SEVEN RIVERS
REGIONAL MEDICAL CENTER

Hospital and Cardiovascular Center

Physician and Advanced Provider ACLS

Wayne W Ruppert, CVT,CCCC
Cardiovascular Coordinator

Speaker Bio

Wayne Ruppert:

-ACC Certified Cardiovascular Coordinator (2013-Present)

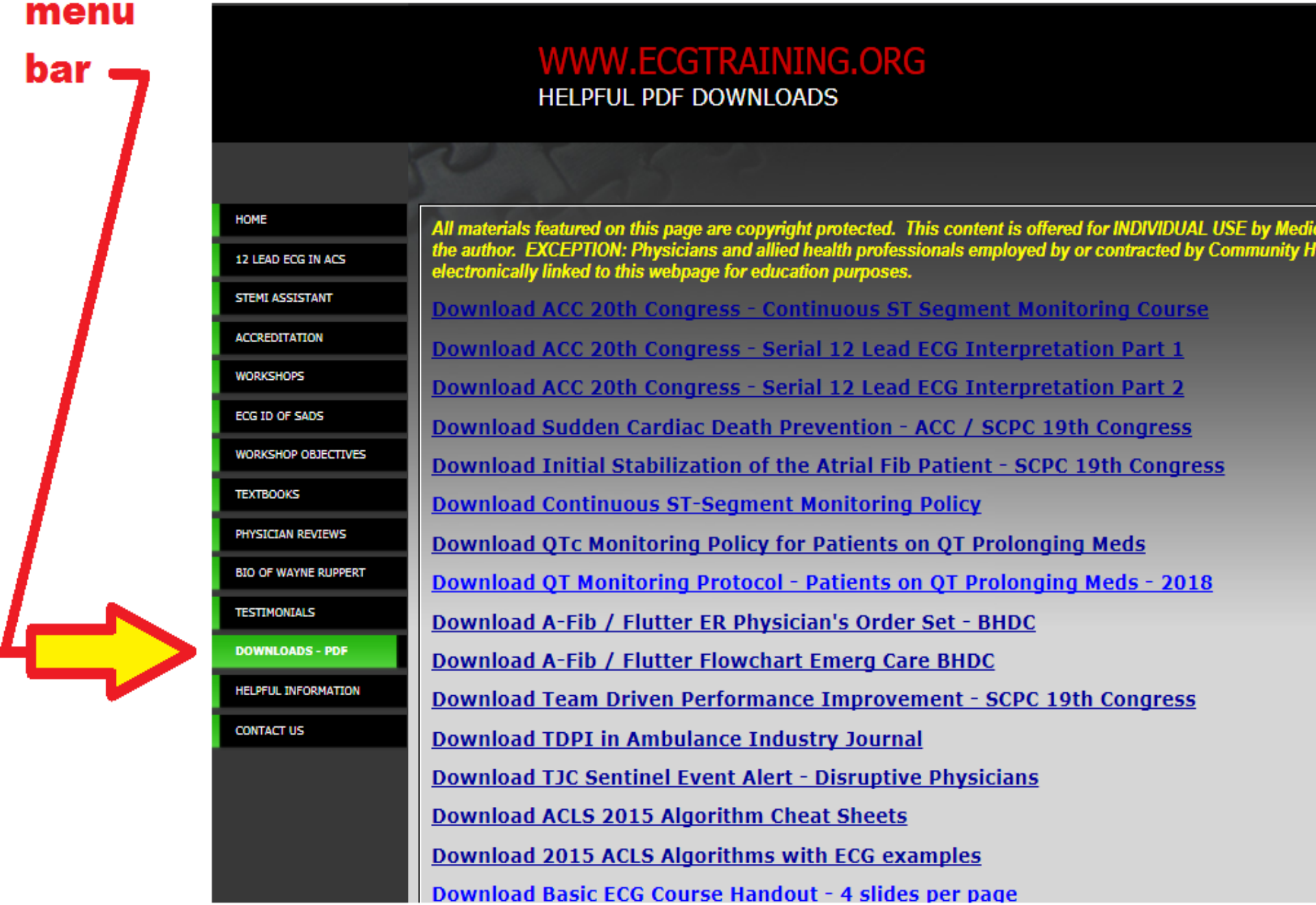
-ACC Conference Speaker 6 presentations 2016 & 2017

-AHA ACLS Instructor (1982 – Present)

-Interventional Cardiovascular and Electrophysiology Technologist (1996- 2013)

-Paramedic (1980 – 1996)

Go to ECGtraining.org then select DOWNLOADS PDF from menu bar



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Download Physician’s Advanced ACLS Course SRRMC 2018

Objectives:

- Review ESSENTIALS of ACLS
- Provide review of review of relevant, critical cardiovascular issues in contemporary practice which fulfill ACC CPC and AF Accreditation Requirements
- Provide ACLS Written and Practical Exams.

Future: Provide up to 4 AMA Category 1 CMEs.

ELECTRICAL THERAPY

	BiPHASIC	MONOPHASIC
SYNCHRONIZED CARDIOVERSION:		
NARROW SVT / REGULAR RHYTHM:	50 - 100j	200j
NARROW QRS, IRREG RHYTHM:	120 - 200j	200j
WIDE QRS / MONOPHASIC / REG:	100j	
DEFIB (unsynchronized):		
WIDE QRS, IRREGULAR: (TORSADES / POLYMORPHIC VT)	DEFIB 120 - 200j	360j
V-FIB / PULSELESS VT:	120 - 200 j subsequent doses may be equivalent or escalated>	360j

If Initial Shock Not Successful:

- Consider repositioning pads
  - Anterior / Posterior placement
  - Change polarity (reverse pad locations)
- Increase energy (joules)
- If monophasic current delivery used, try biphasic
- Apply pressure to anterior pad
- Administer meds to lower defibrillation threshold, then repeat defibrillation.

"There is NO SUCH thing as an EP (heart rate) emergency . . .

If the rate's too slow -- PACE IT

If the rate's too fast -- SHOCK IT !"

Dr. James Irwin
Electrophysiologist
St. Joseph's Hospital
Tampa, Florida

" There is NO SUCH thing as an EP (heart rate) emergency . . .

If the rate's too slow -- PACE IT

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. . . and call me in the MORNING.

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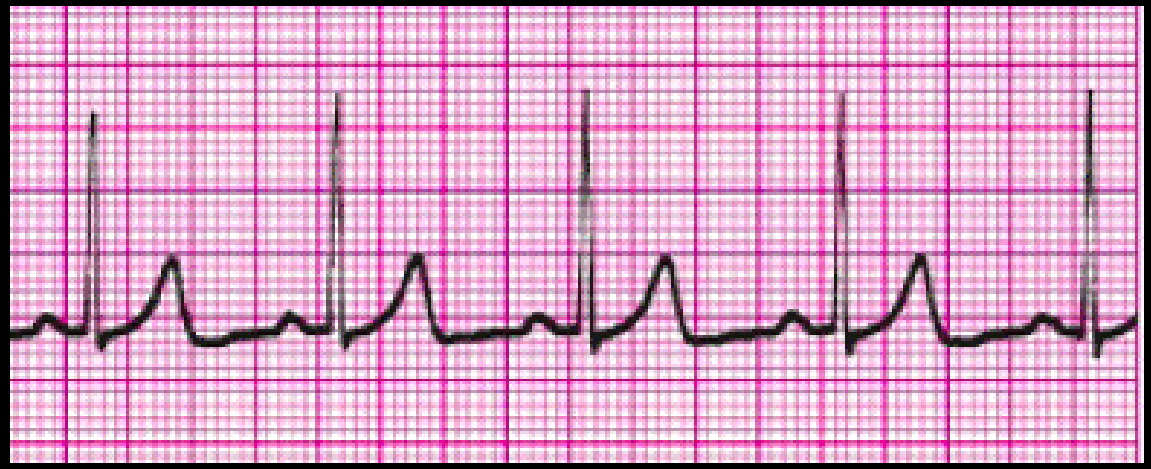
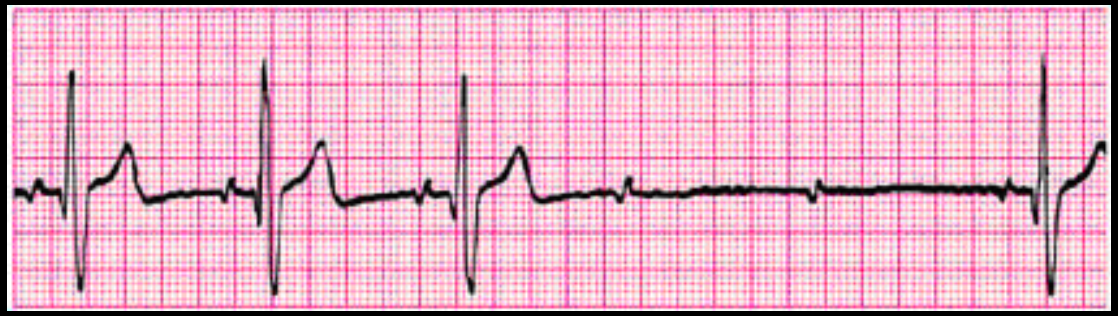
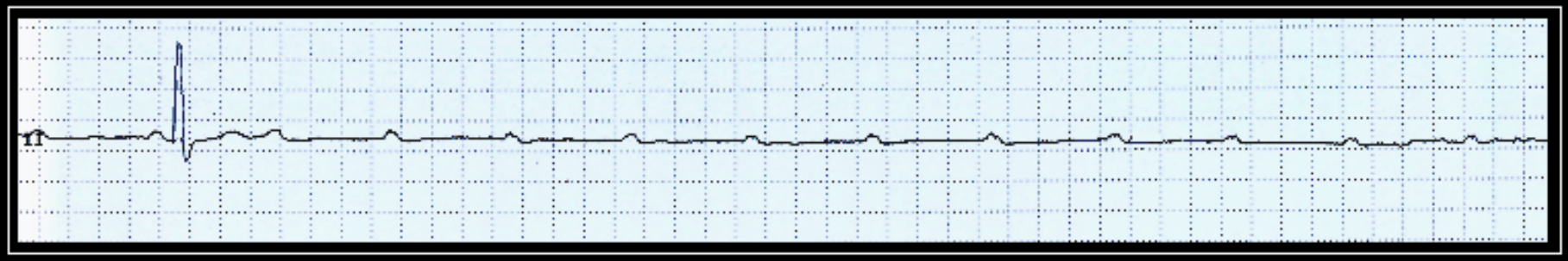




OR

?

ADAMS - STOKES SYNDROME



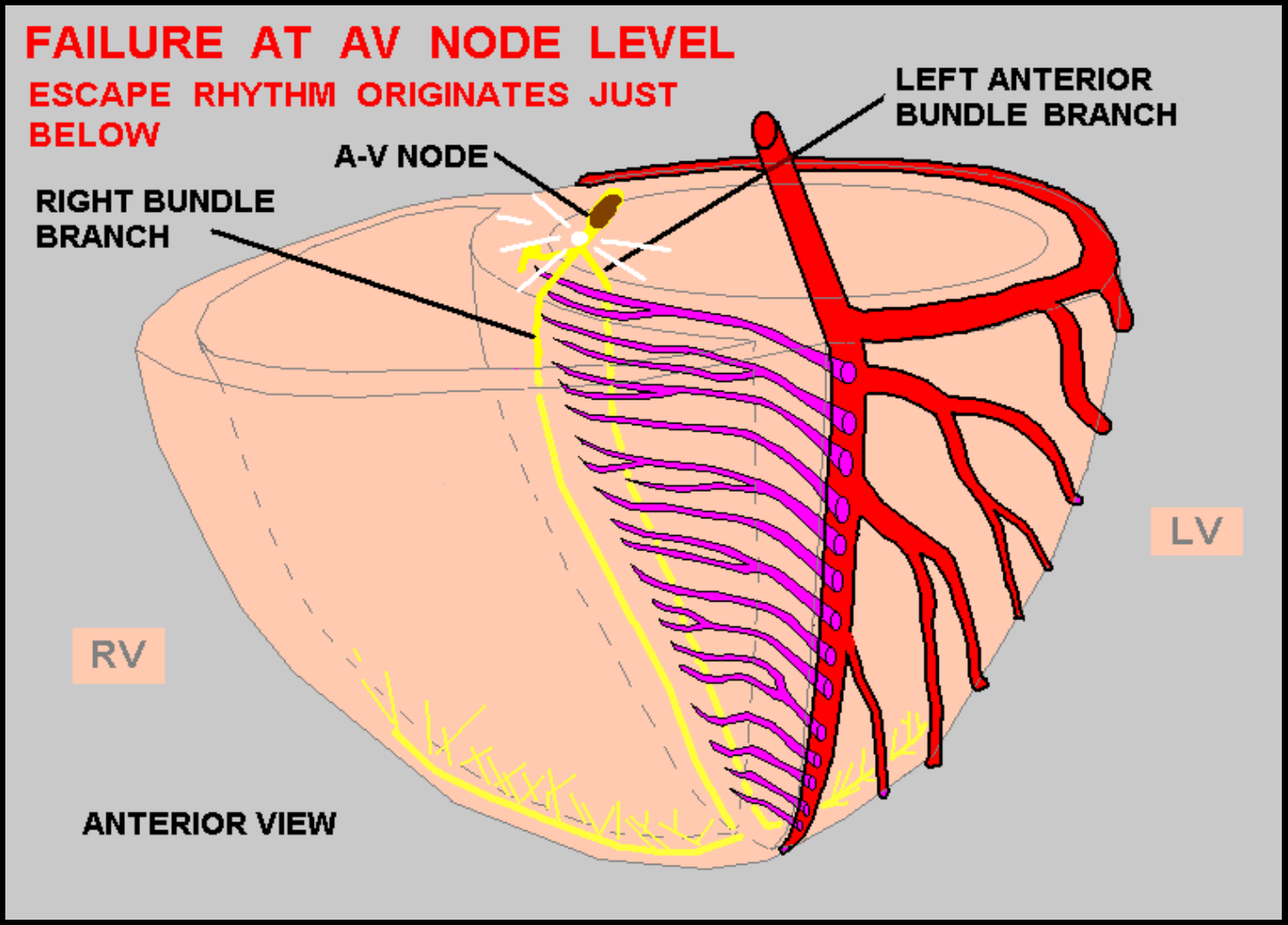
CASE HISTORY:

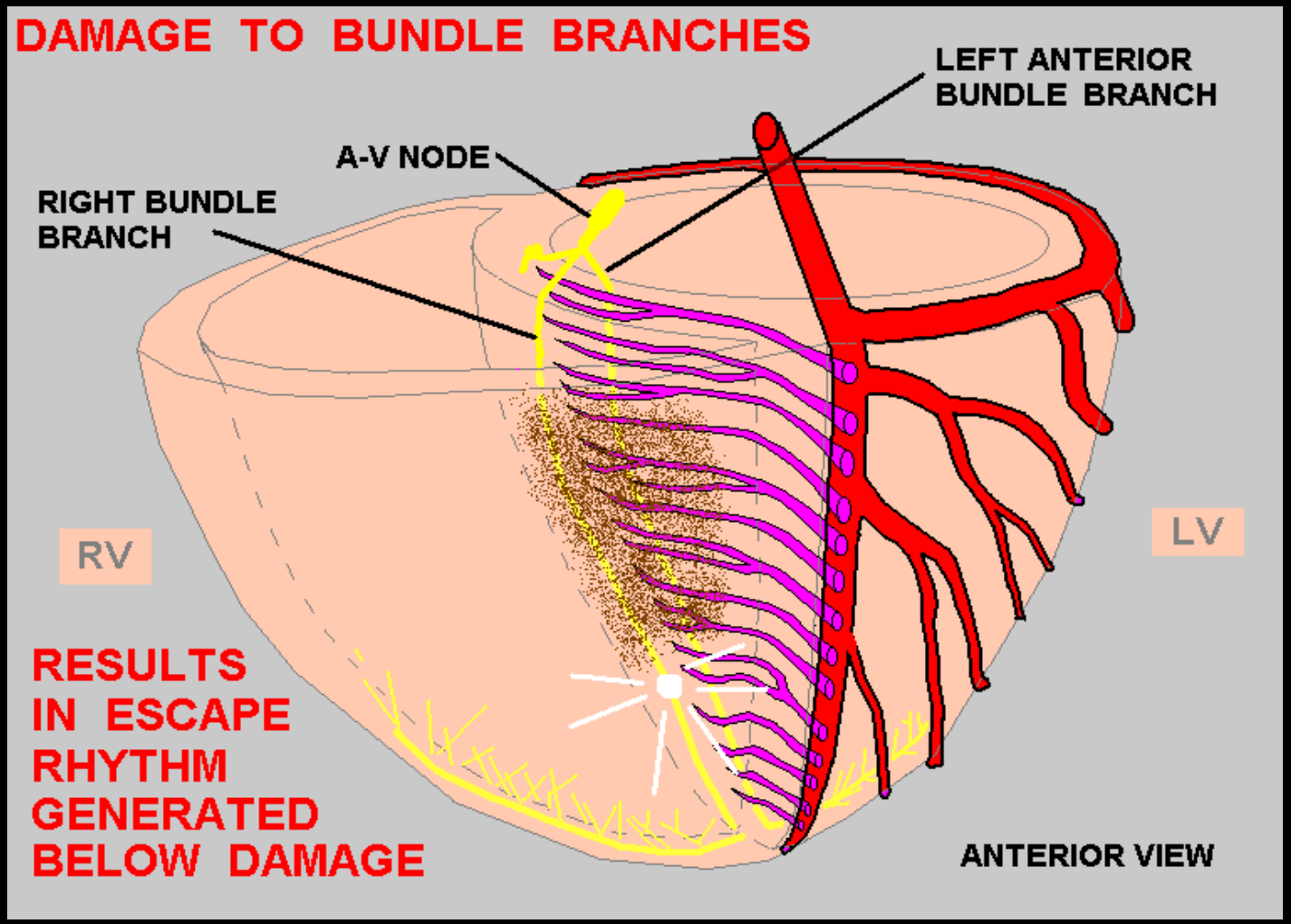
72 y/o male with history of SYNCOPE OF UNKOWN ORIGIN. While undergoing Cardiac Catherization (Left Heart Cath), pt went from NSR rate 76 - 80 to **2nd o TYPE II HEART BLOCK,** which quickly deteriorated into **VENTRICULAR STANDSTILL .**

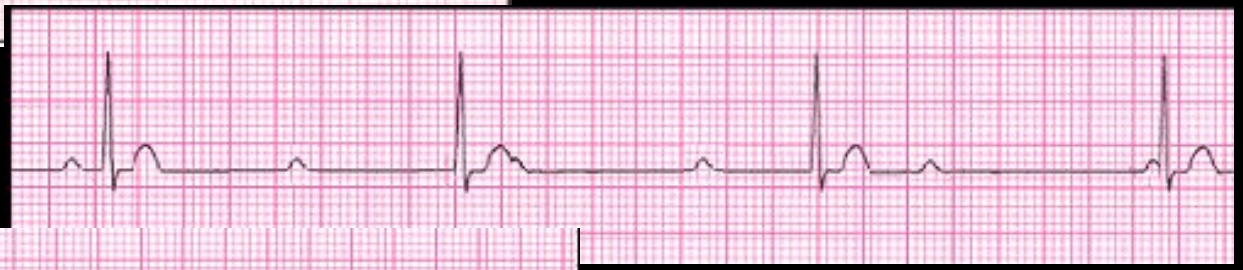
TX: CPR, Atropine, Transvenous Pacemaker, followed by Permanent Pacemaker Implanation. Patient experienced full recovery, was discharged.

FAILURE AT AV NODE LEVEL

ESCAPE RHYTHM ORIGINATES JUST BELOW




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



COMMON Rx !

- SYMPTOMATIC BRADYCARDIAS
- HEART BLOCKS with SLOW VENTRICULAR RATES
  (patient symptomatic)

Tx:

- ✓ ABC s
- ✓ GENERAL SUPPORTIVE CARE
- ✓ BRADYCARDIA ALGORITHM

As per ACC/AHA Guidelines:

- **2013 STEMI**
- **2014 NSTE-ACS**

Refrain from administering Oxygen to ACS / suspected ACS patients unless SAO2 <90 %, or patient exhibits signs of hypoxemia or respiratory distress.

SYMPTOMATIC BRADYCARDIA

- **ABC s + GENERAL SUPPORTIVE CARE**

- **ATROPINE 0.5 mg. IV**
  - MAY REPEAT 0.5 mg. DOSES IF NEEDED
  - MAXIMUM 3.0 mg.

- **TRANSCUTANEOUS PACEMAKER**
  - PREFERRED PRIMARY Tx FOR HIGH GRADE A-V BLOCK

SYMPTOMATIC BRADYCARDIA

- **DOPAMINE gtt.**
  2 - 10 mcg / kg. / min. INFUSION RATE
  IF PACING NOT AVAILABLE or EFFECTIVE

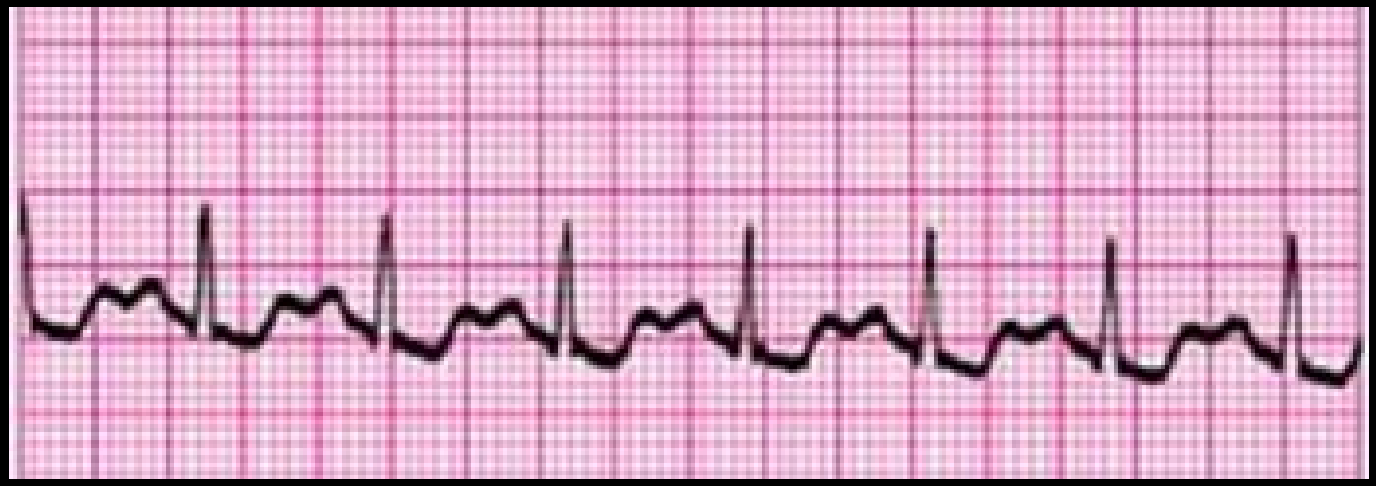
- **EPINEPHRINE gtt.**
  2 - 10 mcg / min INFUSION RATE
  IF PACING NOT AVAILABLE or EFFECTIVE

- **TRANSVENOUS PACEMAKER**

CAUSES of HEART BLOCK

☞ THINK:

- ISCHEMIA / INFARCTION
  - AV NODE — INFERIOR WALL (RCA or CIRCUMFLEX LESIONS)
  - HIS / BUNDLE BRANCHES — ANTERIOR WALL (LAD LESIONS)
- ⊖ DROMOTROPIC MEDS — DIGITALIS / BETA & Ca++ CH BLOCKERS
- INFILTRATIVE DISEASE — AMYLOIDOSIS / HEMOCHROMATOSIS
- INFLAMMATORY DISEASE — PERICARDITIS / MYOCARDITIS / RHEUMATIC DISORDERS
- LEV'S DISEASE — LENEGRE'S SYNDROME
- AORTIC / MITRAL ANNULAR CALCIFICATION



ACLS TACHYCARDIA GUIDELINES

150

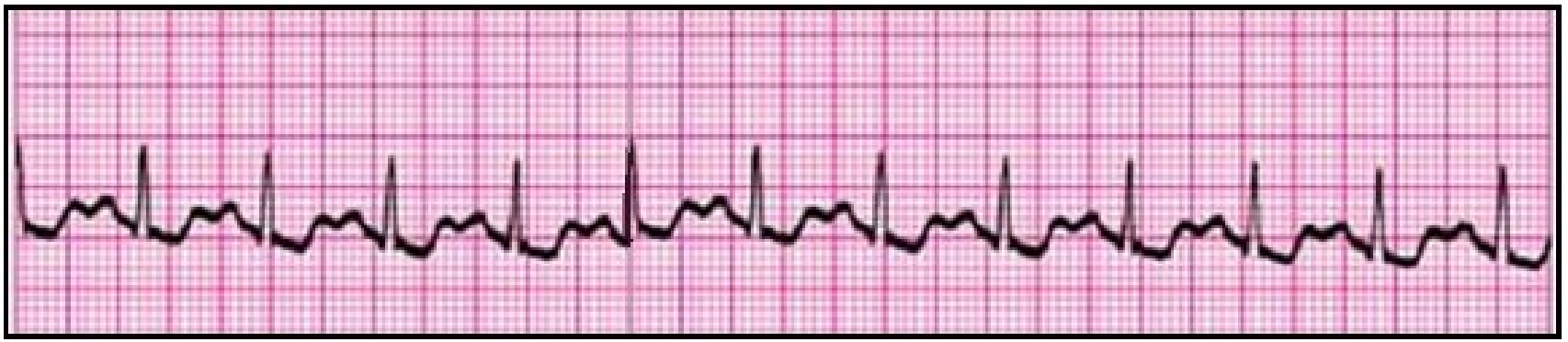
100

SVT

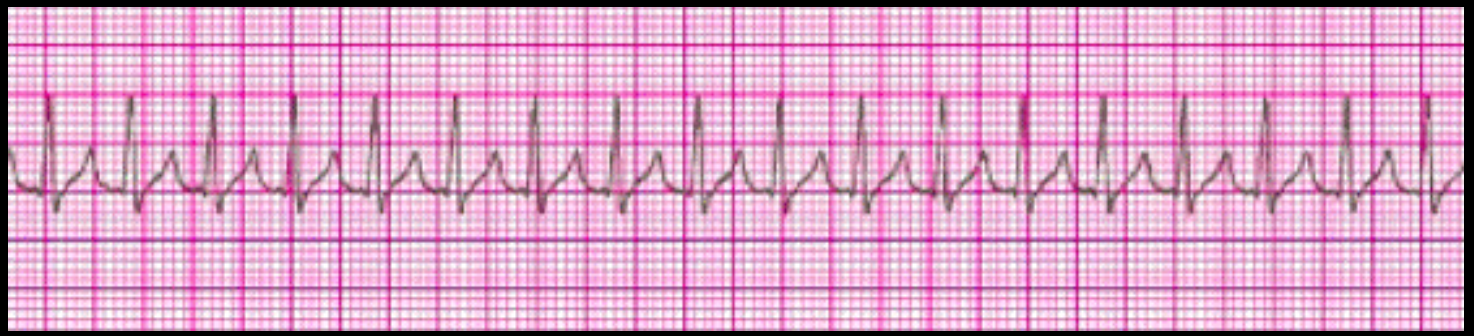
SINUS TACH

NSR

THIS RHYTHM IS: SINUS TACHYCARDIA



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



SVT - UNSTABLE PATIENT (NARROW QRS)

ABC s + GENERAL SUPPORTIVE CARE

(OXYGEN, ECG / VS / SAO2 MONITORING, IV ACCESS)

IMMEDIATE SYNCHRONIZED CARDIOVERSION

- CONSIDER SEDATION

—— ADENOSINE - IF IT DOES NOT DELAY CARDIOVERSION !

- SYNCHRONIZED CARDIOVERSION

REGULAR RHYTHM:
50 - 100 j biphasic

IRREGULAR RHYTHM:
100 - 200 j biphasic

------- monophasic = 200 j ---------

SVT - STABLE PATIENT (NARROW QRS)

ABCs + GENERAL SUPPORTIVE CARE

REGULAR RHYTHM

- VAGAL MANEUVERS
- ADENOSINE 6 mg / 12 mg

IRREGULAR RHYTHM

POSSIBLE ATRIAL FIB or
MULTIFOCAL ATRIAL TACH

- BETA BLOCKERS
- CALCIUM CHANNEL BLOCKER
- TREAT UNDERLYING CAUSE (THE Hs and Ts)
- " EXPERT CONSULTATION "

THE " H's " and the " T's "

- HYPOVOLEMIA
- HYPOXIA
- HYDROGEN ION (Ph)
- HYPOGLYCEMIA
- HYPOTHERMIA

- TOXINS
- TAMPONADE (CARDIAC)
- TENSION PNEUMOTHORAX
- THROMBOSIS (CORONARY or PULMONARY)
- TRAUMA

APR–2004 ST. JOSEPH'S HOSPITAL

55 yr
Male Caucasian

Loc:3 Option:23

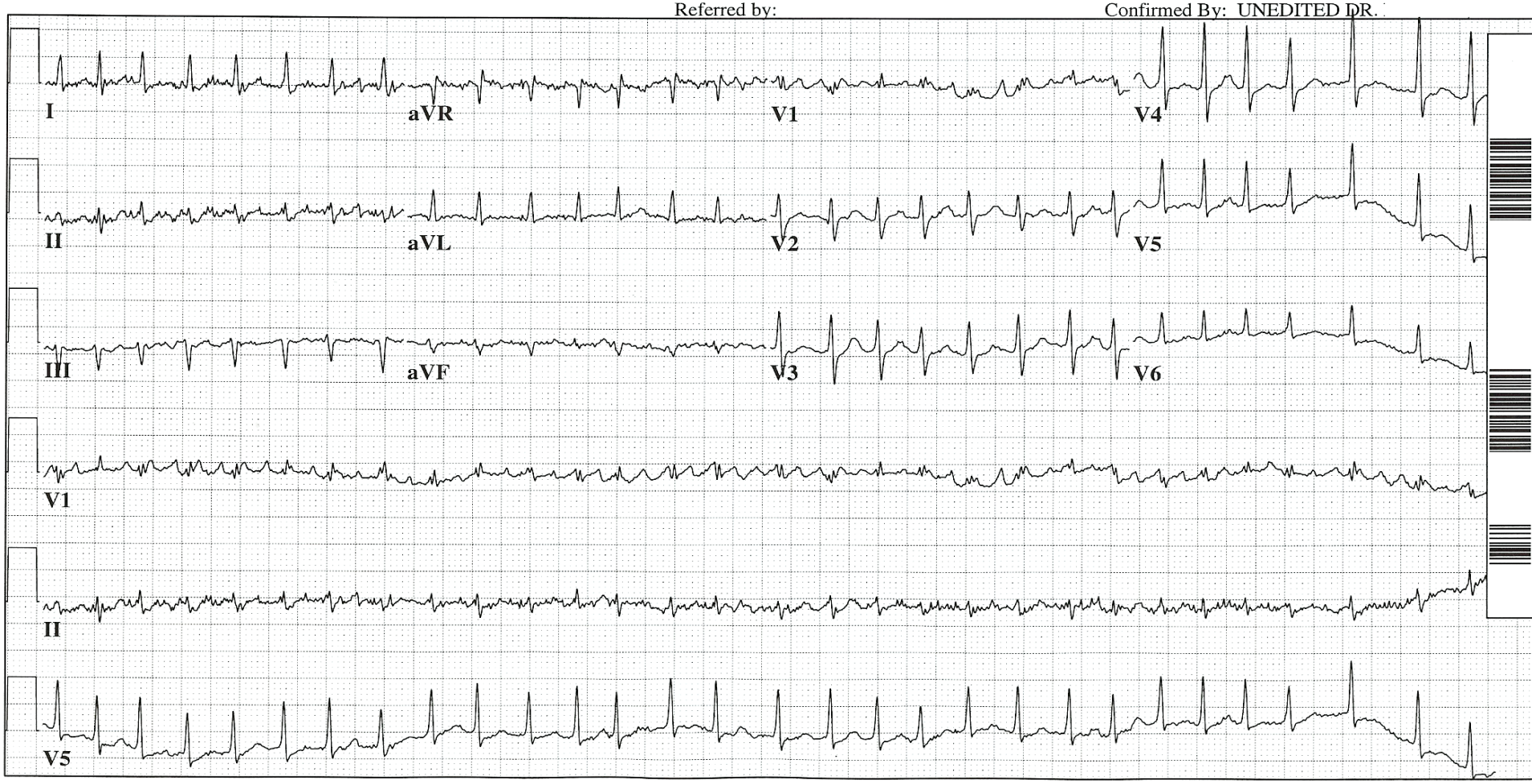
Vent. rate	178	BPM
PR interval	*	ms
QRS duration	90	ms
QT/QTc	264/454	ms
P–R–T axes	* –19	46

Technician:

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

Atrial fibrillation with rapid ventricular response
with premature ventricular or aberrantly conducted complexes
Nonspecific ST abnormality , probably digitalis effect
Abnormal ECG
When compared with ECG of 30–JUL–1998 15:14,
Atrial fibrillation has replaced Sinus rhythm
Vent. rate has increased BY 109 BPM ...

Referred by: Confirmed By: UNEDITED DR.



25mm/s 10mm/mV 40Hz 005C 12SL 235 CID: 2

EID:10 EDT:

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Post Emergency Cardioversion;

Anticoagulation Strategy:

- **AF/AFL Duration <48 hours, but patient has high risk of stroke (CHA_2DS_2-VASc Stroke Risk Score 2 or more):**
- **AF/AFL Duration 48 hours or more (all patients):**

anticoagulation should be initiated as soon as possible and continued for at least 4 weeks after cardioversion unless contraindicated.

CLASS I, LOE C

SVT - STABLE PATIENT (NARROW QRS)

