Session I

The INTEGRATED LIFESAVING ECG

Bravera Health Seven Rivers

Bravera Health Brooksville

Bravera Citrus Hills

Bravera Health Spring Hill

Wayne W Ruppert, CVT, CCCC, NREMT-P
Bravera Market Regional Cardiovascular Coordinator
Bravera Health Hospitals:
Brooksville – Spring Hill – Seven Rivers

“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:

“The Integrated, Lifesaving Lead ECG”

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

“The Integrated, Lifesaving Lead ECG”

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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

“The Integrated, Lifesaving Lead ECG”

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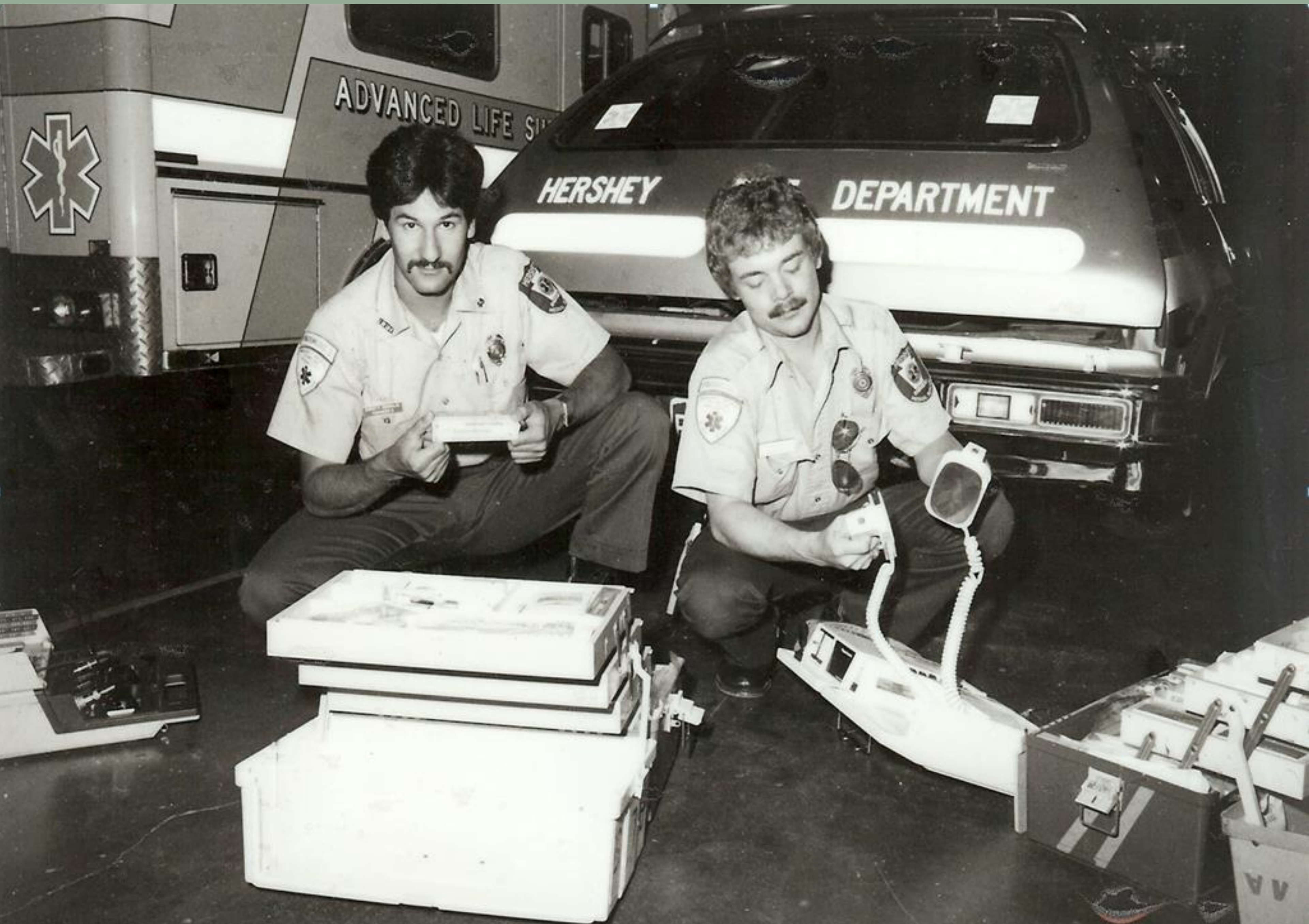
- Acute Coronary Syndromes
- Sudden Arrhythmia Death Syndromes (SADS)

We do not cover “Axis Deviation or Axis Rotation” in today’s course. While helpful to know, these items are not critical to identifying ACS or SADS conditions.

Welcome !

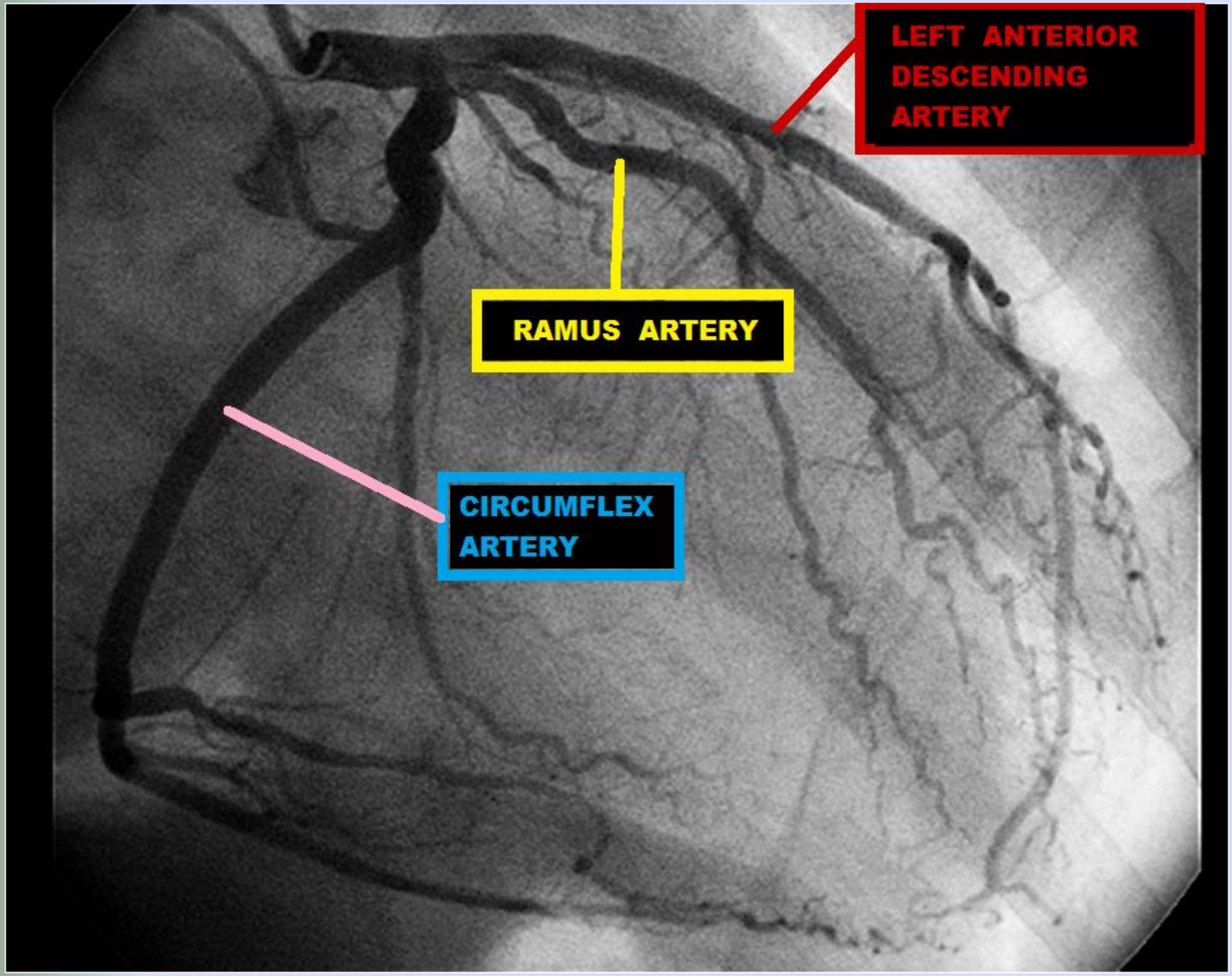


Paramedics Christ Megoulas and Wayne Ruppert, Hershey, PA Fire Department, 1982

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

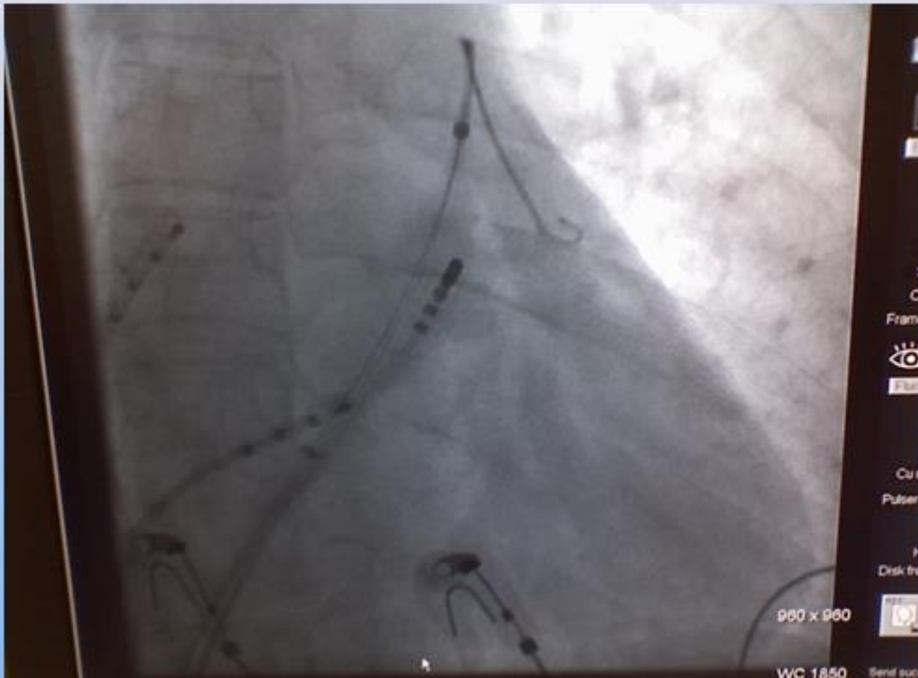
Wayne Ruppert and Dr. James Irwin, St Joseph’s Hospital, Tampa, 7/29/2004

Cardiac Cath Lab Advantage:



Correlation of ECG leads with SPECIFIC cardiac anatomic structures.

Electrophysiology Lab Case Studies



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

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

EP Lab Advantage:

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

Wayne Ruppert – Bio:

- 1978 – 1996 EMT-Paramedic
- 1996 – 2012 Interventional Cardiovascular Technologist Cardiac Cath Lab and Electrophysiology Labs
- 2012 – Present Cardiovascular Programs Director / Coordinator

Wayne Ruppert - Bio:

- Cardiovascular Coordinator 2012-present (coordinated 7 successful accreditations)
- Interventional Cardiovascular / Electrophysiology Technologist, 1995-Present. (Approx 13,000 patients)
- Author of: "**12 Lead ECG Interpretation in Acute Coronary Syndrome with Case Studies from the Cardiac Cath Lab**," 2010, TriGen publishing / Ingram Books
- Author of: "**STEMI Assistant**," 2014, TriGen publishing / Ingram Books
- Florida Nursing CE Provider # 50-12998
- 12 Lead ECG Instructor, 1994-present (multiple hospitals, USF College of Medicine 1994)
- ACLS Instructor: 1982 - 2022
- Website: www.ECGtraining.org

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)
  - American Heart Association (AHA) Circulation
  - ACC/AHA Guidelines
  - New England Journal of Medicine

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  - ACC/AHA Guidelines
  - New England Journal of Medicine
- Two peer reviewed, published textbooks

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

This book prepares you to:

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

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

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

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

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

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

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

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

Humberto Coto, MD, FACP, FACC
- Chief of Interventional Cardiology
St. Joseph's Hospital
Tampa, Florida

ISBN 978-0-9829172-1-3
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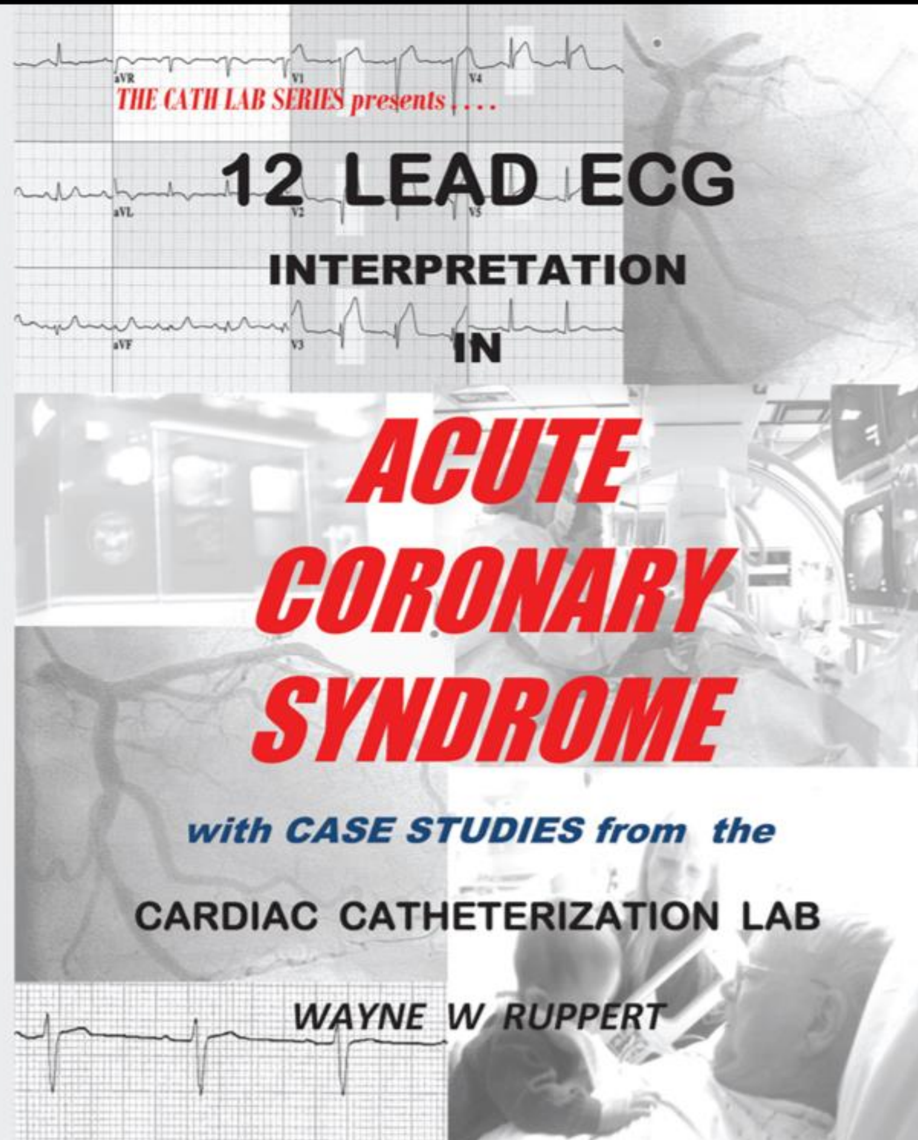
12 LEAD ECG INTERPRETATION in ACUTE CORONARY SYNDROME with CASE STUDIES from the CATH LAB -- *WAYNE RUPPERT*



www.TriGenPress.com

www.ECGtraining.org

BarnesandNoble.com

Amazon.com

TEXTBOOK REVIEWED BY:

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

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

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

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

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

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2010 - Current

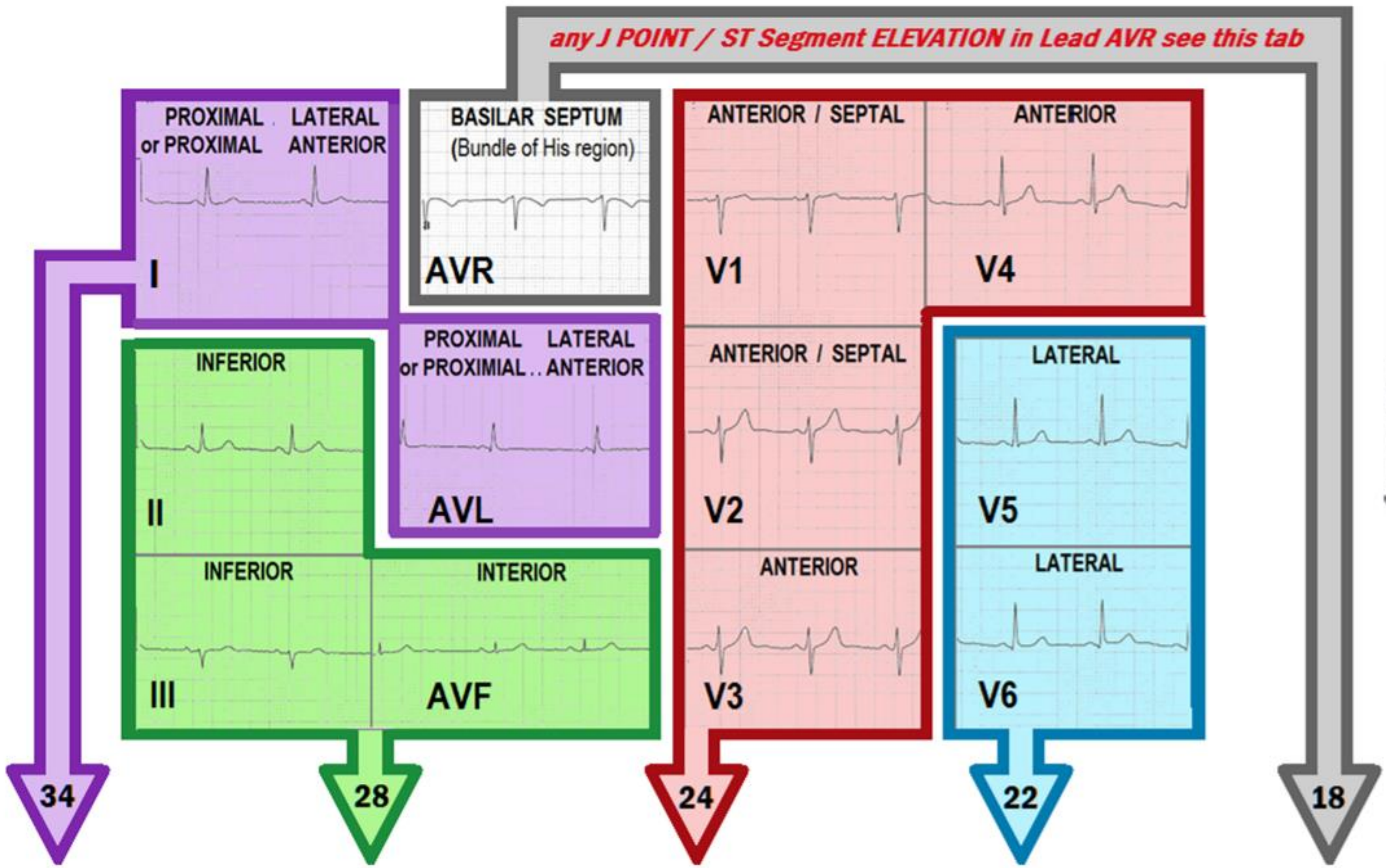
STEMI Assistant

by Wayne Ruppert

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

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

CRASH CART EMERGENCY REFERENCE



Bayfront Health
Dade City

TEXTBOOK REVIEWED BY:

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

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

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

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

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2010 - Current

STEMI Assistant

[Tutorial Video]

[Free download – electronic copy (PDF file)]

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Wayne.ruppert@bayfronthealth.com

Helpful Web Resources:

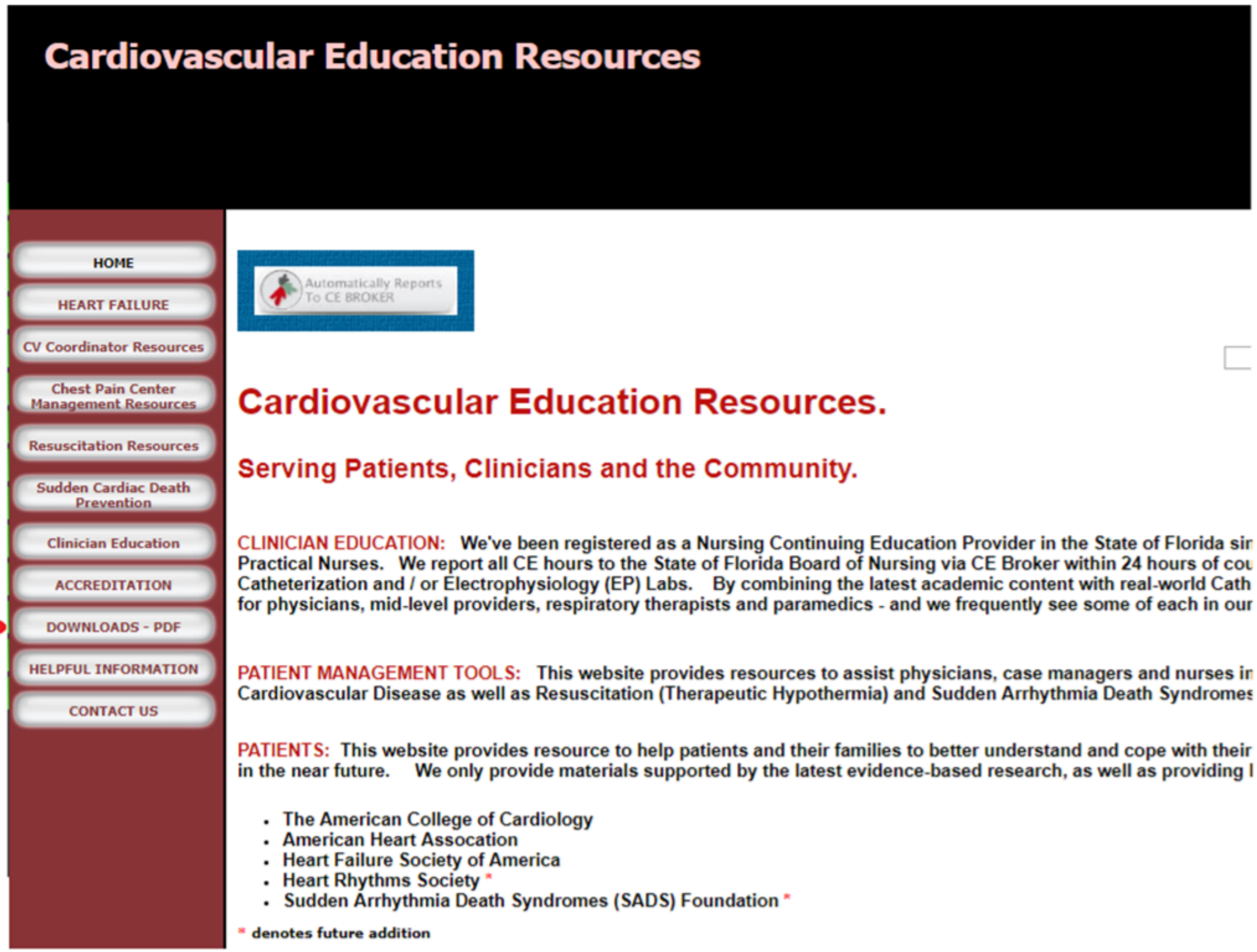
www.practicalclinicalskills.com

www.skillstat.com/tools/ecg-simulator

www.ECGtraining.org

1. Go to: www.ECGtraining.org

2. Select "Downloads PDF" from menu bar



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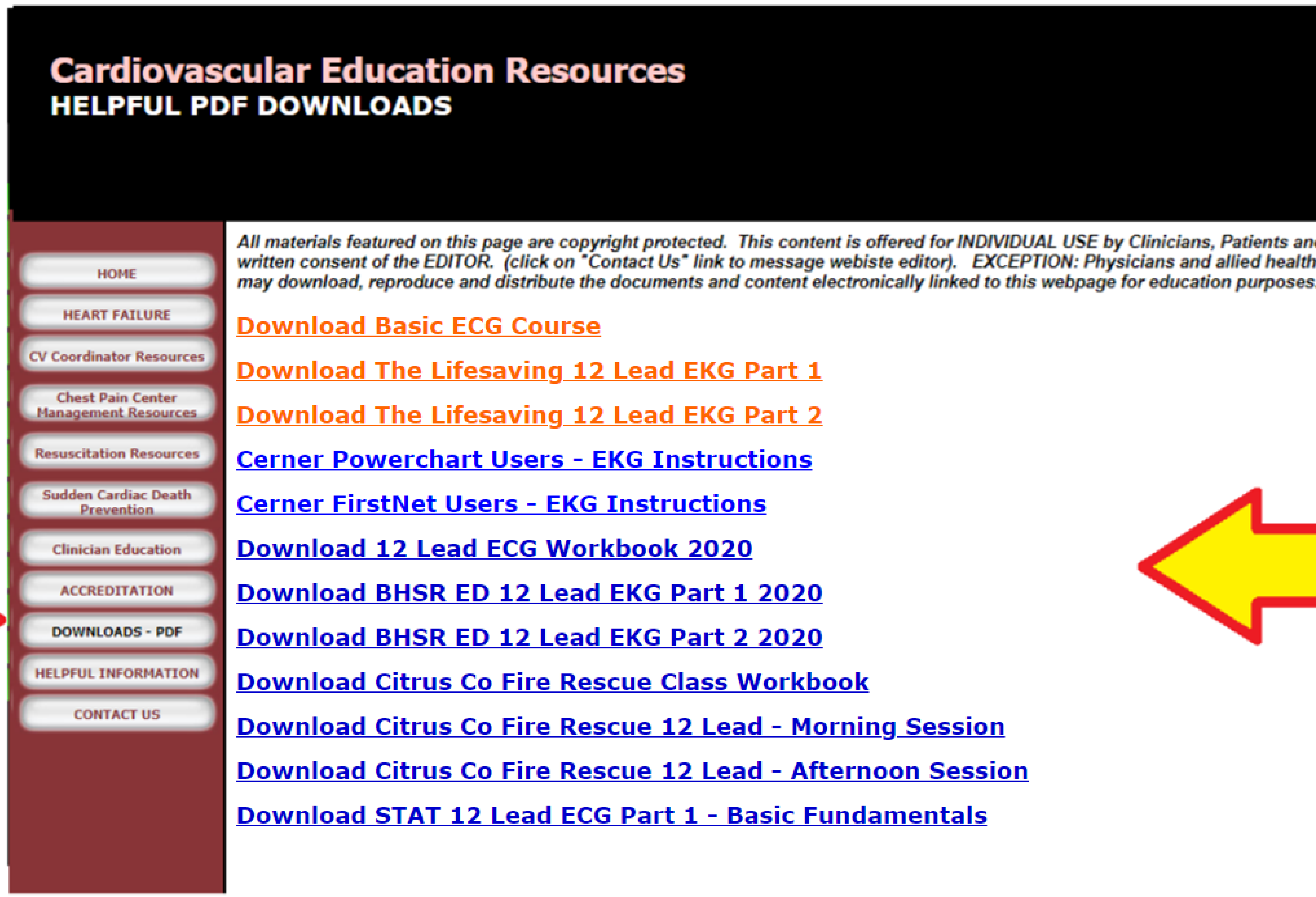
2. Select "Downloads PDF" from menu bar

3. Select your courses



SUGGESTION *for optimal learning…….*

To get the most from this class:

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

Don’t worry.

Don’t worry.

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

Don't worry.

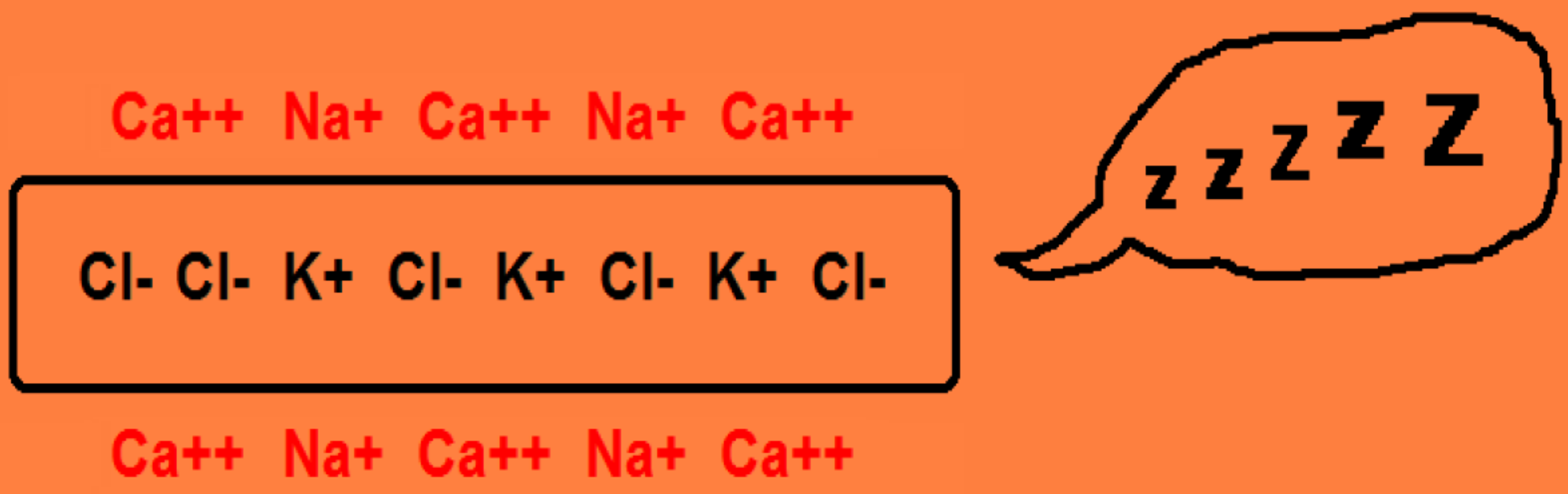
This course will be NOWHERE NEAR as much fun as a roller coaster.

But it will move as fast.

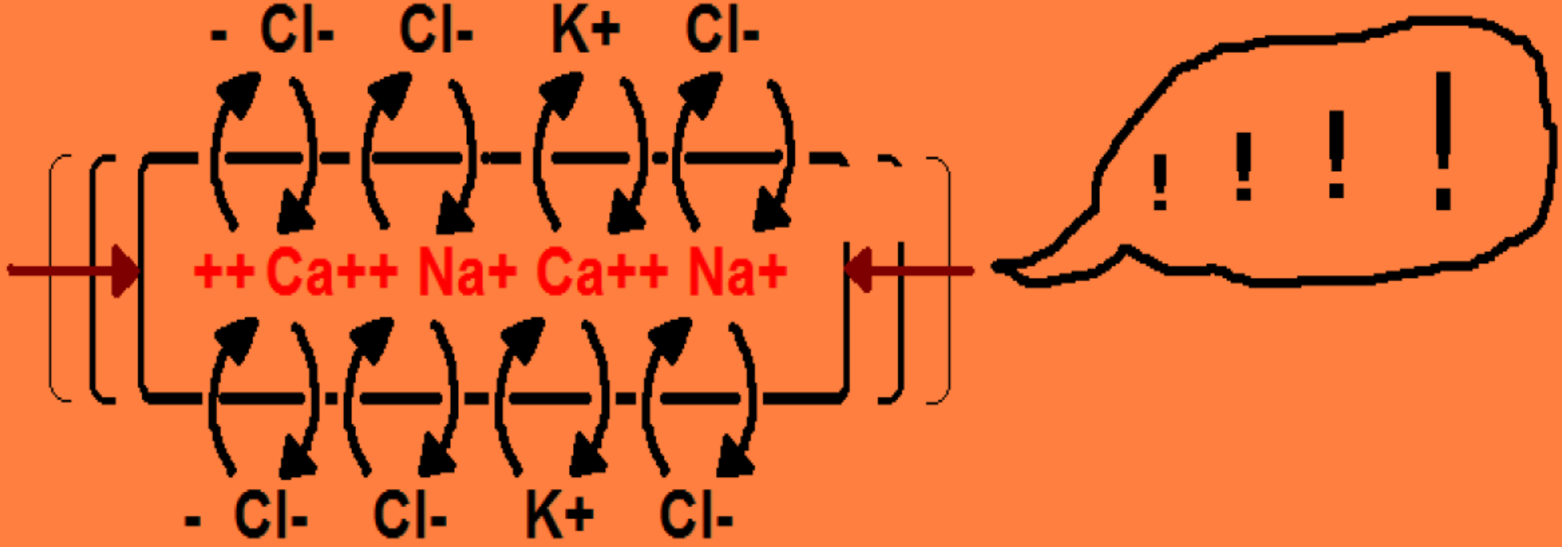
Cardiac A & P "101" . . . .

- **Action Potential of Ventricular Muscle Cells**

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



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



. . . THE CELL CONTRACTS !

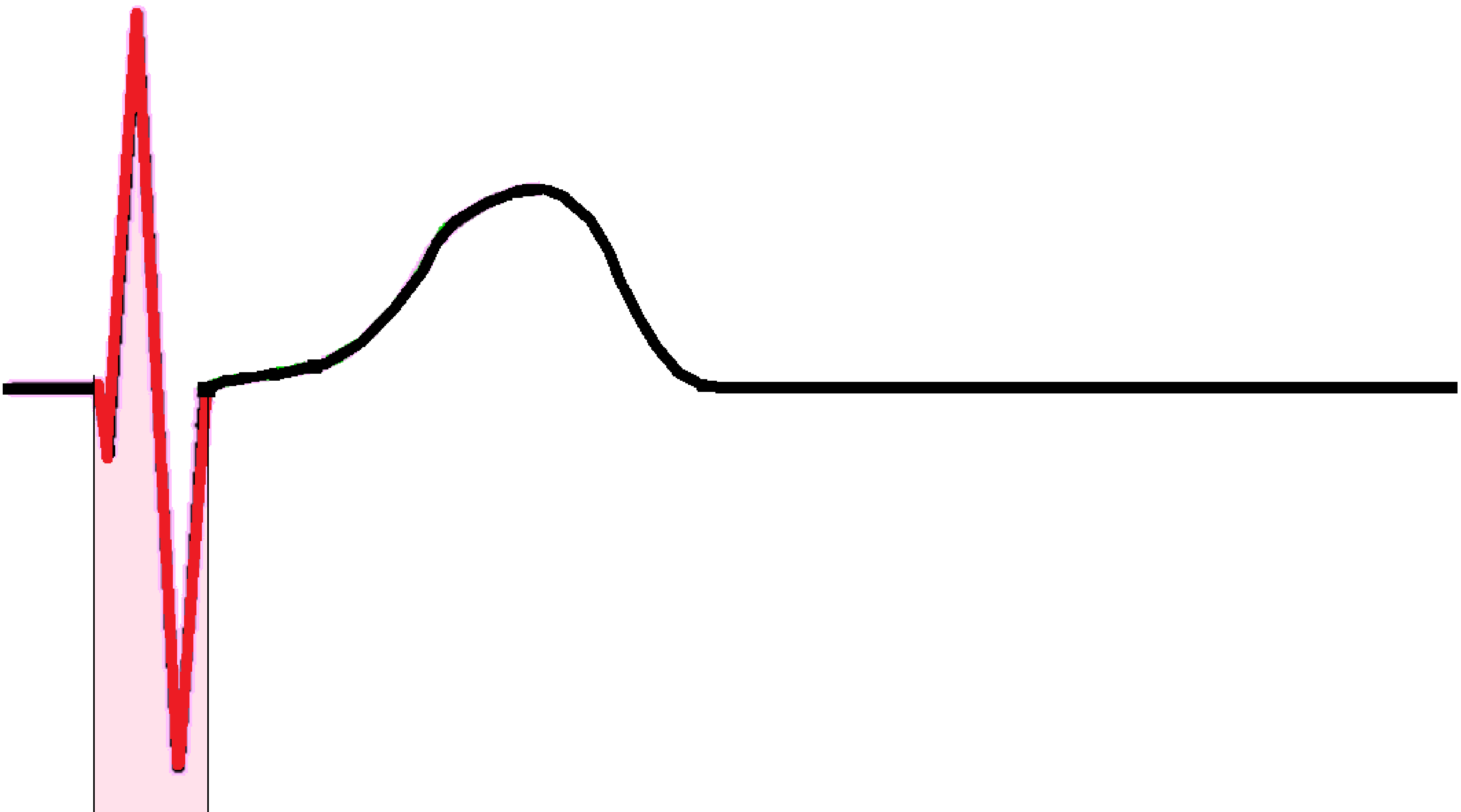
THIS (OF COURSE) IS KNOW AS . . .

DEPOLARIZATION

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

Ventricular Depolarization:

- Is represented by the **QRS Complex**



QRS Complex = Ventricular Depolarization

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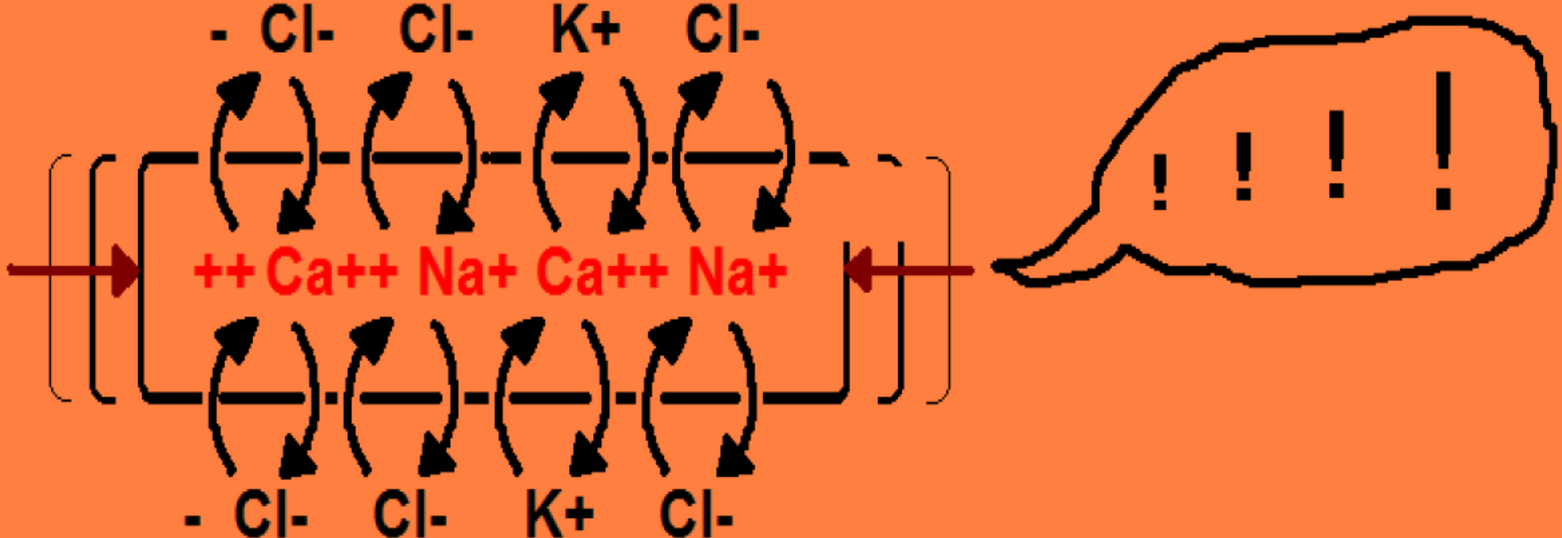
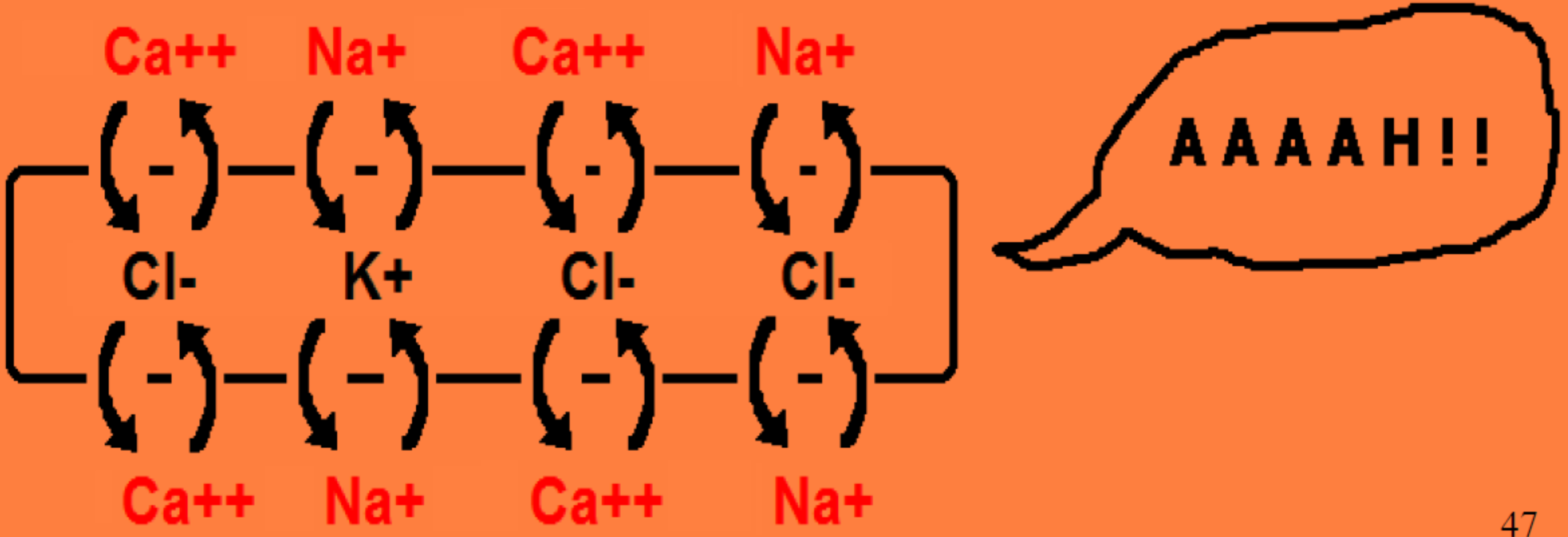


. . . THE CELL CONTRACTS!

AFTER DEPOLARIZATION, THE CELLS RELAX.

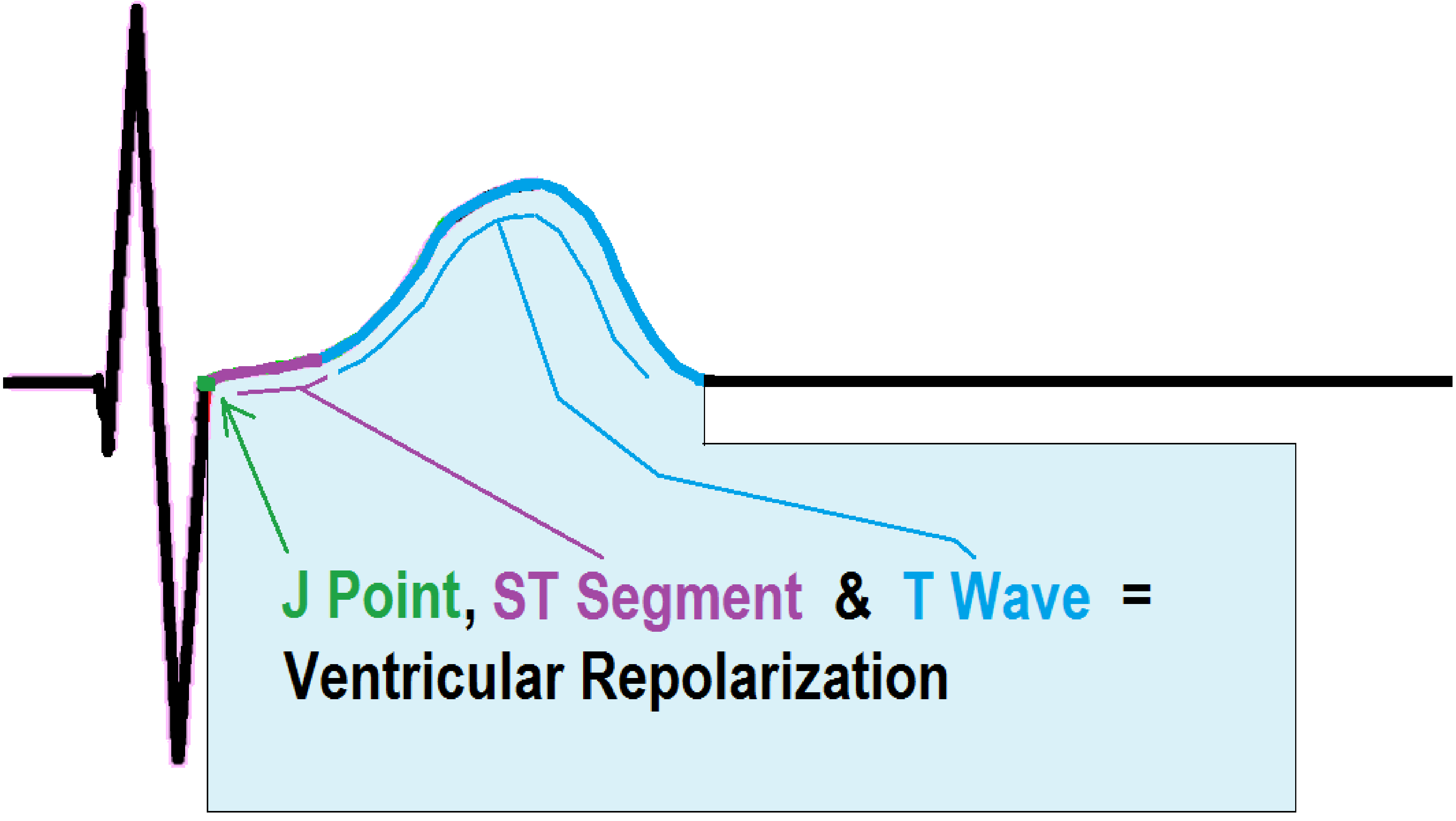
THE IONS RETURN TO THEIR ORIGINAL POSITIONS --

THIS PROCESS IS KNOWN AS REPOLARIZATION

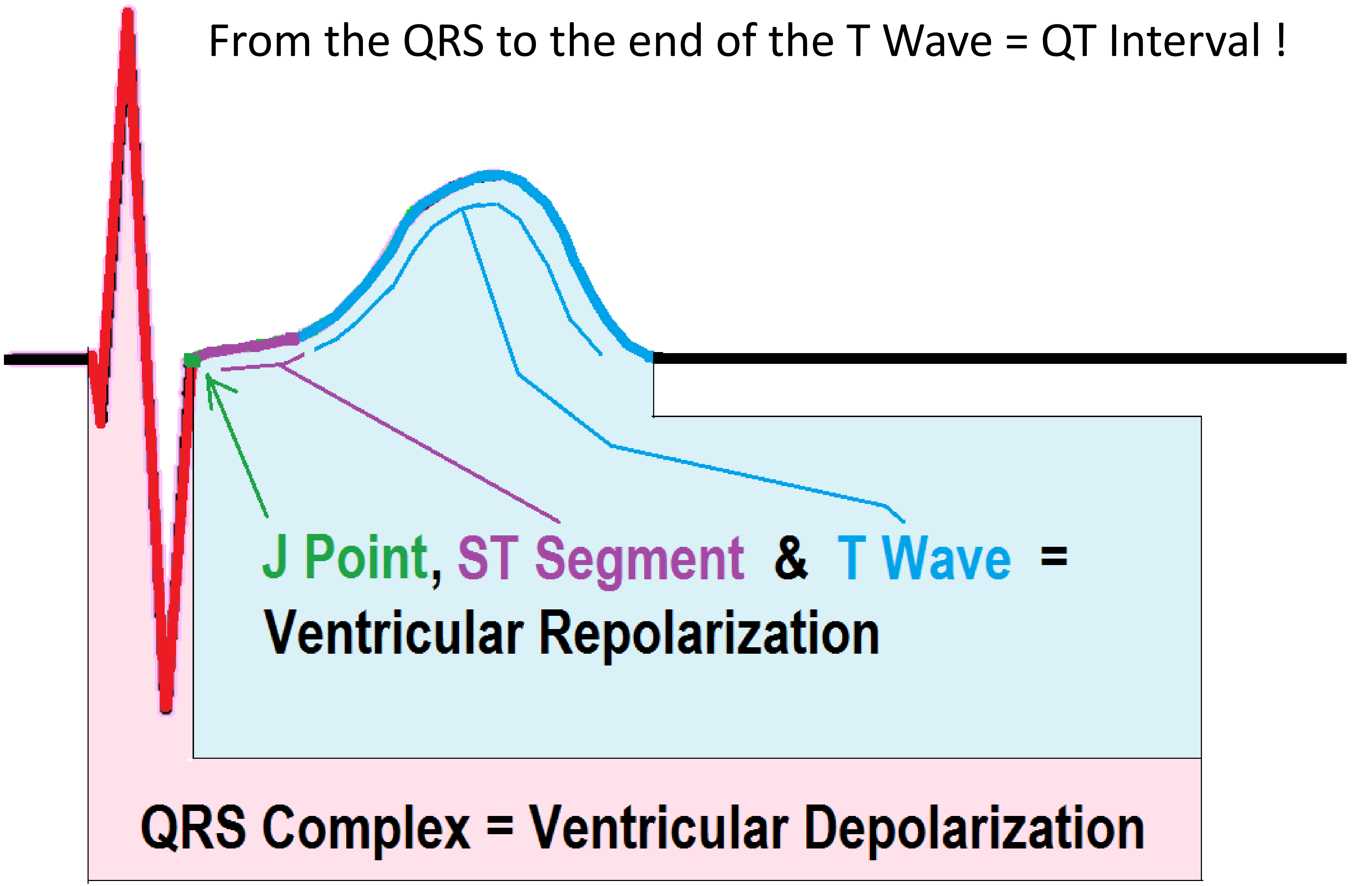


Repolarization on the ECG:

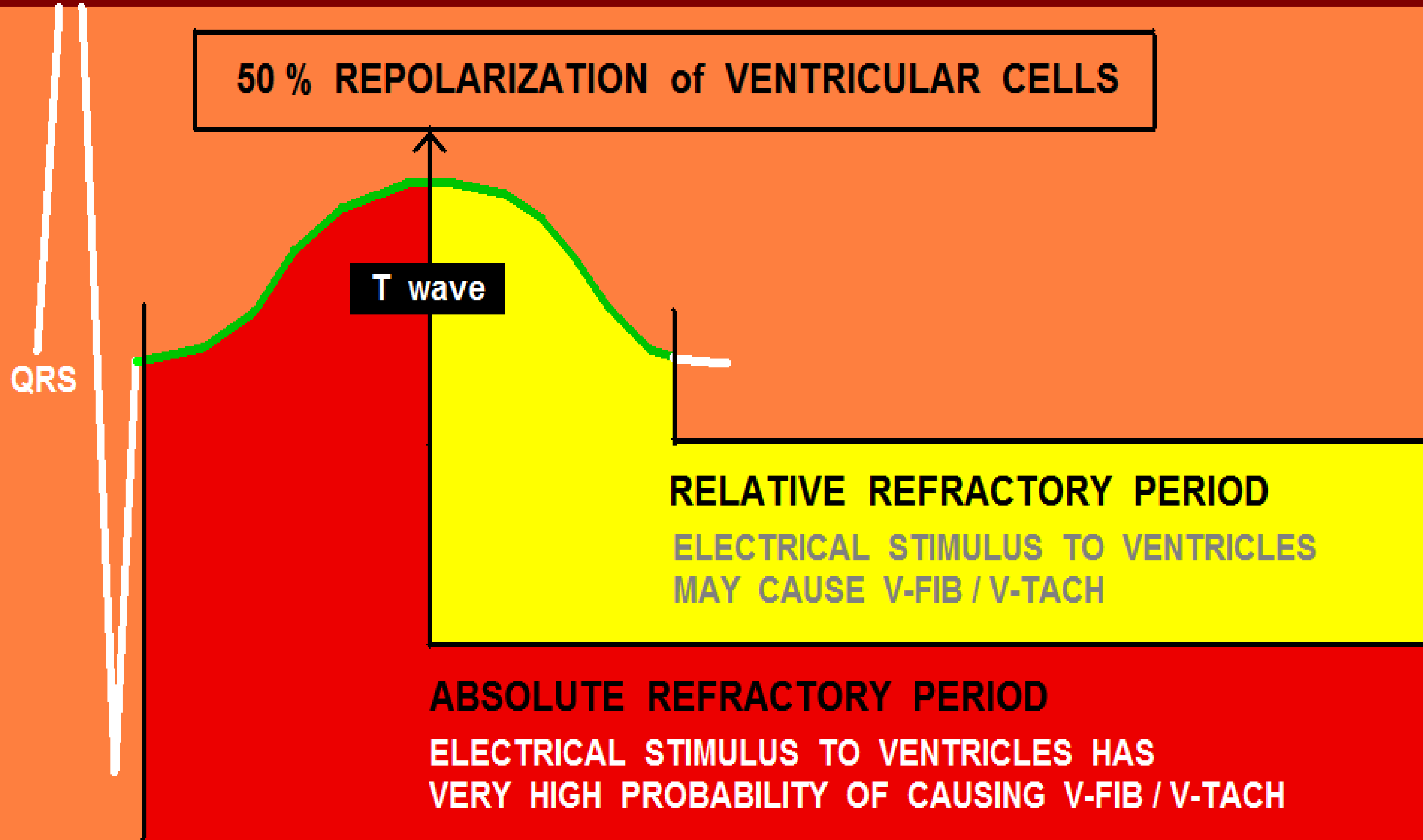
- Is represented by the:
  - **J Point**
  - **ST Segment**
  - **T Wave**



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"



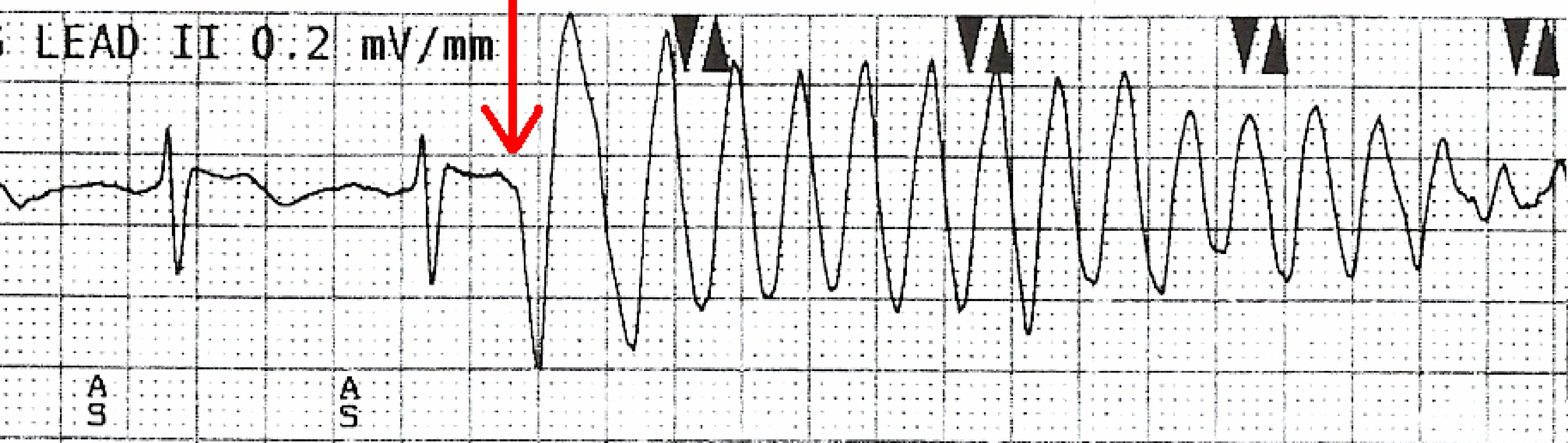
ROUTINE TEST OF ICD

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

08-Sep-2006 18:01:47

Test Started

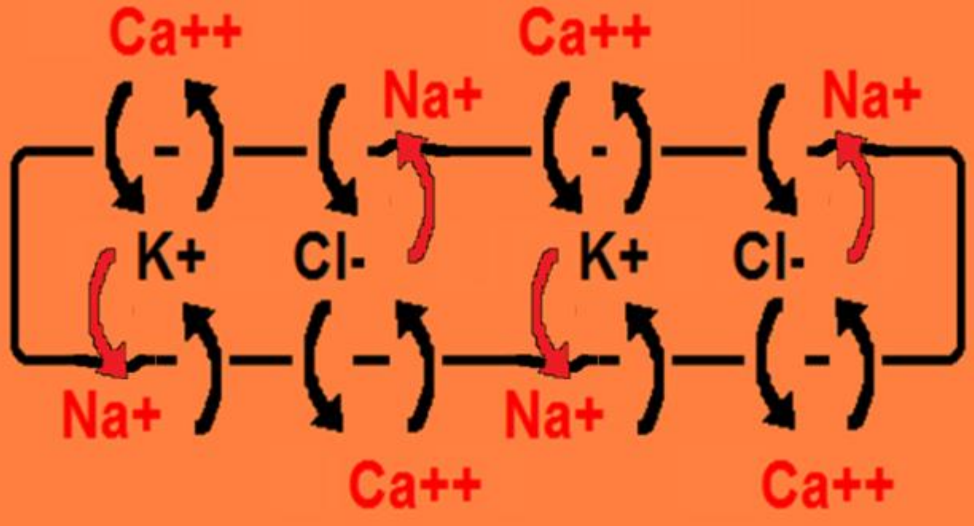
SPECIAL THANKS TO:
Ray Heinley
Medtronic Corporation
for this contribution



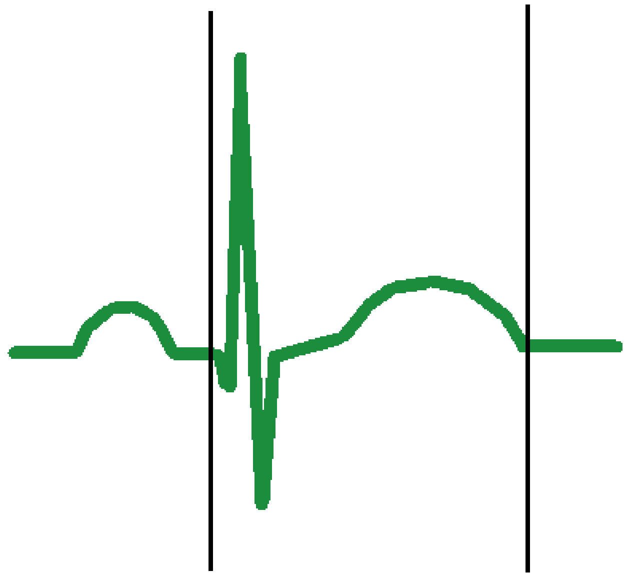
CARDIAC ANATOMY and PHYSIOLOGY "101"

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

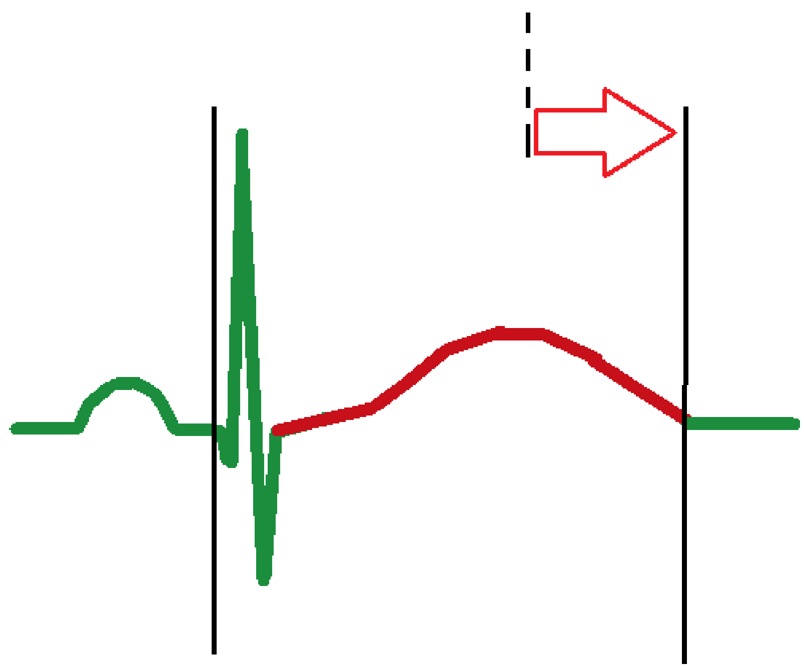
. . . . this can DELAY REPOLARIZATION, which will show on the ECG as "QT Prolongation"



Normal
QT Interval



ABNORMAL
(prolonged)
QT Interval



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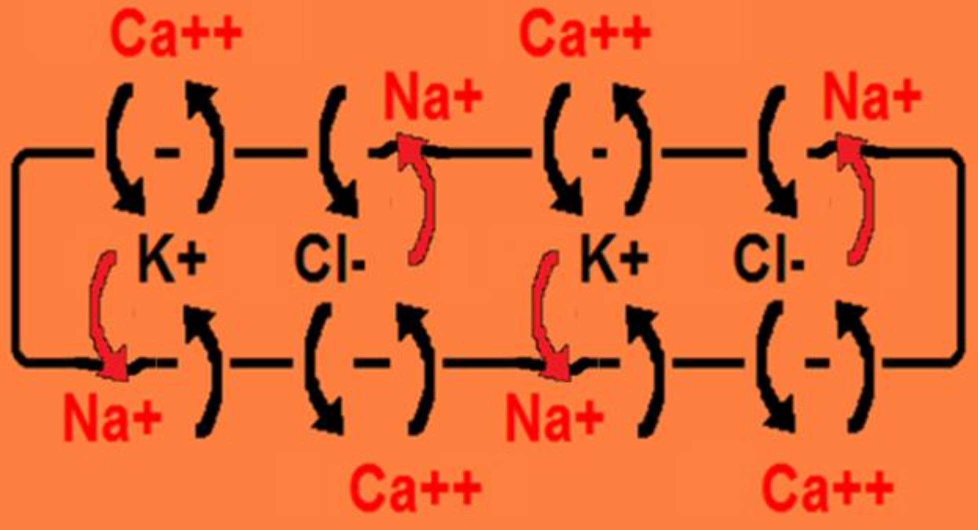
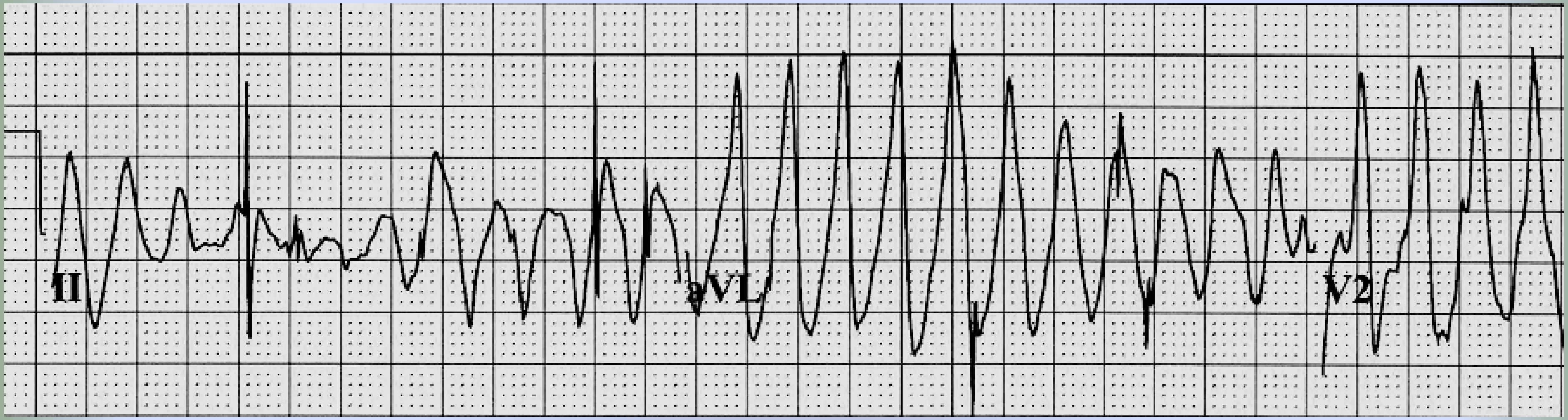


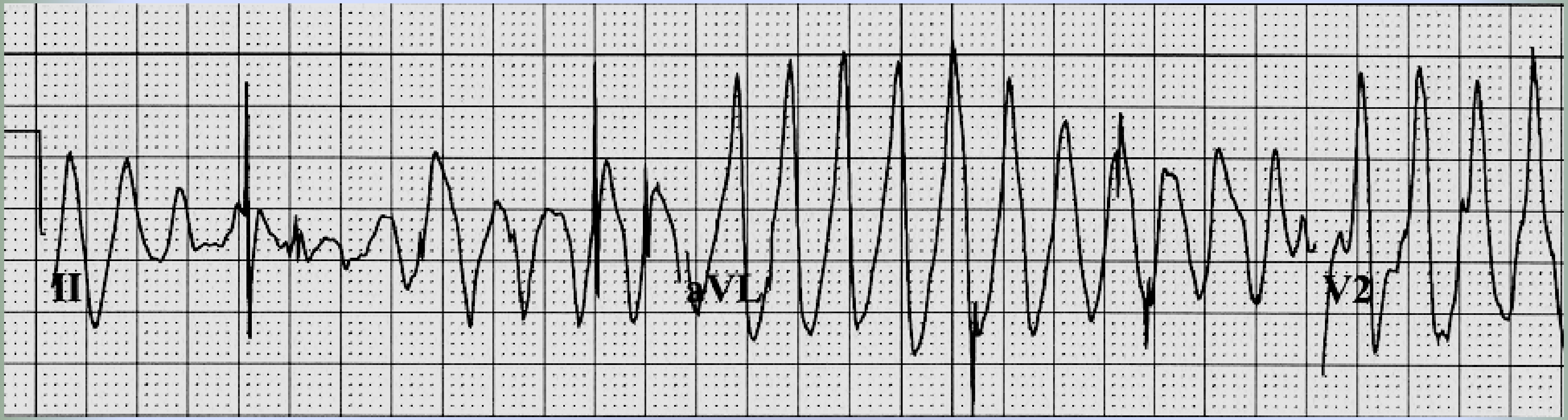
which can lead to Torsades . . . Cardiac Arrest . . . and *SUDDEN DEATH.*

Torsades de Pointes (TdP)



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

Torsades de Pointes (TdP)



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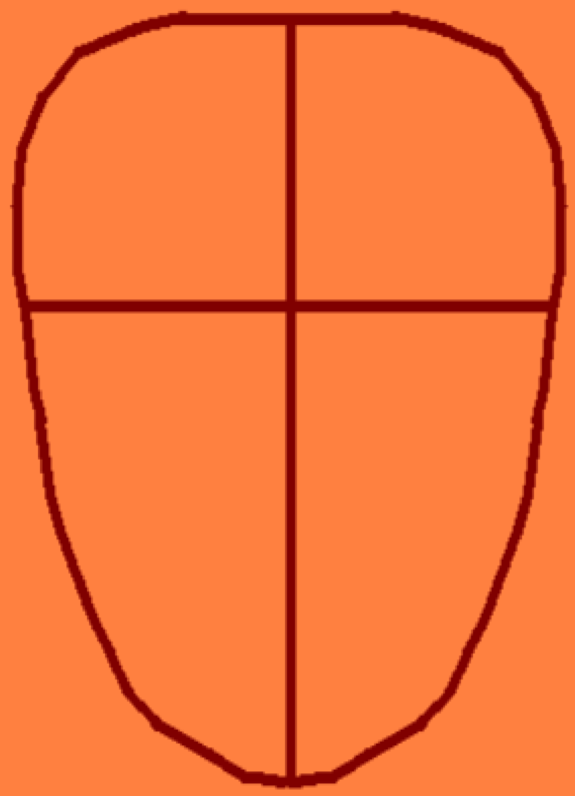
Cardiac A & P "101" . . . .

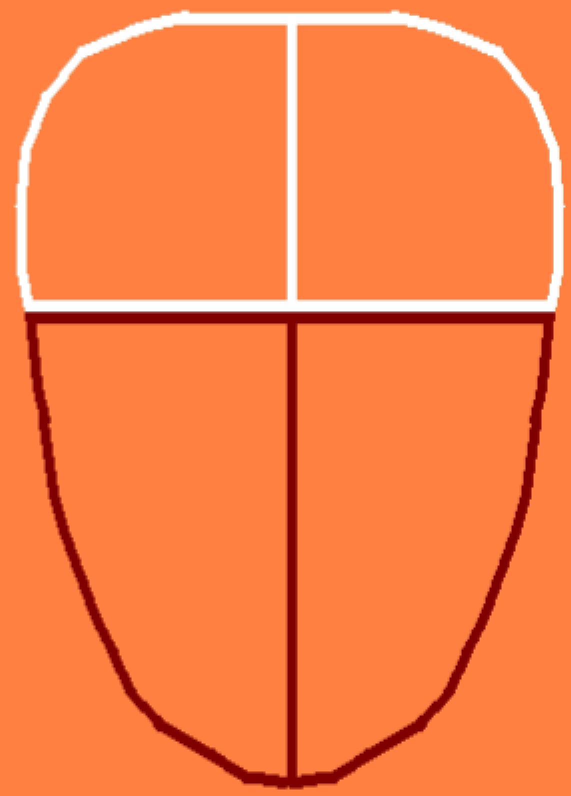
- Action Potential of Ventricular Muscle Cells
- **Rapid basic review heart structure**

FOUR CHAMBERED PUMP



FOUR CHAMBERED PUMP . . .

2 ATRIUM

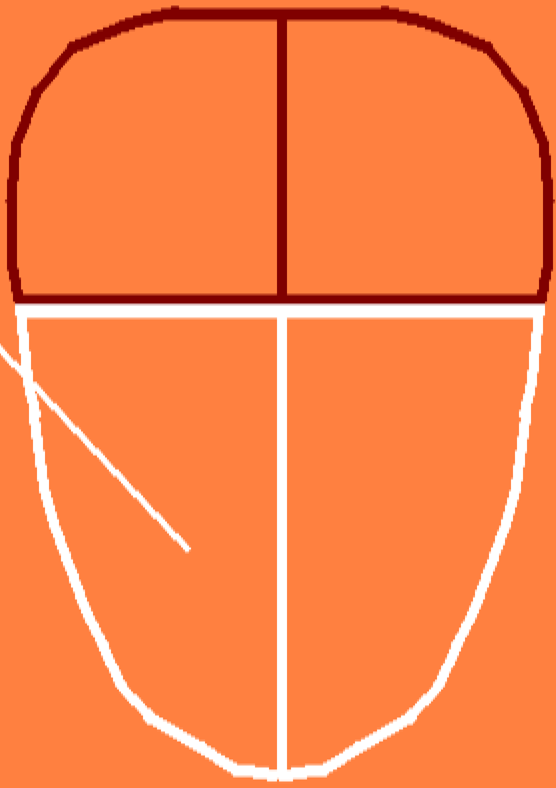


PRIMARY JOB:

"PACK VENTRICLES FULL OF BLOOD"

FOUR CHAMBERED PUMP . . .

2 VENTRICLES



<u>PRIMARY JOB:</u>

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

WHEN FUNCTIONING PROPERLY,

THE ATRIUM SUPPLY

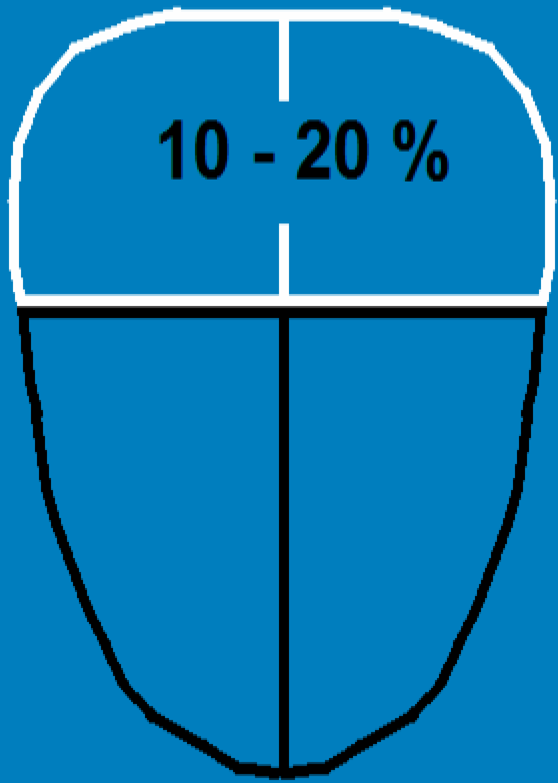
APPROXIMATELY

WHAT

PERCENTAGE

OF THE

CARDIAC OUTPUT ?



