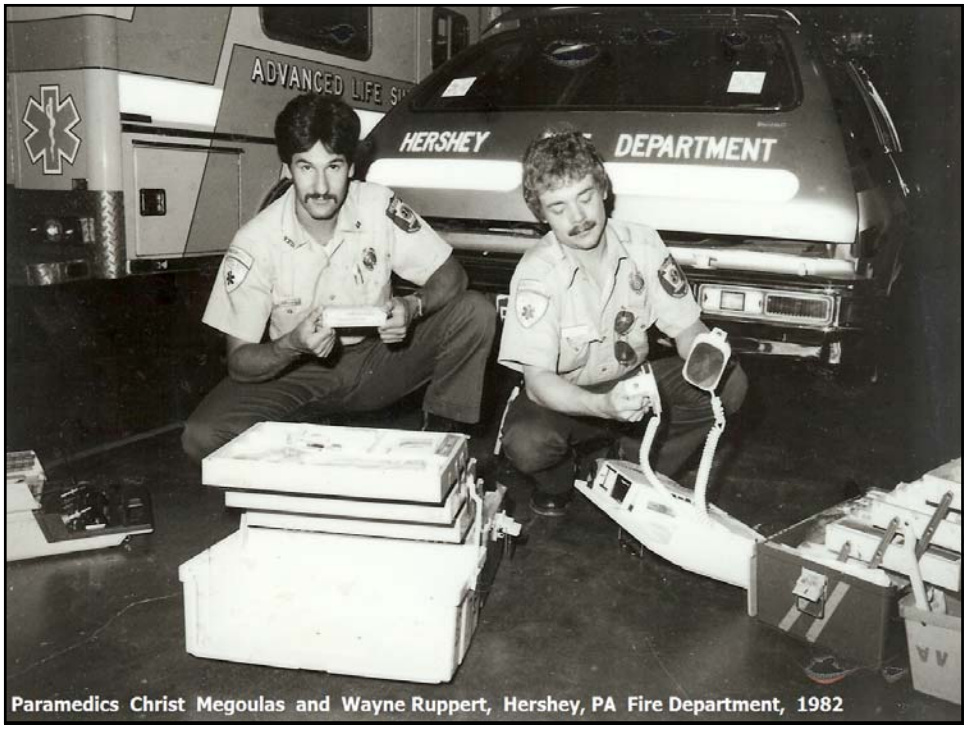
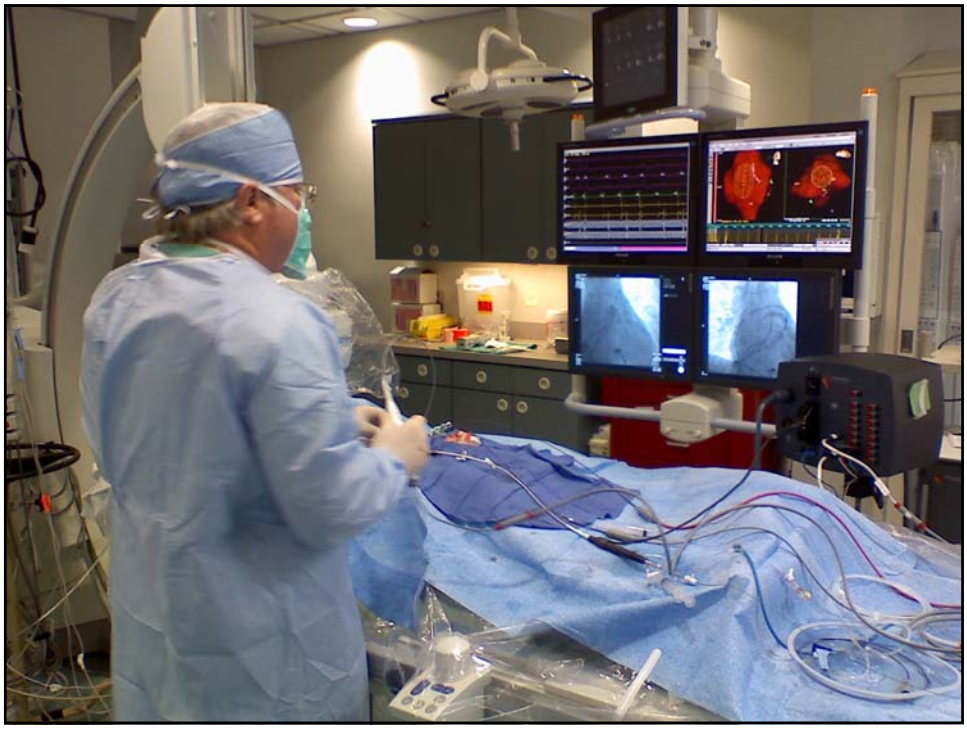
ECG Identification of LETHAL CONDITIONS