ABC s + GENERAL SUPPORTIVE CARE

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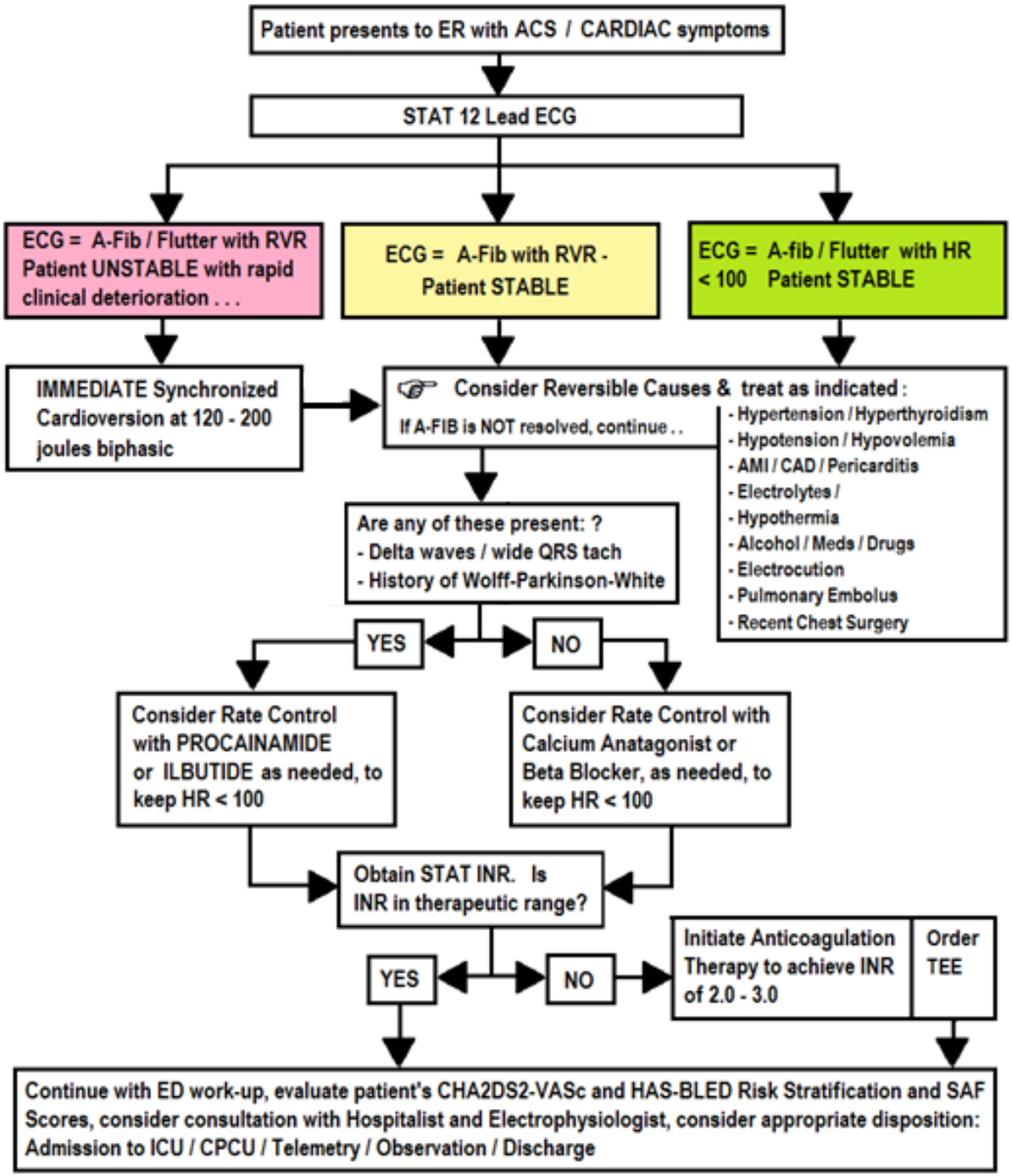
Patient presents to ER with ACS / CARDIAC symptoms
STAT 12 Lead ECG
ECG = A-Fib / Flutter with RVR Patient UNSTABLE with rapid clinical deterioration . . .
ECG = A-Fib with RVR - Patient STABLE
ECG = A-fib / Flutter with HR < 100 Patient STABLE
IMMEDIATE Synchronized Cardioversion at 120 - 200 joules biphasic
Consider Reversible Causes & treat as indicated :
If A-FIB is NOT resolved, continue . .
- Hypertension / Hyperthyroidism
- Hypotension / Hypovolemia
- AMI / CAD / Pericarditis
- Electrolytes /
- Hypothermia
- Alcohol / Meds / Drugs
- Electrocution
- Pulmonary Embolus
- Recent Chest Surgery
Are any of these present: ?
- Delta waves / wide QRS tach
- History of Wolff-Parkinson-White
YES
NO
Consider Rate Control with PROCAINAMIDE or ILBUTIDE as needed, to keep HR < 100
Consider Rate Control with Calcium Anatagonist or Beta Blocker, as needed, to keep HR < 100
Obtain STAT INR. Is INR in therapeutic range?
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Order TEE
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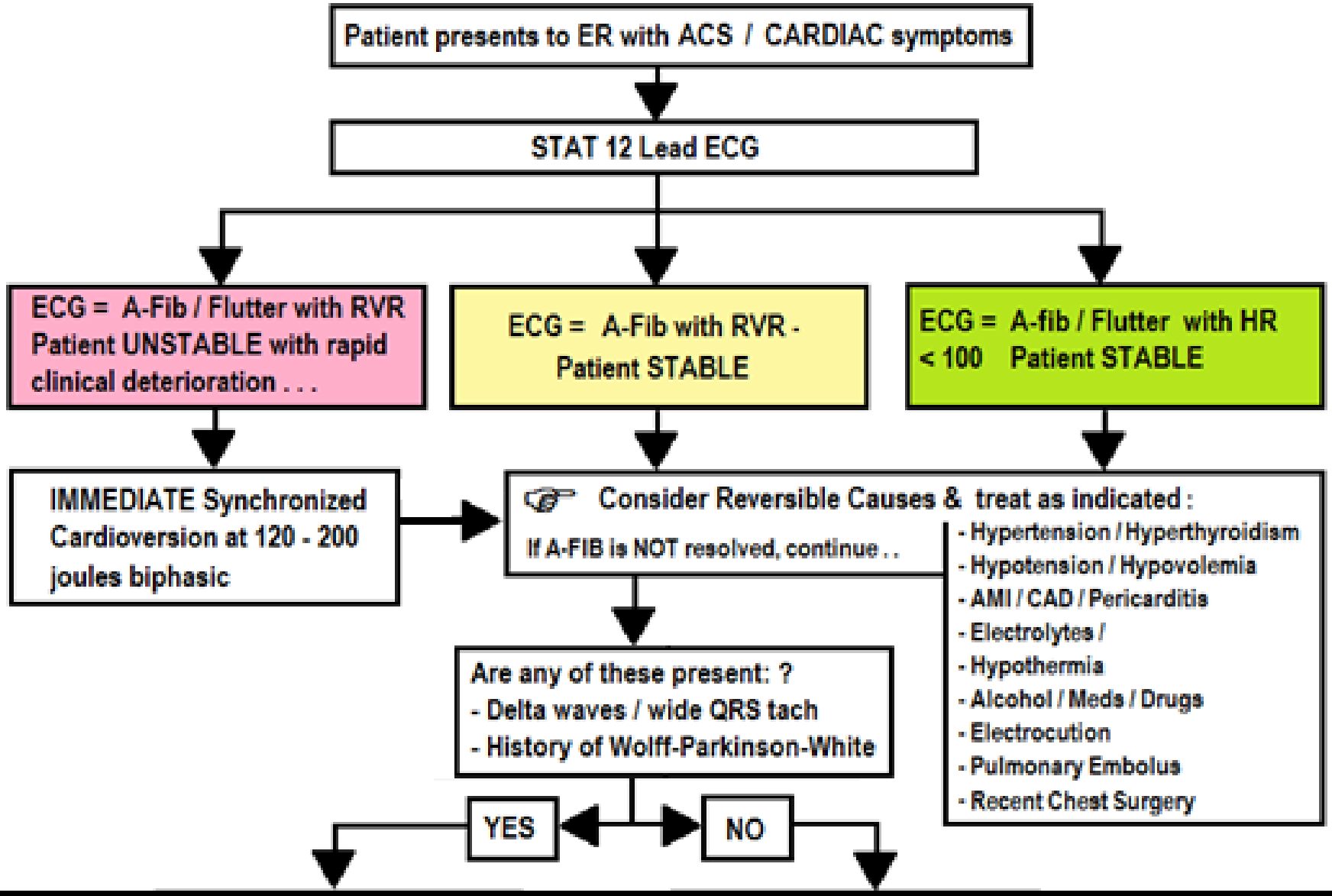
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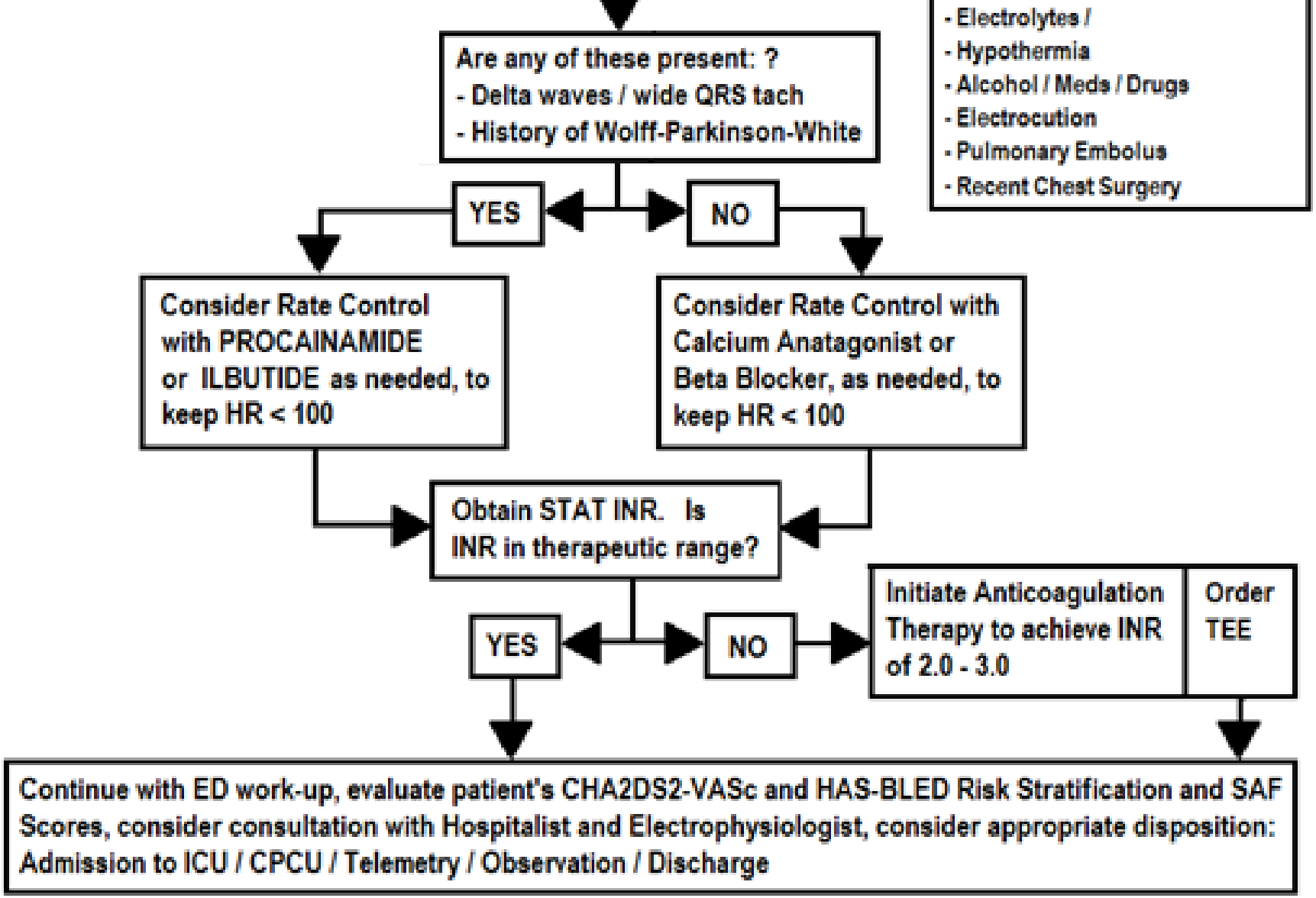
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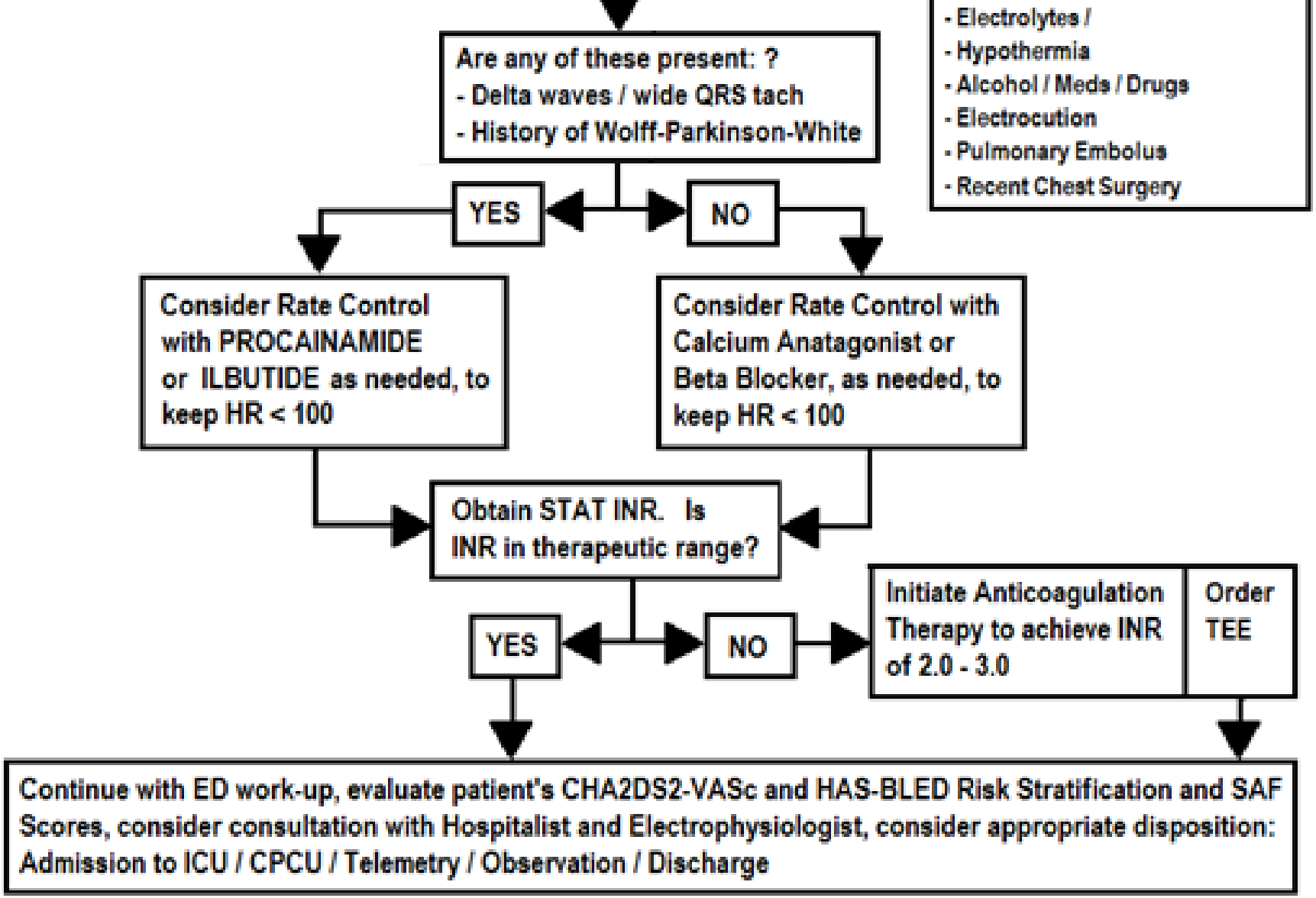


Pay attention to "Wide vs. Narrow" QRS Complexes . . .

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%

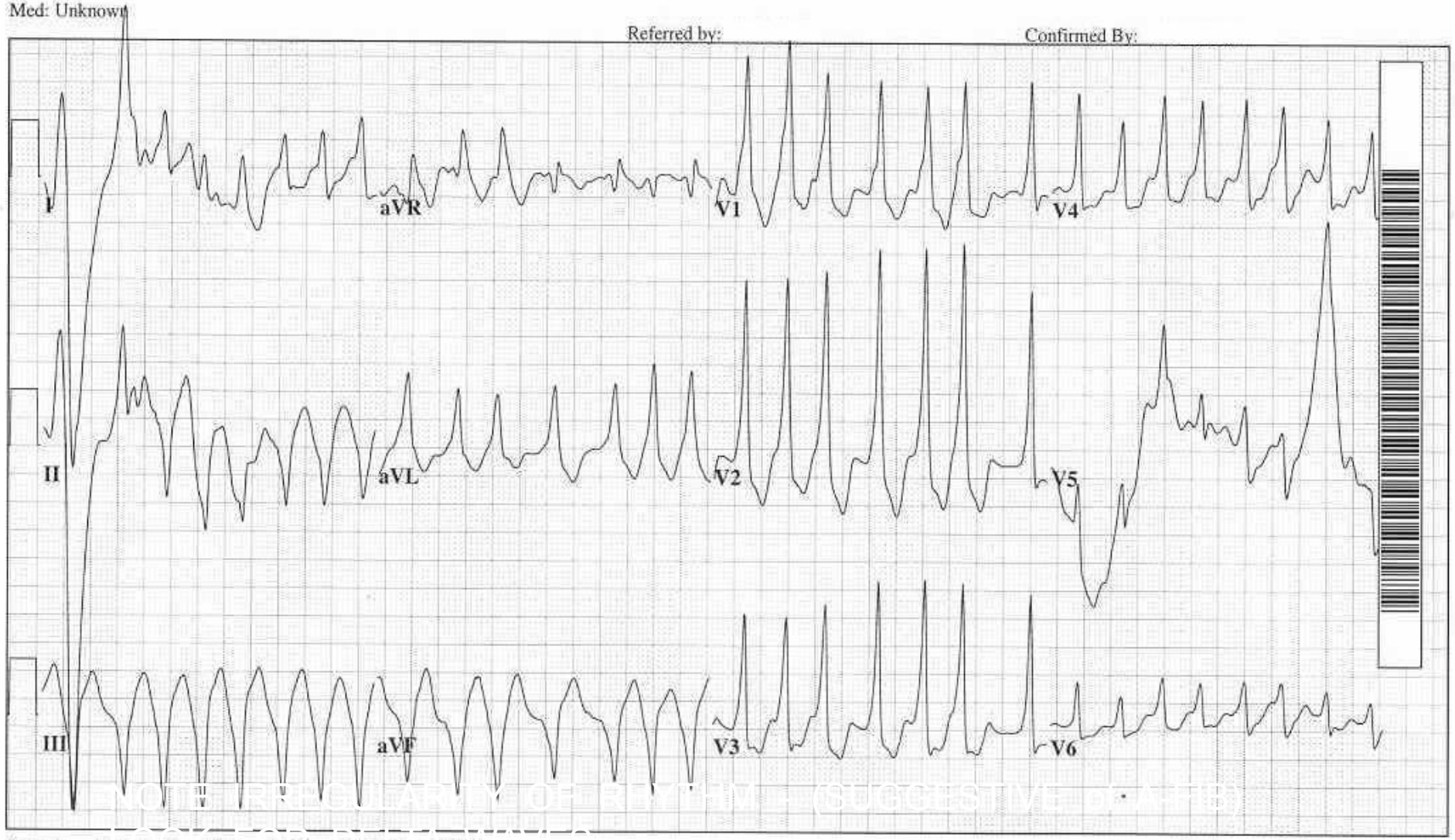
ST. JOSEPH'S HOSPITAL-

37 yr		Vent. rate	180	BPM	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:



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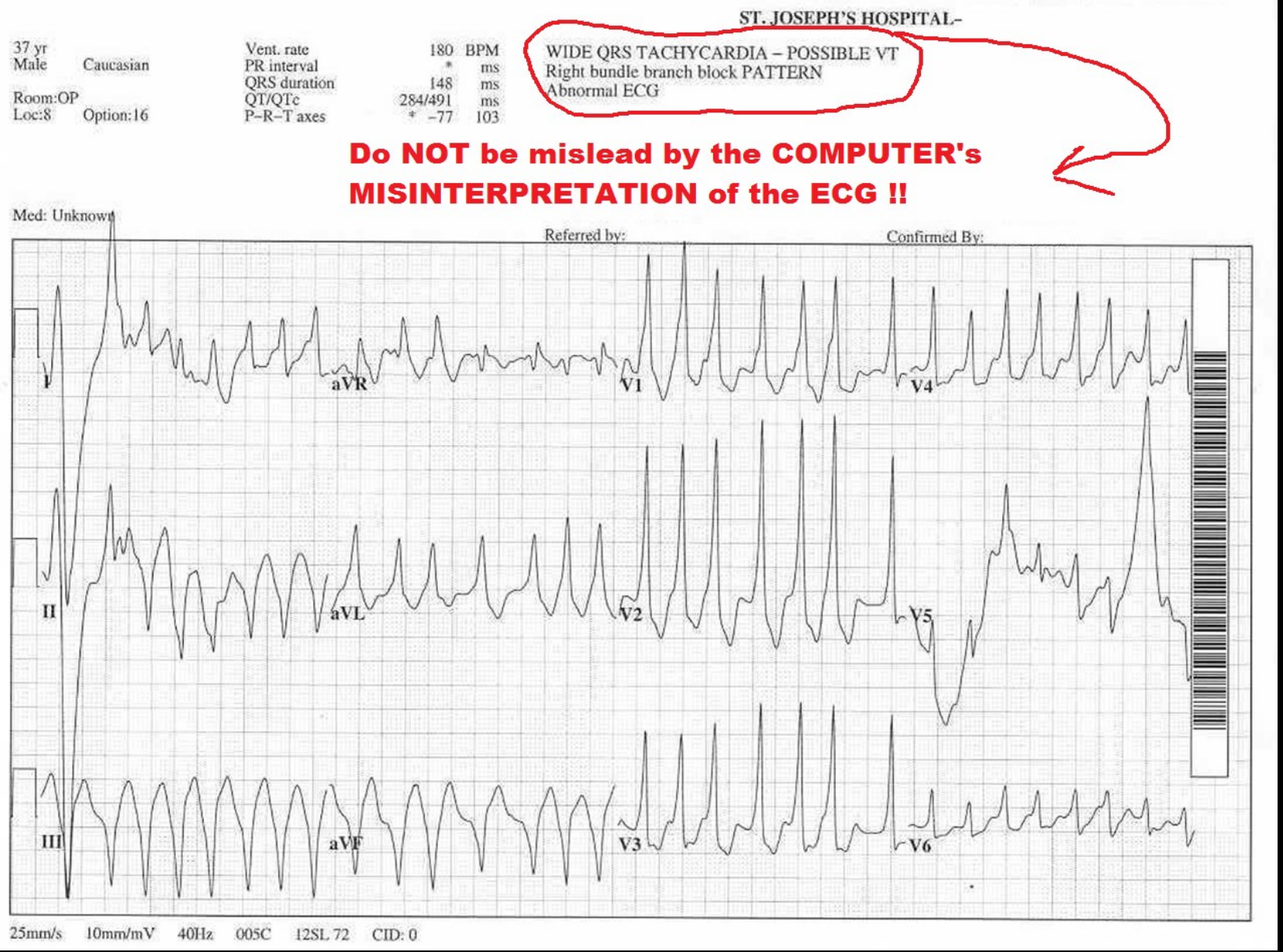


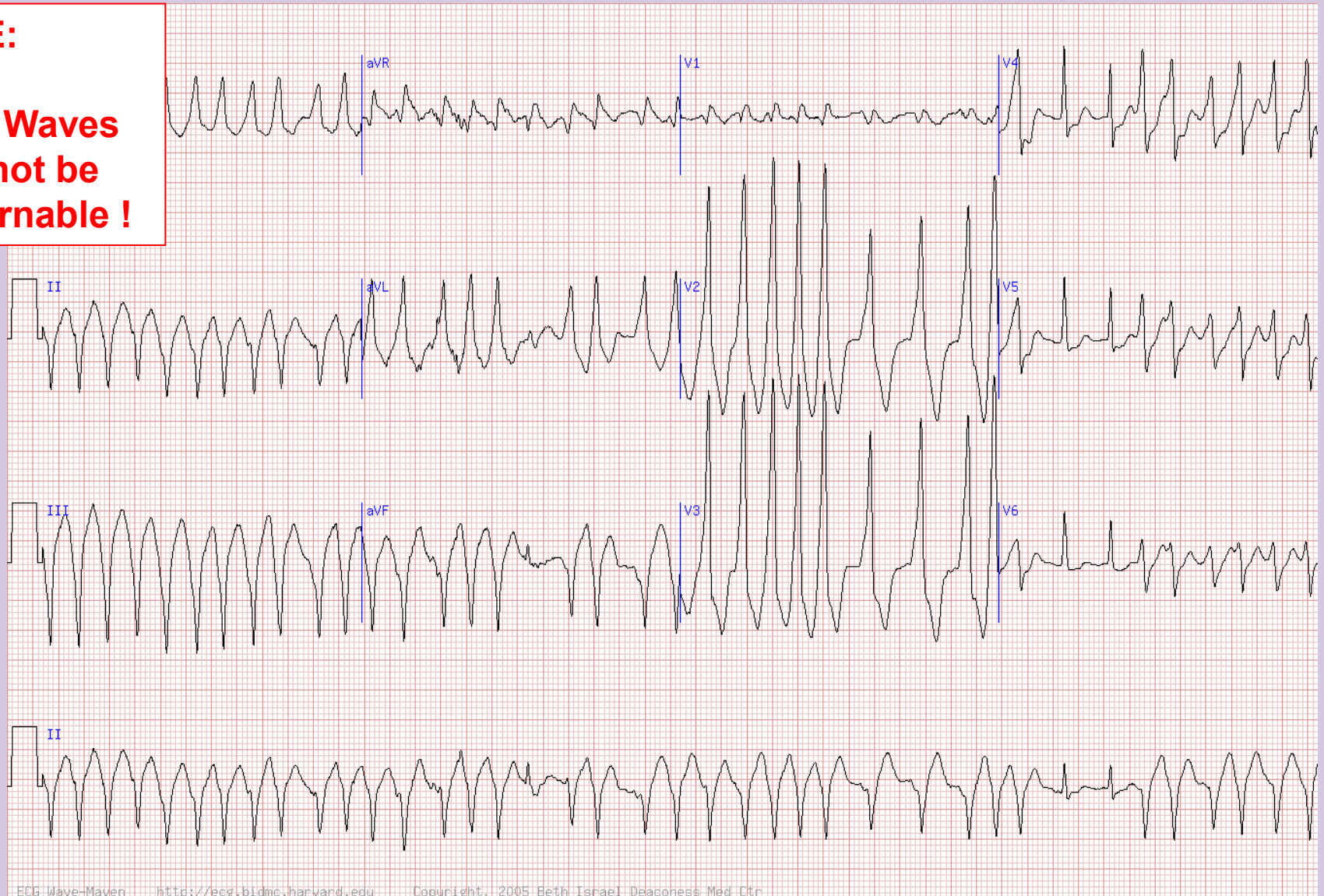
-**NOTE IRREGULARITY OF RHYTHM** - (SUGGESTIVE of A-FIB)
-**WIDE QRS – Consider Bypass Tract** (W-P-W)
-DELTA WAVES ? **(may or may not be visible).**

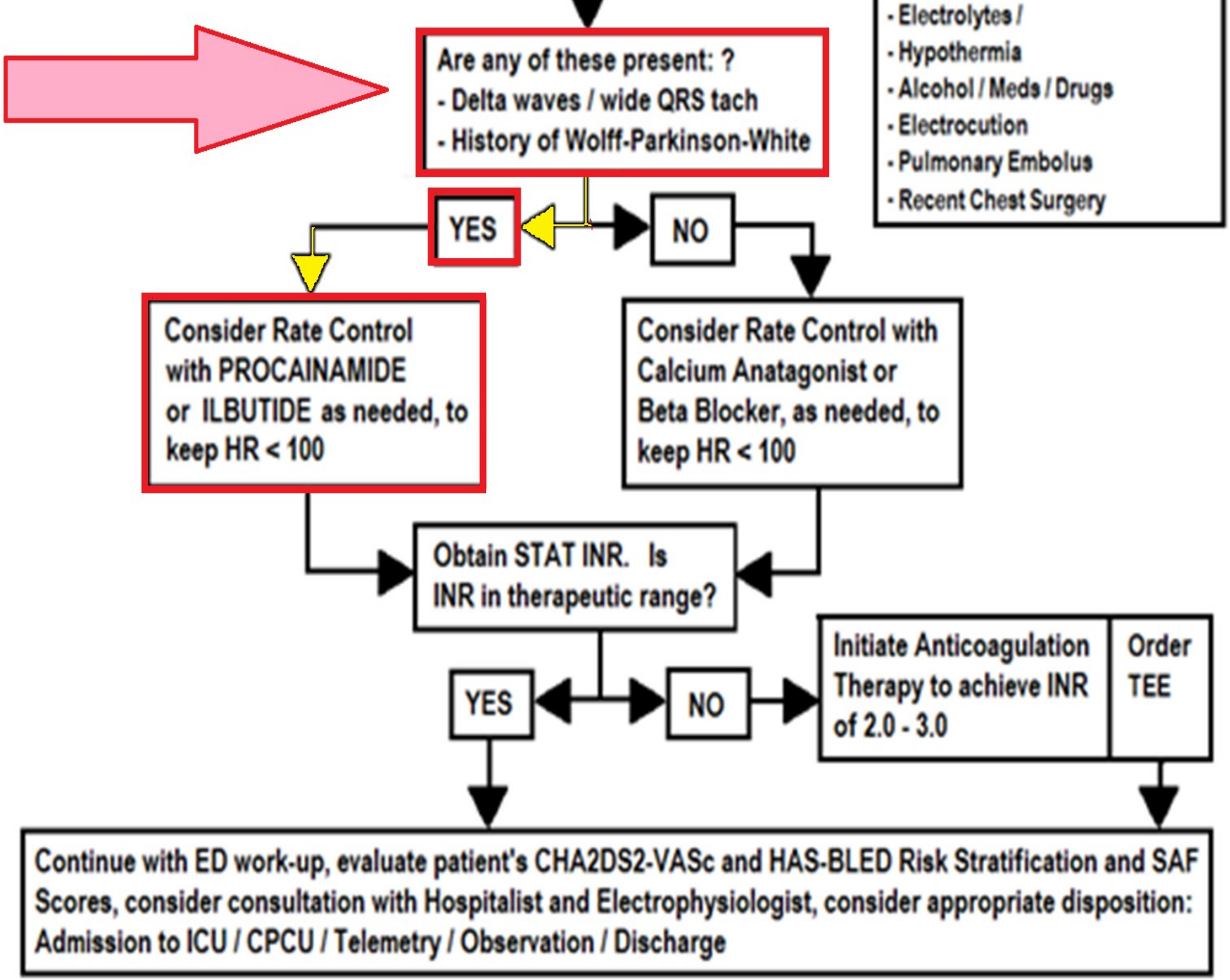
CHARACTERISTICS of W-P-W with Afib & RVR:

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

NOTE:

Delta Waves may not be discernable !

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Continue with ED work-up, evaluate patient's CHA2DS2-VASc and HAS-BLED Risk Stratification and SAF Scores, consider consultation with Hospitalist and Electrophysiologist, consider appropriate disposition: Admission to ICU / CPCU / Telemetry / Observation / Discharge

Afib/AFL: Pt. Hemodynamically Stable:

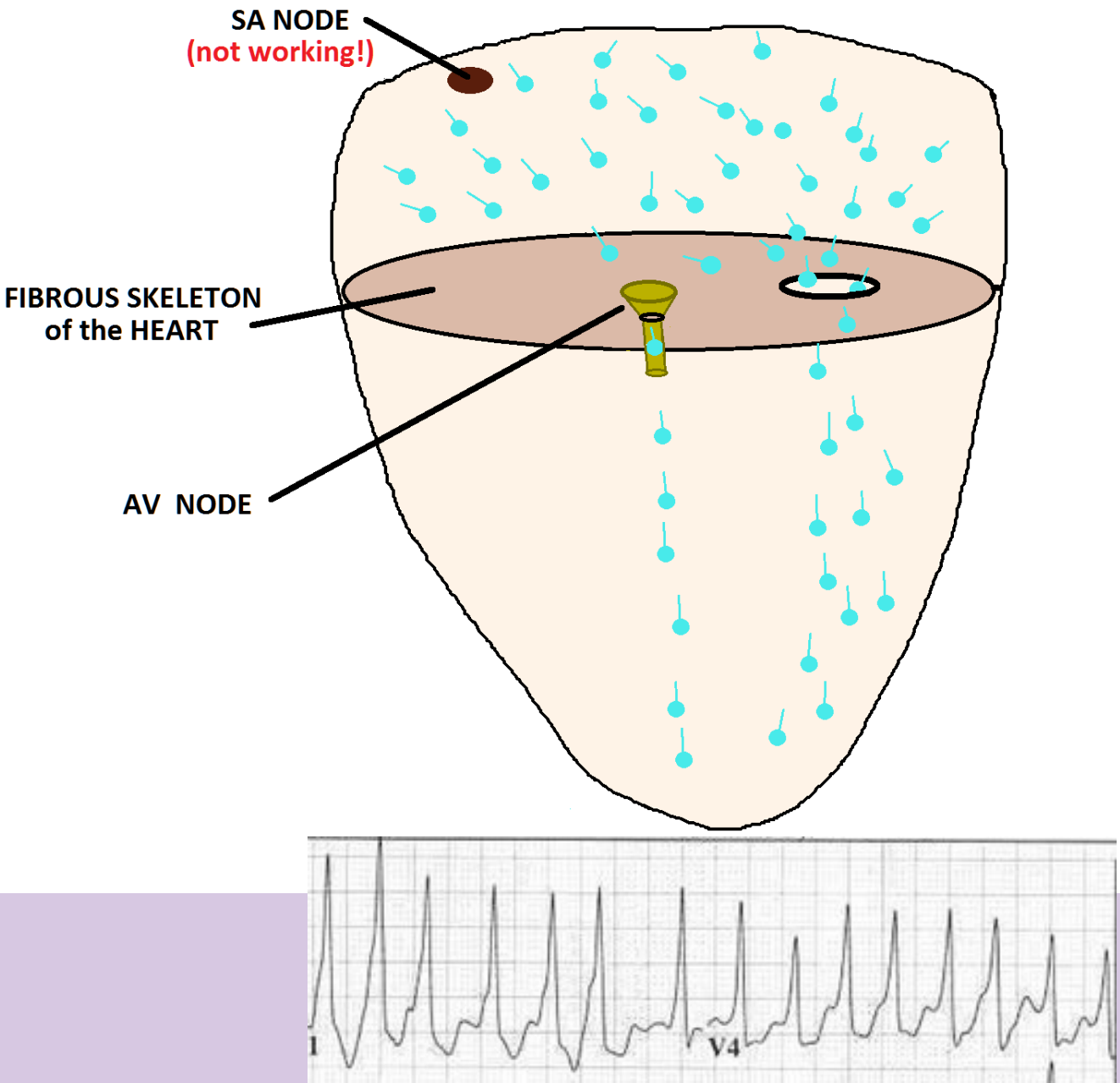
- **Rate control strategy:**
  - **QRS Complexes Wide. If Delta waves are present or if unable to rule out pre-excitation:**

    Administration of intravenous amiodarone, adenosine, digoxin (oral or intravenous), or nondihydropyridine calcium channel antagonists (oral or intravenous) in patients with Wolff-Parkinson-White syndrome who have pre-excited AF is potentially harmful because these drugs accelerate the ventricular rate and are known to precipitate VENTRICULAR FIBRILLATION

    Class III (harm), LOE B

    Source: 2014 AHA/ACC/HRS Guideline for the Management of Patients With Atrial Fibrillation

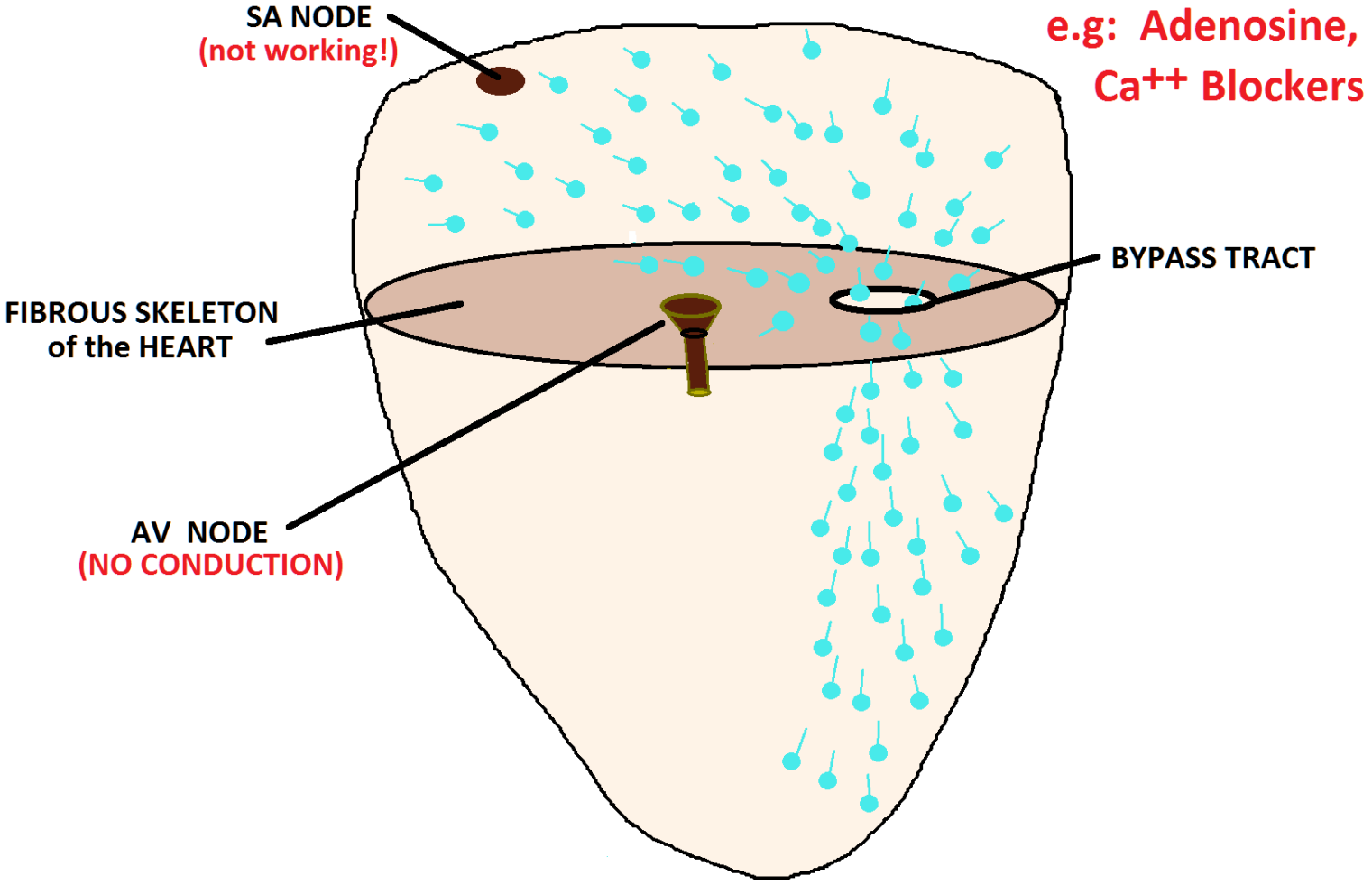
Atrial Fibrillation with Wolff-Parkinson White

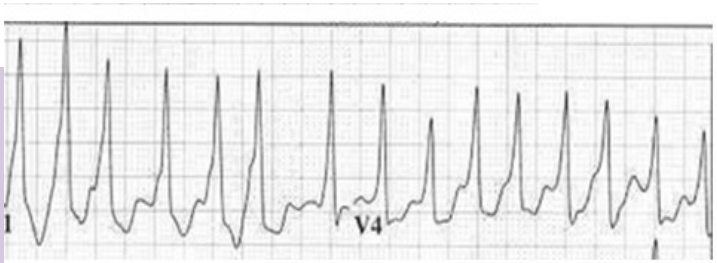


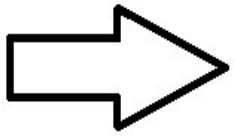
Atrial Fibrillation with Wolff-Parkinson White

with AV NODAL BLOCKING AGENTS

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







AF / AFL with Ventricular Rate >100 Patient is Hemodynamically Stable

- **Rate control strategy**
  - **QRS Complexes Wide (>120ms). If Delta waves are present or if unable to rule out pre-excitation:**

    Intravenous **procainamide** or **ibutilide** (Corvert) to restore sinus rhythm or slow the ventricular rate is recommended for patients with pre-excited AF and rapid ventricular response who are not hemodynamically compromised .

    Class I, LOE C

AHA ACLS 2015 with ADDED CONSIDERATIONS for WPW with A-FIB and RVR

WIDE COMPLEX TACHYCARDIA (QRS > 120 ms)

MONOPHASIC

ABC s

NO PULSE

GO TO
V - FIB
ALGORITHM !

PULSE - UNSTABLE

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

PULSE - STABLE

- O2, IV-IO, EKG
- MEDS:
  - ~~ADENOSINE 6-12 (RAPID BOLUS)~~
  - PROCAINAMIDE (20-50mg/min)
  - ~~AMIODARONE~~
  - ILBUTILIDE

Case Progression . . . .

- ED Physician ordered Diltiazem bolus and maintenance infusion.
- Veteran ED RN expressed concern that rhythm could be Afib with Bypass Tract. Physician became visibly angered and ordered RN to administer the Diltiazem ***as ordered.***
- **During Diltiazem bolus, patient converted to Ventricular Fibrillation**.
- Post Defibrillation 12 Lead ECG revealed . . . .

