MED FLEET AMBULANCE

The Lifesaving 12 Lead ECG

Part 1

Presented by:

Wayne W Ruppert, CVT, CCC, NREMT-P

Welcome !

Wayne Ruppert – Bio:

- 1978 First Responder (EMR Equivalent)
- 1980 EMT Pennsylvania
- 1982 - 1996 Paramedic (PA, NREMT, VA, FL)
- 1996 - 2012 Interventional Cardiovascular Technologist, Cardiac Cath Lab & Electrophysiology (EP) Lab
- 2012 - 2023 Cardiovascular Accreditations Regional Director, Bayfront Hosptials
- PRESENT: ALS Coordinator, MedFleet

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

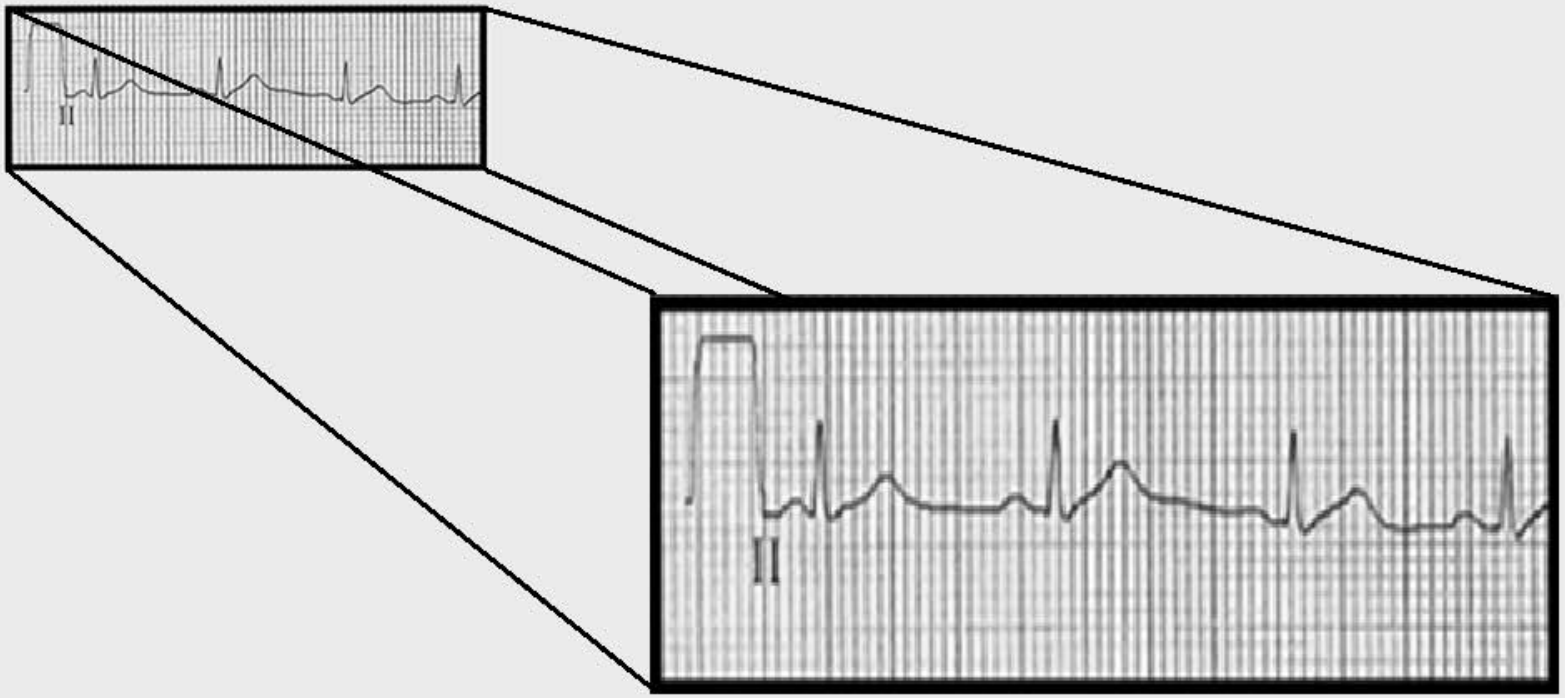
54 y/o male

CAO x 4, anxious and restless.

Clutching chest.

Skin ashen, diaphoretic, cold

C/O "susternal chest pressure" x 1 hour.



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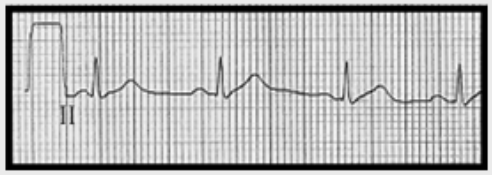
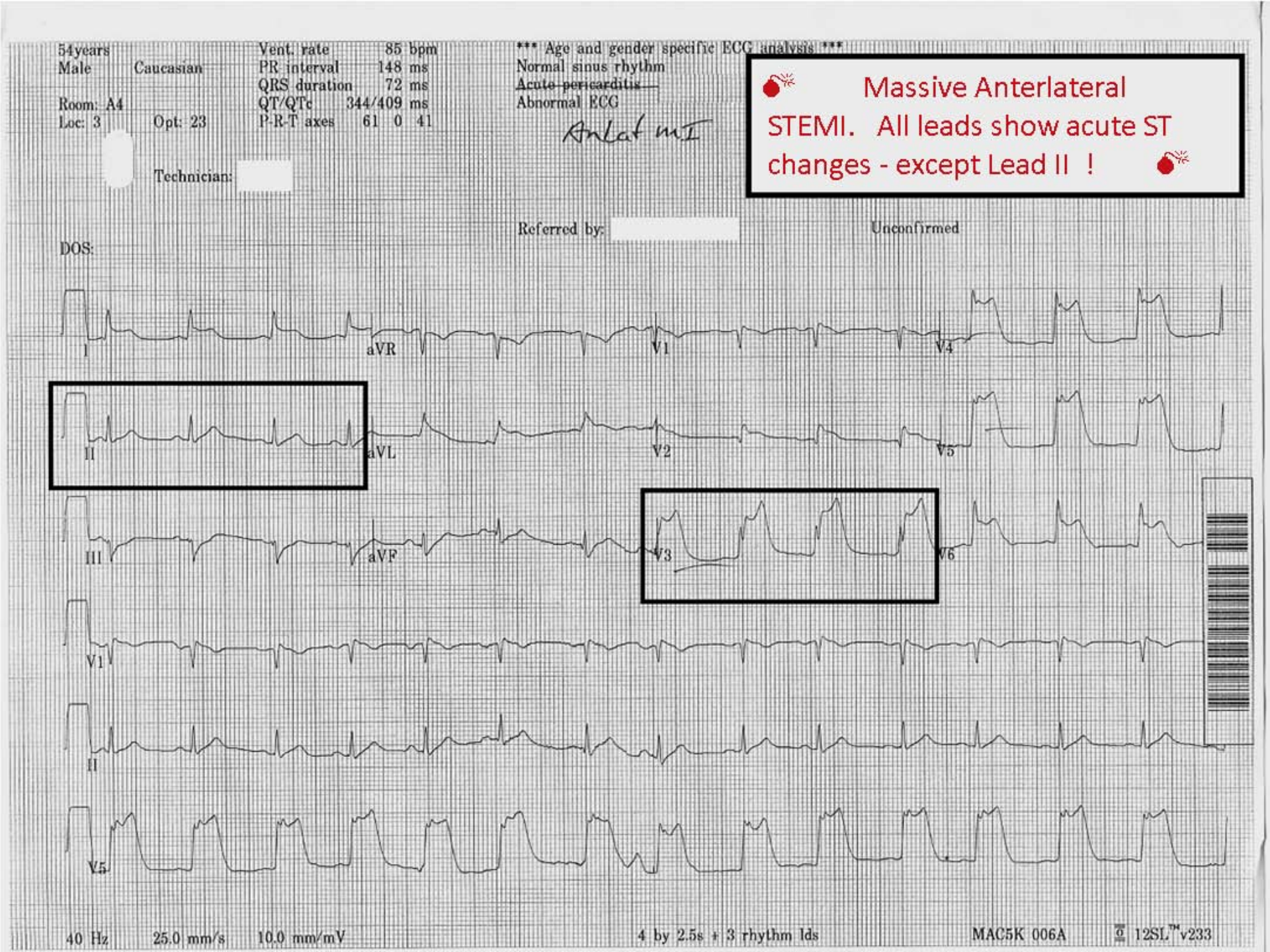



54years
Male
Caucasian
Room: A4
Loc: 3
Opt: 23
Technician:
Vent. rate 85 bpm
PR interval 148 ms
QRS duration 72 ms
QT/QTc 344/409 ms
P-R-T axes 61 0 41
*** Age and gender specific ECG analysis ***
Normal sinus rhythm
Acute pericarditis
Abnormal ECG
AnLat MI
Referred by:
Unconfirmed
DOS:
Massive Anterlateral STEMI. All leads show acute ST changes - except Lead II !
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
MAC5K 006A
12SL™v233

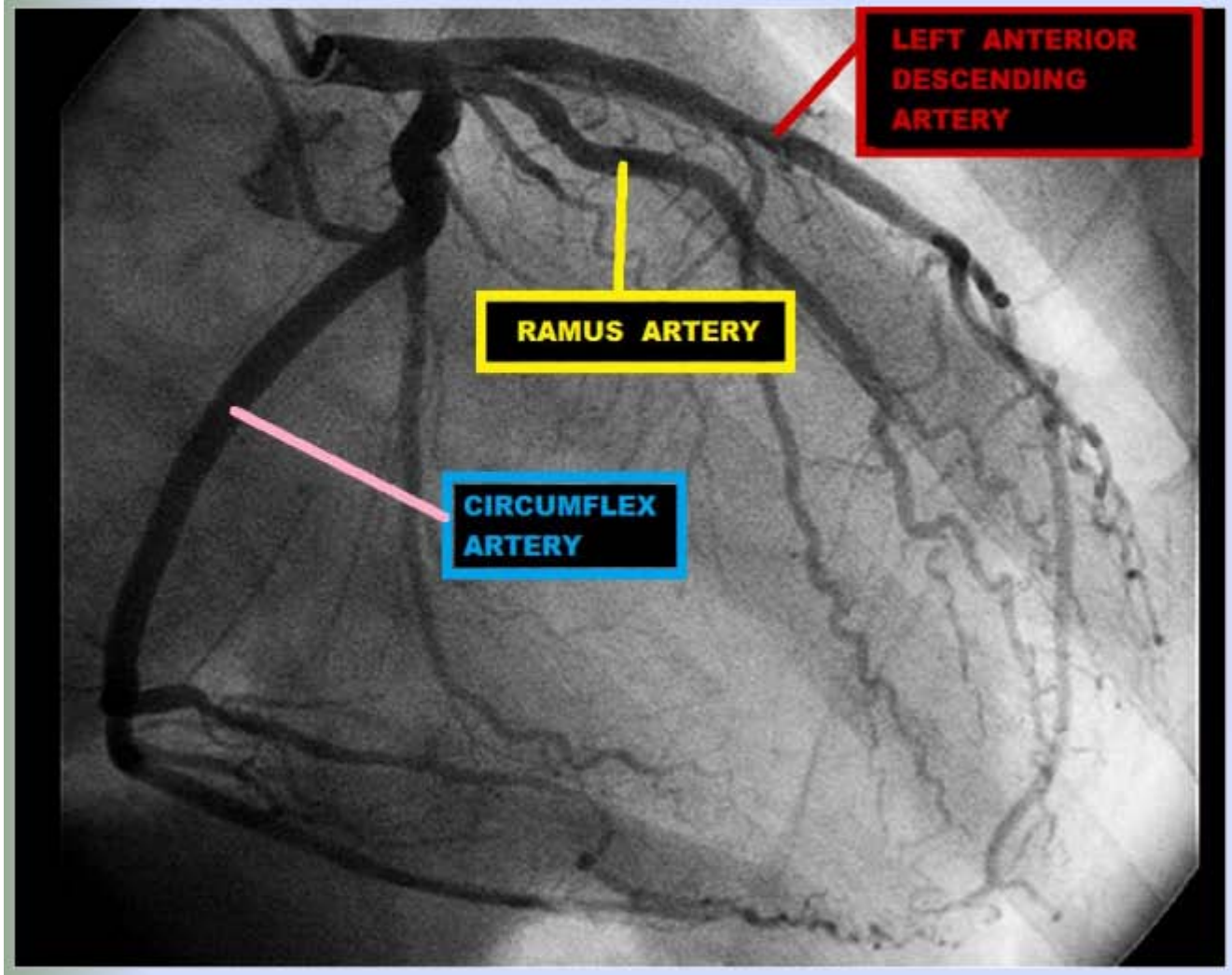
Paramedics Christ Megoulas and Wayne Ruppert, Hershey, PA Fire Department, 1982

Paramedics Christ Megoulas and Wayne Ruppert, 2024

-- both still active – (but off duty, for this pic, LOL)!

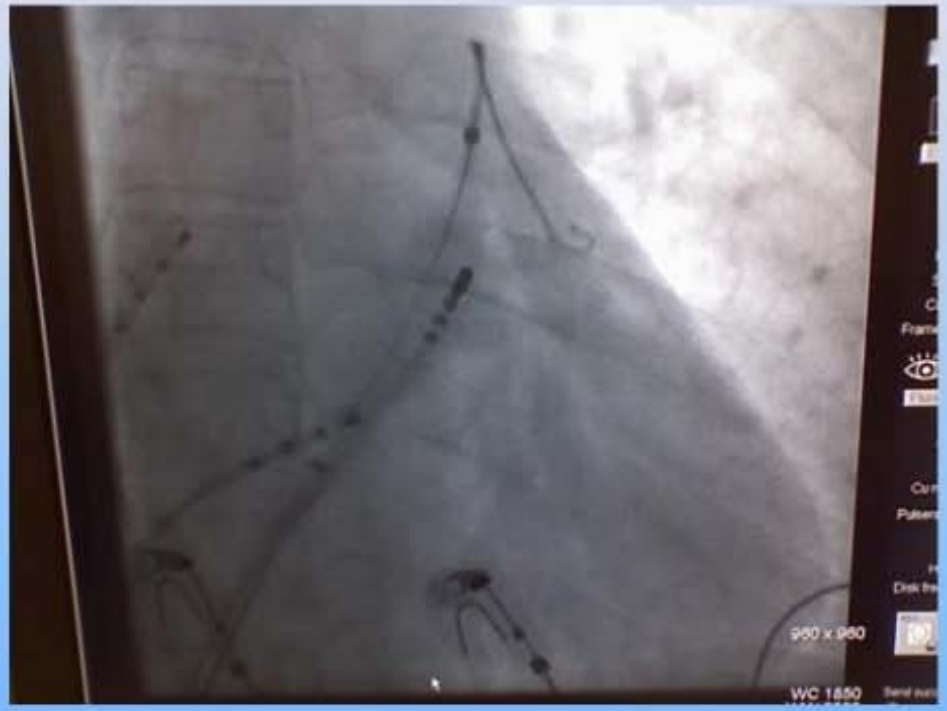

13,000 – 15,000 EP and Cath Lab cases between 1996 - Today
Wayne Ruppert and Dr. James Irwin, St Joseph's Hospital, Tampa, 7/29/2004
7.29 06:55

Cardiac Cath Lab Advantage:



Correlation of ECG leads with SPECIFIC cardiac anatomic structures.

Electrophysiology Lab Case Studies

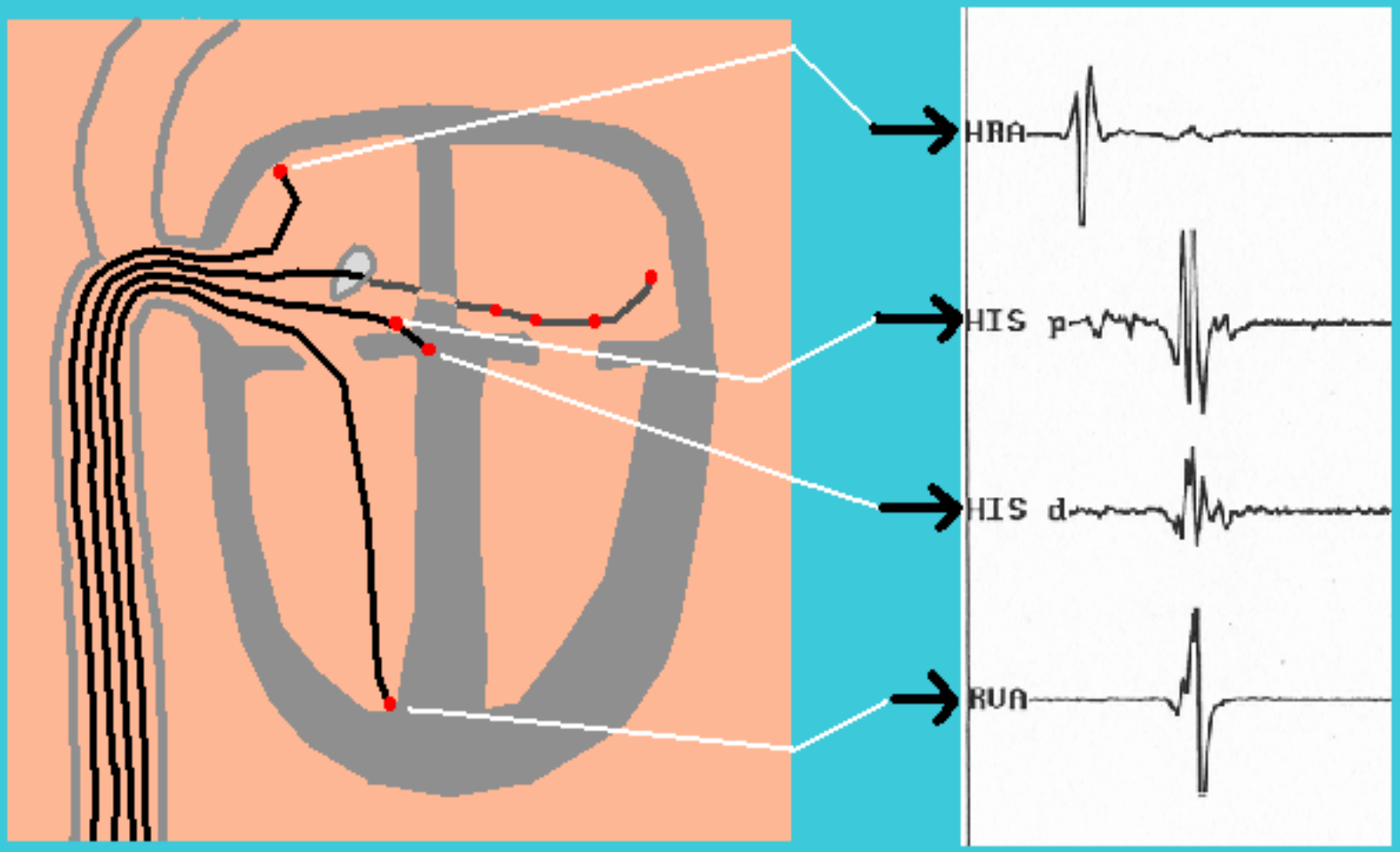


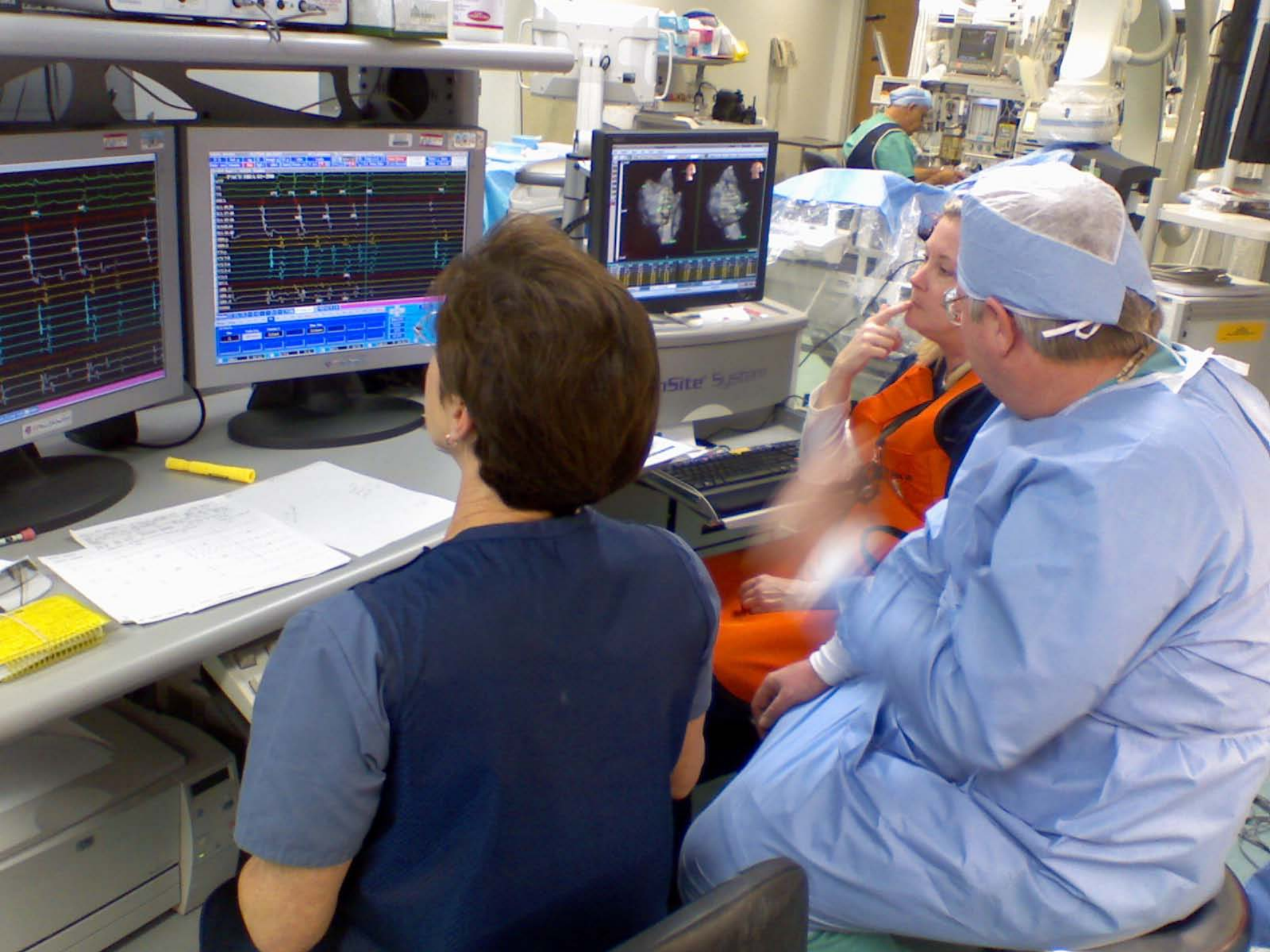
EP Catheters within the heart used for obtaining the Electrogram (the "internal ECG") Tracing and for Pace-mapping, an integral component of an EP study

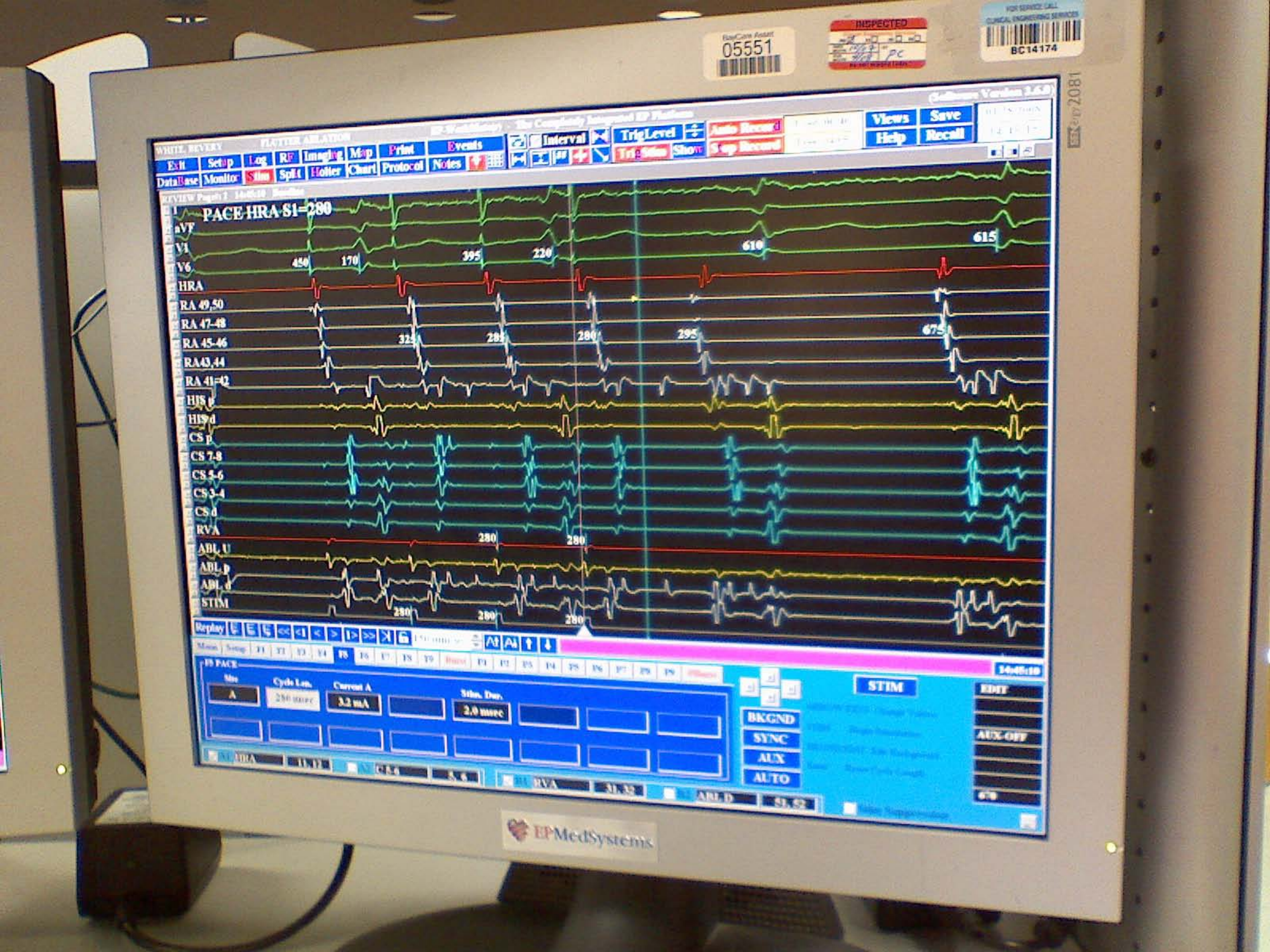
Author Wayne Ruppert conducting Pace-mapping during EP study at the St Joseph's Hospital Heart Institute, Pediatric Electrophysiology Program, Tampa, FL in 2004

THE ELECTROGRAM - THE INTERNAL EKG

THE ELECTROPHYSIOLOGY (EP) LAB



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Notes
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TrigLevel
Show
Views
Save
Help
Recall
PACE HRA S1=280
I
aVF
V1
V6
HRA
RA 49,50
RA 47-48
RA 45-46
RA43,44
RA 41-42
HIS p
HIS d
CS p
CS 7-8
CS 5-6
CS 3-4
CS d
RVA
ABL U
ABL p
ABL d
STIM
450
170
395
220
610
615
325
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675
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Cycle Len.
Current A
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A
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STIM
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BKGND
SYNC
AUX
AUTO
HRA
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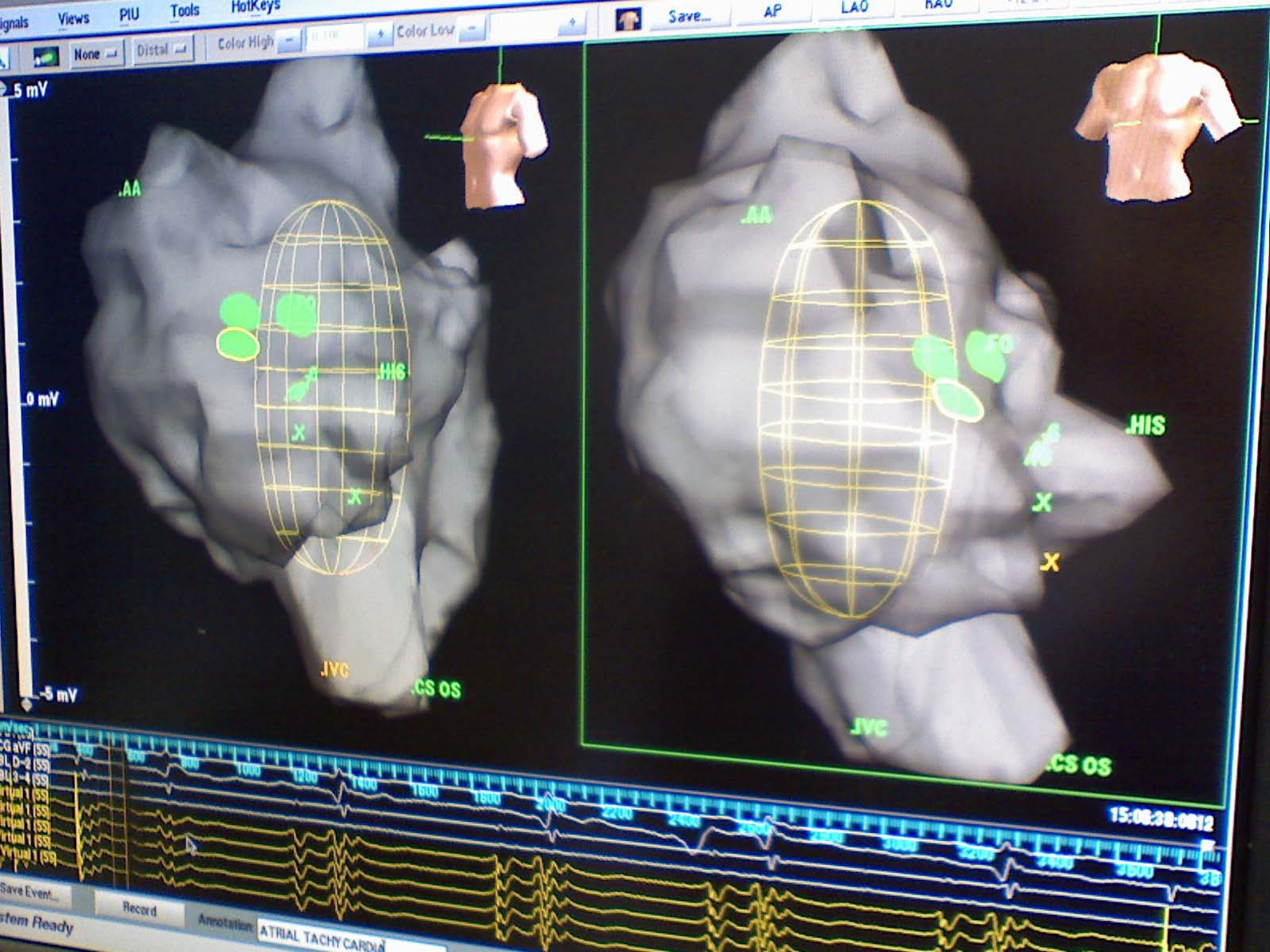
ABL D
EPMedSystems


Views
PIU
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Save...
AP
LAO
RAO
None
Distal
5 mV
0 mV
-5 mV
.AA
.HIS
.JVC
.CS OS
Save Event...
Record
Annotation:
ATRIAL TACHYCARDIA

There is NO paper handout today..

….. You can DOWNLOAD this full PowerPoint and many other ECG resources at:

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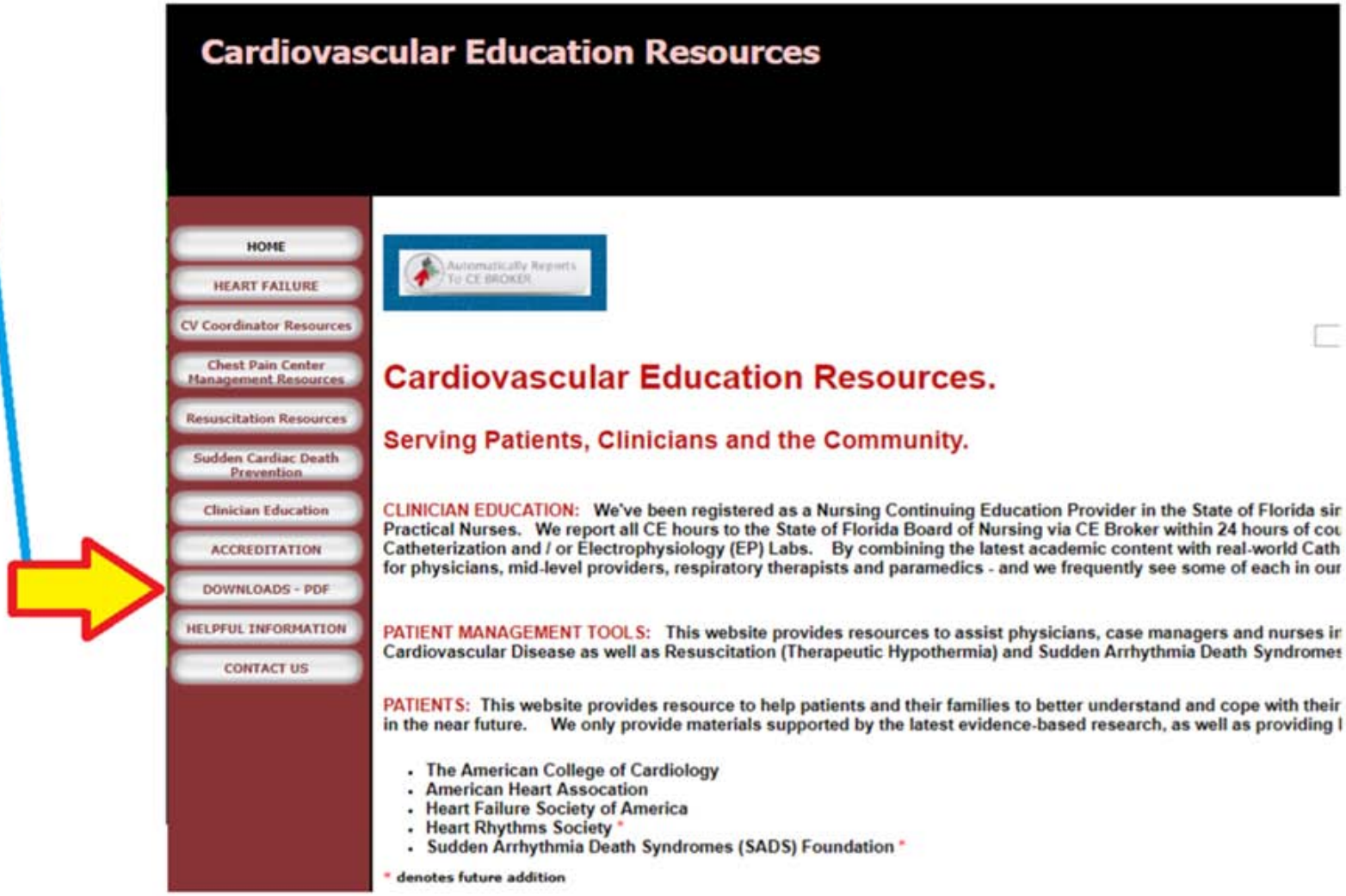
2. Select "Downloads PDF" from menu bar



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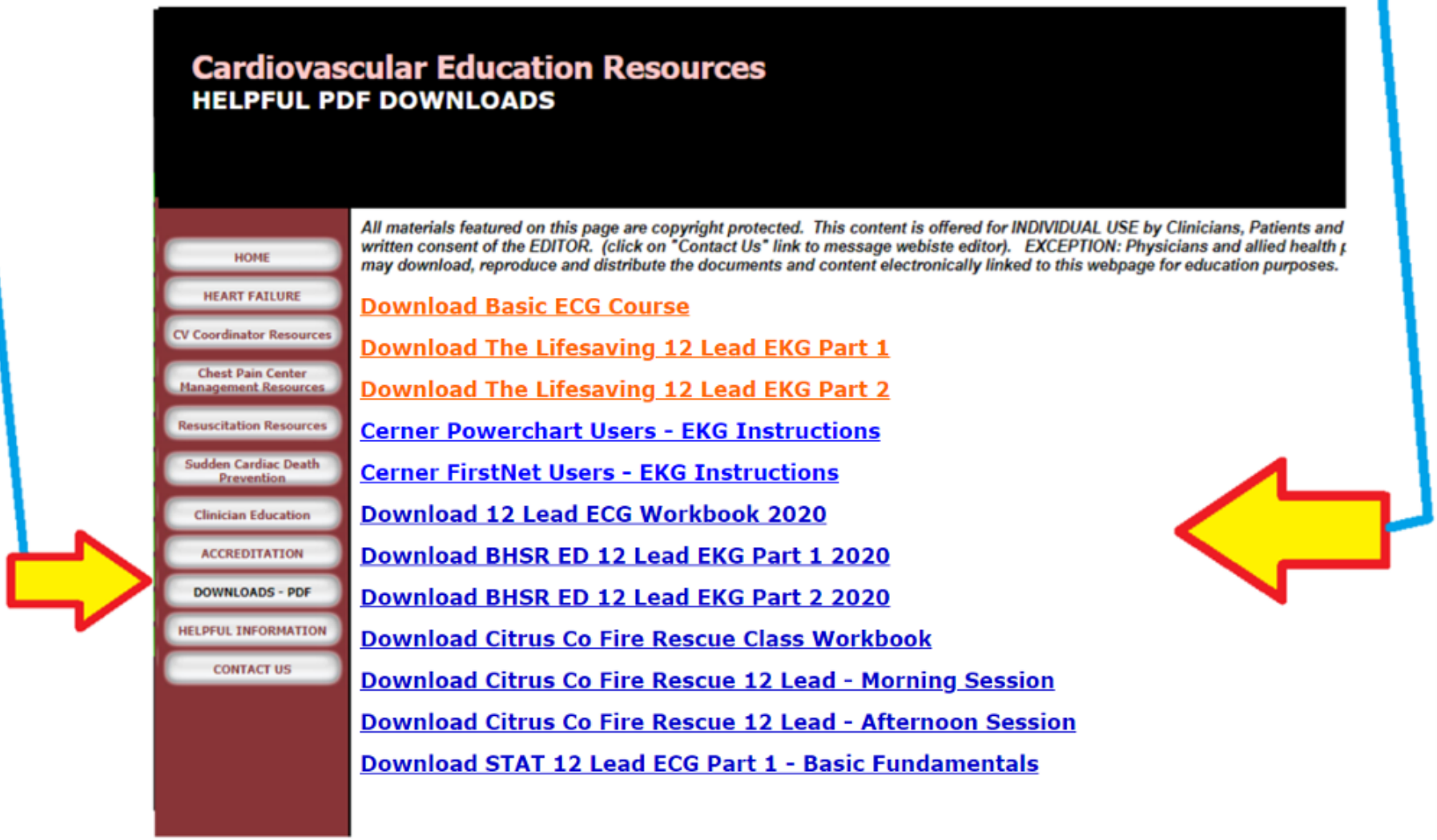

“The Integrated, Lifesaving Lead ECG”

There are multiple applications for the 12 Lead ECG, just as there are multiple facets for learning how to interpret 12 Lead ECGs. This course focuses on ECG diagnostic indicators of:

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- Acute Coronary Syndromes
  - STEMI
  - NSTEMI
  - Unstable Angina
  - Low Risk Chest Pain

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- Acute Coronary Syndromes
- Sudden Arrhythmia Death Syndromes (SADS)
  - Long QT Syndrome (LQTS)
  - Brugada Syndrome (BrS)
  - Hypertrophic Cardiomyopathy (HCM)
  - Arrhythmogenic Right Ventricular Dysplasia (ARVD)
  - Catecholinergic Polymorphic Ventricular Tachycardia (CPVT)
  - Wolff-Parkinson-White (WPW) Syndrome

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- Acute Coronary Syndromes
- Sudden Arrhythmia Death Syndromes (SADS)

This course DOES NOT include many "traditional ECG course contents," such as Fascicular Blocks, Chamber Hypertrophy, & other "less lethal cardiac abnormalities." Our 3 day course covers all of this and more.

Source of Curriculum:

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

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- Current Evidence-based Research
  - Journal of the American College of Cardiology (JACC)
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  - 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

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with CASE STUDIES from the CARDIAC CATHETERIZATION LAB

WAYNE W RUPPERT

Published in 2010. Working on UPDATE.

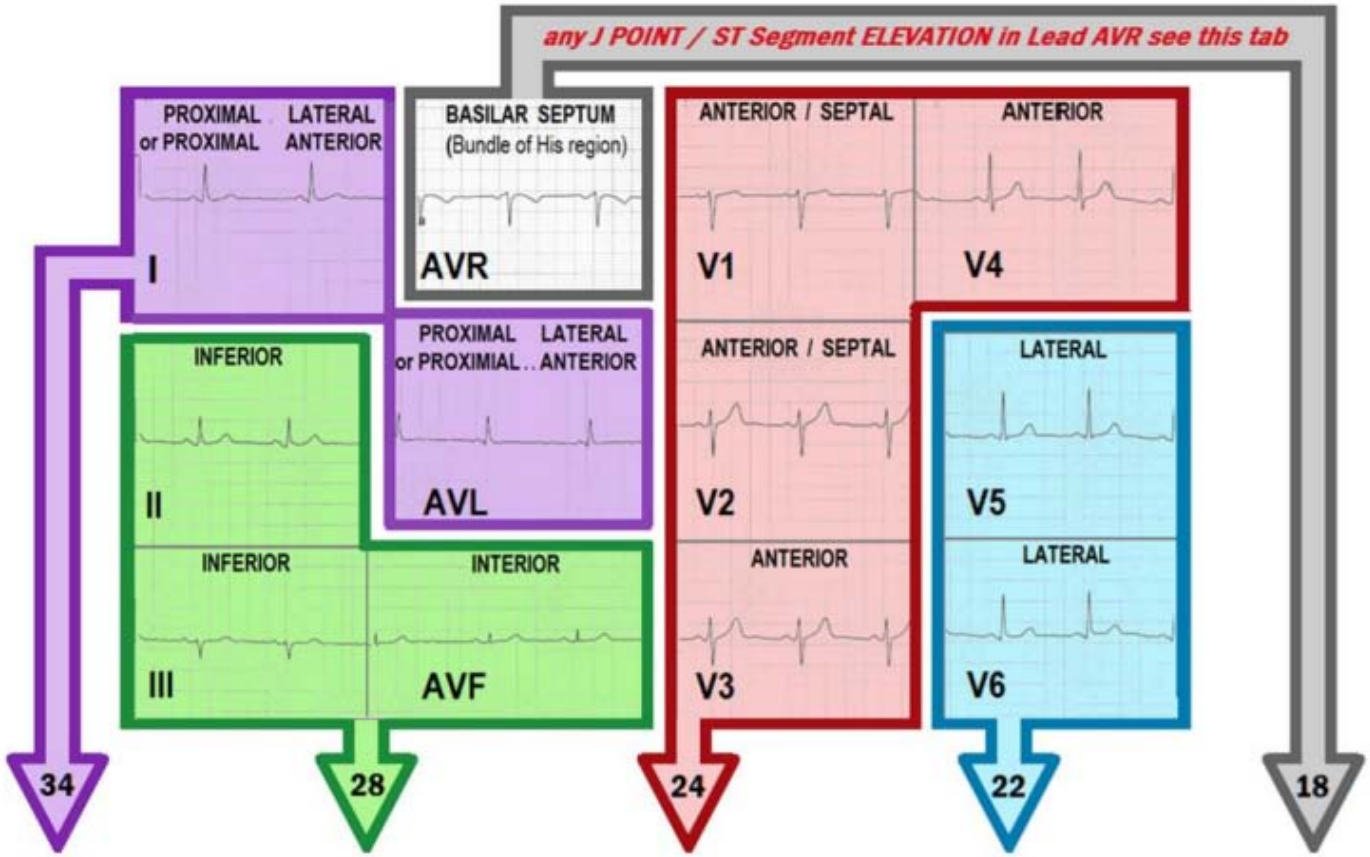
STEMI Assistant

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



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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…….*

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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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- In other words, *"just go along for the ride."*

Don’t worry.

This course will be NOWHERE NEAR as much fun as a roller coaster.

Don’t worry.

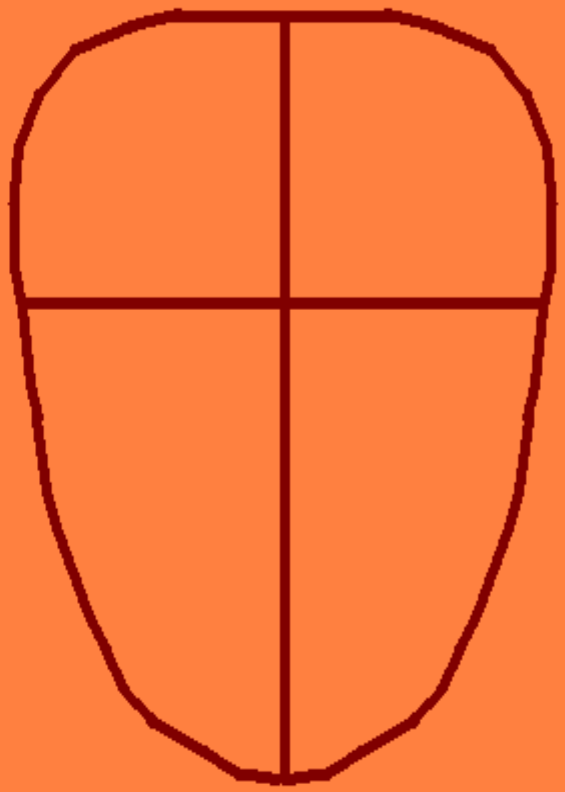
This course will be
NOWHERE NEAR
as much fun as a
roller coaster.

But it will move as fast.

Cardiac A & P “101”...

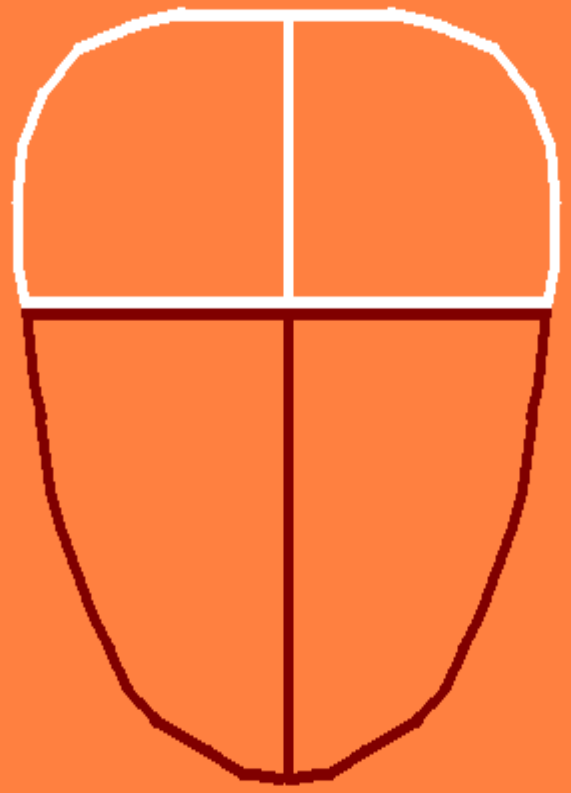
- **Heart structure – basic 4 chambers**

FOUR CHAMBERED PUMP



FOUR CHAMBERED PUMP . . .

2 ATRIUM

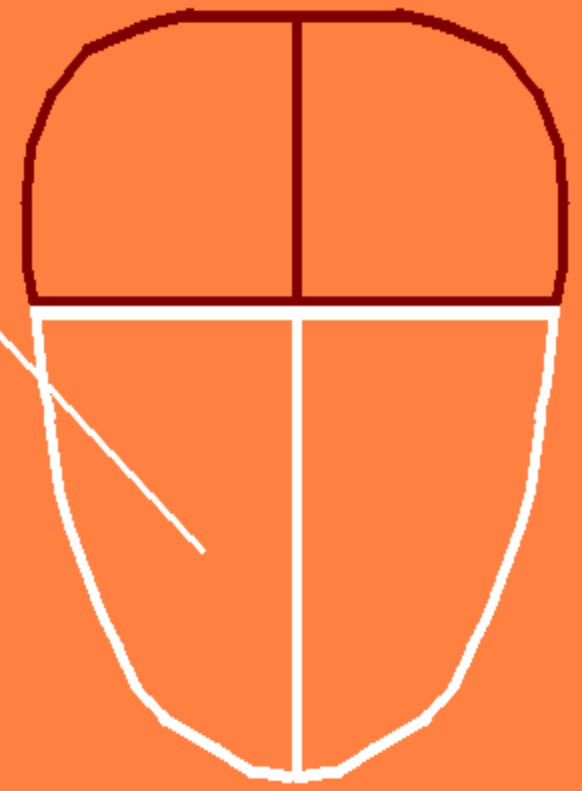


PRIMARY JOB:

"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

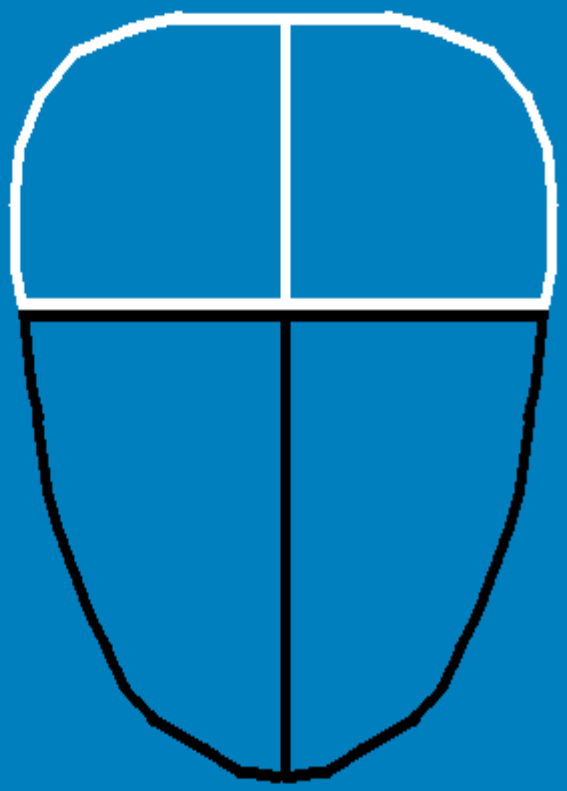
APPROXIMATELY

WHAT

PERCENTAGE

OF THE

CARDIAC OUTPUT ?



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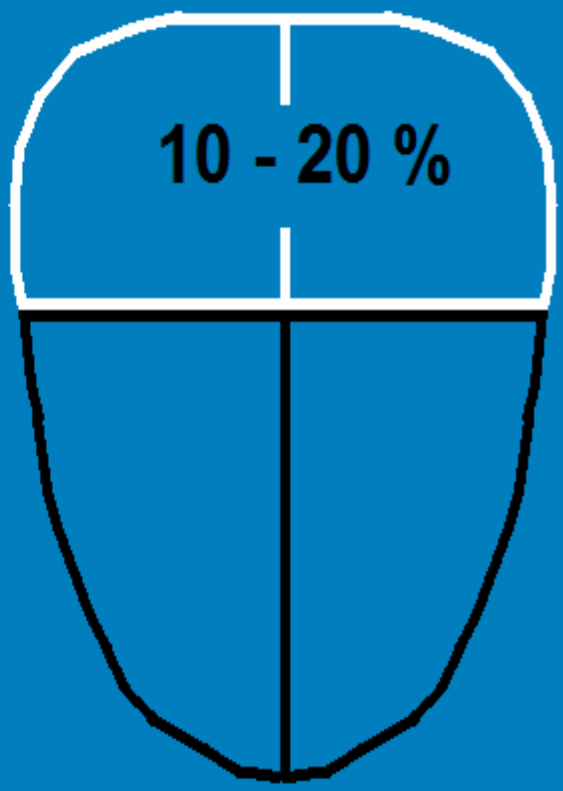


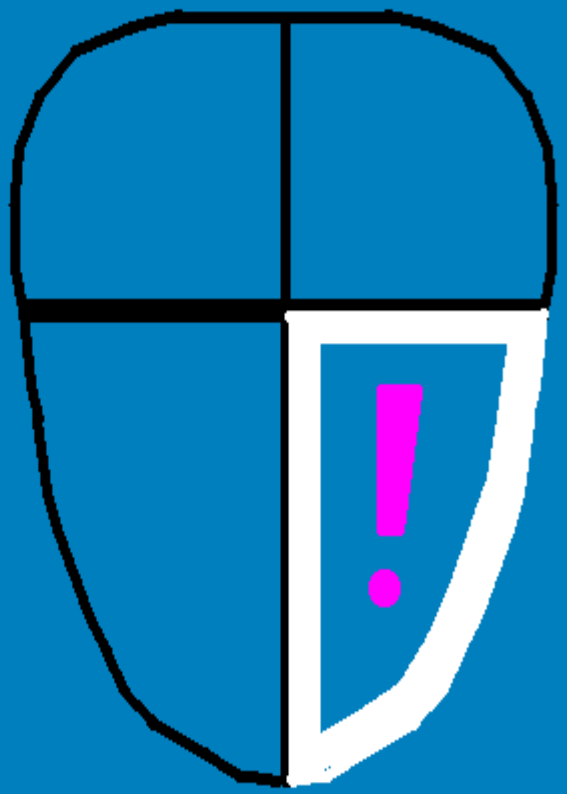
THE CHAMBER MOST IMPORTANT TO KEEPING THE PATIENT ALIVE

(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 structure – basic 4 chambers
- **Action Potential of Ventricular Muscle Cells**

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

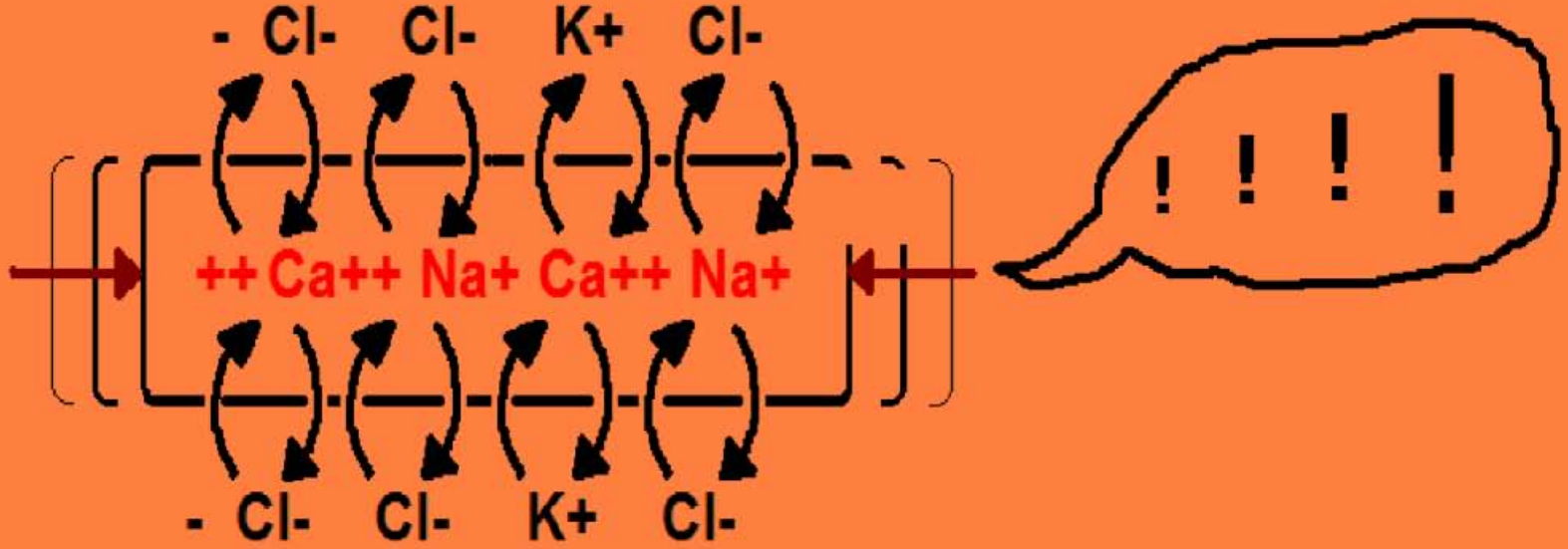
Ca++ Na+ Ca++ Na+ Ca++

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!

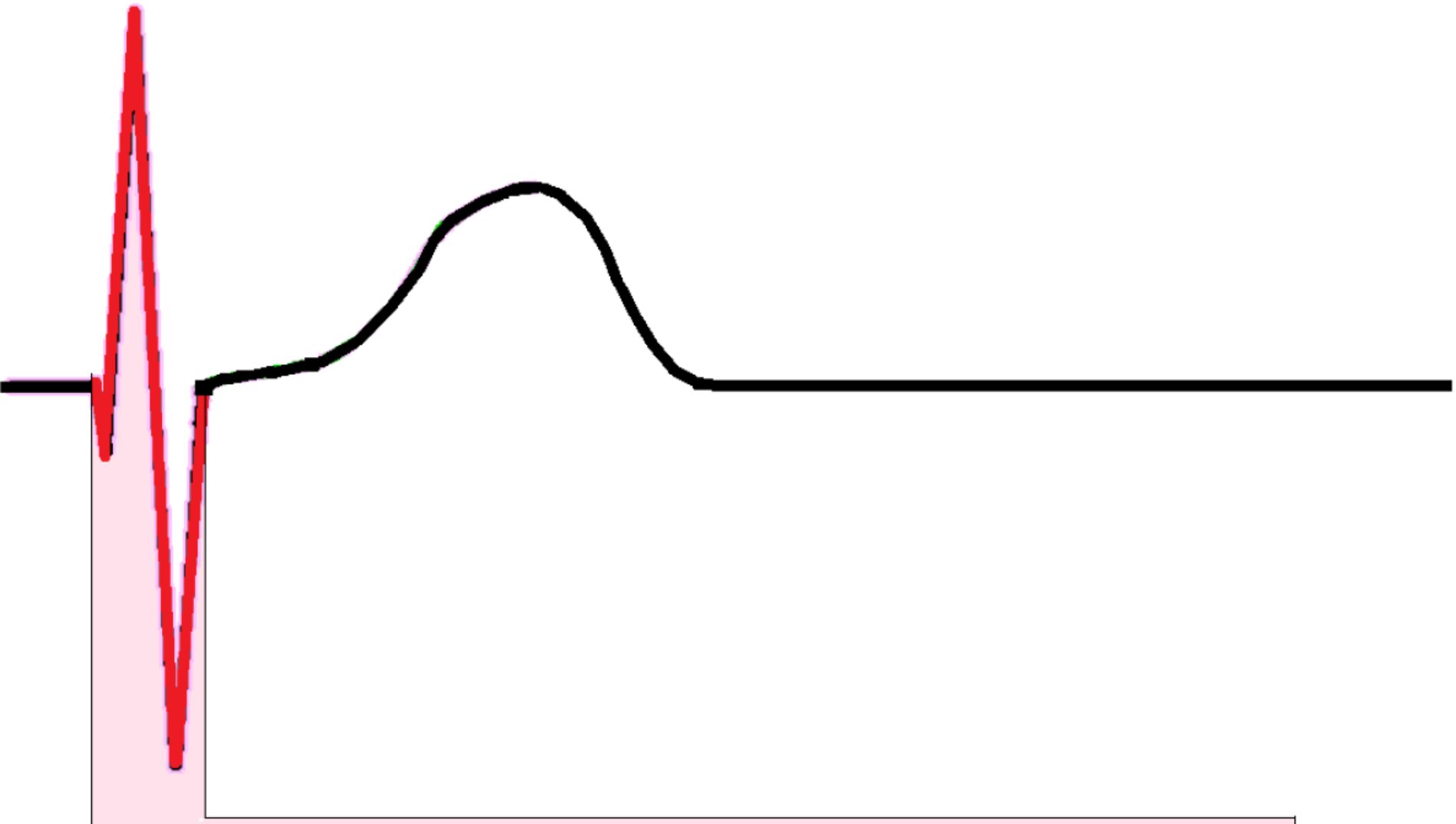
THIS (OF COURSE) IS KNOW AS . . .

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

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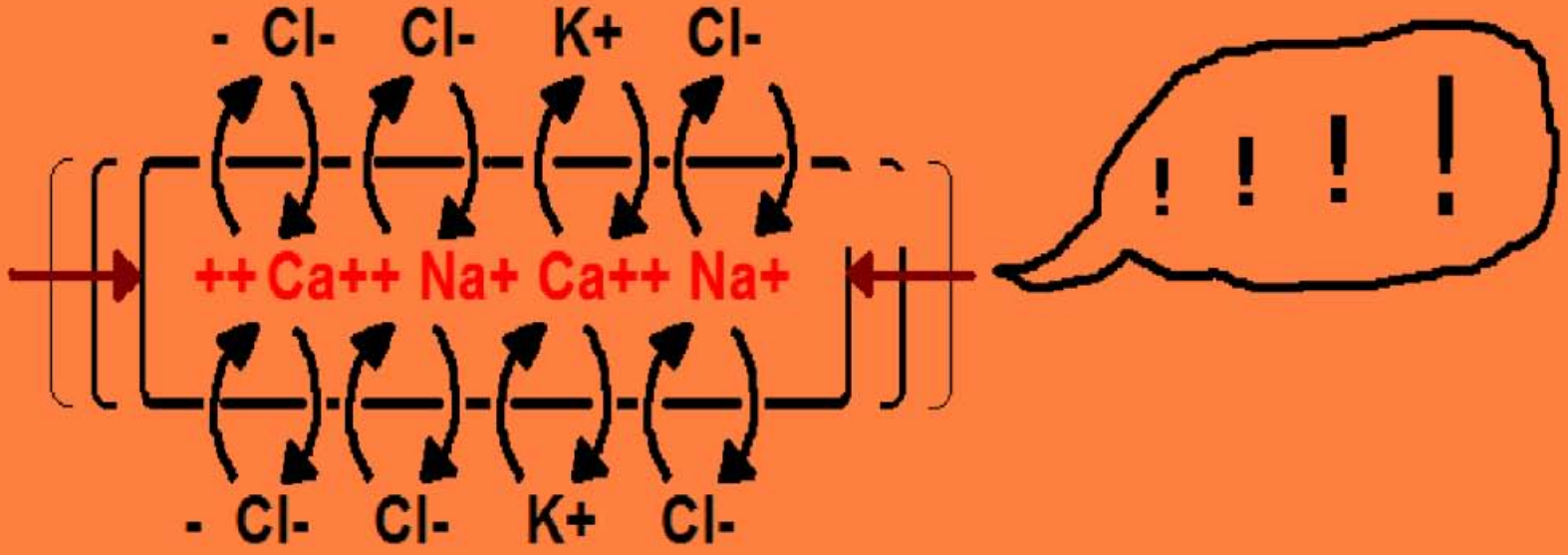


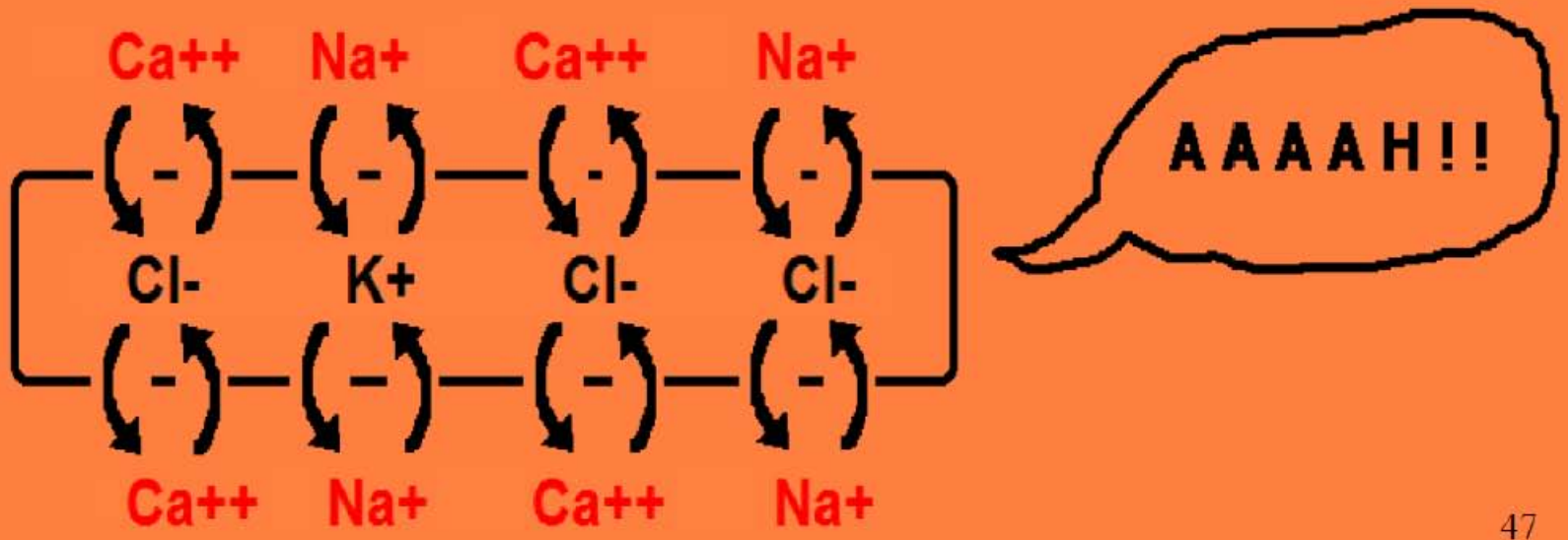
. . . THE CELL CONTRACTS!

CARDIAC ANATOMY and PHYSIOLOGY "101"

AFTER DEPOLARIZATION, THE CELLS RELAX.

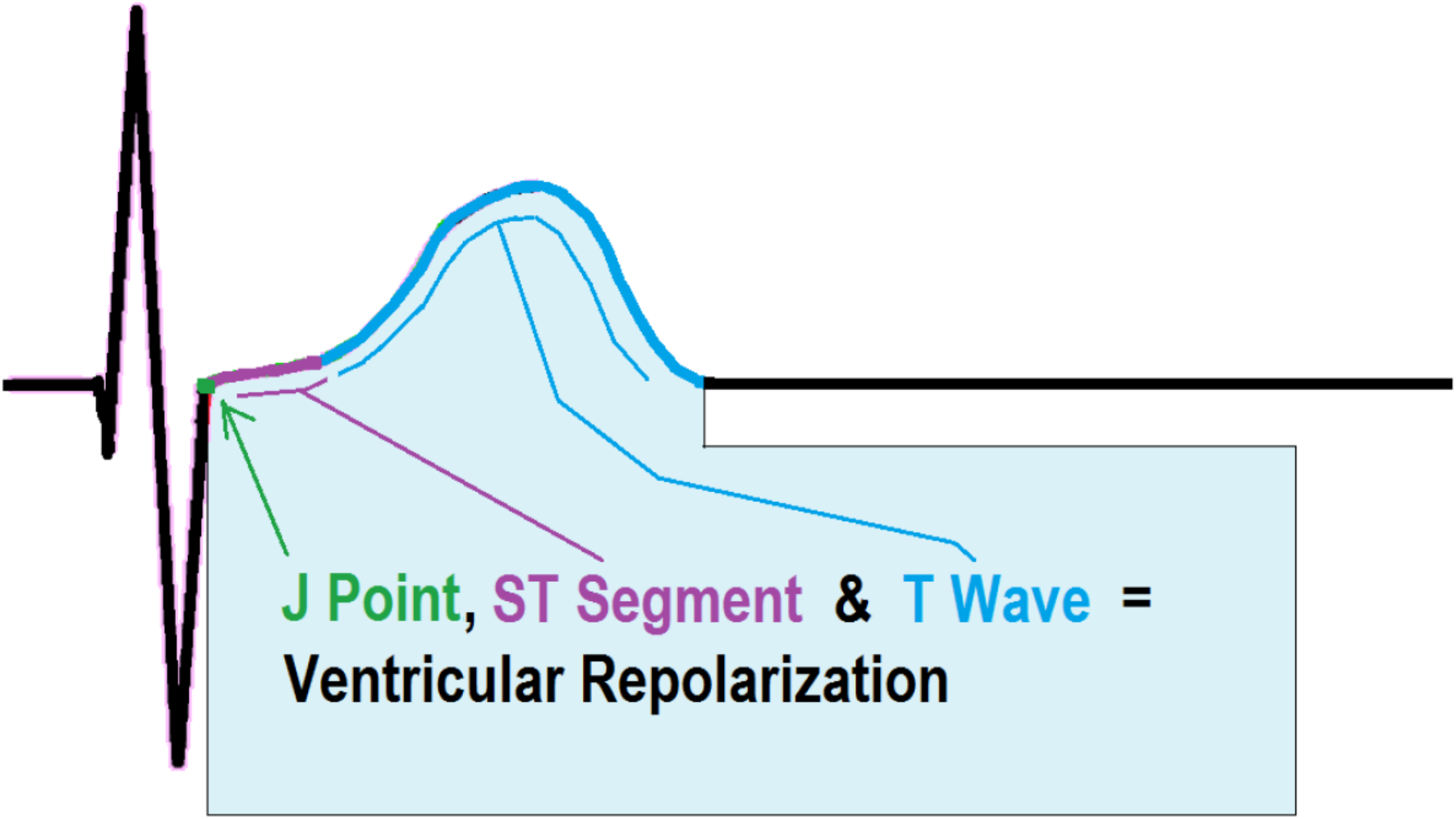
THE IONS RETURN TO THEIR ORIGINAL POSITIONS --

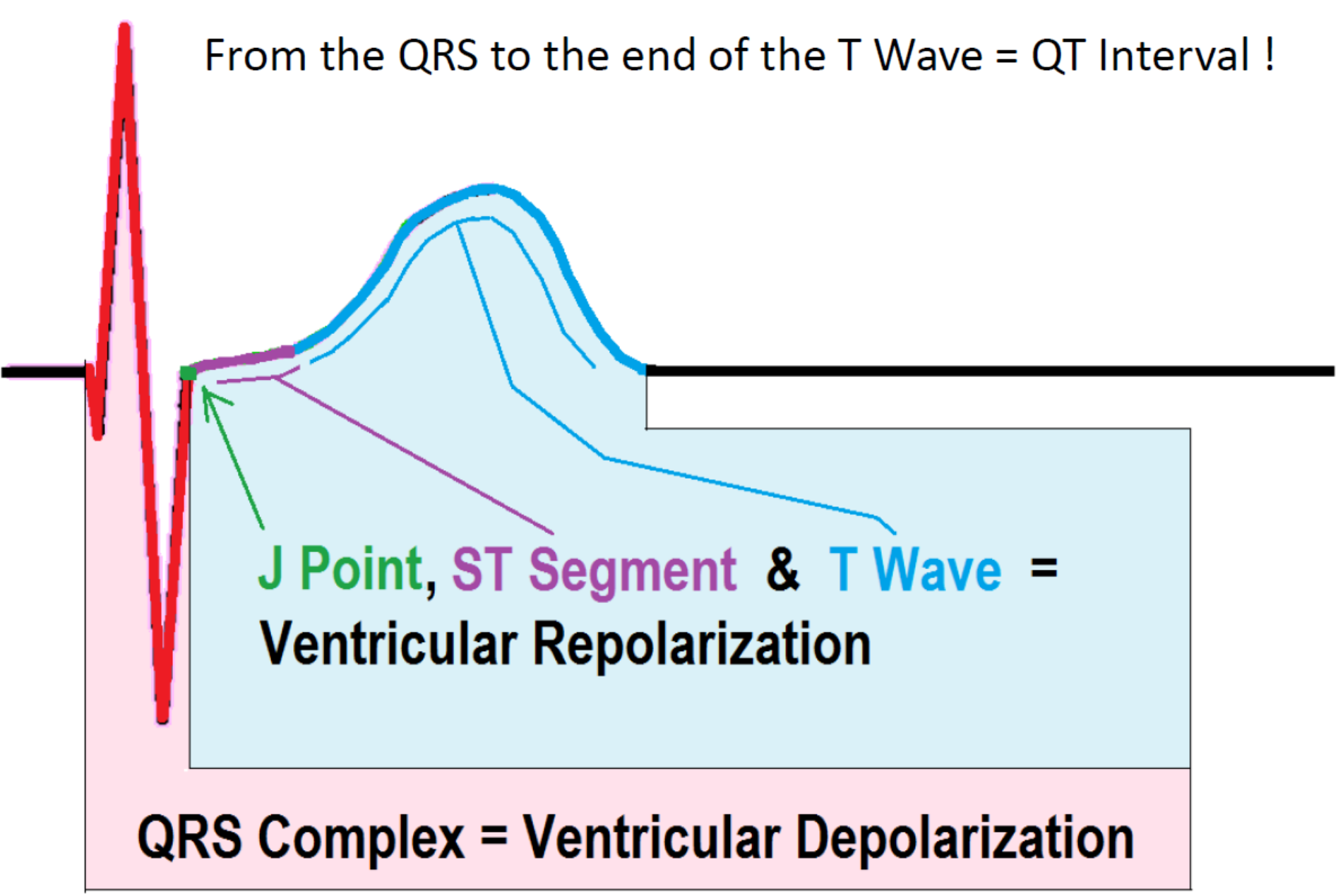
THIS PROCESS IS KNOWN AS REPOLARIZATION



Repolarization on the ECG:

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

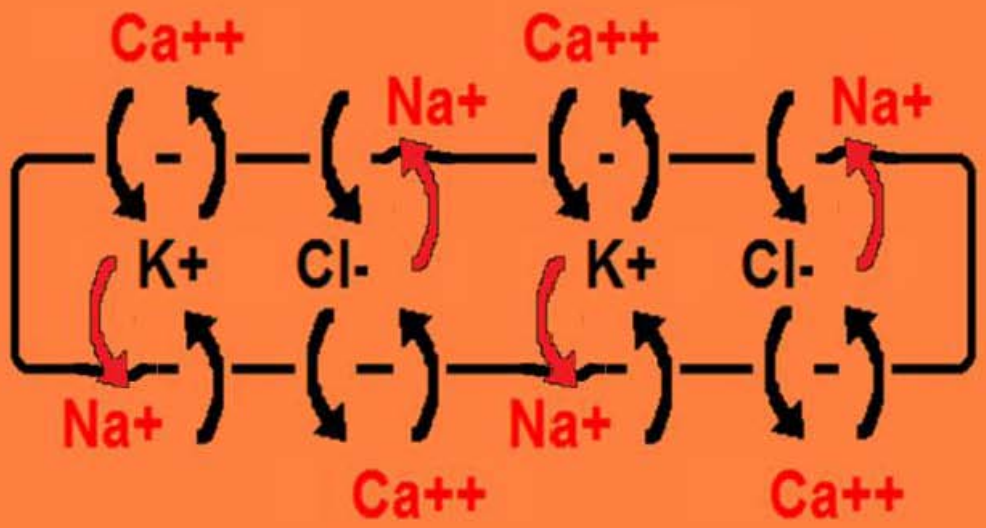

J Point, ST Segment & T Wave =
Ventricular Repolarization


From the QRS to the end of the T Wave = QT Interval !
J Point, ST Segment & T Wave =
Ventricular Repolarization
QRS Complex = Ventricular Depolarization

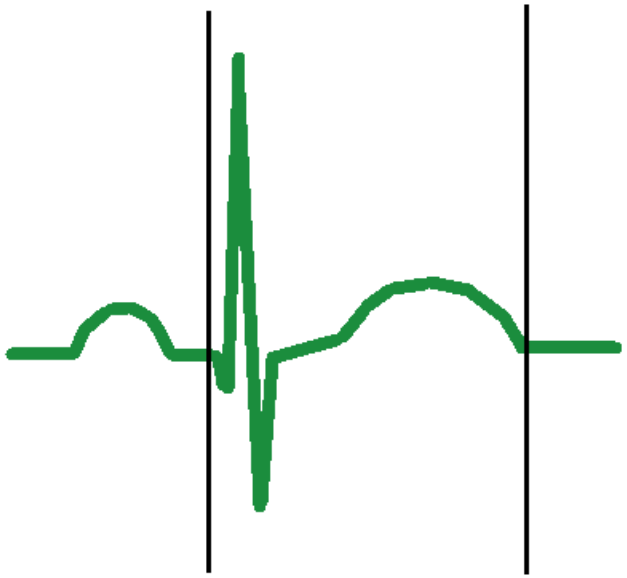
CARDIAC ANATOMY and PHYSIOLOGY "101"

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

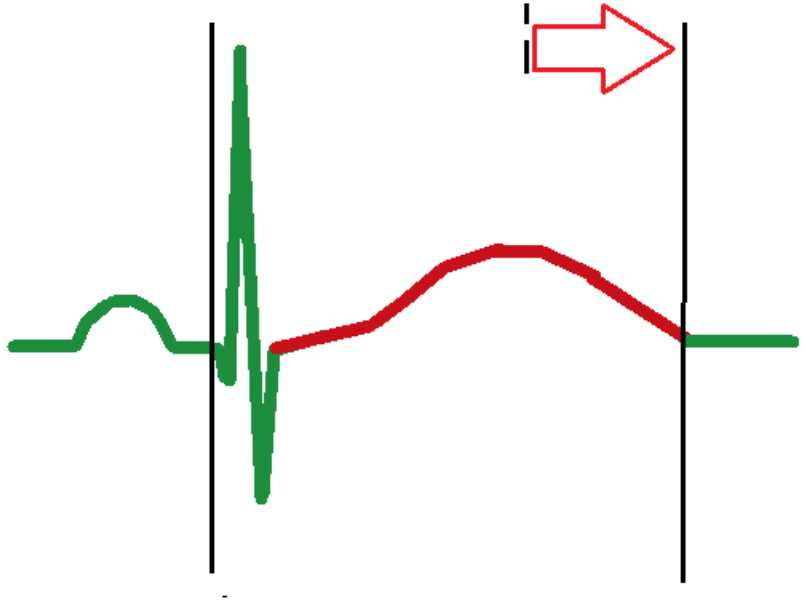
. . . . this can DELAY REPOLARIZATION, which will show on the ECG as "QT Prolongation"



Normal
QT Interval



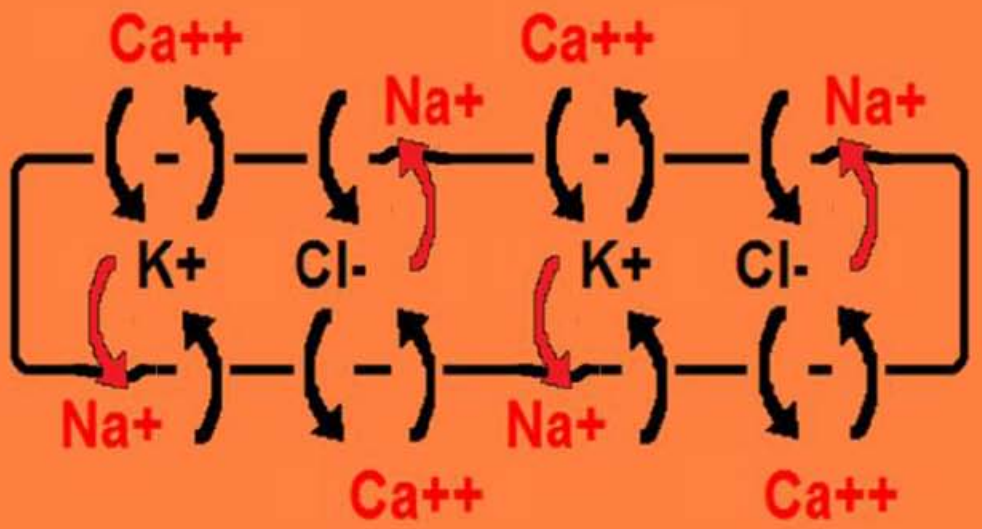
ABNORMAL
(prolonged)
QT Interval



CARDIAC ANATOMY and PHYSIOLOGY "101"

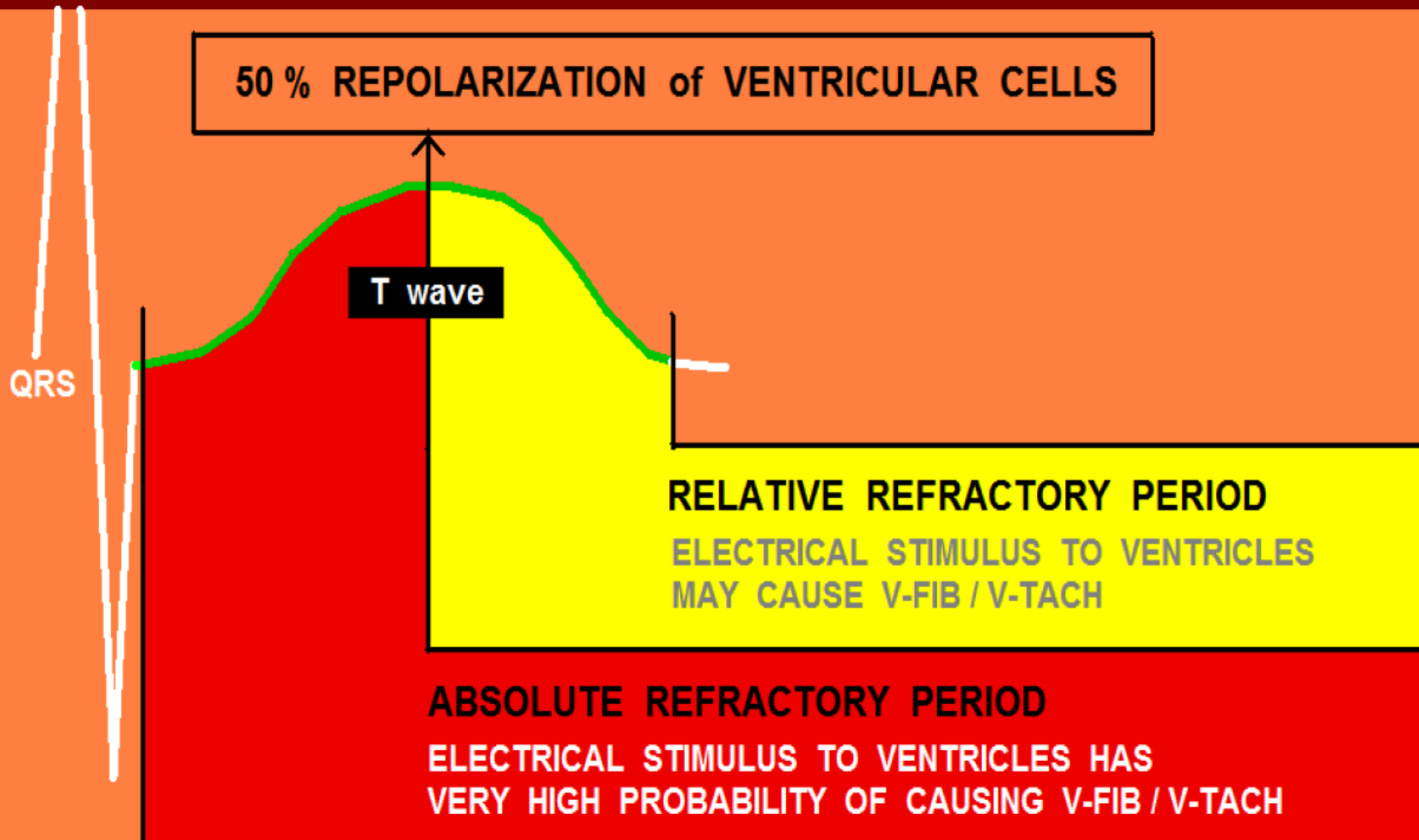
When ION CHANNELS are MALFORMED, the abnormal channel shape may DELAY the transfer of IONS . . . . .

