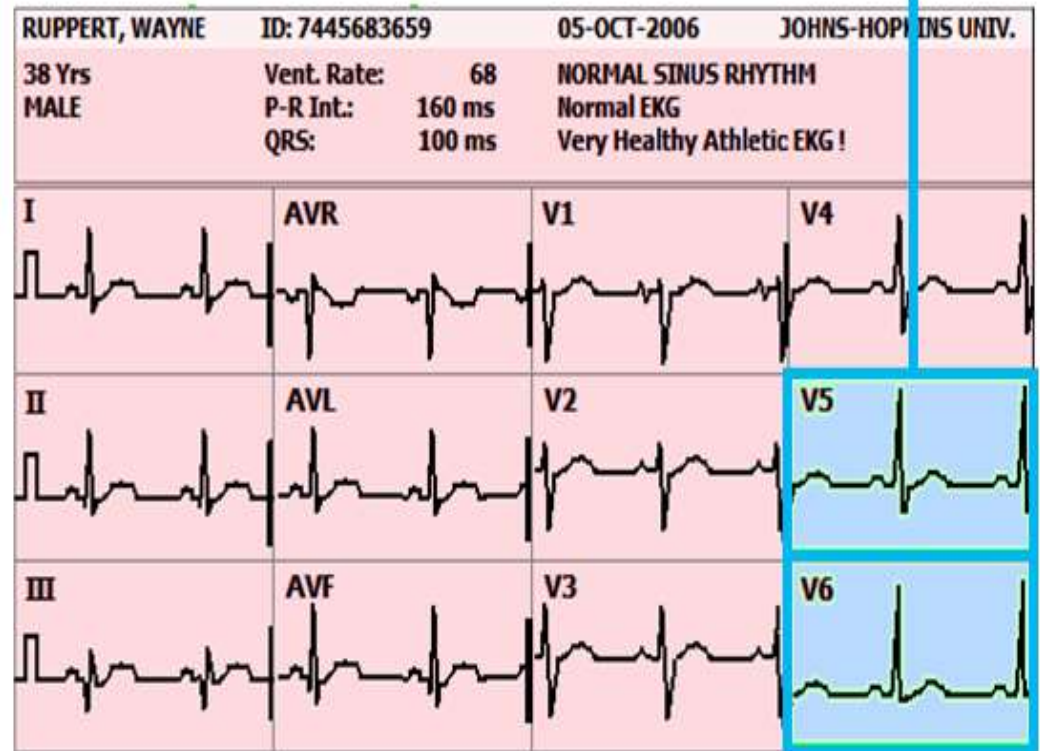
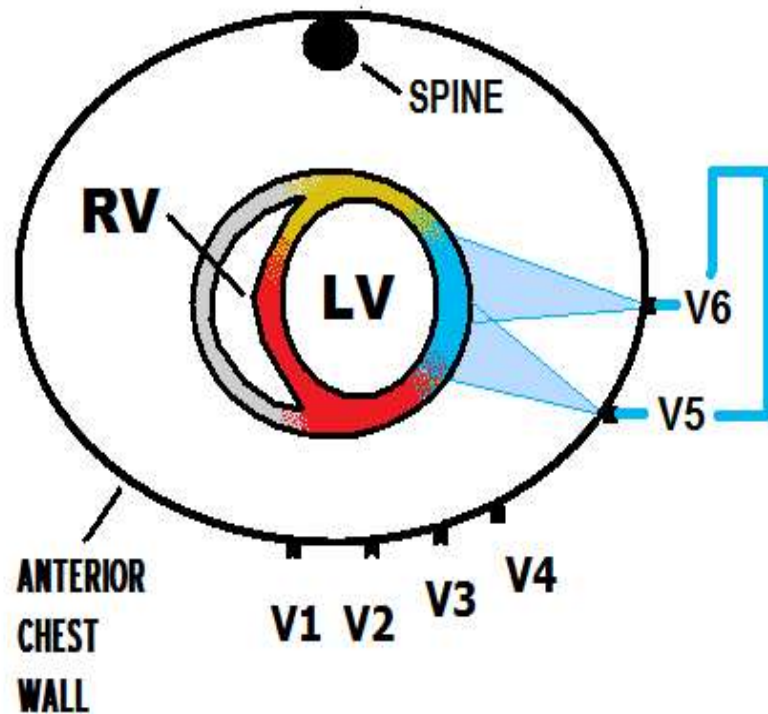
DOMINANT RCA

75-80% of POPULATION



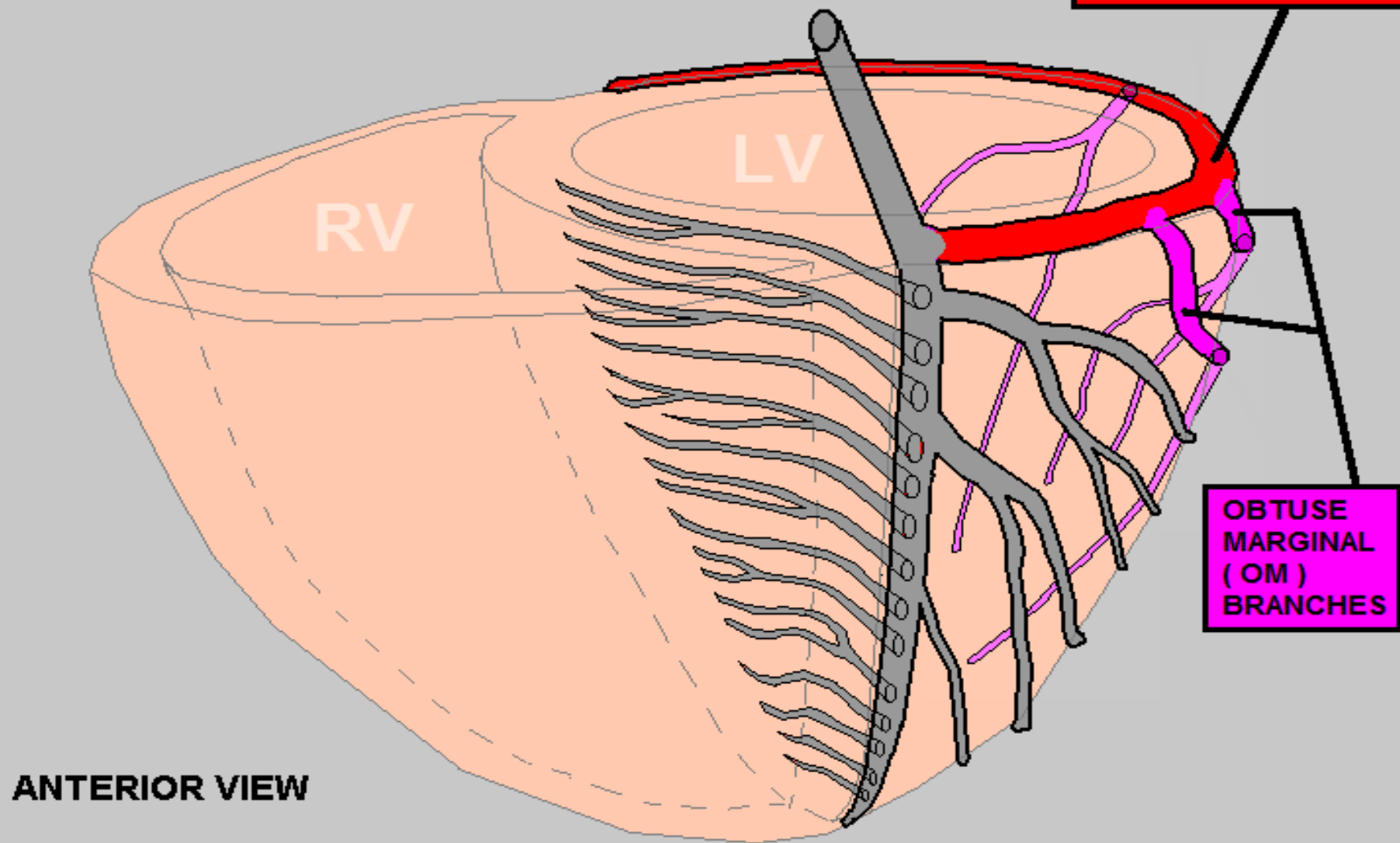
**In MOST PEOPLE (75-80 % pop.)
the POSTERIOR WALL is fed by
TWO different arteries: the RCA
and the CIRCUMFLEX**

V5 - V6 VIEW THE LATERAL WALL of the LEFT VENTRICLE



Which Coronary Artery typically Supplies the LATERAL WALL ?

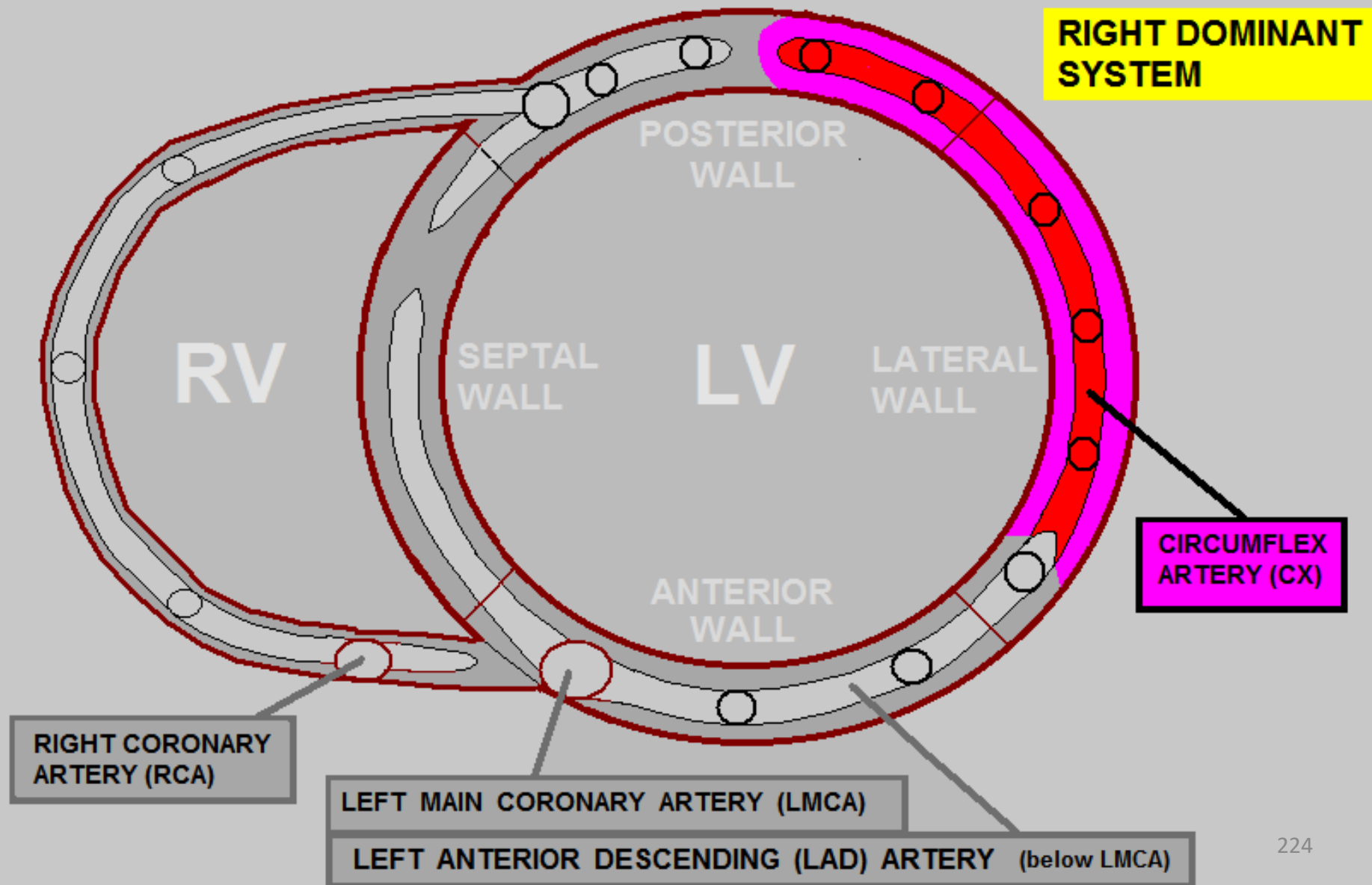
LEFT CORONARY ARTERY SYSTEM



CIRCUMFLEX ARTERY (CX) DISTRIBUTION



SUPPLIES 20 - 30 % of the LV MUSCLE MASS





HELPFUL HINT... *MEMORIZE THIS!*

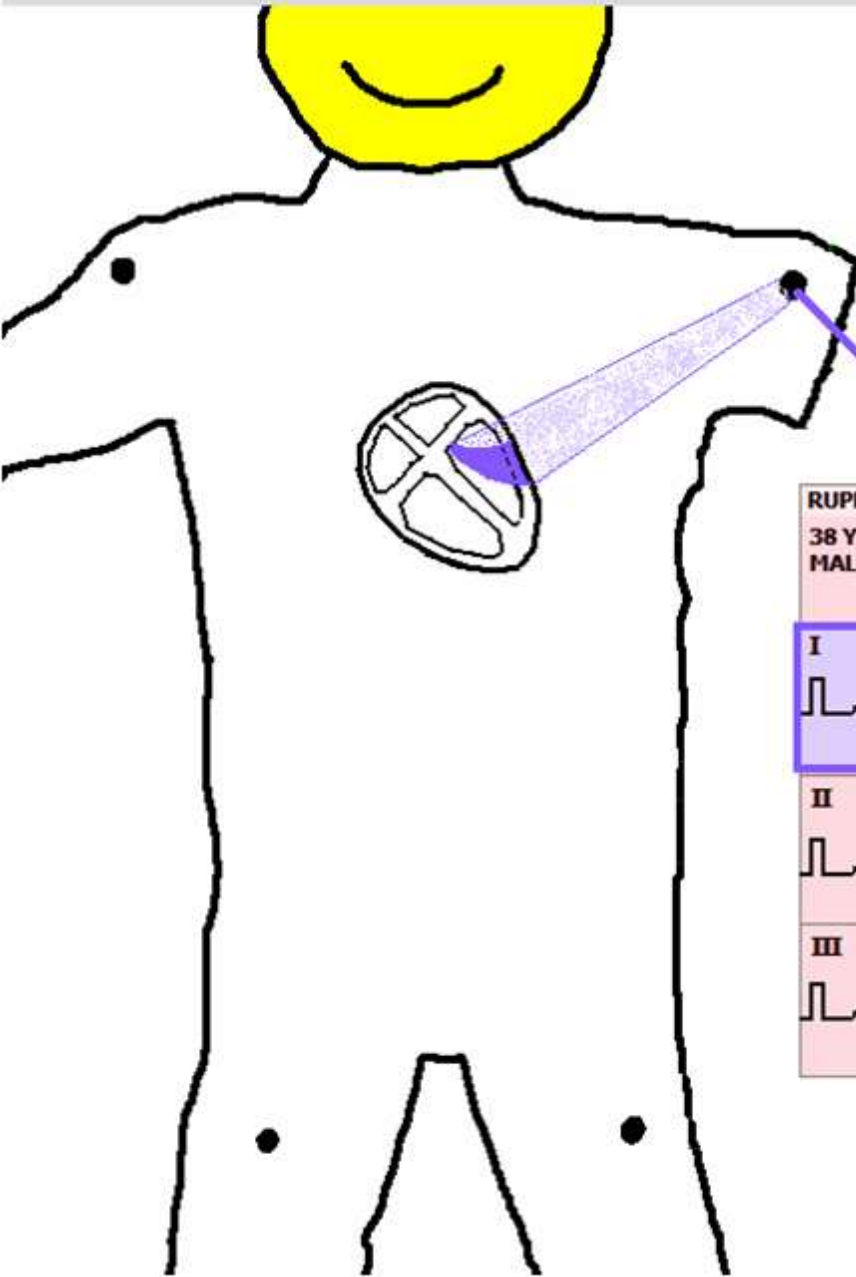


CIRCUMFLEX ARTERY (CX)

RIGHT DOMINANT
SYSTEMS

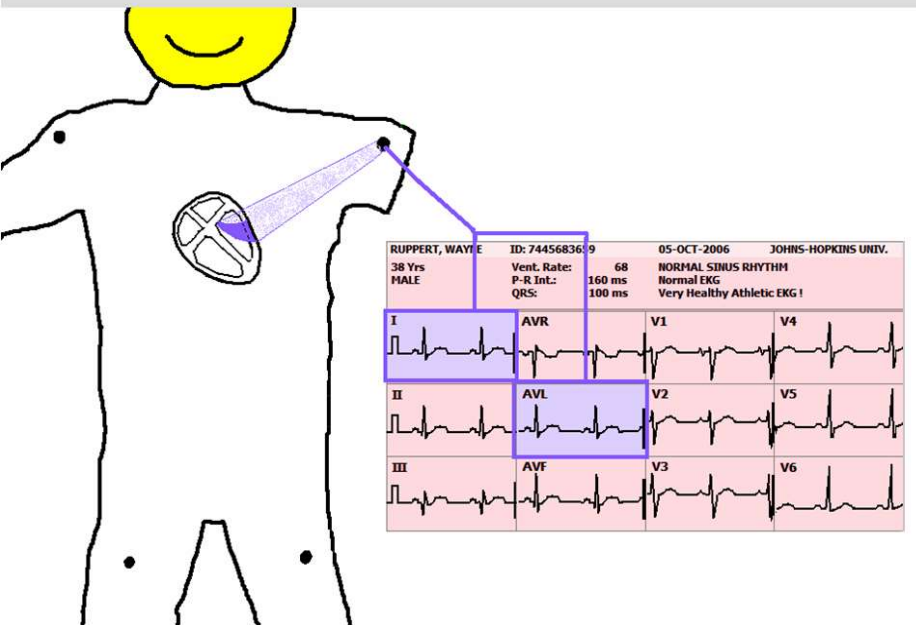
- ▶ LEFT ATRIUM
- ▶ SINUS NODE (5% of the population)
- ▶ LEFT VENTRICLE: 20 - 30 % of muscle mass
 - LATERAL WALL
 - up to 1/2 of POSTERIOR WALL

LEADS I and aVL VIEW the LATERAL - ANTERIOR WALL

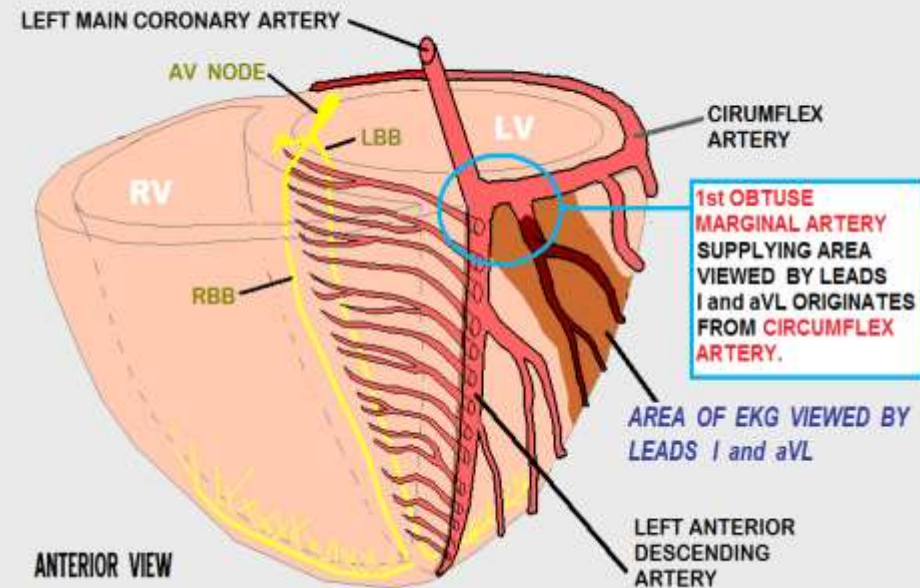


RUPPERT, WAYNE		ID: 744568369	05-OCT-2006	JOHNS-HOPKINS UNIV.
38 Yrs MALE		Vent. Rate: 68 P-R Int.: 160 ms QRS: 100 ms	NORMAL SINUS RHYTHM Normal EKG Very Healthy Athletic EKG !	
I	AVR	V1	V4	
II	AVL	V2	V5	
III	AVF	V3	V6	

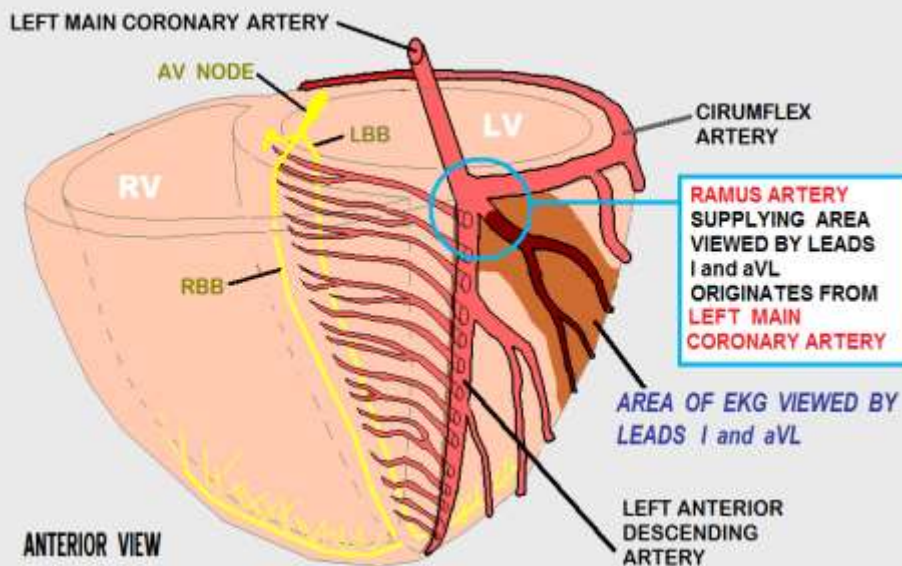
LEADS I and aVL VIEW the LATERAL - ANTERIOR WALL



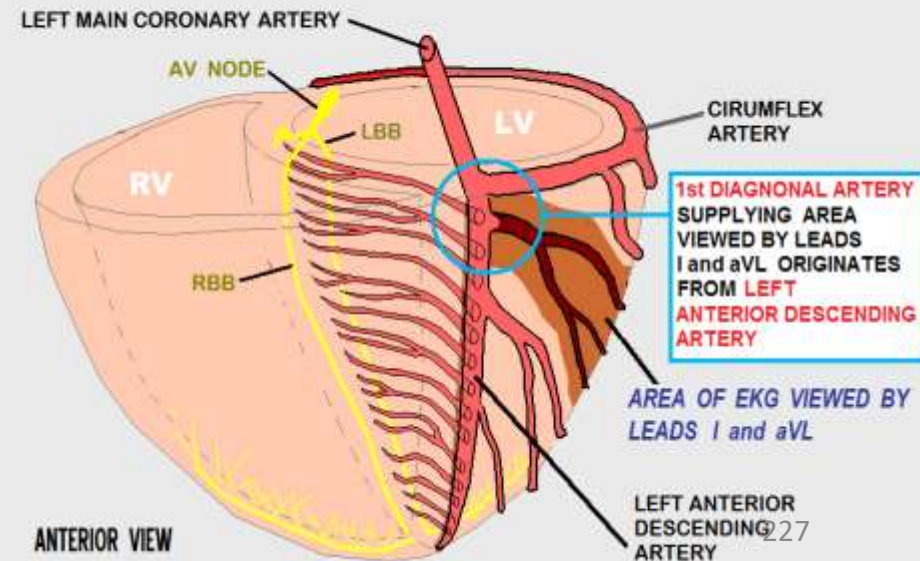
OCCCLUSION of OBTUSE MARGINAL ARTERY



OCCCLUSION of RAMUS ARTERY



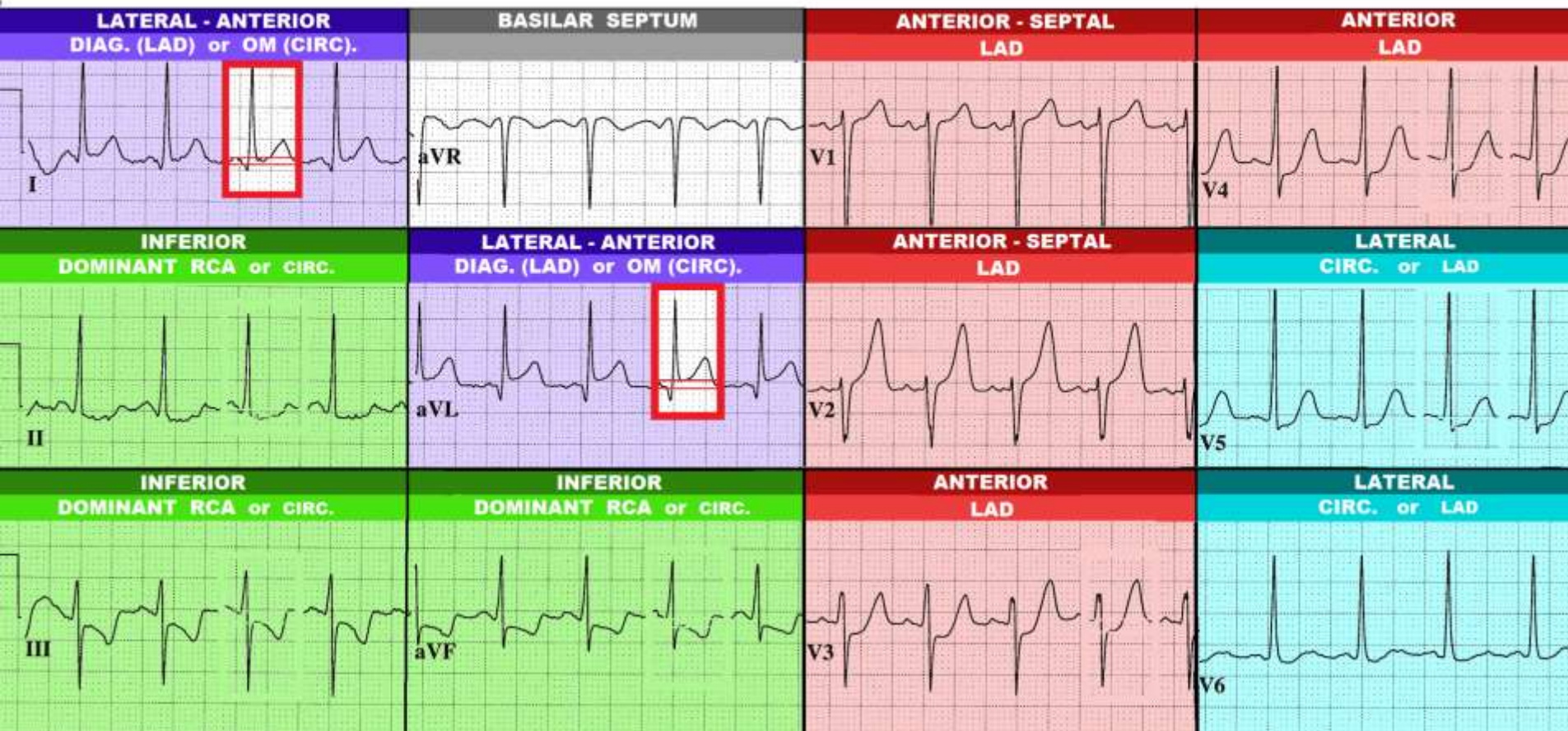
OCCCLUSION of DIAGONAL ARTERY



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

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

ST SEGMENT ELEVATION

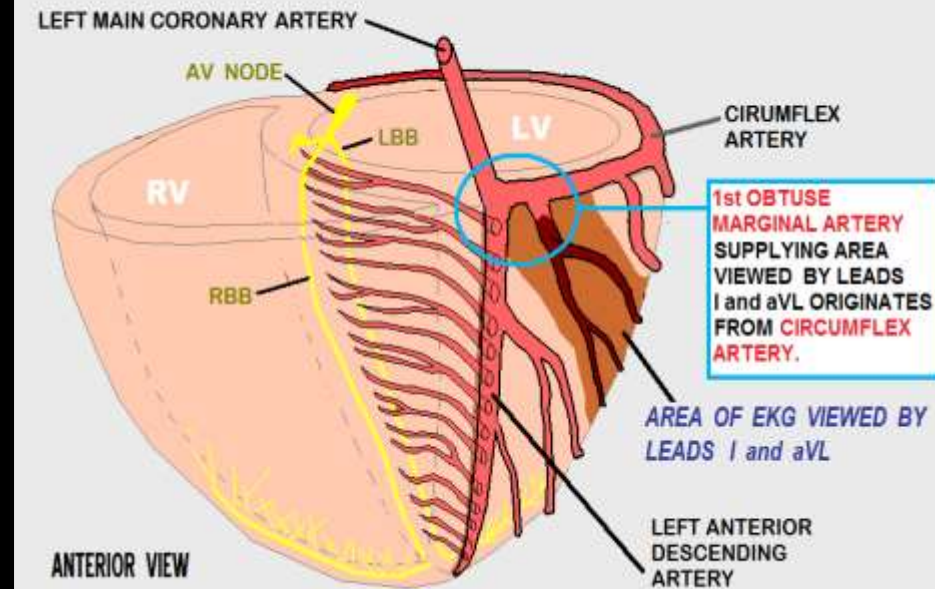


ST Segment elevation ONLY in Leads I and aVL

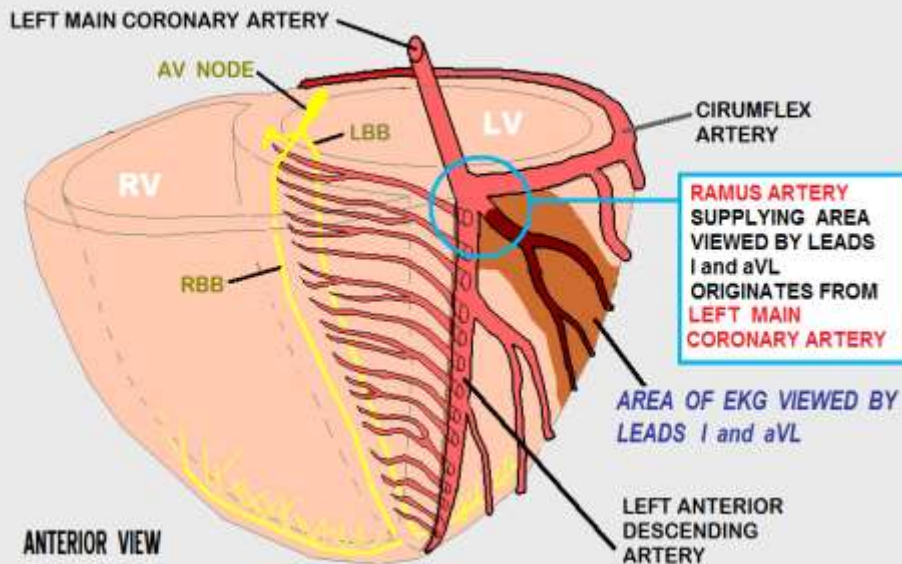
ST Elevation isolated to Leads I and aVL - usually indicates the "Culprit Artery" is most likely One of the following:

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

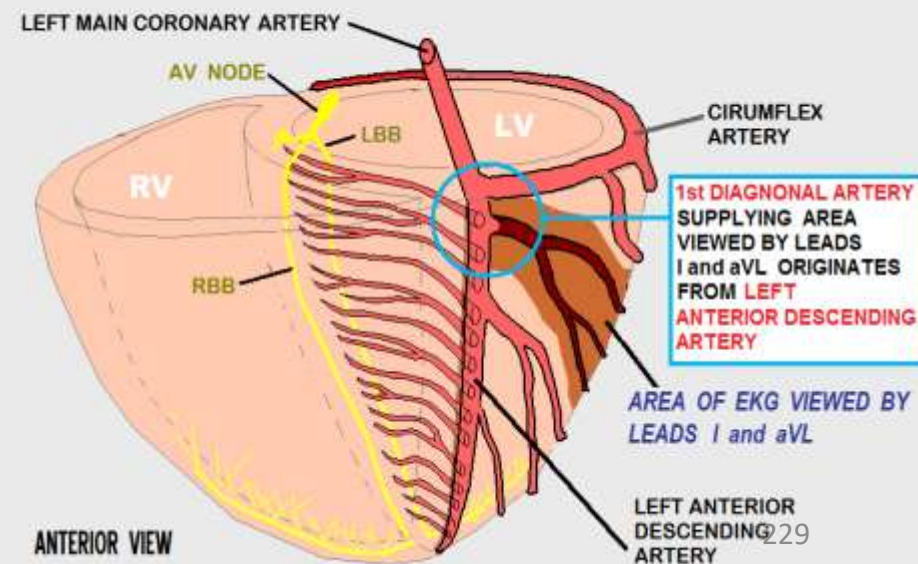
OCCCLUSION of OBTUSE MARGINAL ARTERY



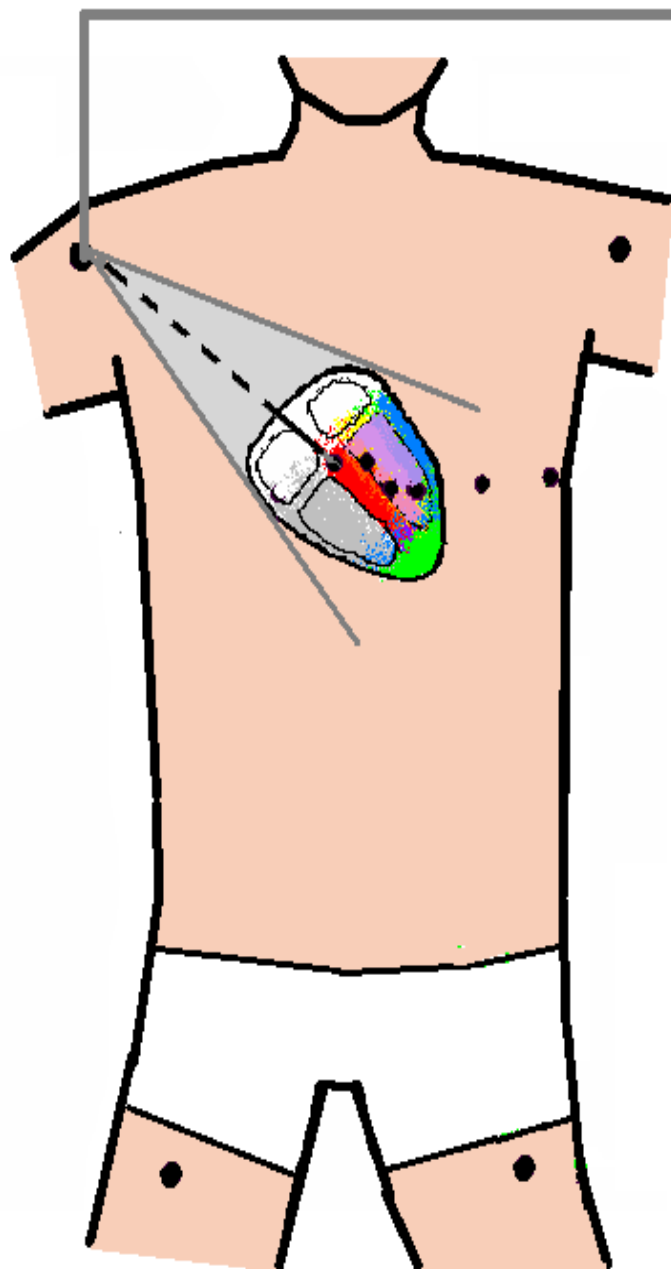
OCCCLUSION of RAMUS ARTERY



OCCCLUSION of DIAGONAL ARTERY



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

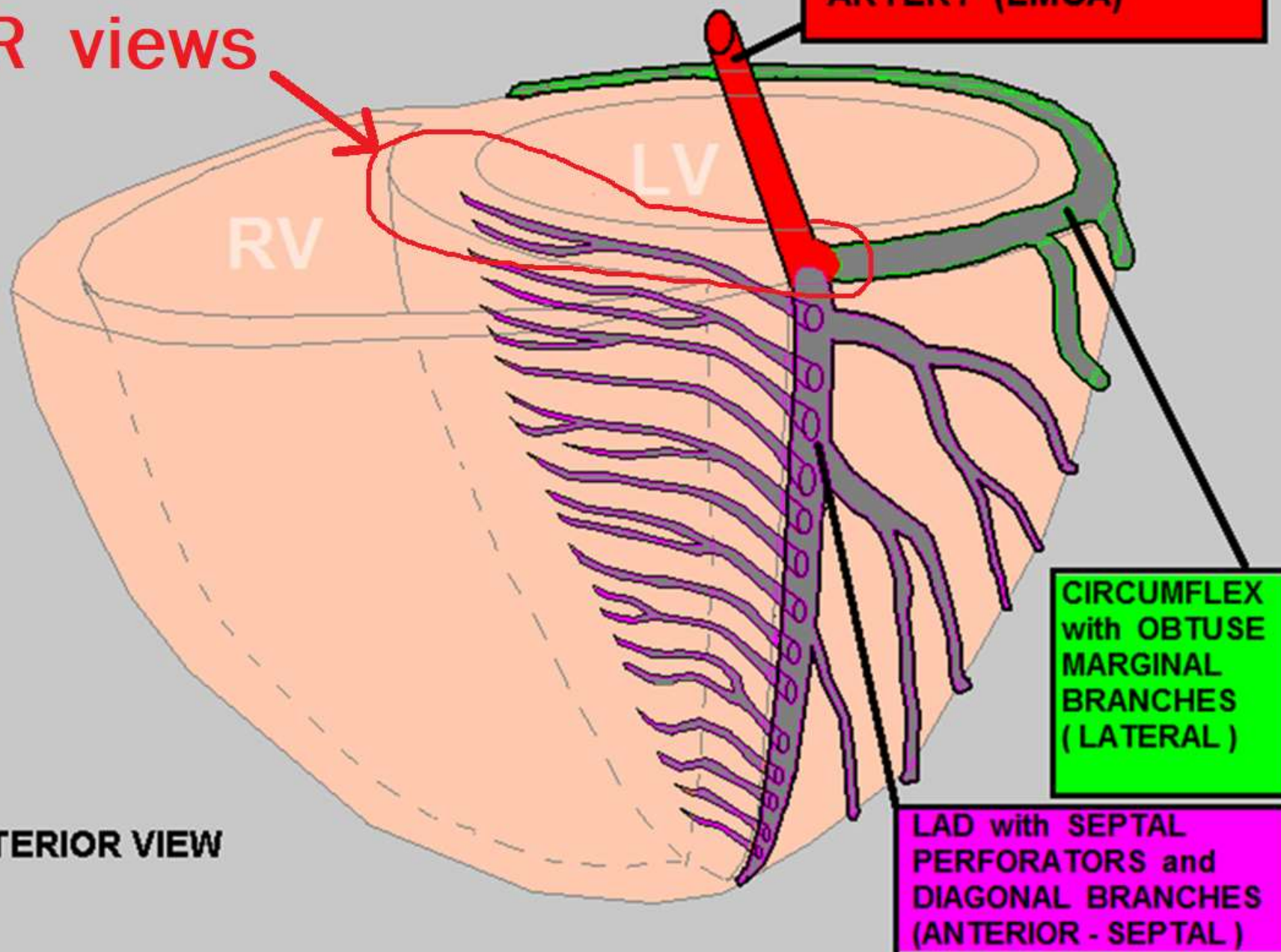


RUPPERT, WAYNE		ID: 7445683	59	05-OCT-2006	JOHNS-HOPKINS UNIV.
38 Yrs		Vent. Rate:	68	NORMAL SINUS RHYTHM	
MALE		P-R Int.:	160 ms	Normal EKG	
		QRS:	100 ms	Very Healthy Athletic EKG !	
I	AVR	V1	V4		
II	AVL	V2	V5		
III	AVF	V3	V6		

LEFT CORONARY ARTERY SYSTEM

AVR views

ANTERIOR VIEW



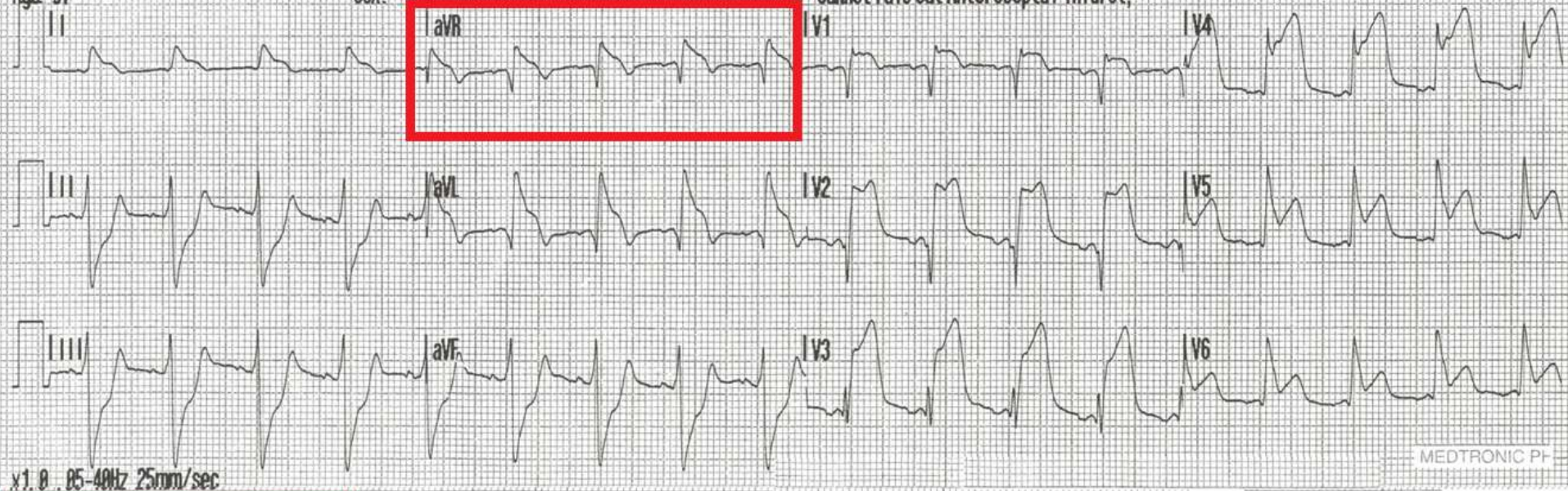
When LEAD AVR shows ST
Elevation:

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

Name: 12-Lead 4 HR 107 bpm
 ID: 06 Oct 07 12:44:13
 Patient ID: PR 0.154s QRS 0.102s
 Incident: QT/QTc 0.332s/0.443s
 Age 37 Sex: P-QRS-T Axes 89° -62° 44°

- *** ACUTE MI SUSPECTED ***
- Abnormal ECG **Unconfirmed**
- Sinus tachycardia
- Left anterior fascicular block
- Cannot rule out Anteroseptal infarct,

**ACUTE STEMI caused by
 LEFT MAIN CORONARY
 ARTERY OCCLUSION**

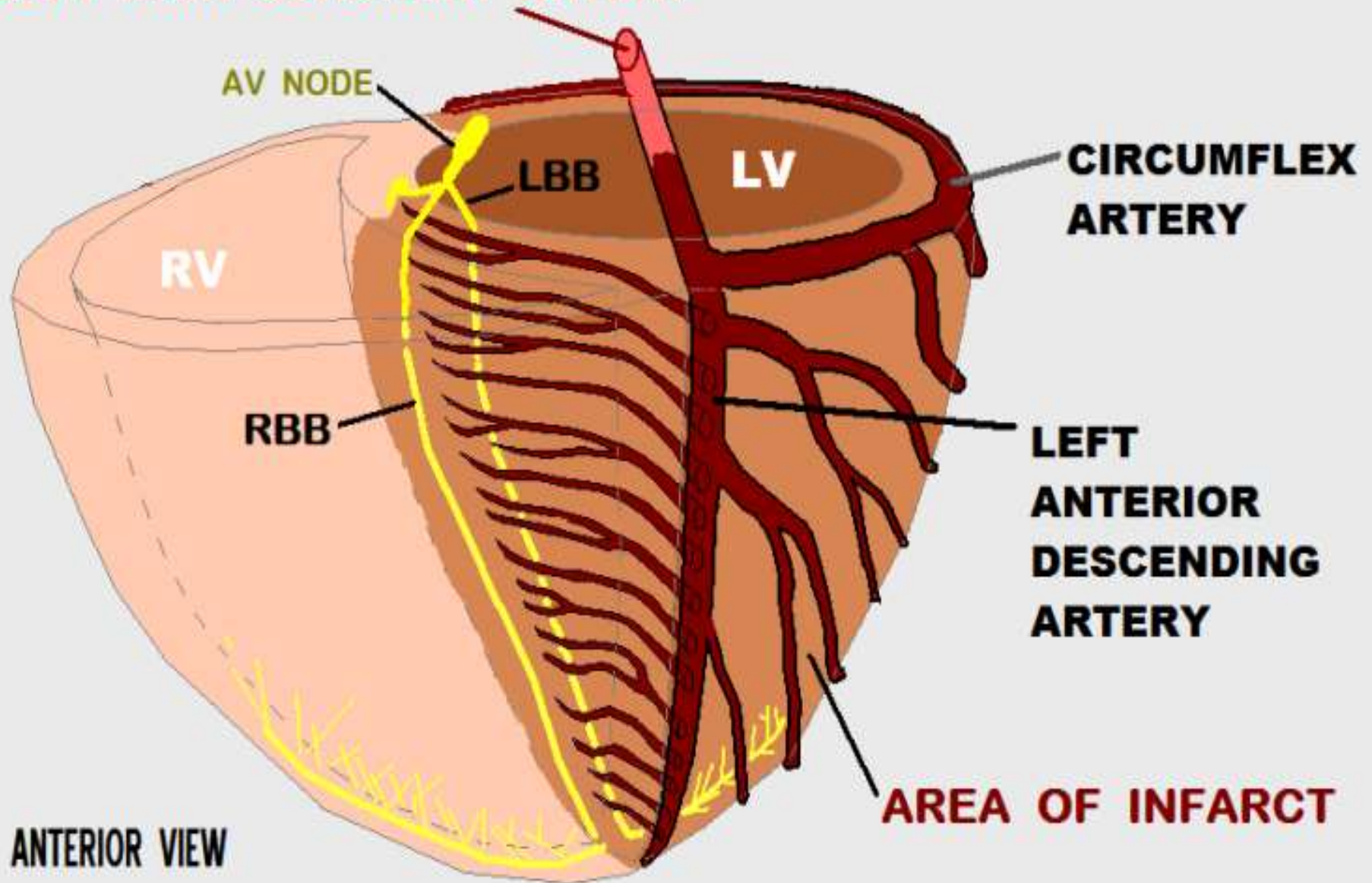


**ECG CLUES of ACUTE
 STEMI caused by
 LEFT MAIN CORONARY
 ARTERY OCCLUSION:**

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

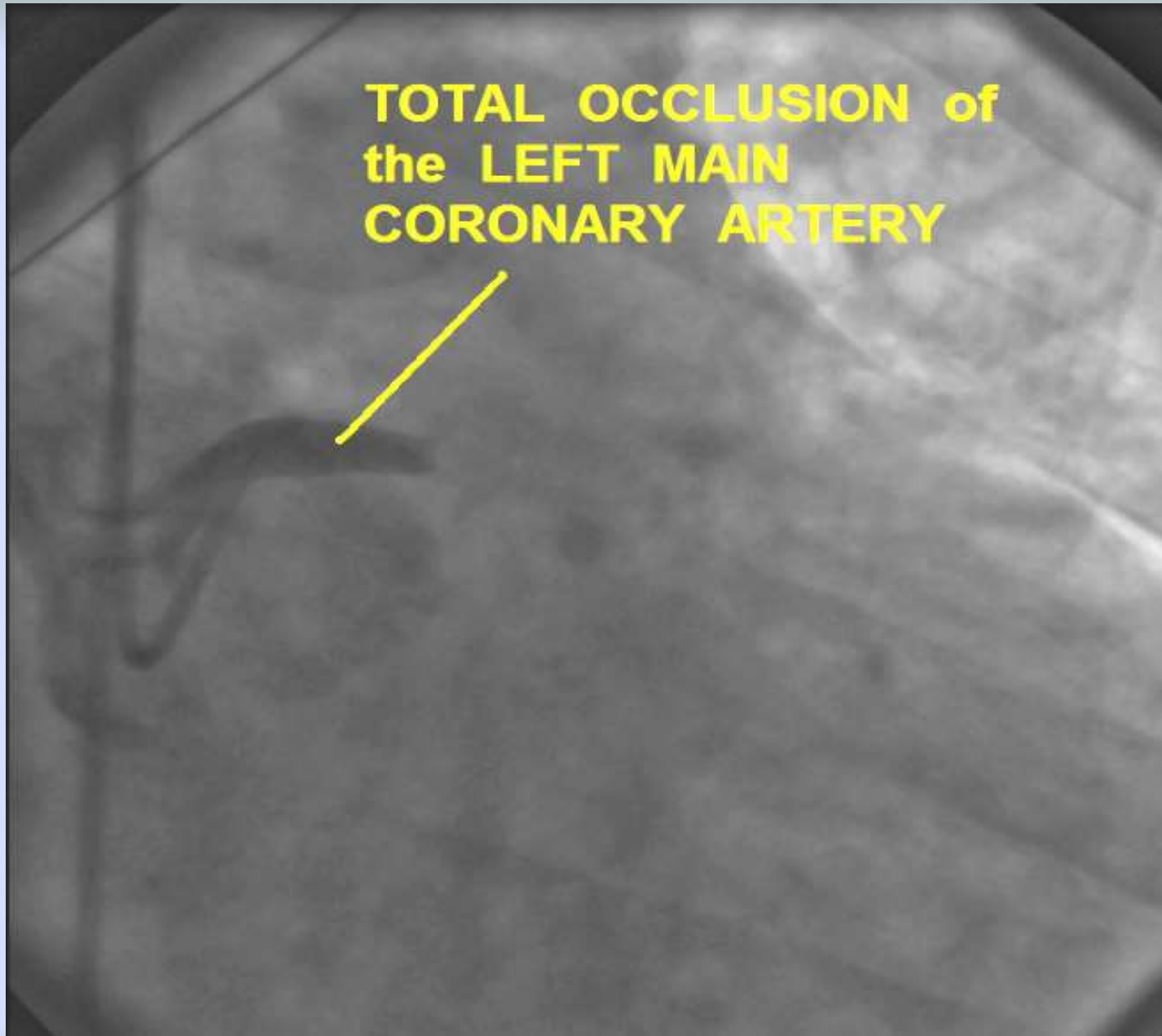
OCCLUSION of the LEFT MAIN CORONARY ARTERY

LEFT MAIN CORONARY ARTERY



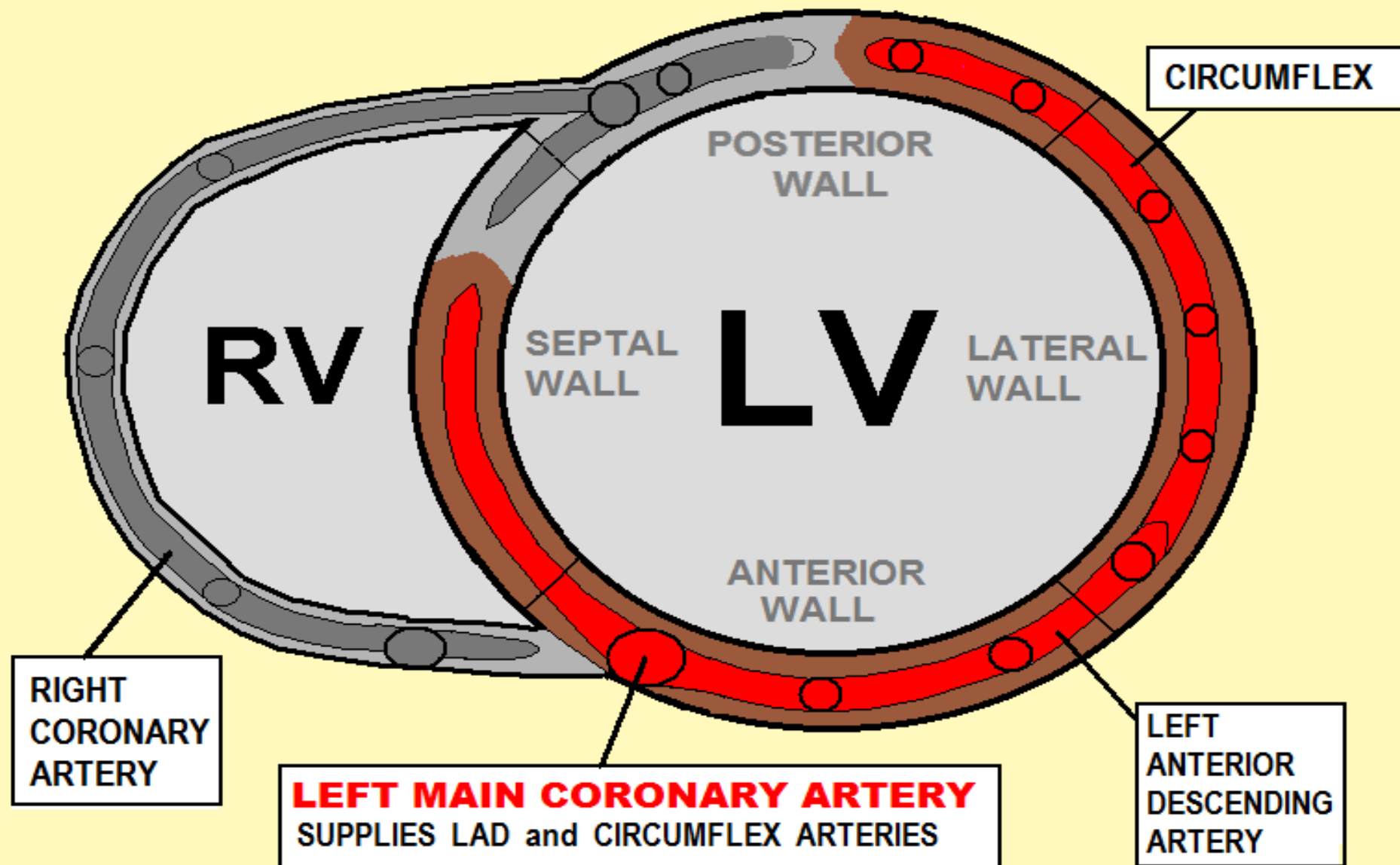
ANTERIOR VIEW

**TOTAL OCCLUSION of
the LEFT MAIN
CORONARY ARTERY**



The LEFT MAIN CORONARY ARTERY

SUPPLIES 75 - 100 % of the LEFT VENTRICULAR MUSCLE MASS



When LEAD AVR shows ST
Elevation:

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

When LEAD AVR shows ST Elevation:

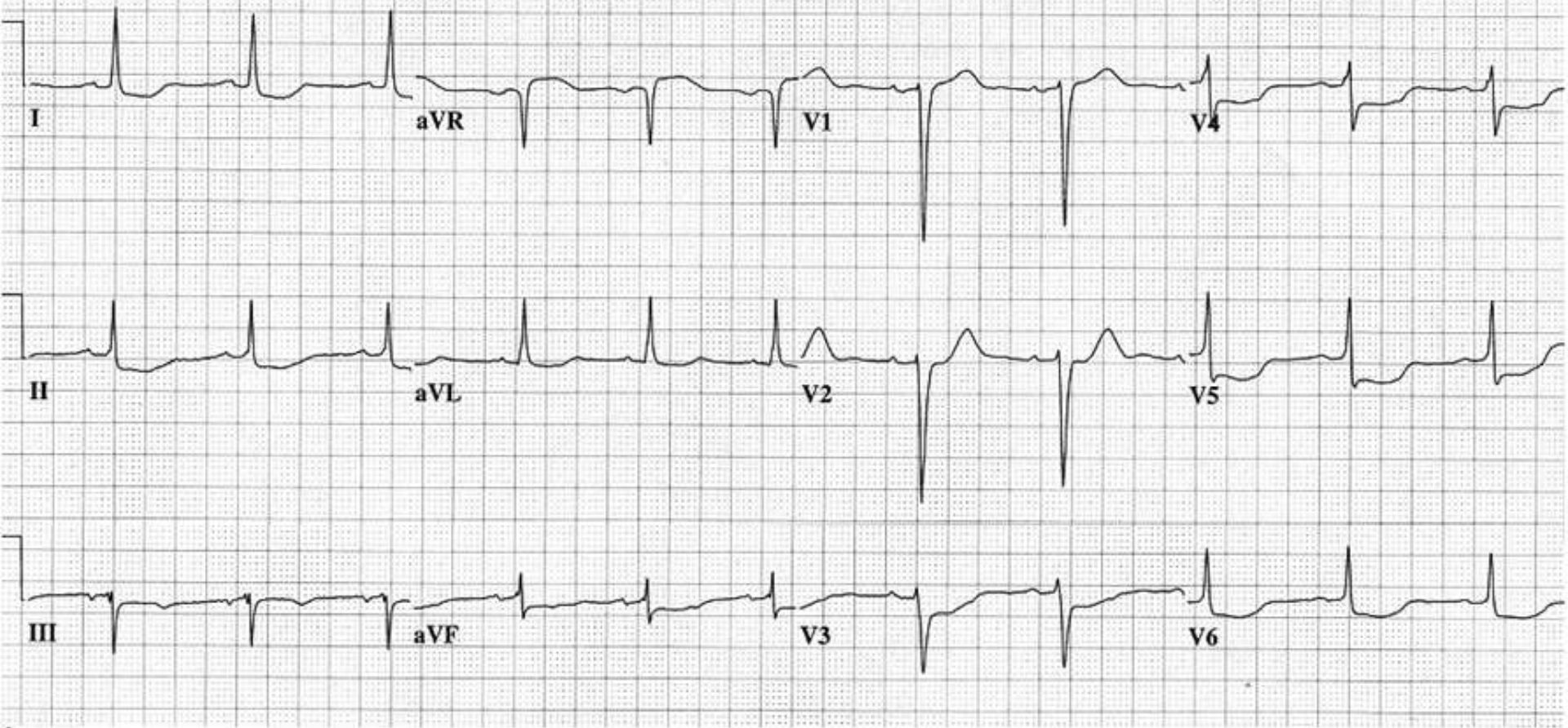
- **STEMI:** consider occlusion of the Left Main Coronary Artery.
- **NSTEMI and Unstable Angina** consider LMCA Occlusion – or **TRIPLE VESSEL DISEASE**

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

67 yr
Female Hispanic
Room:S7
Loc:3 Option:23

Vent. rate	67	BPM
PR interval	188	ms
QRS duration	106	ms
QT/QTc	458/483	ms
P-R-T axes	27 -3 -111	

OS:



67 yr
Female Hispanic
Room: S7
Loc: 3 Option: 23

Vent. rate 67 BPM
PR interval 188 ms
QRS duration 106 ms
QT/QTc 458/483 ms
P-R-T axes 27 -3 -111

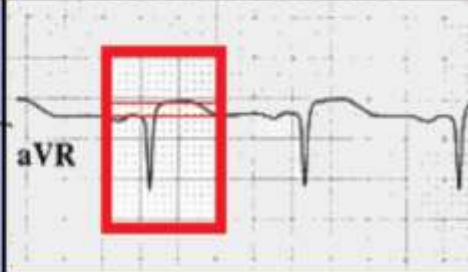
ST SEGMENT ELEVATION

ST SEGMENT DEPRESSION

**LATERAL - ANTERIOR
DIAG (LAD) or OM (CIRC)**



**BASILAR SEPTAL
1st SEPTAL PERF.**



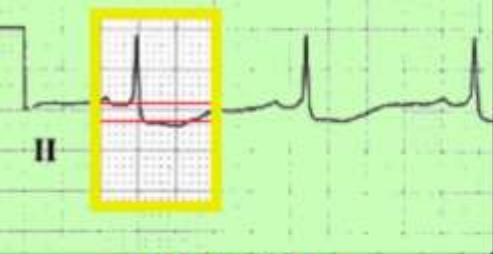
**ANTERIOR SEPTAL
LAD**



**ANTERIOR
LAD**



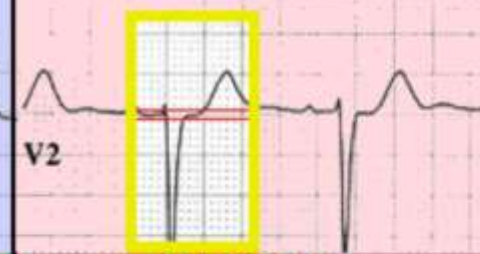
**INFERIOR
RCA or CIRC.**



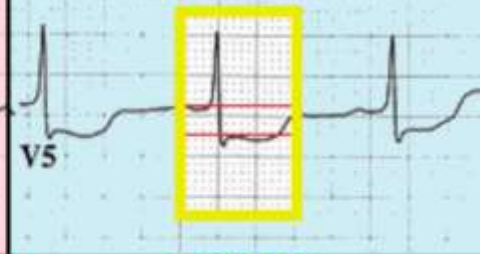
**LATERAL - ANTERIOR
DIAG (LAD) or OM (CIRC)**



**ANTERIOR SEPTAL
LAD**



**LATERAL
CIRC. or LAD**



**INFERIOR
RCA or CIRC.**



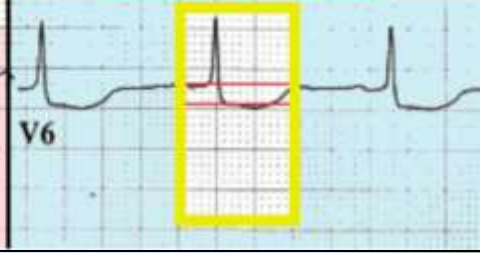
**INFERIOR
RCA or CIRC.**



**ANTERIOR
LAD**



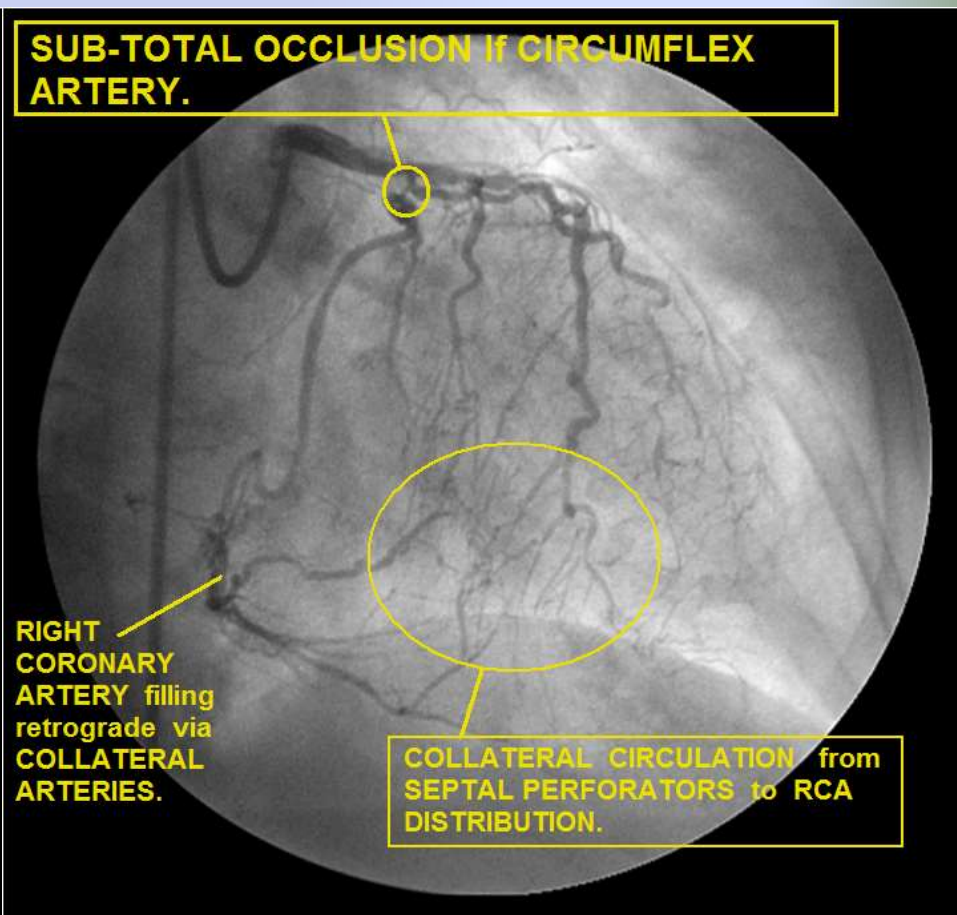
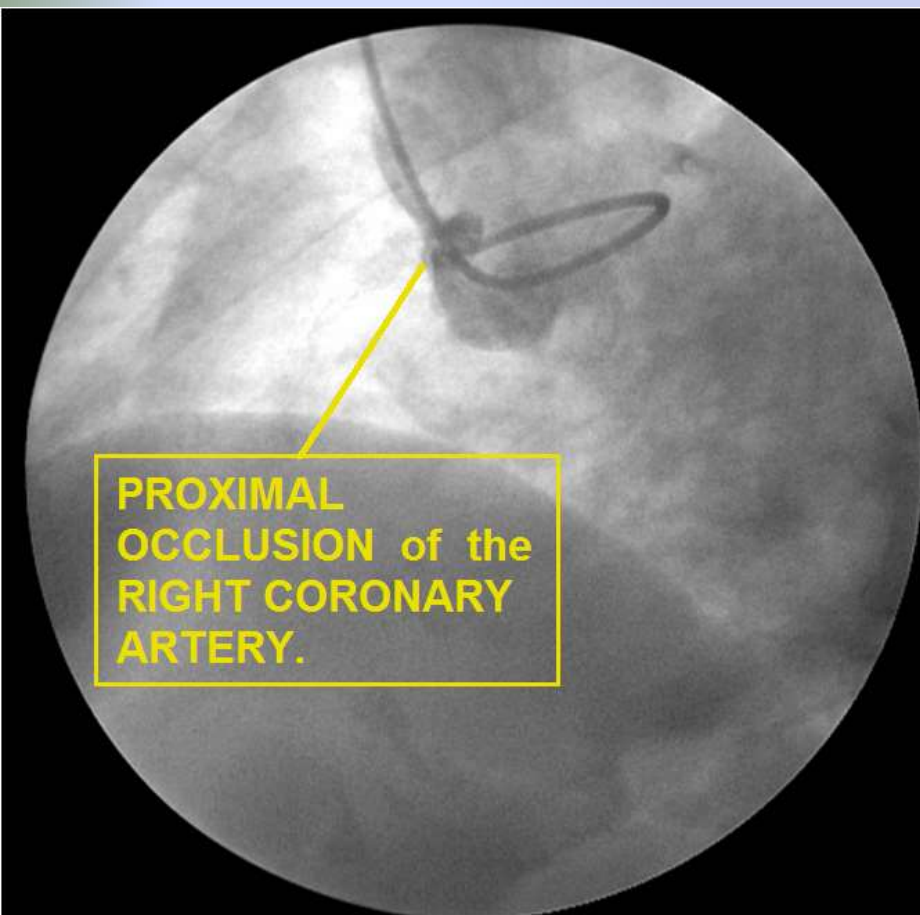
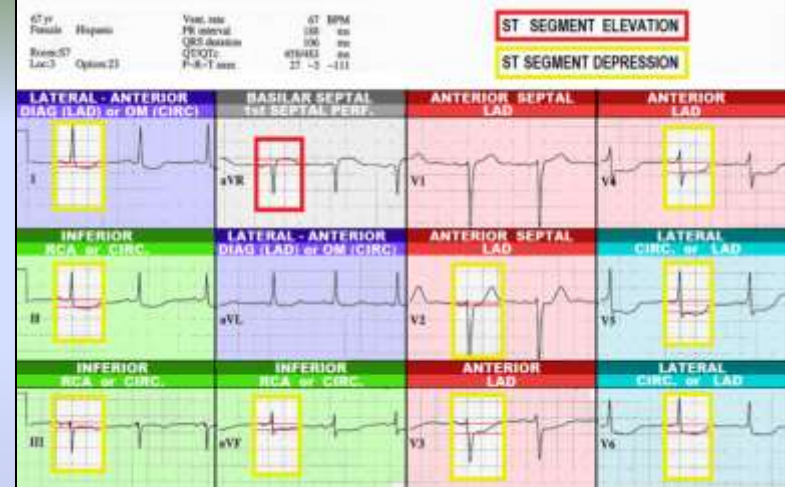
**LATERAL
CIRC. or LAD**



GLOBAL ISCHEMIA

- ST Elevation Lead aVR
- ST Depression in 8 or more other Leads
- Indicates either SUB-TOTALLY OCCLUDED LEFT MAIN CORONARY ARTERY – or – TRIPLE VESSEL DISEASE.
- ***MOST PATIENTS WITH THIS ECG PRESENTATION REQUIRE OPEN HEART SURGERY.***

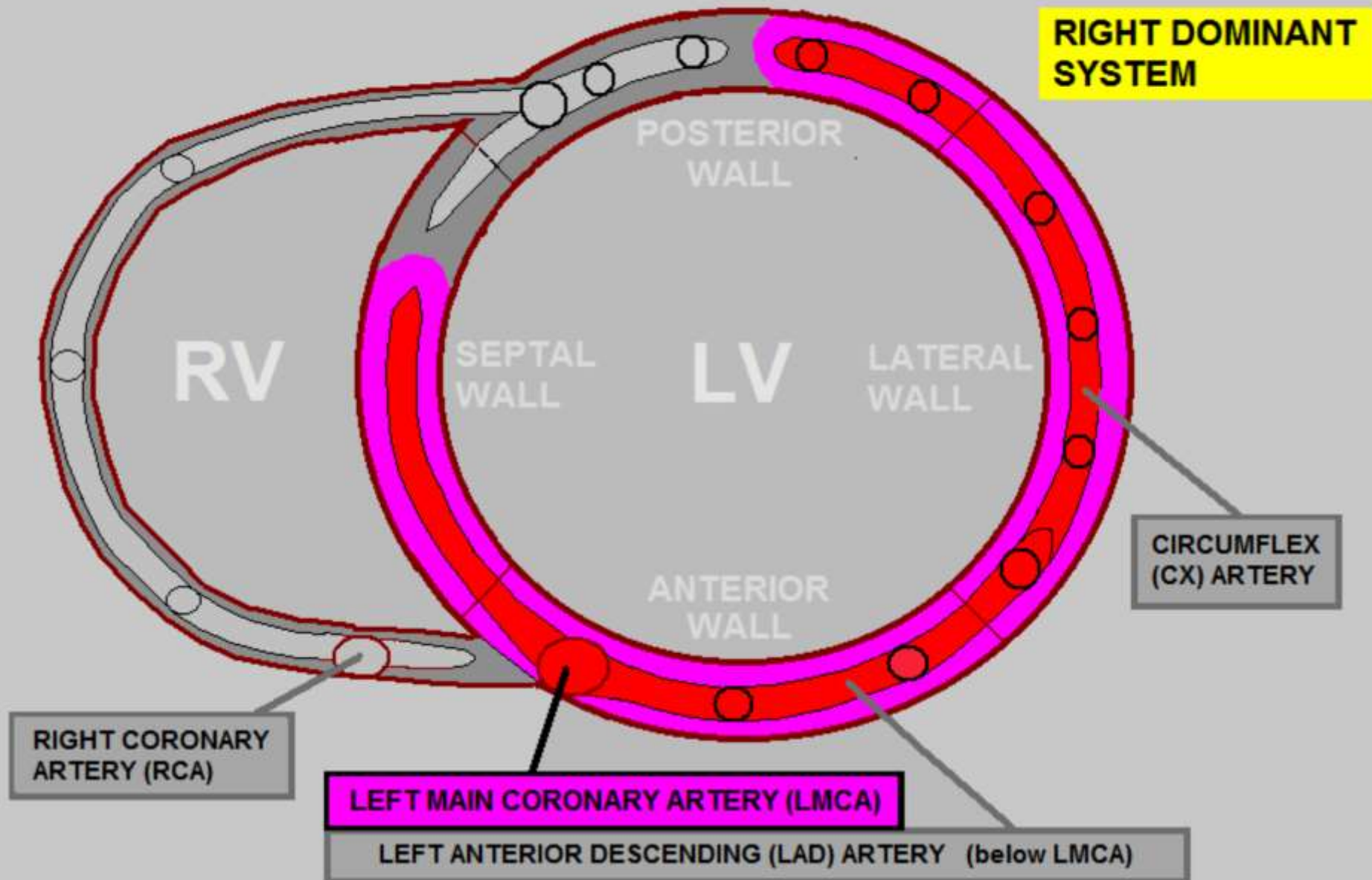
Critical Triple Vessel Disease = *STAT Coronary Artery Bypass Surgery*



cutaway view of the

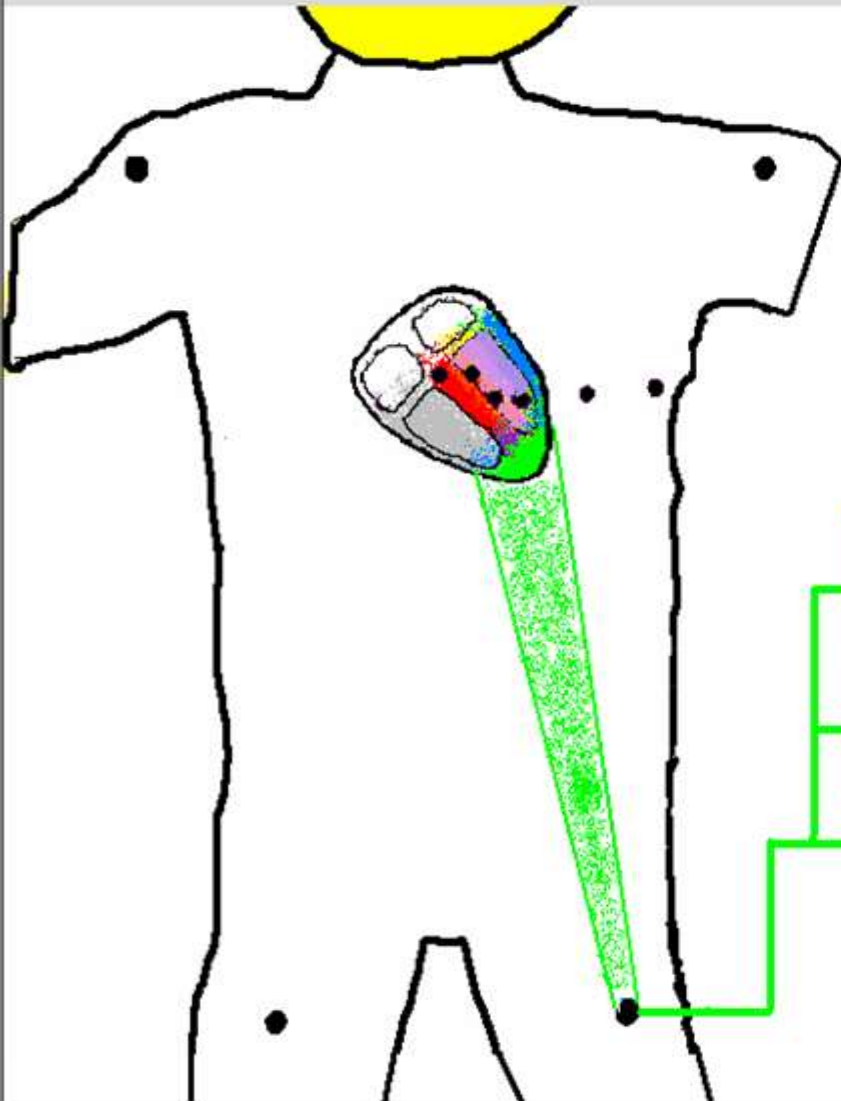
LEFT MAIN CORONARY ARTERY (LMCA)

👉 SUPPLIES APPROXIMATELY 75% OF LV MUSCLE MASS



LEADS II, III, and aVF VIEW

INFERIOR WALL of the LEFT VENTRICLE



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

Which CORONARY ARTERY usually supplies the INFERIOR WALL?

