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

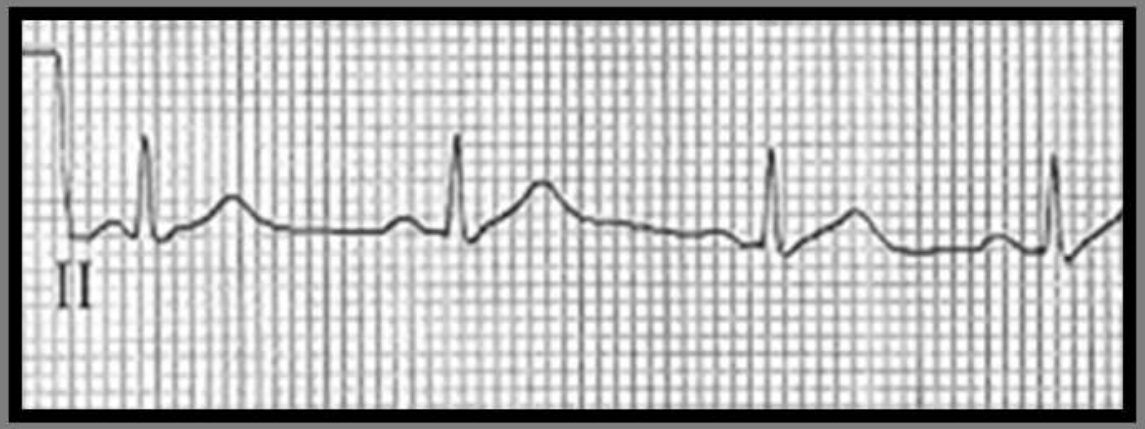
The Lifesaving 12 Lead ECG: Part 1

Wayne W Ruppert, CVT, CCCC, NREMT-P
Regional Director of Clinical Outreach &
Cardiovascular Accredited Programs:
Chest Pain Center, Heart Failure

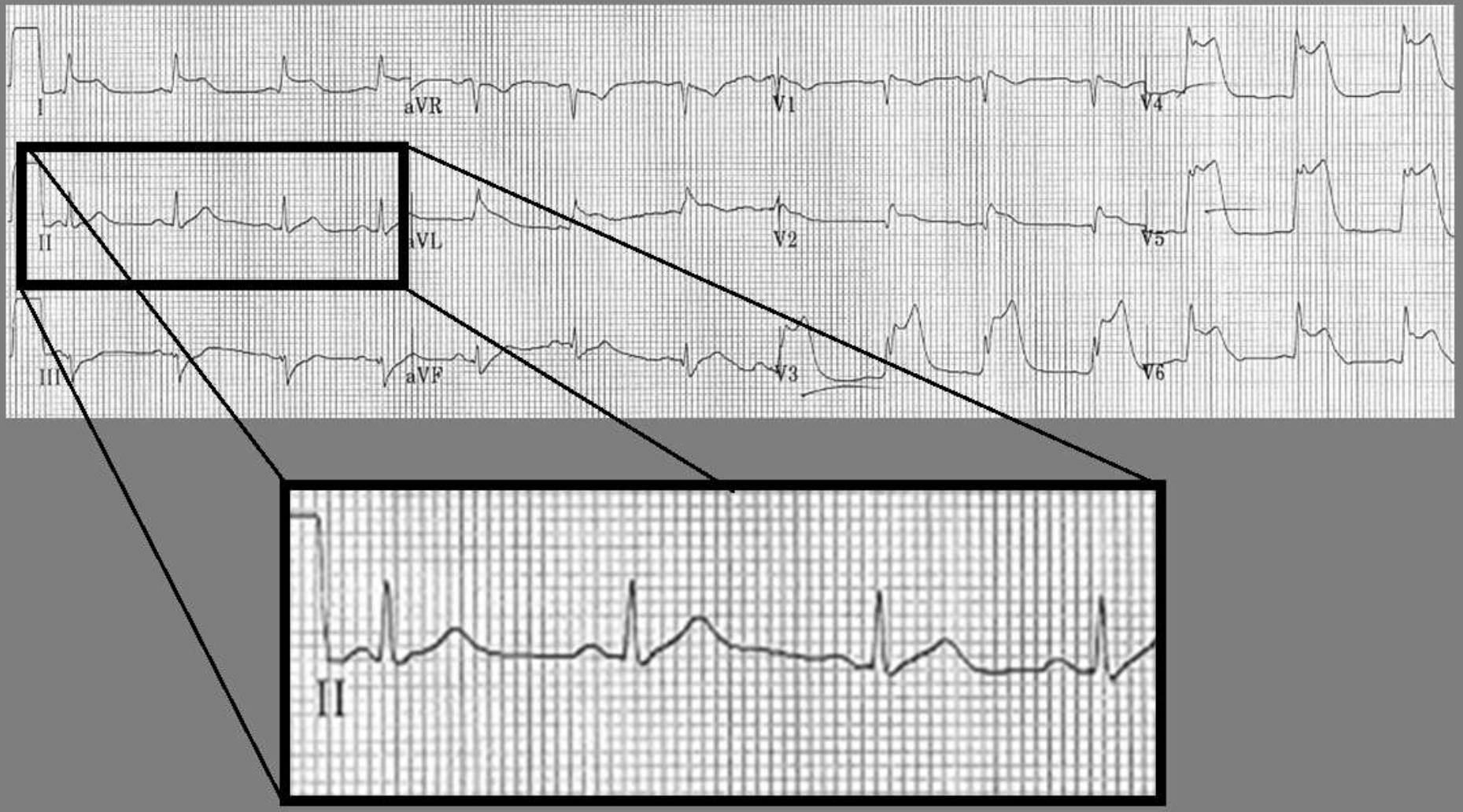
Welcome !

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

49 year old male - "Crushing chest pressure" . . . .



49 year old male - "Crushing chest pressure" . . . .



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

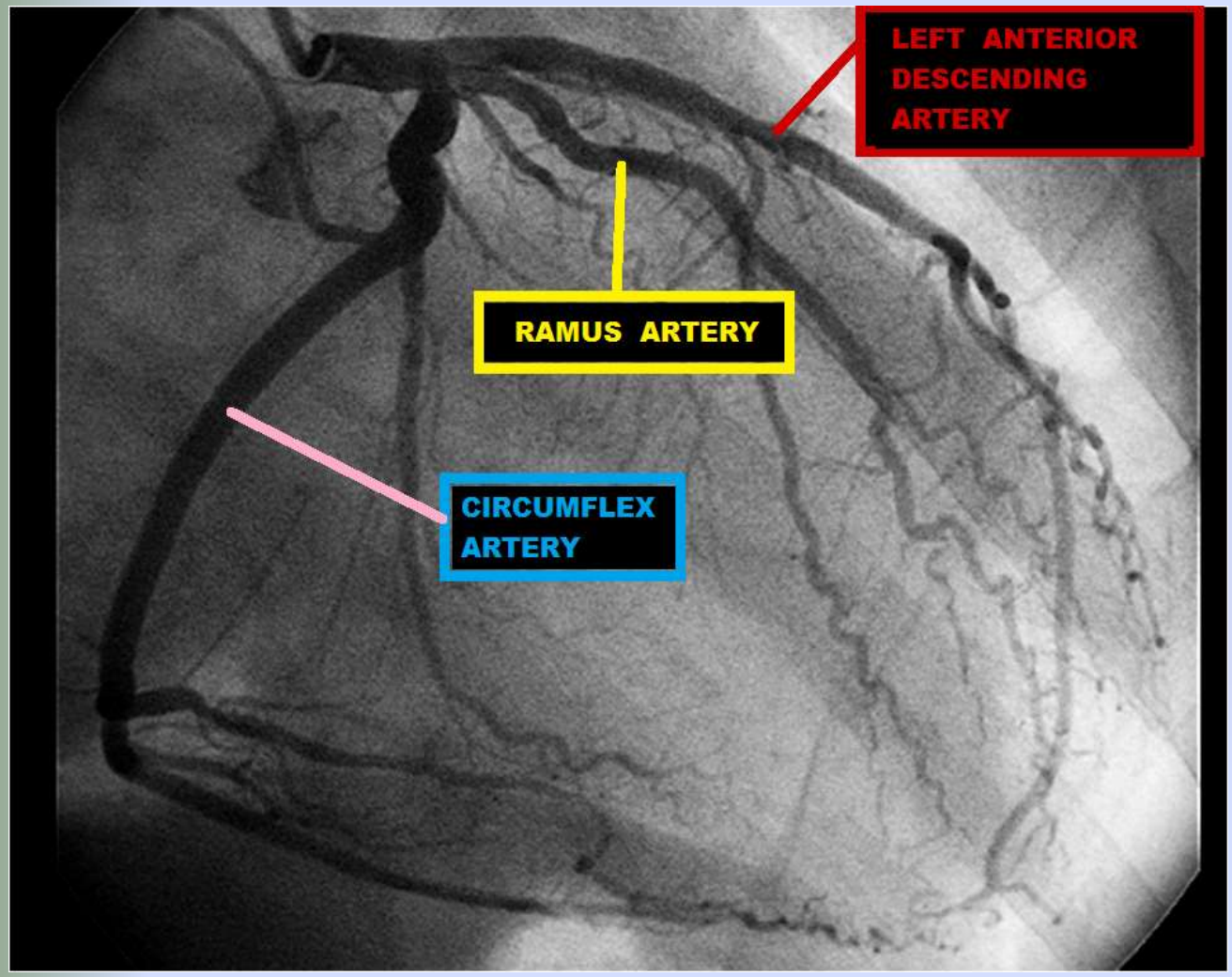
Source of Curriculum:

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

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

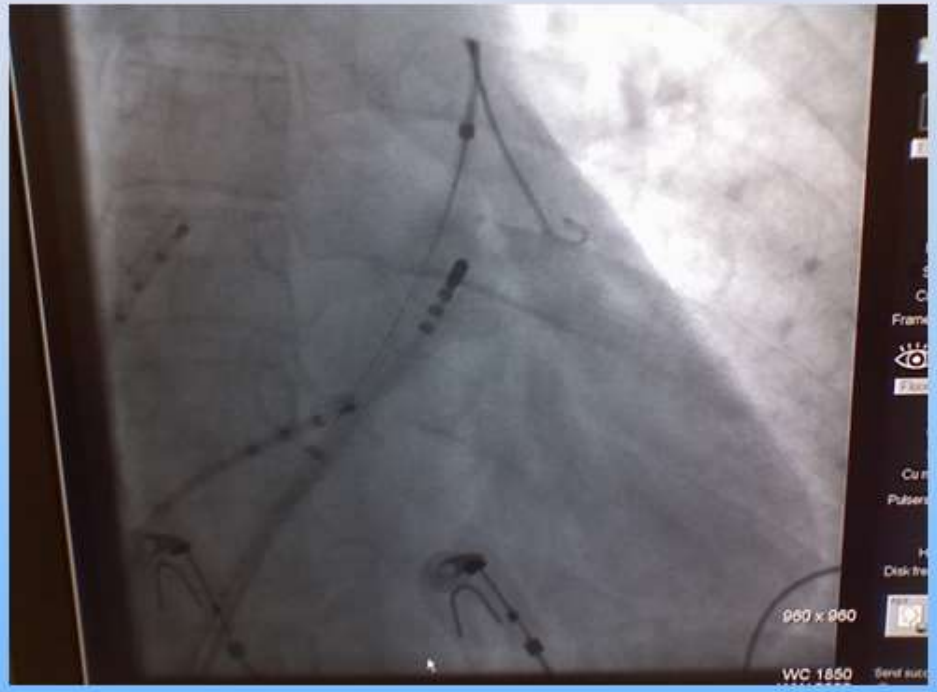
Wayne Ruppert and Dr. James Irwin, St Joseph's Hospital, Tampa, 7/29/2004

Cardiac Cath Lab Advantage:



Correlation of ECG leads with SPECIFIC cardiac anatomic structures.

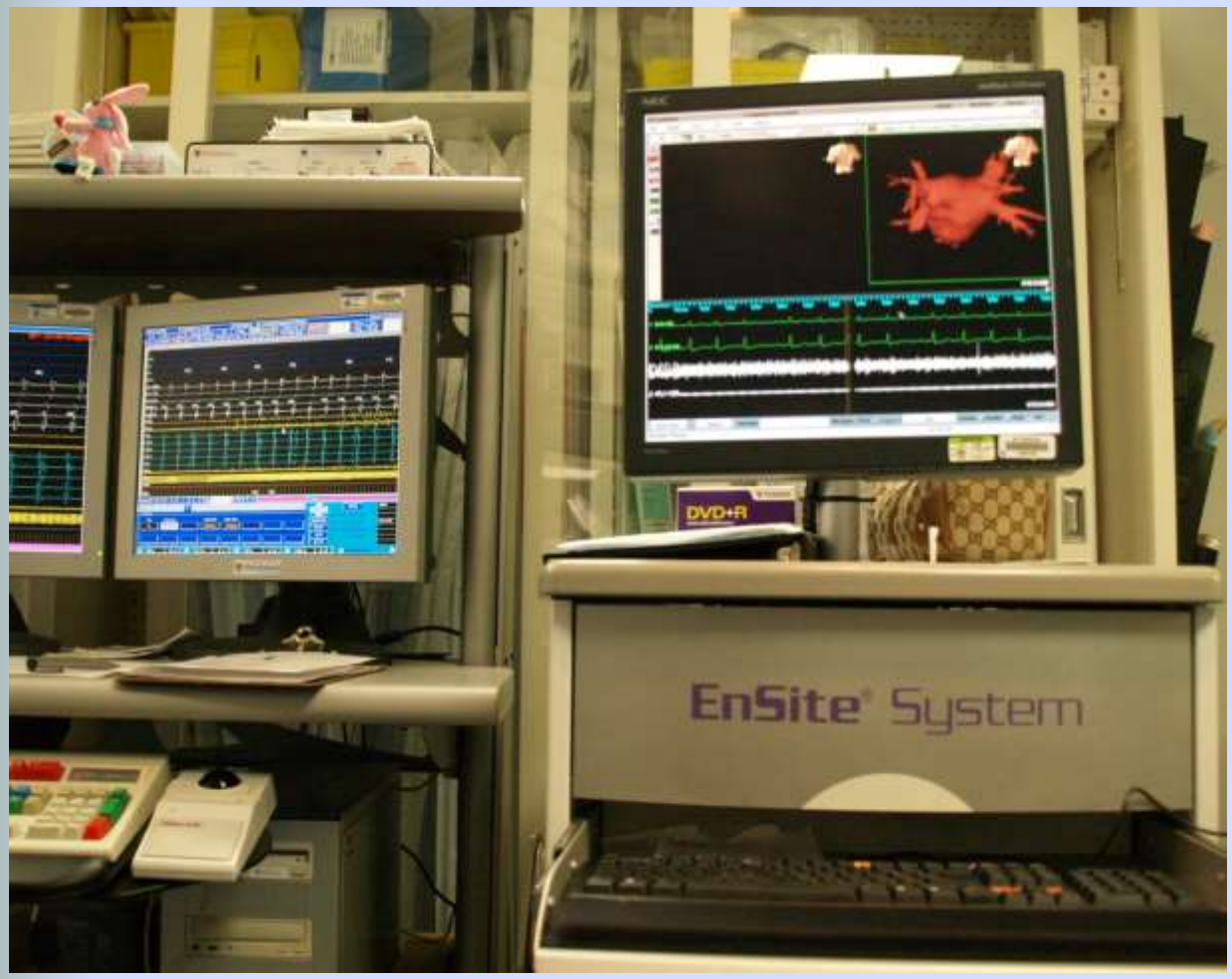
Electrophysiology Lab Case Studies



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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  - 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

ISBN 978-0-9829172-1-3
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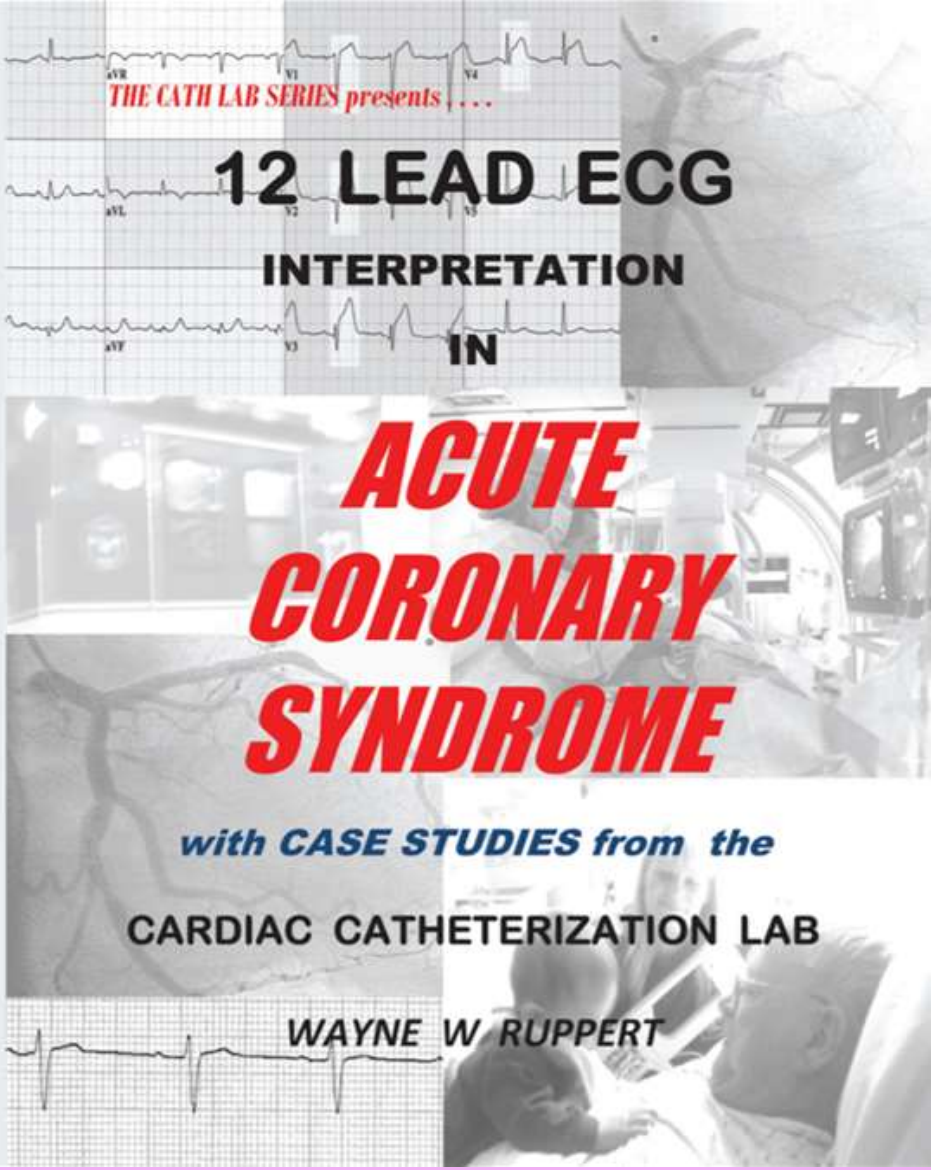
9 780982 917213

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



[Amazon.com](#)

[Barnes and Noble](#)

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

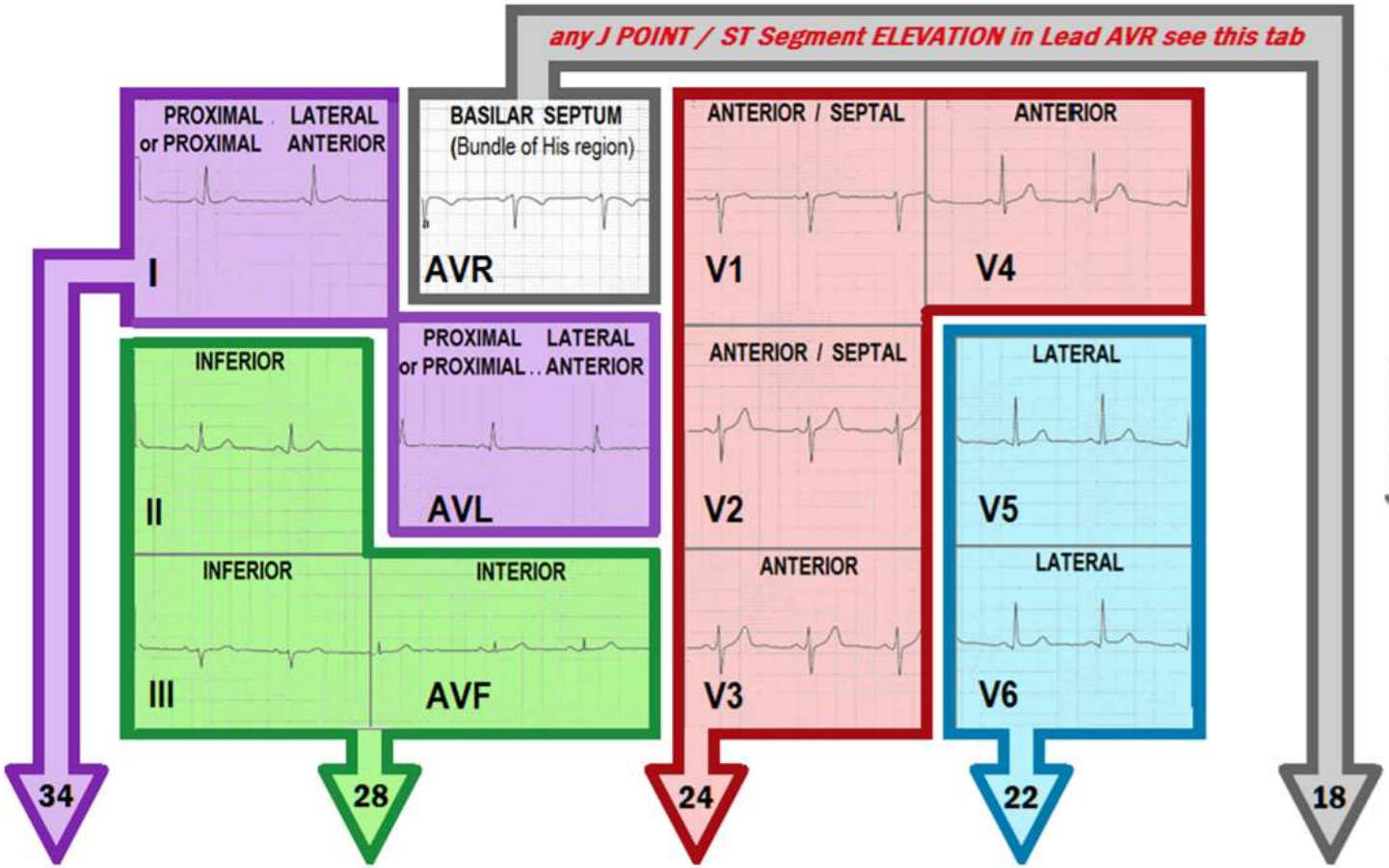
STEMI Assistant

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, Utrech, 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

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**
   - **Catecholinergic 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.

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- Do not try to write down or memorize every point.
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- For now …. Simply LISTEN to everything that is said. If it “makes sense,” then you’re learning.

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- DOWNLOAD this PowerPoint in its entirety – review and study it at you 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**
  - **Arrythmogenic Right Ventricular Cardiomyopathy**
  - **Brugada Syndrome**
- **Application of The HEART Score**
- **Acute Coronary Syndromes**

  With Cath Lab Case Studies

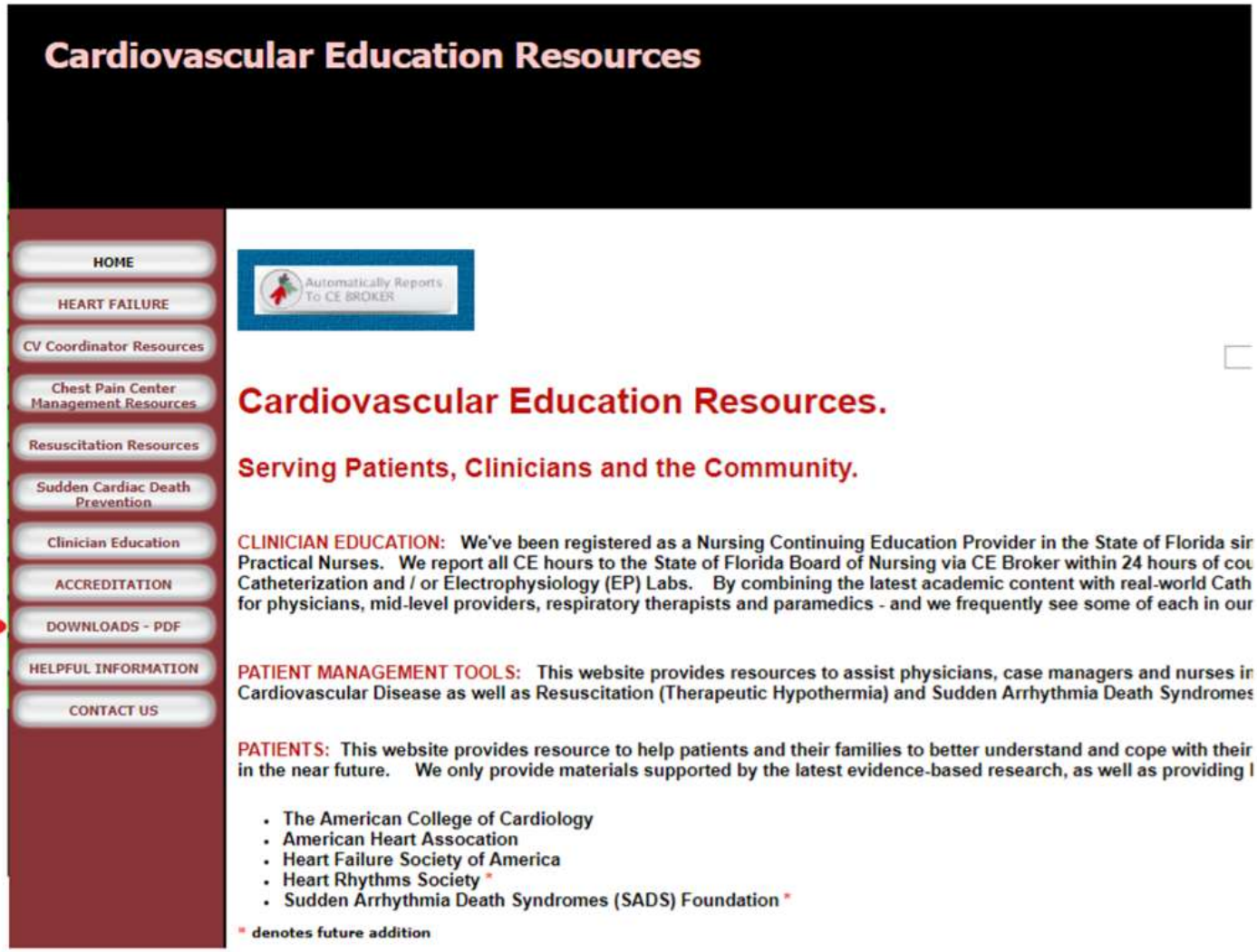
Helpful Web Resources:

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



1. Go to: www.ECGtraining.org
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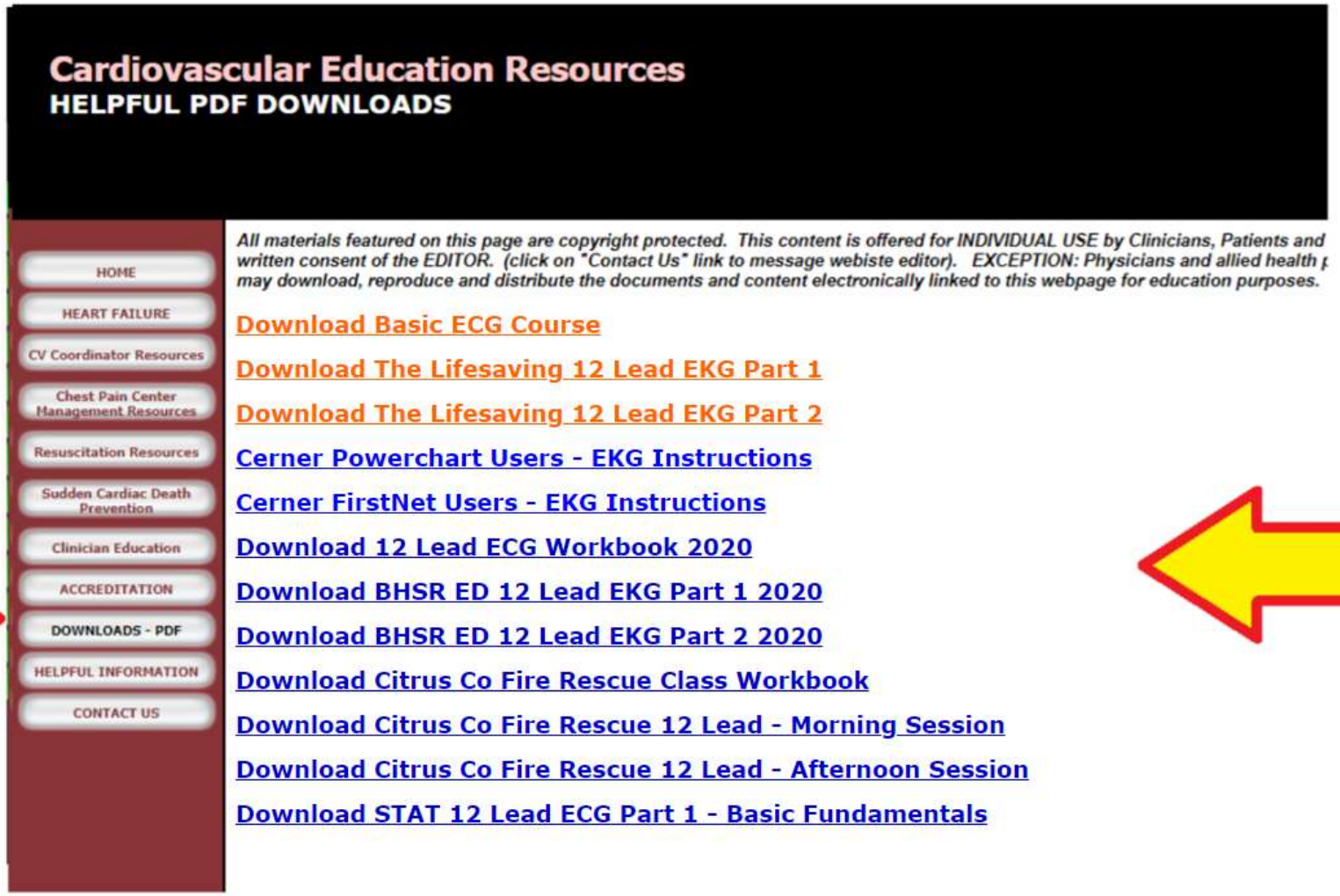
3. Select your courses



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)

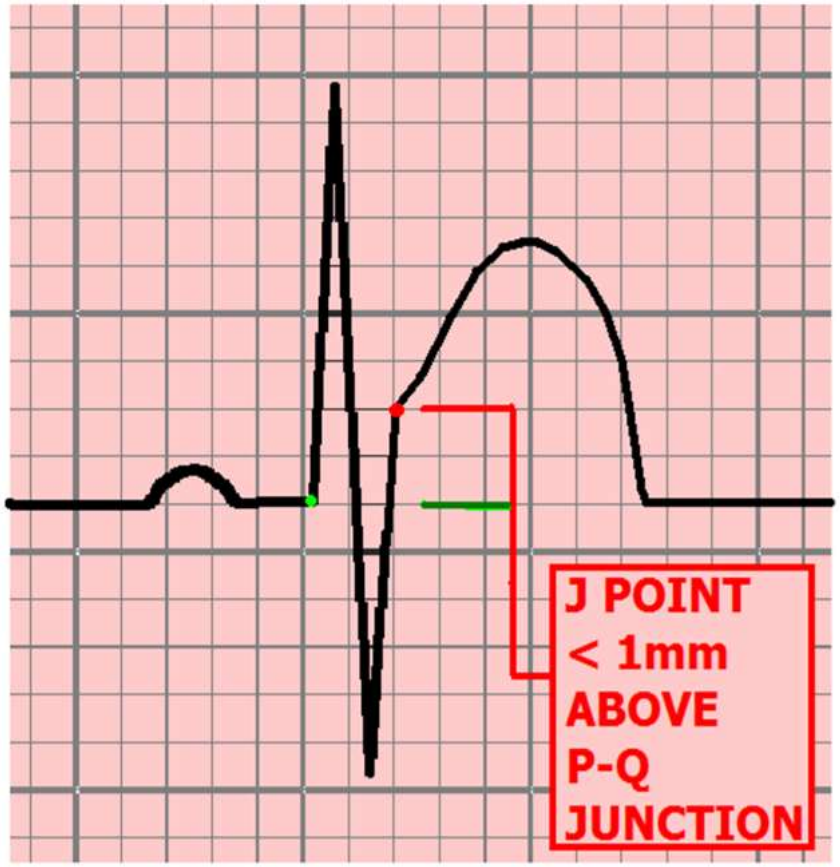
↓ SPECIFICITY

(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.

ST-Segment Elevation in Normal Circumstances and in Various Conditions

Table 1. ST-Segment Elevation in Normal Circumstances and in Various Conditions.

Condition	Features
Normal (so-called male pattern)	Seen in approximately 90 percent of healthy young men; therefore, normal Elevation of 1–3 mm Most marked in V_2 Concave
Early repolarization	Most marked in V_4, with notching at J point Tall, upright T waves Reciprocal ST depression in aVR, not in aVL, when limb leads are involved
ST elevation of normal variant	Seen in V_3 through V_4 with inverted T waves Short QT, high QRS voltage
Left ventricular hypertrophy	Concave Other features of left ventricular hypertrophy
Left bundle-branch block	Concave ST-segment deviation discordant from the QRS
Acute pericarditis	Diffuse ST-segment elevation Reciprocal ST-segment depression in aVR, not in aVL Elevation seldom >5 mm PR-segment depression
Hyperkalemia	Other features of hyperkalemia present: Widened QRS and tall, peaked, tented T waves Low-amplitude or absent P waves ST segment usually downsloping
Brugada syndrome	rSR' in V_1 and V_2 ST-segment elevation in V_1 and V_2, typically downsloping
Pulmonary embolism	Changes simulating myocardial infarction seen often in both inferior and anteroseptal leads
Cardioversion	Striking ST-segment elevation, often >10 mm, but lasting only a minute or two immediately after direct-current shock
Prinzmetal's angina	Same as ST-segment elevation in infarction, but transient
Acute myocardial infarction	ST segment with a plateau or shoulder or upsloping Reciprocal behavior between aVL and III

Wang K et al. N Engl J Med 2003;349:2128-2135

The NEW ENGLAND JOURNAL of MEDICINE

77 Years Male 7/2/2015 9:44:46

1North (06)

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

--AXIS--

P	59
QRS	14
T	33

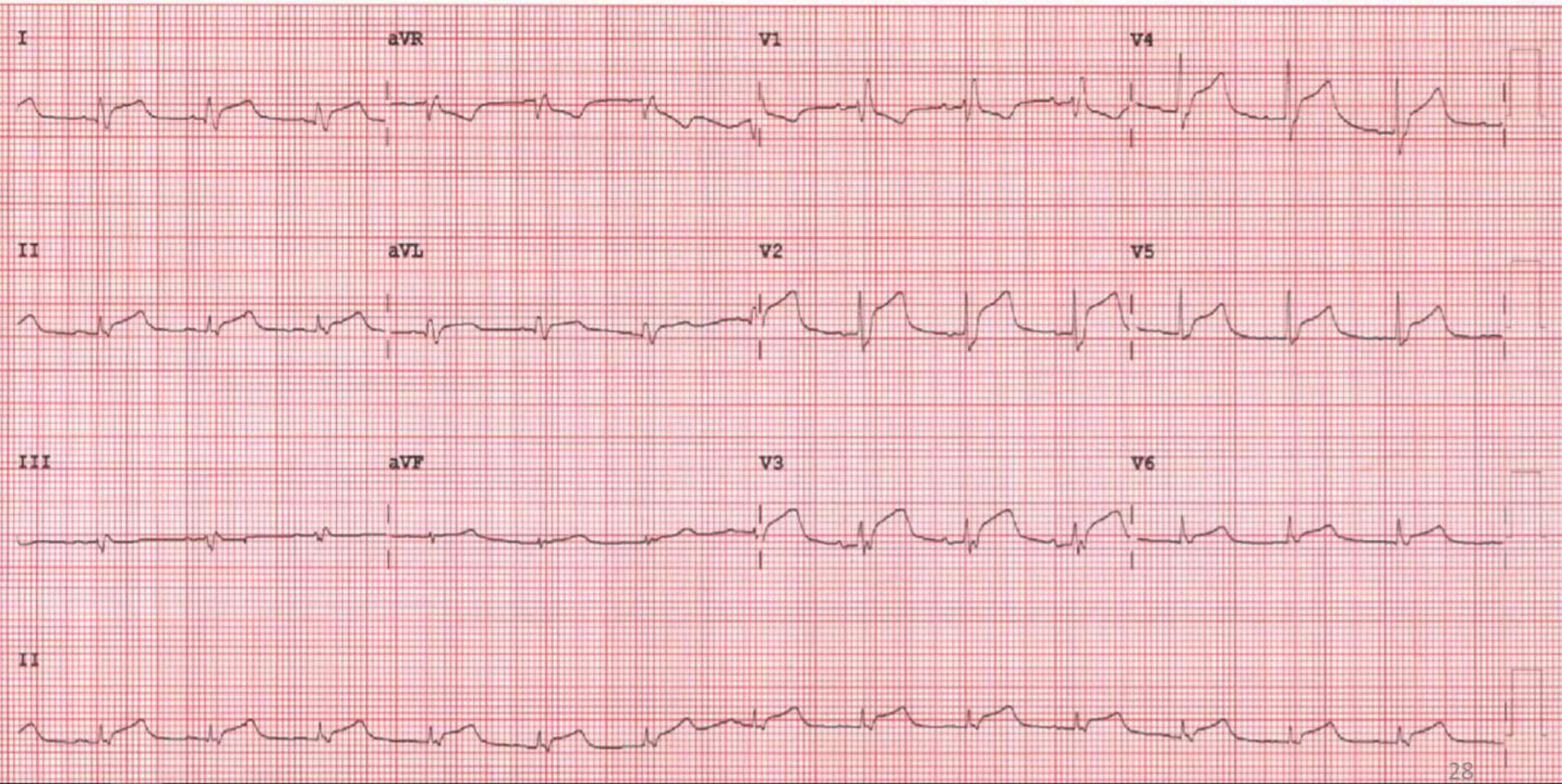
12 Lead; Standard Placement

- ABNORMAL ECG -

>>> Acute MI <<<

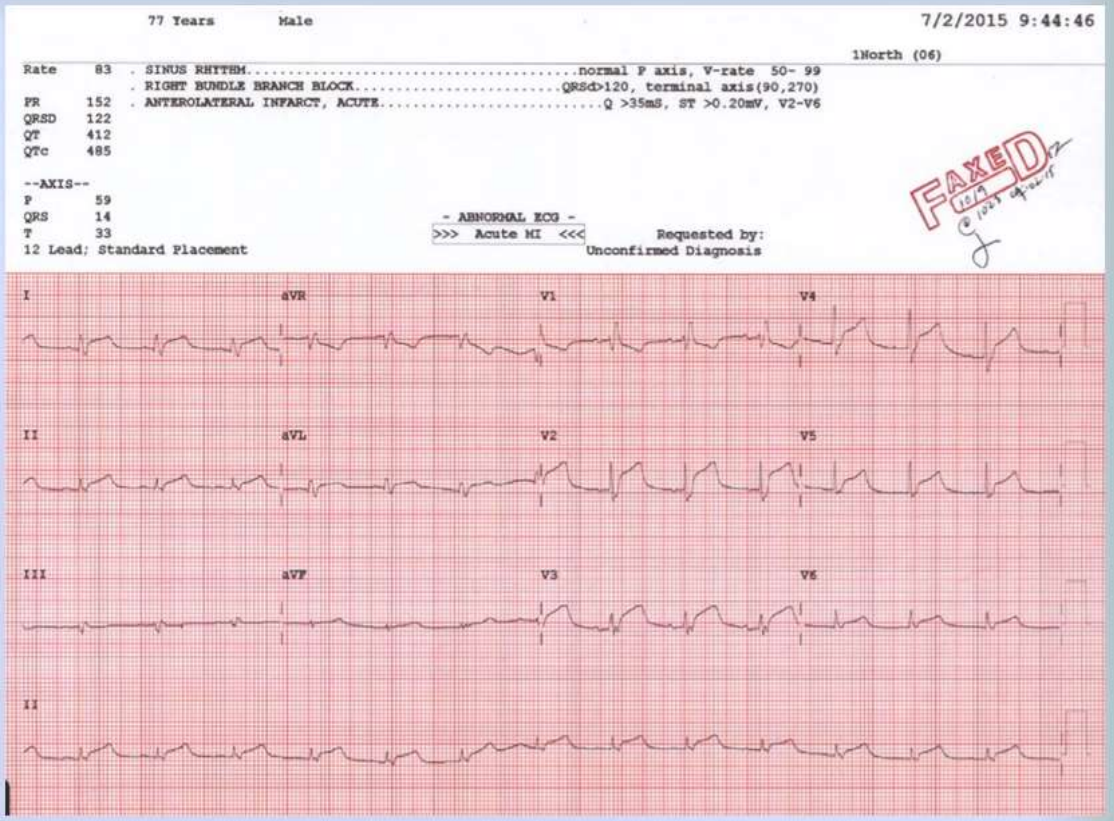
Requested by:
Unconfirmed Diagnosis

FAXED 10/9 @ 1023 cf 10.2.15



Patient:

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



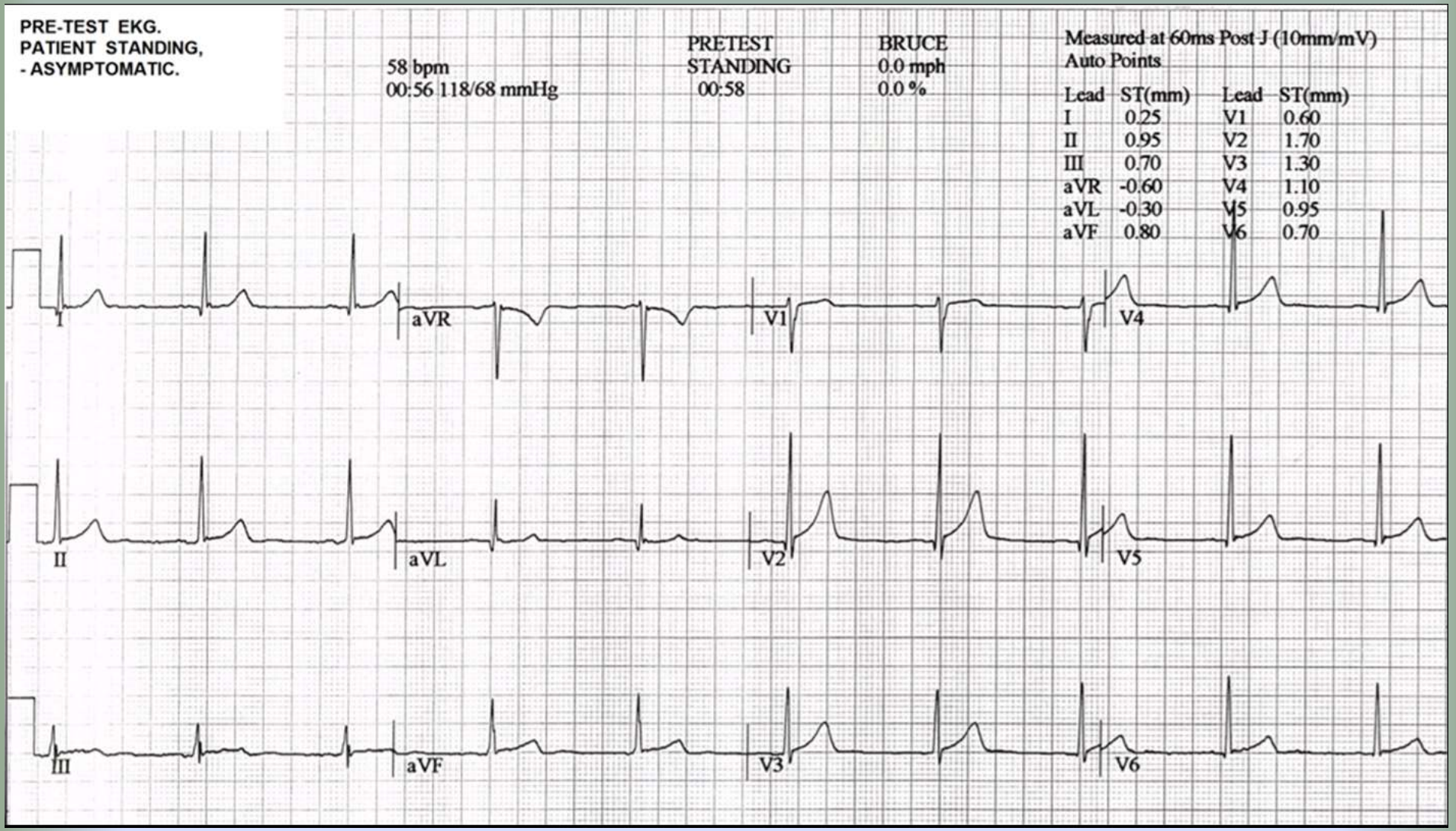
EARLY REPOLARIZATION. This degree of ST Elevation in early repolarization is VERY RARE: The only such ECG I have seen in approximately 13,000 cardiac catheterizations.

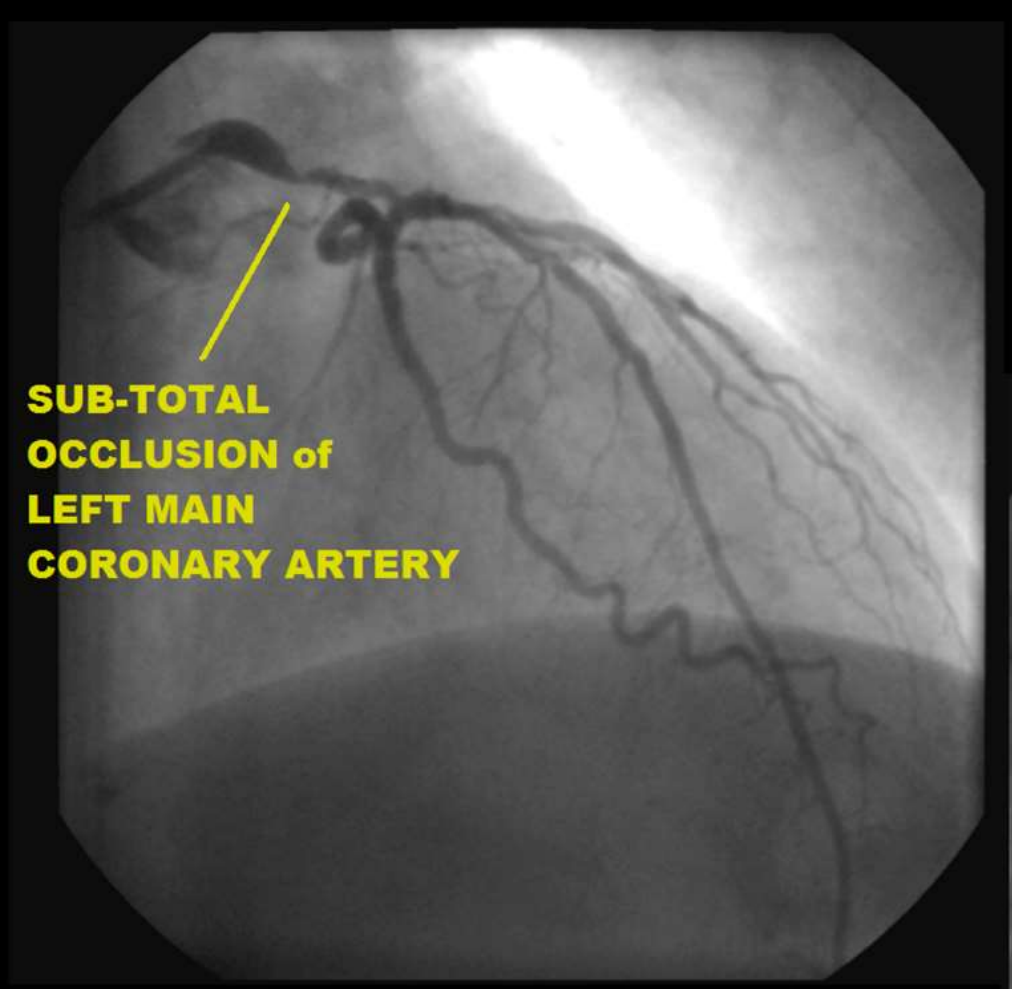
EKGs in PERSPECTIVE, con't:

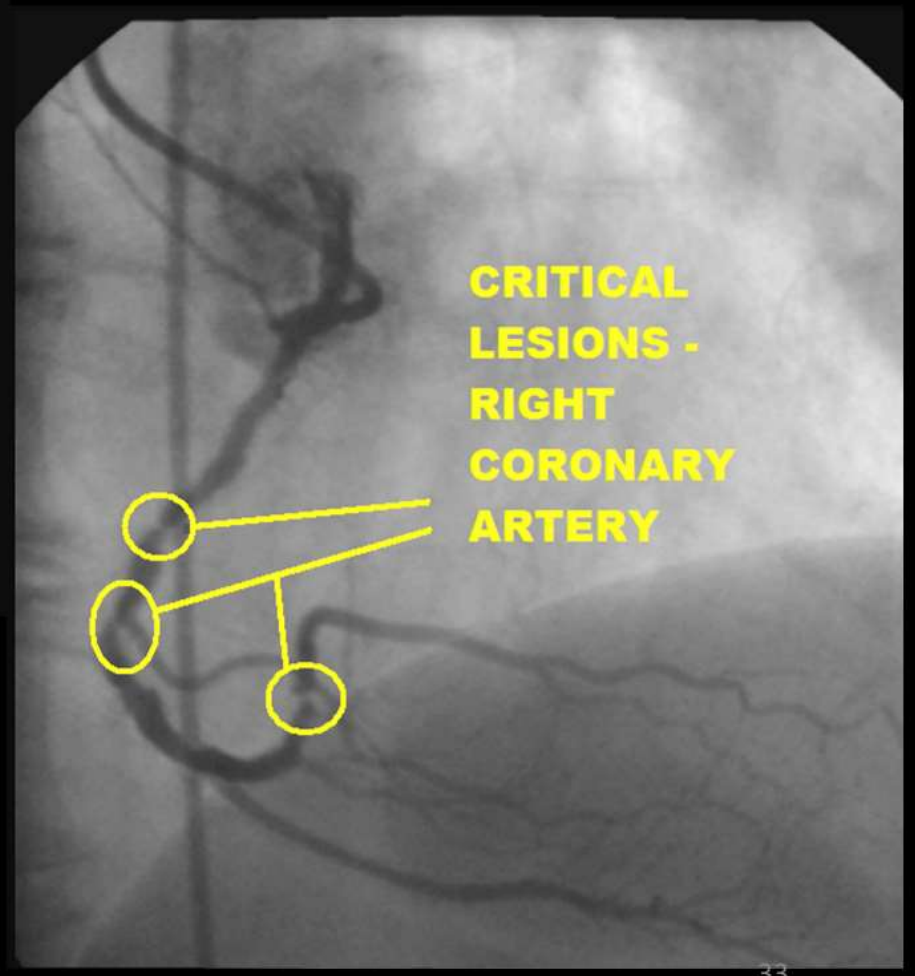
One of the MOST MISLEADING scenarios of all is when the EKG APPEARS PERFECTLY NORMAL . . .

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

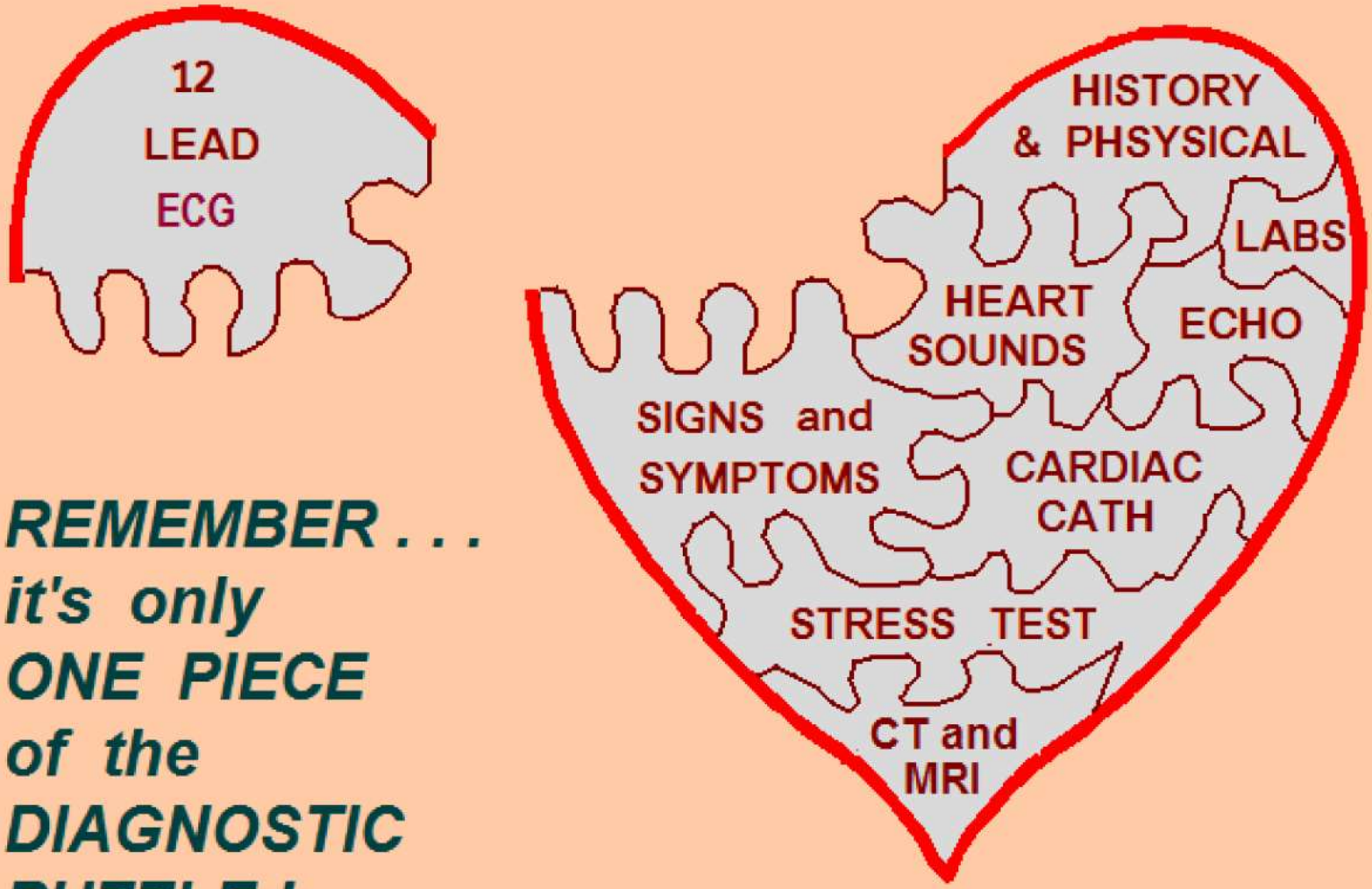

SUB-TOTAL
OCCLUSION of
LEFT MAIN
CORONARY ARTERY


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.***

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.

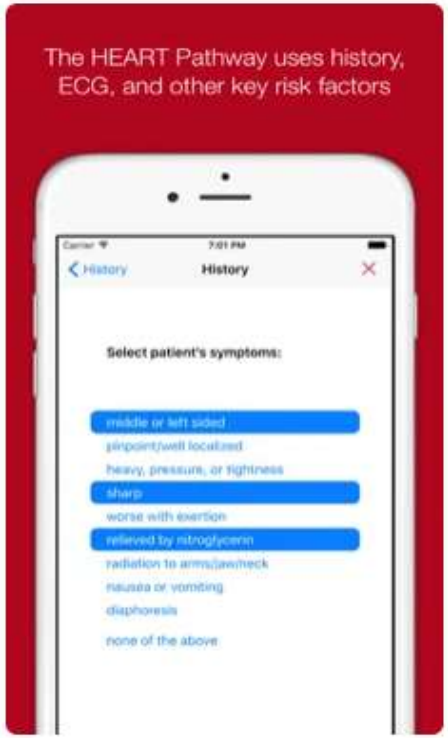
Impathiq

Designed for iPhone

★★★★½ 4.5 • 13 Ratings

Free

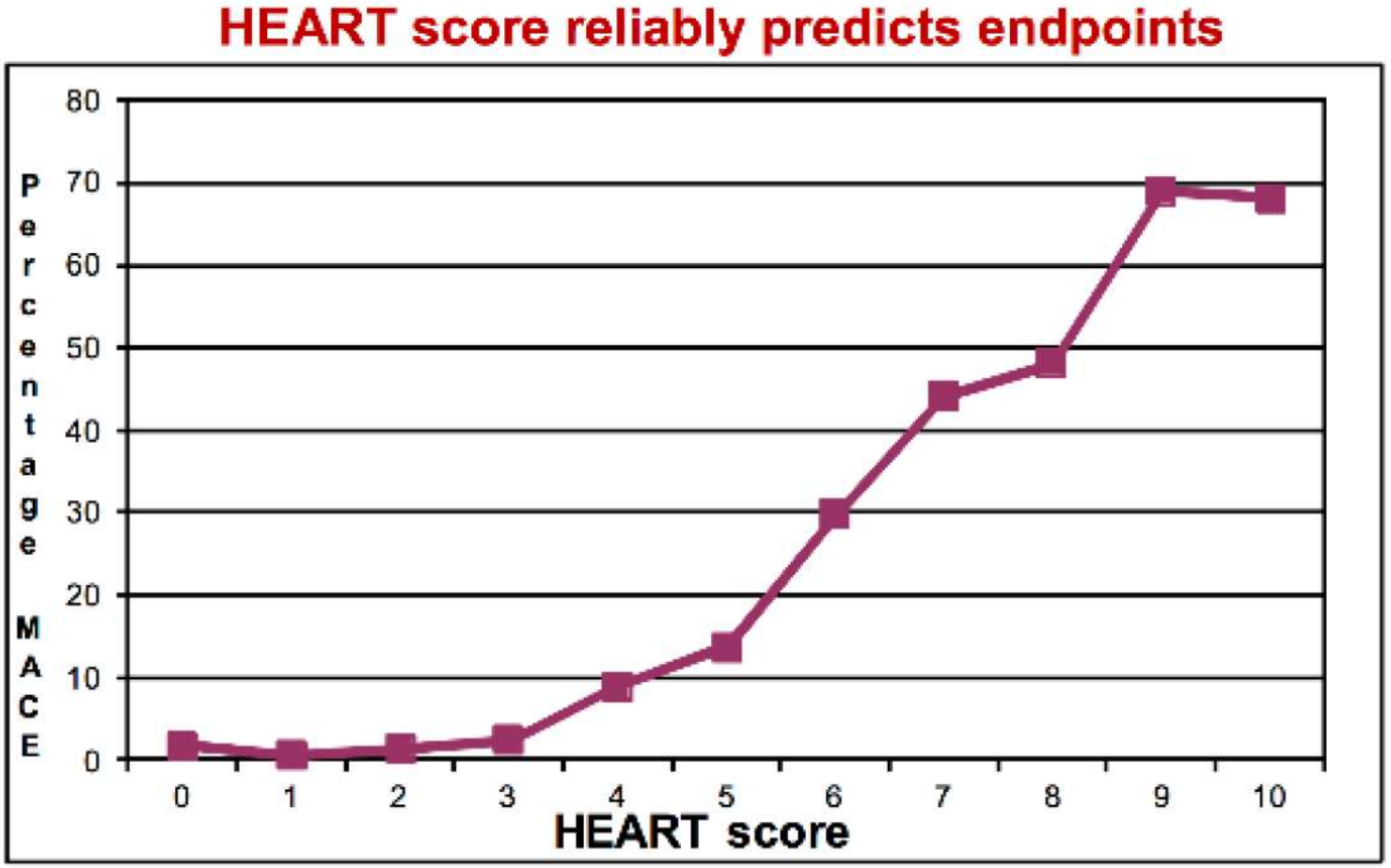
iPhone Screenshots



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



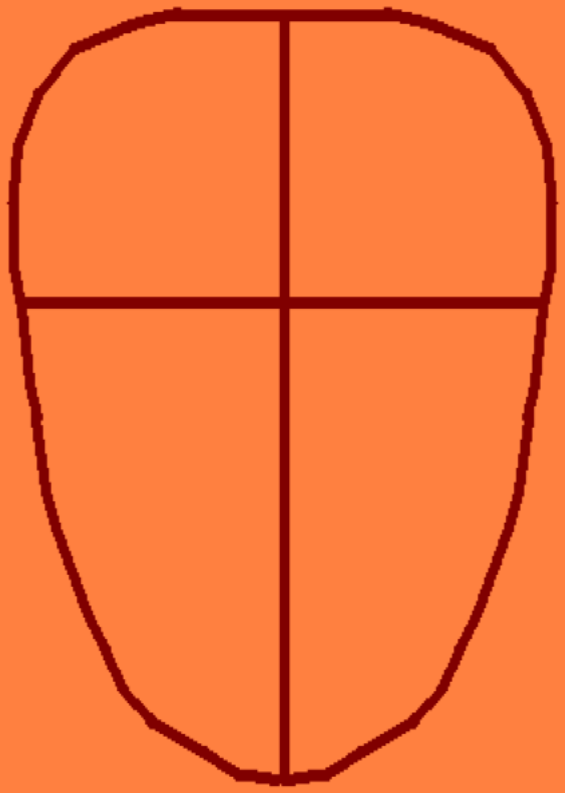
More on
HEART SCORE
in our ACS Case Studies
in Session 2 . . .

“Cardiac A & P 101”

“Cardiac A & P 101”

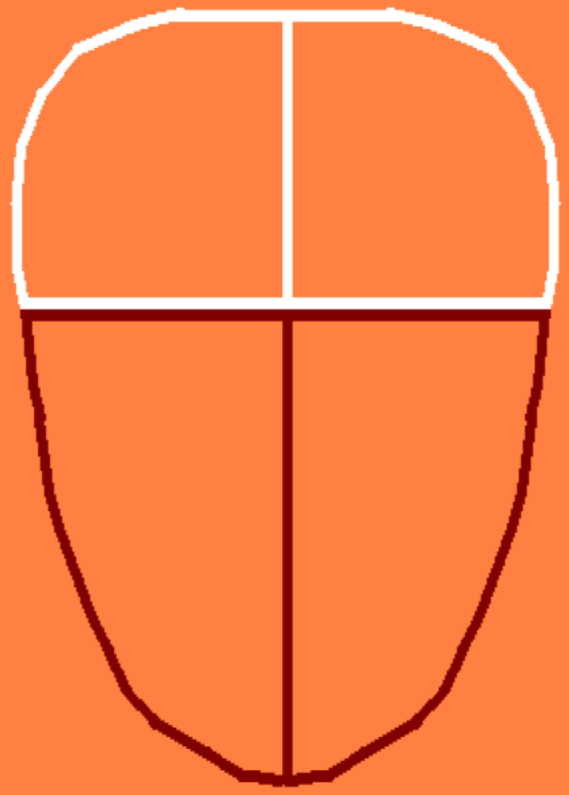
- **Heart Chambers**

FOUR CHAMBERED PUMP



FOUR CHAMBERED PUMP . . .

2 ATRIUM

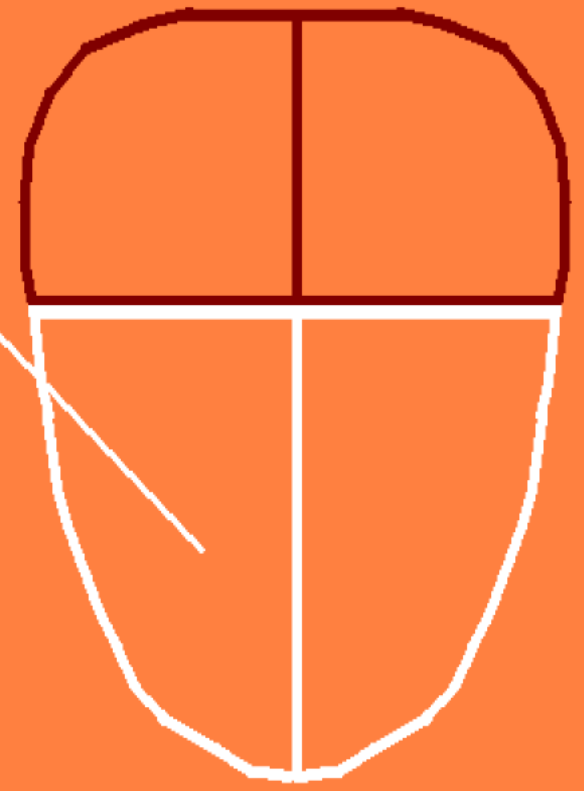


PRIMARY JOB:

"PACK VENTRICLES FULL OF BLOOD"

FOUR CHAMBERED PUMP . . .

2 VENTRICLES



PRIMARY JOB:

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

WHEN FUNCTIONING PROPERLY,

THE ATRIUM SUPPLY

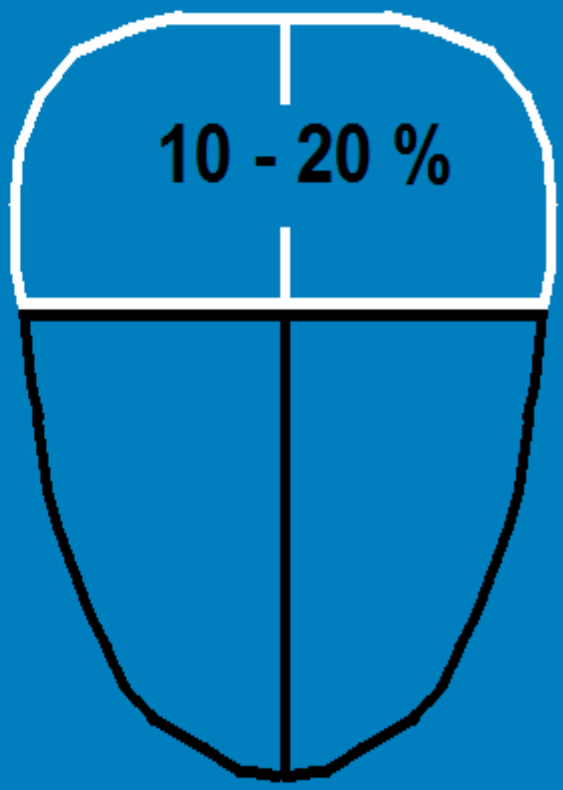
APPROXIMATELY

WHAT

PERCENTAGE

OF THE

CARDIAC OUTPUT ?



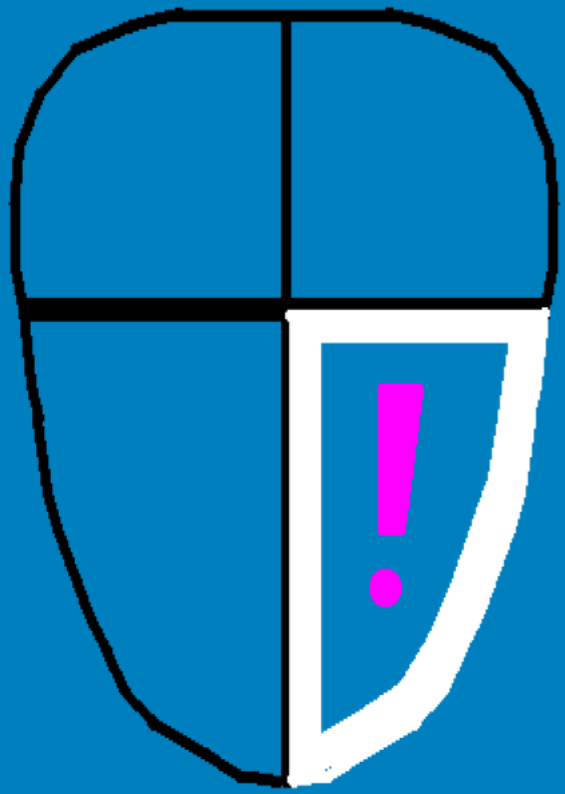
THE CHAMBER MOST IMPORTANT TO KEEPING THE PATIENT ALIVE

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

IS THE

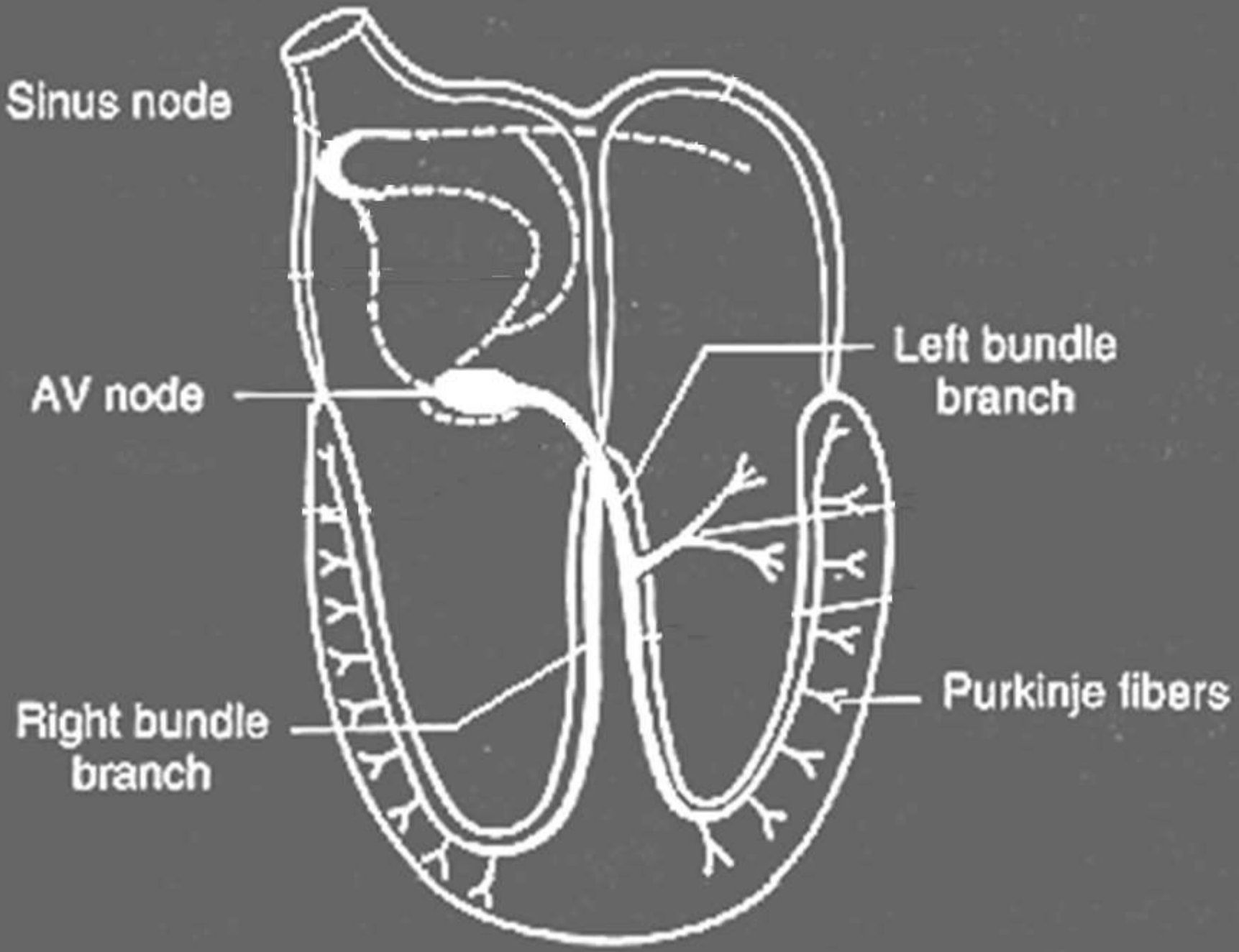
LEFT VENTRICLE

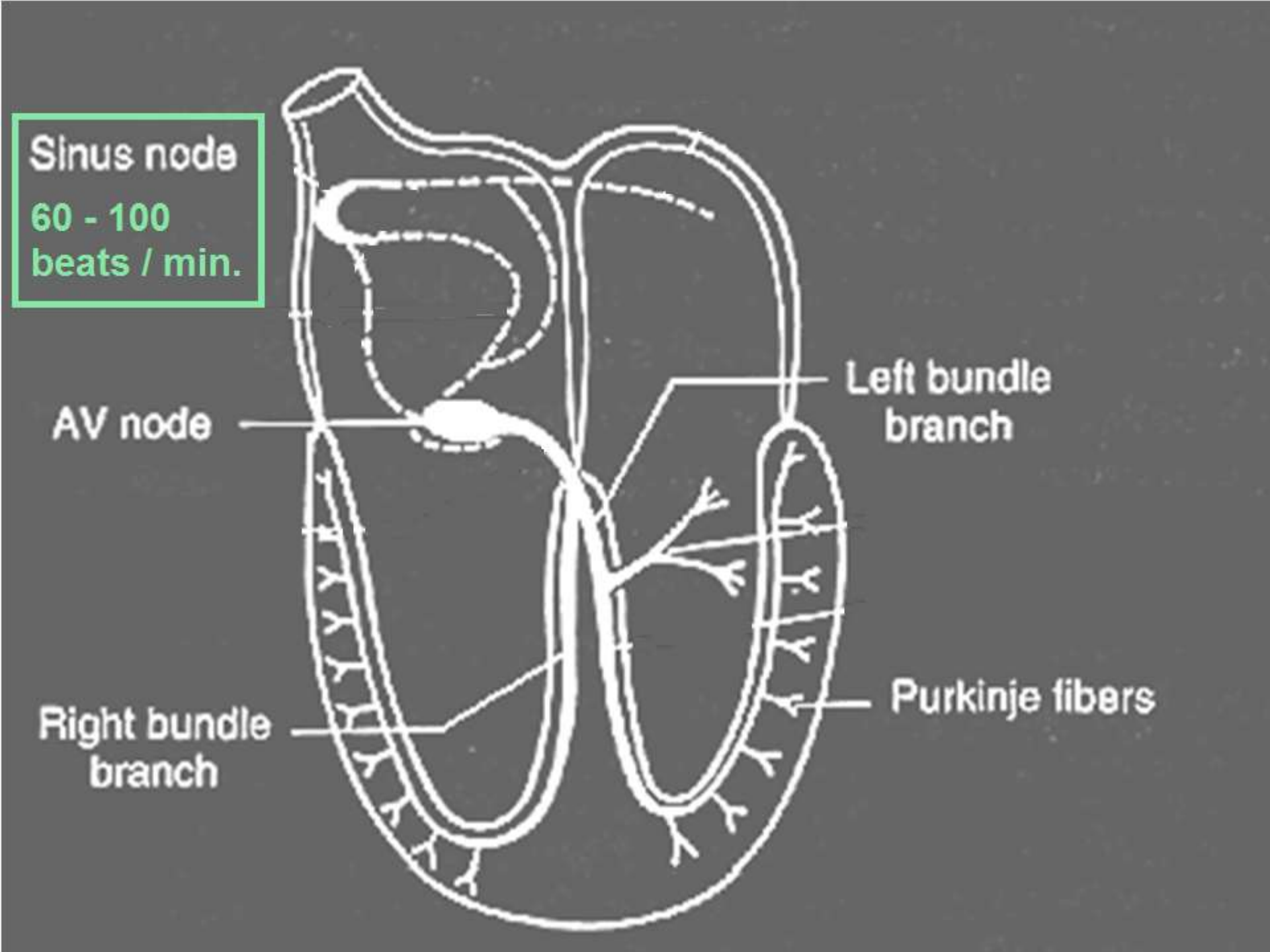
WHICH WE WILL REFER TO AS THE PUMP

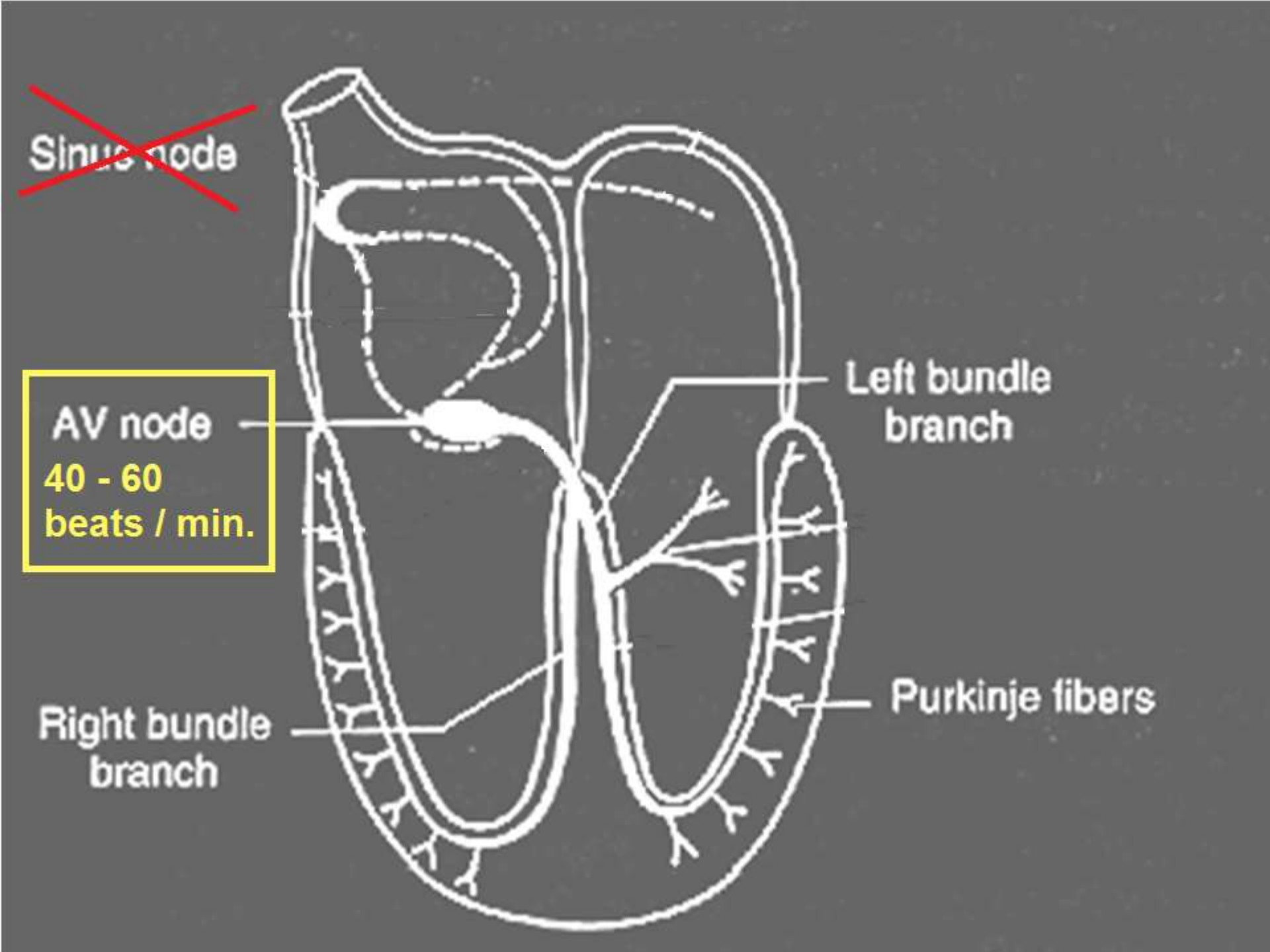


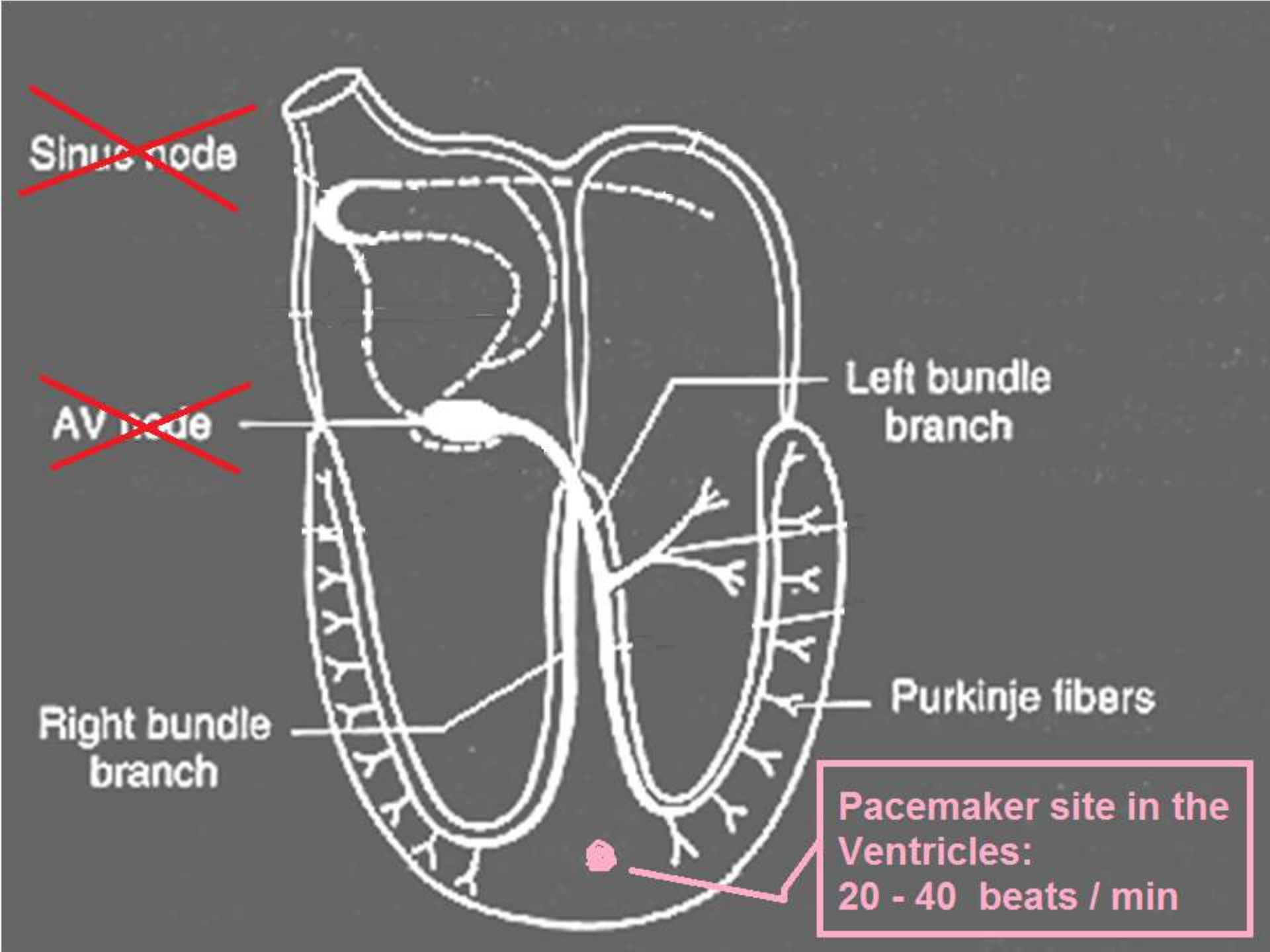
“Cardiac A & P 101”

- Heart Chambers
- **Heart Electrical System & ECG Waveforms**


Sinus node
AV node
Left bundle branch
Right 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.
Left bundle
branch
Right 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

“Cardiac A & P 101”

- Heart Chambers
- Heart Electrical System & ECG Waveforms
- **Depolarization and Repolarization**

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

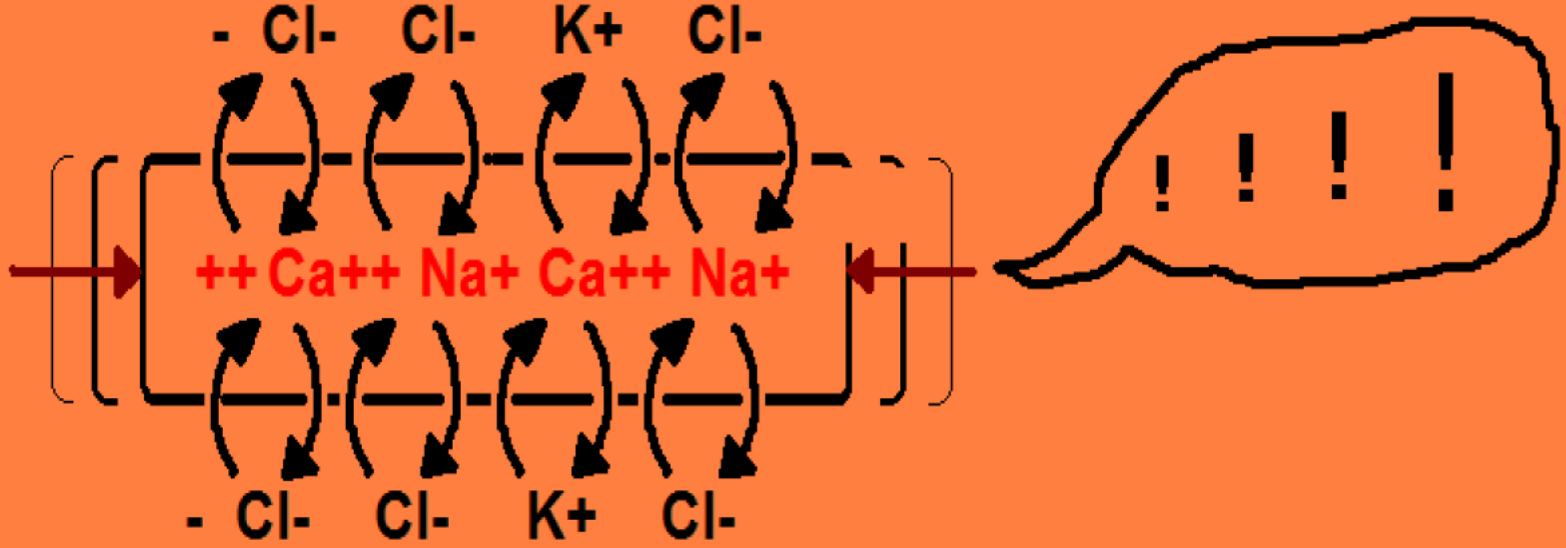
Ca++ Na+ Ca++ Na+ Ca++

Cl- Cl- K+ Cl- K+ Cl- K+ Cl-

Ca++ Na+ Ca++ Na+ Ca++

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"

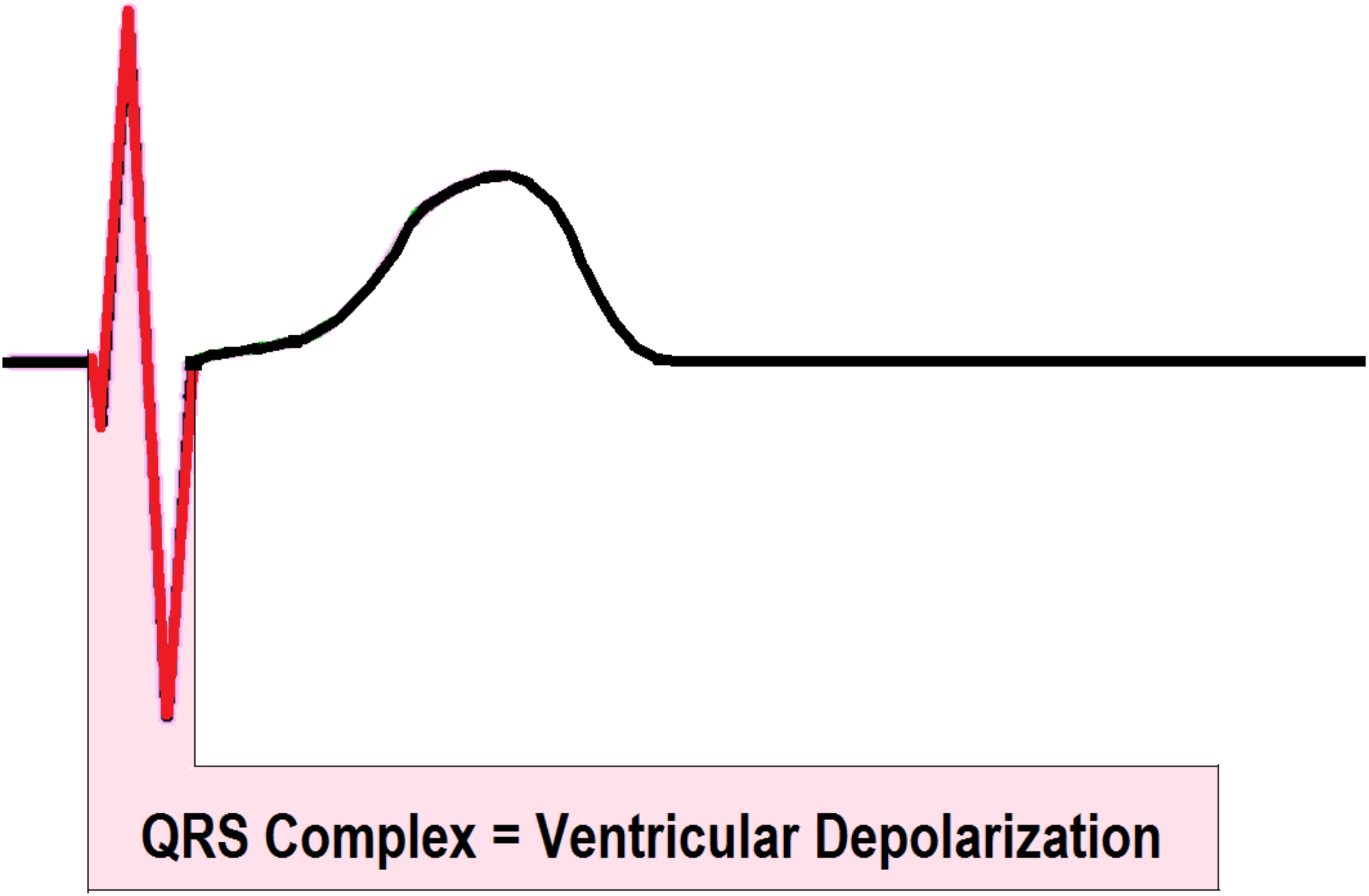
THIS (OF COURSE) IS KNOW AS . . .

DEPOLARIZATION

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

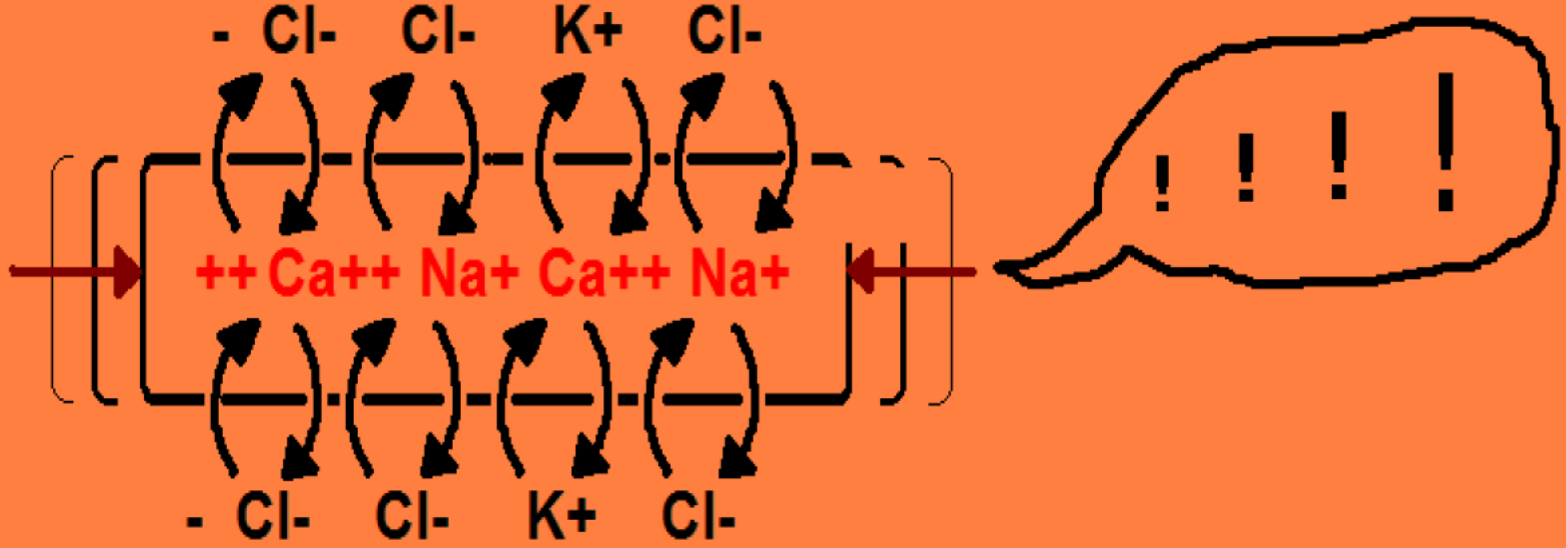
Ventricular Depolarization:

- Is represented by the **QRS Complex**


QRS Complex = Ventricular Depolarization

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 . . . .



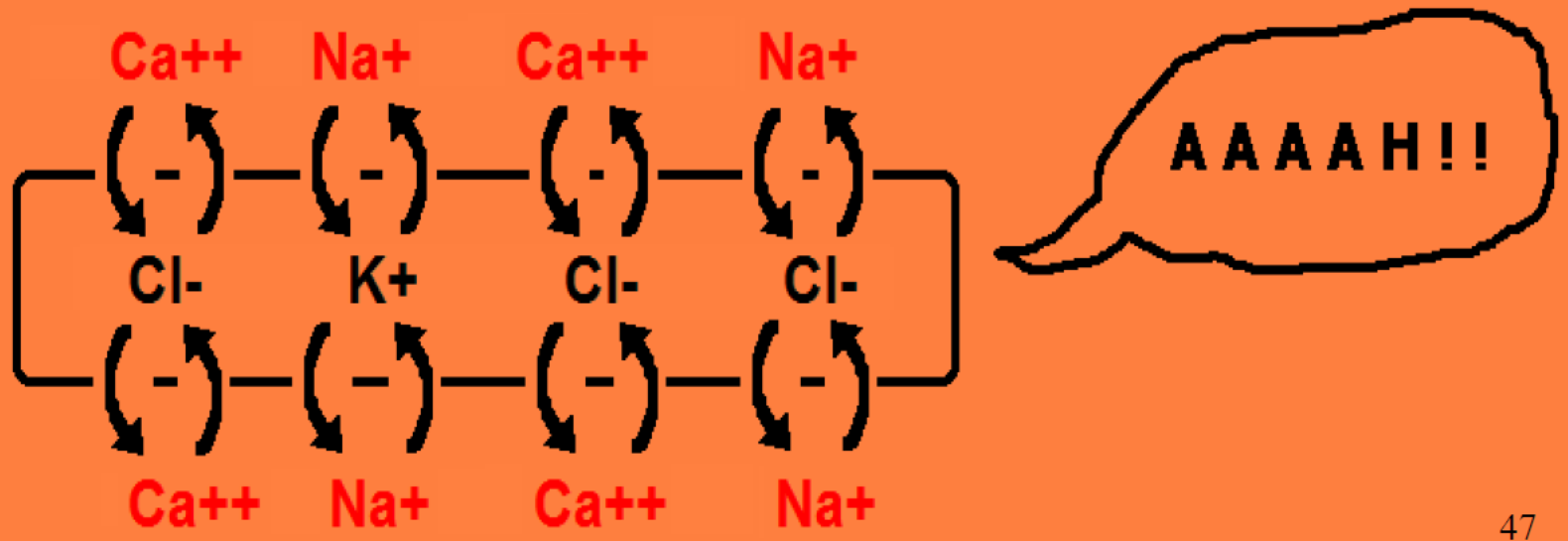
. . . THE CELL CONTRACTS!

CARDIAC ANATOMY and PHYSIOLOGY "101"

AFTER DEPOLARIZATION, THE CELLS RELAX.

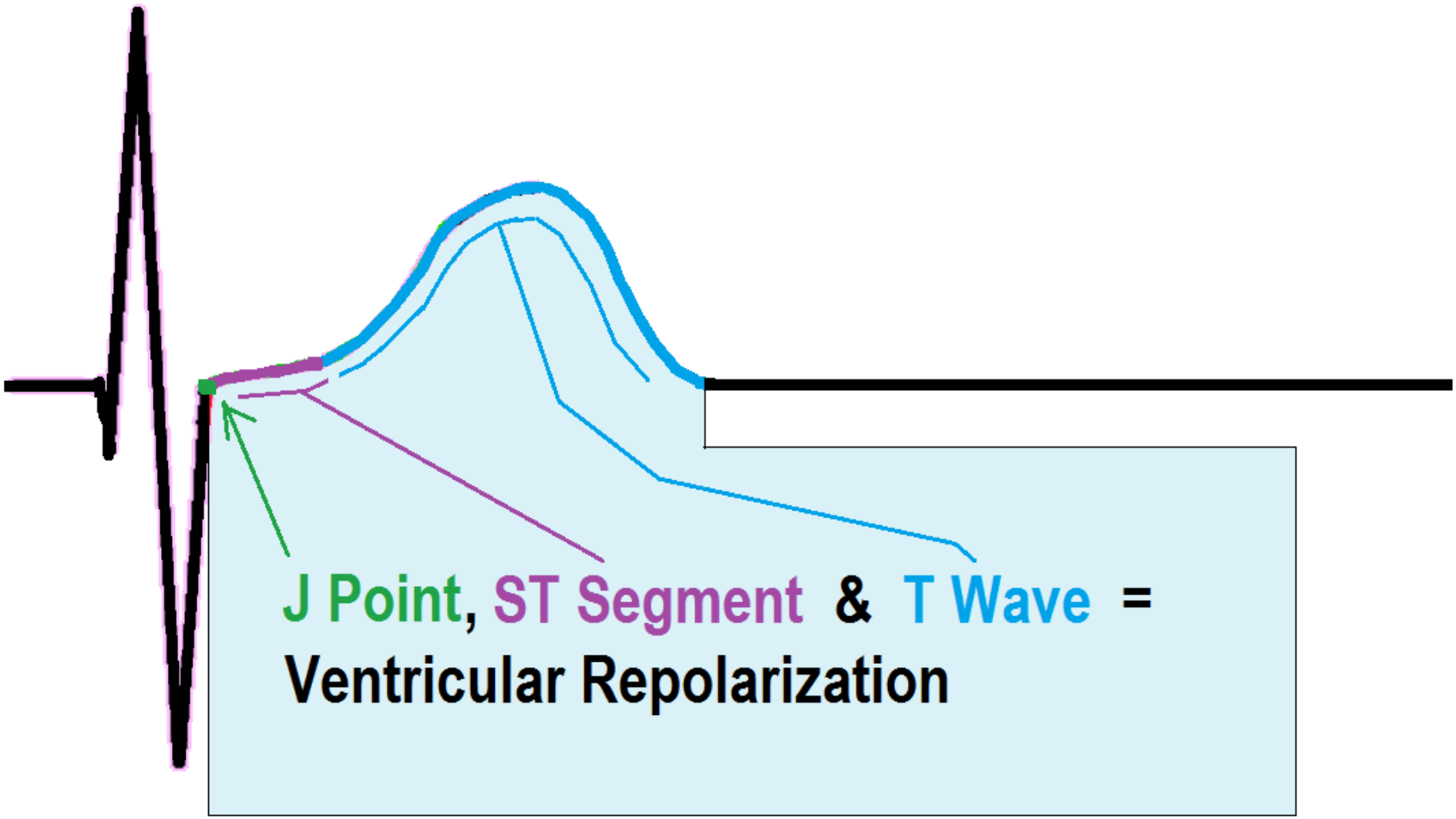
THE IONS RETURN TO THEIR ORIGINAL POSITIONS --

THIS PROCESS IS KNOWN AS REPOLARIZATION

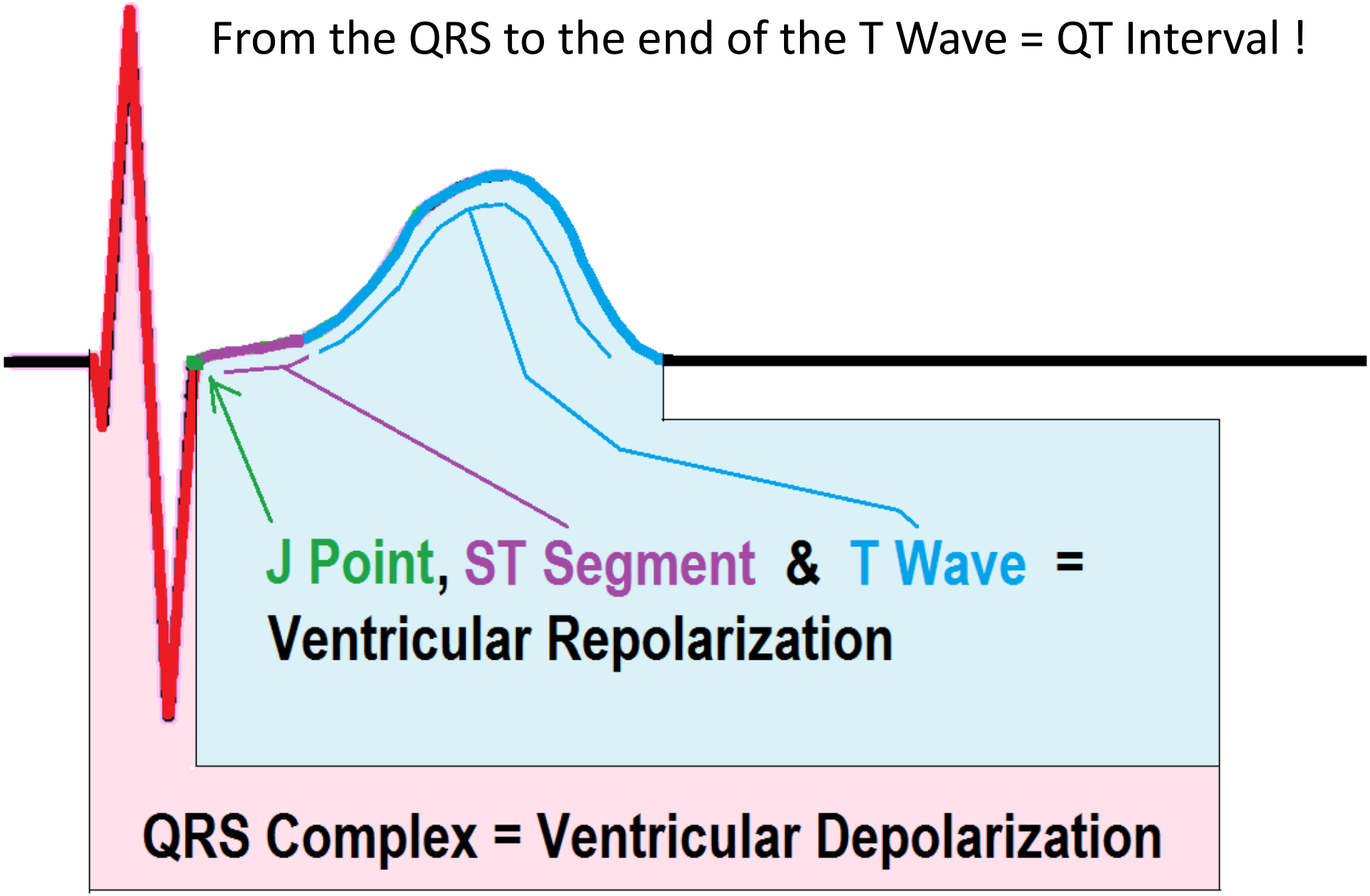


Repolarization on the ECG:

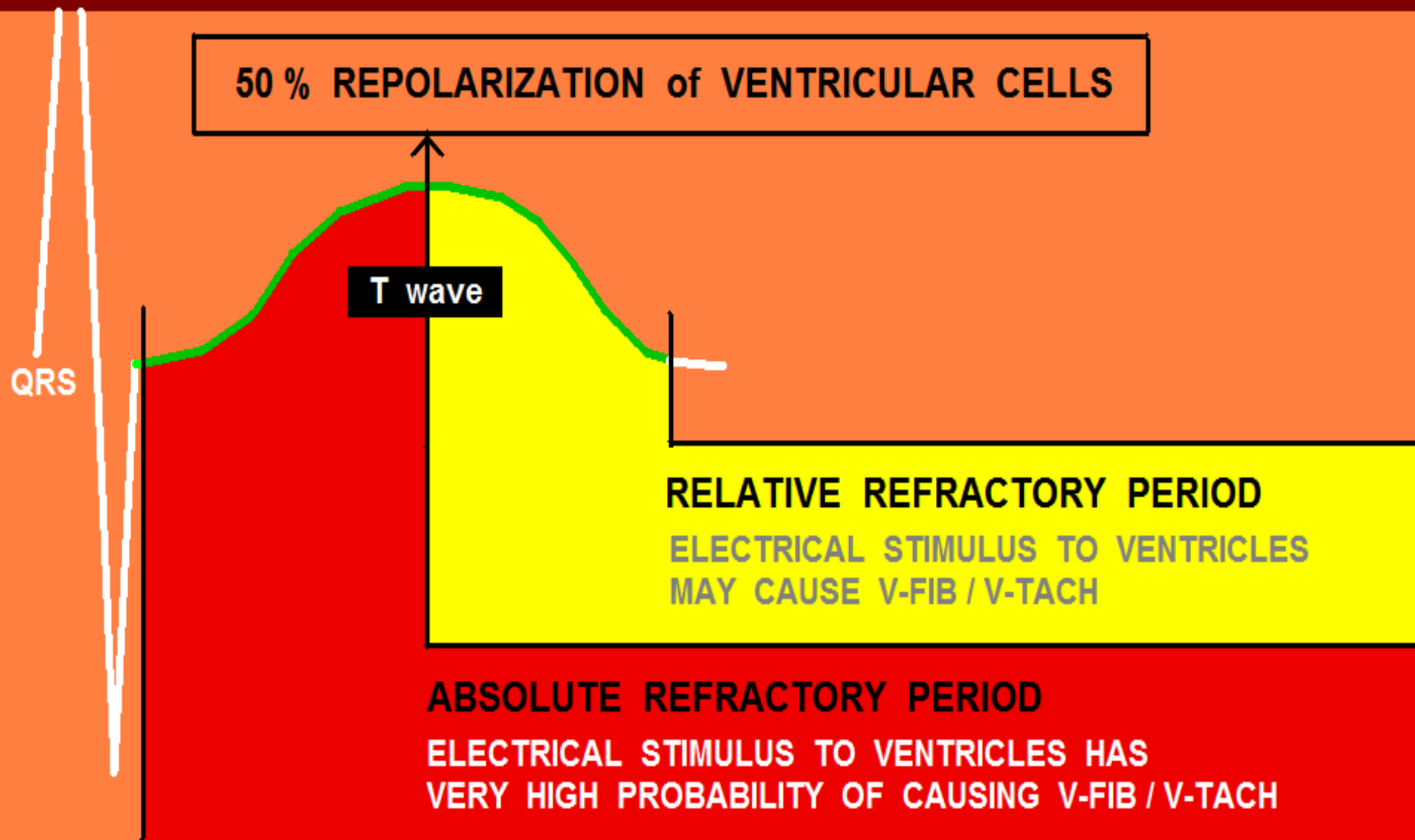
- Is represented by the:
  - **J Point**
  - **ST Segment**
  - **T Wave**


J Point, ST Segment & T Wave =
Ventricular Repolarization

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



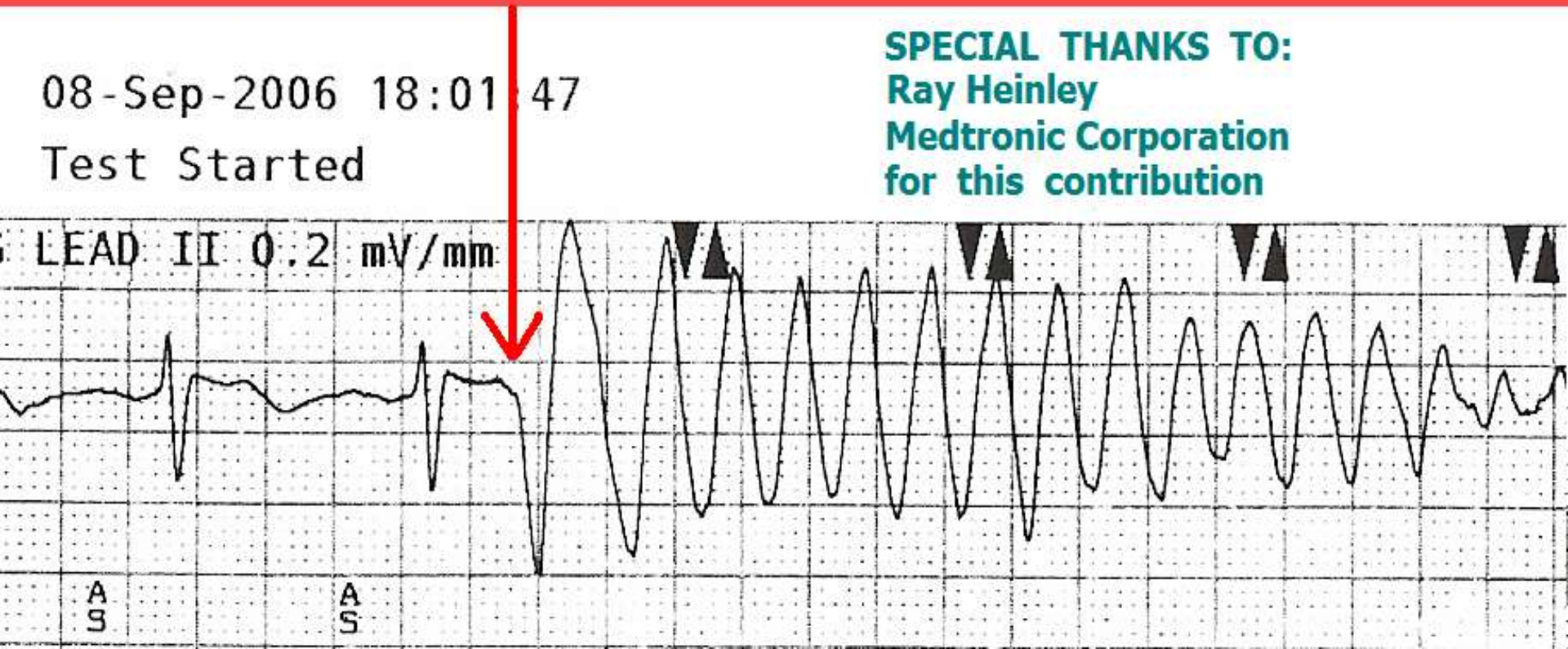
CARDIAC ANATOMY and PHYSIOLOGY "101"



ROUTINE TEST OF ICD

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

SPECIAL THANKS TO:
Ray Heinley
Medtronic Corporation
for this contribution




VOLTCRAFT®
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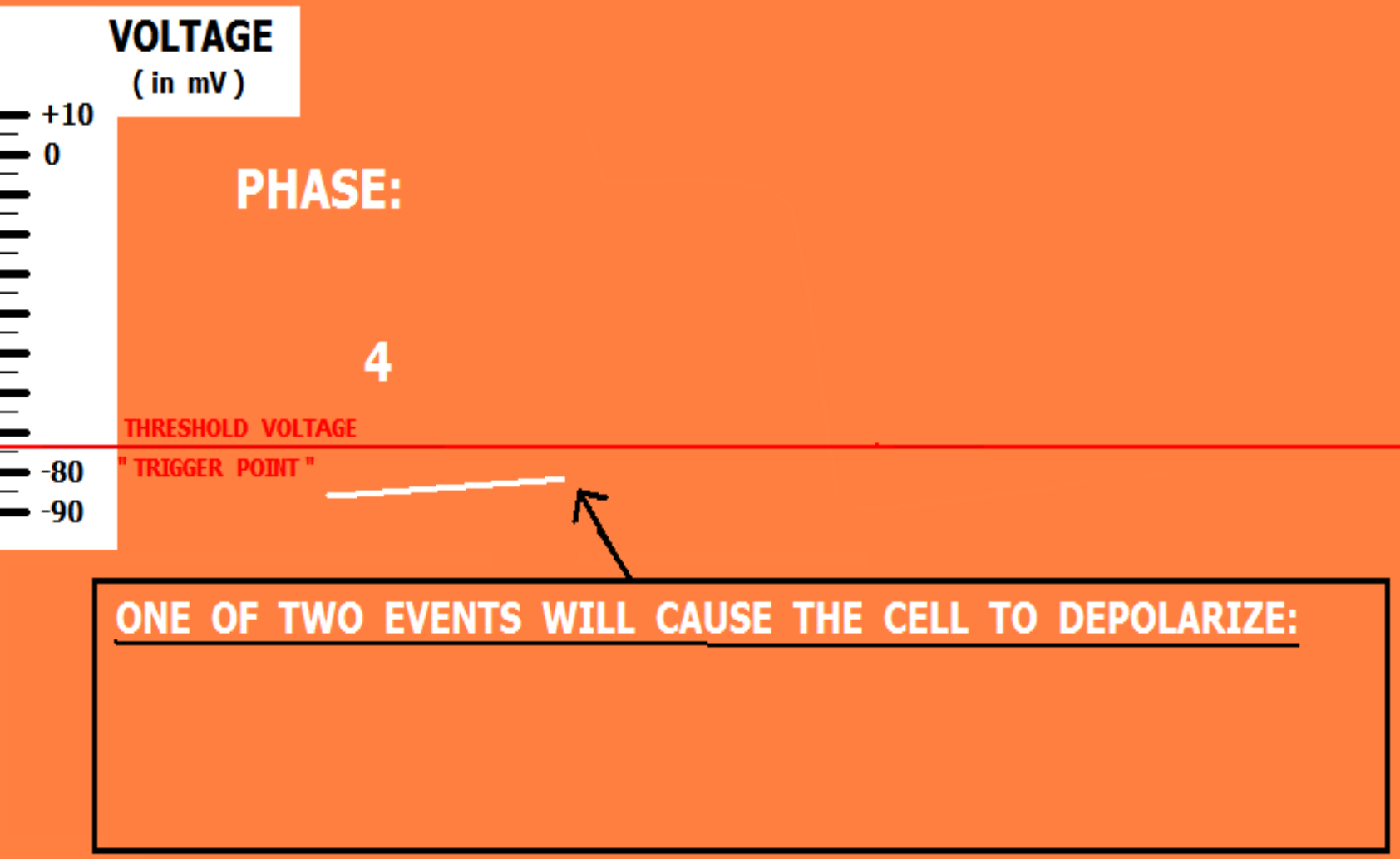
DCΩ/AC
UP
DOWN
PNP
NPN
Ω
FREQ
CAP
hFE
TEMP
TEMP (ROOM)
LOGIC
A
mA
V
mV
AUTORANGE DMM
FUSED
500V MA
MAX 30 sec EACH 15 min
20A
mA
VOLTCRAFT®