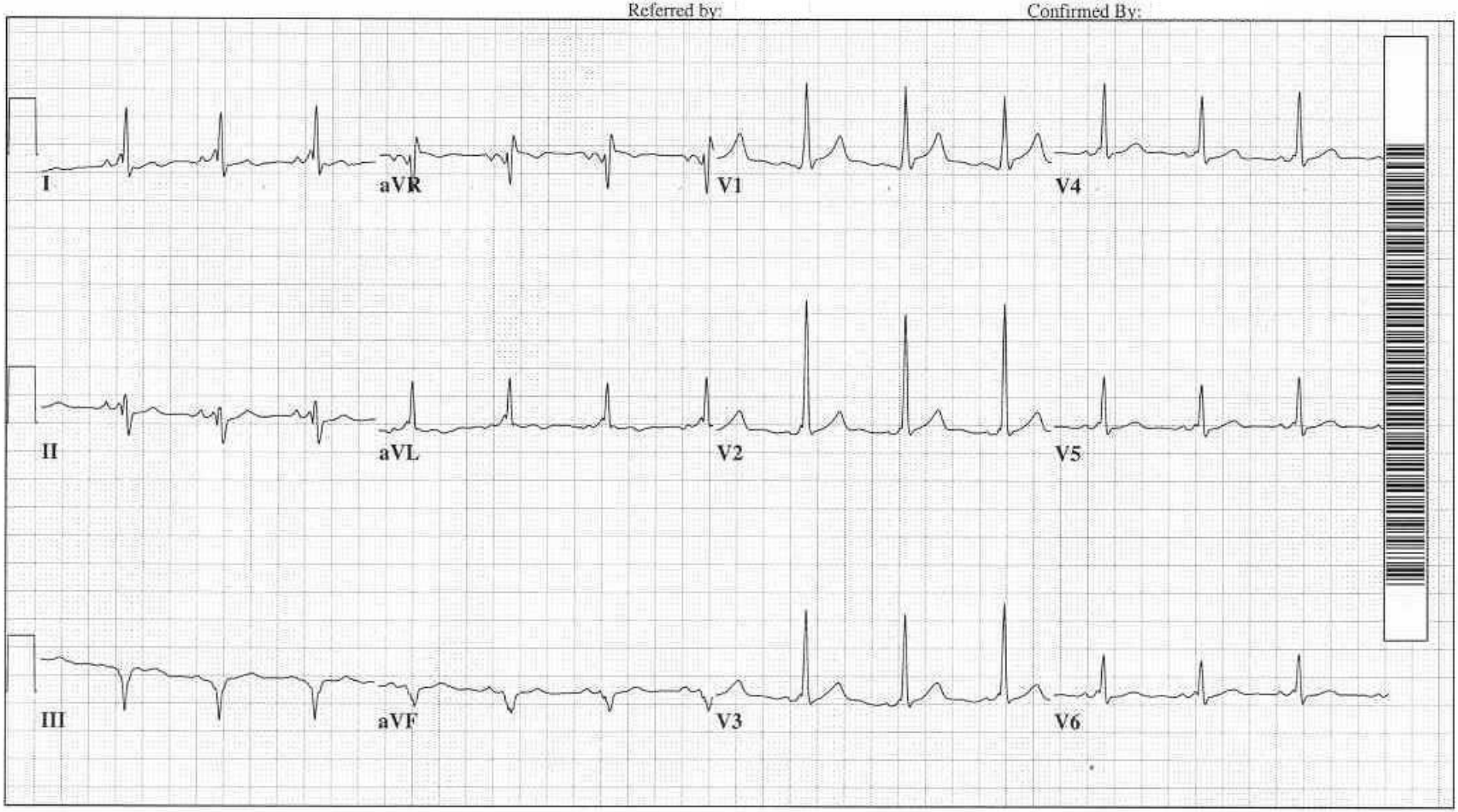
37 yr
Male Caucasian

Room:OP
Loc:8 Option:19

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

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

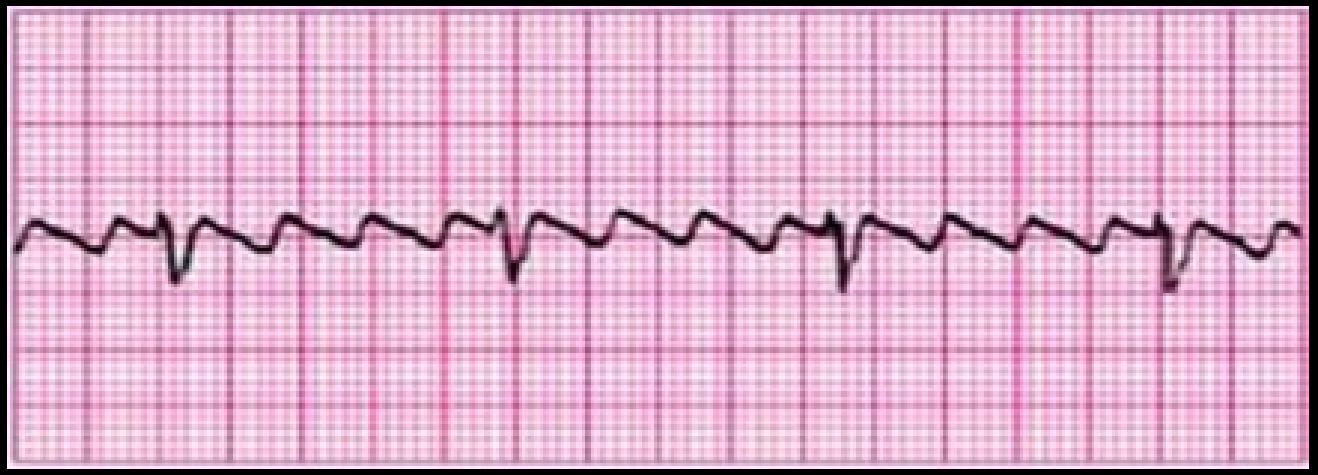
Referred by: Confirmed By:

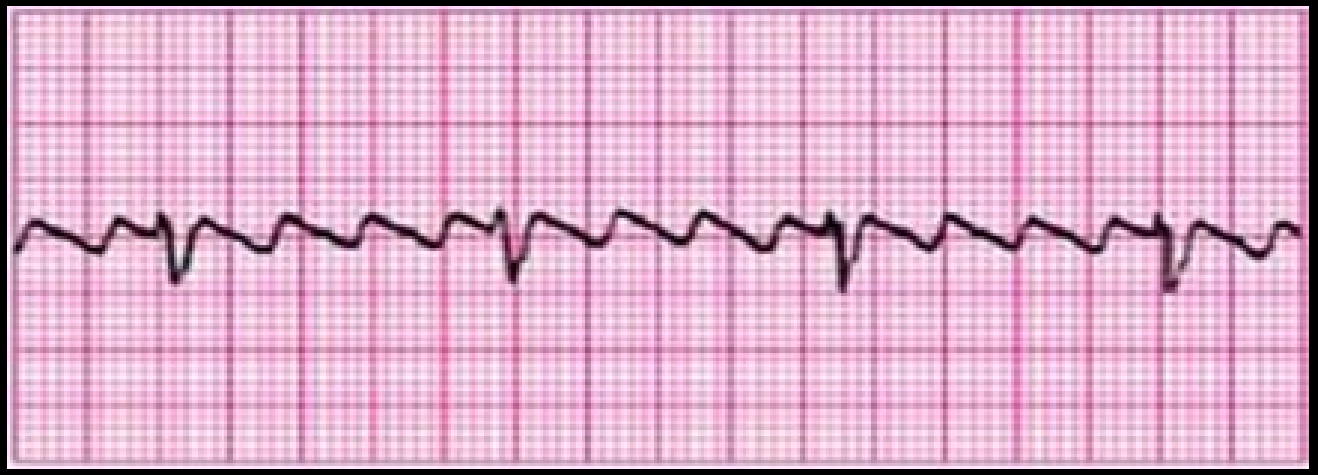


25mm/s 10mm/mV 40Hz 005C 12SL 78 CID: 0

AHA ACLS Advocates TEAM CONCEPT:

- Team Leader coordinates overall patient management.
- Asking other Team Members for their ideas and suggestions is encouraged.
- Consider suggestions with open mind, then implement or veto suggestion as indicated.
- ***When orders are given, staff must repeat order back to team leader. THIS IS CALLED: "CLOSED-LOOP COMMUNICATION."***





-NEED FOR EMERGENT TX BASED ON VENTRICULAR RATE

-OFTEN OBSERVED INTERMITTENTLY WITH A-FIB

-IF CARDIOVERSION NEEDED, CONSIDER R/O ATRIAL THROMBUS

68 yr
Male Hispanic

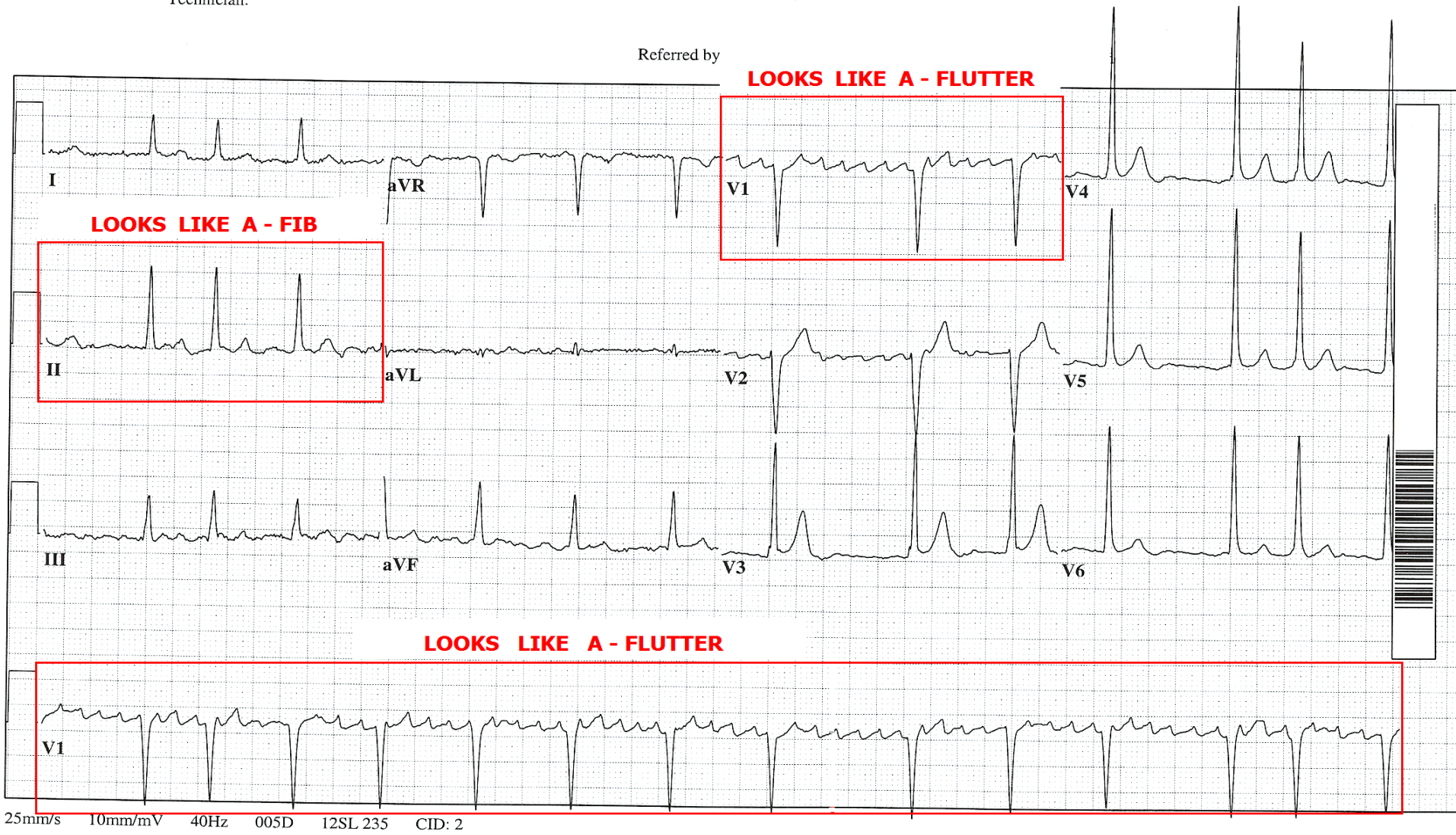
Room:VAM
Loc:3 Option:23

Vent. rate	85	BPM
PR interval	*	ms
QRS duration	100	ms
QT/QTc	342/406	ms
P–R–T axes	* 58	46

"**UNEDITED COPY: REPORT IS COMPUTER GENERATED ONLY, WITHOUT PHYSICIAN INTERPRETATION".
Atrial fibrillation
Voltage criteria for left ventricular hypertrophy
Abnormal ECG
When compared with ECG of 19–NOV–2006 07:39,
No significant change was found

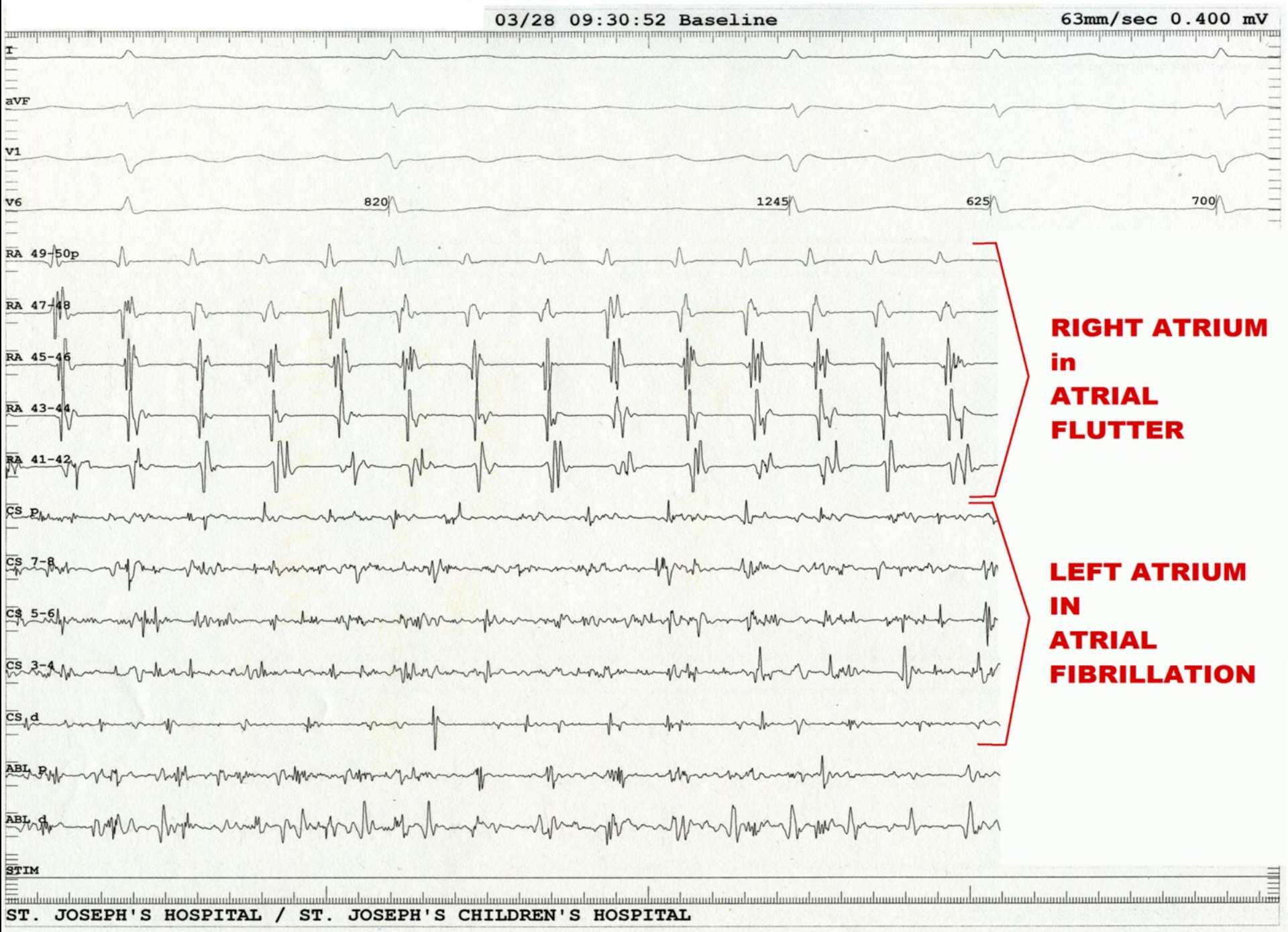
Technician:

Referred by



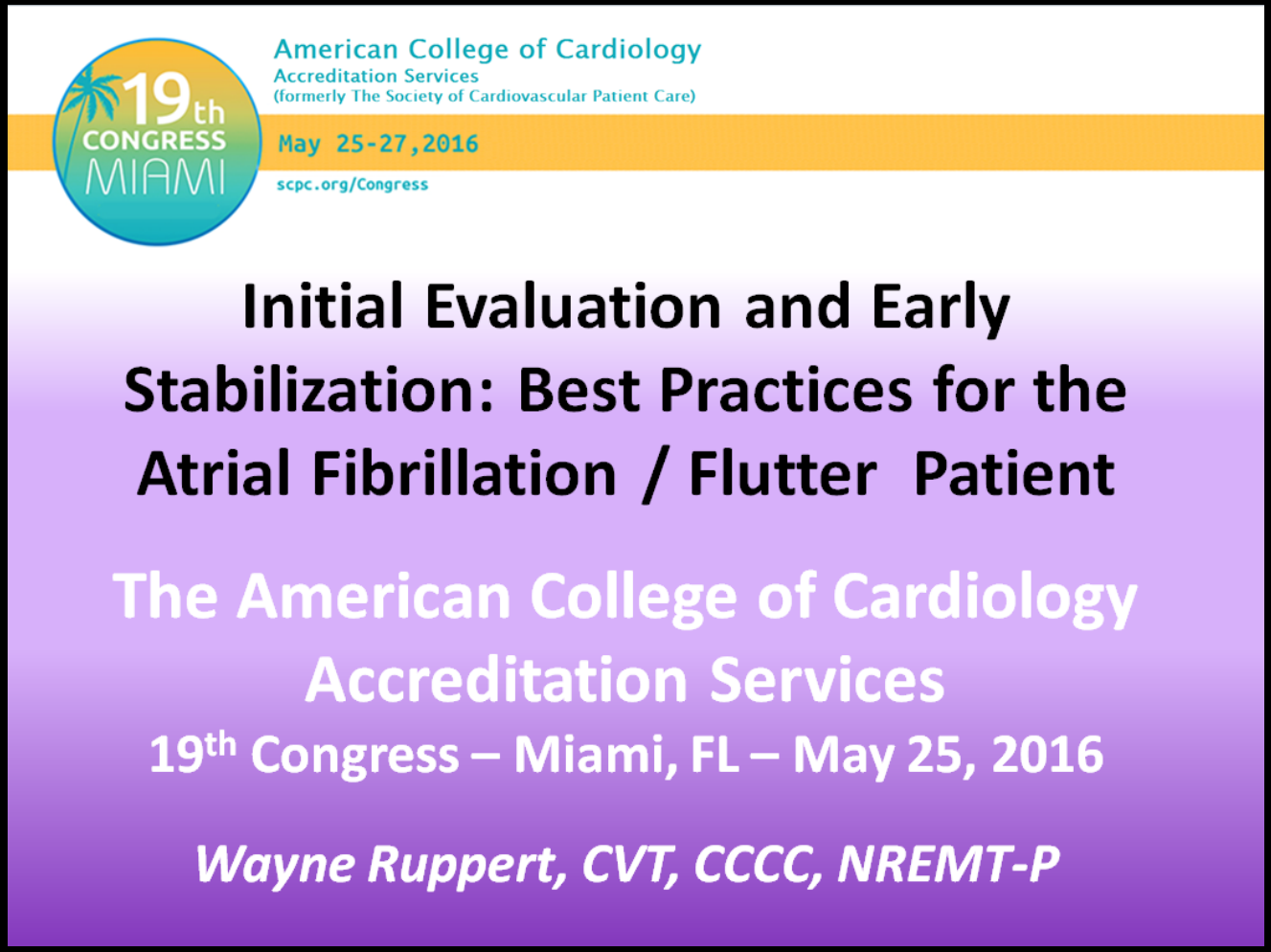
25mm/s 10mm/mV 40Hz 005D 12SL 235 CID: 2

"ATRIAL FIB - FLUTTER"

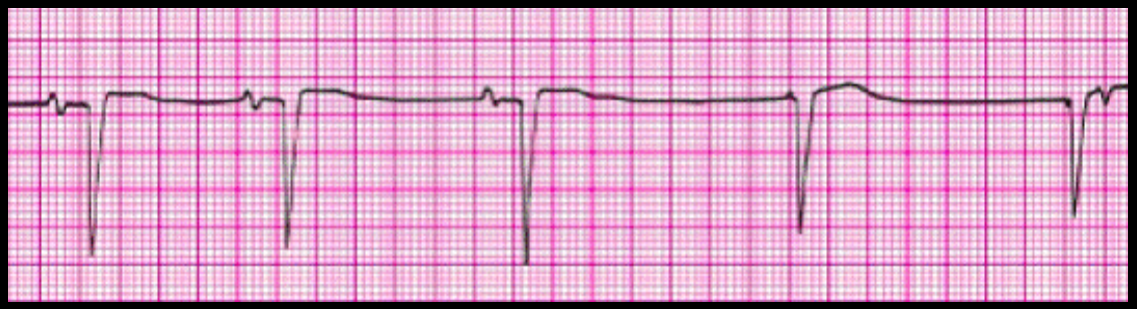


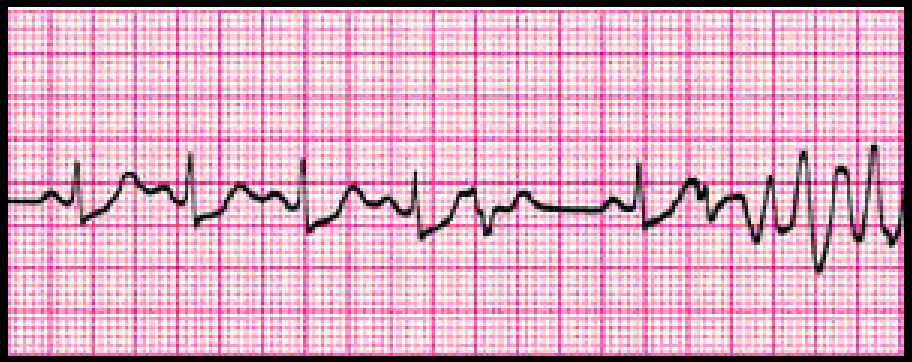
☞ Management of A-Flutter: same considerations as A-Fib.

For MORE INFO on the Mgmt. of Afib / AFL,



CLICK HERE to download presentation "Initial Evaluation and Early Stabilization: Best Practices for the AF/AFL Pt."





THOUGHTS TO CONSIDER
FOR MANAGEMENT OF VENTRICULAR ECTOPY:

1. **DOES IT POSE AN IMMEDIATE THREAT TO THE PATIENT'S WELL-BEING (e.g. R on T, RUNS OF VT, or EXCESSIVE FREQUENCY) ?**

2. **DOES IT IMPAIR THE PATIENT'S VENTRICULAR FUNCTION (e.g. - Frequent RV OUTFLOW TRACT PVCs).**

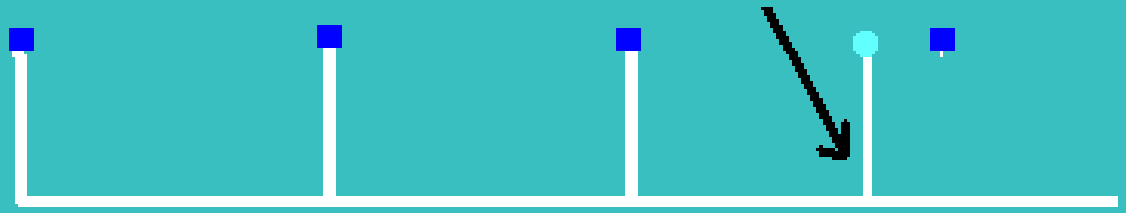
3. **ETIOLOGY ? (AMI, ISCHEMIA, etc)**

IMMEDIATE THERAPEUTIC INTERVENTION:
- **PROCAINAMIDE (BE AWARE OF QT INTERVAL)**
- **AMIODARONE (BE AWARE of QT INTERVAL – USE OF AMIODARONE MAY DELAY SUCCESSFUL EP STUDY FOR SEVERAL WEEKS !!)**
- **LIDOCAINE**

CLASSIFICATIONS OF ECTOPY

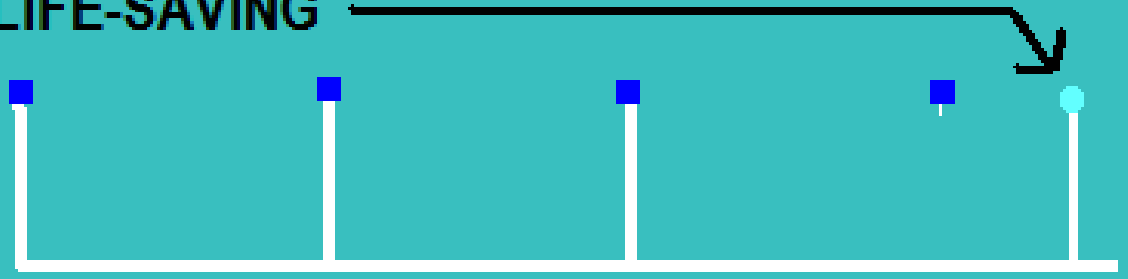
1. PREMATURE

THE ECTOPIC BEAT COMES BEFORE THE NEXT REGULARLY EXPECTED BEAT (IT's EARLY !)



2. END-DIASTOLIC, ESCAPE, or COMPENSATORY

THE ECTOPIC BEAT COMES AFTER A REGULAR BEAT FAILS TO HAPPEN. END-DIASTOLIC BEATS MAY BE LIFE-SAVING



CAUSES OF ECTOPY

1. PREMATURE

- HYPOXIA
- IRRITABILITY
- CHANGES IS SYMPATHETIC / PARASYMPATHETIC TONE
- DAMAGE TO MYOCARDIUM CAUSING CHANGES IN AUTOMATICITY (such as from MI / NECROSIS, etc.).
- MEDICATIONS / SUBSTANCES
- ELECTROLYTES

2. END-DIASTOLIC, ESCAPE, or COMPENSATORY

- FAILUARE OF SA NODE
- FAILURE OF AV NODE

WHEN THESE FAIL TO PRODUCE OR PROPOGATE AN IMPULSE, ESCAPE FOCI MAY TAKE OVER PACING THE HEART BY PRODUCING END-DIASTOLIC BEATS

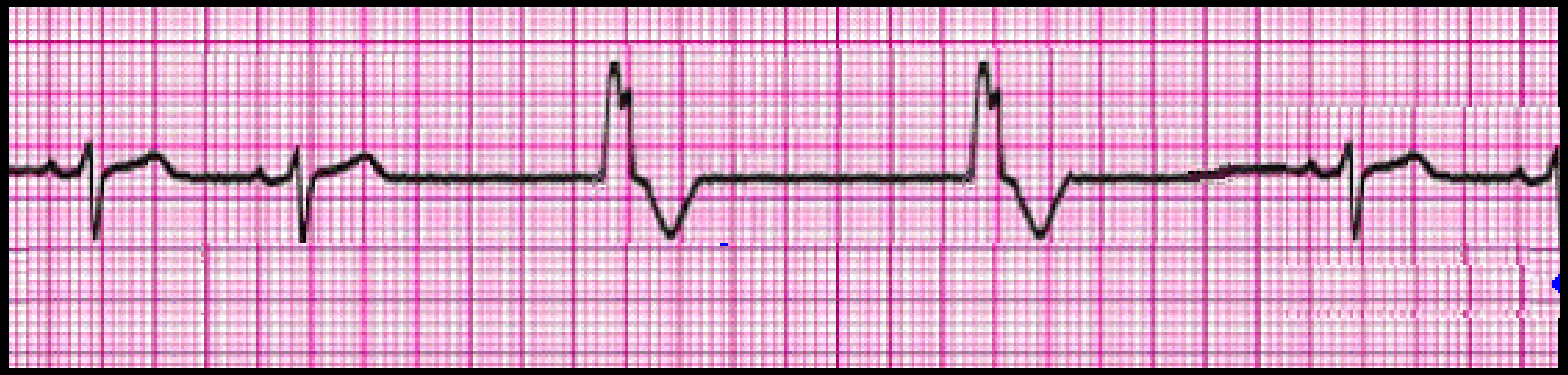
SIMPLY STATED,

1. **PREMATURE BEATS ----** **BAD**

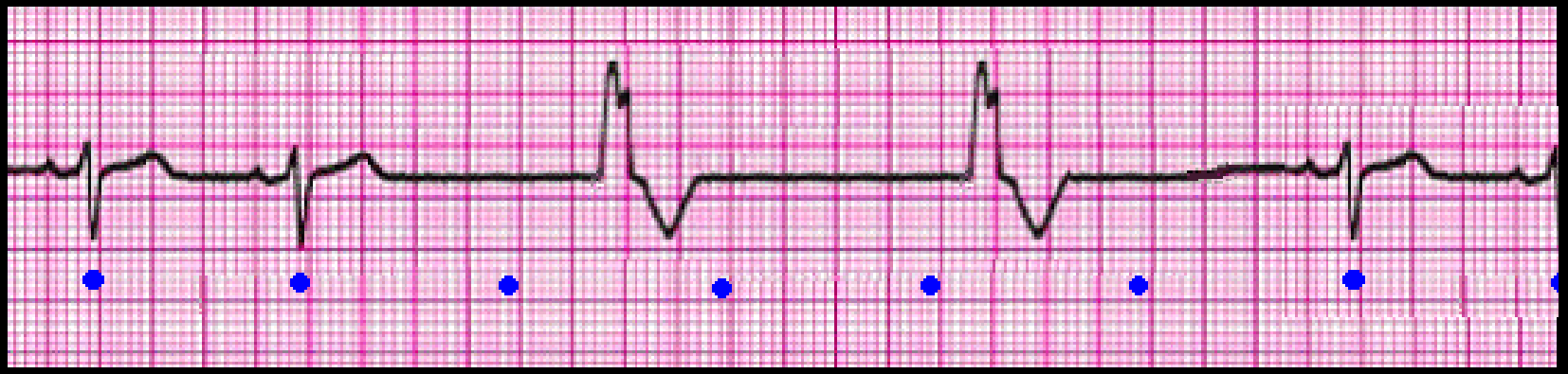
IN SOME CASES WE MUST ELIMINATE PREMATURE BEATS TO PROTECT THE PATIENT

2. **END-DIASTOLIC or ESCAPE BEATS ----** **GOOD**

ELIMINATION OF END-DIASTOLIC BEATS COULD BE DEADLY



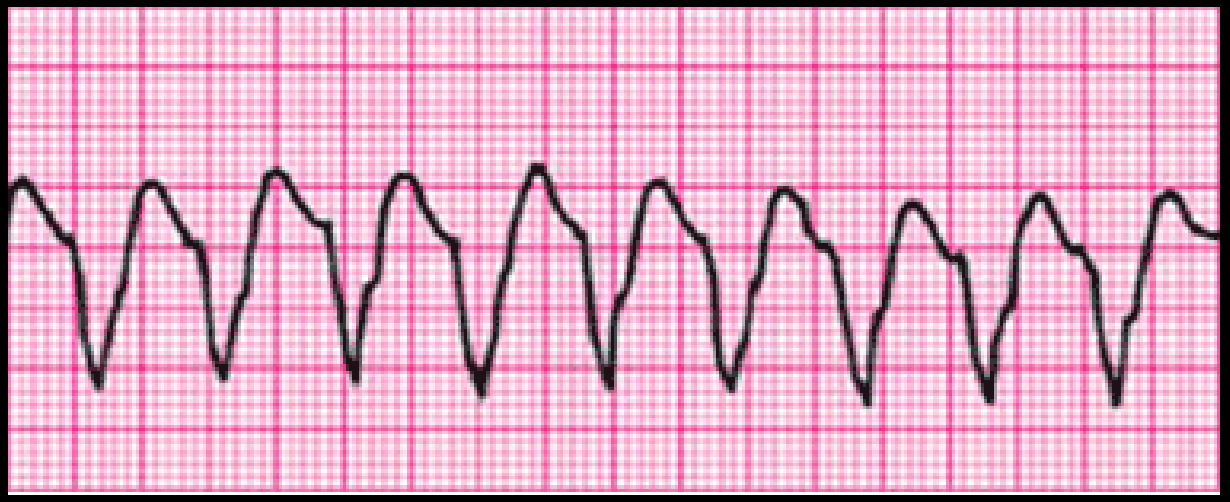
THIS RHYTHM IS: SINUS ARREST w/ VENT. ESCAPE



TREATMENT / INTERVENTION (S):

- EMERGENT TREATMENT IS TRANSCUTANEOUS PACING.
- TREAT UNDERLYING CAUSE OF SINUS / AV ARREST
- ***DO NOT*** ATTEMPT TO SUPRESS VENTRICULAR ESCAPE BEATS WITHOUT HAVING BACK-UP TRANSCUTANEOUS / TRANSVENOUS PACING ATTACHED TO PATIENT !!!

WIDE COMPLEX TACHYCARDIA . . . .



IT COULD BE:

- MONOPHASIC VT

- SVT WITH BUNDLE BRANCH BLOCK

- ANTEDROMIC RECIPROCATING BYPASS TRACT MEDIATED TACHYCARDIA (W-P-W).

WIDE COMPLEX TACHYCARDIA (QRS > 120 ms)

MONOPHASIC

ABC s

NO PULSE

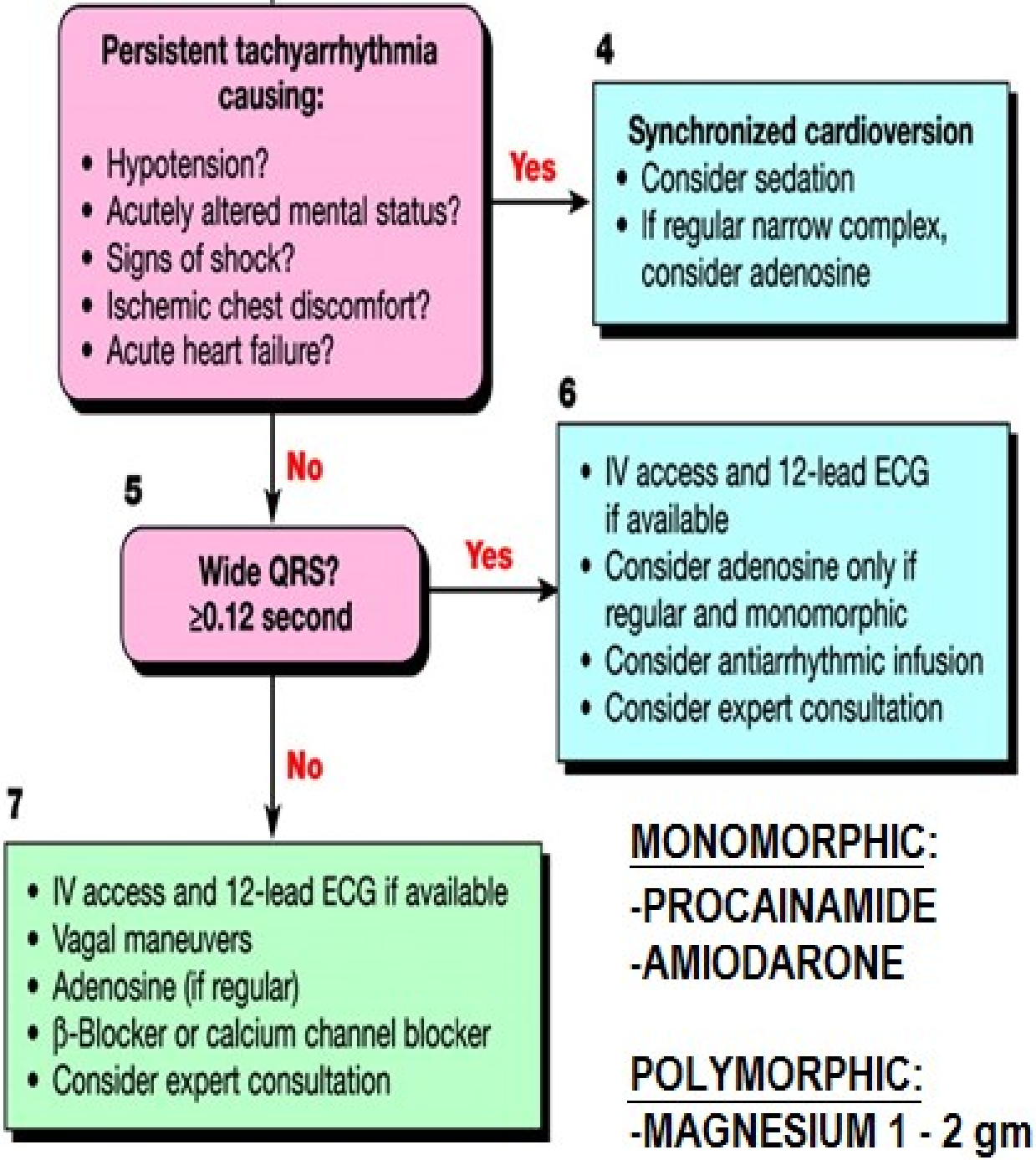
GO TO
V - FIB
ALGORITHM !

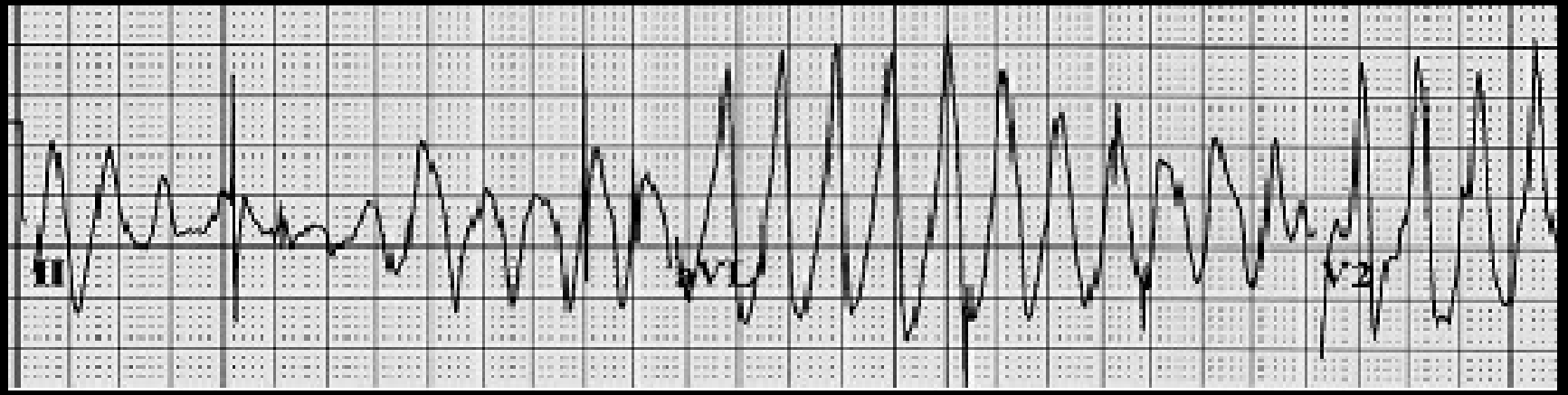
PULSE - UNSTABLE

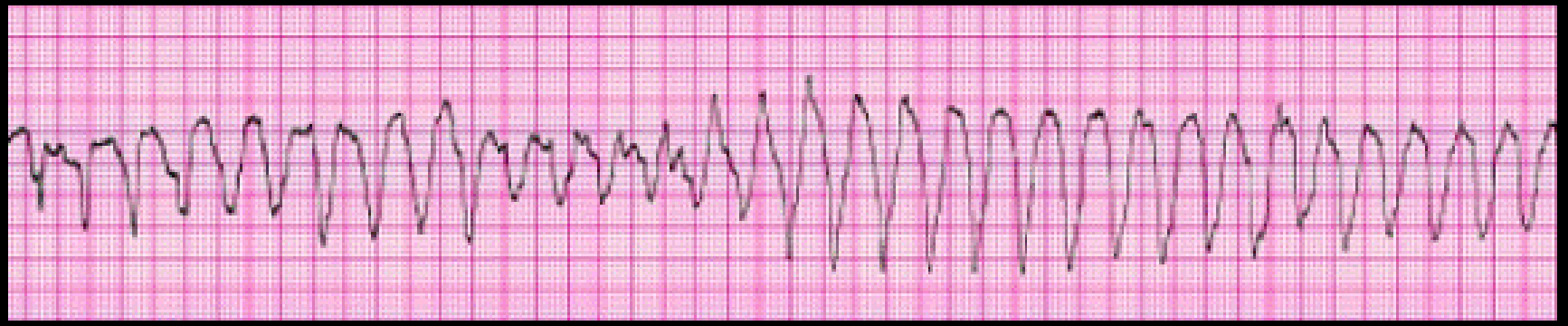
- IMMEDIATE SYNC. CARDIOVERSION:
  - 100 j biphasic
  - consider sedation
- INCREASE joules
- MEDS:
  - PROCAINAMIDE
  - AMIODARONE

PULSE - STABLE

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


Persistent tachyarrhythmia causing:
• Hypotension?
• Acutely altered mental status?
• Signs of shock?
• Ischemic chest discomfort?
• Acute heart failure?
Yes
4
Synchronized cardioversion
• Consider sedation
• If regular narrow complex, consider adenosine
No
5
Wide QRS?
≥0.12 second
Yes
6
• IV access and 12-lead ECG if available
• Consider adenosine only if regular and monomorphic
• Consider antiarrhythmic infusion
• Consider expert consultation
No
7
• IV access and 12-lead ECG if available
• Vagal maneuvers
• Adenosine (if regular)
• β-Blocker or calcium channel blocker
• Consider expert consultation
MONOMORPHIC:
-PROCAINAMIDE
-AMIODARONE
POLYMORPHIC:
-MAGNESIUM 1 - 2 gm

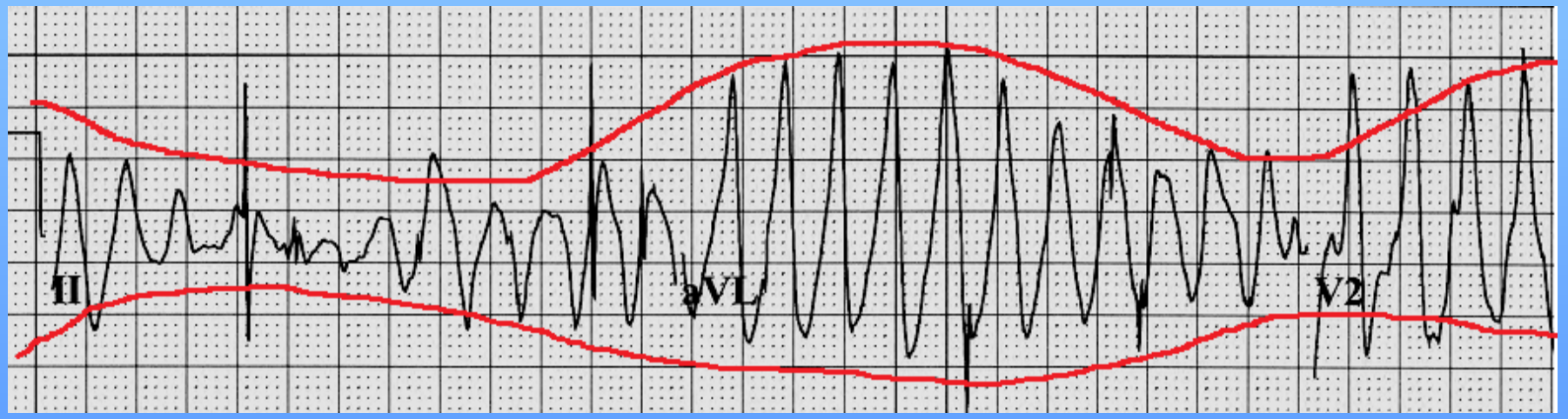

II
V1
V2



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

resembles . . . . .

a piece of Twisted Ribbon !