**DOMINANT RIGHT
CORONARY ARTERY**

A-V NODE

**SA
NODE**

LV

RBB

**PLV &
AV
NODAL**

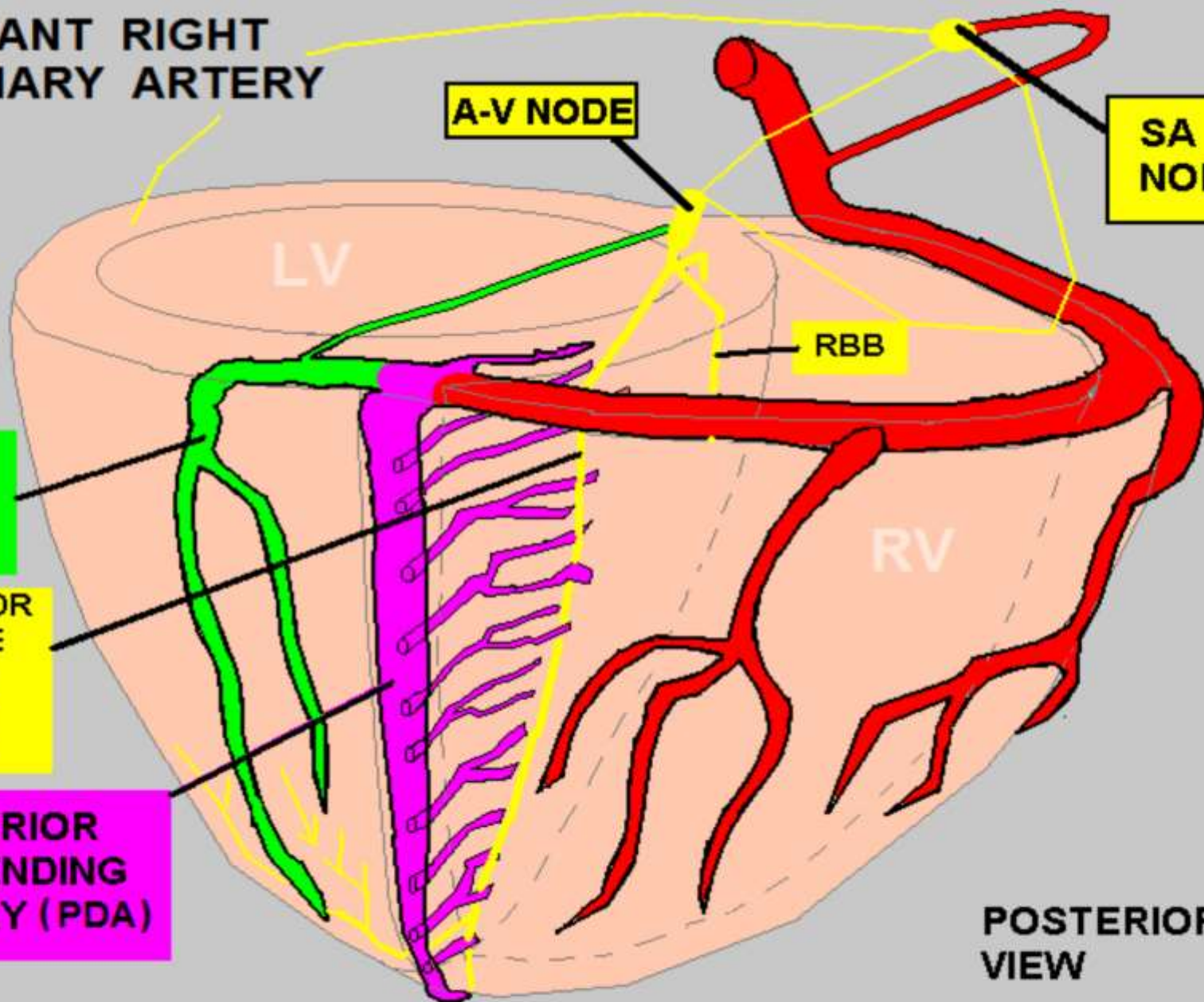
**POSTERIOR
FASCICLE
OF LEFT
BUNDLE
BRANCH**

**POSTERIOR
DESCENDING
ARTERY (PDA)**

RV

**POSTERIOR
VIEW**

75 - 80% of the POPULATION HAVE THIS CORONARY ARTERY ANATOMY





HELPFUL HINT... *MEMORIZE THIS!*

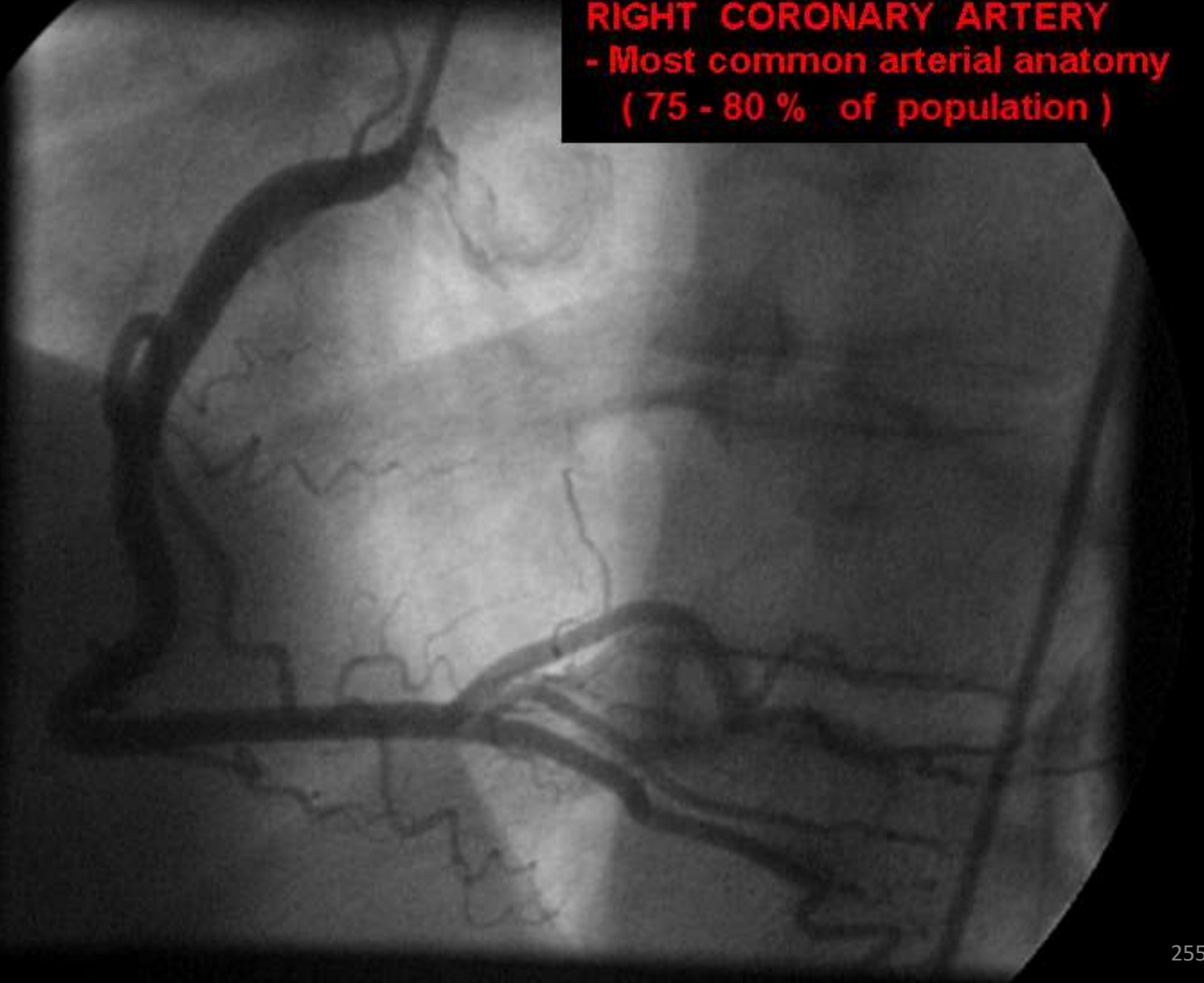


RIGHT CORONARY ARTERY (RCA)

RIGHT DOMINANT
SYSTEMS

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

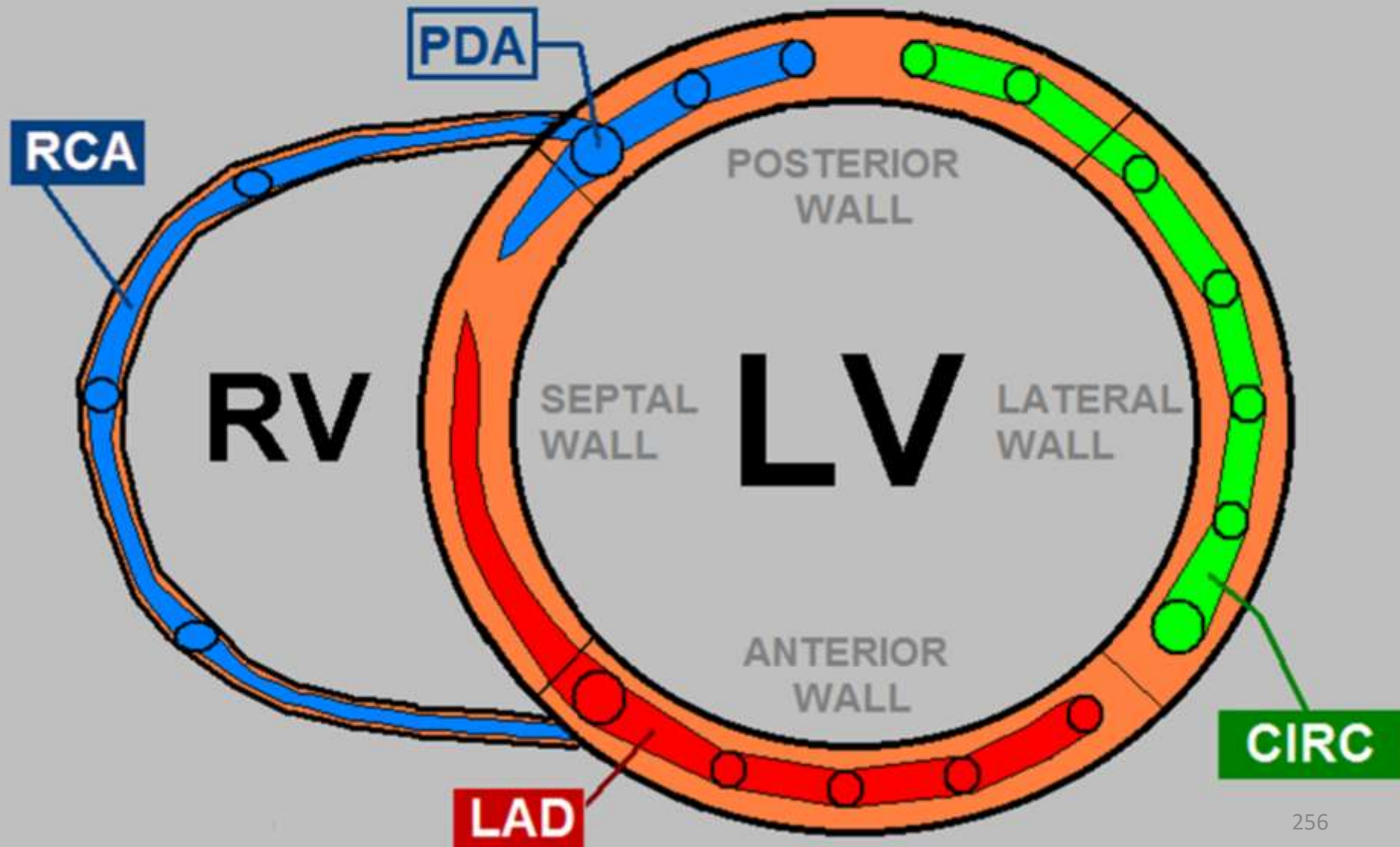
**DOMINANT
RIGHT CORONARY ARTERY**
- Most common arterial anatomy
(75 - 80 % of population)



ARTERIAL DISTRIBUTION - MYOCARDIUM

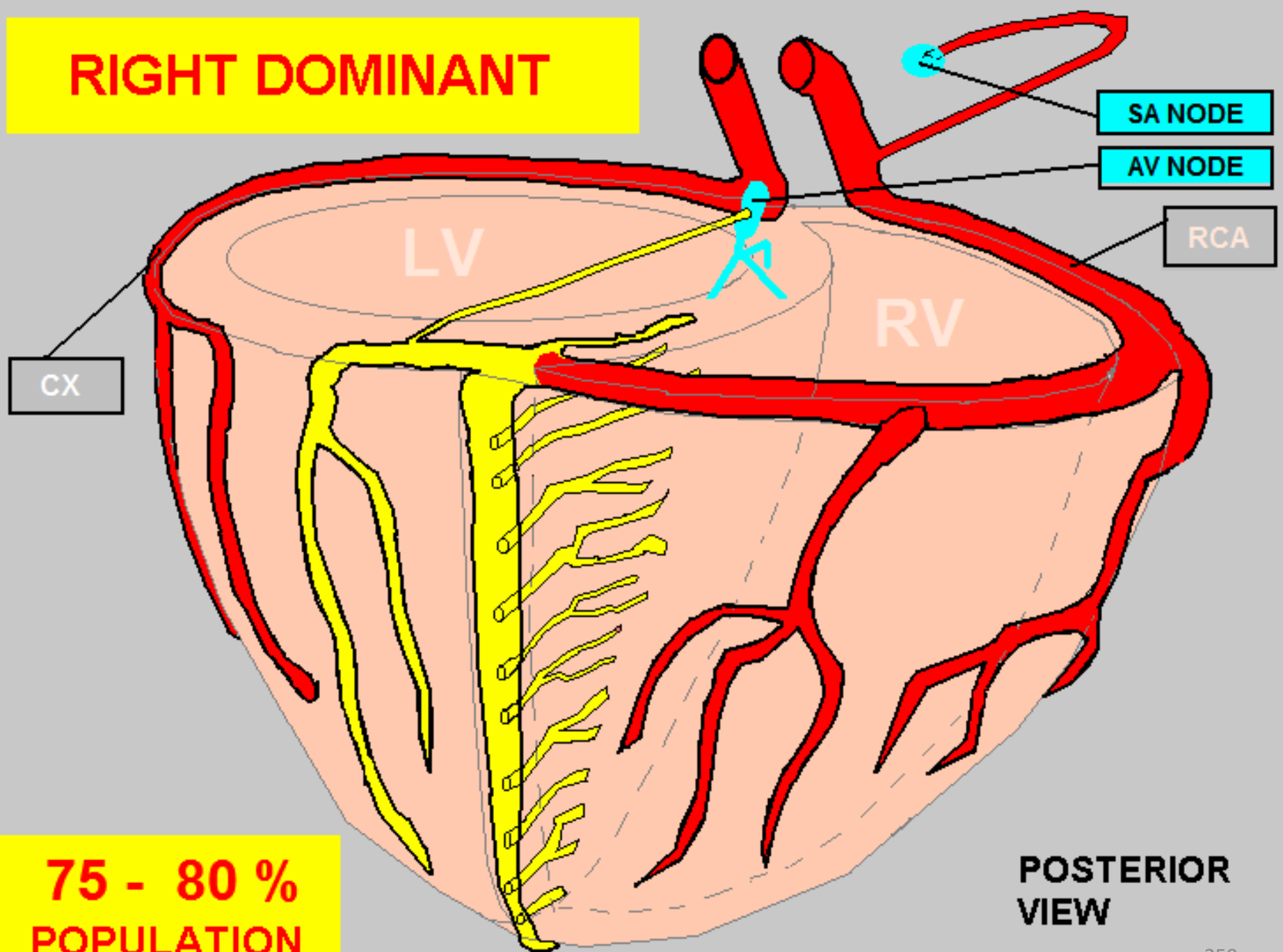
DOMINANT RCA

75-80 % of POPULATION



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

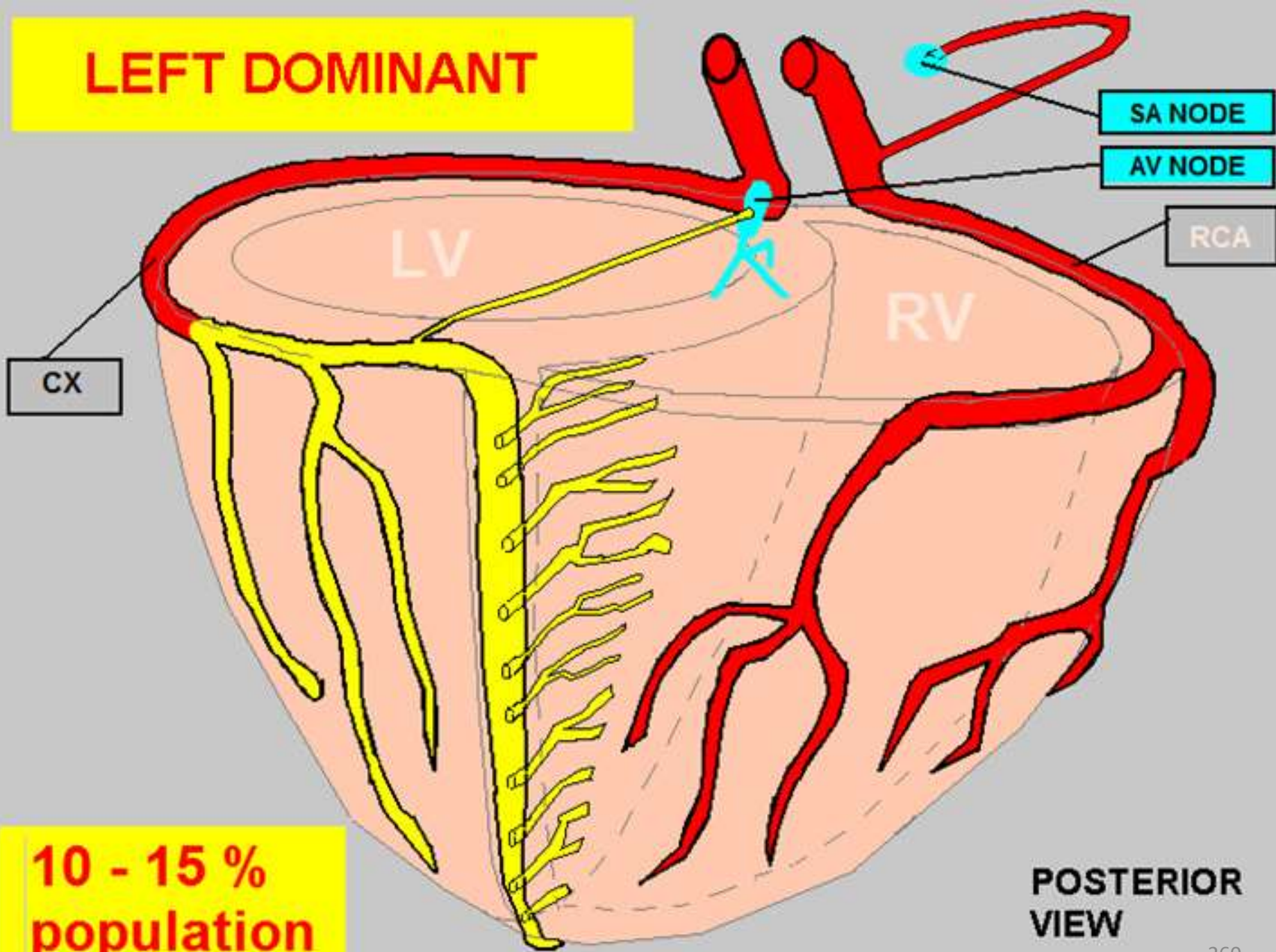
RIGHT DOMINANT



75 - 80 %
POPULATION

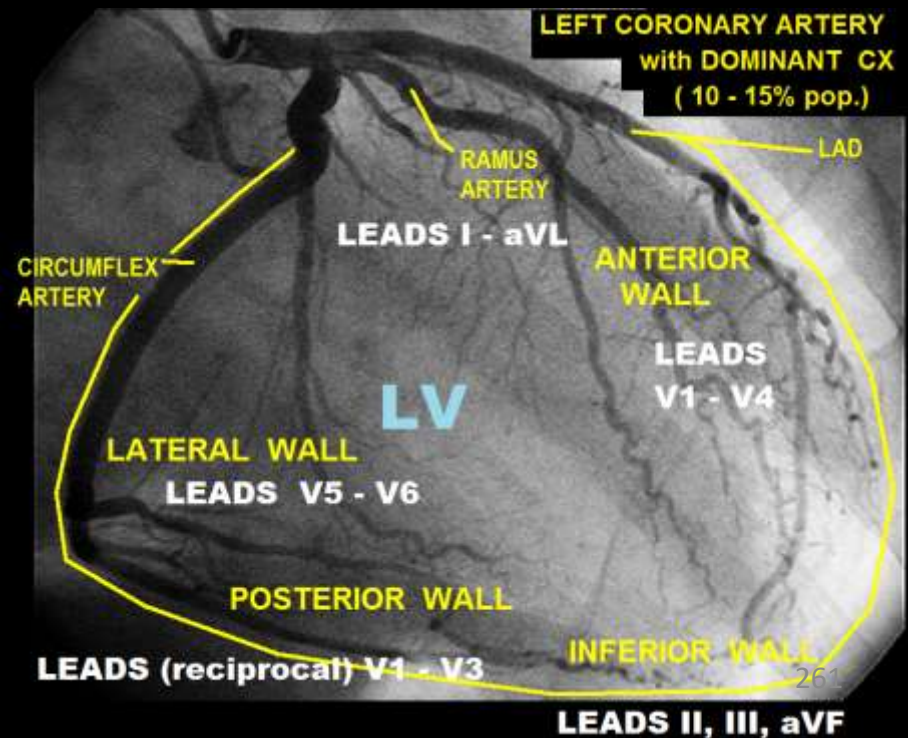
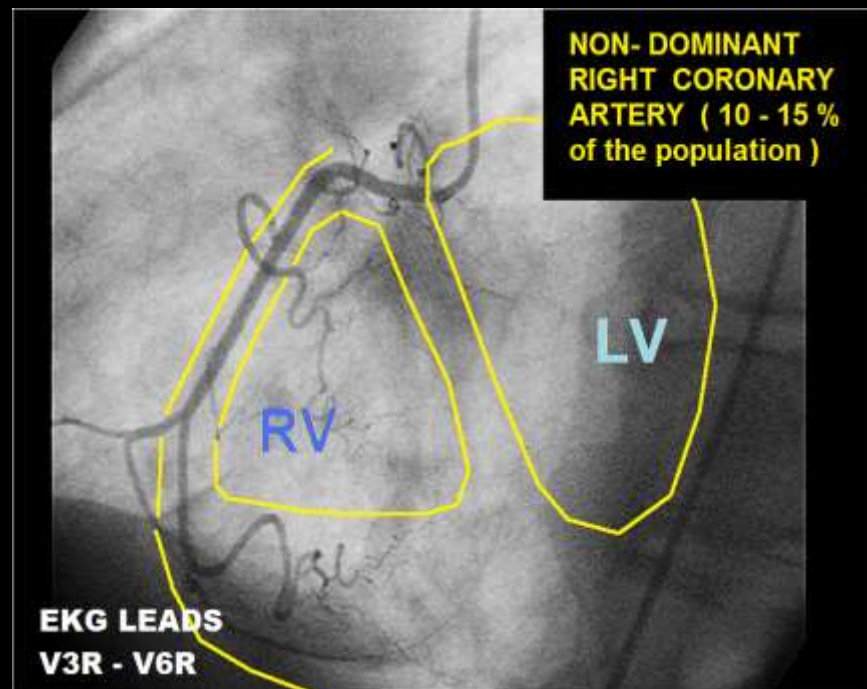
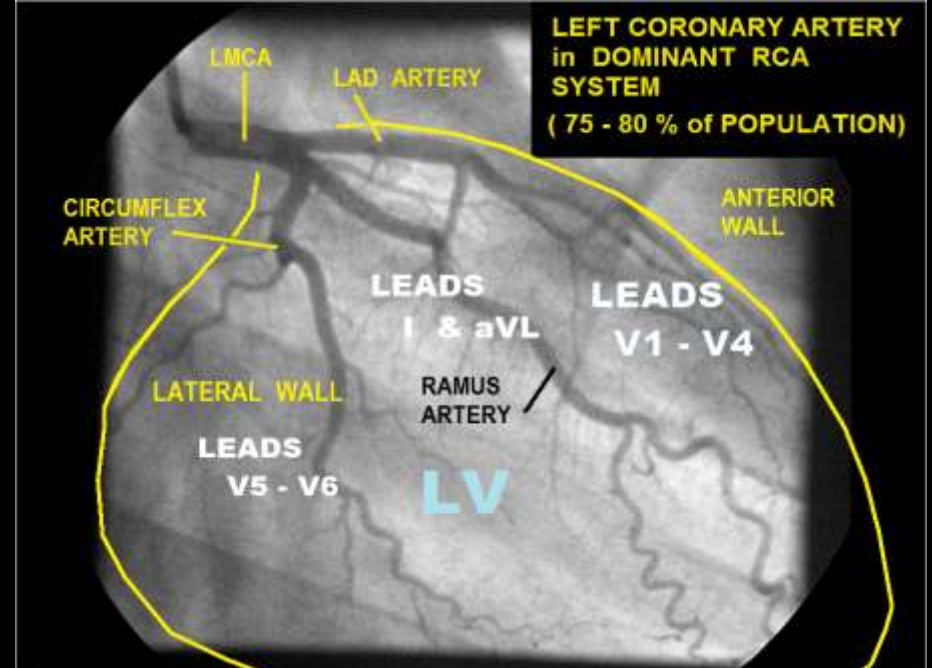
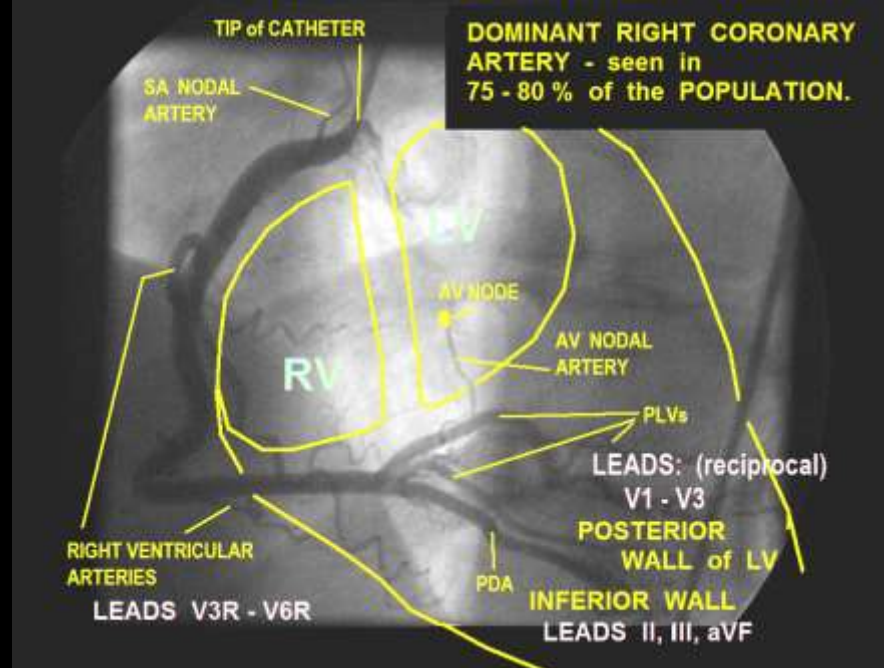
POSTERIOR
VIEW

LEFT DOMINANT

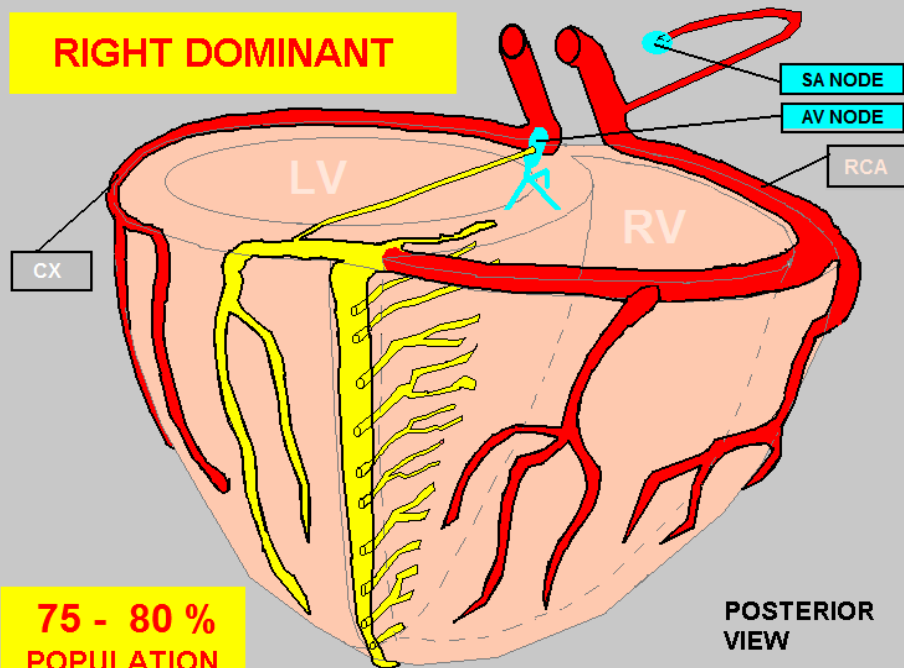


10 - 15 %
population

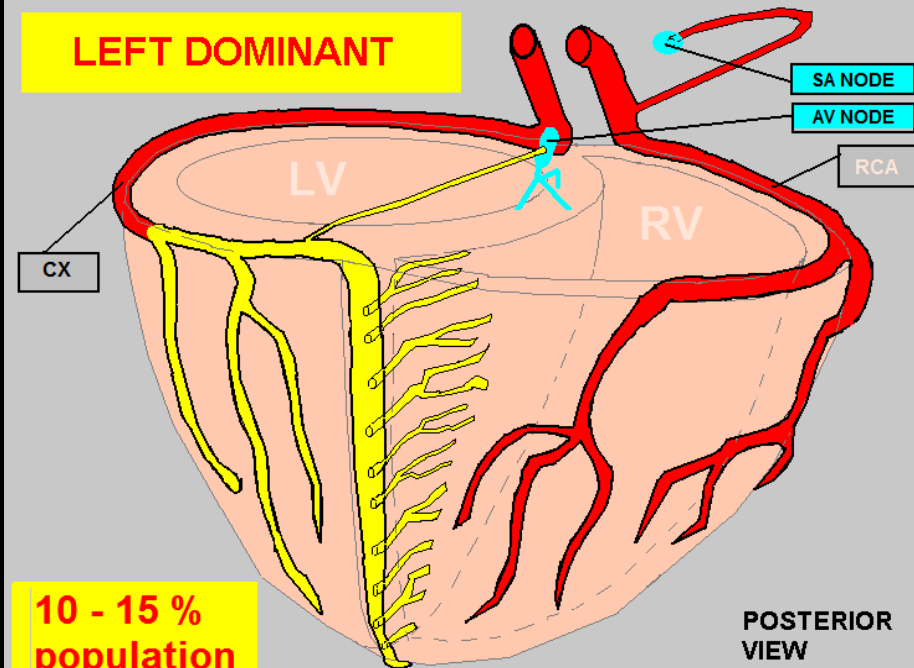
POSTERIOR
VIEW



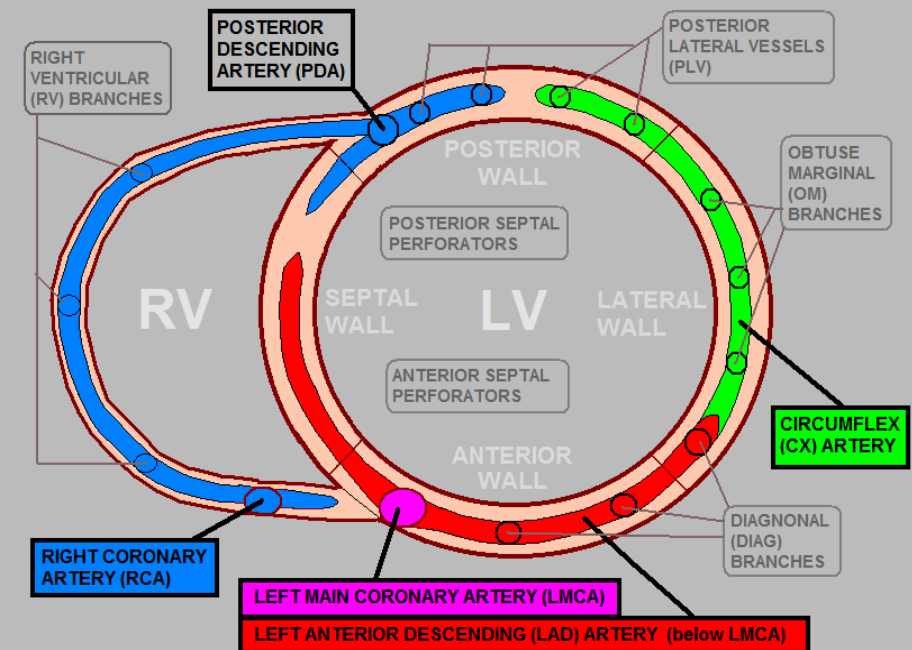
RIGHT DOMINANT



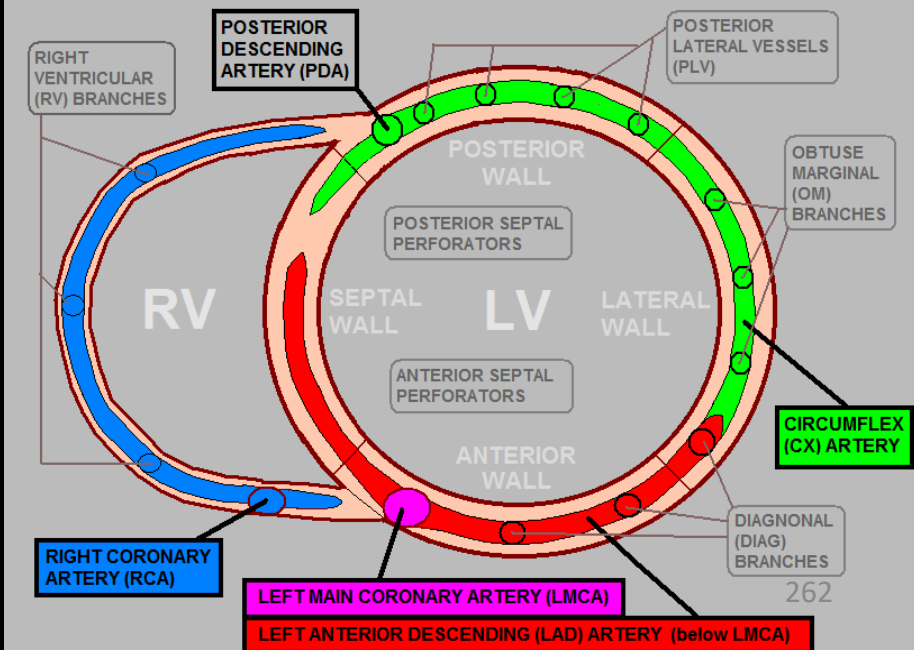
LEFT DOMINANT



CORONARY ARTERIAL DISTRIBUTIONS - RIGHT DOMINANT SYSTEM



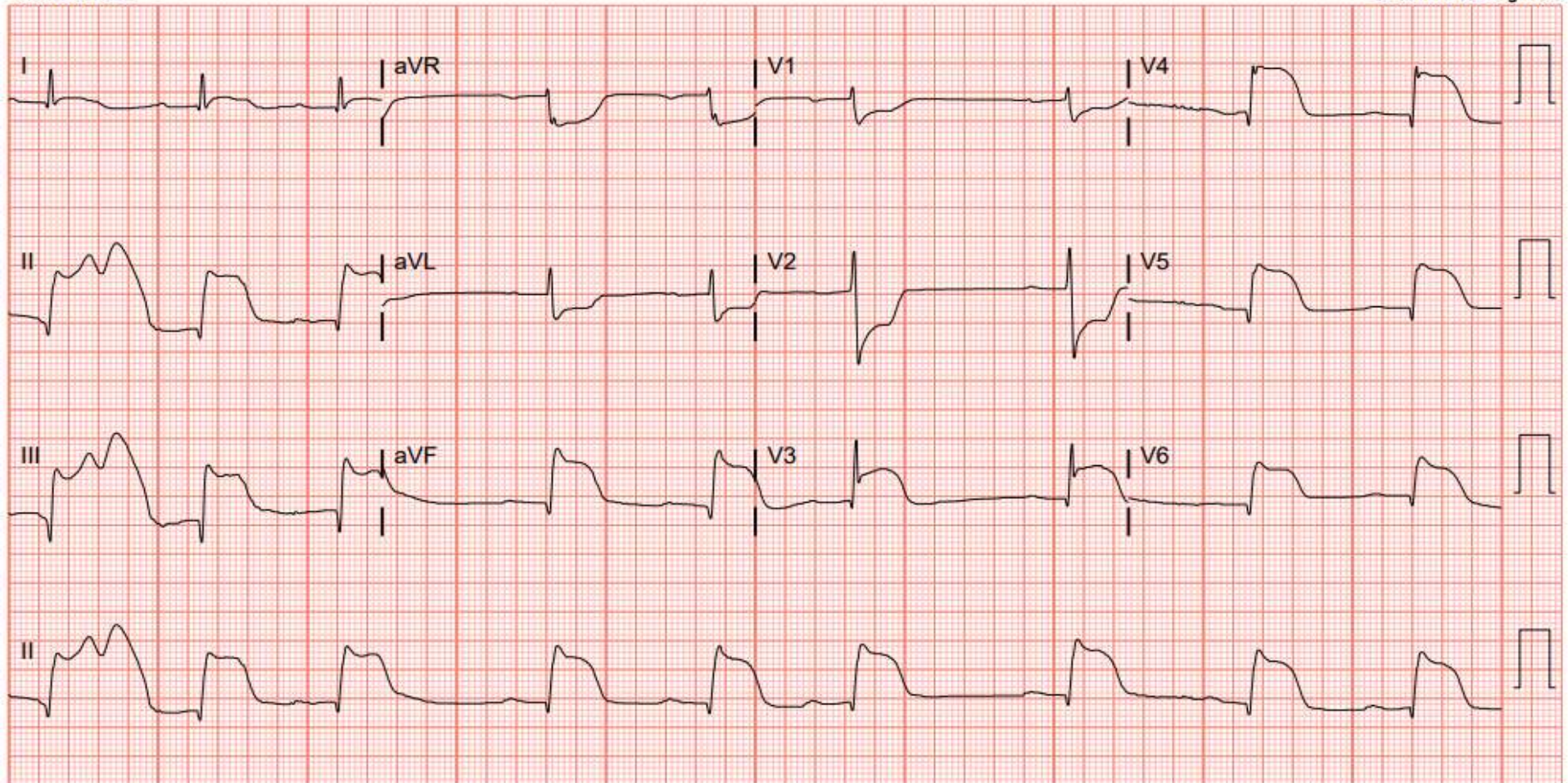
CORONARY ARTERIAL DISTRIBUTIONS - LEFT DOMINANT SYSTEMS



Rate	54	Sinus or ectopic atrial rhythm
PR	329	Atrial premature complex
QRSd	139	Prolonged PR interval
QT	437	Nonspecific intraventricular conduction delay
QTc	415	Inferoposterior infarct, acute (LCx)
-Axis-		Anterolateral infarct, acute
P	-83	Baseline wander in lead(s) V3,V4
QRS	80	
T	77	NO PREVIOUS ECG AVAILABLE FOR COMPARISON

Req Provider: Xandus Chen

- Abnormal ECG - Unconfirmed Diagnosis



CIRCUMFLEX ARTERY (CX)

- NON-DOMINANT CX:

CX = 15 - 30% OF LV MASS

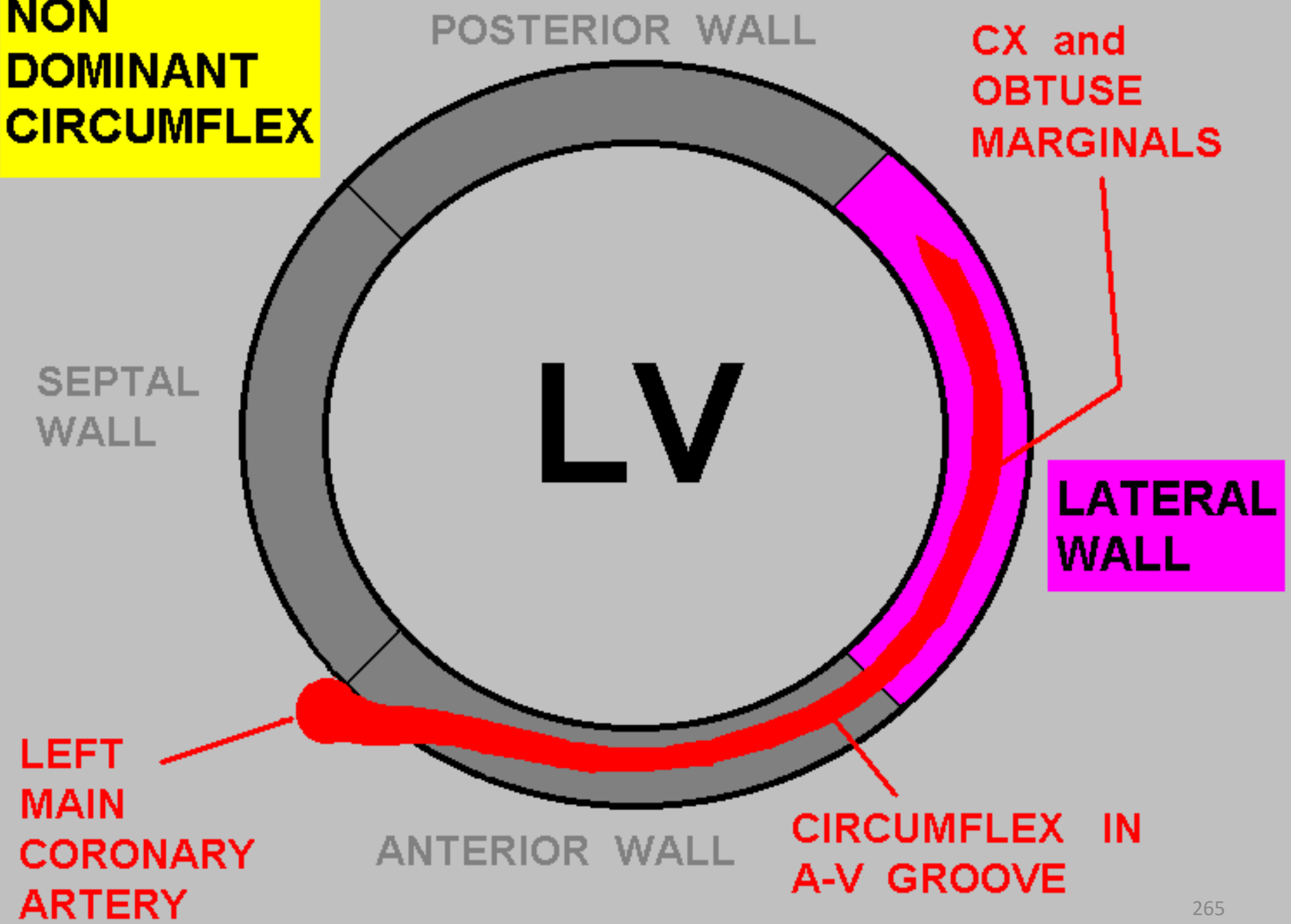
- DOMINANT CX:

CX = 15 - 30% OF LV MASS

+ PDA = 15 - 25% OF LV MASS

TOTAL 30 - 55% OF LV MASS

**NON
DOMINANT
CIRCUMFLEX**



**DOMINANT
CIRCUMFLEX**

POSTERIOR WALL

**CX and
OBTUSE
MARGINALS**

PLVs

PDA

SEPTAL FEEDERS

LV

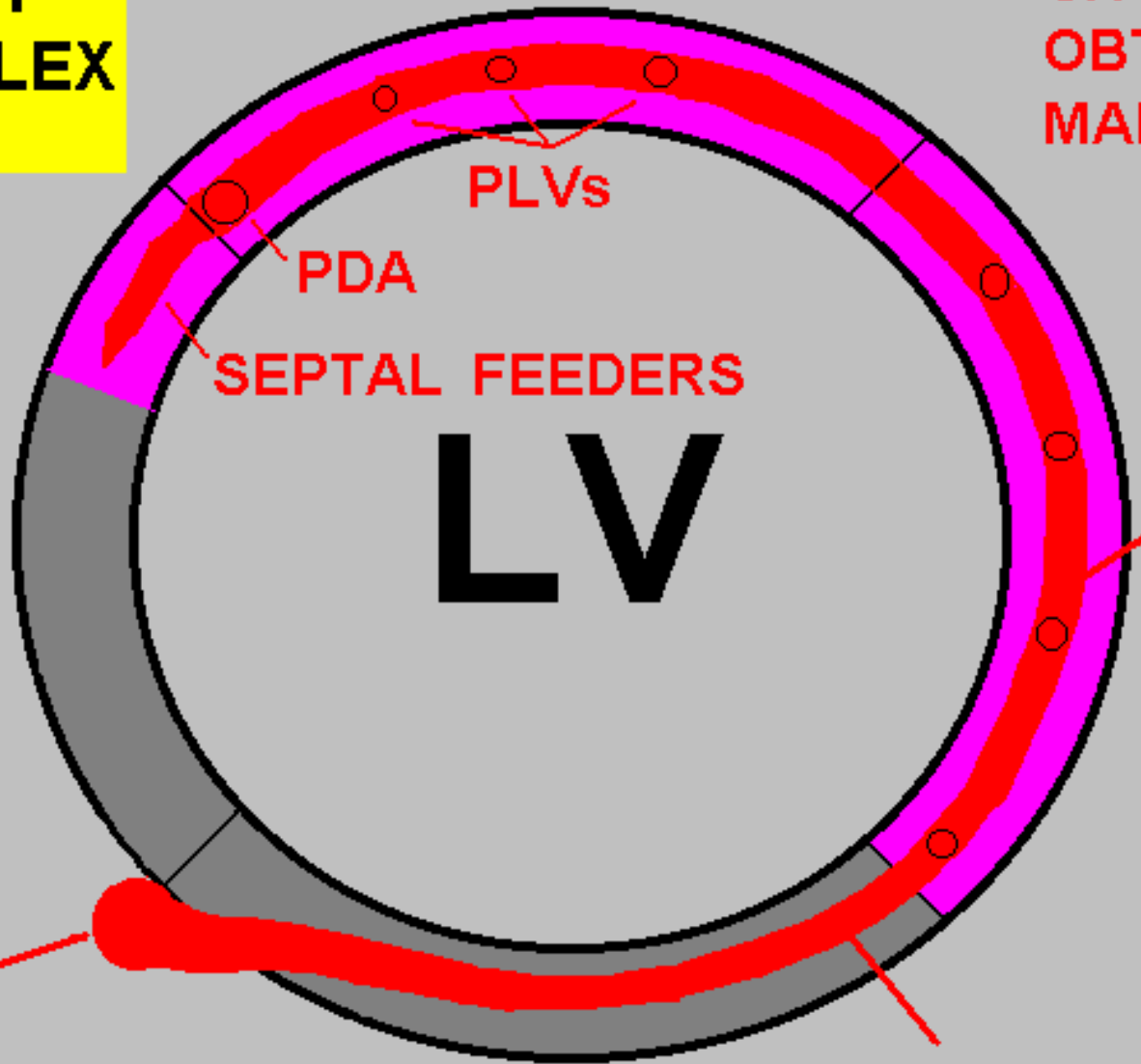
**SEPTAL
WALL**

**LATERAL
WALL**

**LEFT
MAIN
CORONARY
ARTERY**

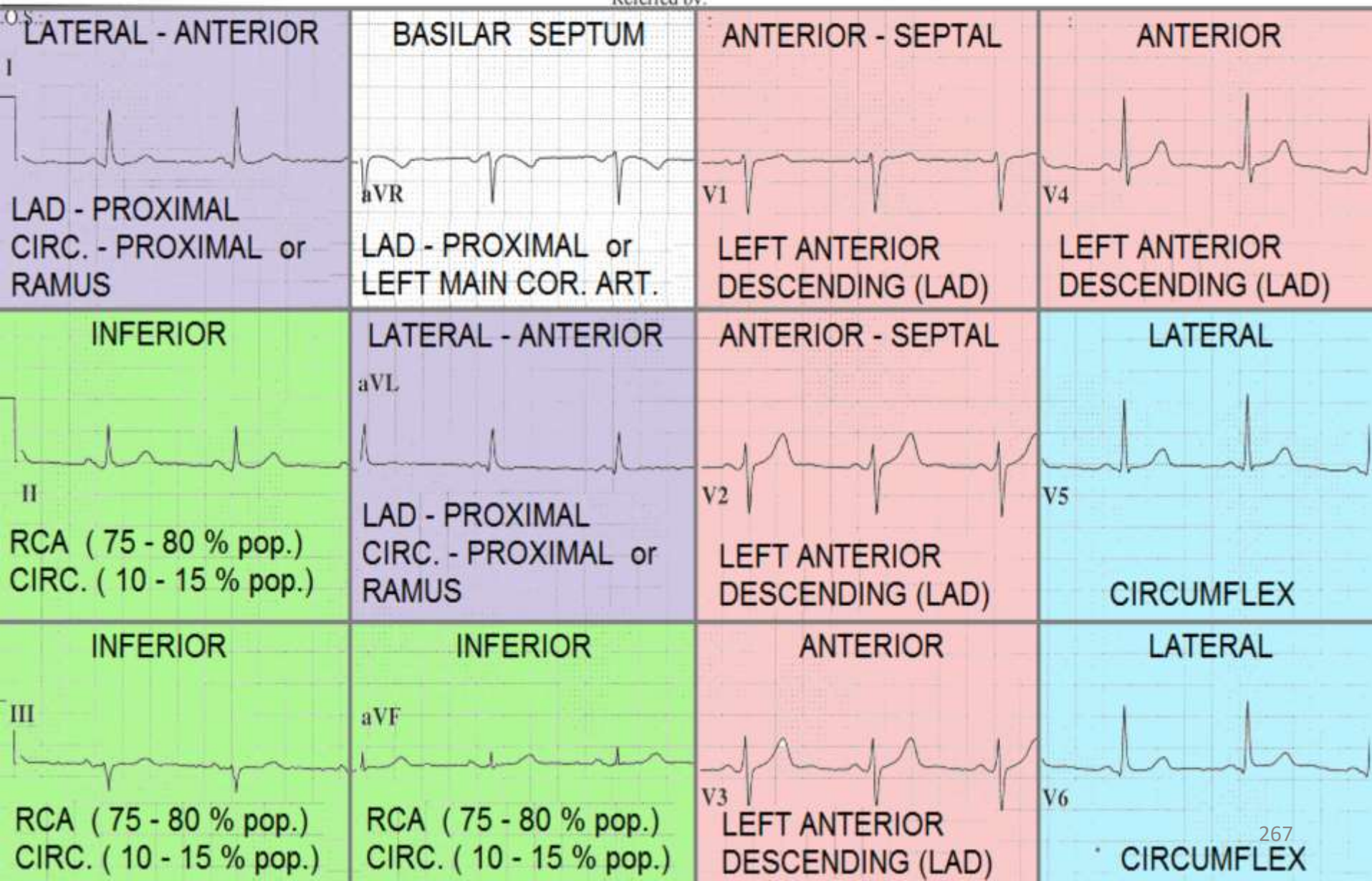
ANTERIOR WALL

**CIRCUMFLEX IN
A-V GROOVE**



Vent. rate	64	BPM	Normal sinus rhythm
PR interval	130	ms	Normal ECG
QRS duration	96	ms	No previous ECGs available
QT/QTc	396/408	ms	
P-R-T axes	40 11 61		

Referred by:



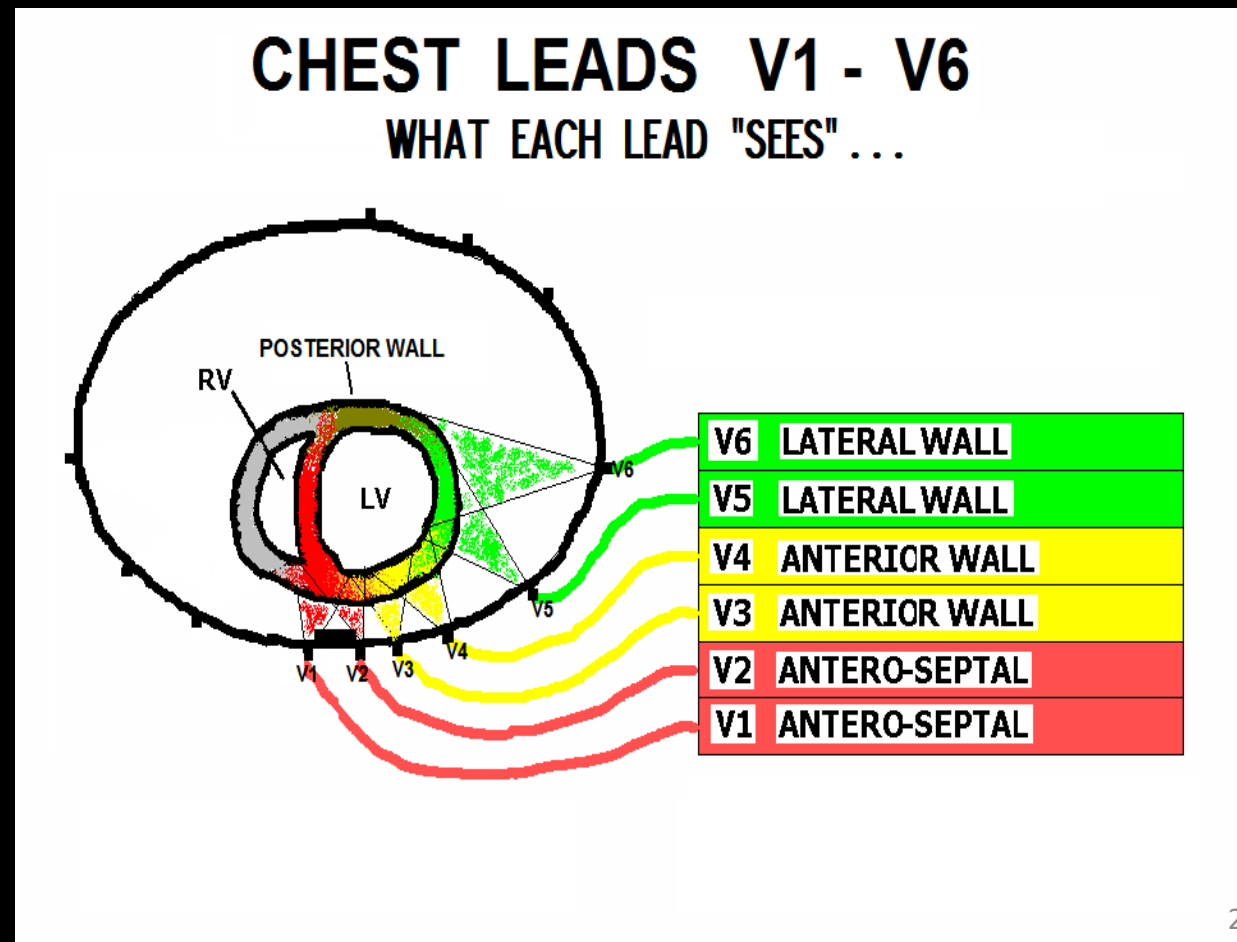
The 12 Lead ECG

Has **TWO** major **BLIND SPOTS**

The **POSTERIOR WALL**

&

**RIGHT
VENTRICLE**



When do we need to see the Right Ventricle?

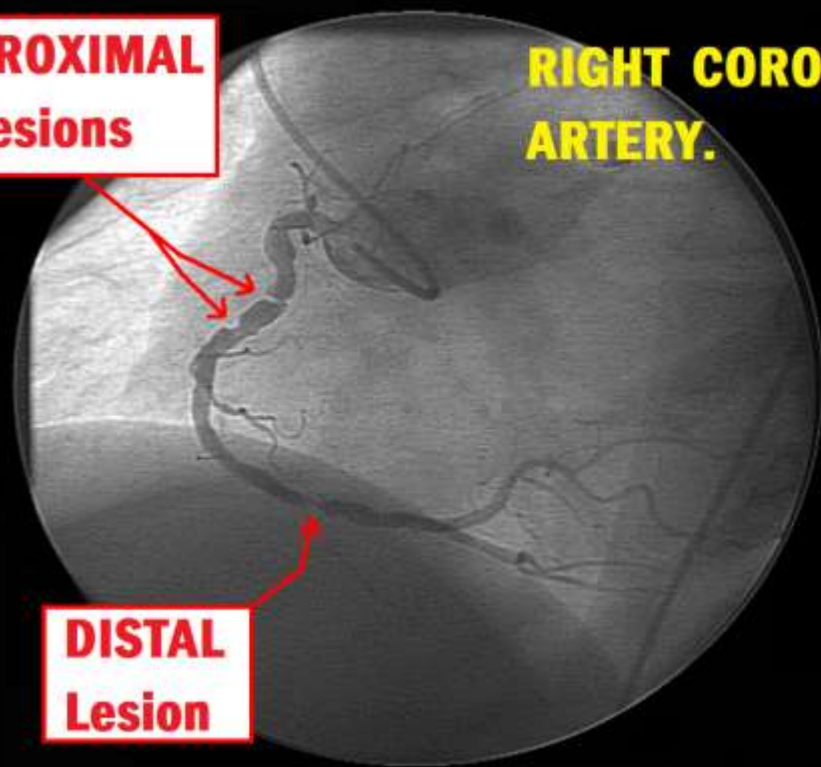
- All Patient with INFERIOR WALL STEMI (ST Elevation in Leads II, III, aVF).

When you see an EKG with **ST Elevation in Leads II, III and AVF** (Inferior Wall STEMI) – you cannot tell if the blockage is in the **PROXIMAL RCA** – or the **DISTAL RCA**.