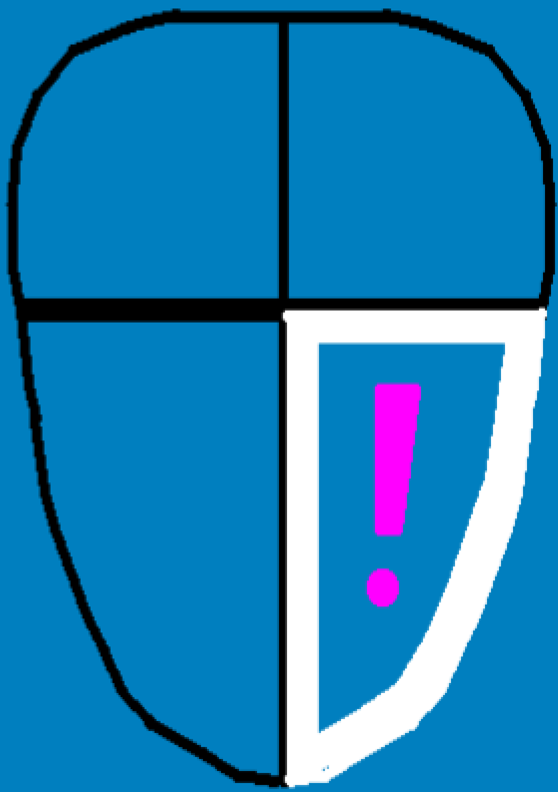
THE CHAMBER MOST IMPORTANT TO KEEPING THE PATIENT ALIVE

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

IS THE

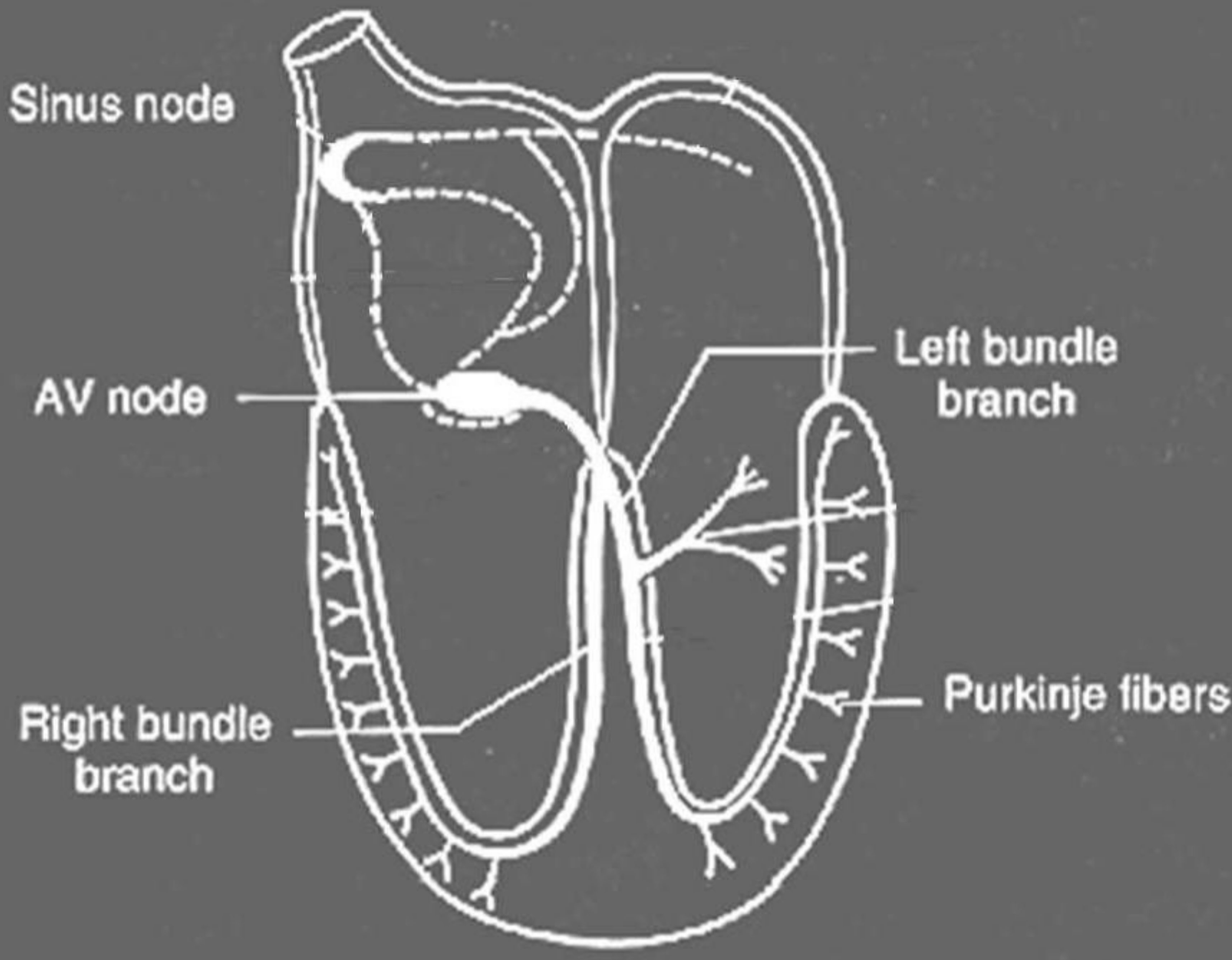
LEFT VENTRICLE

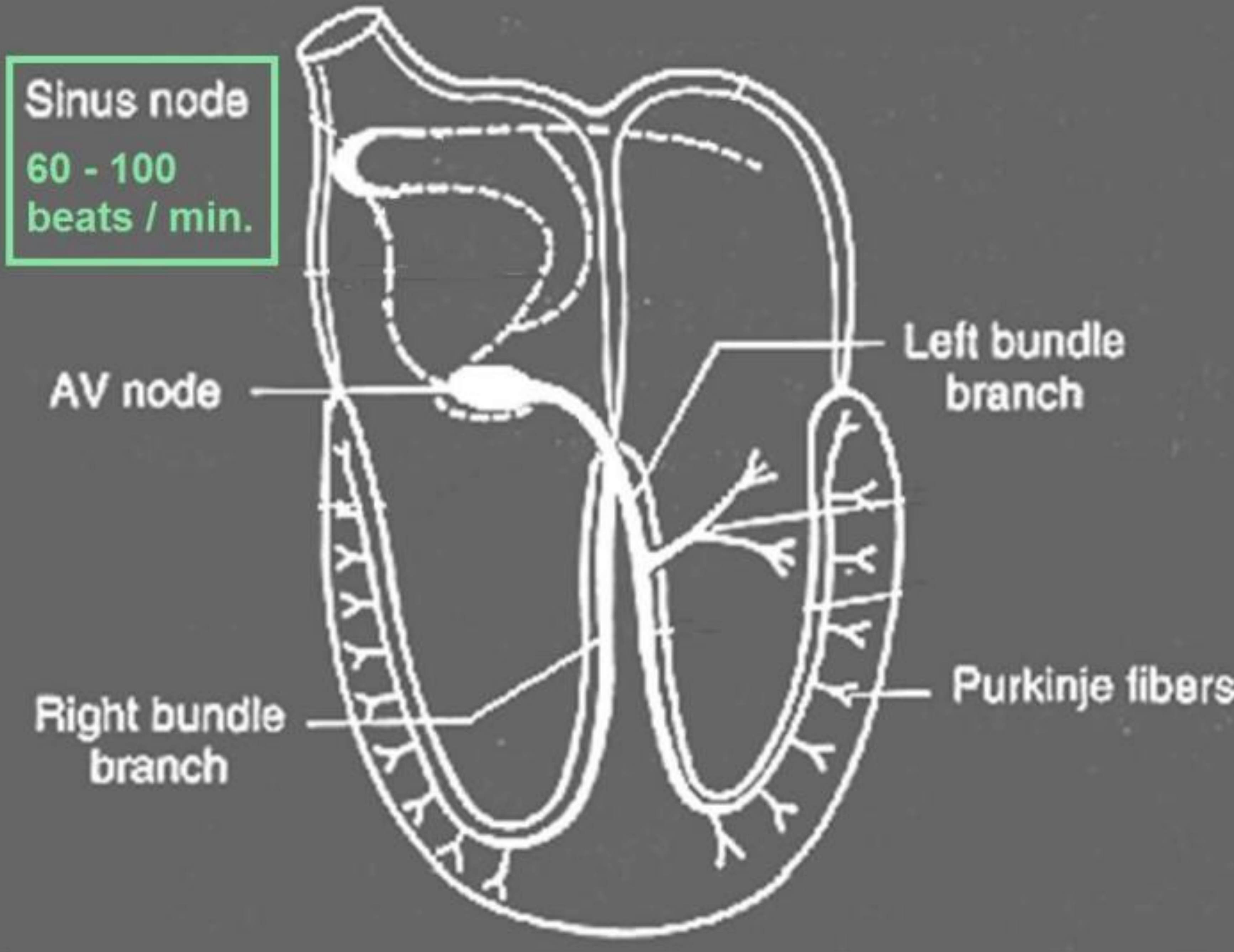
WHICH WE WILL REFER TO AS <u>THE PUMP</u>

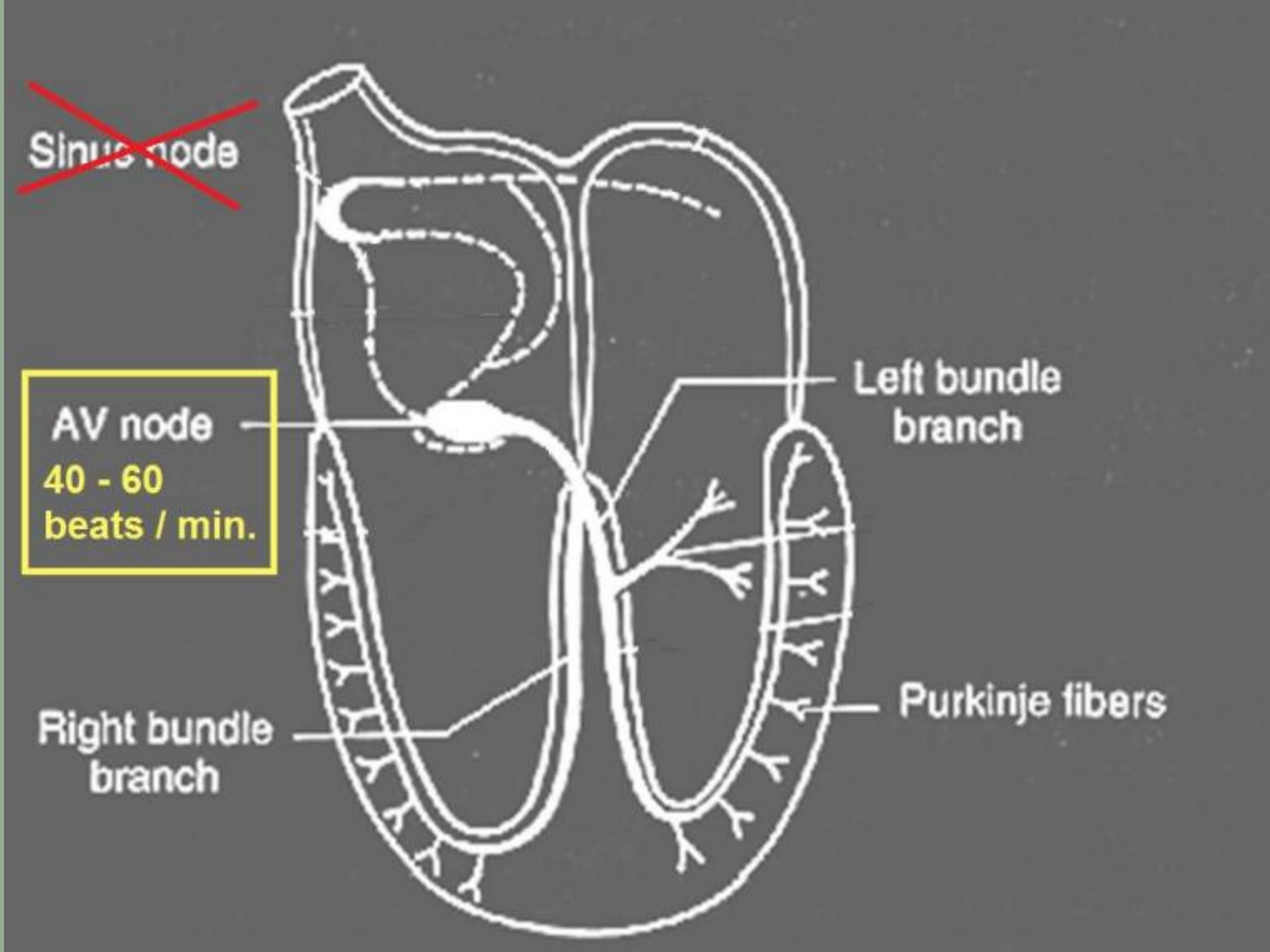


Cardiac A & P "101" . . . .

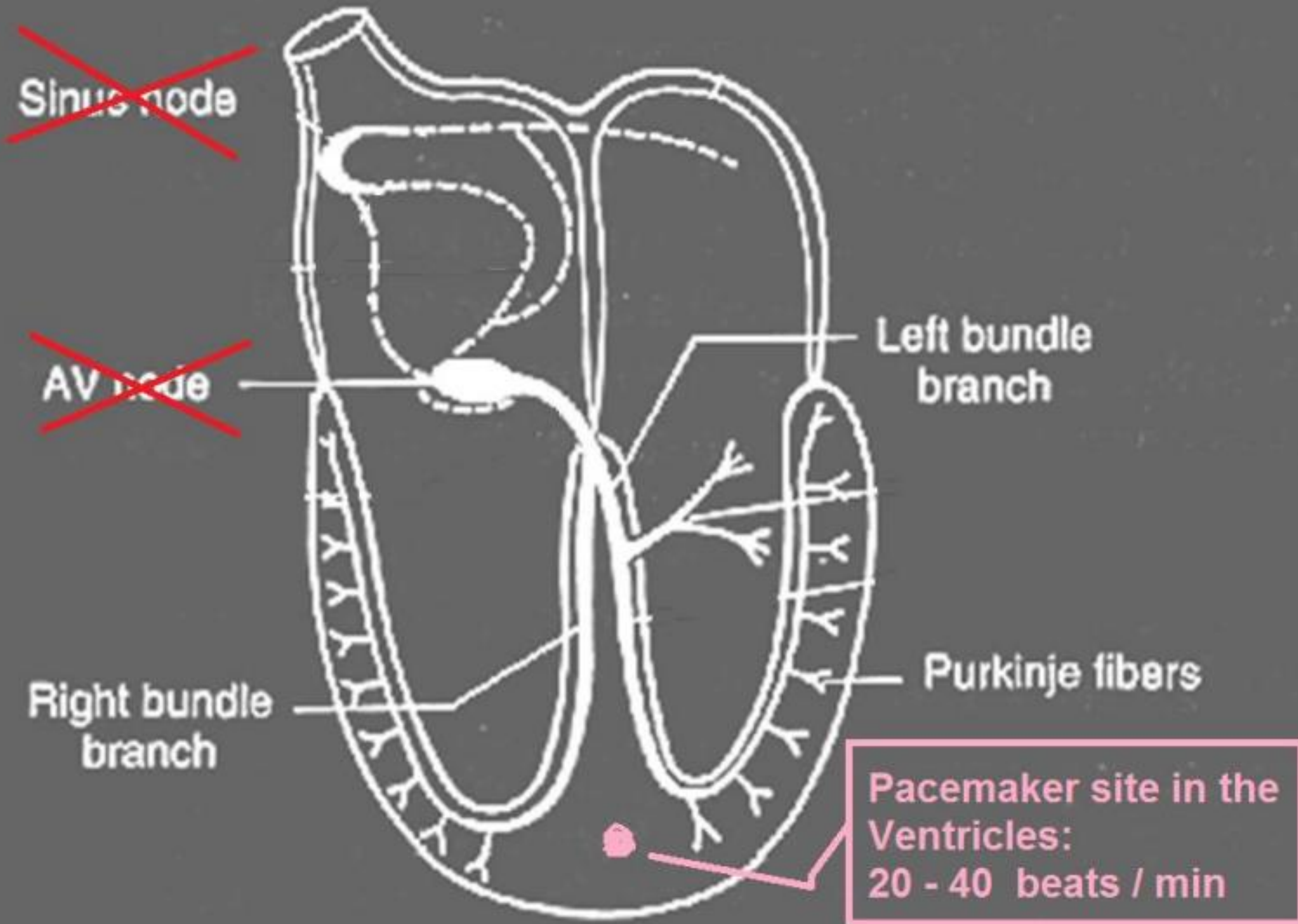
- Action Potential of Ventricular Muscle Cells
- Rapid basic review heart structure
- **Electrical System**


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


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



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

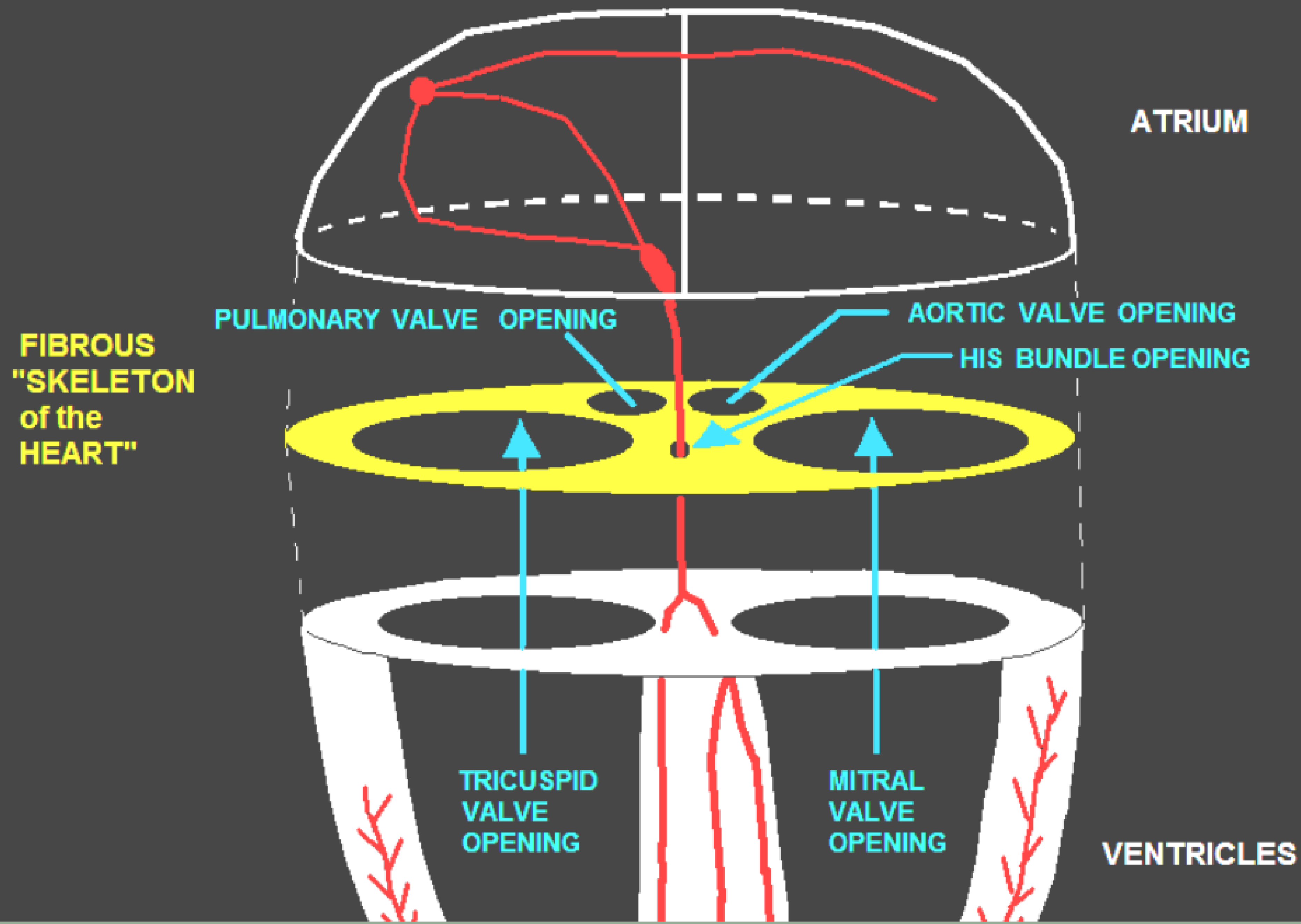


~~Sinus node~~
~~AV node~~
Left bundle branch
Purkinje fibers
Right bundle branch
Pacemaker site in the Ventricles:
20 - 40 beats / min

Cardiac A & P "101" . . . .

- Action Potential of Ventricular Muscle Cells
- Rapid basic review heart structure
- Electrical System
- **Fibrous Skeleton of the Heart**

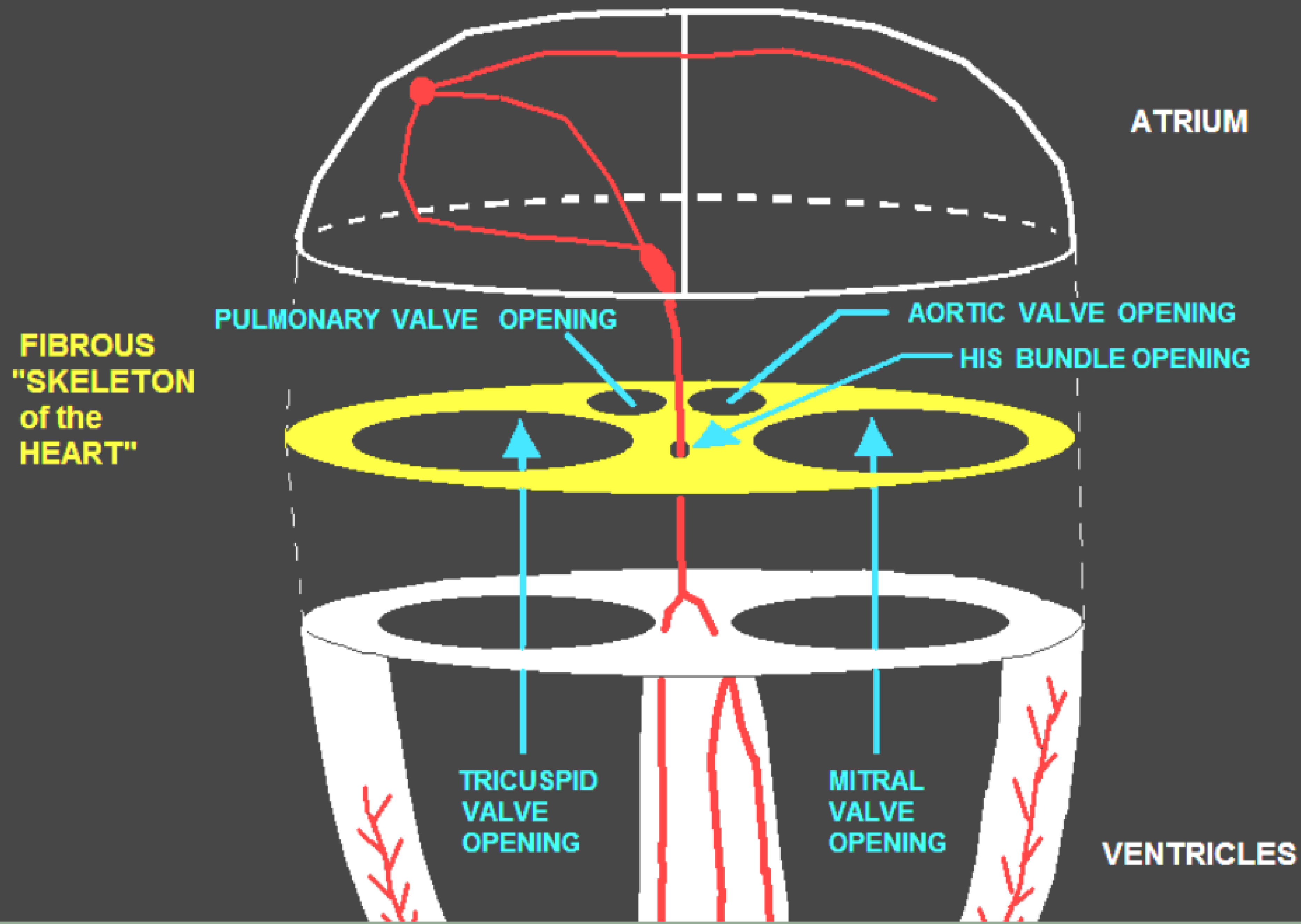
THE "SKELETON OF THE HEART"

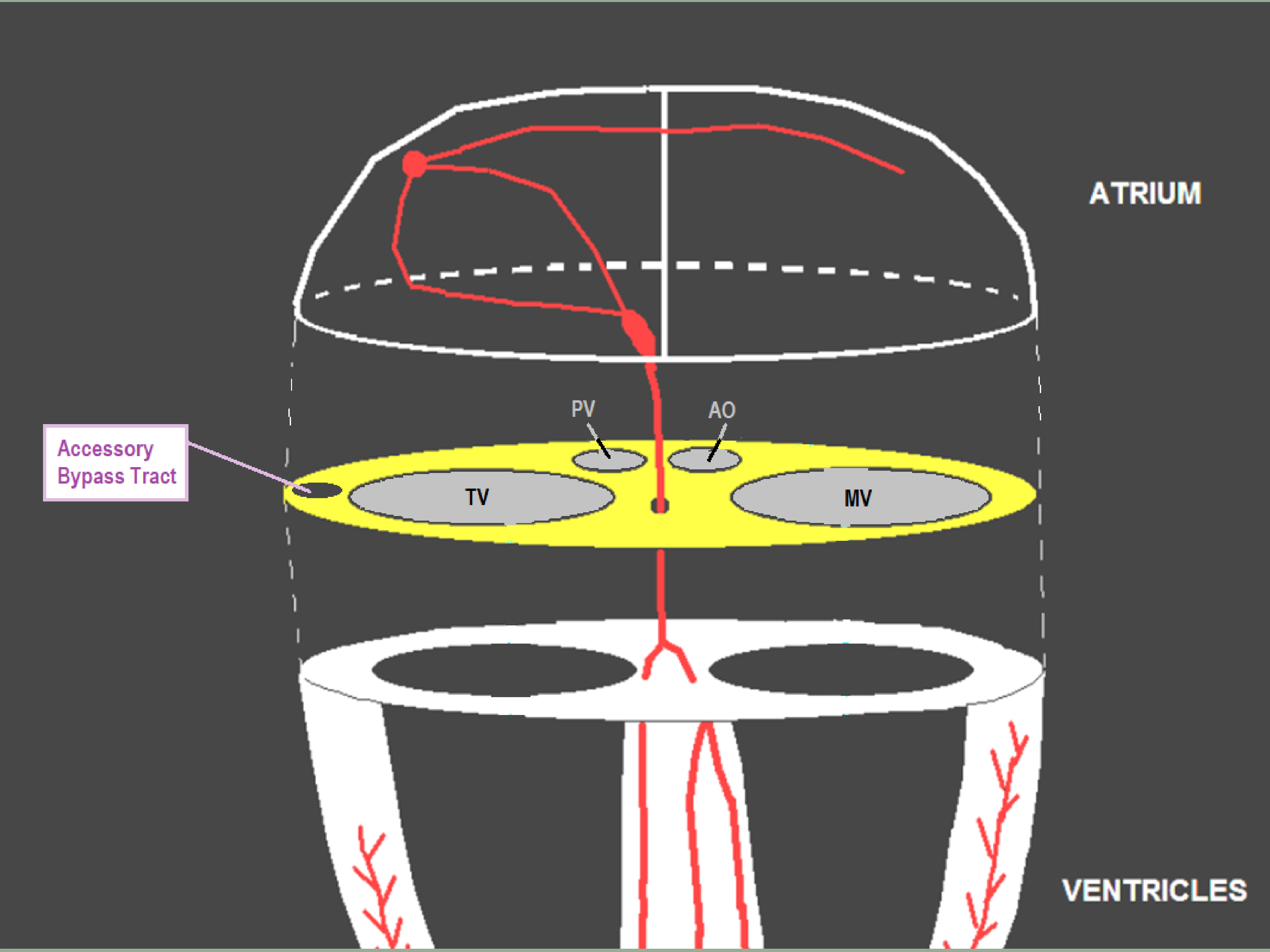


Cardiac A & P "101" . . . .

- Action Potential of Ventricular Muscle Cells
- Rapid basic review heart structure
- Electrical System
- Fibrous Skeleton of the Heart
- **Pathophysiology of Accessory Bypass Tracts (cause of Wolff-Parkinson-White Syndrome)**

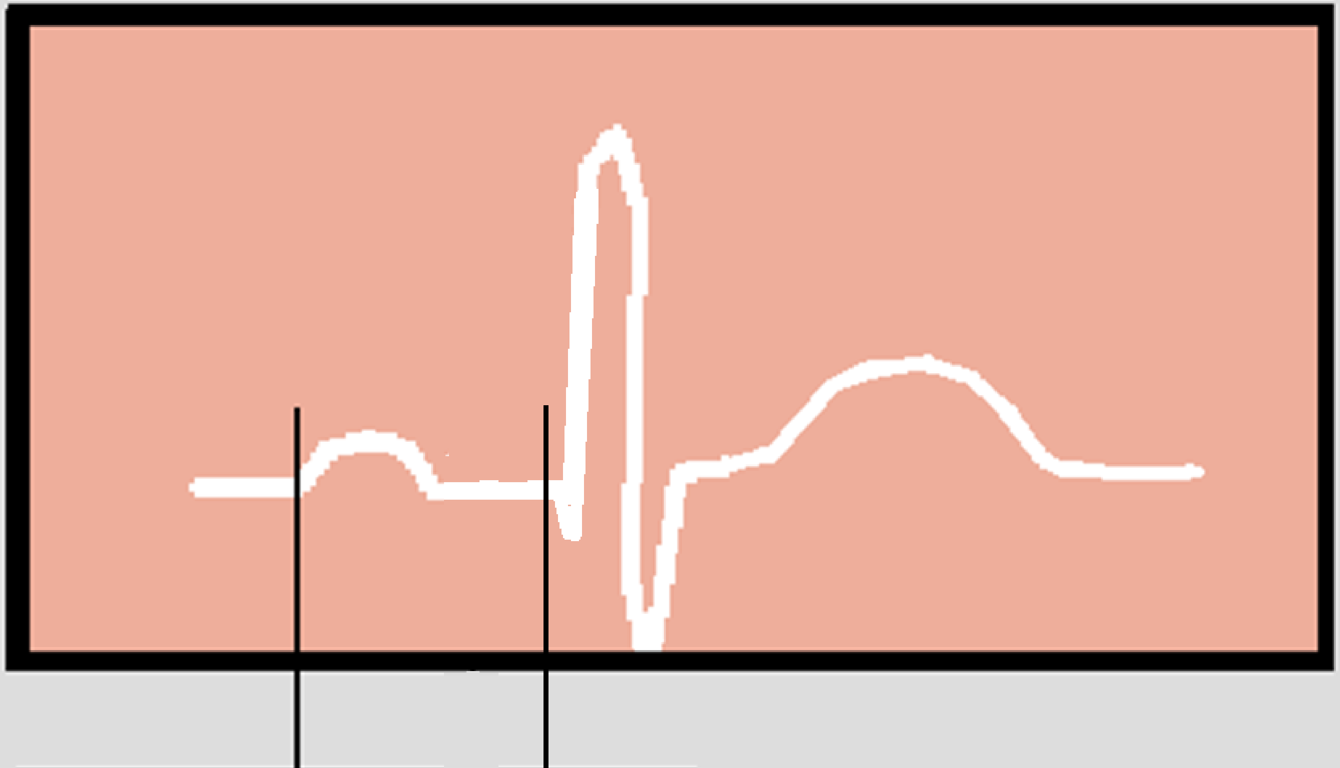
THE "SKELETON OF THE HEART"




ATRIUM
PV
AO
Accessory
Bypass Tract
TV
MV
VENTRICLES

WOLFF-PARKINSON-WHITE

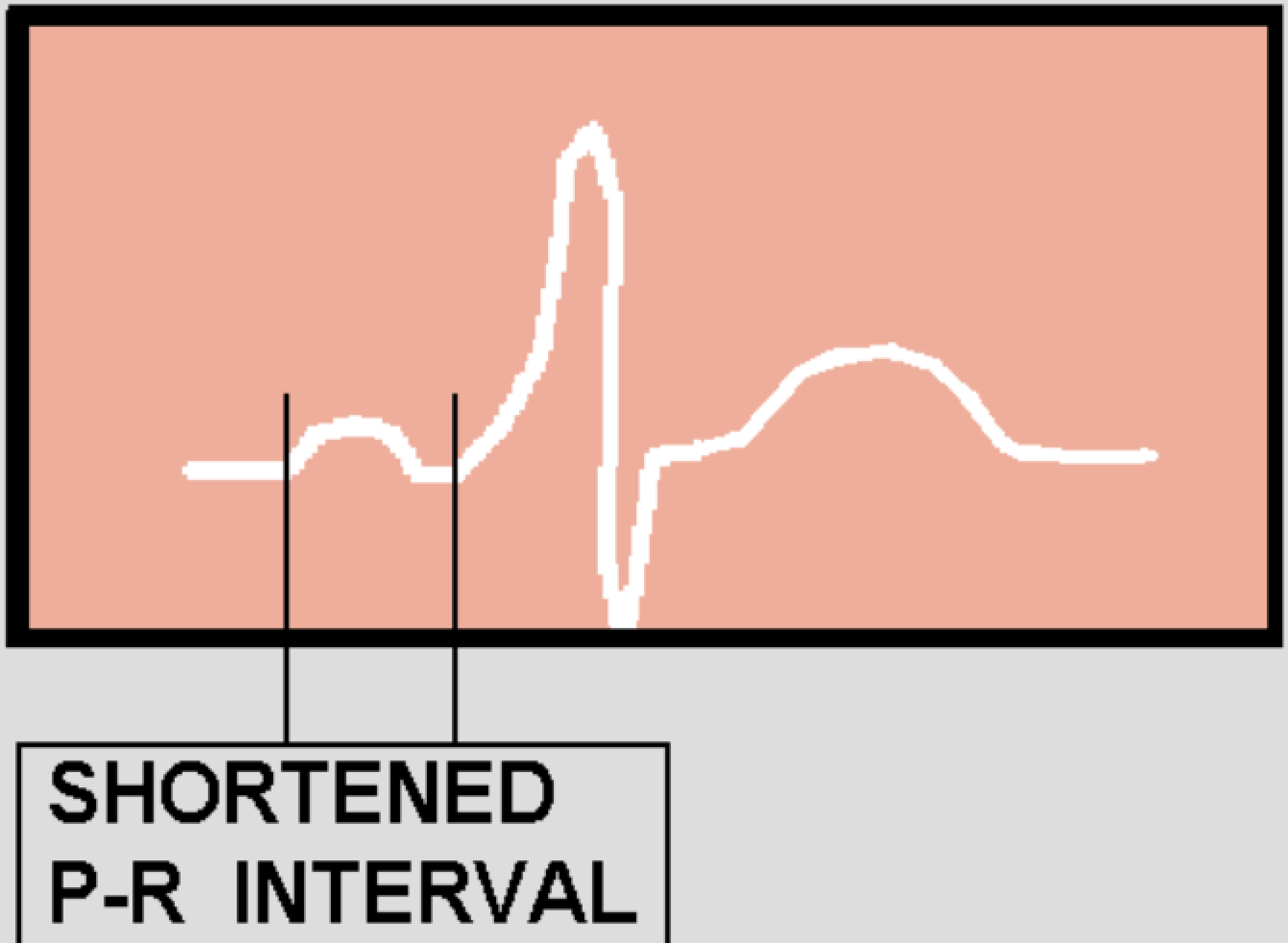
THE NORMAL ECG . . . .



NORMAL
P-R INTERVAL

WOLFF-PARKINSON-WHITE

EKG CHARACTERISTICS

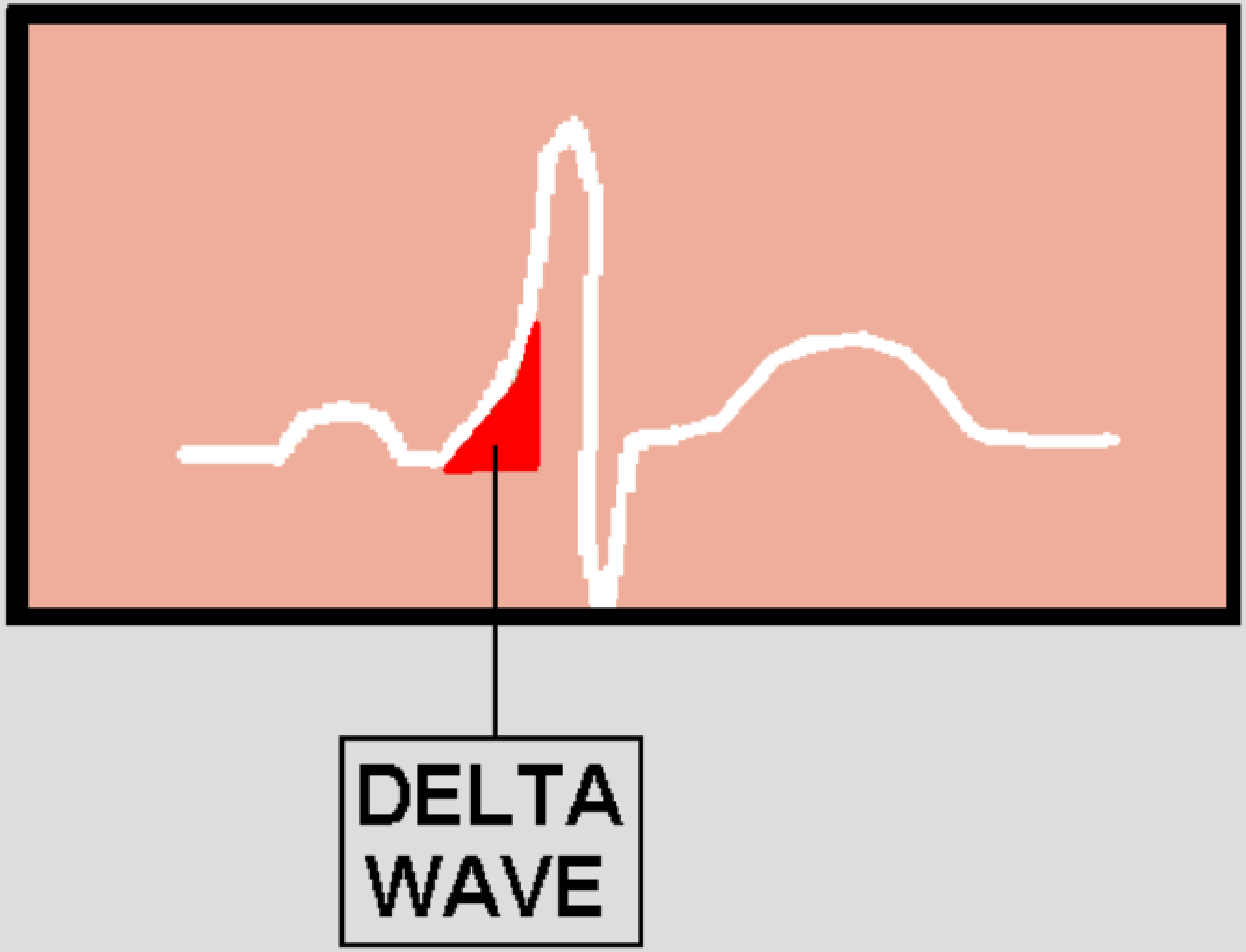


WOLFF-PARKINSON-WHITE

EKG CHARACTERISTICS

WOLFF-PARKINSON-WHITE

EKG CHARACTERISTICS



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

16 yr
Female Caucasian

Room:REC
Loc:20 Option:50

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

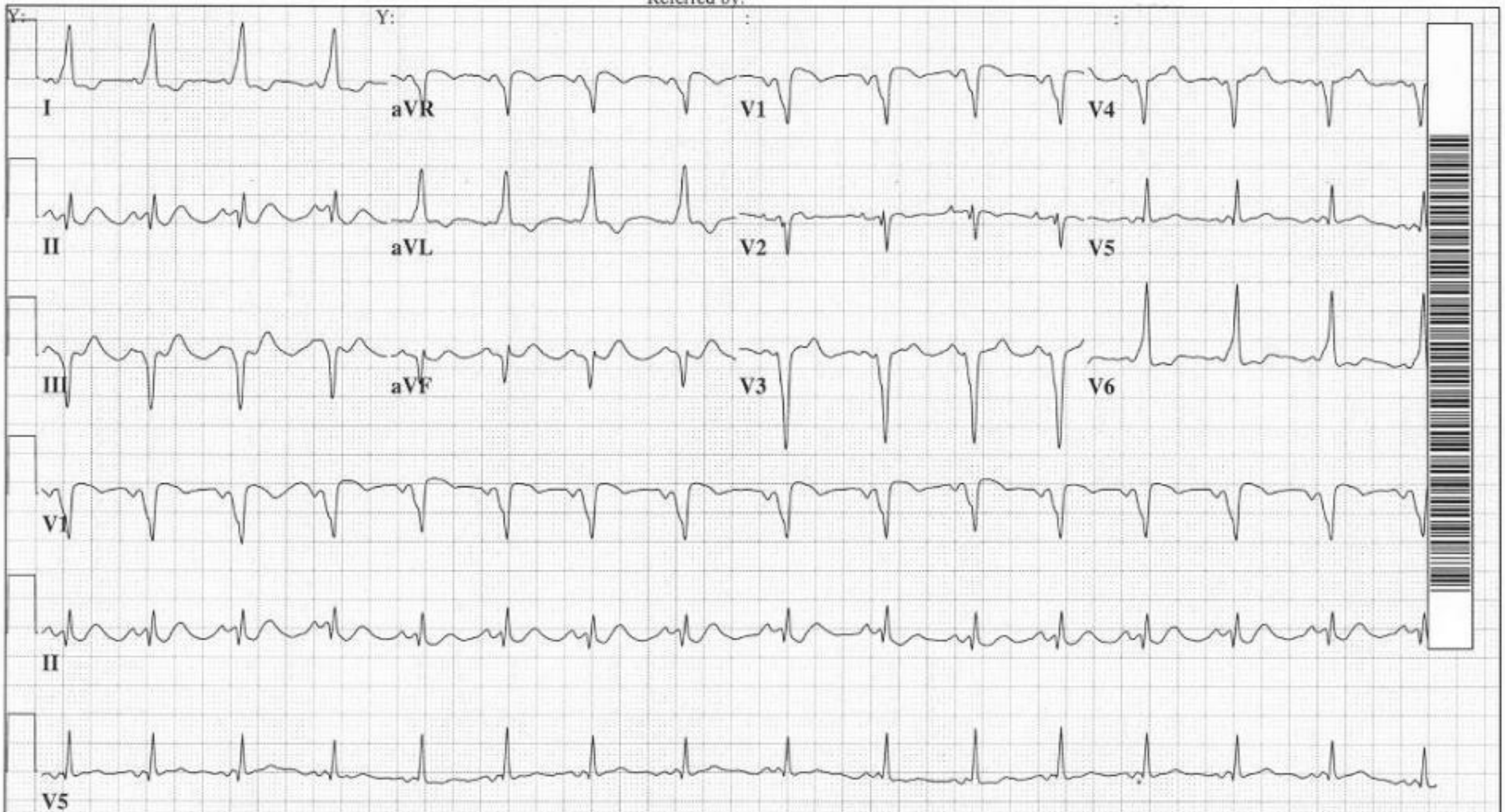
Normal sinus rhythm with sinus arrhythmia
~~Left atrial enlargement~~
~~Anterior infarct , age undetermined~~
~~Inferior infarct , age undetermined~~
~~ST & T wave abnormality, consider lateral ischemia~~
Wolff–Parkinson–White
Abnormal ECG
No previous ECGs available

History:Unknown
Technician: DP
Test ind:EKG

EKG CLASS #WR030100 60783

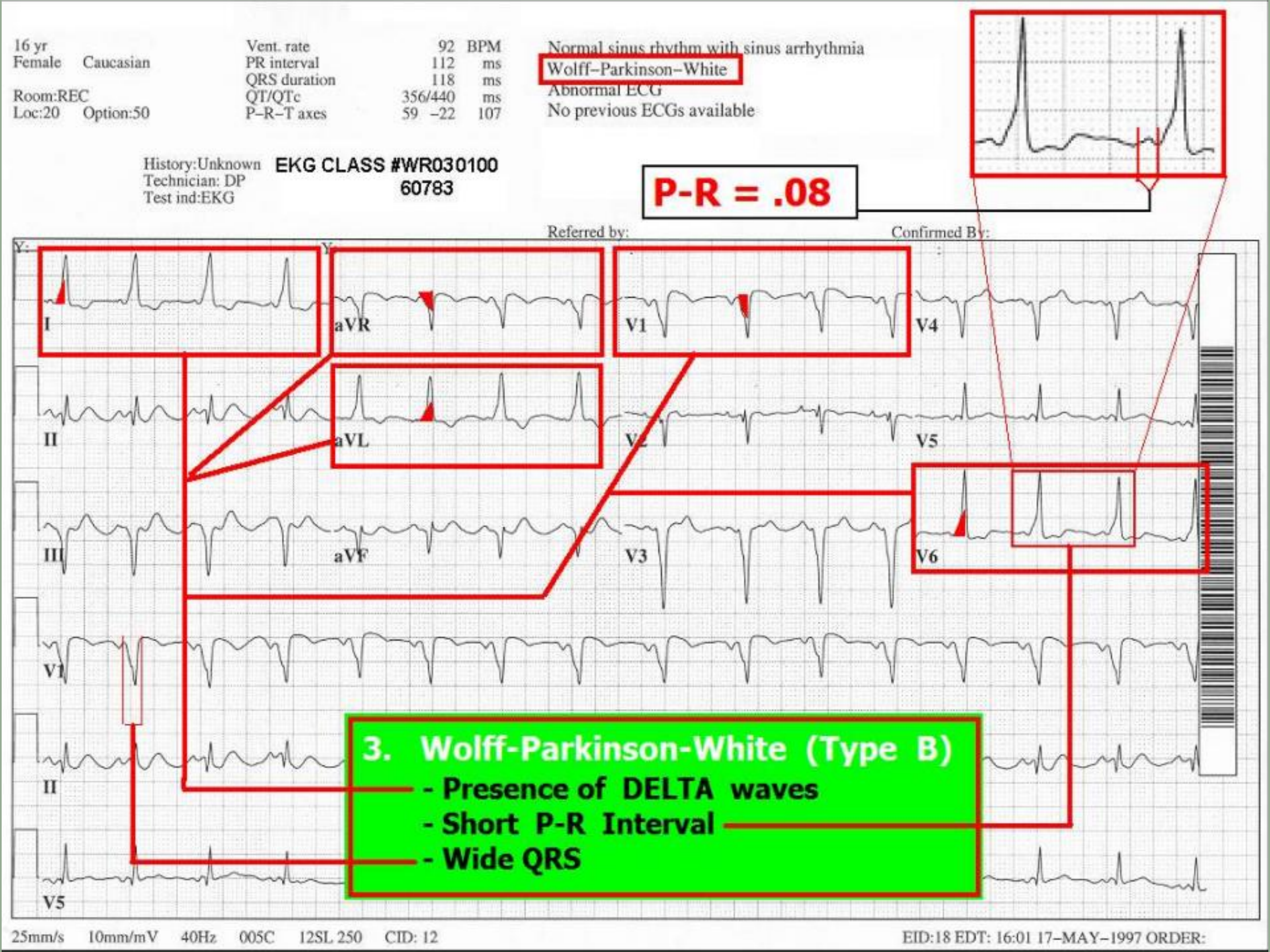
WOLFF-PARKINSON-WHITE TYPE B

Referred by:



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

EID:18 EDT: 16:01 17–MAY–1997 ORDER:


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

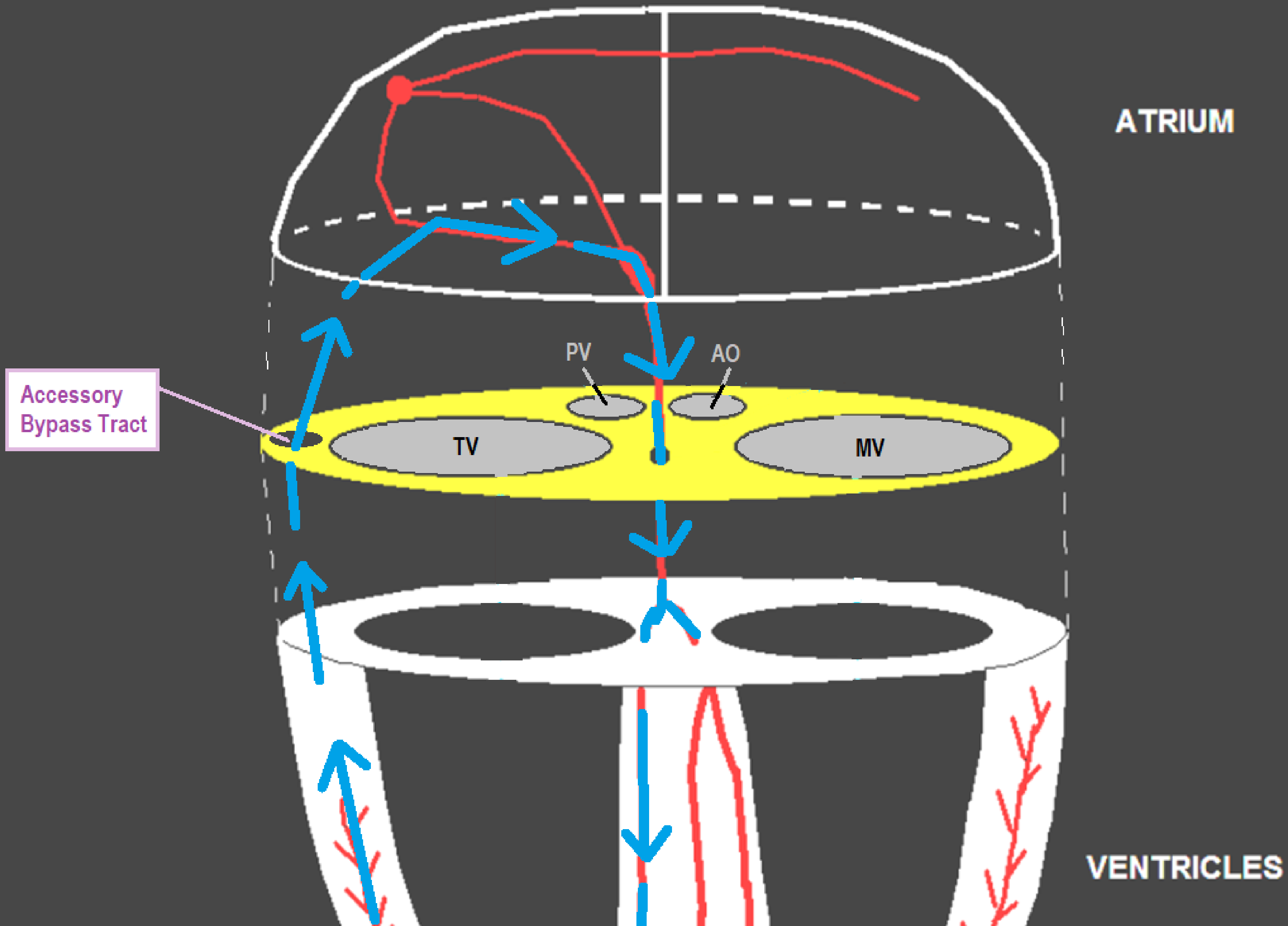
Patient Profile: Wolff-Parkinson-White:
- Typically Pediatric / Young Adult
- May not know they have it
- May experience episodes of "palpitations" or "Very Fast Heartbeat."

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

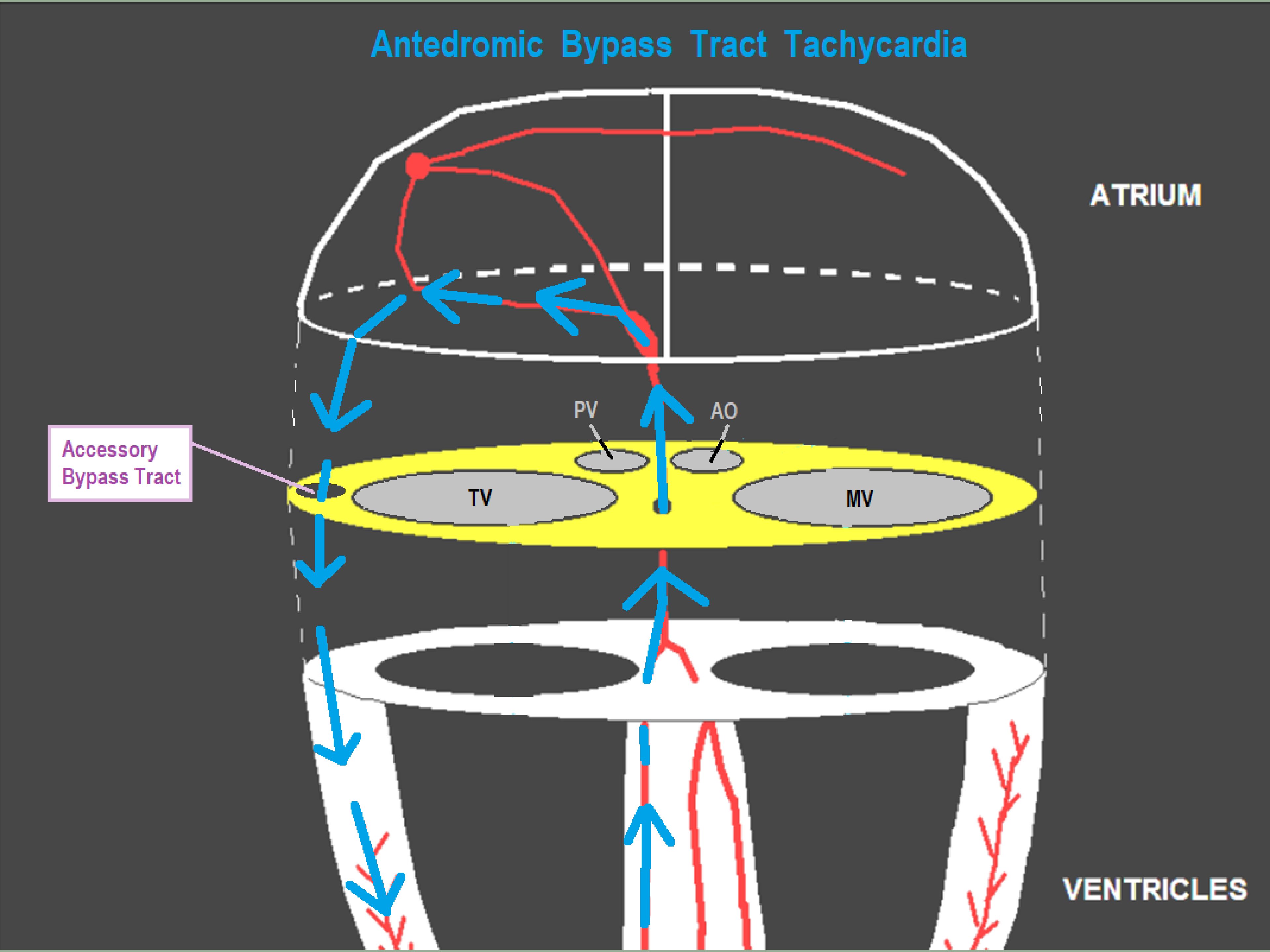


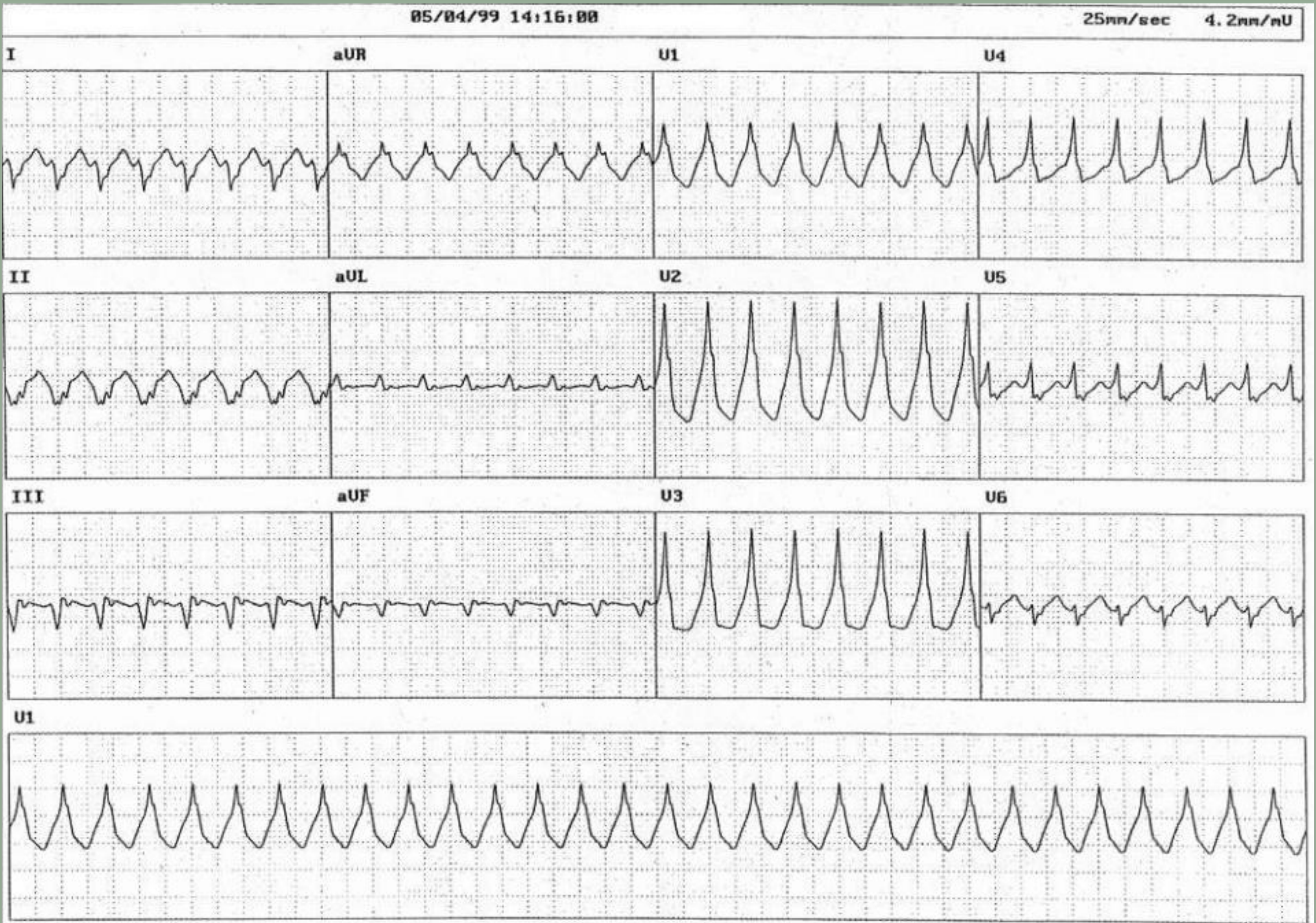
SVT

- 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
ATRIUM
PV
AO
Accessory
Bypass Tract
TV
MV
VENTRICLES



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-

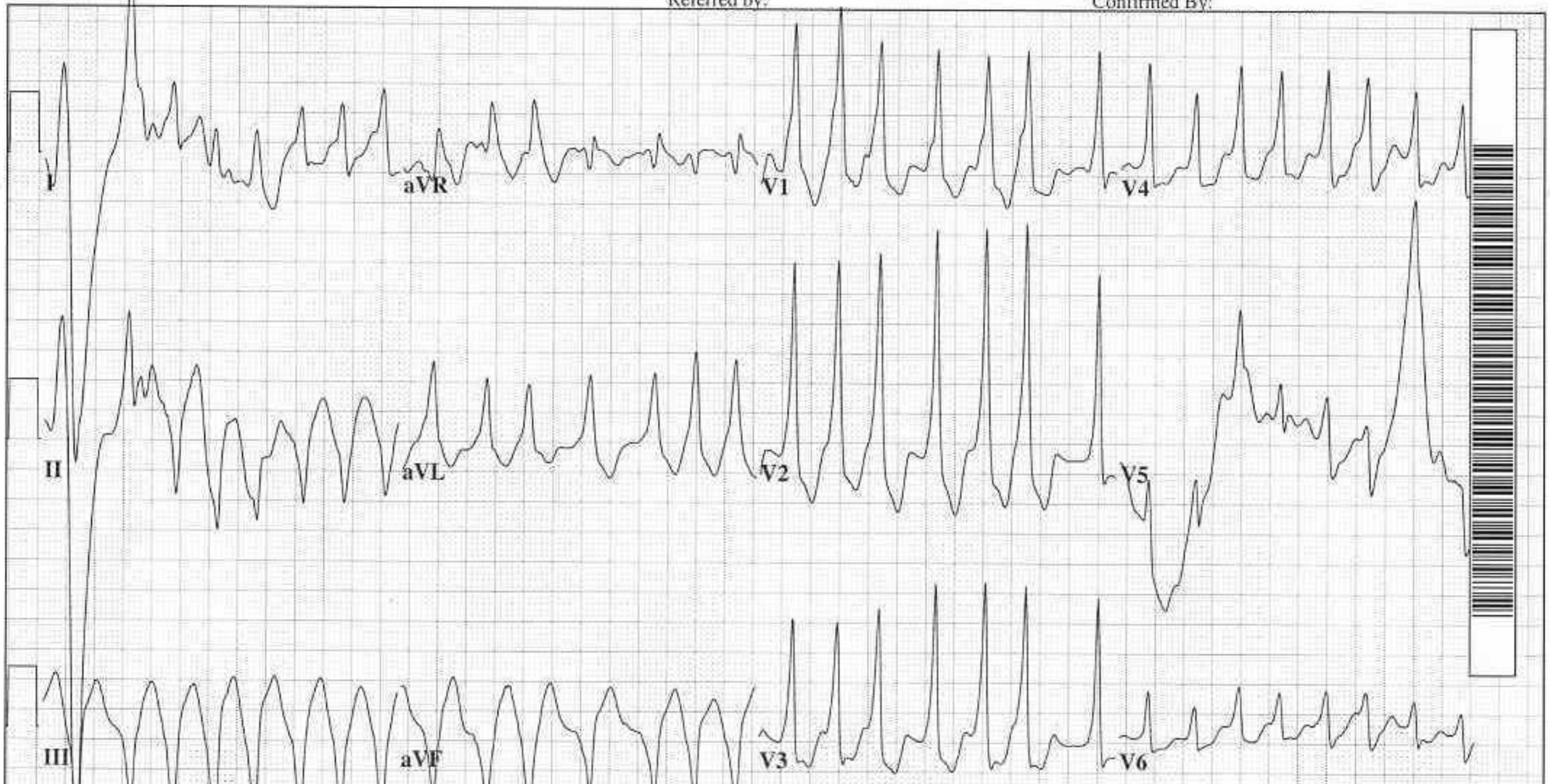
37 yr		Vent. rate	180	BPM
Male	Caucasian	PR interval	*	ms
		QRS duration	148	ms
Room:OP		QT/QTc	284/491	ms
Loc:8	Option:16	P-R-T axes	* -77	103

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

Med: Unknown

Referred by:

Confirmed By:



NOTE IRREGULARITY OF RHYTHM – (SUGGESTIVE OF AFIB)

LOOK FOR DELTA WAVES

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

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.***

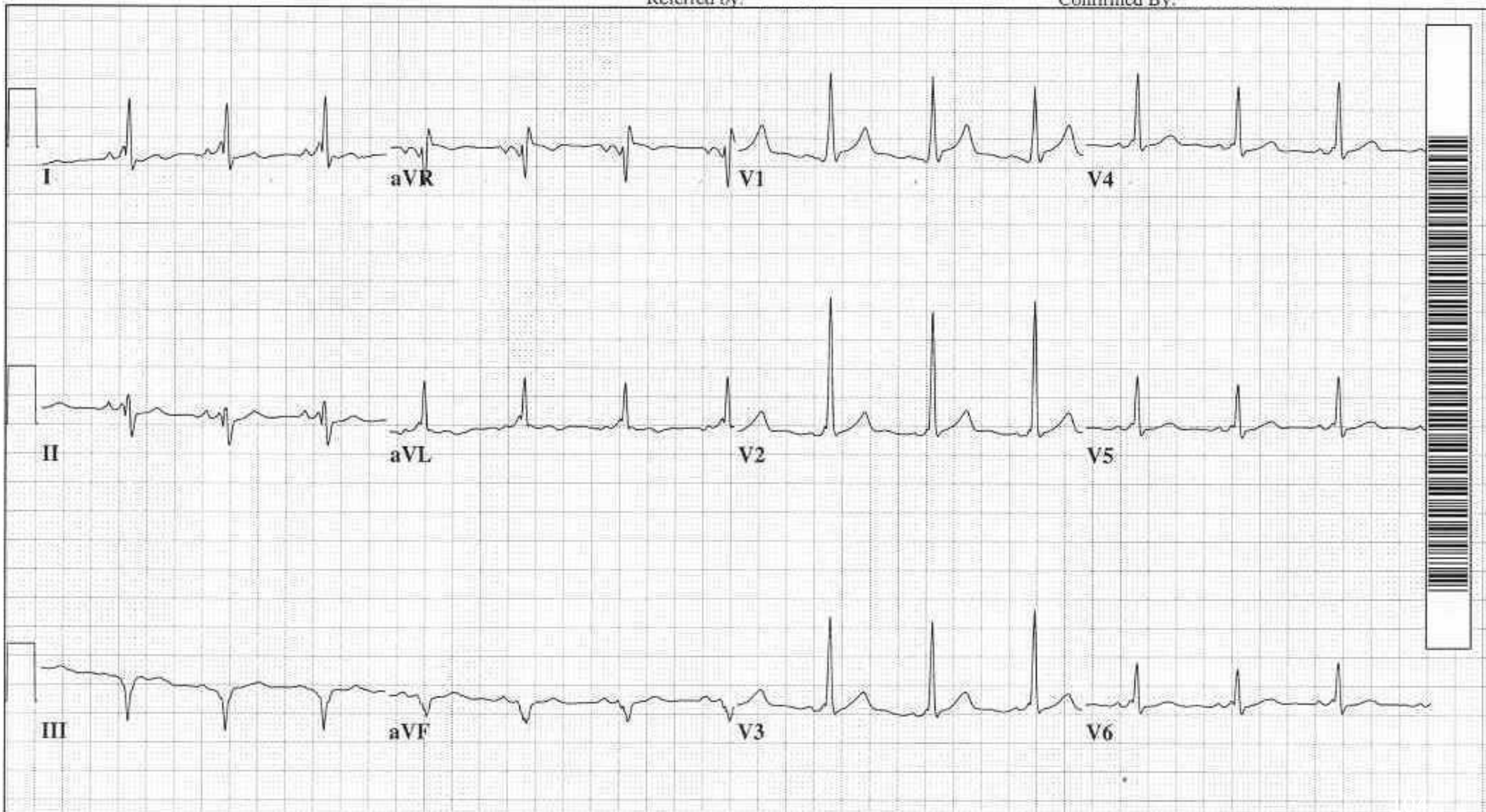
37 y/o male

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

A 12 Lead EKG obtained revealed . . . . .

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

Referred by: Confirmed By:



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

ST. JOSEPH'S HOSPITAL-

37 yr		Vent. rate	180	BPM	WIDE QRS TACHYCARDIA – POSSIBLE VT
Male	Caucasian	PR interval	*	ms	Right bundle branch block PATTERN
		QRS duration	148	ms	Abnormal ECG
Room:OP		QT/QTc	284/491	ms	
Loc:8	Option:16	P-R-T axes	* −77	103	

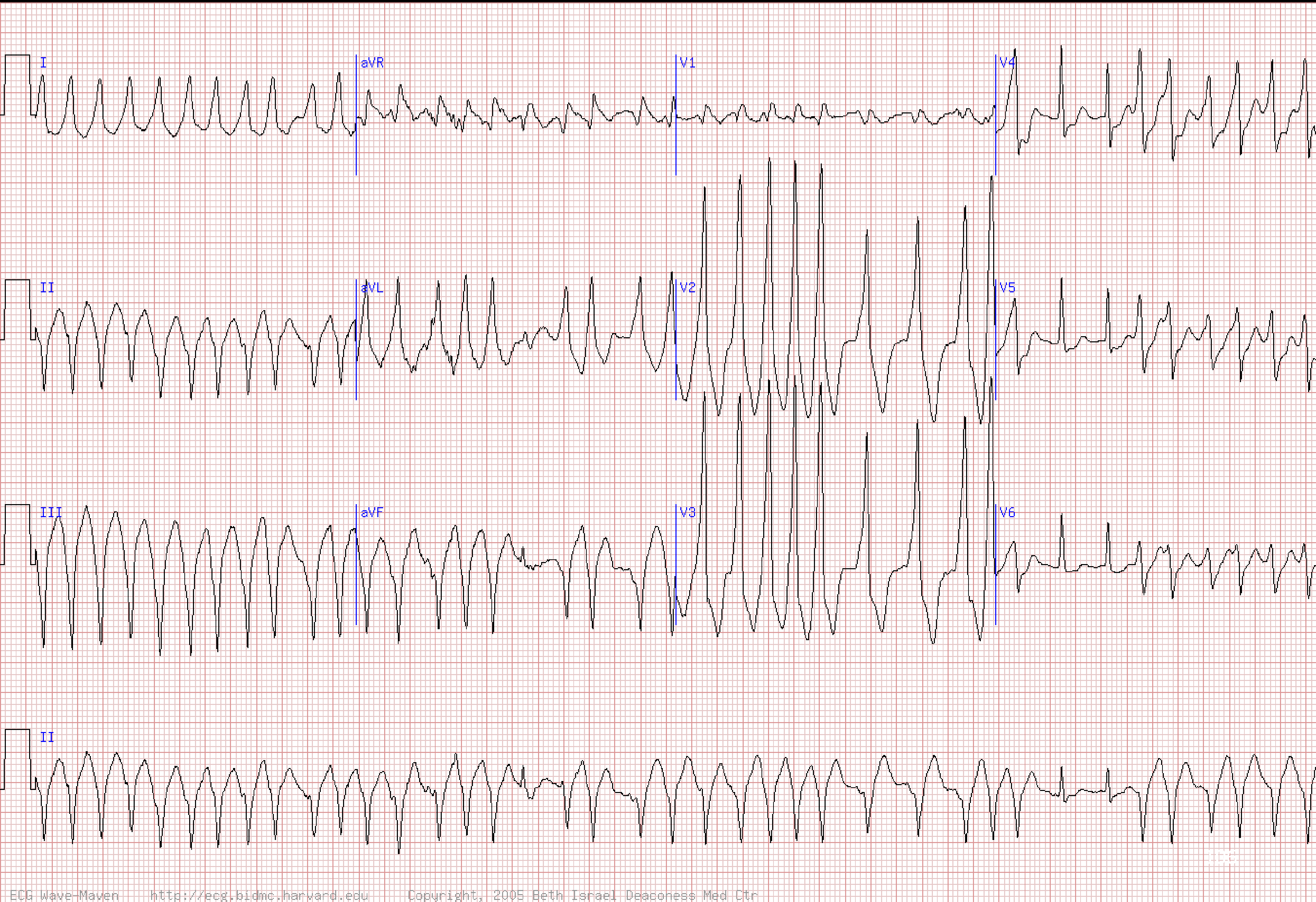
Med: Unknown

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

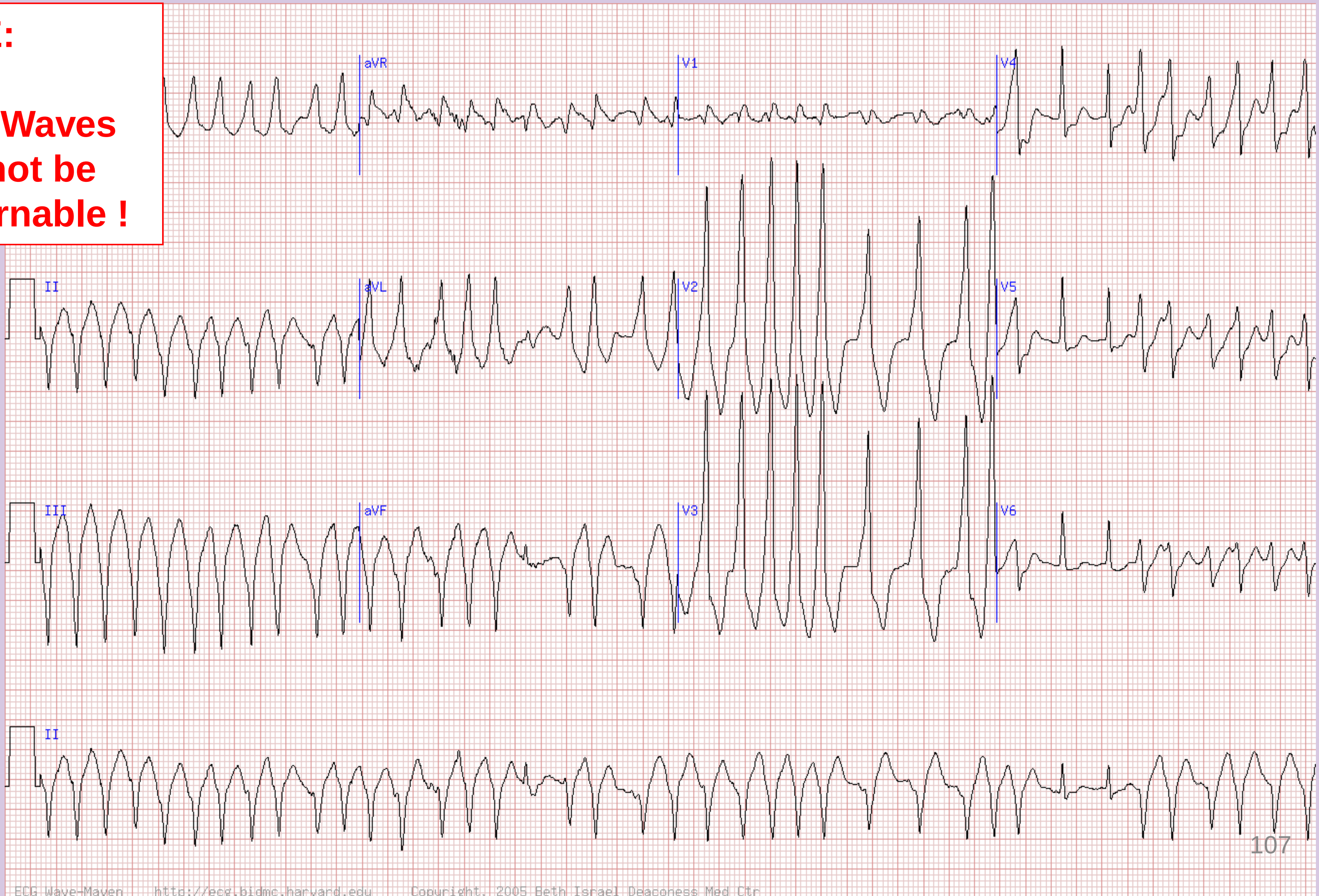


CHARACTERISTICS of W-P-W with Afib & RVR:

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

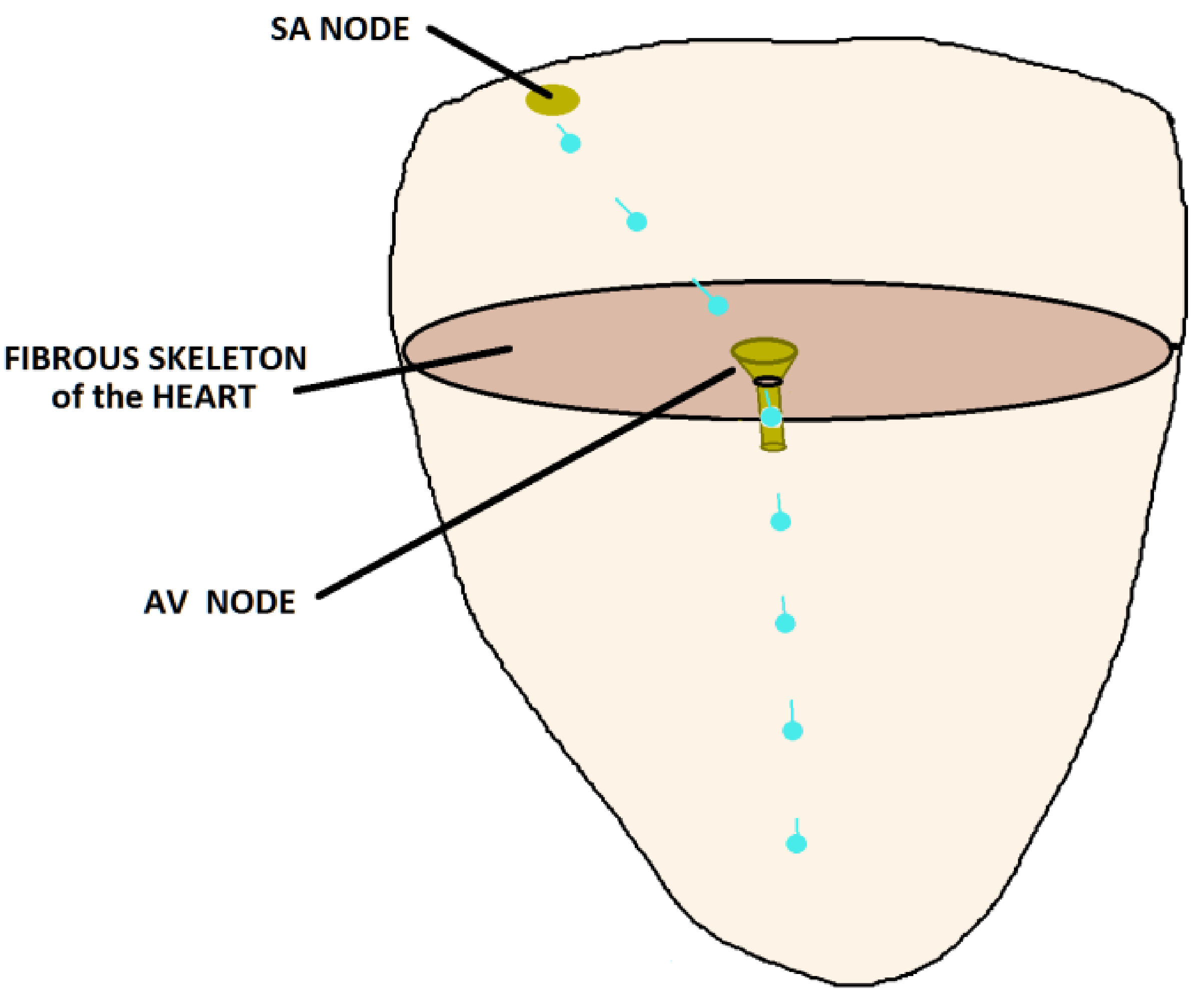
NOTE:

Delta Waves may not be discernable !