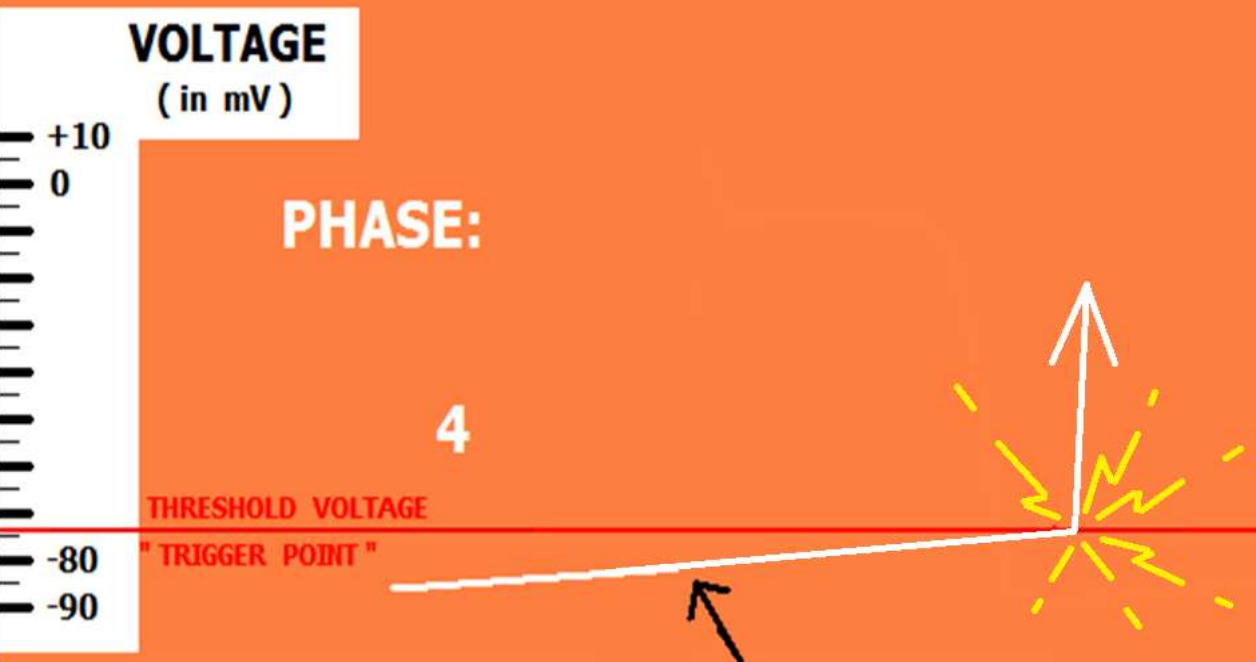
CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL



CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL

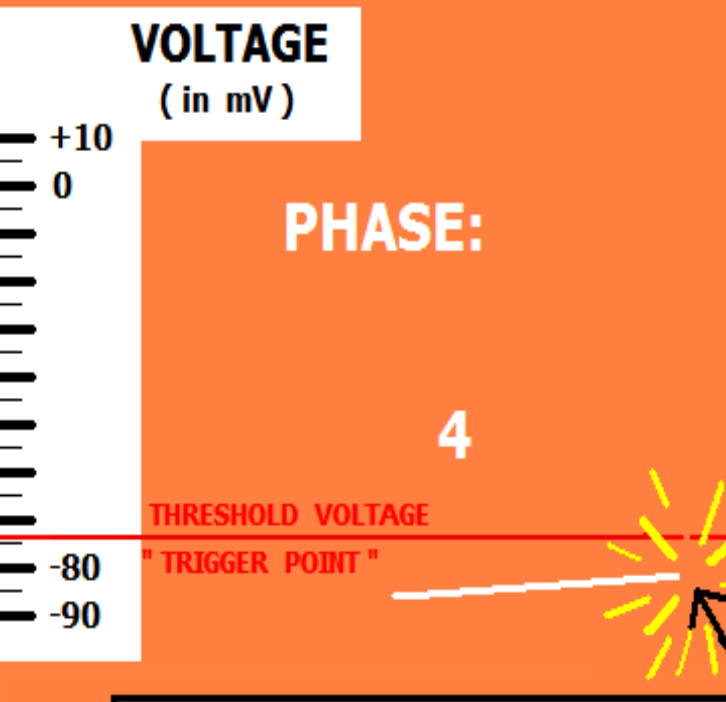


ONE OF TWO EVENTS WILL CAUSE THE CELL TO DEPOLARIZE:

Cell "leaks out" enough negative Ions that it reaches the THRESHOLD VOLTAGE (trigger point) .... and it "self-depolarizes" (automaticity)

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

PHASE:

4

THRESHOLD VOLTAGE

" TRIGGER POINT "

CELL " STATUS: "

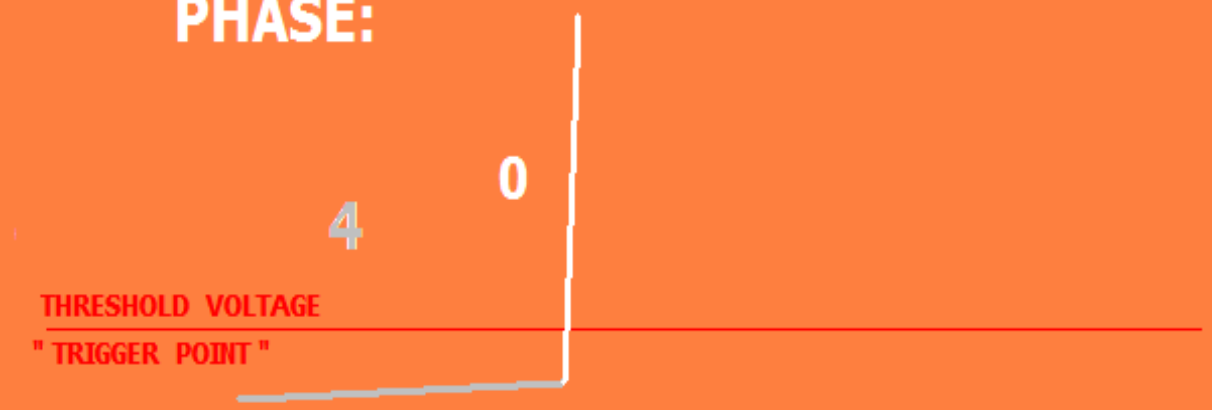
4:
- CELL COMPLETELY REPOLARIZED
- -80 to -90 mV CHARGE
- SLIGHT "LEAKAGE" OF IONS

ECG

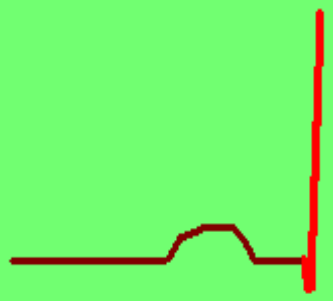
CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL

PHASE:



ECG



CELL " STATUS: "

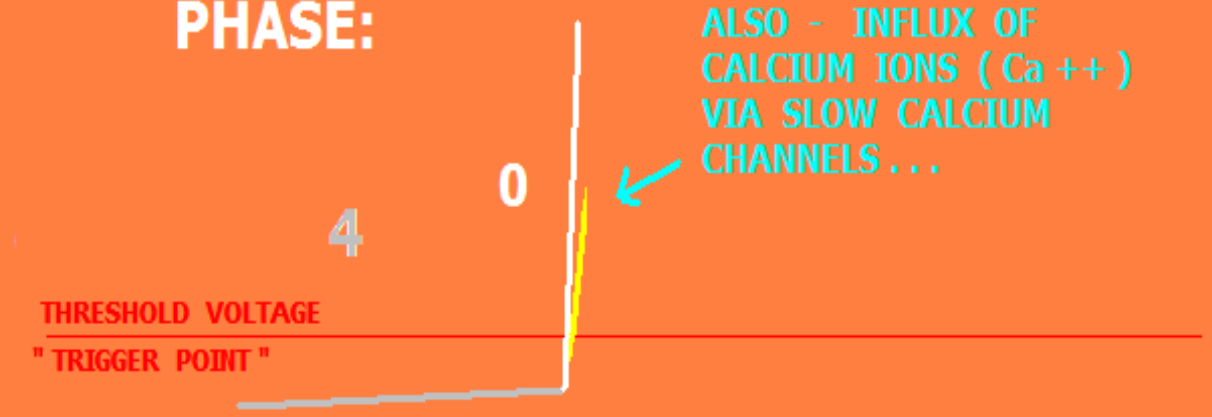
4
- CELL REPOLARIZED
- -80 to -90 mV CHARGE
- SLIGHT " LEAKAGE " OF IONS

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

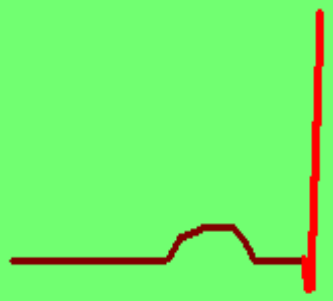
CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL

PHASE:



ECG



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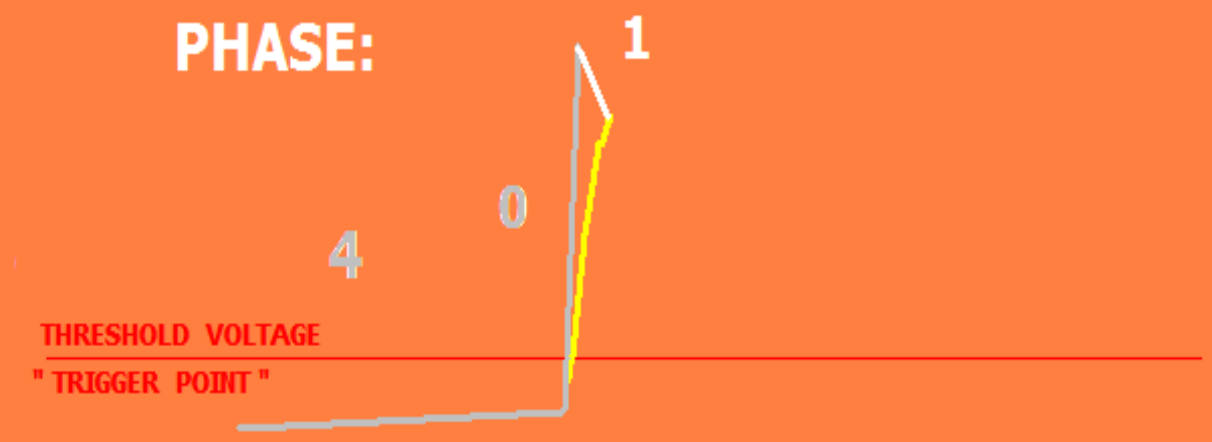
CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL

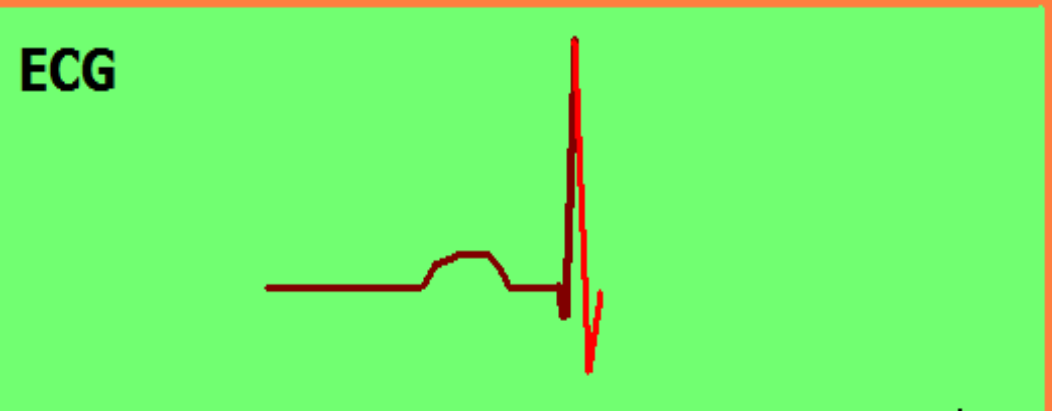


CELL " STATUS: "

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

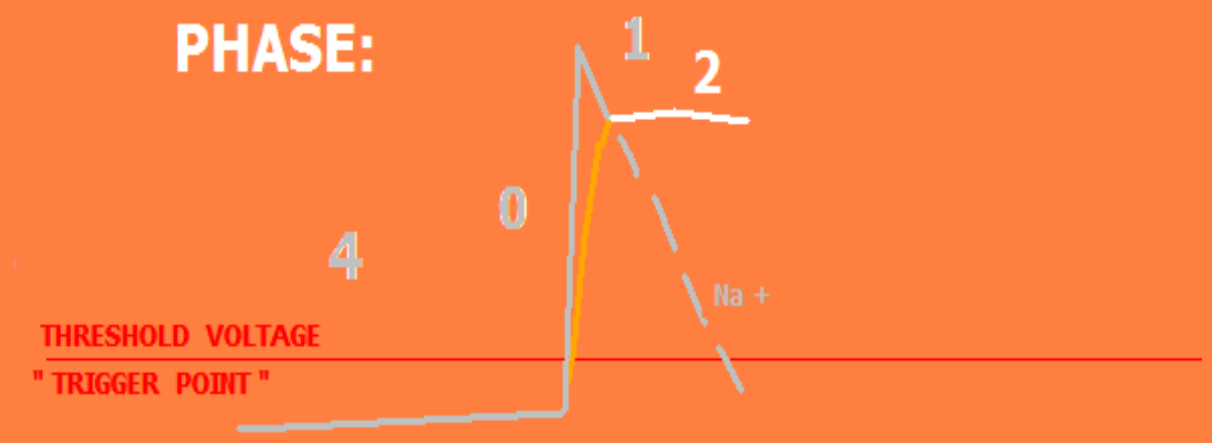
0
- RAPID INFLUX OF + CHARGED SODIUM IONS
- CELL DEPOLARIZATION

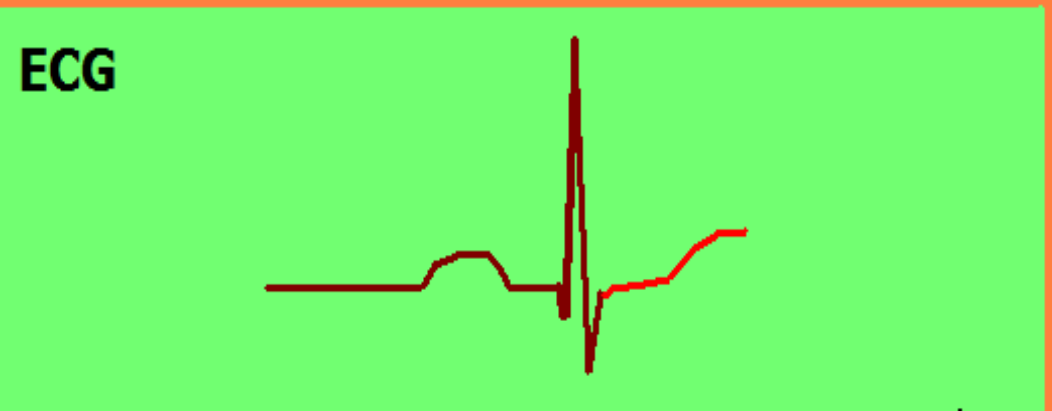
1:
- SODIUM IONS BEGIN TO EXIT THE CELL
- THIS BEGINS THE REPOLARIZATION PROCESS



CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL





CELL " STATUS: "

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- CELL REPOLARIZED
- -80 to -90 mV CHARGE
- SLIGHT " LEAKAGE " OF IONS

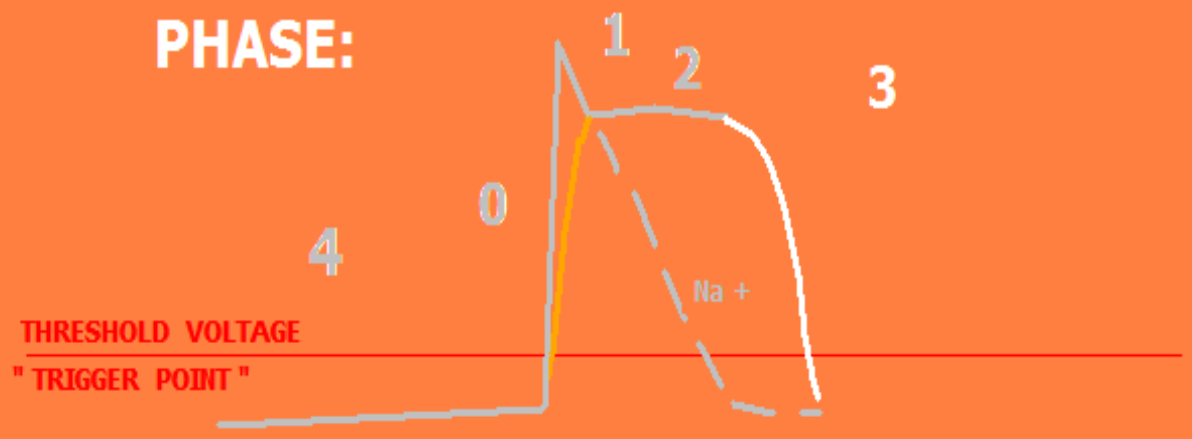
0
- RAPID INFLUX OF + CHARGED SODIUM IONS
- CELL DEPOLARIZATION

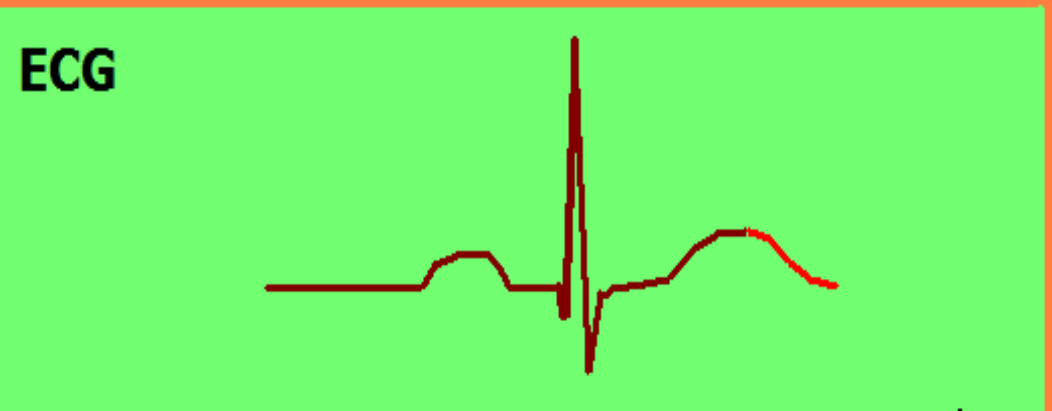
1
- SODIUM EXITS CELL
- REPOLARIZATION BEGINS

2
- CALCIUM IONS ARE COMPLETING THEIR " SLOW ENTRY " OF CARDIAC CELLS, PROLONGING THE ACTION POTENTIAL

CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL





CELL " STATUS: "

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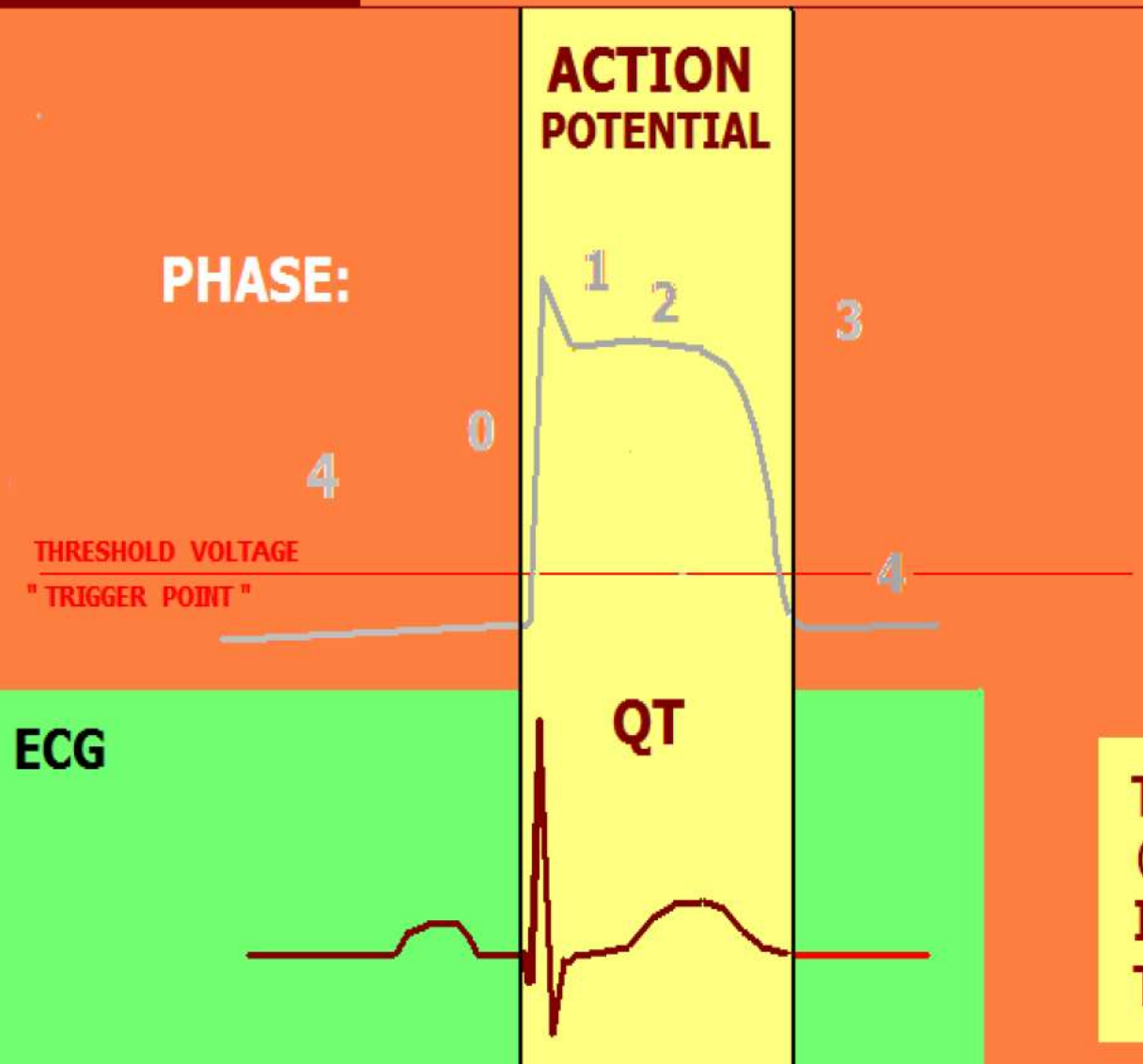
  - SLIGHT " LEAKAGE " OF IONS
- 0
  - RAPID INFLUX OF + CHARGED SODIUM IONS
  - CELL DEPOLARIZATION
- 1
  - SODIUM EXITS CELL
  - REPOLARIZATION BEGINS
- 2
  - CALCIUM IONS CONTINUE TO ENTER CELL
- 3
  - CALCIUM CHANNELS CLOSE

CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL



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  - 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 CONTINUE TO ENTER CELL
- 3
  - CALCIUM CHANNELS CLOSE

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

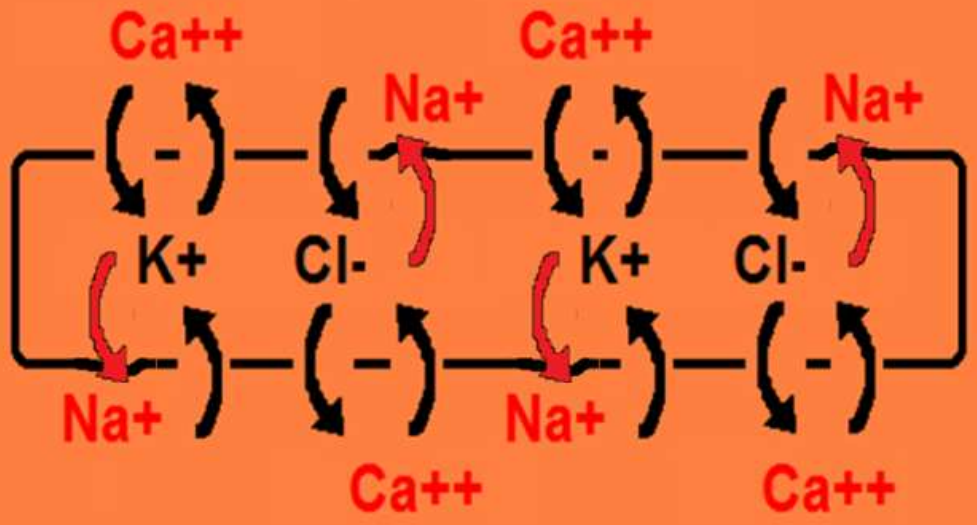
QT Prolongation

- **Congenital** malformation of ion channels
- **Medications** or other substances alter Ion channel function

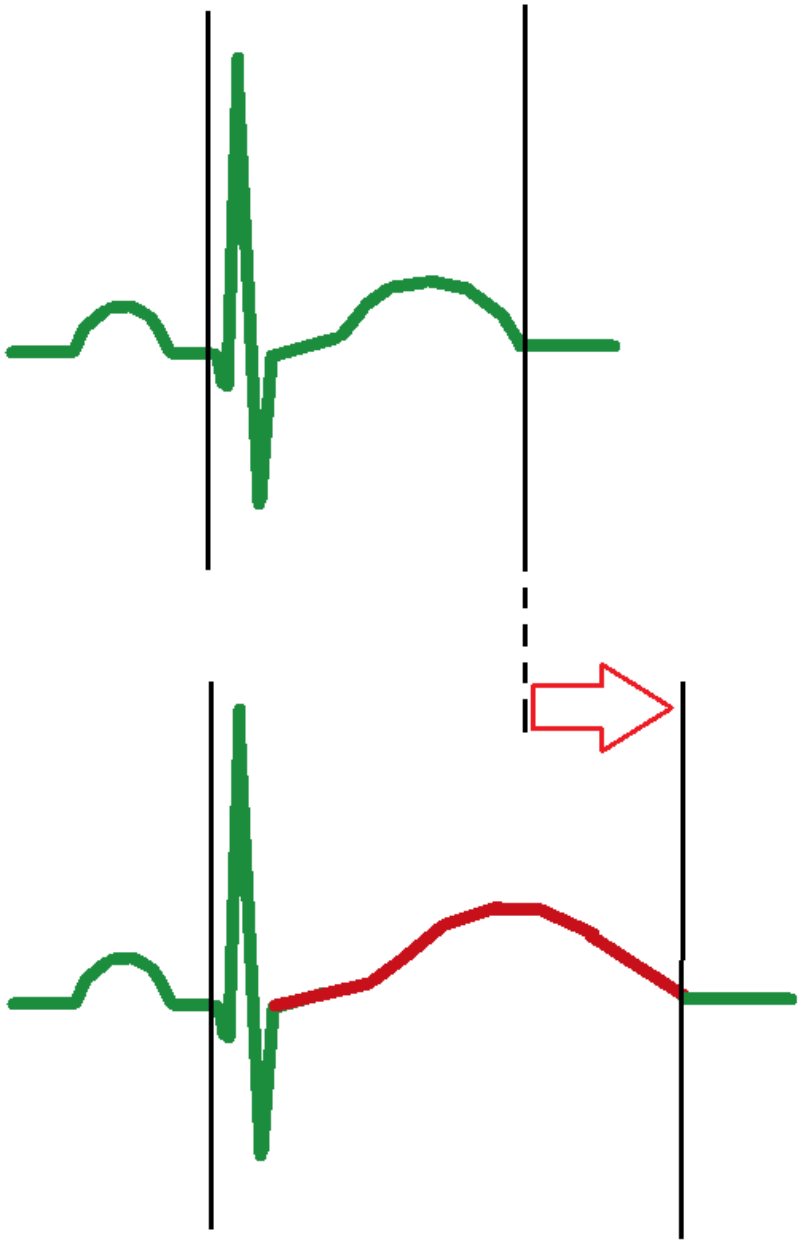
CARDIAC ANATOMY and PHYSIOLOGY "101"

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

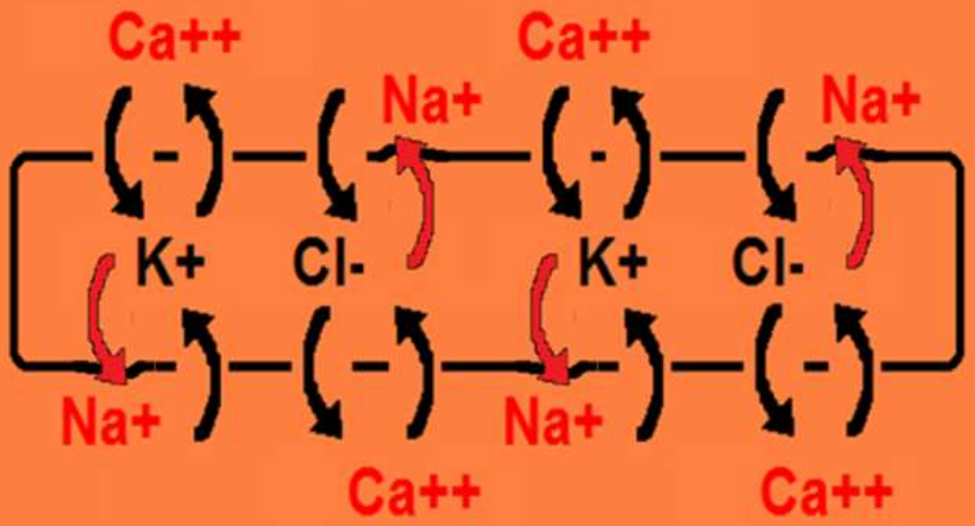


ABNORMAL
(prolonged)
QT Interval

CARDIAC ANATOMY and PHYSIOLOGY "101"

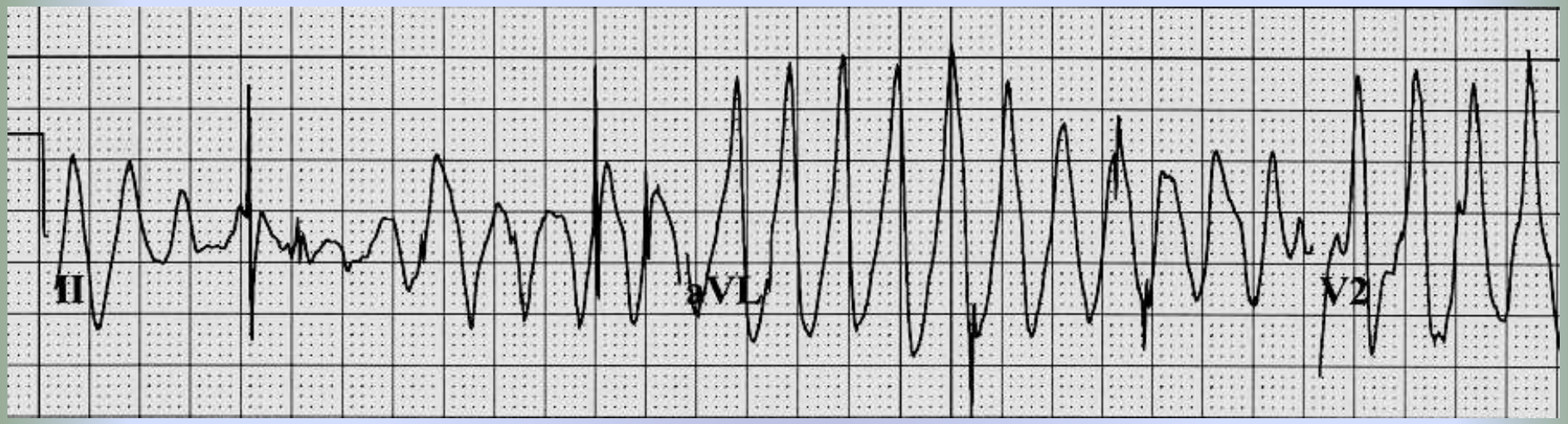
When ION CHANNELS are MALFORMED, the abnormal channel shape may DELAY the transfer of IONS . . . . .

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



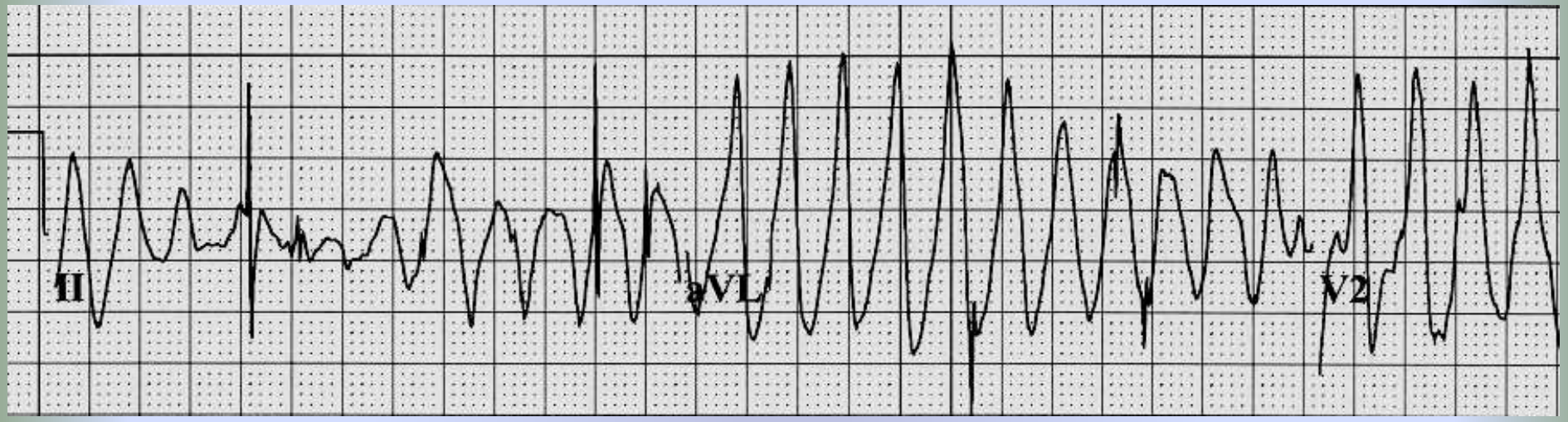
which can lead to Torsades . . . Cardiac Arrest . . . and *SUDDEN DEATH.*

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)



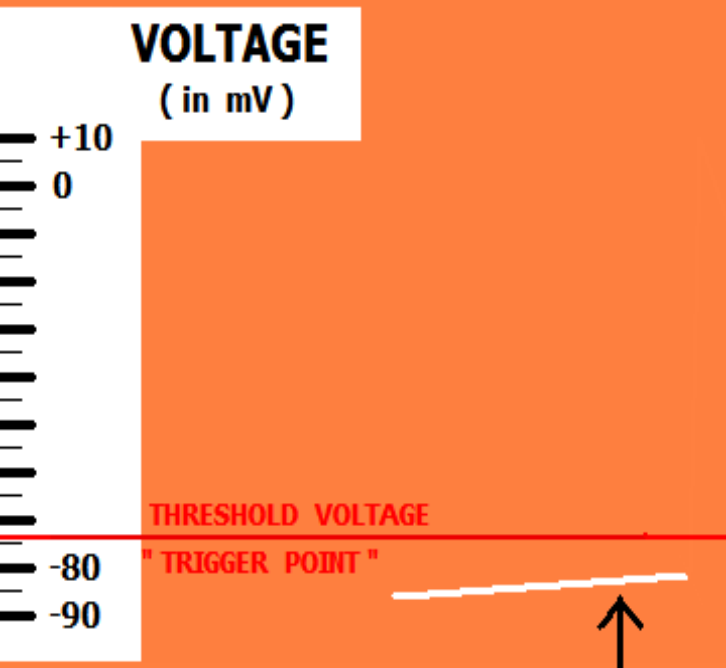
- Common cause: QTc > 600 ms
- Patients typically have little to no cardiac output when in this rhythm
- TdP may self-terminate or deteriorate into **VENTRICULAR FIBRILLATION**

Pacemaker Cells

- When cardiac cells **self-depolarize**

CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL



DURING PHASE 4, CELLS " LEAK OUT " SOME OF THEIR NEGATIVELY CHARGED IONS, CAUSING THE INSIDE OF THE CELL TO HAVE A LESS-NEGATIVE CHARGE . . .

CARDIAC ANATOMY and PHYSIOLOGY "101"

VENTRICULAR MUSCLE CELL ACTION POTENTIAL

PHASE:

4

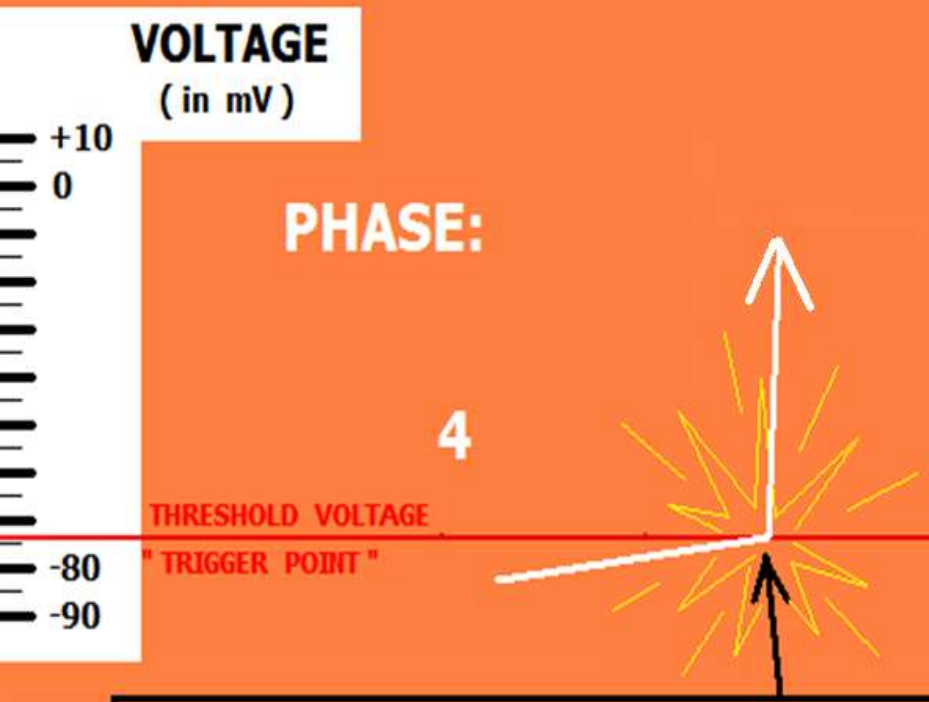
THRESHOLD VOLTAGE
" TRIGGER POINT "

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



THIS IS KNOWN AS ***AUTOMATICITY***

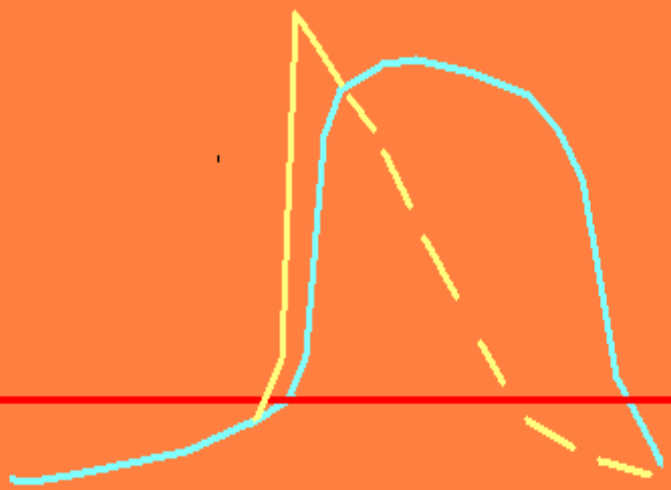
ONE OF TWO EVENTS WILL CAUSE THE CELL TO DEPOLARIZE:

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 ") CAUSING THE CELL TO DEPOLARIZE ITSELF . . . and then in turn, it's neighbors

CARDIAC ANATOMY and PHYSIOLOGY "101"

COMPONENTS OF ACTION POTENTIAL WAVEFORM

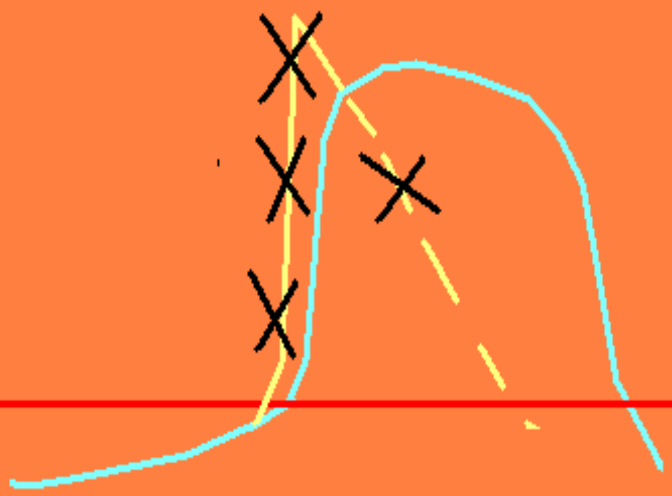
CARDIAC PACEMAKER CELLS
(SINUS NODE and A-V NODE)



CARDIAC ANATOMY and PHYSIOLOGY "101"

COMPONENTS OF ACTION POTENTIAL WAVEFORM

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

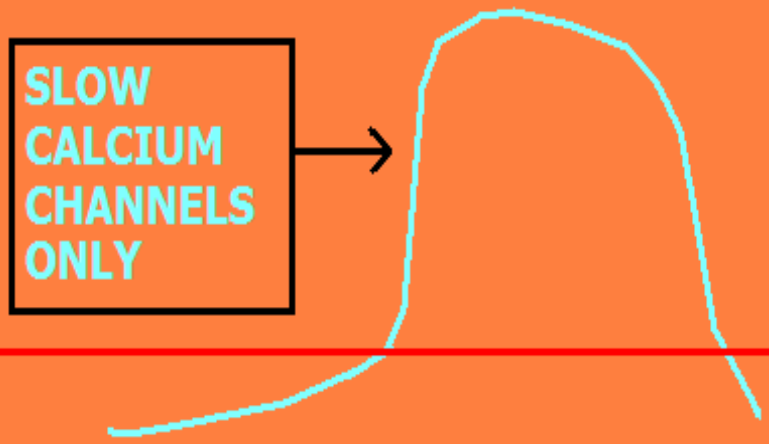


DO NOT HAVE FAST SODIUM CHANNELS . . .

CARDIAC ANATOMY and PHYSIOLOGY "101"

COMPONENTS OF ACTION POTENTIAL WAVEFORM

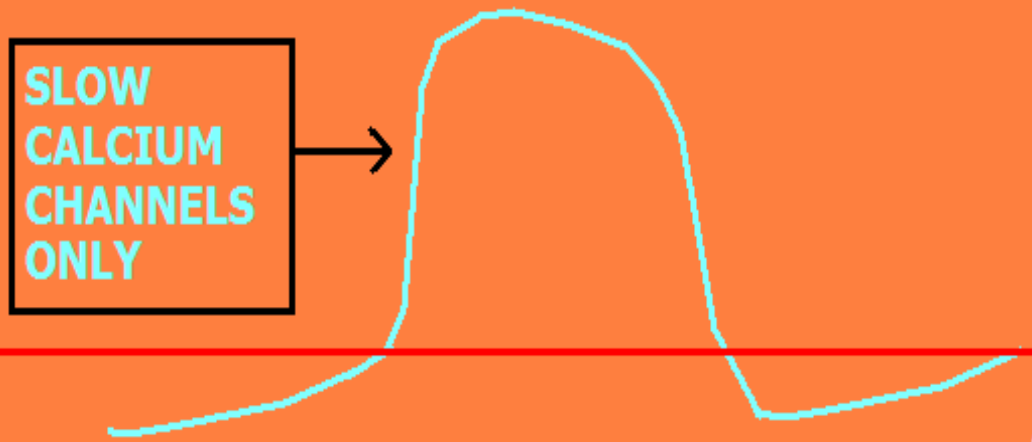
CARDIAC PACEMAKER CELLS
(SINUS NODE and A-V NODE)



CARDIAC ANATOMY and PHYSIOLOGY "101"

COMPONENTS OF ACTION POTENTIAL WAVEFORM

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



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

CARDIAC ANATOMY and PHYSIOLOGY "101"

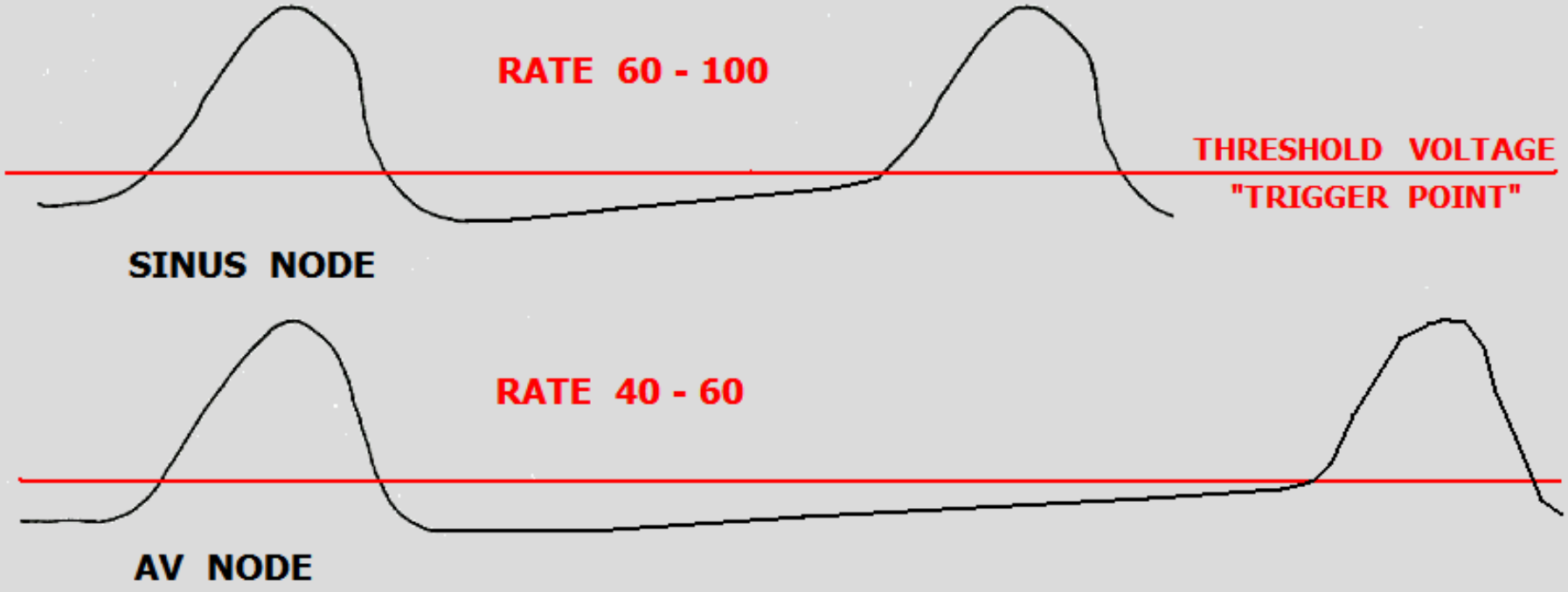
COMPONENTS OF ACTION POTENTIAL WAVEFORM

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

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

DIFFERENCES IN ACTION POTENTIAL IN DIFFERENT TYPES OF HEART CELLS

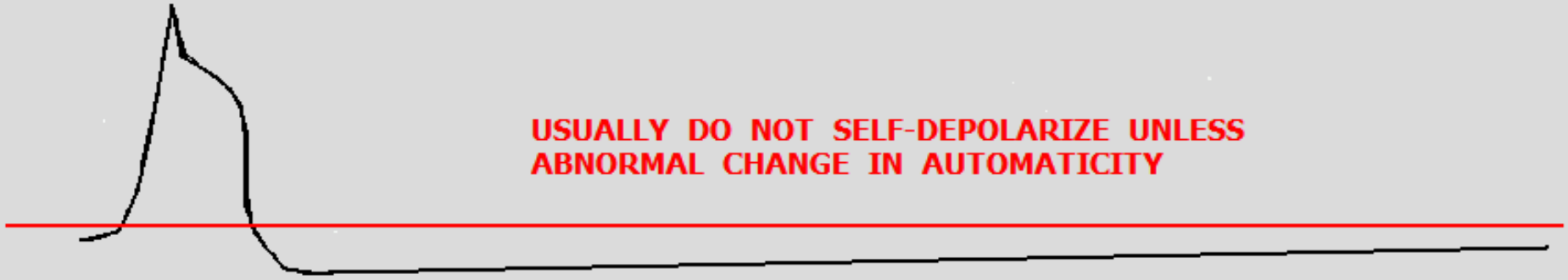
CARDIAC PACEMAKER CELLS



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

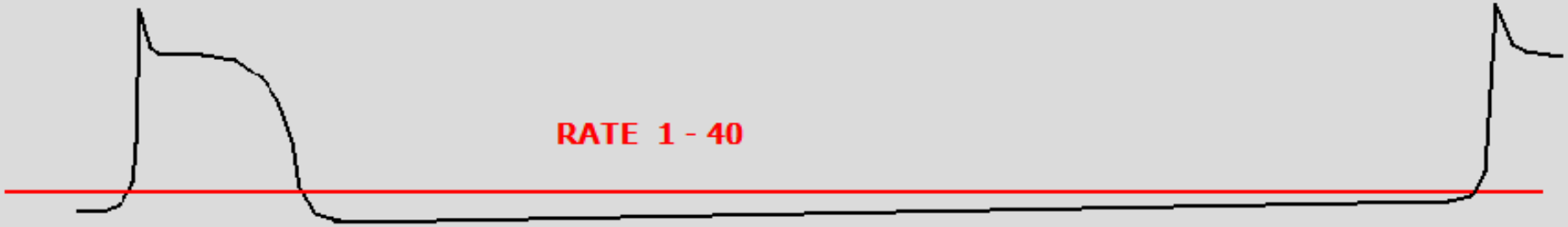
DIFFERENCES IN ACTION POTENTIAL IN DIFFERENT TYPES OF HEART CELLS

MUSCLE and PURKINJE FIBER ACTION POTENTIALS



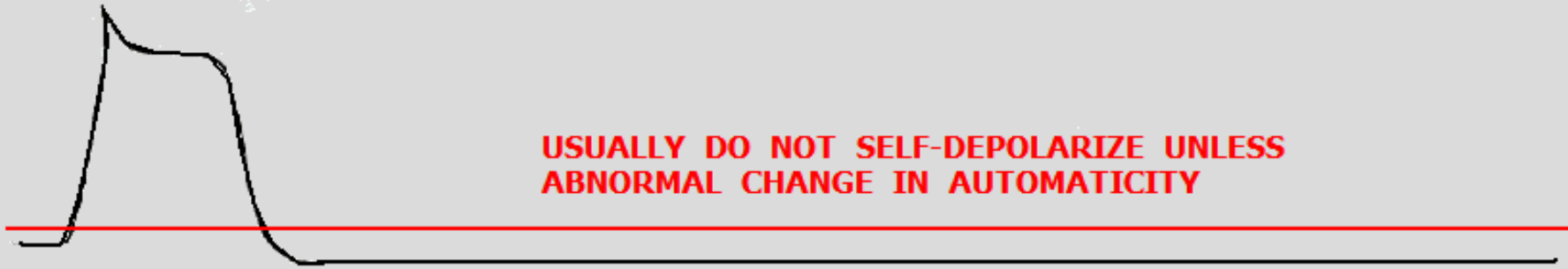
USUALLY DO NOT SELF-DEPOLARIZE UNLESS
ABNORMAL CHANGE IN AUTOMATICITY

ATRIAL MUSCLE



RATE 1 - 40

PURKINJE FIBER



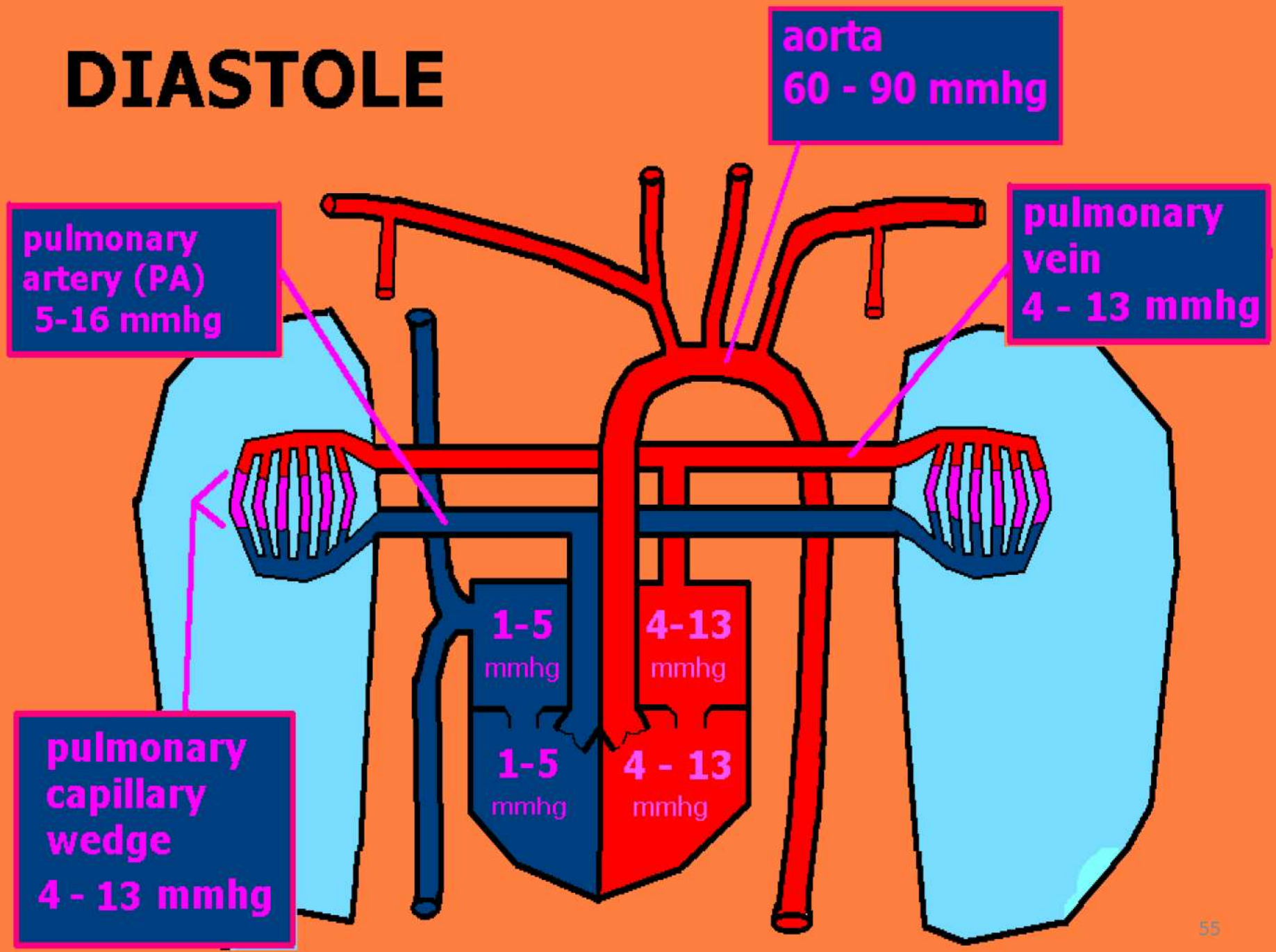
USUALLY DO NOT SELF-DEPOLARIZE UNLESS
ABNORMAL CHANGE IN AUTOMATICITY

VENTRICULAR MUSCLE

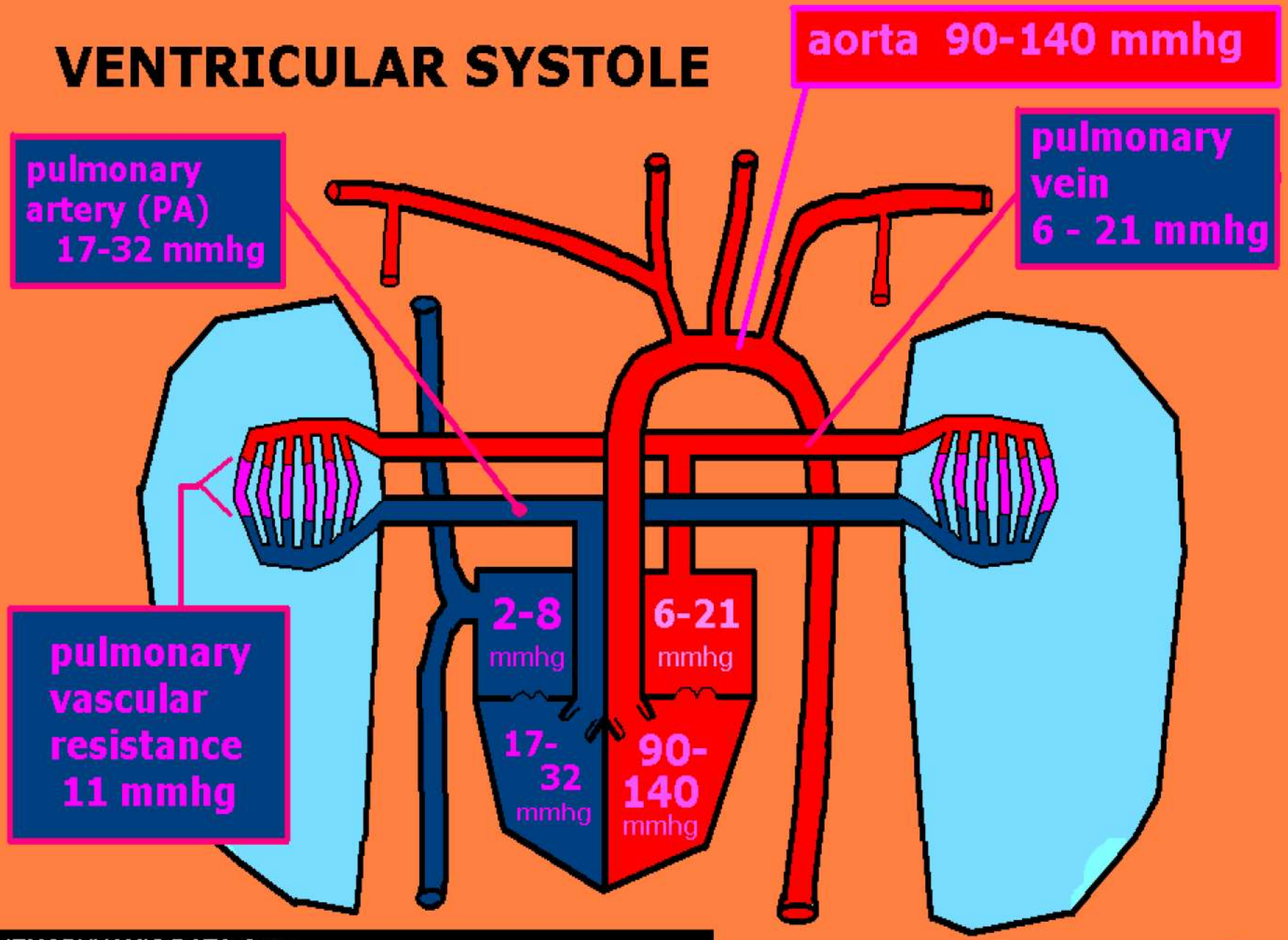
“Cardiac A & P 101”

- Heart Chambers
- Heart Electrical System & ECG Waveforms
- Depolarization and Repolarization
- **Normal Pressures within Heart & Lungs**

DIASTOLE


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

VENTRICULAR SYSTOLE

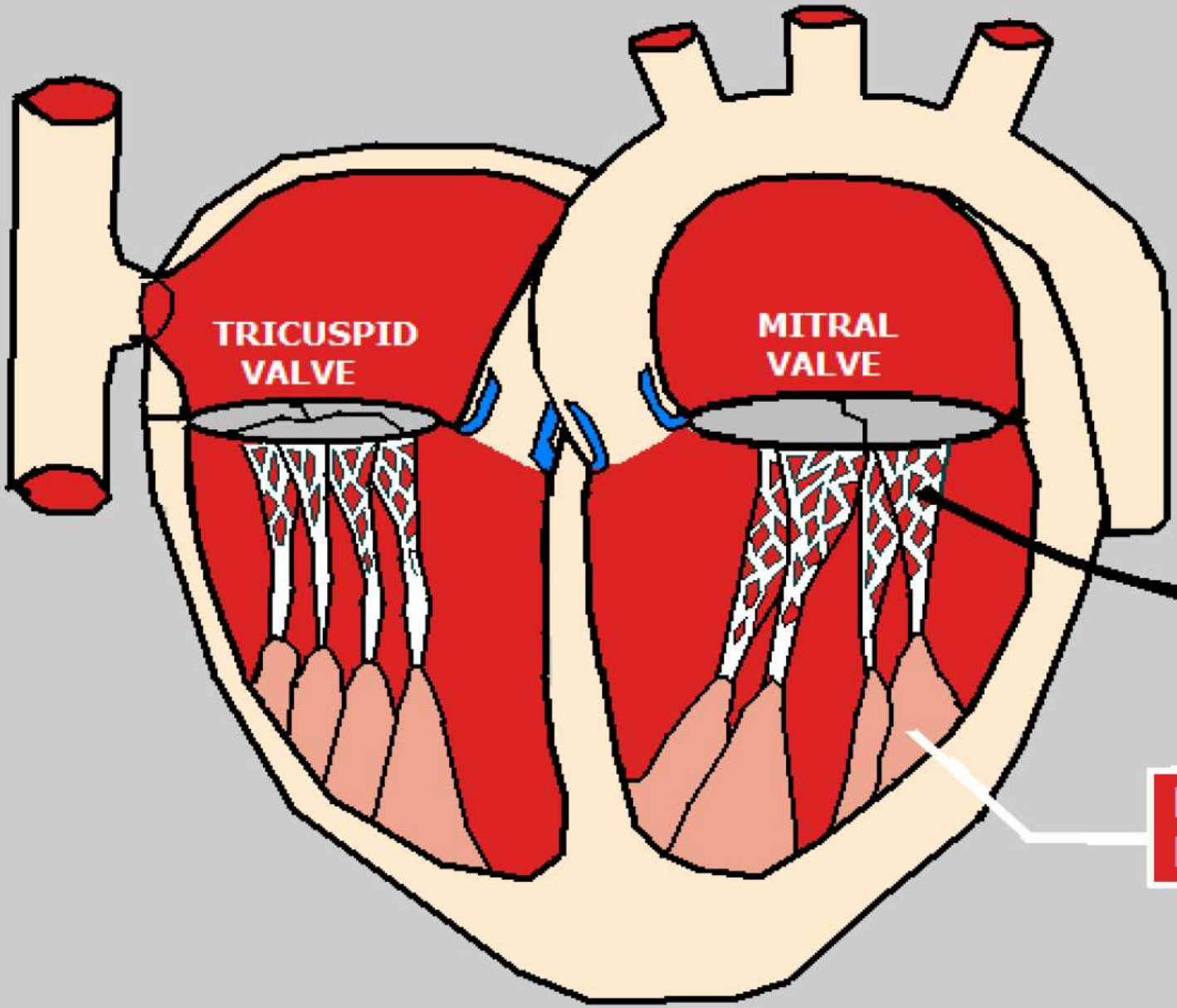


HEMODYNAMIC DATA from:
"The Cardiac Catheterization Handbook," Morton J. Kearn, MD

“Cardiac A & P 101”

- Heart Chambers
- Heart Electrical System & ECG Waveforms
- Action Potential
- Normal Pressures within Heart & Lungs
- **Heart Valves**

ATRIO-VENTRICULAR VALVES



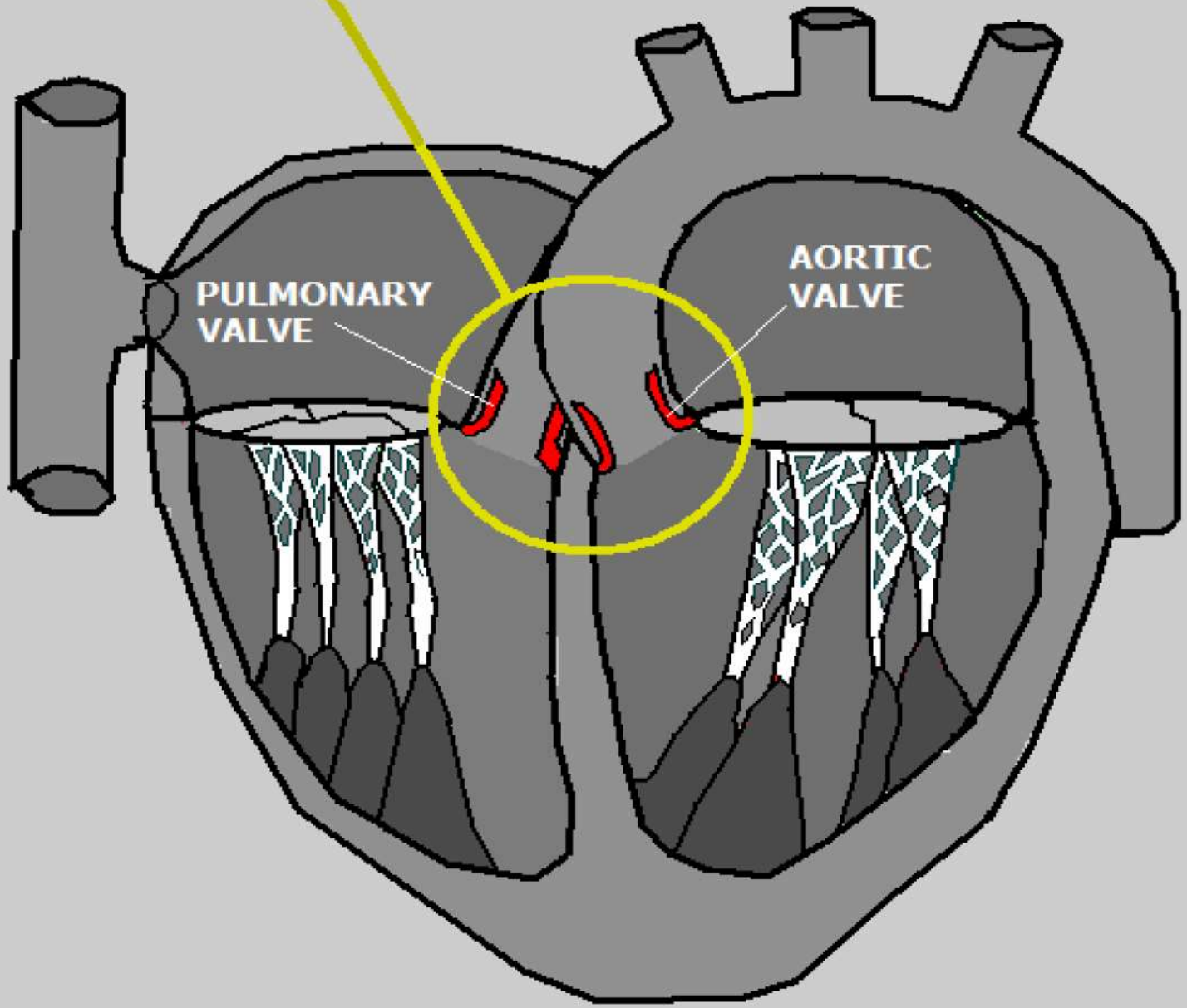
ARE SECURED TO THE ENDO-CARDIAL SURFACE BY THE

CHORDAE TENDINEAE

AND THE

PAPILLARY MUSCLES

THE SEMILUNAR VALVES



“Cardiac A & P 101”

- Heart Chambers
- Heart Electrical System & ECG Waveforms
- Depolarization and Repolarization
- Normal Pressures within Heart & Lungs
- Heart Valves
- **Heart Sounds Overview**

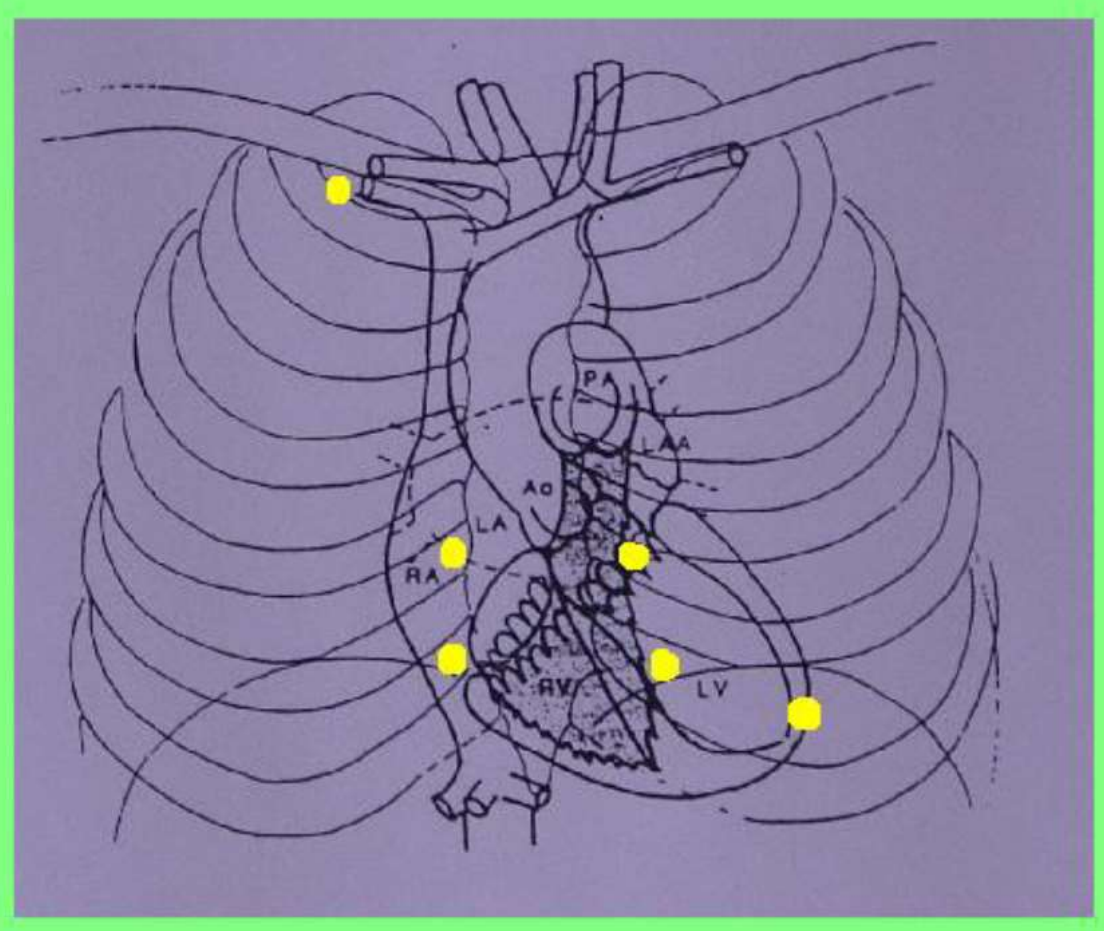
VERY BASIC HEART SOUNDS ASSESSMENT

ABNORMAL EKG CHANGES THAT MAY PRESENT WITH ABNORMAL HEART SOUNDS:

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

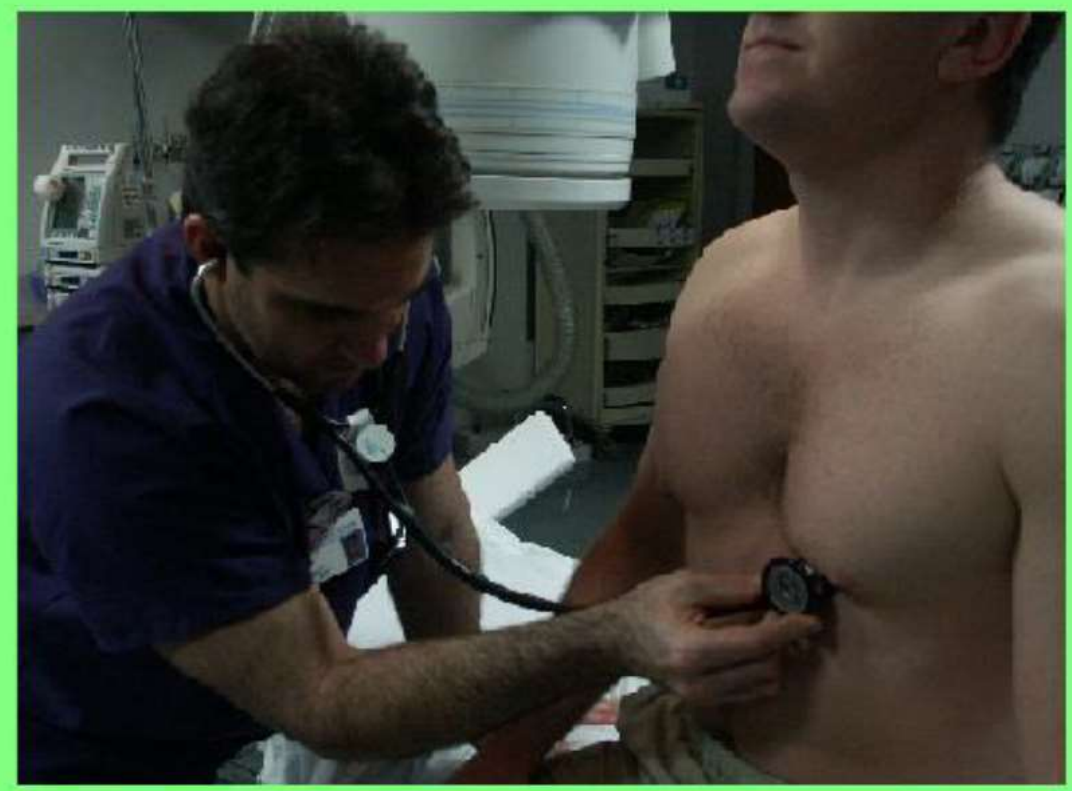
HEART SOUNDS ASSESSMENT

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.

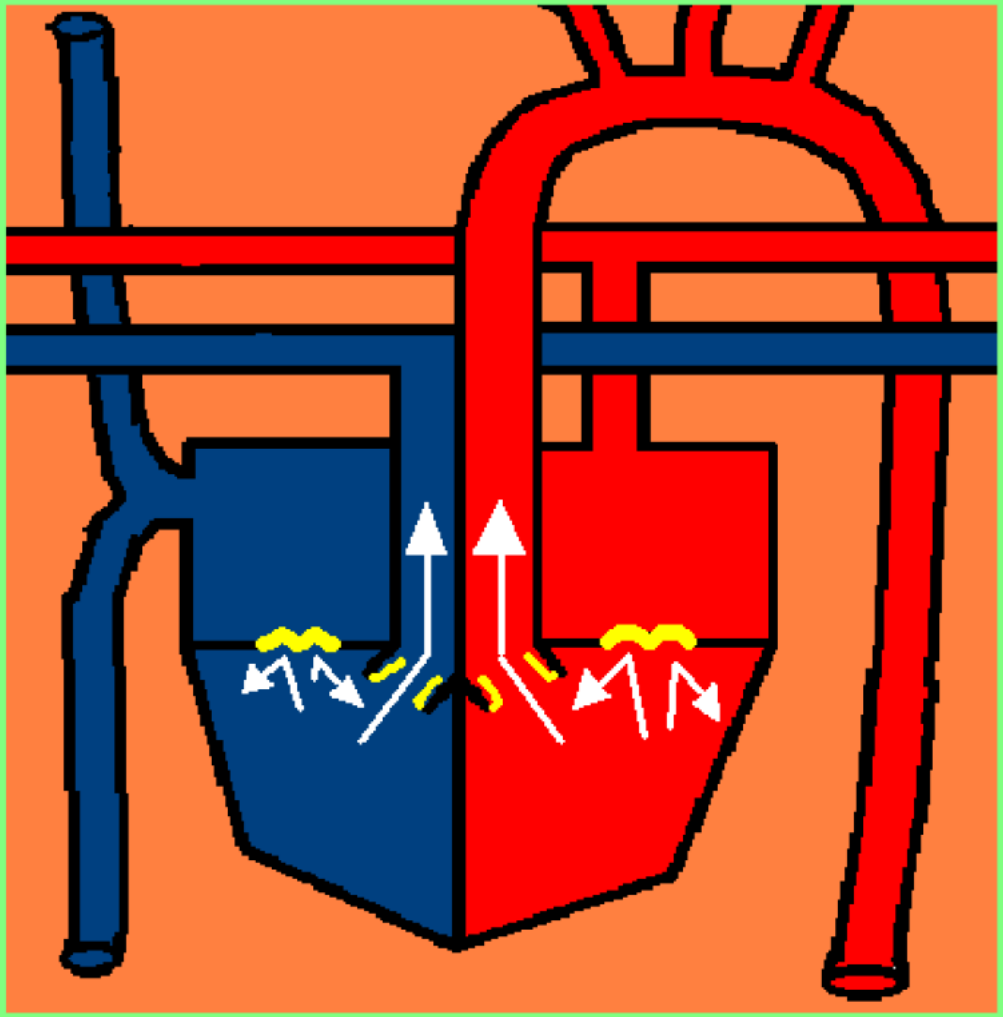
THERE ARE TWO NORMAL HEART SOUNDS, KNOWN AS: S-1 and S-2

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

HEART SOUNDS ASSESSMENT

S-1 BEGINNING OF SYSTOLE.

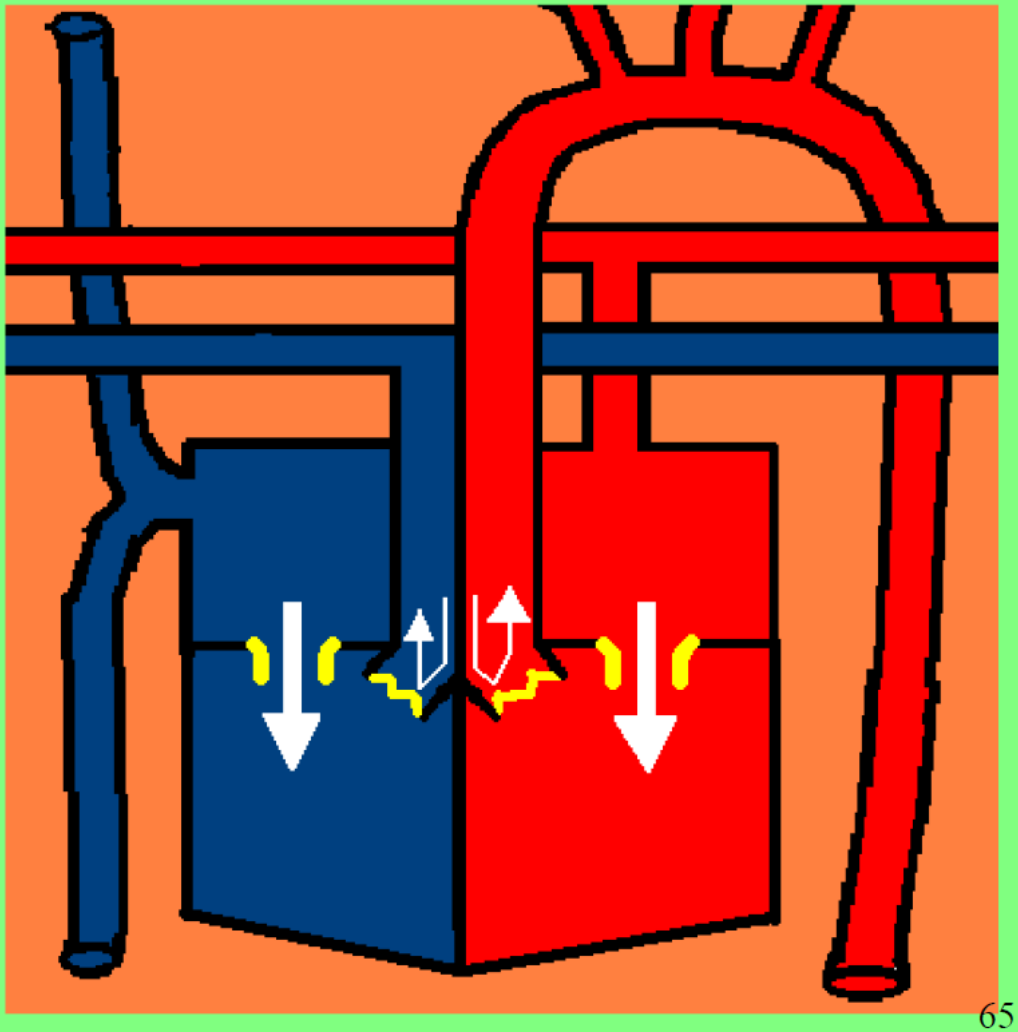
SOUND OF THE MITRAL AND TRICUSPID VALVES CLOSING.



HEART SOUNDS ASSESSMENT

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

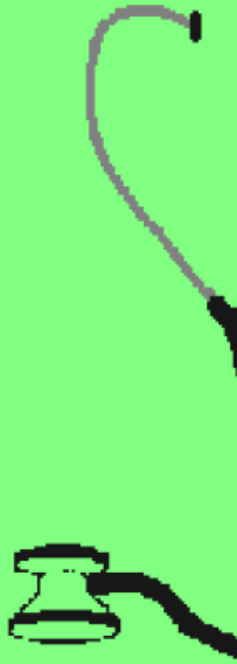
IT IS THE SOUND OF THE AORTIC AND PULMONARY VALVES CLOSING.



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

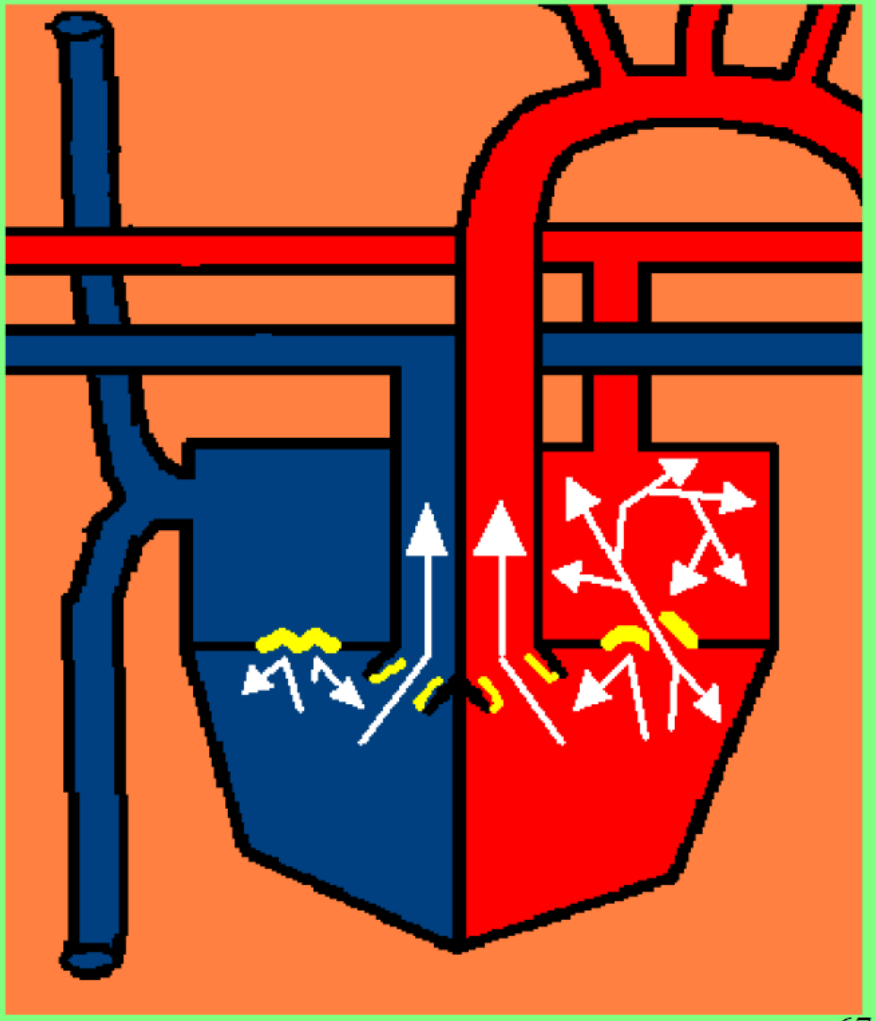
S-1 MURMUR SOUNDS LIKE:

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



CAUSE OF SYSTOLIC (S 1) MURMUR

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



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

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

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

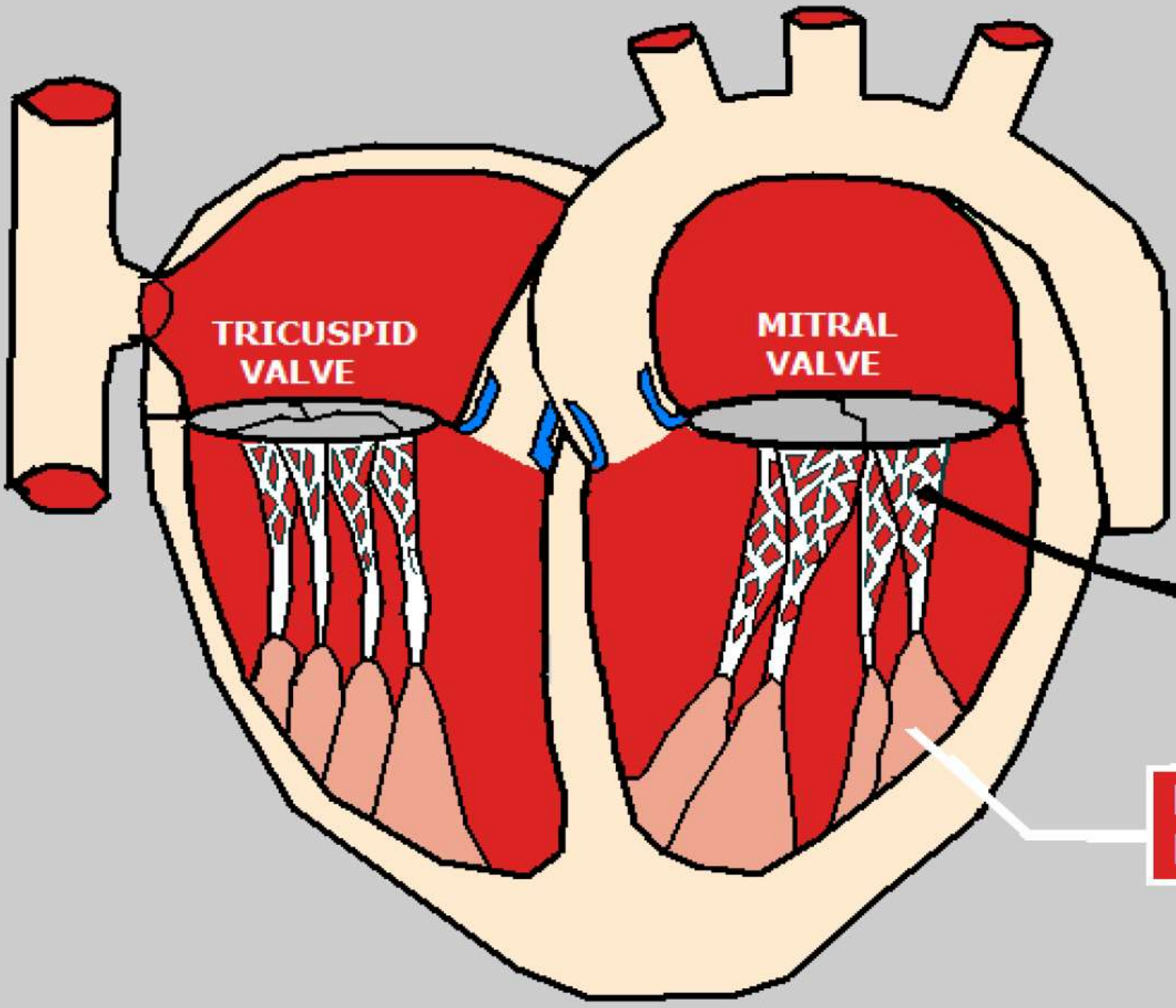
ACUTE Mitral Valve REGURGITATION can be caused by EXTENSIVE "Multi-Site" Myocardial Infarction and Necrosis – which results in PAPILLARY MUSCLE NECROSIS and PAPILLARY MUSCLE TEAR.

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

A Common Cause of ACUTE MITRAL REGURGITATION is:

"Patients who are 7-10 days POST-EXTENSIVE MI," in cases where the "zone of infarction" is large (e.g. "inferior-posterior-lateral") and there was a delay in PCI resulting in large zone of necrosis.

ATRIO-VENTRICULAR VALVES



ARE SECURED TO THE ENDO-CARDIAL SURFACE BY THE

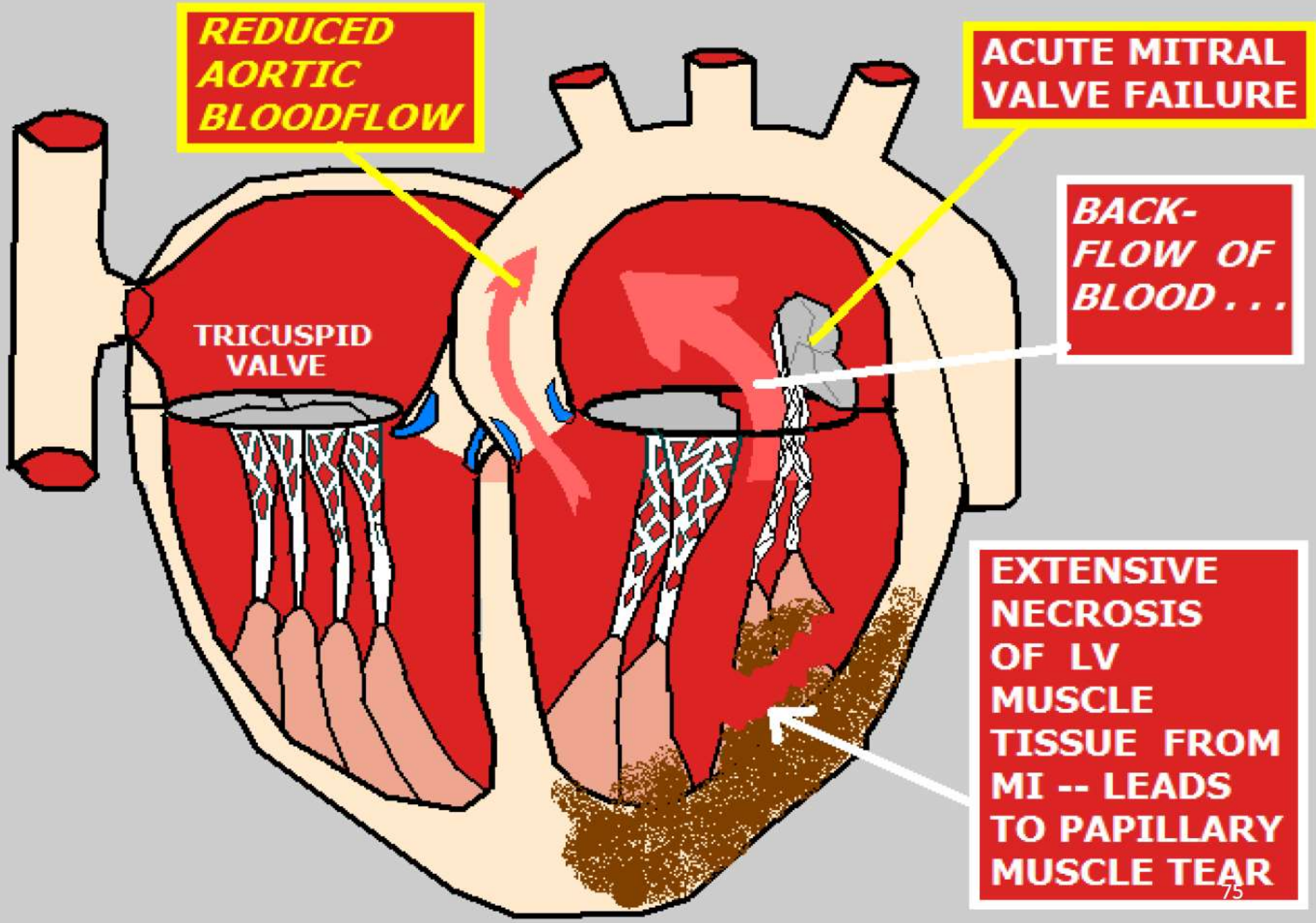
CHORDAE TENDINEAE

AND THE

PAPILLARY MUSCLES

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

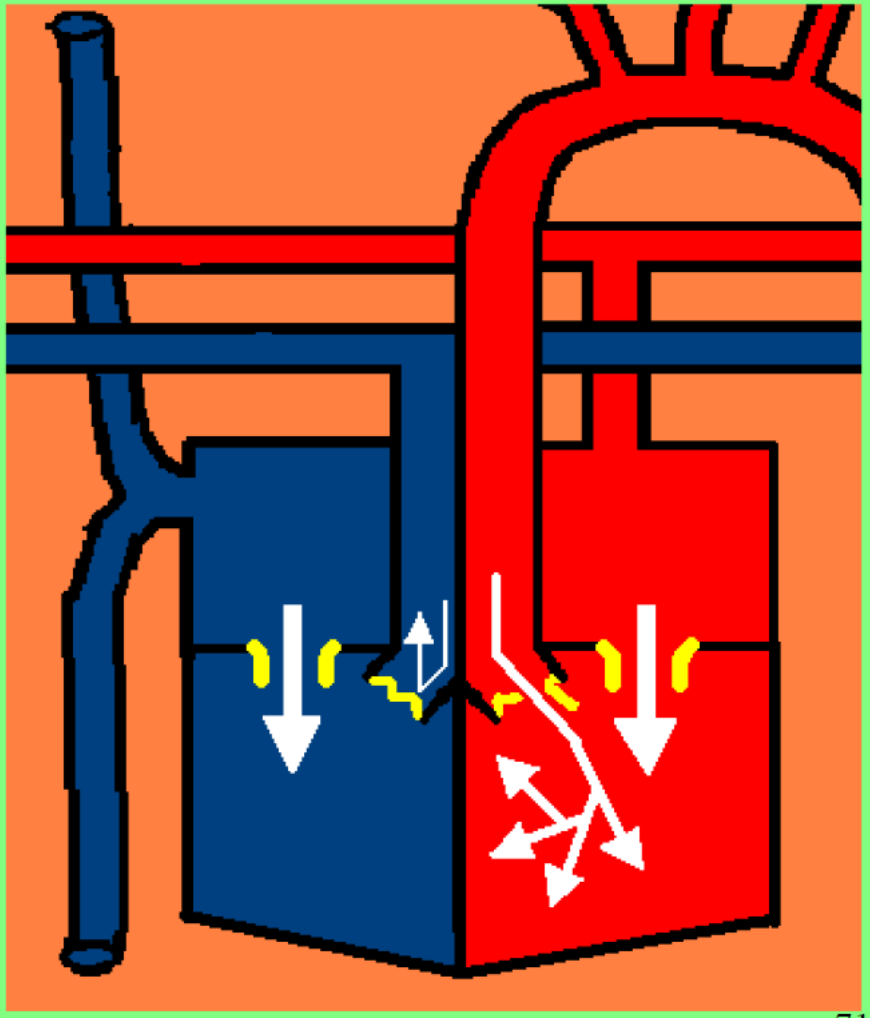
**MURMUR = "SWOOSH"
SOUND CAUSED BY THE
SOUND OF TURBULENCE.**

S-2 MURMUR SOUNDS LIKE:

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

CAUSE OF DIASTOLIC (S2) 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.**

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

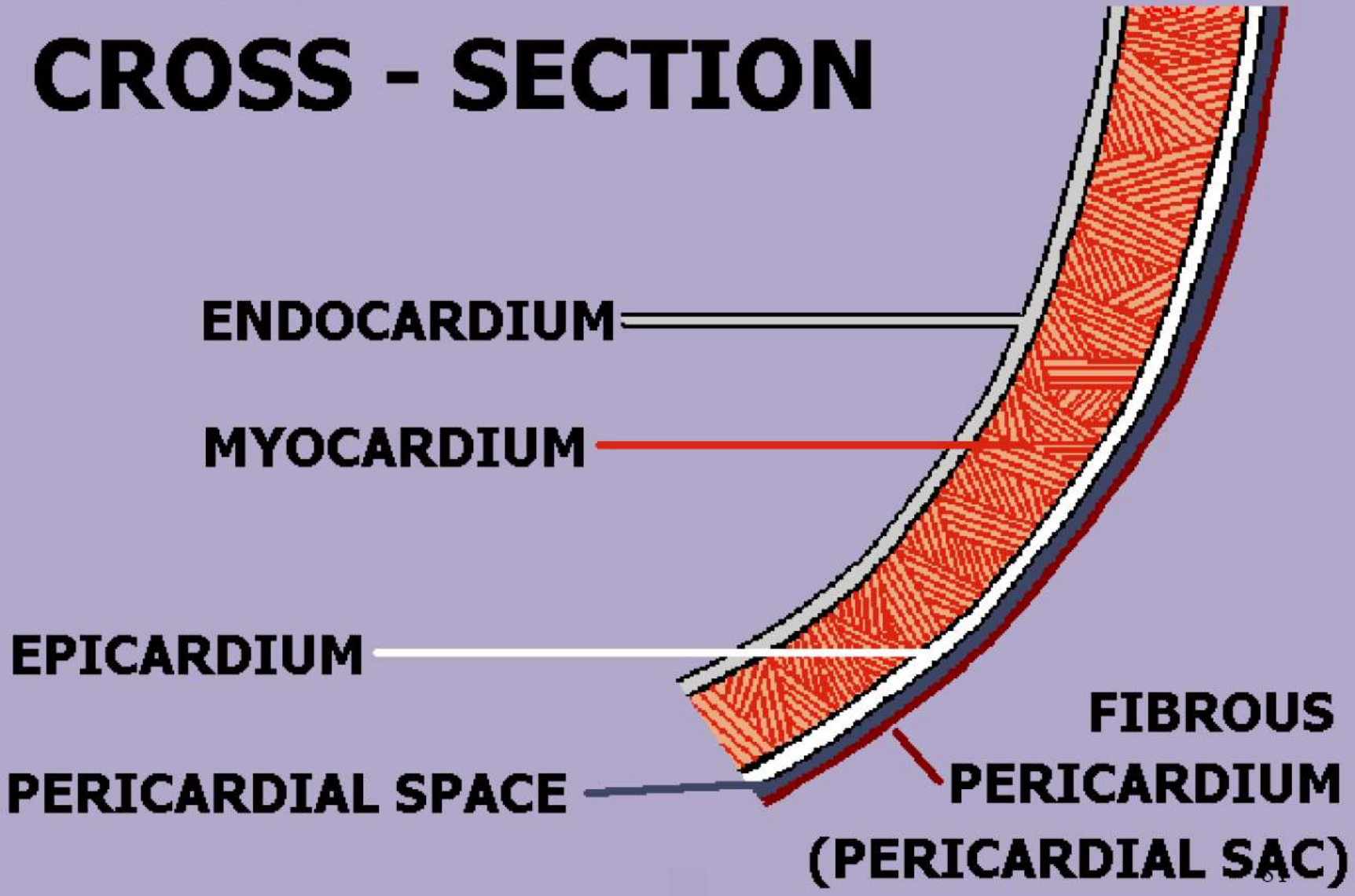
- ASSOCIATED WITH PERICARDITIS
- SOUNDS LIKE THE GENTLE RUBBING OF SANDPAPER
- HAS 3 COMPONENTS: SYSTOLIC, EARLY, and LATE DIASTOLIC

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



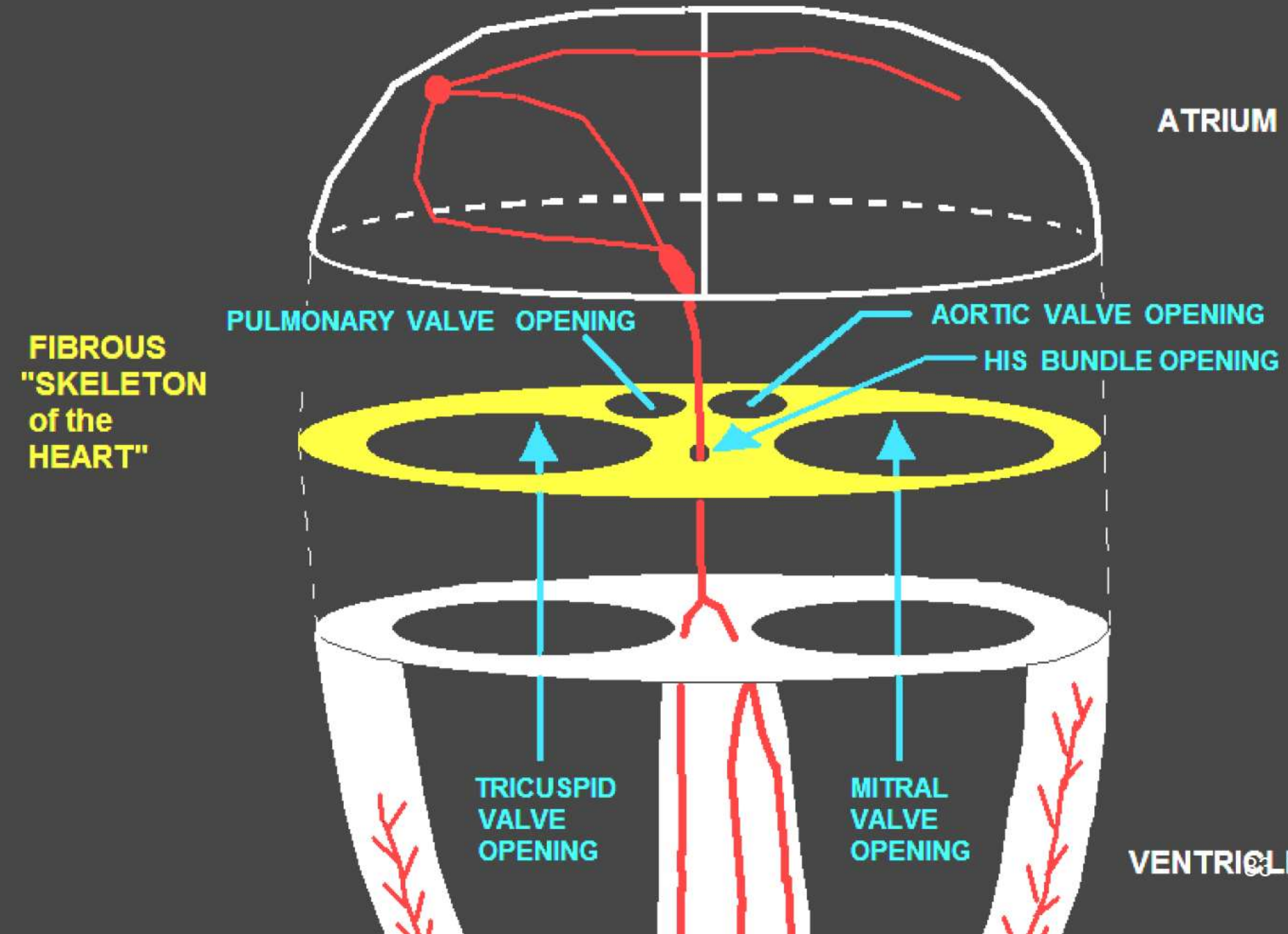
NORMAL AMOUNT OF FLUID IN PERICARDIAL SPACE =

20 - 50 cc

“Cardiac A & P 101”

- Heart Chambers
- Heart Electrical System & ECG Waveforms
- Depolarization and Repolarization
- Normal Pressures within Heart & Lungs
- Heart Valves
- Heart Sounds Overview
- **Fibrous Skeleton of Heart**

THE "SKELETON OF THE HEART"

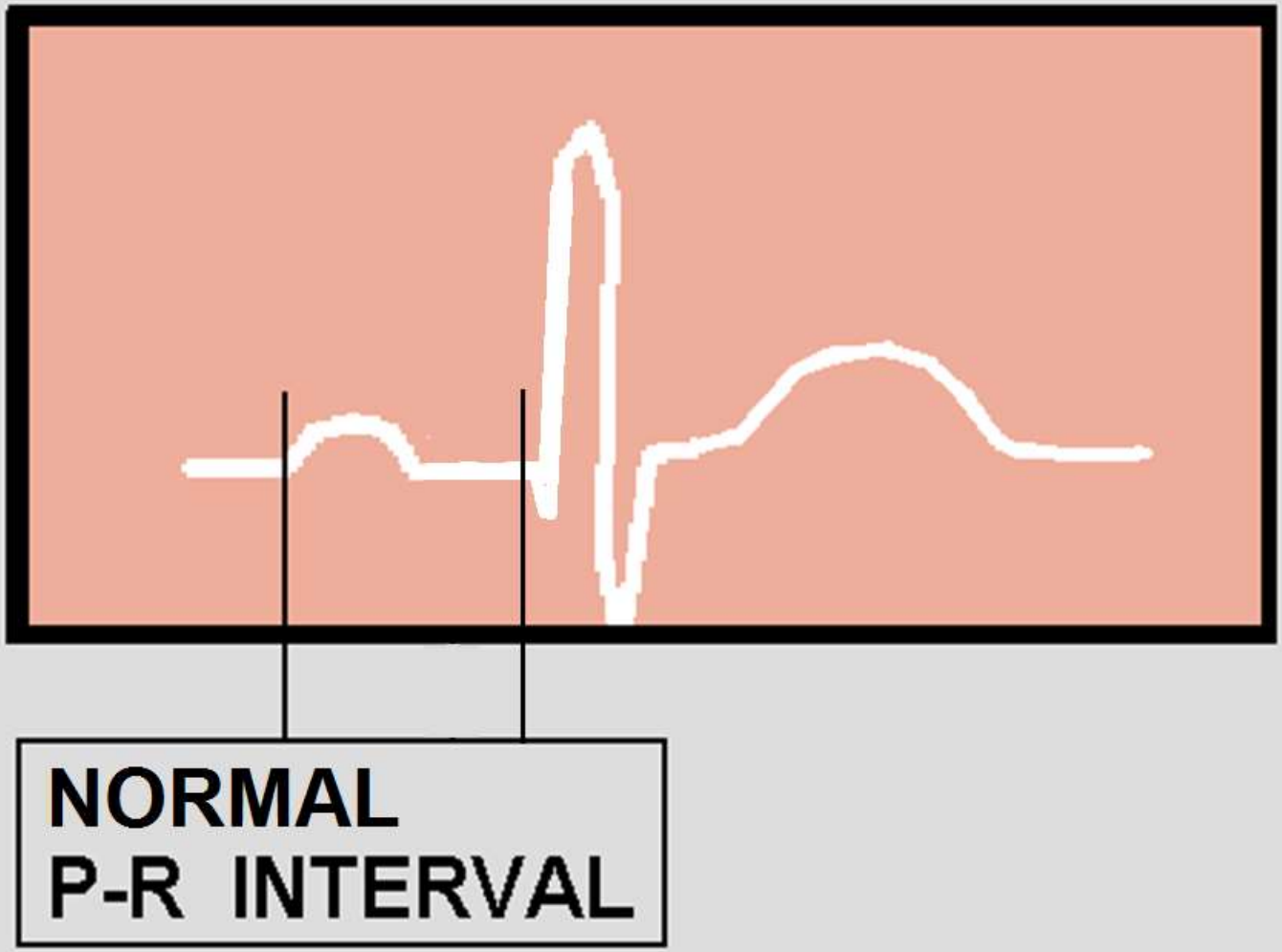


Fibrous Skeleton of the Heart…

- 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**

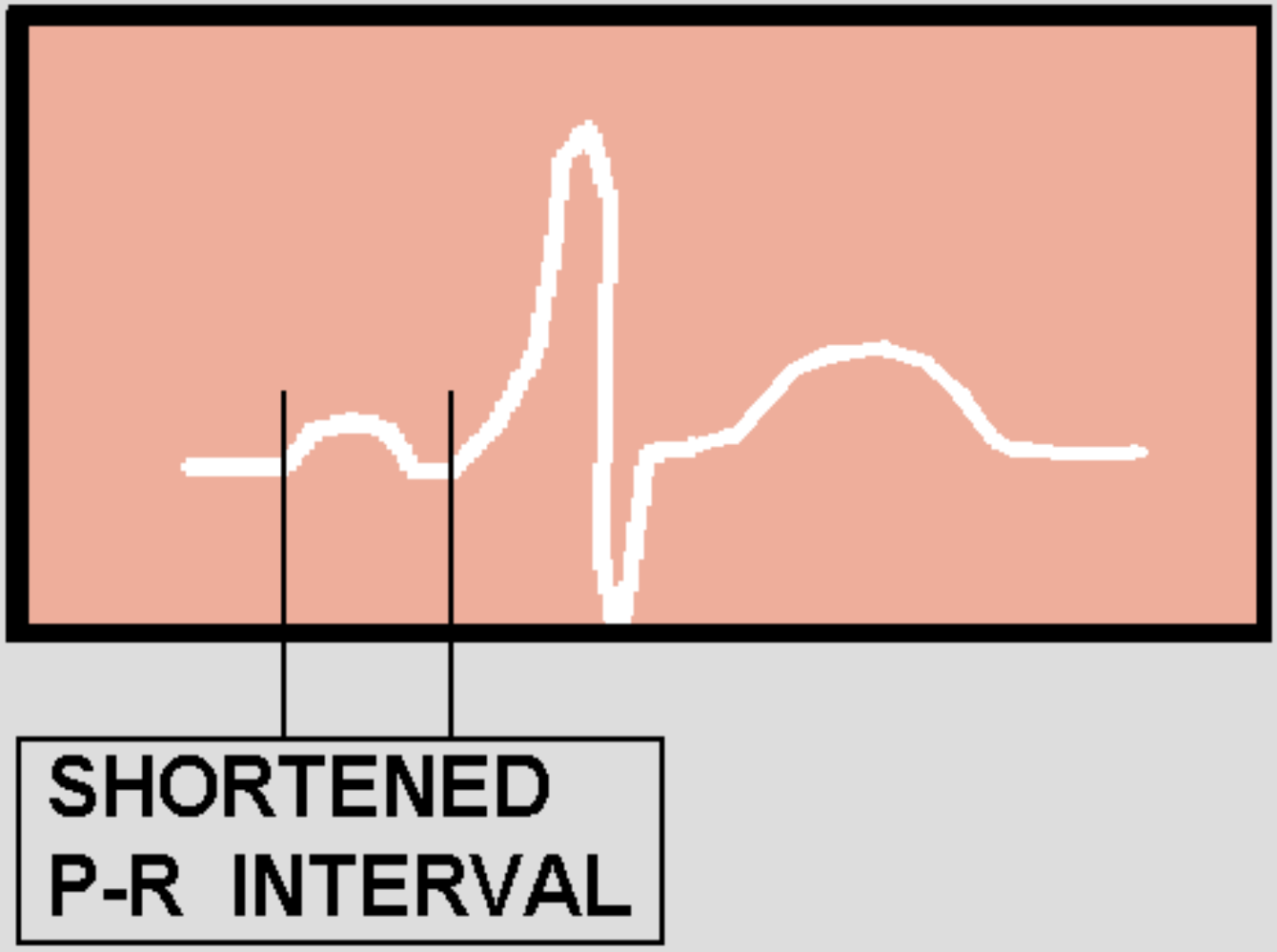
WOLFF-PARKINSON-WHITE

THE NORMAL ECG . . . .