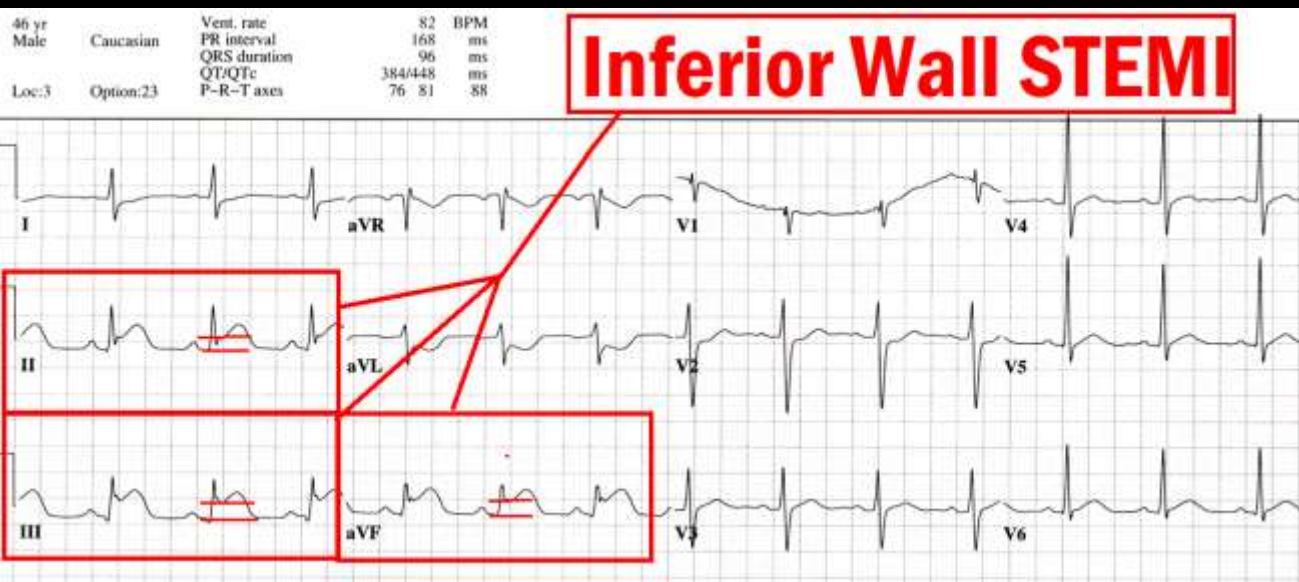
PROXIMAL Lesions

RIGHT CORONARY ARTERY.

DISTAL Lesion



Inferior Wall STEMI



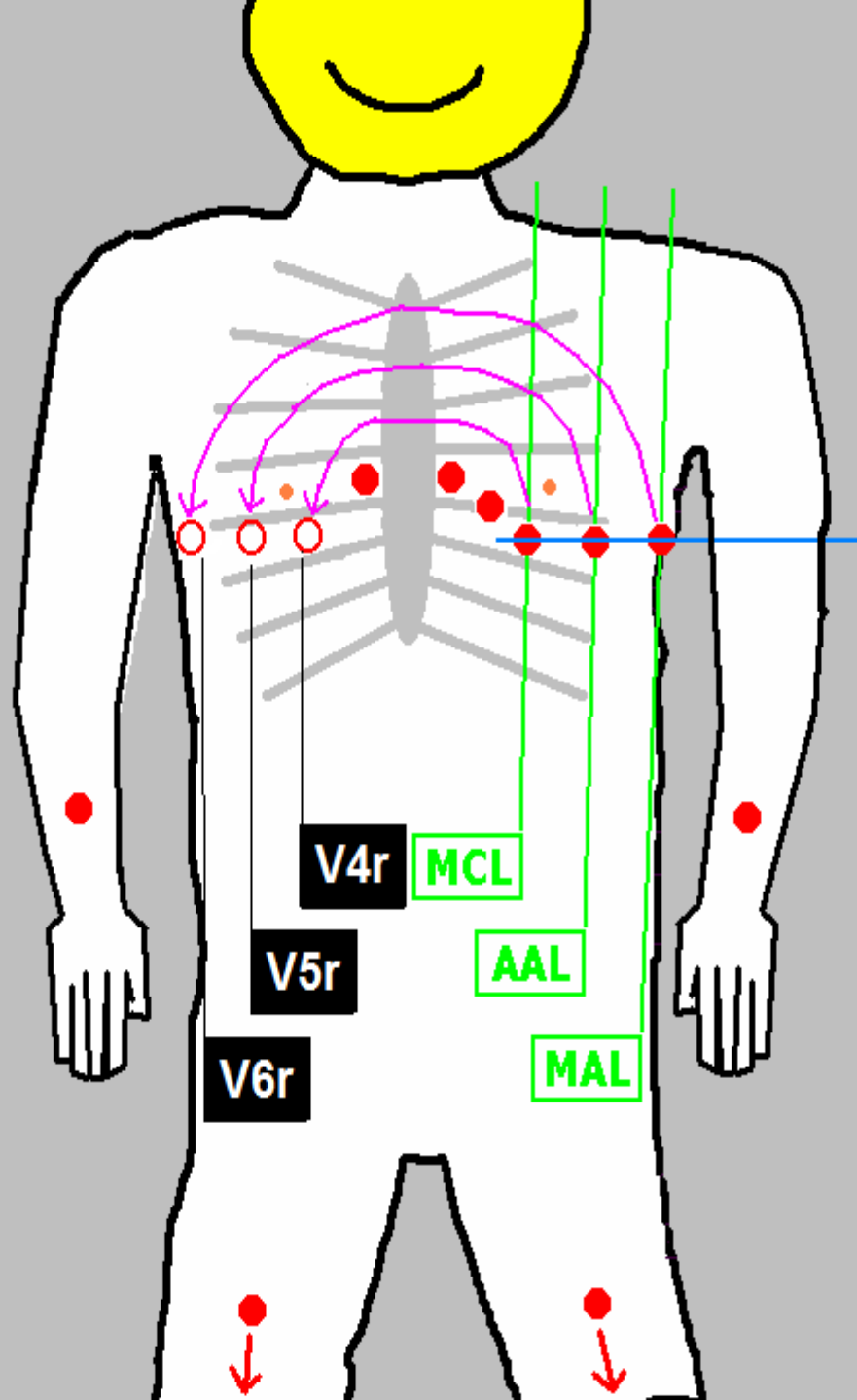
To see the
RIGHT VENTRICLE . . .

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



You must do a

RIGHT - SIDED EKG !!

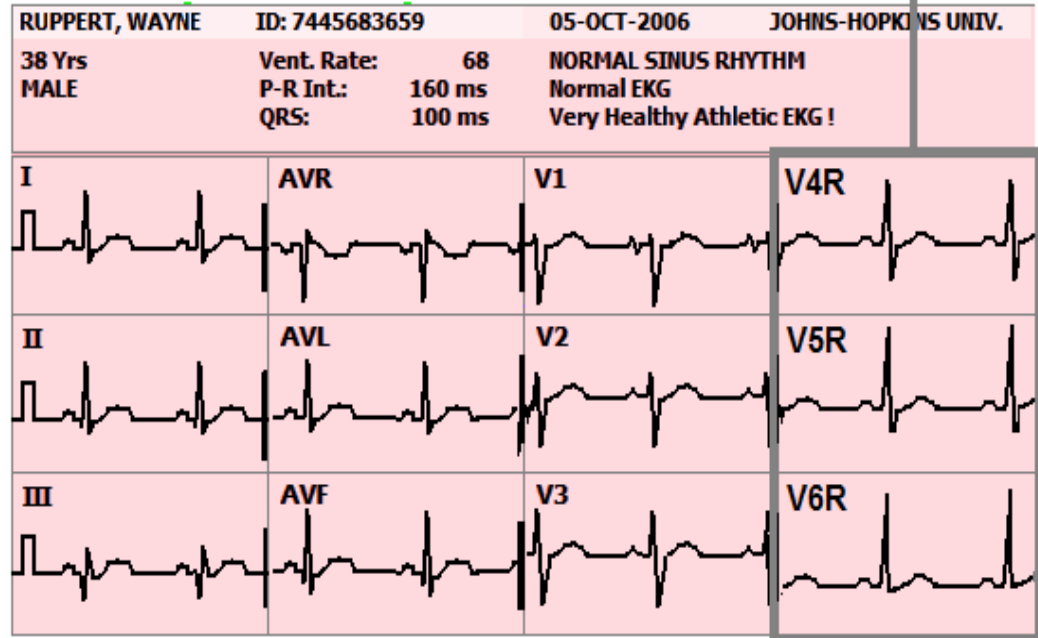
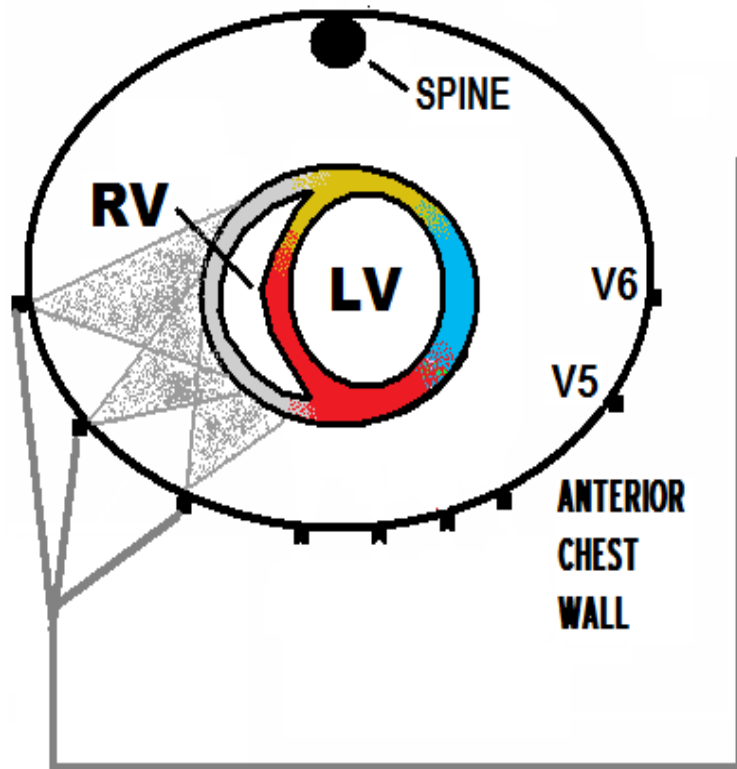


To do a
RIGHT - SIDED EKG . .

**MOVE leads
V4, V5, and V6**

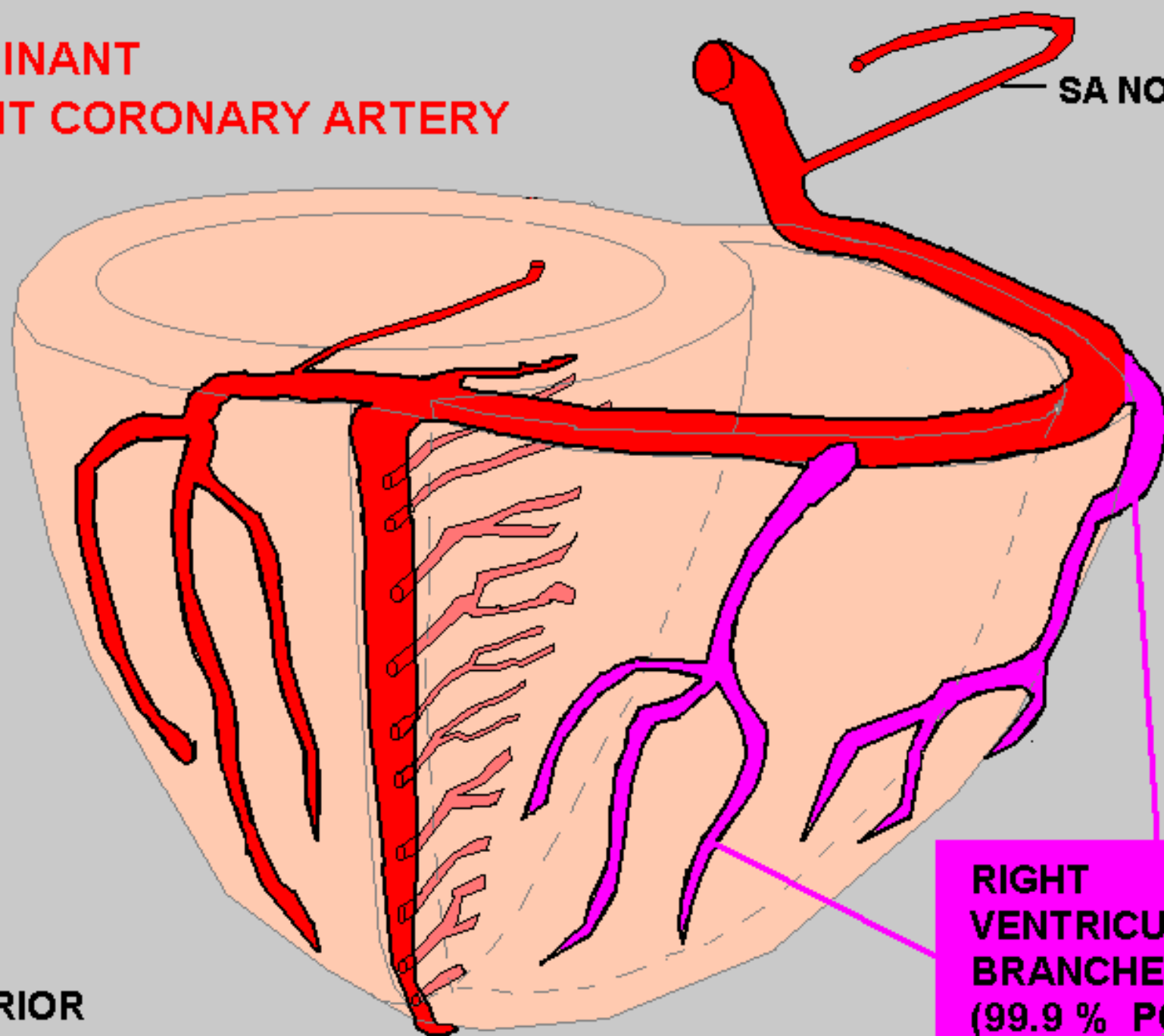
**to the corresponding
placement on the
RIGHT SIDE of patient's
chest . . .**

V4R - V6R VIEW THE RIGHT VENTRICLE



**DOMINANT
RIGHT CORONARY ARTERY**

SA NODAL



**RIGHT
VENTRICULAR
BRANCHES
(99.9 % POP.)**

**POSTERIOR
VIEW**

ID:

46 yo
Male Caucasian

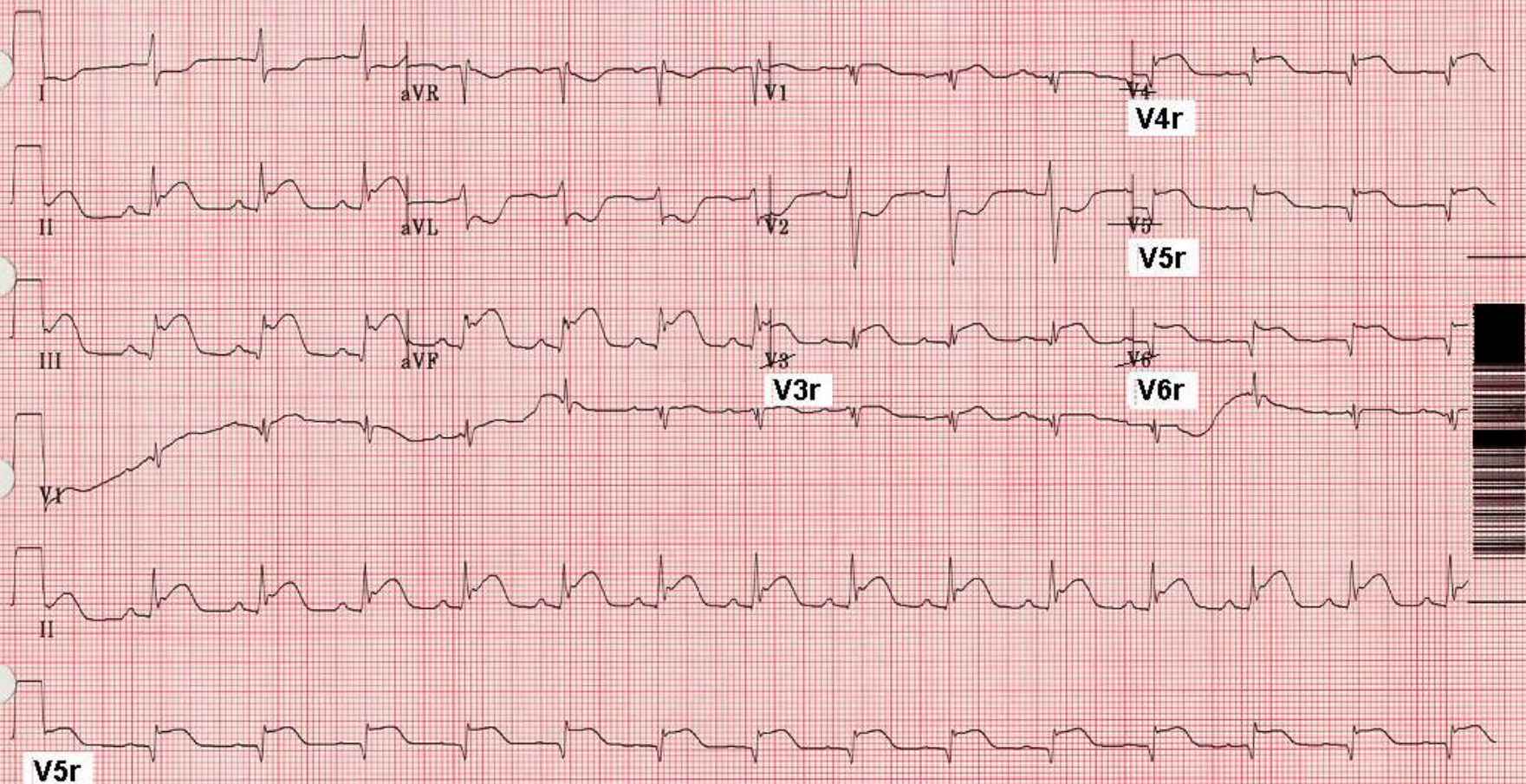
Room: Opt:

Vent. rate 87 bpm
PR interval 176 ms
QRS duration 94 ms
QT/QTc 330/397 ms
P-R-T axes 79 81 102Normal sinus rhythm
~~Anterolateral infarct, possibly acute~~
Inferior injury pattern
***** Acute MI *****
Abnormal ECG**Right Ventricular Infarct**V LEADS
R SIDE

Technician:

Referred by:

Unconfirmed



40 Hz 25.0 mm/s 10.0 mm/mV

4 by 2.5s + 3 rhythm lds

MACVU 003C

12SL™ v250

When do we need to see the Posterior Wall?

- Any time a patient presents with symptoms of ACS and the 12 Lead ECG shows ST Depression in Leads V1, V2, V3 and/or V4.

Whenever you see
ST DEPRESSION in Leads V1 - V4



you must do a

POSTERIOR LEAD ECG

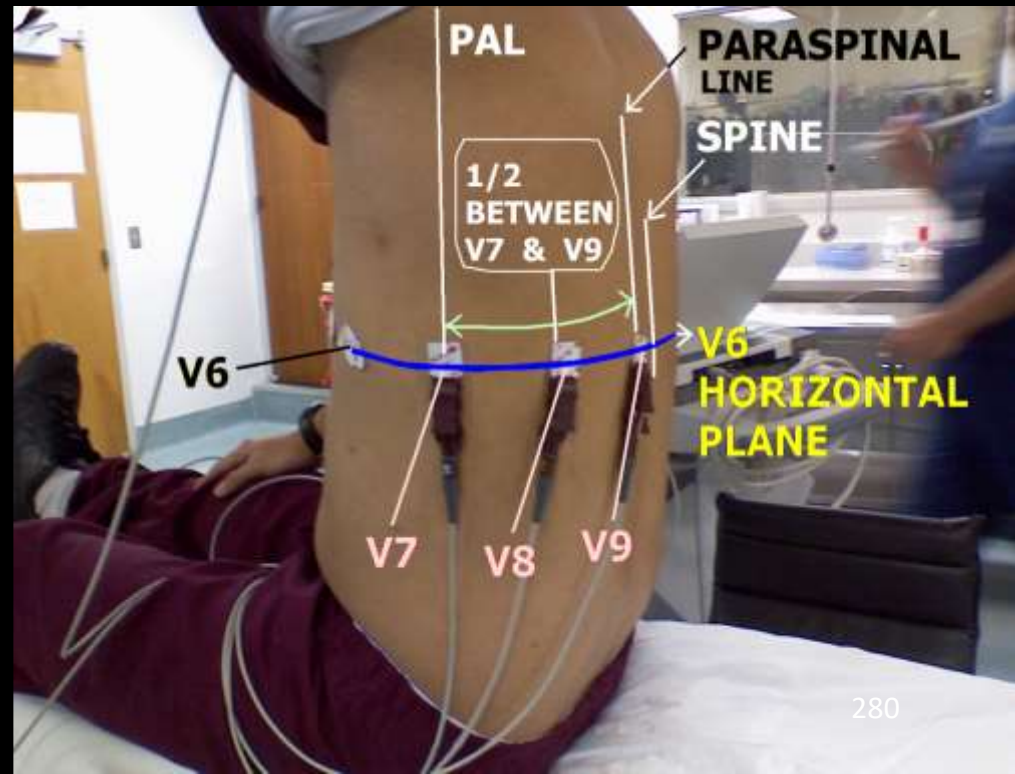
(V7 - V9)

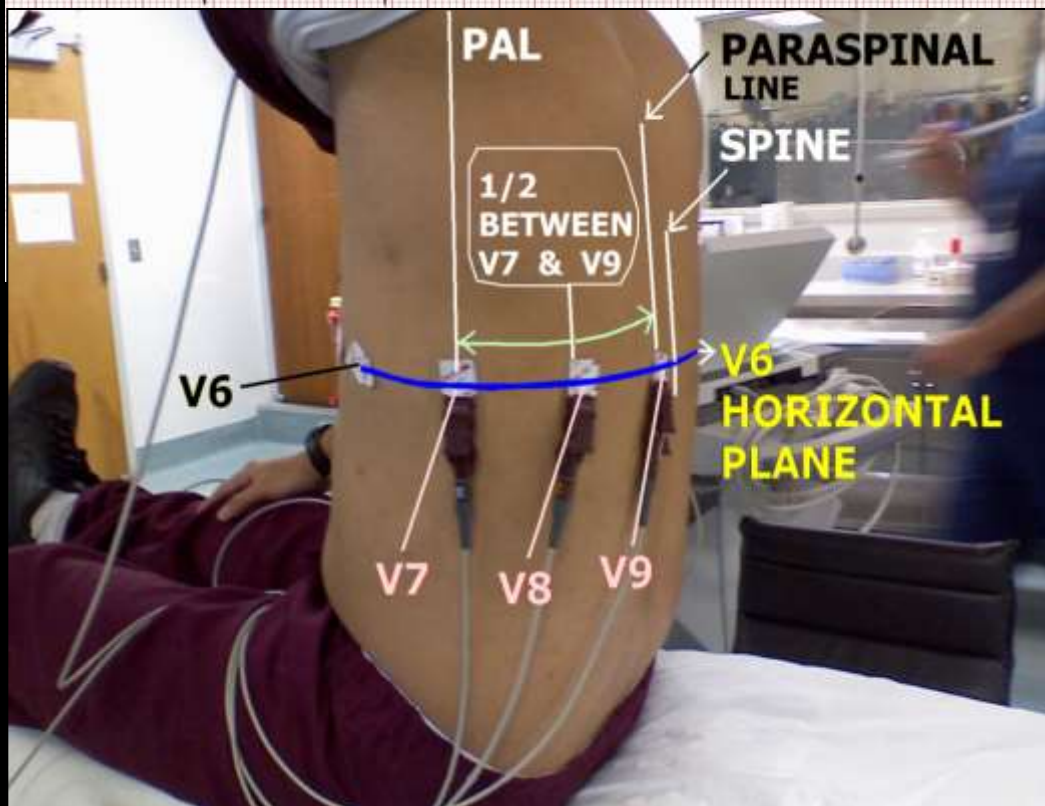
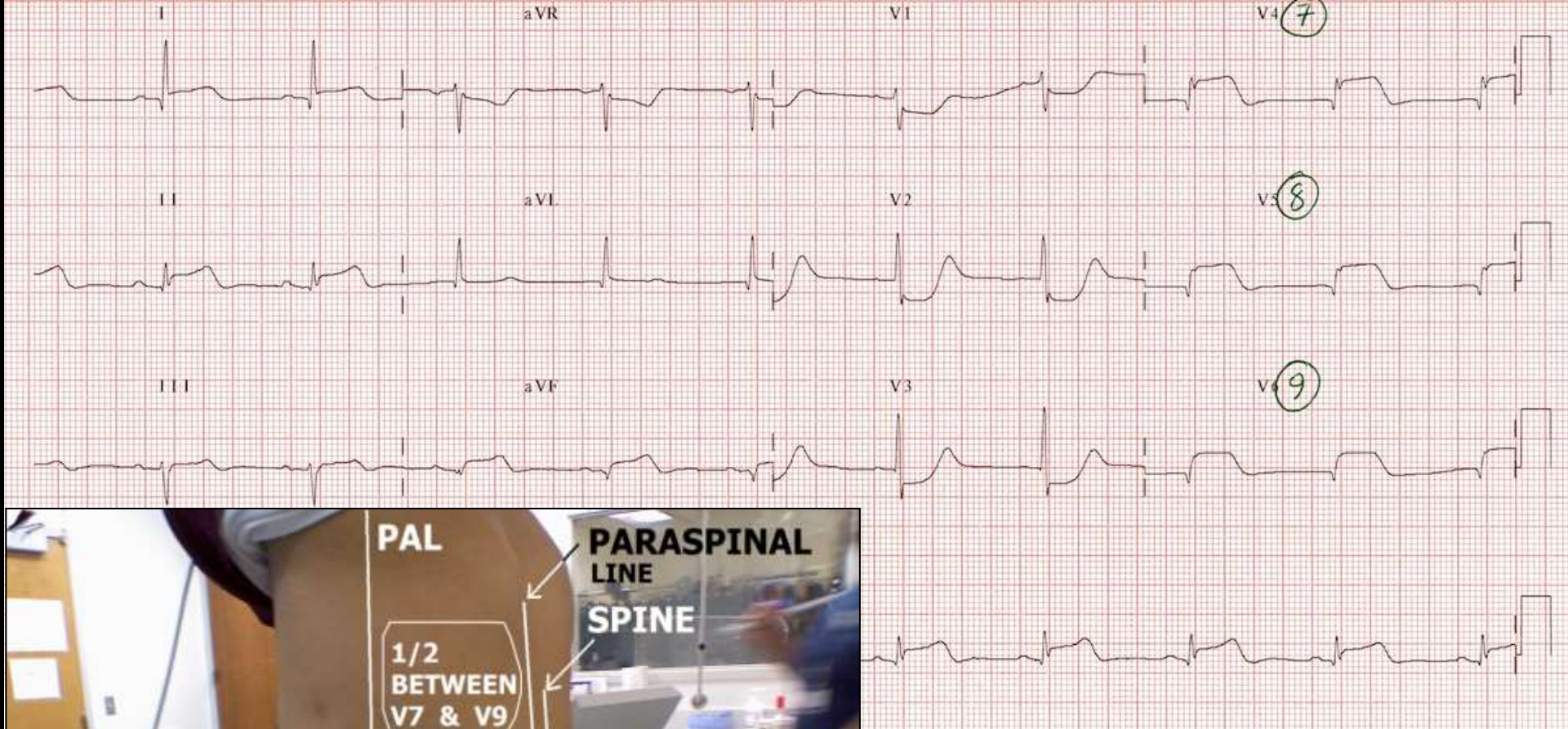
to see if you Patient is having a

POSTERIOR WALL STEMI

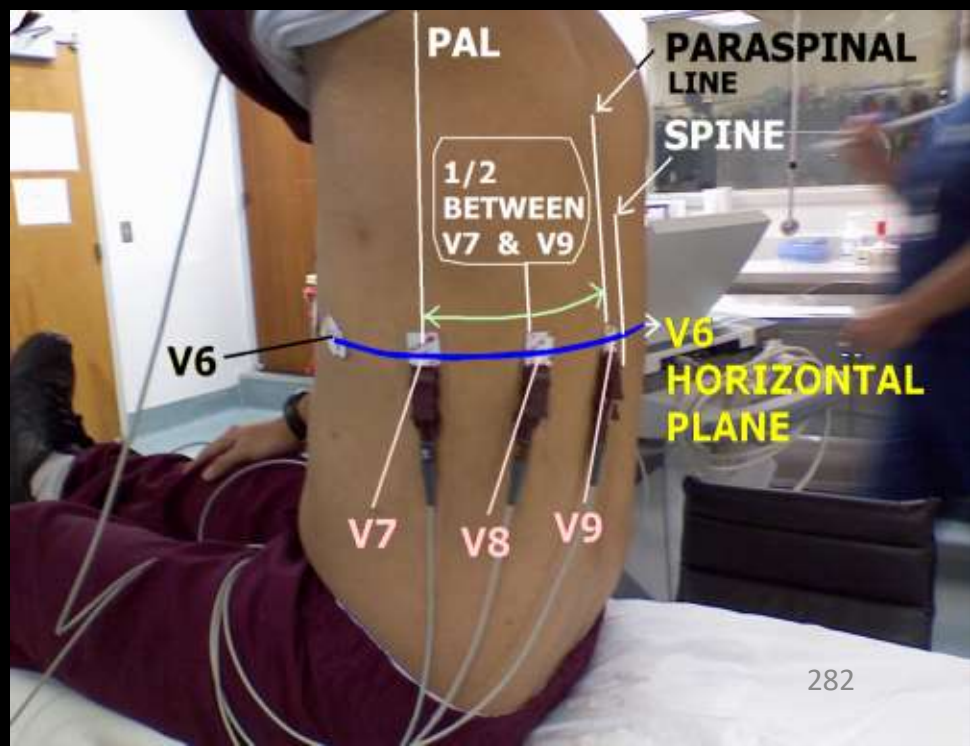
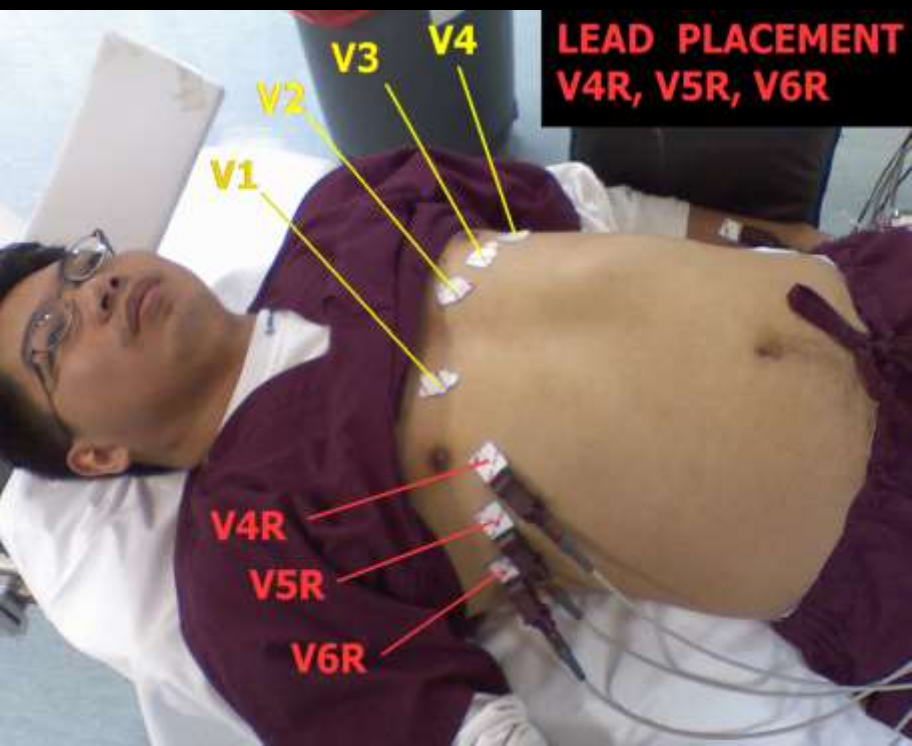
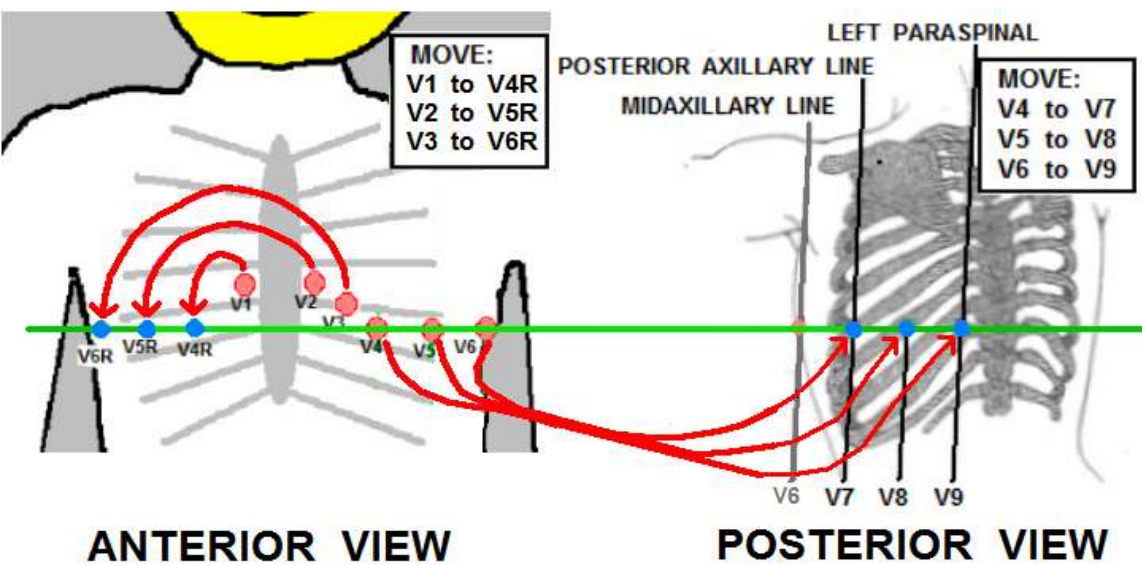
Whenever your patient's ECG exhibits ST DEPRESSION in any of the ANTERIOR LEADS (V1-V4), CONSIDER the possibility of POSTERIOR WALL STEMI !!

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



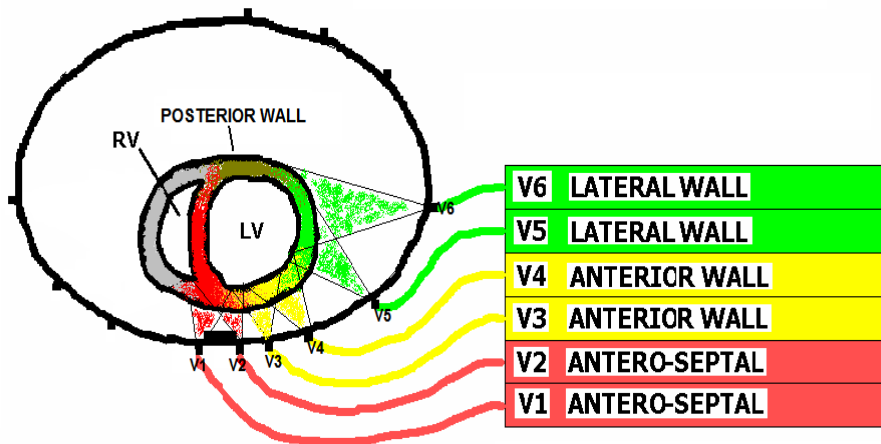


HOW TO REPOSITION 6 CHEST LEADS to OBTAIN 3 R VENTRICLE and 3 POSTERIOR LEADS



CHEST LEADS V1 - V6

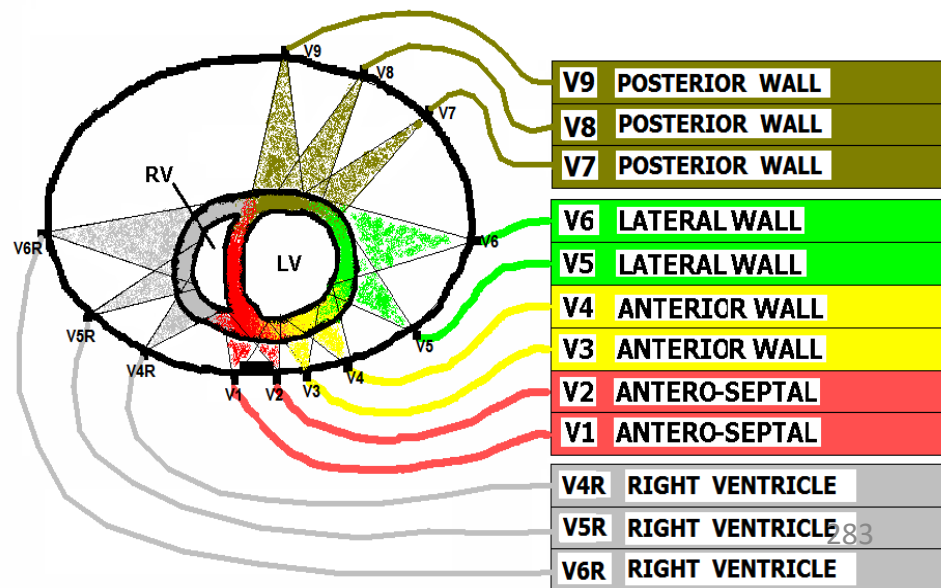
WHAT EACH LEAD "SEES" ...



⇐ The 12 Lead ECG

The 18 Lead ECG ⇒

CHEST LEADS V1 - V6 PLUS V4R, V5R, V6R, and V7, V8, V9
WHAT EACH LEAD "SEES" ...



34 years Vent. rate 58 bpm
Male Asian PR interval 146 ms
Room: QRS duration 82 ms
Opt: QT/QTc 372/365 ms
P-R-T axes 29 82 50

Sinus bradycardia
~~RSR' or QR pattern in V1 suggests right ventricular conduction delay~~
~~Cannot rule out Anteroseptal infarct, age undetermined~~
~~Abnormal ECG~~

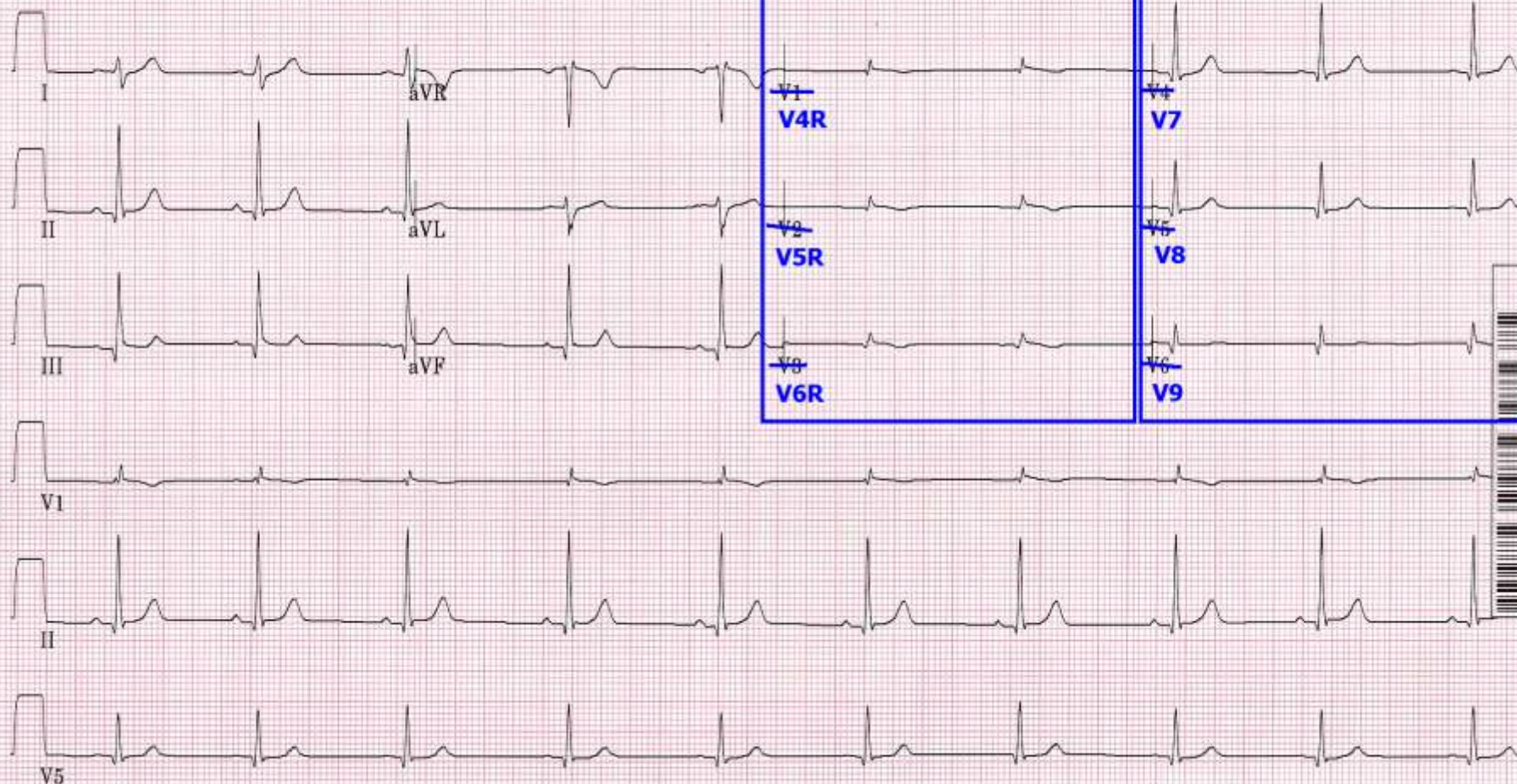
Technician: WR

DOS:

Referred by:

RIGHT VENTRICLE

POSTERIOR WALL



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*

Req Provider: CHARLES NOLES

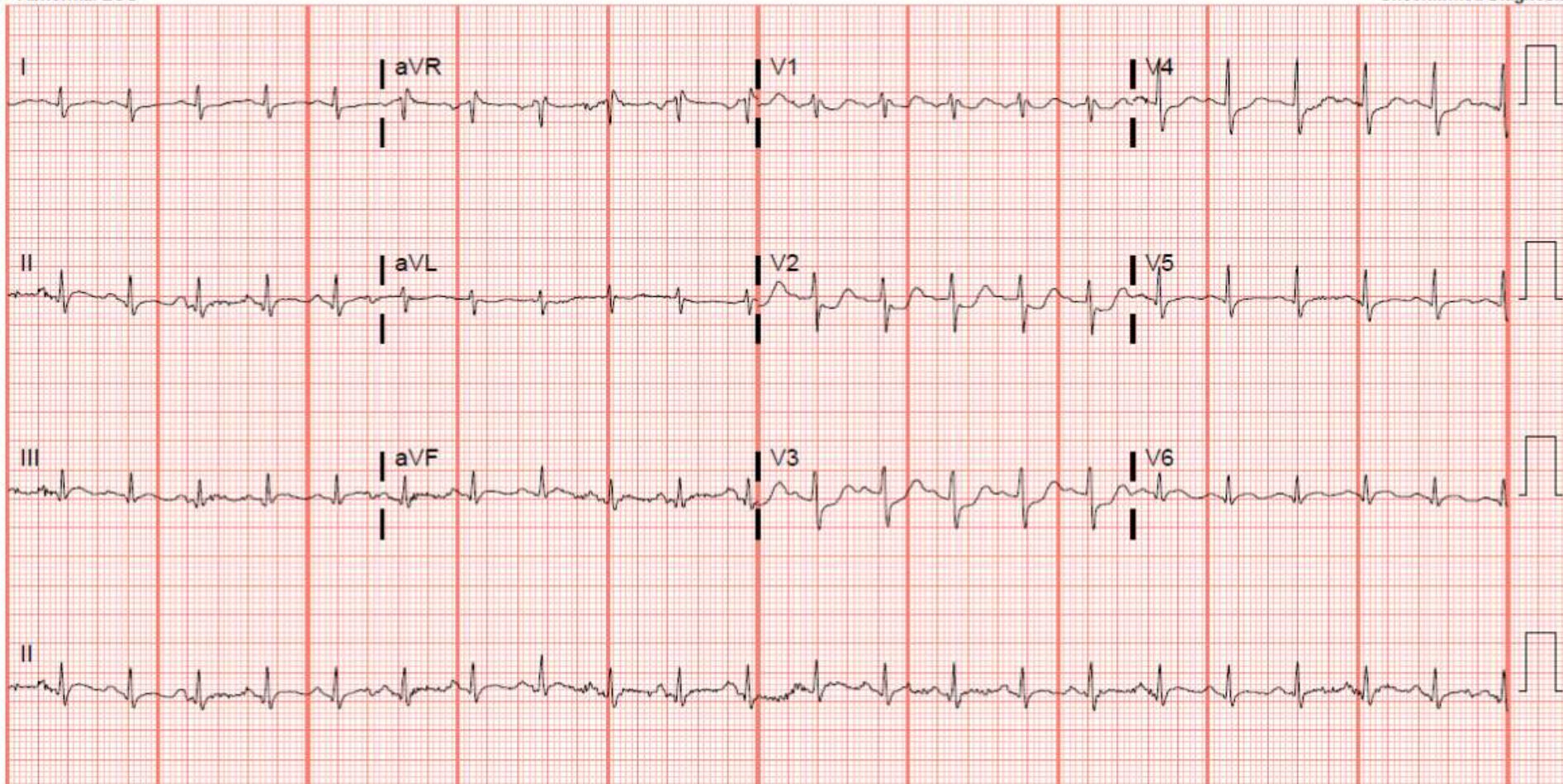
Rate	131	Sinus tachycardia
PR	128	Probable inferior infarct, old
QRSd	92	Posterior infarct, acute (LCx)
QT	317	ST depression V1-V3, suggest recording posterior leads
QTc	468	NO PREVIOUS ECG AVAILABLE FOR COMPARISON

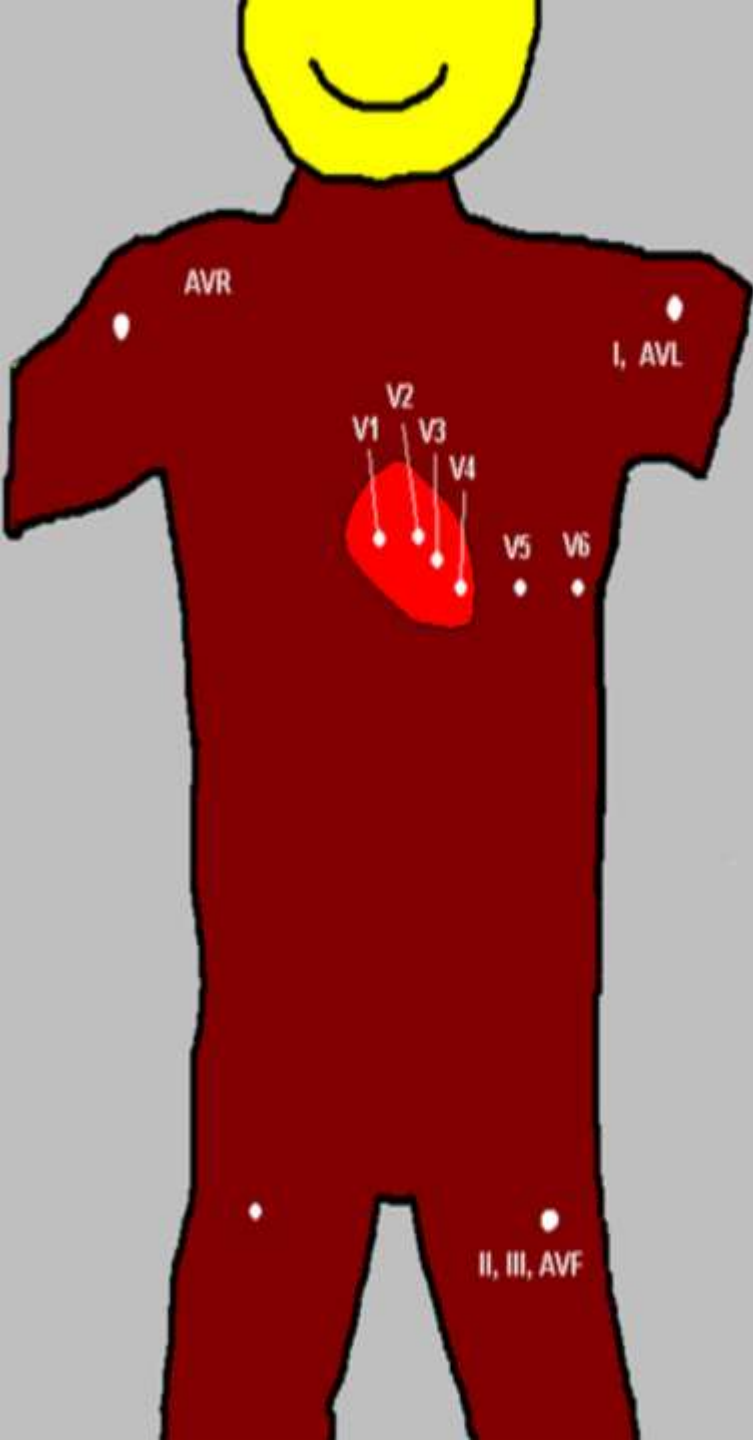
--Axis--

P	65
QRS	83
T	132

- Abnormal ECG -

Unconfirmed Diagnosis





AREAS VIEWED by 12 LEAD ECG

+ TYPICAL CORONARY ARTERIAL DISTRIBUTION

AVR *BASILAR SEPTAL*



1st SEPTAL PERFORATOR

AVL, I LATERAL
ANTERIOR



1st DIAGONAL or RAMUS or
1st OBTUSE MARGINAL

V1, V2 ANTERIOR



LEFT ANTERIOR DESCENDING

SEPTAL



LEFT ANTERIOR DESCENDING

POSTERIOR (recip.)



POSTERIOR LATERAL VESSELS

V3, V4 ANTERIOR



LEFT ANTERIOR DESCENDING

V5, V6 LATERAL



CIRCUMFLEX

II, III, AVF INFERIOR



RIGHT CORONARY ARTERY or
CIRCUMFLEX

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

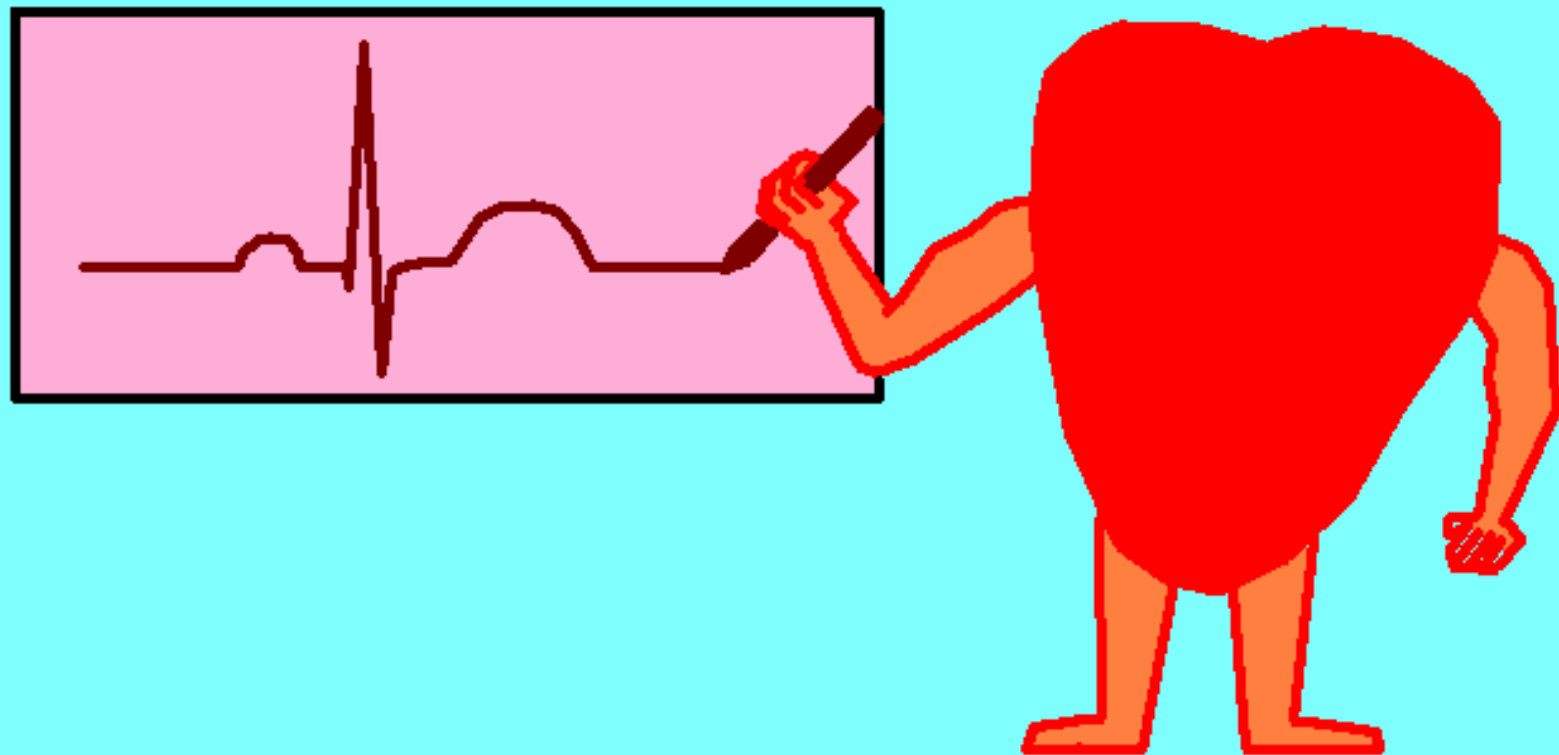
The other 10% of the population have a wide diversity of coronary arterial anatomies. Please see the **DOWNLOADABLE PDF** version of this presentation to view this optional material !!



“ROAD TO FOREVER,” Rt 385, Oklahoma panhandle, 1994

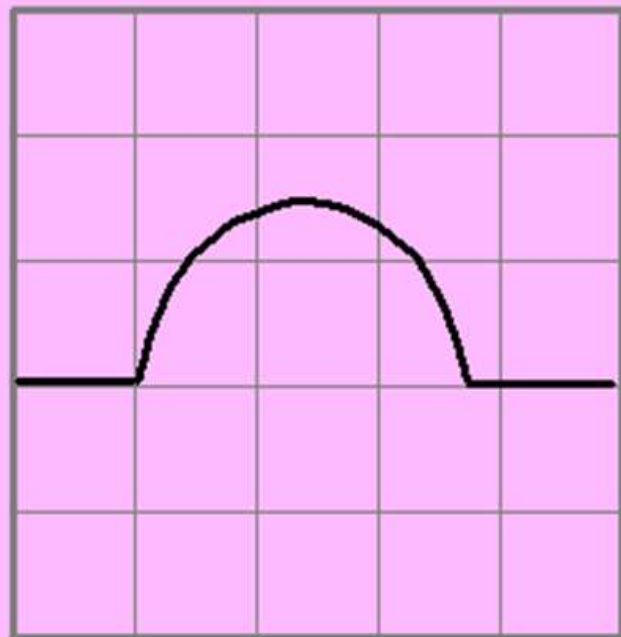
PUTTING IT ALL ON PAPER...

WAVEFORMS and INTERVALS . . .



THE P WAVE

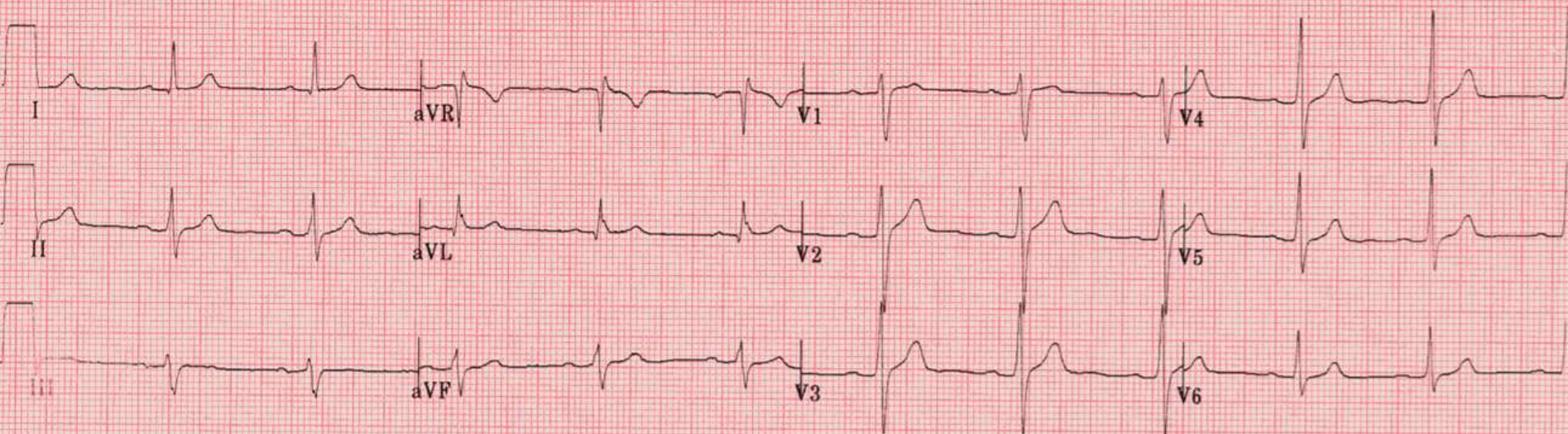
- SHOULD BE UPRIGHT, CONVEX-SHAPED DOME IN ALL LEADS EXCEPT AVR and V1



D.O.S.: TEST

Referred by:

Reviewed by:



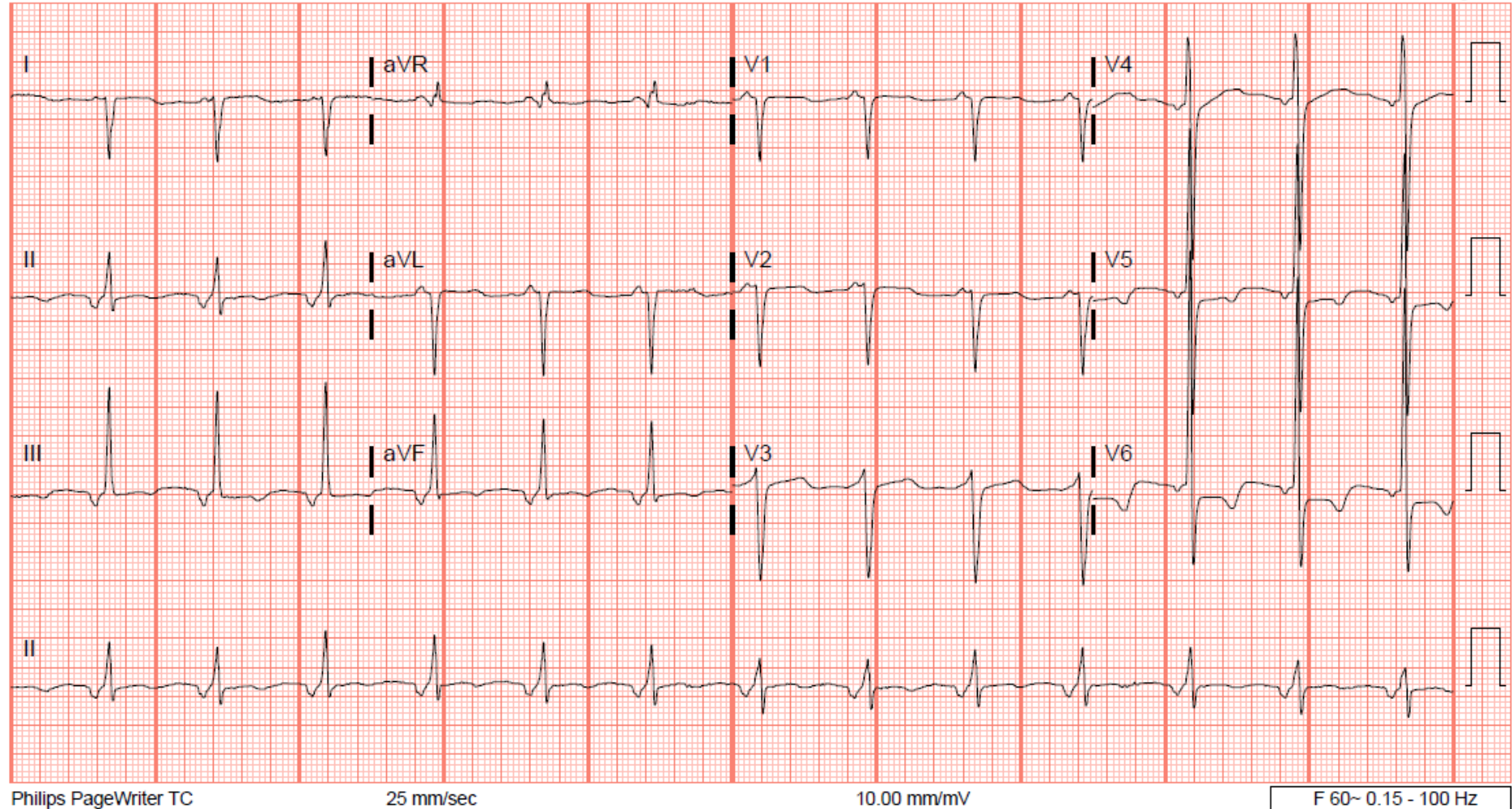
P Wave Axis

- P waves with abnormal axis (“not pointing in the right direction”) may signify ectopic atrial beats.
- When P waves are inverted in most leads with an abnormally short P-R interval ($<120\text{ms}$) the origin of the rhythm may be the AV node (Junctional Rhythm).

Inverted P waves & short P-R interval:

- Abnormal ECG -

Unconfirmed Diagnosis

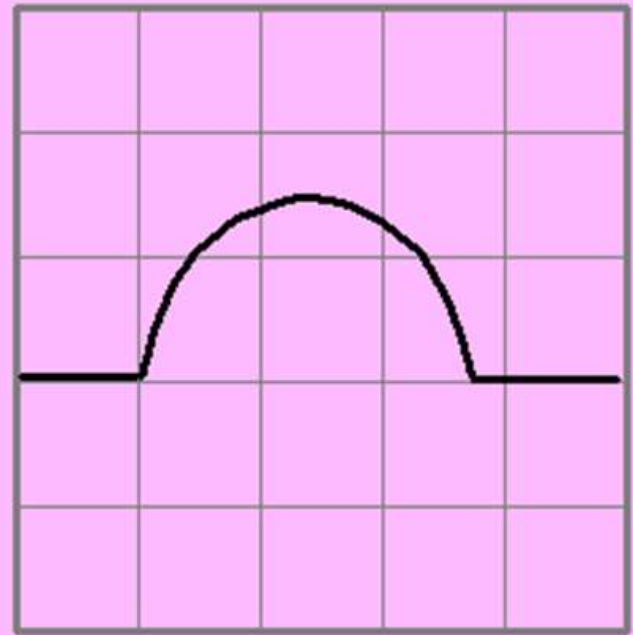


Evaluate P Wave for Atrial Hypertrophy

- Evaluate amplitude and duration in Lead II

THE P WAVE

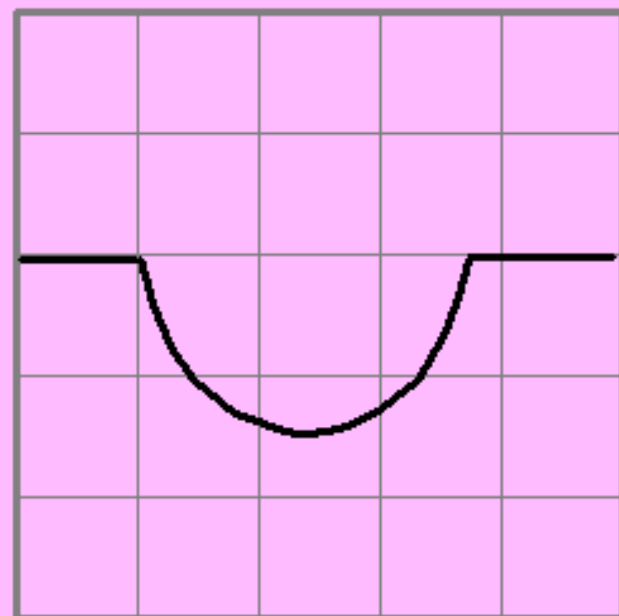
- SHOULD BE UPRIGHT, CONVEX-SHAPED DOME IN ALL LEADS EXCEPT AVR and V1
- SHOULD BE LESS THAN .2 mv (2 mm) HIGH
- SHOULD BE LESS THAN 100 ms (2.5mm) LONG



THE P WAVE

- SHOULD BE INVERTED IN LEAD AVR

LEAD AVR



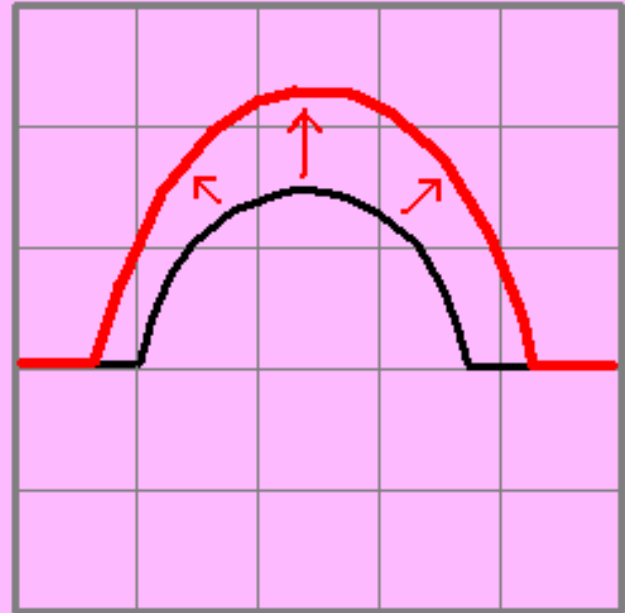
THE P WAVE

When the P WAVE
is

TOO LARGE

We think of

ATRIAL HYPERTROPHY



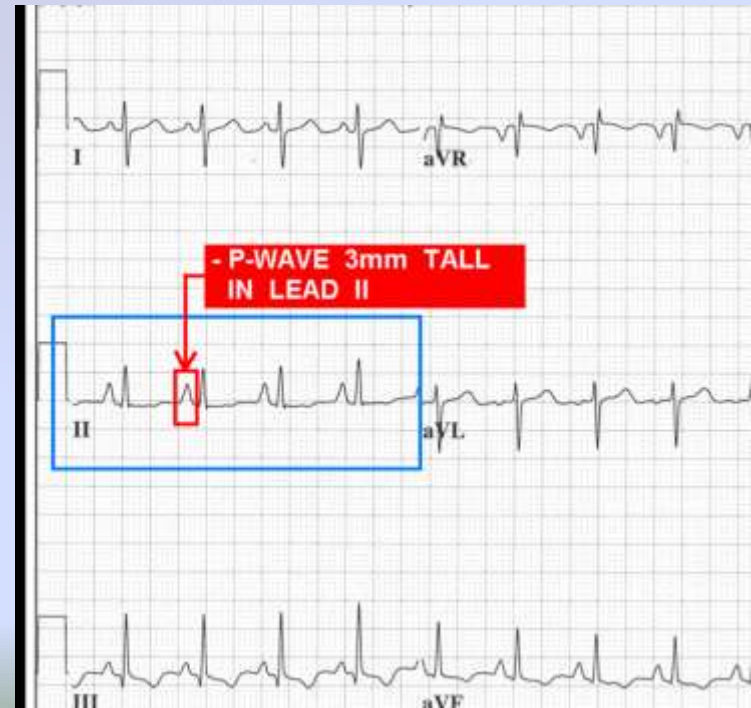
(SPECIFIC CRITERIA FOR ATRIAL HYPERTROPHY IS DISCUSSED IN
MORE DETAIL IN THE "CHAMBER HYPERTROPHY" SECTION)

Evaluate P Wave for Atrial Hypertrophy

- Evaluate amplitude and duration in *Lead II*

Evaluate P Wave for Atrial Hypertrophy

- Evaluate amplitude and duration in Lead II
- If the P wave is “too tall (>2mm) or too long (>2.5mm)” in Lead II, ***then go to Lead V1*** to evaluate P wave



THE P WAVE

IN LEAD V1 MAY BE:

- POSITIVE

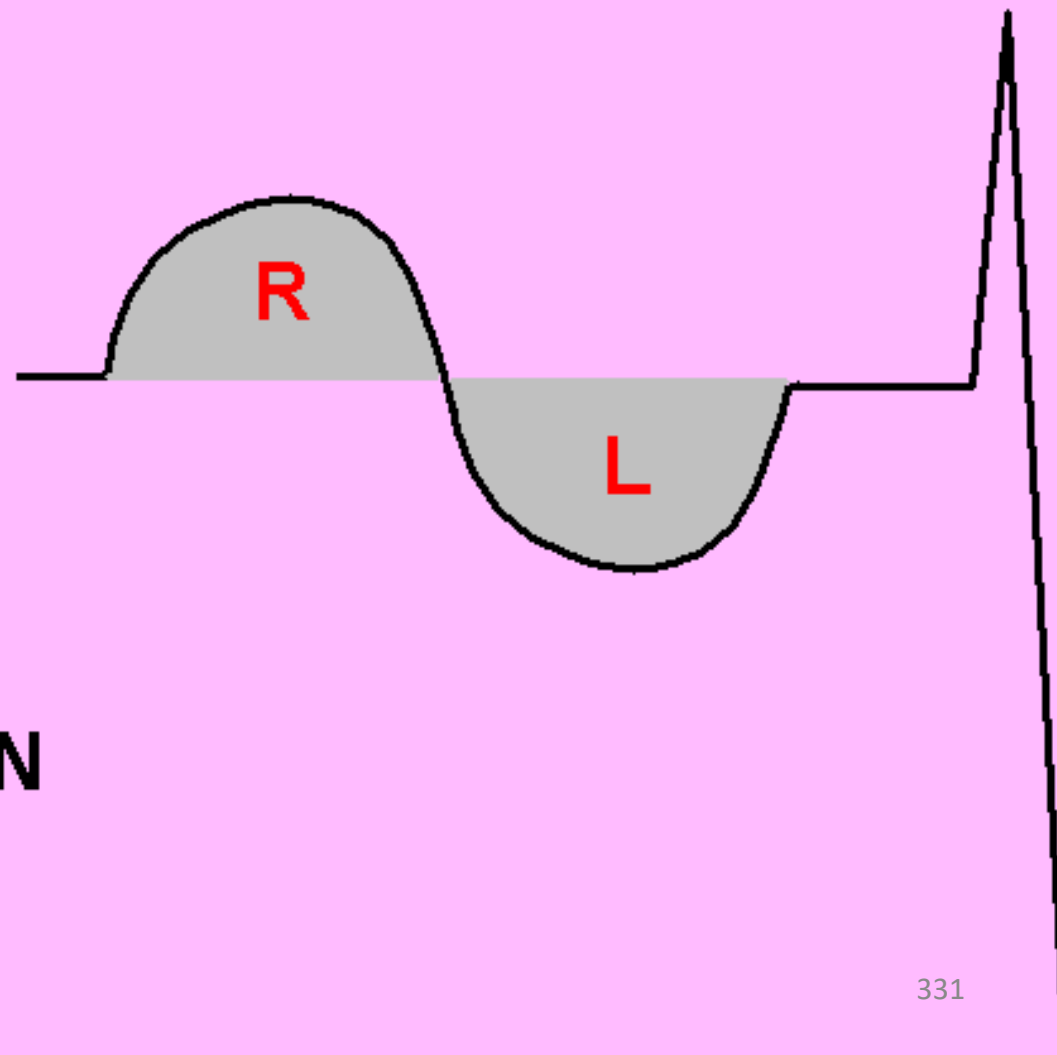


- OR BI-PHASIC



THE P WAVE

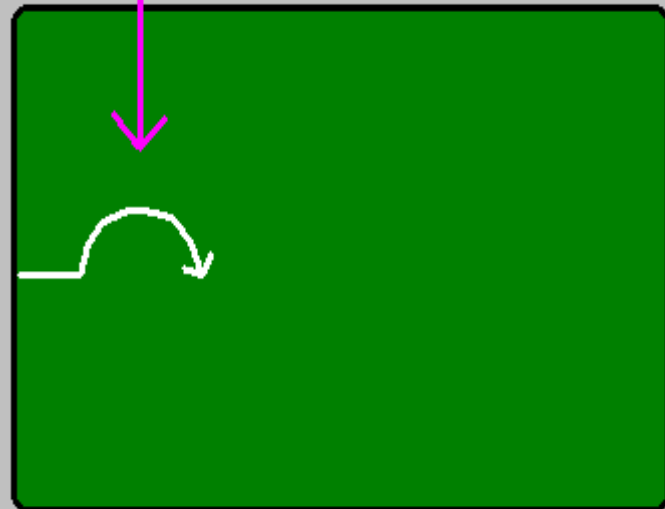
- WHEN THE P WAVE IS BI-PHASIC IN V1, IT DISPLAYS BOTH R and L ATRIAL DEPOLARIZATION



RIGHT ATRIAL DEPOLARIZATION

FIRST 1/2 of
P WAVE

LEAD
V1



A diagram of a human torso showing a heart model. A red arrow points from the heart towards the lungs, indicating the direction of blood flow. The heart is labeled with a red cross and a red arrow. The lungs are labeled with a red cross and a red arrow. The diagram is labeled with 'G' and 'H'.