WIDE COMPLEX TACHYCARDIA
TORSADES de POINTES

(QRS > 120 ms)

ABC s

NO PULSE

GO TO
V - FIB
ALGORITHM !

PULSE - UNSTABLE

- IMMEDIATE
UNSYNCHRONIZED
DEFIBRILLATION
120-200 j biphasic
360 monophasic

PULSE - STABLE

- O2 / IV / EKG
- MAGNESIUM
1 - 2 gm OVER
5 - 60 min, THEN
INFUSION

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

Patient Experiencing Runs of TdP:

- *DC QT prolonging Meds IMMEDIATELY (consider alternate therapy - pharmacy?)*
- *Magnesium Sulfate 1-2 grams IV infused over 5-60 minutes*
- *STAT 12 Lead ECG – Assess QT / QTc interval, Abnormal T and U waves . . .*
- *Electrolytes with Serum Mag.*

Determining the QT / QTc

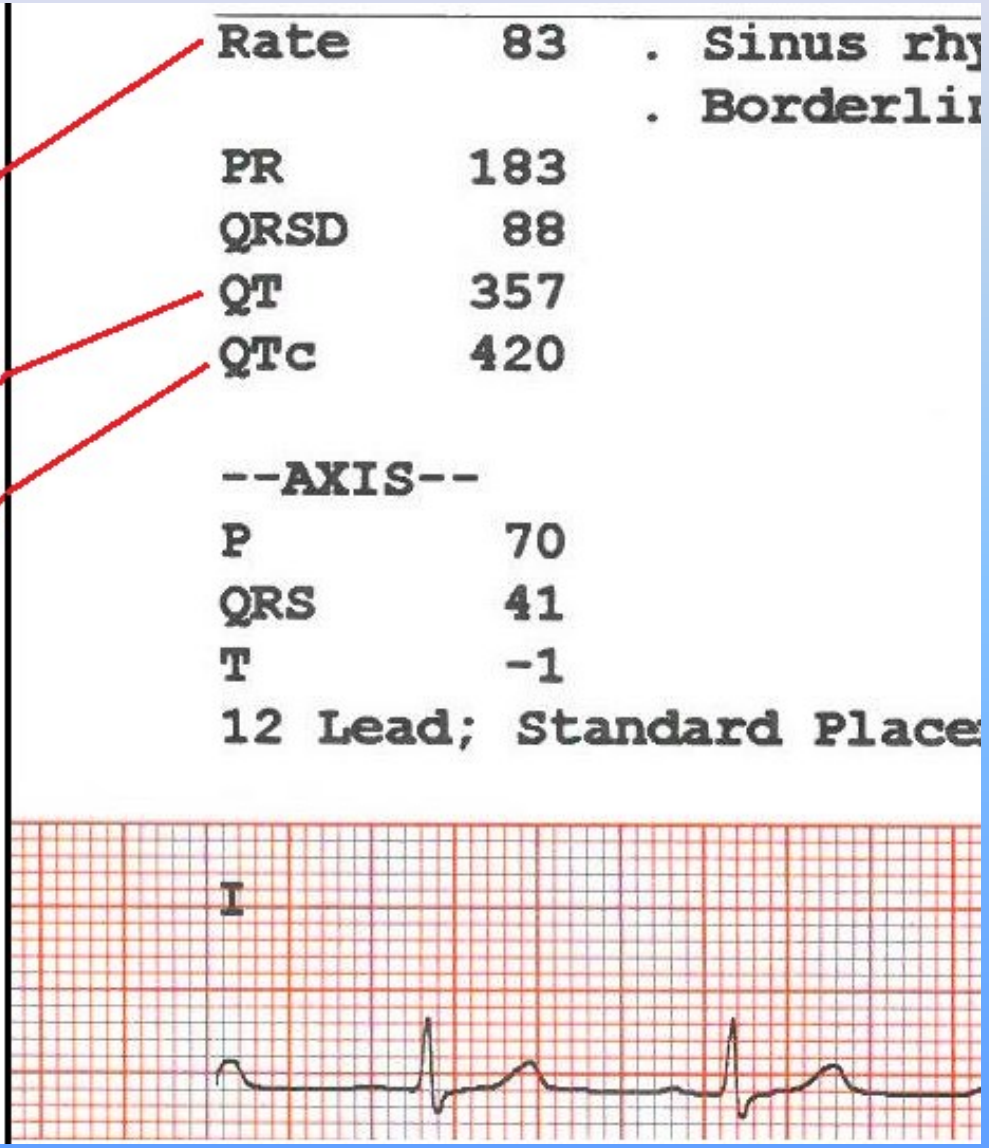
Method 1 – 12 Lead ECG Report:

Standard 12 Lead ECG printout . . .

Heart Rate = 83

QT Interval = 357

QTc = 420



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**

SOURCE: "ACC/AHA/HRS Recommendations for Standardization and Interpretation of the ECG, Part IV: The ST Segment, T and U Waves, and the QT Interval" Rautaharju et al 2009

CONSIDER ORDERING 12 Lead ECG (to asses QT/QTc) on "high risk" patients:

- *History / Fam history of:*
  - *LQTS*
  - *Brugada Syndrome*
  - *Sudden Death*
- *Patients receiving two or more QT prolonging Meds*
- *Patients with "syncope unknown etiology"*

Also for patients with known QT prolongation or "at risk" patients:

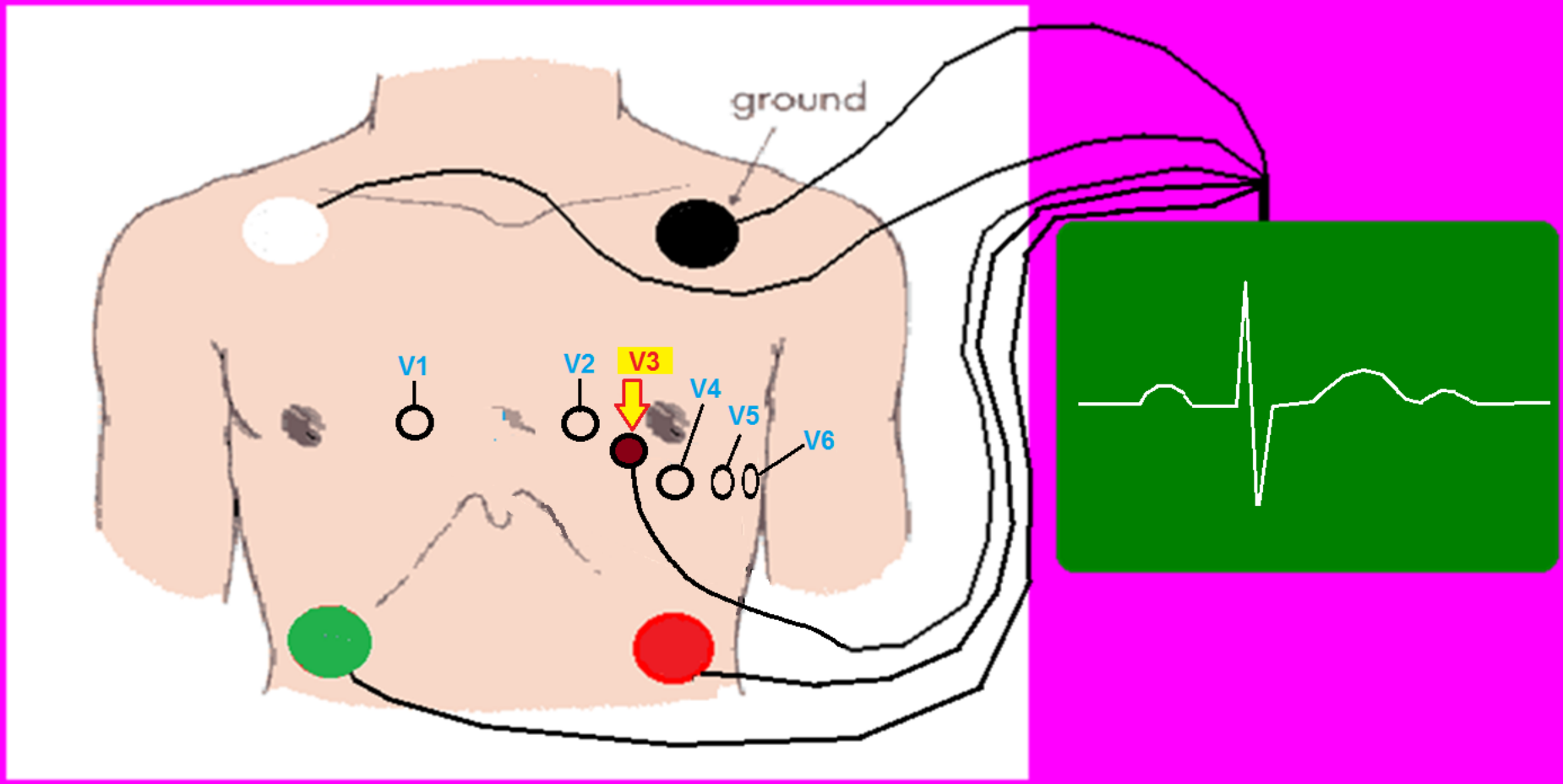
Consider ordering

CONTINUOUS QTc MONITORING

For CPC Accreditation, SRRMC is currently developing a "QTc Monitoring Protocol."

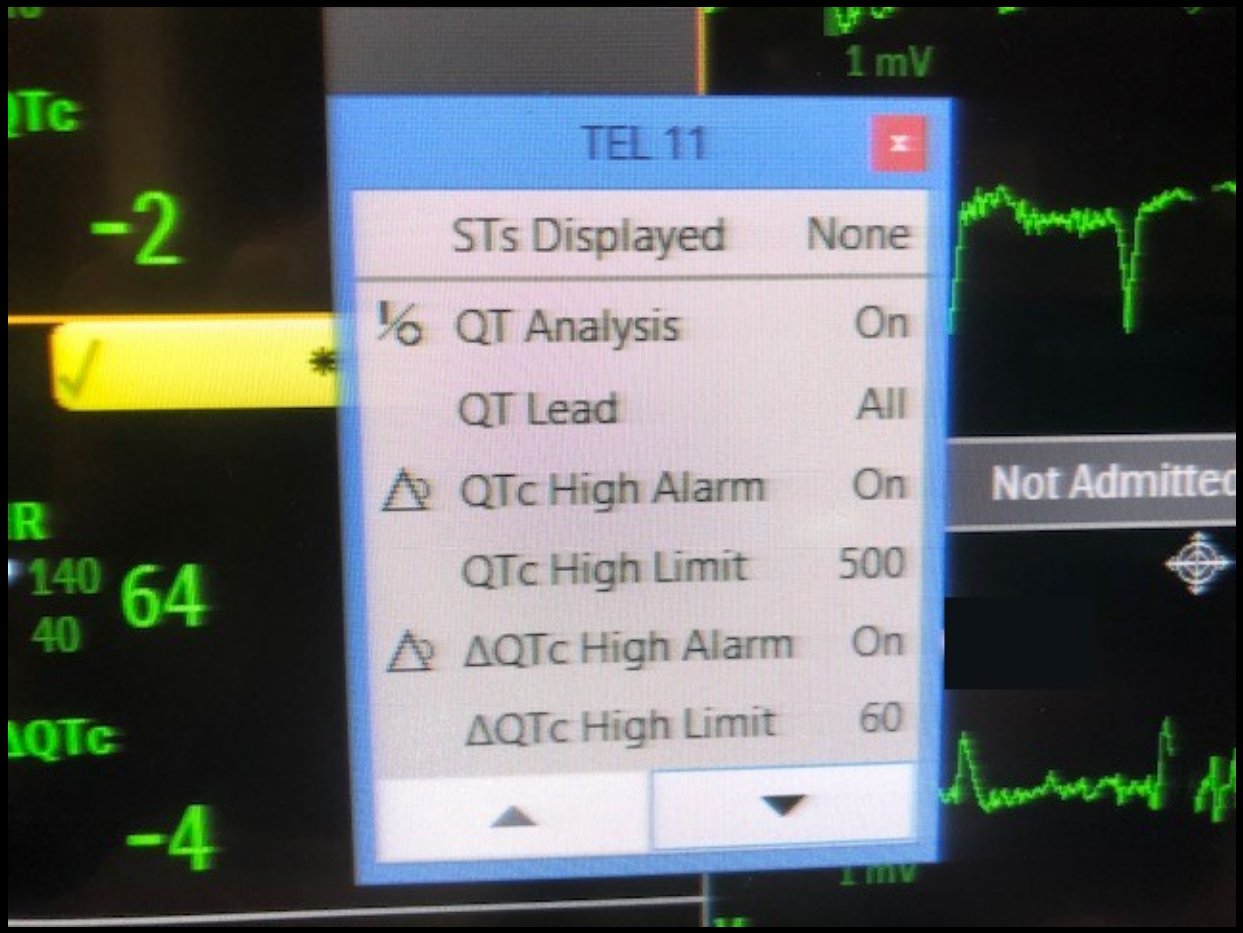
It will include . . . .

LEAD PLACEMENT - V3



5 WIRE TELEMETRY UNIT

At SRRMC: Automated CONTINUOUS QTc MONITORING Available for Tele:



At SRRMC: Automated CONTINUOUS QTc MONITORING Available for Tele:

QT Prolongation -- *D/C QT Prolonging Meds:*

☞ ***Avoidance of Meds that are known to prolong the QT Interval. Click here for current list from CREDIBLEMEDS.ORG***

Commonly used QT prolonging meds include:

-Amiodarone
-Procainamide
-Levaquin
-Erythromycin
-Norpace
-Tequin
-Benadryl
-Ritalin
-Pseudophedrine
-Haloperidol
-Thorazine
-Propulcid
-Zofran
-Ilbutilide

and MANY more!

[LINK to preview EP tools on iTunes website – click here]

//itunes.apple.com/us/app/eptools/id430201878?mt=8

App Store Preview

epTools 17+

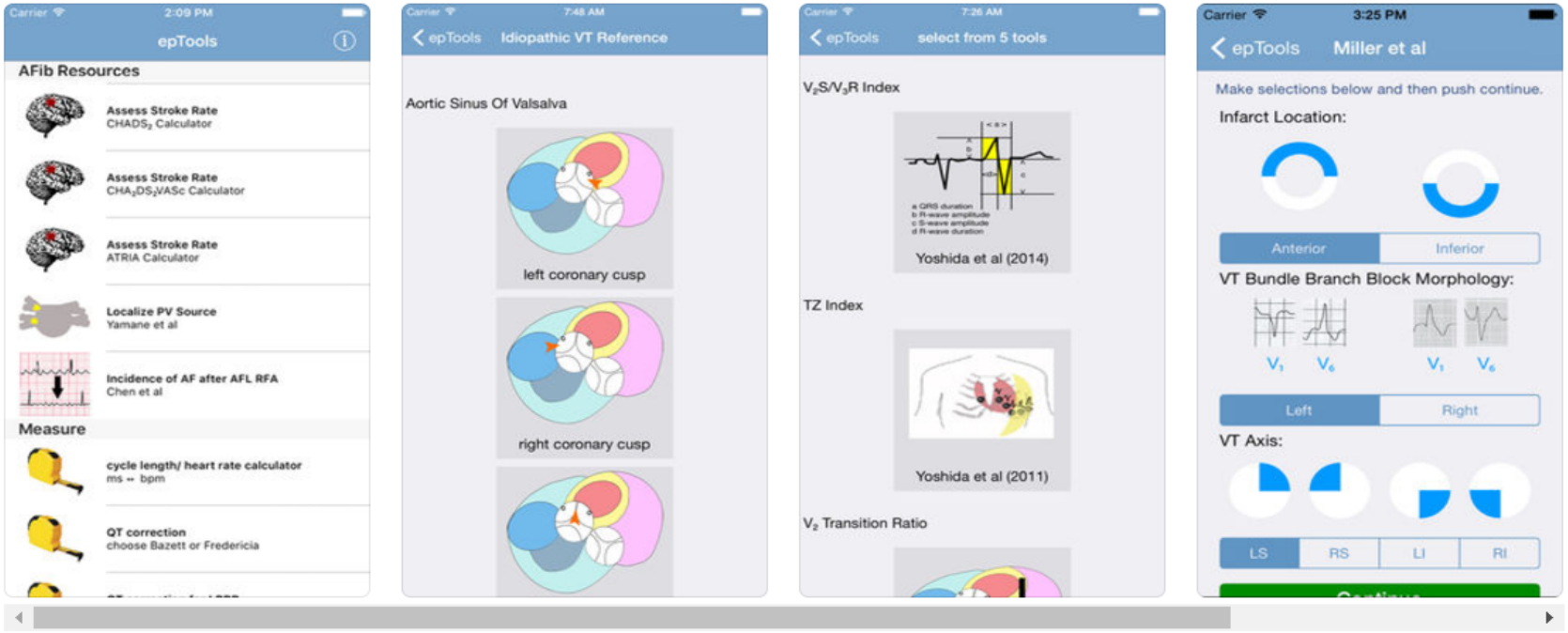
Resources for Cardiac EP

Busy Being Born Solutions, LLC

$5.99

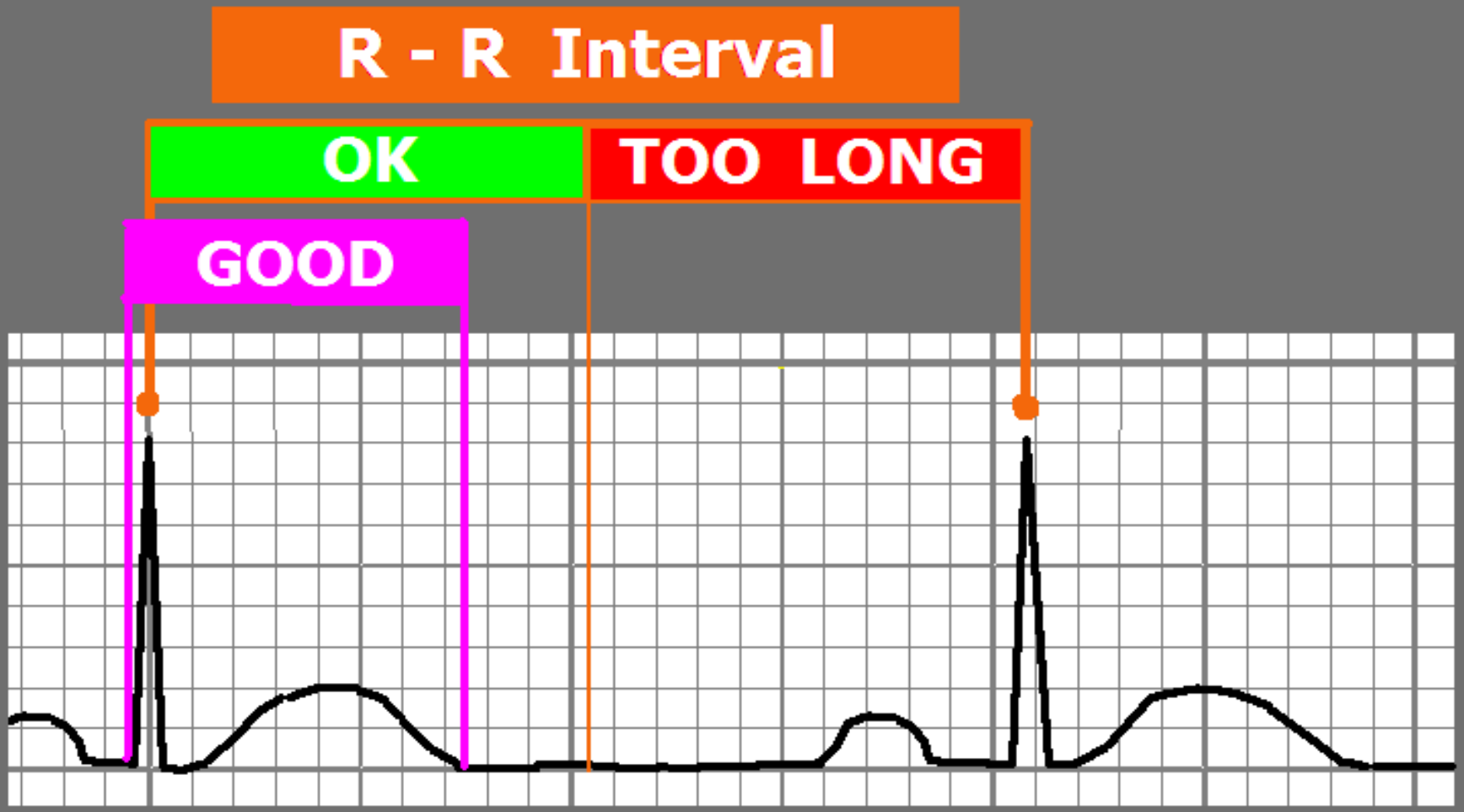
My favorite ECG / Cardiology iPhone APP:
- **has updated list of QT prolonging meds from AZ University (AZCERT)**
- **QTc calculation tools (Bazett's & Fredericia)**

Screenshots iPhone iPad

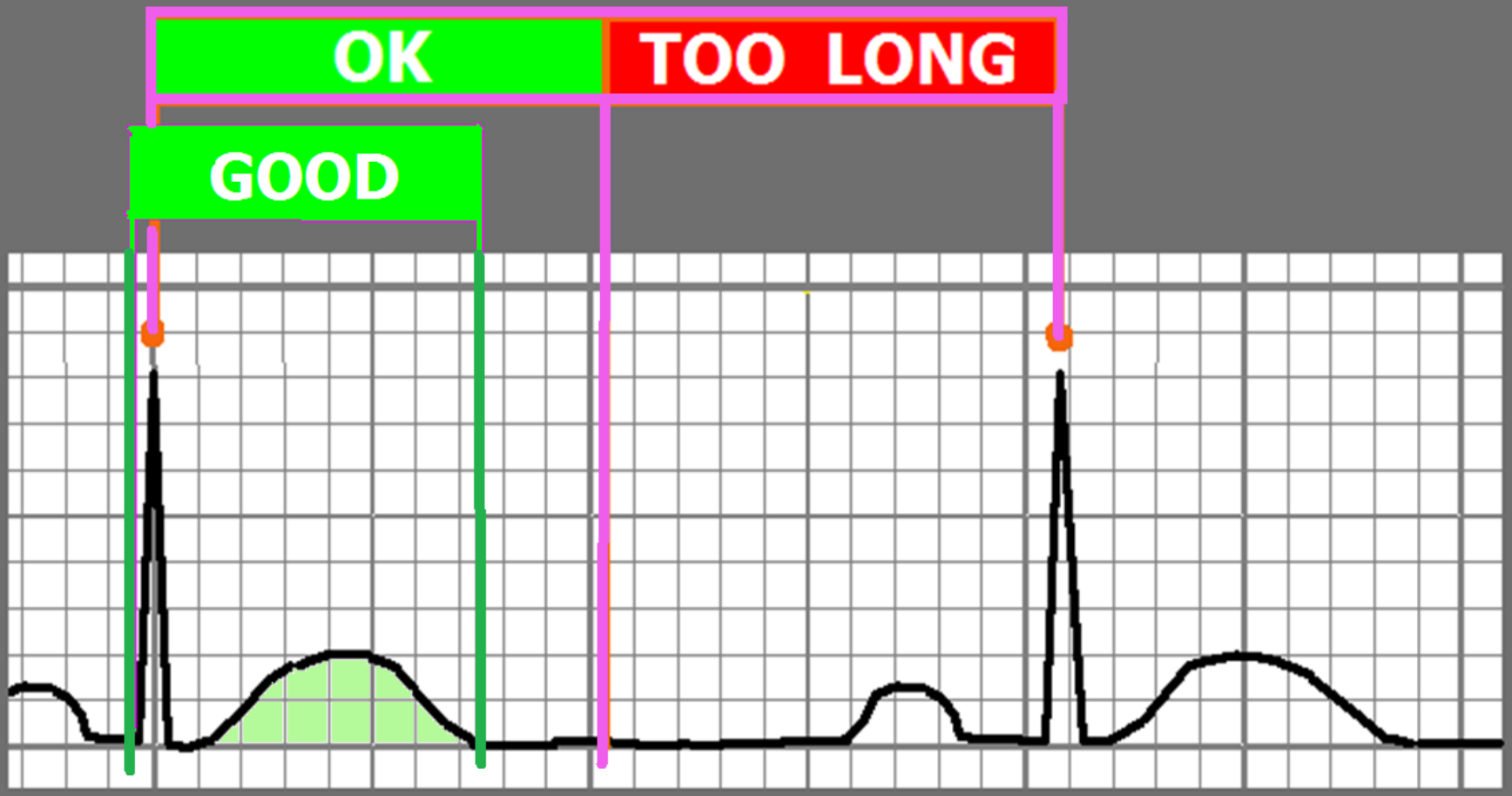


FOR VENTRICULAR RATES BETWEEN 60 – 100 with QRSd <120ms:

The Q - T Interval should be LESS THAN 1/2 the



The Q - T Interval should be LESS THAN 1/2 the R - R Interval



The Q - T Interval should be LESS THAN 1/2 the R - R Interval

Known ECG Indicators of Long QT Syndrome:

- QTc 460ms or longer in females*
- QTc 450ms or longer in males*
- T wave alterans
- U waves 100% size of the T wave**
- U waves merged with T waves
- U wave opposite polarity of T wave
- U waves >0.1mv (1mm on standard calibrated ECG)

*P. Rautaharju, et al, "Standardization and Interpretation of the ECG, Part IV" JACC2009;53, no. 11:982-991

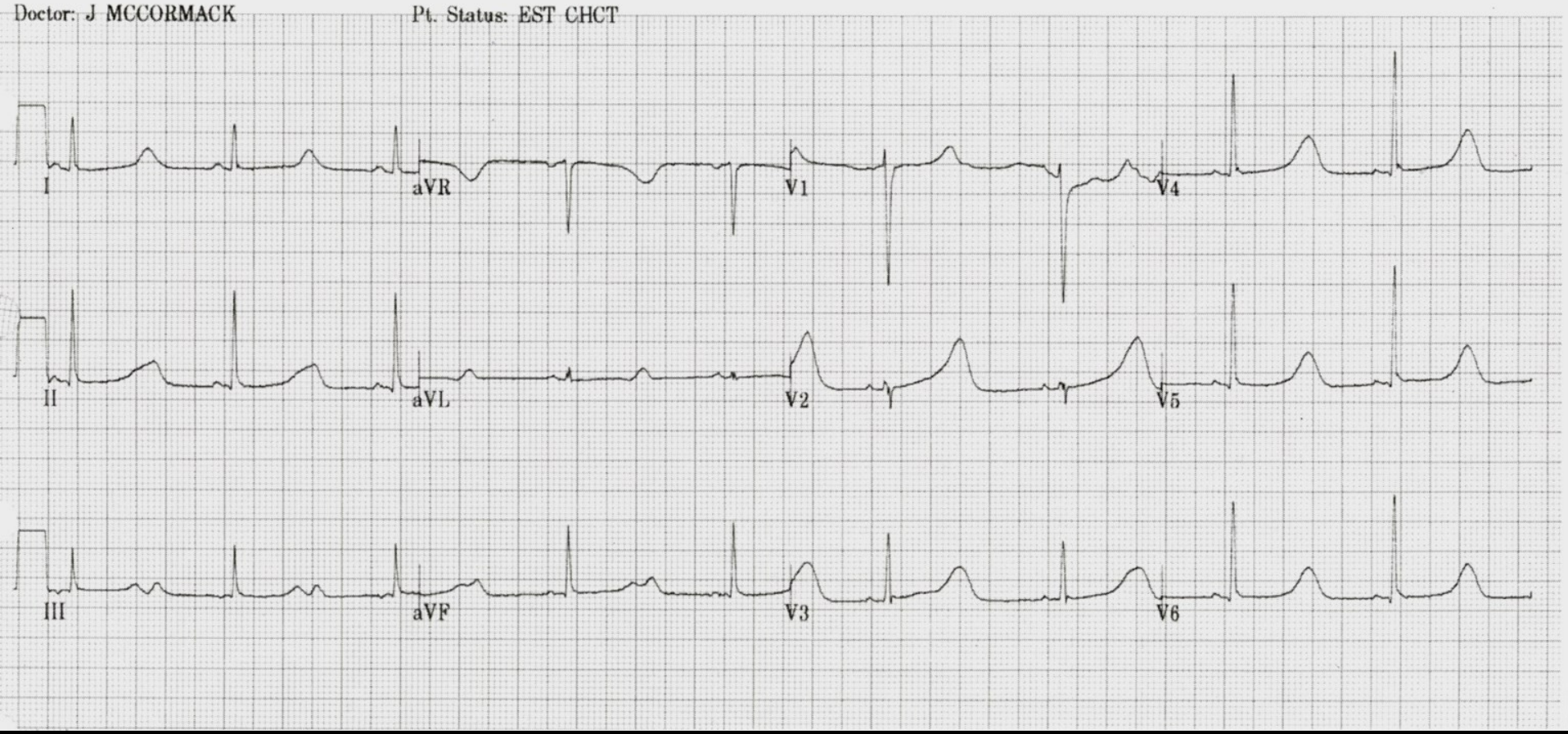
** Medical literature citations reflect variation in the value for U wave amplitude as an indicator of LQTS, ranging from 25 – 100% of the T wave amplitude.

☞ WHEN LQTS IS SUSPECTED, TAKE THE FOLLOWING PRECAUTIONS . . . .

22 y/o FEMALE

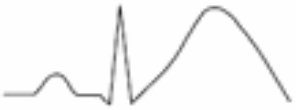
Vent. rate	53 bpm
PR interval	110 ms
QRS duration	84 ms
QT/QTc	678/636 ms
P-R-T axes	25 60 48

PEDIATRIC CARDIOLOGY ASSOCIATES



Patient diagnosed with Epilepsy, Rx: Dilantin, Phenobarbitol, Cerebrex – All INEFFECTIVE at controlling grand mal seizures . . . .

GENETICALLY ACQUIRED LONG QT SYNDROMES:
ECG PATTERNS of 3 MOST COMMON VARIATIONS:

Type	Current	Functional Effect	Frequency Among LQTS	ECG[12,13]	Triggers Lethal Cardiac Event[10]	Penetrance*
LQTS1	K	↓	30%-35%		Exercise (68%) Emotional Stress (14%) Sleep, Repose (9%) Others (19%)	62%
LQTS2	K	↓	25%-30%		Exercise (29%) Emotional Stress (49%) Sleep, Repose (22%)	75%
LQTS3	Na	↑	5%-10%		Exercise (4%) Emotional Stress (12%) Sleep, Repose (64%) Others (20%)	90%

Etiology of Long QT Syndromes:

Congenital (14 known subtypes)

Genetic mutation results in abnormalities of cellular ion channels

Acquired

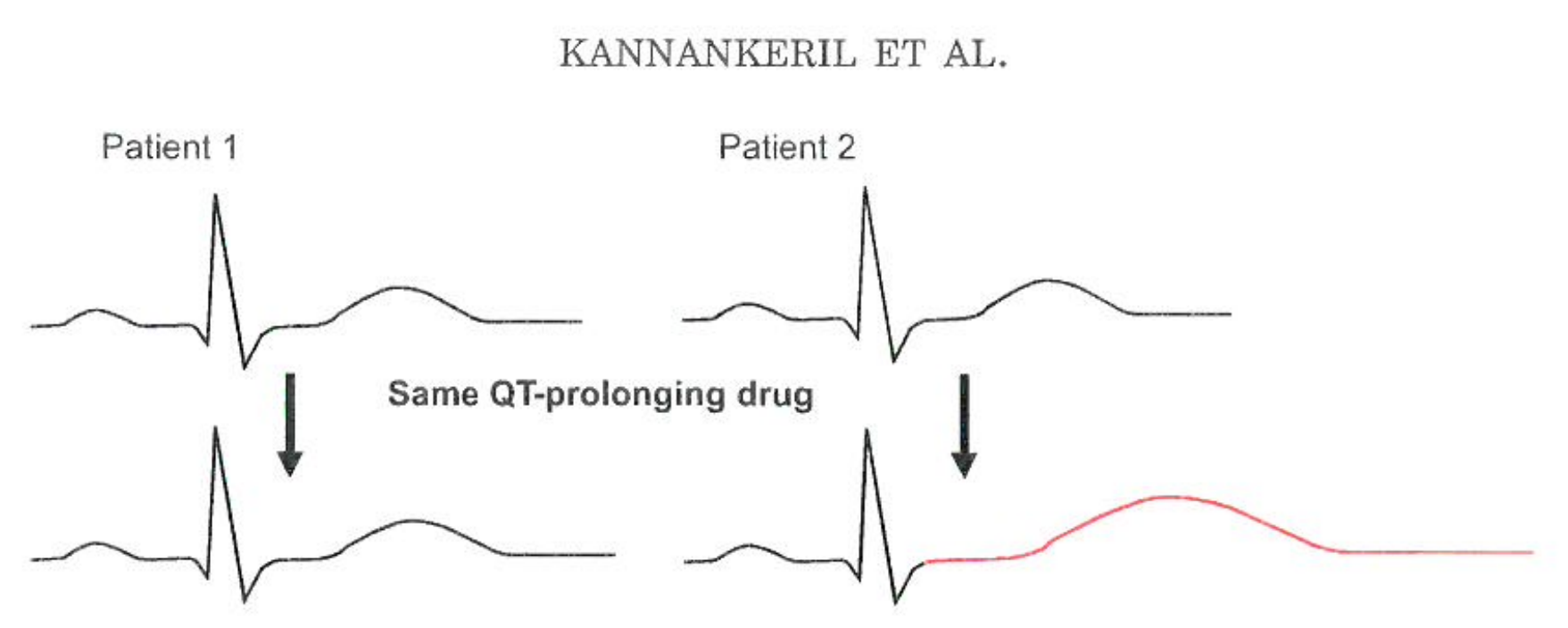
- Drug Induced
- Metabolic/electrolyte induced
- Very low energy diets / anorexia
- CNS & Autonomic nervous system disorders

Miscellaneous

- Coronary Artery Disease
- Mitral Valve Prolapse

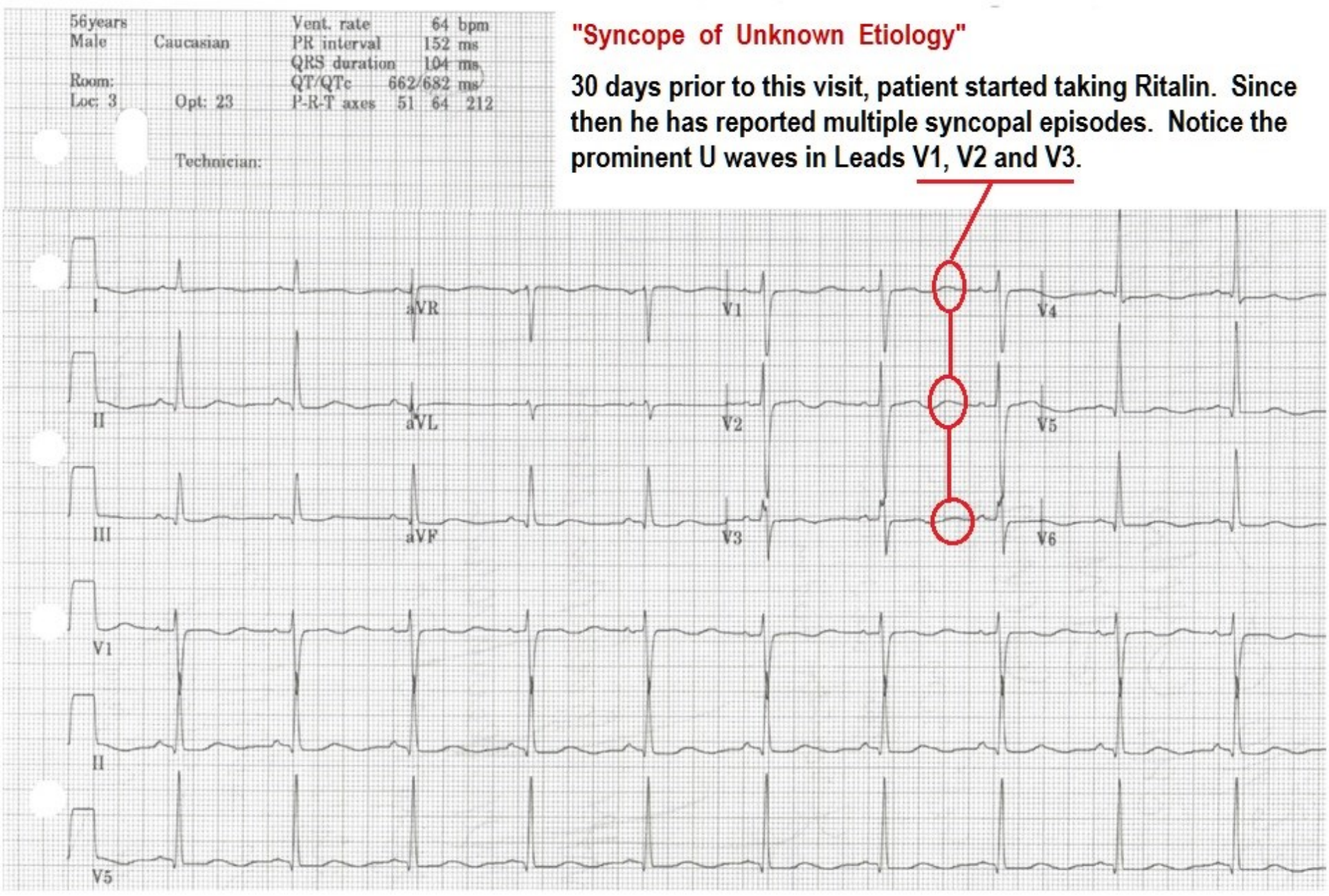
PATIENT 1: NORMAL

PATIENT 2: Genetic susceptibility; sensitivity to QT prolonging drugs:

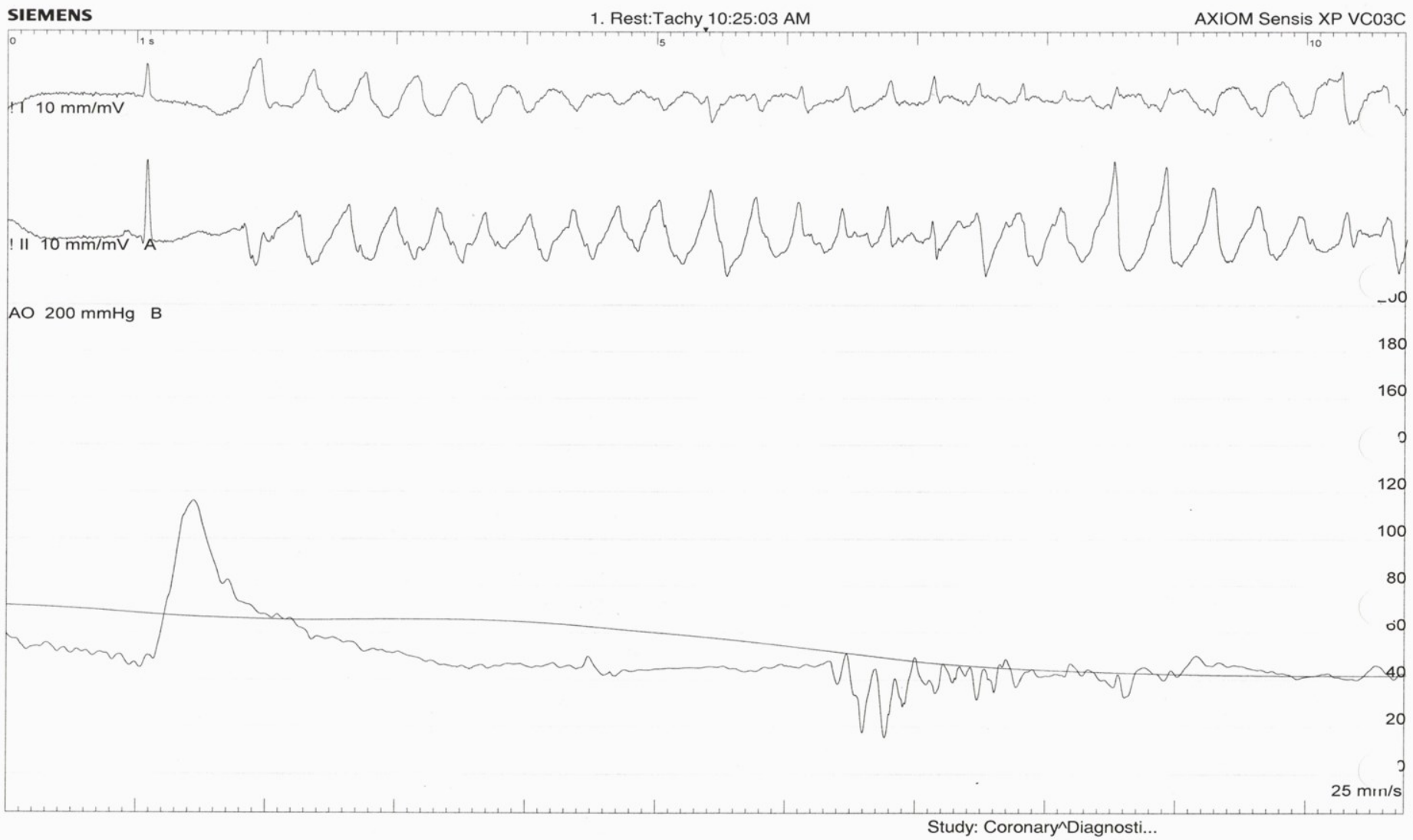


Click here for link to paper by Kannankeril et al (2010 Pharmacological Reviews) that describes genetic susceptibility described above.

