

Bayfront Health Hospitals









Seven Rivers Freestanding ED, Citrus Hills, FL



Bayfront Health Spring Hill, Spring Hill, FL





The Lifesaving 12 Lead ECG: Part 2

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Wayne.ruppert@bayfronthealth.com

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



American College of Cardiology

Accreditation Services (formerly The Society of Cardiovascular Patient Care)

May 25-27,2016

scpc.org/Congress

Elements of Sudden Cardiac Death Prevention Programs

The American College of Cardiology
Accreditation Services

19th Congress – Miami, FL – May 25, 2016

Wayne Ruppert, CVT, CCCC, NREMT-P

To download presentation in PDF: visit: www.ECGtraining.org select: "Downloads - PDF"

Brief, focused ECG excerpts from the presentation given by Wayne Ruppert at the "19th Congress," American College of Cardiology Accreditation Services" national conference, on MAY 25, 2016 Miami, FL

Prevalence SADS Foundation Stats:

- Each year in the United States, 350,000 Americans die suddenly and unexpectedly due to cardiac arrhythmias. Almost 4,000 of them are young people under age 35. (CDC 2002)
- In 30%–50% of sudden cardiac deaths, it is the first clinically identified expression of heart disease
- 10-12% of Sudden Infant Death Syndrome (SIDS) cases are due to Long QT Syndrome.
- LQTS is now known to be 3 times more common in the US than childhood leukemia.
- 1 in 200,000 high school athletes in the US will die suddenly, most without any prior symptoms—JAMA 1996; 276

The SADS Conditions:

- Hypertrophic Cardiomyopathy (HCM)
- Long QT Syndrome (LQTS)
- Short QT Syndrome (SQTS)
- Brugada Syndrome (BrS)
- Arrhythmogenic Right Ventricular Dysplasia (ARVD)
- Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)
- Wolff-Parkinson-White (WPW) Syndrome
- Commotio Cordis
- Less-common conditions (e.g. <u>Marfans</u>, <u>Ehlers-Danlos</u>, <u>Loeys-Dietz Syndromes</u>)

Estimated SADS Prevalence in US Population:

• HCM: 1/500 <u>J Am Coll Cardiol</u>. 2014;64

• BrS: 1/2,500 SADS Foundation

LQTS: 1/2,500 <u>Lenhart,SE 2007 AHA Circ</u>

ARVD: 1/10,000 SADS Foundation

CPVT: 1/10,000 <u>US Nat'l Library of Medicine</u>

• WPW: 1/1,000 <u>Circulation.2011; 124: 746-757</u>

Prevalence

Sudden Deaths in Young Competitive Athletes

B Maron et al; AHA Circulation.2009; 119: 1085-1092

Analysis, causes of 1866 Deaths in the US, 1980 –2006:

Cardiovascular: 56%

Traumatic: 22%

Commotio Cordis: 3%

Heat Stroke: 2%

Other: 17%

Most ACS Cardiac Arrest Patients are over age 30.

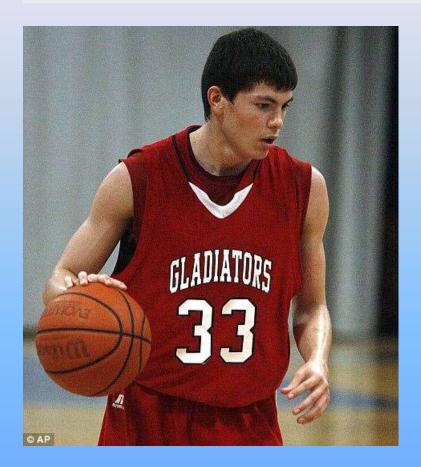
Meet the typical Cardiac Arrest patients affected by SADS....

High School Athlete Dies After Collapsing At Practice August 15, 2011 11:28 PM

Share on email₁₇



Teen basketball player collapses and dies on court - third school boy sportsman to do so in less than a month



By DAILY MAIL REPORTER

UPDATED: 12:03 EST, 14 March 2011

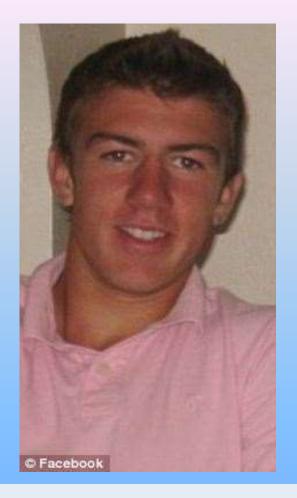
A teenage basketball player has become the third school boy sports man in less than a month to collapse and die while playing.

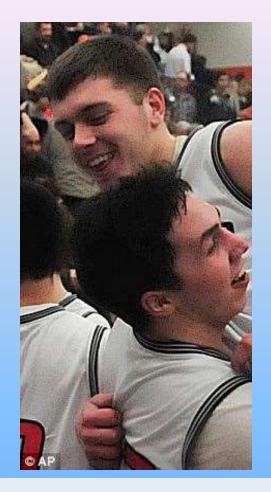
Roma High School junior Robert Garza, 16, was playing in the AAU tournament on Saturday with the Hoopsters, a South Texas club team, when he collapsed without any warning.

His death follows that of Wes Leonard, who died of cardiac arrest from an enlarged heart on March 3 and

Matthew Hammerdorfer,17, who collapsed after taking a tackle to the chest at a rugby match near Denver last week.

Sudden: The death of Robert Garza is the third such school boy death in the last month. The other two both had heart conditions





Tragedy: The death comes only weeks after that of Wes Leonard (right top) and Matthew Hammerdorfer, who collapsed after taking a school rugby match near Denver

Ray-Pec student collapses and dies during track practice Posted, 2015-03-05

Kansas City Star

A senior at Raymore-Peculiar High School collapsed during track practice Wednesday and died at a hospital, according to school officials. ... Click to Continue »

Family and friends mourn popular Boonsboro High School athlete

Michaela Grove 'was just a good kid that didn't follow the crowd, and people liked that'

July 24, 2013 By DAVE McMILLION | davem@herald-mail.com



Family members and friends of a popular Boonsboro High School athlete are mourning her death after she collapsed at a camp in Mercersburg, Pa., on Monday evening.

Michaela Grove's mother, Brenda Grove, said she believes her 16-year-old daughter was involved in a tug-of-war competition at Camp Tohiglo when she fell to the ground in cardiac arrest.

Greg Moyer, 15



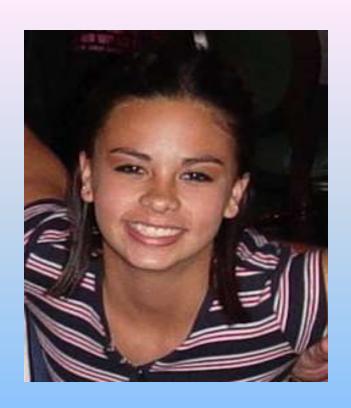
Greg Moyer collapsed and died of sudden cardiac arrest while playing in a high school basketball game in East Stroudsburg, Pennsylvania. His school did not have a automated external defibrillator available and there were no nearby emergency medical services.

Afterwards, a nurse at the hospital emergency room suggested to Greg's parents that they start a fund to help locals schools get AEDs. The Moyers are now involved in AED projects statewide, and Greg's mother, Rachel Moyer, has traveled as far as Hawaii to advocate for school AED legislation and donate AEDs



"Princess George" died at age 3 of sudden cardiac arrest brought on by an undiagnosed heart condition. At the suggestion of the doctor who saw "George" in the emergency room, her brother was subsequently tested for heart problems. He was diagnosed with a heart condition that is, fortunately, treatable.

Jennifer Lynn Balma, their mother, notes that "George" never showed any symptoms of cardiac problems — *until the day she suddenly stopped breathing.*



Olivia Corinne Hoff, 14

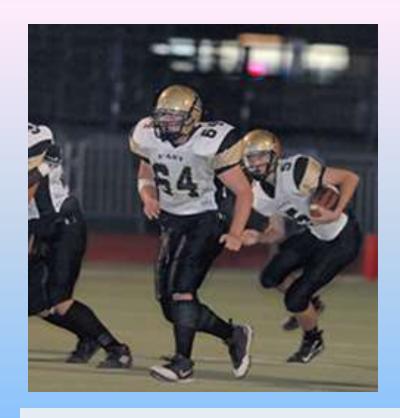
Olivia died at age 14 from sudden cardiac arrest attributed to Long QT Syndrome. The condition was undiagnosed. Olivia, a high school freshman involved in sports and cheerleading, suffered cardiac arrest during the night. Her mother found her unresponsive and called 911. Olivia was subsequently hospitalized, but did not survive.

Her mother, Corinne Ruiz, wrote: "Today, 6 years later, I cry for my daughter every day. Not a day goes by that I don't ask myself: If only I had been told that there are screening tests or preventative treatments."



High school quarterback Reggie Garrett threw his second touchdown pass of the night, walked off the field, and collapsed from sudden cardiac arrest. He died in the ambulance on the way to the hospital in West Orange, Texas.

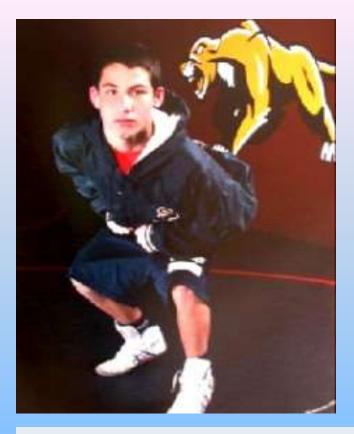
In the news coverage following Garrett's death, Dallas station WFAA.com urged cardiac screening for high school athletes.



Zachary Shrah, 16

High school football player Zachary Schrah collapsed and <u>died of sudden cardiac</u> <u>arrest</u> during football practice in Plano, Texas. His mother, Karen Schrah, has become an advocate for legislation mandating heart screenings as a part of student physicals.

Zachary's death had an impact on the community at large. Heart Hospital Baylor Plano now offers low-cost <u>ECGs</u> and echocardiograms for the area's student athletes.



Eric Paredes, a two-sport high school athlete, had an enlarged heart. But no one knew about it until it was too late. His father, Hector Paredes, found Eric on the kitchen floor, unconscious and not breathing. He administered CPR, but was unable to revive him. Eric died of sudden cardiac arrest.

In Eric's memory, the family has organized <u>electrocardiogram</u> (EKG) screening for other students at Eric's San Diego area high school.



In 2005, Chicago conservationist and wildlife educator Max Schewitz <u>died</u> of sudden cardiac arrhythmia. Since then, the Max Schewitz Foundation, created by his parents, has provided free <u>electrocardiograms</u> (EKGs) for more than 10,000 Chicago-area students through a Screen for Teens program.

According to media reports, the screenings have identified 142 teens who are considered at-risk for sudden cardiac death because of cardiac conditions.

Nick Varrenti, 16



Nick Varrenti played in two high school football games — varsity and junior varsity — on Labor Day weekend. A day later, he <u>suffered sudden cardiac arrest</u> and died. His family learned later that Nick had lived with an undiagnosed heart condition, hypertrophic cardiomyopathy.

Nick's parents created the Nick of Time Foundation, which is dedicated to education schools, athletes, and communities about sudden cardiac arrest, <u>public access defibrillator</u> (PAD) programs, and cardiac screenings.

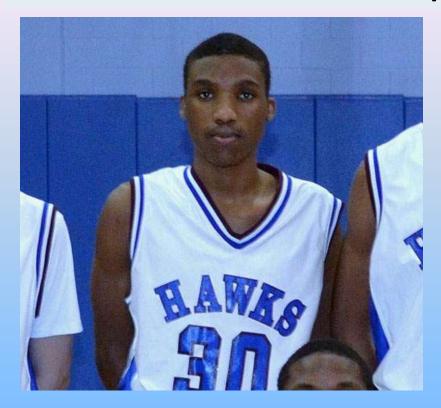
Jimmy Brackett, 22, and Crissy Brackett, 21





The hereditary cardiac disease Long QT Syndrome ran in Jackie Renfrow's family, but she had no idea about it until two of her children died from sudden cardiac arrest.

Brandon athlete dies after collapsing at practice



TAMPA — A Brandon High School senior Milo Meeks died Saturday, one day after conditioning with the basketball team "This is mind blowing," said Ben Bromley, the junior varsity and assistant varsity basketball coach at Armwood.

Jeremy Twining, age 21 Dade City, Florida February 1, 2015

Your Hometown News Source • Dade City News February 12, 2015 • 7B dadecitynews.net

Obituaries

Jeremy Grant Twining



TWINING, Jeremy Grant, 21, of Dade City, joined his savior Jesus in Heaven on Feb. 1, 2015. He was born May 31, 1993. He graduated from Pasco High School and was studying Criminal Justice at Liberty University. He is survived by his parents, John and Julie Twining of Dade City; siblings,

Jonathan, Jessica and James Twining of Dade City; girlfriend, Lydia Tucker of Temple Terrace; paternal grandparents, Dave and Shirley Twining of Tampa; maternal grandparents, Edna Margaret Neatherly of Tampa and Earl and Ginger Hornsby of Cromwell, Conn.; and countless aunts, uncles, and cousins. Jeremy will always be remembered for his contagious laugh, his huge caring heart, and his love for his Lord and Savior Jesus Christ. A private graveside service was held Feb. 6 from the Florida National Cemetery in Bushnell. A memorial service was held at First Baptist Church of Dade City on Feb. 7. In lieu of flowers make send donations to the Sudden Arrhythmia Death Foundation at SADS.org. Hodges Family Funeral Home was in charge of arrangements.

.... And on a more personal note:

This slide added April 27, 2016:

Yesterday, a good friend of my step-daughter collapsed during a tennis game in the Carrollwood community of Tampa, Florida. She was 16 years old.

A physician bystander started CPR, but since no AED was available, she did not survive.

Sudden death was the first indication that she suffered from a cardiac condition. At the current time, her specific diagnosis is unknown.

Entry 5/2/2016: I was advised that the cause of cardiac arrest was Hypertrophic Cardiomyopathy.

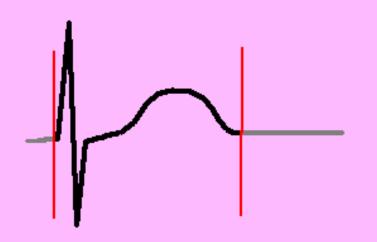


My step-daughter, Caitlin Cameron (right) with her friend, also named Caitlin (left) who collapsed and died during a tennis match on 4/26/16

"Healthcare organizations have an obligation to implement programs, practices, protocols, policies and procedures designed to eliminate the needless mortality of SADS in our communities."

"Healthcare professionals who evaluate young patients have an obligation to be aware of risk factors, signs and symptoms of patients with potential SADS conditions. Those who read ECGs should be aware of the subtle ECG identifiers of SADS conditions."

THE Q-T INTERVAL



 BEGINNING OF QRS COMPLEX TO THE END OF THE T WAVE

- NORMAL VALUES VARY BASED ON HEART RATE
- SEVERAL WAYS TO DETERMINE NORMAL LIMITS

THE *QTc INTERVAL

*QTc = Q-T interval,

	4.10	corrected for heart rate
HEART RATE	MALE	FEMALE
150	0.25	0.28
125	0.26	0.29
100	0.31	0.34
93	0.32	0.35
83	0.34	0.37
71	0.37	0.40
60	0.40	0.44
50	0.44	0.48
43	0.47	0.51

Annals of Internal Medicine, 1988 109:905.

Determining the QTc

Manual calculation:

QT CORRECTION FORMULAS:

Bazett's QTc=QT/√RR

Fredericia QTc=QT/(RR)1/3

Framingham QTc=QT+0.154(1-RR)

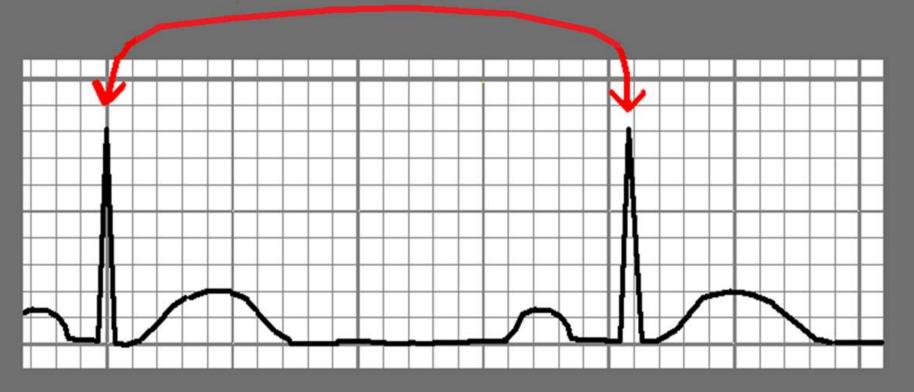
Rautaharju QTp=656/(1+HR/100)

DETERMINING Q-T INTERVAL LIMITS THE "QUICK PEEK" METHOD

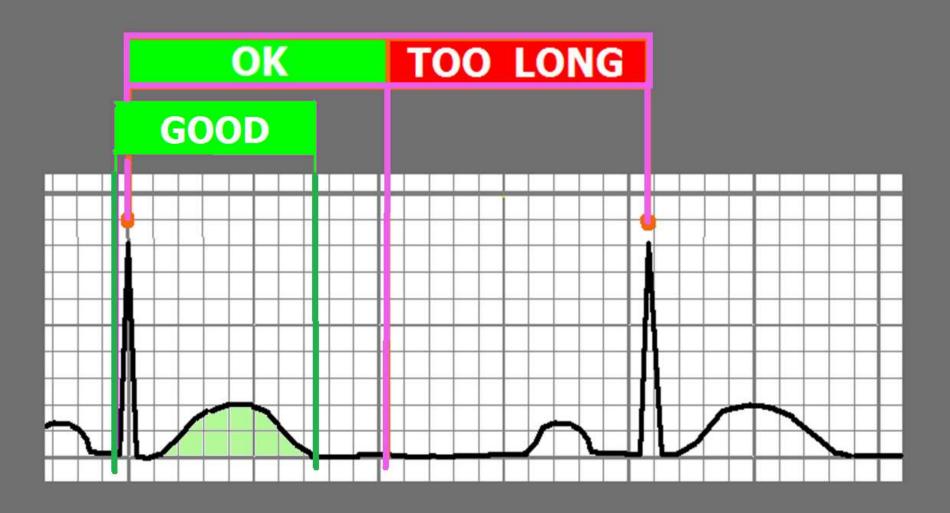


Relatively accurate method to quickly identify patients with abnormal QT Intervals.

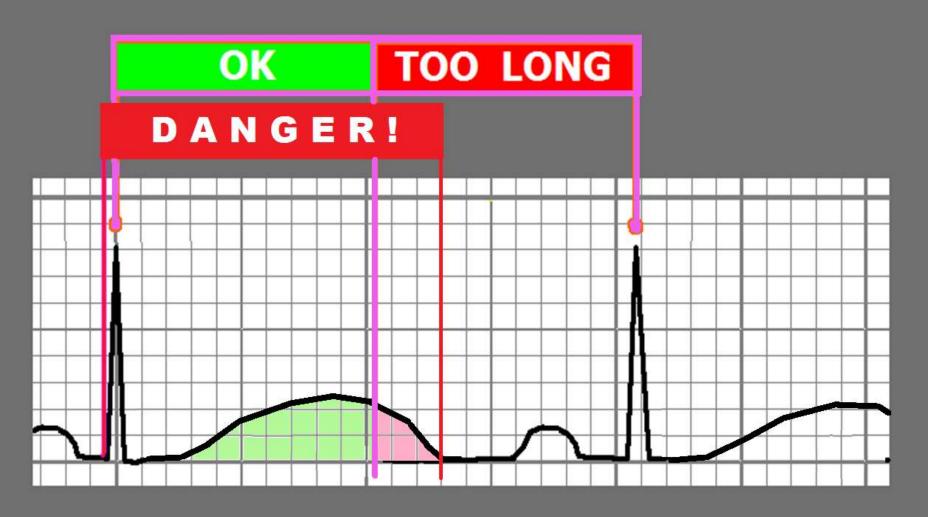
- Applies to patients with normal heart rates (60-100) and narrow QRS (QRSd <120ms)



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

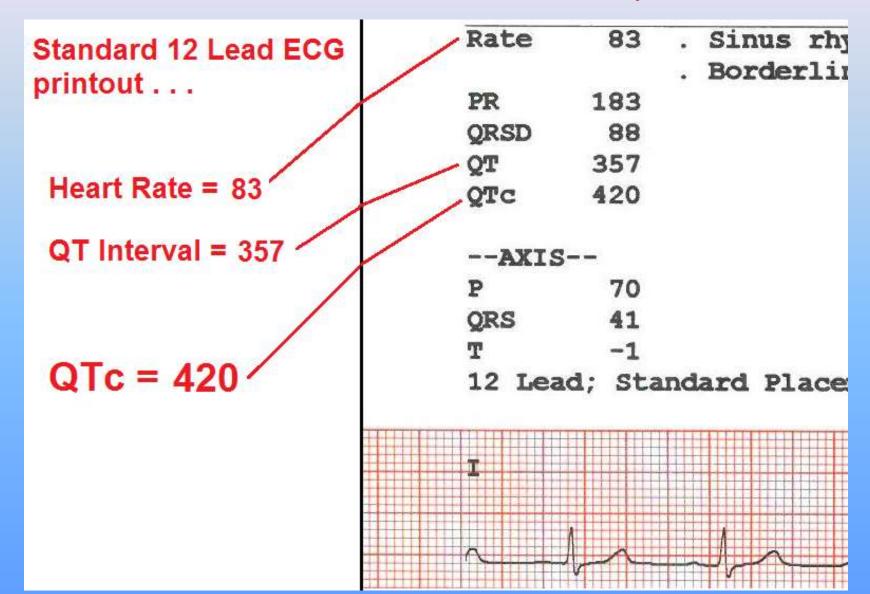


The Q-T Interval should be LESS THAN 1/2 the R-R Interval



Determining the QT / QTc

Method 1 – 12 Lead ECG Report:



Determining the QTc

Method 4, Use a Smartphone App:

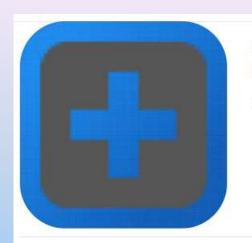
iPhone

- https://itunes.apple.com/us/app/corrected-qtinterval-qtc/id1146177765?mt=8

Android

https://play.google.com/store/apps/details?id=com/store/apps/d

"There's an APP for that!"



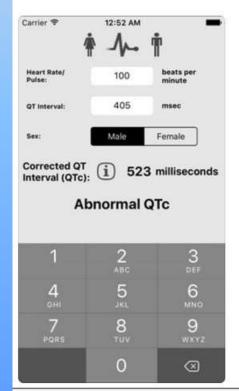
Corrected QT Interval (QTc) 17+

Daniel Juergens

\$0.99

Carrier ?

iPhone Screenshots





12:52 AM

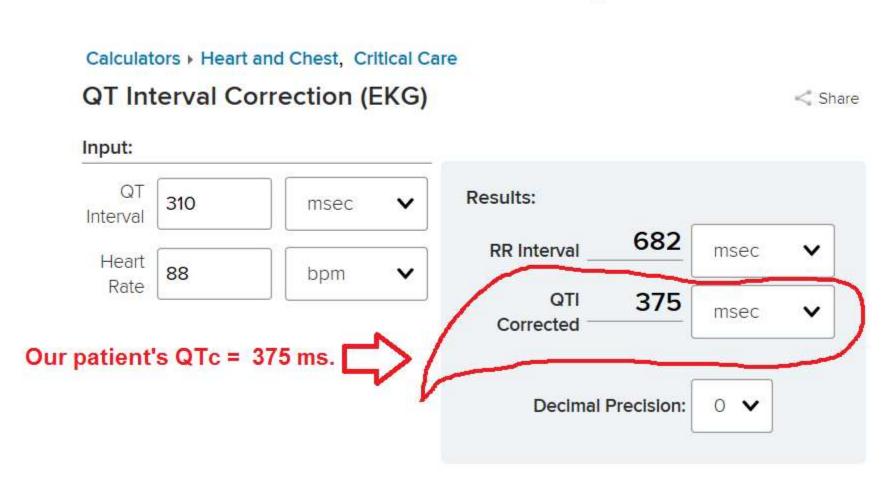


The information contained within this application is for informational purposes only and does not constitute medical or health advice. You should not rely on the information portrayed in this application as an alternative to medical advice from your doctor or any other professional healthcare provider.

Determining the QTc

Method 3, Use a Web-based App:





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

Dysrhythmia Associated with Mortality, Triggered by LQTS: *Torsades de Pointes*



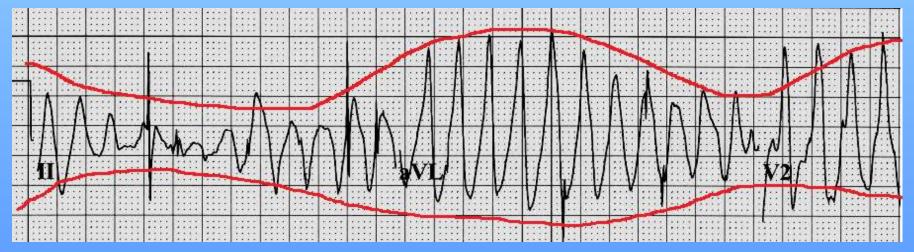
Torsades de Pointes (TdP) – HEMODYNAMICS:

- Decreased to NO Cardiac Output
- Often patient PULSELESS during episode
- Patients often report SYNCOPE when TdP self-terminates.
- May DETERIORATE into VENTRICULAR
 FIBRILLATION and CARDIAC ARREST. ("Sudden Death")

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



a piece of Twisted Ribbon!



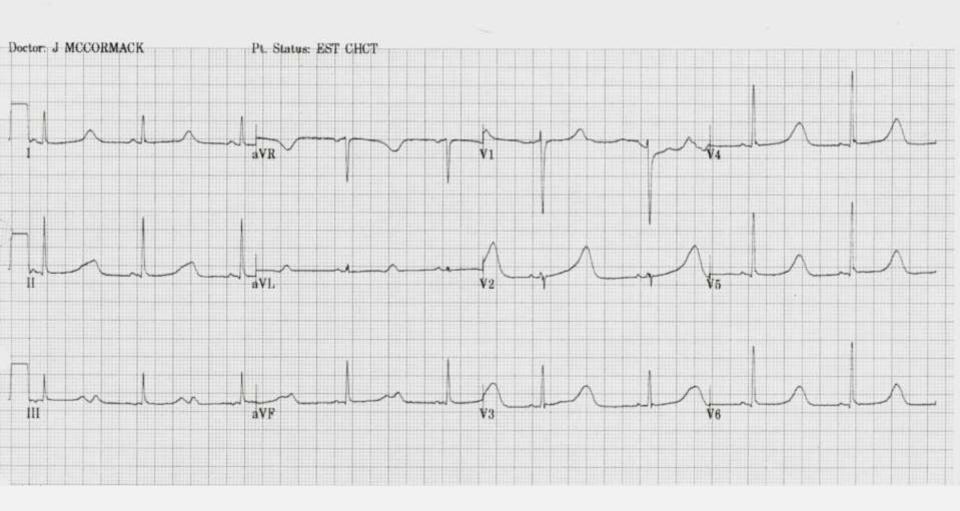
GENETICALLY ACQUIRED LONG QT SYNDROMES:

ECG PATTERNS of 3 MOST COMMON VARIATIONS:

Туре	Current	Functional Effect	Frequency Among LQTS	ECG ^{12,13}	Triggers Lethal Cardiac Event ¹⁰	Penetrance*
LQTS1	к		30%-35%	~~	Exercise (68%) Emotional Stress (14%) Sleep, Repose (9%) Others (19%)	62%
LQTS2	К		25%-30%		Exercise (29%) Emotional Stress (49%) Sleep, Repose (22%)	75%
LQTS3	Na	1	5%-10%		Exercise (4%) Emotional Stress (12%) Sleep, Repose (64%) Others (20%)	90%

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

Chief Complaint: "Grand-Mal Seizures" With NO postictal phase!



WHEN THE "QUICK PEEK" METHOD for QT INTERAL EVALUATION IS APPLIED TO THE ABOVE ECG, WHAT IS THE RESULT?

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

PROLONGED Q - T INTERVAL

THINK:

- CHECK K+ AND MAG LEVELS
- POSSIBILITY OF TORSADES

PROLONGED Q - T INTERVAL

THINK:

- CHECK K+ AND MAG LEVELS
- POSSIBILITY OF TORSADES
- QUESTION MEDS THAT PROLONG Q-T

QT Prolongation -- STAT Intervention:

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 -Ritalin

-Procainamide -Pseudophedrine

-Levaquin -Haloperidol

-Erythromycin -Thorazine

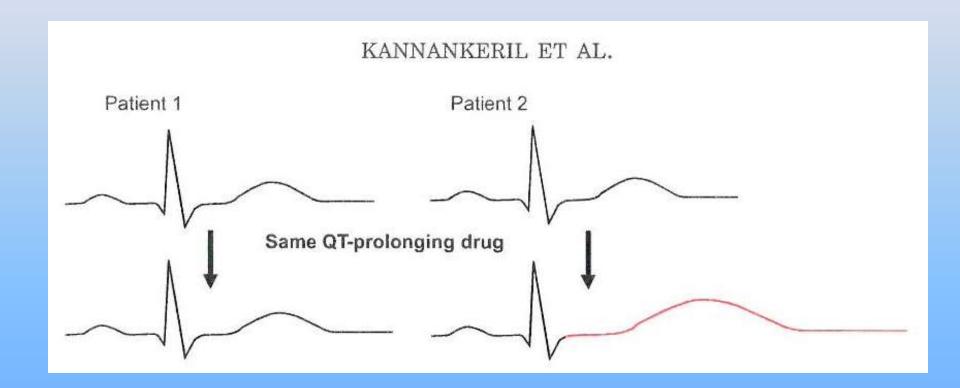
-Norpace -Propulcid

-Tequin -Zofran

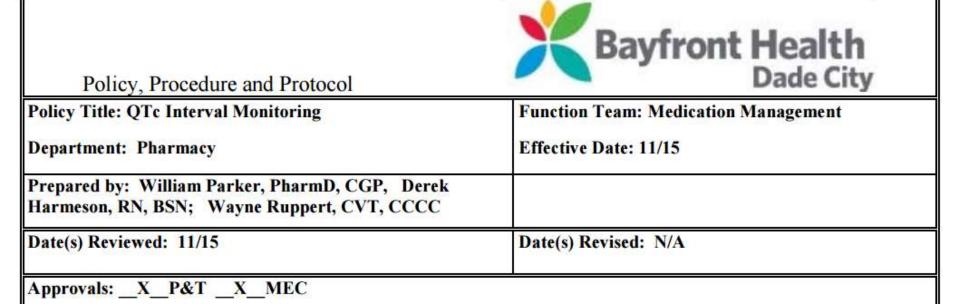
-Benadryl -Ilbutilide *and MANY more!*

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.



PURPOSE:

1.1. To establish a protocol and process by which the Pharmacy and Nursing departments can monitor QTc intervals in patients at high risk for QTc prolongation and subsequently decrease the risk for sudden cardiac death

2. POLICY:

2.1. The Policy, Procedure and Protocol will be utilized selectively and appropriately by the Pharmacy and Nursing staff in order to evaluate and monitor patients at high risk for QTc prolongation and decrease their risk for arrhythmias and sudden cardiac death

Click here to download QTc Interval Monitoring Policy



Results of QTc Monitoring Protocol - Trial - March 8 - March 22

In patients with QTc 500 or more (indicated by red arrow), QT prolonging drugs were discontinued and substituted with non-QT prolonging medications.

	3/8/2016	3/9/2016	3/10/2016	3/11/2016	3/14/2016	3/15/2016	3/16/2016	3/17/2016	3/18/2016	3/21/2016	3/22/2016
PATIENT:	107-141								7-8		
A	389	400									
В	425	437									
C	469	479	528	470	630	500	480				
D	465	426	400	370	470						
- E	559	495	480								
F	418										
G			370	420	460	420	460				
н			390	420							
- 1			416	430							
J			400	400							
К			435								
L			410	400	430	410	440	420	478	430	
- M					510						
N	ı.				480						
0	QTc	Men	Women		470						
P	Abnormal	>450	>460		500						
Q	Panic	500+	500+			400	420	400	413		
R				Ĭ		440					
S						430	440	460			
т	1						400	480			
U								430			
V									491		
w									441	440	44
- X											53
Υ											46
Z											39

QTc Medications - Monitoring Protocol

developed by: William Parker, Director of Pharmacy, Bayfront Health Dade City

Derek Harmeson, Director of ICU/CPCU

Wayne Ruppert, Cardiovascular Coordinator, Bayfront Health Dade City

Bayfront Health Dade City is a 120 bed community hospital with an accredited chest pain center and an interventional cardiac catheterization program in Dade City, Florida.

Click for link to: "Predicting the Unpredictable;
Drug-Induced QT Prolongation and Torsades de
Pointes: J Am Coll Cardiol. 2016;67(13):16391650

Click for link to "AHA ACC Scientific Statement:

Prevention of Torsades de Pointes in the Hospital
Setting," AHA Circulation 2010;

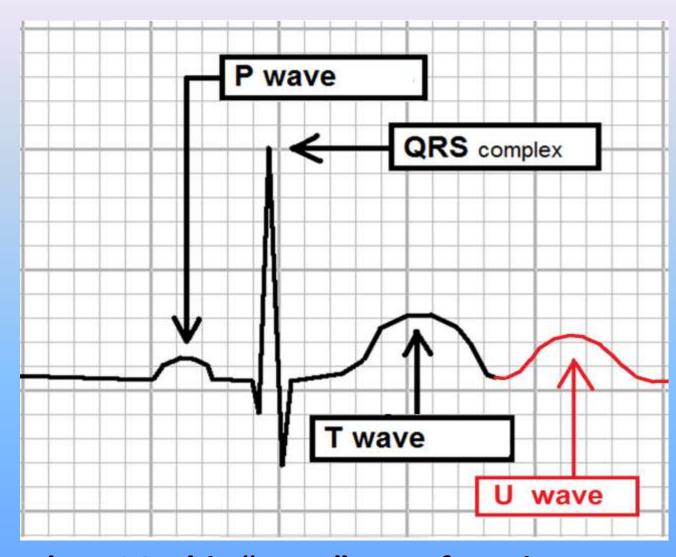
Click for link to hospital model policy & procedure for: "QT Prolonging Medications; QT interval monitoring"

U Waves

Occasionally an extra wave is noted after each T wave.

It typically resembles

"a secondary T wave."



When present on the ECG, this "extra" waveform is referred to as a "U Wave."

U Waves . . .

- Common U wave Etiology:
 - Hypomagnesemia*
 - Hypokalemia*
 - Hypercalcemia*
 - QT prolonging medications*
 - Increased intracranial pressure*
 - Hypothermia*
 - Digitalis (usually shortens the QT Interval)

* These are also causes of QT interval prolongation.

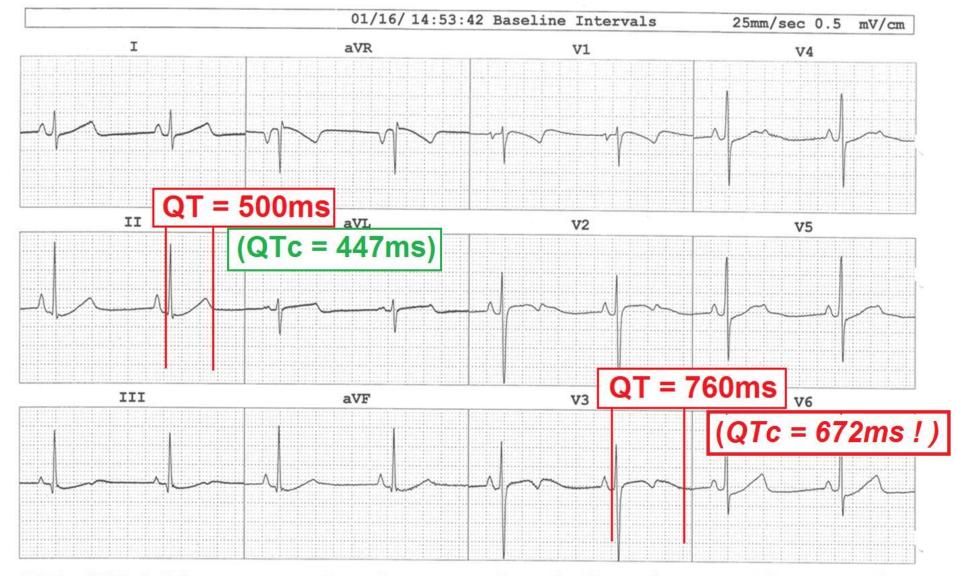
Abnormal U Waves

INCLUDE the U Wave in the QT Interval measurement when any one or more criteria are present:

- U wave 100% (or more) the size of the T wave.
- U wave is INVERTED (opposite polarity of T wave)
- U wave merged with the T wave

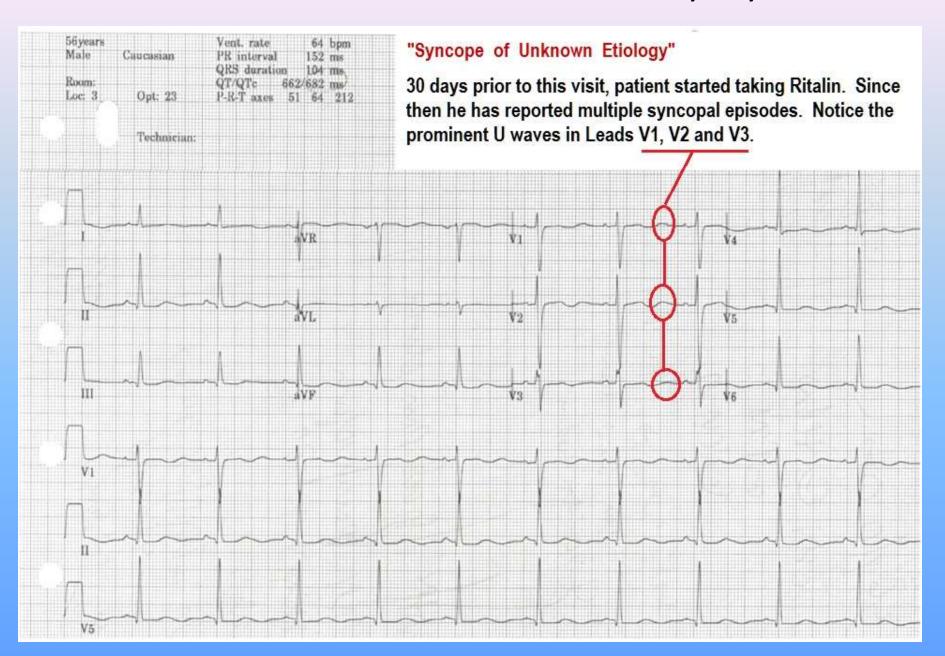
EVIDENCE SOURCE:

ACC/AHA/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram Part IV: The ST Segment, T and U Waves, and the QT Interval.

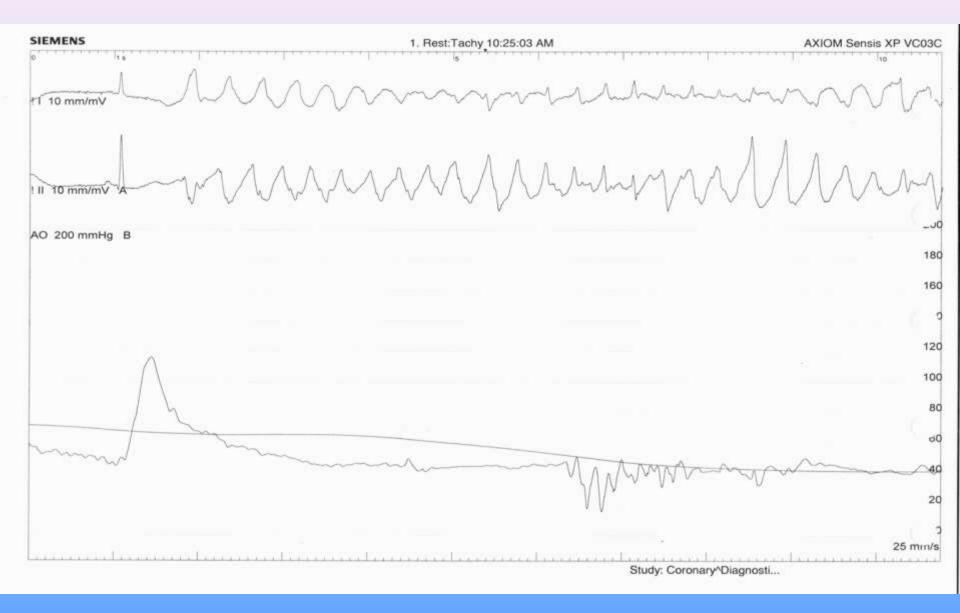


This ECG illustrates the degree of variation that can be noted between different leads on the 12 Lead ECG. ALWAY measure the QT Interval in the lead with the GREATEST value.