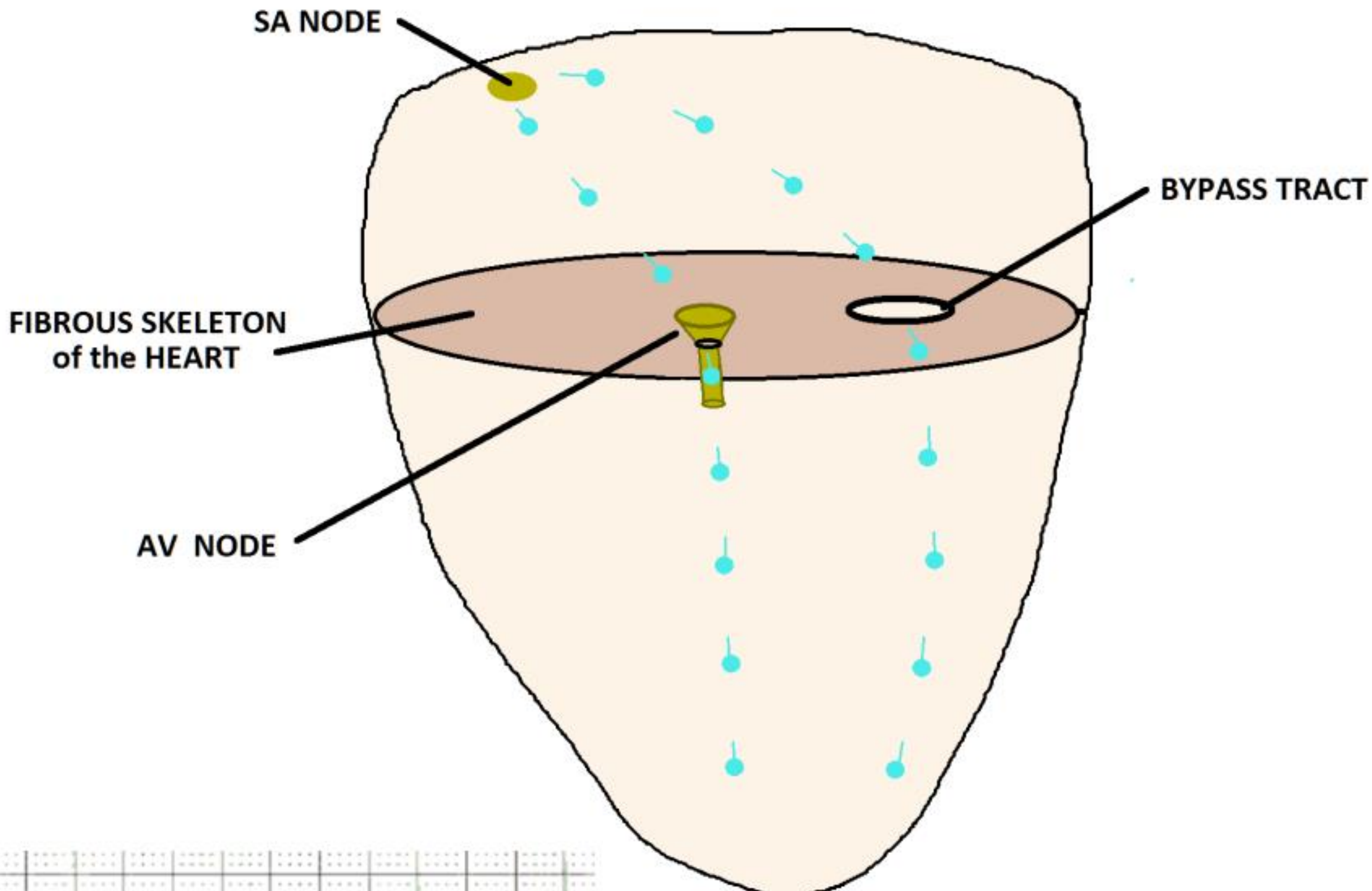


NO AV NODAL BLOCKERS
(e.g. ADENOSINE, CALCIUM CHANNEL BLOCKERS)
FOR WIDE COMPLEX TACHYCARDIAS THAT COULD BE ATRIAL FIBRILLATION with Pre-Excitation (W-P-W)

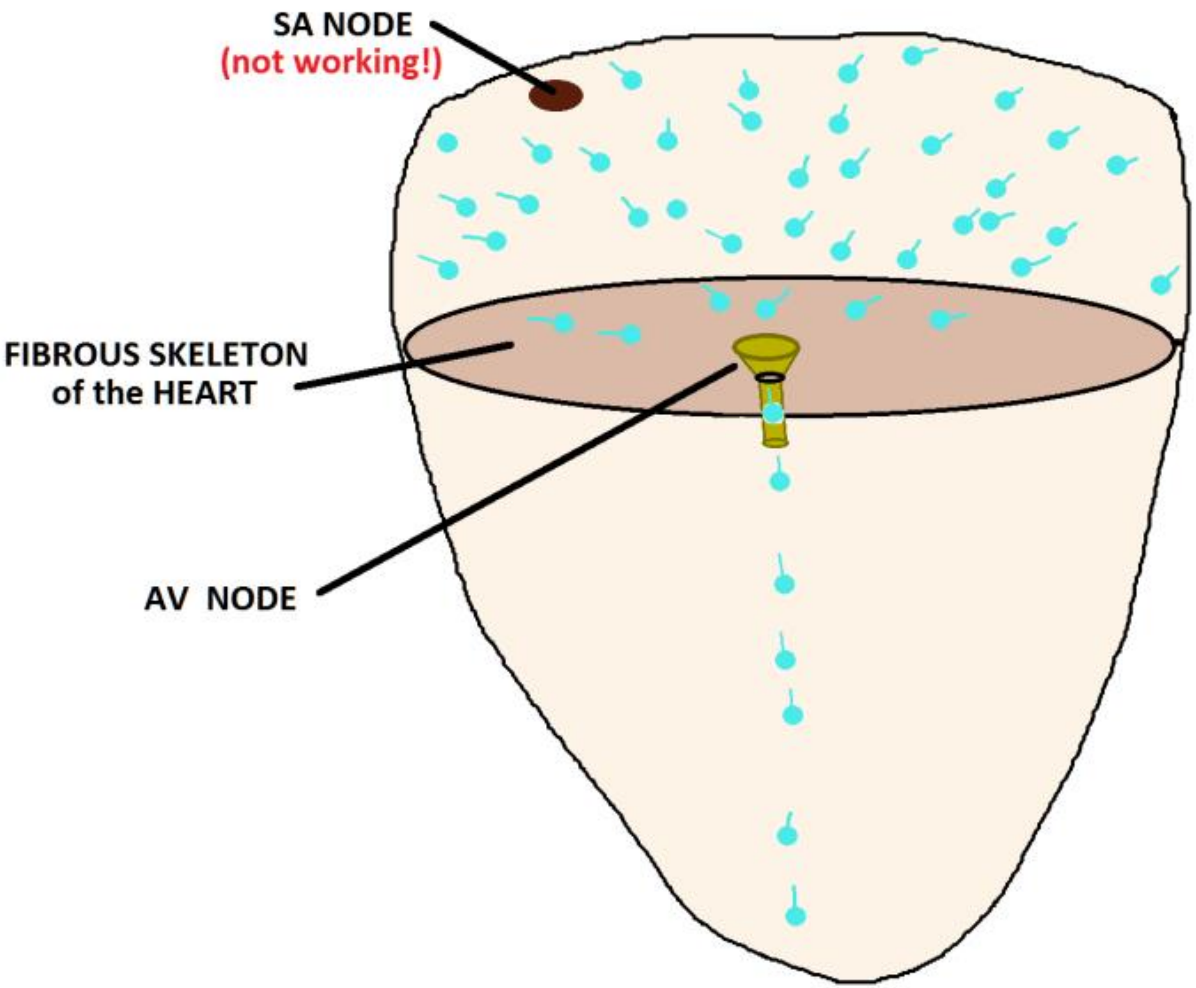
Normal Sinus Rhythm



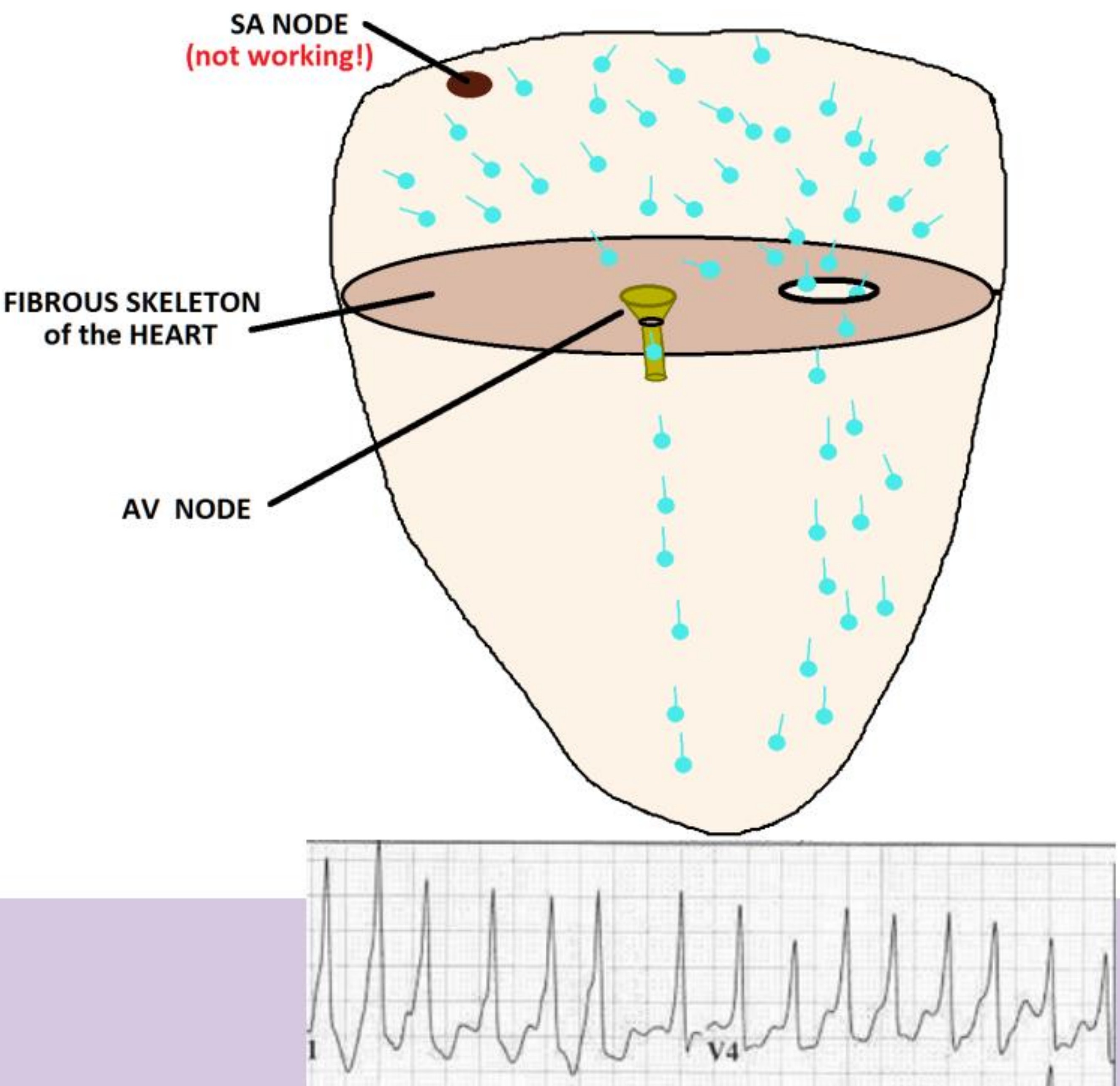
Normal Sinus Rhythm with Wolff-Parkinson White



Atrial Fibrillation



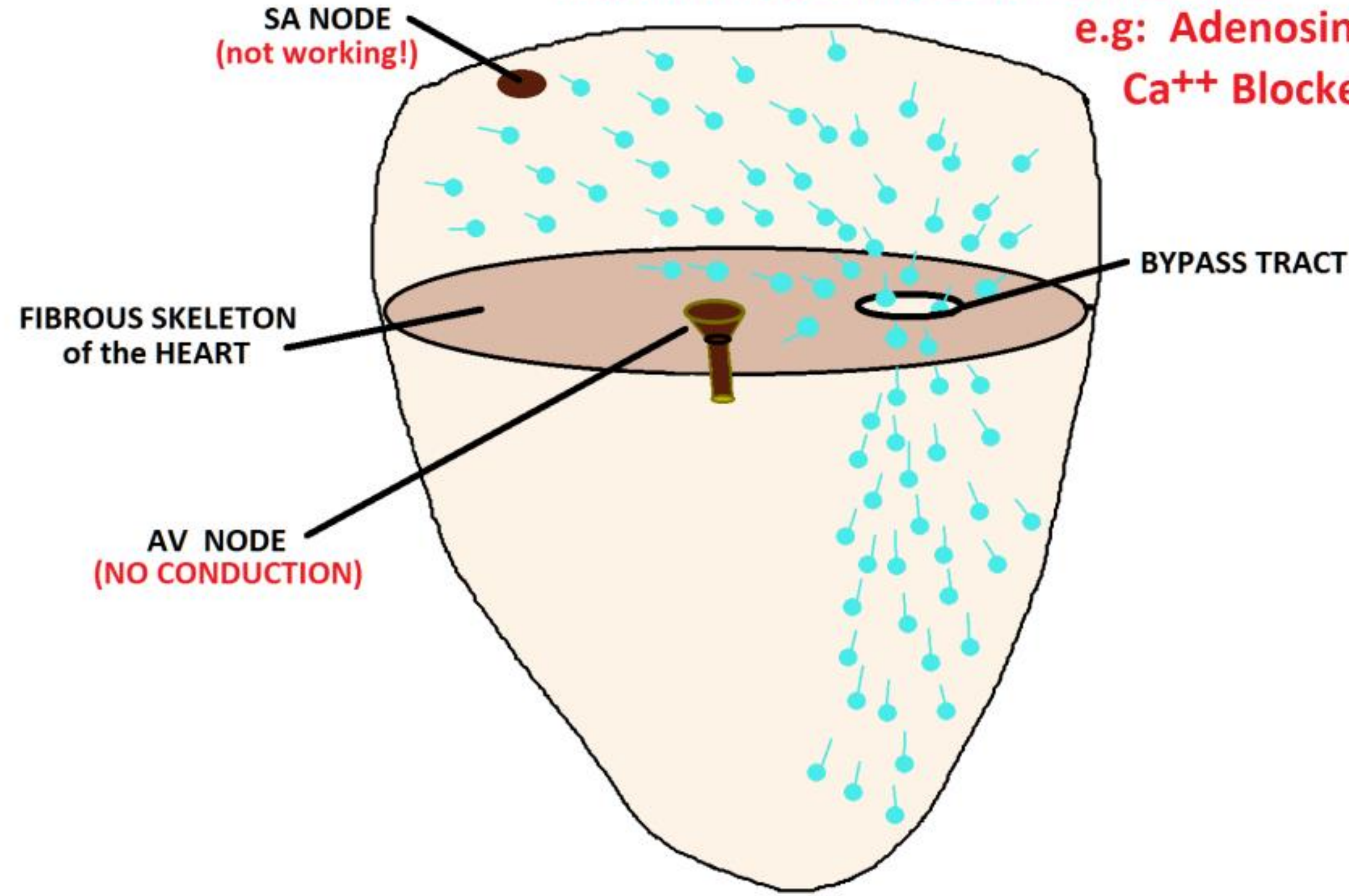
Atrial Fibrillation with Wolff-Parkinson White

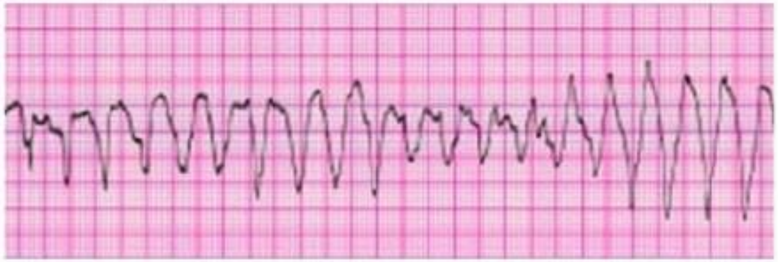


Atrial Fibrillation with Wolff-Parkinson White

with AV NODAL BLOCKING AGENTS

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

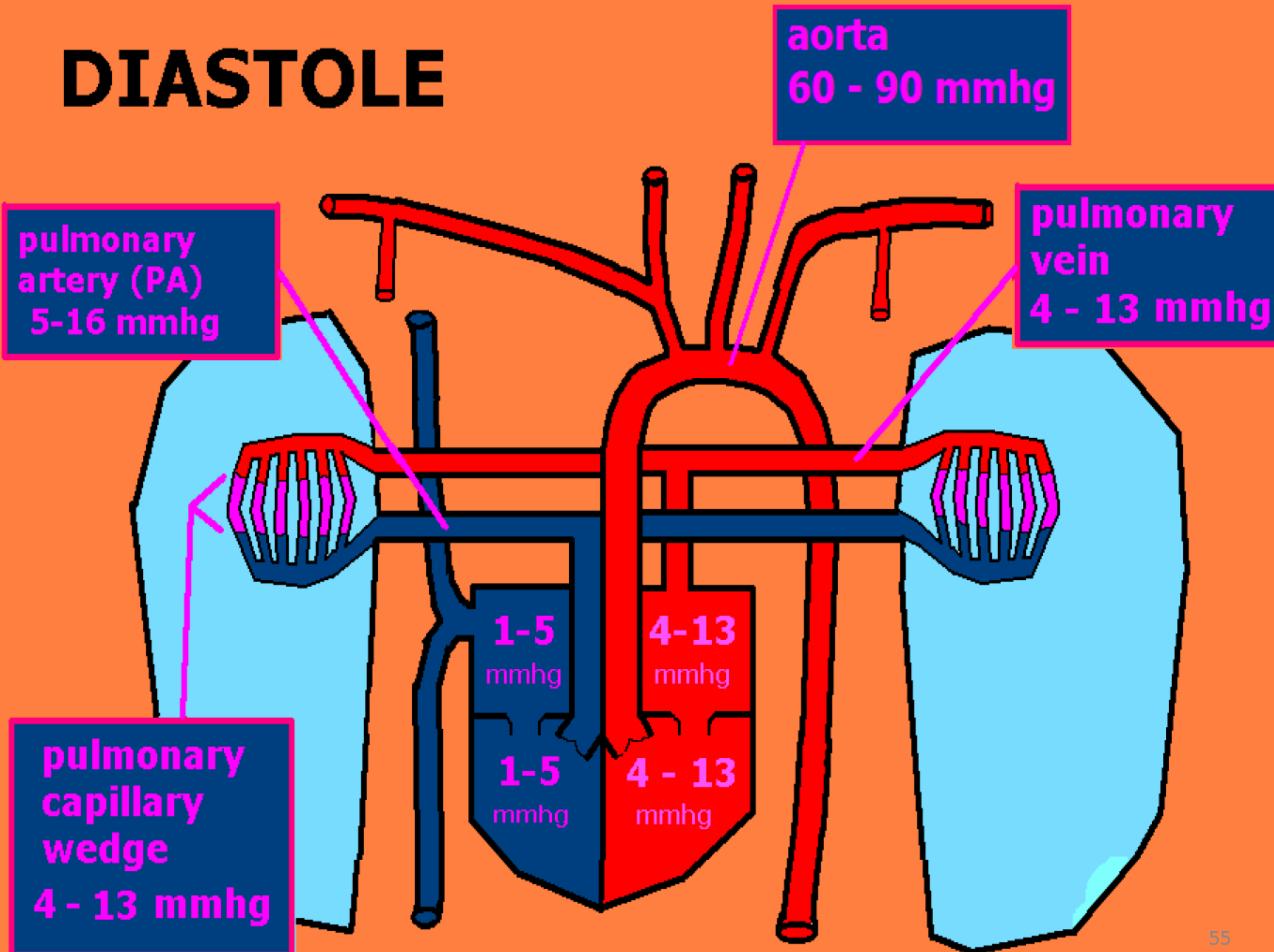




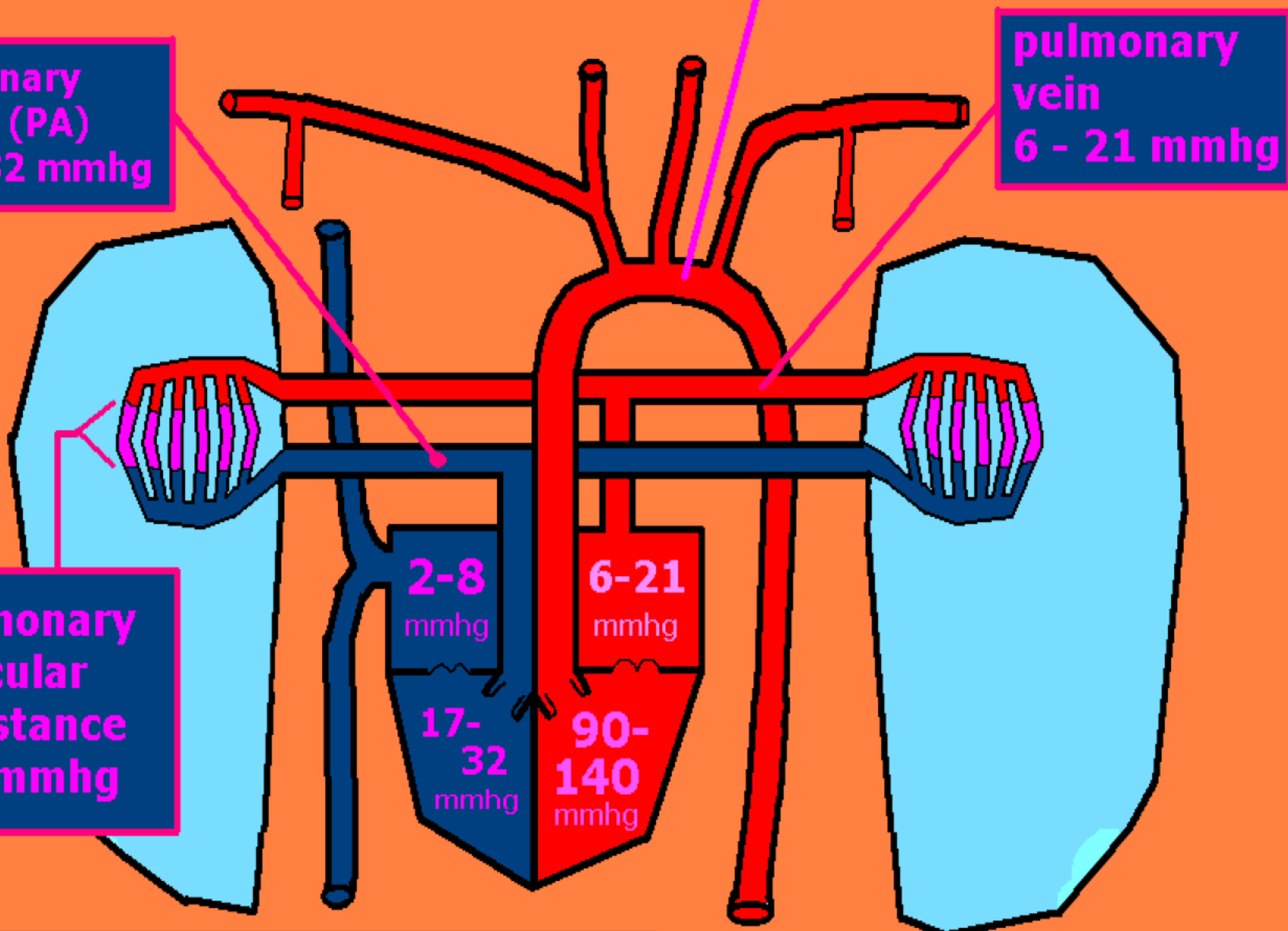
Cardiac A & P "101" . . . .

- Action Potential of Ventricular Muscle Cells
- Rapid basic review heart structure
- Electrical System
- Fibrous Skeleton of the Heart
- Pathophysiology of Accessory Bypass Tracts (cause of Wolff-Parkinson-White Syndrome)
- **Normal Pressures with Heart and Lungs**

DIASTOLE


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

VENTRICULAR SYSTOLE

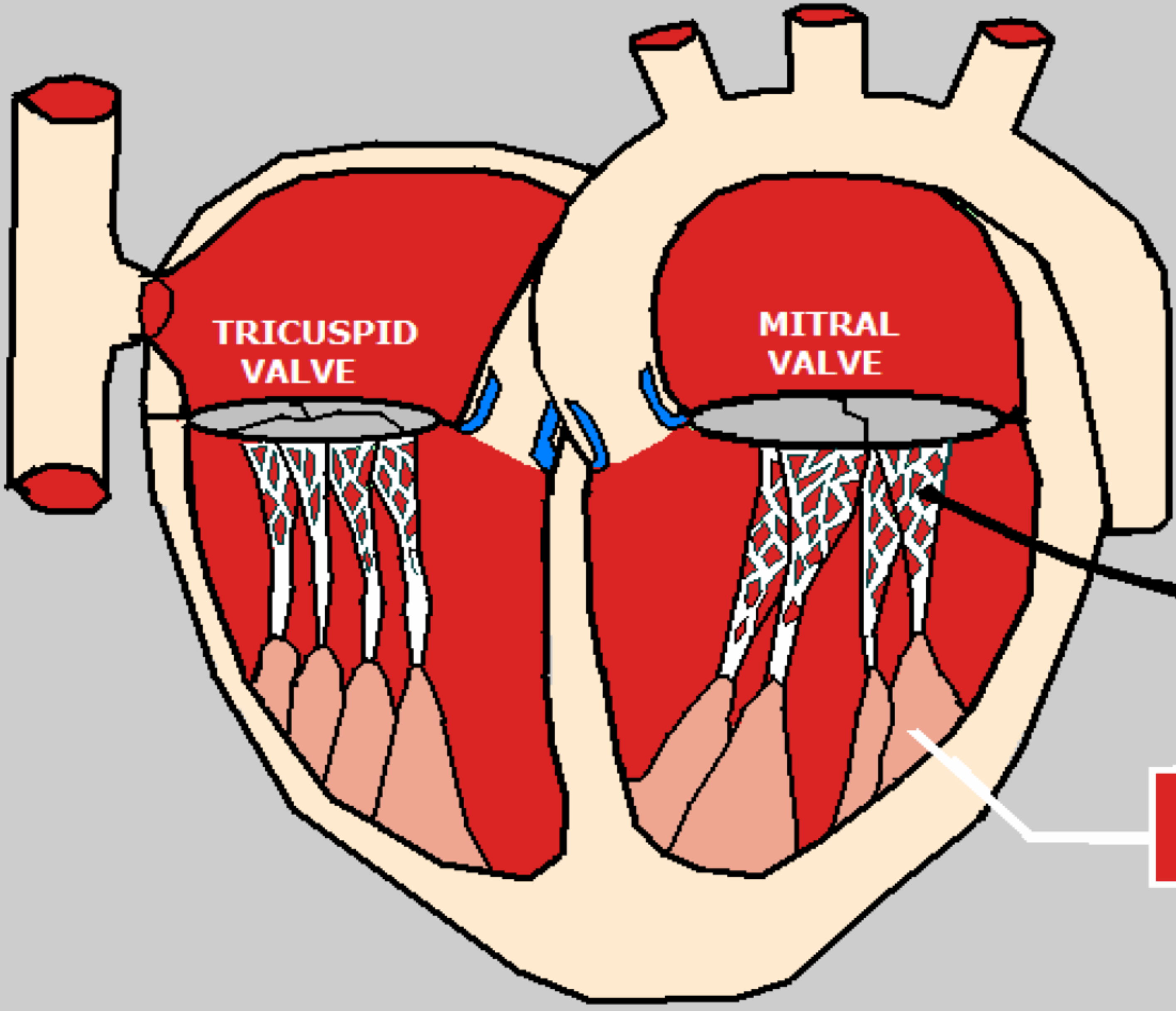


HEMODYNAMIC DATA from:
"The Cardiac Catheterization Handbook," Morton J. Kearn, MD

Cardiac A & P "101" . . . .

- Action Potential of Ventricular Muscle Cells
- Rapid basic review heart structure
- Electrical System
- Fibrous Skeleton of the Heart
- Pathophysiology of Accessory Bypass Tracts (cause of Wolff-Parkinson-White Syndrome)
- Normal Pressures with Heart and Lungs
- **Heart Valves**

ATRIO-VENTRICULAR VALVES



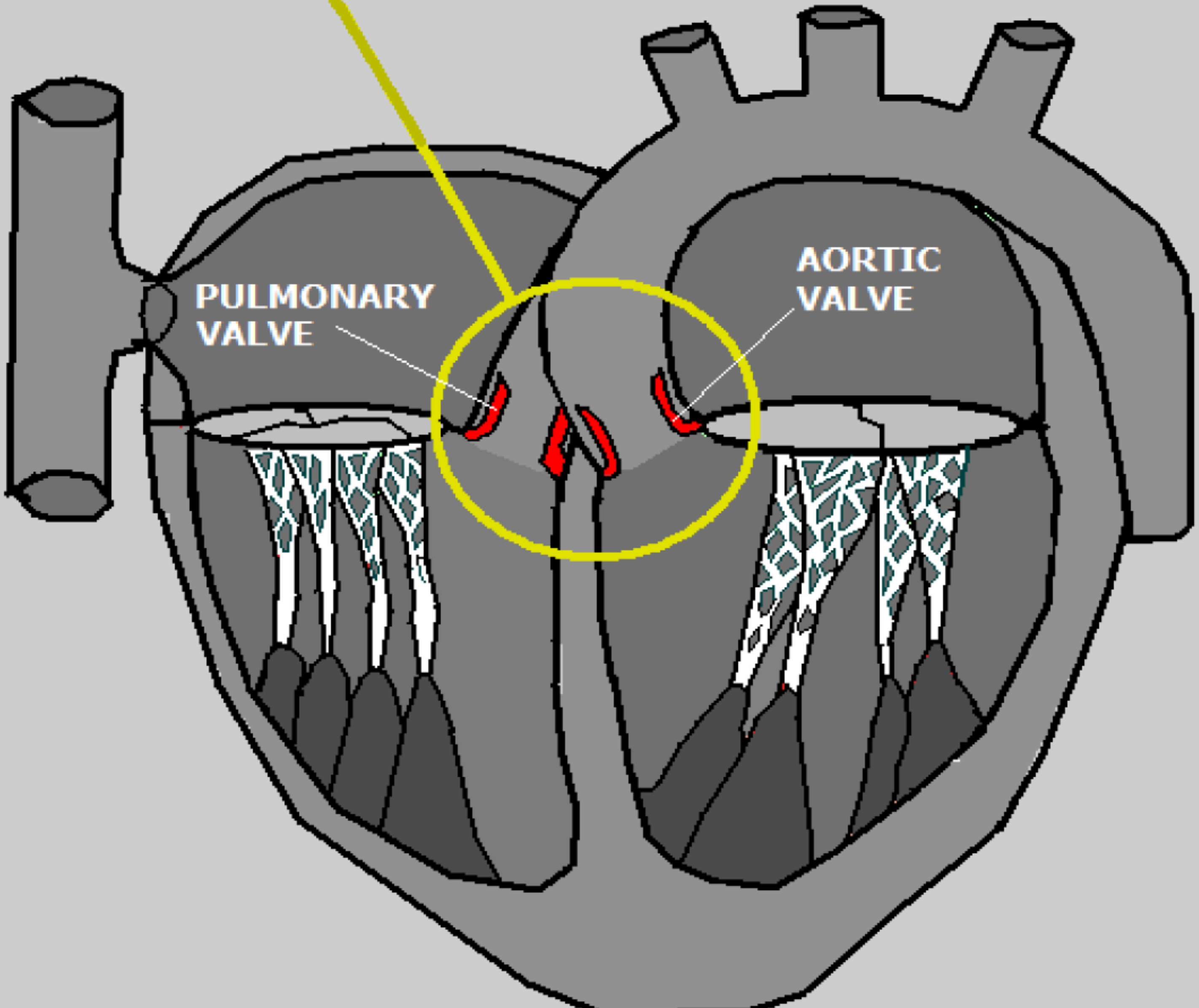
ARE SECURED TO THE ENDO-CARDIAL SURFACE BY THE

CHORDAE TENDINEAE

AND THE

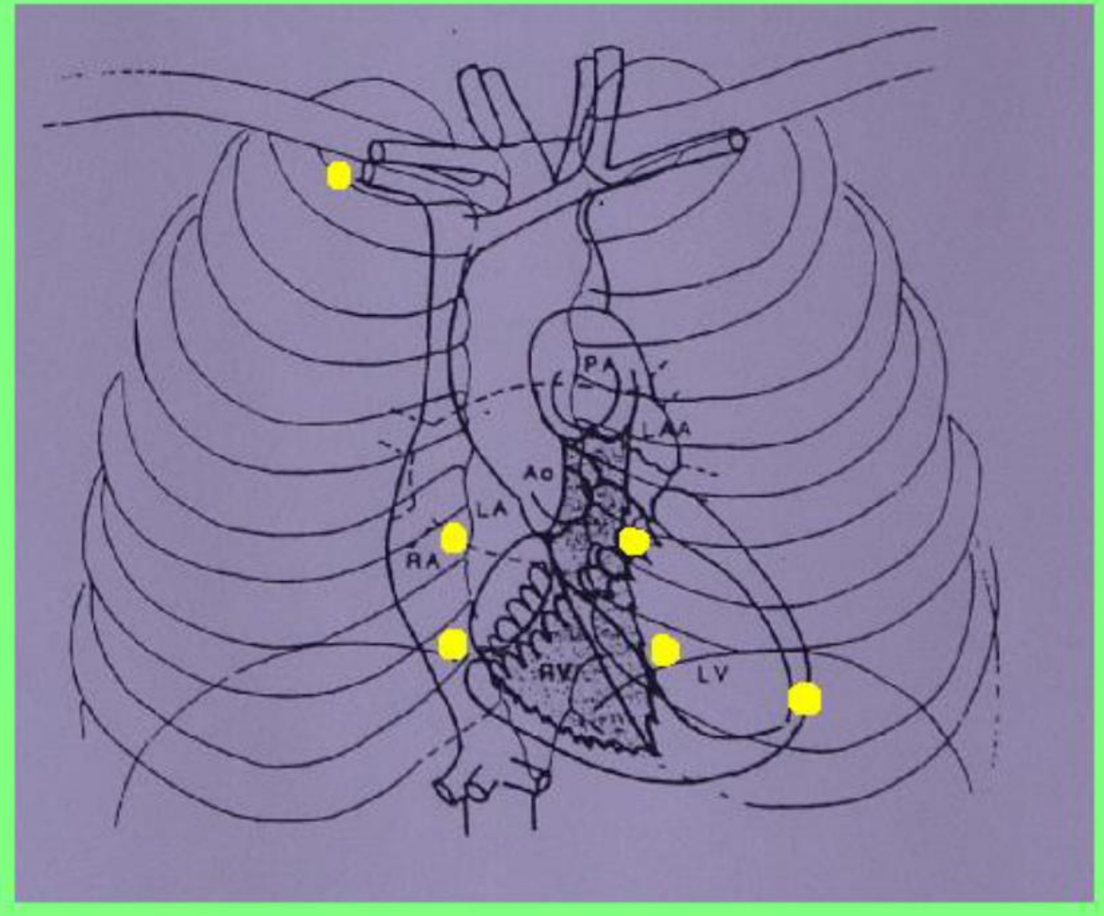
PAPILLARY MUSCLES

THE SEMILUNAR VALVES



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.

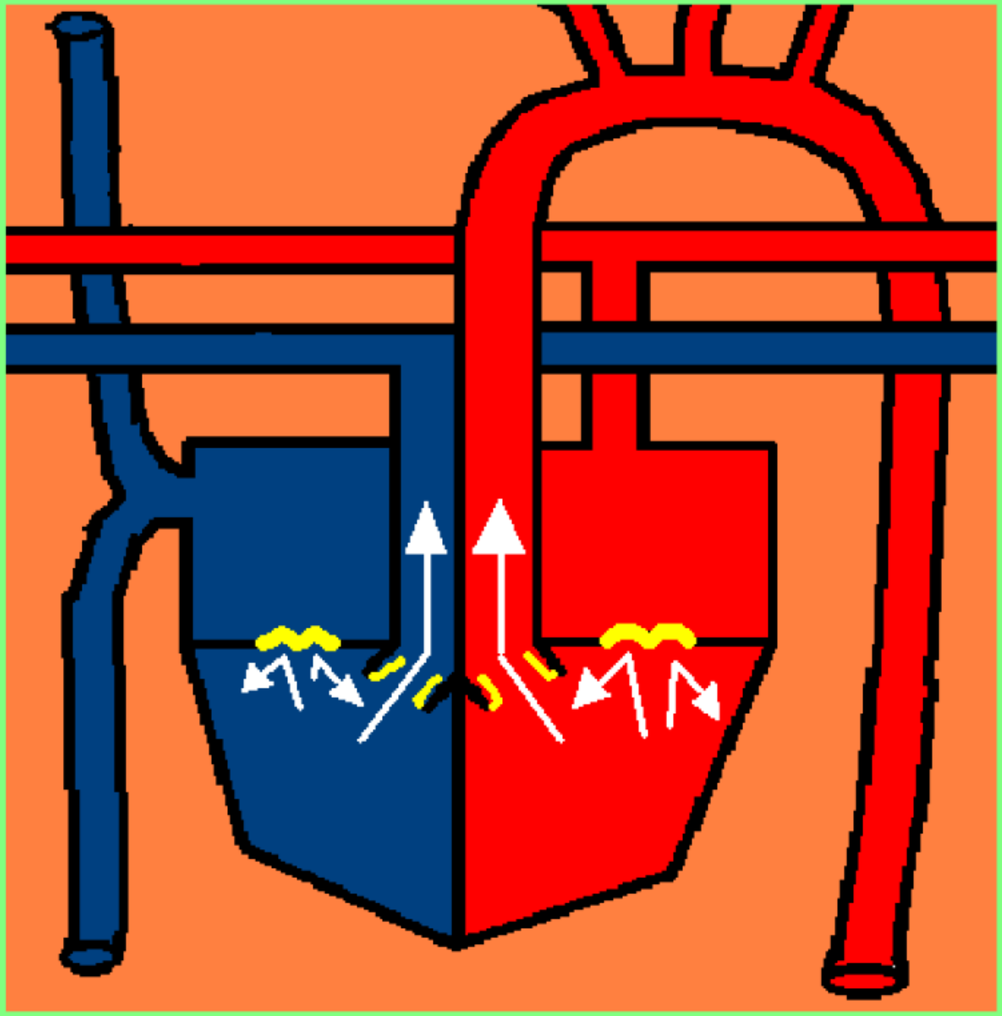
THERE ARE TWO NORMAL HEART SOUNDS, KNOWN AS: **S-1** and **S-2**

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

HEART SOUNDS ASSESSMENT

S-1
BEGINNING
OF
SYSTOLE.

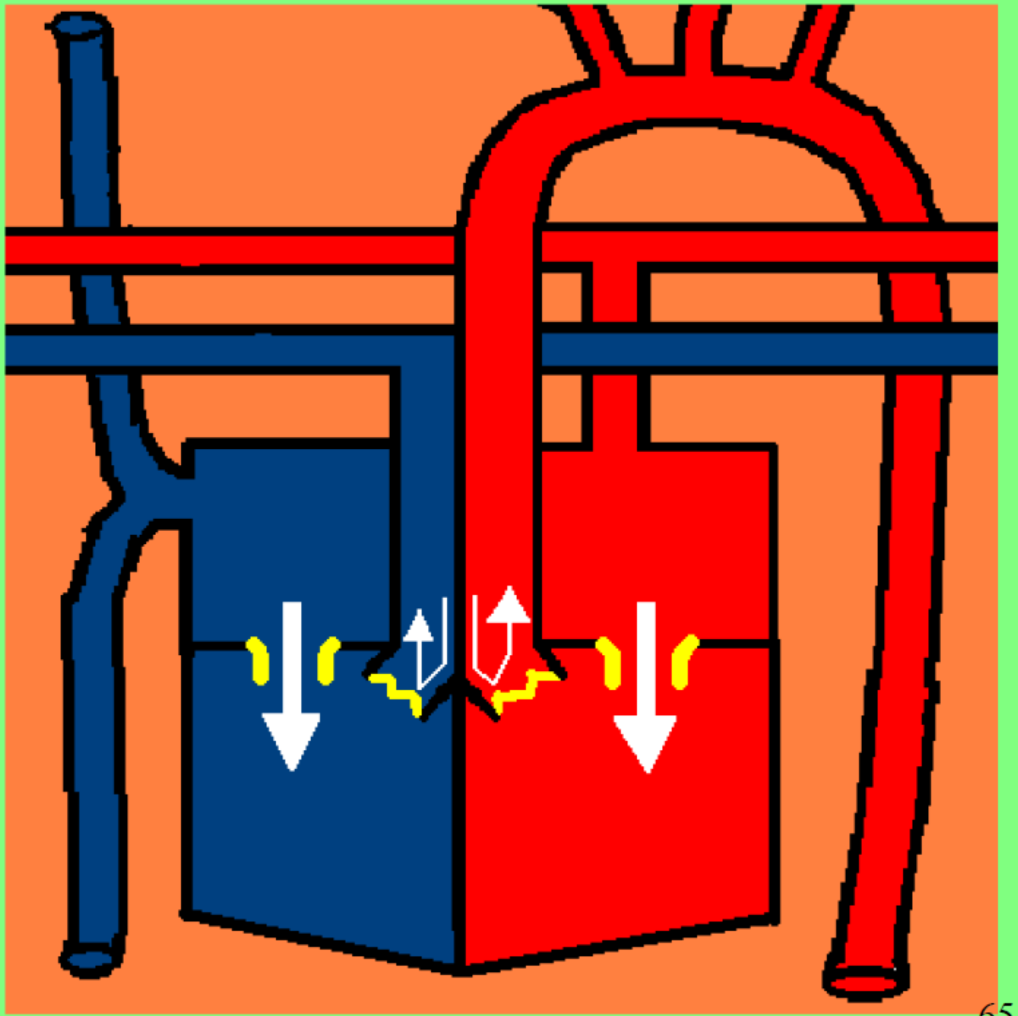
SOUND OF
THE
MITRAL
AND
TRICUSPID
VALVES
CLOSING.



HEART SOUNDS ASSESSMENT

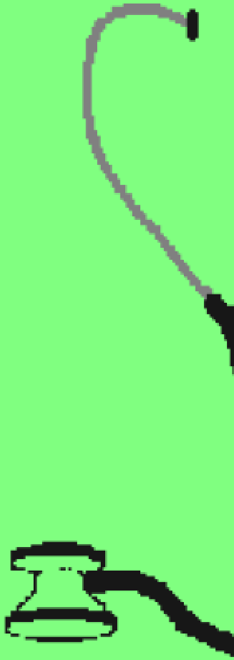
S-2 OCCURS AT THE END OF SYSTOLE (THE BEGINNING OF DIASTOLE).

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



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

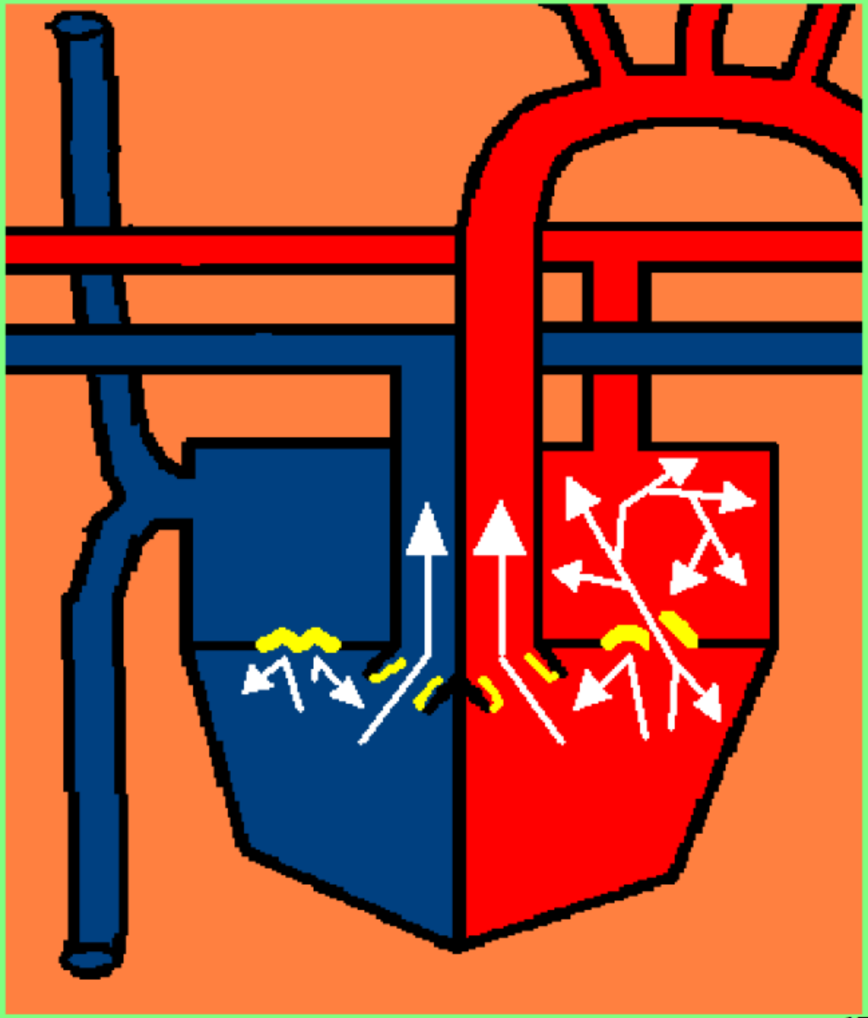
S-1 MURMUR SOUNDS LIKE:



"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).

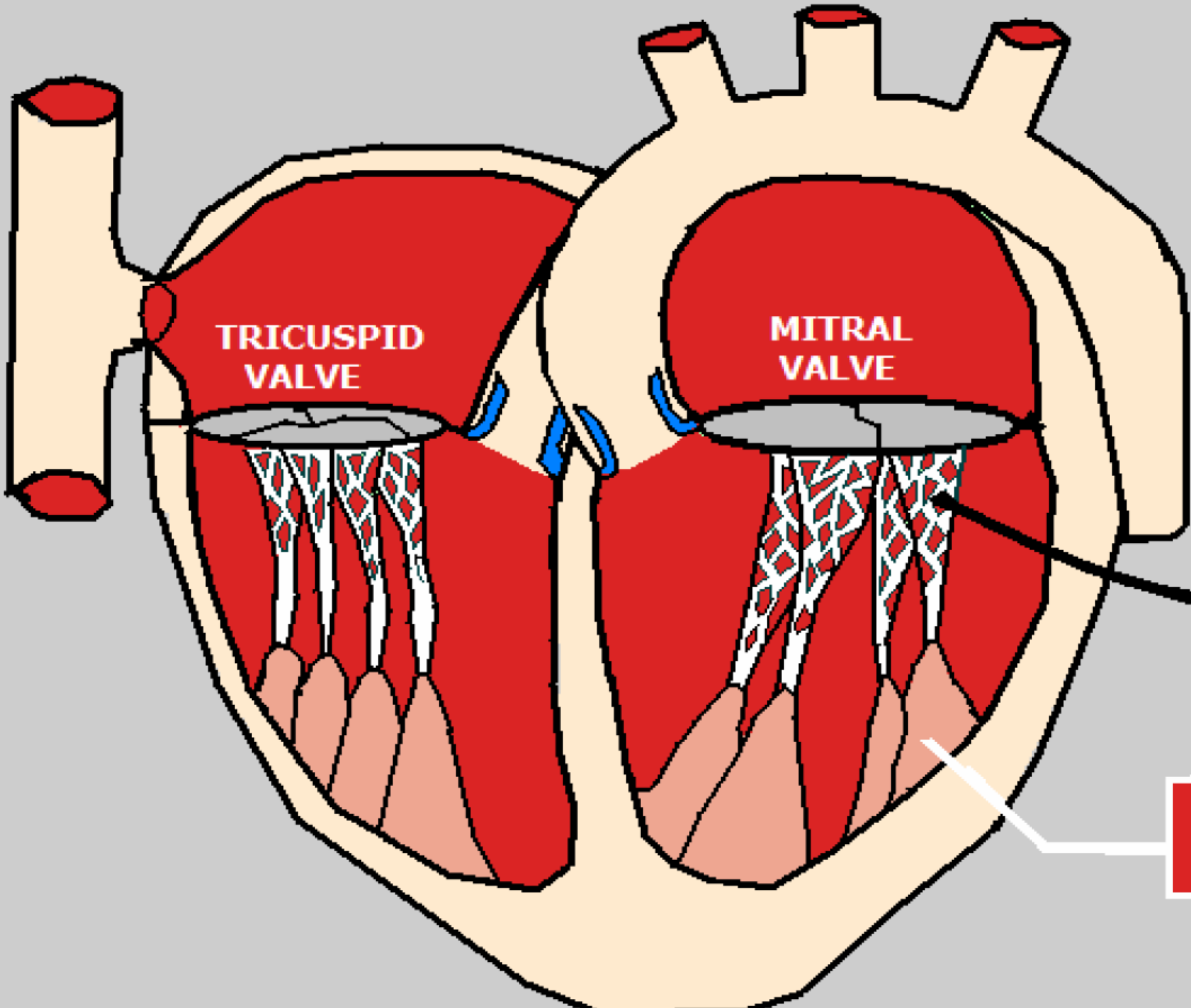
ACUTE Mitral Valve REGURGITATION can be caused by EXTENSIVE "Multi-Site" Myocardial Infarction and Necrosis – which results in PAPILLARY MUSCLE NECROSIS and PAPILLARY MUSCLE TEAR.

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

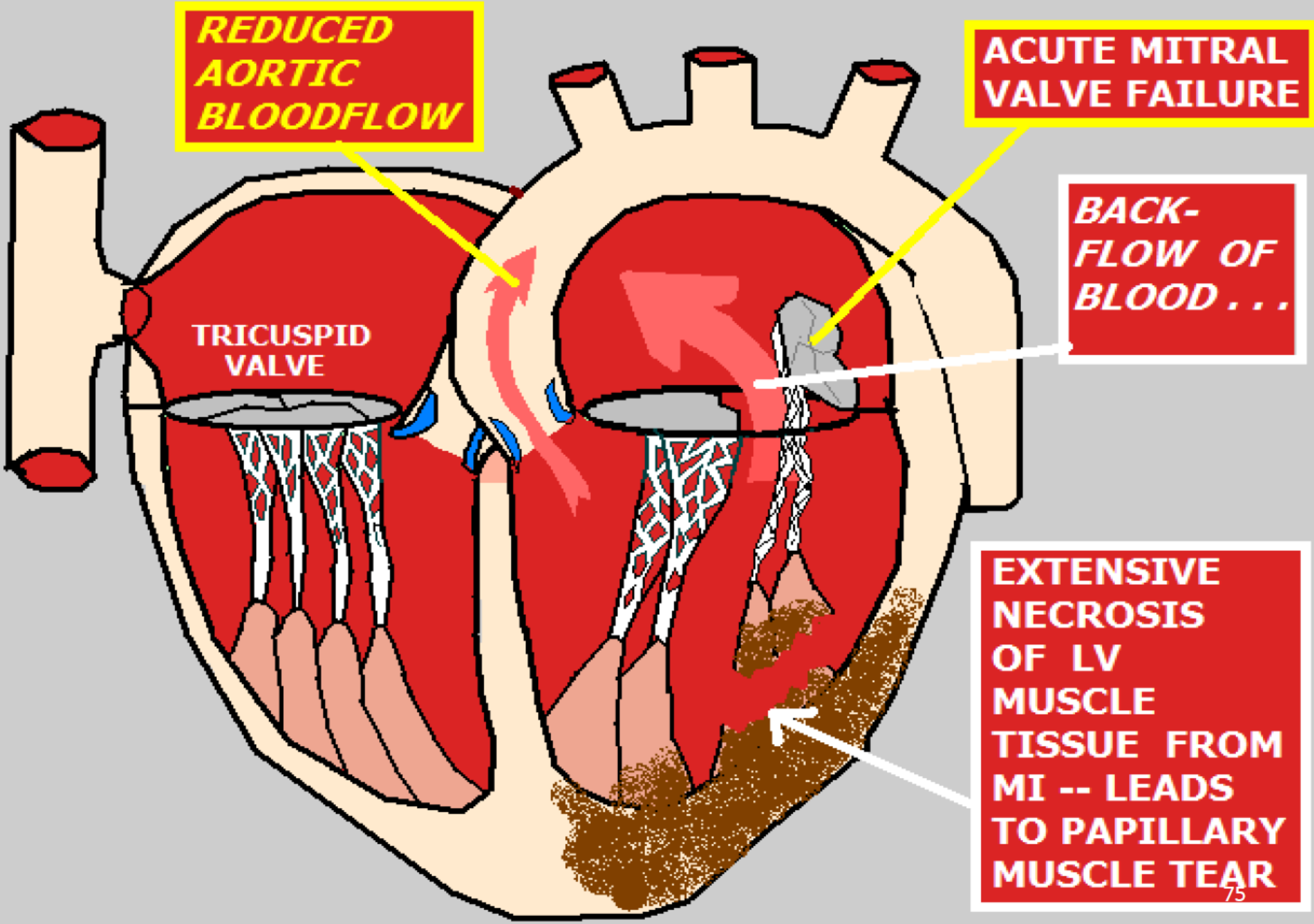
CHORDAE TENDINEAE

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.

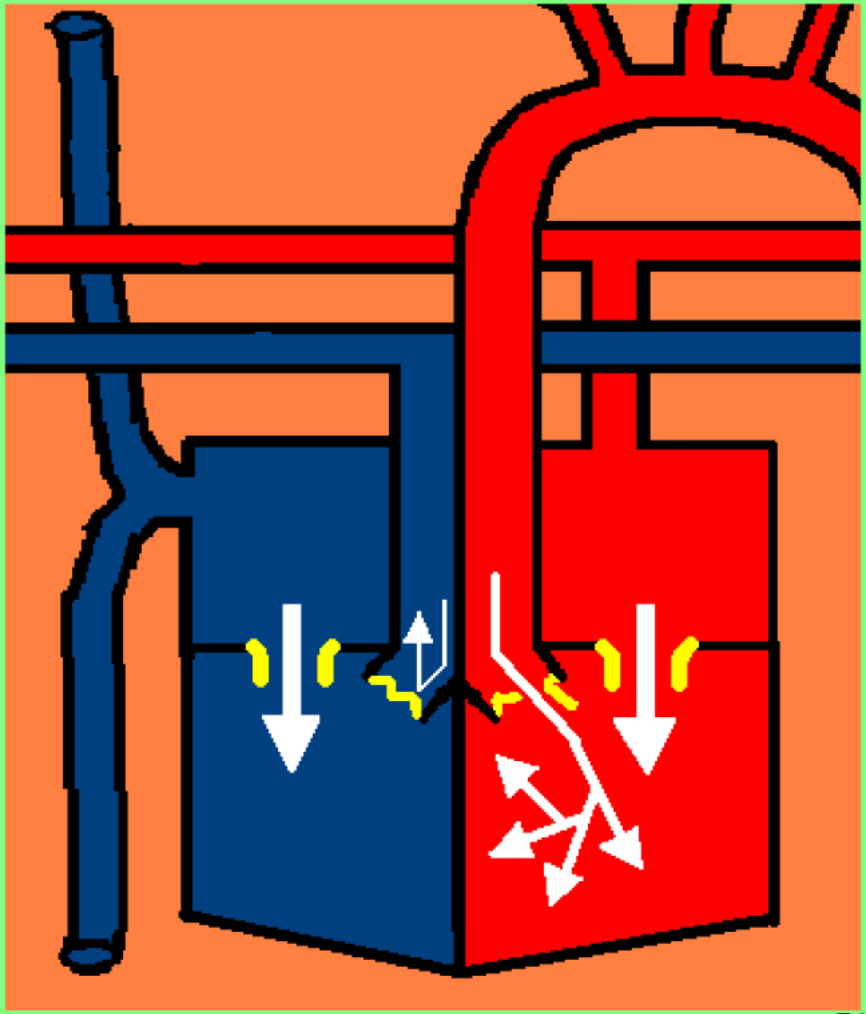
S-2 MURMUR SOUNDS LIKE:

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

CAUSE OF DIASTOLIC (S_2) MURMUR

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

- **CAUSES REGURGITATION**



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

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

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

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:

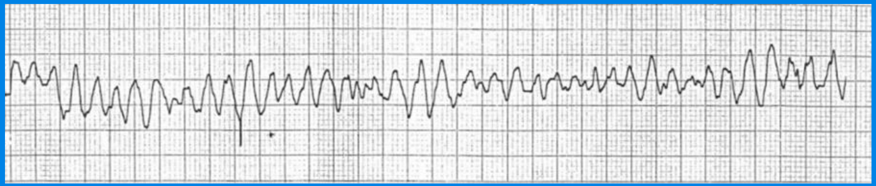
- HEMODYNAMIC STATUS
  - ABCs
  - Shock
- SYMPTOMS
  - Chest Pain / Pressure
  - Other ACS Symptoms
- ECG
  - 12 Lead
  - Single Lead “rhythm strip”

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

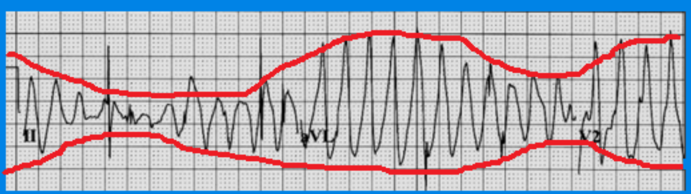
Defib 120-200 or HIGHER
- Epinephrine 1mg IV

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

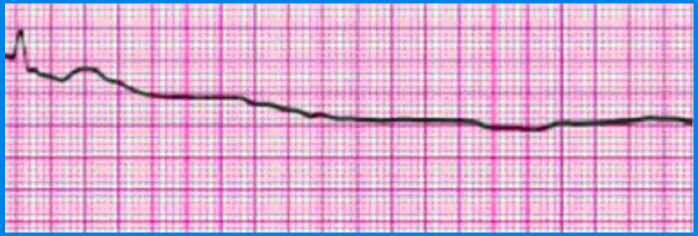
Defib 120-200 or HIGHER
- ~~Amiodarone 300mg~~ – OR –
  Lidocaine 1.0-1.5 mg/kg

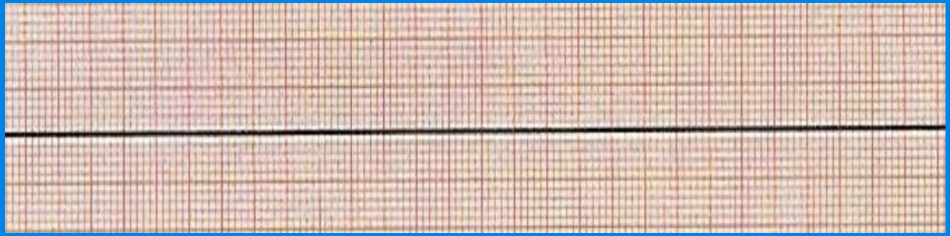
Defib 120-200 or HIGHER
- Epinephrine 1mg IV

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

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

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

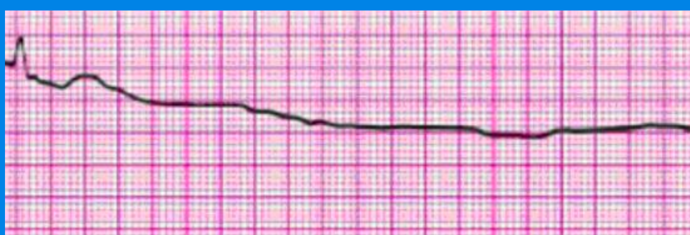




- Continue CPR
- IV/IO Access
- Advanced Airway
- Epinephrine 1mg IV
- Rule out reversible causes
- CONTINUE as per ACLS....

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

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



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

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

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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☞ ***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:

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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

<u>KEY WORDS:</u>

"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:

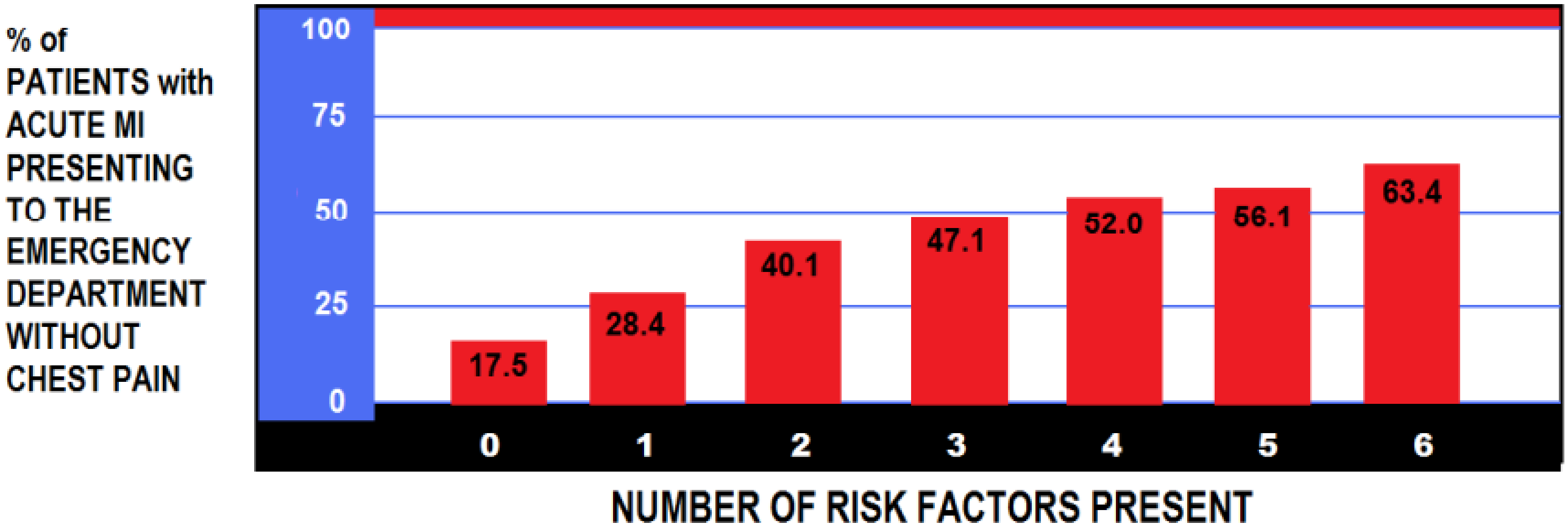
Stroke (previous history of)
Heart failure (previous history of)
Race (non-white)
Elderly (age 75+)
Women
Diabetes mellitus

* 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)	**F**atigue
Indigestion	**A**bdominal pain
Nausea	**C**old sweats
Dizziness	**E**levated heart rate
Syncope	**D**sypnea

Effect of Having Multiple Risk Factors for AMI Without Chest Pain



RISK FACTORS INCLUDE: Stroke (previous), Heart failure (previous), Race (non-white), Elderly (age 75+), Women, Diabtetes

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”

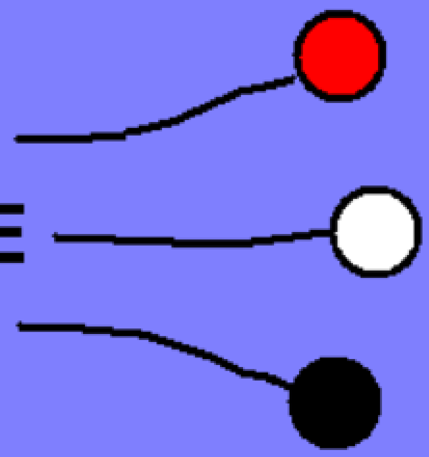
- The 12 Lead ECG has been ordered
- Meanwhile we’ll hook the patient to the ECG monitor . . . . .

THE EKG MACHINE

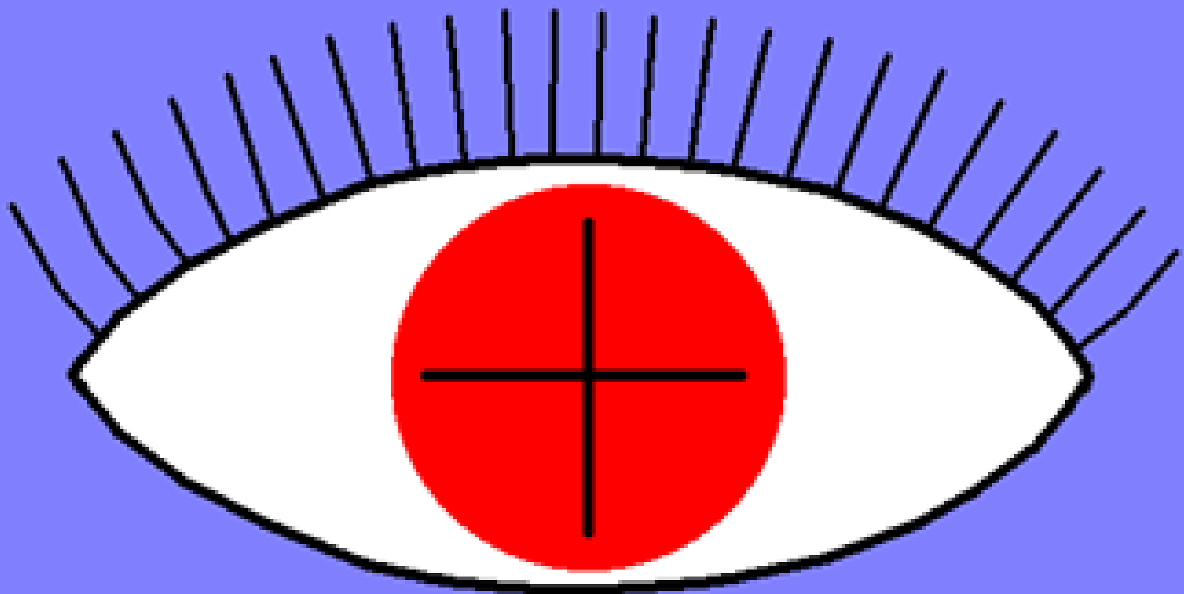
STANDARD 12 LEADS - USES 10 WIRES
(6 CHEST and 4 LIMB)

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

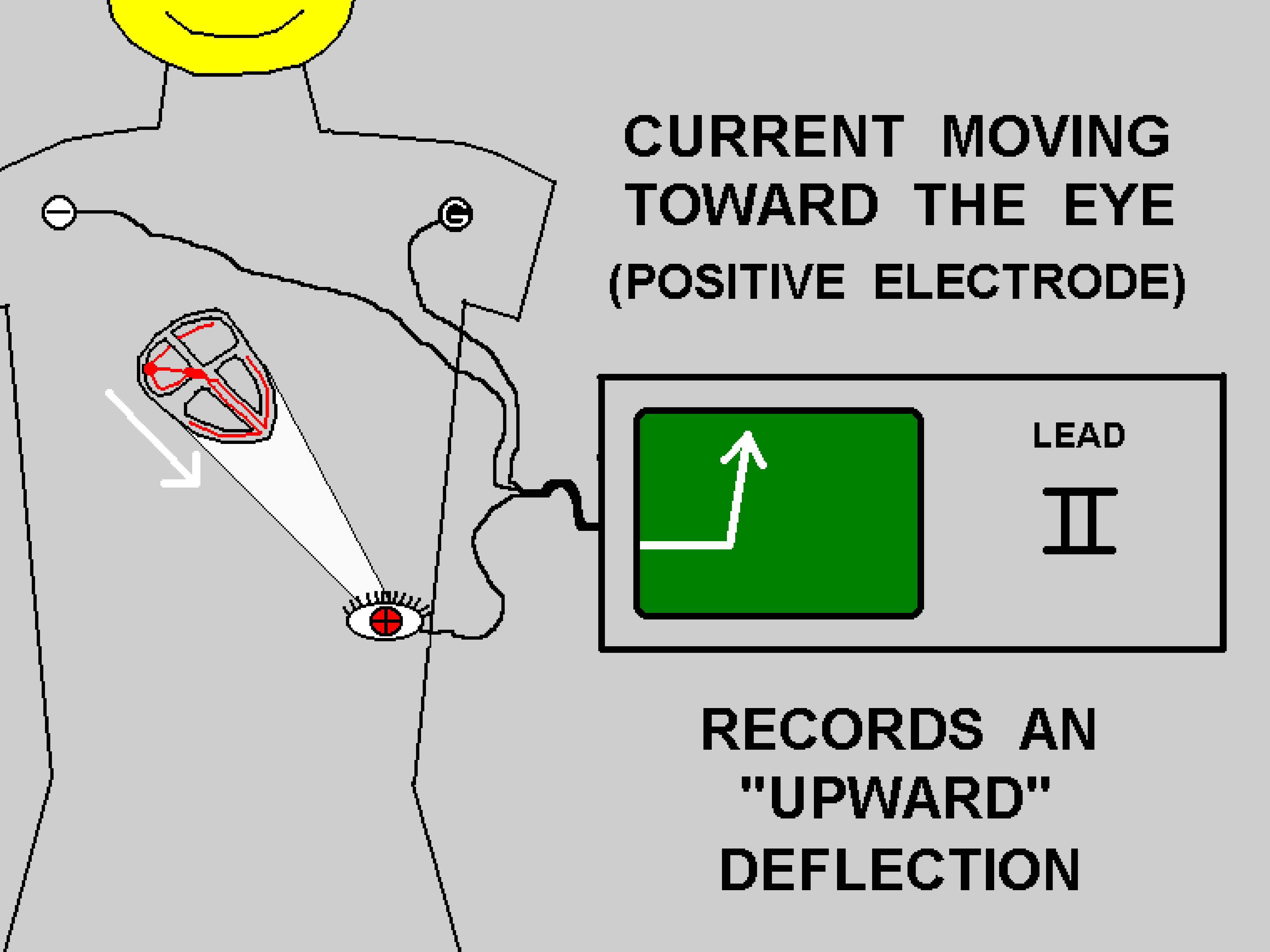
1 POSITIVE ELECTRODE
1 NEGATIVE ELECTRODE
1 GROUND ELECTRODE

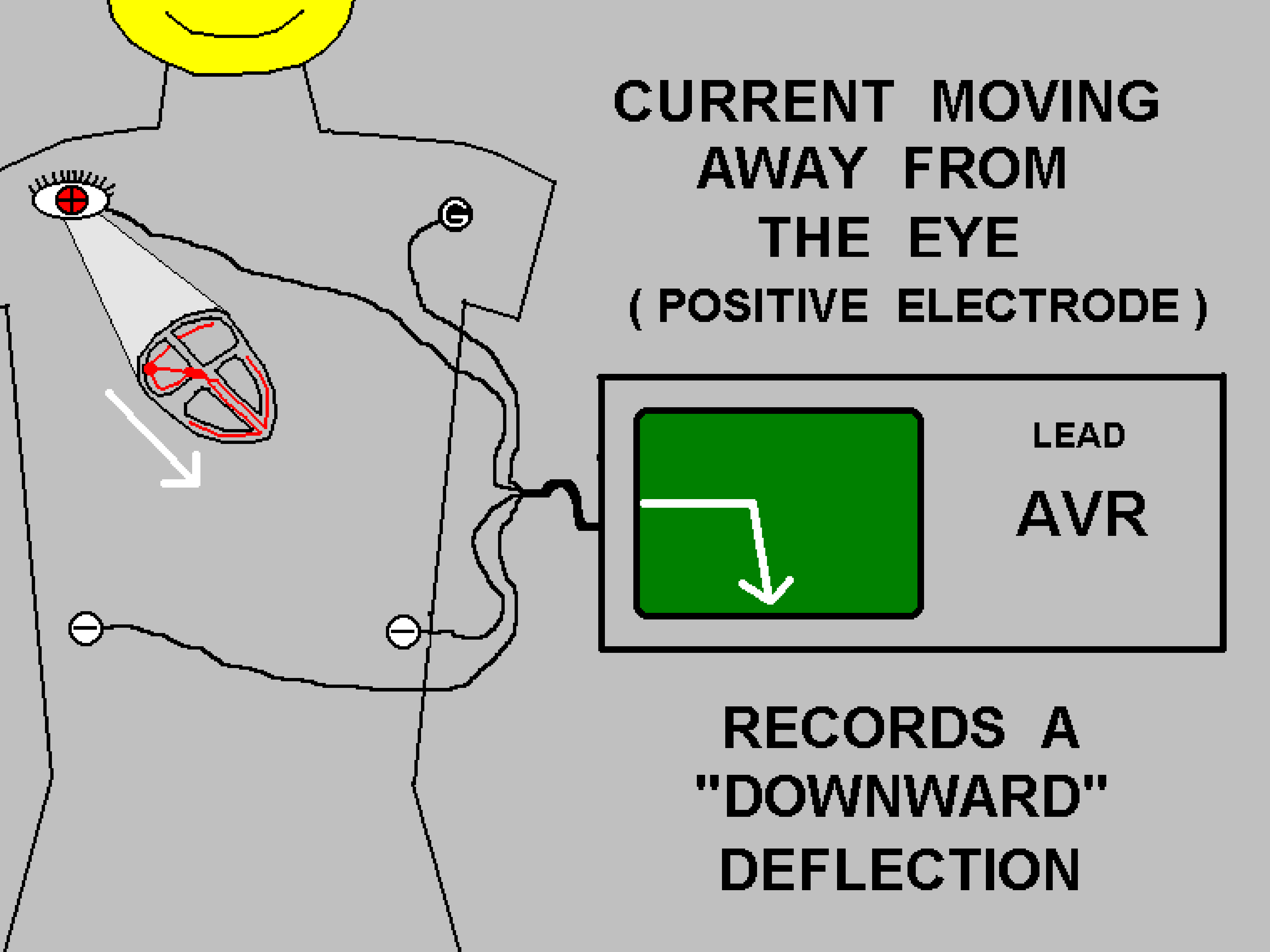


THE POSITIVE ELECTRODE

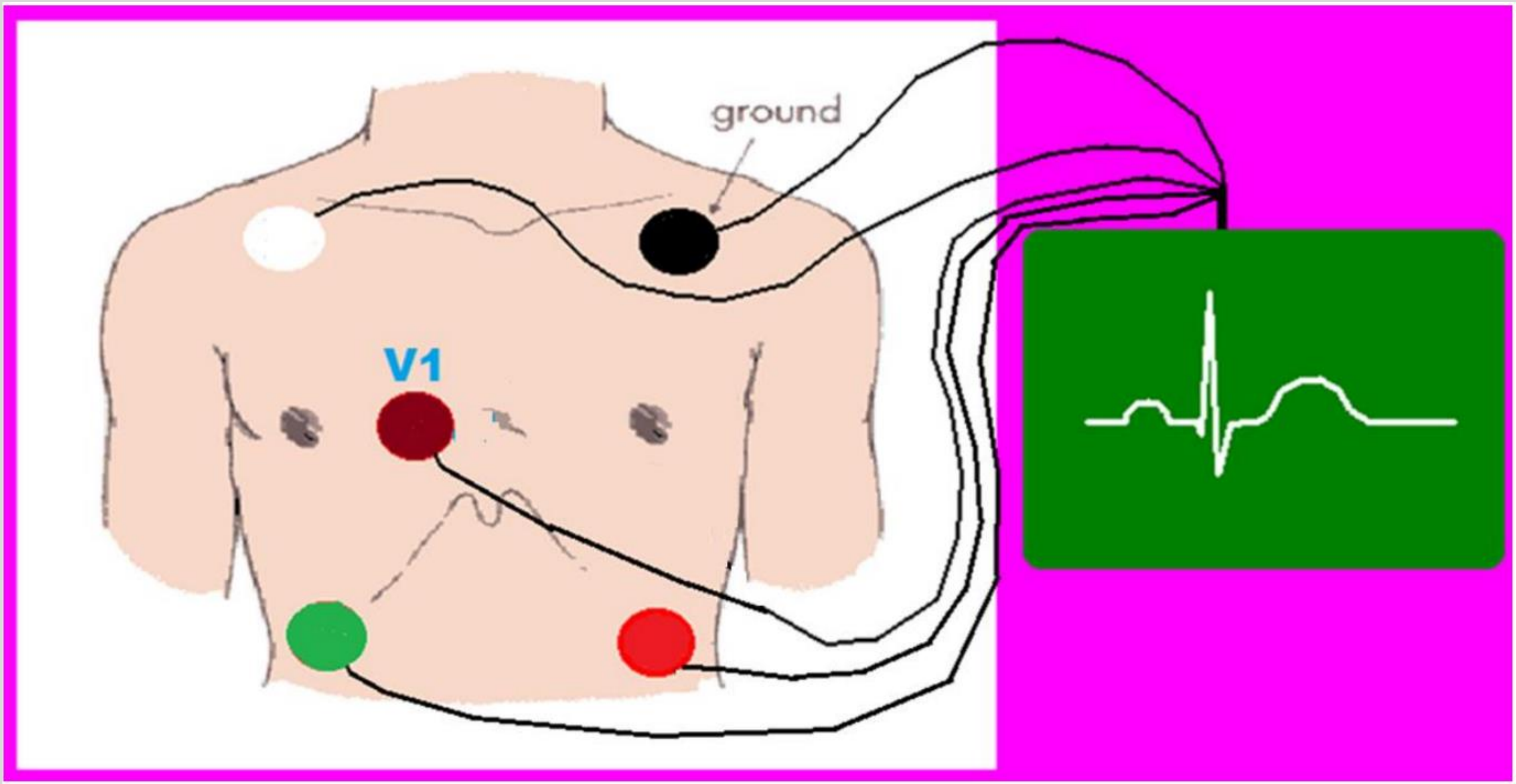


IS THE "EYE" . . .