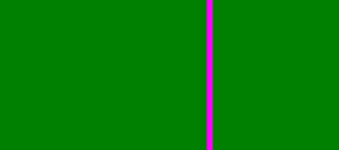


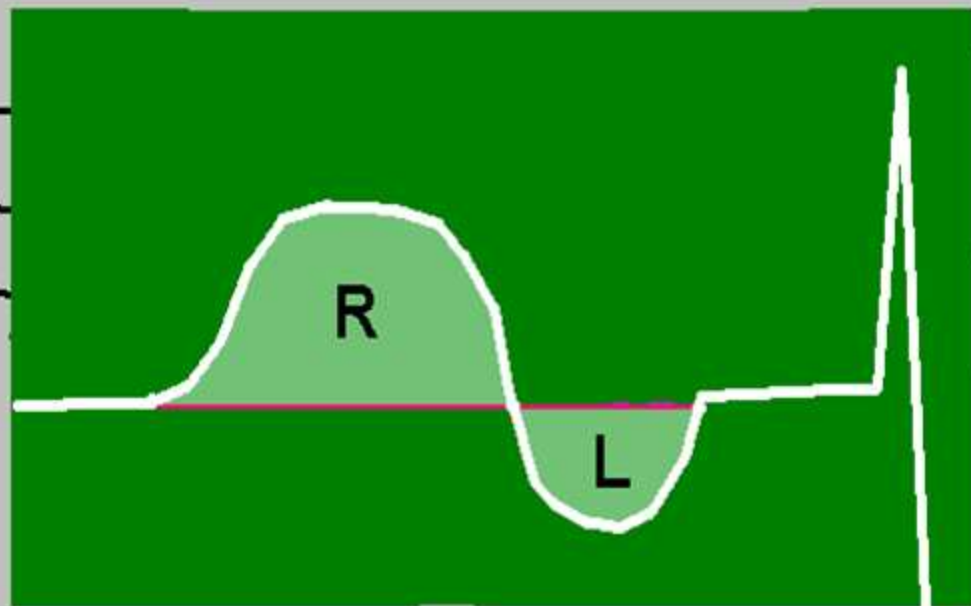
Diagram of ECG lead V1. A green rectangular area represents the lead field. A white line shows the ECG waveform, which starts flat, then rises to a peak and falls to a trough. A pink arrow points down to the peak of the waveform. To the right of the green area, the text "LEAD V1" is displayed.

Evaluate P Wave for Atrial Hypertrophy

- Evaluate amplitude and duration in Lead II
- If the P wave is “too tall ($>2\text{mm}$) or too long ($>2.5\text{mm}$)” in Lead II, ***then go to lead V1*** to evaluate P wave.
- In Lead V1, if the first half (positive deflection) of the P wave is LARGER than the second half (negative deflection) it suggests RIGHT ATRIAL HYPERTROPHY (RAH).

RIGHT ATRIAL ENLARGEMENT

P-WAVE IN V1



02-DEC-1998 00:24:45

ST. JOSEPH'S HOSPITAL-ER ROUTINE RETRIEVAL

29 yr
Male Black
Room:ER
Loc:3 Option:28

Vent. rate 107 BPM
PR interval 132 ms
QRS duration 80 ms
QT/QTc 310/413 ms
P-R-T axes 67 105 -32

EKG CLASS #WR03446043

Sinus tachycardia

Right atrial enlargement

Rightward axis

Pulmonary disease pattern

RSR' or QR pattern in V1 suggests right ventricular

T wave abnormality, consider inferior ischemia

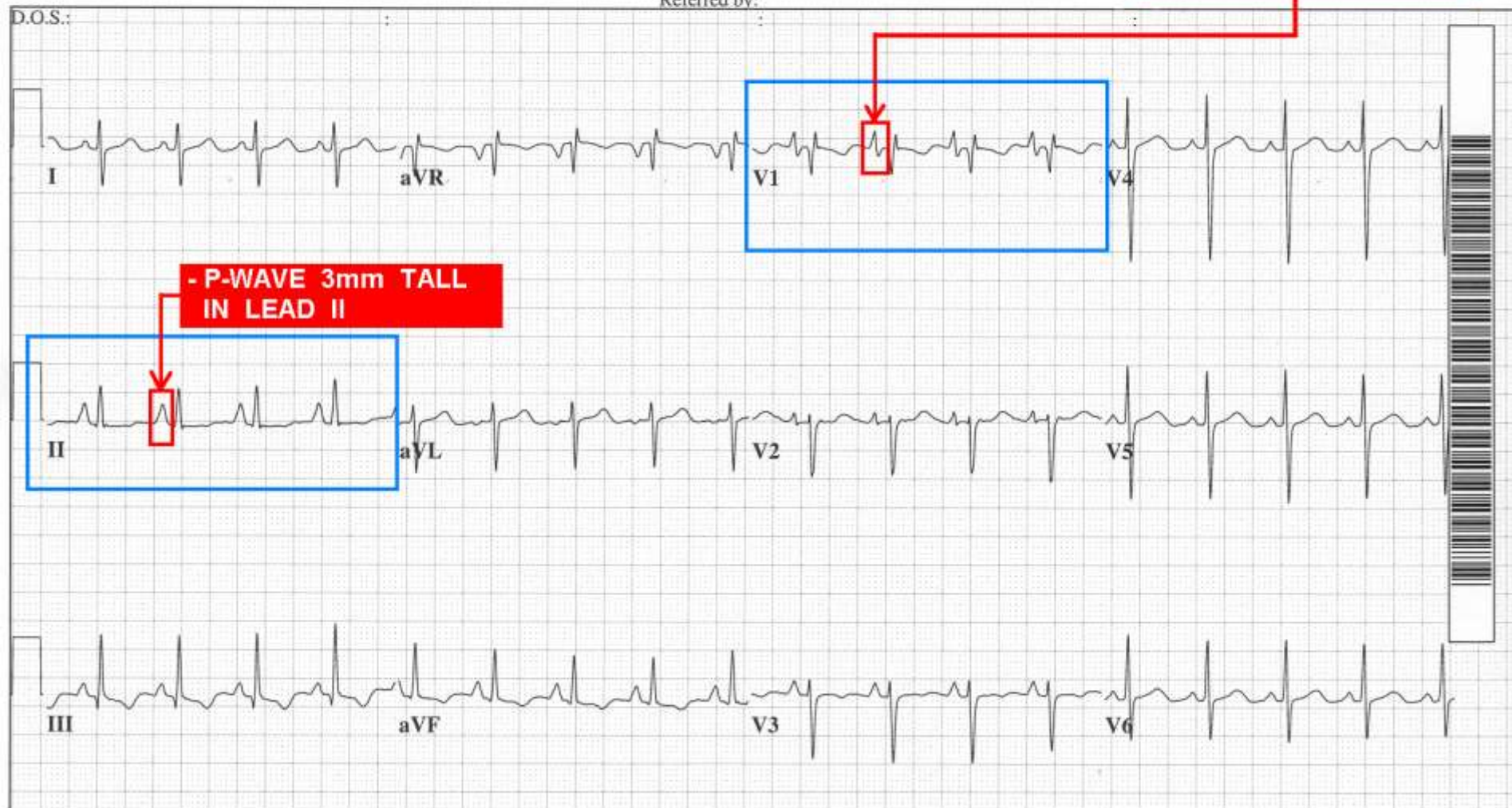
Abnormal ECG

When compared with ECG of 01-OCT-1998 21:45

T wave inversion more evident in Inferior leads ...

Referred by:

- POSITIVE DEFLECTION
TALLER (more
dominant) IN LEAD
V1

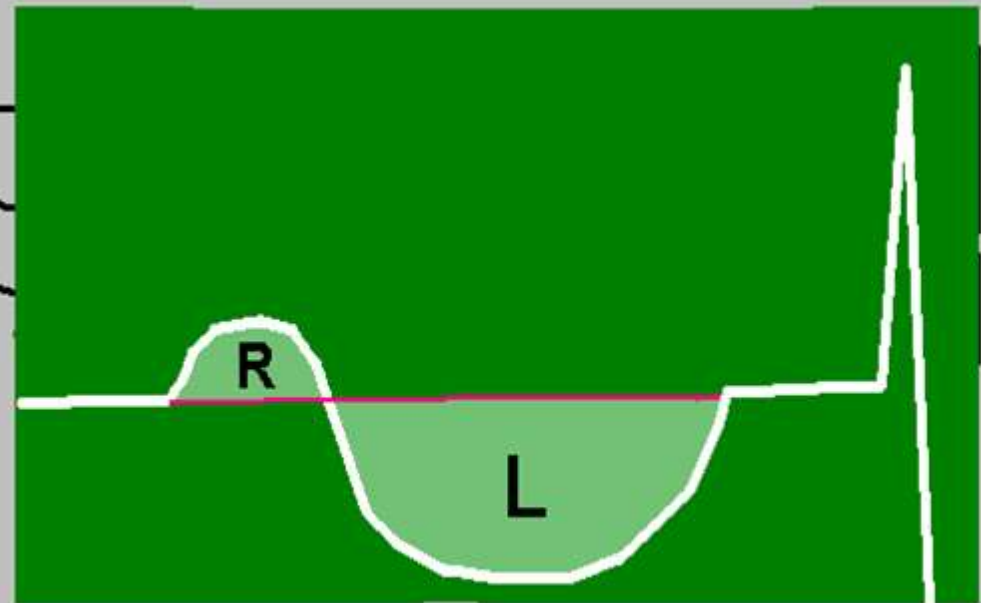


Evaluate P Wave for Atrial Hypertrophy

- Evaluate amplitude and duration in Lead II
- If the P wave is “too tall ($>2\text{mm}$) or too long ($>2.5\text{mm}$)” in Lead II, ***then go to lead V1*** to evaluate P wave.
- In Lead V1, if the first half (positive deflection) of the P wave is LARGER than the second half (negative deflection) it suggests RAH. **If the second half (negative deflection) is larger, it suggests LEFT ATRIAL HYPERTROPY (LAH).**

LEFT ATRIAL ENLARGEMENT

P-WAVE IN V1



77 yr
Male Caucasian
Room: S 1
Loc: 3 Option: 10

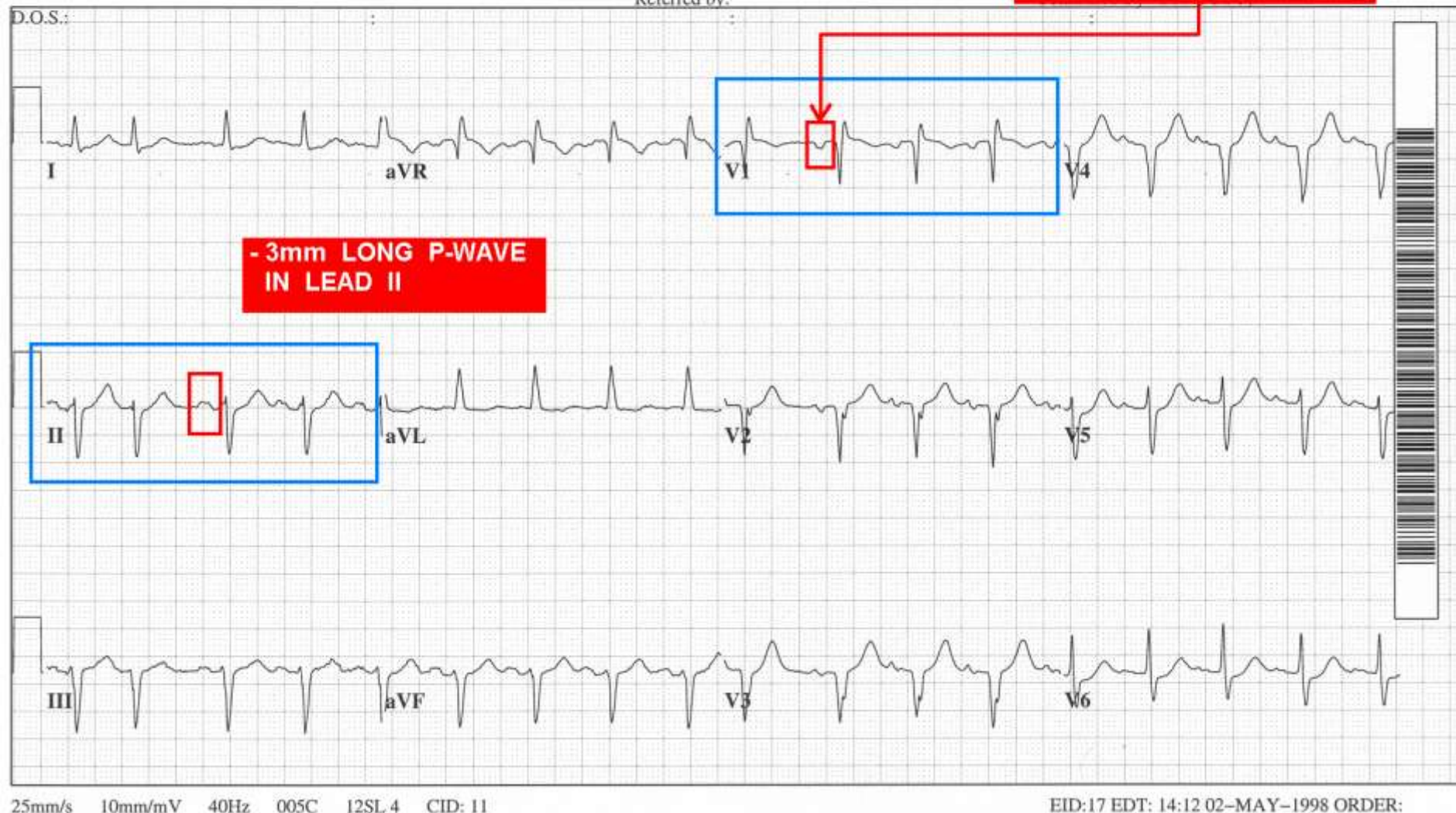
Vent. rate 106 BPM
PR interval 170 ms
QRS duration 104 ms
QT/QTc 374/496 ms
P-R-T axes 67 -66 70

Sinus tachycardia with occasional Premature supraventricular complexes
Left atrial enlargement
Left axis deviation
Incomplete right bundle branch block
Anteroseptal infarct, age undetermined
Abnormal ECG
No previous ECGs available

**- NEGATIVE DEFLECTION
P-WAVE IN LEAD V1**

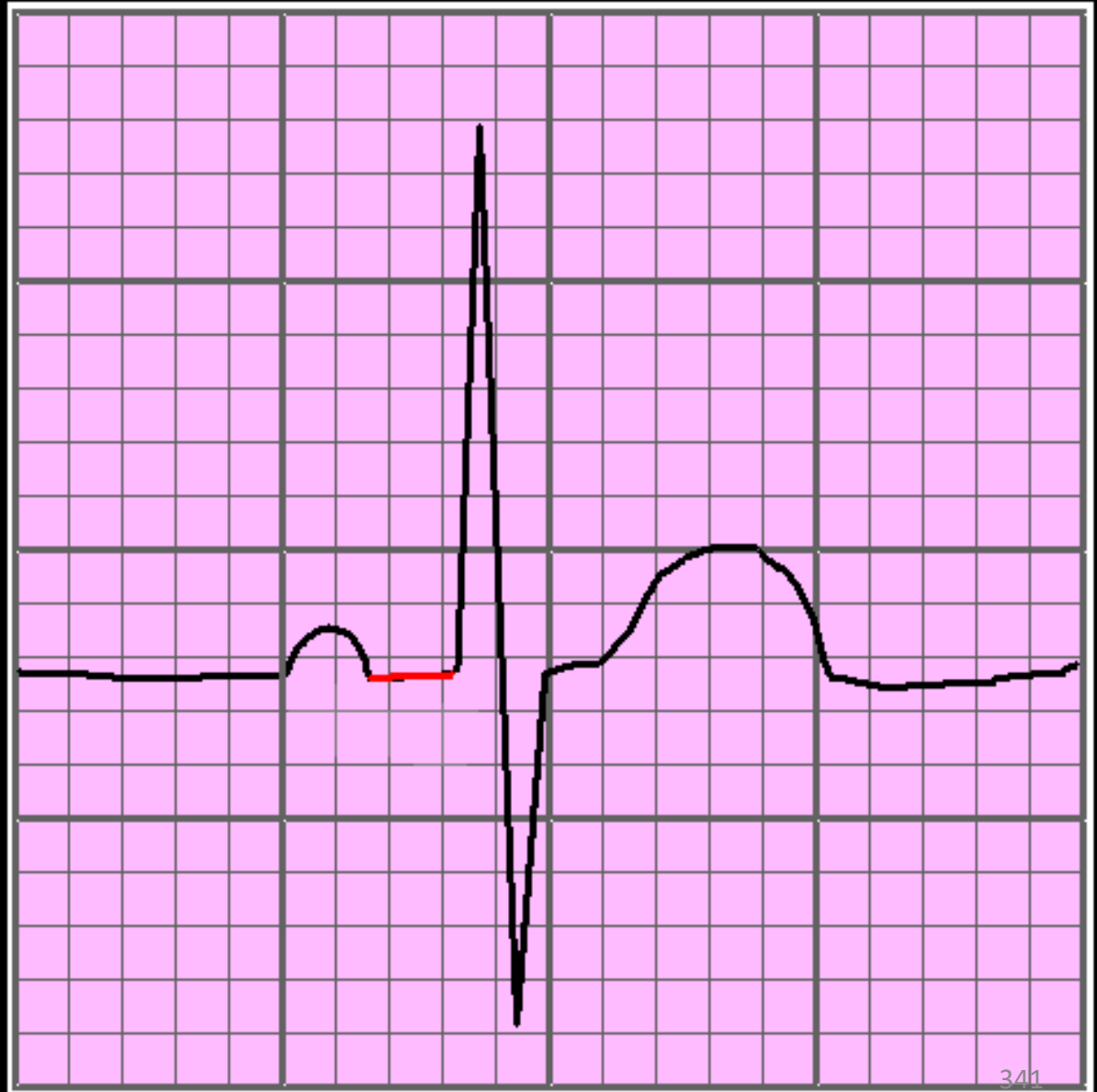
EKG CLASS #WR03651849

Referred by:



THE P-R SEGMENT

SHOULD
RETURN TO
THE
ISO-
ELECTRIC
LINE.



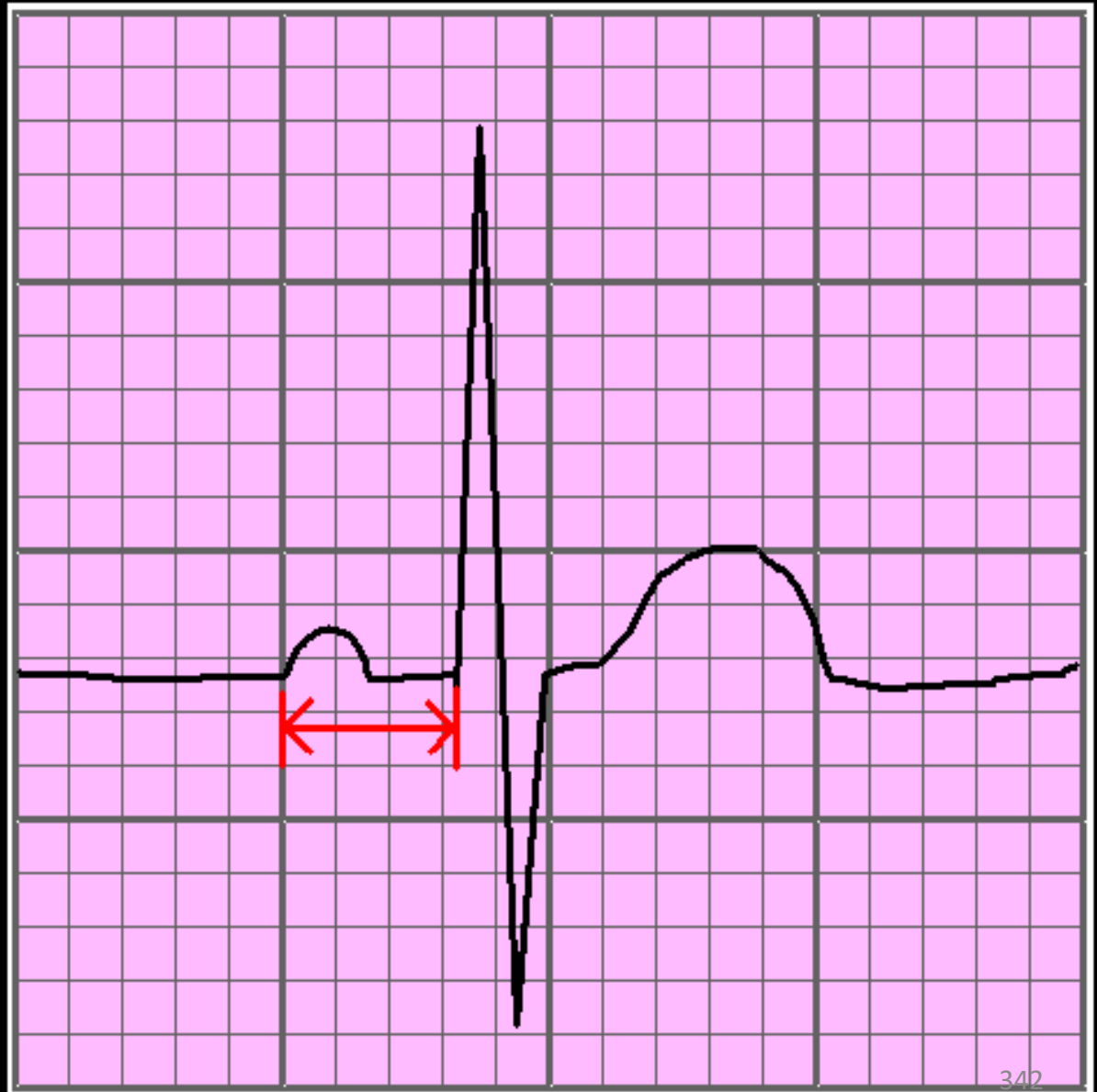
NORMAL P-R INTERVAL

.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 QRS COMPLEX

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



THE QRS COMPLEX

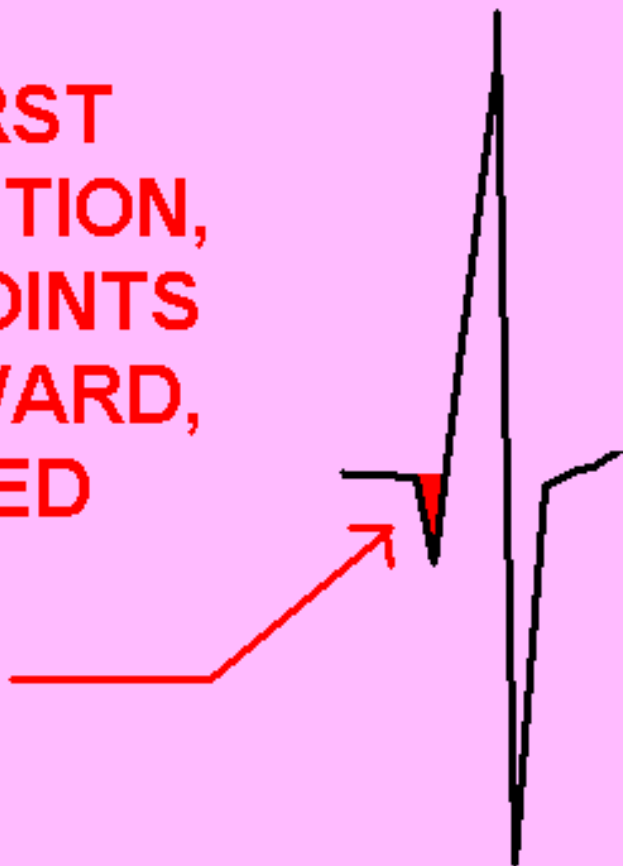
THIS QRS COMPLEX CONSISTS OF
3 DEFLECTIONS



THE QRS COMPLEX

THIS QRS COMPLEX CONSISTS OF
3 DEFLECTIONS

THE FIRST
DEFLECTION,
IF IT POINTS
DOWNWARD,
IS NAMED
THE "Q
WAVE"



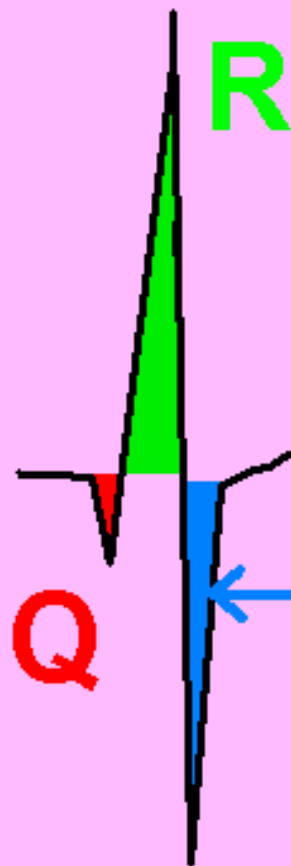
THE QRS COMPLEX

THIS QRS COMPLEX CONSISTS OF
3 DEFLECTIONS



THE QRS COMPLEX

THIS QRS COMPLEX CONSISTS OF
3 DEFLECTIONS

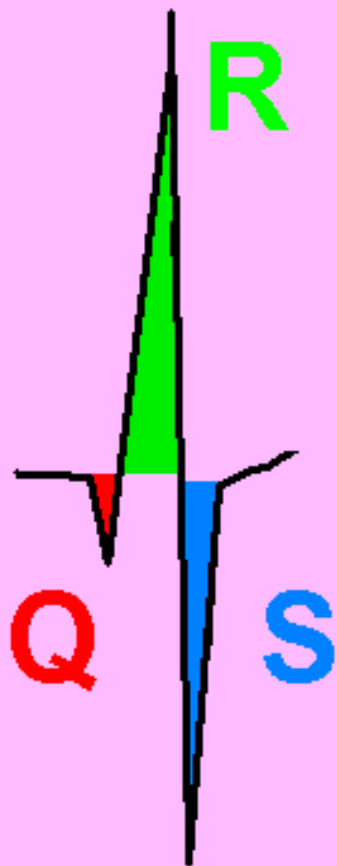


A NEGATIVE
DEFLECTION
AFTER THE
R WAVE IS
CALLED THE
" S " WAVE

THE QRS COMPLEX

THIS QRS COMPLEX CONSISTS OF
3 DEFLECTIONS

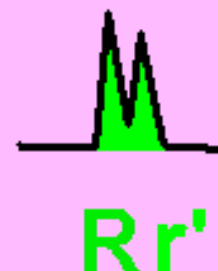
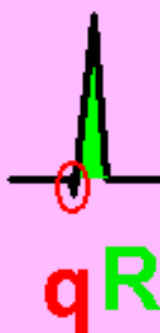
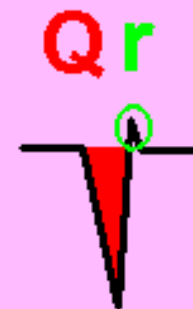
AND IS
THE ONLY
TRUE
"QRS"
COMPLEX



SOME OF
THE OTHER
VARIATIONS
INCLUDE

THE QRS COMPLEX

WHAT ARE THESE COMPLEXES ??



QRS INTERVAL

LESS THAN

.12

OR

120 mSEC



QRS COMPLEX TOO WIDE

WIDER THAN 120 mSEC

THINK:

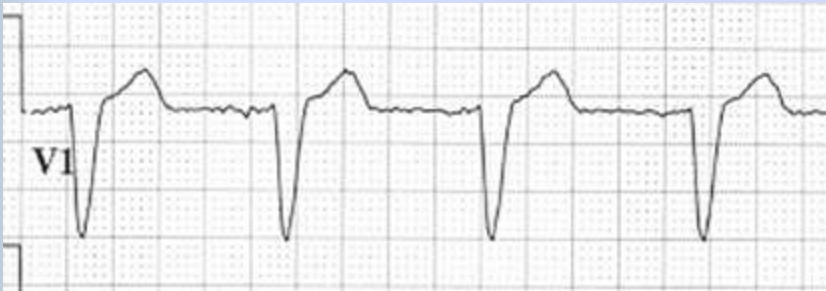
- BUNDLE BRANCH BLOCK
- **VENTRICULAR COMPLEX (ES)**
- PACED RHYTHM
- L VENTRICULAR HYPERTROPHY
- **ELECTROLYTE IMBAL. ($\uparrow K^+$ $\downarrow Ca^{++}$)**
- DELTA WAVE (PRE-EXCITATION)

When the QRS is WIDE (> 3mm):

- If you KNOW the Rhythm is originating ABOVE the Ventricles (such as NSR or any Supraventricular Rhythm) – you should determine if the QRS has a RIGHT or LEFT Bundle Branch Block morphology.

Normal Sinus and Other “Supraventricular Rhythms” with **WIDE QRS (> 120 ms)**

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



Simple “Turn Signal Method” . . .

THE “TURN SIGNAL METHOD” for identifying BUNDLE BRANCH BLOCK

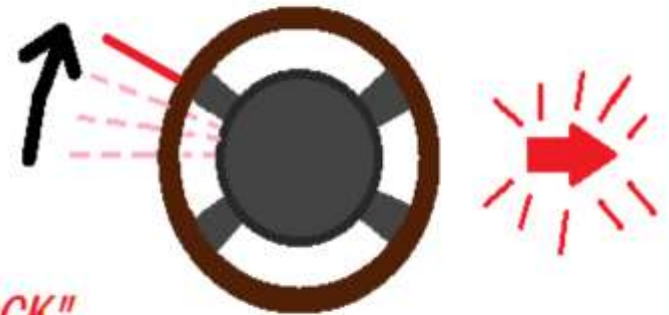
V1

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”

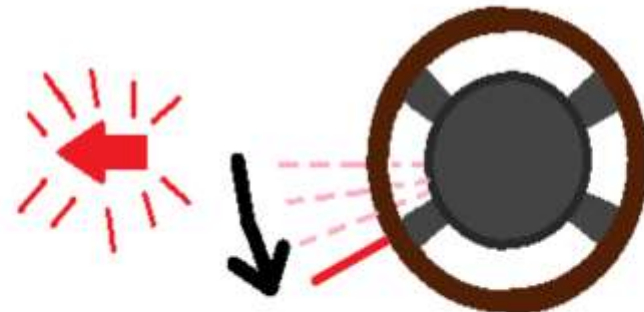


V1

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

THINK:

“QRS points DOWN = LEFT BUNDLE BRANCH BLOCK”



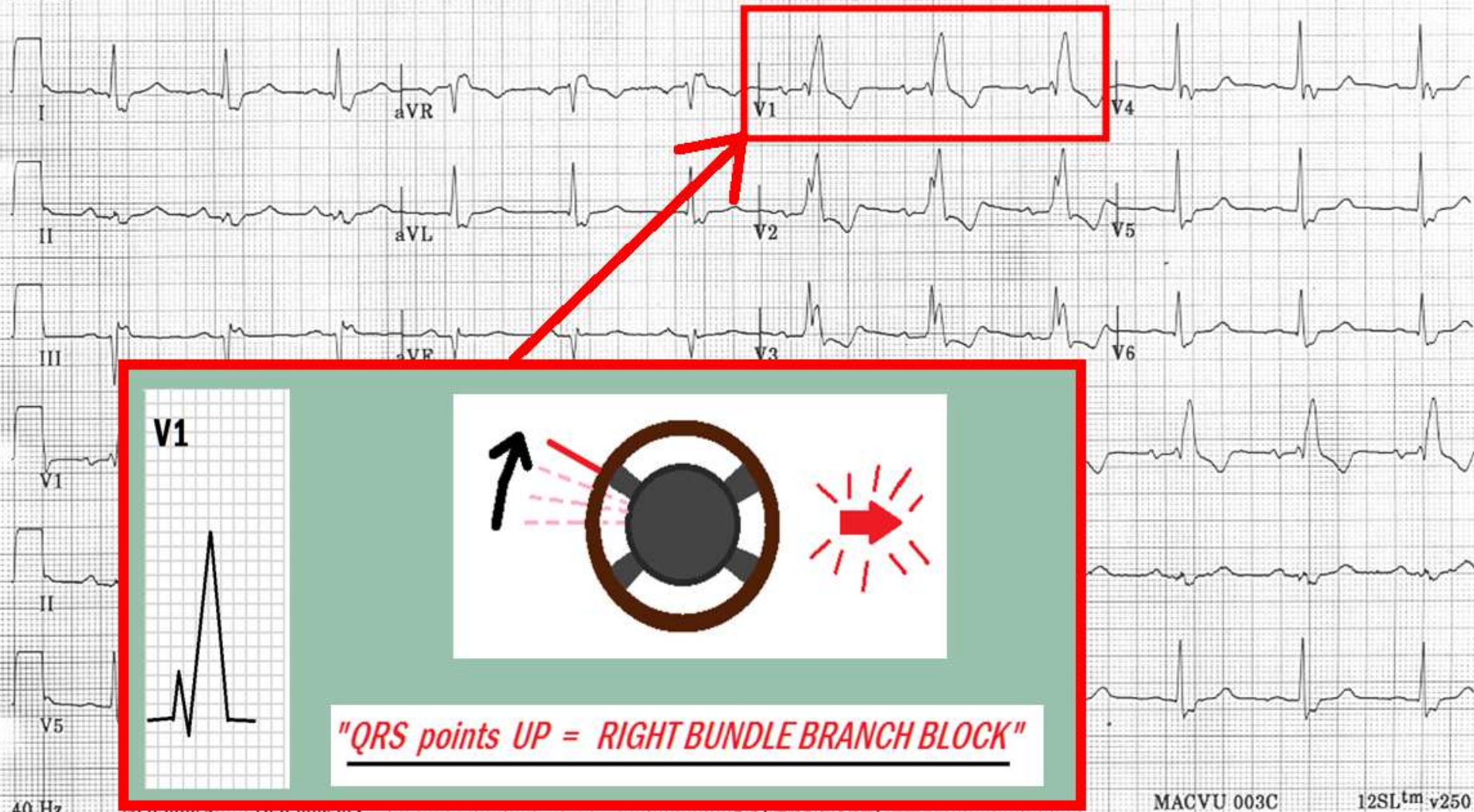
74years
Male Caucasian
Room: Loc: 0
Opt:
Technician: WR

Vent. rate 72 bpm
PR interval 186 ms
QRS duration 166 ms
QT/QTc 436/477 ms
P-R-T axes 57 -32 32

Normal sinus rhythm
Left axis deviation
Right bundle branch block
Inferior infarct, age undetermined
Abnormal ECG

USE LEAD V1 for this technique

D.O.S.:



09:16:40

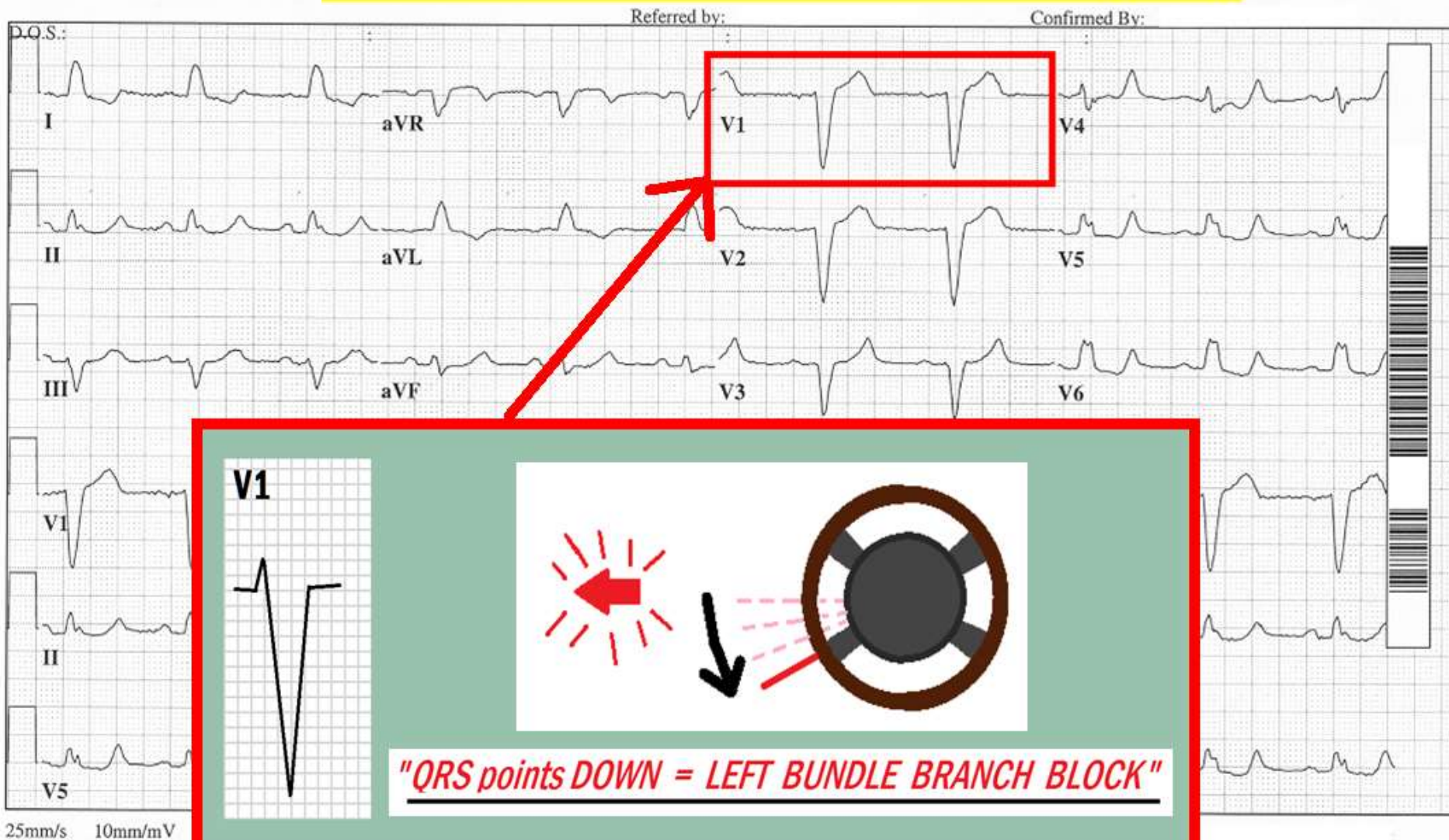
74 yr
Female Caucasian

Vent. rate 64 BPM
PR interval 188 ms
QRS duration 152 ms
QT/QTc 472/486 ms
P-R-T axes 78 3 106
EKG #1A1D03020050

Normal sinus rhythm
Left bundle branch block
Abnormal ECG
When compared with ECG of 28-MAY-2003 06:36,

Technician: WW

USE LEAD V1 for this technique



*More on
Determining
Right – vs – Left
Bundle Branch Block
in Session 2.*

When the QRS is WIDE (> 3mm):

- If you KNOW the Rhythm is originating ABOVE the Ventricles (such as NSR or any Supraventricular Rhythm) – you should determine if the QRS has a RIGHT or LEFT Bundle Branch Block morphology.
- **If you SUSPECT the rhythm is VENTRICULAR in origin, then EVALUATE THE “NADIR”**

THE QRS COMPLEX



Point of NADIR

Measurement from BEGINNING OF QRS COMPLEX to the TIP of the DOMINANT WAVEFORM of the complex in LEAD V1 or V6.

WHY ? ?

It is one (good) clue to help us discern VENTRICULAR beats vs. SUPRA-VENTRICULAR beats with abberancy.

THE QRS COMPLEX

*** How ??**

-- Supraventricular favors < 70ms

-- Ventricular favors > 70ms



* Marriott, Henry J.L. -- a measurement of 70ms or greater from the beginning of the QRS to the peak of the R wave or nadir of the S wave suggestive of rhythms of Ventricular origin.

THE QRS COMPLEX

QRS HEIGHT

is a reflection of the
QRS AMPLITUDE.

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



THE QRS COMPLEX

QRS AMPLITUDE

is influenced by:

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



THE QRS COMPLEX

QRS AMPLITUDE

is measured by finding the **TALLEST POSITIVE DEFLECTION (R WAVE)** and the **DEEPEST NEGATIVE DEFLECTION (S WAVE)** 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

Referred by:

Confirmed By:

TALLEST R WAVE is in LEAD V4 = 11 mm

DEEPEST S WAVE is in LEAD V2 = 8 mm

OVERALL QRS AMPLITUDE = 19 mm

THE QRS COMPLEX

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 !**

14-JUL-1997 14:30:58

ST. JOSEPH'S HOSPITAL-ER ROUTINE RETRIEVAL

17 yr
Male Black
Room:ER
Loc:3 Option:16

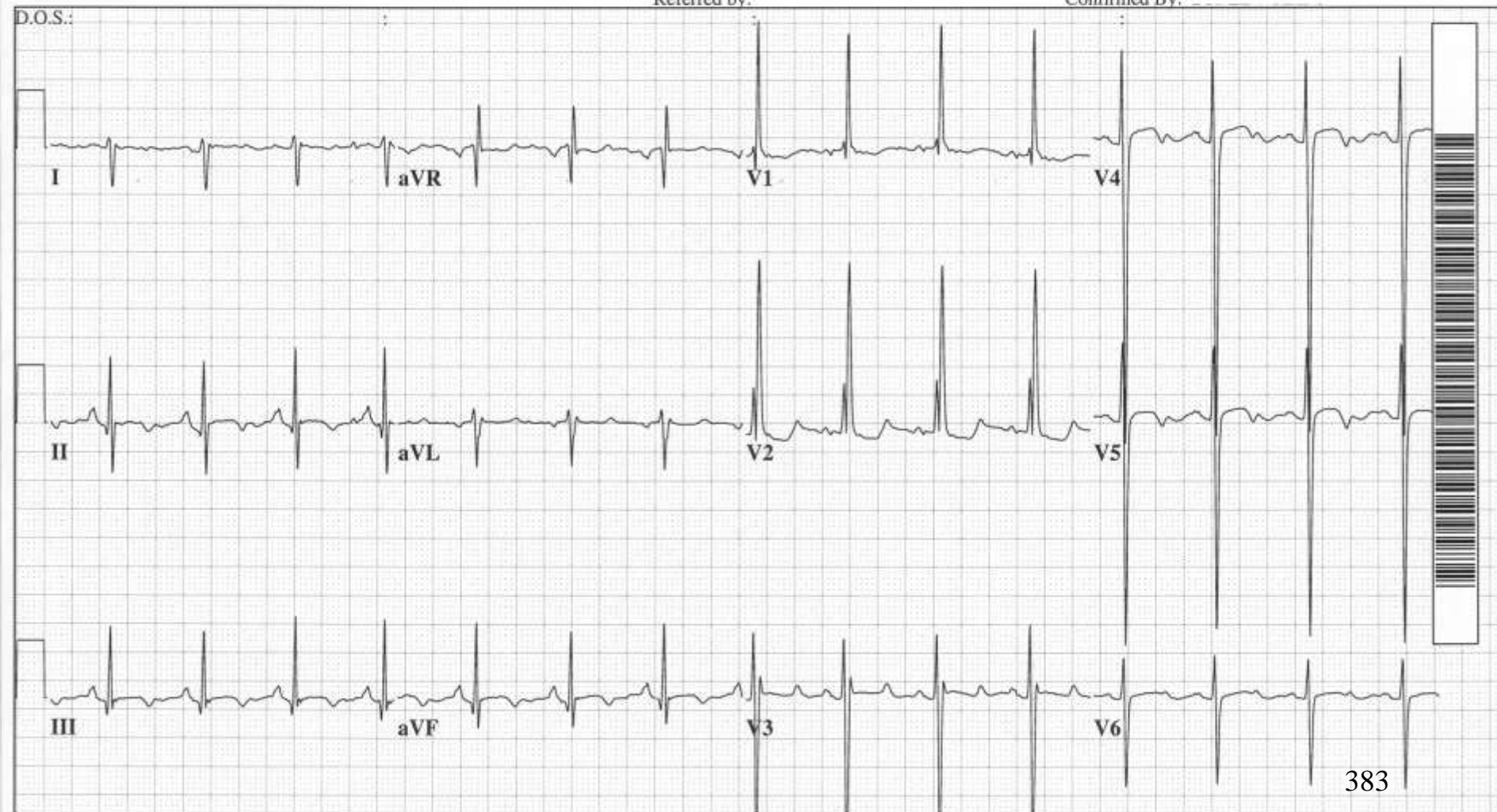
Vent. rate 90 BPM
PR interval 136 ms
QRS duration 94 ms
QT/QTc 378/462 ms
P-R-T axes 77 123 58

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

Referred by:

Confirmed By:



25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 11

EID:11 EDT: 17:04 15-JUL-1997 ORDER:

53 yr
Male Caucasian
Room: ER S3
Loc: 3 Option: 18

Vent. rate 100 BPM
PR interval 198 ms
QRS duration 186 ms
QT/QTc 380/490 ms
P-R-T axes 79 163 -20

Normal sinus rhythm
Left atrial enlargement
Right bundle branch block, plus right ventricular hypertrophy
Left posterior fascicular block
*** Bifascicular block ***

NONSPECIFIC ST CHANGES

Abnormal ECG

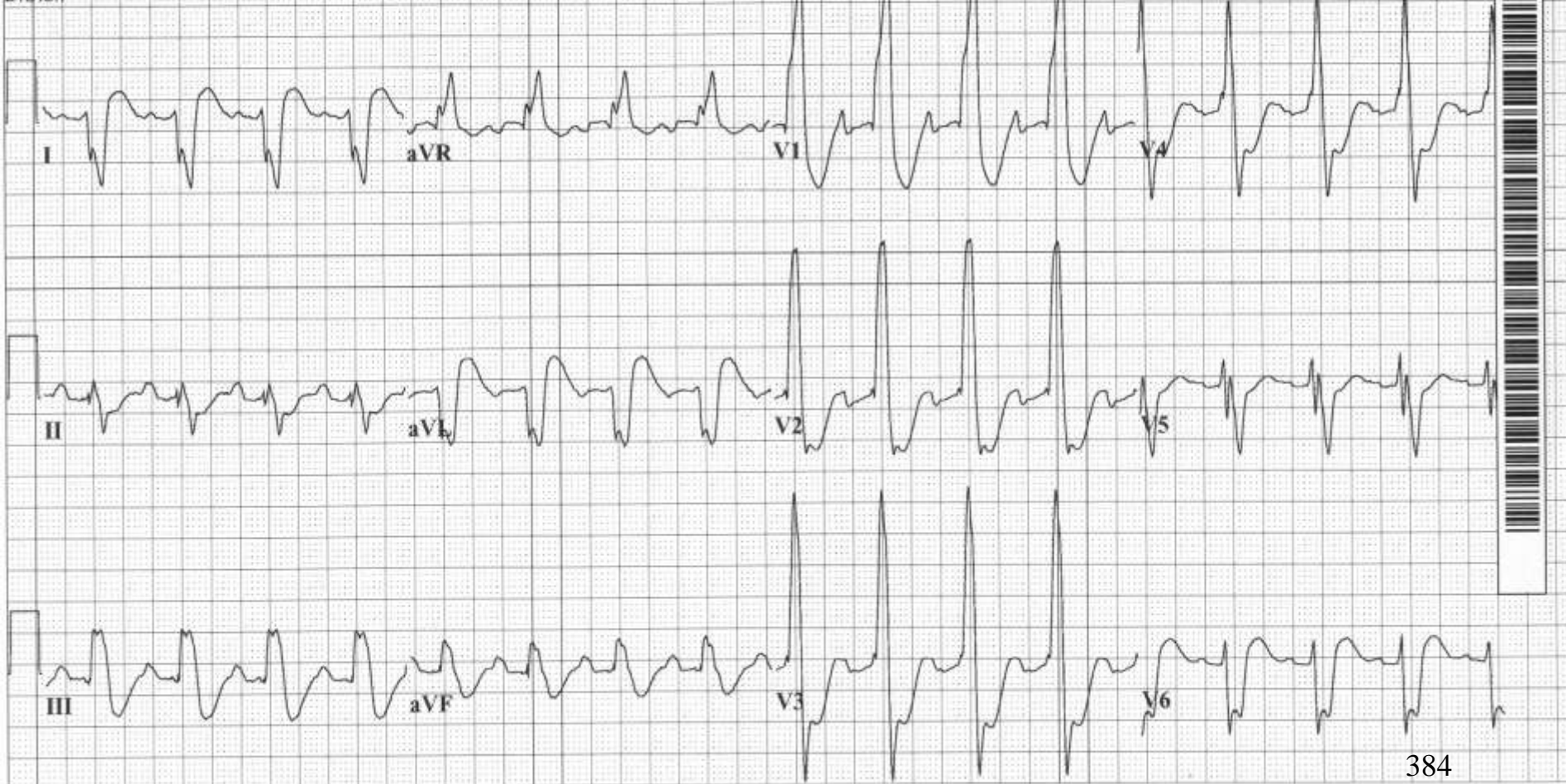
When compared with ECG of 21-APR-1996 11:44,
No significant change was found

EKG CLASS #WR03028722

Referred by:

Confirmed By:

D.O.S.:



384

53 yr
Male Black
Room:ER
Loc:3 Option:23

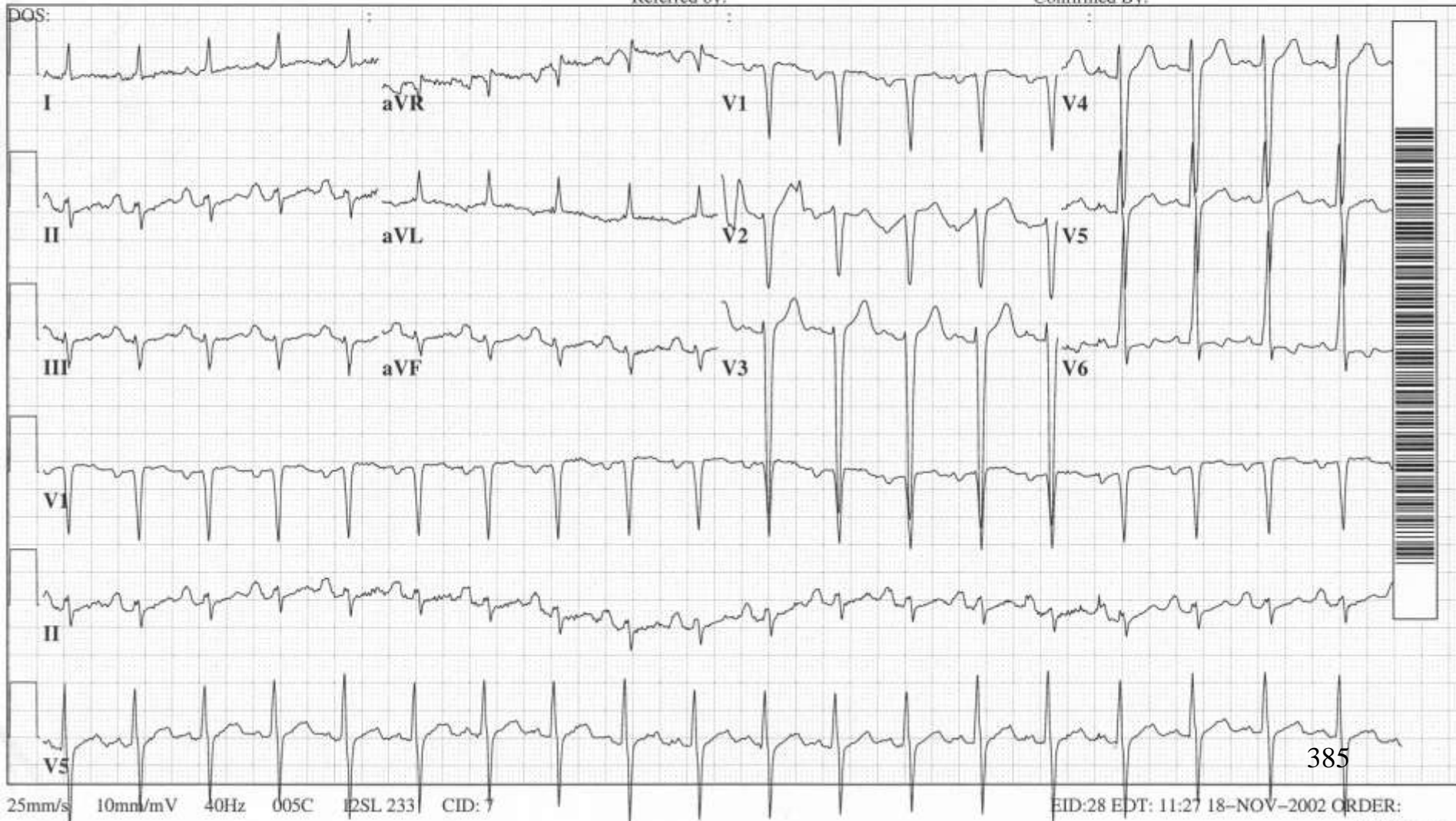
Vent. rate 115 BPM
PR interval 160 ms
QRS duration 92 ms
QT/QTc 316/437 ms
P-R-T axes 76 -39 59

****UNEDITED COPY - REPORT IS COMPUTER GENERATED ONLY, WITHOUT
PHYSICIAN INTERPRETATION**
Sinus tachycardia
Possible Left atrial enlargement
Left axis deviation
Left ventricular hypertrophy
Abnormal ECG
No previous ECGs available

EKG CLASS #WR03896717

Referred by:

Confirmed By:



61 yr
Male Black
Loc:7 Option:35

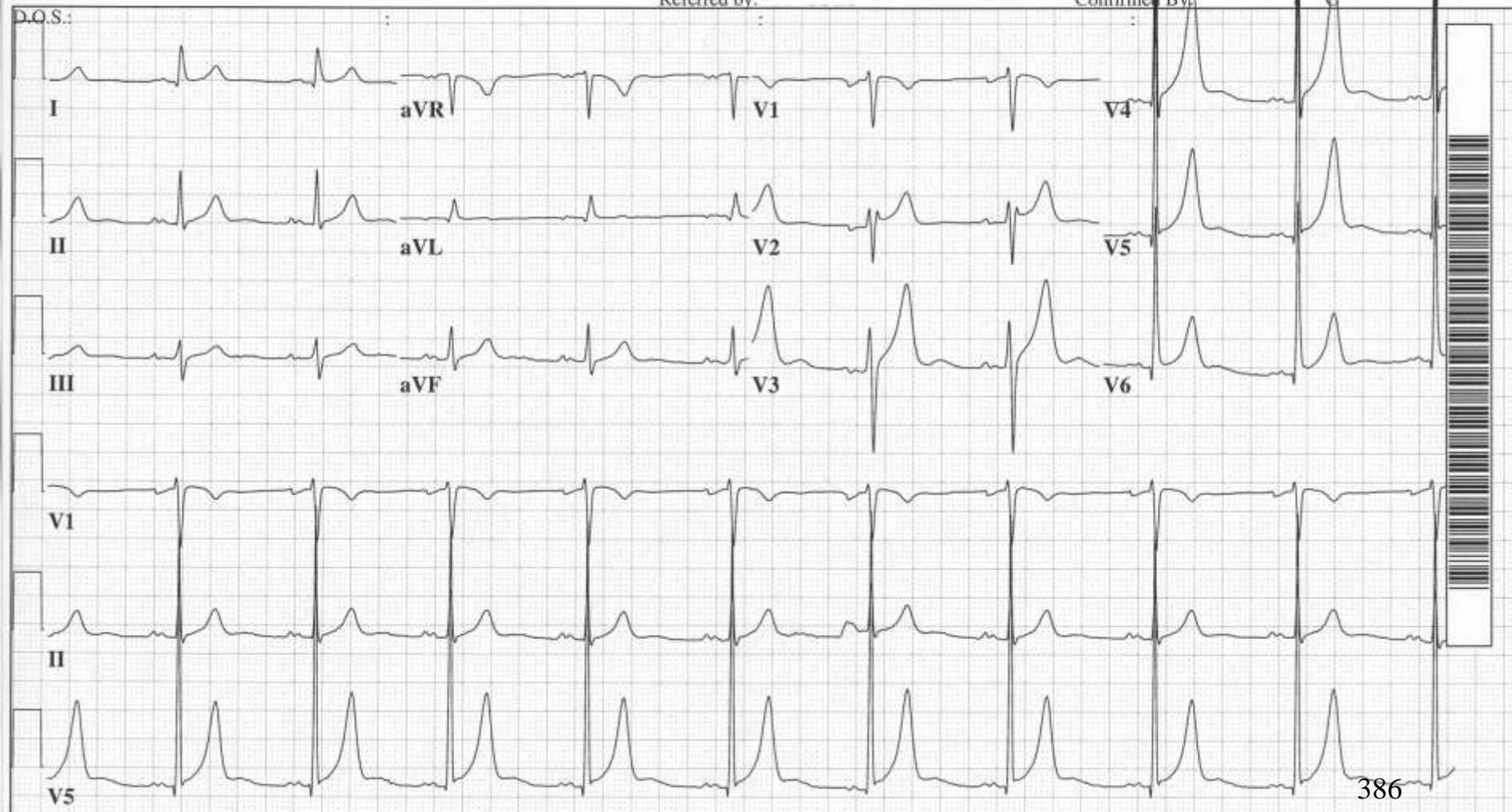
Vent. rate	60	BPM
PR interval	176	ms
QRS duration	90	ms
QT/QTc	400/400	ms
P-R-T axes	62 33	60

Normal sinus rhythm
Voltage criteria for left ventricular hypertrophy
Abnormal ECG
When compared with ECG of 02-SEP-2002 09:00,
Vent. rate has decreased BY 44 BPM

EKG CLASS #WR03503400

Referred by:

Confirmed By:



THE QRS COMPLEX

QRS AMPLITUDE

CRITERIA FOR MINIMUM AMPLITUDE:

Abnormally LOW QRS VOLTAGE occurs when the OVERALL QRS is:

$\leq 0.5 \text{ mV}$ IN ANY LIMB LEAD

— *and* —

$\leq 1.0 \text{ mV}$ 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)

33 yr
Female Black

Room:ATL
Loc:3 Option:23

Vent. rate	132	BPM
PR interval	154	ms
QRS duration	76	ms
QT/QTc	282/417	ms
P-R-T axes	51 17	-80

***unedited copy: report is computer generated only, without physician interpretation".
*** Age and gender specific ECG analysis ***
Sinus tachycardia with occasional , and consecutive
Premature ventricular complexes
Low voltage QRS
ST elevation consider anterolateral injury or acute infarct
***** ACUTE MI *****
Abnormal ECG
No previous ECGs available

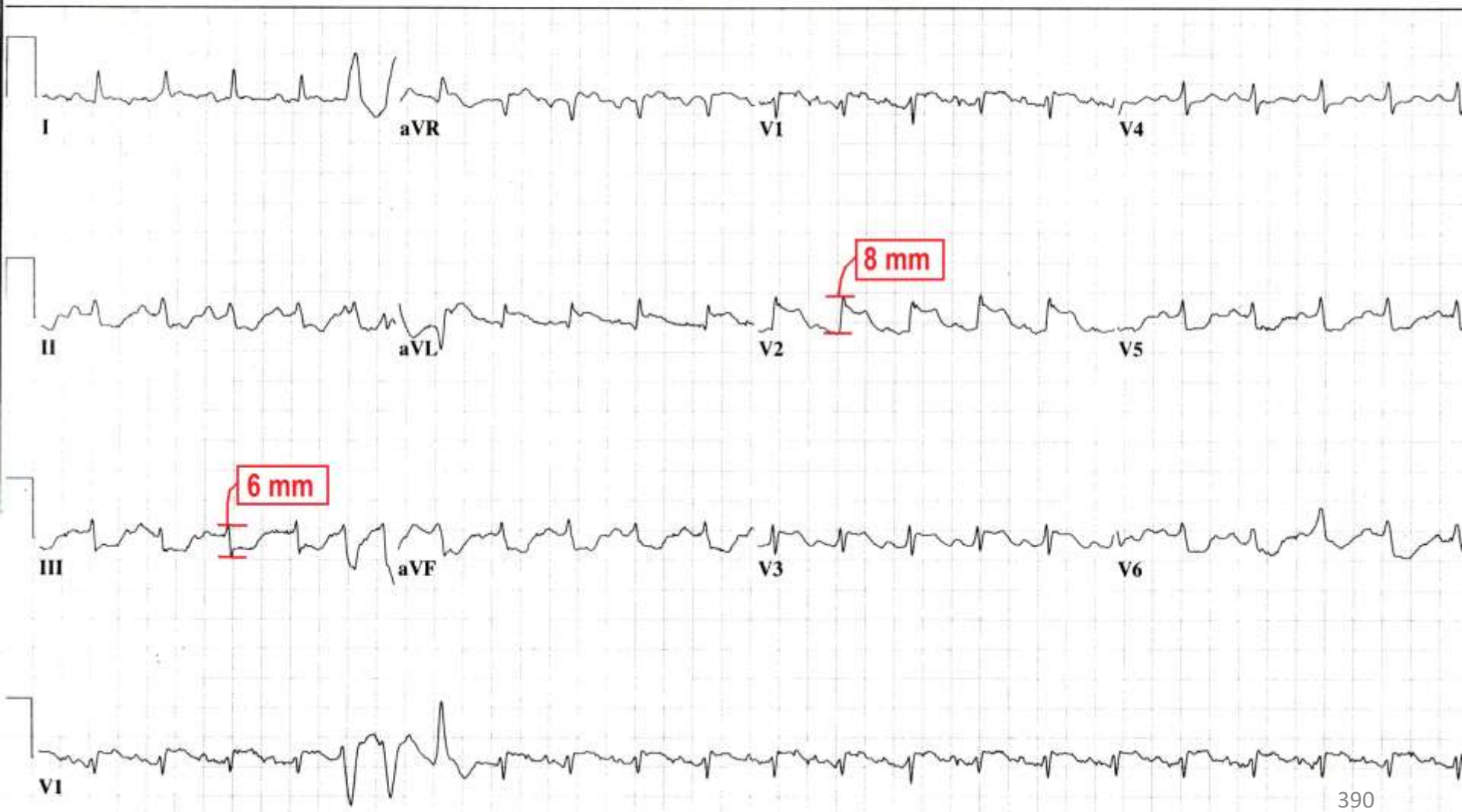


33 yr
Female Black

Room:ATL
Loc:3 Option:23

Vent. rate 132 BPM
PR interval 154 ms
QRS duration 76 ms
QT/QTc 282/417 ms
P-R-T axes 51 17 -80

***unedited copy: report is computer generated only, without physician interpretation".
*** Age and gender specific ECG analysis ***
Sinus tachycardia with occasional , and consecutive
Premature ventricular complexes
Low voltage QRS
ST elevation consider anterolateral injury or acute infarct
***** ACUTE MI *****
Abnormal ECG
No previous ECGs available