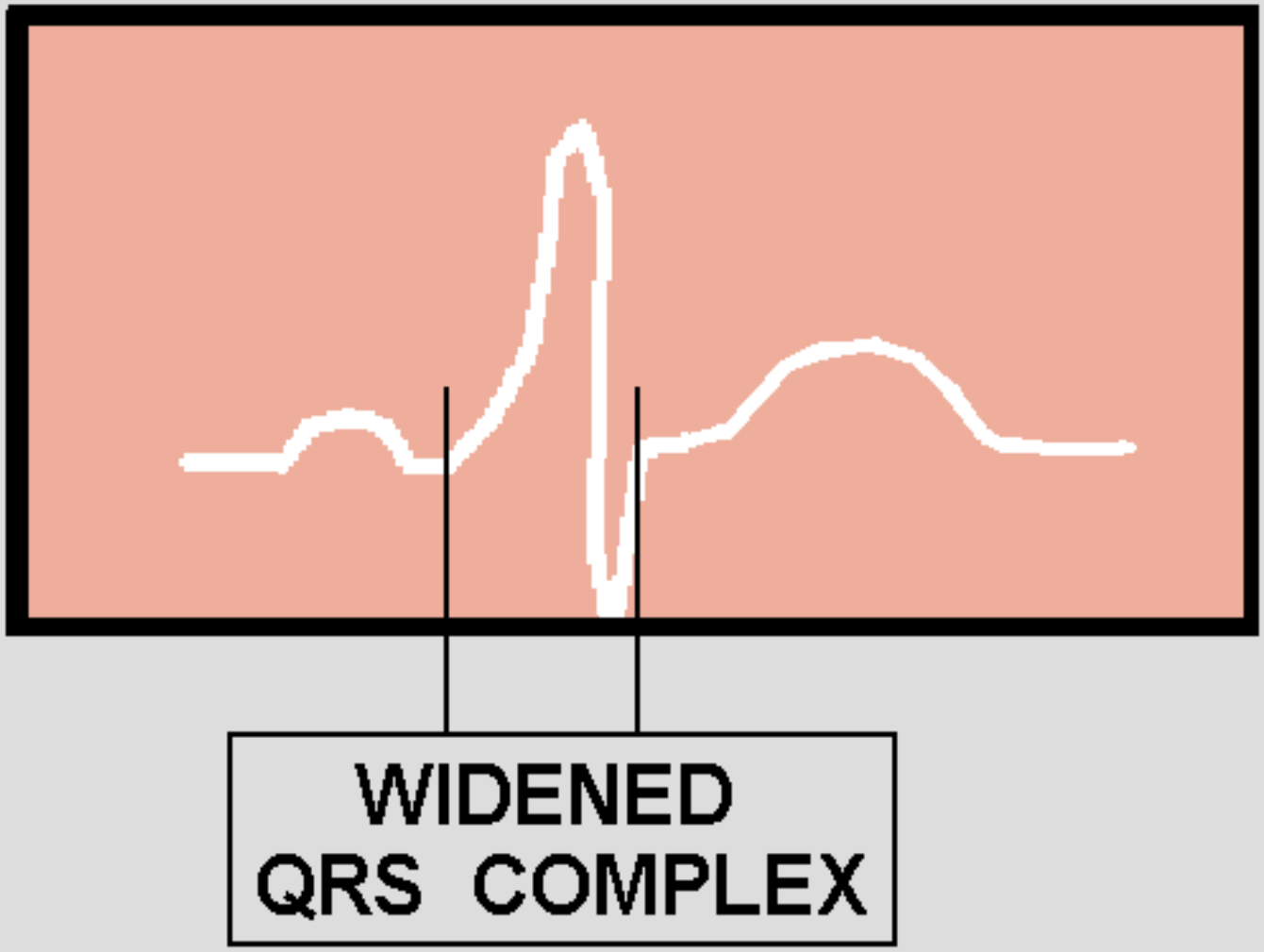
WOLFF-PARKINSON-WHITE

EKG CHARACTERISTICS



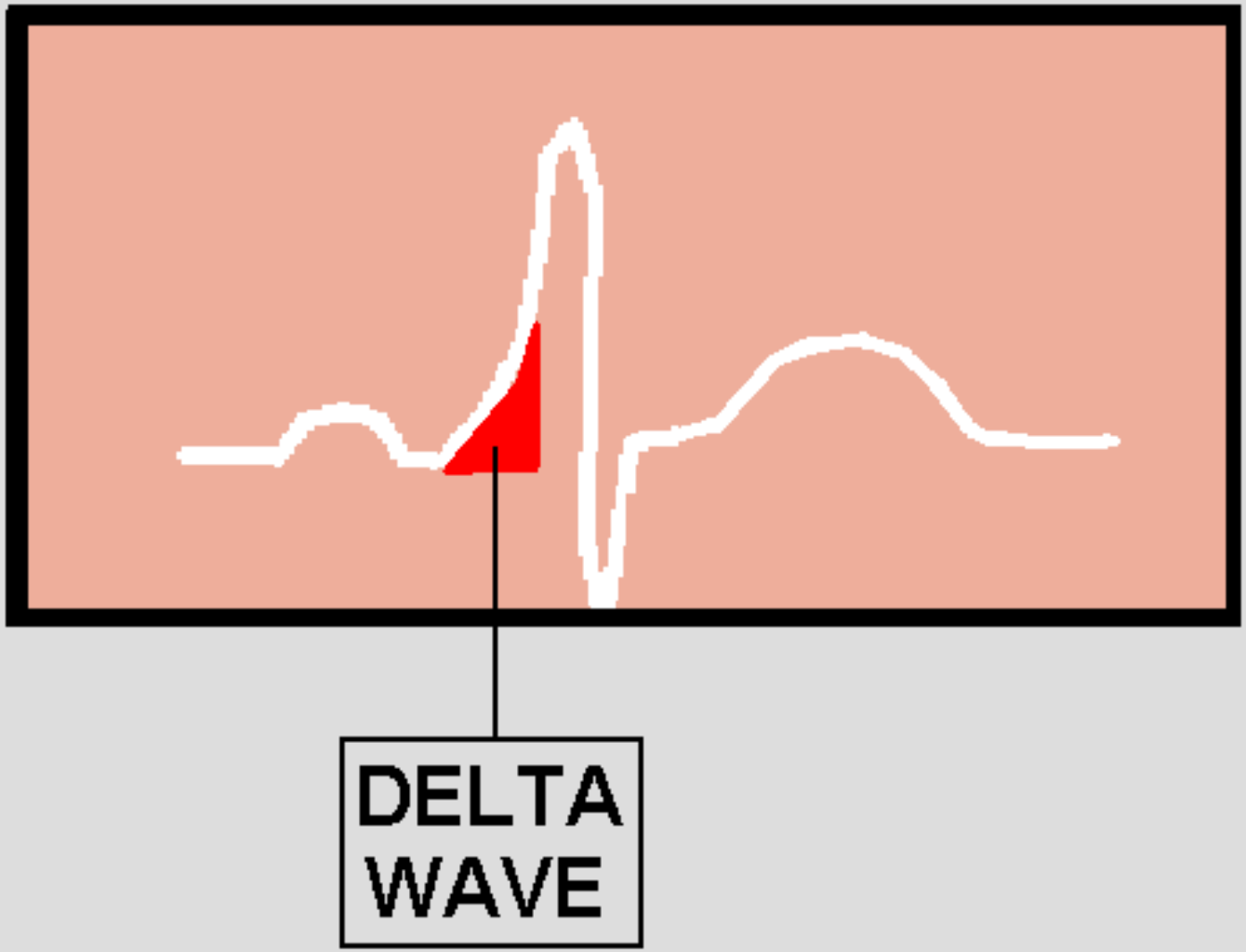
WOLFF-PARKINSON-WHITE

EKG CHARACTERISTICS



WOLFF-PARKINSON-WHITE

EKG CHARACTERISTICS



17-MAY-1997 15:32:09 ST. JOSEPH'S WOMEN'S-WOMEN' ROUTINE RETRIEVAL

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

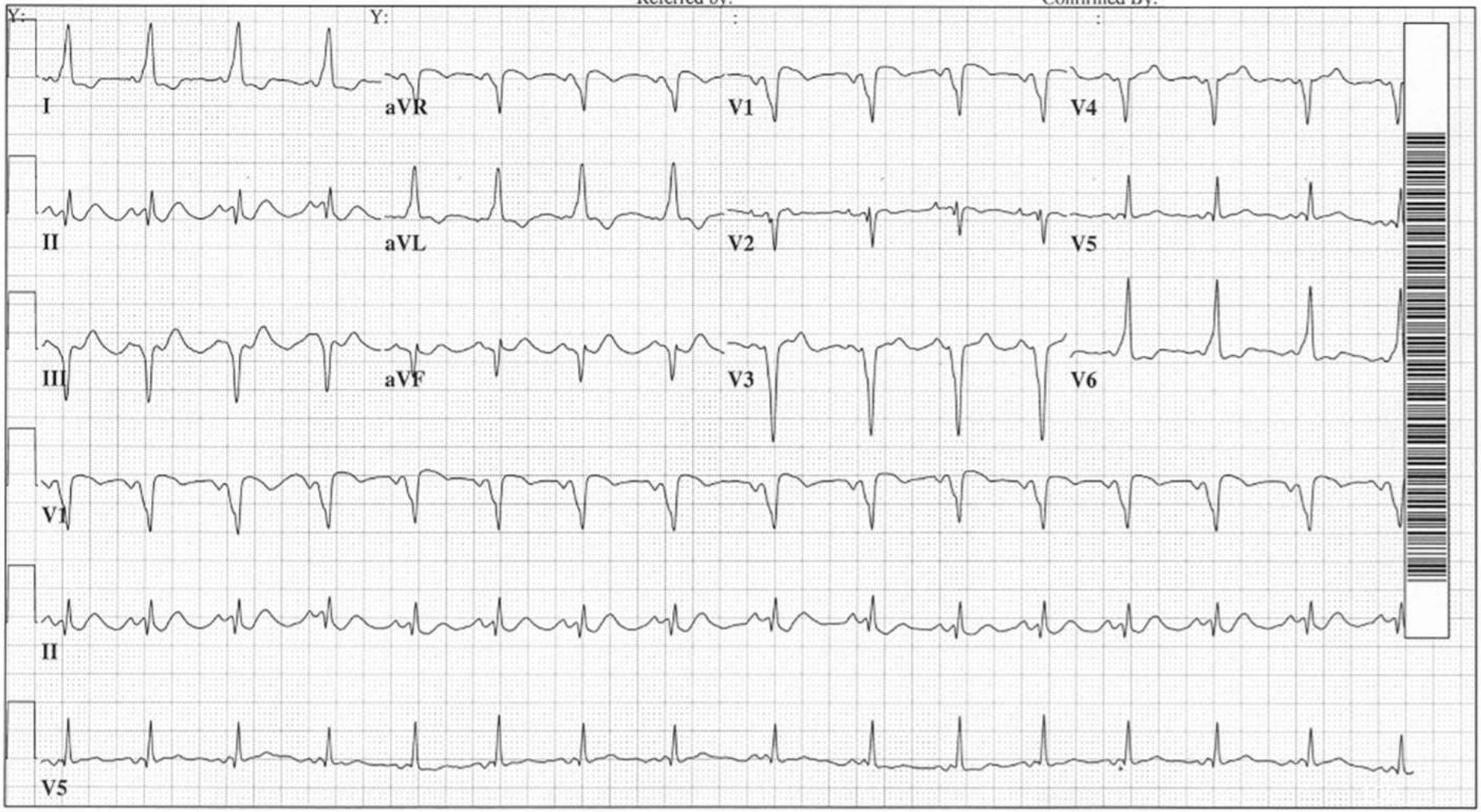
Normal sinus rhythm with sinus arrhythmia
Left atrial enlargement
Anterior infarct , age undetermined
Inferior infarct , age undetermined
ST & T wave abnormality, consider lateral ischemia
Wolff-Parkinson-White
Abnormal ECG
No previous ECGs available

History:Unknown
Technician: DP
Test ind:EKG

EKG CLASS #WR030100
60783

Referred by:

Confirmed By:



25mm/s 10mm/mV 40Hz 005C 12SL 250 CID: 12

EID:18 EDT: 16:01 17-MAY-1997 ORDER:

01-MAY-1999 04:14:17 ST. JOSEPH'S HOSPITAL-IN1464 ROUTINE RETRIEVAL

51 yr
Male Caucasian

Room:540
Loc:5 Option:28

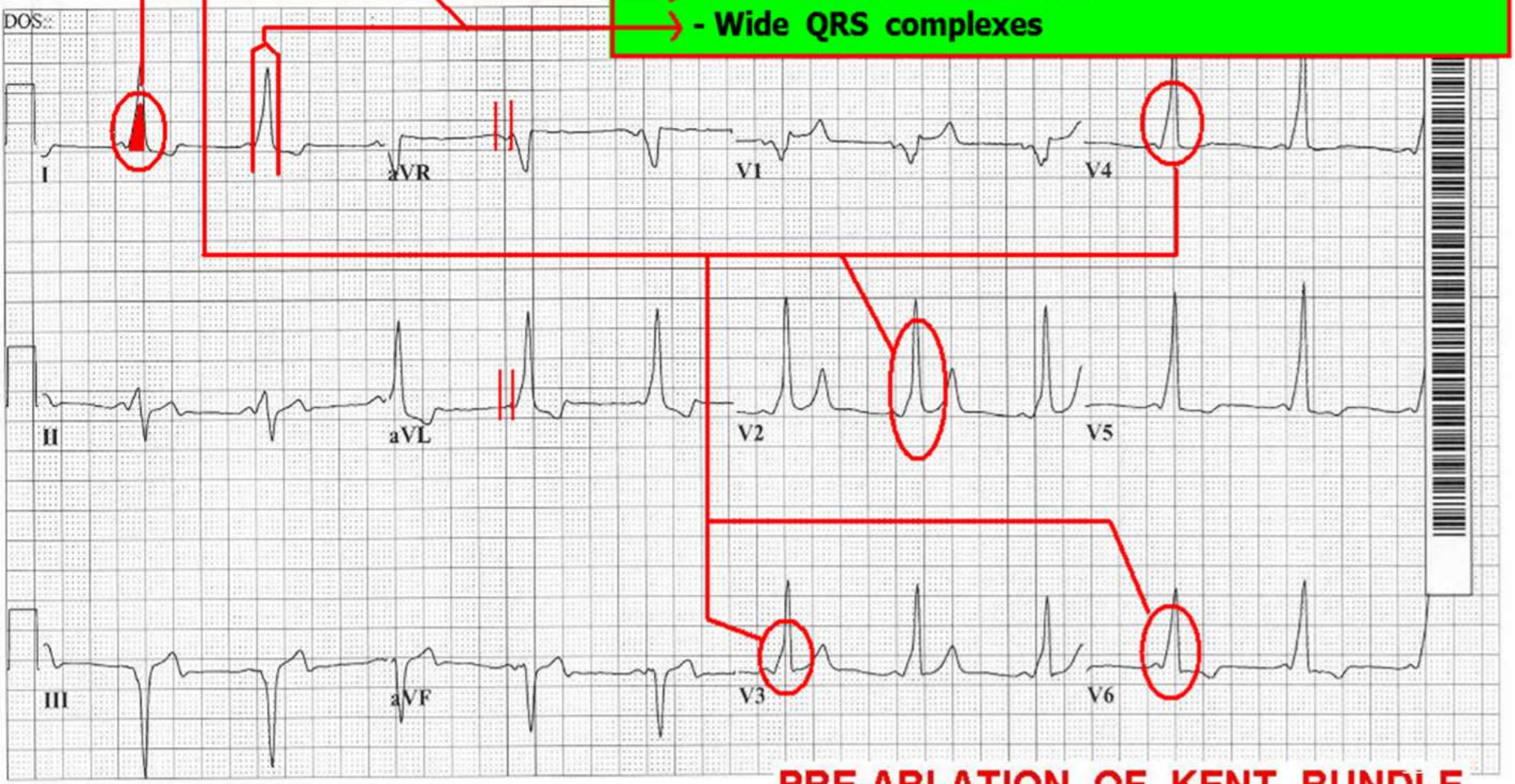
Vent. rate	64	BPM
PR interval	110	ms
QRS duration	146	ms
QT/QTc	418/431	ms
P-R-T axes	50 -36	119

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



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

PRE-ABLATION OF KENT BUNDLE

16 yr
Female Caucasian

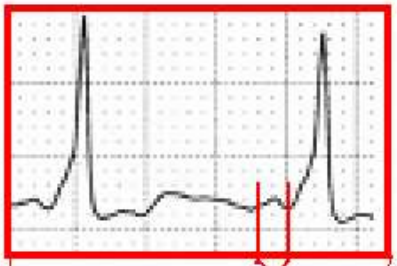
Room:REC
Loc:20 Option:50

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

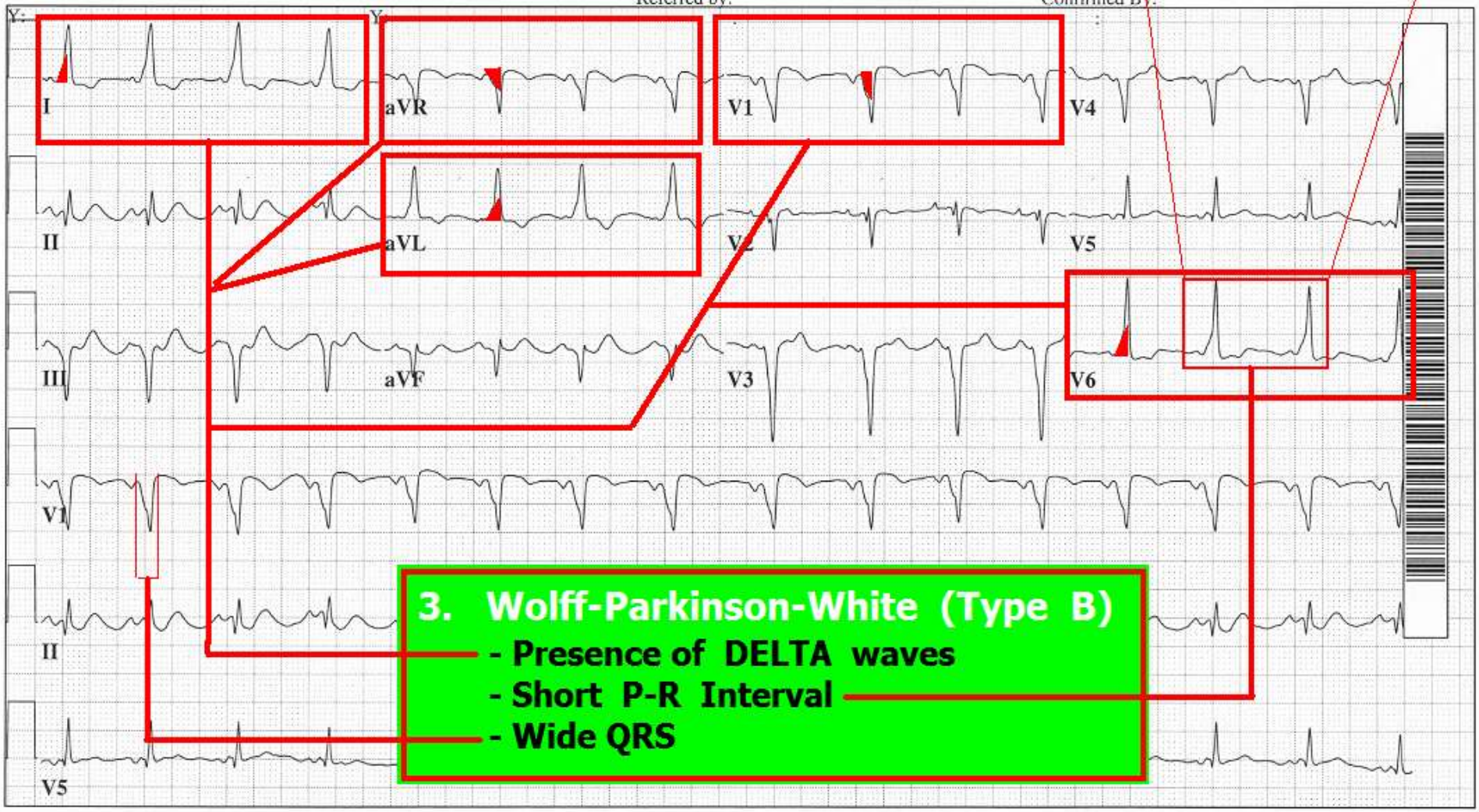
EKG CLASS #WR030100
60783



P-R = .08

Referred by:

Confirmed By:



3. Wolff-Parkinson-White (Type B)

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

25mm/s 10mm/mV 40Hz 005C 12SL 250 CID: 12

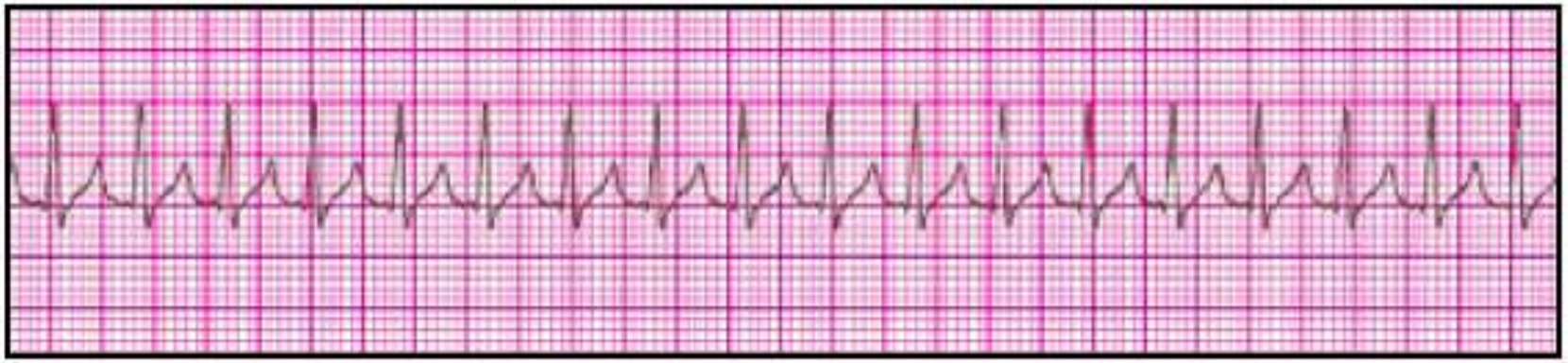
EID:18 EDT: 16:01 17-MAY-1997 ORDER:

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 …….

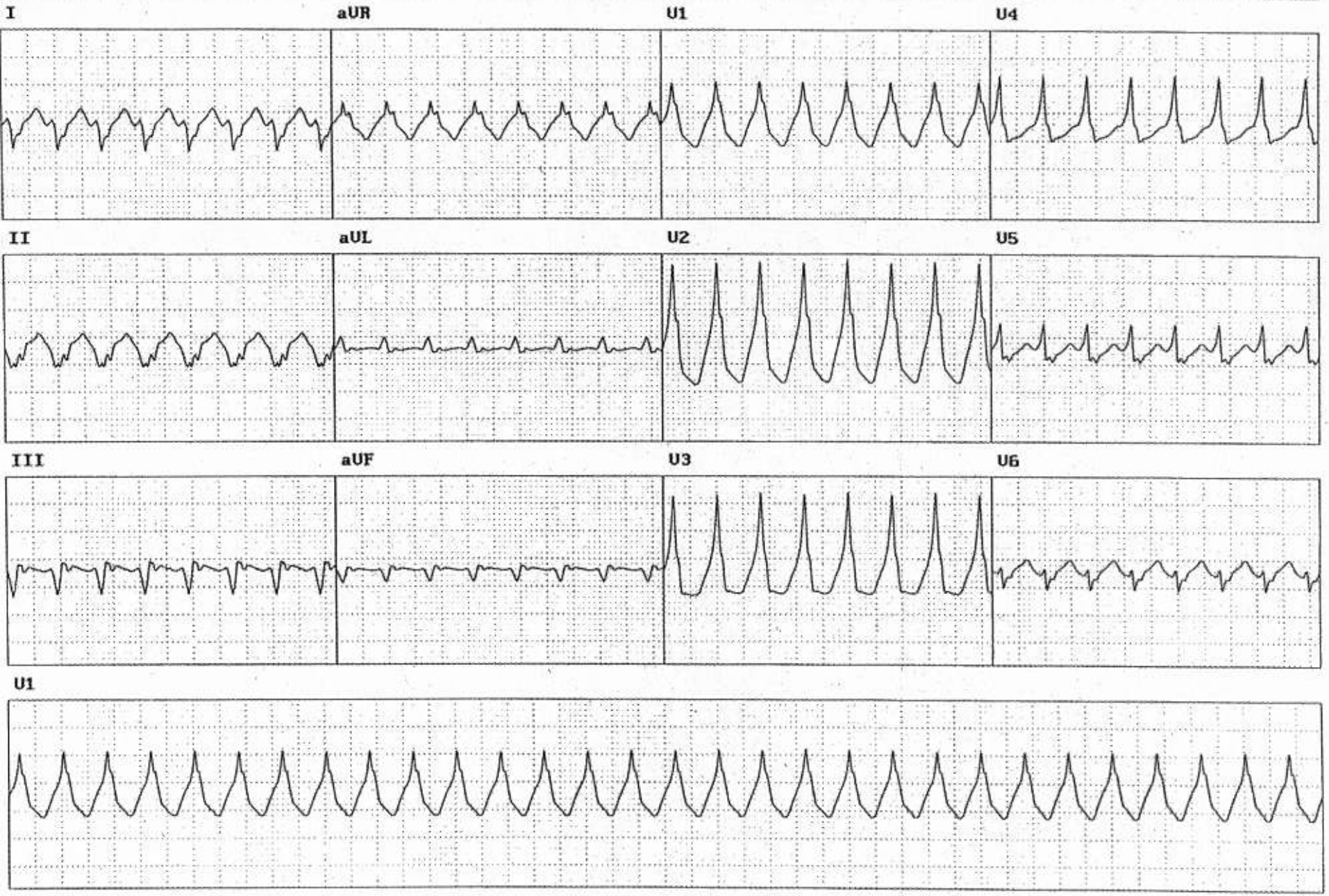
THIS RHYTHM IS: SUPRAVENTRICULAR TACHYCARDIA (SVT)



MAIN IDENTIFICATION CHARACTERISTIC(S): HEART RATE TOO FAST, USUALLY > 150. P WAVES MAY BE "BURIED" IN THE PRECEDING T WAVES. Pt USUALLY C/O "SUDDEN ONSET of HEART RACING," or "PALPITATIONS."

RATE ----------	TACHYCARDIC (usually > 150)	
RHYTHM ----------	REGULAR	
P-R INTERVAL ----	NORMAL or ABNORMAL.	MAY BE IMPOSSIBLE TO SEE DUE TO P WAVE BURIED IN T WAVES
P:QRS RATIO ------	1:1	
QRS INTERVAL ----	NORMAL	

05/04/99 14:16:00 25mm/sec 4.2mm/mU



ST. JOSEPH'S HOSPITAL W-P-W DOUBLE BY-PASS TRACT

Patient Profile: Wolff-Parkinson-White:

- Typically Pediatric / Young Adult

- May not know they have it

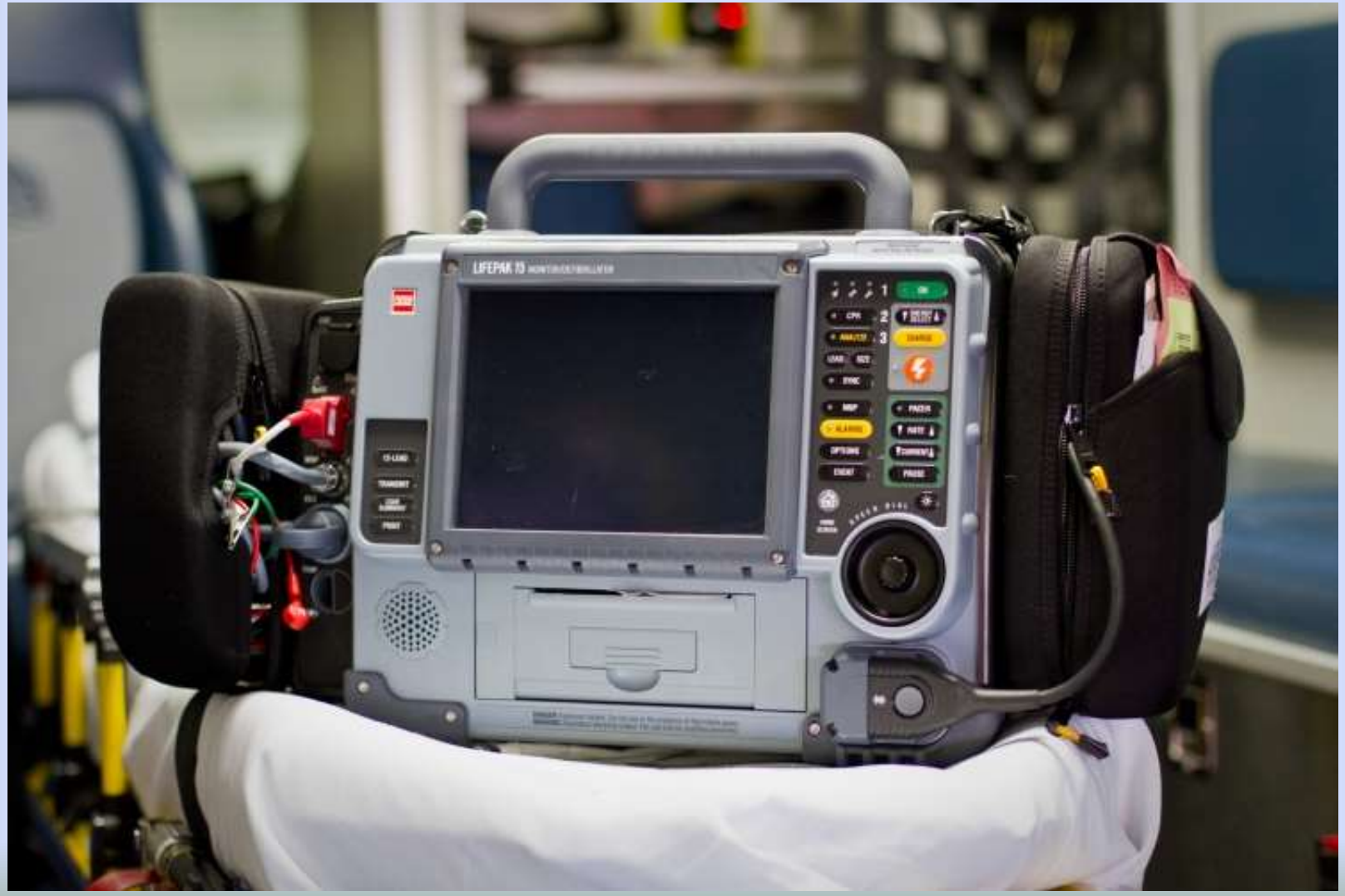
- May experience episodes of "palpitations" or "Very Fast Heartbeat."

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

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

OLD BARN, SHREWSBURY, PA – 2001

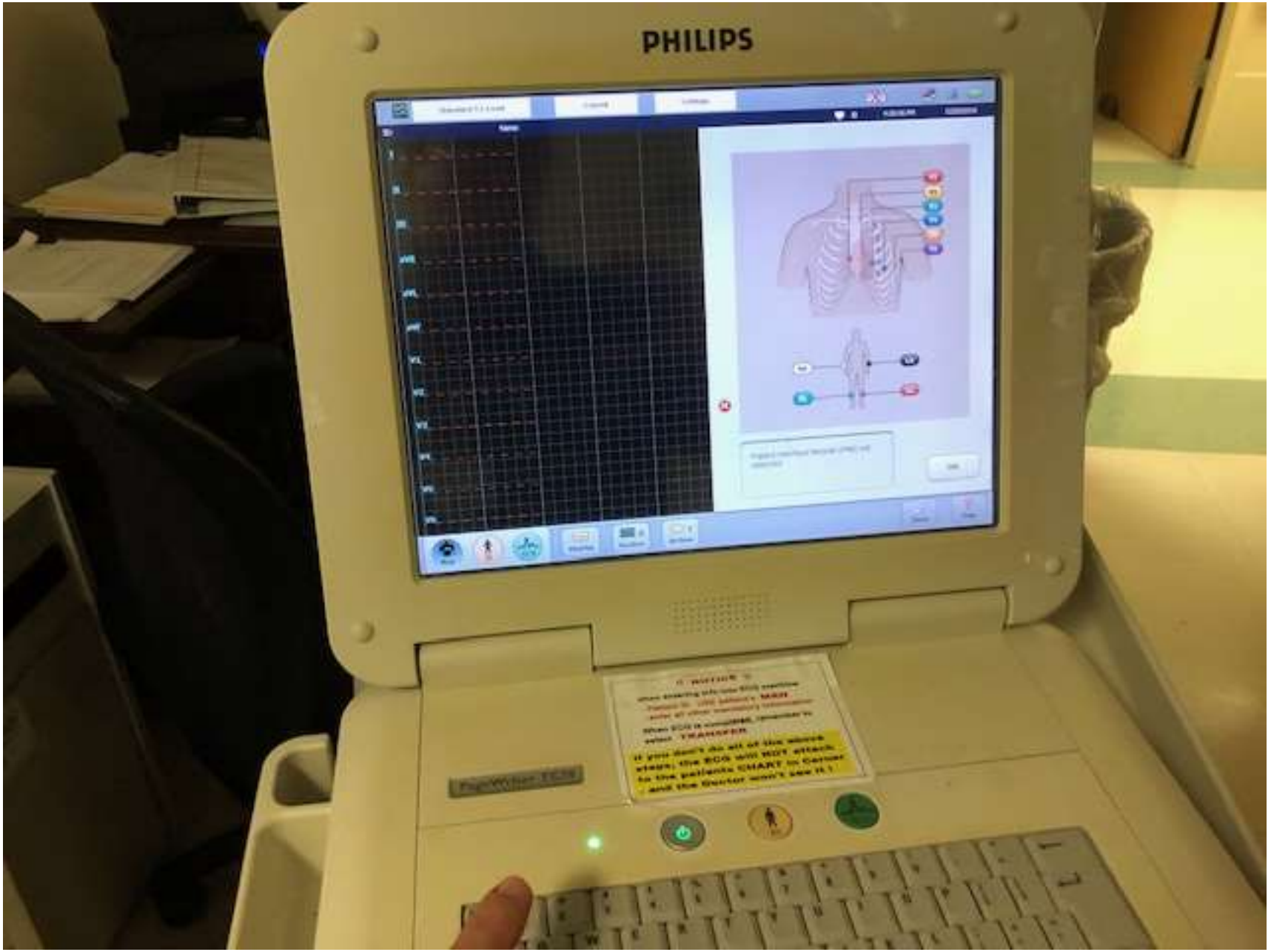
EMS 12 Lead ECG



10 wires . . .

- 4 limb leads
- 6 chest (“V”) leads


Sanitas


PHILIPS

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.

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

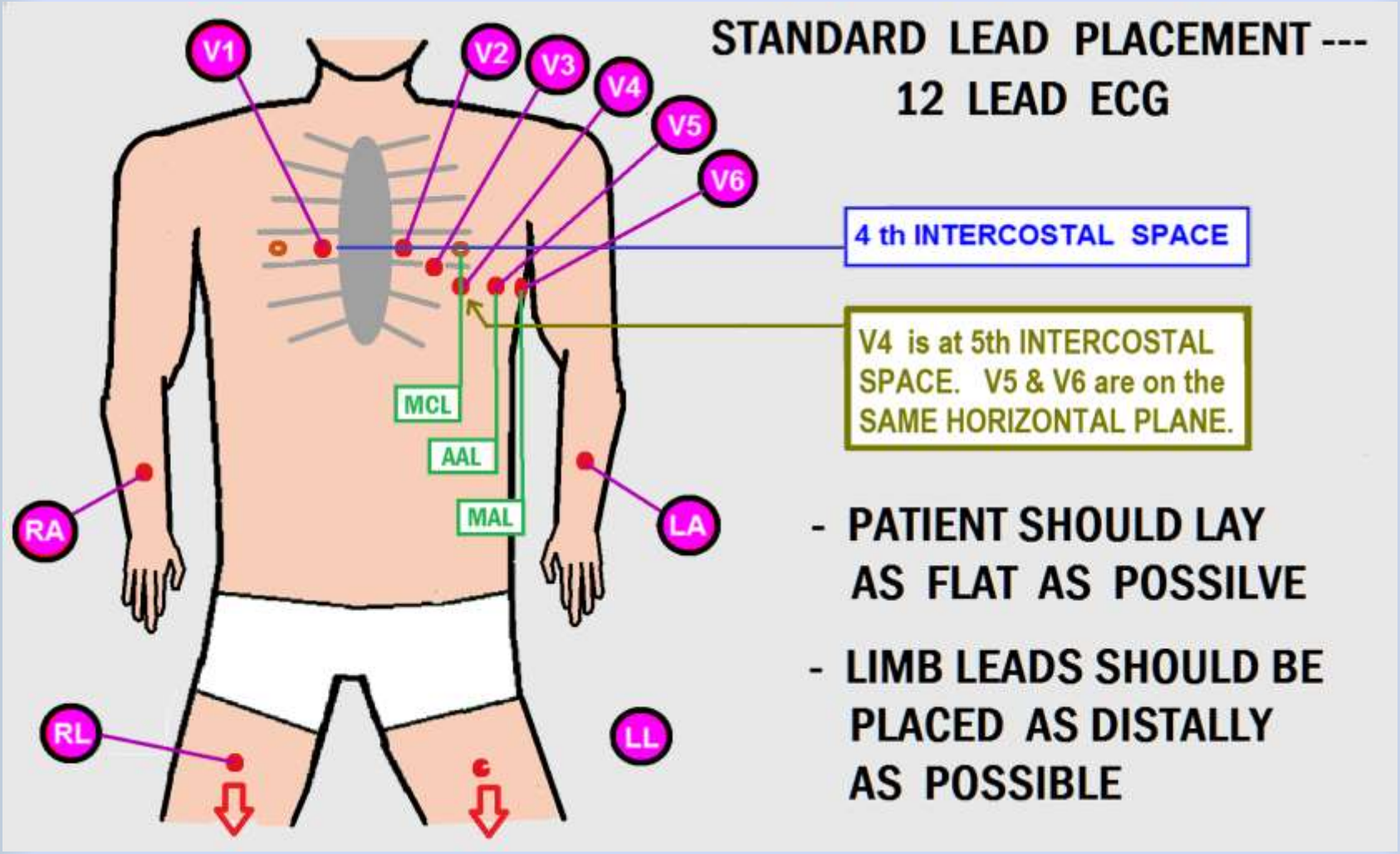
Recommendations for the Standardization and Interpretation of the Electrocardiogram

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

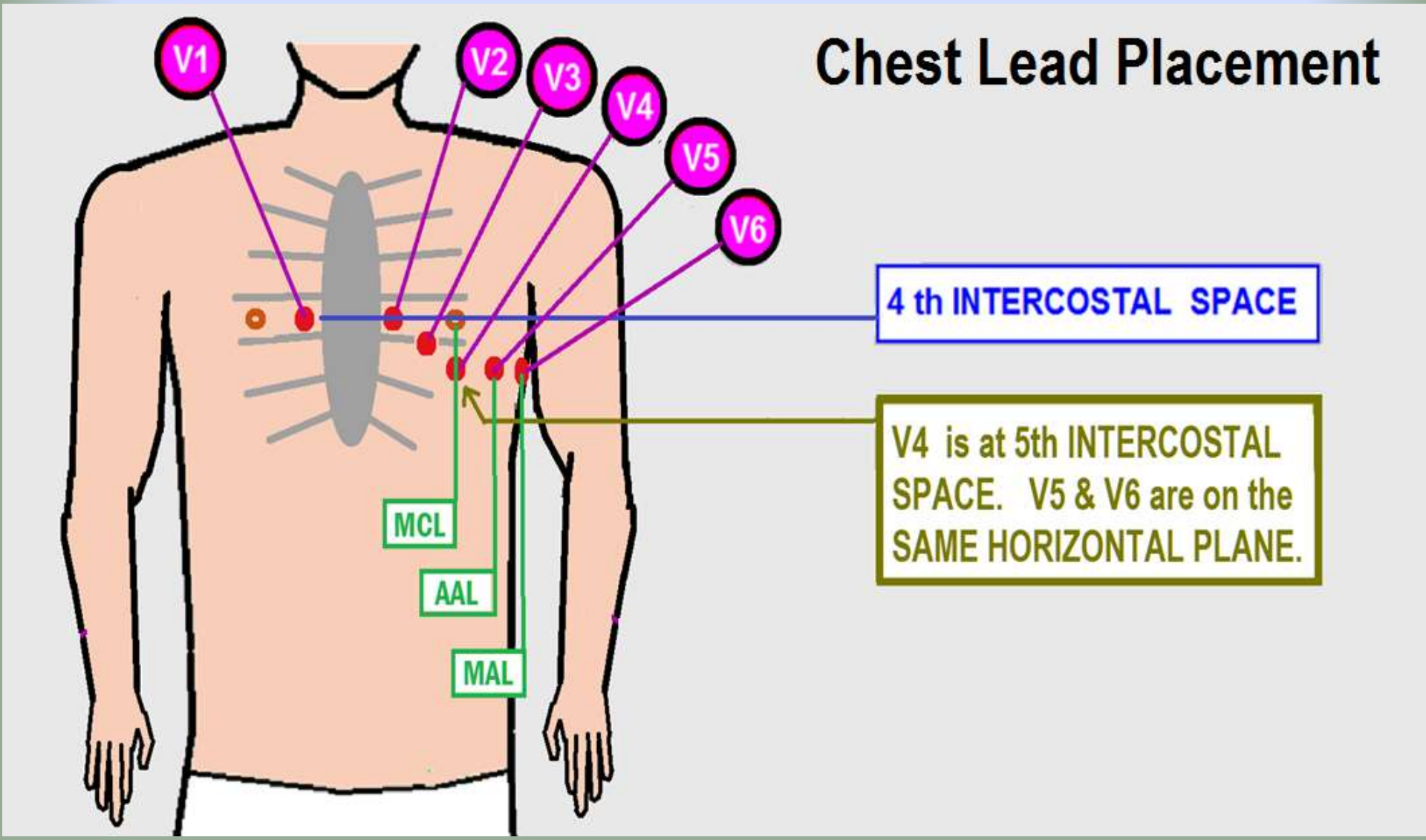
Obtaining the 12 Lead ECG



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:



[redacted] [redacted] [redacted]

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

--AXIS--
P 50
QRS 51
T 44

12 Lead; Standard Placement

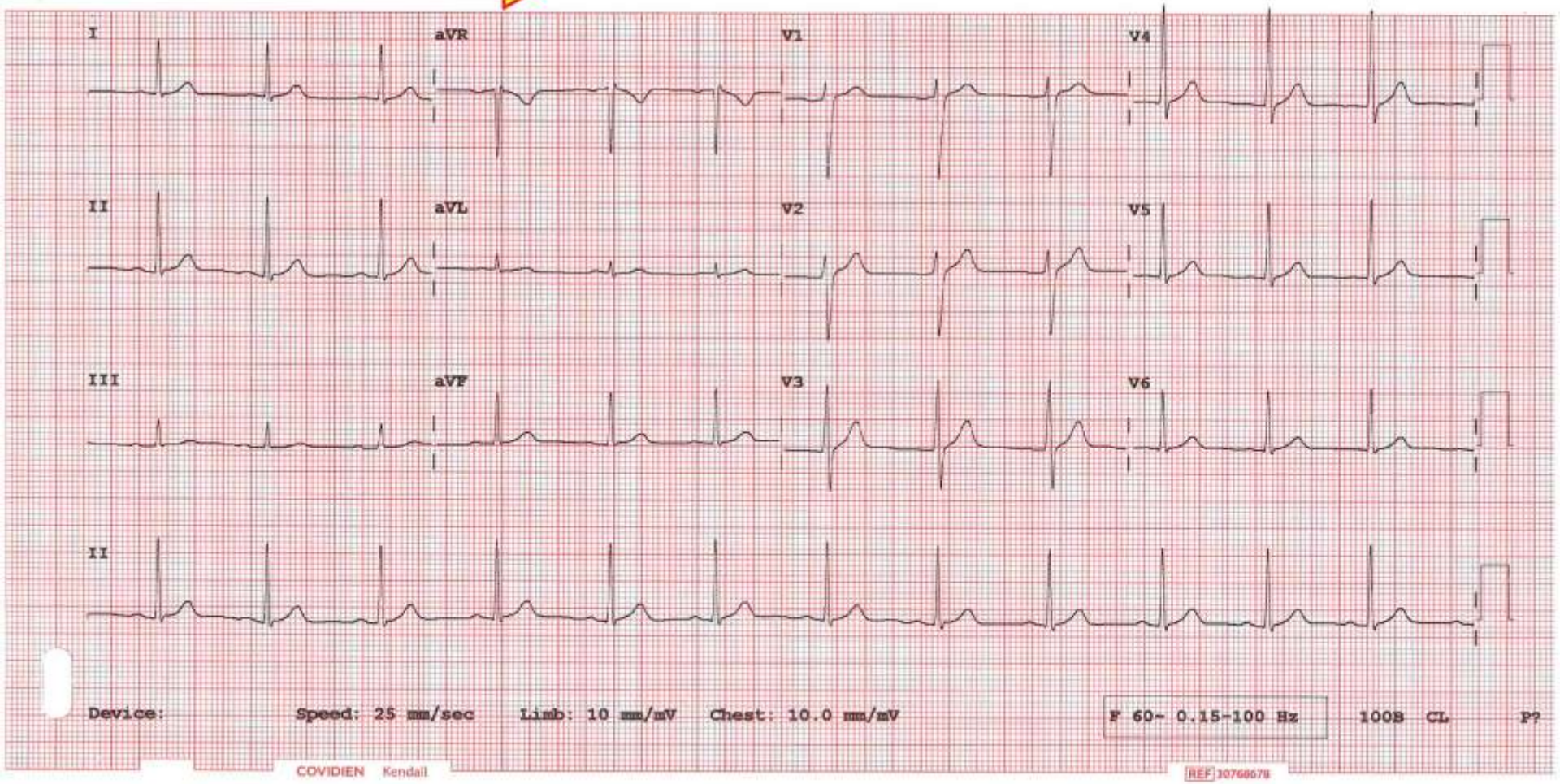
[redacted] TECH SD

[redacted]

- NORMAL ECG -

Unconfirmed Diagnosi

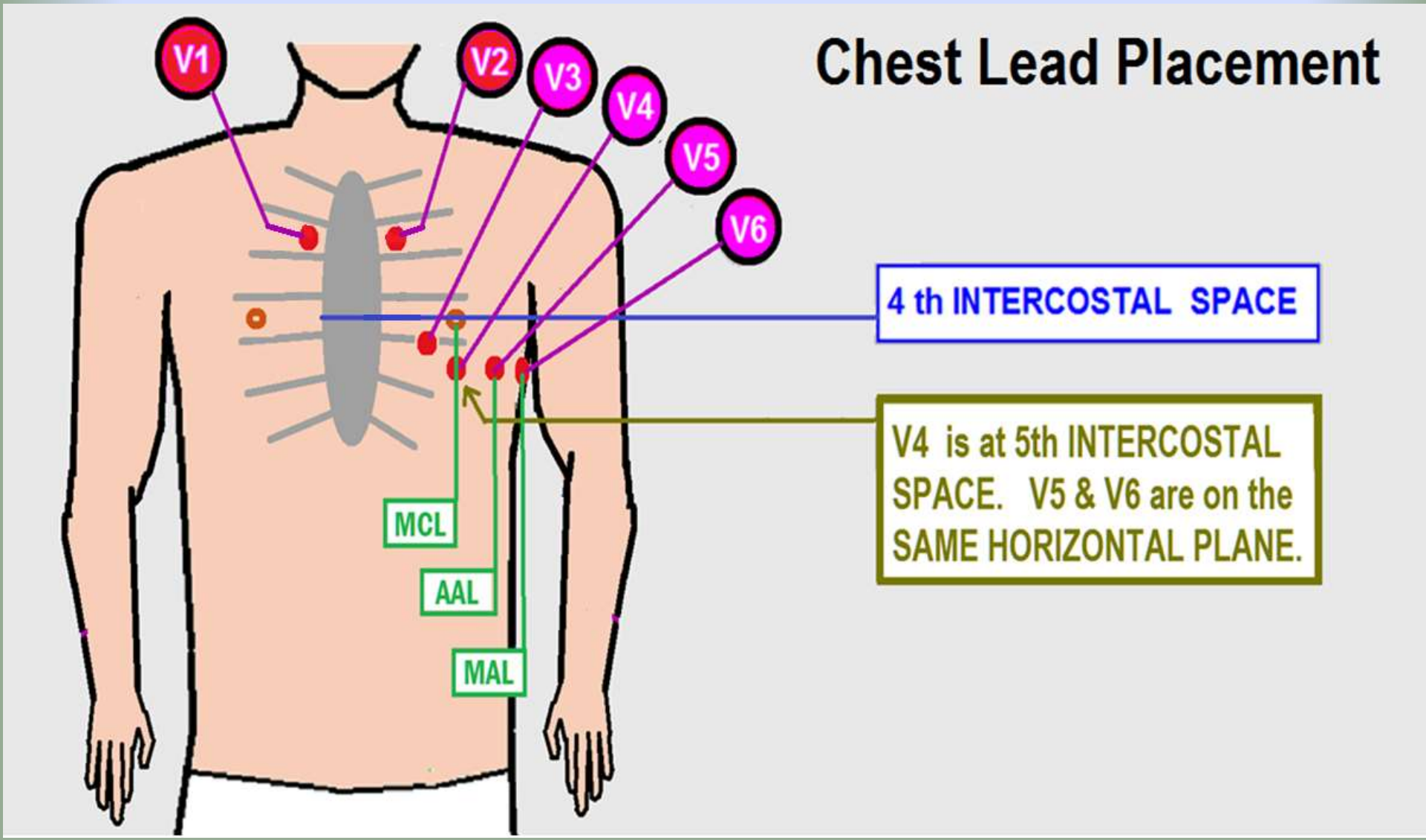
[redacted]



Device: Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10.0 mm/mV F 60- 0.15-100 Hz 100B CL P?

COVIDIEN Kendall REF 30768678

INCORRECT Lead placement:



[redacted]

DOB [redacted]1988 30 Years [redacted] Female [redacted] 5:20:58 AM

(1)

3NE

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

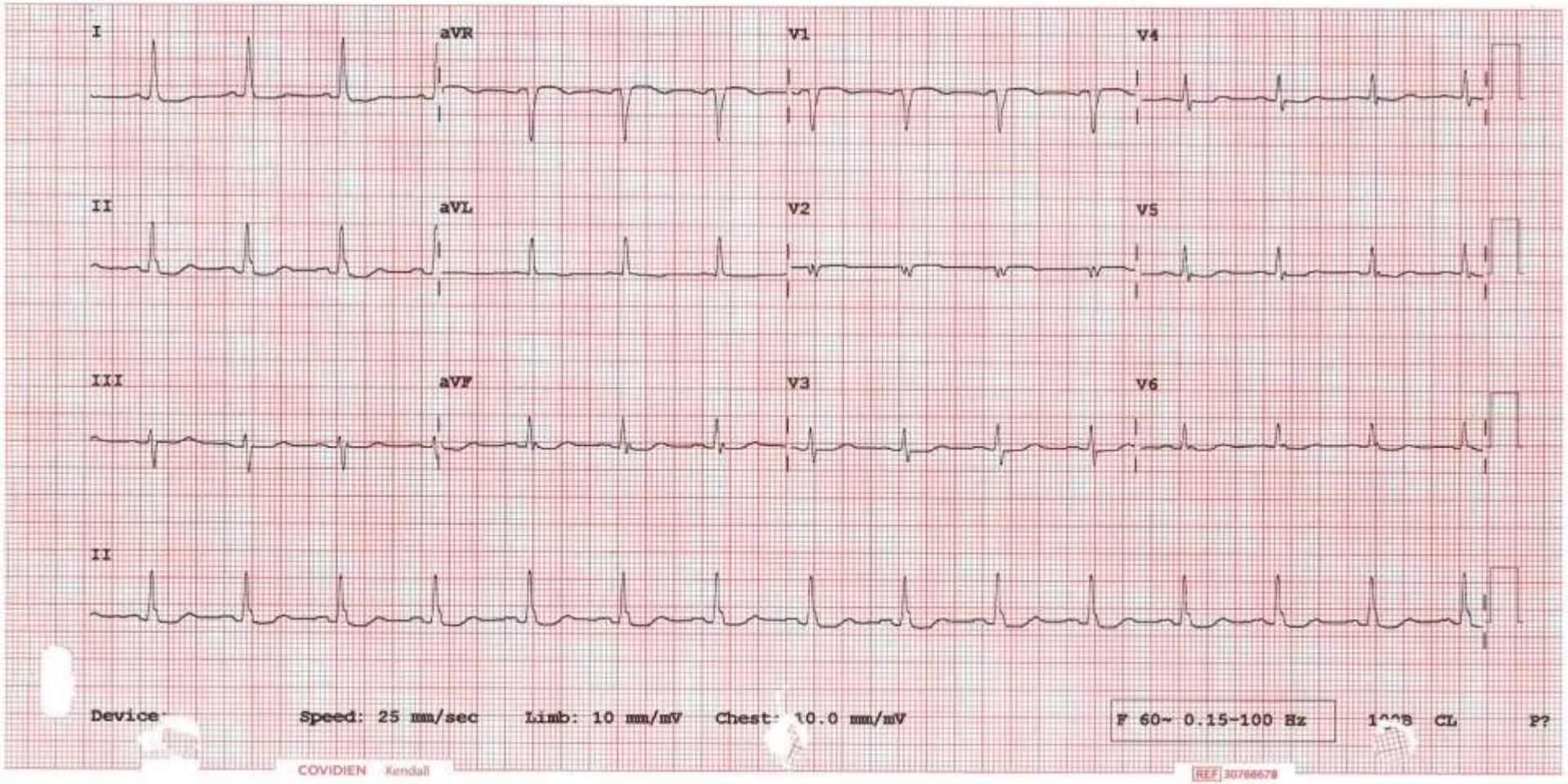
--AXIS--
P 46
QRS 24
T 86

12 Lead; Standard Placement

- ABNORMAL ECG -

Unconfirmed Diagnosis

[redacted]



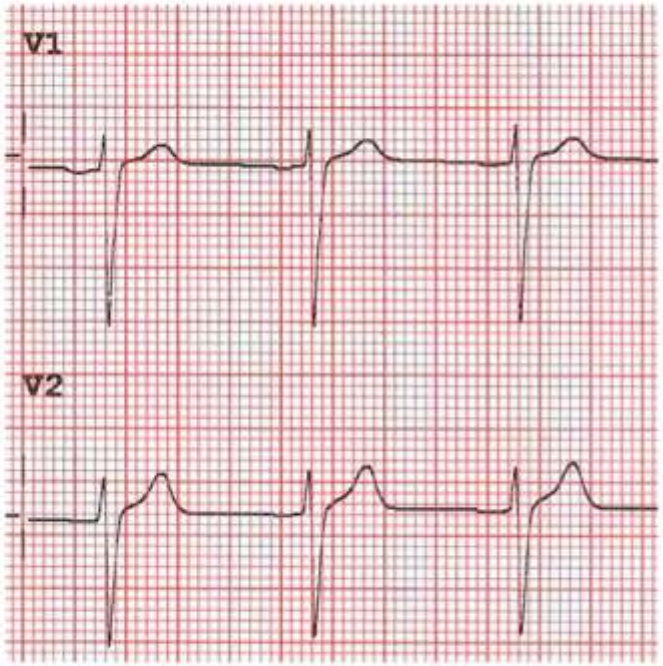
AHA/ACC/HRS Scientific Statement

Recommendations for the Standardization and Interpretation of the Electrocardiogram

Part I: The Electrocardiogram and Its Technology

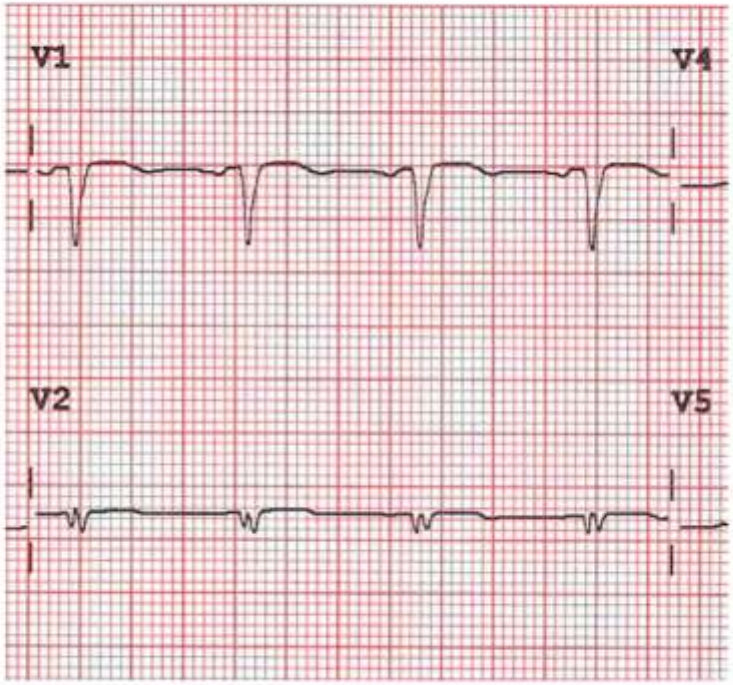
the often profound alterations in waveforms that can result from precordial electrode misplacement.[85,86] A common error is superior misplacement of V_1 and V_2 in the second or third intercostal space. This can result in reduction of initial R-wave amplitude in these leads, approximating 0.1 mV per 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

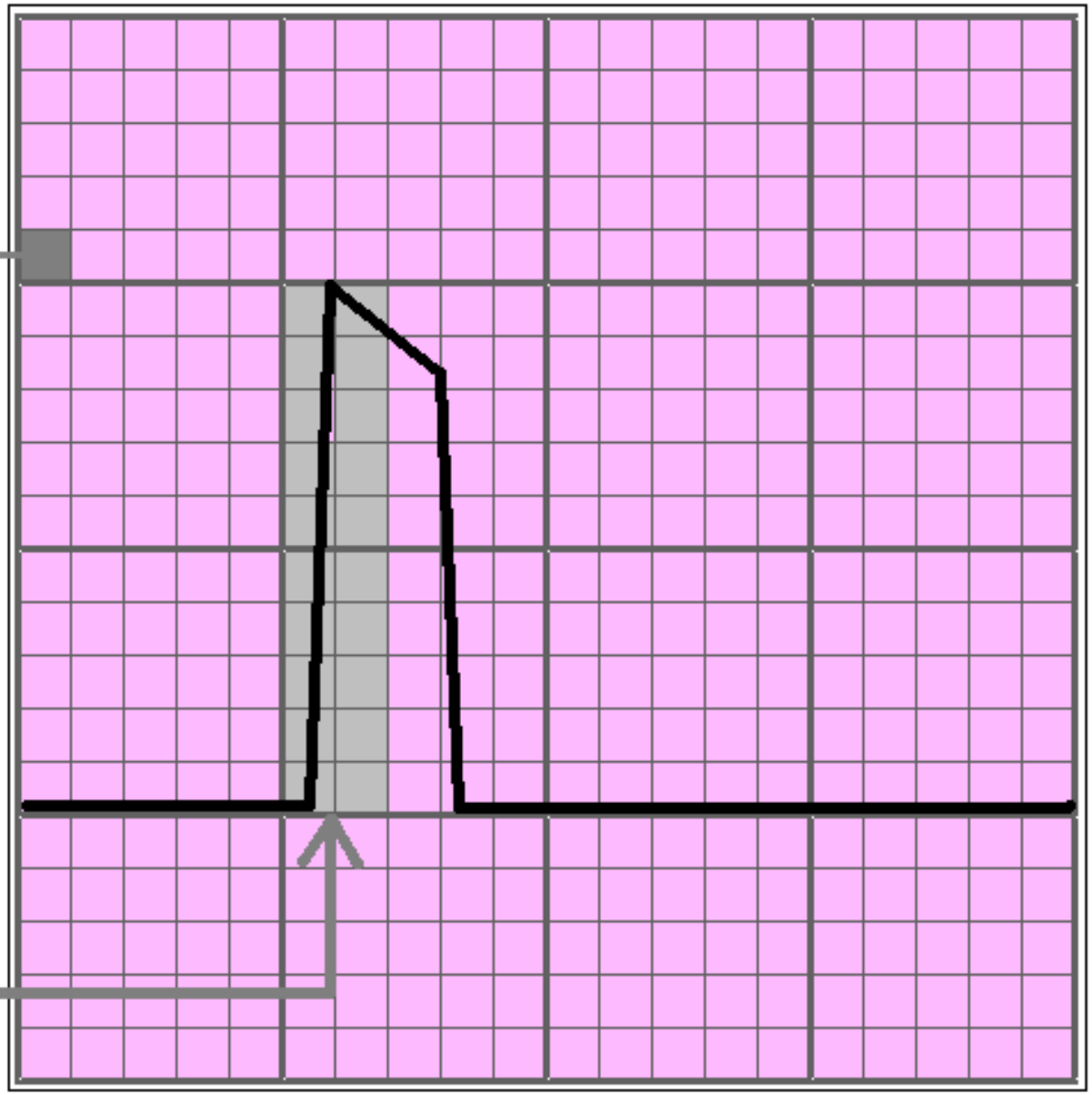
Incorrect Lead Placement



QS = old MI

ECG PAPER - THE VERTICAL AXIS:

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



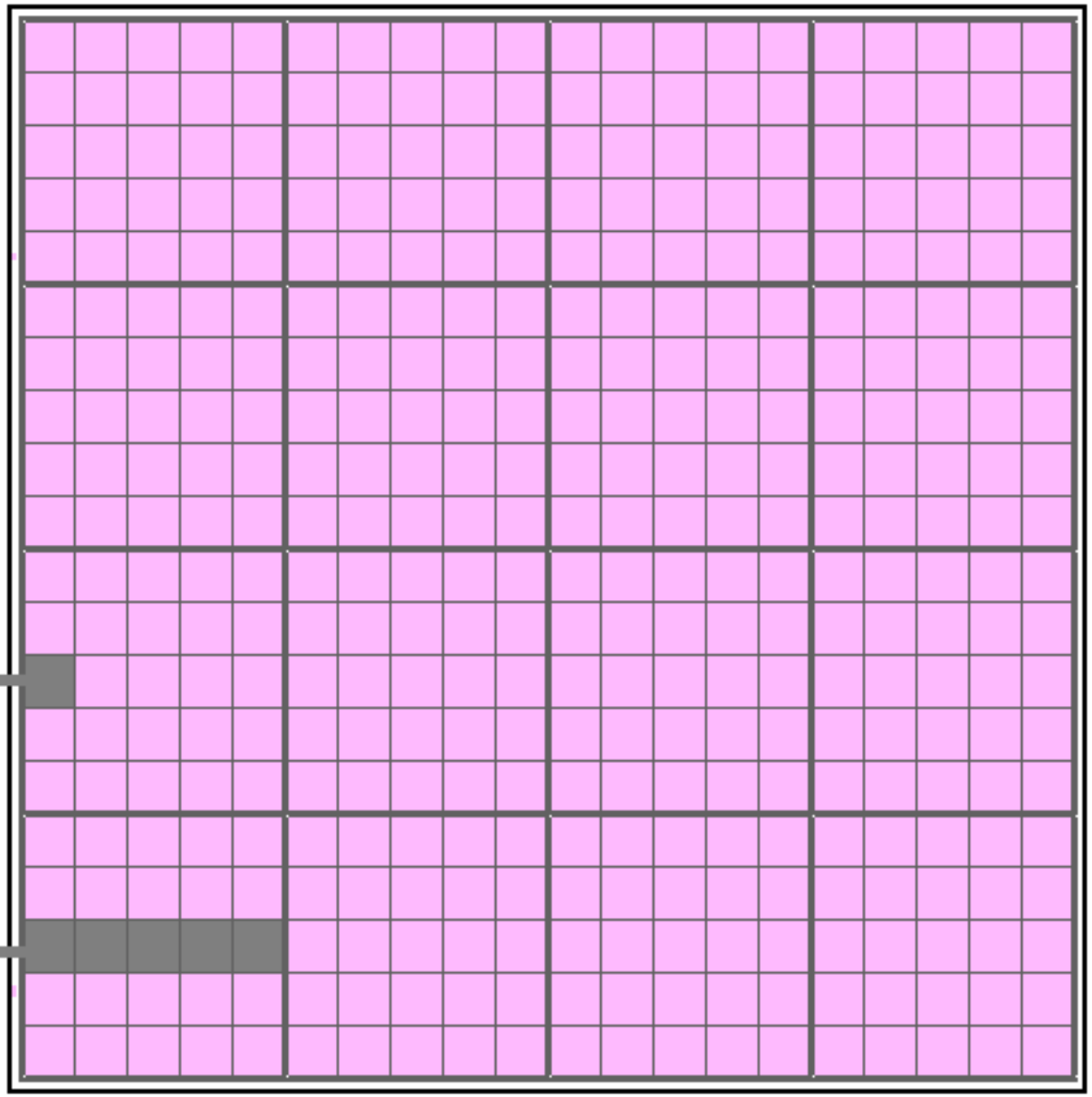
ECG PAPER - THE HORIZONTAL AXIS:

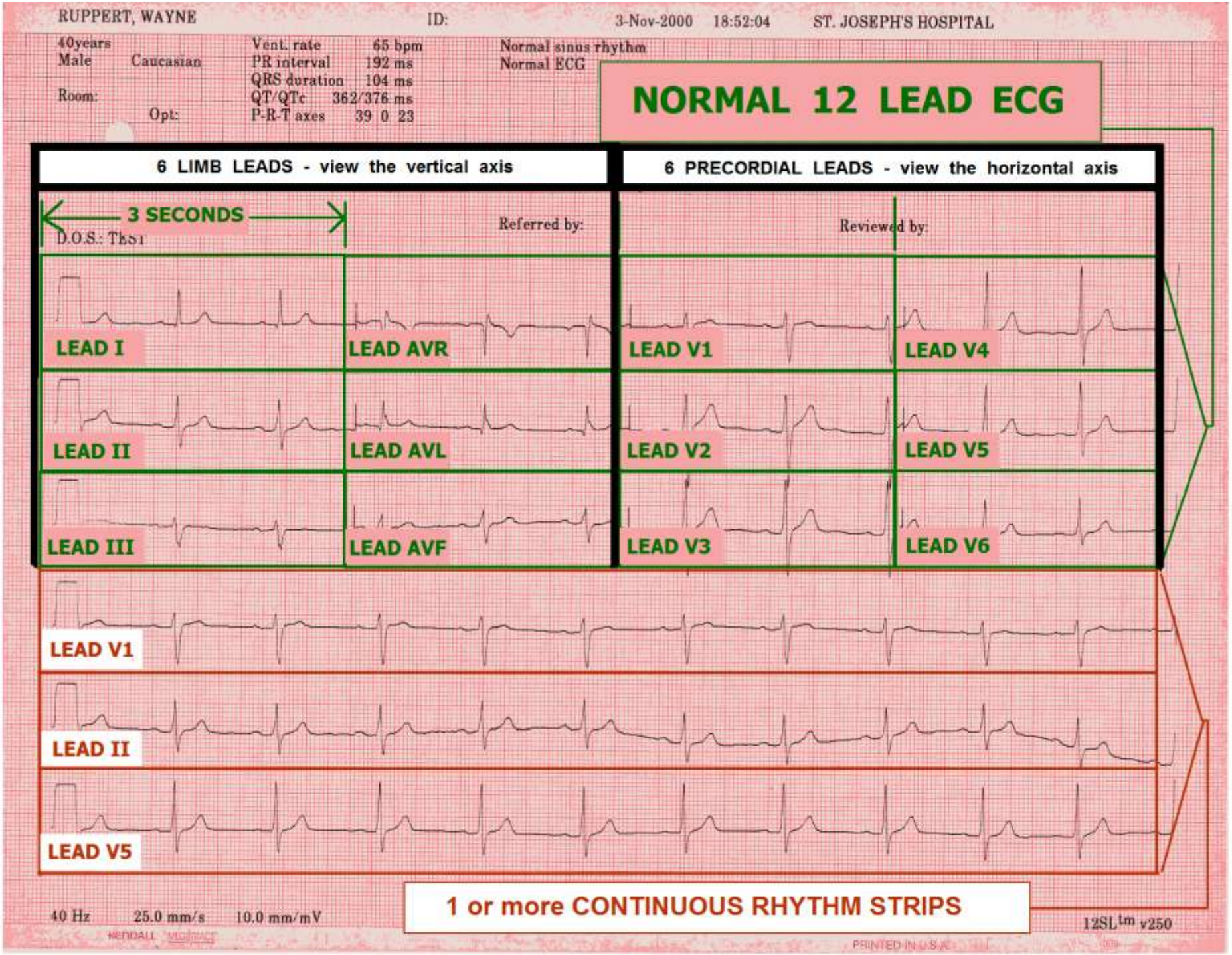
THE HORIZONTAL AXIS REPRESENTS TIME . . .

STANDARD SPEED FOR RECORDING ADULT EKGs = 25 mm / SECOND

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

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




RUPPERT, WAYNE
ID:
3-Nov-2000 18:52:04
ST. JOSEPH'S HOSPITAL
40years
Male
Caucasian
Room:
Opt:
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.: Tbs1
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 STRIPS
12SL tm v250
KENDALL
PRINTED IN U.S.A.

[redacted]

DOB [redacted] 75 Years

Evaluate this EKG for each of the "Normal" criteria seen on the NEXT 2 SLIDES . . . . . .

Rate 76 . Sinus rhythm.

PR 161
QRSD 90
QT 350
QTc 394

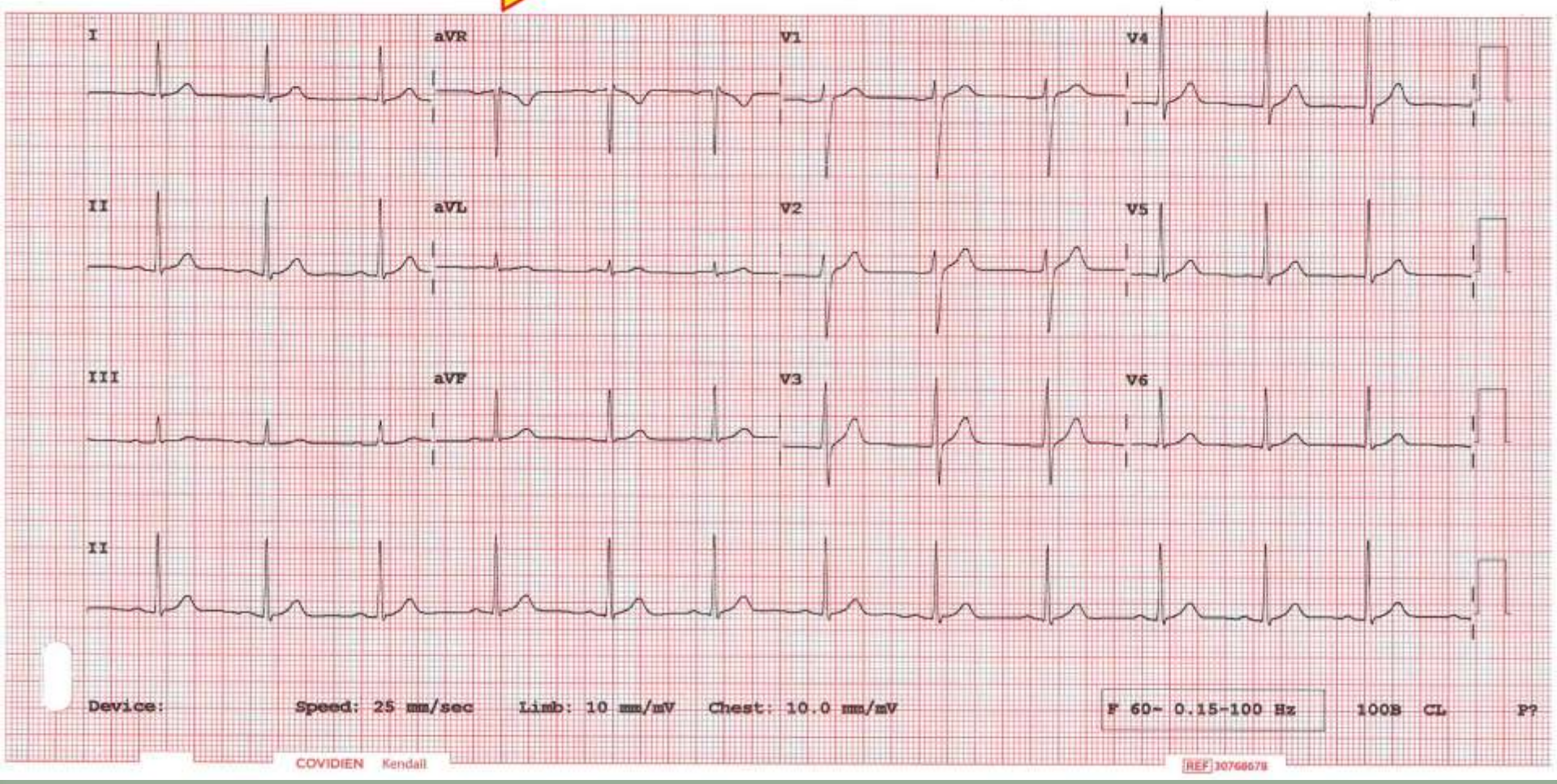
--AXIS--
P 50
QRS 51
T 44

12 Lead; Standard Placement

- NORMAL ECG -

Unconfirmed Diagnosi

[redacted]



The Normal 12 Lead EKG

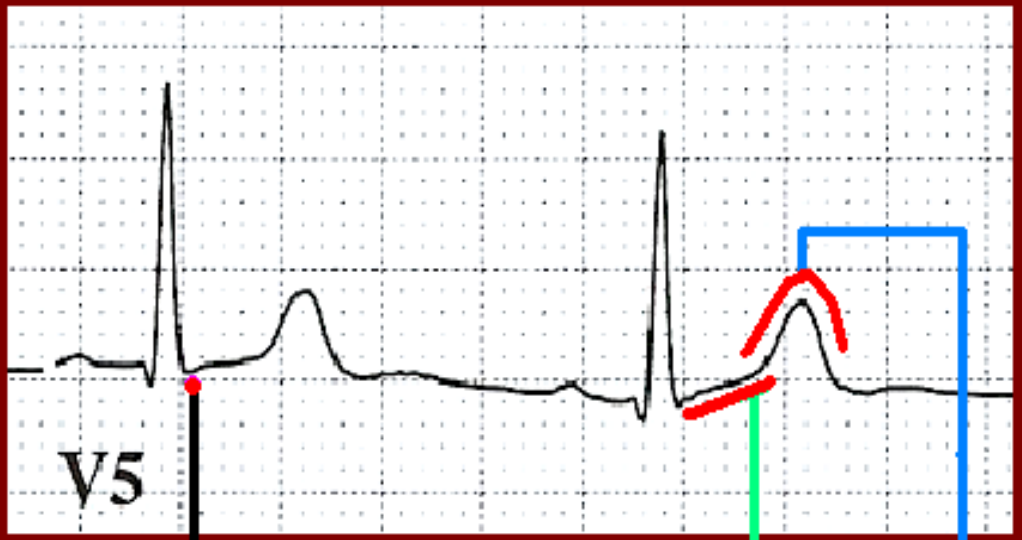
- NSR (rate 60-100, regular rhythm)
- P Waves upright all leads except aVR
- P Waves inverted lead aVR, possibly V1
- QRS upright Leads I, II, III, aVL, aVF, V5, V6
- QRS inverted Leads aVR, V1, V2
- QRS biphasic: Leads V3, V4
- P wave size: up to 2mm tall, 2.5mm long
- QRS height Limb Leads: 5-15mm 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

NORMAL ST - T WAVES

- WHEN QRS WIDTH IS NORMAL (< 120 ms)



ASSESS:

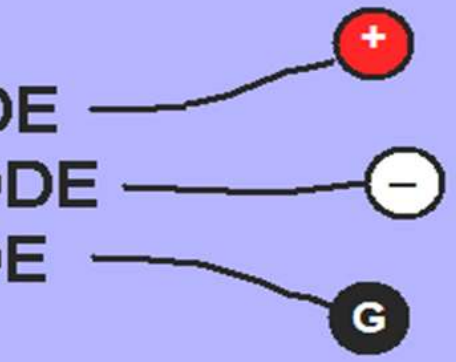
- J POINT: ISOELECTRIC (or < 1 mm dev.)
- ST SEG: SLIGHT, POSITIVE INCLINATION
- T WAVE: UPRIGHT, POSITIVE

☞ *in EVERY LEAD EXCEPT aVR ! !*

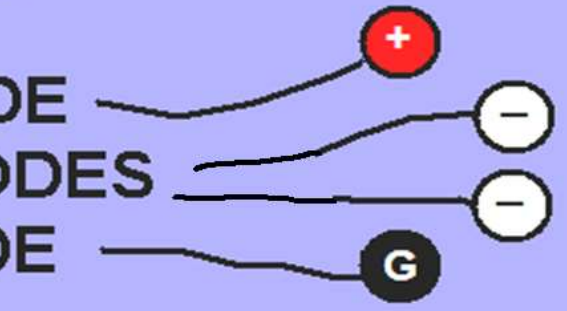
THE ECG MACHINE

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

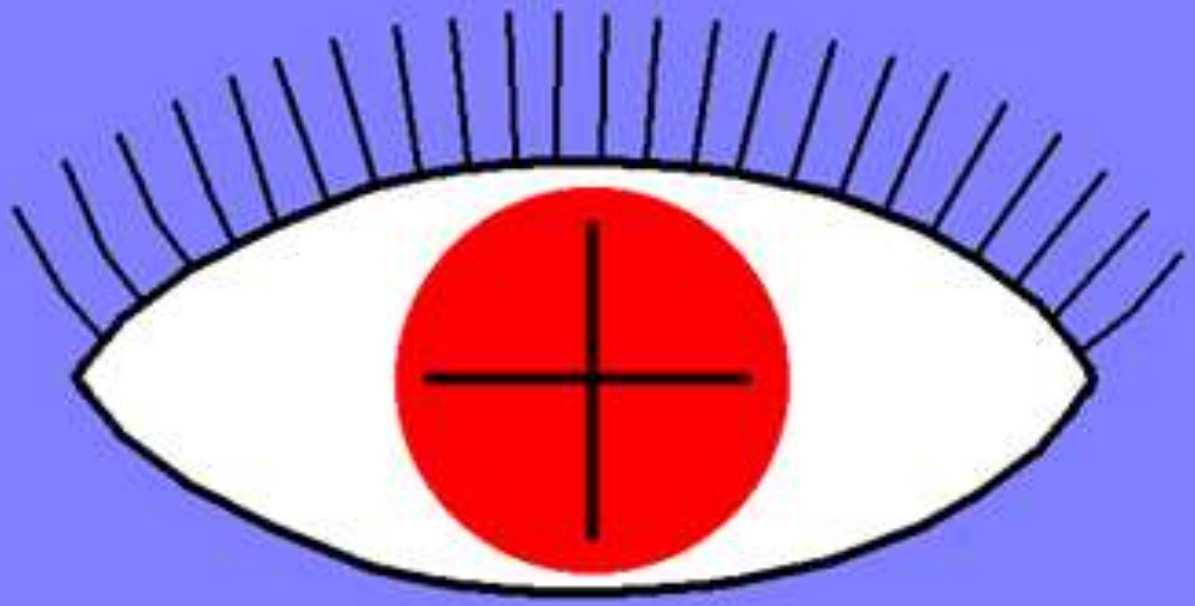
- LEADS I, II, III, and V1, V2, V3, V4, V5, V6



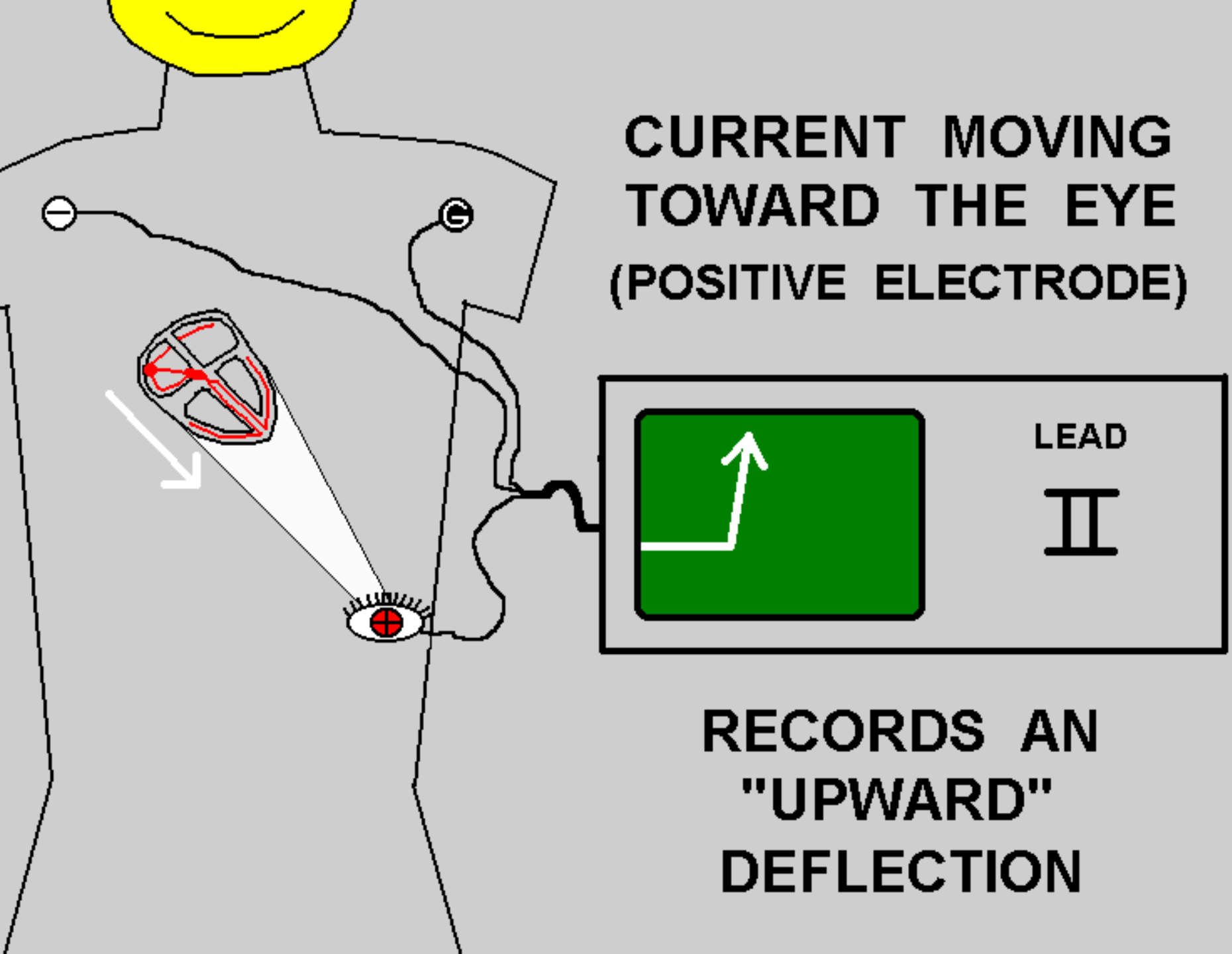
- LEADS AVR, AVL, and AVF

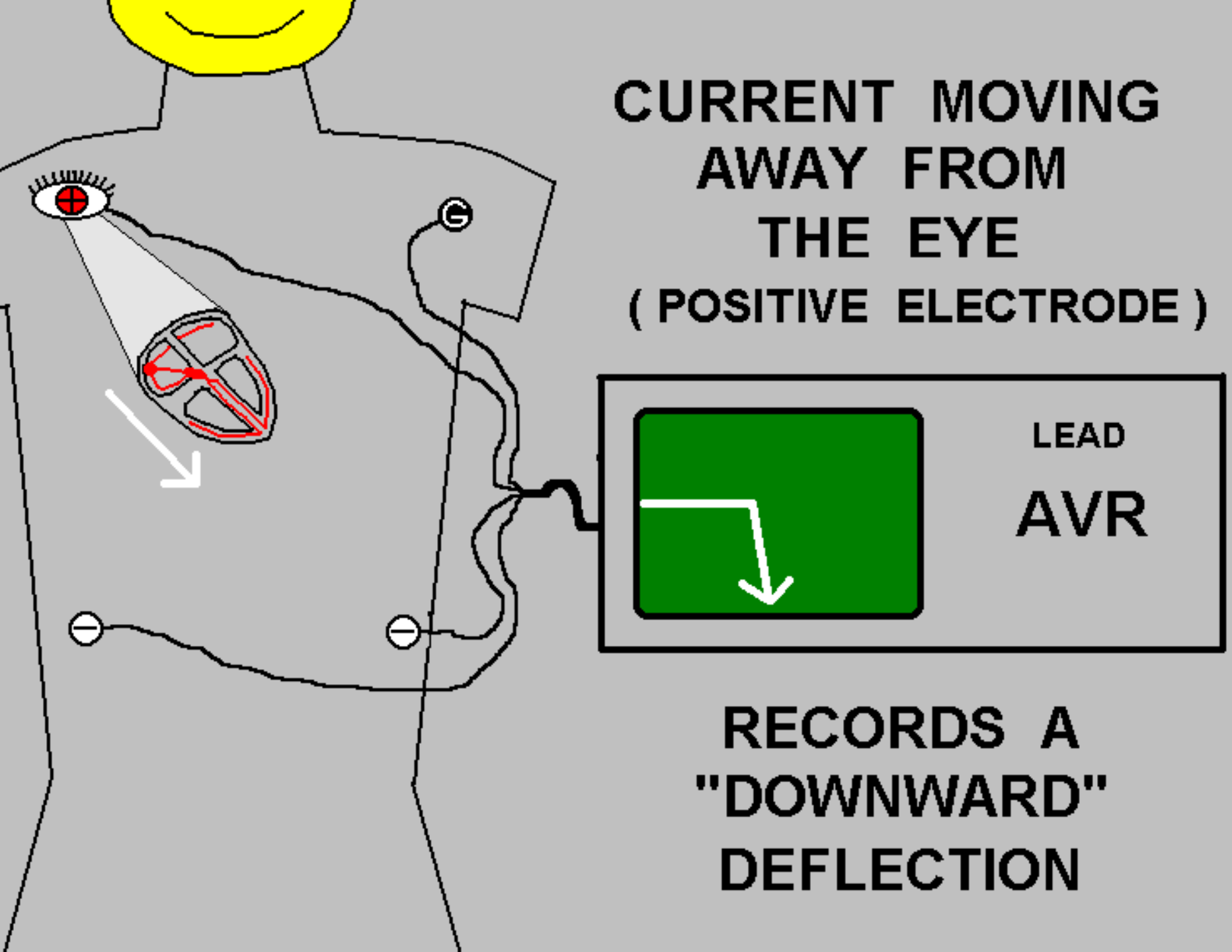


THE POSITIVE ELECTRODE

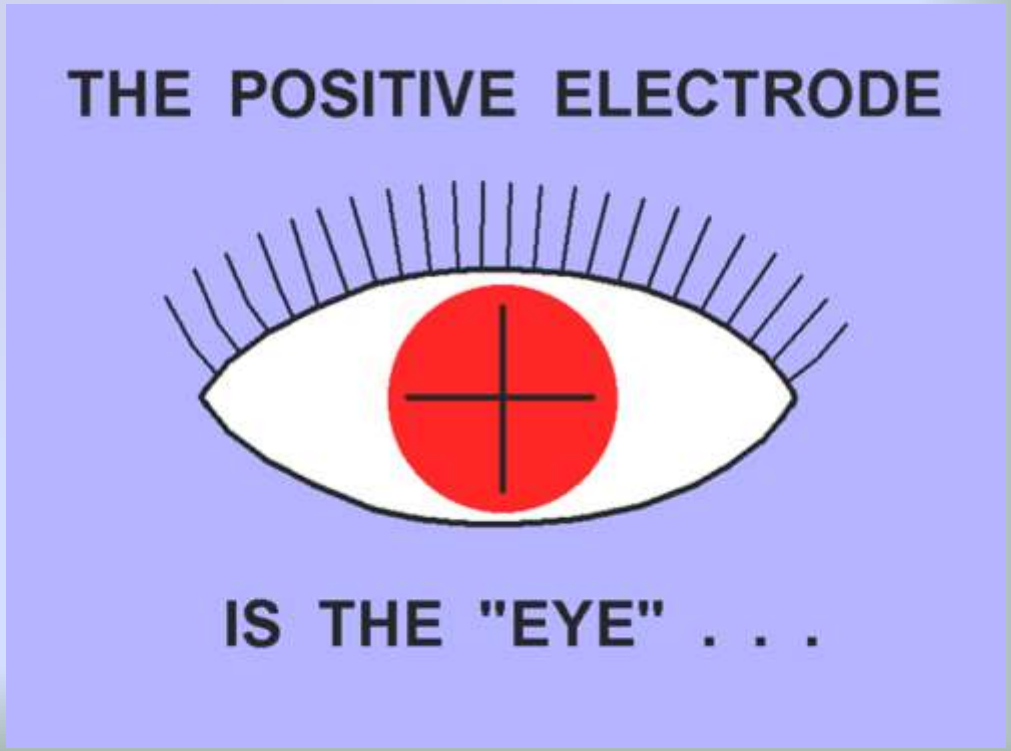


IS THE "EYE" . . .


CURRENT MOVING
TOWARD THE EYE
(POSITIVE ELECTRODE)
LEAD
II
RECORDS AN
"UPWARD"
DEFLECTION

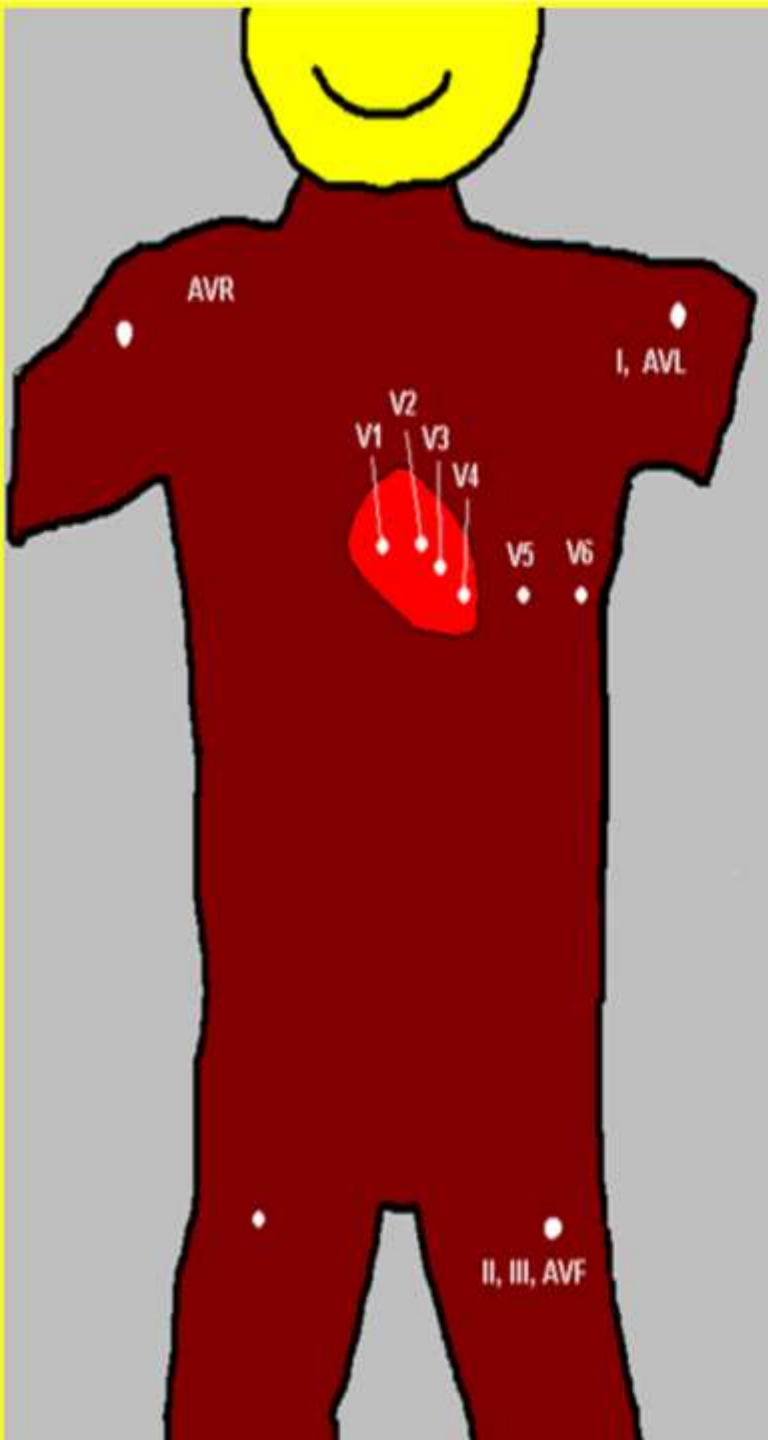

CURRENT MOVING AWAY FROM THE EYE
(POSITIVE ELECTRODE)
LEAD
AVR
RECORDS A "DOWNWARD" DEFLECTION

What part of the HEART does each lead SEE ?



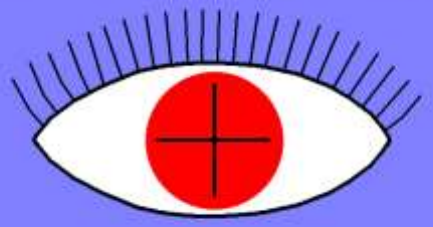
Imagine a body made of clear glass, with only a HEART inside. We dip this body in liquid chocolate, and then scratch holes in each spot where we normally place the ECG leads . . . . .

AREAS VIEWED by 12 LEAD ECG



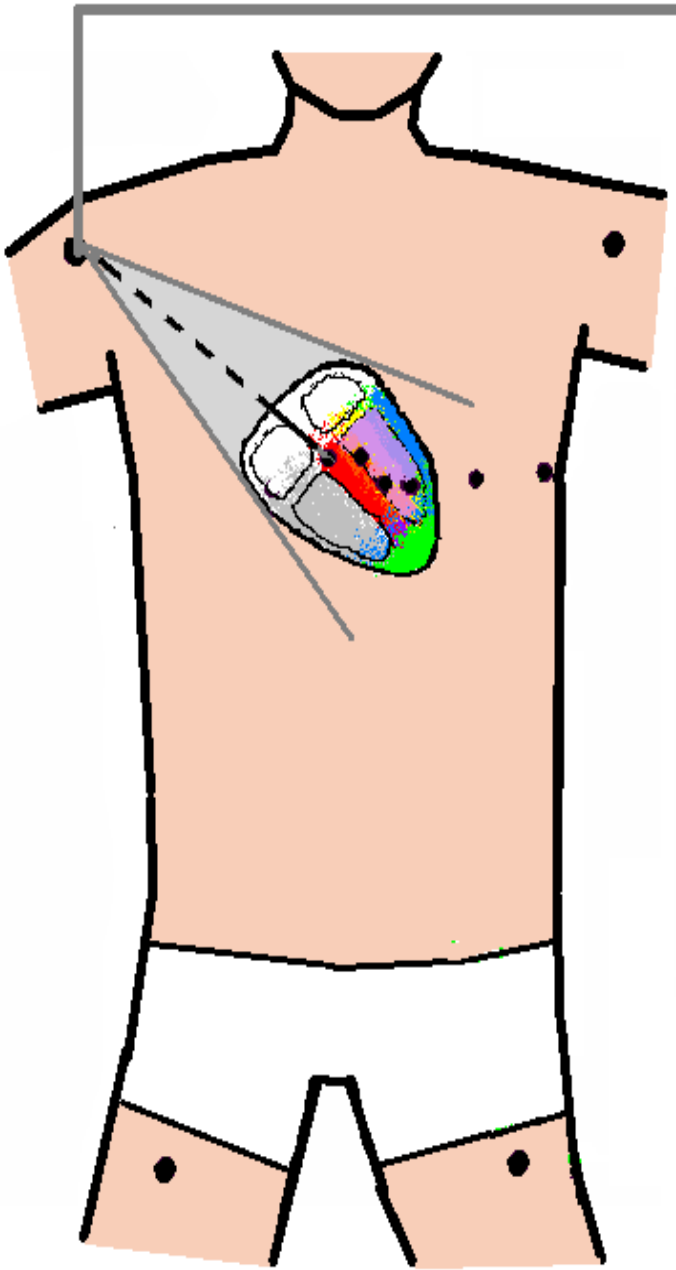
AVR
AVL, I
V1, V2
V3, V4
V5, V6
II, III, AVF

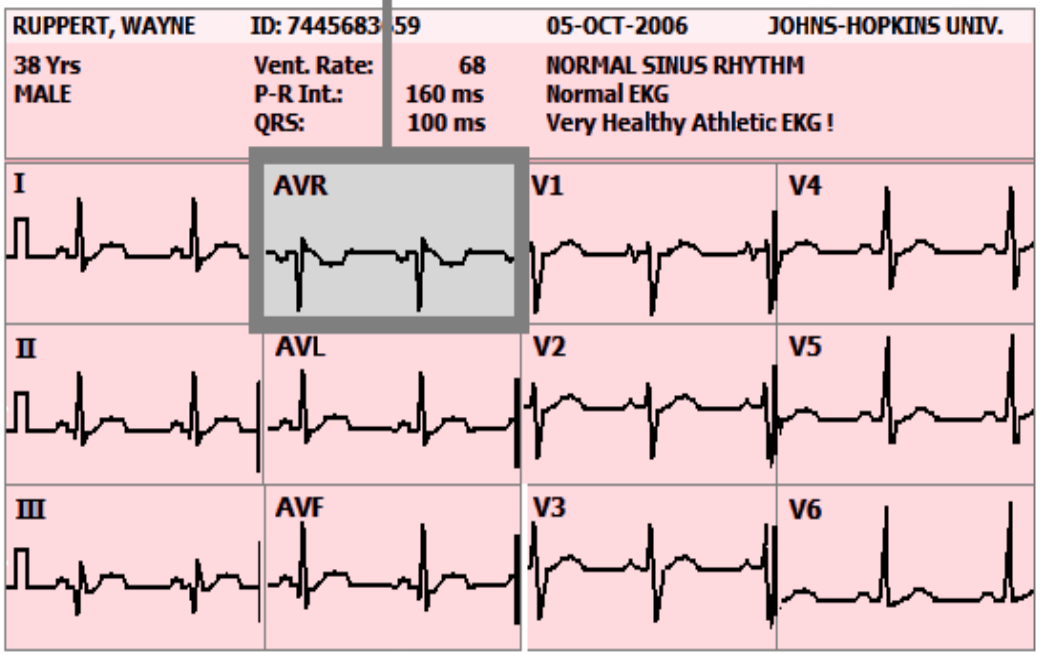
THE POSITIVE ELECTRODE



IS THE "EYE" . . .

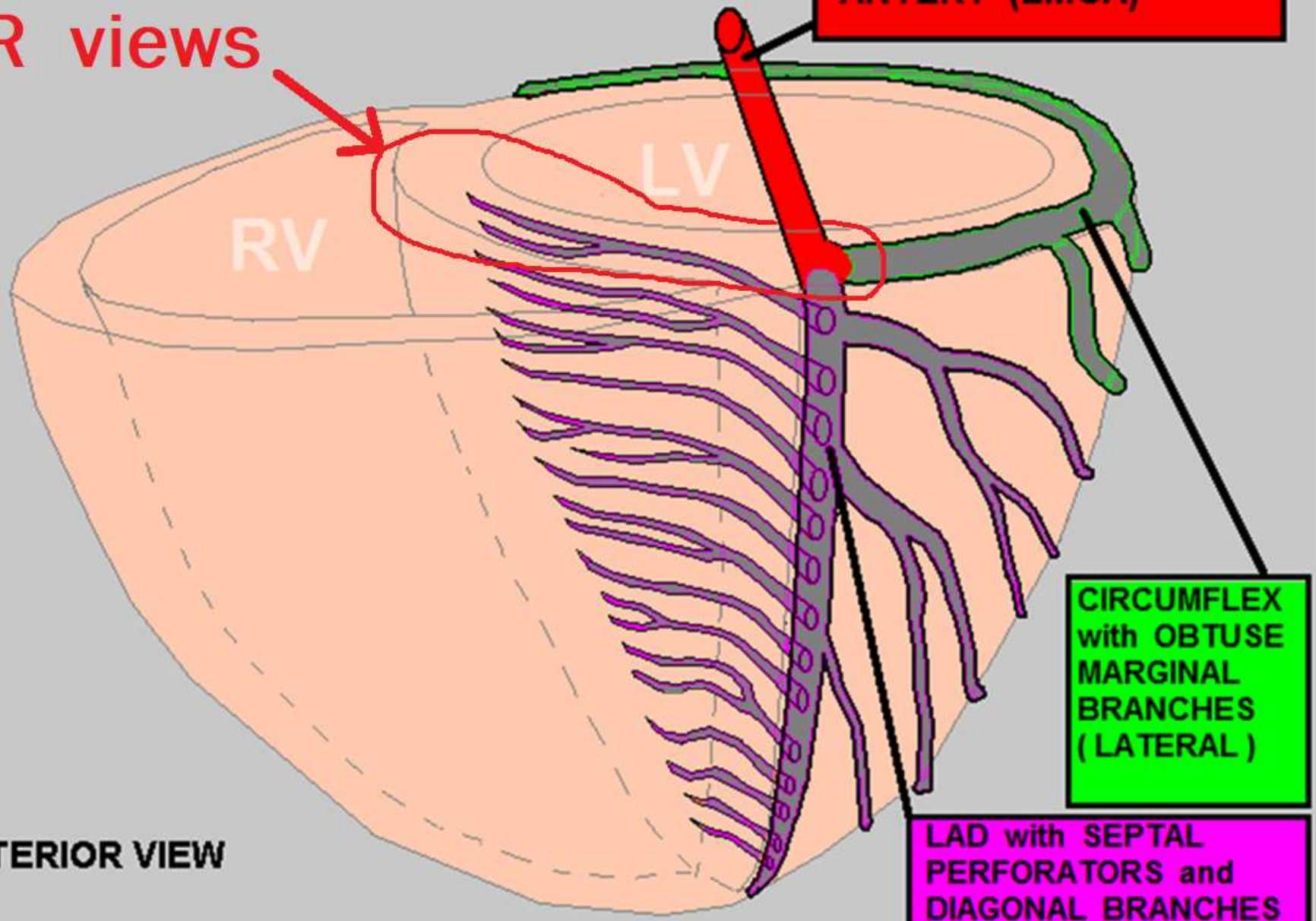
Lead AVR Views the BASILAR SEPTUM (region of the Bundle of His)



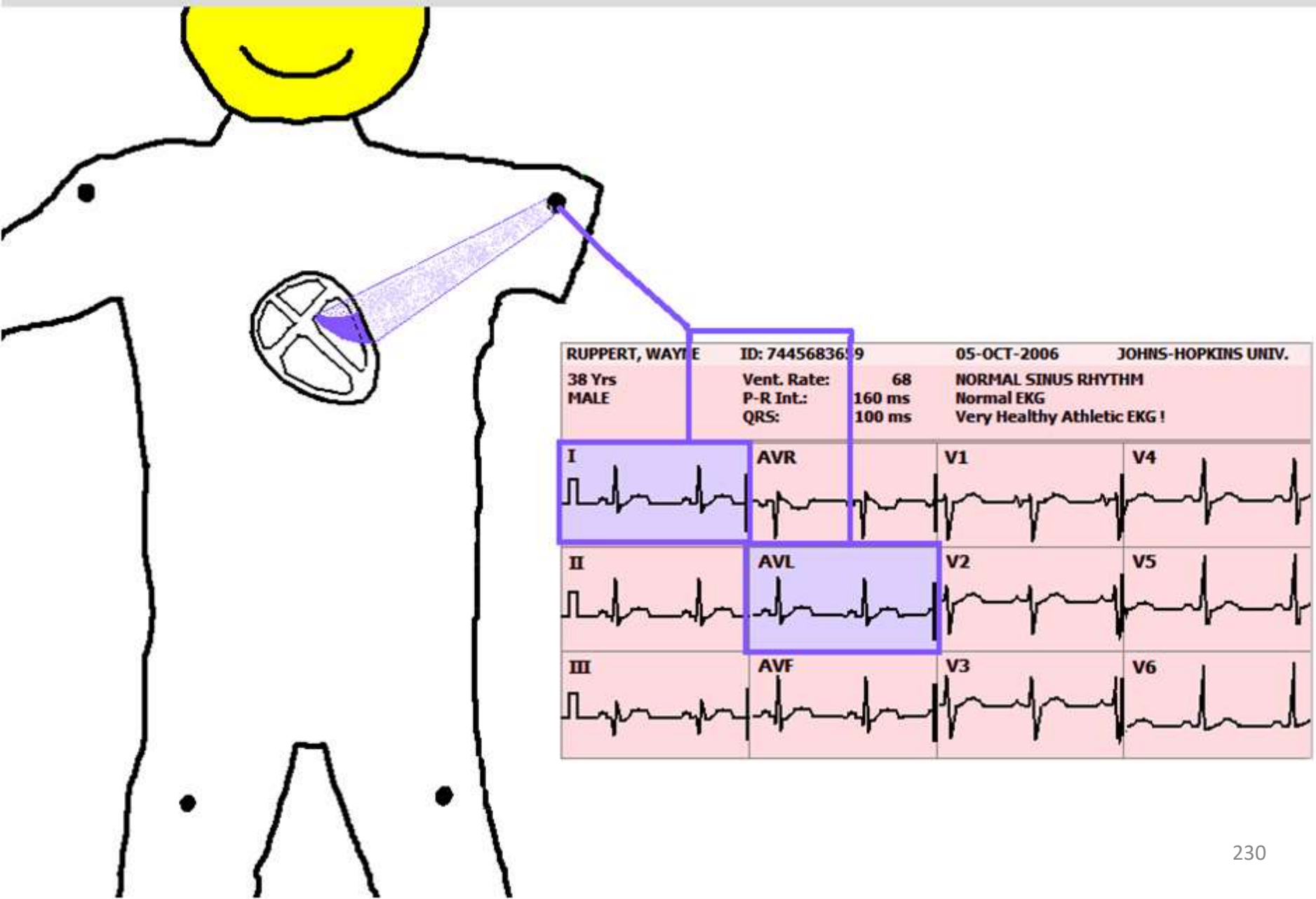


LEFT CORONARY ARTERY SYSTEM

AVR views

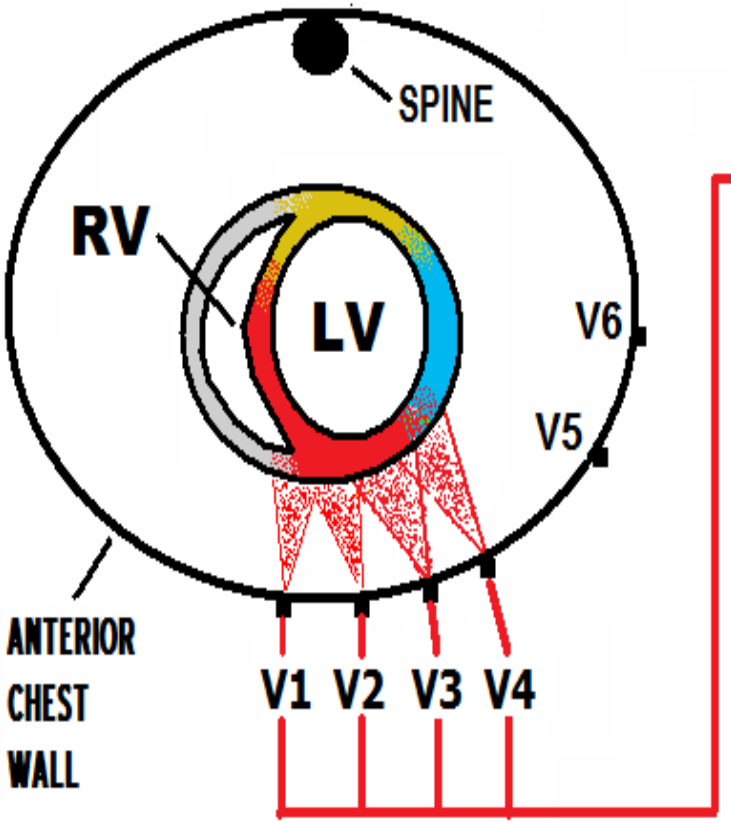


LEADS I and aVL VIEW the LATERAL - ANTERIOR WALL



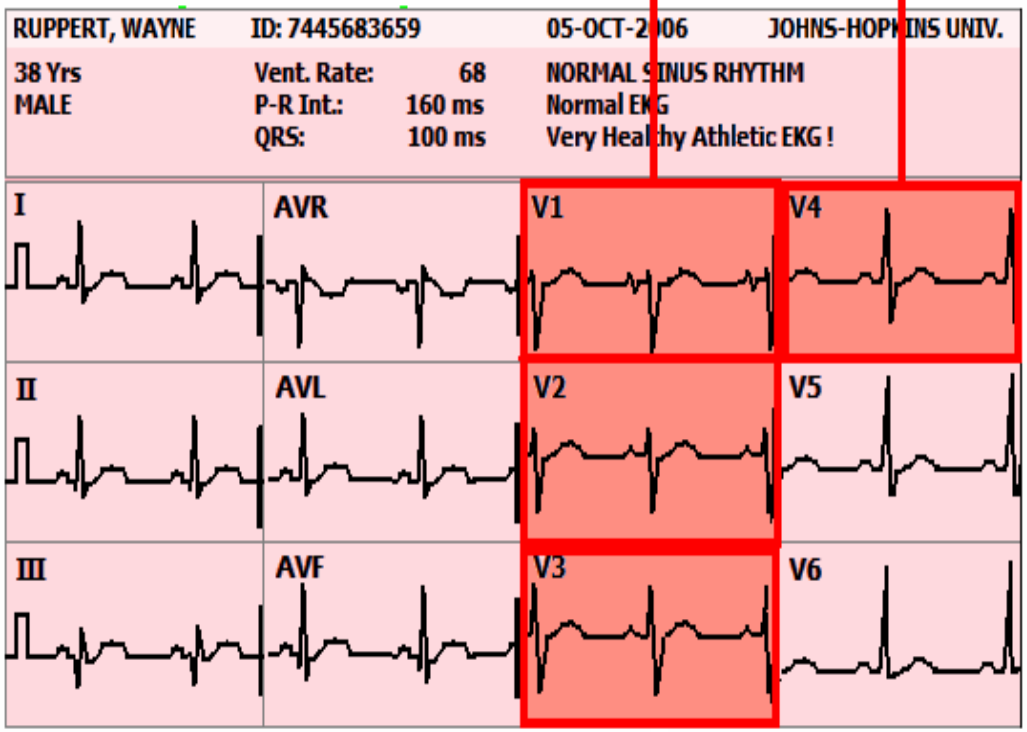
V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL

of the LEFT VENTRICLE



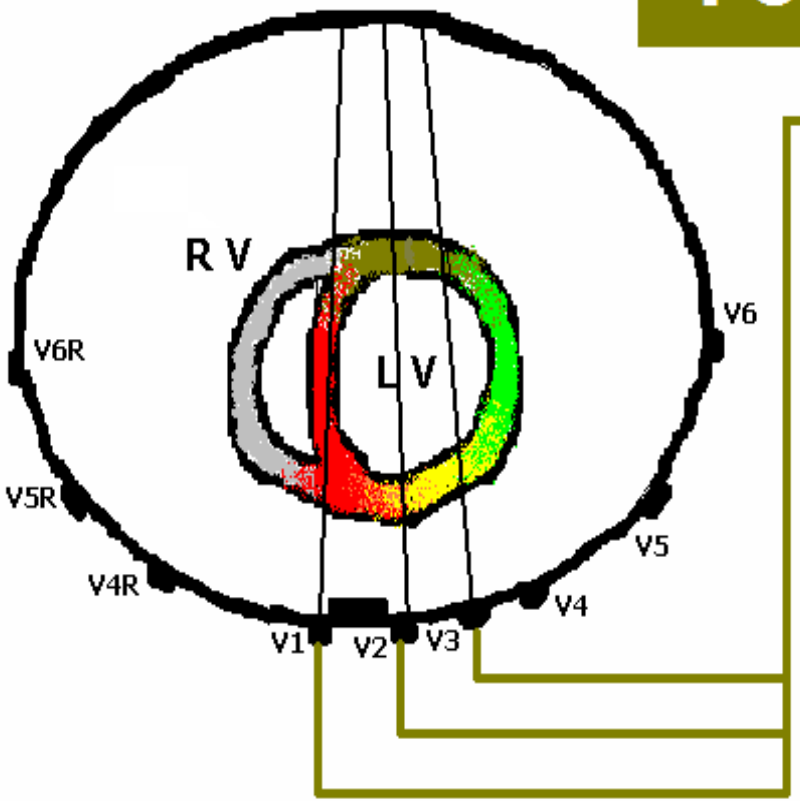
V1, V2 - ANTERIOR / SEPTAL

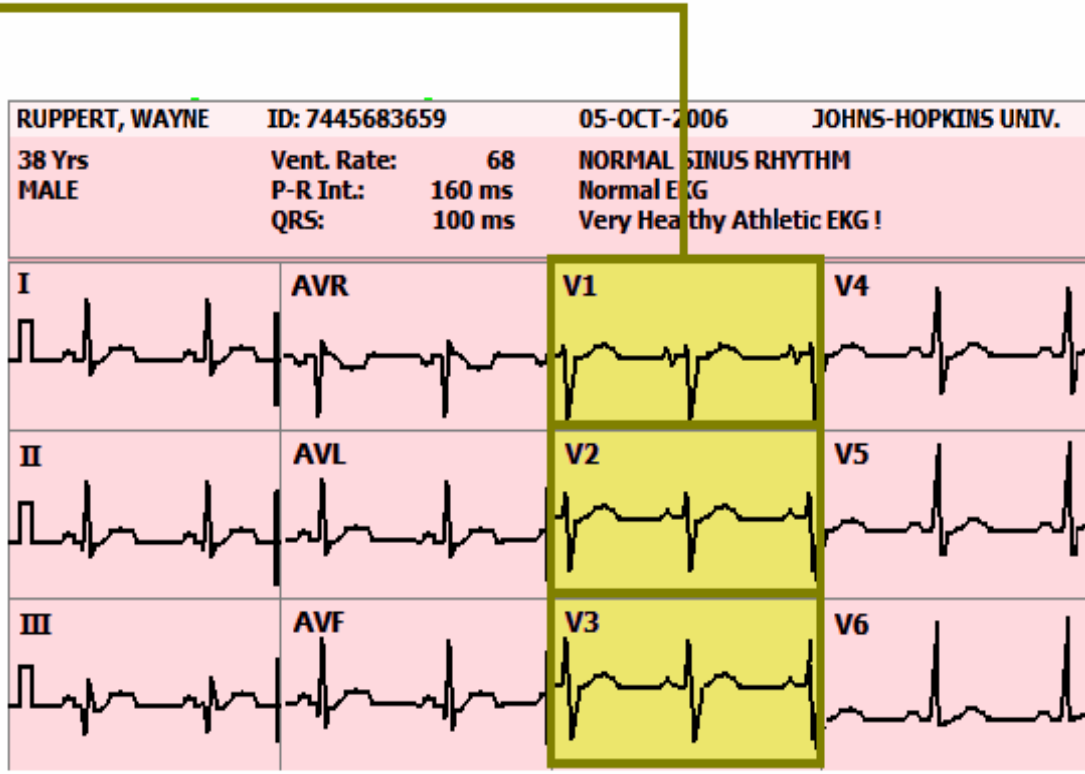
V3, V4 - ANTERIOR



LEADS V1 - V3 *view the*

POSTERIOR WALL



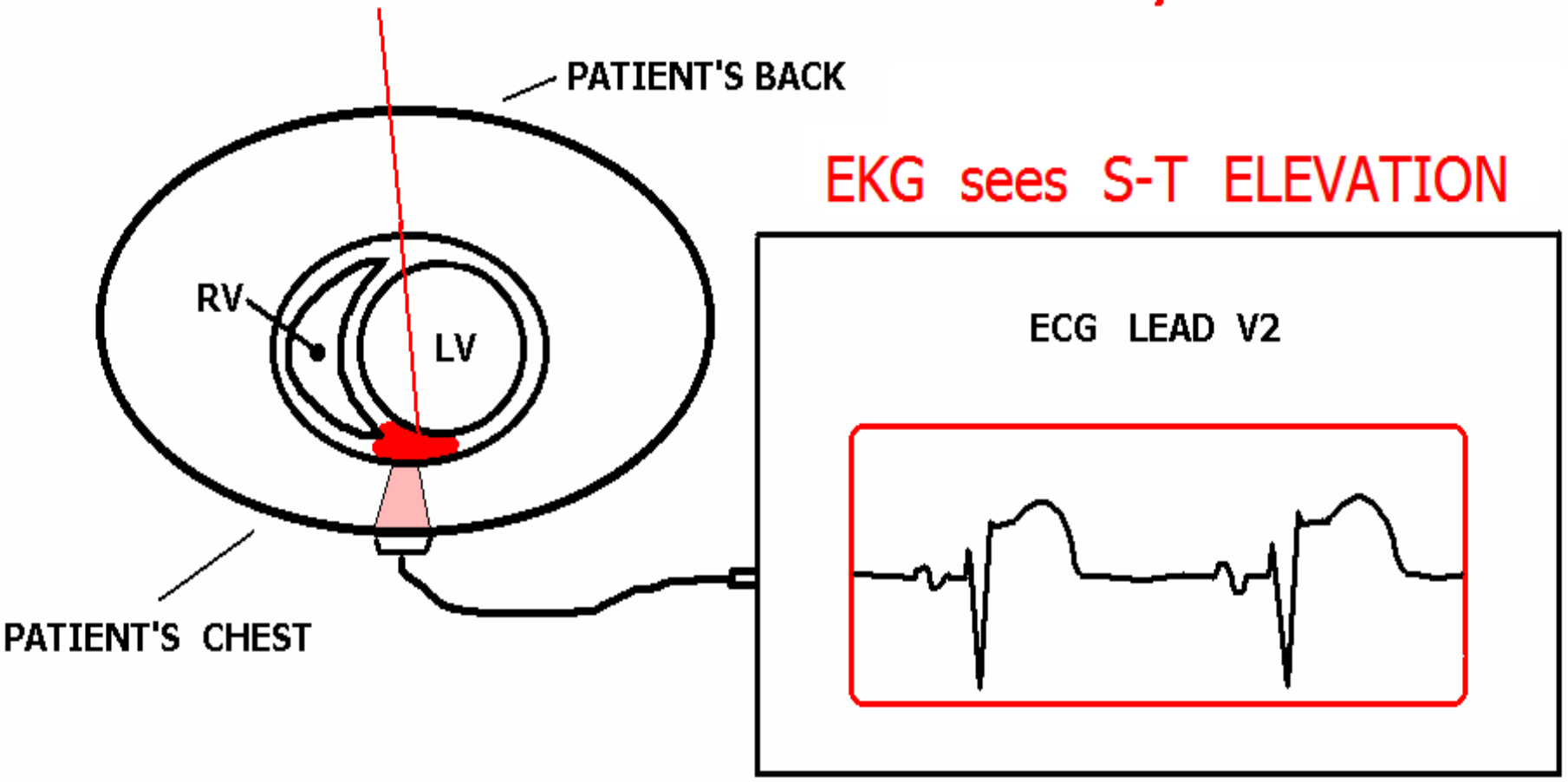


via RECIPROCAL CHANGES.