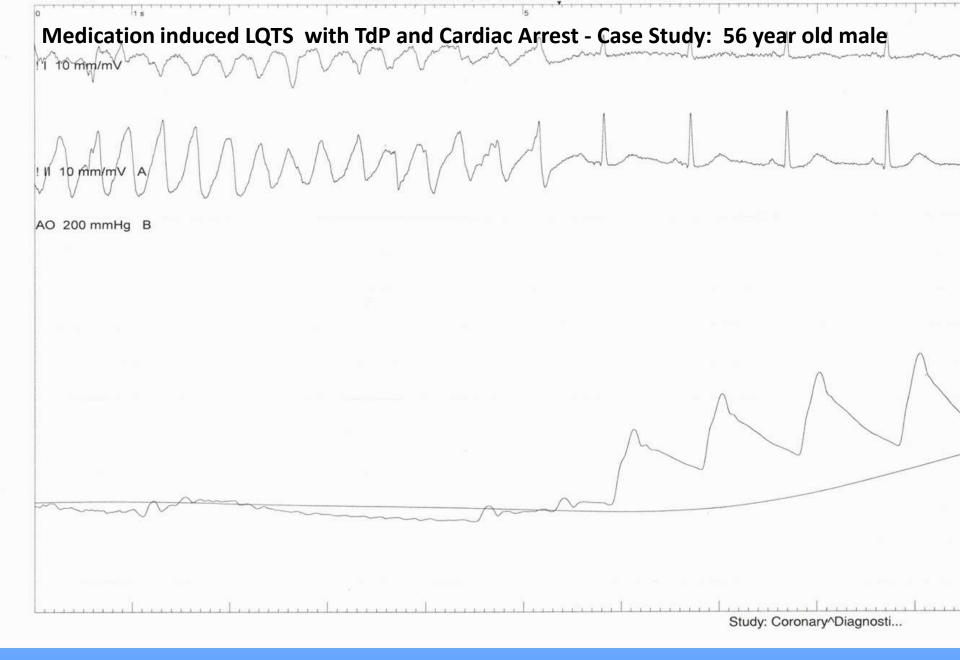
Medication induced LQTS with TdP and Cardiac Arrest - Case Study: 56 year old male



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

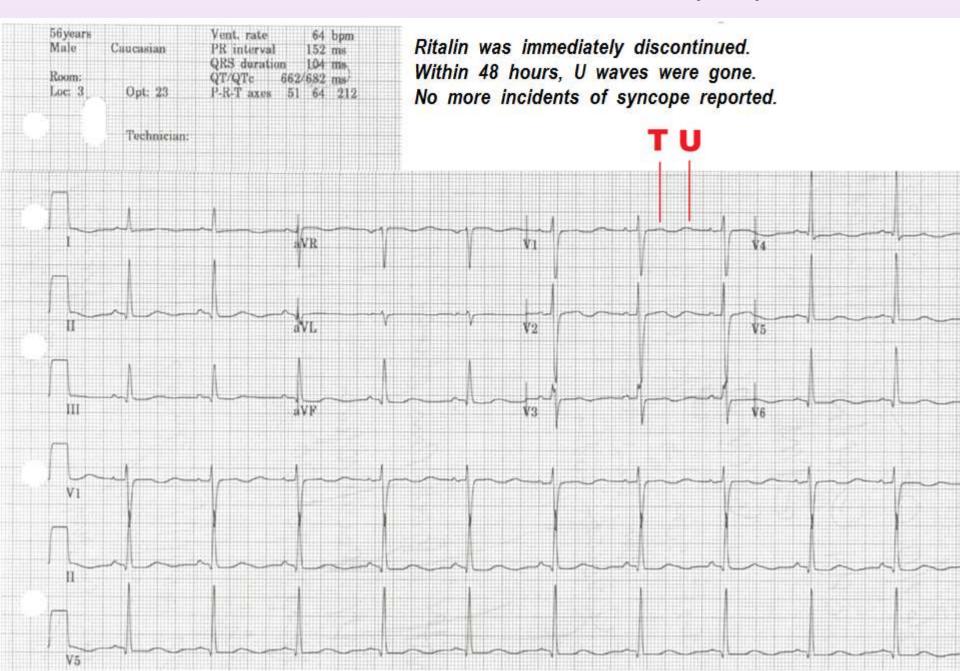


Run of Torsades de Pointes occurred during Cardiac Catheterization . . .



Torsades de Pointes self-terminates just before aborted Defibrillation

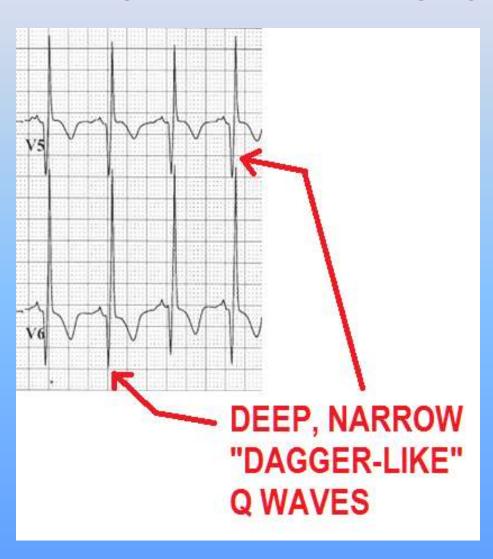
Medication induced LQTS with TdP and Cardiac Arrest - Case Study: 56 year old male



ECG Indicators: Hypertrophic Cardiomyopathy

- ECG may be normal
- Deep, narrow (dagger-like) Q waves

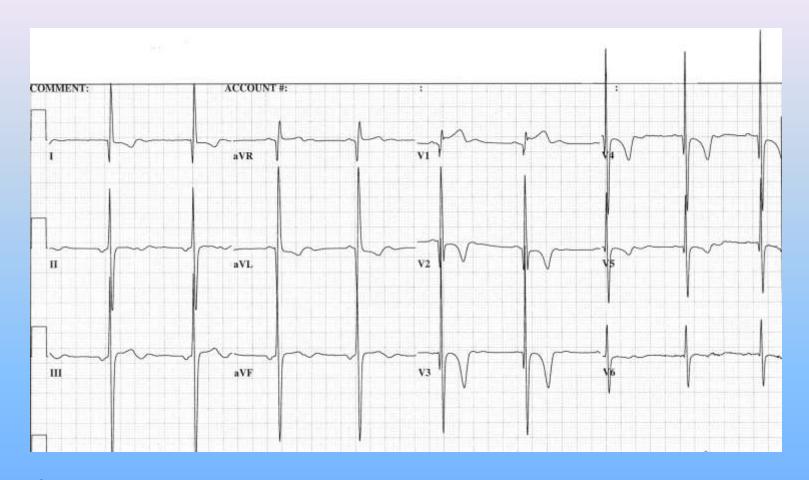
ECG Indicators: Hypertrophic Cardiomyopathy



ECG Indicators: Hypertrophic Cardiomyopathy

- ECG may be normal
- Deep, narrow (dagger-like) Q waves
- Inverted T waves in multiple regions
- <u>Left Ventricular and possibly Left Atrial</u>
 <u>Hypertrophy</u>

Hypertrophic Cardiomyopathy (HCM)



12 Lead ECG Traits:

- QRS Height -- exceeds normal size, "spearing through QRS" in other leads
- Inverted T waves appear in multiple regions (ANTERIOR, LATERAL)
- BiPHASIC T waves in Inferior Leads.
- T WAVES are SYMMETRICAL.

ECG Indicators: Brugada Syndrome

IS THERE ANYTHING ABNORMAL WITH THIS EKG?

37 yr Female Vent. rate 62 BPM Caucasian PR interval 180 QRS duration 88 Room:C4A QT/QTc P-R-T axes 418/424 Option:23 Loc:3 37 22

Normal sinus rhythm Normal ECG No previous ECGs available

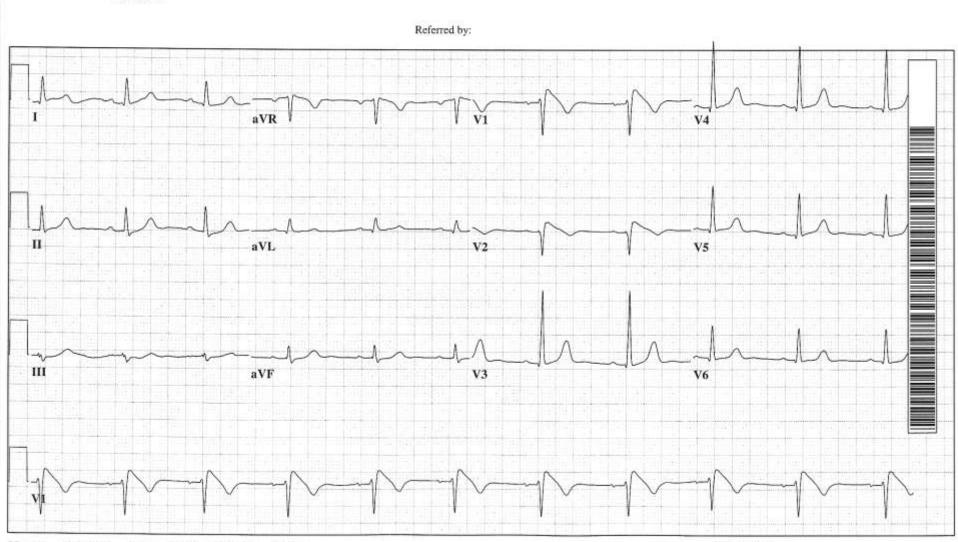
ms

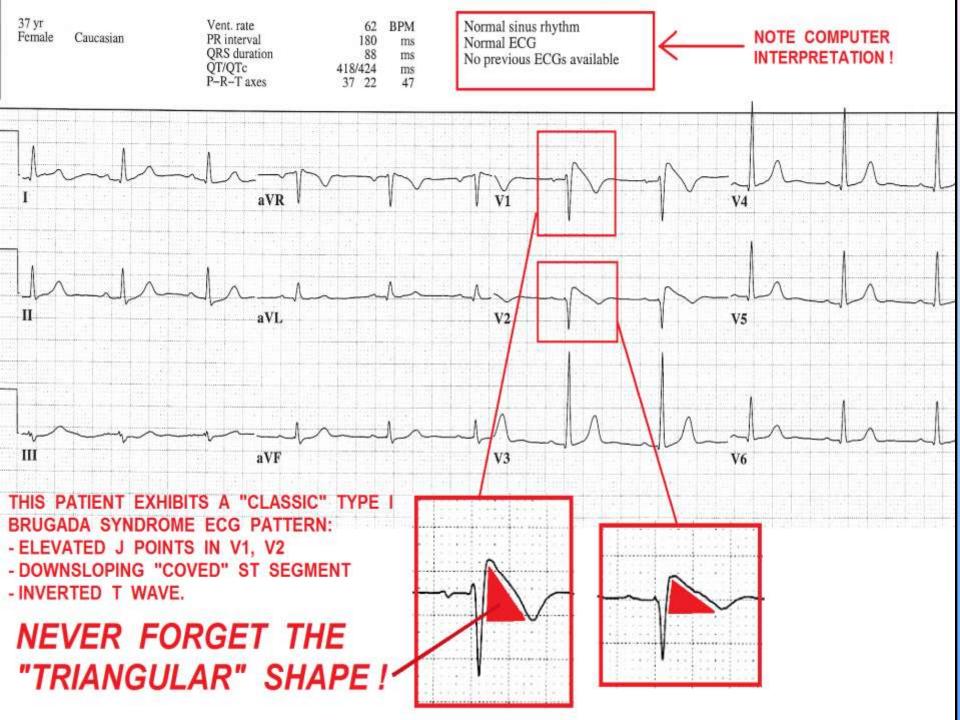
ms

ms

47

Technician:

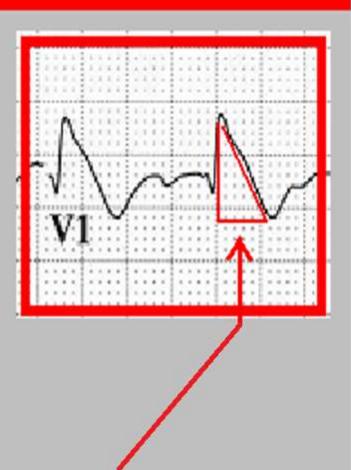




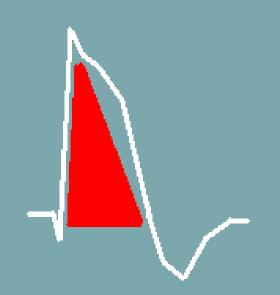
BRUGADA SYNDROME

- 1. RBBB PATTERN
- 2. J POINT ELEVATION V1, V2 and possibly V3
- 3. DOWNWARD SLOPING S-T SEGMENT
- 4. INVERTED T WAVE





PATTERNS of S-T ELEVATION:



BEWARE of the

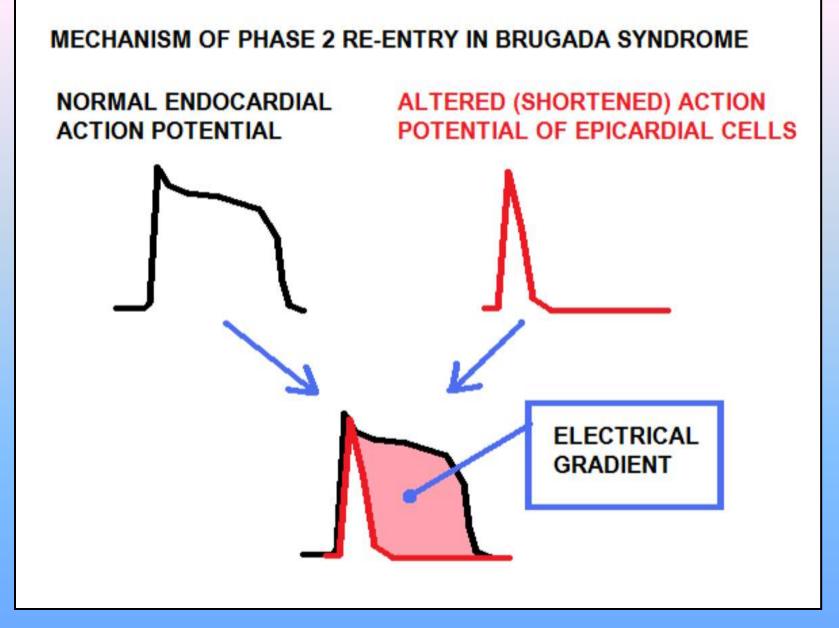
"TRIANGULAR"
SHAPED S-T SEGMENT
IN V1, V2, and sometimes also in V3 . . .

THINK - -



BRUGADA SYNDROME





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

Brugada / Long QT Syndromes cause:



Torsades de Pointes:

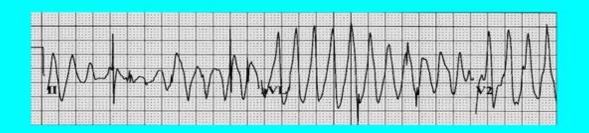
- Decreased to NO Cardiac Output
- Often patient PULSELESS during episode
- Causes SYNCOPE
- Often DETERIORATES into VENTRICULAR FIBRILLATION and CARDIAC ARREST.

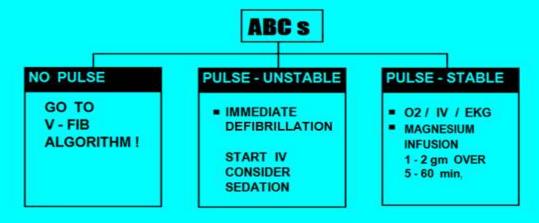
TREATMENT OF TORSADES de POINTES per AHA ACLS 2015:

- -TRANSIENT: MAGNESIUM SULFATE 1 2 gm IV infusion over 5 60 minutes.
- -PERSISTENT, PATIENT UNSTABLE: DEFIBRILLATION
- -CARDIAC ARREST: FOLLOW Ventricular Fibrillation Algorithm. Consider Mag Sulfate as your Antiarrhythmic of choice.

WIDE COMPLEX TACHYCARDIA TORSADES de POINTES

(QRS > 120 ms)





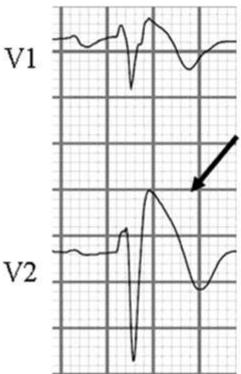
DO NOT give PROCAINAMIDE, AMIODARONE, or SOTALOL to patients with TORSADES or POLYMORPHIC VT !!!

OTHER CONSIDERATIONS:

- EVALUATE BASELINE ECG RHYTHM FOR PRONGED Q-T INVERVAL.
- EVALUATE PATIENT'S MEDS FOR Q-T PROLONGING DRUGS
 - ... if PATIENT HAS BEEN RECEIVING ANY Q-T PROLONGING DRUGS, IMMEDIATELY DISCONTINUE AND CONTACT PHYSICIAN STAT.
- EVALUATE PATIENT HISTORY FOR PREVIOUS EVENTS OF "SYNCOPE OF UNKOWN ETIOLOGY"
- EVALUATE PATIENT FOR FAMILY HISTORY FOR SUDDEN CARDIAC DEATH

REPORT ANY ABNORMAL FINDINGS TO PHYSICIAN.

ECG abnormality diagnostic or suspected of Brugada syndrome.



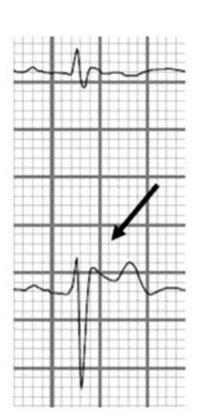
Type 1:

Coved type

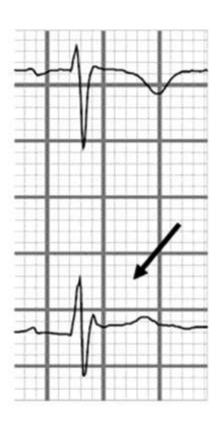
ST-segment

elevation

Association_®



Type 2: saddle-back type ST-segment elevation



Type 3: Saddle-back type "ST-segment elevation"



33 y/o FEMALE

Vent. rate PR interval QRS duration QT/QTc P-R-T axes 129 BPM * ms 112 ms 398/583 ms * 121 -2 Undetermined rhythm

Incomplete right bundle branch block

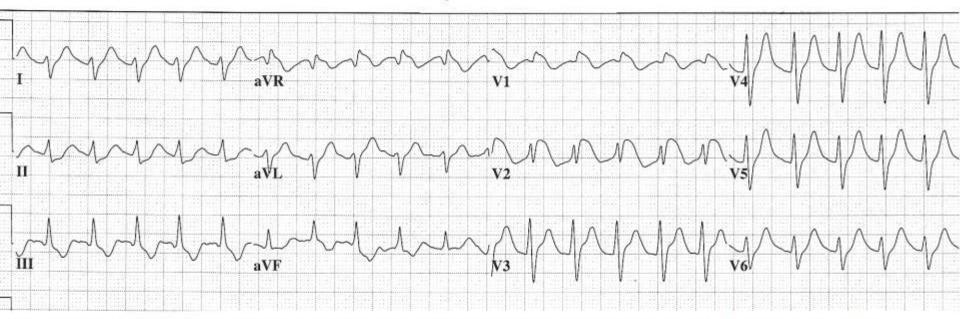
Right ventricular hypertrophy

ST elevation consider anterior injury or acute infarct

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

Abnormal ECG

No previous ECGs available



PT. BROUGHT TO EMERGENCY DEPARTMENT BY EMS AFTER SUFFERING SPONTANEOUS CARDIAC ARREST. PATIENT DID NOT EXPERIENCE ANY SYMPTOMS PRIOR TO COLLAPSE. HAD SEVERAL EPISODES OF NEAR-SYNCOPE IN THE PAST 10 YEARS. CARDIAC CATHETERIZATION REVEALED NO EVIDENCE OF CARDIOVASCULAR DISEASE. NORMAL LV FUNCTION.

DIAGNOSIS: BRUGADA SYNDROME. PT. RECEIVED ICD PRIOR TO HOSPITAL DISCHARGE.

VISIT: www.BRUGADA.org FOR MORE INFORMATION.

42 y/o FEMALE

 Vent. rate
 86
 BPM

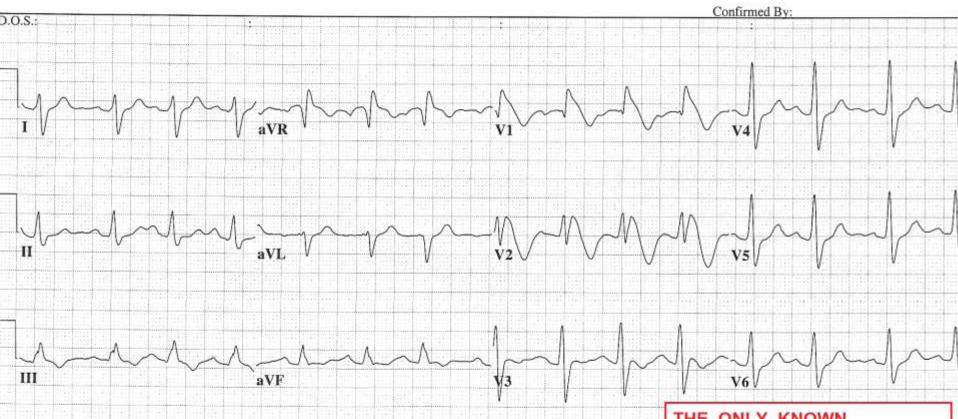
 PR interval
 200
 ms

 QRS duration
 148
 ms

 QT/QTc
 414/495
 ms

 P-R-T axes
 64
 114
 17

Abnormal ECG No previous ECGs available



BRUGADA SYNDROME.

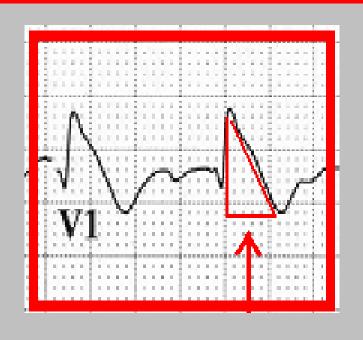
PATIENT HAD HISTORY of SYNCOPE of UNKNOWN ETIOLOGY. FAMILY HISTORY of SUDDEN DEATH of YOUNG, HEALTHY ADULTS.

VISIT: www.BRUGADA.org FOR MORE INFORMATION!

THE ONLY KNOWN
TREATMENT FOR BRUGADA
SYNDROME IS IMPLANTATION
of an ICD. THIS PATIENT
HAD ICD IMPLANTED PRIOR
TO HOSPITAL DISCHARGE.

BRUGADA SYNDROME

- SEVERAL VARIATIONS of this disorder are known to exist.
- CONCEALED and NON-CONCEALED.
- The NON-CONCEALED version HAS THE V1-V3 abnormality VISIBLE at all times.



 The CONCEALED version - pt. has a NORMAL EKG at most times - a DRUG STUDY, an EP STUDY, and / or GENETIC TESTING must be done to rule out or confirm diagnosis.

Arrhythmogenic Right Ventricular Dysplasia

- A genetically acquired myocardial disease associated with paroxysmal ventricular arrhythmias and sudden cardiac death.
- Characterized pathologically by fibro-fatty replacement of the right ventricular myocardium.
- The second most common cause of sudden cardiac death in young people (after HOCM), causing up to 20% of sudden cardiac deaths in patients < 35 yrs of age.
- Typically inherited as an autosomal dominant trait, with variable penetrance and expression (there is an autosomal recessive form called Naxos Disease, which is associated with woolly hair and skin changes).
- More common in men than women (3:1) and in people of Italian or Greek descent.
- Estimated to affect approximately 1 in 5,000 people overall.

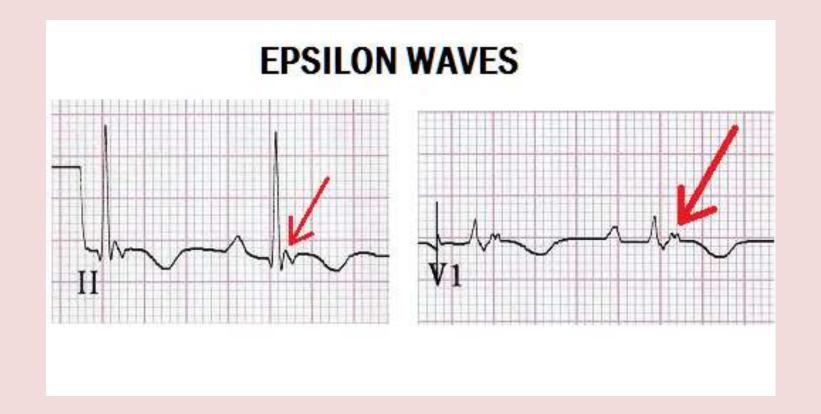
From: 2014 ACC/AHA Guideline on Perioperative Cardiovascular Evaluation and Management of Patients Undergoing Noncardiac Surgery: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

Arrhythmogenic Right Ventricular (RV) Cardiomyopathy and/or Dysplasia:

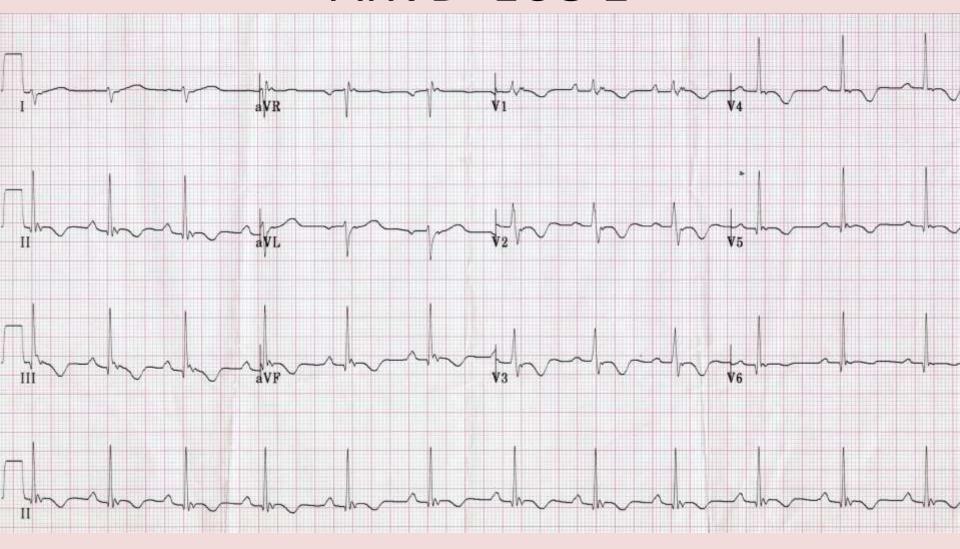
In 1 autopsy study examining a series of 200 cases of sudden death associated with arrhythmogenic RV cardiomyopathy and/or dysplasia, death occurred in 9.5% of cases during the perioperative period. This emphasizes the importance of close perioperative

evaluation and monitoring of these patients for ventricular arrhythmia. Most of these patients require cardiac electrophysiologist involvement and consideration for an implantable cardioverter-defibrillator (ICD) for long-term management.

ARVD – 12 Lead ECG Indicators



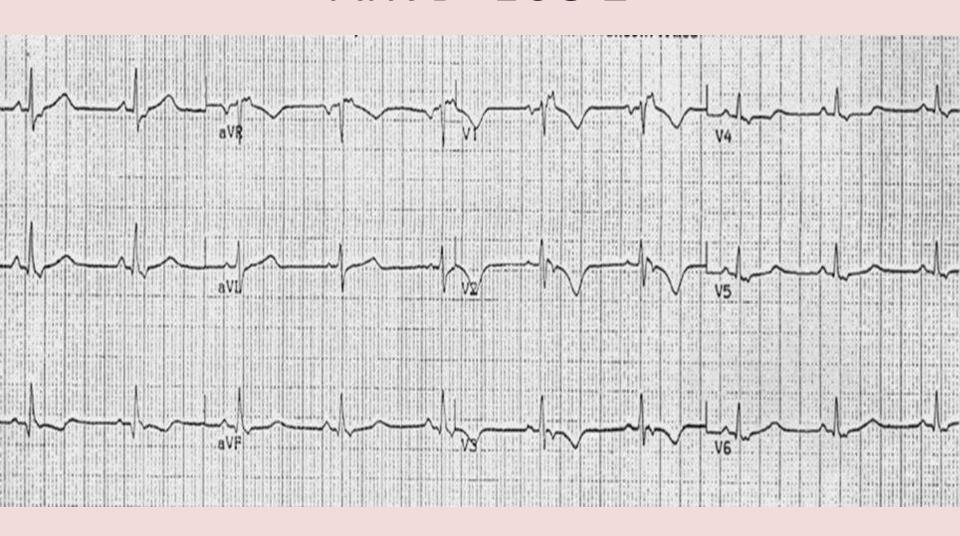
ARVD ECG 1



- 1. "Incomplete RBBB" Pattern
- 2. V1, V2 Rs pattern
- 3. Inverted T waves, symmetrical, Global

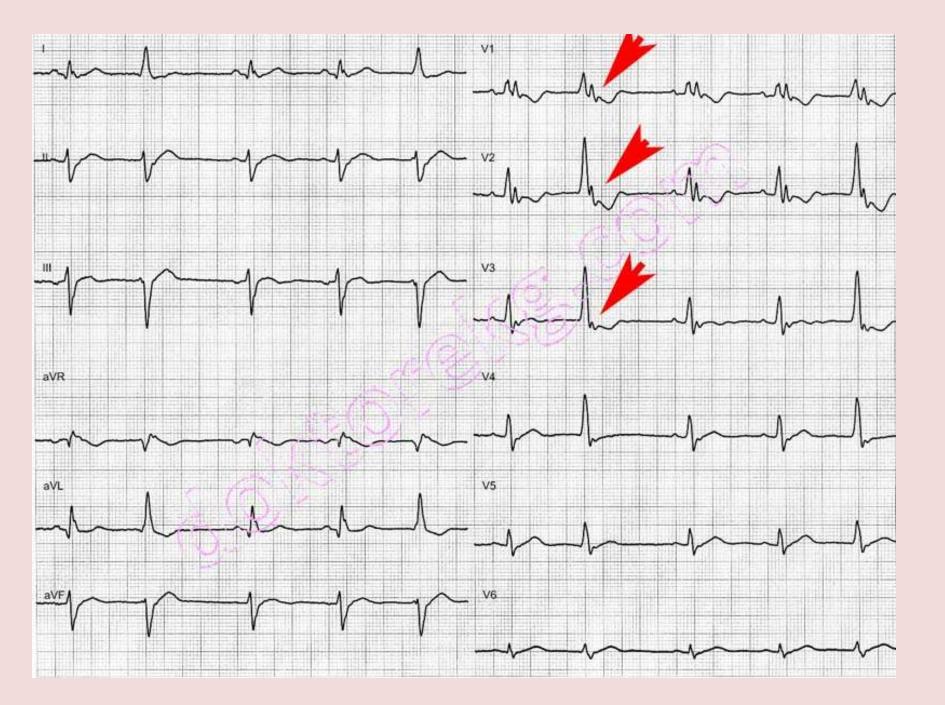
4. Epsilon's waves

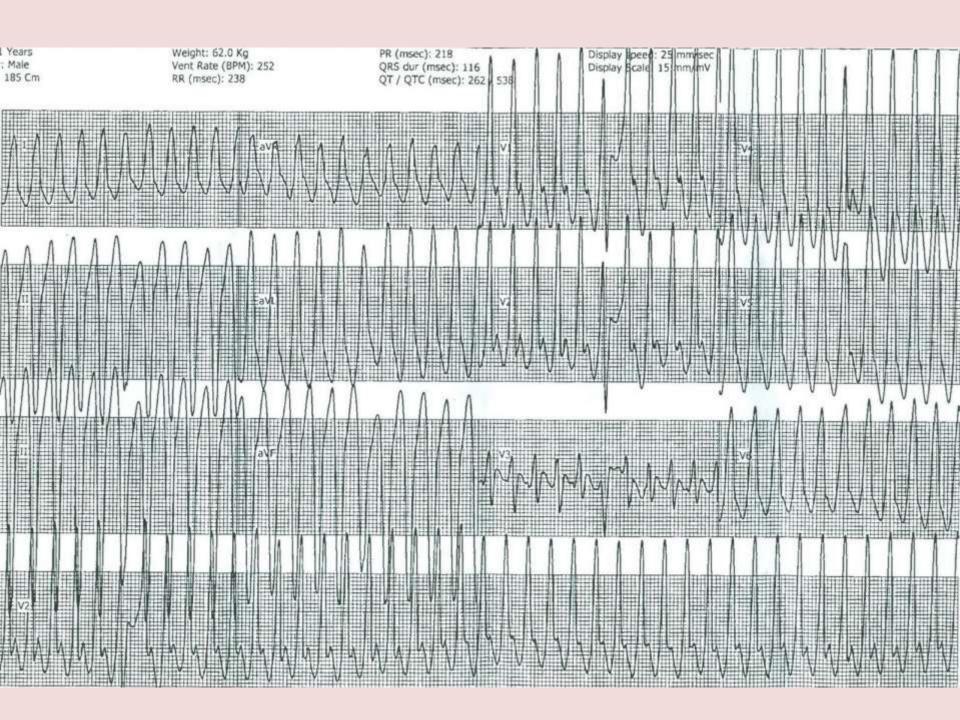
ARVD ECG 2



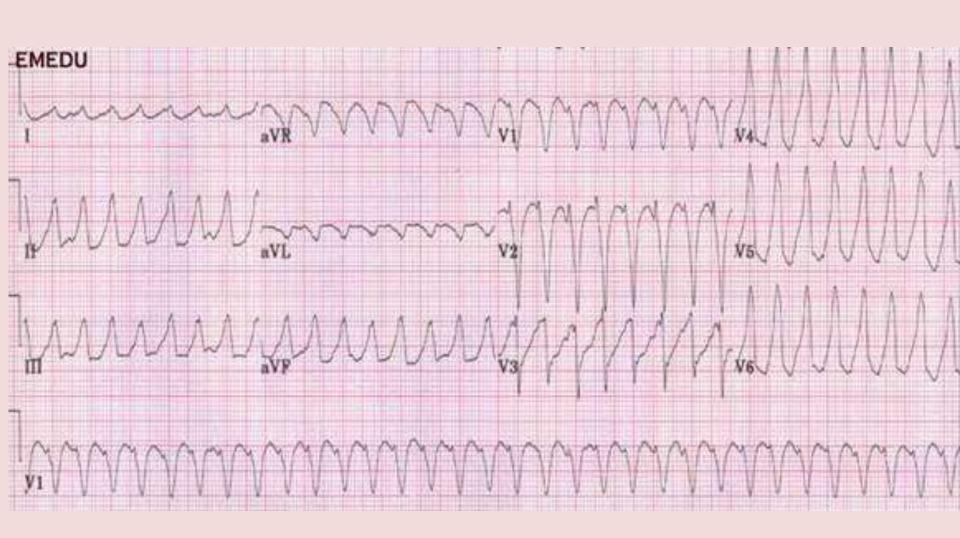
- 1. "Incomplete RBBB" Pattern
- 2. V1, V2 Rs pattern
- 3. Inverted T waves, symmetrical, Global

4. Epsilon's waves





ARVD INDUCED VT



Evidence Based Reference Sources

- 2016 ACC Interassociation Consensus Statement on Cardiovascular Care of College Student-Athletes
- 2014 AHA/ACC Scientific Statement: Assessment of the 12-Lead ECG as a Screening Test for Detection of Cardiovascular Disease in Healthy General Populations of Young People (12–25 Years of Age)
- AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram: Part IV: The ST Segment, T and U Waves, and the QT Interval: Circulation 2009 119: e241-e250
- AHA Circulation: Inherited Arrhythmias; Basic Science for Clinicians
- AHA ACC Scientific Statement Prevention of Torsade de Pointes in Hospital Settings
- AHA ACC QTc Behavior During Exercise and Genetic Testing for the Long-QT Syndrome
- Pharmacology Review: Drug Induced Long QT Syndromes

Evidence Based Reference Sources, cont'

- HRS/EHRA/APHRS Expert Consensus Statement on the Diagnosis and Management of Patients with Inherited Primary Arrhythmia
 Syndromes
- Genetic Determinants of Sudden Cardiac Death: AHA Circulation.2008; 118: 1854-1863
- AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram: Part III: Intraventricular Conduction Disturbances
- AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram: Part V: Electrocardiogram Changes Associated With Cardiac Chamber Hypertrophy
- Arrhythmogenic Disorders of Genetic Origin; Brugada Syndrome:
 Circulation: Arrhythmia and Electrophysiology.2012; 5: 606-616

Other Reference Sources:

www.JACC.org

http://circ.ahajournals.org/



www.SADS.org

THE ACUTE CORONARY SYNDROMES



- STEMI
- NSTEMI
- UNSTABLE ANGINA / OBSTRUCTIVE C.A.D.



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

*Risk factors for atherosclerotic disease:

Hypercholesterolemia Hypertension Diabetes Mellitus

Cigarette smoking Positive family history Obesity \mathbf{H} = chest pain = 2

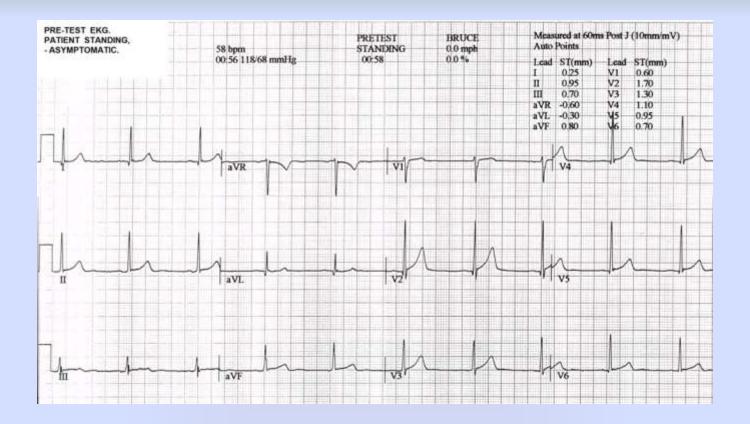
 $\mathbf{E} = \mathbf{ECG} \text{ normal} = \mathbf{0}$

A = 63 = 1

 $\mathbf{R} = 3 \text{ risk fctors} = 2$

T = Trop. NL = 0

HEART Score: = 5



2 patients with the above ECG.

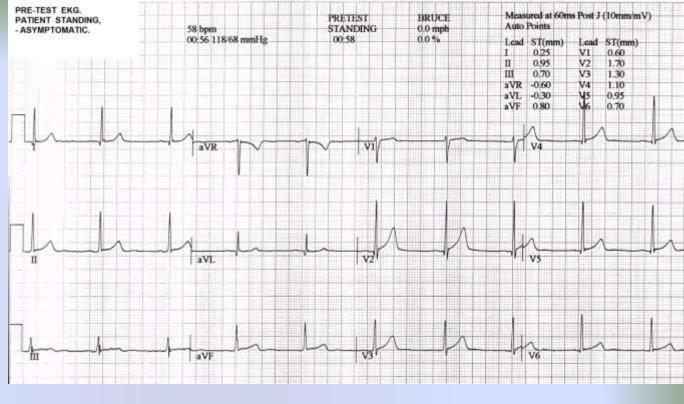
- Patient 1 HEART Score of "0"
- Patient 2 HEART Score of "5"

Should they get the same care ??

PROBLEMS WITH SENSITIVITY . . .

NORMAL ECG.

But



His HEART Score = 5

What could that possibly mean?

4 - 6	Intermed.	Suspect: ACS, Obstructive CAD, Unstable Angina NSTEMI	Admit to hospital, Serial ECGs /Troponins aggressive diagnositic work-up (e.g. Cardiac Cath, CT coronary angio
7 - 10	HIGH	NSTEMI STEMI	STEMI= STAT PCI or thrombolytics. NSTEMI = "urgent" Cardiac Cath

ACS

Dx?