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



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

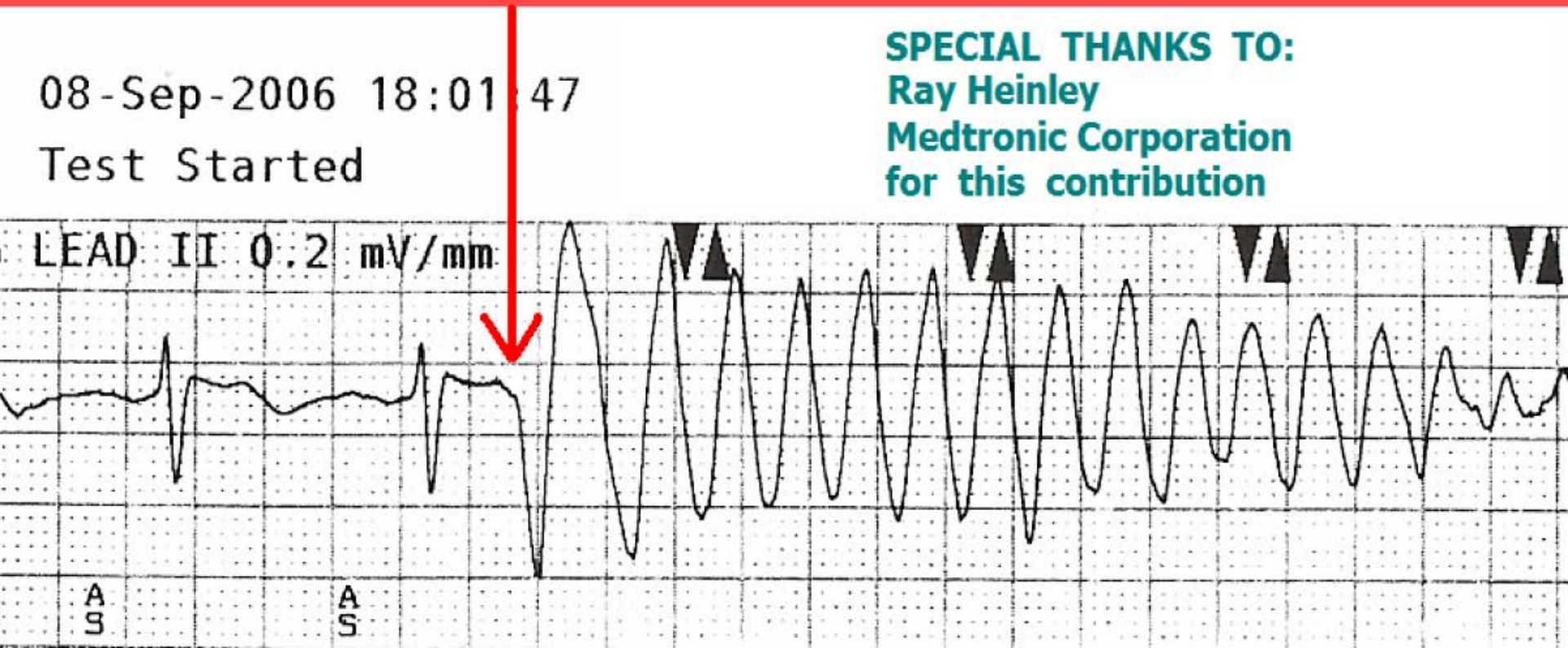
CARDIAC ANATOMY and PHYSIOLOGY "101"



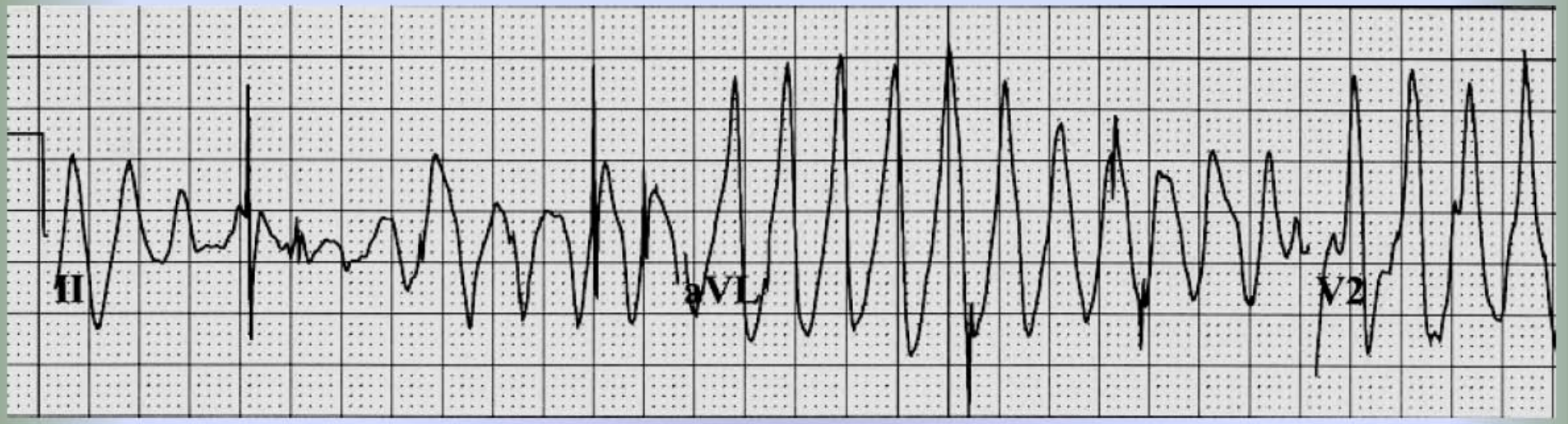
ROUTINE TEST OF ICD

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

SPECIAL THANKS TO:
Ray Heinley
Medtronic Corporation
for this contribution



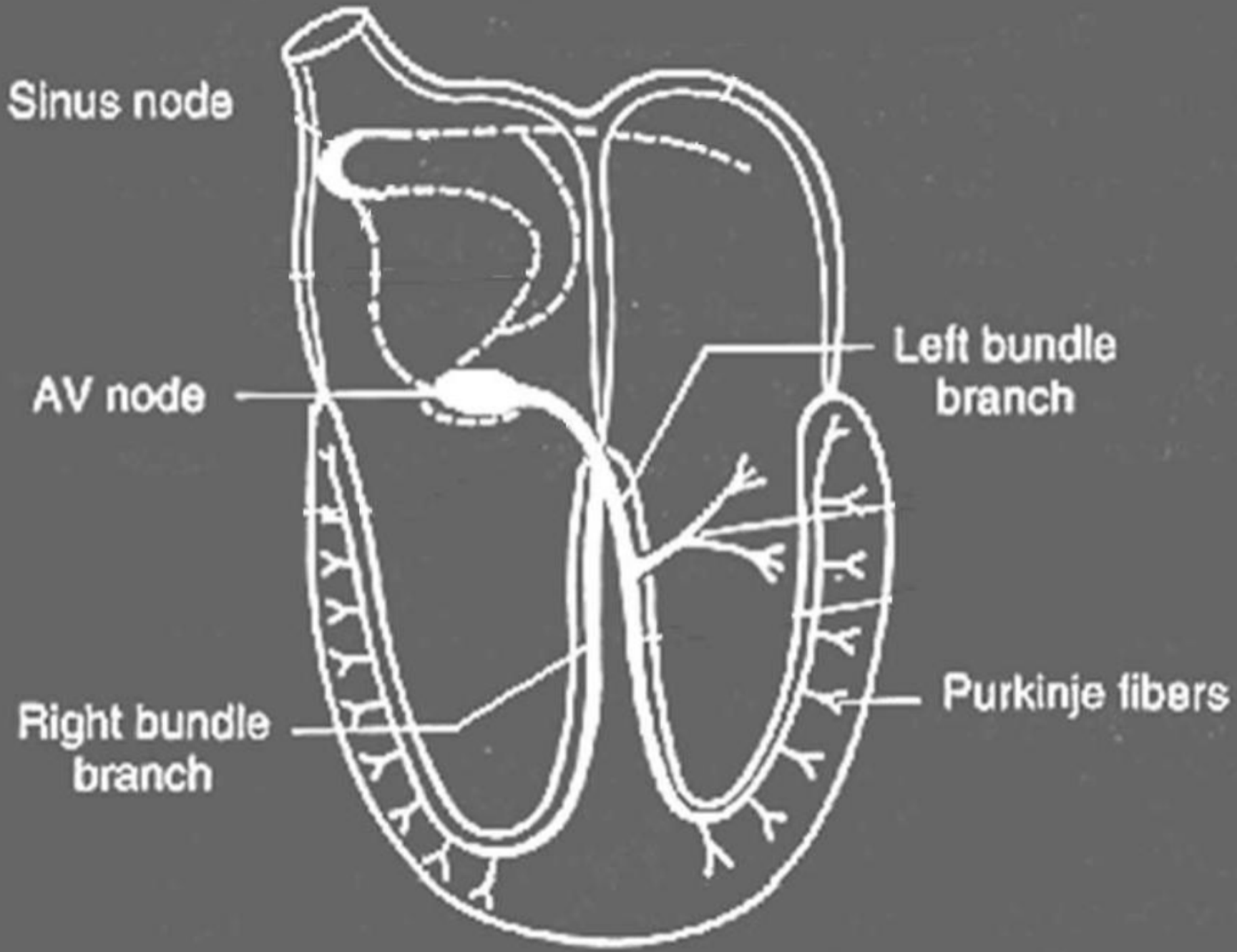
Torsades de Pointes (TdP)

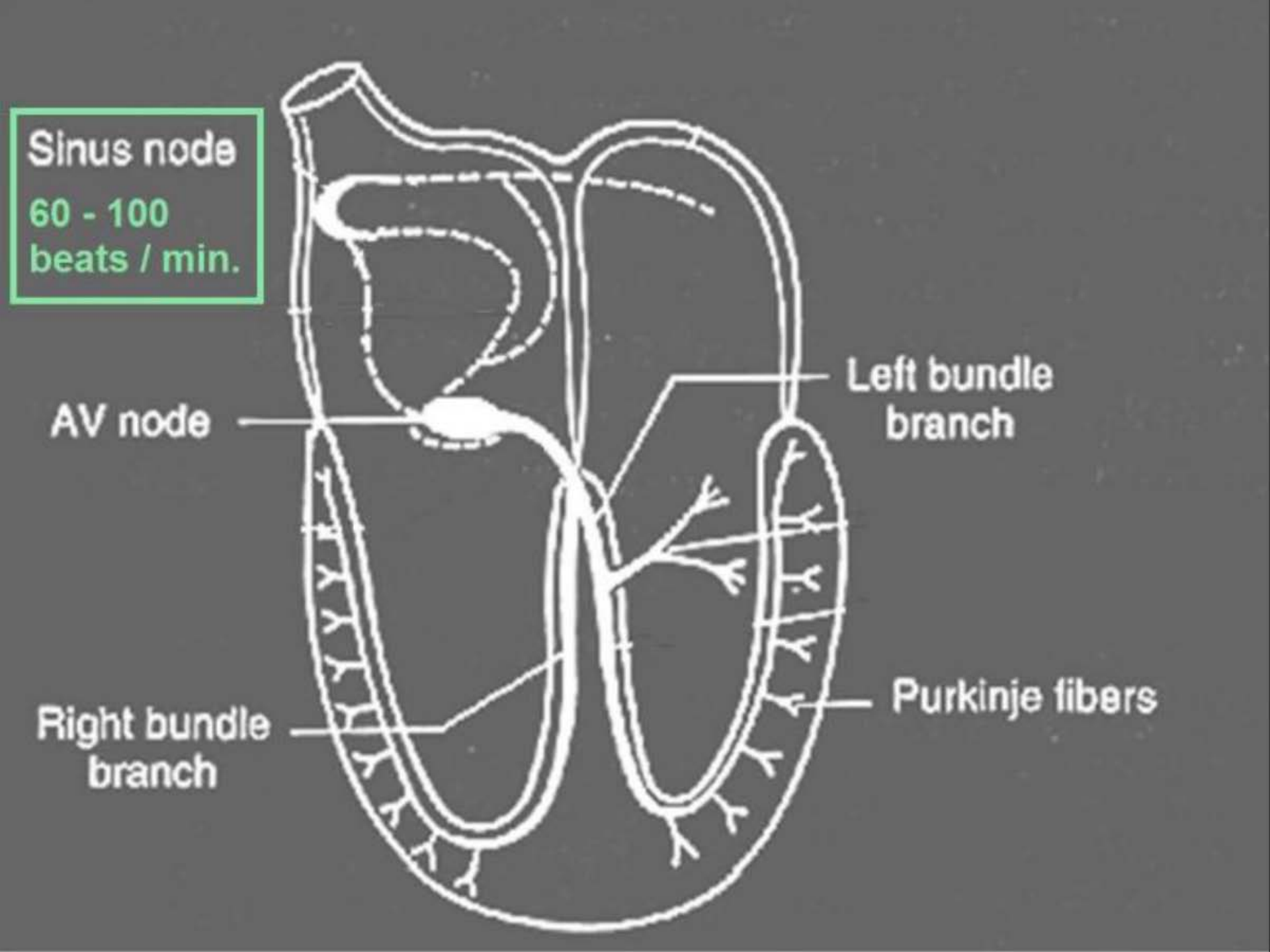


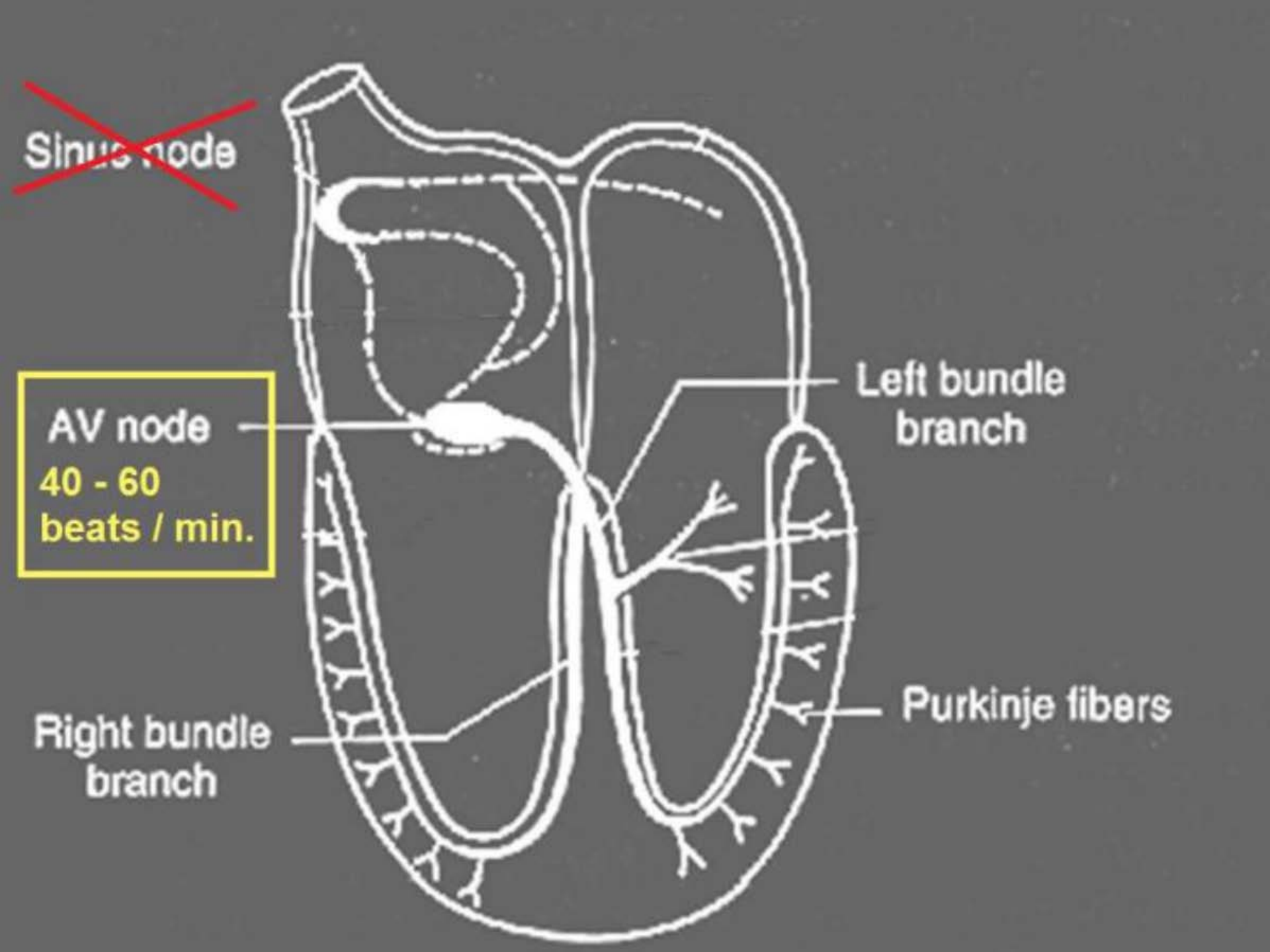
- **Common cause: QTc > 600 ms**
- Patients typically have little to no cardiac output when in this rhythm
- TdP may self-terminate or deteriorate into **VENTRICULAR FIBRILLATION**

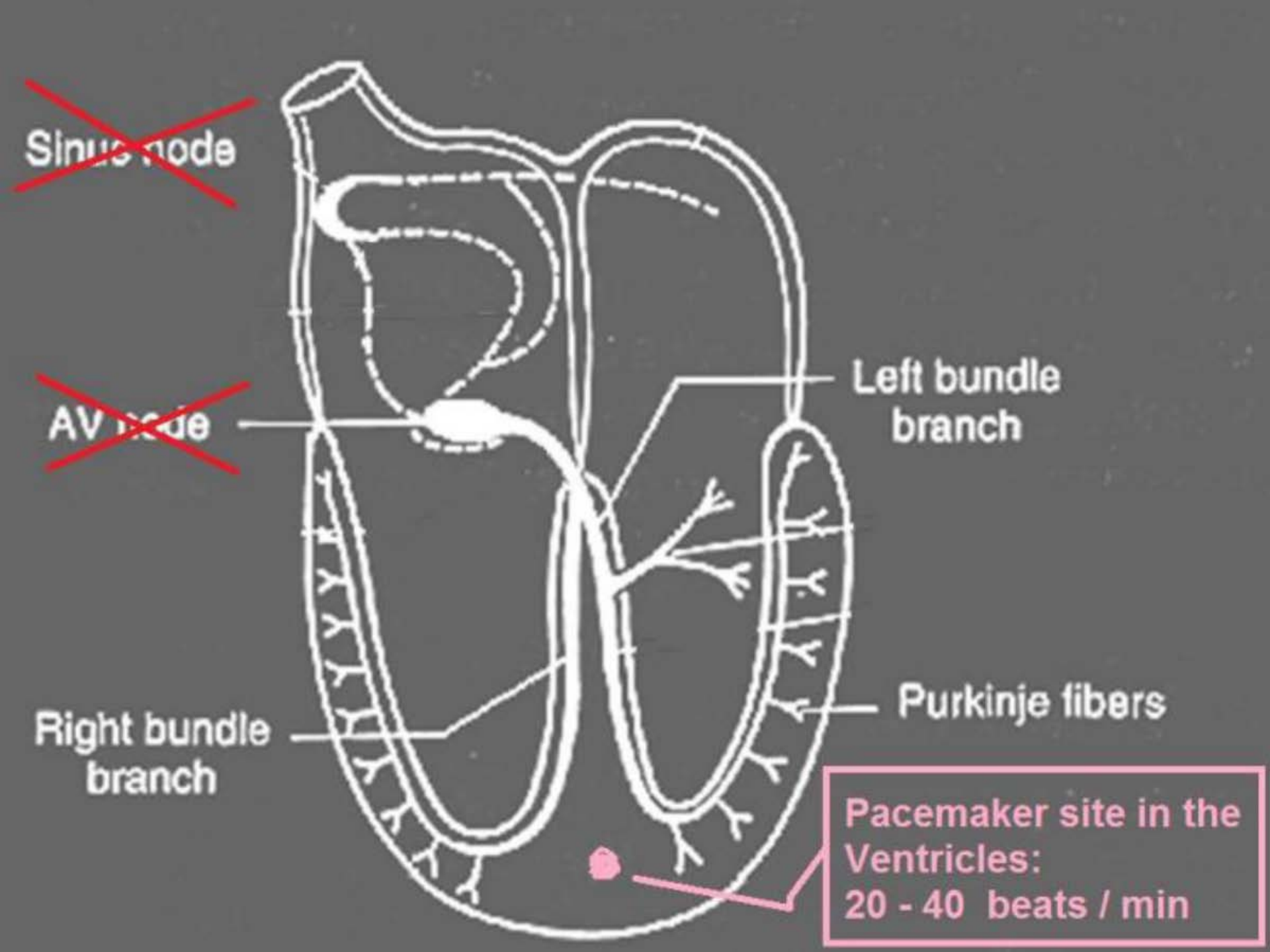
Cardiac A & P “101”...

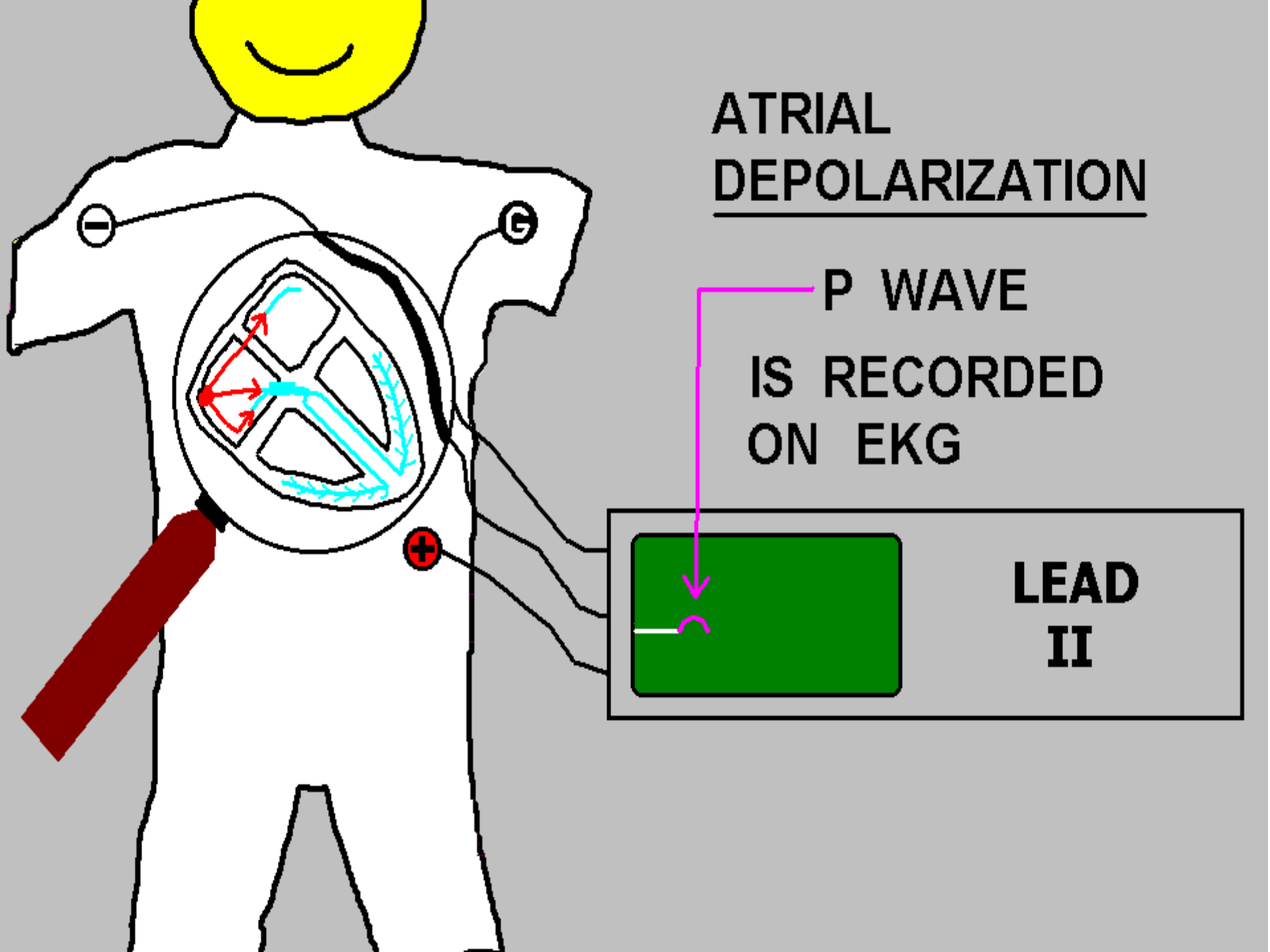
- Heart structure – basic 4 chambers
- Action Potential of Ventricular Muscle Cells
- **Electrical System overview**

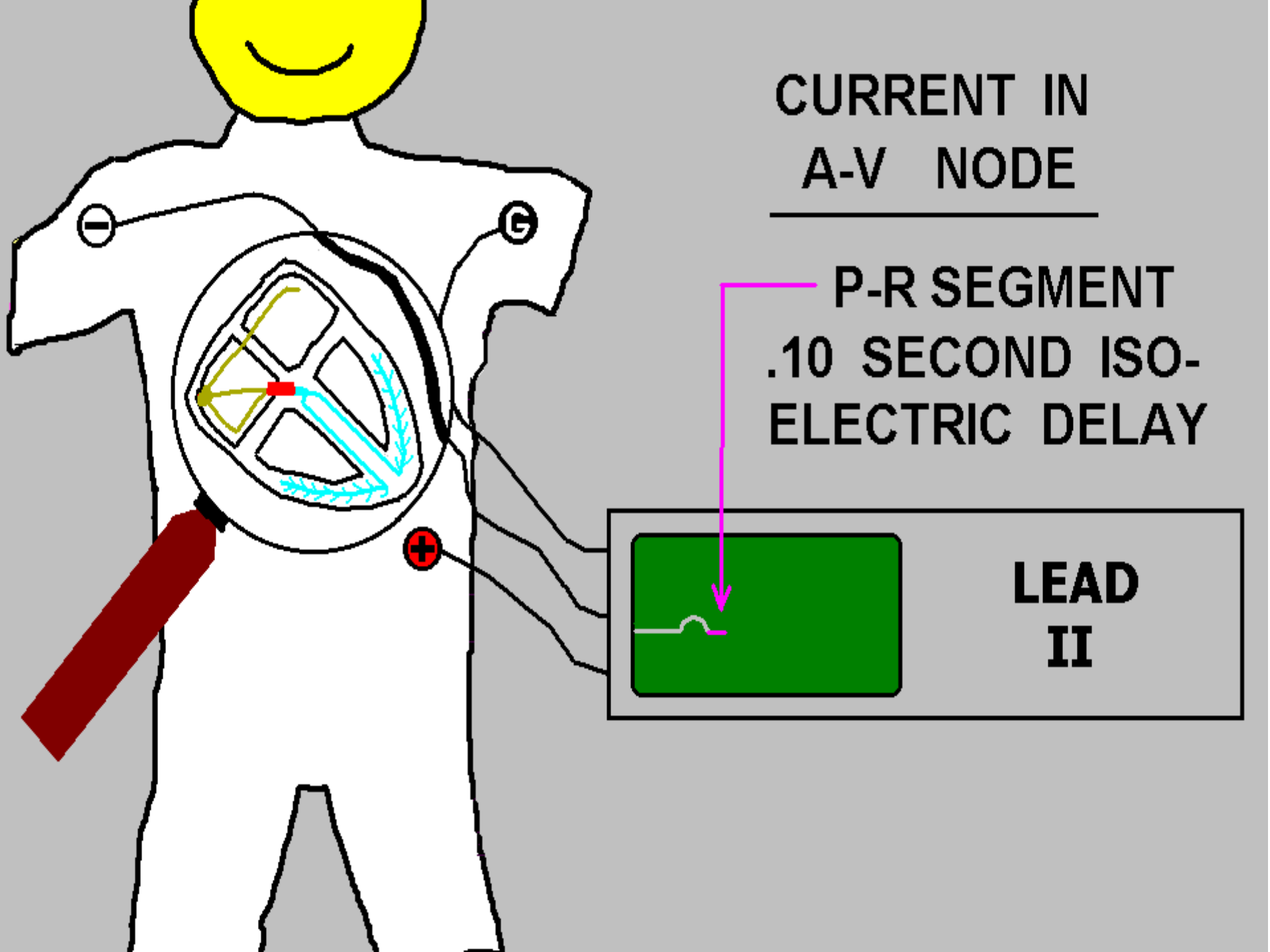

Sinus node
AV node
Left bundle branch
Right bundle branch
Purkinje fibers

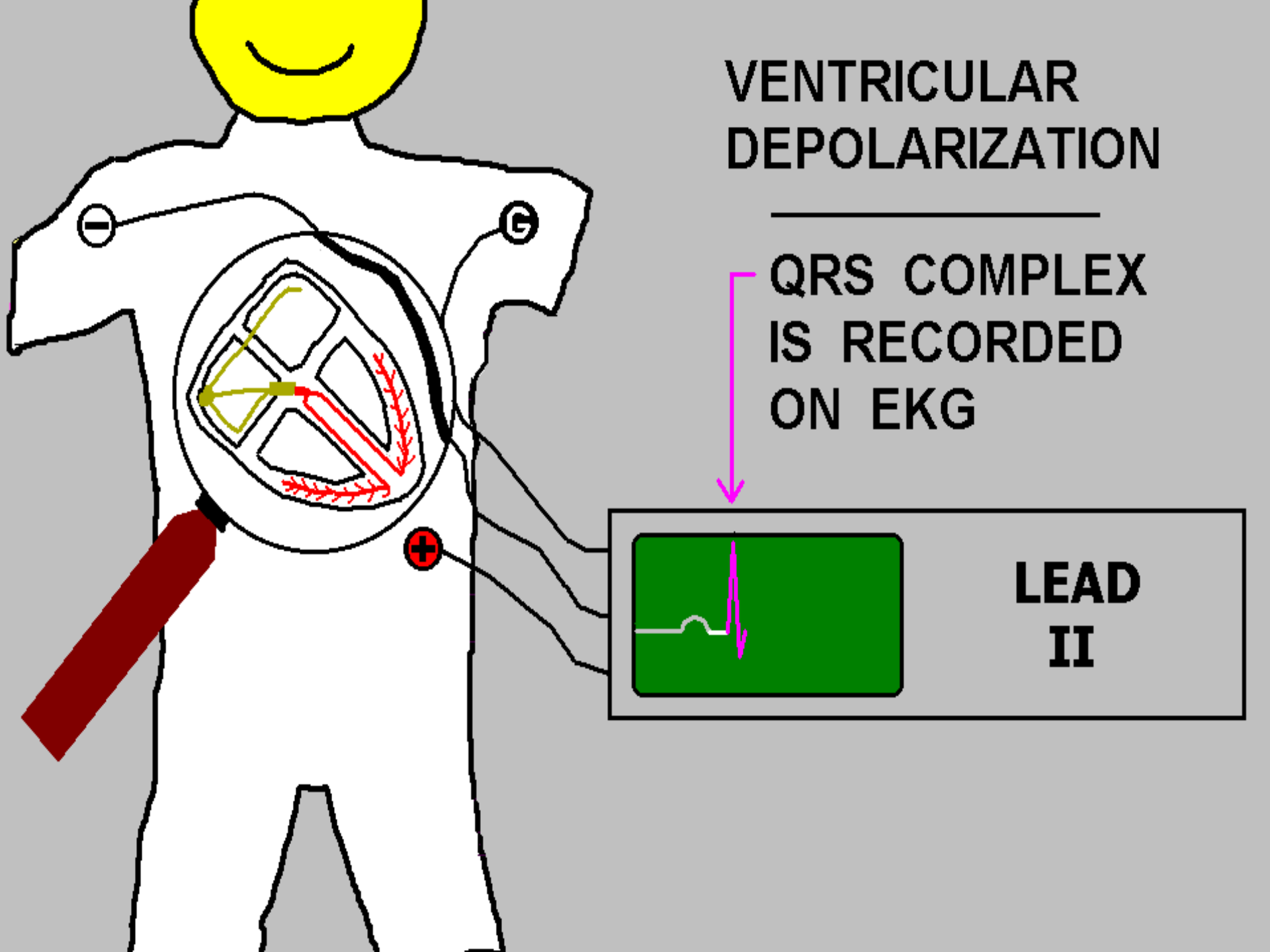

Sinus node
60 - 100
beats / min.
AV node
Left bundle
branch
Right bundle
branch
Purkinje fibers

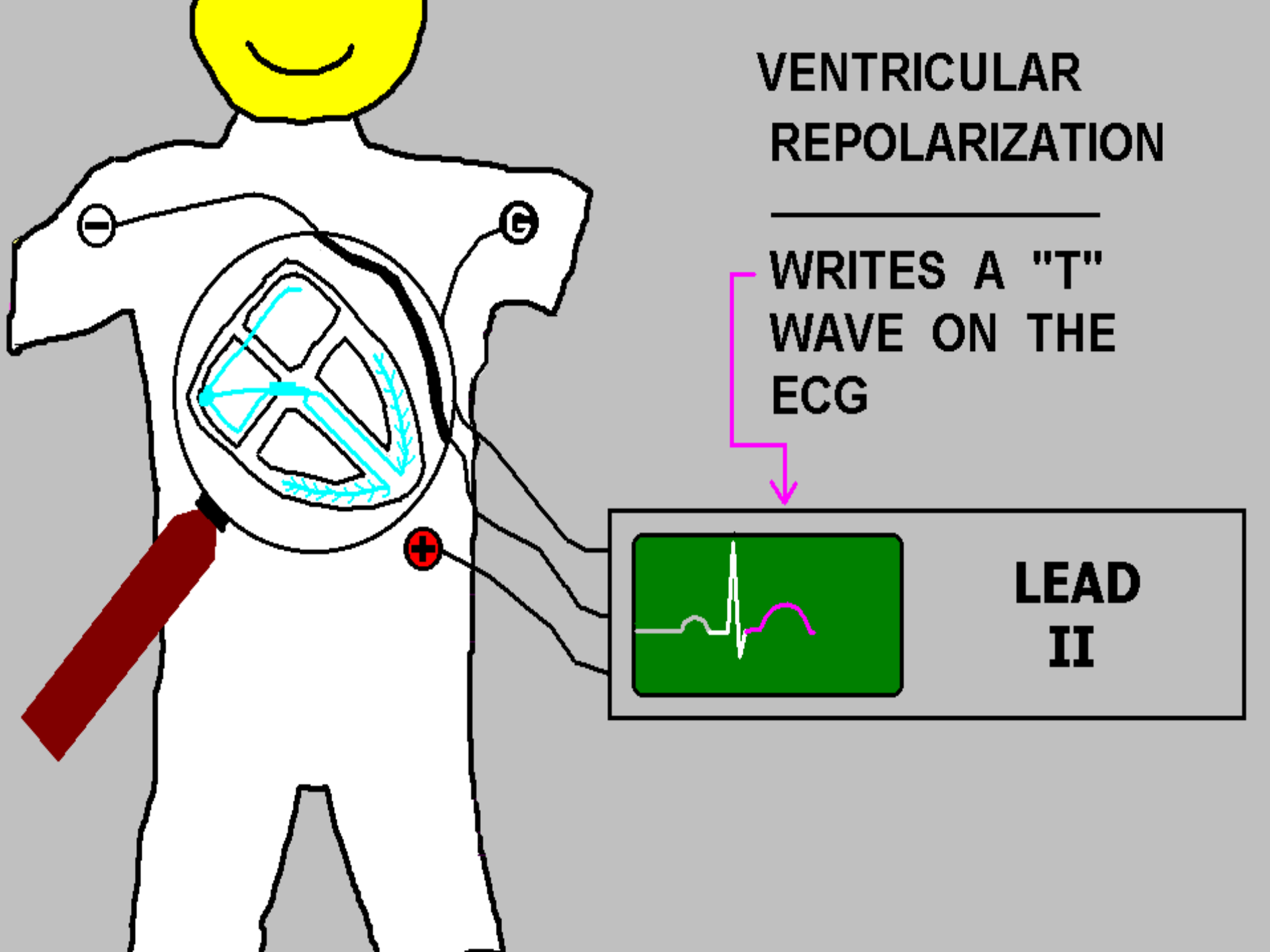

Sinus node
AV node
40 - 60
beats / min.
Left bundle
branch
Right bundle
branch
Purkinje fibers


Sinus node
AV node
Left bundle branch
Right bundle branch
Purkinje fibers
Pacemaker site in the Ventricles:
20 - 40 beats / min


ATRIAL
DEPOLARIZATION
P WAVE
IS RECORDED
ON EKG
LEAD
II


CURRENT IN
A-V NODE
P-R SEGMENT
.10 SECOND ISO-
ELECTRIC DELAY
LEAD
II

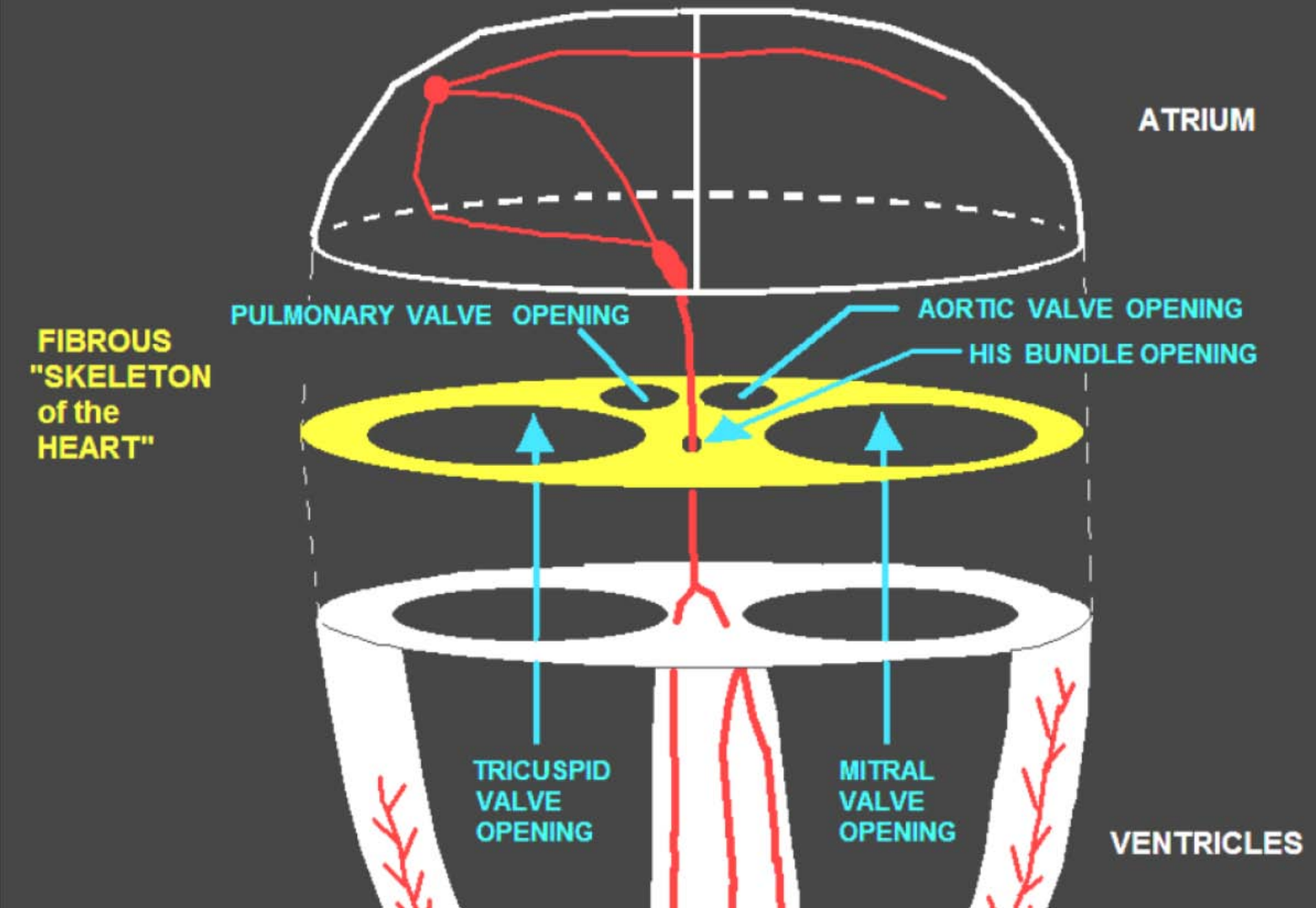

VENTRICULAR
DEPOLARIZATION
QRS COMPLEX
IS RECORDED
ON EKG
LEAD
II

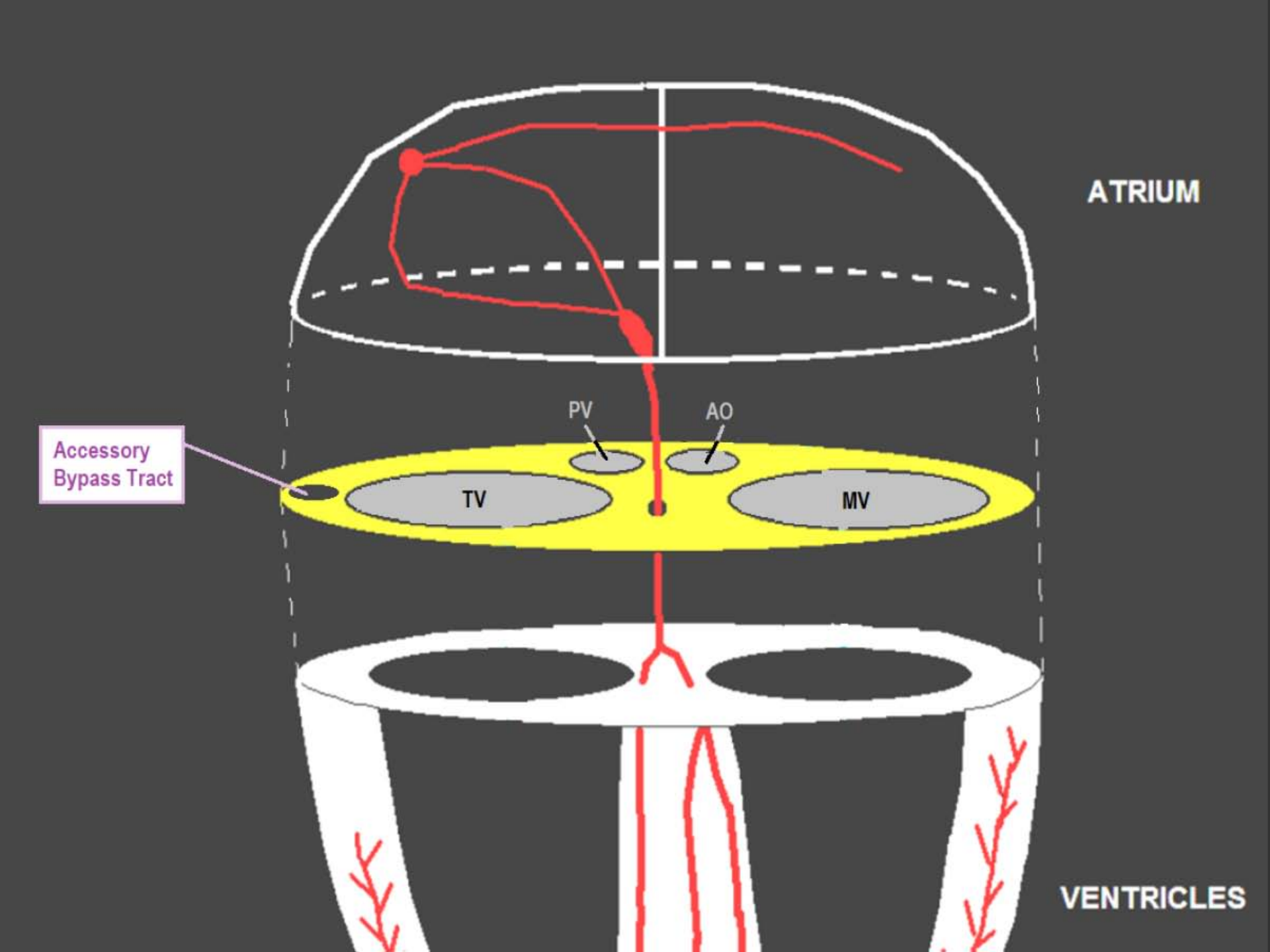

VENTRICULAR
REPOLARIZATION
WRITES A "T"
WAVE ON THE
ECG
LEAD
II

Cardiac A & P “101”...

- Heart structure – basic 4 chambers
- Action Potential of Ventricular Muscle Cells
- Electrical System overview
- **Fibrous Skeleton of Heart**

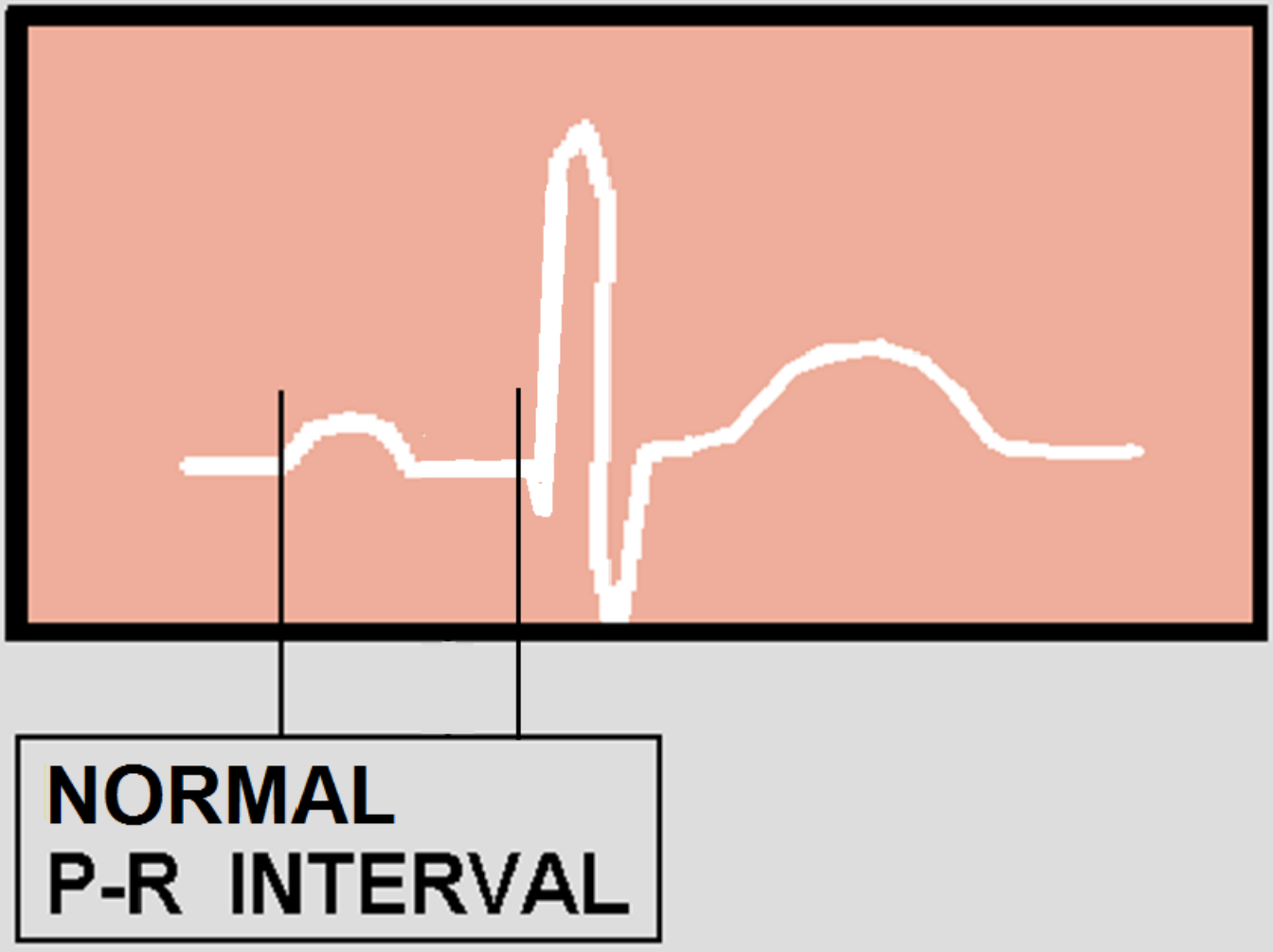
THE "SKELETON OF THE HEART"




ATRIUM
PV
AO
Accessory
Bypass Tract
TV
MV
VENTRICLES

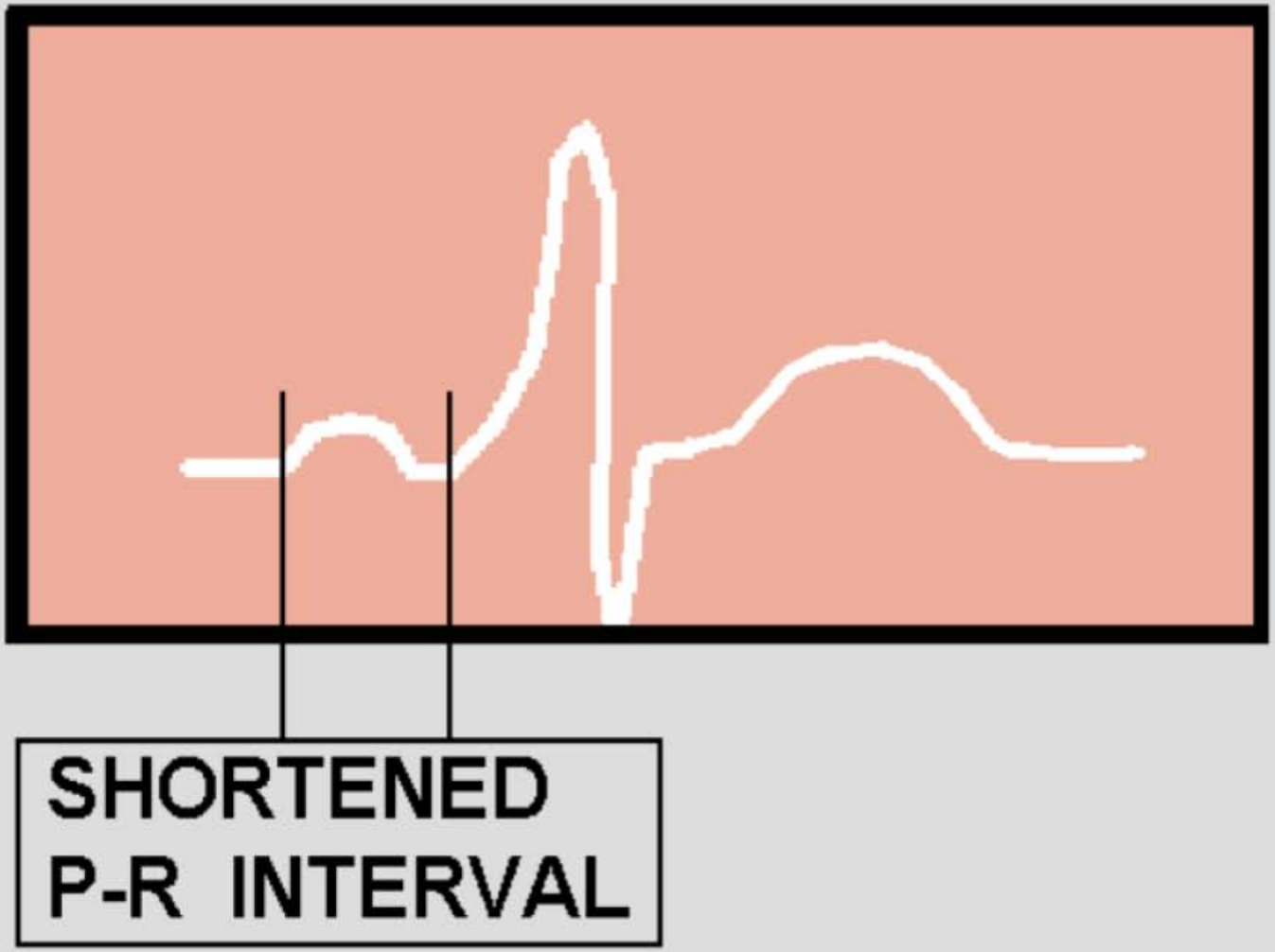
WOLFF-PARKINSON-WHITE

THE NORMAL ECG . . . .



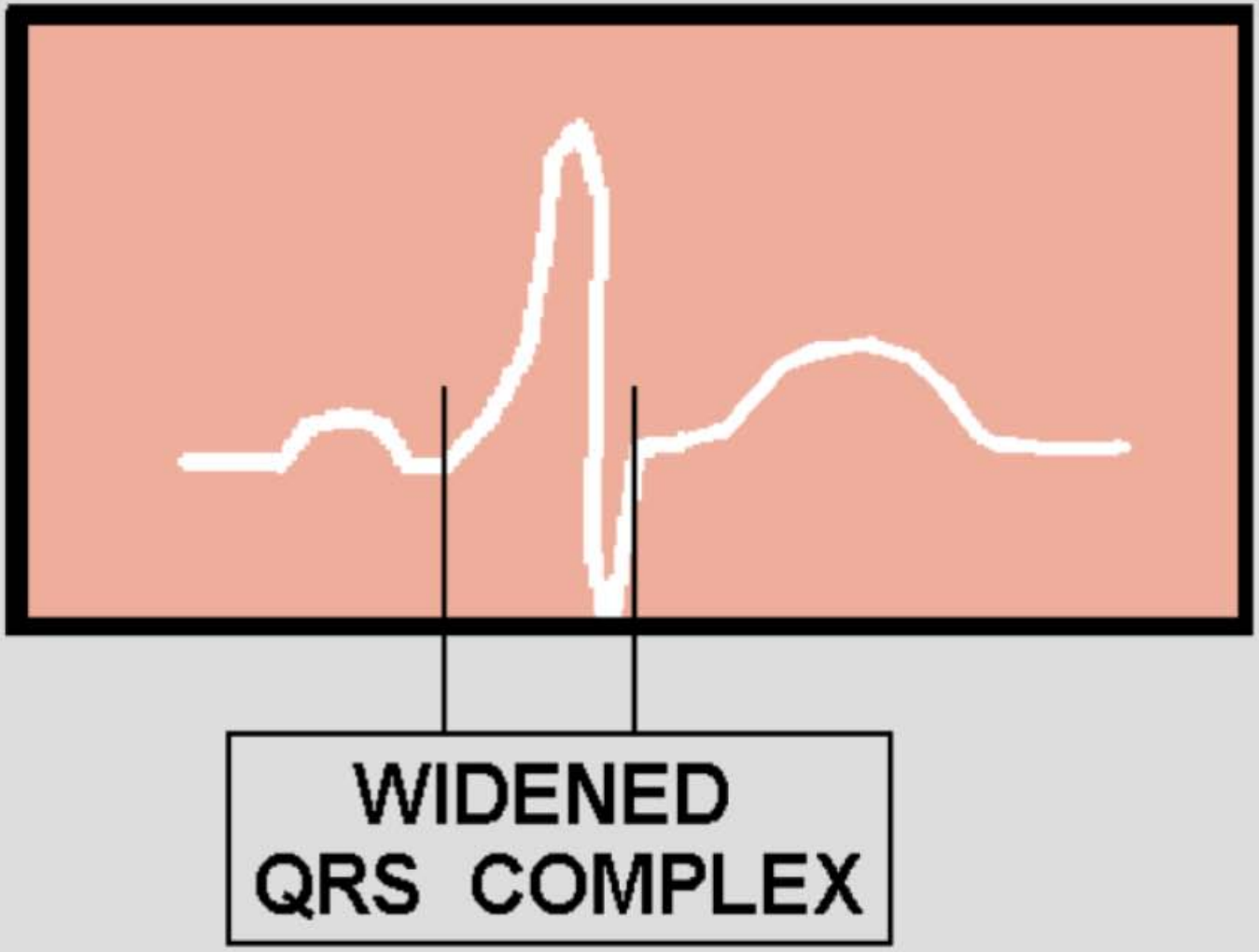
WOLFF-PARKINSON-WHITE

EKG CHARACTERISTICS



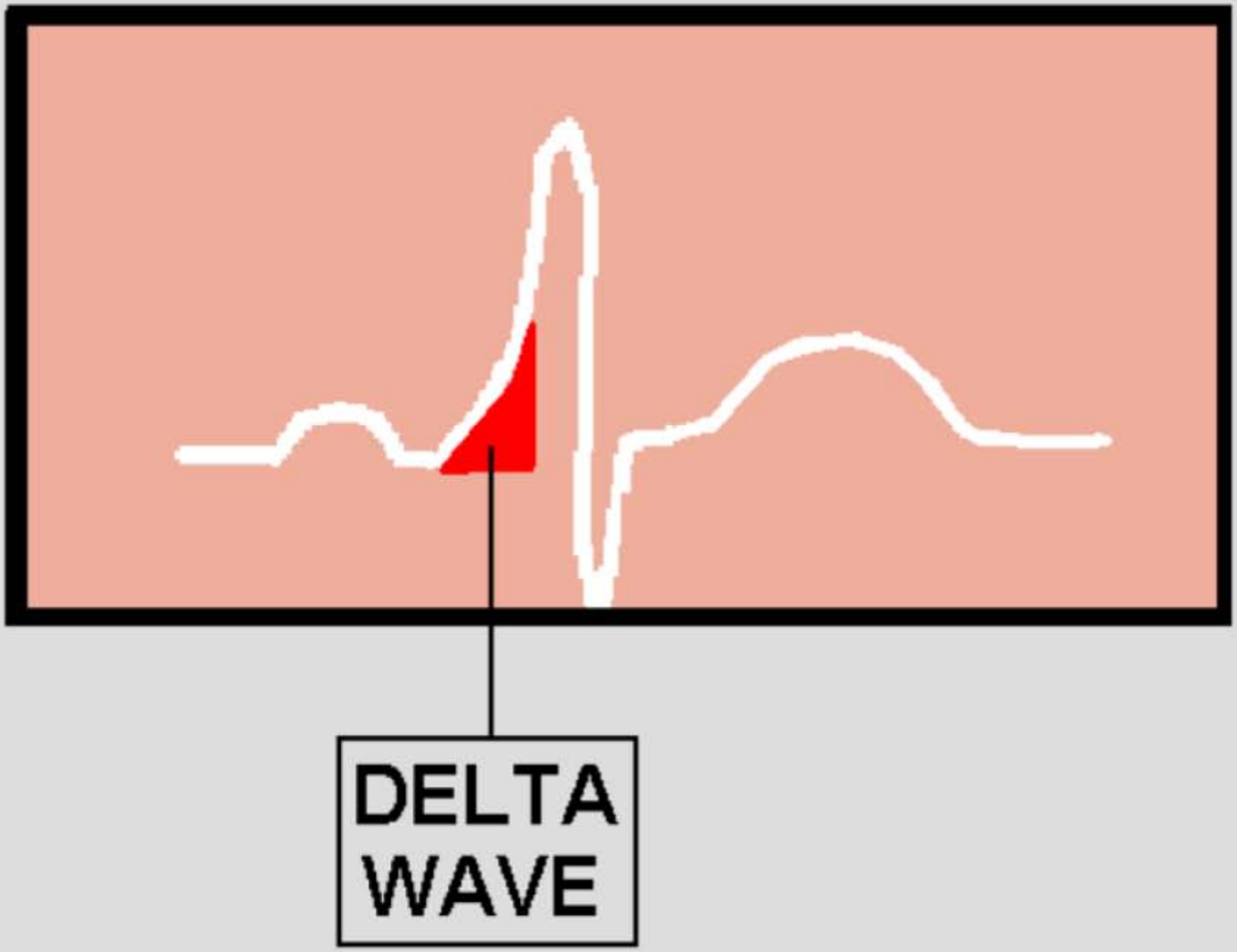
WOLFF-PARKINSON-WHITE

EKG CHARACTERISTICS



WOLFF-PARKINSON-WHITE

EKG CHARACTERISTICS



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

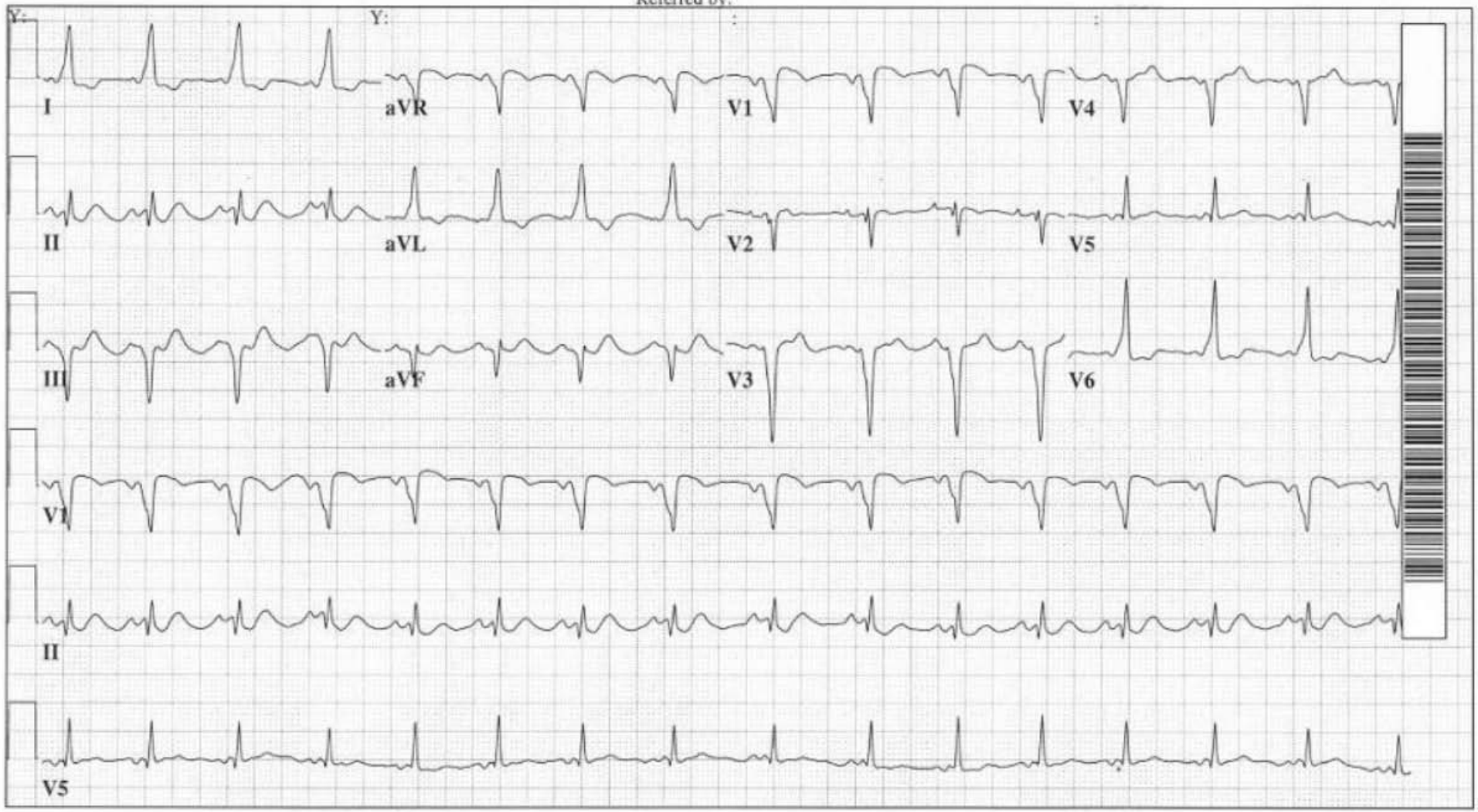
Normal sinus rhythm with sinus arrhythmia
Left atrial enlargement
Anterior infarct , age undetermined
Inferior infarct , age undetermined
ST & T wave abnormality, consider lateral ischemia
Wolff-Parkinson-White
Abnormal ECG
No previous ECGs available

History:Unknown
Technician: DP
Test ind:EKG

EKG CLASS #WR030100
60783

WOLFF-PARKINSON-WHITE TYPE B

Referred by:



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

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

16 yr
Female Caucasian

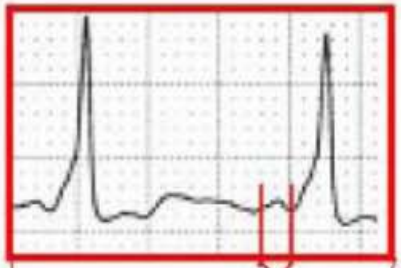
Room:REC
Loc:20 Option:50

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

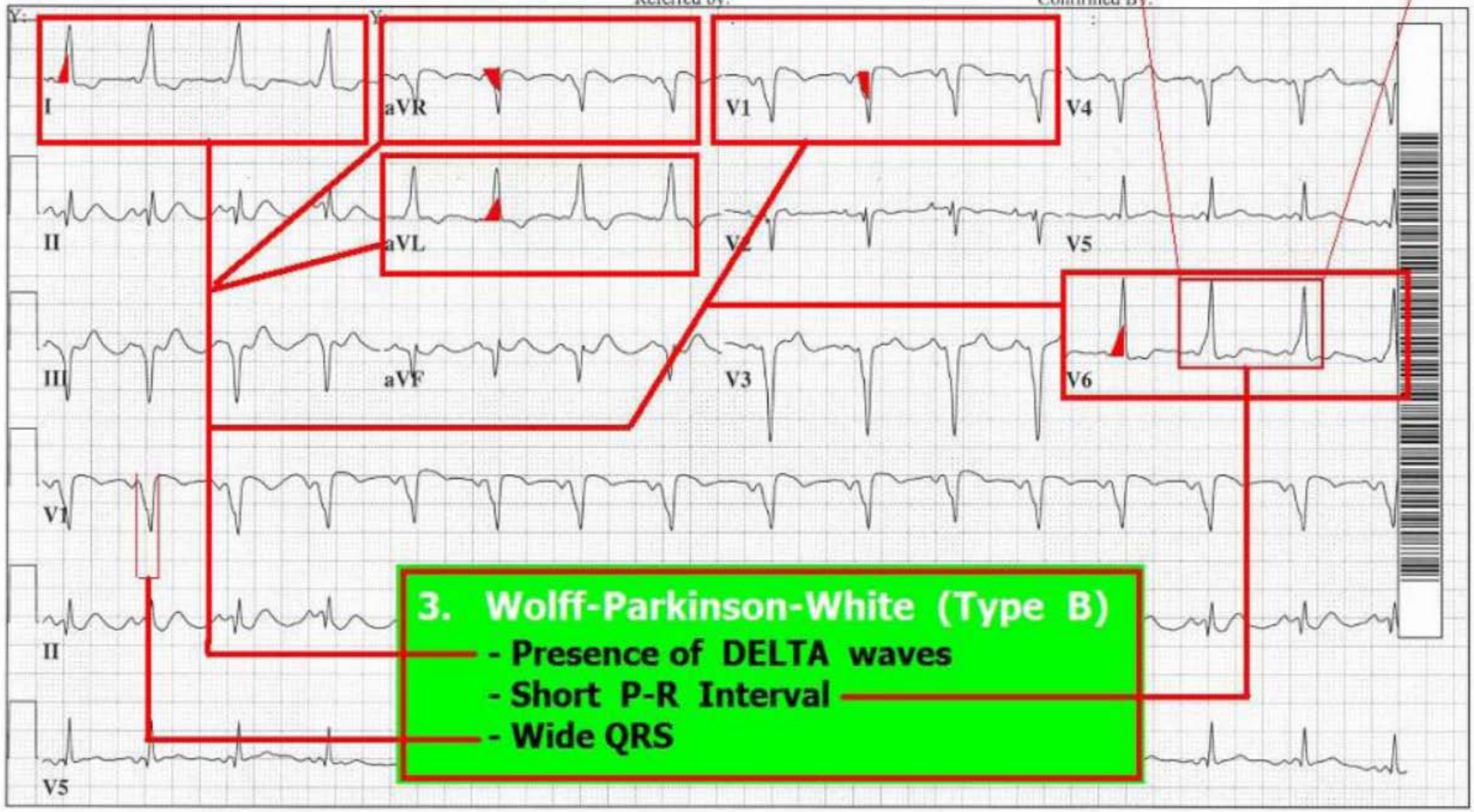
EKG CLASS #WR030100
60783



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:

Patient Profile: Wolff-Parkinson-White:

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

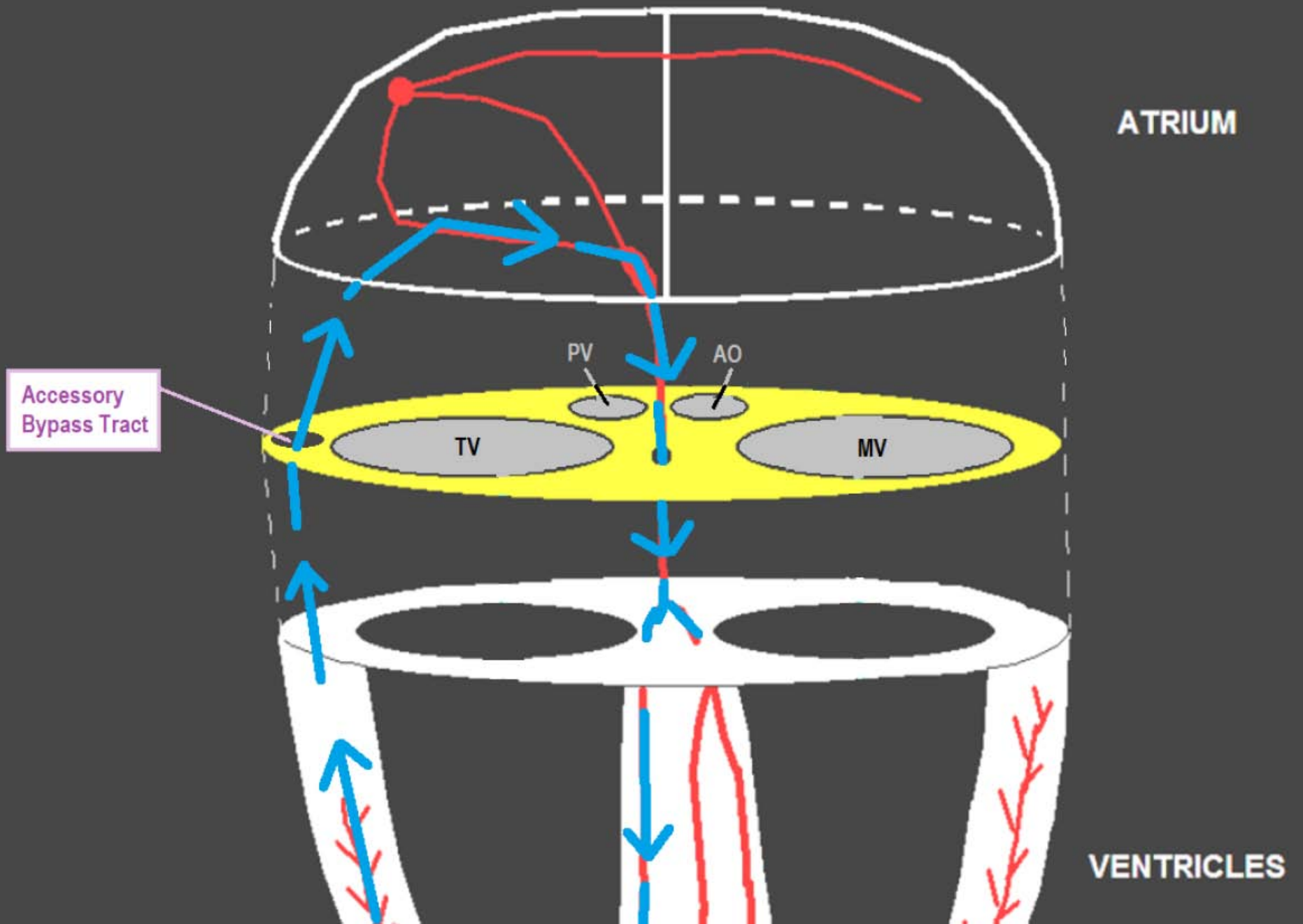
W-P-W may CAUSE A-fib with RVR.

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

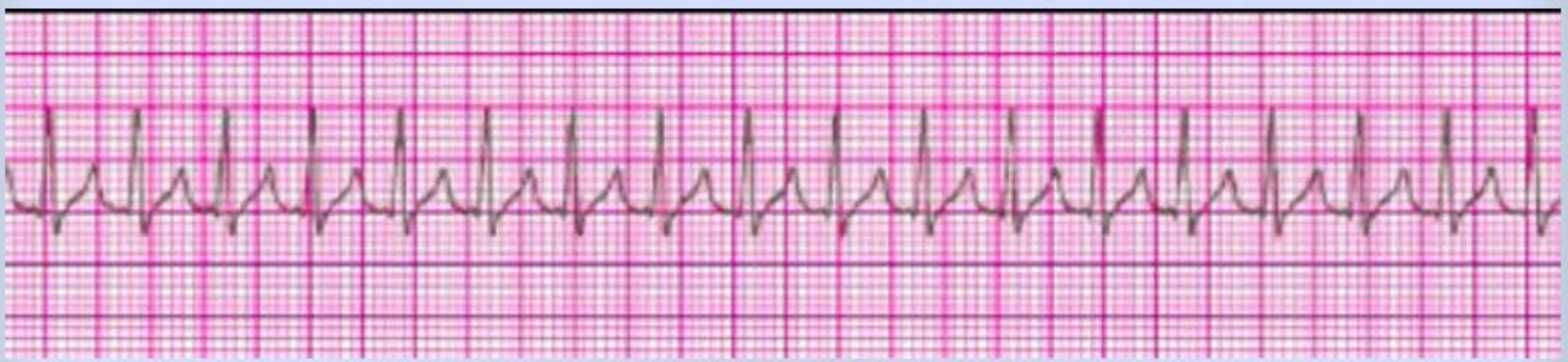
Patients with Bypass Tract Physiology (W-P-W) may present with:

- **Narrow QRS Tachycardia (SVT)**

Orthodromic Bypass Tract Tachycardia



SVT

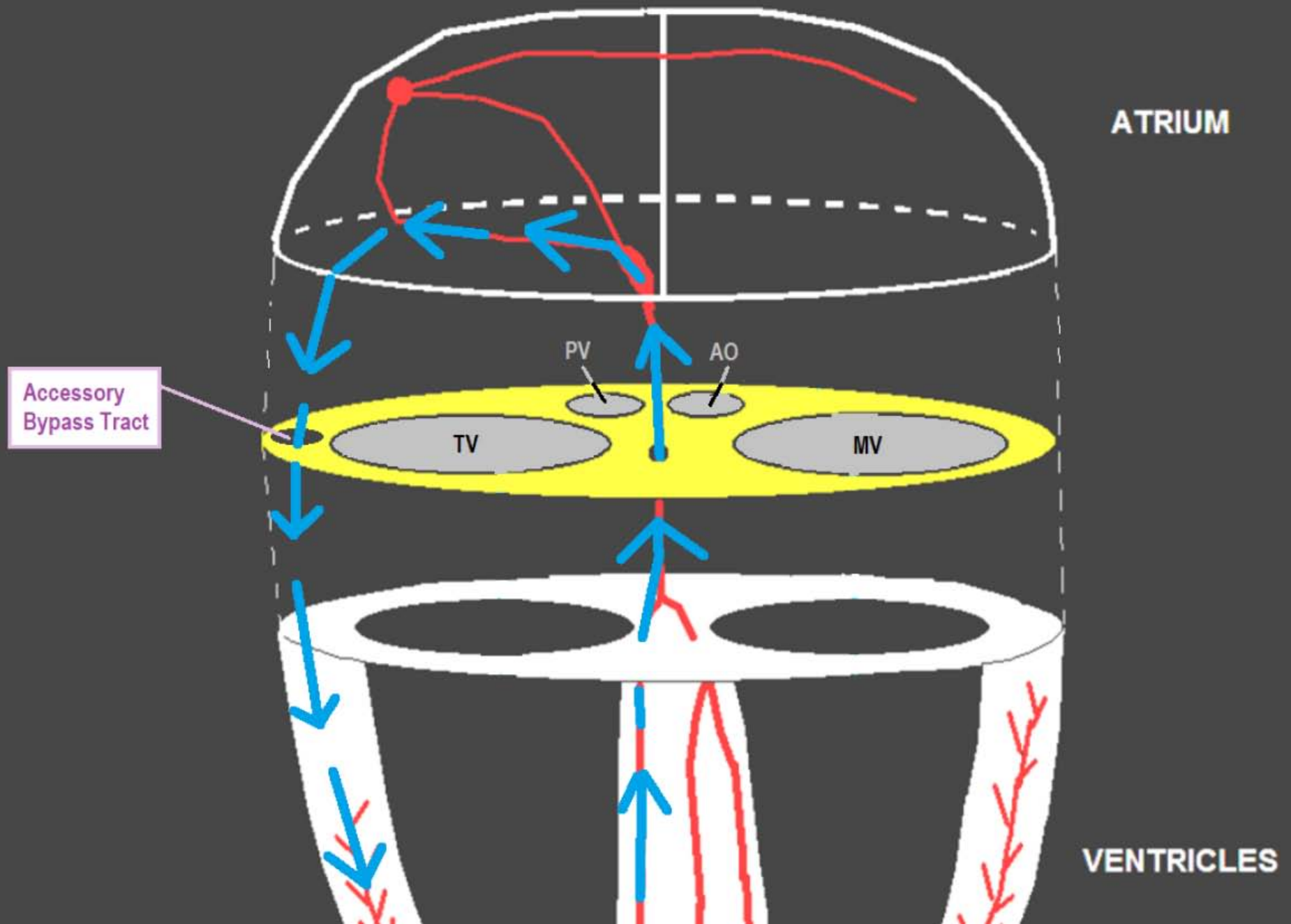


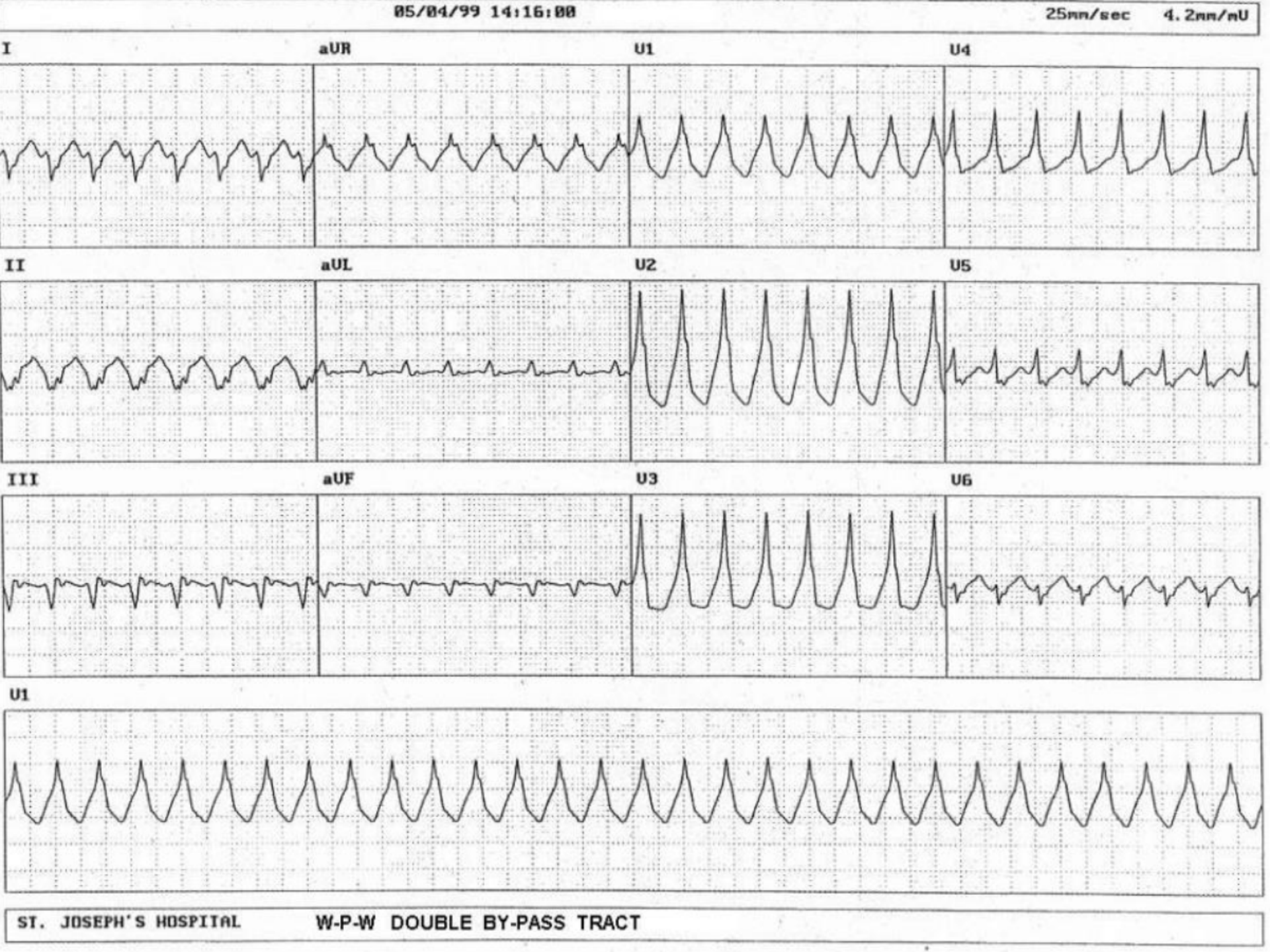
- SVT has numerous causes, including
  - Ectopic Atrial Tachycardia
  - AV Nodal Reentrant Tachycardia (Junctional)
  - Orthodromic Bypass Tract Tachycardia (WPW)
- EP study is often needed to diagnose etiology.

Patients with Bypass Tract Physiology (W-P-W) may present with:

- Narrow QRS Tachycardia (SVT)
- **Wide QRS Tachycardia (mimics V-tach)**

Antedromic Bypass Tract Tachycardia




05/04/99 14:16:00
25mm/sec
4.2mm/mU
I
aUR
U1
U4
II
aUL
U2
U5
III
aUF
U3
U6
U1
ST. JOSEPH'S HOSPITAL
W-P-W DOUBLE BY-PASS TRACT

Patients with Bypass Tract Physiology (W-P-W) may present with:

- Narrow QRS Tachycardia (SVT)
- Wide QRS Tachycardia (mimics V-tach)
- **Atrial Fib with RVR – and a WIDE QRS . . . .**

37 y/o male

Chief Complaint: Lightheadedness, Palpitations, Shortness of Breath

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

PMH: HTN (non-compliant)

37 y/o male

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

Meds: None, NKDA

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

ST. JOSEPH'S HOSPITAL-

37 yr
Male Caucasian

Room:OP
Loc:8 Option:16

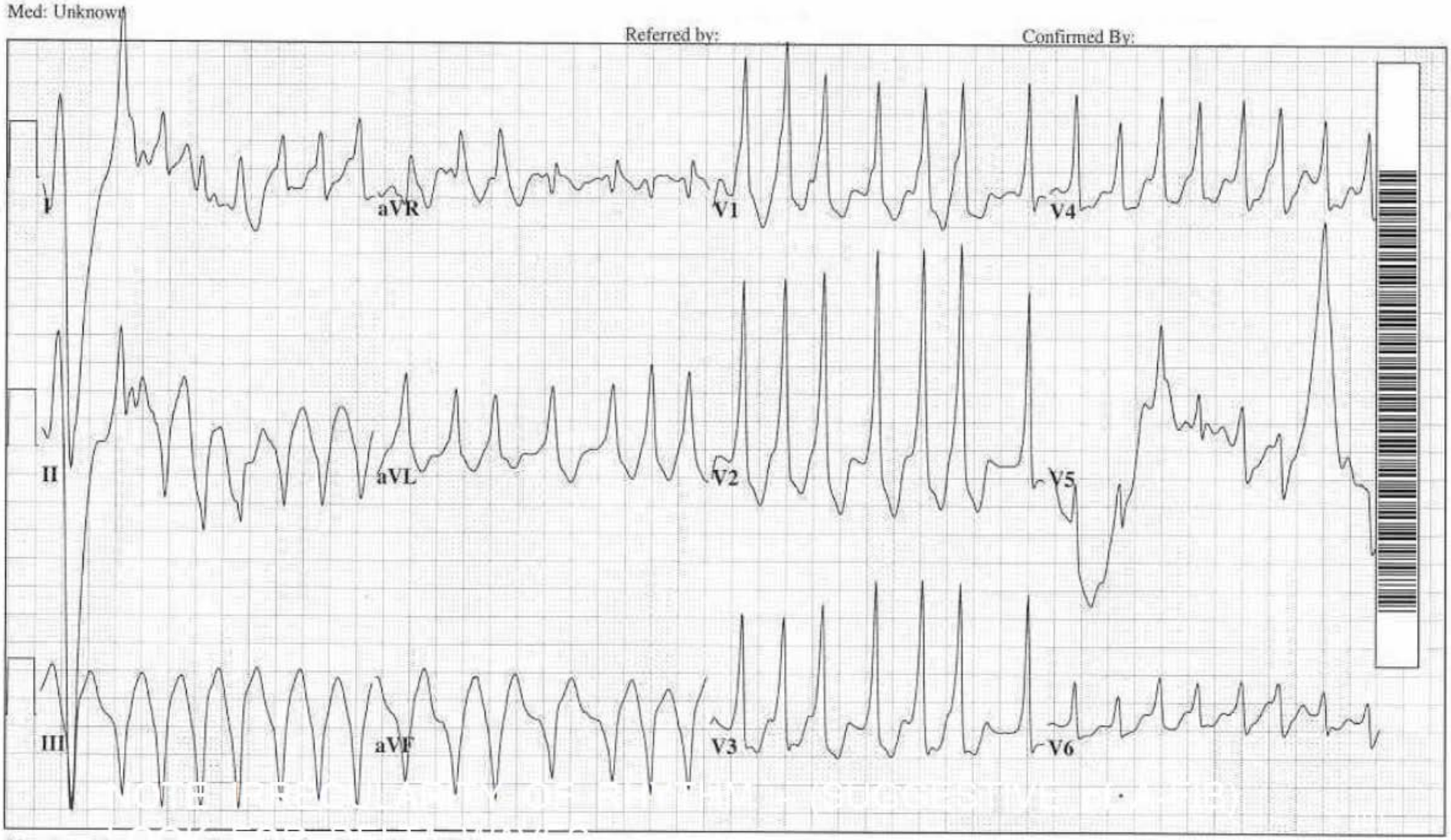
Vent. rate	180	BPM
PR interval	*	ms
QRS duration	148	ms
QT/QTc	284/491	ms
P-R-T axes	* -77	103

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

Med: Unknown

Referred by:

Confirmed By:



25mm/s 10mm/mV 60Hz 305C 12SL 72 CID: 3

Physician correctly identified Atrial Fibrillation with Rapid Ventricular Response.

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

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

37 y/o male

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

A 12 Lead EKG obtained revealed . . . . .