• Q WAVES •

Normal Q Waves

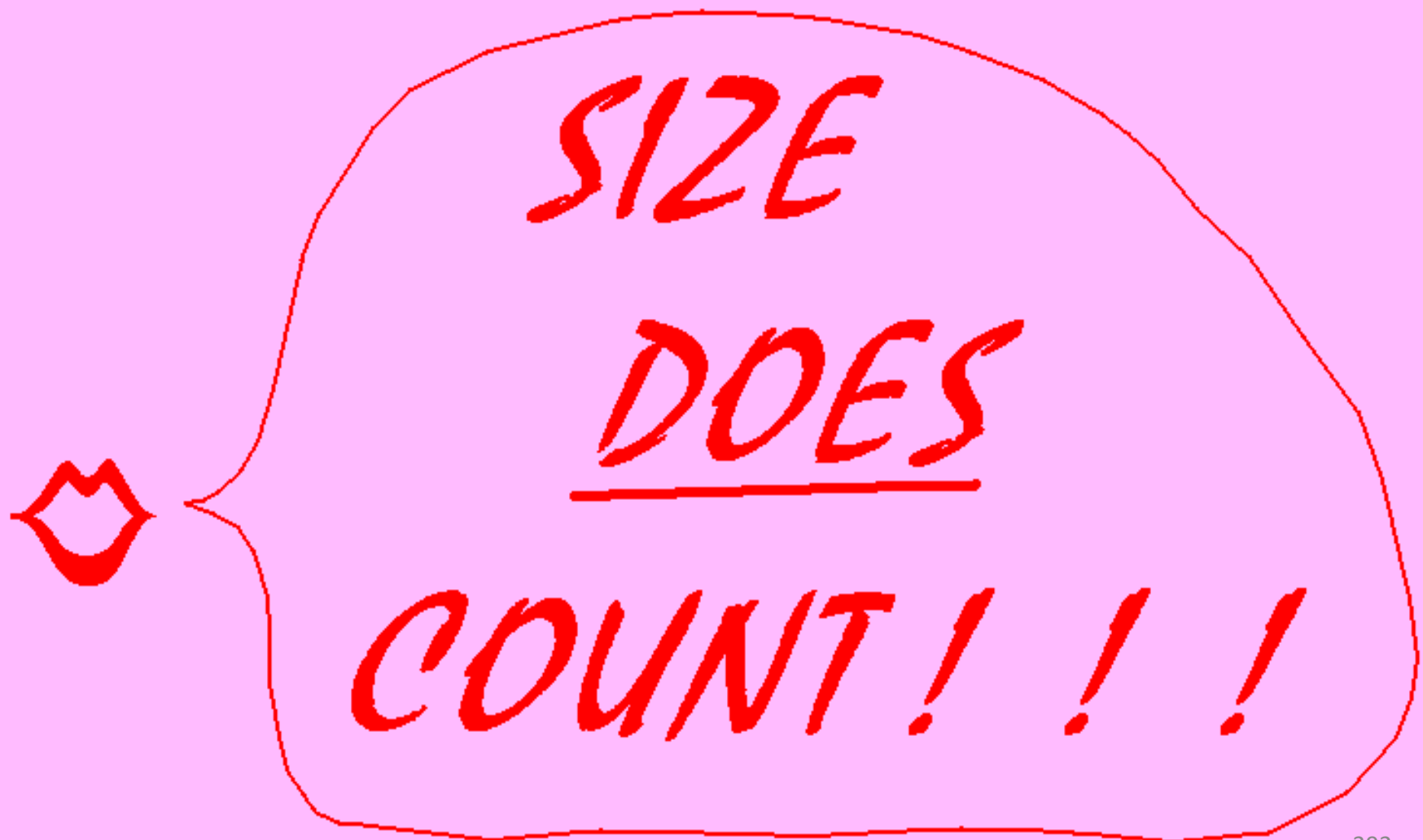
caused by depolarization of
the intraventricular septum

Abnormal Q Waves -

caused by:

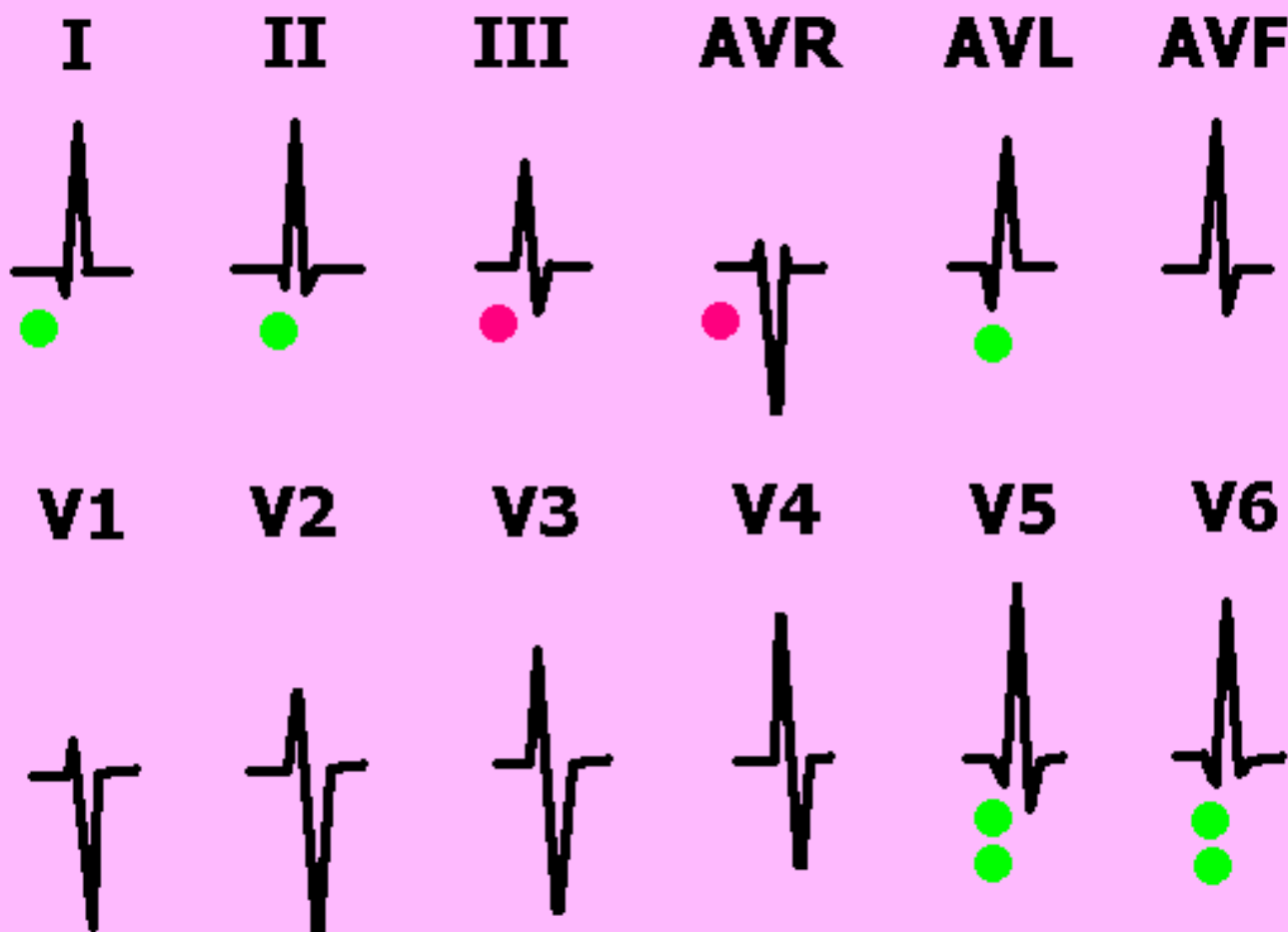
- necrosis (old infarction)
- hypertrophy

• Q WAVES •



LEADS WHERE Q WAVES ARE NORMAL

- Normal Q WAVES caused by SEPTAL DEPOLARIZATION



Q WAVES NORMAL AND FREQUENTLY SEEN



Q WAVES EXPECTED



Q WAVES, IF PRESENT, CAN NORMALLY BE ANY SIZE

THE QRS COMPLEX

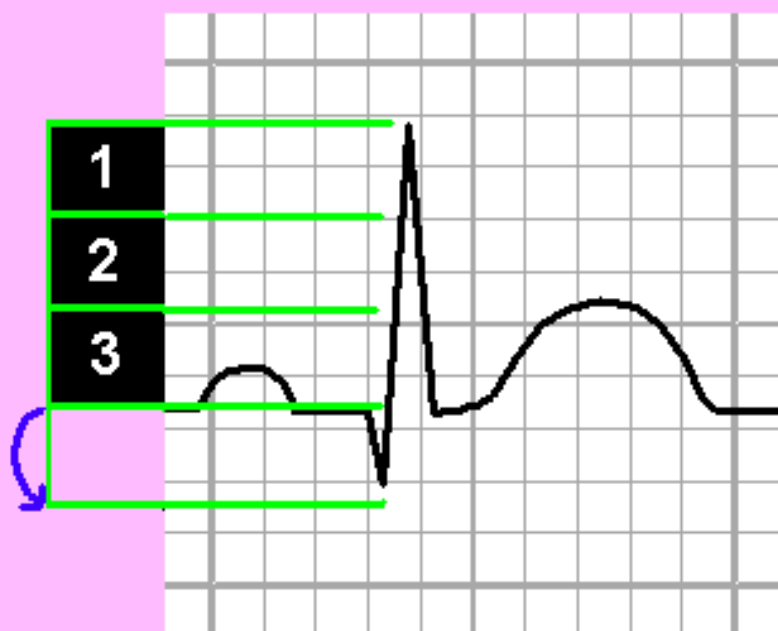
GENERAL RULES FOR NORMAL Q WAVES - WIDTH



**LESS THAN .40
(1 mm) WIDE**

THE QRS COMPLEX

GENERAL RULES FOR NORMAL Q WAVES - HEIGHT

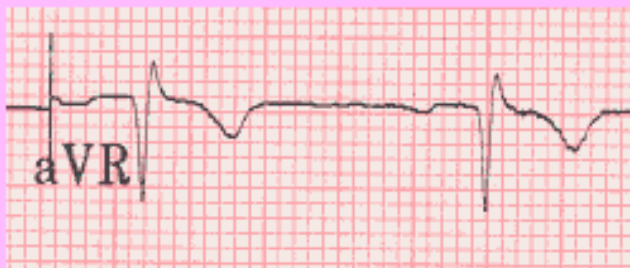


LESS THAN $\frac{1}{3}$ THE
HEIGHT OF THE R WAVE

THE QRS COMPLEX

NORMAL Q WAVES

EXCEPTIONS TO THE RULES



LEAD aVR



LEAD III



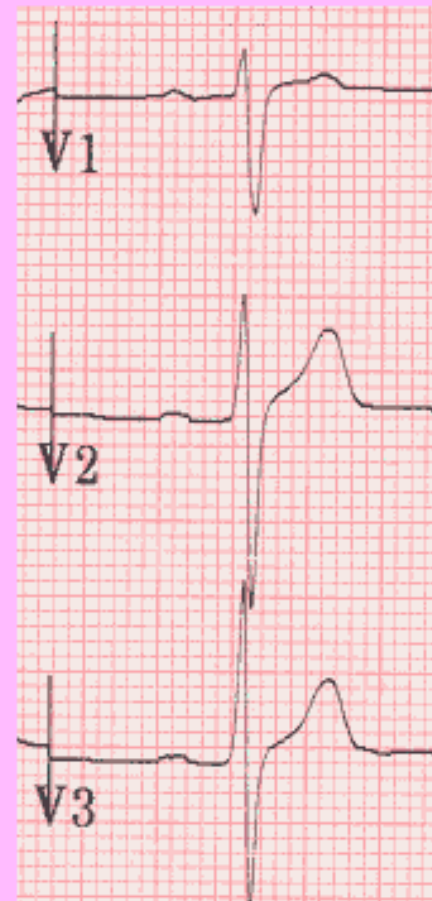
THE Q WAVE CAN BE ANY SIZE

THE QRS COMPLEX

NORMAL Q WAVES EXCEPTIONS TO THE RULES



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



THE QRS COMPLEX

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

NORMAL ST - T WAVES

- WHEN QRS WIDTH IS NORMAL (< 120 ms)

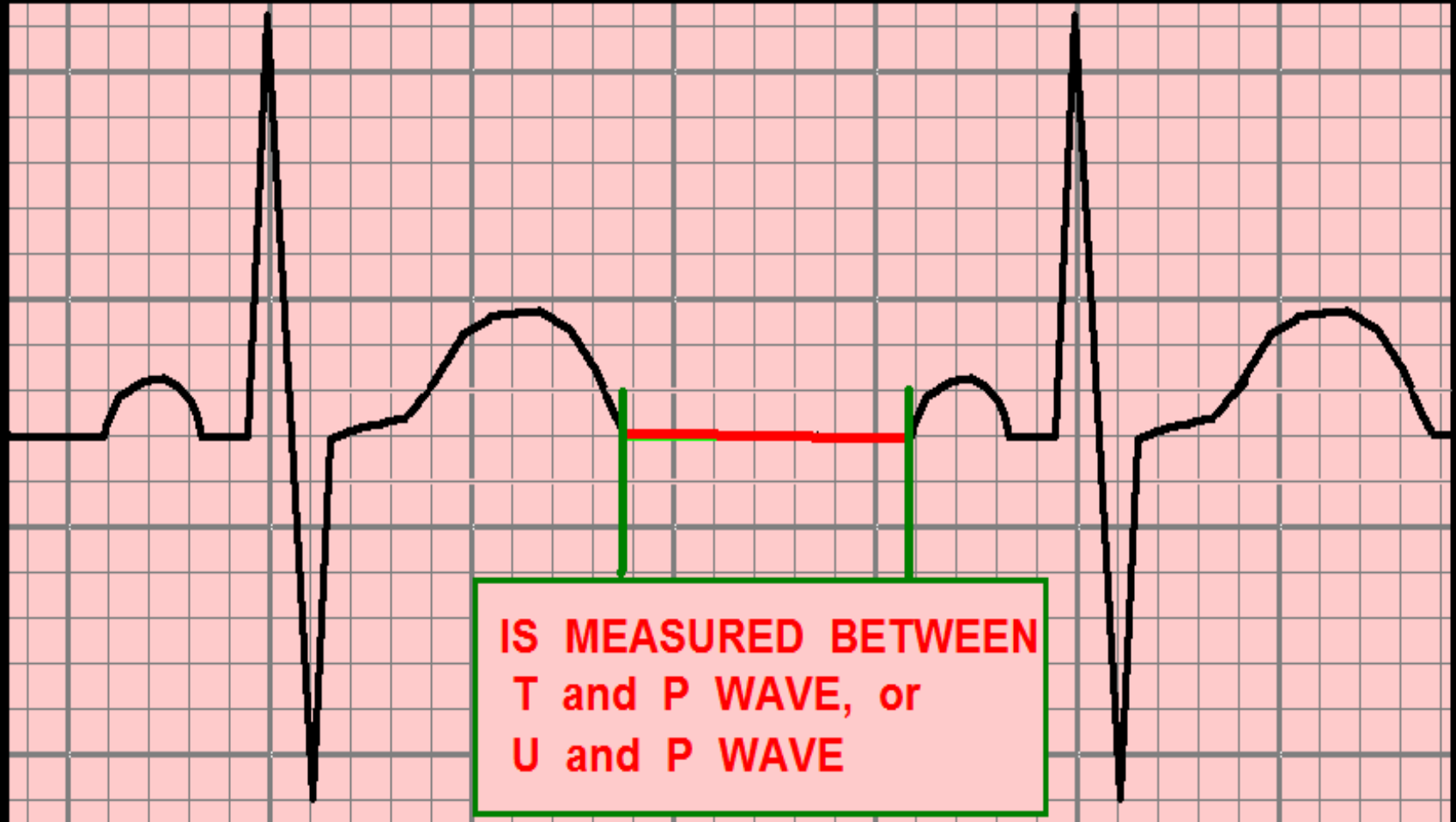
ASSESS:



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

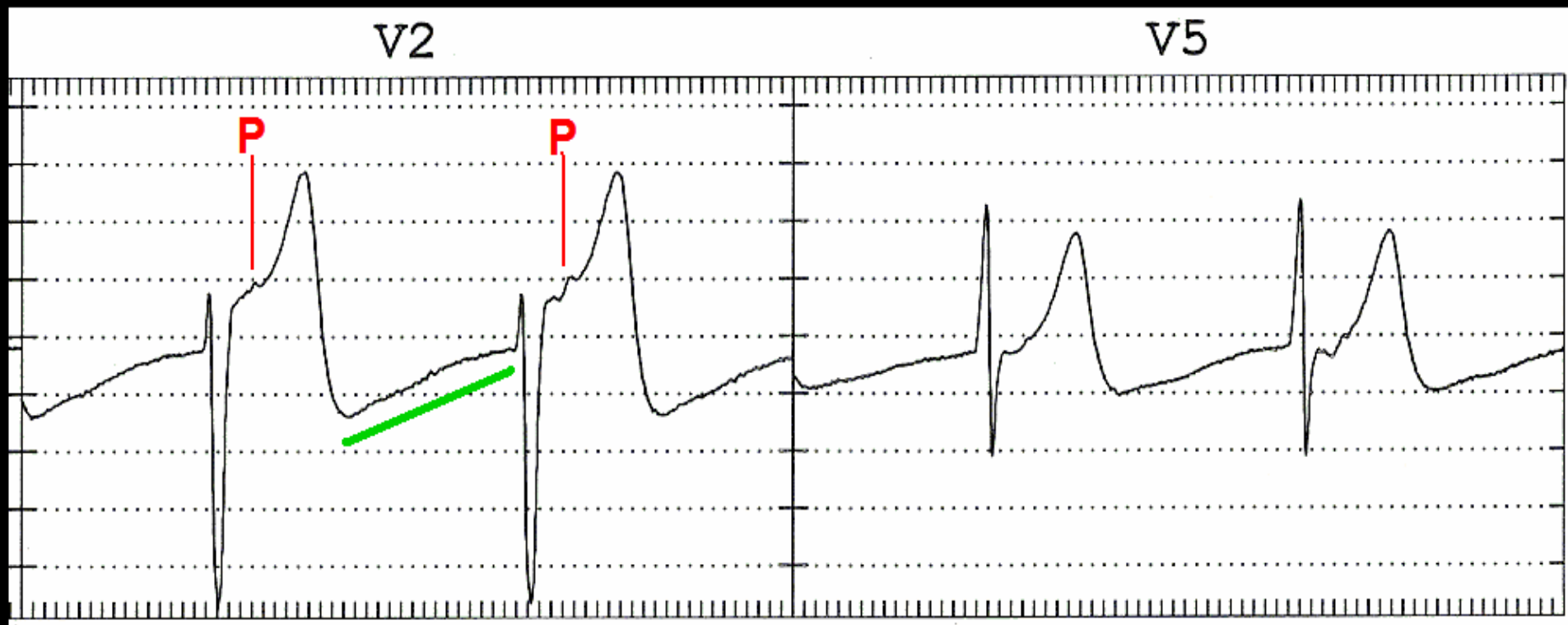
👉 **in EVERY LEAD EXCEPT aVR !!**

THE ISOELECTRIC LINE



THE ISOELECTRIC LINE

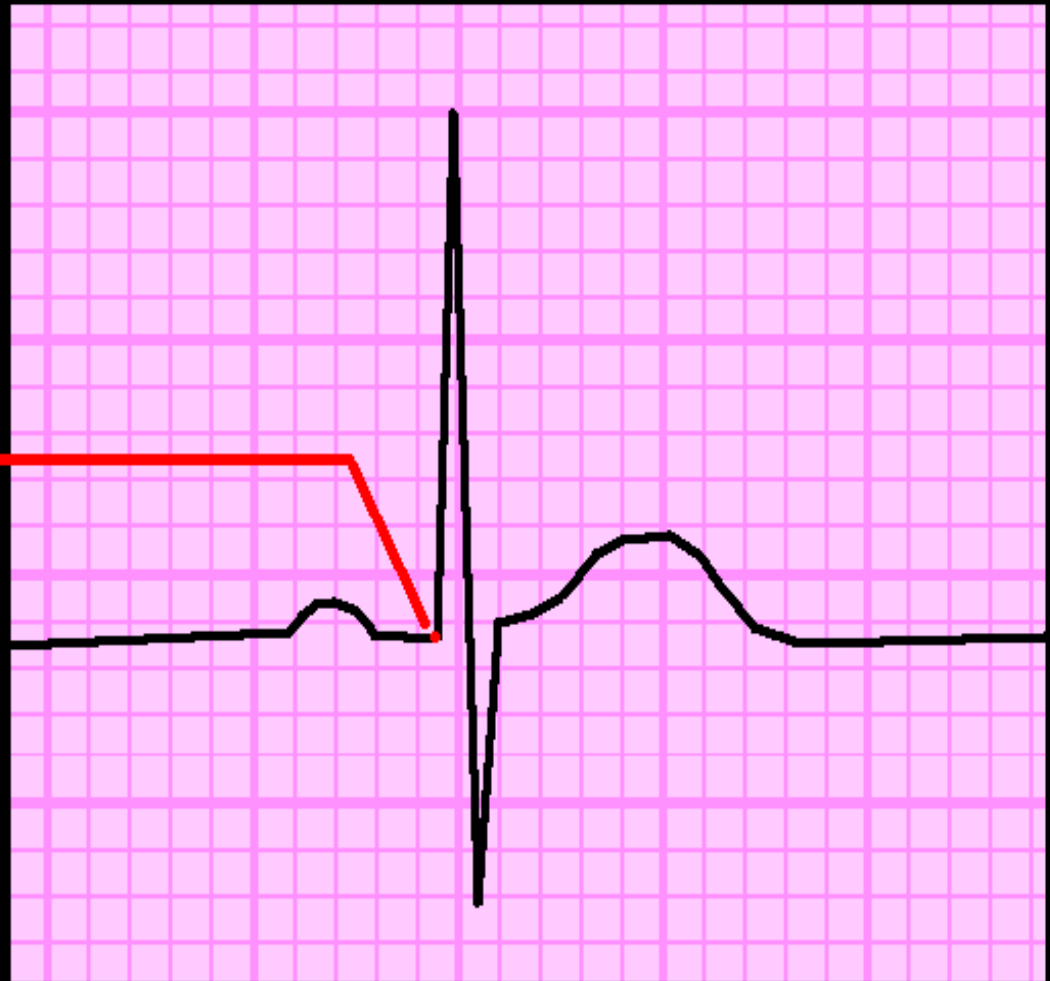
EKG from 13 y/o girl in ACCELERATED JUNCTIONAL RHYTHM.
note: upsloping T-P interval, and P buried in T waves.



THE P-Q JUNCTION

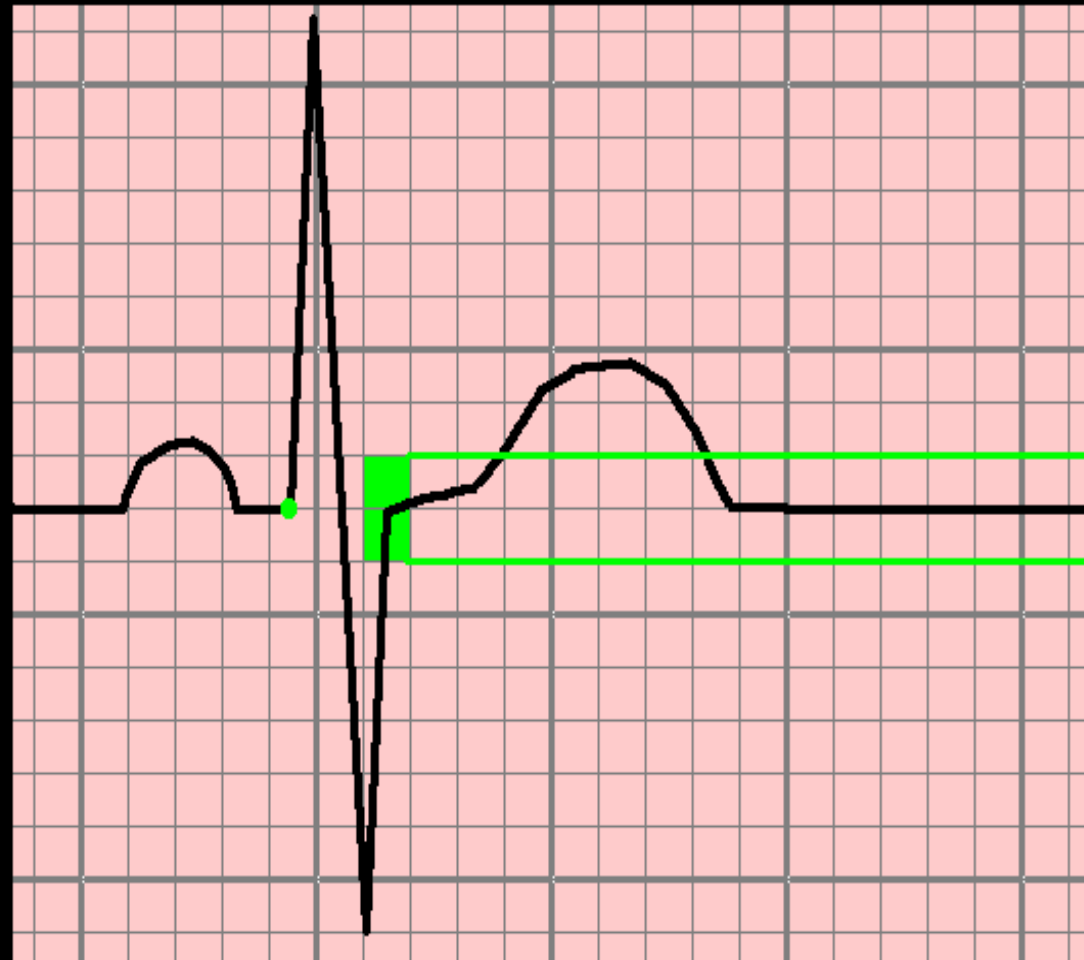
. . . is the POINT
where the P-R
SEGMENT ends
and the QRS
COMPLEX BEGINS.

Used for POINT
OF REFERENCE
for measurement of
the J-POINT and
the S-T SEGMENT –



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

THE J POINT SHOULD BE ..

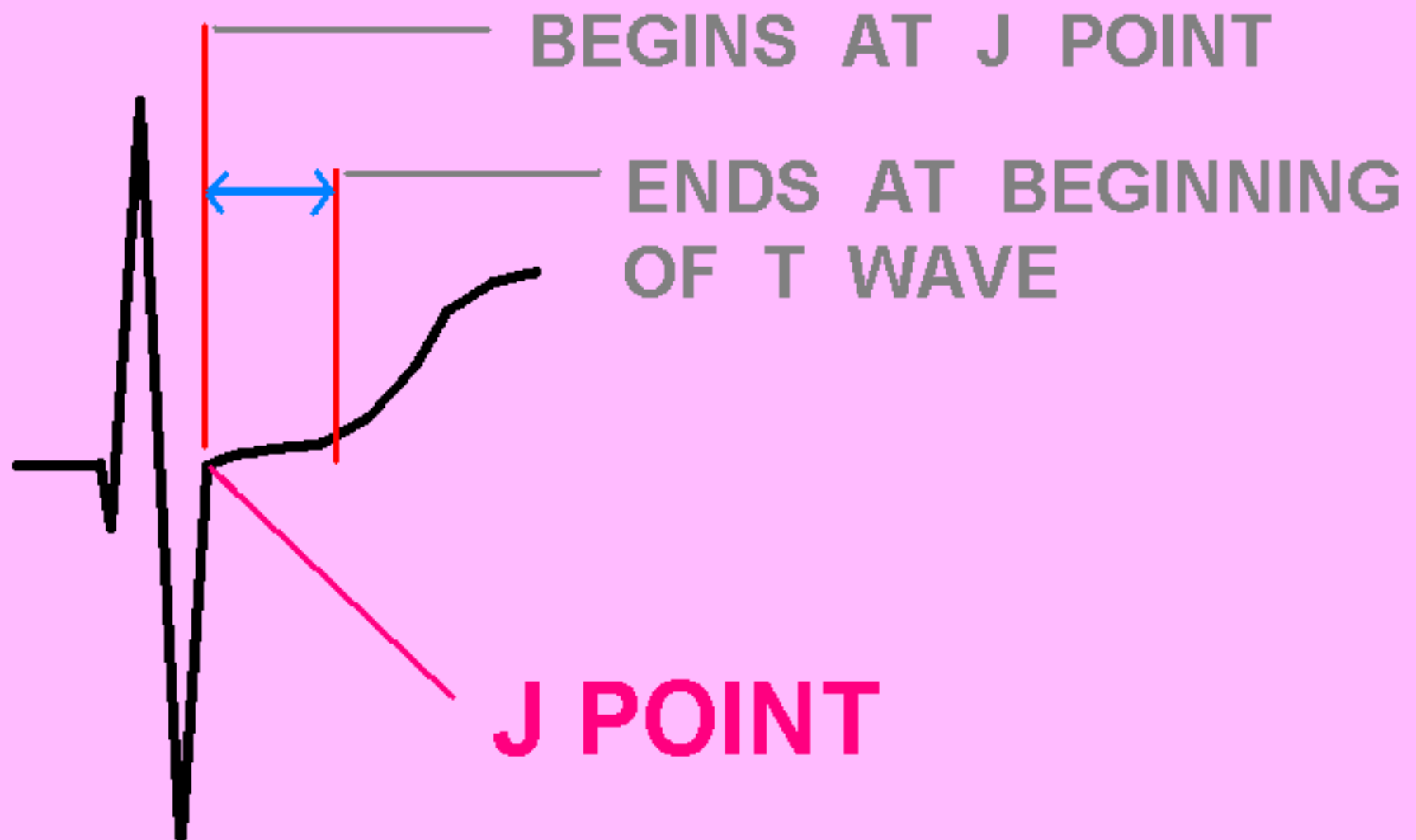


**WITHIN
1 mm
ABOVE**

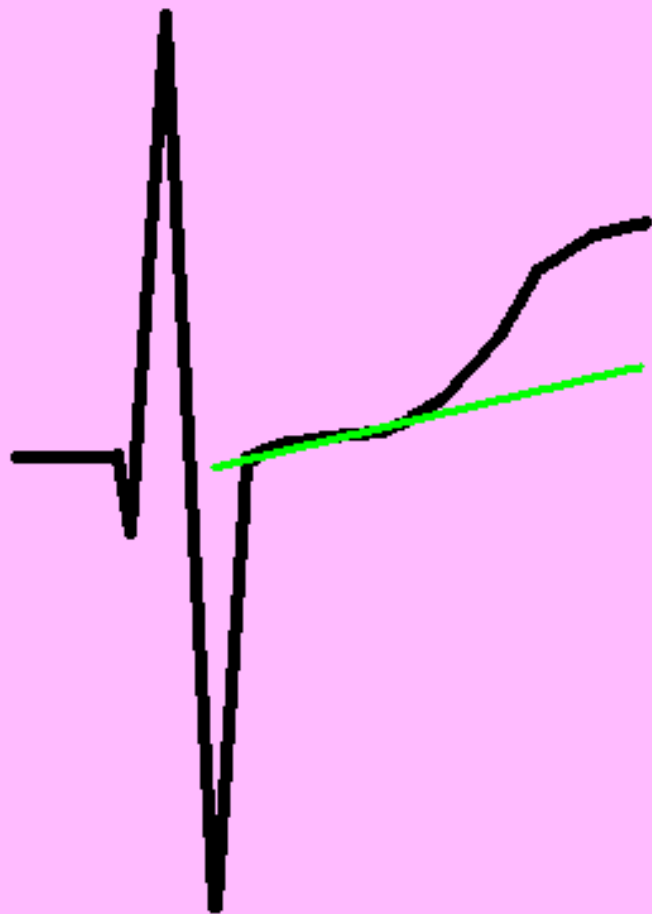
OR

**BELOW
THE
P-Q
JUNCTION**

THE S-T SEGMENT

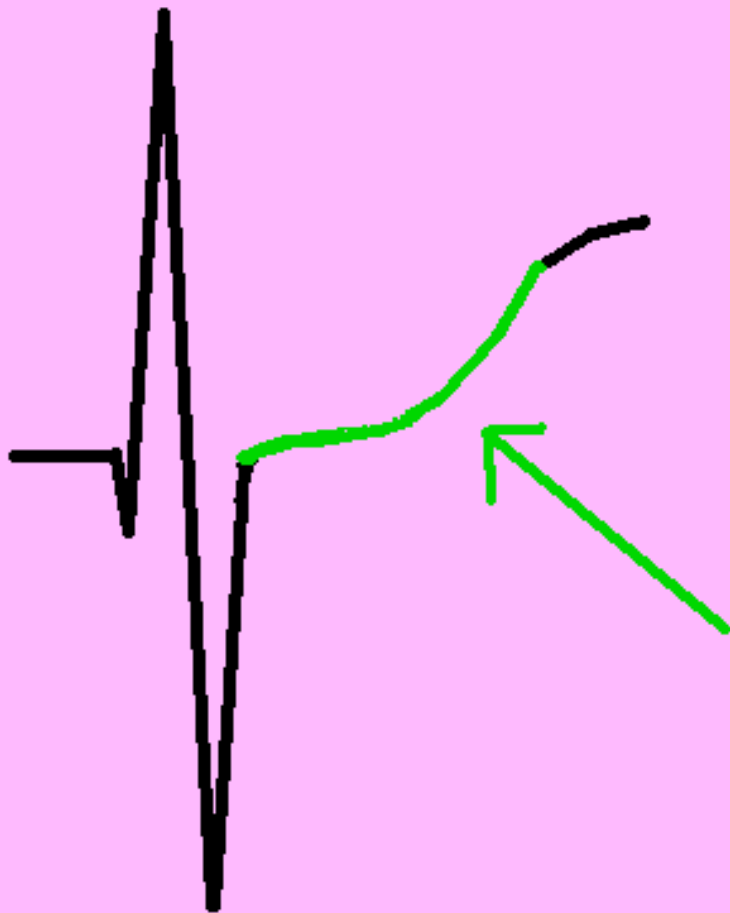


THE S-T SEGMENT



**SHOULD HAVE
A "SLIGHT POSITIVE"
INCLINATION**

THE S-T SEGMENT

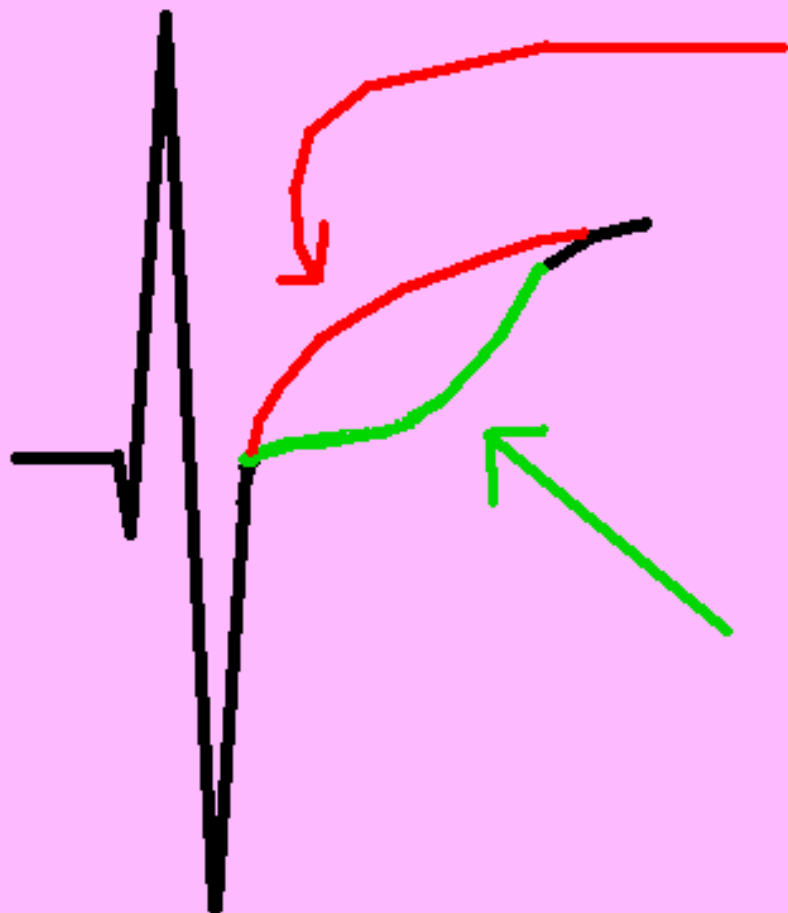


SHOULD BE
"CONCAVE" IN
SHAPE . . .

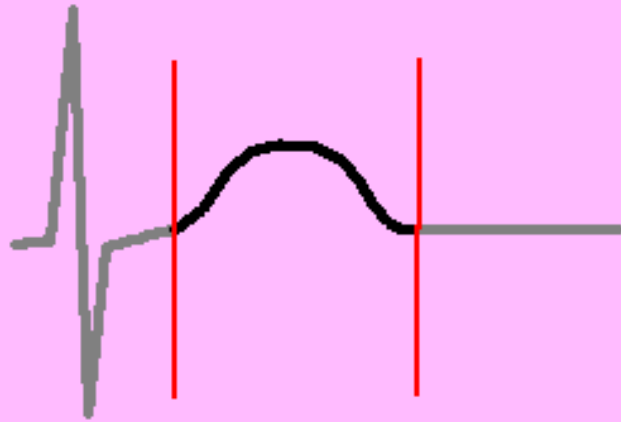
THE S-T SEGMENT

AS OPPOSED TO
"CONVEX" IN
SHAPE

SHOULD BE
"CONCAVE" IN
SHAPE . . .

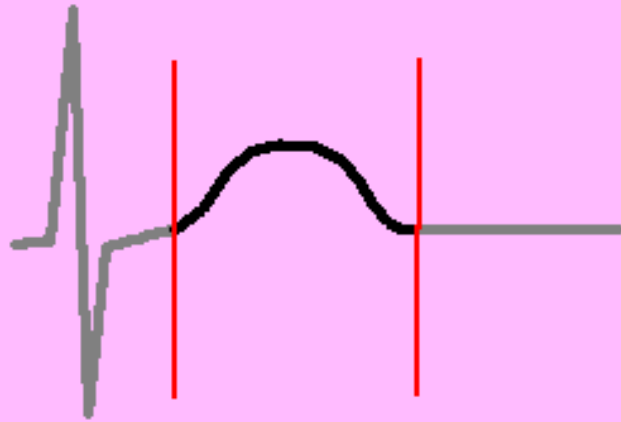


THE T WAVE



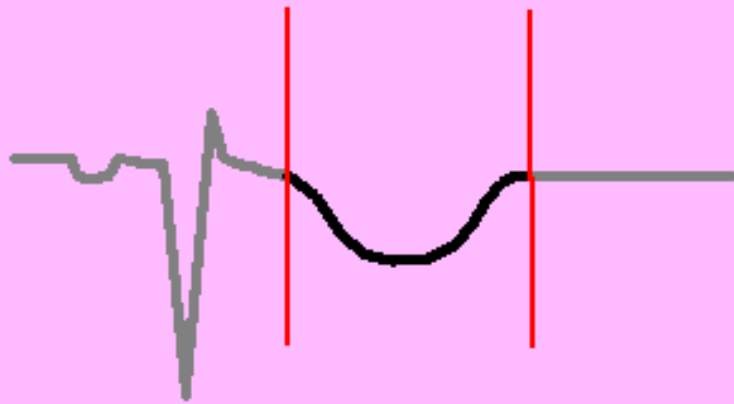
- SHOULD BE A "NICE," ROUNDED, CONVEX SHAPE
- SHOULD BE SYMMETRICAL

THE T WAVE



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

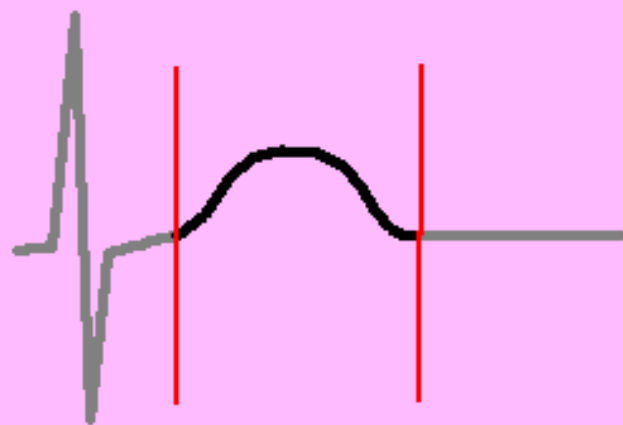
THE T WAVE



**LEAD
AVR**

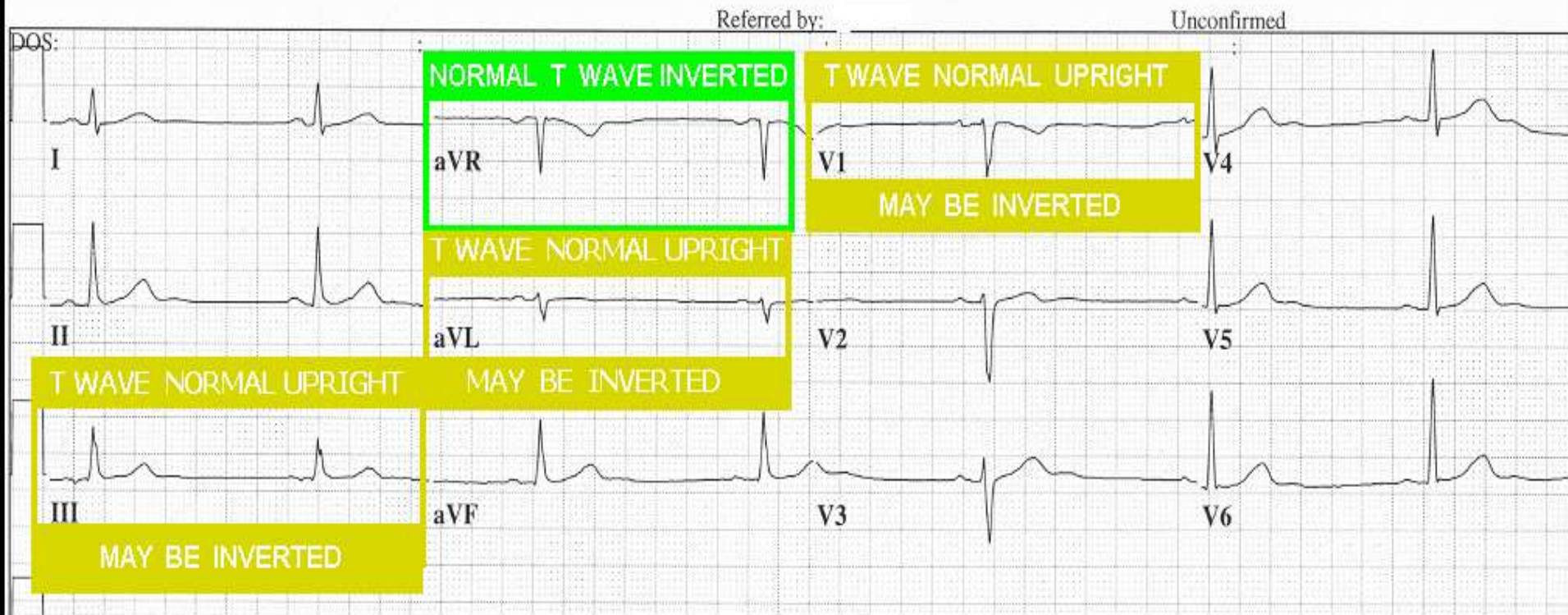
- **REMEMBER, IN LEAD AVR
EVERYTHING
IS
"UPSIDE-DOWN"**

THE T WAVE



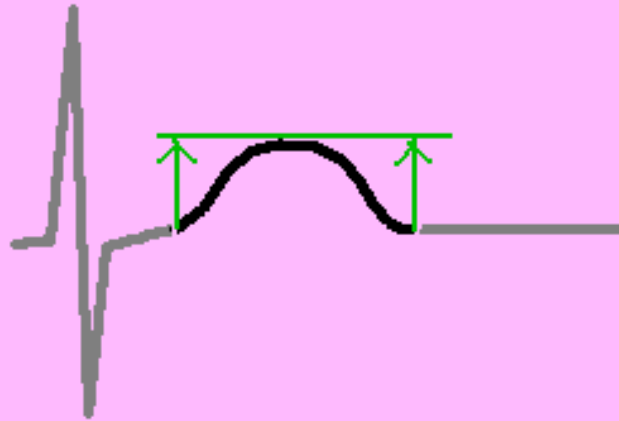
- SHOULD BE A "NICE," ROUNDED, CONVEX SHAPE
- SHOULD BE SYMMETRICAL
- SHOULD BE UPRIGHT IN ALL LEADS, EXCEPT AVR
- MAY BE INVERTED IN LEADS I, III, and V1

Leads where the T WAVE may be INVERTED:



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

THE T WAVE



AMPLITUDE GUIDELINES:

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

HYPER-ACUTE T WAVES - COMMON ETIOLOGIES:



CONDITION:

 **HYPERKALEMIA**

 **ACUTE MI**

 **TRANSMURAL
ISCHEMIA**

 **HYPERTROPHY**

MORE INFORMATION ON HYPERACUTE T WAVES COMING UP SOON . . .

S-T SEGMENT ELEVATION - COMMON ETIOLOGIES:

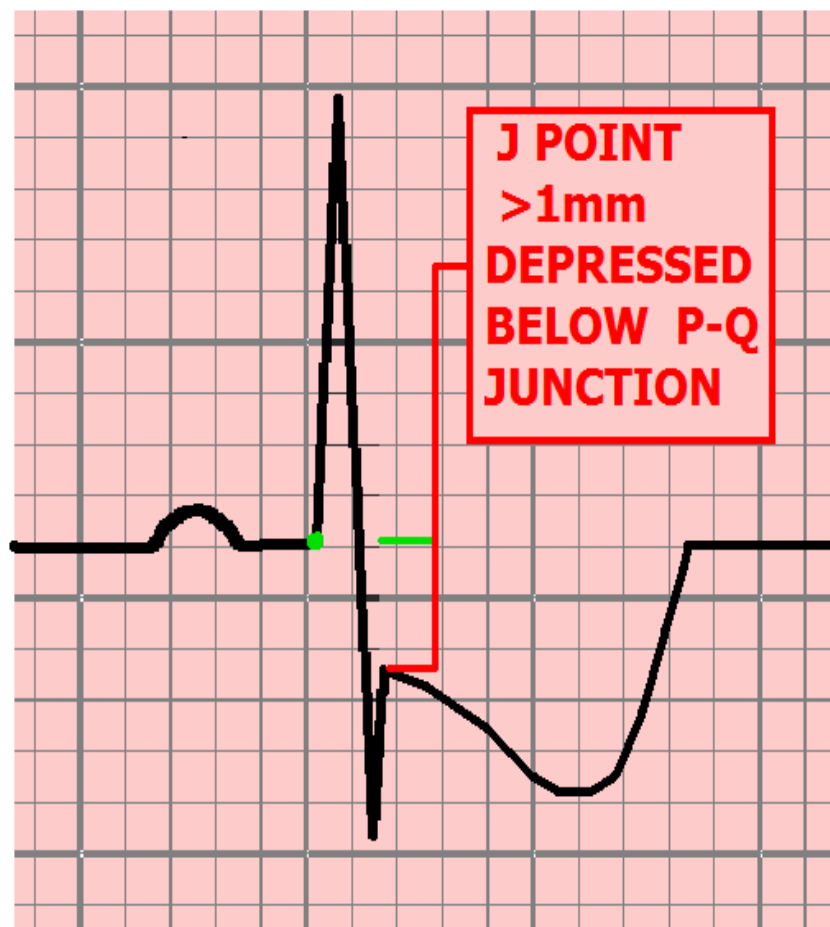


CONDITION:

- **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.**

ON THE NEXT PAGE IN YOUR BOOK ARE SOME EXAMPLES OF THE ABOVE CONDITIONS

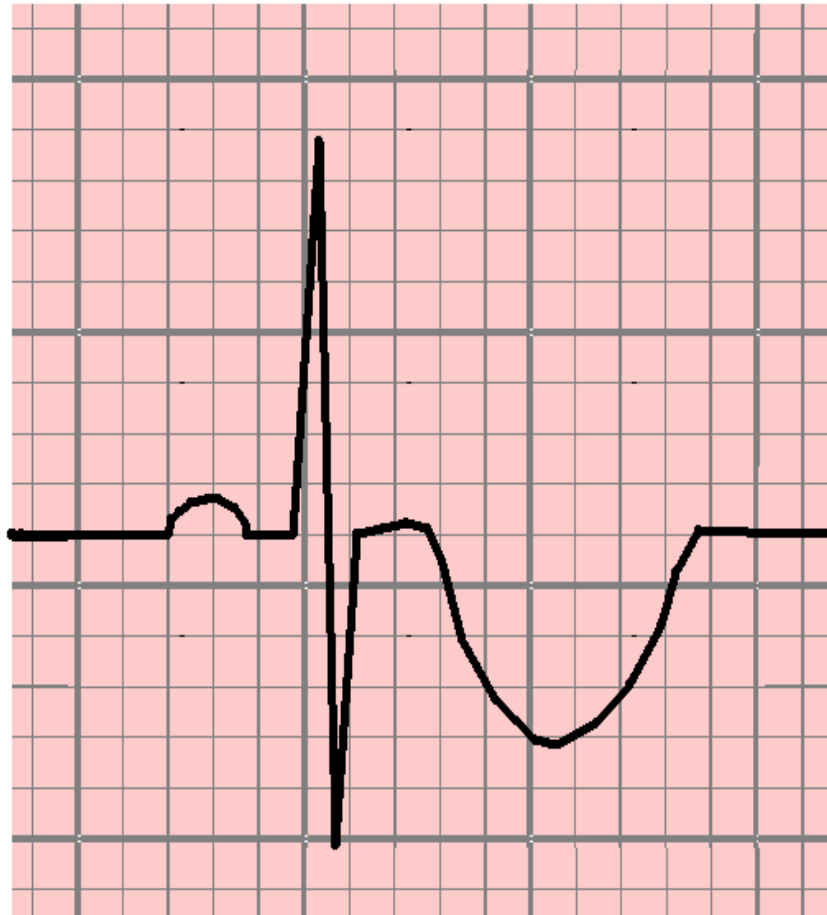
S-T SEGMENT DEPRESSION - COMMON ETIOLOGIES:



CONDITION:

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

T WAVE INVERSION - COMMON ETIOLOGIES:



CONDITION:

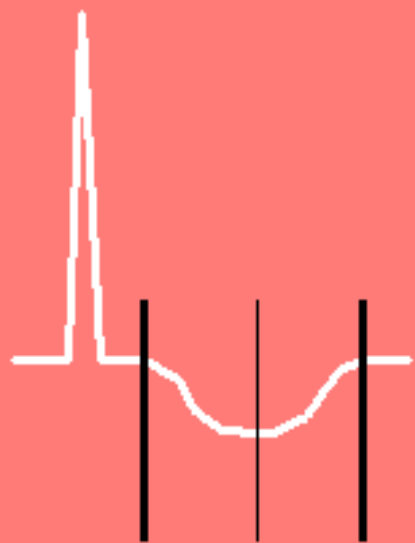
- **MYOCARDITIS**
- **ELECTROLYTE IMBALANCE**
- **ISCHEMIA**
- **POSITIVE STRESS TEST**
- **CEREBRAL DISORDER**
- **MITRAL VALVE PROLAPSE**
- **VENTRICULAR HYPERTROPHY**
- **WOLFF-PARKINSON-WHITE**
- **HYPERVENTILATION**
- **CARDIOACTIVE DRUGS**
- **OLD MI (NECROSIS vs. ISCHEMIA)**
- **DIGITALIS**
-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

CHAMBER ENLARGEMENT

VENTRICULAR STRAIN PATTERNS



T-WAVES ARE INVERTED
and ASYMMETRICAL



symmetrical



asymmetrical

CHAMBER ENLARGEMENT

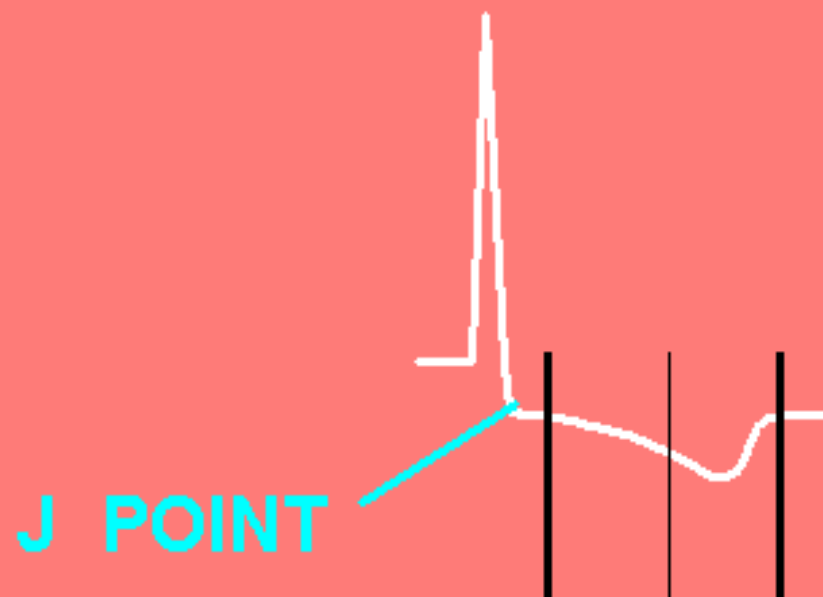
VENTRICULAR STRAIN PATTERNS



**T WAVES ARE INVERTED
AND ASYMMETRICAL**



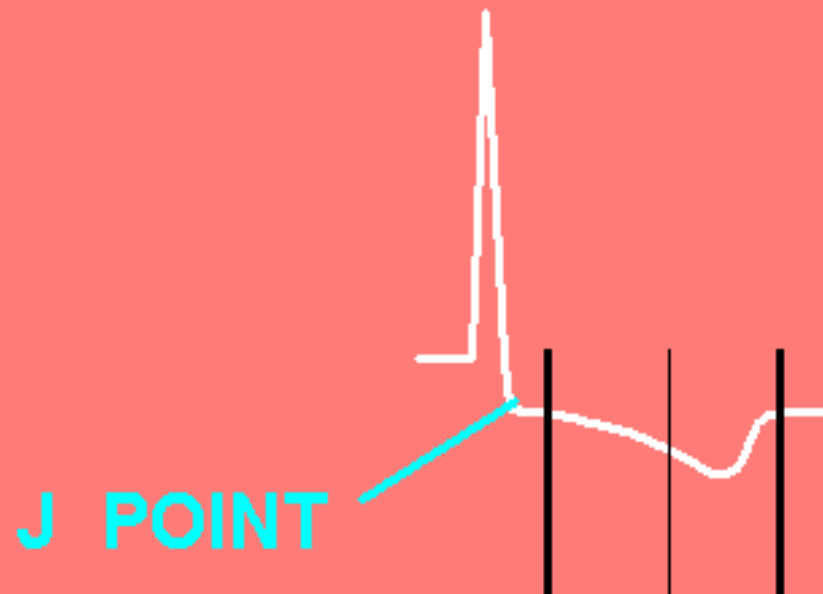
**THERE MAY BE S-T SEGMENT
DEPRESSION**



CHAMBER ENLARGEMENT

VENTRICULAR STRAIN PATTERNS

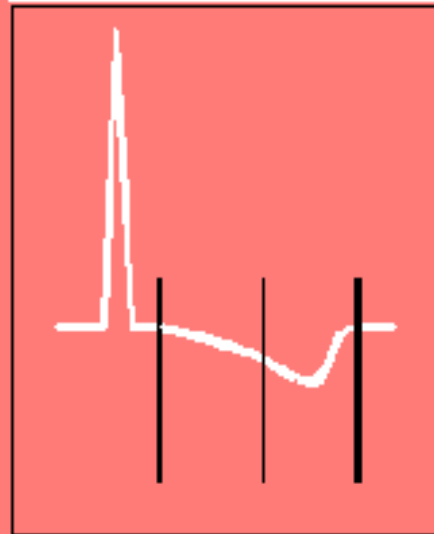
STRAIN PATTERNS ARE INDICATIVE OF
SYSTOLIC OVERLOAD -- THE
VENTRICLES HAVING TO OVERCOME
GREAT FORCE TO EXPEL BLOOD.



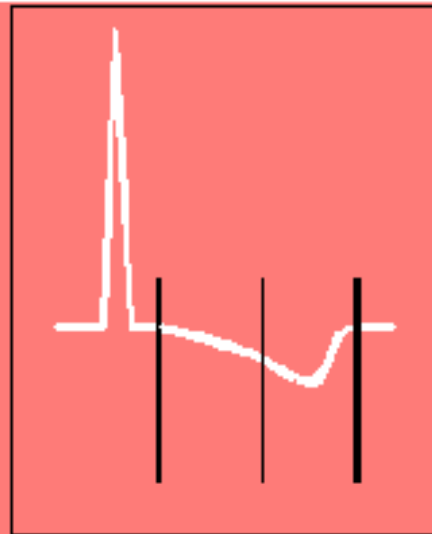
CHAMBER ENLARGEMENT

VENTRICULAR STRAIN PATTERNS

RVH

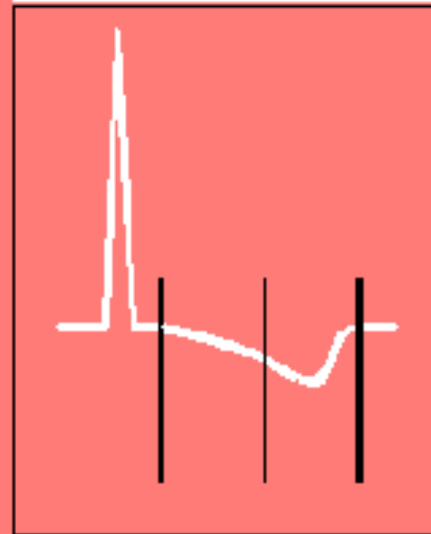


V1

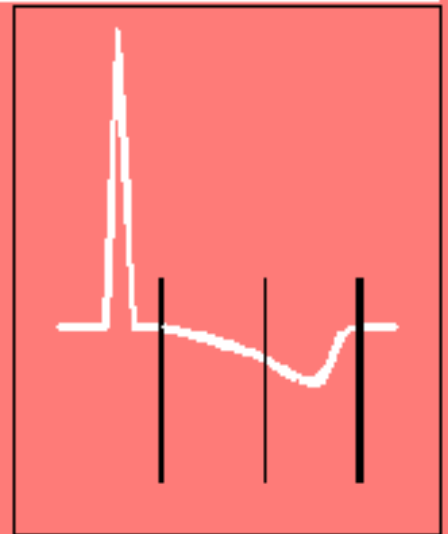


V2

LVH



V5



V6

hang in there !

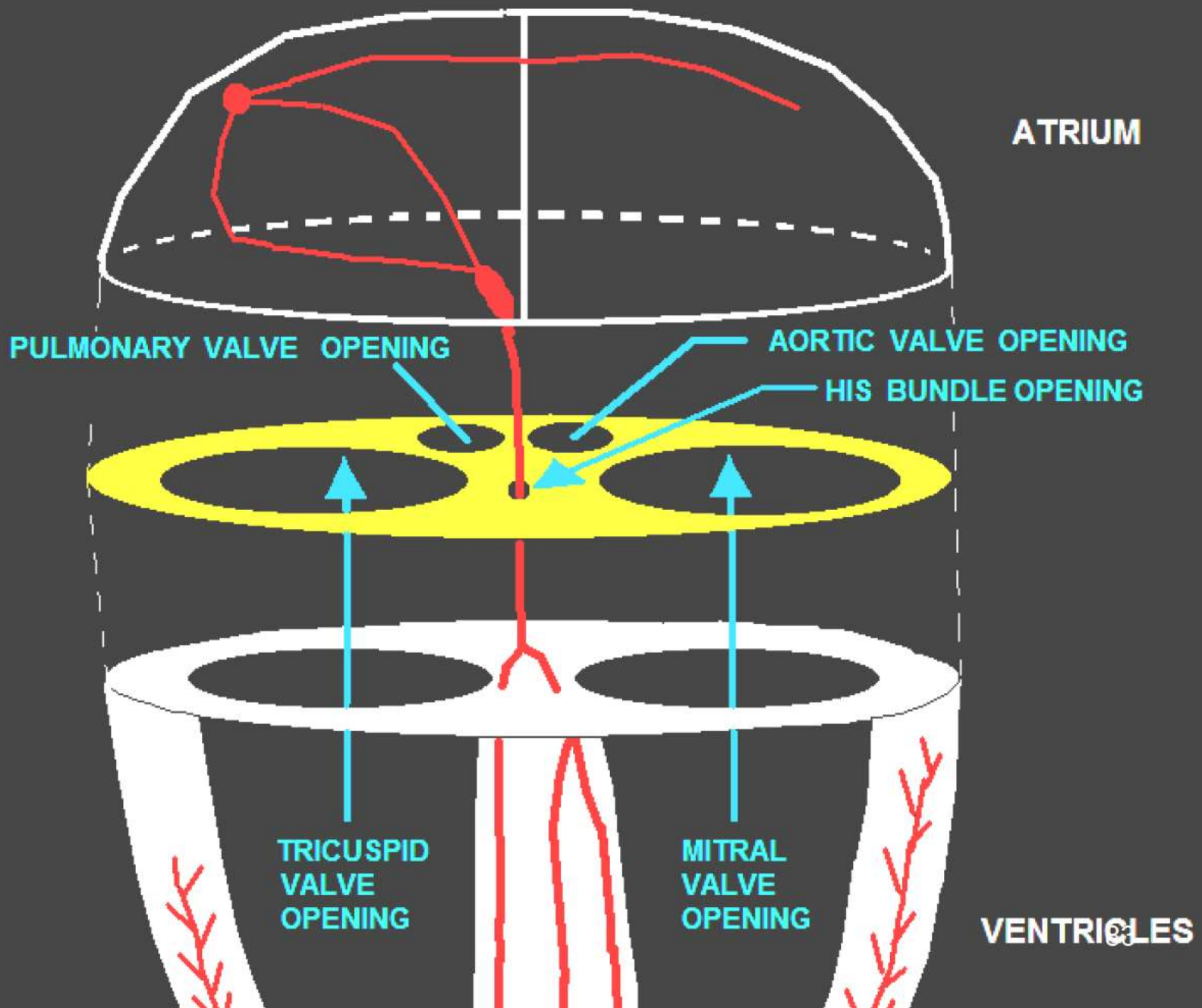
it's almost LUNCH TIME !!!!



Wayne "Will" Ruppert, III

THE "SKELETON OF THE HEART"

**FIBROUS
"SKELETON
of the
HEART"**



Fibrous Skeleton of the Heart...