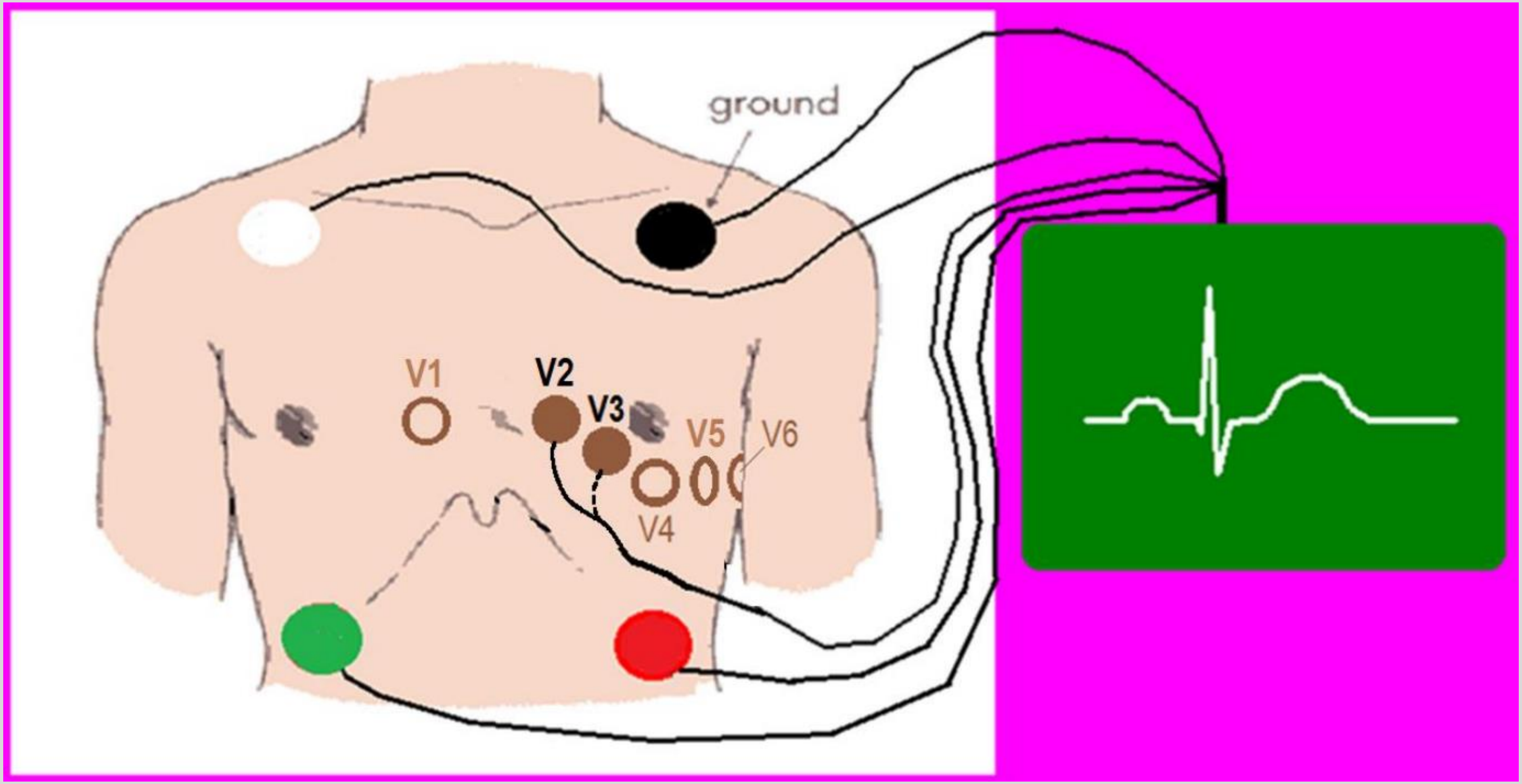

CURRENT MOVING
TOWARD THE EYE
(POSITIVE ELECTRODE)
LEAD
II
RECORDS AN
"UPWARD"
DEFLECTION


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

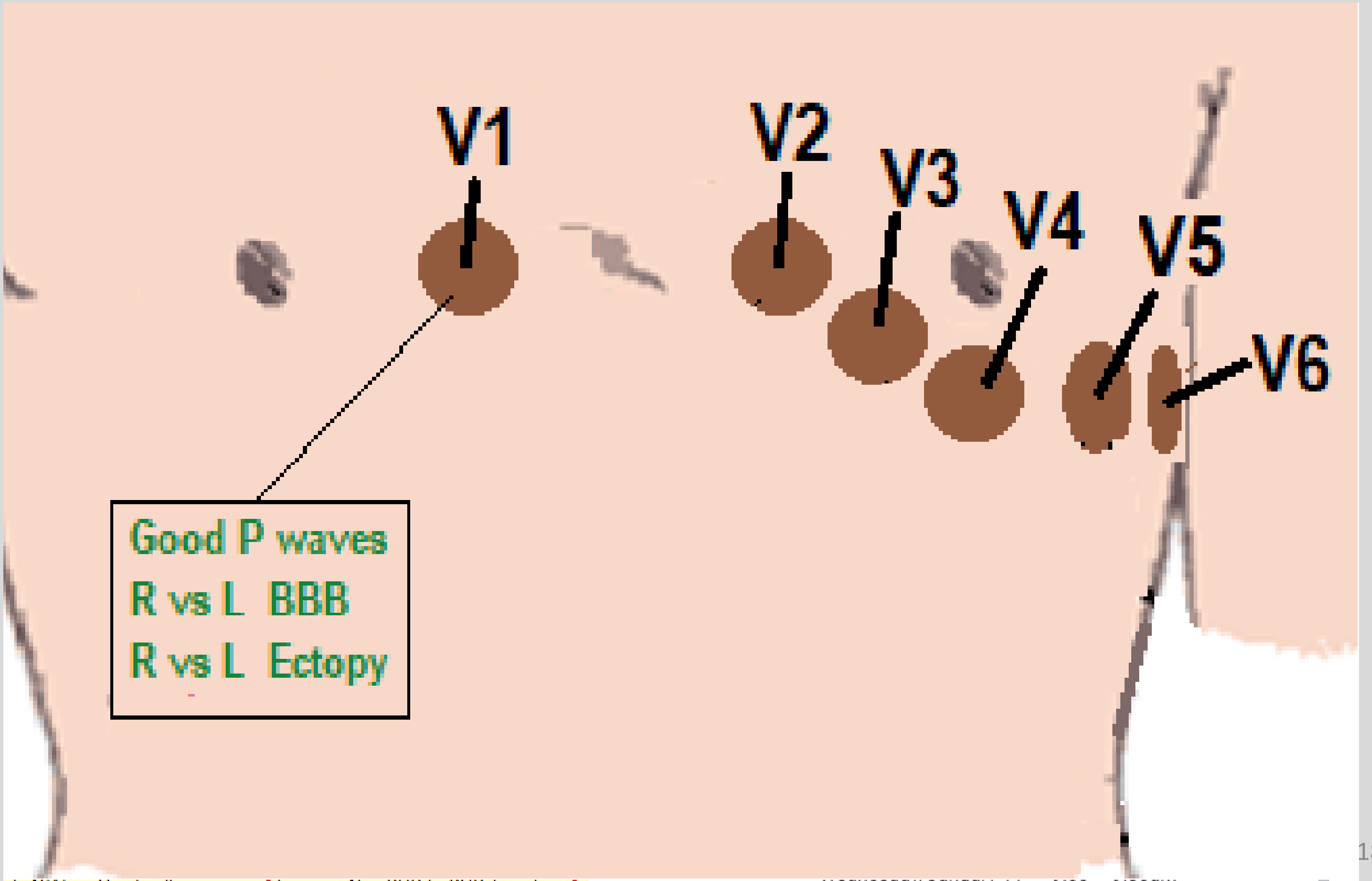
Traditional
ECG Monitoring Lead Placement:



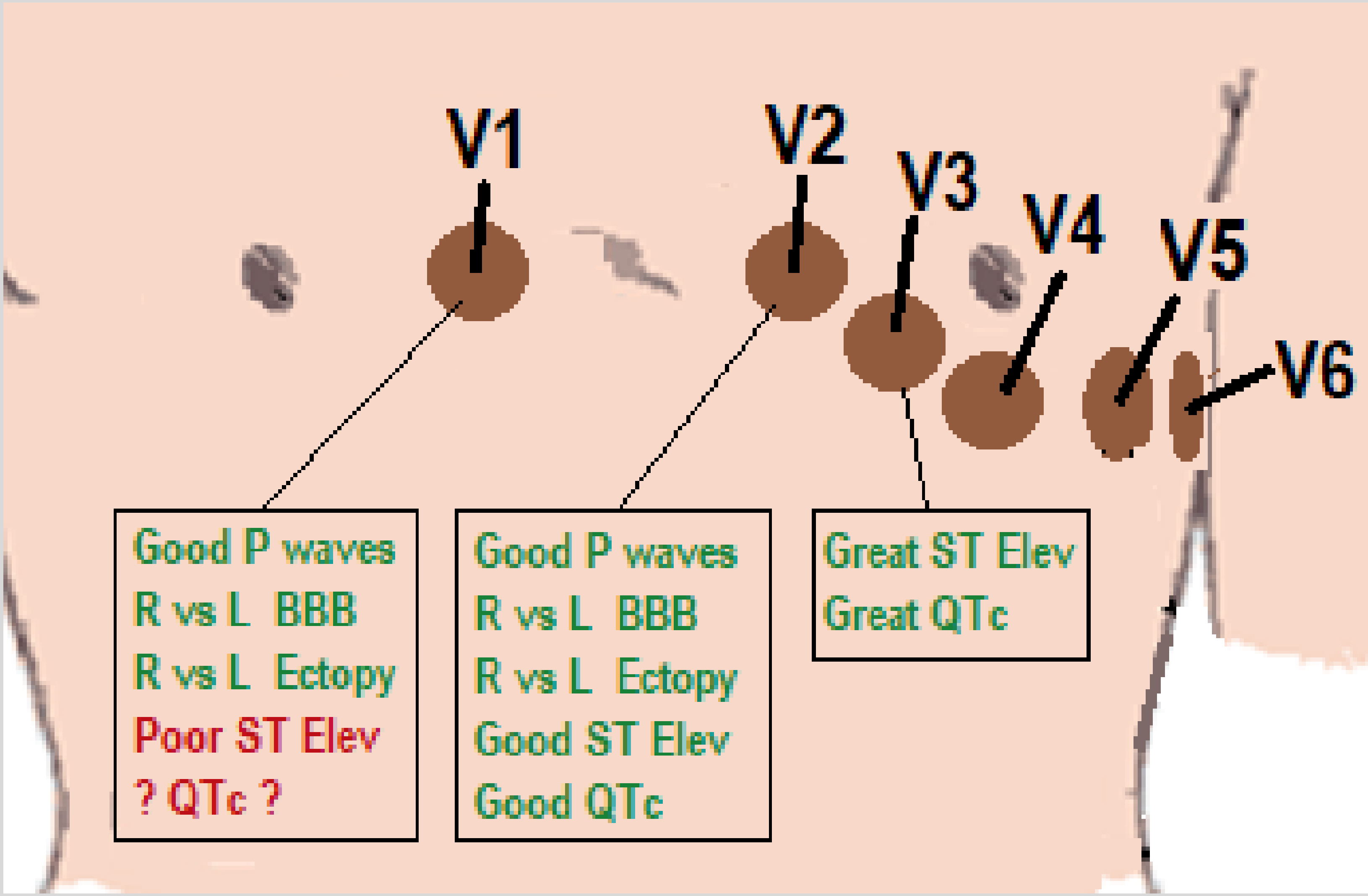
New (2014) Guideline Suggested ECG Monitoring Lead Placement



Traditional Continuous ECG Monitoring Lead: V1



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

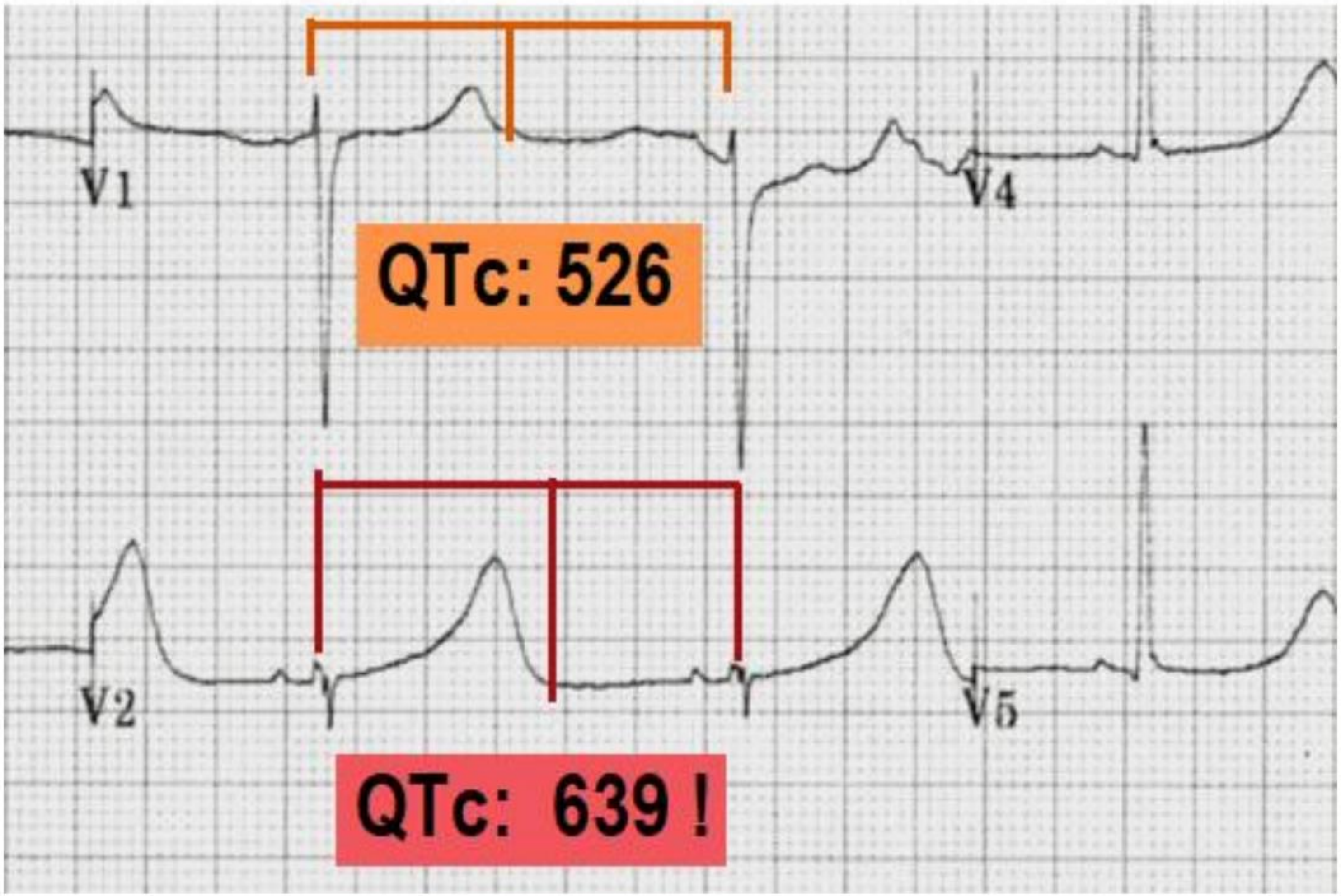


Why not V1 ?

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:

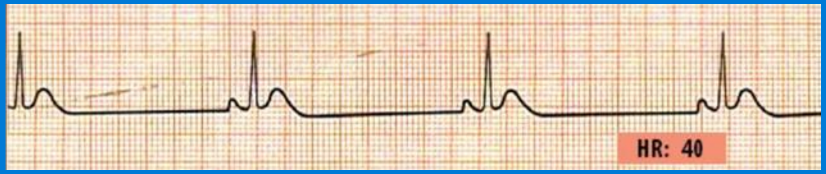
- LESS THAN 50
- GREATER THAN 150

CARDIOGENIC SHOCK

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

☞ ***SPEED UP THE HEART RATE***

(follow ACLS and Protocols)

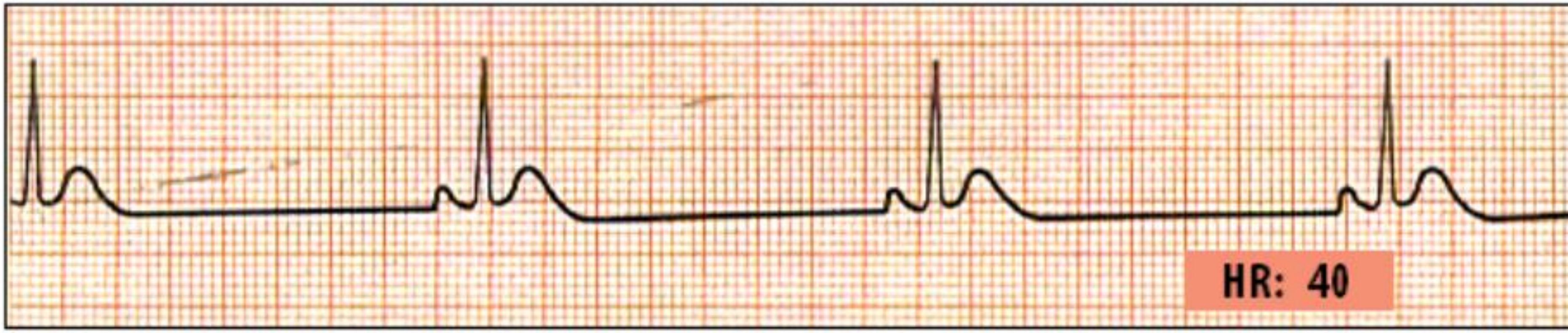


Usual treatment:
- Atropine
- Pacemaker

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.

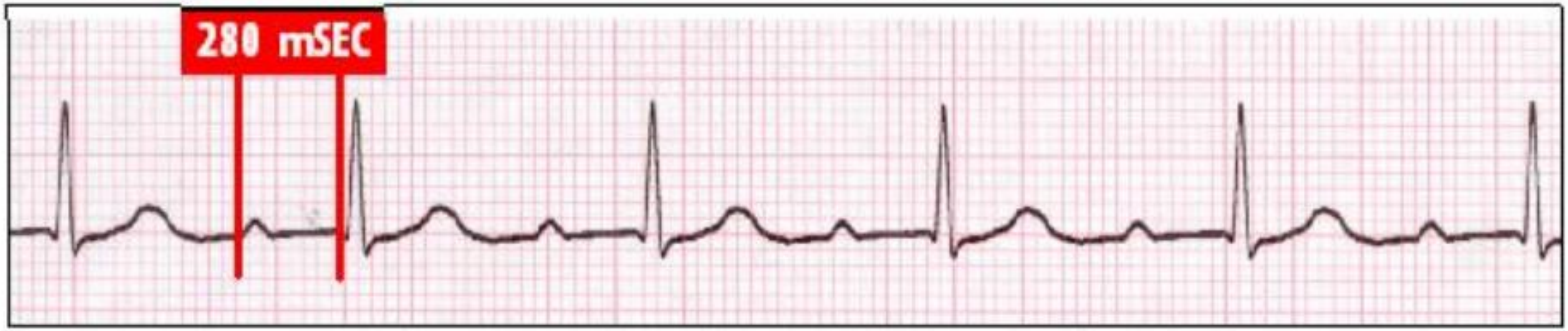
THIS RHYTHM IS: SINUS BRADYCARDIA



WE MUST CONSIDER UNDERLYING CAUSES: AND TREAT THEM:

INCREASED VAGAL TONE →	ATROPINE
BLOCKED SA NODAL ARTERY (INFERIOR WALL MI) →	CARDIAC CATH - PTCA / STENT THROMBOLYTICS
ELECTROLYTE IMBAL. (K+) →	CORRECT ELECTROLYTES
HYPOTHERMIA →	WARM PATIENT
ORGANOPHOSPHATE POISONING →	ATROPINE
ATHLETIC METABOLISM (excellent health!) →	COMPLIMENT PATIENT!

THIS RHYTHM IS: FIRST DEGREE HEART BLOCK



MAIN IDENTIFICATION CHARACTERISTIC(S): P - R INTERVAL TOO LONG - (GREATER THAN 200 mSEC.)

RATE ---------------- NORMAL
RHYTHM ------------ REGULAR
P-R INTERVAL ----- > 200 mSEC.
P: QRS RATIO ------ 1 : 1
QRS INTERVAL ---- NORMAL

P : QRS RATIO

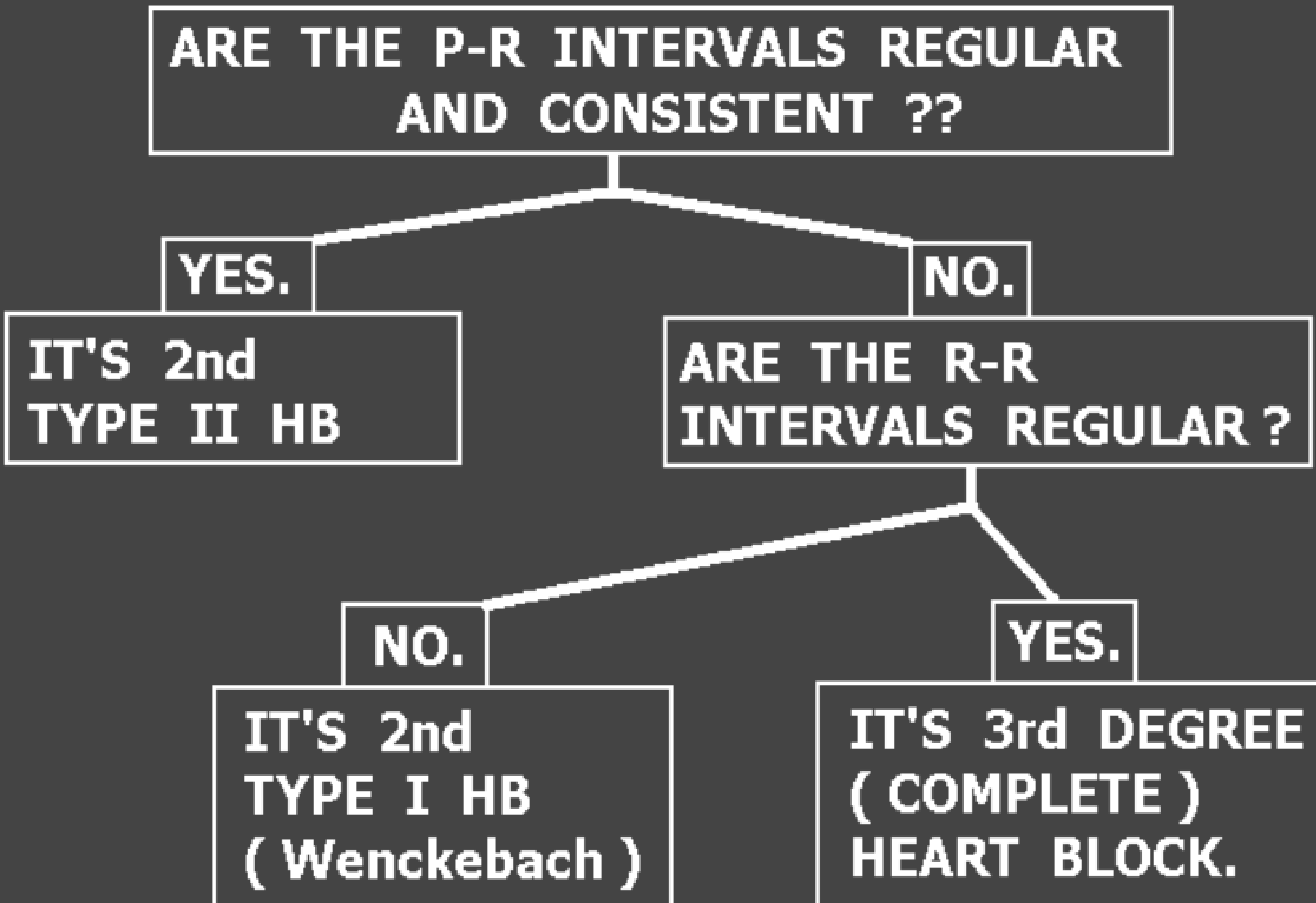
IF GREATER THAN 1 : 1

THINK:

- 2° HEART BLOCK
  (TYPE 1 or 2)
- 3° HEART BLOCK

DIAGNOSING 2nd and 3rd DEGREE HEART BLOCK

MORE P-WAVES THAN QRS COMPLEXES PRESENT.



LET'S TEST THE PROCEDURE . . .

1

2

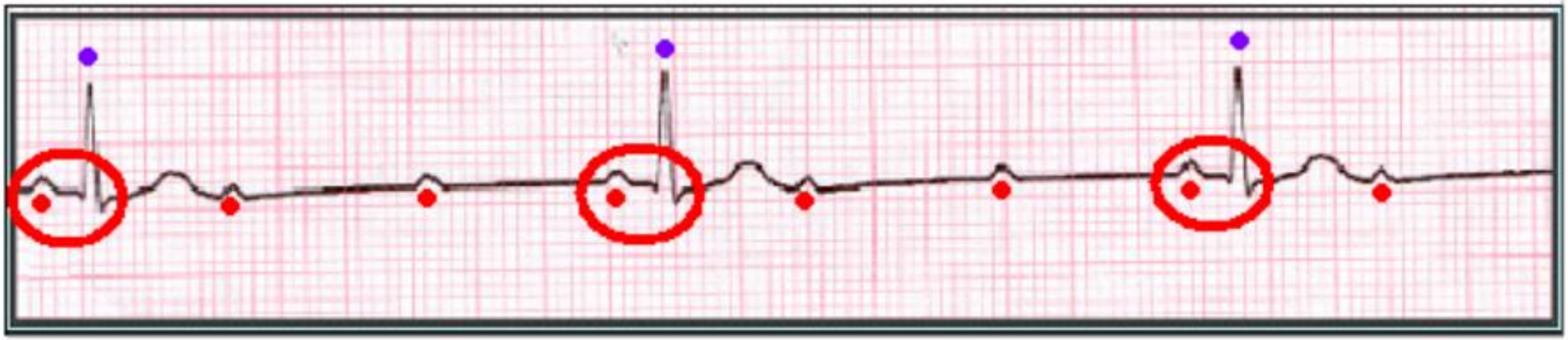
3

THIS RHYTHM IS: 2nd ^{o} TYPE I HB (Wenckebach)

MAIN IDENTIFICATION CHARACTERISTIC(S): P-R INTERVAL GETS PROGRESSIVELY LONGER UNTIL IT DROPS A QRS -- THEN CYCLE REPEATS

RATE ---------------- NORMAL or BRADYCARDIC
RHYTHM ------------- REGULARLY IRREGULAR
P-R INTERVAL ----- VARIES (regularly irregular)
P:QRS RATIO ------ VAIRES (usually 1:1 and 2:1)
QRS INTERVAL ---- NORMAL

THIS RHYTHM IS: 2nd ° TYPE II HEART BLOCK



MAIN IDENTIFICATION CHARACTERISTIC(S): MORE THAN ONE P WAVE FOR EACH QRS -- BUT EVERY QRS HAS A NORMAL, CONSISTENT P-R INTERVAL

RATE ------------------ USUALLY BRADYCARDIC
RHYTHM ------------- USUALLY REGULAR (can be irregular)
P-R INTERVAL ------ NORMAL and CONSISTENT
P:QRS RATIO ------- ≥ 2:1
QRS INTERVAL ----- NORMAL

THIS RHYTHM IS: 3rd^{o} HB $\bar{c}$ JUNCTIONAL ESCAPE

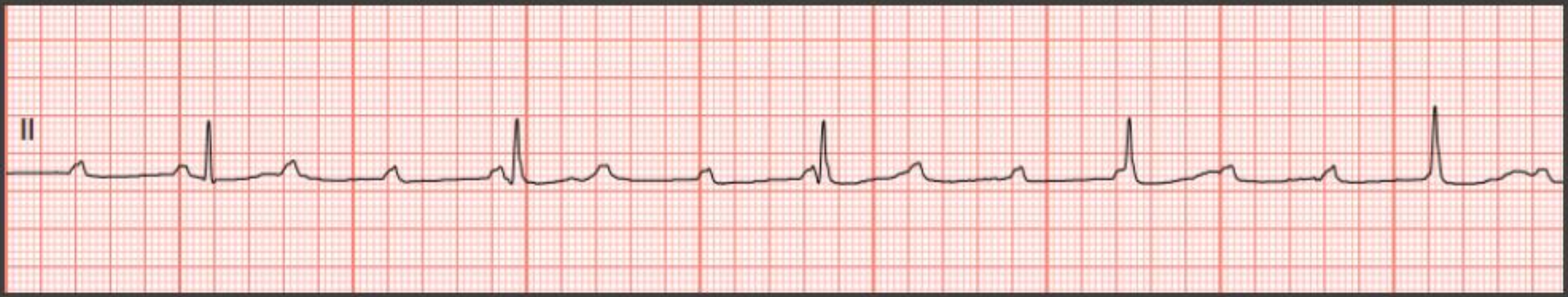
MAIN IDENTIFICATION CHARACTERISTIC(S): P - R INTERVAL INCOSISTENT, P - P INTERVALS REGULAR, R - R INTERVALS REGULAR -- NO RELATIONSHIP BETWEEN P WAVES AND QRS COMPLEXES.

RATE ---------------- USUALLY BRADYCARDIC (40 - 60 JUNCTIONAL RATE)
RHYTHM ----------- REGULAR
P-R INTERVAL ----- INCONSISTENT (irregularly irregular)
P: QRS RATIO ------ VARIES - USUALLY > 2 : 1
QRS INTERVAL ----- NORMAL (< 120 ms) UNLESS PT HAS BUNDLE BRANCH BLOCK

THIS RHYTHM IS: 3rd^{o} HB $\bar{c}$ IDIOVENTRICULAR ESCAPE

MAIN IDENTIFICATION CHARACTERISTIC(S): P - R INTERVALS INCONSISTENT P - P INTERVALS REGULAR, R - R INTERVALS REGULAR. NO RELATIONSHIP BETWEEN P WAVES AND QRS COMPLEXES. QRS COMPLEXES are WIDER THAN 120ms, AND OF SLOW VENTRICULAR RATE (usually < 40)

RATE ------------------ USUALLY BRADYCARDIC (< 40 VENTRICULAR RATE)
RHYTHM ------------- REGULAR
P-R INTERVAL ----- INCONSISTENT (irregularly irregular)
P: QRS RATIO ------ VARIES - USUALLY > 2 : 1
QRS INTERVAL ---- WIDER THAN 120 ms


II

???

THIS RHYTHM IS: JUNCTIONAL RHYTHM

MAIN IDENTIFICATION CHARACTERISTIC(S): P WAVES ABSENT, or LOCATED JUST AFTER QRS (in S-Tseg) or JUST BEFORE QRS (short P-R). WHEN P wave seen, it is INVERTED (upside-down).

- HR USUALLY 40 - 60

RATE ---------------- 40 - 60
RHYTHM ------------ REGULAR
P-R INTERVAL ----- ABSENT or SHORT
P: QRS RATIO ------ 1 : 1
QRS INTERVAL ----- NORMAL

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

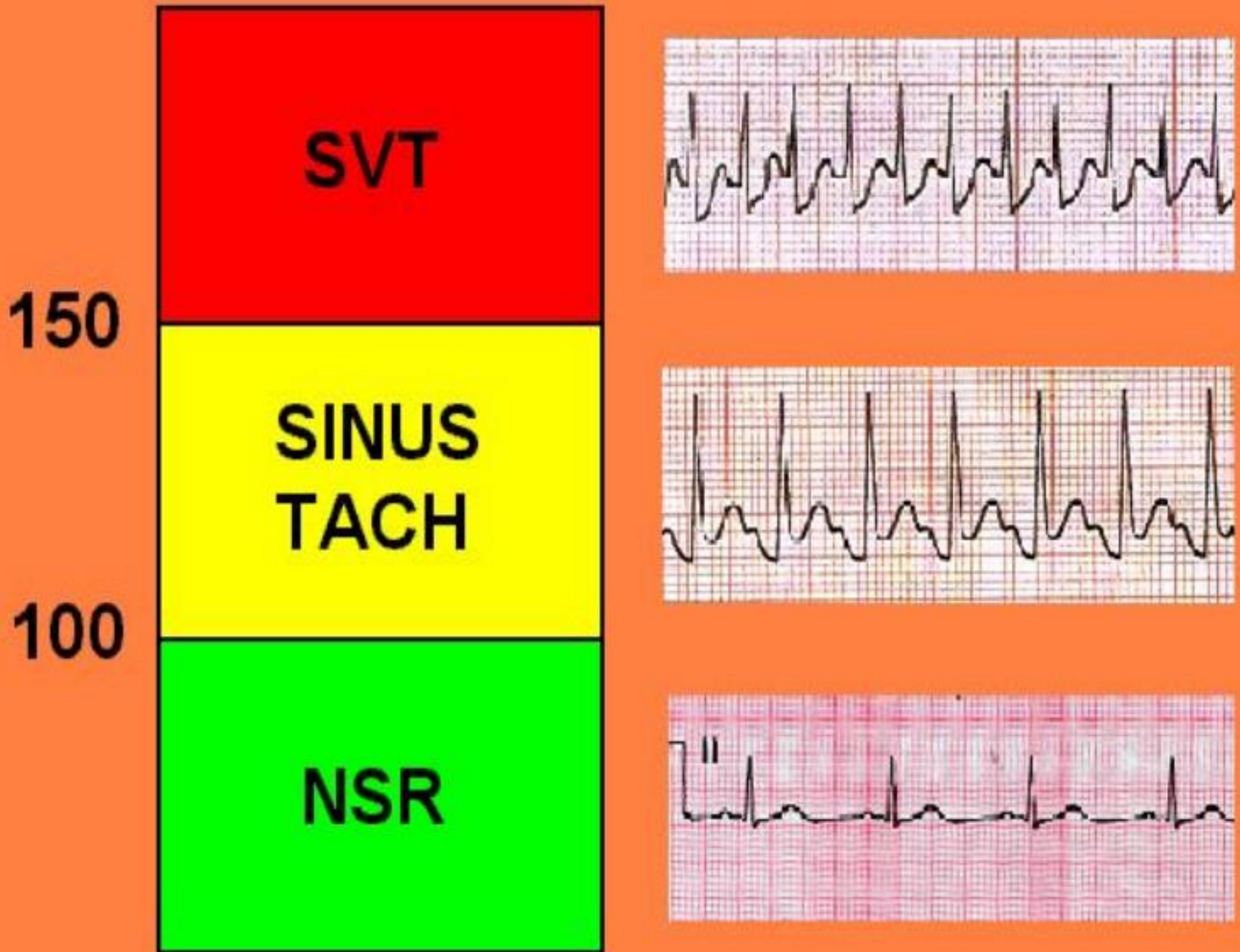
Tachycardias

- 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



THIS RHYTHM IS: SINUS TACHYCARDIA

MAIN IDENTIFICATION CHARACTERISTIC(S): SINUS RHYTHM, RATE HIGHER THAN 100. (ACLS guidelines: heart rate 100 - 150)

RATE ---------------- 100 - 150 (can be > 150)
RHYTHM ----------- REGULAR
P-R INTERVAL ----- NORMAL (120 - 200 ms)
P: QRS RATIO ------ 1 : 1
QRS INTERVAL ---- NORMAL (< 120 ms), (unless Bundle Branch Block present)

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."

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

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

GO TO
V - FIB
ALGORITHM !

PULSE - UNSTABLE

- IMMEDIATE DEFIBRILLATION

START IV
CONSIDER
SEDATION

PULSE - STABLE

- 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**

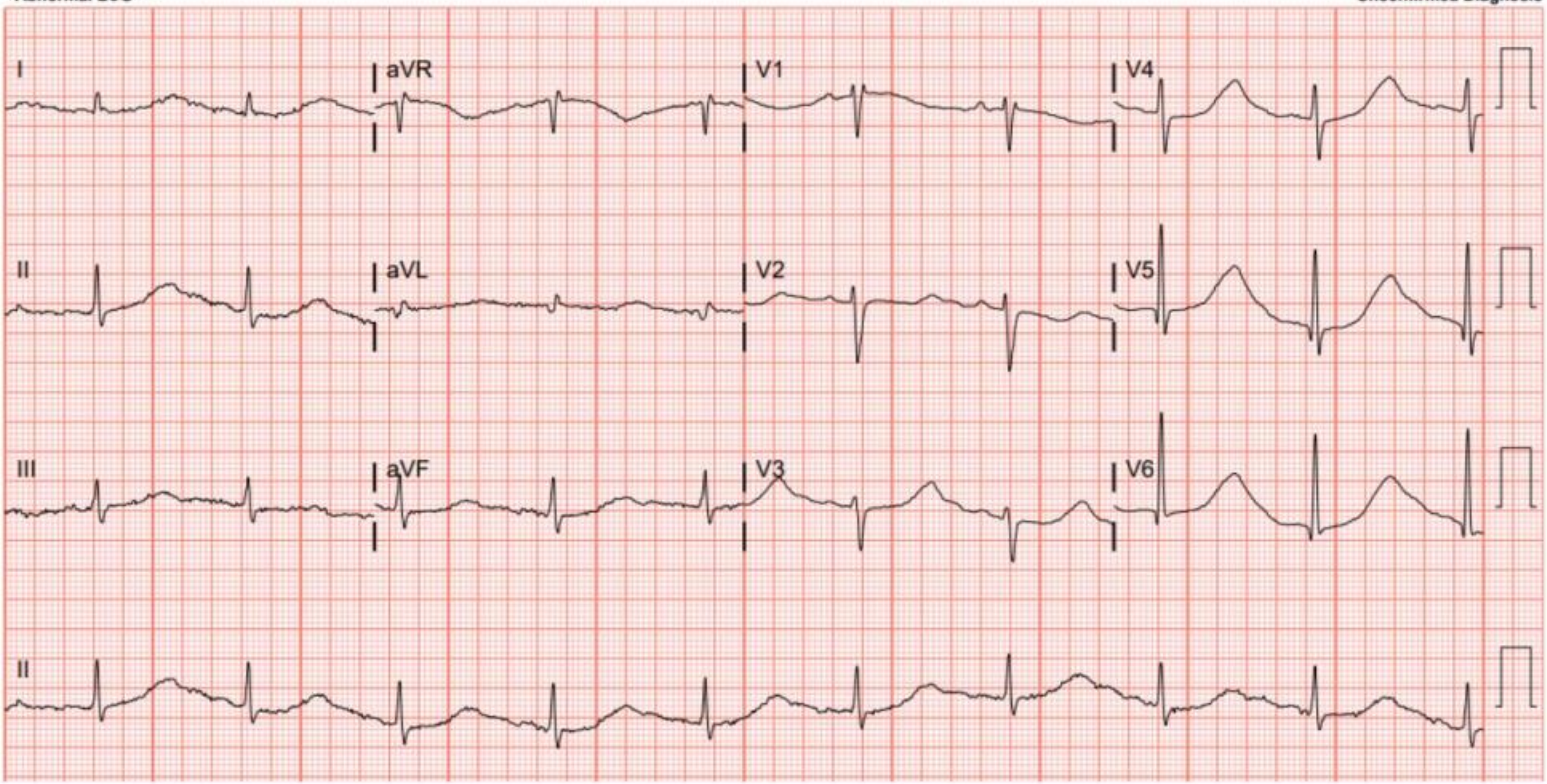
Rate	58	Sinus rhythm
PR	185	IVCD, consider atypical RBBB
QRSd	126	Baseline wander in lead(s) V2,V3,V4,V6
QT	668	COMPARED TO ECG 07/22/2020 16:56:59
QTc	657	SINUS RHYTHM NOW PRESENT
--Axis--		
P	107	
QRS	61	
T	45	

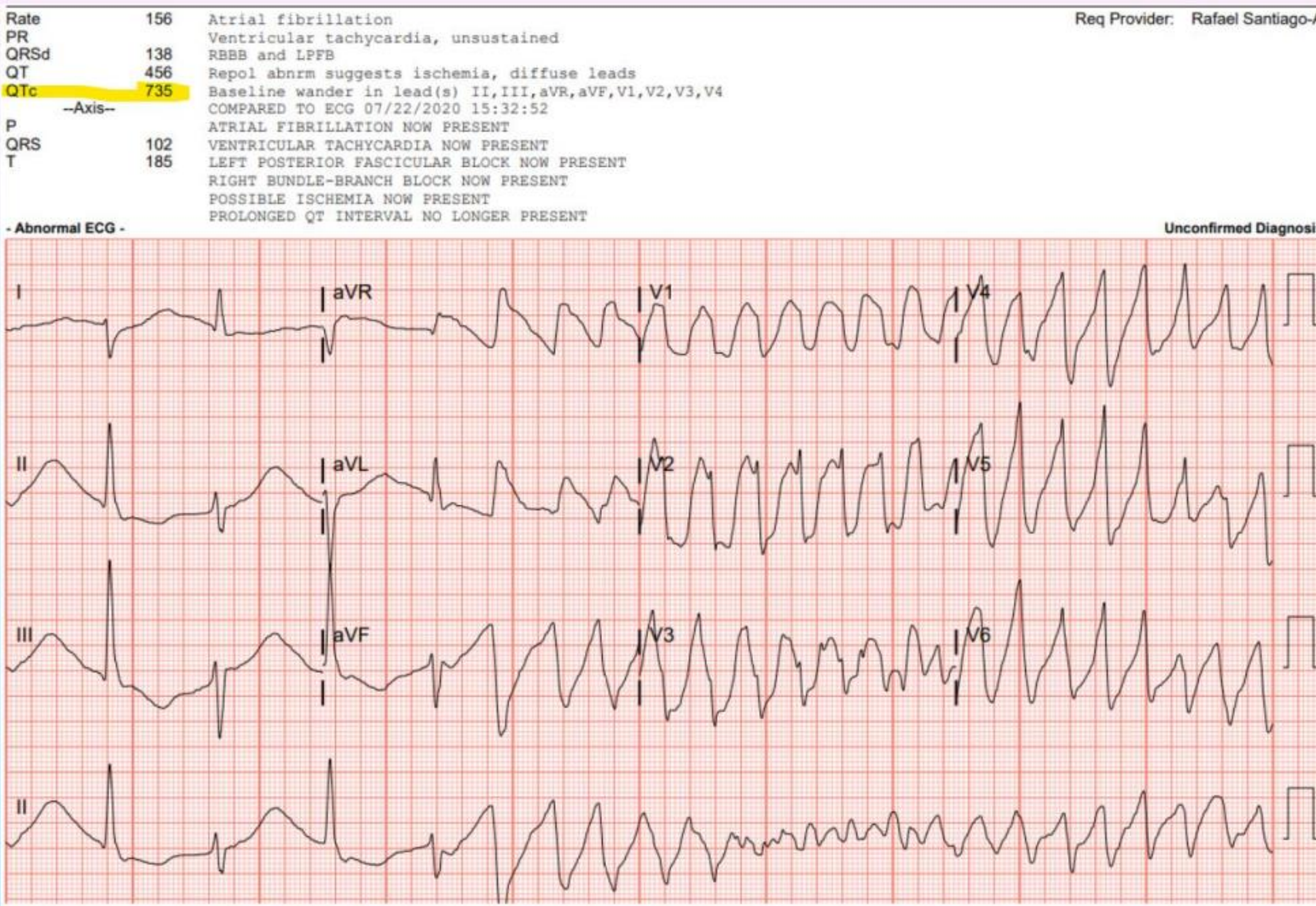
Req Provider: Rafael Santiago-Ap

QTc greater than 600 = "Torsades very possible….."

- Abnormal ECG -

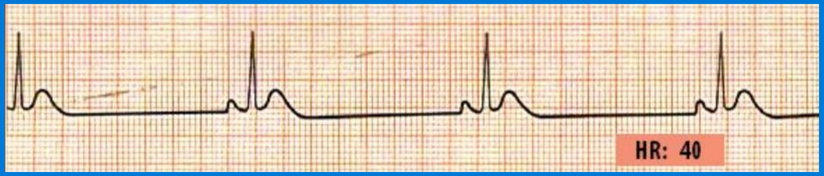
Unconfirmed Diagnosis




Rate 156
PR
QRSd 138
QT 456
QTc 735
--Axis--
P
QRS 102
T 185
Atrial fibrillation
Ventricular tachycardia, unsustained
RBBB and LPFB
Repol abnrm suggests ischemia, diffuse leads
Baseline wander in lead(s) II,III,aVR,aVF,V1,V2,V3,V4
COMPARED TO ECG 07/22/2020 15:32:52
ATRIAL FIBRILLATION NOW PRESENT
VENTRICULAR TACHYCARDIA NOW PRESENT
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
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

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

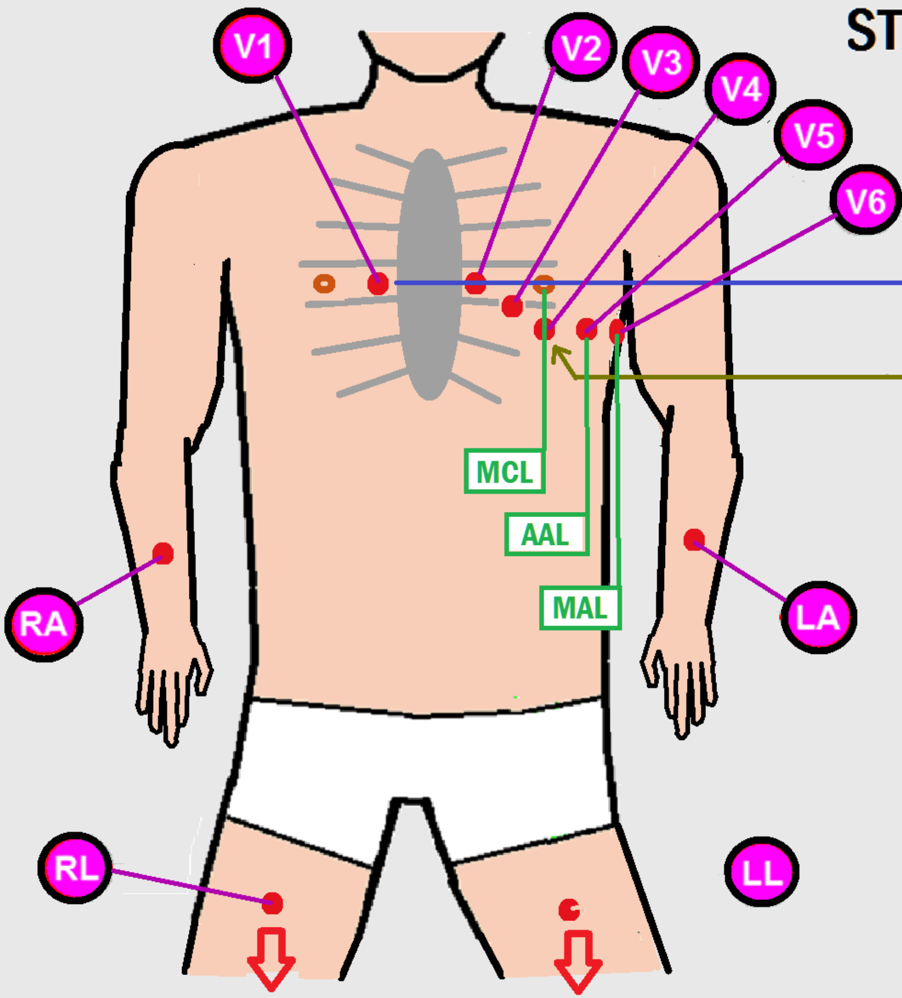
EMS 12 Lead ECG

In-Hospital 12 Lead ECG

10 wires . . .

- 4 limb leads
- 6 chest ("V") leads

Obtaining the 12 Lead ECG



STANDARD LEAD PLACEMENT ---
12 LEAD ECG

4 th INTERCOSTAL SPACE

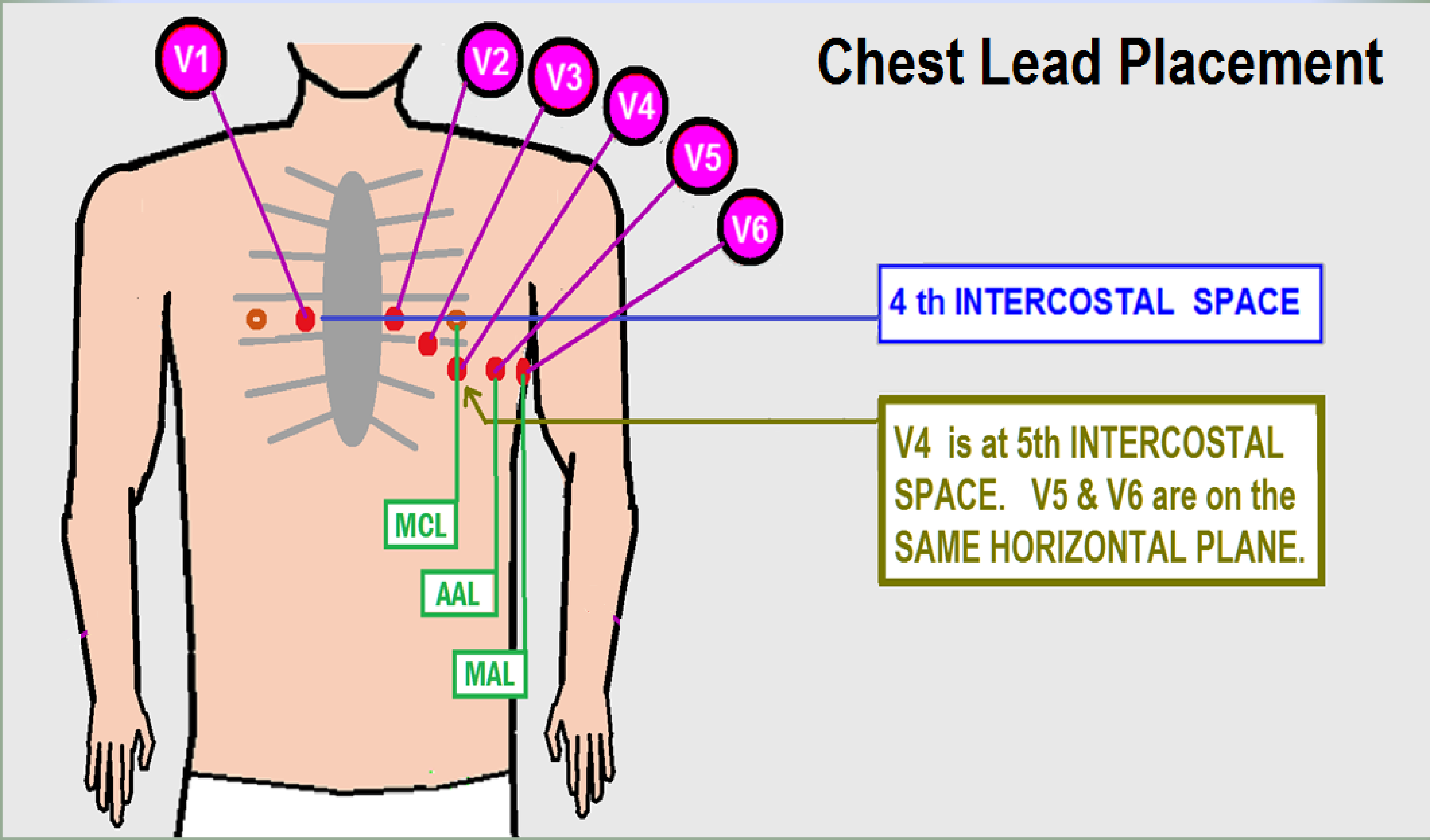
V4 is at 5th INTERCOSTAL SPACE. V5 & V6 are on the SAME HORIZONTAL PLANE.

- PATIENT SHOULD LAY AS FLAT AS POSSILVE
- LIMB LEADS SHOULD BE PLACED AS DISTALLY AS POSSIBLE

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

[redacted] TECH SD

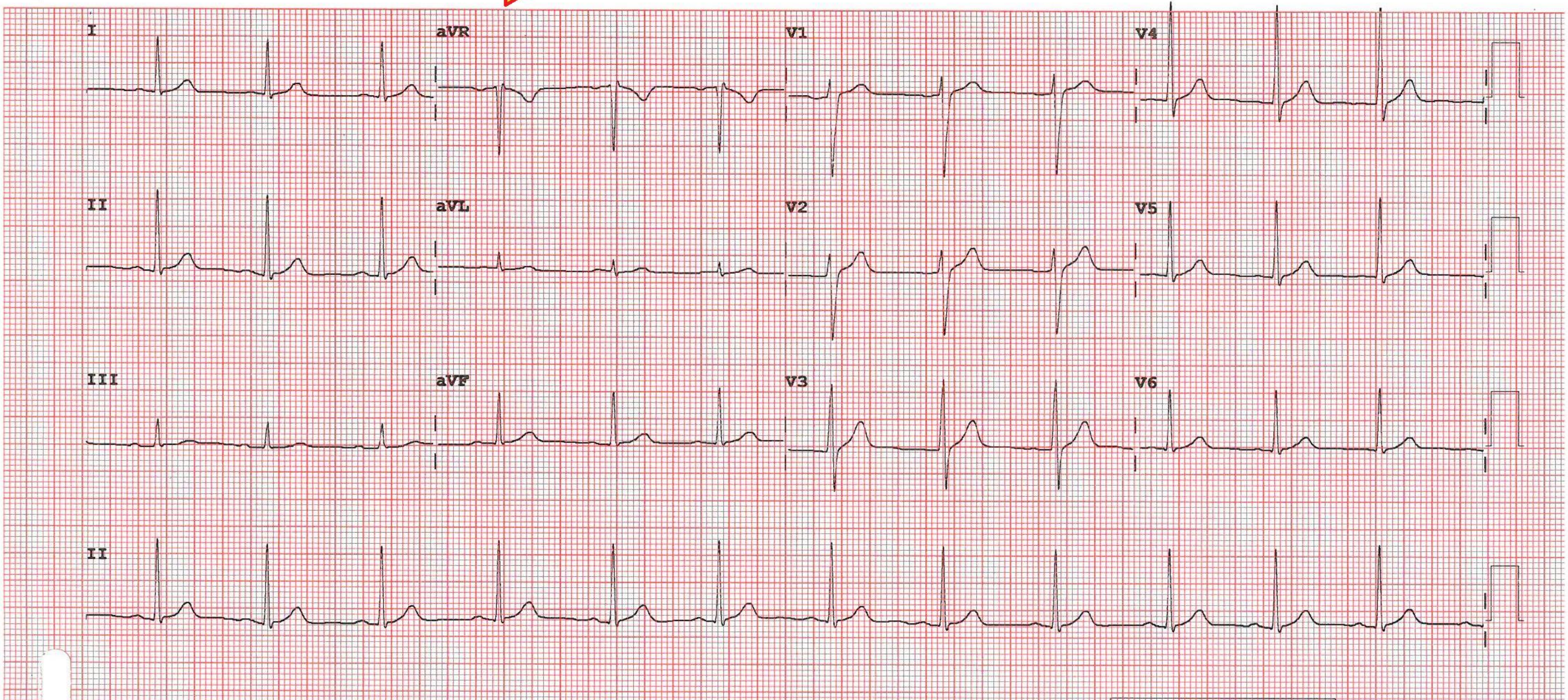
[redacted] [redacted]

--AXIS--
P 50
QRS 51
T 44

12 Lead; Standard Placement

- NORMAL ECG -

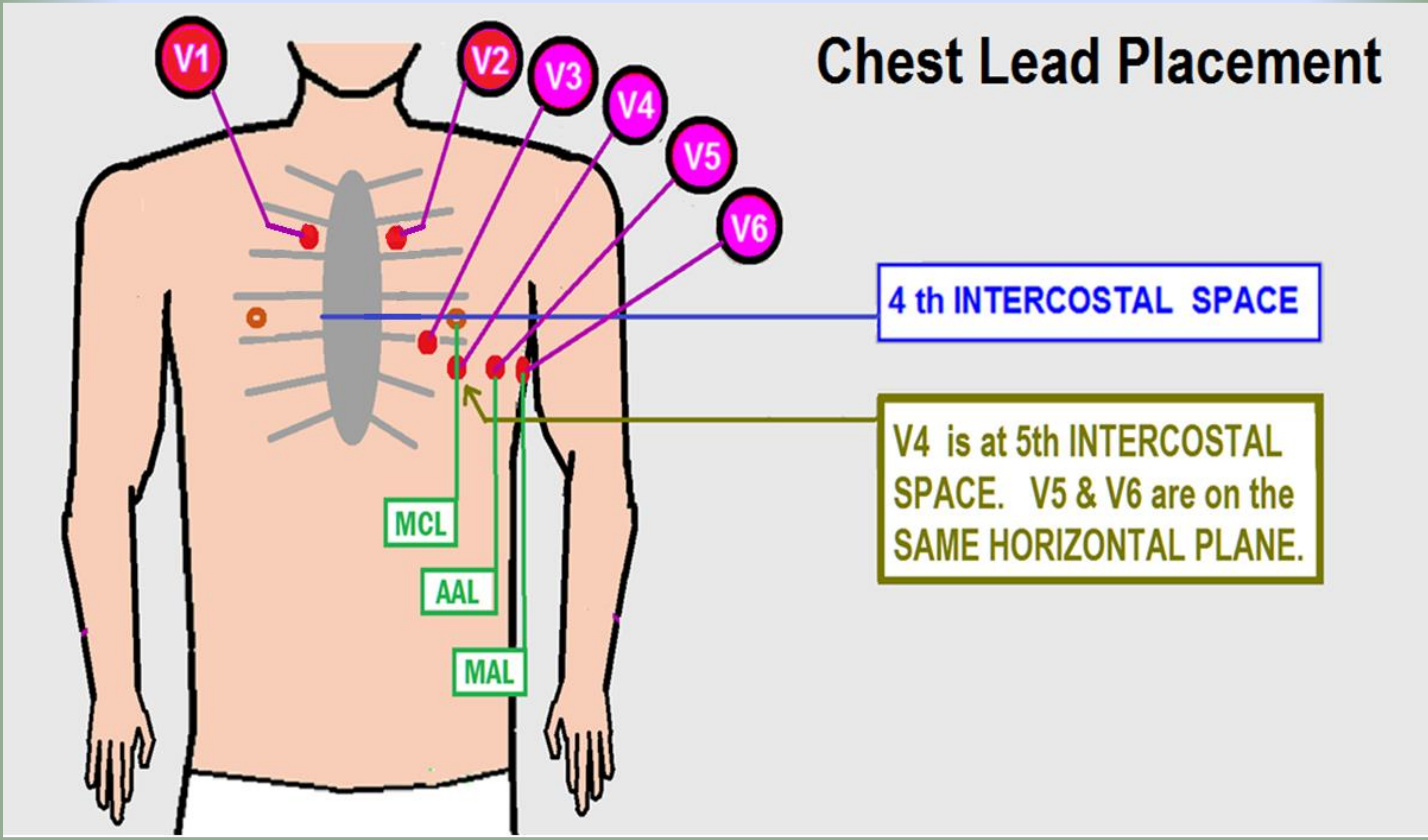
Unconfirmed Diagnosi



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

TEC

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--

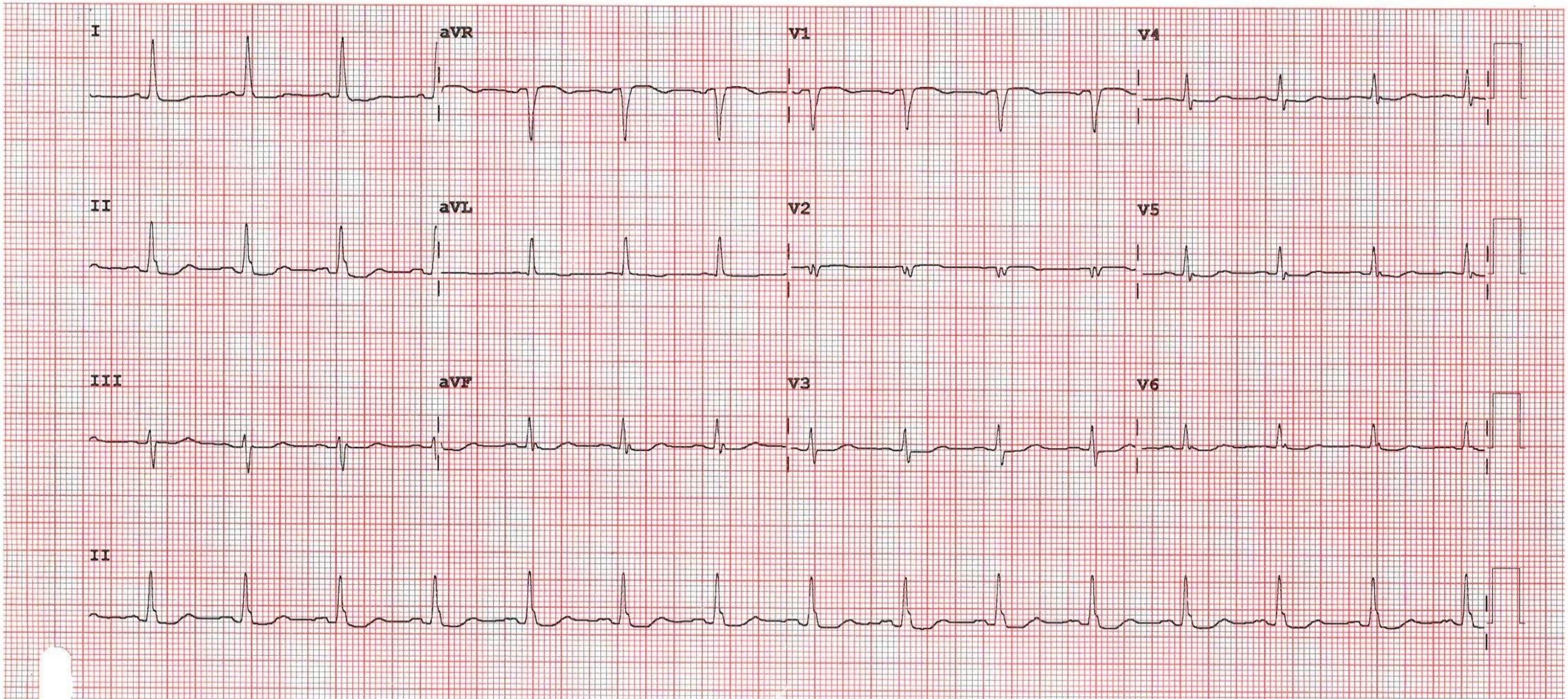
P	46
QRS	24
T	86

[redacted]

- ABNORMAL ECG -

12 Lead; Standard Placement

Unconfirmed Diagnosis



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

COVIDIEN Kendall REF 30768678

AHA/ACC/HRS Scientific Statement

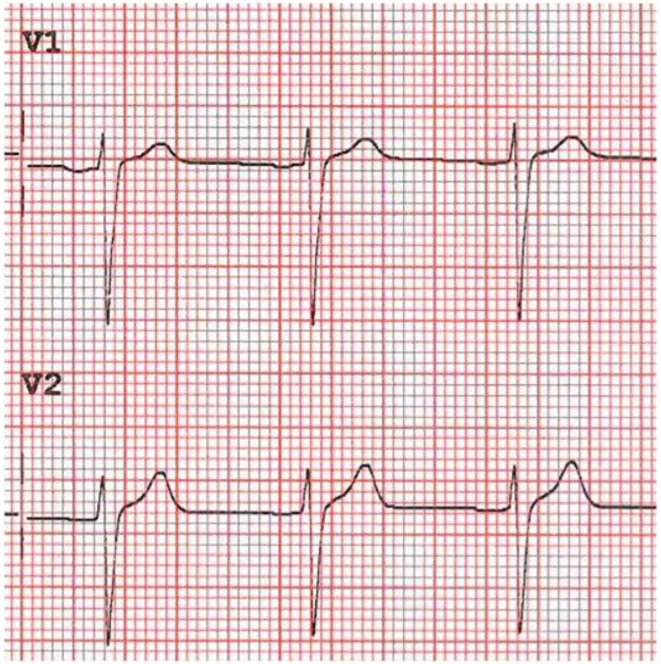
Recommendations for the Standardization and Interpretation of the Electrocardiogram

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]

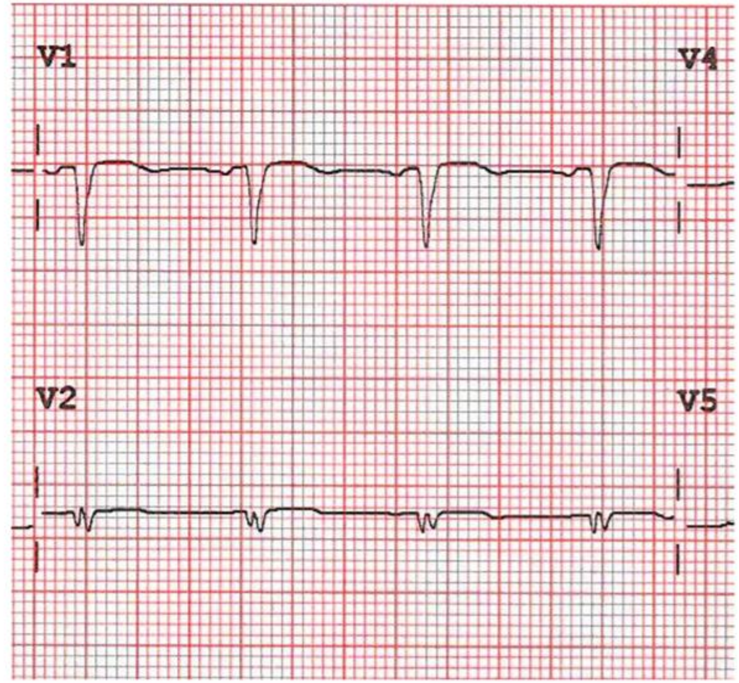
Kligfield et al   Standardization and Interpretation of the ECG, Part I

Correct Lead Placement



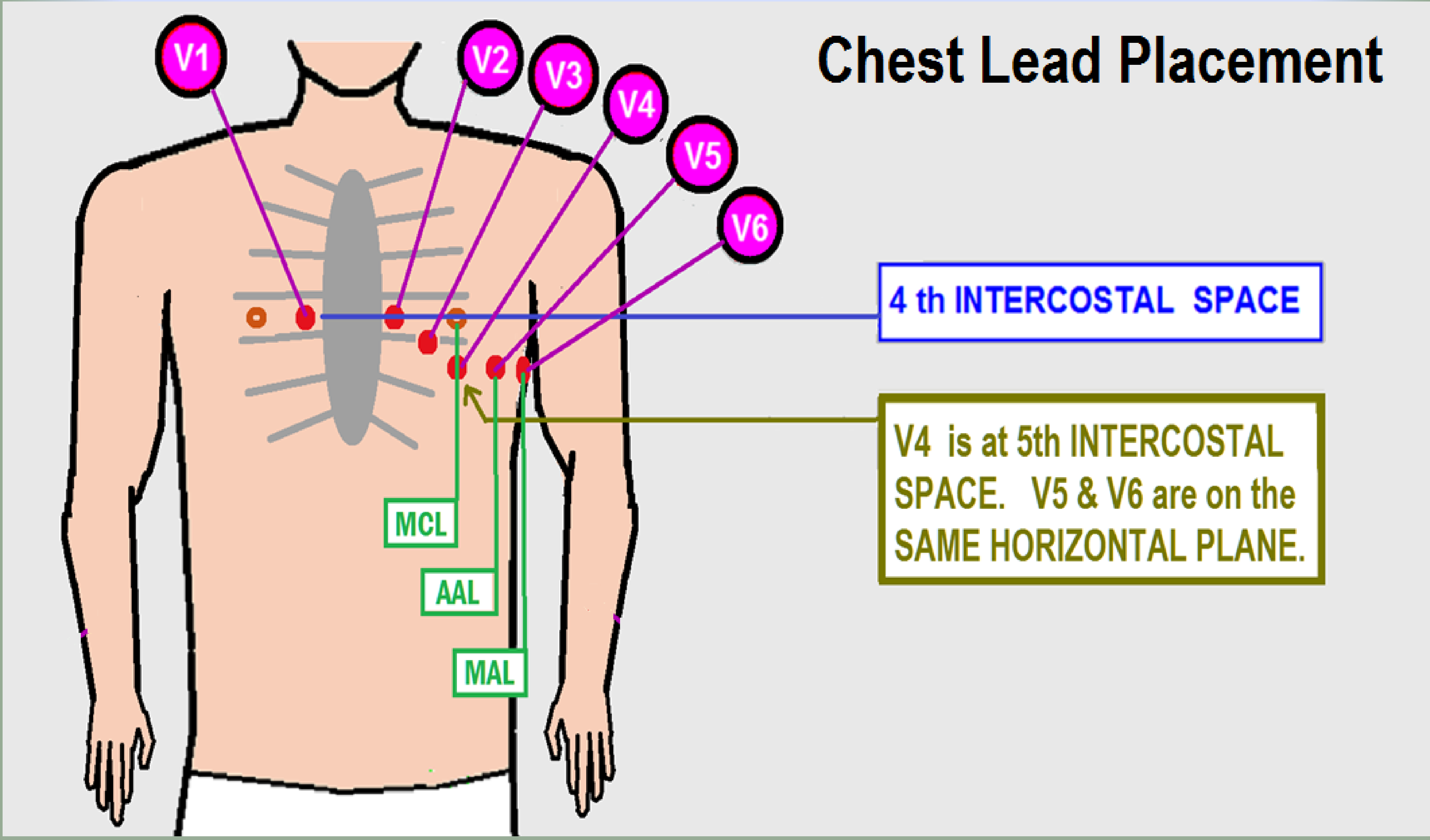
RS = NO old MI

Incorrect Lead Placement

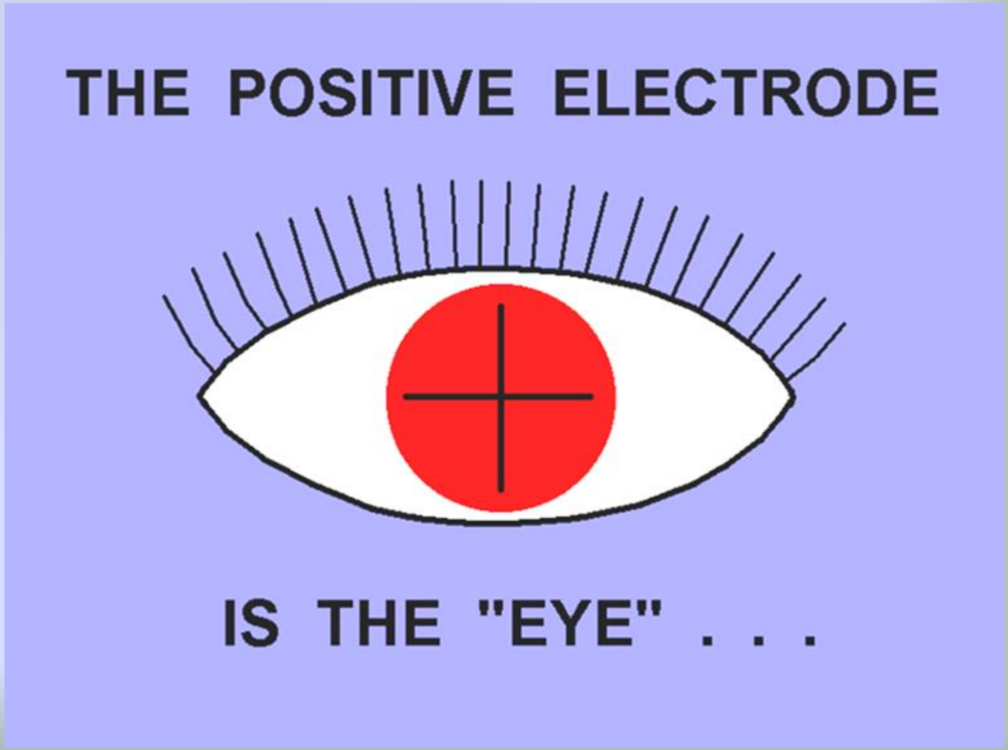


QS = old MI

CORRECT Lead placement:



What part of the HEART does each lead SEE ?



AREAS VIEWED by 12 LEAD ECG

AVR

AVL, I

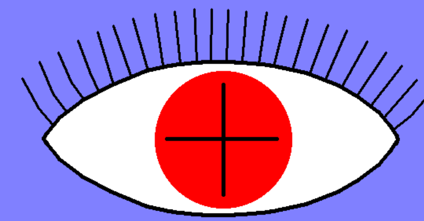
V1, V2

V3, V4

V5, V6

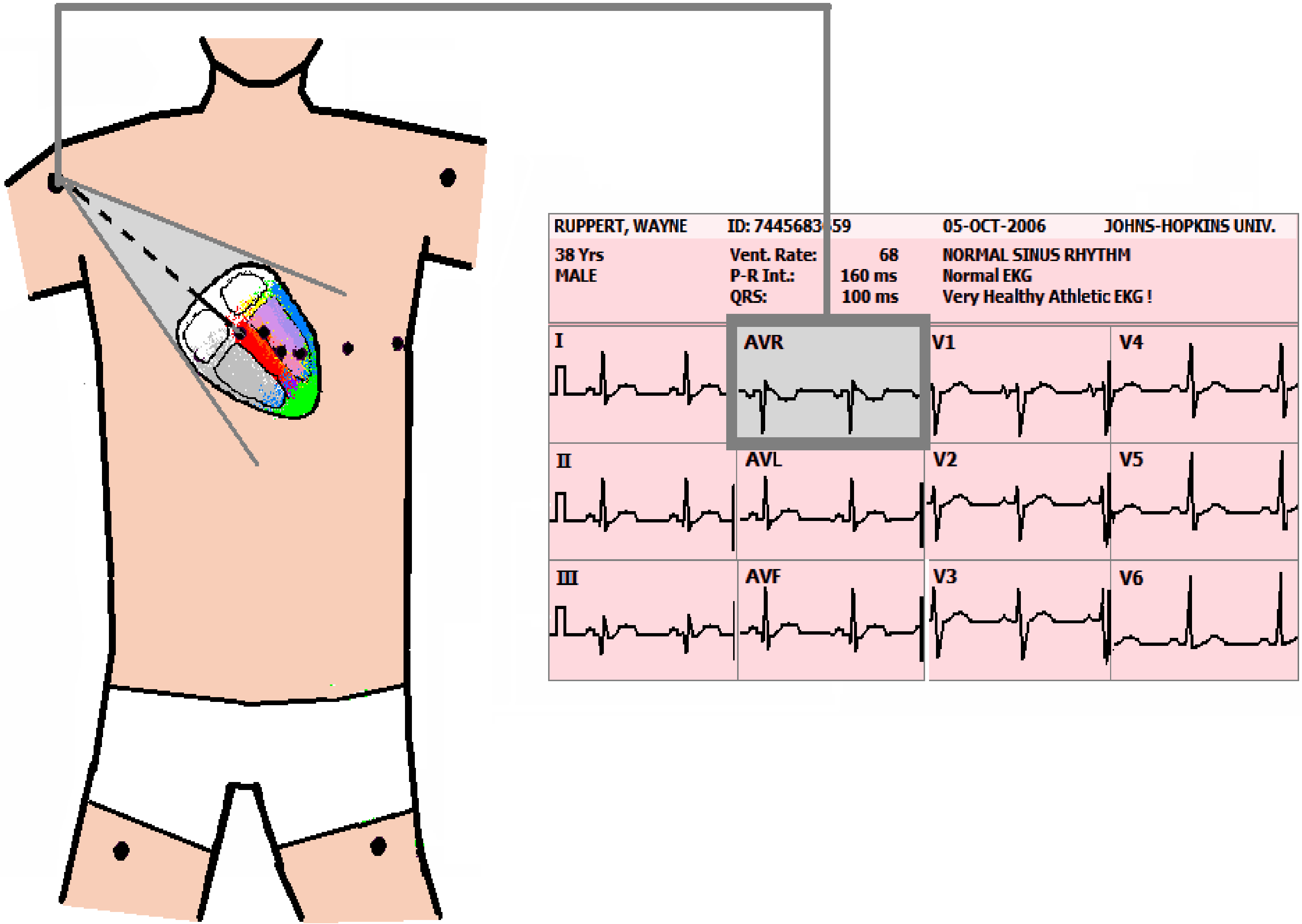
II, III, AVF

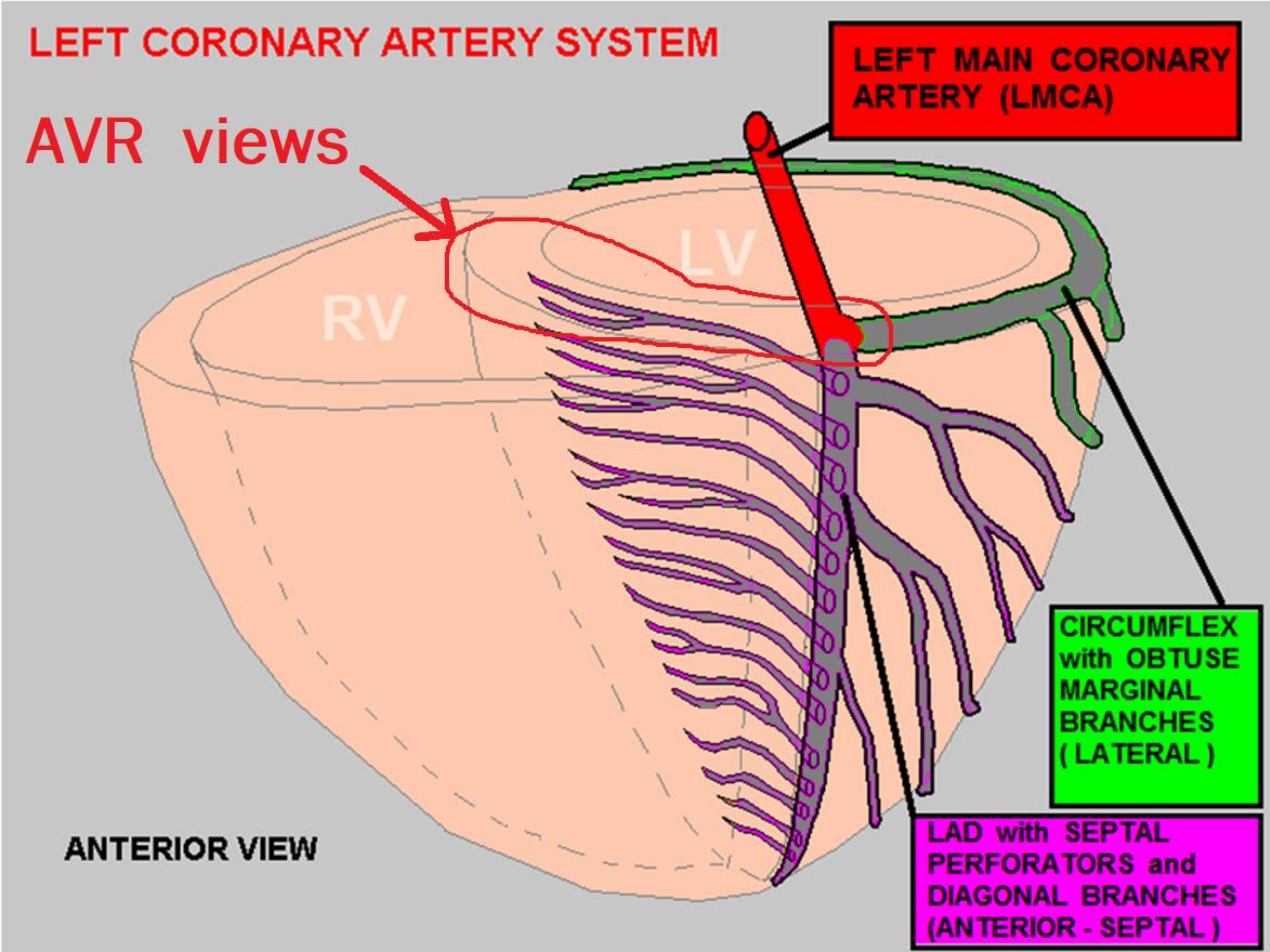
THE POSITIVE ELECTRODE



IS THE "EYE" . . .