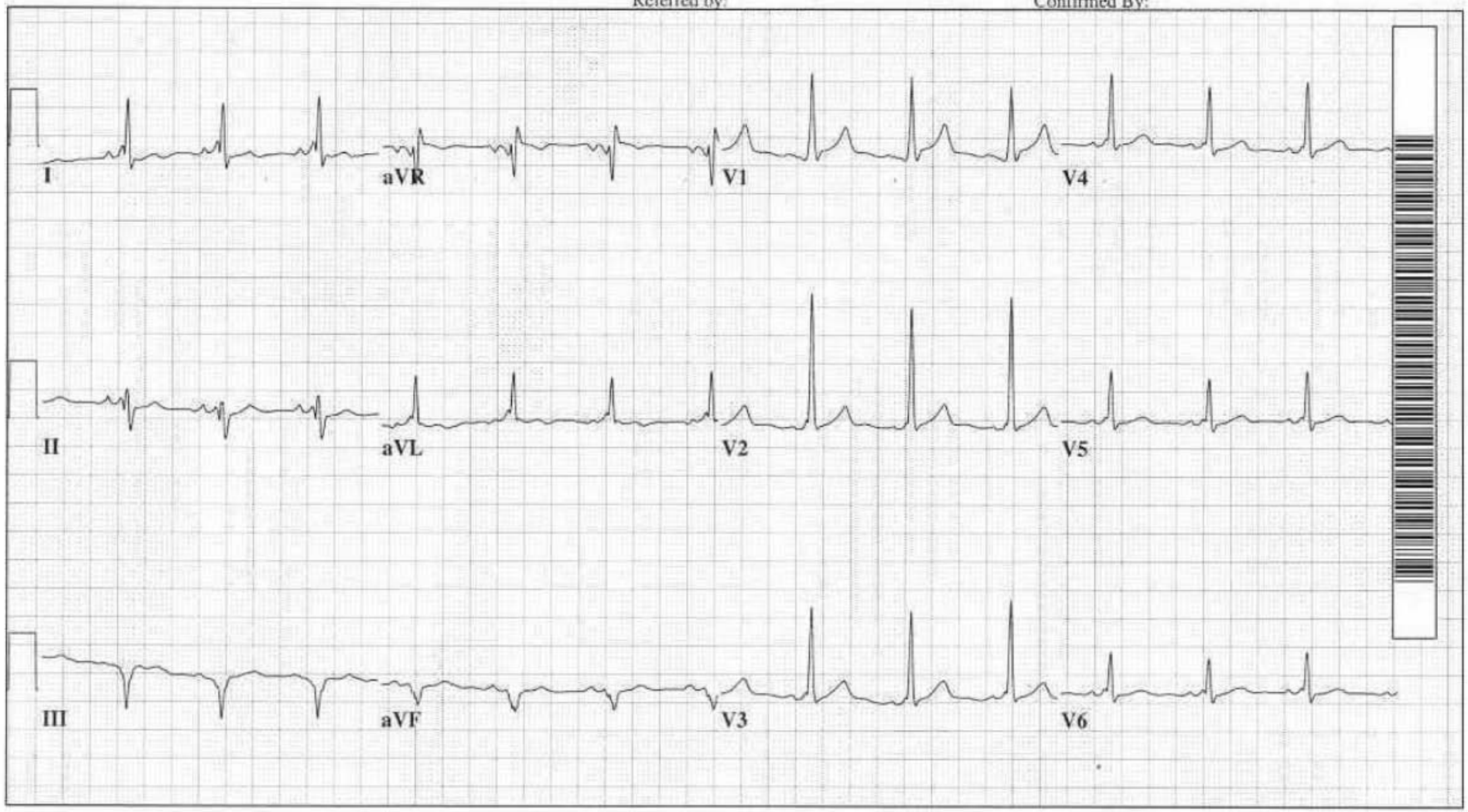
37 yr
Male Caucasian

Room:OP
Loc:8 Option:19

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

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

Referred by: Confirmed By:



25mm/s 10mm/mV 40Hz 005C 12SL 78 CID: 0

ST. JOSEPH'S HOSPITAL-

37 yr
Male Caucasian

Room:OP
Loc:8 Option:16

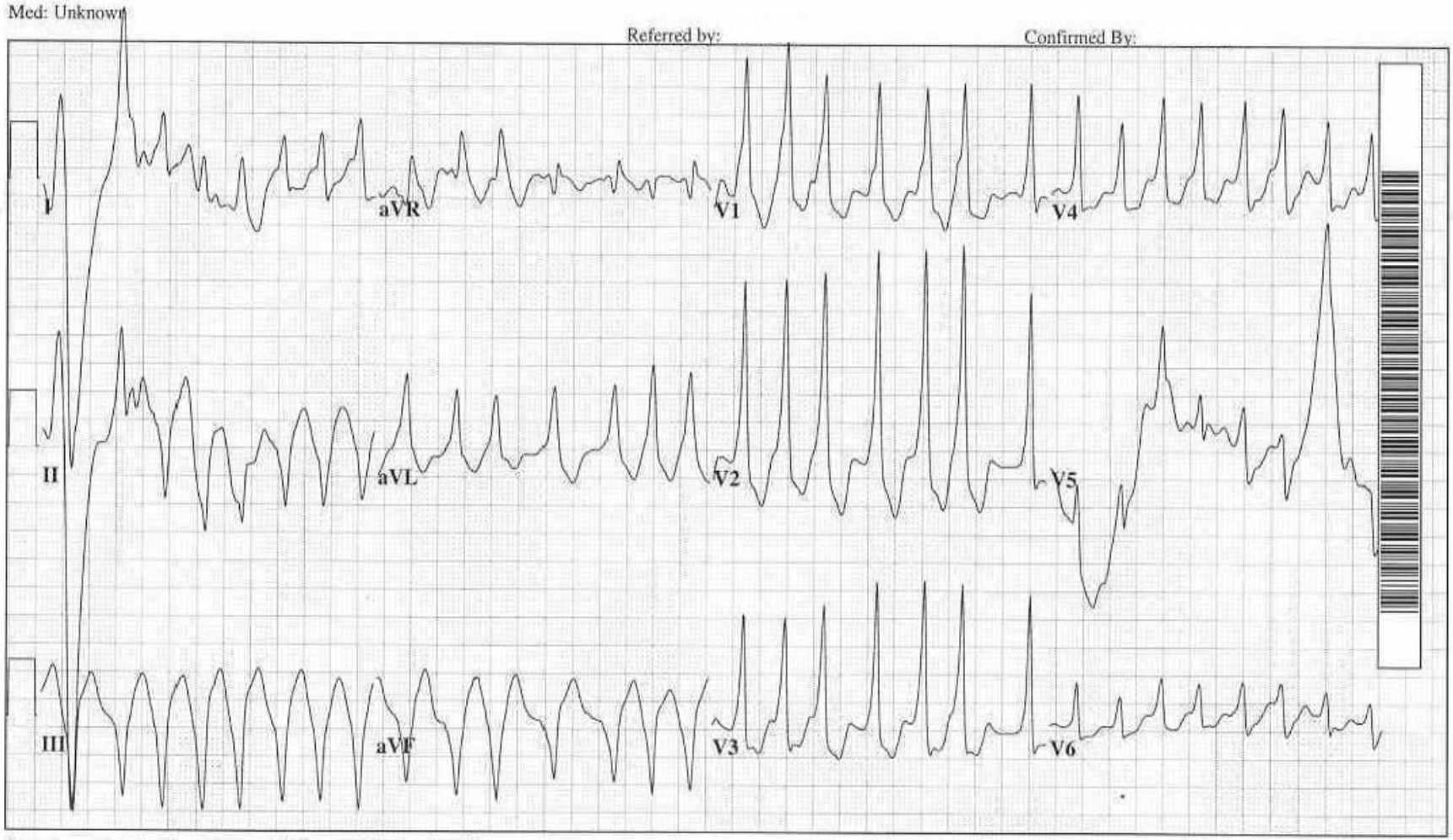
Vent. rate	180	BPM
PR interval	*	ms
QRS duration	148	ms
QT/QTc	284/491	ms
P-R-T axes	* -77	103

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

Med: Unknown

Referred by:

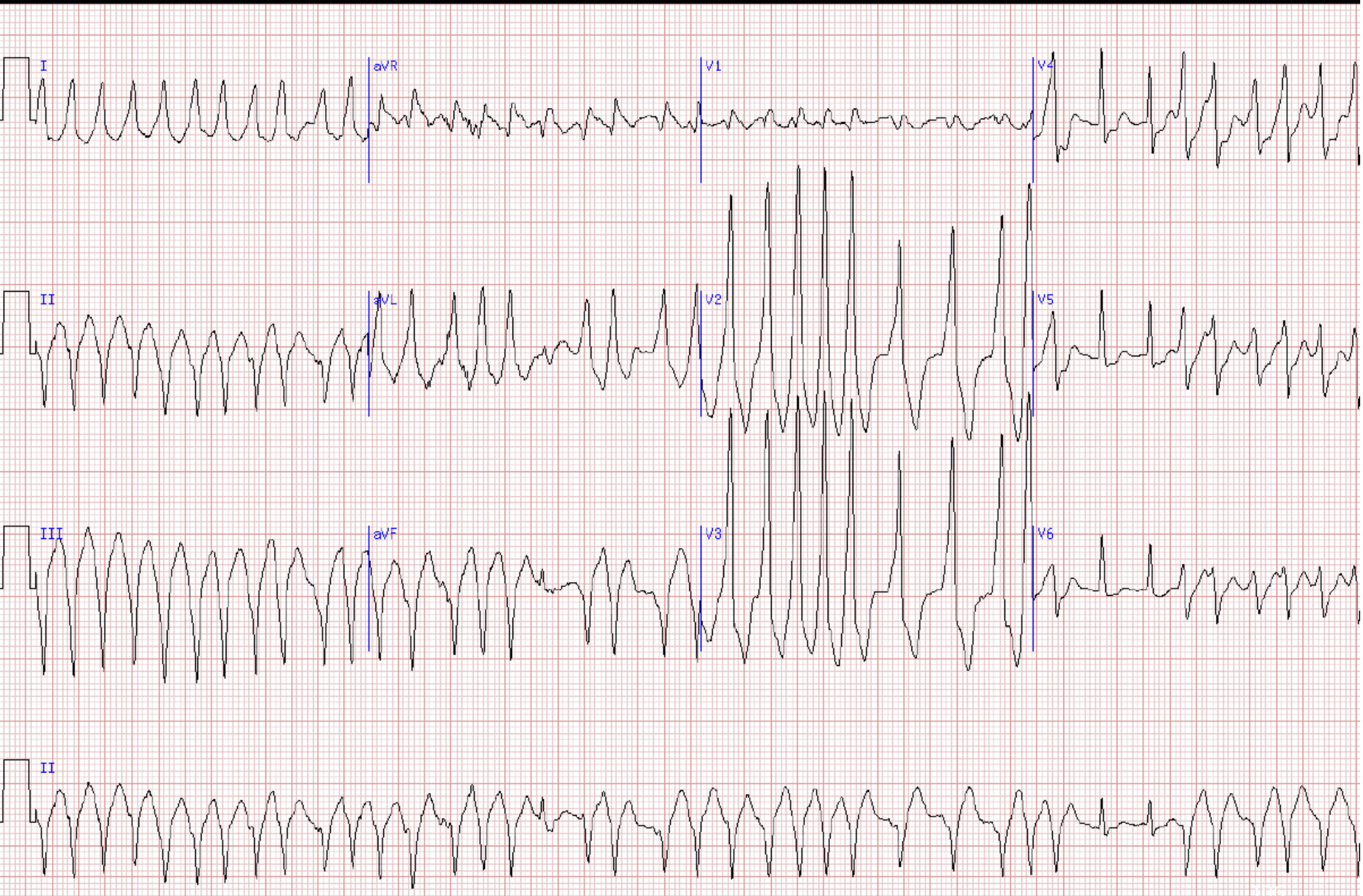
Confirmed By:



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

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

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

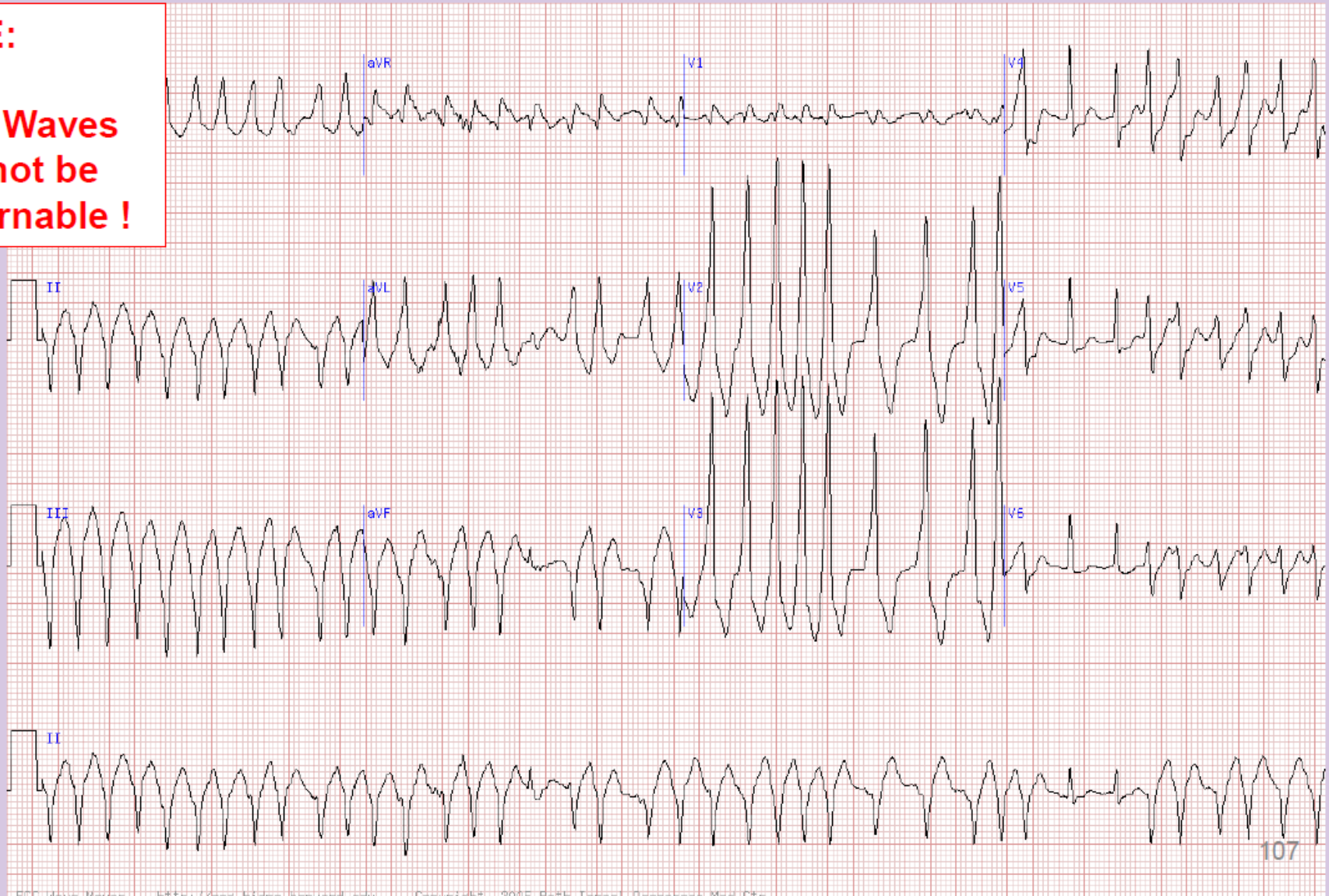


CHARACTERISTICS of W-P-W with Afib & RVR:

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

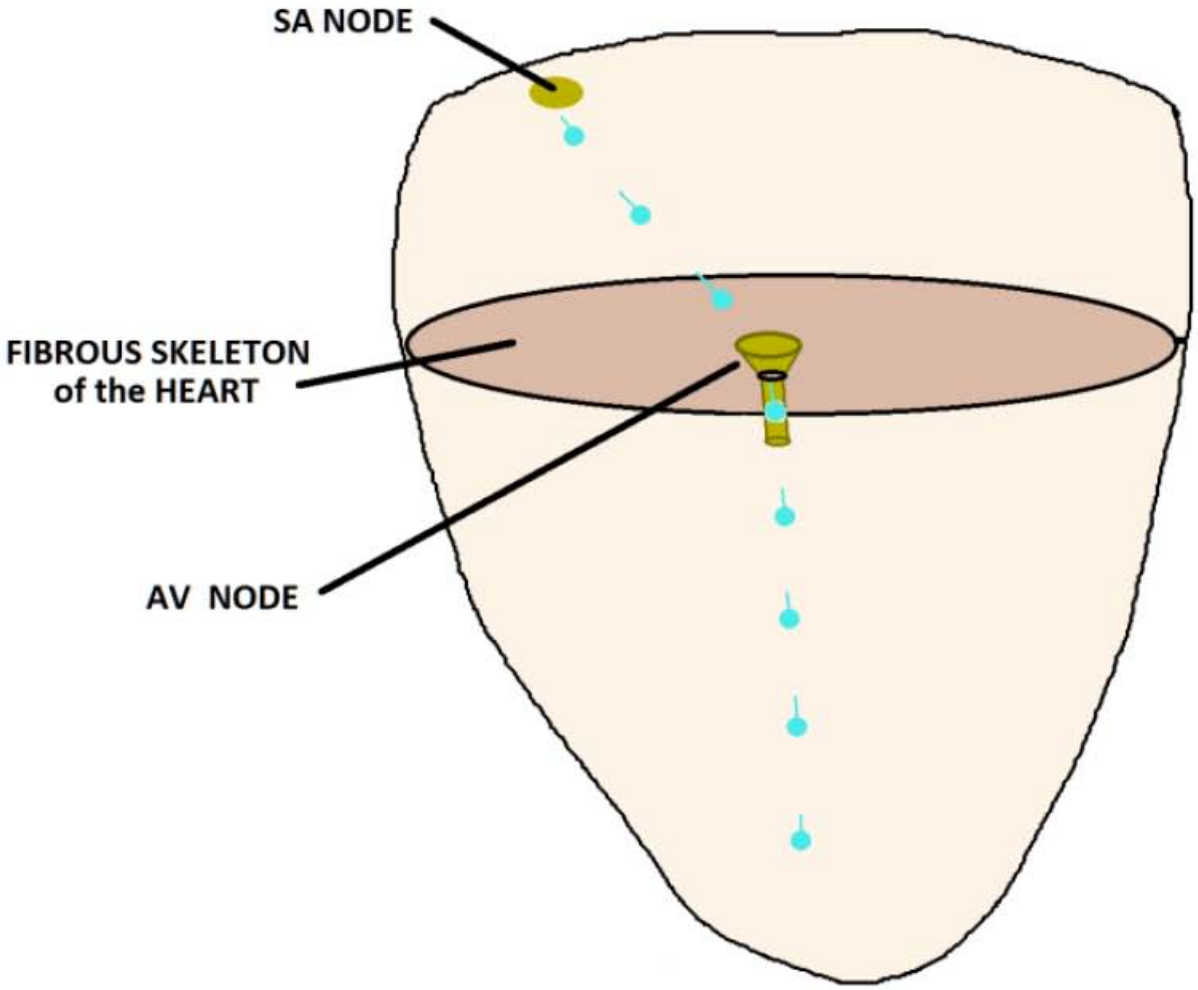
NOTE:

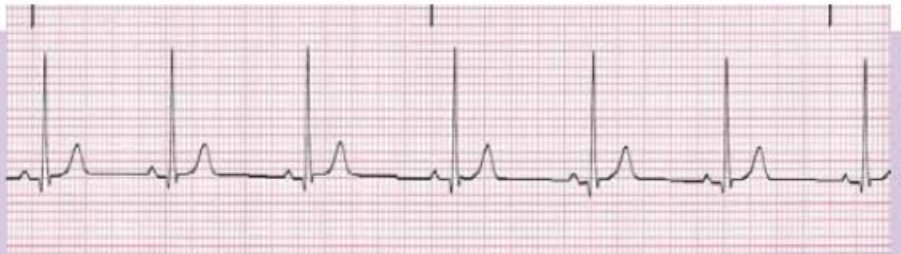
Delta Waves may not be discernable !



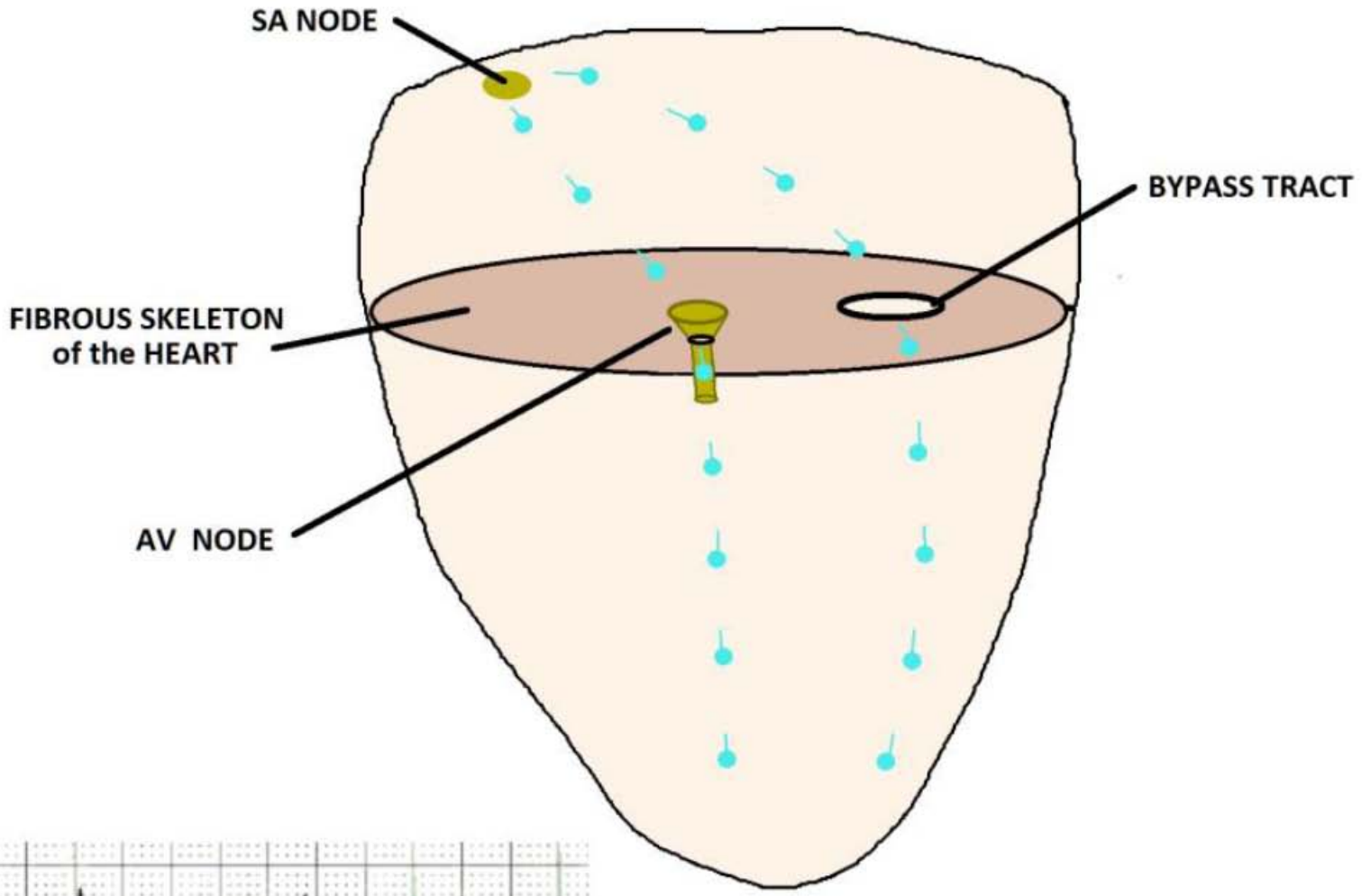
NO AV NODAL BLOCKERS (e.g. ADENOSINE, CALCIUM CHANNEL BLOCKERS) FOR WIDE COMPLEX TACHYCARDIAS THAT COULD BE ATRIAL FIBRILLATION with Pre-Excitation (W-P-W)

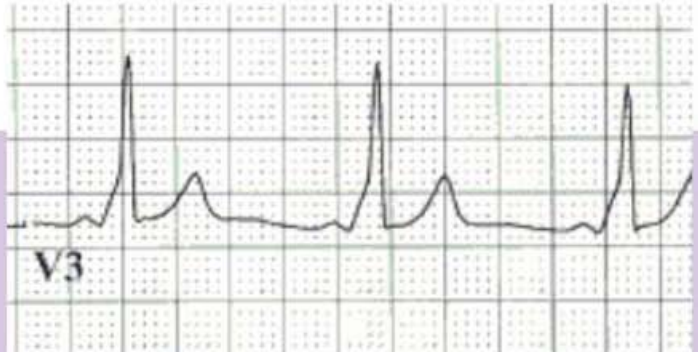
Normal Sinus Rhythm



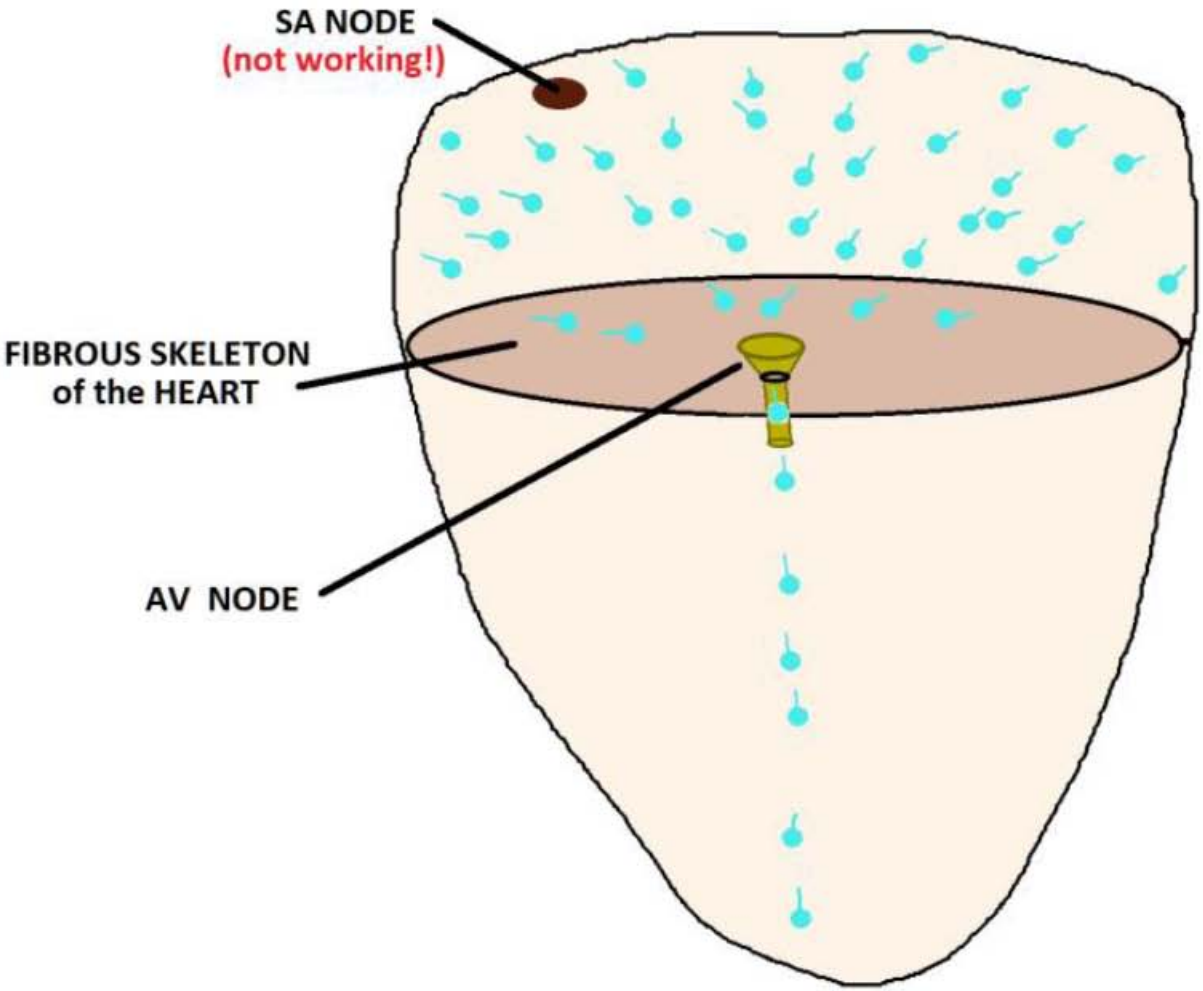


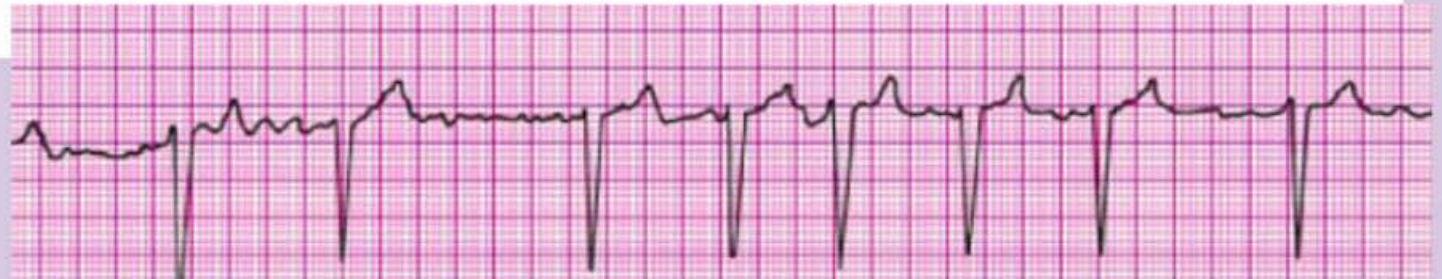
Normal Sinus Rhythm with Wolff-Parkinson White



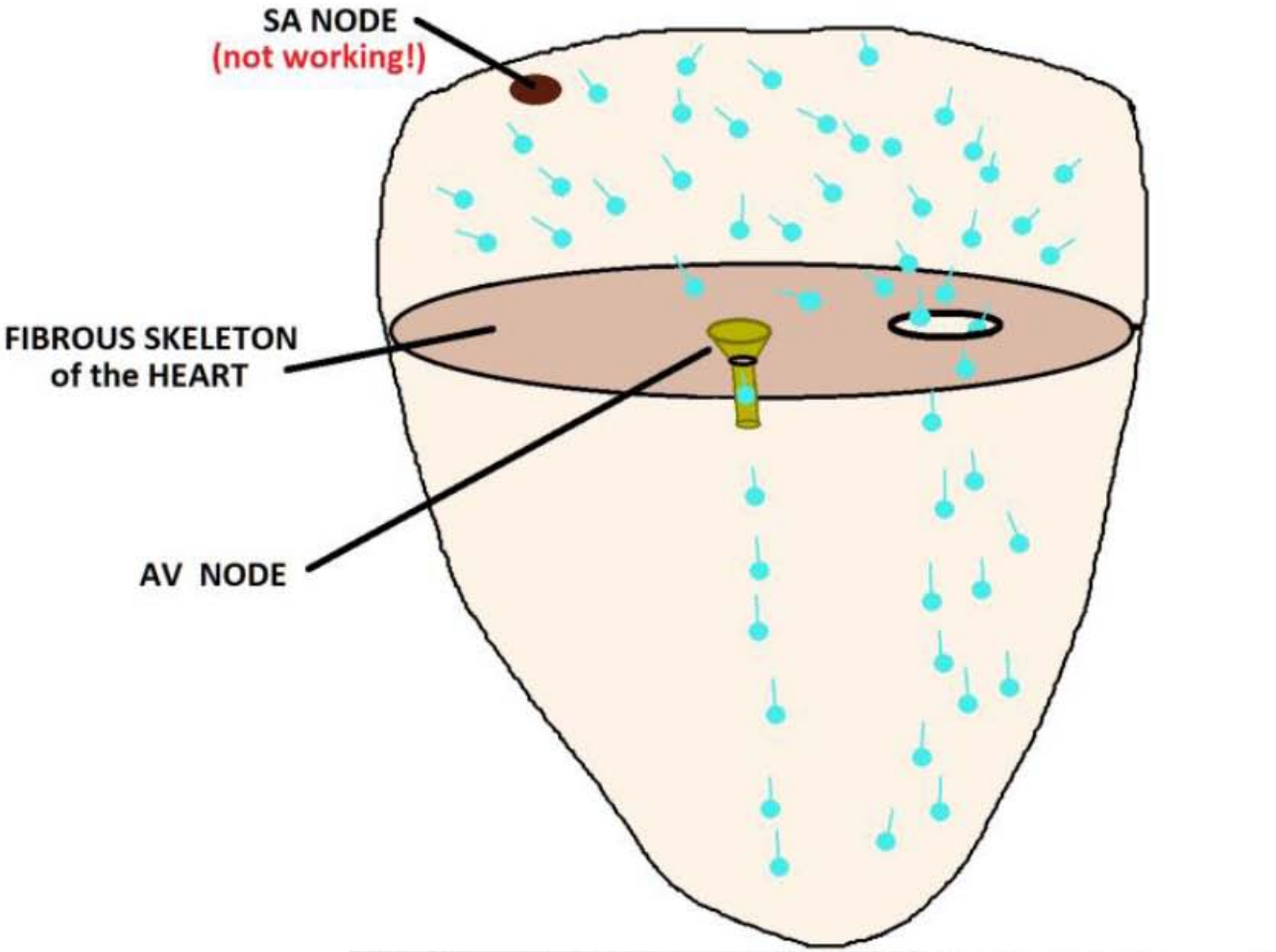


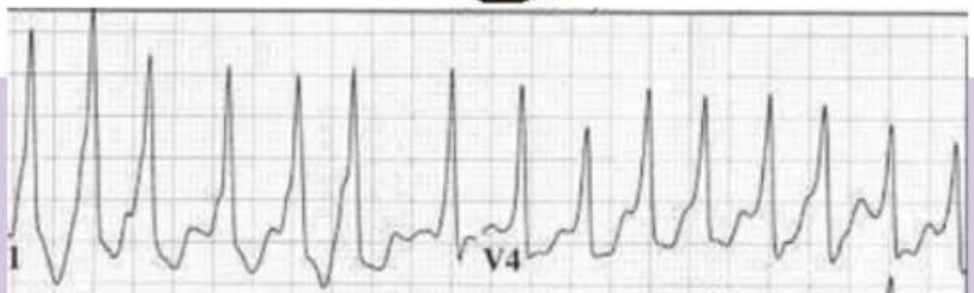
Atrial Fibrillation





Atrial Fibrillation with Wolff-Parkinson White

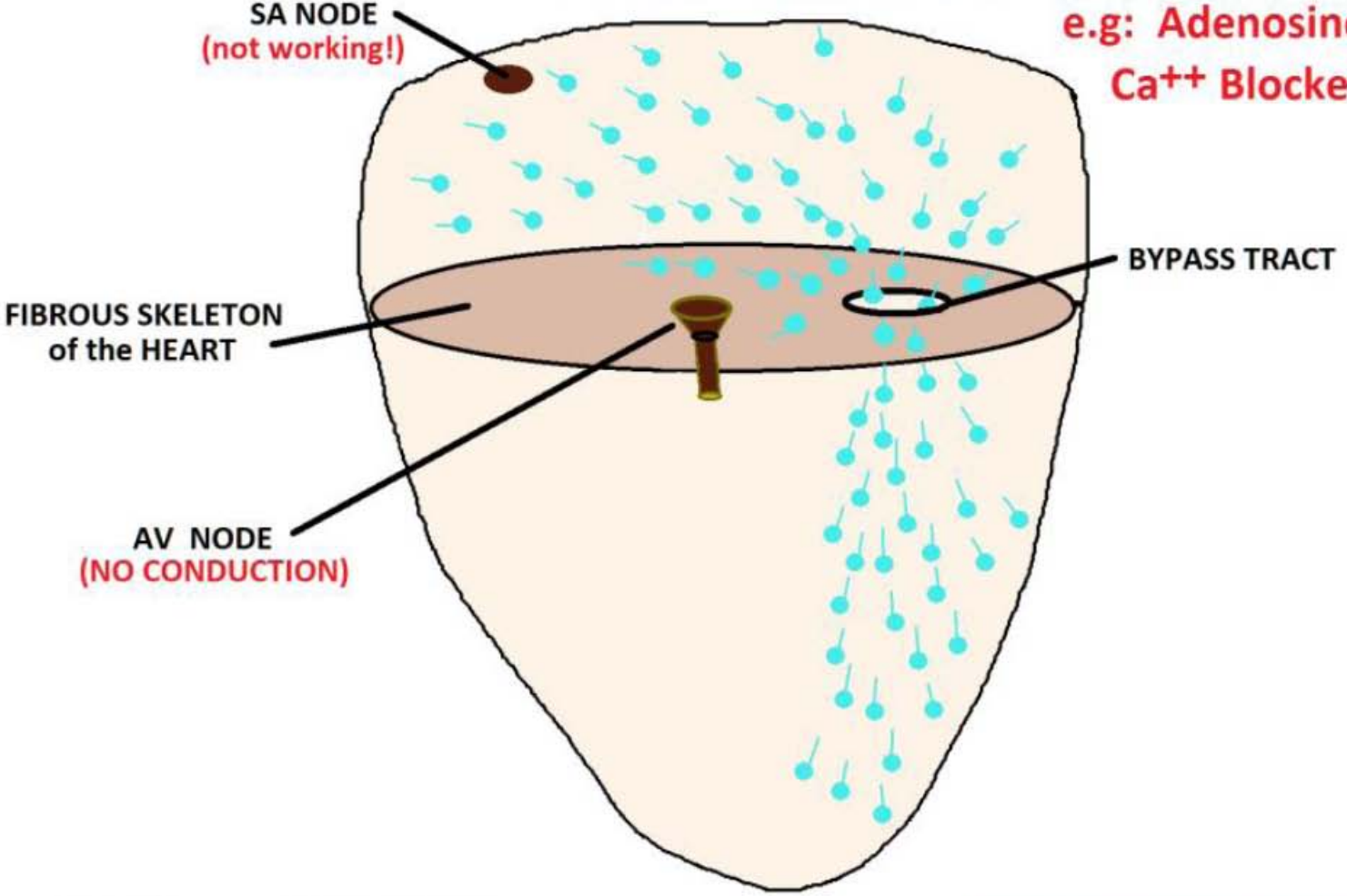


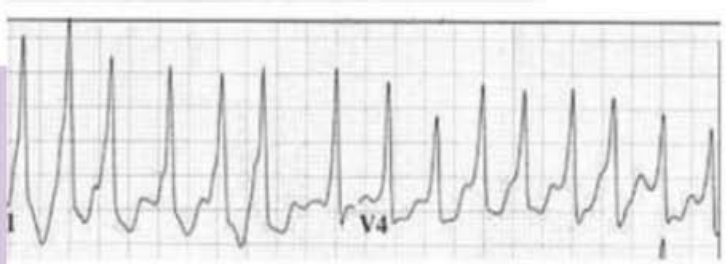


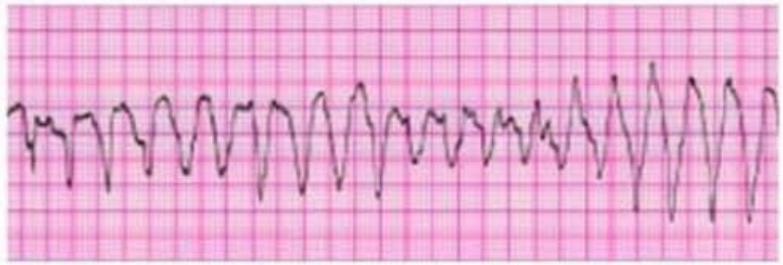
Atrial Fibrillation with Wolff-Parkinson White

with AV NODAL BLOCKING AGENTS

e.g: Adenosine,
Ca^{++} Blockers



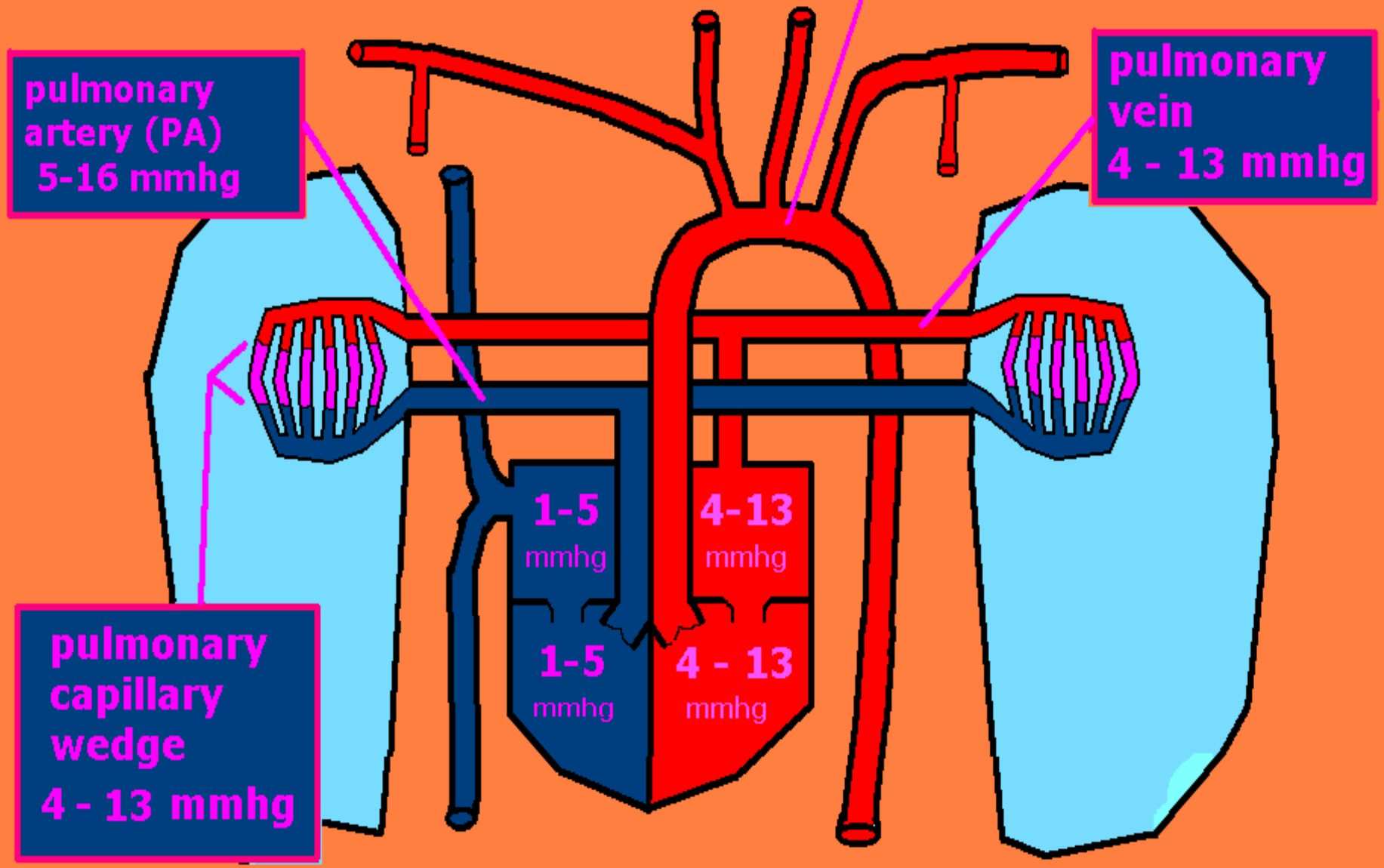




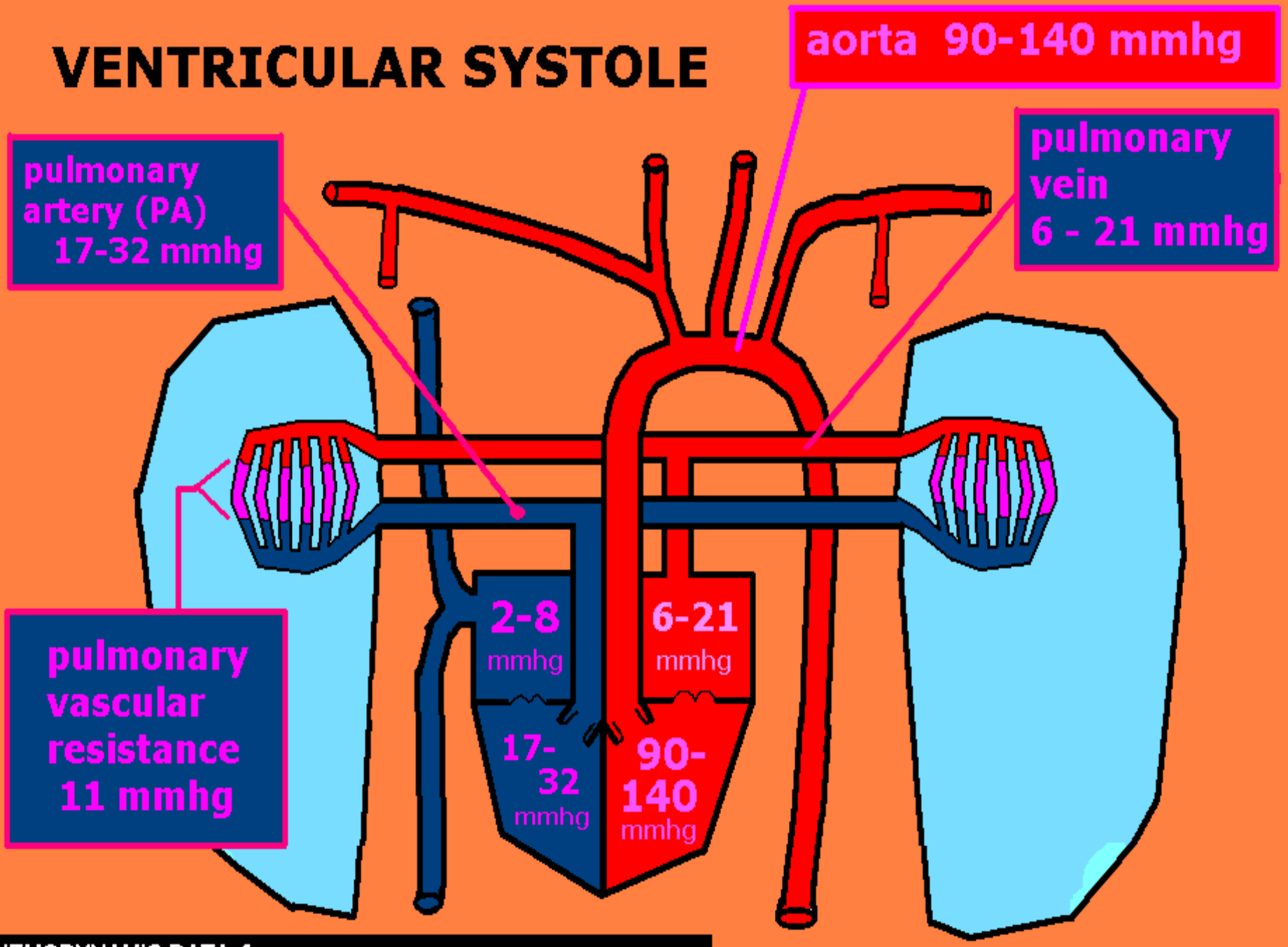
Cardiac A & P "101"...

- Heart structure – basic 4 chambers
- Action Potential of Ventricular Muscle Cells
- Electrical System overview
- Fibrous Skeleton of Heart
- Pathophysiology of Accessory Bypass Tracts (cause of Wolff-Parkinson-White Syndrome)
- **Normal Pressures with Heart and Lungs**
- **Heart Sounds**

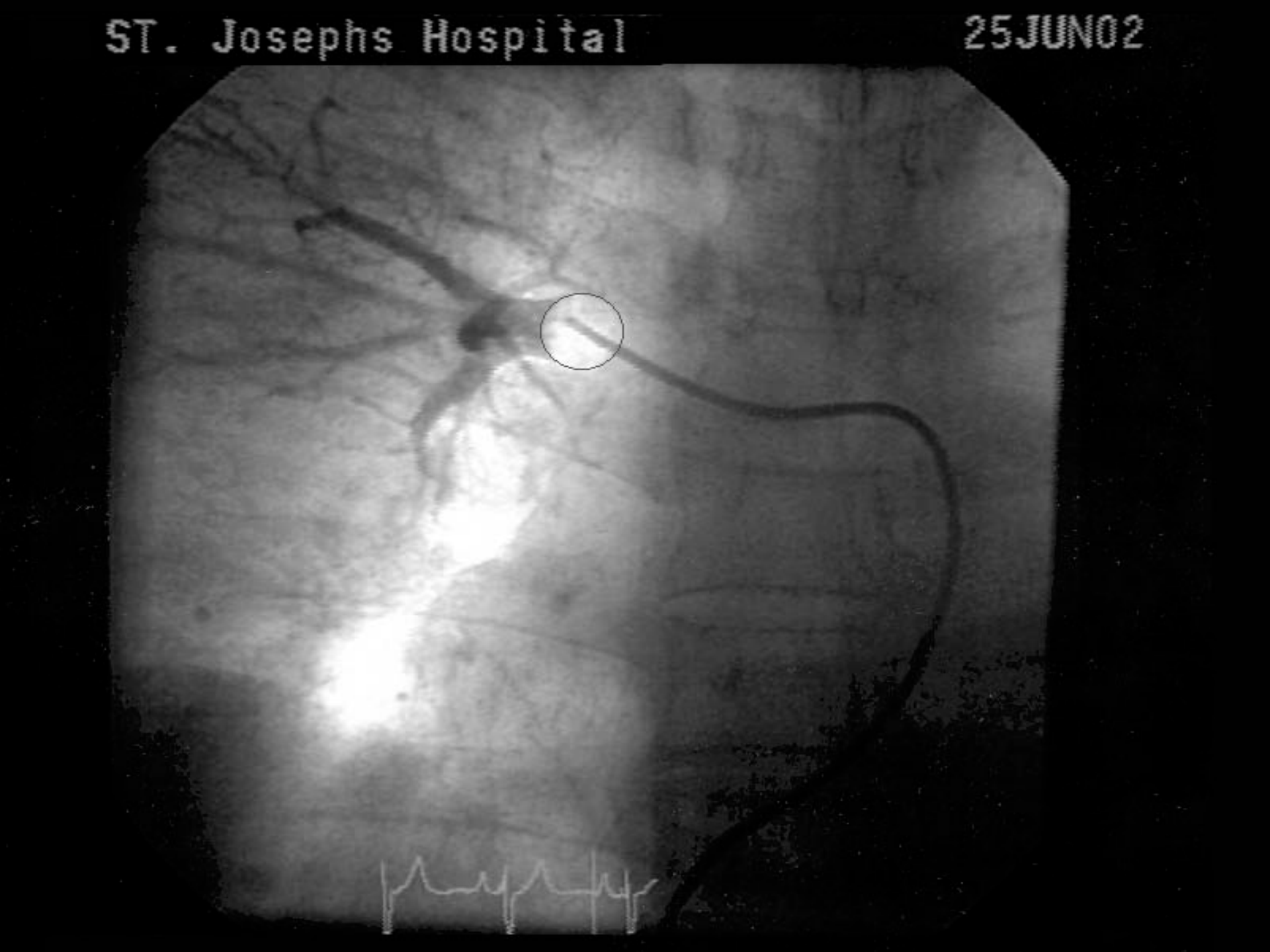
DIASTOLE


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

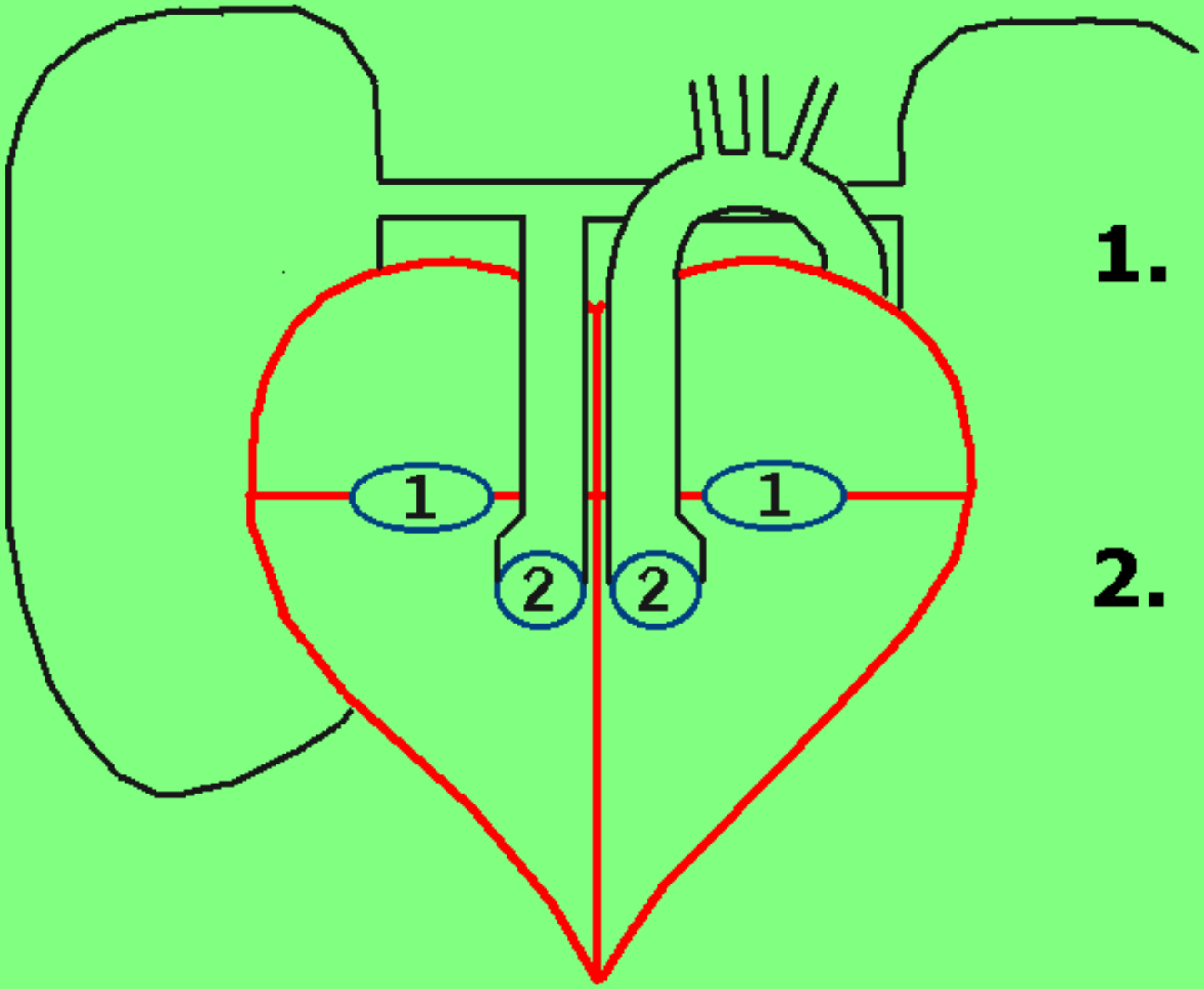
VENTRICULAR SYSTOLE



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


ST. Josephs Hospital
25JUN02

HEART VALVES

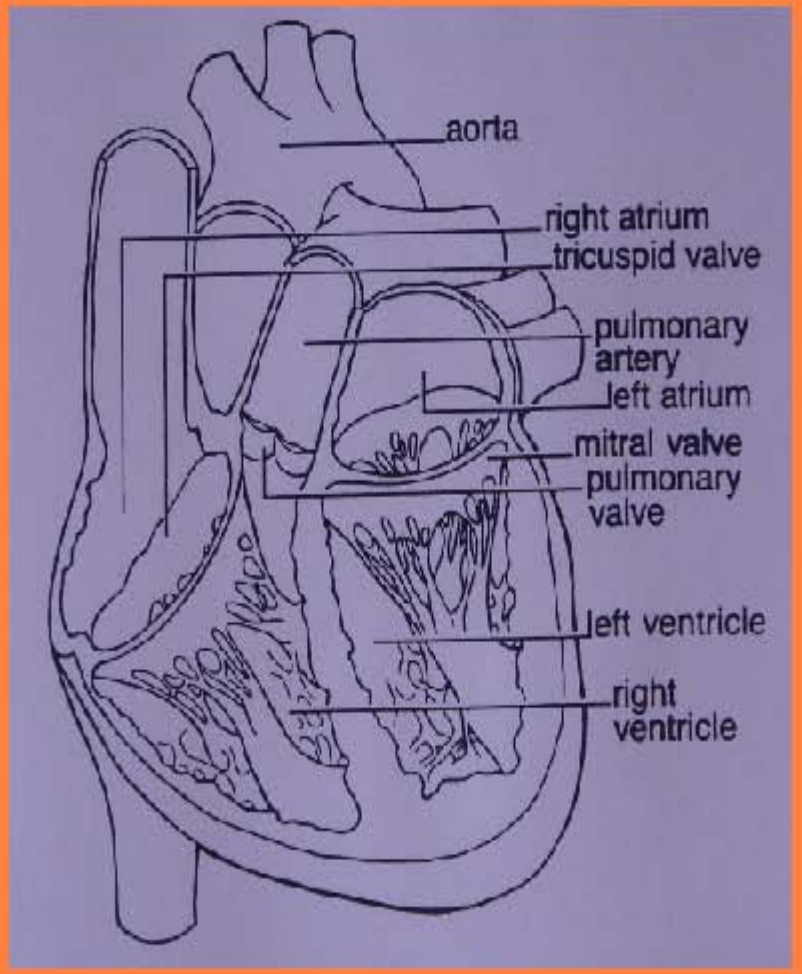


1. A-V VALVES

2. SEMI-LUNAR VALVES

THE ATRIO-VENTRICULAR VALVES

- ARE SECURED TO THE INFERIOR WALL OF THE HEART BY THE PAPILLARY MUSCLES



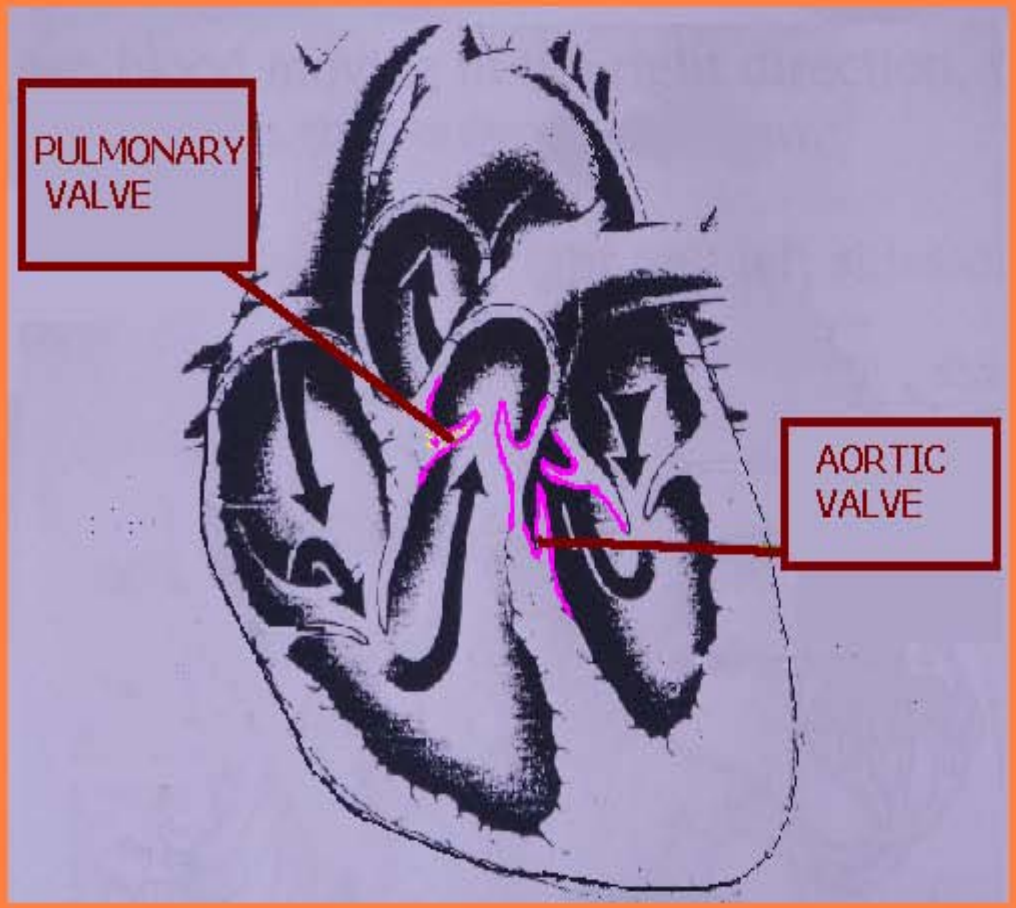
THE ATRIO-VENTRICULAR VALVES

VULNERABLE TO DAMAGE FROM TISSUE NECROSIS, SUCH AS FROM EXTENSIVE MI.

THE SEMI-LUNAR VALVES

PREVENT BACK-FLOW OF BLOOD AFTER SYSTOLE.

ARE NOT AS VULNERABLE TO DAMAGE FROM MI AS THE AV VALVES.



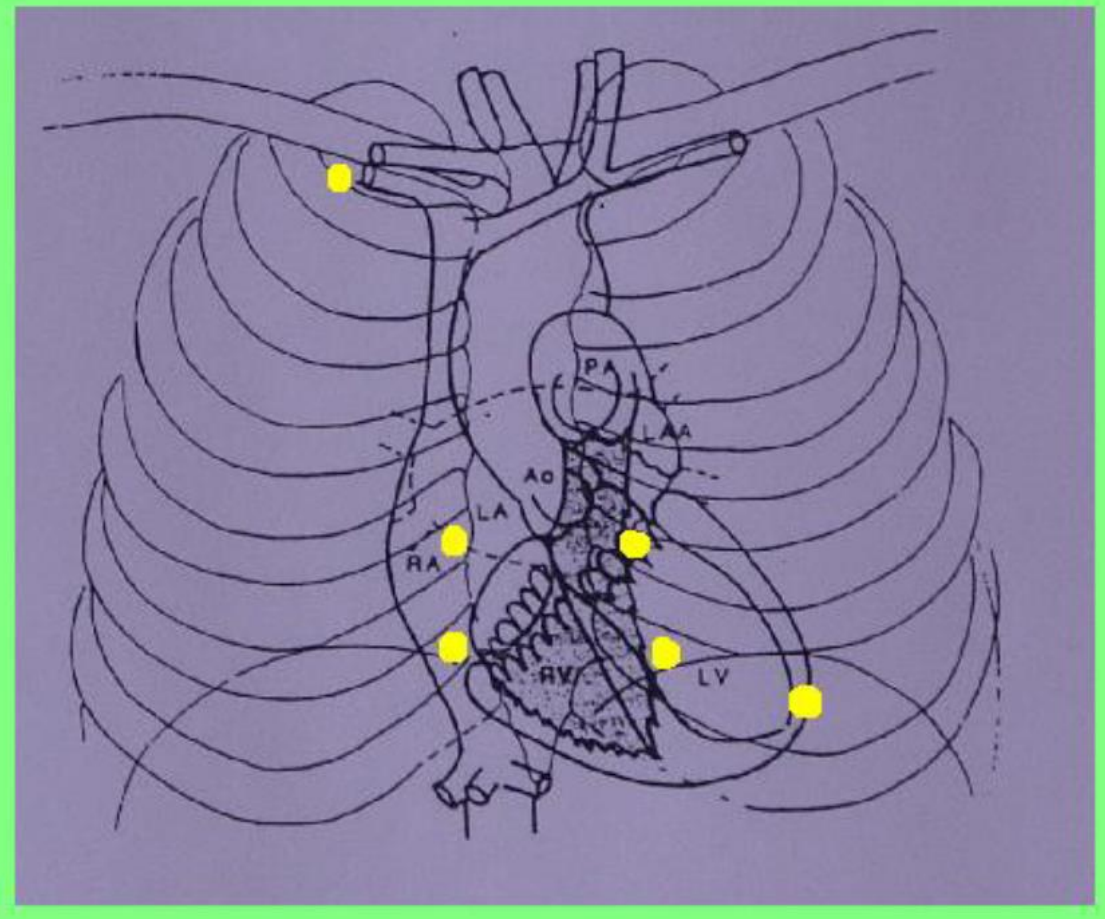
VERY BASIC HEART SOUNDS ASSESSMENT

ABNORMAL EKG CHANGES THAT MAY PRESENT WITH ABNORMAL HEART SOUNDS:

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

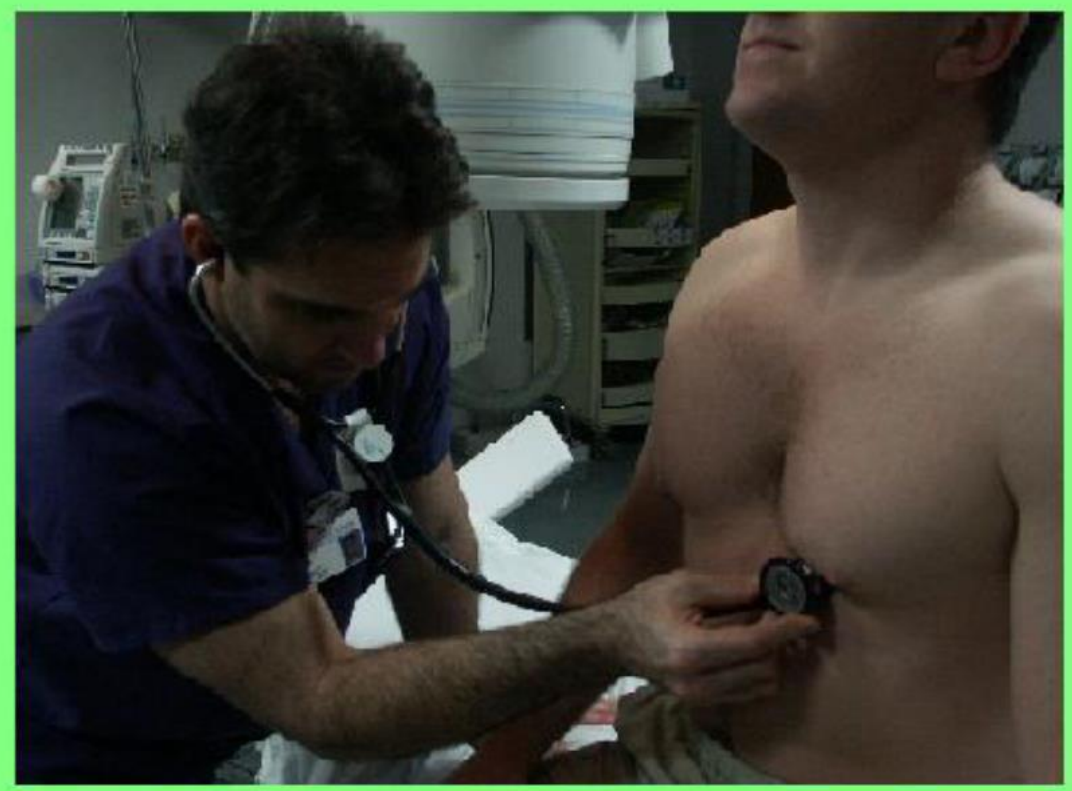
HEART SOUNDS ASSESSMENT

HEART SOUNDS ASSESSMENT



VERY BASIC HEART SOUNDS ASSESSMENT

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



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

HEART SOUNDS ASSESSMENT

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

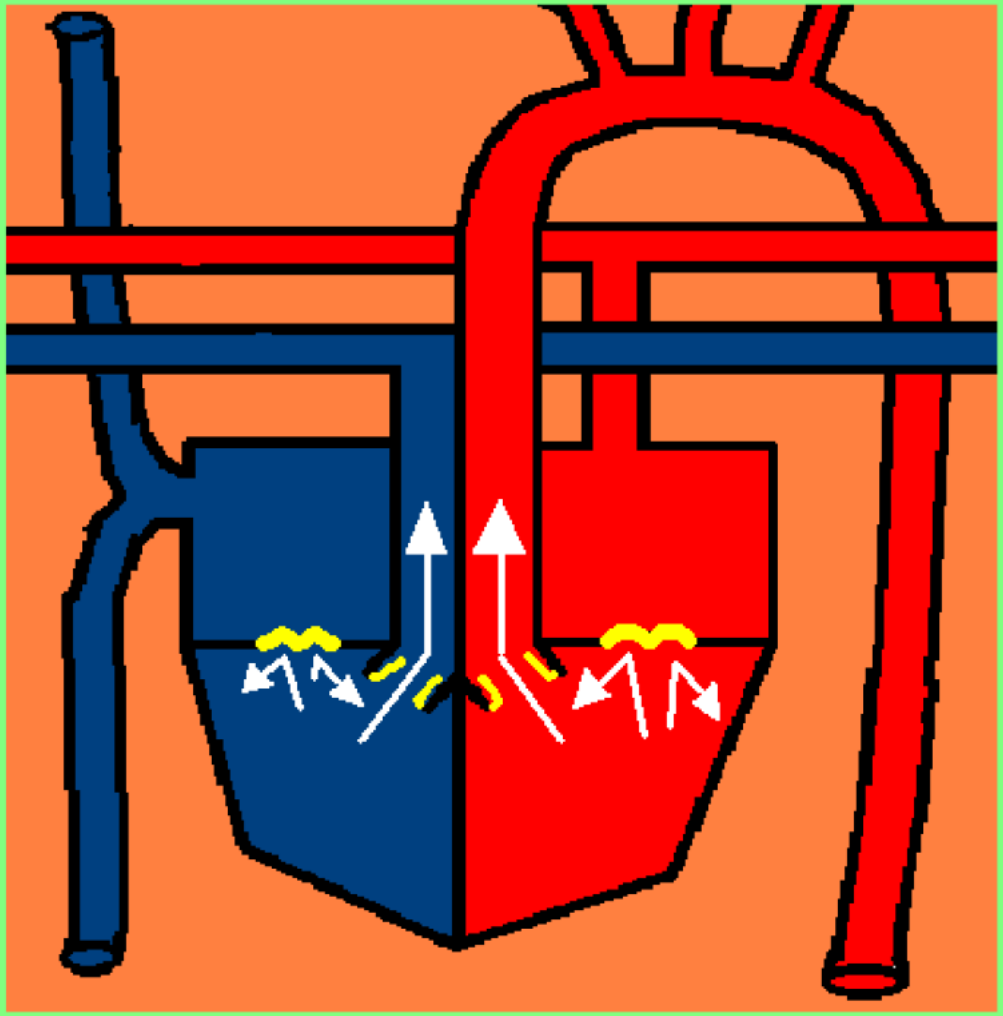
THERE ARE TWO NORMAL HEART SOUNDS, KNOWN AS: S-1 and S-2

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

HEART SOUNDS ASSESSMENT

S-1 BEGINNING OF SYSTOLE.

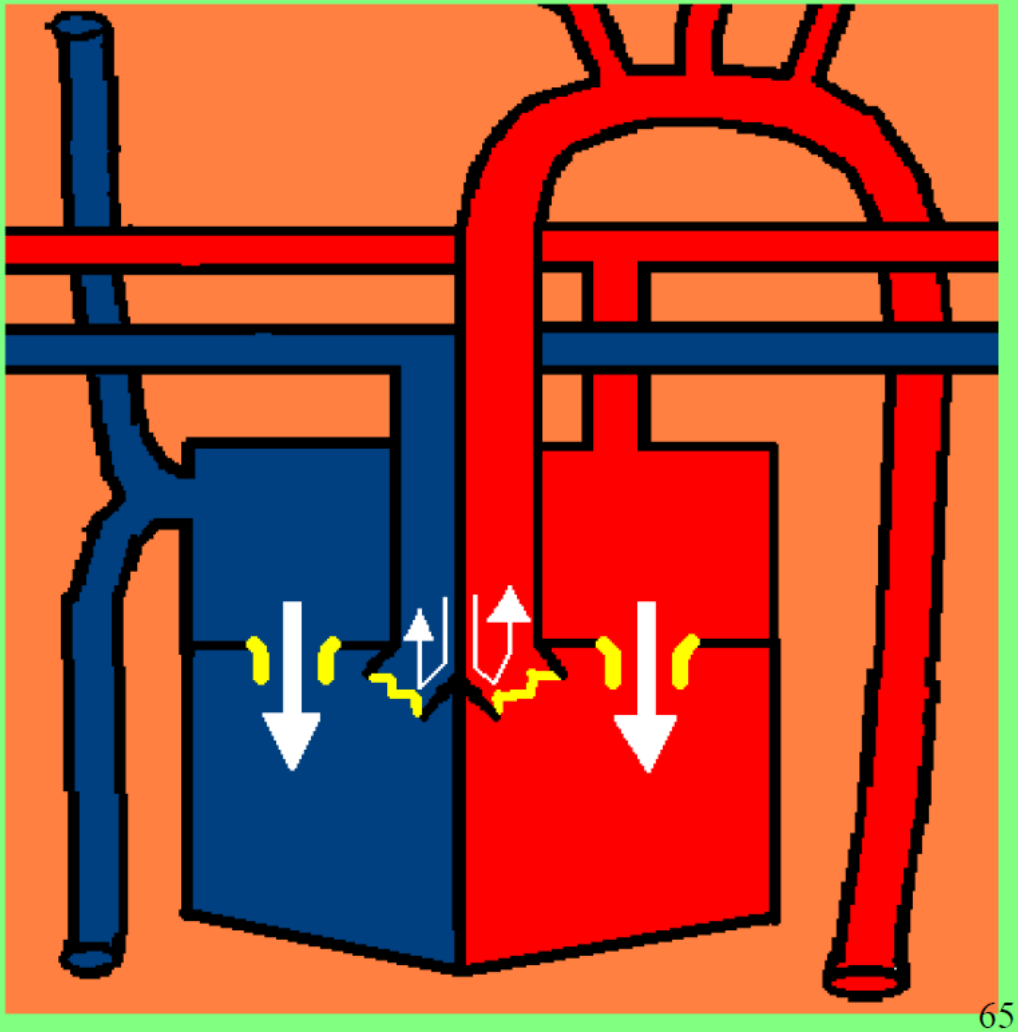
SOUND OF THE MITRAL AND TRICUSPID VALVES CLOSING.



HEART SOUNDS ASSESSMENT

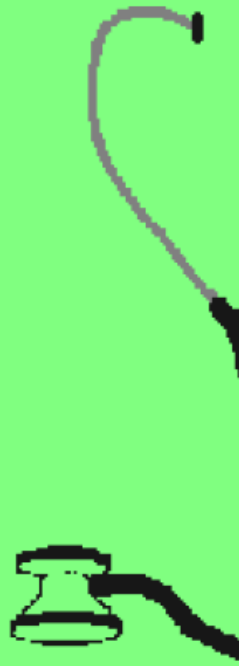
S-2 OCCURS AT THE END OF SYSTOLE (THE BEGINNING OF DIASTOLE).

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



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

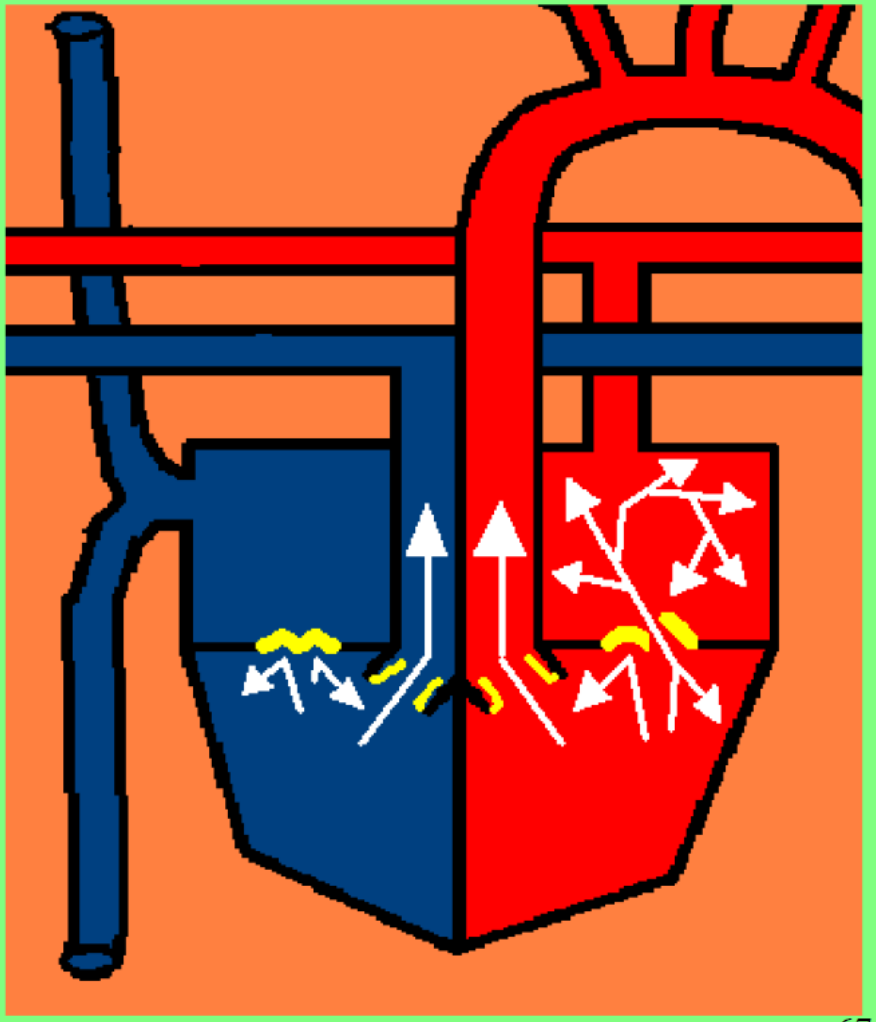
S-1 MURMUR SOUNDS LIKE:



"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).

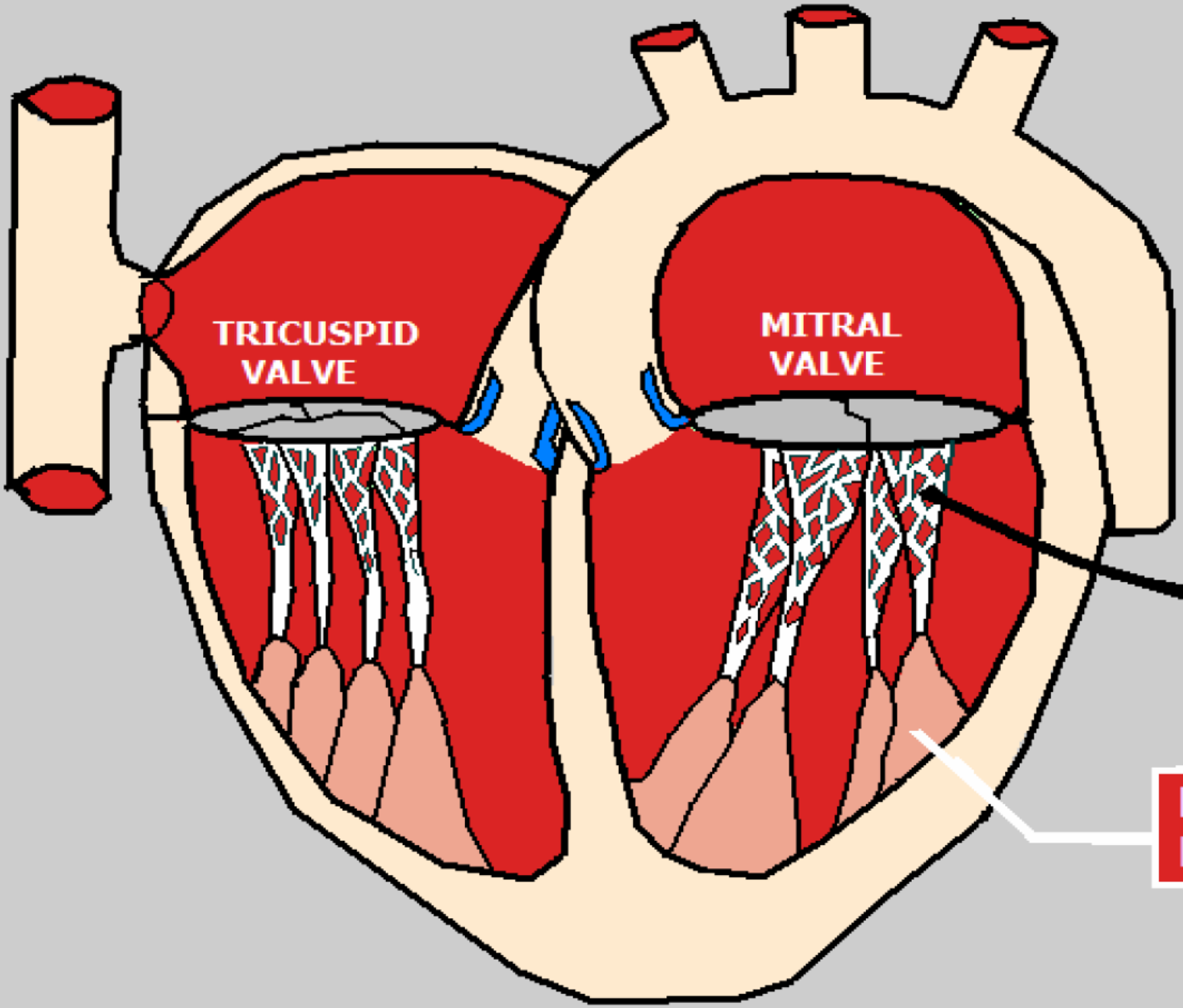
ACUTE Mitral Valve REGURGITATION can be caused by EXTENSIVE "Multi-Site" Myocardial Infarction and Necrosis – which results in PAPILLARY MUSCLE NECROSIS and PAPILLARY MUSCLE TEAR.

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

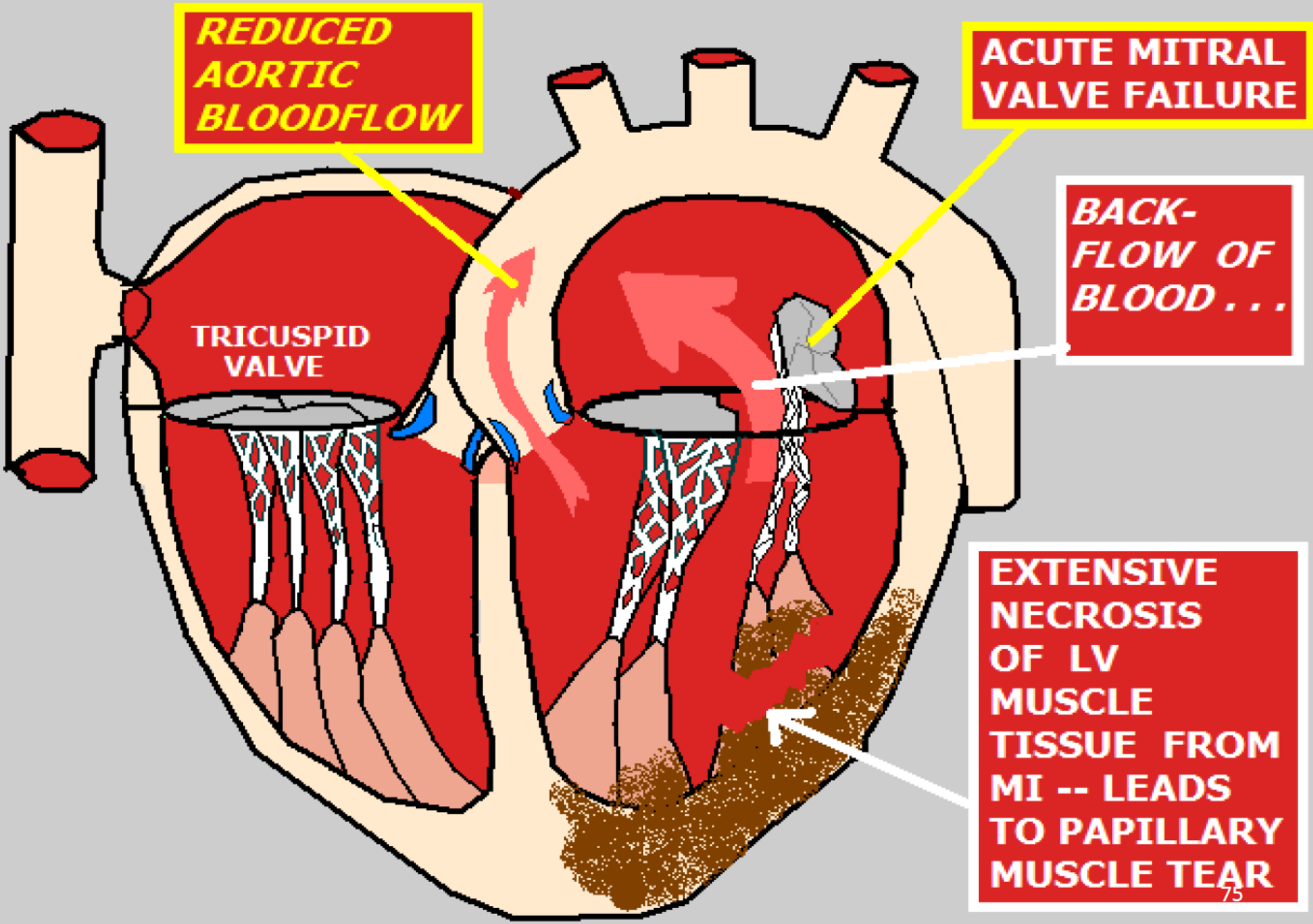
CHORDAE TENDINEAE

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

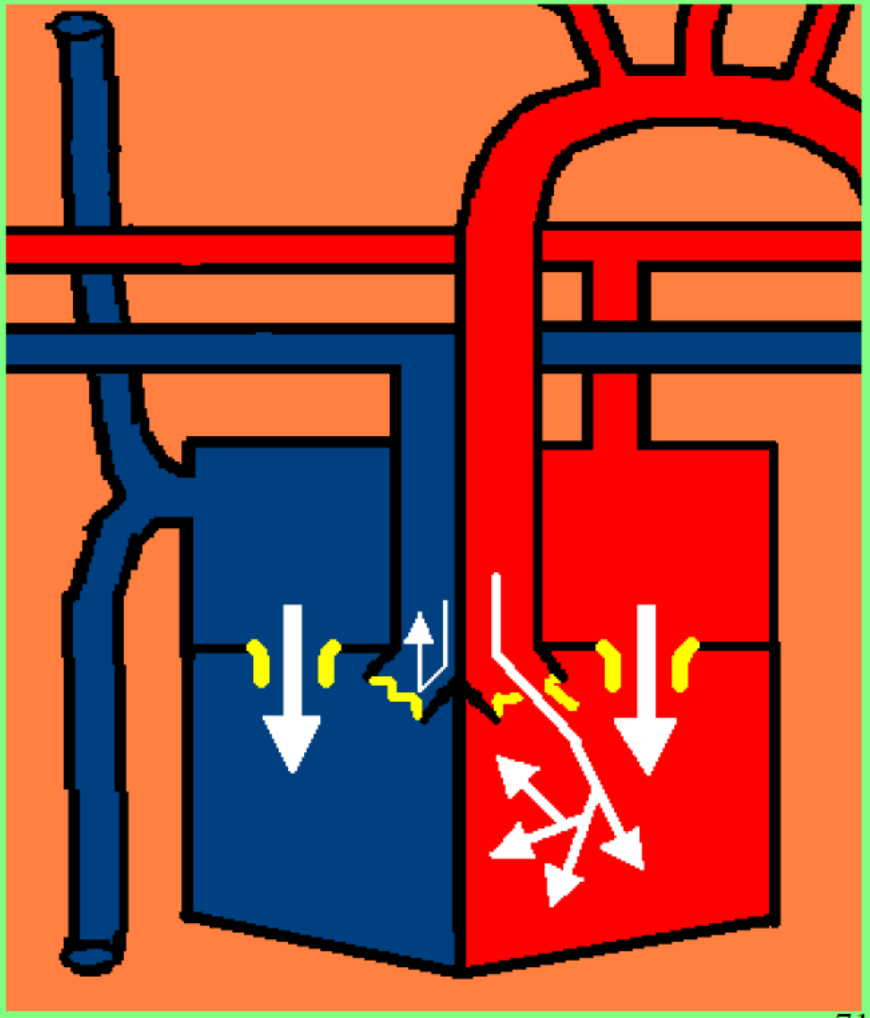
MURMUR = "SWOOSH"
SOUND CAUSED BY THE
SOUND OF TURBULENCE.

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.**

Access
University of Washington
Department of Medicine

Heart Sound Simulator

Integrated ECG:

PATIENT'S HEMODYNAMIC STATUS

+

SYMPTOMS

+

ECG

OLD BARN, SHREWSBURY, PA – 2001

Integrated ECG:

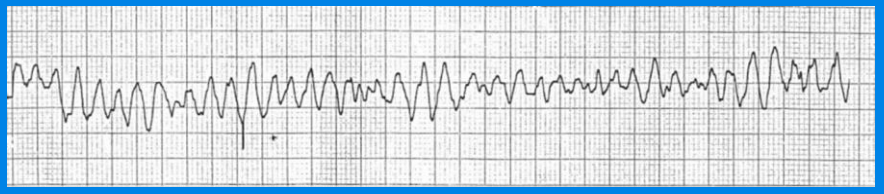
- HEMODYNAMIC STATUS
  - ABCs
  - Shock
- SYMPTOMS
  - Chest Pain / Pressure
  - Other ACS Symptoms
- ECG
  - 12 Lead
  - Single Lead “rhythm strip”

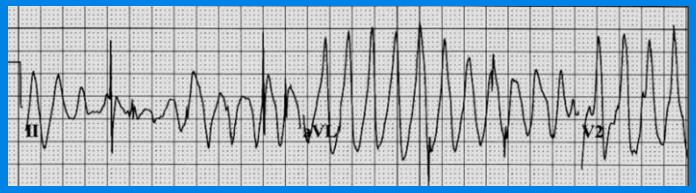
Integrated ECG:

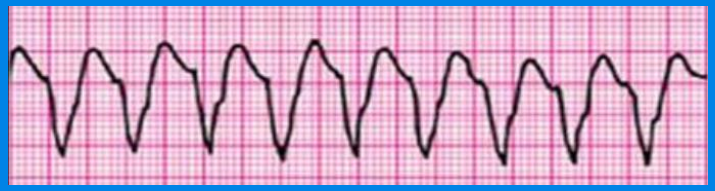
- HEMODYNAMIC STATUS
  - ABCs (Airway open? + Breathing? + Pulse?)

If there is **NO PULSE** . . . .

- **Start CPR**
- **Apply ECG** – determine rhythm- shockable?
  - **SHOCKABLE:** V-fib / V-tach / Torsades







Defib 120-200 BiPhasic
- IV Access
- Advanced Airway

Defib 120-200 or HIGHER
- Epinephrine 1mg IV

Defib 120-200 or HIGHER
- Amiodarone 300mg - OR – Lidocaine 1.0 -1.5 mg/kg

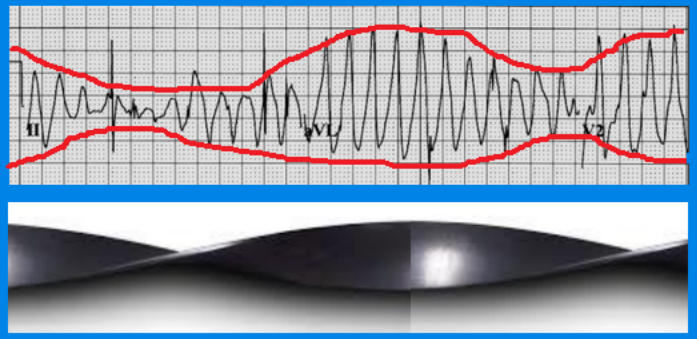
Defib 120-200 or HIGHER
- Epinephrine 1mg IV

Defib 120-200 or HIGHER
- CONTINUE as per ACLS….

If there is **NO PULSE** . . . .

- **Start CPR**
- **Apply ECG** – determine rhythm- shockable?
  - **SHOCKABLE:** V-fib / V-tach / Torsades

Torsades de Pointes . . . the QRS pattern resembles a "TWISTED RIBBON" . . . .



CONSIDER using Lidocaine in place of Amiodarone due to the increased possibility of QT PROLONGATION . . .

Defib 120-200 BiPhasic
- IV Access
- Advanced Airway

Defib 120-200 or HIGHER
- Epinephrine 1mg IV

Defib 120-200 or HIGHER
- ~~Amiodarone 300mg~~ – OR –
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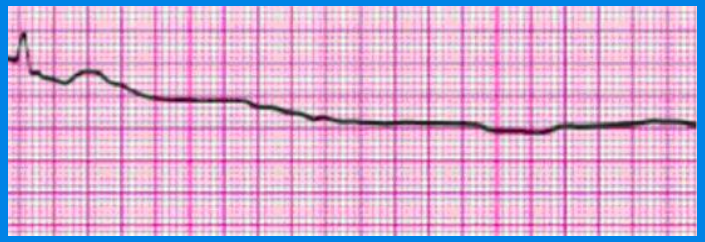
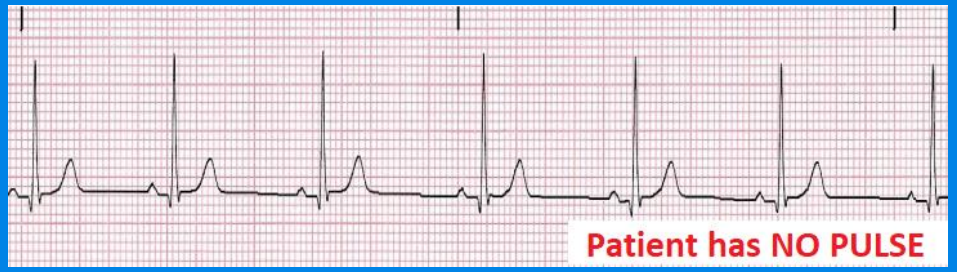
Defib 120-200 or HIGHER
- Epinephrine 1mg IV

Defib 120-200 or HIGHER
- CONTINUE as per ACLS….

If there is **NO PULSE** . . . .

- **Start CPR**
- **Apply ECG** – determine rhythm- shockable?
  - **NOT SHOCKABLE:** Agonal Rhythm / Asystole / PEA

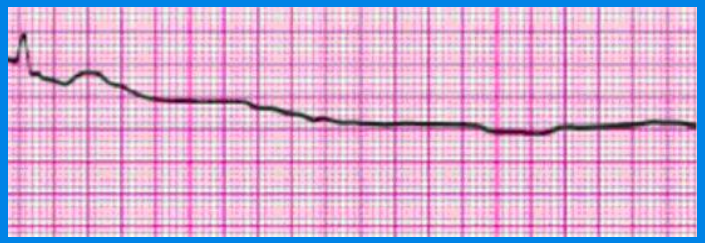




- Continue CPR
- IV/IO Access
- Advanced Airway
- Epinephrine 1mg IV
- Rule out reversible causes
- CONTINUE as per ACLS….

If there is **NO PULSE** . . . .

- **Start CPR**
- **Apply ECG** – determine rhythm- shockable?
  - **NOT SHOCKABLE:** Agonal Rhythm / Asystole / PEA



- Continue CPR
- IV/IO Access
- Advanced Airway
- Epinephrine 1mg IV
- Rule out reversible causes
- CONTINUE as per ACLS….

if the patient
HAS A PULSE with
AGONAL COMPLEXES . . . .
IMMEDIATELY BEGIN TRANSCUTANEOUS PACING -
you will probably save the patient's life !

Integrated ECG:

- HEMODYNAMIC STATUS
  - ABCs
  - Shock Assessment

SHOCK ASSESSMENT

SHOCK =

INADEQUTE TISSUE PERFUSION

- STARTS THE INSTANT YOU SEE PATIENT
- ENDS WHEN YOU REACH THE PATIENT'S SIDE

SHOCK ASSESSMENT

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

SHOCK is the CORRIDOR to DEATH

SHOCK – FIND CAUSE . . .

- HYPOVOLEMIC (internal or external bleeding)
- OBSTRUCTIVE (PE / tamponade)
- PSYCHOGENIC (sudden fear – self-correcting)
- NEUROLOGICAL (spinal injury)
- INSULIN (hypoglycemia)
- SEPTIC (systemic infection)
- CARDIOGENIC (abnormal heart rate or contractility)

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- SEPTIC (systemic infection)
- ☞ ***CARDIOGENIC (abnormal heart rate or contractility)***

*Initial Patient Workup:

- Determine CHIEF COMPLAINT
- STAT 12 Lead ECG (if indicated)
- Continuous ECG Monitoring
- Vital signs
- Verbal history
- O2 (if indicated)
- IV (if indicated)

* *Appropriate order of events varies based on a case-by-case basis*

*Initial Patient Workup:

- **Determine CHIEF COMPLAINT**
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Integrated ECG:

- HEMODYNAMIC STATUS
  - ABCs
  - Shock
- SYMPTOMS
  - Chest Pain / Pressure

Integrated ECG:

- HEMODYNAMIC STATUS
  - ABCs
  - Shock
- SYMPTOMS
  - Chest Pain / Pressure = ***STAT 12 LEAD ECG !!!***

    (within 10 minutes) !!

CHIEF COMPLAINT

KEY WORDS:

"CHEST: PAIN / HEAVINESS / PRESSURE/ FUNNY FEELING IN," etc.

SHORTNESS BREATH

DIZZINESS / LIGHTHEADEDNESS

ETC. ETC. ETC.

INFARCTION

SYMTOMS OF MYOCARDIAL INFARCTION:

1. CHEST PAIN:
   - Substernal - can radiate to neck, shoulders, jaw, L or R arm
   - Pain described as "Dull Pain" or "Pressure" or "Heaviness" - but can be sharp
   - Usually NOT effected by DEEP INSPIRATION, POSITION, or MOVEMENT

INFARCTION

SYMTOMS OF MYOCARDIAL INFARCTION:

1. CHEST PAIN
2. SHORTNESS OF BREATH
   May or may not be present.

INFARCTION

SYMTOMS OF MYOCARDIAL INFARCTION:

1. CHEST PAIN
2. SHORTNESS OF BREATH
3. NAUSEA
   May or may not be present

INFARCTION

SYMTOMS OF MYOCARDIAL INFARCTION:

1. CHEST PAIN
2. SHORTNESS OF BREATH
3. NAUSEA
4. COLD, CLAMMY, PALE SKIN
   and other signs of hypoperfusion
   may be present

INFARCTION

- - - "Classic Symptoms" - - -

QUICK ASSESSMENT "SHORT FORM"

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

Integrated ECG:

- HEMODYNAMIC STATUS
  - ABCs
  - Shock
- SYMPTOMS
  - Chest Pain / Pressure
  - Other ACS Symptoms

Integrated ECG:

- HEMODYNAMIC STATUS
  - ABCs
  - Shock
- SYMPTOMS
  - Chest Pain / Pressure
  - Other ACS Symptoms = ***STAT 12 LEAD ECG !!!***

    (within 10 minutes) !!

ATYPICAL SYMPTOMS of ACS

? ? ?