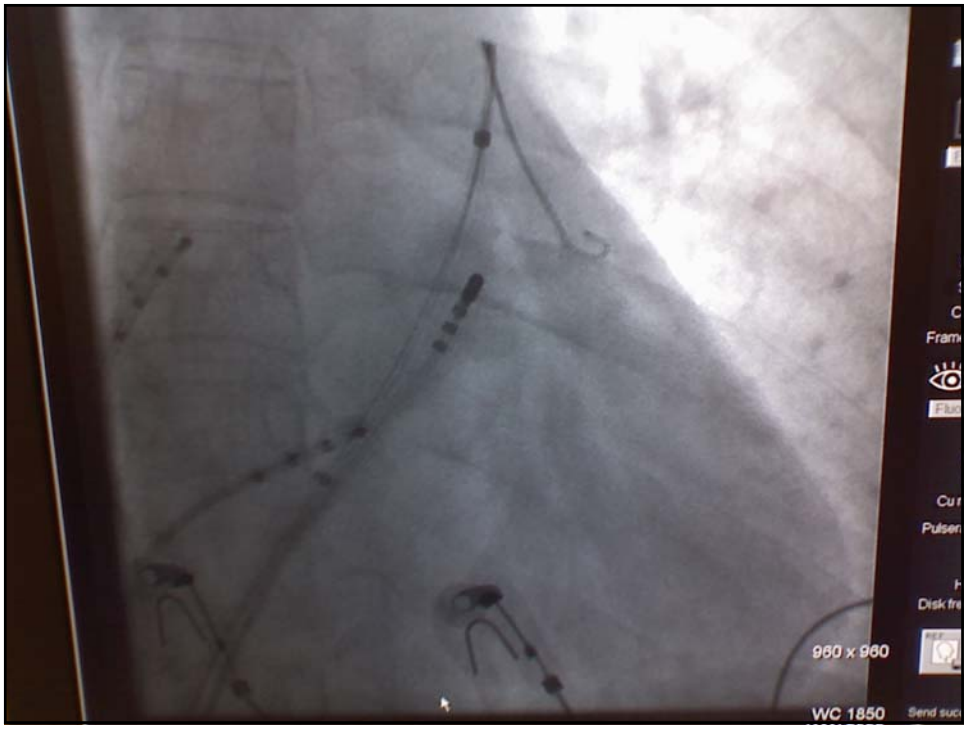
WAYNE W RUPPERT, CVT, CCC, NREMT-P

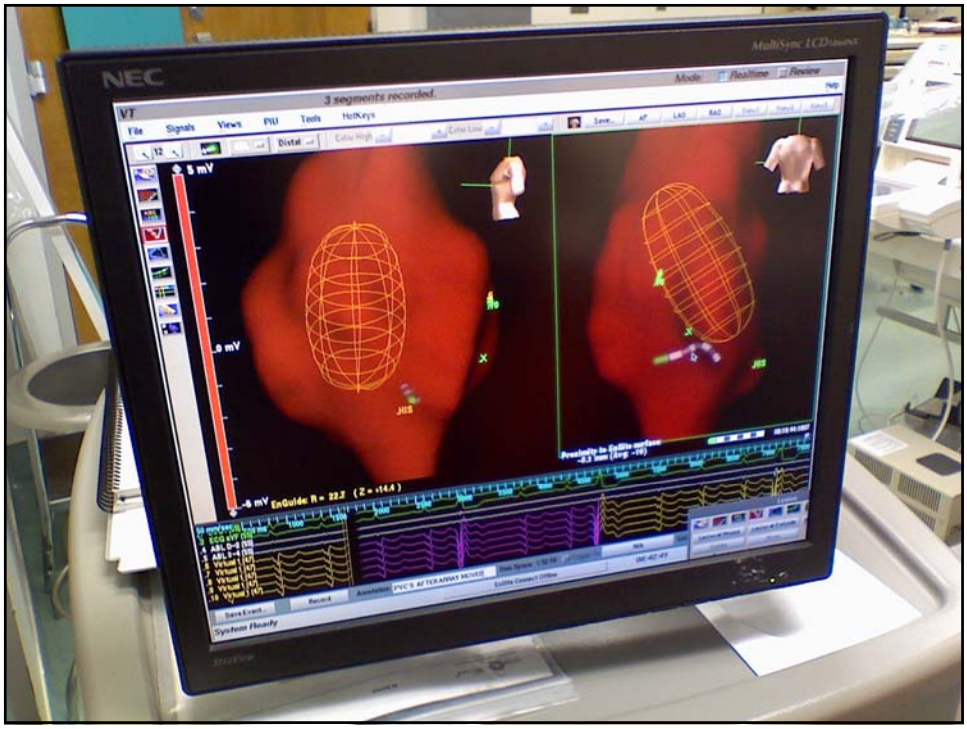
Cardiovascular Clinical Coordinator
Cardiac Data Specialist
Interventional CV/EP Technologist
Bayfront Health Dade City
Dade City, FL