HOW EKG VIEWS INDICATIVE CHANGES

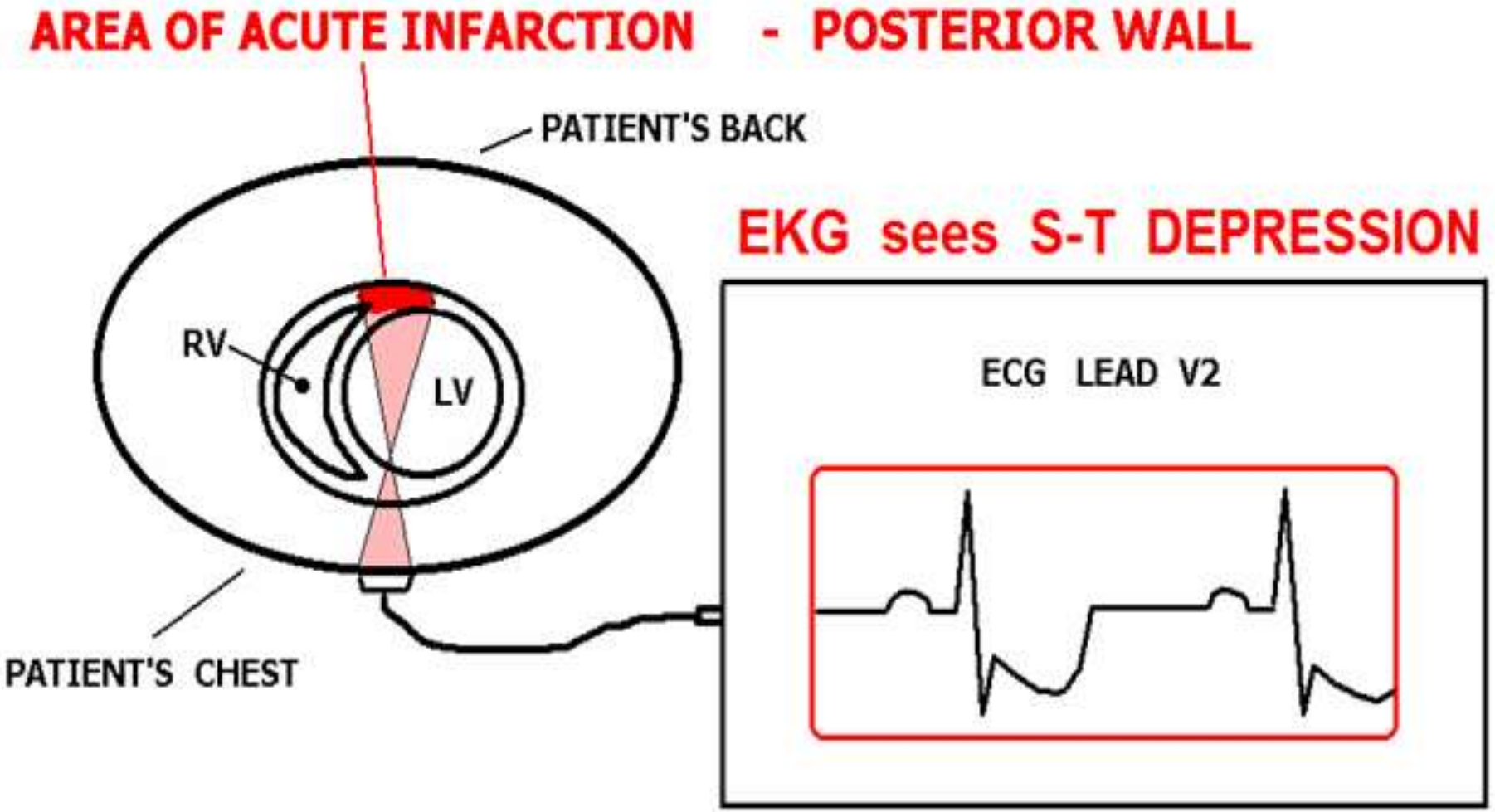
EXAMPLE:

AREA OF ACUTE INFARCTION - ANTERIOR/SEPTAL

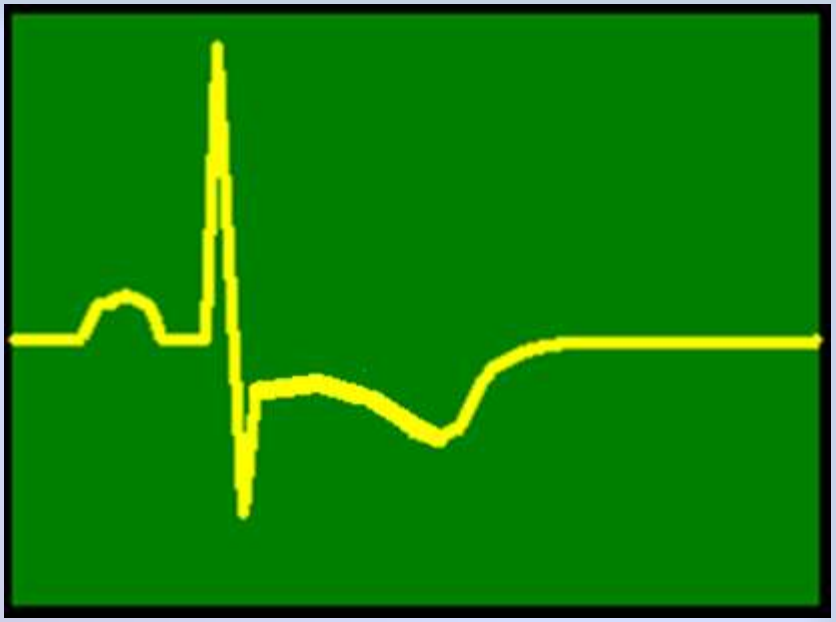


HOW EKG VIEWS RECIPROCAL CHANGES

EXAMPLE:



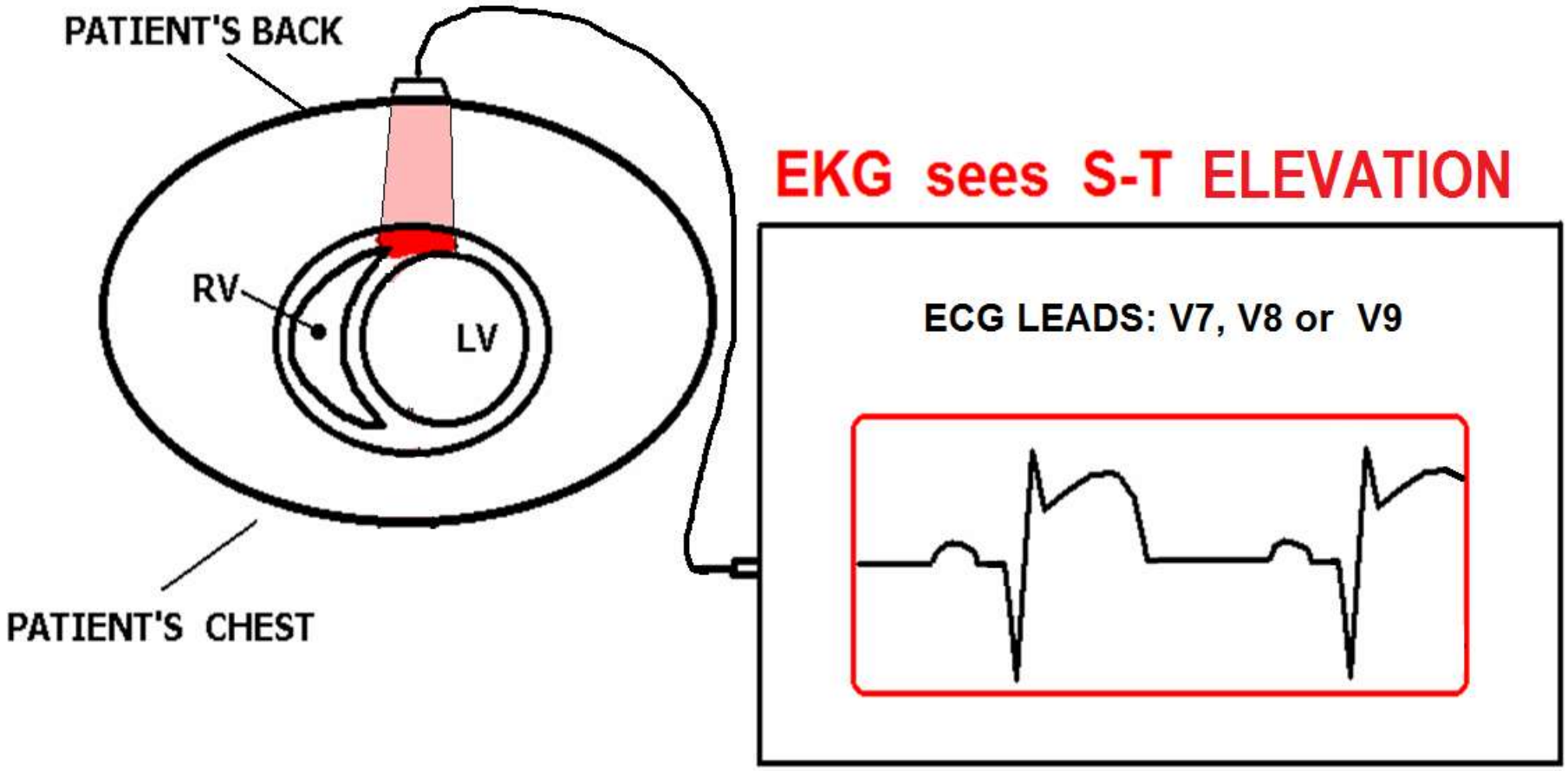
ST Depression can indicate:



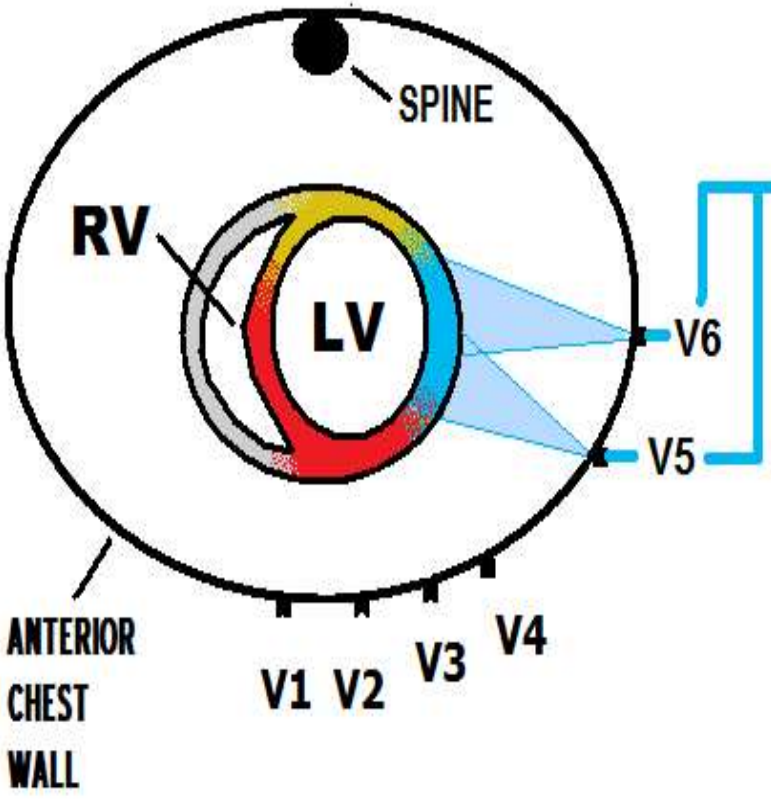
- ISCHEMIA
- “Partial-wall thickness” MI (NSTEMI)
- STEMI (in the opposite side of the heart)
- Other things (like RBBB, certain medications, etc).

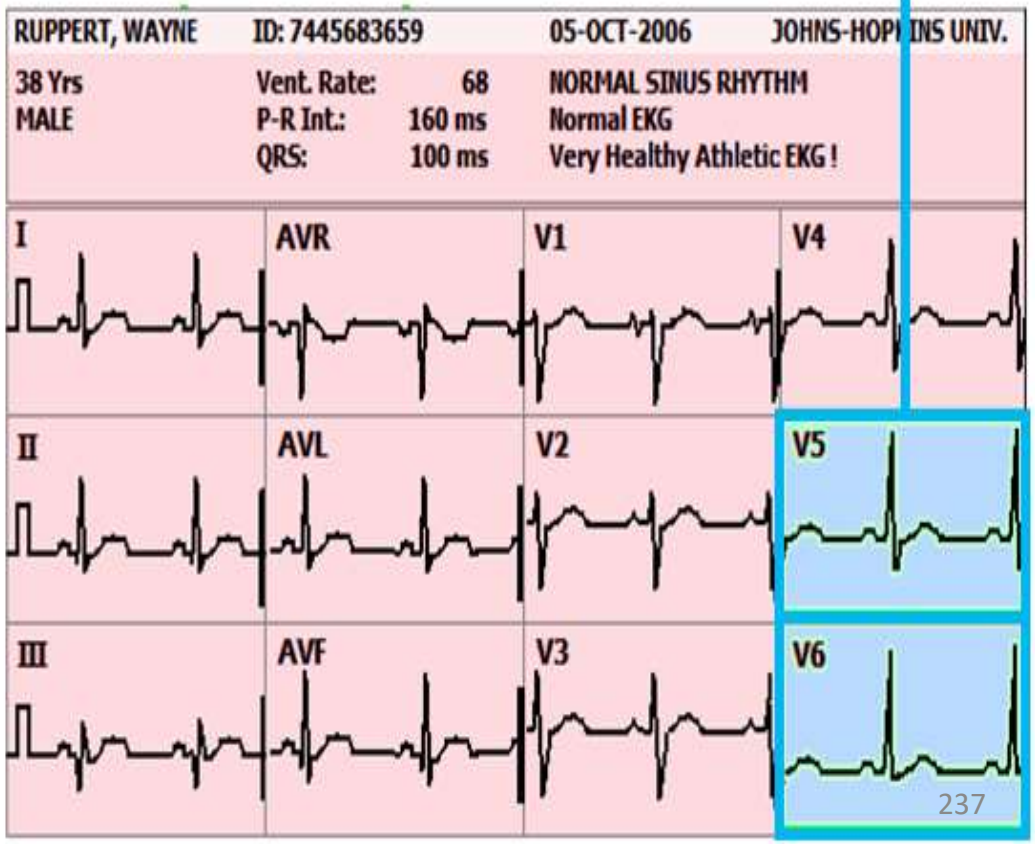
If we put ECG leads on the BACK of a PATIENT who is having an

ACUTE POSTERIOR WALL MI . . . . .

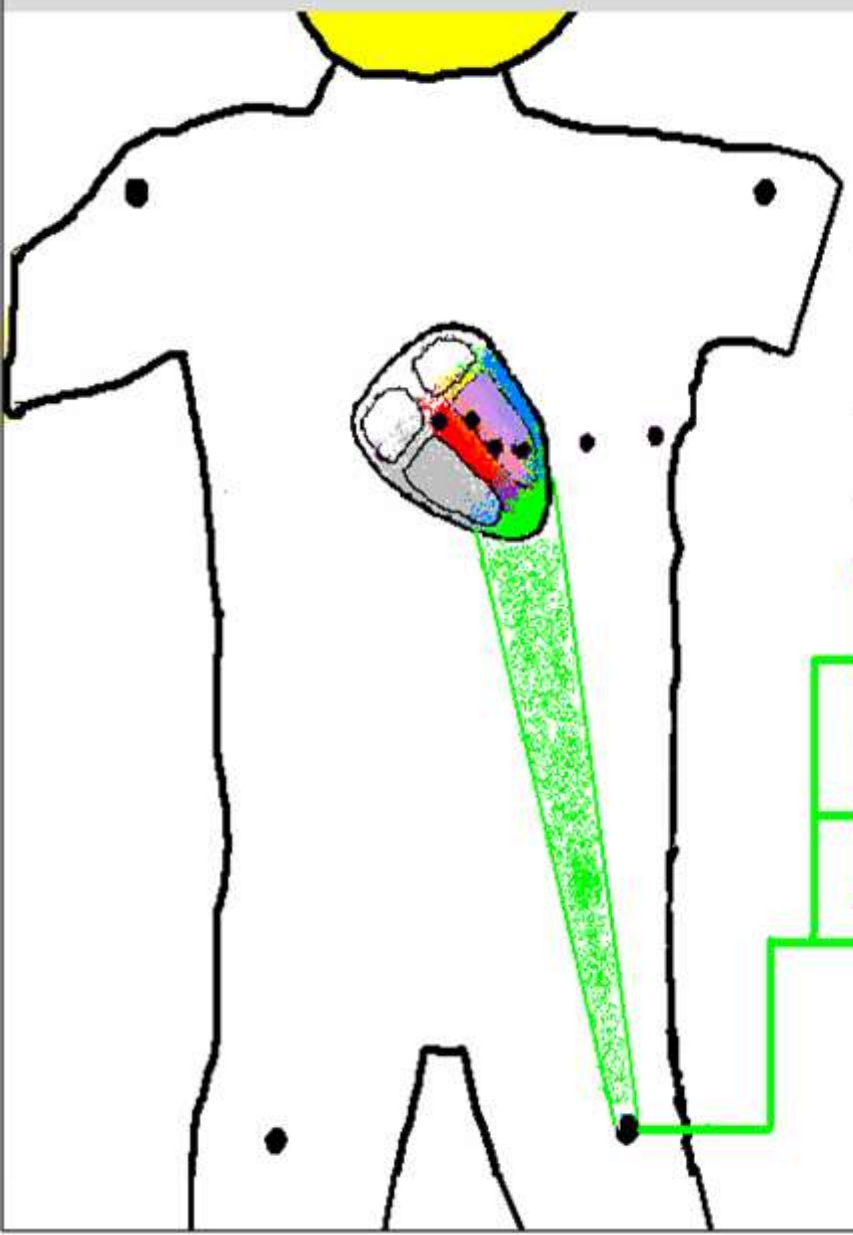


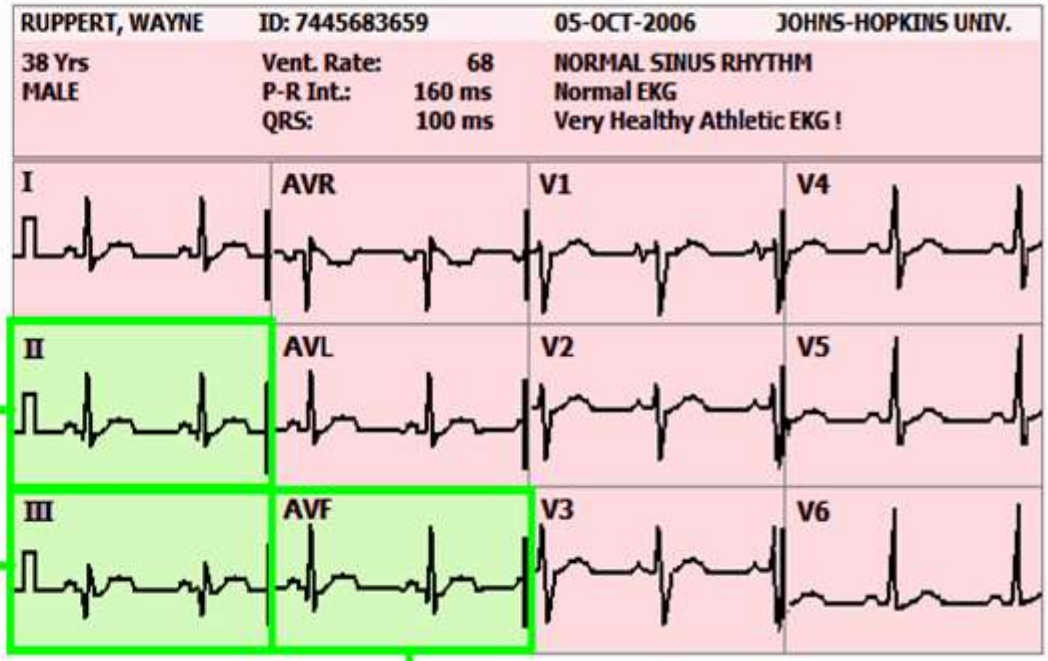
V5 - V6 VIEW THE LATERAL WALL
of the LEFT VENTRICLE

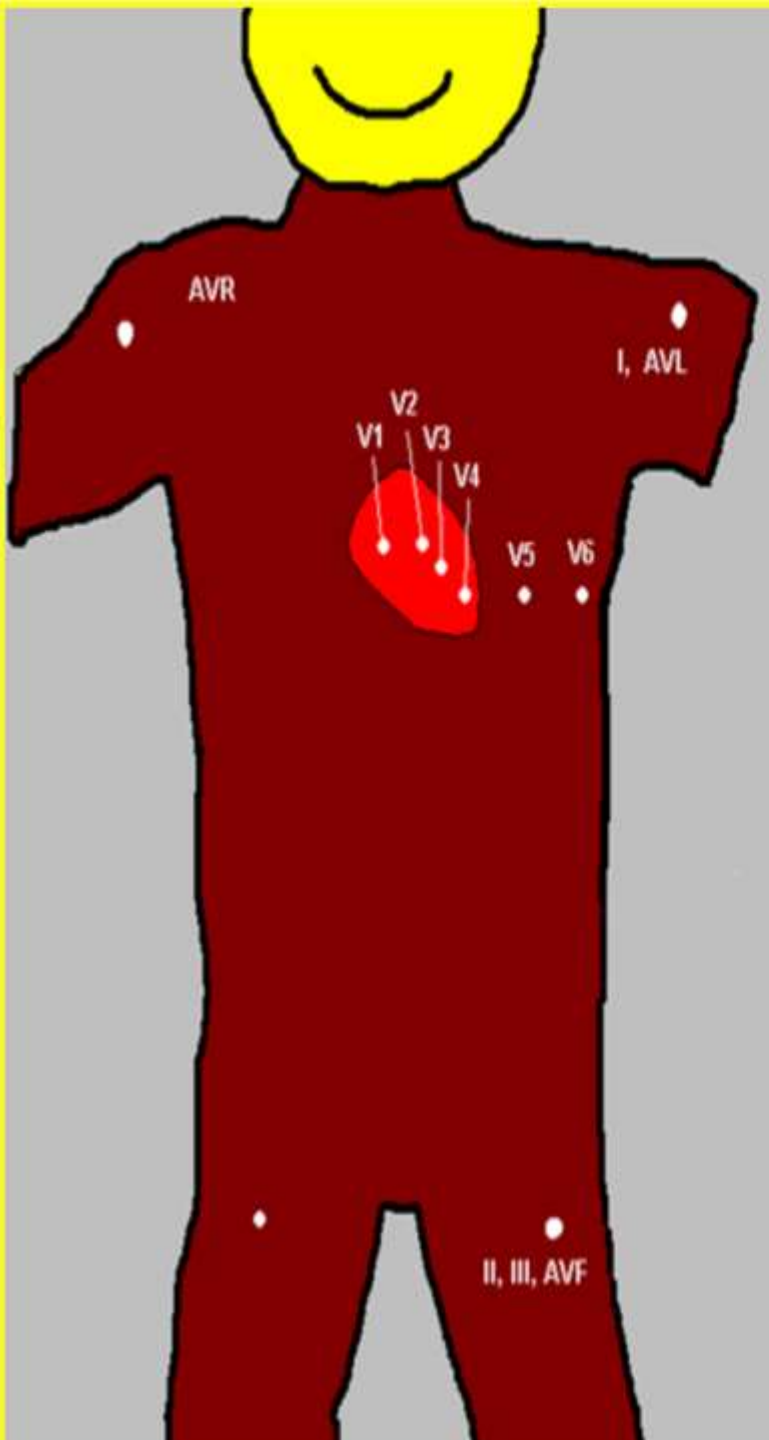




LEADS II, III, and aVF VIEW
INFERIOR WALL of the LEFT VENTRICLE







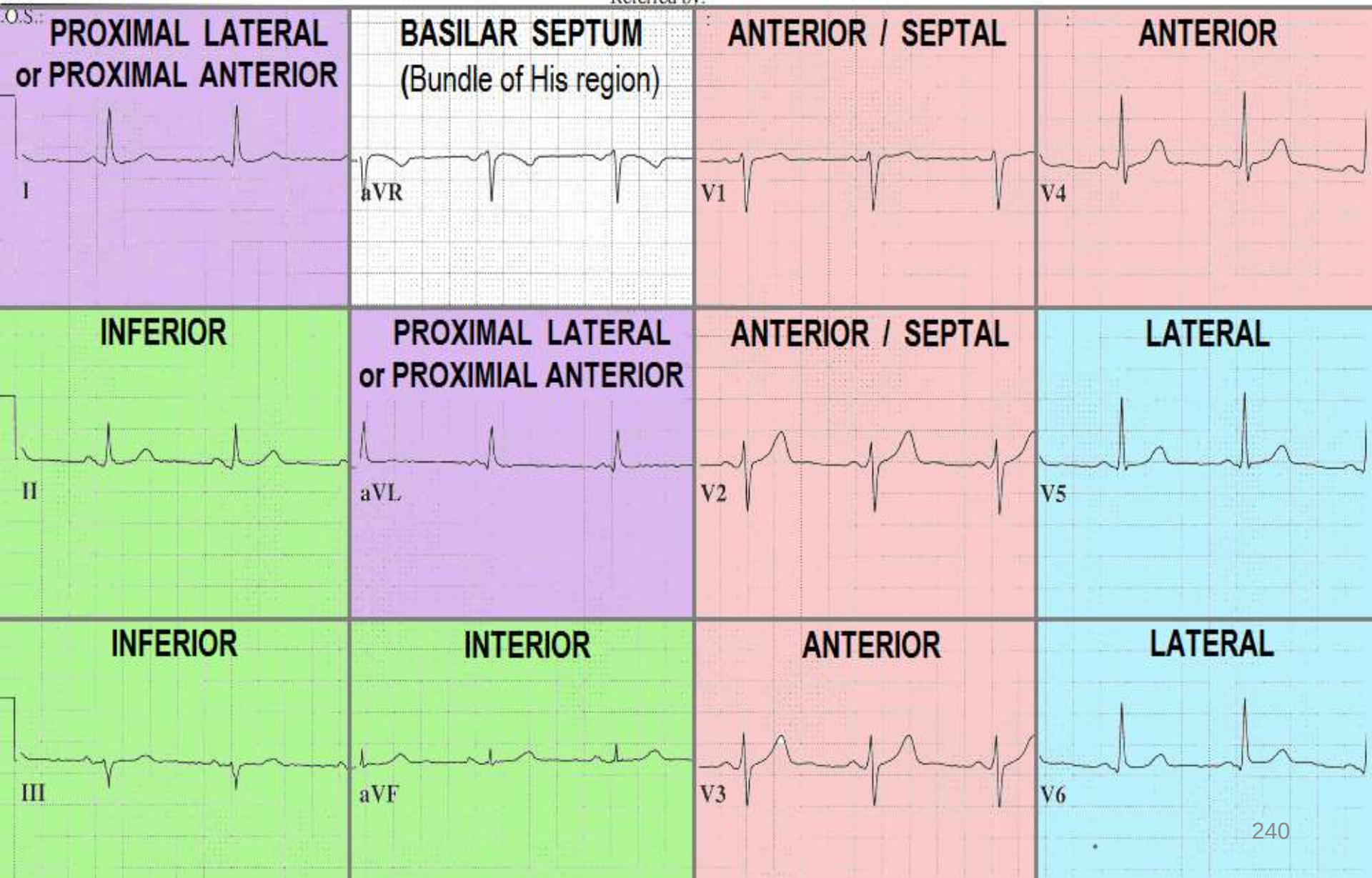
AREAS VIEWED by 12 LEAD ECG

AVR	*BASILAR SEPTAL*
AVL, I	LATERAL ANTERIOR
V1, V2	ANTERIOR
	SEPTAL
	POSTERIOR (recip.)
V3, V4	ANTERIOR
V5, V6	LATERAL
II, III, AVF	INFERIOR

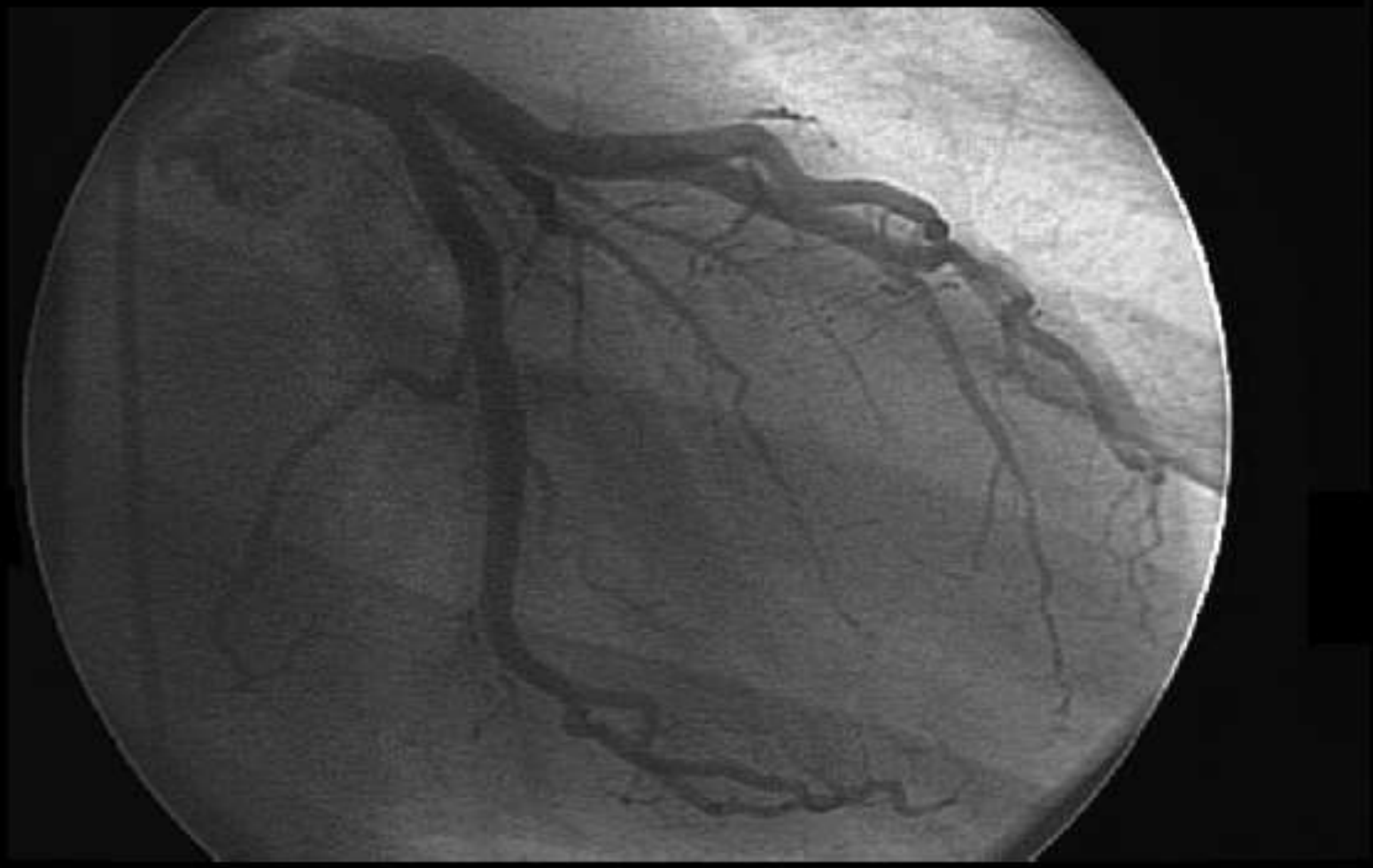
Vent. rate	64	BPM
PR interval	130	ms
QRS duration	96	ms
QT/QTc	396/408	ms
P–R–T axes	40 11	61

Normal sinus rhythm
Normal ECG
No previous ECGs available

Referred by:

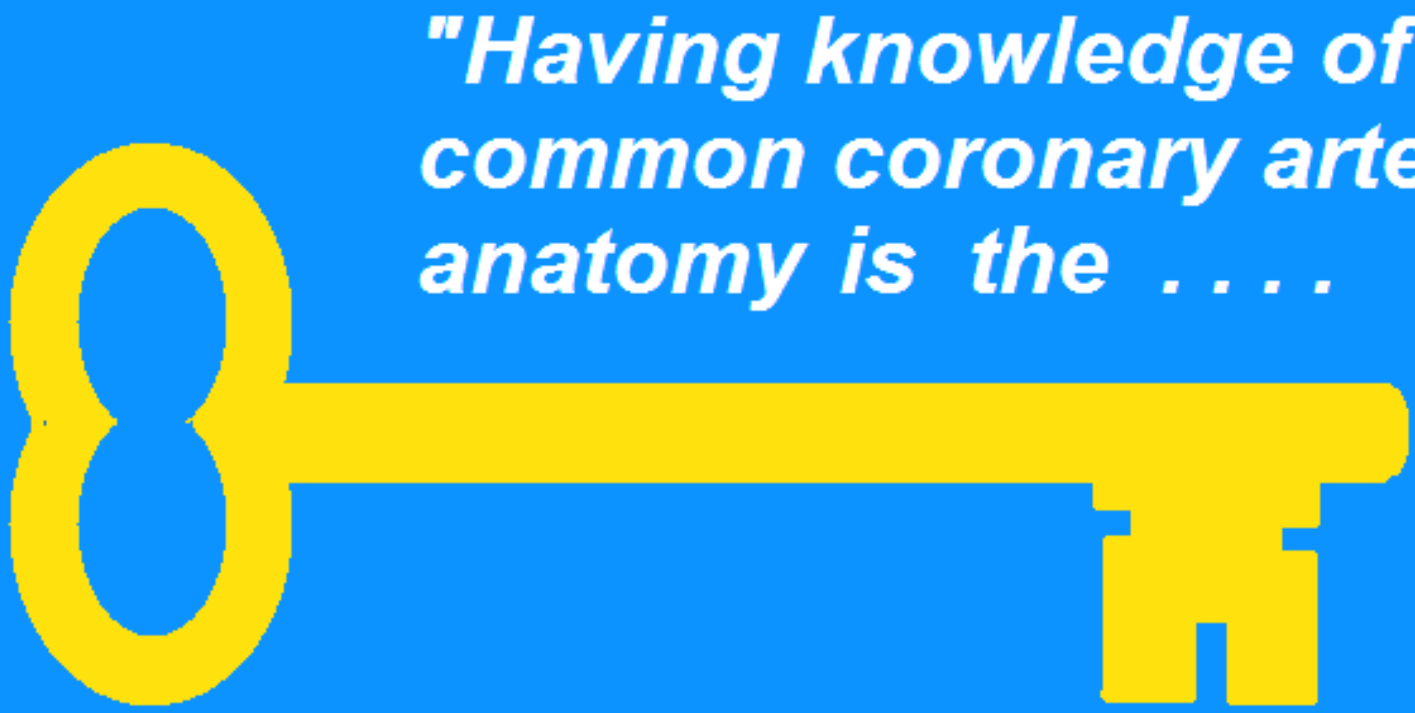


THE CORONARY



ARTERIES

STRUCTURES SERVED BY THE CORONARY ARTERIES



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

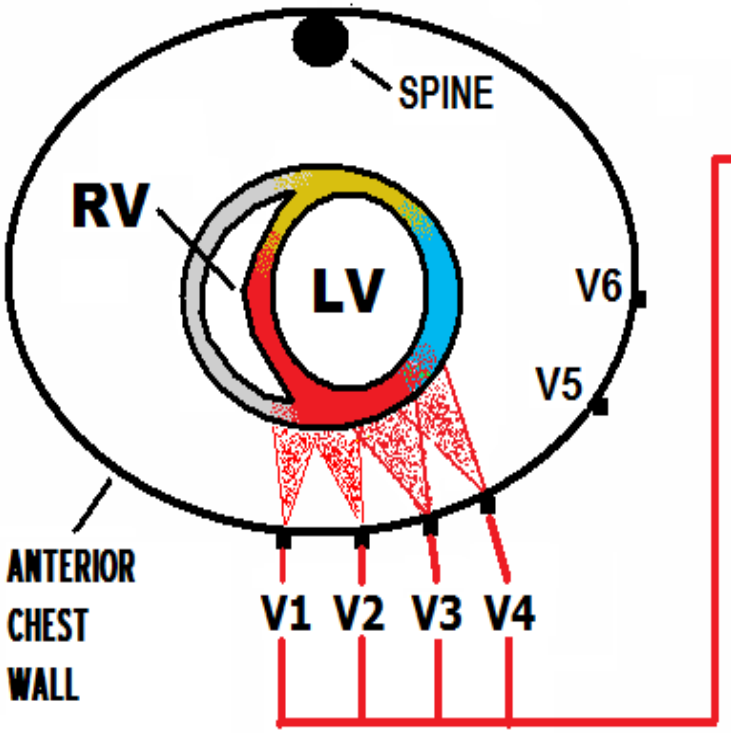
The 12 Lead ECG becomes your "crystal ball !!"

INTERPRET THE EKG, THEN:

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

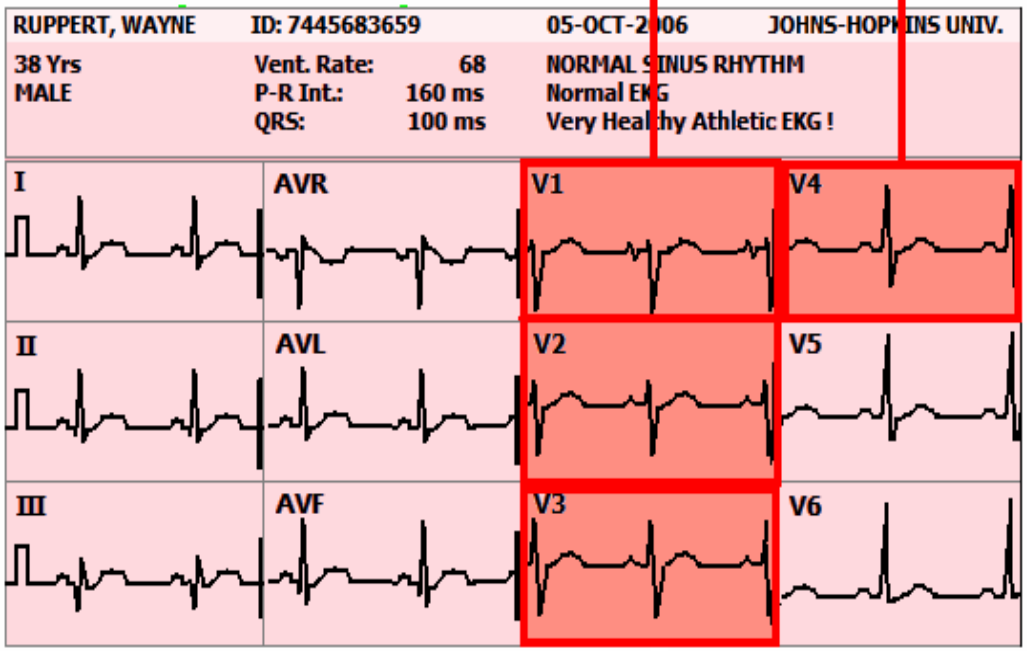
V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL

of the LEFT VENTRICLE



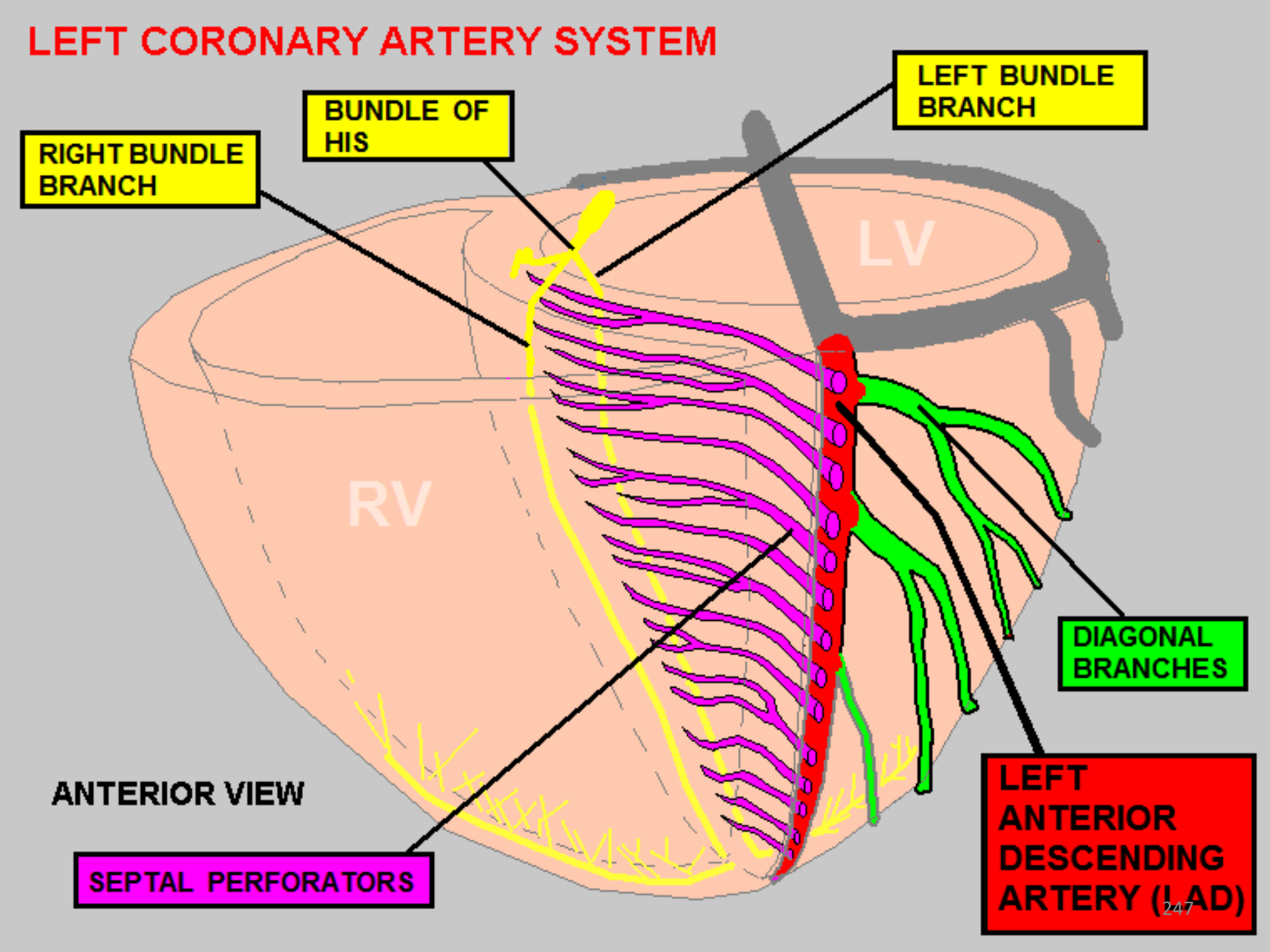
V1, V2 - ANTERIOR / SEPTAL

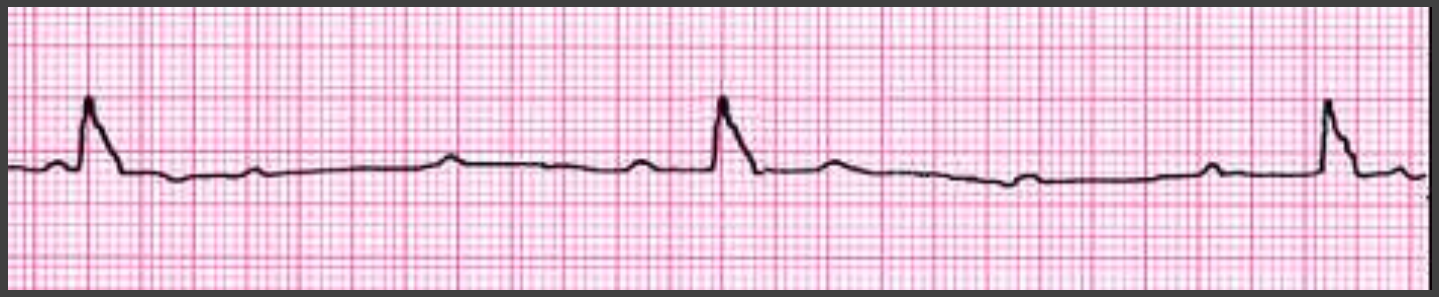
V3, V4 - ANTERIOR

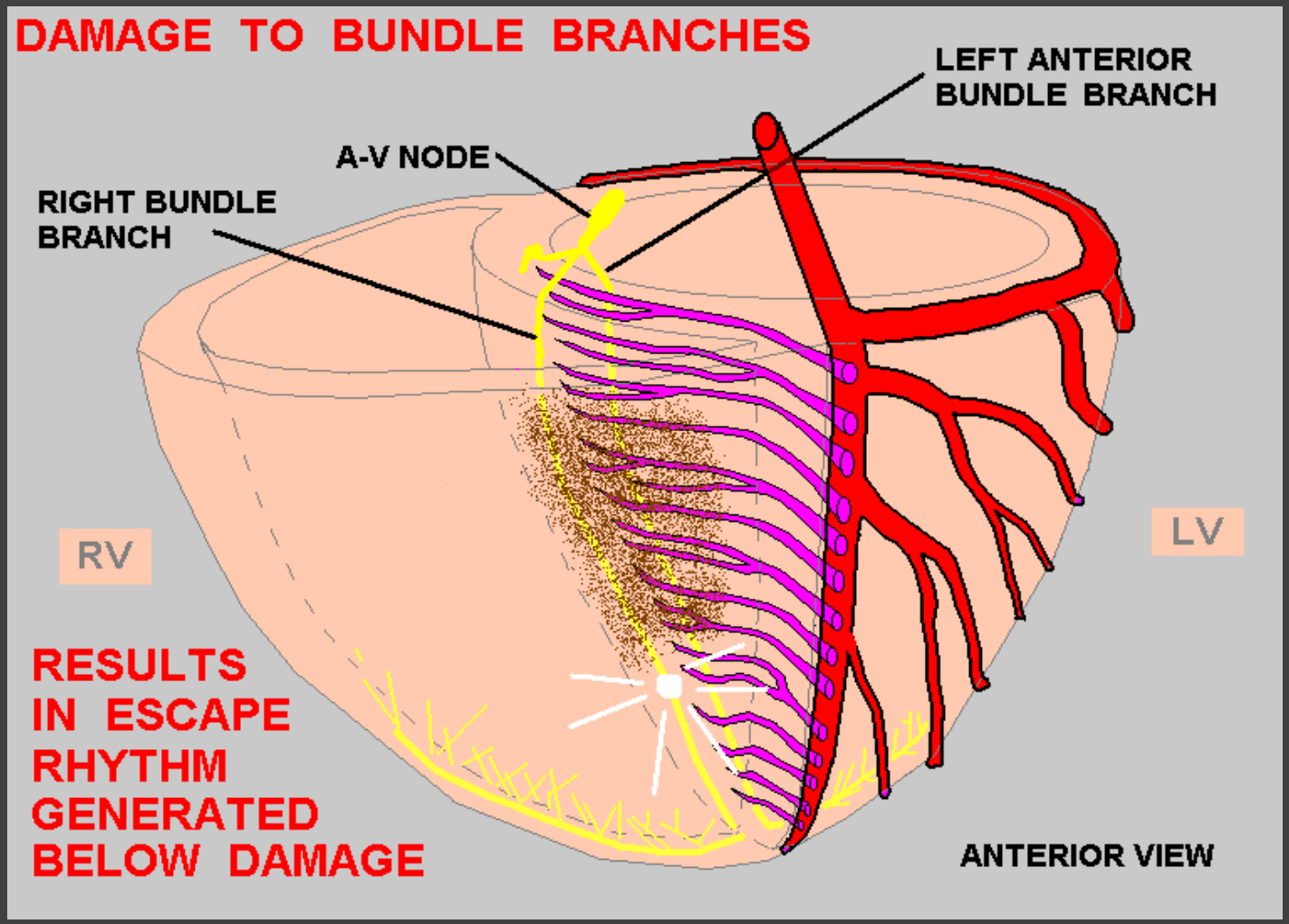


Which Coronary Artery typically Supplies the ANTERIOR WALL ?

LEFT CORONARY ARTERY SYSTEM



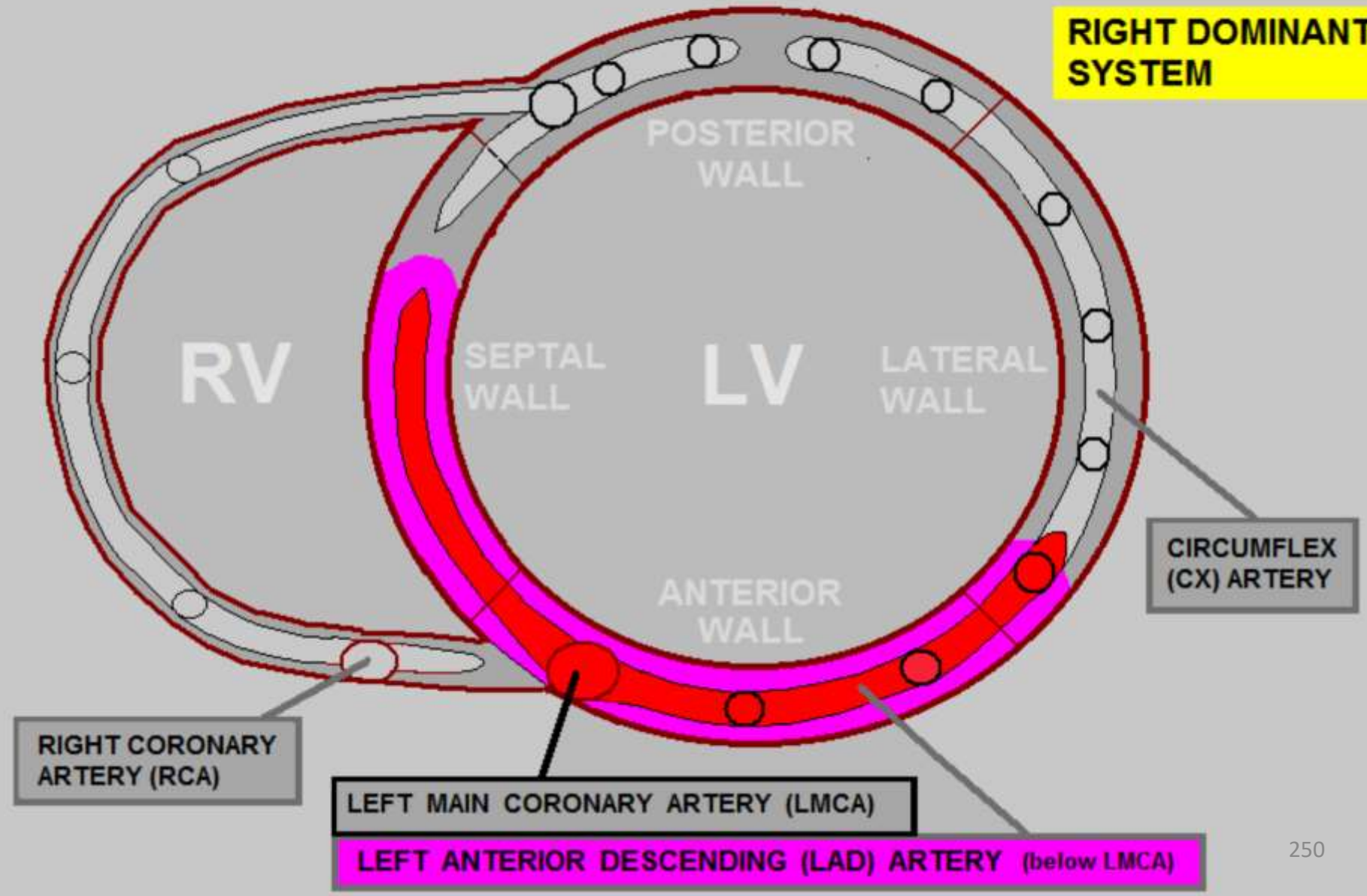



DAMAGE TO BUNDLE BRANCHES
LEFT ANTERIOR BUNDLE BRANCH
A-V NODE
RIGHT BUNDLE BRANCH
RV
LV
RESULTS IN ESCAPE RHYTHM GENERATED BELOW DAMAGE
ANTERIOR VIEW

cutaway view of the

LEFT ANTERIOR DESCENDING ARTERY (LAD)

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



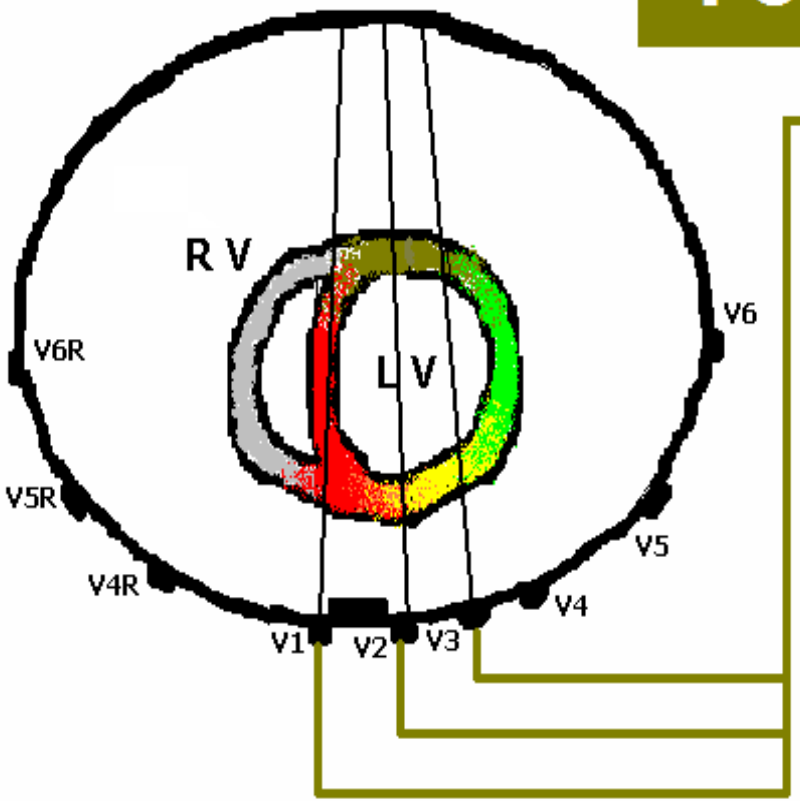
HELPFUL HINT . . . MEMORIZE THIS !

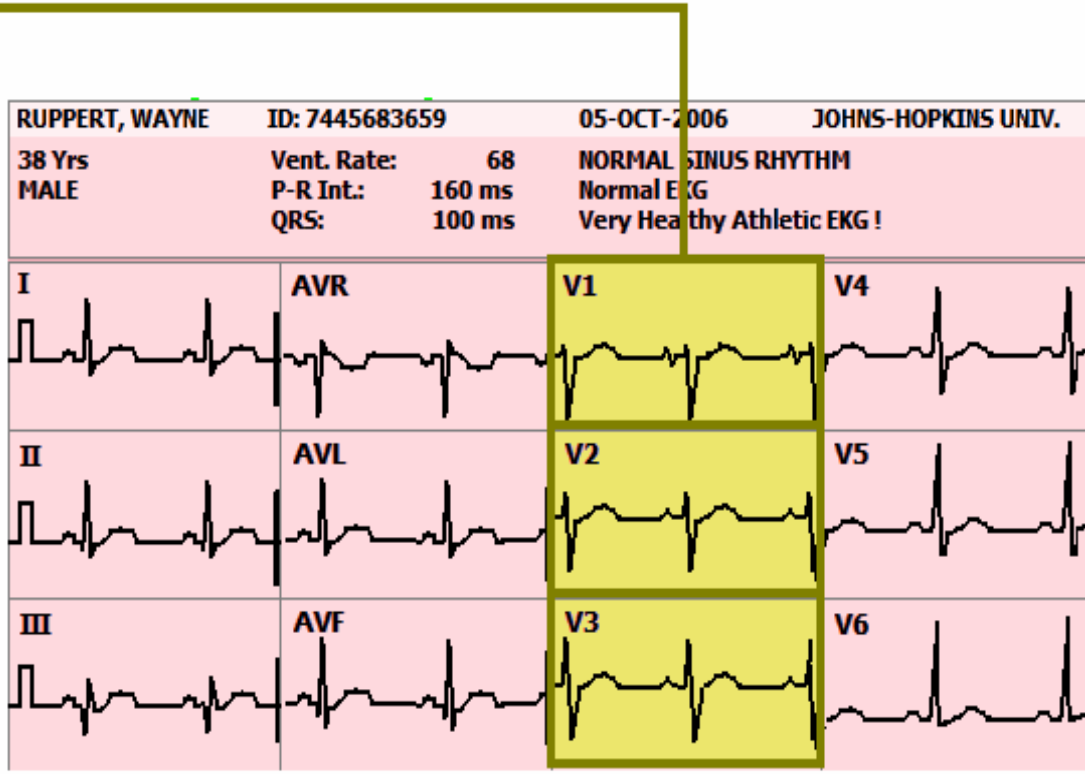
LEFT ANTERIOR DESCENDING ARTERY (LAD)

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

LEADS V1 - V3 *view the*

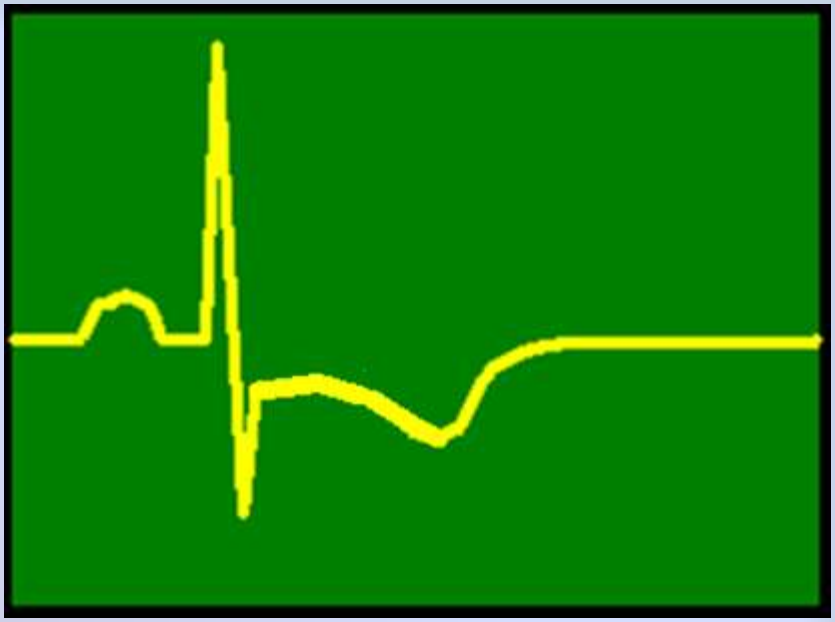
POSTERIOR WALL





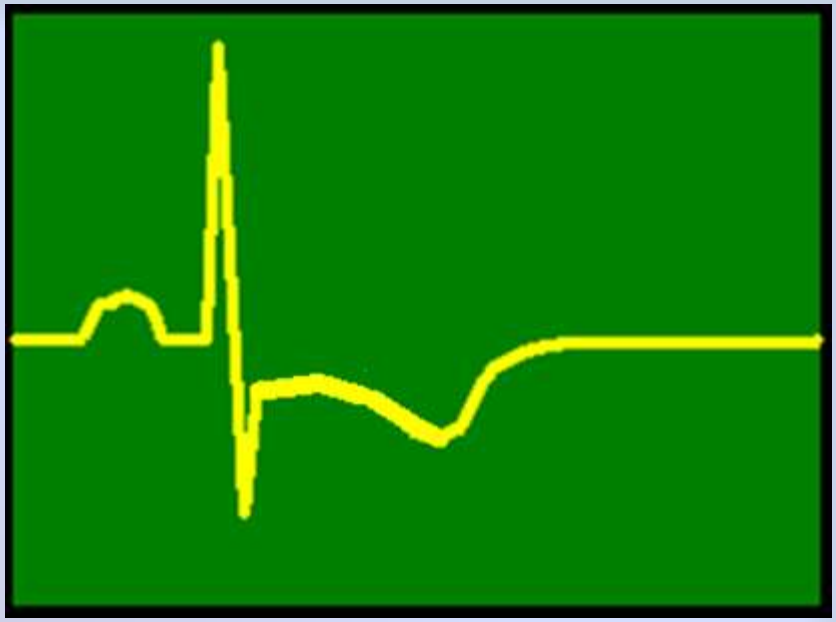
via RECIPROCAL CHANGES.

ST Depression in Leads V1 – V4:



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

ST Depression in Leads V1 – V4:

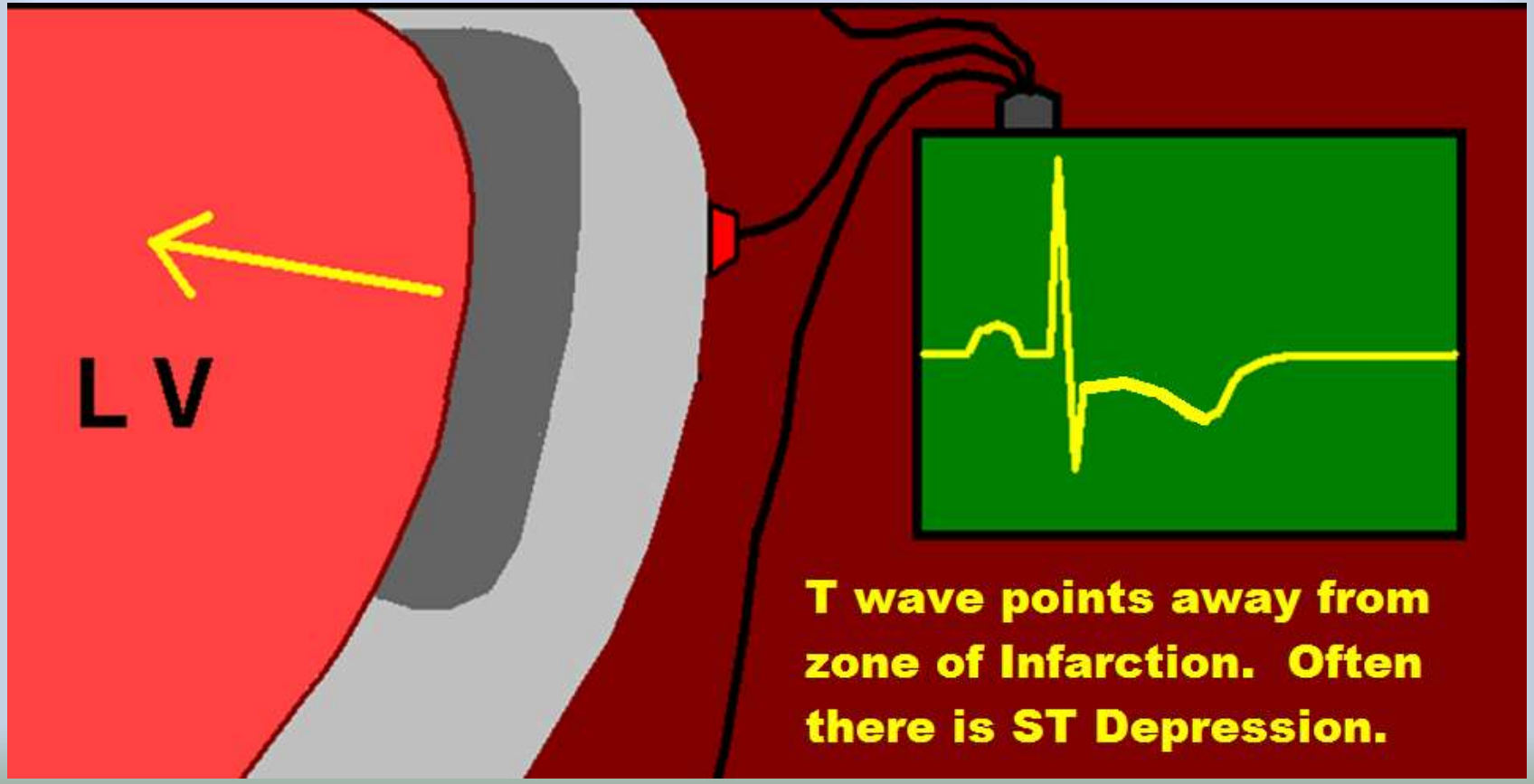


- Direct view of ISCHEMIA (anterior wall)
- Direct view of NSTEMI (anterior wall)

Non-STEMI (NSTEMI)

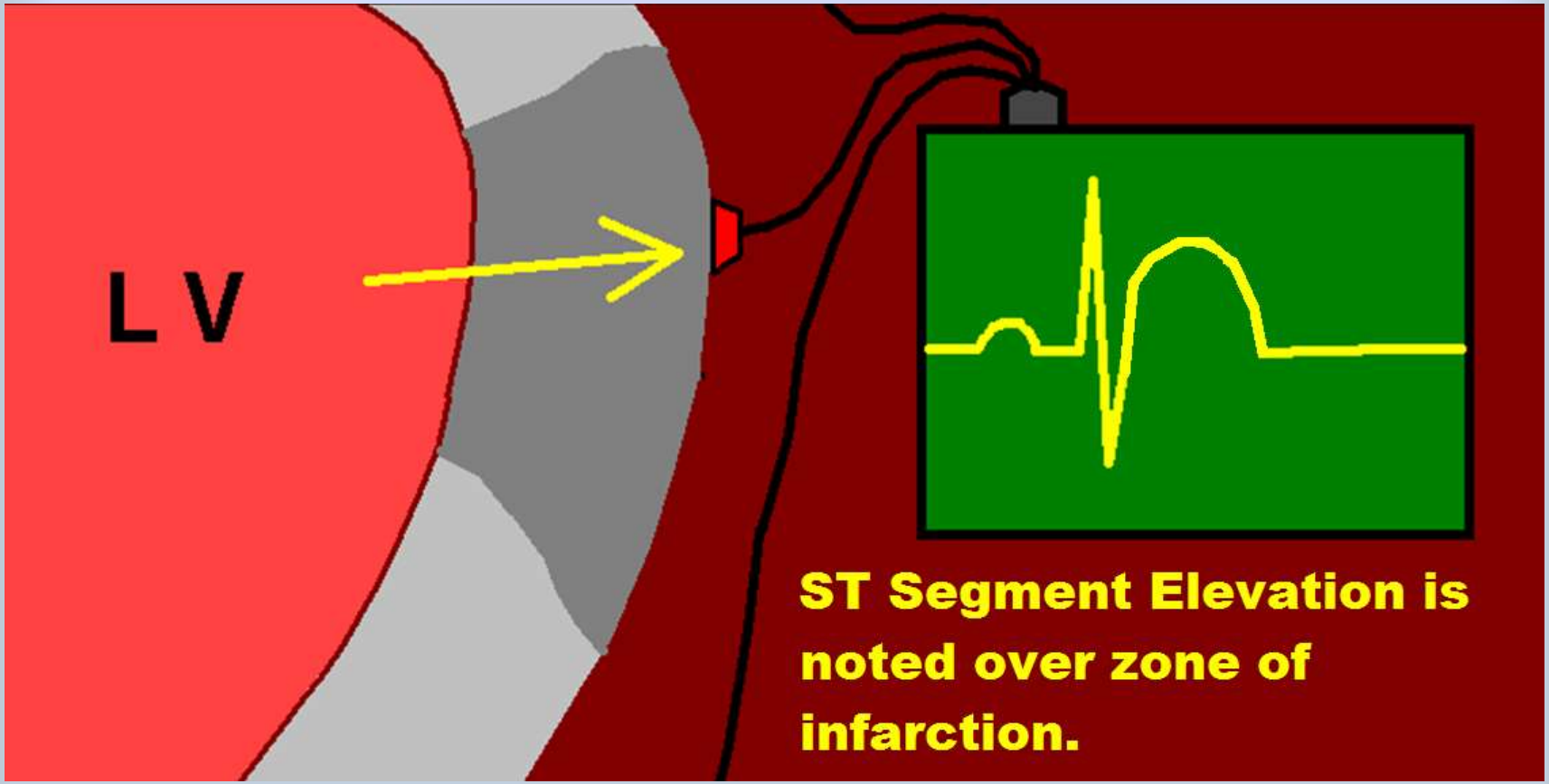
Non-ST Segment Elevation Myocardial Infarction.

"sub-endocardial MI" . . . "partial wall thickness"

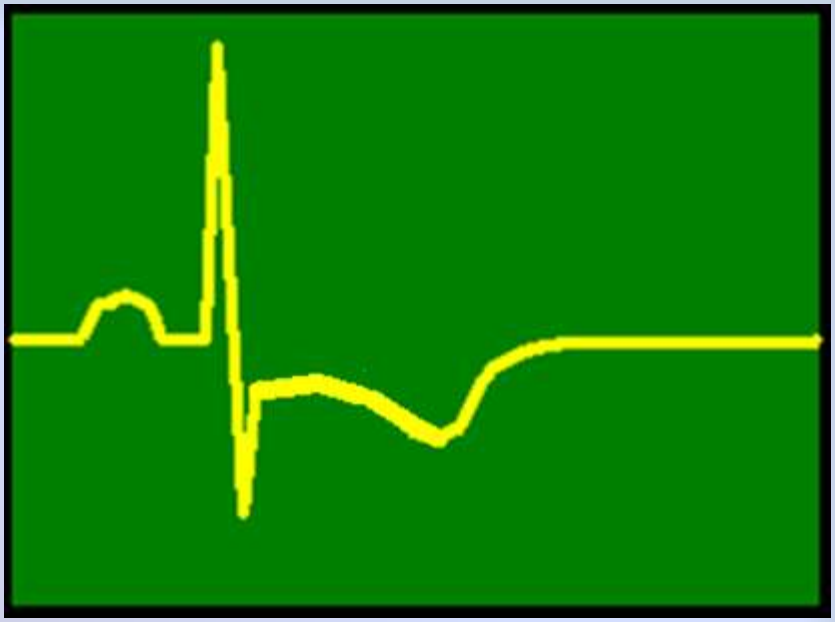


STEMI

- ST Segment Elevation Myocardial Infarction.



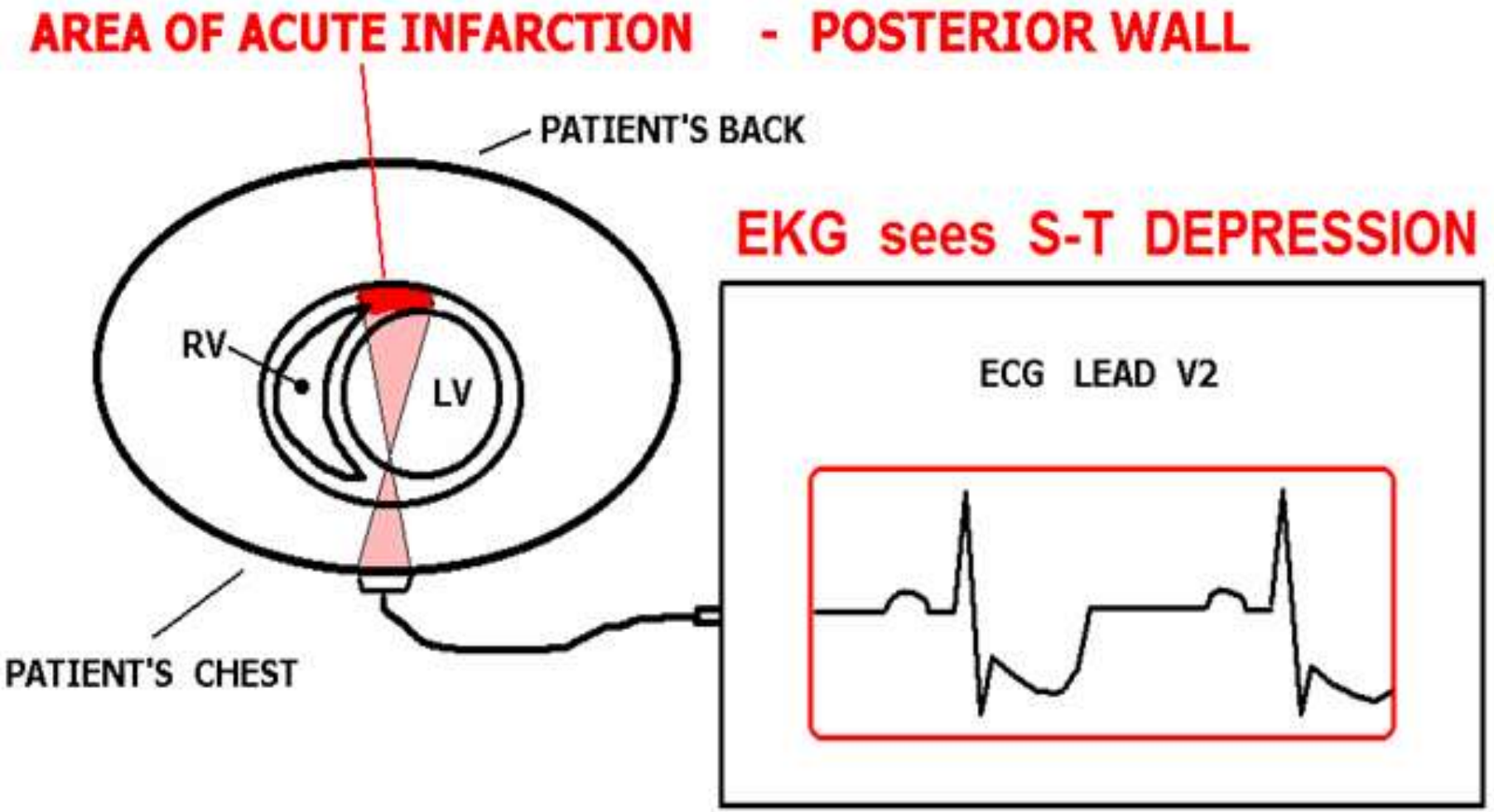
ST Depression in Leads V1 – V4:



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

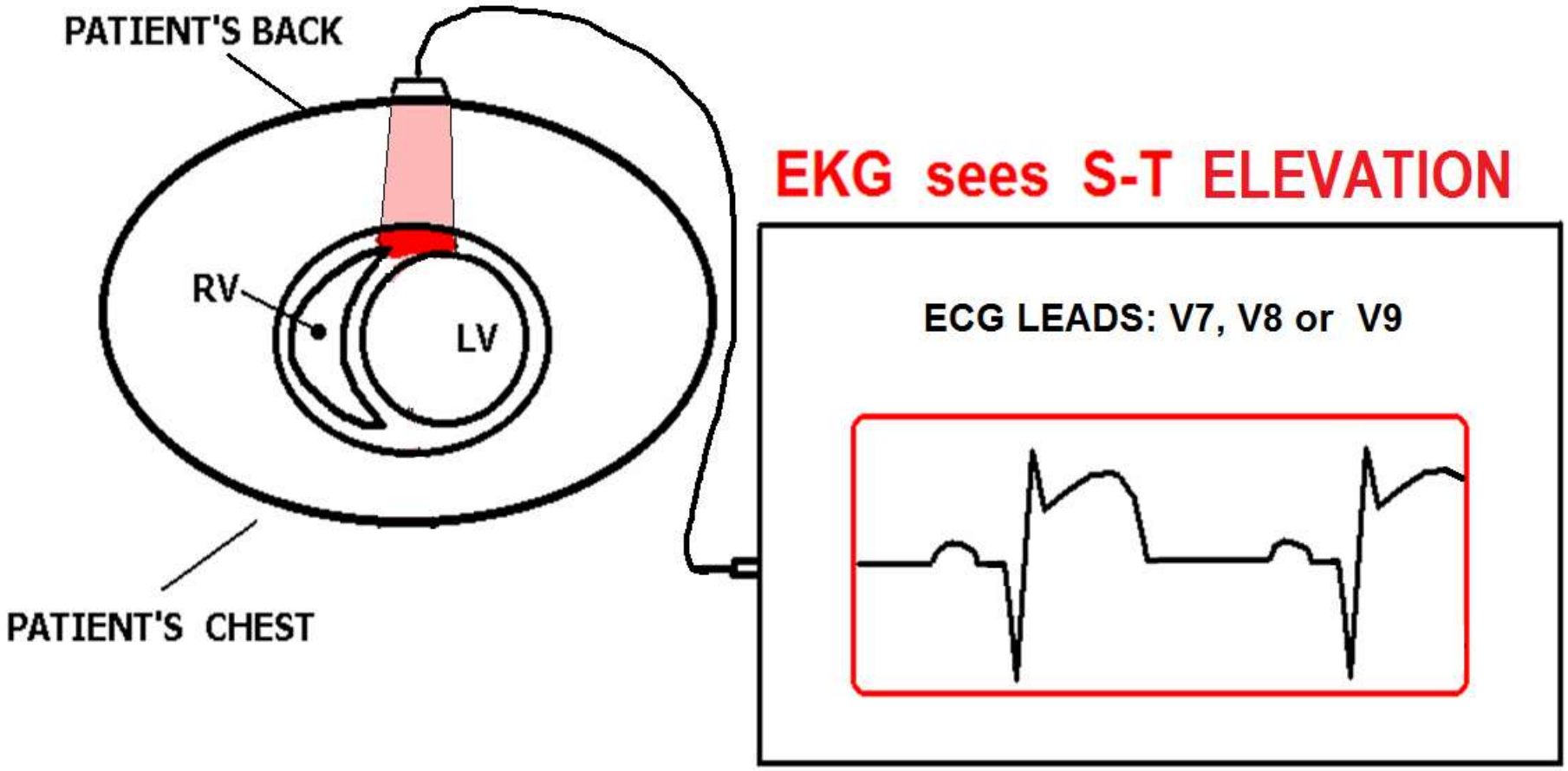
HOW EKG VIEWS RECIPROCAL CHANGES

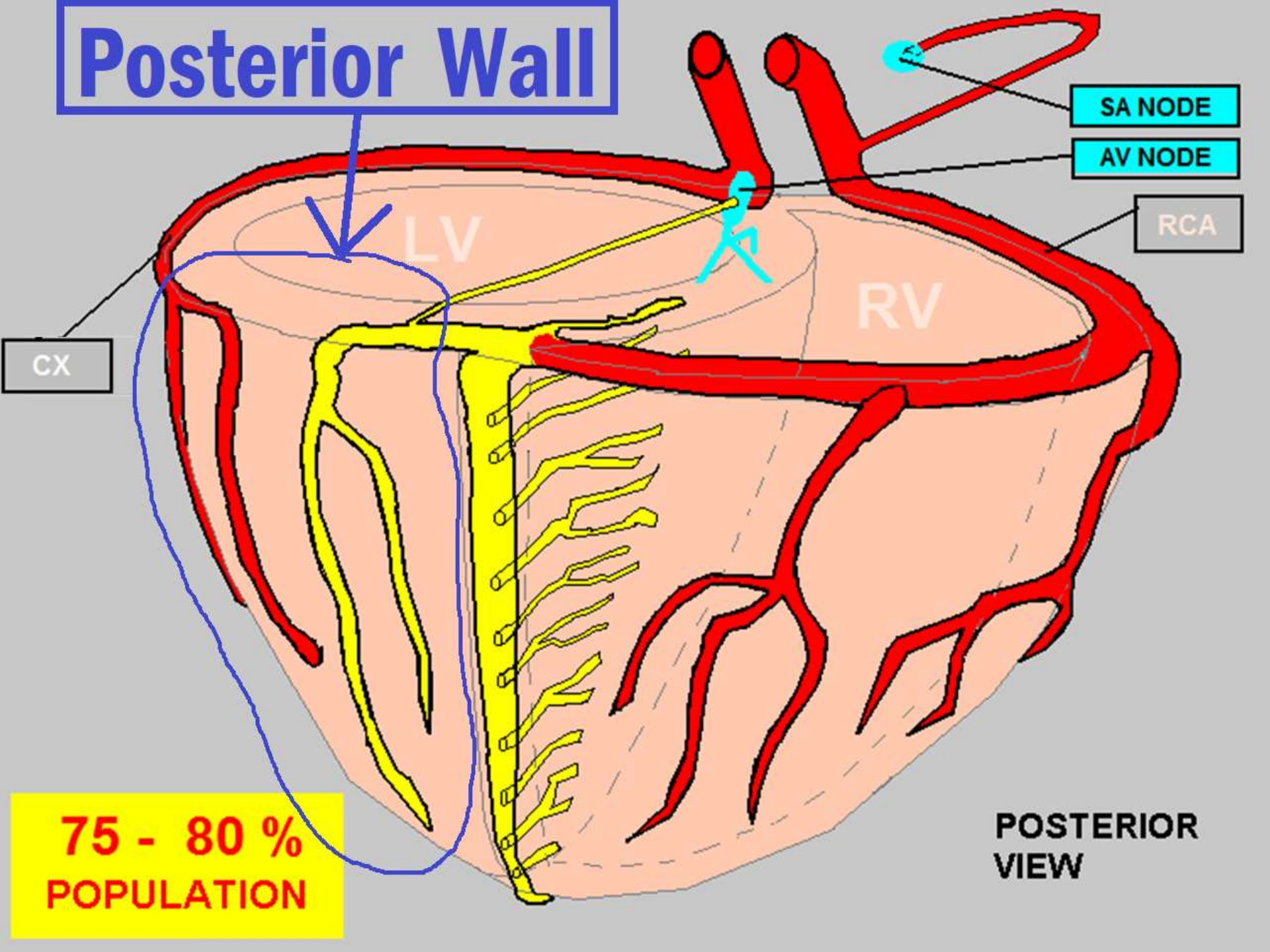
EXAMPLE:



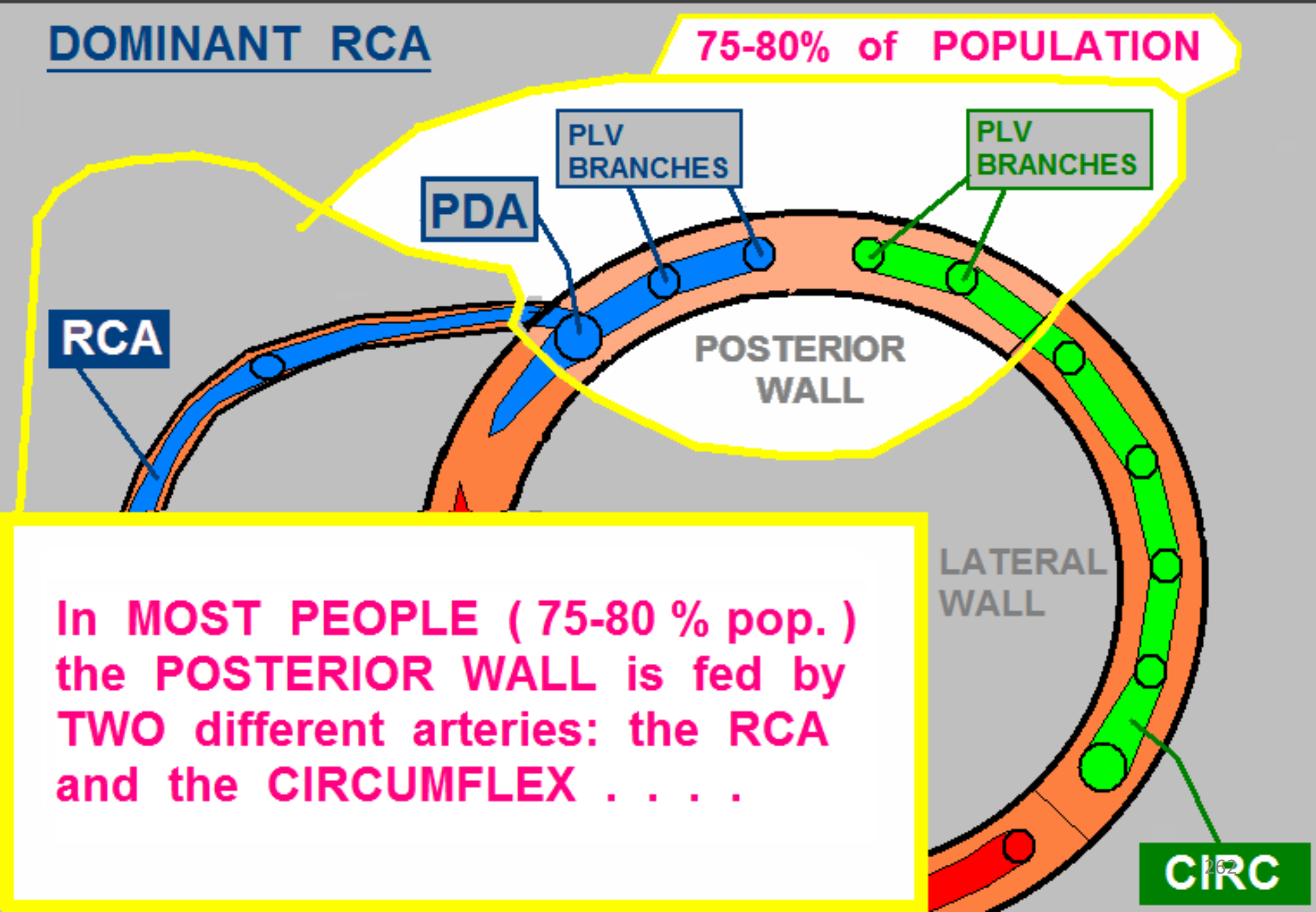
If we put ECG leads on the BACK of a PATIENT who is having an

ACUTE POSTERIOR WALL MI . . . . .



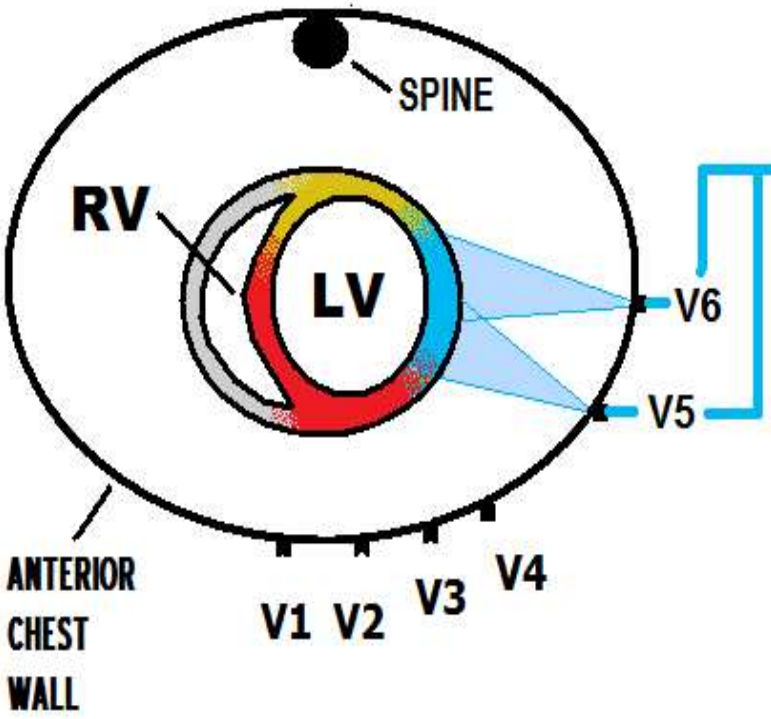

Posterior Wall
SA NODE
AV NODE
RCA
LV
RV
CX
75 - 80 %
POPULATION
POSTERIOR
VIEW

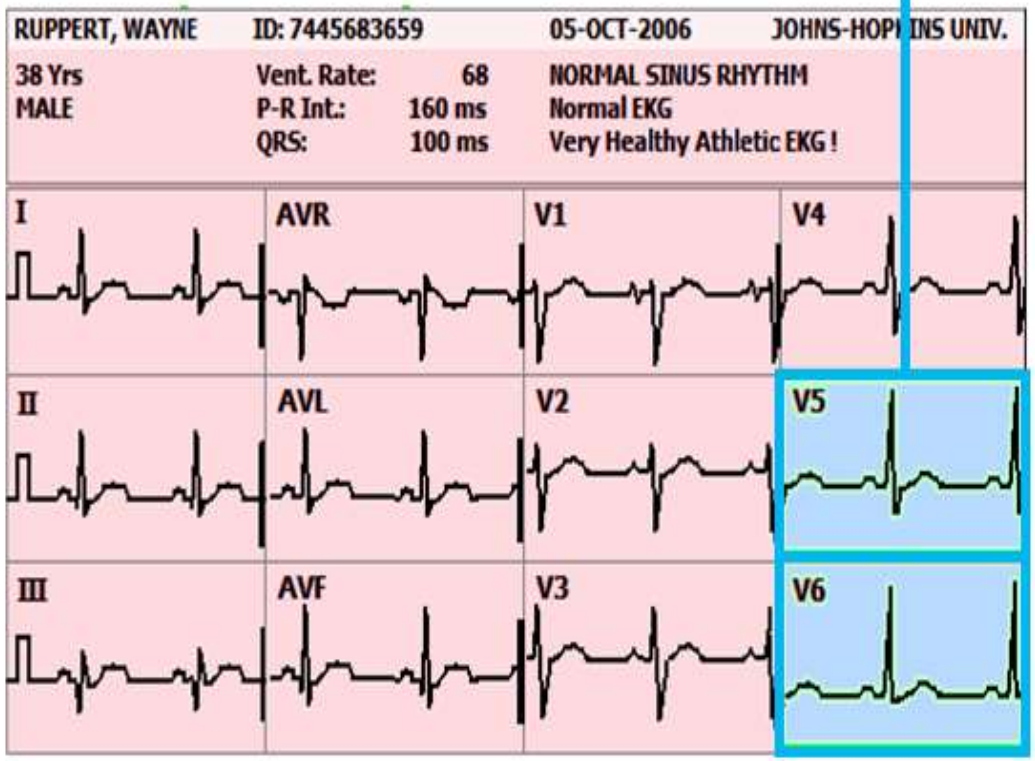
POSTERIOR WALL BLOOD SUPPLY



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

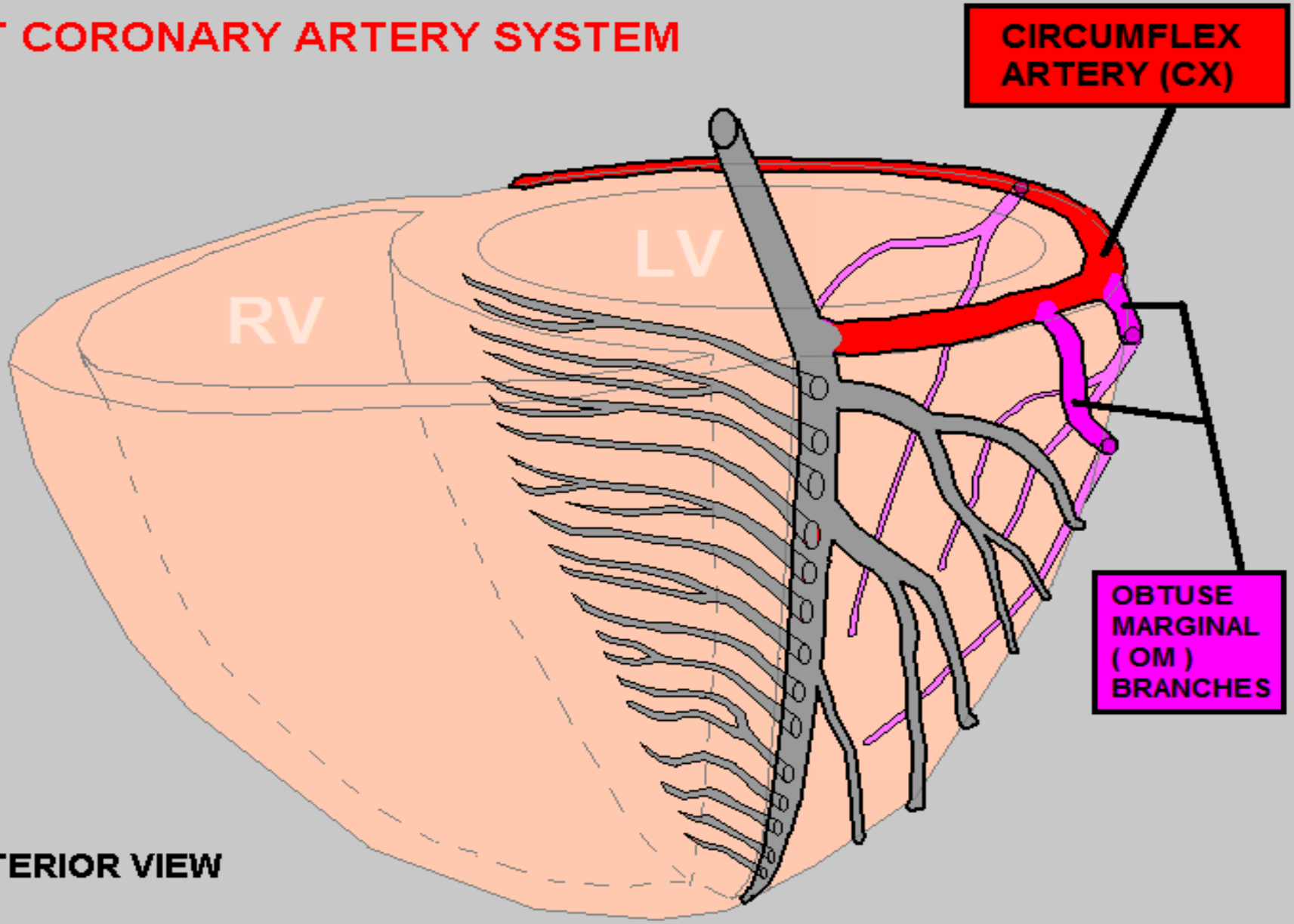
V5 - V6 VIEW THE LATERAL WALL
of the LEFT VENTRICLE





Which Coronary Artery typically Supplies the LATERAL WALL ?

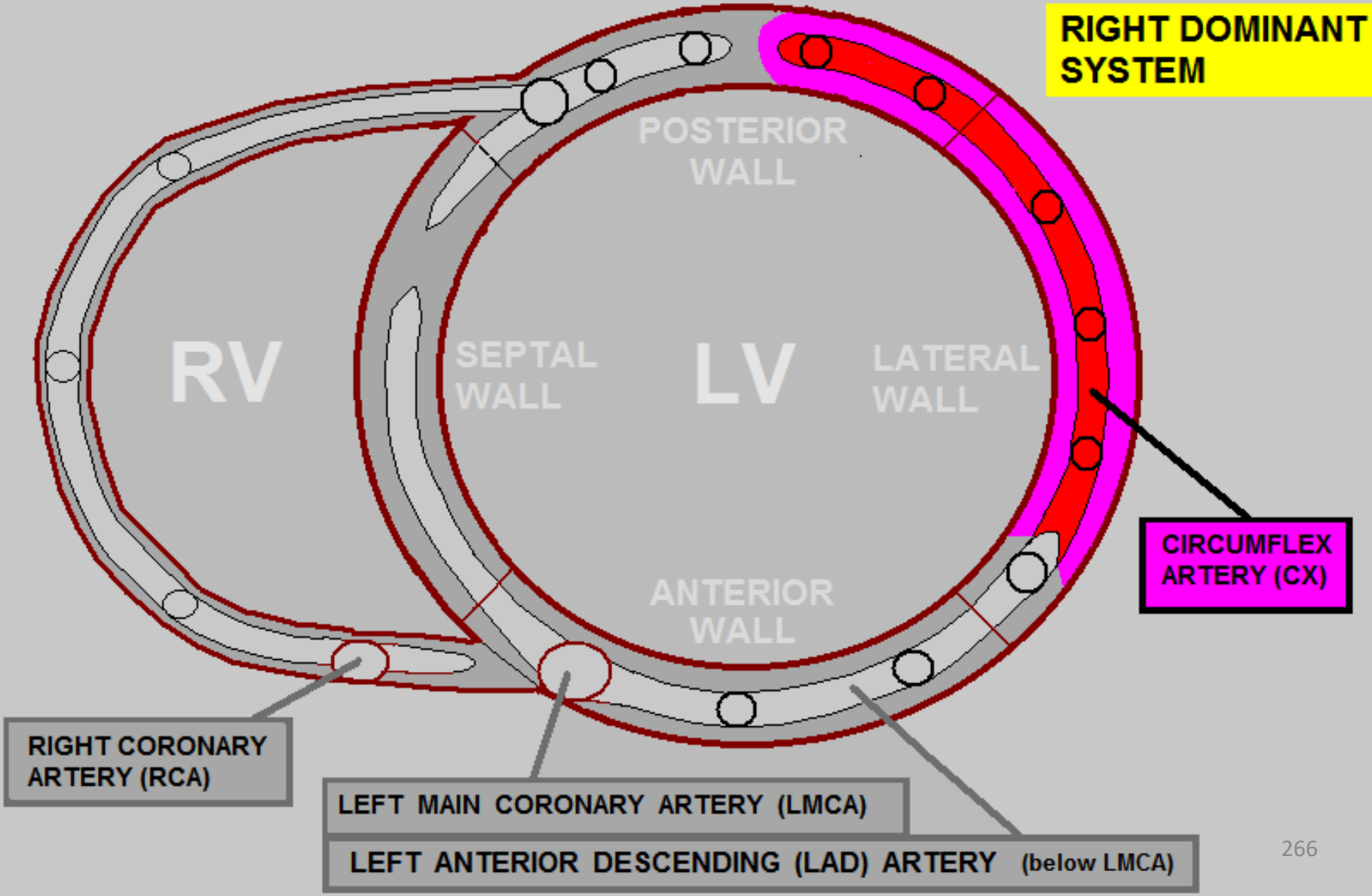
LEFT CORONARY ARTERY SYSTEM



cutaway view of the

CIRCUMFLEX ARTERY (CX) DISTRIBUTION

SUPPLIES 20 - 30 % of the LV MUSCLE MASS



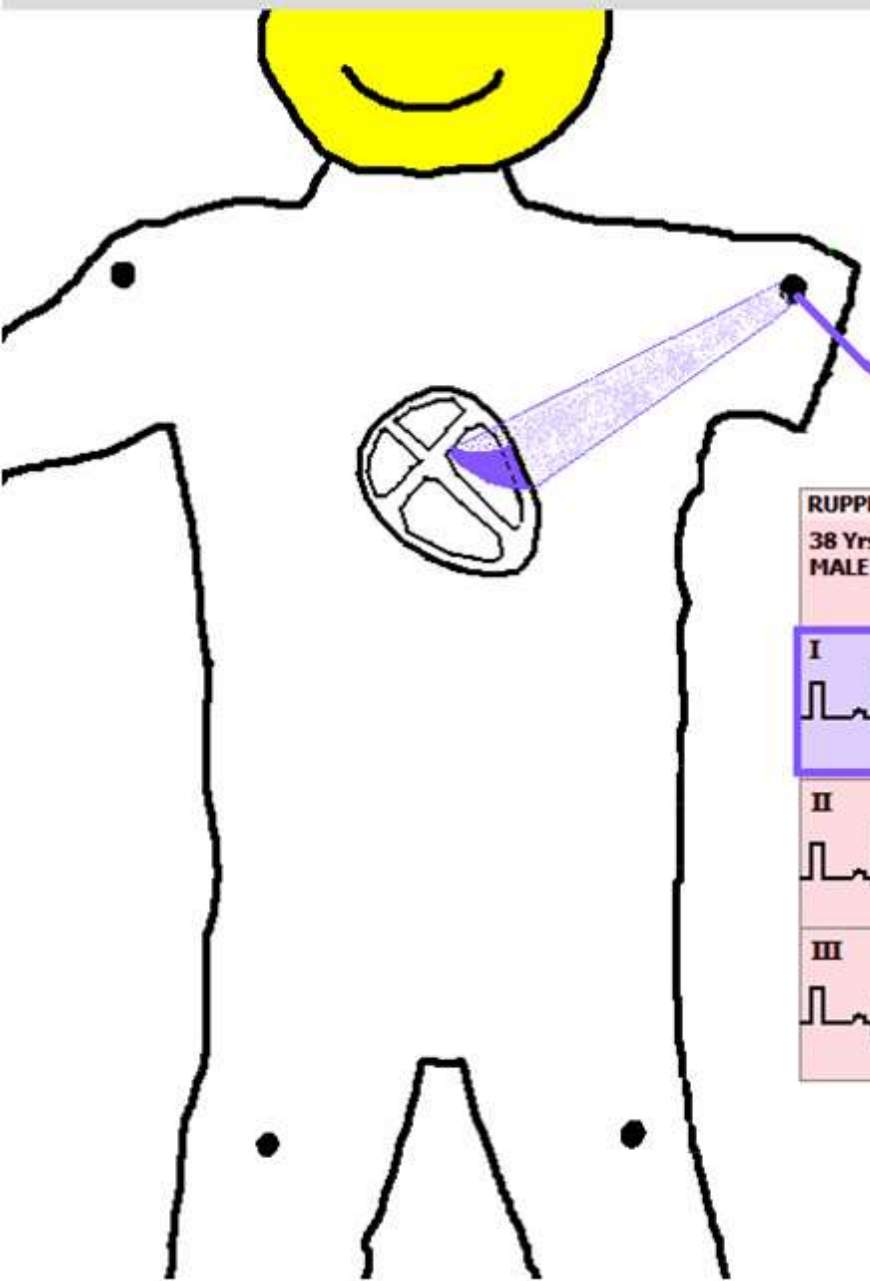
HELPFUL HINT . . . MEMORIZE THIS !