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

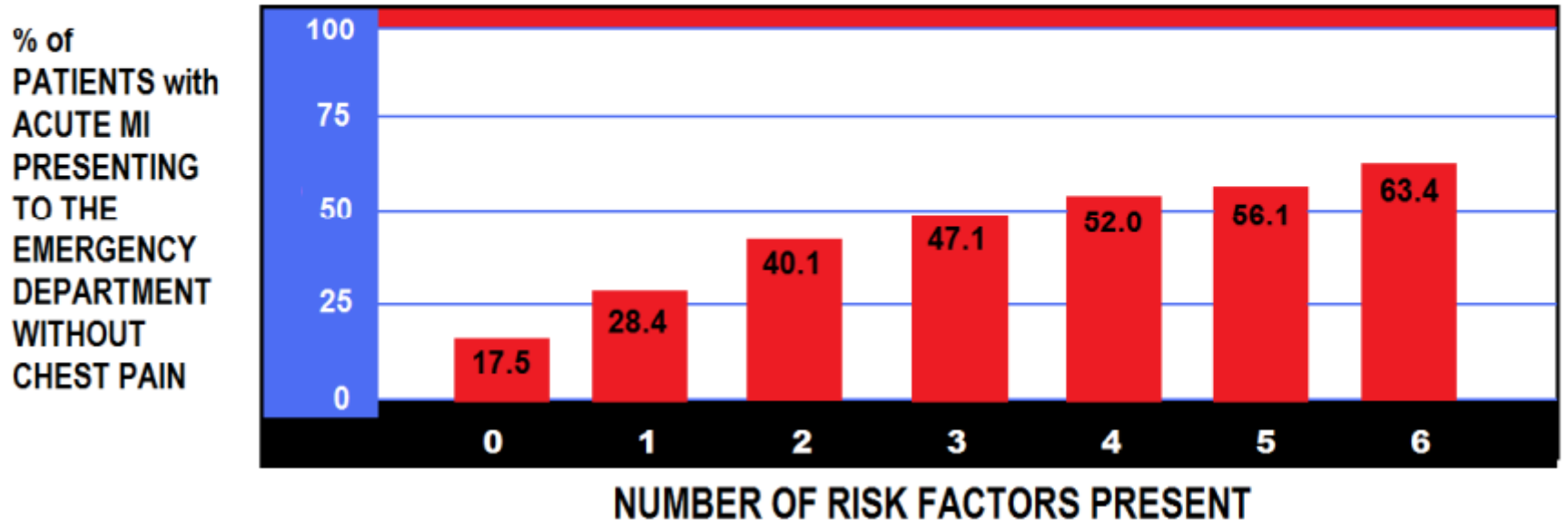
Stroke (previous history of)
Heart failure (previous history of)
Race (non-white)
Elderly (age 75+)
Women
Diabetes mellitus

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

Common atypical complaints associated with AMI without chest pain include:

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

Effect of Having Multiple Risk Factors for AMI Without Chest Pain



RISK FACTORS INCLUDE: **S**troke (previous), **H**eart failure (previous), **R**ace (non-white), **E**lderly (age 75+), **W**omen, **D**iabtetes

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

Integrated ECG:

- HEMODYNAMIC STATUS
  - ABCs
  - Shock
- SYMPTOMS
  - Chest Pain / Pressure
  - Other ACS Symptoms
- ECG
  - 12 Lead
  - Single Lead “rhythm strip”

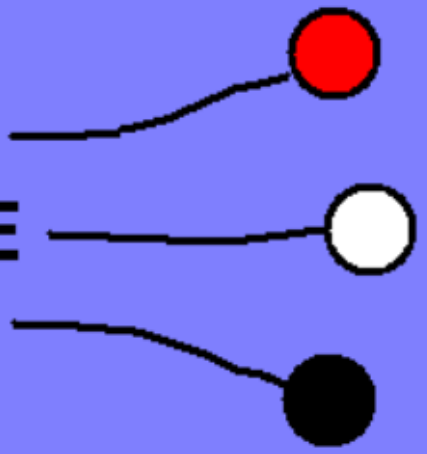
- The 12 Lead ECG has been ordered
- Meanwhile we'll hook the patient to the ECG monitor . . . . .

THE EKG MACHINE

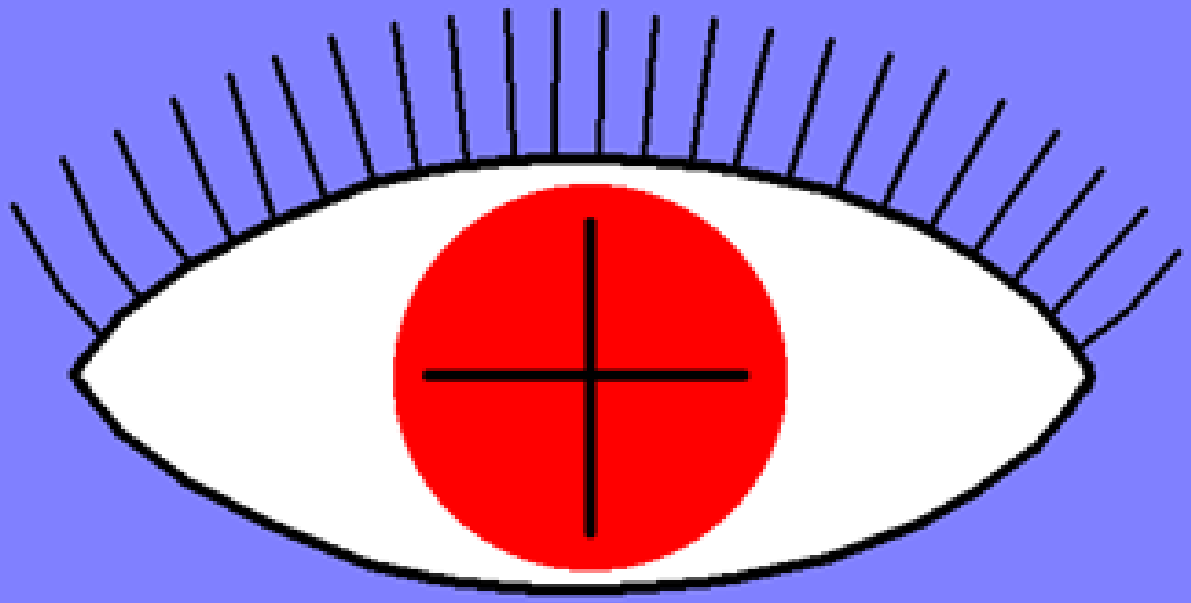
STANDARD 12 LEADS - USES 10 WIRES
(6 CHEST and 4 LIMB)

- I, II, III, and V1, V2, V3, V4, V5, V6
EACH CONSIST OF:

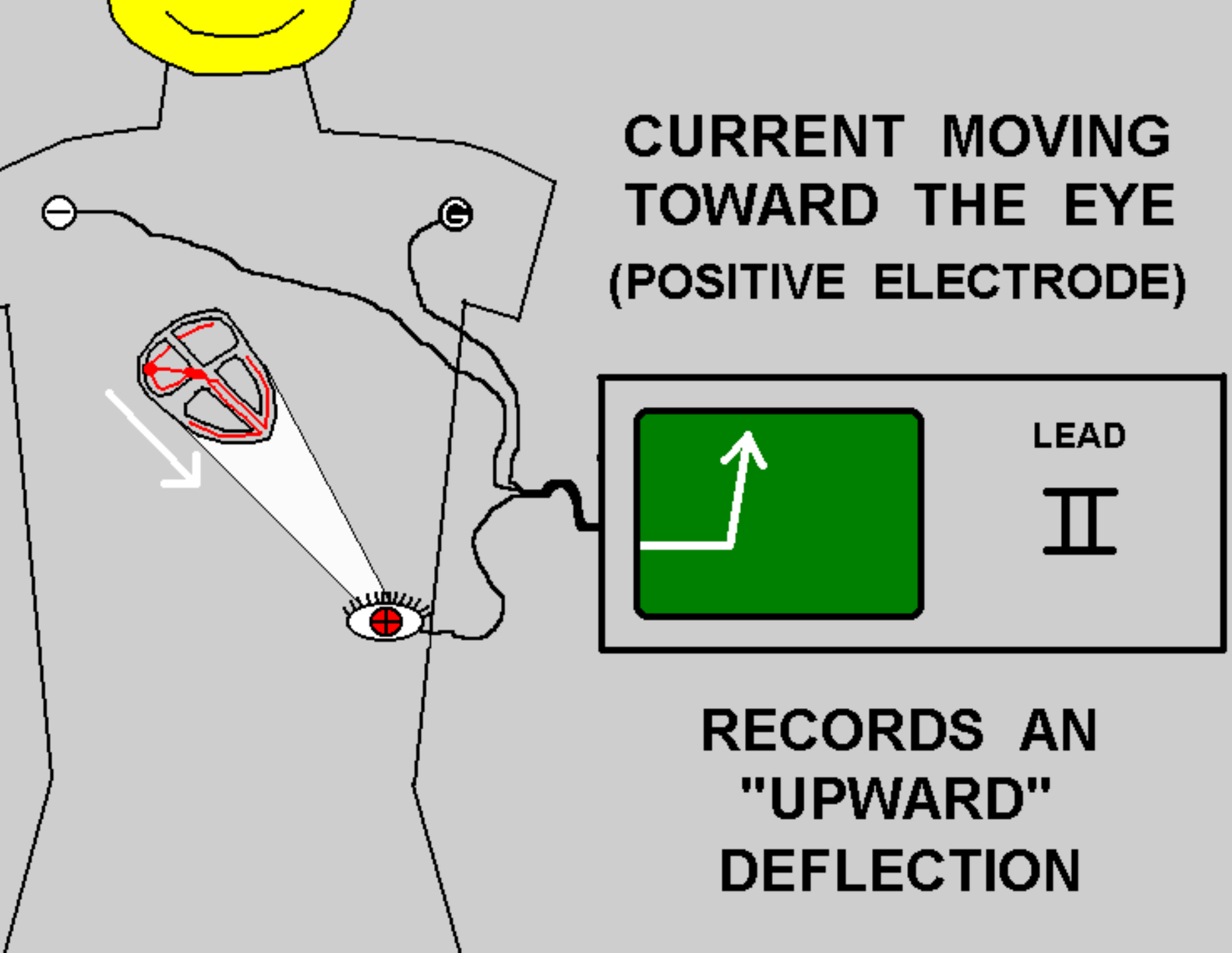
1 POSITIVE ELECTRODE
1 NEGATIVE ELECTRODE
1 GROUND ELECTRODE

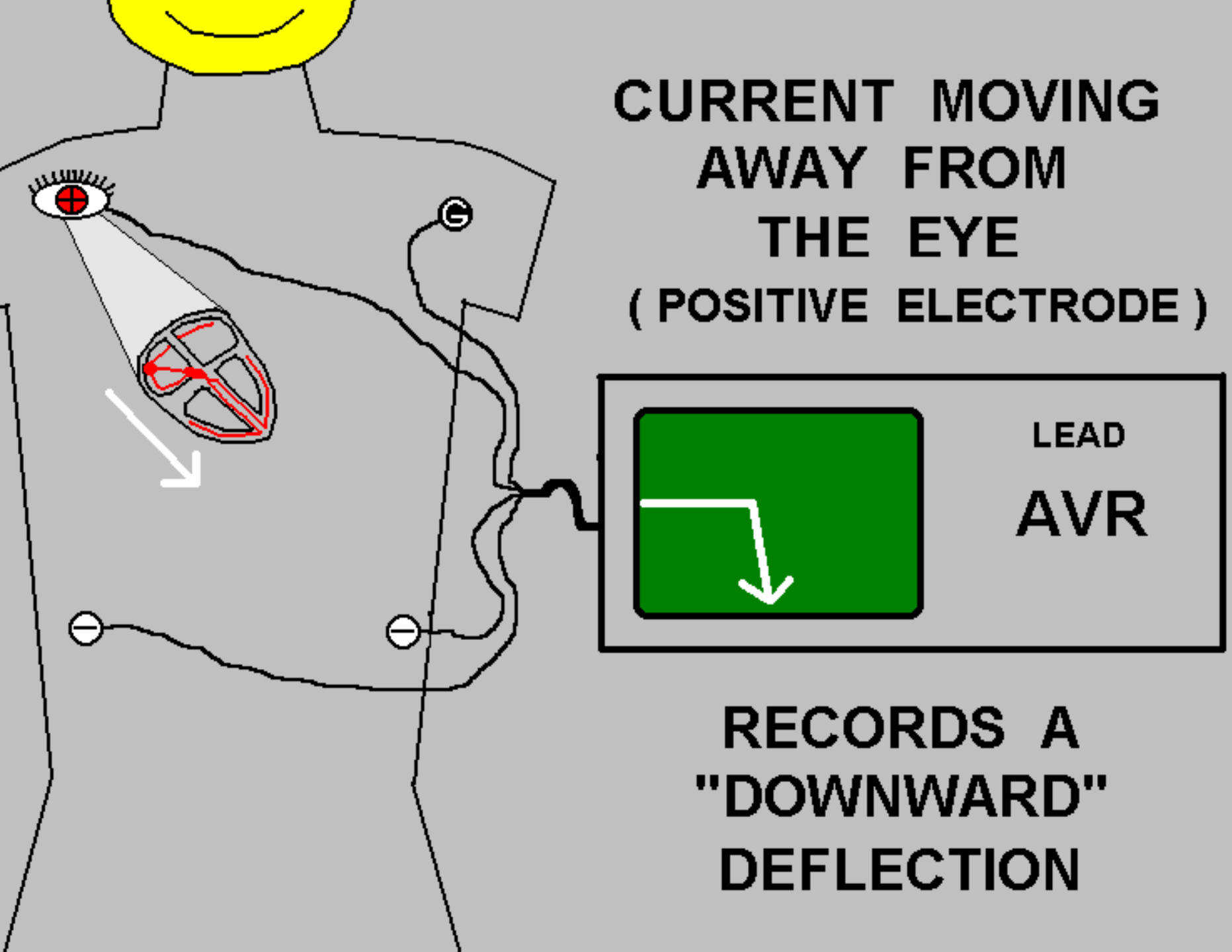


THE POSITIVE ELECTRODE

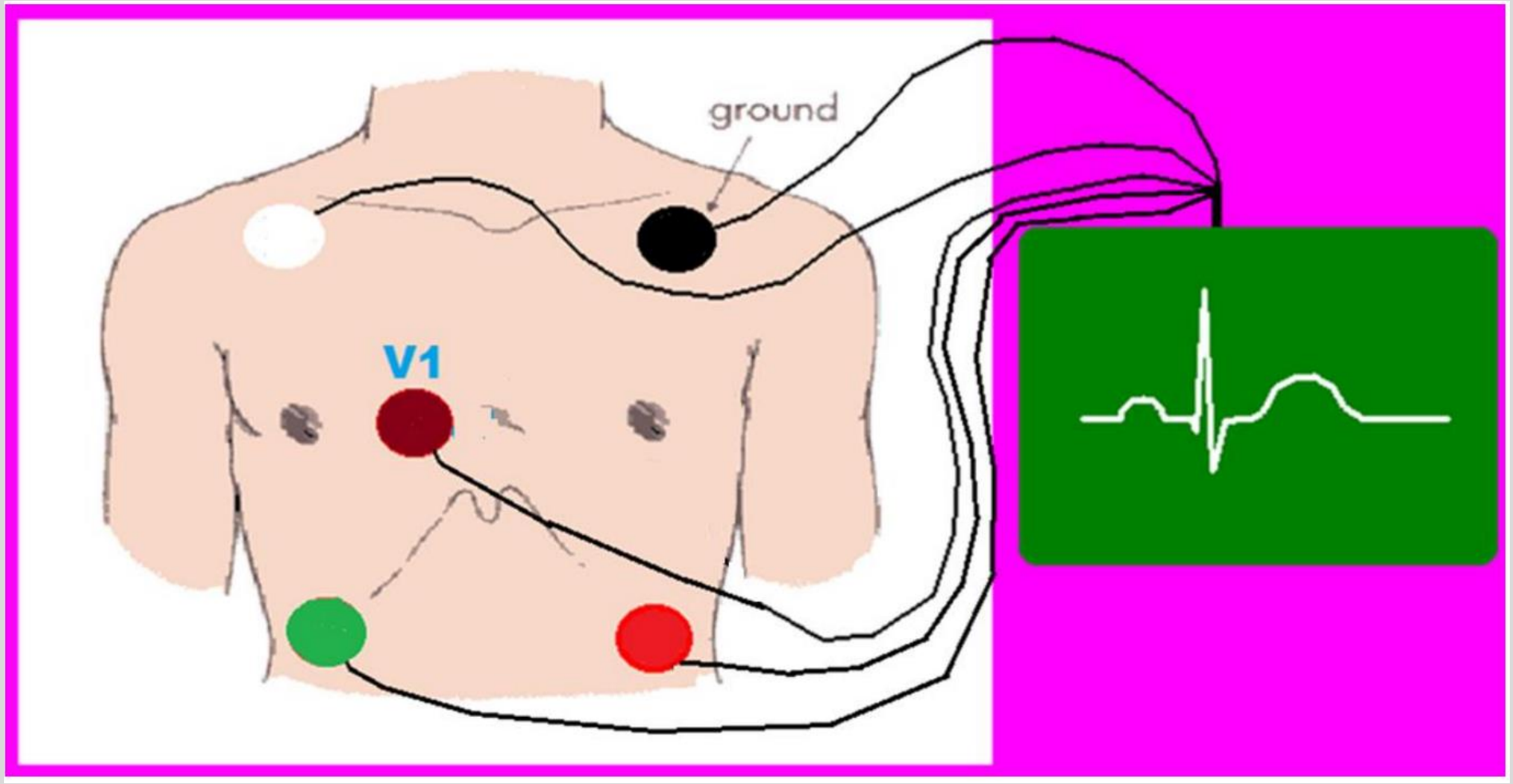


IS THE "EYE" . . .

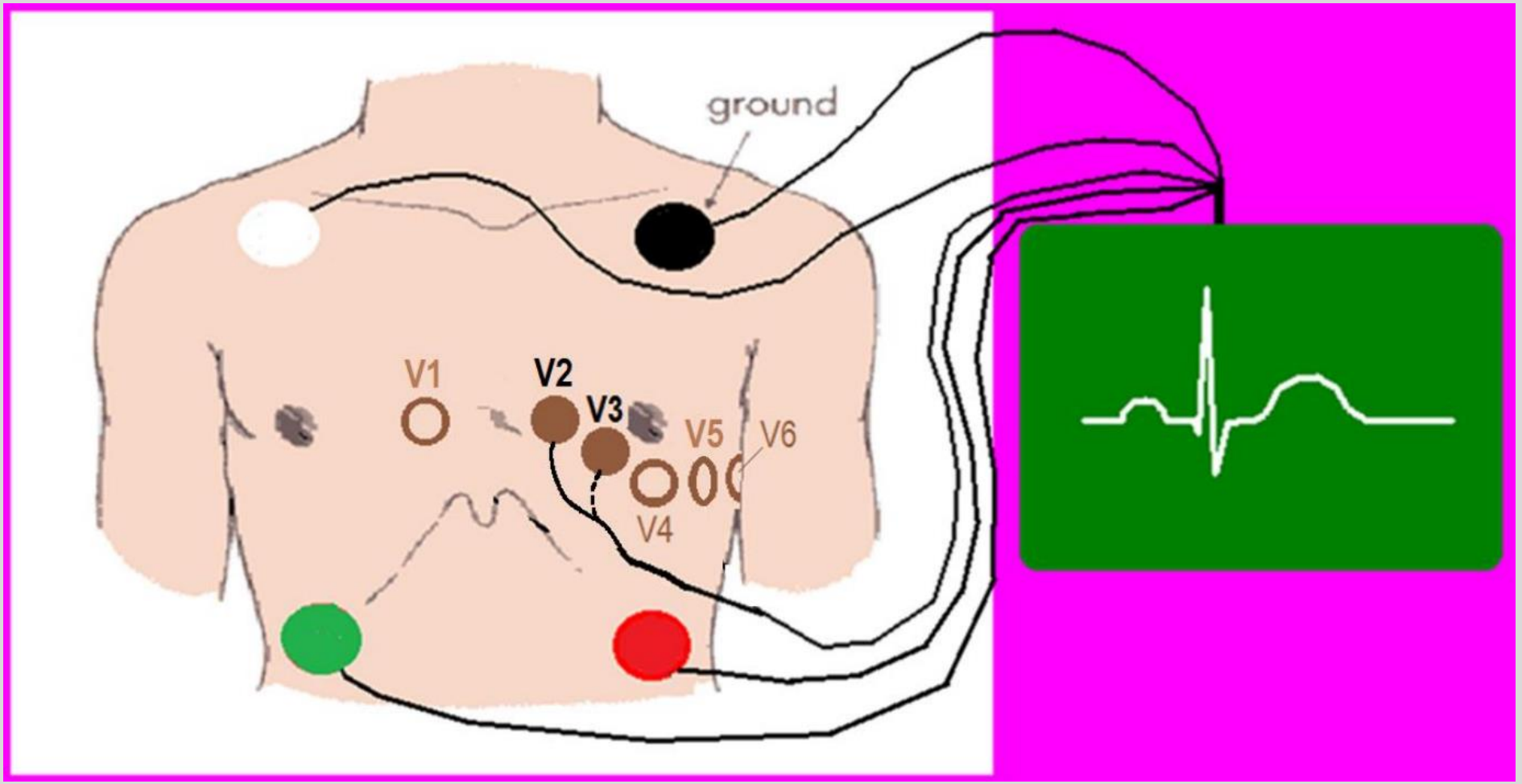

CURRENT MOVING
TOWARD THE EYE
(POSITIVE ELECTRODE)
LEAD
II
RECORDS AN
"UPWARD"
DEFLECTION


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

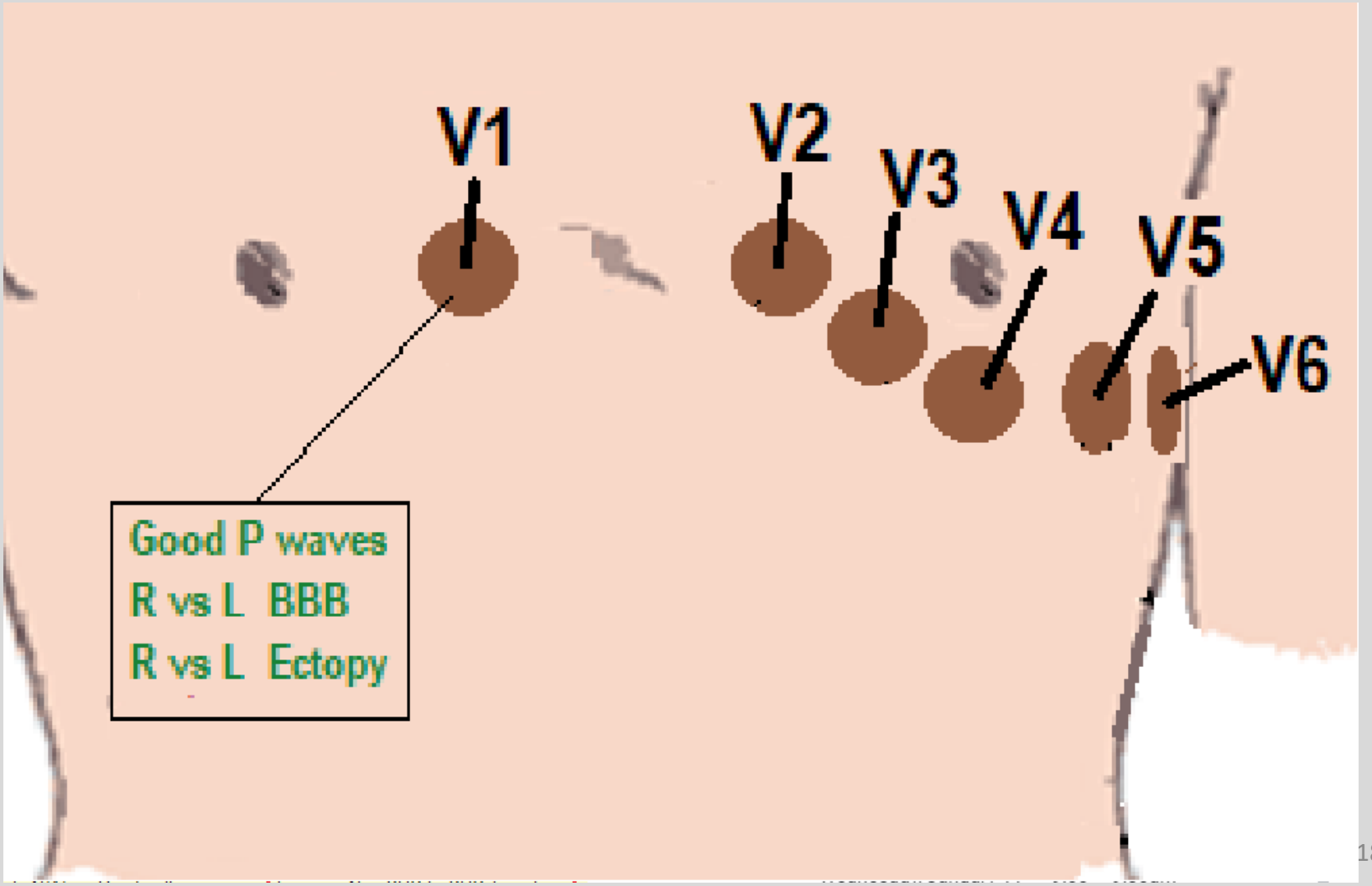
Traditional ECG Monitoring Lead Placement:



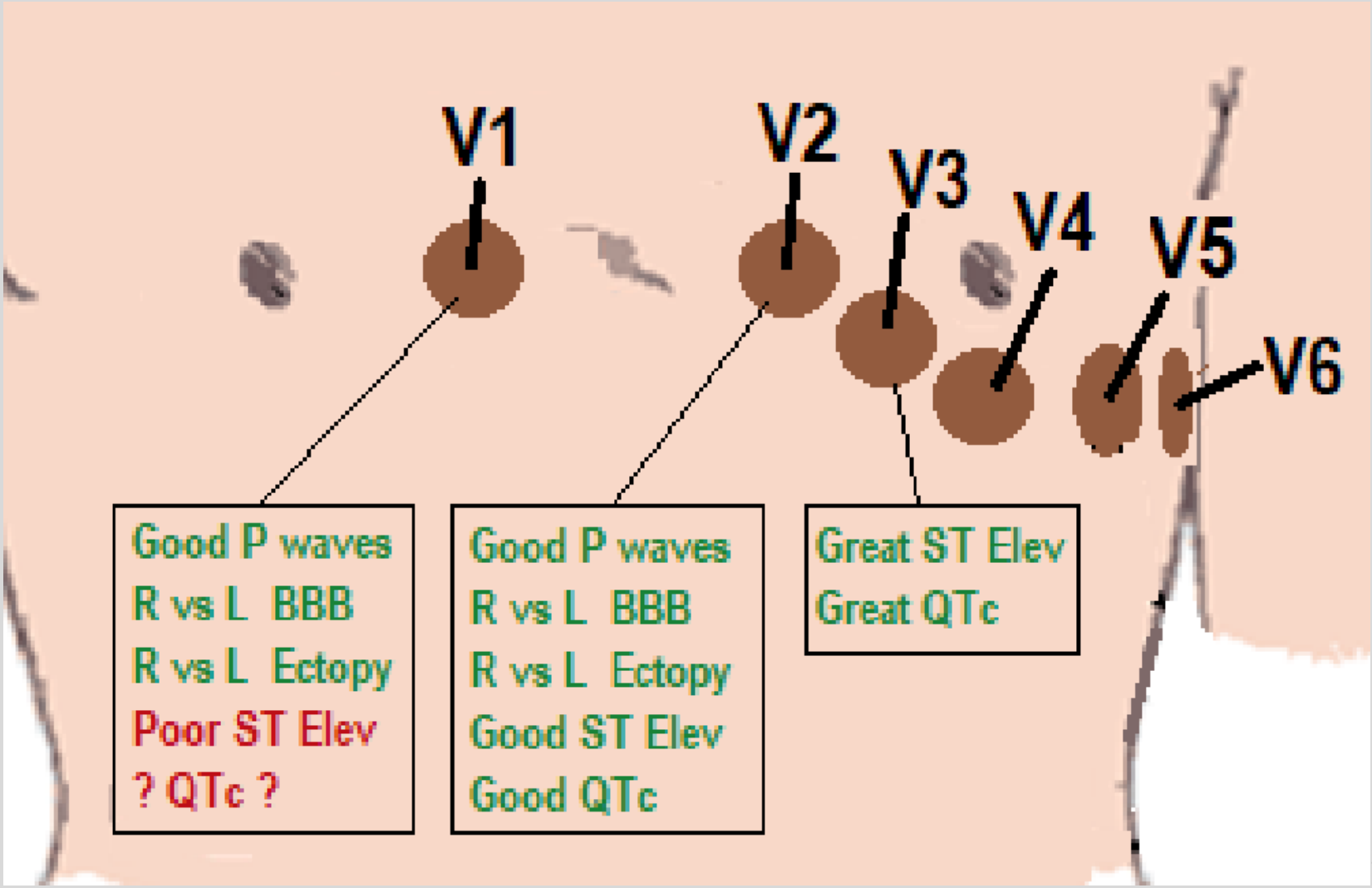
New (2014) Guideline Suggested ECG Monitoring Lead Placement



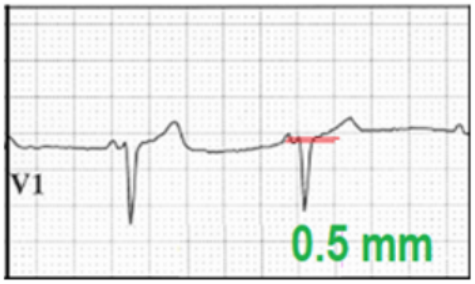
Traditional Continuous ECG Monitoring Lead: V1



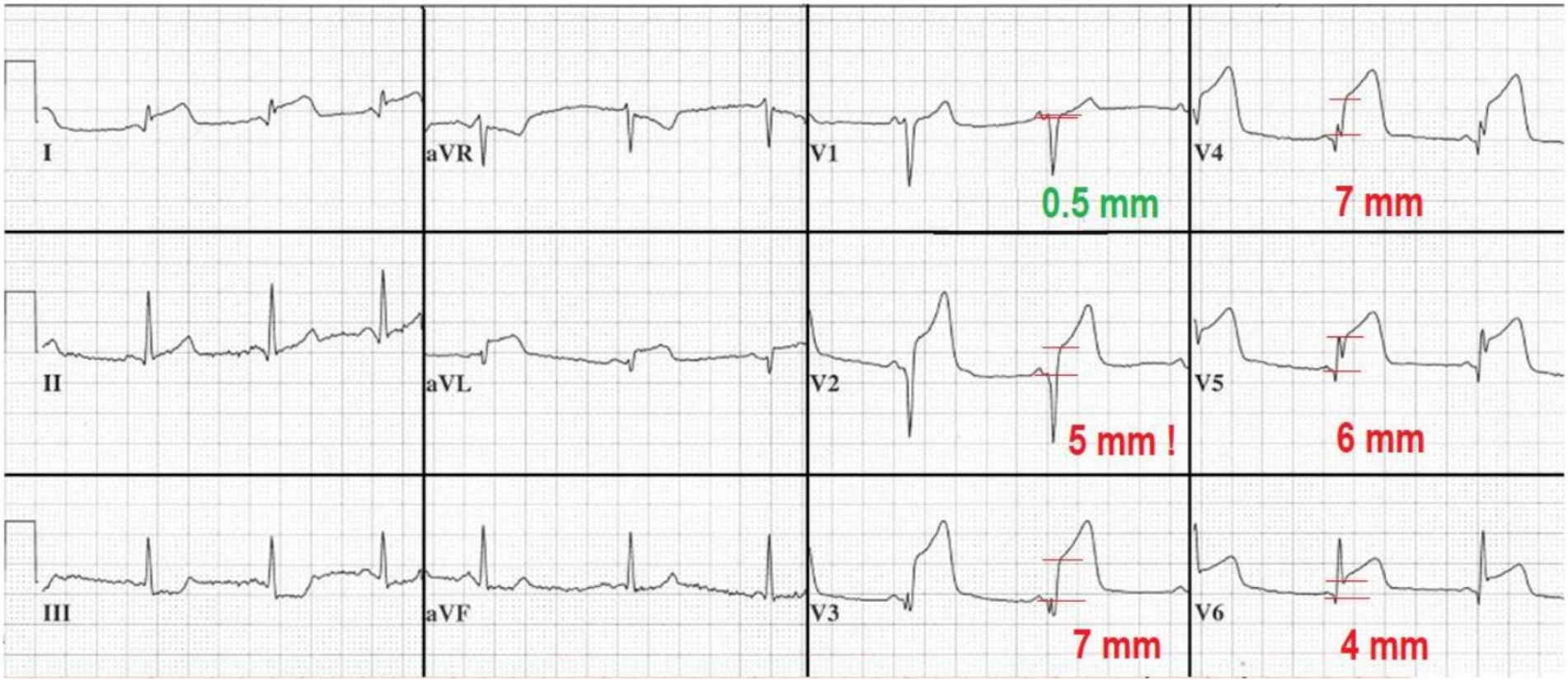
New (2014) Guideline Suggested ECG Monitoring Leads: V2 or V3



Why not V1 ?

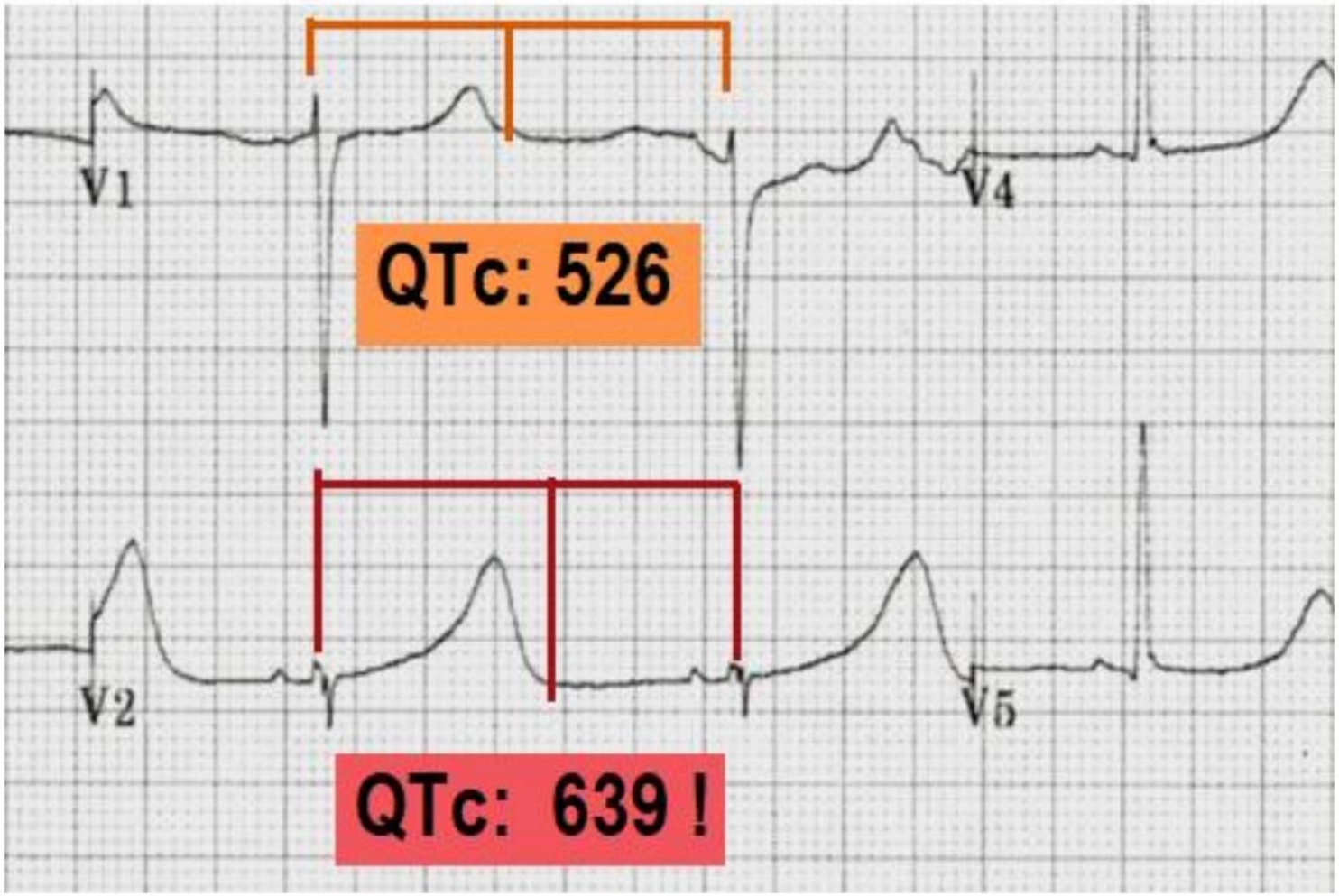


Why not V1 ? *(often won't see STEMI !)*



If you were only monitoring Leads II and V1, you ***would NOT detect this patient's STEMI ! !***

Why not V1 ? (may not detect critical QTc)



- We’ve established continuous ECG monitoring and we assess the rhythm . . . .

CARDIOGENIC SHOCK

- **Heart Rate:**
  - **Should be between 50 – 150**

CARDIOGENIC SHOCK

- **Heart Rate:**
  - **Should be between 50 – 150**

DECREASED CARDIAC OUTPUT may be present when heart rate is:

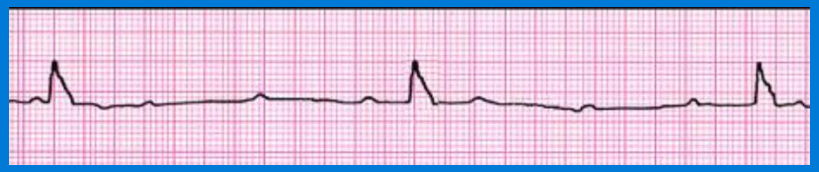
- **LESS THAN 50**
- **GREATER THAN 150**

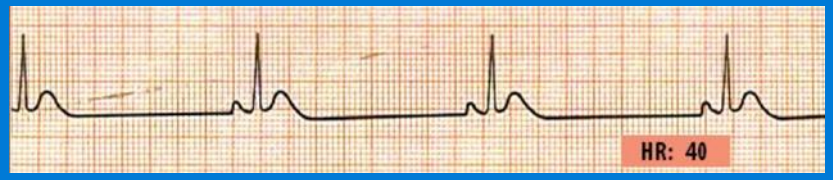
CARDIOGENIC SHOCK

- **Heart Rate**
  - **TOO SLOW (less than 50) with signs of shock:**

☞ ***SPEED UP THE HEART RATE***

(follow ACLS and Protocols)





Usual treatment:
- Atropine
- Pacemaker

Bradycardias & Heart Block

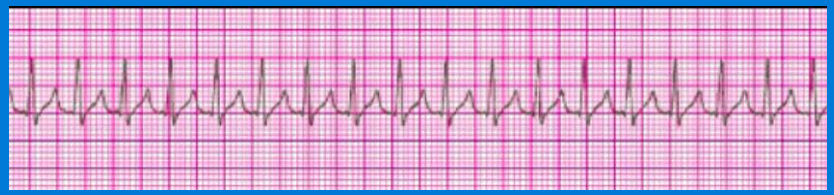
- There are several ECG Rhythms seen with “BRADYCARDIA”
- While you should be able to distinguish each rhythm, what is MORE IMPORTANT is that you simply “identify when the heart rate being TOO SLOW is causing the patient to be symptomatic (SHOCK) . . . .and that you. . .
- KNOW how to treat it.

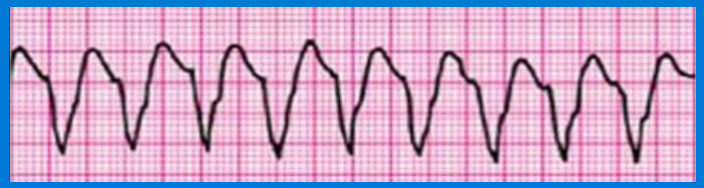
CARDIOGENIC SHOCK

- **Heart Rate**
  - **TOO FAST (greater than 150) with signs of shock:**

☞ ***SLOW the heart rate***

(follow ACLS and Protocols)





Usual treatment:
- Synchronized Cardioversion

Tachycardias

- Is the patient STABLE or UNSTABLE?
- QRS narrow or wide ???
  - Narrow = “not greater than 120 ms” (3 mm)
  - Wide = “greater than 120 ms (3 mm)

ALL UNSTABLE TACHYCARDIAS:

- SYNCHRONIZED CARDIOVERSION
  - As per agency PROTOCOL and/or ACLS
  - NARROW tachycardias = less initial energy
  - WIDE QRS tachycardias AND A-fib RVR = higher initial energy

ACLS TACHYCARDIA GUIDELINES

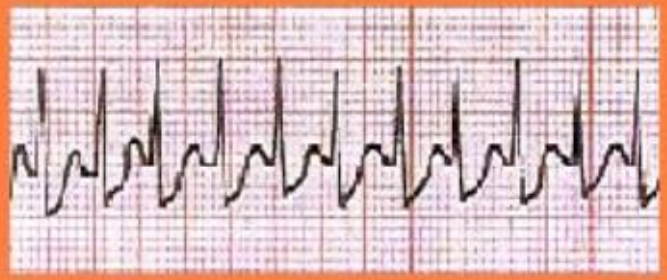
SVT

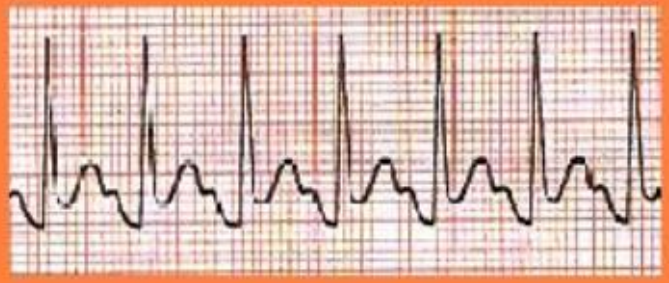
150

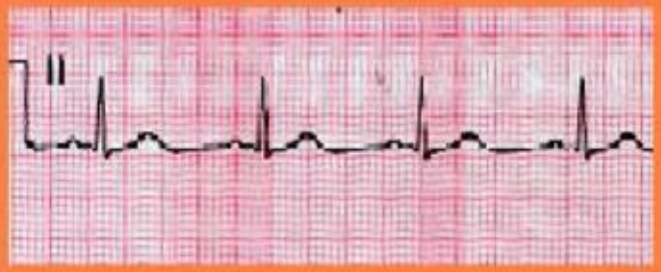
SINUS TACH

100

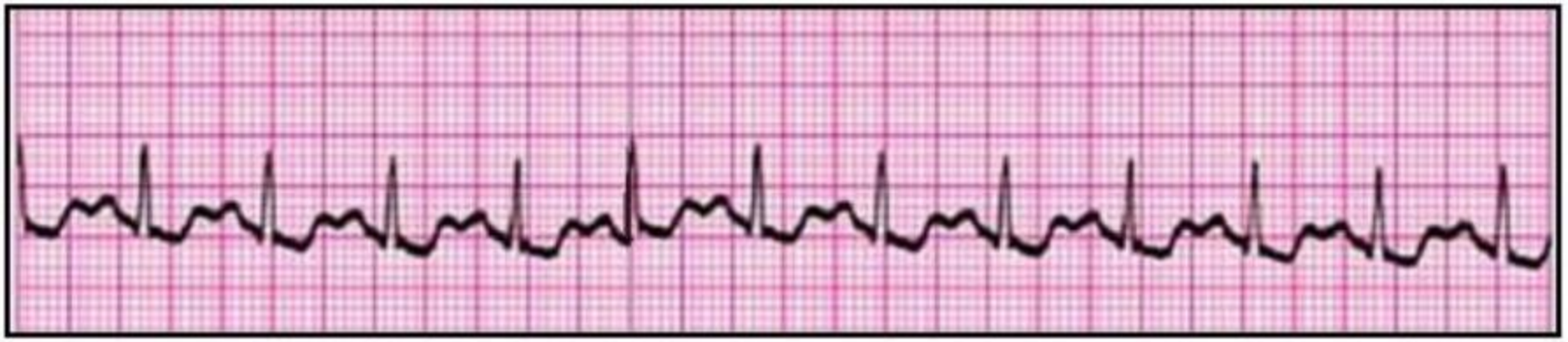
NSR





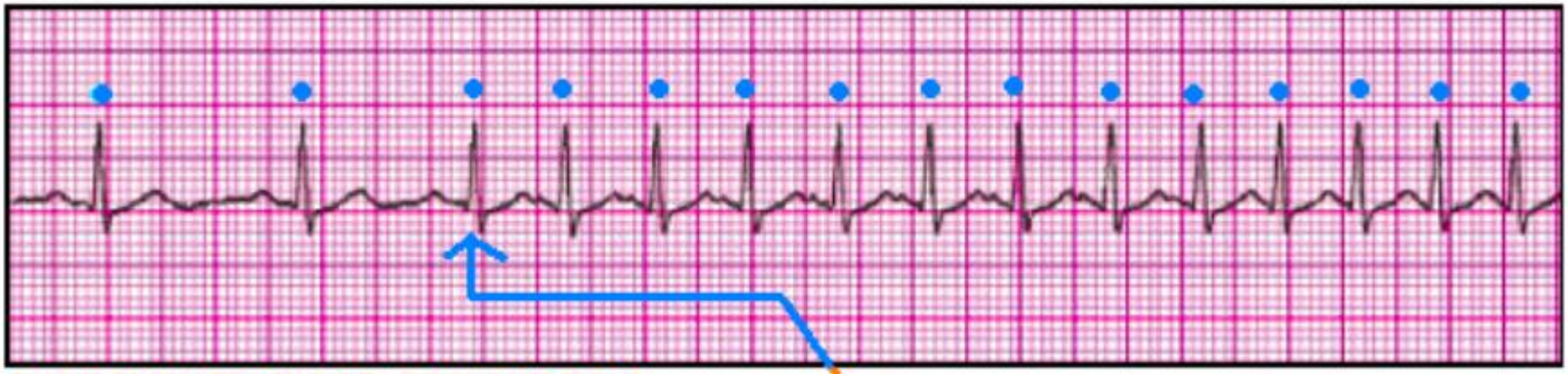


THIS RHYTHM IS: SINUS TACHYCARDIA



WE MUST CONSIDER UNDERLYING CAUSES:		AND TREAT THEM:
ANXIETY / FEAR	→	CALM PATIENT
HYPOVOLEMIA		
DEHYDRATION	→	FLUIDS
BLOOD LOSS	→	STOP BLEEDING
MEDICATION EFFECTS	→	CONSIDER MEDICAL Tx
OTHER ILLNESS	→	IDENTIFY & Tx DISORDER

RHTHYM CLUES . . . .

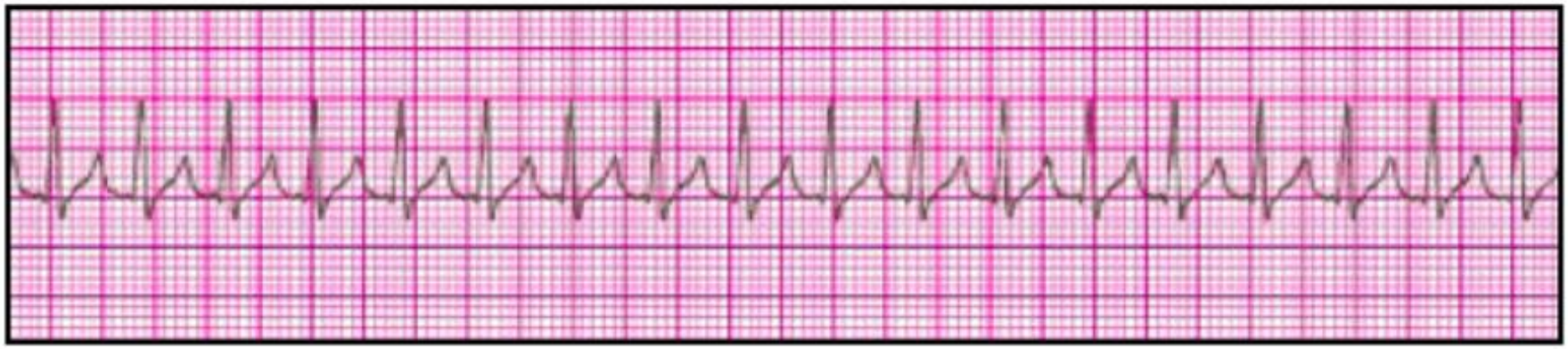


SUPRAVENTRICULAR TACHYCARDIA

SVT is usually PAROXSYMAL -- ie: has a SUDDEN ONSET.

SINUS TACHYCARDIA usually has a " ramp - up " and " ramp - down " period -- a gradual change in HEART RATE.

THIS RHYTHM IS: SUPRAVENTRICULAR TACHYCARDIA (SVT)

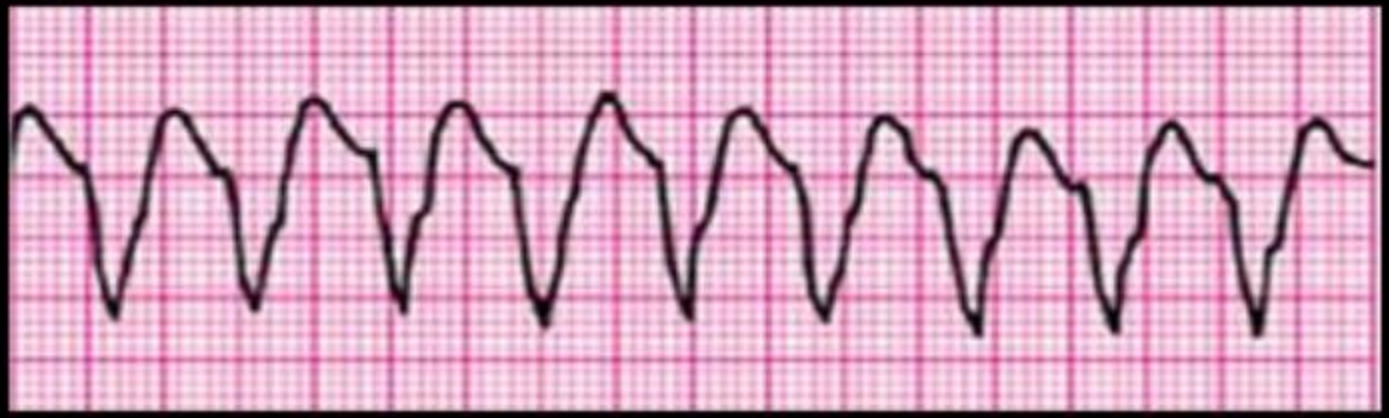


MAIN IDENTIFICATION CHARACTERISTIC(S): HEART RATE TOO FAST, USUALLY > 150. P WAVES MAY BE "BURIED" IN THE PRECEDING T WAVES. Pt USUALLY C/O "SUDDEN ONSET of HEART RACING," or "PALPITATIONS."

TREATMENT / INTERVENTIONS:

BASED ON WHETHER PATIENT IS STABLE or UNSTABLE . . .

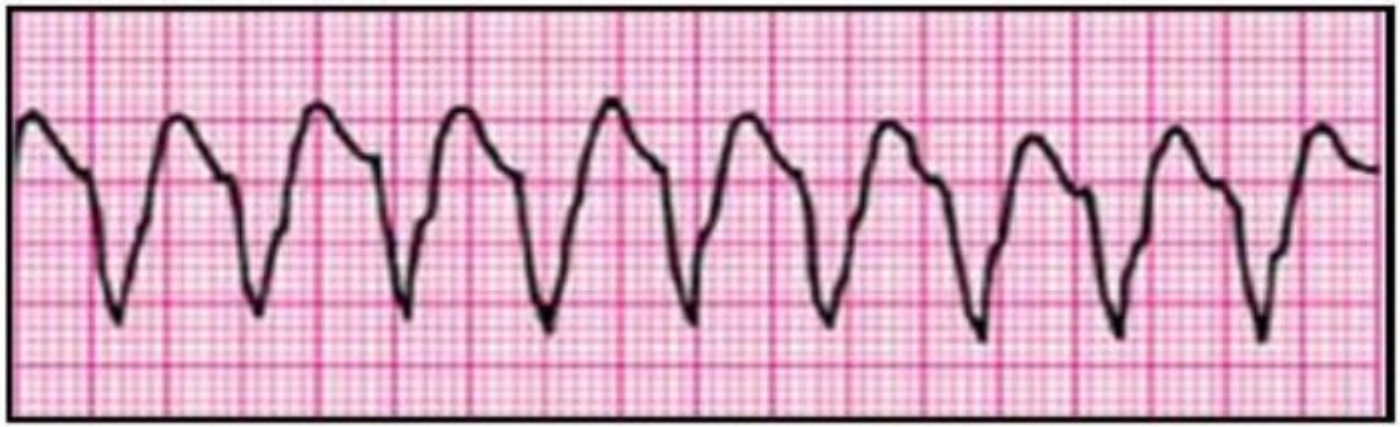
THIS RHYTHM IS:



MAIN IDENTIFICATION CHARACTERISTIC(S):

RATE ----------
RHYTHM ----------
P-R INTERVAL -----
P: QRS RATIO ------
QRS INTERVAL -----

THIS RHYTHM IS: MONOMORPHIC V-TACH



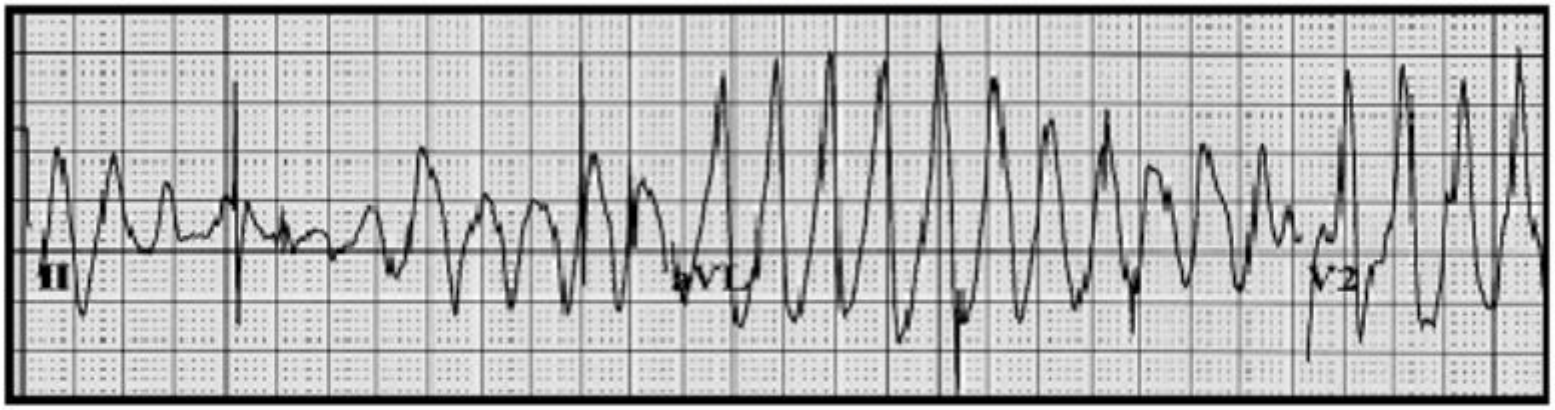
MAIN IDENTIFICATION CHARACTERISTIC(S): WIDE QRS COMPLEXES (> 120 ms)
HR USUALLY BETWEEN 150 - 200; ALL QRS COMPLEXES APPEAR SAME IN SHAPE and DEFELCTION; IF P WAVES SEEN, DISASSOTIATED w/ QRS

RATE ---------- > 100 (usually 150 - 200)
RHYTHM ---------- REGULAR
P-R INTERVAL ----- N/A
P: QRS RATIO ------ N/A
QRS INTERVAL ----- > 120 ms

V-Tach

- NO PULSE – Follow Protocols / ACLS for “V-Fib / V-Tach”
- PULSE – but UNSTABLE – Synchronized Cardioversion
- STABLE - Give MEDS as per Protocols / ACLS

THIS RHYTHM IS: POLYMORPHIC V - TACH



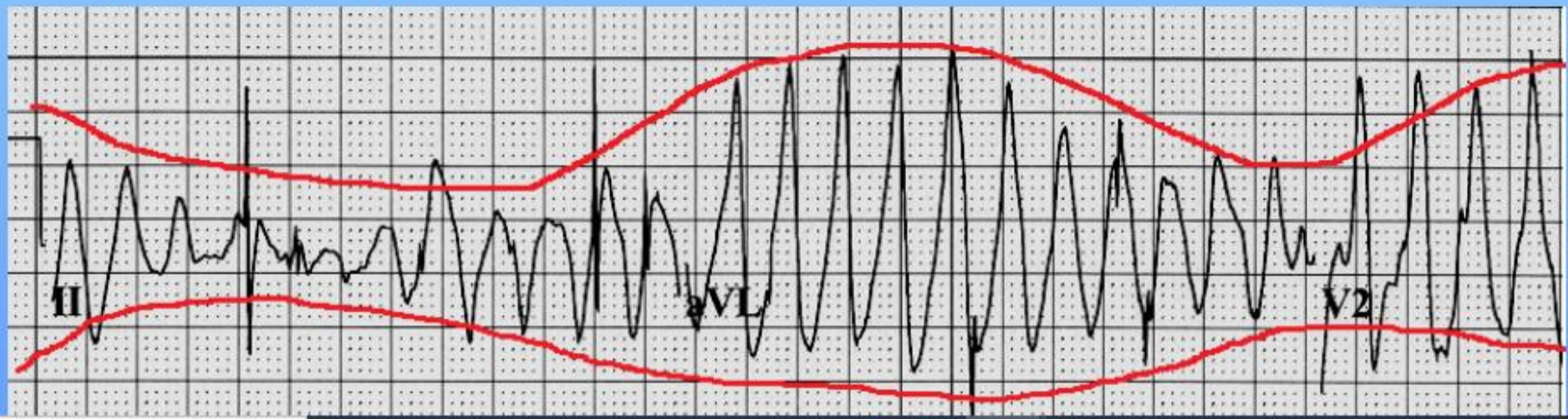
MAIN IDENTIFICATION CHARACTERISTIC(S): WIDE QRS COMPLEXES, MULTIPLE SHAPES AND FORMS, POSITVE AND NEGATIVE DEFLECTIONS, APPEARS TO ROTATE BETWEEN NEGATIVE AND POSITIVE (TWISTING OF POINTS)

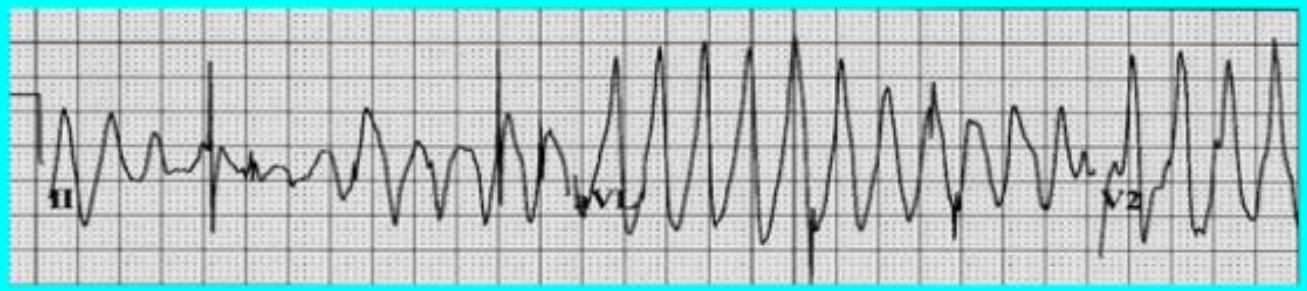
RATE	200 - 300
RHYTHM	VARIES
P-R INTERVAL	N/A
P: QRS RATIO	N/A
QRS INTERVAL	VARIES

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

resembles . . . . .

a piece of Twisted Ribbon !





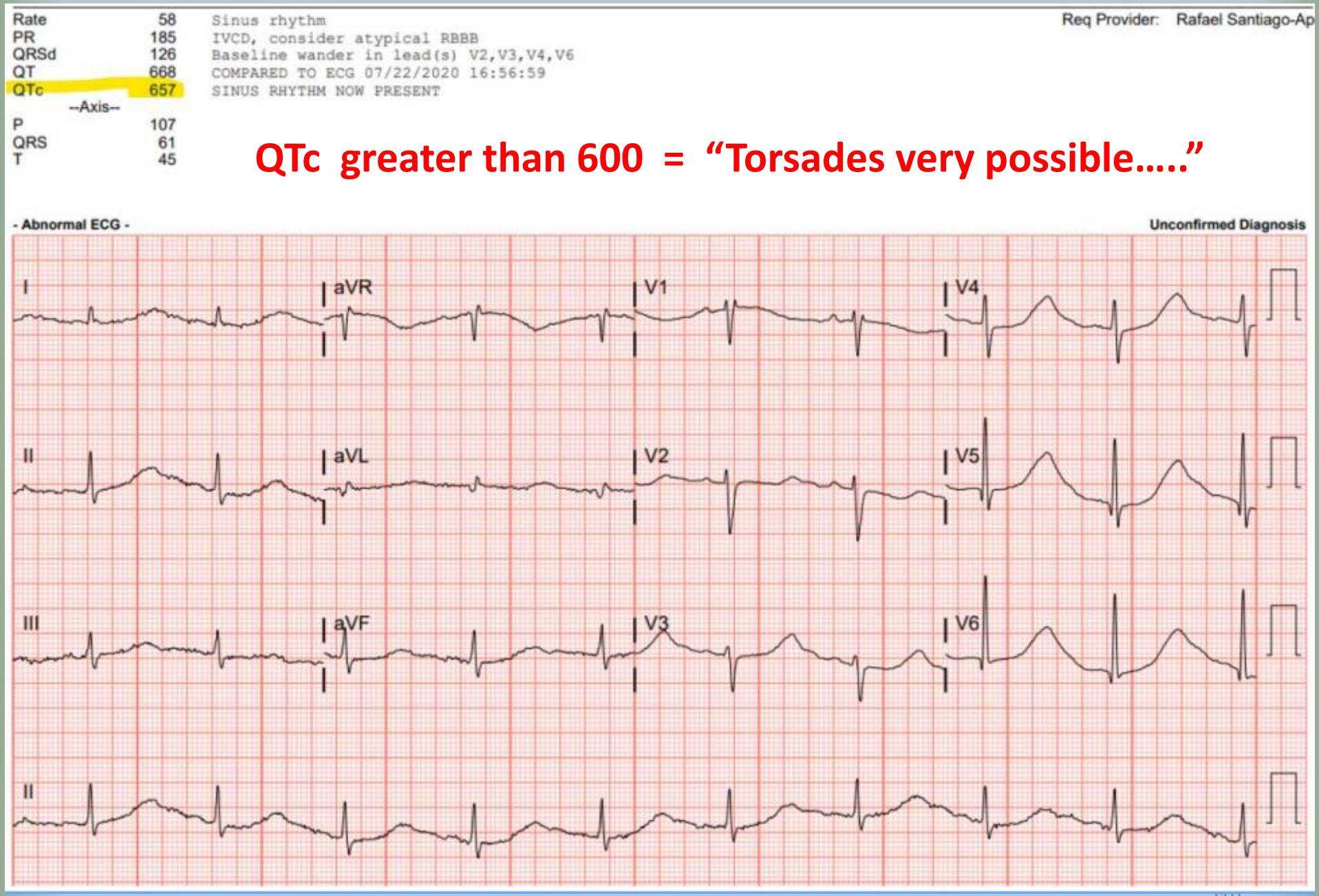
ABC s

NO PULSE	PULSE - UNSTABLE	PULSE - STABLE
GO TO V - FIB ALGORITHM !	▪ IMMEDIATE DEFIBRILLATION START IV CONSIDER SEDATION	▪ O2 / IV / EKG ▪ MAGNESIUM INFUSION 1 - 2 gm OVER 5 - 60 min,

DO NOT give PROCAINAMIDE, AMIODARONE, or SOTALOL to patients with TORSADES or POLYMORPHIC VT !!!

QTc Values:

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

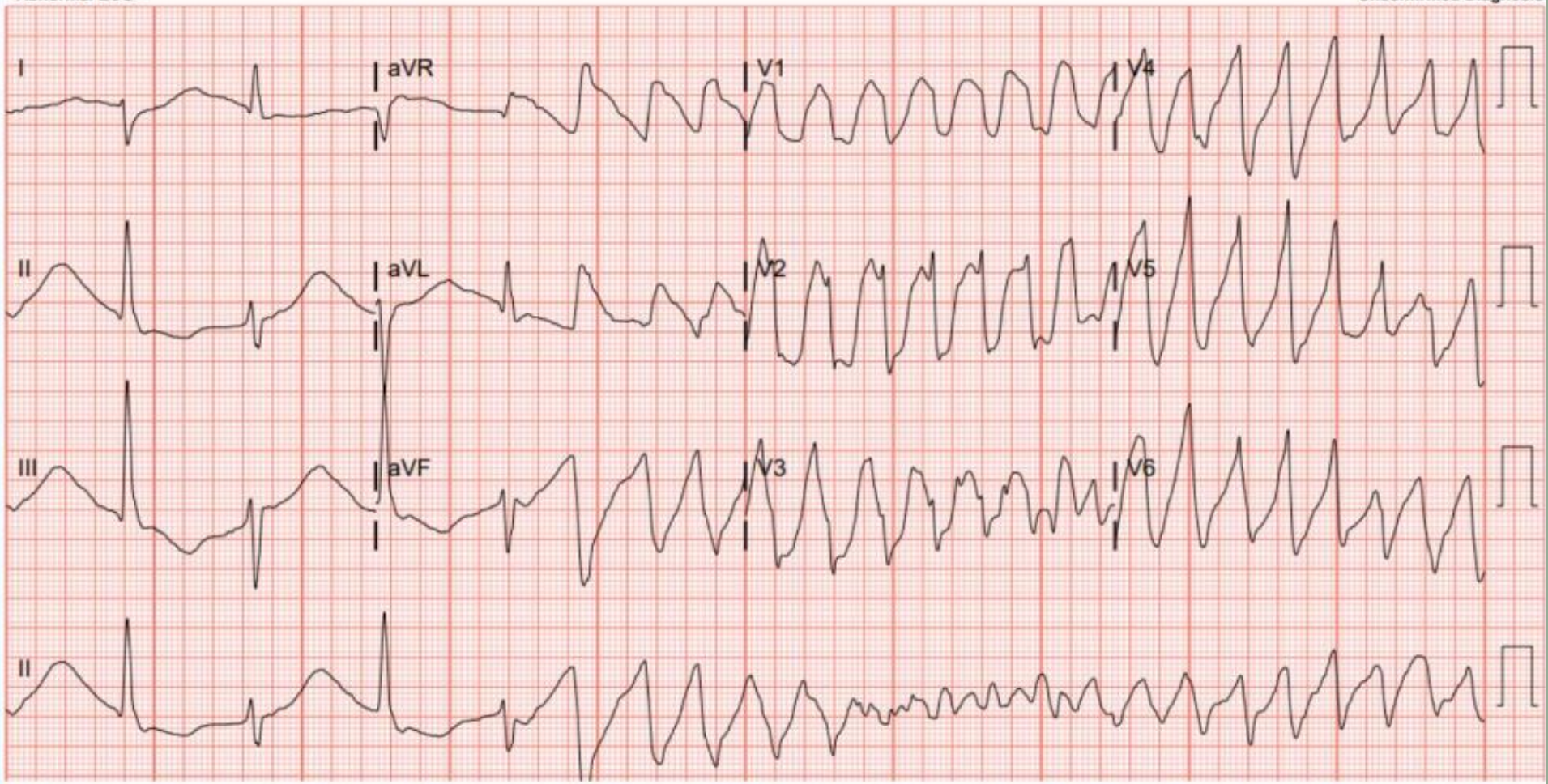

Rate 58
PR 185
QRSd 126
QT 668
QTc 657
--Axis--
P 107
QRS 61
T 45
Sinus rhythm
IVCD, consider atypical RBBB
Baseline wander in lead(s) V2,V3,V4,V6
COMPARED TO ECG 07/22/2020 16:56:59
SINUS RHYTHM NOW PRESENT
Req Provider: Rafael Santiago-Ap
QTc greater than 600 = "Torsades very possible….."
- Abnormal ECG -
Unconfirmed Diagnosis
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

Rate	156	Atrial fibrillation
PR		Ventricular tachycardia, unsustained
QRSd	138	RBBB and LPFB
QT	456	Repol abnrm suggests ischemia, diffuse leads
QTc	735	Baseline wander in lead(s) II,III,aVR,aVF,V1,V2,V3,V4
--Axis--		COMPARED TO ECG 07/22/2020 15:32:52
P		ATRIAL FIBRILLATION NOW PRESENT
QRS	102	VENTRICULAR TACHYCARDIA NOW PRESENT
T	185	LEFT POSTERIOR FASCICULAR BLOCK NOW PRESENT
		RIGHT BUNDLE-BRANCH BLOCK NOW PRESENT
		POSSIBLE ISCHEMIA NOW PRESENT
		PROLONGED QT INTERVAL NO LONGER PRESENT

Req Provider: Rafael Santiago-A

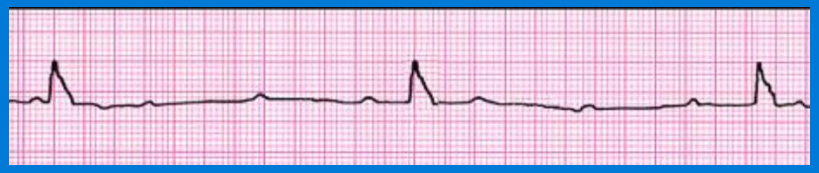
- Abnormal ECG -

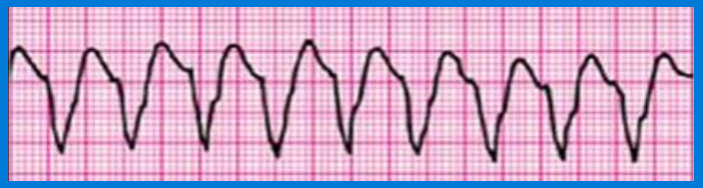
Unconfirmed Diagnosis

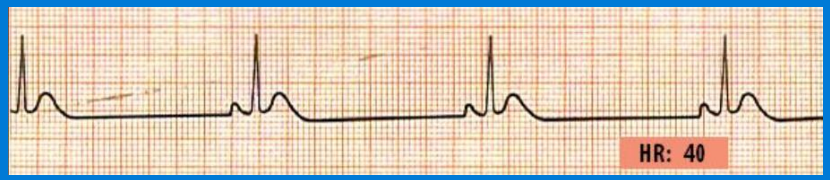


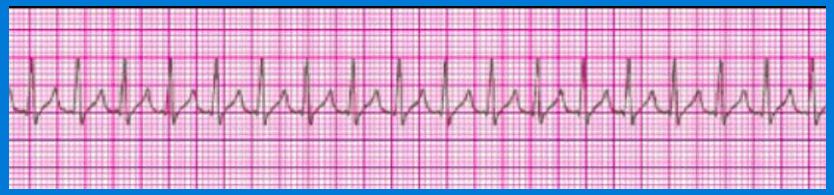
CARDIOGENIC SHOCK

- Heart Rate – TOO SLOW or TOO FAST
  - Wide QRS
  - Narrow QRS







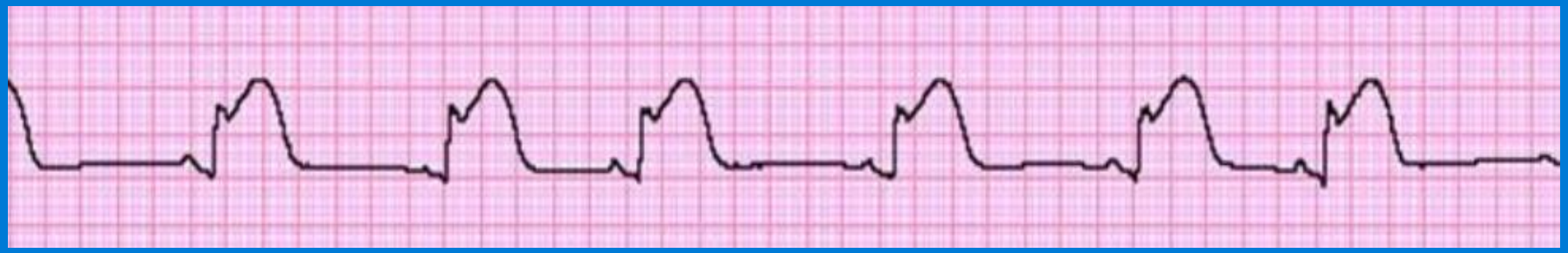


CARDIOGENIC SHOCK

- **Heart Rate:**
  - **Should be between 50 – 150**

CARDIOGENIC SHOCK

- **Heart Rate:**
  - **Should be between 50 – 150**
- **Decreased Contractility:**
  - **STEMI / Acute Coronary Syndrome (vascular)**



CARDIOGENIC SHOCK

- **Heart Rate:**
  - **Should be between 50 – 150**
- **Decreased Contractility:**
  - **STEMI / Acute Coronary Syndrome (vascular)**
  - **Myocarditis (muscle dysfunction)**

CARDIOGENIC SHOCK

- **Heart Rate:**
  - **Should be between 50 – 150**
- **Decreased Contractility:**
  - **STEMI / Acute Coronary Syndrome (vascular)**
  - **Myocarditis (muscle dysfunction)**

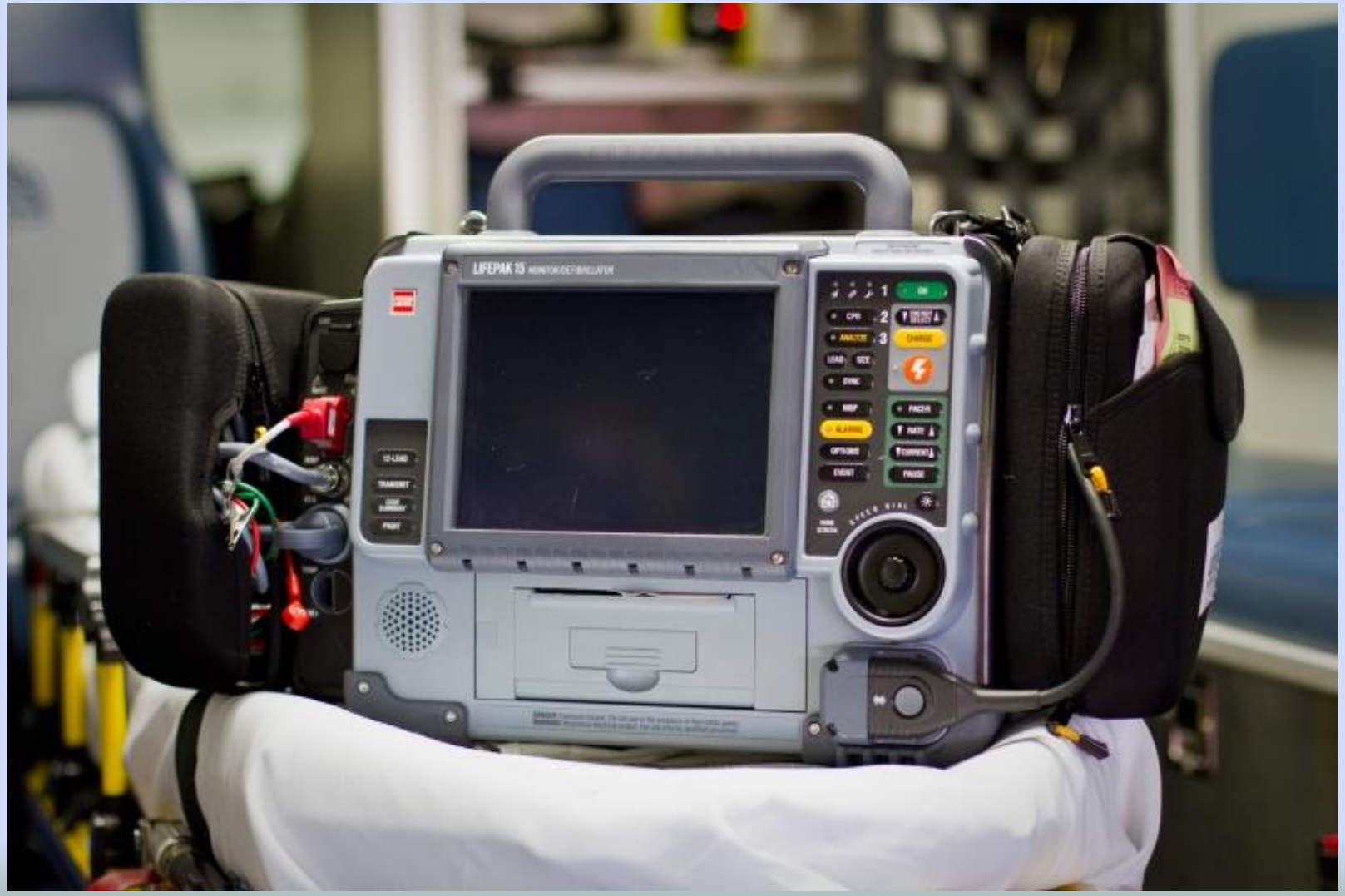
    Often mimics STEMI on the ECG. Often "challenging" for advanced practitioners to diagnose.

Initial Patient Workup:

- If patient has ANY symptoms of ACS, get a

STAT 12 Lead ECG

EMS 12 Lead ECG



In-Hospital 12 Lead ECG

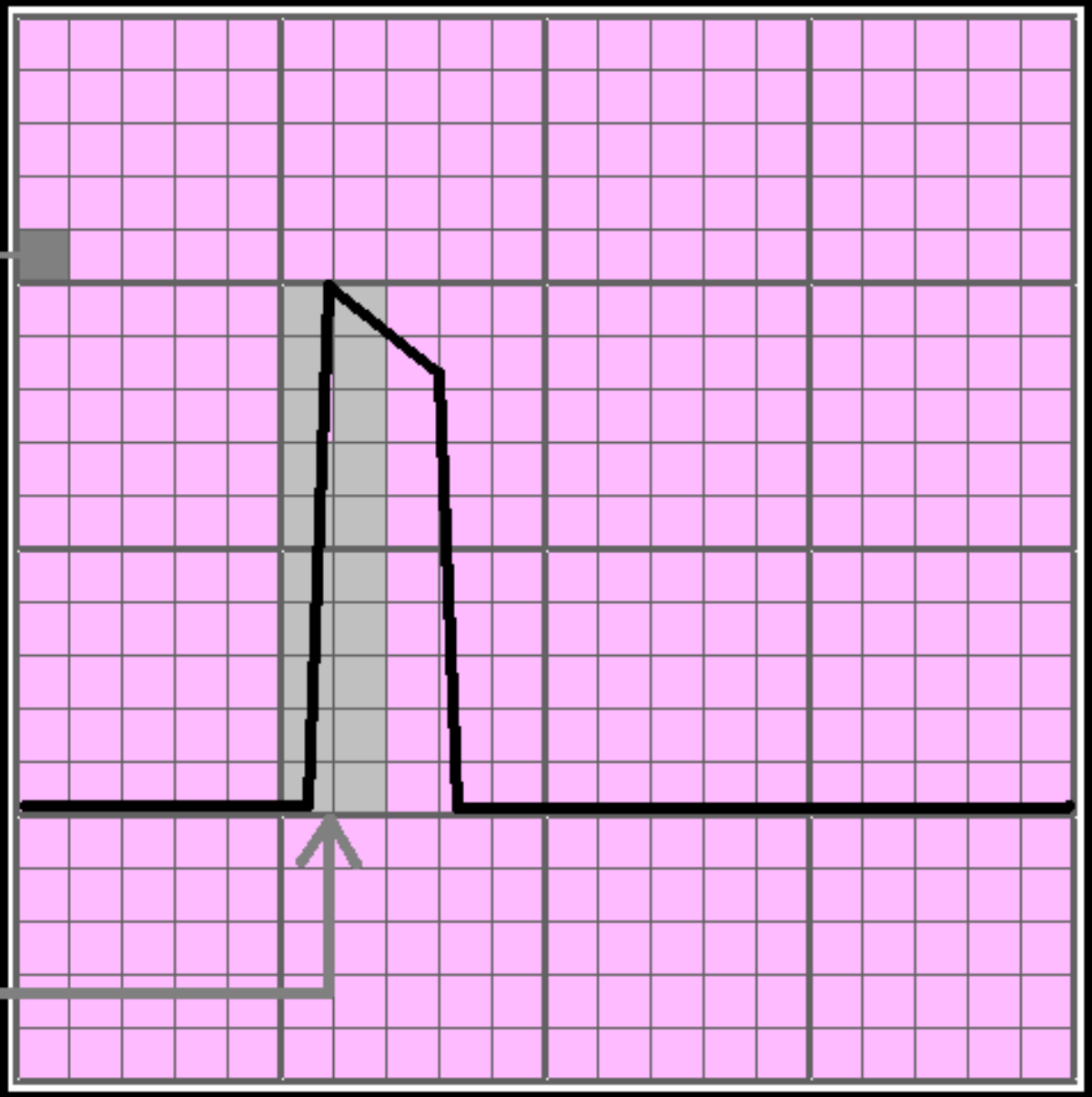
10 wires . . .

- 4 limb leads
- 6 chest (“V”) leads


MEDFLEET-39615815
LP - 8
LIFEPAK 15 MONITOR/DEFIBRILLATOR
ON
CPR
ANALYZE
CHARGE
LEAD
SIZE
SYNC
NIBP
ALARMS
OPTIONS
EVENT
PACER
RATE
CURRENT
PAUSE
12-LEAD
TRANSMIT
PRINT

EKG 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



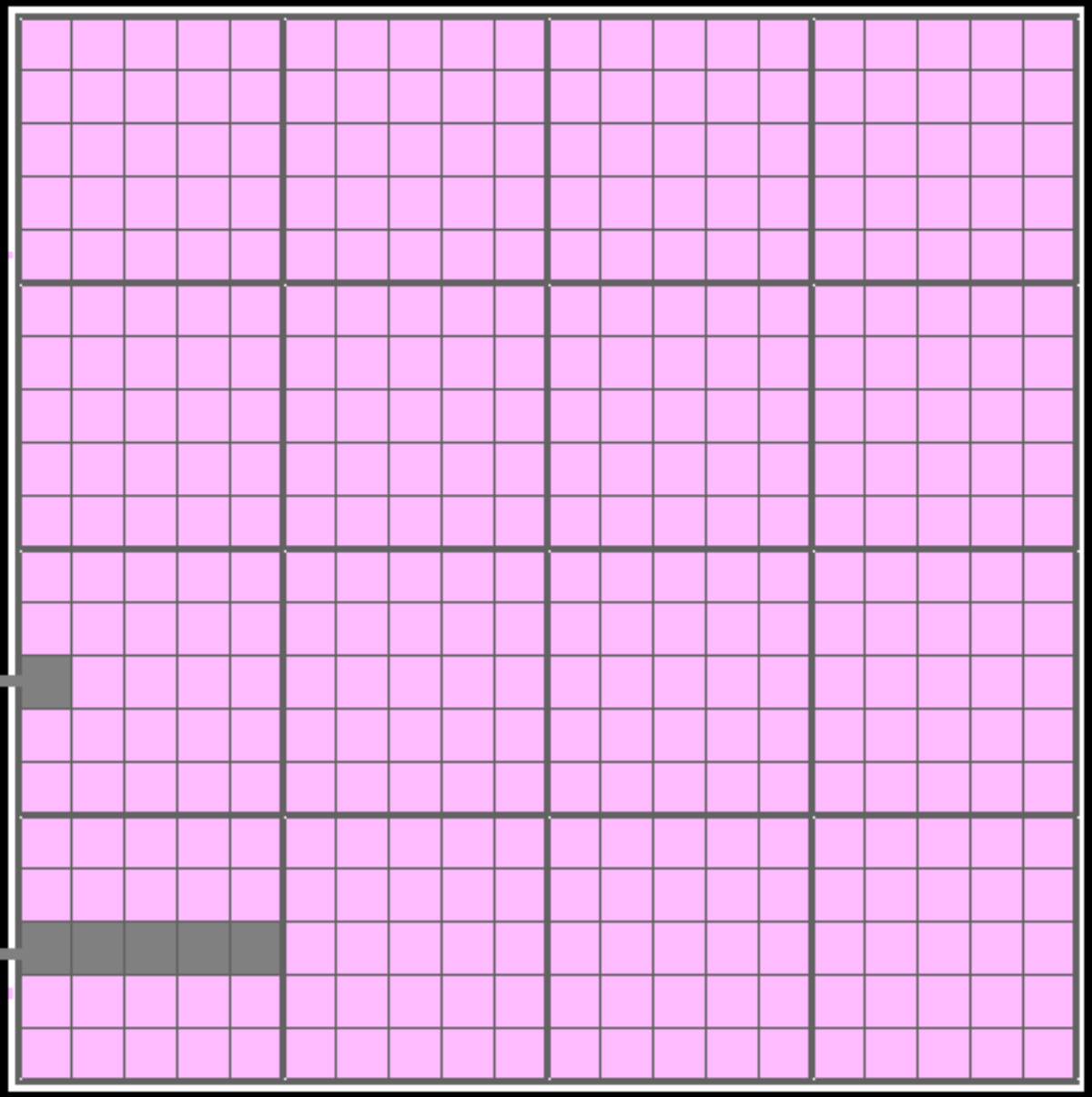
EKG PAPER - THE HORIZONTAL AXIS:

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)

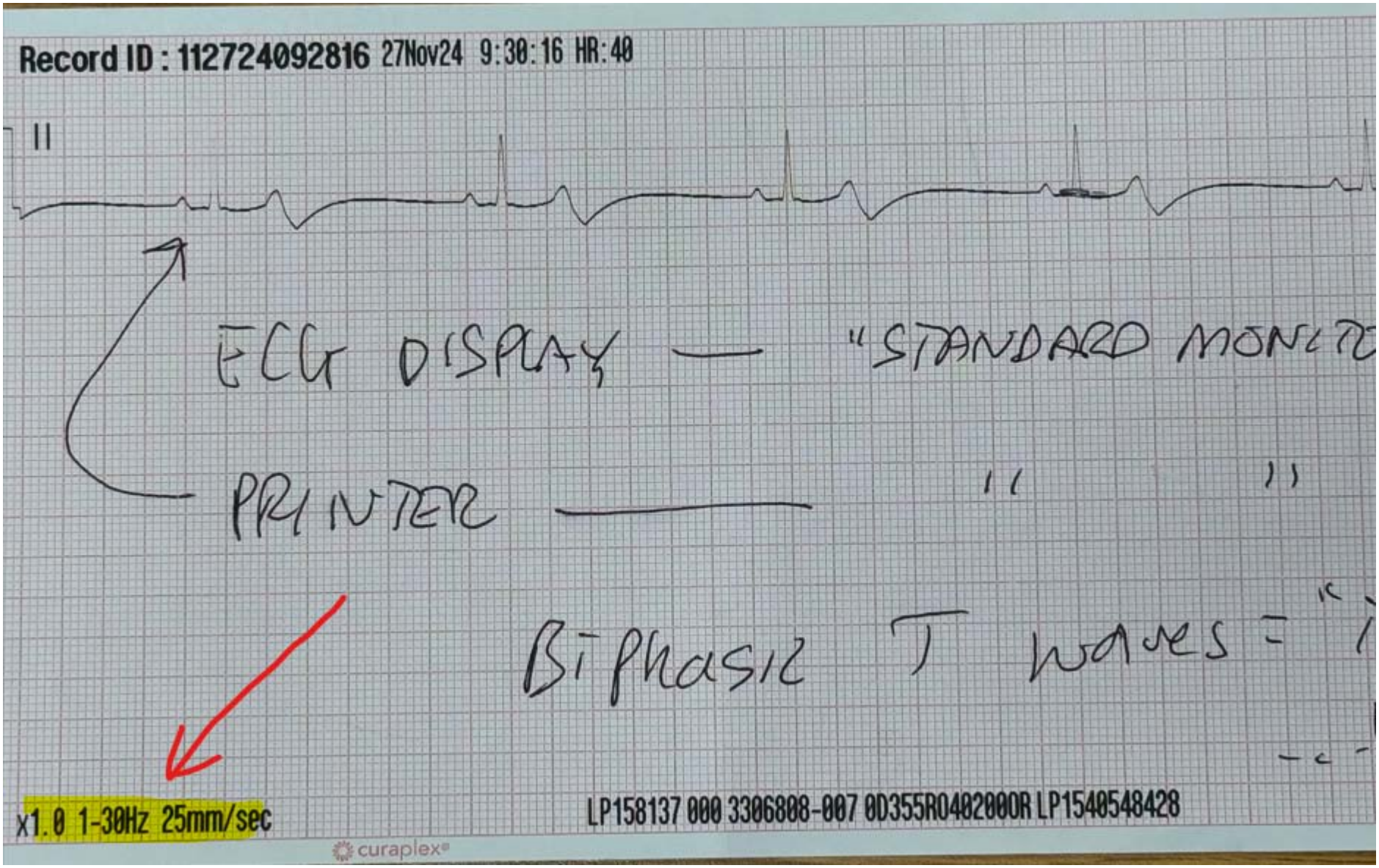


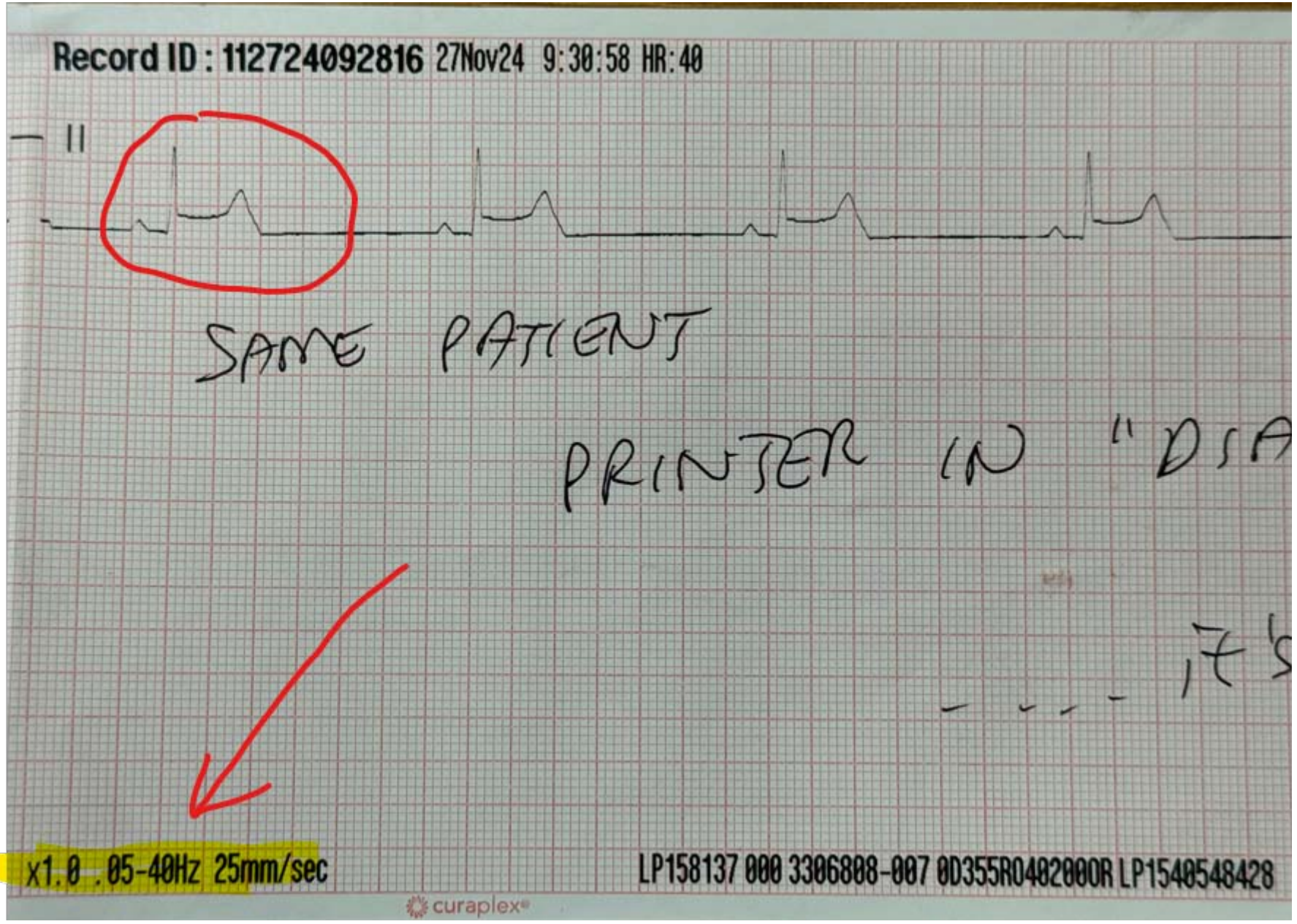
If you're going to use your ECG monitor to evaluate the patient's:

- J Points
- ST Segments
- T Waves

It MUST be in the "DIAGNOSTIC" mode

(Not the normal "Monitoring" mode).....