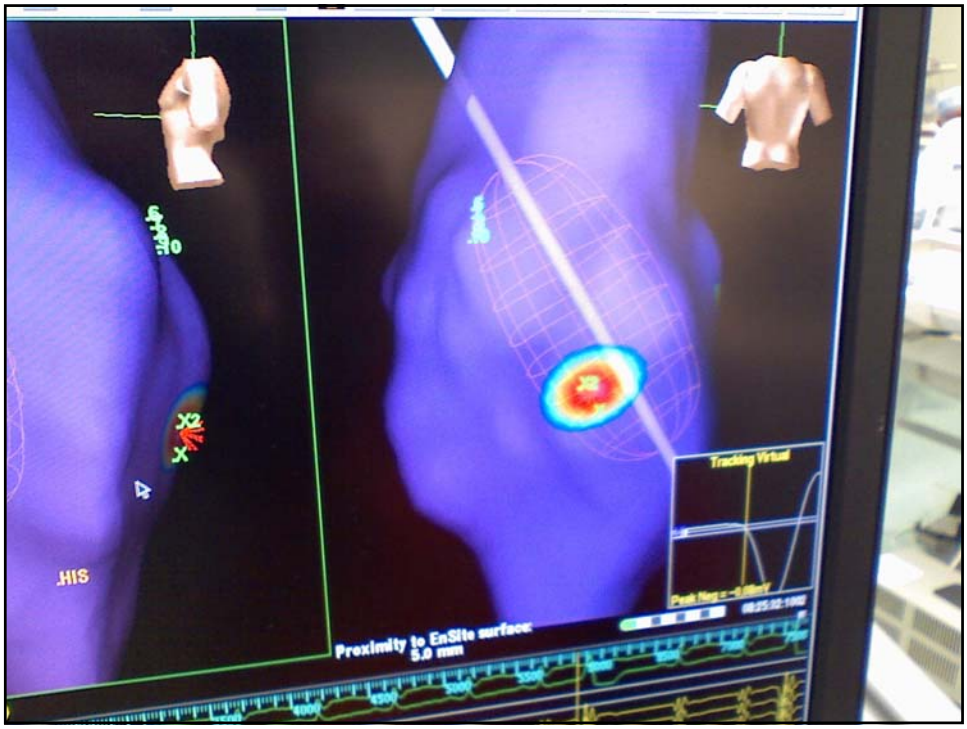


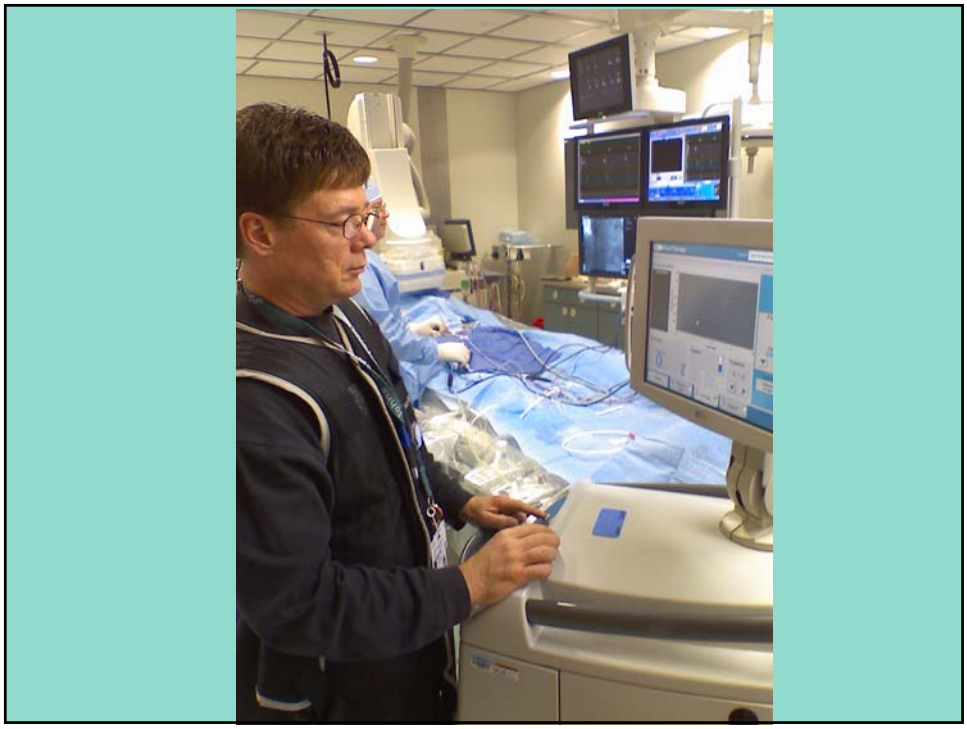
Paramedics Christ Megoulas and Wayne Ruppert, Hershey, PA Fire Department, 1982