Non-ACS

Proposed

Discharge with

follow-up / out-

patient stress

Management

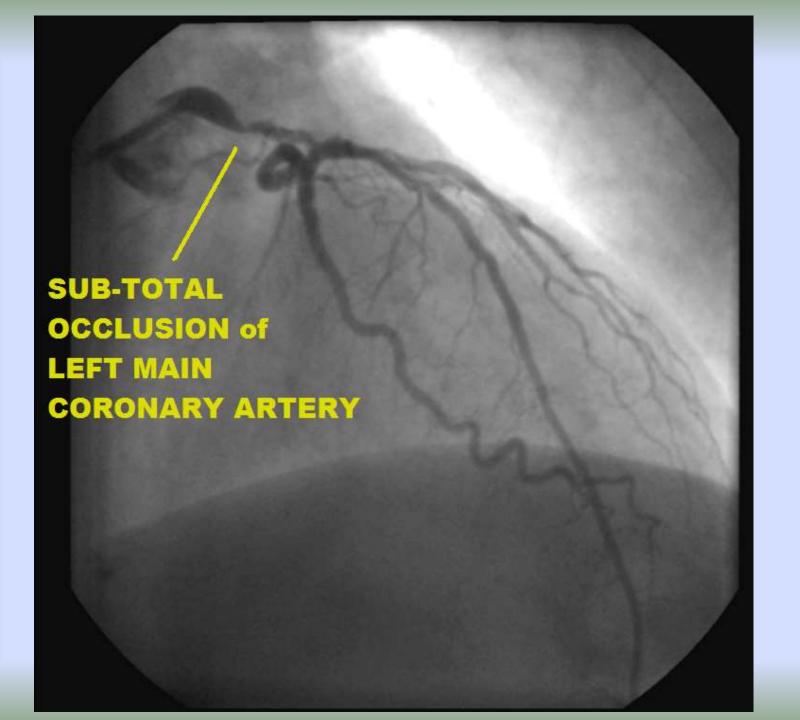
HEART

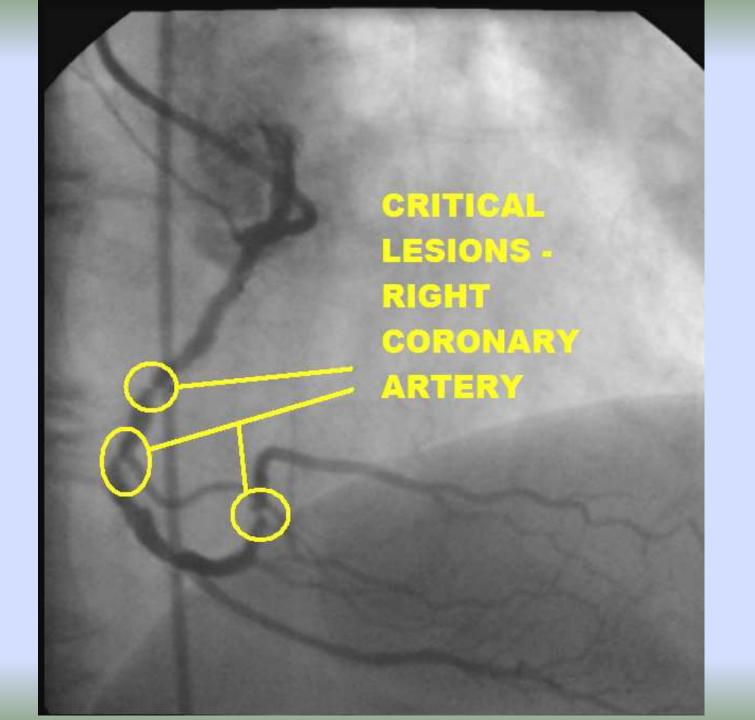
Score

0 - 3

RISK

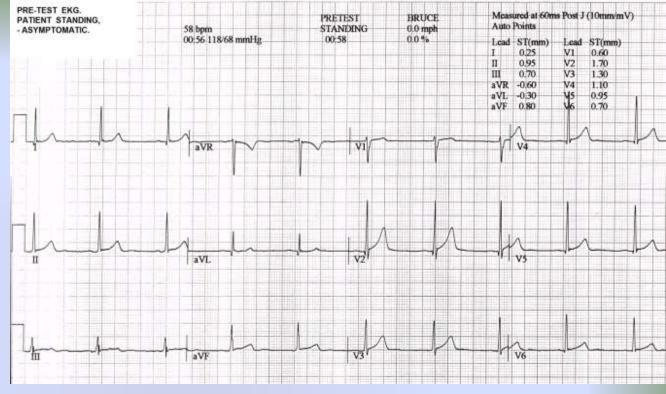
LOW

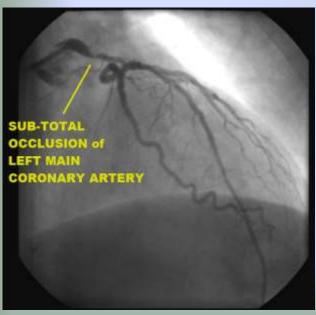


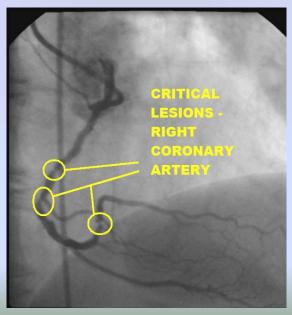


Heart Score 5.

Lethal
Triple
Vessel
Disease =







Emergency
Triple Vessel
Coronary
Artery
Bypass
Surgery

H = HISTORY

- <u>2 Points</u>: "Suspicious" = Typical ACS Symptoms
- 1 Point: "Moderately Suspicious" = Atypical ACS Symptoms
- O Points: No Typical or Atypical Symptoms of ACS



TYPICAL SYPTOMS of ACUTE CORNARY SYNDROME:

- \checkmark
- CHEST PAIN DESCRIBED AS ...
- "HEAVINESS, PRESSURE, DULL PAIN, TIGHTNESS"
- CENTERED IN CHEST, SUBSTERNAL
- MAY RADIATE TO SHOULDERS, JAW, NECK, LEFT or RIGHT ARM
- NOT EFFECTED by:
 - MOVEMENT
 - POSITION
 - DEEP INSPIRATION
- \checkmark
 - SHORTNESS OF BREATH
 - MAY or MAY NOT BE PRESENT
- **√**
 - NAUSEA / VOMITING
 - MAY or MAY NOT 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

stable angina

- SYMPTOMS START DURING PHYSICAL EXERTION.
- 2. SYMPTOMS ARE "PREDICTABLE"



unstable angina

- 1. SYMPTOMS MAY START AT ANY TIME, EVEN DURING REST
- 2. SYMPTOMS ARE <u>NEW</u>, <u>DIFFERENT</u>, or <u>WORSE</u> THAN PREVIOUS EPISODES

BEWARE of the patient with "INTERMITTENT CHEST PAIN"....



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) Fatigue

Indigestion Abdominal pain

Nausea Cold sweats

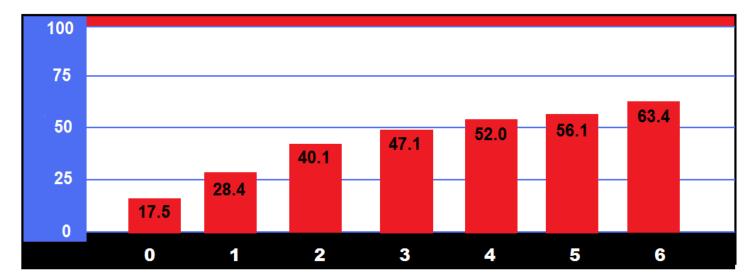
Dizziness Elevated heart rate

Syncope Dsypnea

BOOK PAGE: 70

Effect of Having Multiple Risk Factors for AMI Without Chest Pain

% of PATIENTS with ACUTE MI PRESENTING TO THE EMERGENCY DEPARTMENT WITHOUT CHEST PAIN



NUMBER OF RISK FACTORS PRESENT

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

WOMEN'S MAJOR SYMPTOMS PRIOR TO THEIR HEART ATTACK:

•	UNUSUAL FATIGUE	71 %
•	SLEEP DISTURBANCE	48 %
	SOB	42 %
٠	INDIGESTION	39 %
	ANXIETY	36 %

APPROXIMATELY 78 % OF WOMEN REPORTED EXPERIENCING AT LEAST ONE OF THESE SYMPTOMS FOR MORE THAN ONE MONTH EITHER DAILY OR SEVERAL TIMES PER WEEK PRIOR TO THEIR MI.

WOMEN'S MAJOR SYMPTOMS DURING THEIR HEART ATTACK:

SHORTNESS OF BREATH	58 %
WEAKNESS	55 %
UNUSUAL FATIGUE	43 %
COLD SWEAT	39 %
DIZZINESS	39 %



Circulation, 2003:108;2619-2623

E = ECG

- <u>2 Points:</u> ST Deviation (elevation or depression at the J point of 0.5mv or more)
- 1 Point: Non-specific ST-T wave abnormalities / Non
- O Points: Normal ECG

A = Age

• 2 Points: Age 65 or more

• **1 Point:** Age 46 – 64

• **O Points**: Age 45 or less

R = Risk Factors for CAD

- 2 Points: 3 or more risk factors
- 1 Point: 1 or 2 risk factors
- O Points: No Risk Factors

RISK FACTORS

for the development of

CORONARY ARTERY DISEASE:

- **●** HEREDITY
- $lacktriant{lacktriangle}{}^{**} lacktriangle$ LDL and lacktriangle HDL CHOLESTEROL PROFILES
- **●**** SMOKING
- **●** DIABETES MELLITUS
- OBESITY
- PHYSICAL INACTIVITY
- HYPERTENSION
- AGE OVER 65
- MALE
- HIGH STRESS

T = Troponin

- 2 Points: 3 X Normal
- 1 Point: >1 <3 times normal limit
- <u>O Points</u>: up to normal limit

Modified HEART Score for EMS

- Most EMS units don't have access to "Troponin blood testing."
- The "HEAR" Score ("HEART" minus the Troponin) has been validated by recent a recent study conducted by Cambridge University.
- View Cambridge University Journal article about HEAR Score

OBTAINING THE 12 LEAD ECG

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

Evaluating the ECG for ACS: A TWO-STEP process:

Evaluating the ECG for ACS:

A TWO-STEP process:

STEP 1: Evaluate QRS Width

Evaluating the ECG for ACS:

A TWO-STEP process:

STEP 1: Evaluate QRS Width

STEP 2: Evaluate J Points, STSegment and T waves
in EVERY Lead

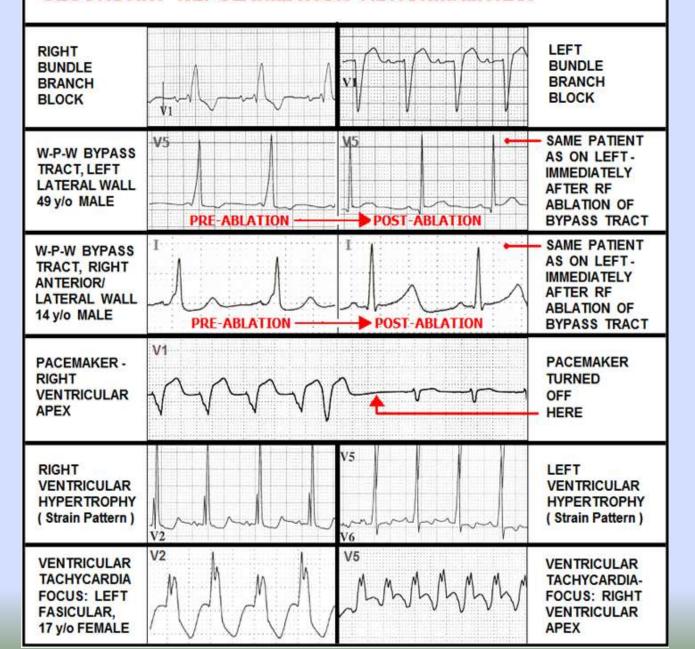
STEP 1 – evaluate QRS width:

- QRS is ABNORMALLY WIDE (>120 ms),
 - indicates DEPOLARIZATION ABNORMALITY
 (e.g. "bundle branch block, Wolff-Parkinson-White Syndrome, etc).

STEP 1 – evaluate QRS width:

- QRS is ABNORMALLY WIDE (>120 ms),
 - indicates DEPOLARIZATION ABNORMALITY
 (e.g. "bundle branch block, Wolff-Parkinson-White Syndrome, etc).
 - DEPOLARIZATION ABNORMALITIES in turn cause REPOLARIZATION ABNORMALITIES, which alters the: *J Points, ST-Segments and/or T Waves*.

CONDITIONS THAT INCREASE QRS DURATION RESULT IN SECONDARY REPOLARIZATION ABNORMALITIES:



Wide QRS present: QRSd > 120ms

 Determine RIGHT vs. LEFT Bundle Branch Block Pattern

Simple "Turn Signal Method" . . .

THE "TURN SIGNAL METHOD" for identifying BUNDLE BRANCH BLOCK

۷1

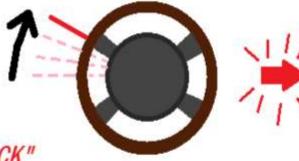
USE LEAD V1 for this technique

To make a RIGHT TURN

you push the turn signal lever UP

THINK:

"QRS points UP = RIGHT BUNDLE BRANCH BLOCK"



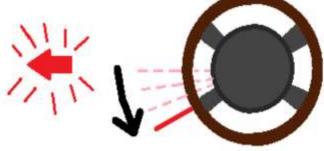
٧1



To make a LEFT TURN

you push the turn signal lever DOWN

THINK:



"QRS points DOWN = LEFT BUNDLE BRANCH BLOCK"

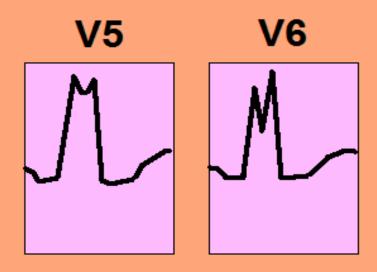
DIAGNOSING BUNDLE BRANCH BLOCK

USING LEADS V1, V2, and V5, V6:

LOCATING RsR' or RR' COMPLEXES:



RIGHT BUNDLE BRANCH BLOCK



LEFT BUNDLE BRANCH BLOCK

From: "Rapid Interpretation of ECGs" by Dale Dubin, MD

The New England Medical Journa/

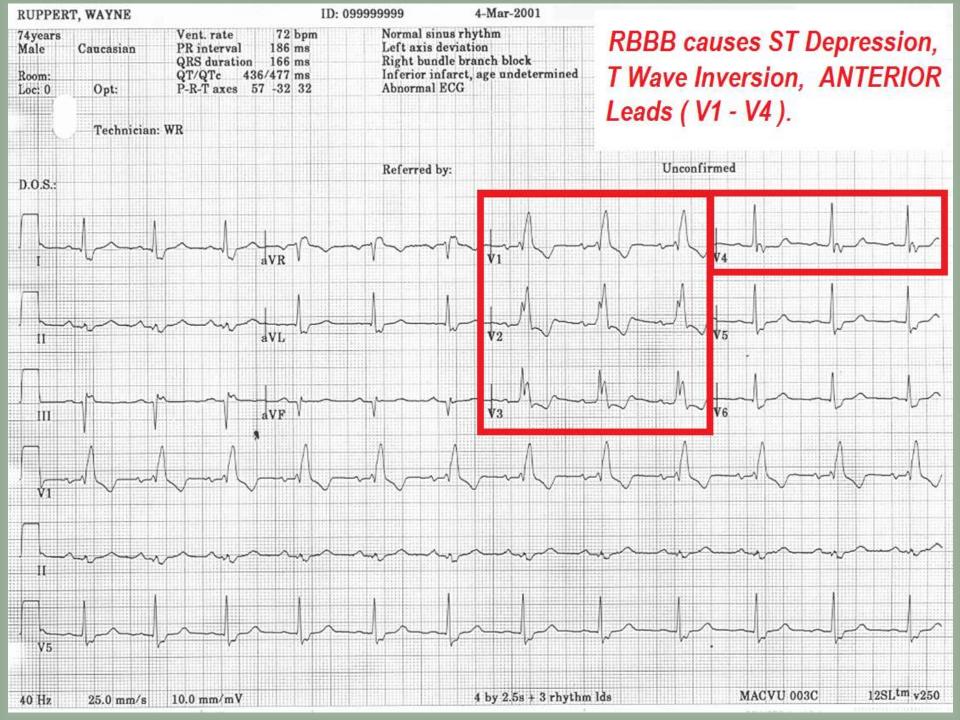


Evaluating the ECG for ACS:

STEP 1 - EVALUATE WIDTH OF QRS: WIDE (> 120 ms) NORMAL (< 120 ms) OF SUPRAVENTRICULAR ORIGIN --DETERMINE QRS MORPHOLOGY: LEFT BUNDLE BRANCH RIGHT BUNDLE BRANCH **BLOCK PATTERN BLOCK PATTERN EVALUATE FOR USE CAUTION --**EVALUATE FOR DO NOT RELY ON ST ELEVATION ST DEPRESSION ST ELEVATION ST DEPRESSION IN USUAL MANNER IN USUAL MANNER AS A MARKER OF ACS. -IS ROUTINELY SEEN IN WIDE QRS COMPLEX WIDE QRS COMPLEX RHYTHMS (both L and R RHYTHMS WITH LBBB **BBB PATTERNS) OFTEN** PATTERN. FOLLOW AHA CAUSE: DEPRESSION of CRITERIA (page 109) FOR J POINTS. ST SEGMENTS. DIAGNOSIS OF STEMI IN & INVERSION OF T WAVES. PRESENCE OF LBBB.

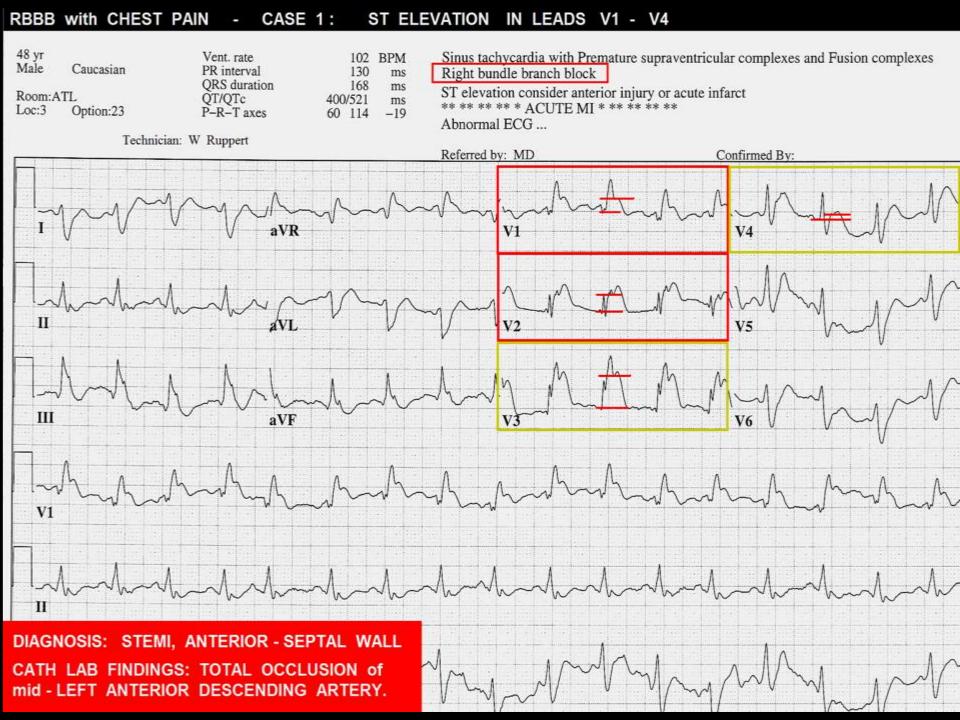
Wide QRS present: (QRSd > 120ms)

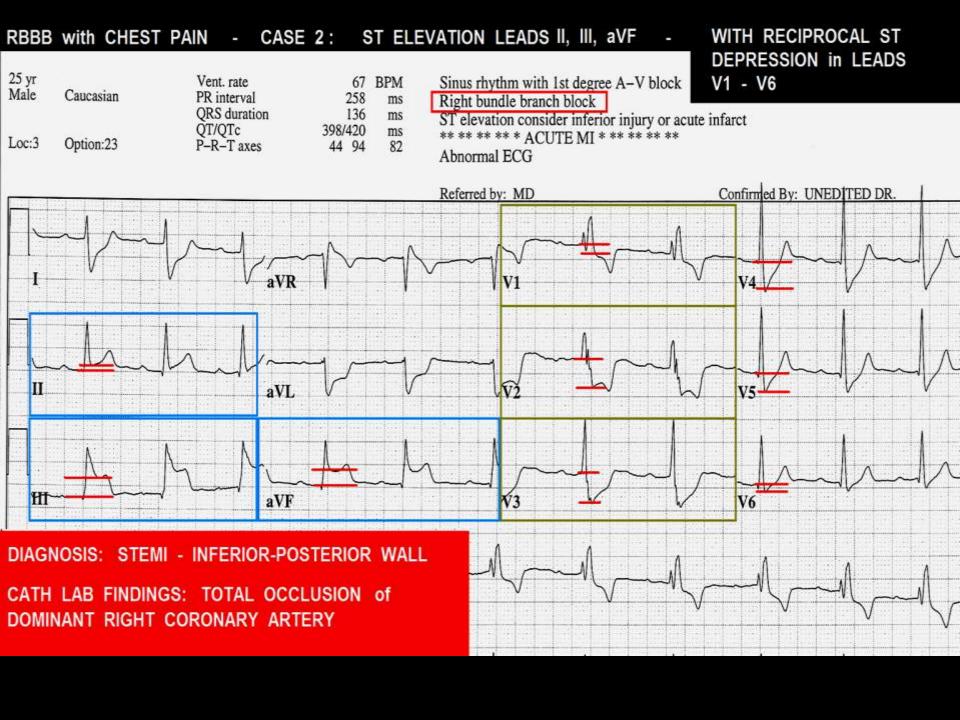
- When RIGHT Bundle Branch Block pattern is present:
 - Precordial Leads typically demonstrate ST
 Depression and T wave Inversion

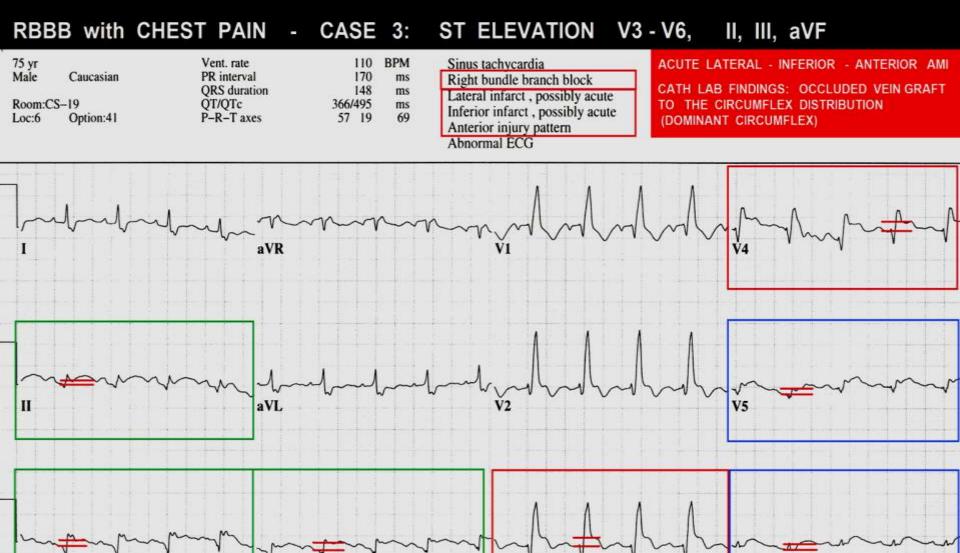


Wide QRS present: (QRSd > 120ms)

- When RIGHT Bundle Branch Block pattern is present:
 - Precordial Leads typically demonstrate ST
 Depression and T wave Inversion
 - DOES NOT MASK STEMI; when ST Elevation is noted, CONSIDER STEMI!!







(QRSd > 120ms)

When LBBB QRS pattern is present:

(QRSd > 120ms)

- When LBBB QRS pattern is present:
 - -ST-Segment Elevation is typically noted in Precordial Leads

(QRSd > 120ms)

- When LBBB QRS pattern is present:
 - ST-Segment Elevation is typically noted in Precordial Leads
 - Can cause up to 5mm of J Point Elevation in normally calibrated ECG (1mm=10mv)

(QRSd > 120ms)

- When LBBB QRS pattern is present:
 - -ST-Segment Elevation is typically noted in Precordial Leads
 - Can cause up to 5mm of J Point Elevation in normally calibrated ECG (1mm=10mv)
 - Does NOT typically cause ST elevation in INFERIOR Leads (II, III and AVF).

2013 ACC/AHA Guideline for Management of STEMI

 ST Elevation of 0.1mv (1mm) or more in leads with Positive Deflection QRS complexes

2013 ACC/AHA Guideline for Management of STEMI

- ST Elevation of 0.1mv (1mm) or more in leads with Positive Deflection QRS complexes
- ST Elevation of 0.5mv (5mm) or more in leads with Negative Deflection QRS complexes

2013 ACC/AHA Guideline for Management of STEMI

- ST Elevation of 0.1mv (1mm) or more in leads with Positive Deflection QRS complexes
- ST Elevation of 0.5mv (5mm) or more in leads with Negative Deflection QRS complexes
- ST Segment Changes as compared with those of older ECGs with LBBB

2013 ACC/AHA Guideline for Management of STEMI

- ST Elevation of 0.1mv (1mm) or more in leads with Positive Deflection QRS complexes
- ST Elevation of 0.5mv (5mm) or more in leads with Negative Deflection QRS complexes
- ST Segment Changes as compared with those of older ECGs with LBBB
- Convex ST Segment

A.H.A. ACLS GUIDELINES

- If patient has a CONFIRMED HISTORY of LBBB, rely on:
 - CARDIAC MARKERS
 - SYMPTOMS
 - RISK FACTOR PROFILE
 - HIGH INDEX OF SUSPICION

for diagnosis of STEMI

- 2. If patient has:
 - a) previously NORMAL ECGs (no LBBB)
 -- or --
 - b) no old ECGs available for comparison

consider diagnosis as STEMI until proven otherwise.



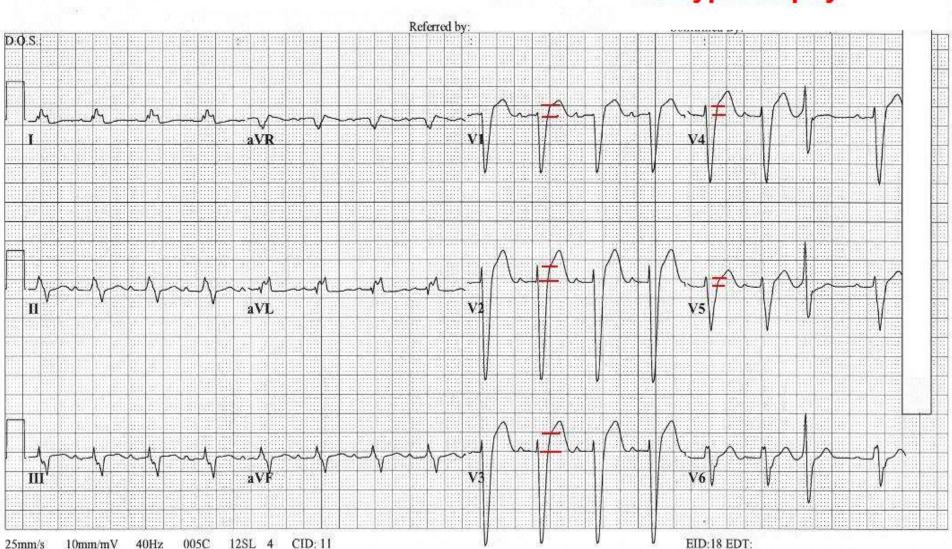
Normal sinus rhythm with occasional Premature ventricular complexes

Left bundle branch block Abnormal ECG - Normal arteries

Normal LV Function

No hypertrophy

Technician: EKG CLASS #WR03602718





HELPFUL INDICATORS FOR ECG DIAGNOSIS OF STEMI in the presence of LBBB:

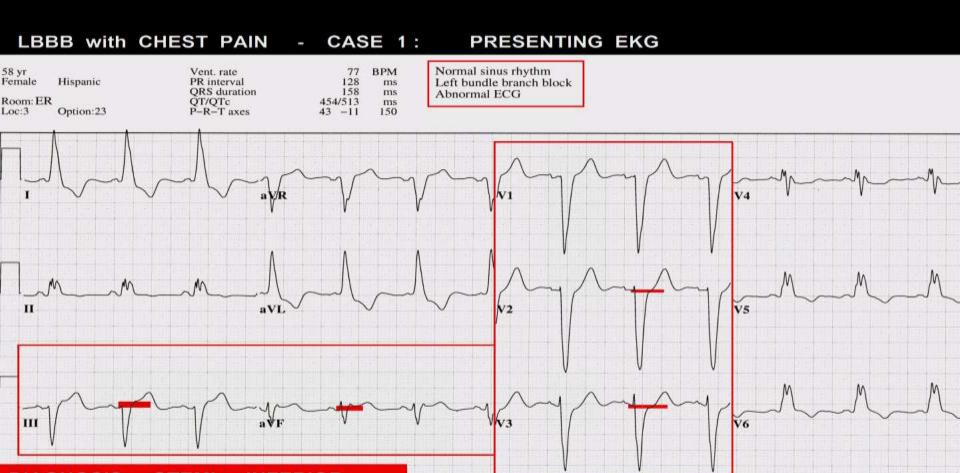
- ST ELEVATION > 5 mm
- COMPARE J POINT, ST SEGMENTS and T WAVES of previous ECG with LBBB to NEW ECG.
- CONVEX ST SEGMENT = poss. MI
 CONCAVE ST SEGMENT = normal
- CONCORDANT ST changes (1 mm or > ST DEPRESSION V1 - V3 or ST ELEVATION LEADS II, III, AVF)
- ST ELEVATION in LEADS II, III, and/or AVF

"Electrocardiographic Diagnosis of Evolving Acute
Myocardial Infarction in the Presence of Left
Bundle-Branch Block" Birnbaum et al, N Engl J Med
1996; 334:481-487

Be advised that in patients with

Left Bundle Branch Block Combined with Ventricular Hypertrophy,

The J Point elevation can exceed 0.5 mv (5mm) above the iso-electric line in patients without ACS.

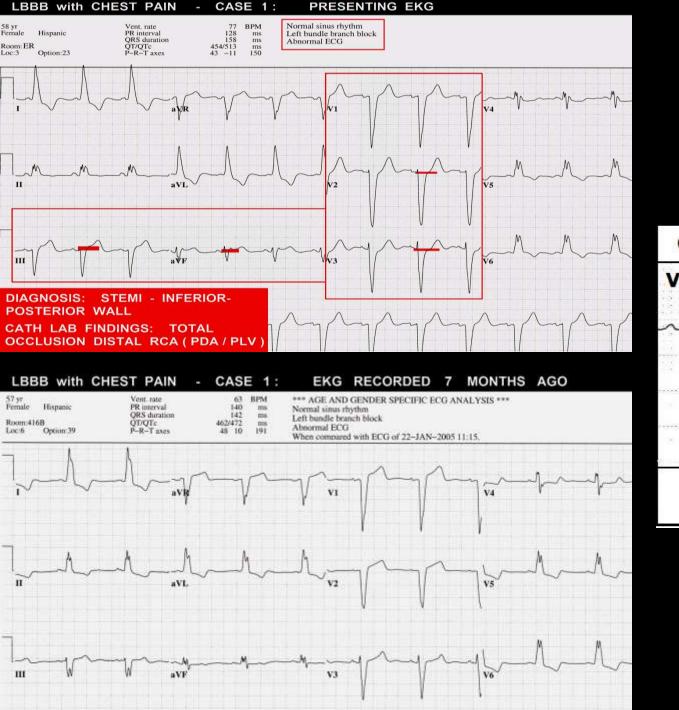


DIAGNOSIS: STEMI - INFERIOR-

POSTERIOR WALL

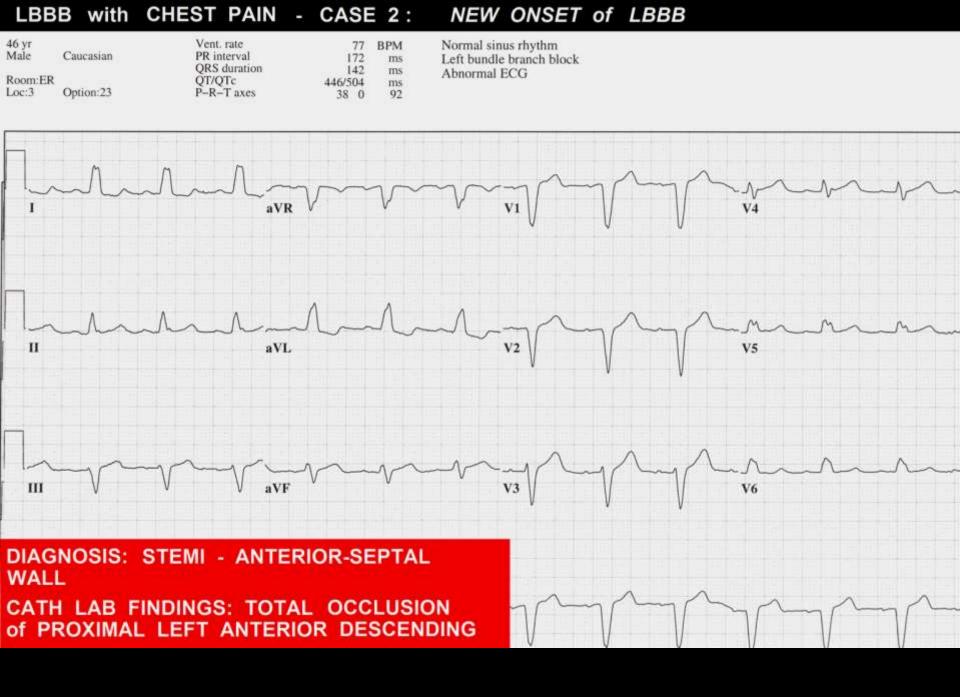
CATH LAB FINDINGS: TOTAL

OCCLUSION DISTAL RCA (PDA / PLV)





CHANGE



Evaluating the ECG for ACS:

STEP 1 - EVALUATE WIDTH OF QRS: WIDE (> 120 ms) NORMAL (< 120 ms) OF SUPRAVENTRICULAR ORIGIN --DETERMINE QRS MORPHOLOGY: LEFT BUNDLE BRANCH RIGHT BUNDLE BRANCH **BLOCK PATTERN BLOCK PATTERN EVALUATE FOR USE CAUTION --**EVALUATE FOR DO NOT RELY ON ST ELEVATION ST DEPRESSION ST ELEVATION ST DEPRESSION IN USUAL MANNER IN USUAL MANNER AS A MARKER OF ACS. -IS ROUTINELY SEEN IN WIDE QRS COMPLEX WIDE QRS COMPLEX RHYTHMS (both L and R RHYTHMS WITH LBBB **BBB PATTERNS) OFTEN** PATTERN. FOLLOW AHA CAUSE: DEPRESSION of CRITERIA (page 109) FOR J POINTS. ST SEGMENTS. DIAGNOSIS OF STEMI IN & INVERSION OF T WAVES. PRESENCE OF LBBB.

Evaluating the ECG for ACS:

Patients with Normal Width QRS (QRSd < 120ms)

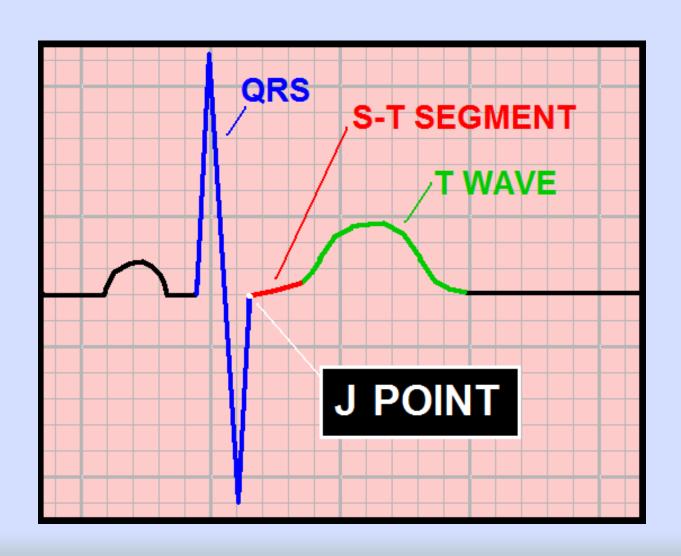
STEP 2 - EVALUATE the EKG for ACS

THE EKG MARKERS USED FOR DETERMINING THE PRESENCE OF ACUTE CORONARY SYNDROME INCLUDE:

- J POINTS
- ST SEGMENTS
- T WAVES

CAREFULLY SCRUTINIZE THESE MARKERS IN EVERY LEAD OF THE 12 LEAD EKG, TO DETERMINE IF THEY ARE NORMAL or ABNORMAL.

Defining NORMAL – QRS <120ms:



NORMAL ST - T WAVES

- WHEN QRS WIDTH IS NORMAL (< 120 ms)



ASSESS:

- J POINT: ISOELECTRIC (or < 1 mm dev.)

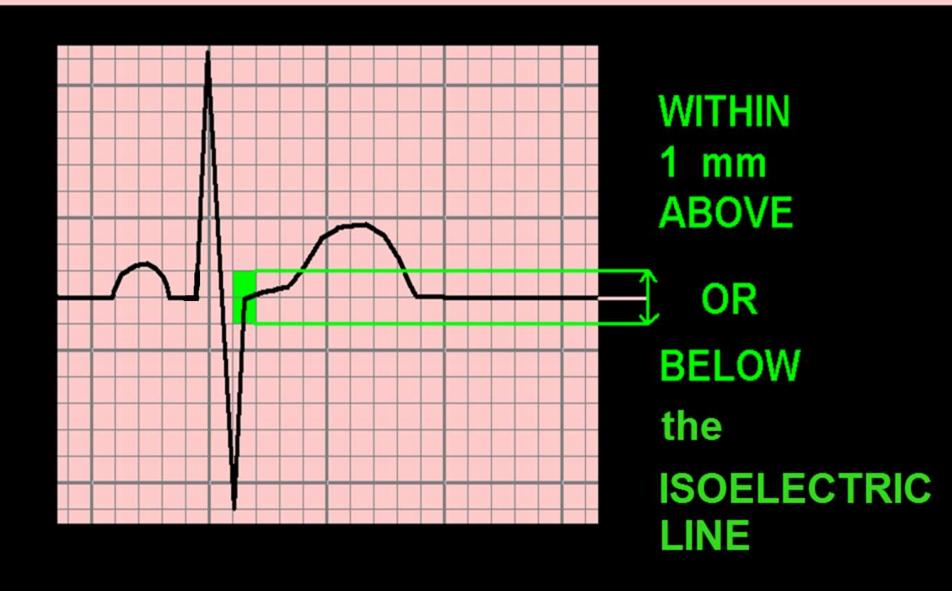
- ST SEG: SLIGHT, POSITIVE INCLINATION -

- T WAVE: UPRIGHT, POSITIVE -

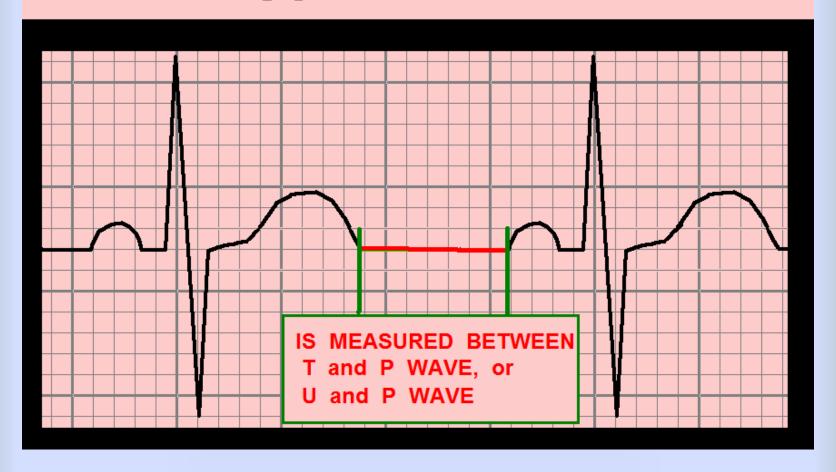


in EVERY LEAD EXCEPT aVR !!

THE J POINT SHOULD BE ...