Record ID : 112724092816 27Nov24 9:30:16 HR:40
II
ECG DISPLAY — "STANDARD MONITO
PRINTER —— " "
BIPHASIC T WAVES = "I
x1.0 1-30Hz 25mm/sec
LP158137 000 3306808-007 0D355R0402000R LP1540548428
curaplex


Record ID : 112724092816 27Nov24 9:30:58 HR:40
II
SAME PATIENT
PRINTER IN "DSA
x1.0 .05-40Hz 25mm/sec
LP158137 000 3306808-007 0D355R0402000R LP1540548428
curaplex

MONITOR mode bandwidth:
1.0 – 30 Hz

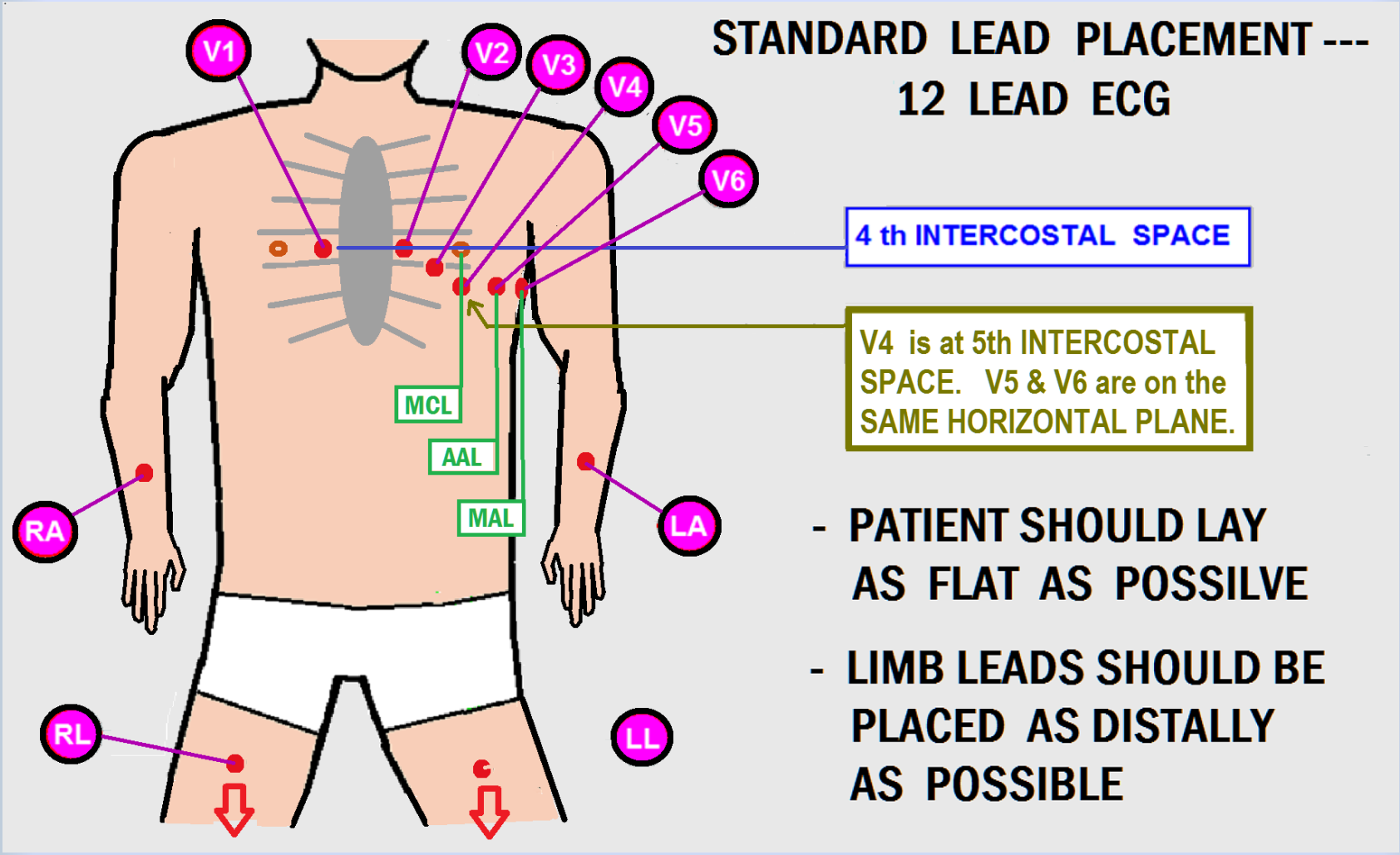
DIAGNOSTIC mode bandwidth:
0.5 - 40 Hz

MONITOR mode bandwidth:
1.0 – 30 Hz

DIAGNOSTIC mode bandwidth:
0.5 - 40 Hz

☞ ***A 12 Lead ECG is ALWAYS recorded in "DIAGNOSTIC Mode"***

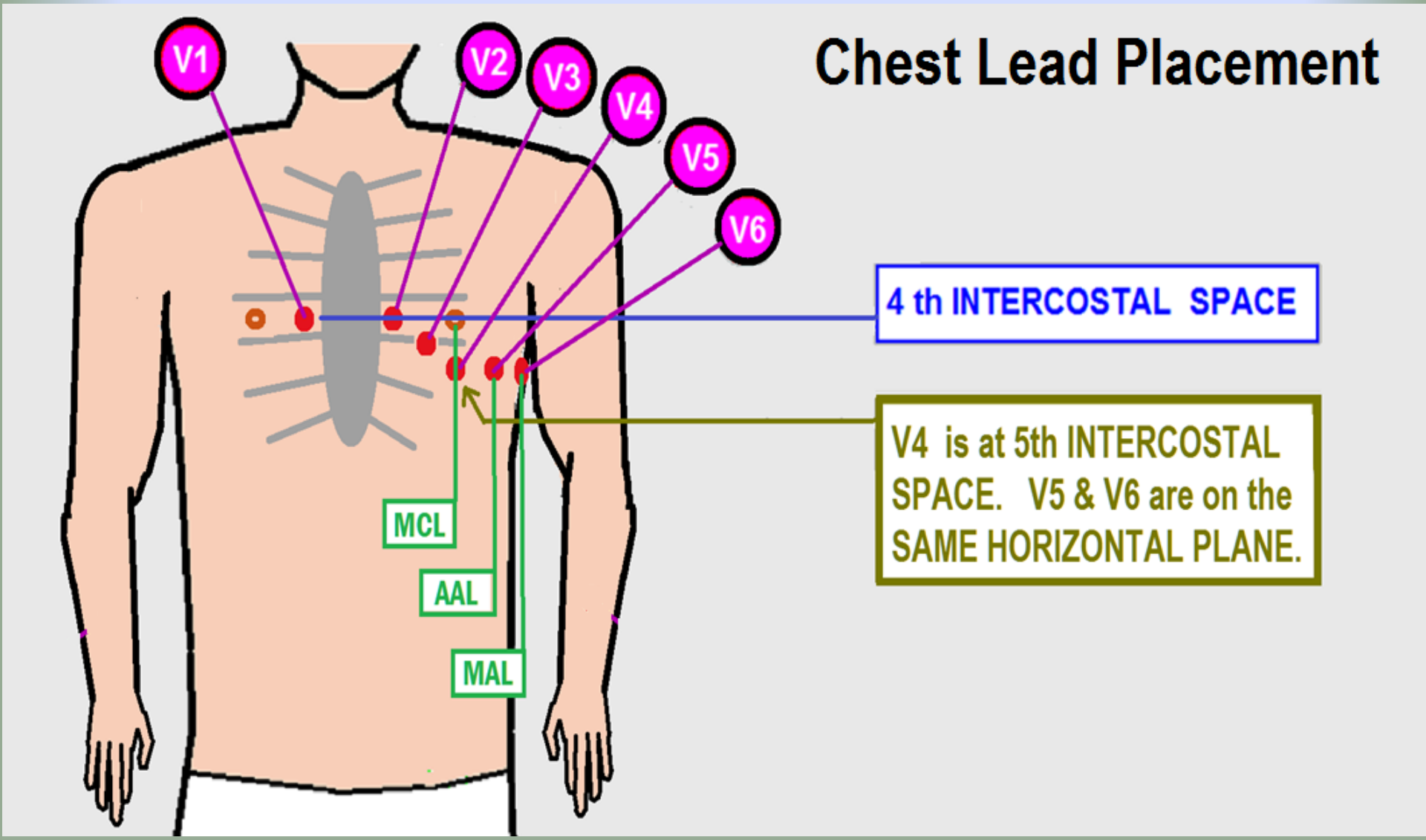
Obtaining the 12 Lead ECG



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

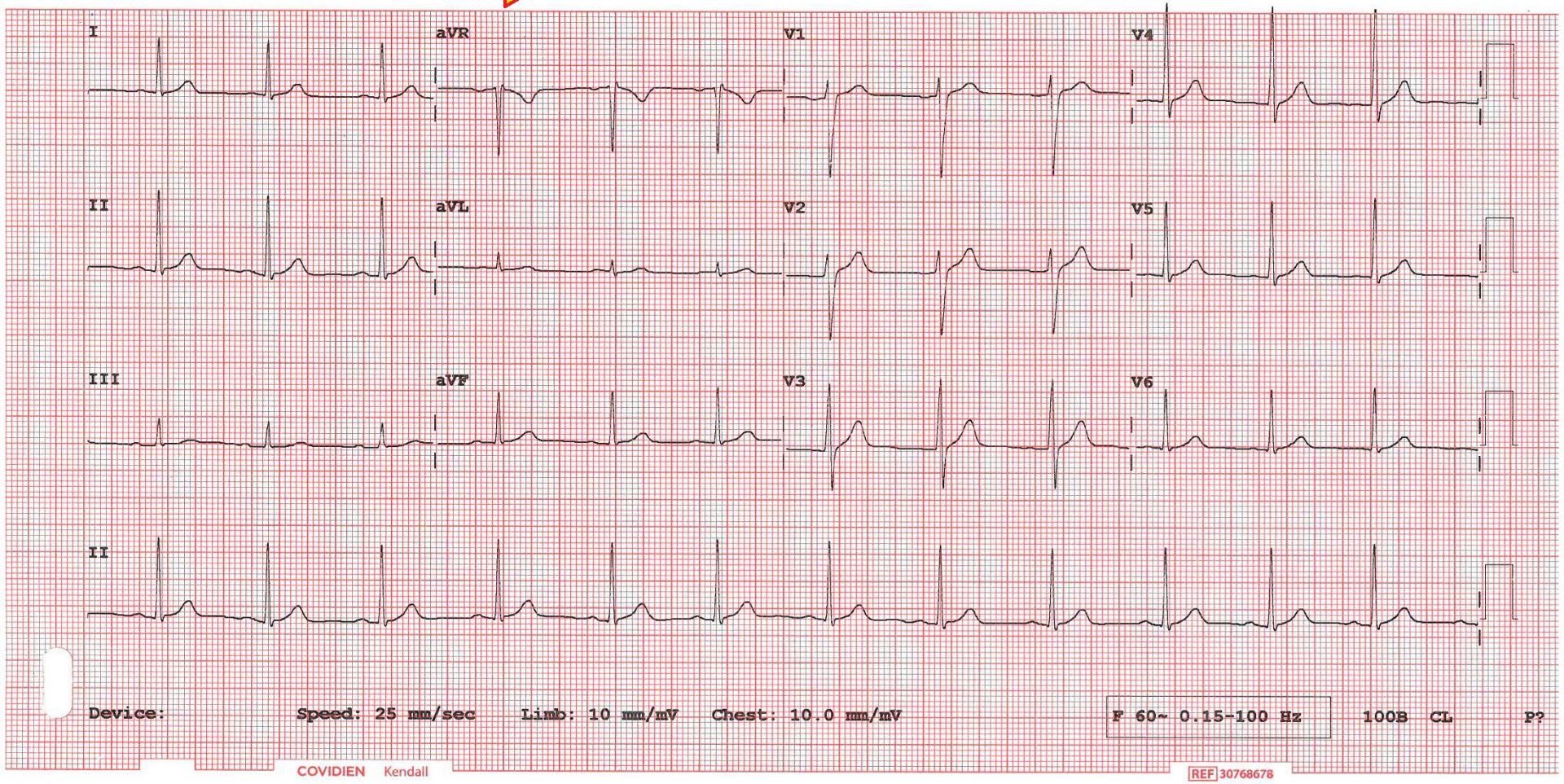
12 Lead; Standard Placement

[redacted] TECH SD

[redacted]

- NORMAL ECG -

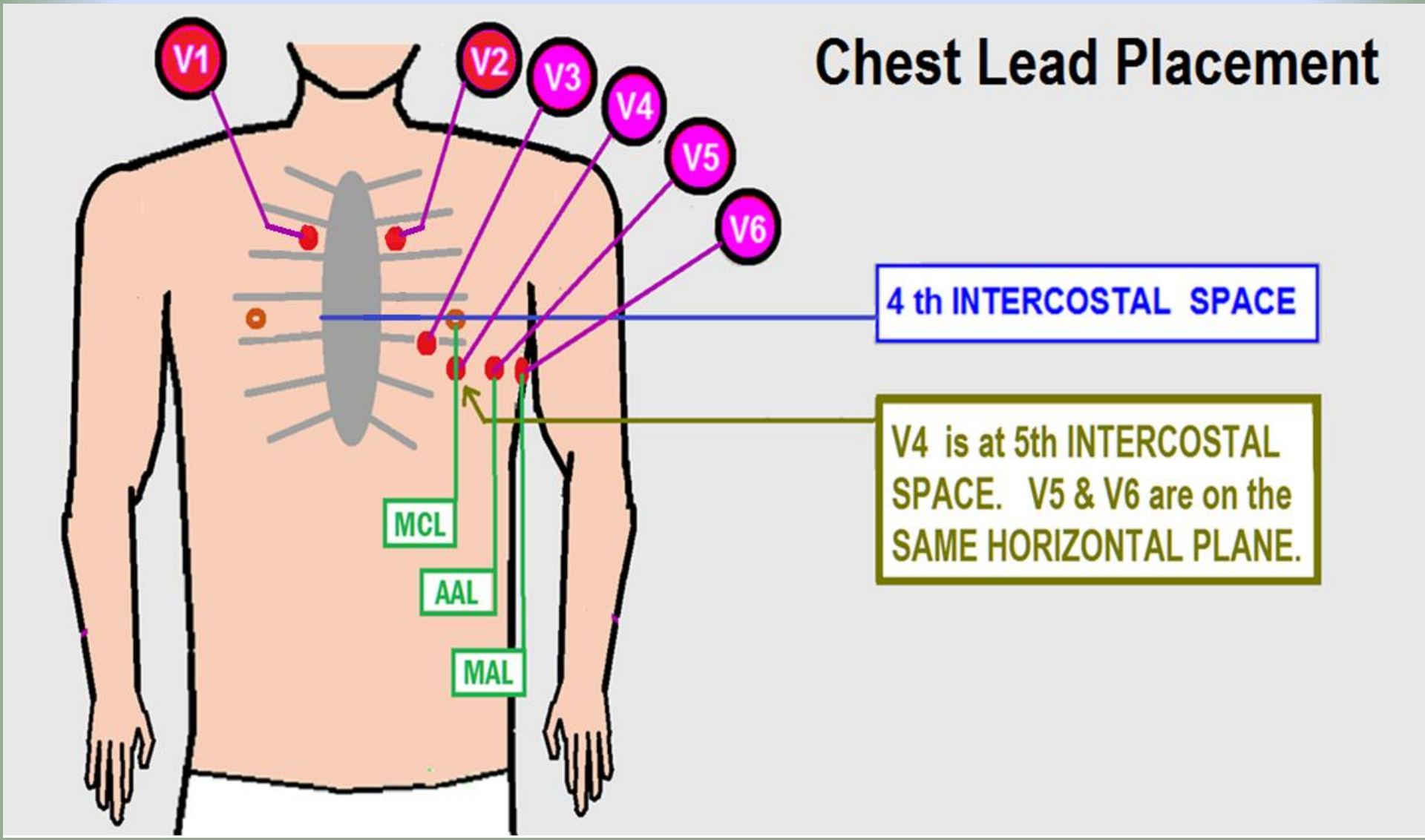
Unconfirmed Diagnosi



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] [redacted] [redacted] 5:20:58 AM

DOB [redacted]1988 30 Years Female (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

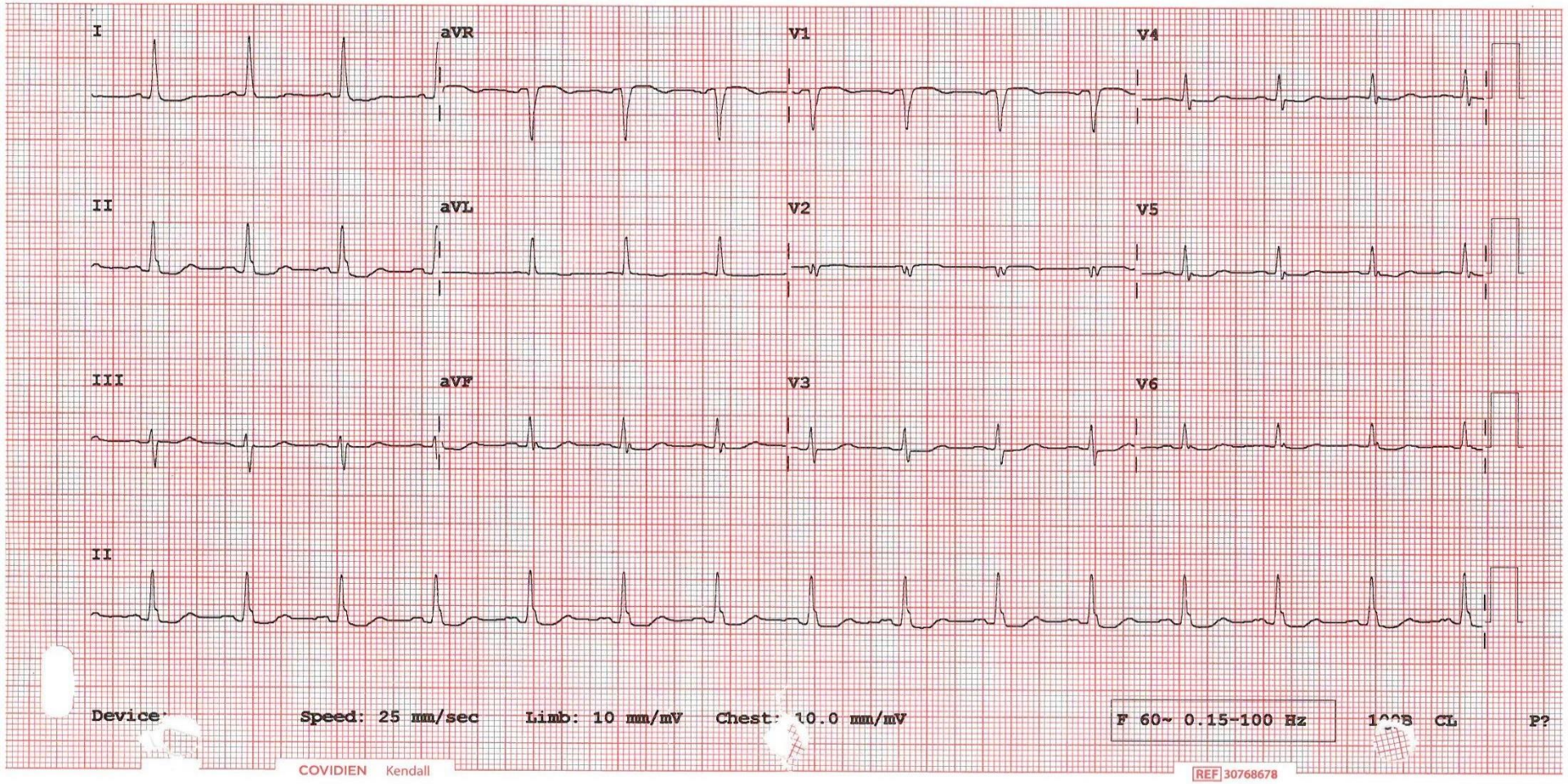
--AXIS--
P 46
QRS 24
T 86

12 Lead; Standard Placement

- ABNORMAL ECG -

Unconfirmed Diagnosis

[redacted]



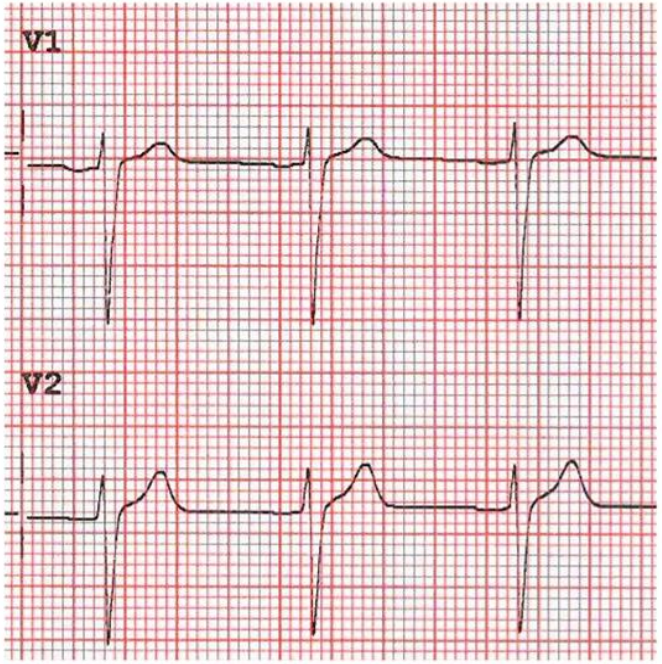
AHA/ACC/HRS Scientific Statement

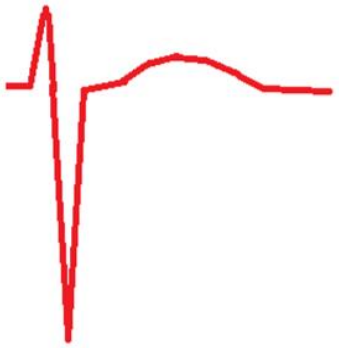
Recommendations for the Standardization and Interpretation of the Electrocardiogram

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]

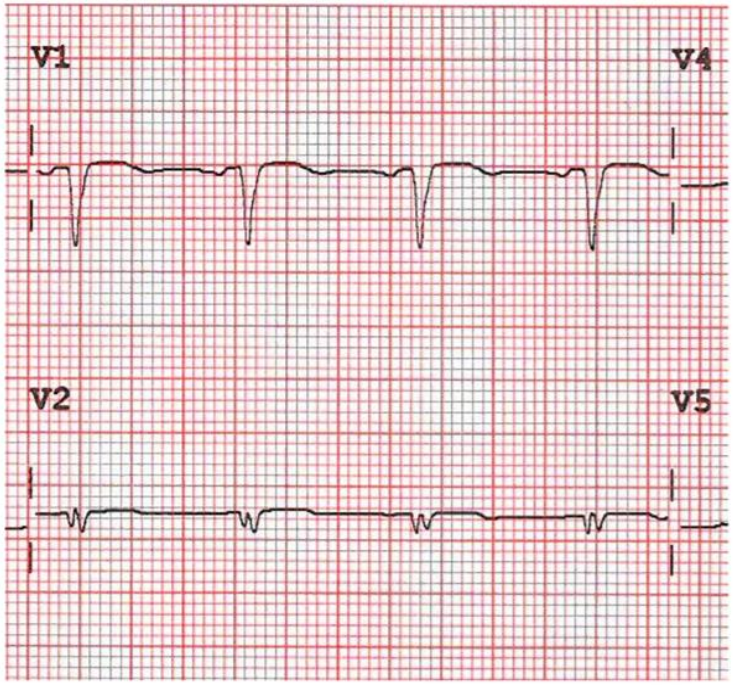
Correct Lead Placement





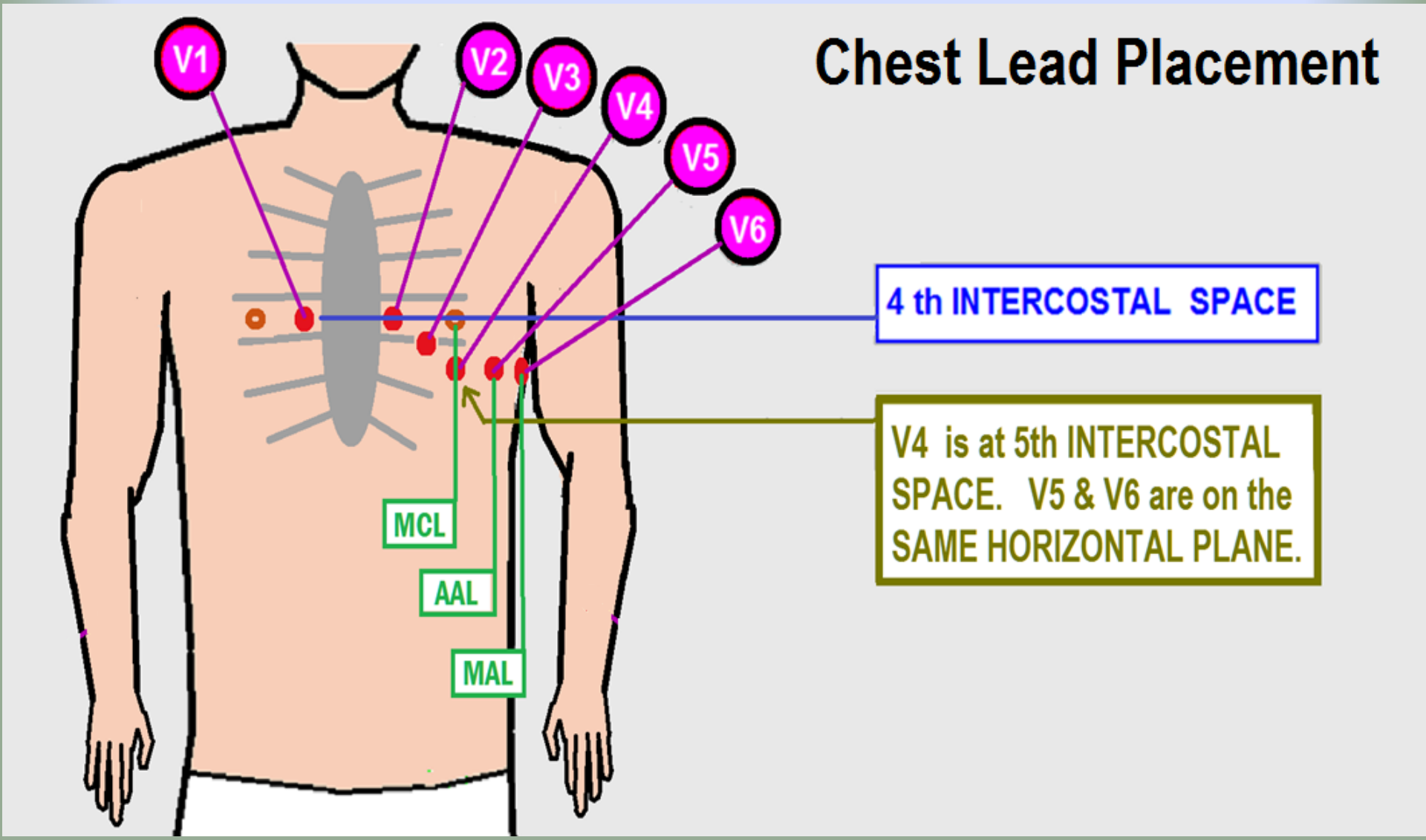
RS = NO old MI

Incorrect Lead Placement

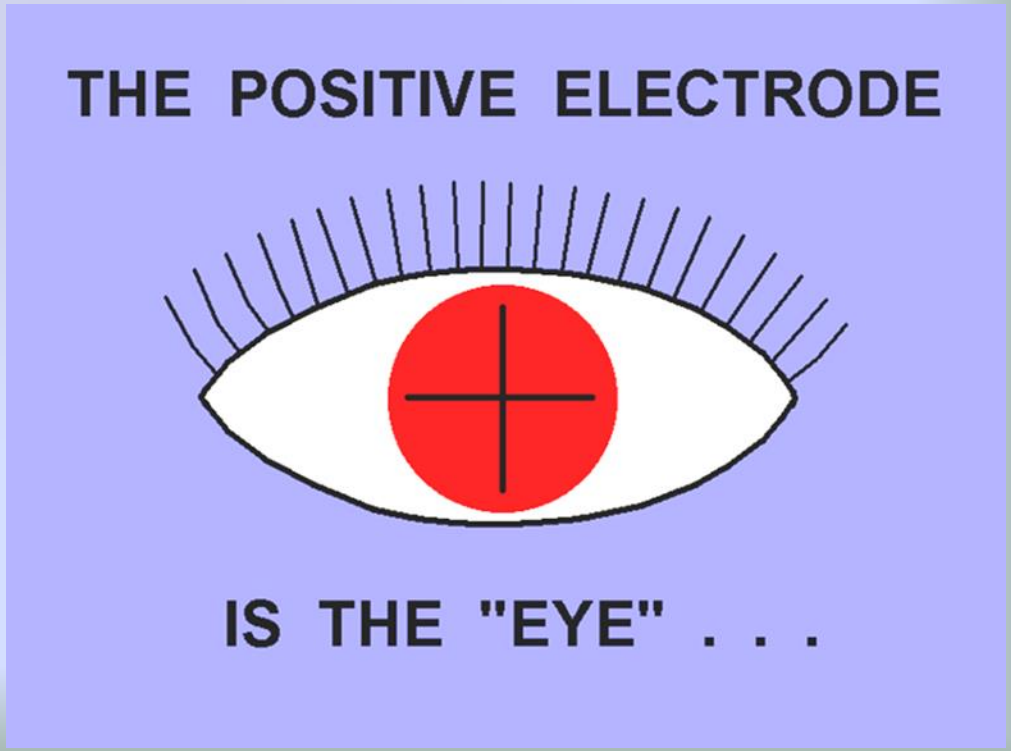


QS = old MI

CORRECT Lead placement:



What part of the HEART does each lead SEE ?



AREAS VIEWED by 12 LEAD ECG

AVR

AVL, I

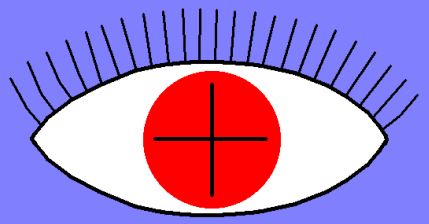
V1, V2

V3, V4

V5, V6

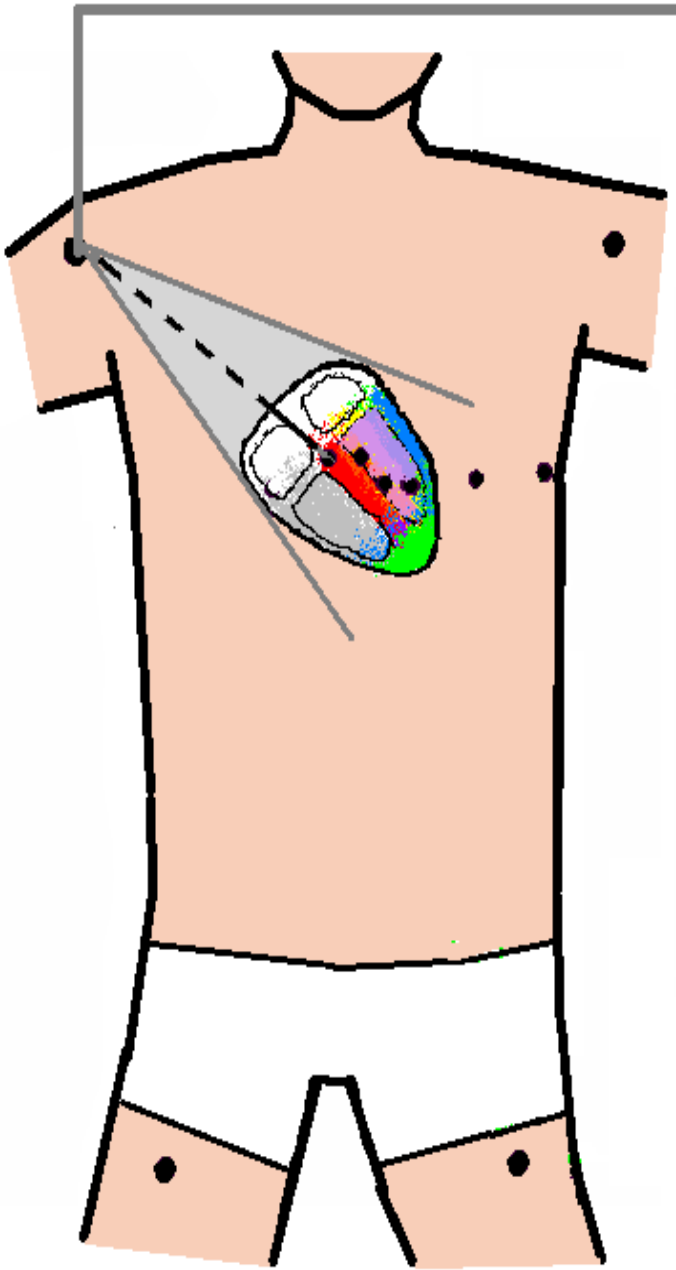
II, III, AVF

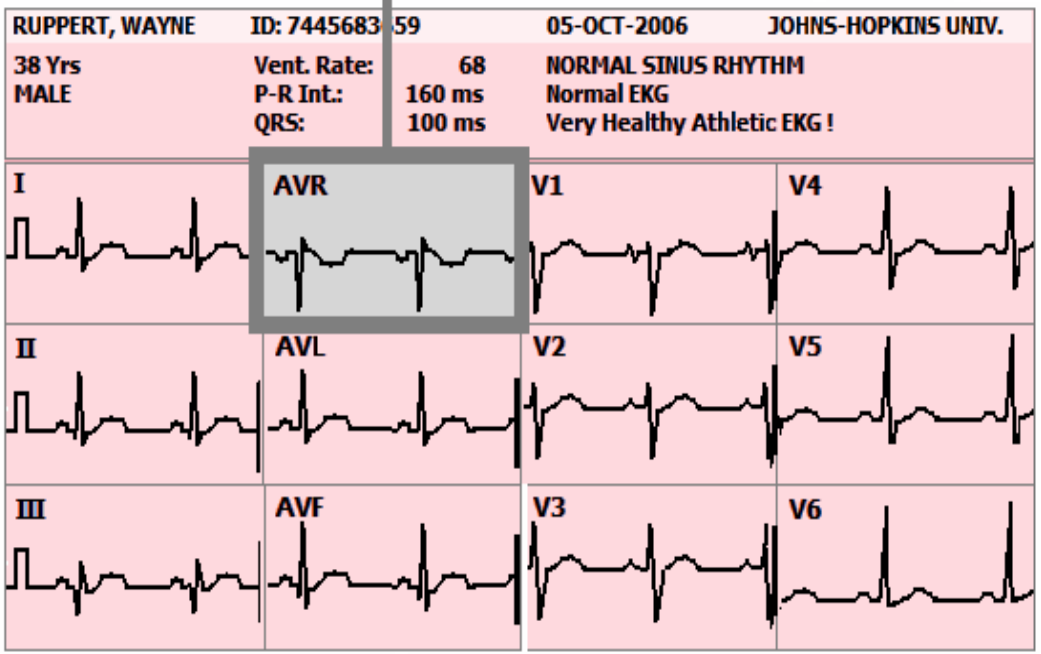
THE POSITIVE ELECTRODE

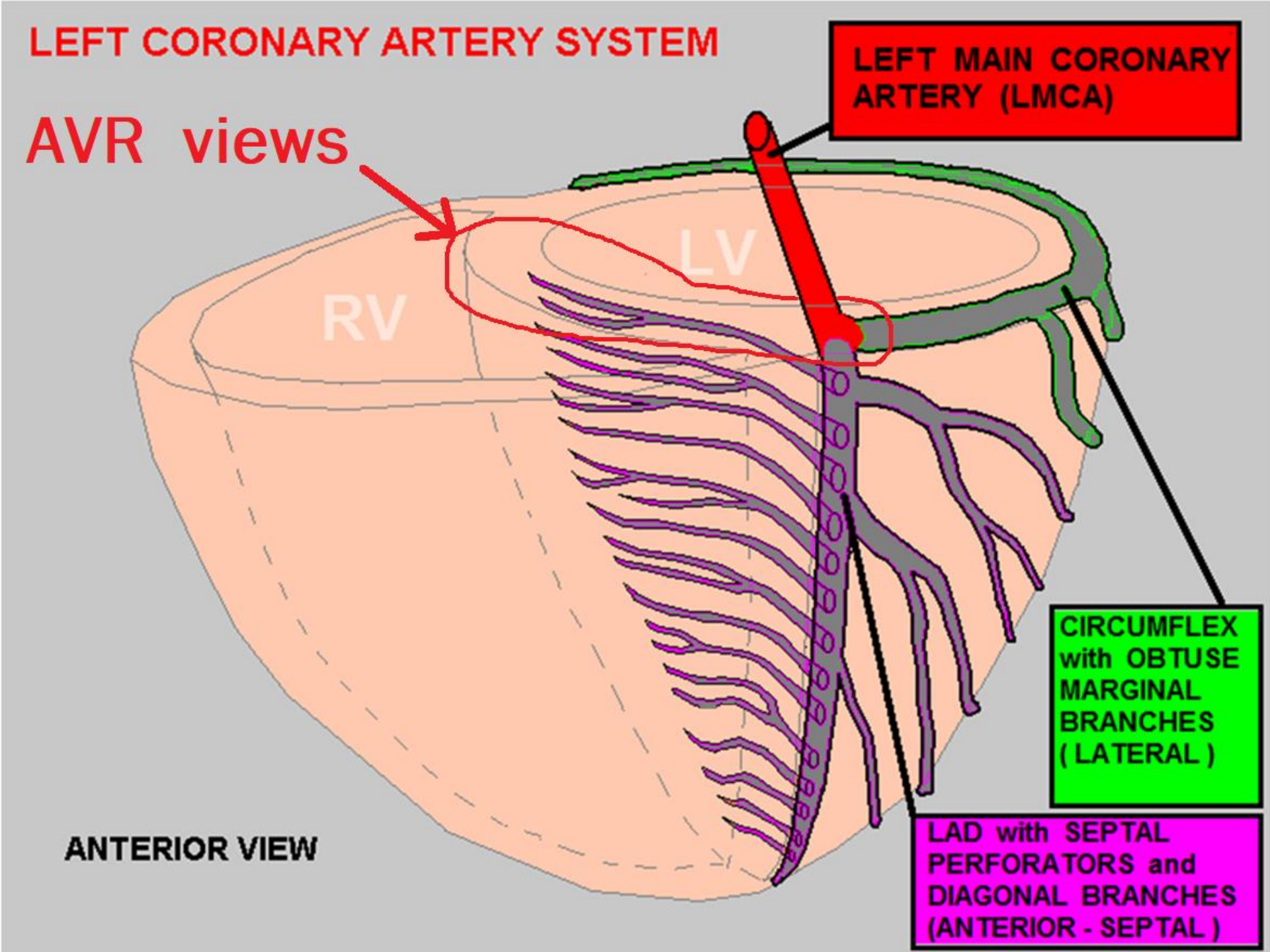


IS THE "EYE" . . .

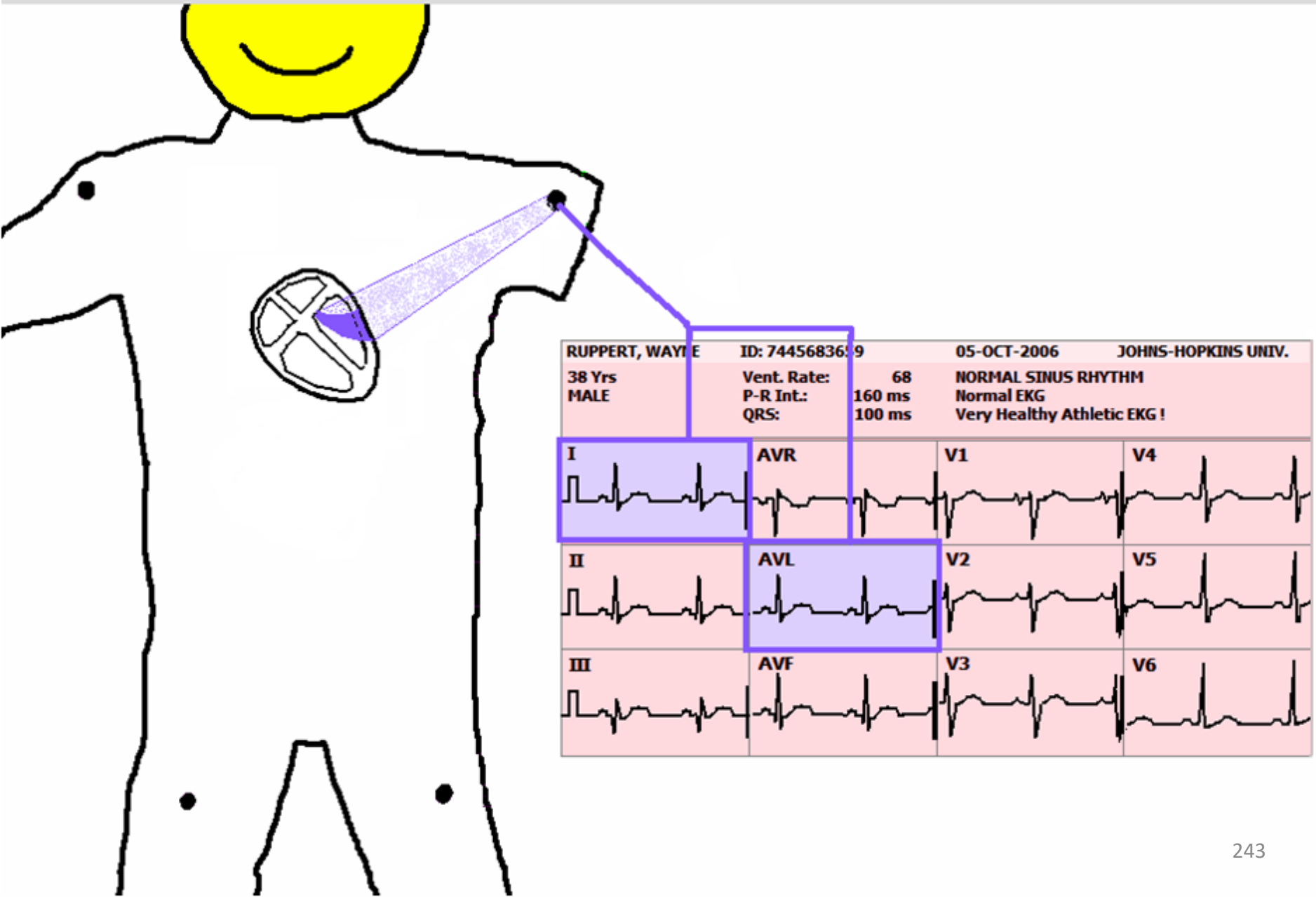
Lead AVR Views the BASILAR SEPTUM (region of the Bundle of His)





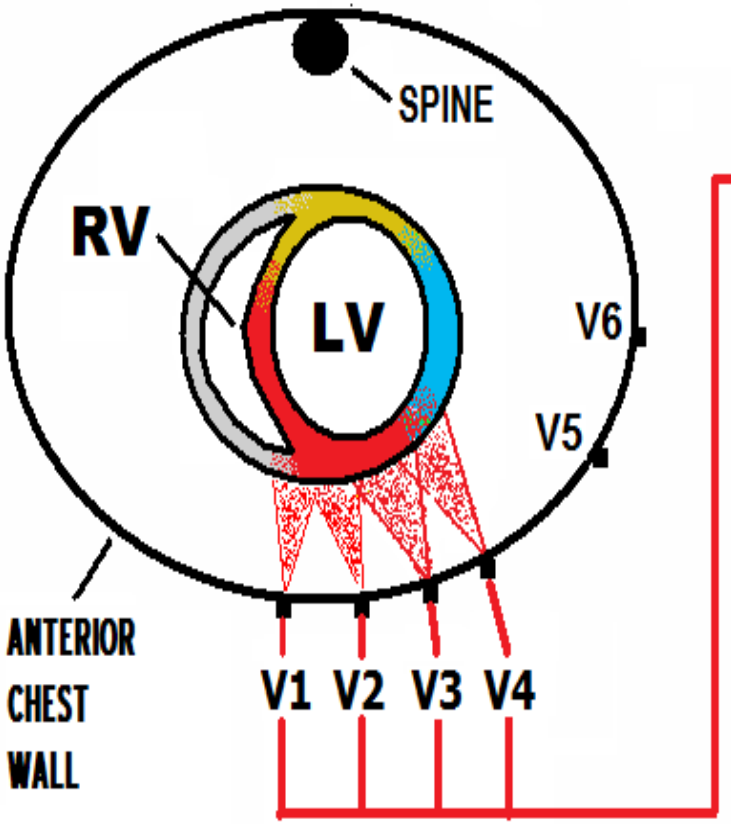

LEFT CORONARY ARTERY SYSTEM
AVR views
LEFT MAIN CORONARY ARTERY (LMCA)
LV
RV
CIRCUMFLEX with OBTUSE MARGINAL BRANCHES (LATERAL)
LAD with SEPTAL PERFORATORS and DIAGONAL BRANCHES (ANTERIOR - SEPTAL)
ANTERIOR VIEW

LEADS I and aVL VIEW the LATERAL - ANTERIOR WALL



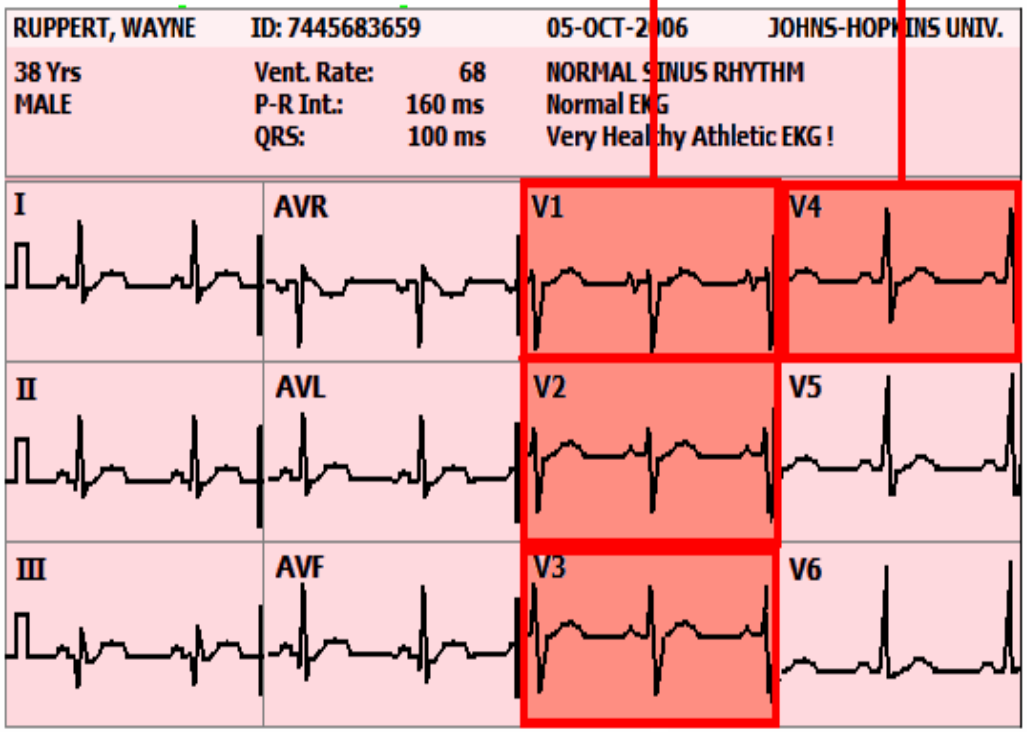
V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL

of the LEFT VENTRICLE



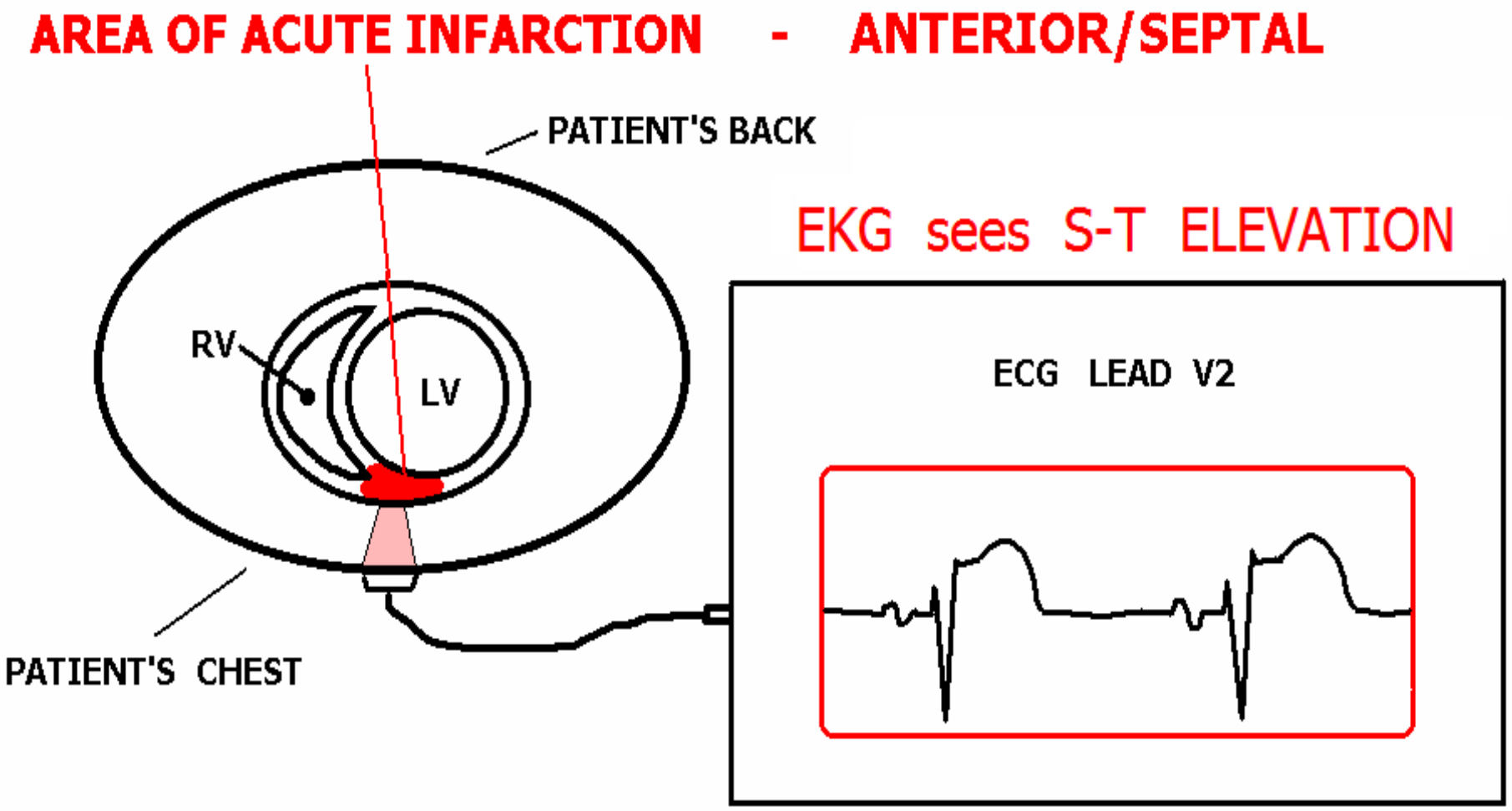
V1, V2 - ANTERIOR / SEPTAL

V3, V4 - ANTERIOR



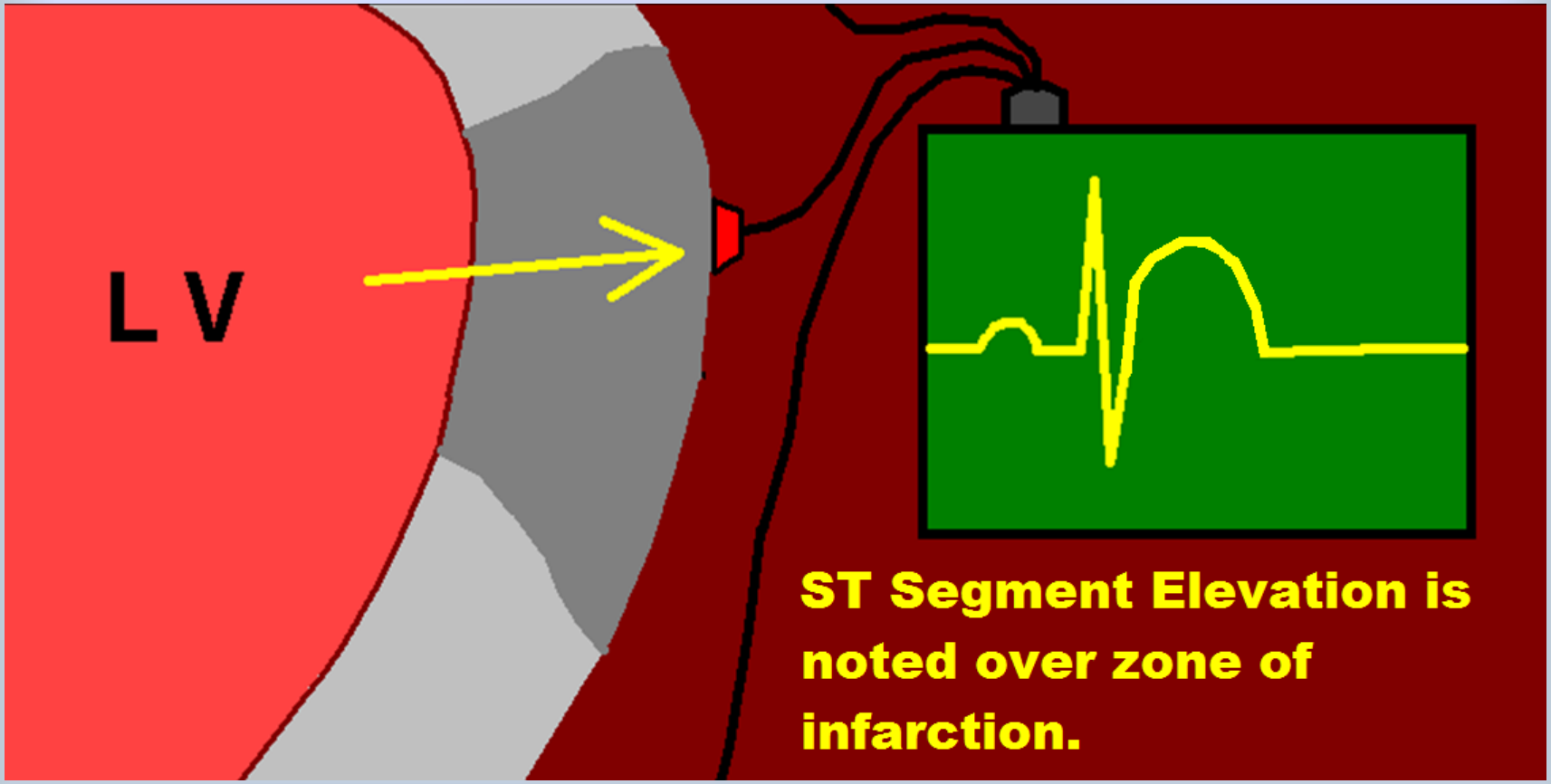
HOW EKG VIEWS INDICATIVE CHANGES

EXAMPLE:



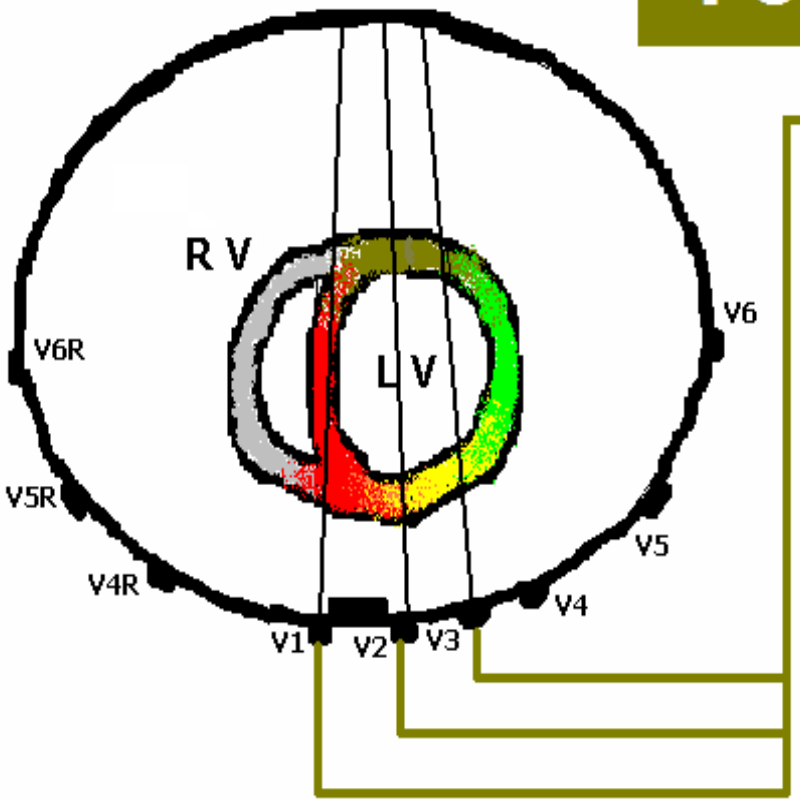
STEMI

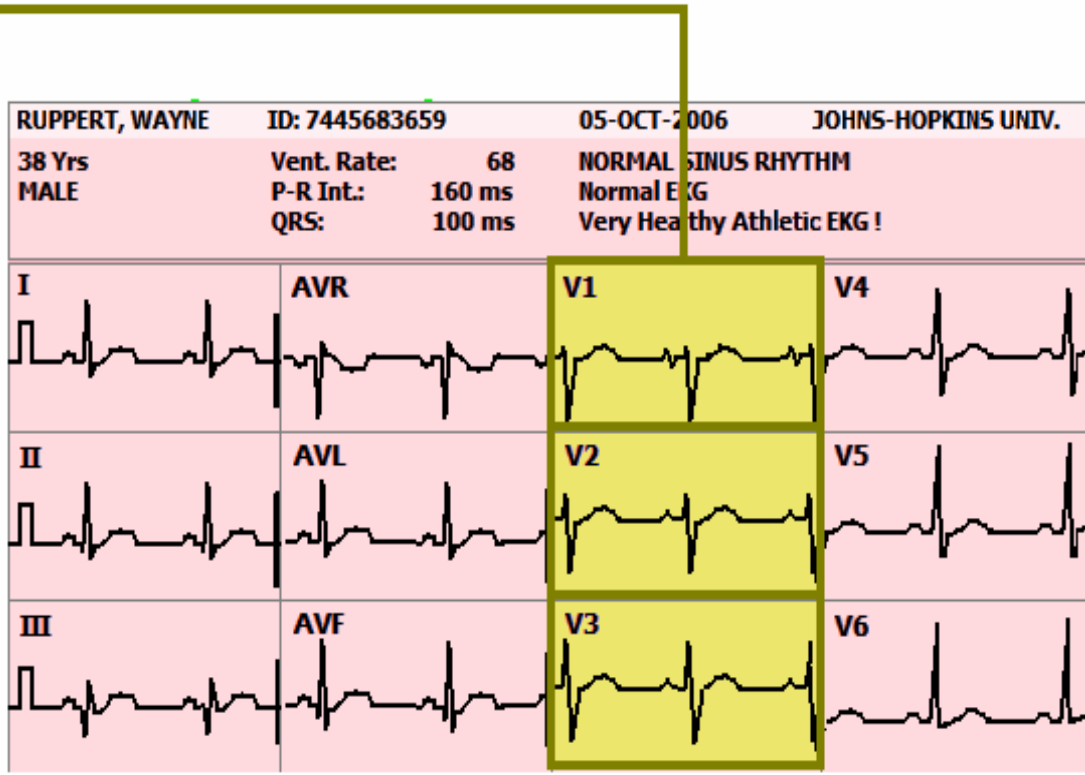
- ST Segment Elevation Myocardial Infarction.



LEADS V1 - V3 *view the*

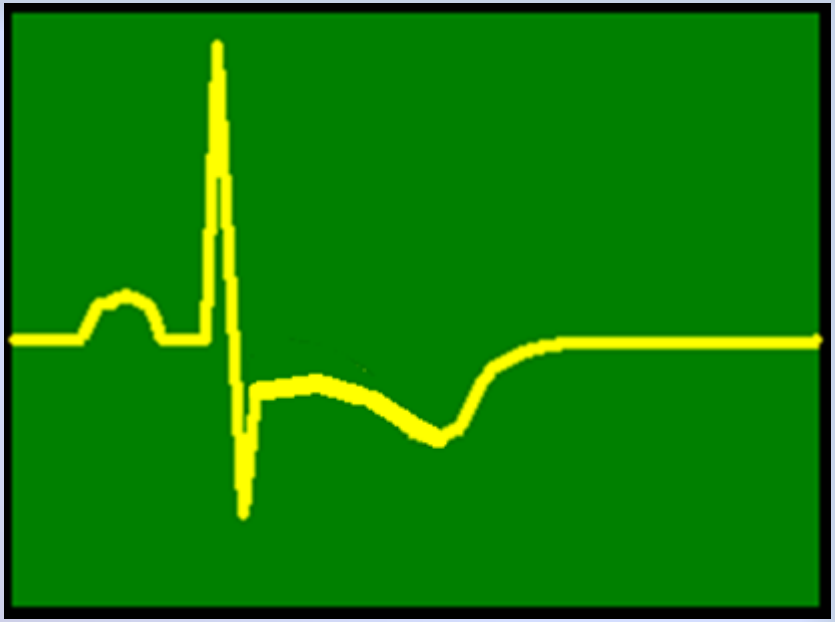
POSTERIOR WALL





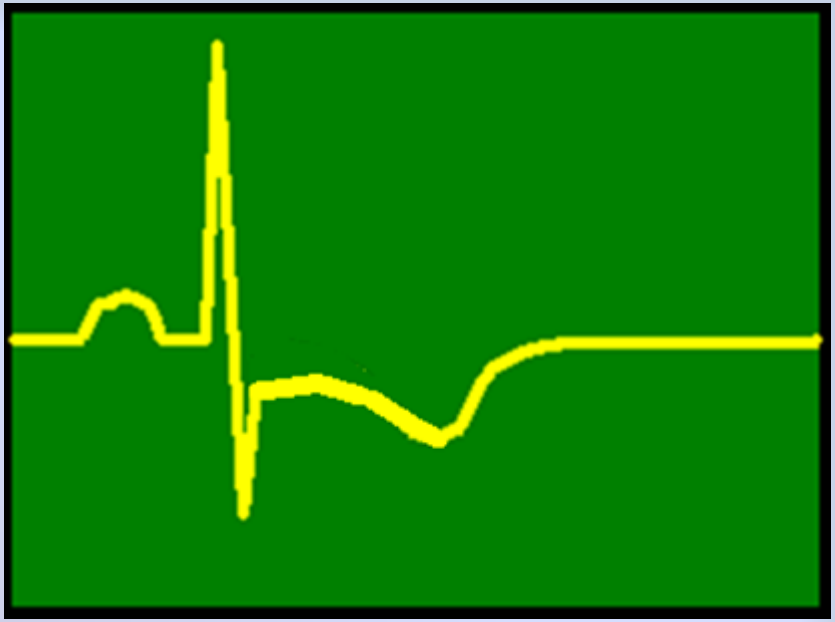
via RECIPROCAL CHANGES.

ST Depression in Leads V1 – V4:



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

ST Depression in Leads V1 – V4:

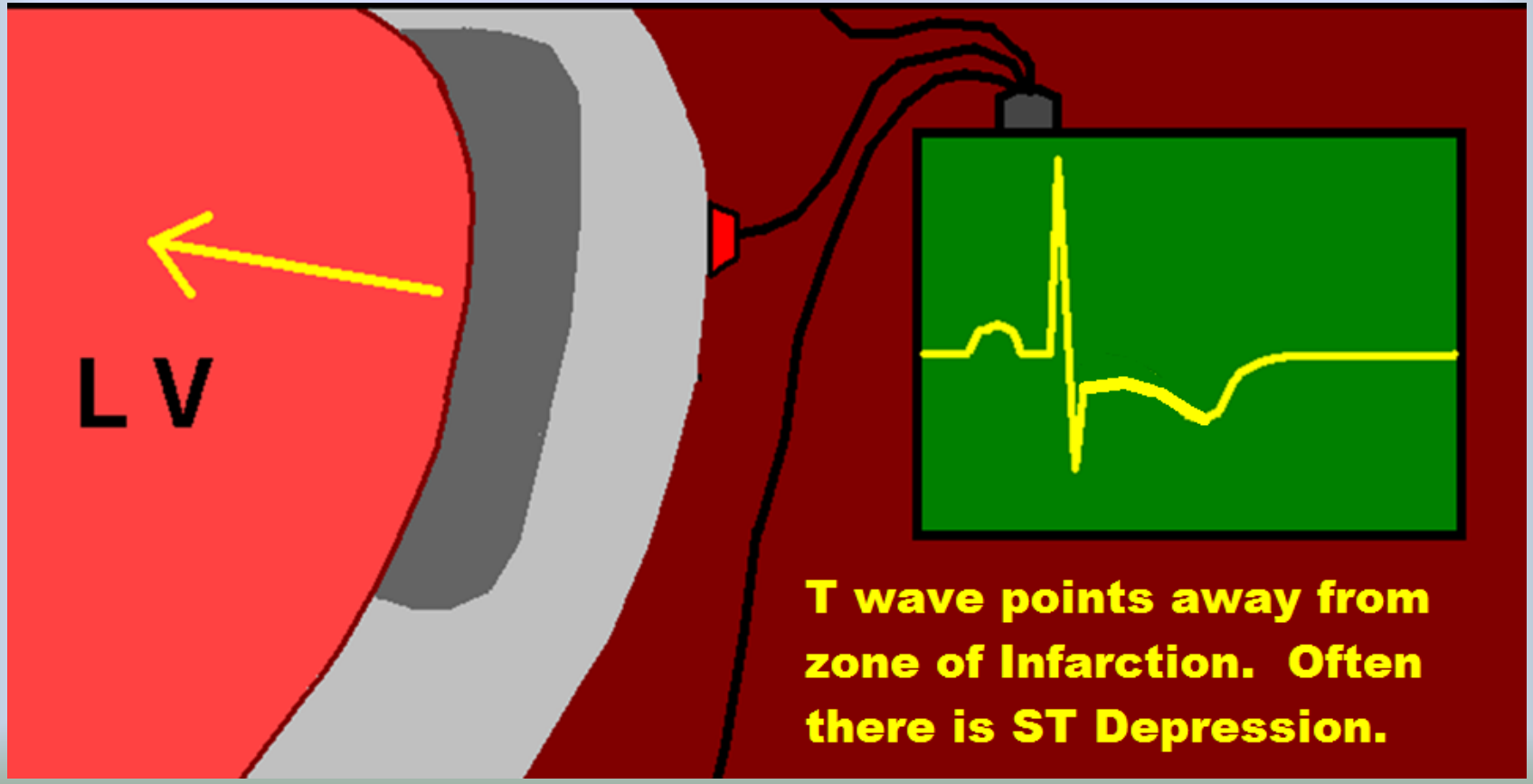


- Direct view of ISCHEMIA (anterior wall)
- Direct view of NSTEMI (anterior wall)

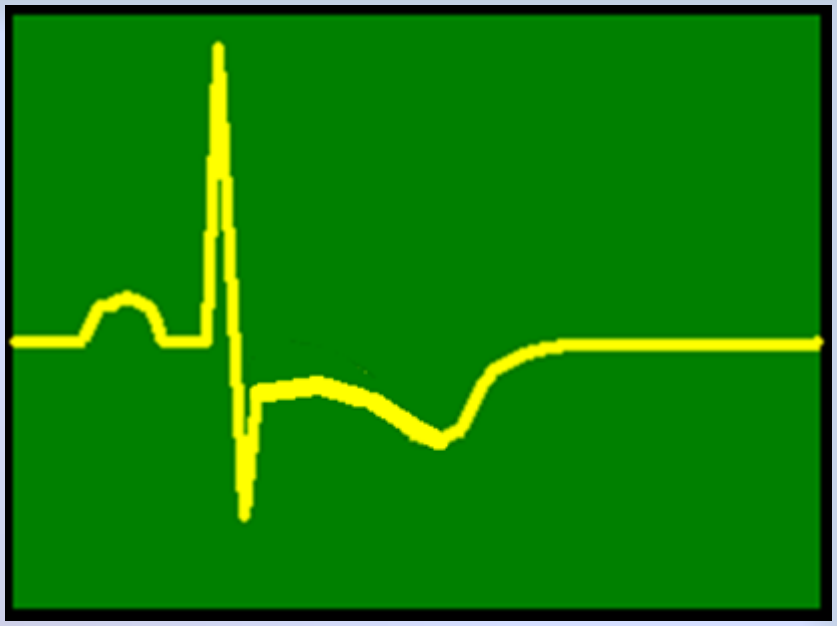
Non-STEMI (NSTEMI)

Non-ST Segment Elevation Myocardial Infarction.

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



ST Depression in Leads V1 – V4:

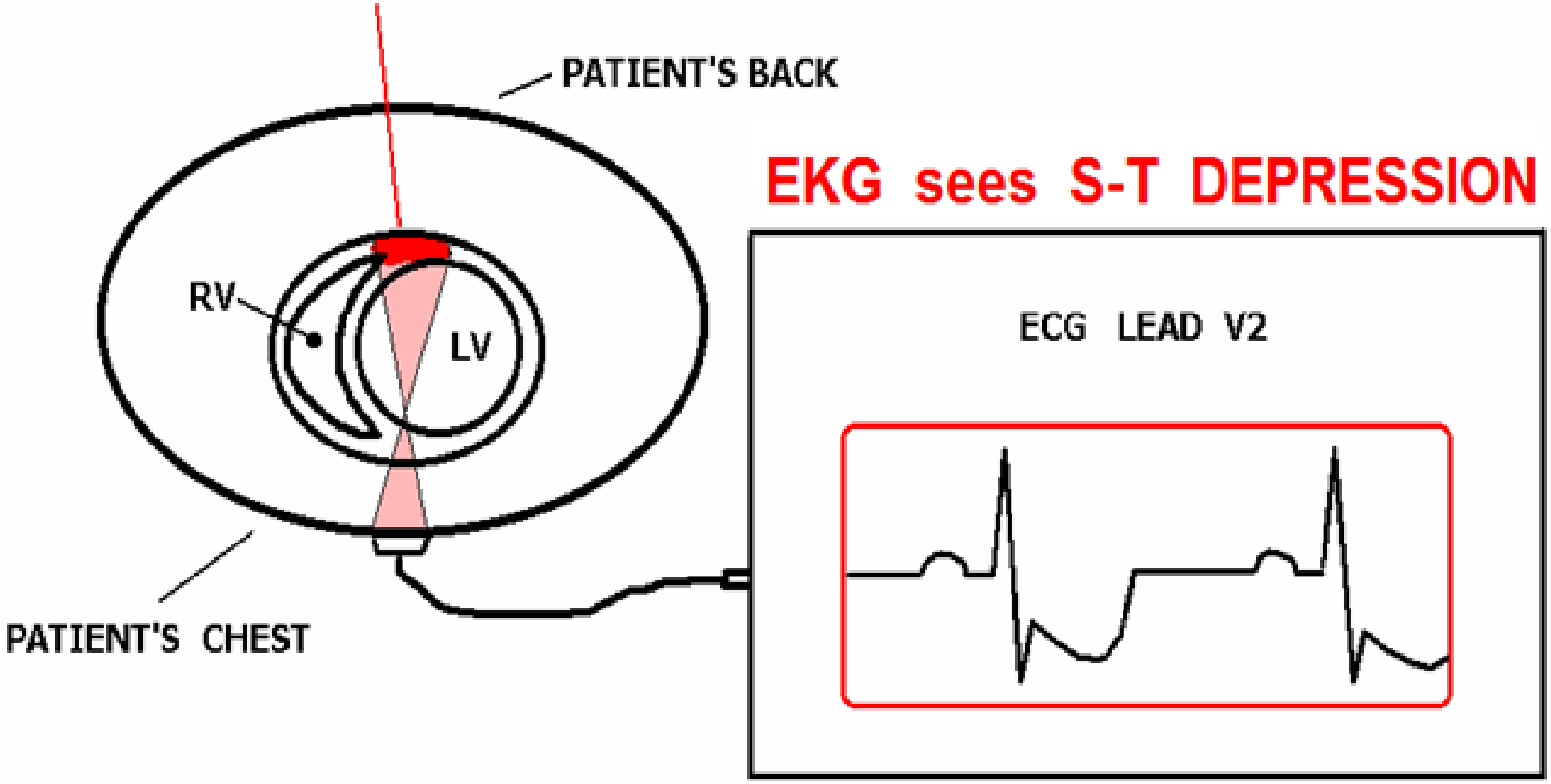


- Direct view of ISCHEMIA (anterior wall)
- Direct view of NSTEMI (anterior wall)
- Reciprocal view of STEMI (opposite side of heart - posterior wall)

HOW EKG VIEWS RECIPROCAL CHANGES

EXAMPLE:

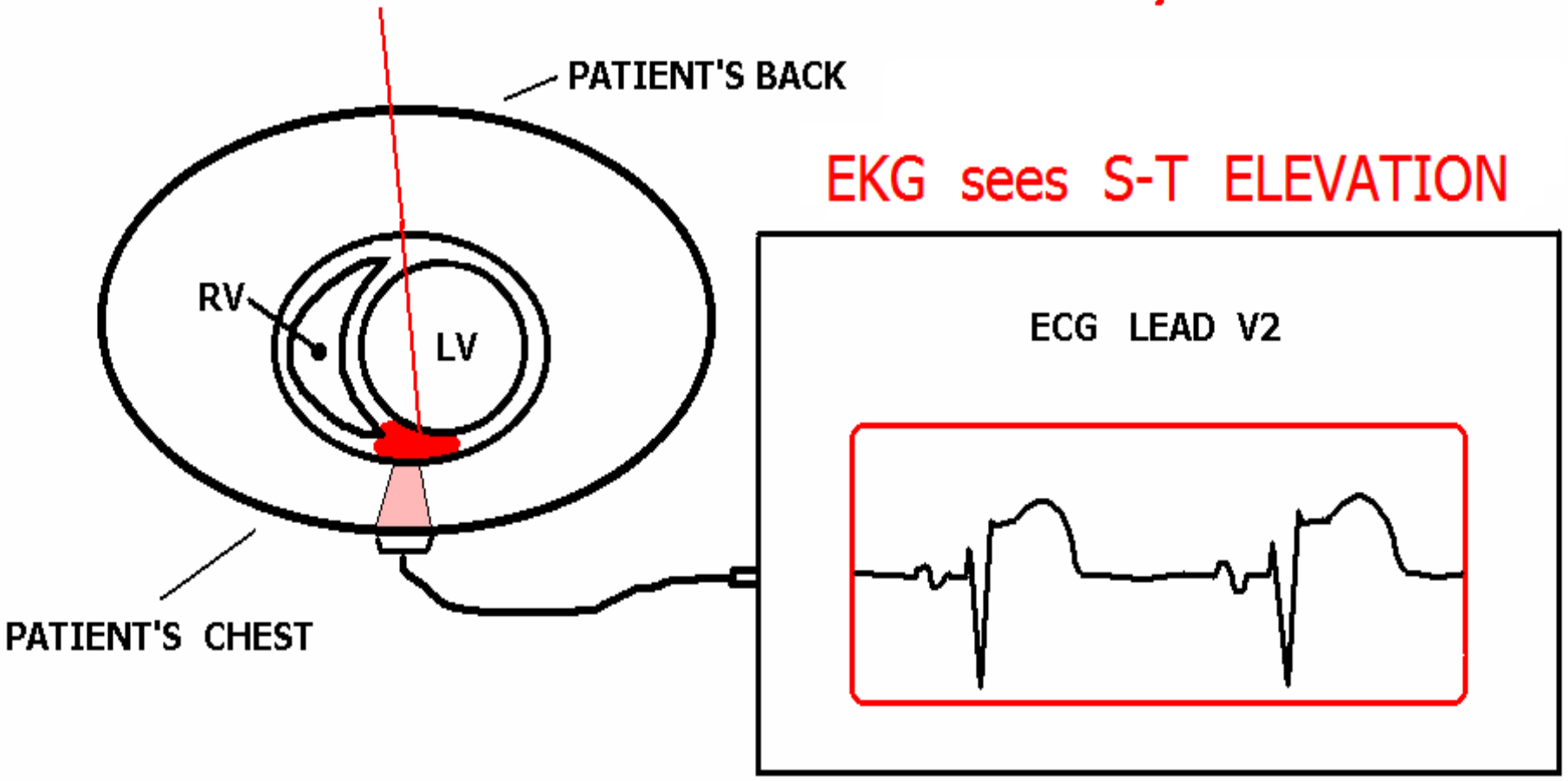
AREA OF ACUTE INFARCTION - POSTERIOR WALL



HOW EKG VIEWS INDICATIVE CHANGES

EXAMPLE:

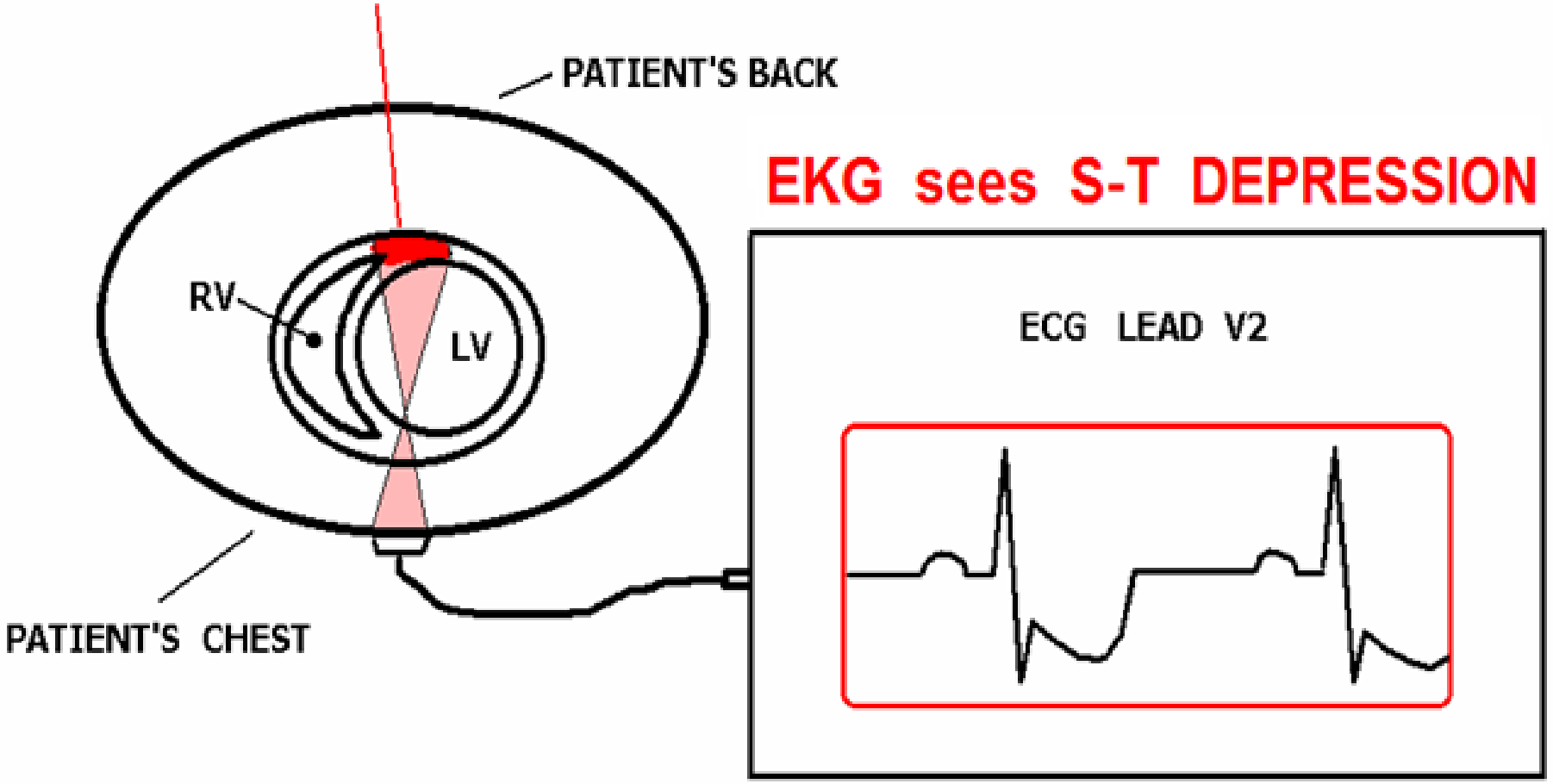
AREA OF ACUTE INFARCTION - ANTERIOR/SEPTAL



HOW EKG VIEWS RECIPROCAL CHANGES

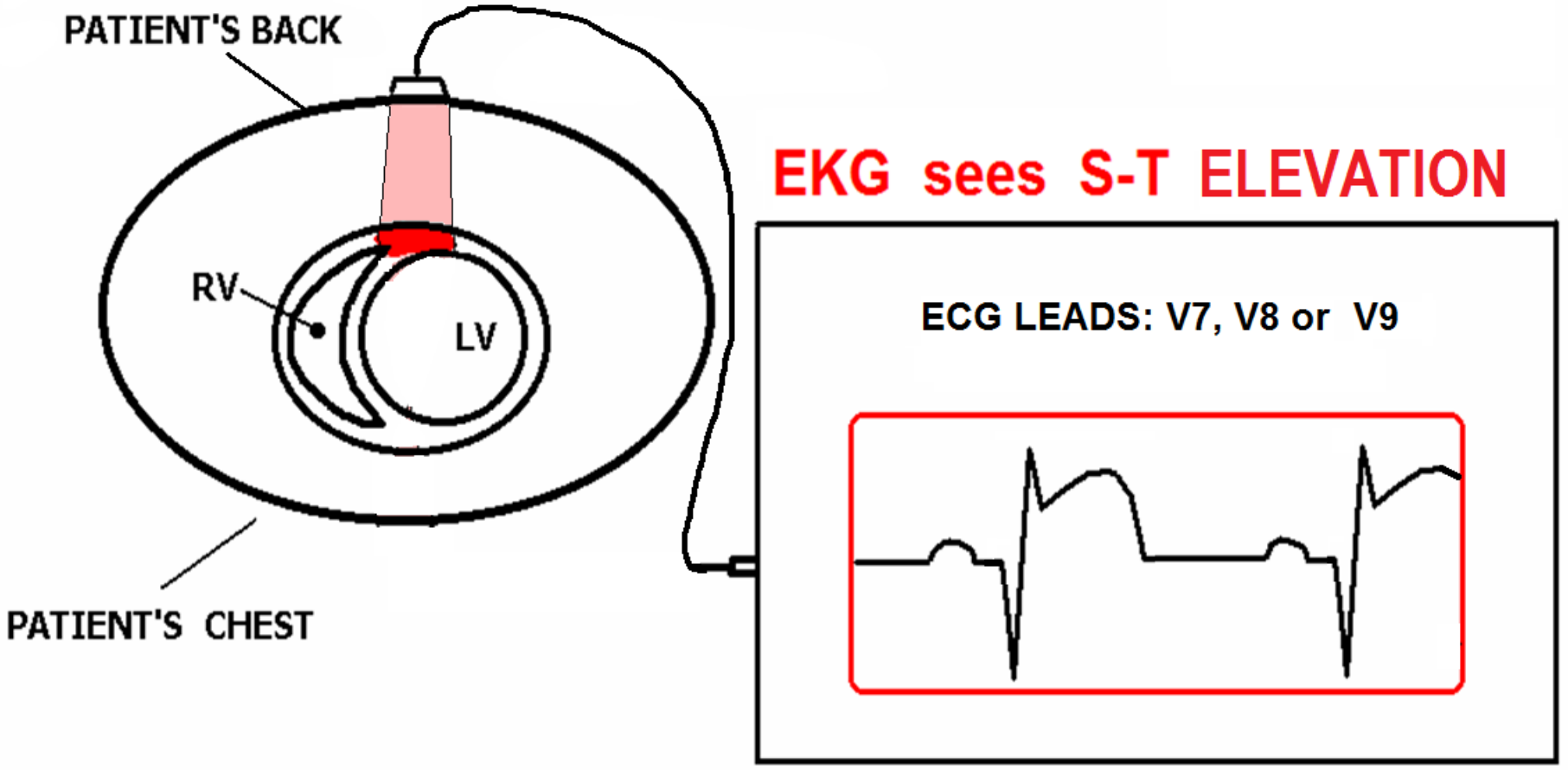
EXAMPLE:

AREA OF ACUTE INFARCTION - POSTERIOR WALL

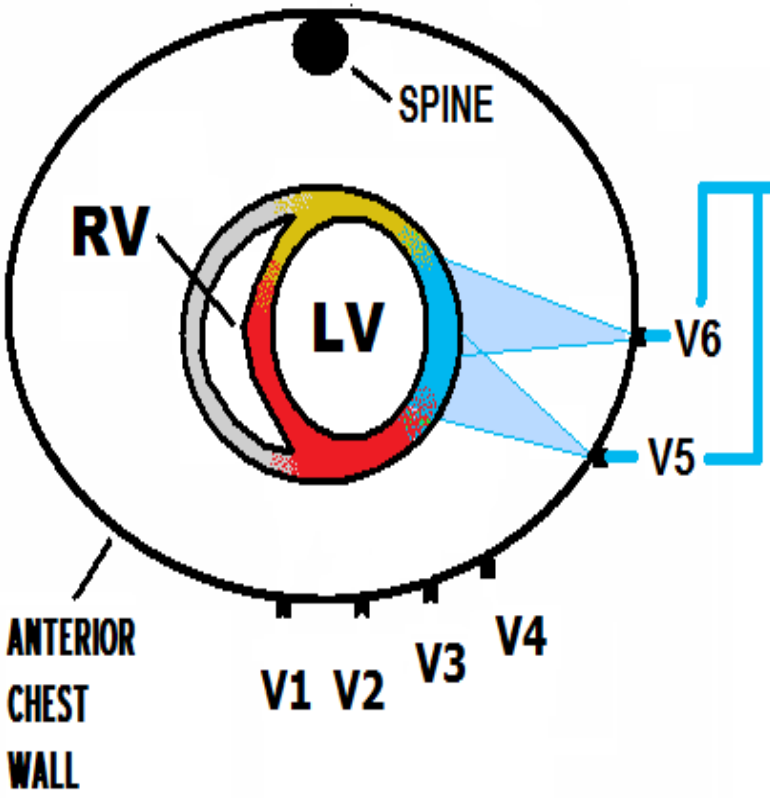


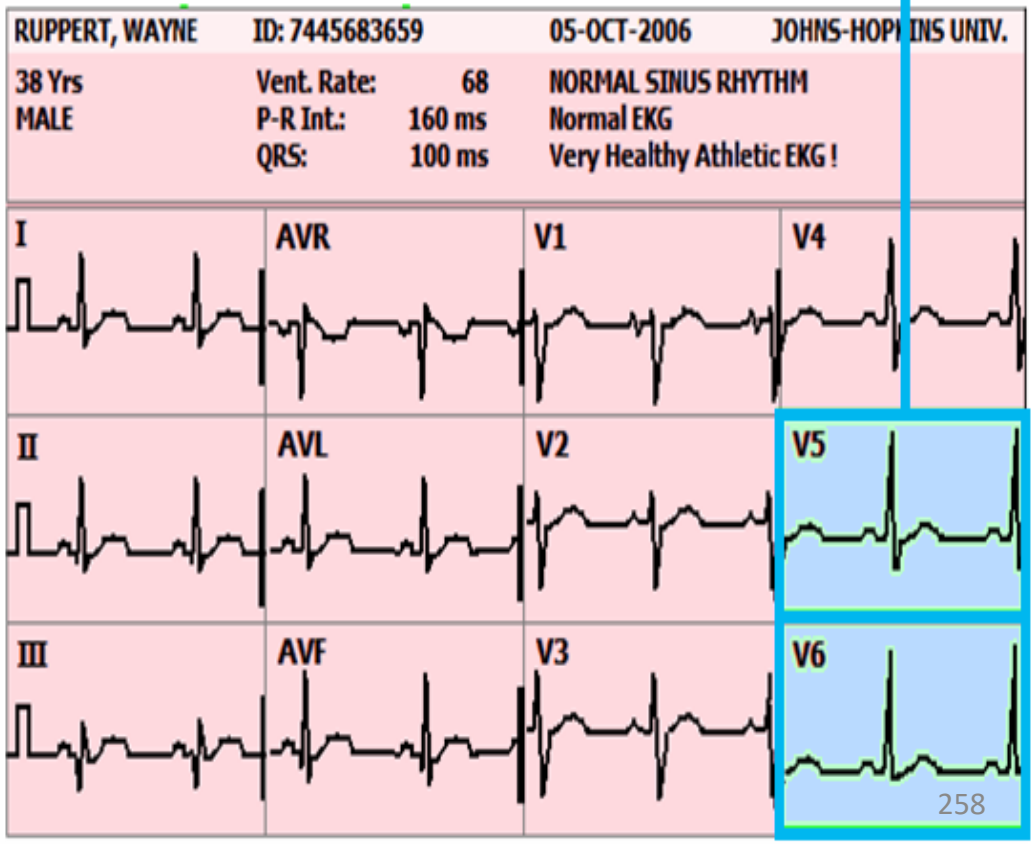
If we put ECG leads on the BACK of a PATIENT who is having an

ACUTE POSTERIOR WALL MI . . . . .