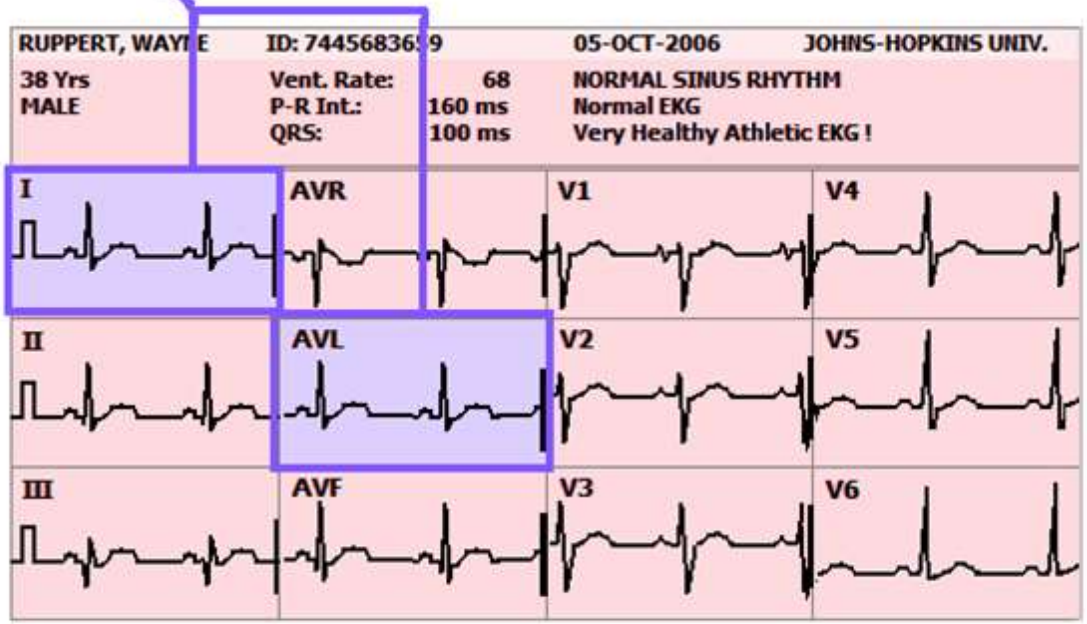
CIRCUMFLEX ARTERY (CX)

RIGHT DOMINANT SYSTEMS

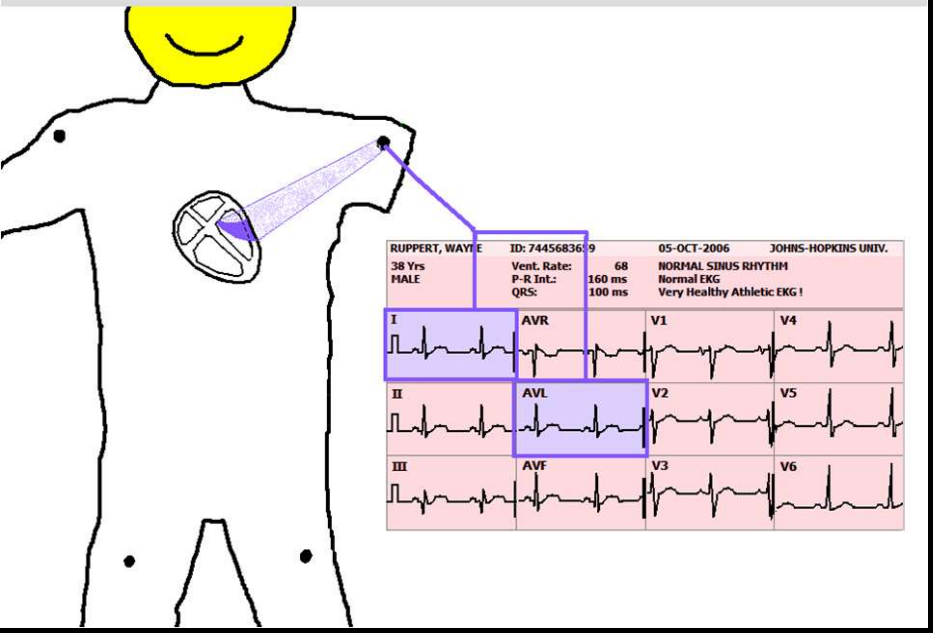
- LEFT ATRIUM
- SINUS NODE (5% of the population)
- LEFT VENTRICLE: 20 - 30 % of muscle mass
  - LATERAL WALL
  - up to 1/2 of POSTERIOR WALL

LEADS I and aVL VIEW the LATERAL - ANTERIOR WALL

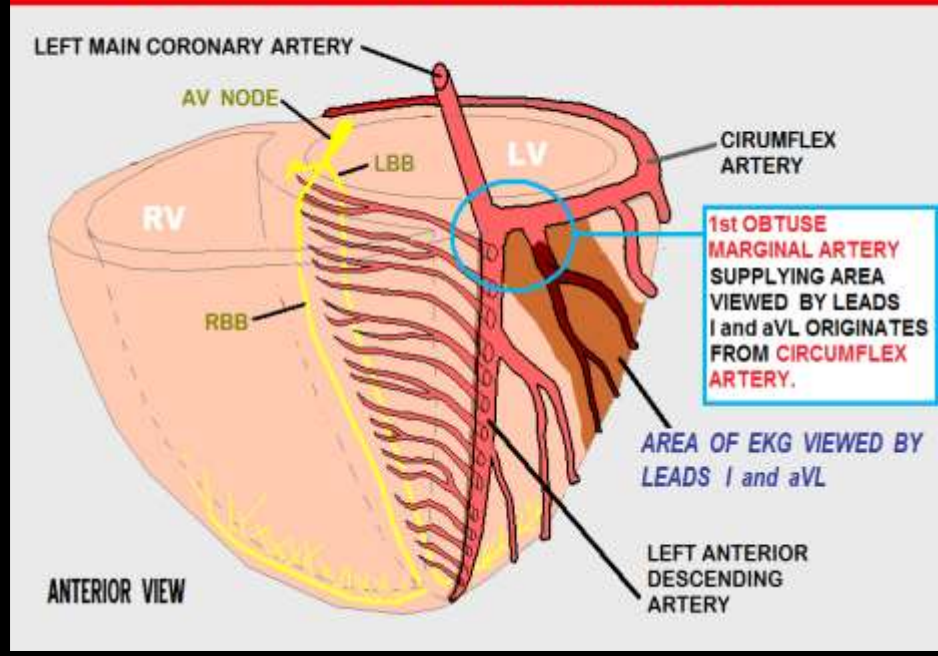




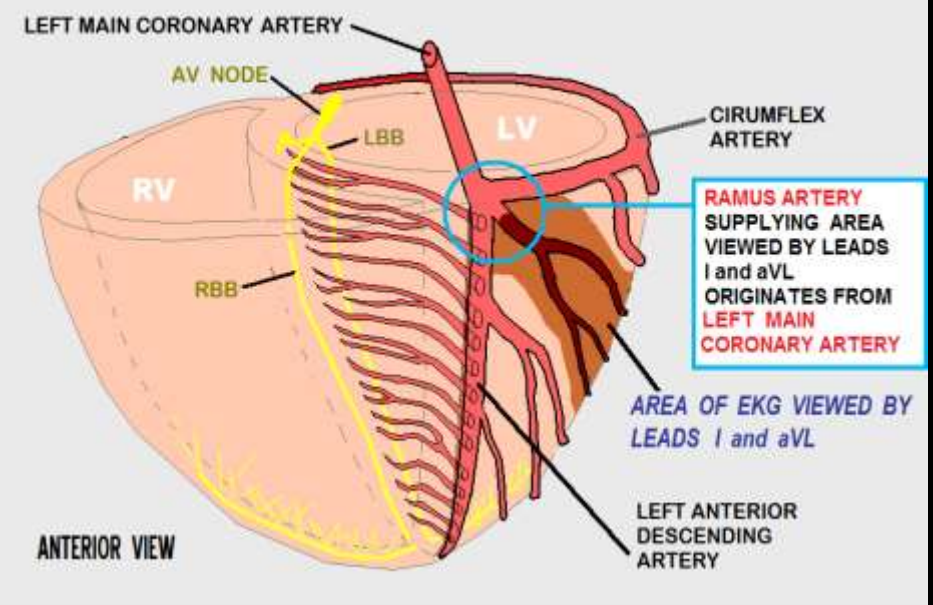
LEADS I and aVL VIEW the LATERAL - ANTERIOR WALL



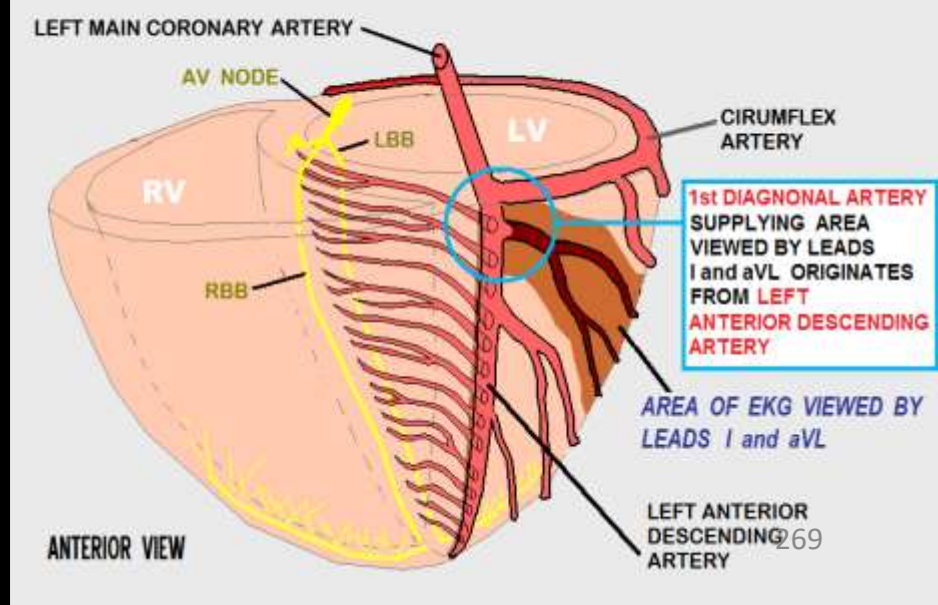
OCCLUSION of OBTUSE MARGINAL ARTERY

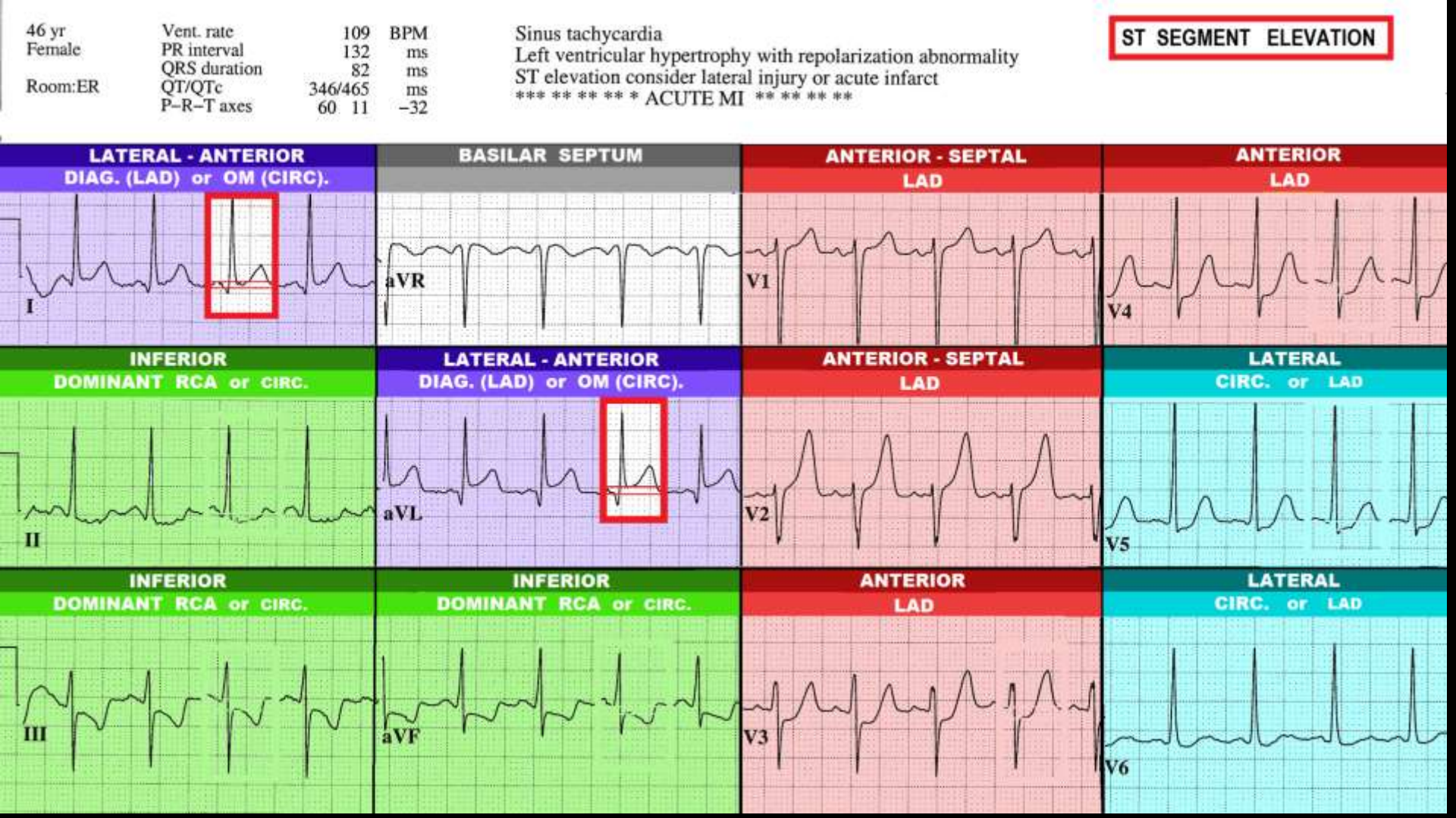


OCCLUSION of RAMUS ARTERY



OCCLUSION of DIAGONAL ARTERY

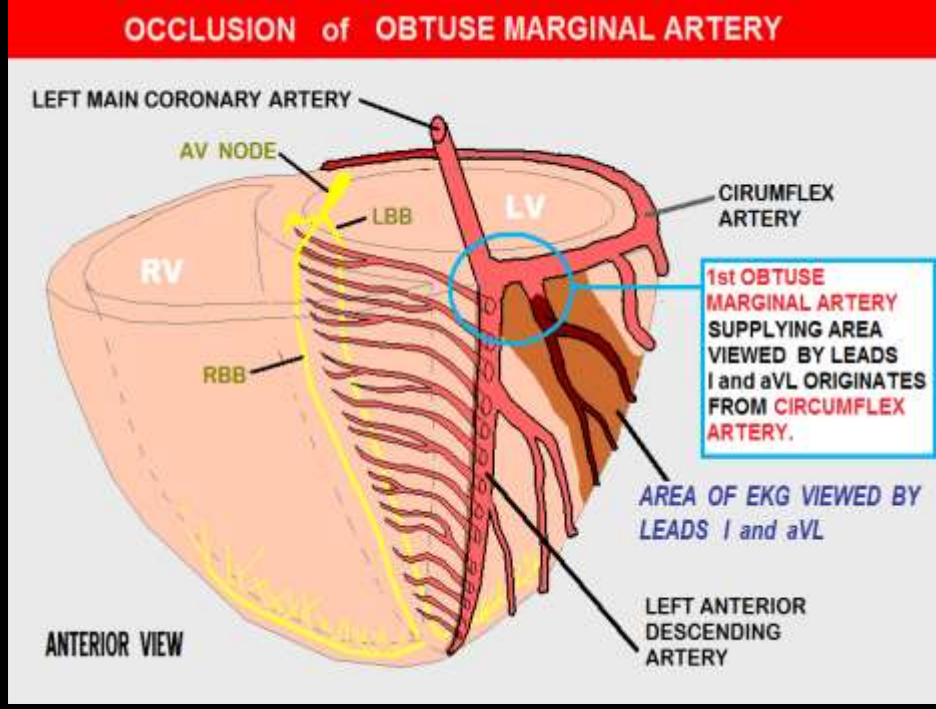


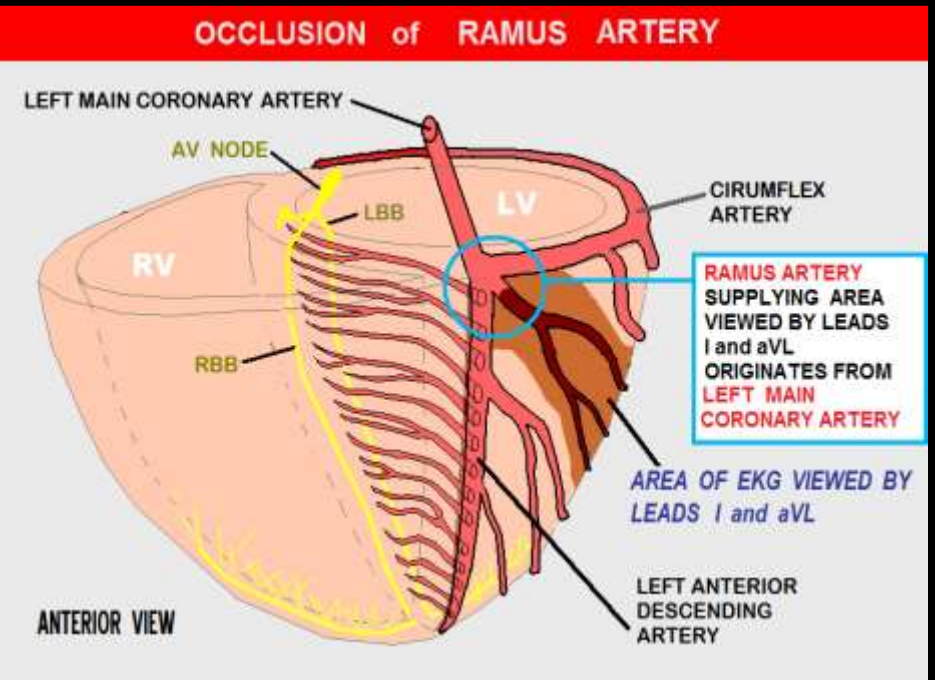


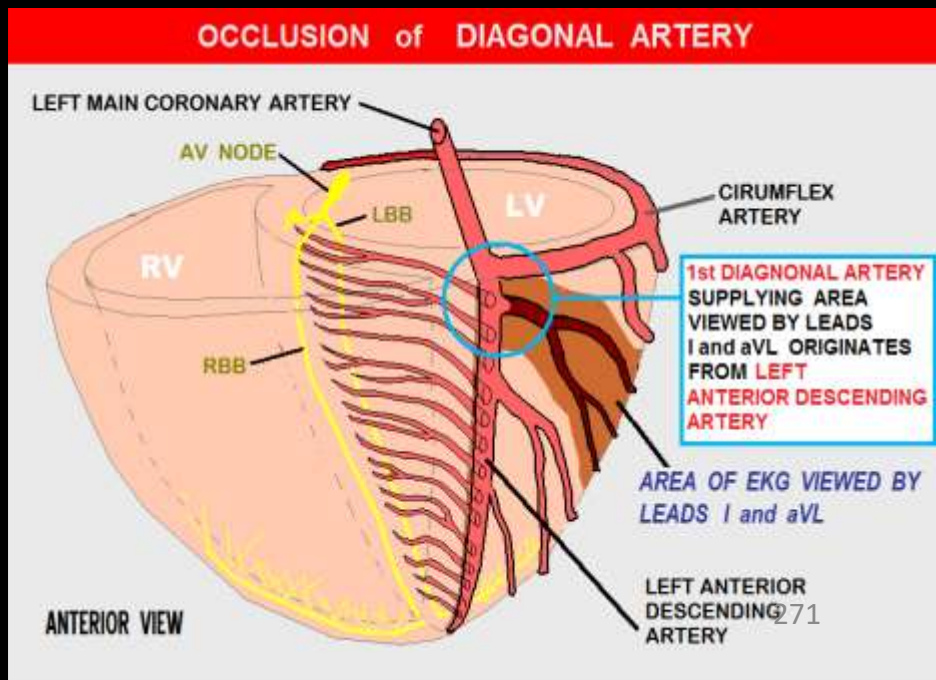
ST Segment elevation ONLY in Leads I and aVL

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

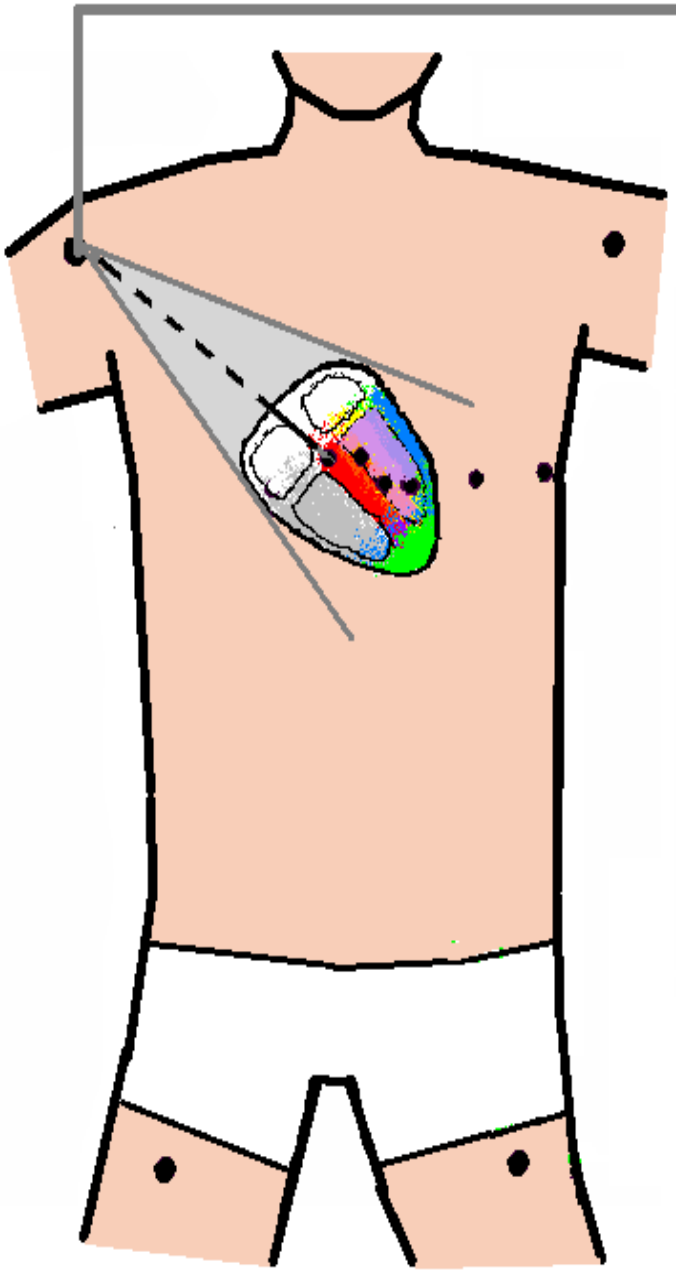
- **RAMUS BRANCH**
- **1st DIAGONAL off of LAD**
- **1st OBTUSE MARGINAL off of CIRCUMFLEX**

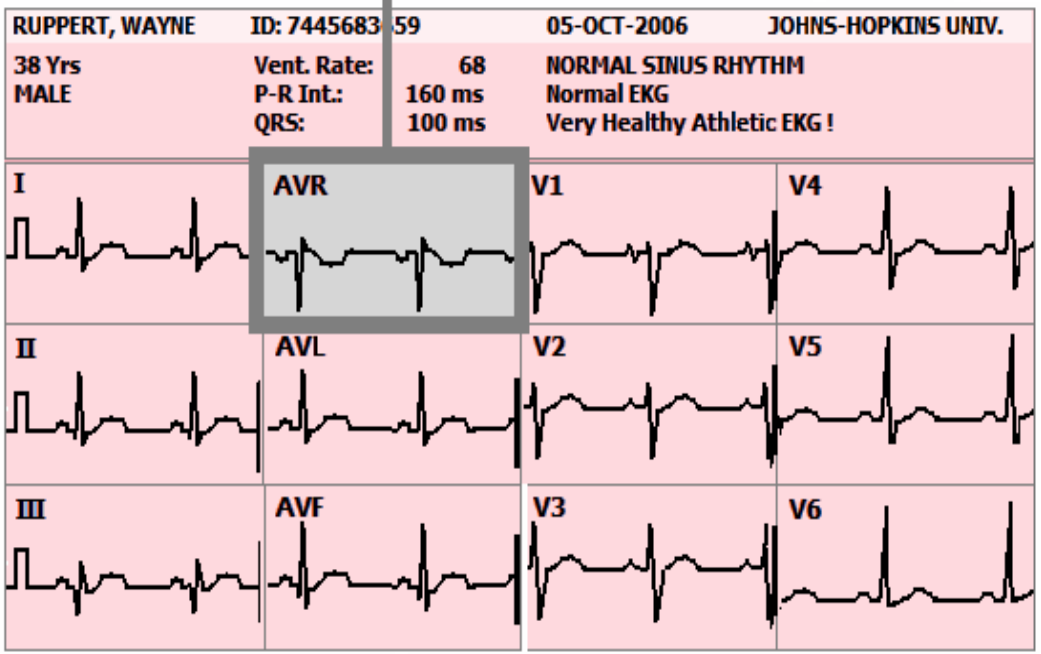






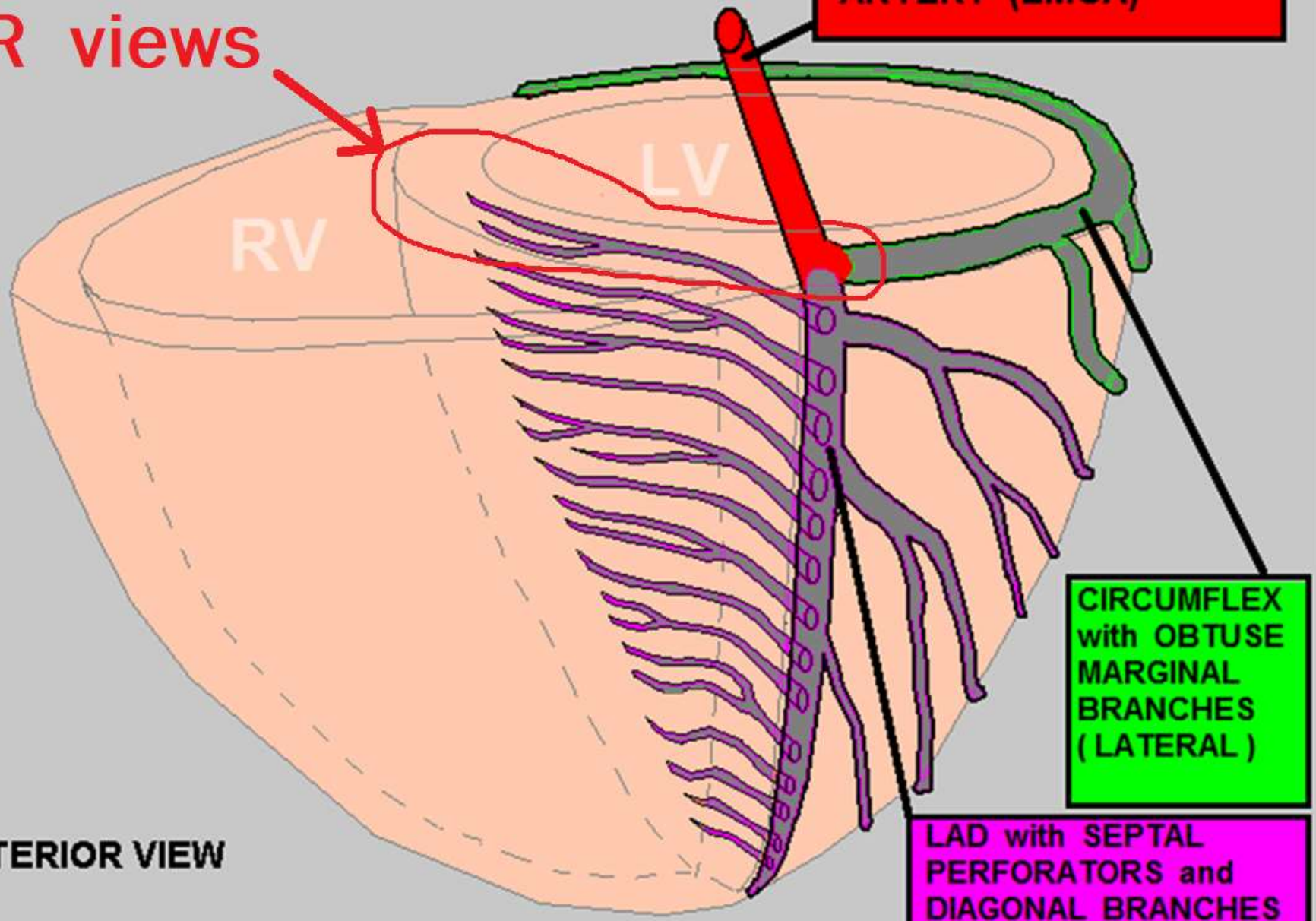
Lead AVR Views the BASILAR SEPTUM (region of the Bundle of His)





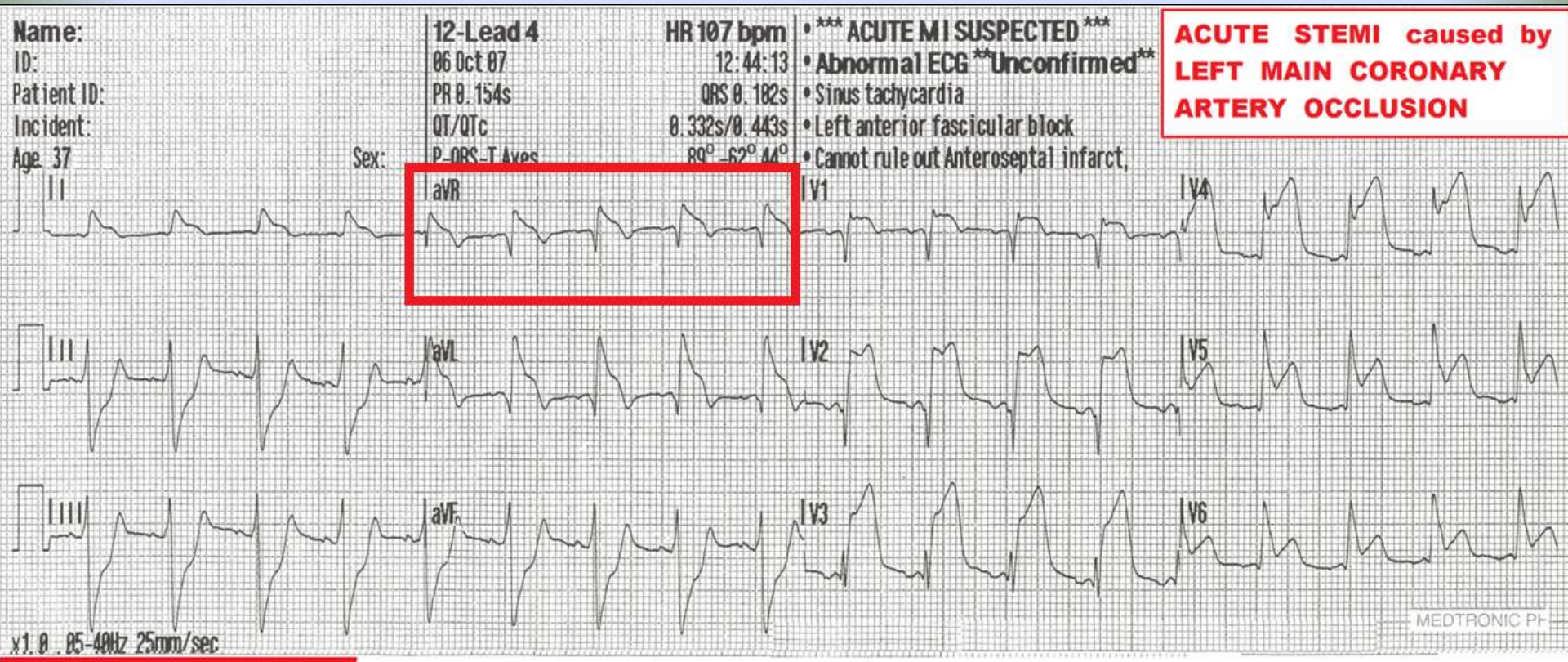
LEFT CORONARY ARTERY SYSTEM

AVR views



When LEAD AVR shows ST Elevation:

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

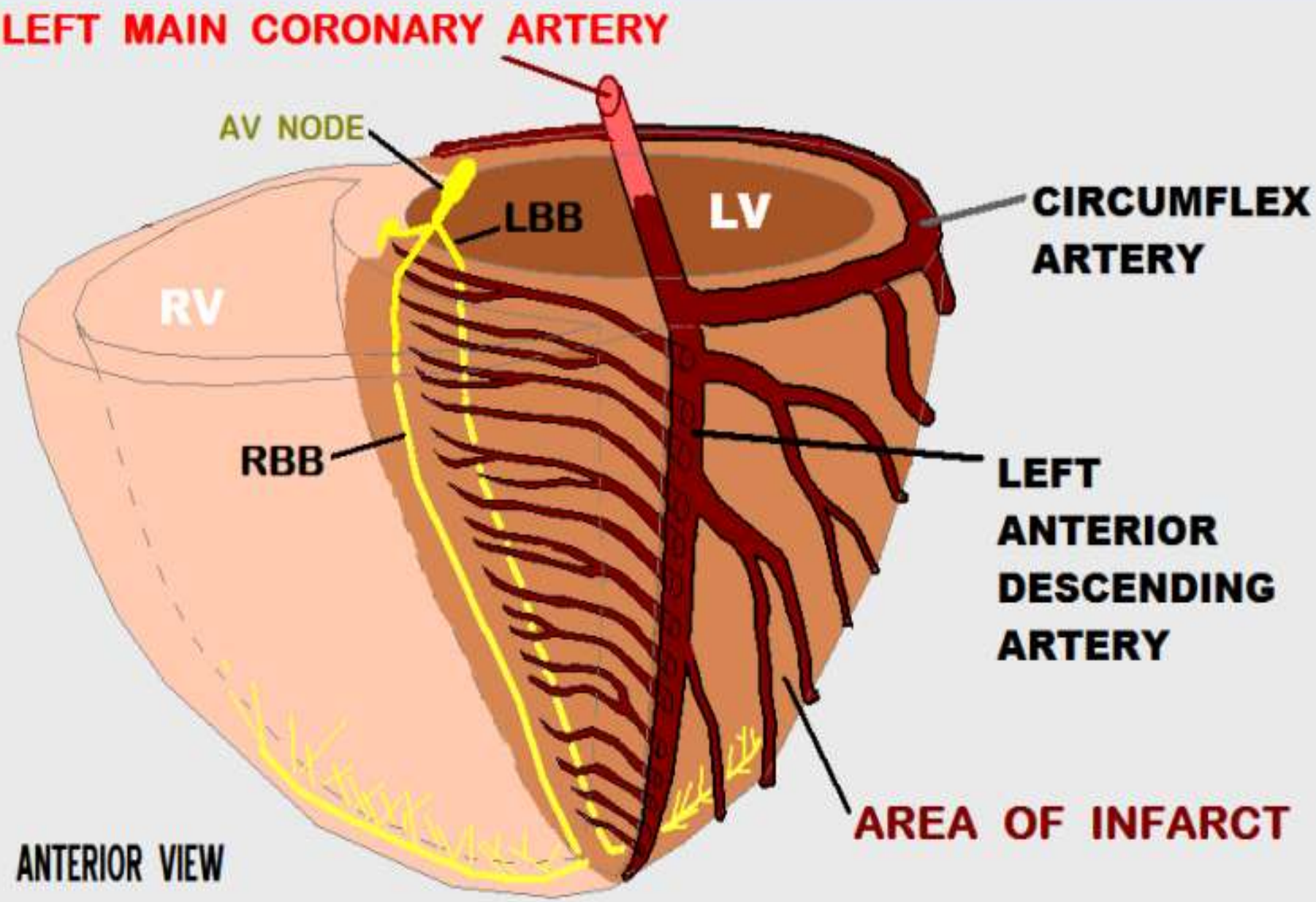


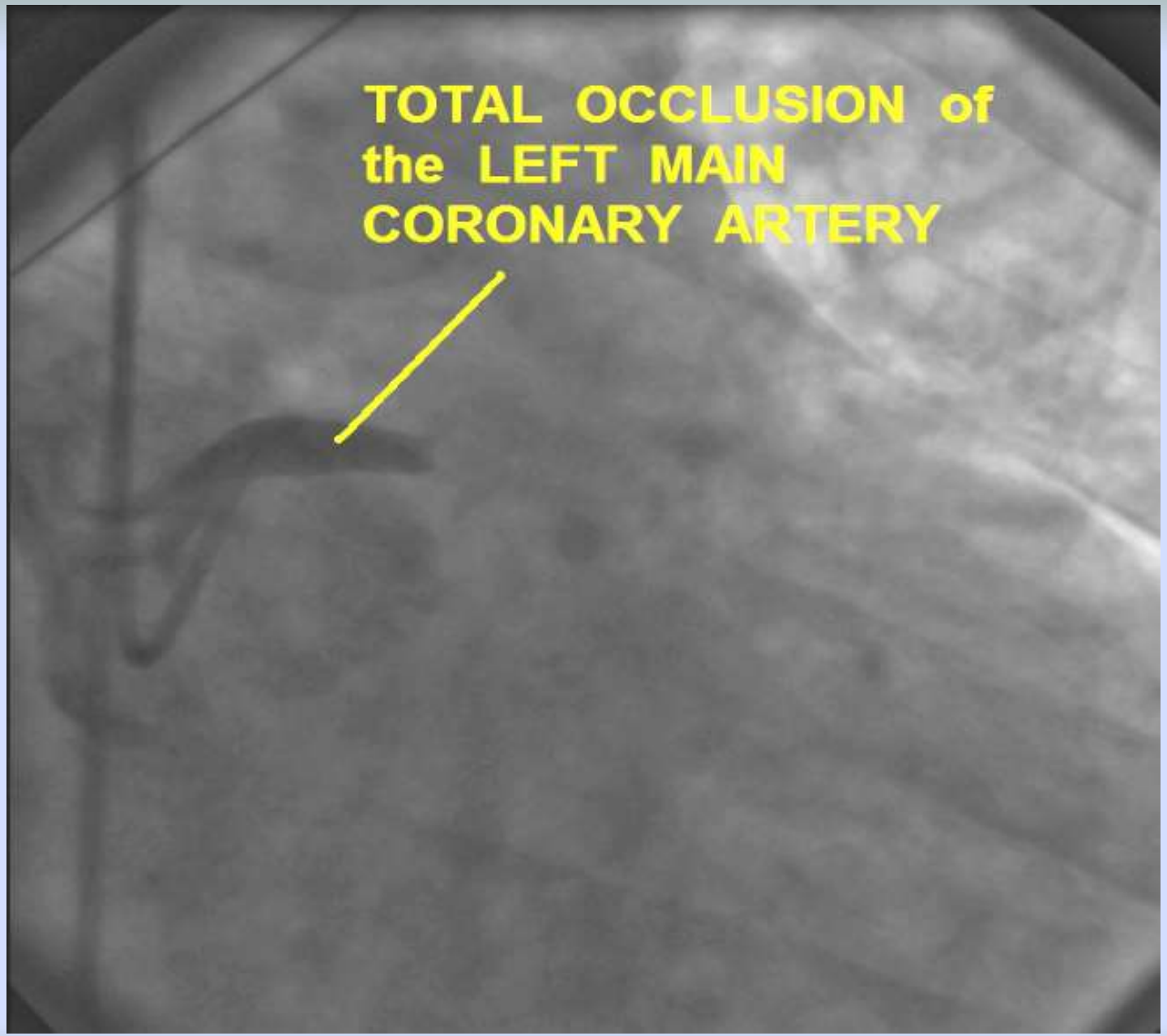
ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION

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

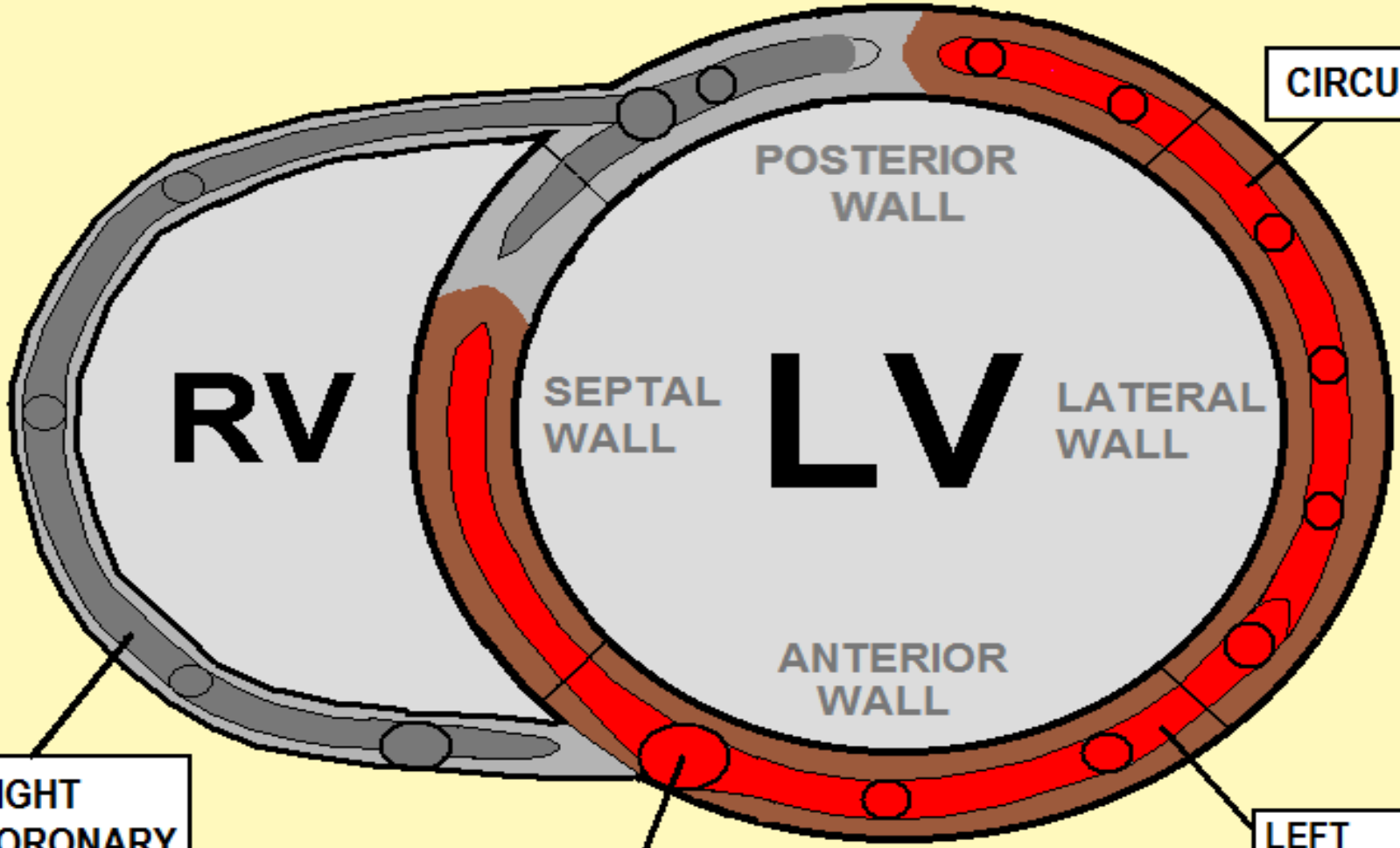
OCCLUSION of the LEFT MAIN CORONARY ARTERY




TOTAL OCCLUSION of
the LEFT MAIN
CORONARY ARTERY

The LEFT MAIN CORONARY ARTERY

SUPPLIES 75 - 100 % of the LEFT VENTRICULAR MUSCLE MASS



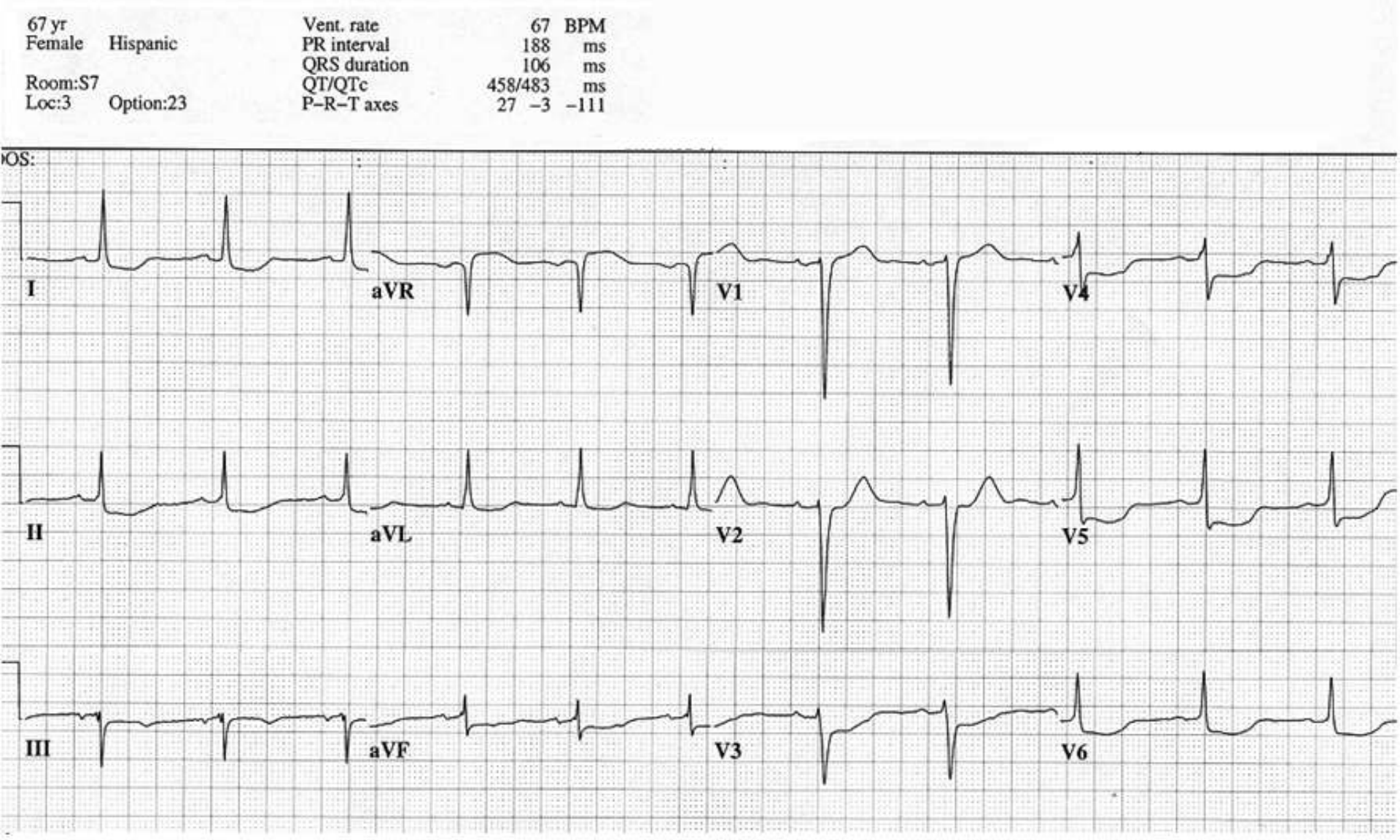
When LEAD AVR shows ST Elevation:

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

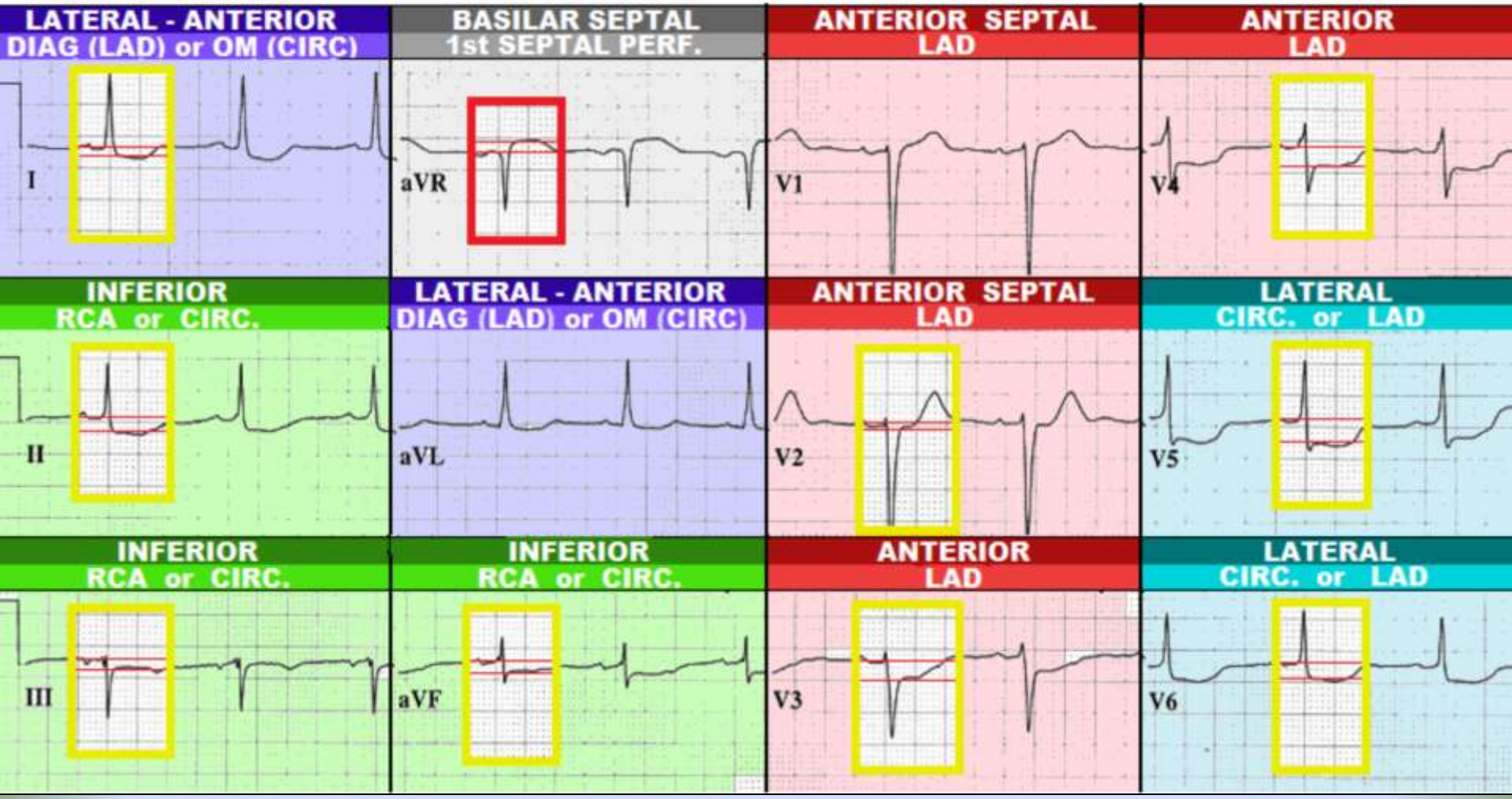
67 yr
Female Hispanic

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

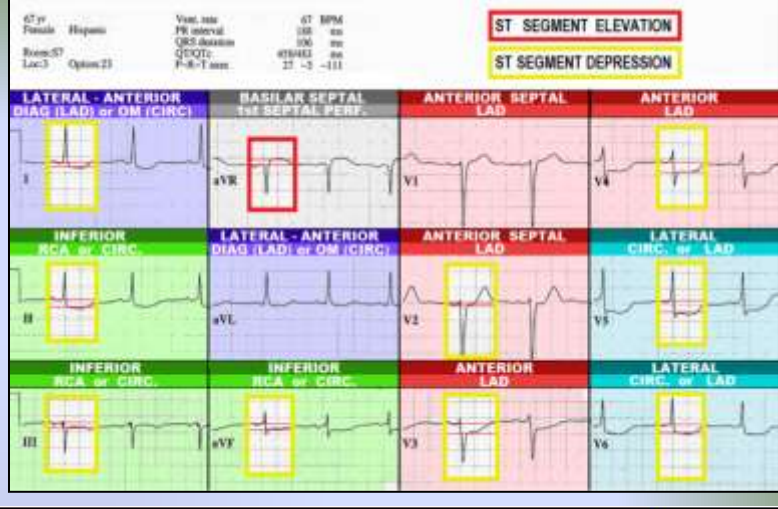
ST SEGMENT DEPRESSION

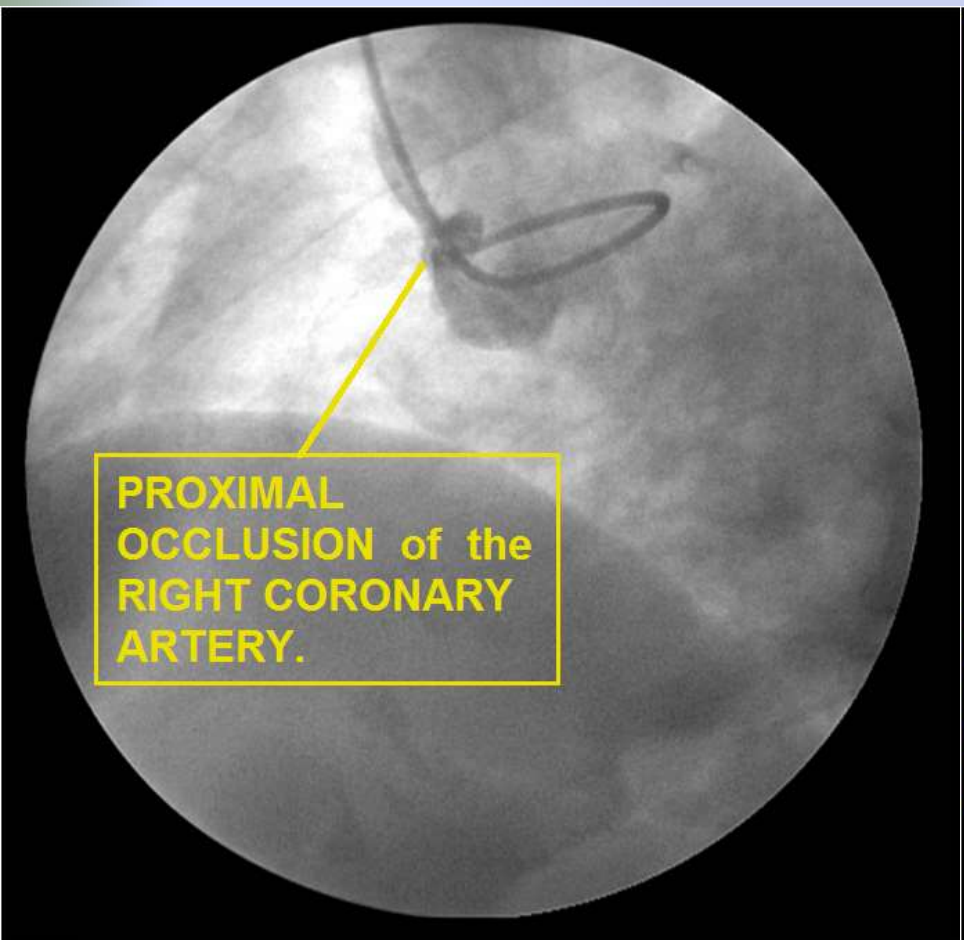


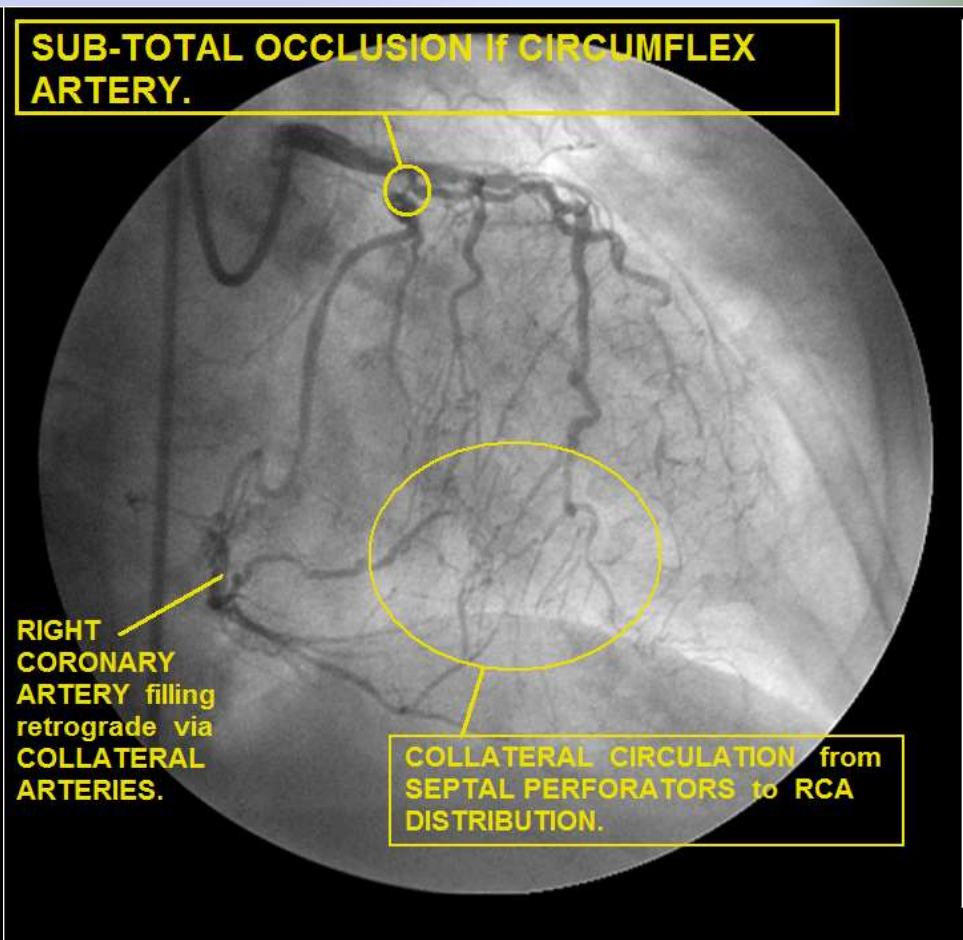
GLOBAL ISCHEMIA

- **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*



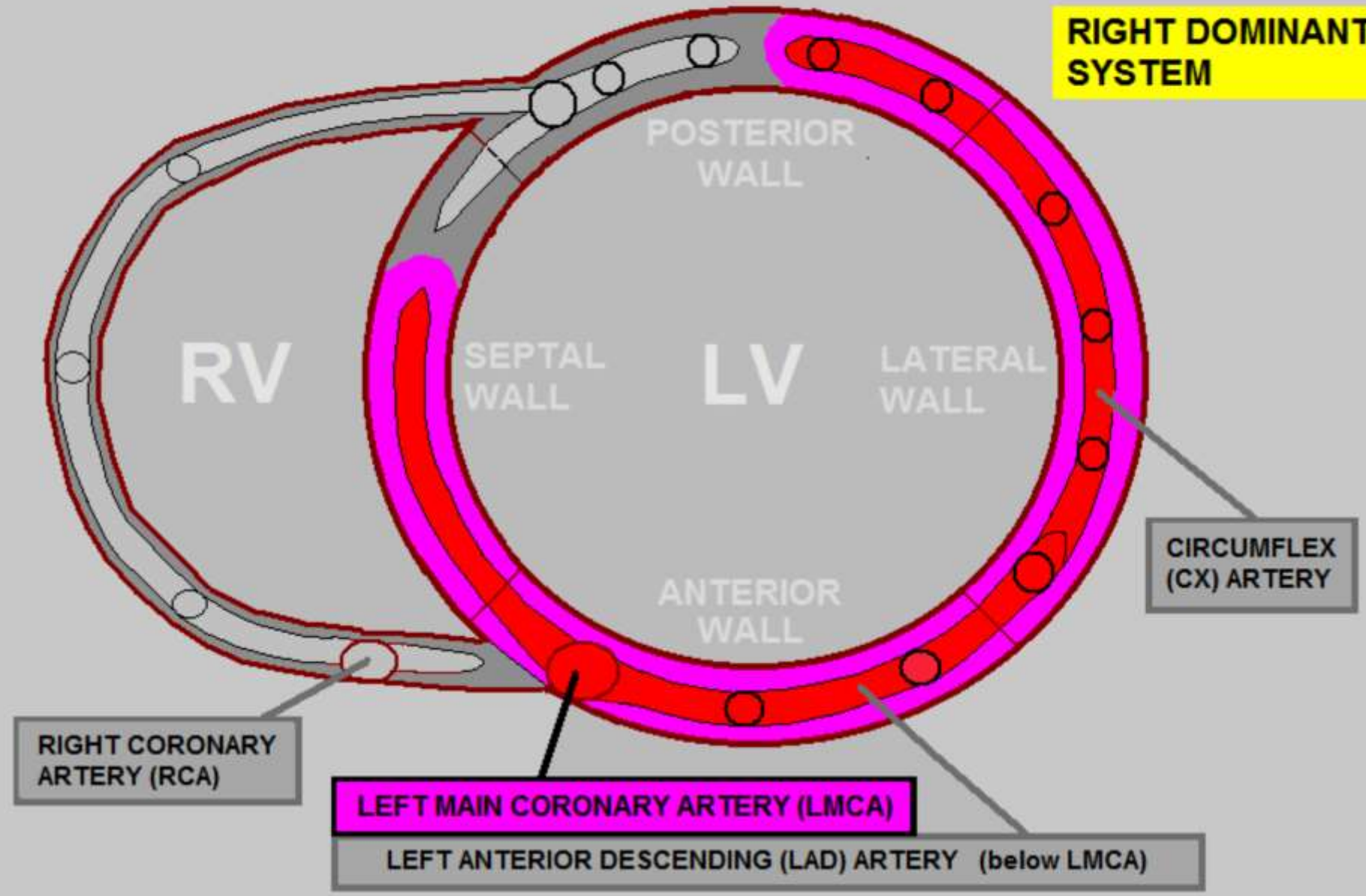




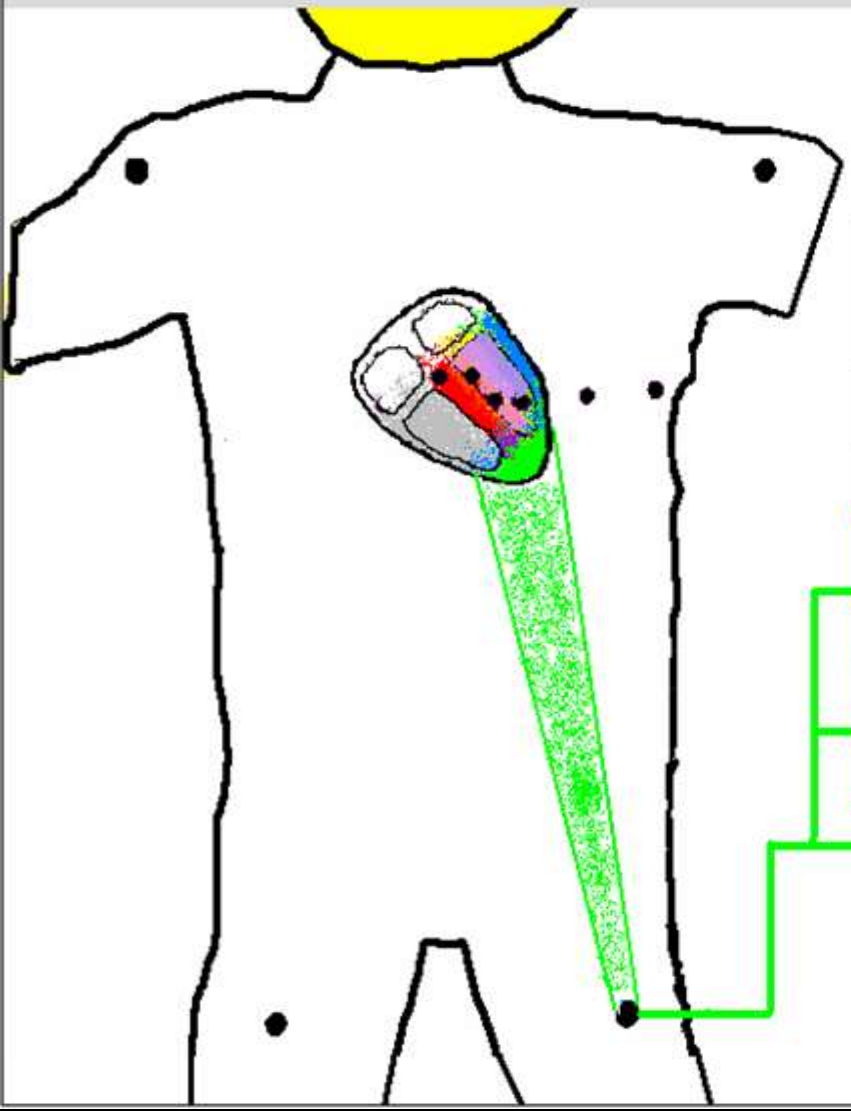
cutaway view of the

LEFT MAIN CORONARY ARTERY (LMCA)

SUPPLIES APPROXIMATELY 75% OF LV MUSCLE MASS

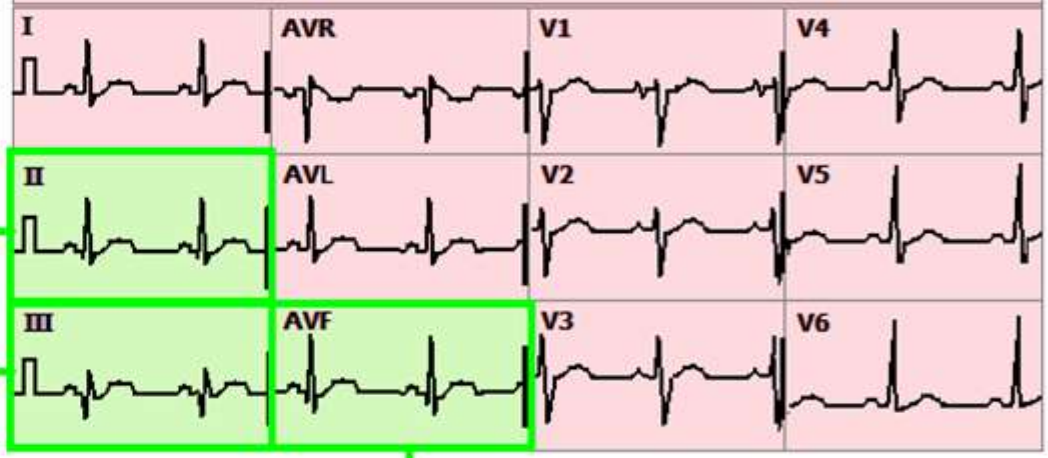


LEADS II, III, and aVF VIEW
INFERIOR WALL of the LEFT VENTRICLE

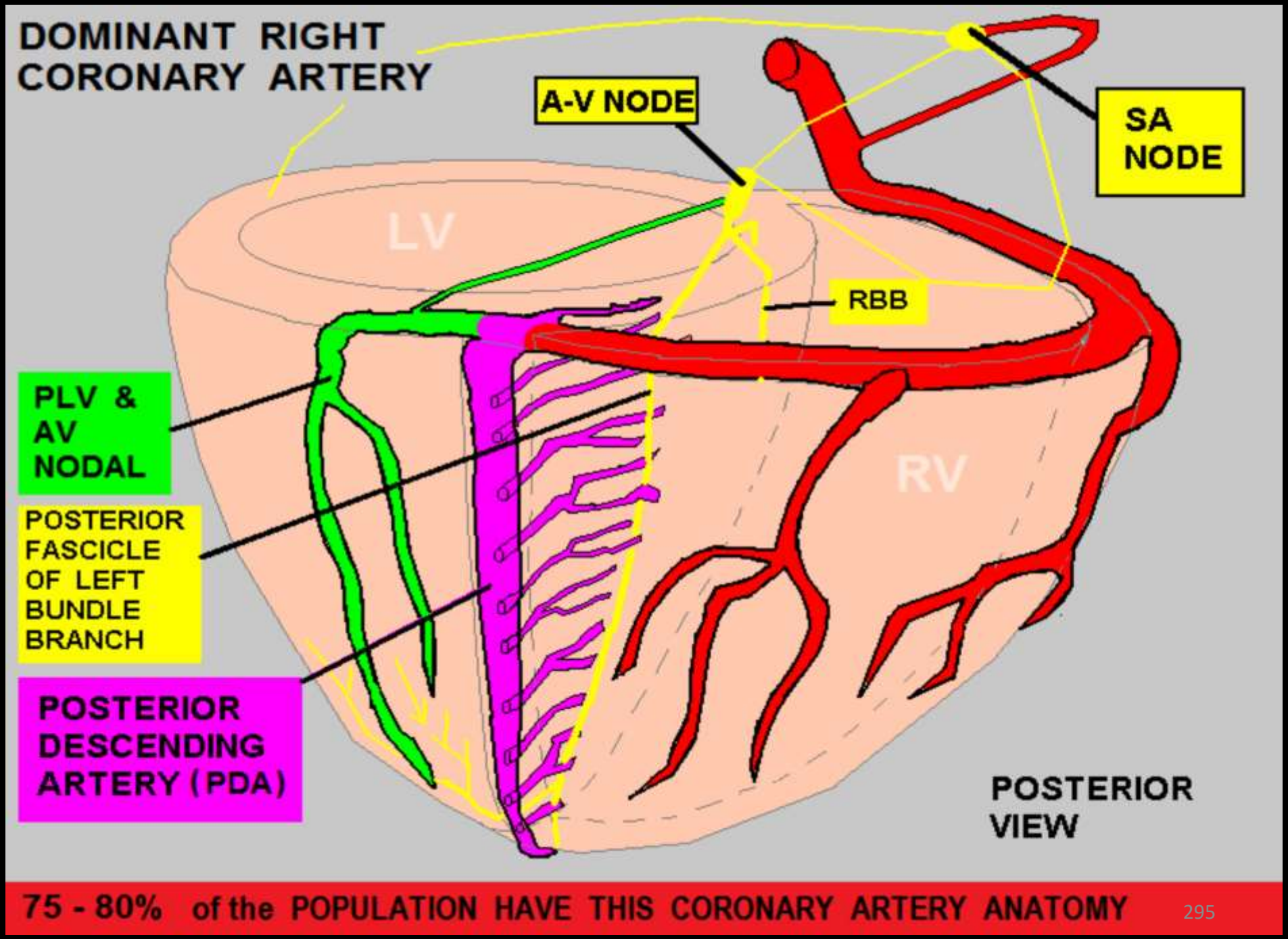


RUPPERT, WAYNE ID: 7445683659 05-OCT-2006 JOHNS-HOPKINS UNIV.

38 Yrs MALE
Vent. Rate: 68
P-R Int.: 160 ms
QRS: 100 ms
NORMAL SINUS RHYTHM
Normal EKG
Very Healthy Athletic EKG !



Which CORONARY ARTERY usually supplies the INFERIOR WALL?

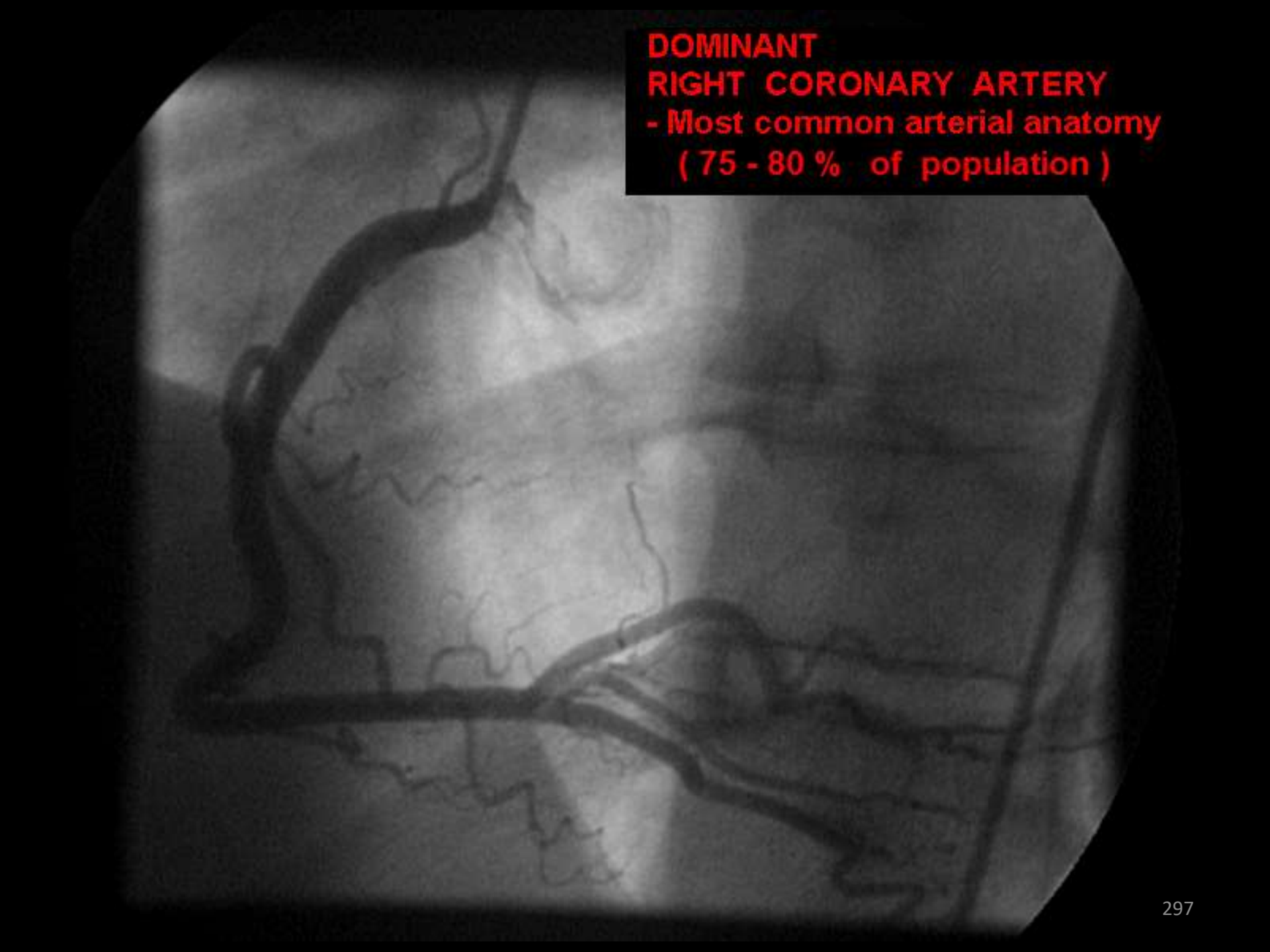

DOMINANT RIGHT CORONARY ARTERY
A-V NODE
SA NODE
LV
RBB
RV
PLV & AV NODAL
POSTERIOR FASCICLE OF LEFT BUNDLE BRANCH
POSTERIOR DESCENDING ARTERY (PDA)
POSTERIOR VIEW
75 - 80% of the POPULATION HAVE THIS CORONARY ARTERY ANATOMY

HELPFUL HINT . . . MEMORIZE THIS !

RIGHT CORONARY ARTERY (RCA)

RIGHT DOMINANT SYSTEMS

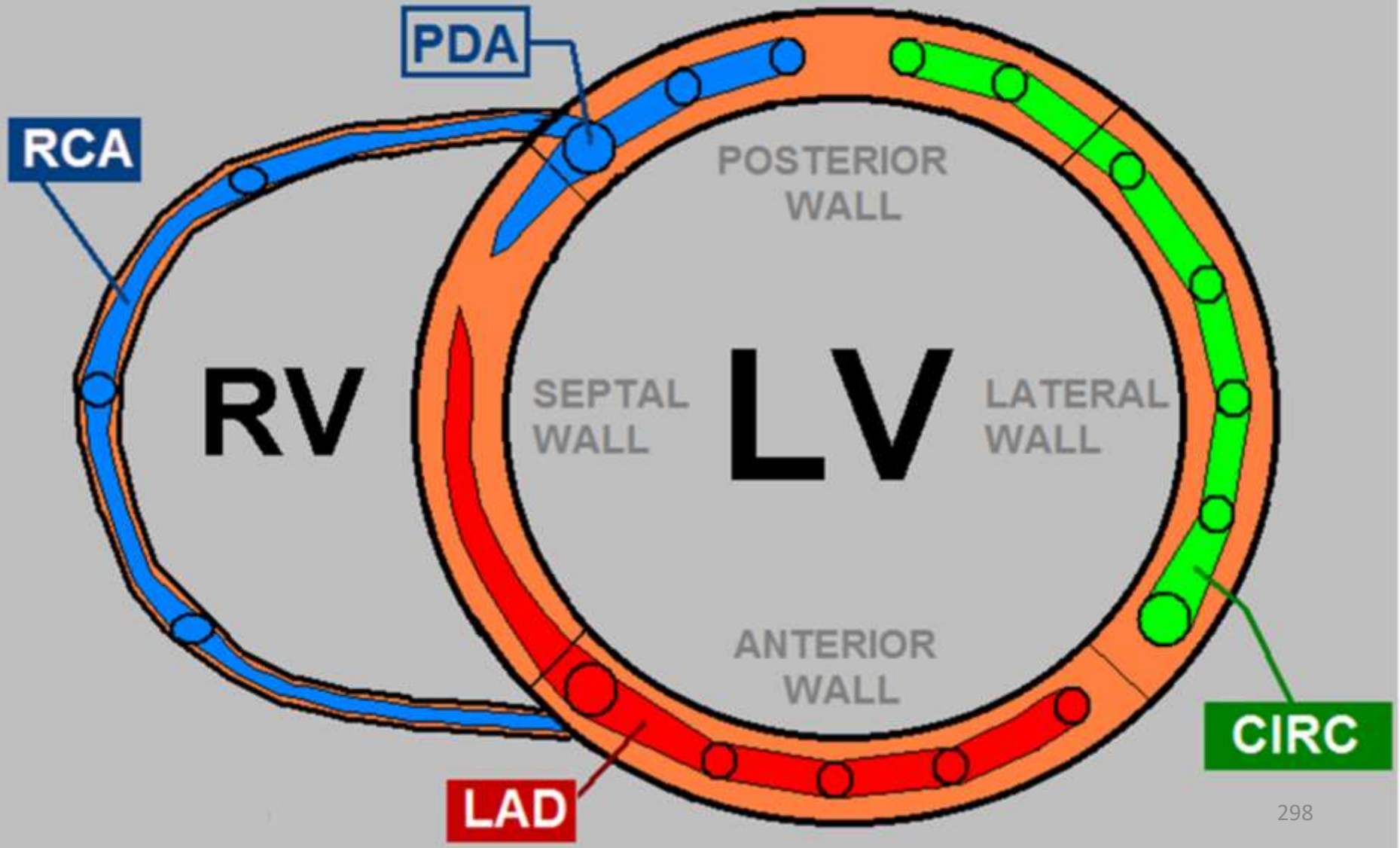
- RIGHT ATRIUM
- SINUS NODE (55% of the population)
- RIGHT VENTRICLE - 100 % of muscle mass
- LEFT VENTRICLE: 15 - 25 % of muscle mass
  - INFERIOR WALL
  - approx. 1/2 of POSTERIOR WALL
- AV NODE


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

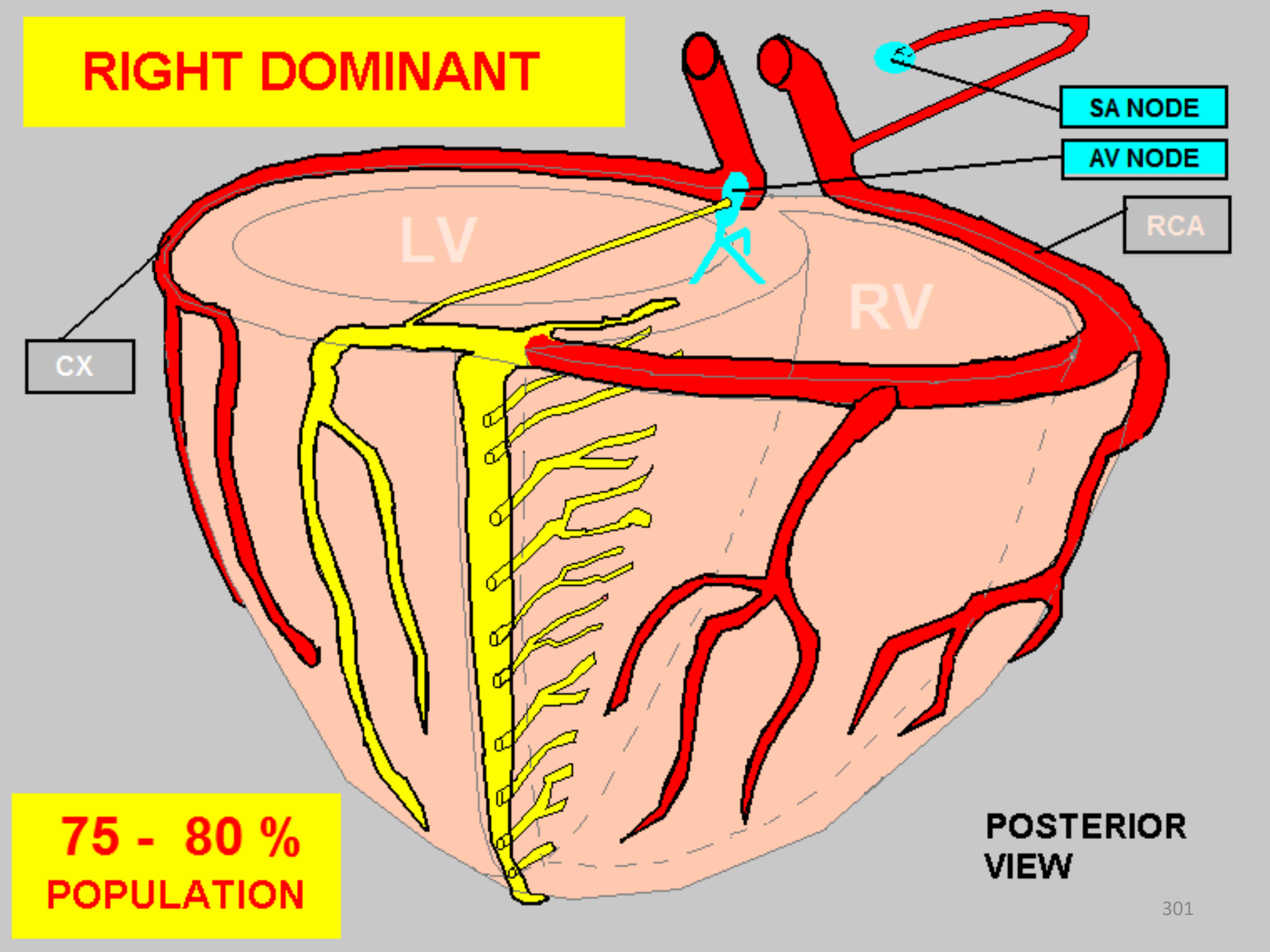
ARTERIAL DISTRIBUTION - MYOCARDIUM

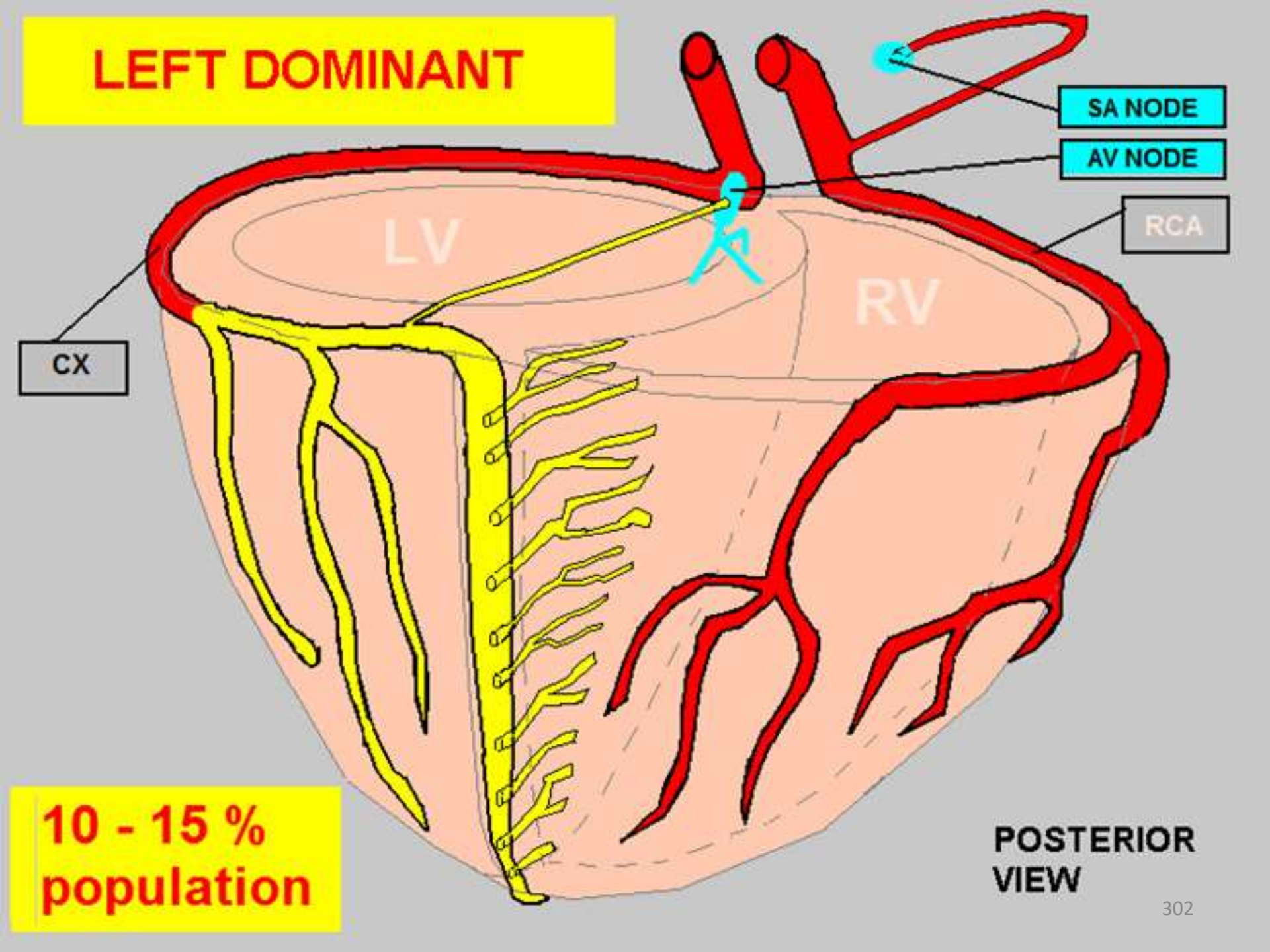
DOMINANT RCA

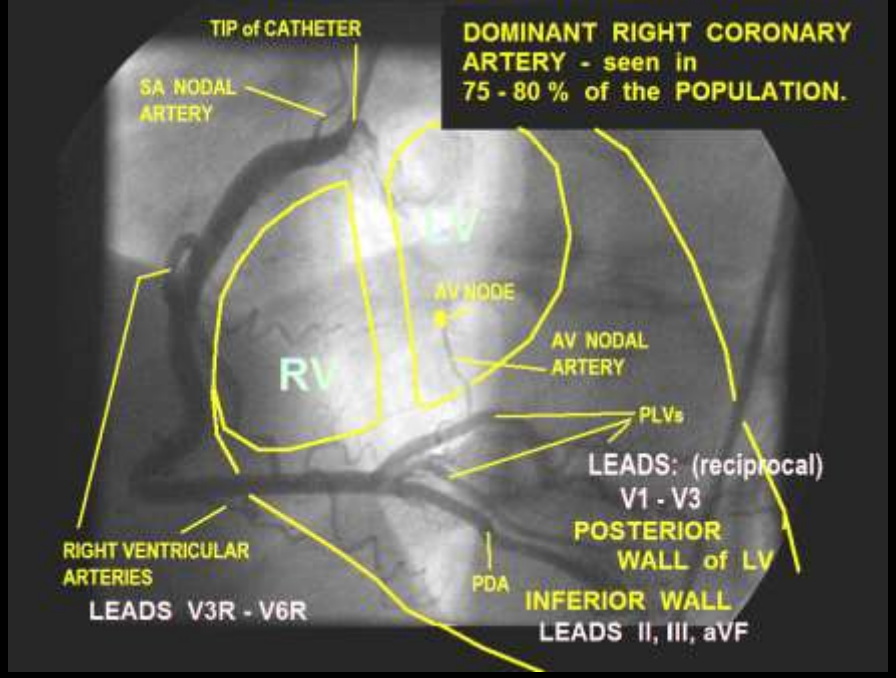
75-80 % of POPULATION

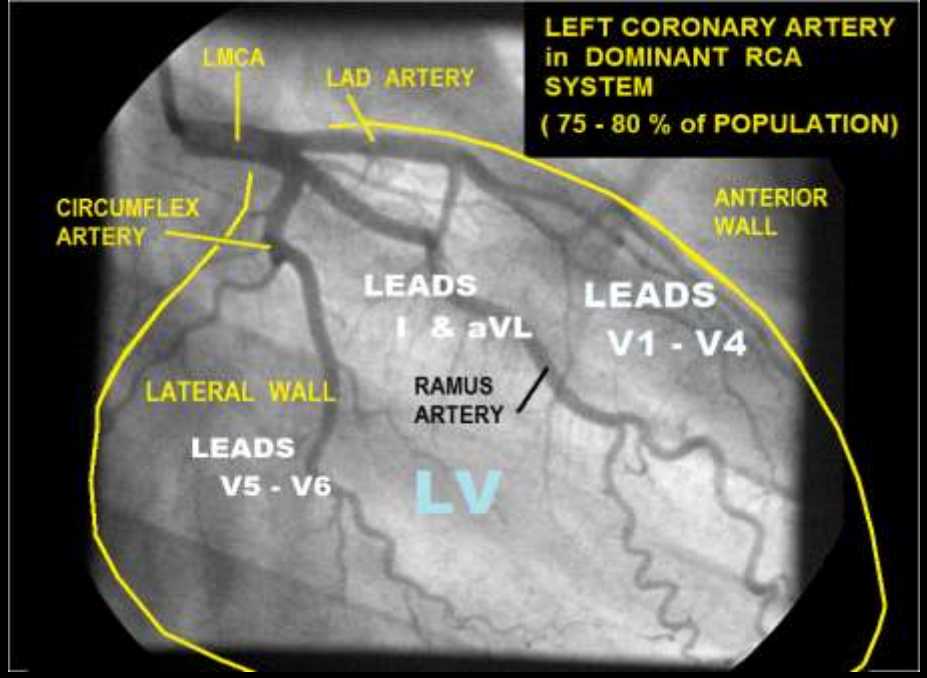


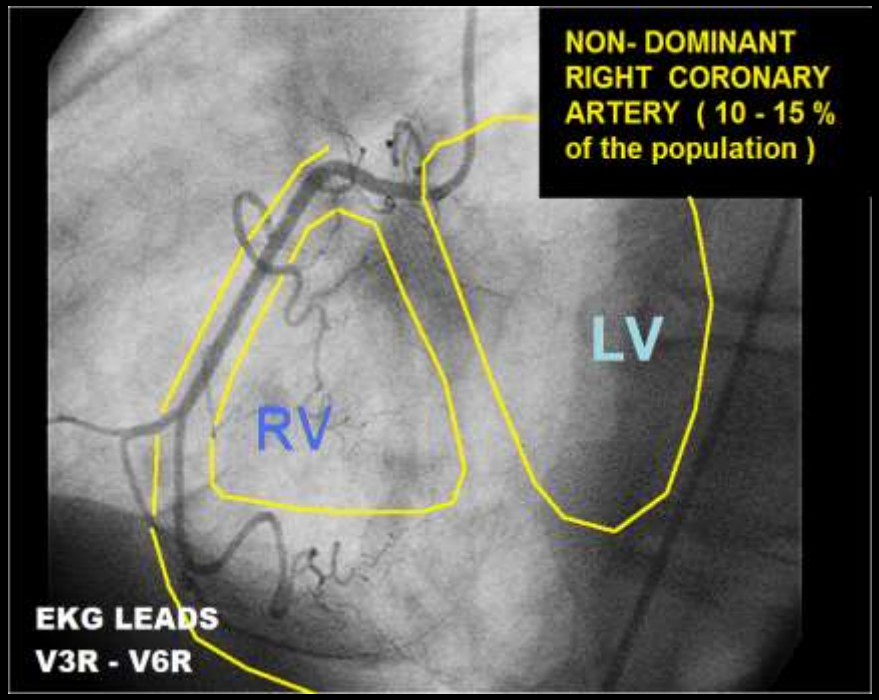
So if the Right Coronary Artery Is DOMINANT in 75 – 80% of the POPULATION, what accounts for the Other 20 – 25% ??

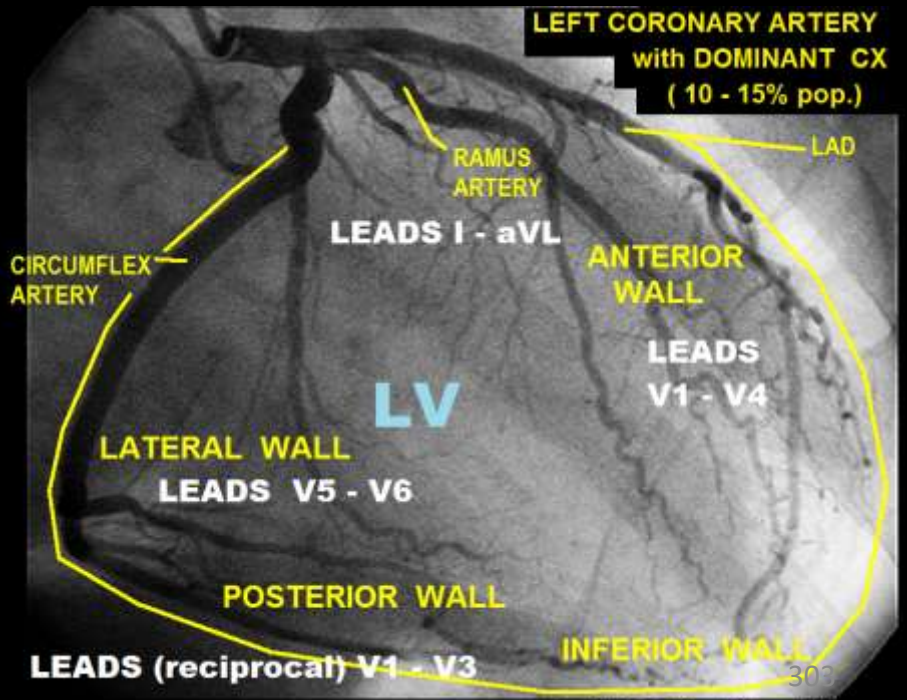

RIGHT DOMINANT
SA NODE
AV NODE
RCA
LV
RV
CX
75 - 80 %
POPULATION
POSTERIOR
VIEW

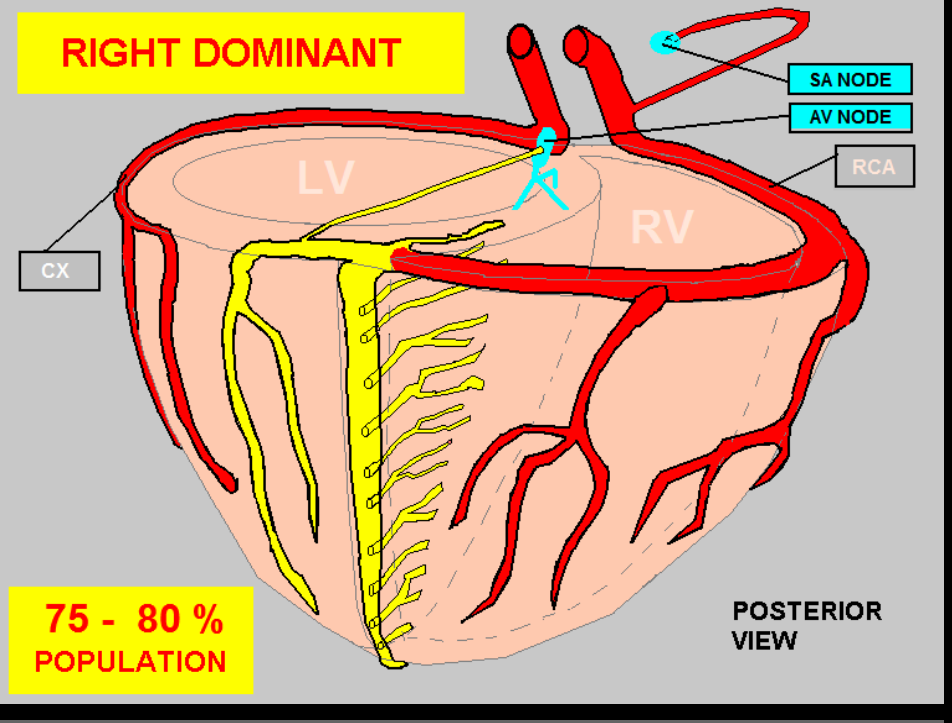

LEFT DOMINANT
SA NODE
AV NODE
RCA
LV
RV
CX
10 - 15 %
population
POSTERIOR
VIEW

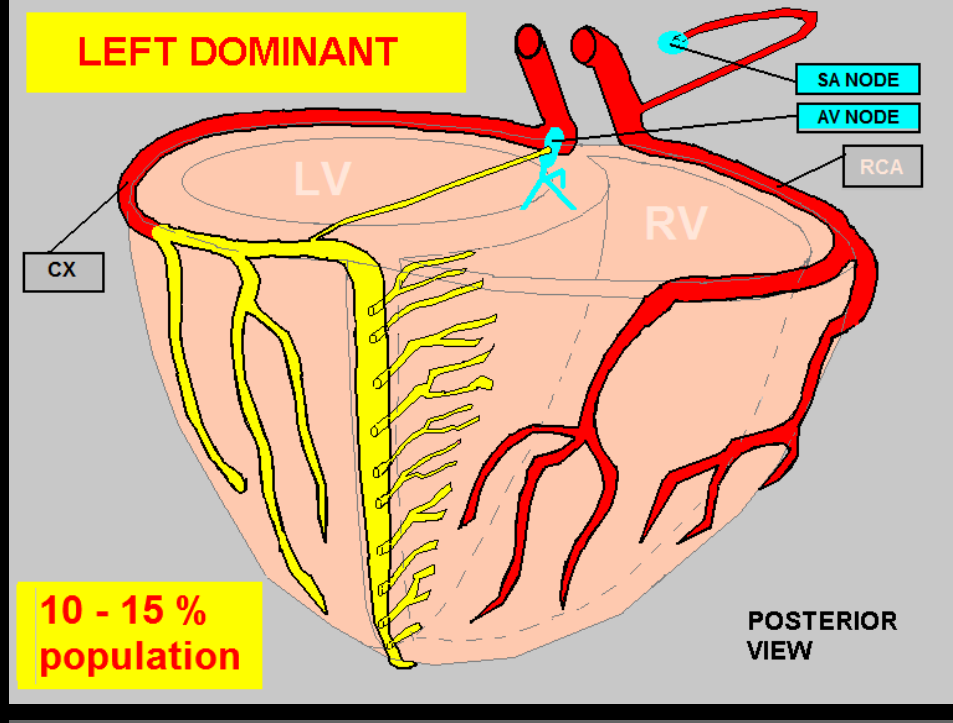

TIP of CATHETER
SA NODAL ARTERY
DOMINANT RIGHT CORONARY ARTERY - seen in 75 - 80 % of the POPULATION.
AV NODE
AV NODAL ARTERY
RV
PLVs
LEADS: (reciprocal) V1 - V3
POSTERIOR WALL of LV
RIGHT VENTRICULAR ARTERIES
PDA
INFERIOR WALL
LEADS II, III, aVF
LEADS V3R - V6R

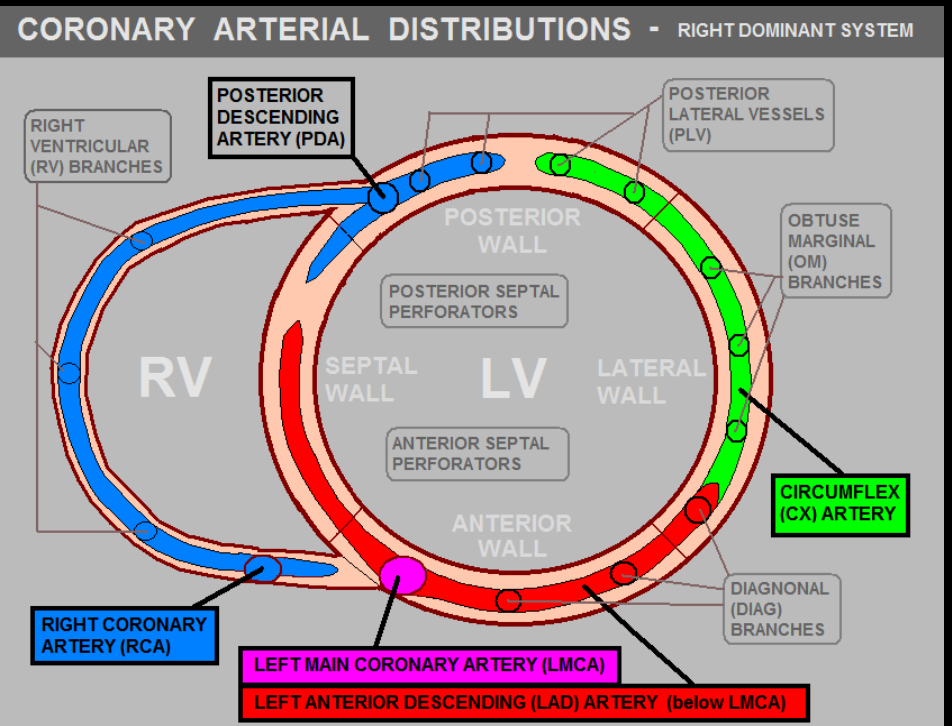

LMCA
LAD ARTERY
LEFT CORONARY ARTERY in DOMINANT RCA SYSTEM
(75 - 80 % of POPULATION)
CIRCUMFLEX ARTERY
ANTERIOR WALL
LEADS I & aVL
LEADS V1 - V4
LATERAL WALL
RAMUS ARTERY
LEADS V5 - V6
LV

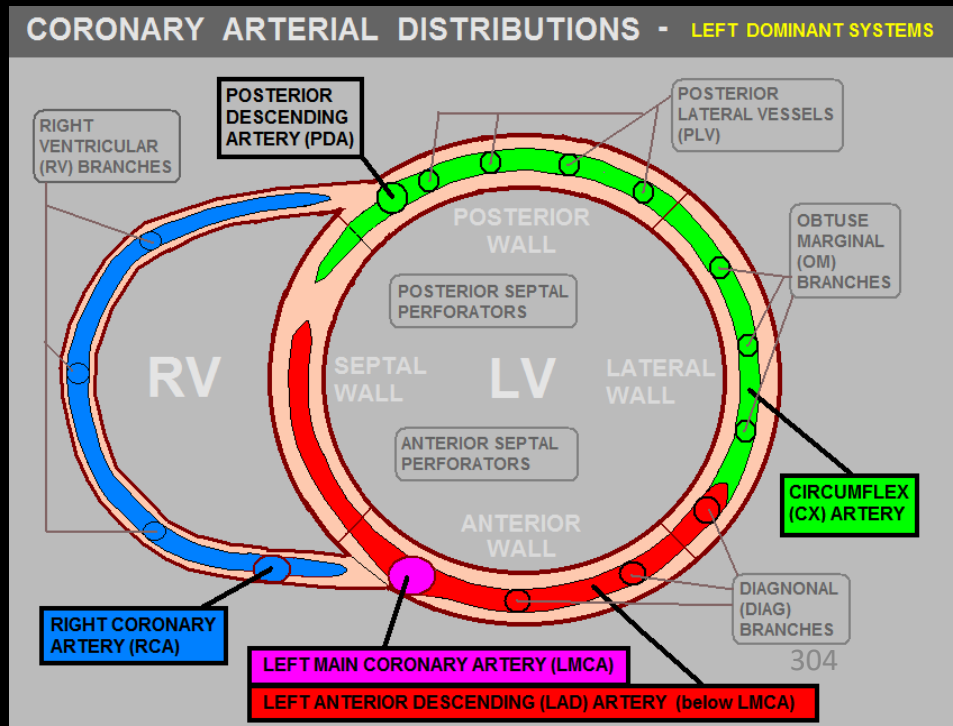

NON- DOMINANT RIGHT CORONARY ARTERY (10 - 15 % of the population)
LV
RV
EKG LEADS V3R - V6R


LEFT CORONARY ARTERY with DOMINANT CX (10 - 15% pop.)
RAMUS ARTERY
LAD
LEADS I - aVL
CIRCUMFLEX ARTERY
ANTERIOR WALL
LEADS V1 - V4
LV
LATERAL WALL
LEADS V5 - V6
POSTERIOR WALL
INFERIOR WALL
LEADS (reciprocal) V1 - V3
LEADS II, III, aVF


RIGHT DOMINANT
SA NODE
AV NODE
RCA
CX
LV
RV
75 - 80 %
POPULATION
POSTERIOR
VIEW


LEFT DOMINANT
SA NODE
AV NODE
RCA
CX
LV
RV
10 - 15 %
population
POSTERIOR
VIEW


CORONARY ARTERIAL DISTRIBUTIONS - RIGHT DOMINANT SYSTEM
POSTERIOR DESCENDING ARTERY (PDA)
POSTERIOR LATERAL VESSELS (PLV)
RIGHT VENTRICULAR (RV) BRANCHES
OBTUSE MARGINAL (OM) BRANCHES
POSTERIOR WALL
POSTERIOR SEPTAL PERFORATORS
RV
SEPTAL WALL
LV
LATERAL WALL
ANTERIOR SEPTAL PERFORATORS
ANTERIOR WALL
CIRCUMFLEX (CX) ARTERY
DIAGNONAL (DIAG) BRANCHES
RIGHT CORONARY ARTERY (RCA)
LEFT MAIN CORONARY ARTERY (LMCA)
LEFT ANTERIOR DESCENDING (LAD) ARTERY (below LMCA)


CORONARY ARTERIAL DISTRIBUTIONS - LEFT DOMINANT SYSTEMS
POSTERIOR DESCENDING ARTERY (PDA)
POSTERIOR LATERAL VESSELS (PLV)
RIGHT VENTRICULAR (RV) BRANCHES
OBTUSE MARGINAL (OM) BRANCHES
POSTERIOR WALL
POSTERIOR SEPTAL PERFORATORS
RV
SEPTAL WALL
LV
LATERAL WALL
ANTERIOR SEPTAL PERFORATORS
ANTERIOR WALL
CIRCUMFLEX (CX) ARTERY
DIAGNONAL (DIAG) BRANCHES
RIGHT CORONARY ARTERY (RCA)
LEFT MAIN CORONARY ARTERY (LMCA)
LEFT ANTERIOR DESCENDING (LAD) ARTERY (below LMCA)

Pat ID [redacted]
01/20/2021 07:46:46
08/17/1955 65 yrs
Account # [redacted]
RX
DX

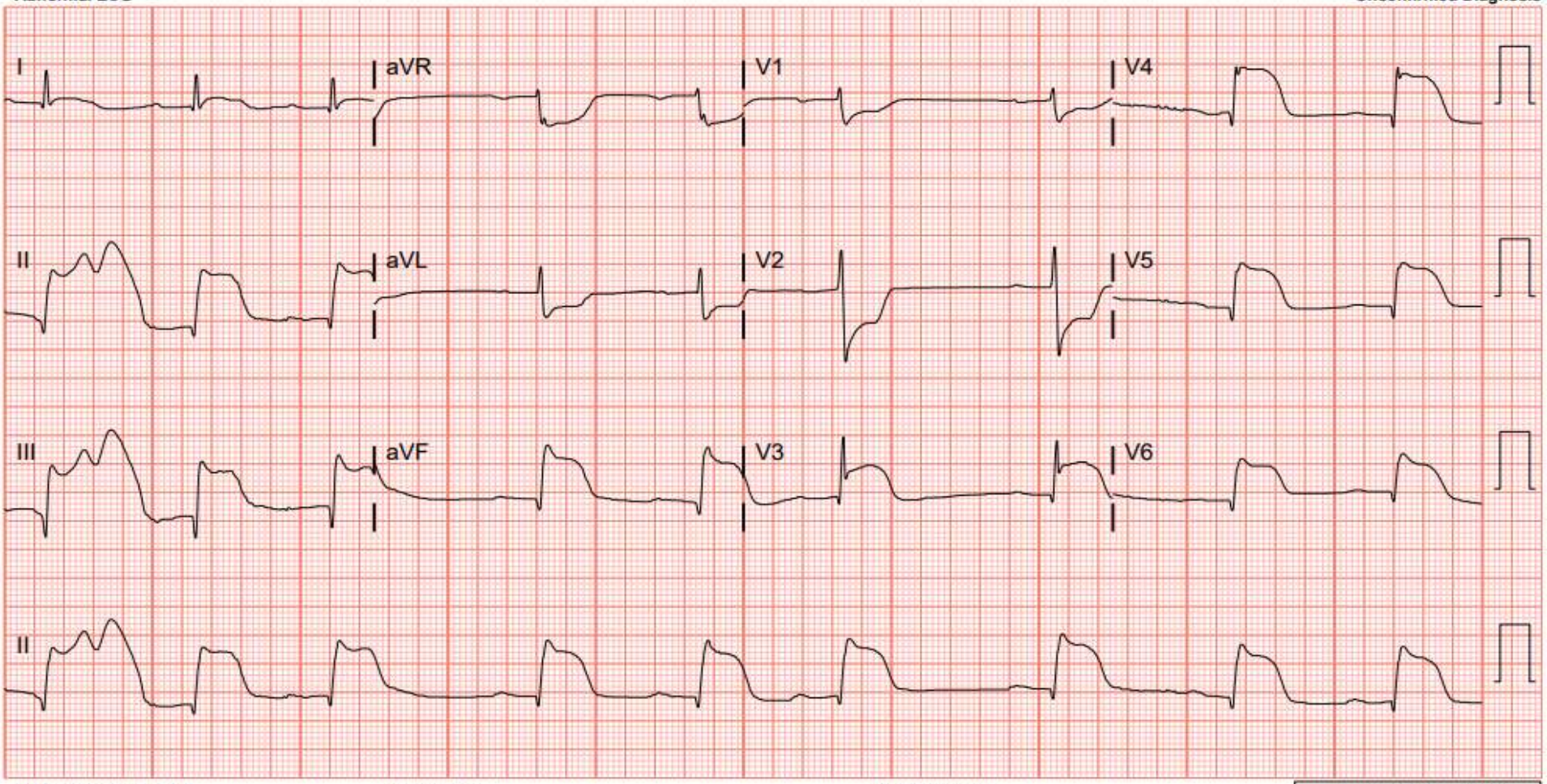
Bayfront Health Seven Rivers ED
Dept ED
Room EDWR
Tech mg

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	NO PREVIOUS ECG AVAILABLE FOR COMPARISON
T	77	

Req Provider: Xandus Chen

- Abnormal ECG -

Unconfirmed Diagnosis



Philips PageWriter TC 25 mm/sec 10.00 mm/mV F 60~ 0.15 - 100 Hz

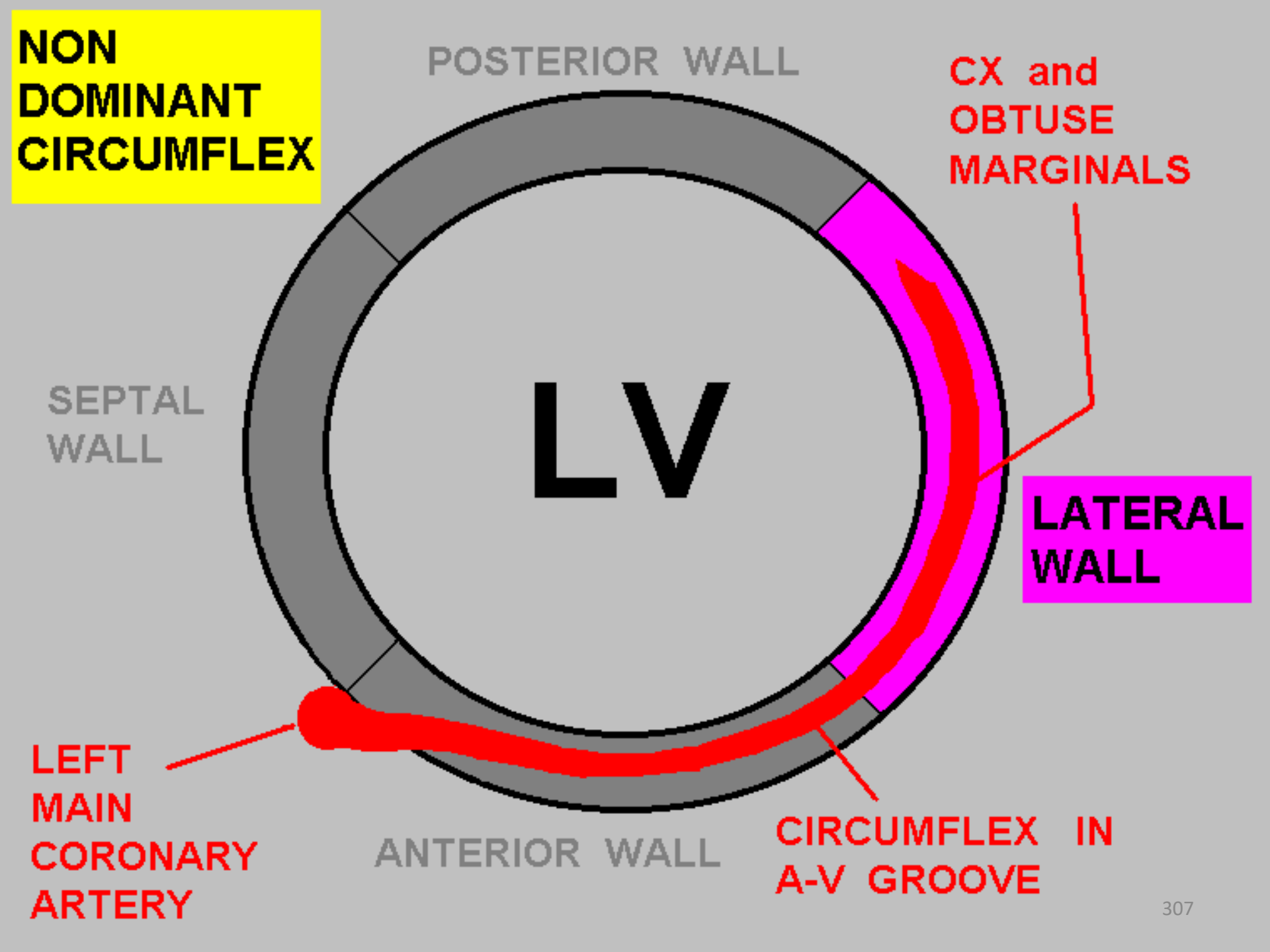
CIRCUMFLEX ARTERY (CX)

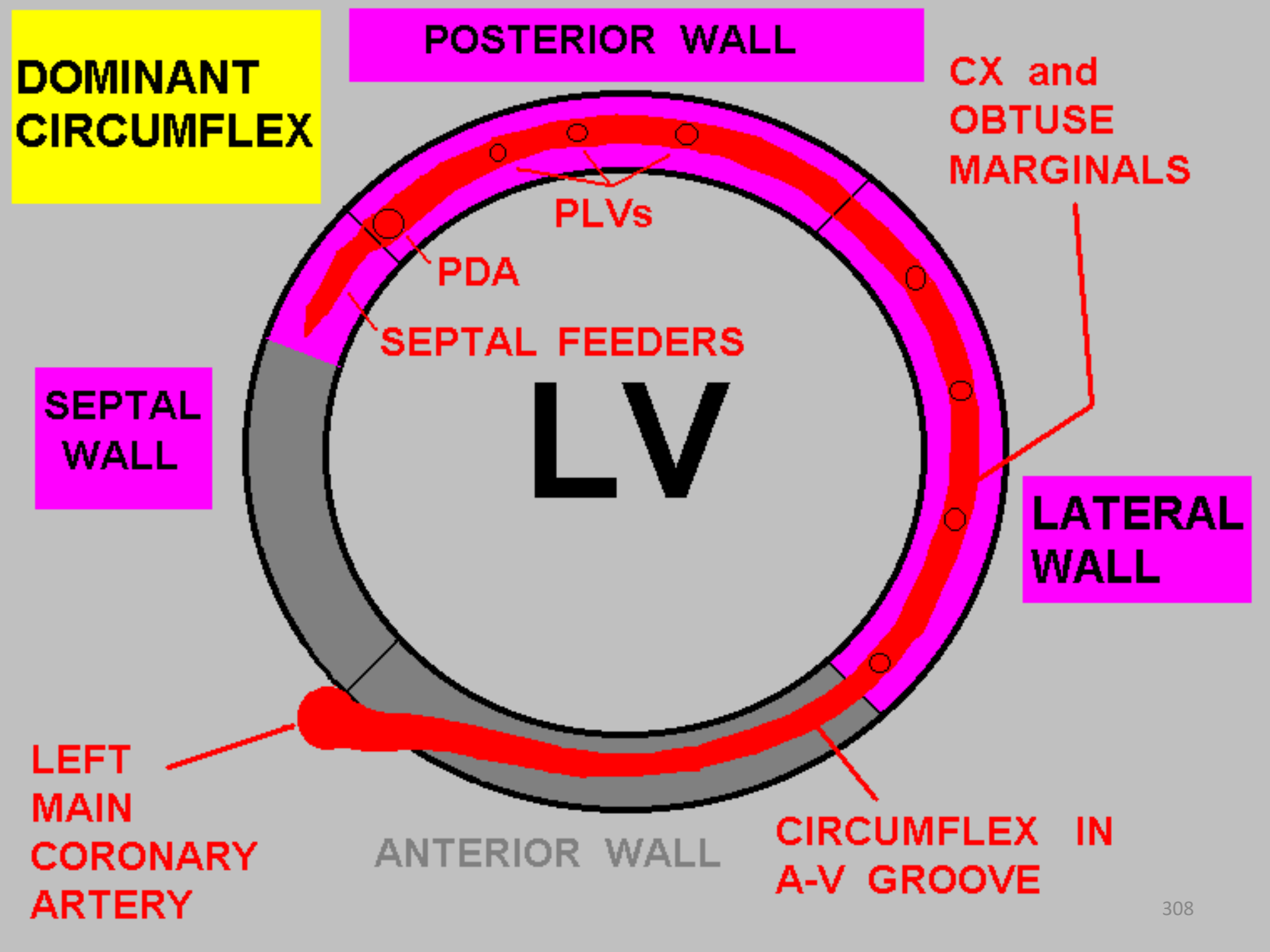
- NON-DOMINANT CX:

CX = 15 - 30% OF LV MASS

- DOMINANT CX:

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

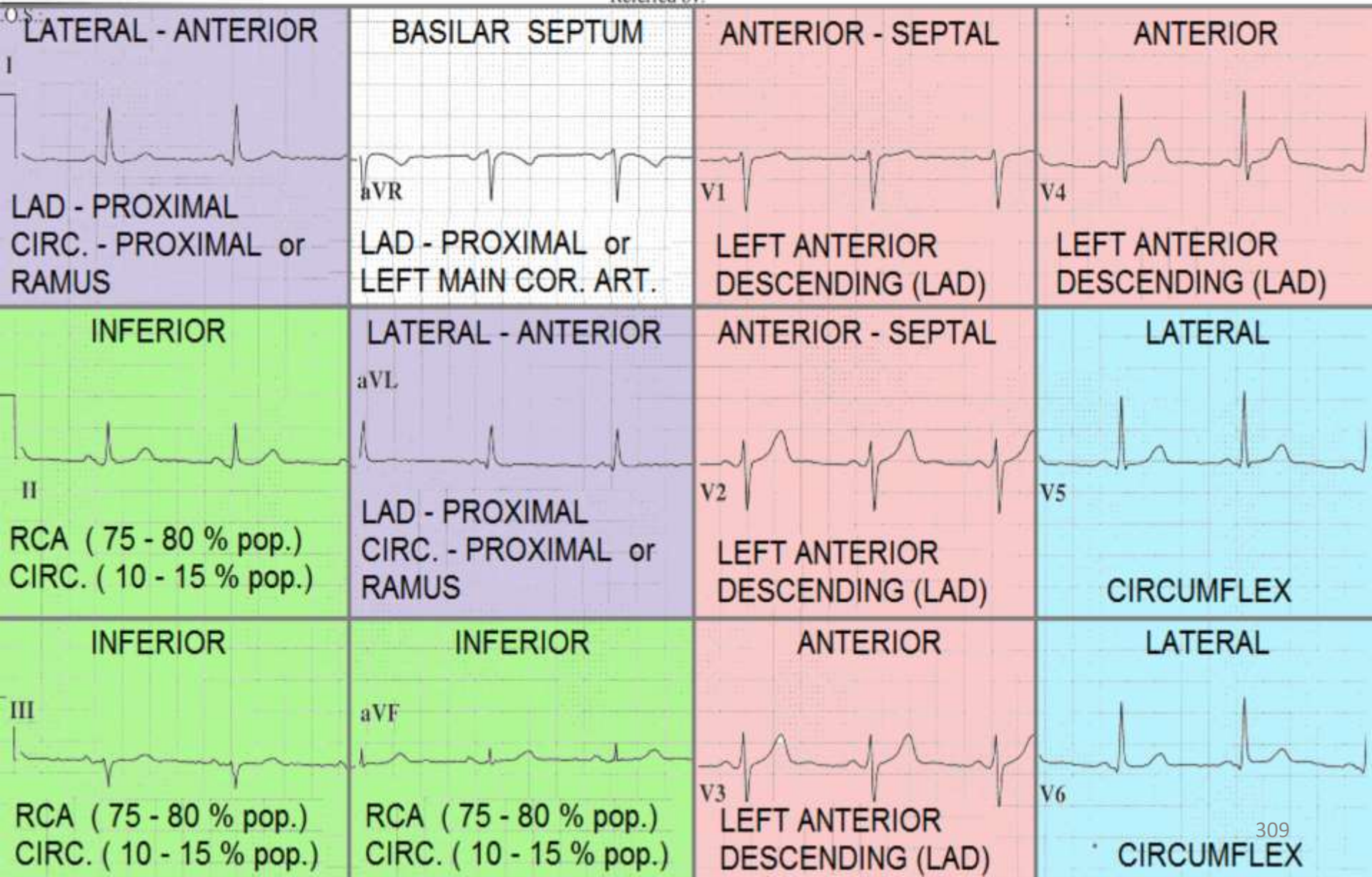

NON DOMINANT CIRCUMFLEX
POSTERIOR WALL
CX and OBTUSE MARGINALS
SEPTAL WALL
LV
LATERAL WALL
LEFT MAIN CORONARY ARTERY
ANTERIOR WALL
CIRCUMFLEX IN A-V GROOVE


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

Vent. rate	64	BPM
PR interval	130	ms
QRS duration	96	ms
QT/QTc	396/408	ms
P–R–T axes	40 11	61

Normal sinus rhythm
Normal ECG
No previous ECGs available

Referred by:

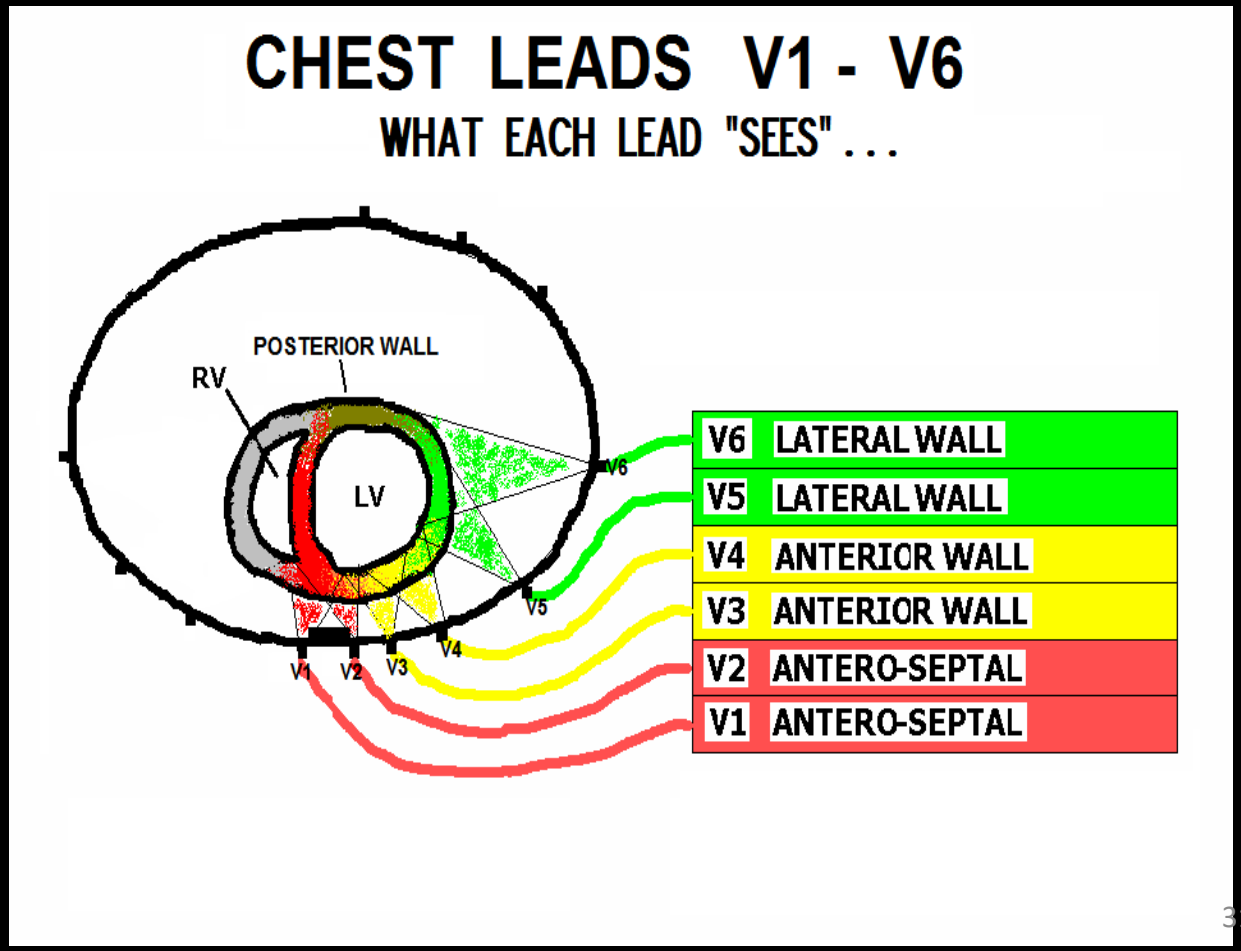


The 12 Lead ECG
Has TWO major BLIND SPOTS . . . . .