THE ISOELECTRIC LINE

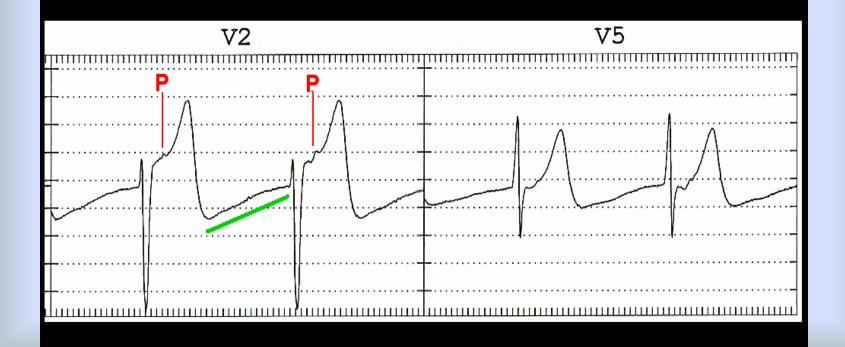


. . .the "flat line" between ECG complexes, when there is no detectable electrical activity . . .

The Isoelectric Line - it's not always isoelectric!

THE ISOELECTRIC LINE

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



THE P-Q JUNCTION

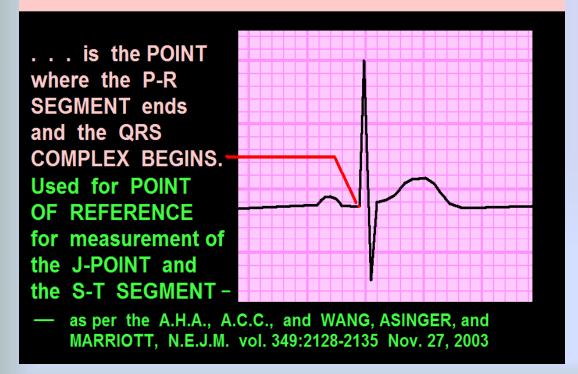
. . . is the POINT where the P-R SEGMENT ends and the QRS COMPLEX BEGINS. **Used for POINT** OF REFERENCE for measurement of the J-POINT and the S-T SEGMENT -



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

Use the P-Q junction as a reference point for measuring the J Point and ST-Segment when "iso-electric line is





not iso-electric!

Defining NORMAL:

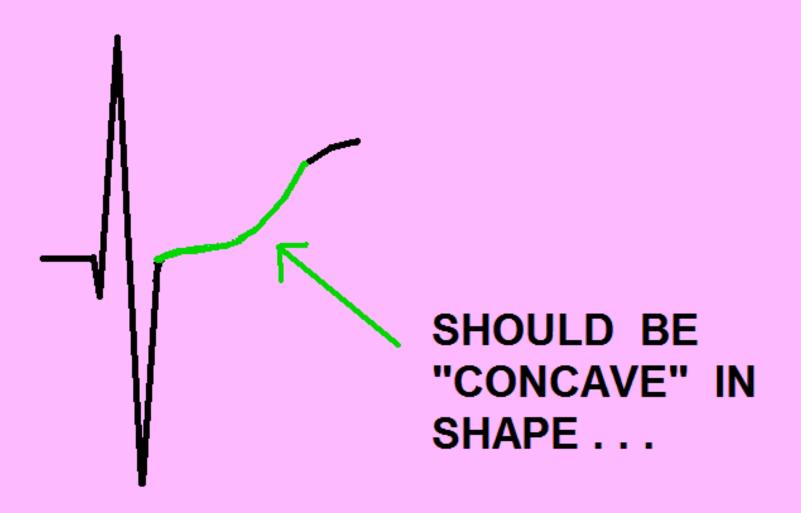
THE J POINT SHOULD BE ...



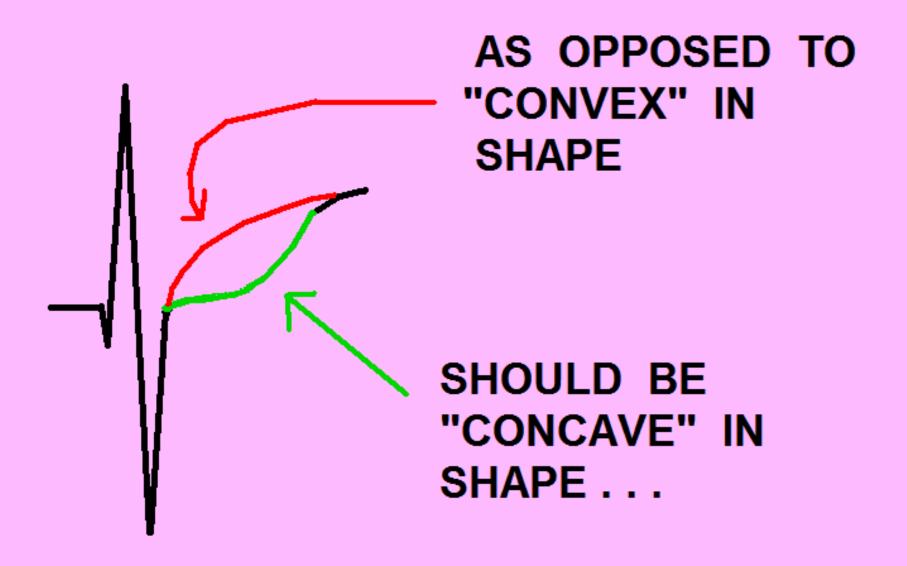
THE S-T SEGMENT

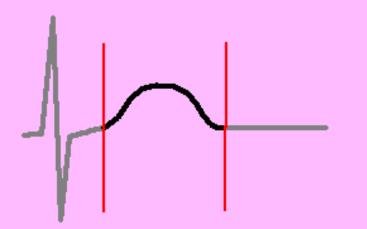


THE S-T SEGMENT



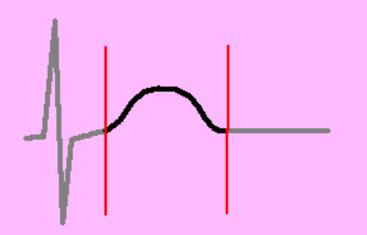
THE S-T SEGMENT





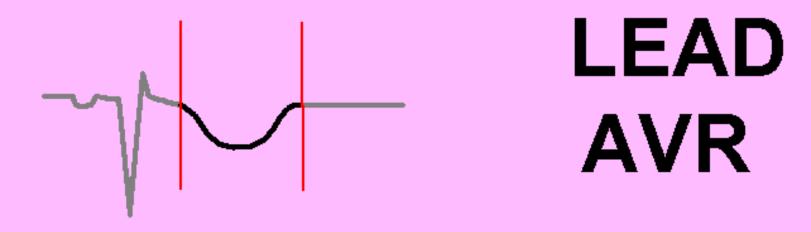
SHOULD BE
 A "NICE,"
 ROUNDED,
 CONVEX SHAPE

SHOULD BE SYMMETRICAL



SHOULD BE
 A "NICE,"
 ROUNDED,
 CONVEX SHAPE

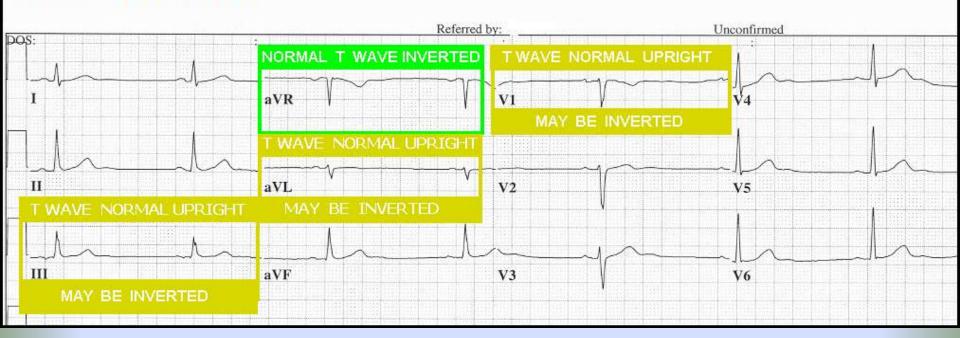
- SHOULD BE SYMMETRICAL
- SHOULD BE UPRIGHT IN ALL LEADS, EXCEPT AVR

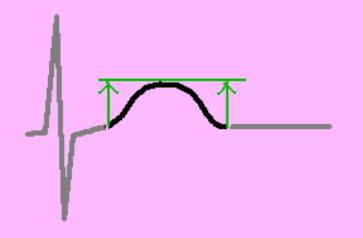


REMEMBER, IN LEAD AVR
 EVERYTHING
 IS
 "UPSIDE-DOWN"

Normal Variants: T Wave Inversion

Leads where the T WAVE may be INVERTED:





AMPLITUDE GUIDELINES:

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

NORMAL ST - T WAVES

- WHEN QRS WIDTH IS NORMAL (< 120 ms)



ASSESS:

- J POINT: ISOELECTRIC (or < 1 mm dev.)

- ST SEG: SLIGHT, POSITIVE INCLINATION -

- T WAVE: UPRIGHT, POSITIVE -



in EVERY LEAD EXCEPT aVR !!

ECG Indicators of ACS in Patients with Normal Width QRS Complexes (QRS duration < 120 ms)

Multiple patterns of ABNORMAL:

- J Point
- ST-Segment
- T Wave configurations may indicate ACS.

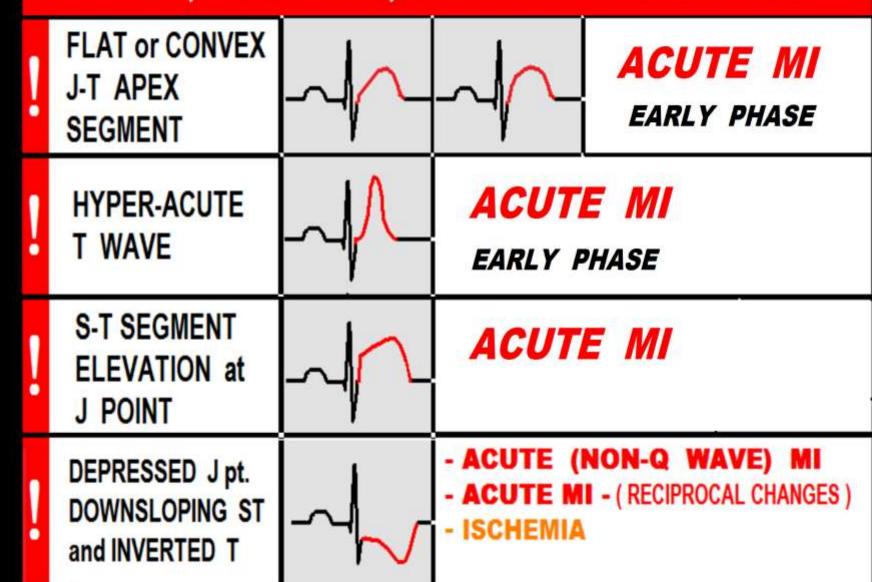
Remember, "IF IT'S NOT NORMAL, it's ABNORMAL!"

EKG PATTERNS Of ACS & ISCHEMIA S-T SEGMENT - ACUTE MI **ACUTE PERICARDITIS ELEVATION** at MYOCARDITIS J POINT EARLY REPOLARIZATION FLAT or CONVEX - ACUTE MI J-T APEX ISCHEMIA SEGMENT **HYPERKALEMIA** HYPER-ACUTE - TRANSMURAL ISCHEMIA ACUTE MI T WAVE HYPERTROPHY - ACUTE (NON-Q WAVE) MI DEPRESSED J pt. - ACUTE MI - (RECIPROCAL CHANGES) DOWNSLOPING ST - ISCHEMIA and INVERTED T - MYOCARDITIS INVERTED ELECTROLYTE IMBAL. T WAVE - ISCHEMIA - ACUTE MI (NOT COMMON) SHARP S-T - ISCHEMIA T ANGLE **BI-PHASIC** SUB-TOTAL LAD LESION T WAVE VASOSPASM HYPERTROPHY (WELLEN'S) DEPRESSED J - ISCHEMIA POINT with UPSLOPING ST - ISCHEMIA DOWNSLOPING S-T SEGMENT FLAT S-T - ISCHEMIA SEGMENT > 120 ms LOW VOLTAGE - ISCHEMIA T WAVE WITH NORMAL QRS U WAVE POLARITY - ISCHEMIA OPPOSITE THAT OF T WAVE

BOOK PAGE: 83

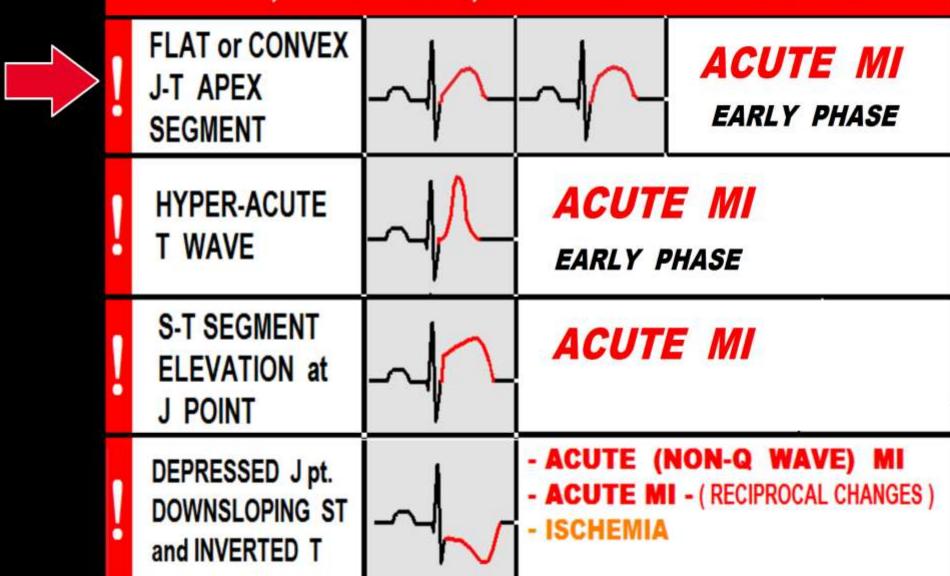
PATTERNS of ACS & ISCHEMIA

-- J POINT, ST SEGMENT, and T WAVE ABNORMALITIES --



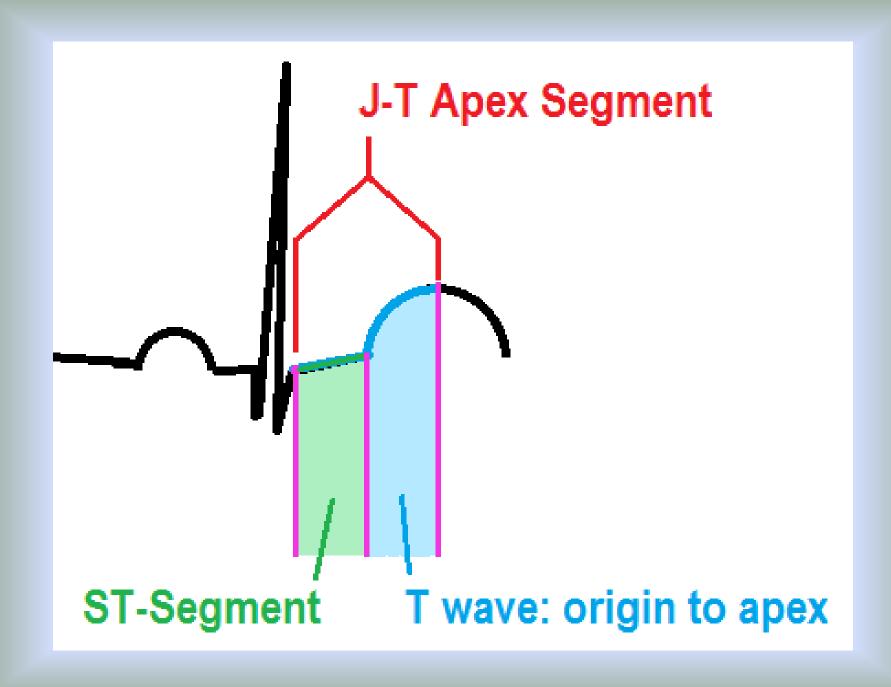
PATTERNS of ACS & ISCHEMIA

-- J POINT, ST SEGMENT, and T WAVE ABNORMALITIES --

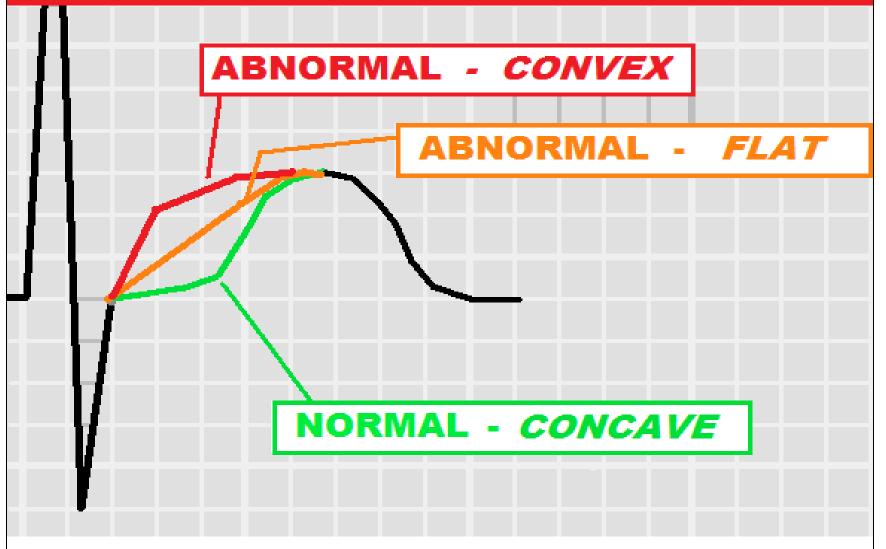


ECG Patterns associated with "EARLY PHASE MI:"

- J-T Apex abnormalities
- Hyper-Acute T Waves
- ST-T Wave Changes



J-T APEX SEGMENT VARIATIONS



PATTERNS of EARLY INFARCTION

-- FLAT and CONVEX J-T APEX SEGMENTS

WHEN EVALUATING for ST SEGMENT ELEVATION

From: AMERICAN HEART ASSOCIATION **ACLS 2005 REVISIONS**

During NORMAL STATES of PERFUSION, the J POINT is ISOELECTRIC and the ST SEGMENT has a

PTCA of the Left Anterior Descending artery (figure B)

CONCAVE appearance.

When measured 40 ms beyond the J POINT (noted by the RED DOT), the ST SEGMENT elevation is less than 1mm.

from a 54 year old male while resting (figure A), and during

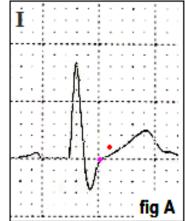


fig B

During a 20 second BALLOON OCCLUSION of the patient's LAD during routine PTCA, the ST segment

J POINT

"JPOINT plus 40 ms"

shows ST ELEVATION > 1 mm

INFARCTION -EARLY PHASE

NORMAL

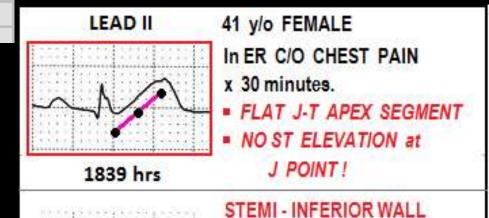
ST SEGMENT

PATTERN

assumes a CONVEX shape. When measured 40 ms beyond the J POINT, the ST segment is elevated > 1 mm. This phenonemon is seen routinely in the cath lab prior to the occurance of ST **ELEVATION at the J POINT** during PTCA and STENTING.

Both figures were recorded

J POINT END of ST SEGMENT T WAVE APEX FLAT J-T APEX SEGMENT CONSIDER EARLY PHASE of ACUTE MI



IS NOTED.

1850 hrs

11 MINUTES LATER, S-T

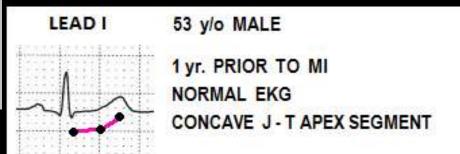
CATH LAB FINDINGS:

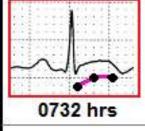
ELEVATION at the J POINT

TOTAL OCCLUSION of the

RIGHT CORONARY ARTERY

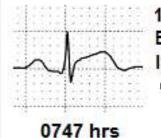
J POINT END of ST SEGMENT T WAVE APEX CONVEX J-T APEX SEGMENT CONSIDER EARLY PHASE of ACUTE MI!





STEMI LATERAL WALL • CONVEX J-T APEX SEGMENT

- MINIMAL ST ELEVATION
- at J POINT



- 15 MINUTES LATER, S-T ELEVATION at the J POINT IS NOTED.
- CATH LAB FINDINGS: TOTAL OCCLUSION OF CIRCUMFLEX ARTERY

CASE STUDY: ABNORMAL J-T APEX SEGMENTS

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

56 y/o MALE presents to ED with complaint of "INTERMITTENT SUBSTERNAL & SUB-EPIGASTRIC PRESSURE" x 3 HOURS. PMHx of ESOPHAGEAL REFLUX. NO other significant past medical history.

RISK FACTOR PROFILE:

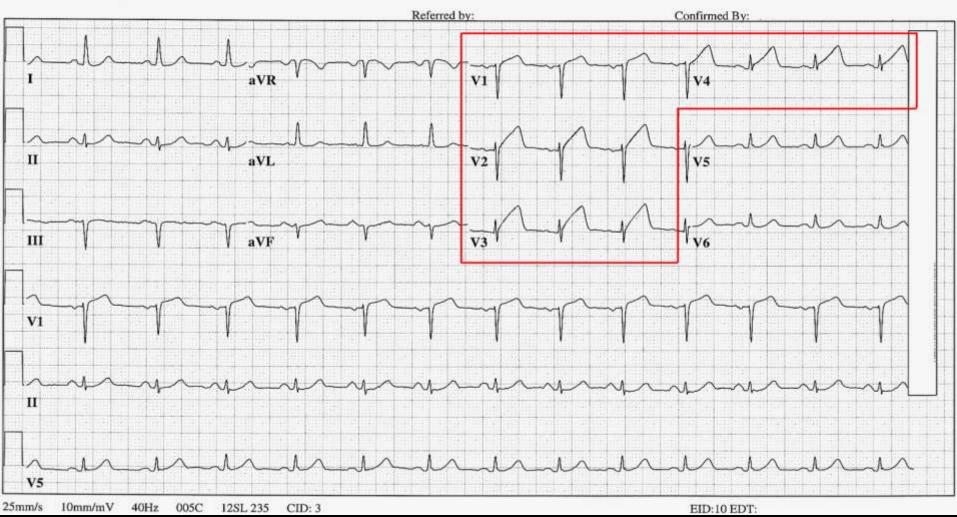
- FAMILY HISTORY father died of MI at age 62
- ☑ PREVIOUS CIGARETTE SMOKER quit 15 years ago.
- ✓ CHOLESTEROL DOES NOT KNOW; "never had it checked."
- ✓ OBESITY

PHYSICAL EXAM: Patient supine on exam table, mildly anxious, currently complaining of "mild indigestion," skin is warm, pale, dry; REST OF EXAM is UNREMARKABLE.

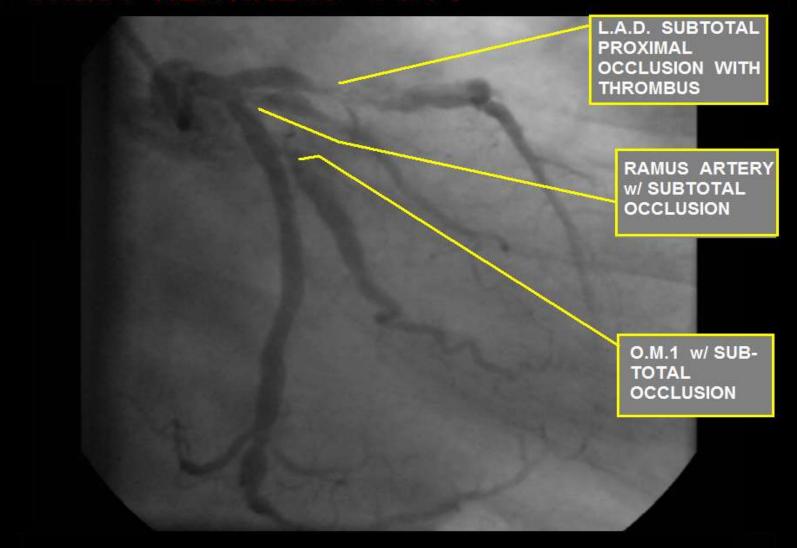
VITAL SIGNS: BP 142/94, P 80, R 20, SAO2 98%

LABS: JUST OBTAINED, RESULTS NOT AVAILABLE YET.

56 yr Vent. rate 80 BPM **UNEDITED COPY - REPORT IS COMPUTER GENERATED ONLY, WITHOUT Male Caucasian PR interval 154 PHYSICIAN INTERPRETATION QRS duration 78 ms Normal sinus rhythm Room: A9 QT/QTc 380/438 ms Normal ECG Loc:3 Option:23 P-R-T axes 51 -24 38 No previous ECGs available Technician: W Ruppert



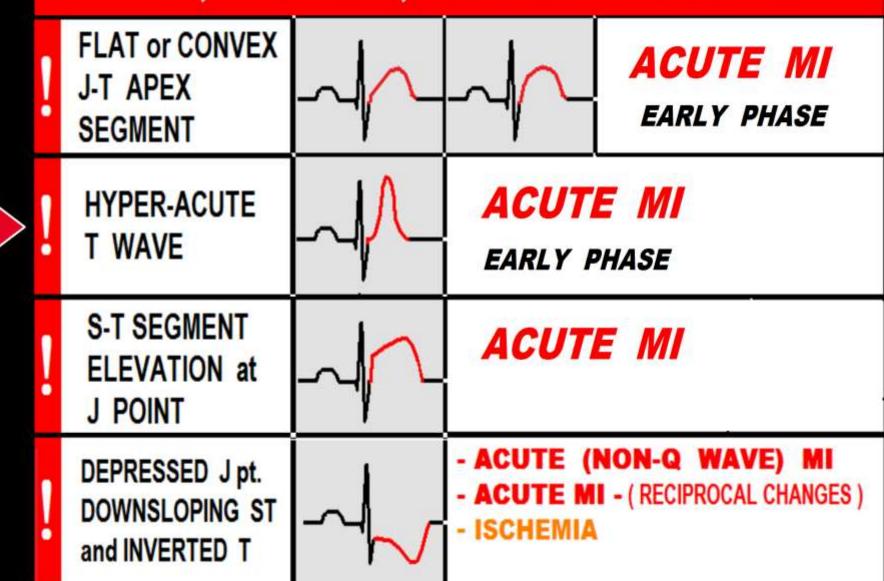
CASE STUDY: 56 y/o male with INTERMITTENT "CHEST HEAVINESS"



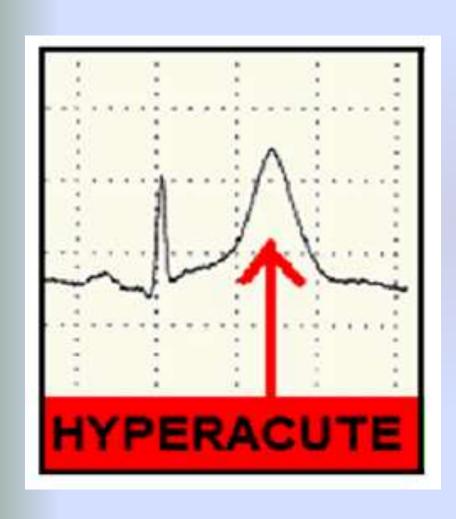
TREATMENT PLAN: EMERGENCY CORONARY ARTERY BYPASS SURGERY (4 VESSEL)

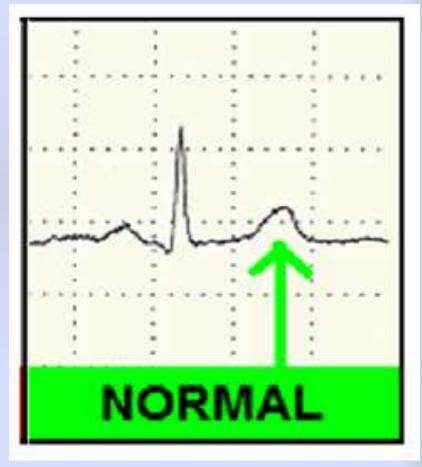
PATTERNS of ACS & ISCHEMIA

-- J POINT, ST SEGMENT, and T WAVE ABNORMALITIES --

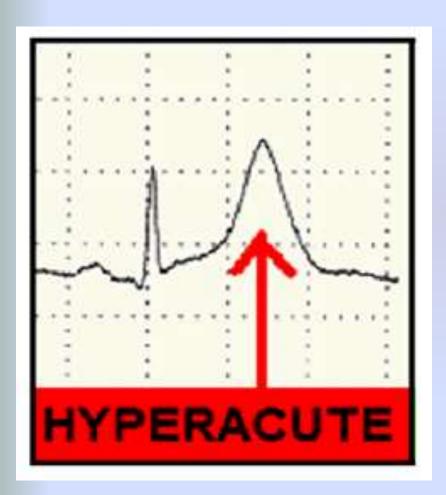


T waves should not be HYPERACUTE



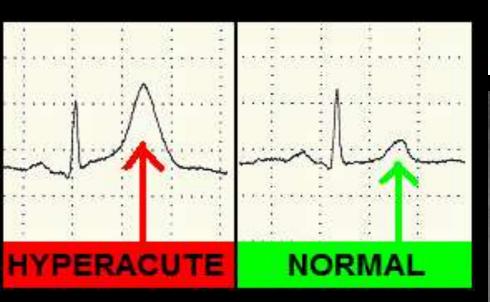


HYPERACUTE T Waves may indicate:



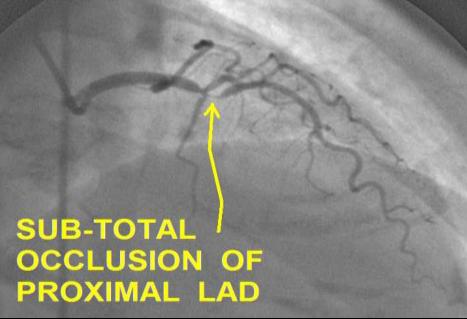
- Early phase Acute MI
- Transmural ischemia (usually seen in one region of the ECG)
- Hyperkalemia (seen globally across ECG)
- Hypertrophy

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



Helpful Clue: Hyper-Acute T Waves

 GLOBAL Hyper-acute T Waves (in leads viewing multiple myocardial regions / arterial distributions) favors HYPERKALEMIA 55years Female Caucasian

Room:

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

Sinus bradyc Possible Left atrial enlargement Borderline ECG

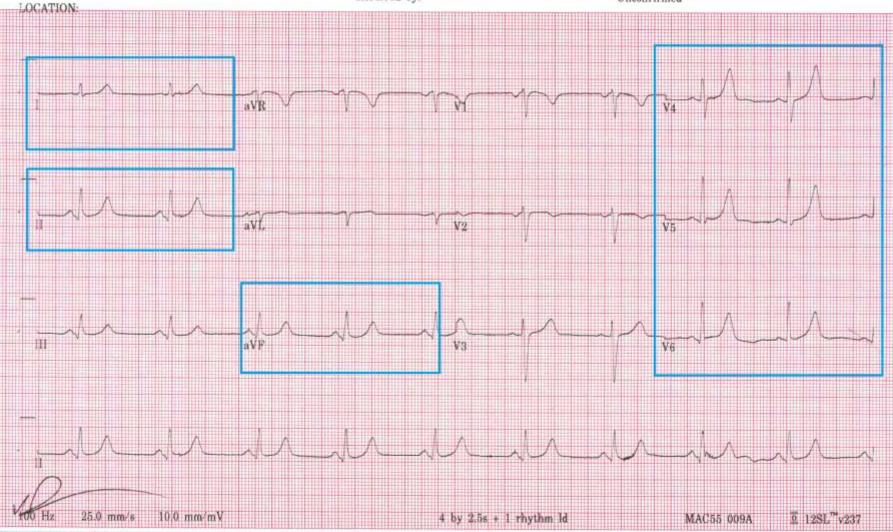
ER ATTENDING REVIEW

K + = 6.7

Technician: Test ind:



Unconfirmed



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

CASE STUDY: HYPERACUTE T WAVES

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

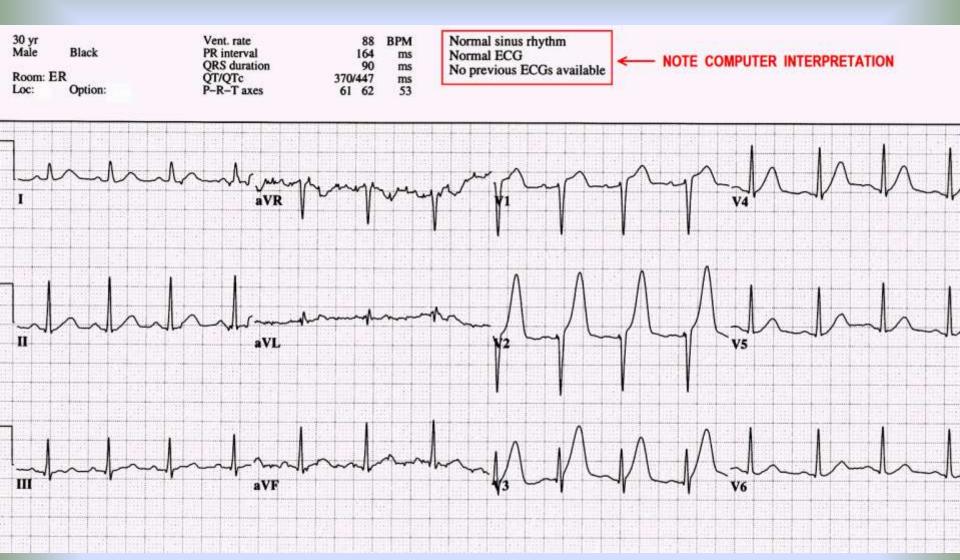
30 y/o male presents to ER via EMS, c/o sudden onset of dull chest pain x 40 min. Pain level varies, not effected by position, movement or deep inspiration. No associated symptoms.

RISK FACTOR PROFILE: NONE. CHOLESTEROL UNKNOWN.

PHYSICAL EXAM: Patient is supine on exam table, CAO x 4, anxious, restless, skin pale, cool, dry. Patient c/o chest pressure, "7" on 1 - 10 scale, uneffected by position, movement, deep inspiration. Lungs clear. HS: NL S1, S2, no rubs, murmurs, gallops

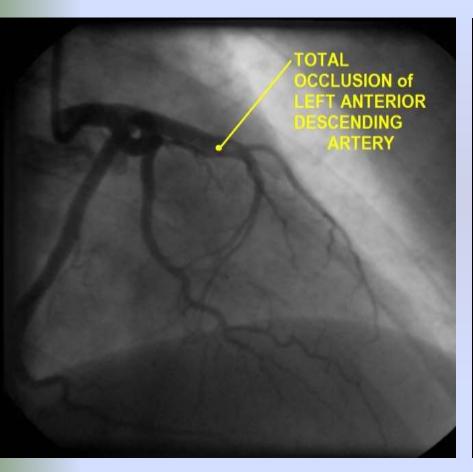
VITAL SIGNS: BP 136/88 P 90 R 20 SAO2 98%

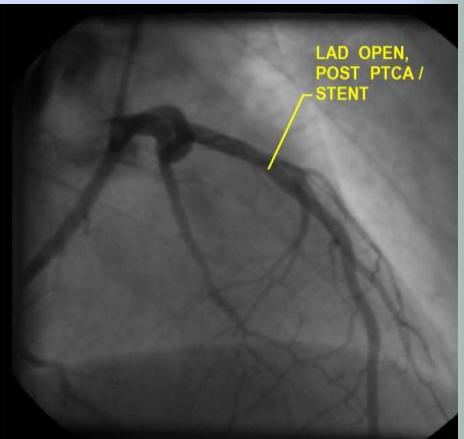
DIAGNOSTIC TESTING: 1st TROPONIN I - ultra: <0.07



CORONARY ARTERIAL DISTRIBUTIONS: 30 yr Male Vent. rate **BPM** Normal sinus rhythm 88 Black PR interval 164 Normal ECG ms V1 - V4 = LEFT ANTERIOR DESCENDING (LAD) QRS duration 90 ms No previous ECGs available I, AVL = DIAGONAL (DIAG) off the LAD or OBTUSE MARGINAL (OM) off CIRCUMFLEX (CX) Room: ER QT/QTc 370/447 ms P-R-T axes HIGHLIGHTED AREAS = Loc: Option: 53 61 62 V5, V6 = CIRCUMFLEX HYPERACUTE T WAVES II, III, AVF = RIGHT CORONARY ARTERY or CX LATERAL - ANTERIOR WALL ANTERIOR - SEPTAL WALL ANTERIOR WALL aVR INFERIOR WALL LATERAL - ANTERIOR WALL ANTERIOR - SEPTAL WALL LATERAL WALL aVL INFERIOR WALL INFERIOR WALL ANTERIOR WALL LATERAL WALL

Cath Lab findings:





Dynamic ST-T Wave Changes:

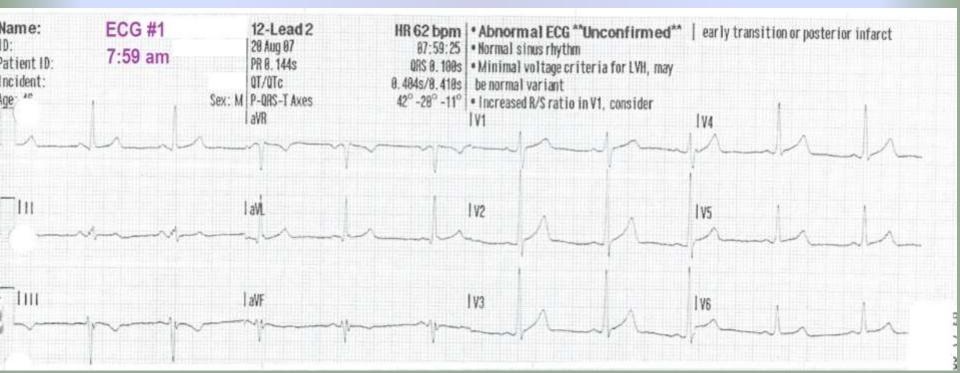
 Other than HEART RATE related variations (which affect intervals), J Points, ST-Segments and T Waves SHOULD NOT CHANGE.

Dynamic ST-T Wave Changes:

- Other than HEART RATE related variations (which affect intervals), J Points, ST-Segments and T Waves SHOULD NOT CHANGE.
- When changes to J Points, ST-Segments and/or T waves are NOTED, consider EVOLVING MYOCARDIAL ISCHEMIA and/or EARLY PHASE MI, until proven otherwise.

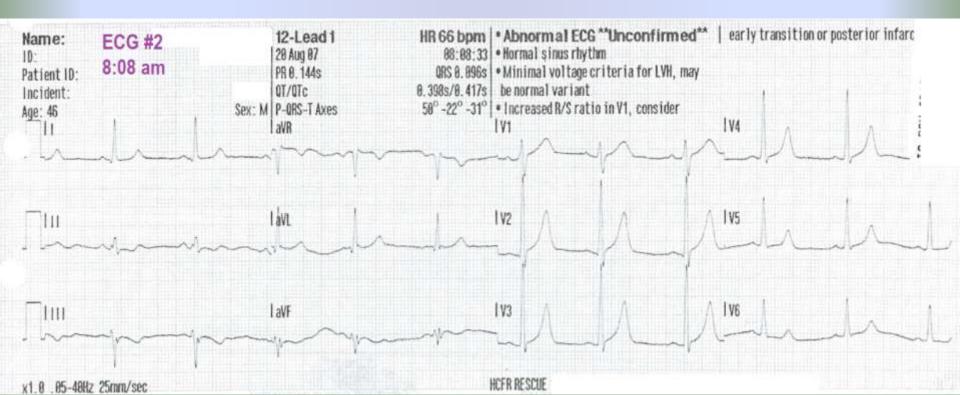
46 year old male

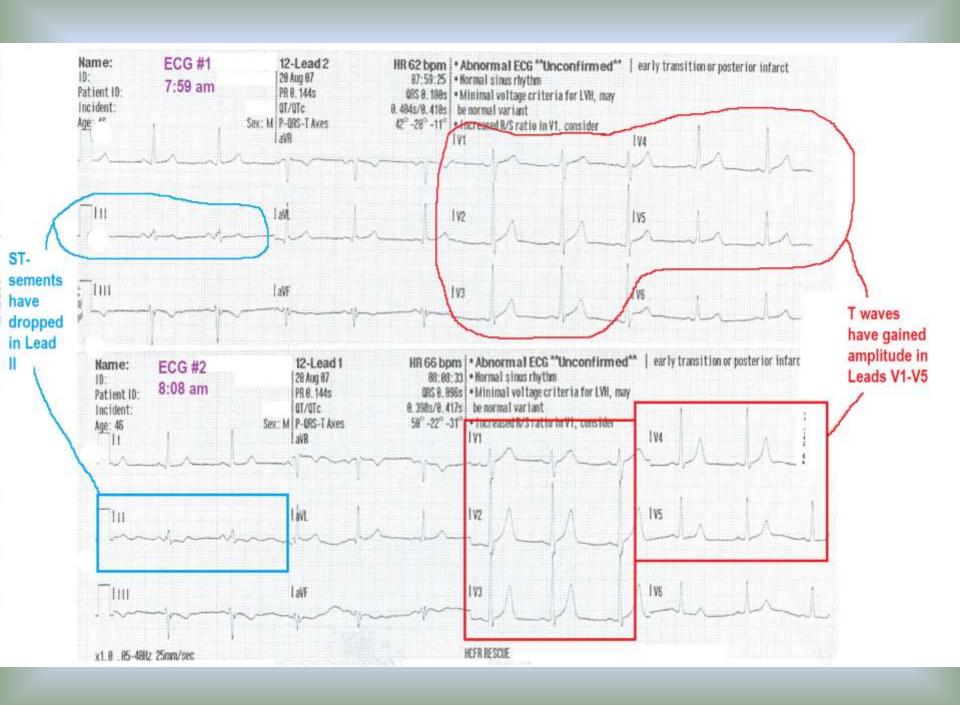
- Exertional dyspnea X "several weeks"
- Intermittent chest pressure X last 3 hours.
 Currently pain free.