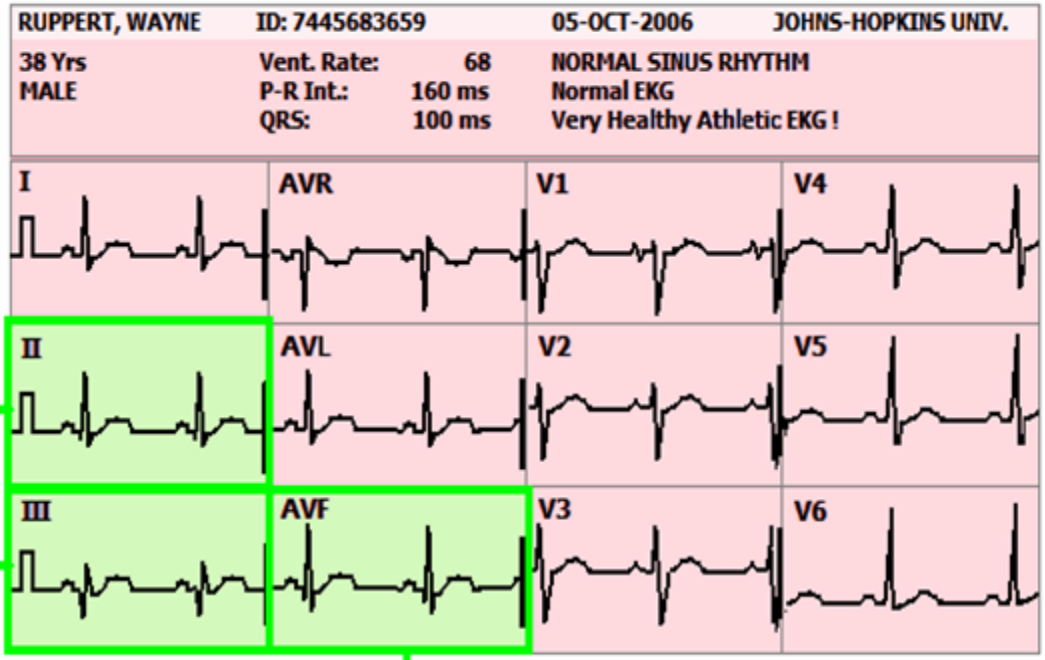


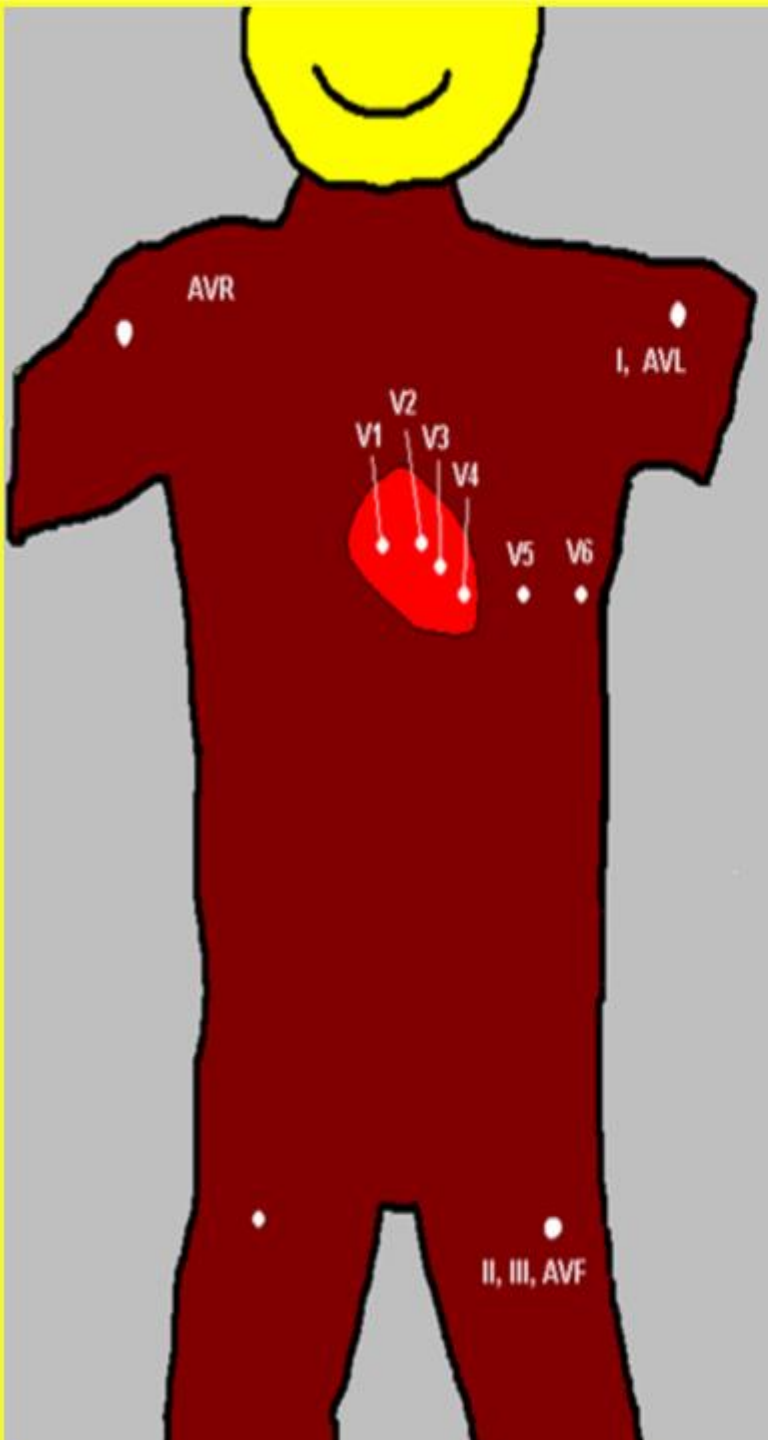
V5 - V6 VIEW THE LATERAL WALL
of the LEFT VENTRICLE





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





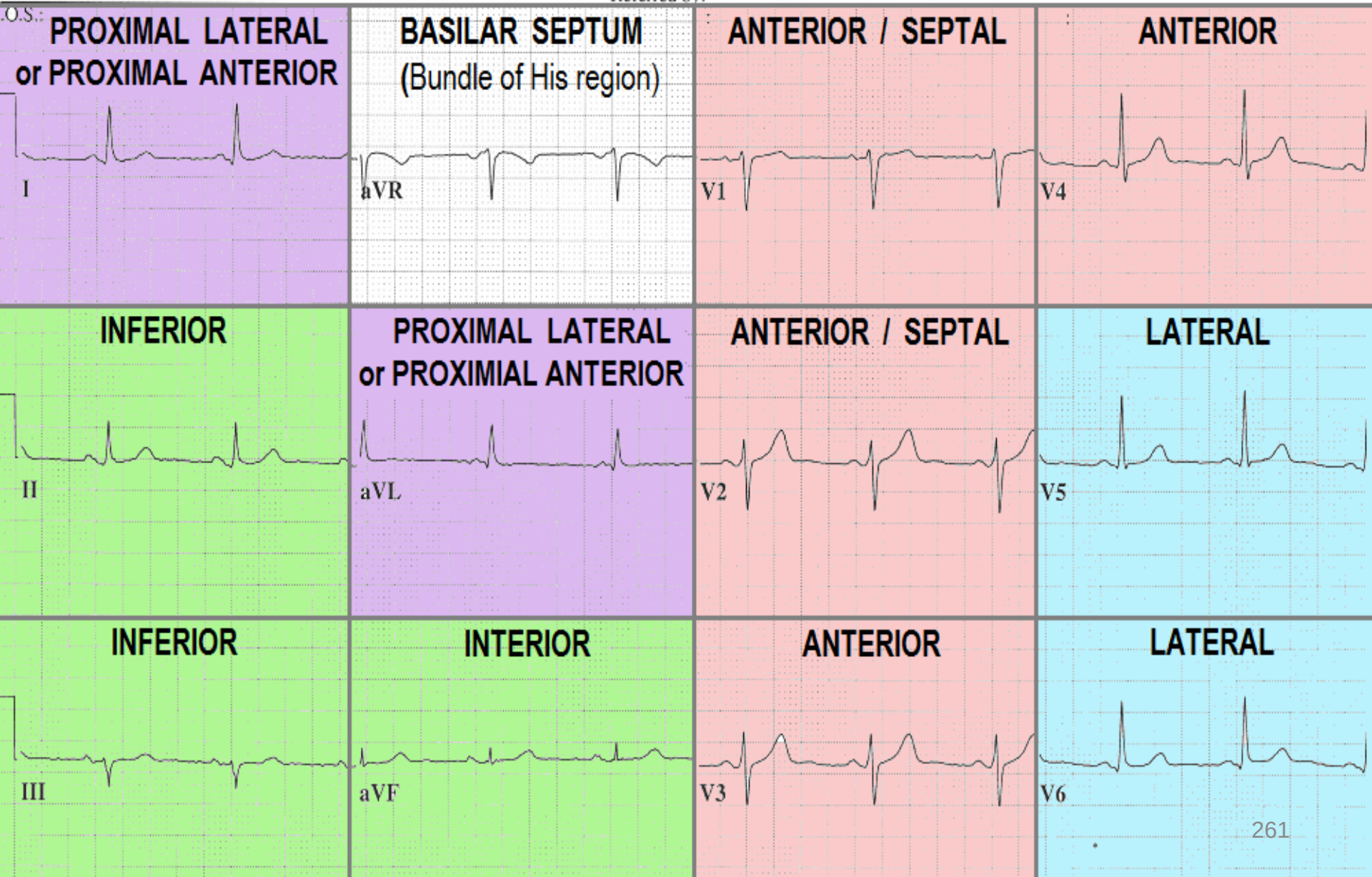
AREAS VIEWED by 12 LEAD ECG

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

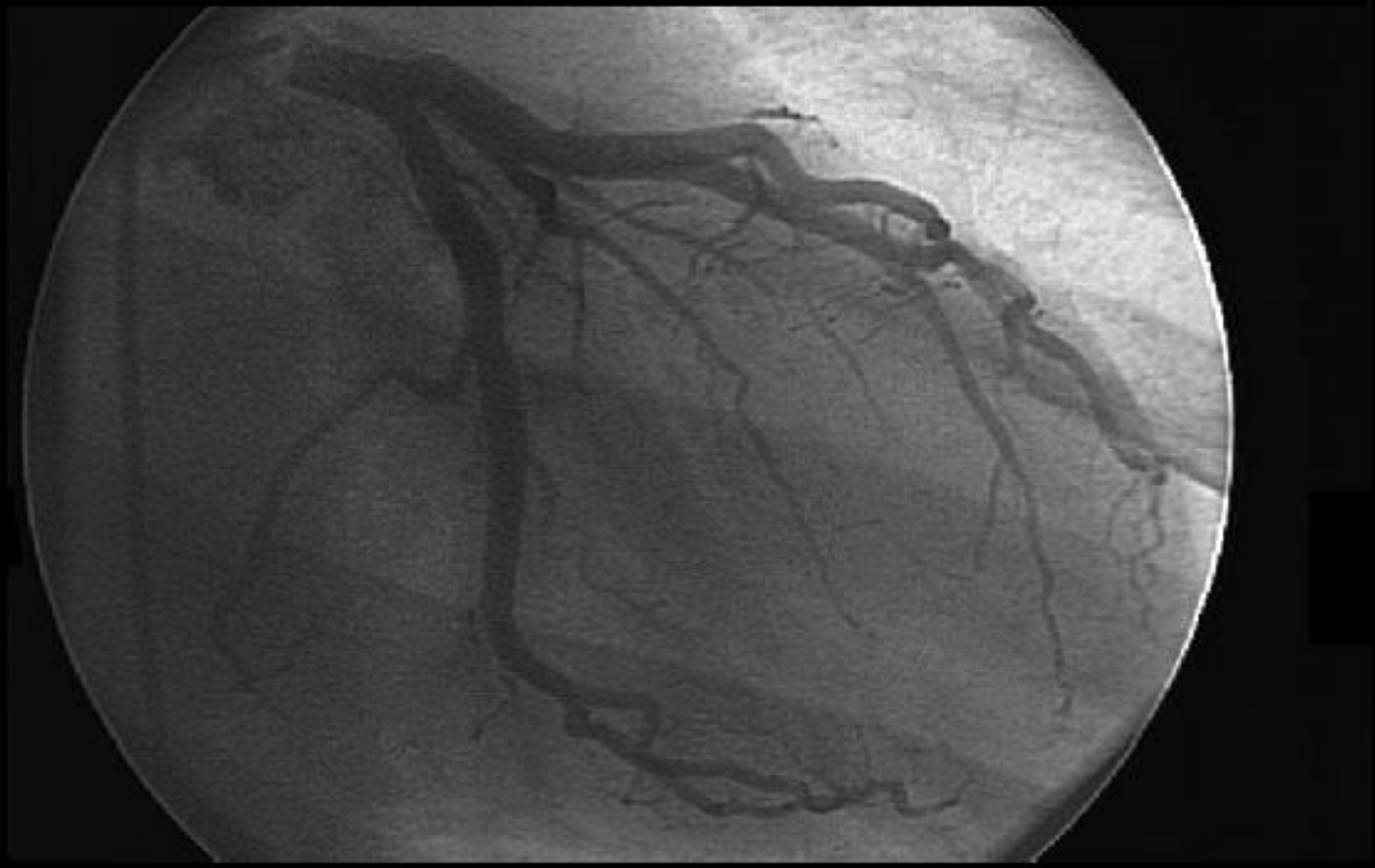
Vent. rate	64	BPM
PR interval	130	ms
QRS duration	96	ms
QT/QTc	396/408	ms
P–R–T axes	40 11	61

Normal sinus rhythm
Normal ECG
No previous ECGs available

Referred by:

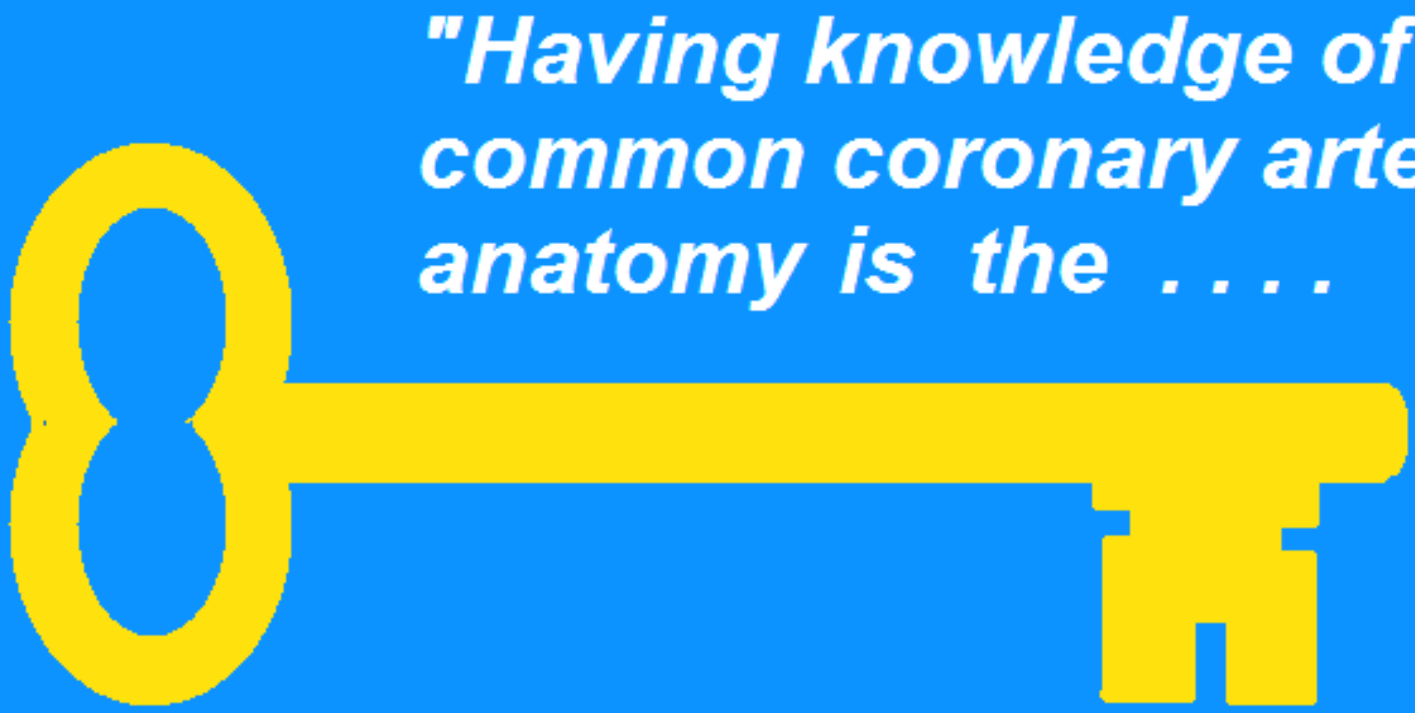


THE CORONARY



ARTERIES

STRUCTURES SERVED BY THE CORONARY ARTERIES



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

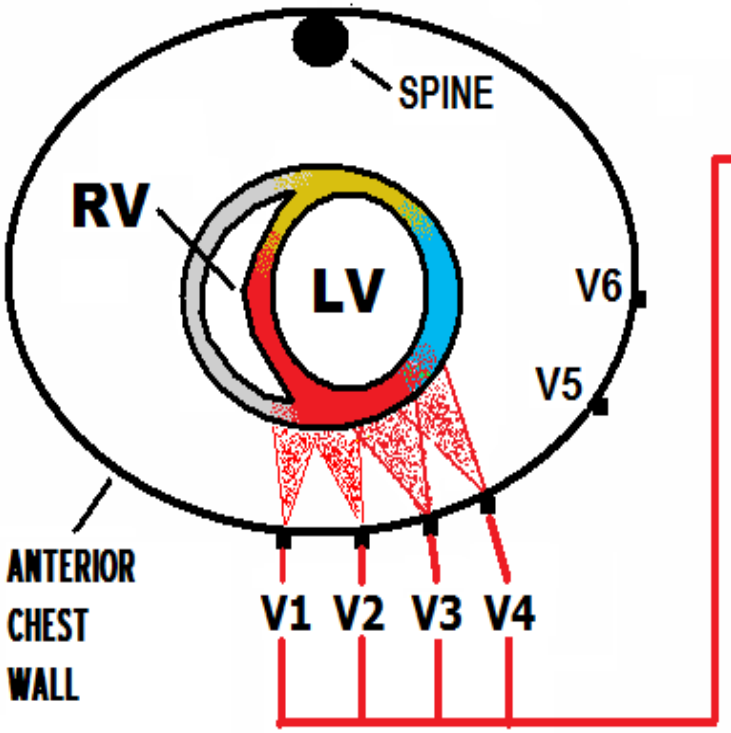
The 12 Lead ECG becomes your "crystal ball !!"

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 !***

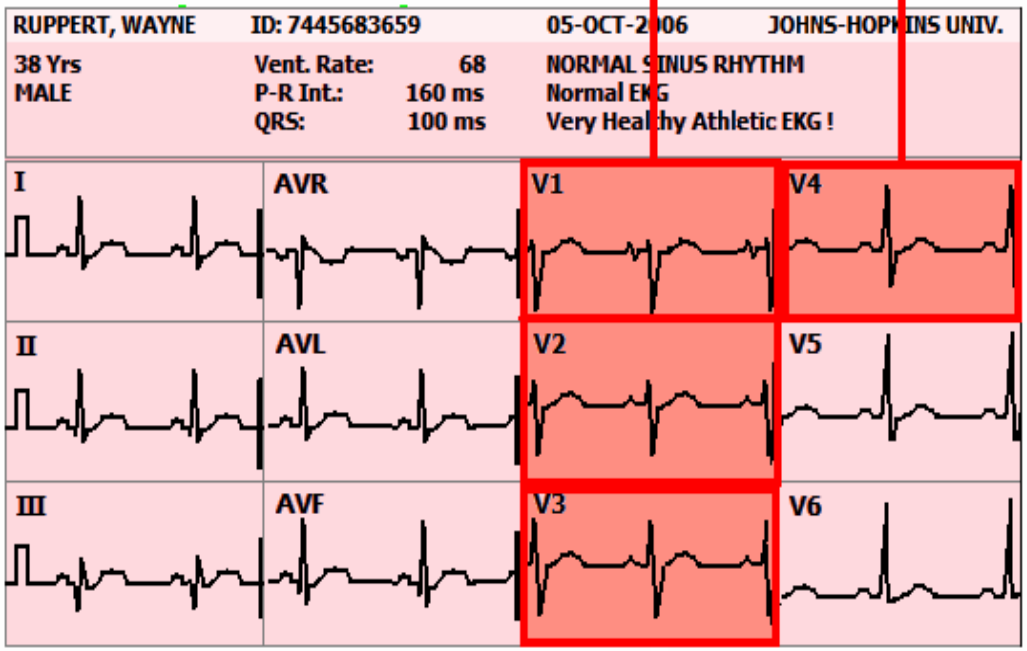
V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL

of the LEFT VENTRICLE



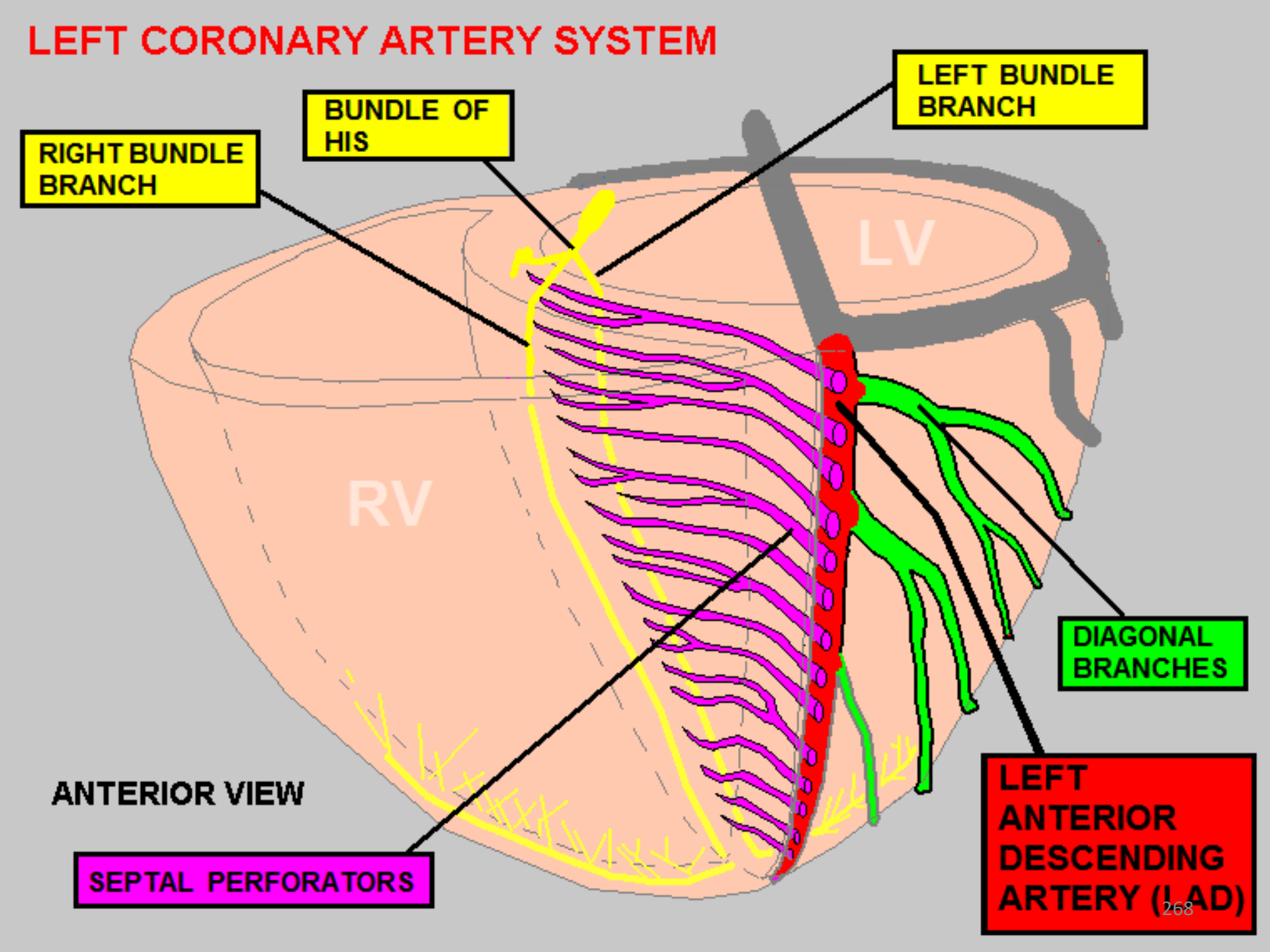
V1, V2 - ANTERIOR / SEPTAL

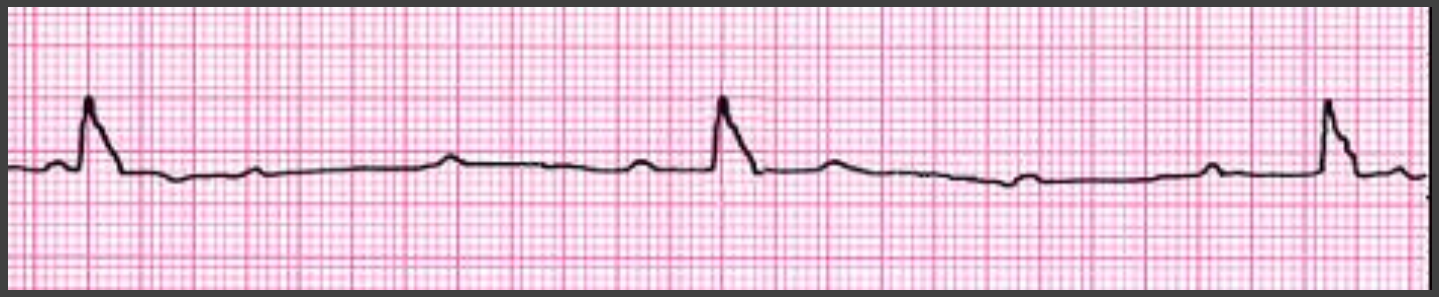
V3, V4 - ANTERIOR

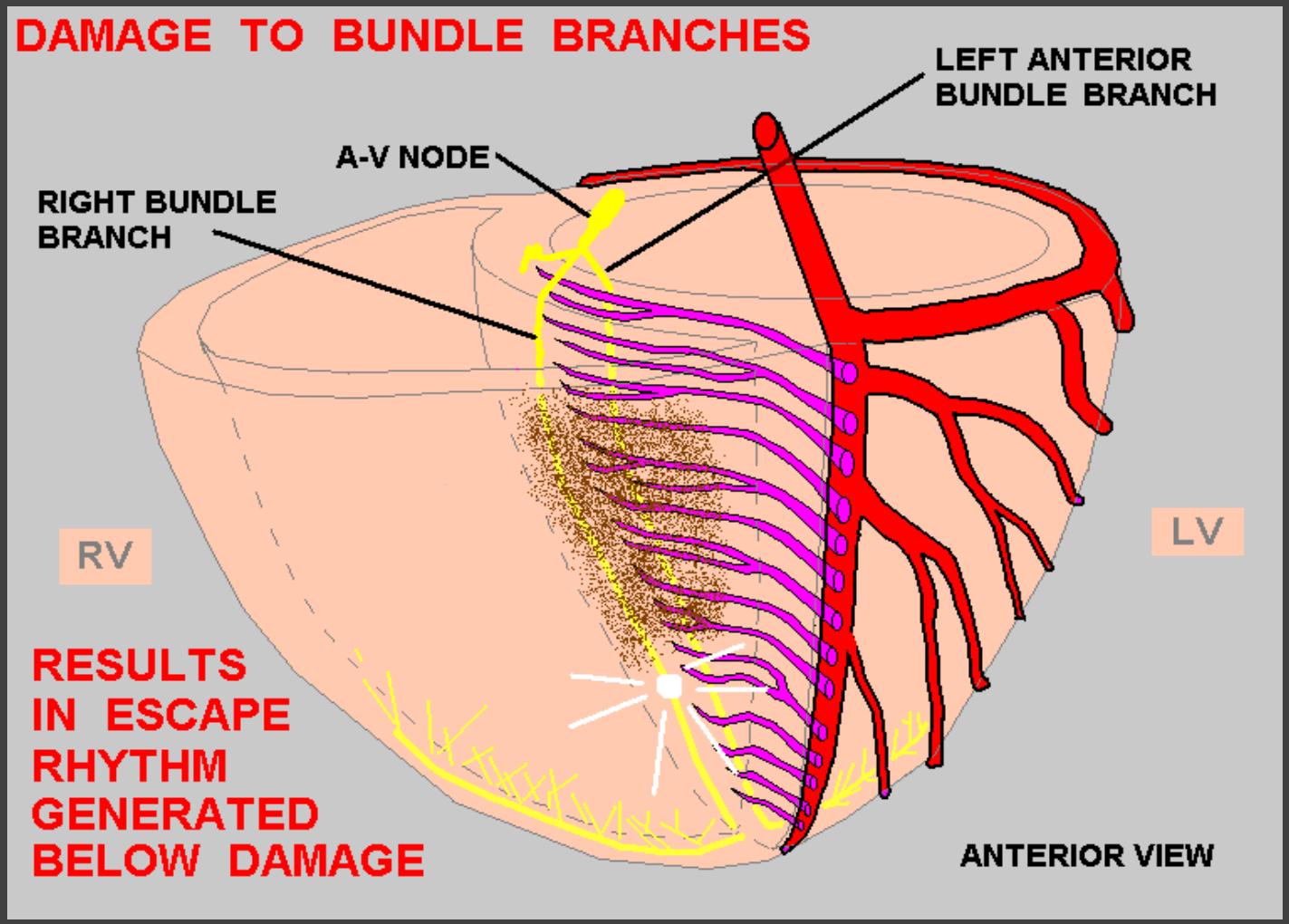


Which Coronary Artery typically Supplies the ANTERIOR WALL ?

LEFT CORONARY ARTERY SYSTEM



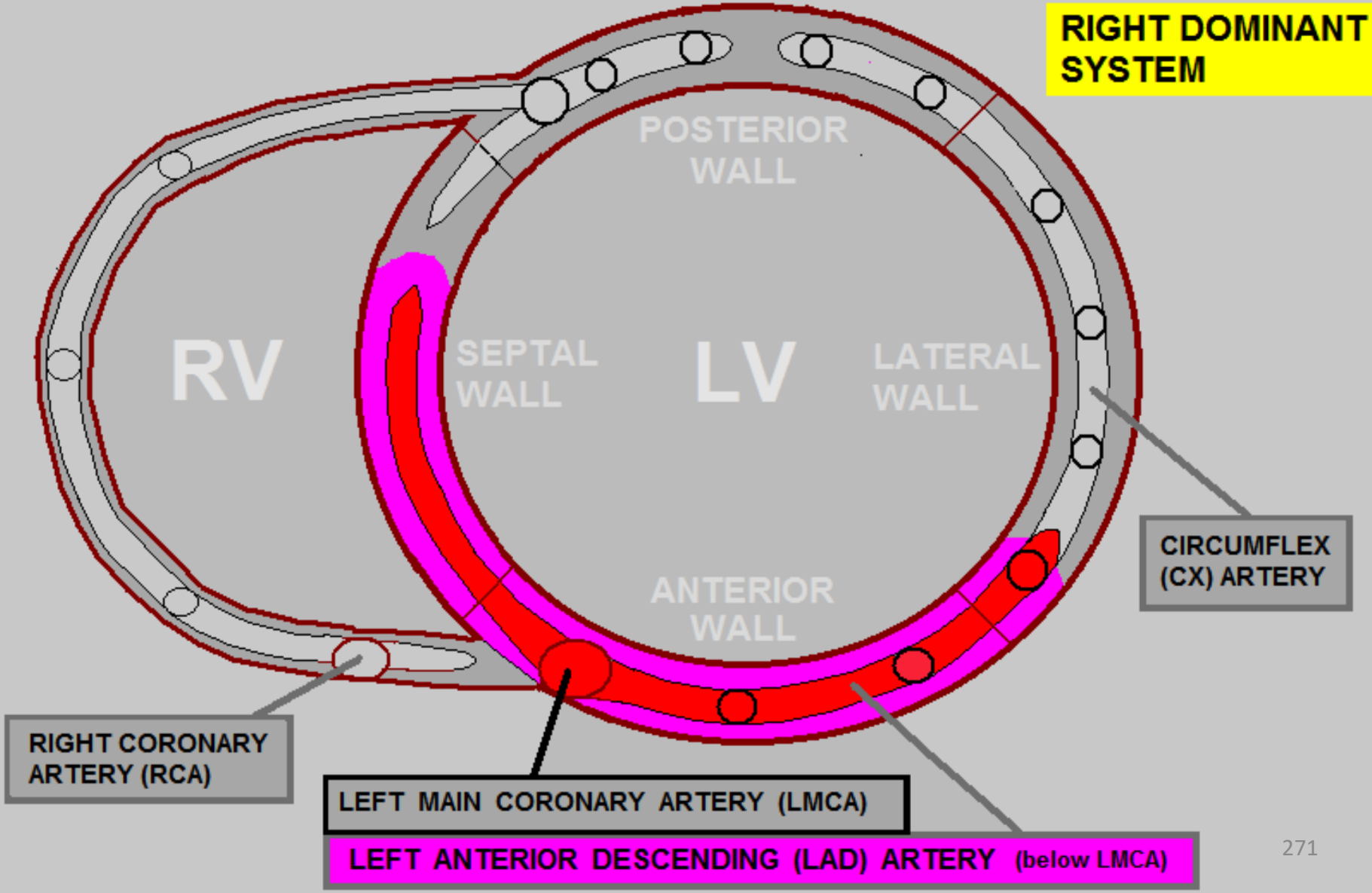



DAMAGE TO BUNDLE BRANCHES
LEFT ANTERIOR BUNDLE BRANCH
A-V NODE
RIGHT BUNDLE BRANCH
RV
LV
RESULTS IN ESCAPE RHYTHM GENERATED BELOW DAMAGE
ANTERIOR VIEW

cutaway view of the

LEFT ANTERIOR DESCENDING ARTERY (LAD)

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



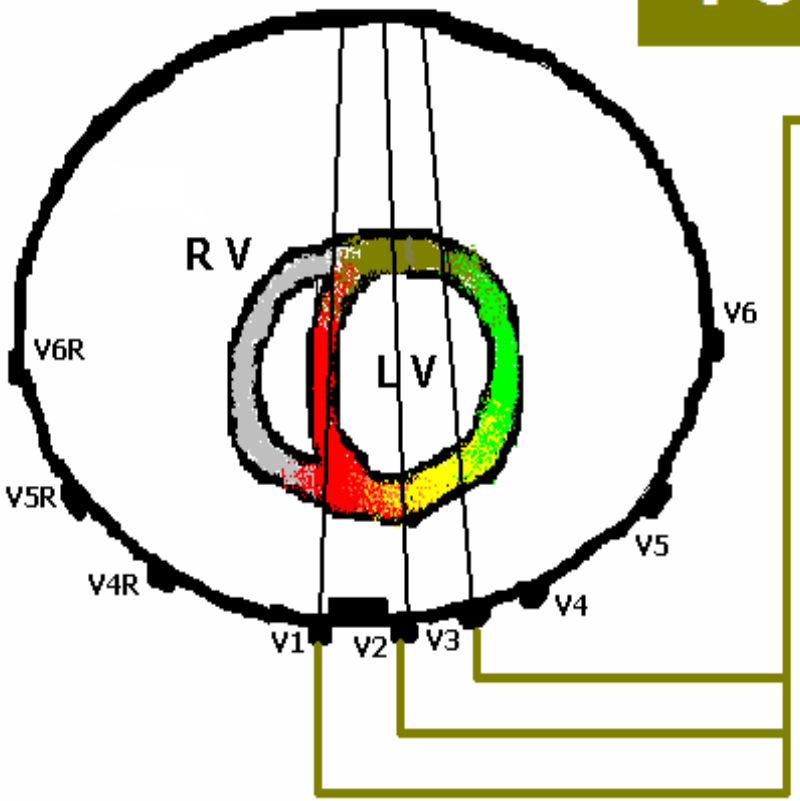
HELPFUL HINT . . . MEMORIZE THIS !

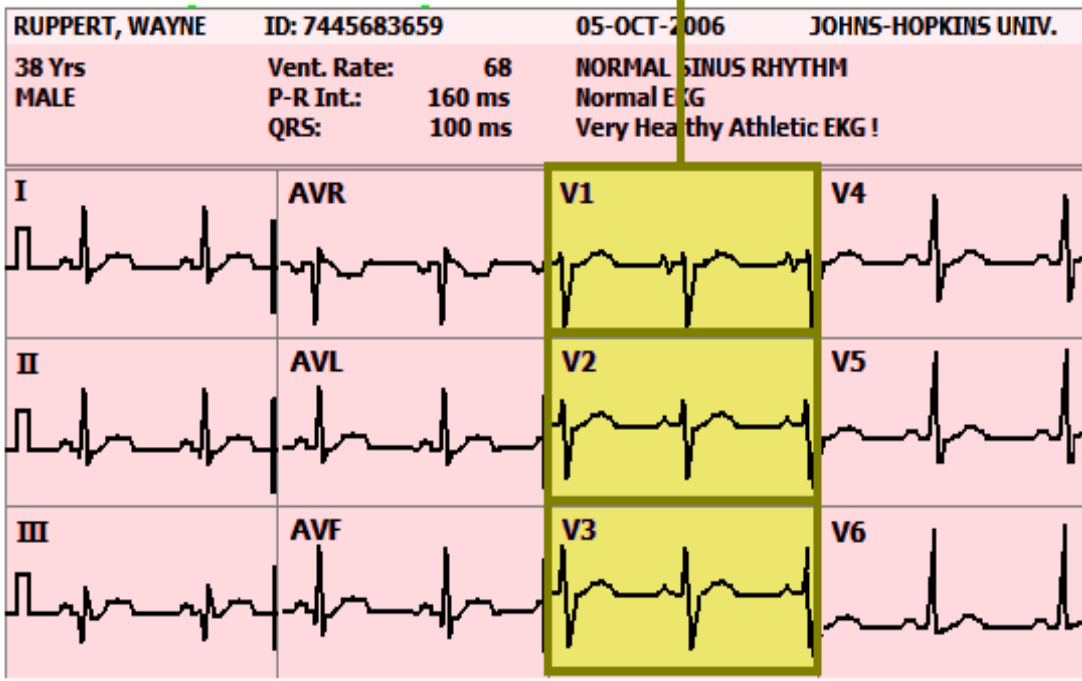
LEFT ANTERIOR DESCENDING ARTERY (LAD)

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

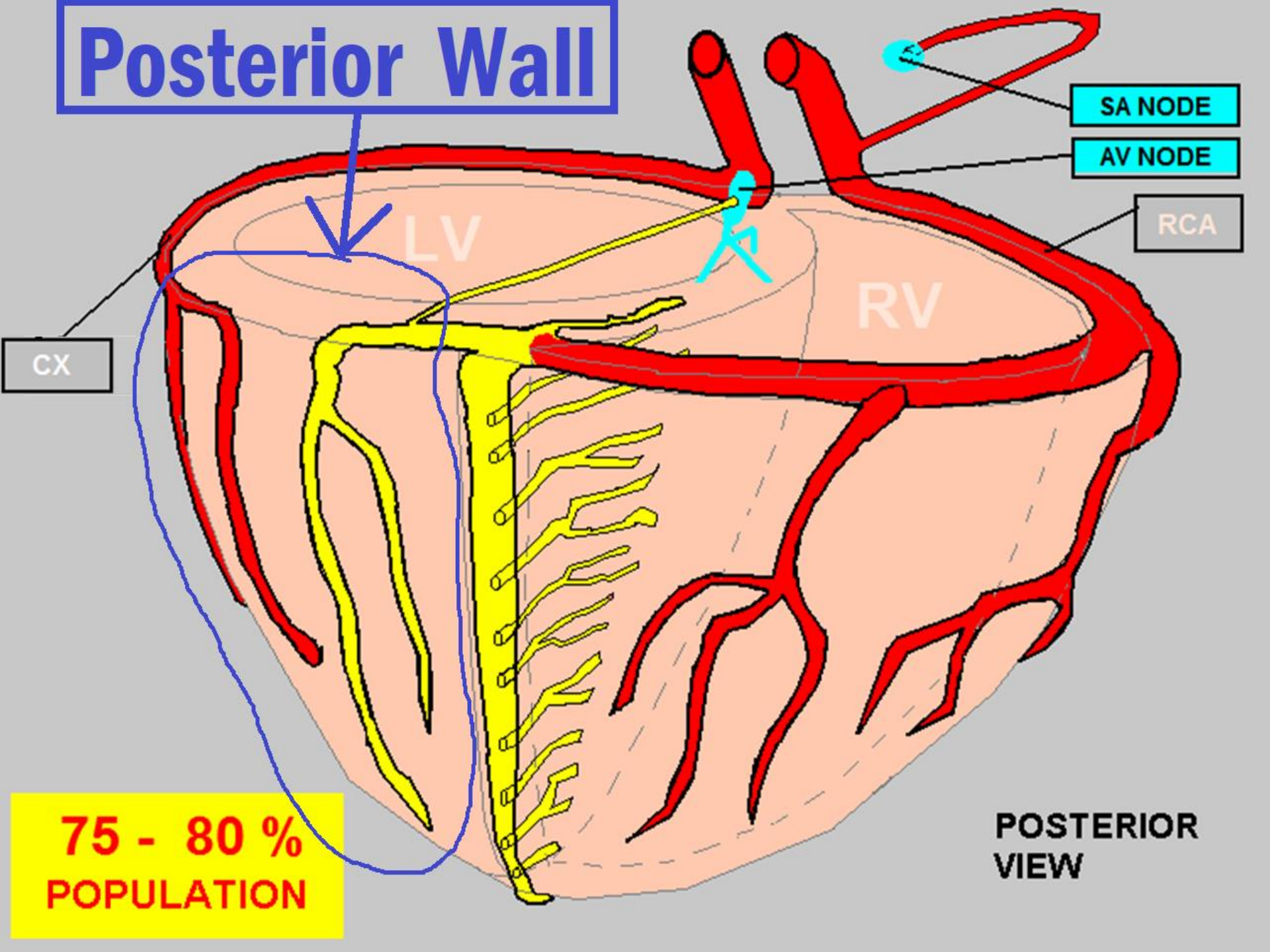
LEADS V1 - V3 *view the*

POSTERIOR WALL

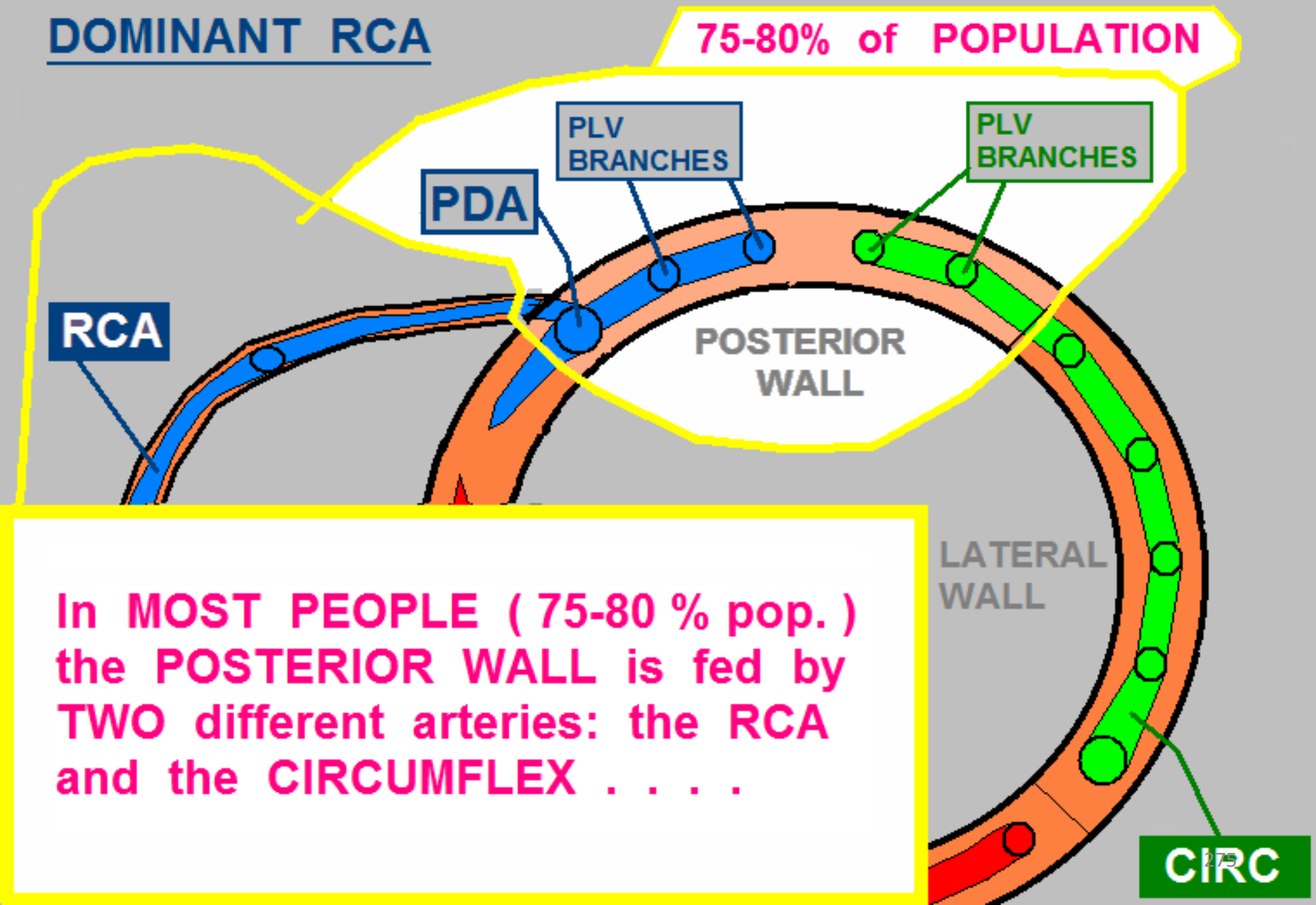




via RECIPROCAL CHANGES.

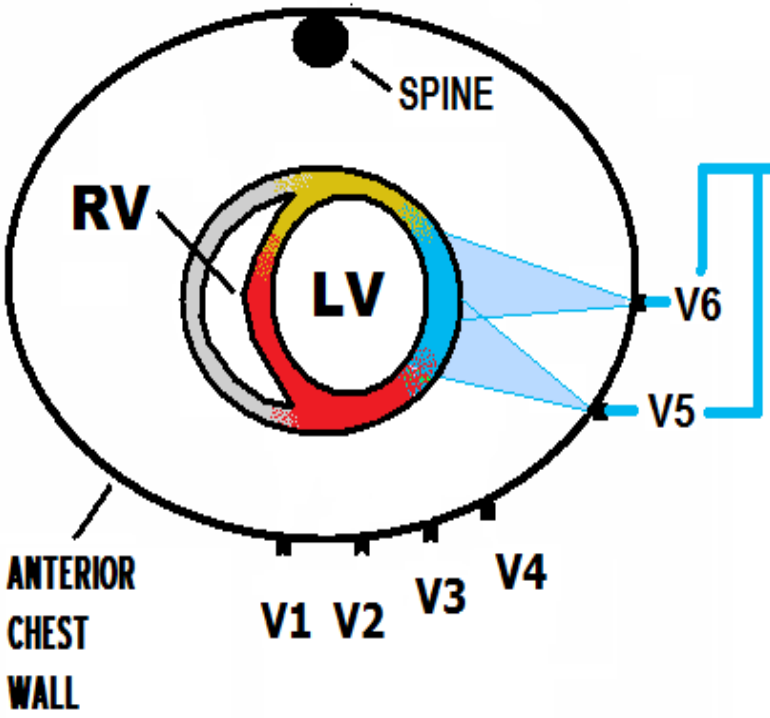

Posterior Wall
SA NODE
AV NODE
RCA
LV
RV
CX
75 - 80 %
POPULATION
POSTERIOR
VIEW

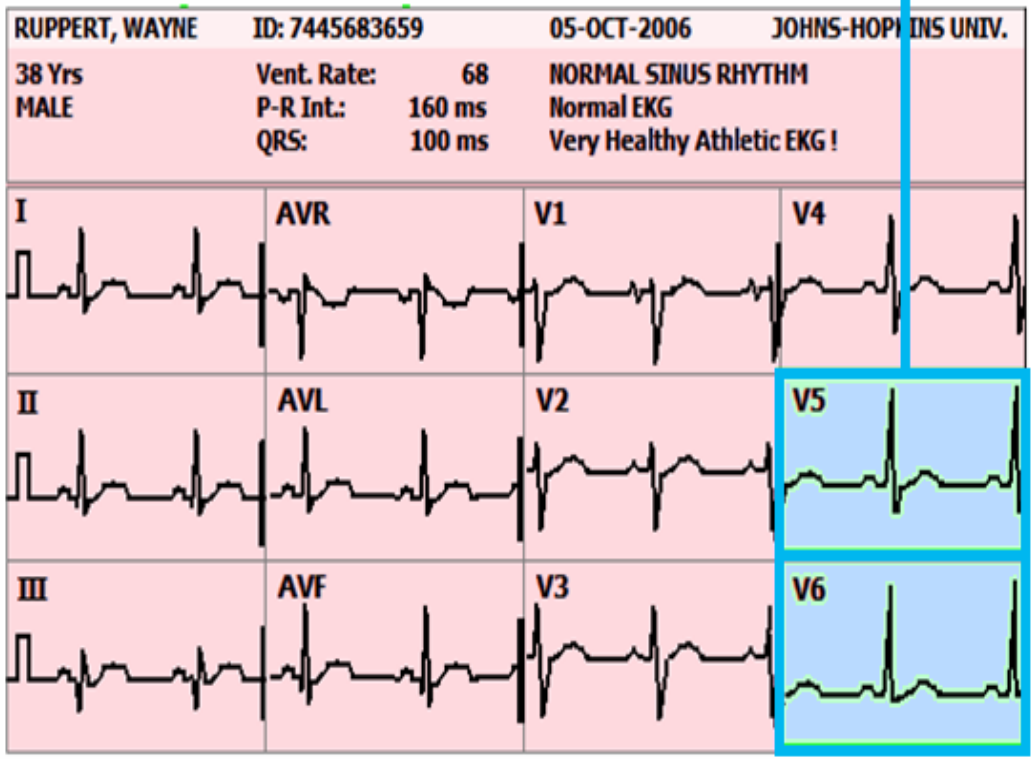
POSTERIOR WALL BLOOD SUPPLY



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

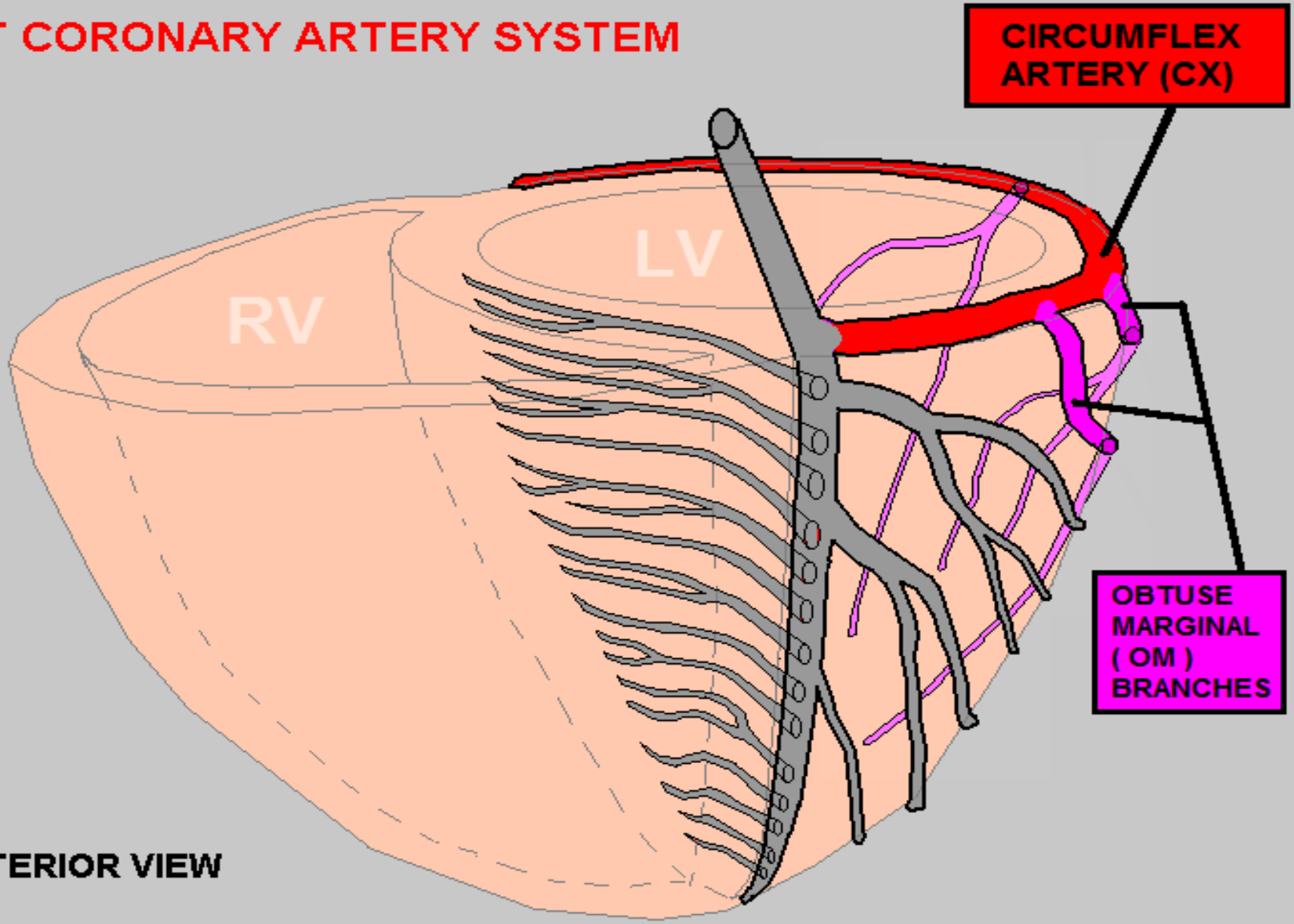
V5 - V6 VIEW THE LATERAL WALL
of the LEFT VENTRICLE





Which Coronary Artery typically Supplies the LATERAL WALL ?

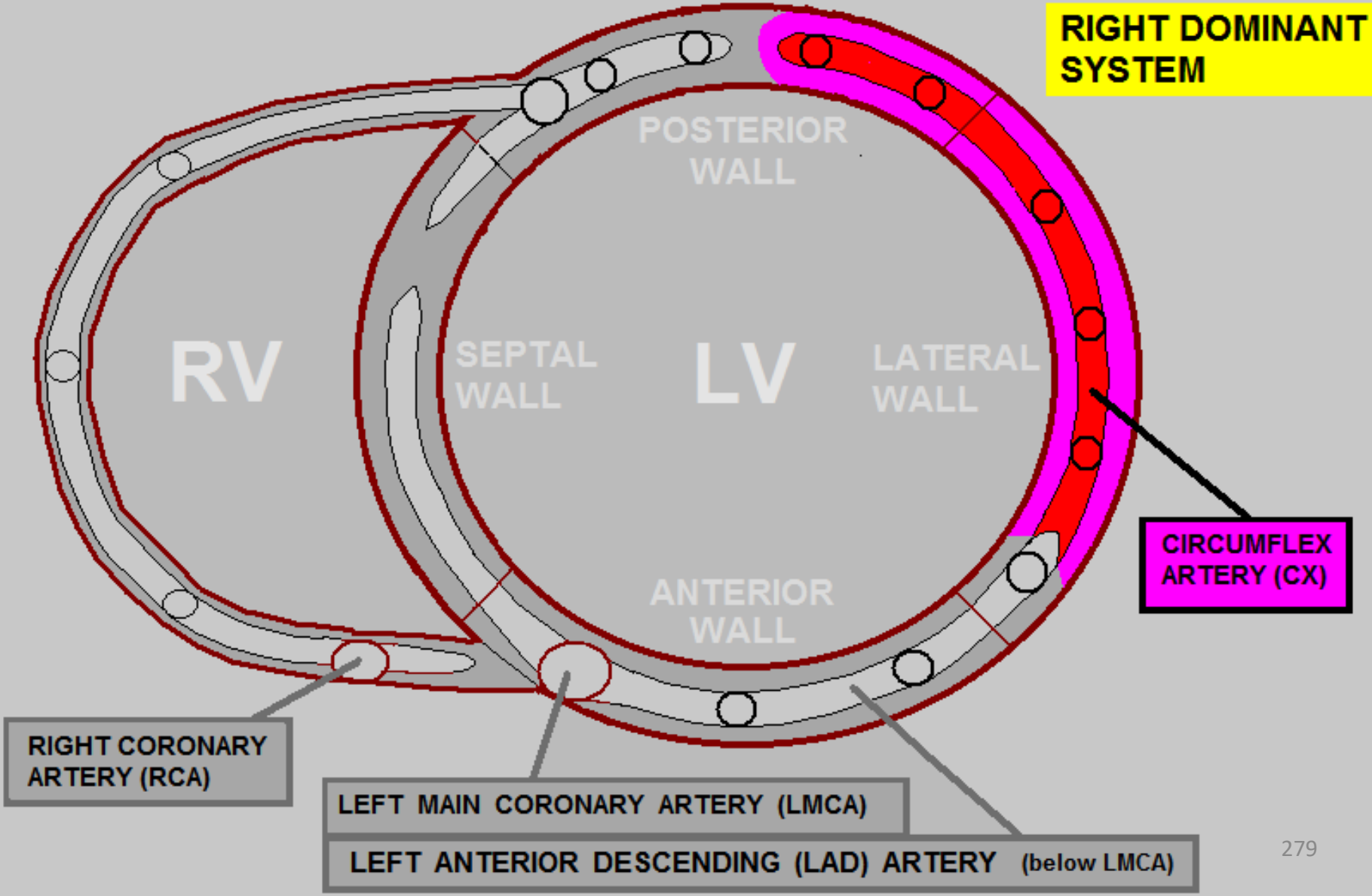
LEFT CORONARY ARTERY SYSTEM



cutaway view of the

CIRCUMFLEX ARTERY (CX) DISTRIBUTION

SUPPLIES 20 - 30 % of the LV MUSCLE MASS



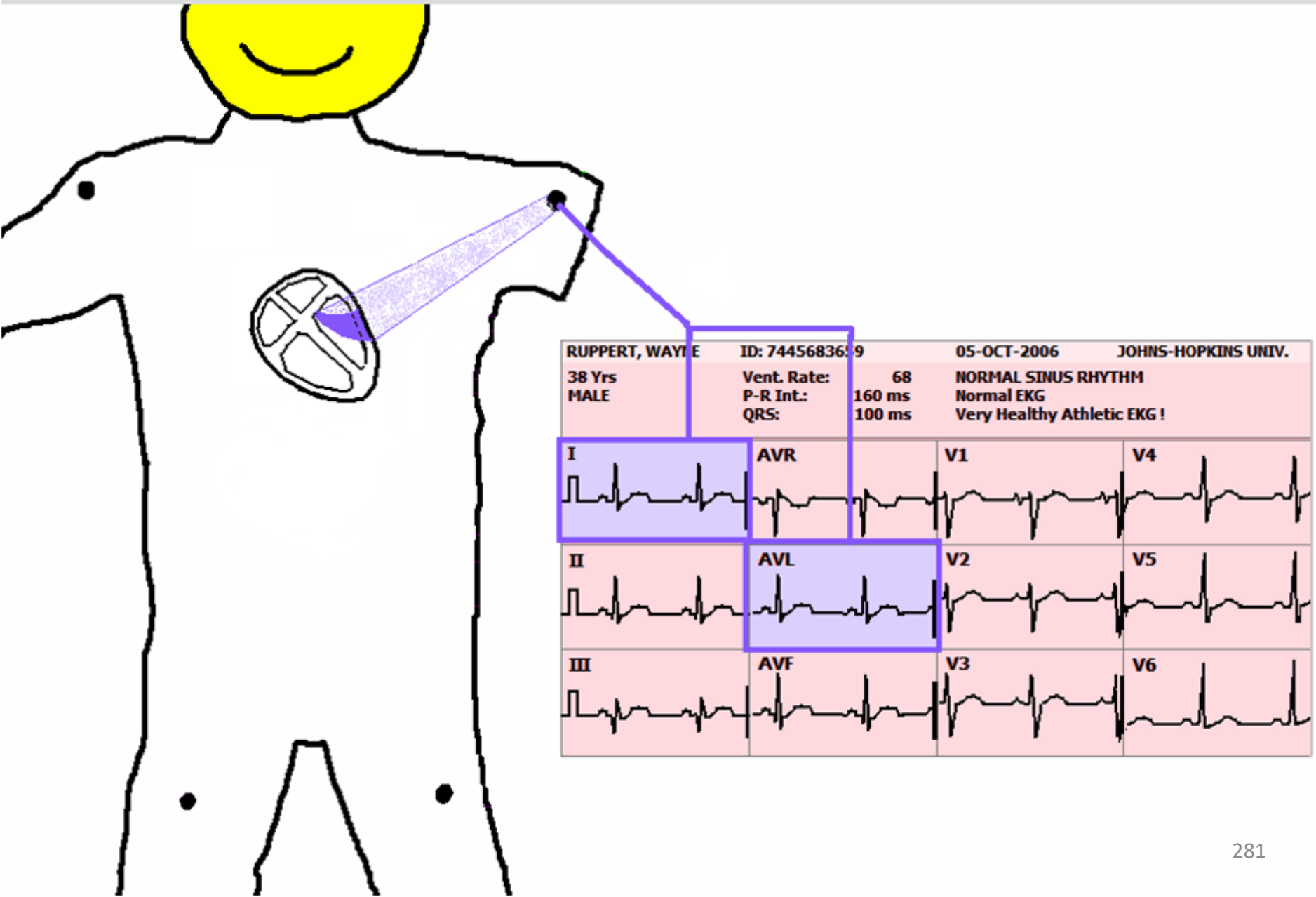
HELPFUL HINT . . . MEMORIZE THIS !

CIRCUMFLEX ARTERY (CX)

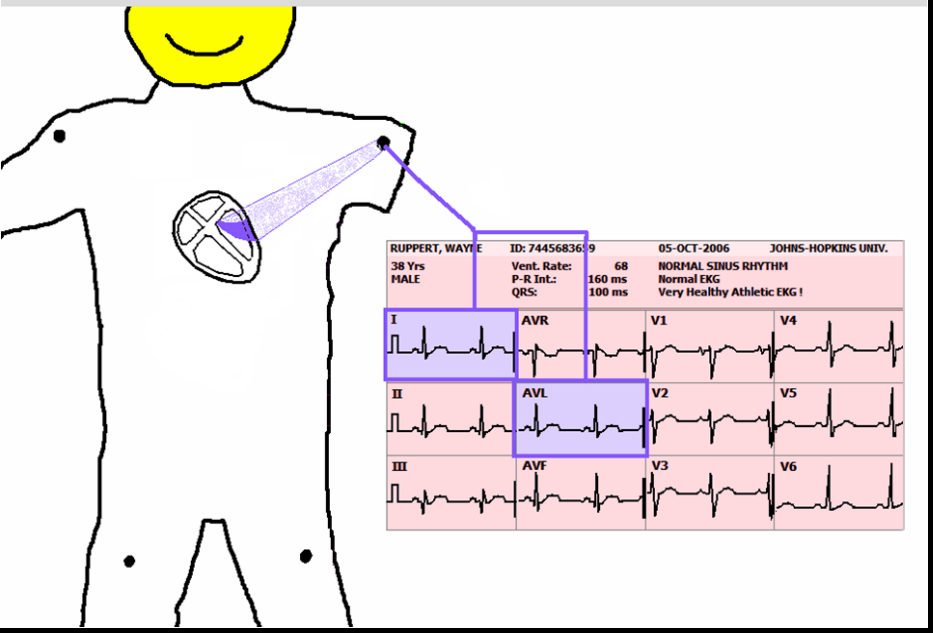
RIGHT DOMINANT SYSTEMS

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

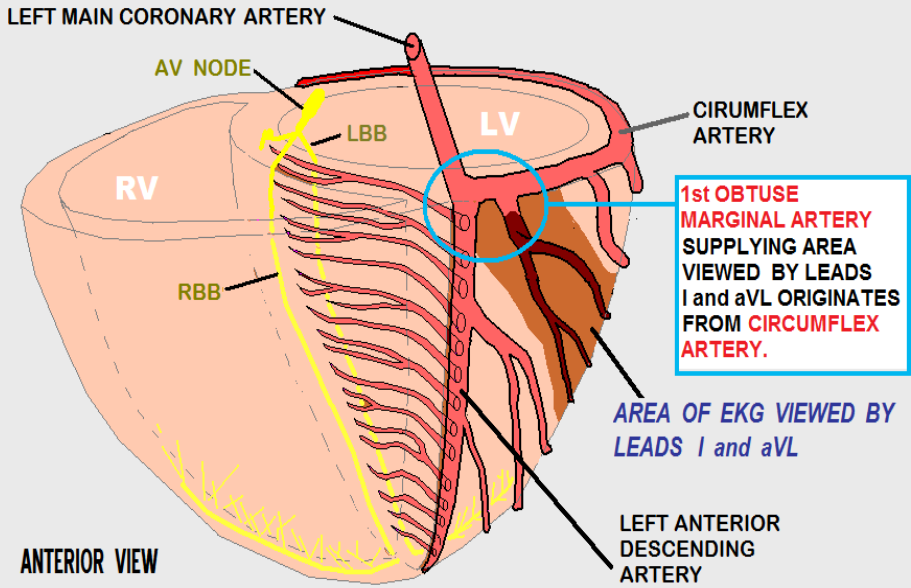
LEADS I and aVL VIEW the LATERAL - ANTERIOR WALL



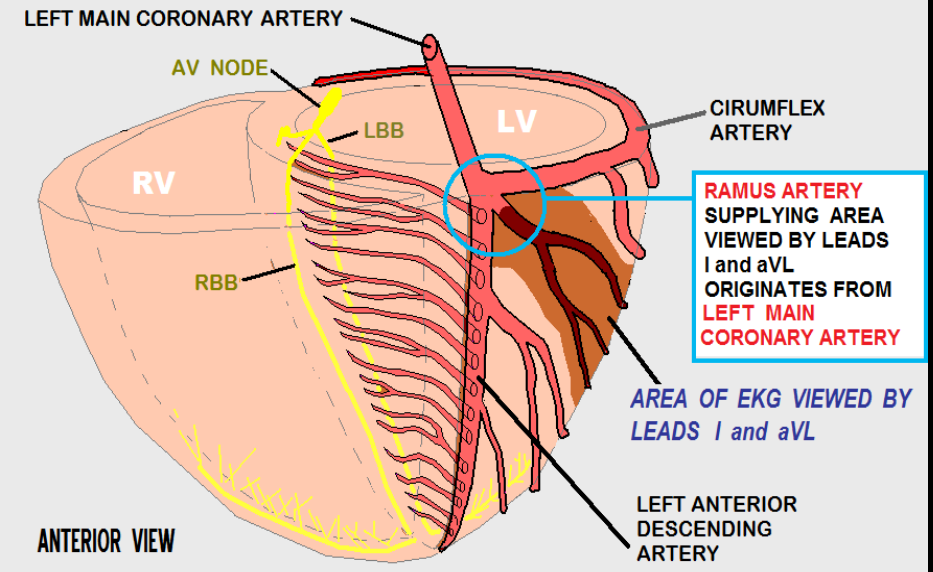
LEADS I and aVL VIEW the LATERAL - ANTERIOR WALL



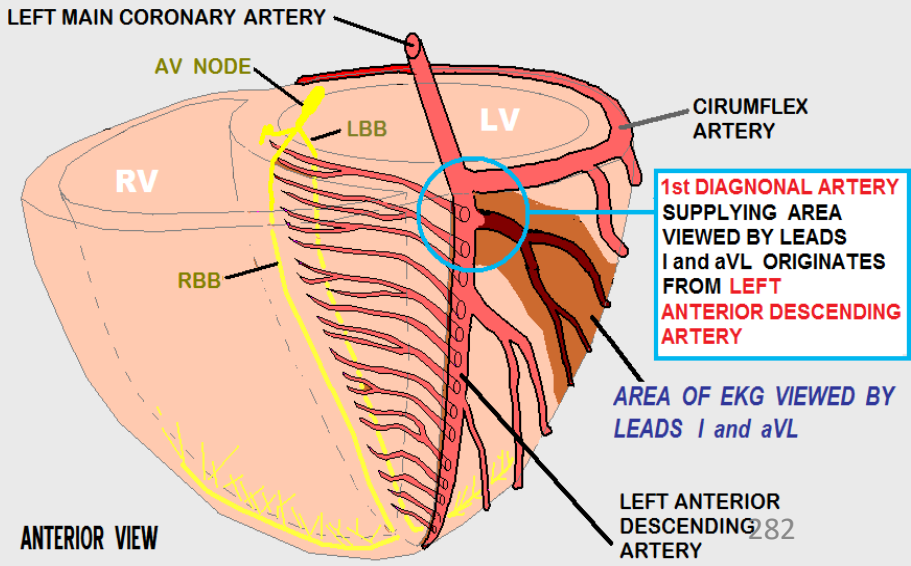
OCCLUSION of OBTUSE MARGINAL ARTERY

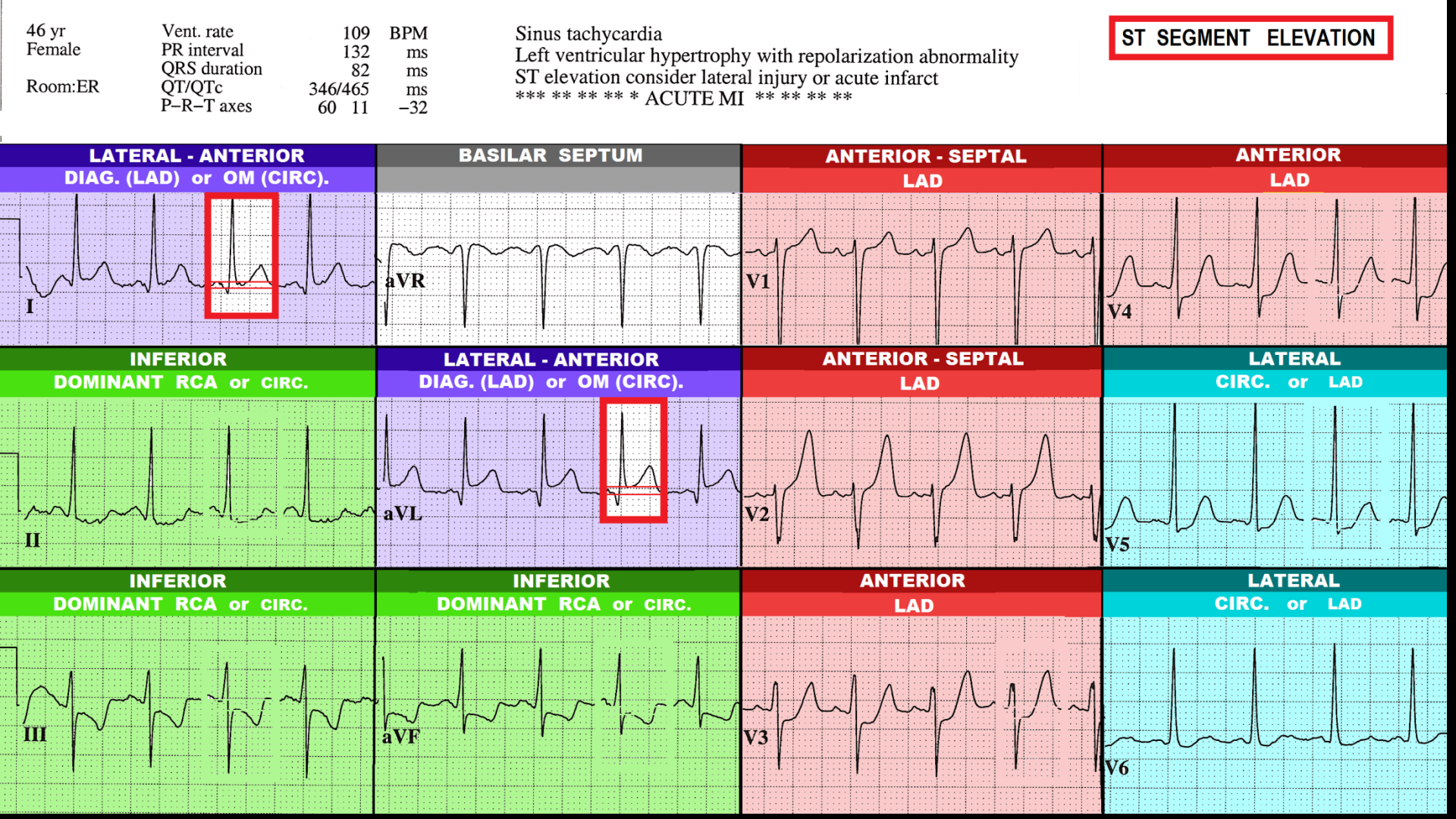


OCCLUSION of RAMUS ARTERY



OCCLUSION of DIAGONAL ARTERY

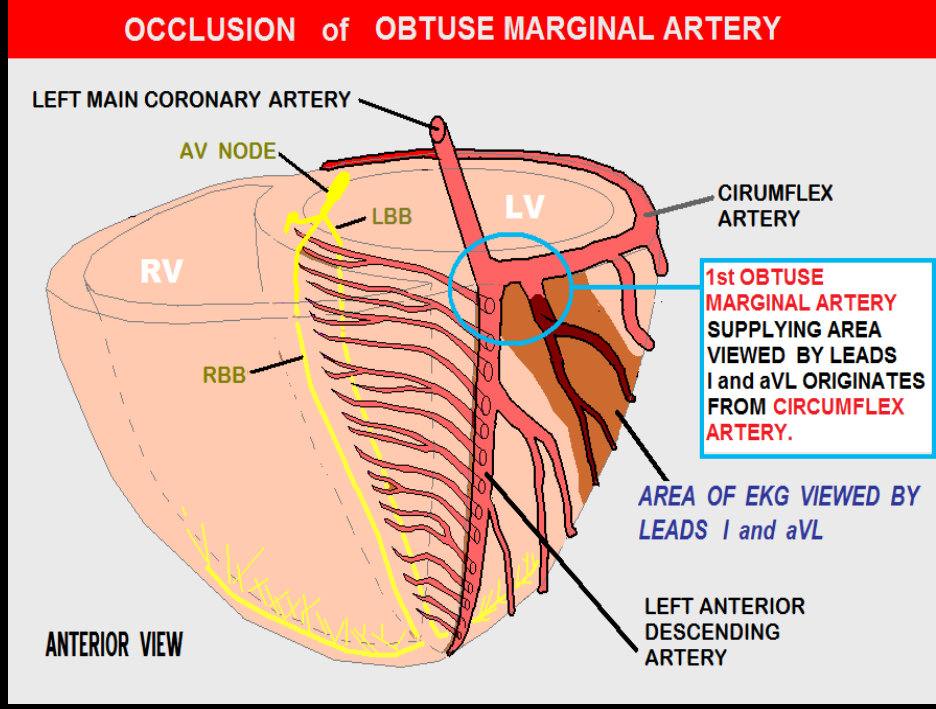


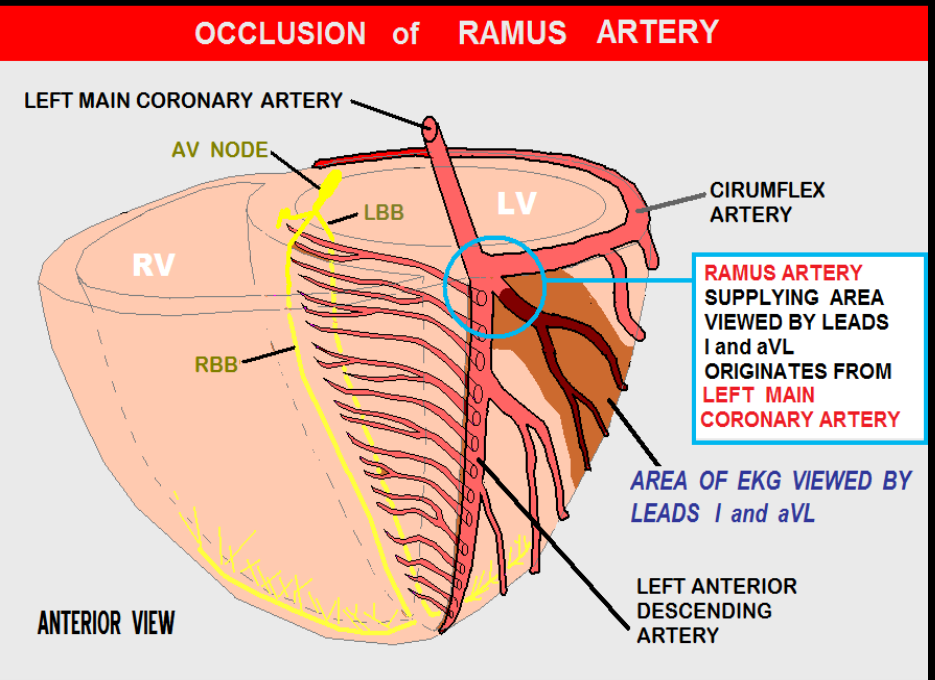


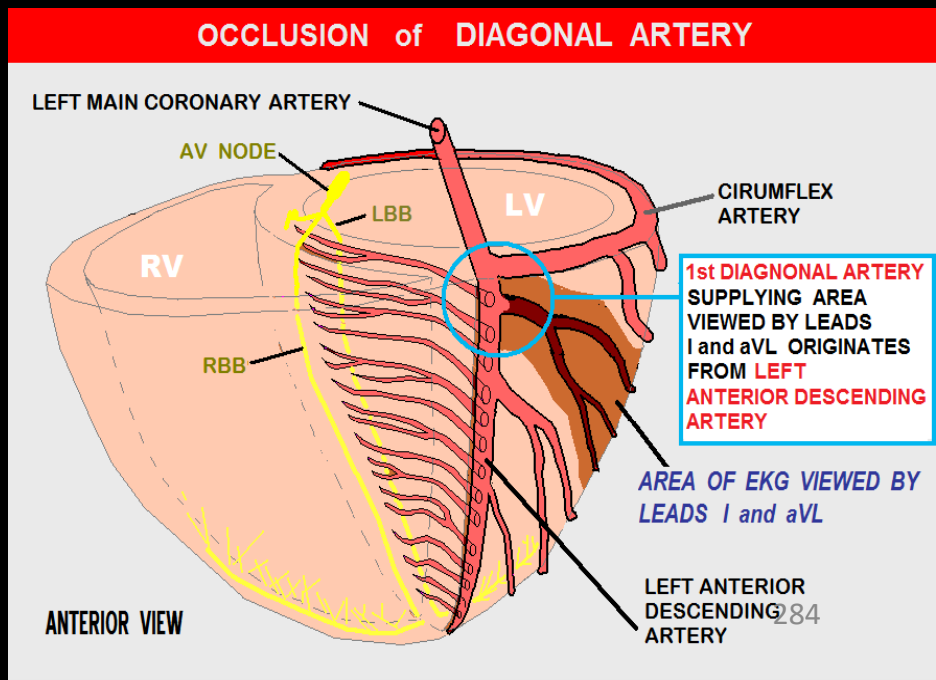
ST Segment elevation ONLY in Leads I and aVL

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

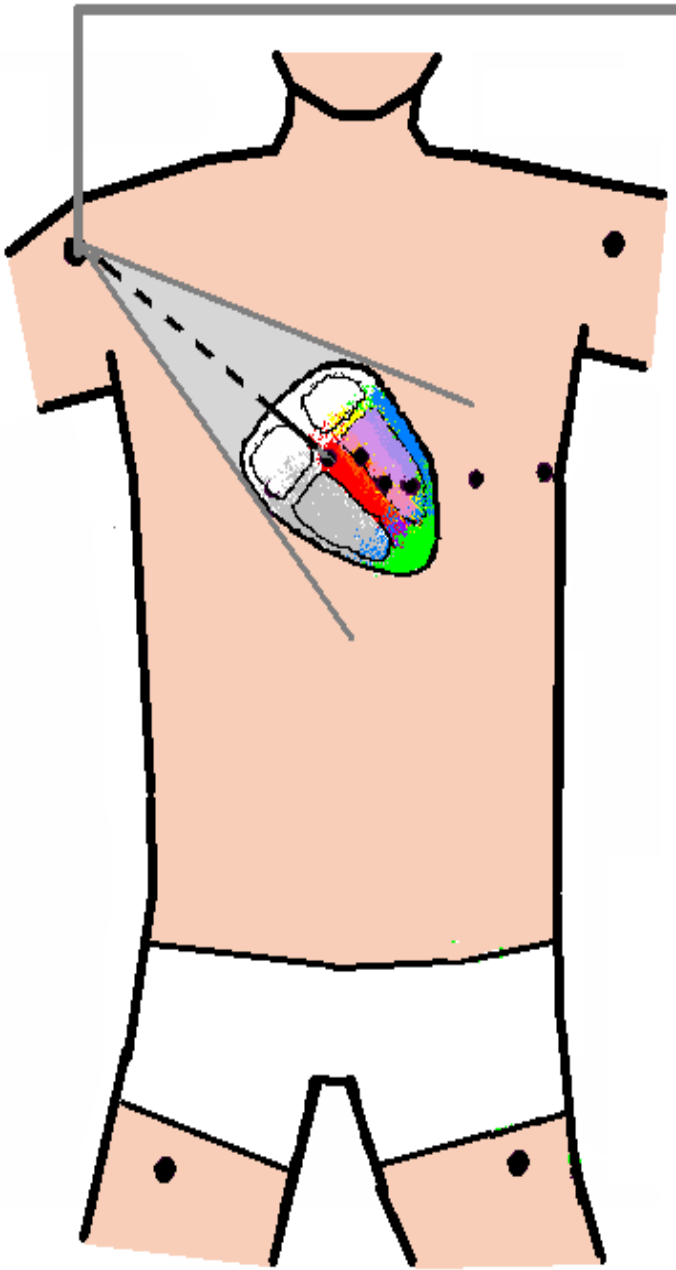
- **RAMUS BRANCH**
- **1st DIAGONAL off of LAD**
- **1st OBTUSE MARGINAL off of CIRCUMFLEX**

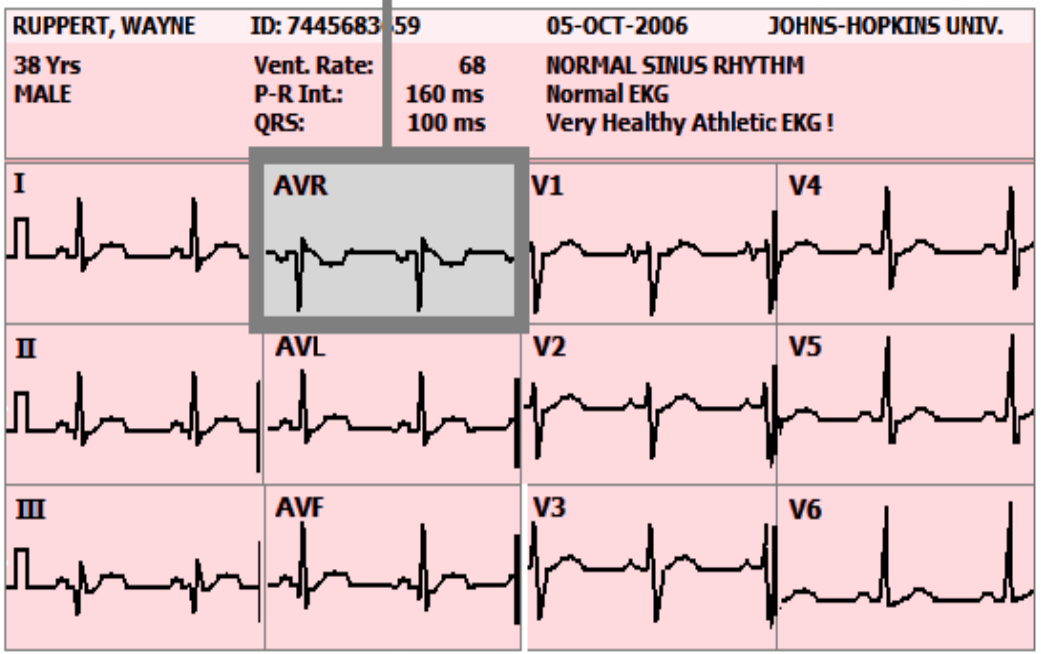


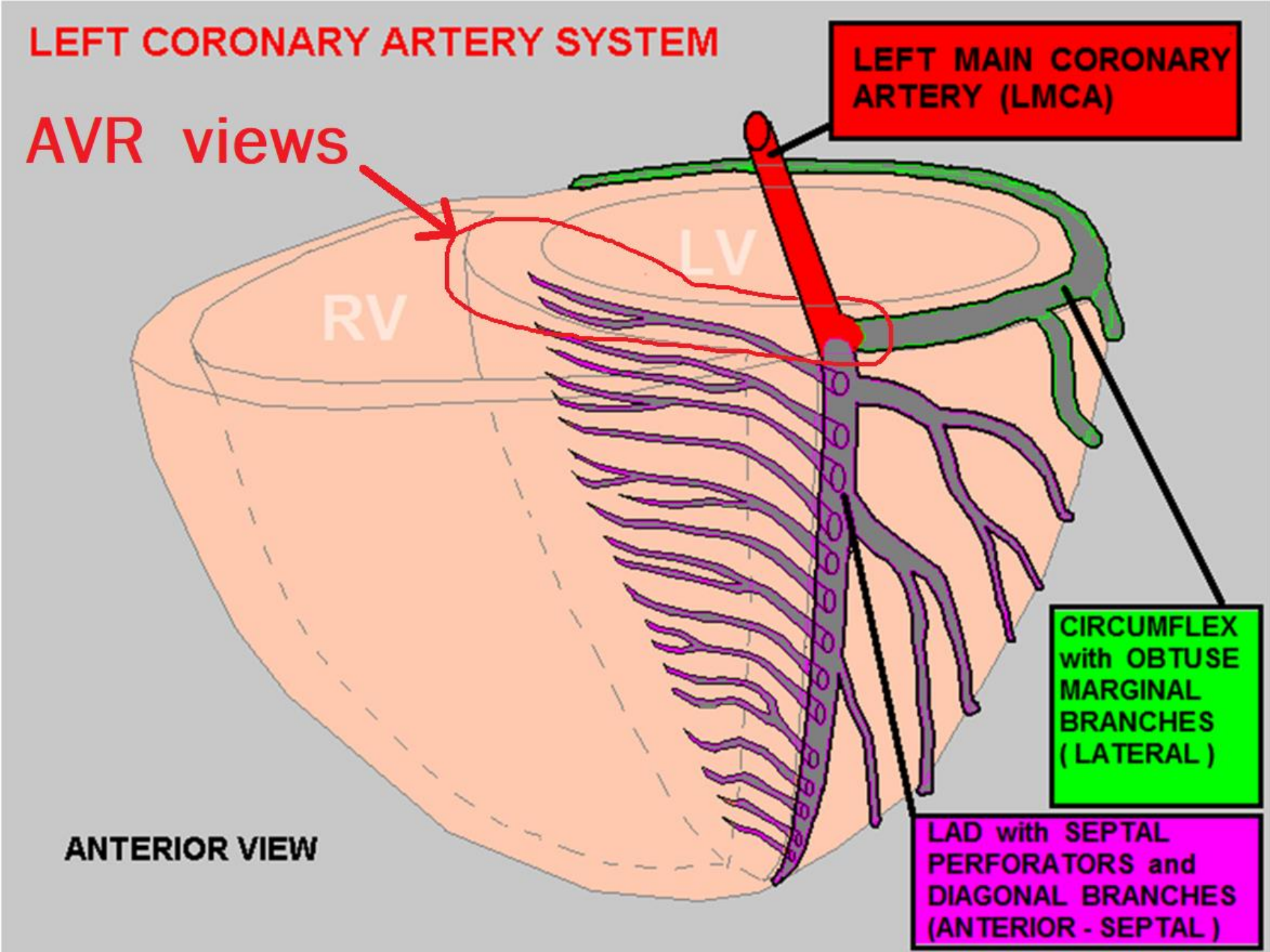




Lead AVR Views the BASILAR SEPTUM (region of the Bundle of His)