Medication induced LQTS with TdP and Cardiac Arrest - Case Study: 56 year old male

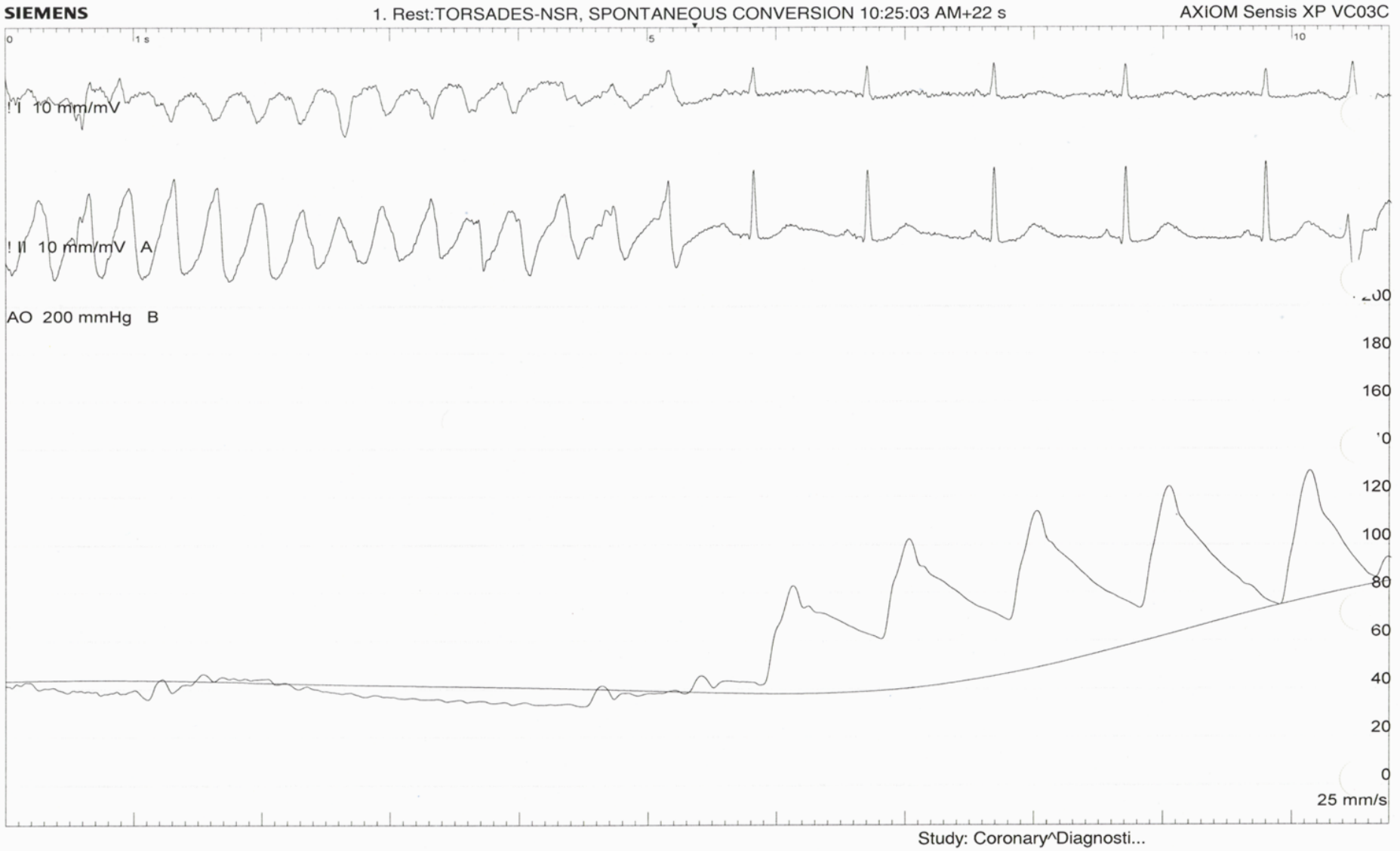


Medication induced LQTS with TdP and Cardiac Arrest - Case Study: 56 year old male



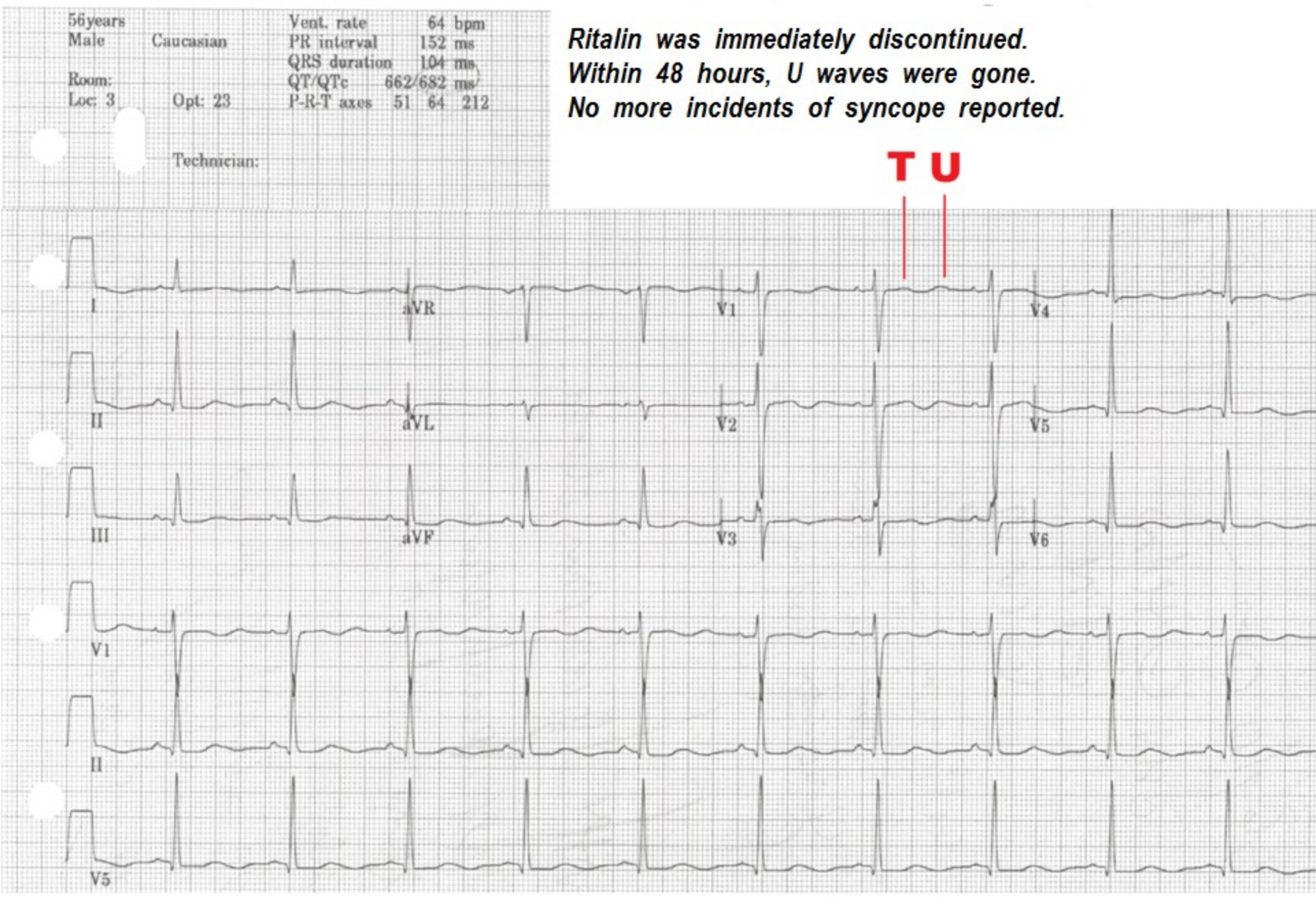
Run of Torsades de Pointes occurred during Cardiac Catheterization . . .

Medication induced LQTS with TdP and Cardiac Arrest - Case Study: 56 year old male

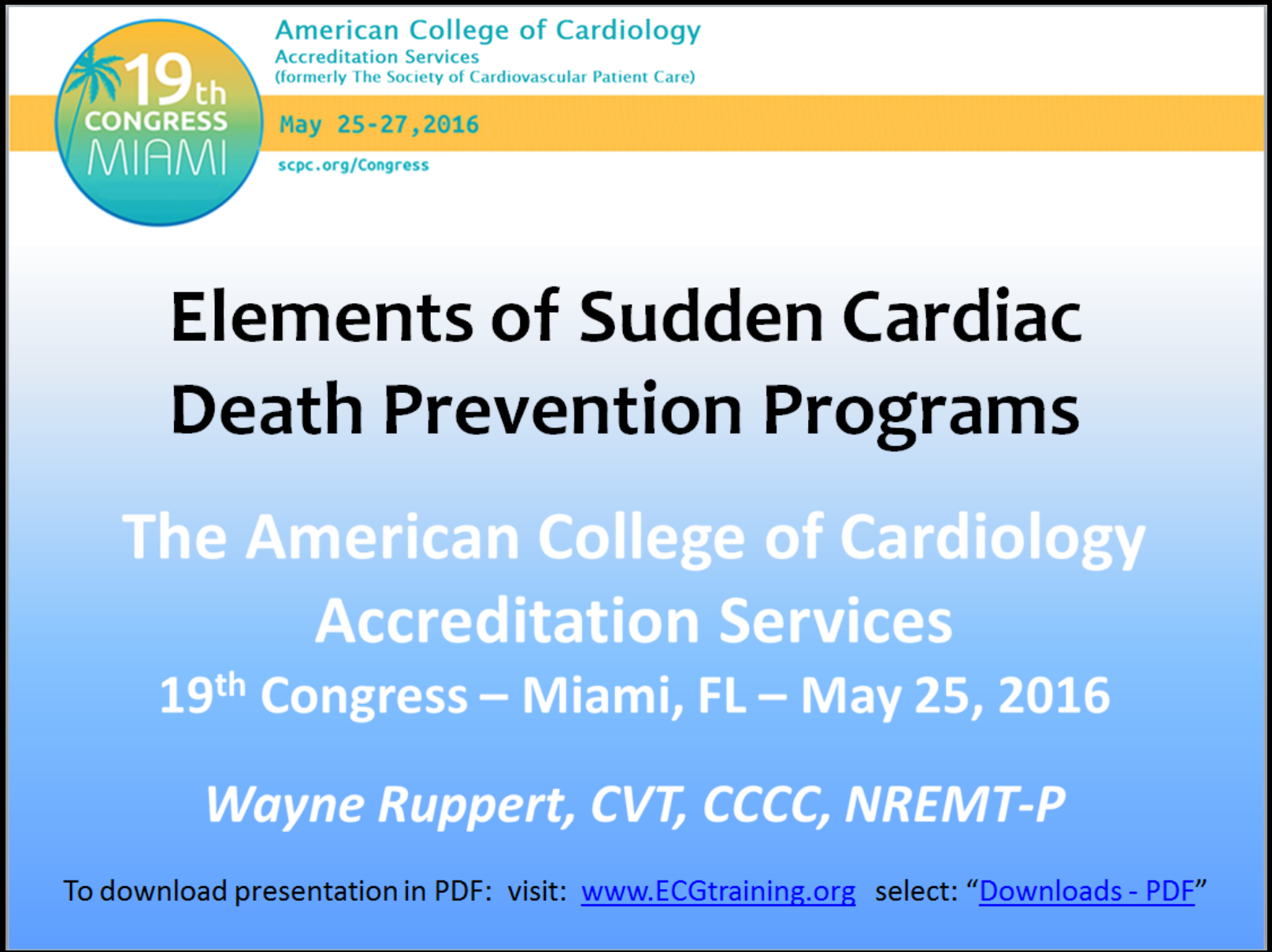


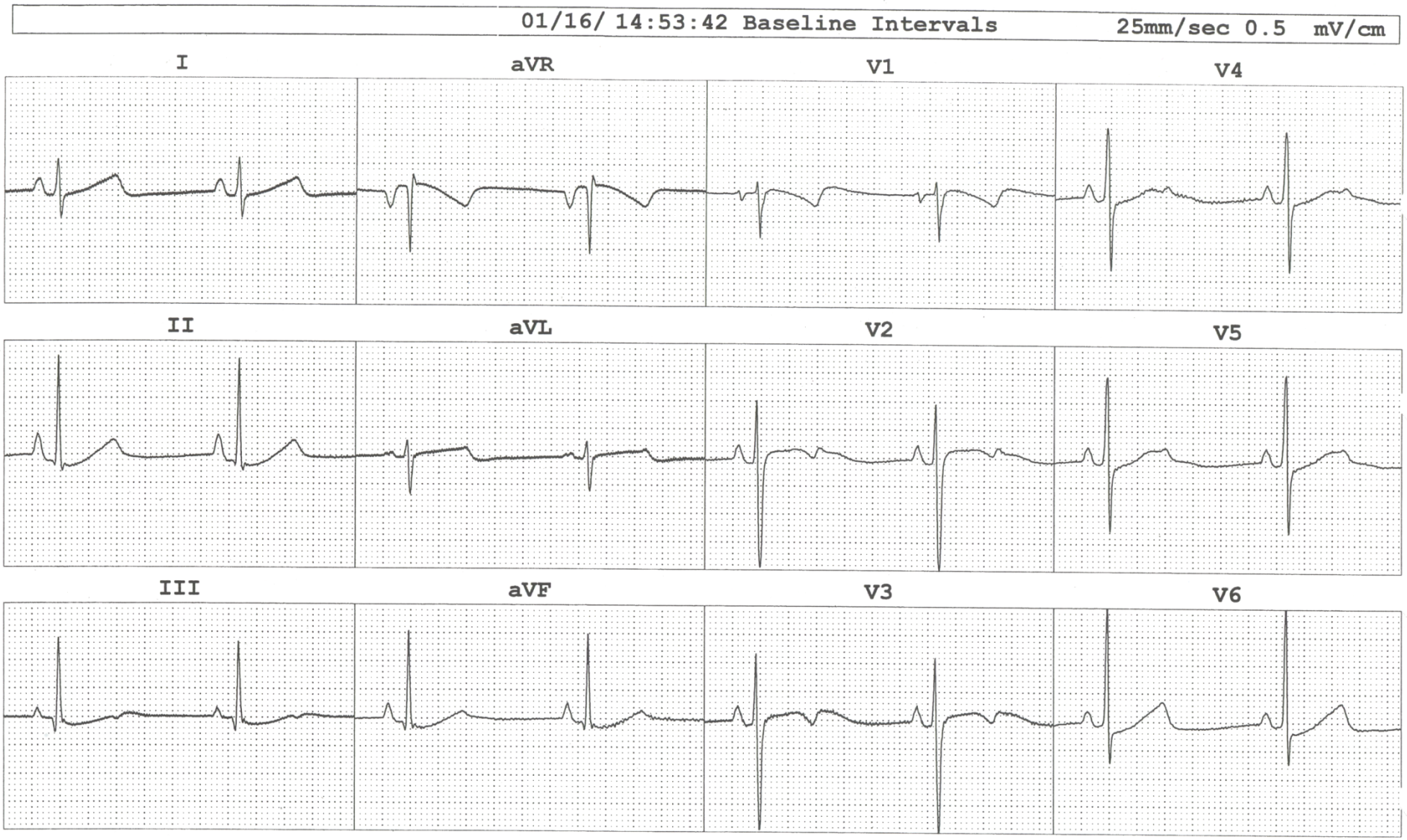
Torsades de Pointes self-terminates just before aborted Defibrillation

Medication induced LQTS with TdP and Cardiac Arrest - Case Study: 56 year old male

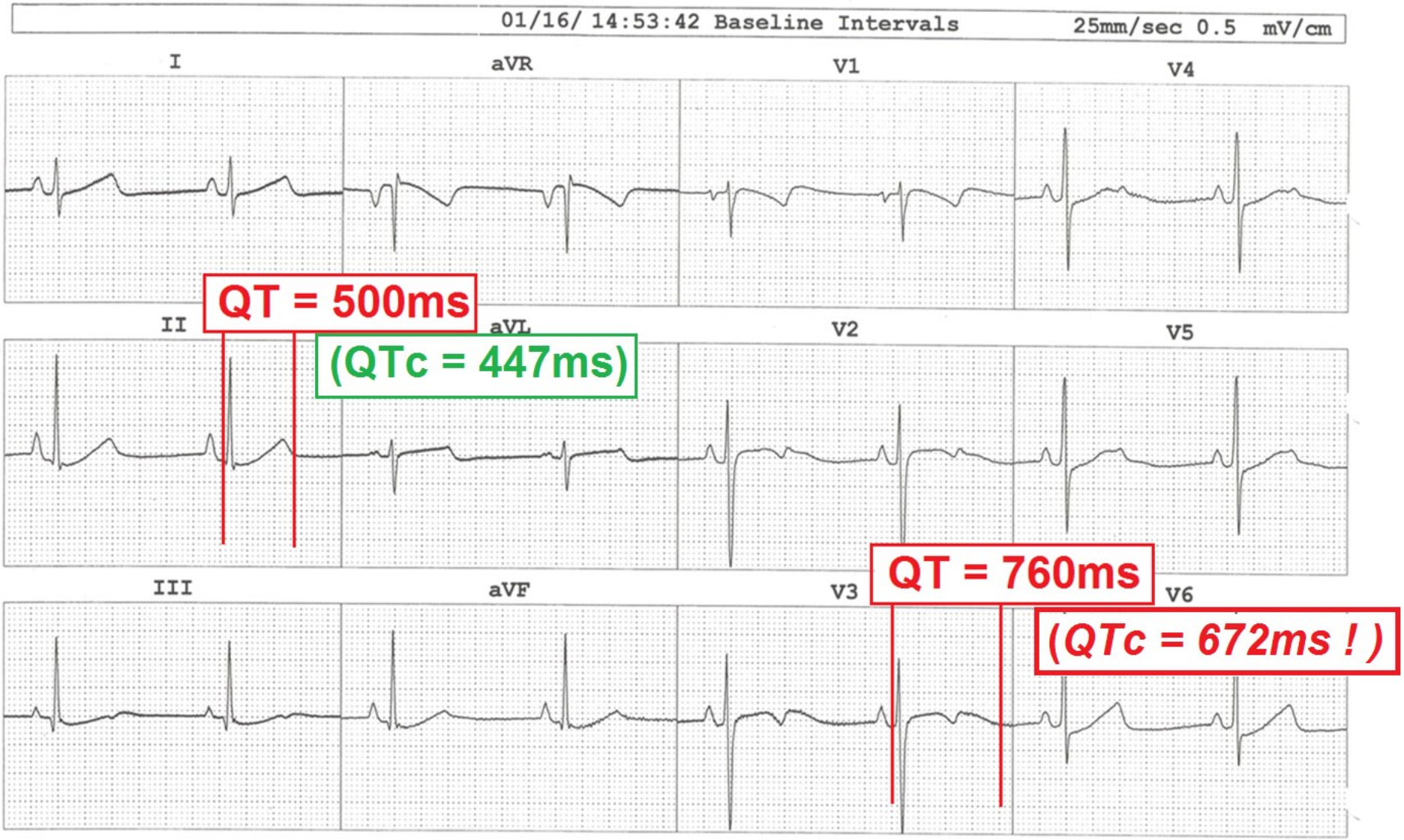


CLICK HERE to download "A SHORT Course in LONG QT Syndrome," a focused excerpt from:





15 year old male , suffered sudden cardiac arrest. Successful out-of-hospital resuscitation with CPR / AED. His ECG is shown below:



This ECG illustrates the degree of variation that can be noted between different leads on the 12 Lead ECG. ALWAY measure the QT Interval in the lead with the GREATEST value.

IS THERE ANYTHING ABNORMAL WITH THIS EKG ?

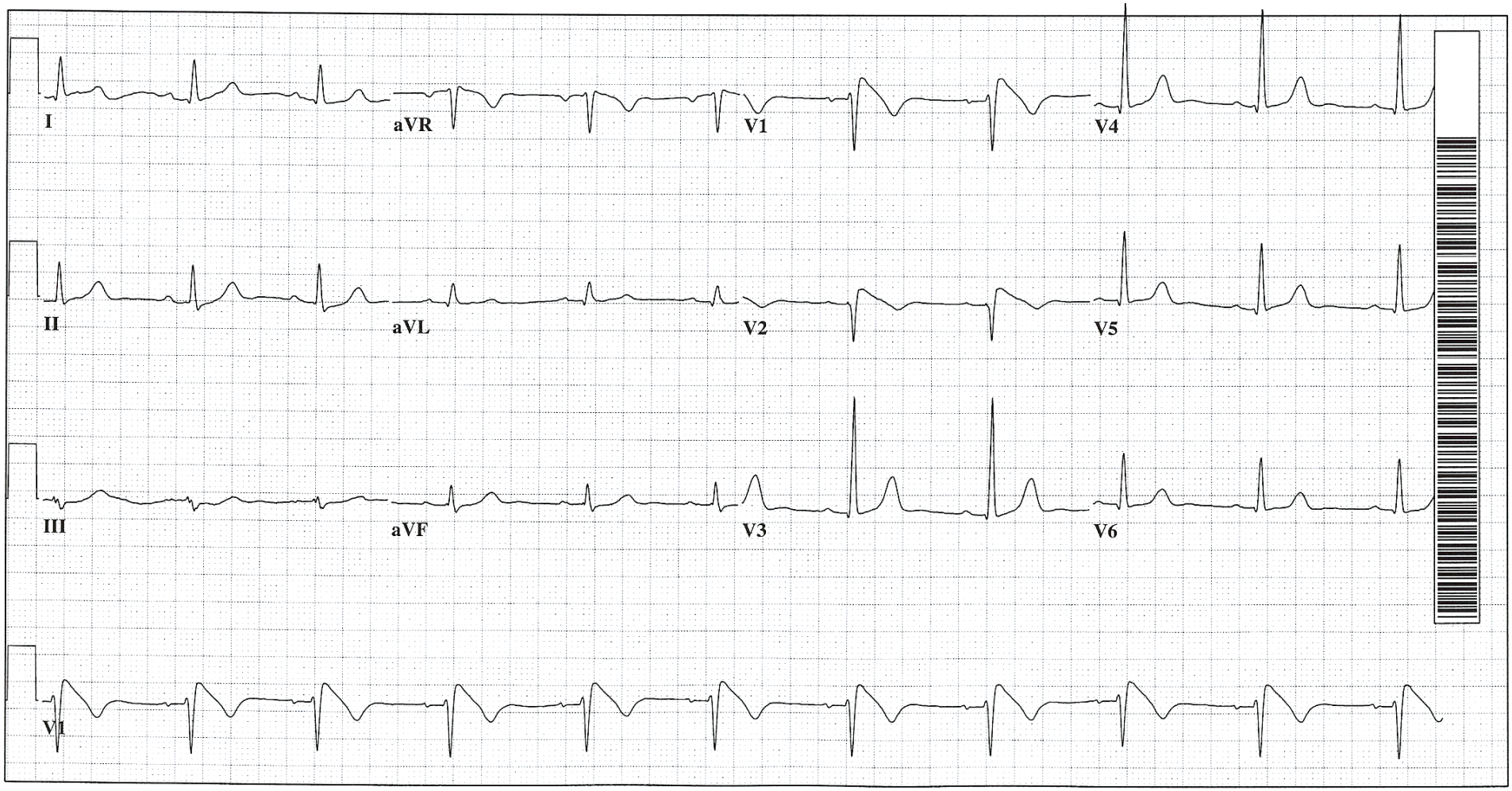
37 yr
Female Caucasian

Room:C4A
Loc:3 Option:23

Vent. rate	62	BPM
PR interval	180	ms
QRS duration	88	ms
QT/QTc	418/424	ms
P–R–T axes	37 22	47

Normal sinus rhythm
Normal ECG
No previous ECGs available

ECG of 37 year old female who suffered syncopal episode while driving, crashed into a tree, resulting in minor soft tissue injuries. . . . .



25mm/s 10mm/mV 40Hz 005D 12SL 235 CID: 3

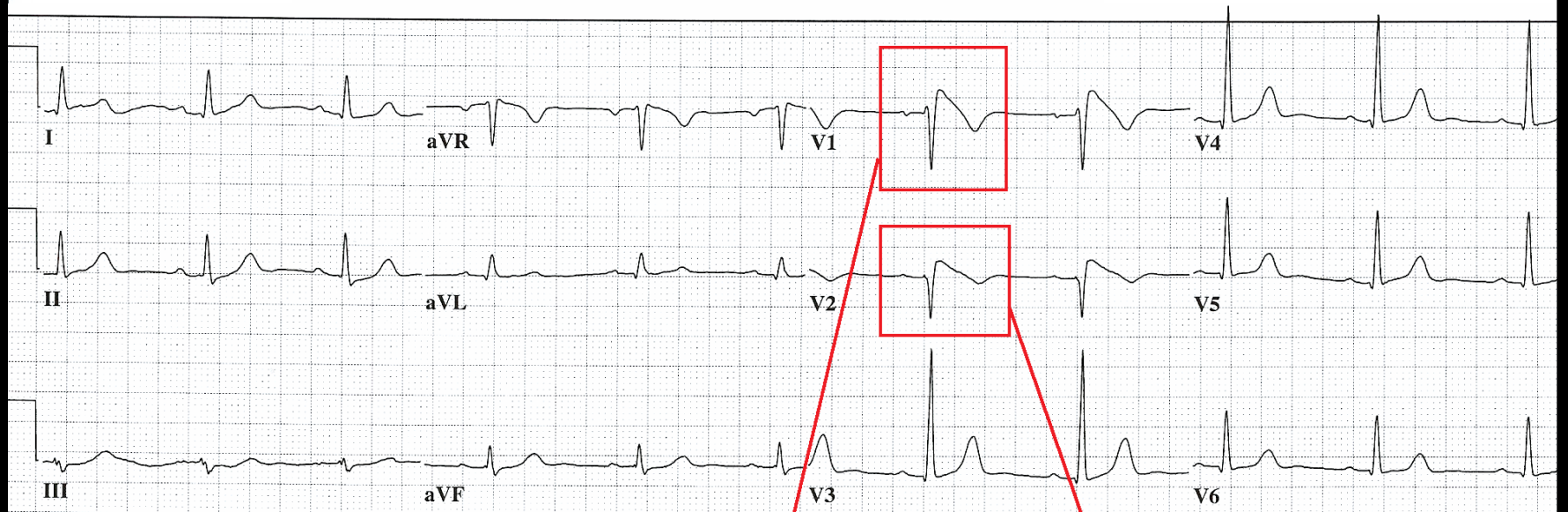
EID:46 EDT:

37 yr
Female Caucasian

Vent. rate	62	BPM
PR interval	180	ms
QRS duration	88	ms
QT/QTc	418/424	ms
P–R–T axes	37 22	47

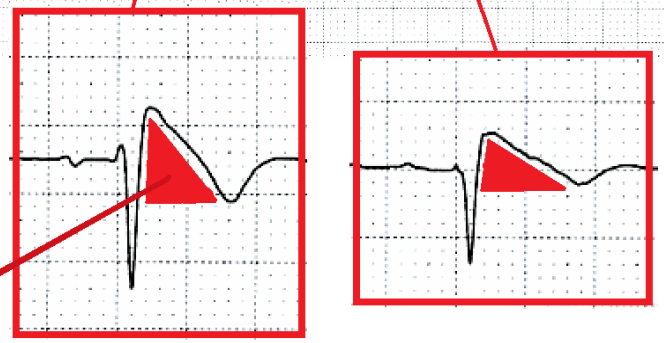
Normal sinus rhythm
Normal ECG
No previous ECGs available

NOTE COMPUTER INTERPRETATION !



THIS PATIENT EXHIBITS A "CLASSIC" TYPE I BRUGADA SYNDROME ECG PATTERN:
- ELEVATED J POINTS IN V1, V2
- DOWNSLOPING "COVED" ST SEGMENT
- INVERTED T WAVE.

NEVER FORGET THE "TRIANGULAR" SHAPE !



37 yr
Female Caucasian

Room:C4A
Loc:3 Option:23

Vent. rate	62	BPM
PR interval	180	ms
QRS duration	88	ms
QT/QTc	418/424	ms
P–R–T axes	37 22	47

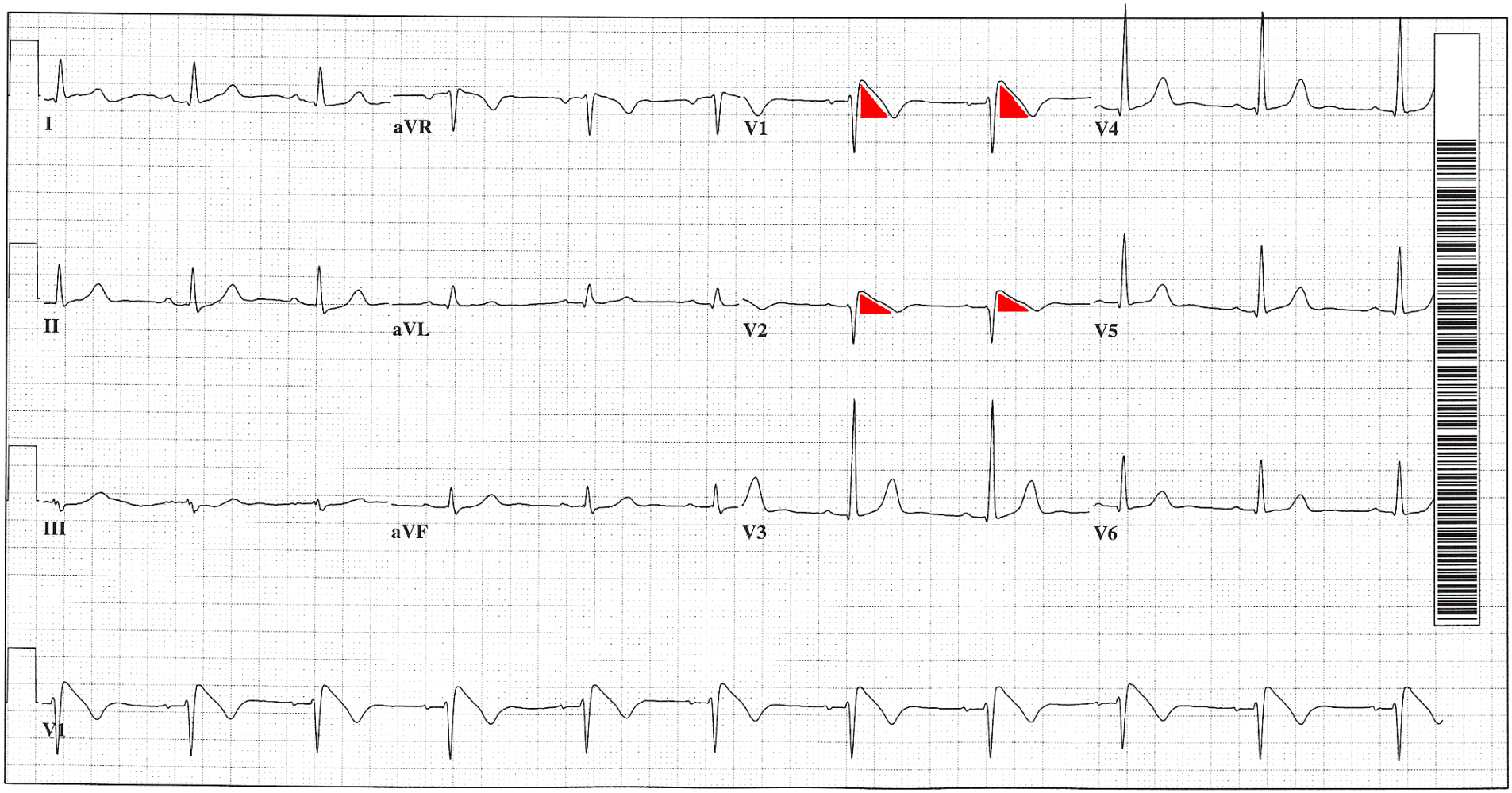
Normal sinus rhythm
Normal ECG
No previous ECGs available

SUSPECT
BRUGADA SYNDROME

- NOTE THE "TRIANGULAR" SHAPED S-T COMPLEXES . . . .

Technician:

Referred by:



25mm/s 10mm/mV 40Hz 005D 12SL 235 CID: 3

EID:46 EDT:

PATTERNS of S-T ELEVATION :

BEWARE of the

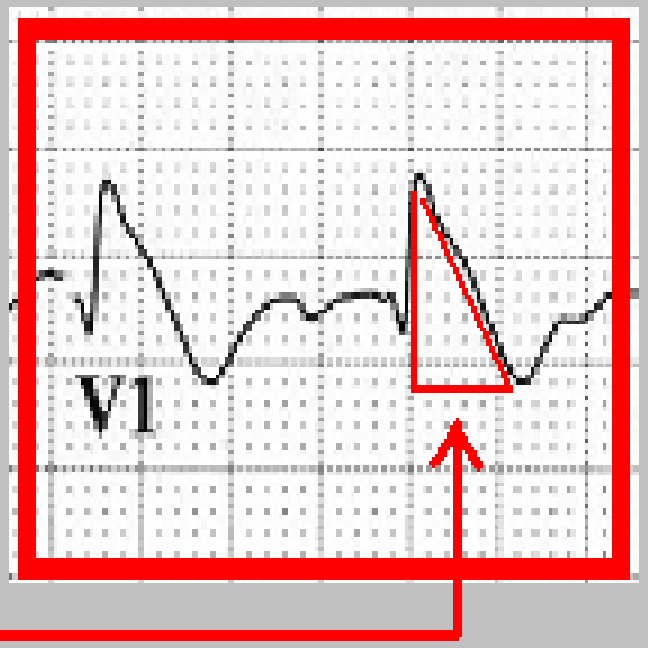
" TRIANGULAR "
SHAPED S-T SEGMENT
IN V1, V2, and some-
times also in V3 . . .

THINK - -

BRUGADA SYNDROME

BRUGADA SYNDROME

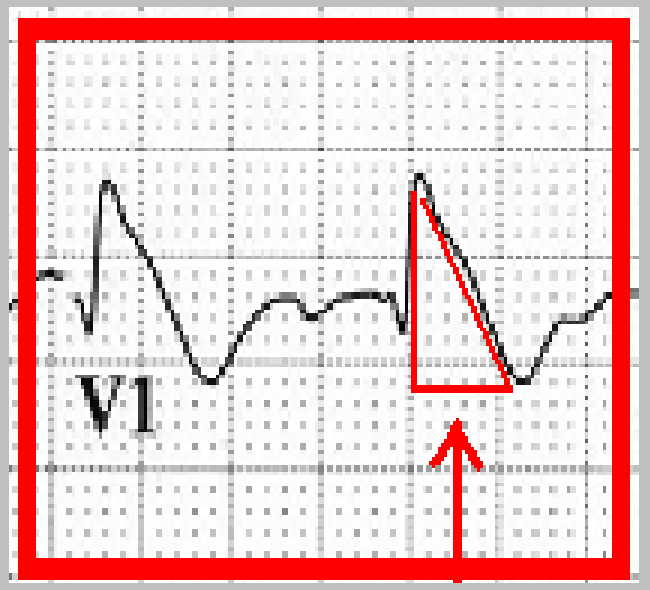
1. RBBB PATTERN
2. S-T ELEVATION
   V1, V2, possibly V3
3. ATYPICAL "TRIANGLE" SHAPED S-T SEGMENT



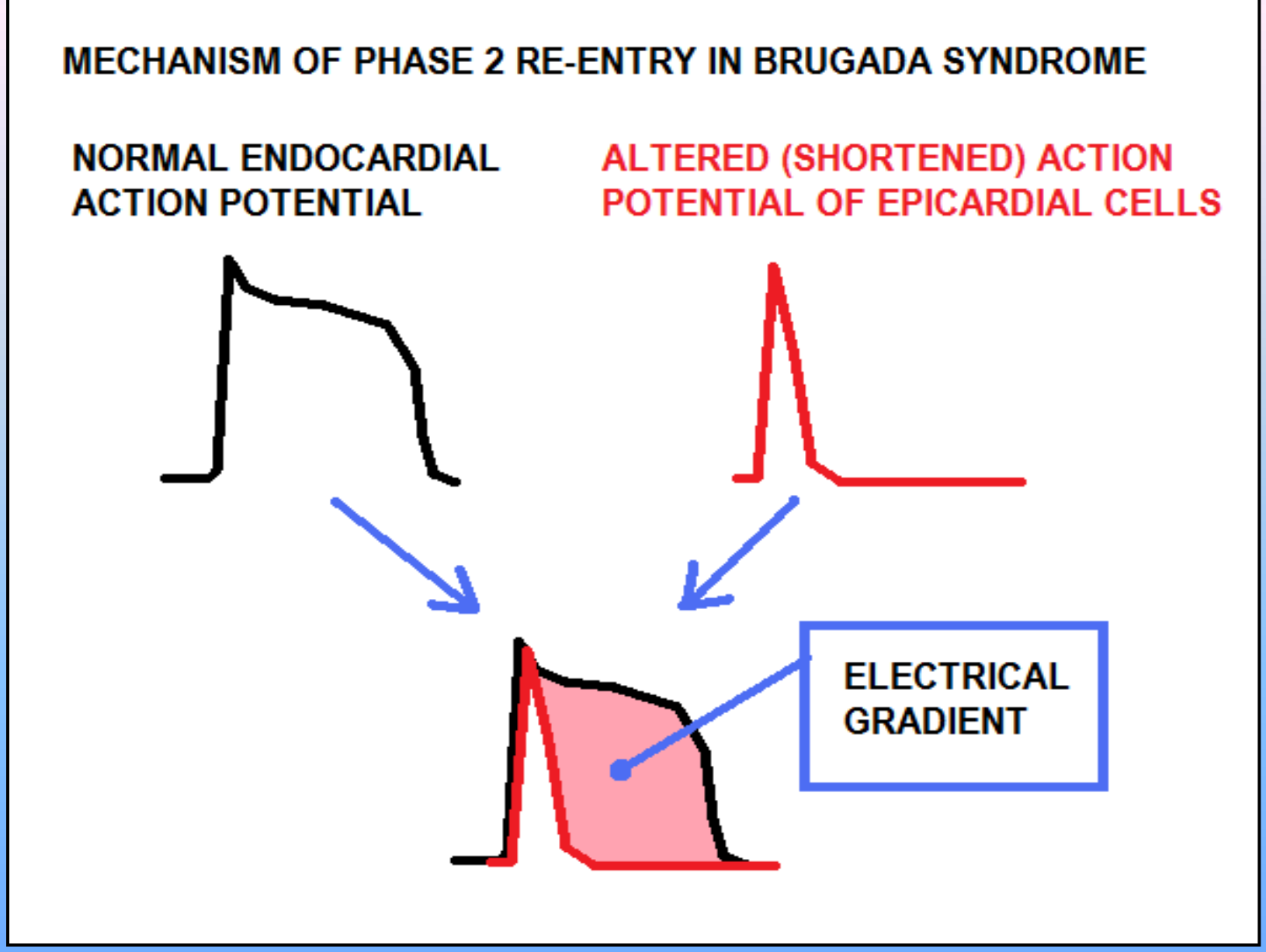
4. USUALLY EFFECTS YOUNG, HEALTHY PEOPLE
5. CAUSES SUDDEN DEATH by TORSADES

BRUGADA SYNDROME

- GENETIC DISORDER - GENE SCN5A, which encodes CARDIAC SODIUM CHANNELS.
- CAUSES EARLY RIGHT VENTRICULAR SUB-EPICARDIAL REPOLARIZATION



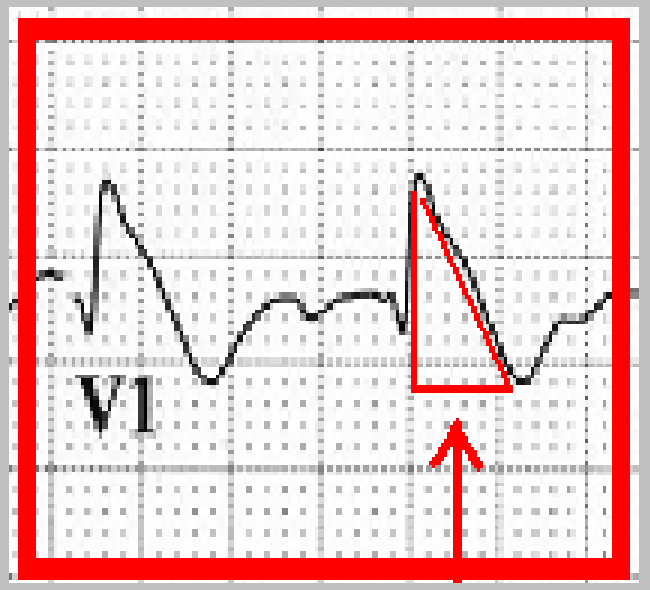
- CAUSES RUNS OF TORSADES de POINTES, and SUDDEN DEATH from TORSADES and V-FIB.
- IS BELIEVED TO CAUSE 4 - 12 % of ALL SUDDEN DEATHS, and 50 % of ALL CARDIAC DEATHS where pt. has a STRUCTUALLY NORMAL HEART.