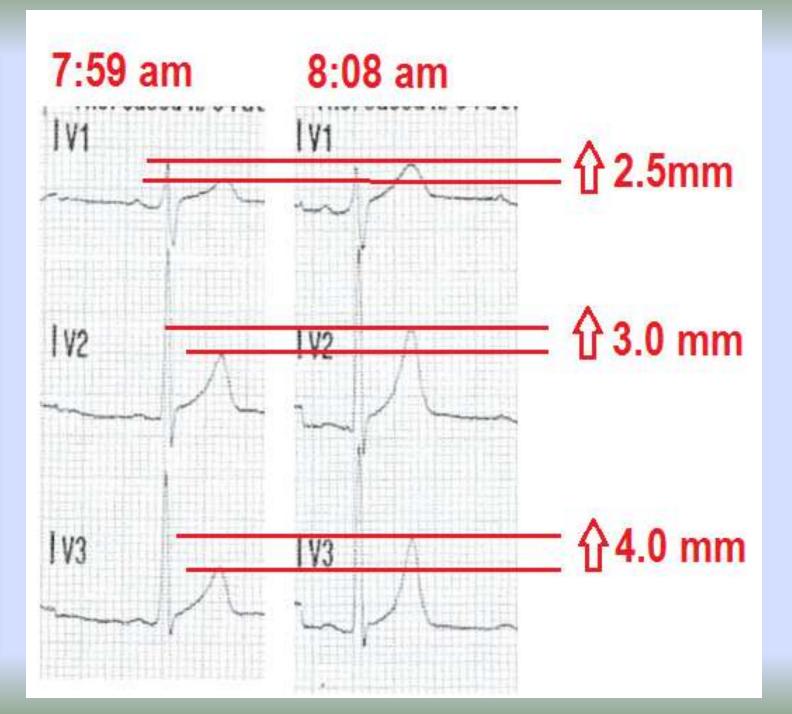


46 year old male: ECG 1

 Chest pressure has returned, "5" on 1-10 scale. 2nd ECG obtained due to "change in symptoms":







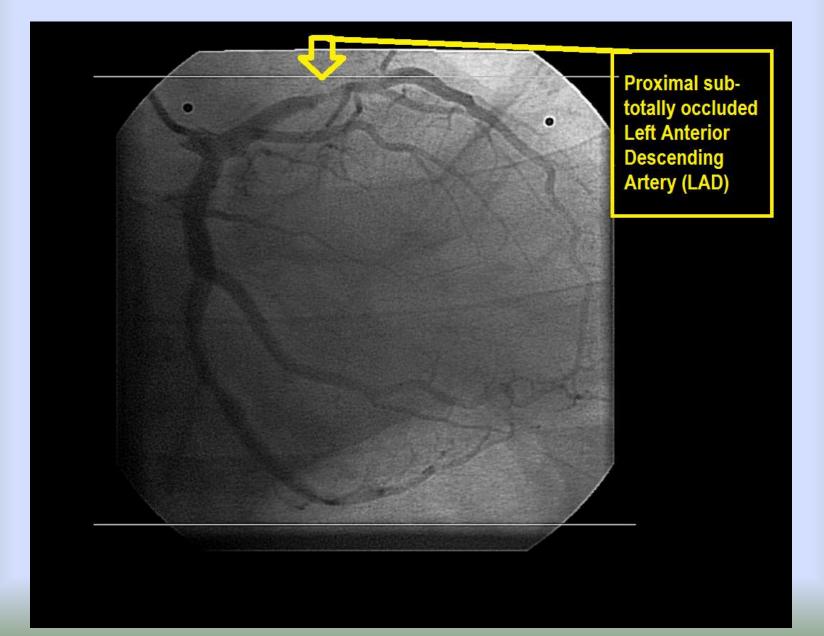
ST-Segment Depression

7:59 am

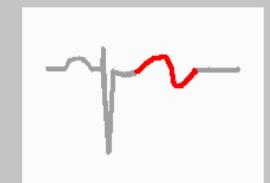
8:08 am



Cath Lab Angiography:



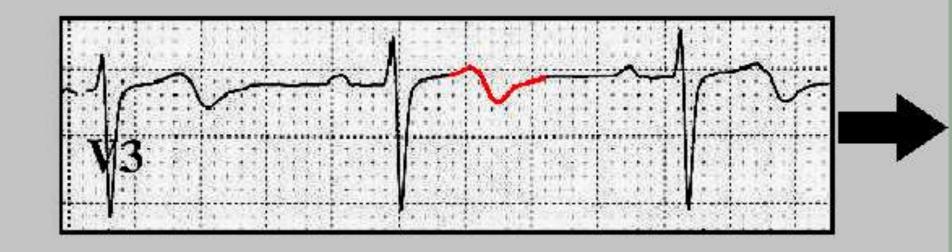
ISCHEMIA



BI-PHASIC T WAVE

- SUB-TOTAL OCCLUSION of LEFT ANTERIOR DESCENDING ARTERY (when noted in V1-V4)
- LEFT VENTRICULAR HYPERTROPHY
- COCAINE INDUCED VASOSPASM

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

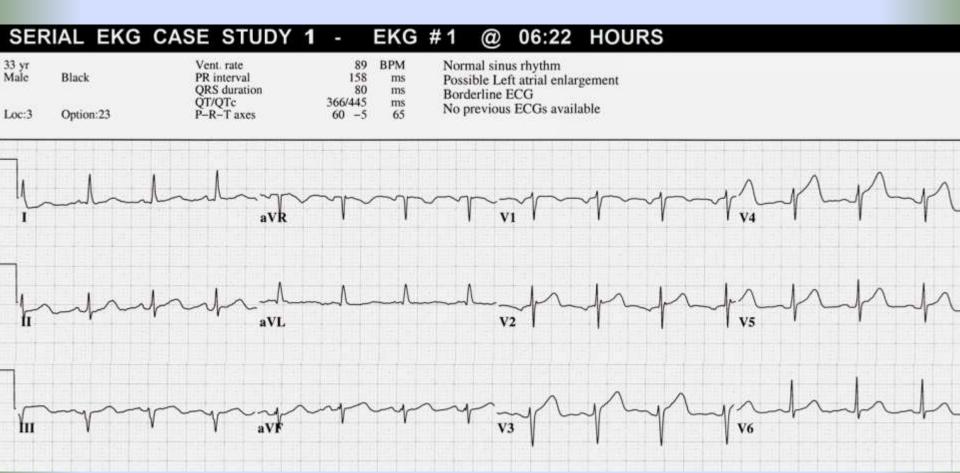
Wellen's Syndrome EPIDEMIOLOGY & PROGNOSIS:

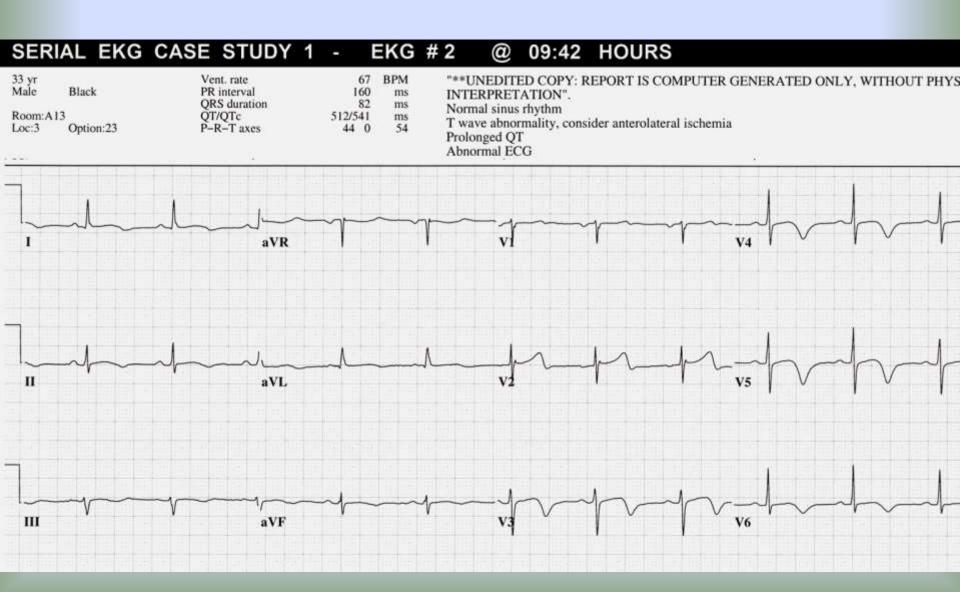
- Present in 14-18% of patients admitted with unstable angina
- 75% patients not treated developed extensive Anterior MI within 3 weeks.
- Median Average time from presentation to Acute Myocardial Infarction – 8 days

Sources: <u>H Wellens et. Al, Am Heart J 1982;</u>

v103(4) 730-736

- 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.





DYNAMIC ST-T Wave Changes ARE PRESENT!!

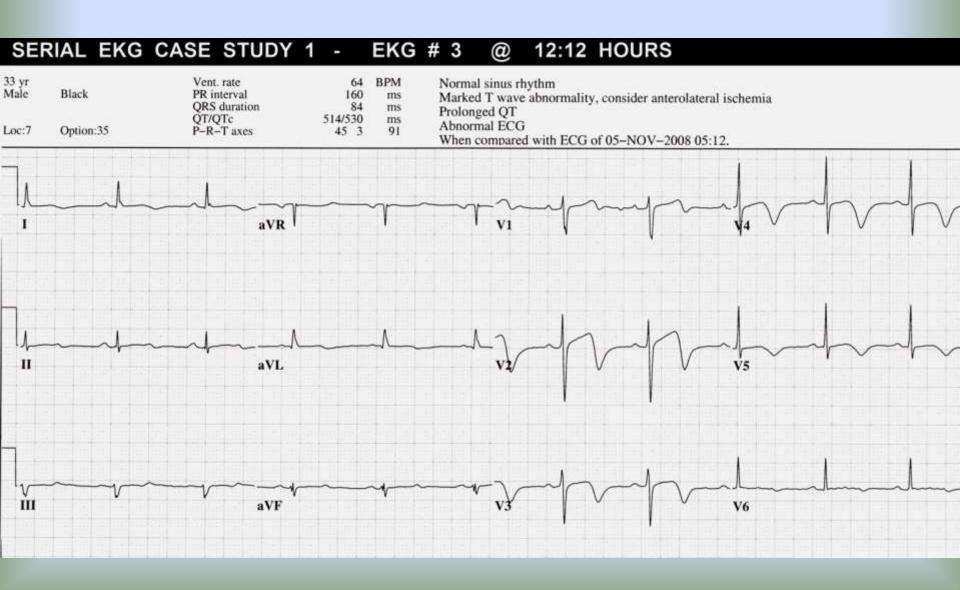
NOW

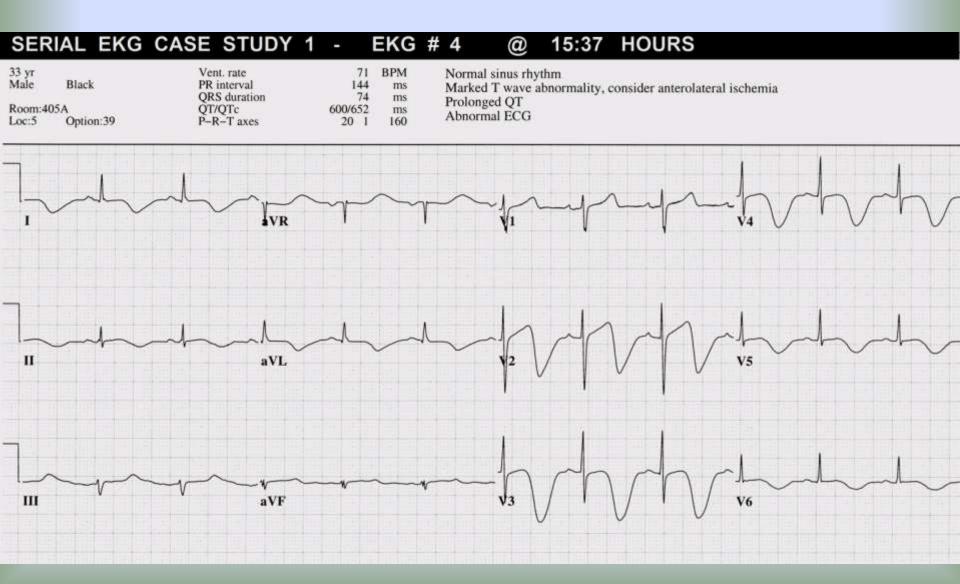
is the time for the

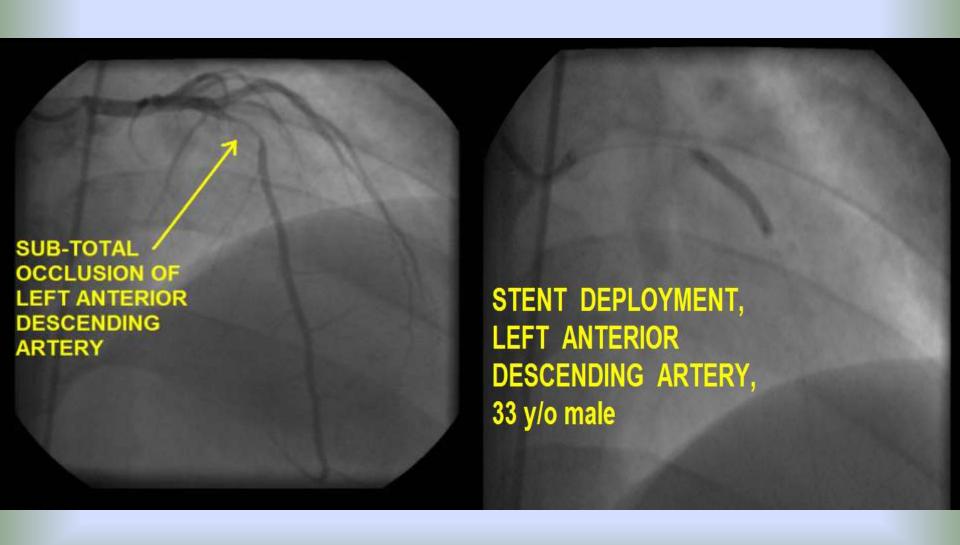
STAT CALL

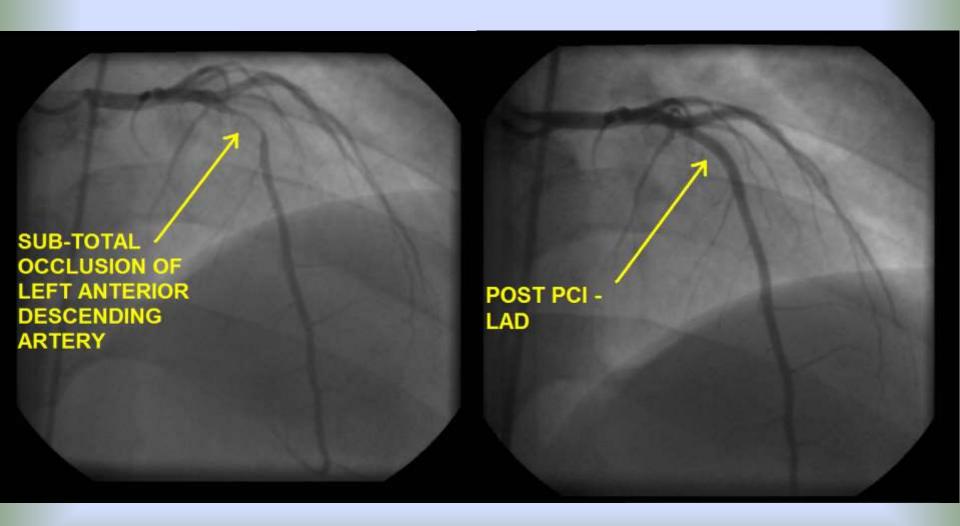
to the

CARDIOLOGIST !!!!







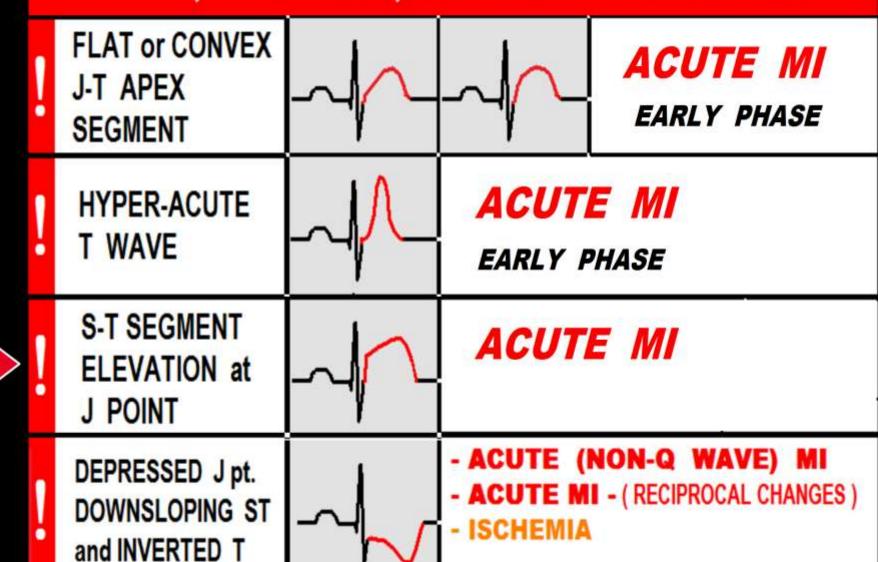


Additional Resources:

Wellen's Syndrome, NEJM case study

PATTERNS of ACS & ISCHEMIA

-- J POINT, ST SEGMENT, and T WAVE ABNORMALITIES --



ECG CRITERIA for DIAGNOSIS of STEMI: (ST ELEVATION @ J POINT)

*LEADS V2 and V3:

MALES AGE 40 and up ----- 2.0 mm

(MALES LESS THAN 40----- 2.5 mm)

FEMALES ----- 1.5 mm

ALL OTHER LEADS: 1.0 mm or more,

in TWO or more

CONTIGUOUS LEADS

* P. Rautaharju et al, "Standardization and Interpretation of the ECG," JACC 2009;(53)No.11:982-991

STEMI Criteria for 18 Lead ECGs:

Right-Sided Chest Leads (V3R - V6R): <u>0.5</u> mm

Posterior Chest Leads (V7 – V9): <u>0.5</u> mm

* P. Rautaharju et al, "Standardization and Interpretation of the ECG," JACC 2009;(53)No.11:982-991

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

Recommendations

- 1. For men 40 years of age and older, the threshold value for abnormal J-point elevation should be 0.2 mV (2 mm) in leads V₂ and V₃ and 0.1 mV (1 mm) in all other leads.
- 2. For men less than 40 years of age, the threshold values for abnormal J-point elevation in leads V₂ and V₃ should be 0.25 mV (2.5 mm).
- 3. For women, the threshold value for abnormal J-point elevation should be 0.15 mV (1.5 mm) in leads V_2 and V_3 and greater than 0.1 mV (1 mm) in all other leads.
- 4. For men and women, the threshold for abnormal J-point elevation in V₃R and V₄R should be 0.05 mV (0.5 mm), except for males less than 30 years of age, for whom 0.1 mV (1 mm) is more appropriate.
- 5. For men and women, the threshold value for abnormal J-point elevation in V₇ through V₉ should be 0.05 mV (0.5 mm).
- 6. For men and women of all ages, the threshold value for abnormal J-point depression should be -0.05 mV (-0.5 mm) in leads V_2 and V_3 and -0.1 mV (-1 mm) in all other leads.

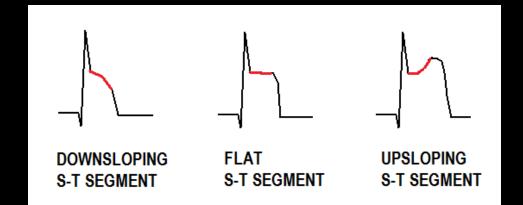
ST SEGMENT ELEVATION:

S-T SEGMENTS ELEVATE WITHIN SECONDS OF CORONARY ARTERY OCCLUSION:



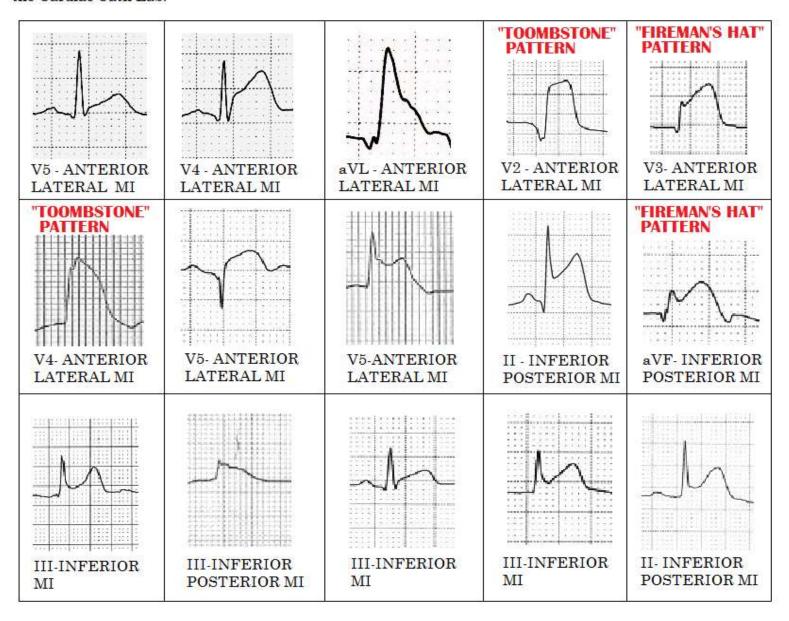
IN THIS CASE, a normal response to balloon occlusion of the RIGHT CORONARY ARTERY during PTCA in the CARDIAC CATH LAB

3 COMMON PATTERNS of ST SEGMENT ELEVATION From ACUTE MI:



ST SEGMENT ELEVATION in ACUTE MI:

The following samples are from patients with ACUTE MI, as confirmed by discovery of total arterial occlusion in the Cardiac Cath Lab:



Reciprocal S-T Segment Depression *may* or *may not* be present during STEMI.

Reciprocal S-T Segment Depression may or may not be present during STEMI.

The presence of S-T Depression on an EKG which exhibits significant S-T elevation is a fairly reliable indicator that STEMI is the diagnosis.

Reciprocal S-T Segment Depression may or may not be present during STEMI.

The presence of S-T Depression on an EKG which exhibits significant S-T elevation is a fairly reliable indicator that STEMI is the diagnosis.

However the *lack of Reciprocal S-T Depression* DOES NOT rule out STEMI.

ACUTE MI

COMPLICATIONS TO ANTICIPATE FOR ALL MI PATIENTS:

- **LETHAL DYSRHYTHMIAS**
- **CARDIAC ARREST**
- FAILURE OF STRUCTURE(S)
 SERVED BY THE BLOCKED ARTERY





"NOWHERE", NEW MEXICO, 1994



 Correlation of ECG Leads with Coronary Arterial Anatomy and the STRUCTURES SERVED by the OCCLUDED ARTERY



Correlation of ECG Leads with Coronary
 Arterial Anatomy and the STRUCTURES
 SERVED by the OCCLUDED ARTERY
 Will serve as a "crystal ball," allowing
 you to ANTICIPATE complications of STEMI



Correlation of ECG Leads with Coronary
 Arterial Anatomy and the STRUCTURES
 SERVED by the OCCLUDED ARTERY
 Will serve as a "crystal ball," allowing
 you to ANTICIPATE complications of STEMI
 BEFORE they occur !!



"Having knowledge of common coronary artery anatomy is the

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

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

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!

STEMI Case Studies, excerpts from "12 Lead ECG Interpretation in ACS with Case Studies from the Cardiac Cath Lab."

CASE STUDY 1 - STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

72 y/o male, c/o CHEST "HEAVINESS," started 20 minutes before calling 911. Pain is "8" on 1-10 scale, also c/o mild shortness of breath. Has had same pain "intermittently" x 2 weeks.

RISK FACTOR PROFILE:

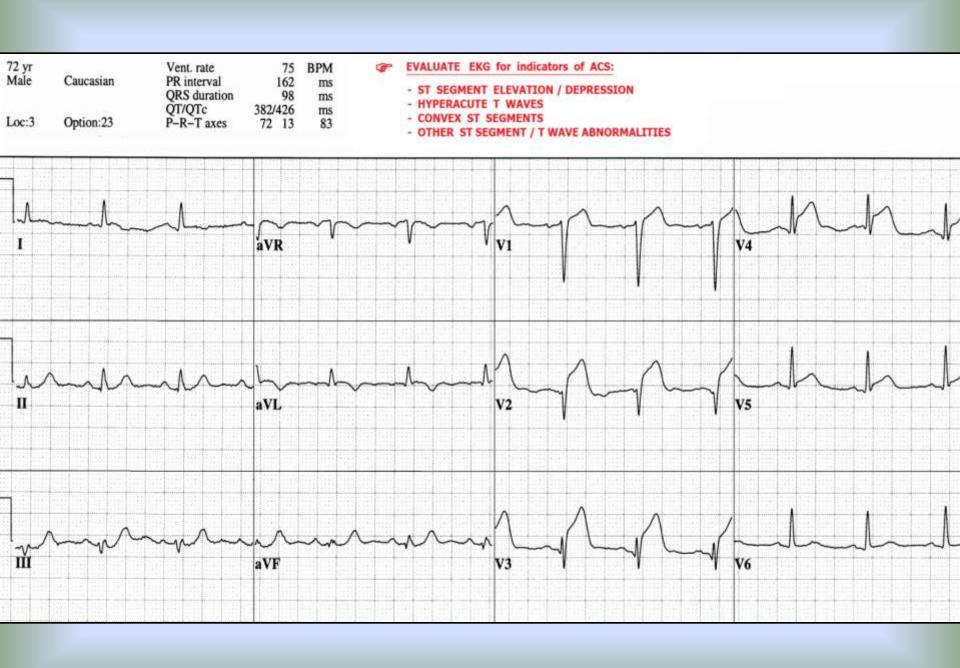
- FAMILY HISTORY father died of MI at age 77
- FORMER CIGARETTE SMOKER smoked for 30 year quit 27 years ago
- DIABETES oral meds and diet controlled
- HIGH CHOLESTEROL controlled with STATIN meds
- AGE: OVER 65

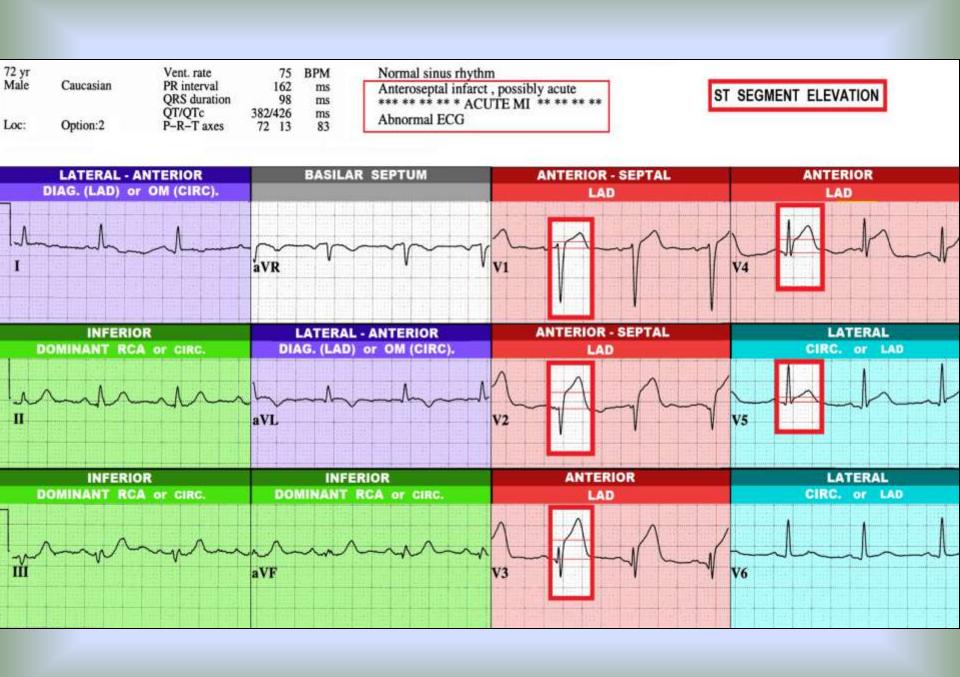
PHYSICAL EXAM: Patient calm, alert, oriented X 4, skin cool, dry, pale.

No JVD, Lungs clear bilaterally. Heart sounds normal S1, S2. No peripheral edema.

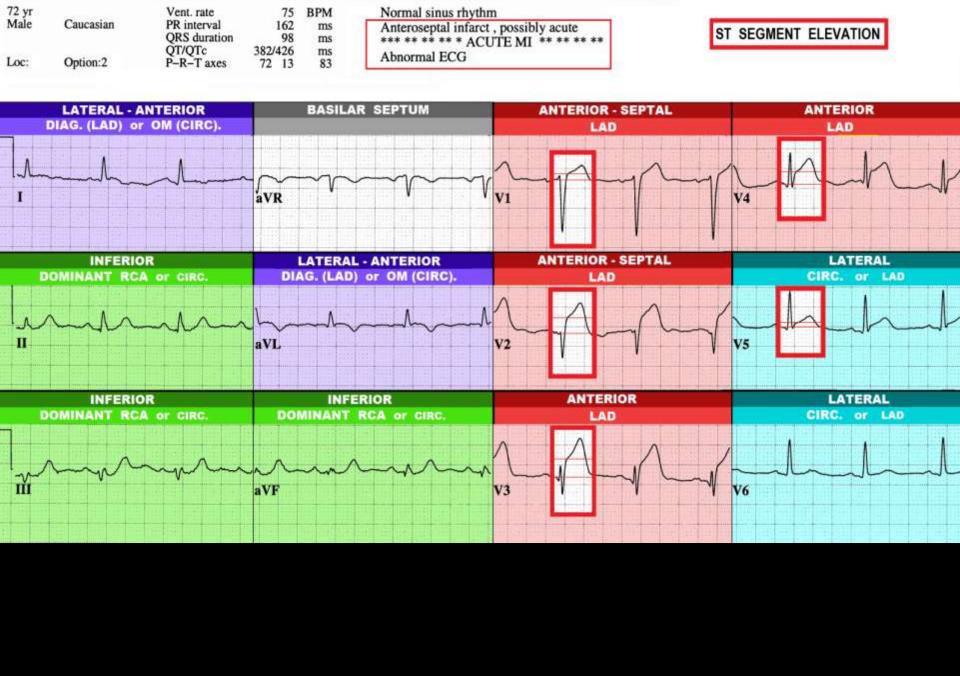
VITAL SIGNS: BP: 100/64, P: 75, R: 20, SAO2: 94%

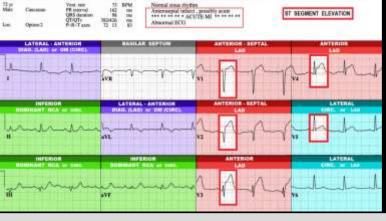
LABS: FIRST TROPONIN: 6.4



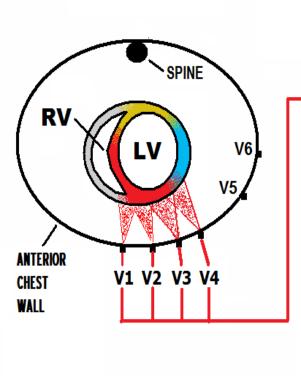


Note: There is NO Reciprocal ST Depression on this STEMI ECG!

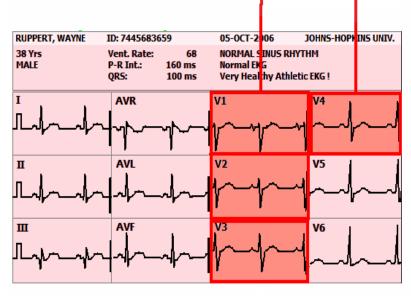




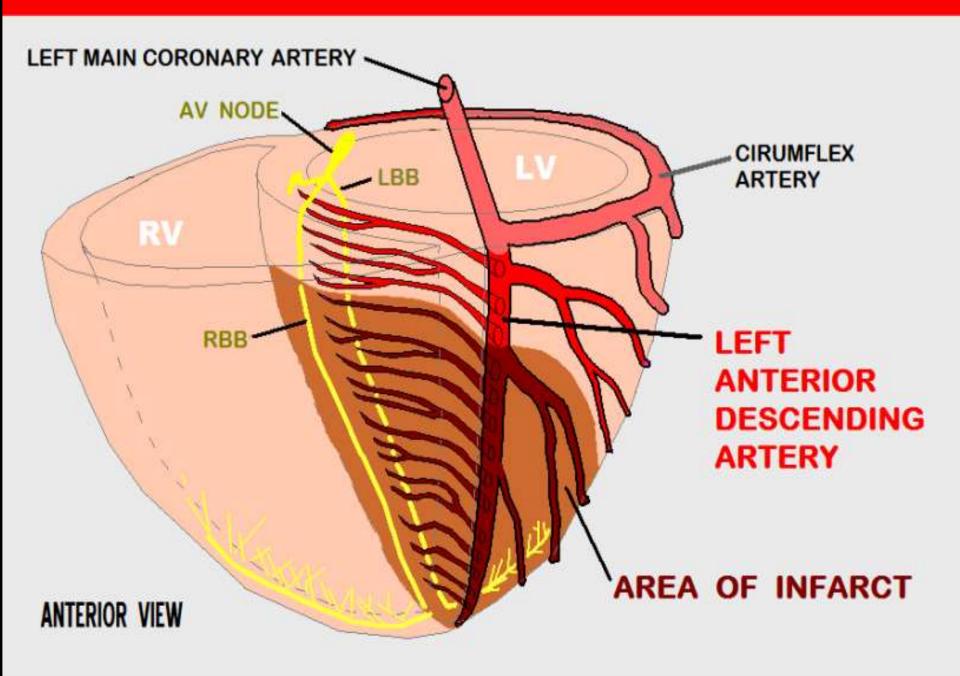
V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL of the LEFT VENTRICLE

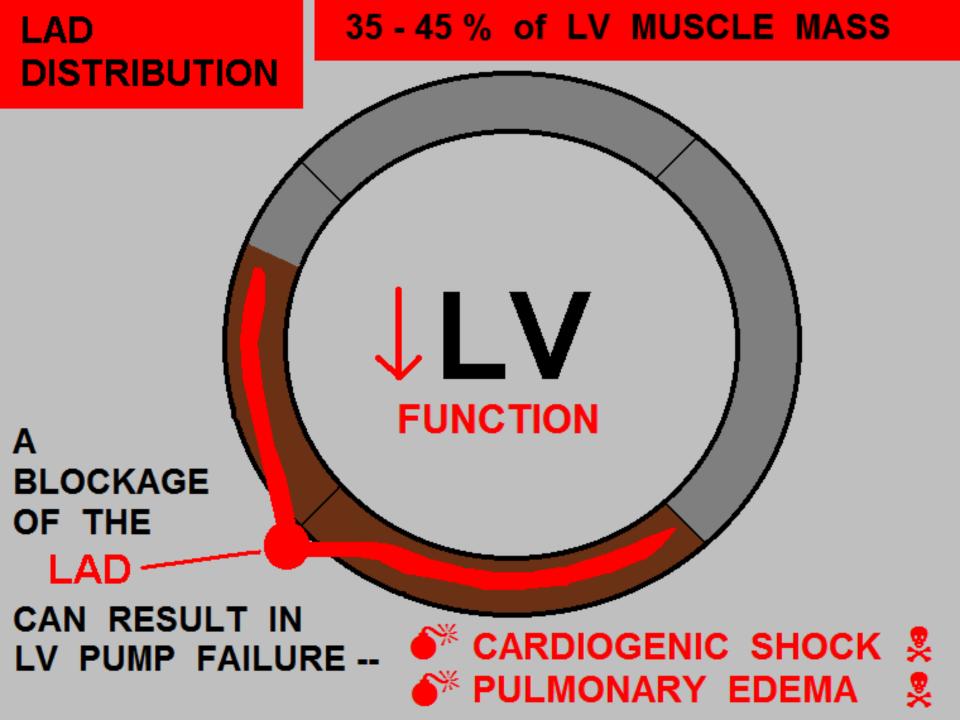


V1, V2 - ANTERIOR / SEPTAL V3, V4 - ANTERIOR



OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY







LEFT ANTERIOR DESCENDING ARTERY (LAD)

- ANTERIOR WALL OF LEFT VENTRICLE
- 35 45 % OF LEFT VENTRICLE MUSCLE MASS
 - SEPTUM, ANTERIOR 2/3
- **BUNDLE BRANCHES**
 - ANTERIOR-MEDIAL PAPILLARY MUSCLE

ANTICIPATED COMPLICATIONS of ANTERIOR-SEPTAL WALL STEMI & POSSIBLE INDICATED INTERVENTIONS: - CARDIAC ARREST - CARDIAC DYSRHYTHMIAS (VT / VF) ACLS (antiarrhythmics)

- INTRA-AORTIC BALLOON PUMP

(use caution with fluid challenges

(use caution with dieuretics due to

pump failure and hypotension)

due to PULMONARY EDEMA)

- CPAP

- ET INTUBATION

TRANSCUTANEOUS or

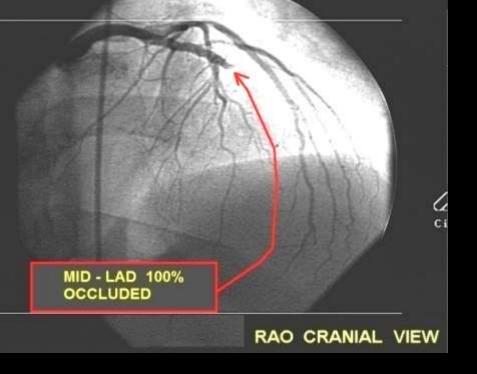
TRANSVENOUS PACING

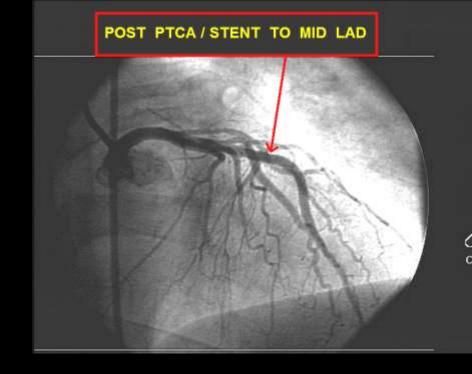
ACLS (antiarrhythmics)
INOTROPE THERAPY:
-DOPAMINE / DOBUTAMINE /
LEVOPHED

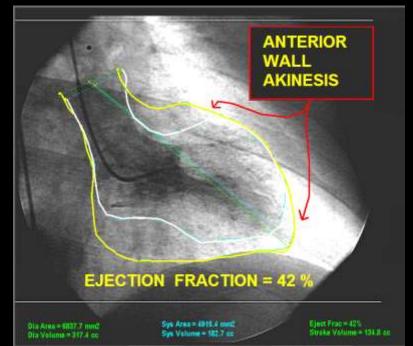
- PULMONARY EDEMA

- 3rd DEGREE HEART BLOCK - NOT

RESPONSIVE TO ATROPINE







CASE STUDY 2: STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

46 y/o Female walks into ED TRIAGE, with chief complaint of EPIGASTRIC PAIN, NAUSEA and WEAKNESS. Symptoms have been intermittent for last two days. She was awakened early this morning with the above symptoms, which are now PERSISTENT.