The POSTERIOR WALL

&

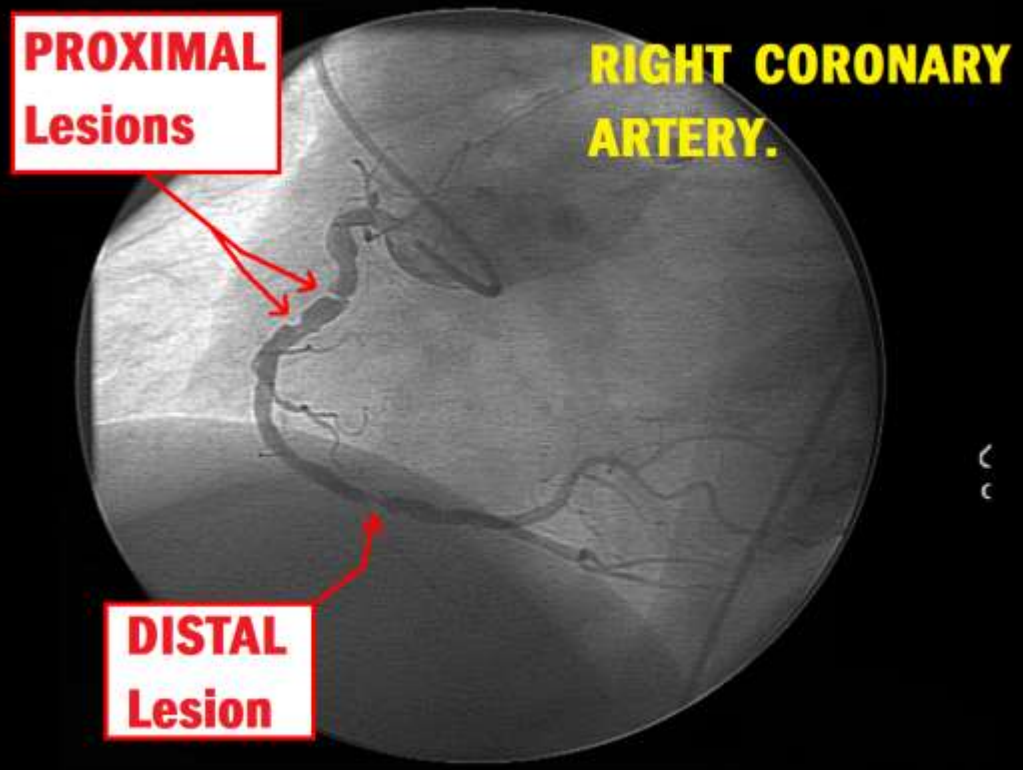
RIGHT VENTRICLE

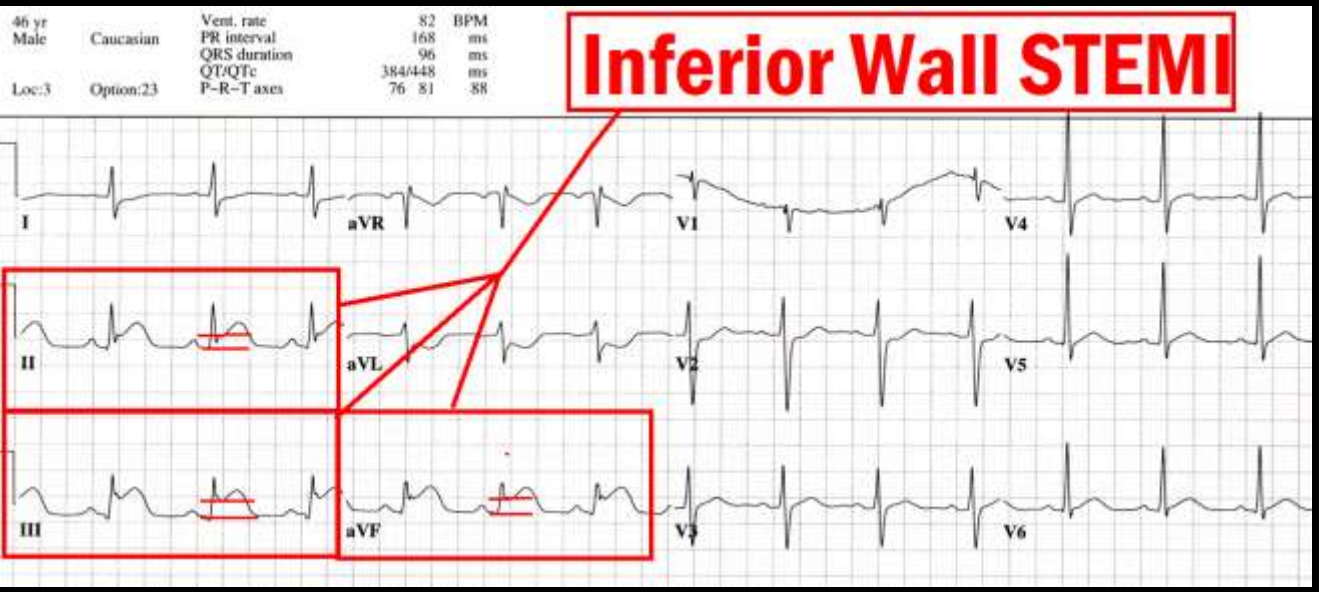


When do we need to see the Right Ventricle?

- 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.





To see the

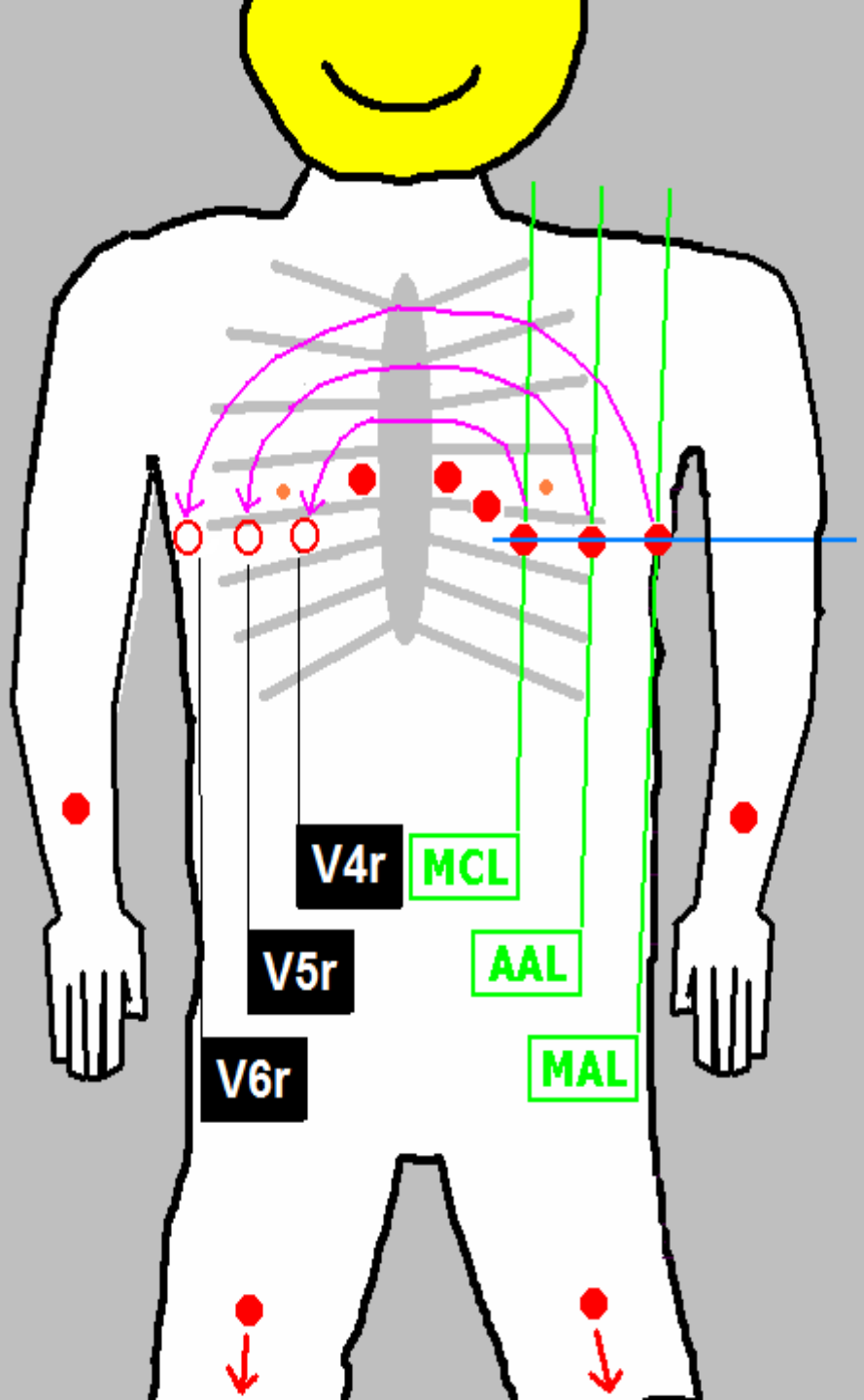
RIGHT VENTRICLE . . .

. . . such as in cases of

INFERIOR WALL M.I.

☞ You must do a

RIGHT - SIDED EKG ! !

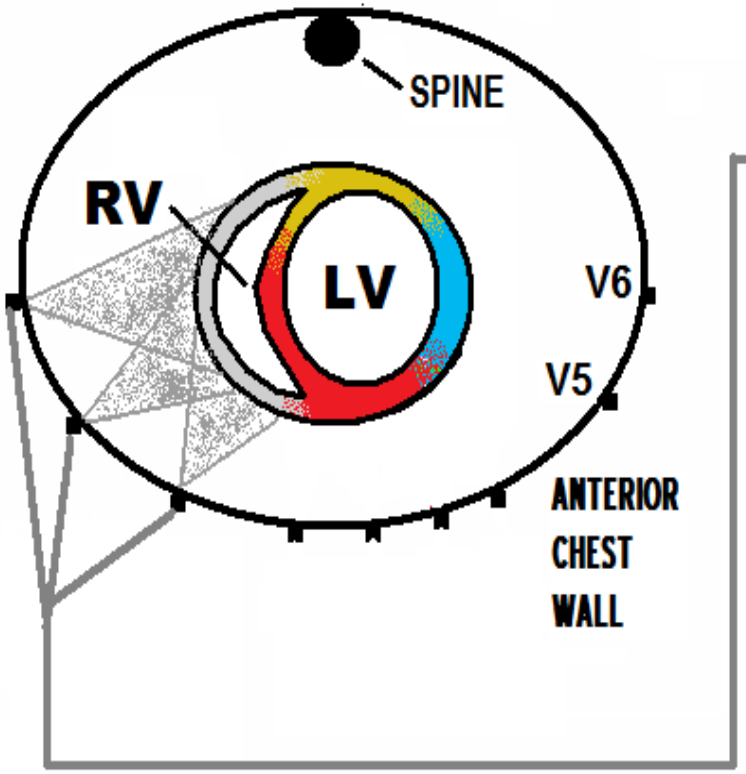


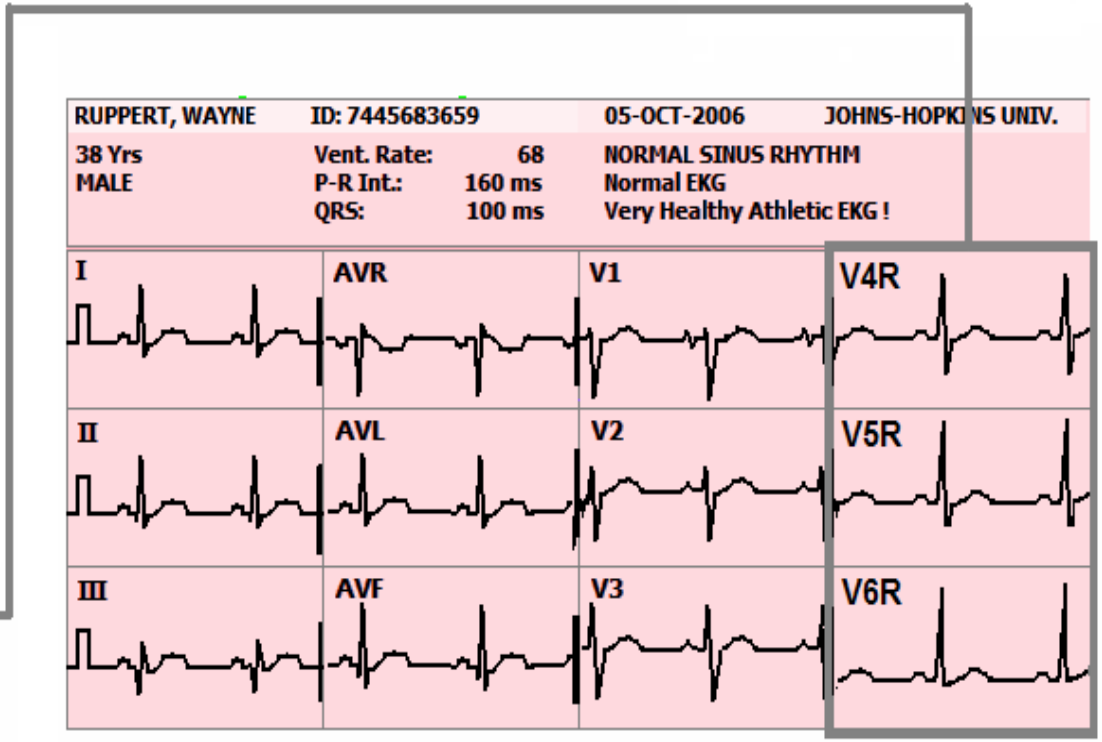
To do a
RIGHT - SIDED EKG . .

MOVE leads
V4, V5, and V6

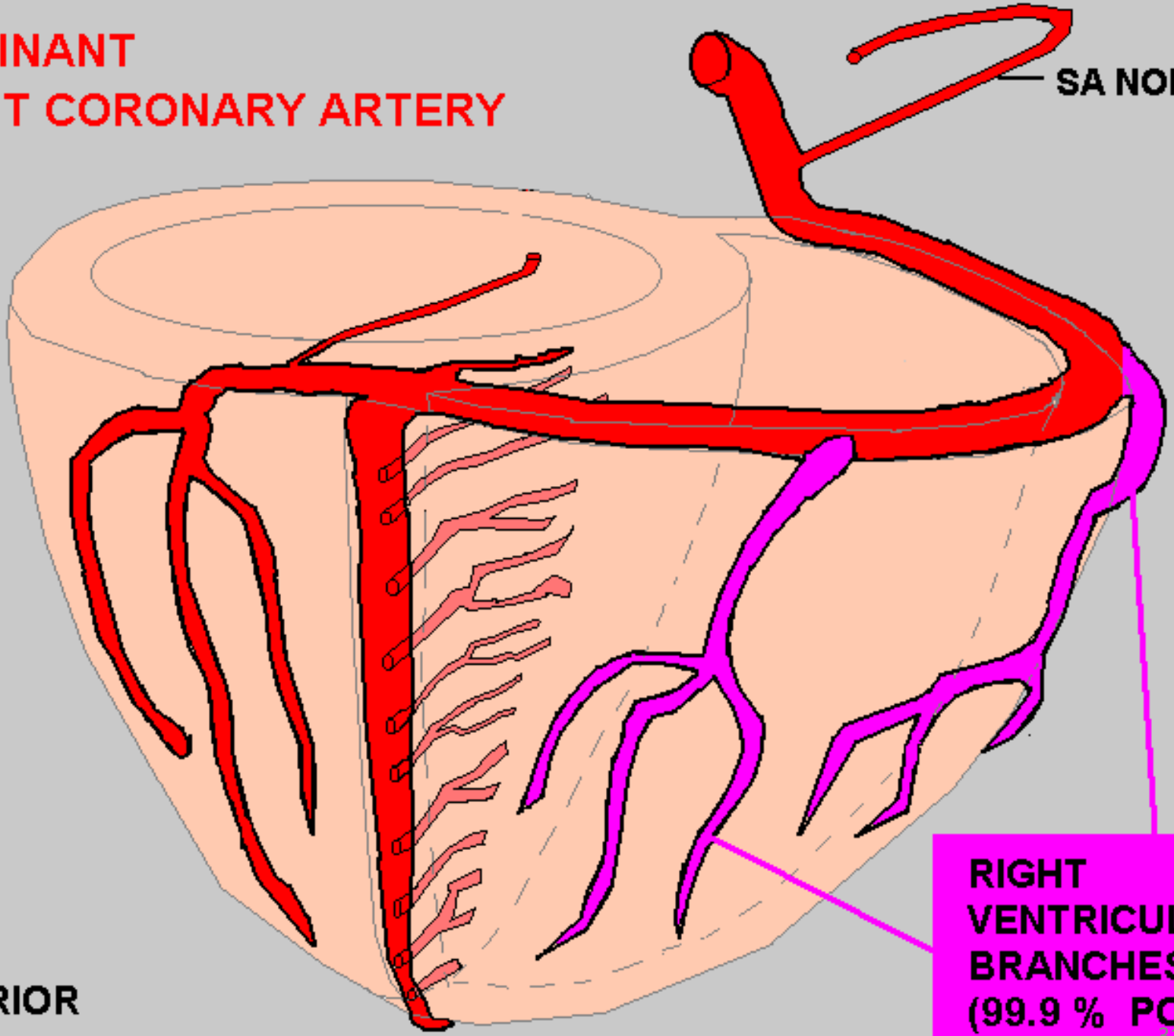
to the corresponding placement on the RIGHT SIDE of patient's chest . . .

V4R - V6R VIEW THE RIGHT VENTRICLE





DOMINANT
RIGHT CORONARY ARTERY



ID:

46 yo
Male Caucasian

Room:

Opt:

Technician:

Vent. rate	87 bpm
PR interval	176 ms
QRS duration	94 ms
QT/QTc	330/397 ms
P-R-T axes	79 81 102

Normal sinus rhythm
~~Anterolateral infarct, possibly acute~~
Inferior injury pattern
** ** ** ** * Acute MI * ** ** ** **
Abnormal ECG

Right Ventricular Infarct

V LEADS
R SIDE

Referred by:

Unconfirmed

I aVR V1 ~~V4~~ V4r

II aVL V2 ~~V5~~ V5r

III aVF ~~V3~~ V3r ~~V6~~ V6r

VI

II

V5r

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

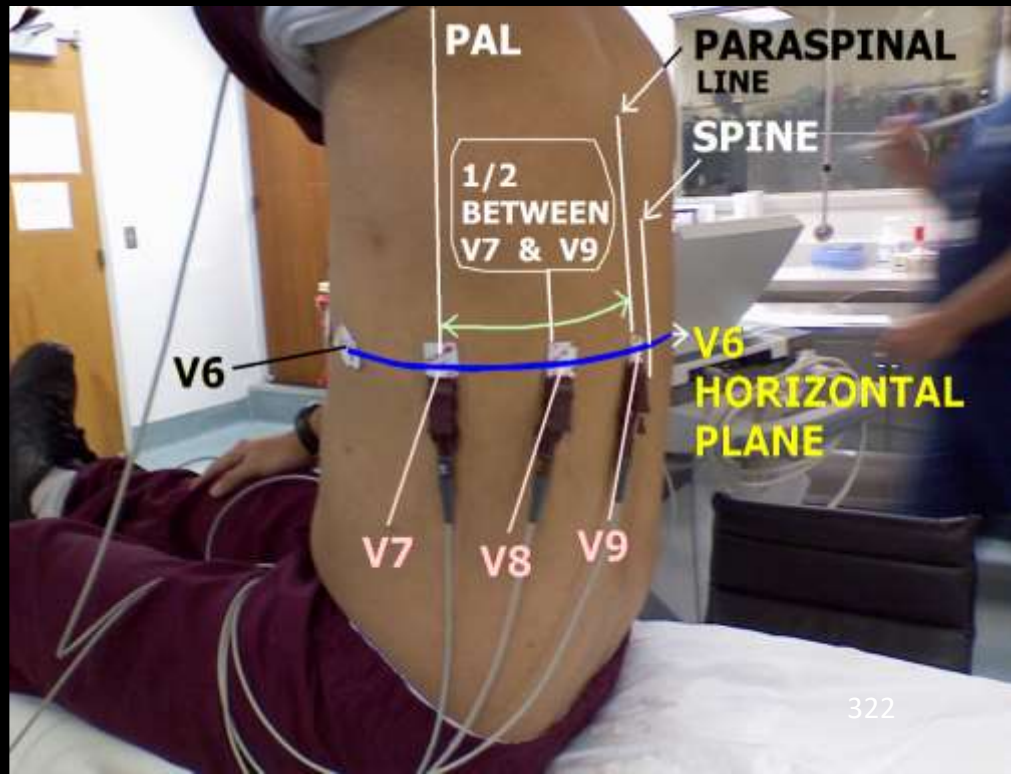
(V7 - V9)

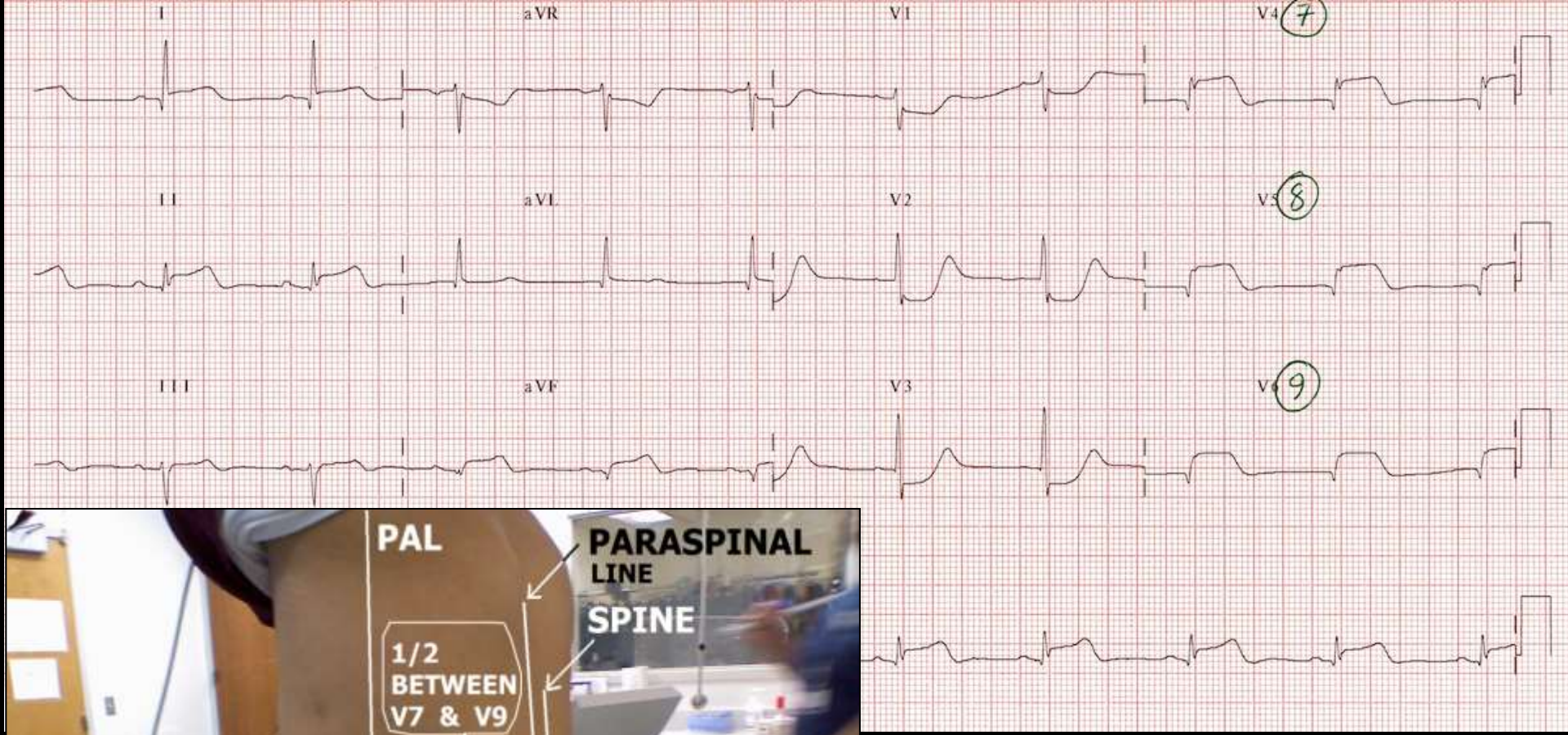
to see if you Patient is having a

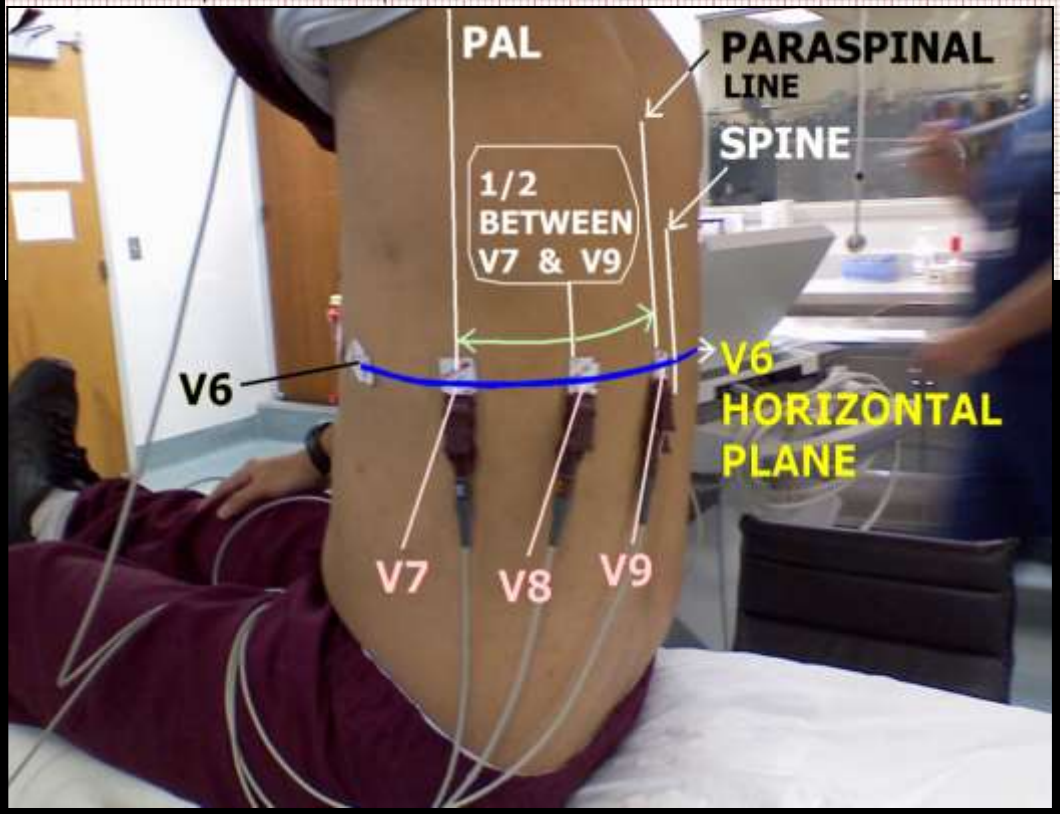
POSTERIOR WALL STEMI

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

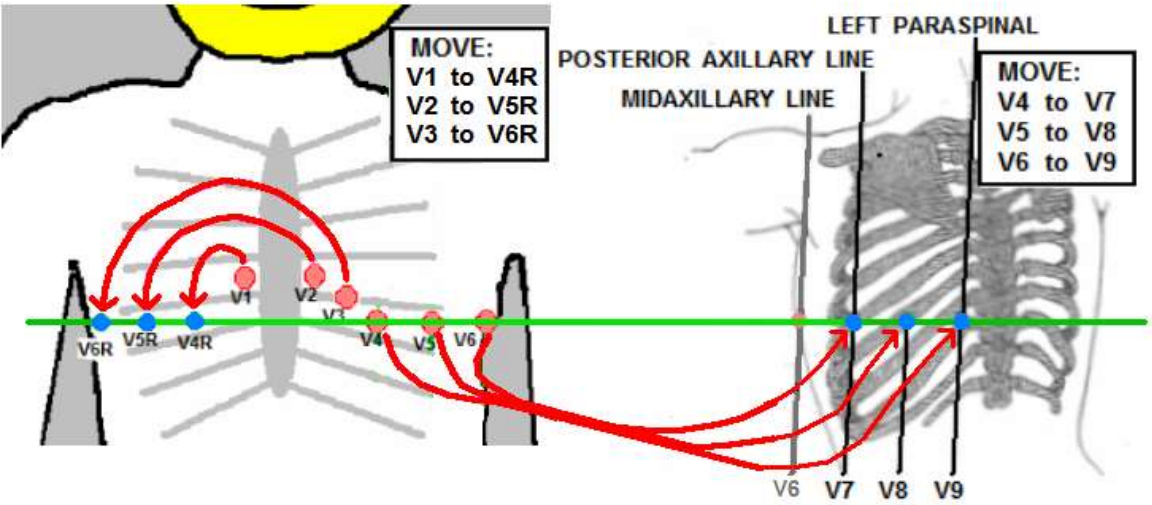
. . . To DIGANOSE Posterior Wall STEMI, we should see LEADS V7 – V9 !!




I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
7
8
9

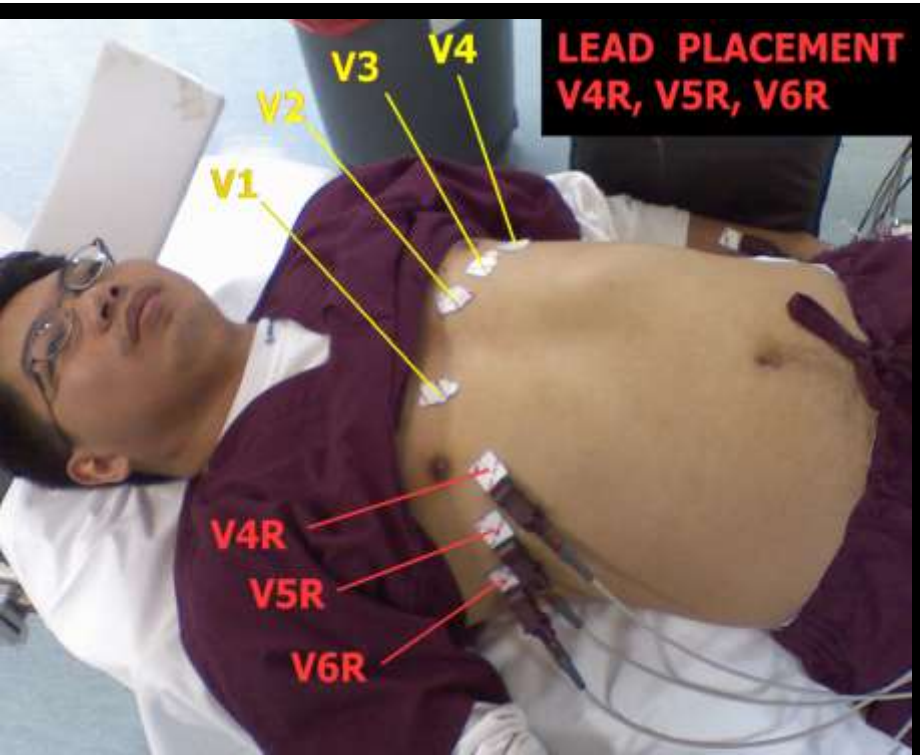

PAL
PARASPINAL LINE
SPINE
1/2 BETWEEN V7 & V9
V6
V6 HORIZONTAL PLANE
V7
V8
V9

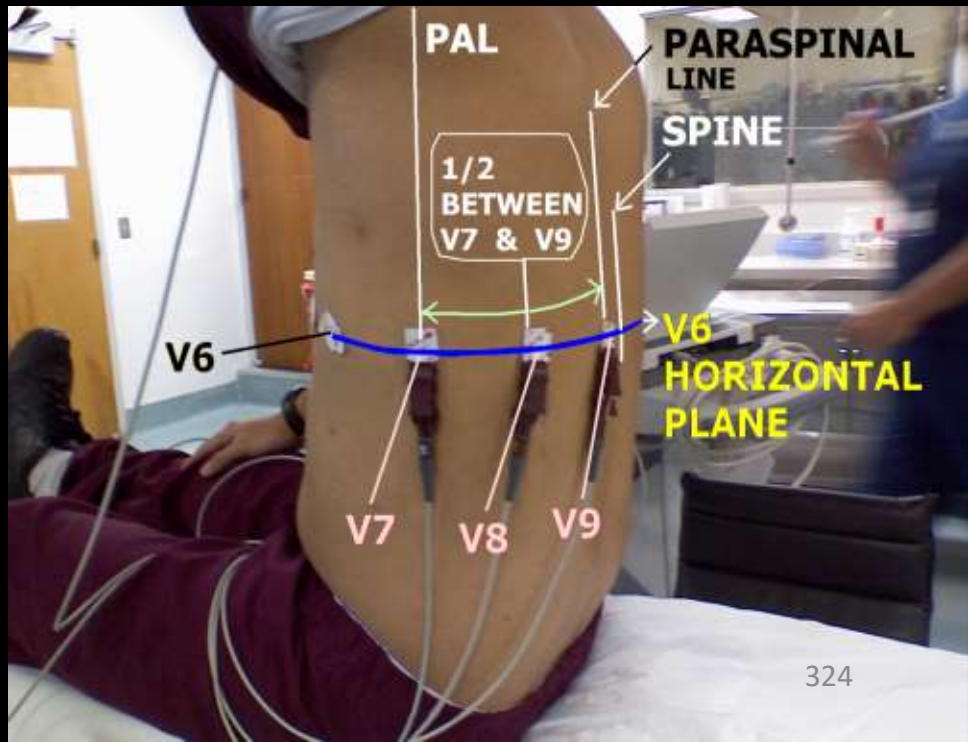
HOW TO REPOSITION 6 CHEST LEADS to OBTAIN 3 R VENTRICLE and 3 POSTERIOR LEADS

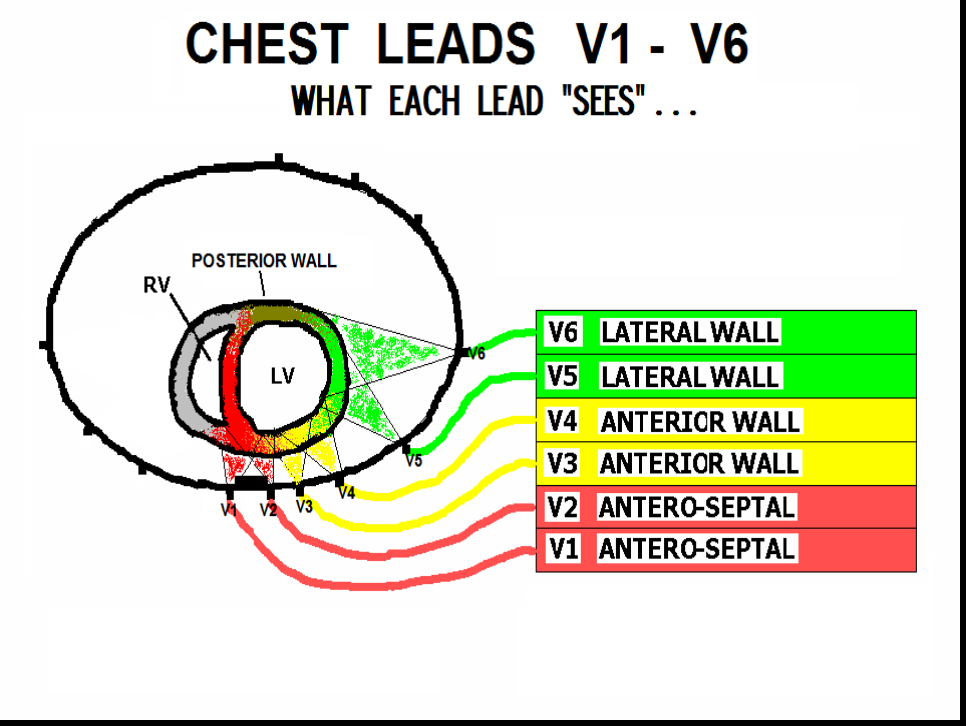


ANTERIOR VIEW

POSTERIOR VIEW

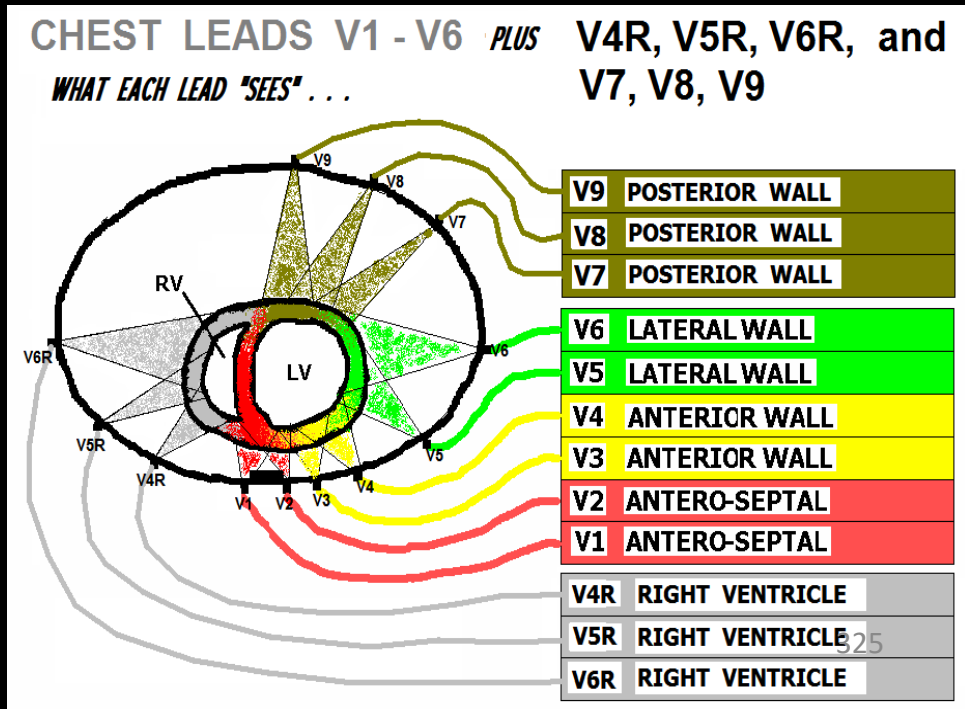






⇦ The 12 Lead ECG

The 18 Lead ECG ⇨



34years
Male Asian

Room:

Opt:

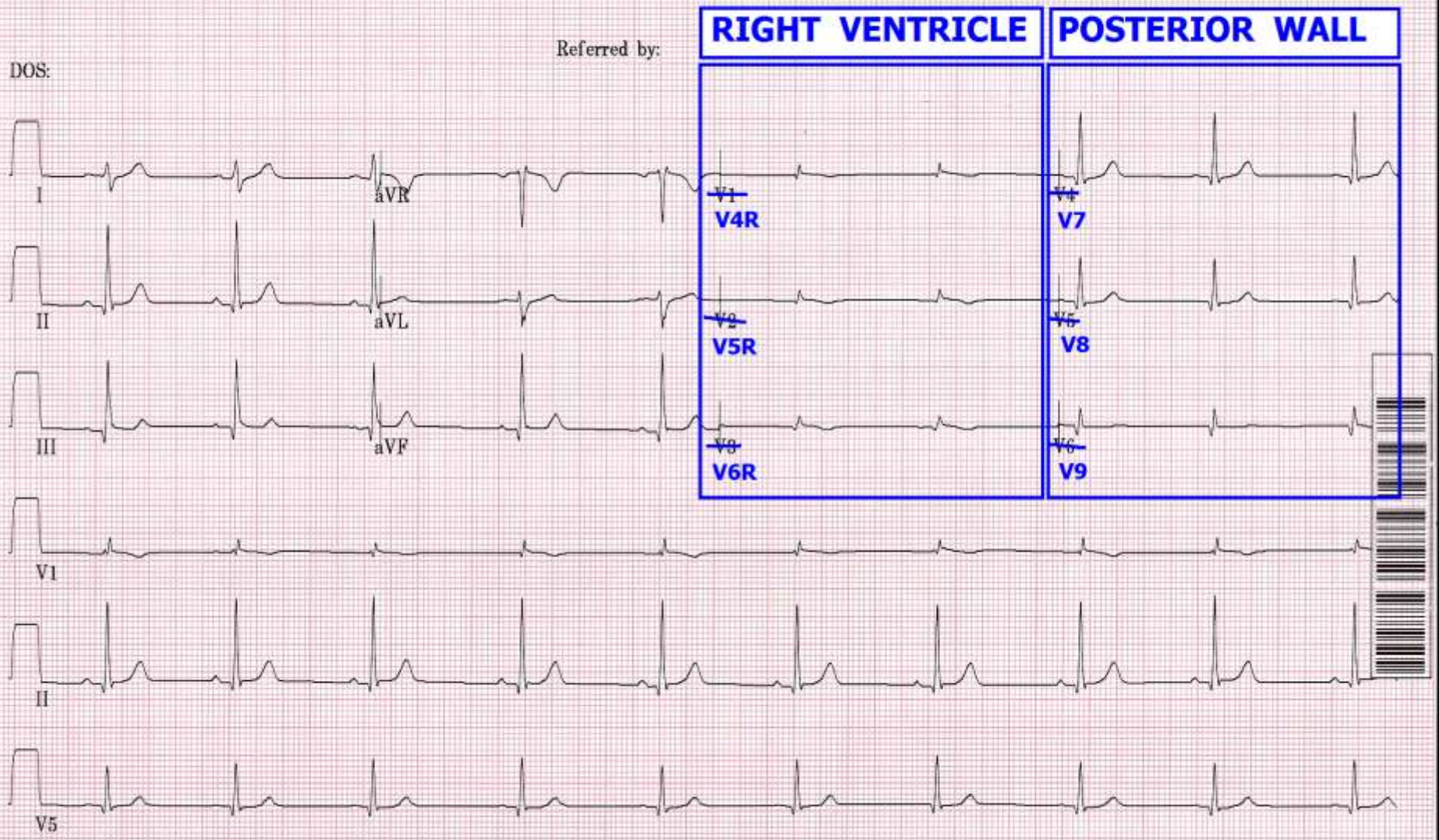
Technician: WR

Vent. rate 58 bpm
PR interval 146 ms
QRS duration 82 ms
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~~

Referred by:

DOS:



40 Hz 25.0 mm/s 10.0 mm/mV 4 by 2.5s + 3 rhythm lds MAC5K 007A.2 12SL™v235

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

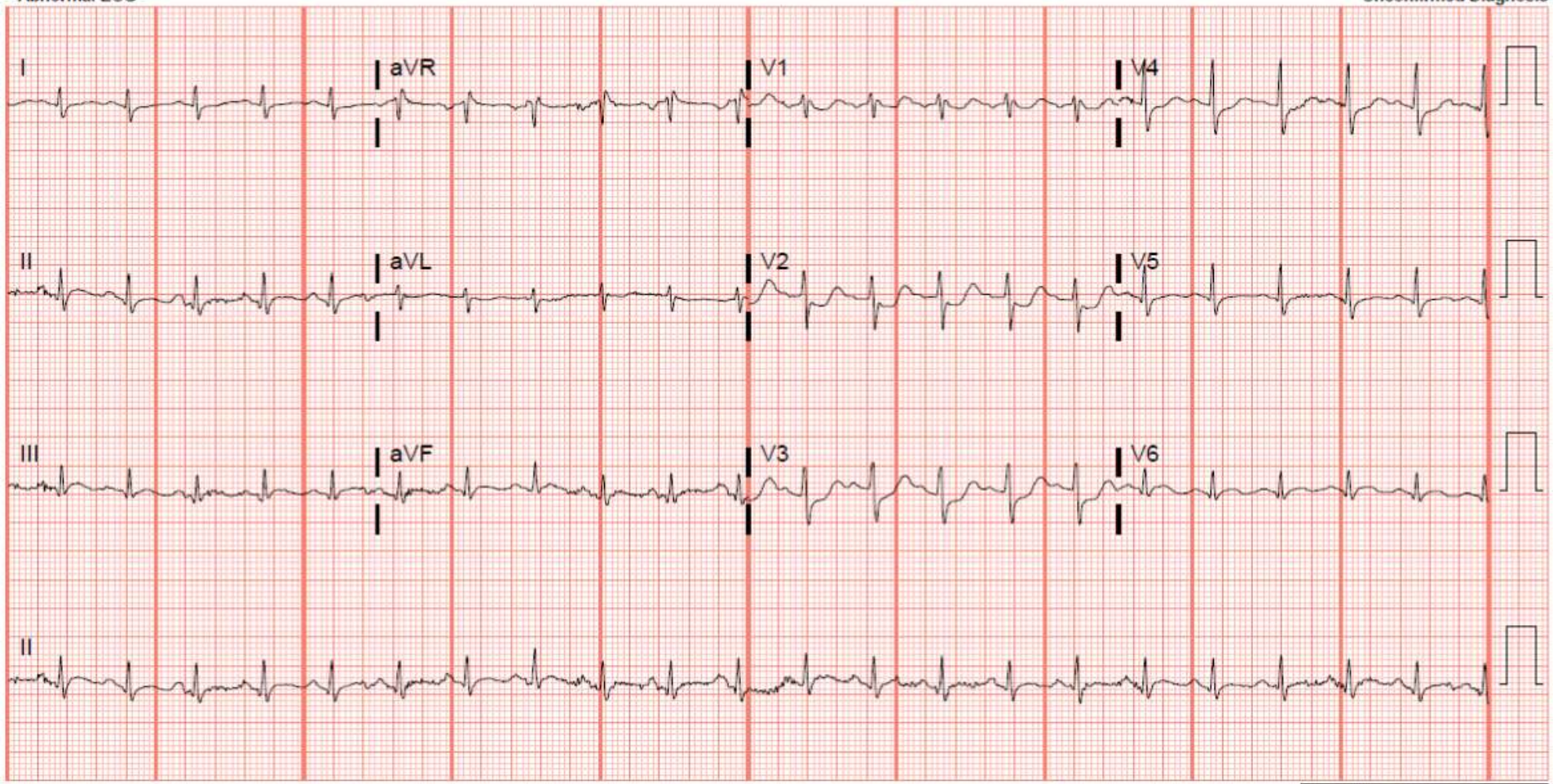
Pat ID [redacted]
[redacted] 2019 22:07:54
[redacted] 46 yrs
[redacted]
Caucasian Female
Account # [redacted]
Bayfront Health Seven Rivers ED
Dept ED
Room [redacted]
Tech LDC
RX
DX

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	

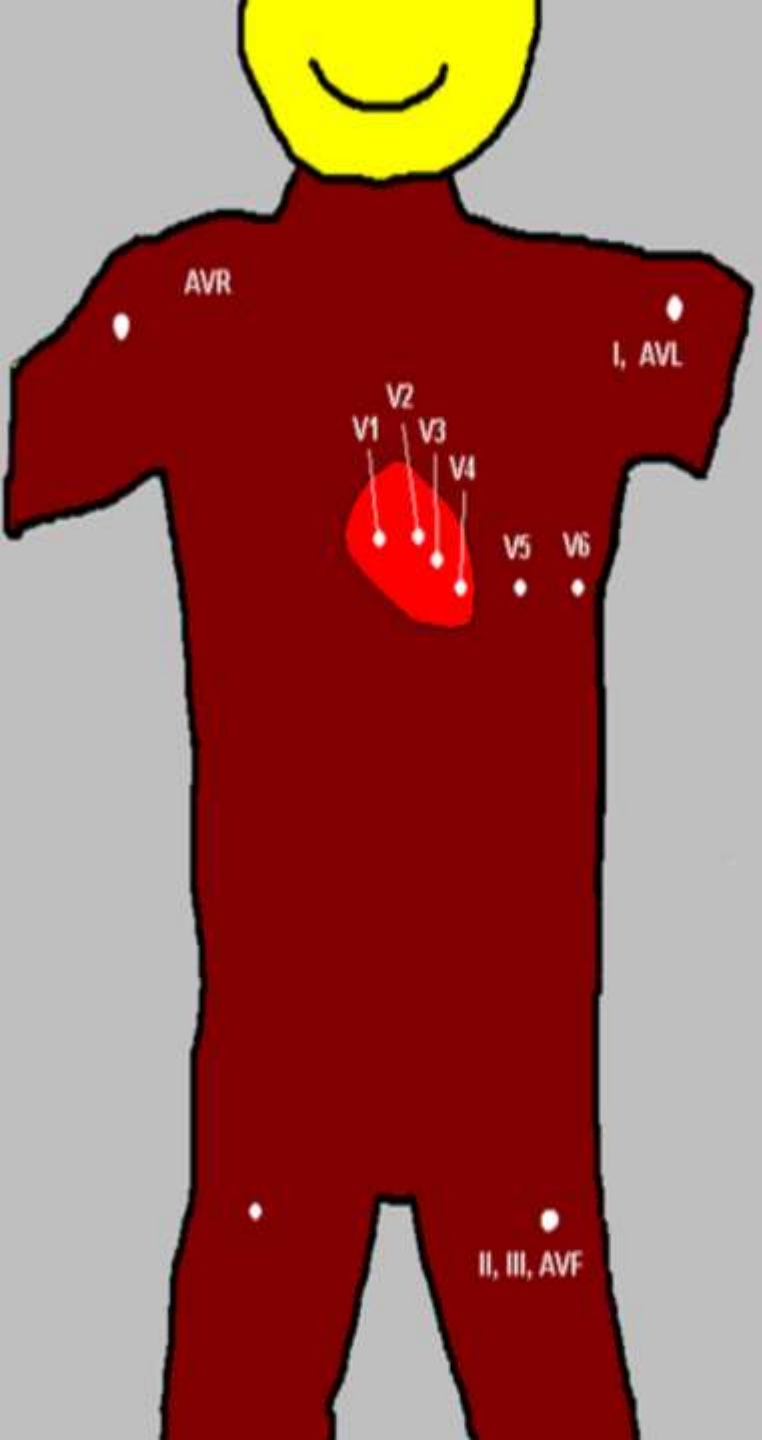
Req Provider: CHARLES NOLES

- Abnormal ECG -

Unconfirmed Diagnosis



Philips PageWriter TC 25 mm/sec 10.00 mm/mV F 60~ 0.15 - 100 Hz



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

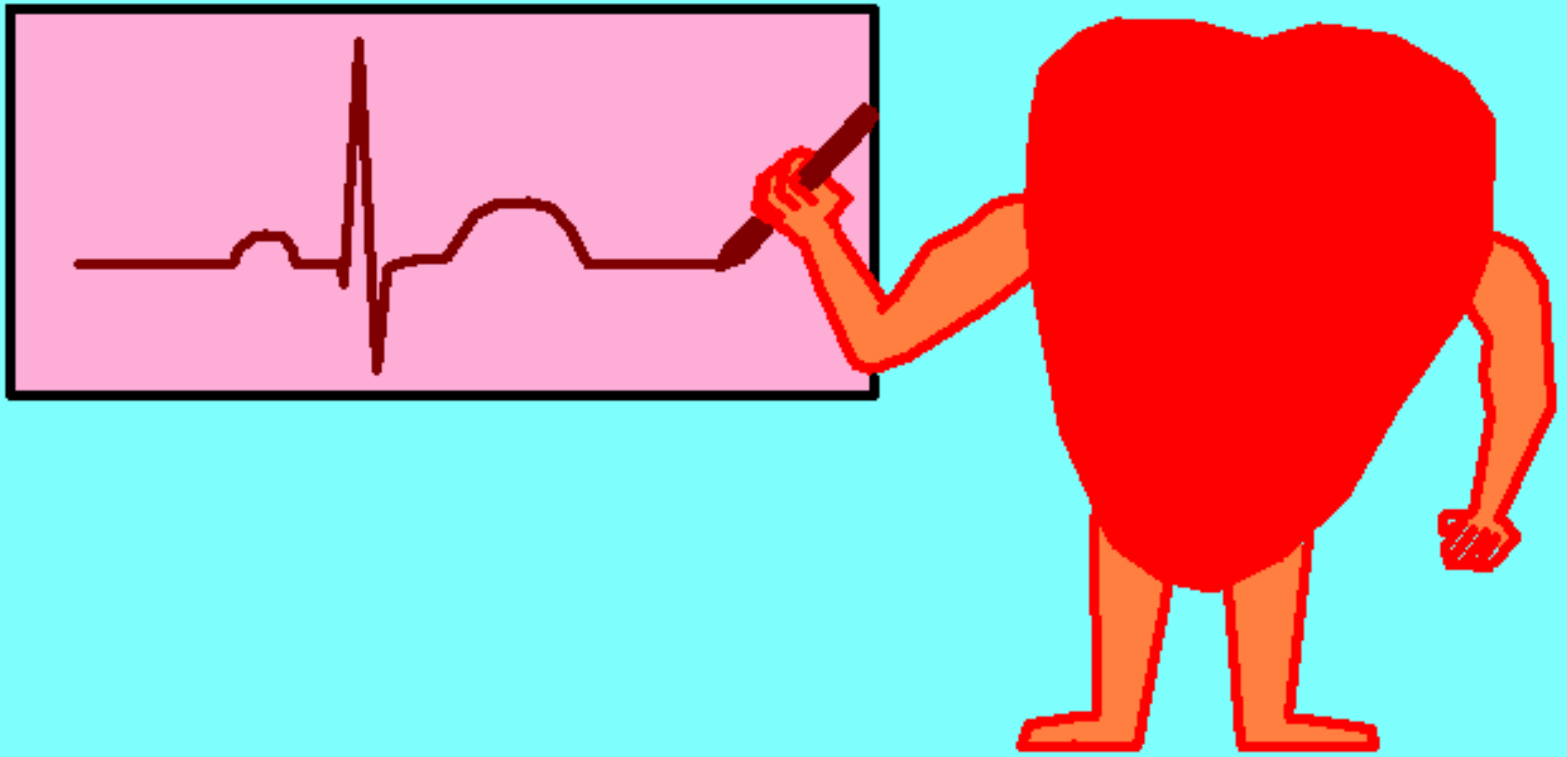
RIGHT DOMINANT and LEFT DOMINANT systems account for approximately 90 % of the population. . . . .

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

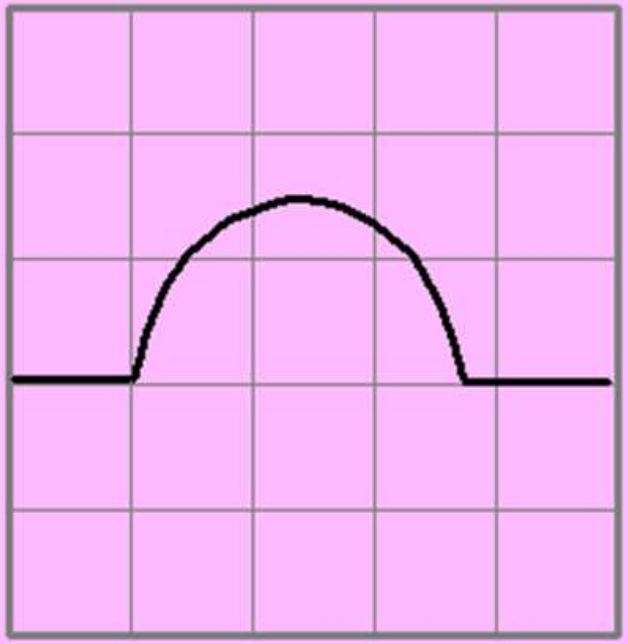
PUTTING IT ALL ON PAPER . . .

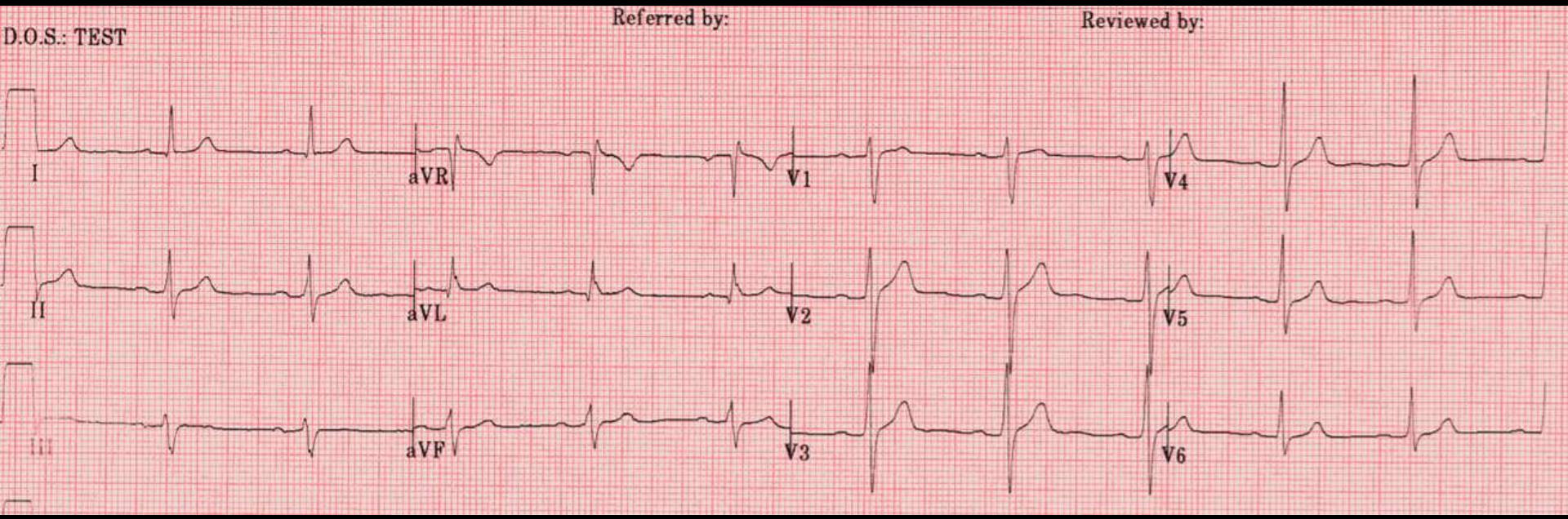
WAVEFORMS and INTERVALS . . .



THE P WAVE

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

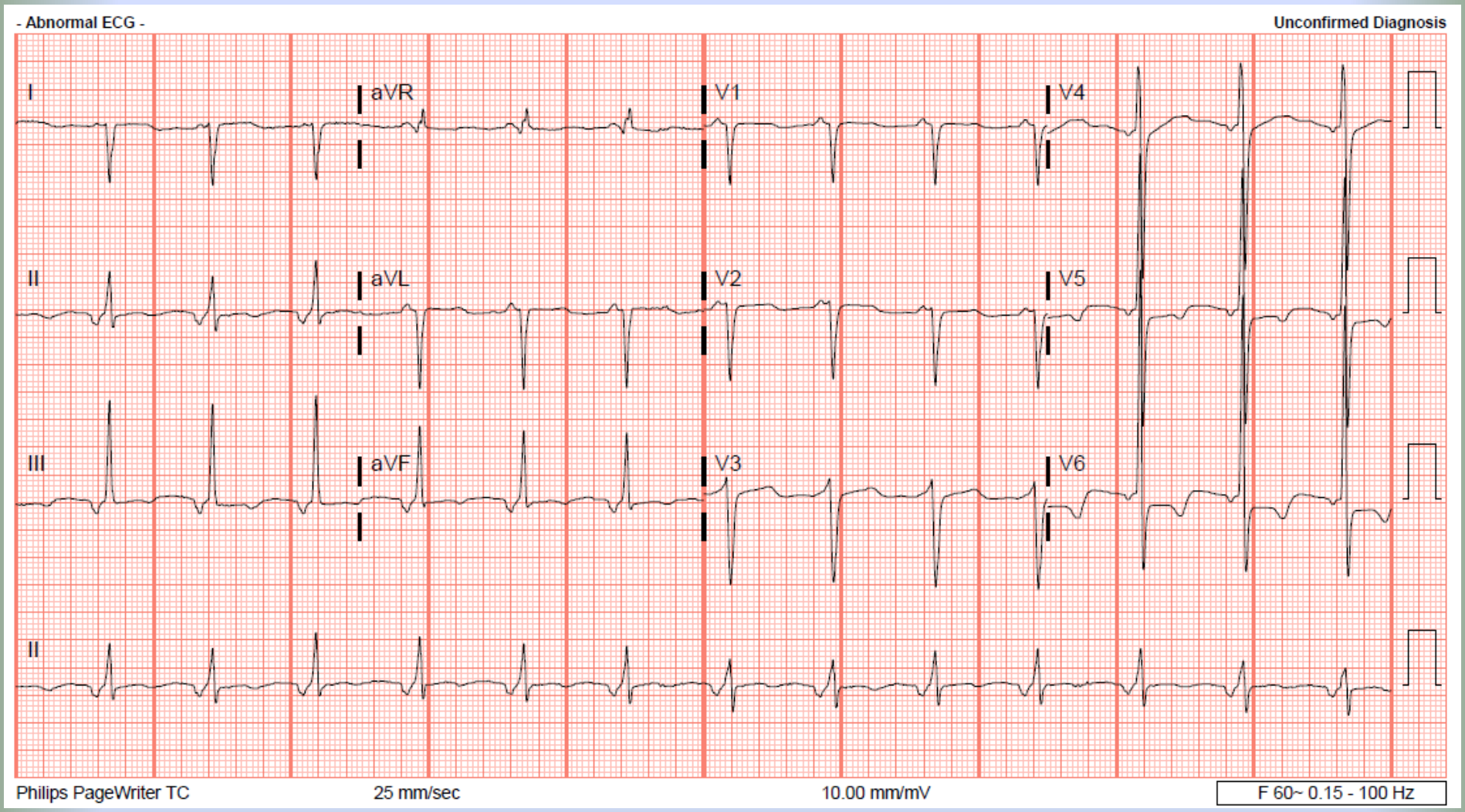



D.O.S.: TEST
Referred by:
Reviewed by:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

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 (<120ms) the origin of the rhythm may be the AV node (Junctional Rhythm).

Inverted P waves & short P-R interval:

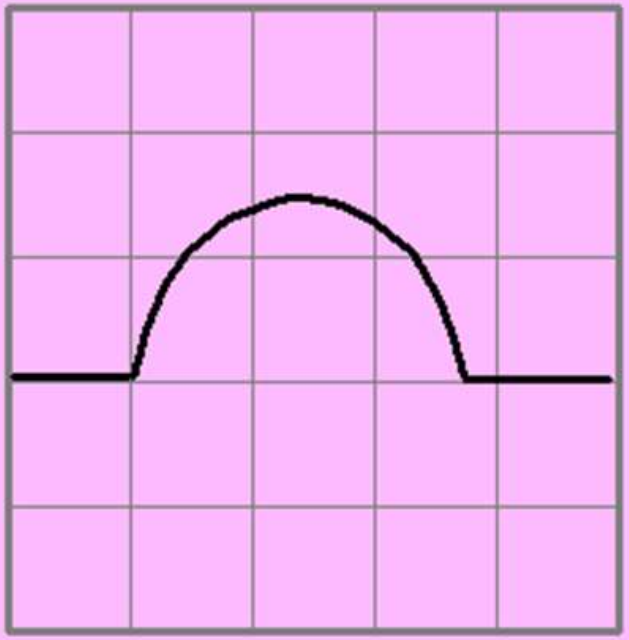


Evaluate P Wave for Atrial Hypertrophy

- Evaluate amplitude and duration in Lead II

THE P WAVE

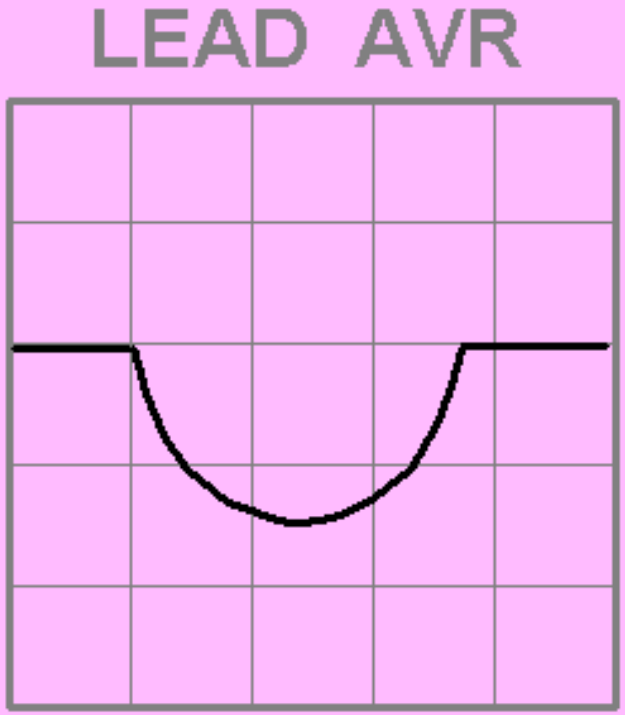
- SHOULD BE UPRIGHT, CONVEX-SHAPED DOME IN ALL LEADS EXCEPT AVR and V1



- 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



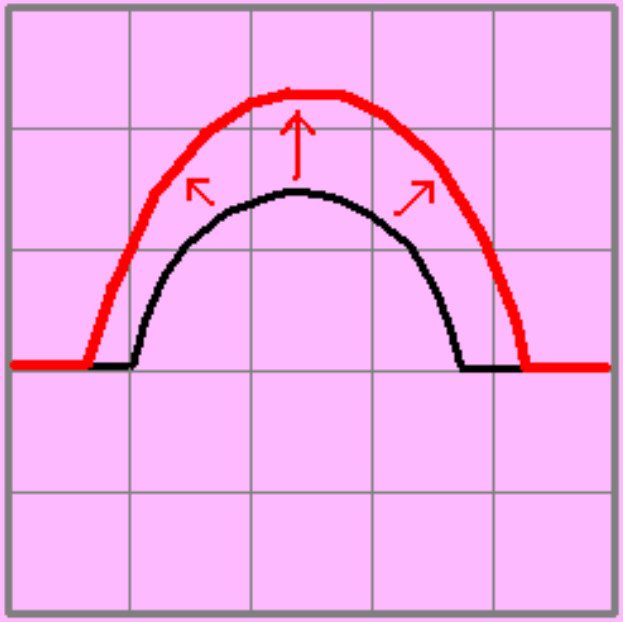
THE P WAVE

When the P WAVE

is

TOO LARGE

We think of



ATRIAL HYPERTROPHY

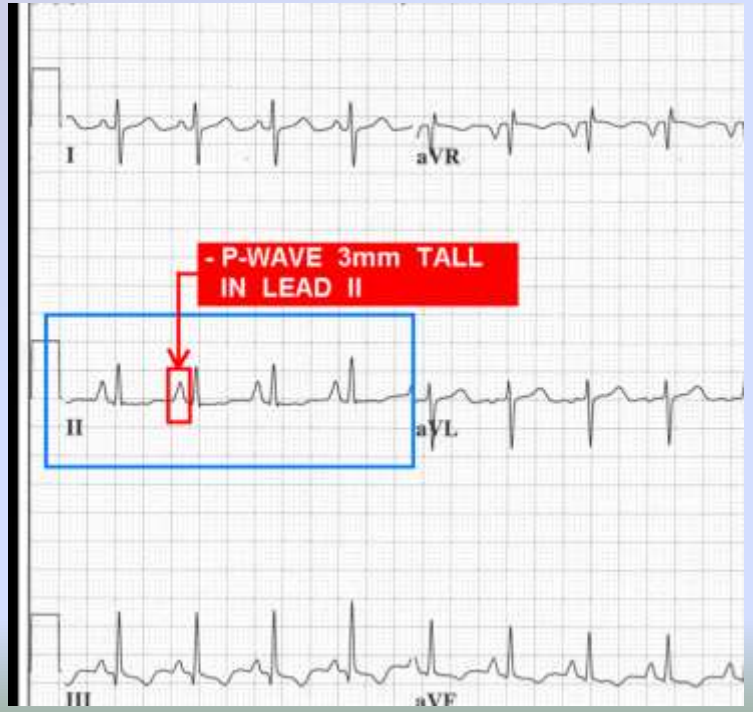
(SPECIFIC CRITERIA FOR ATRIAL HYPERTROPHY IS DISCUSSED IN MORE DETAIL IN THE "CHAMBER HYPERTROPHY" SECTION)

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 it “too tall (>2mm) or too long (>2.5mm)” in Lead II, ***then go to Lead V1*** to evaluate P wave . . . .



THE P WAVE

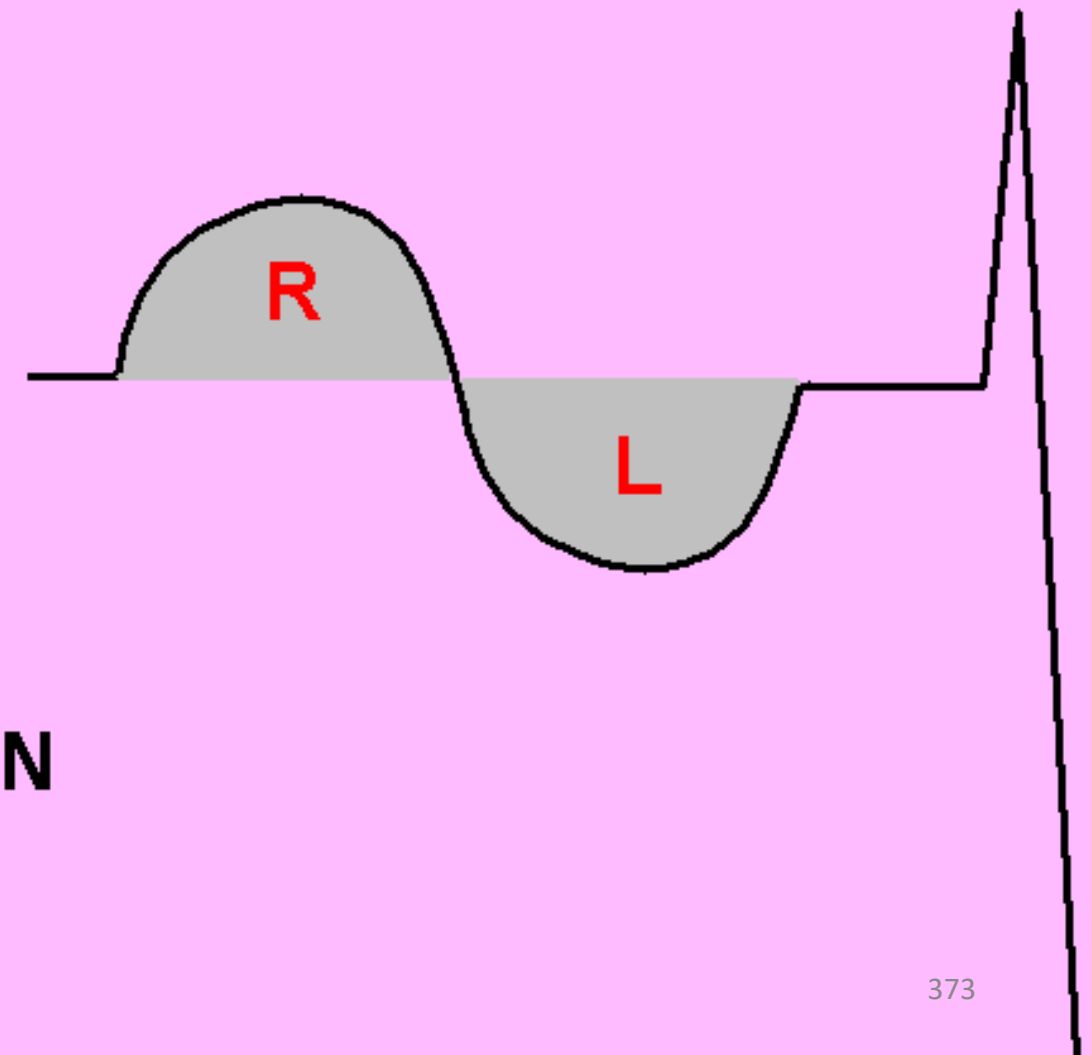
IN LEAD V1 MAY BE:

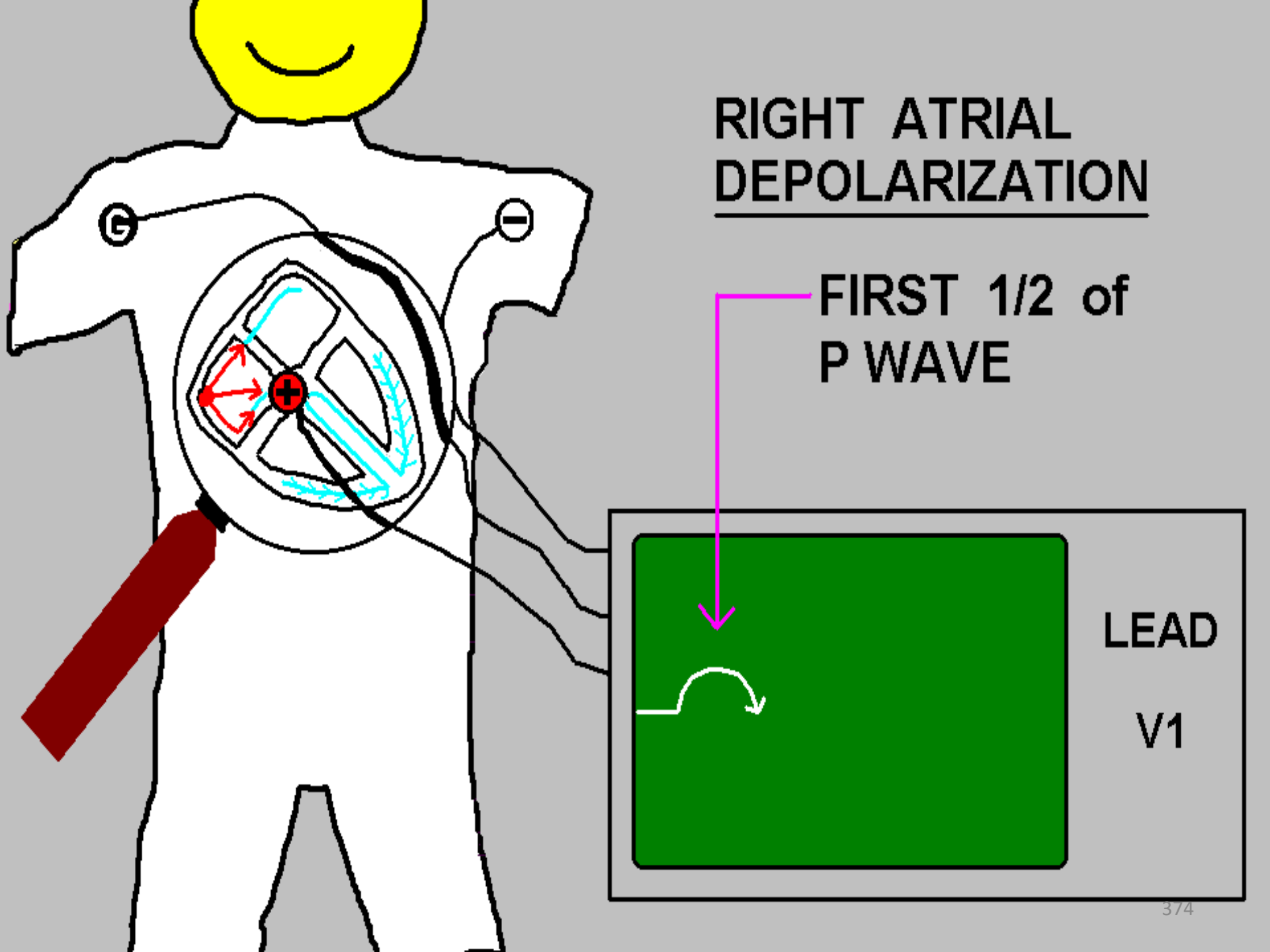
- POSITIVE

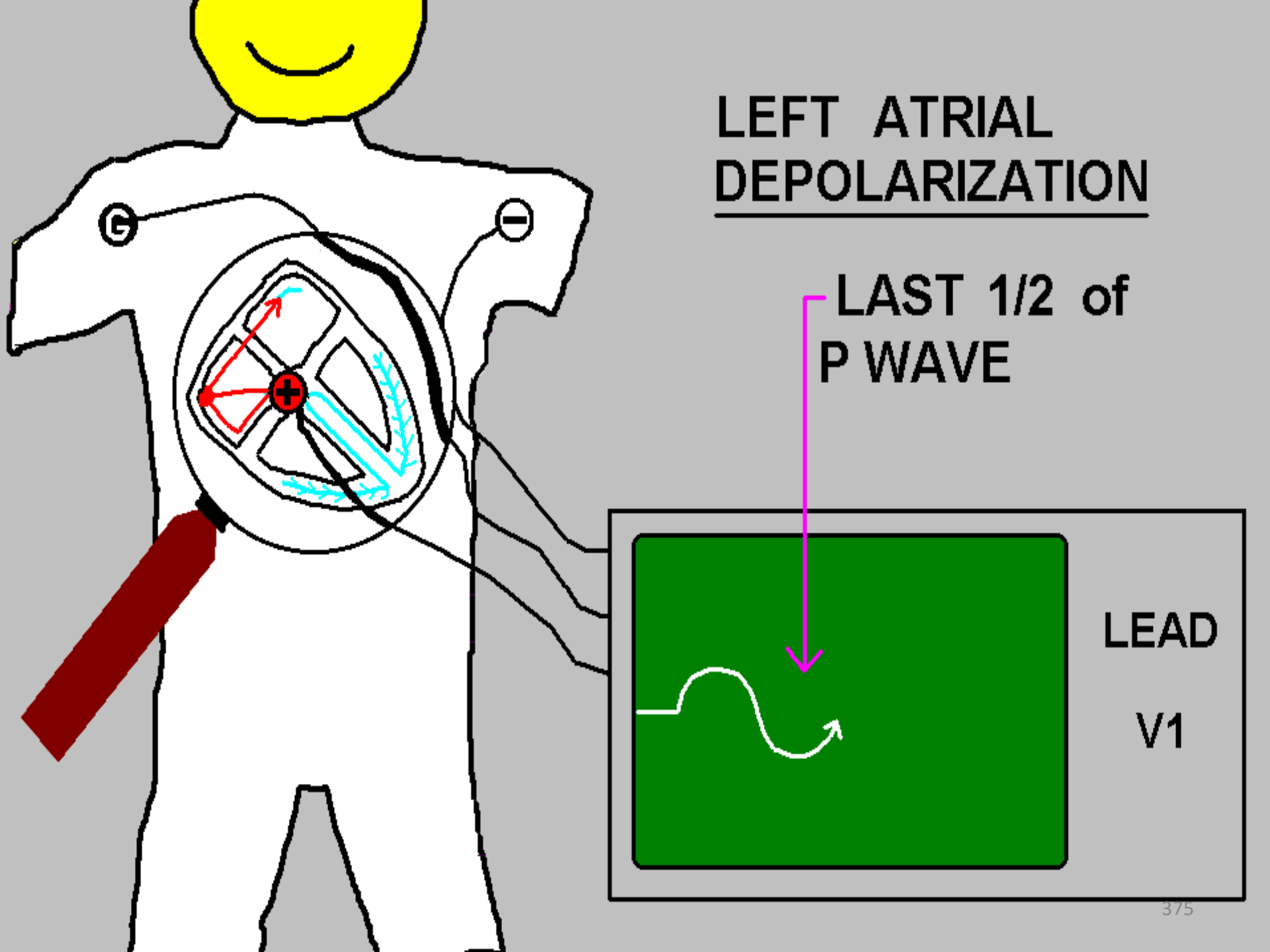
- OR BI-PHASIC

THE P WAVE

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



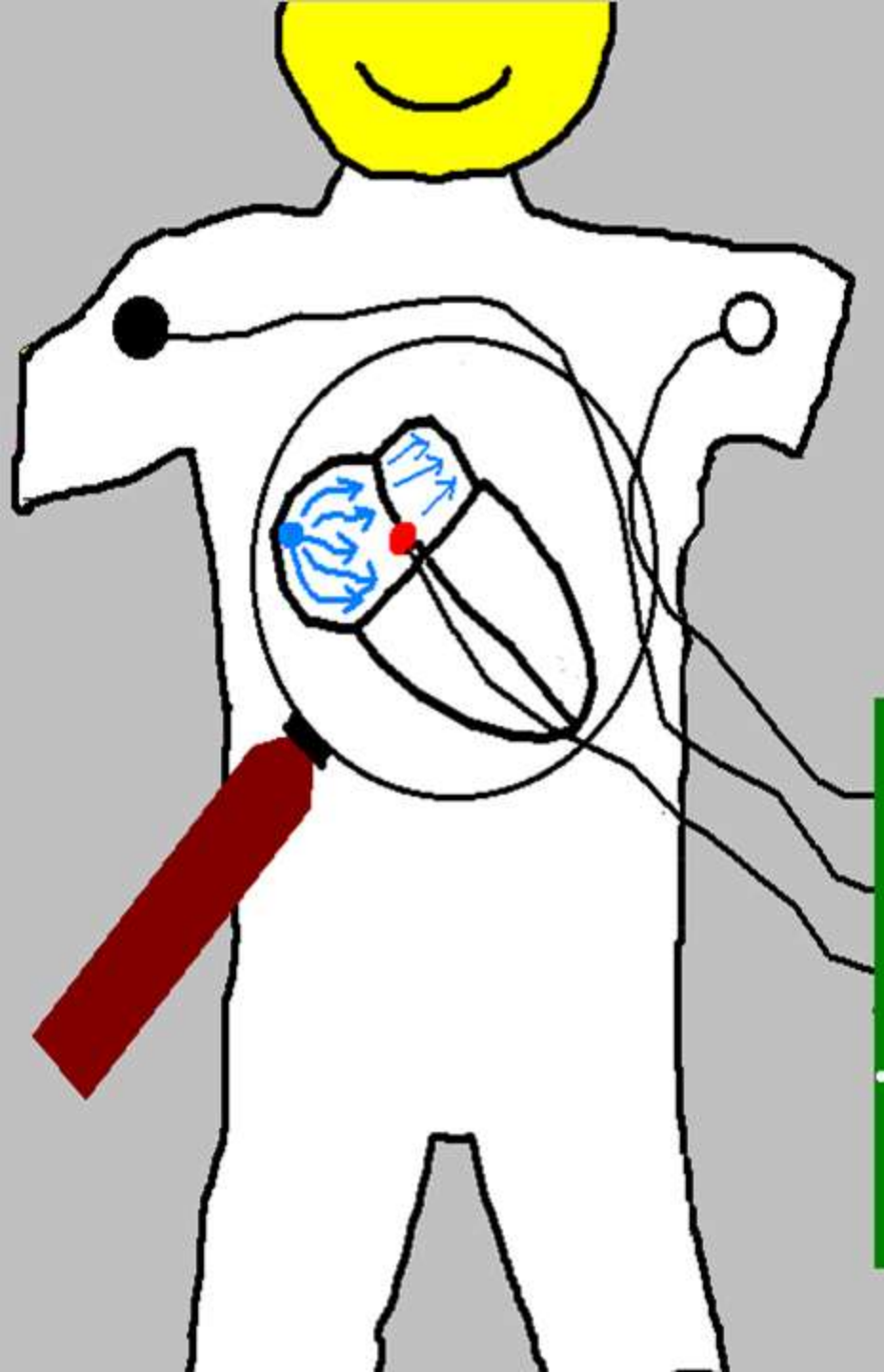

RIGHT ATRIAL
DEPOLARIZATION
FIRST 1/2 of
P WAVE
LEAD
V1


LEFT ATRIAL
DEPOLARIZATION
LAST 1/2 of
P WAVE
LEAD
V1

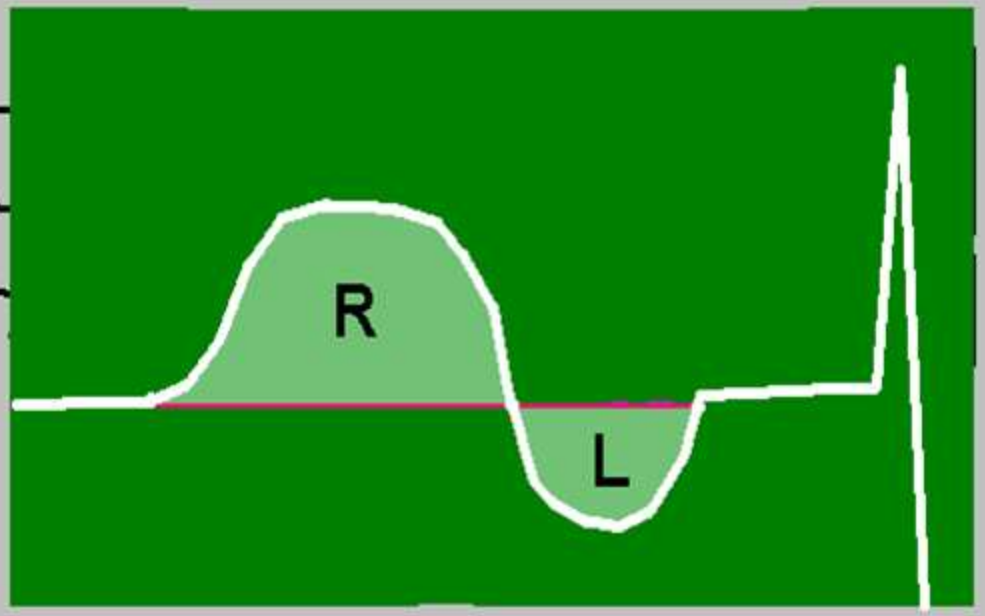
Evaluate P Wave for Atrial Hypertrophy

- Evaluate amplitude and duration in Lead II
- If the P wave it “too tall (>2mm) or too long (>2.5mm)” 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

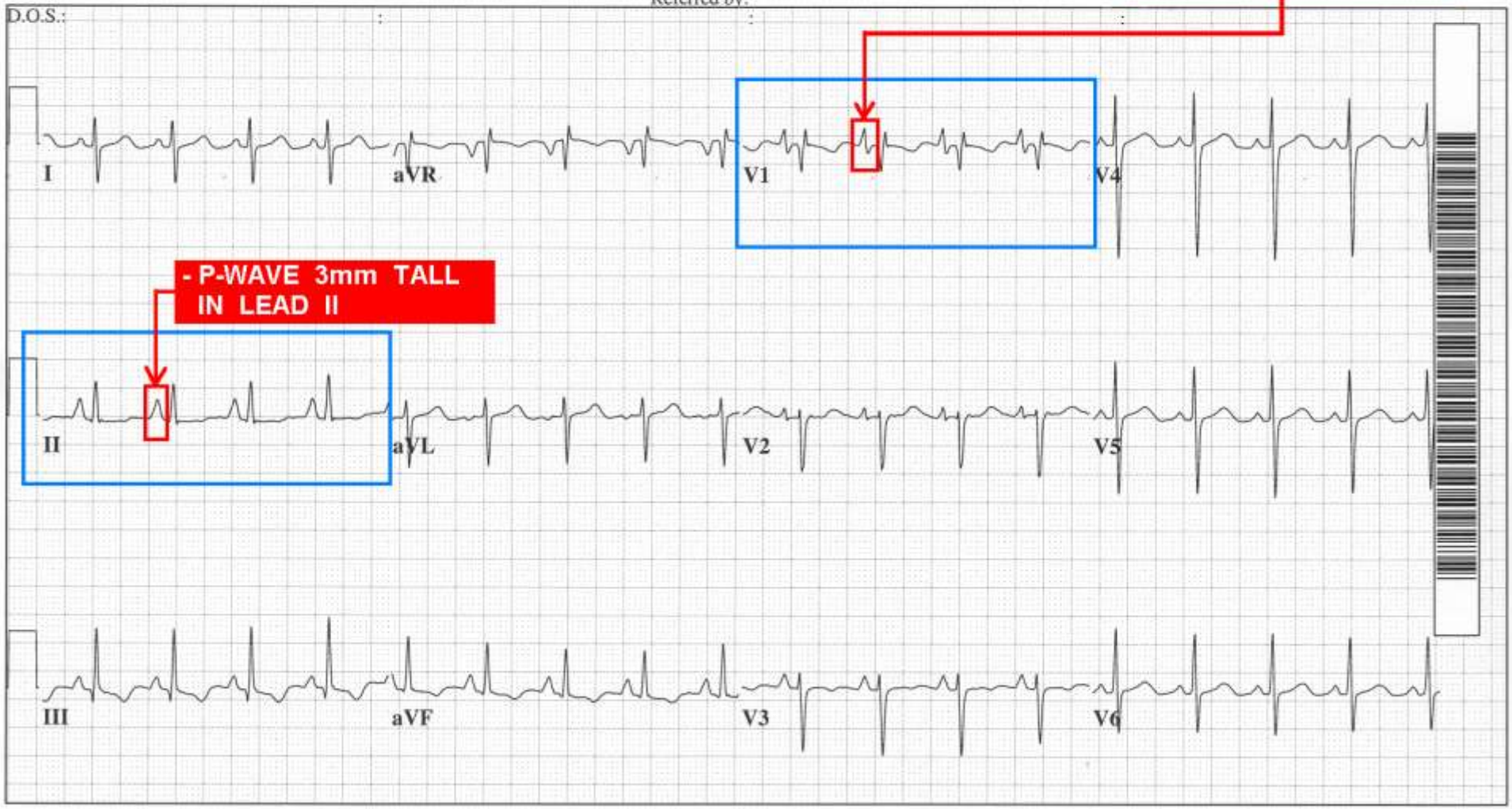
Sinus tachycardia
Right atrial enlargement
Rightward axis
Pulmonary disease pattern
RSR' or QR pattern in V1 suggests right ventricular
T wave abnormality, consider inferior ischemia
Abnormal ECG
When compared with ECG of 01-OCT-1998 21:45
T wave inversion more evident in Inferior leads ...

Referred by:

EKG CLASS #WR03446043

- POSITIVE DEFLECTION TALLER (more dominant) IN LEAD V1

- P-WAVE 3mm TALL IN LEAD II



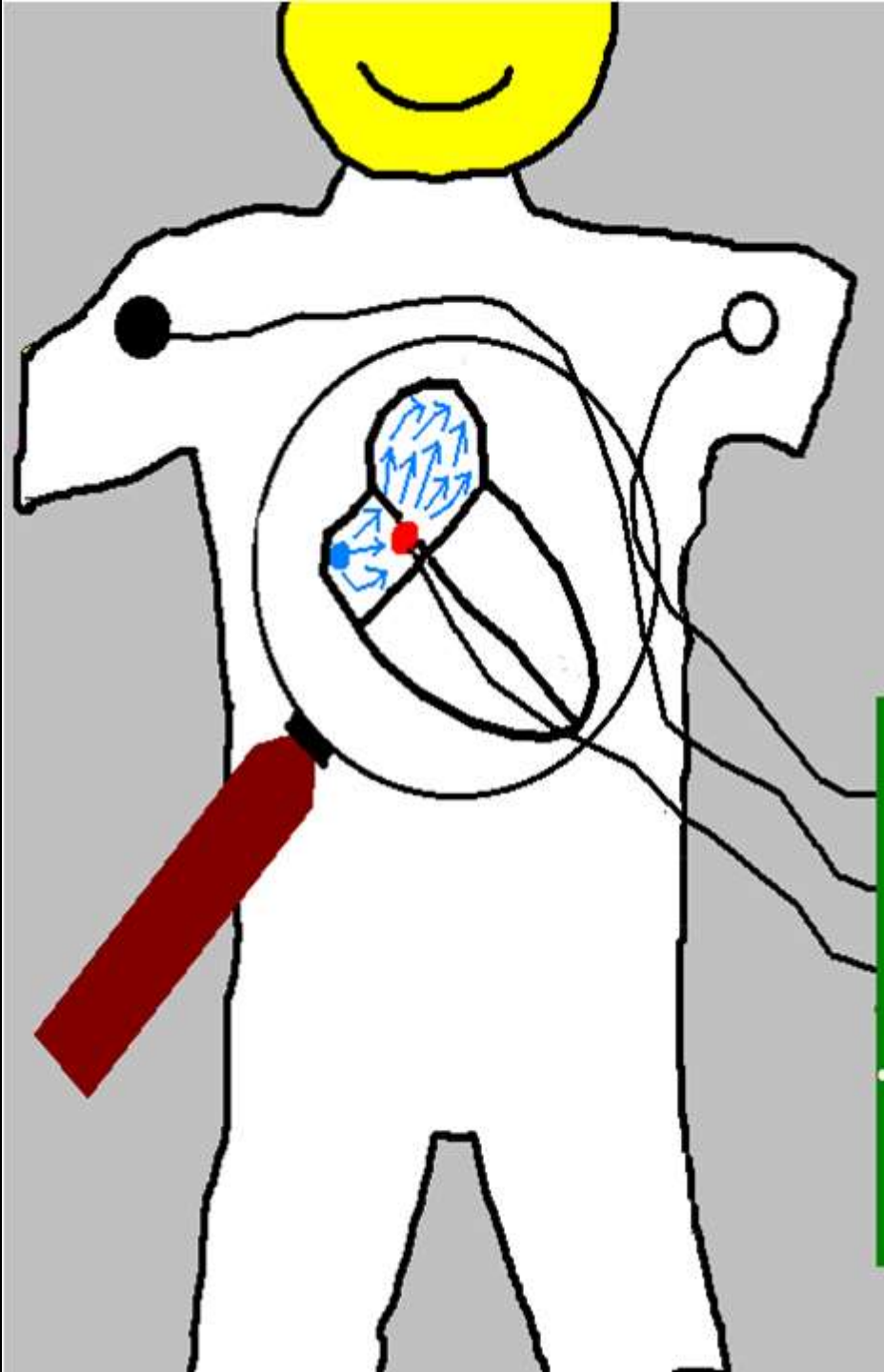
25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 13

EID:17 EDT: 07:28 02-DEC-1998 ORDER:

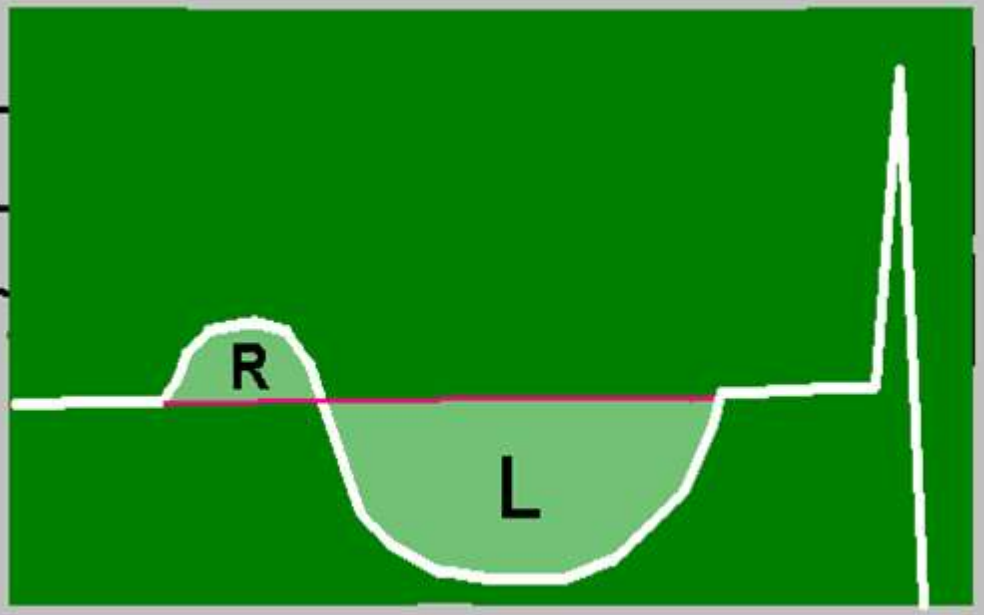
Evaluate P Wave for Atrial Hypertrophy

- Evaluate amplitude and duration in Lead II
- If the P wave it “too tall (>2mm) or too long (>2.5mm)” 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



01–MAY–1998 03:09:15 ST. JOSEPH'S HOSPITAL–ER ROUTINE RETRIEVAL

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

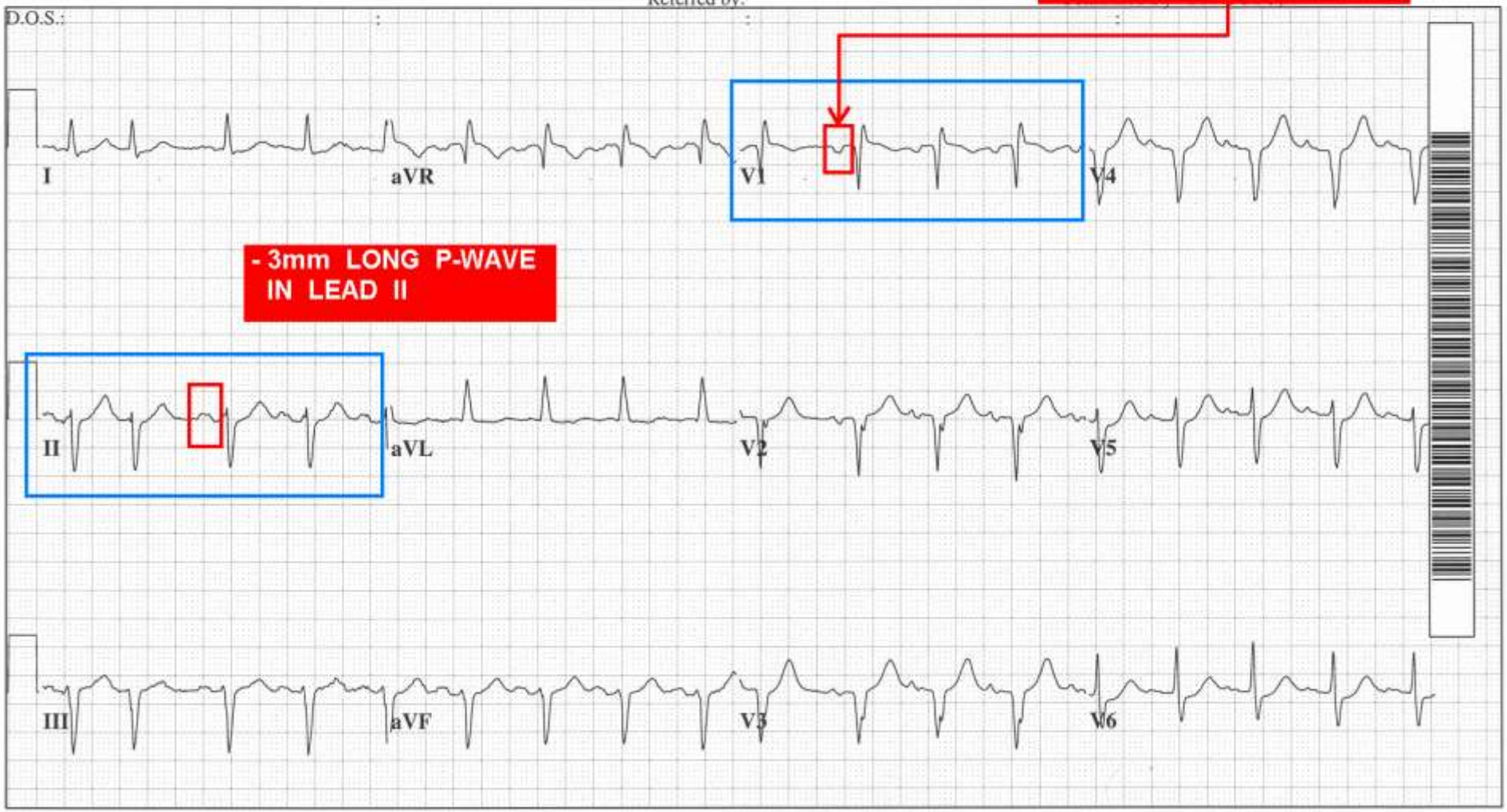
Sinus tachycardia with occasional Premature supraventricular complexes
Left atrial enlargement
Left axis deviation
Incomplete right bundle branch block
Anteroseptal infarct , age undetermined
Abnormal ECG
No previous ECGs available

EKG CLASS #WR03651849

- NEGATIVE DEFLECTION P-WAVE IN LEAD V1

Referred by:

D.O.S.:



- 3mm LONG P-WAVE IN LEAD II

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

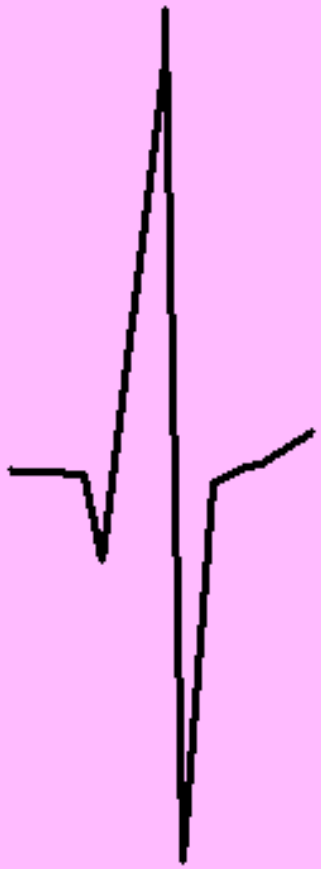
EID:17 EDT: 14:12 02–MAY–1998 ORDER:

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

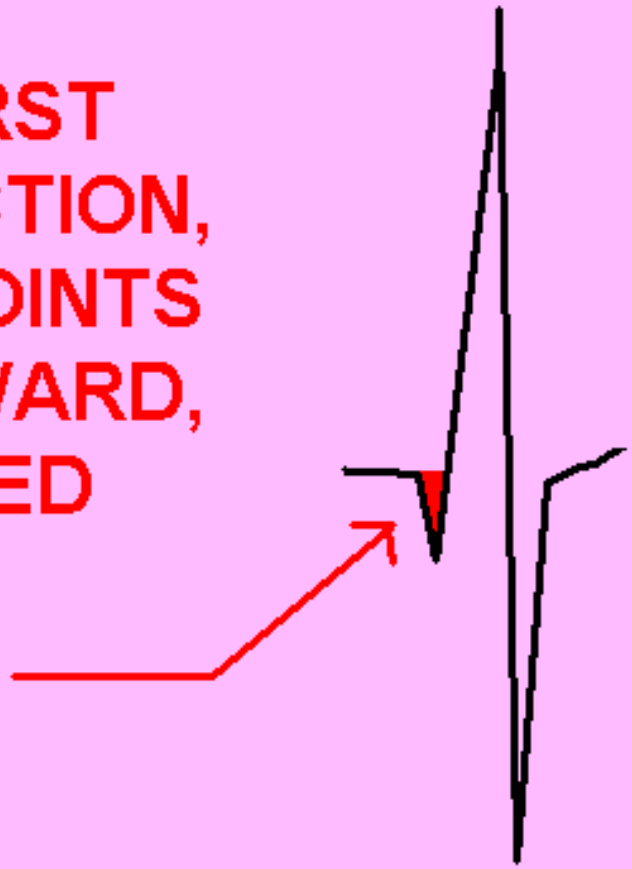
THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .



THE QRS COMPLEX

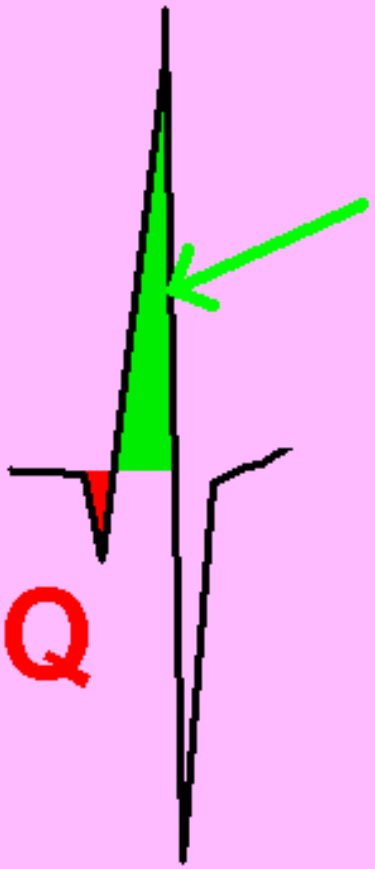
THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .

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



THE QRS COMPLEX

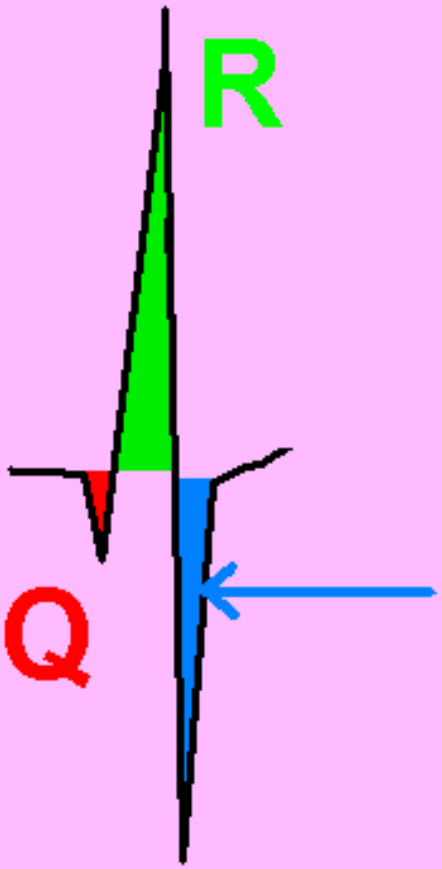
THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .



THE FIRST POSITIVE DEFECTION IS KNOW AS THE "R" WAVE

THE QRS COMPLEX

THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .

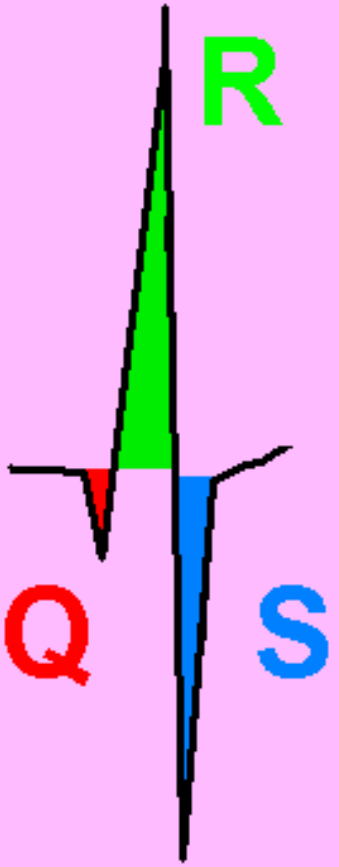


A NEGATIVE DEFLECTION AFTER THE R WAVE IS CALLED THE " S " WAVE

THE QRS COMPLEX

THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .

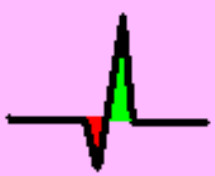
AND IS THE ONLY TRUE "QRS" COMPLEX



SOME OF THE OTHER VARIATIONS INCLUDE . . . .

THE QRS COMPLEX

WHAT ARE THESE COMPLEXES ??



qR

RS

R

Rsr'

Qr

QS

qRSr'

qR

rS

Rr'

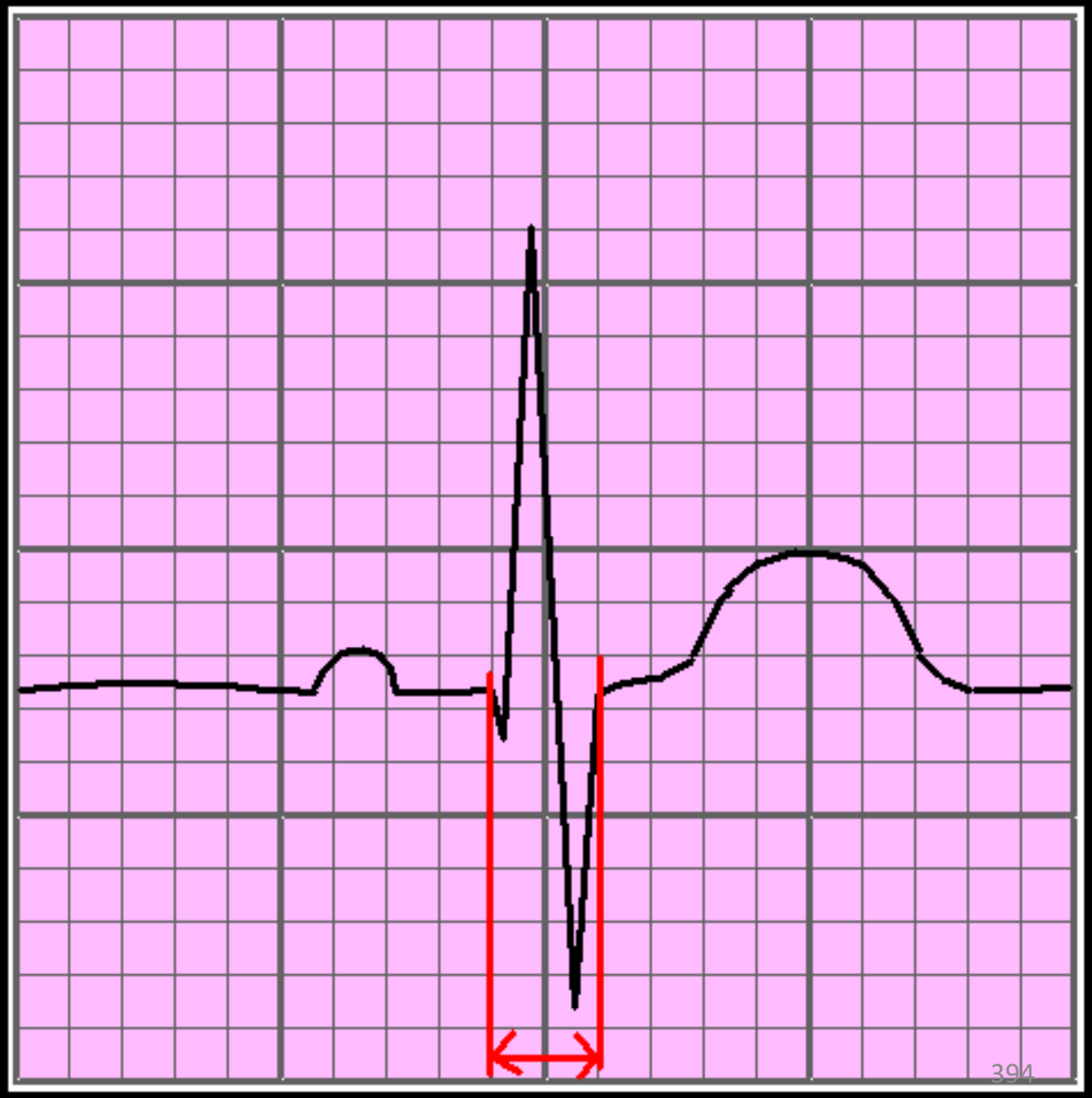
QRS INTERVAL

LESS THAN

.12

OR

120 mSEC



QRS COMPLEX TOO WIDE

WIDER THAN 120 mSEC

THINK:

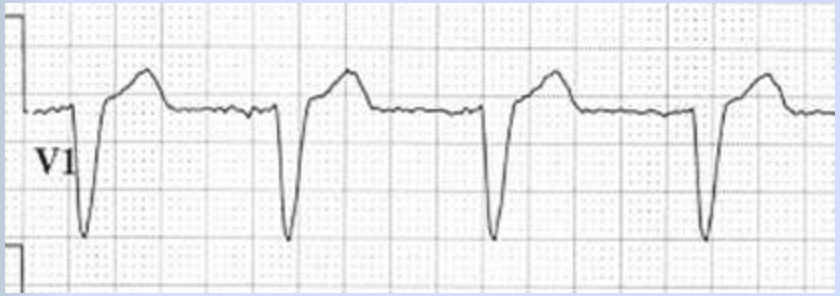
- BUNDLE BRANCH BLOCK
- VENTRICULAR COMPEX (ES)
- PACED RHYTHM
- L VENTRICULAR HYPERTROPHY
- ELECTROLYTE IMBAL. ($\uparrow K^+$ $\downarrow Ca^{++}$)
- DELTA WAVE (PRE-EXCITATION)

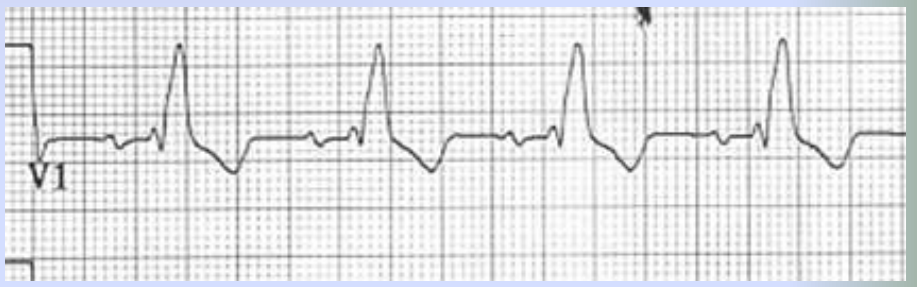
When the QRS is WIDE (> 3mm):

- **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**

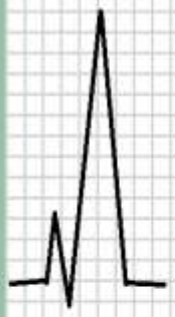




Simple “Turn Signal Method” . . .