Trigger for Torsades de Pointes – ECTOPIC BEAT during The "ELECTRICAL GRADIENT" phase shown above.

BRUGADA SYNDROME - TESTING

- For CONCEALED cases, a drug study of AJMALINE, FLECAINIDE, or PROCAINAMIDE can UNMASK the "tell-tale" TRIANGULAR COMPLEXES of V1 and V2.



- IN EP STUDIES, a PROLONGED H-V INTERVAL may be observed.
- GENETIC TESTING is performed by THE RAMON A. BRUGADA FOUNDATION.

CLICK HERE to download 2017 ACC AHA HRS Guideline- Eval and Mgmt of Syncope

2017 ACC/AHA/HRS guideline for the evaluation and management of patients with syncope

A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society

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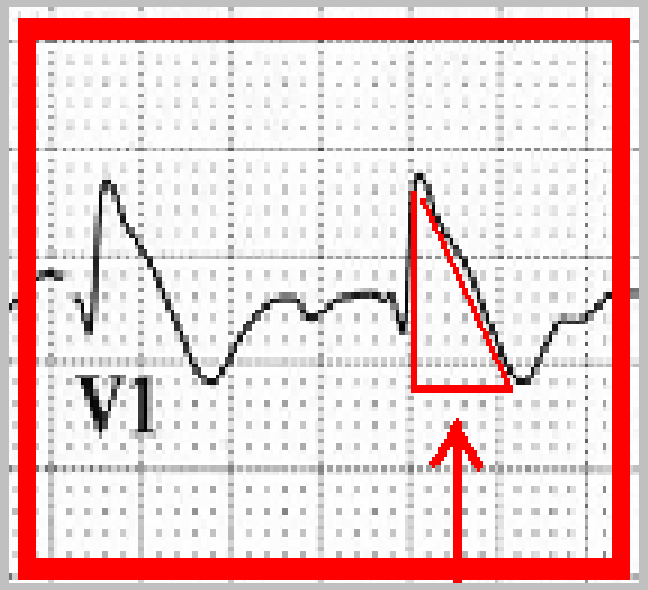
BRUGADA SYNDROME - TREATMENT

ICD implantation is the only known effective treatment to date.



www.BRUGADA.org

V-FIB & PULSELESS VT

V - FIB	
MONOMORPHIC V - TACH	
TORSADES de POINTES / Polymorphic VT	

ABCs (CAB)

CPR -- — - SEND FOR MONITOR / DEFIB or AED; - CALL CODE or 911 (DELEGATE !)

ASSESS ECG — SHOCK x 1 — 120 - 200 j BiPHASIC / 360 j MONOPHASIC

Switch Rescuers — CPR - 2 min — - START IV / IO; - ADVANCED AIRWAY

ASSESS ECG — SHOCK x 1 — 120 - 200 j BiPHASIC / 360 j MONOPHASIC

Switch Rescuers — CPR - 2 min — - EPI 1 mg every 5 min

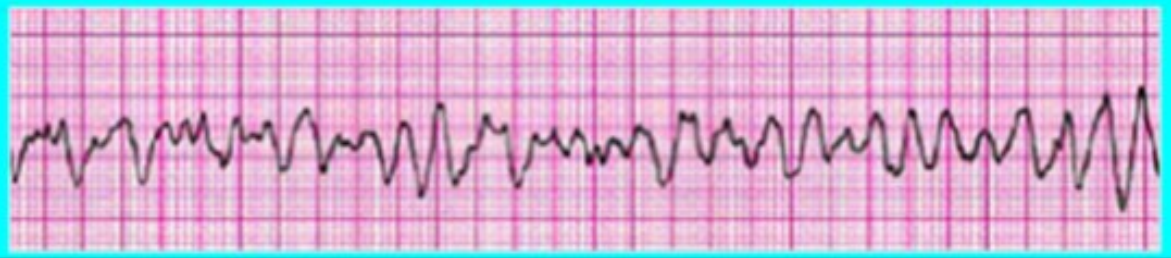
ASSESS ECG — SHOCK x 1 — 120 - 200 j BiPHASIC / 360 j MONOPHASIC

Switch Rescuers — CPR - 2 min — - AMIODARONE (1st DOSE - 300 mg) (2nd DOSE - 150 mg)

AFTER AMIODARONE - CONSIDER MAGNESIUM SULFATE 1 - 2 gm AND / OR LIDOCAINE 1.5 mg/kg

PULSELESS TORSADES: USE MAGNESIUM SULFATE FIRST

V-FIB & PULSELESS VT

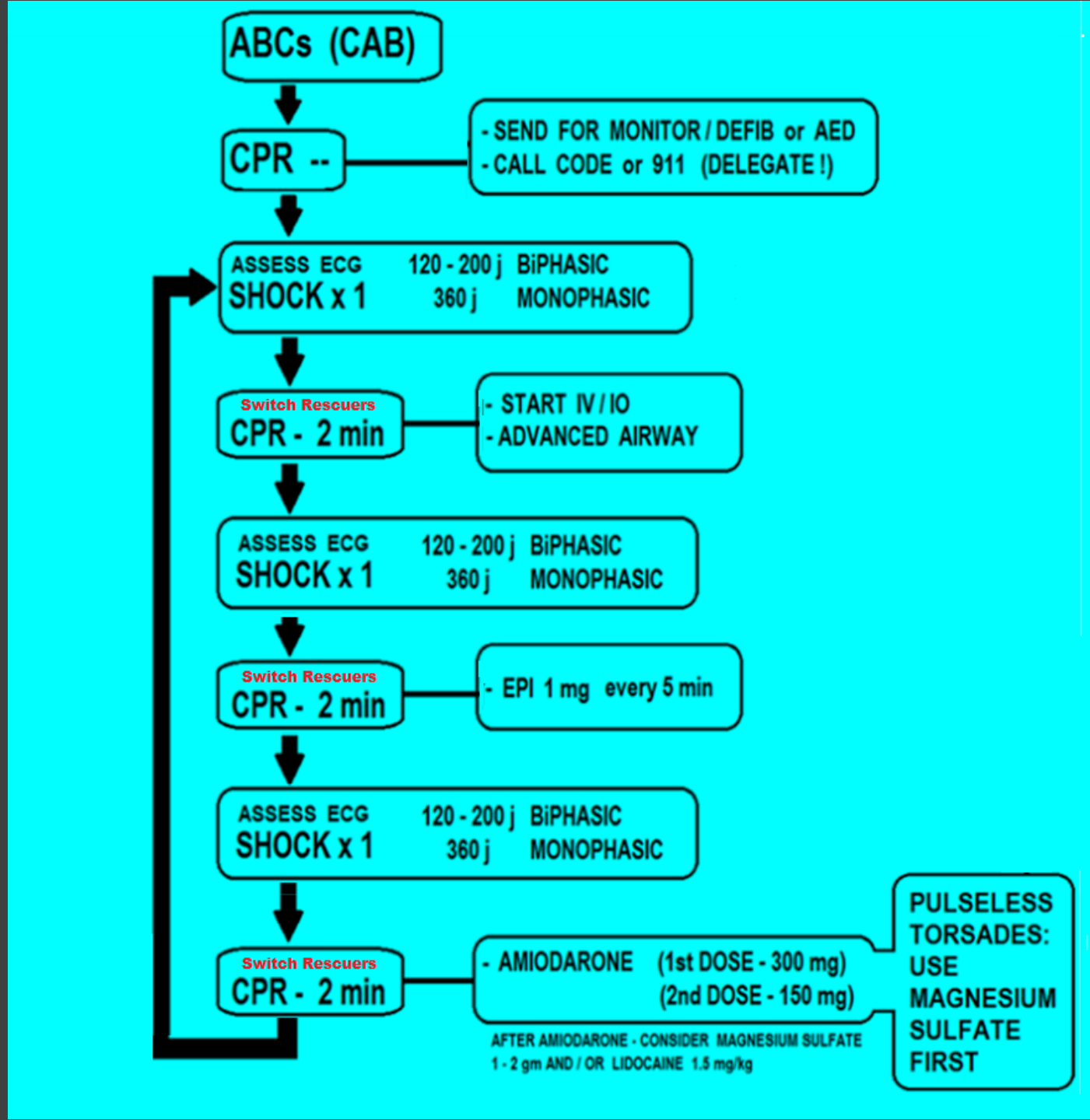
V - FIB	
MONOMORPHIC V - TACH	
TORSADES de POINTES / Polymorphic VT	

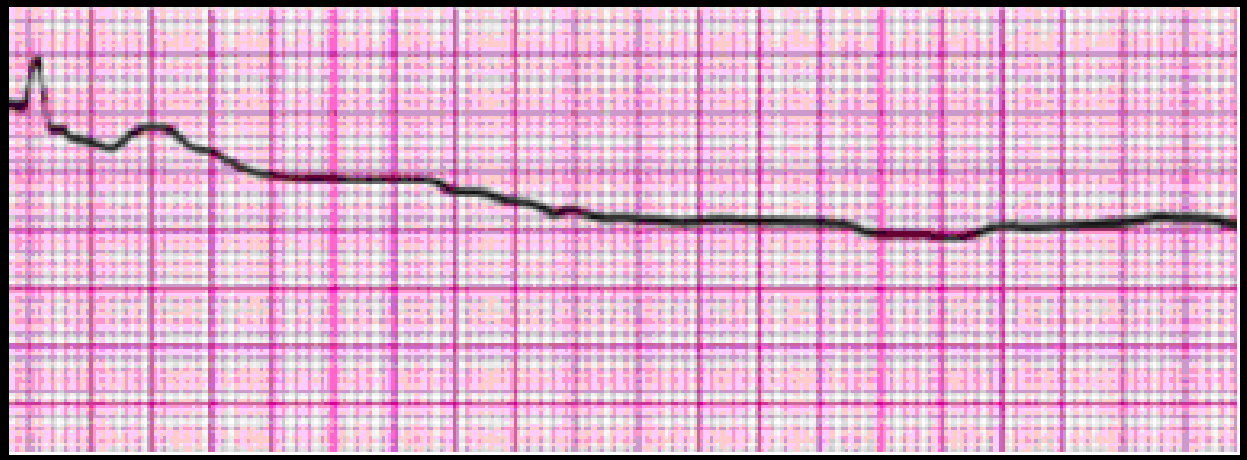
ABCs (CAB)

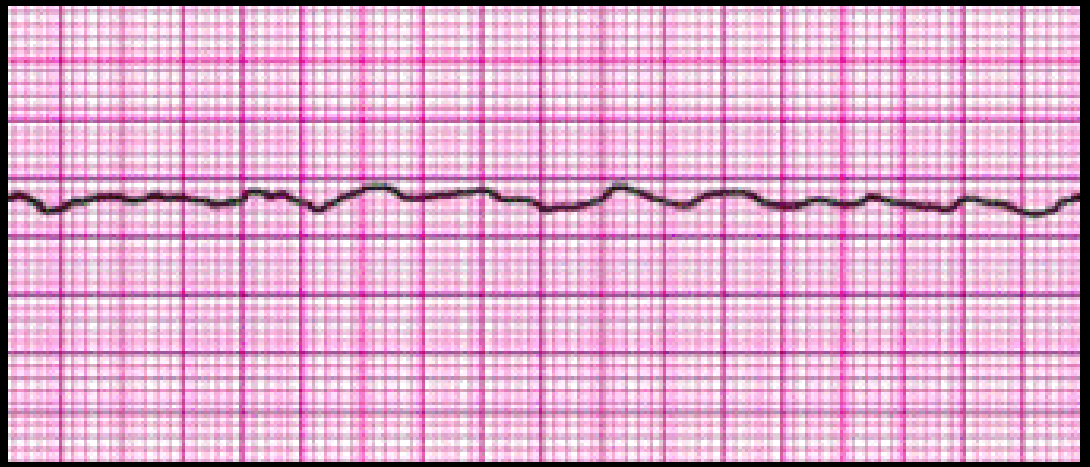
CPR --

- SEND FOR MONITOR / DEFIB or AED
- CALL CODE or 911 (DELEGATE !)

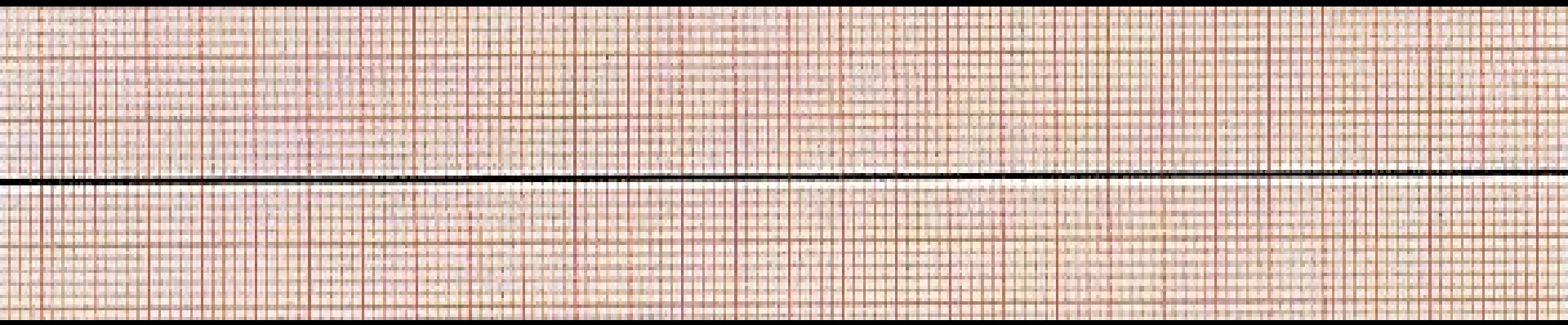
VF & Pulseless VT Algorithm





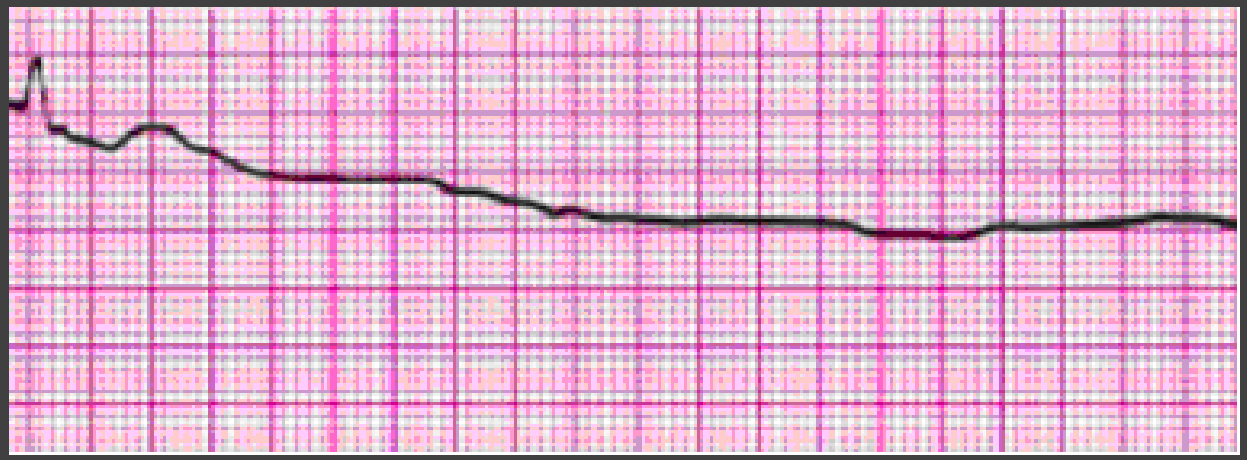


CPR
|
IV / AIRWAY
|
EPI 1 mg
|

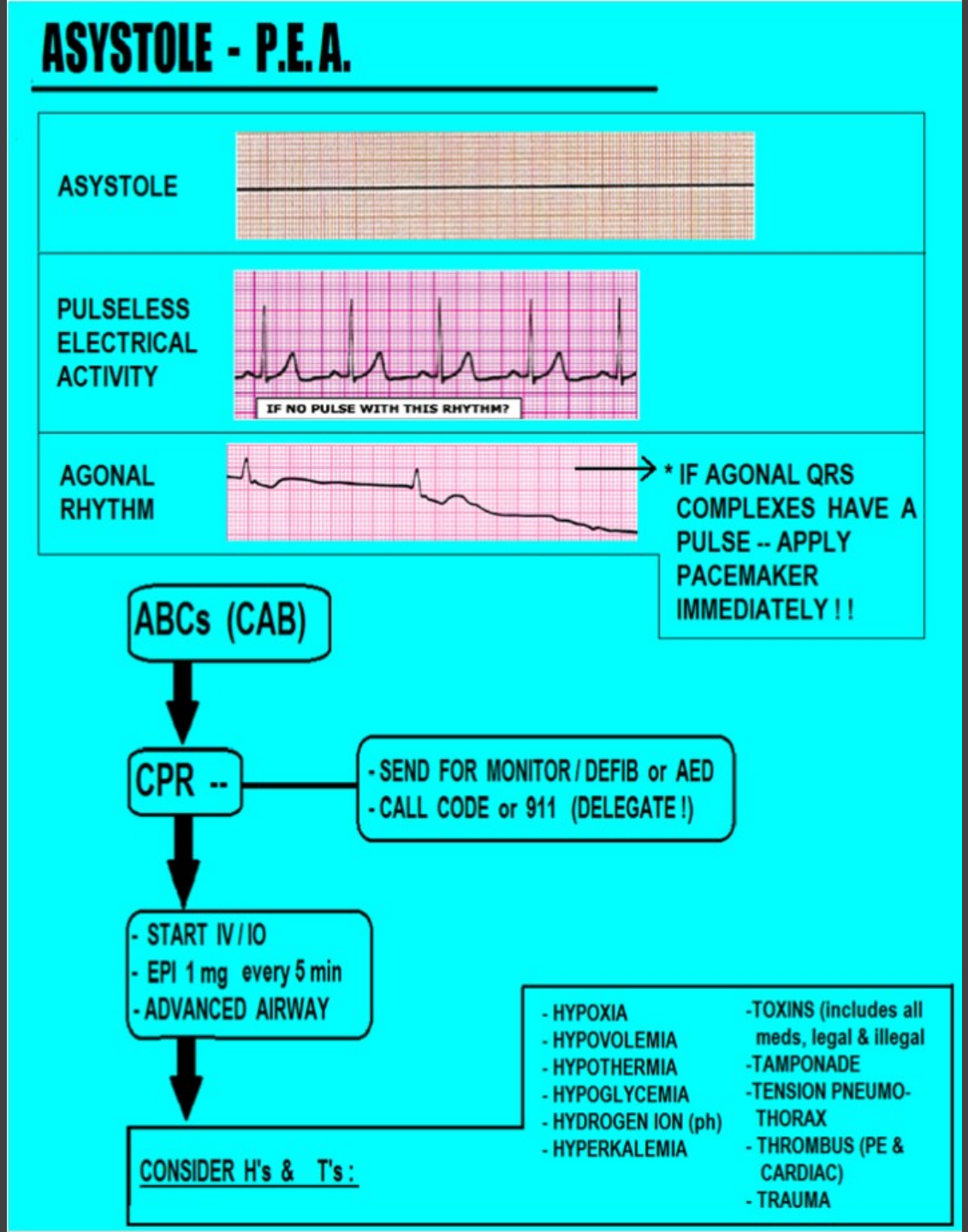


If QRS complexes have a PULSE then apply

PACEMAKER ! !

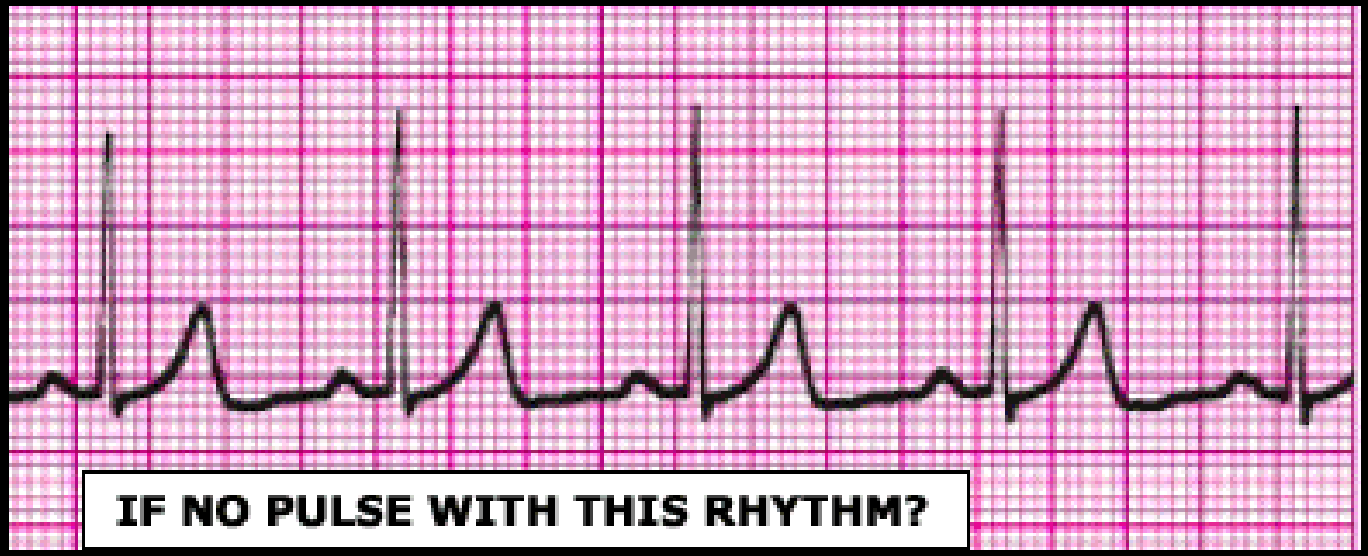


CPR
|
IV / AIRWAY
|
EPI 1 mg
|



Every TWO MINUTES . . .

- Do PULSE CHECK / ECG eval.
- SWITCH CHEST Compressors



CPR

|

IV / AIRWAY

|

EPI 1 mg

AND THEN . . . . ?

THE " H's " and the " T's "

- HYPOVOLEMIA
- HYPOXIA
- HYDROGEN ION (Ph)
- HYPOGLYCEMIA
- HYPOTHERMIA
- HYPERKALEMIA

- TOXINS
- TAMPONADE (CARDIAC)
- TENSION PNEUMOTHORAX
- THROMBOSIS (CORONARY or PULMONARY)
- TRAUMA

ECG Indicators of STEMI:

“Abnormal ST Elevation in TWO or more CONTINGUOUS LEADS” . . .

“Abnormal ST Elevation” . . .

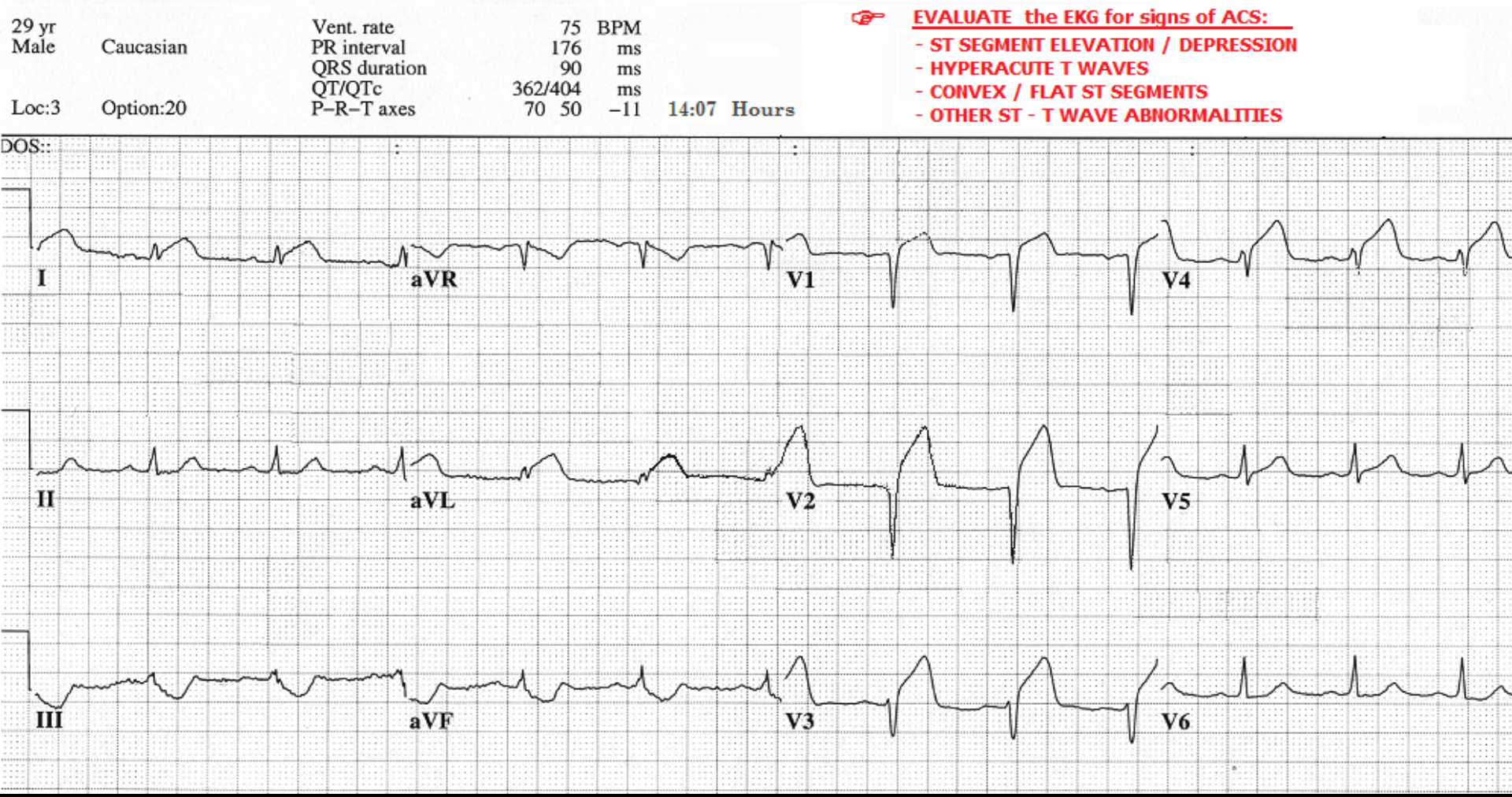
Abnormal ST Elevation Criteria: ACC/AHA 2009
“Standardization and Interpretation of the ECG, Part VI Acute Ischemia and Infarction,” Galen Wagner, et al

Leads V2 & V3:

- Men < age 40: up to 2.5 mm (.25mv)
- Men 40 +: up to 2.0 mm (.20mv)
- Women (all): up to 1.5 mm (.15mv)

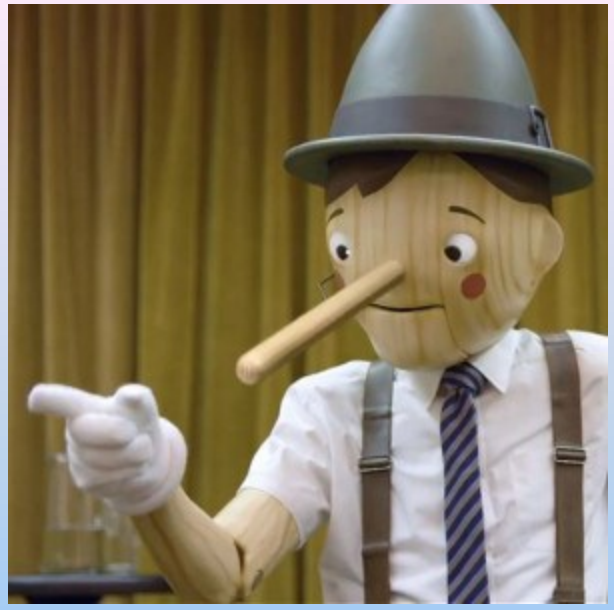
All other Leads of 12 Lead ECG:

- All patients: up to 1.0 mm (.10mv)



Not all STEMIs this obvious . . .

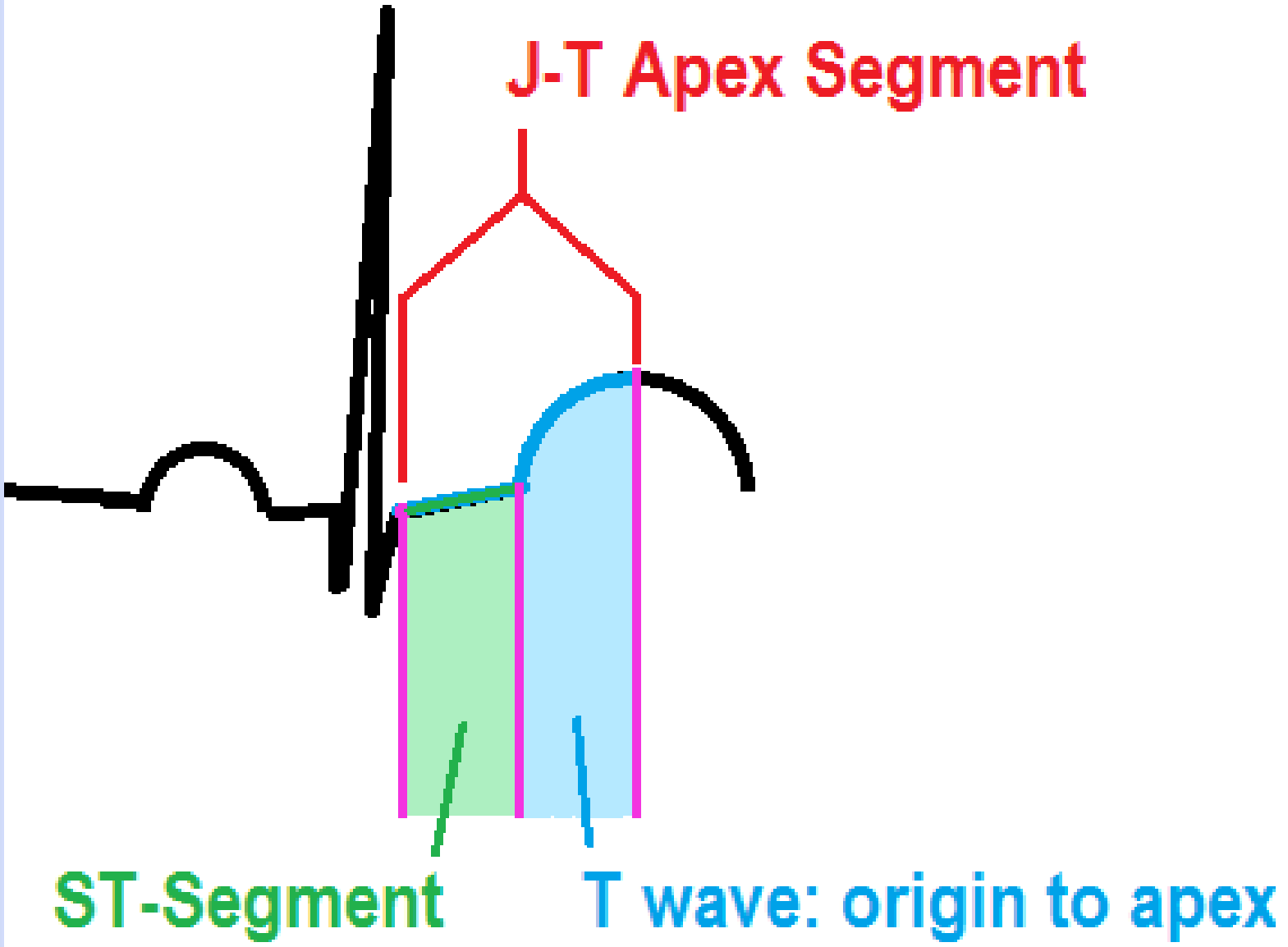
EKGs DON'T ALWAYS TELL THE TRUTH. . .



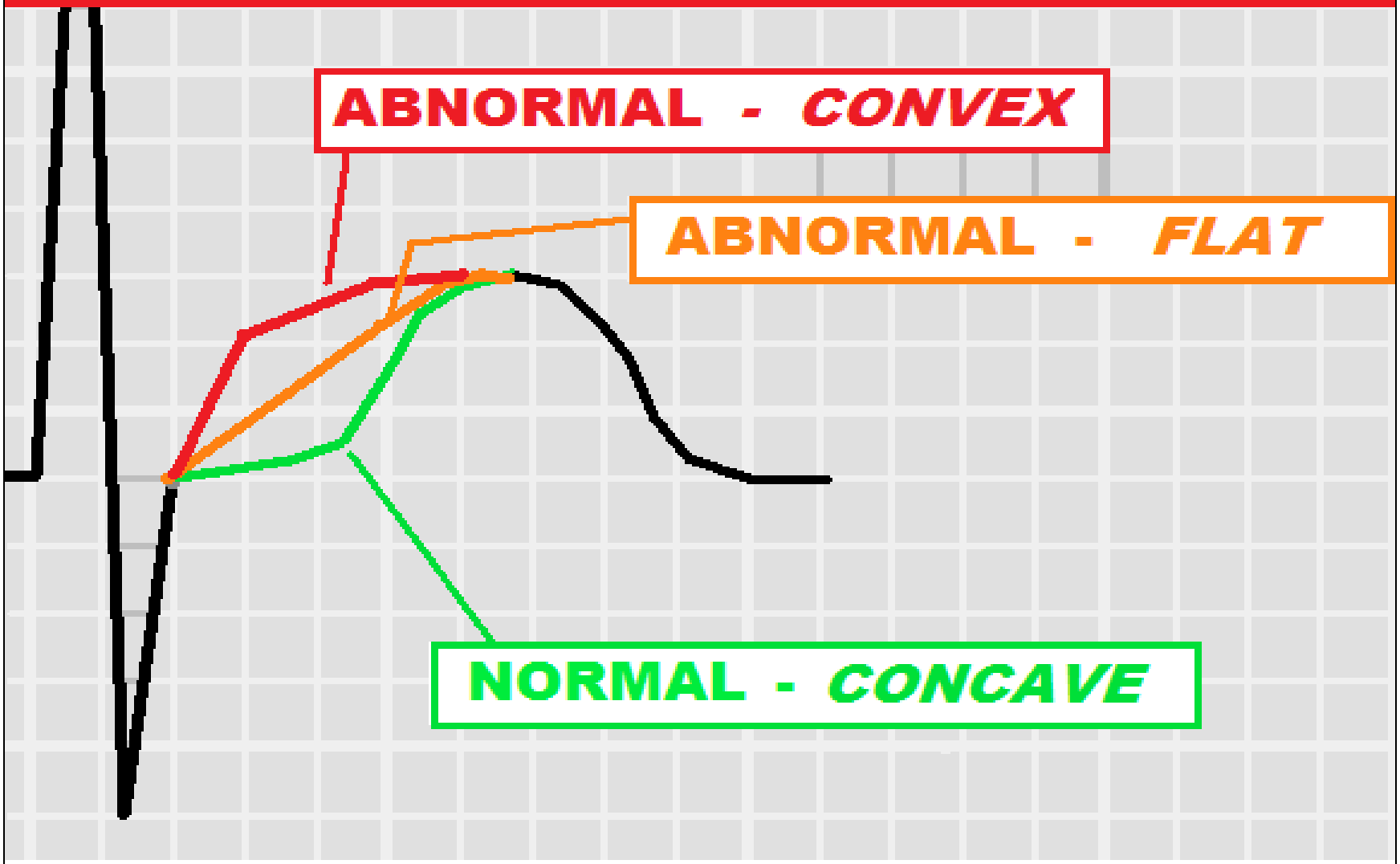
"When evaluating the ECG , there is always an undesirable degree of LACK OF SENSITIVITY ("false negatives") and LACK OF SPECIFICITY ("false positives").