RISK FACTOR PROFILE:

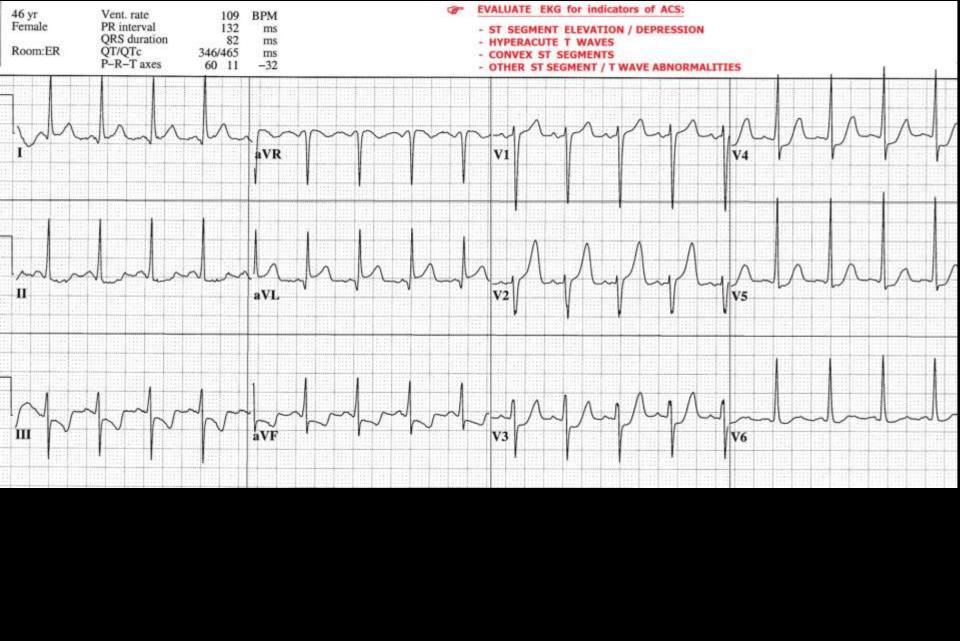
- FAMILY HISTORY father died of CAD, older brother had CABG, age 39
- DIABETES diet controlled
- HYPERTENSION

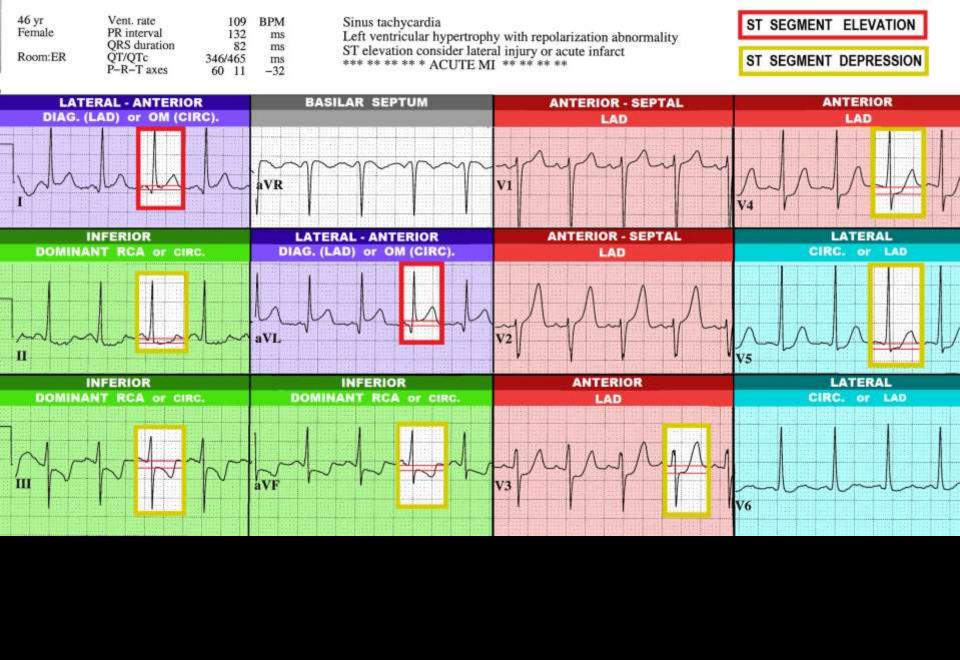
PHYSICAL EXAM: Pt. CAOx4, anxious, SKIN cold, clammy, diaphoretic. No JVD.

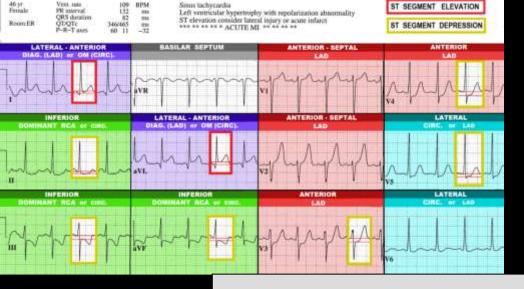
Lungs: clear, bilaterally. Heart Sounds: Normal S1, S2.

VITAL SIGNS: BP: 168/98, P: 110, R: 24, SAO2: 97% on O2 4 LPM via nasal canula

LABS: TROPONIN ultra = 2.8

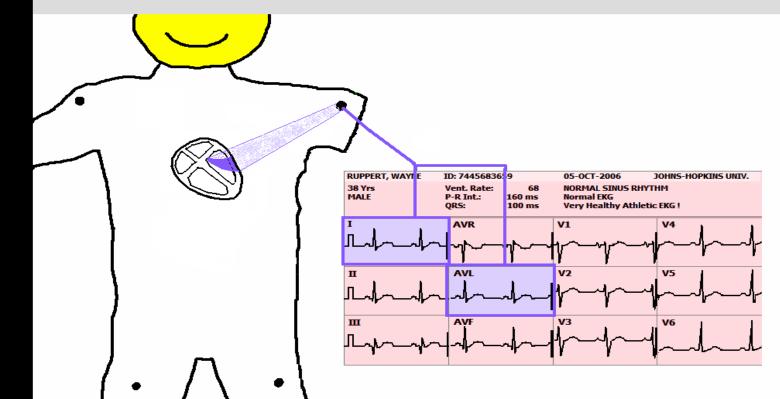


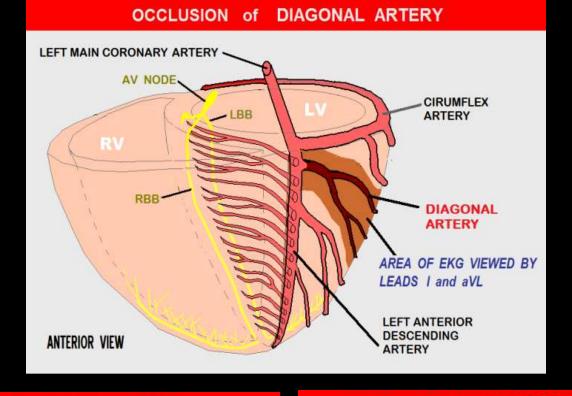


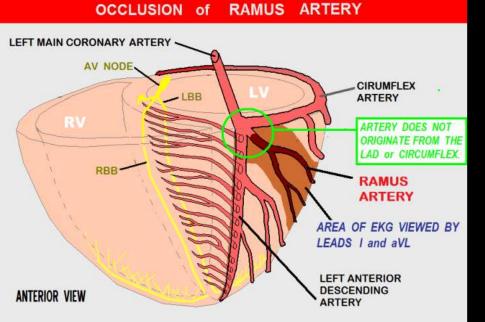


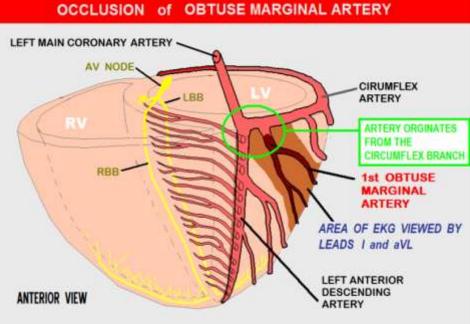
Vent. mie.

LEADS I and aVL view the ANTERIOR-LATERAL JUNCTION

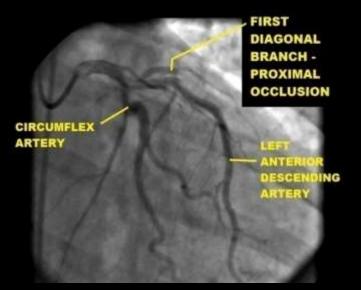








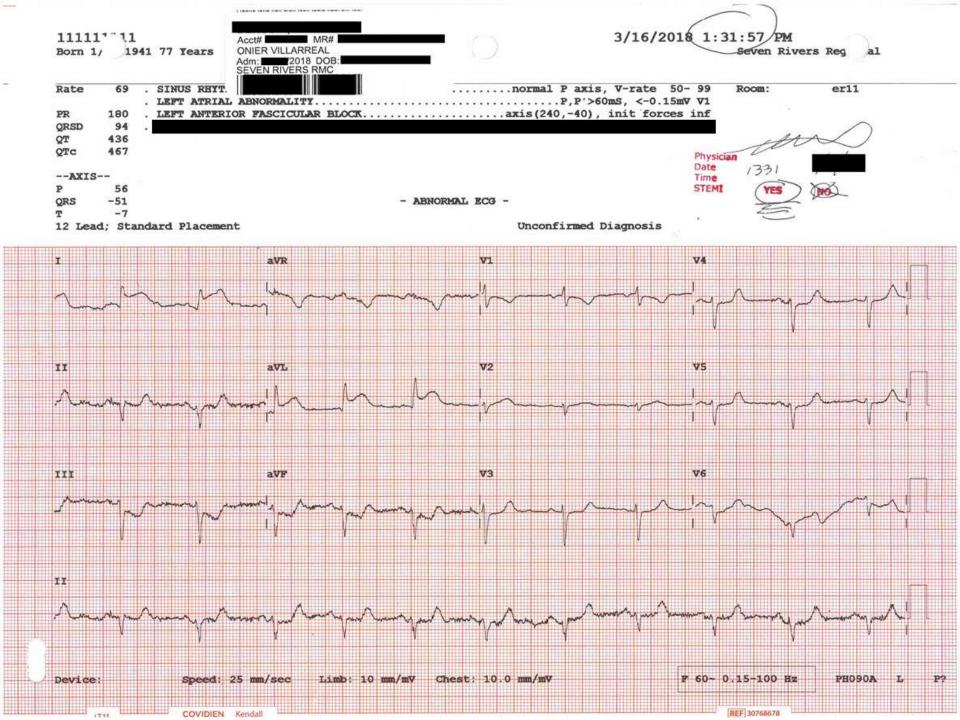
CASE PROGRESSION: As the patient was being prepared for transport to the Cardiac Cath Lab, she experienced an episode of Ventricular Fibrillation.

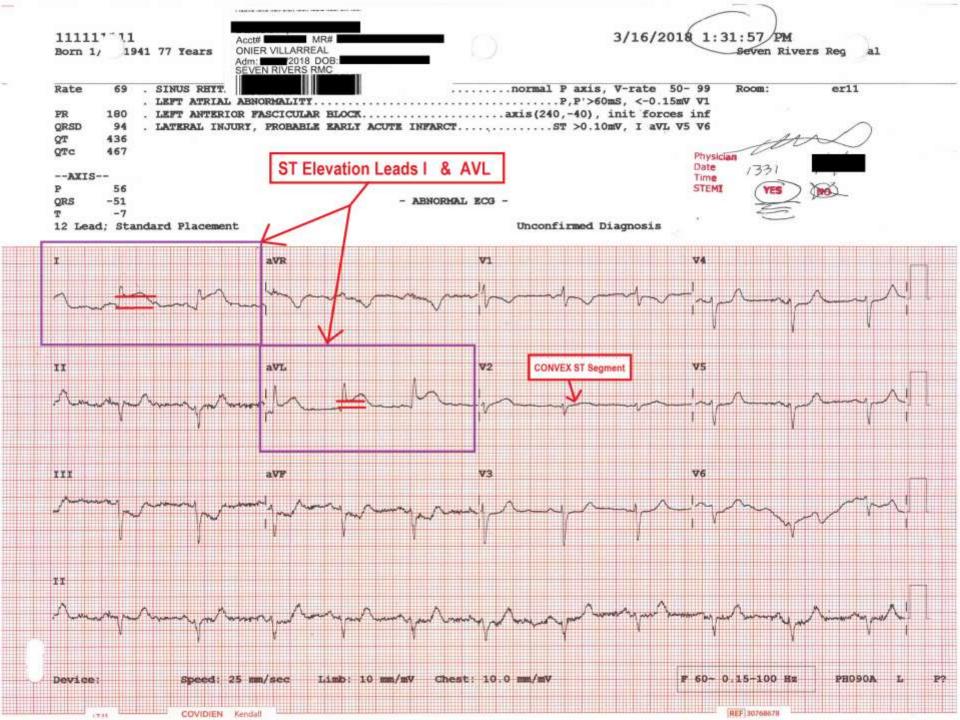


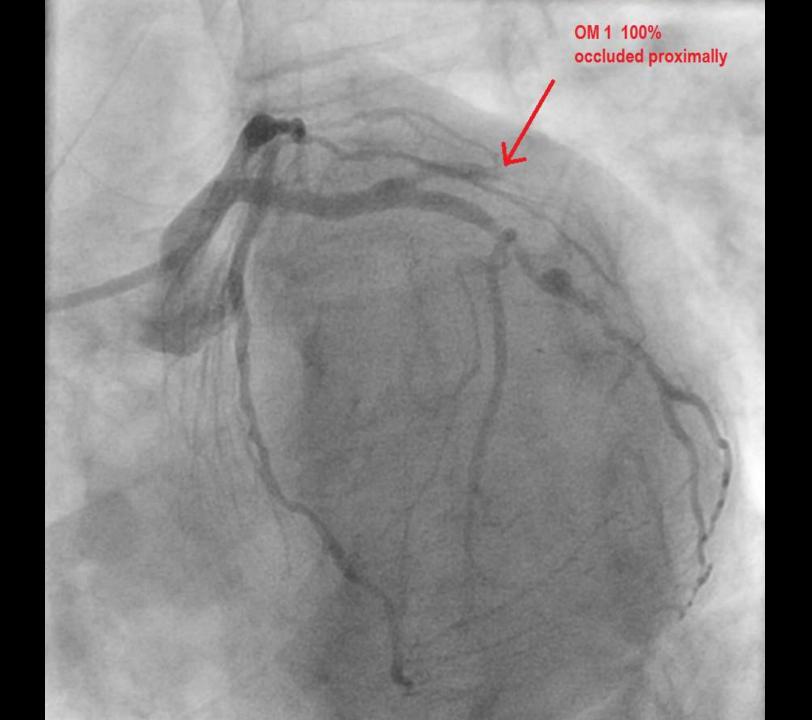


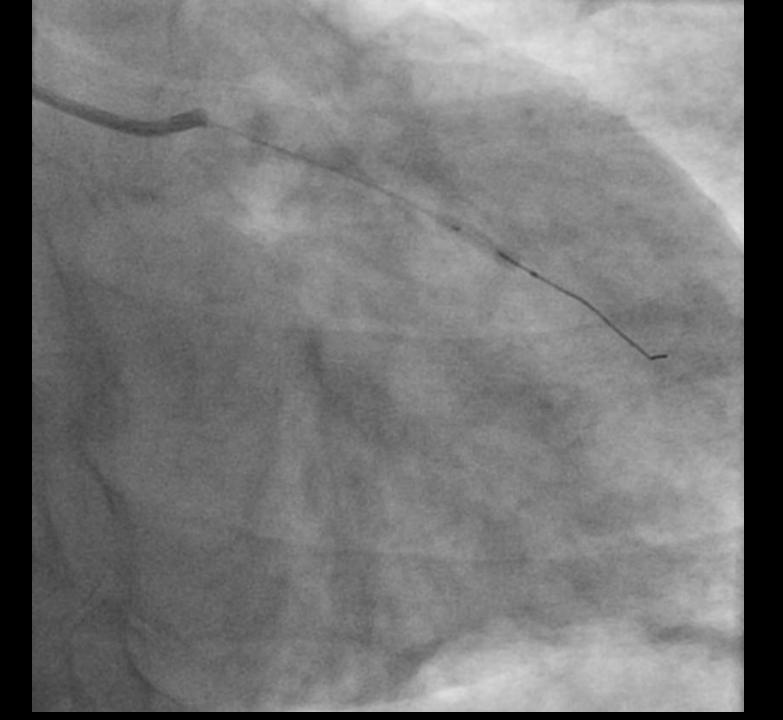


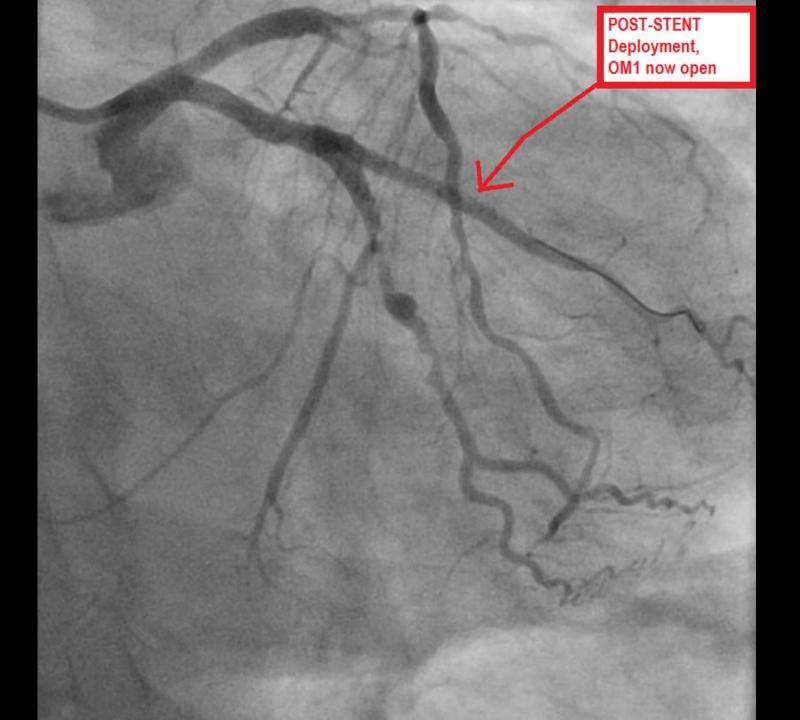












CASE STUDY 3: STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

29 y/o male presents to the ER c/o "HEAVY CHEST PRESSURE" x 30 minutes. The patient states he was playing football with friends after eating a large meal. Pt. also c/o nausea. Denies DIB.

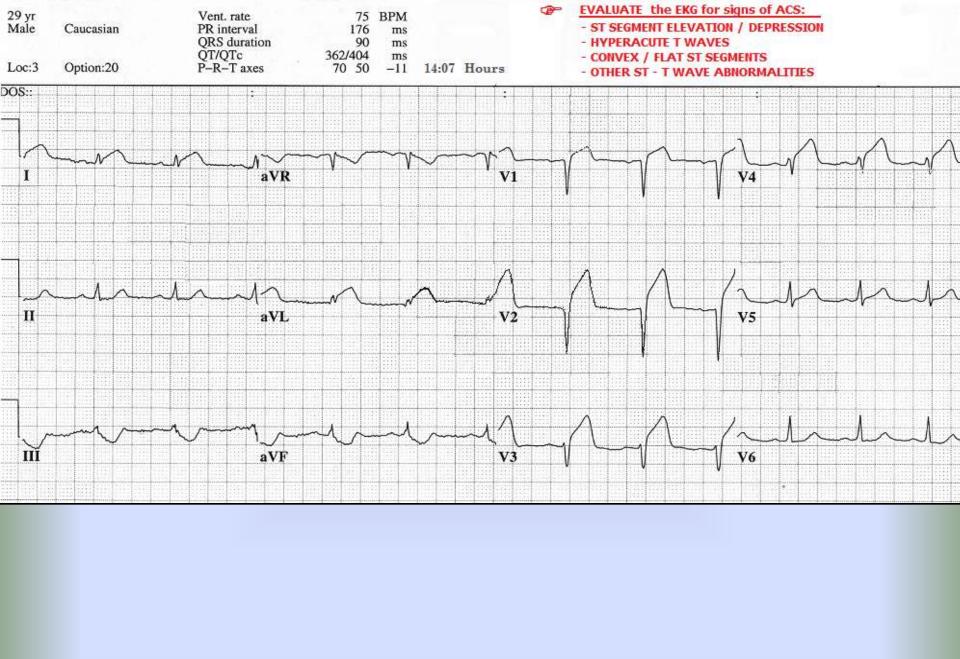
RISK FACTOR PROFILE:

- FAMILY HISTORY father died of MI age 46
- CURRENT CIGARETTE SMOKER
- "MILD" HYPERTENSION untreated
- ✓ CHOLESTEROL unknown "never had it checked."

PHYSICAL EXAM: Patient alert, oriented X 4, skin cool, dry, pale. Patient restless. No JVD, Lungs clear bilaterally. Heart sounds normal S1, S2. No peripheral edema.

VITAL SIGNS: BP: 104/78, P: 76, R: 20, SAO2: 96%

LABS: INITIAL CARDIAC MARKERS - NEGATIVE



29 yr Male

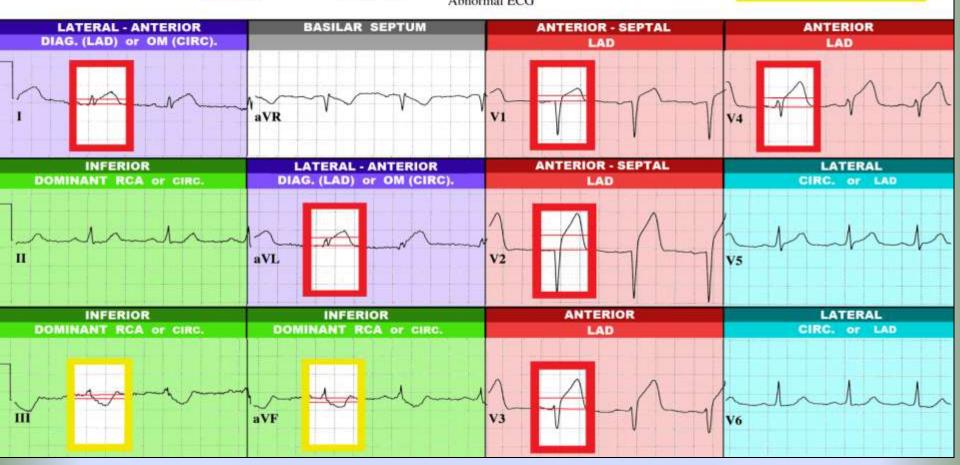
Caucasian

Vent. rate
PR interval
QRS duration
QT/QTc
P-R-T axes

75 BPM 176 ms 90 ms 362/404 ms 70 50 -11 Normal sinus rhythm
Septal infarct, possibly acute
Anterolateral injury pattern
*** ** ** * ACUTE MI ** ** **
Abnormal ECG

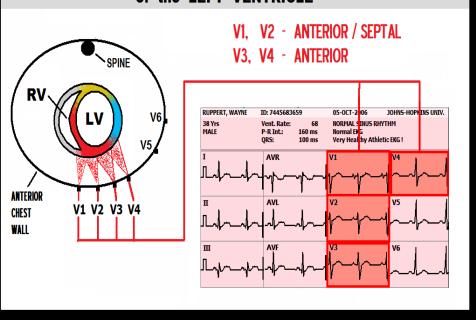
ST SEGMENT ELEVATION

ST SEGMENT DEPRESSION

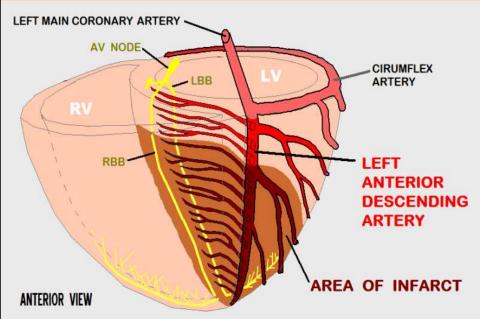


- Reciprocal ST Depression is NOW PRESENT
- Additional ST Elevation is present in Leads I, AVL

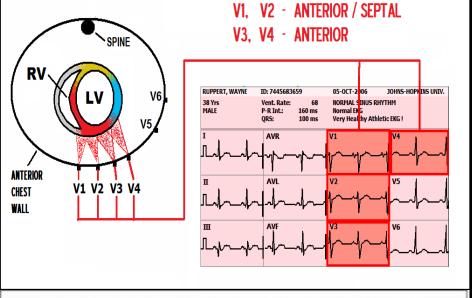
V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL of the LEFT VENTRICLE



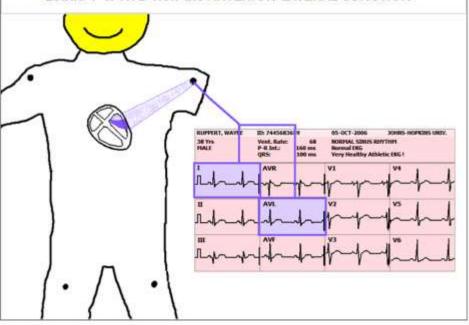
OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY



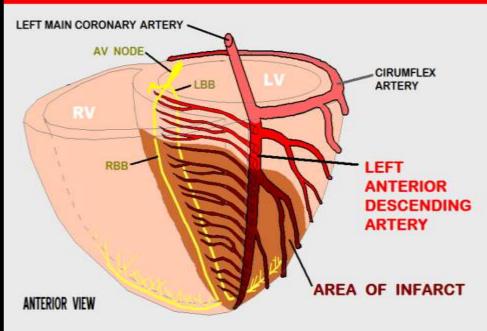
V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL of the LEFT VENTRICLE



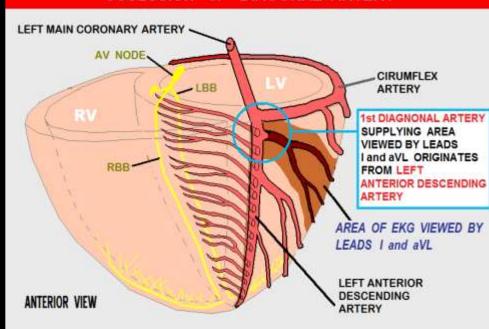
Leads I & AVL view the ANTERIOR-LATERAL JUNCTION

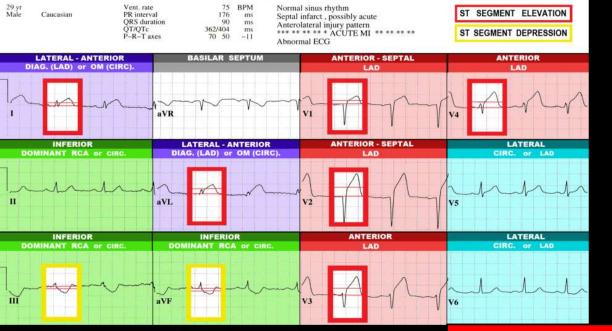


OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY



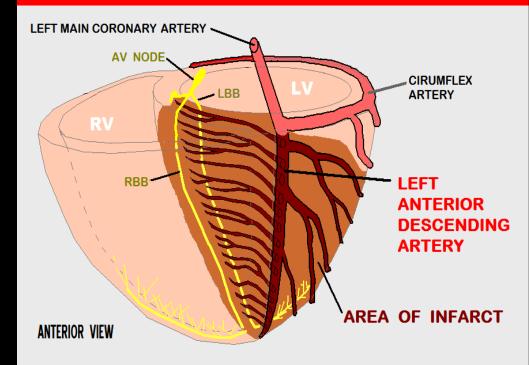
OCCLUSION of DIAGONAL ARTERY



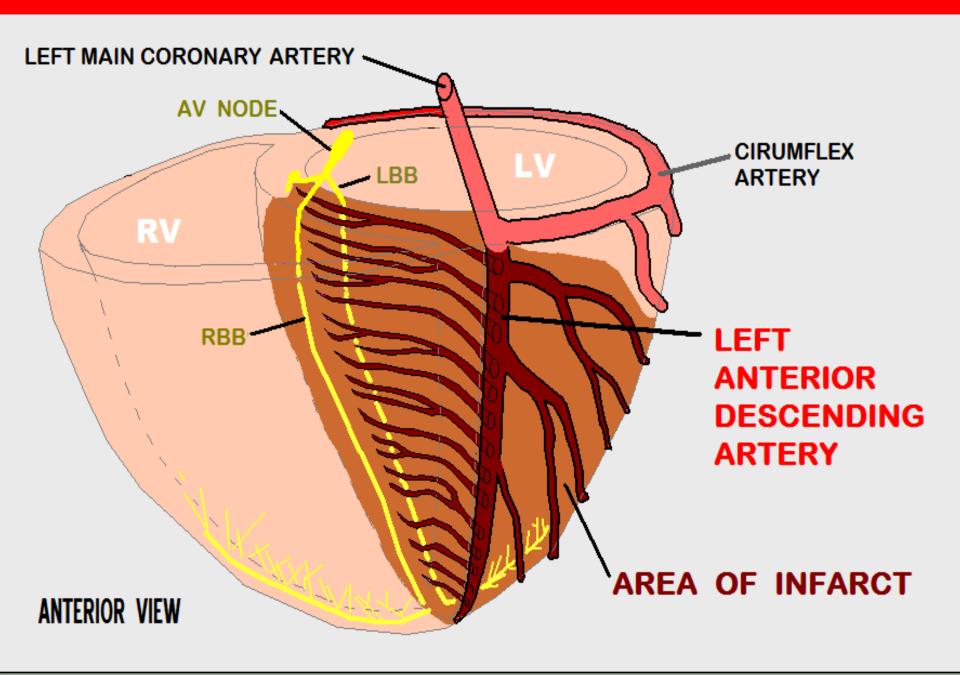


Vent. rate

OCCLUSION of PROXIMAL LEFT ANTERIOR DESCENDING ARTERY



OCCLUSION of PROXIMAL LEFT ANTERIOR DESCENDING ARTERY



ANTICIPATED COMPLICATIONS of ANTERIOR-SEPTAL WALL STEMI & POSSIBLE INDICATED INTERVENTIONS: BCLS / ACLS - CARDIAC ARREST **ACLS** (antiarrhythmics)

-DOPAMINE / DOBUTAMINE /

- INTRA-AORTIC BALLOON PUMP

(use caution with fluid challenges

(use caution with dieuretics due to

pump failure and hypotension)

due to PULMONARY EDEMA)

LEVOPHED

- CPAP

- ET INTUBATION

TRANSCUTANEOUS or

TRANSVENOUS PACING

- CARDIAC DYSRHYTHMIAS (VT / VF) - PUMP FAILURE with **INOTROPE THERAPY:** CARDIOGENIC SHOCK

- PULMONARY EDEMA

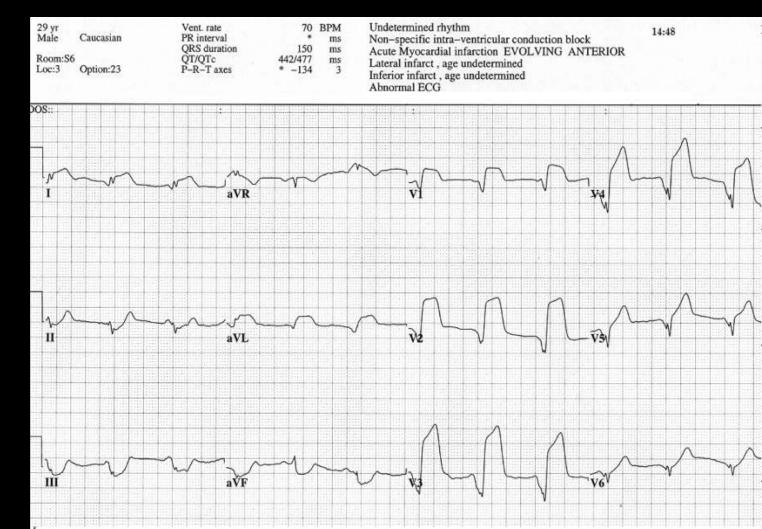
- 3rd DEGREE HEART BLOCK - NOT

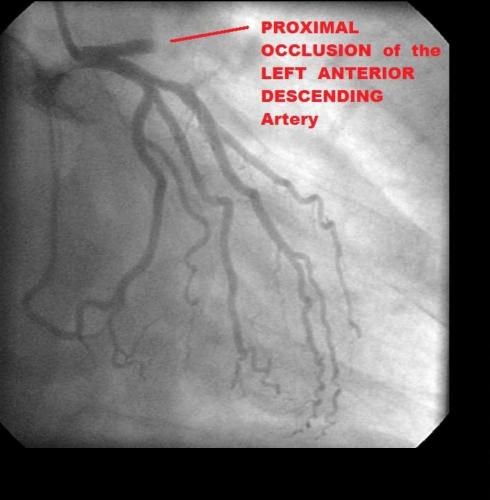
RESPONSIVE TO ATROPINE

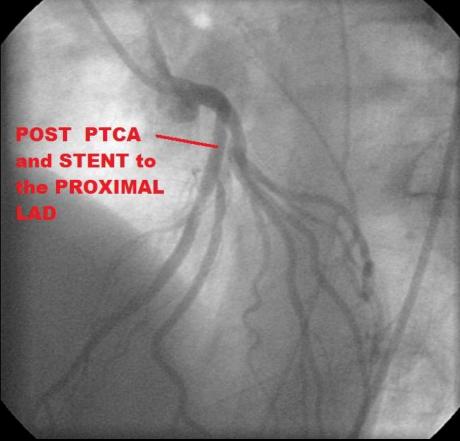
WHILE AWAITING THE CATH LAB TEAM, THE PATIENT BEGAN VOMITING. SKIN BECAME ASHEN & DIAPHORETIC. REPEAT BP = 50/30.

WHILE AWAITING THE CATH LAB TEAM, THE PATIENT BEGAN VOMITING. SKIN BECAME ASHEN & DIAPHORETIC. REPEAT BP = 50/30.

-WHAT THERAPEUTIC INTERVENTIONS SHOULD BE IMPLMENTED AT THIS POINT?



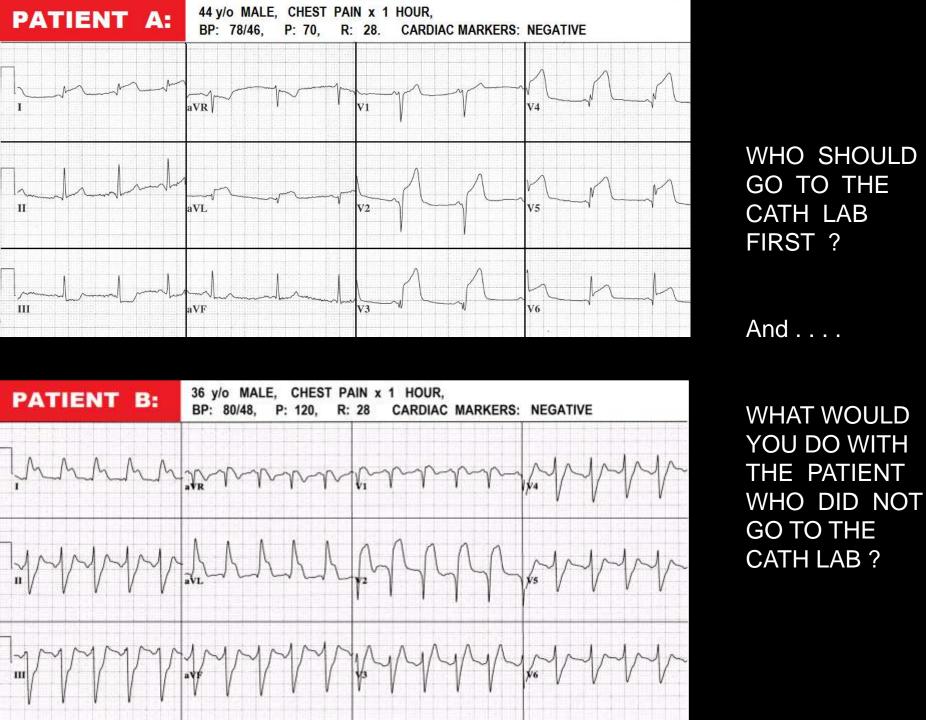


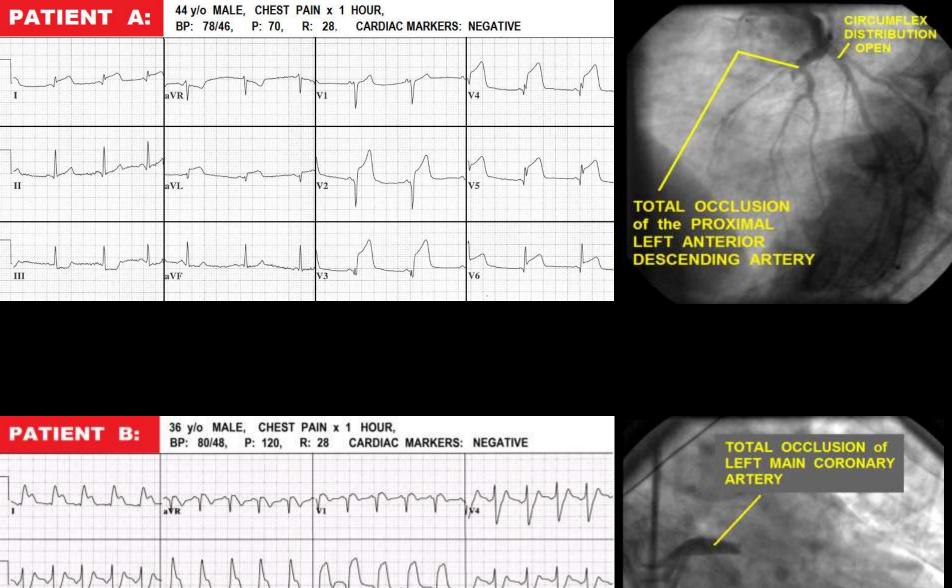


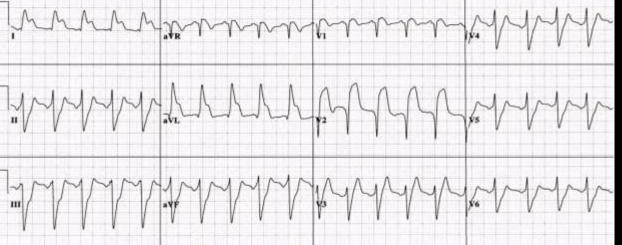
CASE STUDY 4: CRITICAL DECISIONS SCENARIO

As per current AHA recommendations, your hospital's policy is to send every STEMI patient to the Cardiac Catheterization Lab for emergency PCI.

You are the ranking medical officer on duty in the ED when two acute STEMI patients arrive, ten minutes apart. The Cath Lab has one lab open, and can take ONE patient immediately. Both patients duration of symptoms and state of hemodynamic stability are similar.