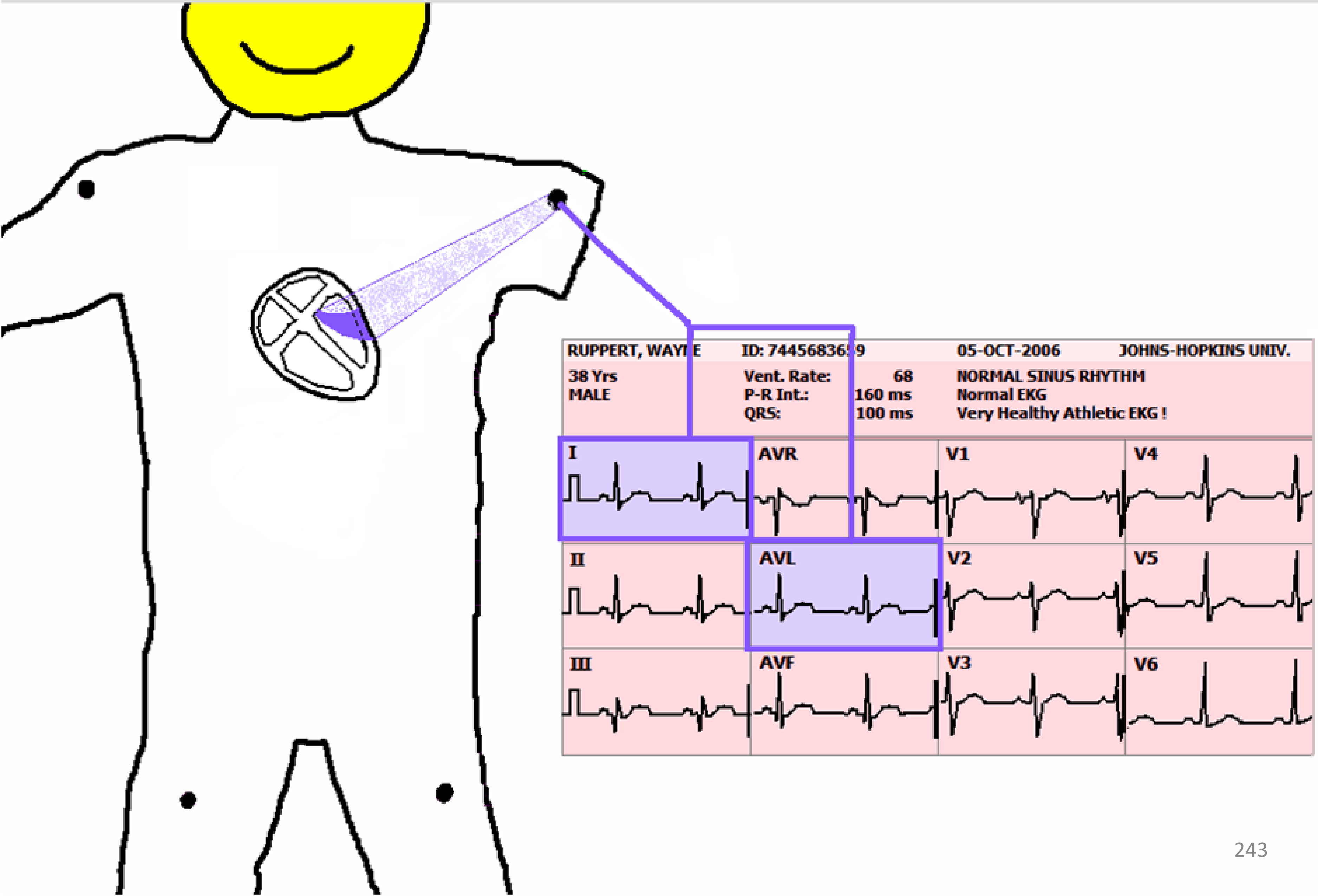
Lead AVR Views the BASILAR SEPTUM (region of the Bundle of His)





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

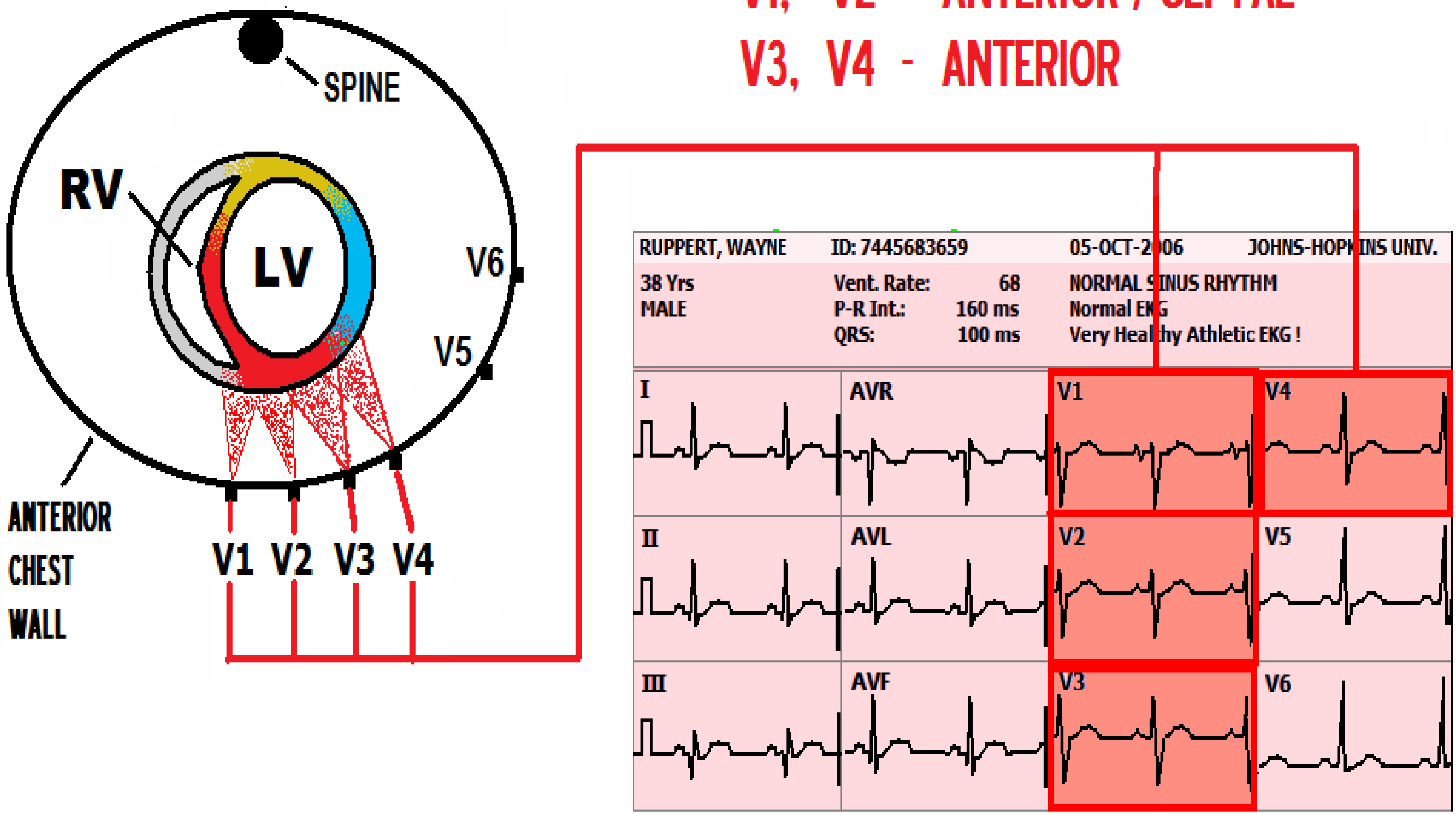
LEADS I and aVL VIEW the LATERAL - ANTERIOR WALL



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

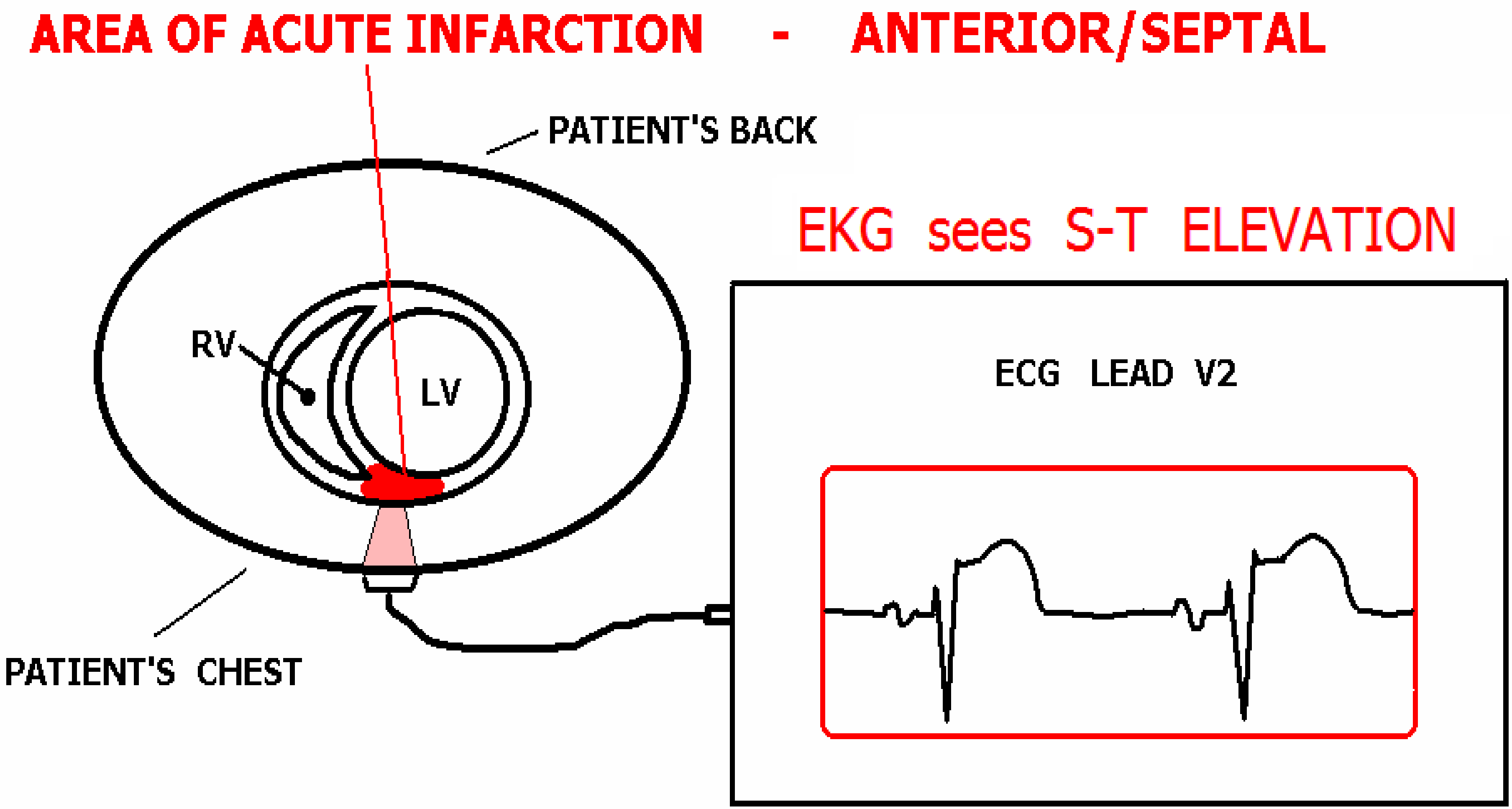
V1, V2 - ANTERIOR / SEPTAL

V3, V4 - ANTERIOR



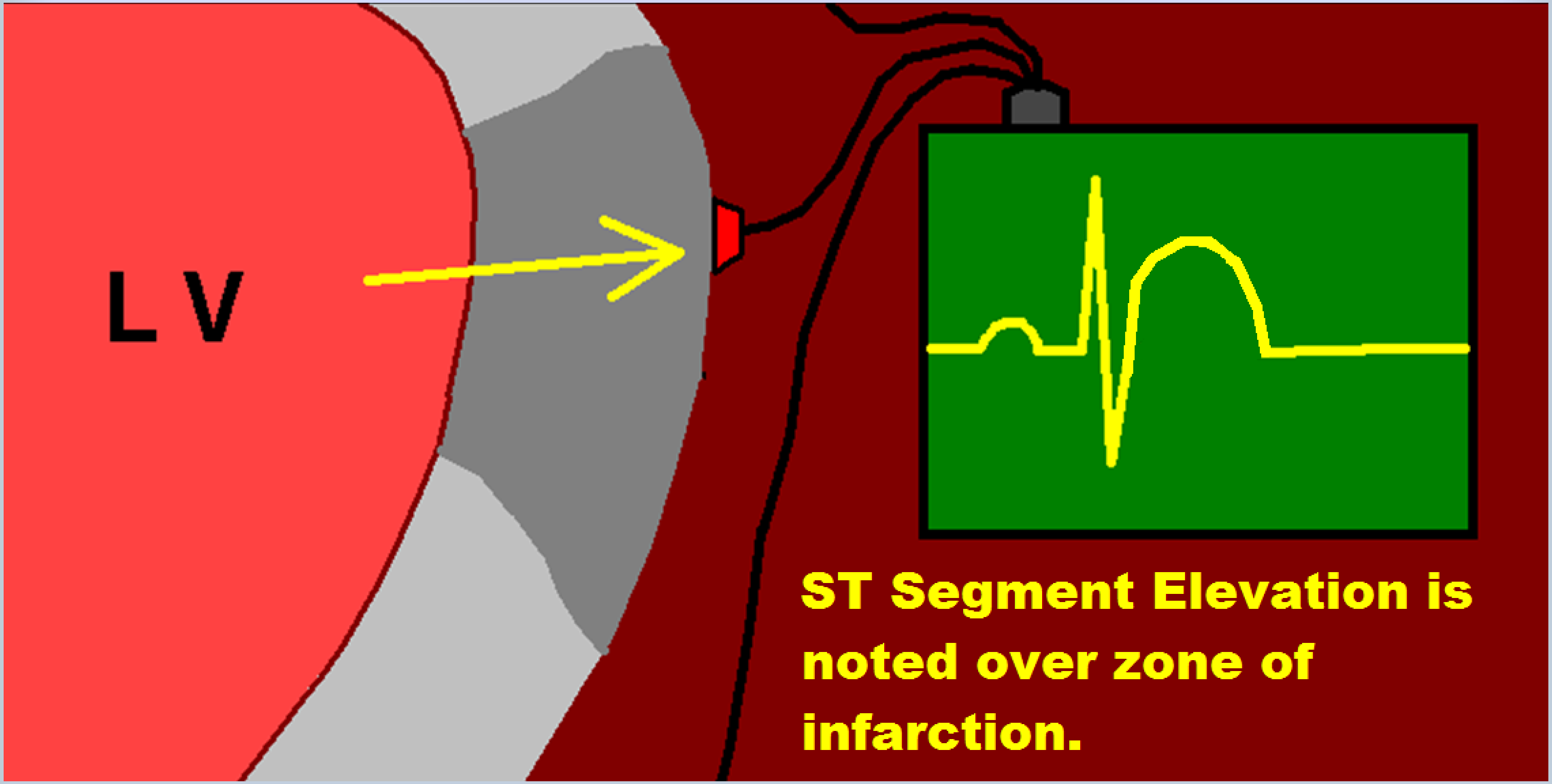
HOW EKG VIEWS INDICATIVE CHANGES

EXAMPLE:



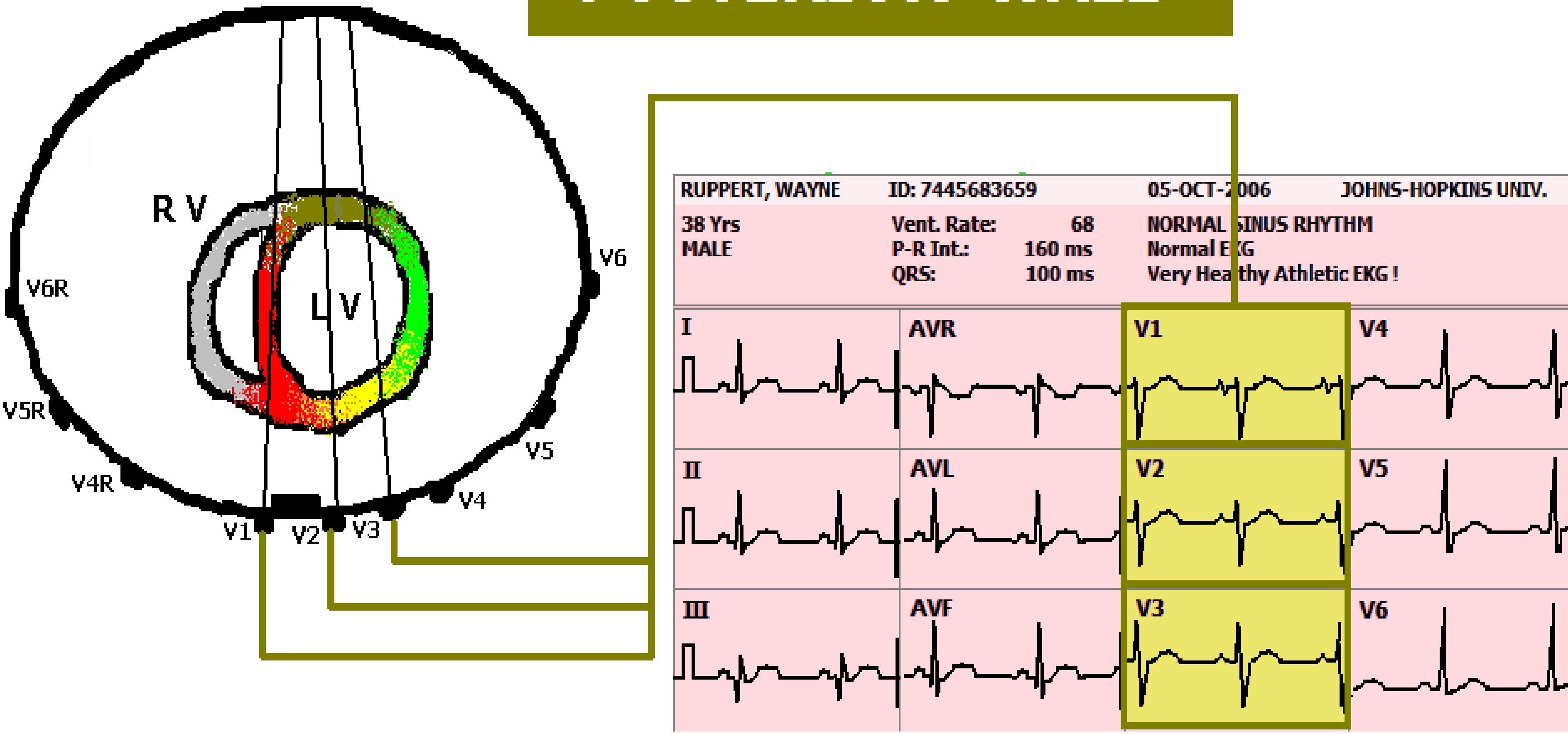
STEMI

- ST Segment Elevation Myocardial Infarction.



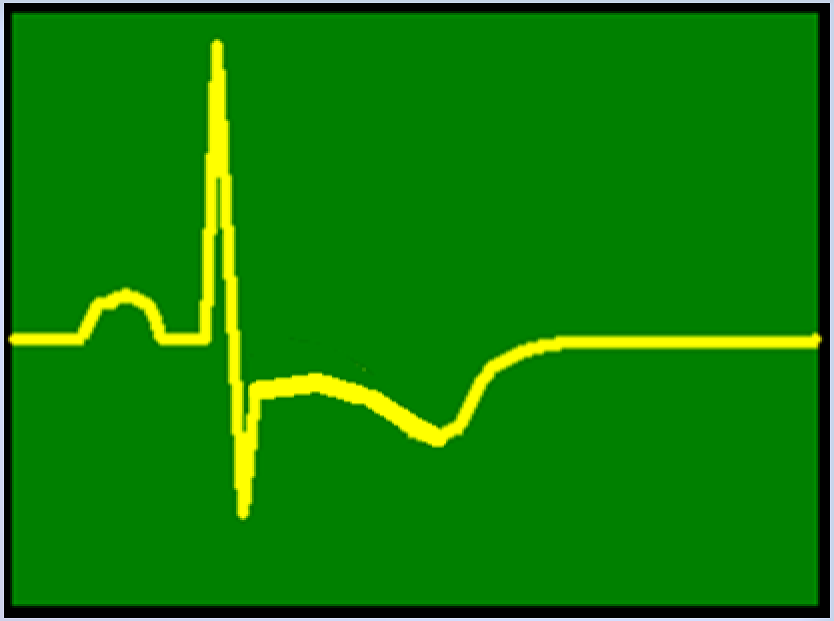
LEADS V1 - V3 *view the*

POSTERIOR WALL



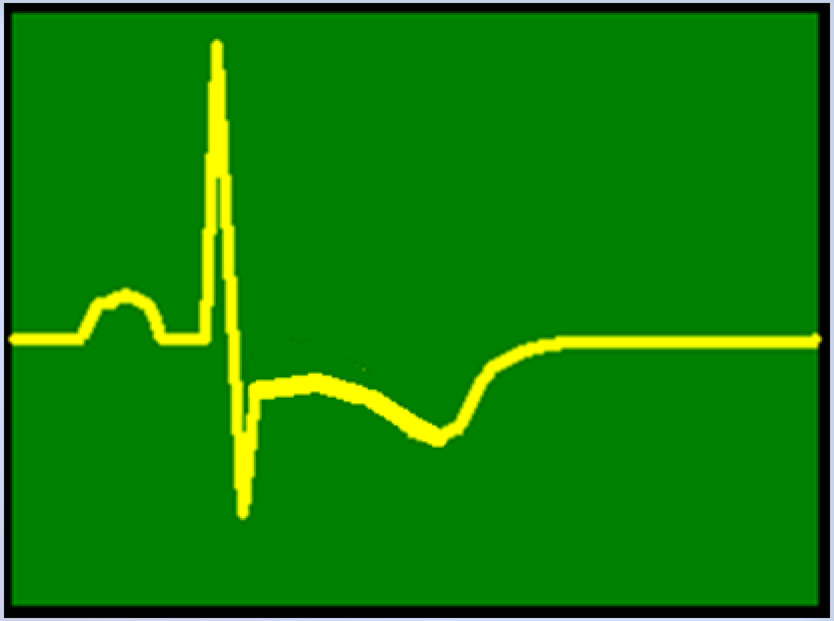
via RECIPROCAL CHANGES.

ST Depression in Leads V1 – V4:



- Direct view of ISCHEMIA (anterior wall)

ST Depression in Leads V1 – V4:

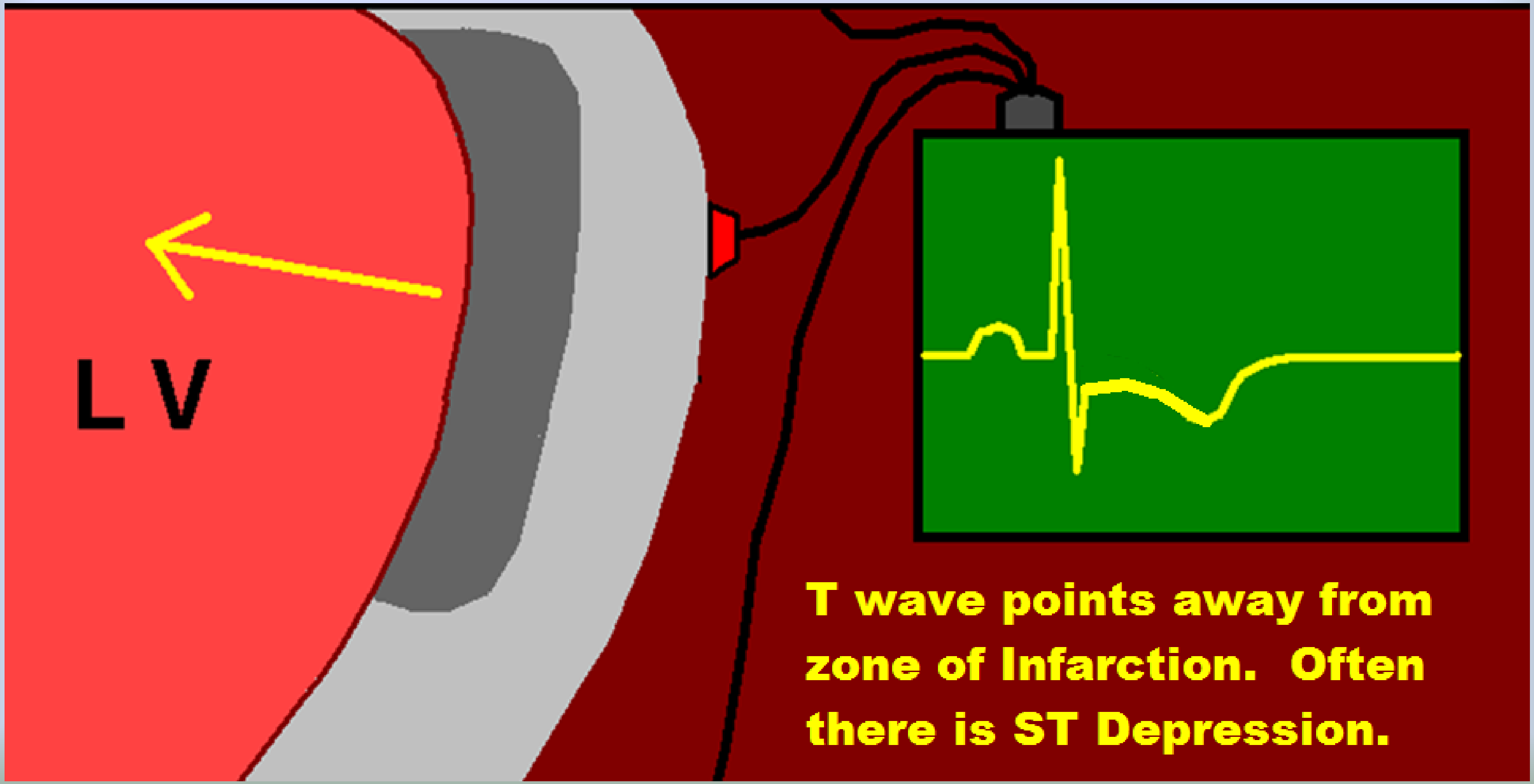


- **Direct view of ISCHEMIA (anterior wall)**
- **Direct view of NSTEMI (anterior wall)**

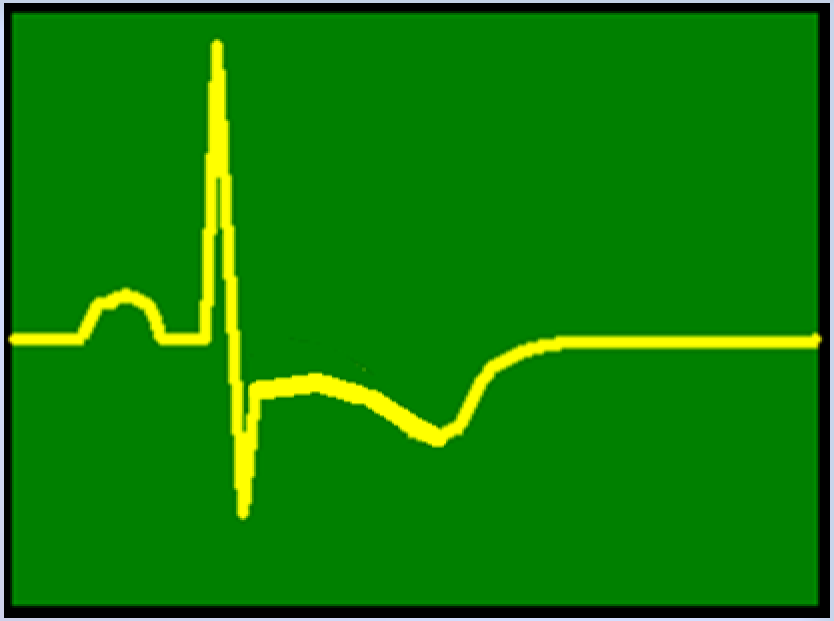
Non-STEMI (NSTEMI)

Non-ST Segment Elevation Myocardial Infarction.

“sub-endocardial MI” . . . “partial wall thickness”



ST Depression in Leads V1 – V4:

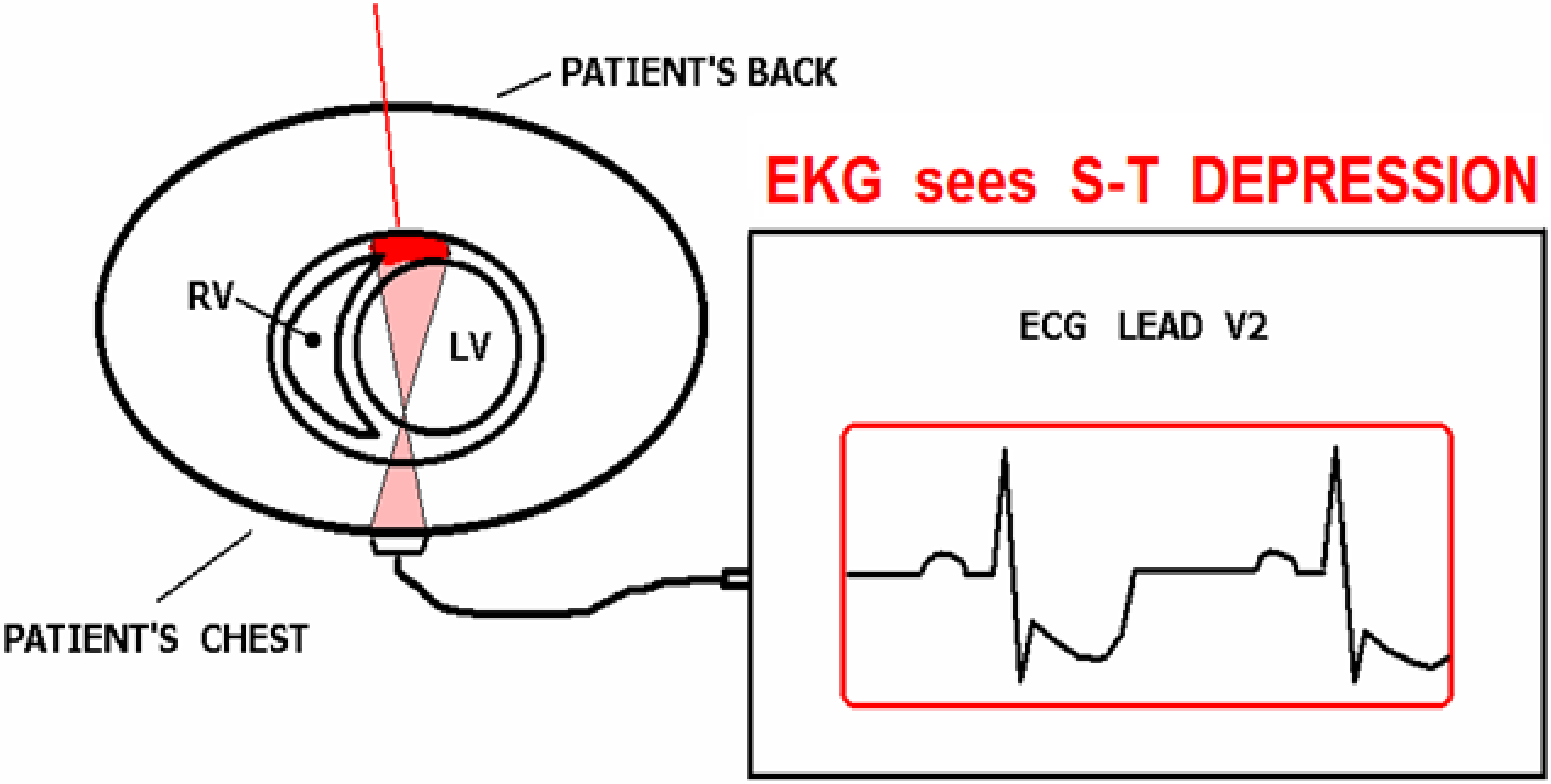


- Direct view of ISCHEMIA (anterior wall)
- Direct view of NSTEMI (anterior wall)
- Reciprocal view of STEMI (opposite side of heart - posterior wall)

HOW EKG VIEWS RECIPROCAL CHANGES

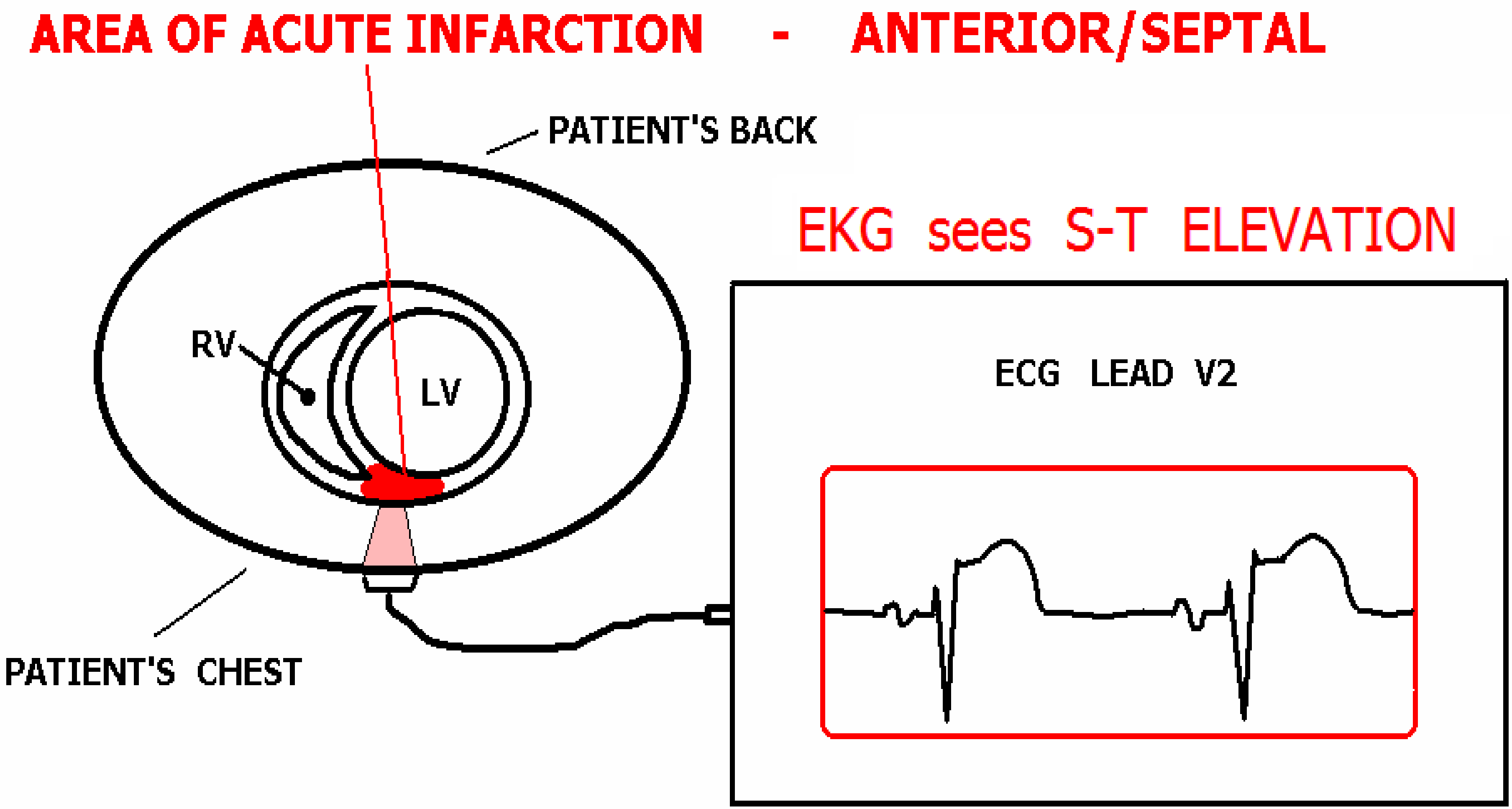
EXAMPLE:

AREA OF ACUTE INFARCTION - POSTERIOR WALL



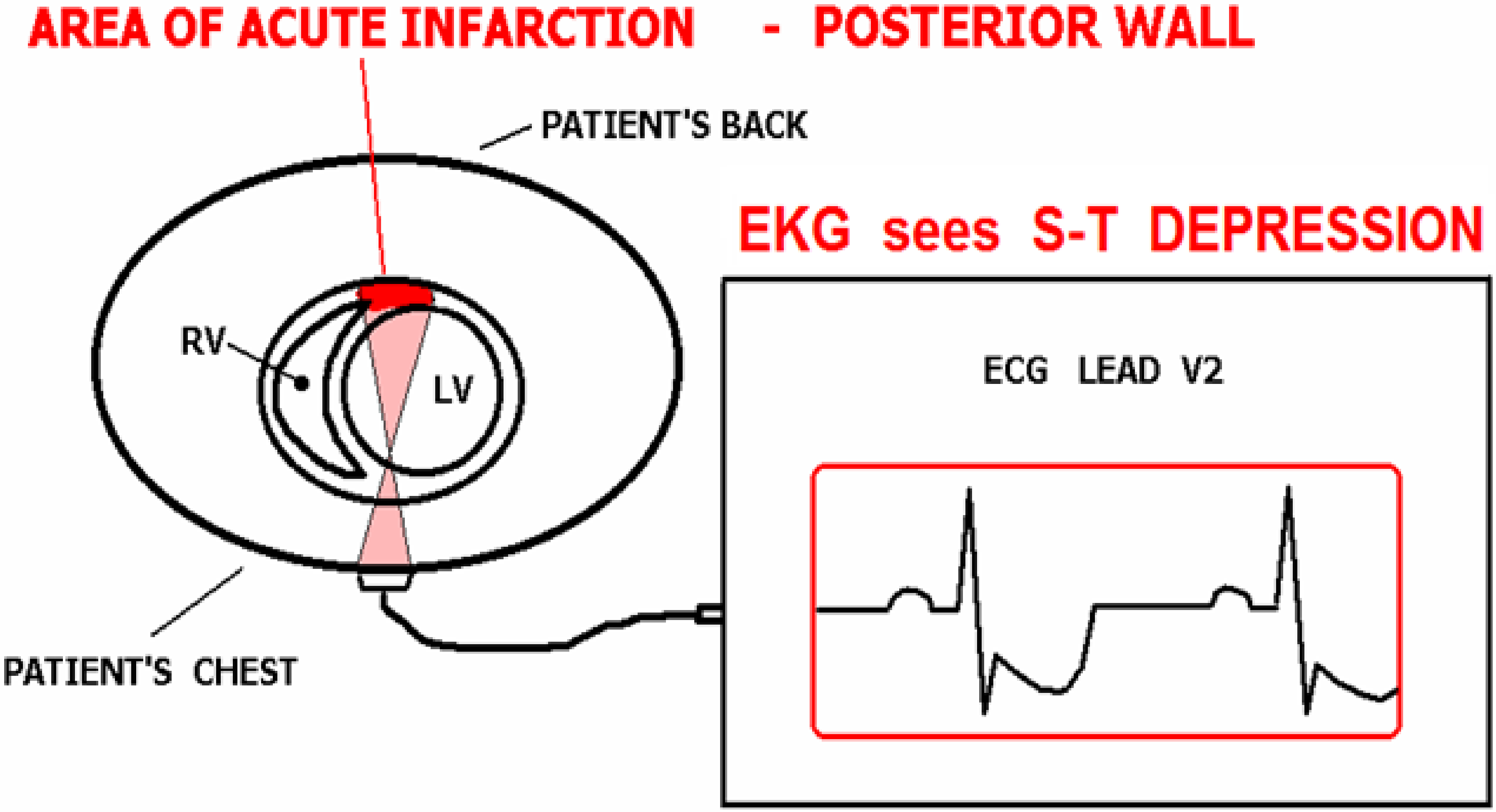
HOW EKG VIEWS INDICATIVE CHANGES

EXAMPLE:



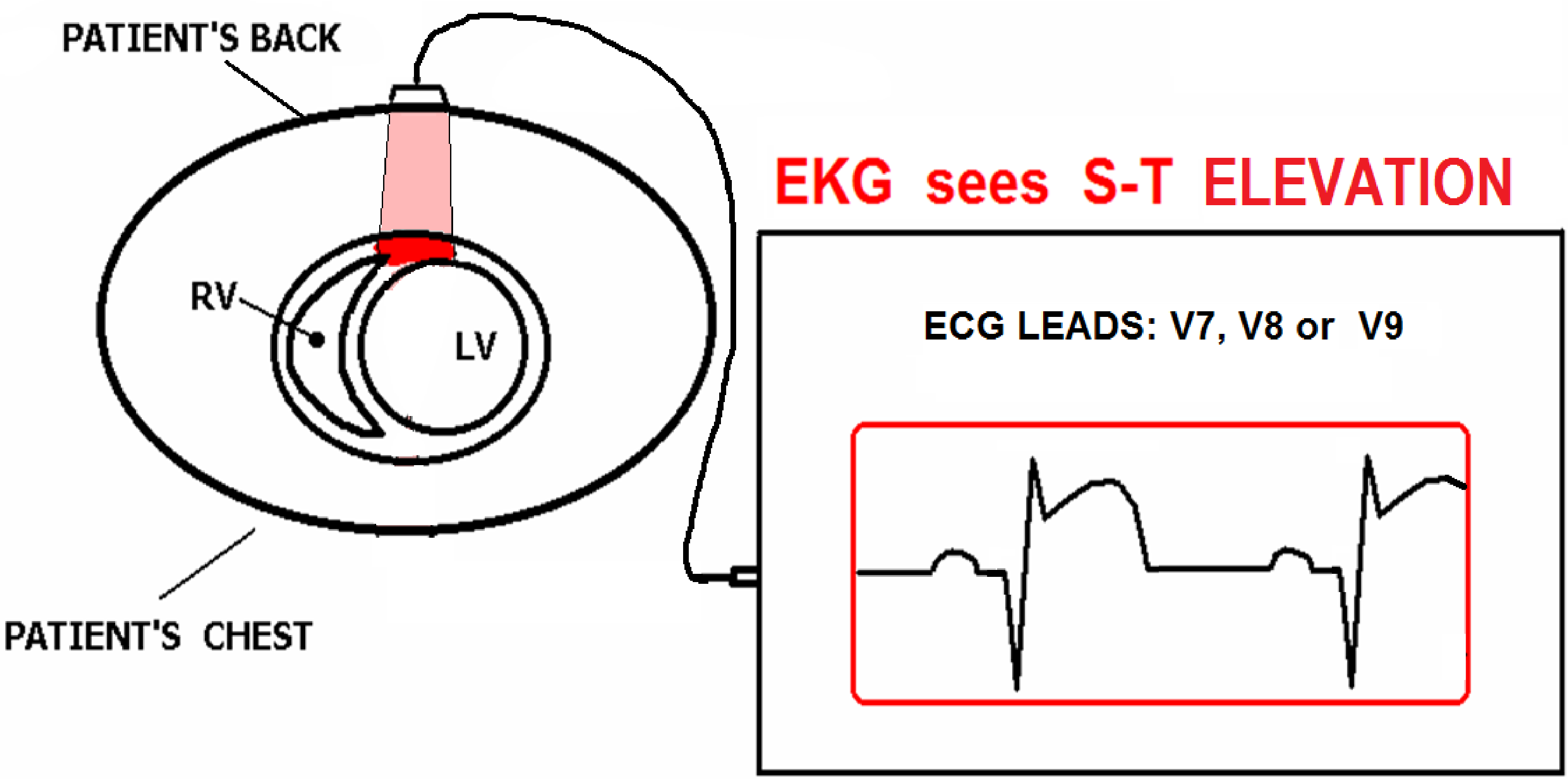
HOW EKG VIEWS RECIPROCAL CHANGES

EXAMPLE:

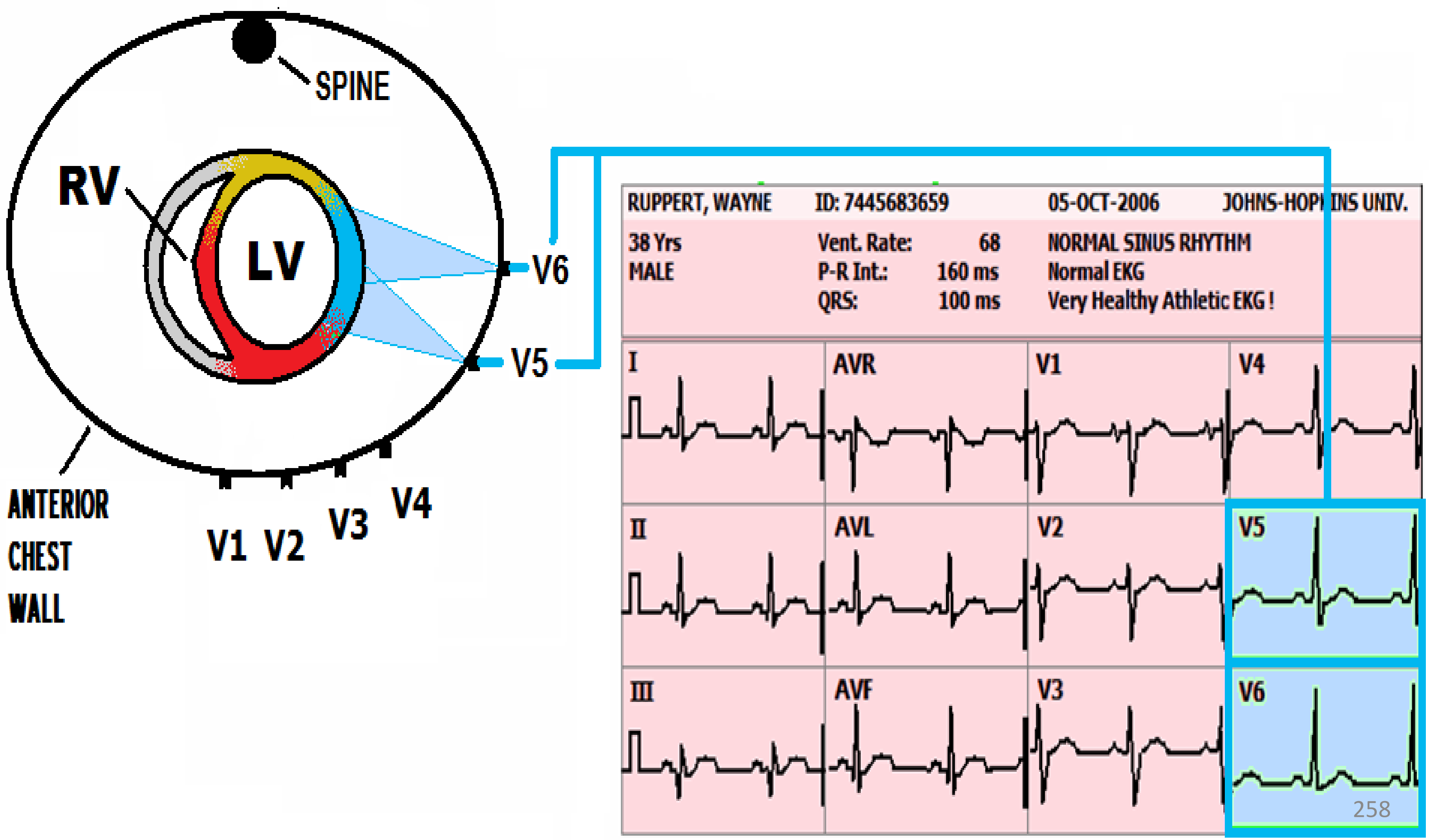


If we put ECG leads on the BACK of a PATIENT who is having an

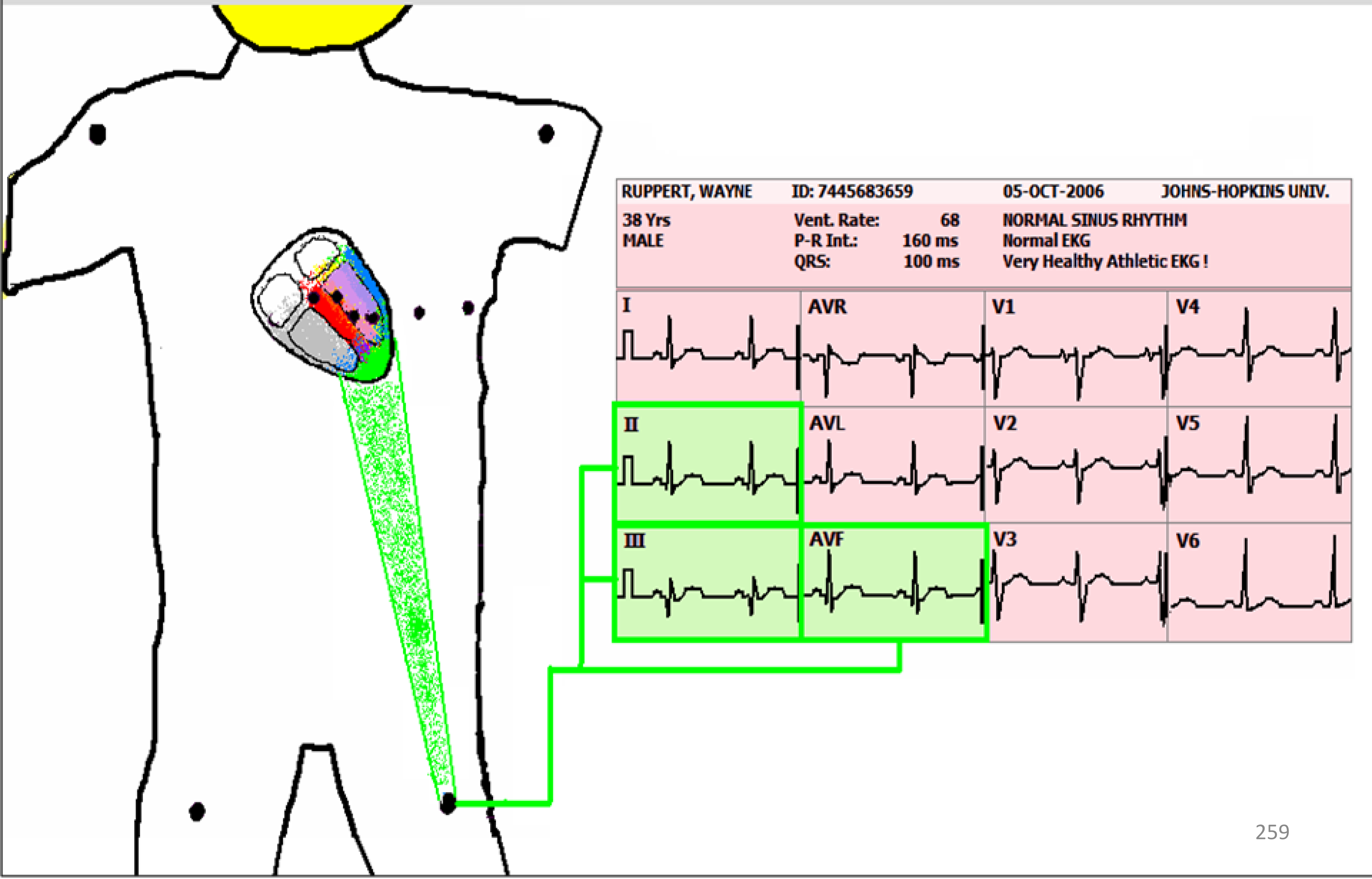
ACUTE POSTERIOR WALL MI . . . . .

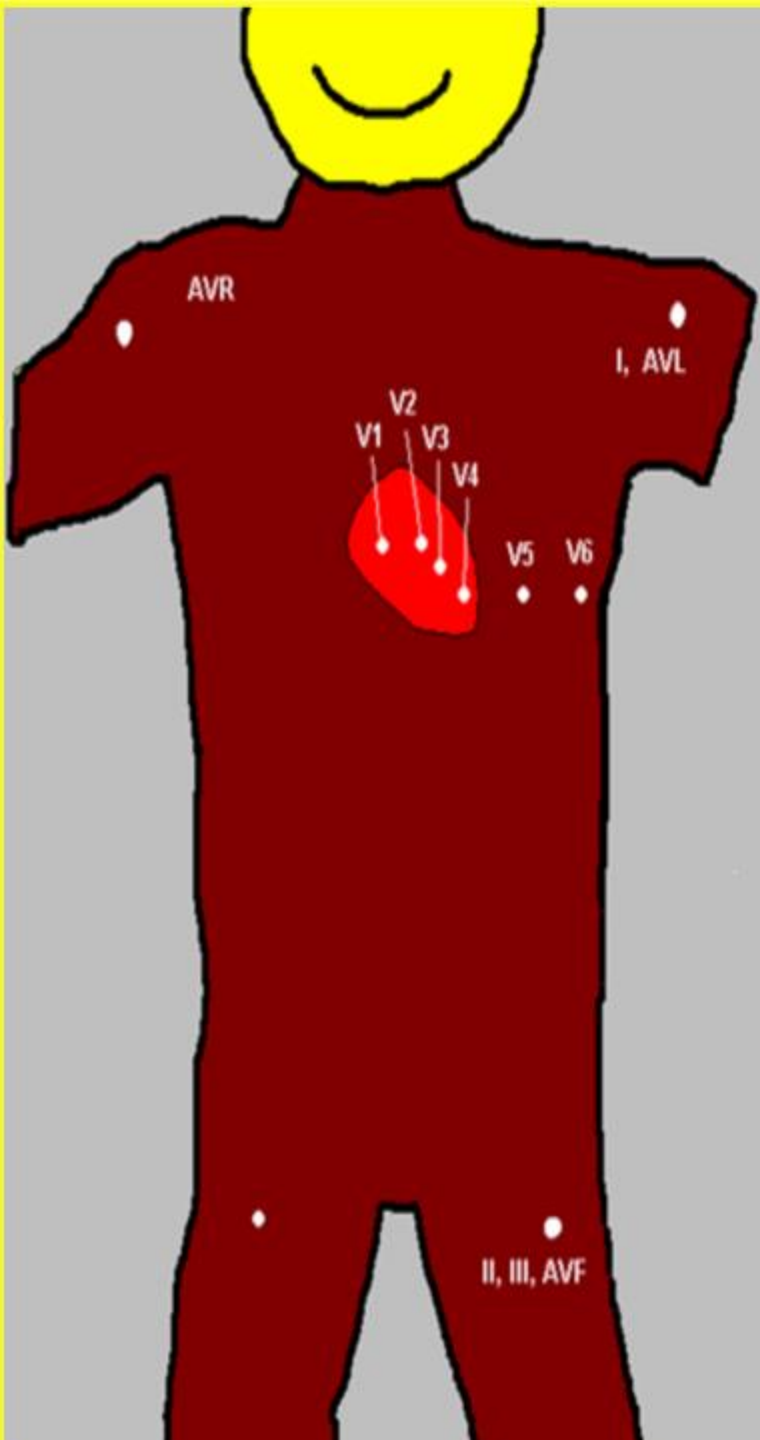


V5 - V6 VIEW THE LATERAL WALL of the LEFT VENTRICLE



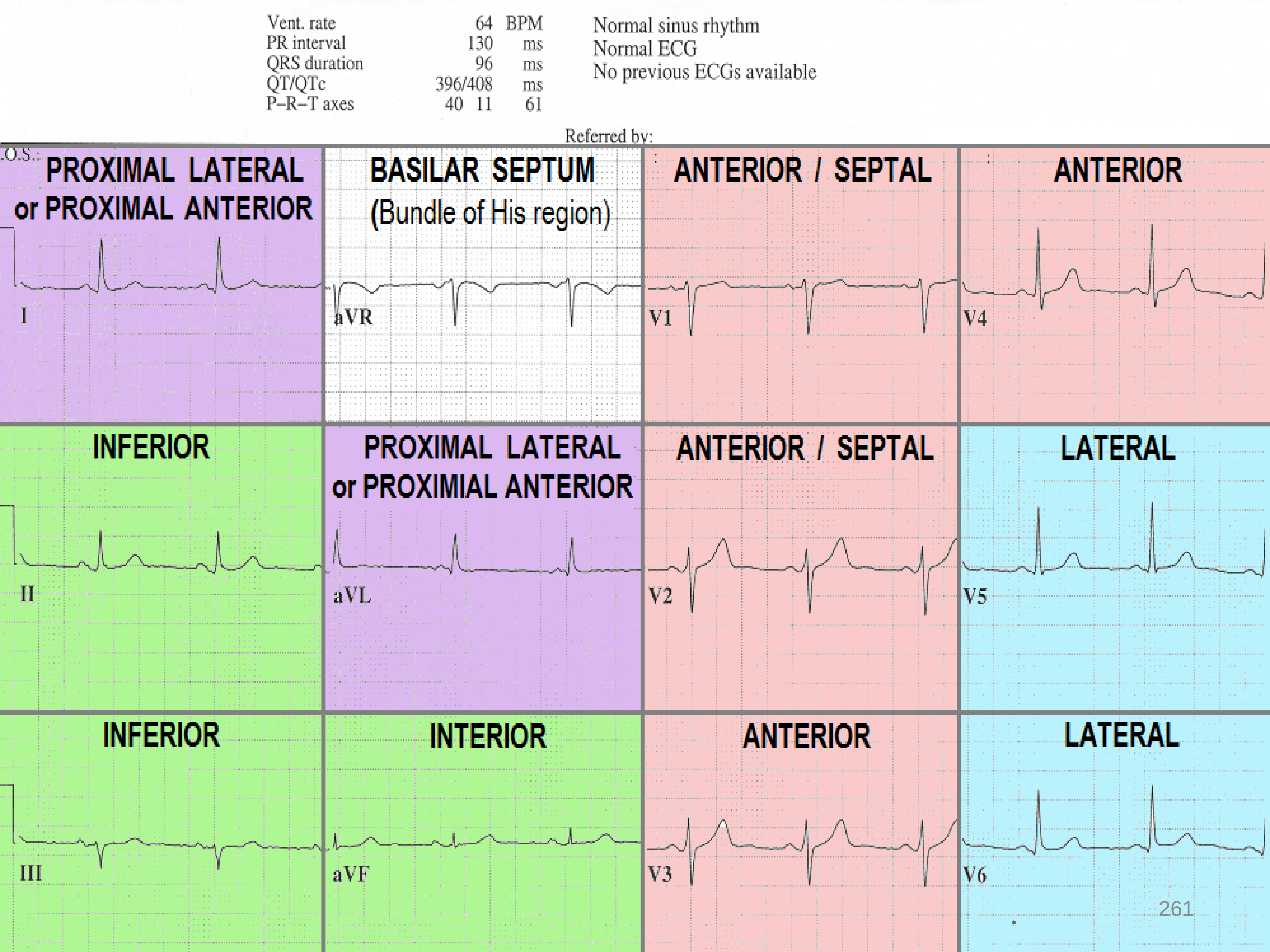
LEADS II, III, and aVF VIEW
INFERIOR WALL of the LEFT VENTRICLE





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

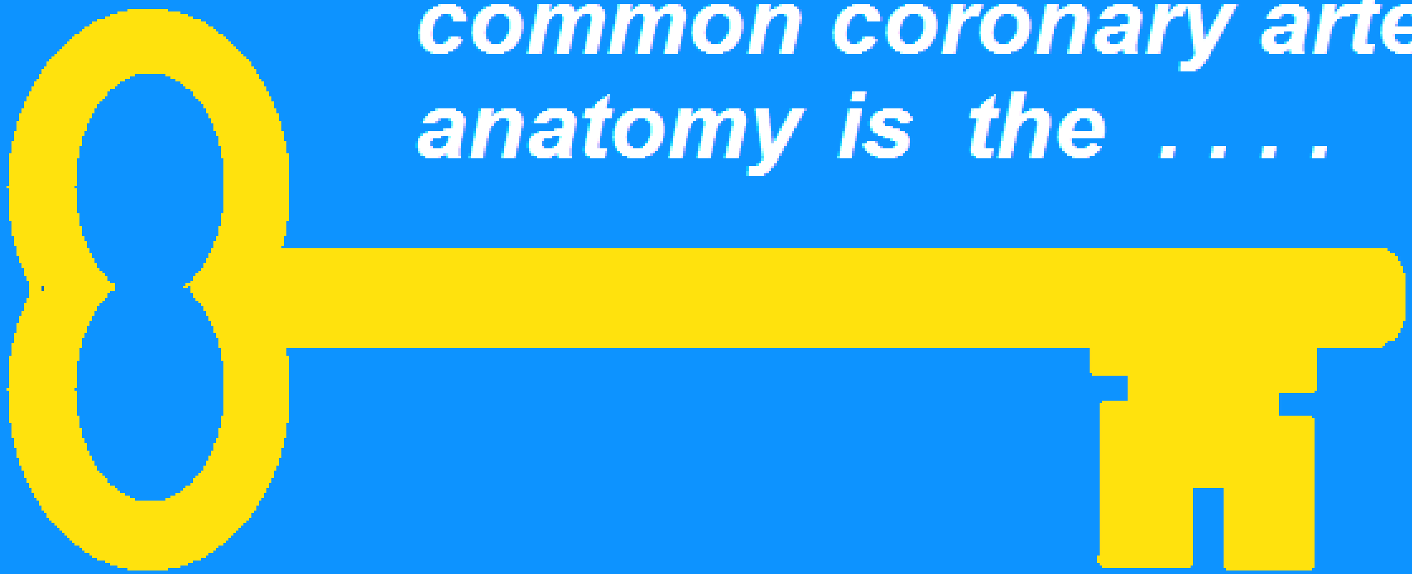

Vent. rate 64 BPM
PR interval 130 ms
QRS duration 96 ms
QT/QTc 396/408 ms
P–R–T axes 40 11 61
Normal sinus rhythm
Normal ECG
No previous ECGs available
Referred by:
PROXIMAL LATERAL or PROXIMAL ANTERIOR
I
BASILAR SEPTUM (Bundle of His region)
aVR
ANTERIOR / SEPTAL
V1
ANTERIOR
V4
INFERIOR
II
PROXIMAL LATERAL or PROXIMIAL ANTERIOR
aVL
ANTERIOR / SEPTAL
V2
LATERAL
V5
INFERIOR
III
INTERIOR
aVF
ANTERIOR
V3
LATERAL
V6

THE CORONARY

ARTERIES

STRUCTURES SERVED BY THE CORONARY ARTERIES

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



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

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

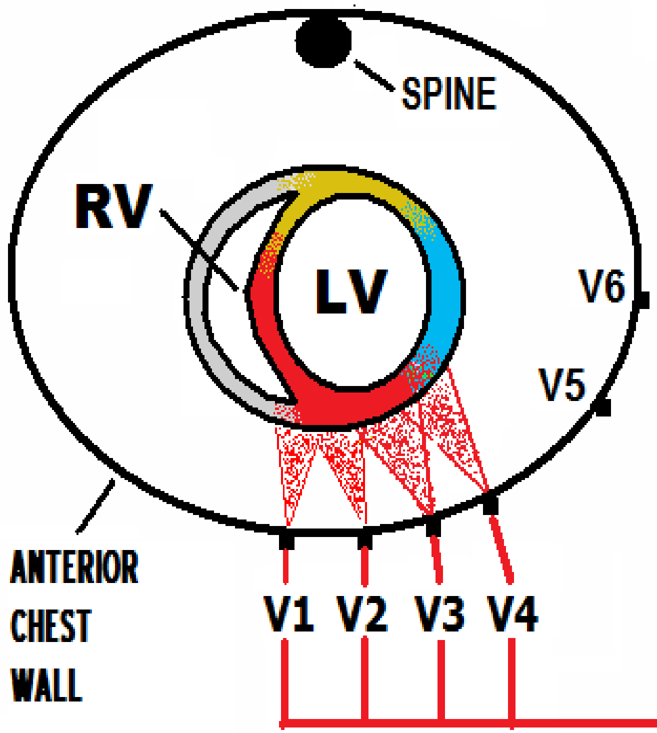
The 12 Lead ECG becomes your "crystal ball !!"

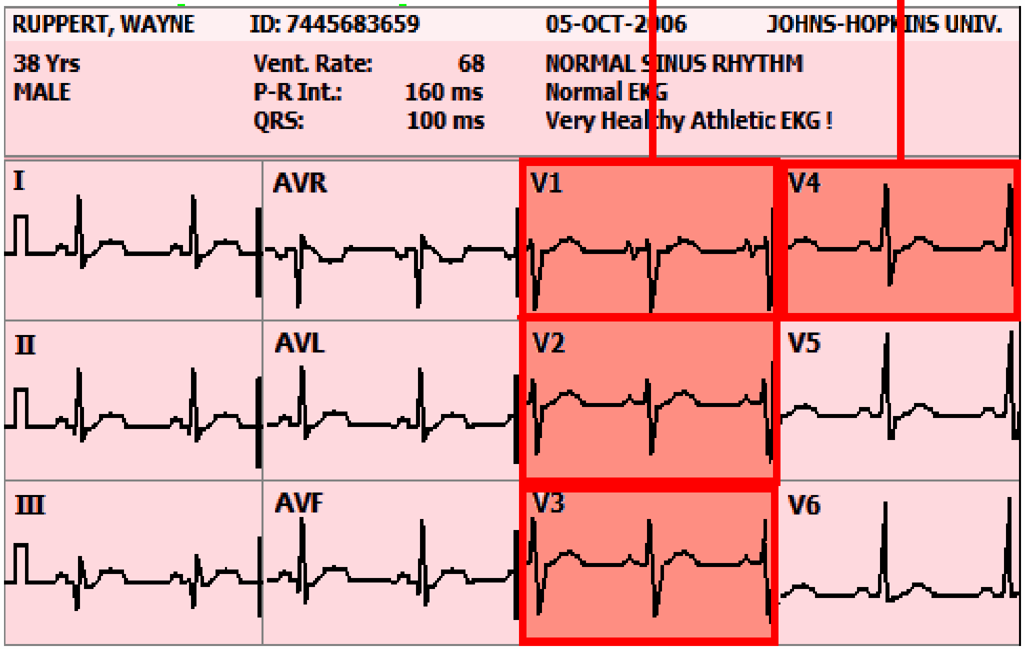
INTERPRET THE EKG, THEN:

- 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

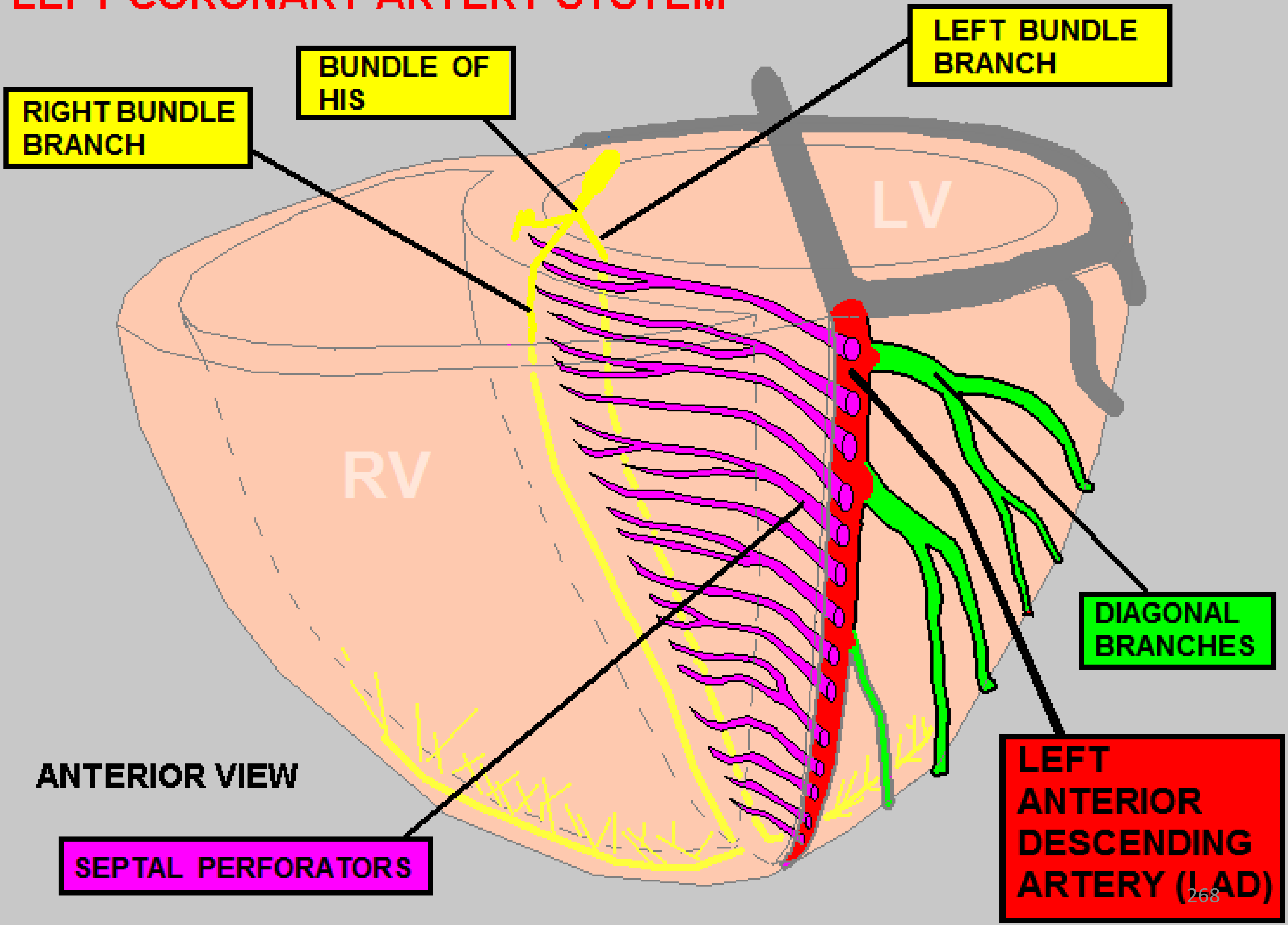
V1, V2 - ANTERIOR / SEPTAL
V3, V4 - ANTERIOR



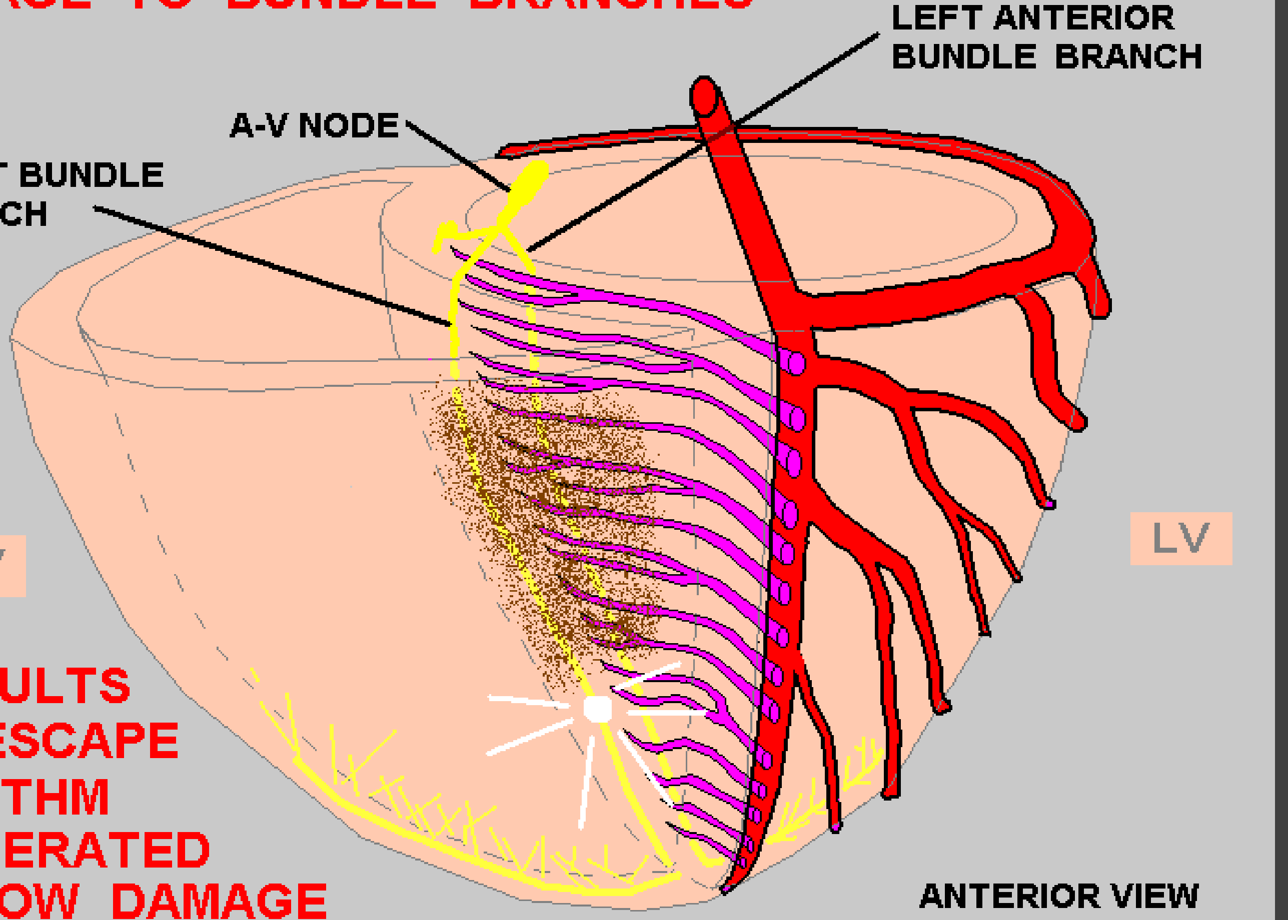


Which Coronary Artery typically Supplies the ANTERIOR WALL ?

LEFT CORONARY ARTERY SYSTEM



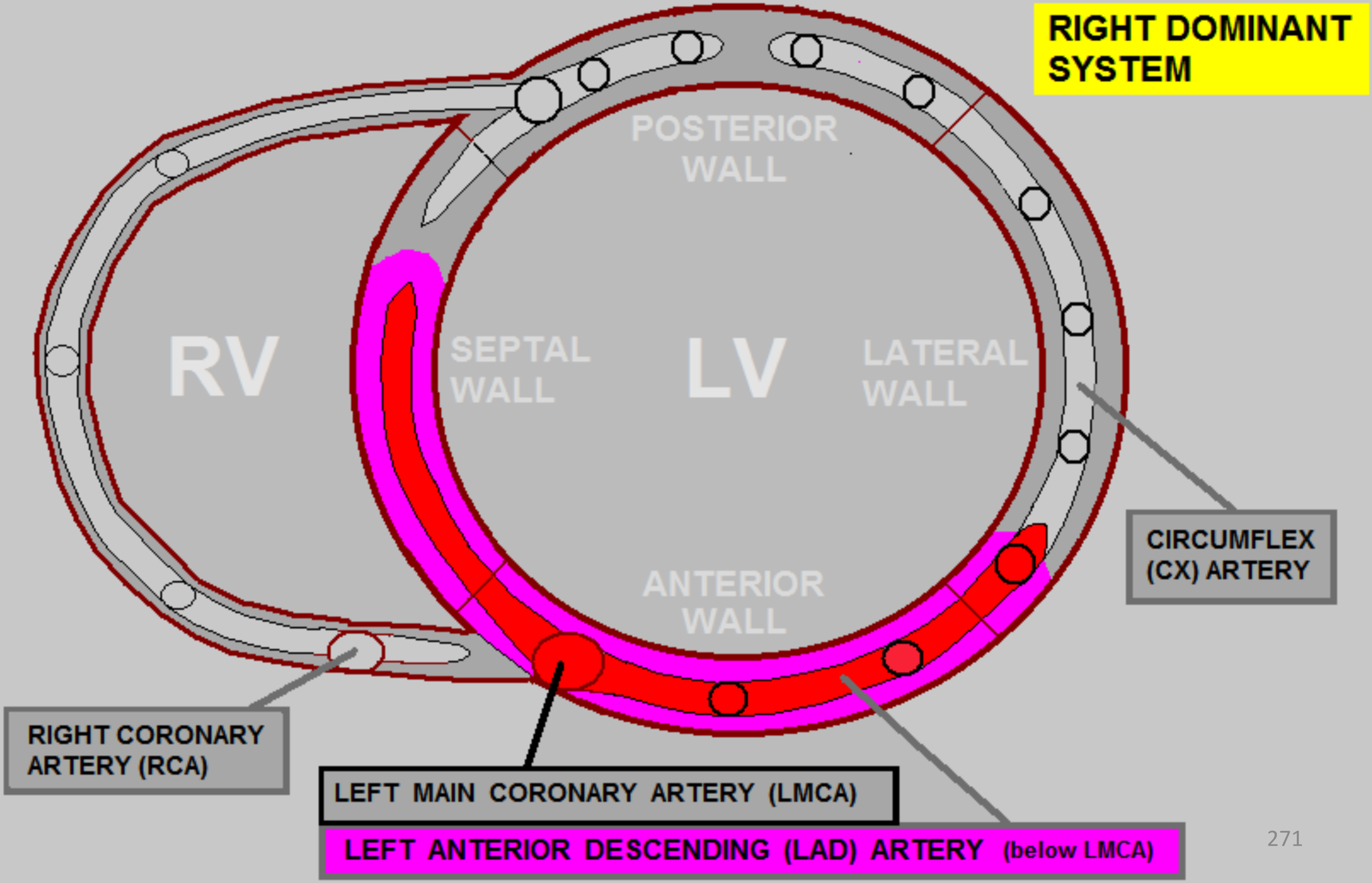
DAMAGE TO BUNDLE BRANCHES



RESULTS IN ESCAPE RHYTHM GENERATED BELOW DAMAGE *cutaway view of the*

LEFT ANTERIOR DESCENDING ARTERY (LAD)

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



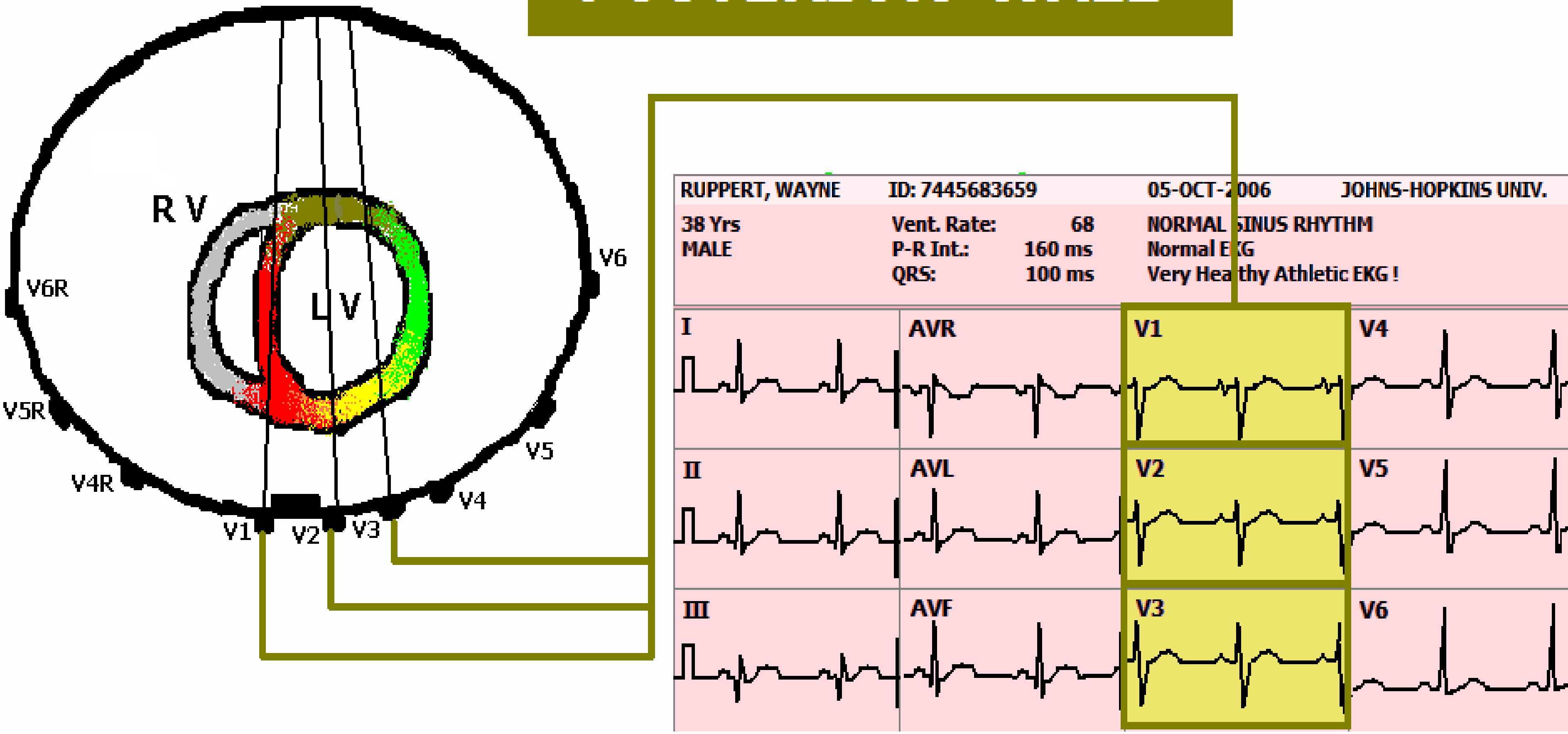
HELPFUL HINT . . . MEMORIZE THIS !