960 x 960
WC 1850


NEC
3 segments recorded
File
Signals
Views
PIU
Tools
HotKeys
5 mV
0 mV
-5 mV
System Ready


Tracking Virtual
Proximity to EnSite surface:
5.0 mm



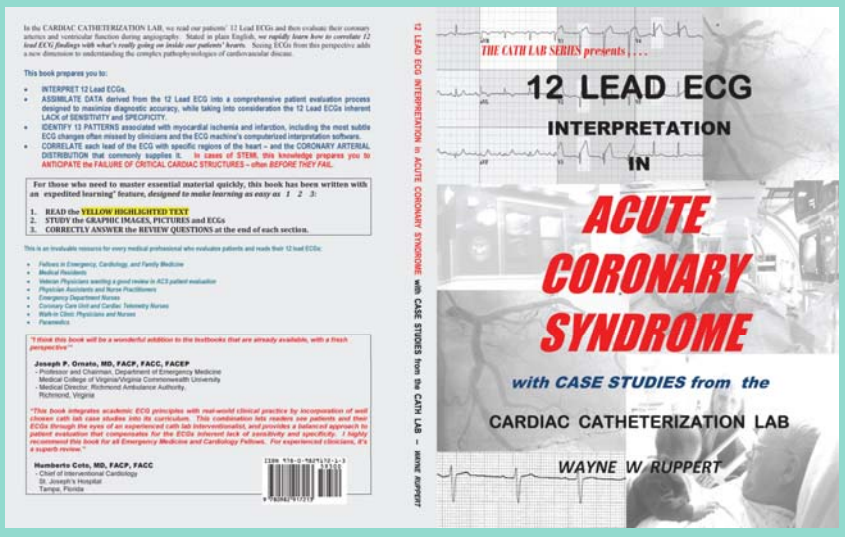
Wayne Ruppert bio:

2012 – Present: Bayfront Health Dade City
Cardiovascular Clinical Coordinator
Stroke Coordinator
Principal Investigator, "Simple Acute Coronary Syndrome (SACS Risk Stratification Score – Scientific Validation Study and Comparison to Modified TIMI and HEART ACS Risk Stratification Scores, NIH #NCT

1994 – 2012: St Joseph's Hospital, Tampa, FL
Interventional Cardiovascular Technologist
Cardiac Electrophysiology Technologist
12 Lead ECG Instructor, Education Department

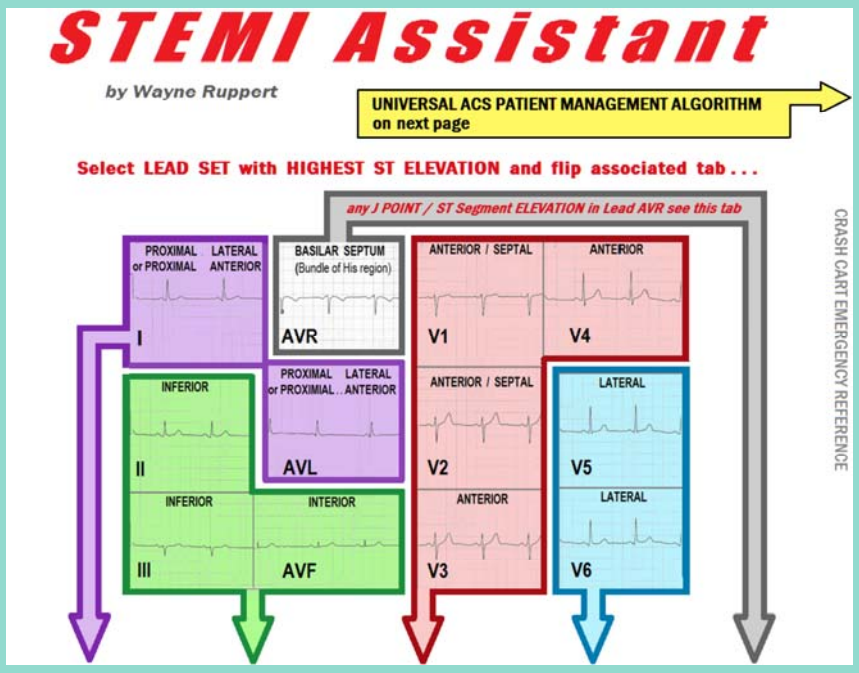
2010: Author, Editor, "12 Lead ECG Interpretation in Acute Coronary Syndrome with Case Studies from the Cardiac Catheterization Lab," 310 page textbook marketed by Ingram Book Company.