THE "TURN SIGNAL METHOD" for identifying BUNDLE BRANCH BLOCK

V1



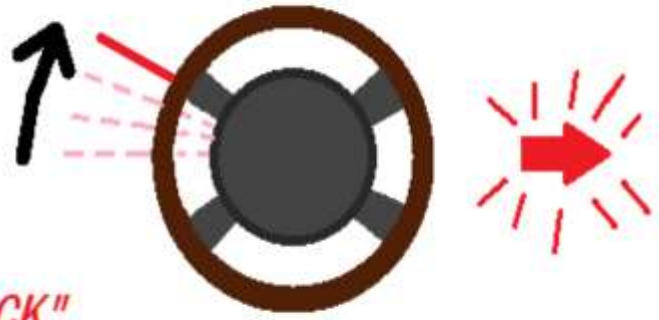
USE LEAD V1 for this technique

To make a **RIGHT TURN**

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

THINK:

"QRS points UP = RIGHT BUNDLE BRANCH BLOCK"



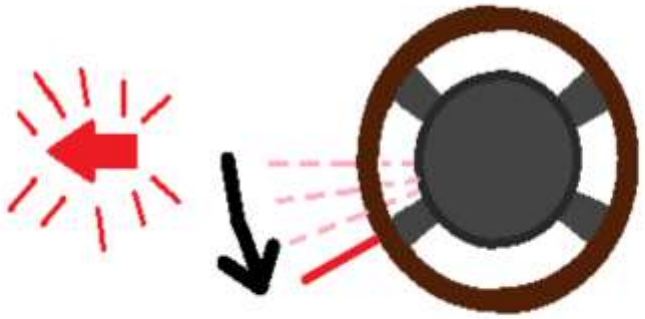
V1

To make a **LEFT TURN**

you push the turn signal lever **DOWN** . . . .

THINK:

"QRS points DOWN = LEFT BUNDLE BRANCH BLOCK"



RUPPERT, WAYNE ID: 099999999 4-Mar-2001

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

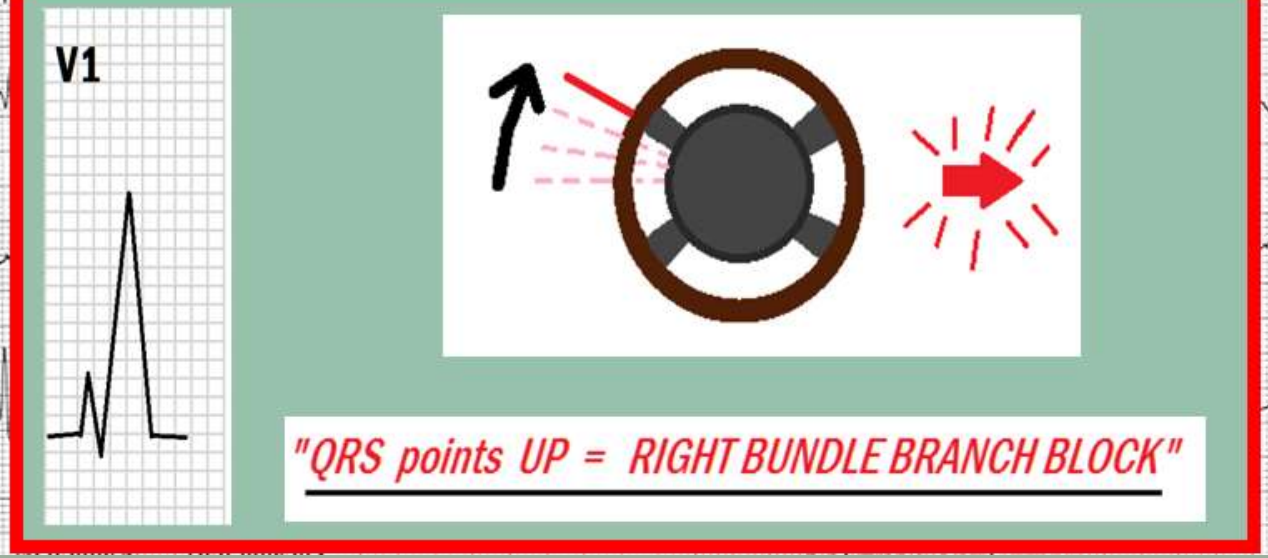
Vent. rate	72	bpm
PR interval	186	ms
QRS duration	166	ms
QT/QTc	436/477	ms
P-R-T axes	57 -32 32	

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.:

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



40 Hz MACVU 003C 12SL^{tm} v250

09:16:40

74 yr
Female Caucasian

Loc:7 Option:35

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

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

EKG #WD03029959

Technician: WW

USE LEAD V1 for this technique

Referred by:

Confirmed By:

D.O.S.:

I aVR V1 V4

II aVL V2 V5

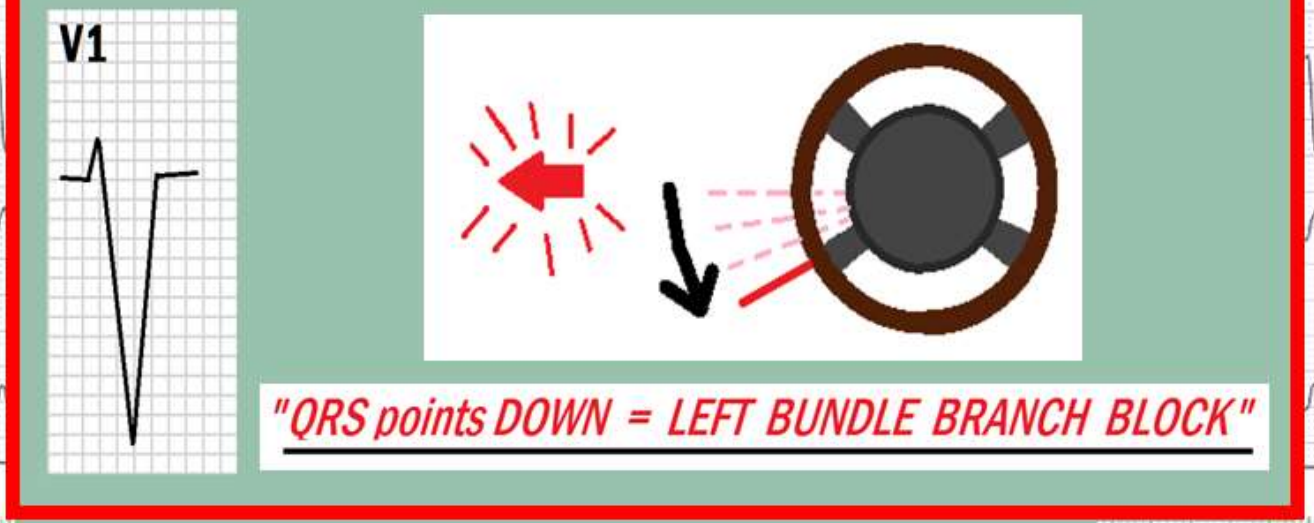
III aVF V3 V6

V1

II

V5

25mm/s 10mm/mV



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

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

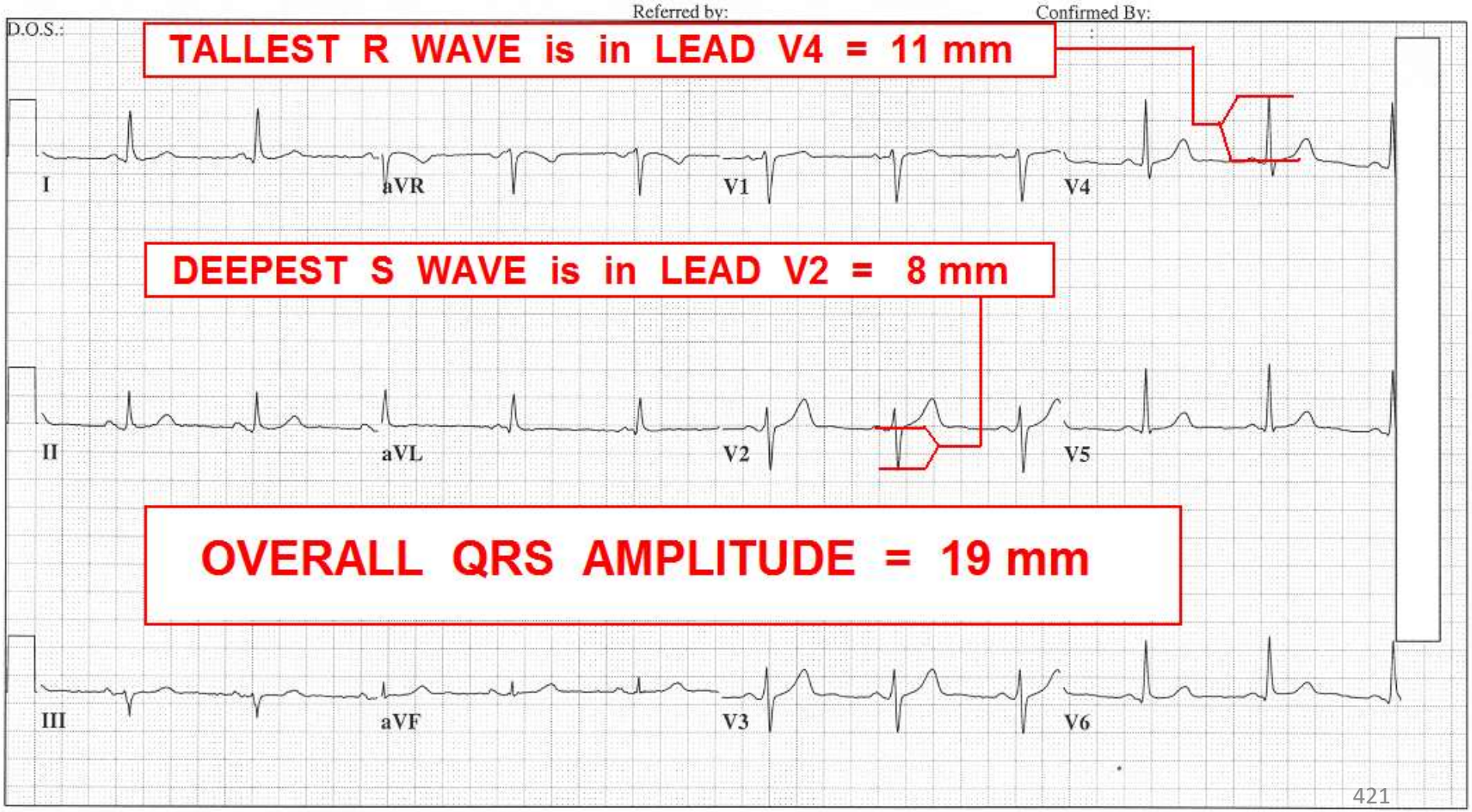
THE QRS COMPLEX

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 . . . .



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

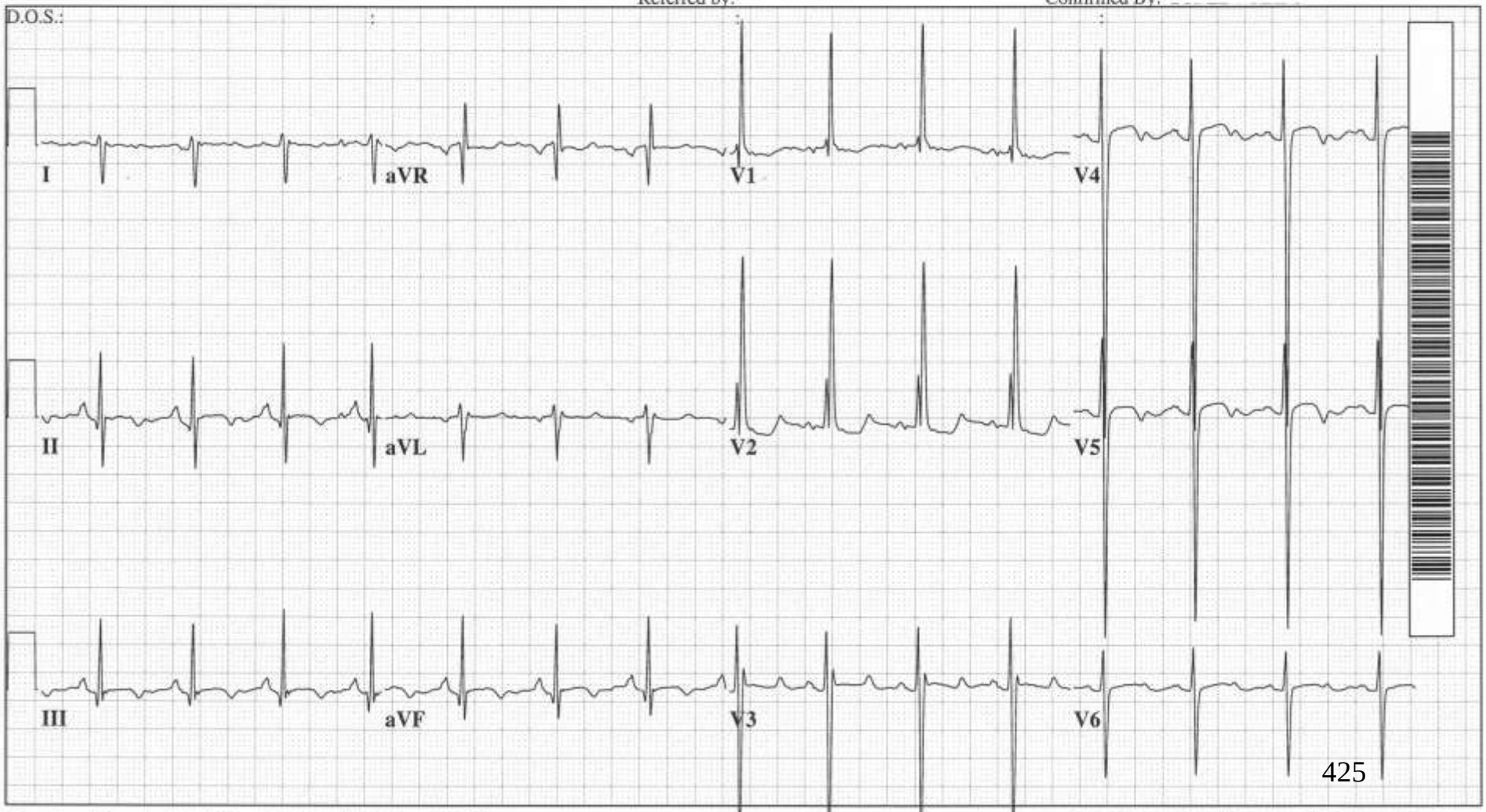
Vent. rate	90	BPM
PR interval	136	ms
QRS duration	94	ms
QT/QTc	378/462	ms
P–R–T axes	77 123	58

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:

D.O.S.:



425

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

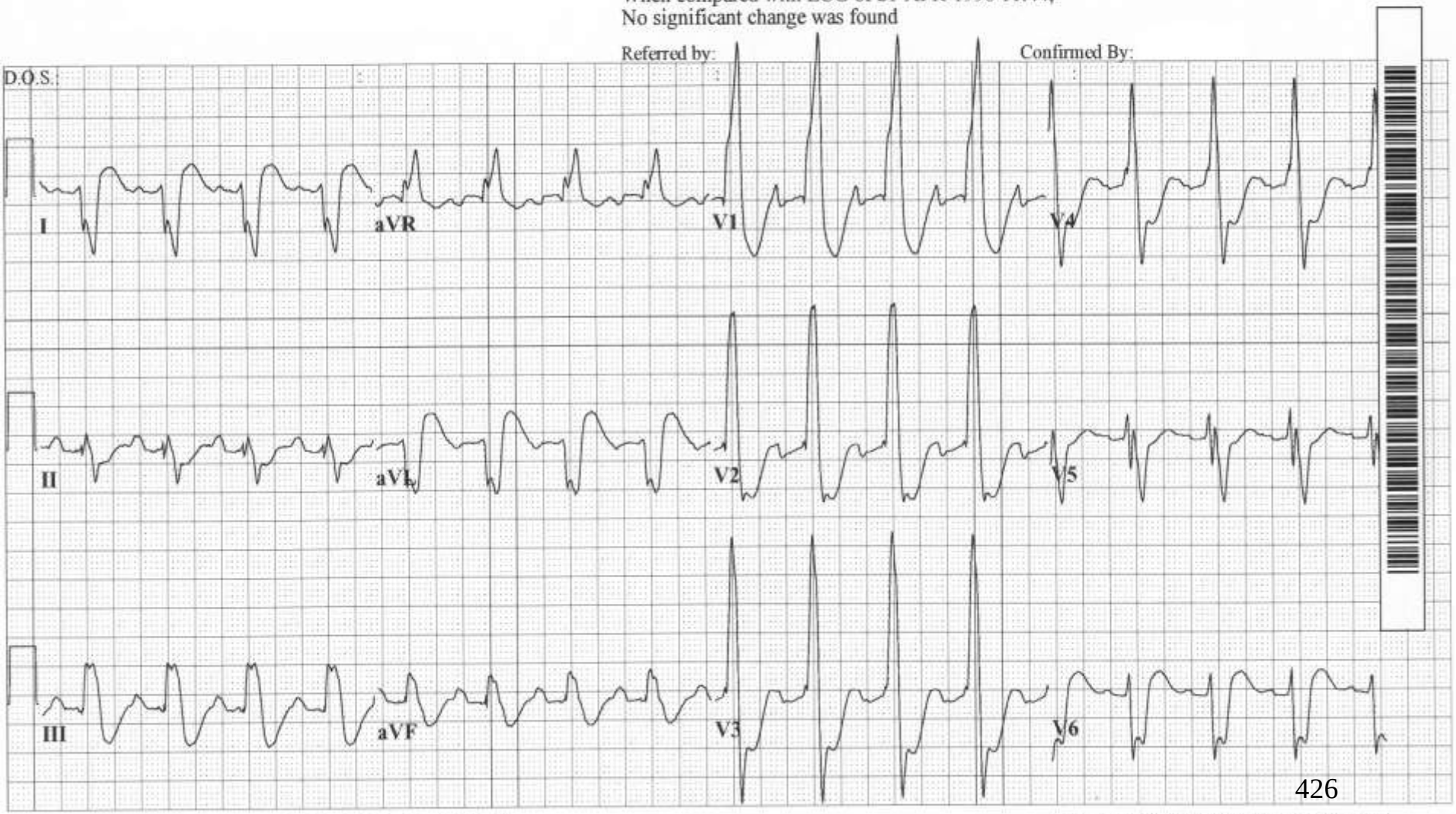
Vent. rate	100	BPM
PR interval	198	ms
QRS duration	186	ms
QT/QTc	380/490	ms
P-R-T axes	79 163 -20	

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.:



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

EID:13 EDT: 23:01 20-JUN-1997 ORDER:

18–NOV–2002 02:15:46 ST. JOSEPH'S HOSPITAL–ER 1ST PREVIOUS

53 yr
Male Black

Room:ER
Loc:3 Option:23

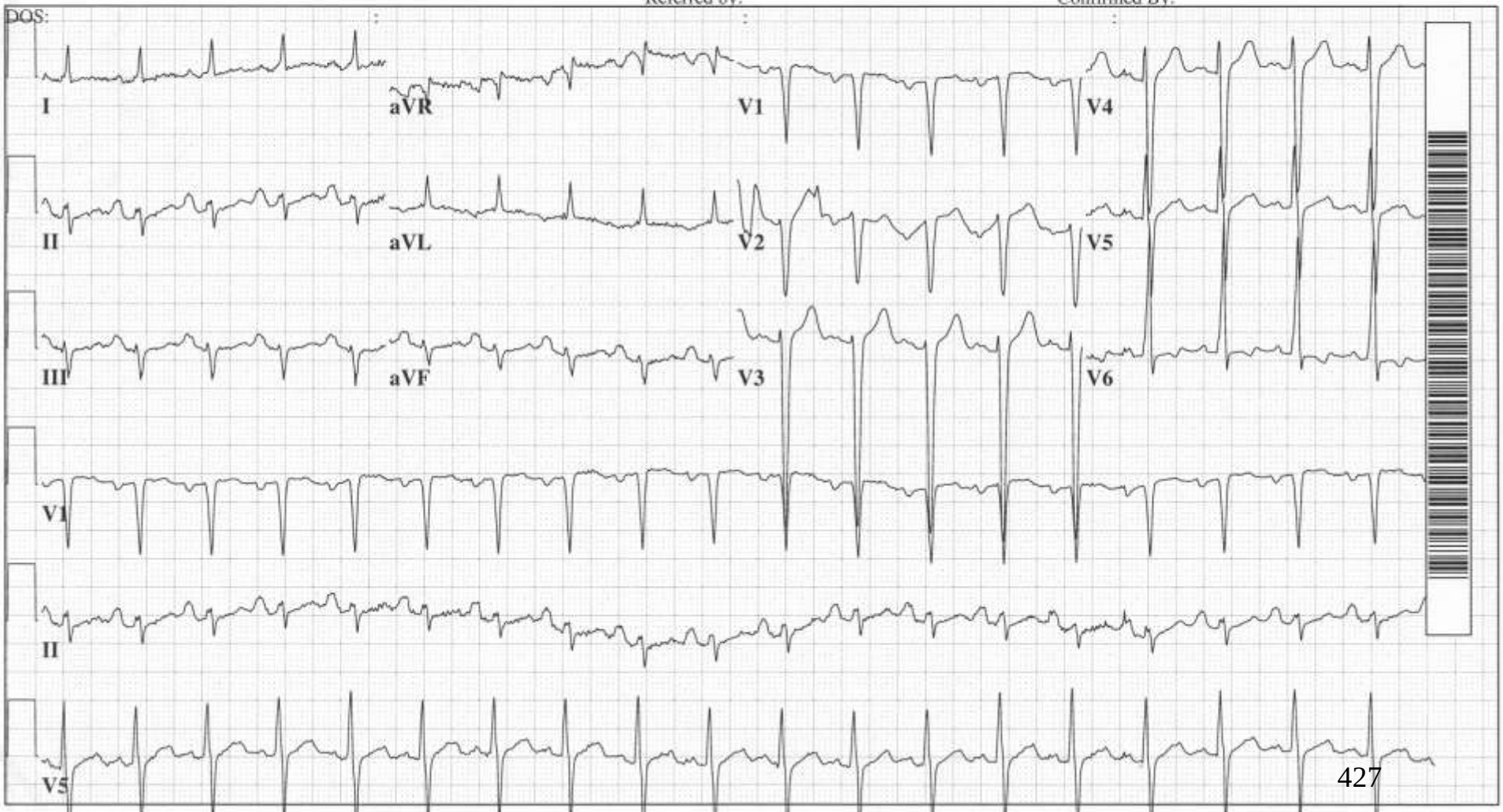
Vent. rate	115	BPM
PR interval	160	ms
QRS duration	92	ms
QT/QTc	316/437	ms
P–R–T axes	76 –39	59

**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:



25mm/s 10mm/mV 40Hz 005C 12SL 233 CID: 7

EID:28 EDT: 11:27 18–NOV–2002 ORDER:

10–APR–2003 11:49:36 ST. JOSEPH'S HOSPITAL–CCR ROUTINE RETRIEVAL

61 yr
Male Black

Loc:7 Option:35

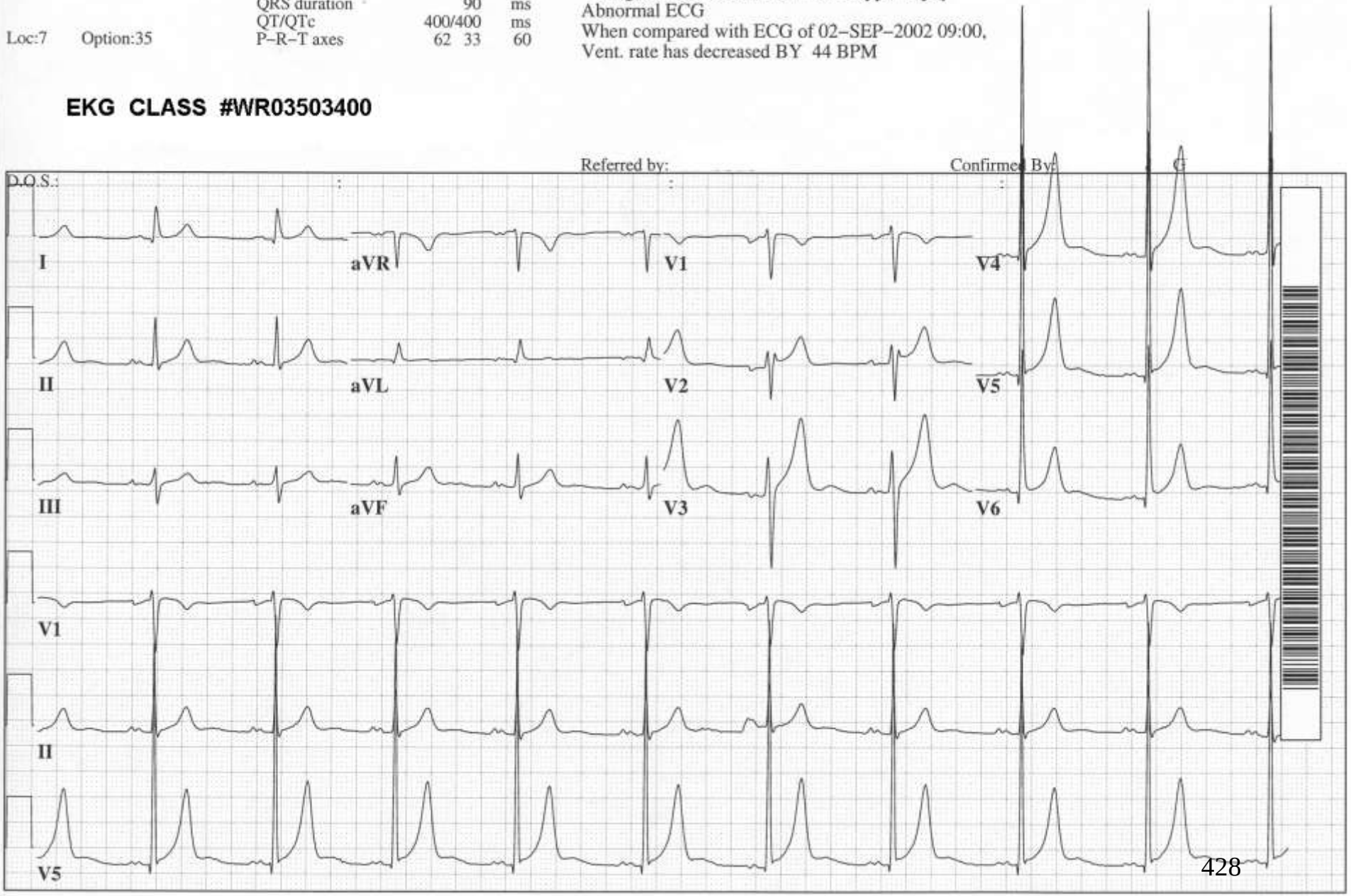
Vent. rate	60	BPM
PR interval	176	ms
QRS duration	90	ms
QT/QTc	400/400	ms
P–R–T axes	62 33	60

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:

D.O.S.:



25mm/s 10mm/mV 40Hz 005C 12SL 229 CID: 0

EID:28 EDT: 10:43 14–APR–2003 ORDER:

THE QRS COMPLEX

QRS AMPLITUDE

CRITERIA FOR MINIMUM AMPLITUDE:

Abnormally LOW QRS VOLTAGE occurs when the OVERALL QRS is:

≤ 0.5 mV IN ANY LIMB LEAD

— *and* —

≤ 1.0 mV IN ANY PRECORDIAL LEAD

OVERALL QRS AMPLITUDE TOO LOW:

(VERTICAL QRS SIZE)

THINK (in absence of obvious OBESITY) :

MYOCARDITIS / CONSTRICTIVE PERICARDITIS

EFFUSIONS / TAMPONADE

COPD c HYPERINFLATION

AMYLOIDOSIS (abnormal protein accumulation in organs)

SCLERODERMA (abnormal hardening of skin)

HEMACHROMOTOSIS (excessive iron buildup in blood / organs)

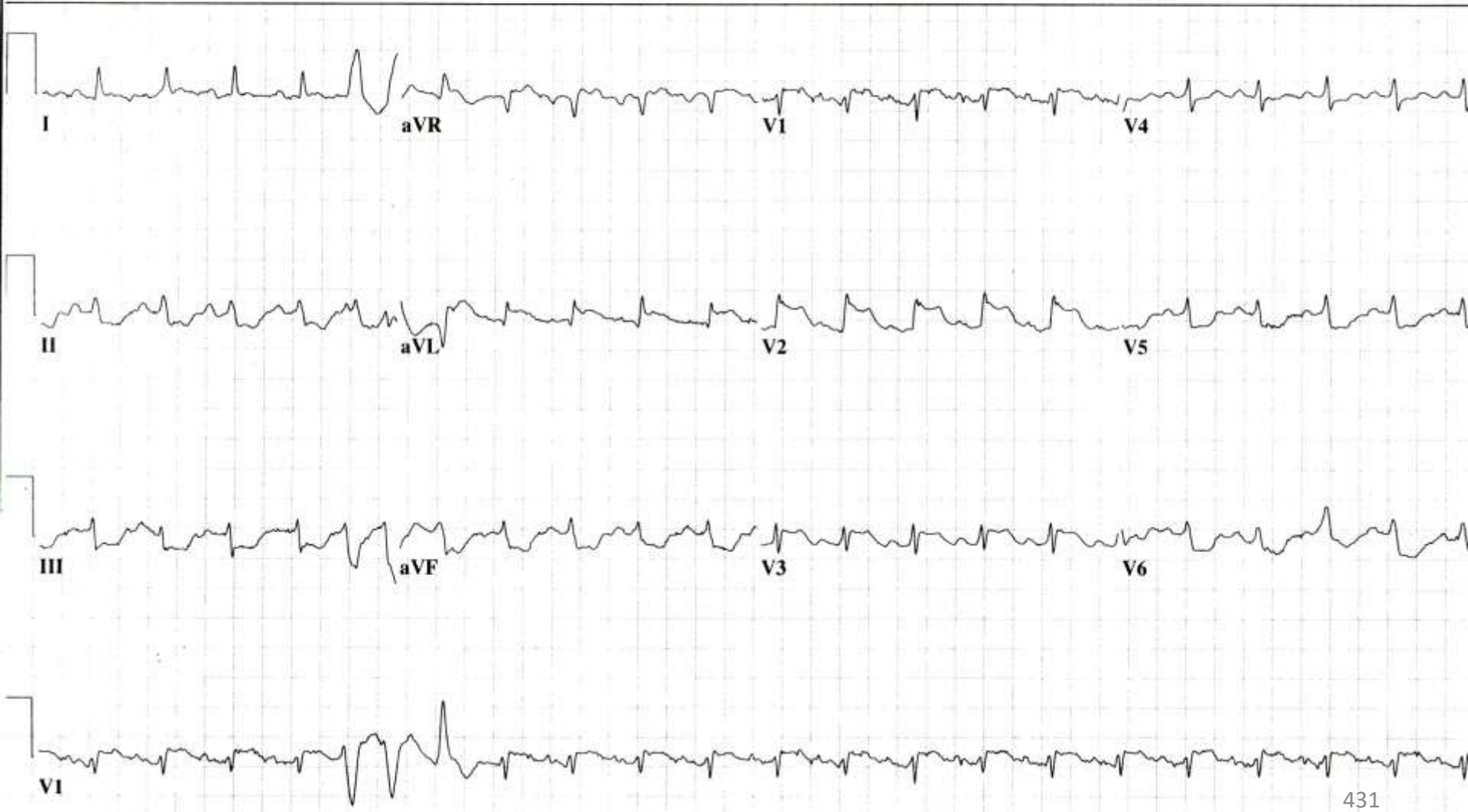
MYXEDEMA (thyroid disorder)

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
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ST elevation consider anterolateral injury or acute infarct
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Abnormal ECG
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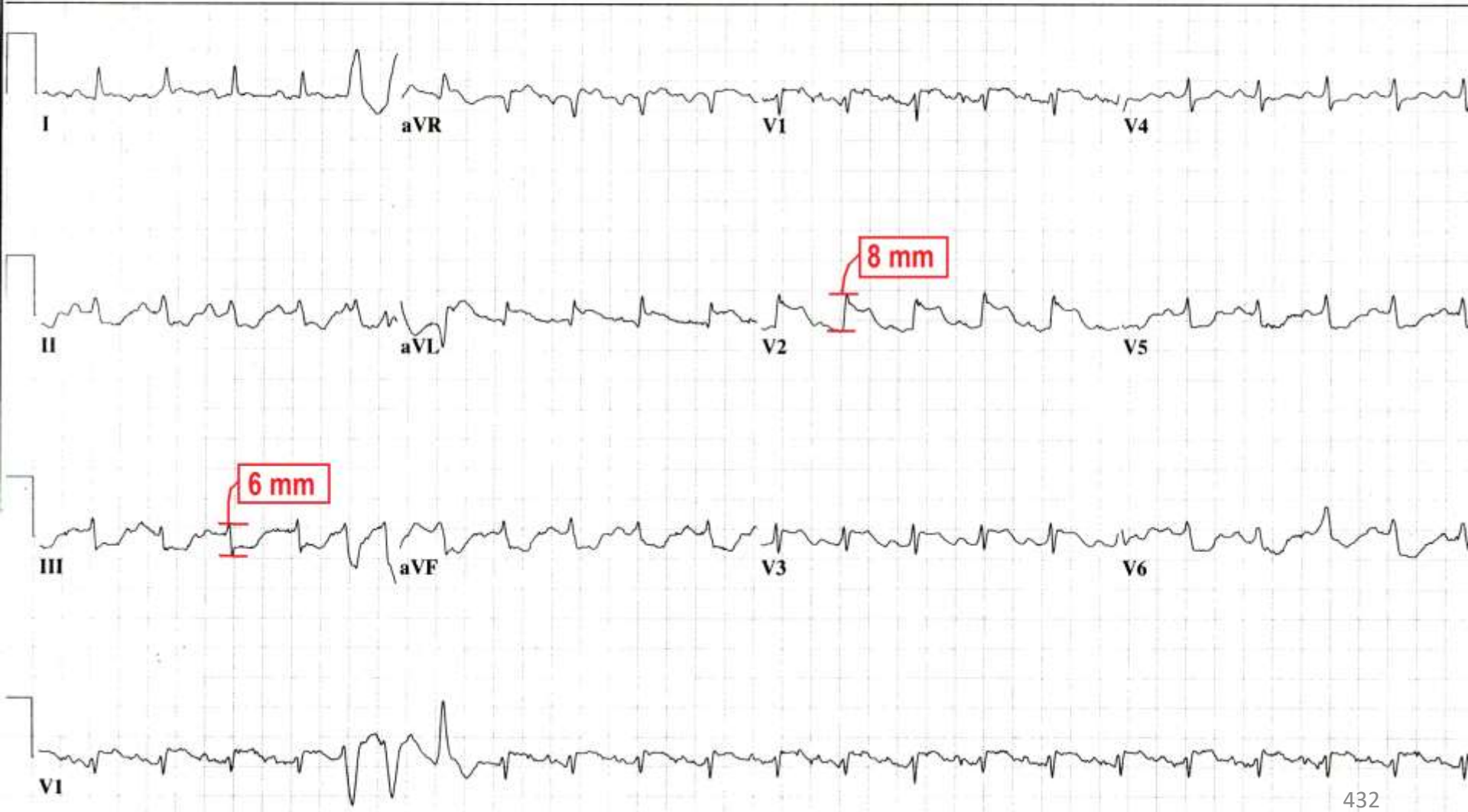


• Q WAVES •

Normal Q Waves

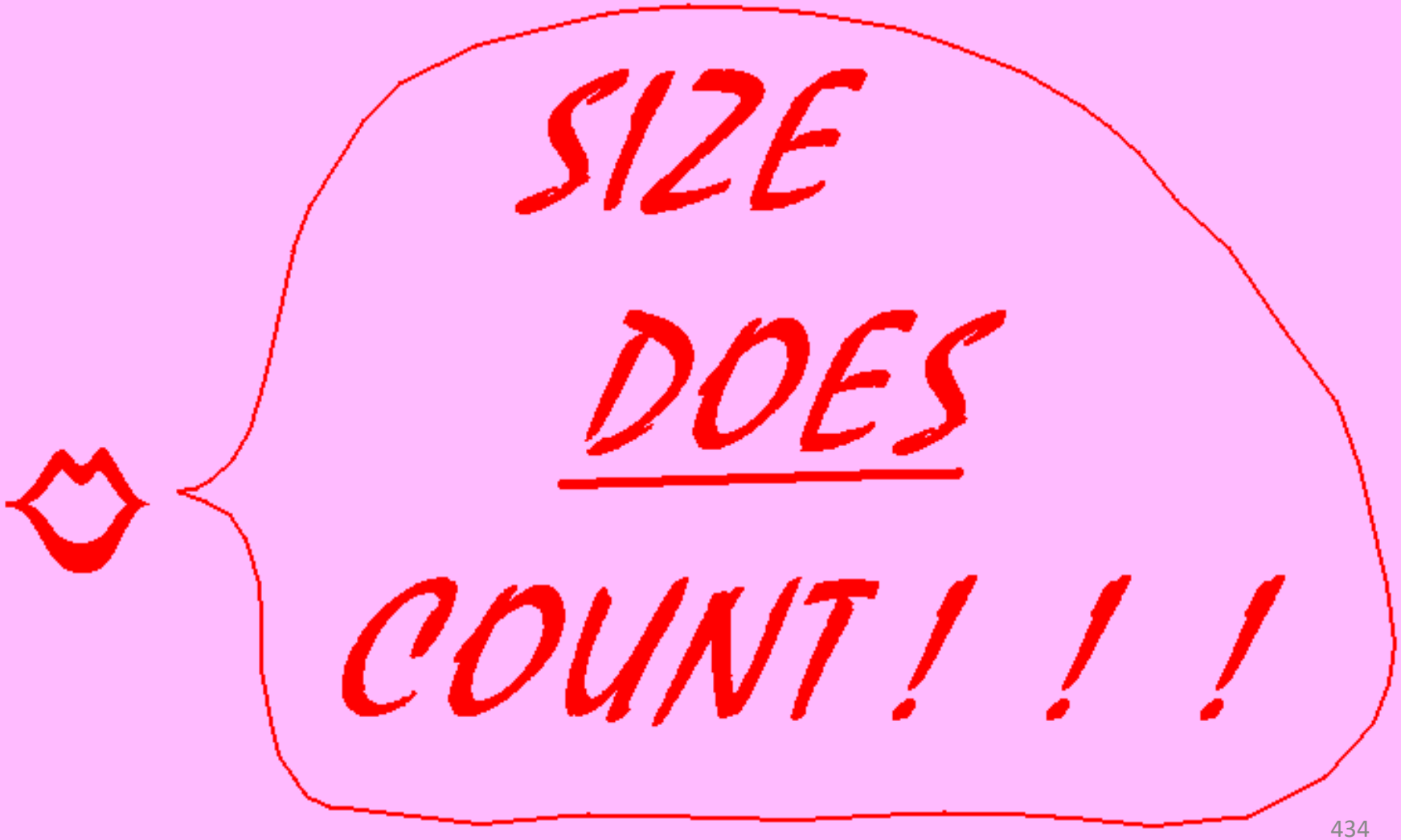
caused by depolarization of the intraventricular septum

Abnormal Q Waves -

caused by:

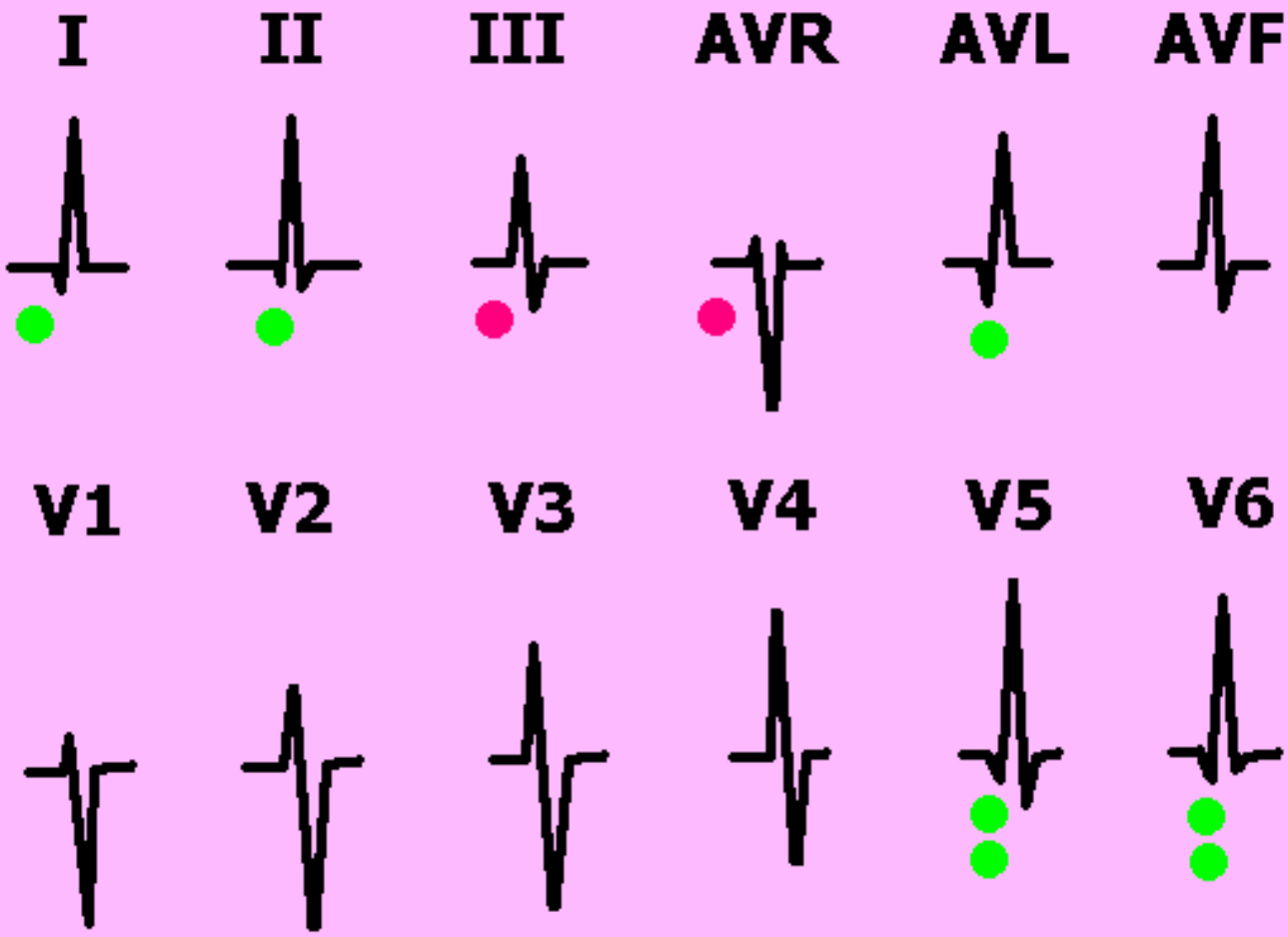
- necrosis (old infarction)
- hypertrophy

• Q WAVES •



LEADS WHERE Q WAVES ARE NORMAL

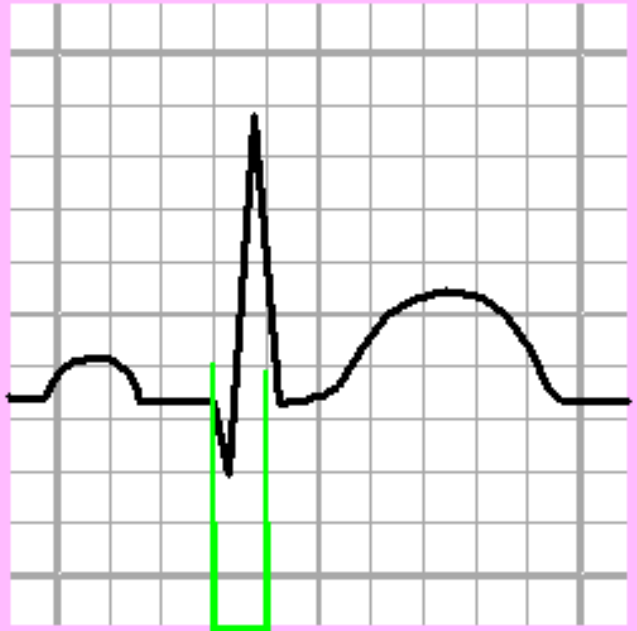
- Normal Q WAVES caused by SEPTAL DEPOLARIZATION



Q WAVES NORMAL AND FREQUENTLY SEEN	Q WAVES EXPECTED	Q WAVES, IF PRESENT, CAN NORMALLY BE ANY SIZE

THE QRS COMPLEX

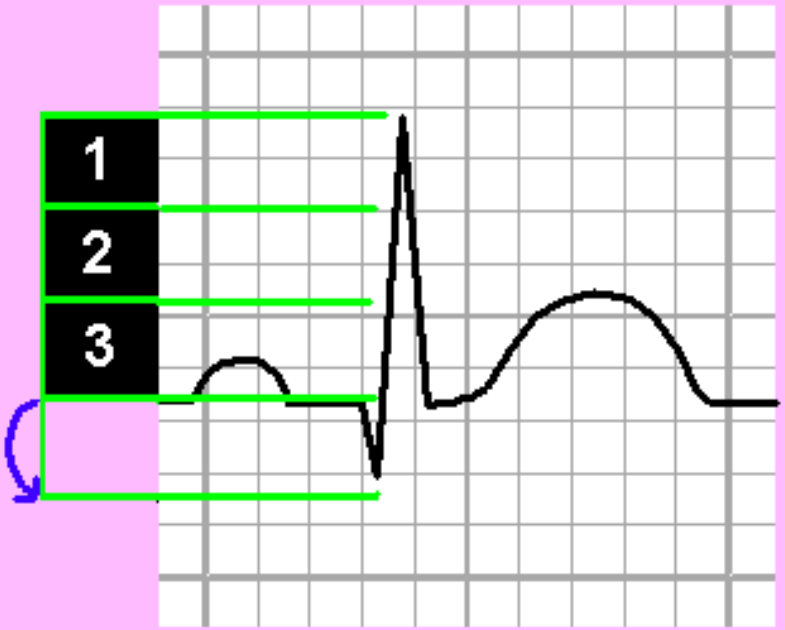
GENERAL RULES FOR NORMAL Q WAVES - WIDTH



LESS THAN .40 (1 mm) WIDE

THE QRS COMPLEX

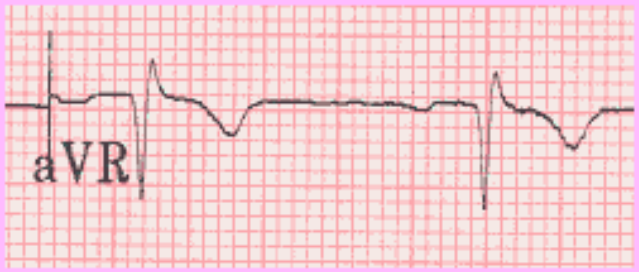
GENERAL RULES FOR NORMAL Q WAVES - HEIGHT



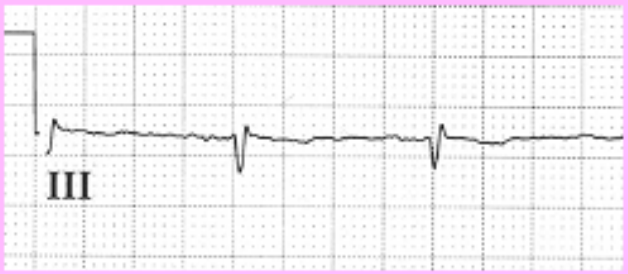
☞ LESS THAN 1/3 THE HEIGHT OF THE R WAVE

THE QRS COMPLEX

NORMAL Q WAVES
EXCEPTIONS TO THE RULES



LEAD AVR



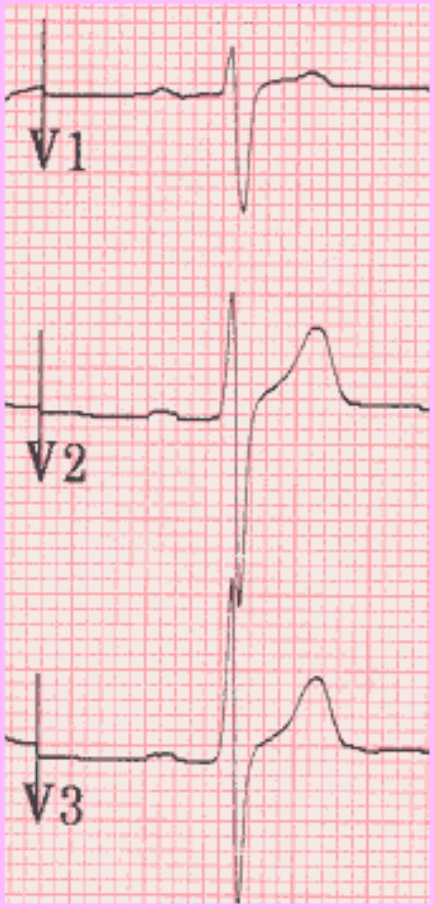
LEAD Ⅲ

☞ 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:

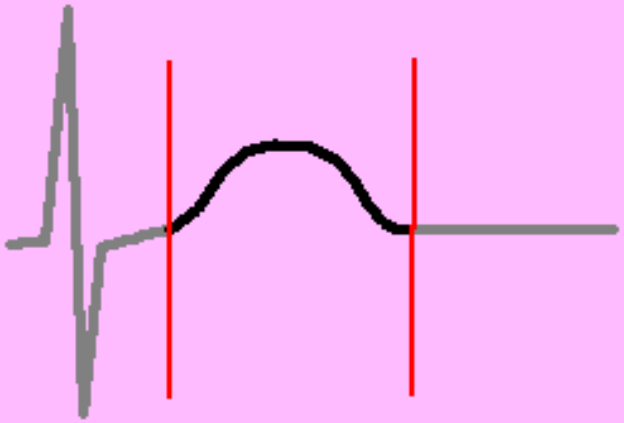
- Q WAVES SHOULD BE LESS THAN .40 WIDE (1 mm)

- 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

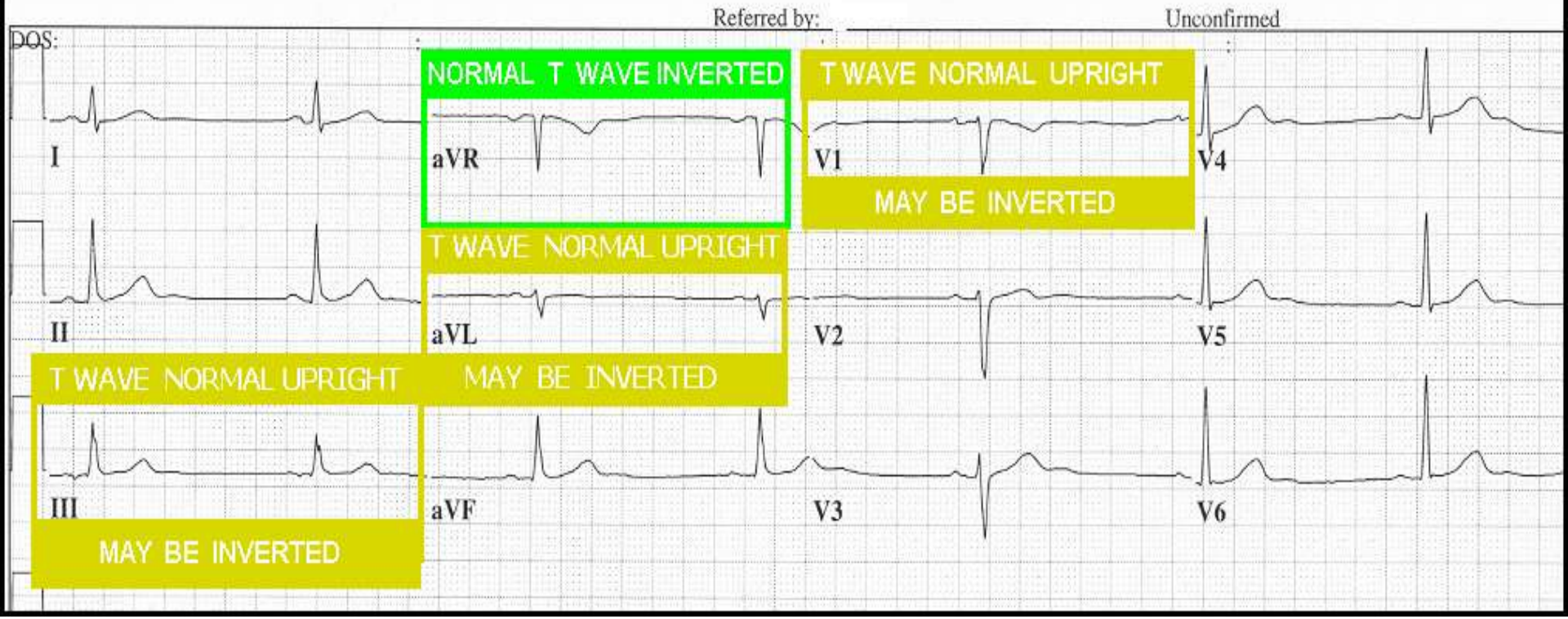
- THERE SHOULD BE NO Q WAVES IN LEADS V1, V2, or V3

THE T WAVE



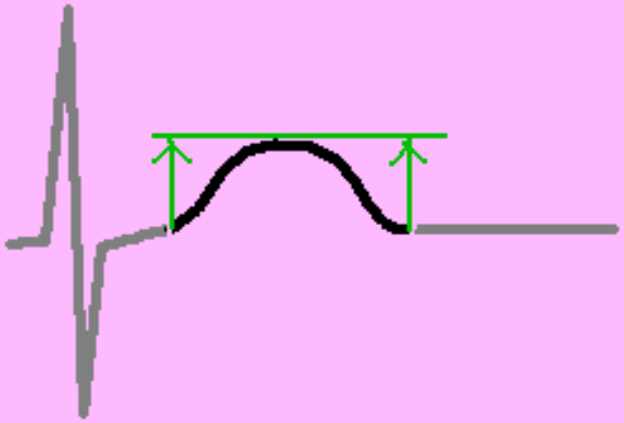
- SHOULD BE A "NICE," ROUNDED, CONVEX SHAPE
- SHOULD BE SYMMETRICAL
- SHOULD BE UPRIGHT IN ALL LEADS, EXCEPT AVR
- MAY BE INVERTED IN LEADS I, III, and V1

Leads where the T WAVE may be INVERTED:



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

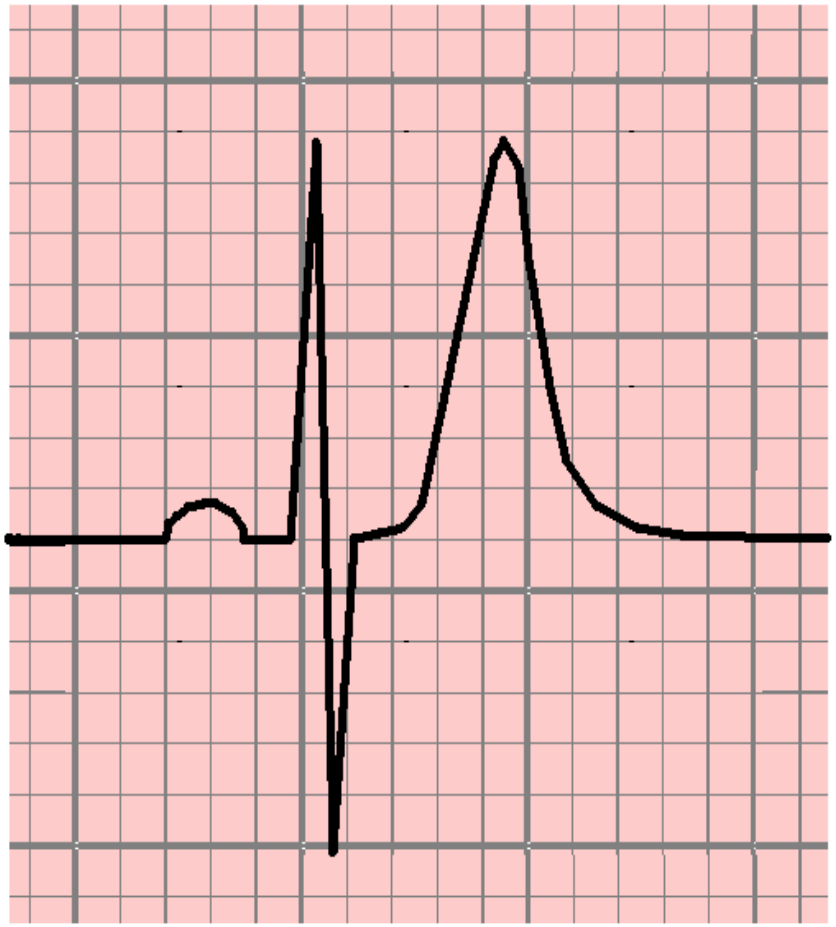
THE T WAVE



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:

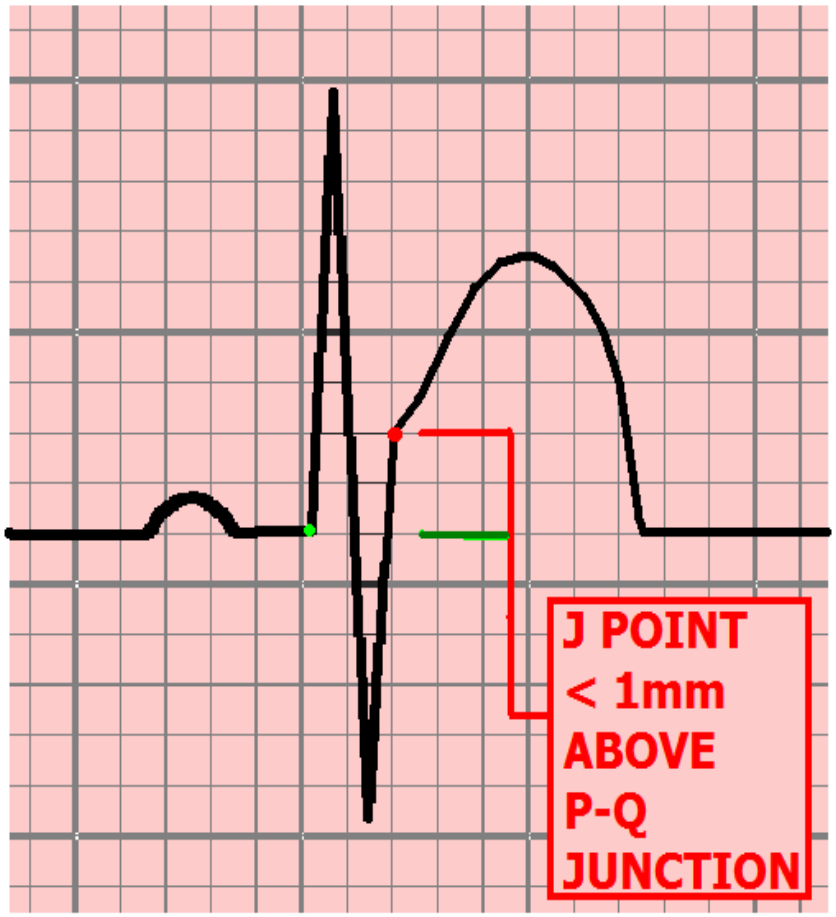


CONDITION:

- 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.

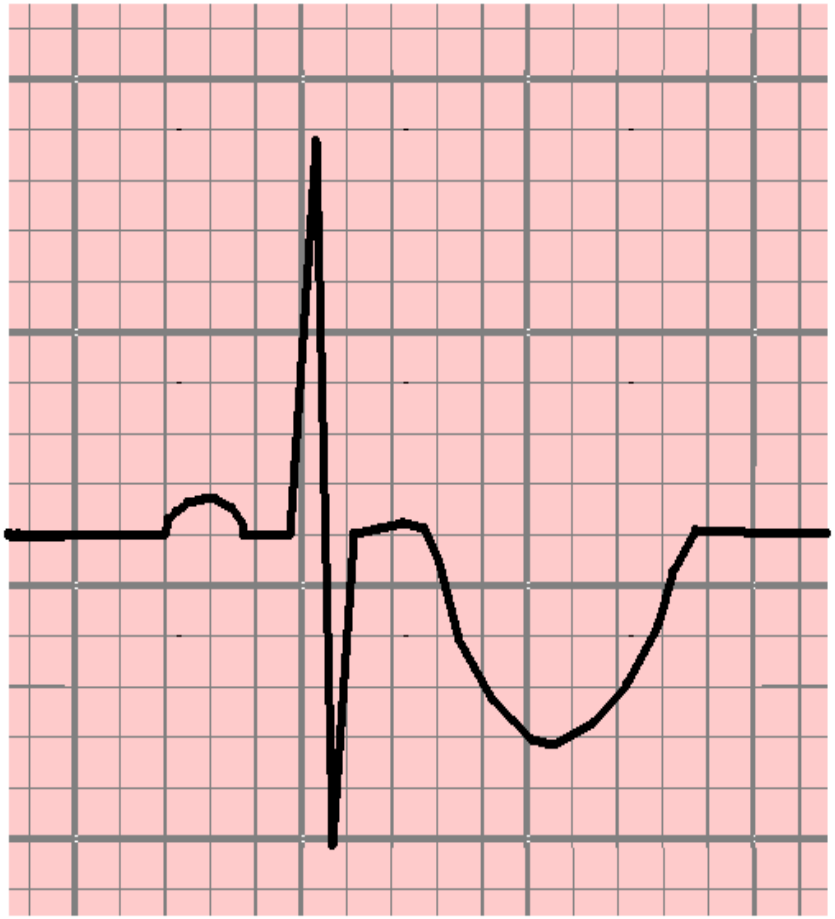
ON THE NEXT PAGE IN YOUR BOOK ARE SOME EXAMPLES OF THE ABOVE CONDITIONS

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:



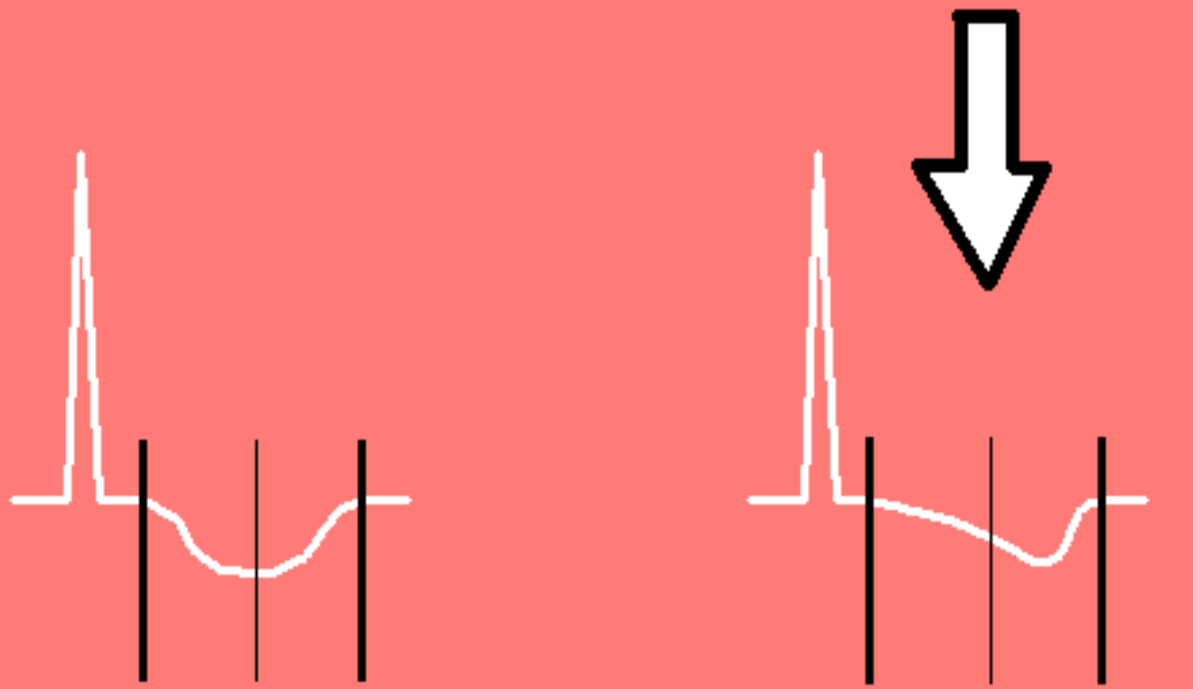
CONDITION:

- 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

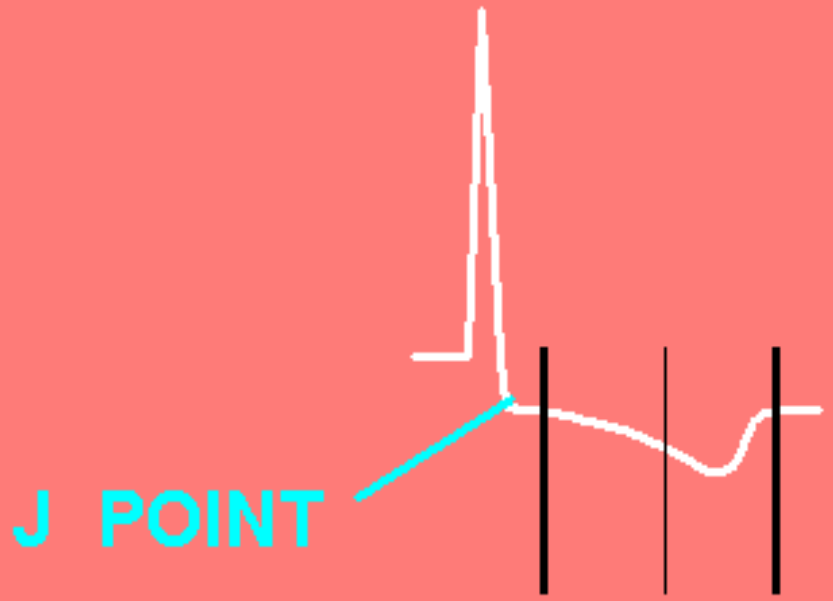
☞ T-WAVES ARE INVERTED and ASYMMETRICAL



CHAMBER ENLARGEMENT

VENTRICULAR STRAIN PATTERNS

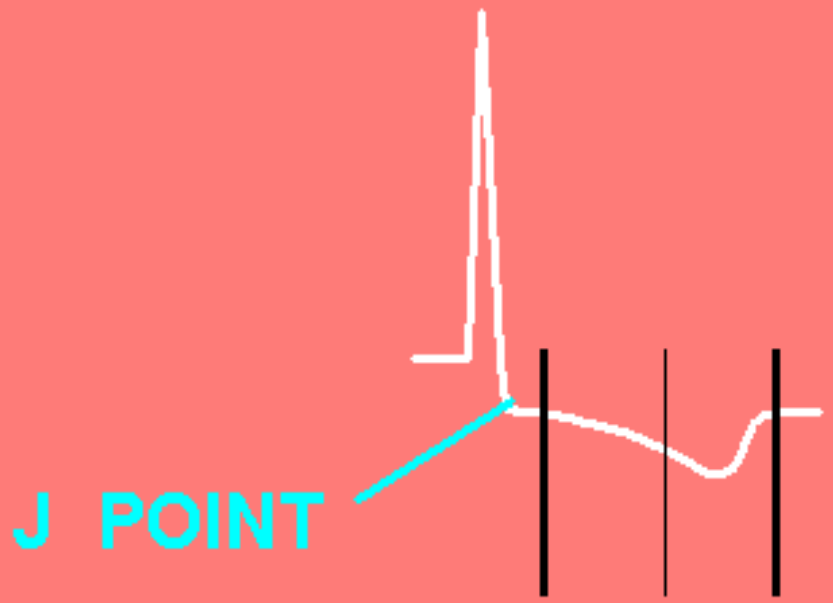
- T WAVES ARE INVERTED AND ASMMETRICAL
- THERE MAY BE S-T SEGMENT DEPRESSION



CHAMBER ENLARGEMENT

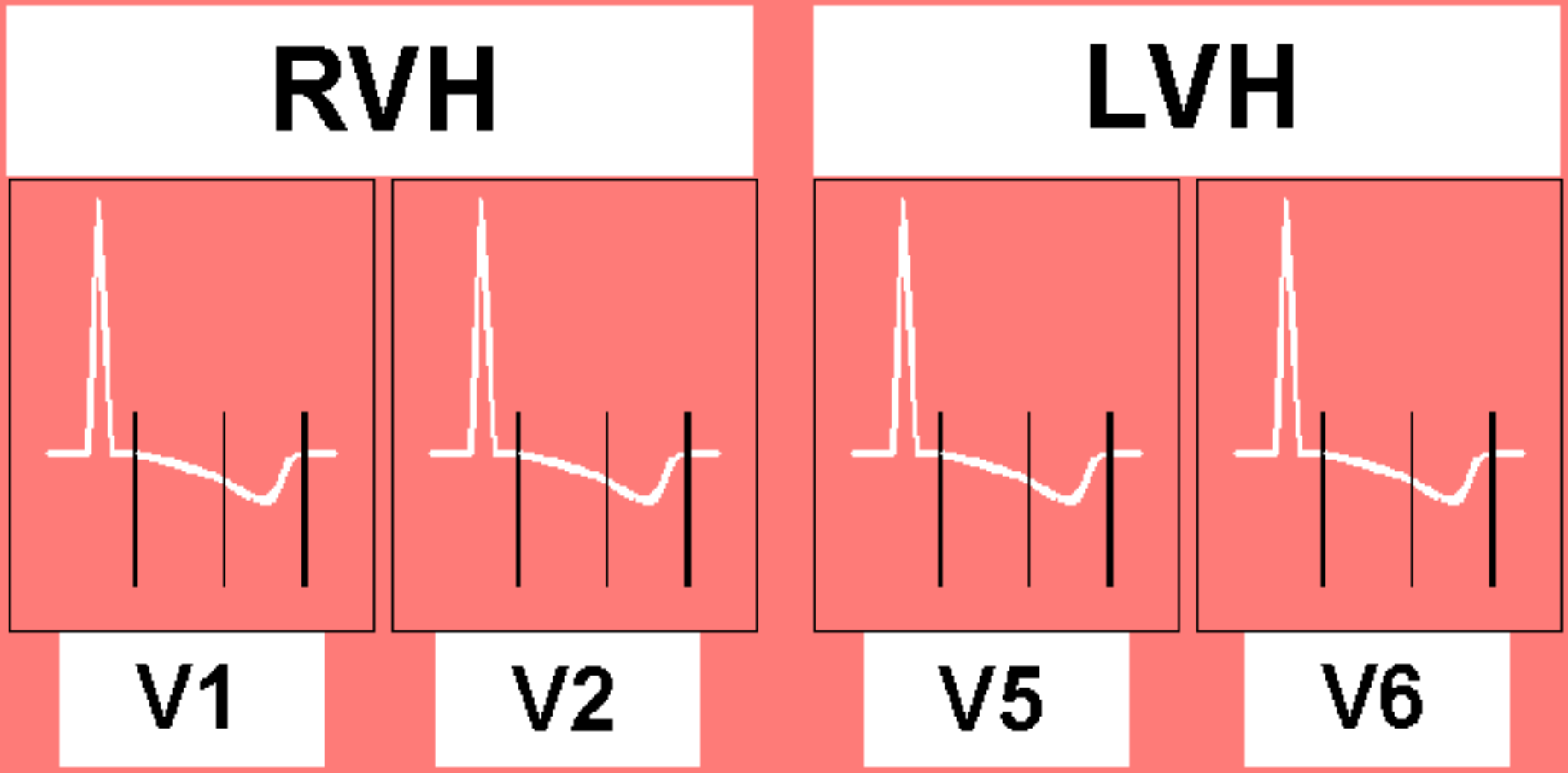
VENTRICULAR STRAIN PATTERNS

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



CHAMBER ENLARGEMENT

VENTRICULAR STRAIN PATTERNS

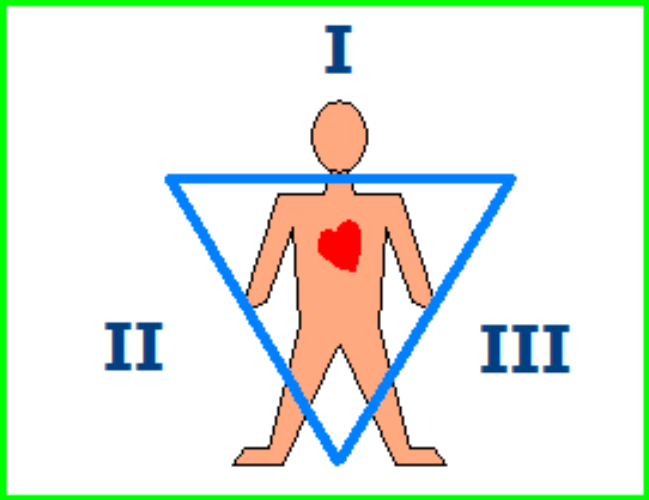



hang in there ! . . . .
it's almost LUNCH TIME ! ! ! !
Wayne "Will" Ruppert, III

EVALUATE THE AXIS IN BOTH PLANES

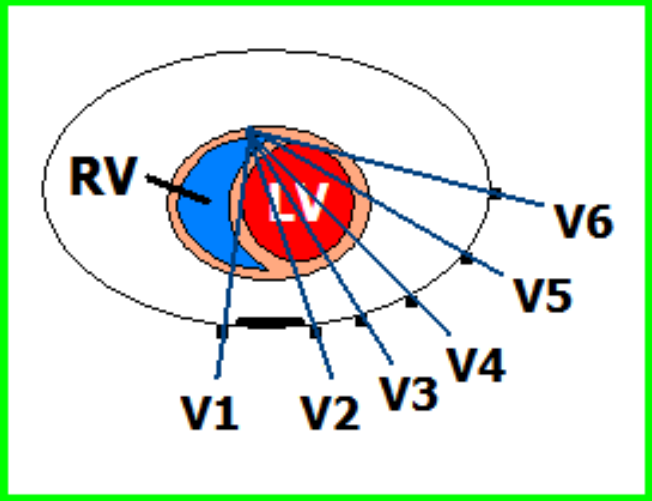
- **VERTICAL**

" AXIS DEVIATION "



- **HORIZONTAL**

" AXIS ROTATION "



AXIS DEVIATION

	LEAD I	LEAD AVF
NORMAL		
LEFT		
RIGHT		
FAR RIGHT		

66 yr
Male Caucasian

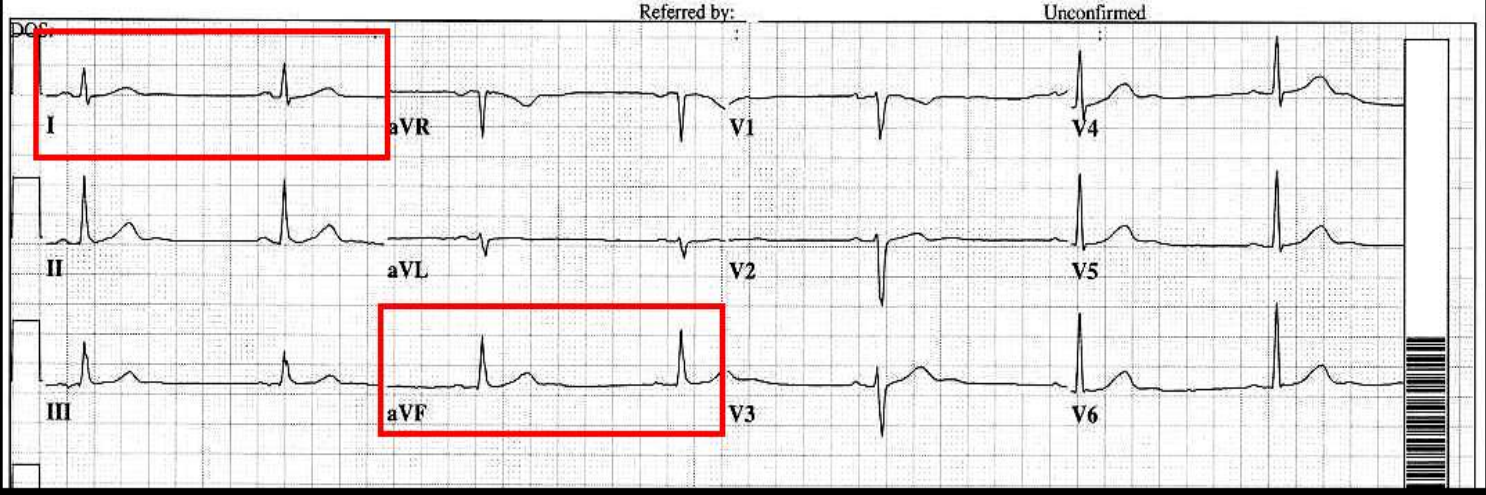
Room:401A
Loc:6 Option:16

Vent. rate	41	BPM
PR interval	192	ms
QRS duration	94	ms
QT/QTc	526/433	ms
P–R–T axes	38 70	58

NORMAL AXIS

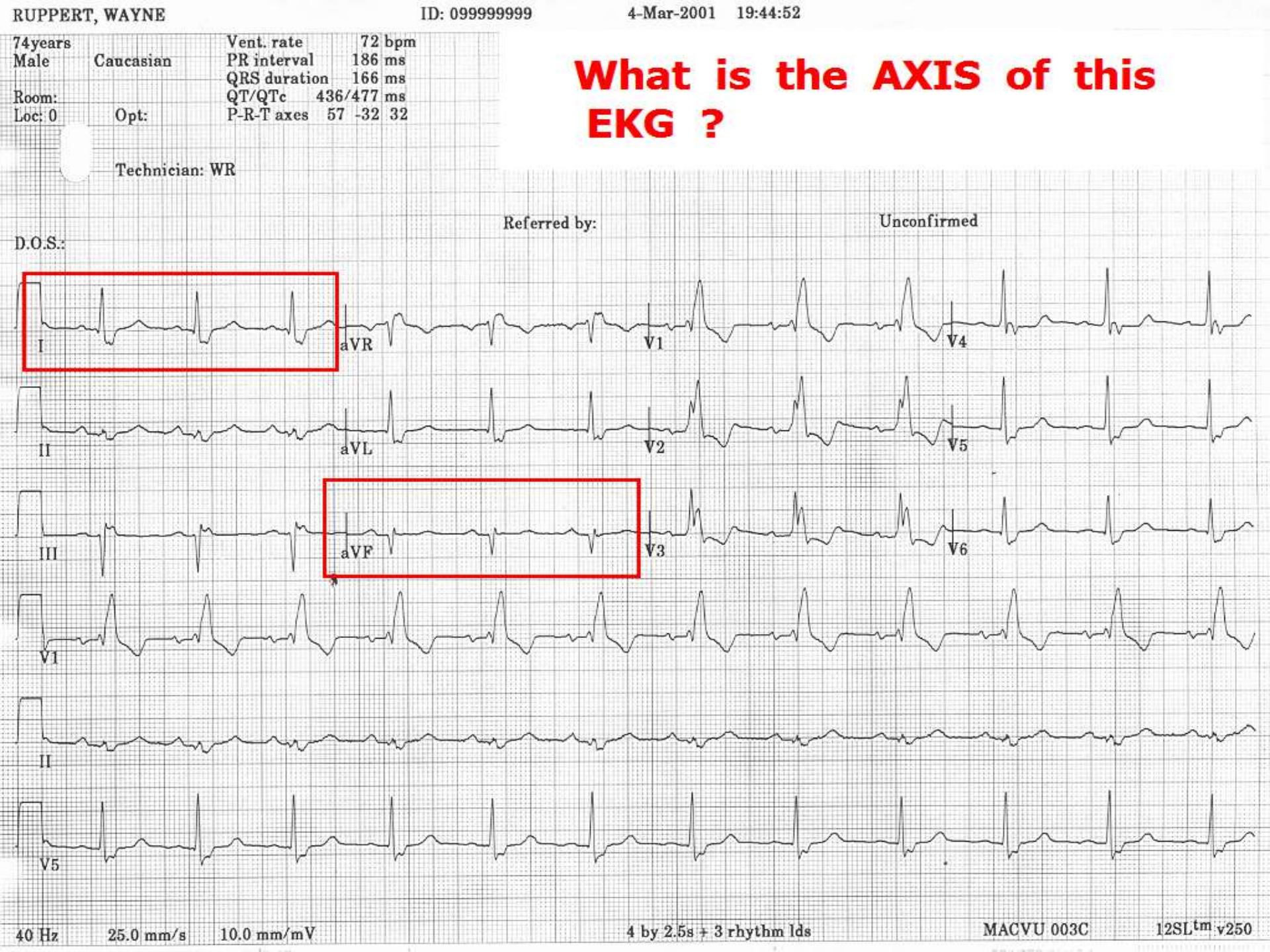
Technician:

Referred by: Unconfirmed



AXIS DEVIATION

	LEAD I	LEAD AVF
NORMAL	⅄	⅄
LEFT	⅄	V
RIGHT	V	⅄
FAR RIGHT	V	V


RUPPERT, WAYNE
ID: 099999999
4-Mar-2001 19:44:52
74years
Male
Caucasian
Room:
Loc: 0
Opt:
Vent. rate 72 bpm
PR interval 186 ms
QRS duration 166 ms
QT/QTc 436/477 ms
P-R-T axes 57 -32 32
Technician: WR
What is the AXIS of this EKG ?
Referred by:
Unconfirmed
D.O.S.:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
40 Hz
25.0 mm/s
10.0 mm/mV
4 by 2.5s + 3 rhythm lds
MACVU 003C
12SL tm v250

☞ 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

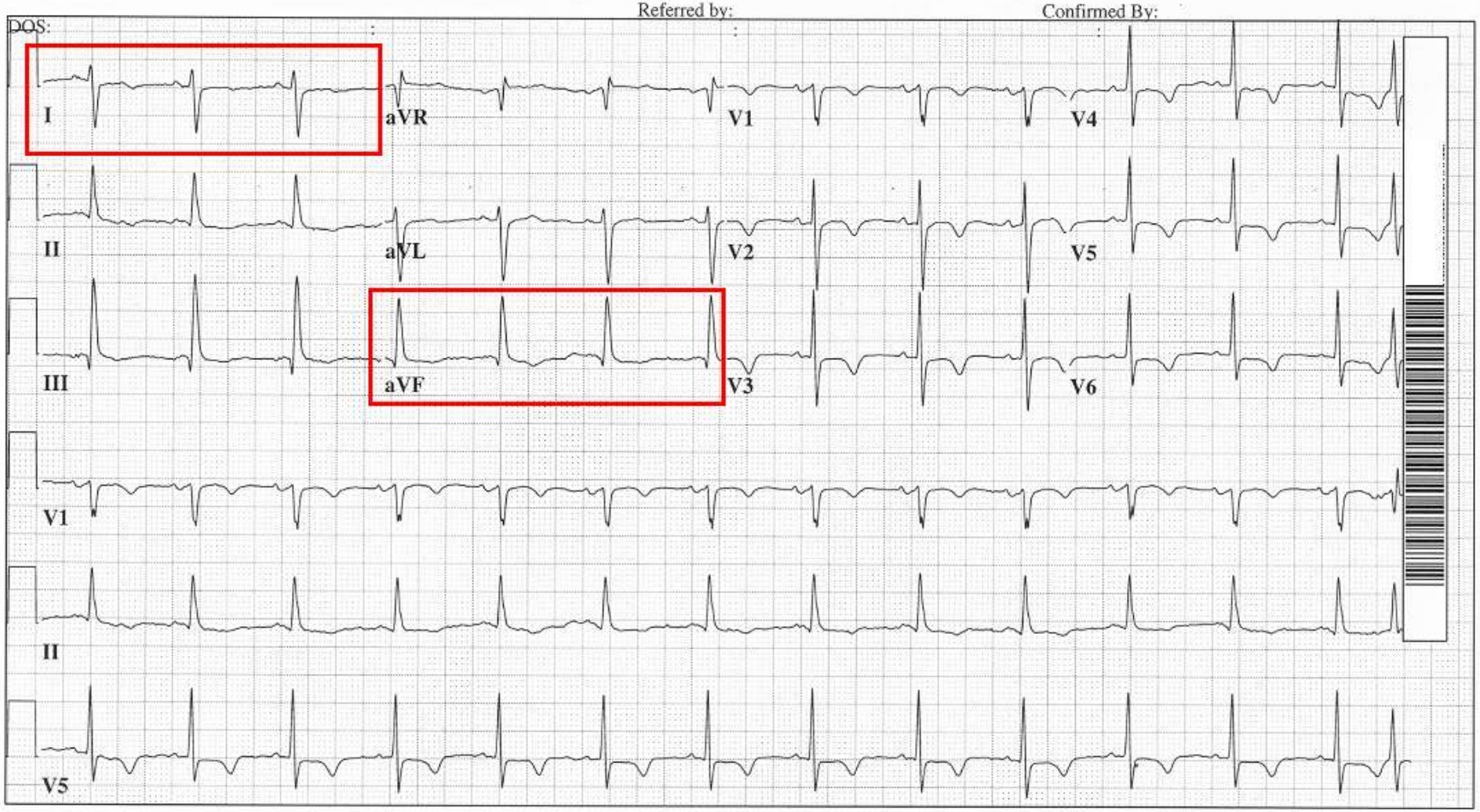
Room:303A
Loc:6 Option:11

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:



25mm/s 10mm/mV 40Hz 005C 12SL 233 CID: 6

EID:13 EDT:

☞ AXIS DEVIATION

	LEAD I	LEAD AVF
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 FASICULAR BLOCK
- **PULMONARY EMBOLUS**
- DEXTROCARDIA
- C.O.P.D.
- ATRIAL / VENTRICULAR SEPTAL DEFECTS

02:55:00

Male Caucasian

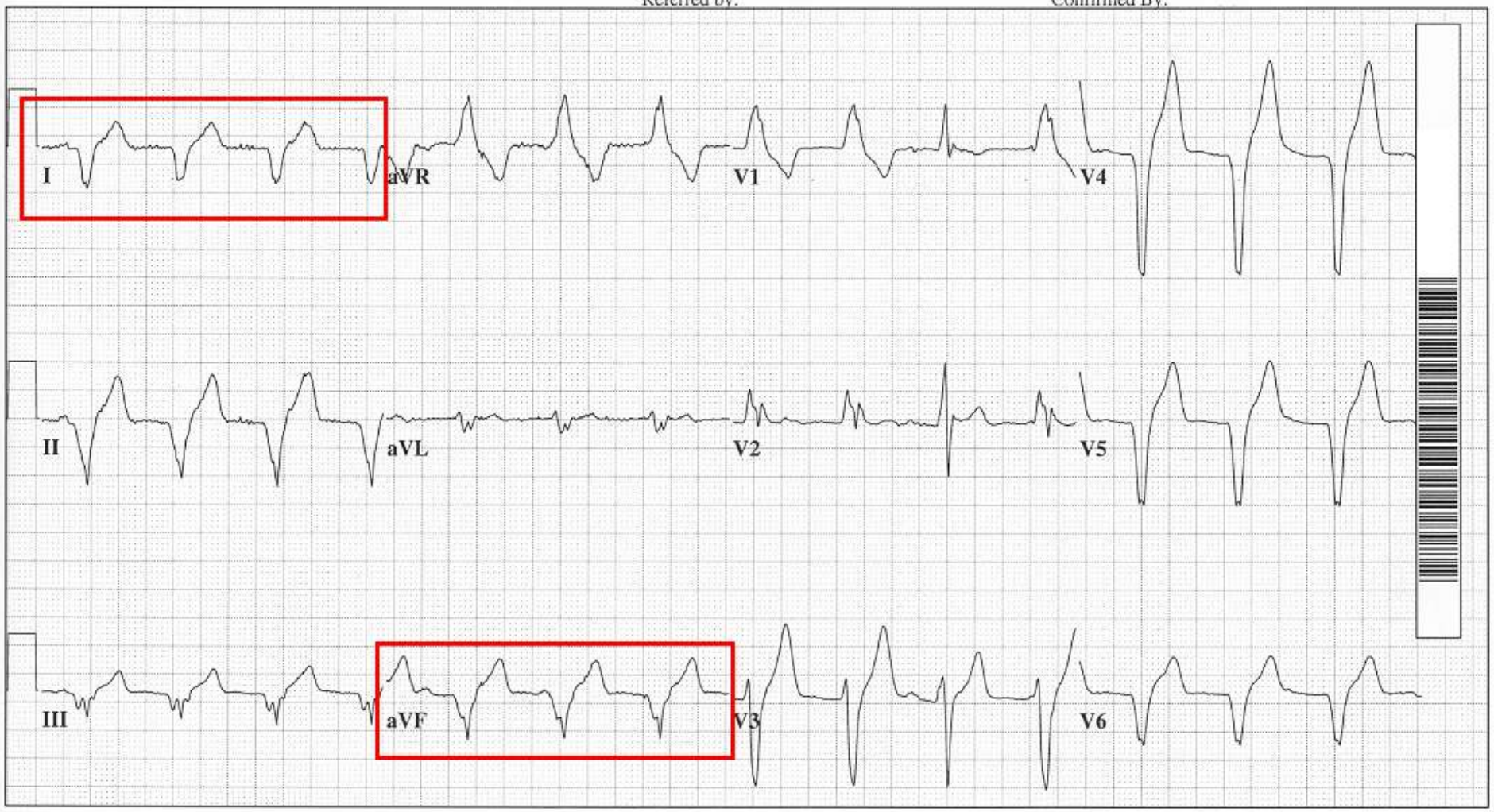
Room:5
Loc:1

Vent. rate	92	BPM
PR interval	*	
QRS duration	172	ms
QT/QTc	420/520	ms
P–R–T axes	* –123	61

ACCELERATED IDIOVENTRICULAR RHYTHM

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		

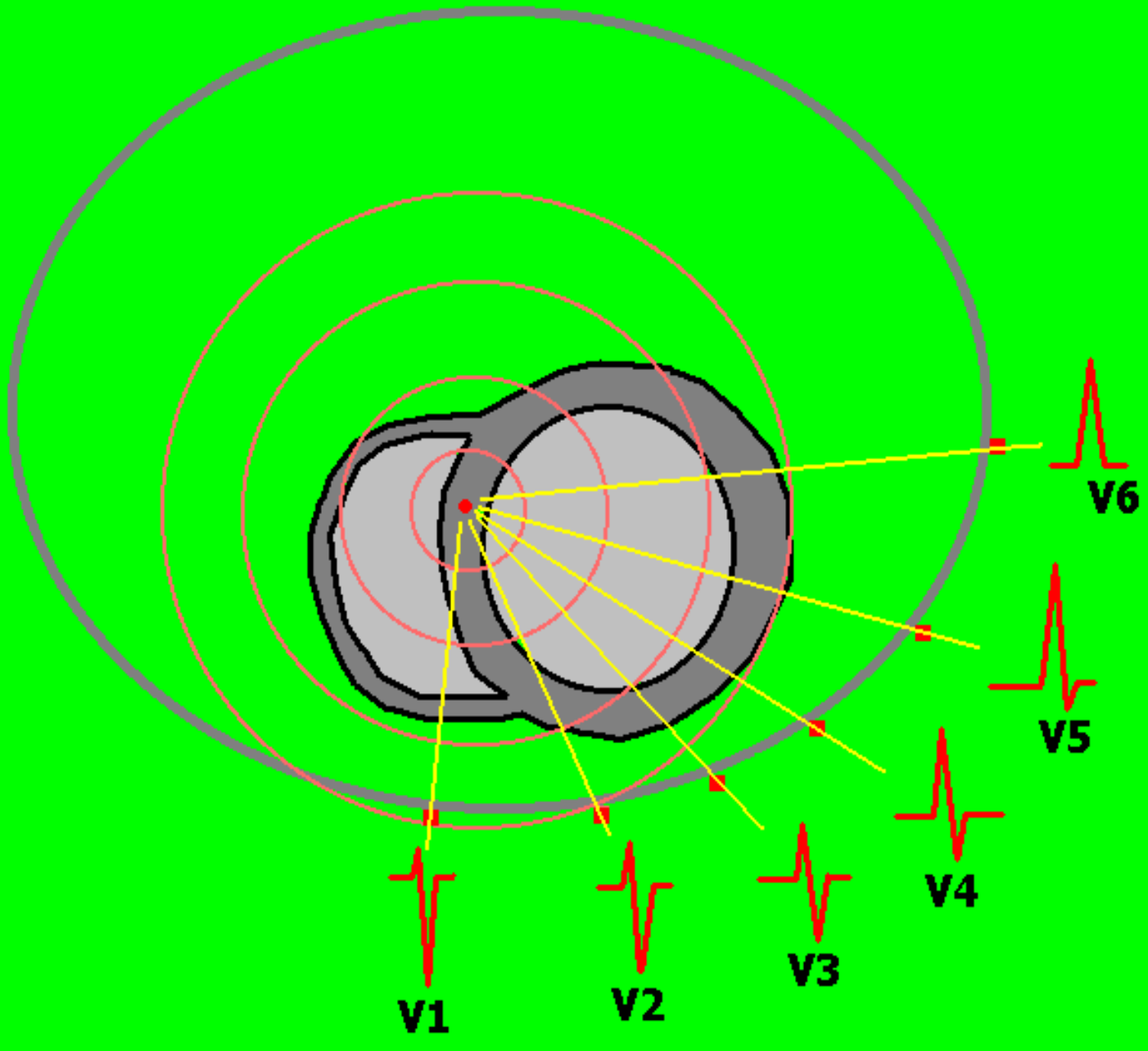
COMMON CONDITIONS WHICH *MAY* CAUSE

(NO-MAN'S LAND AXIS)

FAR RIGHT AXIS DEVIATION:

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

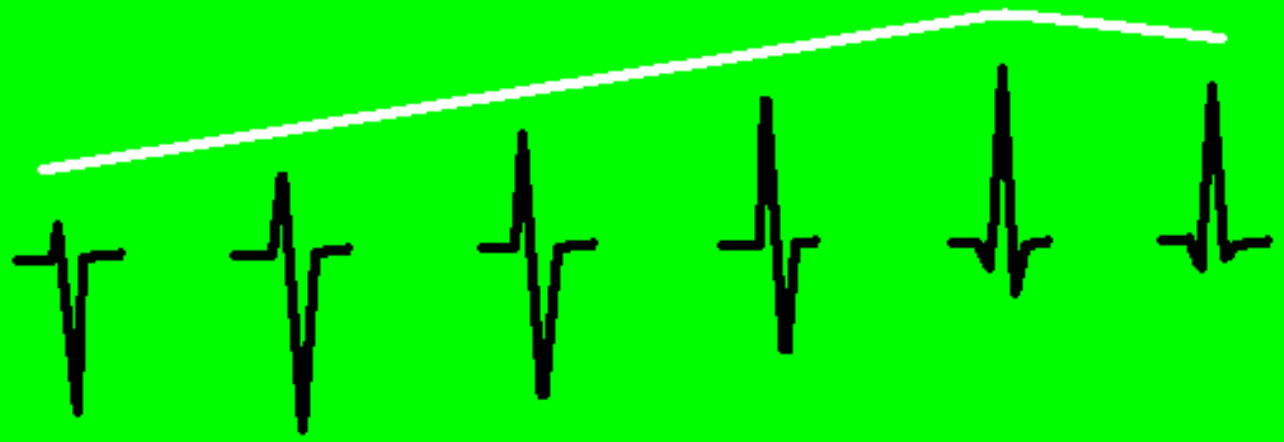
AXIS ROTATION



ASSESSING AXIS ROTATION:

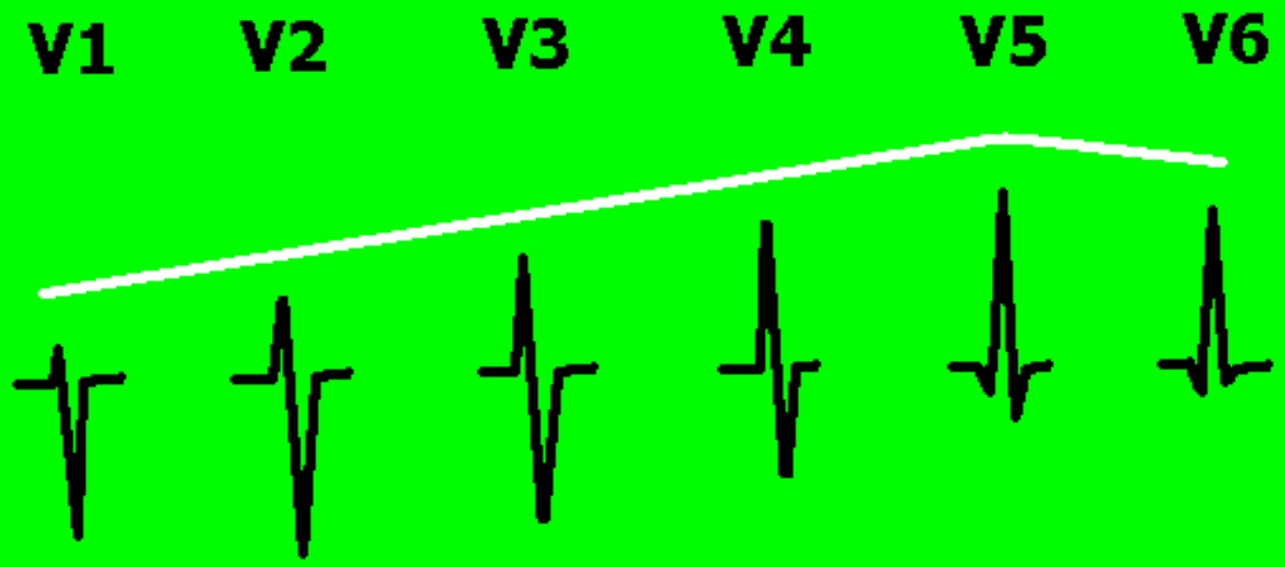
V1 V2 V3 V4 V5 V6

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

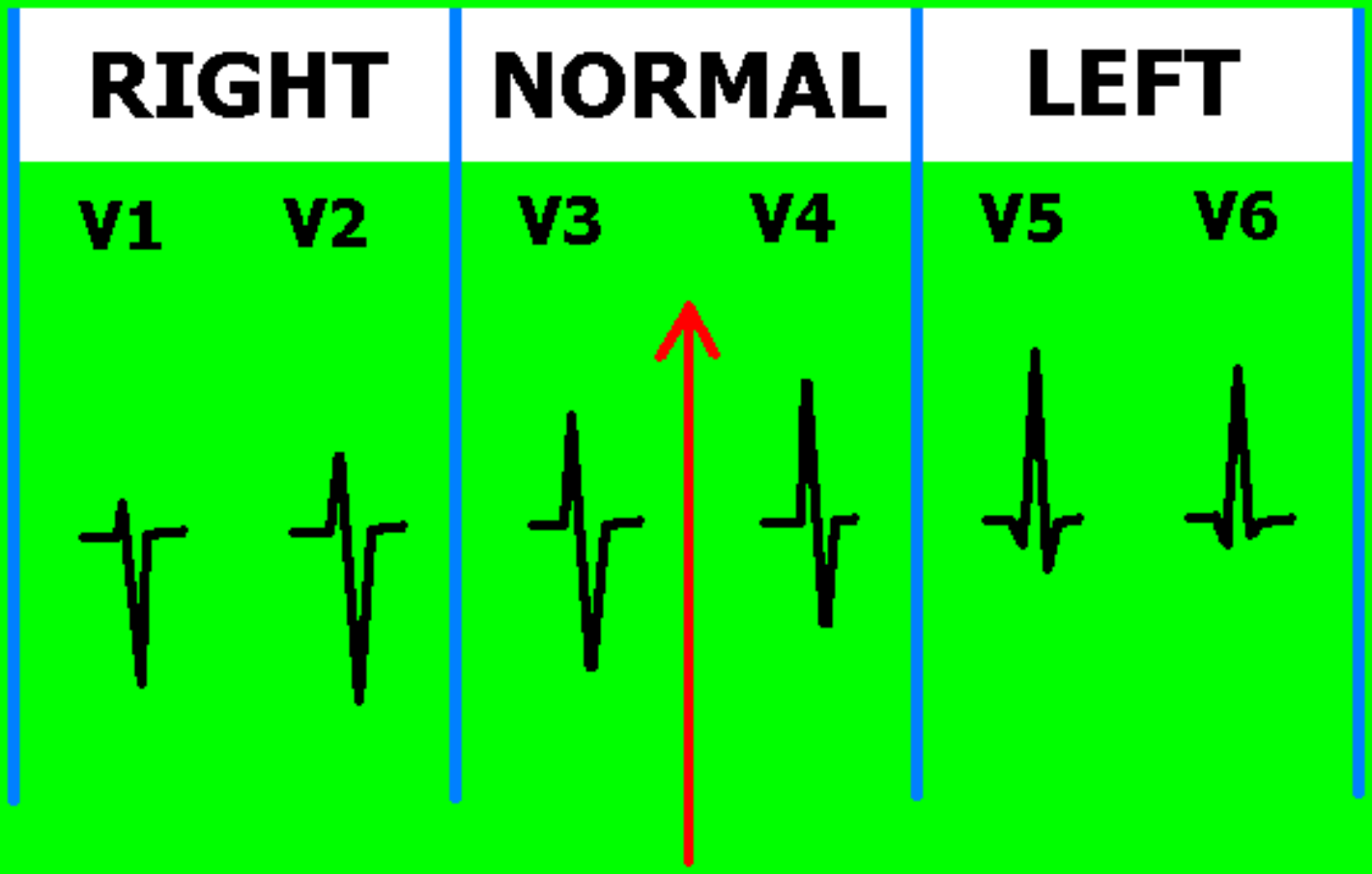
OCCURS IN THE LEAD WHERE THE QRS IS THE MOST **BIPHASIC**

AXIS ROTATION

IMPORTANT TRANSITION RULE

"Transition shifts TOWARD HYPERTROPHY and AWAY FROM NECROSIS."

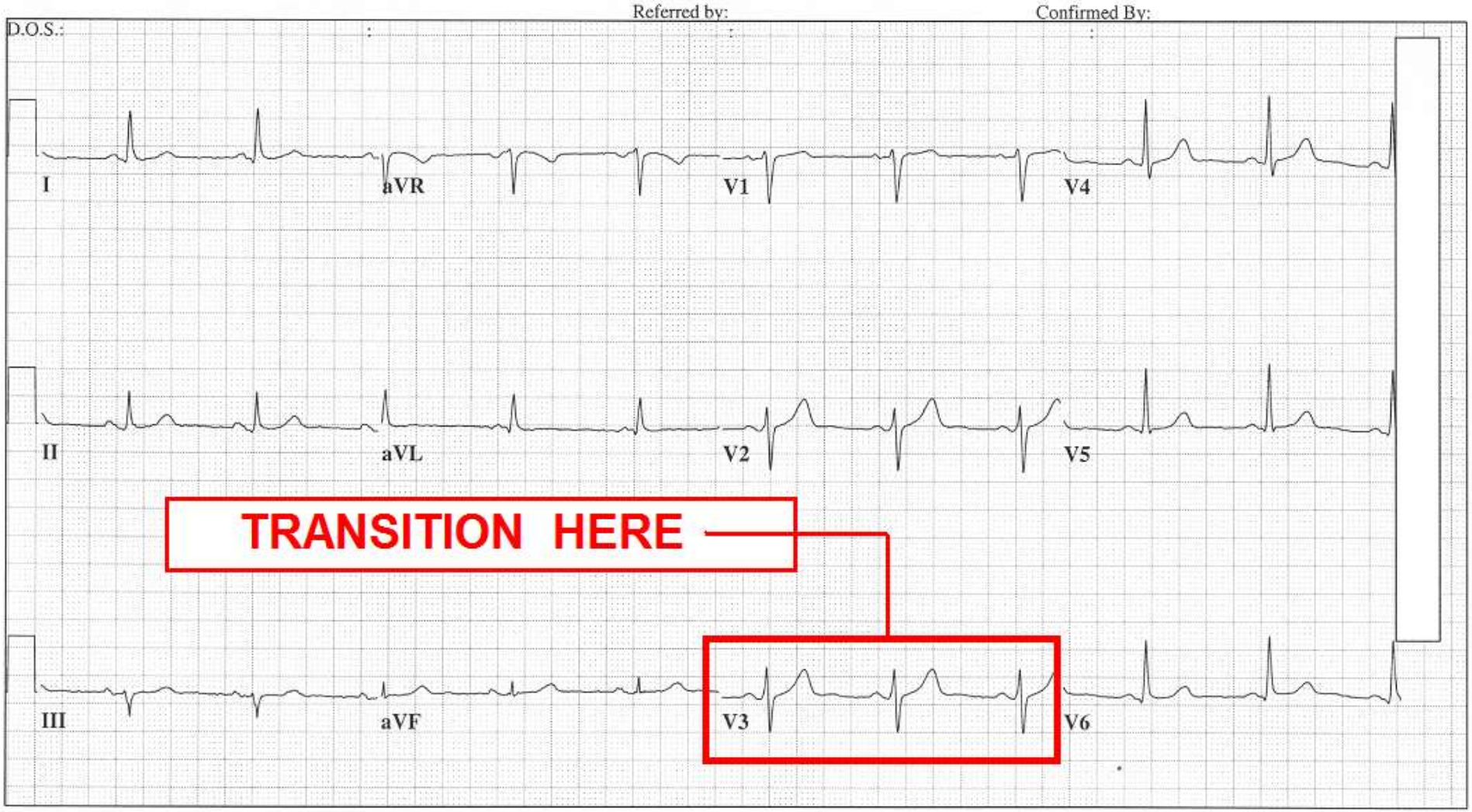
AXIS ROTATION



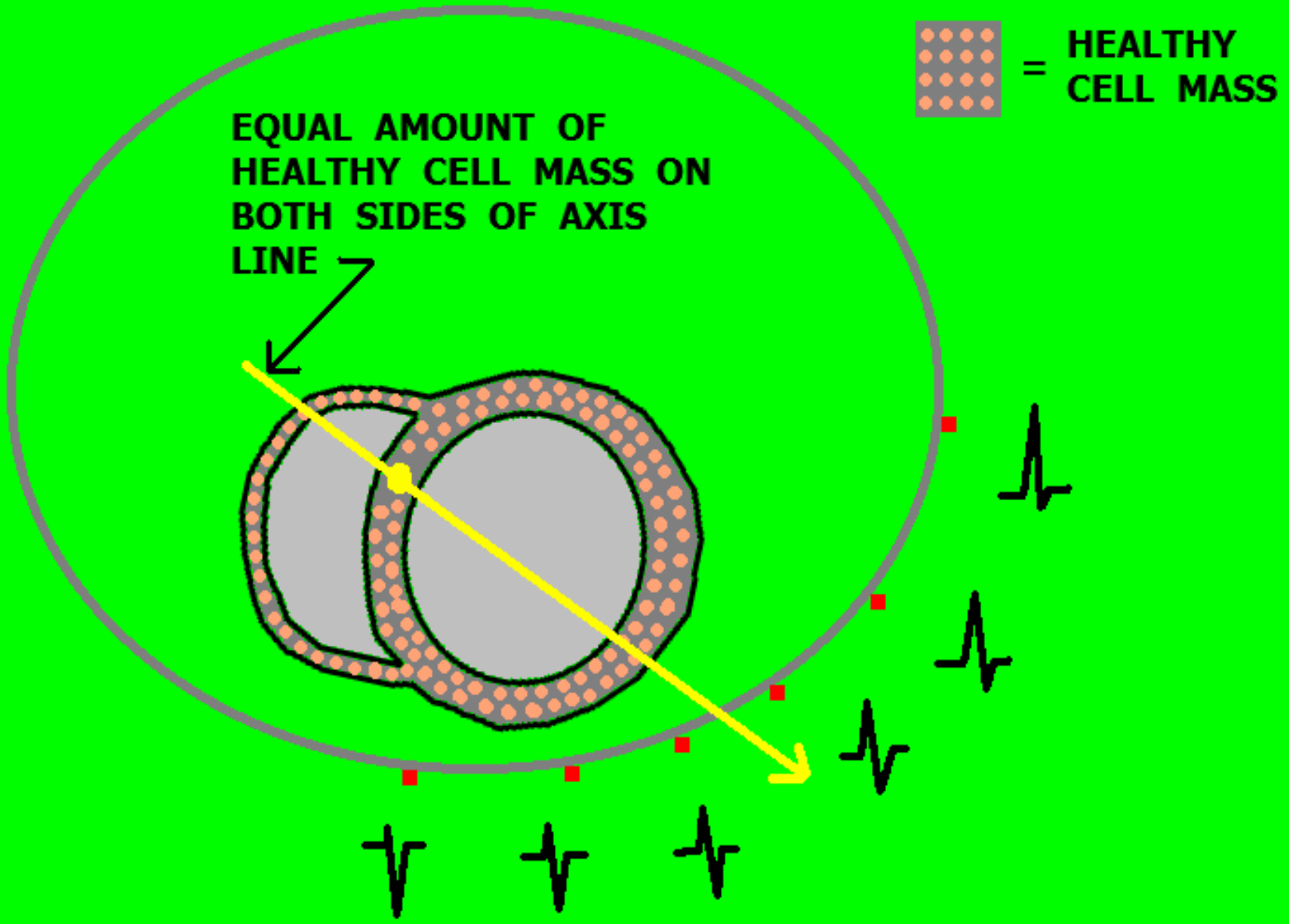
TRANSITION SHOULD OCCUR IN LEADS V3 or V4

NORMAL TRANSITION

IS BETWEEN LEADS V3 and V4



NORMAL TRANSITION



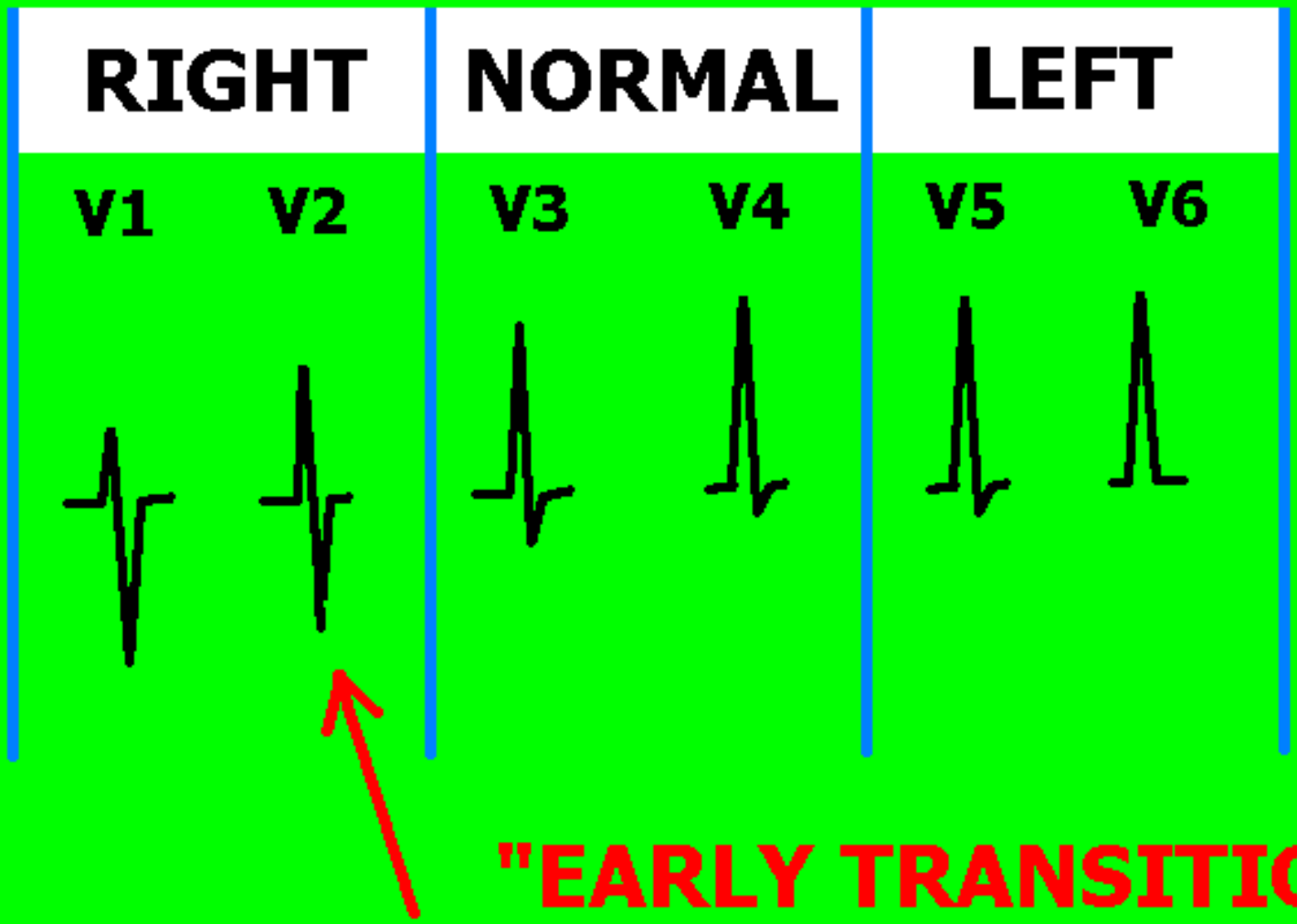
ASSESSING AXIS ROTATION:

IMPORTANT NOTES:

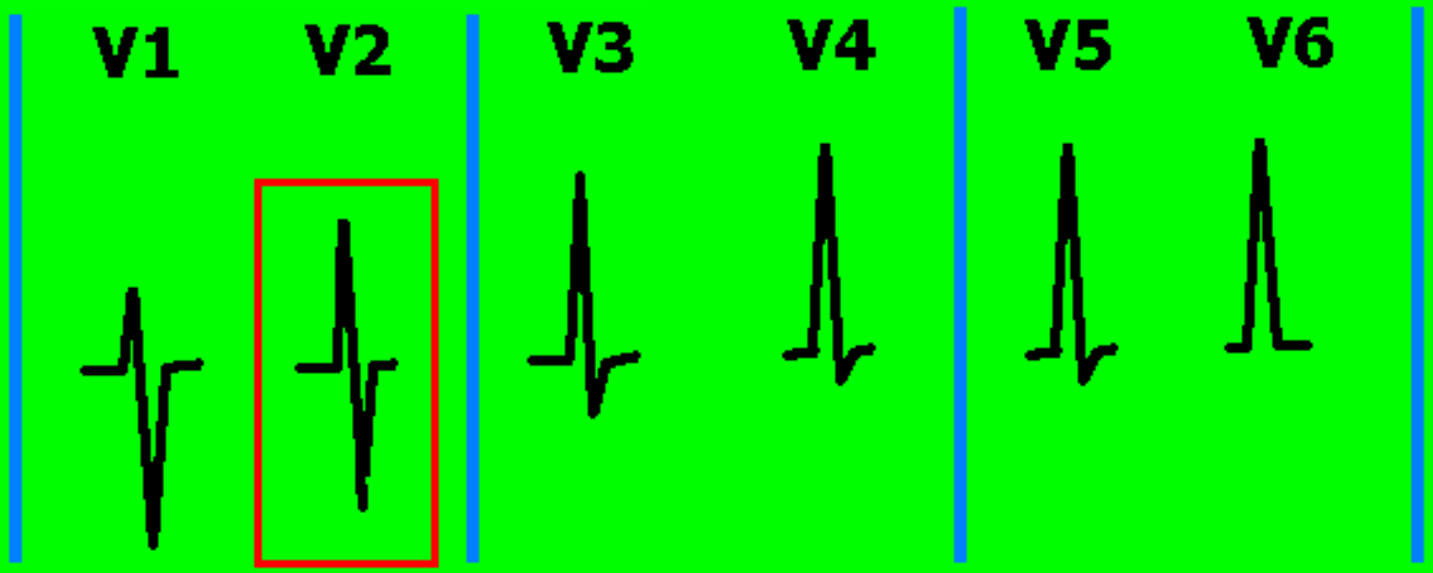
- As with all EKG-made DIAGNOSES, you must consider the TOTALITY of the PATIENT'S PRESENTATION. This includes the PATIENT'S CLINICAL PRESENTATION, RISK FACTOR PROFILE, and your INDEX OF SUSPICION.