LEFT ANTERIOR DESCENDING ARTERY (LAD)

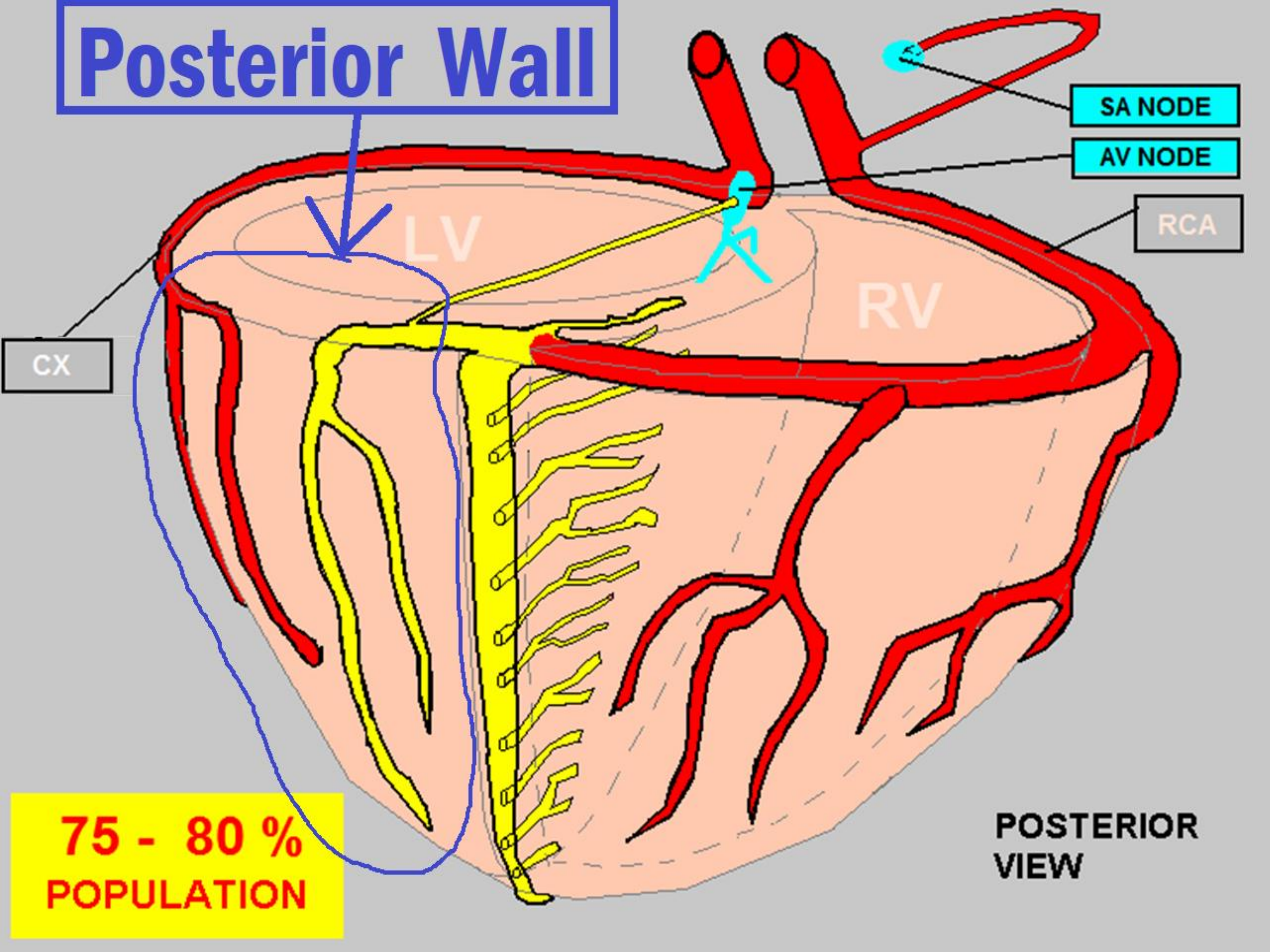
- BUNDLE OF HIS
- BUNDLE BRANCHES ( )
- 35 - 45 % OF LV MUSCLE MASS
  - ANTERIOR WALL
  - SEPTAL WALL (anterior 2/3)

LEADS V1 - V3 *view the*

POSTERIOR WALL

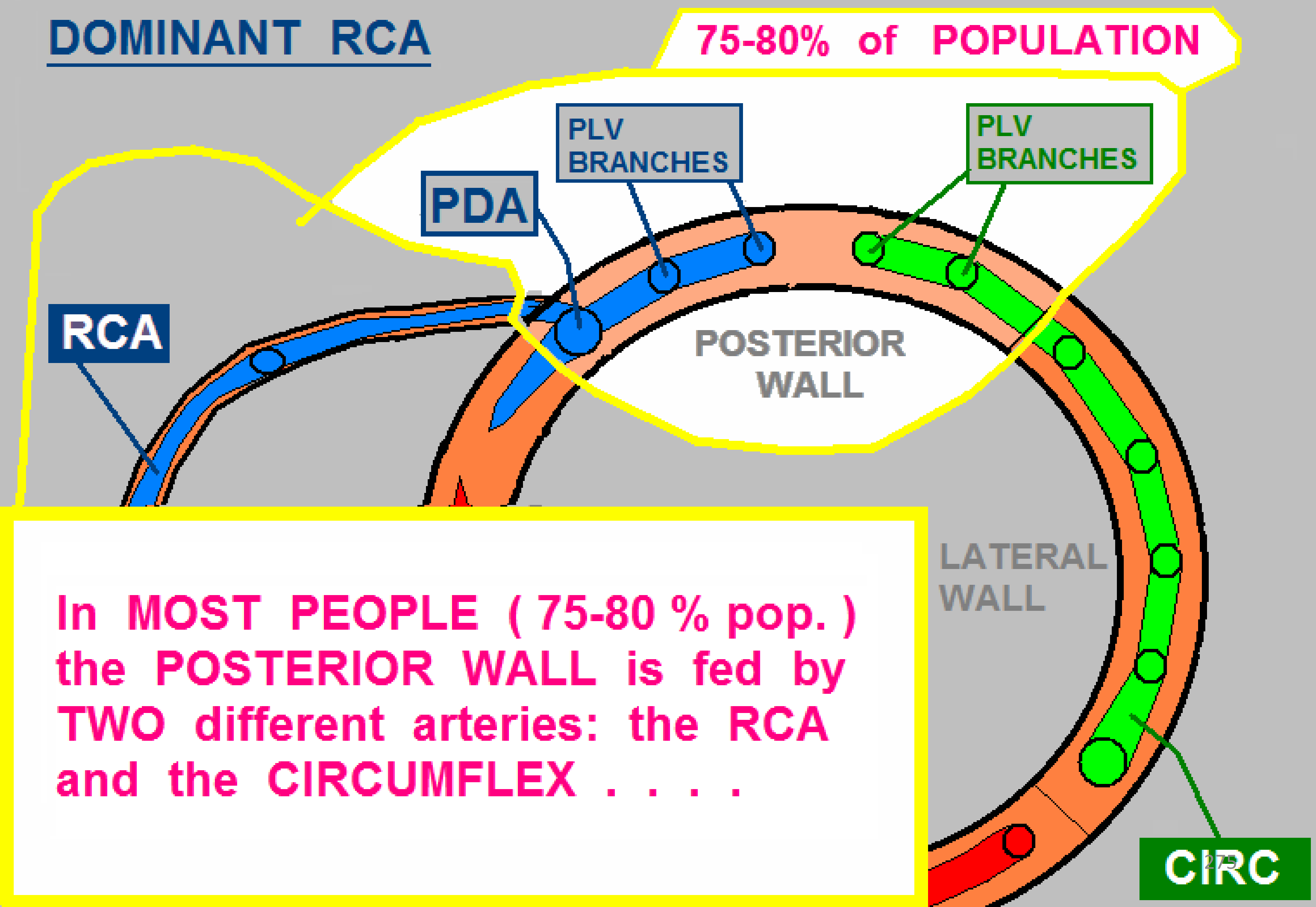


via RECIPROCAL CHANGES.



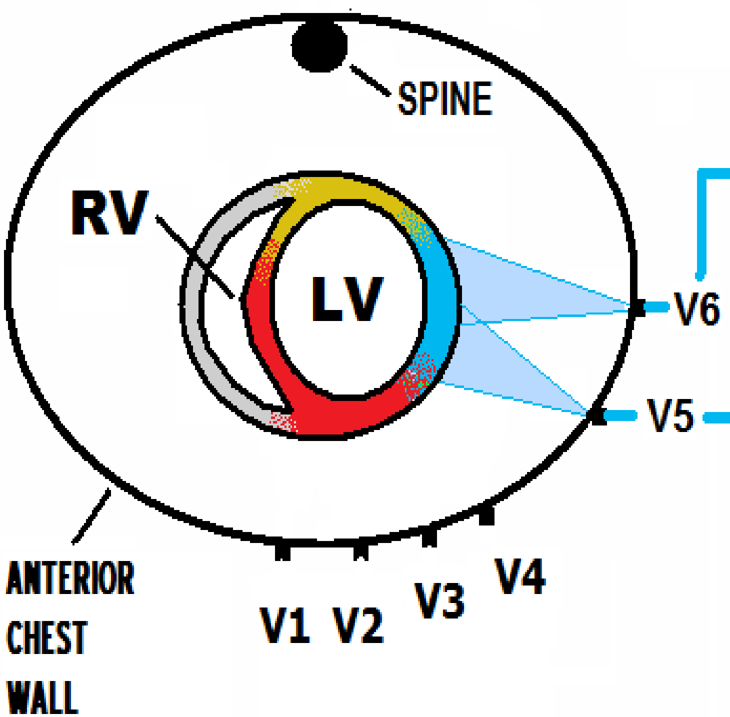
Posterior Wall
SA NODE
AV NODE
RCA
LV
RV
CX
75 - 80 %
POPULATION
POSTERIOR
VIEW

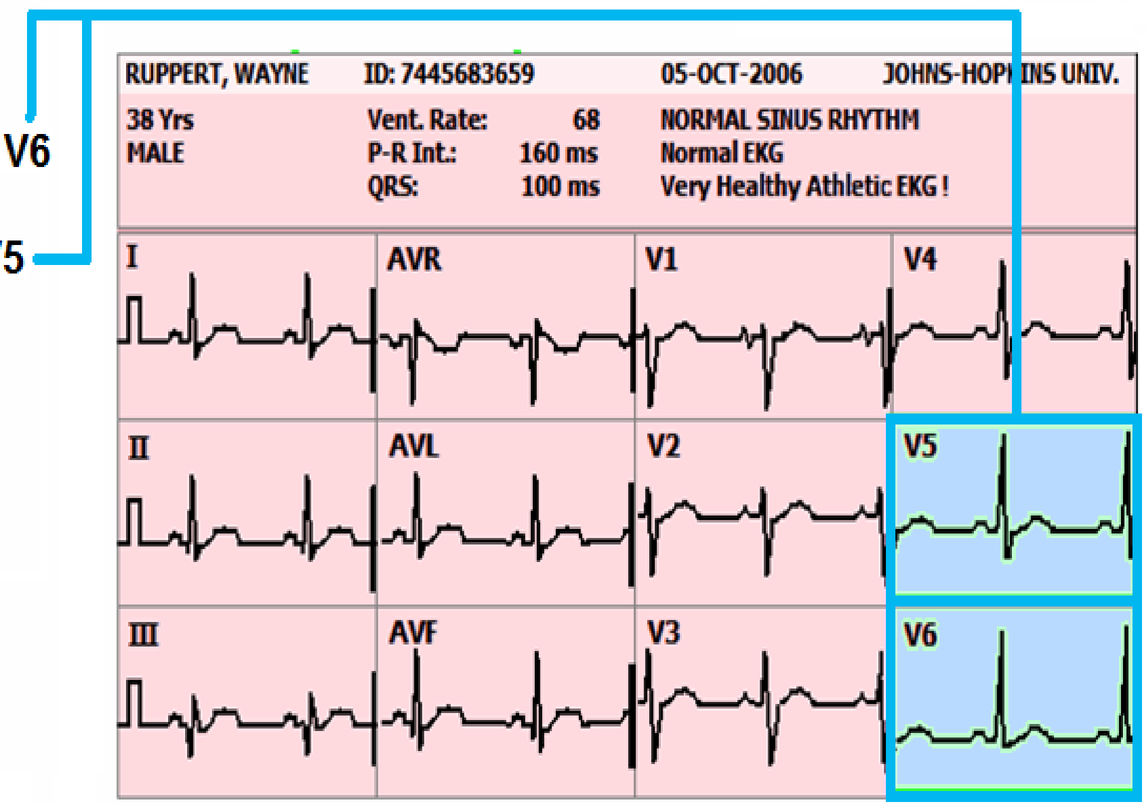
POSTERIOR WALL BLOOD SUPPLY



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

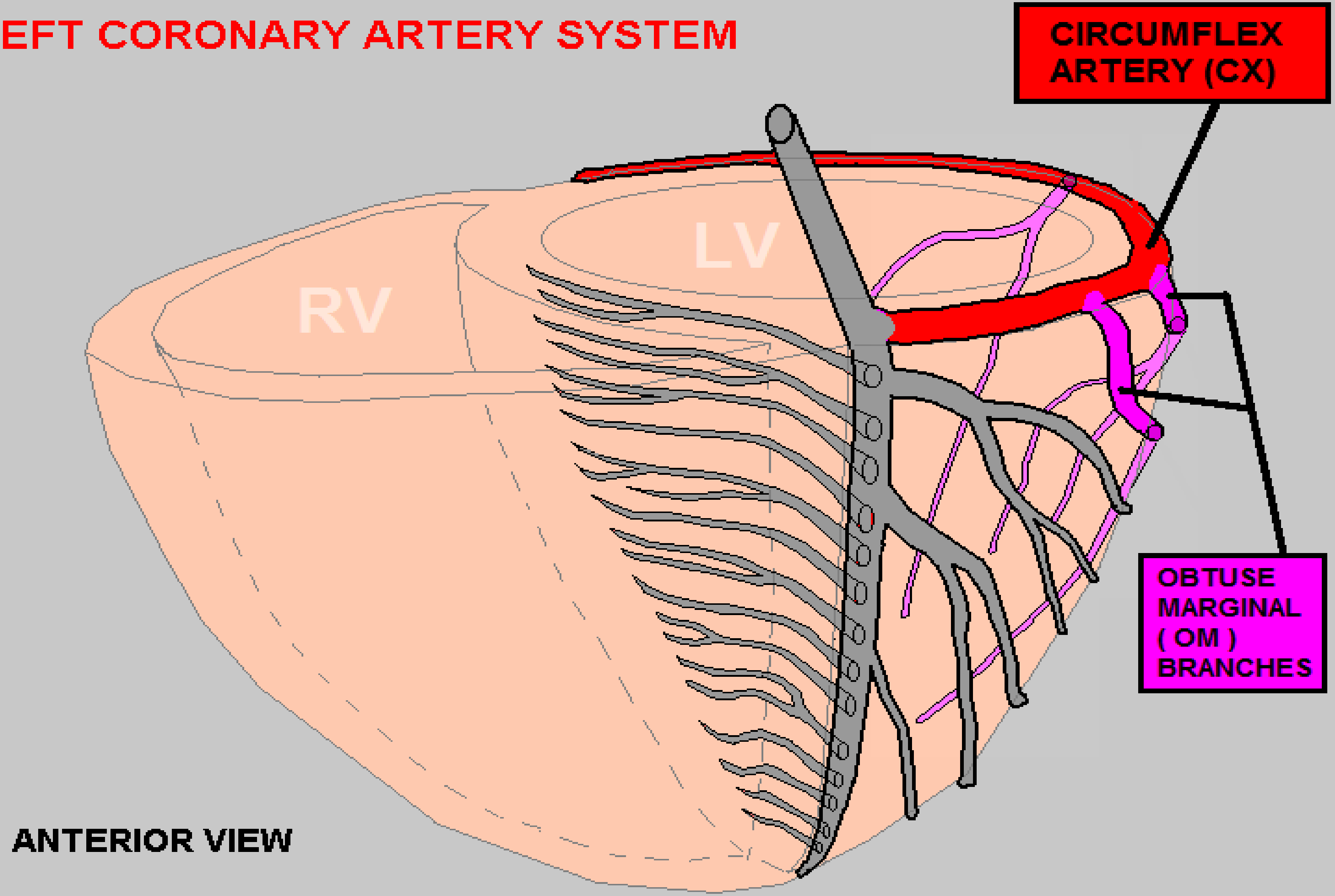
V5 - V6 VIEW THE LATERAL WALL
of the LEFT VENTRICLE





Which Coronary Artery typically Supplies the LATERAL WALL ?

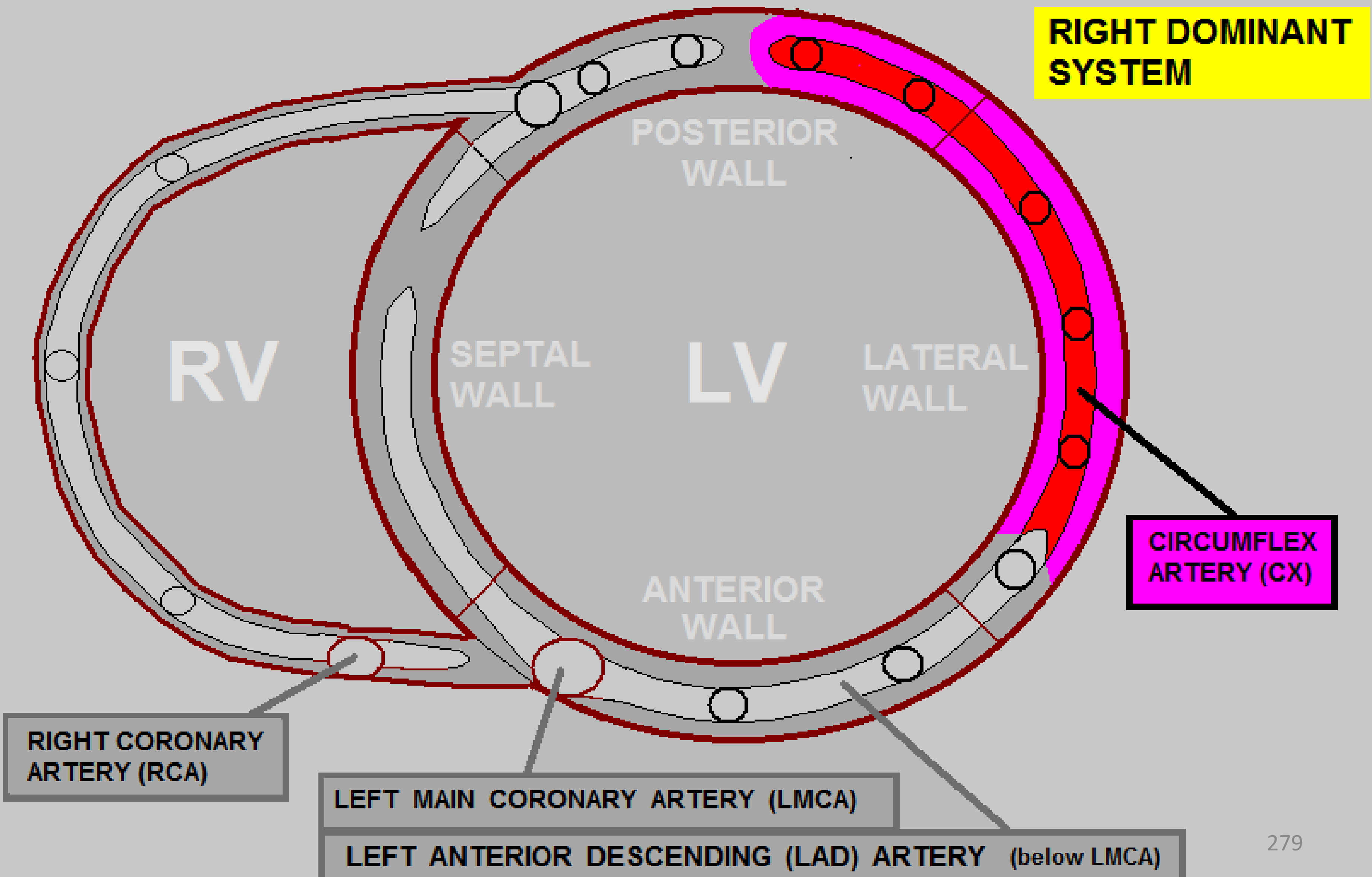
LEFT CORONARY ARTERY SYSTEM



cutaway view of the

CIRCUMFLEX ARTERY (CX) DISTRIBUTION

SUPPLIES 20 - 30 % of the LV MUSCLE MASS



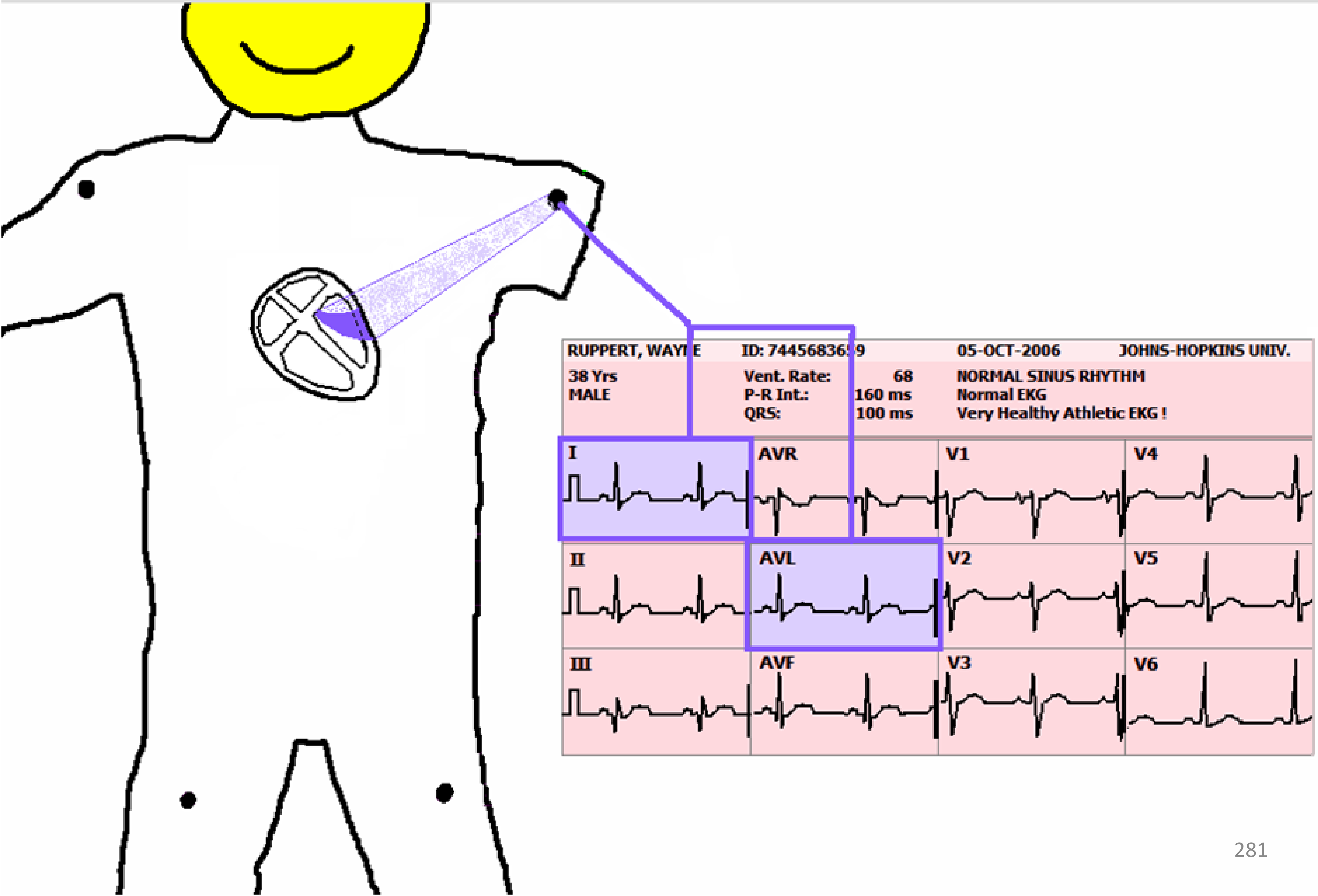
HELPFUL HINT... MEMORIZE THIS !

CIRCUMFLEX ARTERY (CX)

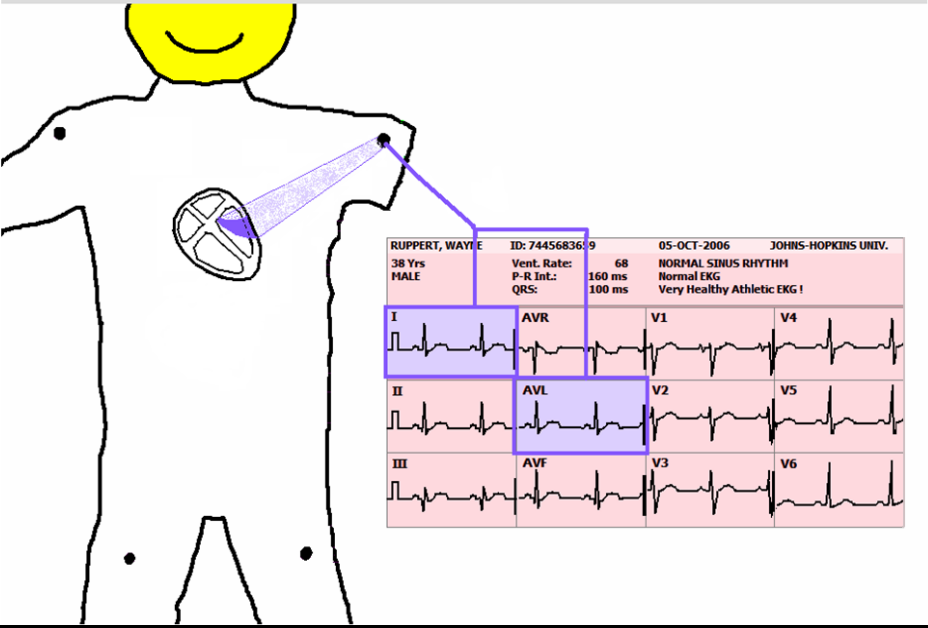
RIGHT DOMINANT SYSTEMS

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

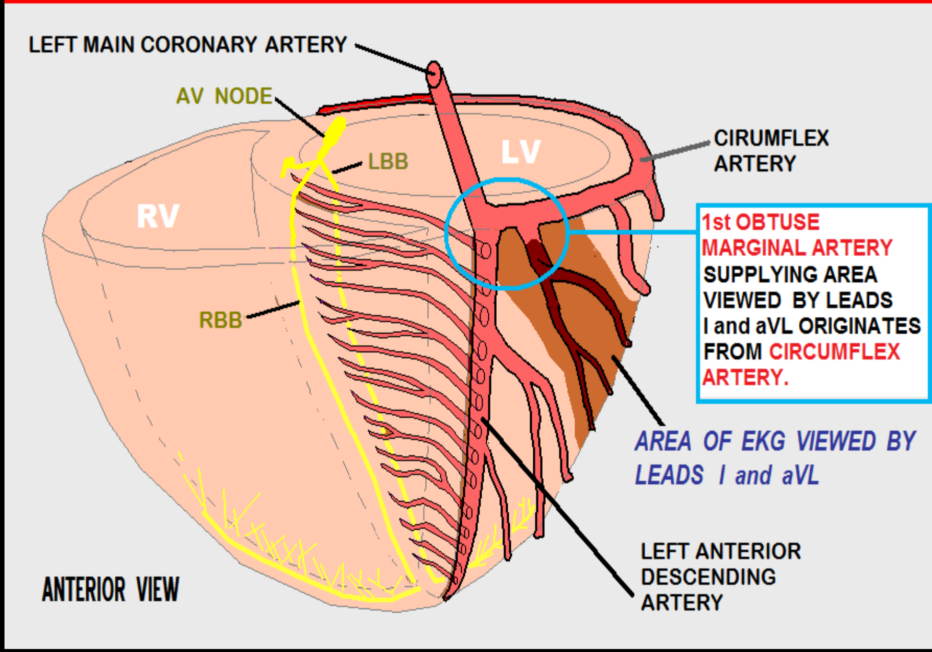
LEADS I and aVL VIEW the LATERAL - ANTERIOR WALL



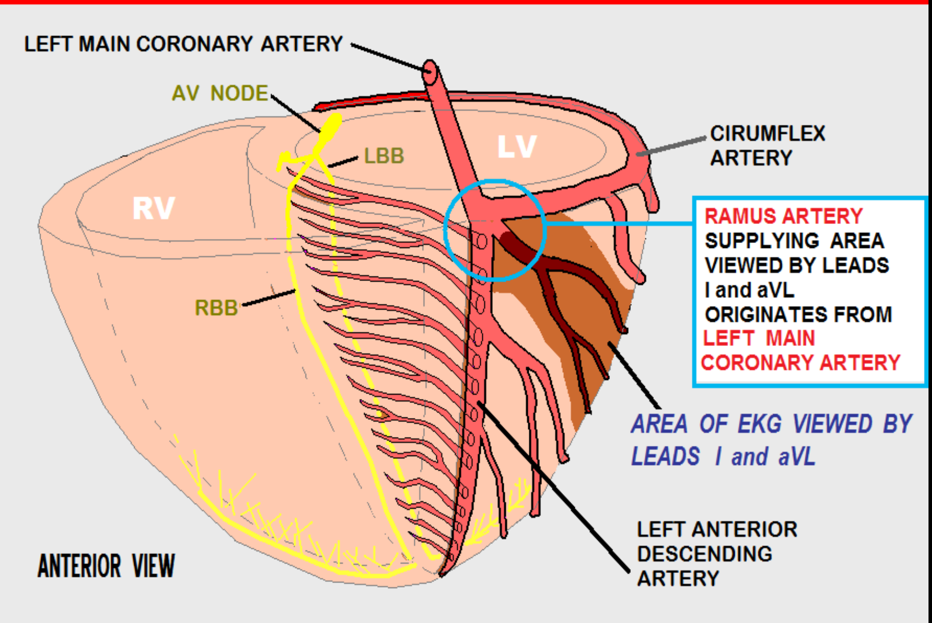
LEADS I and aVL VIEW the LATERAL - ANTERIOR WALL



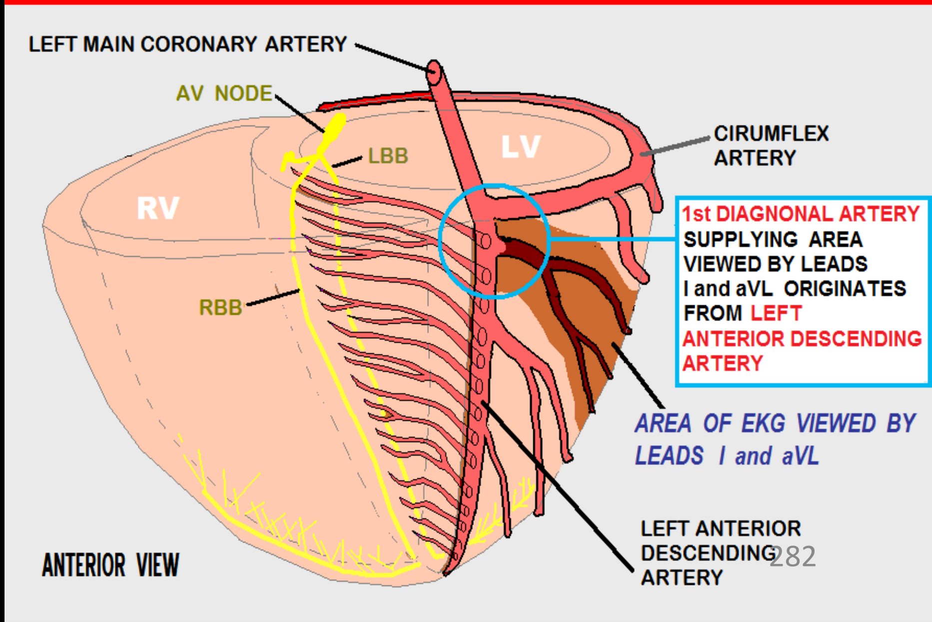
OCCLUSION of OBTUSE MARGINAL ARTERY

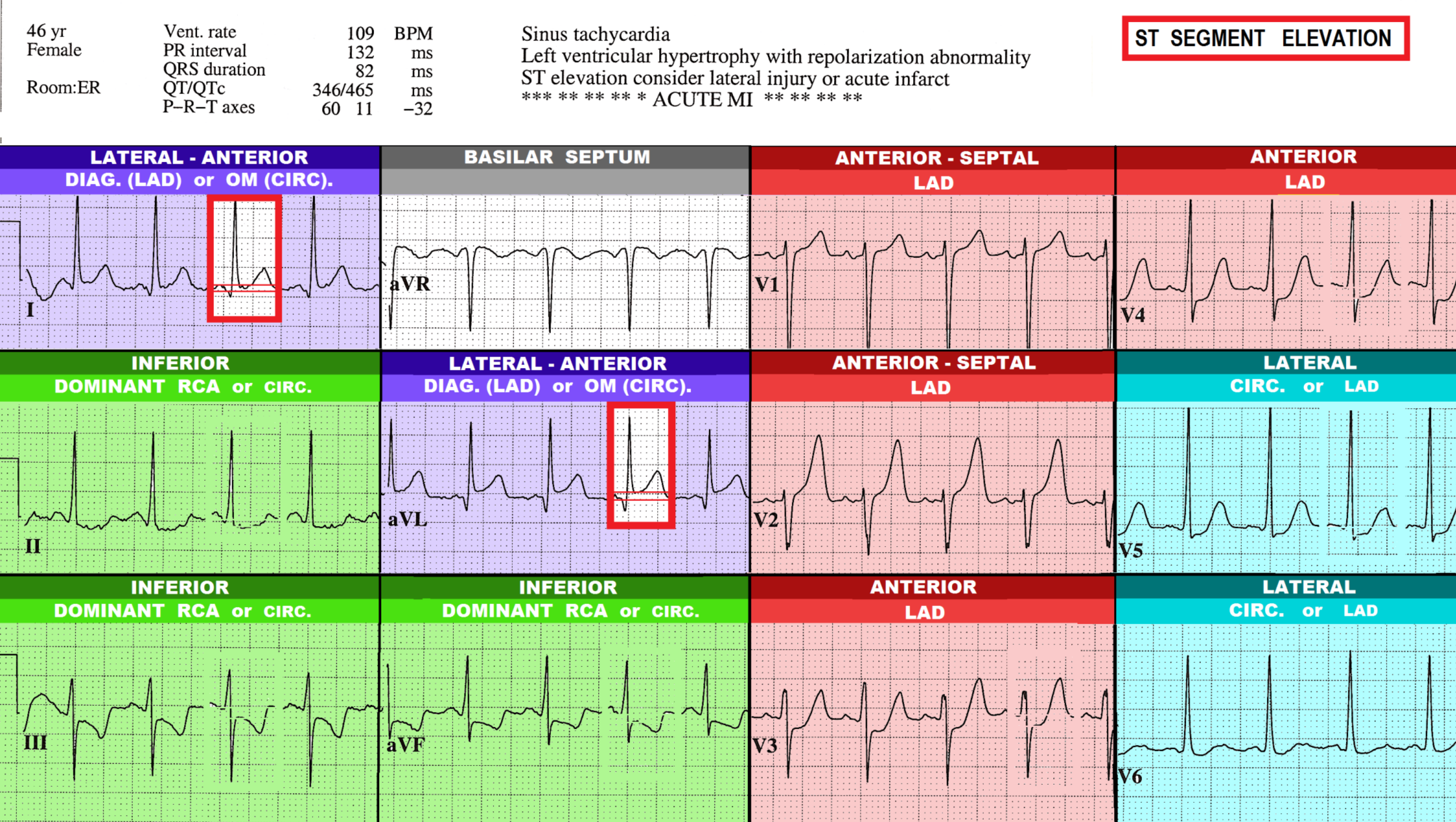


OCCLUSION of RAMUS ARTERY



OCCLUSION of DIAGONAL ARTERY

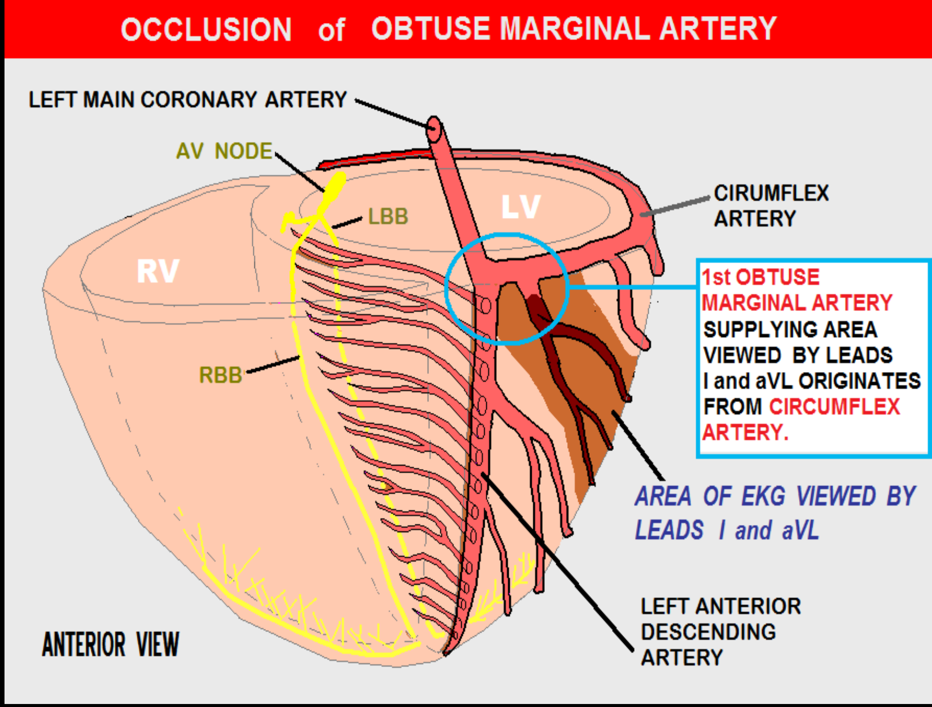


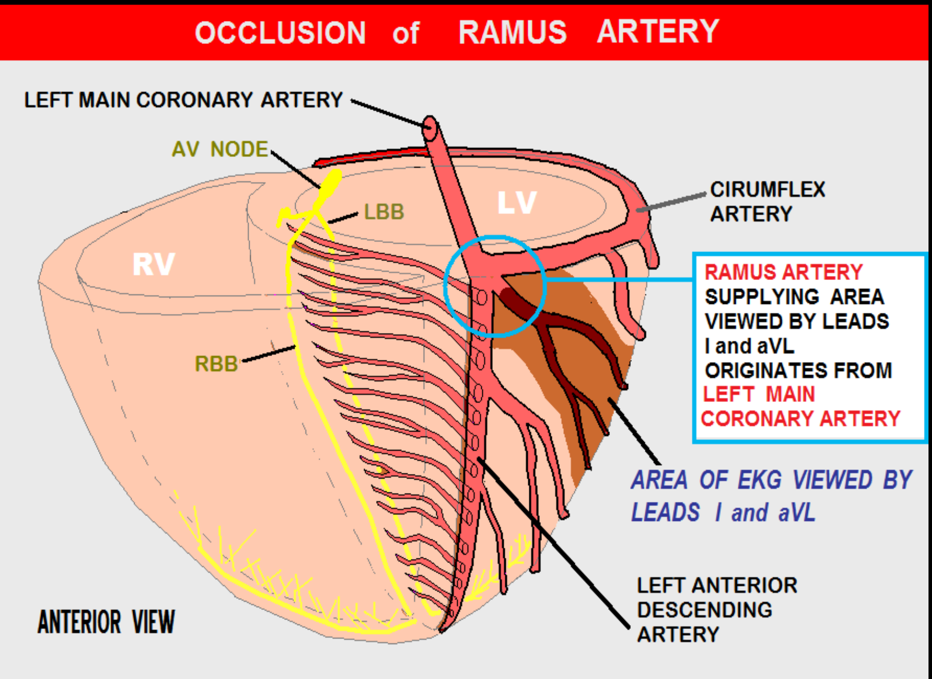


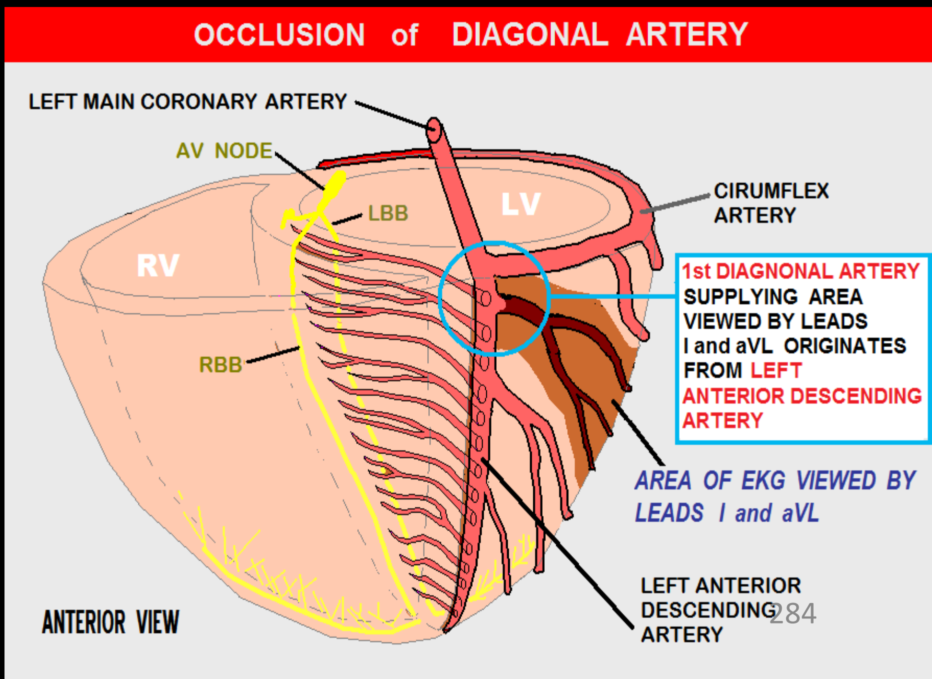
ST Segment elevation ONLY in Leads I and aVL

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

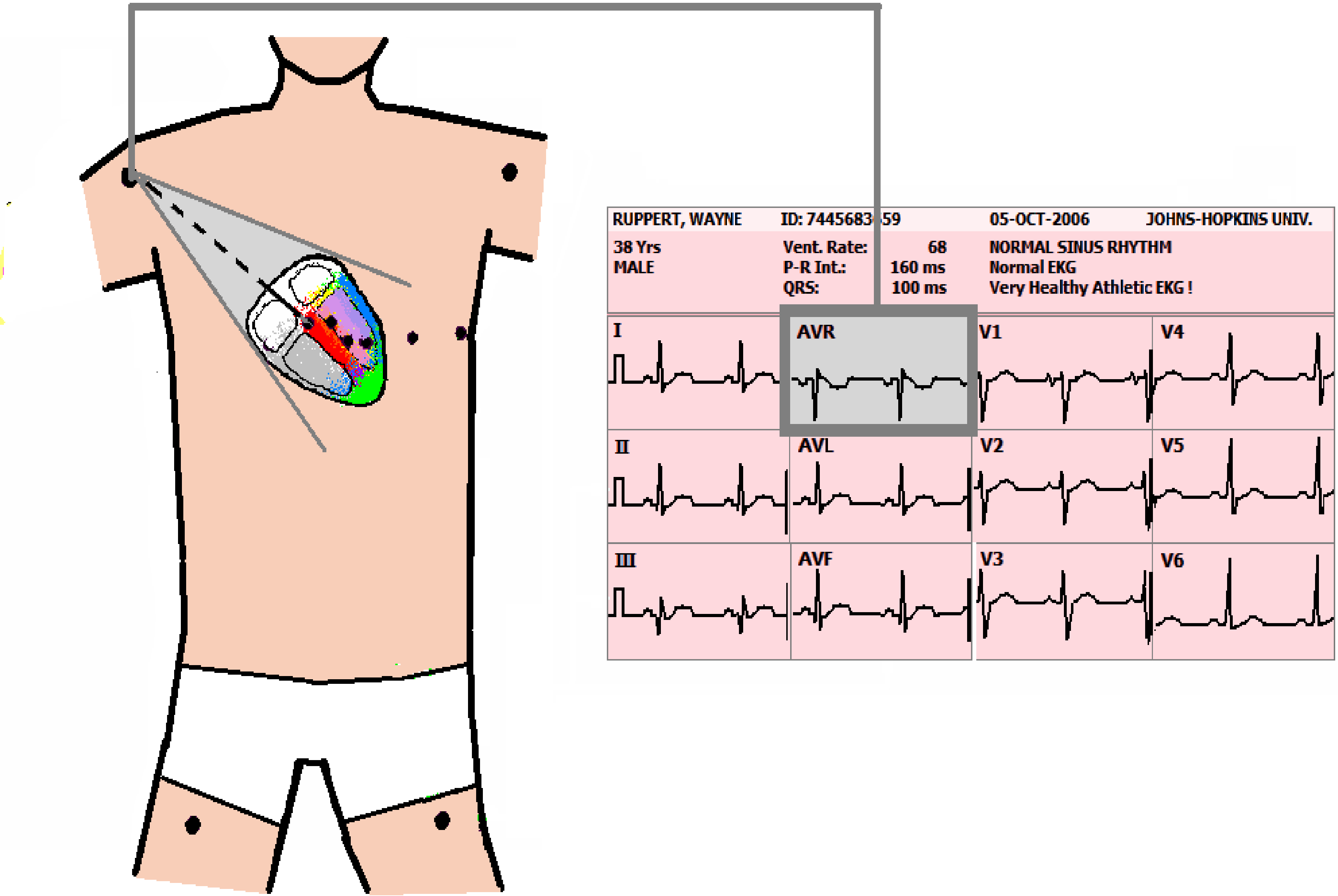
- RAMUS BRANCH
- 1st DIAGONAL off of LAD
- 1st OBTUSE MARGINAL off of CIRCUMFLEX

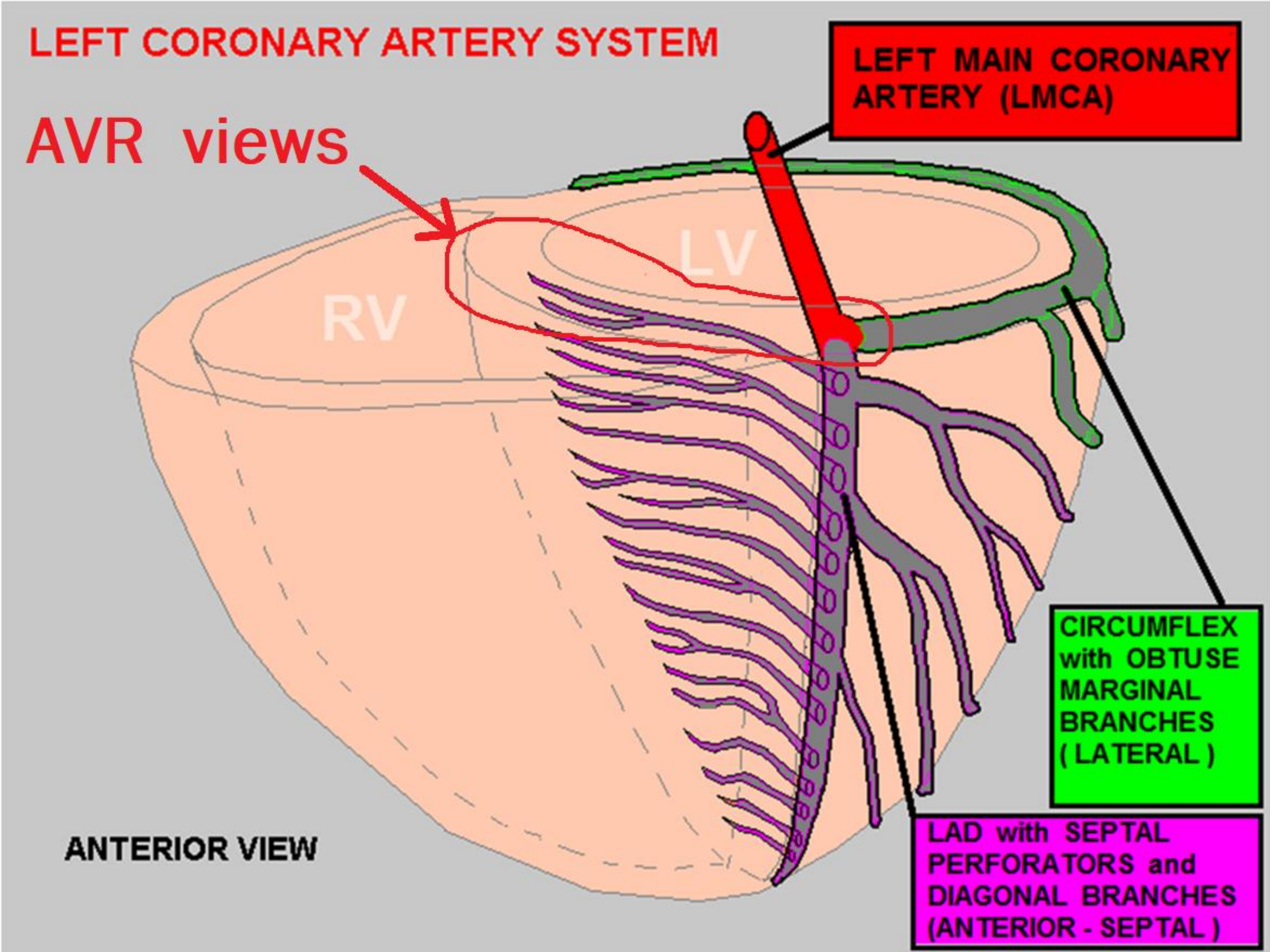






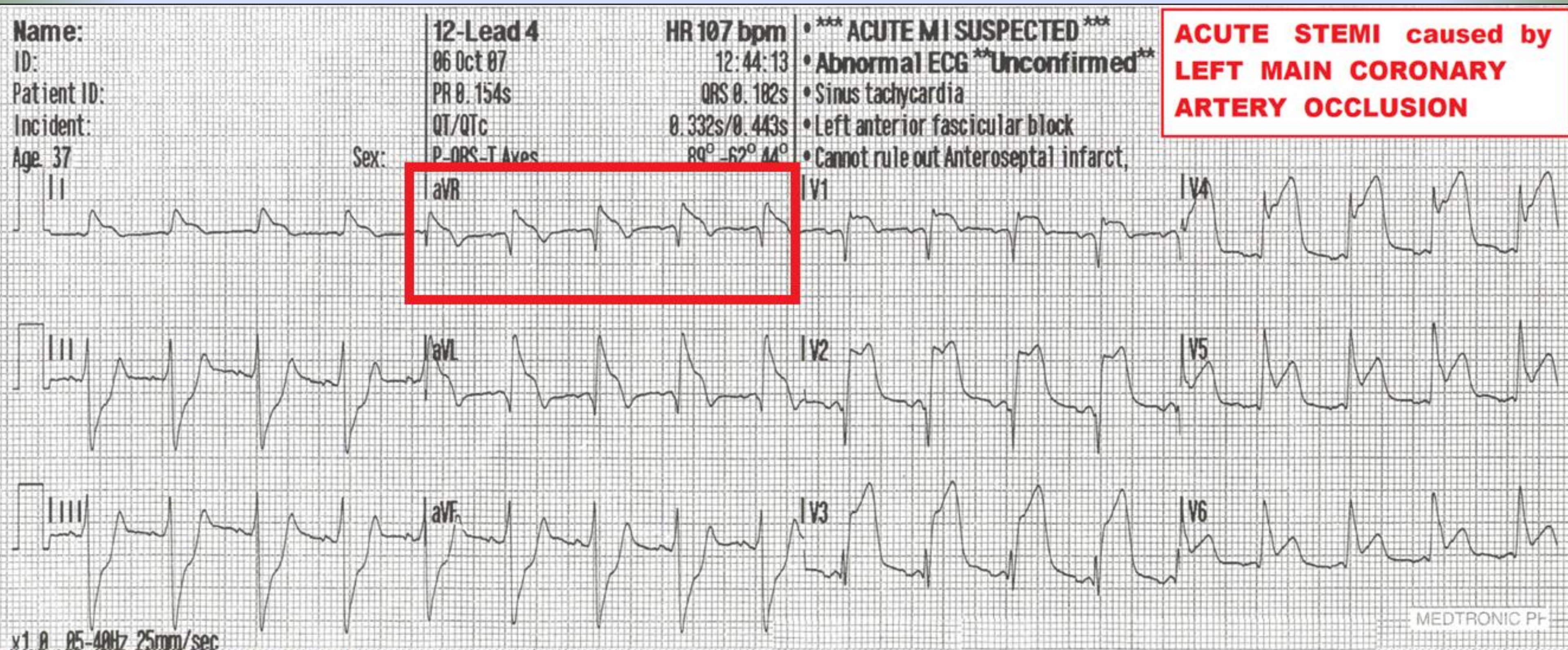
Lead AVR Views the BASILAR SEPTUM (region of the Bundle of His)




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.

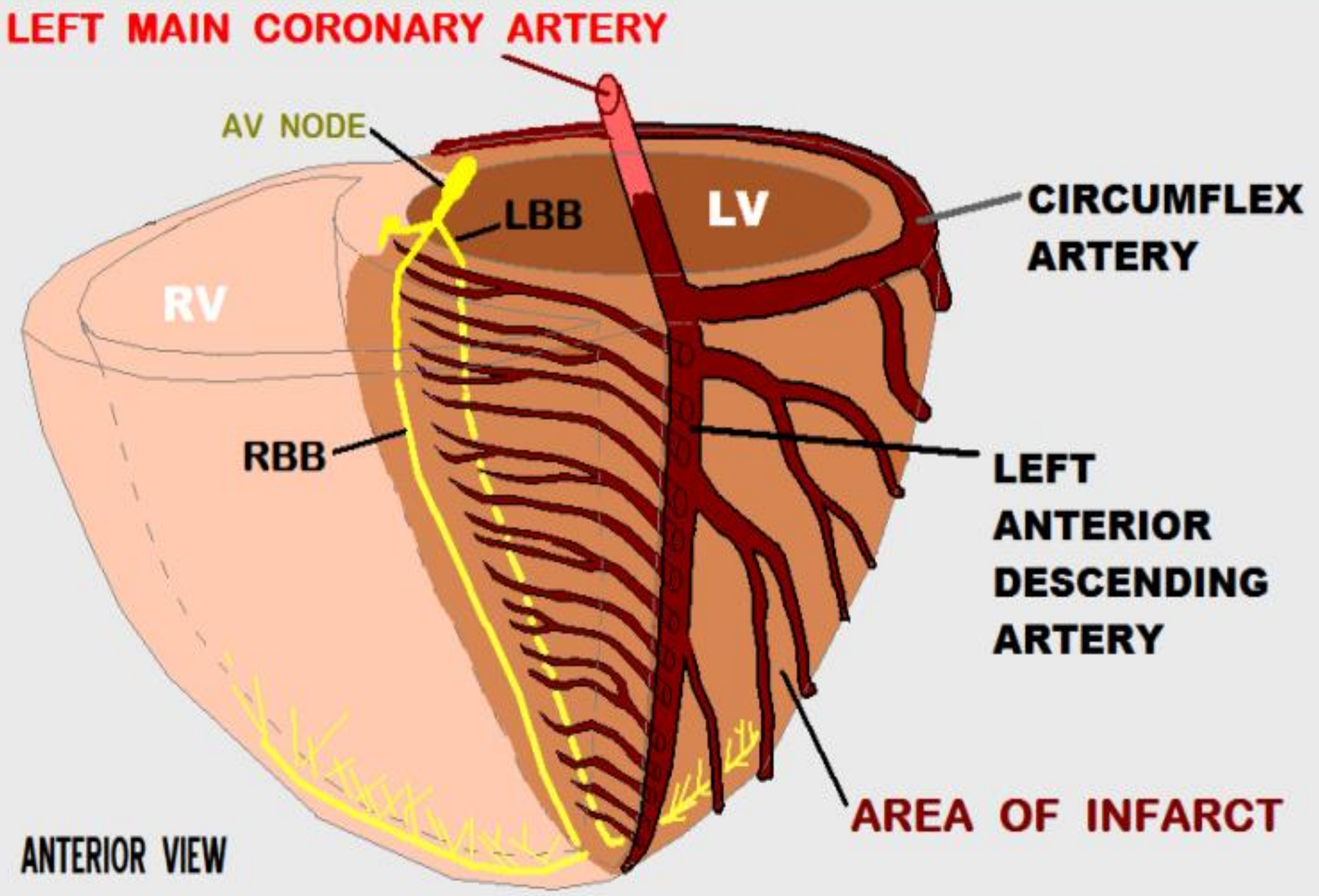


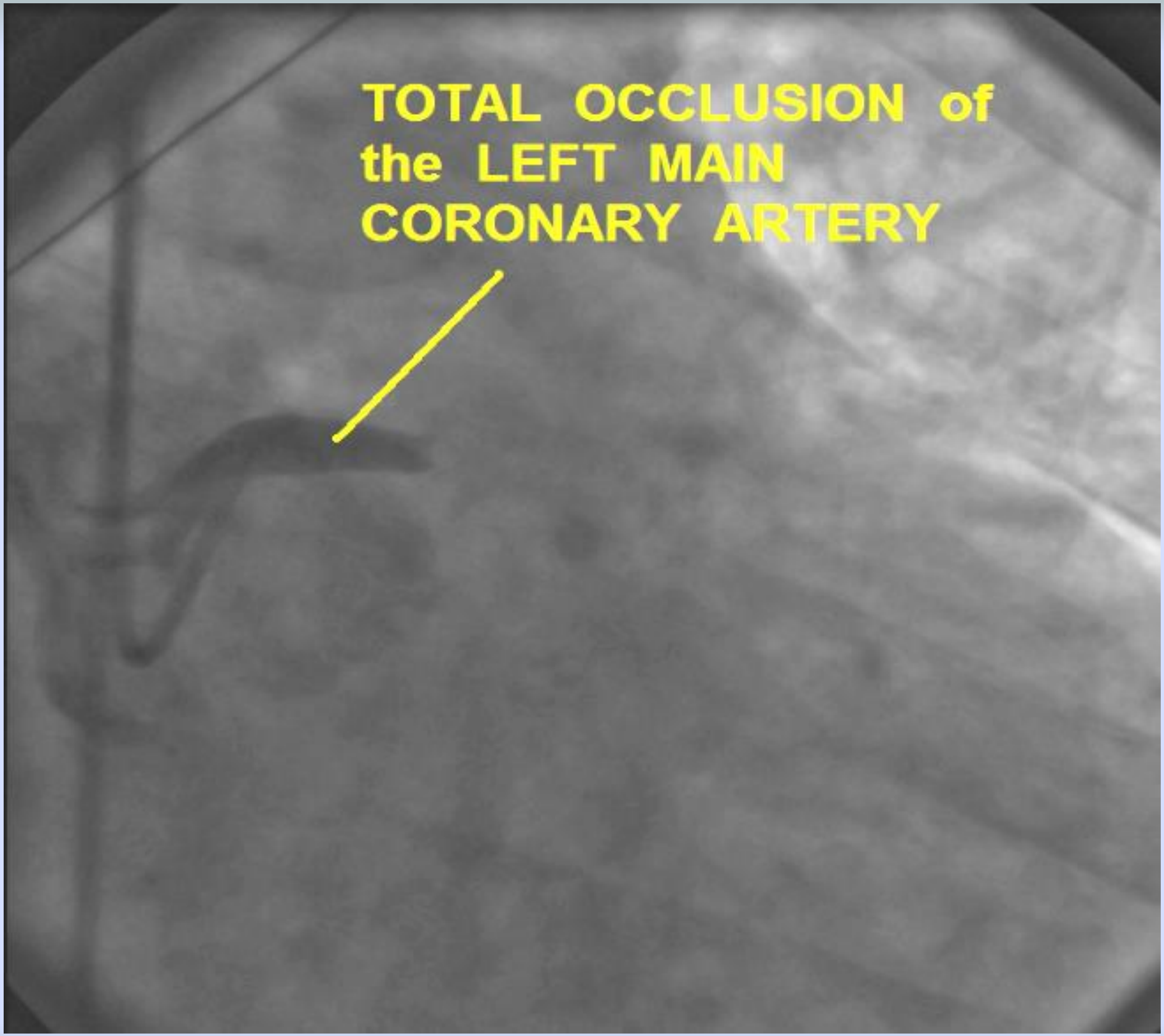
ACUTE STEMI caused by LEFT MAIN CORONARY ARTERY OCCLUSION

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

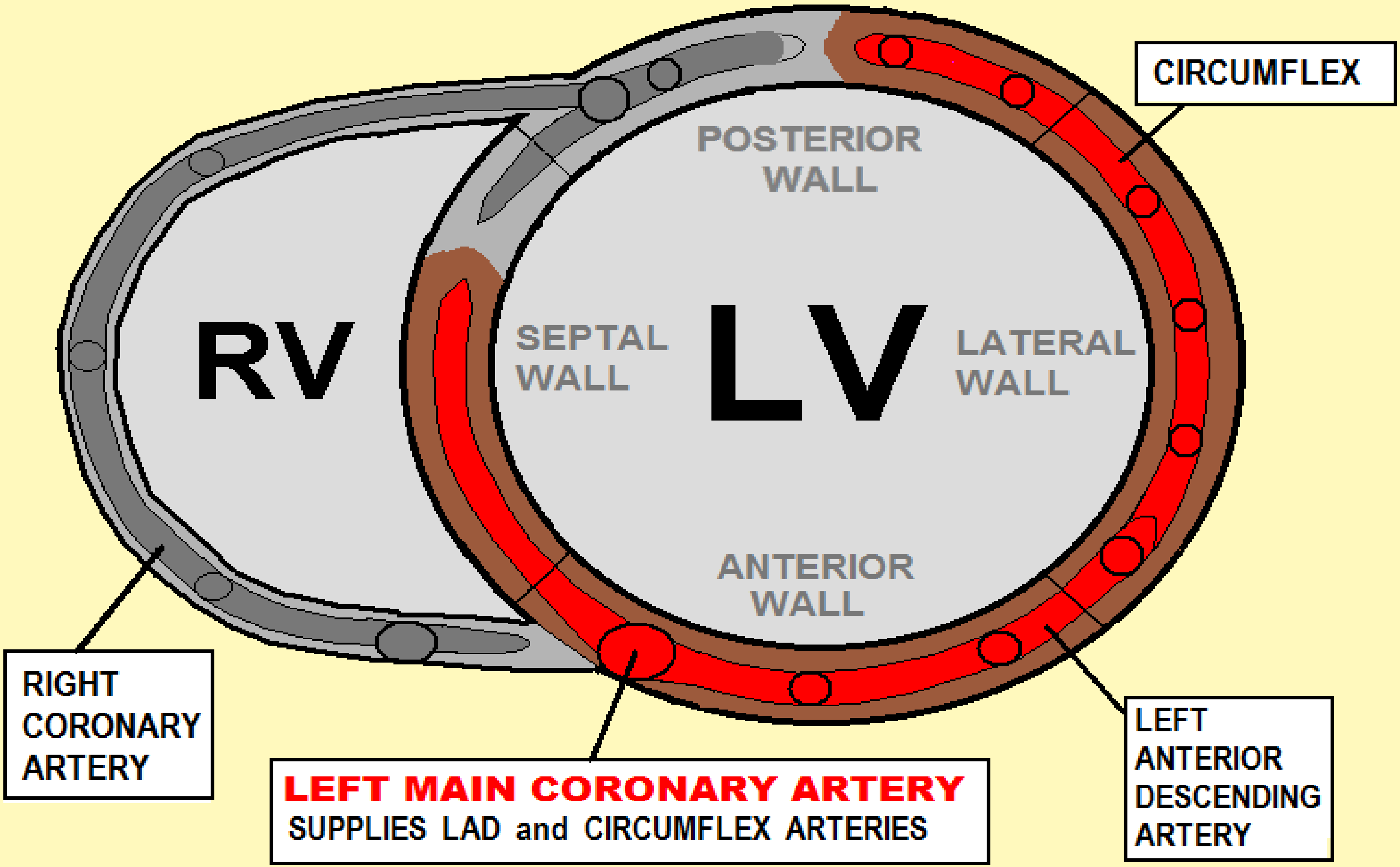
OCCLUSION of the LEFT MAIN CORONARY ARTERY




TOTAL OCCLUSION of
the LEFT MAIN
CORONARY ARTERY

The LEFT MAIN CORONARY ARTERY

SUPPLIES 75 - 100 % of the LEFT VENTRICULAR MUSCLE MASS



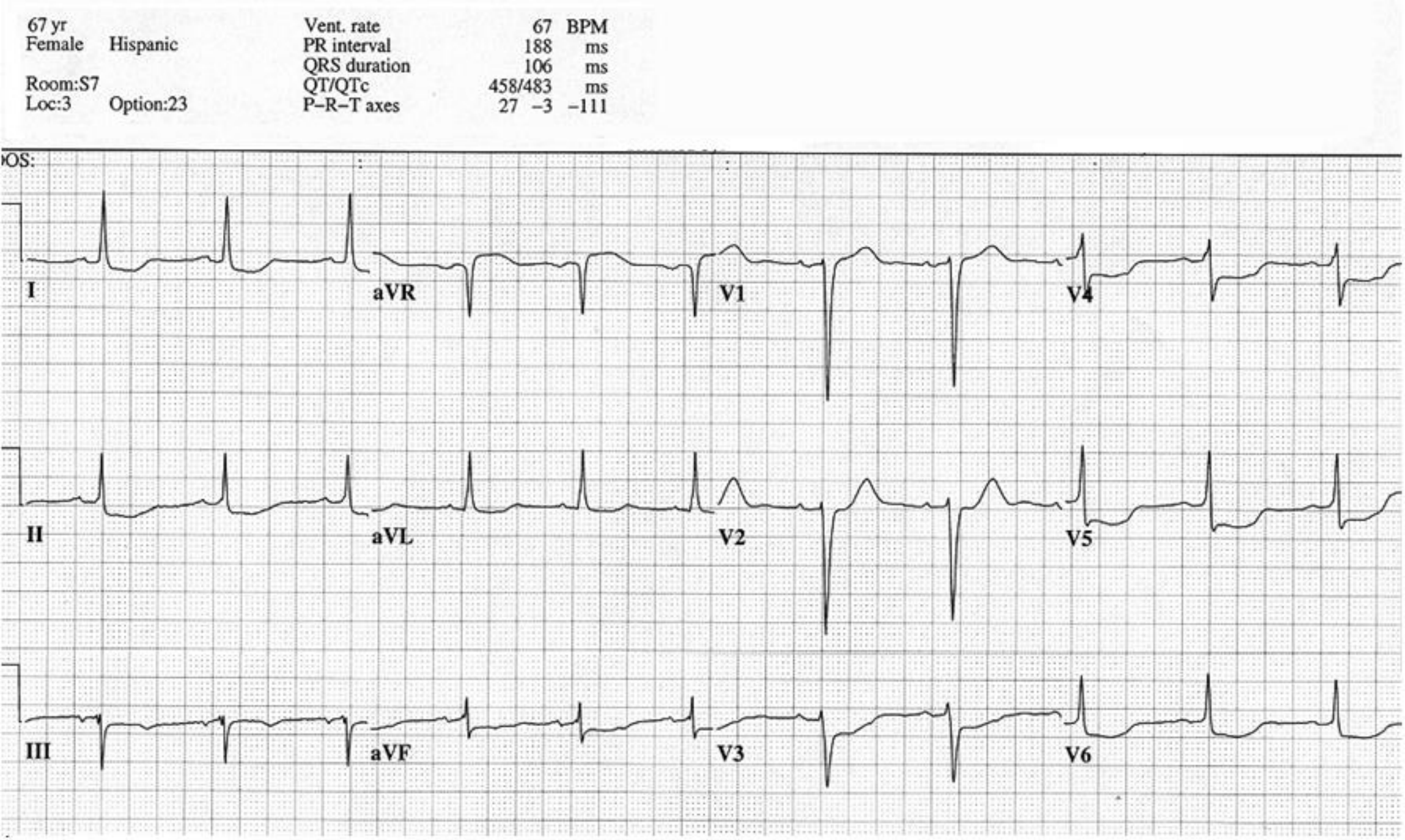
When LEAD AVR shows ST Elevation:

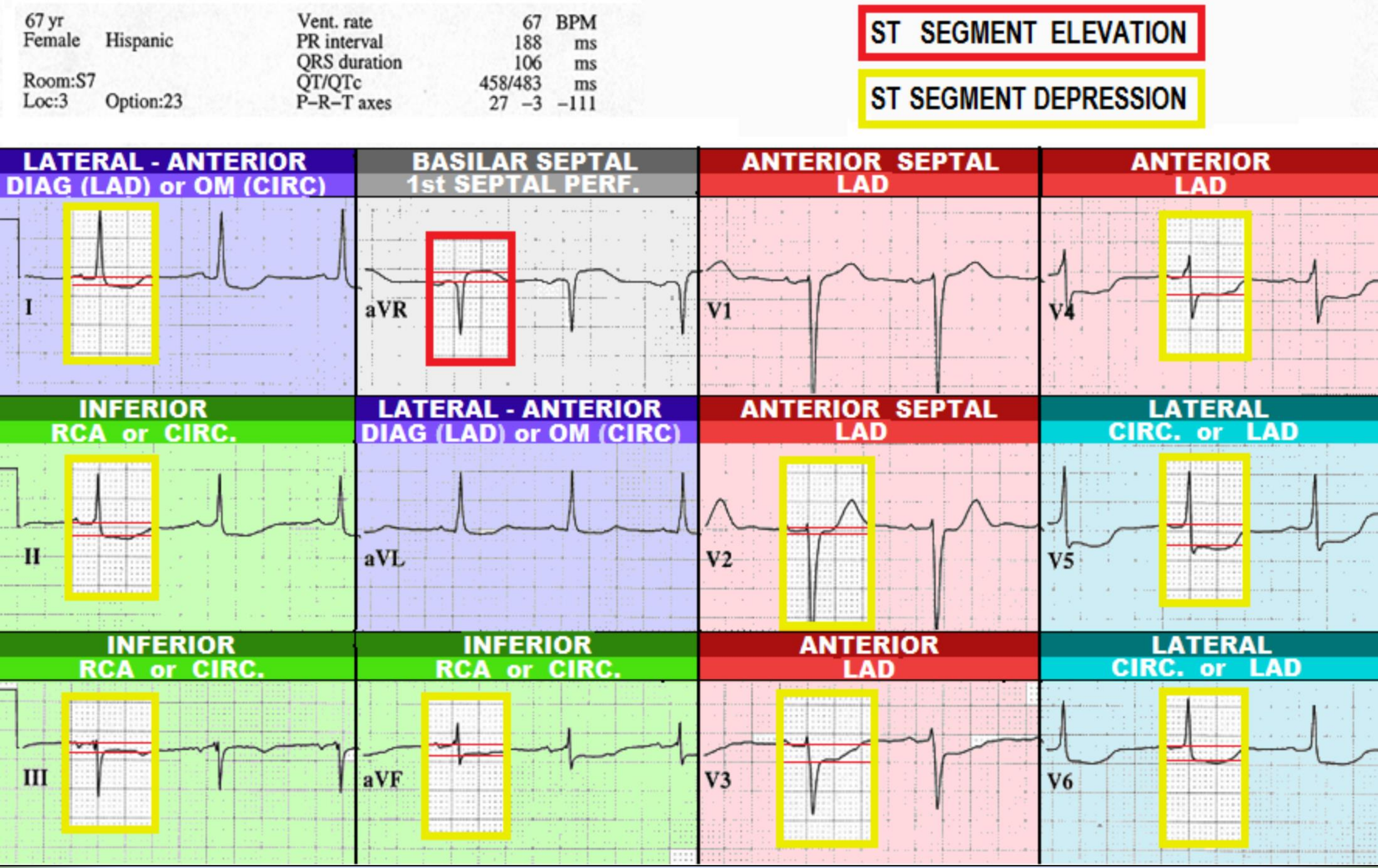
- **STEMI:** consider occlusion of the Left Main Coronary Artery.

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

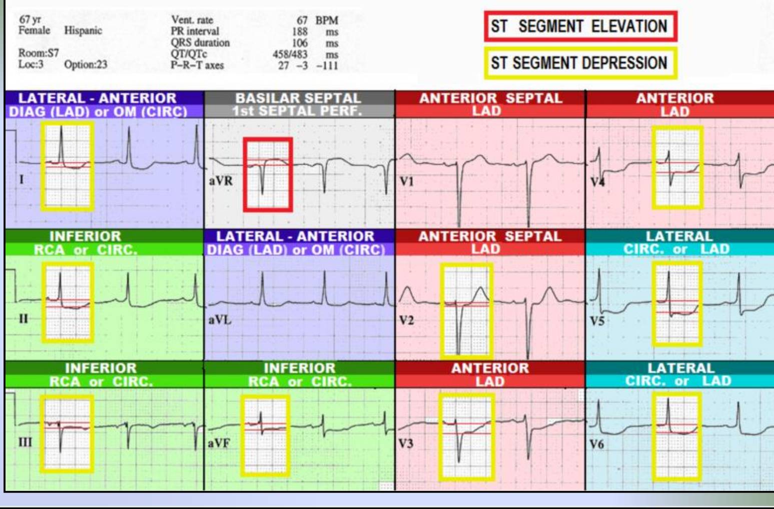


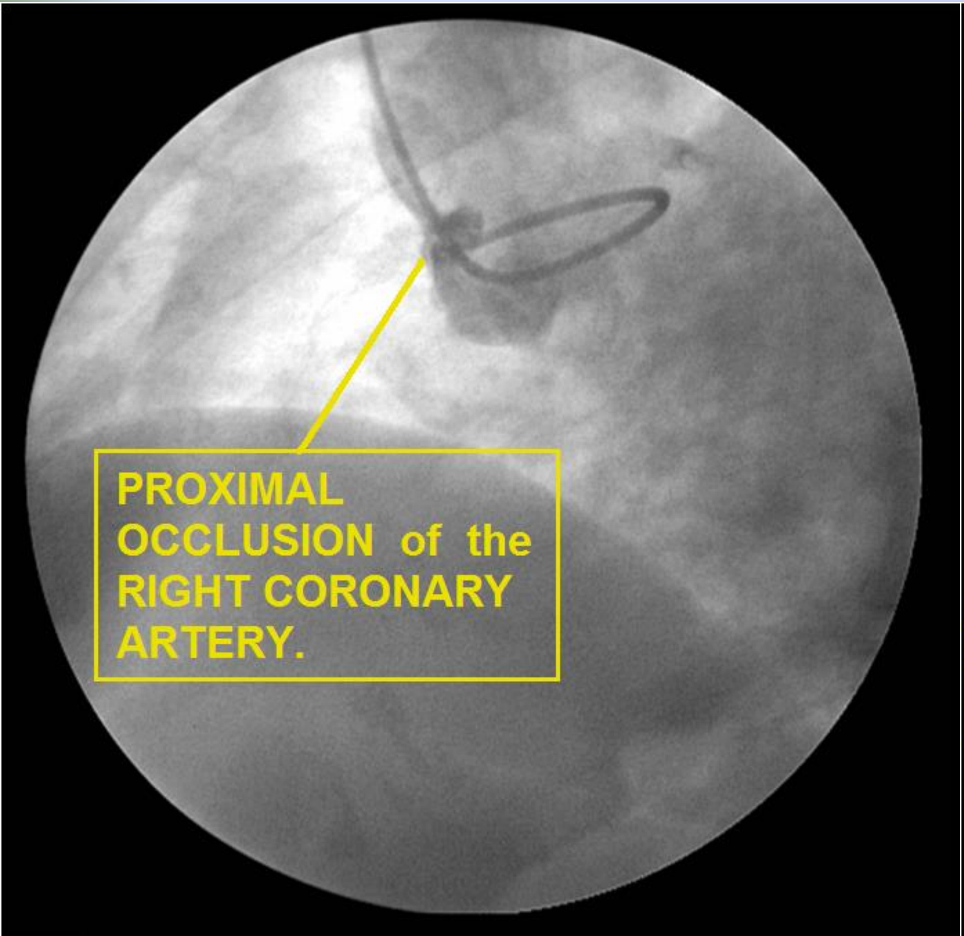
67 yr
Female Hispanic
Room:S7
Loc:3 Option:23
Vent. rate 67 BPM
PR interval 188 ms
QRS duration 106 ms
QT/QTc 458/483 ms
P–R–T axes 27 –3 –111
ST SEGMENT ELEVATION
ST SEGMENT DEPRESSION
LATERAL - ANTERIOR
DIAG (LAD) or OM (CIRC)
I
BASILAR SEPTAL
1st SEPTAL PERF.
aVR
ANTERIOR SEPTAL
LAD
V1
ANTERIOR
LAD
V4
INFERIOR
RCA or CIRC.
II
LATERAL - ANTERIOR
DIAG (LAD) or OM (CIRC)
aVL
ANTERIOR SEPTAL
LAD
V2
LATERAL
CIRC. or LAD
V5
INFERIOR
RCA or CIRC.
III
INFERIOR
RCA or CIRC.
aVF
ANTERIOR
LAD
V3
LATERAL
CIRC. or LAD
V6

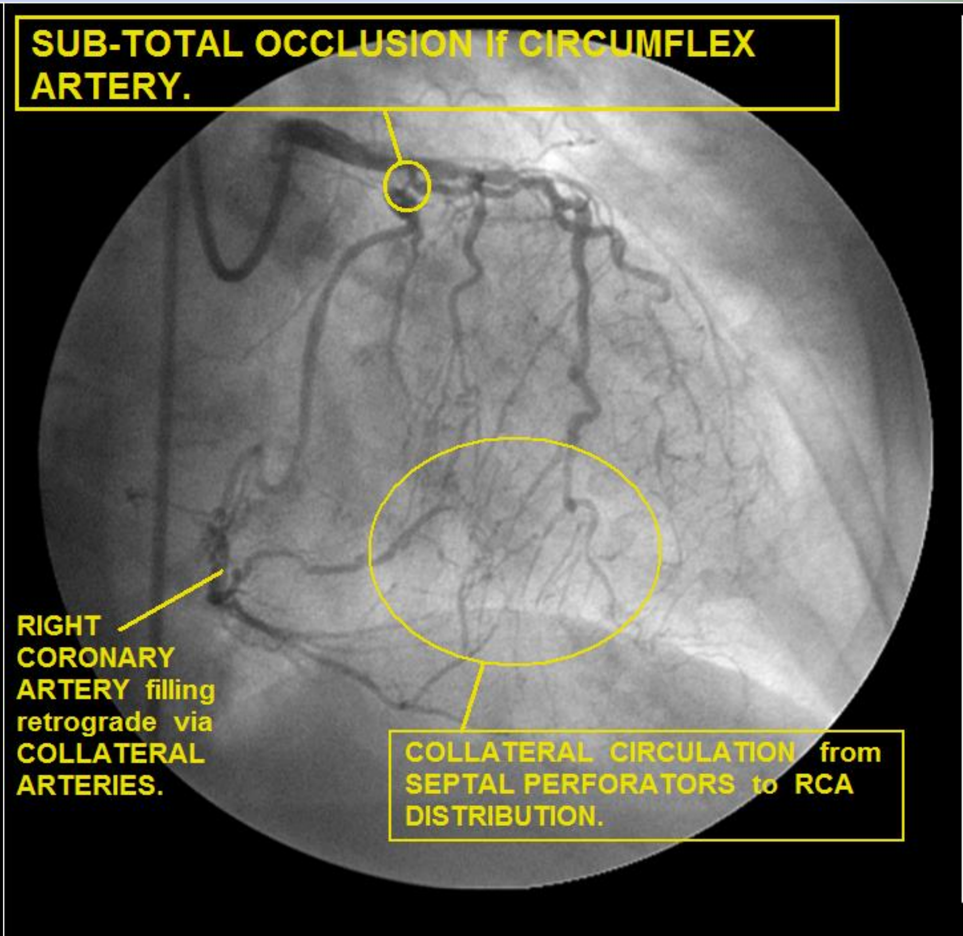
GLOBAL ISCHEMIA

- **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*



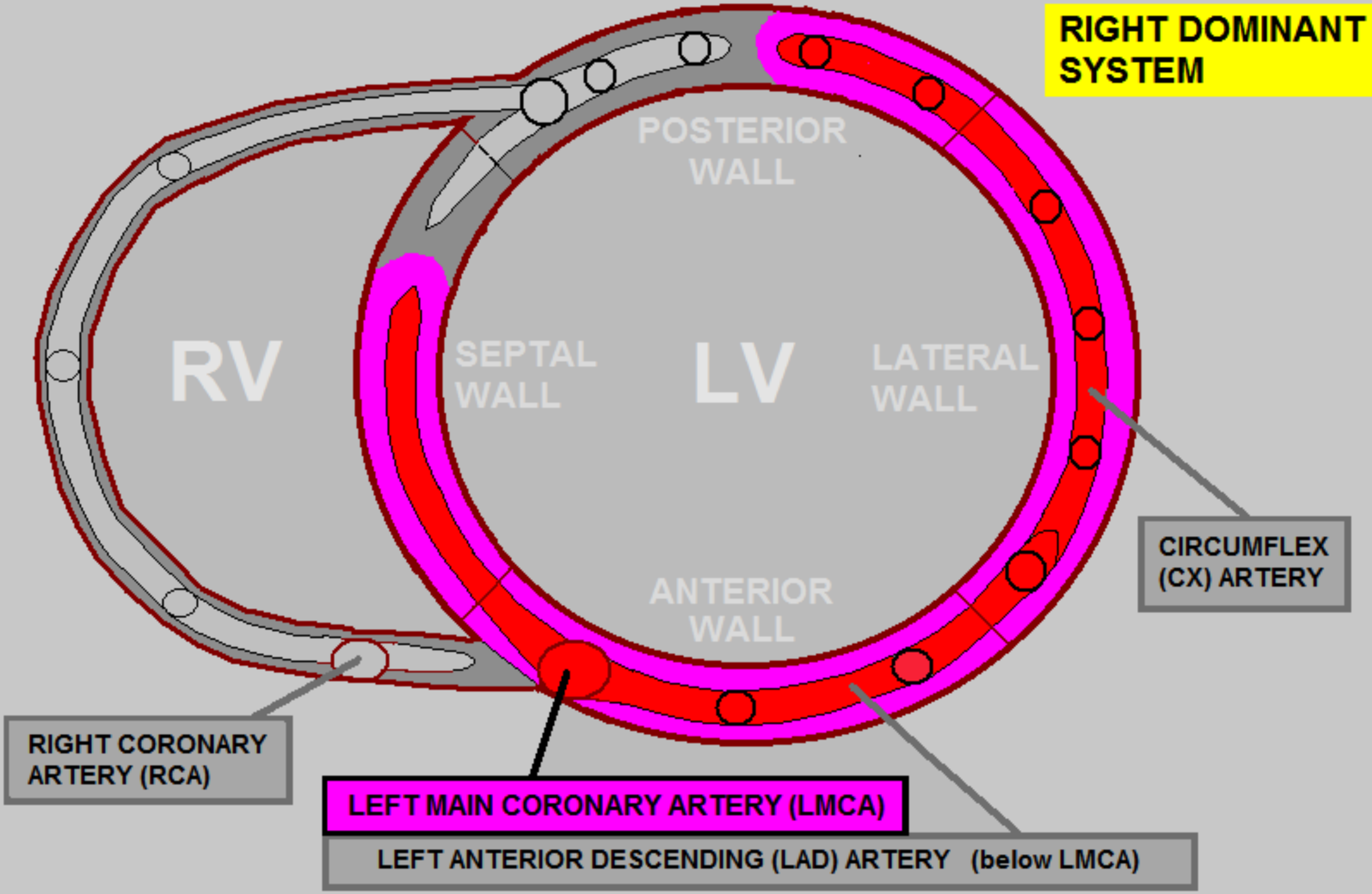




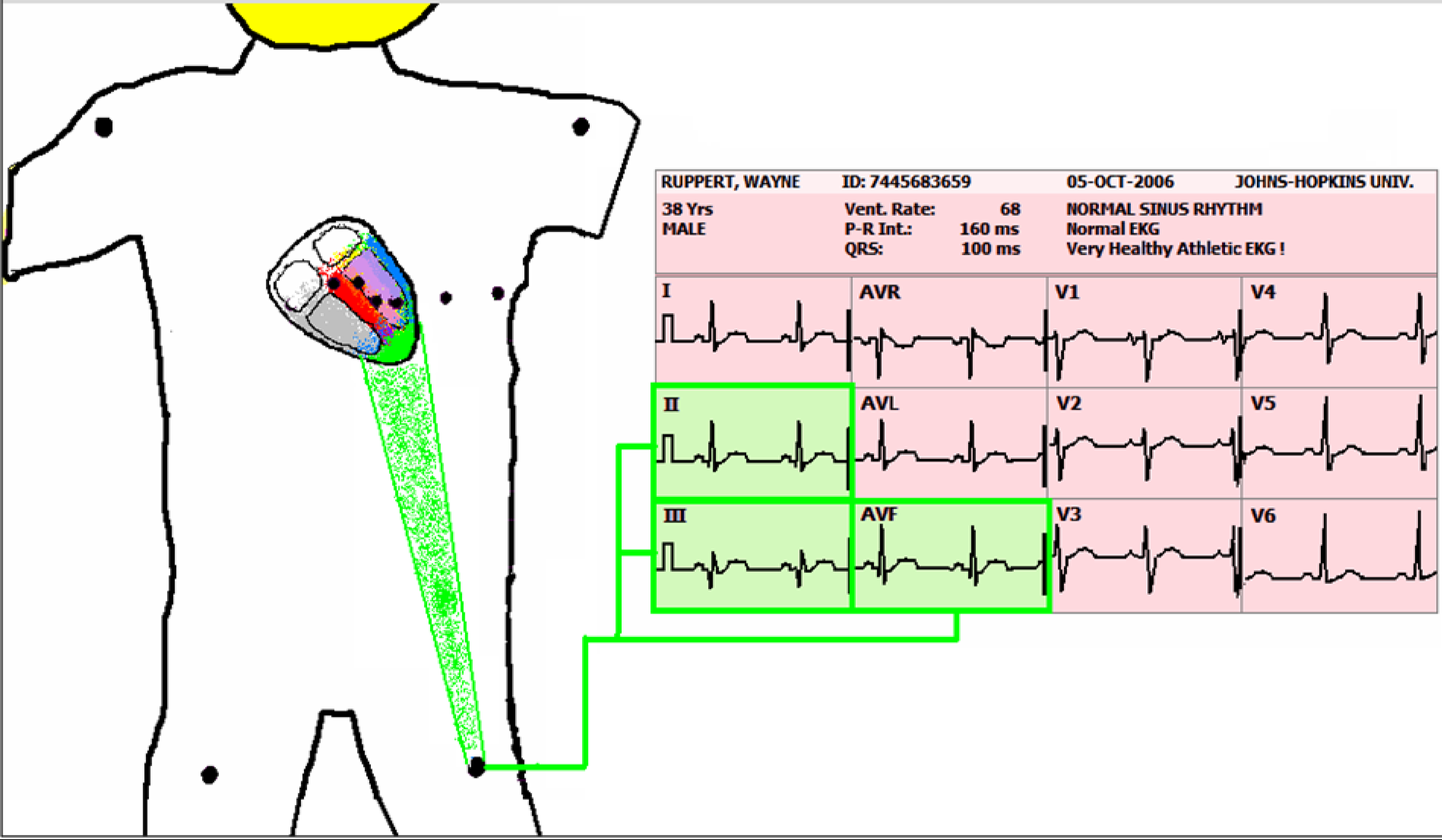
cutaway view of the

LEFT MAIN CORONARY ARTERY (LMCA)

☛ **SUPPLIES APPROXIMATELY 75% OF LV MUSCLE MASS**

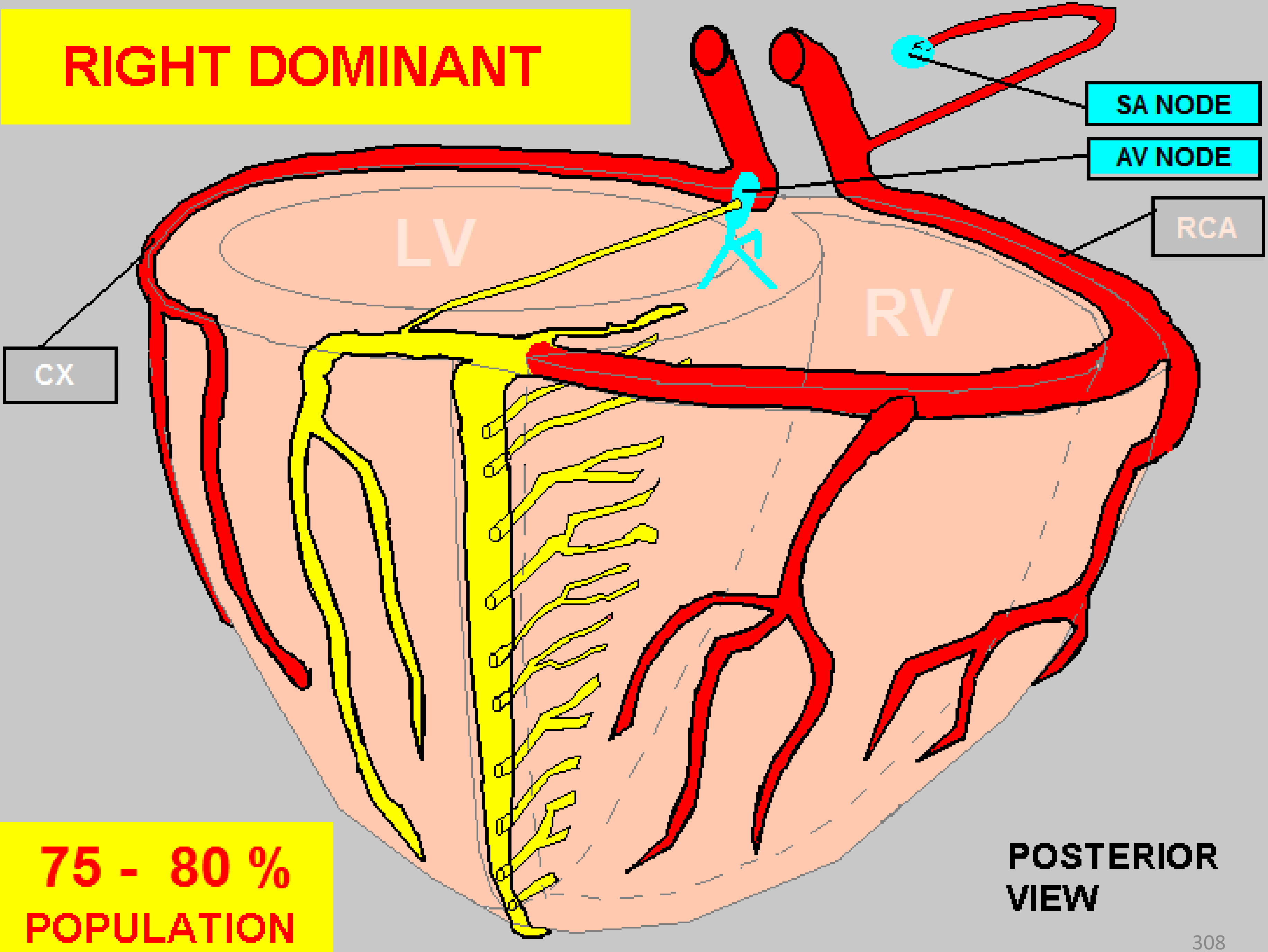


LEADS II, III, and aVF VIEW
INFERIOR WALL of the LEFT VENTRICLE



Which CORONARY ARTERY usually supplies the INFERIOR WALL ?