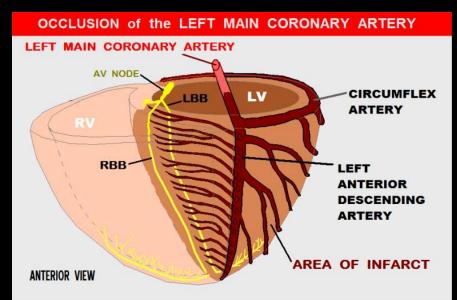


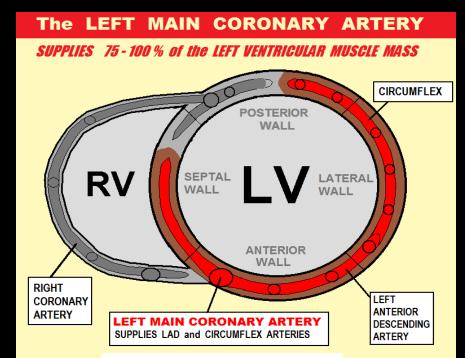
PATIENT A:

LEFT MAIN CORONARY ARTERY AV NODE AV NODE LEFT ANTERIOR DESCENDING ARTERY LEFT ANTERIOR DESCENDING ARTERY AREA OF INFARCT

SUPPLIES 40-50% OF THE LEFT VENTRICULAR MUSCLE MASS CIRCUMFLEX POSTERIOR WALL ANTERIOR WALL RIGHT CORONARY ARTERY LEFT ANTERIOR DESCEDING ARTERY = APPROXIMATELY 45% LV MUSCLE MASS

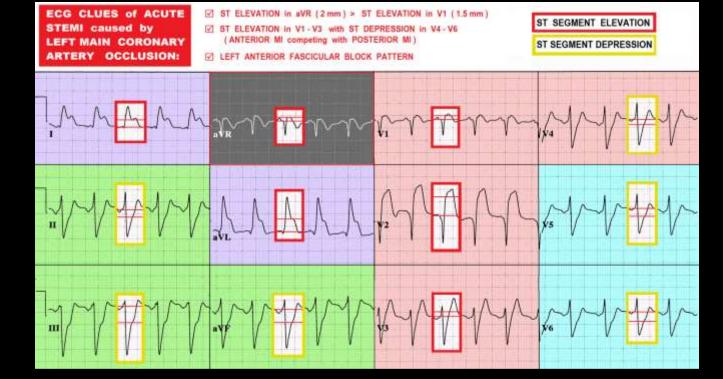
PATIENT B:



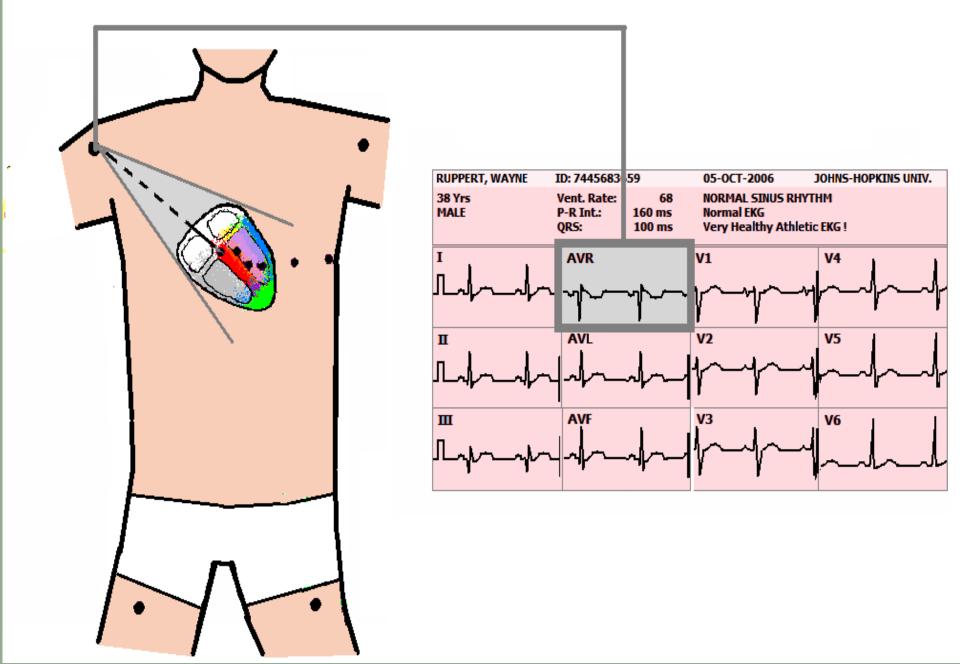


FCG Clues... for IDENTIFYING STEMI CAUSED BY LEFT MAIN CORONARY ARTERY occlusion:

- ☑ ST ELEVATION in ANTERIOR LEADS (V1 V4) and LATERAL LEADS (V5 & V6)
- ✓ ST DEPRESSION or ISOELCTRIC J POINTS may be seen in V LEADS.... mainly V2 and/or V3 caused by COMPETING FORCES of ANTERIOR vs. POSTERIOR WALL MI.*+
 - → NOTE: it is very unusual to see ST DEPRESSION in V LEADS with isolated ANTERIOR WALL MI when caused by occluded LAD.
- ✓ ST ELEVATION in AVR is GREATER THAN ST ELEVATION in V1*+
- ✓ ST ELEVATION in AVR GREATER THAN 0.5 mm
- ☑ ST ELEVATION in LEAD I and AVL (caused by NO FLOW to DIAGONAL / OBTUSE MARGINAL BRANCHES)*
- ✓ ST DEPRESSION in LEADS II, III, and AVF. (in cases of LMCA occlusion of DOMINANT CIRCUMFLEX, leads II, III, and AVF may show ST ELEVATION or ISOELECTRIC | POINTS)**
- ✓ NEW / PRESUMABLY NEW RBBB, and/or LEFT ANTERIOR FASICULAR BLOCK**
 - * Kurisu et al, HEART 2004, SEPTEMBER: 90 (9): 1059-1060
 - + Yamaji et al, JACC vol. 38, No. 5, 2001, November 1, 2001:1348-54

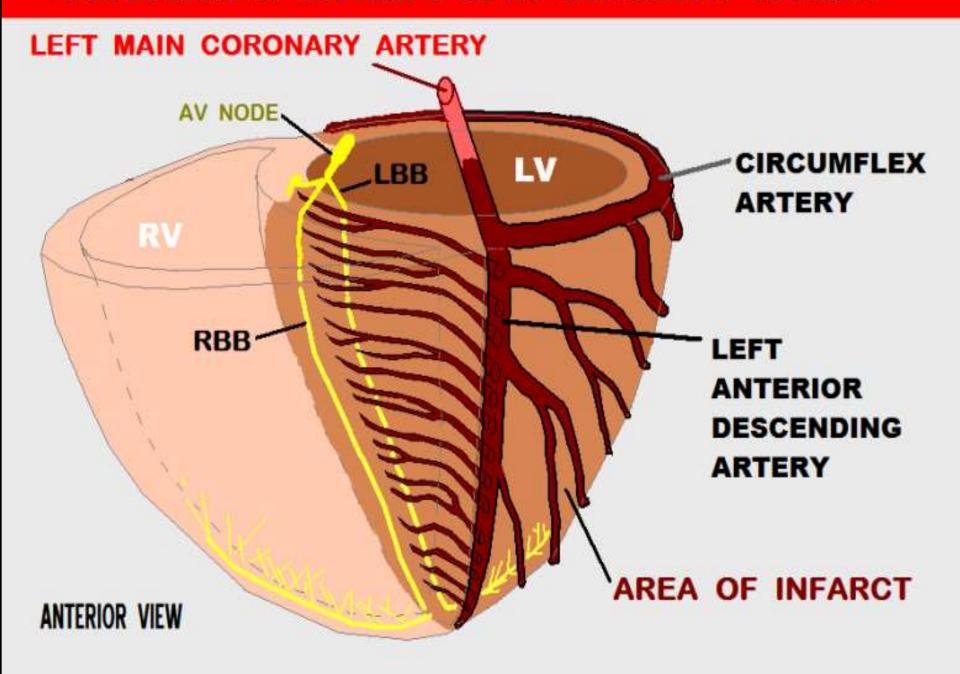


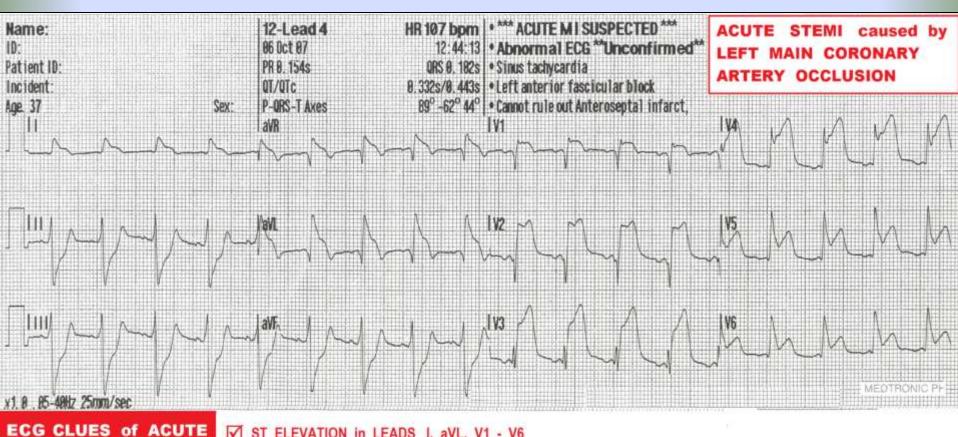
Lead AVR Views the BASILAR SEPTUM (region of the Bundle of His)



In STEMI with ST-Segment Elevation in Lead AVR, This is indicative of Left Main Coronary Artery Occlusion . . .

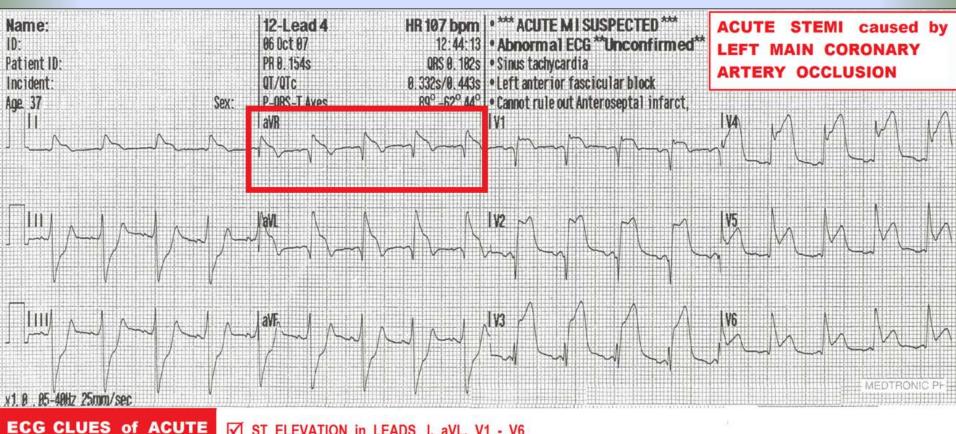
OCCLUSION of the LEFT MAIN CORONARY ARTERY





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

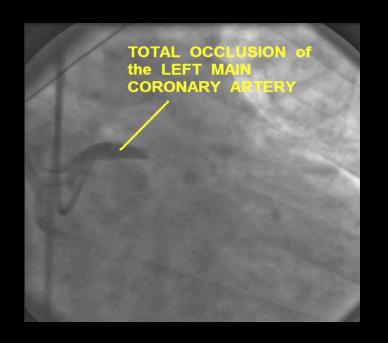
- ST ELEVATION in LEADS I, aVL, V1 V6
- ST ELEVATION in aVR GREATER THAN 0.5 mm
- ST ELEVATION in aVR GREATER THAN LEAD V1
- LEFT ANTERIOR FASCICULAR BLOCK PATTERN

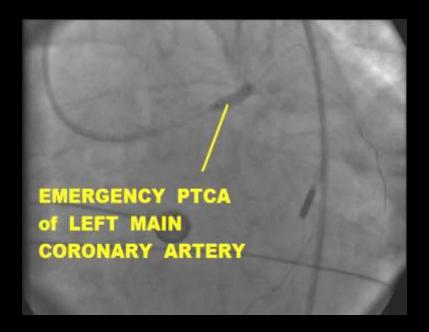


STEMI caused by **LEFT MAIN CORONARY OCCLUSION:** ARTERY

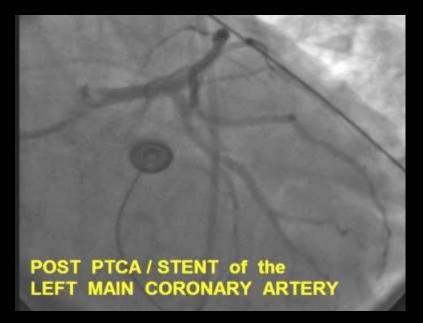
- ST ELEVATION in LEADS I, aVL, V1 V6
- ST ELEVATION in aVR GREATER THAN 0.5 mm
- ST ELEVATION in aVR GREATER THAN LEAD V1
- ✓ LEFT ANTERIOR FASCICULAR BLOCK PATTERN

TOTAL OCCLUSION of the LEFT MAIN CORONARY ARTERY





Despite the dismal mortality rate associated with STEMI from total LMCA occlusion, this patient survived and was later discharged. His EF is estimated at approximately 30%. He received an ICD, and is currently stable.



CASE STUDY 4: CRITICAL DECISIONS SCENARIO

CONCLUSIONS:

QUESTION 1: WHICH PATIENT SHOULD BE TAKEN FIRST FOR IMMEDIATE CARDIAC

CATHETERIZATION for EMERGENCY PCI?

ANSWER: PATIENT B was taken emergently to the Cardiac Cath Lab - both the ED physician

and the Interventional Cardiologist correctly identified the EKG patterns

of LMCA occlusion.

QUESTION 2: WHAT COURSE OF ACTION SHOULD BE TAKEN WITH THE PATIENT NOT CHOSEN

TO BE SENT TO THE CATH LAB FIRST?

ANSWER: PATIENT A received thrombolytic therapy in the ED. It was determined that

THROMBOLYTIC THERAPY would achieve the FASTEST ROUTE to REPERFUSION --

-- by at least 60 minutes.

FCG Clues... for identifying stemi caused by LEFT MAIN CORONARY ARTERY occlusion:

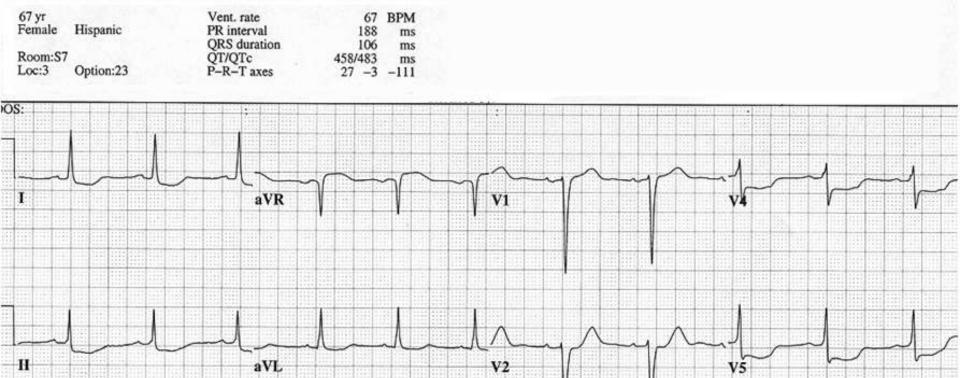
- ☑ ST ELEVATION in ANTERIOR LEADS (V1 V4) and LATERAL LEADS (V5 & V6)
- ☑ ST DEPRESSION or ISOELCTRIC J POINTS may be seen in V LEADS.... mainly V2 and/or V3 caused by COMPETING FORCES of ANTERIOR vs. POSTERIOR WALL MI.**
 - → NOTE: it is very unusual to see ST DEPRESSION in V LEADS with isolated ANTERIOR WALL MI when caused by occluded LAD.
- ✓ ST ELEVATION in AVR is GREATER THAN ST ELEVATION in V1*+
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- ☑ ST ELEVATION in LEAD I and AVL (caused by NO FLOW to DIAGONAL / OBTUSE MARGINAL BRANCHES)*
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 - * Kurisu et al, HEART 2004, SEPTEMBER: 90 (9): 1059-1060
 - + Yamaji et al, JACC vol. 38, No. 5, 2001, November 1, 2001:1348-54

Yamaji et al, JACC vol 38, No 5, 2001: 1348-54

Electrocardiogram patterns in acute left main occlusion: J Electrocardiol. 2008 Nov-Dec;41(6):626-9.

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**

- "In patients with:
 - Angina at rest
- ST Elevation in AVR and ST Depression in 8 or more ECG leads (global ischemia), it is reported with a 75% predictive accuracy of 3-vessel or left main coronary artery stenosis"...
- Wagner et al, 2009 ACC/AHA Standardization and Interpretation of the ECG, Part VI, ACS.



V3

V6

Ш

aVF

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

 Vent. rate
 67
 BPM

 PR interval
 188
 ms

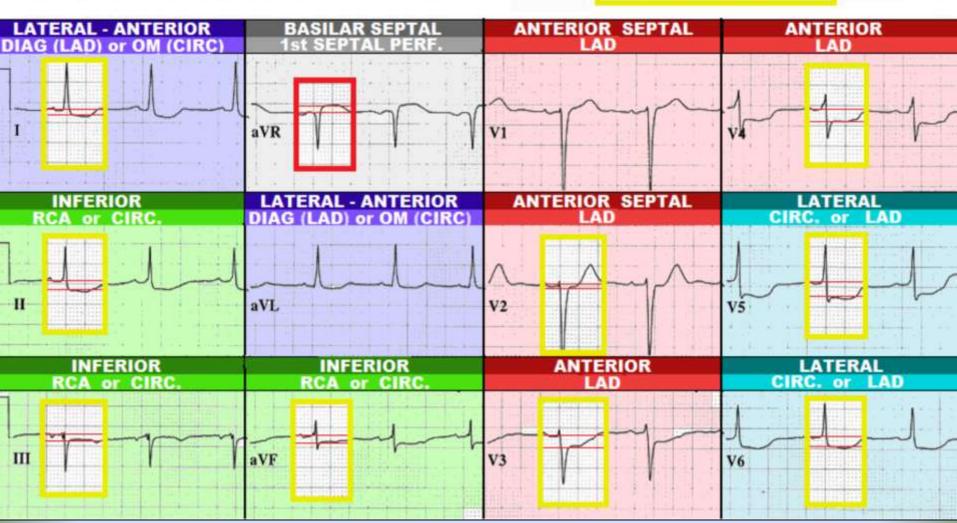
 QRS duration
 106
 ms

 QT/QTc
 458/483
 ms

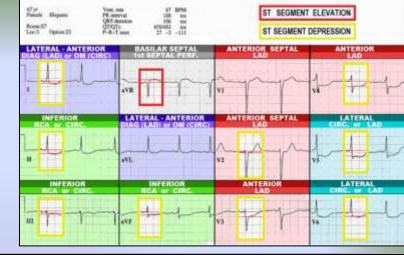
 P-R-T axes
 27
 -3
 -111

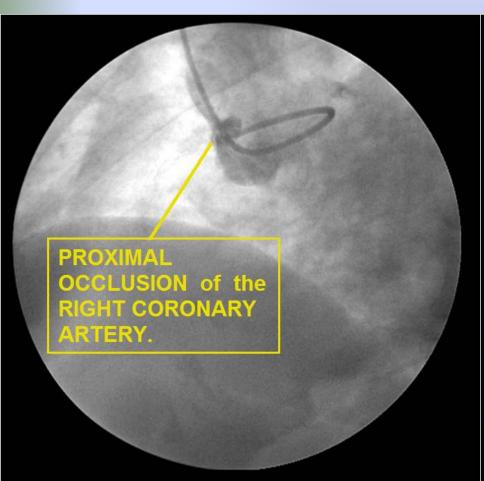
ST SEGMENT ELEVATION

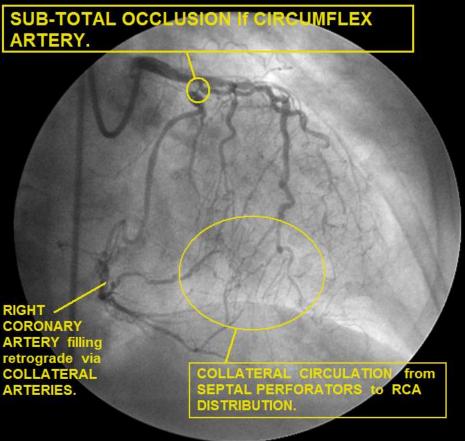
ST SEGMENT DEPRESSION



Critical Triple Vessel Disease = STAT Coronary Artery Bypass Surgery







ANTICIPATED COMPLICATIONS of GLOBAL ISCHEMIA with POSSIBLE NSTEMI -- INTERVENTIONS to be CONSIDERED:

Patients with CHEST PAIN at REST and this ECG	PREHOSPITAL: if patient has no hospital
presentation have a 75% incidence of severe LMCA	preference consider transport to Chest Pain
STENOSIS and/orTRIPLE - VESSEL DISEASE in	Center WITH Open Heart Surgery
such cases Coronary Artery Bypass Surgery (CABG)	capabilities IF nearby.
is frequently indicated.	
	HOSPITAL: consider use of SHORT-ACTING
	intravenous GP IIb/IIIa receptor agonists
- ACTIVE CHEST PAIN	ACUTE CHEST PAIN PROTOCOL
- ISCHEMIA - CONSIDER DYSRHYTHMIAS	ACLS PROTOCOL
- INCREASED PROBABILITY of IMMINENT	1. AGGRESSIVE SERIAL TROPONIN and
MYOCARDIAL INFARCTION	SERIAL ECG PROTOCOLS (2014 AHA / ACC

/ NSTE-ACS Guidelines)

Cardiac Catheterization

2. Positive TROPONIN: consider STAT / early

Excerpt from **STEM/ASSISTANT**

CASE STUDY 7 - STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

46 yr. old MALE arrives in ER, C/O SUDDEN ONSET OF CHEST PRESSURE 45 MINUTES AGO. PAIN IS CONSTANT, PRESSURE-LIKE, AND NOT EFFECTED BY POSITION, MOVEMENT or DEEP INSPIRATION. ALSO C/O D.I.B.

RISK FACTOR PROFILE:

● CURRENT CIGARTTE SMOKER x 18 YEARS**

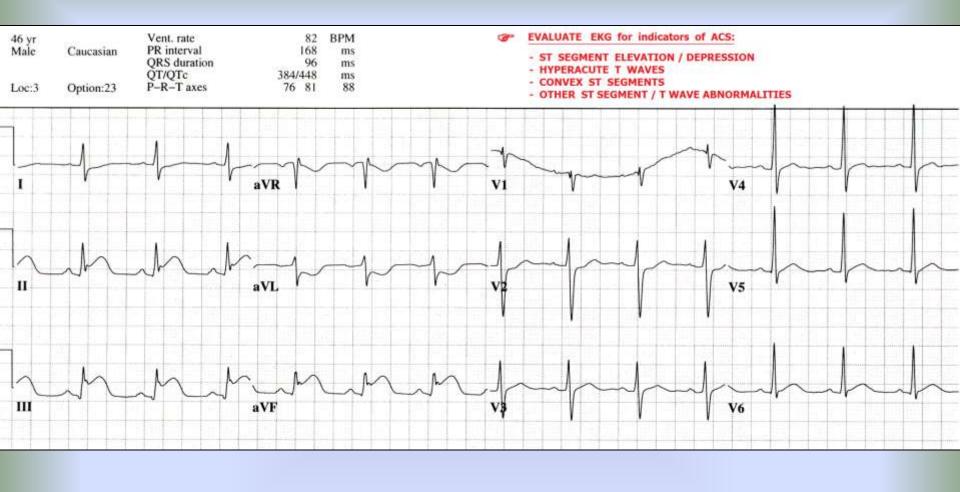
HYPERTENSION

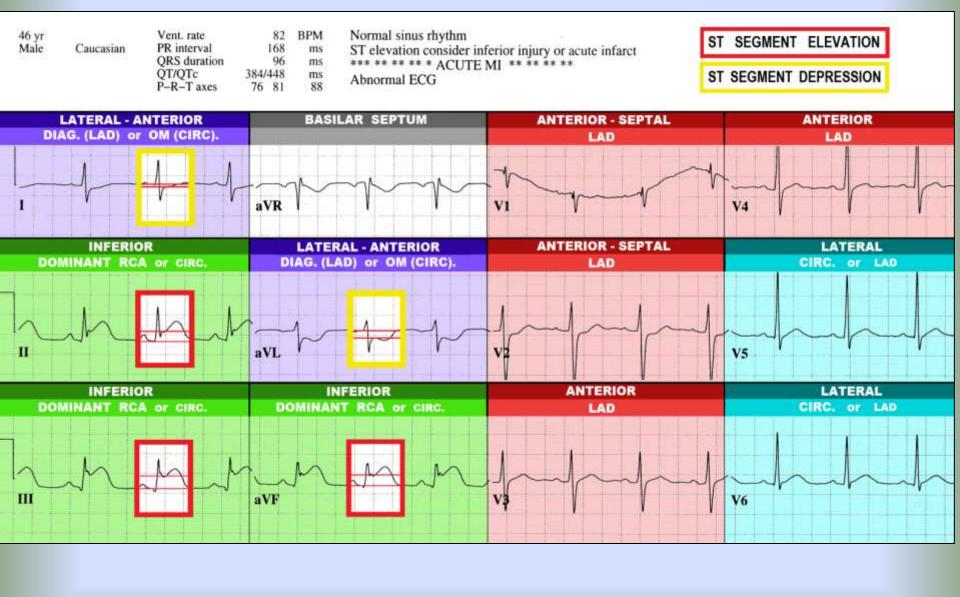
♠™ HIGH LDL CHOLESTEROL

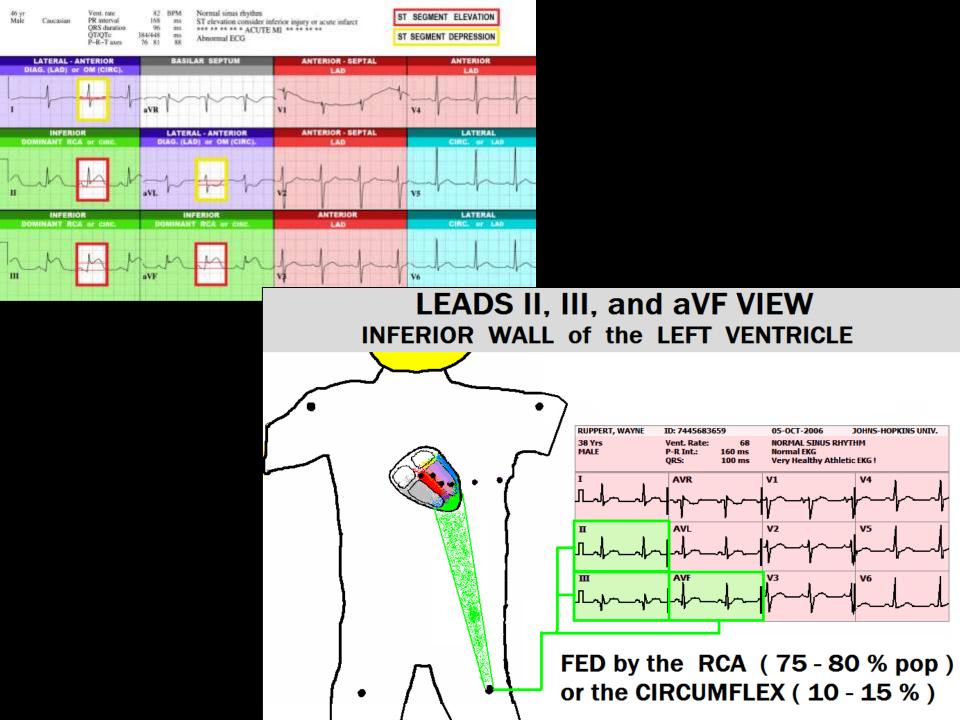
PHYSICAL EXAM: Patient is alert & oriented x 4, skin warm, dry, color normal. Non-anxious Lungs clear, normal S1, S2. No JVD, No ankle edema.

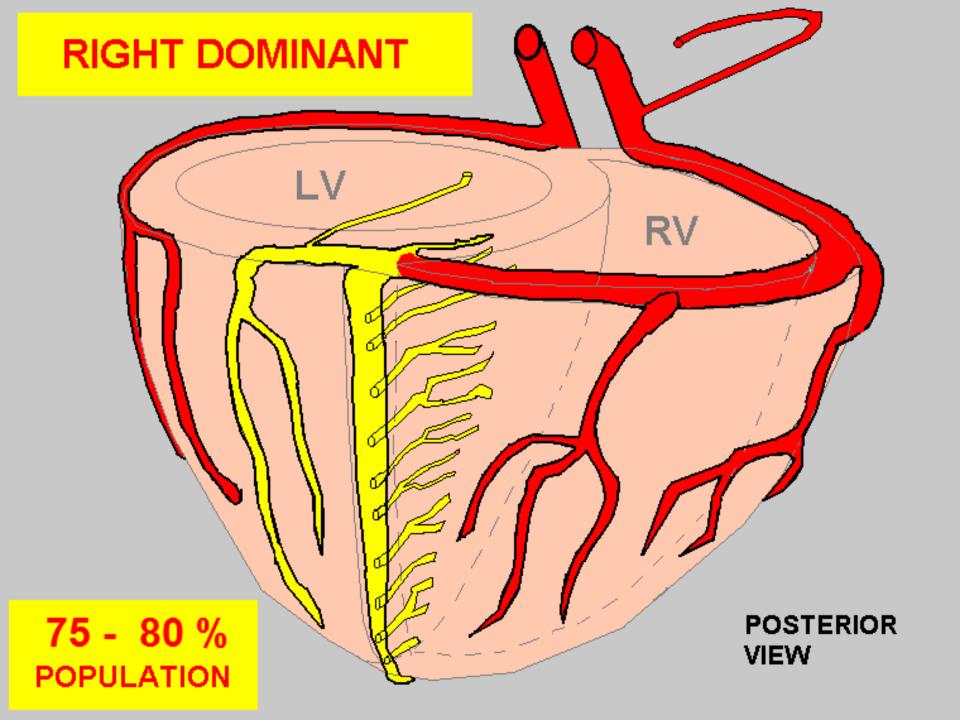
VITAL SIGNS: BP: 136/88 P: 88 R: 20 SAO2: 100% on 4 LPM O2

LABS: TROPONIN: < .04









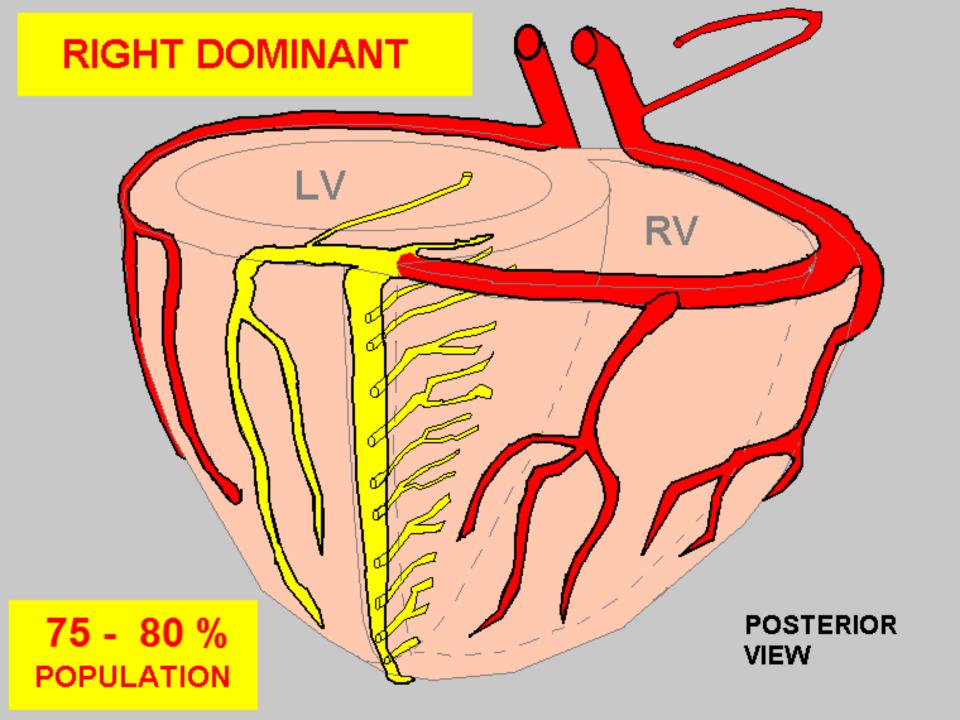




RIGHT CORONARY ARTERY (RCA)

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



A standard

12 LEAD EKG

Does NOT show the

RIGHT VENTRICLE

To see the RIGHT VENTRICLE . . .

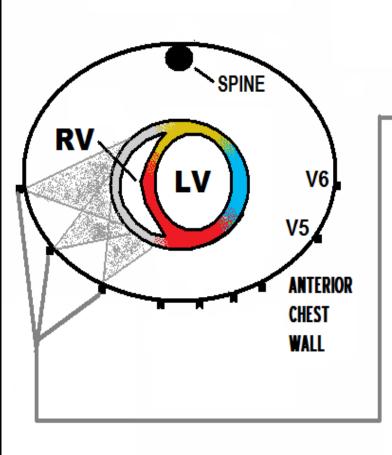
... such as in cases of

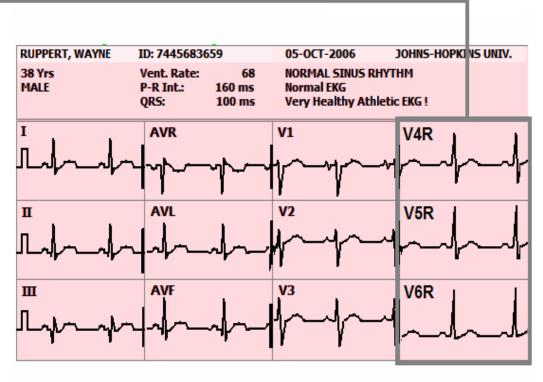
INFERIOR WALL M.I.

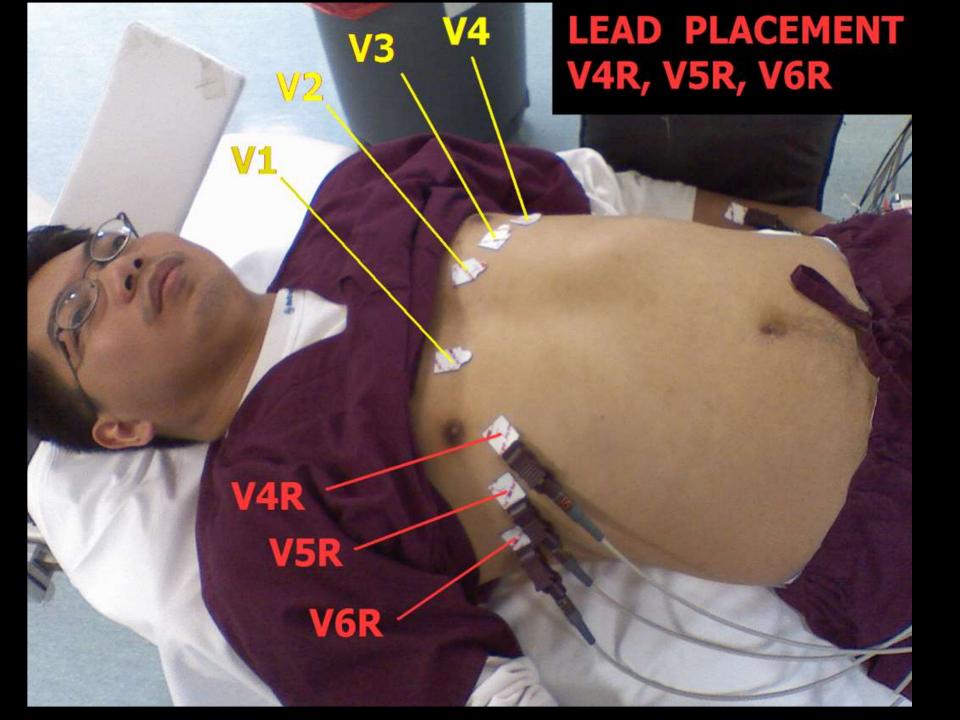
You must do a

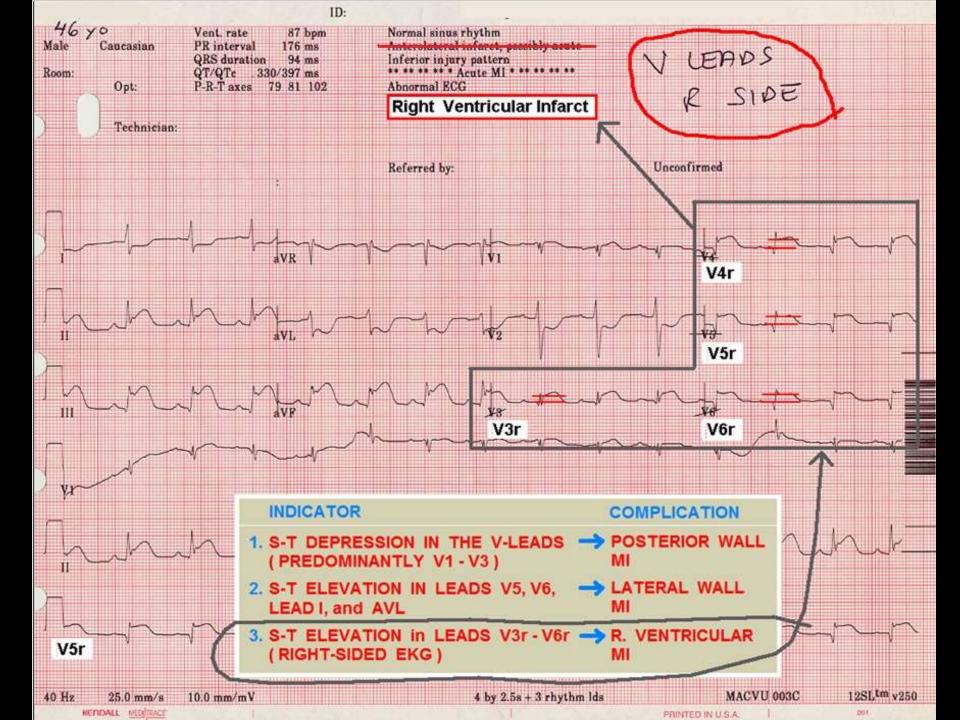
RIGHT - SIDED EKG!!

V4R - V6R VIEW THE RIGHT VENTRICLE

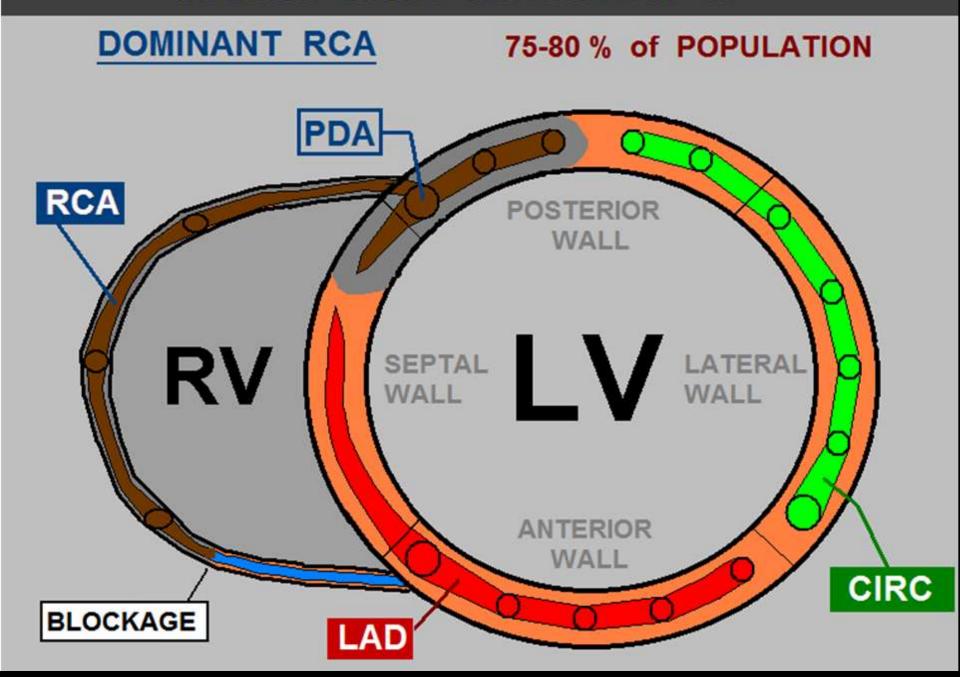








INFERIOR - RIGHT VENTRICULAR MI



ANTICIPATED COMPLICATIONS of INFERIOR WALL STEMI secondary to RCA Occlusion & POSSIBLE INDICATED INTERVENTIONS:

- CARDIAC ARREST BCLS / ACLS

ACLS (antiarrhythmics)

- CARDIAC DYSRHYTHMIAS (VT / VF) - SINUS BRADYCARDIA (follow ACLS and/or UNIT protocols) - HEART BLOCKS (1st, 2nd & 3rd Degree HB)

ATROPINE 0.5mg, REPEAT as needed UP TO 3mg. ATROPINE 0.5mg, REPEAT as needed UP TO 3mg, Transcutaneous Pacing, (follow ACLS and/or UNIT protocols)

- The standard 12 Lead ECG does NOT view - RIGHT VENTRICULAR MYOCARDIAL INFARCTION the Right Ventricle. - You must do a RIGHT-SIDED ECG to see if RV MI is present. - Do NOT give any Inferior Wall STEMI patient been RULED OUT.