- Rarely taught But it's so important to understanding cardiac function and ECGs.....
- It's a disk-shaped structure separating the atrium from the ventricles.
- Secures the heart valves.
- Acts as an electrical insulator, blocks electrical current.....
- **An abnormal hole (BYPASS TRACT) allows current to “leak” between atrium and ventricles**

WOLFF-PARKINSON-WHITE

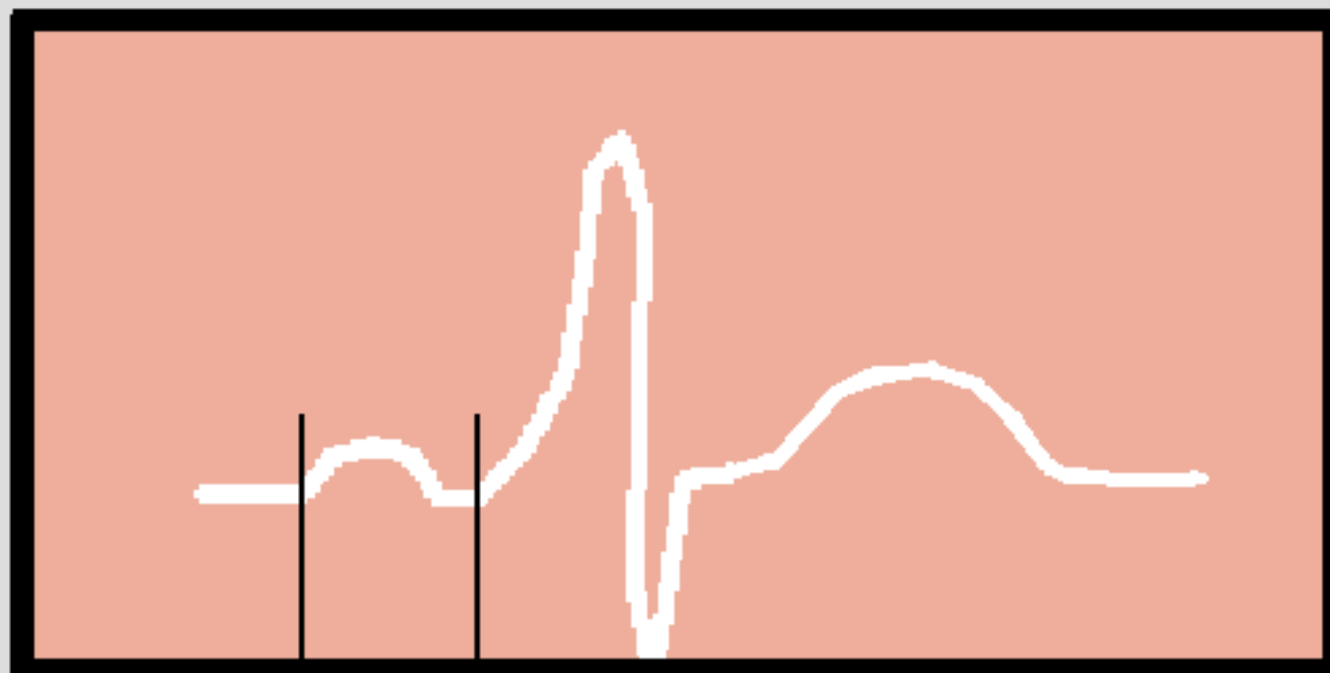
THE NORMAL ECG



**NORMAL
P-R INTERVAL**

WOLFF-PARKINSON-WHITE

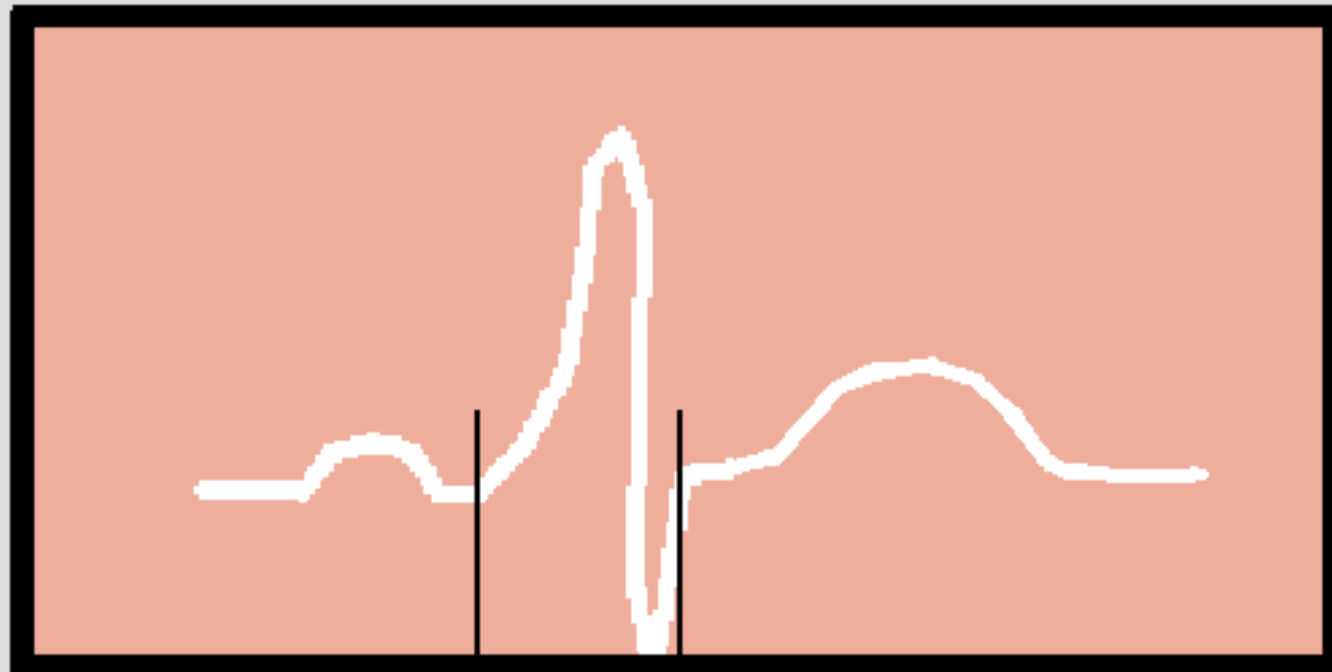
EKG CHARACTERISTICS



SHORTENED
P-R INTERVAL

WOLFF-PARKINSON-WHITE

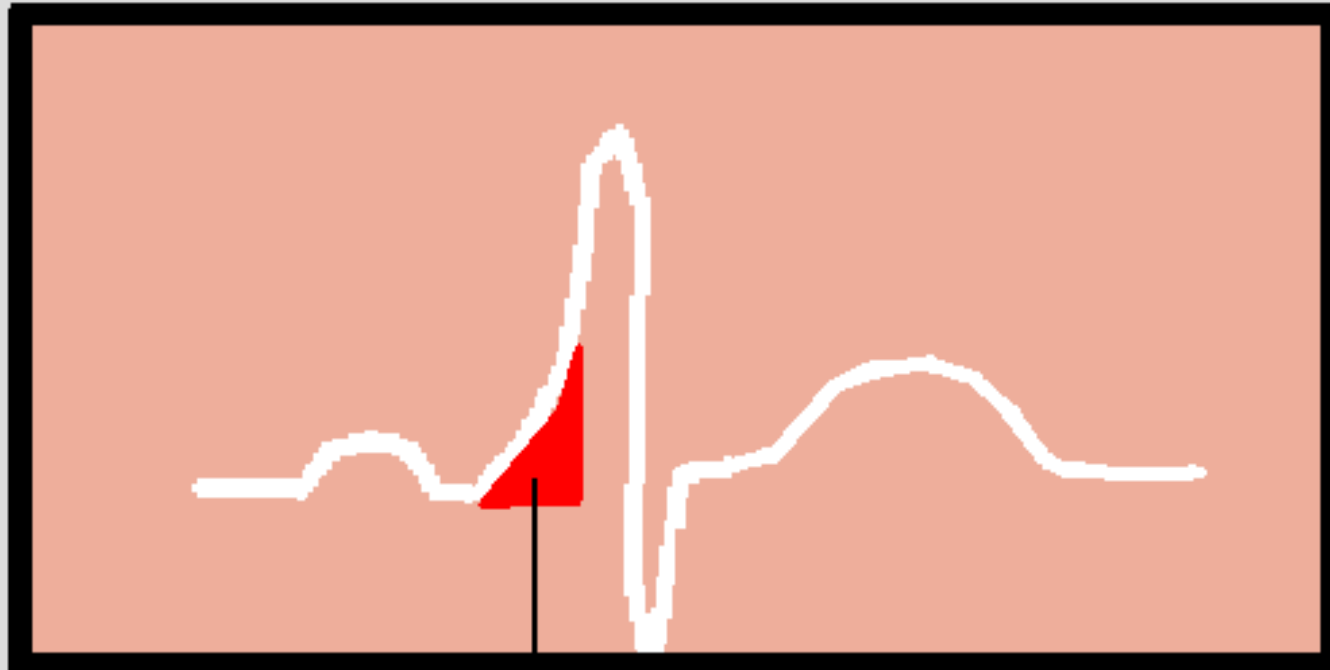
EKG CHARACTERISTICS



WIDENED
QRS COMPLEX

WOLFF-PARKINSON-WHITE

EKG CHARACTERISTICS



DELTA
WAVE

01-MAY-1999 04:14:17

ST. JOSEPH'S HOSPITAL-IN1464 ROUTINE RETRIEVAL

51 yr
Male Caucasian
Room:540
Loc:5 Option:28

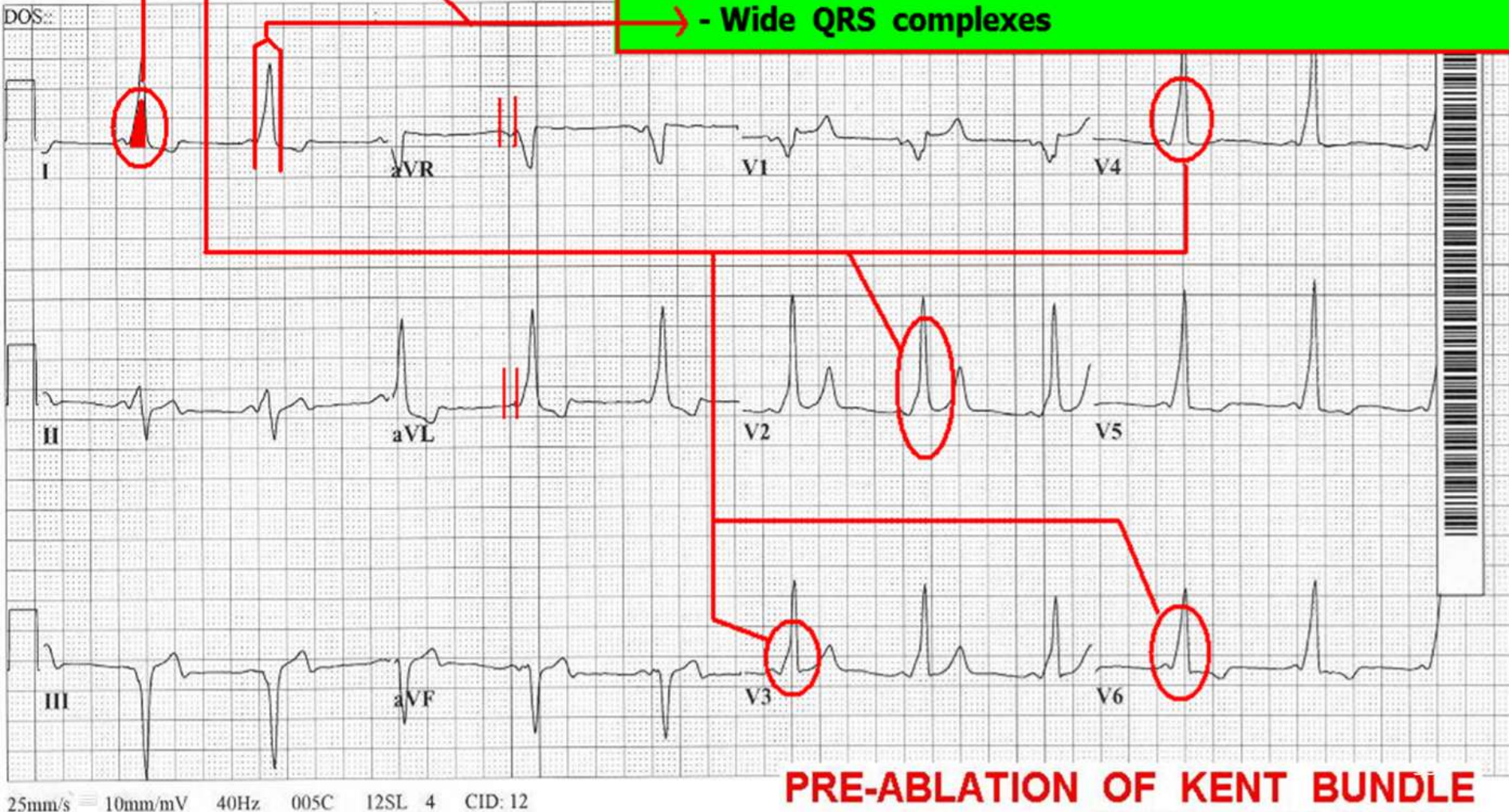
Vent rate	64	BPM
PR interval	110	ms
QRS duration	146	ms
QT/QTc	418/431	ms
P-R-T axes	50 -36 119	

Normal sinus rhythm
Wolff-Parkinson-White
Abnormal ECG
No previous ECGs available

Technician EKG CLASS #WR03696205

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

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



16 yr
Female Caucasian
Room: REC
Loc: 20 Option: 50

Vent. rate 92 BPM
PR interval 112 ms
QRS duration 118 ms
QT/QTc 356/440 ms
P-R-T axes 59 -22 107

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

Normal sinus rhythm with sinus arrhythmia

Left atrial enlargement

Anterior infarct, age undetermined

Inferior infarct, age undetermined

ST & T wave abnormality, consider lateral ischemia

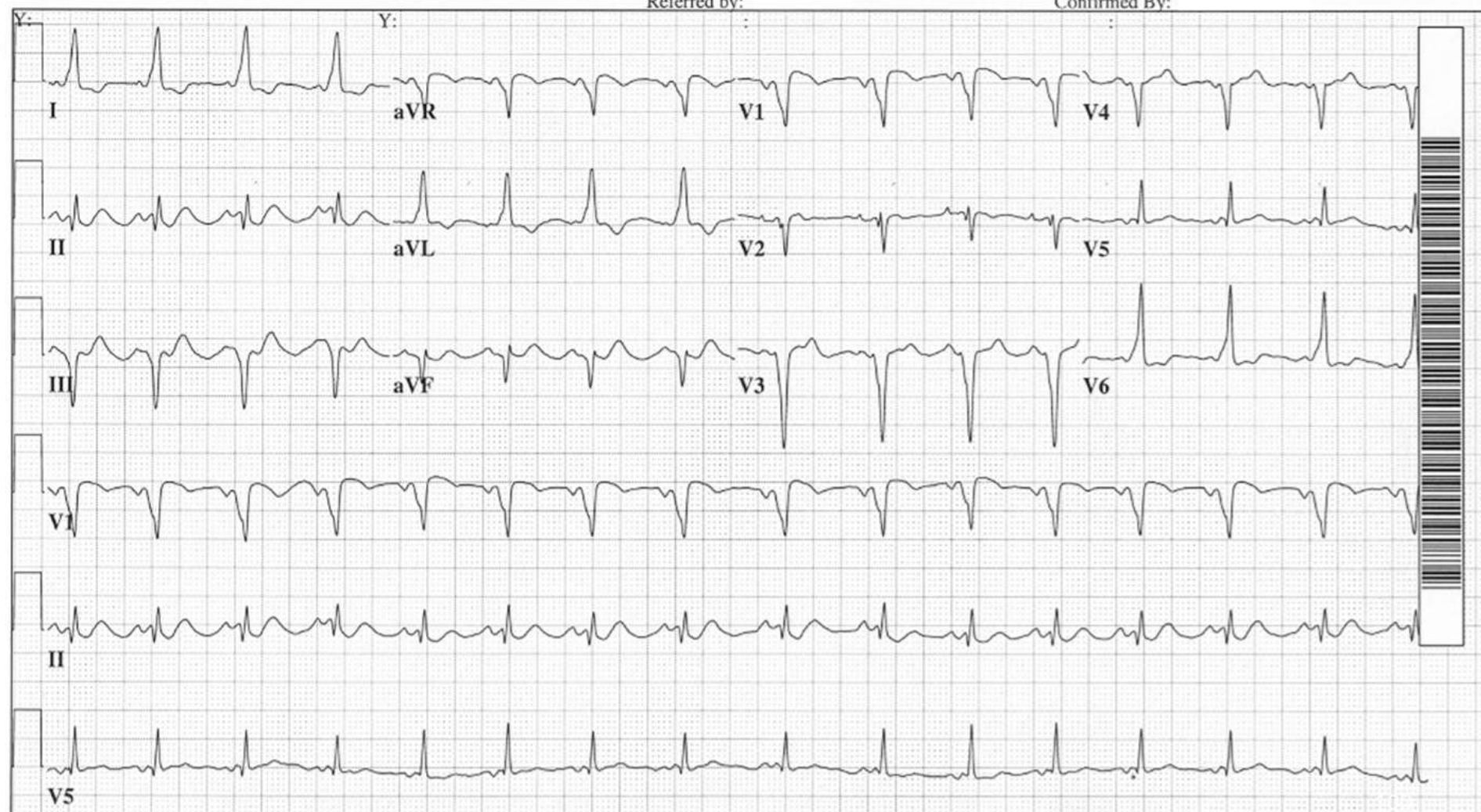
Wolff-Parkinson-White

Abnormal ECG

No previous ECGs available

Referred by:

Confirmed By:



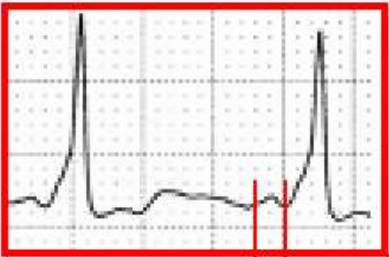
16 yr
Female Caucasian
Room: REC
Loc: 20 Option: 50

Vent. rate 92 BPM
PR interval 112 ms
QRS duration 118 ms
QT/QTc 356/440 ms
P-R-T axes 59 -22 107

Normal sinus rhythm with sinus arrhythmia
Wolff-Parkinson-White
Abnormal ECG
No previous ECGs available

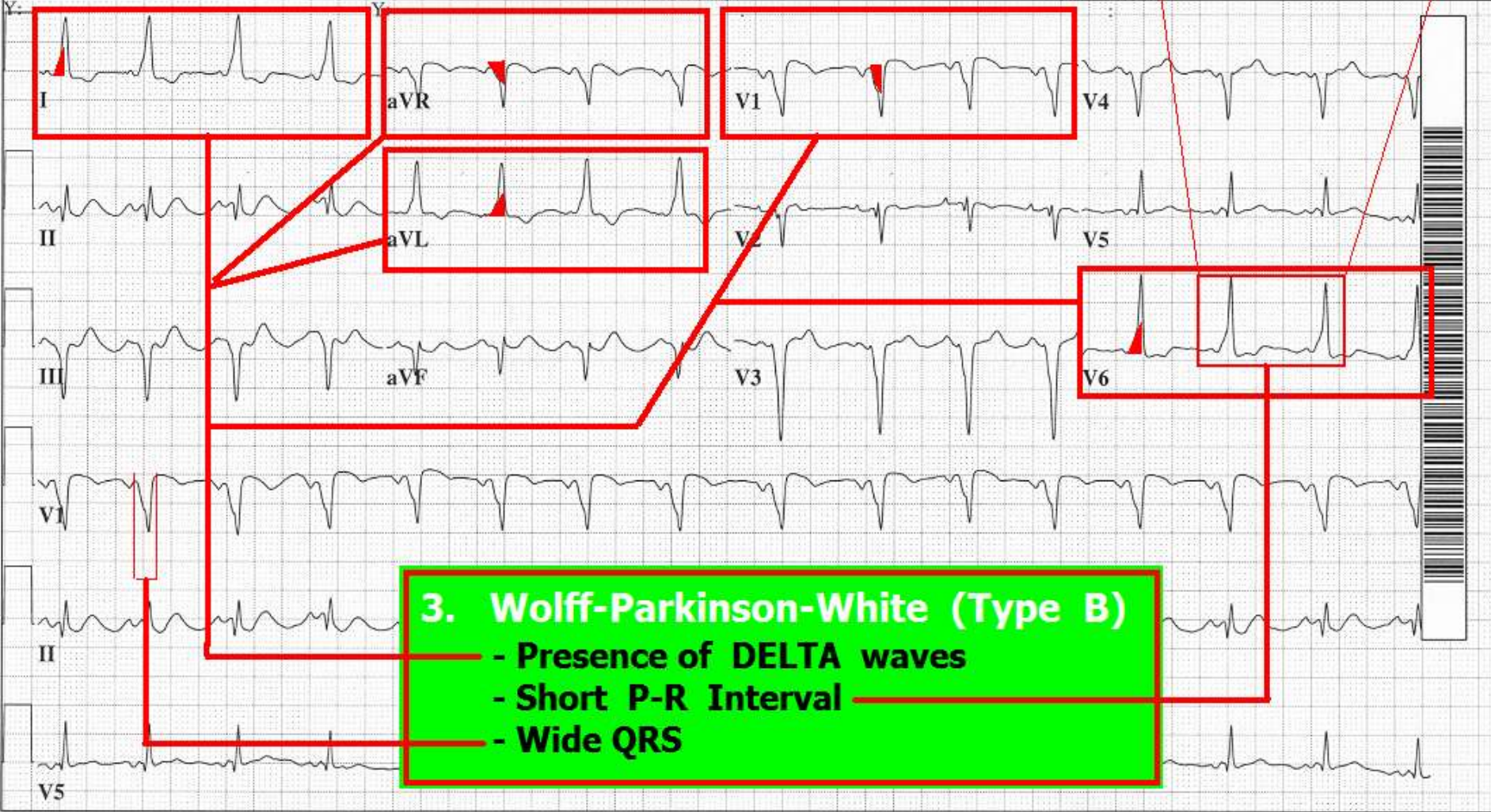
History: Unknown
Technician: DP
Test ind: EKG
EKG CLASS #WR030100
60783

P-R = .08



Referred by:

Confirmed By:

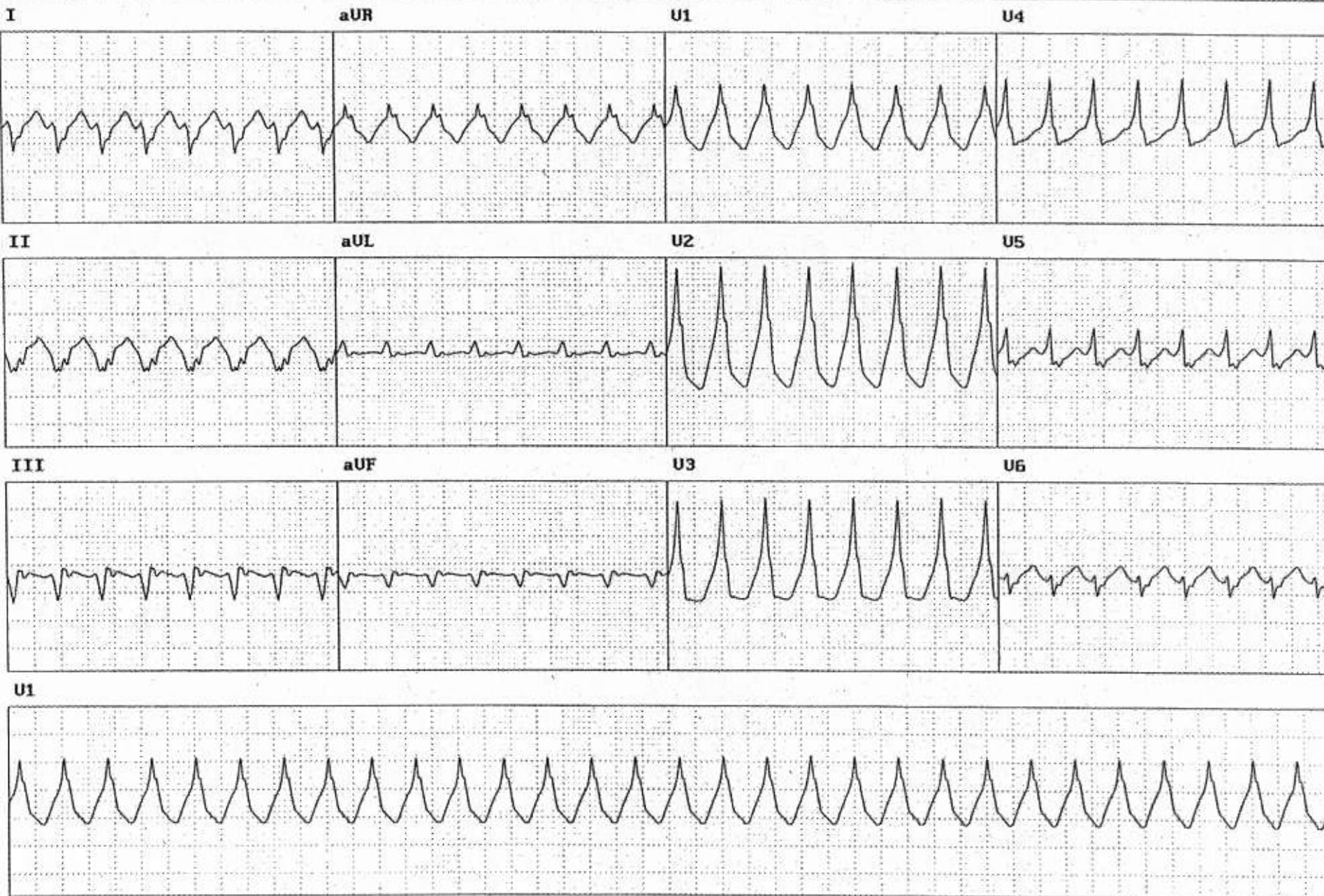


3. Wolff-Parkinson-White (Type B)
- Presence of DELTA waves
- Short P-R Interval
- Wide QRS

W-P-W patients often experience
Tachycardias:

- Narrow QRS Tachycardia (SVT)
- Wide QRS Tachycardia (mimics V-Tach.

The same patient can present with
narrow QRS SVT, and at another
time, Wide QRS Tachycardia



Patients with Wolff-Parkinson-White:

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

W-P-W may CAUSE A-fib with RVR.

Patients may present with symptoms of “palpitations,” “heart racing,” “light-headedness,” or “passing out”

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%

37 yr
Male Caucasian
Room:OP
Loc:8 Option:16

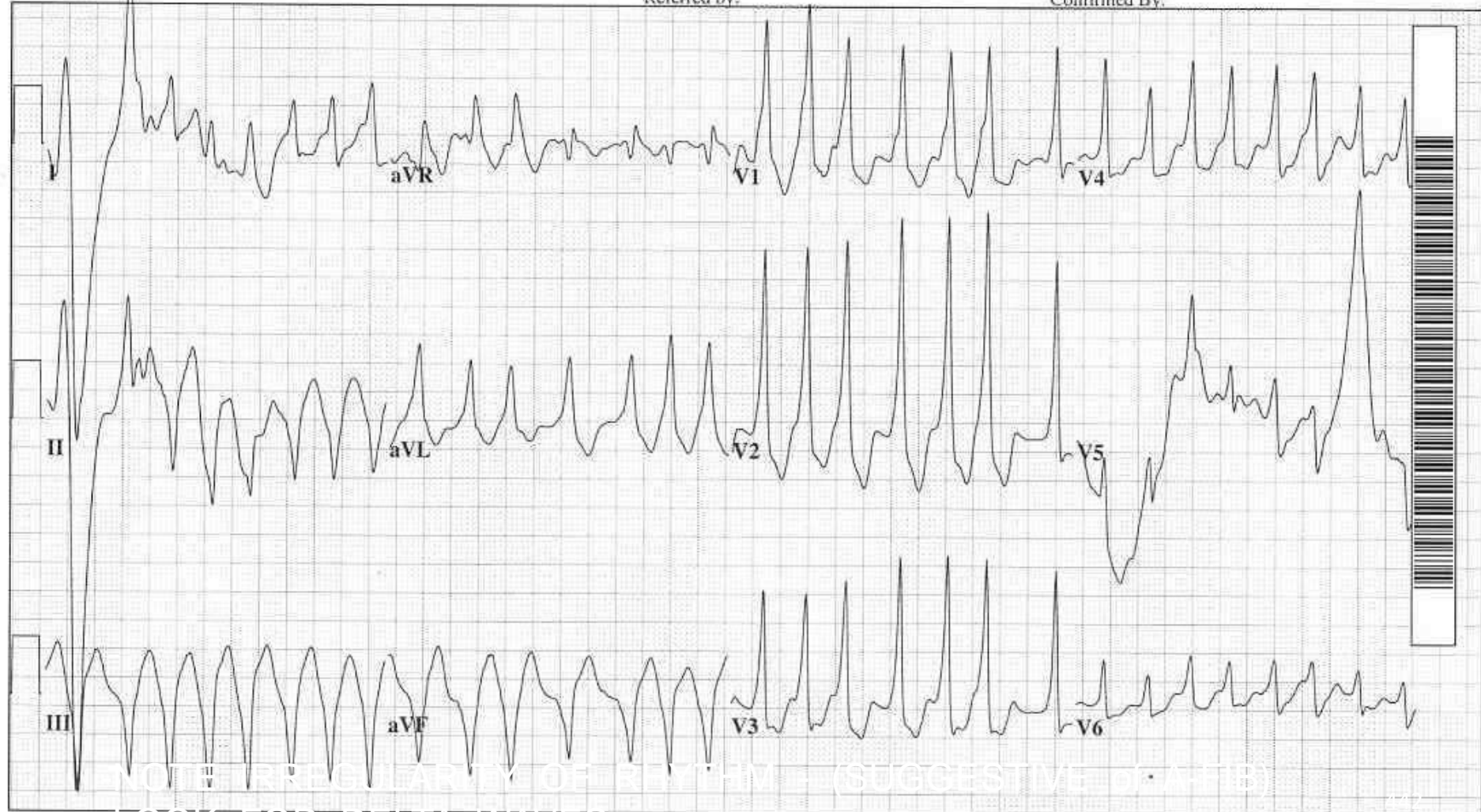
Vent. rate 180 BPM
PR interval * ms
QRS duration 148 ms
QT/QTc 284/491 ms
P-R-T axes * -77 103

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

Med: Unknown

Referred by:

Confirmed By:



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 y/o male

After the patient was defibrillated, sinus rhythm with good perfusion was restored.

A 12 Lead EKG obtained revealed

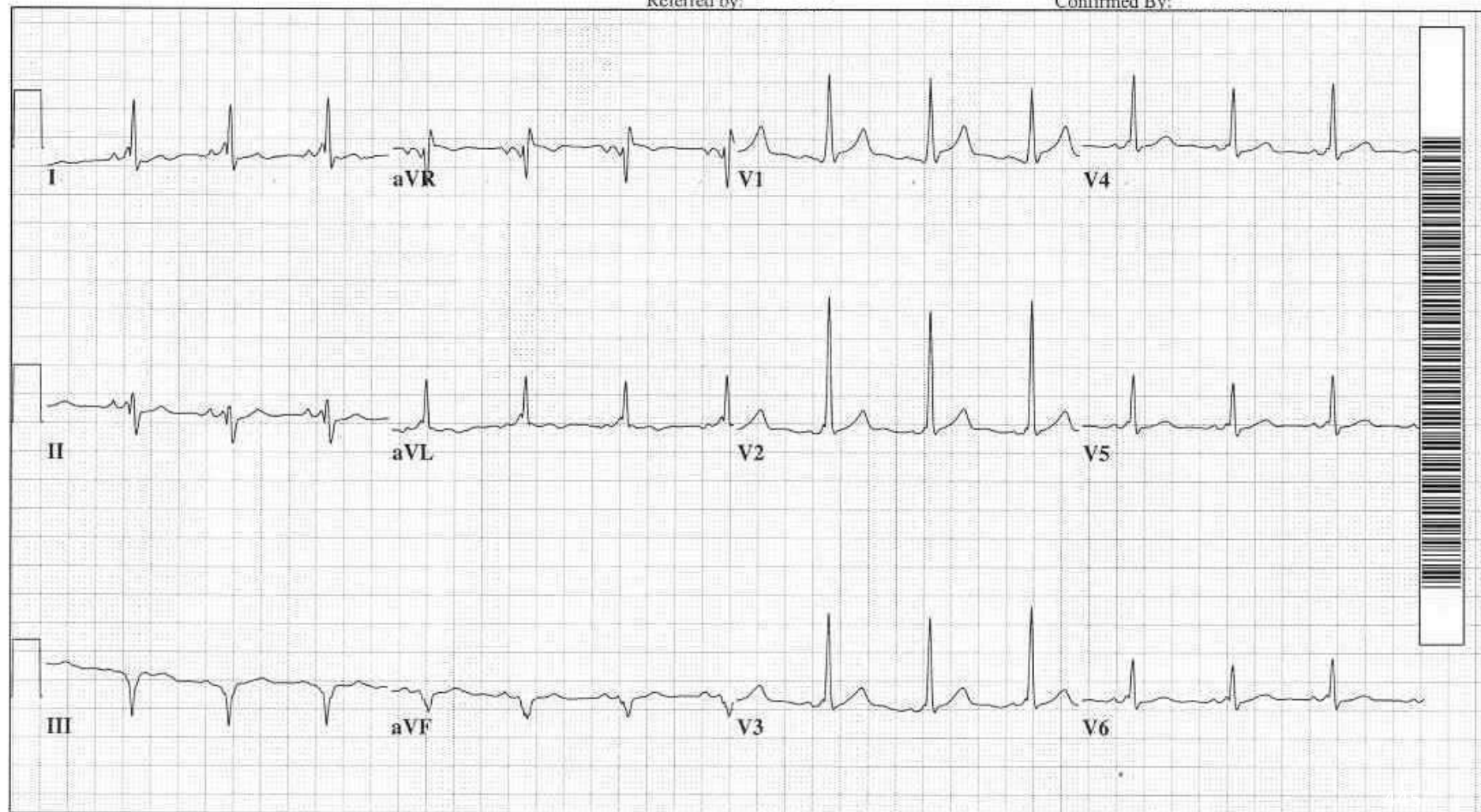
37 yr
Male Caucasian
Room:OP
Loc:8 Option:19

Vent. rate 82 BPM
PR interval 132 ms
QRS duration 128 ms
QT/QTc 392/458 ms
P-R-T axes 77 -44 154

Normal sinus rhythm
Ventricular pre-excitation, WPW pattern type A
Abnormal ECG

Referred by:

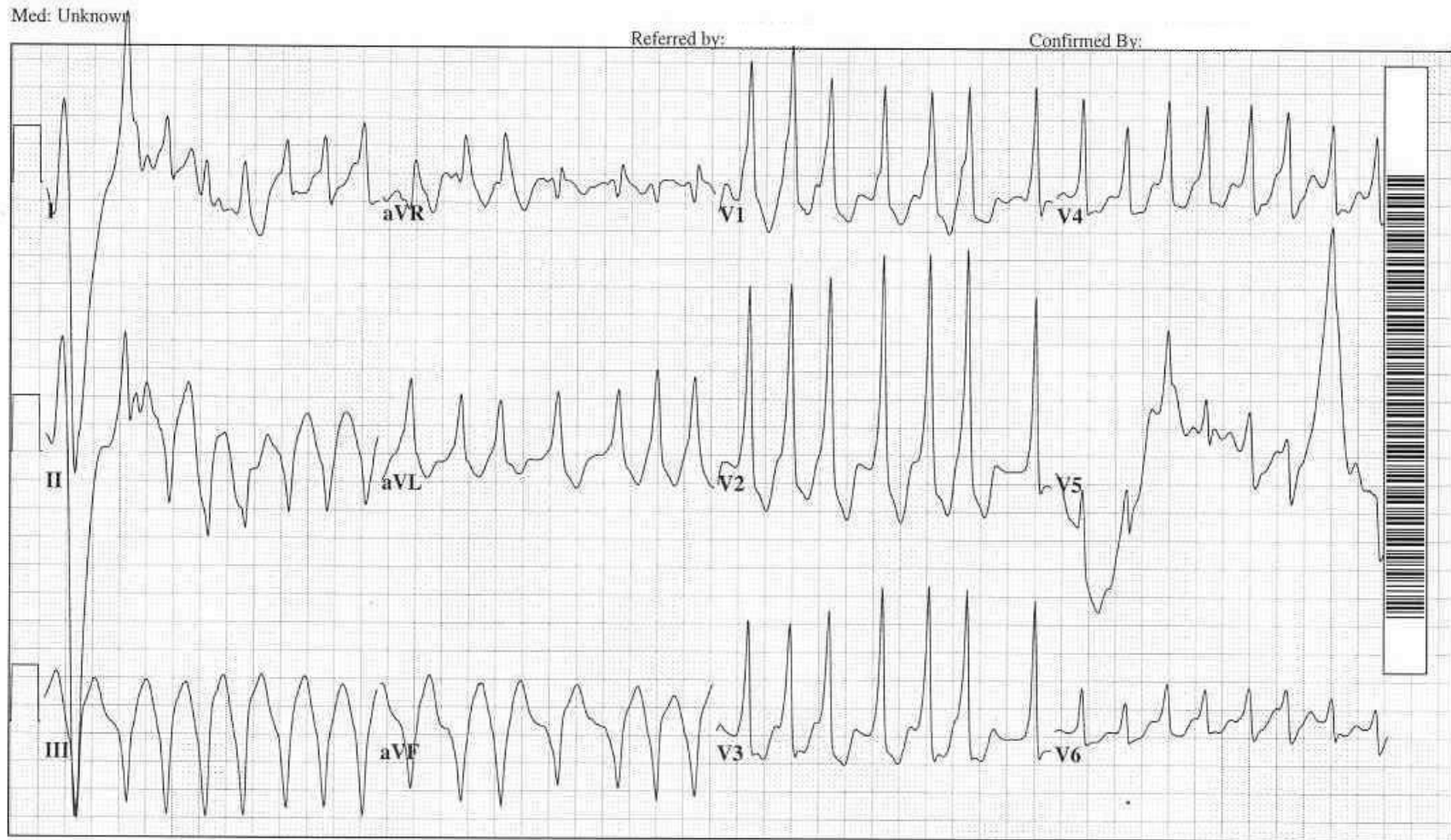
Confirmed By:



37 yr
Male Caucasian
Room: OP
Loc: 8 Option: 16

Vent. rate 180 BPM
PR interval * ms
QRS duration 148 ms
QT/QTc 284/491 ms
P-R-T axes * -77 103

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



25mm/s 10mm/mV 40Hz 005C 12SL 72 CID: 0

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

17 year old male: W-P-W with Afib & RVR



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

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

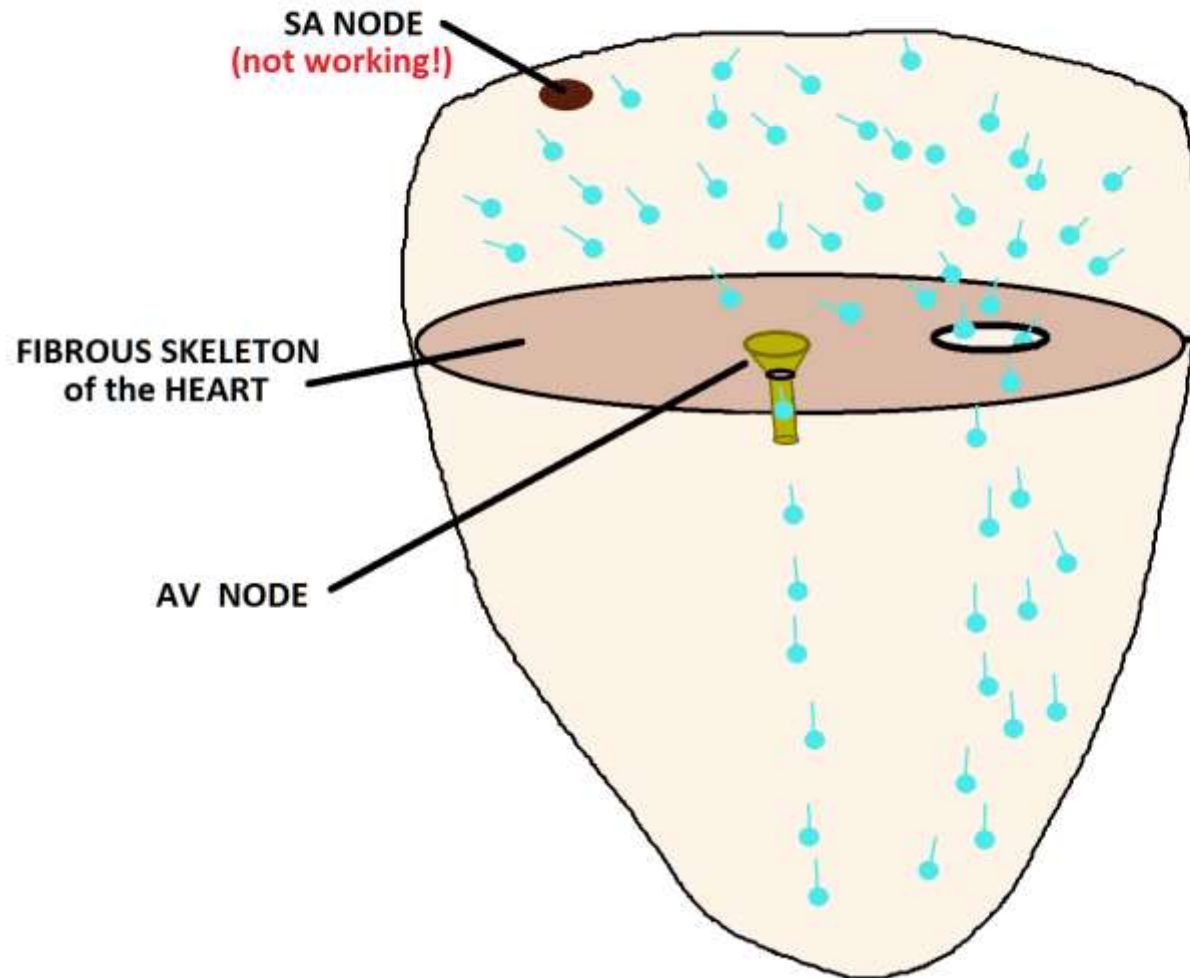
NOTE:

**Delta Waves
may not be
discernable !**



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

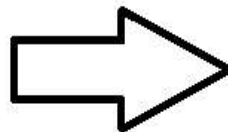
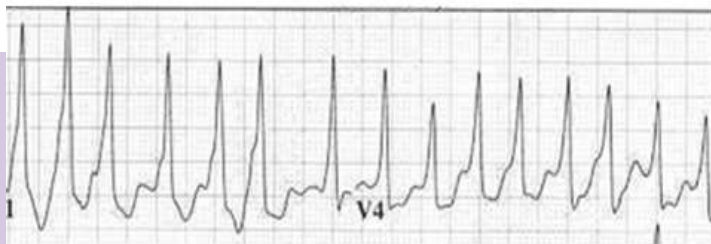
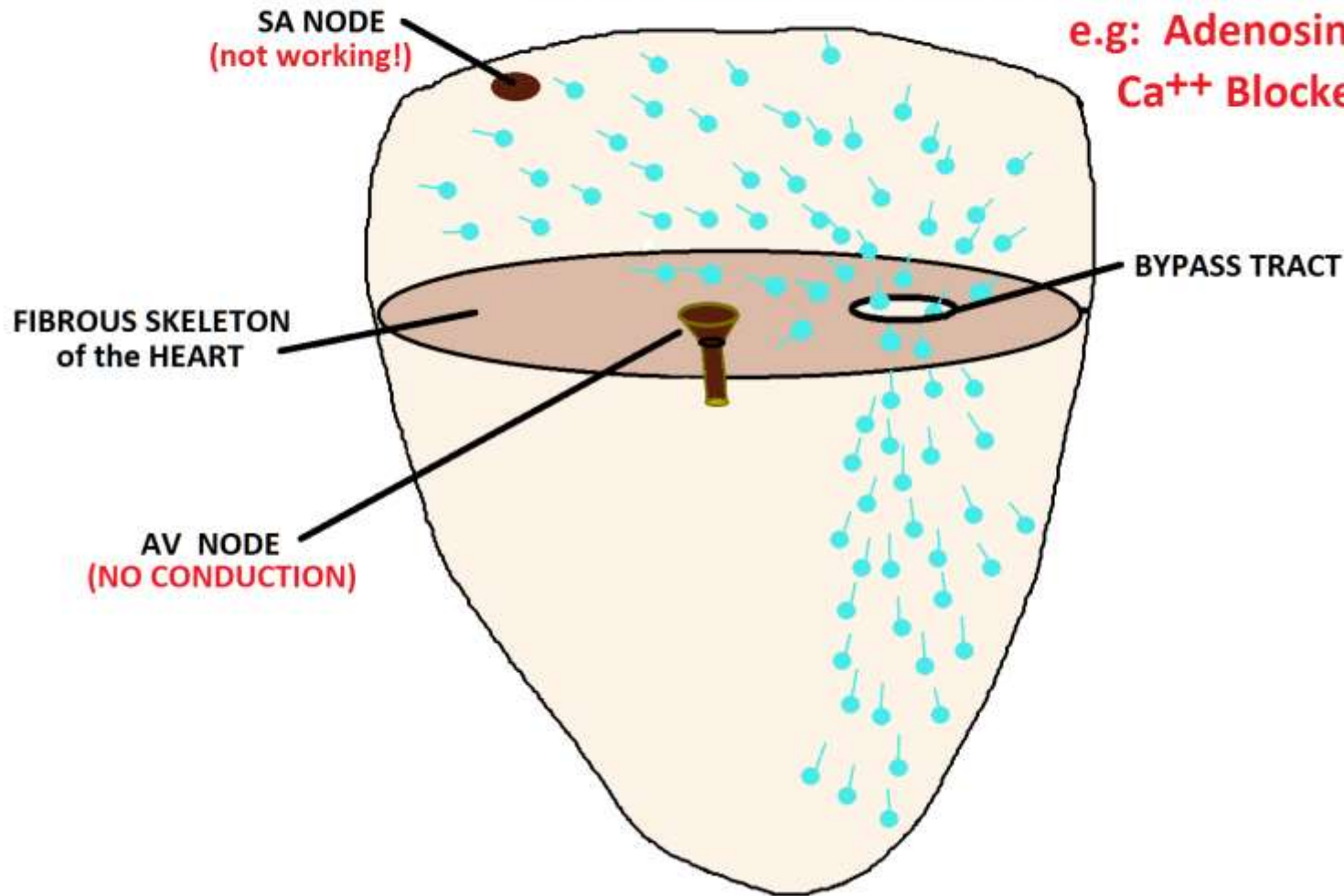
Atrial Fibrillation with Wolff-Parkinson White

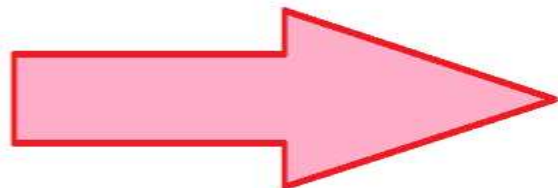


Atrial Fibrillation with Wolff-Parkinson White

with AV NODAL BLOCKING AGENTS

e.g: Adenosine,
Ca⁺⁺ Blockers





Are any of these present: ?

- Delta waves / wide QRS tach
- History of Wolff-Parkinson-White

- Electrolytes /
- Hypothermia
- Alcohol / Meds / Drugs
- Electrocution
- Pulmonary Embolus
- Recent Chest Surgery

YES

NO

Consider Rate Control with PROCAINAMIDE or ILBUTIDE as needed, to keep HR < 100

Consider Rate Control with Calcium Antagonist or Beta Blocker, as needed, to keep HR < 100

Obtain STAT INR. Is INR in therapeutic range?

YES

NO

Initiate Anticoagulation Therapy to achieve INR of 2.0 - 3.0

Order TEE

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

ABCs

NO PULSE

GO TO
V - FIB
ALGORITHM !

PULSE - UNSTABLE

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

PULSE - STABLE

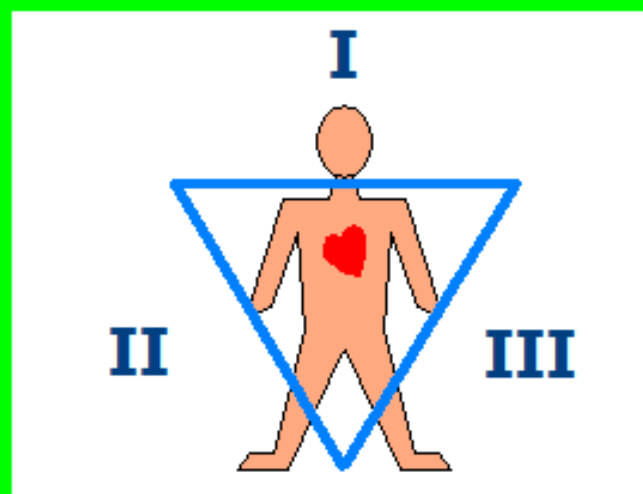
- O2, IV-IO, EKG
- MEDS:
 - ~~• ADENOSINE 6-12 (only if BENIGN)~~
 - PROCAINAMIDE (20-50mg/min)
 - ~~• AMIODARONE (150 mg 10min + 1mg/ min INFUSION)~~



EVALUATE THE AXIS IN BOTH PLANES

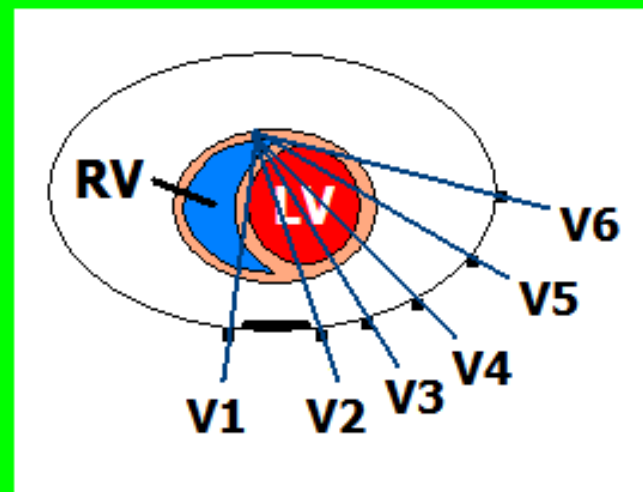
- **VERTICAL**

" **AXIS DEVIATION** "



- **HORIZONTAL**

" **AXIS ROTATION** "





AXIS DEVIATION

LEAD I

LEAD AVF

NORMAL



LEFT



RIGHT



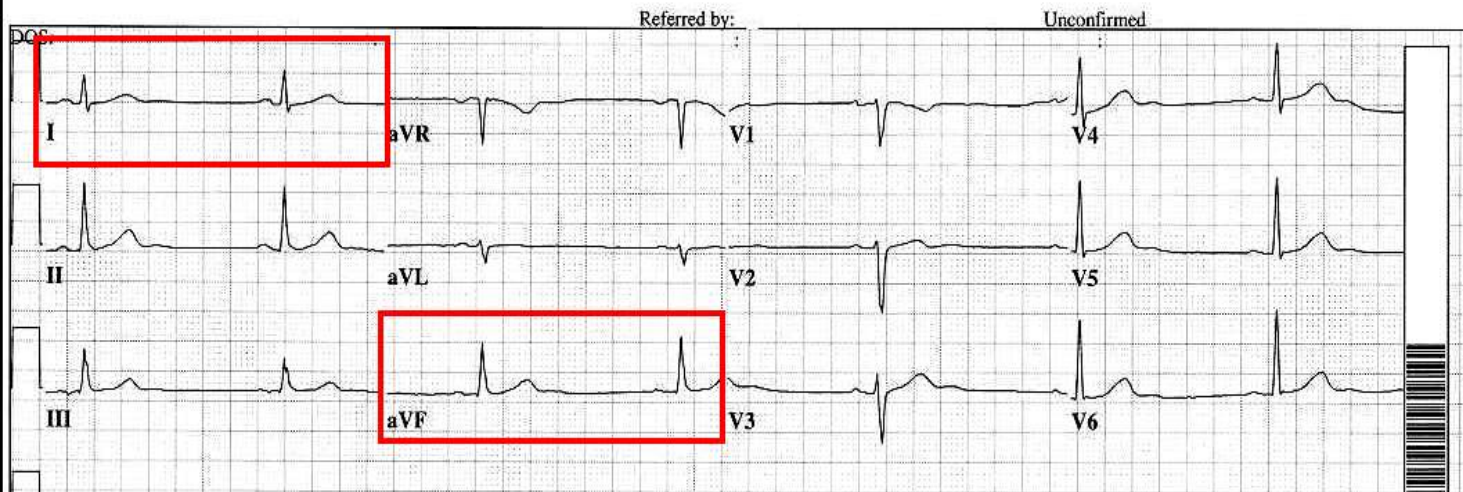
FAR RIGHT



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

NORMAL AXIS

Technician:



⚡️ AXIS DEVIATION

	<u>LEAD I</u>	<u>LEAD aVF</u>
NORMAL		
LEFT		
RIGHT		
FAR RIGHT		

74years		Vent. rate	72 bpm
Male	Caucasian	PR interval	186 ms
		QRS duration	166 ms
Room:		QT/QTc	436/477 ms
Loc: 0	Opt:	P-R-T axes	57 -32 32

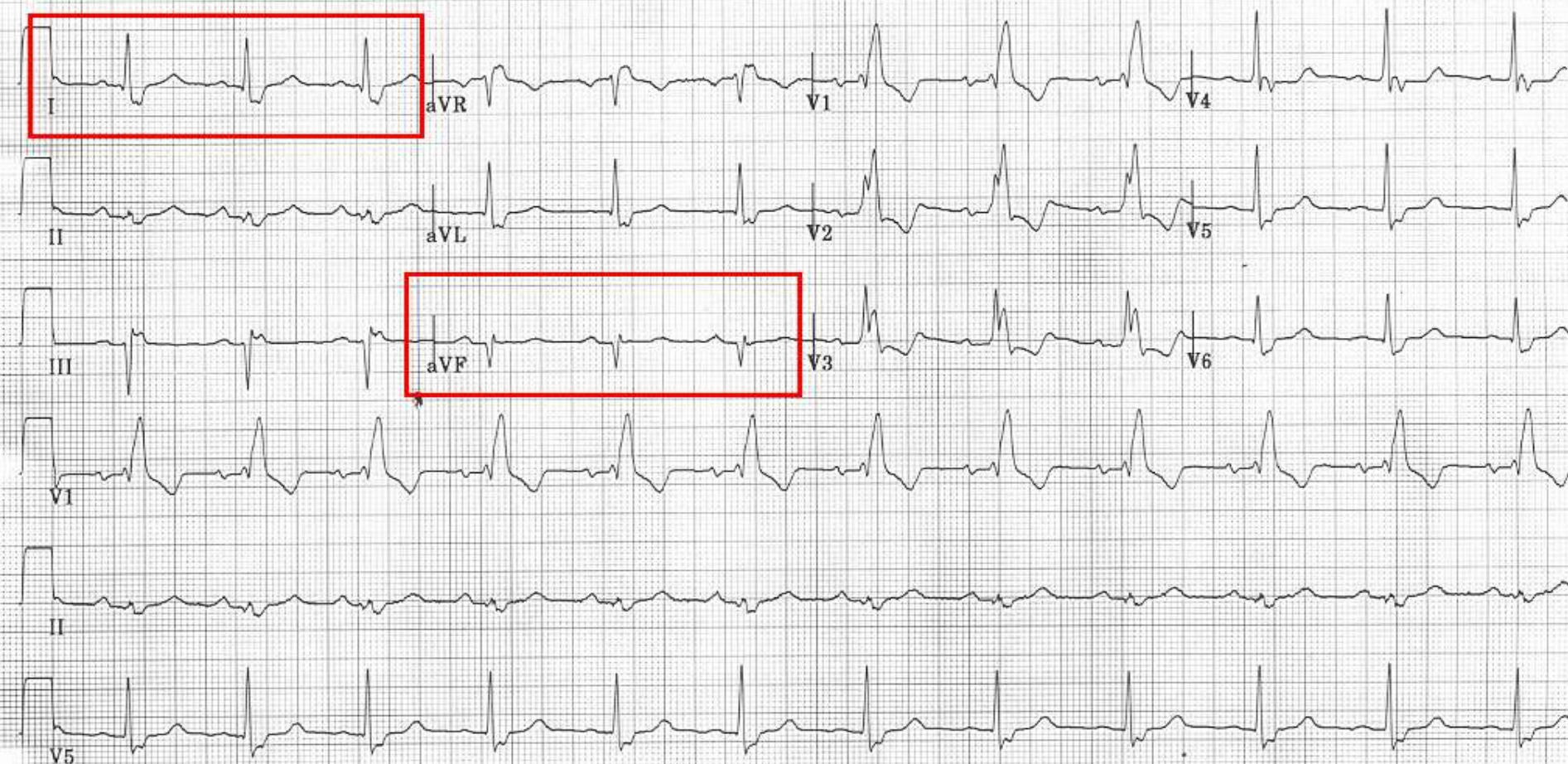
Technician: WR

What is the AXIS of this EKG ?

Referred by:

Unconfirmed

D.O.S.:





AXIS DEVIATION

LEAD I

LEAD AVF

NORMAL



LEFT











RIGHT



FAR RIGHT



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
-  WOLFF-PARKINSON-WHITE (types A & B)

11:18:02

81 yr
Female Hispanic
Room:303A
Loc:6 Option:11

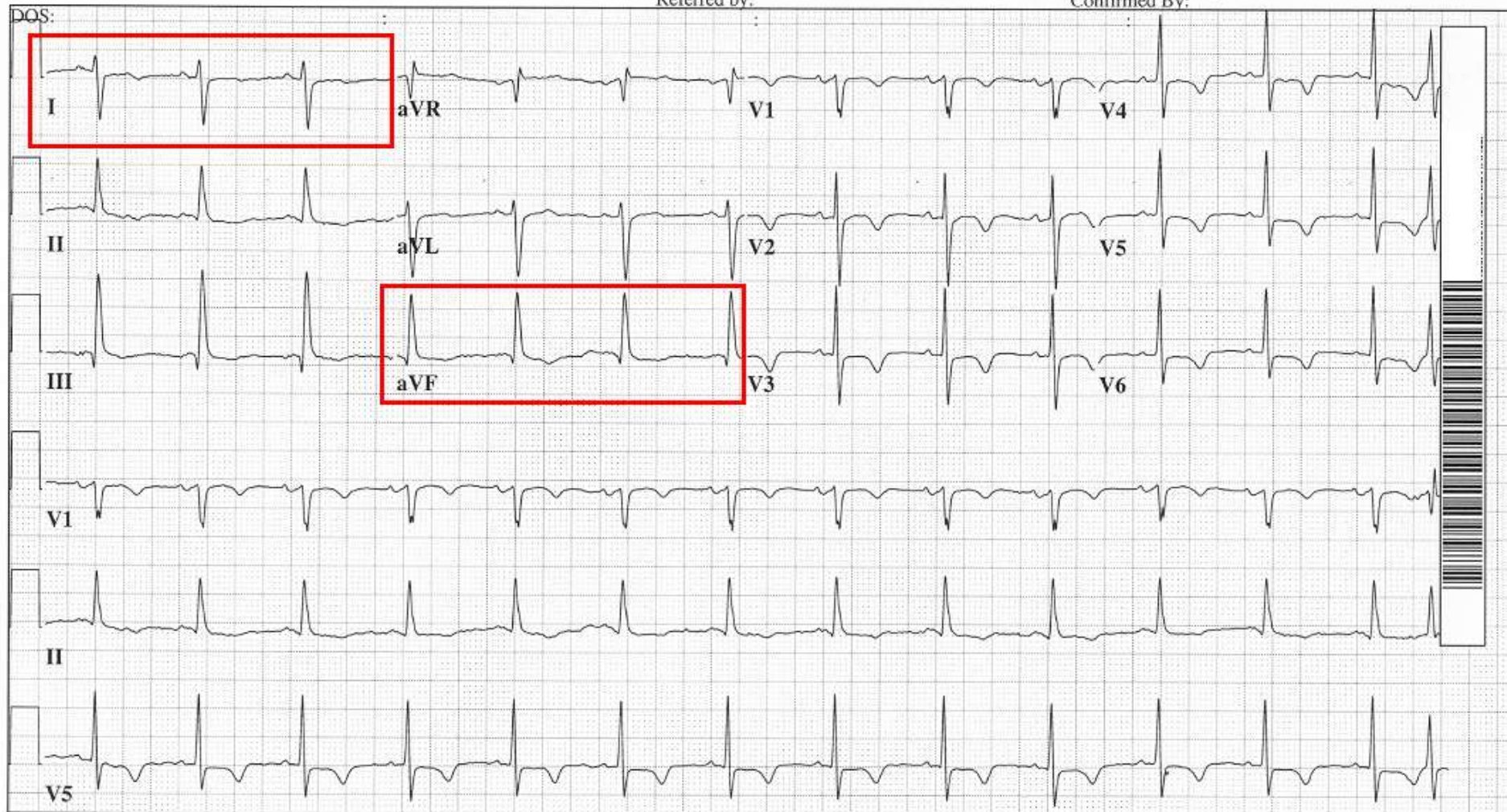
Vent. rate 82 BPM
PR interval 128 ms
QRS duration 86 ms
QT/QTc 392/457 ms
P-R-T axes 38 112 -142

What is the AXIS of this EKG ?

Technician: EKG CLASS CODE
WR03899892









Referred by:

Confirmed By:





AXIS DEVIATION

	<u>LEAD I</u>	<u>LEAD AVF</u>
NORMAL		
LEFT		
RIGHT		
FAR RIGHT		

COMMON CONDITIONS WHICH *MAY* CAUSE RIGHT AXIS DEVIATION:

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

02:55:00

Male Caucasian

Vent. rate 92 BPM

PR interval *

QRS duration 172 ms

QT/QTc 420/520 ms

P-R-T axes * -123 61

ACCELERATED IDIOVENTRICULAR RHYTHM

Room:5

Loc:1

EKG CLASS CODE #WR03611255

Referred by:

Confirmed By:



25mm/s 10mm/mV 100Hz 005C 12SL 4 CID: 5

EID:17 EDT:



AXIS DEVIATION

LEAD I

LEAD AVF

NORMAL



LEFT








RIGHT



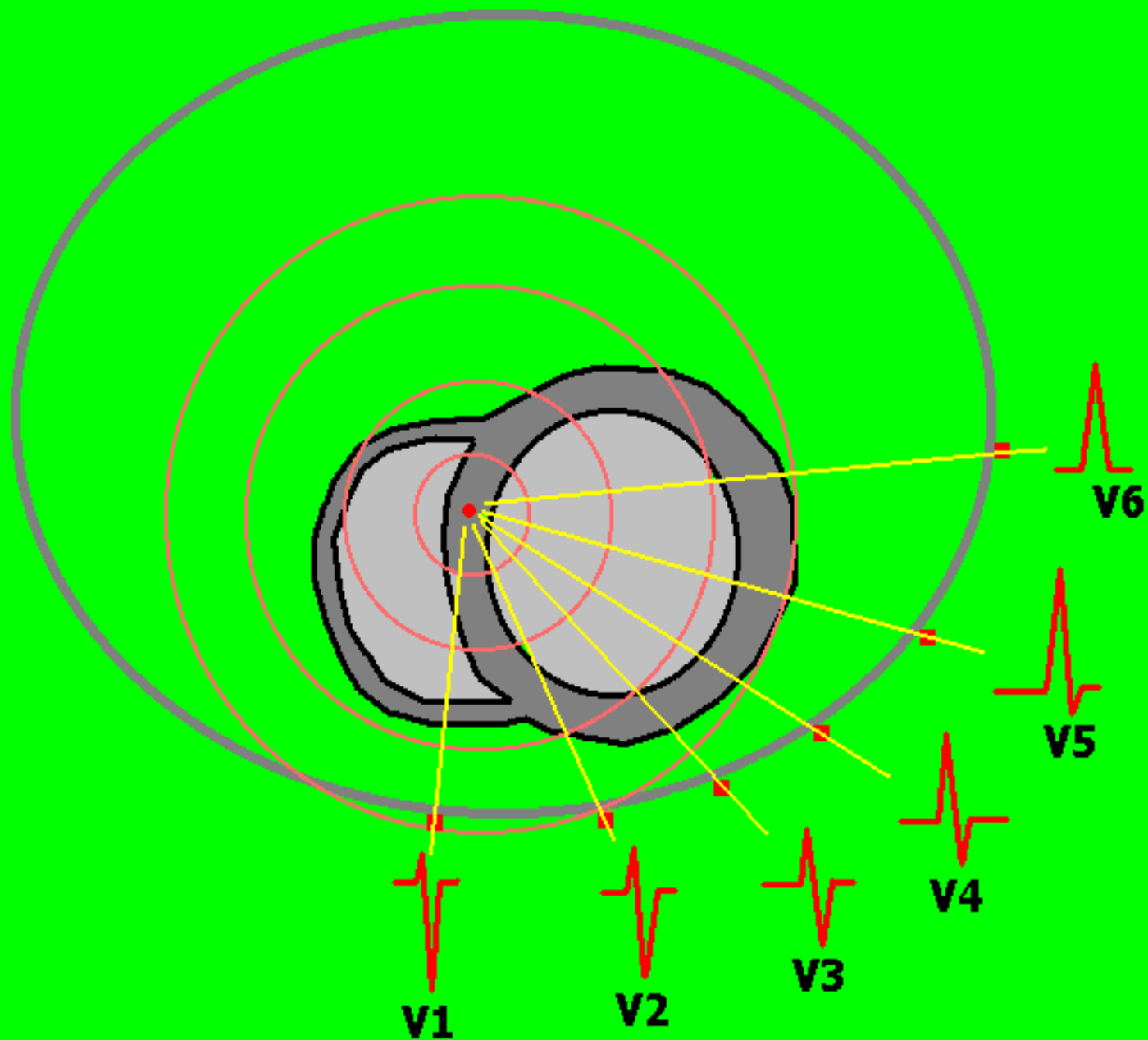
FAR RIGHT



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**

AXIS ROTATION



ASSESSING AXIS ROTATION:

V1

V2

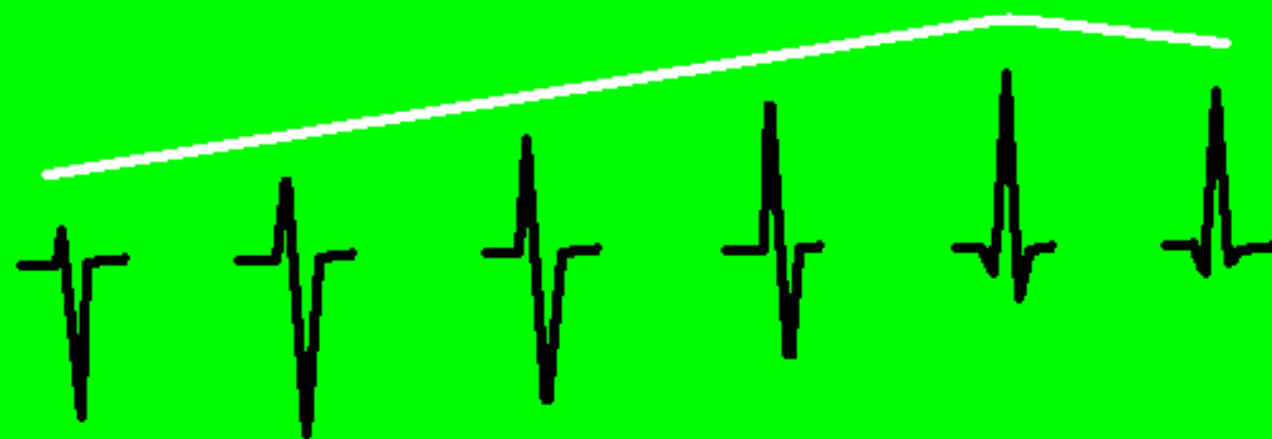
V3

V4

V5

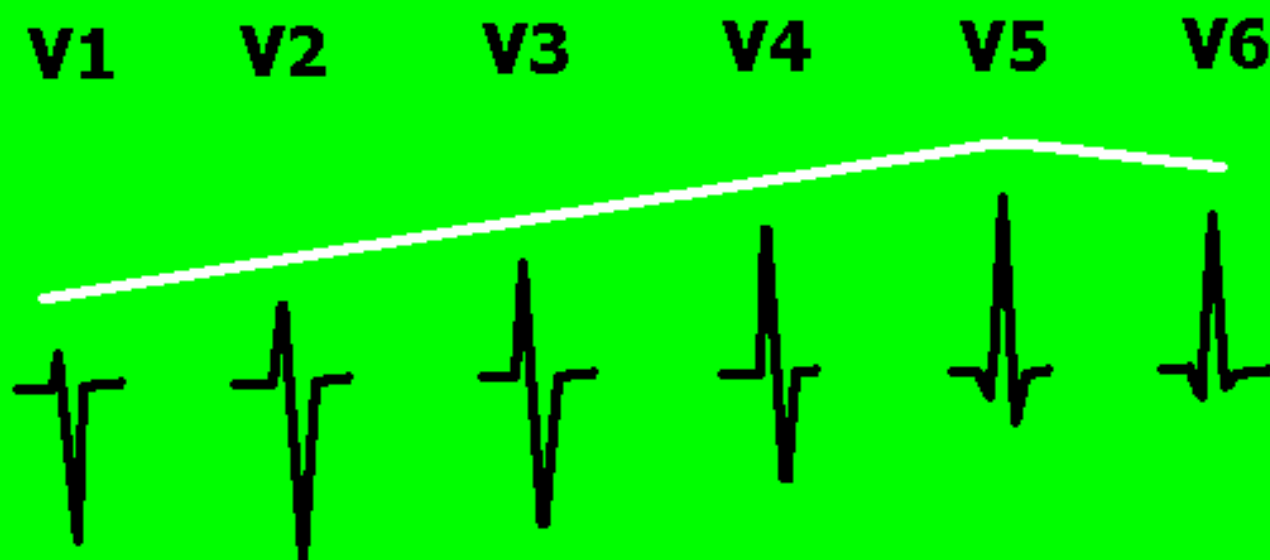
V6

1. 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**

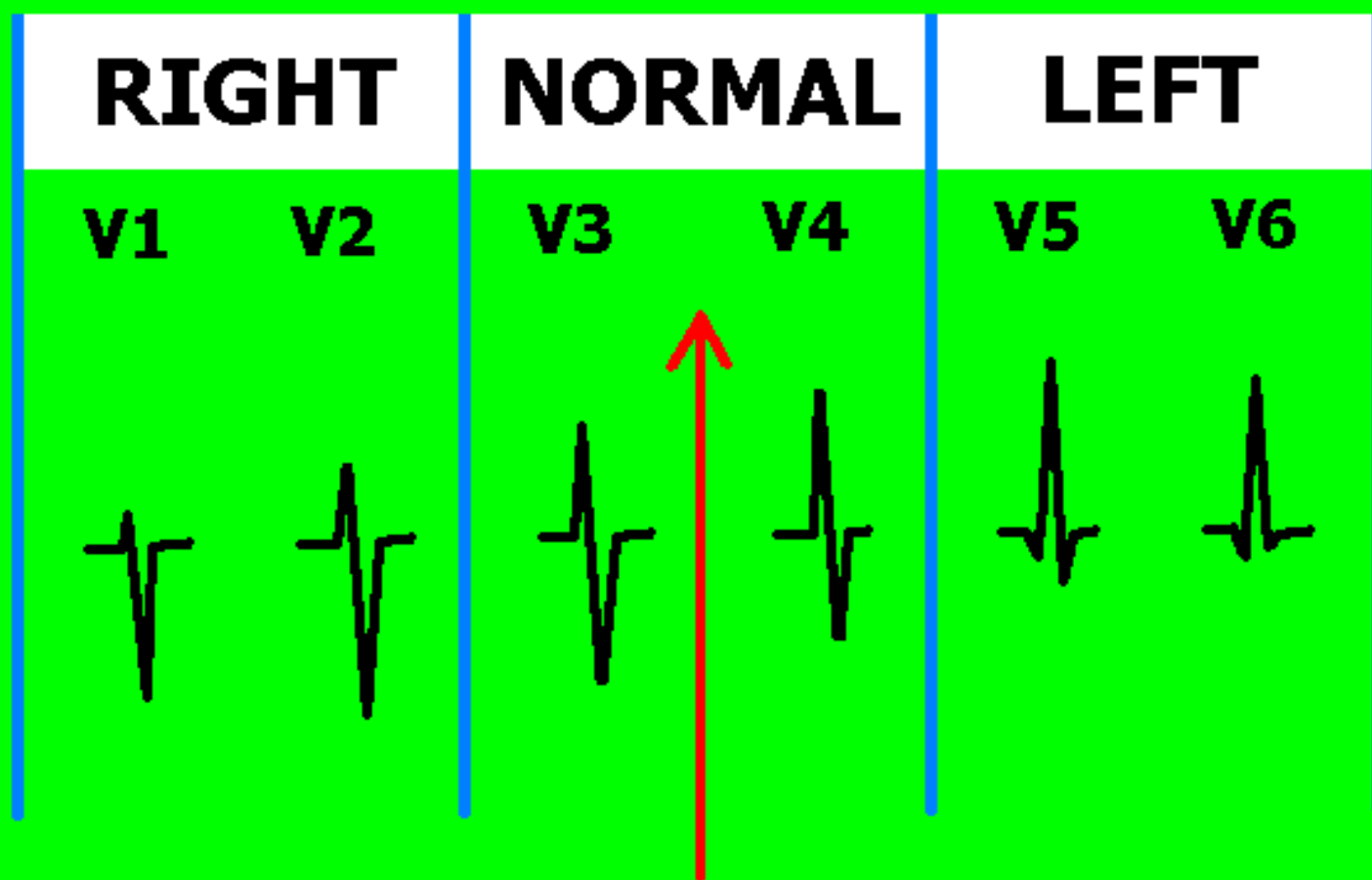
AXIS ROTATION

IMPORTANT TRANSITION RULE



**"Transition shifts TOWARD
HYPERTROPHY and AWAY
FROM NECROSIS."**

AXIS ROTATION

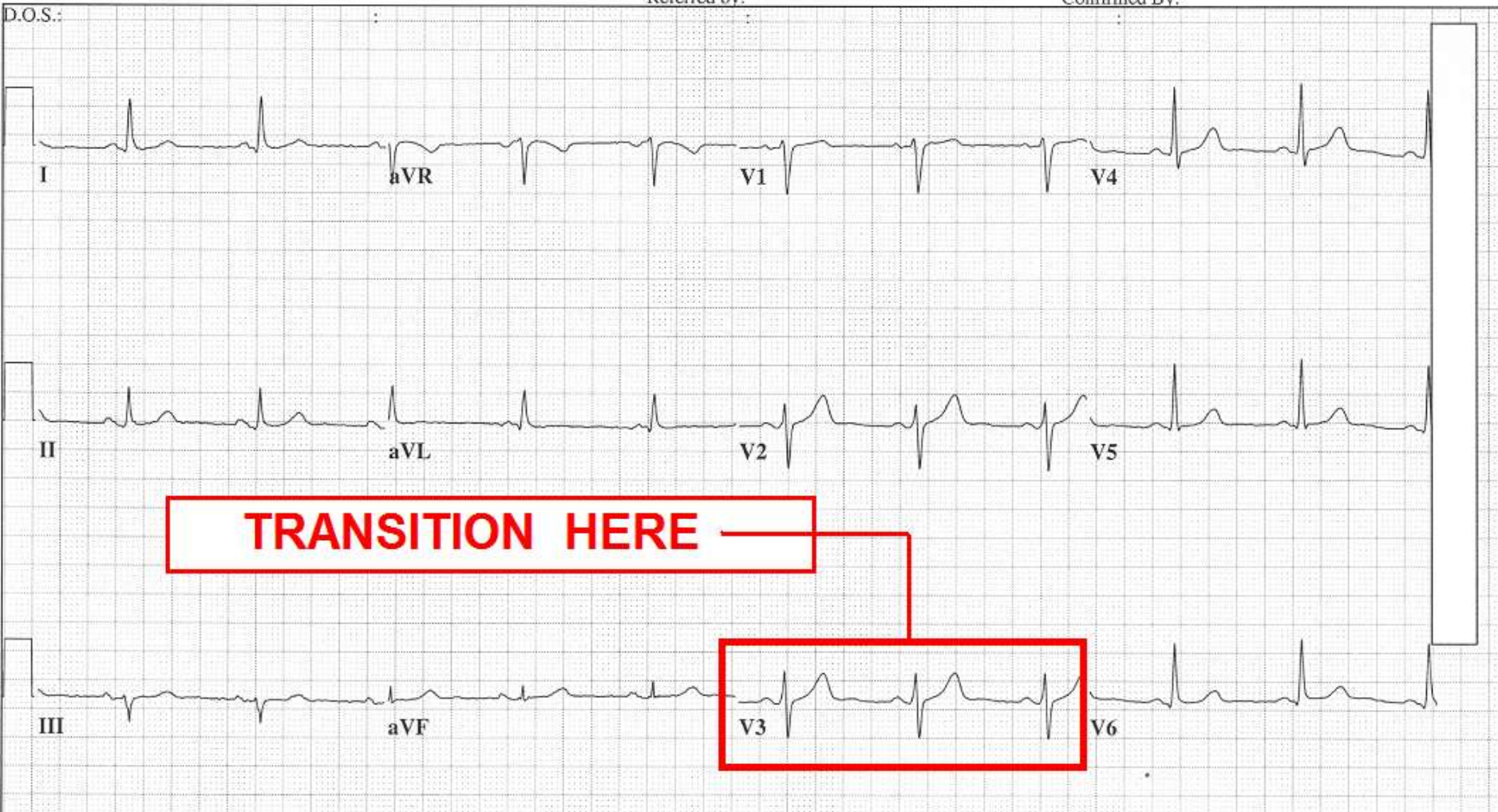


**TRANSITION SHOULD
OCCUR IN LEADS V3 or V4**

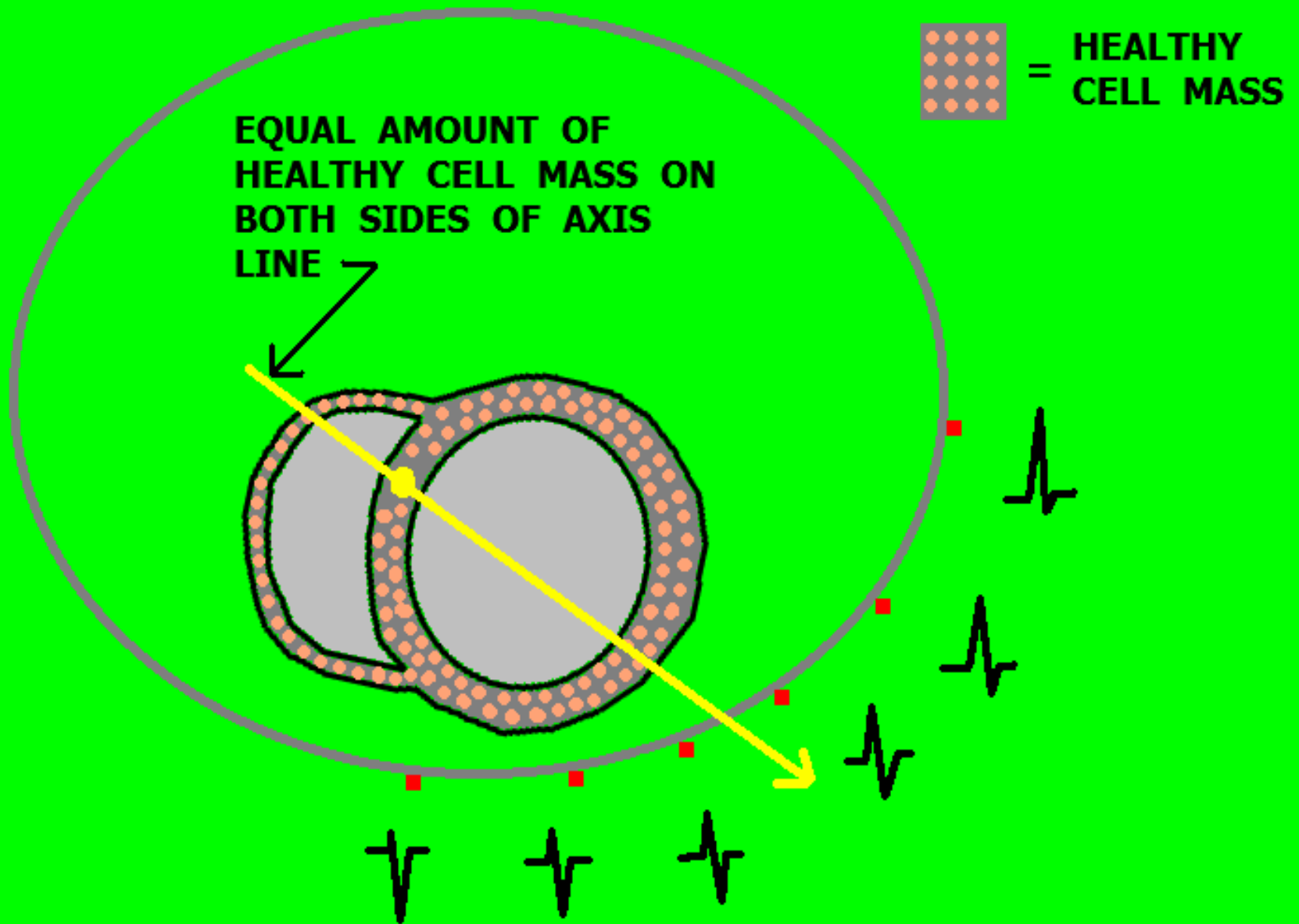
NORMAL TRANSITION IS BETWEEN LEADS V3 and V4

Referred by:

Confirmed By:



NORMAL TRANSITION

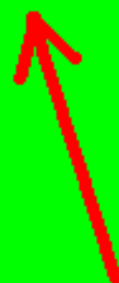
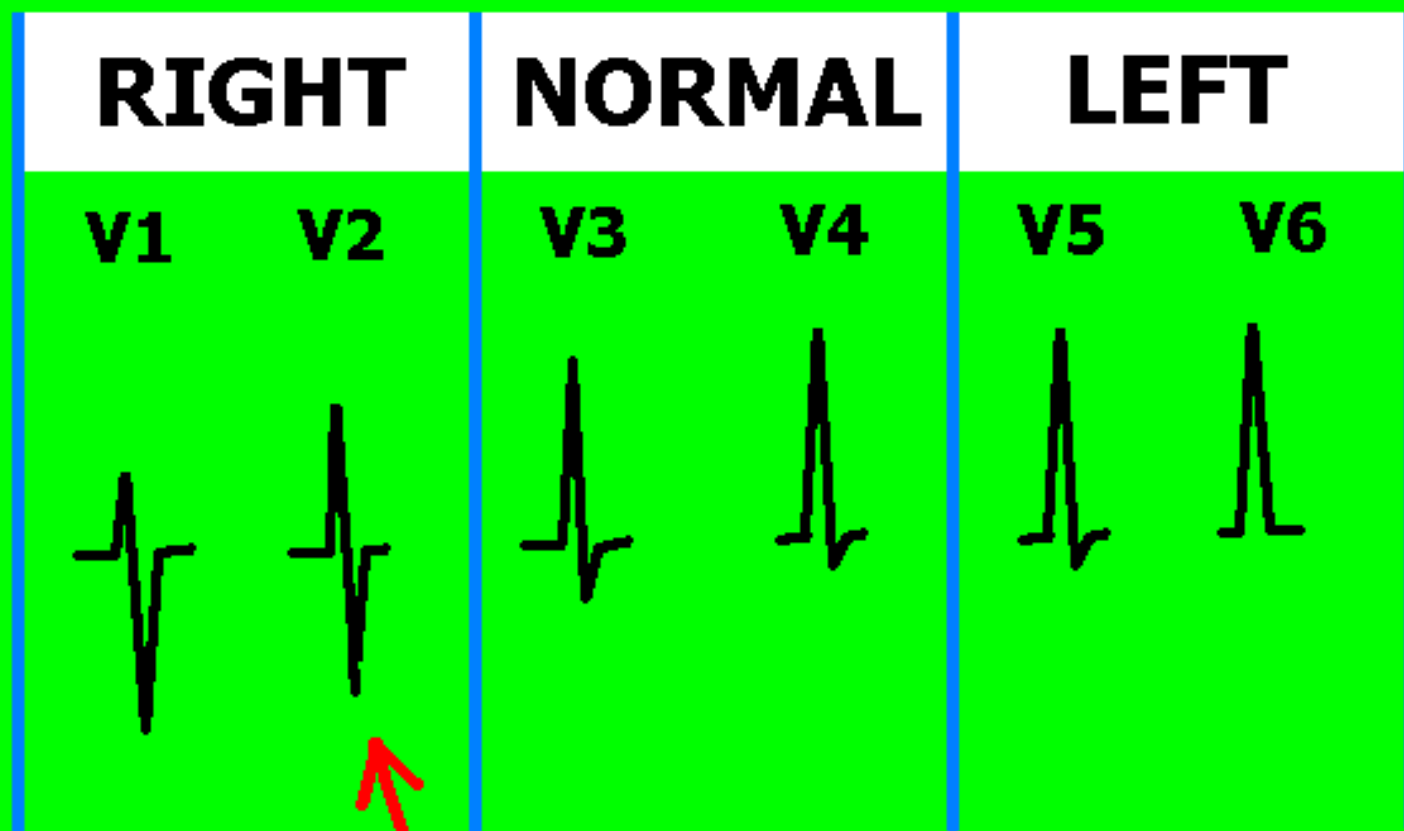


ASSESSING AXIS ROTATION:

IMPORTANT NOTES:

- ➡ As with all EKG-made DIAGNOSES, you must consider the TOTALITY of the PATIENT'S PRESENTATION. This includes the PATIENT'S CLINICAL PRESENTATION, RISK FACTOR PROFILE, and your INDEX OF SUSPICION.
- ➡ Validate all EKG-suspected DIAGNOSES with Additional, MORE ACCURATE diagnostic testing, e.g.: CARDIAC ECHO, CARDIAC CATHERIZATION, ELECTROPHYSIOLOGIC TESTING, MRI, etc.

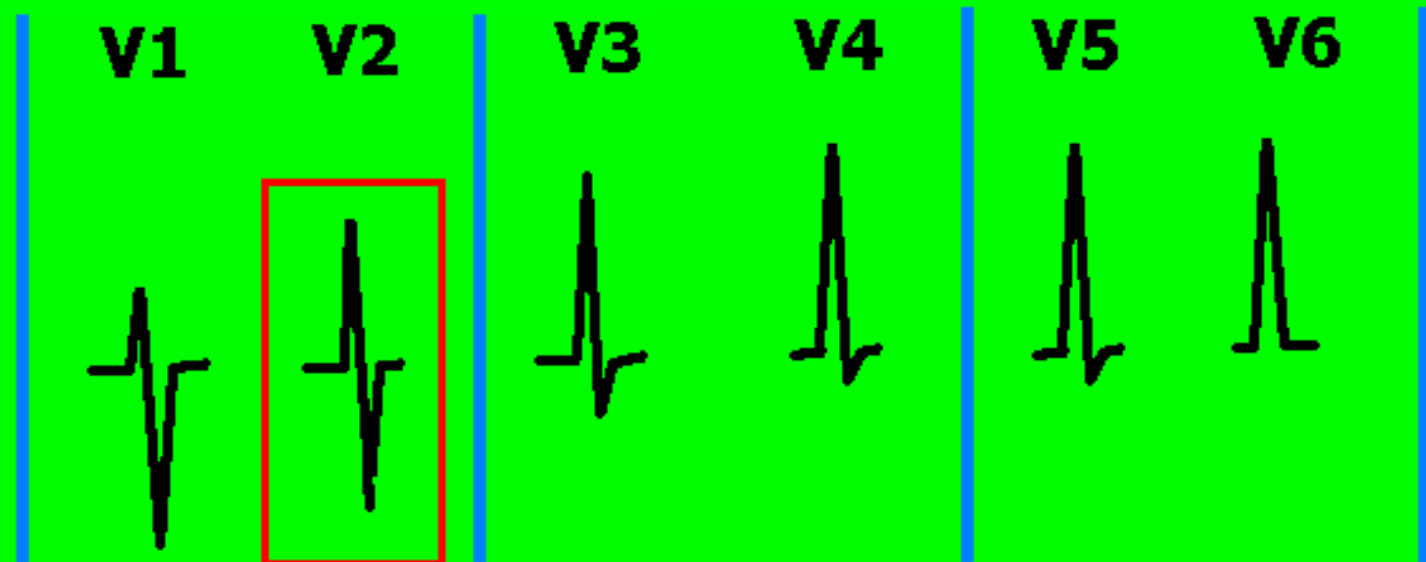
AXIS ROTATION



"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

74years
Male Caucasian
Room: Loc: 0
Opt:
Technician: WR

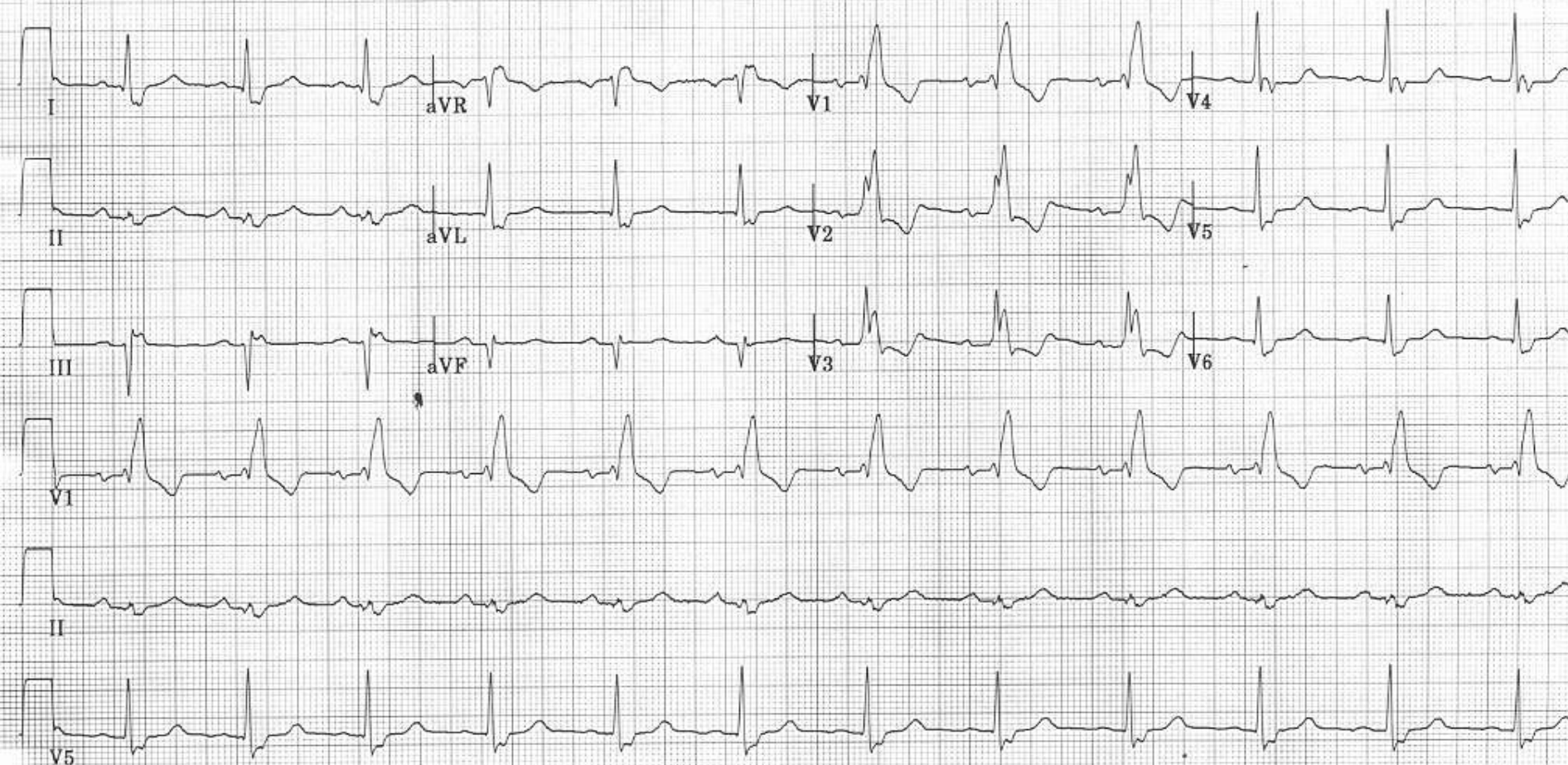
Vent. rate 72 bpm
PR interval 186 ms
QRS duration 166 ms
QT/QTc 436/477 ms
P-R-T axes 57 -32 32

What is the cause of EARLY TRANSITION in this EKG? -- Use the list of COMMON CAUSES OF EARLY TRANSITION to rule out different causes . . .

Referred by:

Unconfirmed

D.O.S.:



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 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

74years
Male Caucasian
Room: 0
Loc: 0
Opt:
Technician: WR

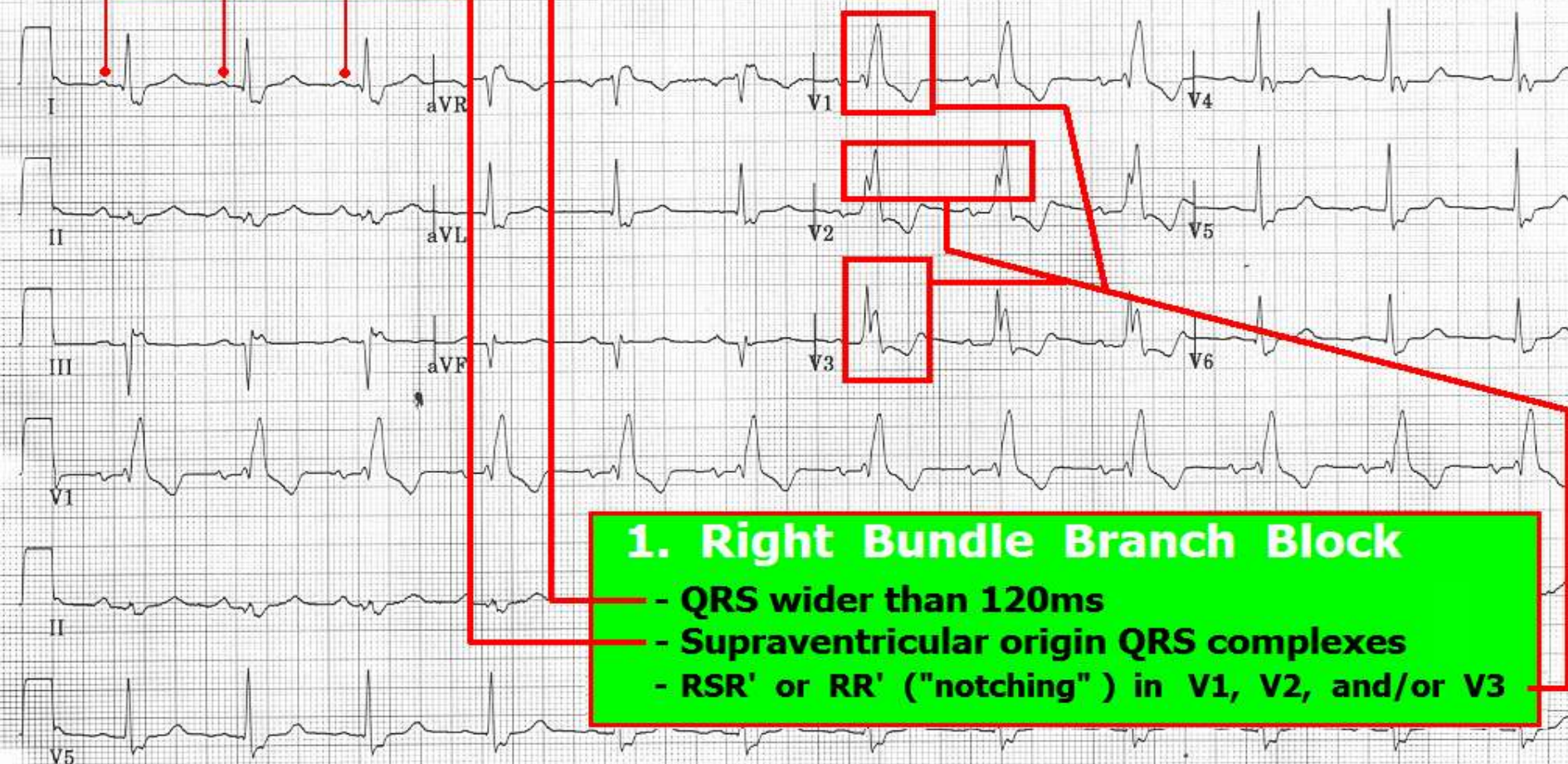
Vent. rate 72 bpm
PR interval 186 ms
QRS duration 166 ms
QT/QTc 436/477 ms
P-R-T axes 57 -32 32

Normal sinus rhythm
Left axis deviation
Right bundle branch block
Inferior infarct, age undetermined
Abnormal ECG

Referred by:

Unconfirmed

**P Waves precede each
QRS w/ reg. P-R int.**



1. Right Bundle Branch Block

- QRS wider than 120ms
- Supraventricular origin QRS complexes
- RSR' or RR' ("notching") in V1, V2, and/or V3

31 yr
Male Black
Room:ER
Loc:3 Option:16

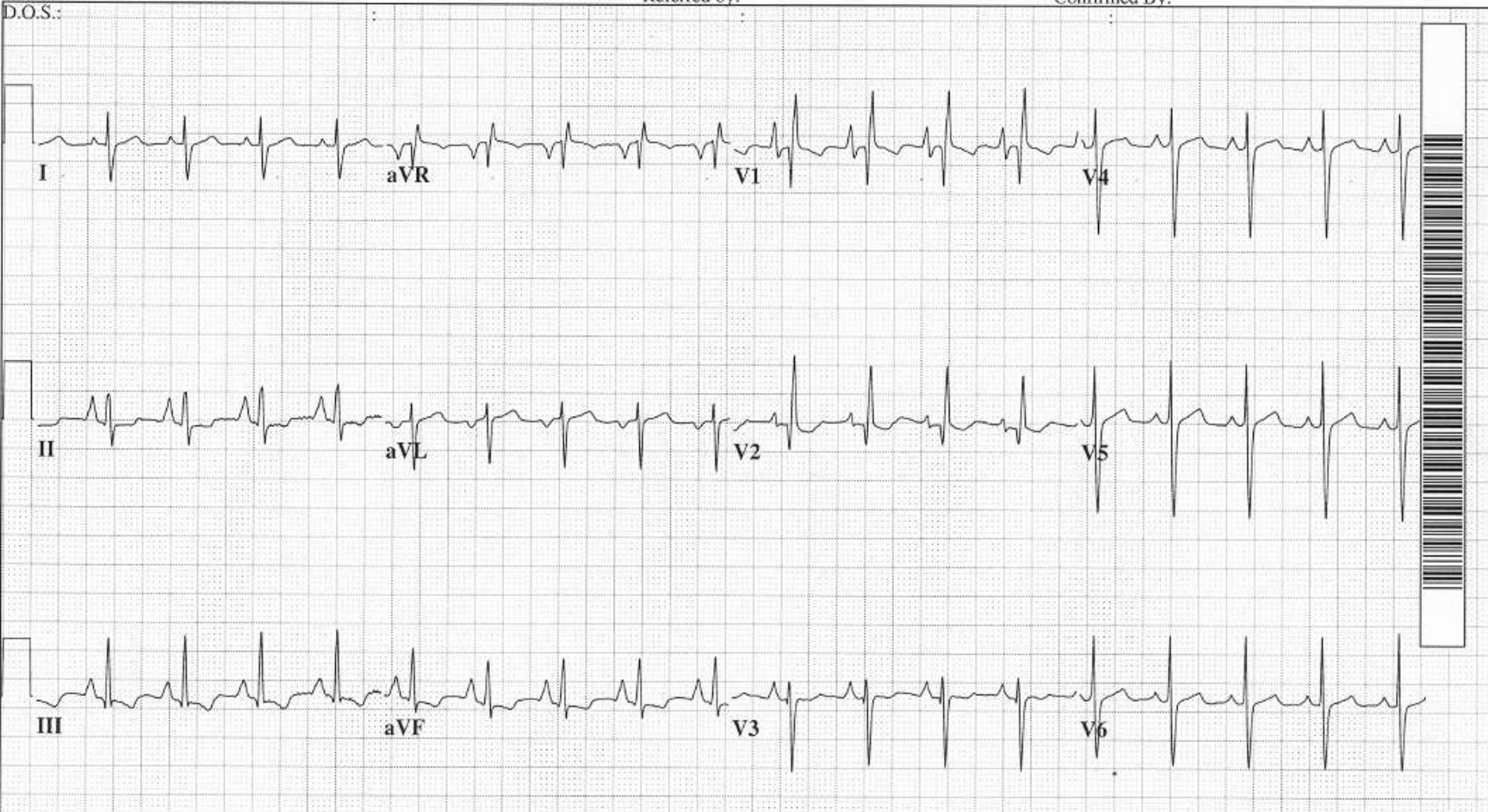
Vent. rate 109 BPM
PR interval 122 ms
QRS duration 84 ms
QT/QTc 296/398 ms
P-R-T axes 79 117 -27

**What is the cause of
EARLY TRANSITION in
this EKG ?**

Technician: EKG CLASS #WR03446043

Referred by:

Confirmed By:



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 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~~

31 yr
Male Black
Room:ER
Loc:3 Option:16

Vent. rate 109 BPM
PR interval 122 ms
QRS duration 84 ms
QT/QTc 296/398 ms
P-R-T axes 79 117 -27

Sinus tachycardia

Right atrial enlargement

Right axis deviation

Right ventricular hypertrophy

Cannot rule out Anteroseptal infarct (cited on or before 13-SEP-1999)

ST & T wave abnormality, consider inferior ischemia

Abnormal ECG

When compared with ECG of 16-FEB-2000 13:11,

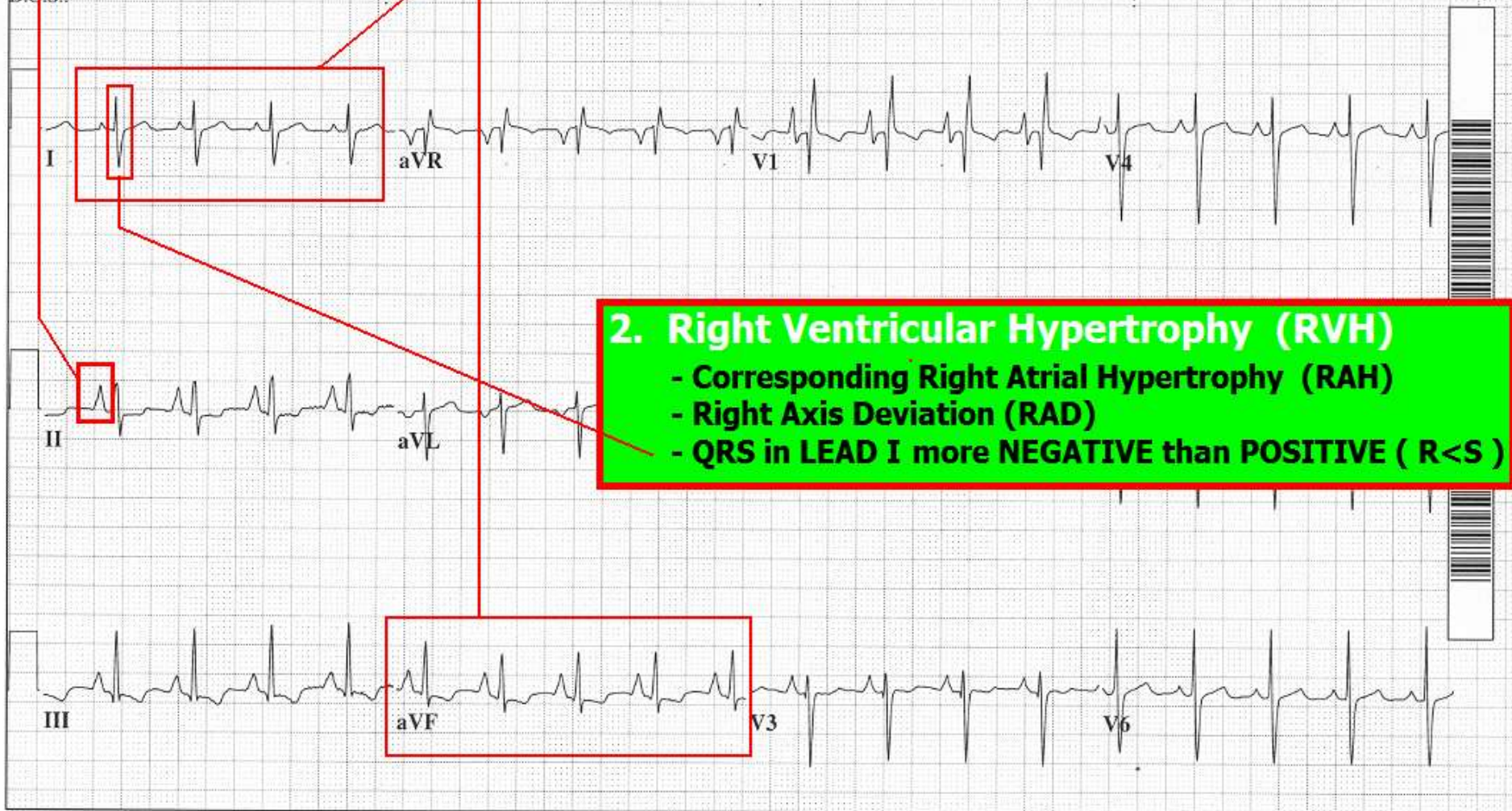
ST now depressed in Inferior leads ...

Technician: EKG CLASS #WR03446043

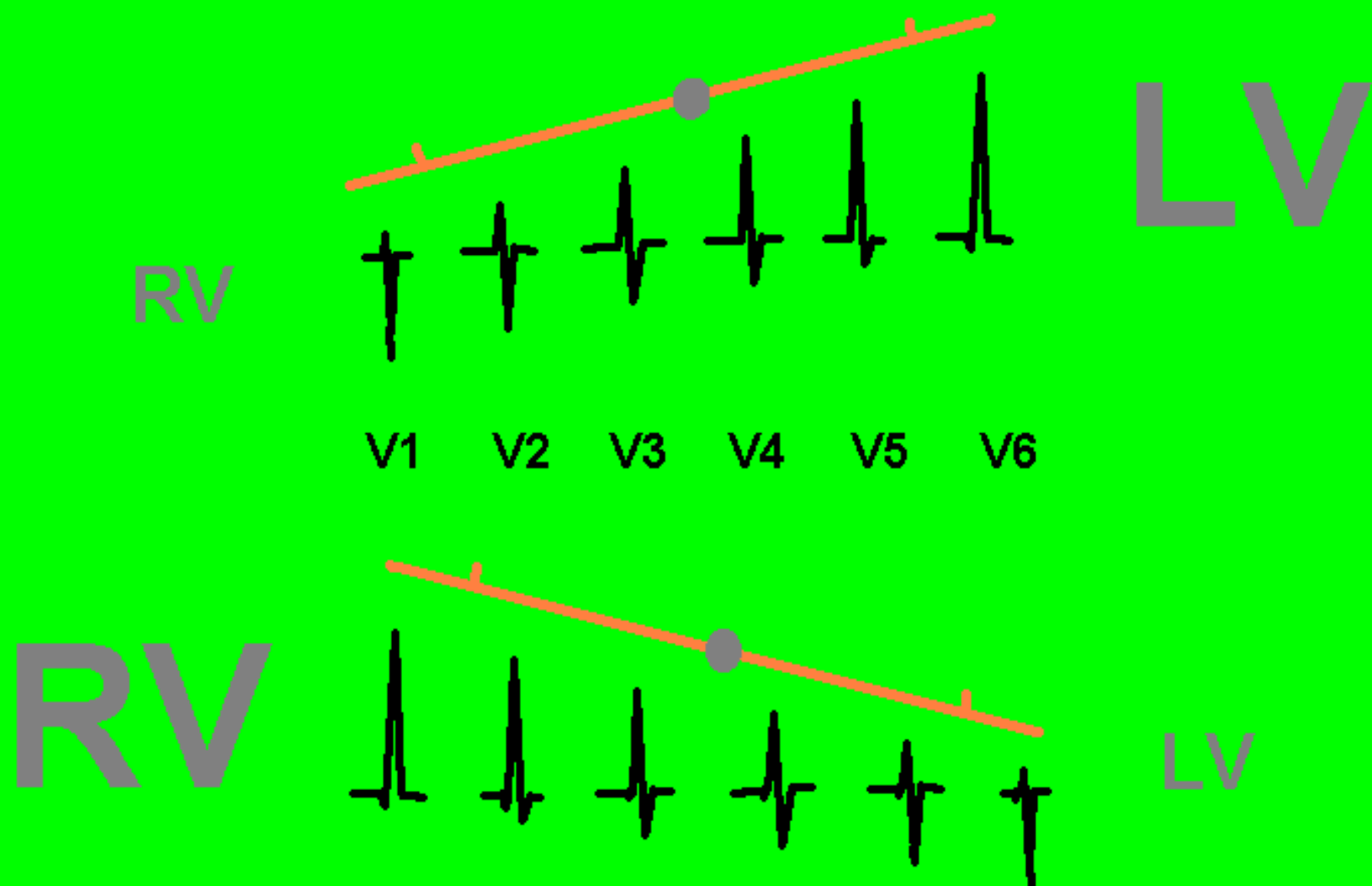
Referred by:

Confirmed By:

D.O.S.:



"SEE-SAW EFFECT" of RVH on R WAVE PROGRESSION



14-JUL-1997 14:30:58

ST. JOSEPH'S HOSPITAL-ER ROUTINE RETRIEVAL

17 yr
Male Black
Room:ER
Loc:3 Option:16

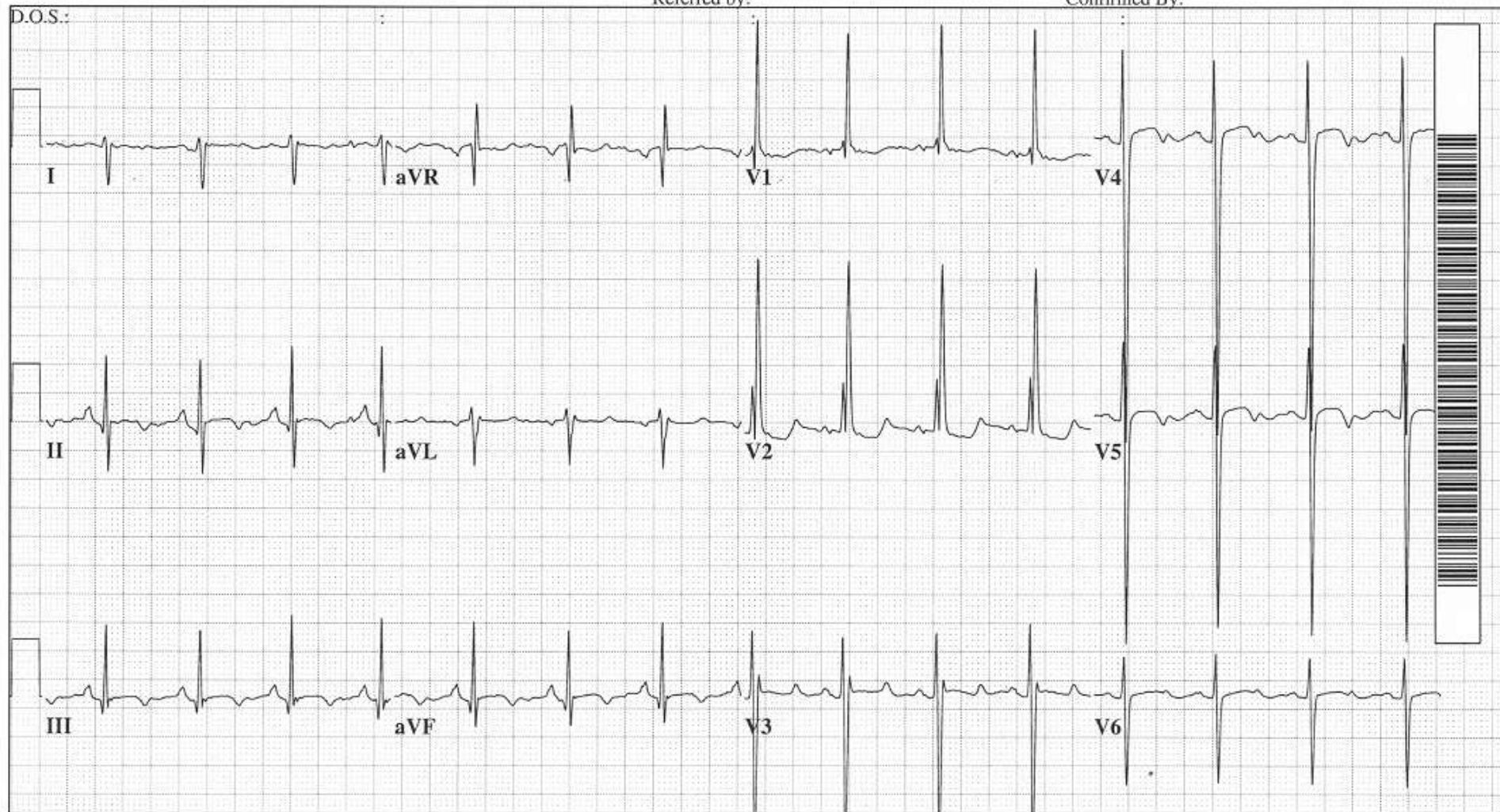
Vent. rate 90 BPM
PR interval 136 ms
QRS duration 94 ms
QT/QTc 378/462 ms
P-R-T axes 77 123 58

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

Technician: EKG CLASS #WR03616941

Referred by:

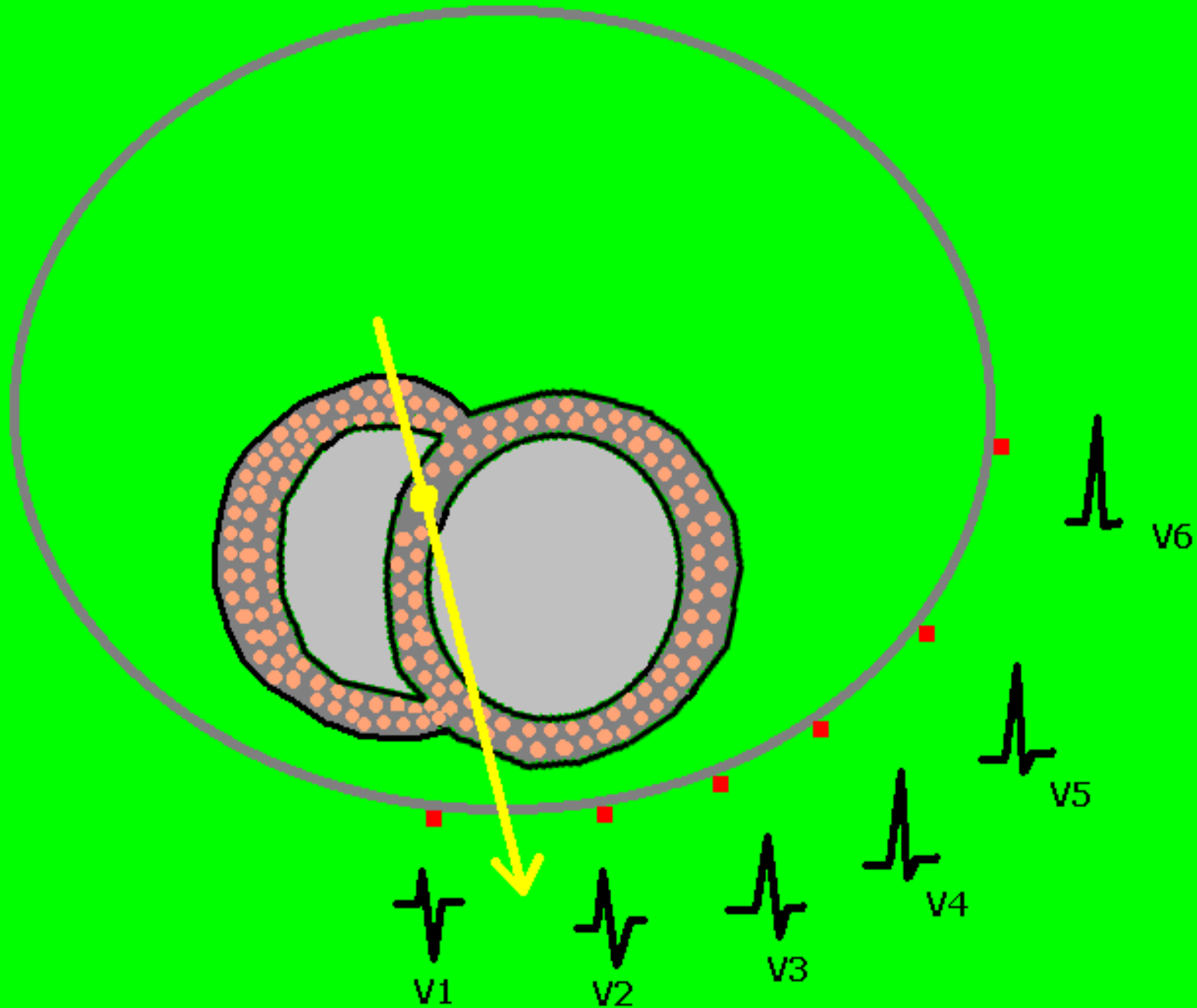
Confirmed By:



25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 11

EID:11 EDT: 17:04 15-JUL-1997 ORDER:

RIGHT VENTRICULAR HYPERTROPHY



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 EARLY TRANSITION

.....SOME HELPFUL CLUES:

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

- ~~- QRS wider than 120ms~~
- ~~- Supraventricular rhythm (normal P : QRS relationship)~~
- ~~- PSR' 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~~

Male Caucasian
Room:CCU3
Loc:1 Option:1

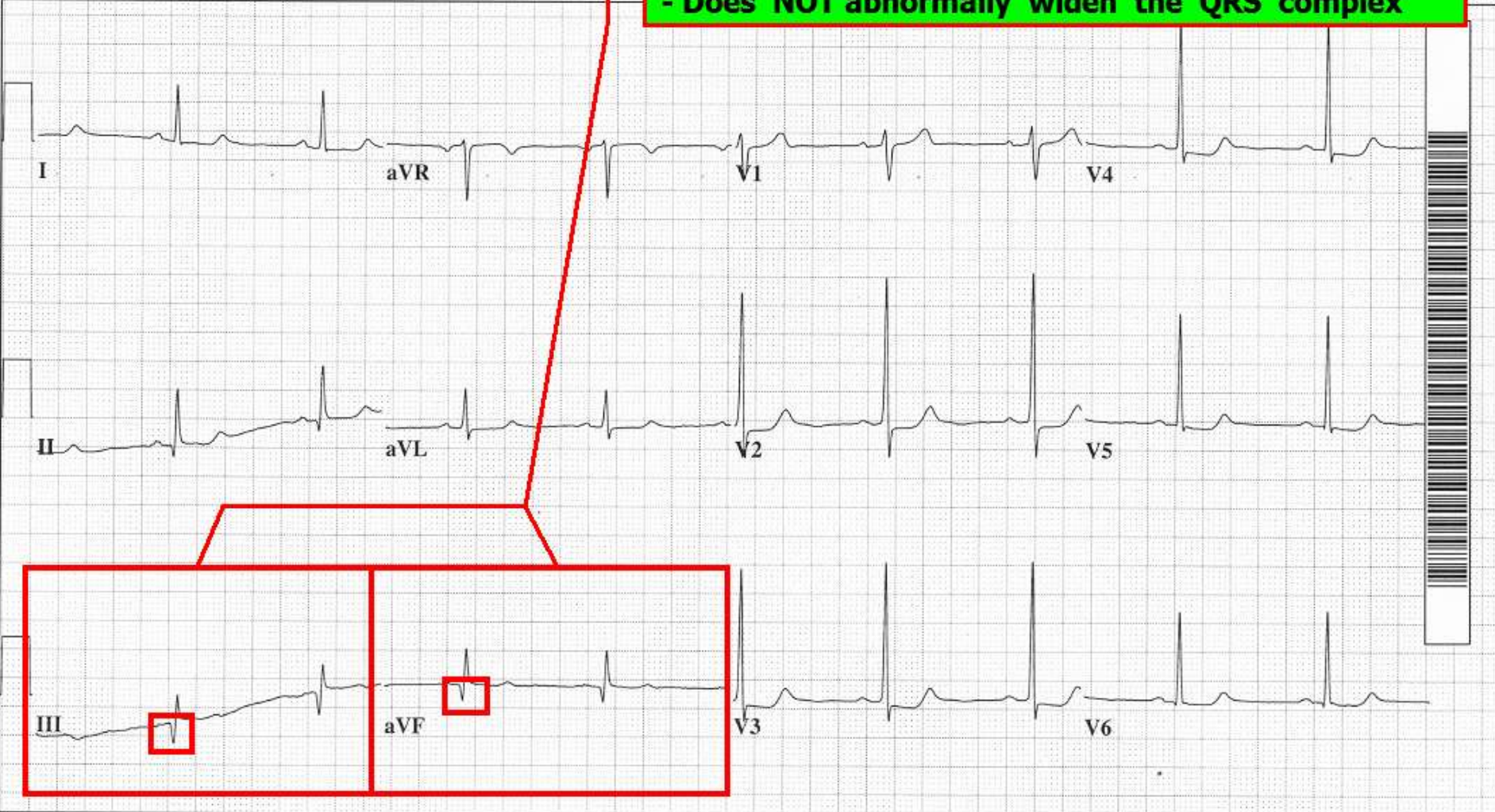
Vent. rate 58 BPM
PR interval 168 ms
QRS duration 84 ms
QT/QTc 424/416 ms
P-R-T axes 18 28 29

Sinus bradycardia
Inferior-posterior infarct (cited on or before 27-APR-1997)
~~Abnormal ECG~~
When compared with ECG of 30-APR-1997 13:39,
No significant change was found

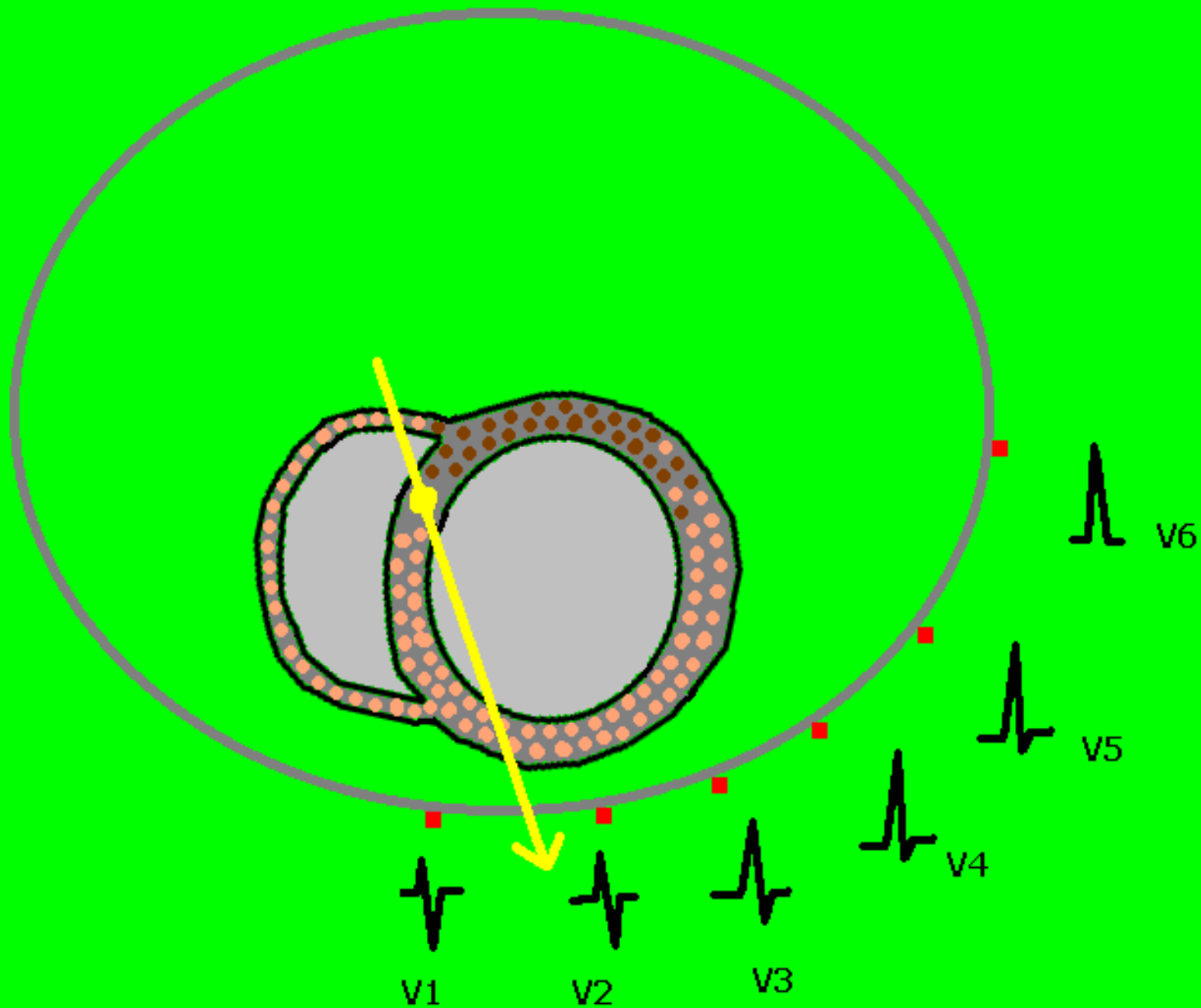
Technician ID: EKG CLASS #WR03602216

Med: Unknown

Old Posterior Wall MI
→ **Usually accompanied by OLD INFERIOR WALL MI**
- Does NOT abnormally widen the QRS complex



OLD POSTERIOR WALL M.I.



01-MAY-

04:14:17

51 yr
Male Caucasian
Room:540
Loc:5 Option:28

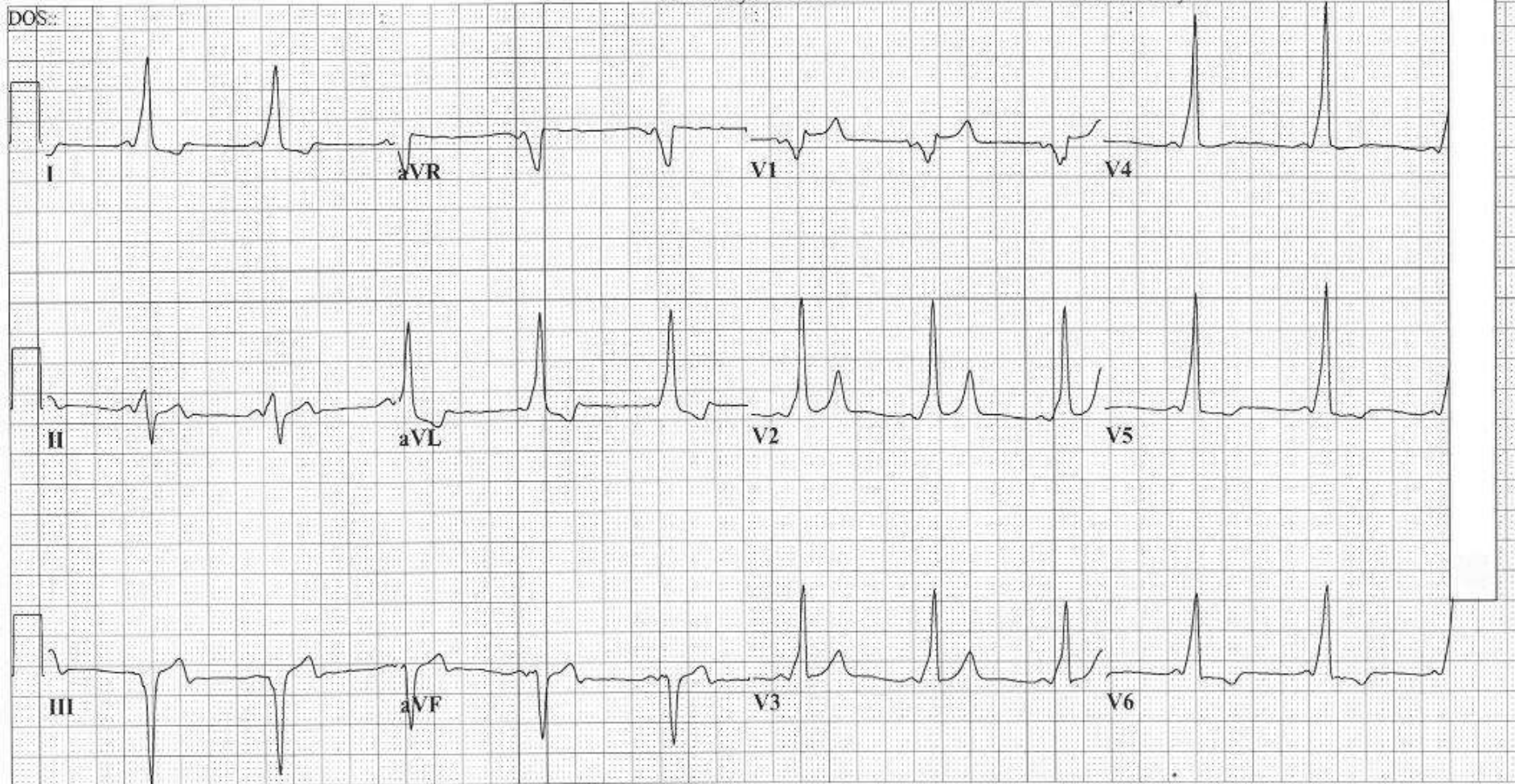
Vent. rate 64 BPM
PR interval 110 ms
QRS duration 146 ms
QT/QTc 418/431 ms
P-R-T axes 50 -36 119

**What is the cause of
EARLY TRANSITION
in this EKG ?**

Technician EKG CLASS #WR03696205

Referred by: _____

Confirmed By: _____



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 EARLY TRANSITION

.....SOME HELPFUL CLUES:

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

- ~~- QRS wider than 120ms~~
- ~~- Supraventricular rhythm (normal P : QRS relationship)~~
- ~~- PSR' 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

01-MAY-1999 04:14:17

ST. JOSEPH'S HOSPITAL-IN1464 ROUTINE RETRIEVAL

51 yr
Male Caucasian
Room:540
Loc:5 Option:28

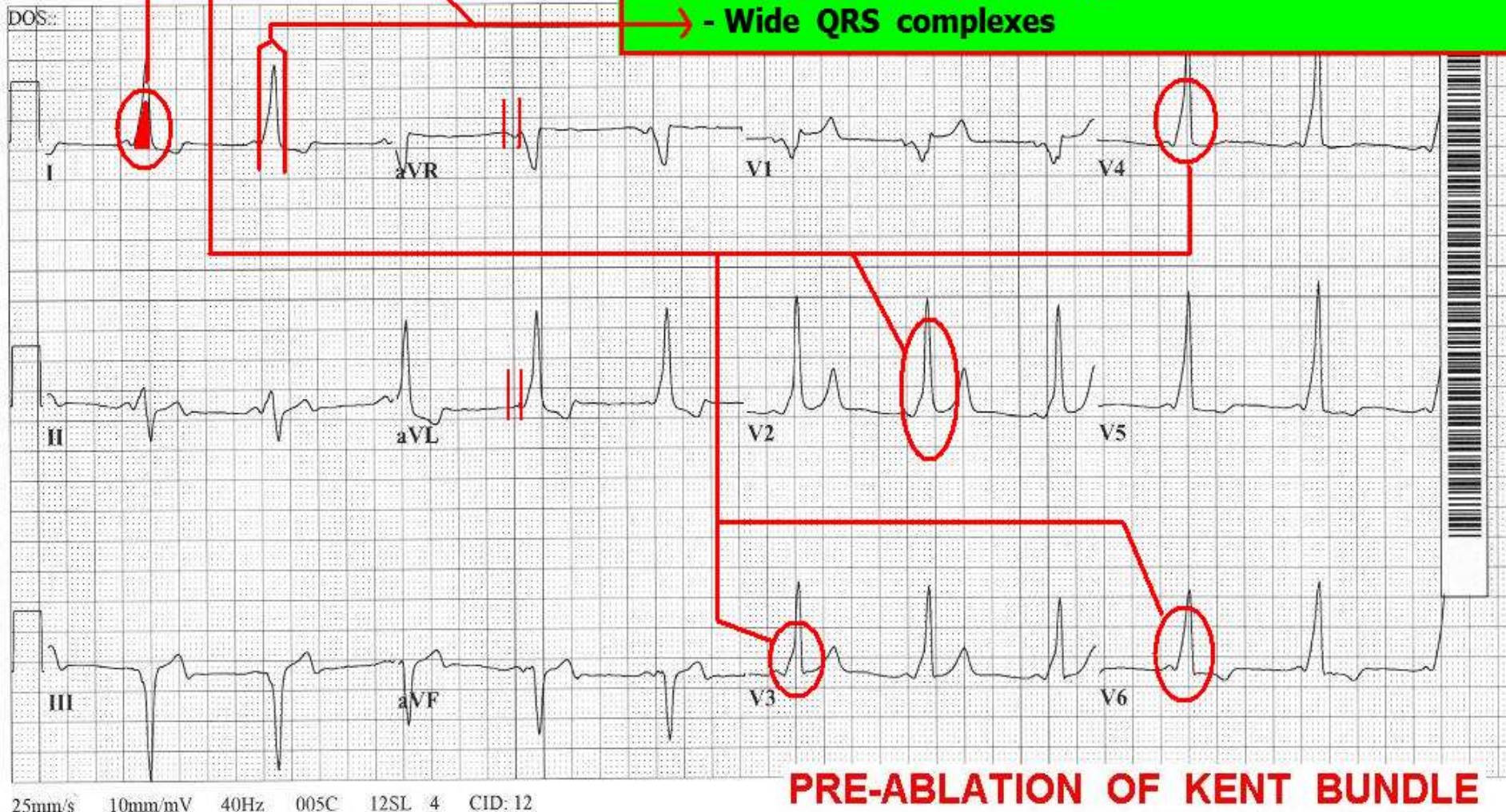
Vent rate	64	BPM
PR interval	110	ms
QRS duration	146	ms
QT/QTc	418/431	ms
P-R-T axes	50 -36 119	

Normal sinus rhythm
Wolff-Parkinson-White
Abnormal ECG
No previous ECGs available

Technician EKG CLASS #WR03696205

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

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

**PRE-ABLATION OF KENT BUNDLE**

04-MAY-1999 04:47:41

ST. JOSEPH'S HOSPITAL-IN1464 ROUTINE RETRIEVAL

51 yr
Male Caucasian
Room: 426
Loc: 5 Option: 28

Vent. rate 69 BPM
PR interval 184 ms
QRS duration 88 ms
QT/QTc 392/420 ms
P-R-T axes 60 69 -50

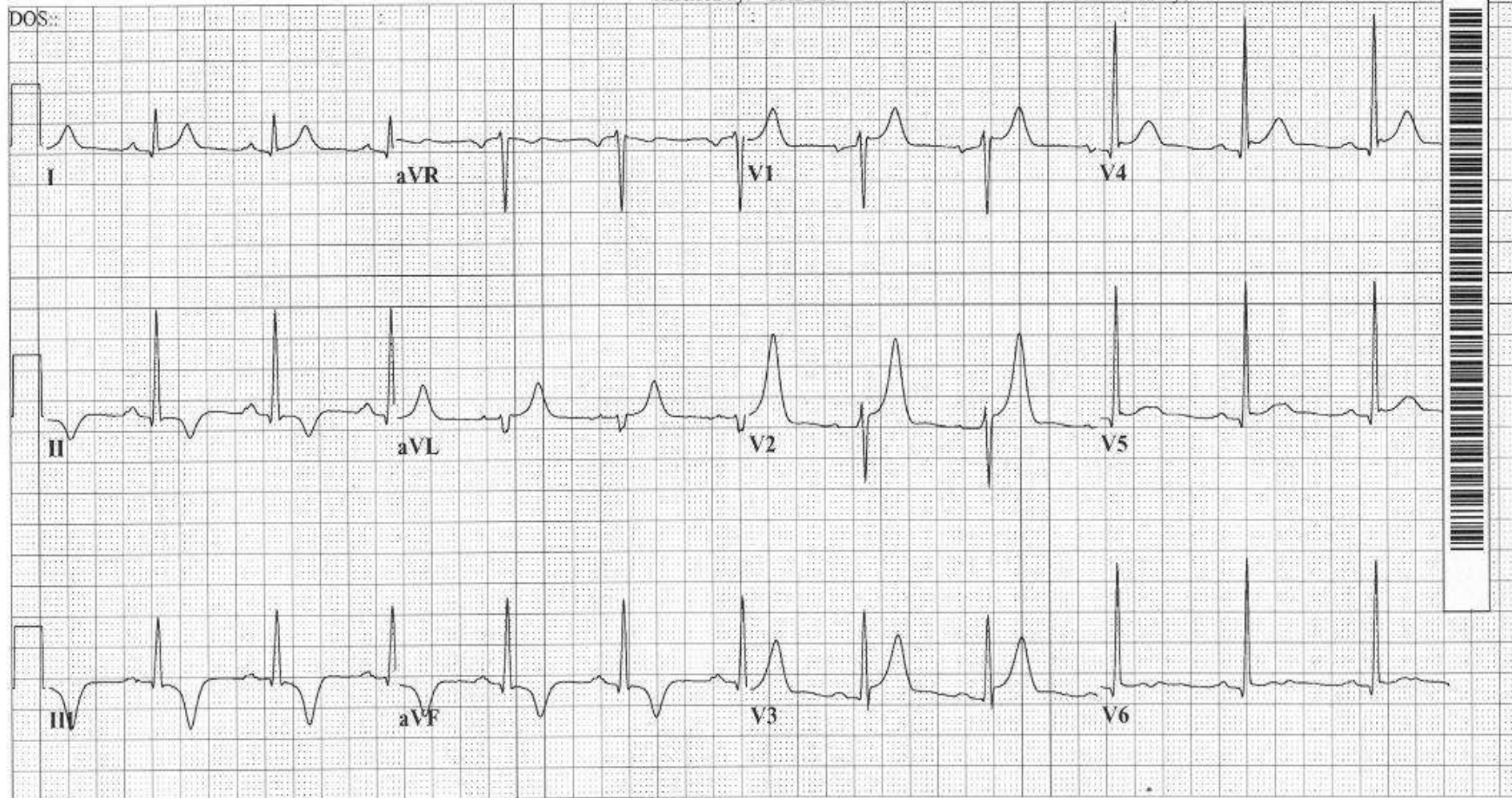
Normal sinus rhythm
Marked T wave abnormality, consider inferior ischemia
Abnormal ECG
When compared with ECG of 01-MAY-1999 21:36,
Wolff-Parkinson-White is no longer Present