RIGHT DOMINANT



75 - 80 %
POPULATION

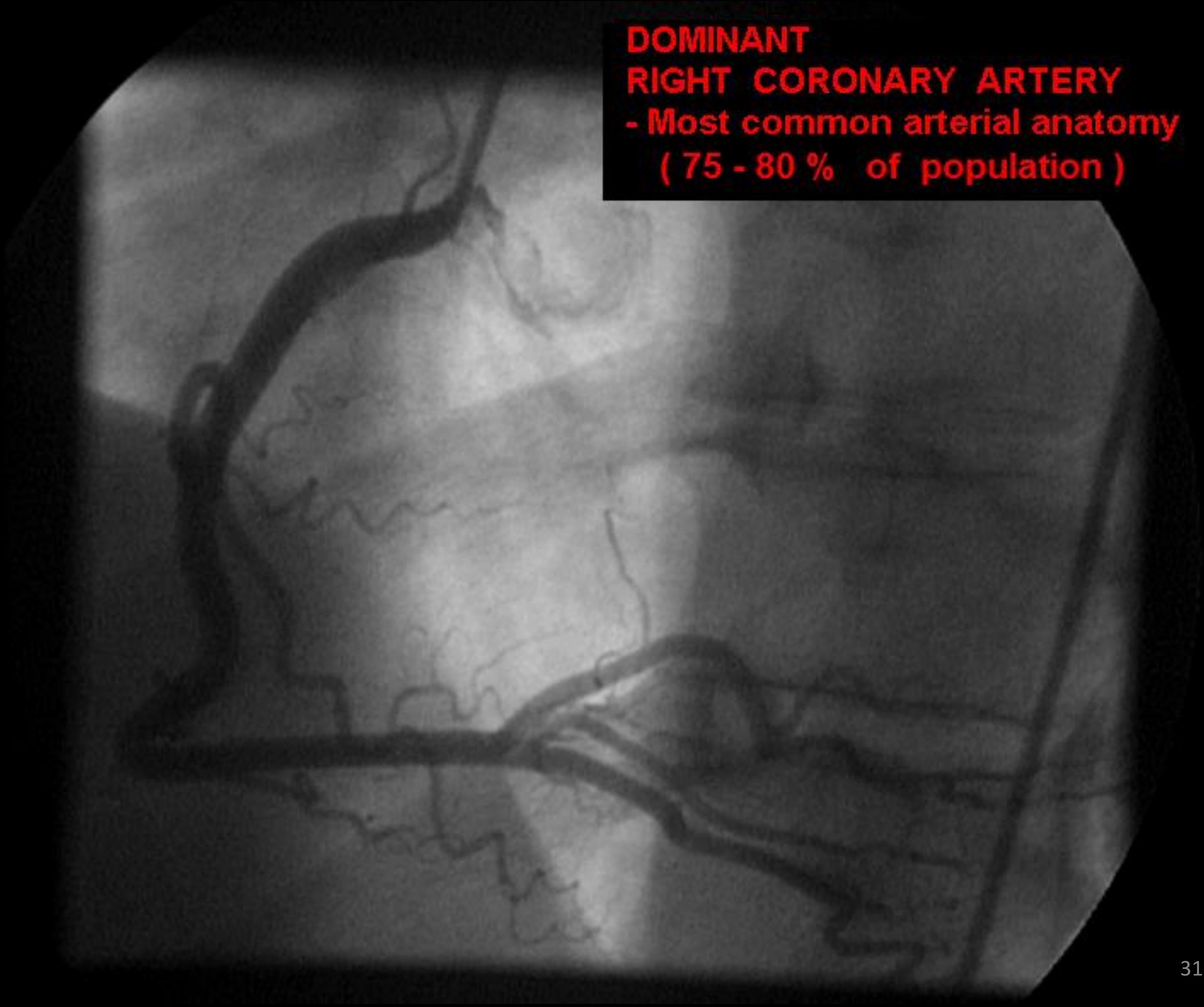
POSTERIOR VIEW

HELPFUL HINT . . . MEMORIZE THIS !

RIGHT CORONARY ARTERY (RCA)

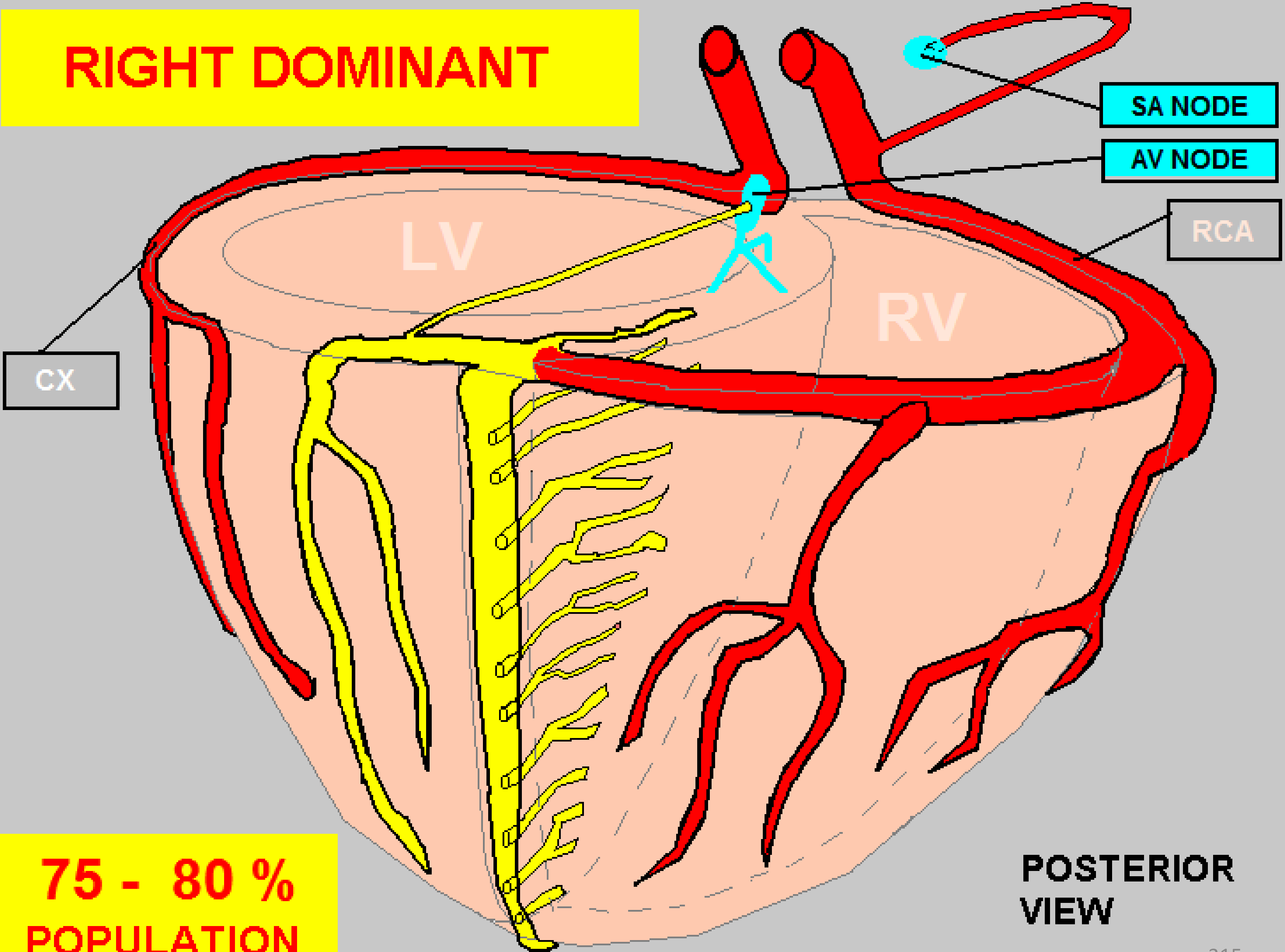
RIGHT DOMINANT SYSTEMS

- 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% ??

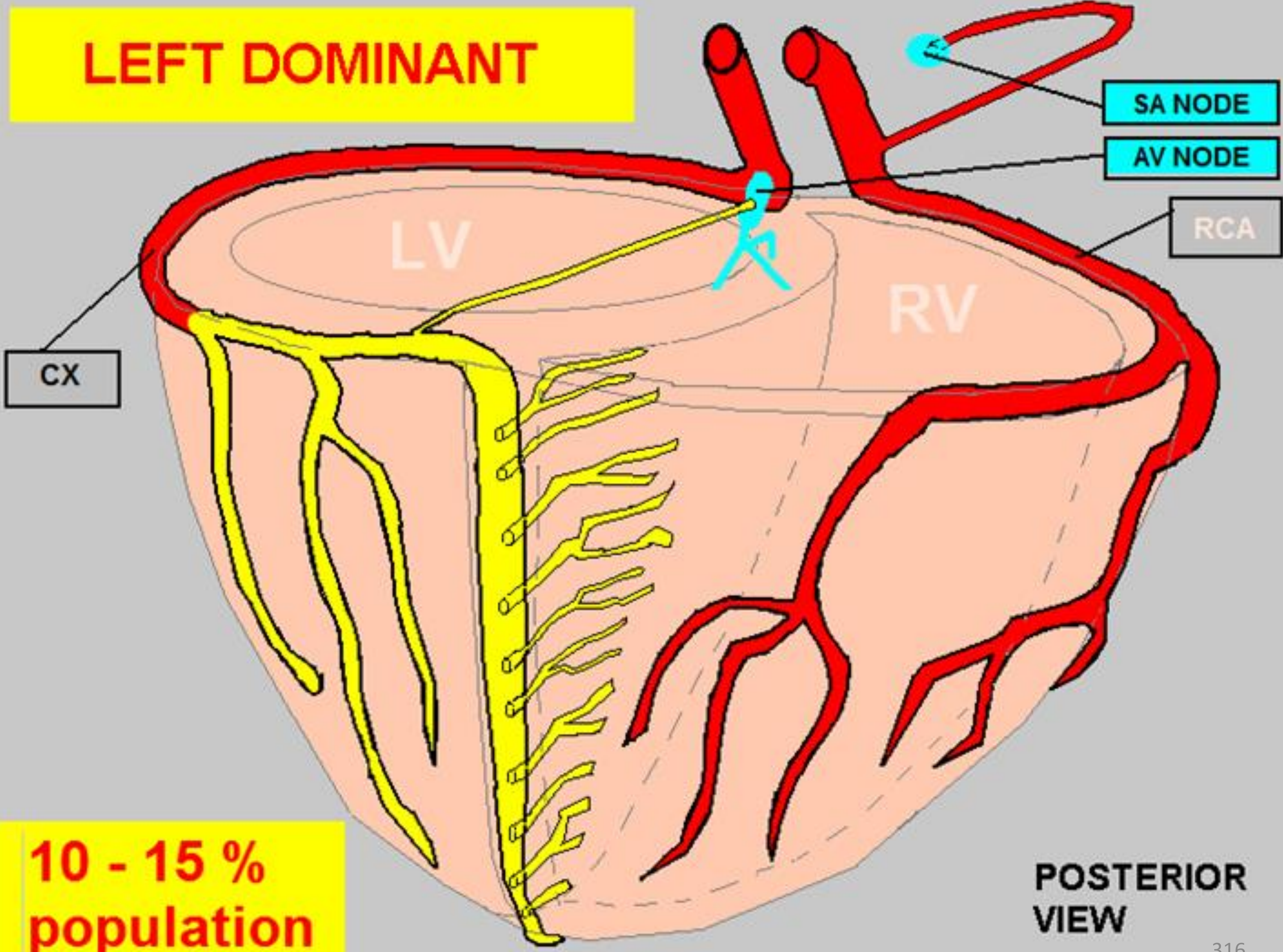
RIGHT DOMINANT



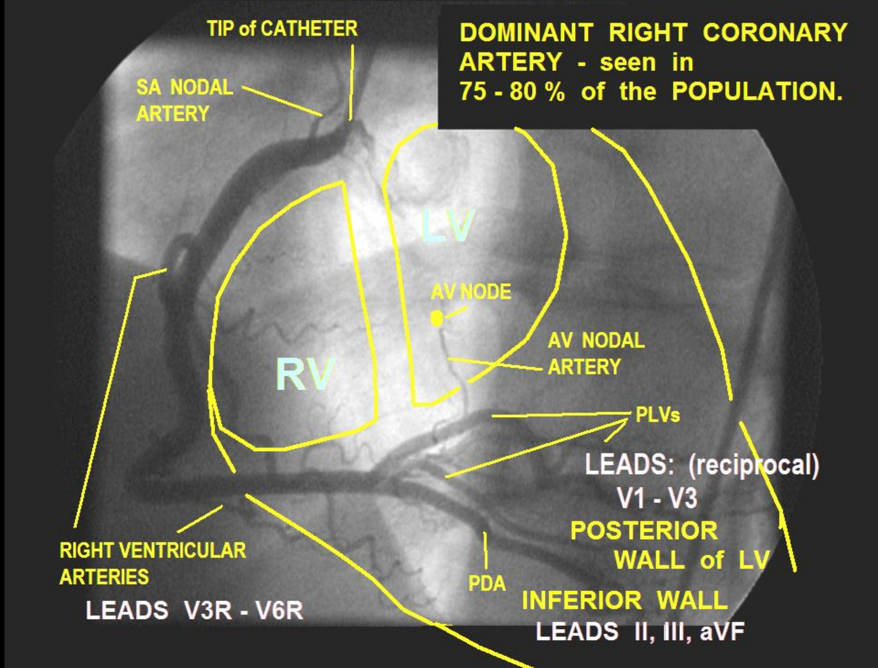
75 - 80 %
POPULATION

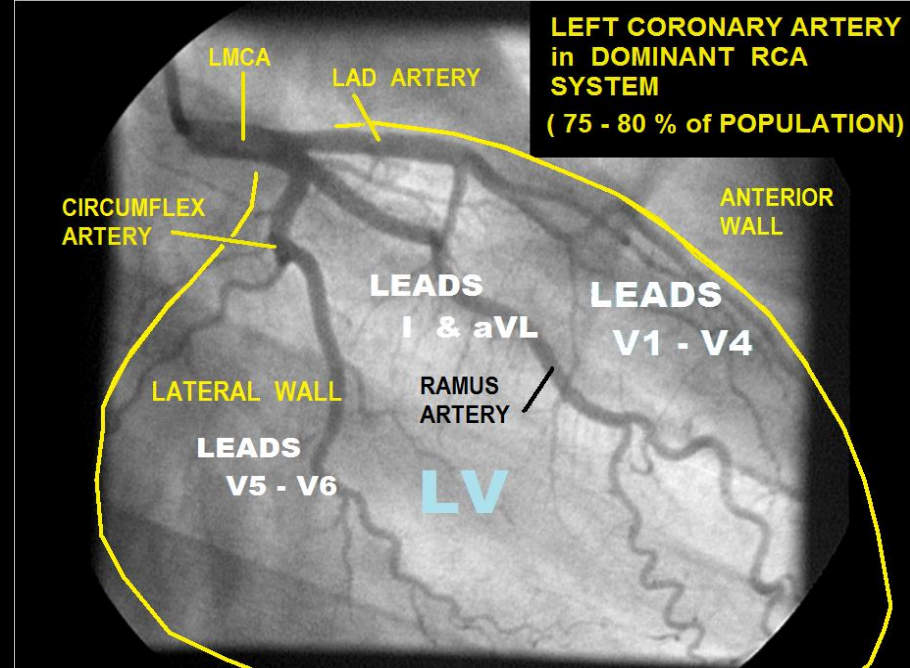
POSTERIOR VIEW

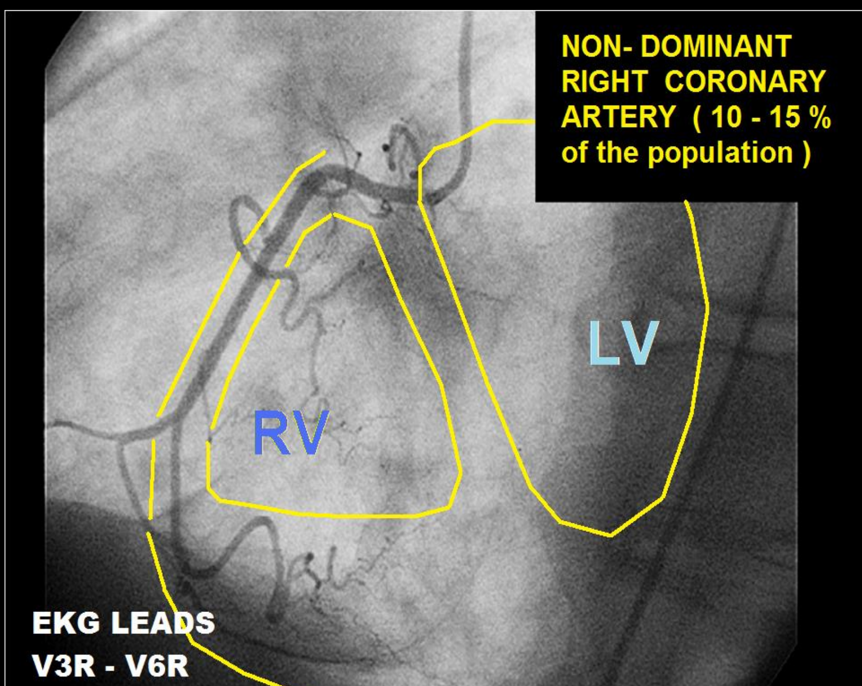
LEFT DOMINANT

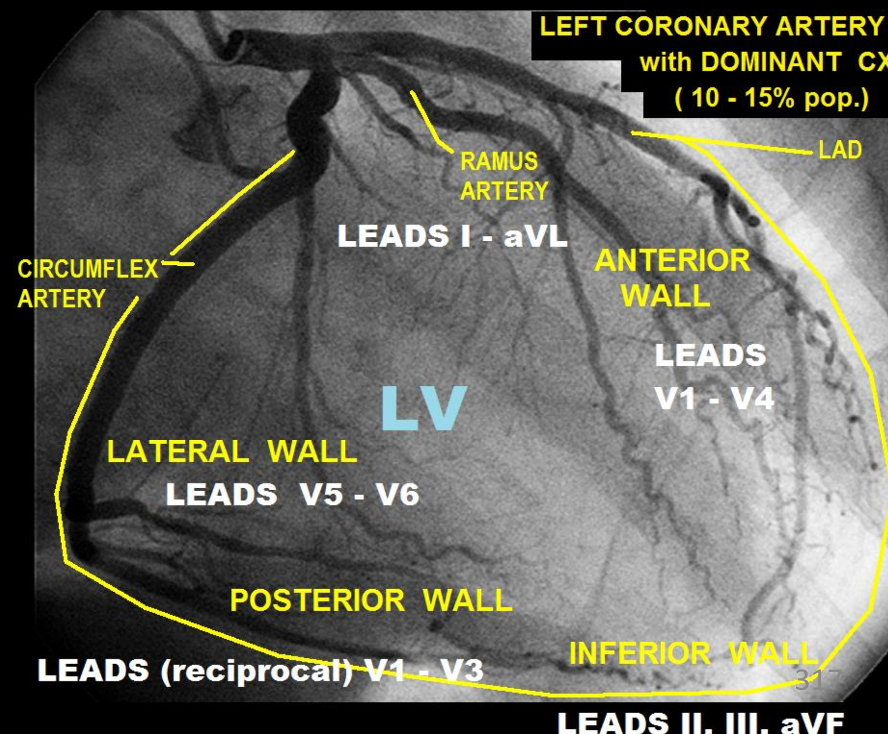


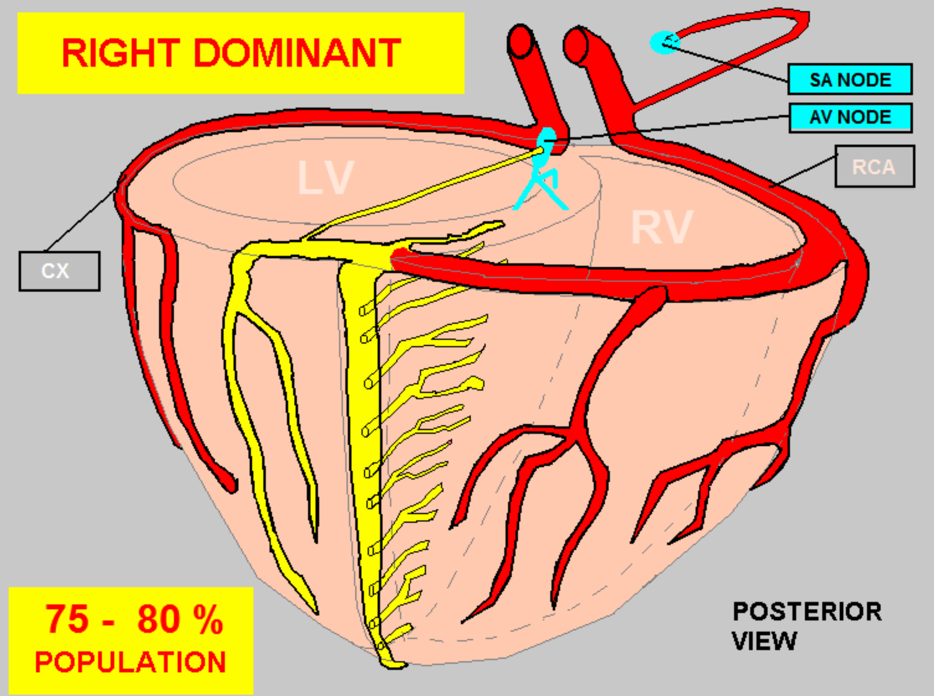
10 - 15 % population

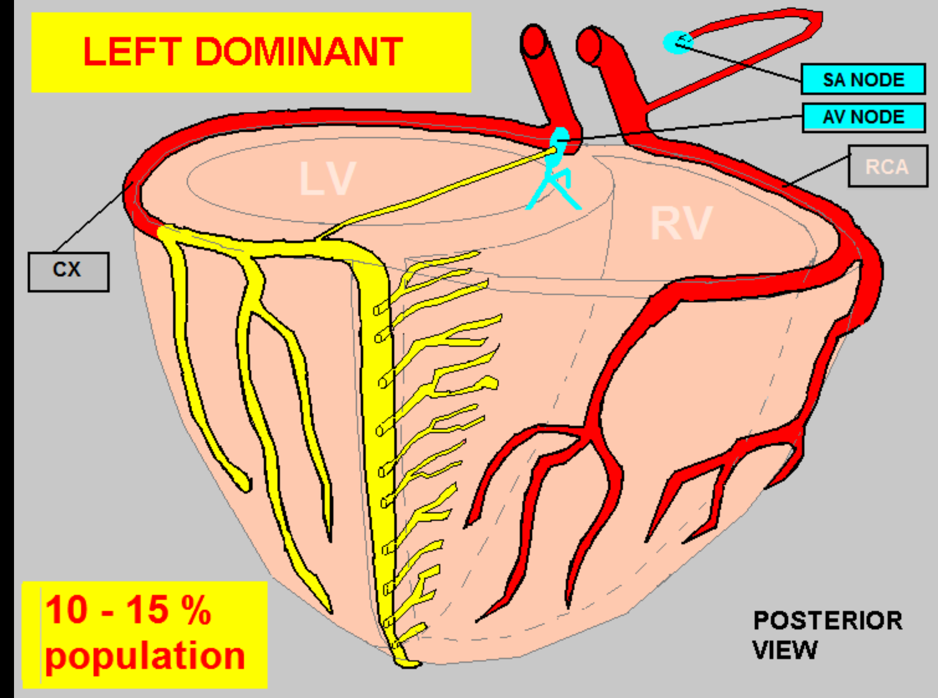

TIP of CATHETER
DOMINANT RIGHT CORONARY ARTERY - seen in 75 - 80 % of the POPULATION.
SA NODAL ARTERY
LV
AV NODE
RV
AV NODAL ARTERY
PLVs
LEADS: (reciprocal) V1 - V3
POSTERIOR WALL of LV
RIGHT VENTRICULAR ARTERIES
LEADS V3R - V6R
PDA
INFERIOR WALL
LEADS II, III, aVF

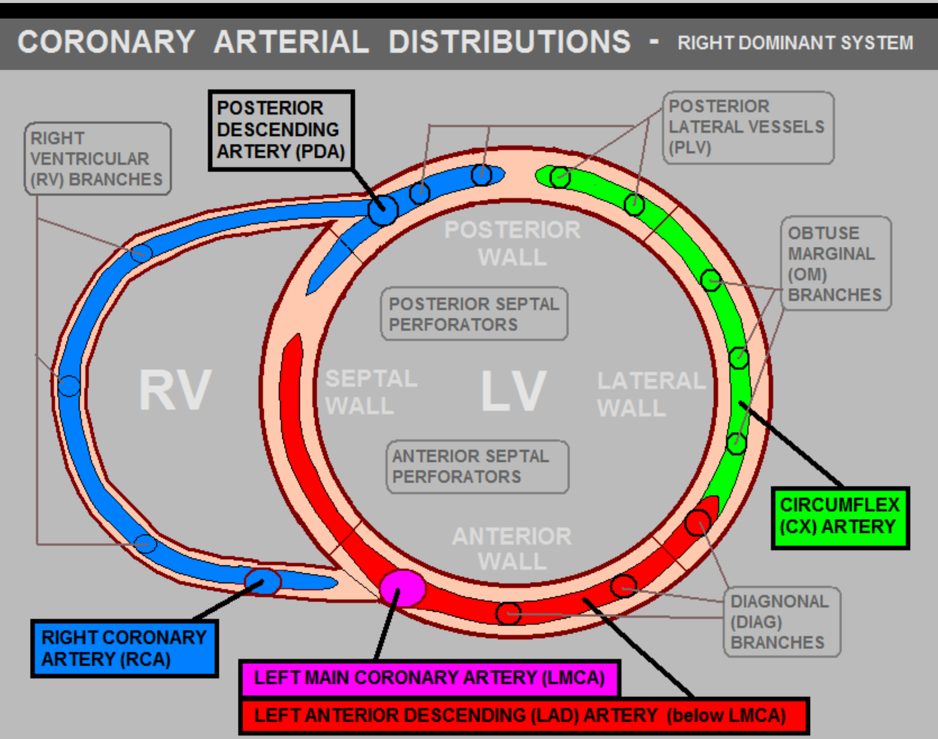

LMCA
LAD ARTERY
LEFT CORONARY ARTERY in DOMINANT RCA SYSTEM
(75 - 80 % of POPULATION)
CIRCUMFLEX ARTERY
ANTERIOR WALL
LEADS I & aVL
LEADS V1 - V4
RAMUS ARTERY
LATERAL WALL
LEADS V5 - V6
LV

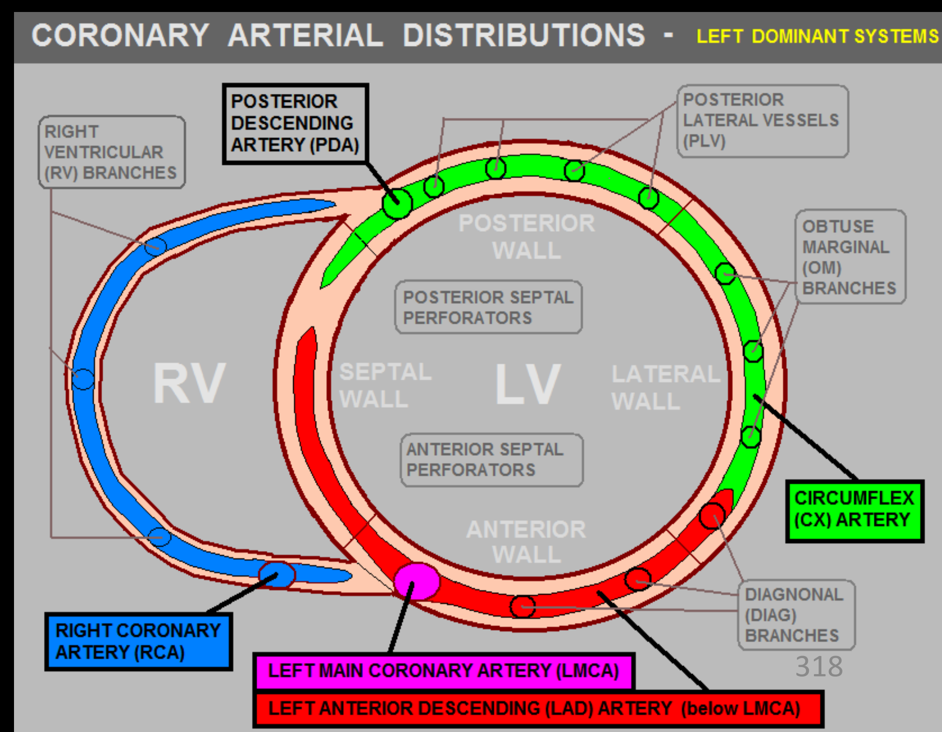

NON- DOMINANT RIGHT CORONARY ARTERY (10 - 15 % of the population)
LV
RV
EKG LEADS V3R - V6R


LEFT CORONARY ARTERY with DOMINANT CX (10 - 15% pop.)
LAD
RAMUS ARTERY
LEADS I - aVL
ANTERIOR WALL
CIRCUMFLEX ARTERY
LEADS V1 - V4
LV
LATERAL WALL
LEADS V5 - V6
POSTERIOR WALL
LEADS (reciprocal) V1 - V3
INFERIOR WALL
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
RIGHT VENTRICULAR (RV) BRANCHES
POSTERIOR DESCENDING ARTERY (PDA)
POSTERIOR LATERAL VESSELS (PLV)
POSTERIOR WALL
OBTUSE MARGINAL (OM) BRANCHES
POSTERIOR SEPTAL PERFORATORS
RV
SEPTAL WALL
LV
LATERAL WALL
ANTERIOR SEPTAL PERFORATORS
CIRCUMFLEX (CX) ARTERY
ANTERIOR WALL
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
RIGHT VENTRICULAR (RV) BRANCHES
POSTERIOR DESCENDING ARTERY (PDA)
POSTERIOR LATERAL VESSELS (PLV)
POSTERIOR WALL
OBTUSE MARGINAL (OM) BRANCHES
POSTERIOR SEPTAL PERFORATORS
RV
SEPTAL WALL
LV
LATERAL WALL
ANTERIOR SEPTAL PERFORATORS
CIRCUMFLEX (CX) ARTERY
ANTERIOR WALL
DIAGNONAL (DIAG) BRANCHES
RIGHT CORONARY ARTERY (RCA)
LEFT MAIN CORONARY ARTERY (LMCA)
LEFT ANTERIOR DESCENDING (LAD) ARTERY (below LMCA)

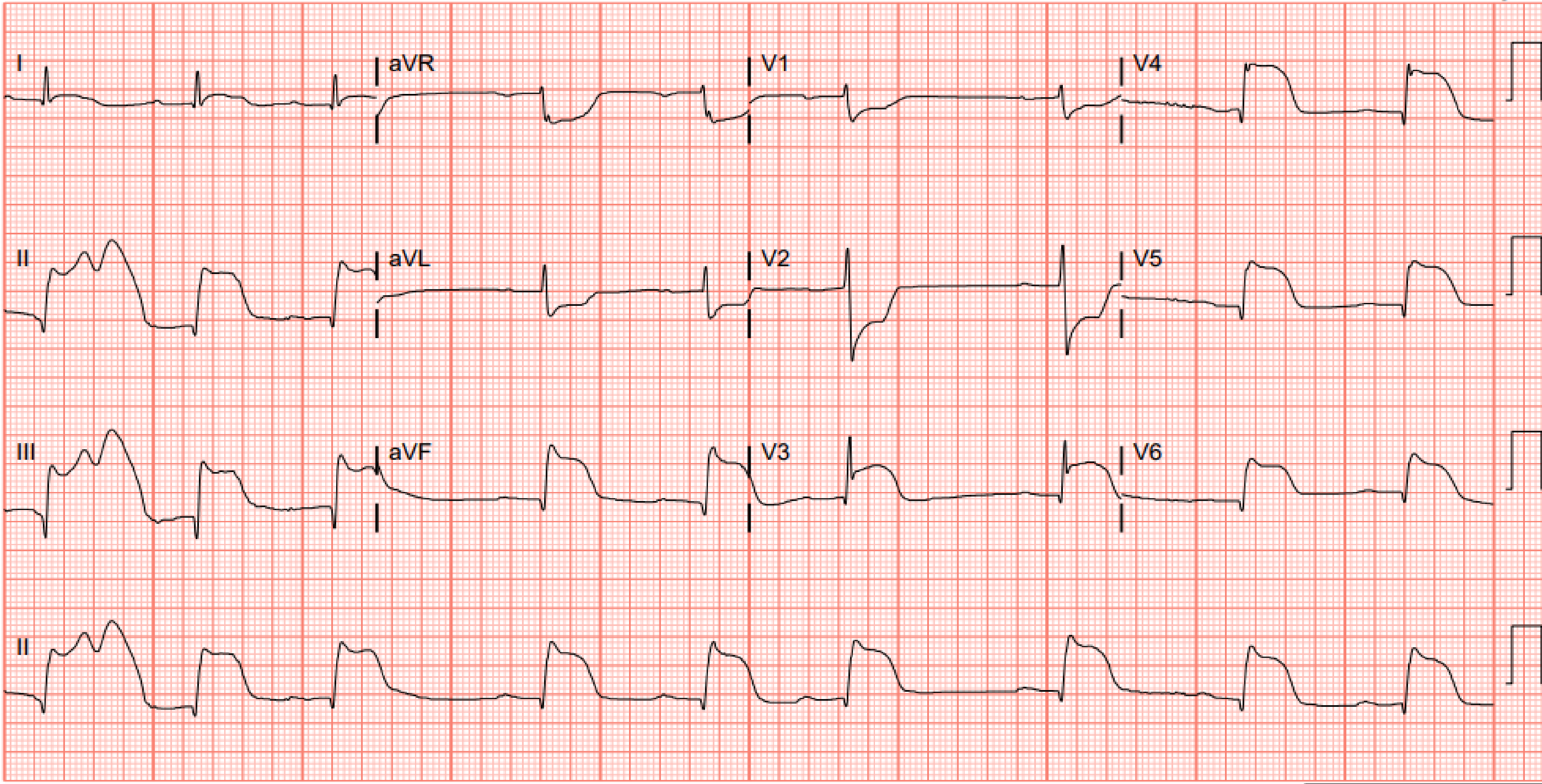
Pat ID [redacted] 01/20/2021 07:46:46 [redacted]
08/17/1955 65 yrs
RX Account # [redacted]
DX

Bayfront Health Seven Rivers ED
Dept ED
Room EDWR
Tech mg

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

Req Provider: Xandus Chen

- Abnormal ECG - **Unconfirmed Diagnosis**



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

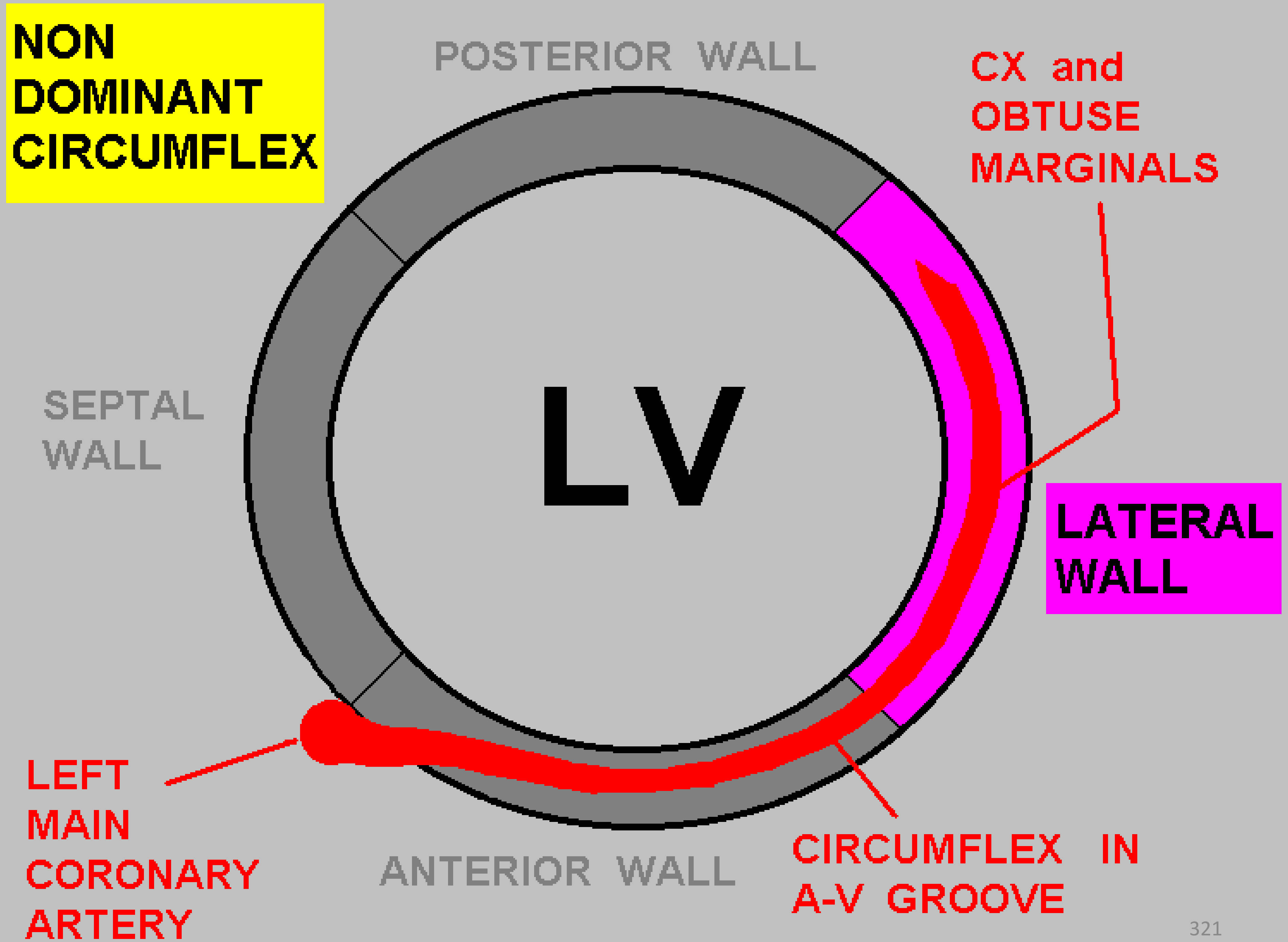
CIRCUMFLEX ARTERY (CX)

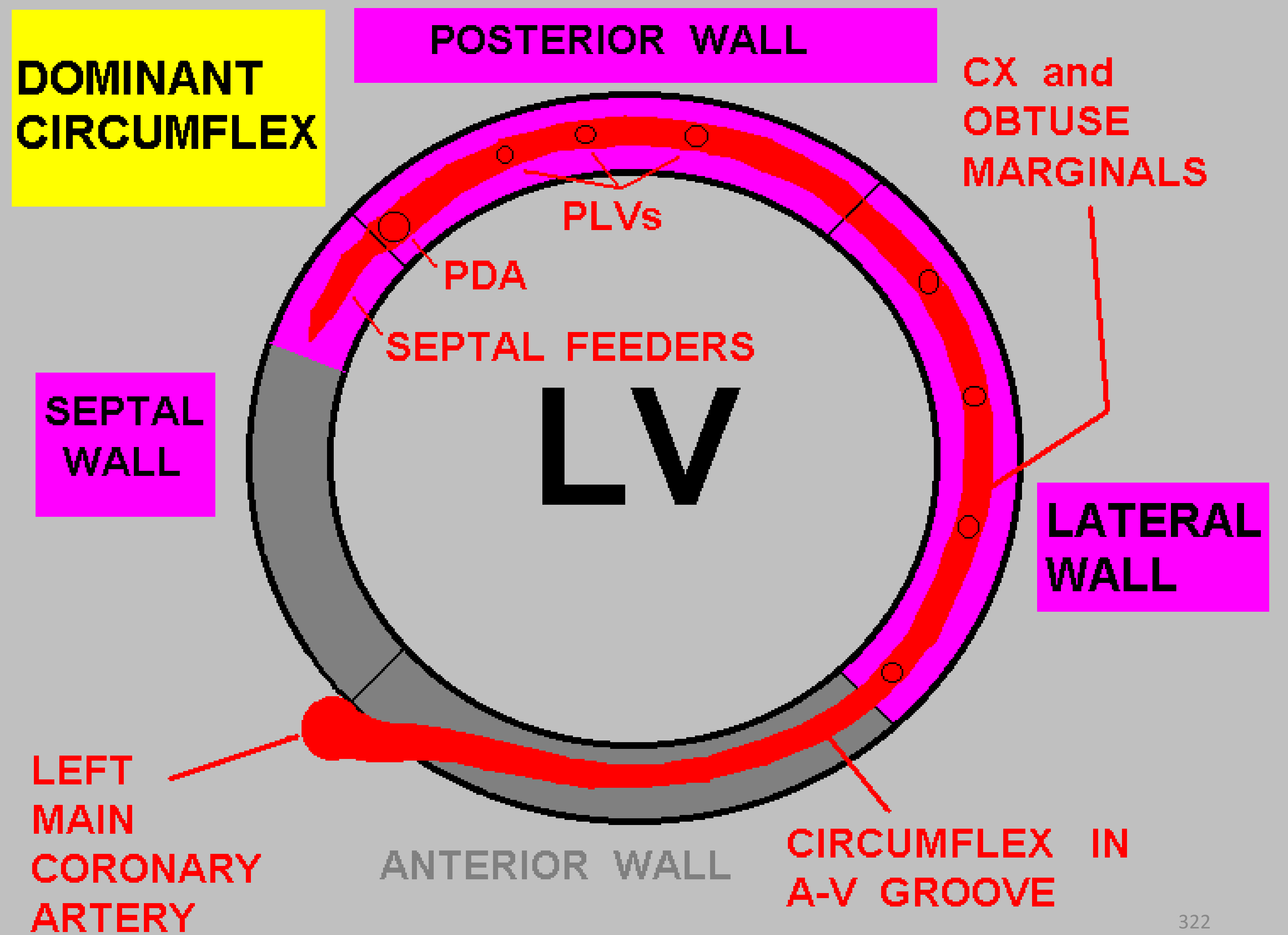
- NON-DOMINANT CX:

CX = 15 - 30% OF LV MASS

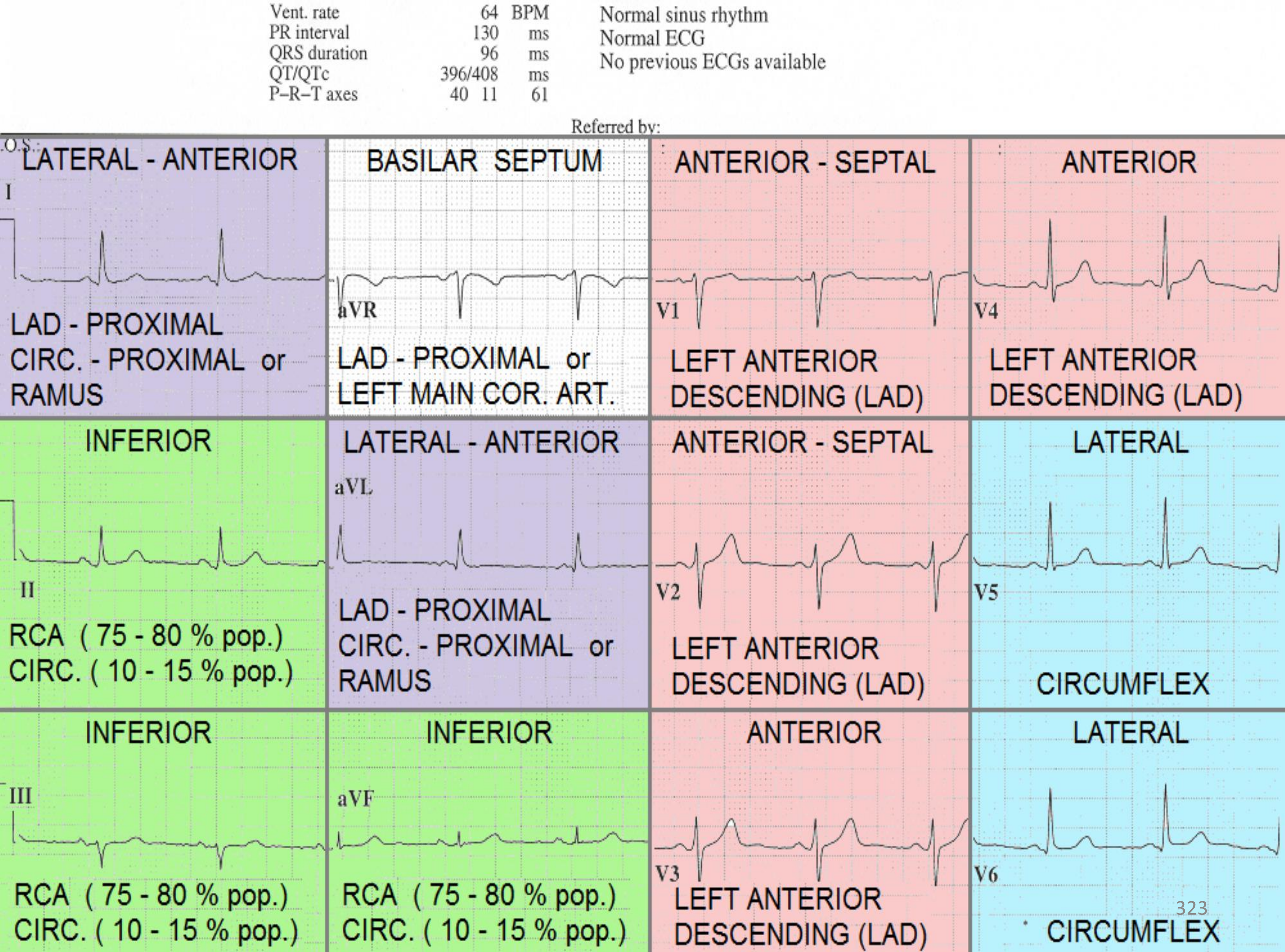
- DOMINANT CX:

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


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



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


Vent. rate 64 BPM
PR interval 130 ms
QRS duration 96 ms
QT/QTc 396/408 ms
P–R–T axes 40 11 61
Normal sinus rhythm
Normal ECG
No previous ECGs available
Referred by:
LATERAL - ANTERIOR
I
LAD - PROXIMAL
CIRC. - PROXIMAL or
RAMUS
BASILAR SEPTUM
aVR
LAD - PROXIMAL or
LEFT MAIN COR. ART.
ANTERIOR - SEPTAL
V1
LEFT ANTERIOR
DESCENDING (LAD)
ANTERIOR
V4
LEFT ANTERIOR
DESCENDING (LAD)
INFERIOR
II
RCA (75 - 80 % pop.)
CIRC. (10 - 15 % pop.)
LATERAL - ANTERIOR
aVL
LAD - PROXIMAL
CIRC. - PROXIMAL or
RAMUS
ANTERIOR - SEPTAL
V2
LEFT ANTERIOR
DESCENDING (LAD)
LATERAL
V5
CIRCUMFLEX
INFERIOR
III
RCA (75 - 80 % pop.)
CIRC. (10 - 15 % pop.)
INFERIOR
aVF
RCA (75 - 80 % pop.)
CIRC. (10 - 15 % pop.)
ANTERIOR
V3
LEFT ANTERIOR
DESCENDING (LAD)
LATERAL
V6
CIRCUMFLEX

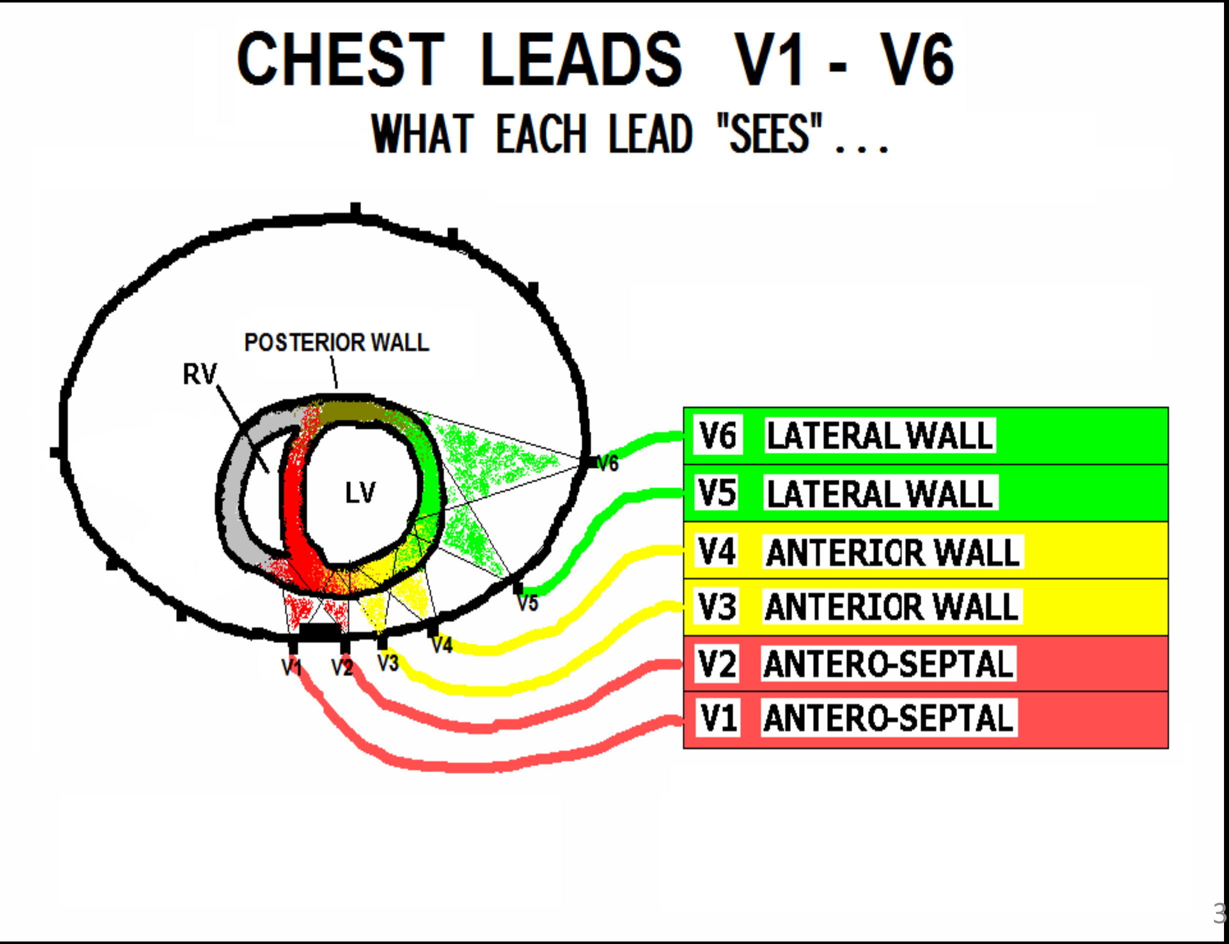
The 12 Lead ECG

Has TWO major BLIND SPOTS . . . . .

The POSTERIOR WALL

&

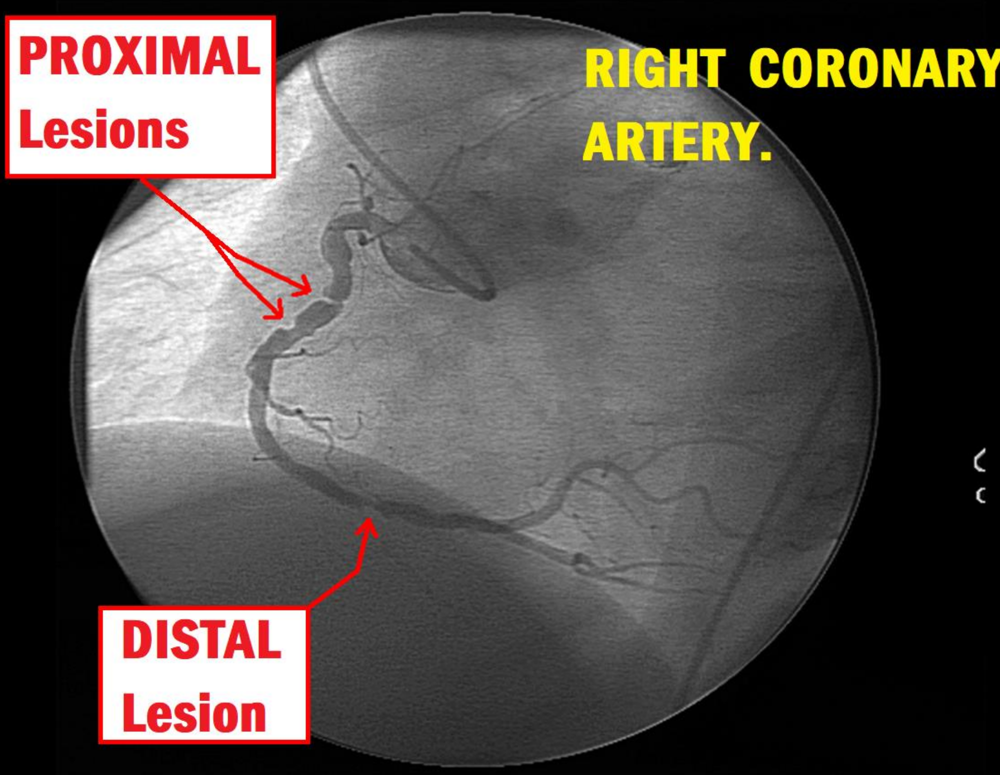
RIGHT VENTRICLE

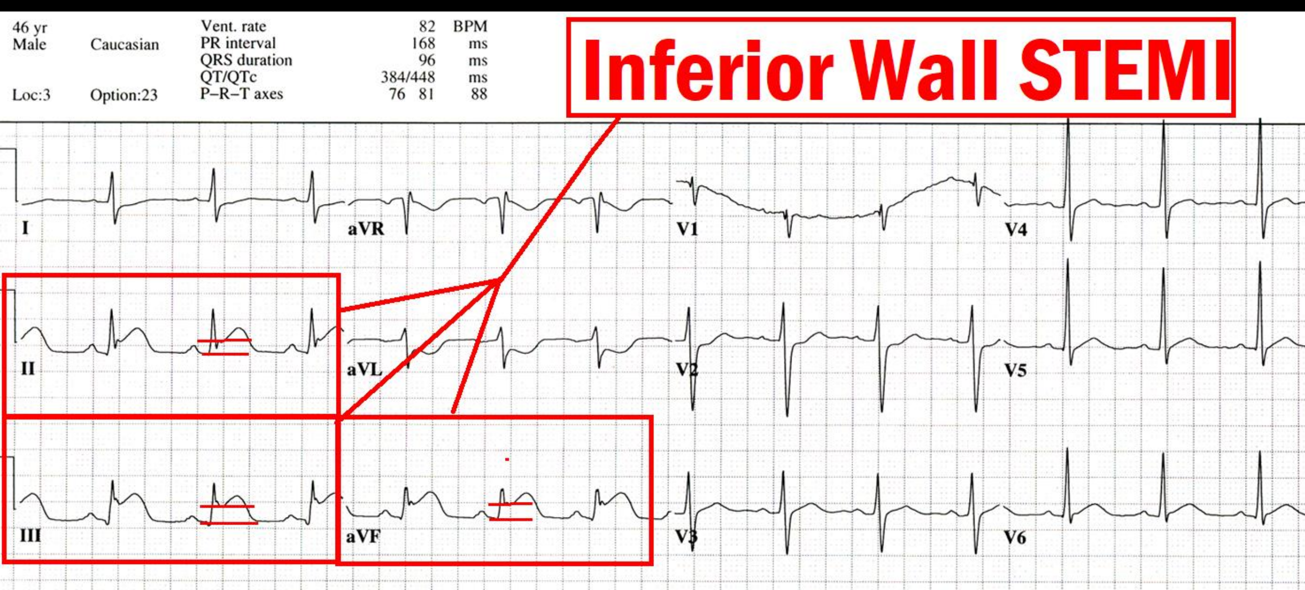


When do we need to see the Right Ventricle?

- 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 . . .

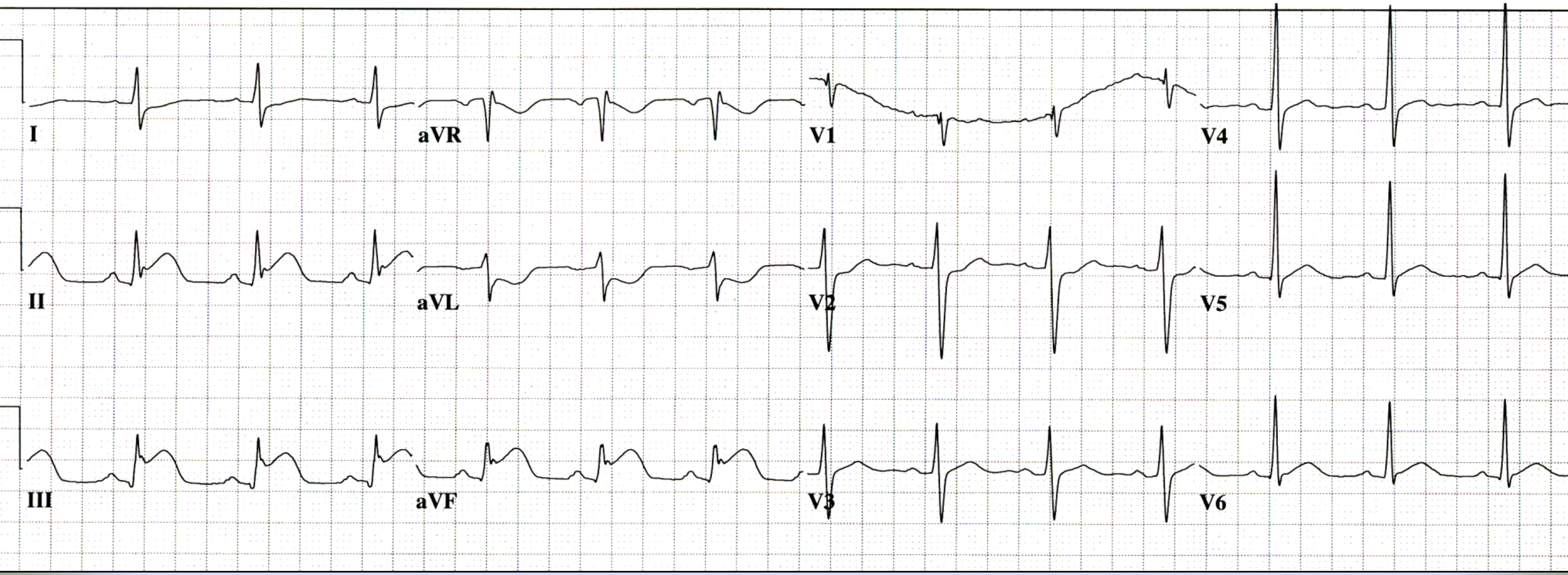
. . . such as in cases of
INFERIOR WALL M.I.

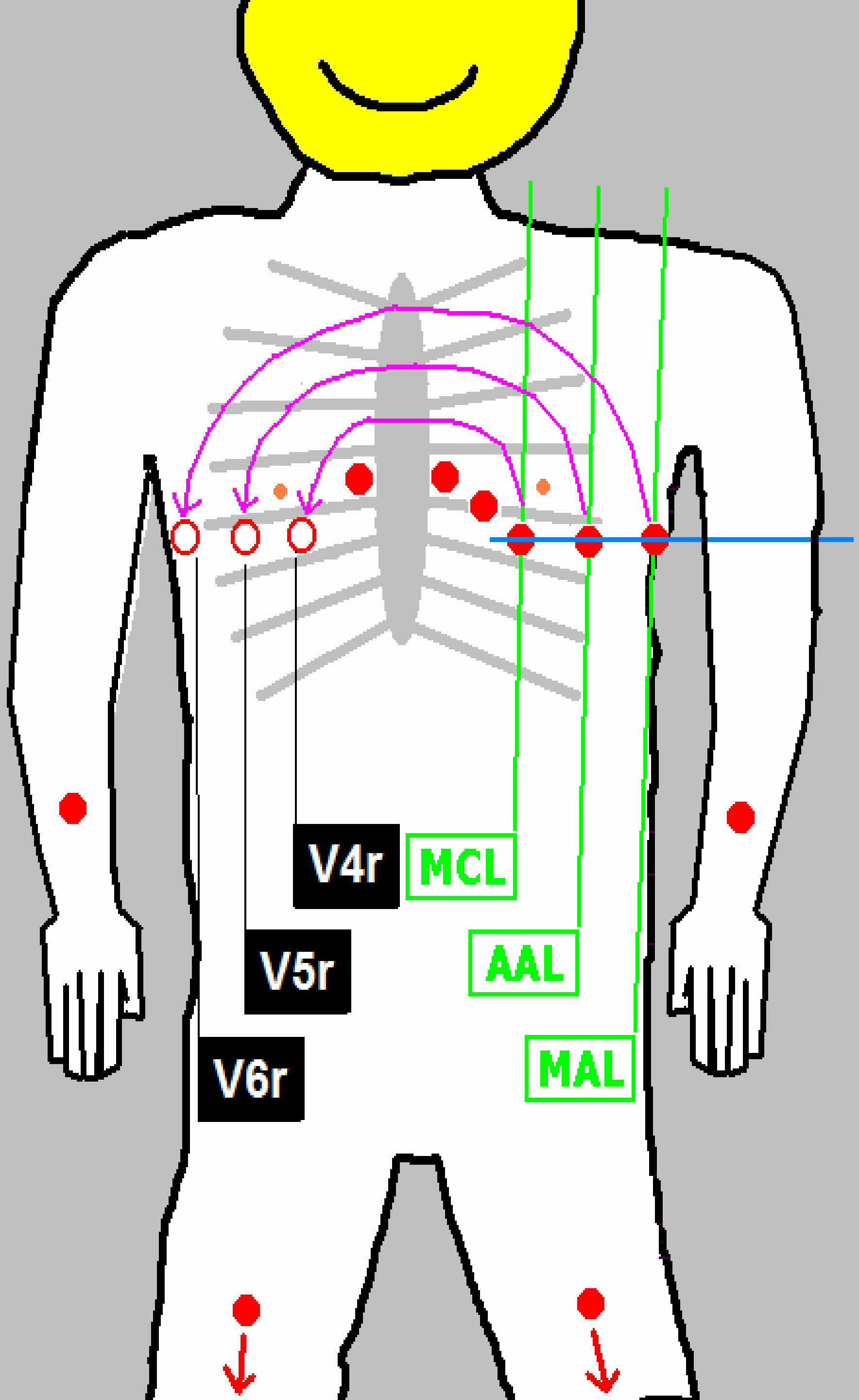
☞ You must do a
RIGHT - SIDED EKG ! !

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



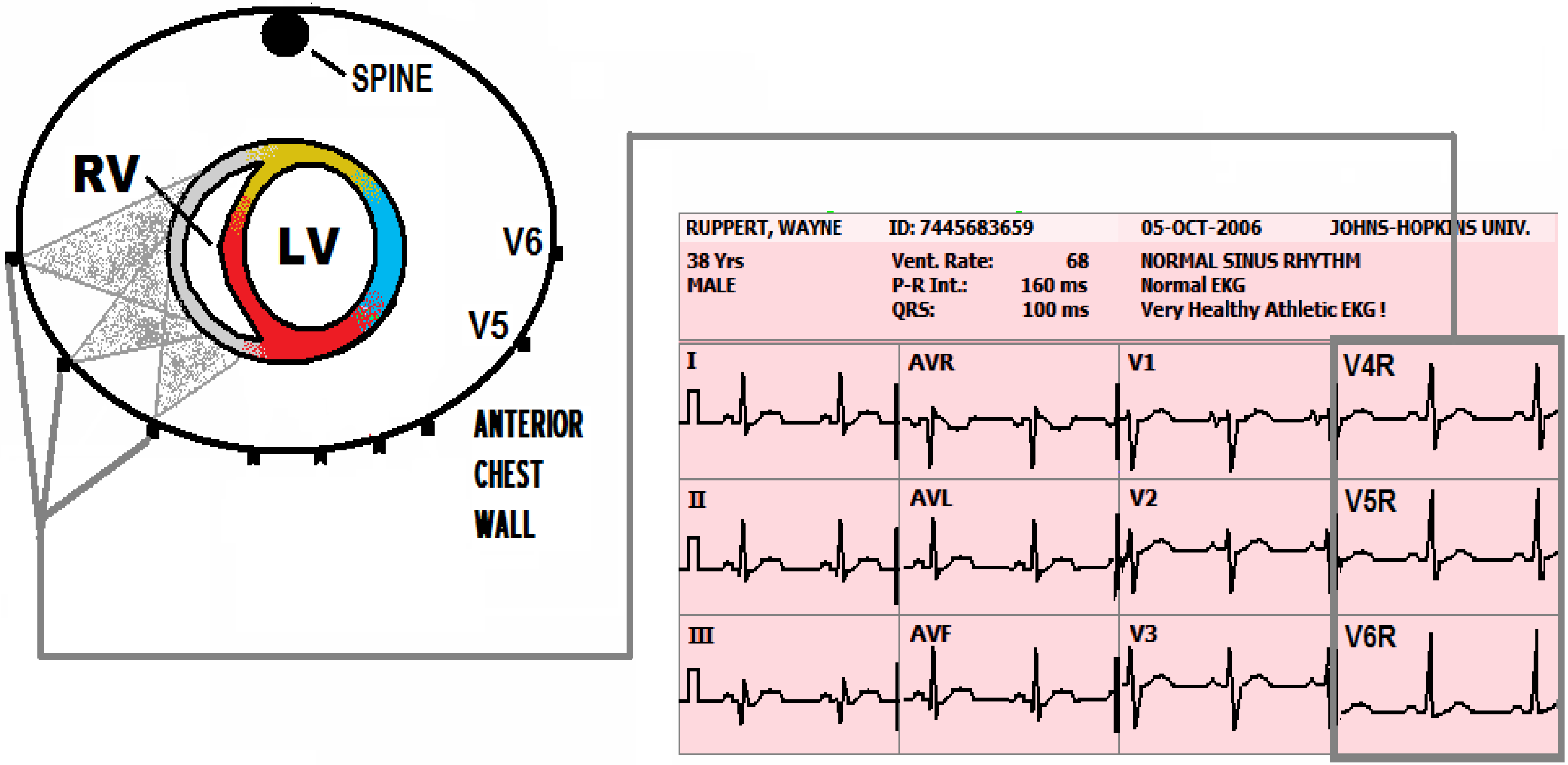


To do a
RIGHT - SIDED EKG . .