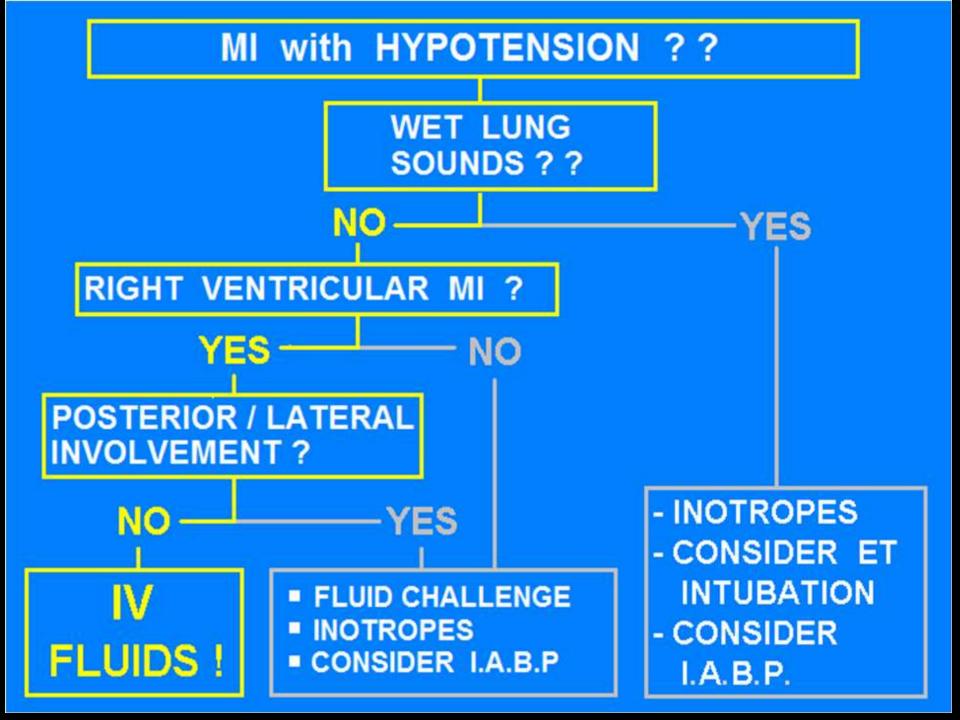
NITRATES or DIURETICS until RV MI has - POSTERIOR WALL INFARCTION - POSTERIOR WALL MI presents on the 12 Lead ECG as ST DEPRESSION in Leads V1 -

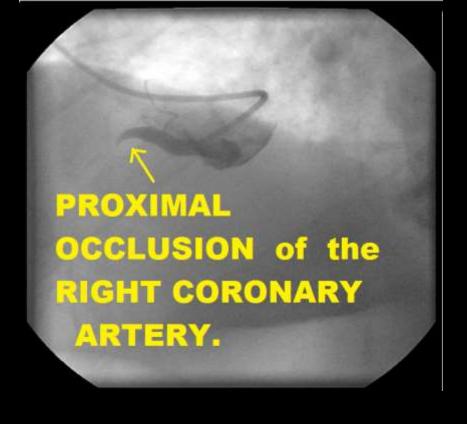
V3.

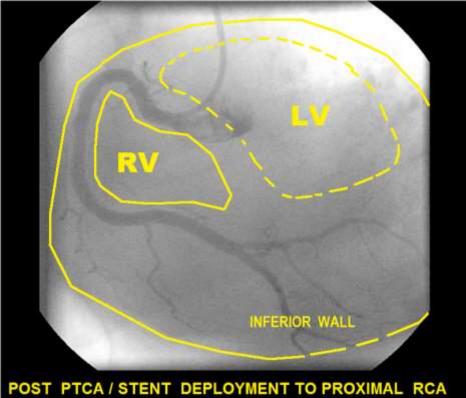
ON THIS FCG.

- POSTERIOR WALL MI is NOT PRESENT

If this patient becomes HYPOTENSIVE....







IN **EVERY** CASE of

INFERIOR WALL STEMI

You must first *RULE OUT*RIGHT VENTRICULAR MI

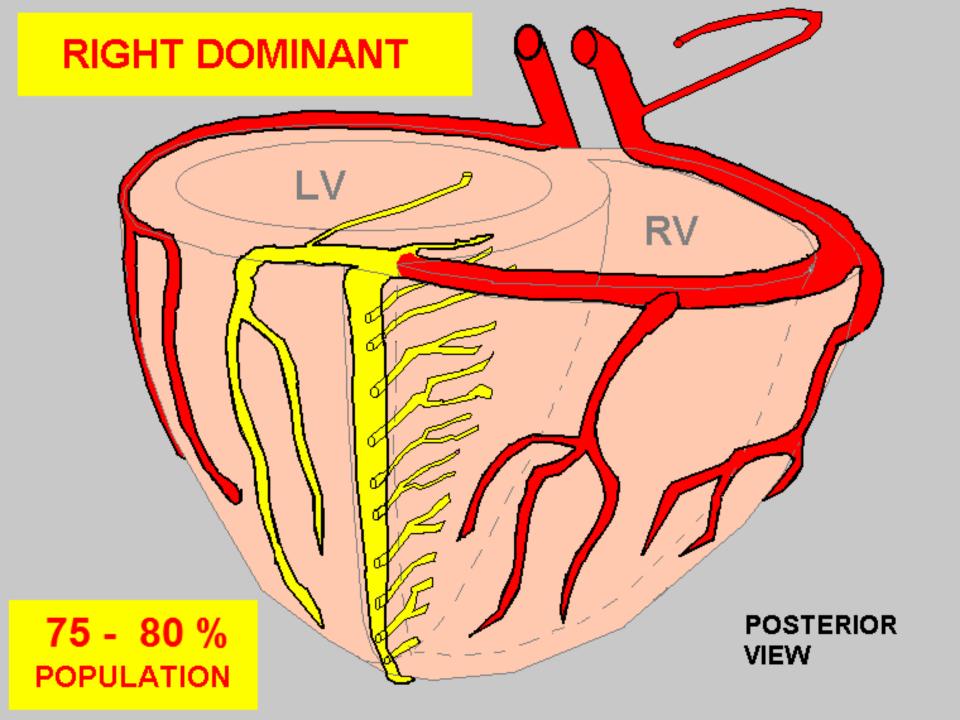
BEFORE giving any:

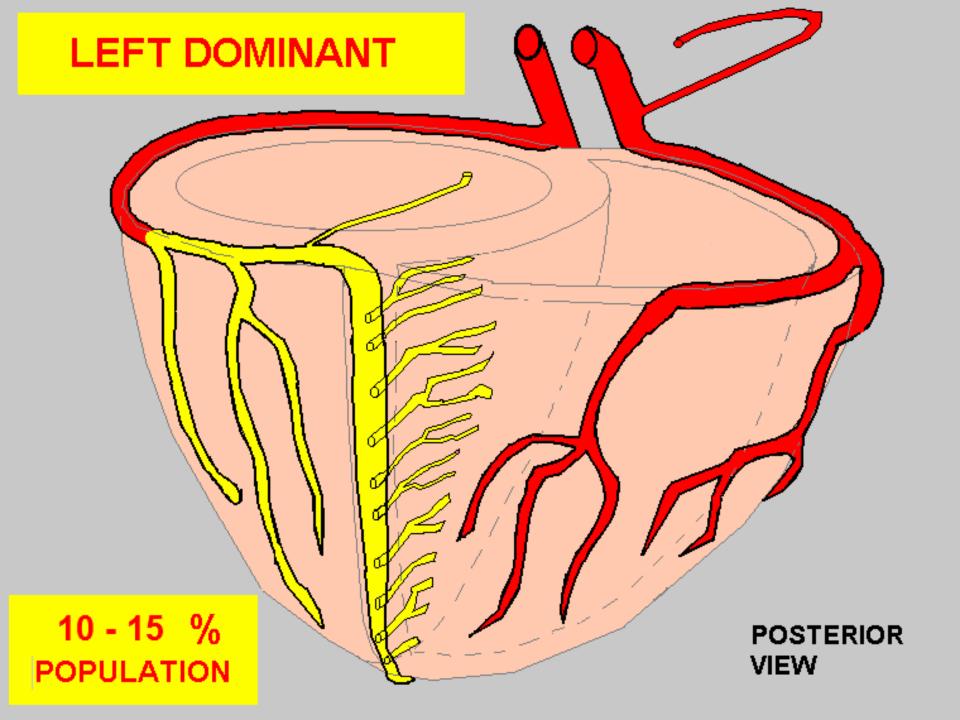
- NITROGLYCERIN
- Diuretics

Nitroglycerin & Diuretics are CLASS III CONTRINDICATED in RIGHT VENTRICULAR MI!!*

They precipitate SEVERE HYPOTENSION

* A.H.A. ACLS 2010 / 2015





CASE STUDY 9 - STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

42 y/o MALE arrived via EMS, c/o "HEAVY CHEST PRESSURE," SHORTNESS of BREATH X 40 min. He has experienced V-FIB and been DEFIBRILLATED multiple times

RISK FACTOR PROFILE:

- **●** CIGARETTE SMOKER**
- HYPERTENSION
- **●** HIGH LDL CHOLESTEROL**

PHYSICAL EXAM: Patient is alert & oriented x 4, ANXIOUS, with COOL, PALE, DIAPHORETIC SKIN. C/O NAUSEA, and is VOMITING. LUNG SOUNDS: COARSE CRACKLES, BASES, bilaterally

VITAL SIGNS: BP: 80/40 P: 70 R: 32 SAO2: 92 % on 15 LPM O2

LABS: TROPONIN: < .04

SHORY ASSESSMENT

LOC: **ANXIOUS AWAKE** RESTLESS **ALERT &** LETHARGIC ORIENTED UNCONSCIOUS

PALE / ASHEN SKIN: CYANOTIC COOL DIAPHORETIC

NORMAL HUE WARM DRY

TACHYPNEA BREATHING: WEAK / THREADY PULSE: TOO FAST or SLOW

STRONG

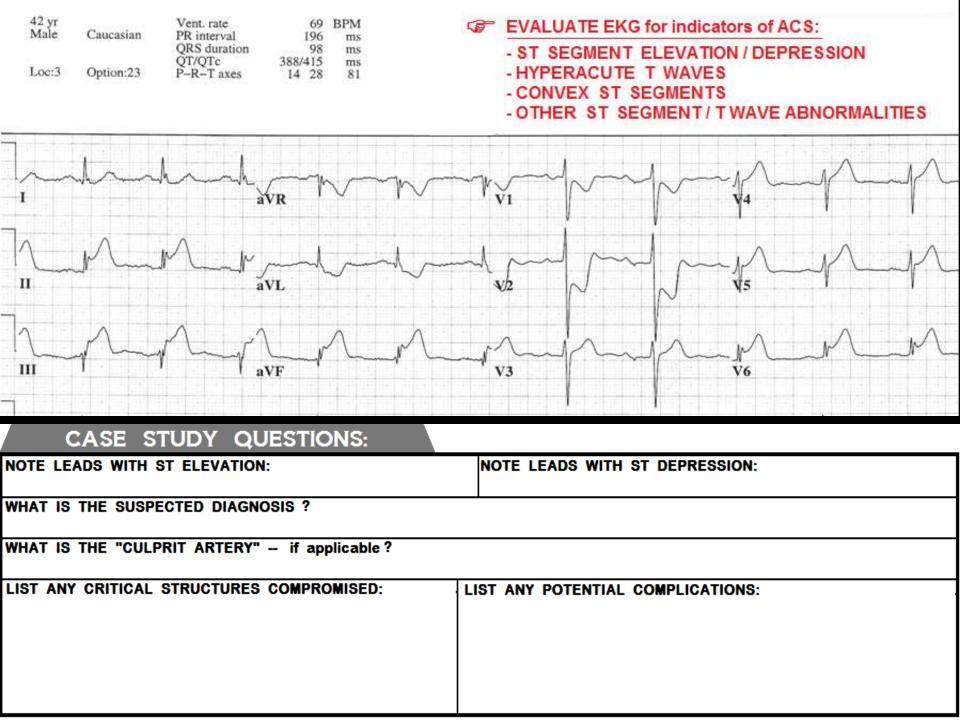
NORMAL

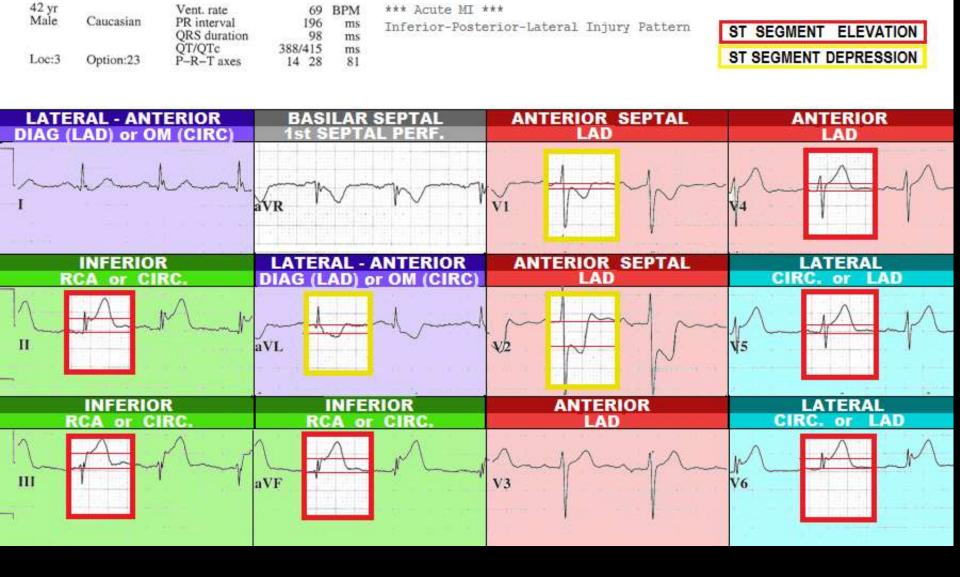
STATUS:



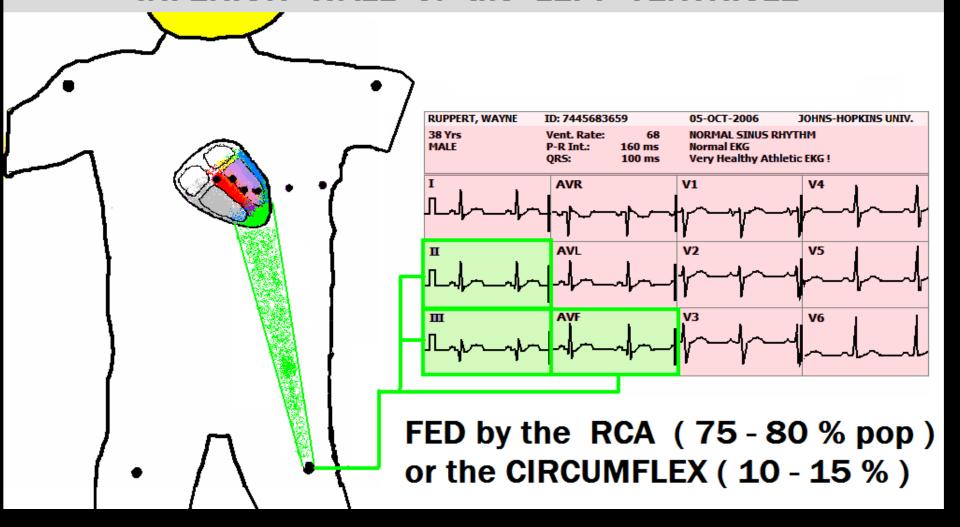


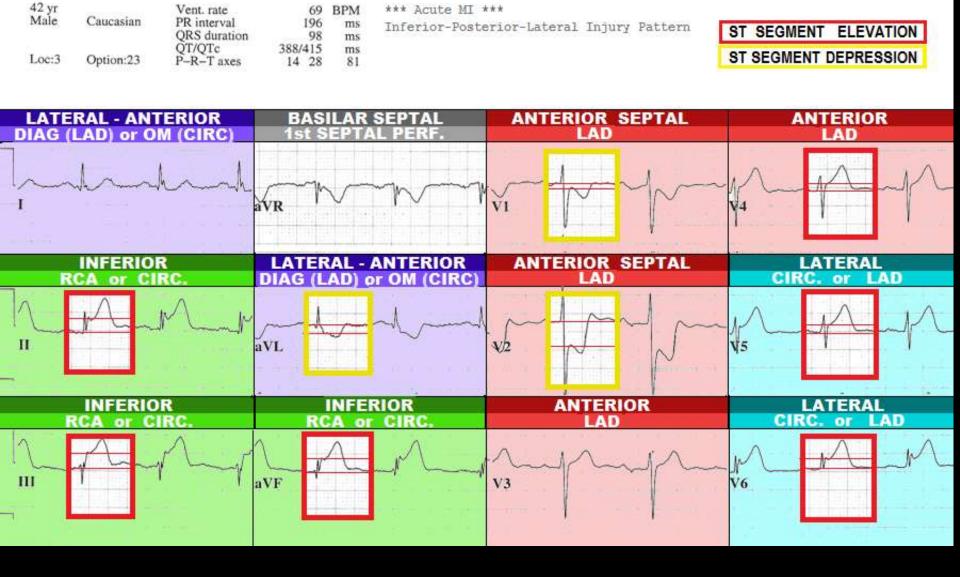
NORMAL



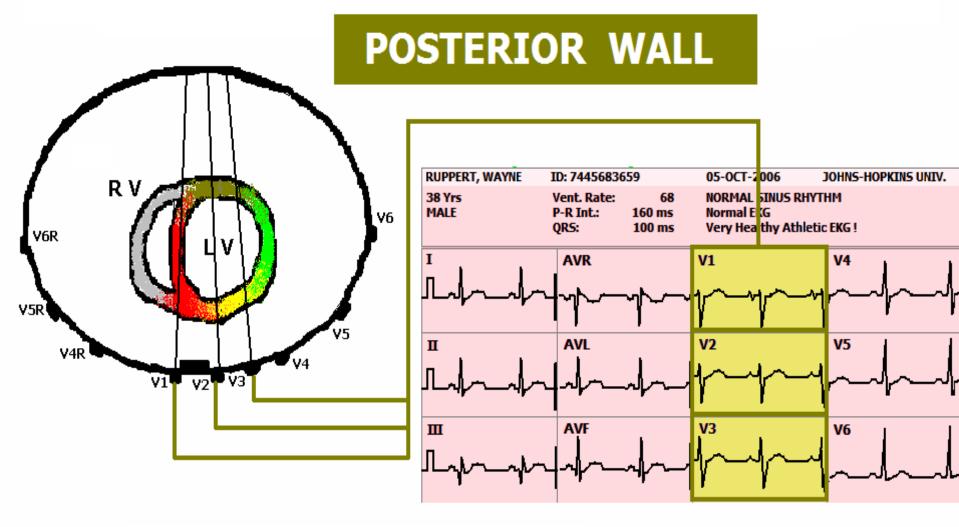


LEADS II, III, and aVF VIEW INFERIOR WALL of the LEFT VENTRICLE





LEADS V1 - V3 view the

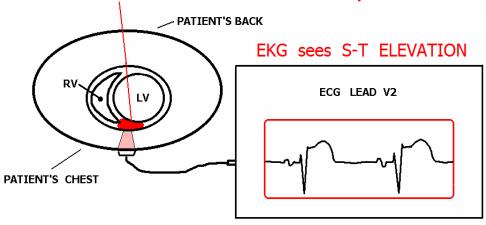


via RECIPROCAL CHANGES.

HOW EKG VIEWS INDICATIVE CHANGES

EXAMPLE:

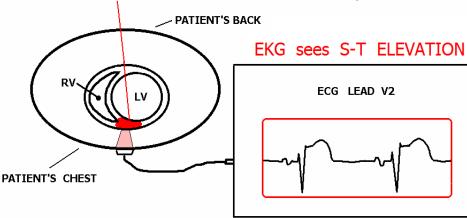




HOW EKG VIEWS INDICATIVE CHANGES

EXAMPLE:

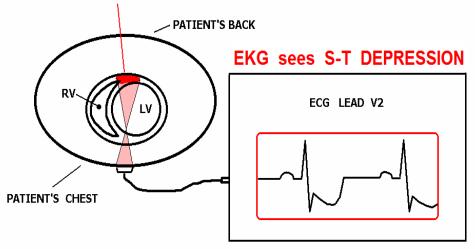
AREA OF ACUTE INFARCTION - ANTERIOR/SEPTAL

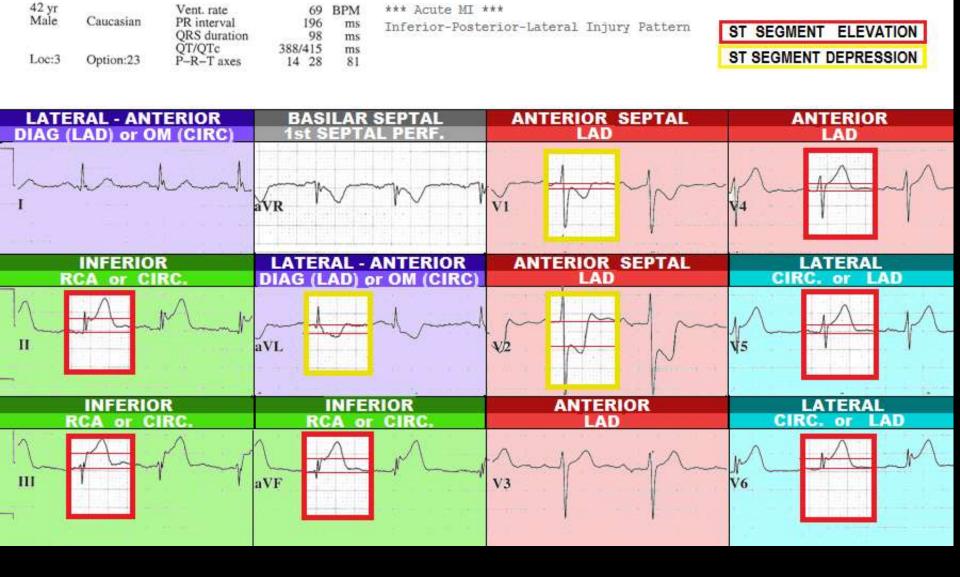


HOW EKG VIEWS RECIPROCAL CHANGES

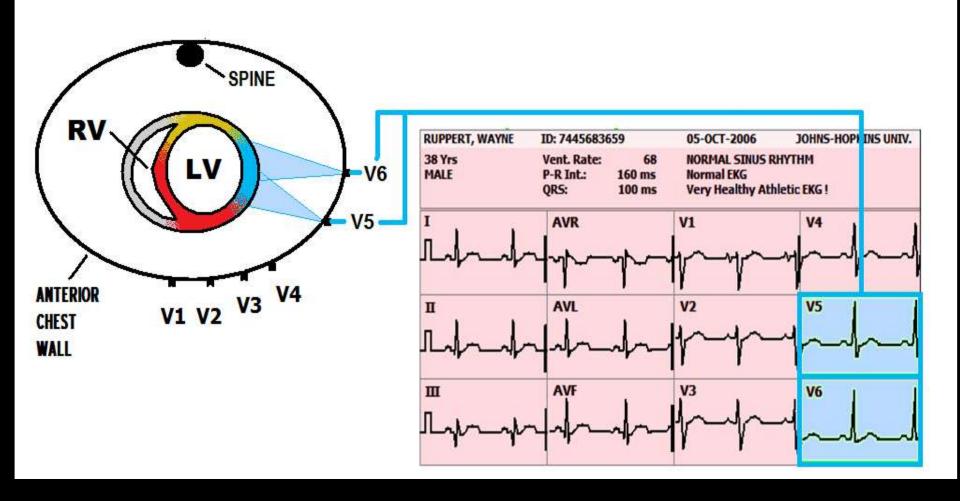
EXAMPLE:

AREA OF ACUTE INFARCTION - POSTERIOR WALL



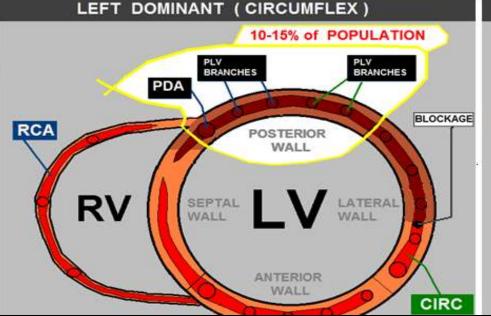


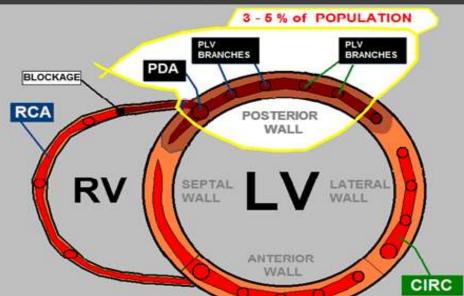
V5 - V6 VIEW THE LATERAL WALL of the LEFT VENTRICLE



INDICATIONS for 18 Lead ECG include:

- INFERIOR WALL MI
- ST Depression in LEADS V1-V4

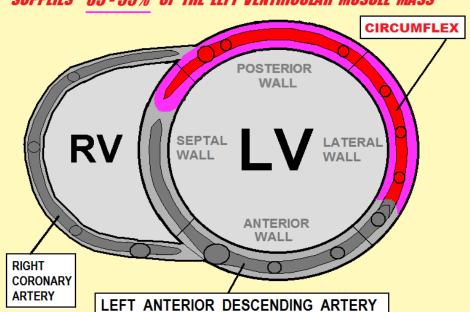




"EXTREME RIGHT DOMINANT" RCA

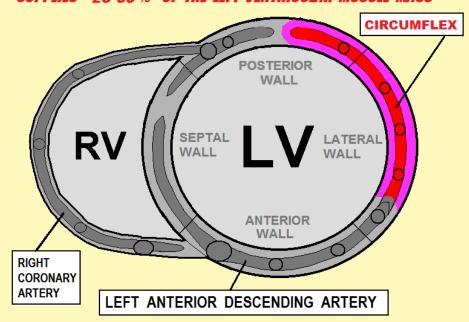
The DOMINANT CIRCUMFLEX ARTERY...

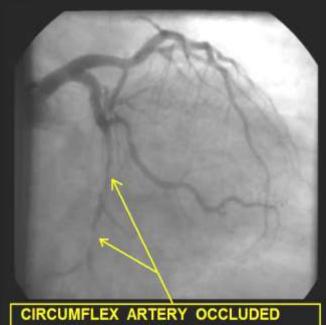
SUPPLIES 35 - 55% OF THE LEFT VENTRICULAR MUSCLE MASS



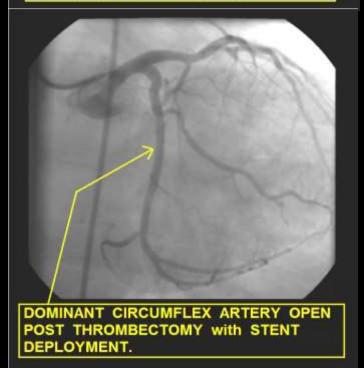
The NON - DOMINANT CIRCUMFLEX ARTERY

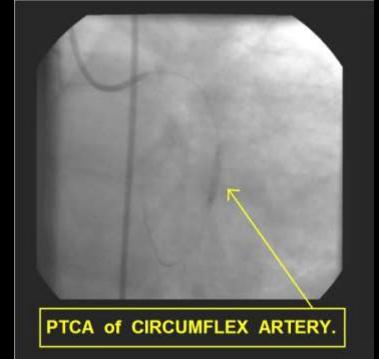
SUPPLIES 25-30 % OF THE LEFT VENTRICULAR MUSCLE MASS





with significant THROMBUS.





LVEF = 4.3%

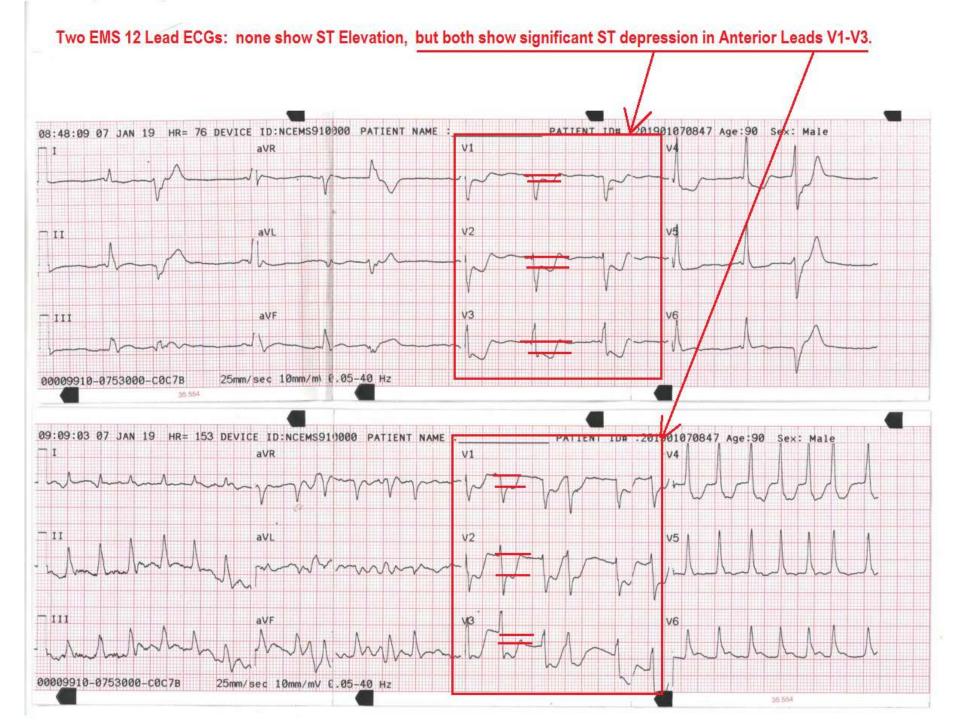
LVEF = 4.3%

Dia Area = 11.0 cm² Sys Area = 8.7 cm² Eject Frec = 4.3%

Dia Volume = 27.7 mi Sys Volume = 16.8 mi Stroke Volume = 11.9 mi

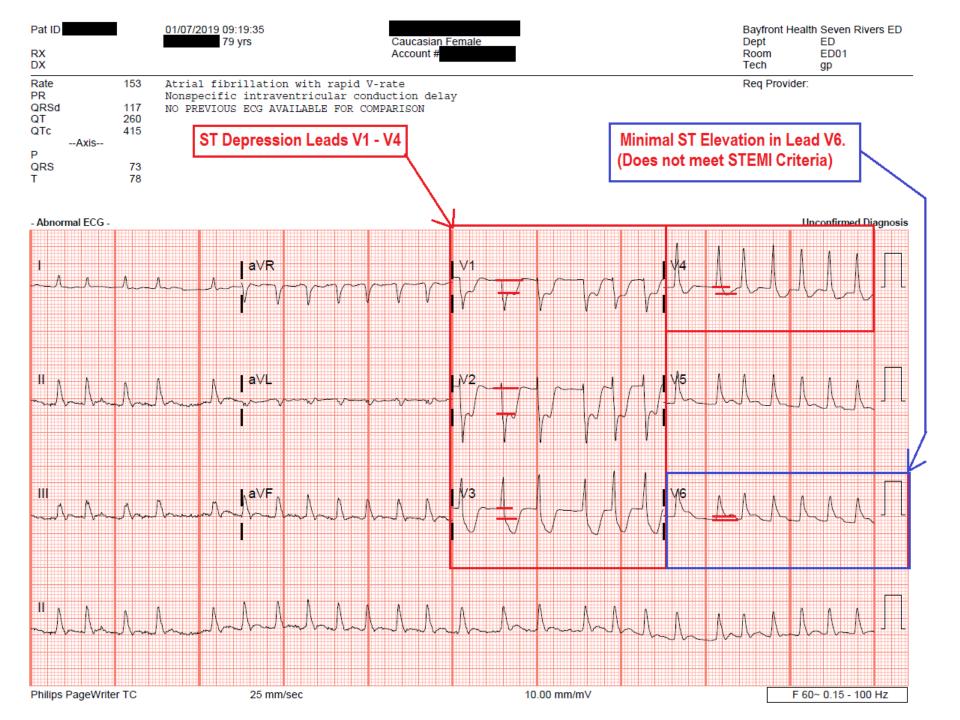
Case Study- January 2019

- 79 y/o female complaining of "L arm pain, and minimal chest pain"
- EMS 12 Lead ECGs show ST Depression in Anterior Leads V1-V4. There is NO ST Elevation.....



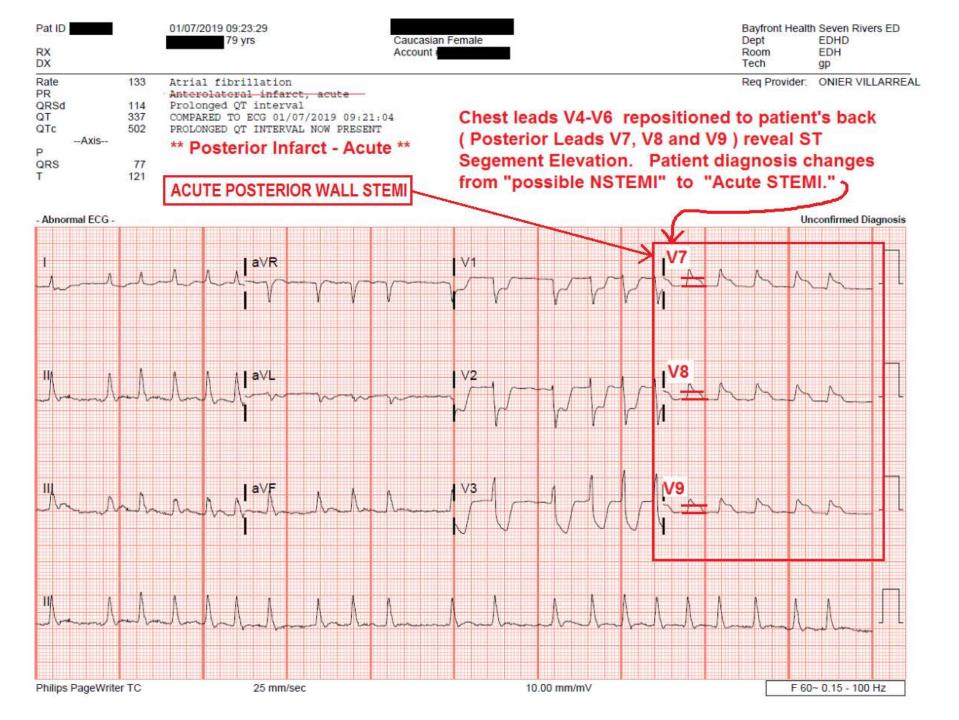
Initial Exam in ED

 Upon arrival in ED, 12 Lead ECG confirmed EMS findings: ST Depression in Leads V1-V4.



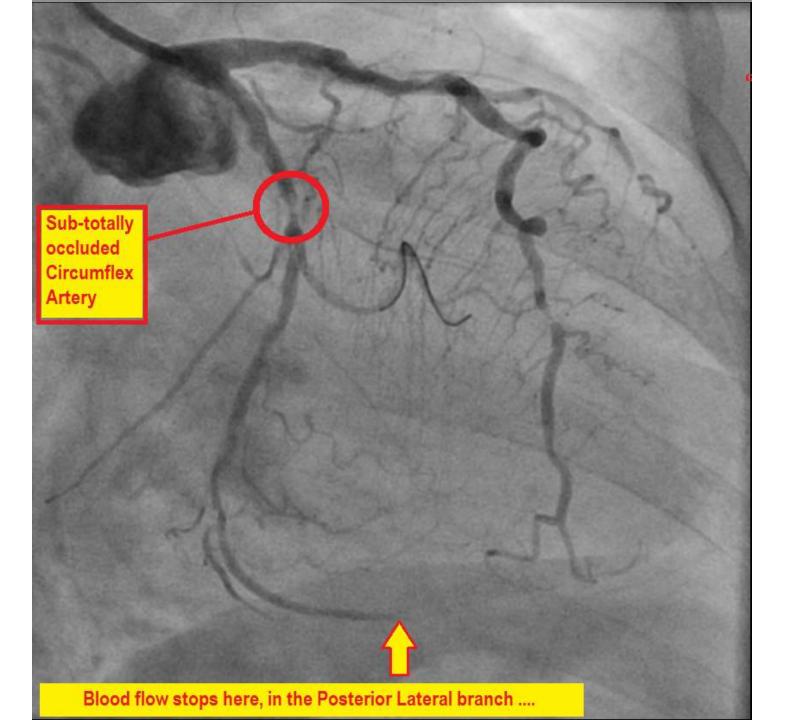
Causes of ST Depression V1-V4

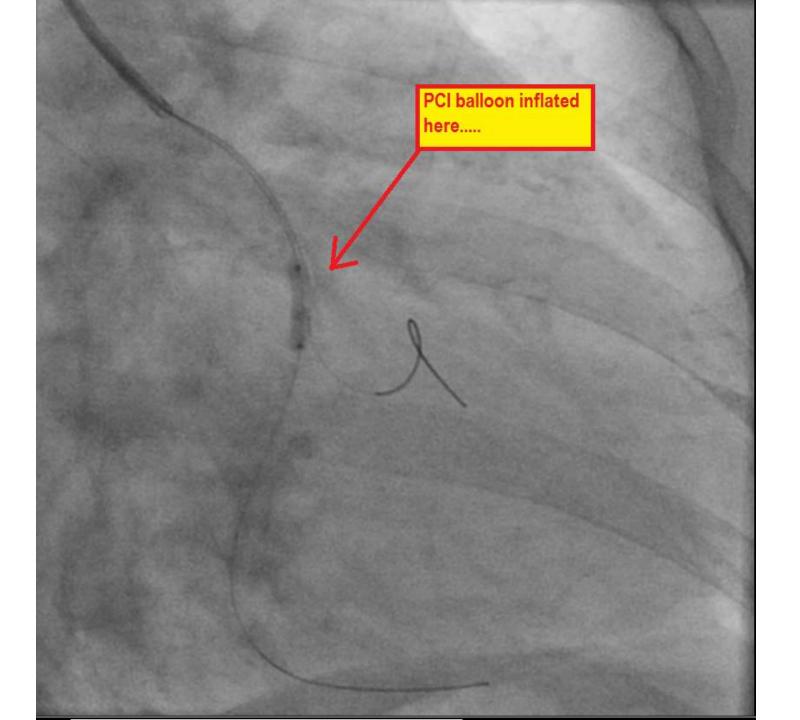
- Anterior Wall ischemia
- Anterior Wall NSTEMI (partial wall thickness myocardial infarction)
- Posterior Wall STEMI

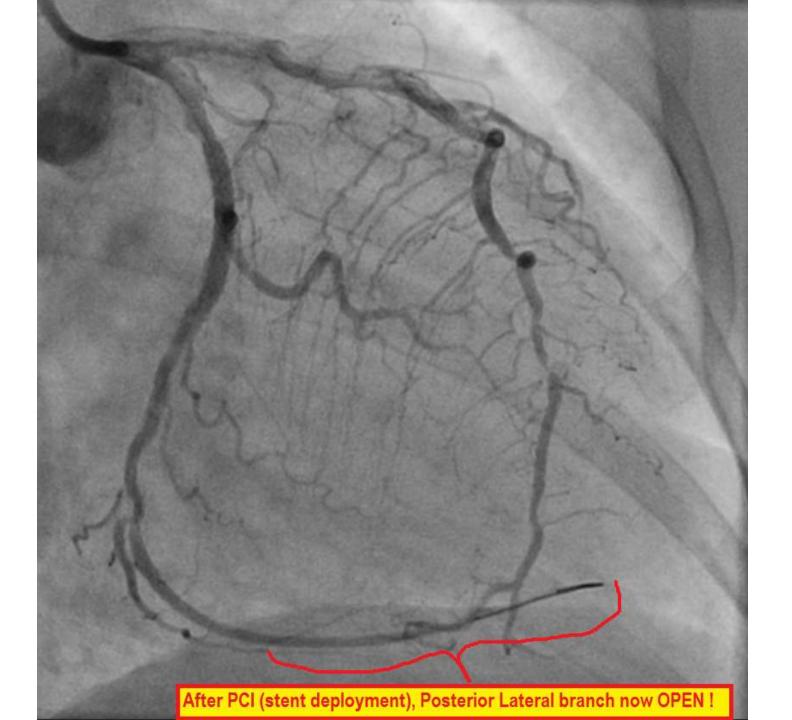


STEMI Alert!

Upon seeing "Significant ST Elevation in TWO or more CONTIGUOUS LEADS, the ED physician diagnosed "Posterior Wall STEMI," a STEMI Alert was issued, and the patient was taken immediately to the cardiac cath lab, where the following images were obtained......







SUMMARY

- Whenever ST Depression is noted in Anterior Leads (V1-V4), it could indicate that Acute Posterior Wall STEMI is present.
- To rule-out Posterior Wall STEMI, a "posterior lead ECG" (V7 – V9) must be obtained.
- In THIS CASE, Posterior Wall STEMI was diagnosed via Posterior Lead ECG.
- STEMI Alert was issued, with a Door-to-PCI time of 53 minutes.

YOU MADE IT !!!

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My top two reasons for giving everything in life the best I have to offer.