- Validate all EKG-suspected DIAGNOSES with Additional, MORE ACCURATE diagnostic testing, e.g.: CARDIAC ECHO, CARDIAC CATHERIZATION, ELECTROPHYSIOLOGIC TESTING, MRI, etc.

AXIS ROTATION



* 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

RUPPERT, WAYNE ID: 099999999 4-Mar-2001 19:44:52 ST JOSEPHS HOSPITAL

74years
Male Caucasian

Room:
Loc: 0 Opt:

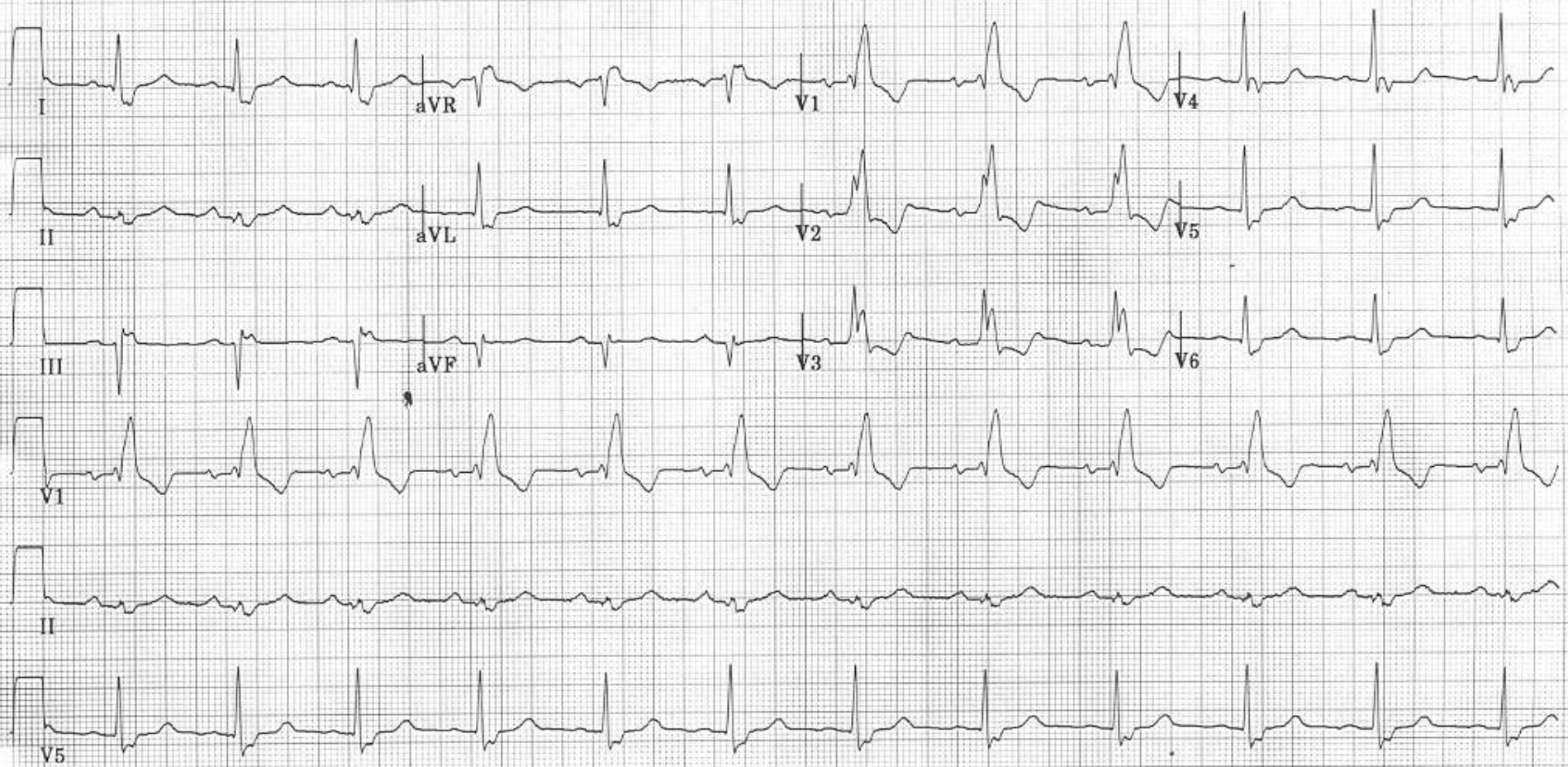
Technician: WR

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.:



40 Hz 25.0 mm/s 10.0 mm/mV 4 by 2.5s + 3 rhythm lds MACVU 003C 12SL^{tm} v250

COMMON CAUSES OF EARLY TRANSITION

. . . . SOME HELPFUL CLUES:

1. **Right Bundle Branch Block (RBBB)**
   - QRS wider than 120ms
   - Supraventricular rhythm (normal P : QRS relationship)
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   - Presence of Delta Waves
   - Wide QRS complexes

COMMON CAUSES OF EARLY TRANSITION

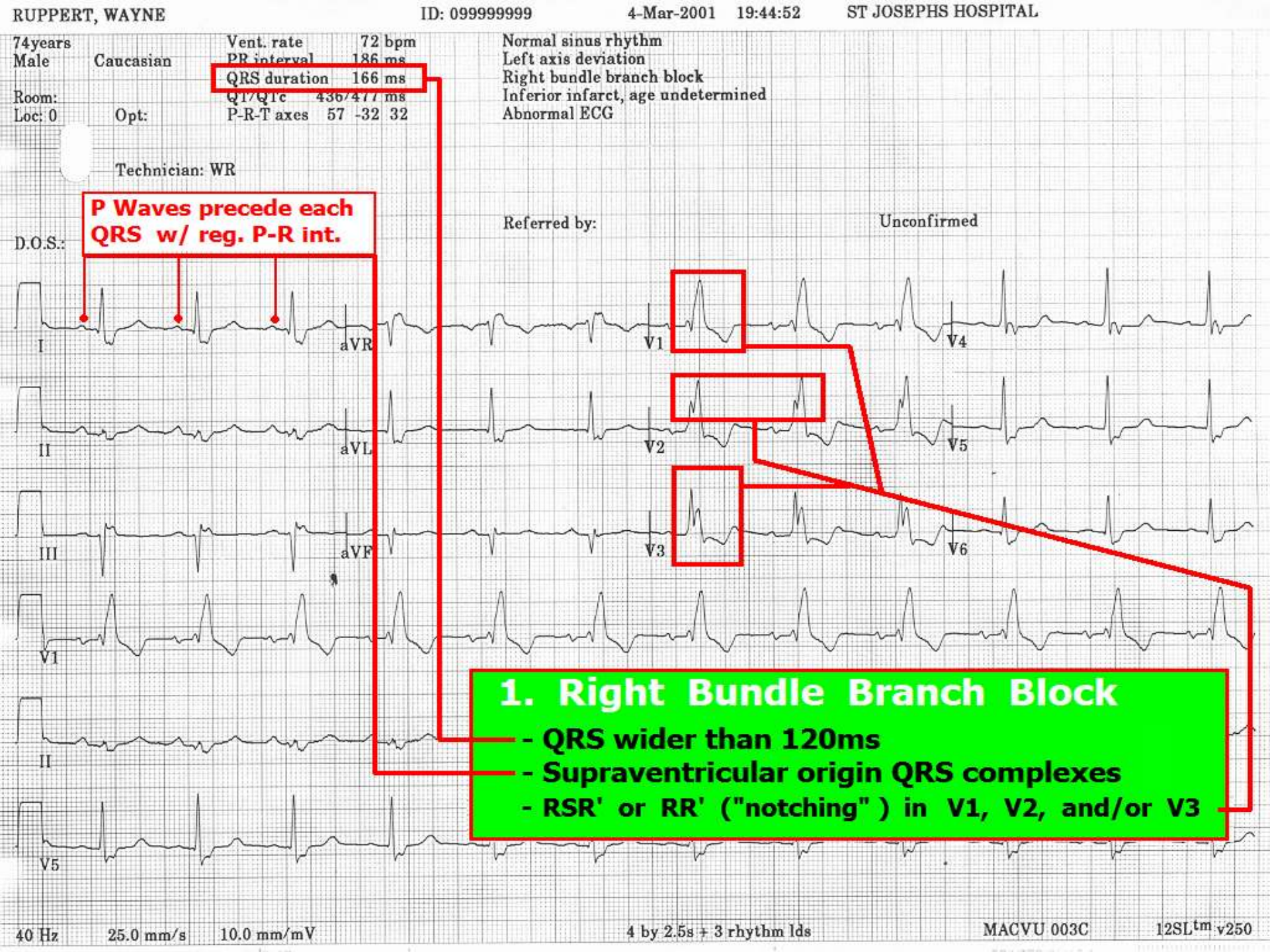
. . . . SOME HELPFUL CLUES:

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~~


RUPPERT, WAYNE
ID: 099999999
4-Mar-2001 19:44:52
ST JOSEPHS HOSPITAL
74years
Male
Caucasian
Vent. rate 72 bpm
PR interval 186 ms
QRS duration 166 ms
Q1/Q1c 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
Room:
Loc: 0
Opt:
Technician: WR
P Waves precede each QRS w/ reg. P-R int.
Referred by:
Unconfirmed
D.O.S.:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
1. Right Bundle Branch Block
- QRS wider than 120ms
- Supraventricular origin QRS complexes
- RSR' or RR' ("notching") in V1, V2, and/or V3
40 Hz
25.0 mm/s
10.0 mm/mV
4 by 2.5s + 3 rhythm lds
MACVU 003C
12SL tm v250

15–MAR–2000 10:29:05 ST. JOSEPH'S HOSPITAL–ER ROUTINE RETRIEVAL

31 yr
Male Black

Room:ER
Loc:3 Option:16

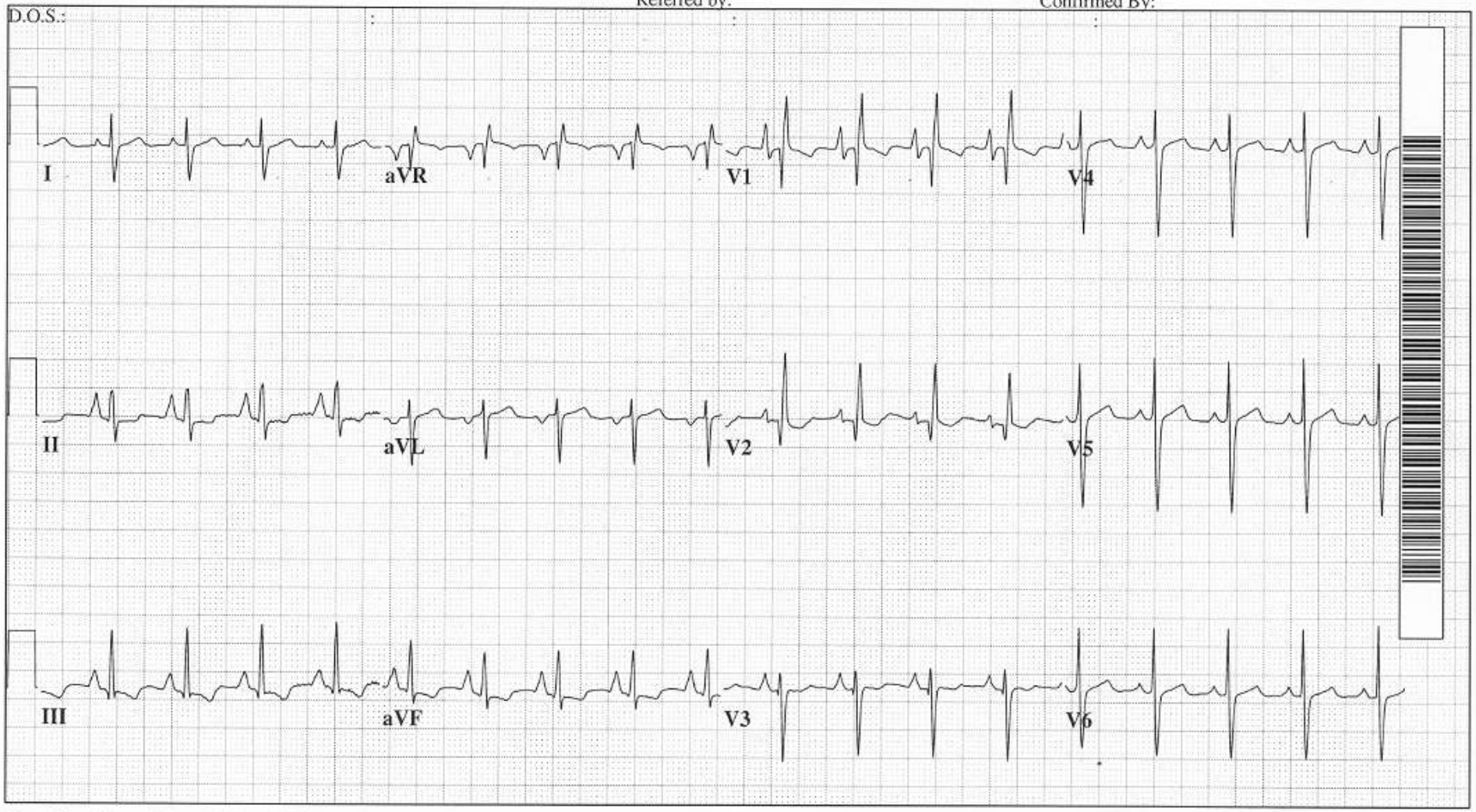
Vent. rate	109	BPM
PR interval	122	ms
QRS duration	84	ms
QT/QTc	296/398	ms
P–R–T axes	79 117	–27

Technician: EKG CLASS #WR03446043

What is the cause of EARLY TRANSITION in this EKG ?

Referred by: Confirmed By:

D.O.S.:



25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 13

EID:17 EDT: 08:56 20–MAR–2000 ORDER:

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

15–MAR–2000 10:29:05 ST. JOSEPH'S HOSPITAL–ER ROUTINE RETRIEVAL

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.:

I aVR V1 V4

II aVL

III aVF V3 V6

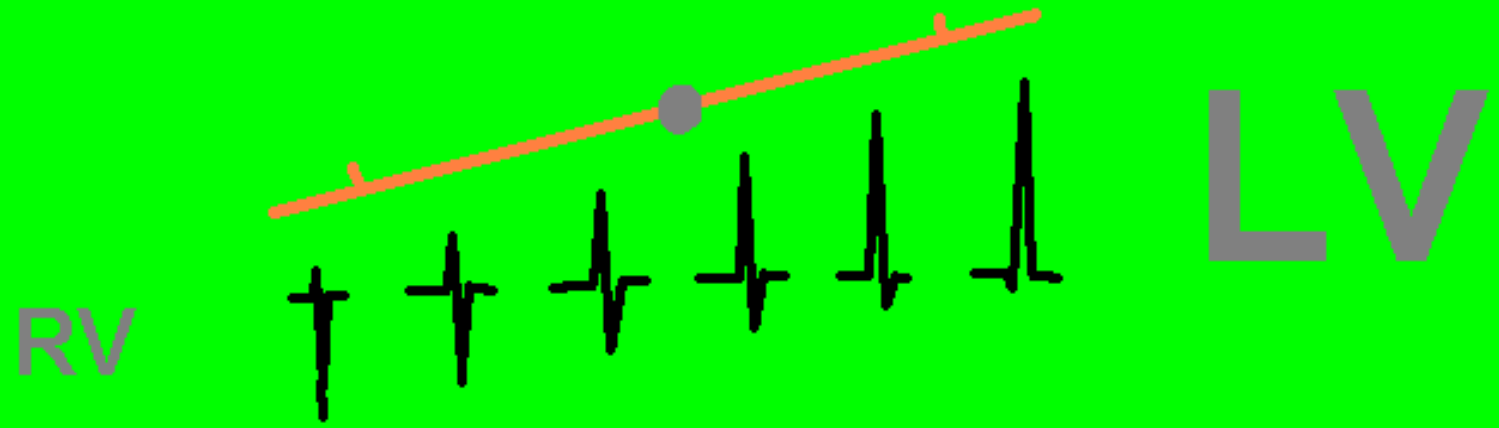
2. Right Ventricular Hypertrophy (RVH)

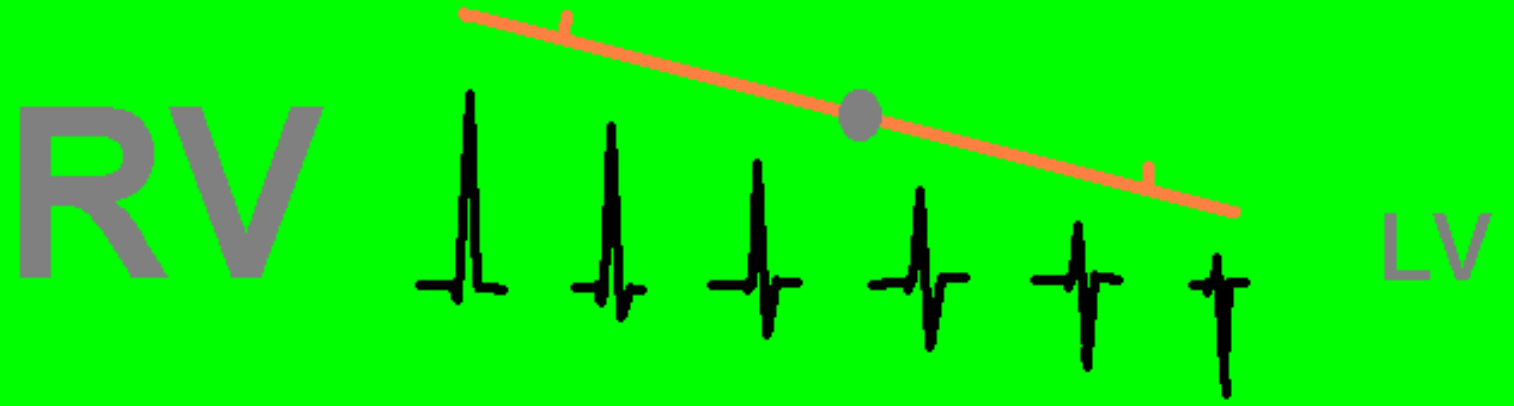
- **Corresponding Right Atrial Hypertrophy (RAH)**
- **Right Axis Deviation (RAD)**
- **QRS in LEAD I more NEGATIVE than POSITIVE (R<S)**

25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 13

EID:17 EDT: 08:56 20–MAR–2000 ORDER:

"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

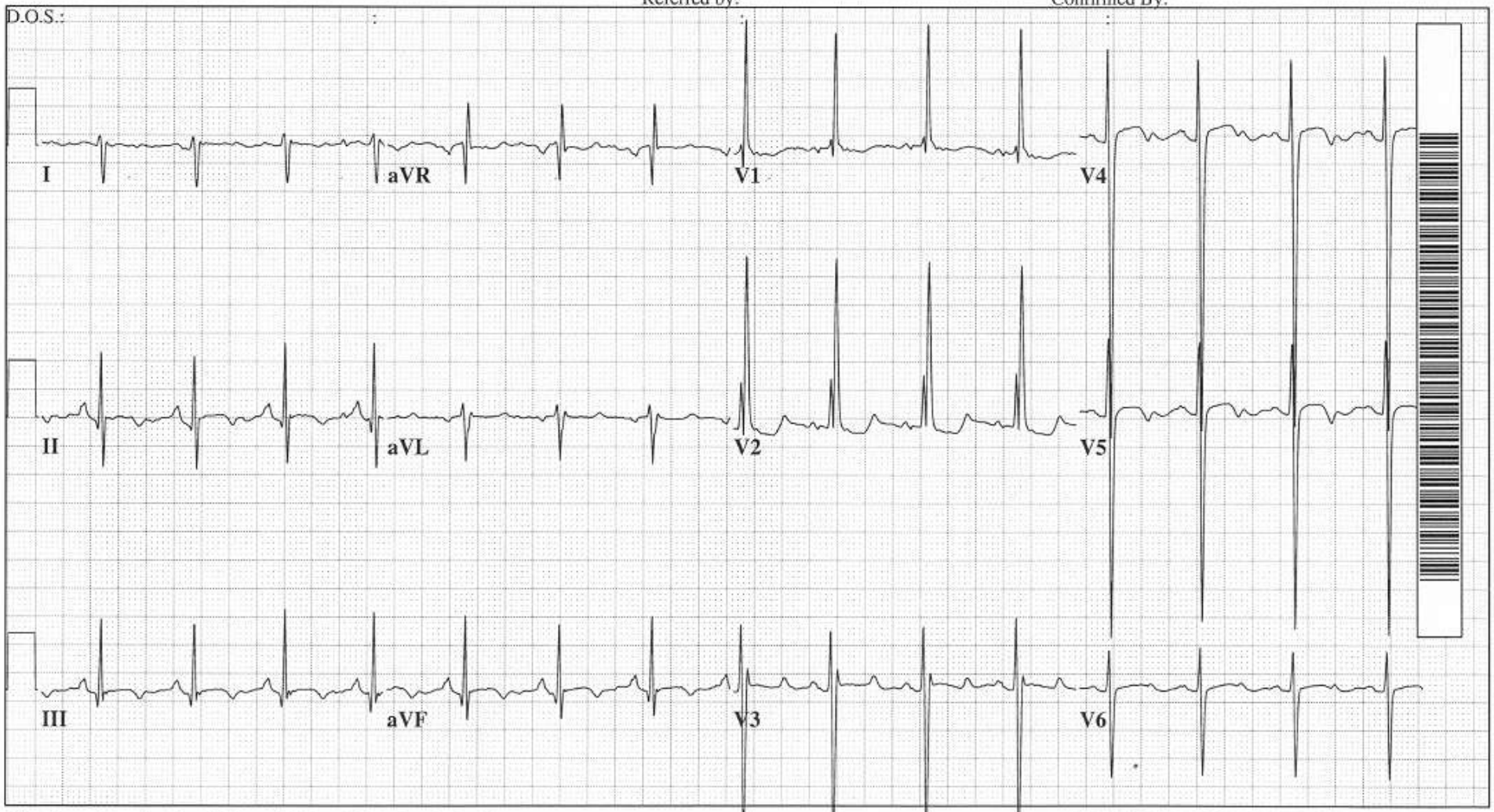
Vent. rate	90	BPM
PR interval	136	ms
QRS duration	94	ms
QT/QTc	378/462	ms
P-R-T axes	77 123	58

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:

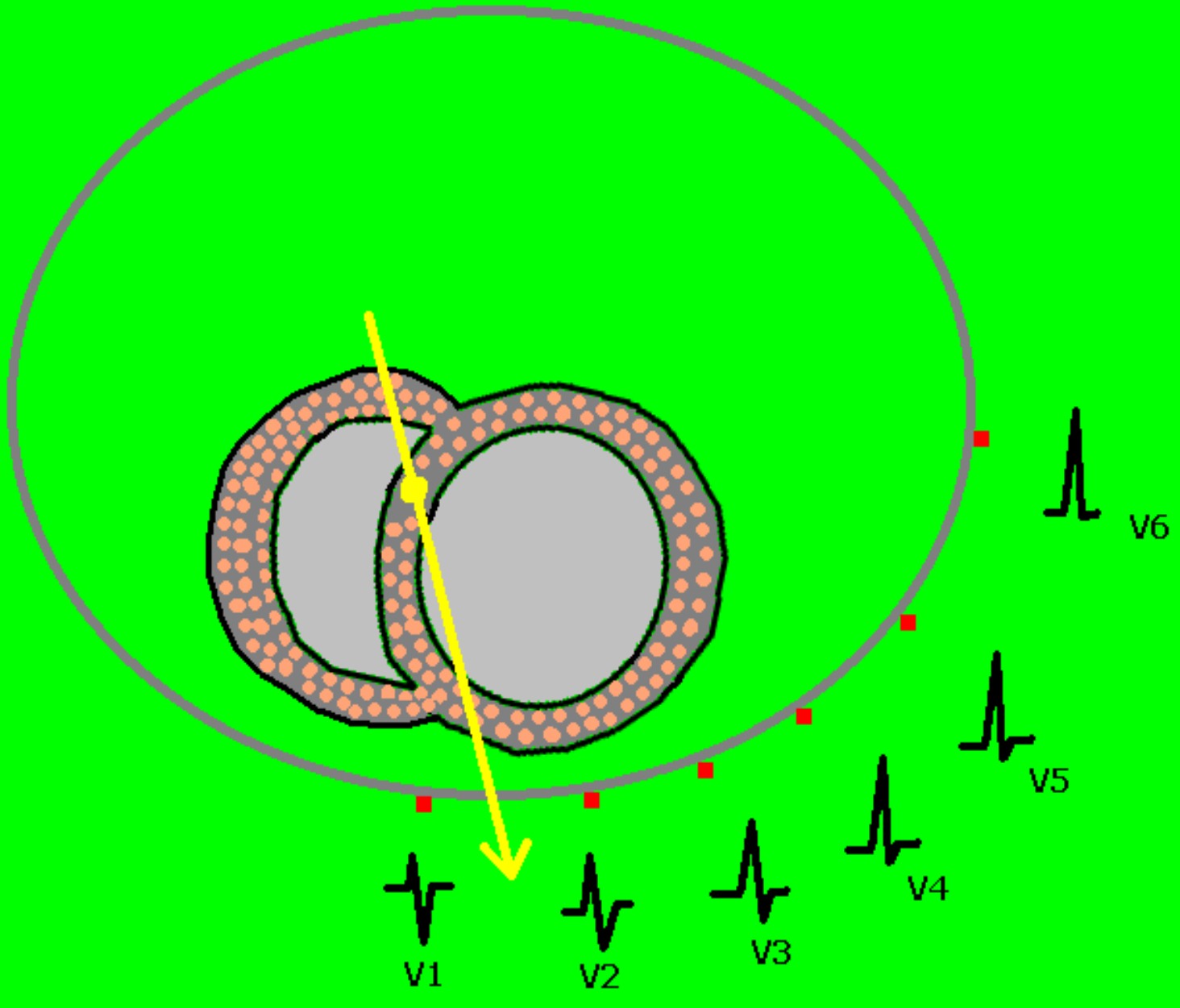
D.O.S.:



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

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

RIGHT VENTRICULAR HYPERTROPHY



Male Caucasian

Room:CCU3
Loc:1 Option:1

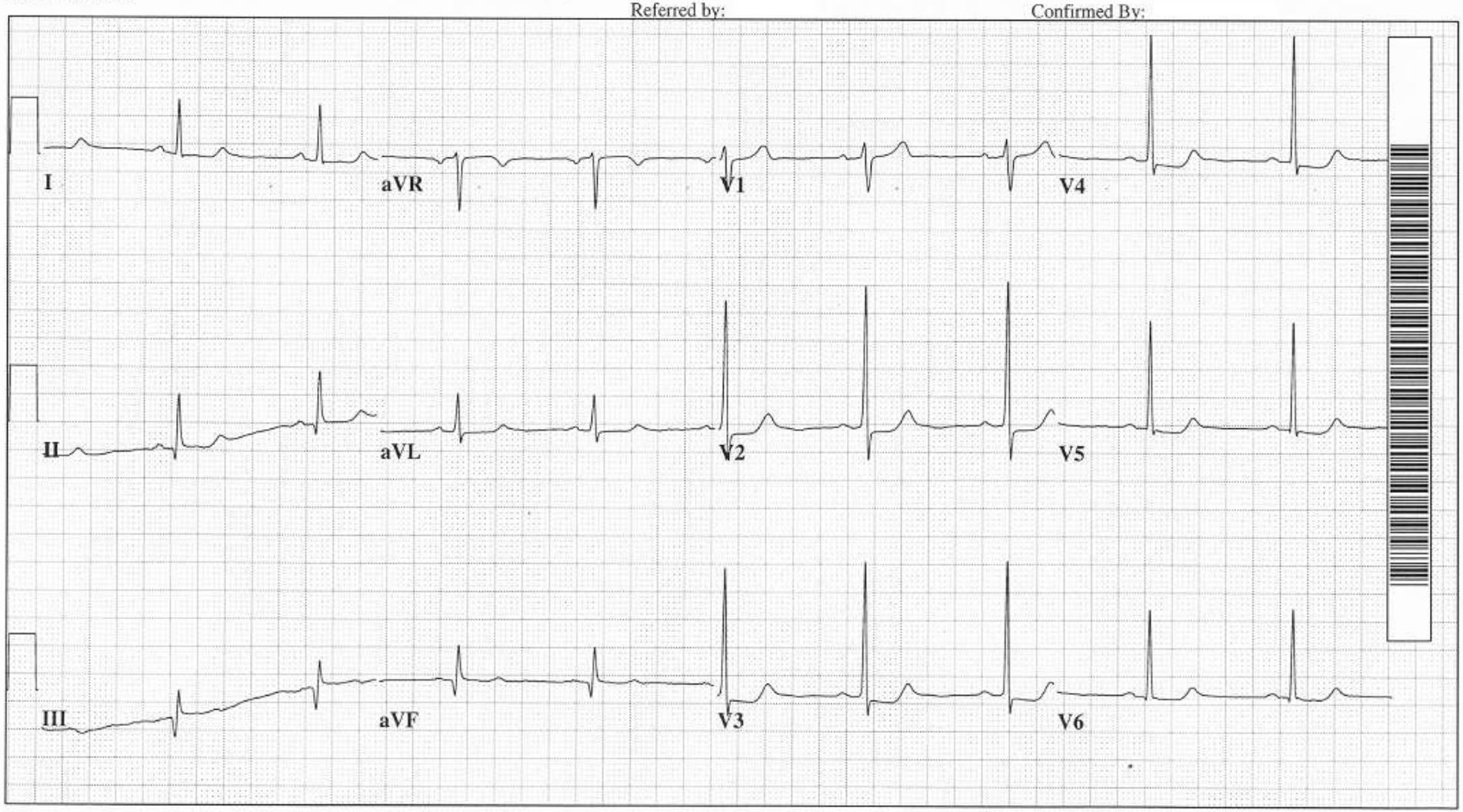
Vent. rate	58	BPM
PR interval	168	ms
QRS duration	84	ms
QT/QTc	424/416	ms
P–R–T axes	18 28	29

What is the cause of EARLY TRANSITION in this EKG ?

Technician ID: EKG CLASS #WR03602216

Med: Unknown

Referred by: Confirmed By:



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

EID:18 EDT: 21:53 06–MAY–1997 ORDER:

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

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

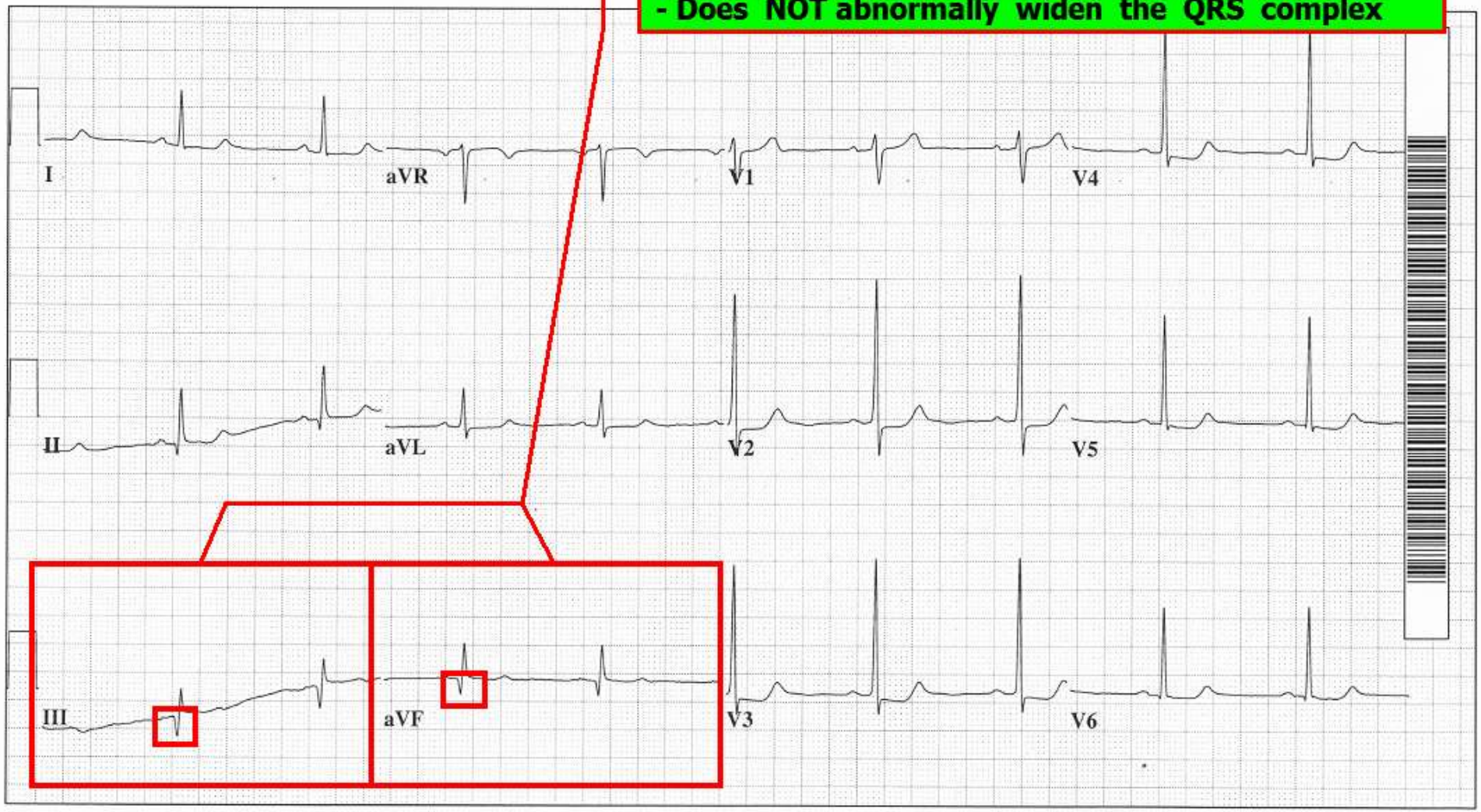
Sinus bradycardia
Inferior–posterior infarct (cited on or before 27–APR–1997)
~~Abnormal ECG~~
When compared with ECG of 30–APR–1997 13:39,
No significant change was found

Technician ID: EKG CLASS #WR03602216

Med: Unknown

Old Posterior Wall MI

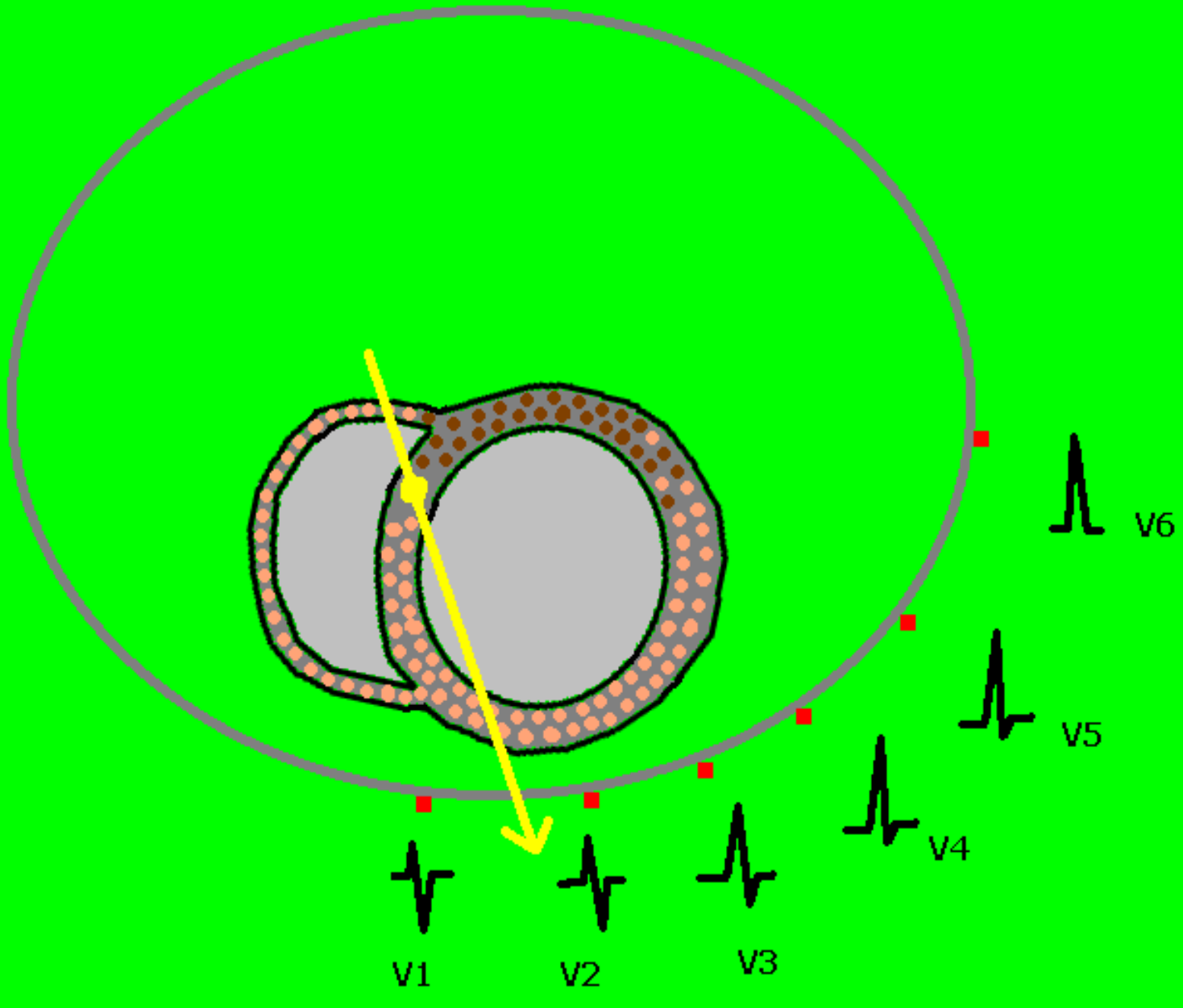
→ **Usually accompanied by OLD INFERIOR WALL MI**
- Does NOT abnormally widen the QRS complex



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

EID:18 EDT: 21:53 06–MAY–1997 ORDER:

OLD POSTERIOR WALL M.I.



01-MAY- 04:14:17

51 yr
Male Caucasian

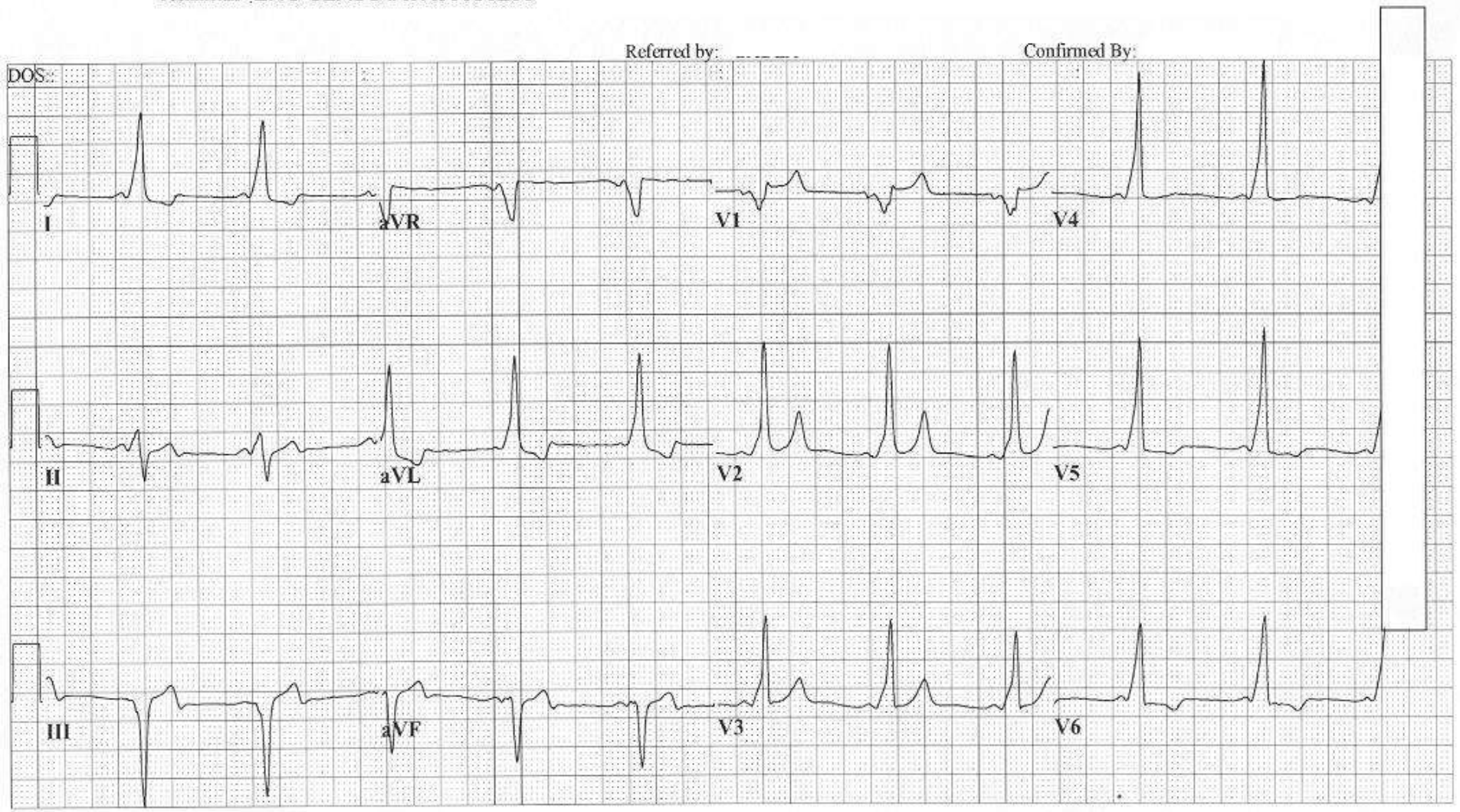
Room:540
Loc:5 Option:28

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:



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

EID:17 EDT: 08:49 01-MAY-1999 ORDER:

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

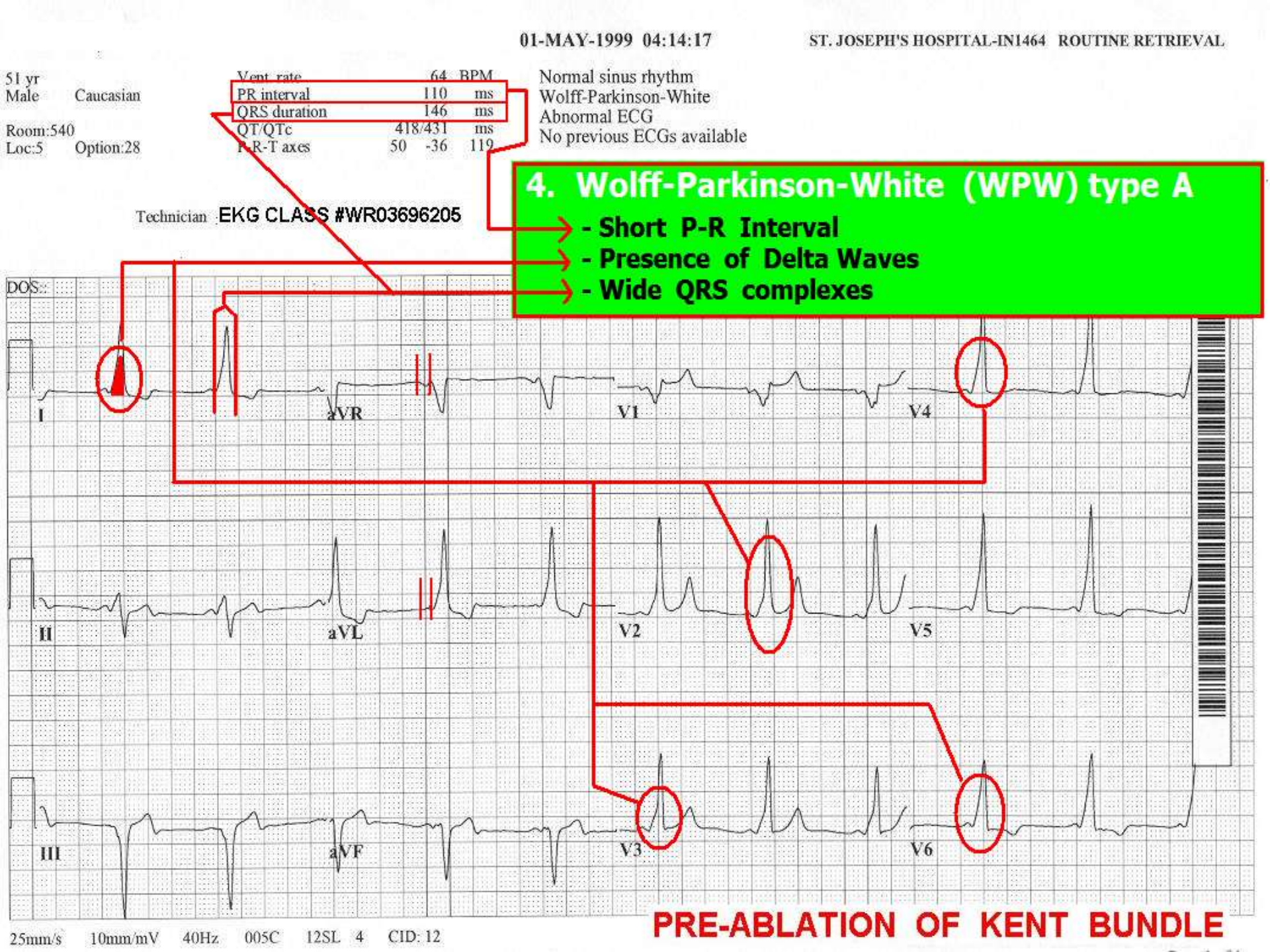
. . . . SOME HELPFUL CLUES:

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


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
DOS::
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 12
PRE-ABLATION OF KENT BUNDLE

51 yr
Male Caucasian

Room:426
Loc:5 Option:28

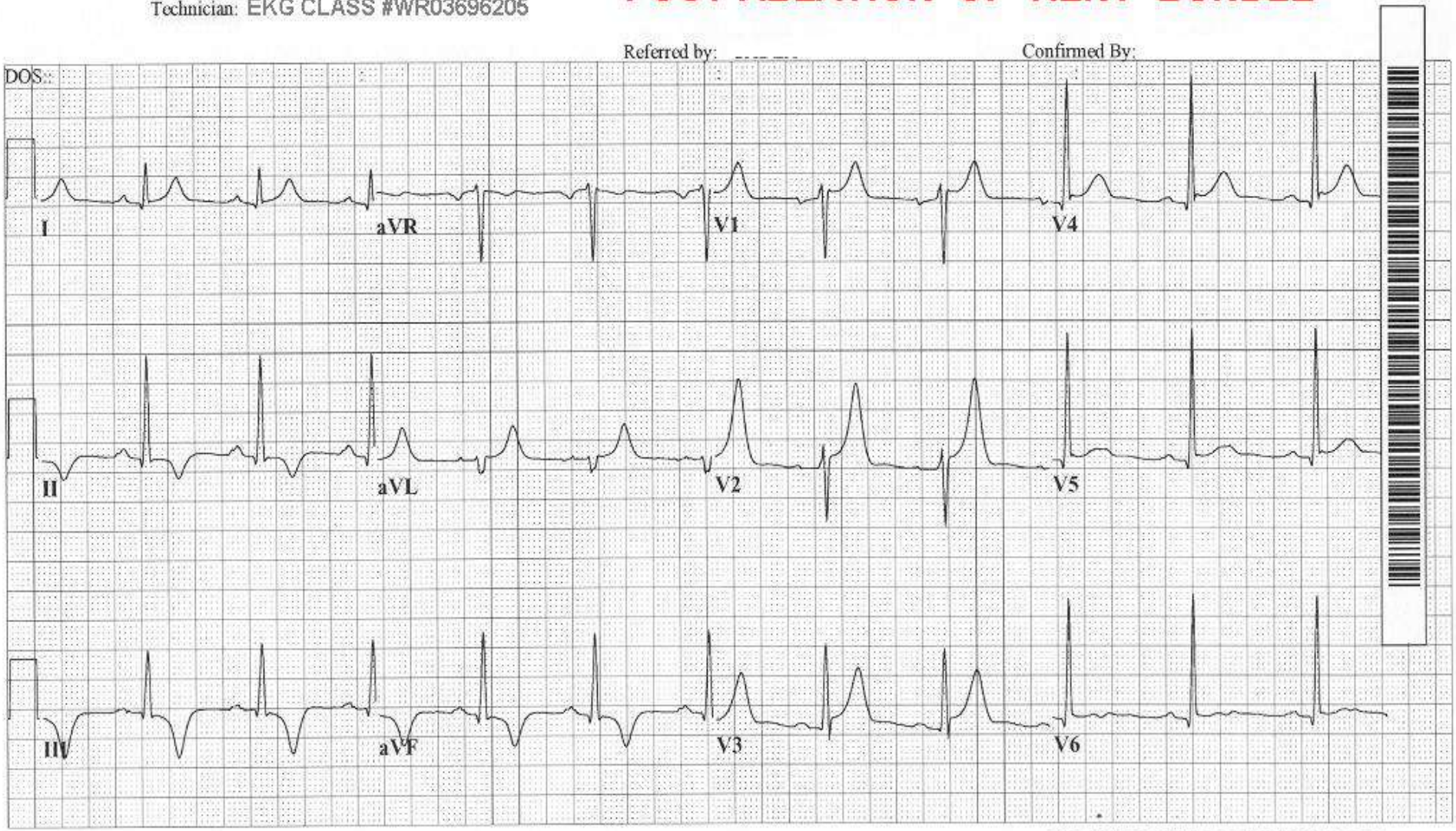
Vent. rate	69	BPM
PR interval	184	ms
QRS duration	88	ms
QT/QTc	392/420	ms
P-R-T axes	60 69	-50

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

POST-ABLATION OF KENT BUNDLE

Technician: EKG CLASS #WR03696205

Referred by: Confirmed By:

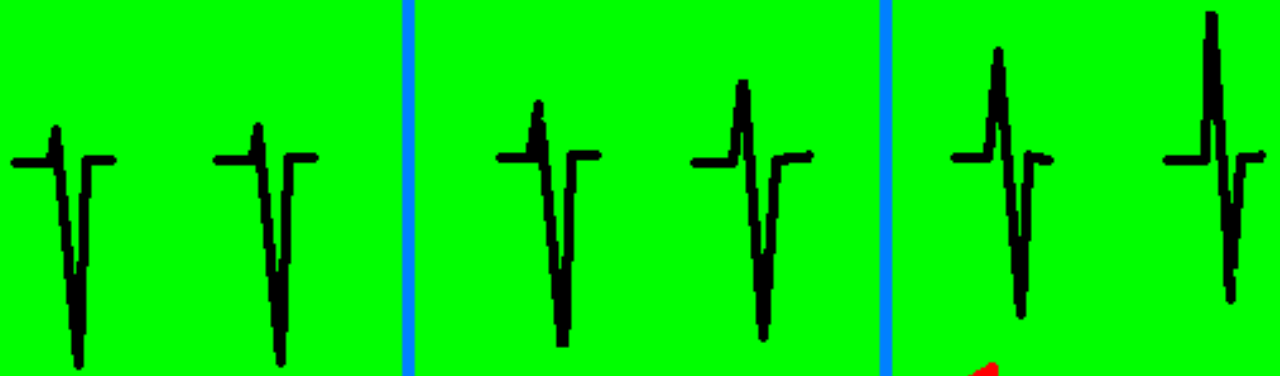


25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 12

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

AXIS ROTATION

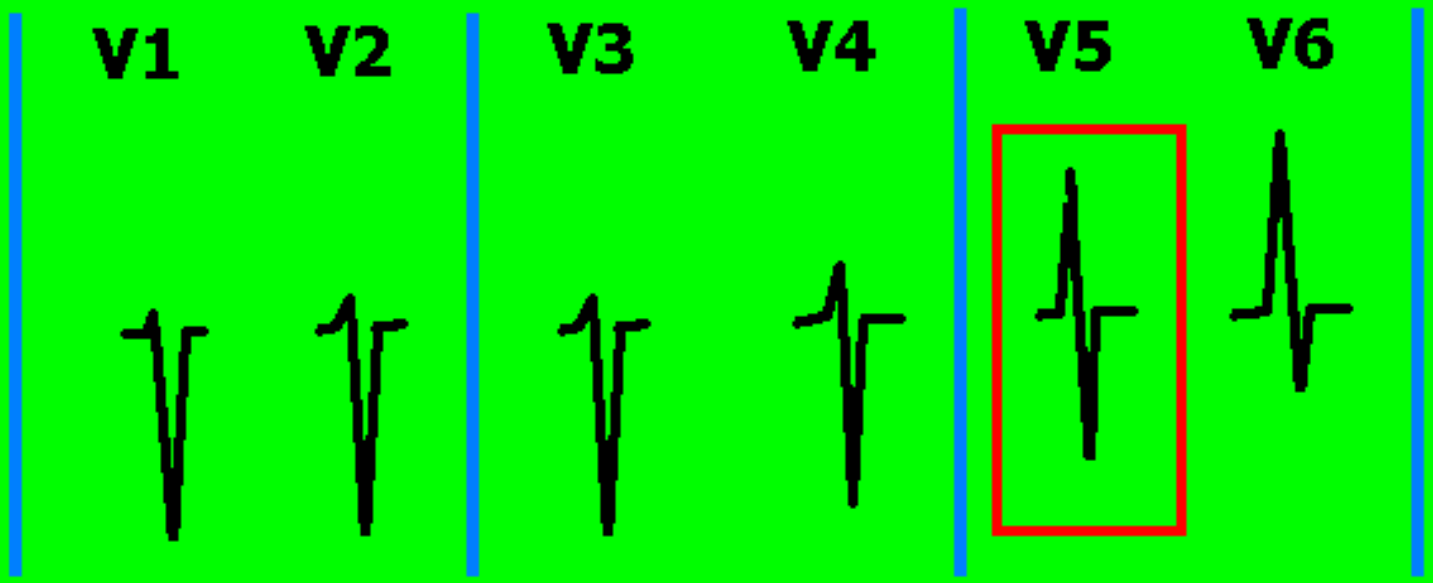
RIGHT		NORMAL		LEFT	
V1	V2	V3	V4	V5	V6



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

28-MAR-1997 05:46:00

91 yr
Female Caucasian

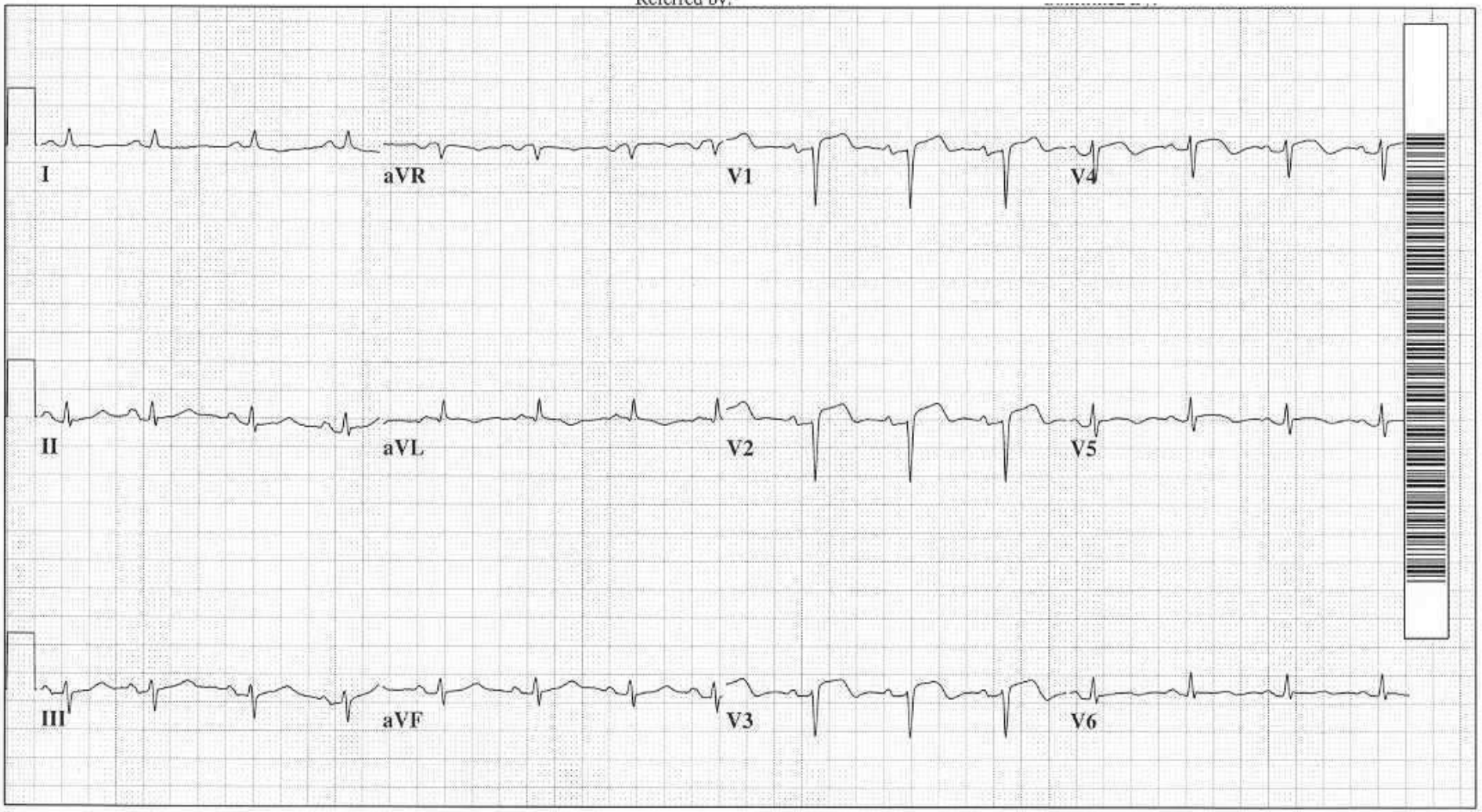
Room:3
Loc:1 Option:1

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

Technician ID: EKG CLASS # WR03110848

What is the cause of LATE TRANSITION in this EKG ?

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

91 yr
Female Caucasian

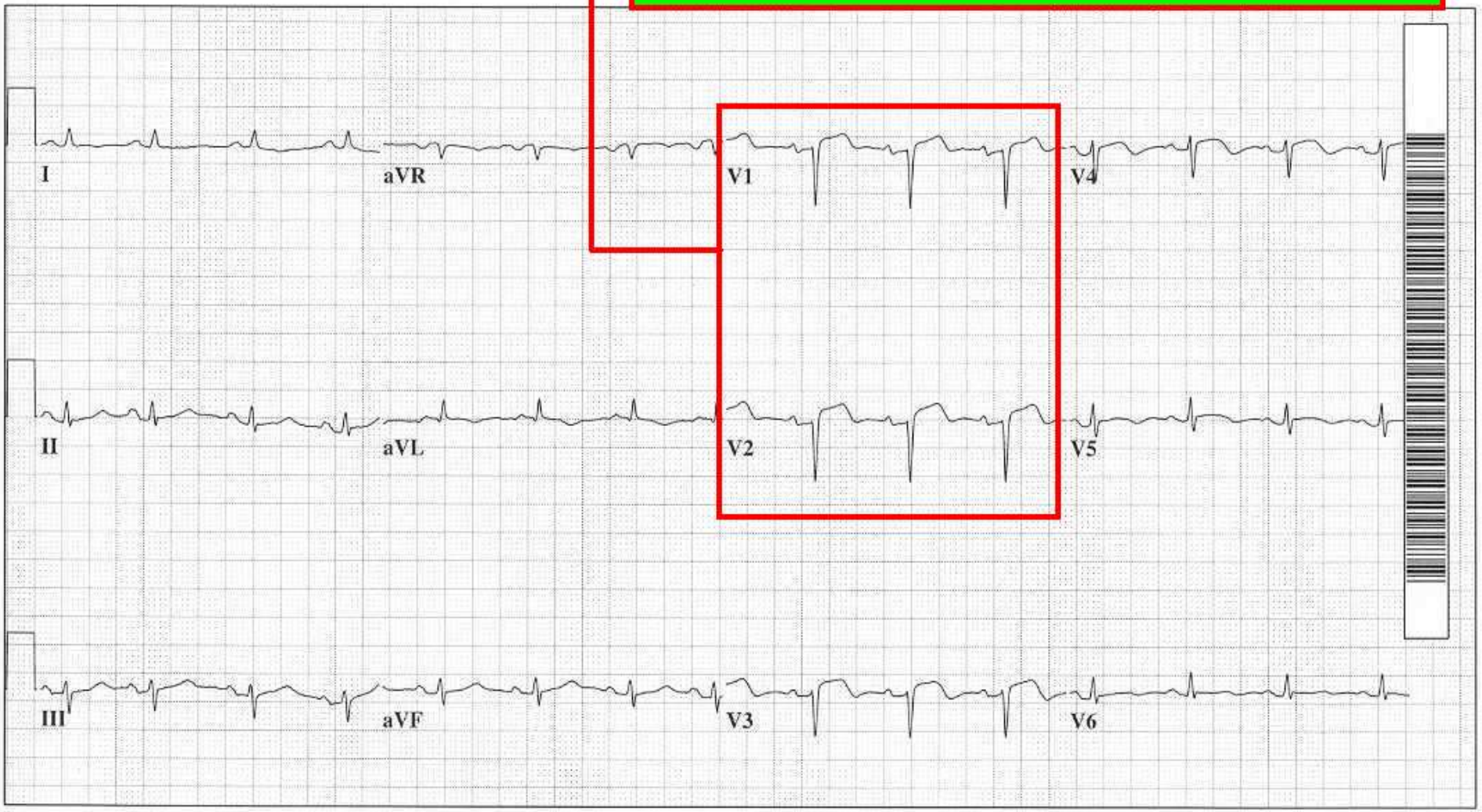
Room:3
Loc:1 Option:1

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



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

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

27-MAR-1991 13:29:00 ST. JOSEPH'S HOSPITAL-IN 65+ ROUTINE RETRIEVAL

85 yr
Female Caucasian

Room:715A
Loc:6 Option:19

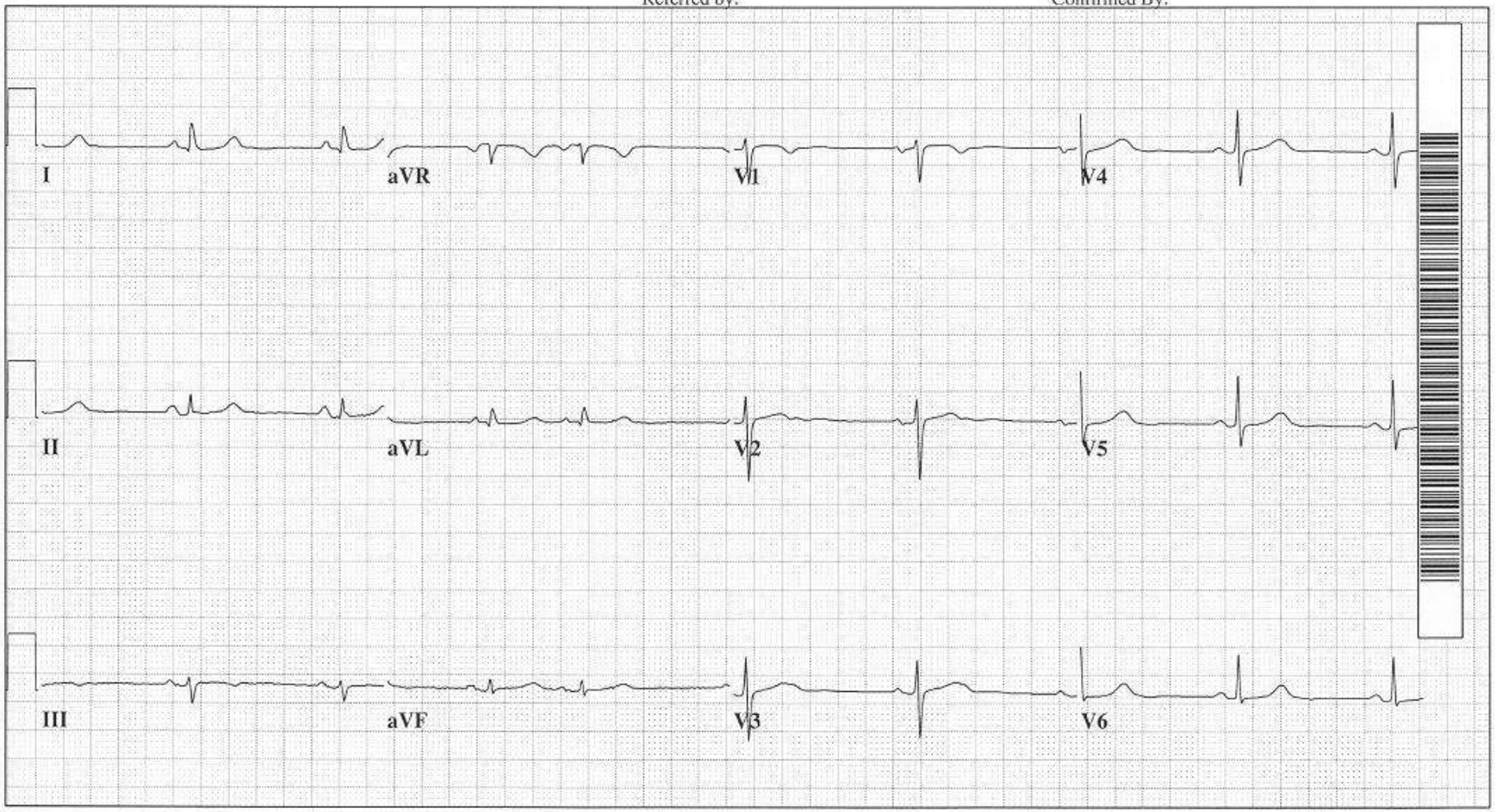
Vent. rate	55	BPM
PR interval	152	ms
QRS duration	76	ms
QT/QTc	432/413	ms
P-R-T axes	40 14	34

Sinus bradycardia with occasional Premature supraventricular complexes
Otherwise normal ECG

EKG CLASS # WR03110848

PRE-INFARCTION EKG

Referred by: Confirmed By:



25mm/s 10mm/mV 40Hz 005C 12SL 68 CID: 0

EID:Cnvrtd EDT: ORDER:

27-MAR-1997 12:42:11 ST. JOSEPH'S HOSPITAL-ER ROUTINE RETRIEVAL

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

Technician: EKG CLASS# WR03110848

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,

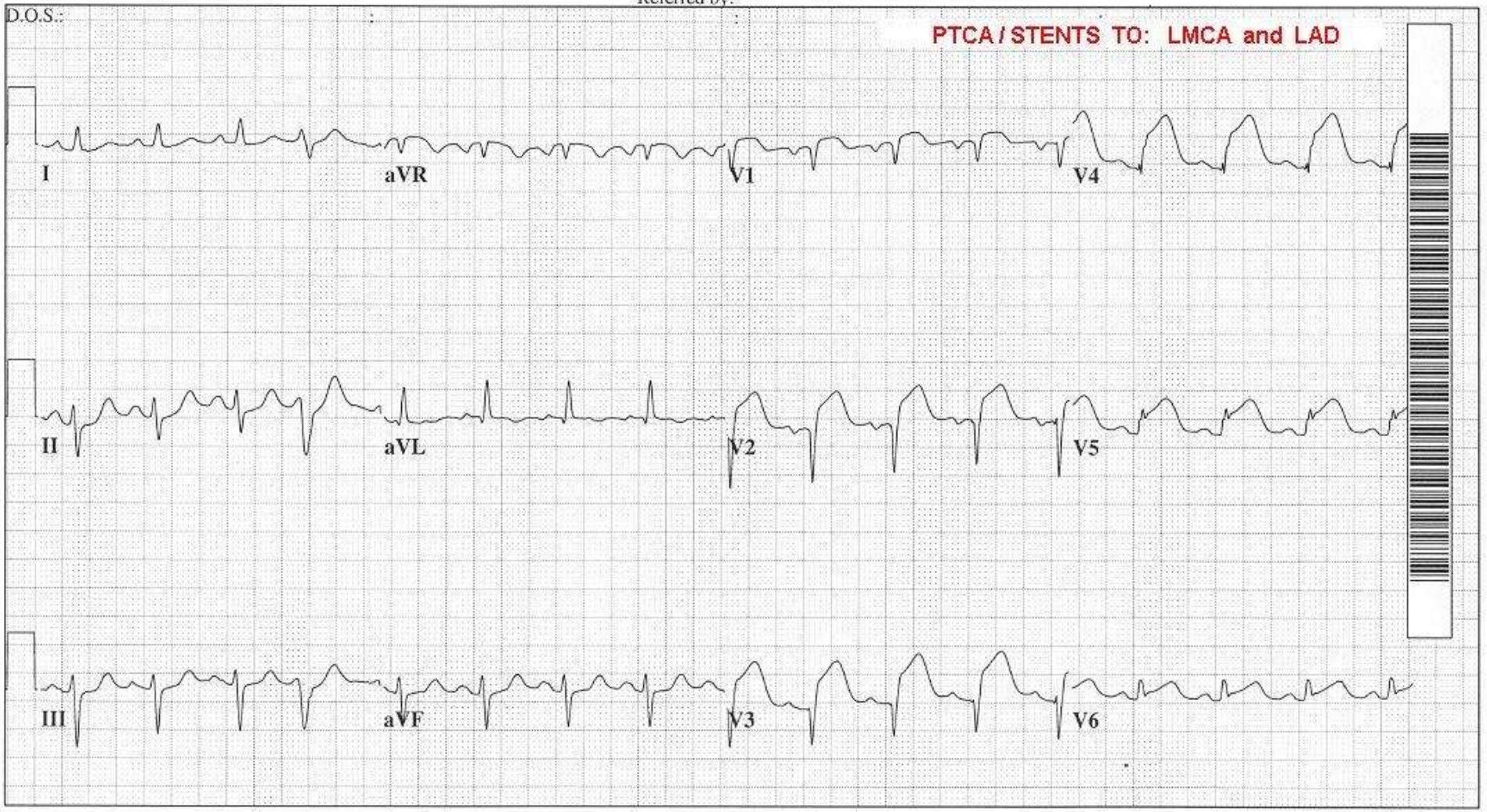
Referred by:

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

CPK: 2,471
CK/MB: 483
CK INDEX: 14

D.O.S.:

PTCA / STENTS TO: LMCA and LAD



25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 1

EID:13 EDT: 18:31 28-MAR-1997 ORDER:

28–MAR–1997 05:46:00 ST. JOSEPH'S HOSPITAL–CCU ROUTINE RETRIEVAL

91 yr
Female Caucasian

Room:3
Loc:1 Option:1

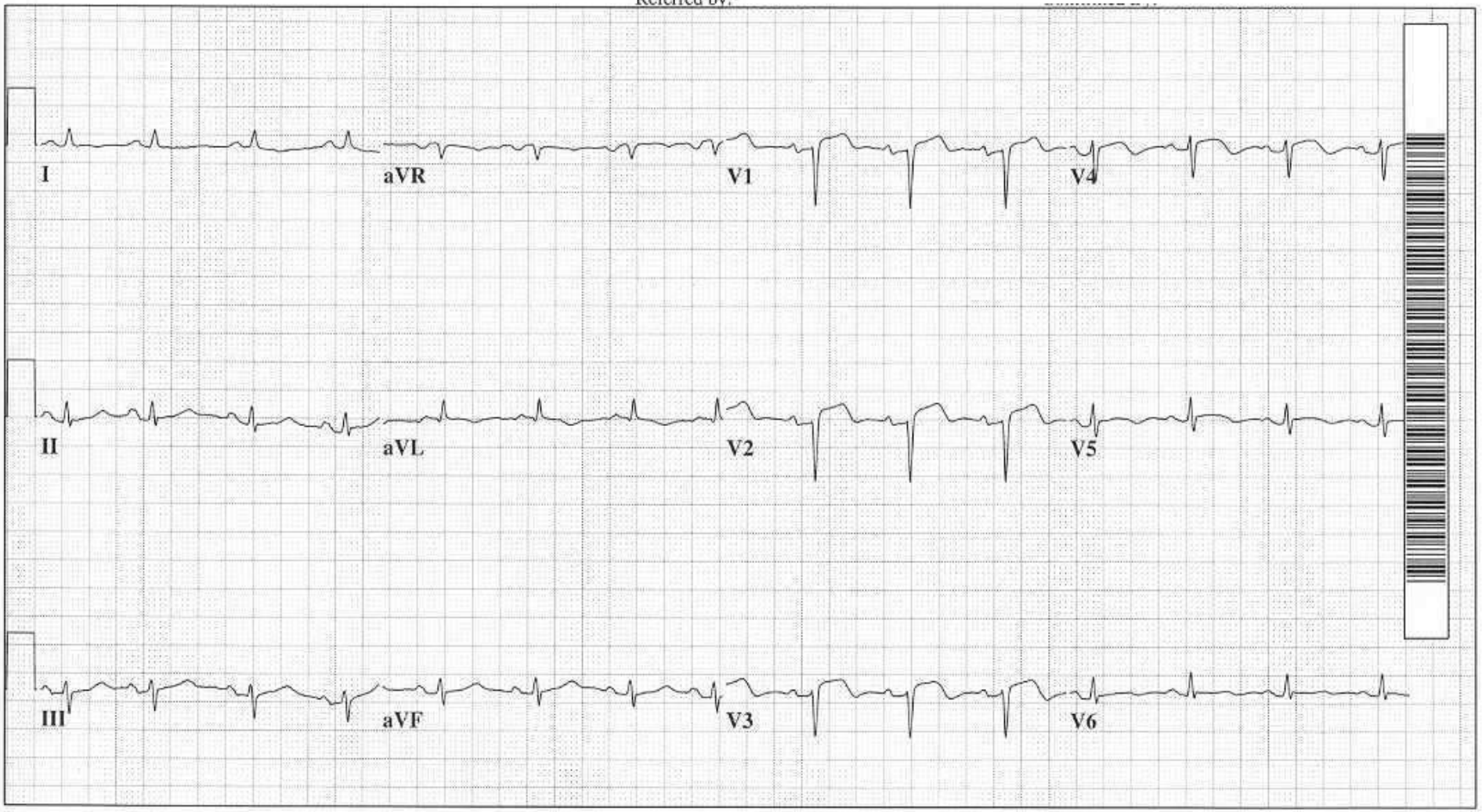
Vent. rate	87	BPM
PR interval	156	ms
QRS duration *	80	ms
QT/QTc	332/399	ms
P–R–T axes	45 4	96

Technician ID: EKG CLASS # WR03110848

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

EKG POST - INFARCTION

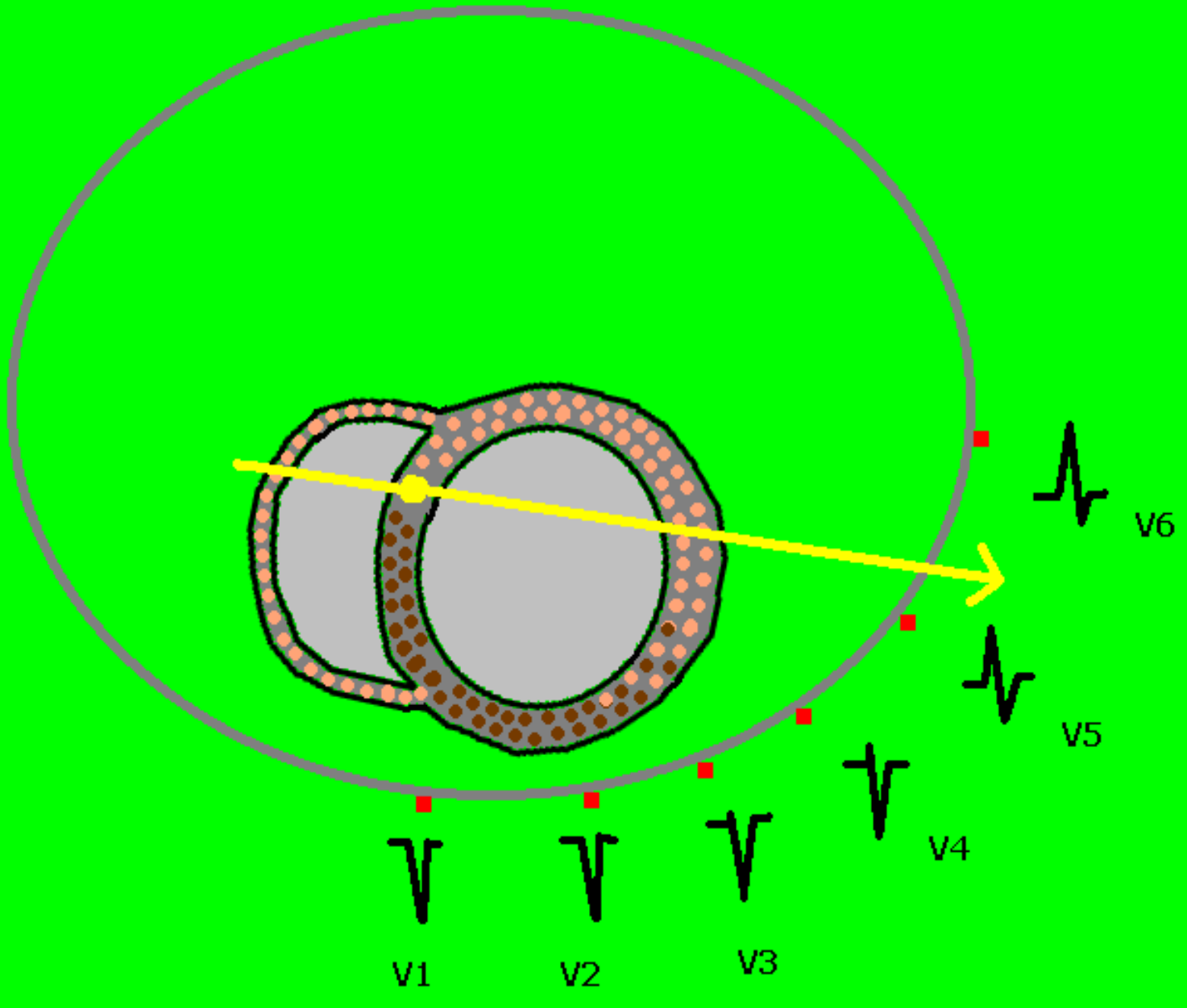
Referred by:



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

EID:16 EDT: 11:25 30–MAR–1997 ORDER:

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 <u>OLD ANTERIOR MI !</u>

- **OBTAIN A THOROUGH PATIENT HISTORY**
- **OBTAIN COPIES OF <u>OLD EKGs</u>, IF AVAILABLE**

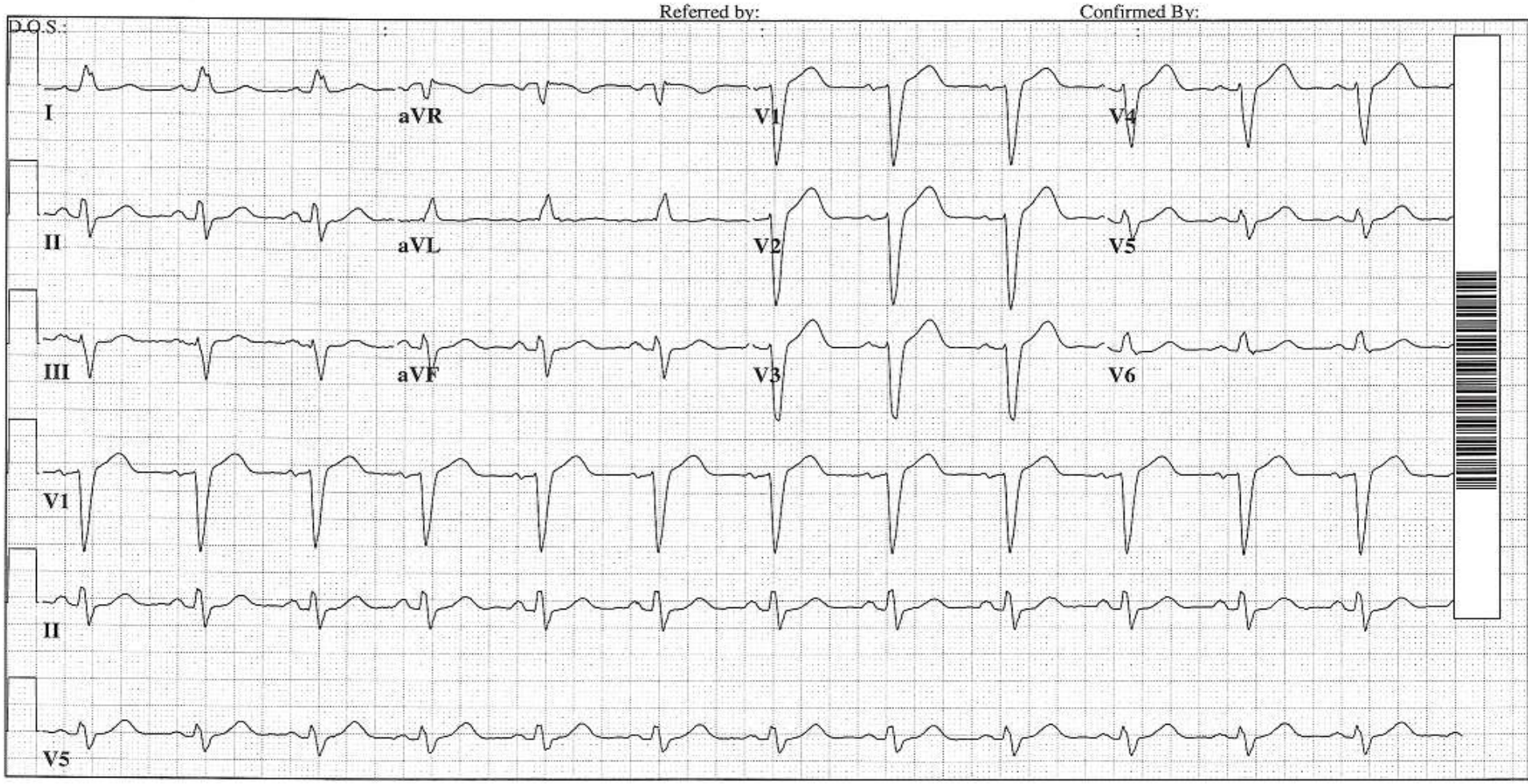
11:36:49

74 yr
Female Caucasian

Loc:7 Option:35

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

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

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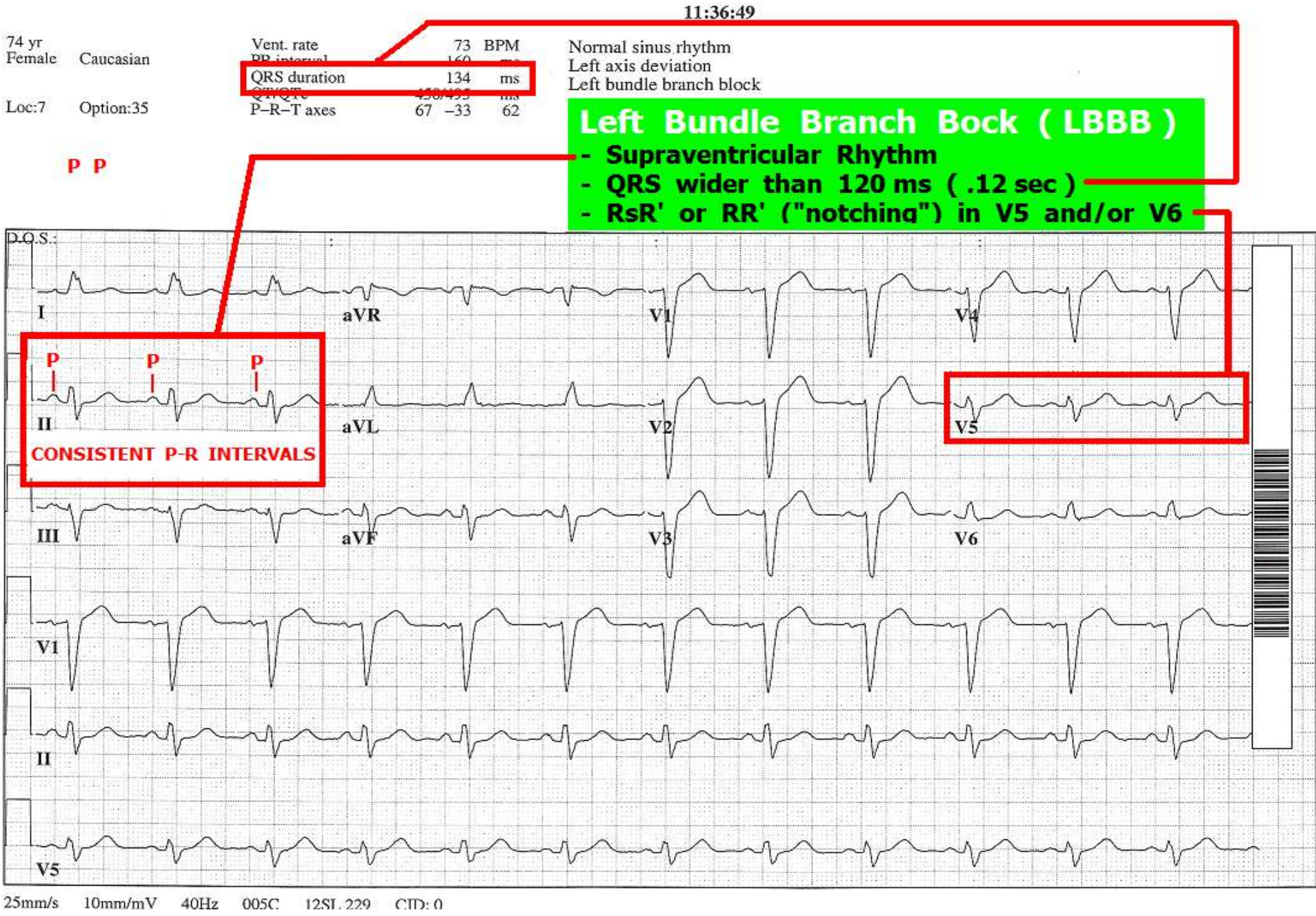

11:36:49
74 yr
Female
Caucasian
Loc:7
Option:35
Vent. rate 73 BPM
QRS duration 134 ms
P–R–T axes 67 –33 62
Normal sinus rhythm
Left axis deviation
Left bundle branch block
Left Bundle Branch Bock (LBBB)
- Supraventricular Rhythm
- QRS wider than 120 ms (.12 sec)
- RsR' or RR' ("notching") in V5 and/or V6
P P
P P P
CONSISTENT P-R INTERVALS
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 40Hz 005C 12SL 229 CID: 0

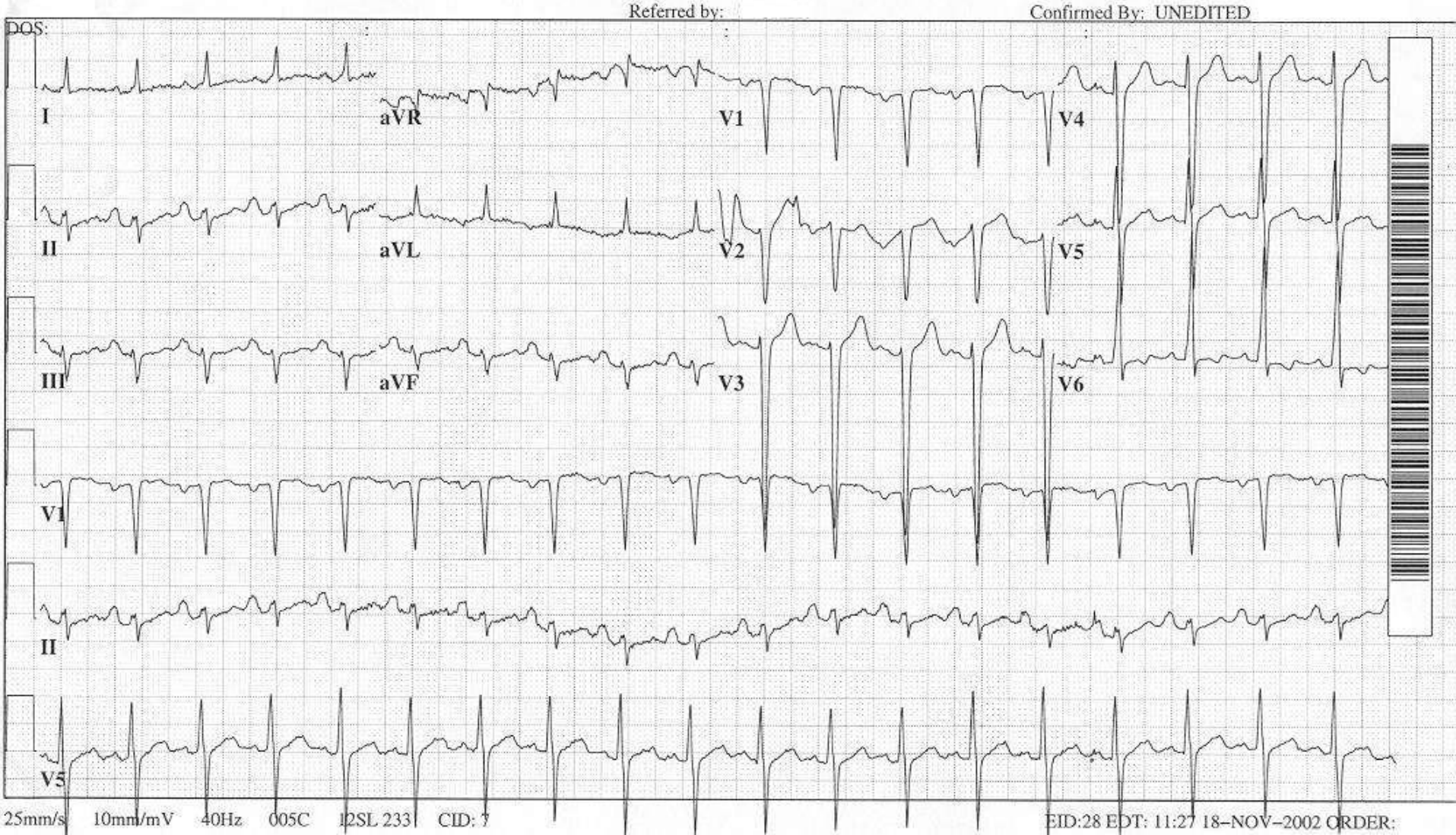
53 yr
Male Black

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

EKG CLASS #WR03896717

What is the cause of LATE TRANSITION in this EKG ?



COMMON CAUSES OF LATE TRANSITION

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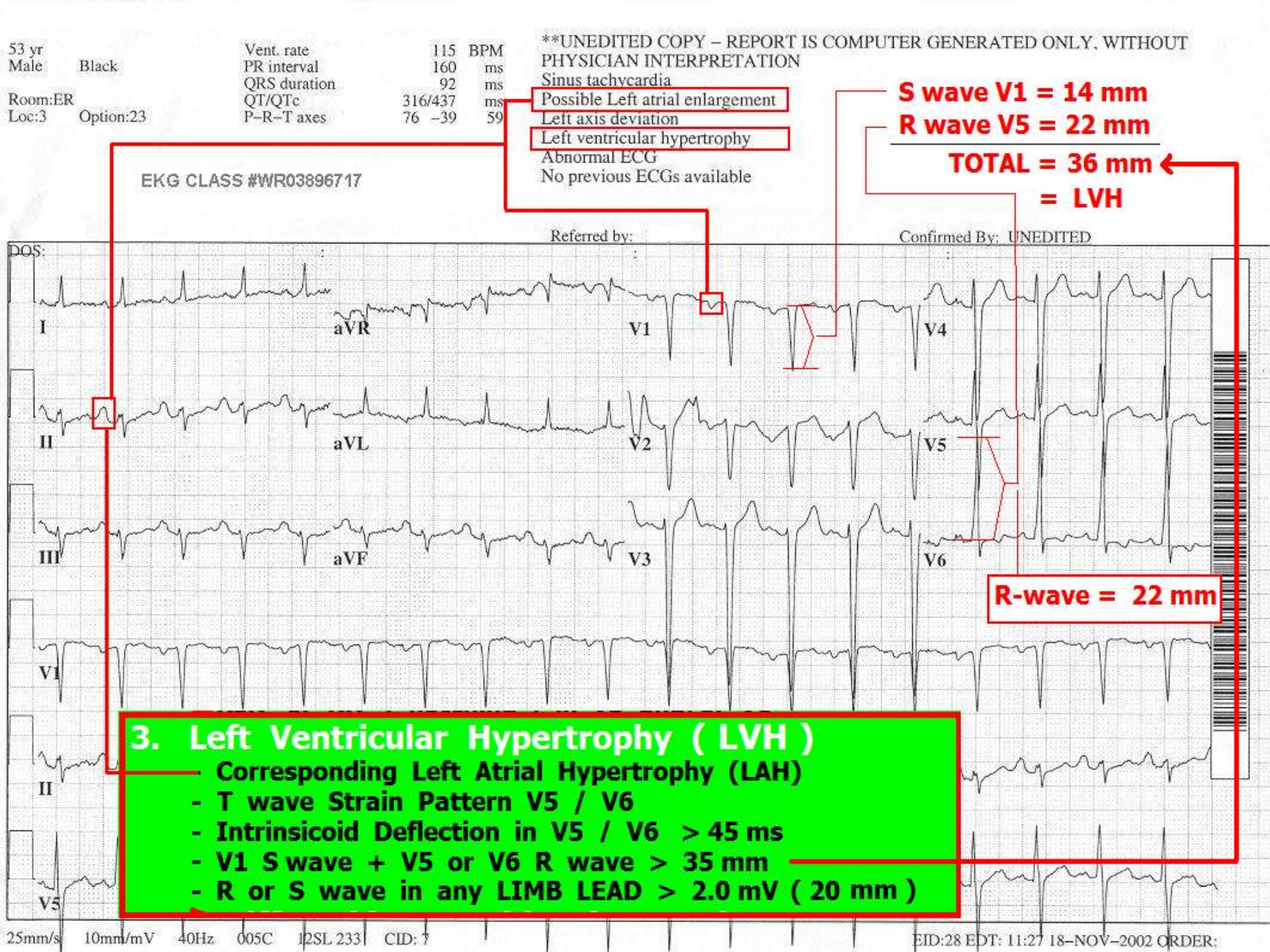
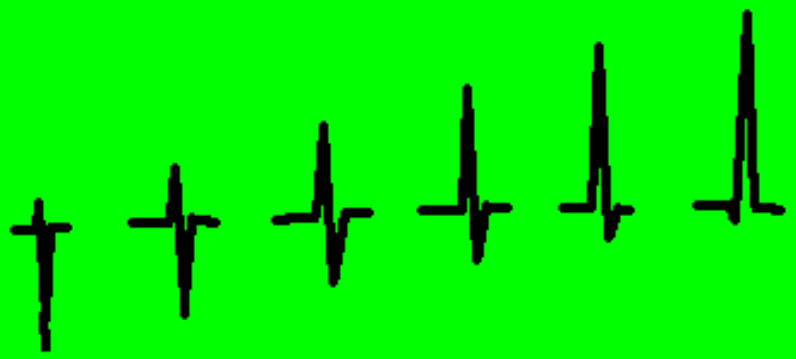
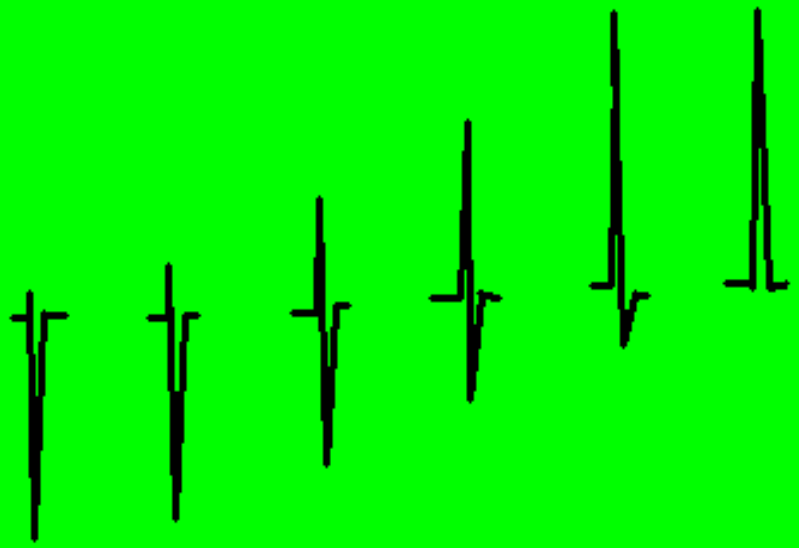

53 yr
Male Black
Room:ER
Loc:3 Option:23
Vent. rate 115 BPM
PR interval 160 ms
QRS duration 92 ms
QT/QTc 316/437 ms
P-R-T axes 76 -39 59
**UNEDITED COPY – REPORT IS COMPUTER GENERATED ONLY, WITHOUT PHYSICIAN INTERPRETATION
Sinus tachycardia
Possible Left atrial enlargement
Left axis deviation
Left ventricular hypertrophy
Abnormal ECG
No previous ECGs available
EKG CLASS #WR03896717
Referred by:
Confirmed By: UNEDITED
DOS:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
S wave V1 = 14 mm
R wave V5 = 22 mm
TOTAL = 36 mm
= LVH
R-wave = 22 mm
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)
25mm/s 10mm/mV 40Hz 005C 12SL 233 CID: 7
EID:28 EDT: 11:27 18-NOV-2002 ORDER:

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



NORMAL

V1 V2 V3 V4 V5 V6



LVH

19-JUN-1995 22:39:00 ST. JOSEPH'S HOSPITAL-TCHD ROUTINE RETRIEVAL

26 yr
Male Black

Room:703A
Loc:8 Option:25

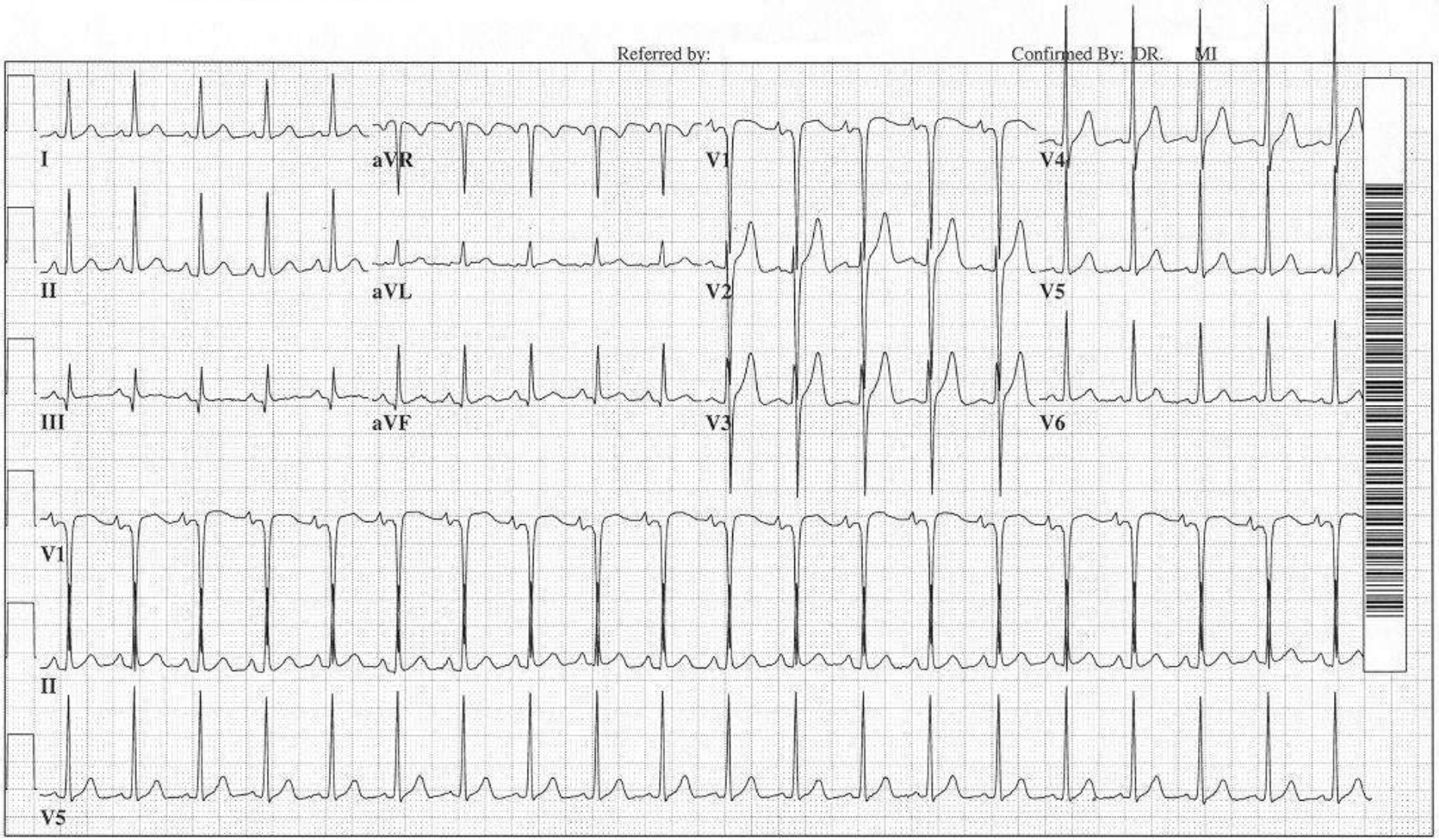
Vent. rate	119	BPM
PR interval	126	ms
QRS duration	78	ms
QT/QTc	282/397	ms
P-R-T axes	68 46	41

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

EKG CLASS #WR03446043

Referred by:

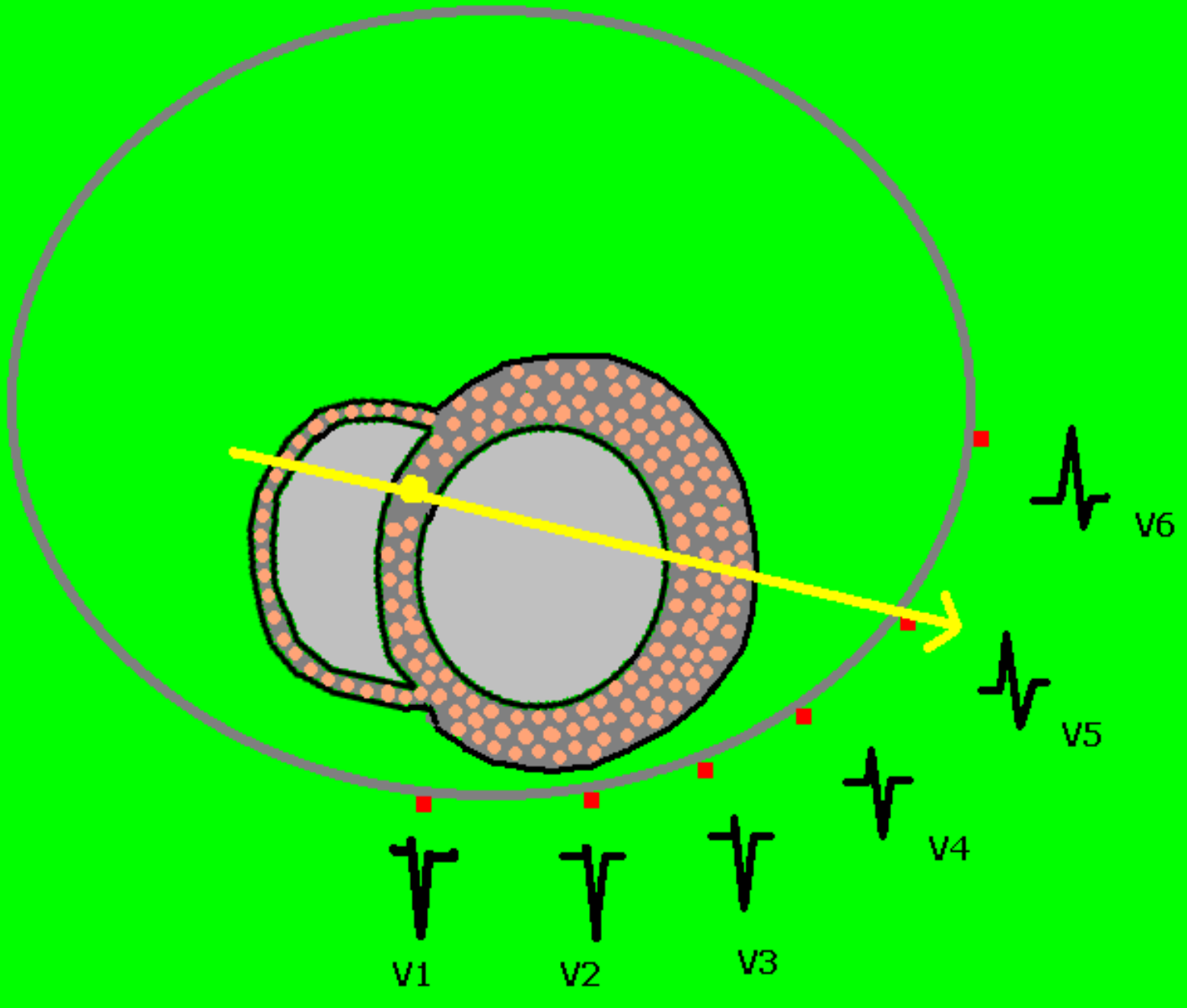
Confirmed By: DR. MI



25mm/s 10mm/mV 40Hz 005C 12SL 250 CID: 0

EID:Cnvrtd EDT: ORDER:

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

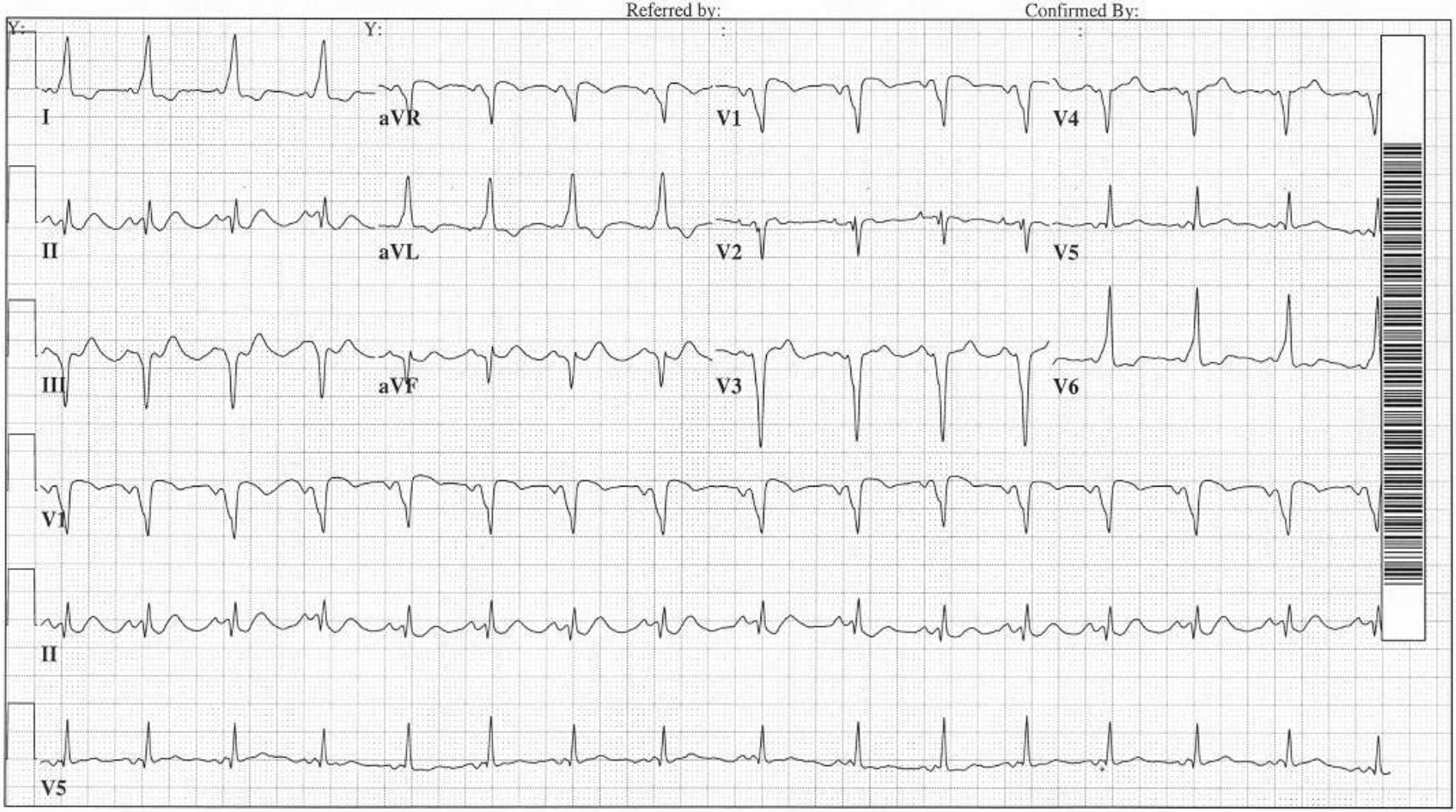
History:Unknown
Technician: DP
Test ind:EKG

EKG CLASS #WR030100
60783

what is the cause of LATE TRANSITION on this EKG ?

Referred by:

Confirmed By:



25mm/s 10mm/mV 40Hz 005C 12SL 250 CID: 12

EID:18 EDT: 16:01 17–MAY–1997 ORDER:

COMMON CAUSES OF LATE TRANSITION

. . . . WITH SOME *COMMON* HELPFUL CLUES:

1. **Old Anterior MI**
   - Q Waves in V1, V2, and /or V3
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2. **Left Bundle Branch Bock (LBBB)**
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   - QRS wider than 120 ms (.12 sec)
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   - Presence of DELTA waves
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   - Wide QRS (> 120 ms)

16 yr
Female Caucasian

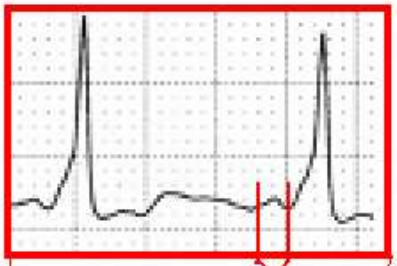
Room:REC
Loc:20 Option:50

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

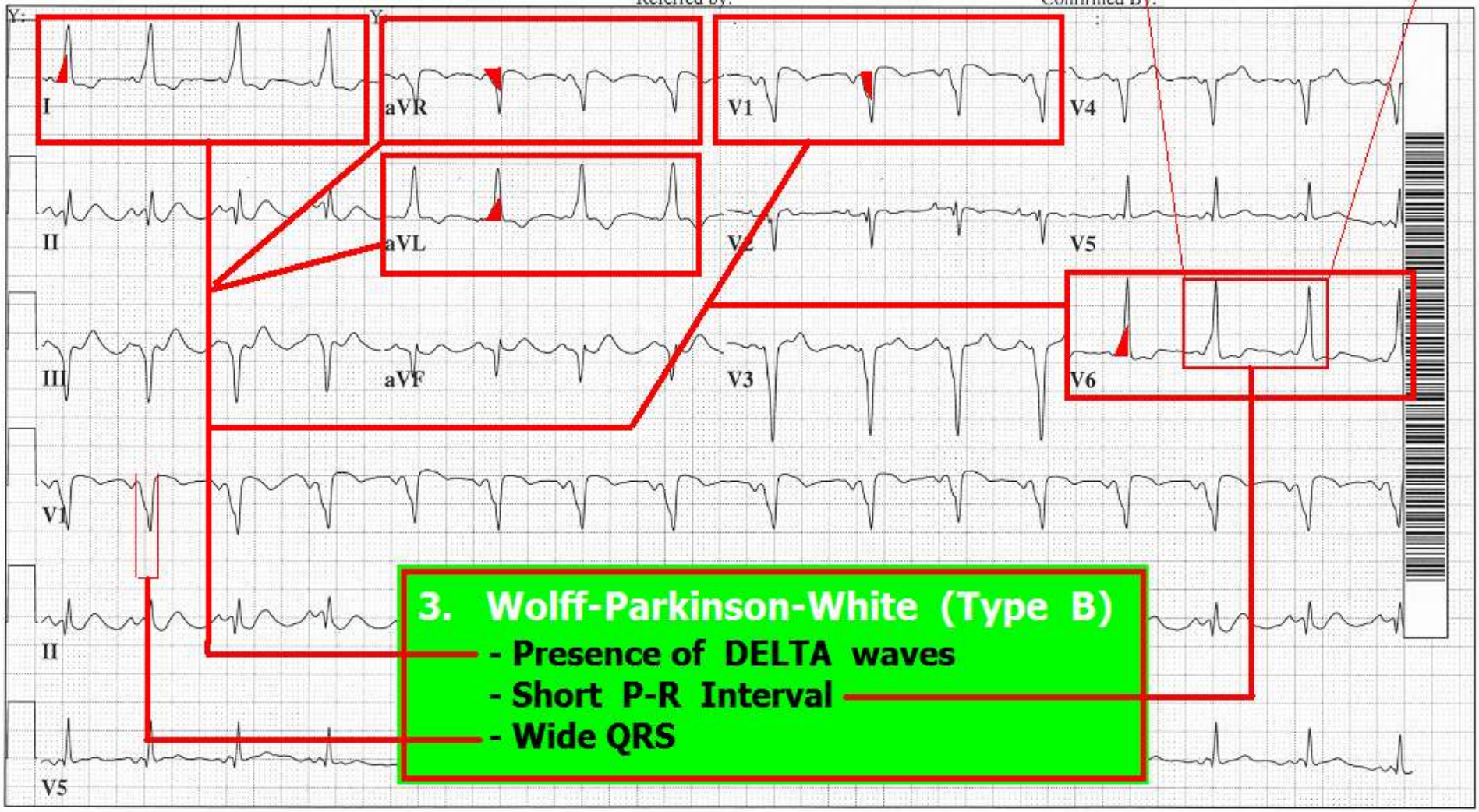
EKG CLASS #WR030100
60783



P-R = .08

Referred by:

Confirmed By:



3. Wolff-Parkinson-White (Type B)

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

25mm/s 10mm/mV 40Hz 005C 12SL 250 CID: 12

EID:18 EDT: 16:01 17-MAY-1997 ORDER:

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