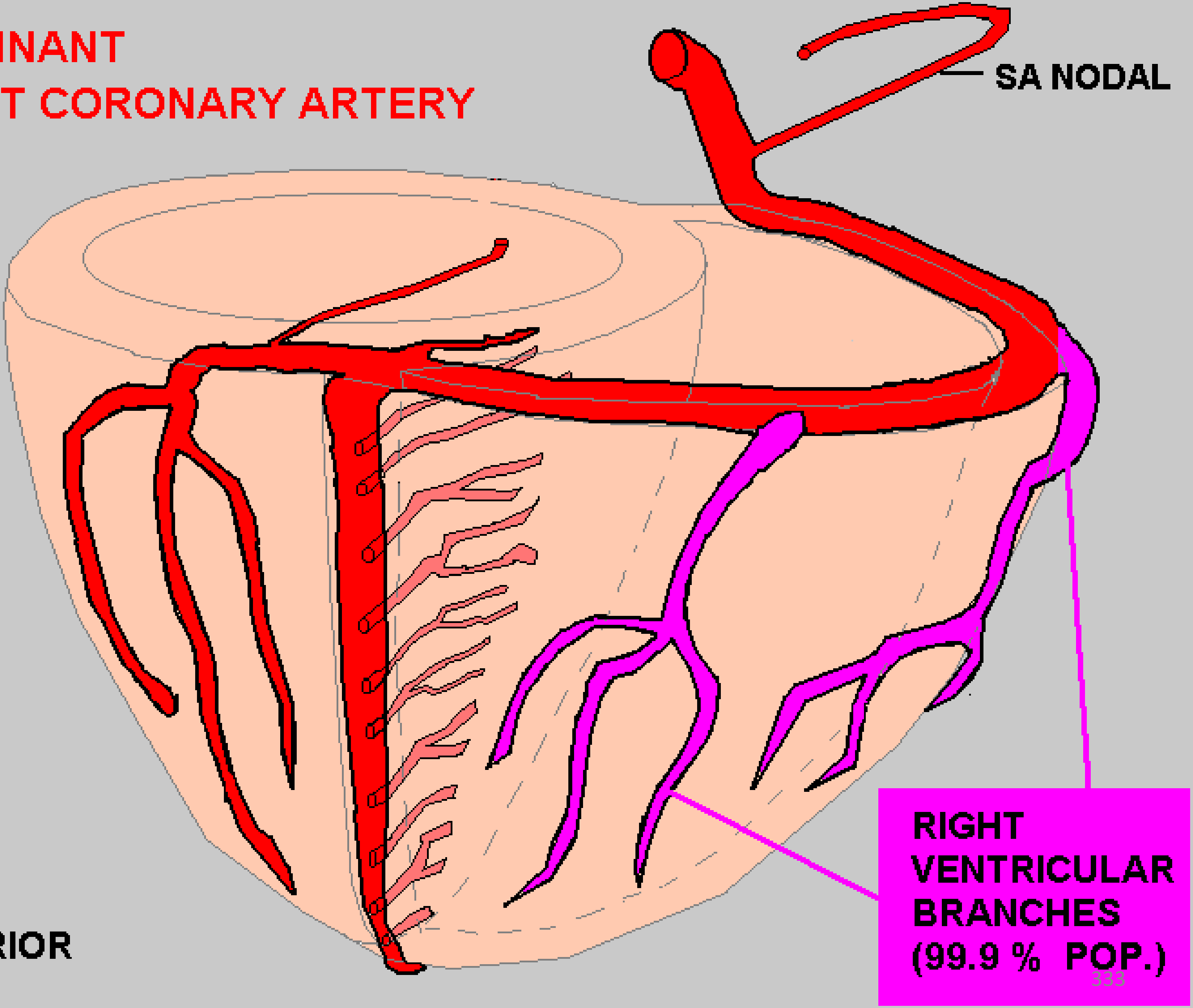
MOVE leads
V4, V5, and V6

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

V4R - V6R VIEW THE RIGHT VENTRICLE



DOMINANT RIGHT CORONARY ARTERY



POSTERIOR VIEW

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

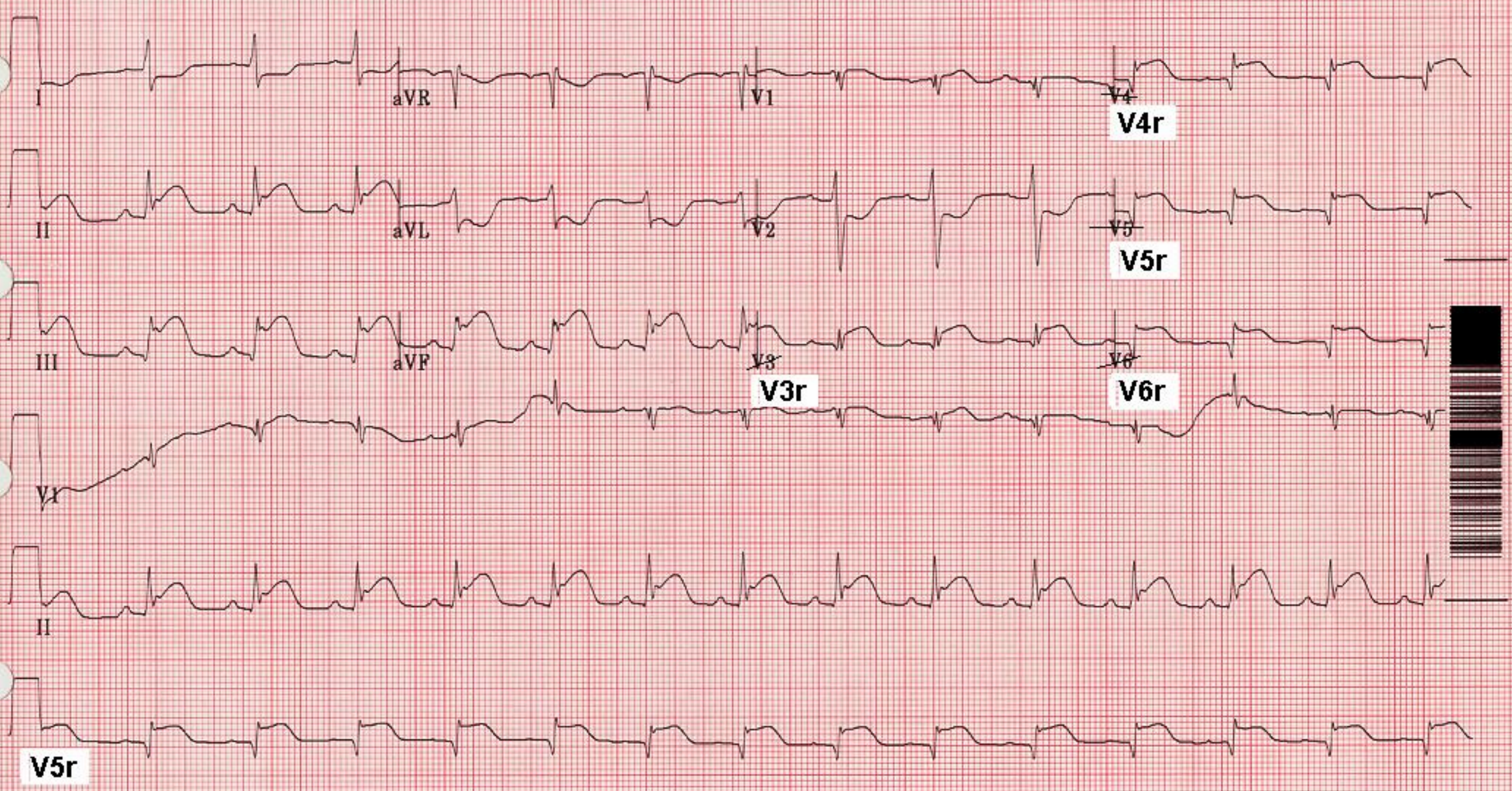
Normal sinus rhythm
~~Anterolateral infarct, possibly acute~~
Inferior injury pattern
** ** ** ** * Acute MI * ** ** ** **
Abnormal ECG

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™ v250

KENDALL MEDI-TRACE PRINTED IN U.S.A. 201

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]

RX
DX

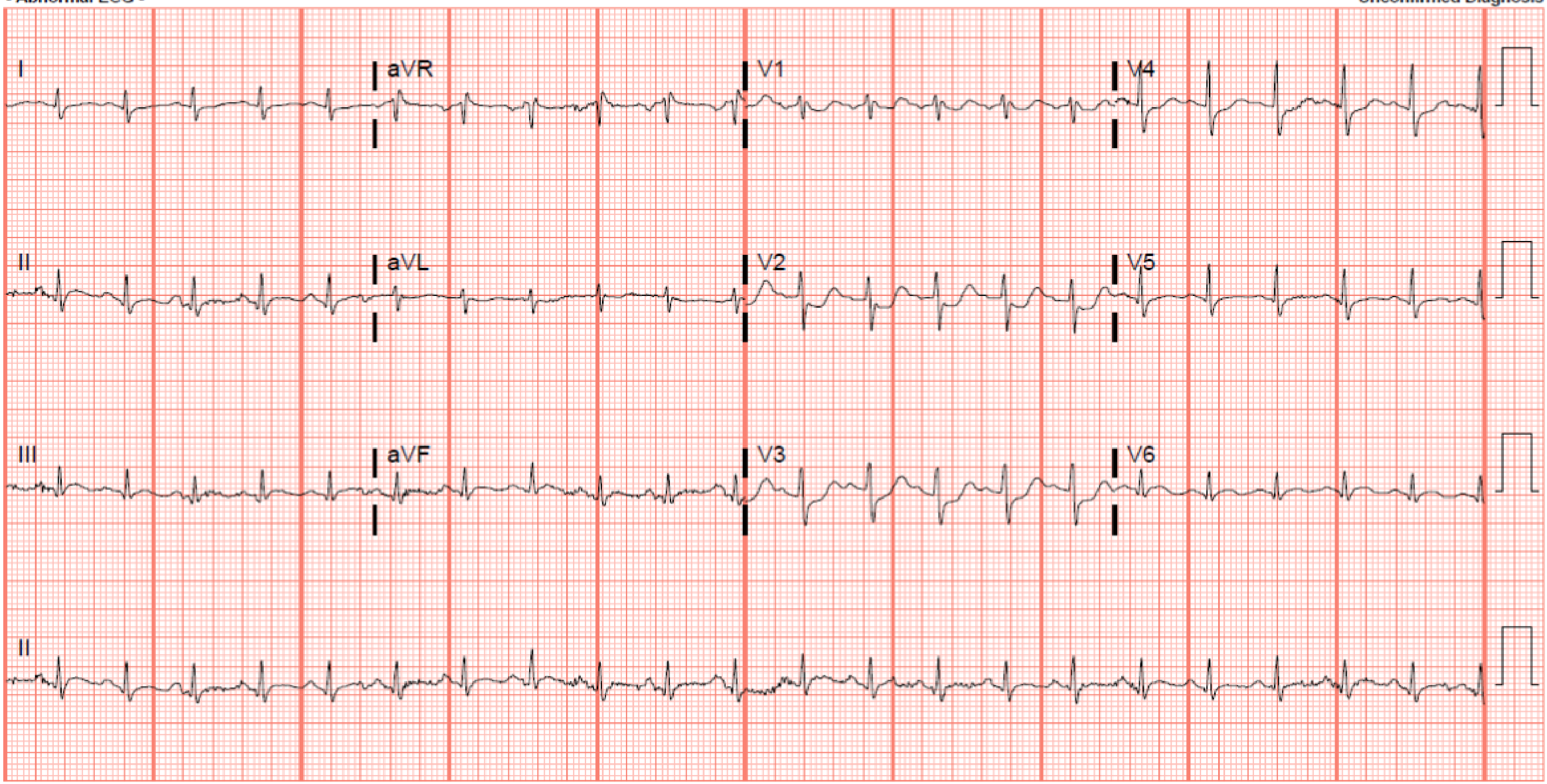
Bayfront Health Seven Rivers ED
Dept ED
Room [redacted]
Tech LDC

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

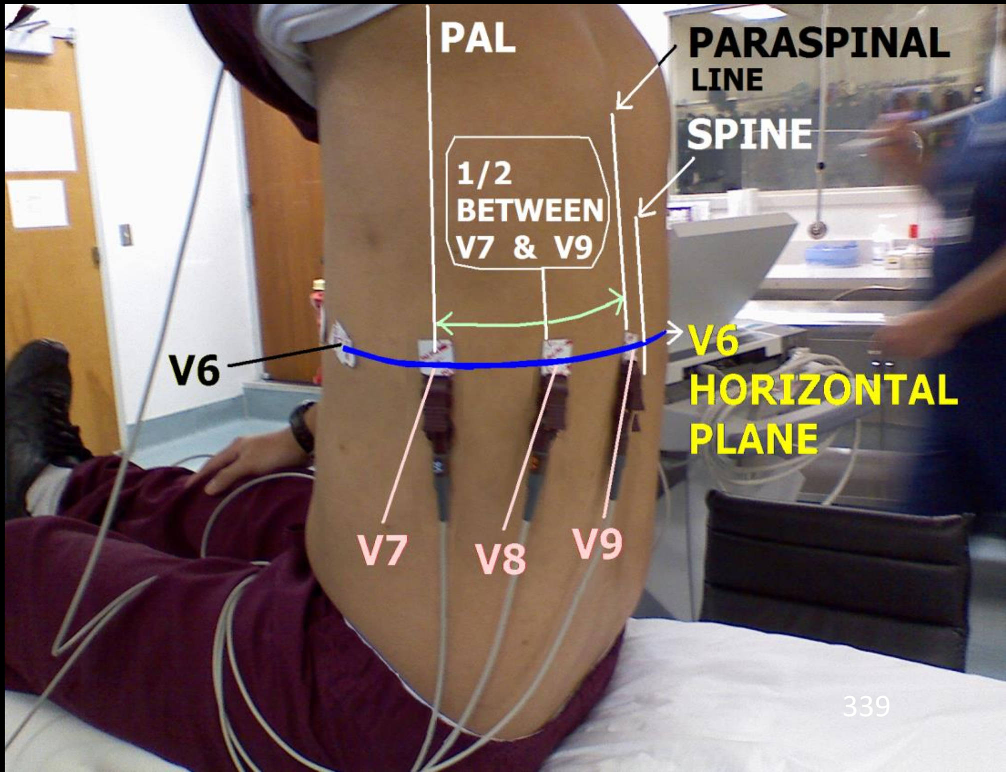
(V7 - V9)

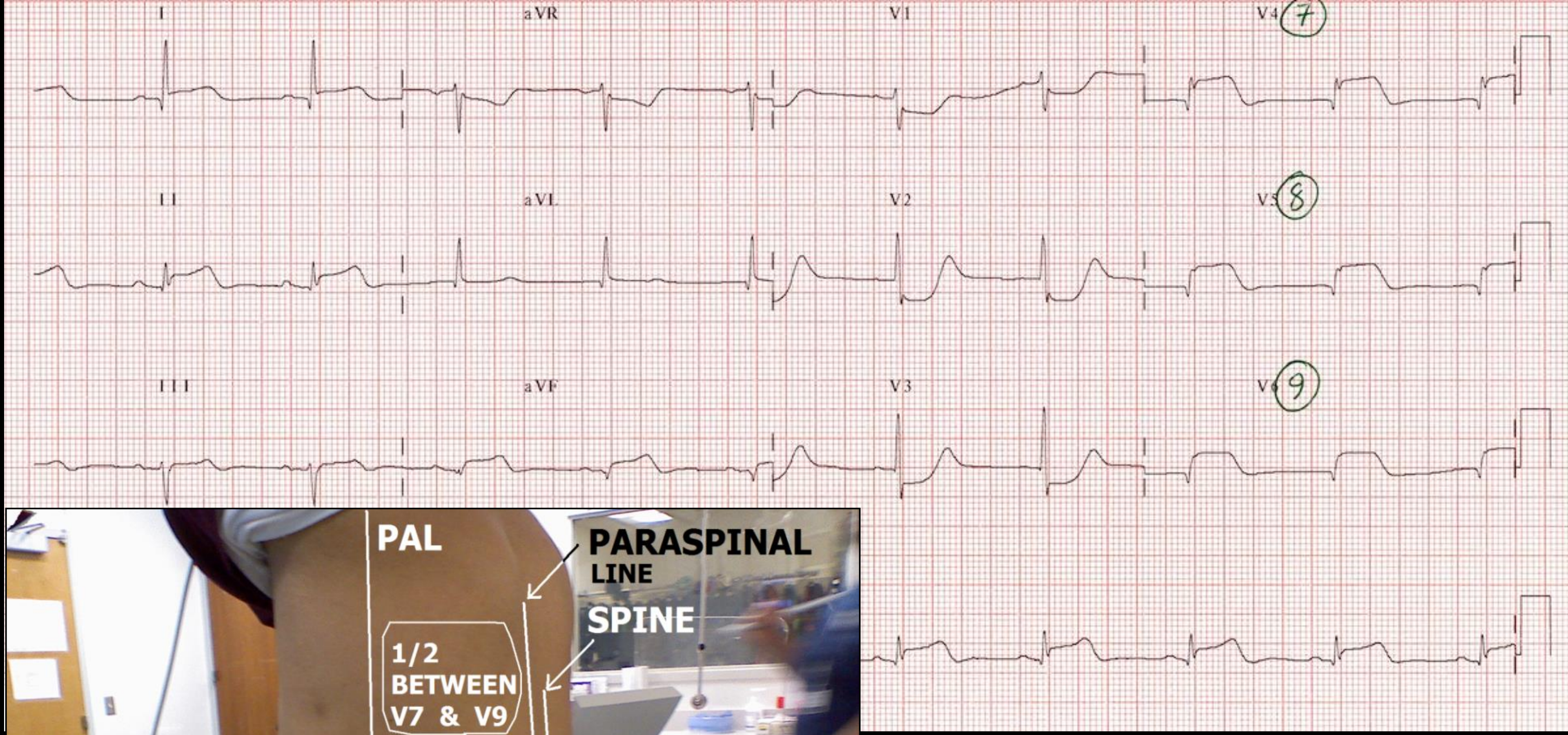
to see if you Patient is having a

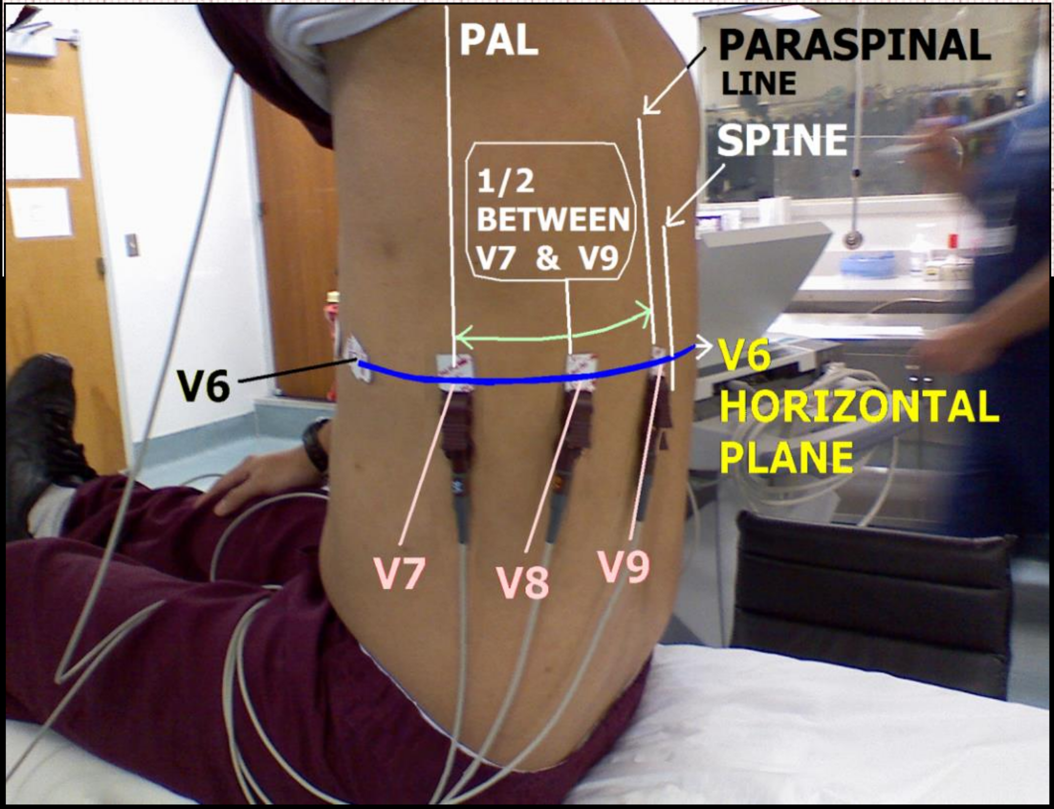
POSTERIOR WALL STEMI

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

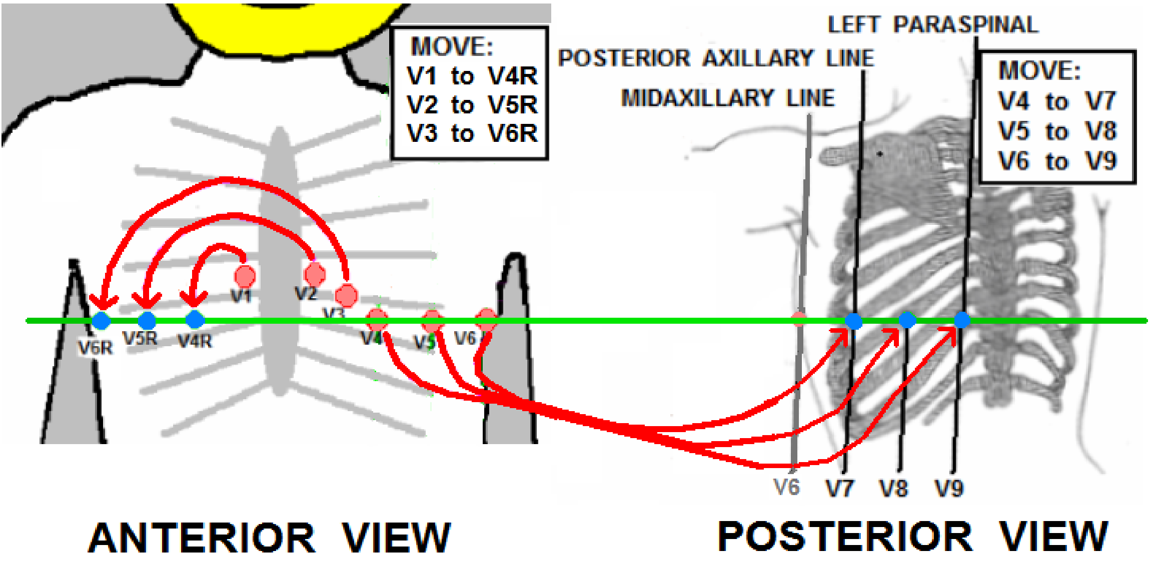
. . . To DIGANOSE Posterior Wall STEMI, we should see LEADS V7 – V9 !!

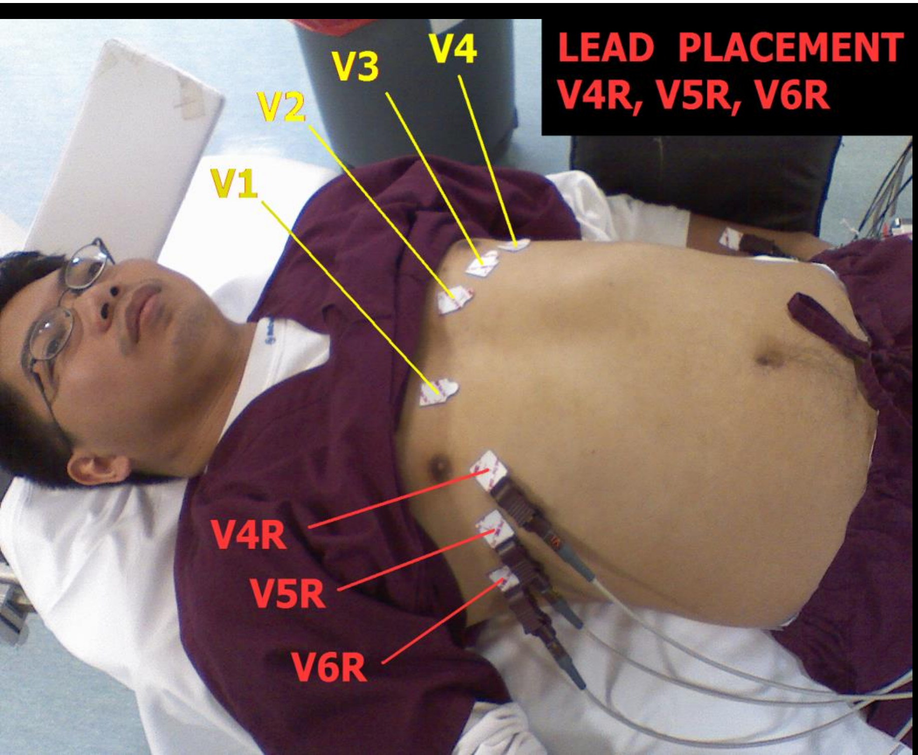


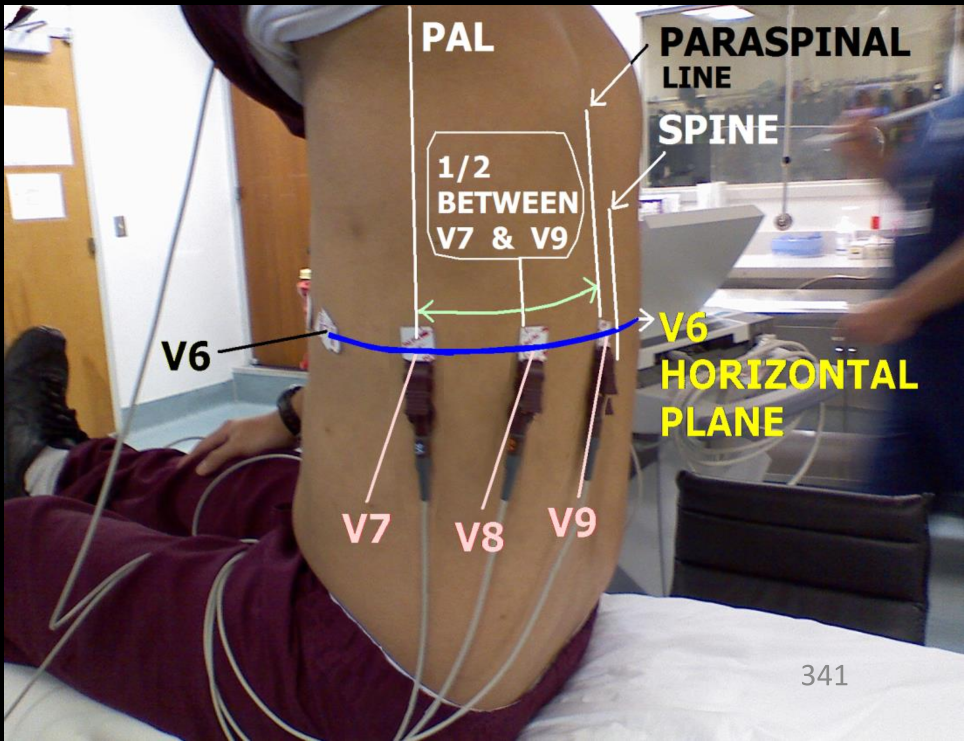

I
aVR
V1
V4 7
II
aVL
V2
V5 8
III
aVF
V3
V6 9

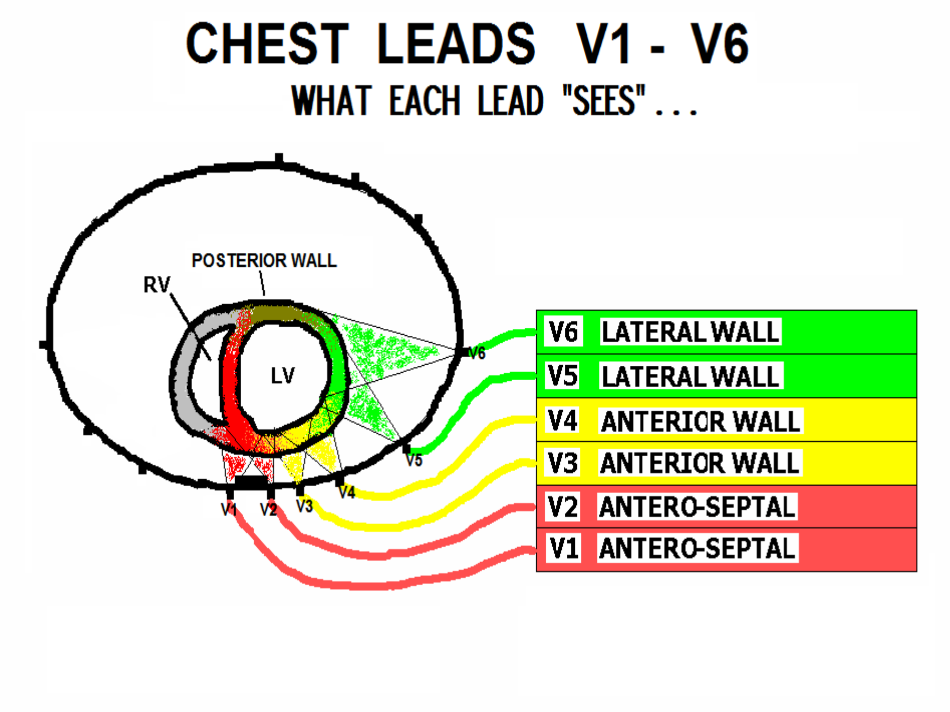

PAL
PARASPINAL LINE
SPINE
1/2 BETWEEN V7 & V9
V6
V6 HORIZONTAL PLANE
V7
V8
V9

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



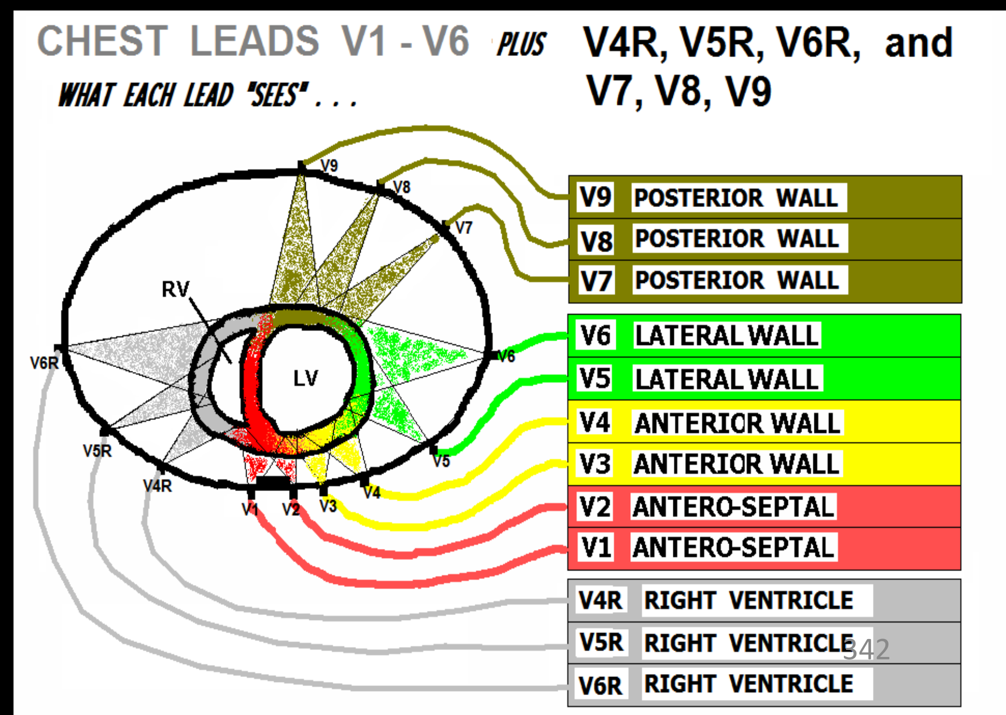


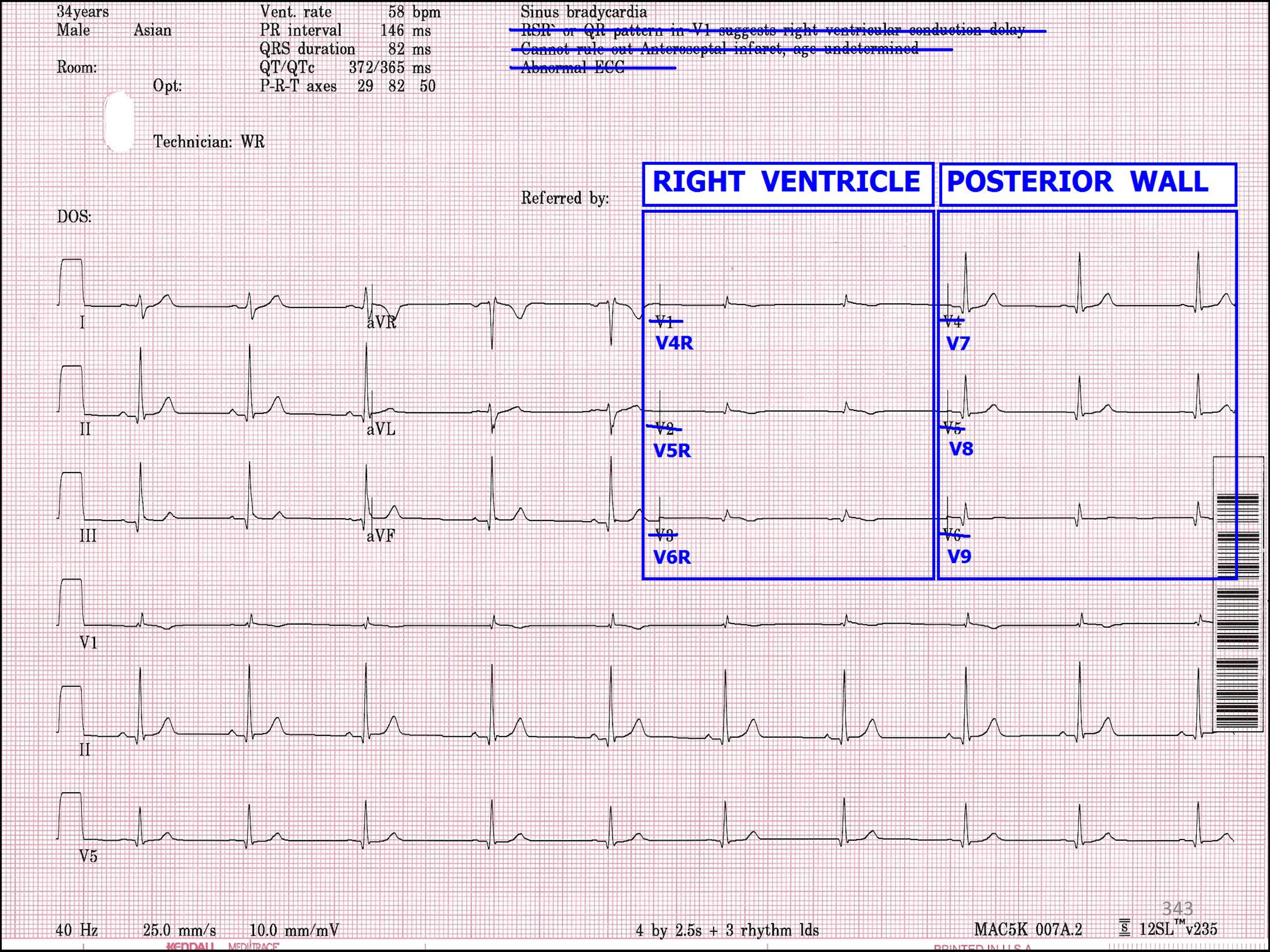




⇦ The 12 Lead ECG

The 18 Lead ECG ⇨




34years
Male Asian
Room:
Opt:
Technician: WR
DOS:
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:
RIGHT VENTRICLE
POSTERIOR WALL
I
II
III
aVR
aVL
aVF
~~V1~~ V4R
~~V2~~ V5R
~~V3~~ V6R
~~V4~~ V7
~~V5~~ V8
~~V6~~ V9
V1
II
V5
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 !!!

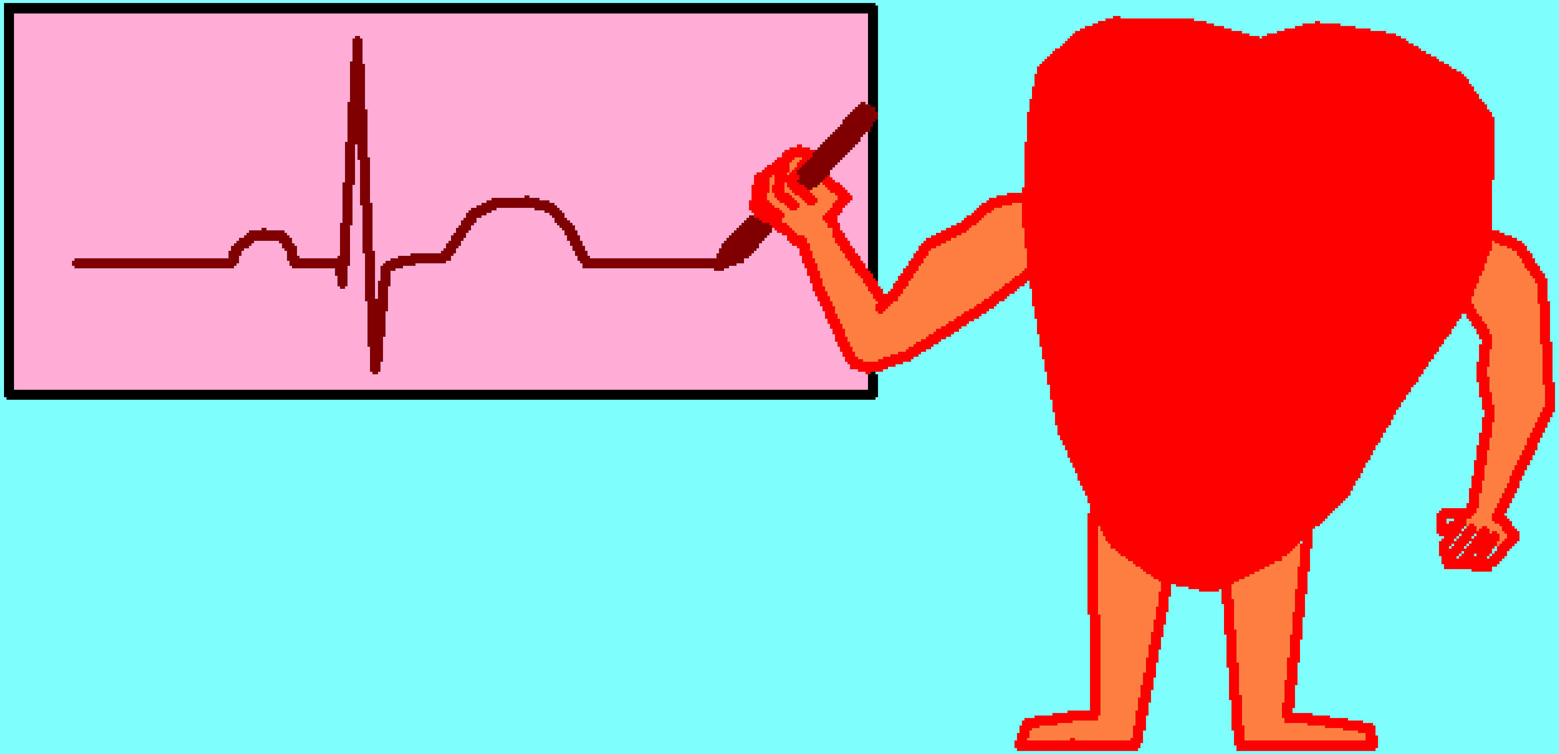
POSTERIOR WALL MI usually accompanies INFERIOR and/or LATERAL WALL MI !!!

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

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

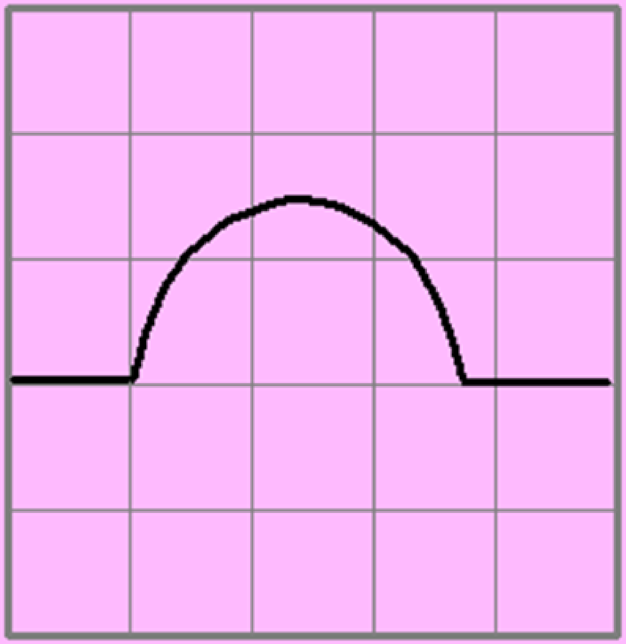
PUTTING IT ALL ON PAPER . . .

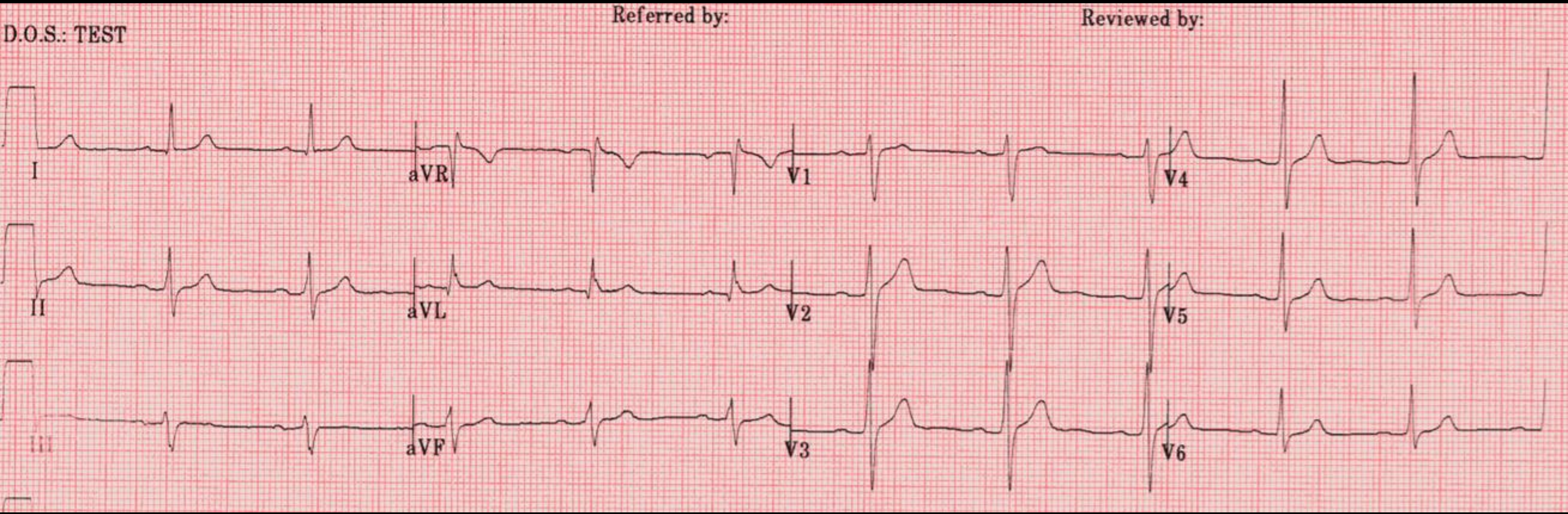
WAVEFORMS and INTERVALS . . .



THE P WAVE

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

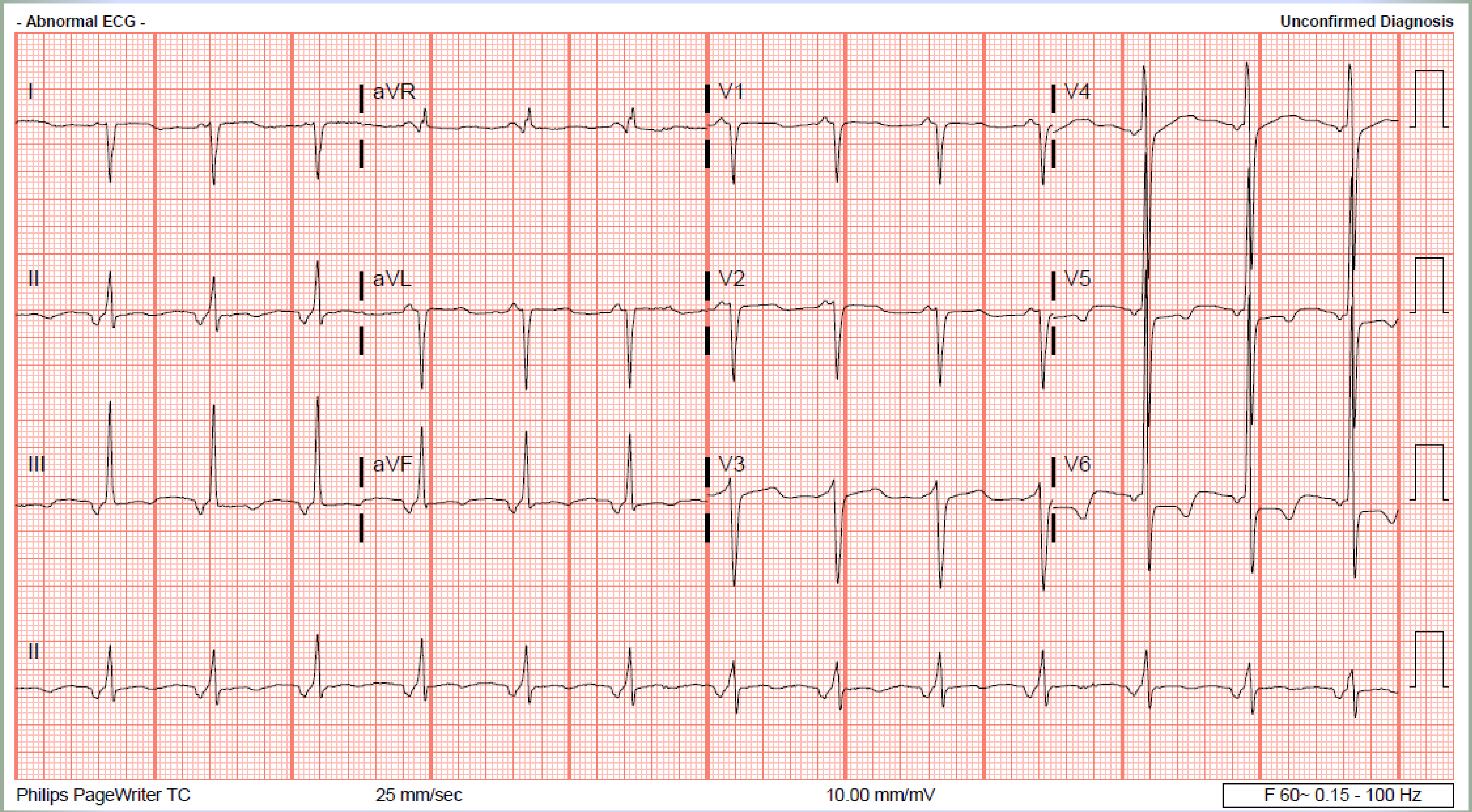



D.O.S.: TEST
Referred by:
Reviewed by:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

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

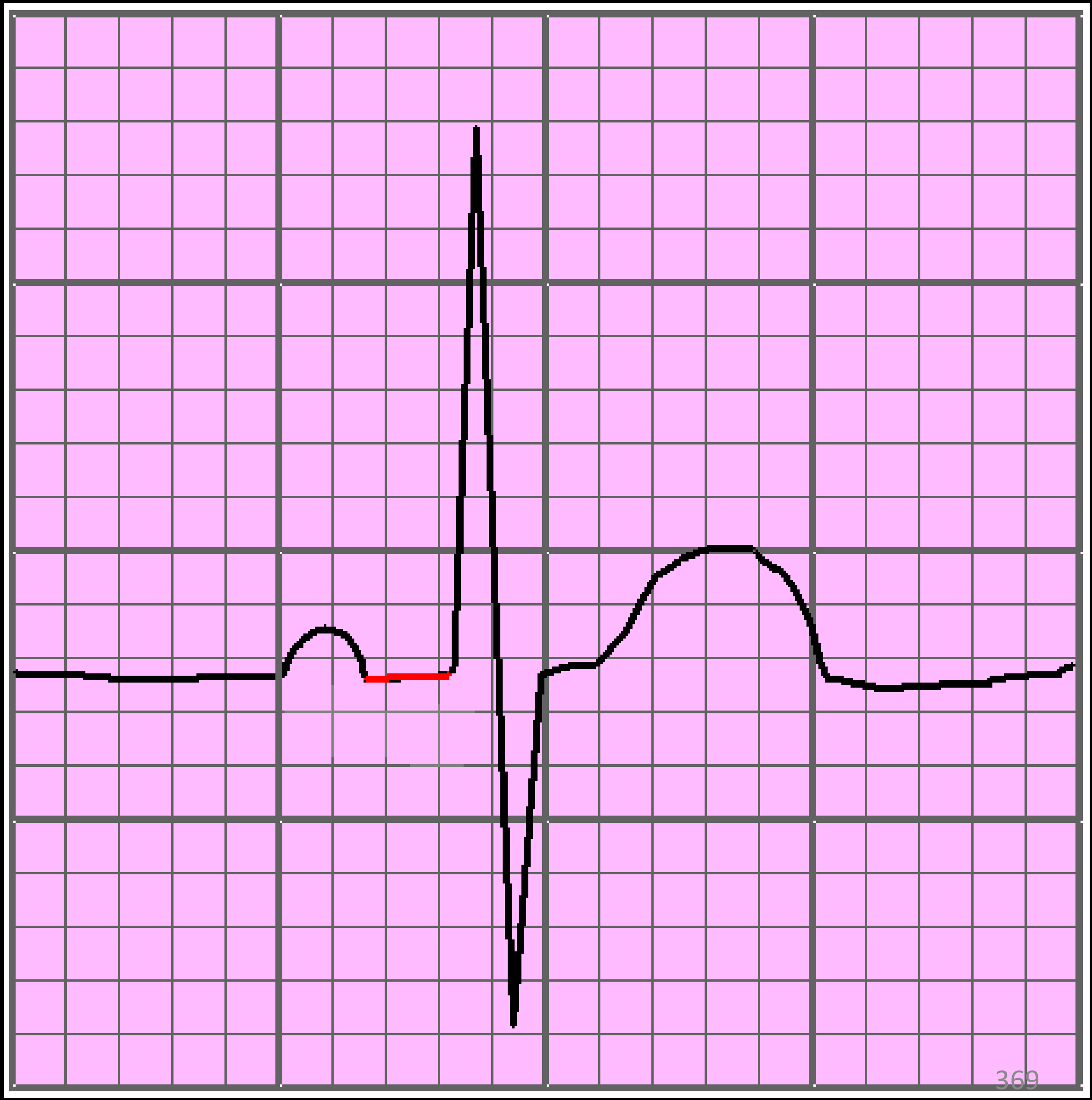
RETURN TO

THE

ISO-

ELECTRIC

LINE.



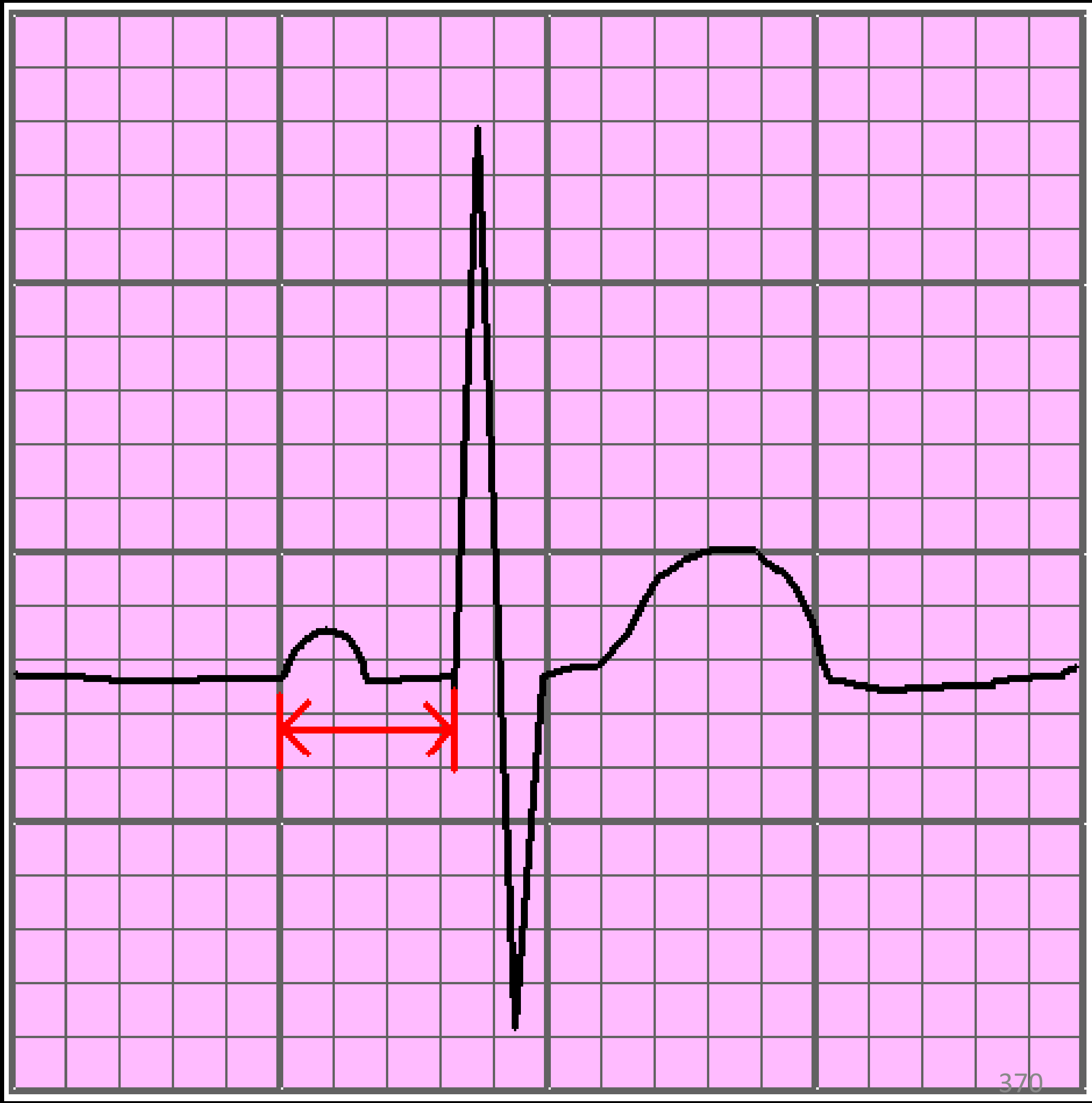
NORMAL P-R INTERVAL

.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

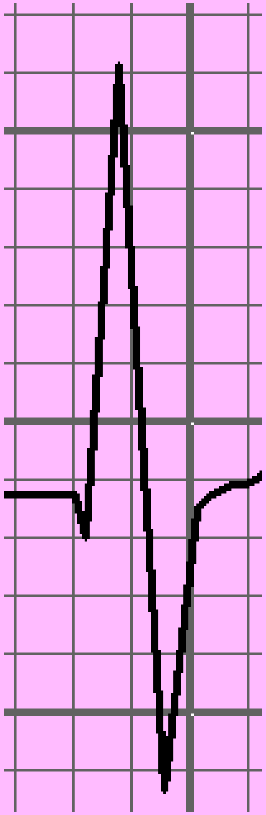
GREATER THAN 200 mSEC

THINK:

- HEART BLOCK

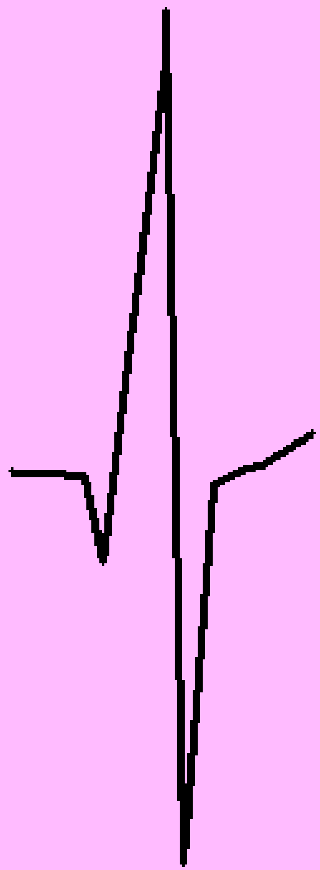
THE QRS COMPLEX

- 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

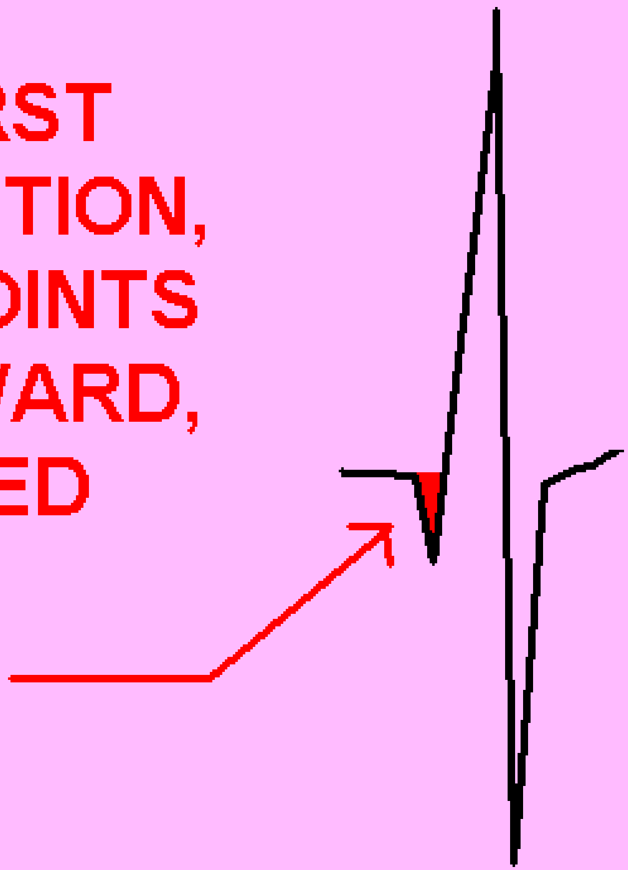
THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .



THE QRS COMPLEX

THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .

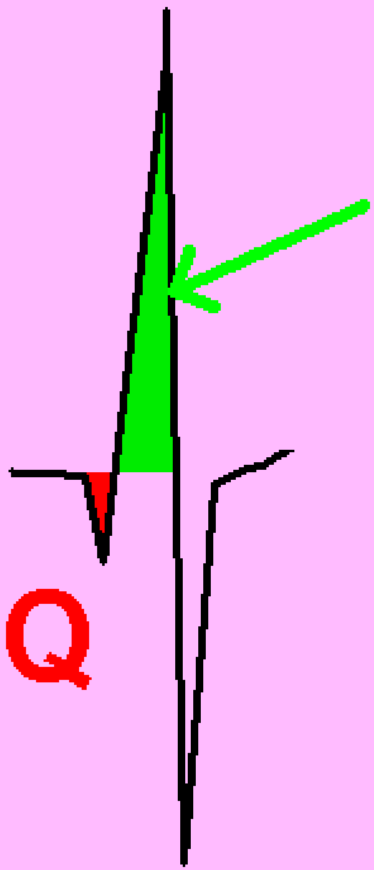
THE FIRST DEFLECTION, IF IT POINTS DOWNWARD, IS NAMED THE "Q WAVE"



THE QRS COMPLEX

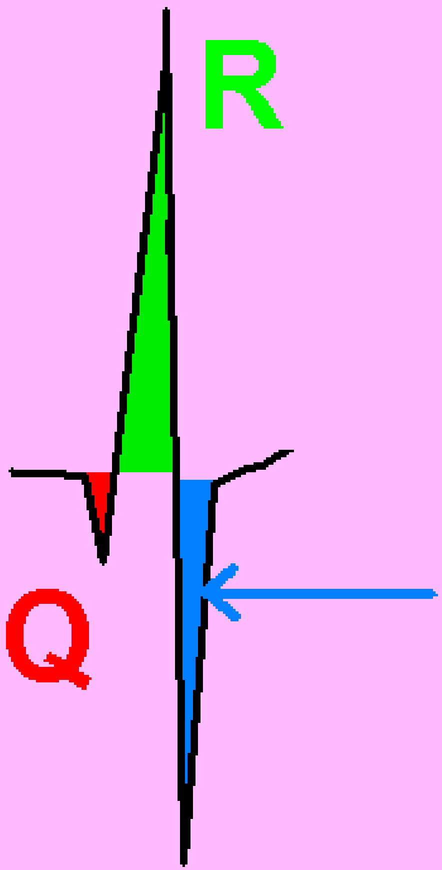
THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .

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



THE QRS COMPLEX

THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .

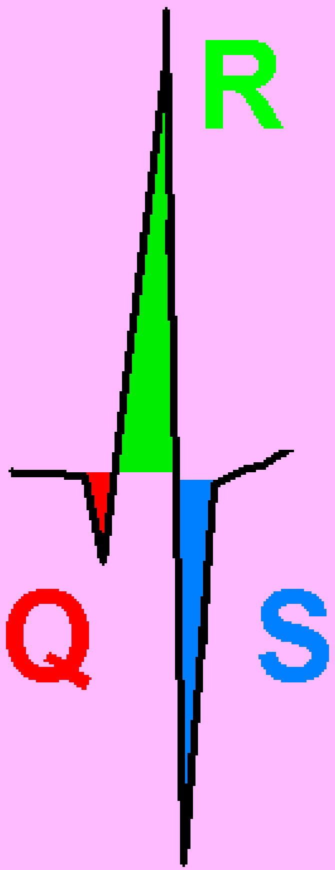


A NEGATIVE DEFLECTION AFTER THE R WAVE IS CALLED THE " S " WAVE

THE QRS COMPLEX

THIS QRS COMPLEX CONSISTS OF 3 DEFLECTIONS . . . .

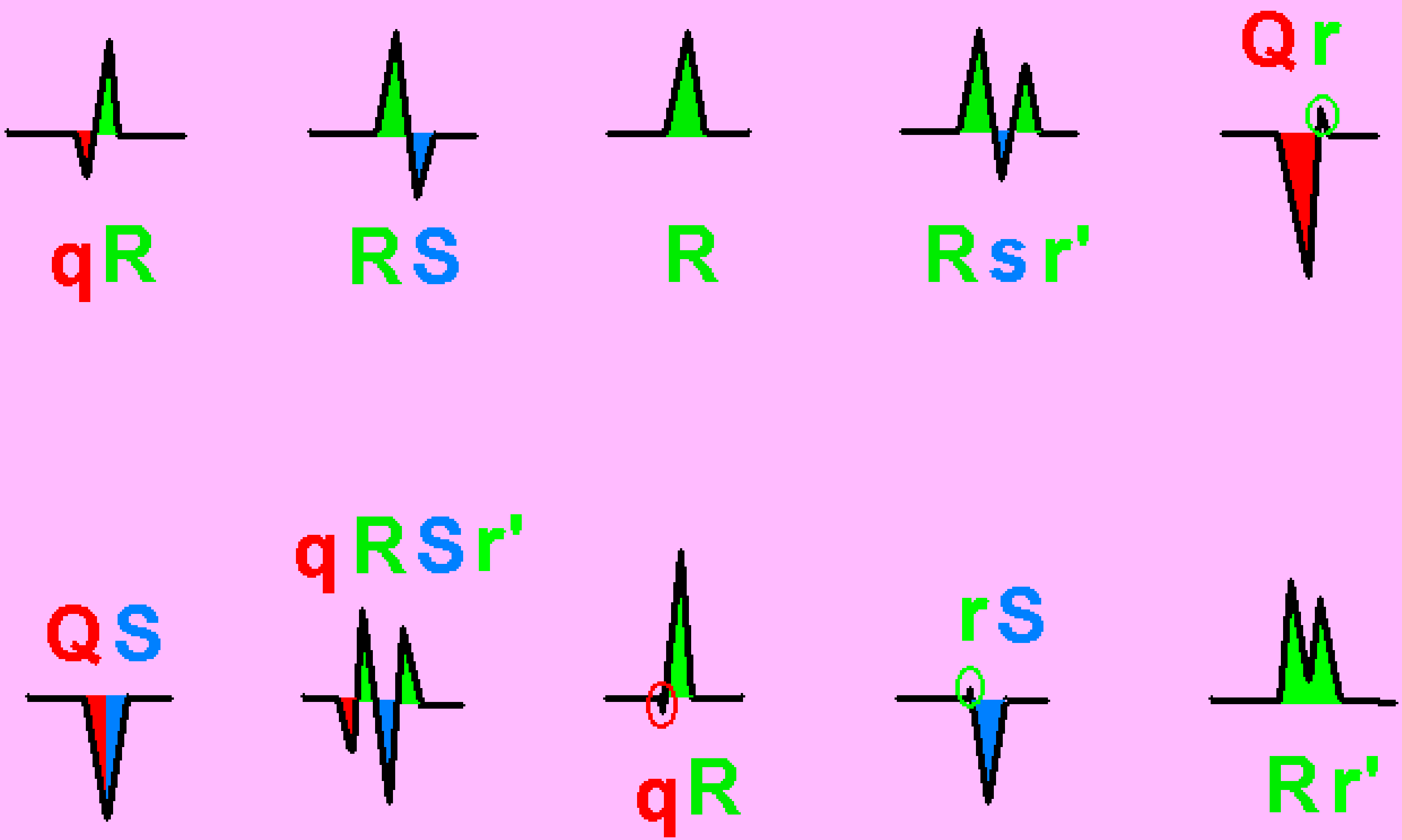
AND IS THE <u>ONLY</u> TRUE "QRS" COMPLEX



SOME OF THE OTHER VARIATIONS INCLUDE . . . .

THE QRS COMPLEX

WHAT ARE THESE COMPLEXES ??



QRS INTERVAL

LESS THAN

.12

OR

120 mSEC

QRS COMPLEX TOO WIDE

WIDER THAN 120 mSEC

THINK:

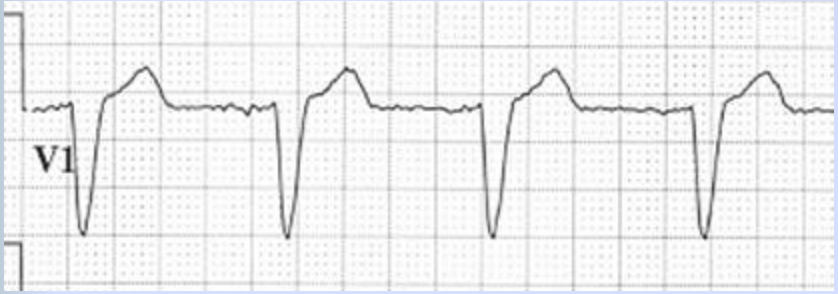
- BUNDLE BRANCH BLOCK
- VENTRICULAR COMPEX (ES)
- PACED RHYTHM
- L VENTRICULAR HYPERTROPHY
- ELECTROLYTE IMBAL. (↑K^+ ↓Ca^{++})
- DELTA WAVE (PRE-EXCITATION)

When the QRS is WIDE (> 3mm):

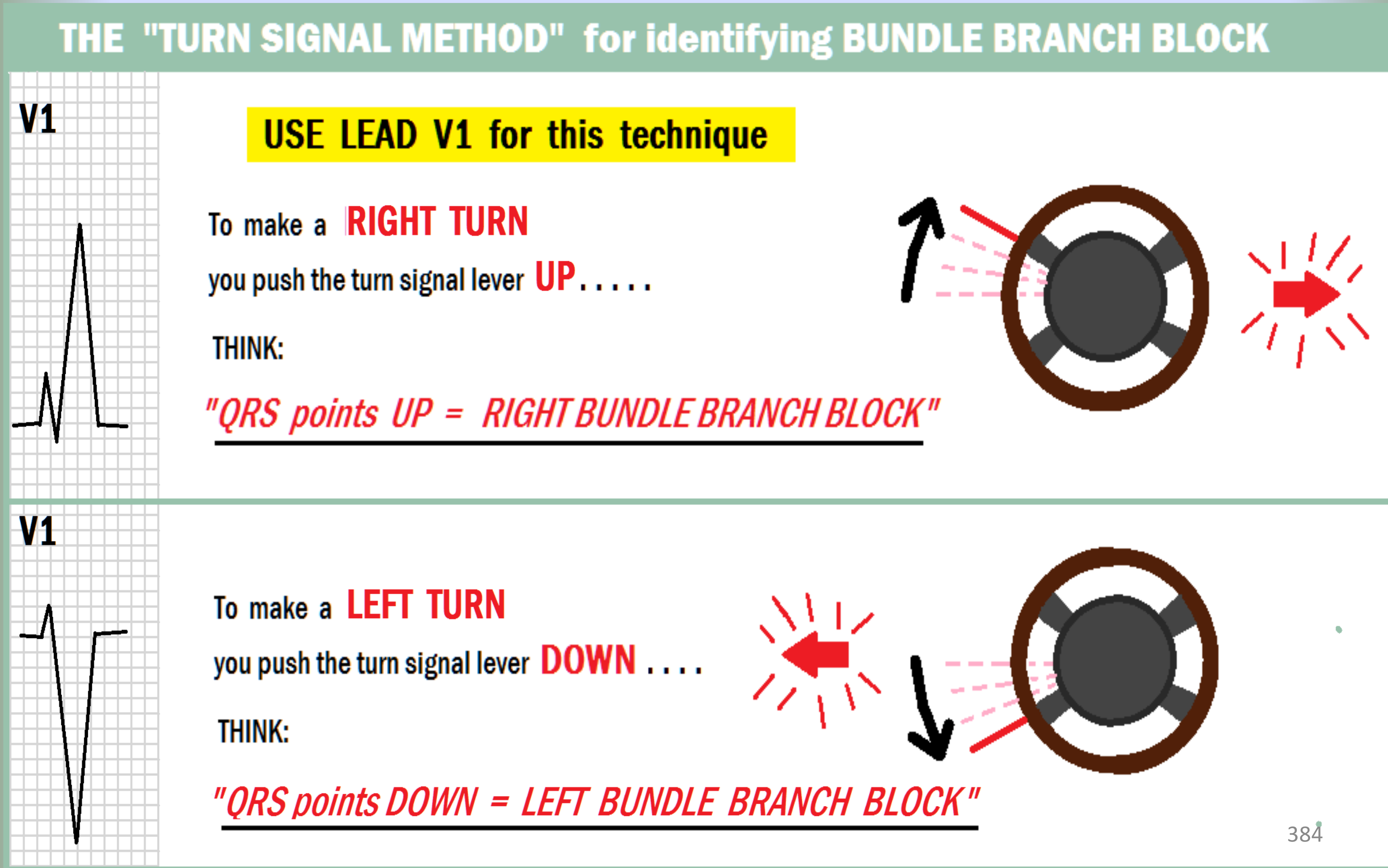
- **If you KNOW the Rhythm is originating ABOVE the Ventricles (such as NSR or any Supraventricular Rhythm) – you should determine if the QRS has a RIGHT or LEFT Bundle Branch Block morphology.**

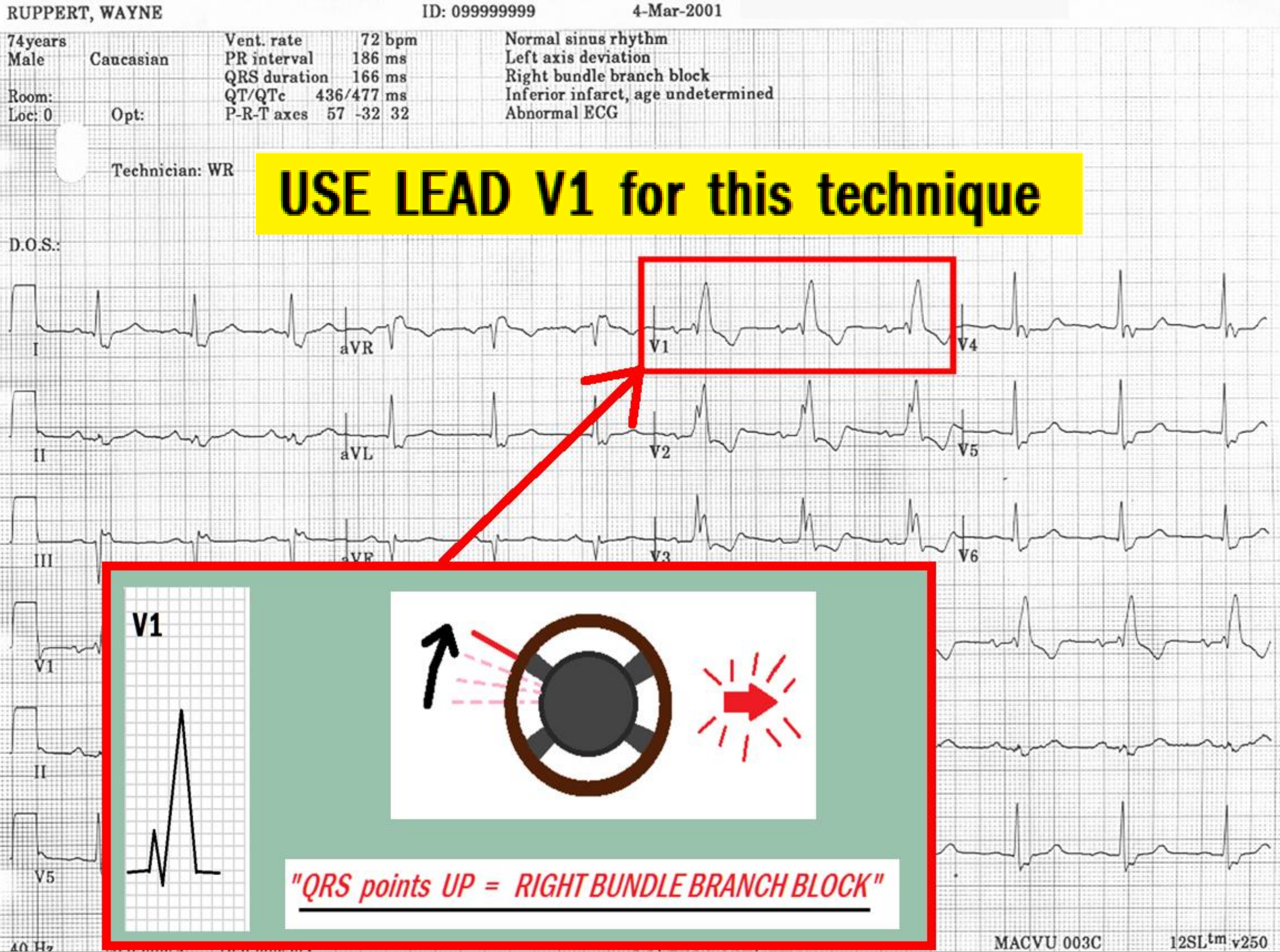
Normal Sinus and Other "Supraventricular Rhythms" with WIDE QRS (> 120 ms)

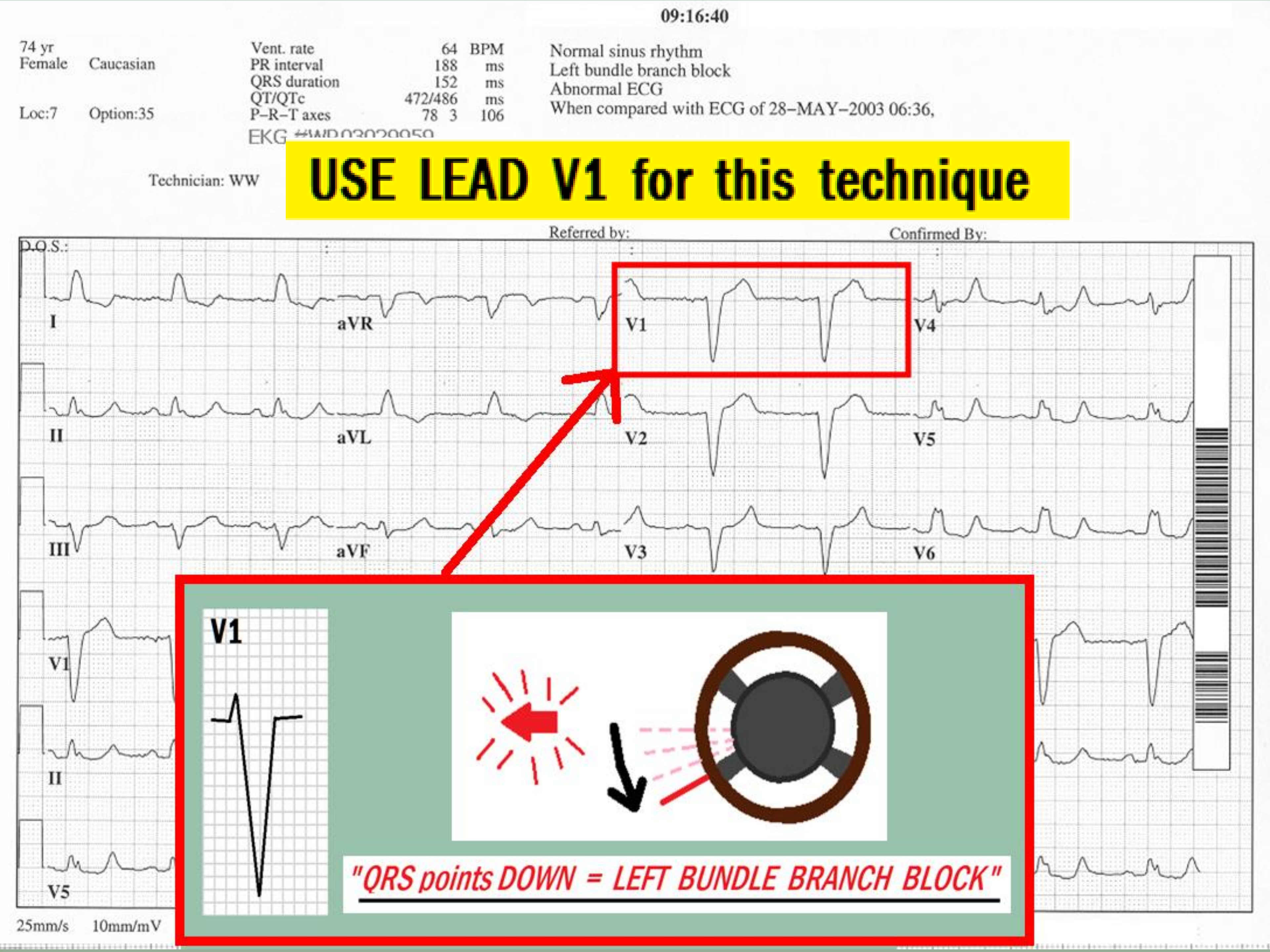
- **Determine LEFT vs. RIGHT Bundle Branch Block Pattern**



Simple “Turn Signal Method” . . .




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
V3
V6
V1
II
V5
V1
"QRS points UP = RIGHT BUNDLE BRANCH BLOCK"
MACVU 003C
12SLtm v250



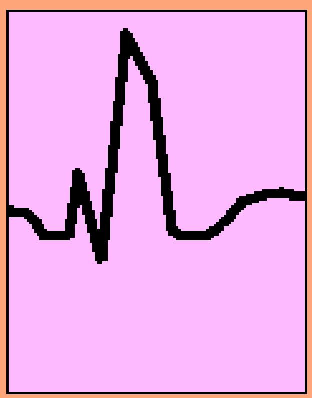
09:16:40
74 yr
Female Caucasian
Loc:7 Option:35
Vent. rate 64 BPM
PR interval 188 ms
QRS duration 152 ms
QT/QTc 472/486 ms
P-R-T axes 78 3 106
Normal sinus rhythm
Left bundle branch block
Abnormal ECG
When compared with ECG of 28-MAY-2003 06:36,
Technician: WW
USE LEAD V1 for this technique
Referred 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
V1
"QRS points DOWN = LEFT BUNDLE BRANCH BLOCK"

DIAGNOSING BUNDLE BRANCH BLOCK

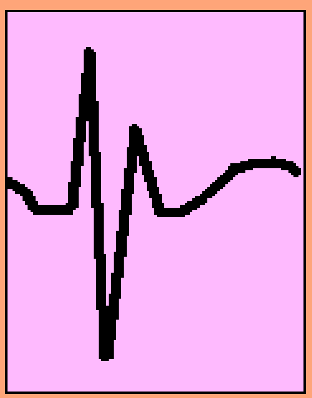
USING LEADS V1, V2, and V5, V6:

LOCATING RsR' or RR' COMPLEXES:

V1

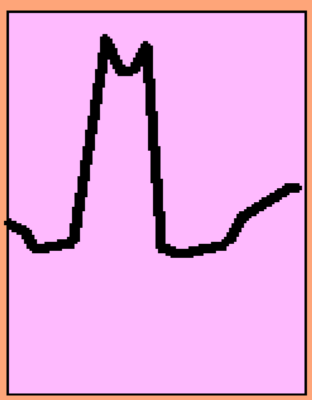


V2

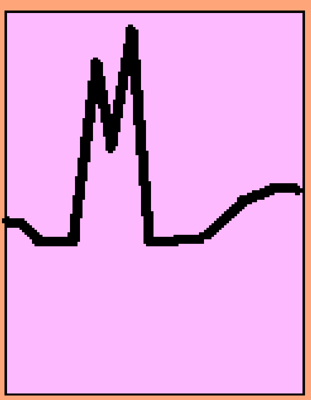


RIGHT BUNDLE BRANCH BLOCK

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