ECG Patterns associated with "EARLY PHASE MI:"

- *J-T Apex abnormalities*
- *Hyper-Acute T Waves*
- *Dynamic ST-T Wave Changes on Serial ECGs*


J-T Apex Segment
ST-Segment
T wave: origin to apex

J - T APEX SEGMENT VARIATIONS



PATTERNS of EARLY INFARCTION

-- FLAT and CONVEX J-T APEX SEGMENTS

56 yr
Male Caucasian

Room:A9
Loc:3 Option:23

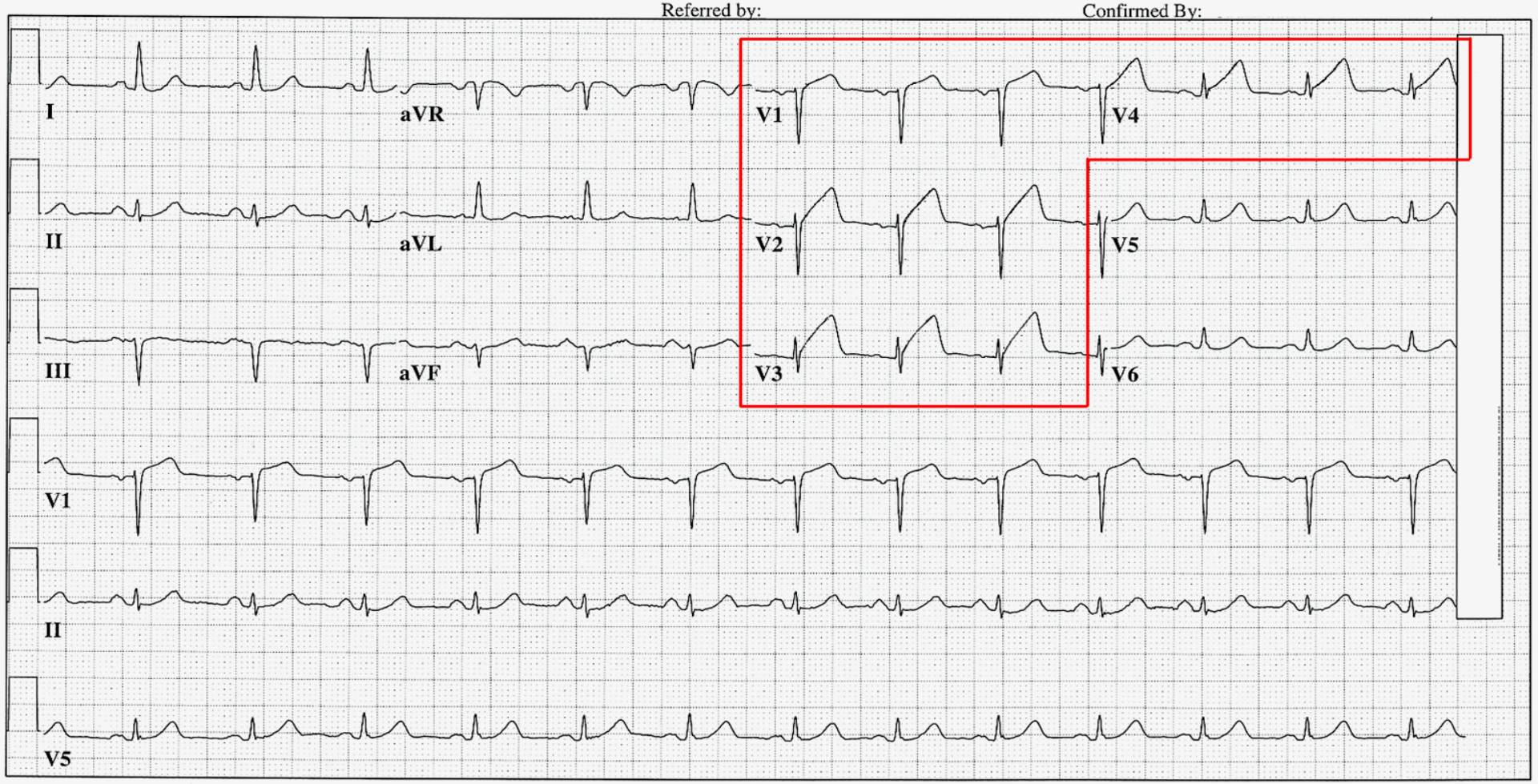
Vent. rate	80	BPM
PR interval	154	ms
QRS duration	78	ms
QT/QTc	380/438	ms
P–R–T axes	51 –24	38

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

Normal sinus rhythm
Normal ECG
No previous ECGs available

Technician: W Ruppert

Referred by: Confirmed By:

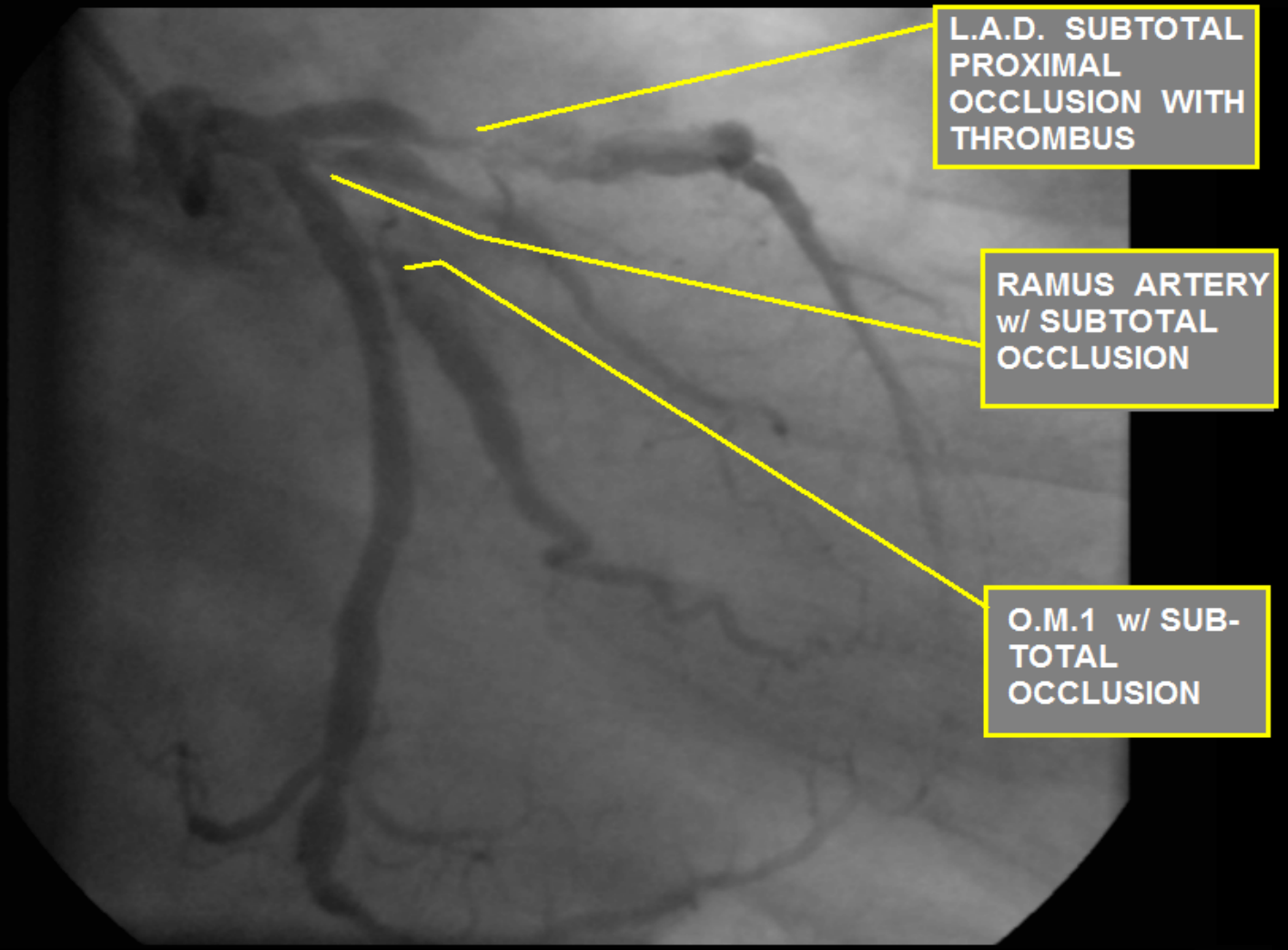


25mm/s 10mm/mV 40Hz 005C 12SL 235 CID: 3

EID:10 EDT:

ECG COMPUTER DOES NOT NOTICE THE CONVEX J-T APEX SEGMENTS !

CASE STUDY: 56 y/o male with INTERMITTENT "CHEST HEAVINESS" . . . .

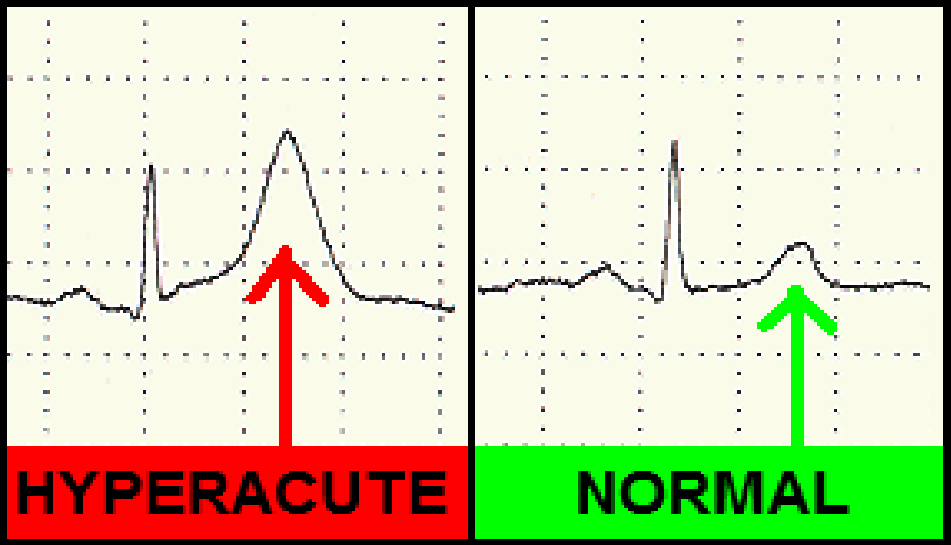


TREATMENT PLAN : EMERGENCY CORONARY ARTERY BYPASS SURGERY (4 VESSEL)

ECG Patterns associated with "EARLY PHASE MI:"

- *J-T Apex abnormalities*
- *Hyper-Acute T Waves*
- *Dynamic ST-T Wave Changes on Serial ECGs*

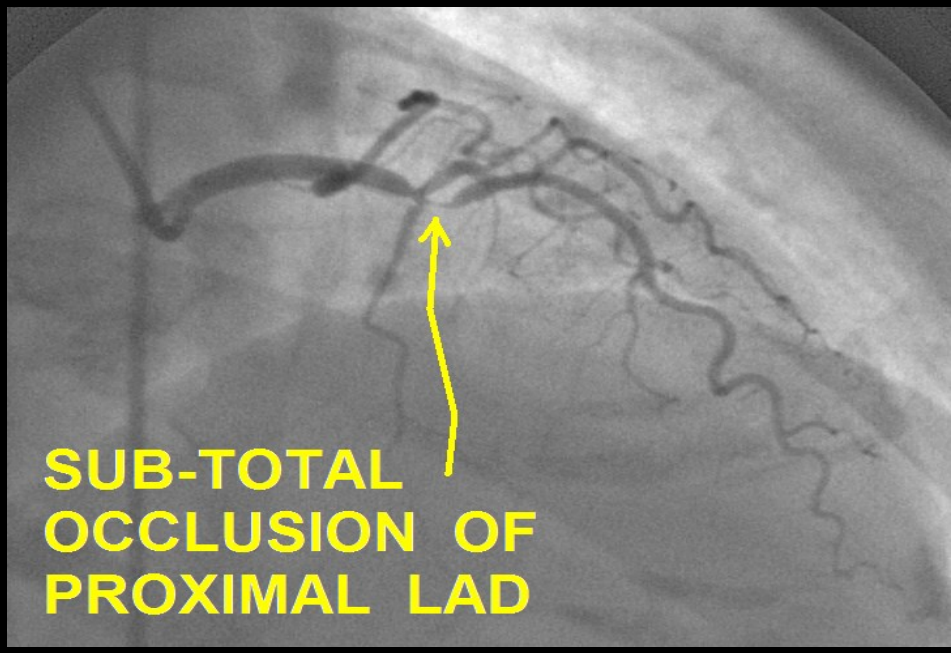
HYPERACUTE T WAVES



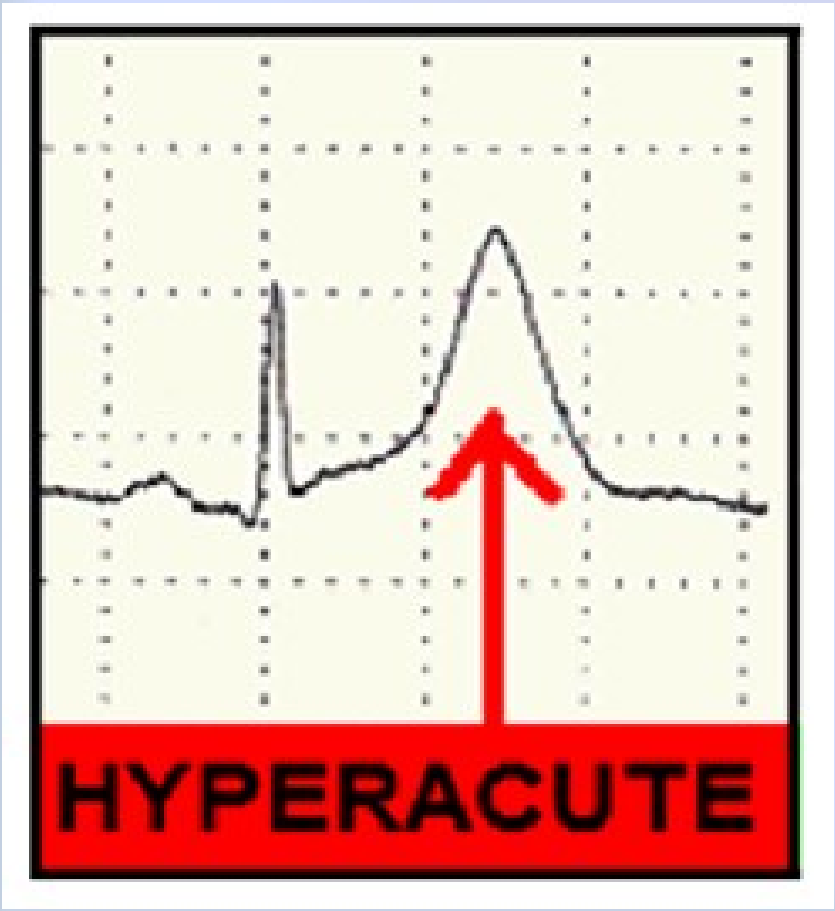
BOOK PAGE: 88

HYPER-ACUTE T WAVES - COMMON ETIOLOGIES:

CONDITION:	SEE PAGE(S):
HYPERKALEMIA	xx - xx
ACUTE MI	xx - xx
TRANS-MURAL ISCHEMIA	xx - xx
HYPERTROPHY	xx - xx



HYPERACUTE T Waves may indicate:



- **Early phase Acute MI**
- **Transmural ischemia** (usually seen in one region of the ECG)
- **Hyperkalemia** (seen globally across ECG)
- **Hypertrophy**

Helpful Clue: Hyper-Acute T Waves

- **GLOBAL Hyper-acute T Waves** (in leads viewing multiple myocardial regions / arterial distributions) **favors HYPERKALEMIA**

ID: 23-Nov- REGIONAL MEDICAL CENTER

55years
Female Caucasian

Room:

Vent. rate	57 bpm
PR interval	150 ms
QRS duration	102 ms
QT/QTc	472/459 ms
P-R-T axes	76 70 58

Sinus bradyc a
Possible Left atrial enlargement
Borderline ECG

ER ATTENDING REVIEW
NO STEMI
TIME 1511

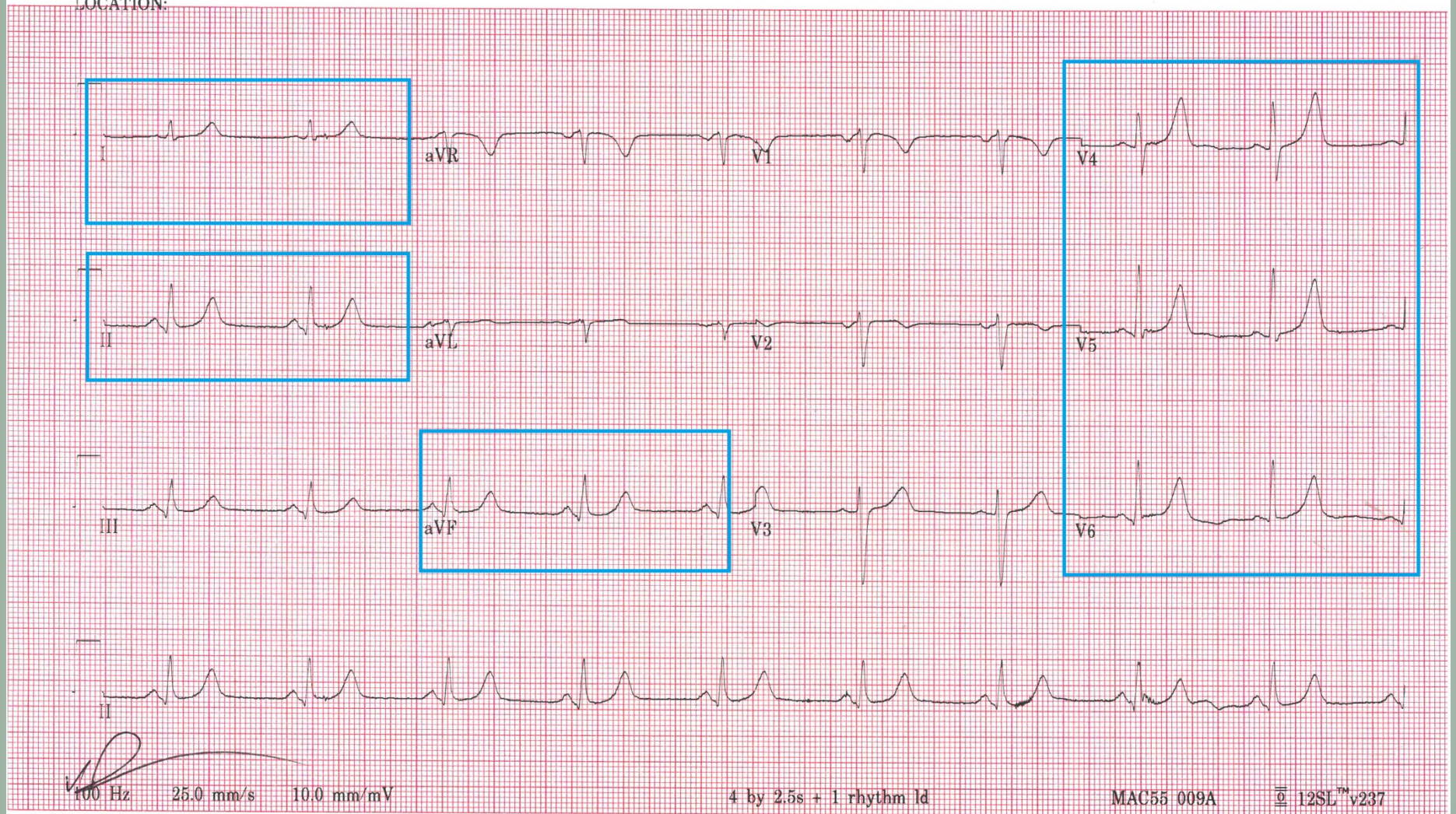
K+ = 6.7

Technician:
Test ind:

Referred by:

Unconfirmed

LOCATION:



100 Hz 25.0 mm/s 10.0 mm/mV 4 by 2.5s + 1 rhythm ld MAC55 009A 12SL™v237

Helpful Clue: Hyper-Acute T Waves

- **GLOBAL Hyper-acute T Waves** (in leads viewing multiple myocardial regions / arterial distributions) **favors HYPERKALEMIA**

- **Hyper-acute T Wave noted in ONE ARTERIAL DISTRIBUTION** (Anterior / Lateral / Inferior) favors TRANSMURAL ISCHEMIA / Early Phase Acute MI

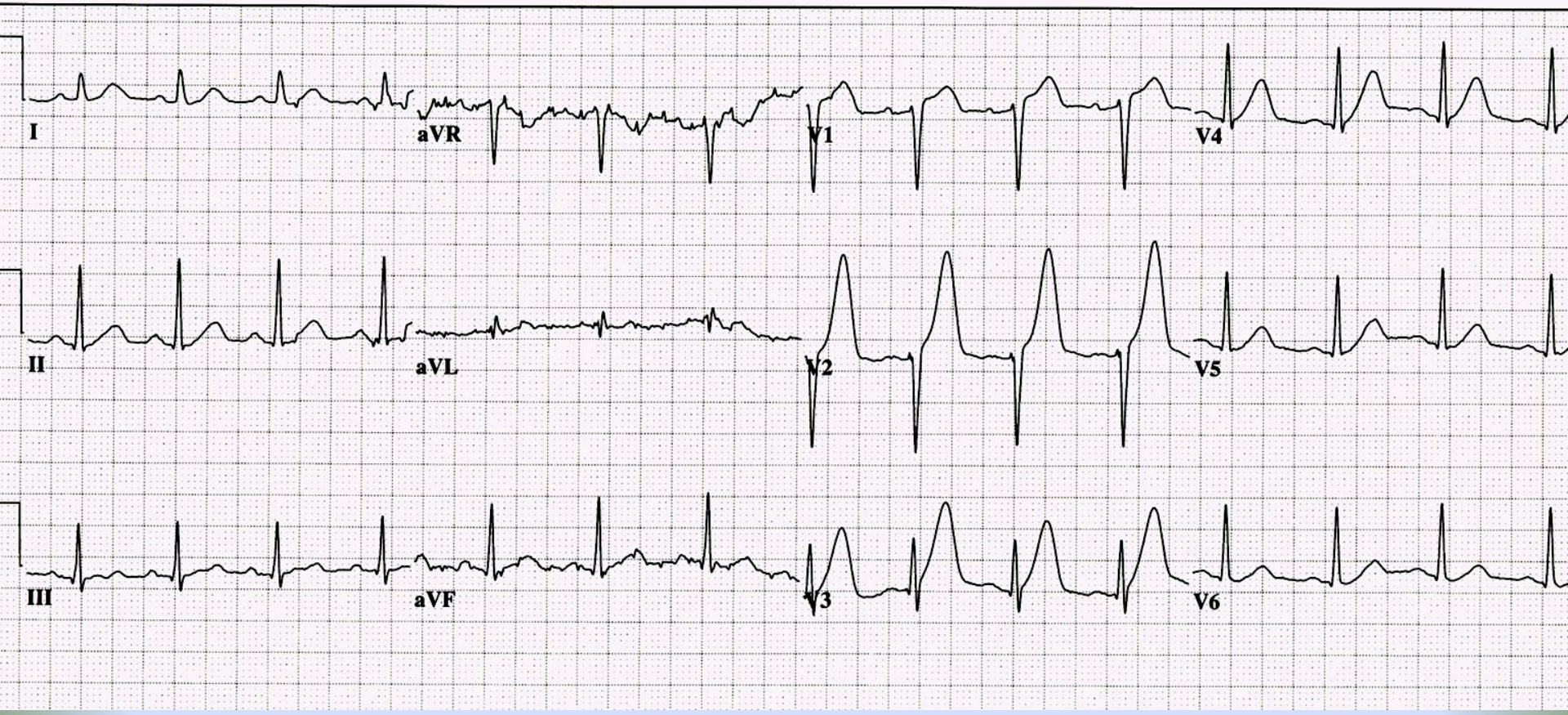
30 yr
Male Black

Room: ER
Loc: Option:

Vent. rate	88	BPM
PR interval	164	ms
QRS duration	90	ms
QT/QTc	370/447	ms
P–R–T axes	61 62	53

Normal sinus rhythm
Normal ECG
No previous ECGs available

← NOTE COMPUTER INTERPRETATION



30 yr
Male Black

Room: ER
Loc: Option:

Vent. rate	88	BPM
PR interval	164	ms
QRS duration	90	ms
QT/QTc	370/447	ms
P–R–T axes	61 62	53

Normal sinus rhythm
Normal ECG
No previous ECGs available

HIGHLIGHTED AREAS = HYPERACUTE T WAVES

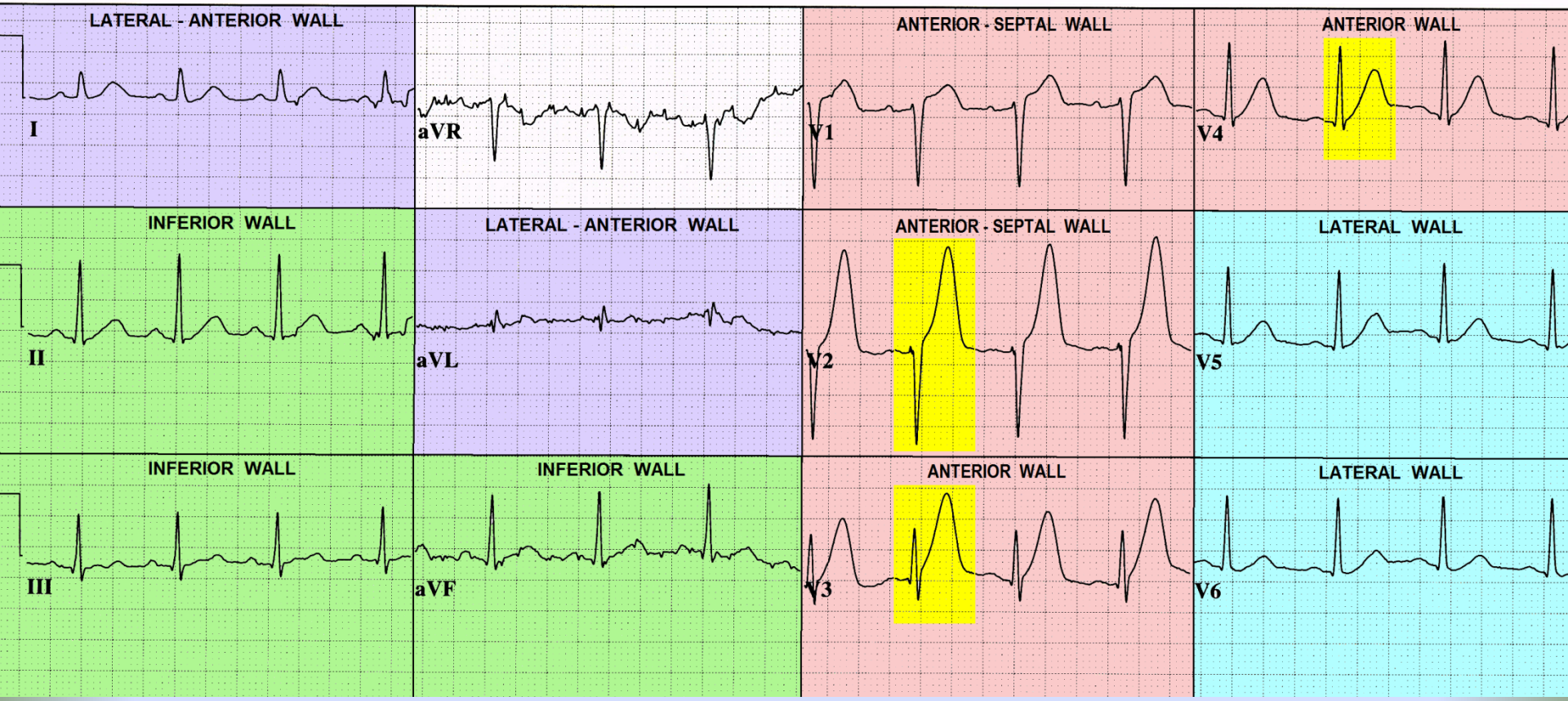
CORONARY ARTERIAL DISTRIBUTIONS:

V1 - V4 = LEFT ANTERIOR DESCENDING (LAD)

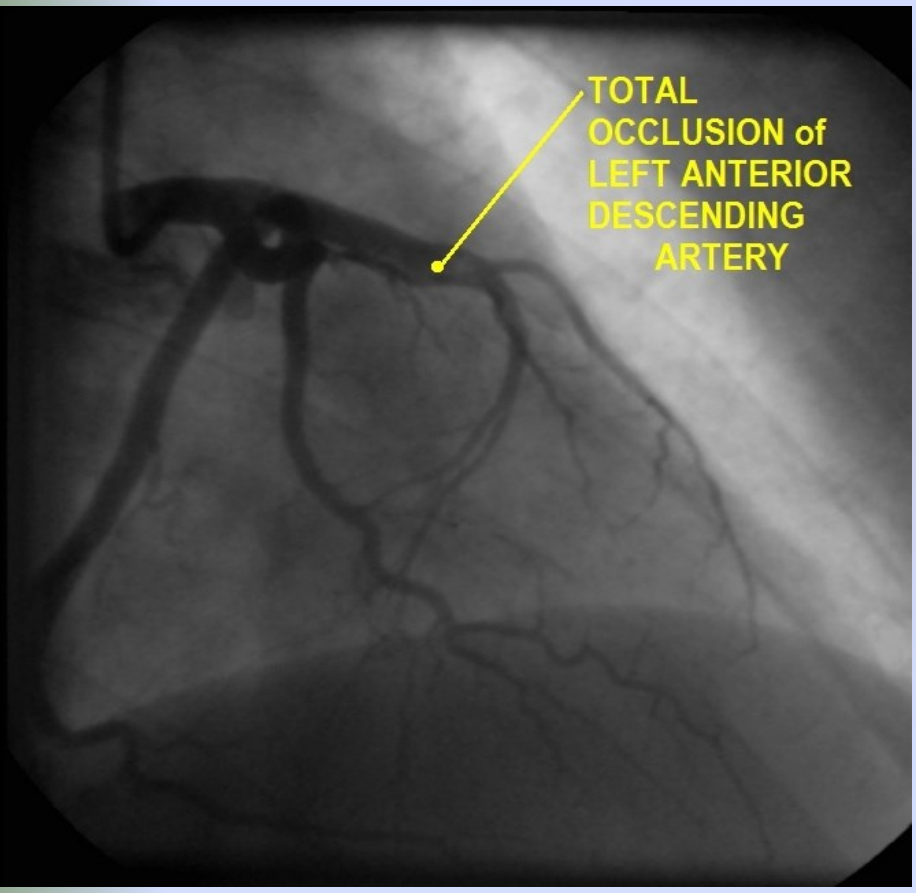
I, AVL = DIAGONAL (DIAG) off the LAD or OBTUSE MARGINAL (OM) off CIRCUMFLEX (CX)

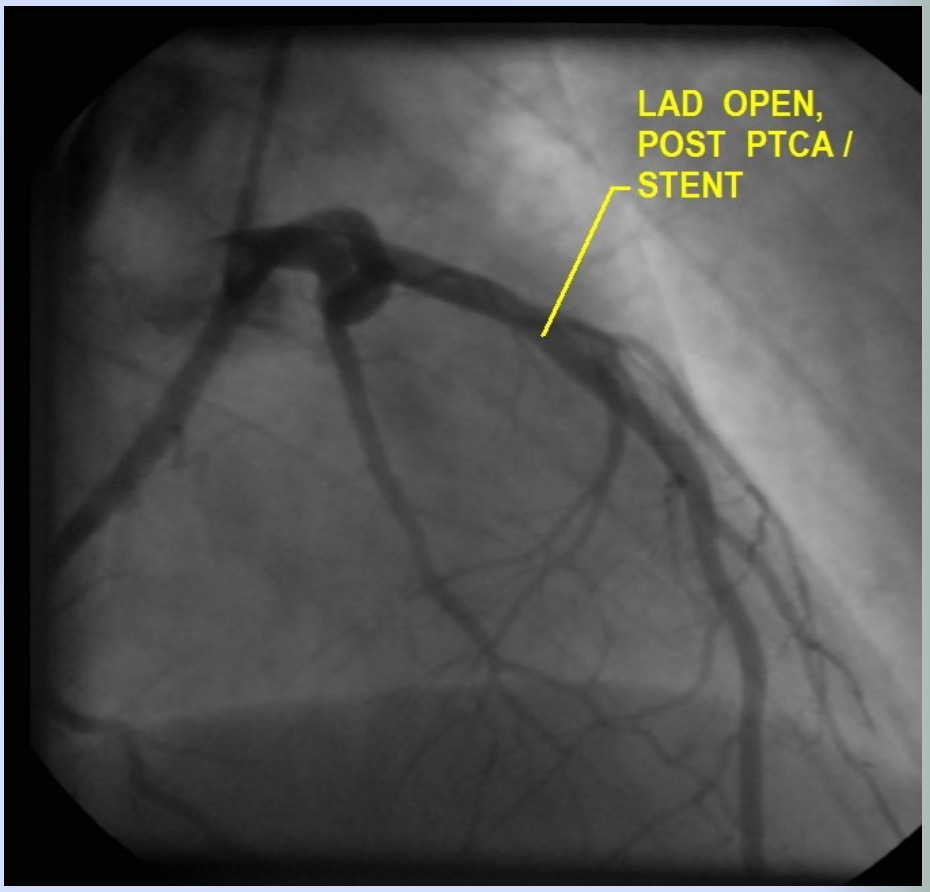
V5, V6 = CIRCUMFLEX

II, III, AVF = RIGHT CORONARY ARTERY or CX



Cath Lab findings:





ECG Patterns associated with "EARLY PHASE MI:"

- *J-T Apex abnormalities*
- *Hyper-Acute T Waves*
- *Dynamic ST-T Wave Changes on Serial ECGs*

12:33:40

Seven R[illegible]rs Regiona
Dept: 2 so[illegible]
Room: er

Rate	75	. SINUS RHYTHM.....normal P axis, V-rate 50- 99
		. CONSIDER ANTEROSEPTAL INFARCT.....Q >30mS, V1 V2
PR	140	. BORDERLINE REPOLARIZATION ABNORMALITY.....ST dep & abnormal T
QRSD	90	. BASELINE WANDER IN LEAD(S) V1,V2
QT	376	
QTc	420	

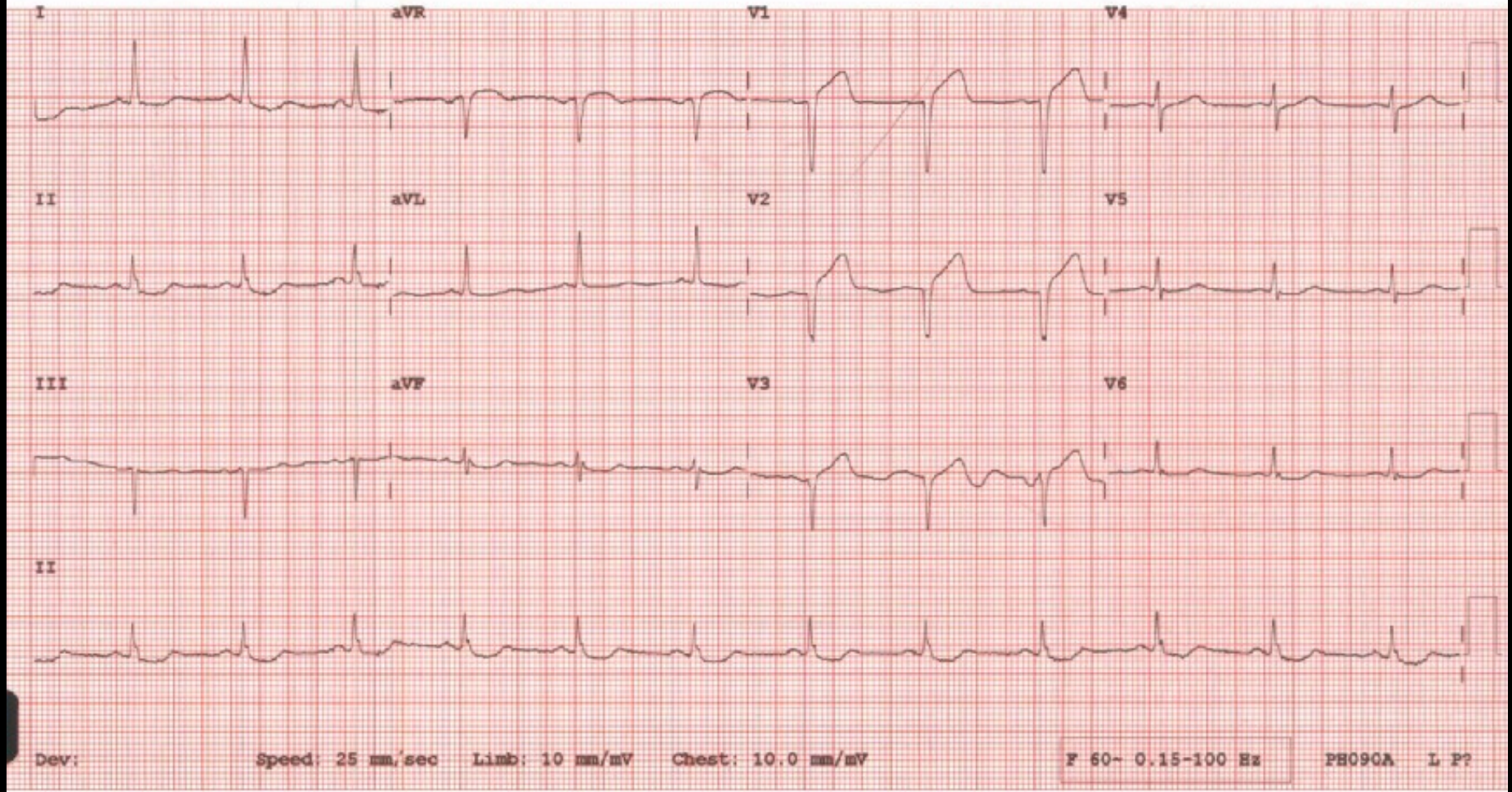
--AXIS--

P	35
QRS	6
T	193

SEVEN RIVERS REGIONAL MED CTR

- ABNORMAL ECG -

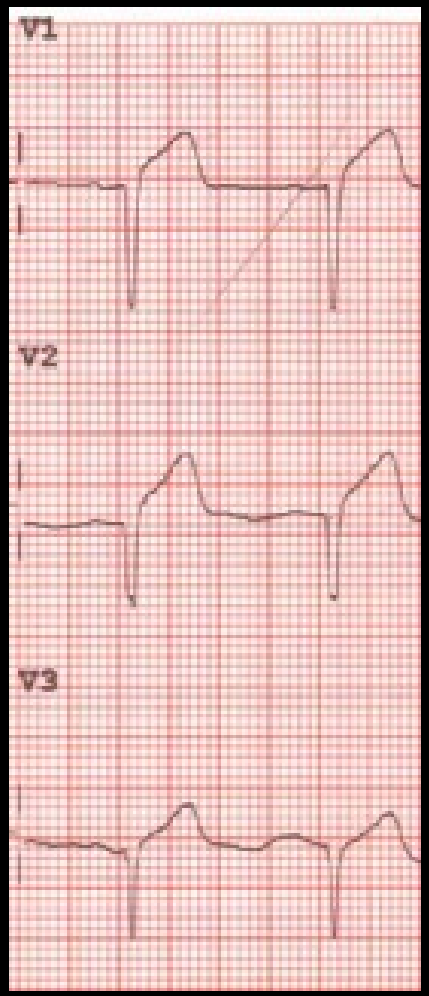
Unconfirmed Diagnosis



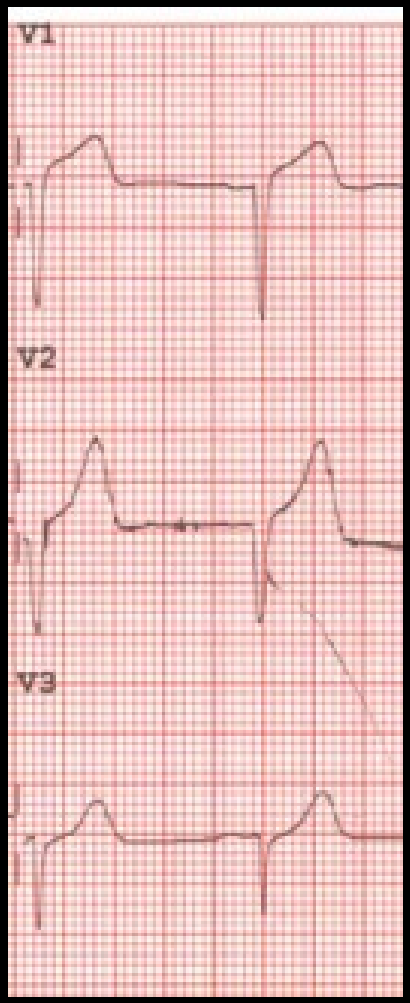
Dev: Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10.0 mm/mV F 60~ 0.15-100 Hz PH090A L P?