Technician: EKG CLASS #WR03696205

POST-ABLATION OF KENT BUNDLE

Referred by: _____

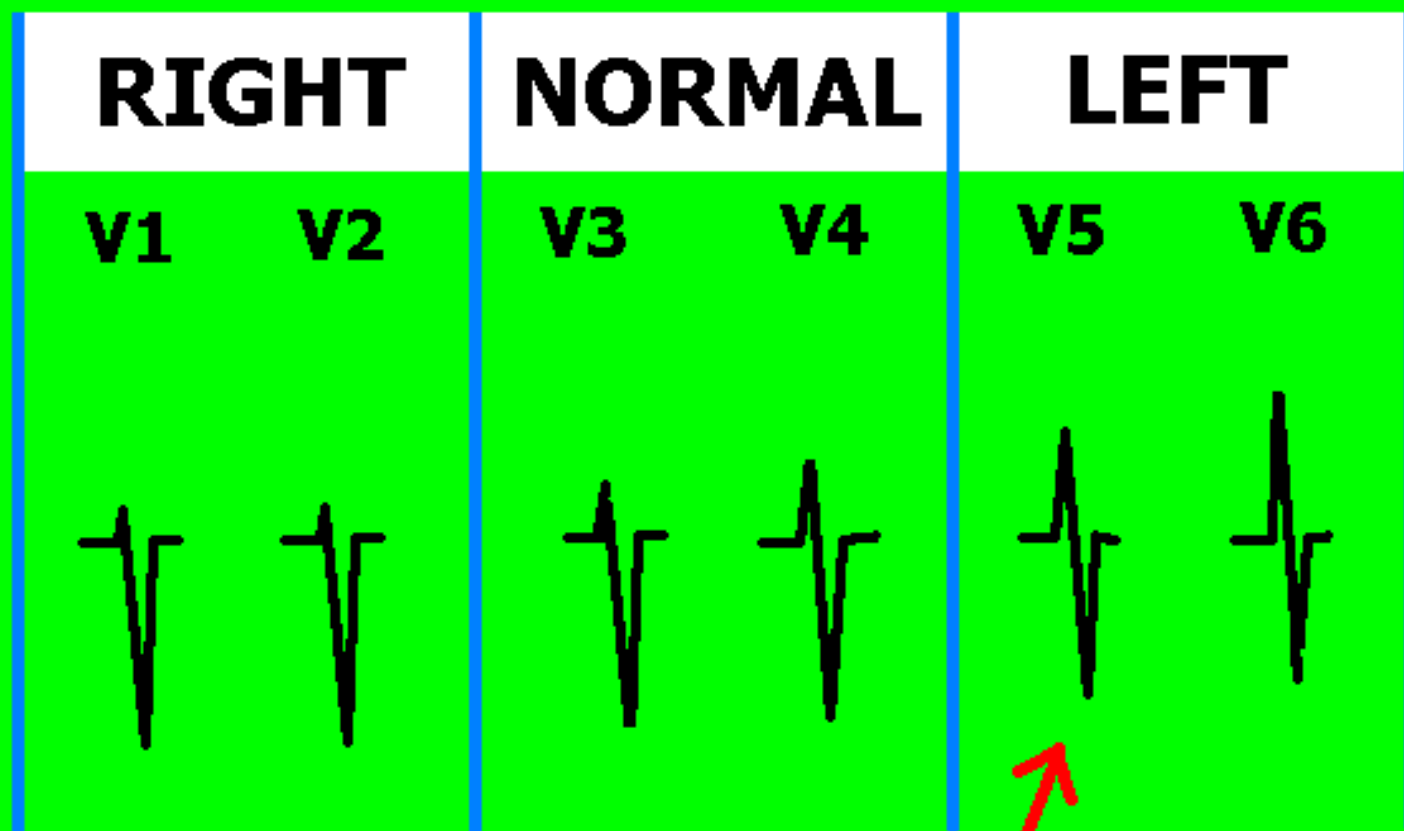
Confirmed By: _____



25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 12

EID:17 EDT: 09:08 04-MAY-1999 ORDER:

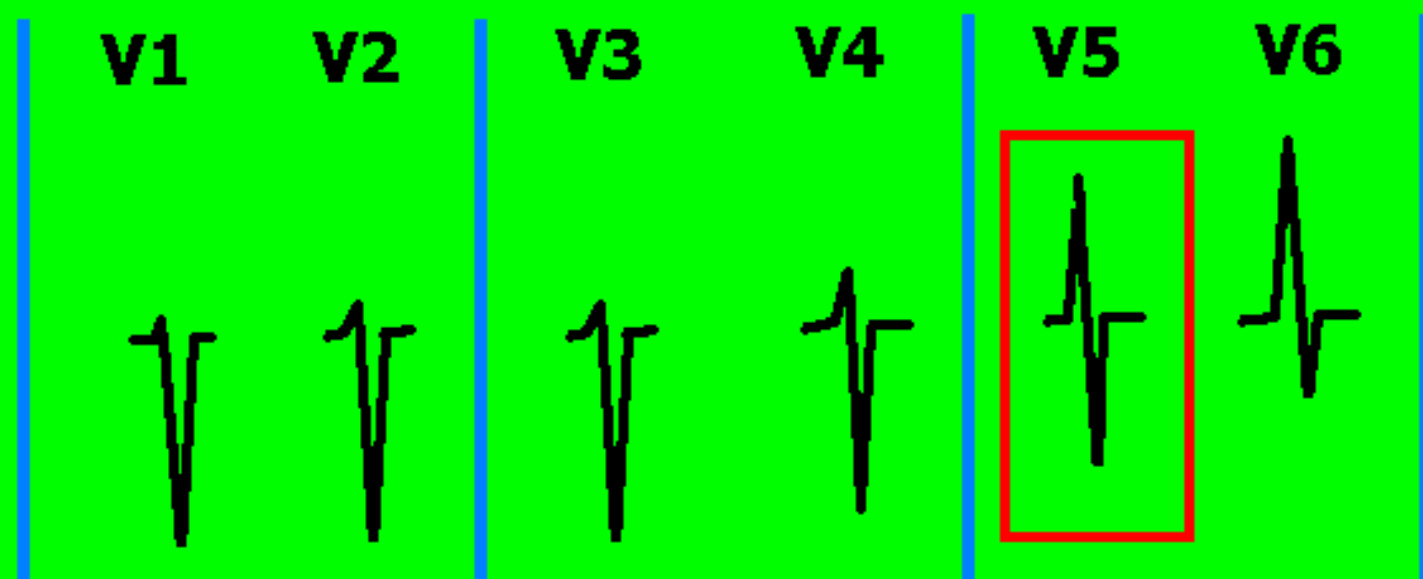
AXIS ROTATION



"LATE TRANSITION"

"SHIFTED TO THE LEFT"

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 Block (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)
- Wide QRS (> 120 ms)

28-MAR-1997 05:46:00

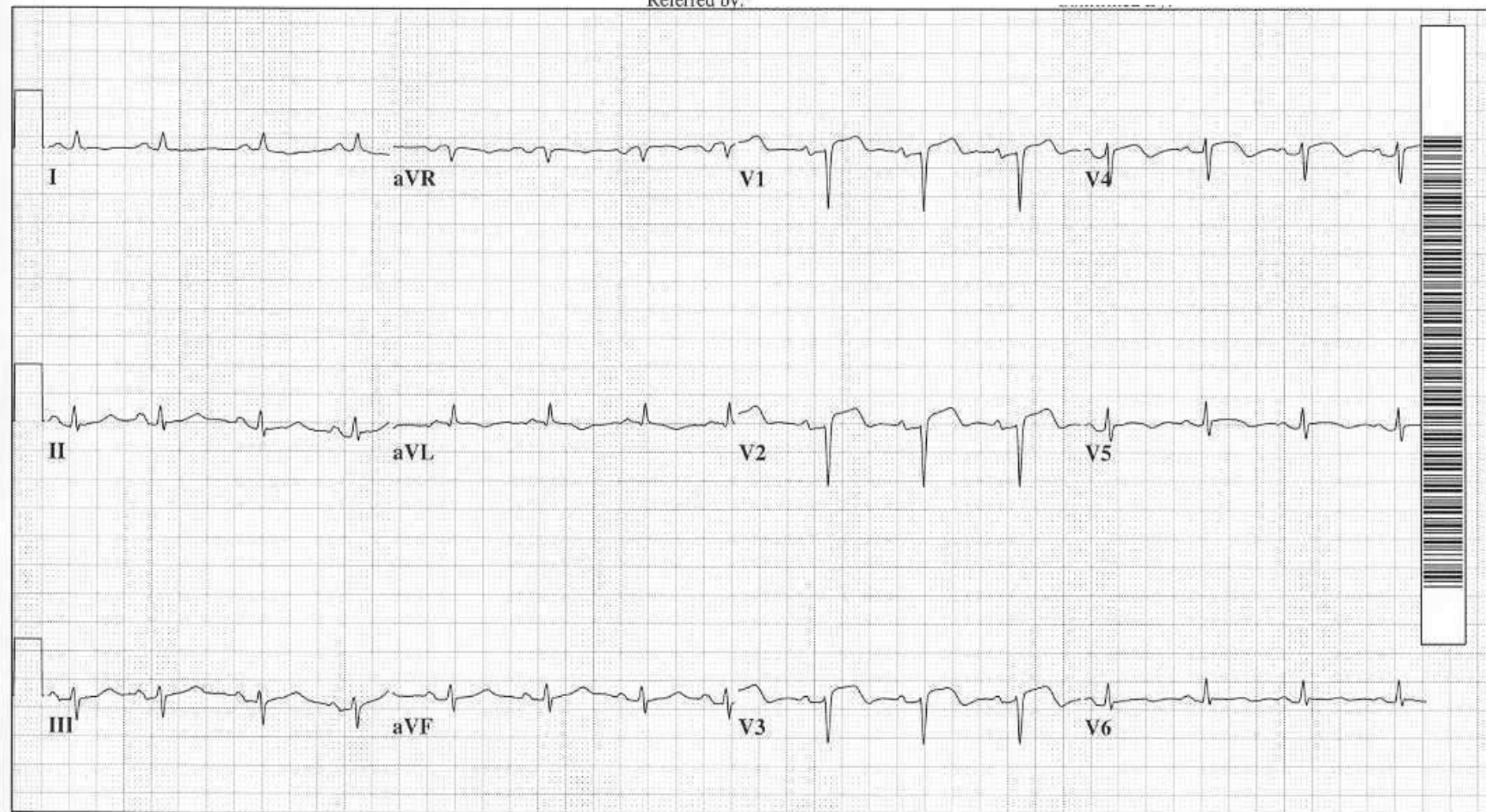
91 yr
Female Caucasian
Room:3
Loc:1 Option:1

Vent. rate 87 BPM
PR interval 156 ms
QRS duration * 80 ms
QT/QTc 332/399 ms
P-R-T axes 45 4 96

**What is the cause of
LATE TRANSITION in
this EKG ?**

Technician ID: EKG CLASS # WR03110848

Referred by:



25mm/s 10mm/mV 100Hz 005C 12SL 4 CID: 5

EID:16 EDT: 11:25 30-MAR-1997 ORDER:

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 Block (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)
- Wide QRS (> 120 ms)

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 Block (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)
- Wide QRS (> 120 ms)

91 yr
Female Caucasian
Room:3
Loc:1 Option:1

Vent. rate 87 BPM
PR interval 156 ms
QRS duration * 80 ms
QT/QTc 332/399 ms
P-R-T axes 45 4 96

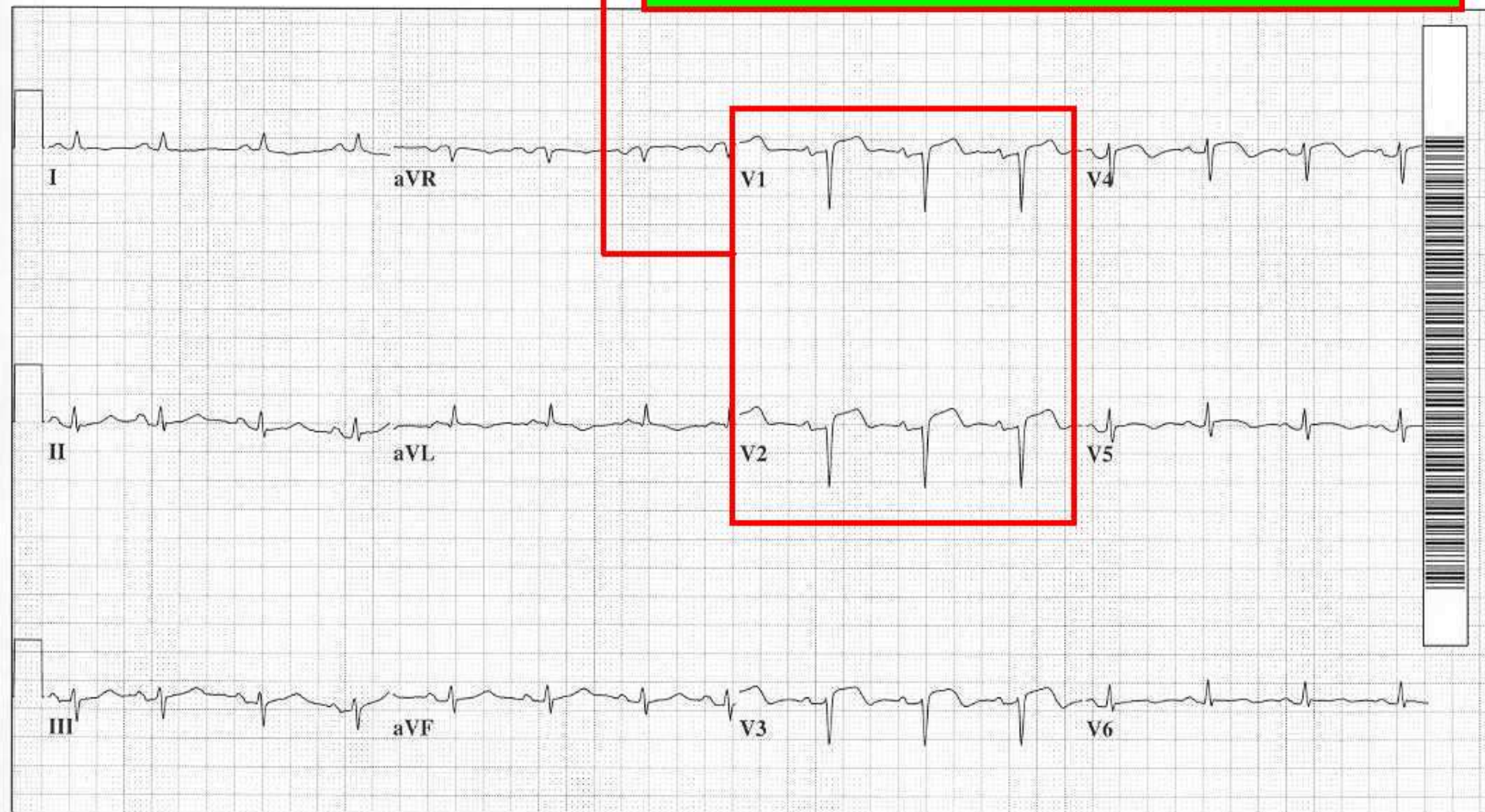
Normal sinus rhythm

Possible Anterior infarct (cited on or before 27-MAR-1997)
Abnormal ECG

Technician ID: EKG CLASS # WR03110848

Old Anterior MI

- Q waves in V1, V2, V3 and/or V4
- other causes of LATE TRANSITION ruled out



85 yr
Female Caucasian

Vent. rate 55 BPM
PR interval 152 ms
QRS duration 76 ms
QT/QTc 432/413 ms
P-R-T axes 40 14 34

Sinus bradycardia with occasional Premature supraventricular complexes
Otherwise normal ECG

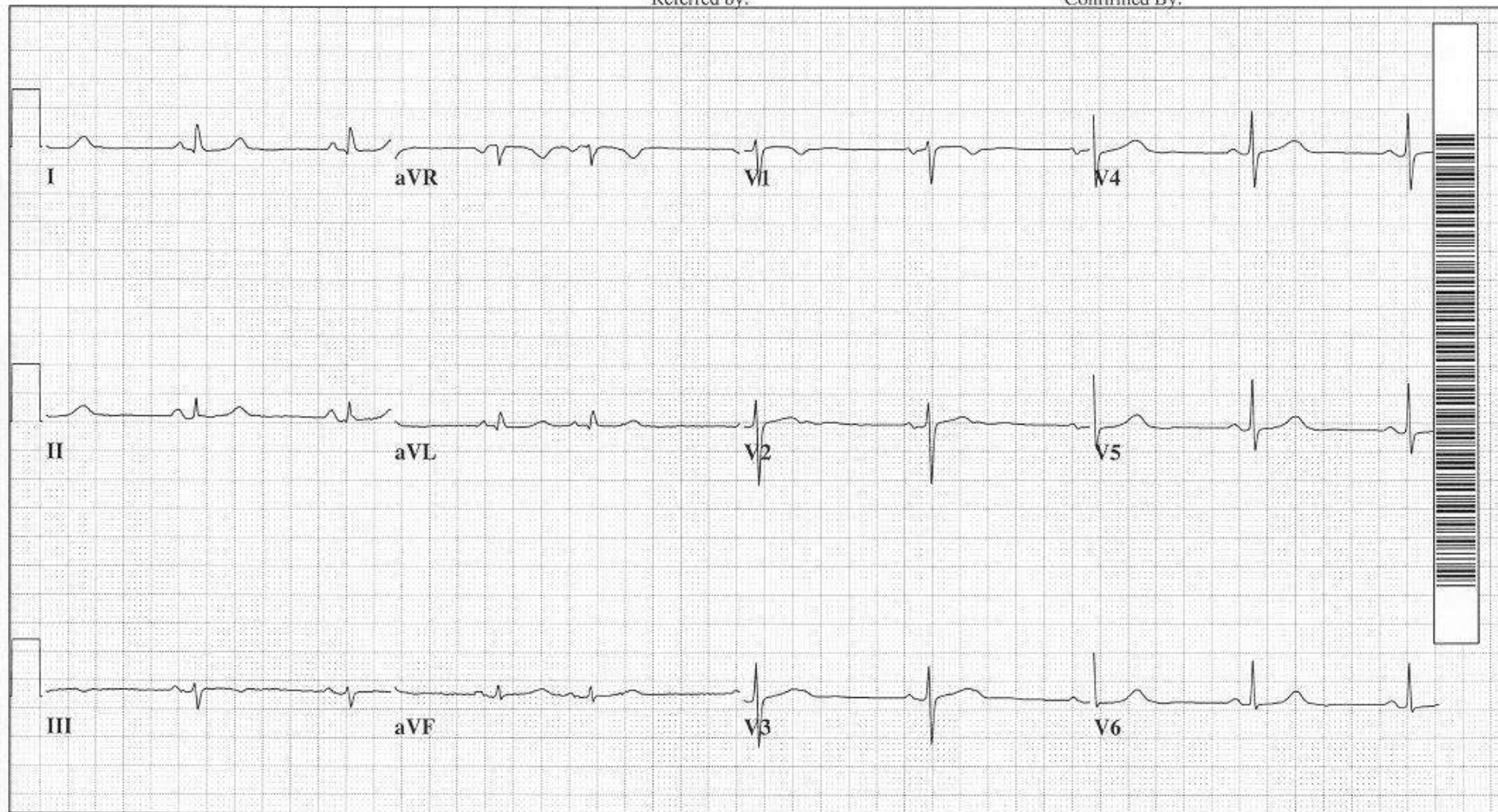
Room: 715A
Loc: 6 Option: 19

EKG CLASS # WR03110848

PRE-INFARCTION EKG

Referred by:

Confirmed By:



91 yr
Female Caucasian
Room: ER
Loc: 3 Option: 17

Vent. rate 100 BPM
PR interval 166 ms
QRS duration 80 ms
QT/QTc 360/464 ms
P-R-T axes 52 -38 70

Normal sinus rhythm with frequent, and consecutive Premature ventricular and fusion complexes

Left atrial enlargement

Left axis deviation

Septal infarct, possibly acute

Anterolateral injury pattern

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

Abnormal ECG

When compared with ECG of 27-MAR-1991 13:29,

Referred by:

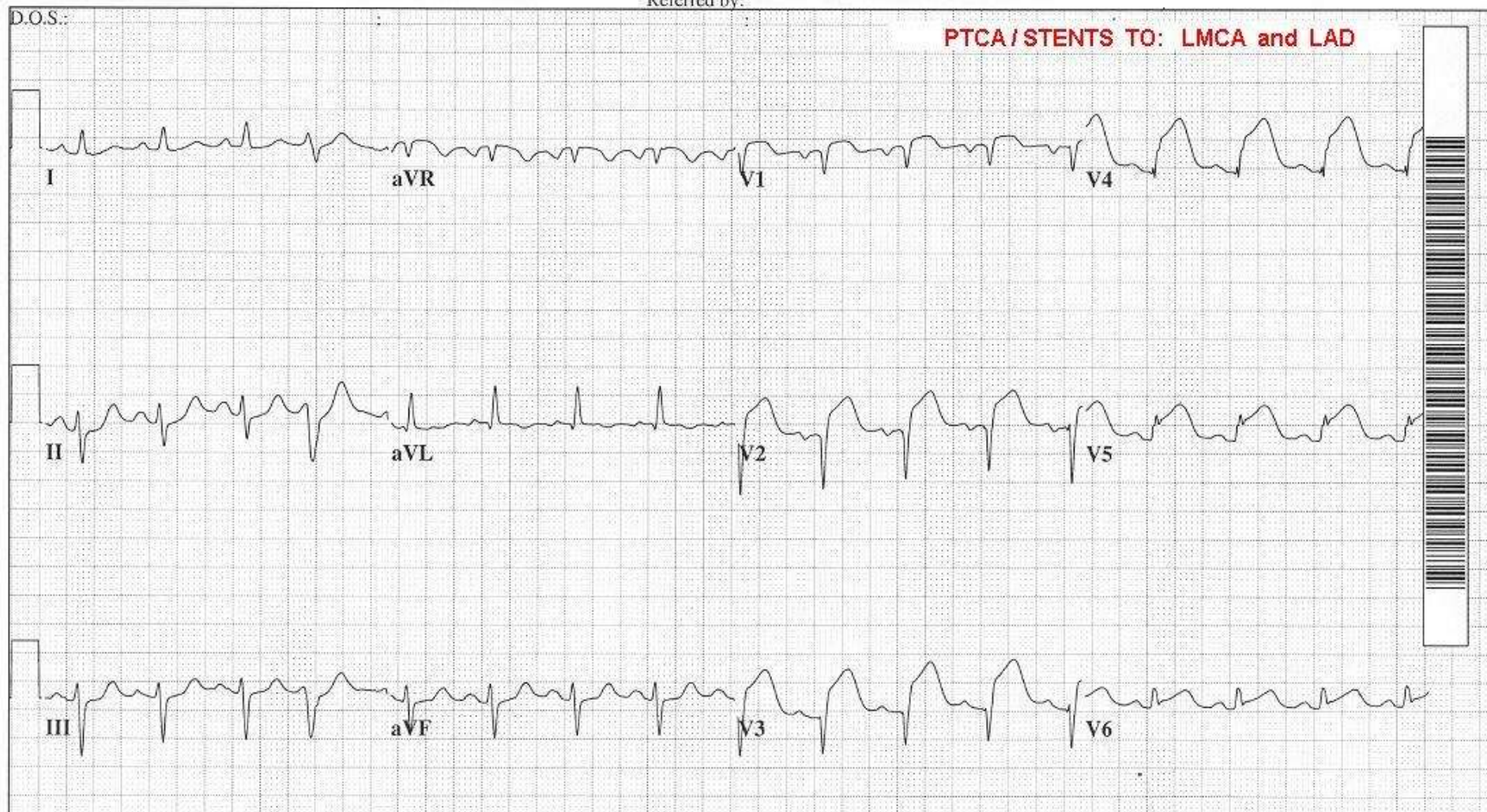
SUDDEN ONSET CHEST PAIN
-WAITED "SEVERAL HOURS"
BEFORE SEEKING HELP
-ER - DIRECTLY TO CATH LAB

CPK: 2,471

CK/MB: 483

CK INDEX: 14

Technician: EKG CLASS# WR03110848



91 yr
Female Caucasian
Room:3
Loc:1 Option:1

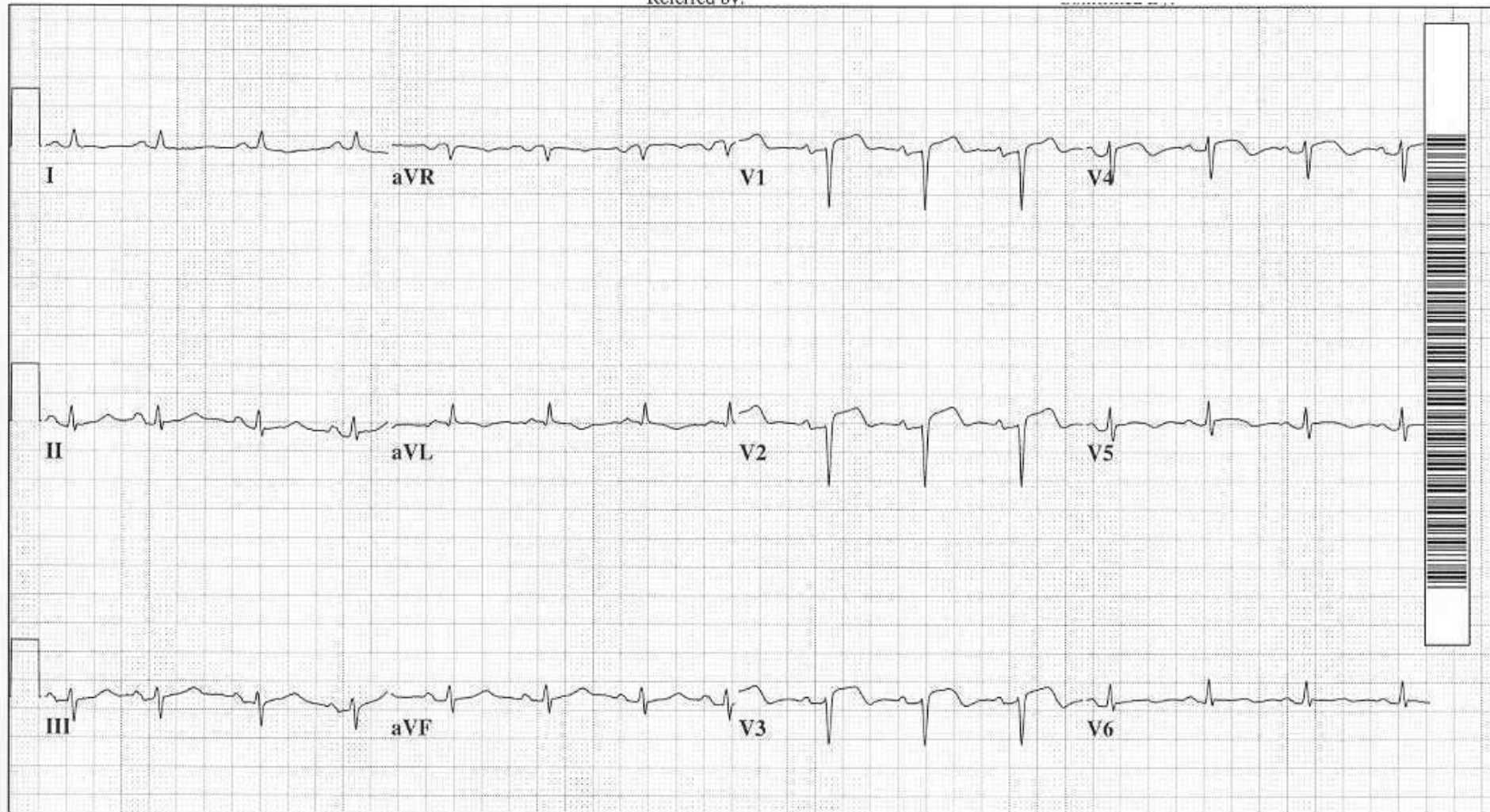
Vent. rate 87 BPM
PR interval 156 ms
QRS duration * 80 ms
QT/QTc 332/399 ms
P-R-T axes 45 4 96

Normal sinus rhythm
Possible Anterior infarct (cited on or before 27-MAR-1997)
Abnormal ECG
When compared with ECG of 27-MAR-1997 16:26 (UNCONFIRMED),
QRS duration has decreased
Questionable change in initial forces of Anteroseptal leads
Non-specific change in ST segment in Lateral leads
QT has shortened

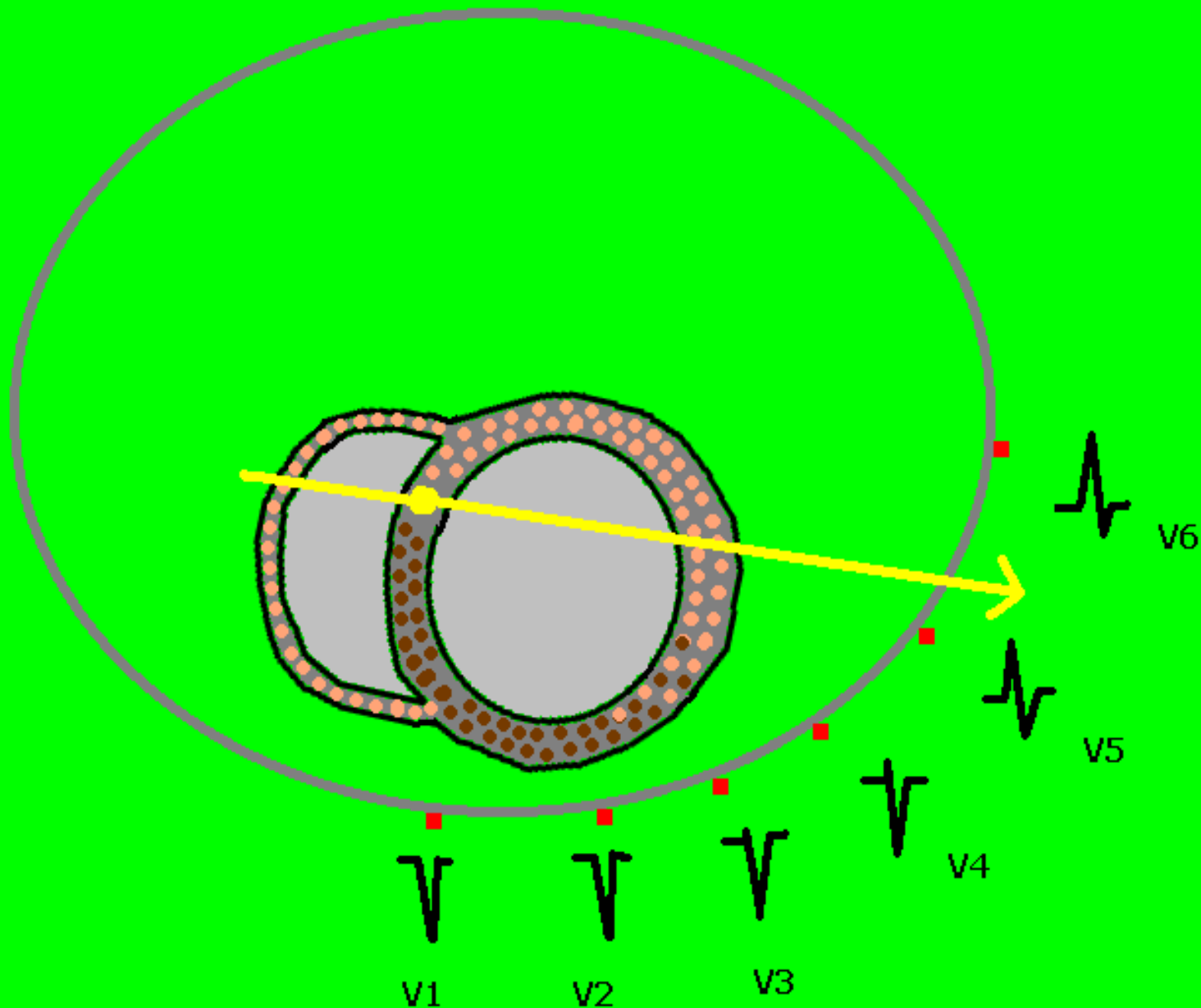
Technician ID: EKG CLASS # WR03110848

EKG POST - INFARCTION

Referred by:



OLD ANTERIOR-SEPTAL WALL M.I.



COMMON CAUSES OF LATE TRANSITION

.....SOME HELPFUL CLUES:



**When you have an EKG with
LATE TRANSITION, which has
NO OBVIOUS CAUSE . . .**

Supect OLD ANTERIOR MI !

- OBTAIN A THOROUGH PATIENT HISTORY**
- OBTAIN COPIES OF OLD EKGs,
IF AVAILABLE**

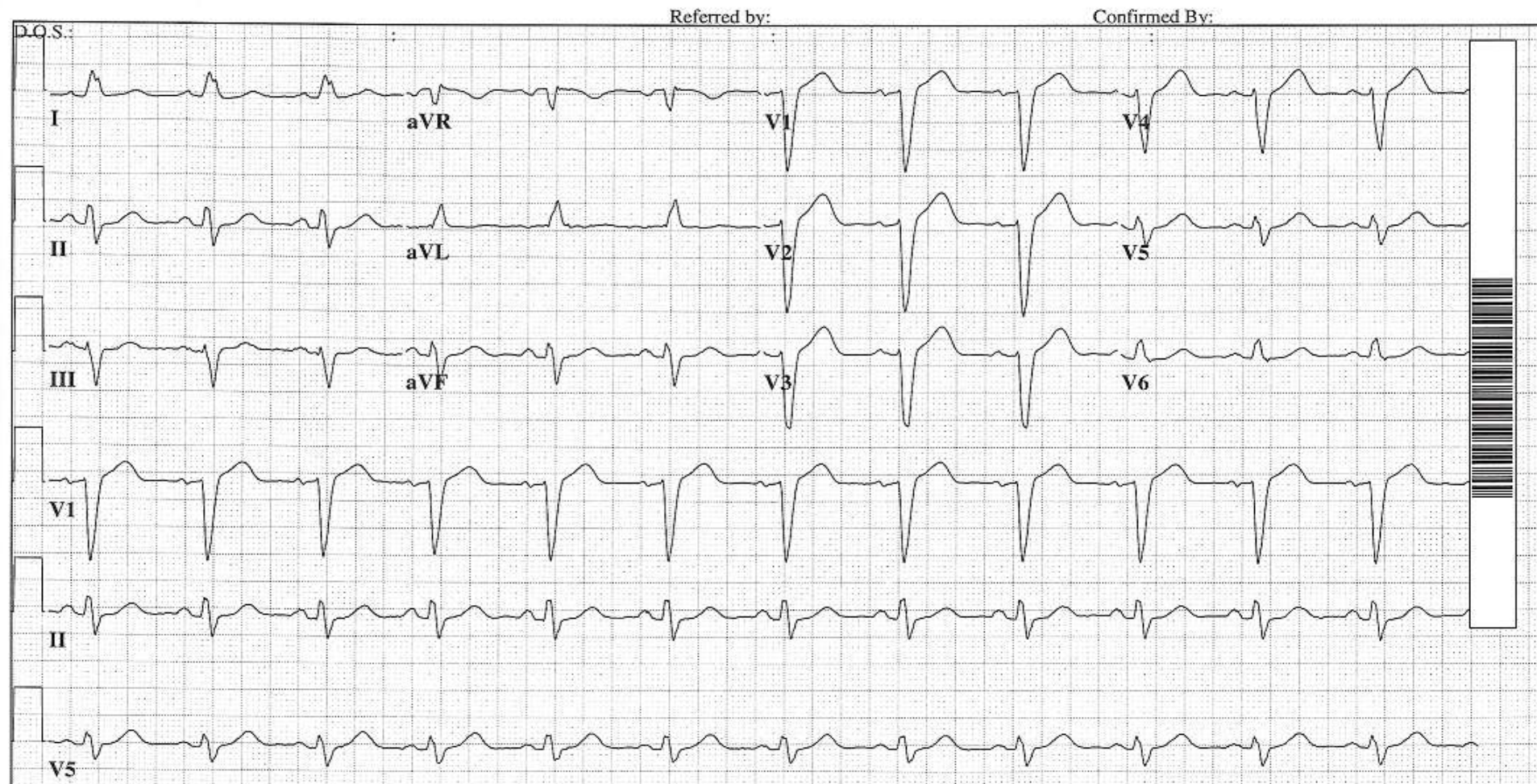
11:36:49

74 yr
Female Caucasian

Vent. rate	73	BPM
PR interval	160	ms
QRS duration	134	ms
QT/QTc	450/495	ms
P-R-T axes	67 -33	62

Loc:7 Option:35

**What is the cause of LATE
TRANSITION in this EKG ?**



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 Block (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)
- Wide QRS (> 120 ms)

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 Block (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)
- Wide QRS (> 120 ms)

11:36:49

74 yr
Female Caucasian

Vent. rate	73	BPM
PR interval	160	ms
QRS duration	134	ms
QT/QTc	438/435	ms
P-R-T axes	67 -33 62	

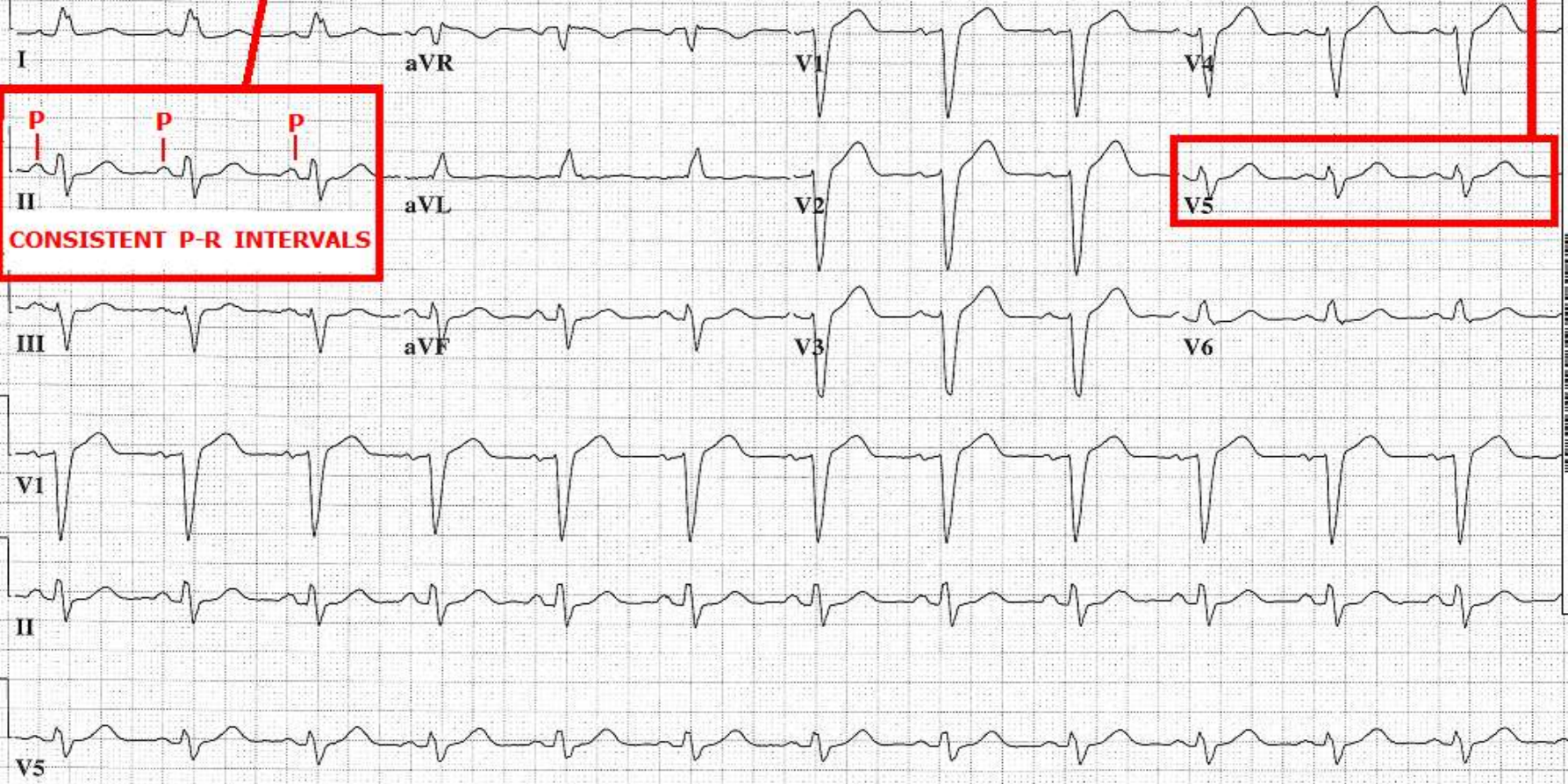
Normal sinus rhythm
Left axis deviation
Left bundle branch block

Left Bundle Branch Block (LBBB)

- Supraventricular Rhythm
- QRS wider than 120 ms (.12 sec)
- RsR' or RR' ("notching") in V5 and/or V6

P P

P.Q.S.:



53 yr
Male Black
Room:ER
Loc:3 Option:23

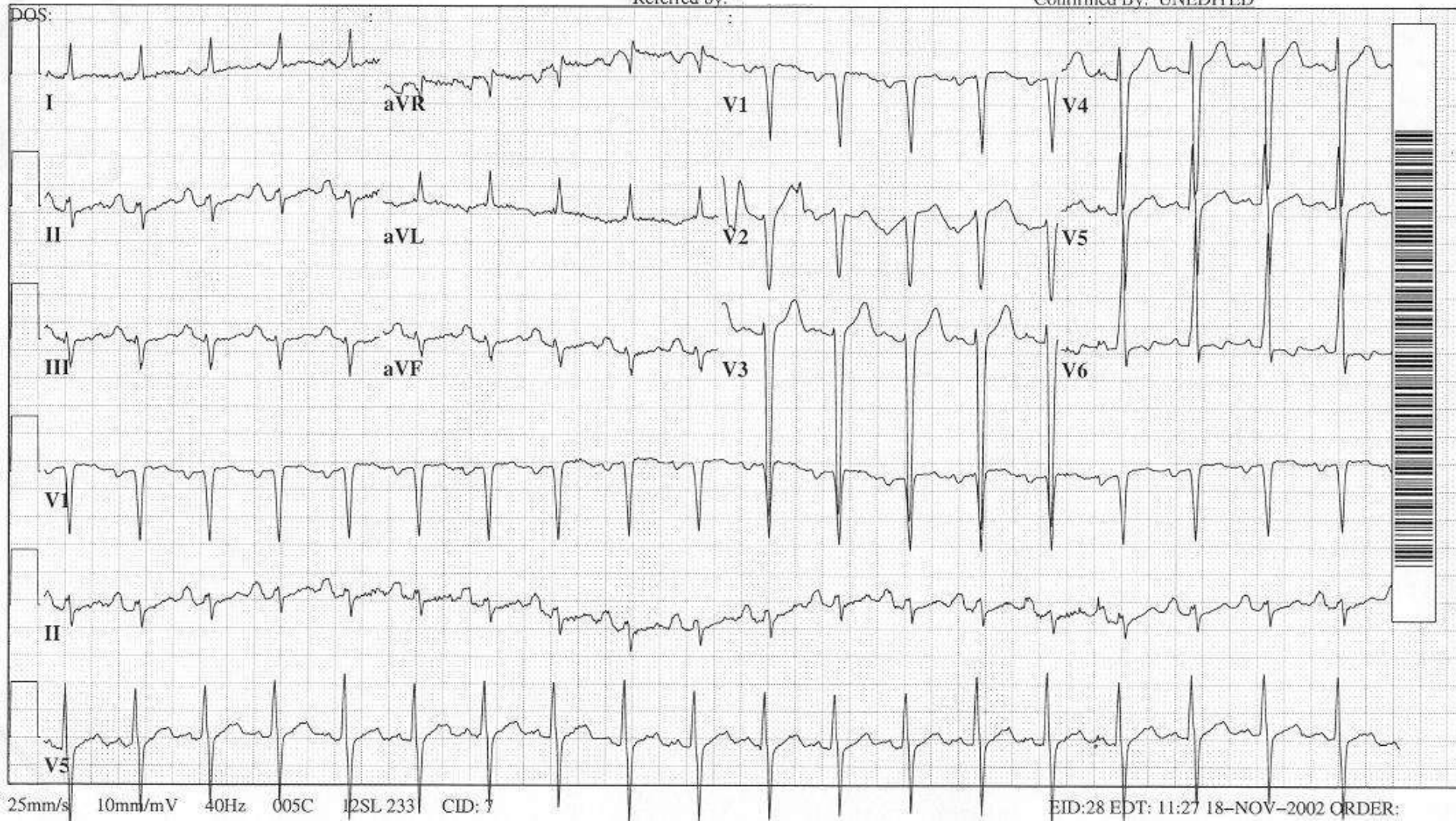
Vent. rate 115 BPM
PR interval 160 ms
QRS duration 92 ms
QT/QTc 316/437 ms
P-R-T axes 76 -39 59

**What is the cause of
LATE TRANSITION in
this EKG ?**

EKG CLASS #WR03896717

Referred by:

Confirmed By: UNEDITED



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 Block (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)
- Wide QRS (> 120 ms)

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 Block (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)~~
- ~~- Wide QRS (> 120 ms)~~

53 yr
Male Black
Room:ER
Loc:3 Option:23

Vent. rate 115 BPM
PR interval 160 ms
QRS duration 92 ms
QT/QTc 316/437 ms
P-R-T axes 76 -39 59

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

Sinus tachycardia

Possible Left atrial enlargement

Left axis deviation

Left ventricular hypertrophy

Abnormal ECG

No previous ECGs available

S wave V1 = 14 mm

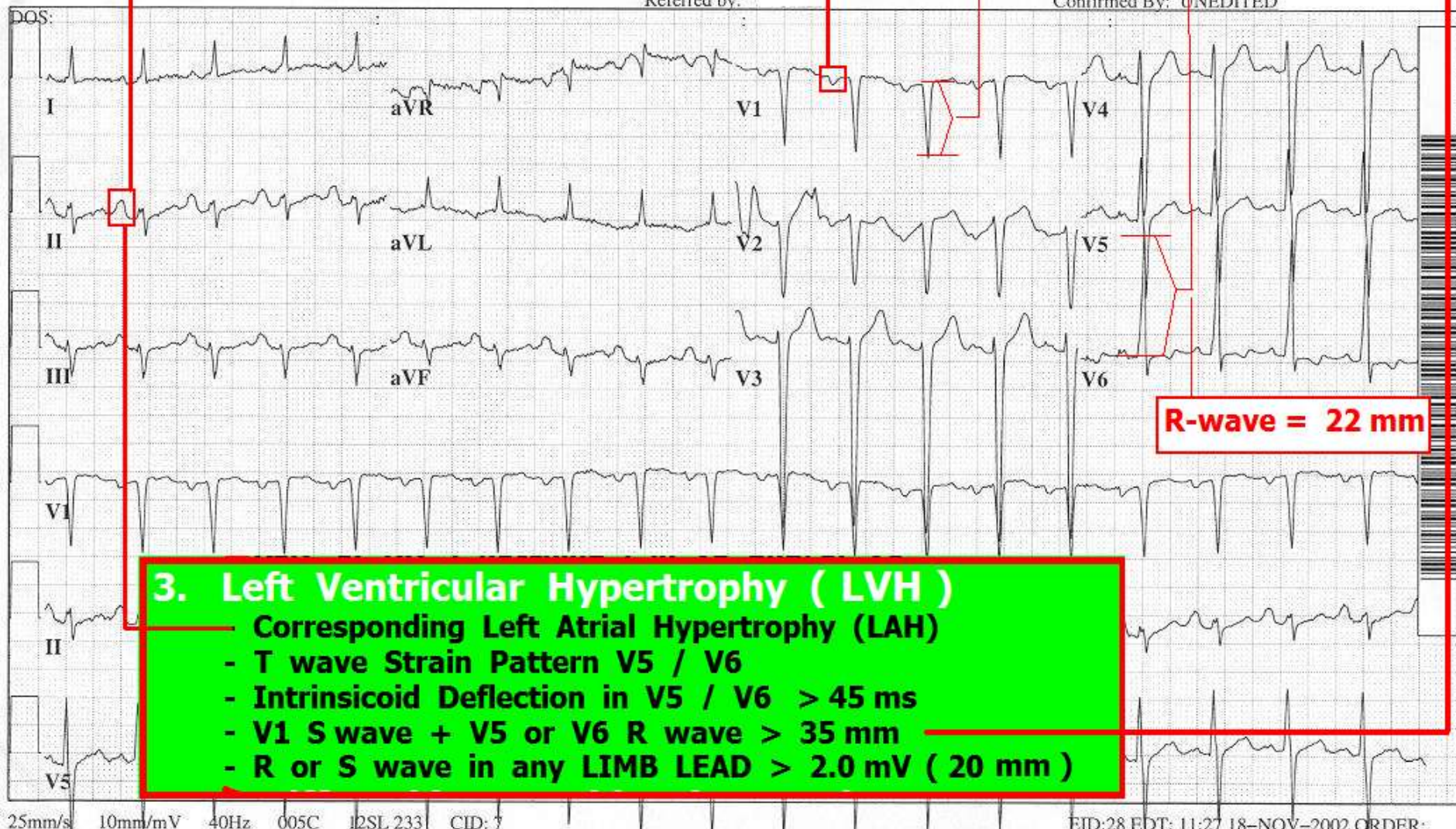
R wave V5 = 22 mm

TOTAL = 36 mm
= LVH

EKG CLASS #WR03896717

Referred by:

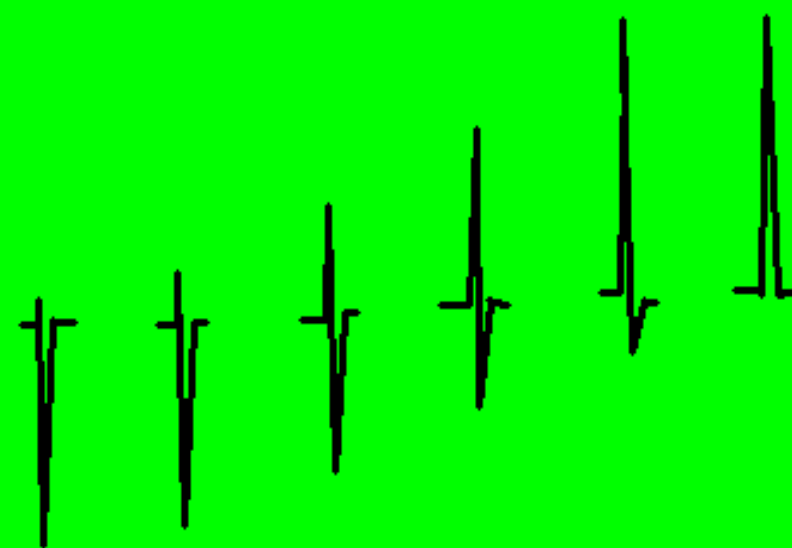
Confirmed By: UNEDITED



**"EXAGGERATED" QRS SIZE in V leads
FROM LEFT VENTRICULAR HYPERTROPHY**



NORMAL



LVH

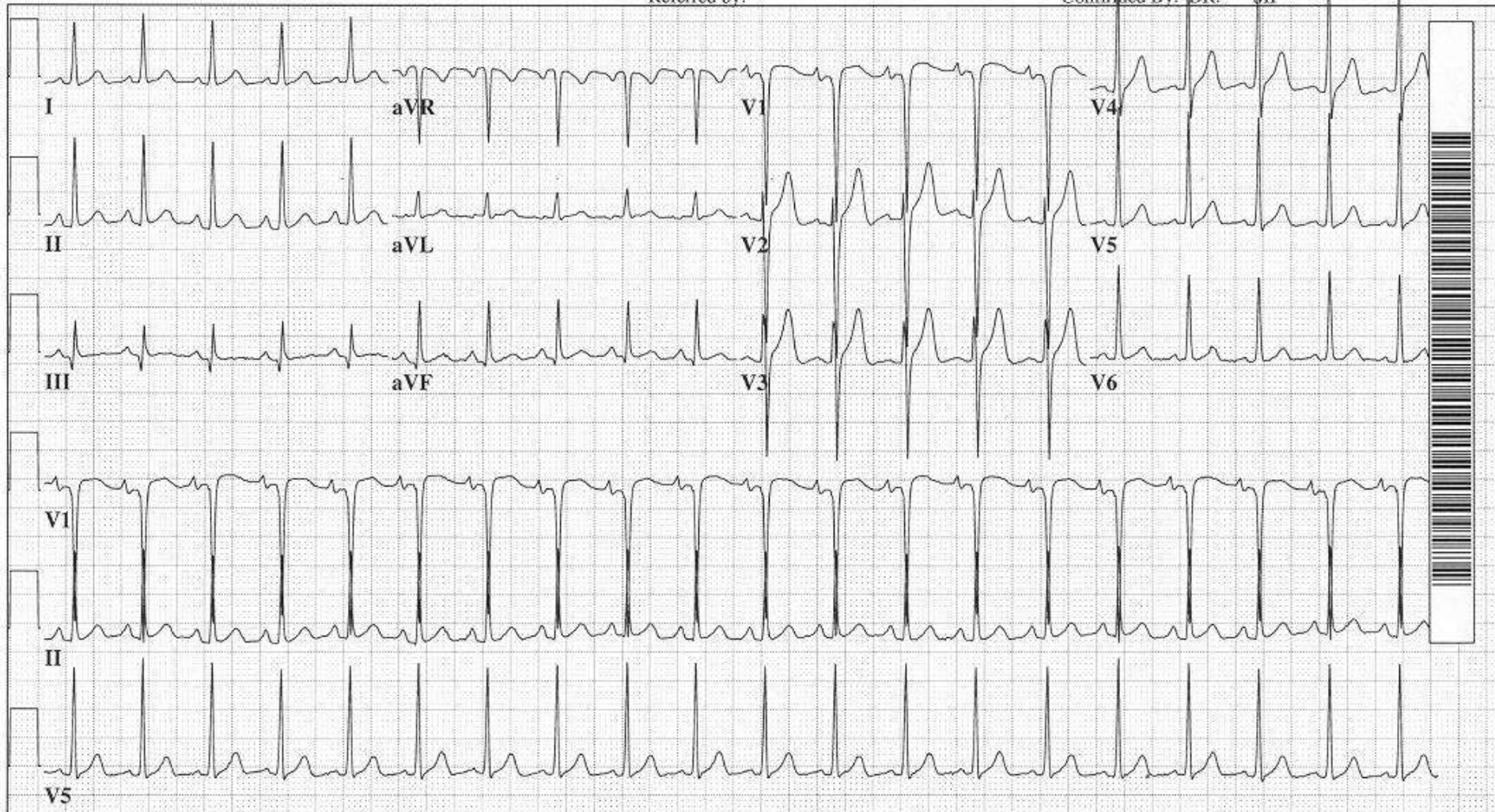
Vent. rate	119	BPM
PR interval	126	ms
QRS duration	78	ms
QT/QTc	282/397	ms
P-R-T axes	68 46	41

Sinus tachycardia
Minimal voltage criteria for LVH, may be normal variant
Borderline ECG

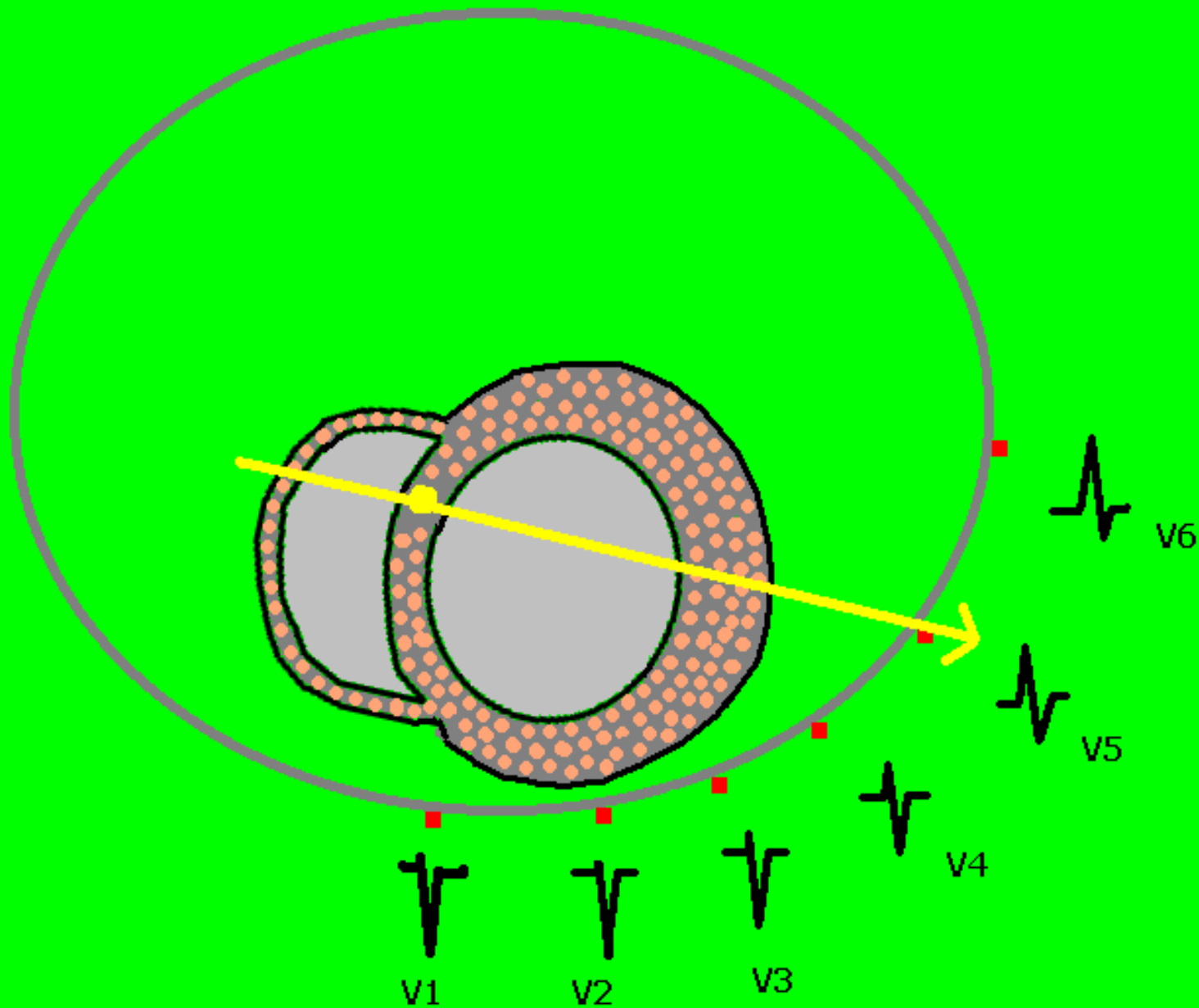
EKG CLASS #WR03446043

Referred by: _____

Confirmed By: DR. MI



LEFT VENTRICULAR HYPERTROPHY

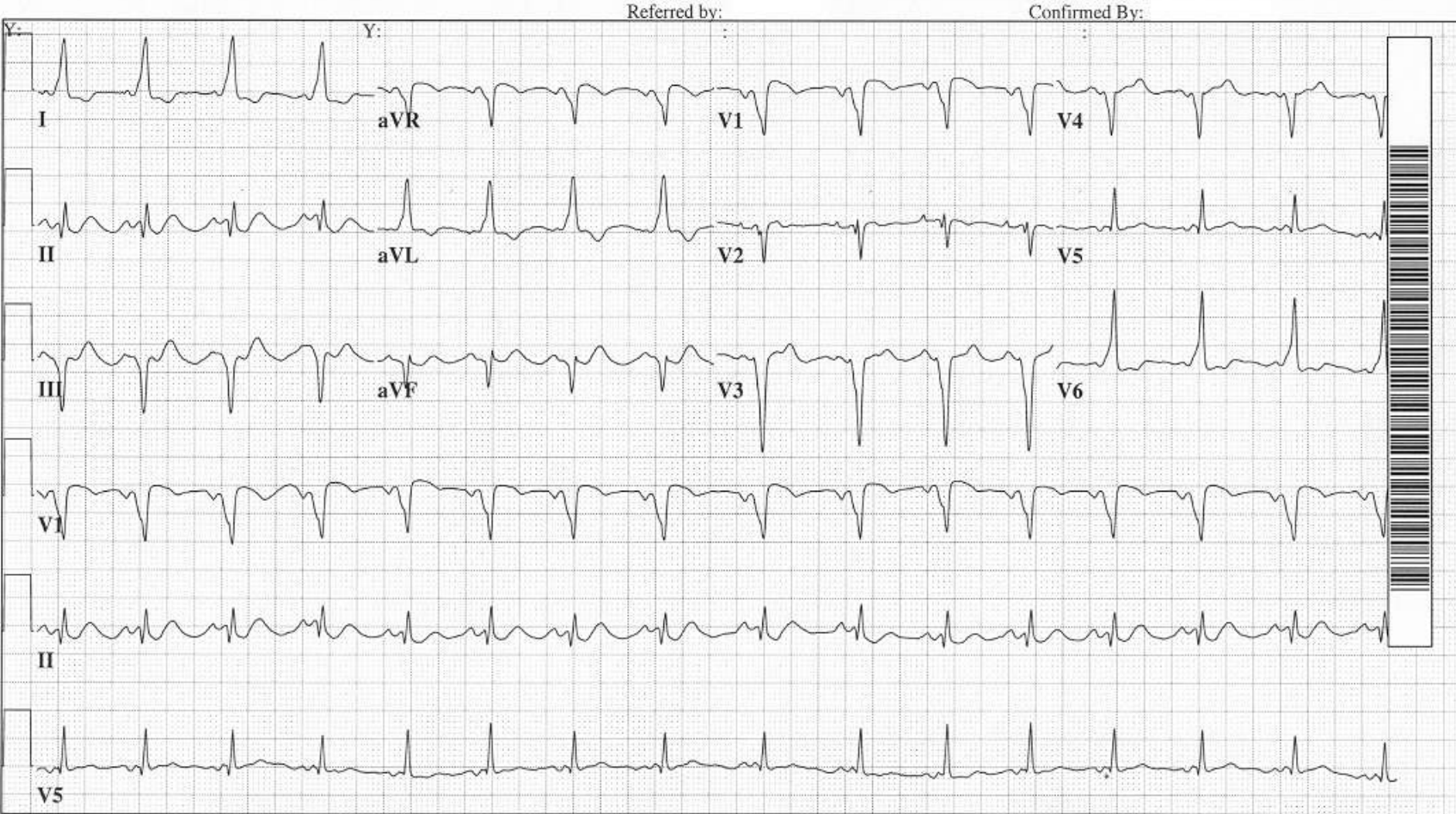


16 yr
Female Caucasian
Room: REC
Loc: 20 Option: 50

Vent. rate 92 BPM
PR interval 112 ms
QRS duration 118 ms
QT/QTc 356/440 ms
P-R-T axes 59 -22 107

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

**what is the cause of
LATE TRANSITION on
this EKG ?**



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 Block (LBBB)

- Supraventricular Rhythm
- QRS wider than 120 ms (.12 sec)
- RsR' or RR' ("notching") in V5 and/or V6

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- Corresponding Left Atrial Hypertrophy (LAH)
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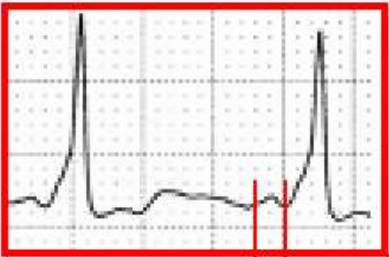
16 yr
Female Caucasian
Room: REC
Loc: 20 Option: 50

Vent. rate 92 BPM
PR interval 112 ms
QRS duration 118 ms
QT/QTc 356/440 ms
P-R-T axes 59 -22 107

Normal sinus rhythm with sinus arrhythmia
Wolff-Parkinson-White
Abnormal ECG
No previous ECGs available

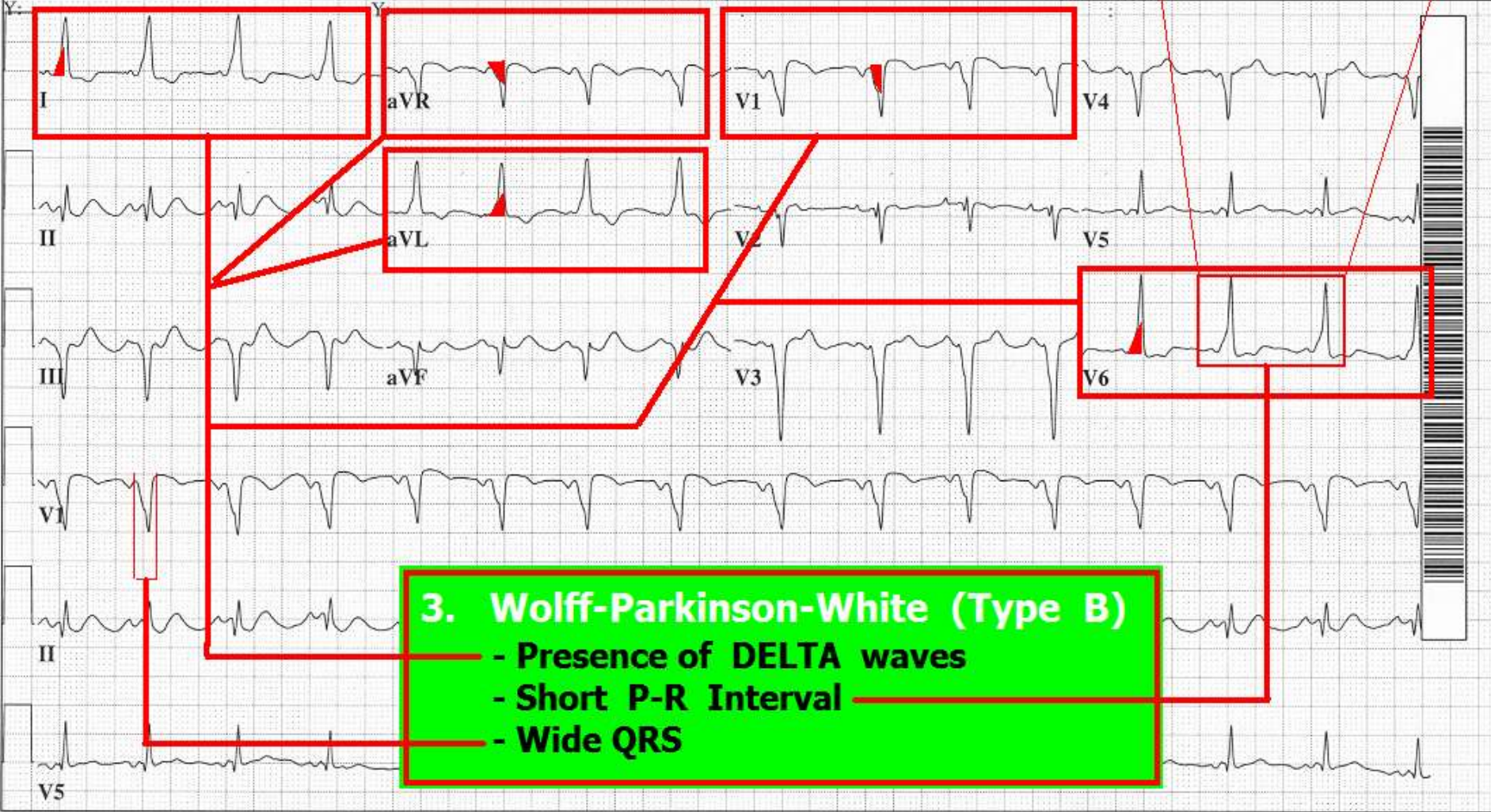
History: Unknown
Technician: DP
Test ind: EKG
EKG CLASS #WR030100
60783

P-R = .08



Referred by:

Confirmed By:



3. Wolff-Parkinson-White (Type B)

- Presence of DELTA waves
- Short P-R Interval
- Wide QRS



MOM and DAD at Lee's Diner, York, PA 2006