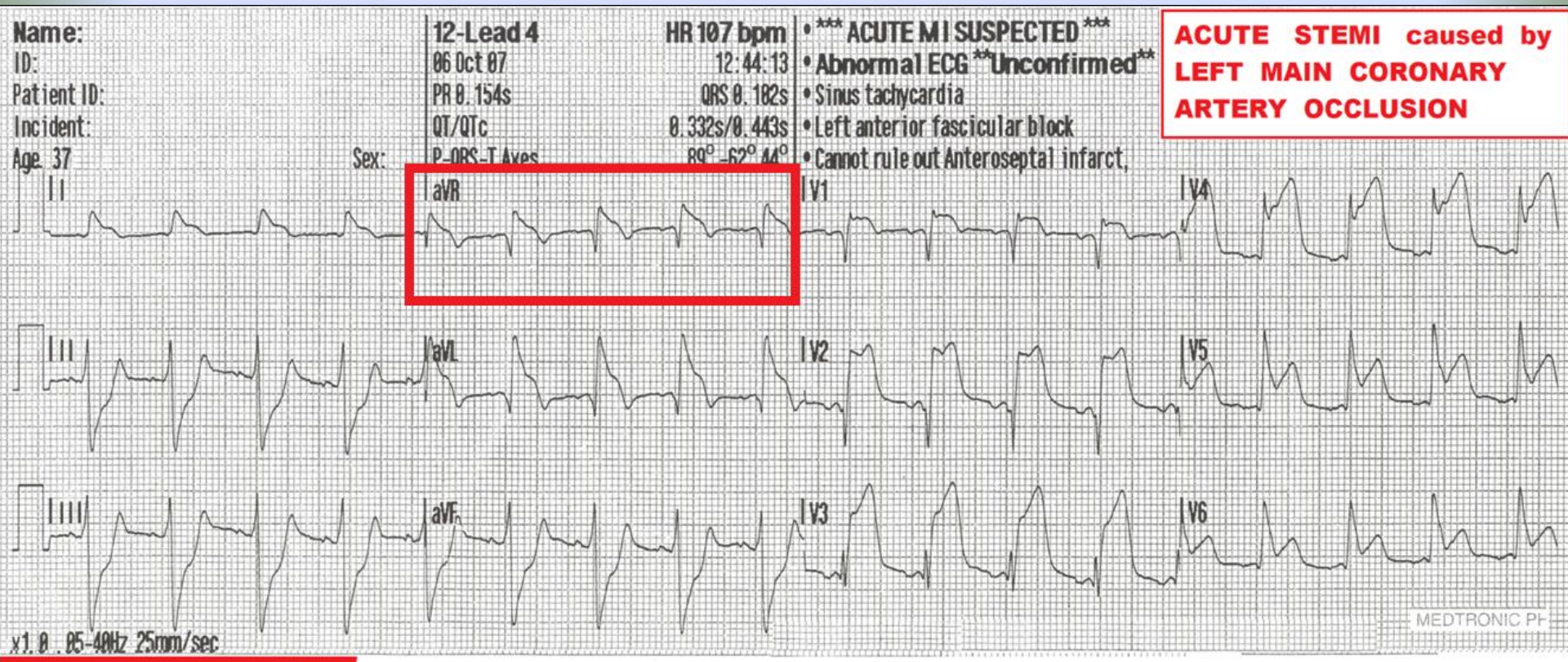





LEFT CORONARY ARTERY SYSTEM
AVR views
LEFT MAIN CORONARY ARTERY (LMCA)
LV
RV
CIRCUMFLEX with OBTUSE MARGINAL BRANCHES (LATERAL)
LAD with SEPTAL PERFORATORS and DIAGONAL BRANCHES (ANTERIOR - SEPTAL)
ANTERIOR VIEW

When LEAD AVR shows ST Elevation:

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

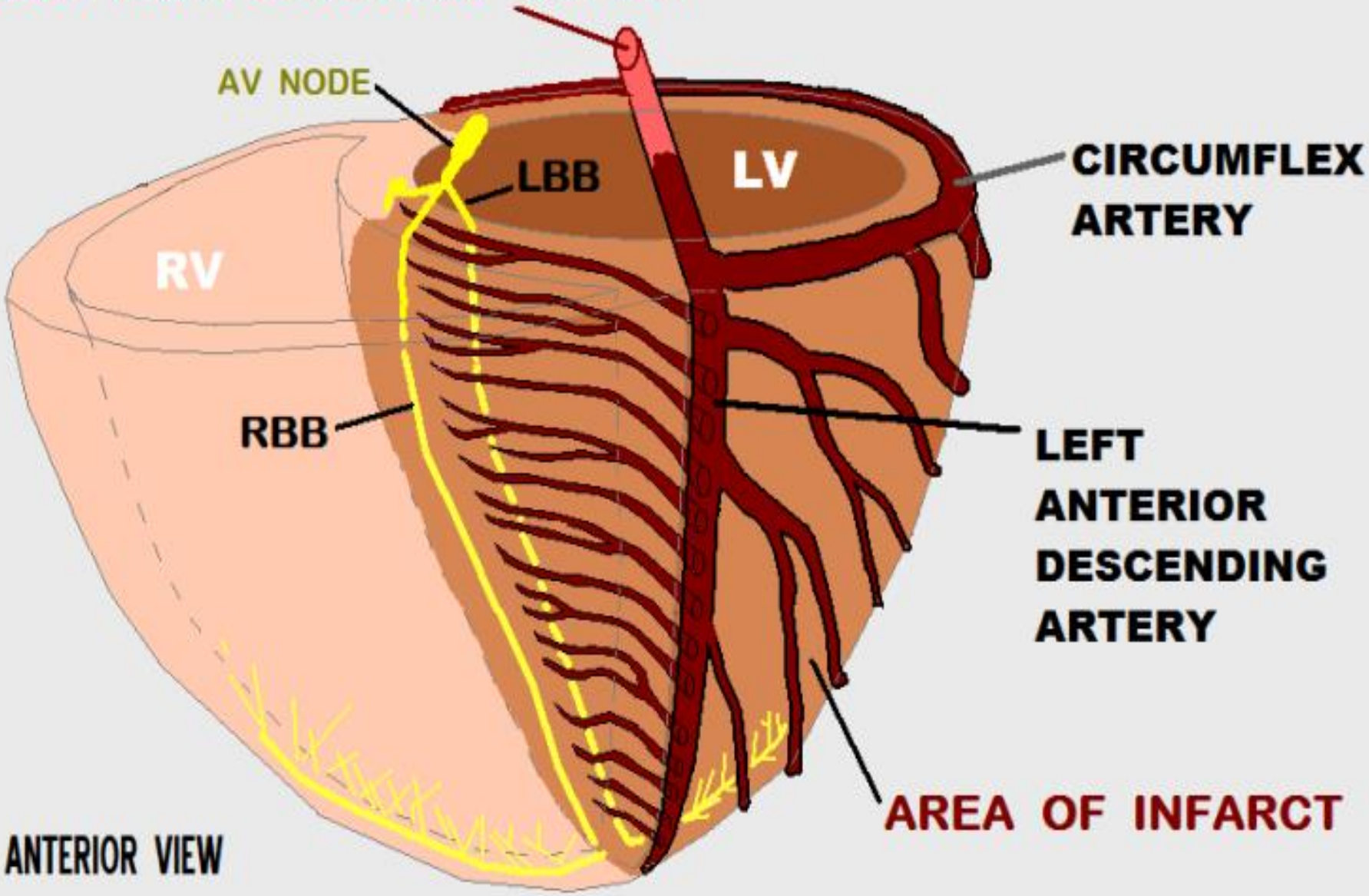


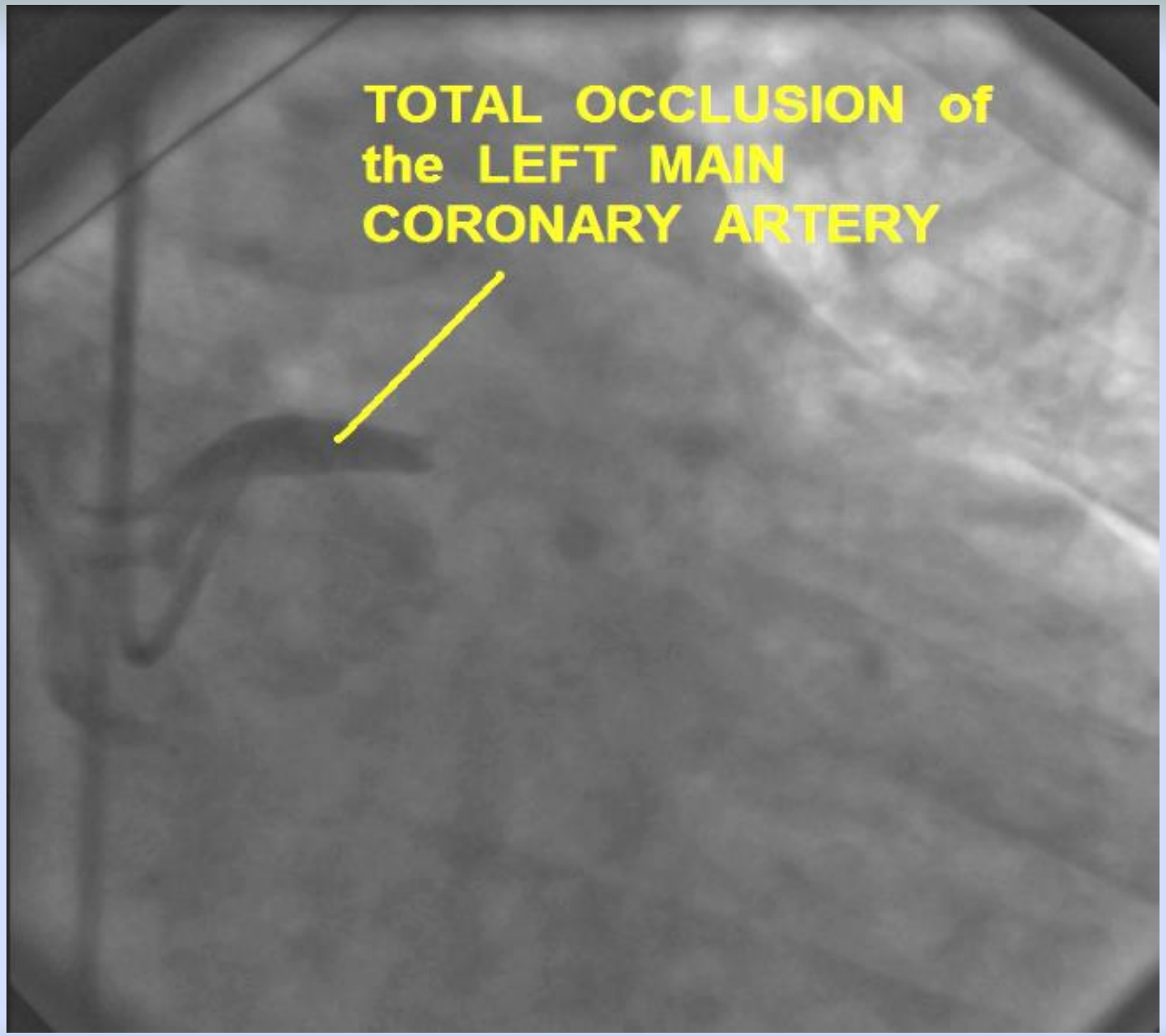
ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION

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

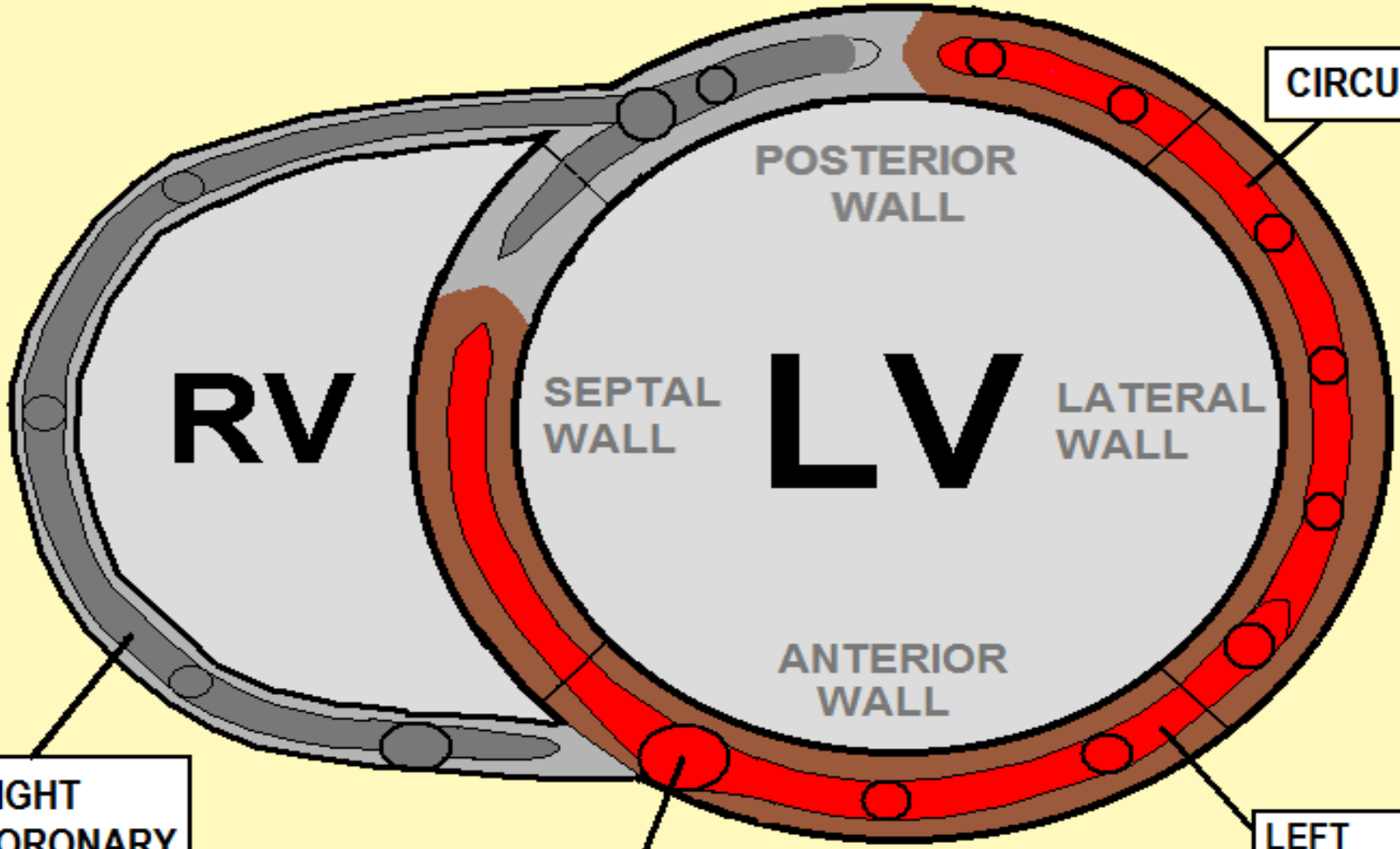
OCCLUSION of the LEFT MAIN CORONARY ARTERY




TOTAL OCCLUSION of
the LEFT MAIN
CORONARY ARTERY

The LEFT MAIN CORONARY ARTERY

SUPPLIES 75 - 100 % of the LEFT VENTRICULAR MUSCLE MASS



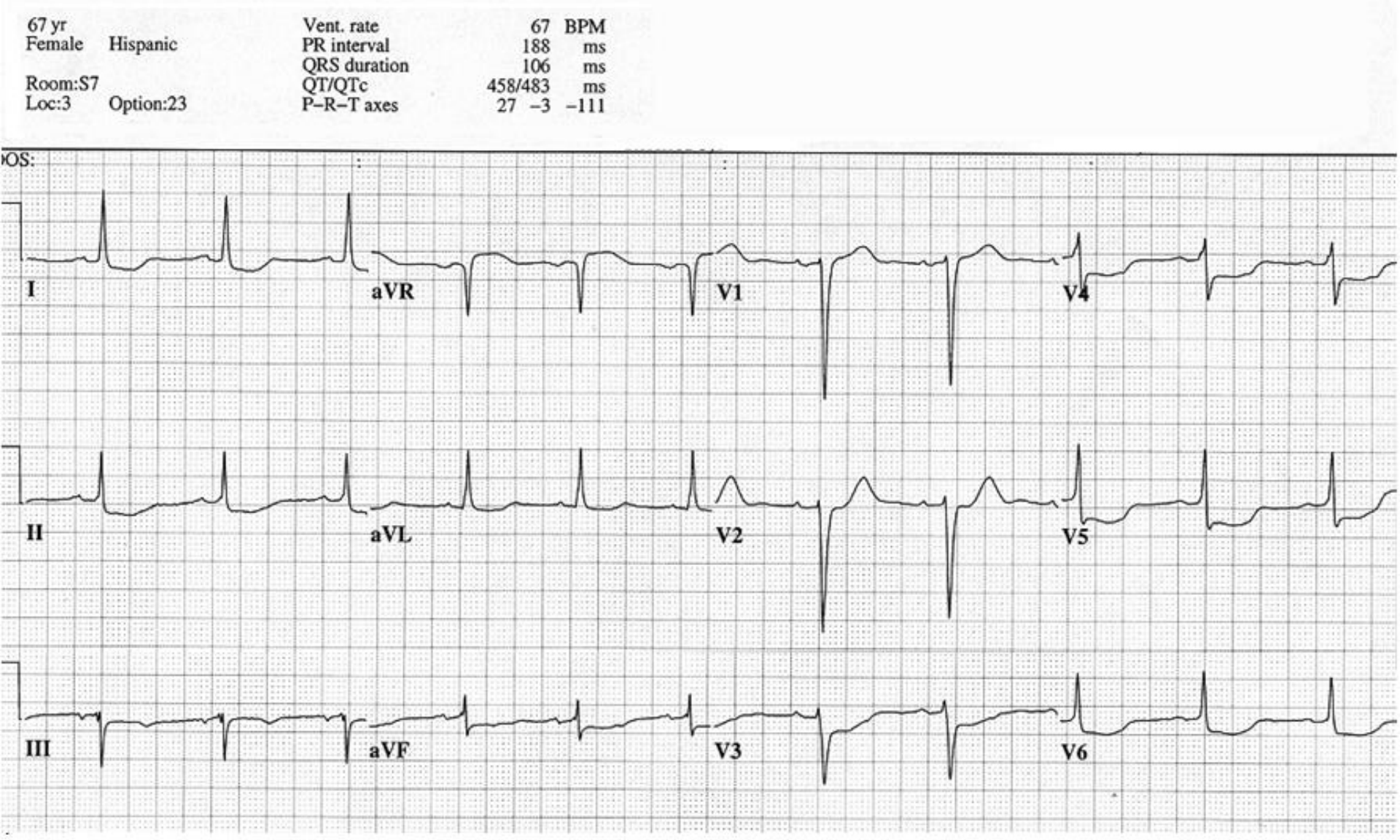
When LEAD AVR shows ST Elevation:

- **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

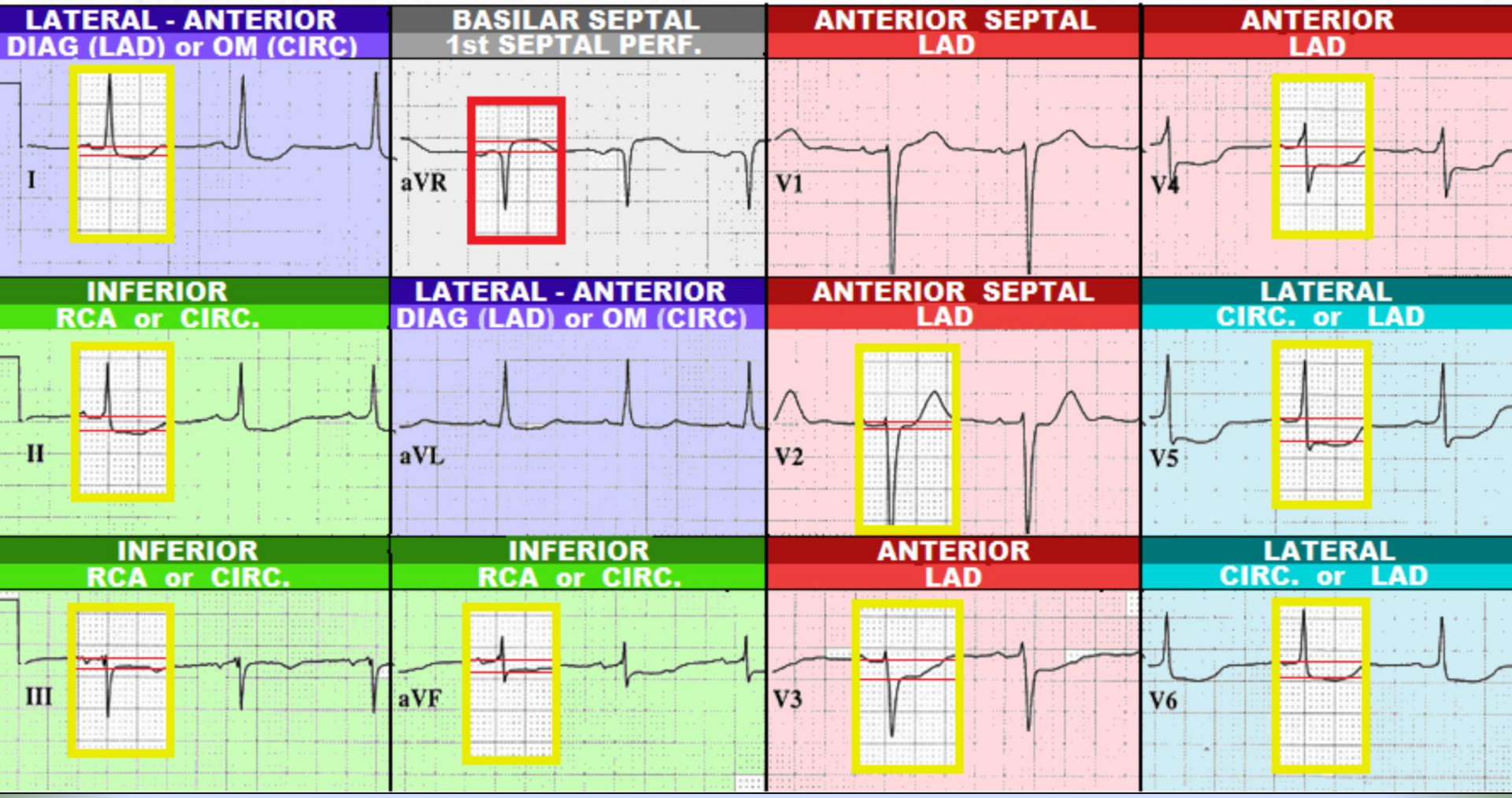
67 yr
Female Hispanic

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

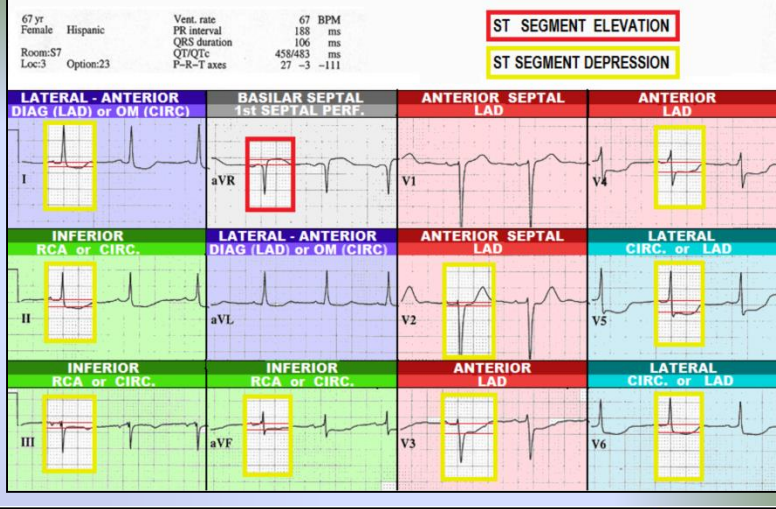
ST SEGMENT DEPRESSION

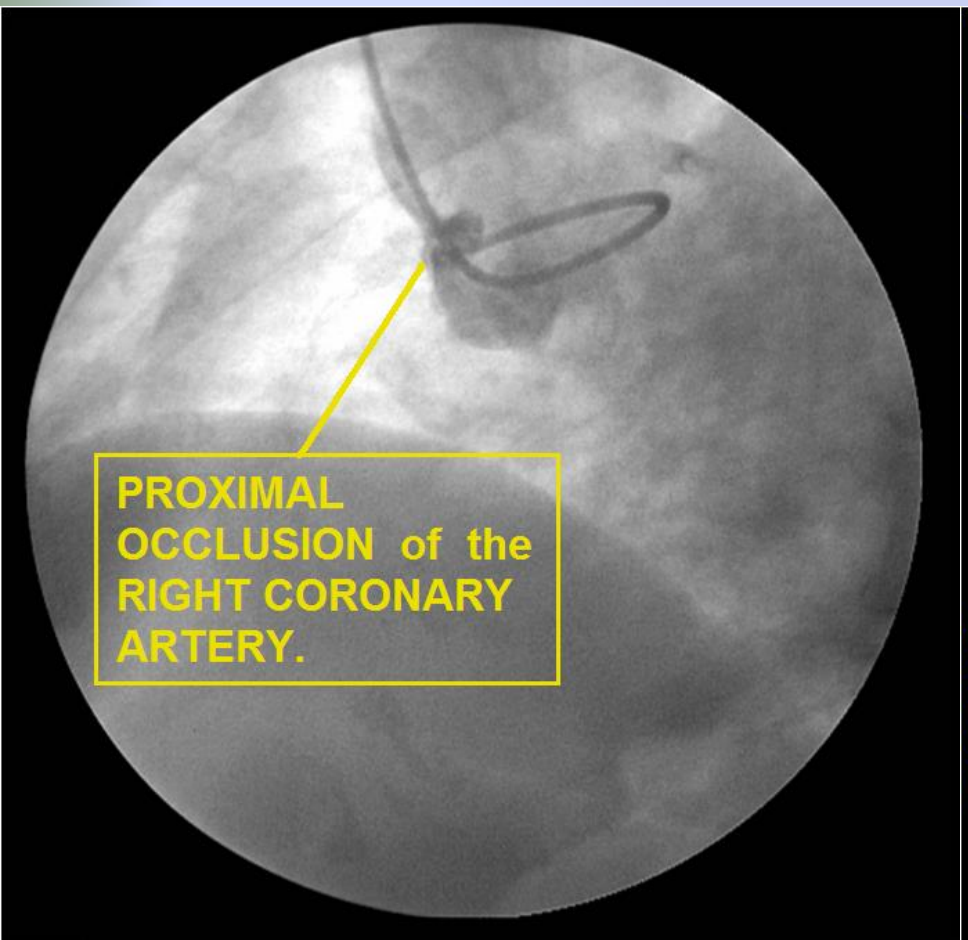


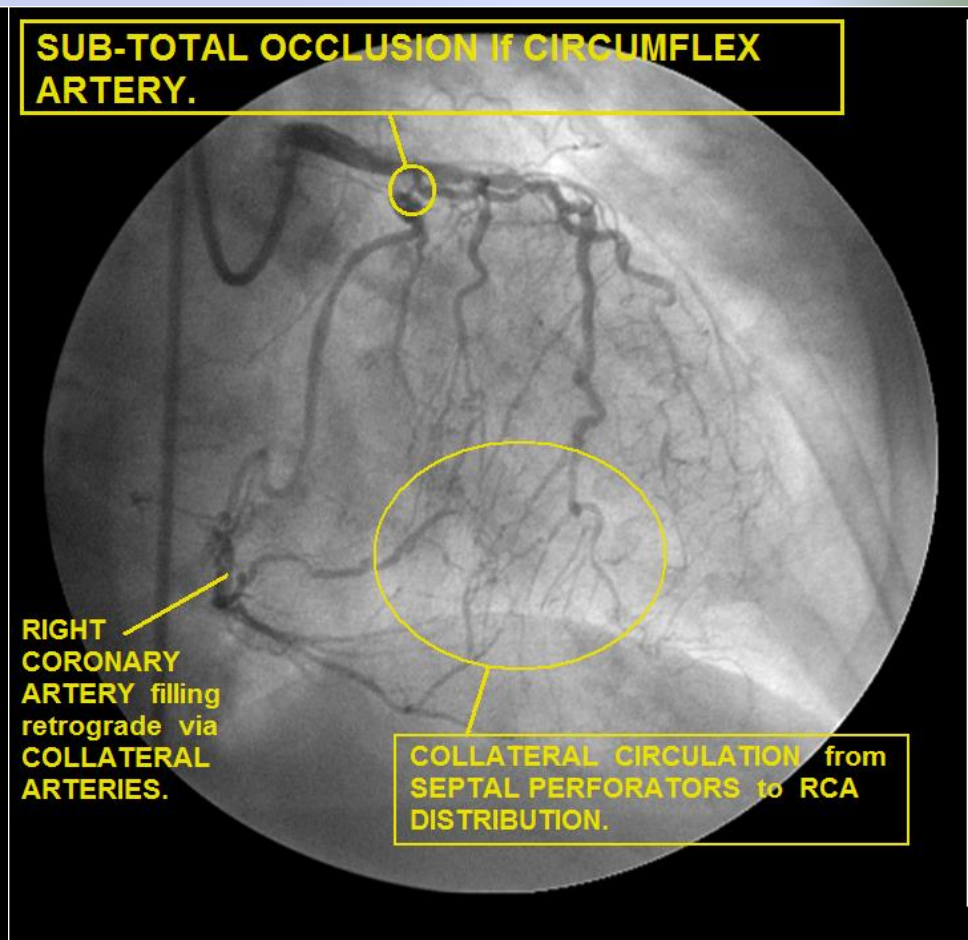
GLOBAL ISCHEMIA

- **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*



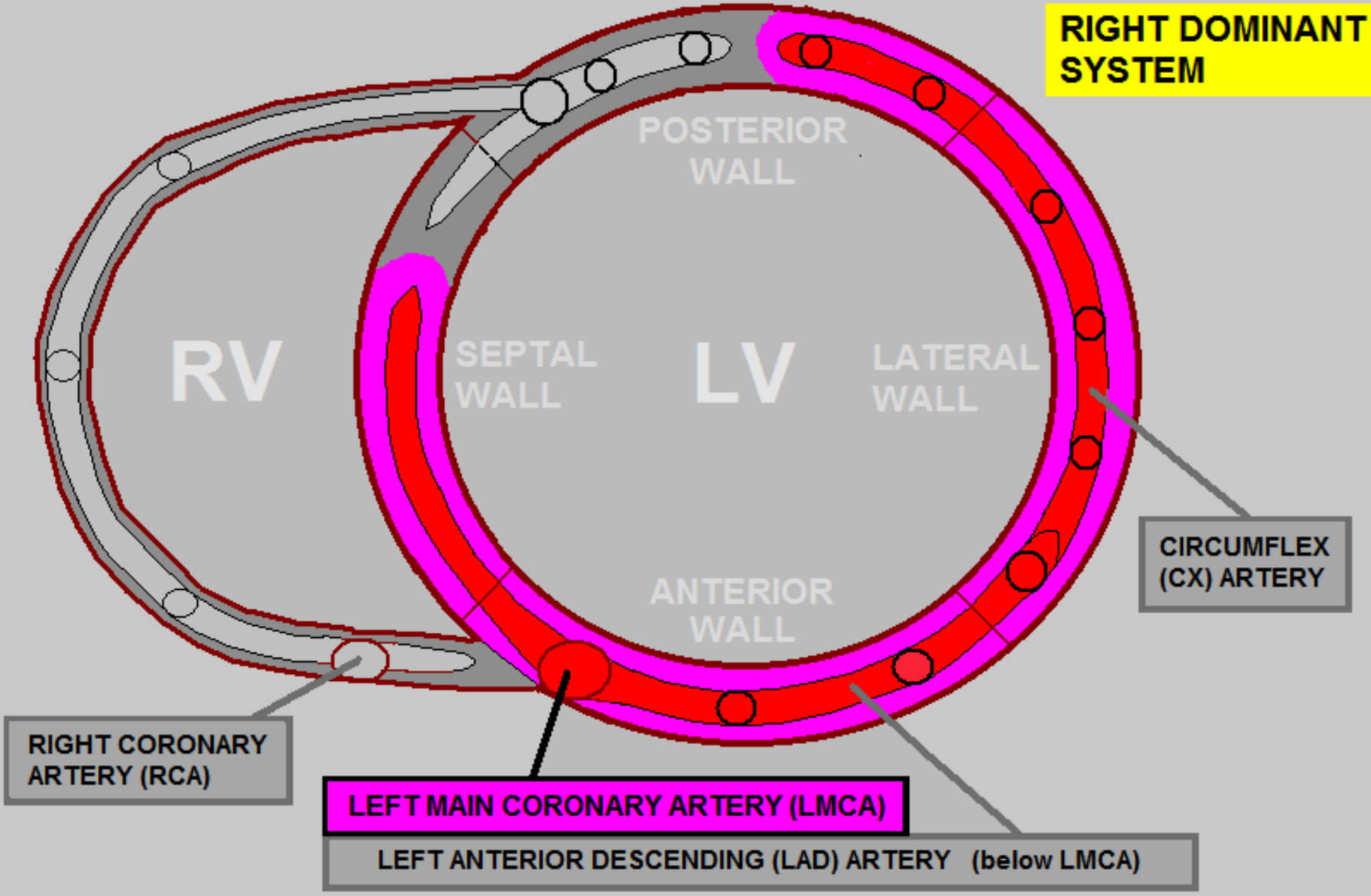




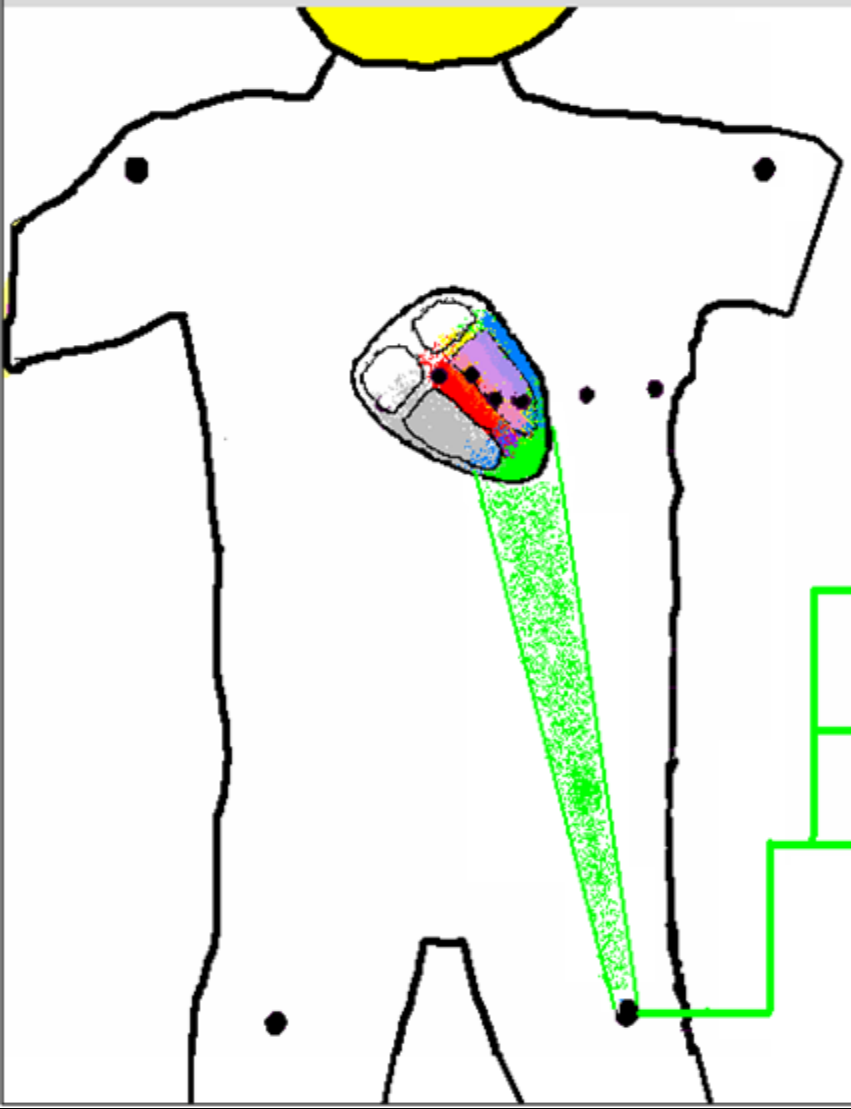
cutaway view of the

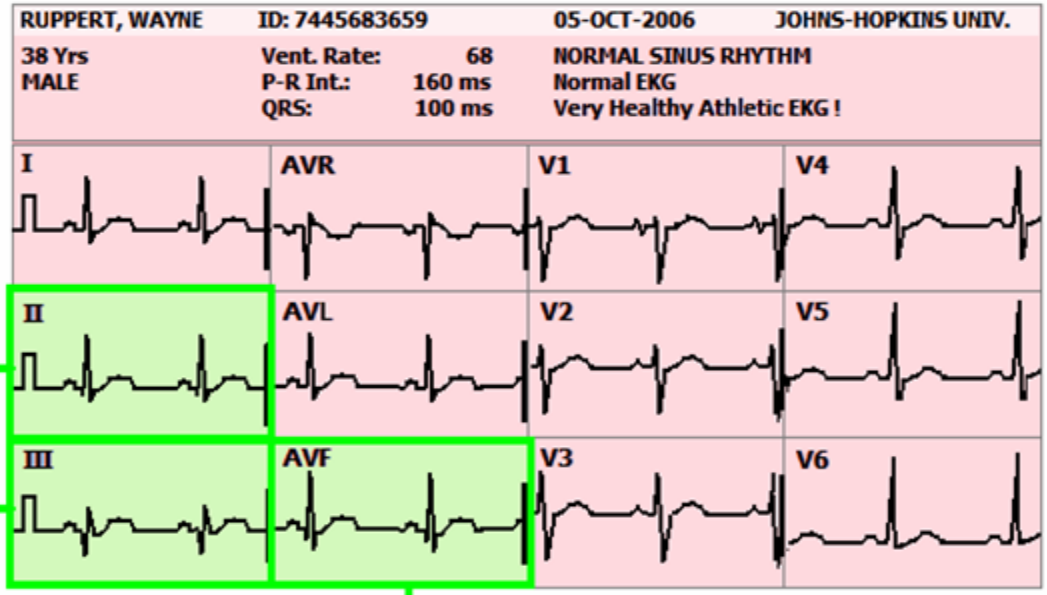
LEFT MAIN CORONARY ARTERY (LMCA)

SUPPLIES APPROXIMATELY 75% OF LV MUSCLE MASS

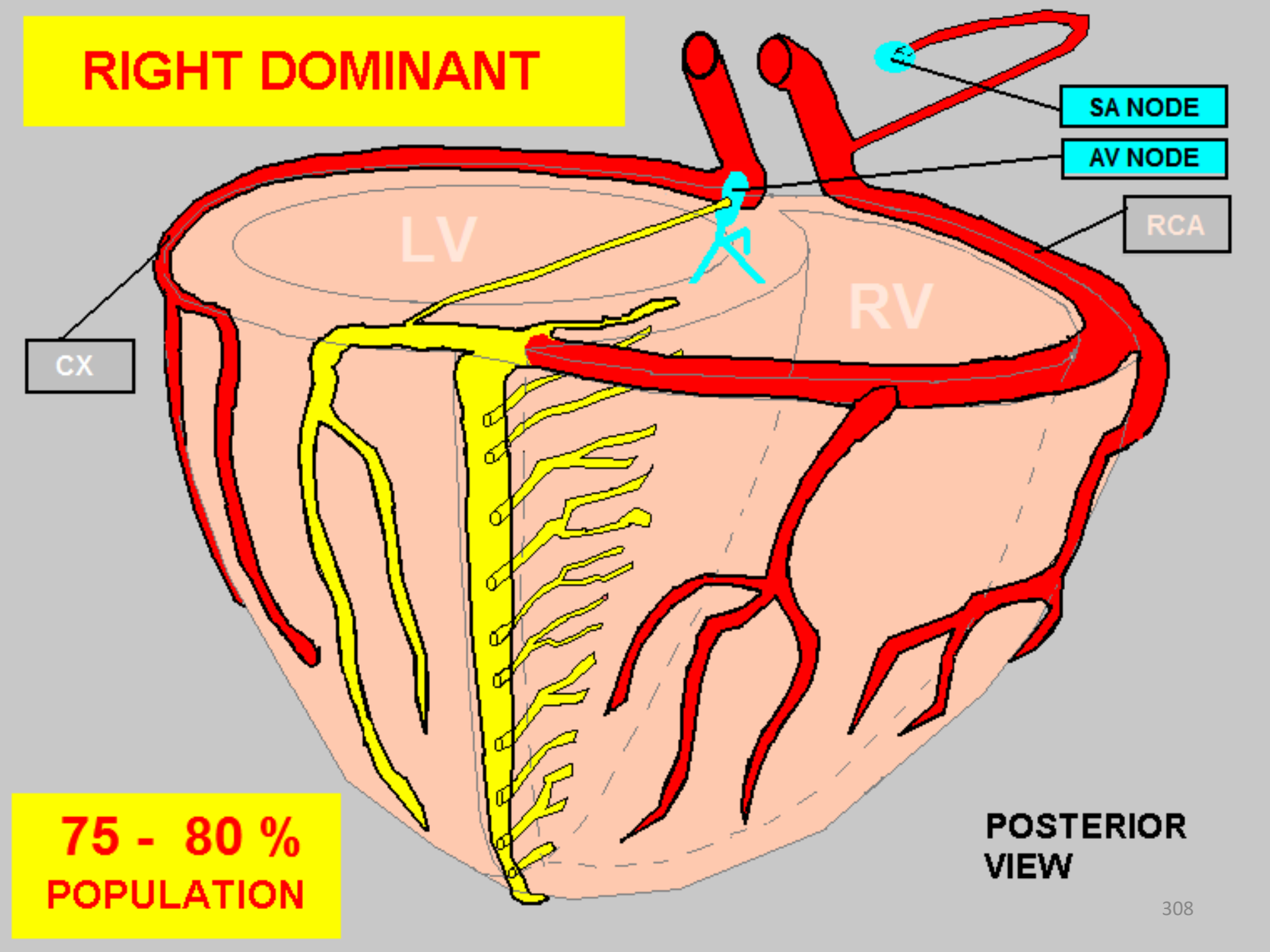


LEADS II, III, and aVF VIEW
INFERIOR WALL of the LEFT VENTRICLE





Which CORONARY ARTERY usually supplies the INFERIOR WALL?

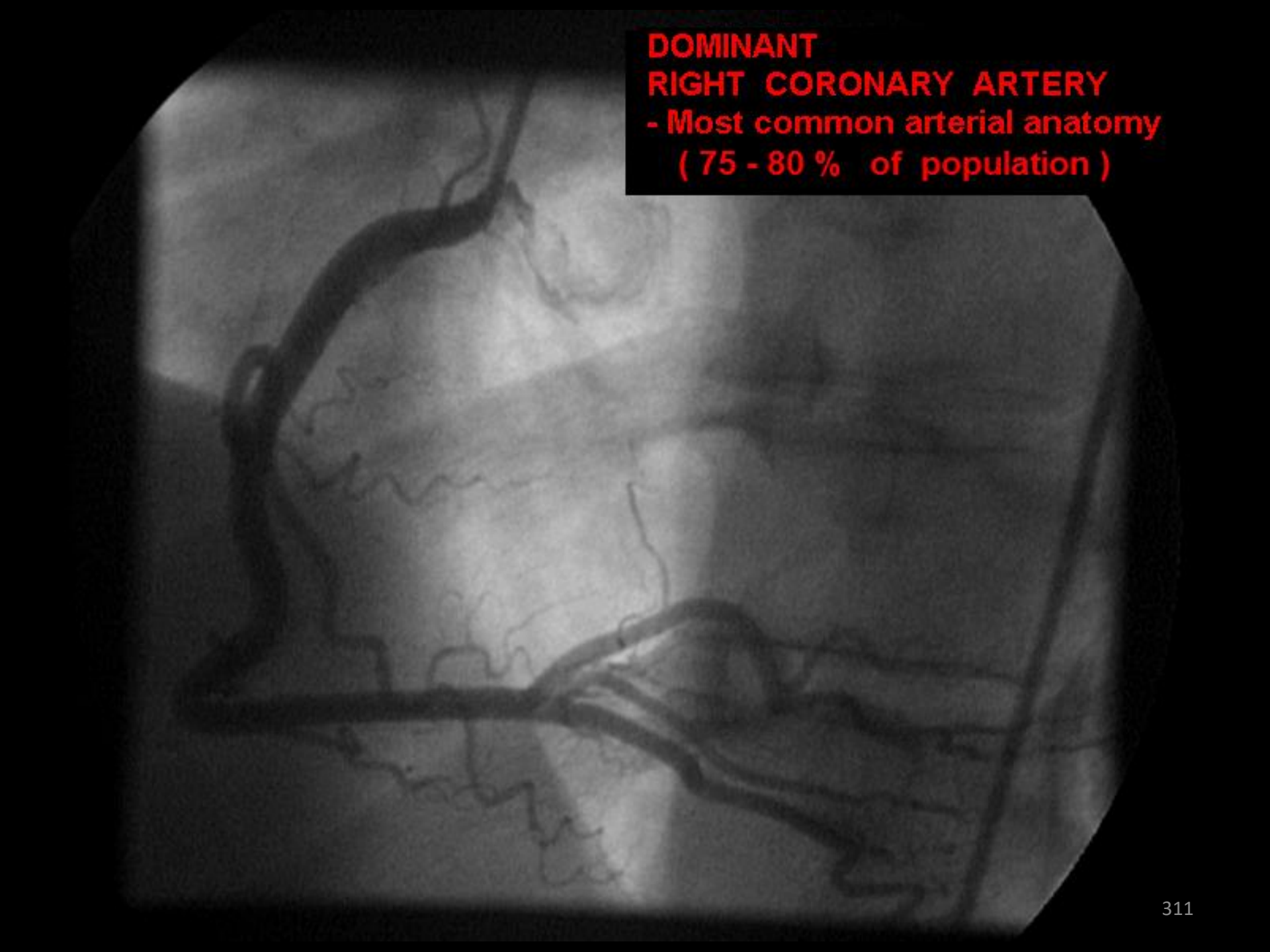

RIGHT DOMINANT
SA NODE
AV NODE
RCA
LV
RV
CX
75 - 80 %
POPULATION
POSTERIOR
VIEW

HELPFUL HINT . . . MEMORIZE THIS !

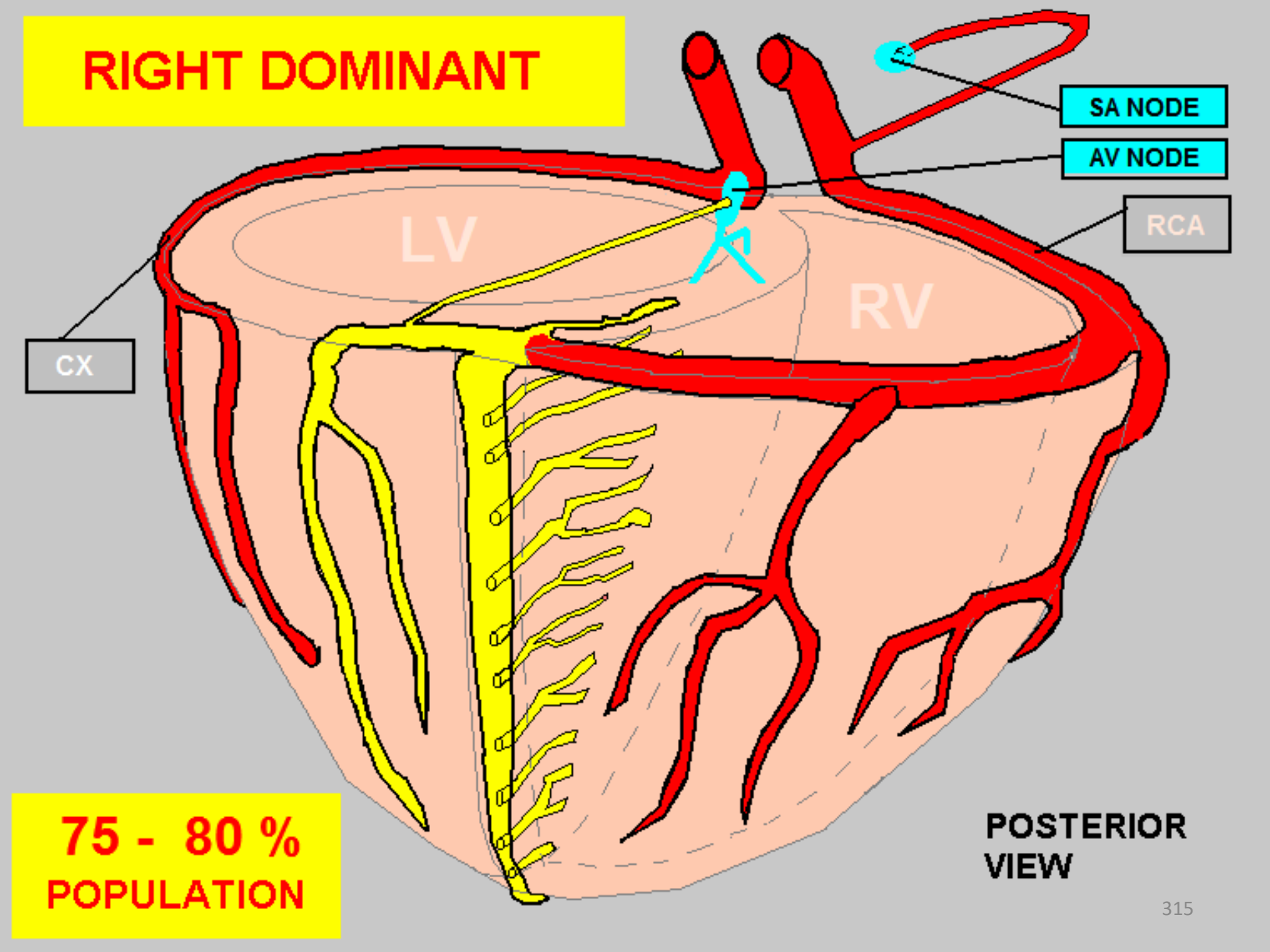
RIGHT CORONARY ARTERY (RCA)

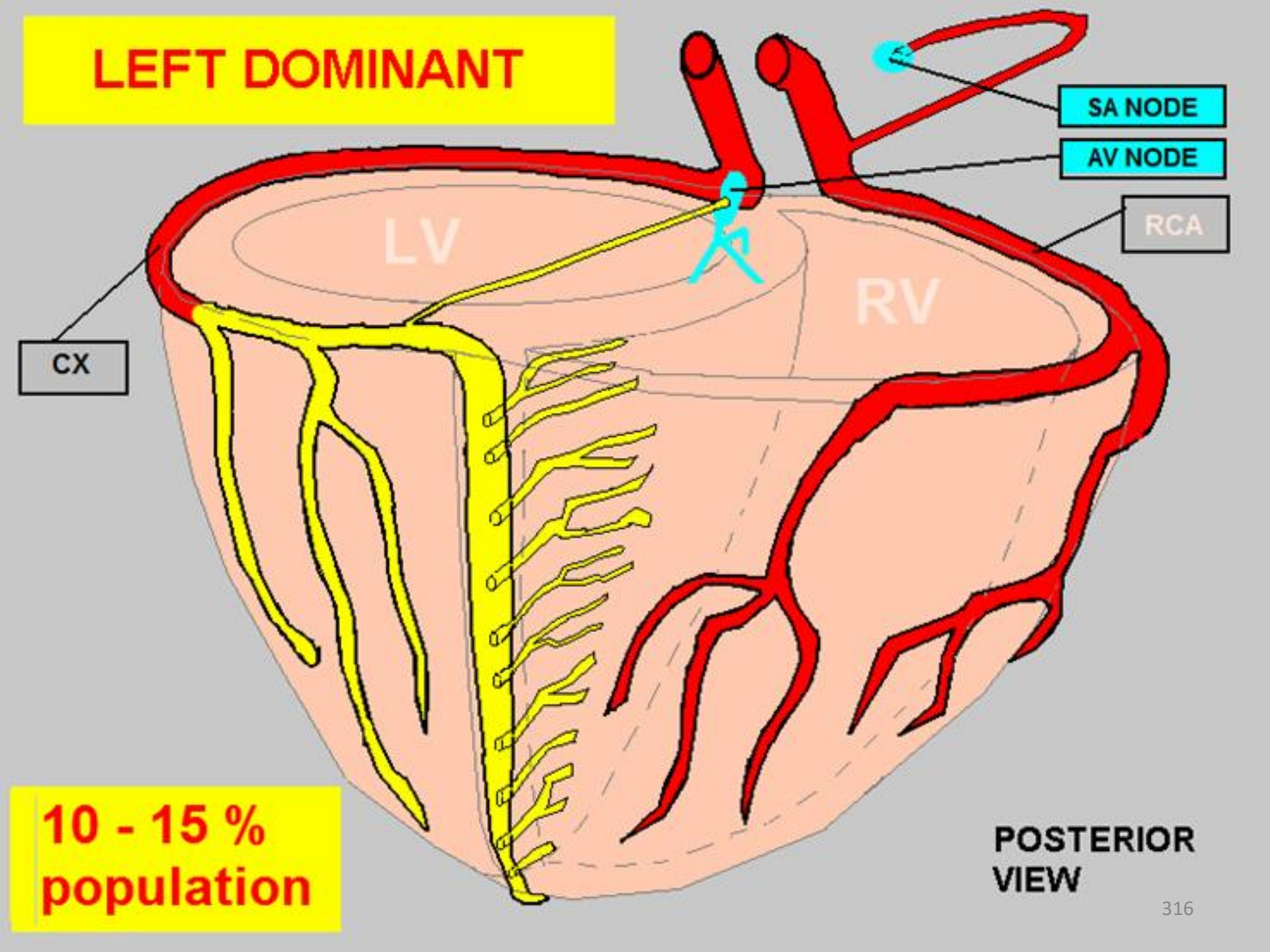
RIGHT DOMINANT SYSTEMS

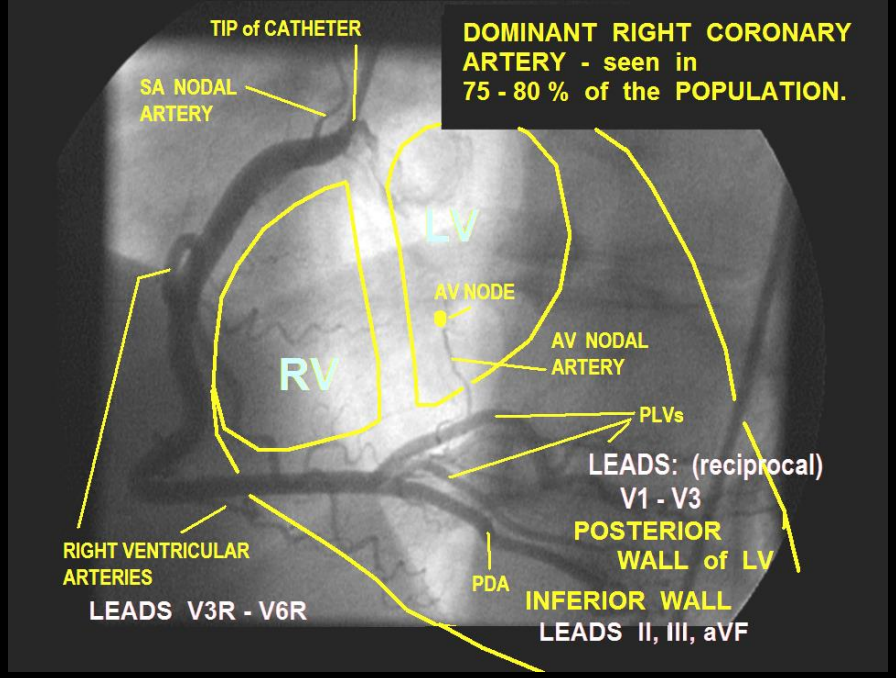
- RIGHT ATRIUM
- SINUS NODE (55% of the population)
- RIGHT VENTRICLE - 100 % of muscle mass
- LEFT VENTRICLE: 15 - 25 % of muscle mass
  - INFERIOR WALL
  - approx. 1/2 of POSTERIOR WALL
- AV NODE

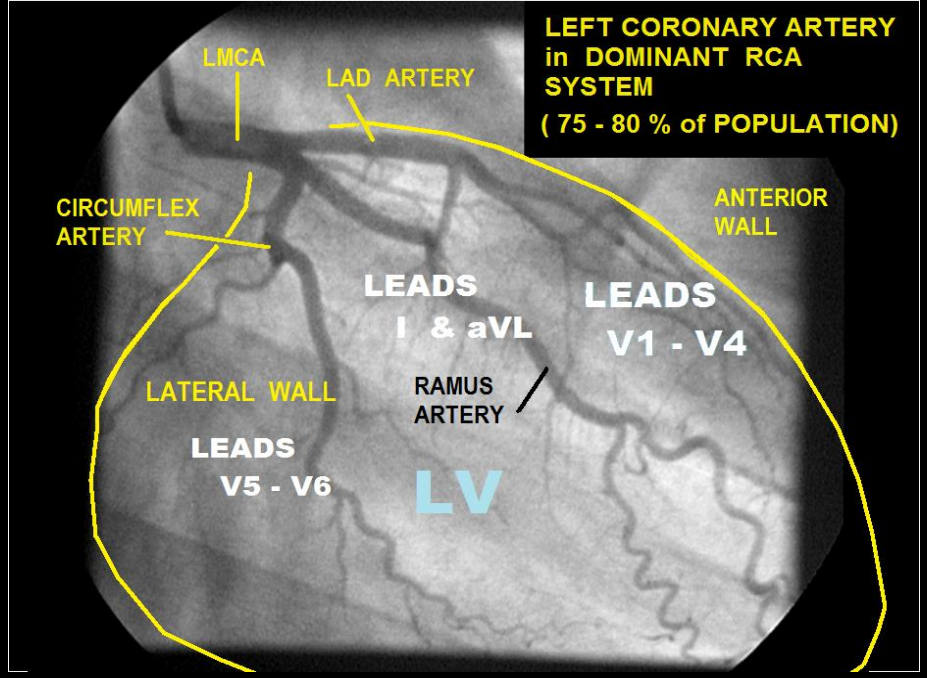

DOMINANT
RIGHT CORONARY ARTERY
- Most common arterial anatomy
(75 - 80 % of population)

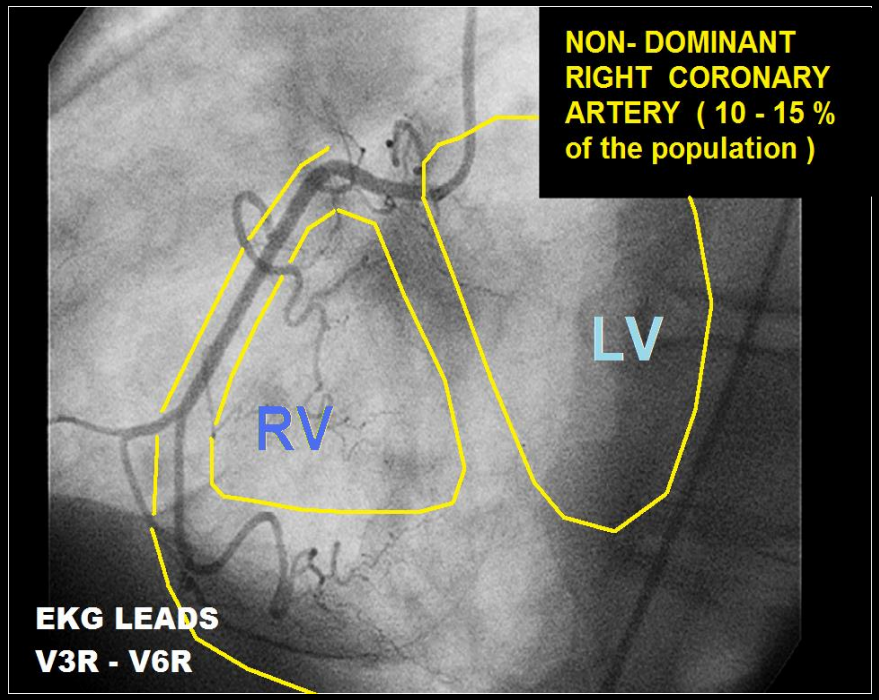
So if the Right Coronary Artery Is DOMINANT in 75 – 80% of the POPULATION, what accounts for the Other 20 – 25% ??

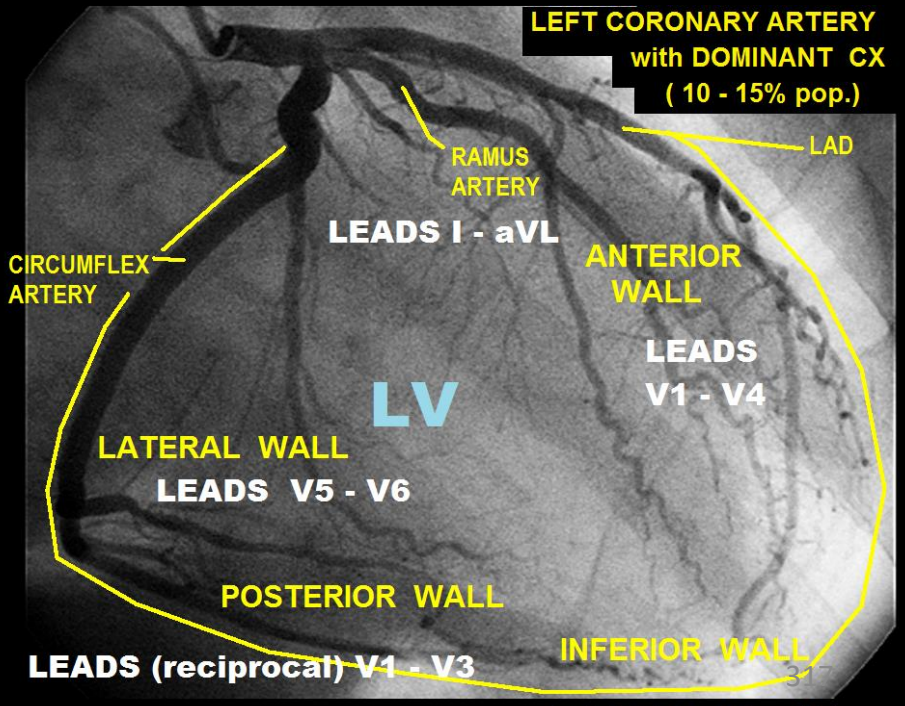

RIGHT DOMINANT
SA NODE
AV NODE
RCA
LV
RV
CX
75 - 80 %
POPULATION
POSTERIOR
VIEW

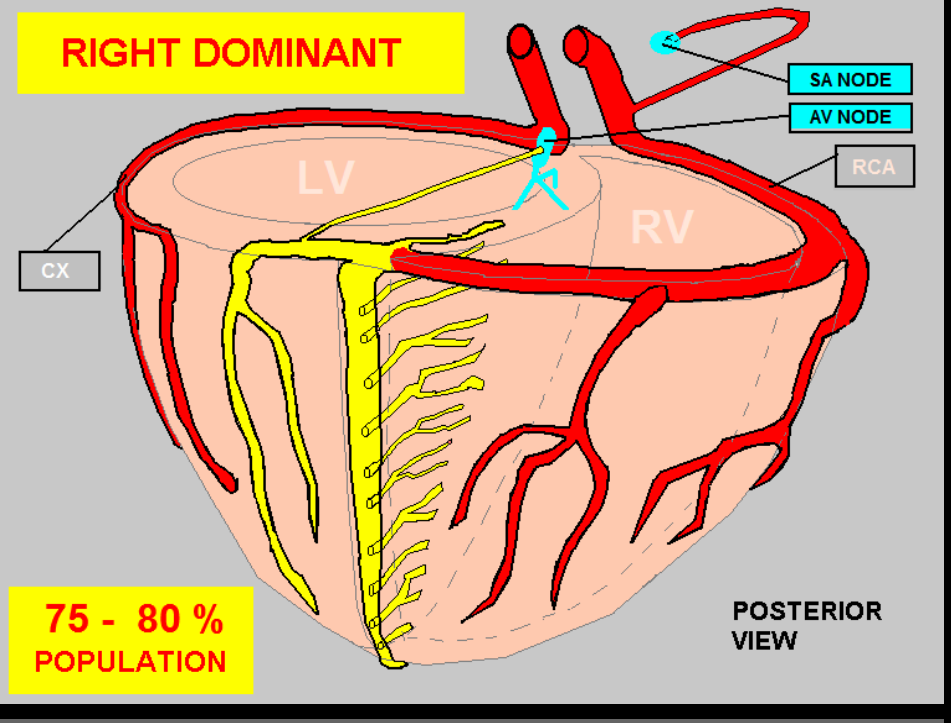

LEFT DOMINANT
SA NODE
AV NODE
RCA
LV
RV
CX
10 - 15 %
population
POSTERIOR
VIEW

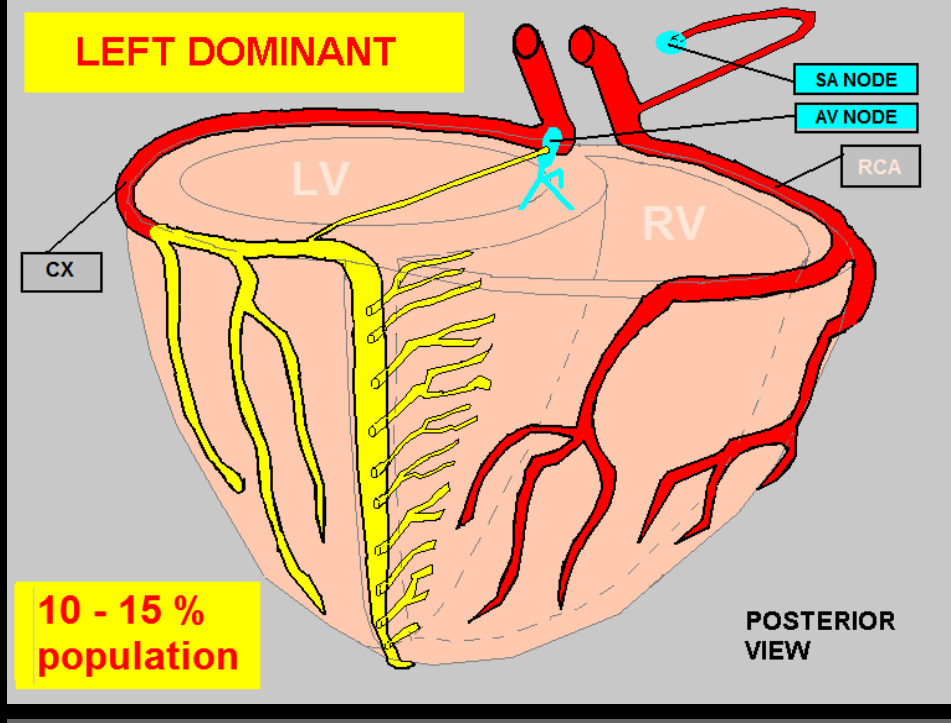

TIP of CATHETER
SA NODAL ARTERY
DOMINANT RIGHT CORONARY ARTERY - seen in 75 - 80 % of the POPULATION.
LV
AV NODE
AV NODAL ARTERY
RV
PLVs
LEADS: (reciprocal) V1 - V3
POSTERIOR WALL of LV
RIGHT VENTRICULAR ARTERIES
LEADS V3R - V6R
PDA
INFERIOR WALL
LEADS II, III, aVF

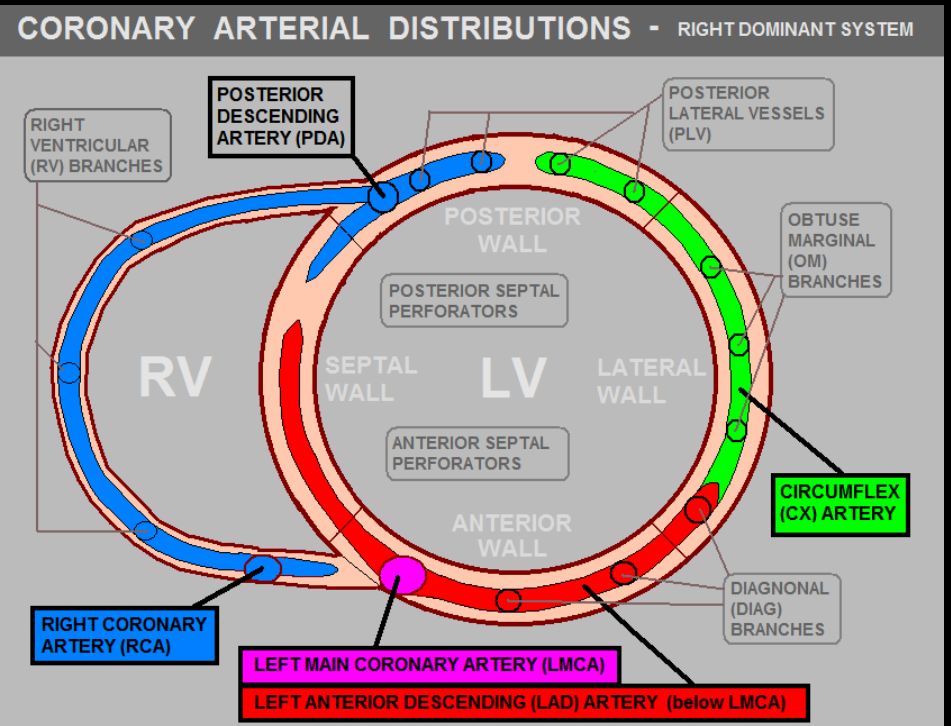

LMCA
LAD ARTERY
LEFT CORONARY ARTERY in DOMINANT RCA SYSTEM
(75 - 80 % of POPULATION)
CIRCUMFLEX ARTERY
ANTERIOR WALL
LEADS I & aVL
LEADS V1 - V4
LATERAL WALL
RAMUS ARTERY
LEADS V5 - V6
LV

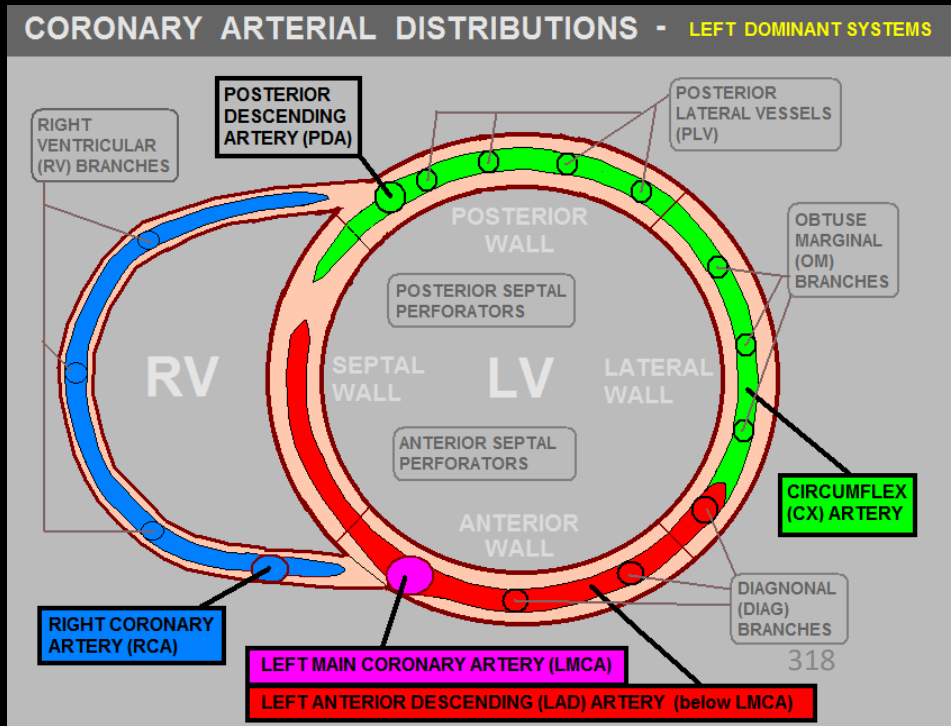

NON- DOMINANT RIGHT CORONARY ARTERY (10 - 15 % of the population)
LV
RV
EKG LEADS V3R - V6R


LEFT CORONARY ARTERY with DOMINANT CX (10 - 15% pop.)
LAD
RAMUS ARTERY
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

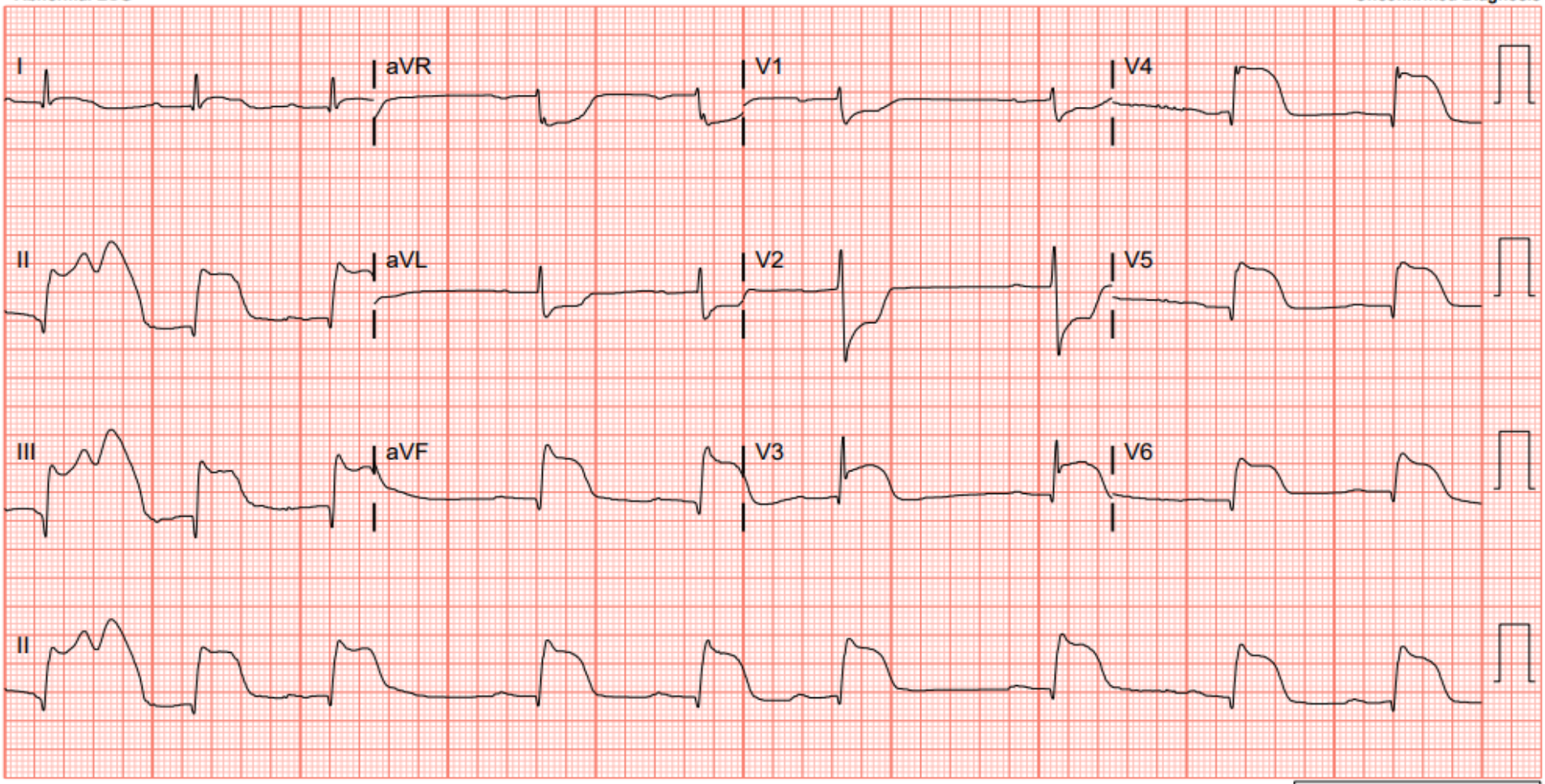
Bayfront Health Seven Rivers ED
Dept ED
Room EDWR
Tech mg

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

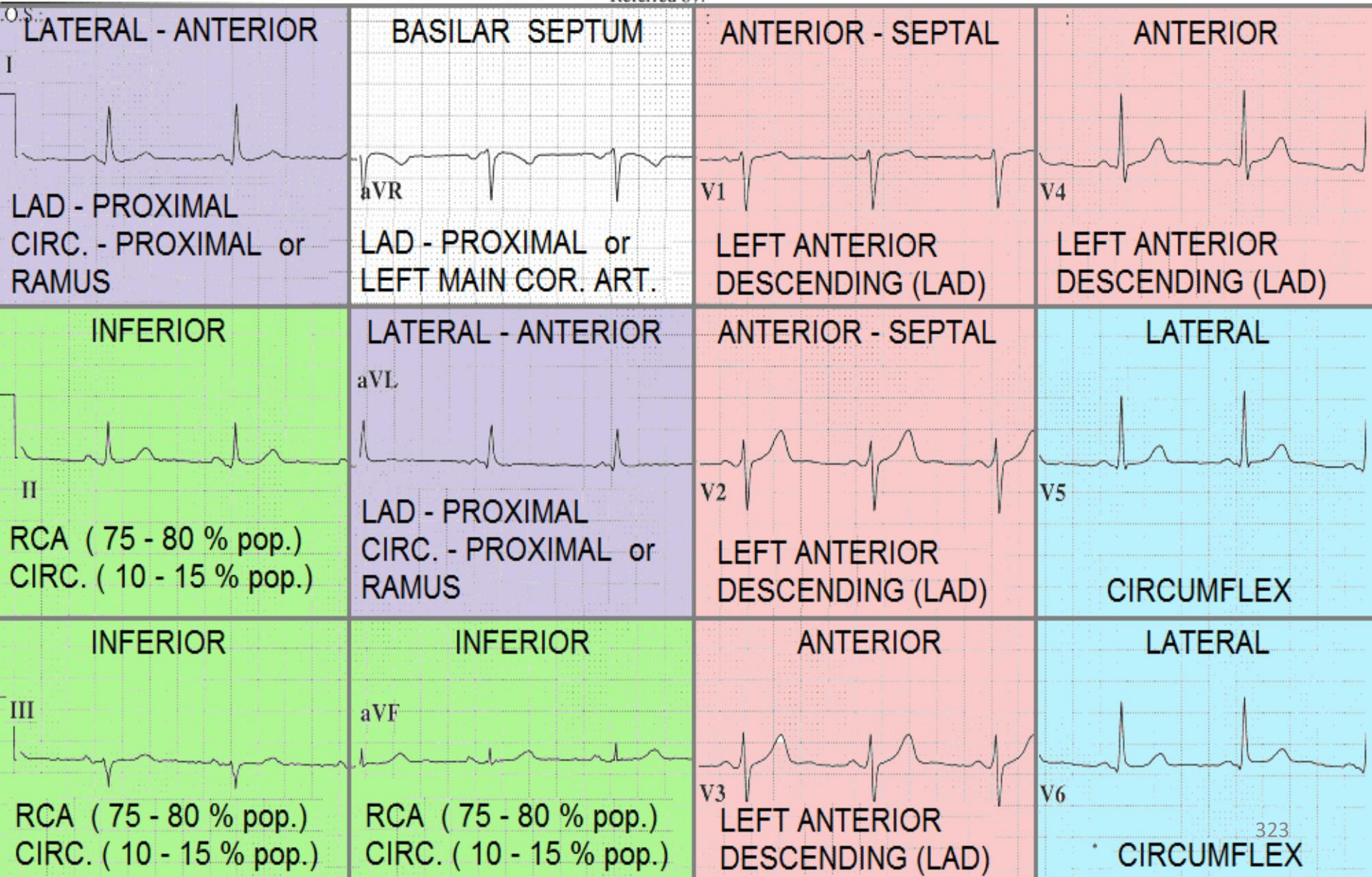
- DOMINANT CX:

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

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:

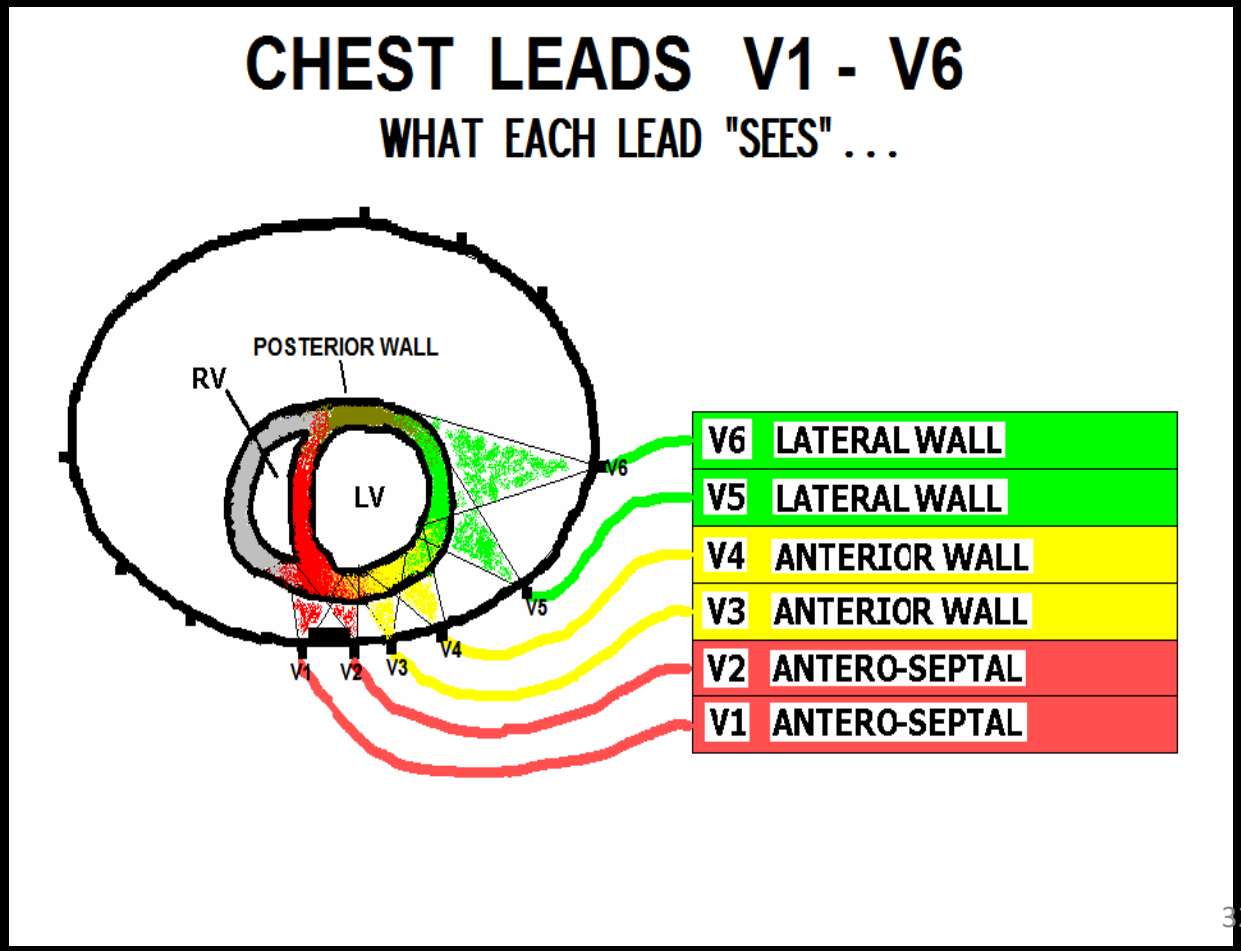


The 12 Lead ECG
Has TWO major BLIND SPOTS . . . . .

The POSTERIOR WALL

&

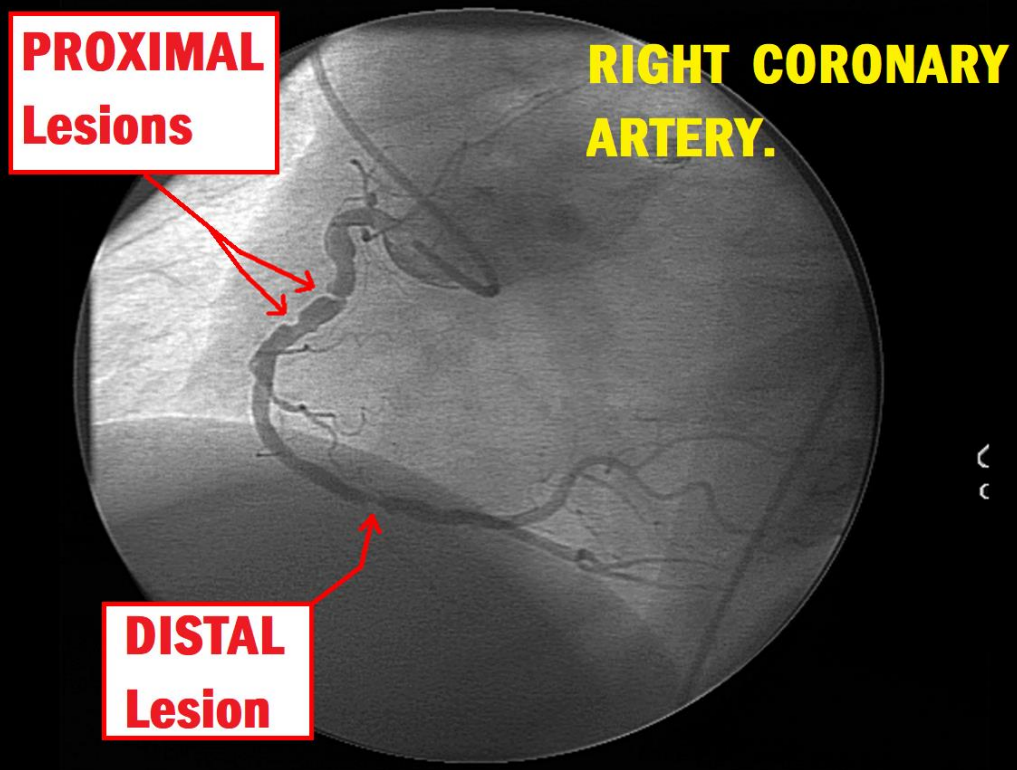
RIGHT VENTRICLE

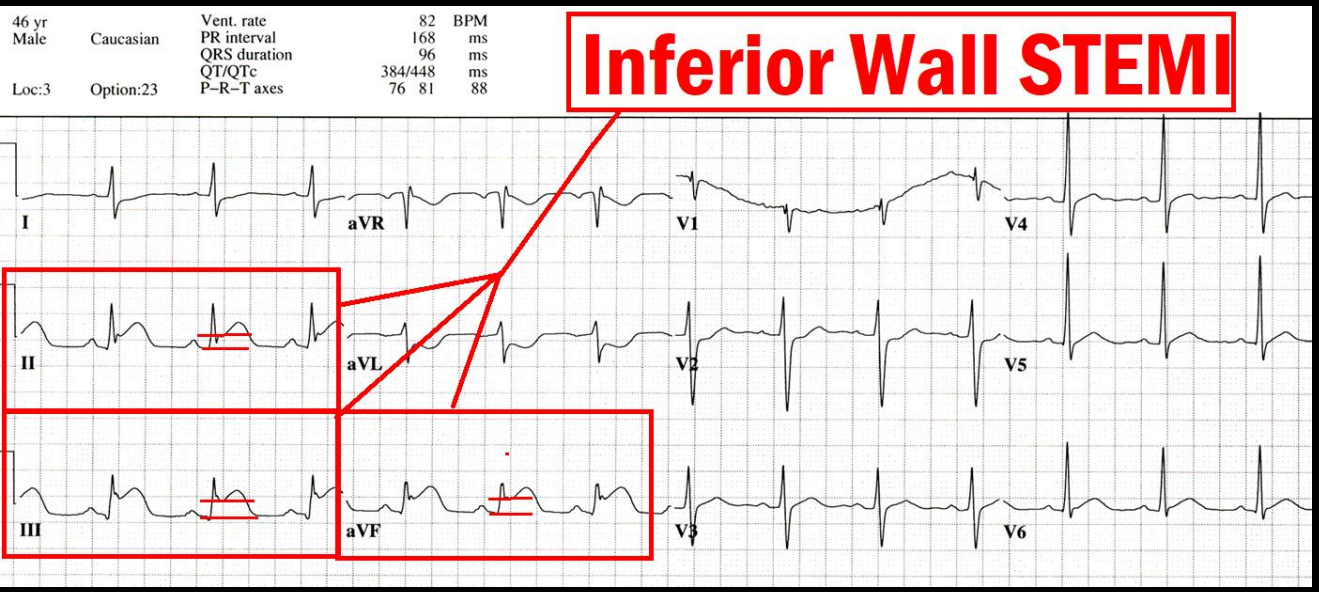


When do we need to see the Right Ventricle?