3. Dynmamic ST-T Wave Changes in Serial ECGs. Recorded at SRRMC

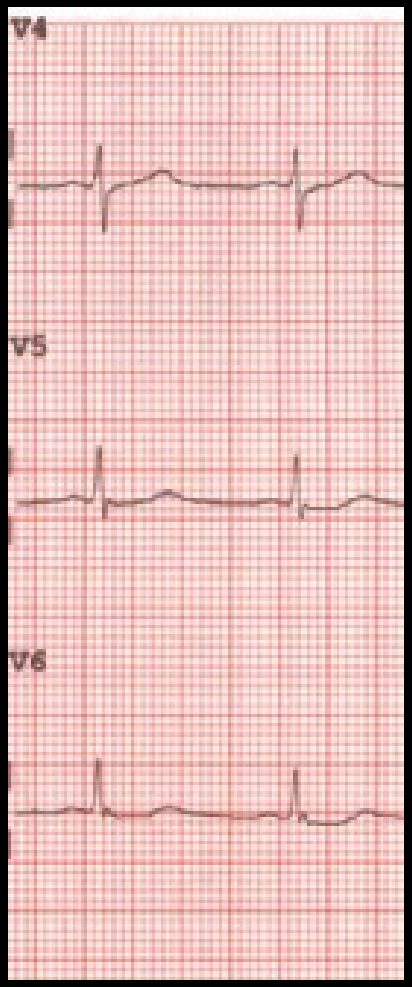
1st ECG



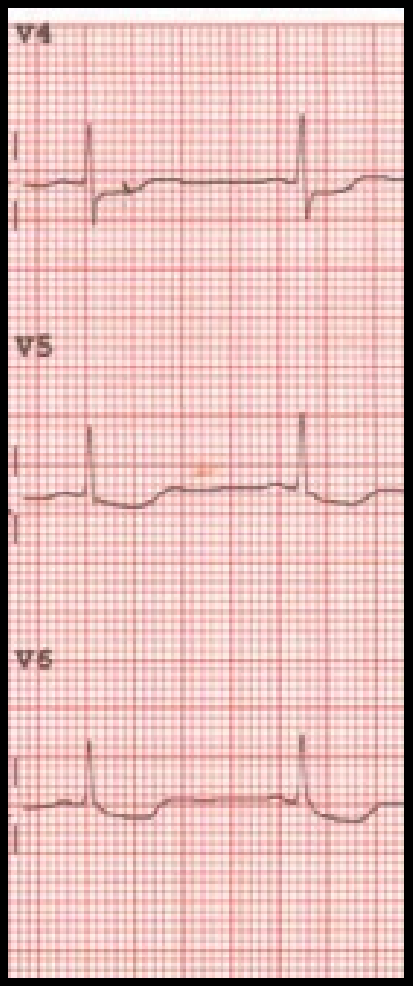
2nd ECG

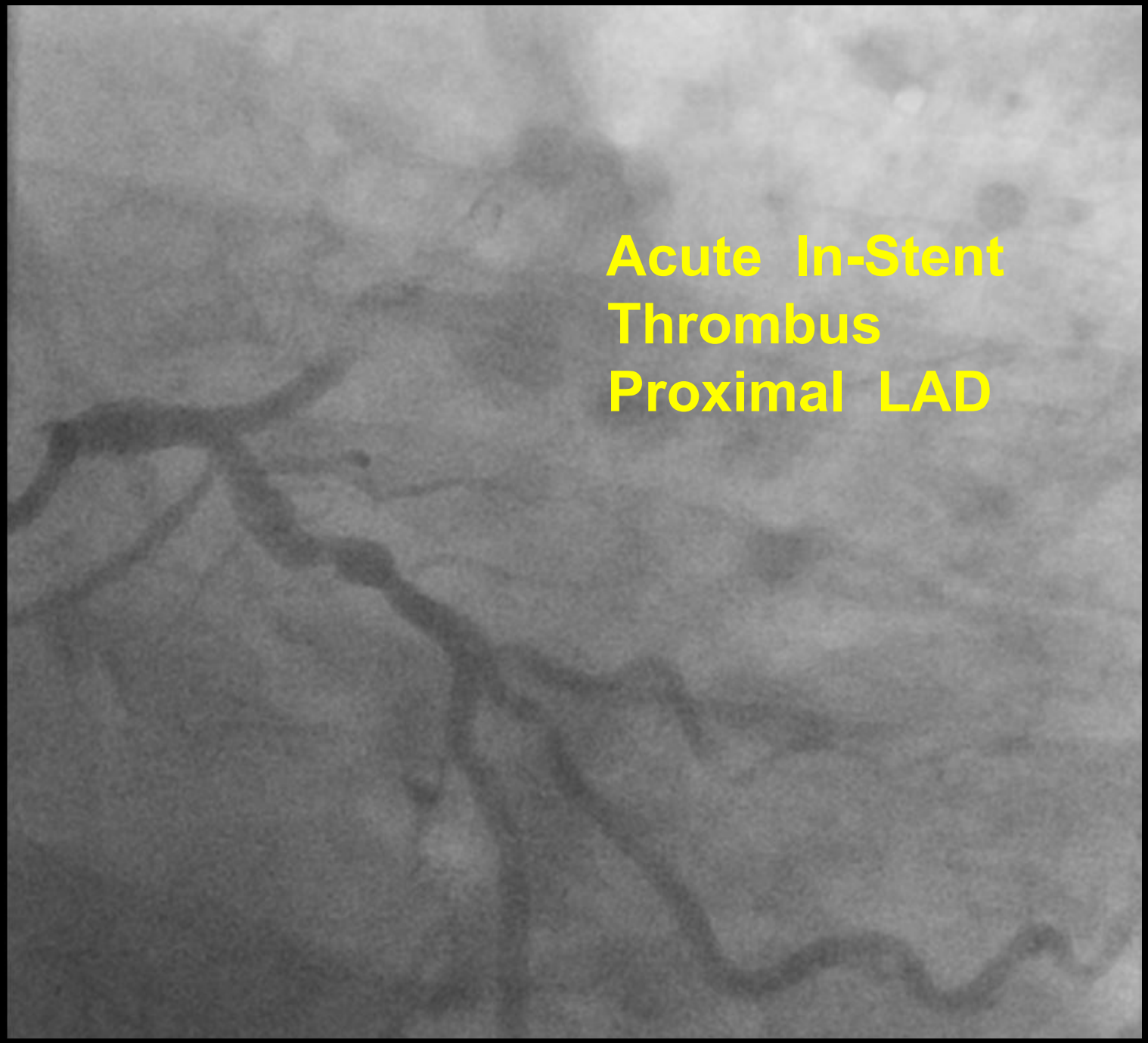


1st ECG



2nd ECG

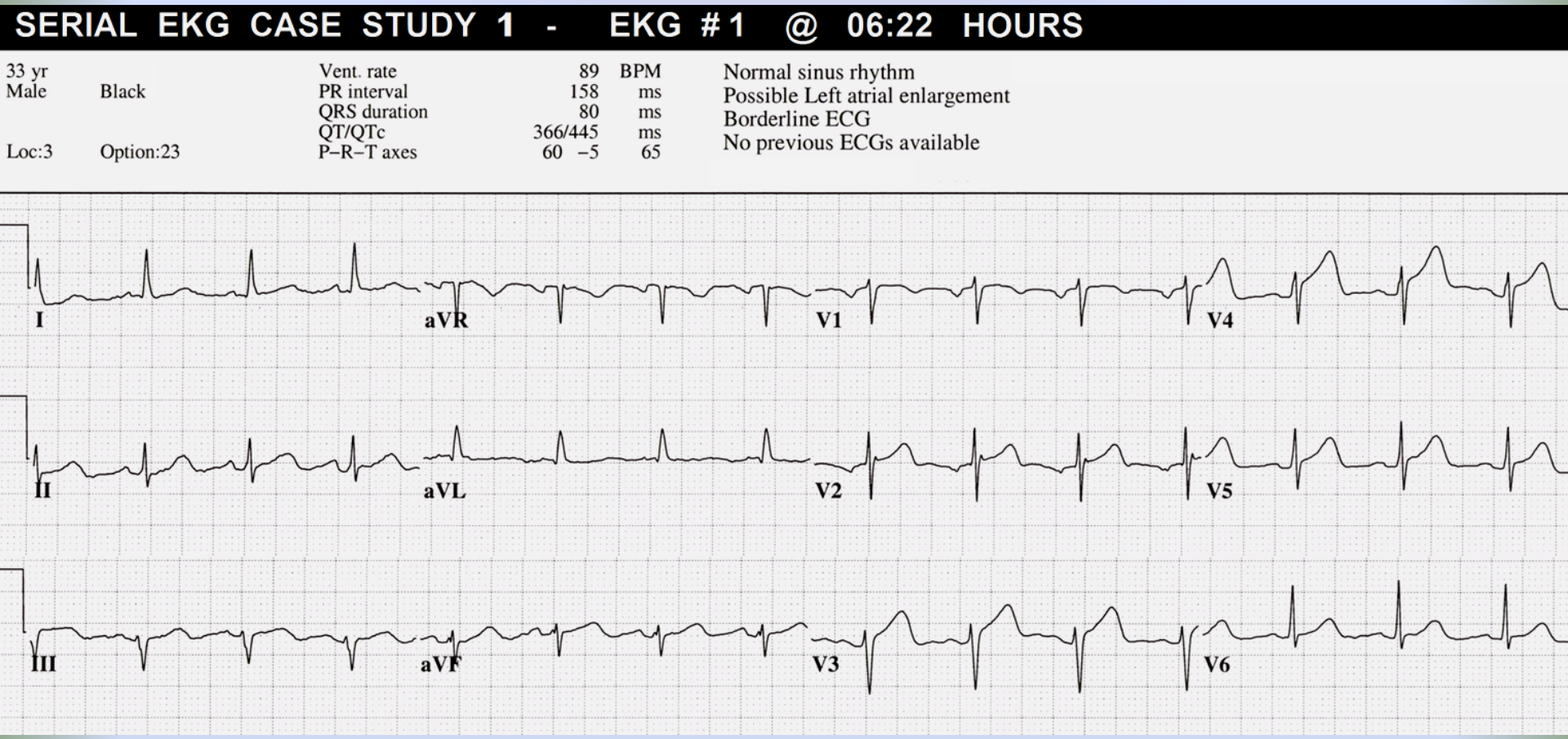



Acute In-Stent
Thrombus
Proximal LAD

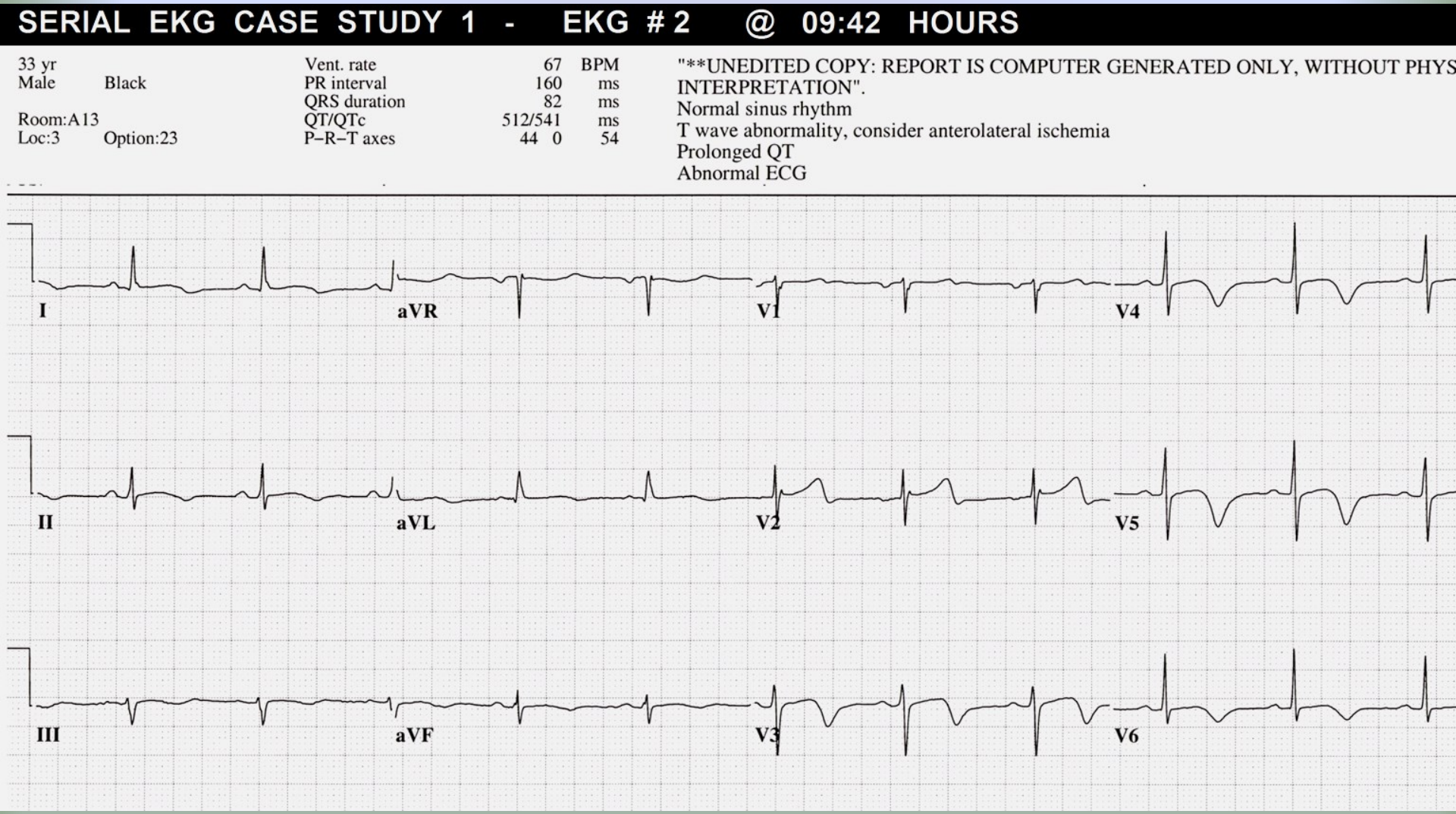
Wellen's Syndrome Case Study

- 33 y/o male
- Chief complaint "sharp, pleuritic quality chest pain, intermittent, recent history lower respiratory infection with productive cough."
- ED physician attributed the ST elevation in precordial leads to "early repolarization," due to patient age, gender, race (African American) and concave nature of ST-segments.

Wellen's Syndrome Case Study



Wellen's Syndrome Case Study

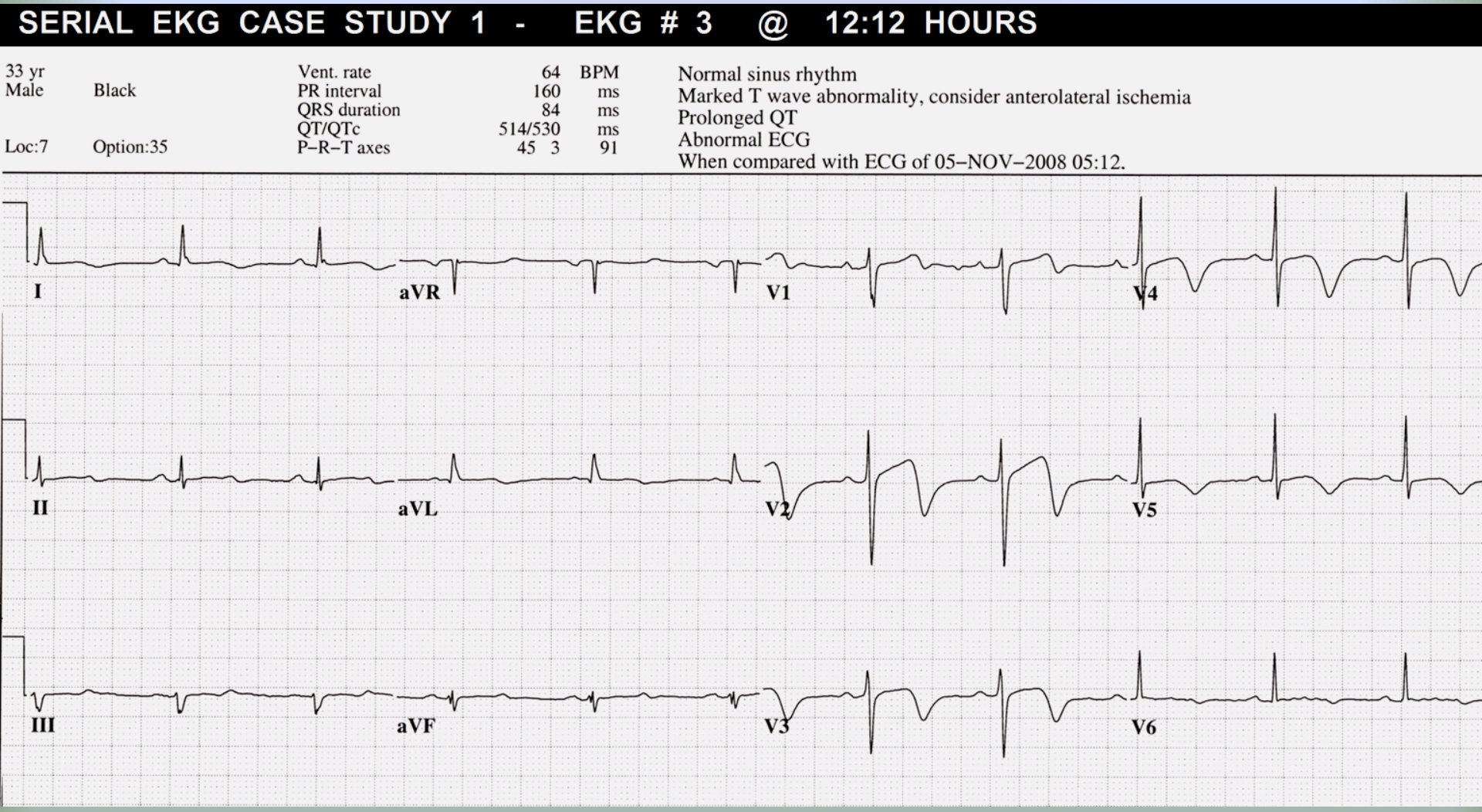


DYNAMIC ST-T Wave Changes ARE PRESENT !!

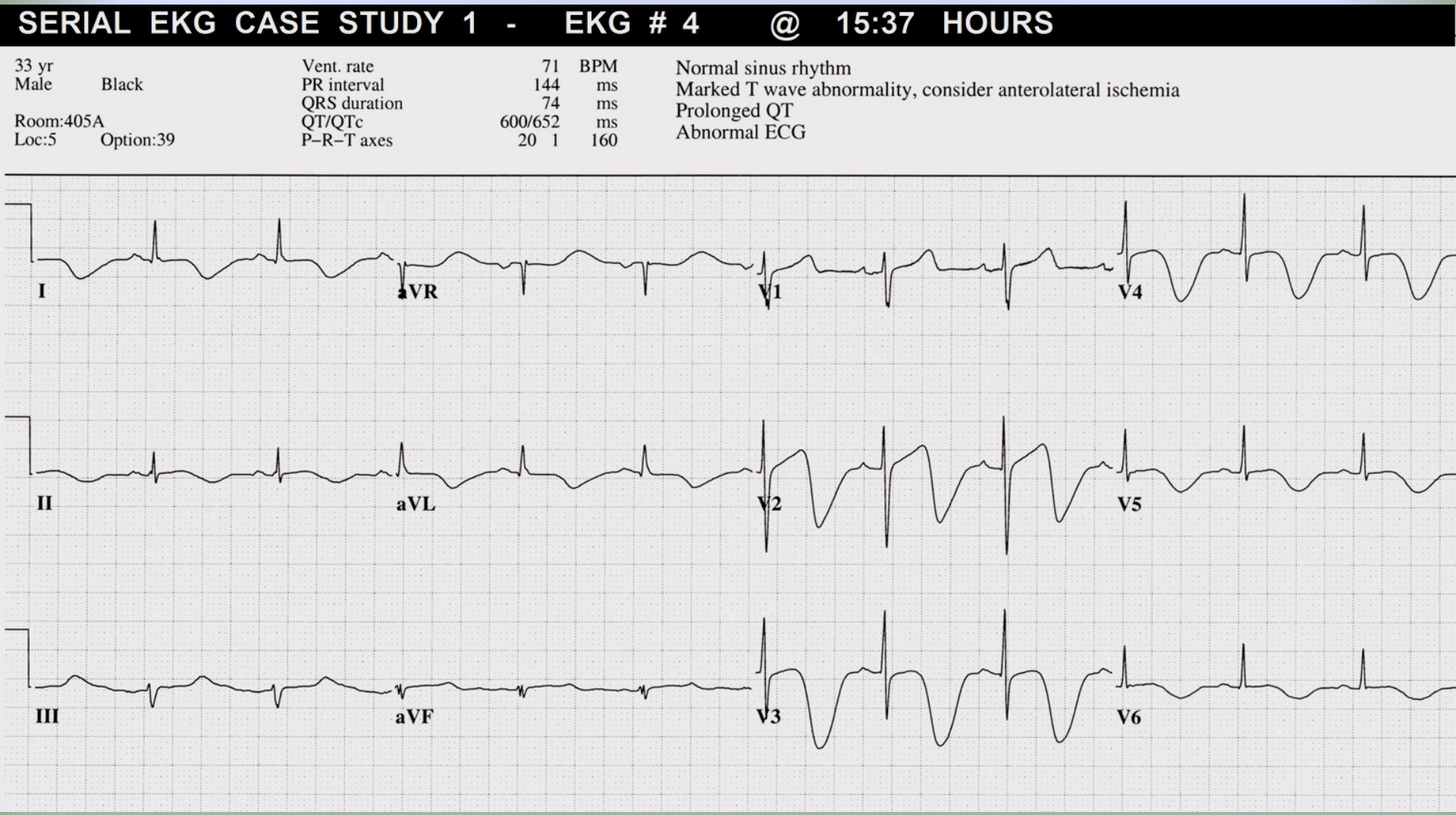
NOW

is the time for the STAT CALL to the CARDIOLOGIST !!!!

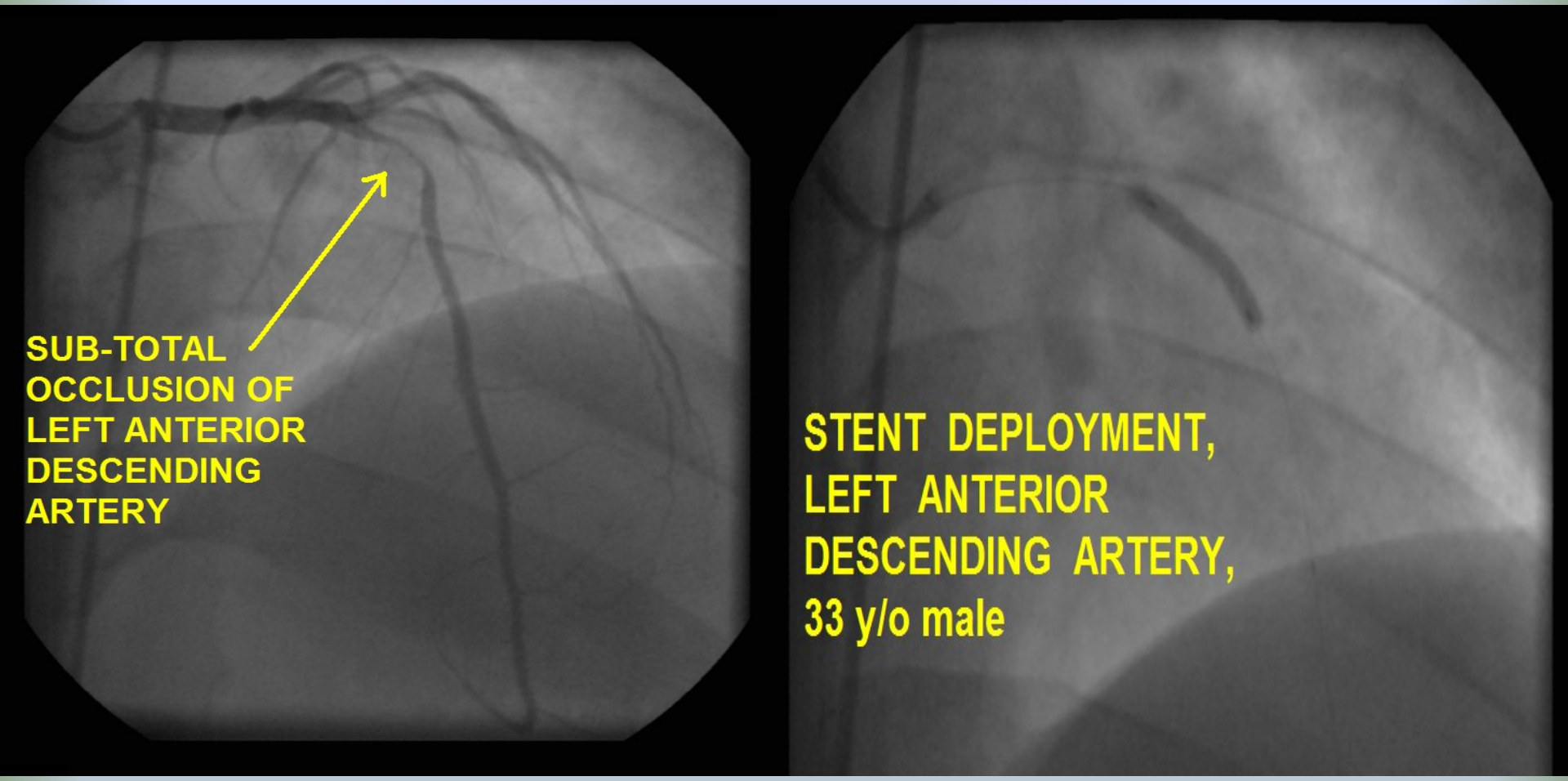
Wellen's Syndrome Case Study



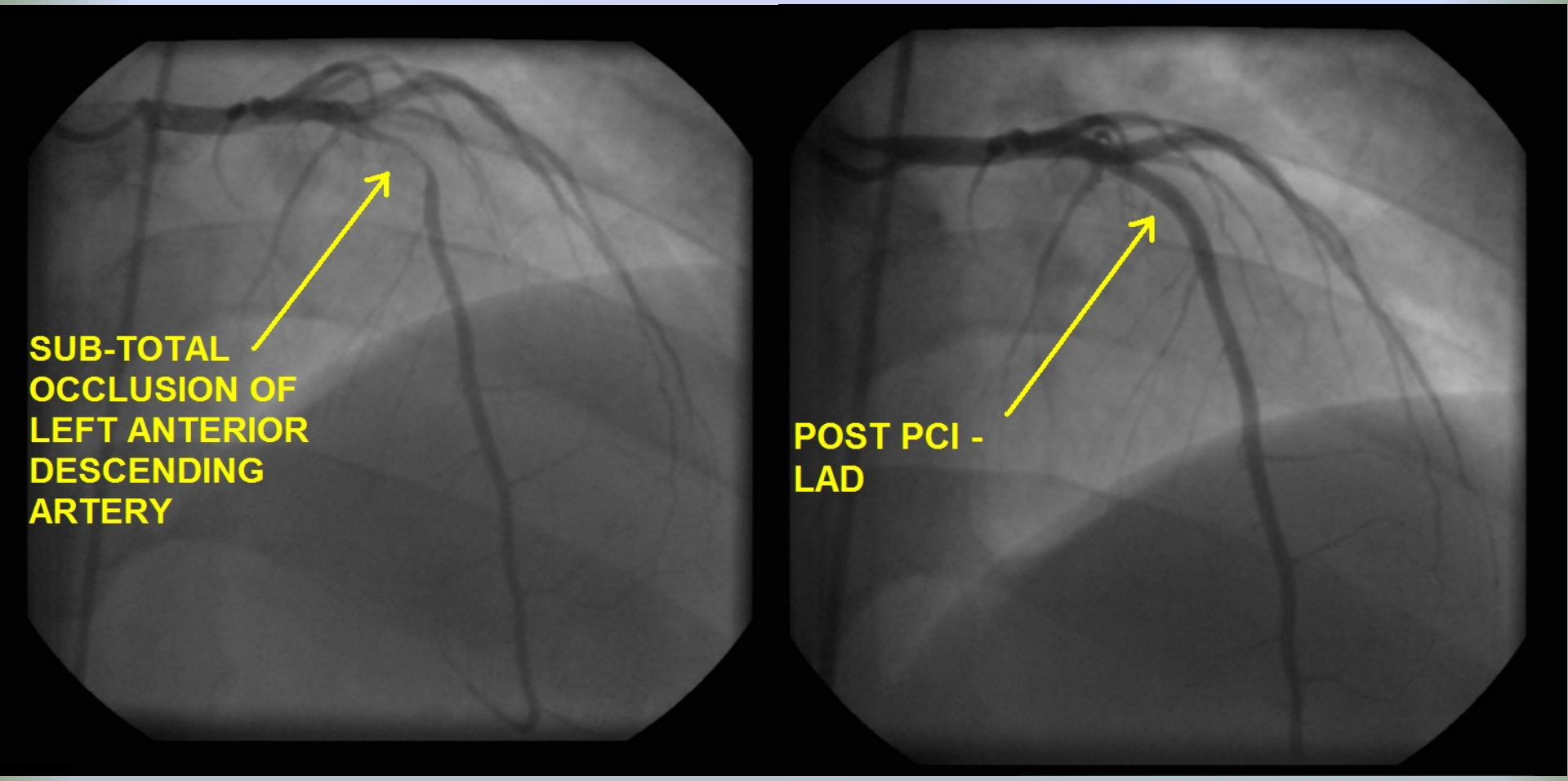
Wellen's Syndrome Case Study



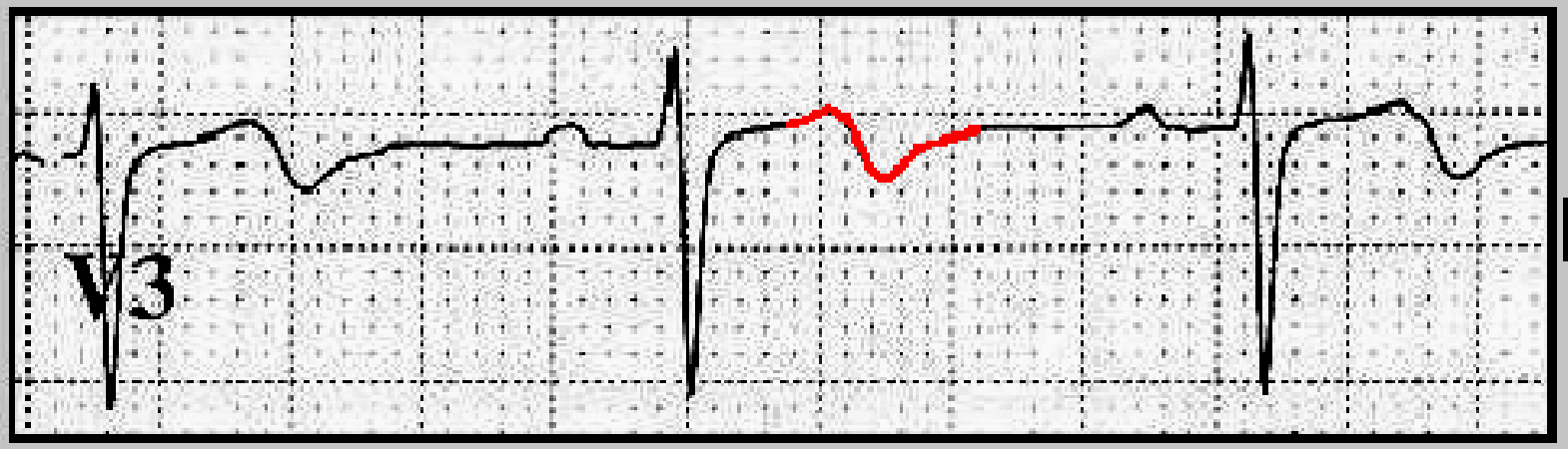
Wellen's Syndrome Case Study



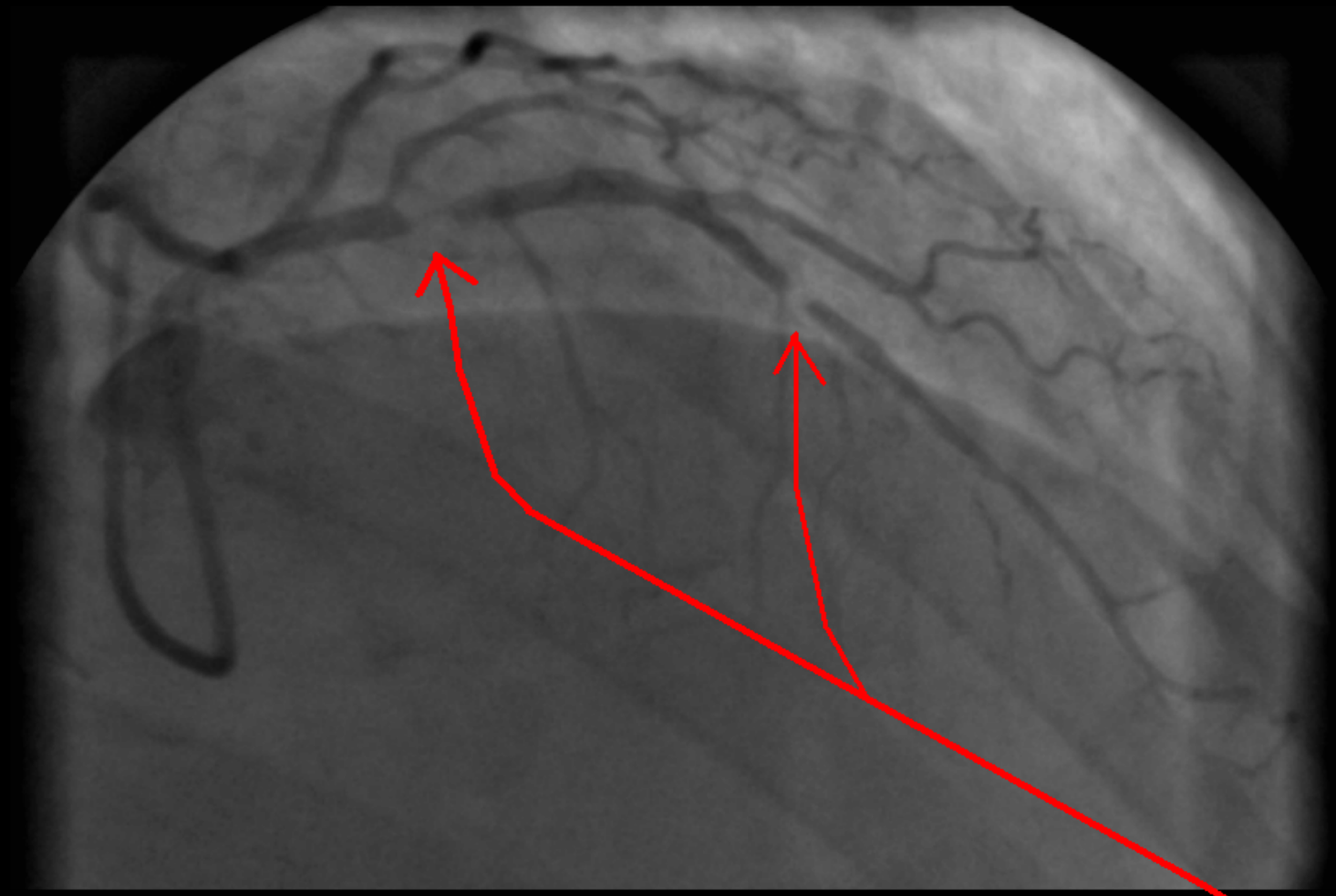
Wellen's Syndrome Case Study



BI-PHASIC T WAVES



58 y/o MALE WITH SUB-TOTAL OCCLUSIONS OF THE LEFT ANTERIOR DESCENDING ARTERY



58 y/o MALE WITH "WELLEN'S WARNING." PT HAS SUB-TOTALLY OCCLUDED LAD X 2

Classic "Wellen's Syndrome:"

- Characteristic T wave changes
  - Biphasic T waves
  - Inverted T waves
- History of anginal chest pain
- Normal or minimally elevated cardiac markers
- ECG without Q waves, without significant ST-segment elevation, and with normal precordial R-wave progression

Wellen's Syndrome ETIOLOGY:

- Critical Lesion, Proximal LAD
- Coronary Artery Vasospasm
- Cocaine use (vasospasm)
- Increased myocardial oxygen demand
- Generalized Hypoxia / anemia / low H&H

Wellen's Syndrome EPIDEMIOLOGY & PROGNOSIS:

- **Present in 14-18% of patients admitted with unstable angina**
- **75% patients not treated developed extensive Anterior MI within 3 weeks.**
- ***Median Average time from presentation to Acute Myocardial Infarction – 8 days***

Sources: **H Wellens et. Al, Am Heart J 1982; v103(4) 730-736**

CLICK HERE to download complete ACC 20th Congress 12 Lead ECG Serial ECG Workshop

American College of Cardiology
20th Congress 2017

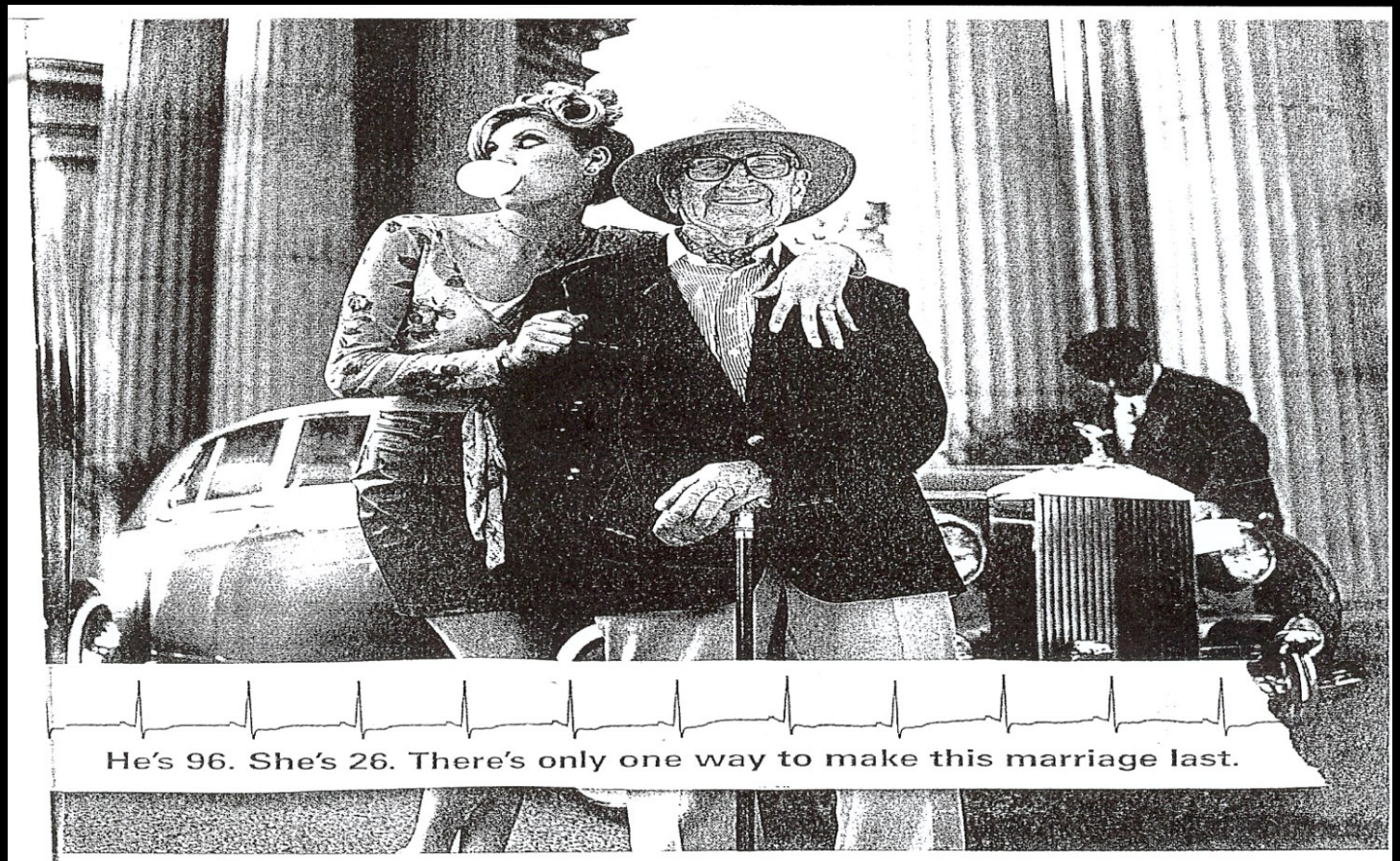
Red Rock Resort, Las Vegas
October 25 & 26, 2017

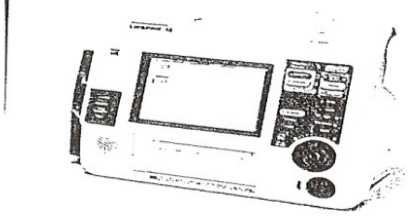
Observation Medicine ECG Instructor Workshop

Serial 12 Lead ECG Interpretation

By: Wayne W Ruppert, CVT, CCCC, NREMT-P

QUESTIONS ? ? ?





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