- 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.

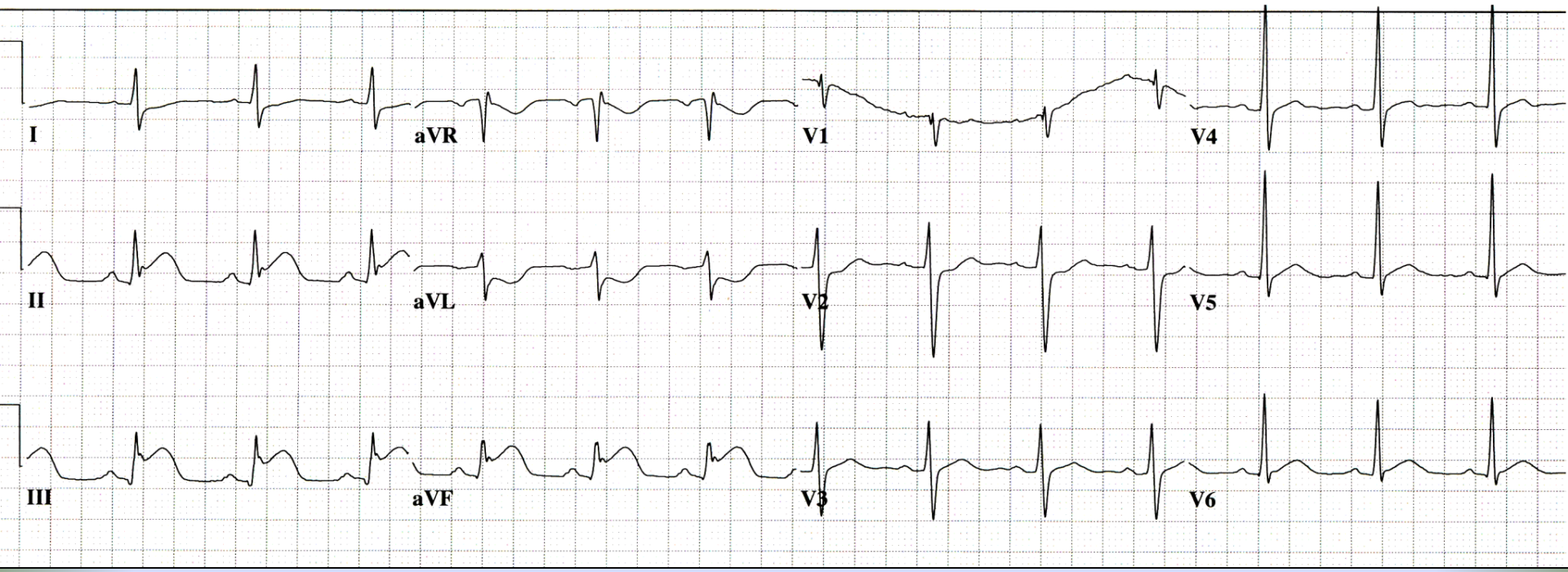
☞ You must do a

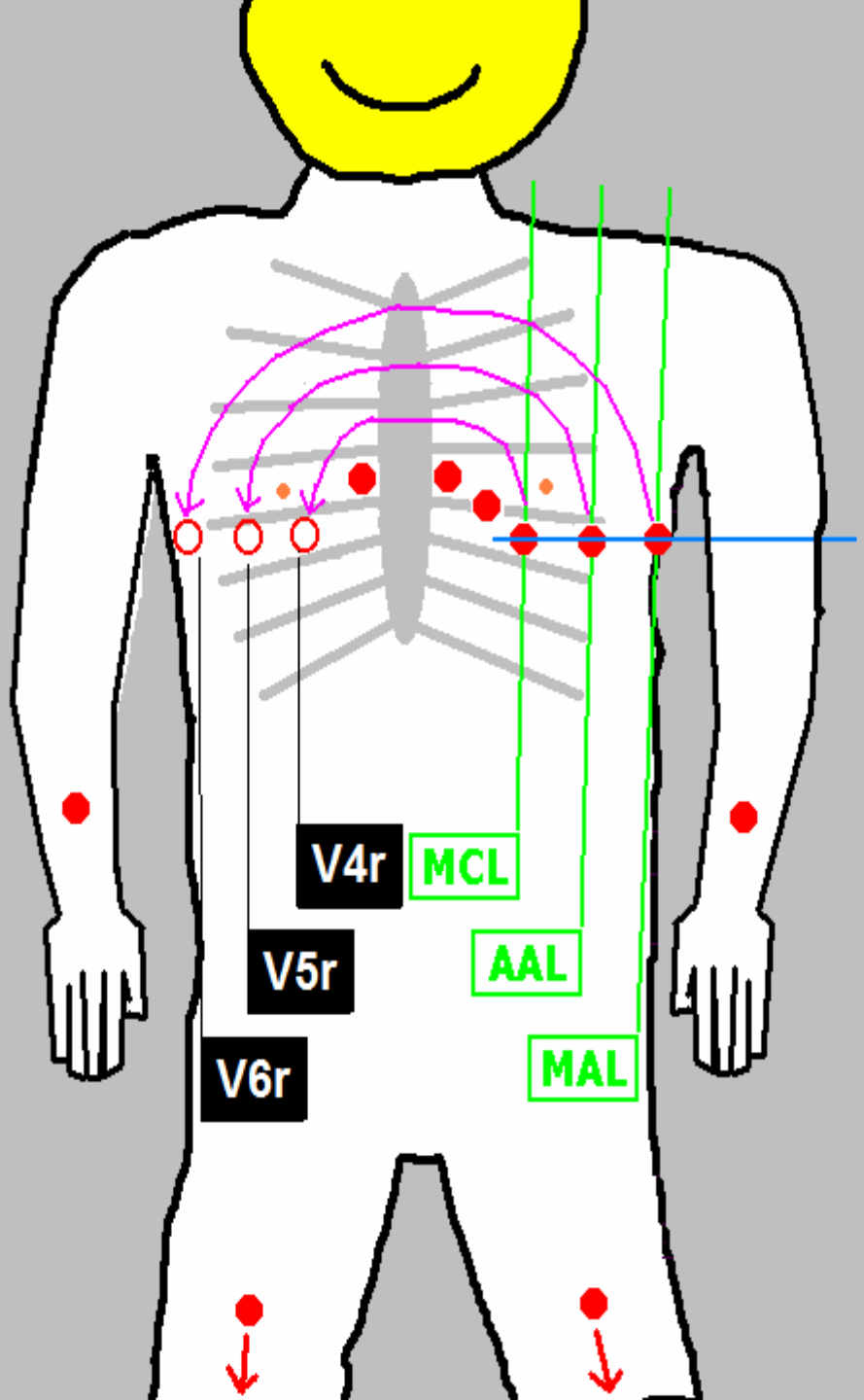
RIGHT - SIDED EKG ! !

46 yr		Vent. rate	82	BPM
Male	Caucasian	PR interval	168	ms
		QRS duration	96	ms
		QT/QTc	384/448	ms
Loc:3	Option:23	P–R–T axes	76 81	88

EVALUATE EKG for indicators of ACS:

- **ST SEGMENT ELEVATION / DEPRESSION**
- **HYPERACUTE T WAVES**
- **CONVEX ST SEGMENTS**
- **OTHER ST SEGMENT / T WAVE ABNORMALITIES**



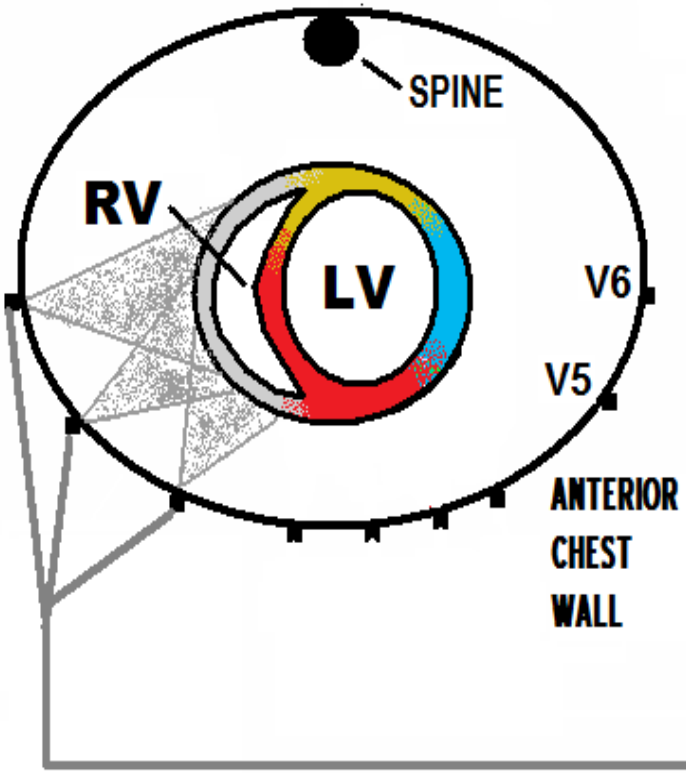


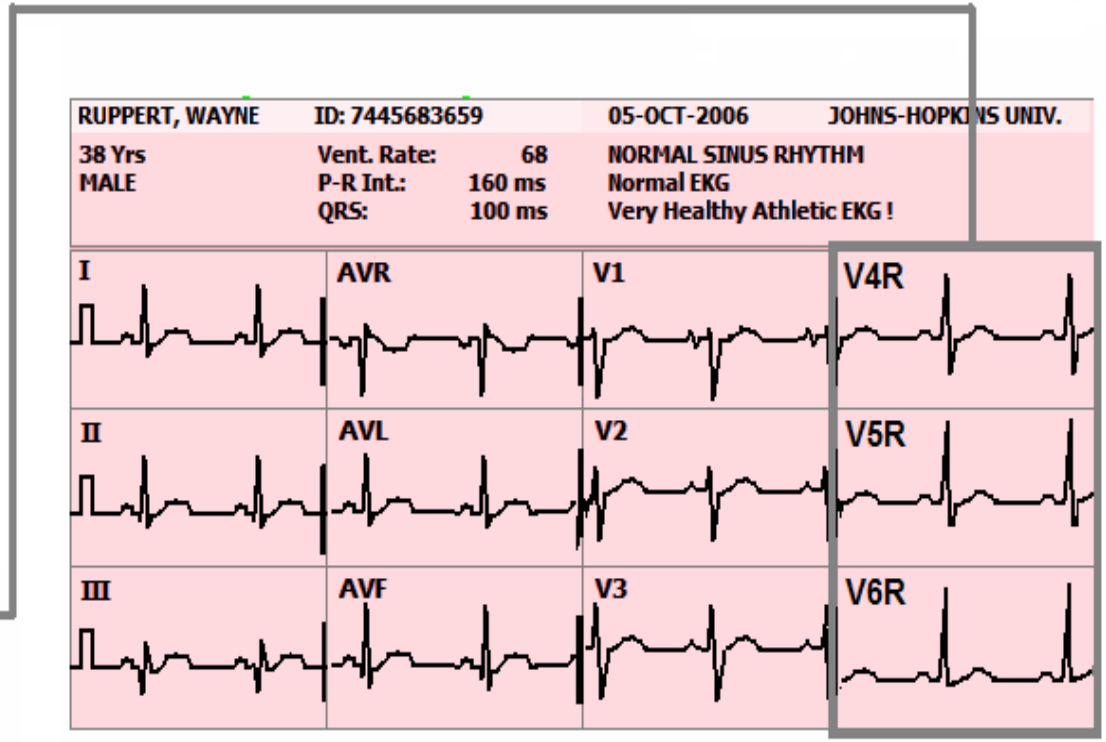
To do a
RIGHT - SIDED EKG . .

MOVE leads
V4, V5, and V6

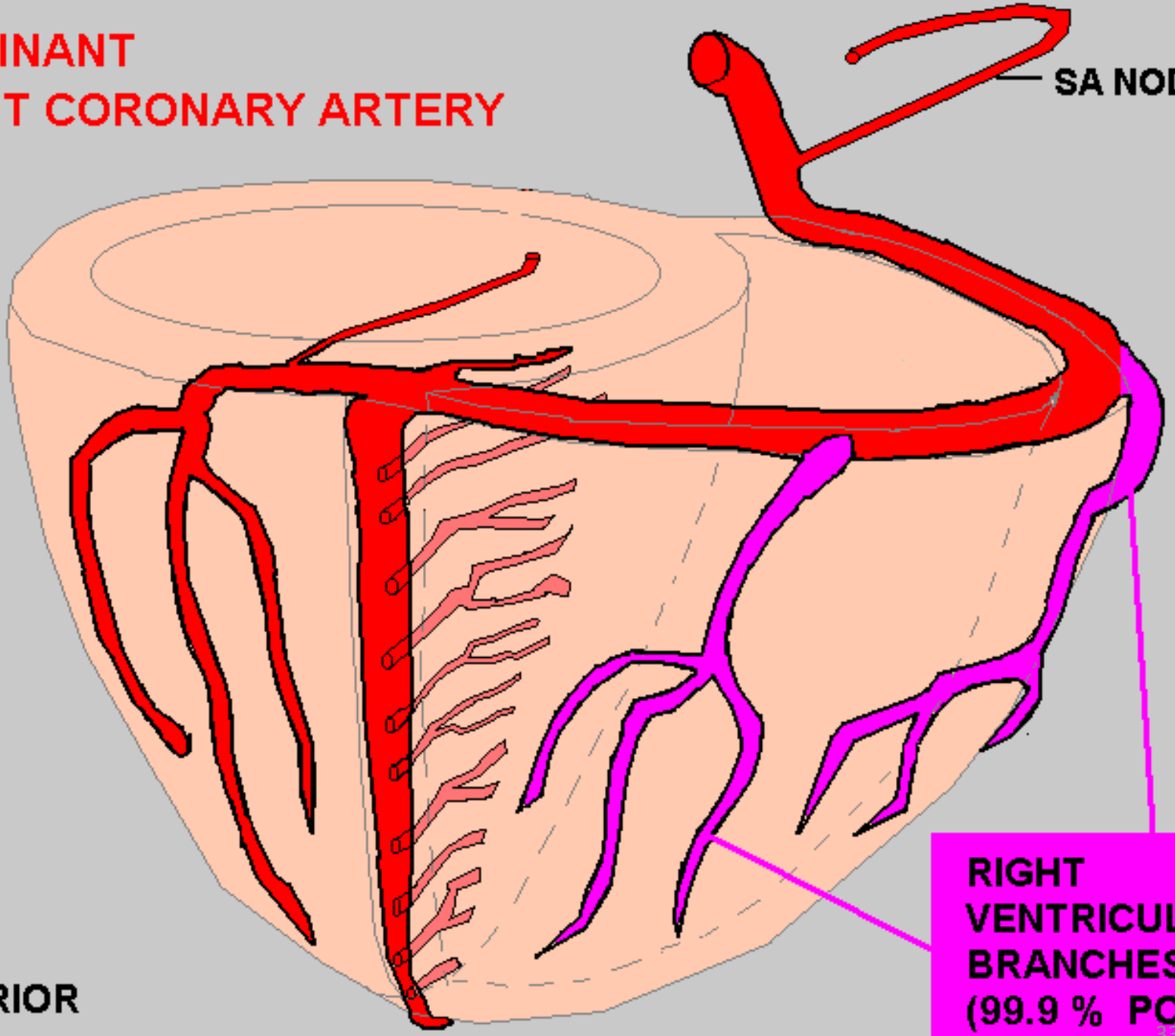
to the corresponding placement on the RIGHT SIDE of patient's chest . . .

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

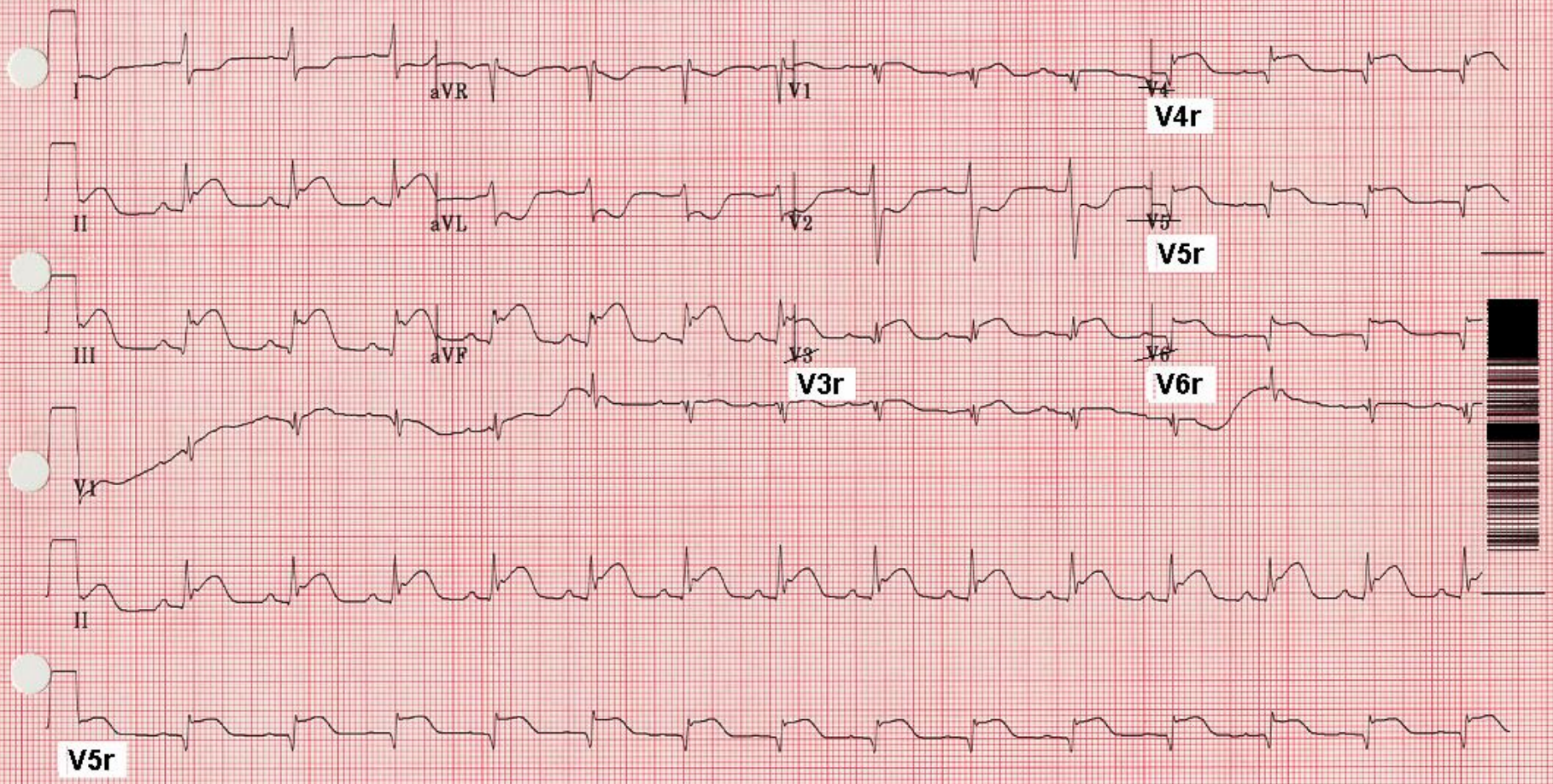
Normal sinus rhythm
~~Anterolateral infarct, possibly acute~~
Inferior injury pattern
** ** ** ** * Acute MI * ** ** ** **
Abnormal ECG

Right Ventricular Infarct

V LEADS
R SIDE

Referred by:

Unconfirmed



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

When do we need to see the Posterior Wall?

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.**

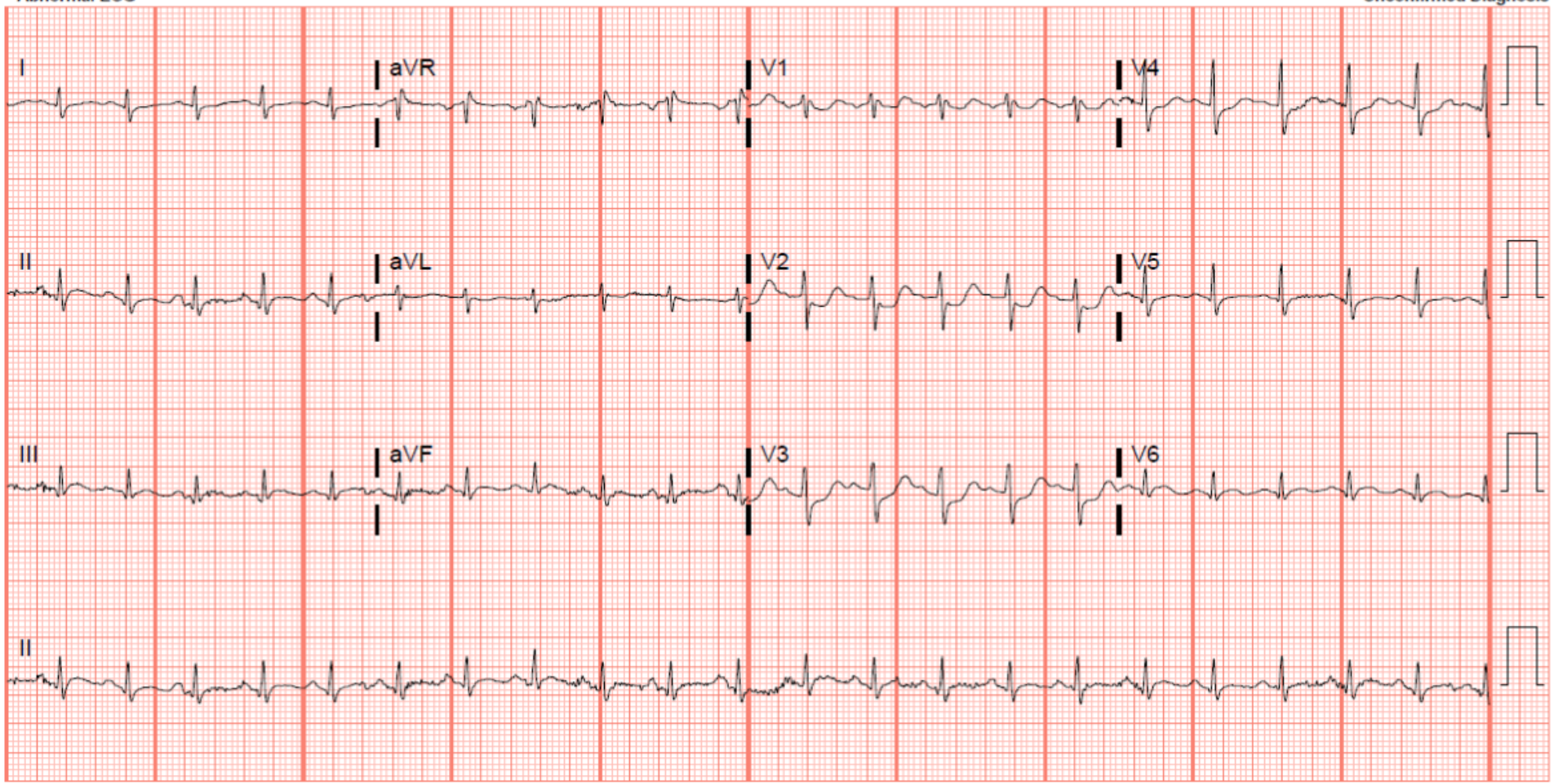
Pat ID [redacted]
[redacted]2019 22:07:54
[redacted] 46 yrs
[redacted]
Caucasian Female
Account # [redacted]
Bayfront Health Seven Rivers ED
Dept ED
Room [redacted]
Tech LDC
RX
DX

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

Whenever you see

ST DEPRESSION in Leads V1 - V4

you must do a

POSTERIOR LEAD ECG

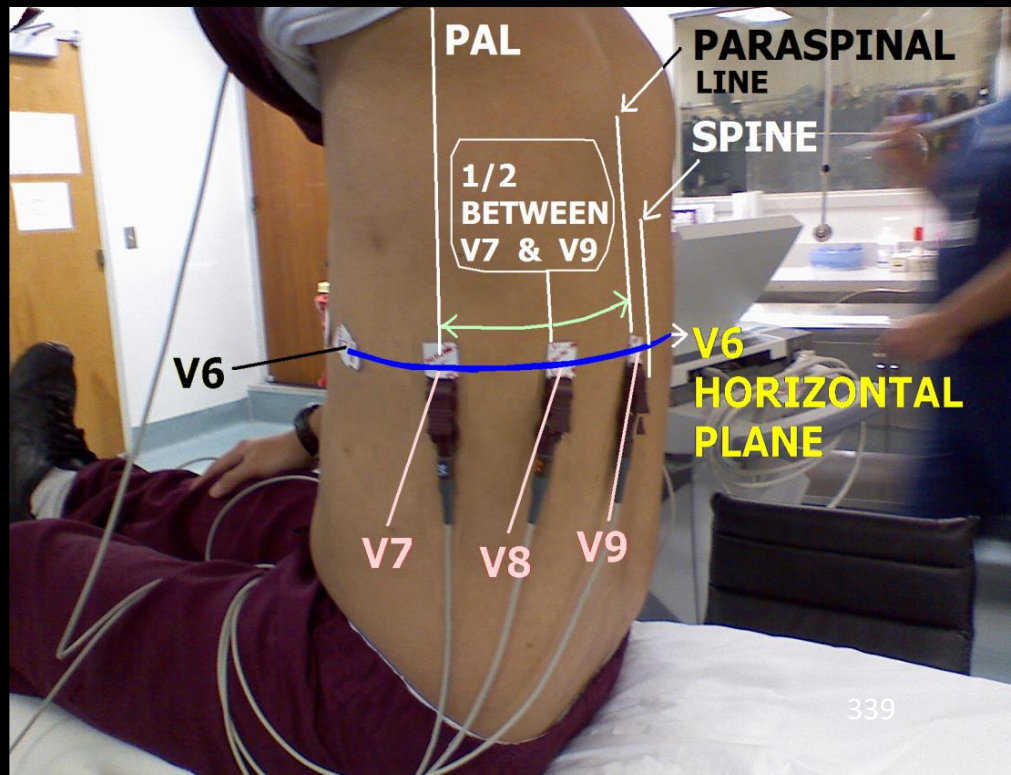
(V7 - V9)

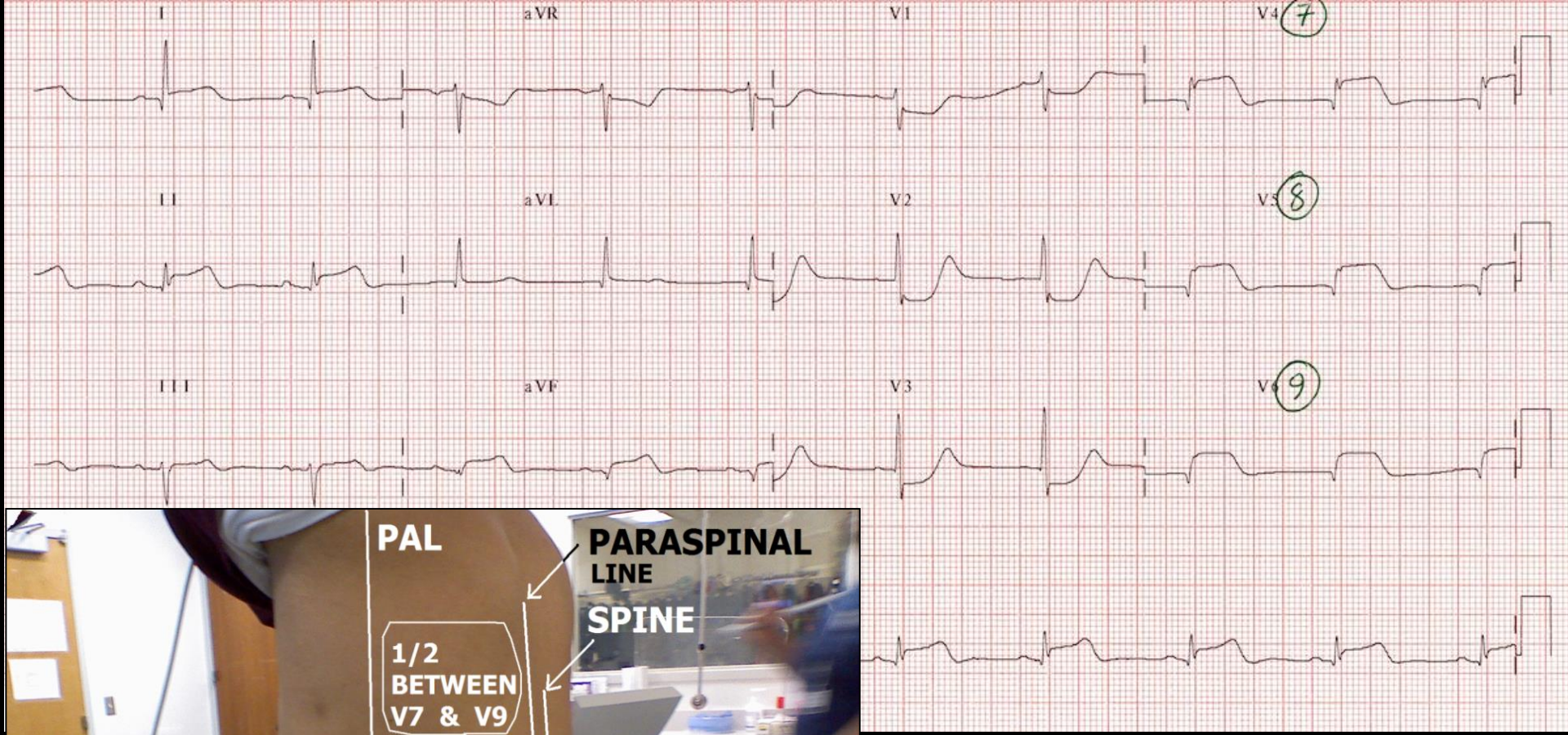
to see if you Patient is having a

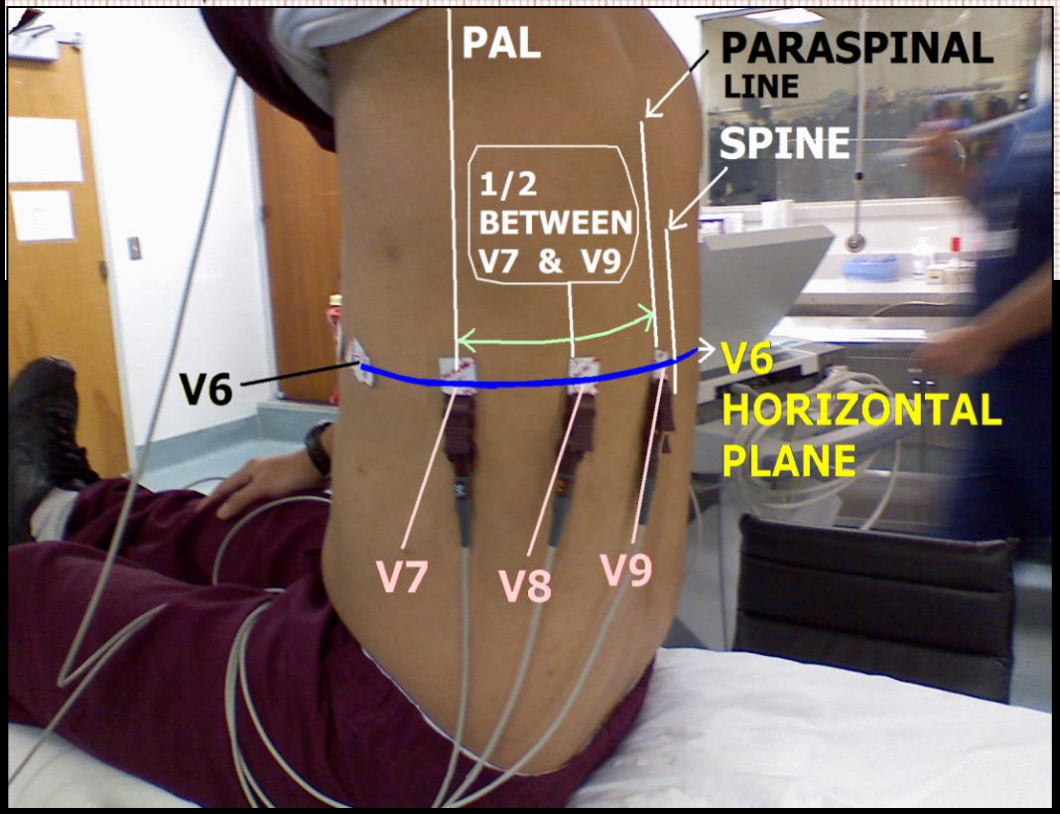
POSTERIOR WALL STEMI

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

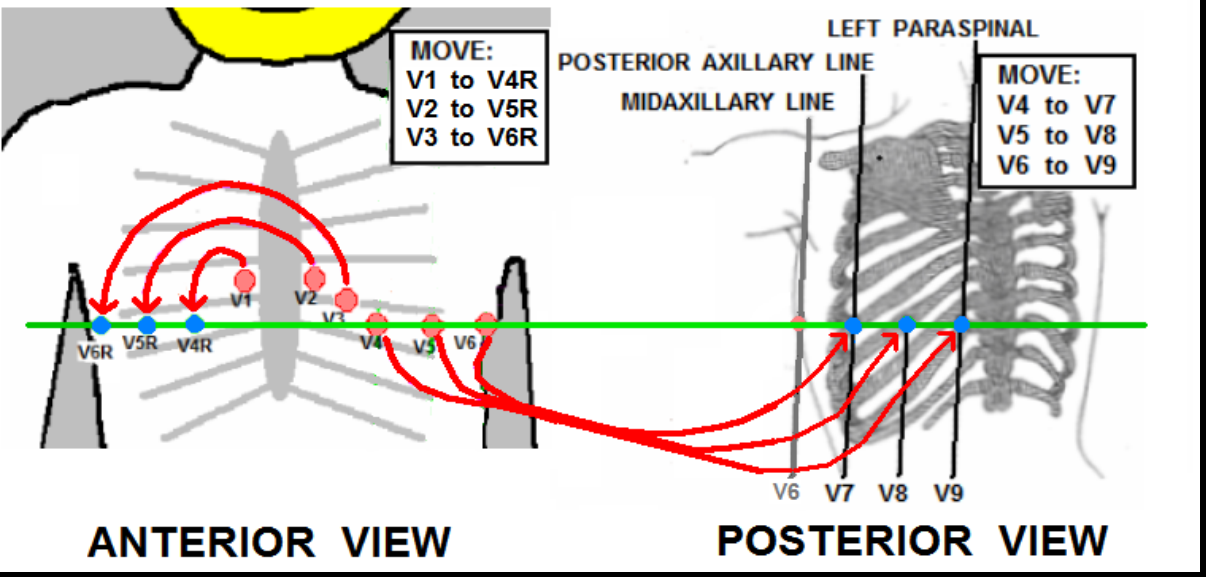
. . . To DIGANOSE Posterior Wall STEMI, we should see LEADS V7 – V9 !!




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

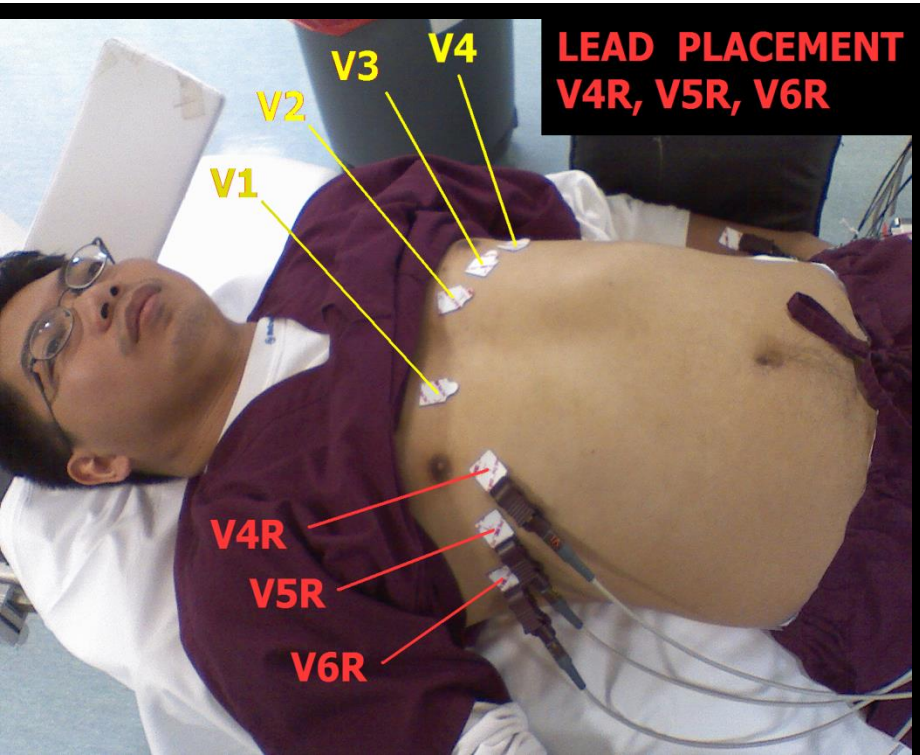

PAL
PARASPINAL
LINE
SPINE
1/2
BETWEEN
V7 & V9
V6
V6
HORIZONTAL
PLANE
V7
V8
V9

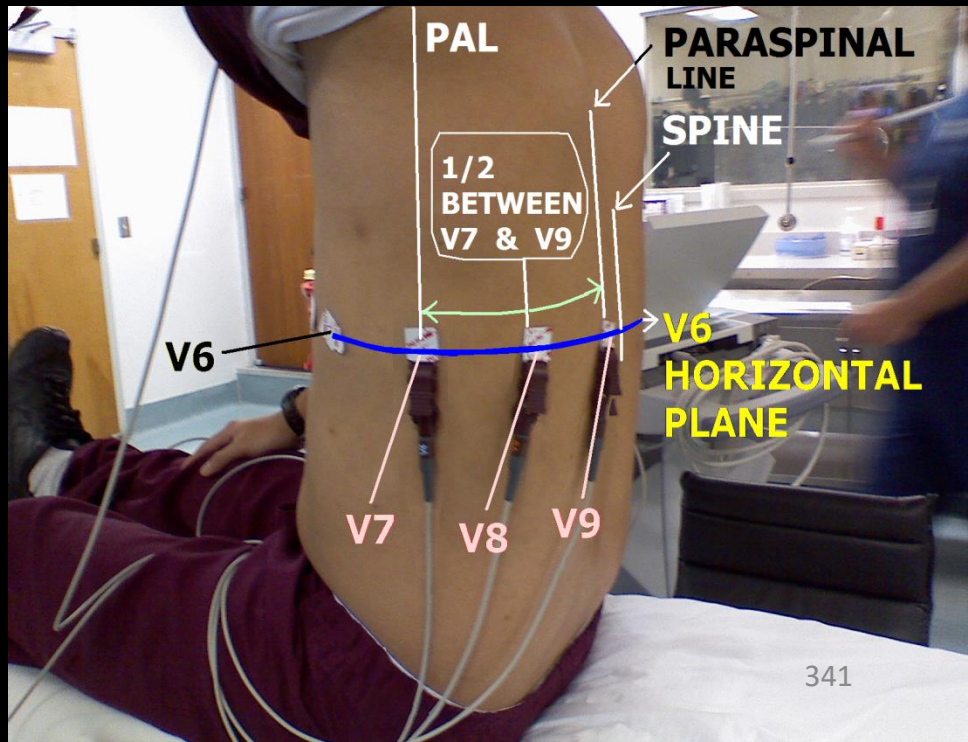
HOW TO REPOSITION 6 CHEST LEADS to OBTAIN 3 R VENTRICLE and 3 POSTERIOR LEADS

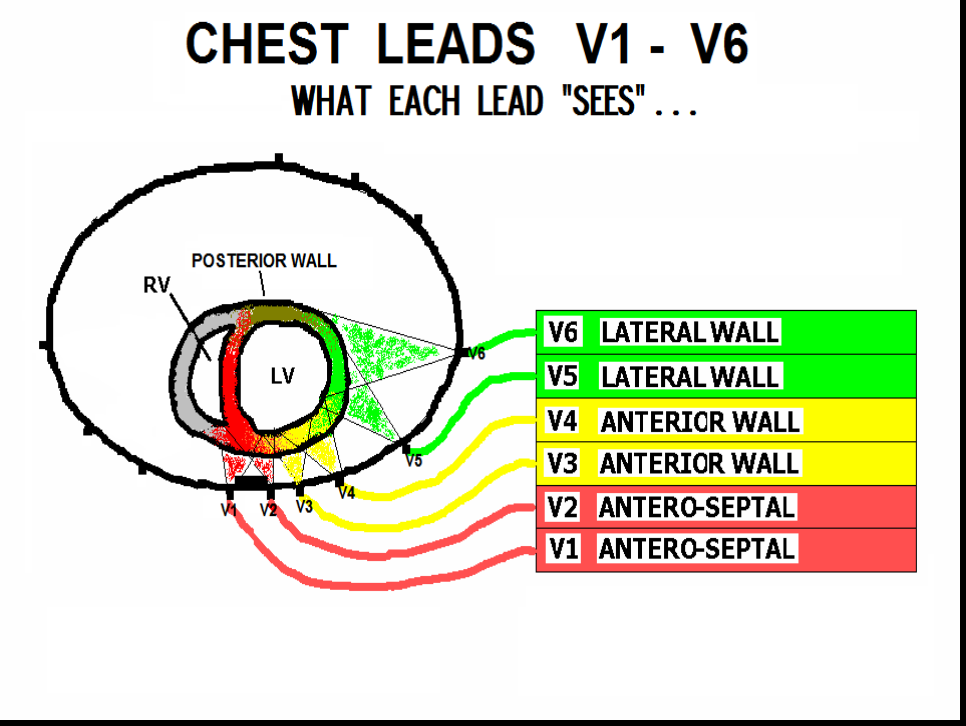


ANTERIOR VIEW

POSTERIOR VIEW

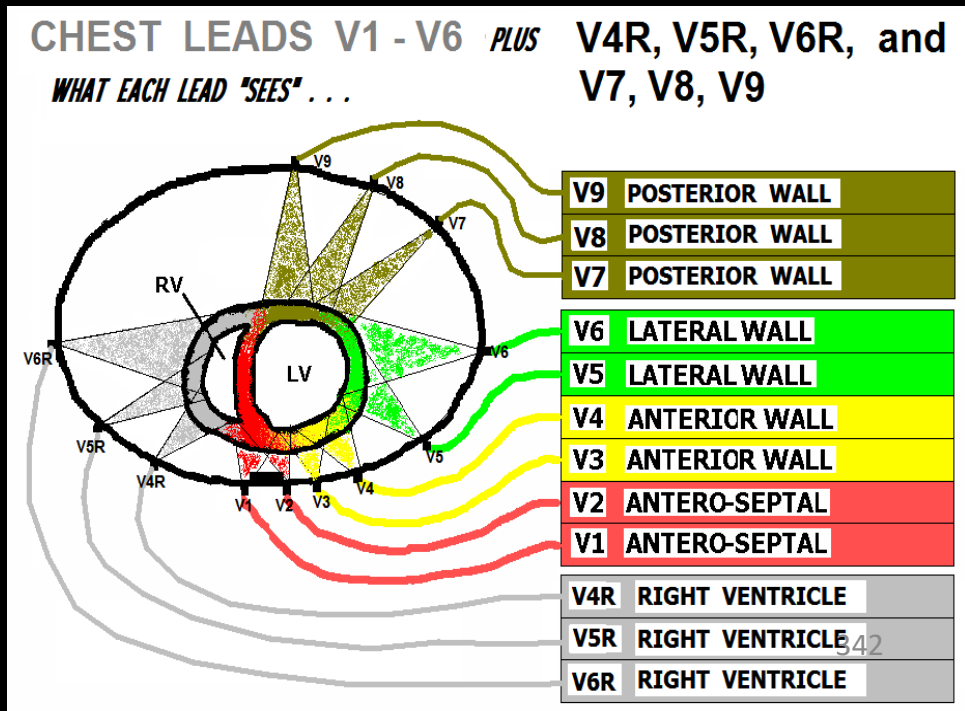






⇦ The 12 Lead ECG

The 18 Lead ECG ⇨



34years
Male Asian

Room:

Opt:

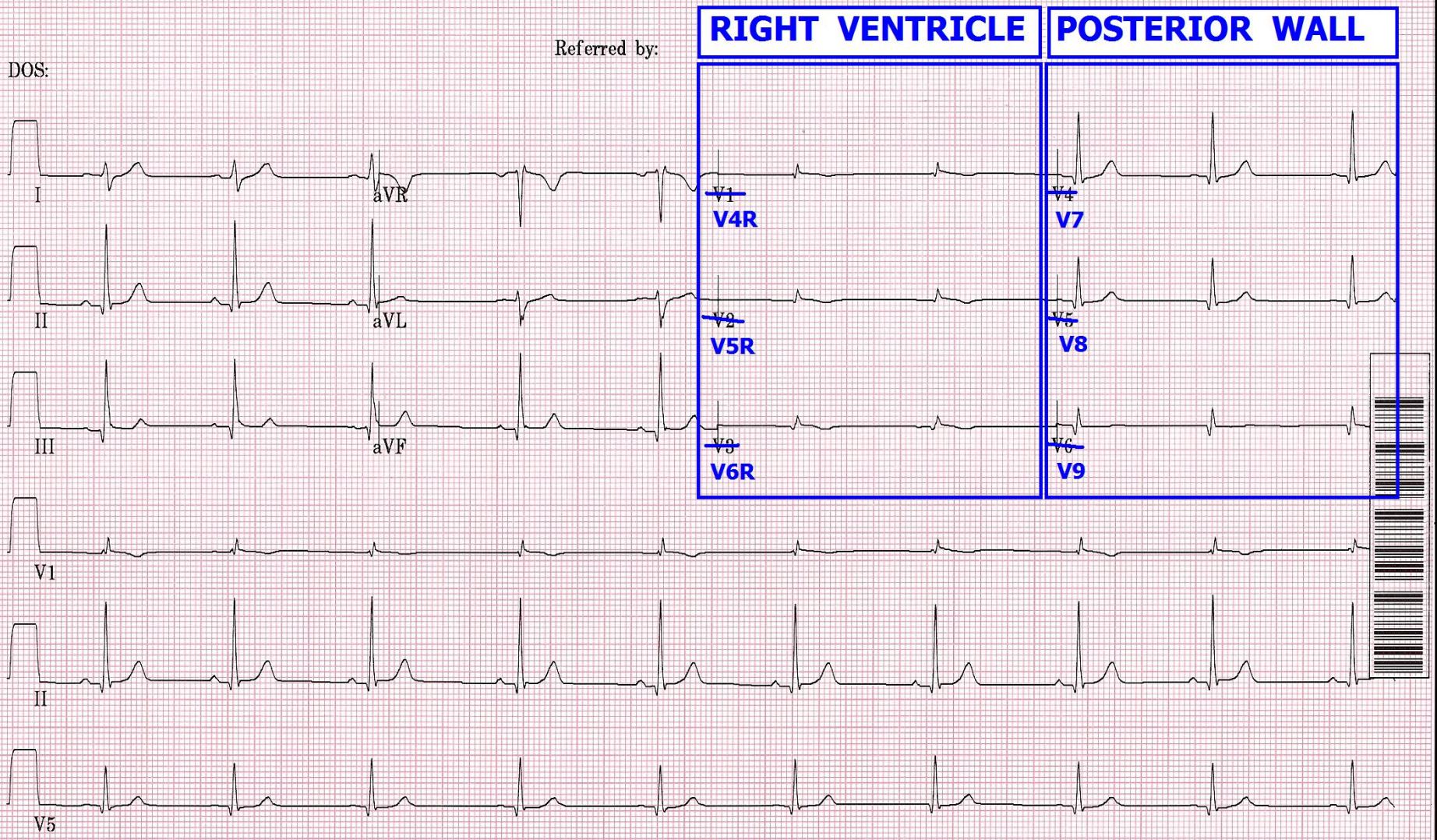
Technician: WR

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 !!!

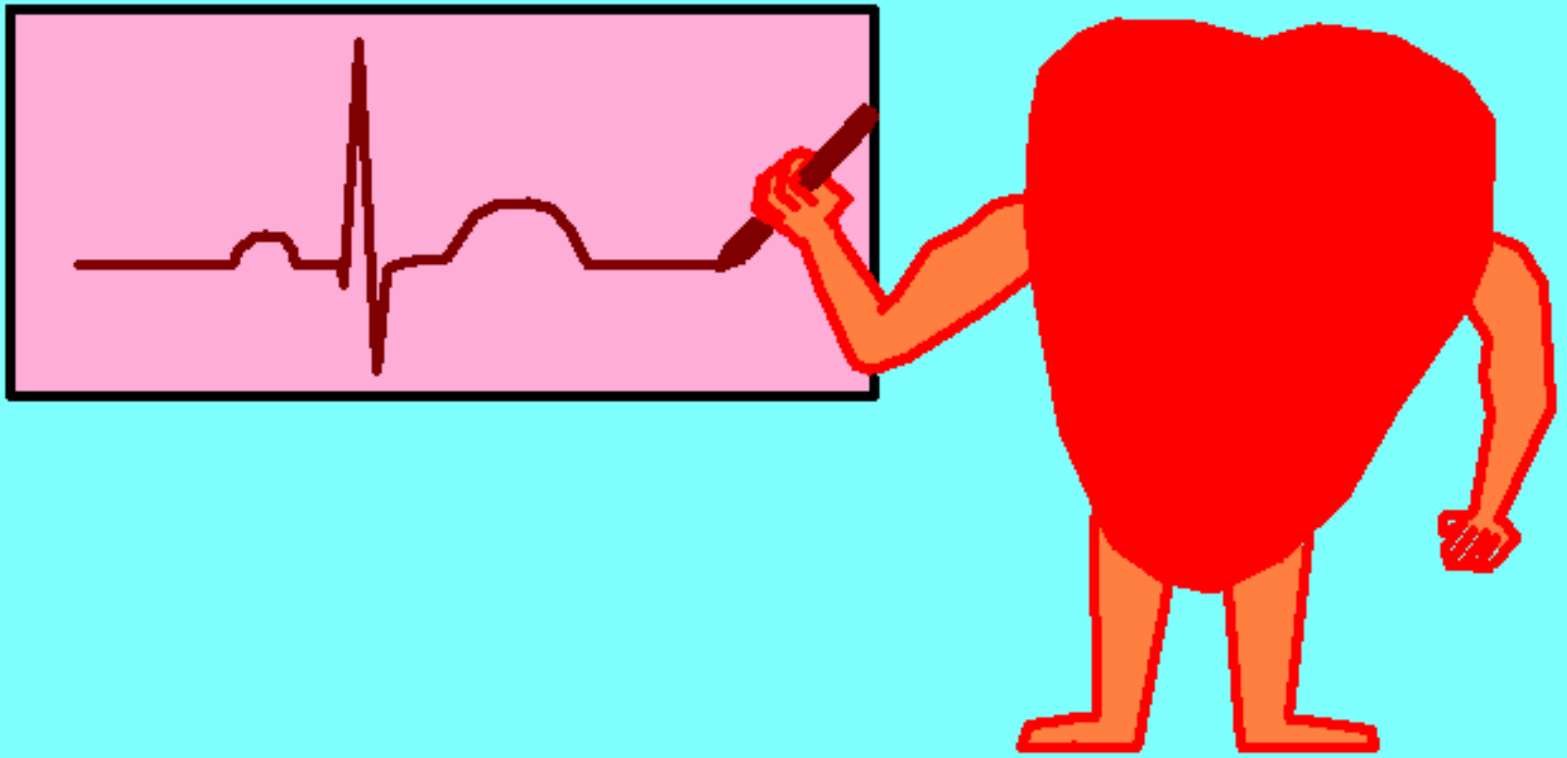
POSTERIOR WALL MI usually accompanies INFERIOR and/or LATERAL WALL MI !!!

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

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

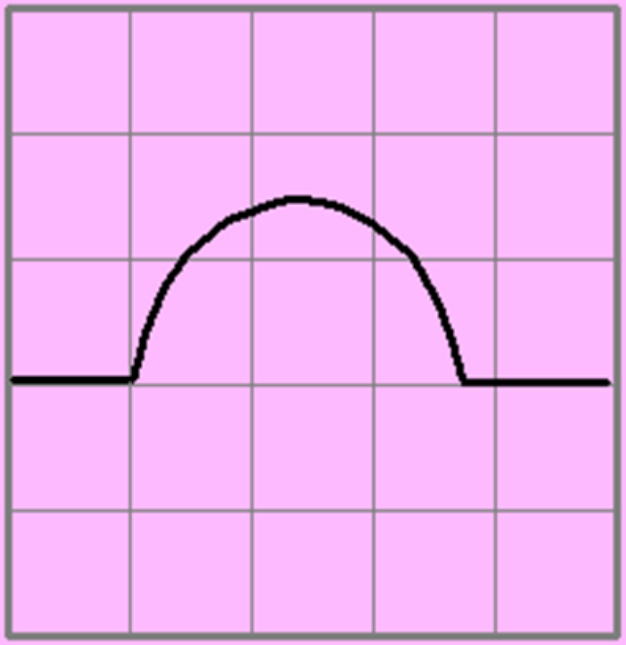
PUTTING IT ALL ON PAPER . . .

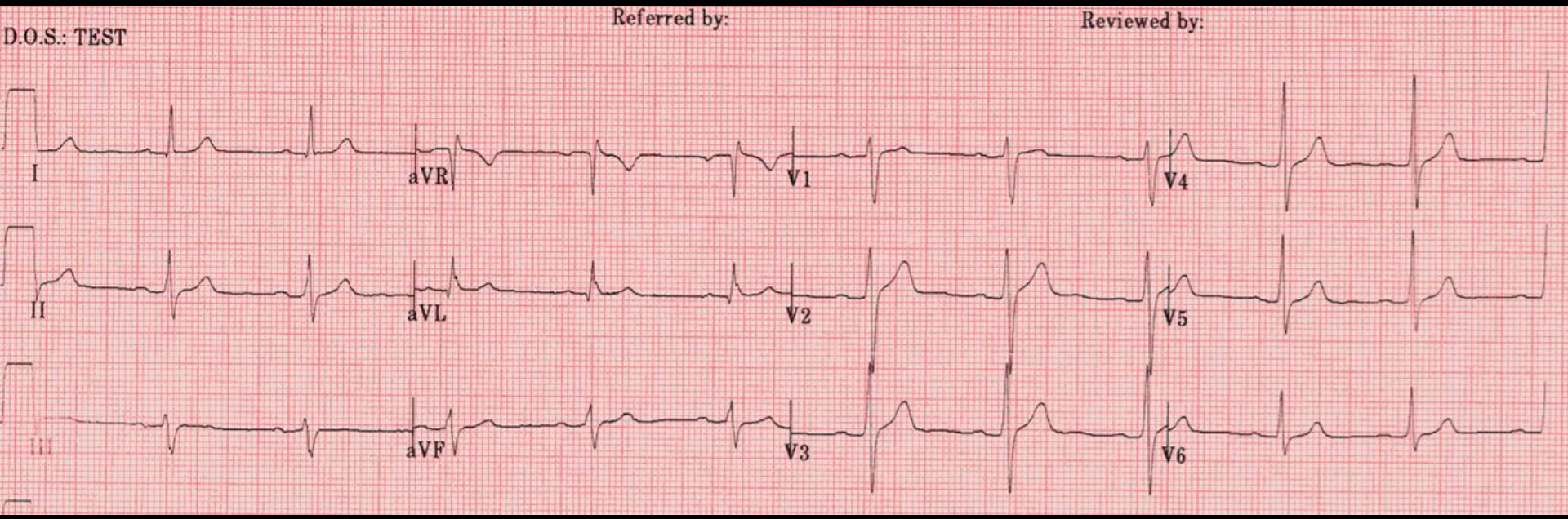
WAVEFORMS and INTERVALS . . .



THE P WAVE

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

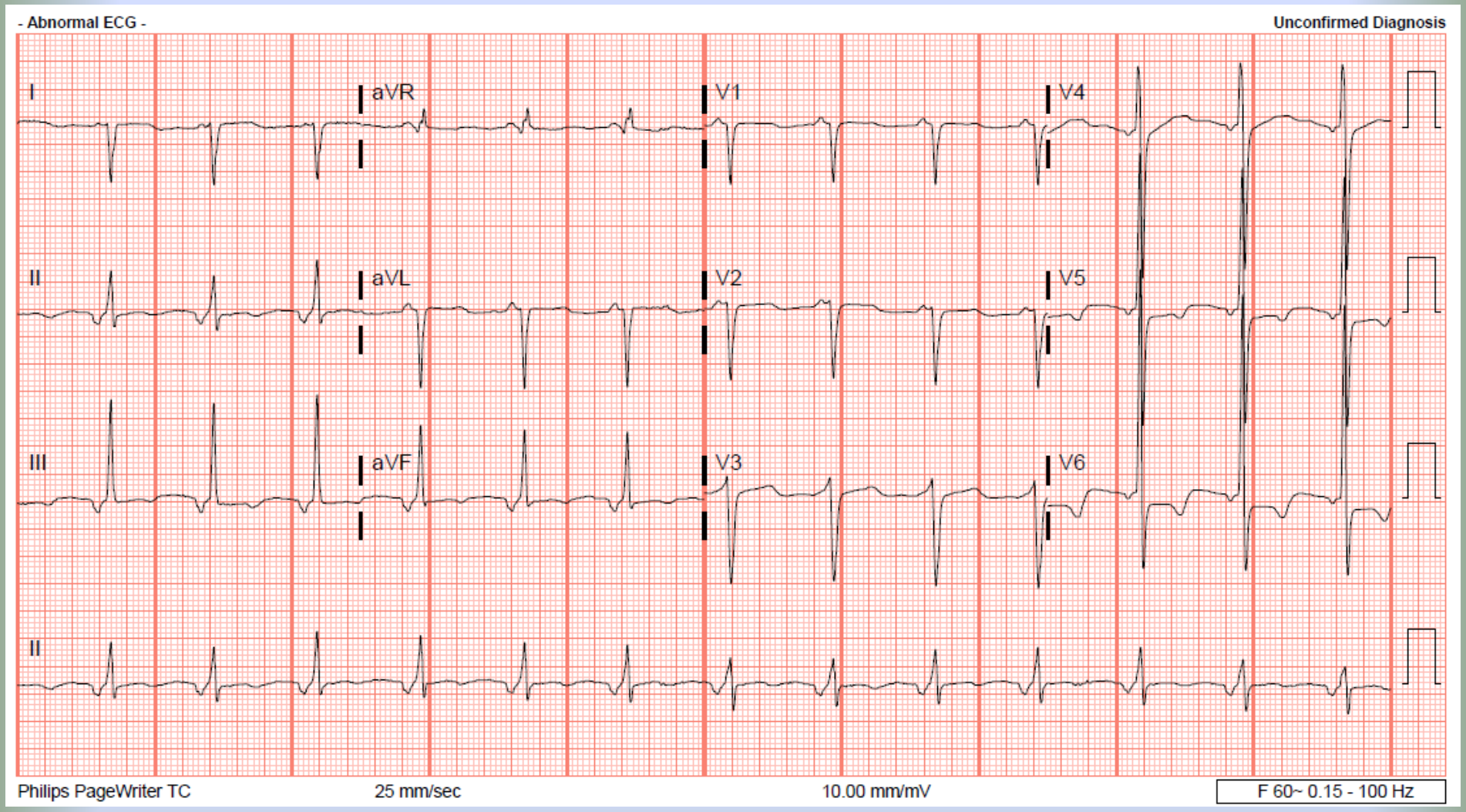



D.O.S.: TEST
Referred by:
Reviewed by:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

P Wave Axis

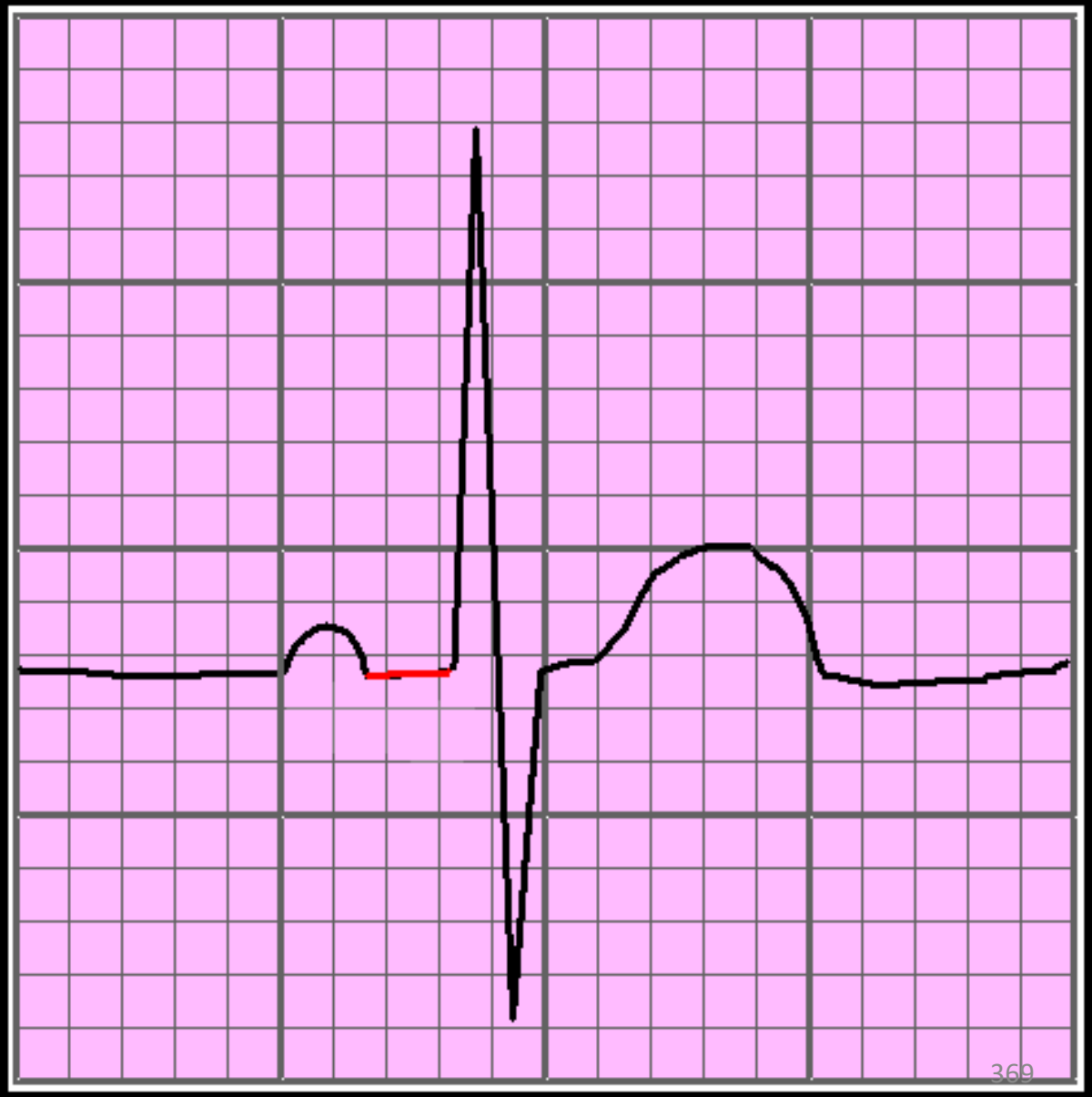
- P waves with abnormal axis (“not pointing in the right direction”) may signify ectopic atrial beats.
- When P waves are inverted in most leads with an abnormally short P-R interval (<120ms) the origin of the rhythm may be the AV node (Junctional Rhythm).

Inverted P waves & short P-R interval:



THE P-R SEGMENT

SHOULD RETURN TO THE ISO-ELECTRIC LINE.

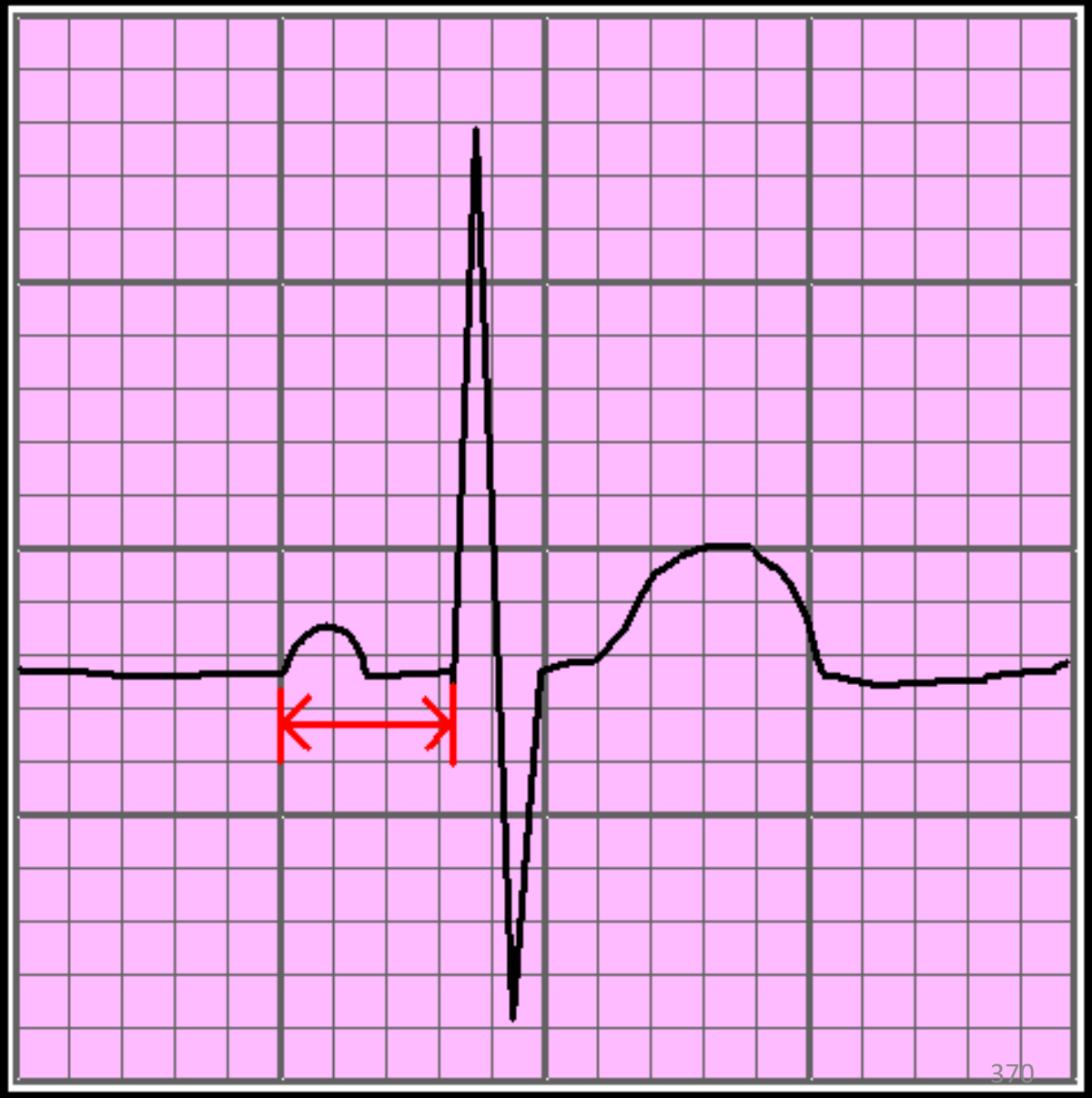


NORMAL P-R INTERVAL

.12 - .20 SEC

or

120 - 200 mSEC



P - R INTERVAL TOO SHORT . . .

LESS THAN 120 mSEC

THINK:

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

P - R INTERVAL TOO LONG

GREATER THAN 200 mSEC

THINK:

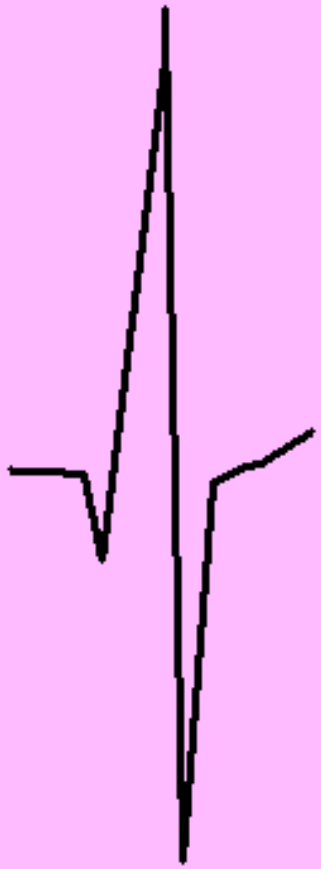
- HEART BLOCK

THE QRS COMPLEX

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

THE QRS COMPLEX

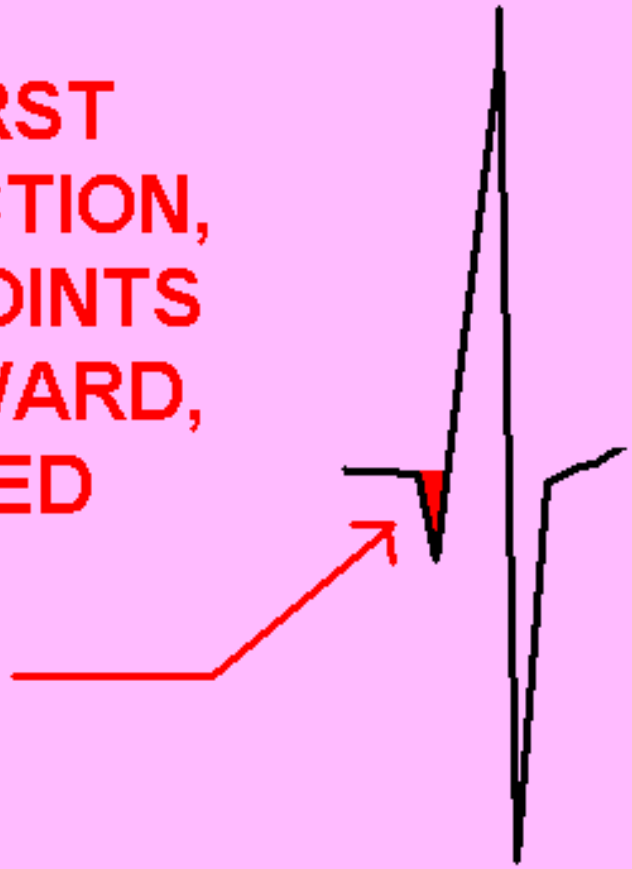
THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .



THE QRS COMPLEX

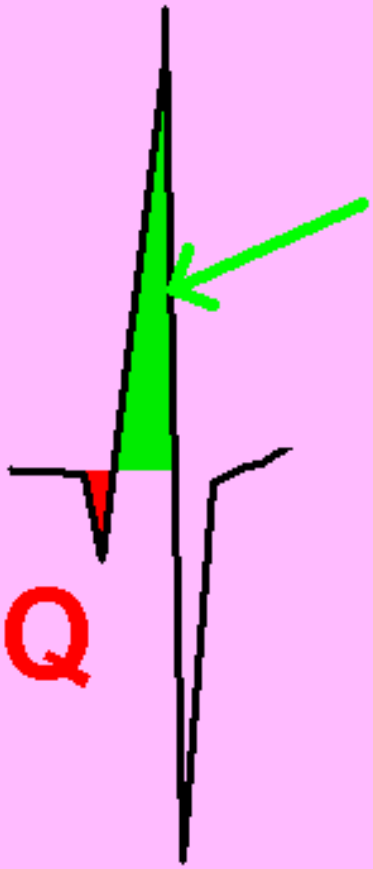
THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .

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



THE QRS COMPLEX

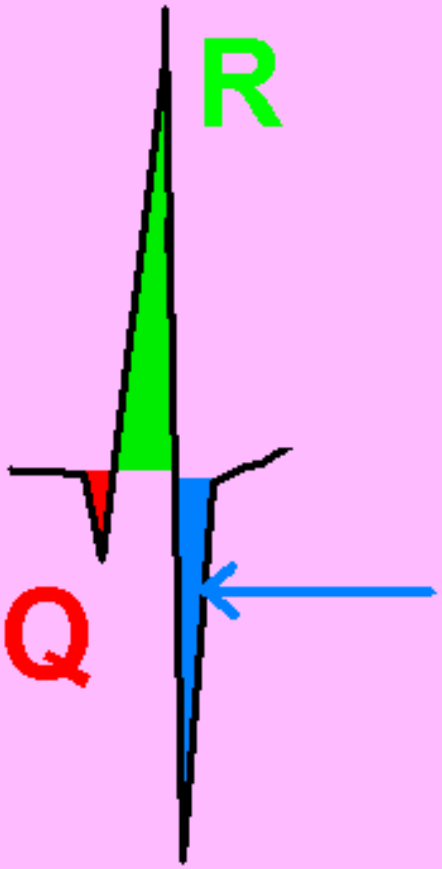
THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .



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

THE QRS COMPLEX

THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .

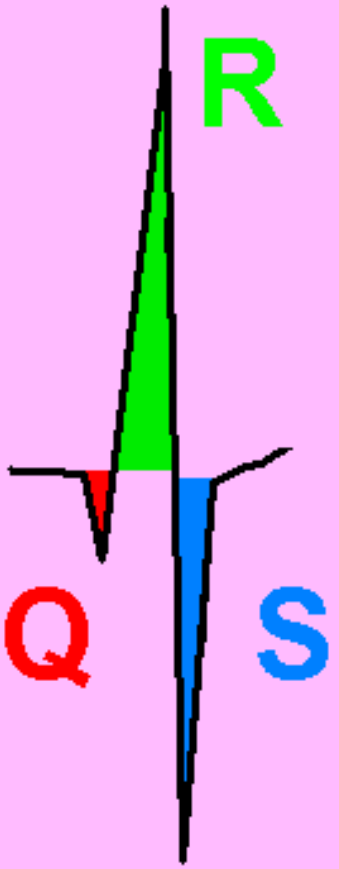


A NEGATIVE DEFLECTION AFTER THE R WAVE IS CALLED THE "S" WAVE

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'

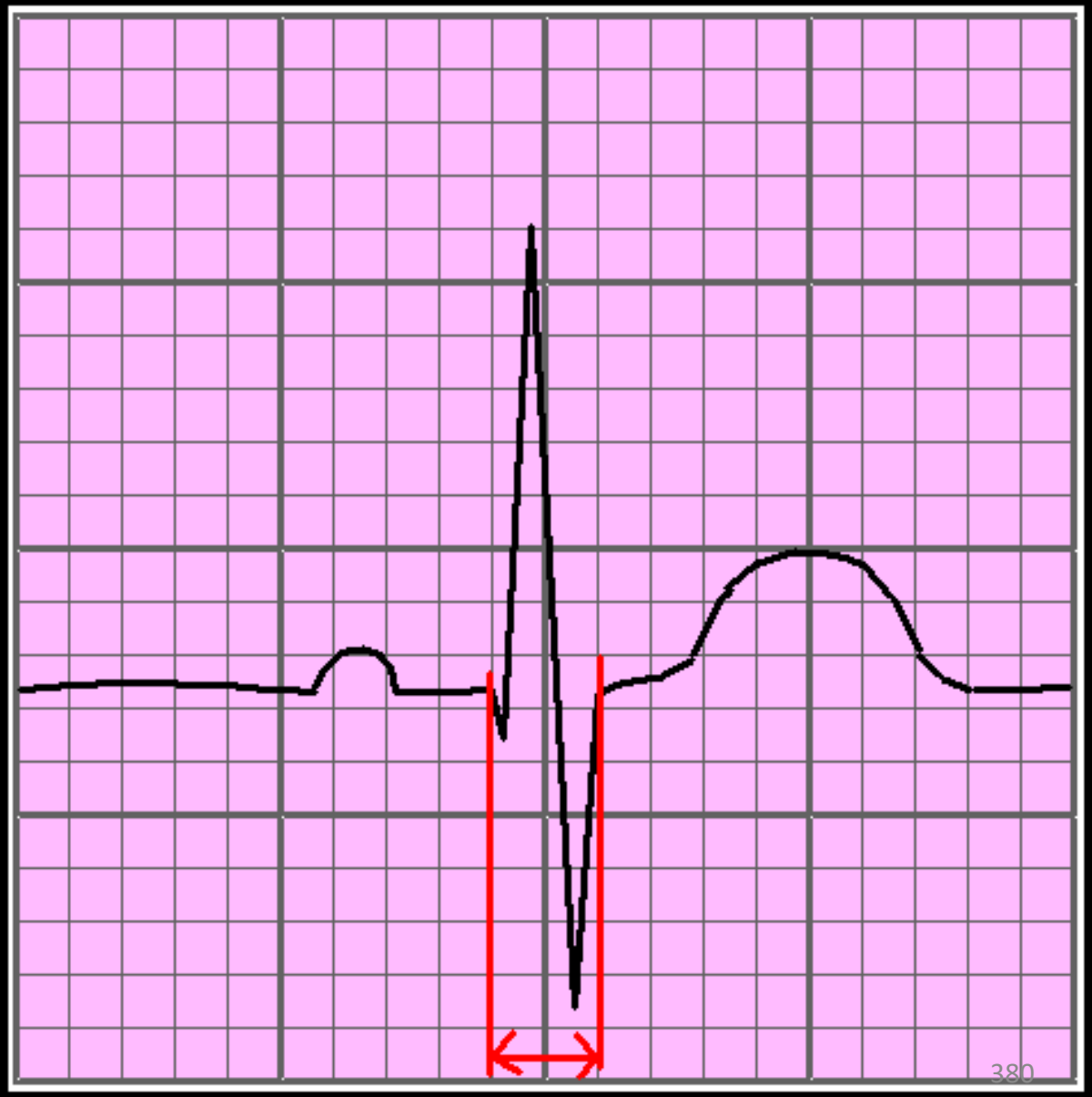
QRS INTERVAL

LESS THAN

.12

OR

120 mSEC



QRS COMPLEX TOO WIDE

WIDER THAN 120 mSEC

THINK:

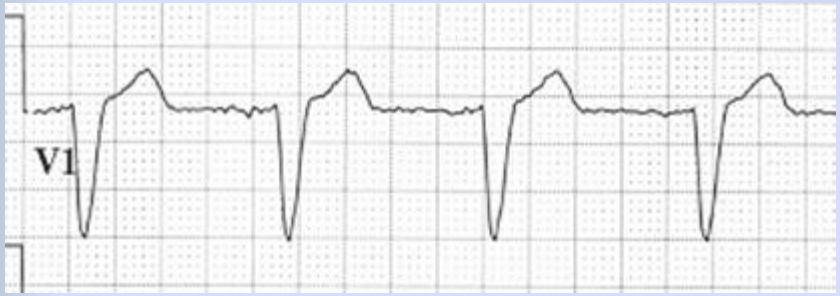
- BUNDLE BRANCH BLOCK
- VENTRICULAR COMPEX (ES)
- PACED RHYTHM
- L VENTRICULAR HYPERTROPHY
- ELECTROLYTE IMBAL. (↑K^+ ↓Ca^{++})
- DELTA WAVE (PRE-EXCITATION)

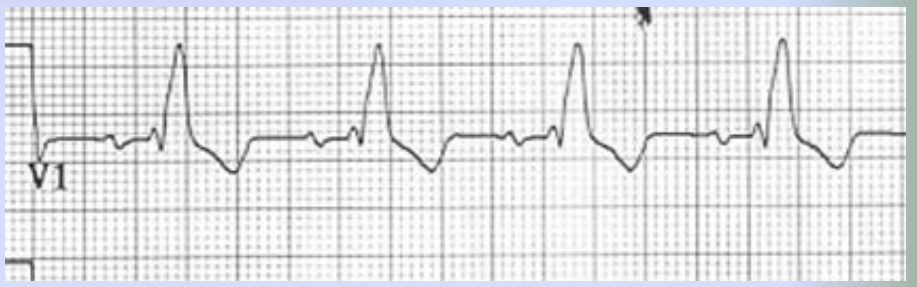
When the QRS is WIDE (> 3mm):

- **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)

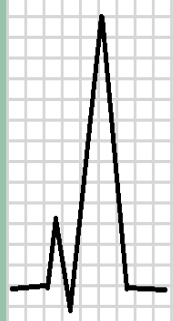
- **Determine LEFT vs. RIGHT Bundle Branch Block Pattern**





Simple “Turn Signal Method” . . .

THE "TURN SIGNAL METHOD" for identifying BUNDLE BRANCH BLOCK



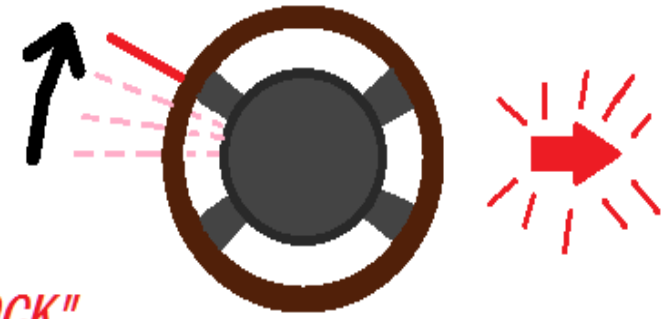
USE LEAD V1 for this technique

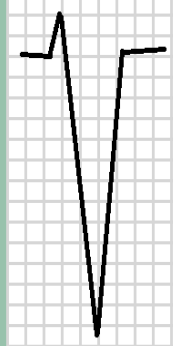
To make a **RIGHT TURN**

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

THINK:

"QRS points UP = RIGHT BUNDLE BRANCH BLOCK"



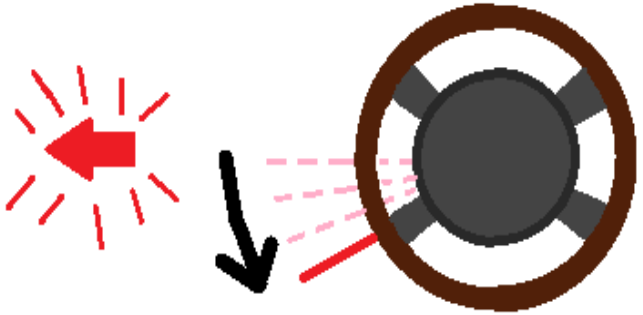


To make a **LEFT TURN**

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

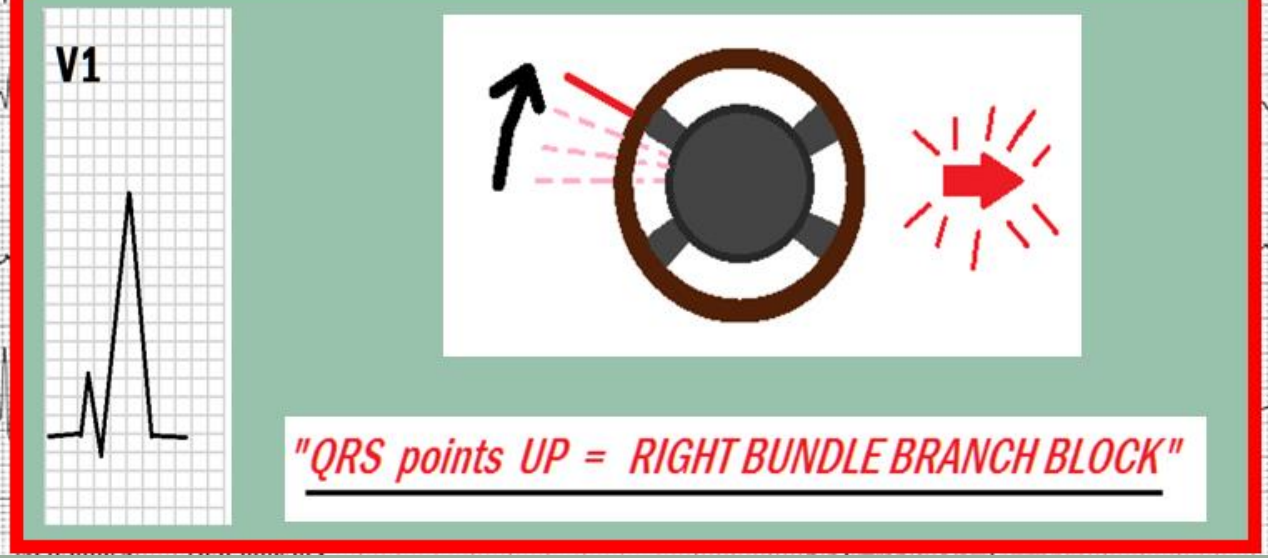
Vent. rate	72	bpm
PR interval	186	ms
QRS duration	166	ms
QT/QTc	436/477	ms
P-R-T axes	57 -32 32	

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 #WP03029959

Technician: WW

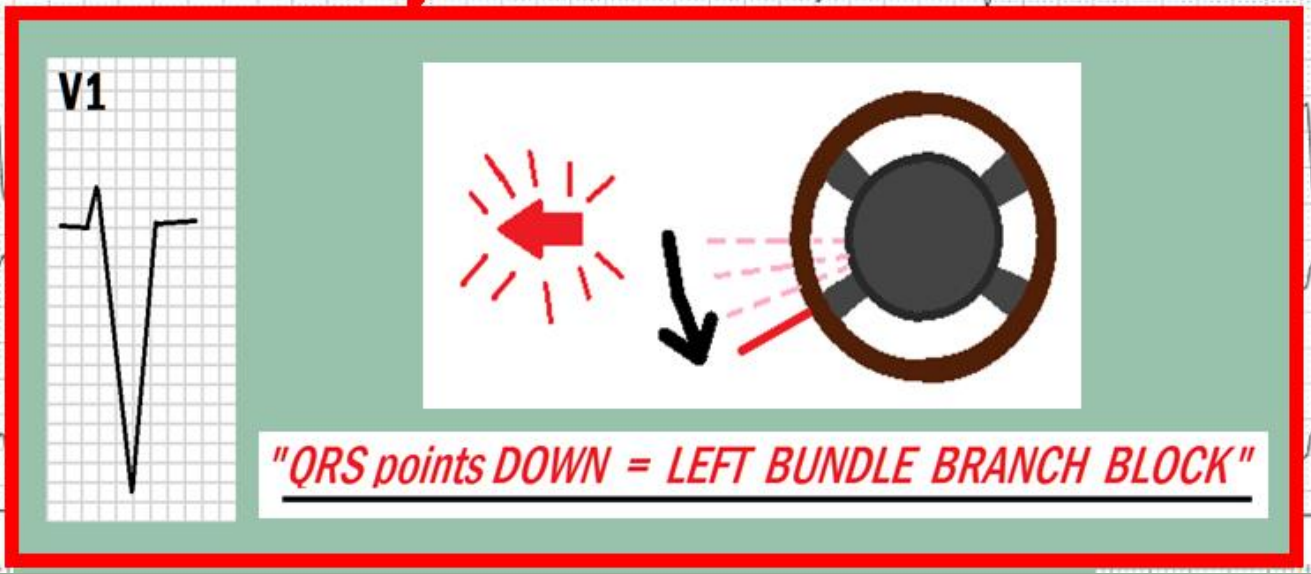
USE LEAD V1 for this technique

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

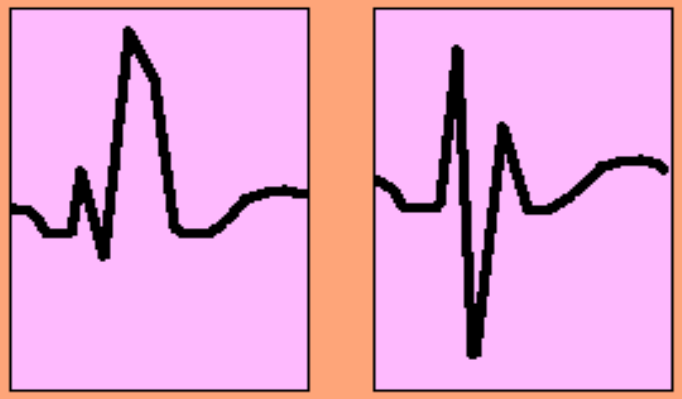


DIAGNOSING BUNDLE BRANCH BLOCK

USING LEADS V1, V2, and V5, V6:

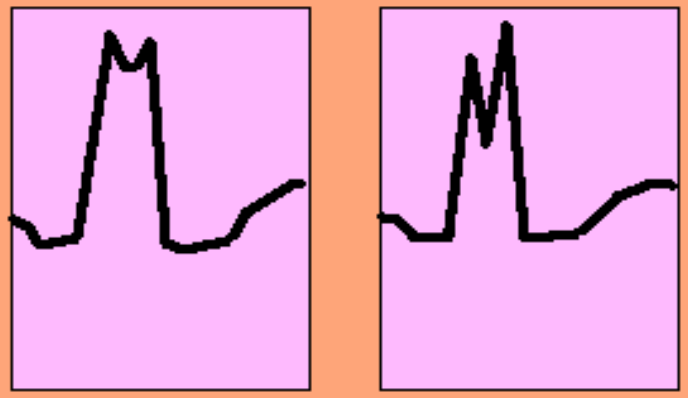
LOCATING RsR' or RR' COMPLEXES:

V1 V2



RIGHT BUNDLE BRANCH BLOCK

V5 V6



LEFT BUNDLE BRANCH BLOCK

From: “Rapid Interpretation of ECGs” by Dale Dubin, MD

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