-1982 – present, Paramedic (National Registry, Pennsylvania, Florida)
-1982 - present, AHA ACLS Instructor
-1988 – present, AHA PALS Instructor



www.TriGenPress.com
www.ECGtraining.org

BarnesandNoble.com
Amazon.com



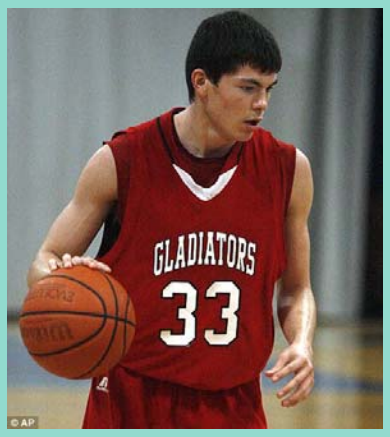
what this class is all about . . . . . .

High School Athlete Dies After Collapsing AtPractice

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



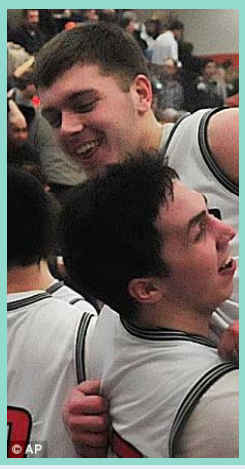
By DAILY MAIL REPORTER
UPDATED: 12:03 EST, 14 March 2011

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 |

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."***

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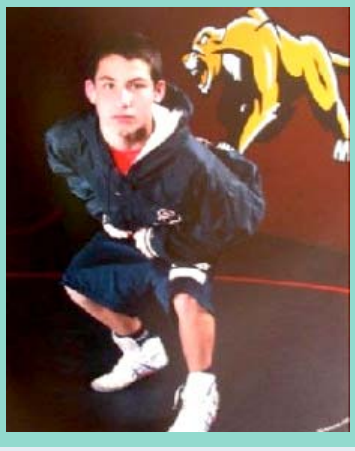
High school quarterback Reggie Garrett threw his second touchdown pass of the night, walked off the field, and collapsed from sudden cardiac arrest. He died in the ambulance on the way to the hospital in West Orange, Texas. In the news coverage following Garrett's death, Dallas station WFAA.com

urged cardiac screening for high school athletes.

Zachary Shrah, 16
High school football player Zachary Schrah collapsed and died of sudden cardiac arrest 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 ECGs 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 electrocardiogram (EKG) screening for other students at Eric's San Diego area high school.

In 2005, Chicago conservationist and wildlife educator Max Schewitz died of sudden cardiac arrhythmia. Since then, the Max Schewitz Foundation, created by his parents, has provided freeelectrocardiograms (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 suffered sudden cardiac arrest 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, public access defibrillator (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.***

" It's the tragic stories what we read about in the news . . . . . Happening SOMEWHERE ELSE

. . . . . ***not HERE."***

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 during a pre-season workout.

"This is mind blowing," said Ben Bromley, the junior varsity and assistant varsity basketball coach at Armwood.

He confirmed Meeks' death Saturday afternoon but said doctors could not provide answers about what killed Meeks.

Jeremy Twining, age 23
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.

Sad stories, avoidable deaths?

Every day, 19 kids in American die of sudden cardiac arrest, from toddlers to college athletes. How many of these lives might be saved if communities made automated external defibrillators **(AEDs) readily available?**

How many would be spared if their families, schools, and sports organizations were aware of the benefits of **cardiac screening for heart defects?**

You can't help but ask, when you read these sad stories, were these avoidable deaths?

Don't take a chance. No excuses. PLEASE get an **AED in your child's school and on the athletic field.**

Sad stories, avoidable deaths?

Every day, 19 kids in American die of sudden cardiac arrest, from toddlers to college athletes. How many of these lives might be saved if communities made automated external defibrillators (AEDs) readily available?

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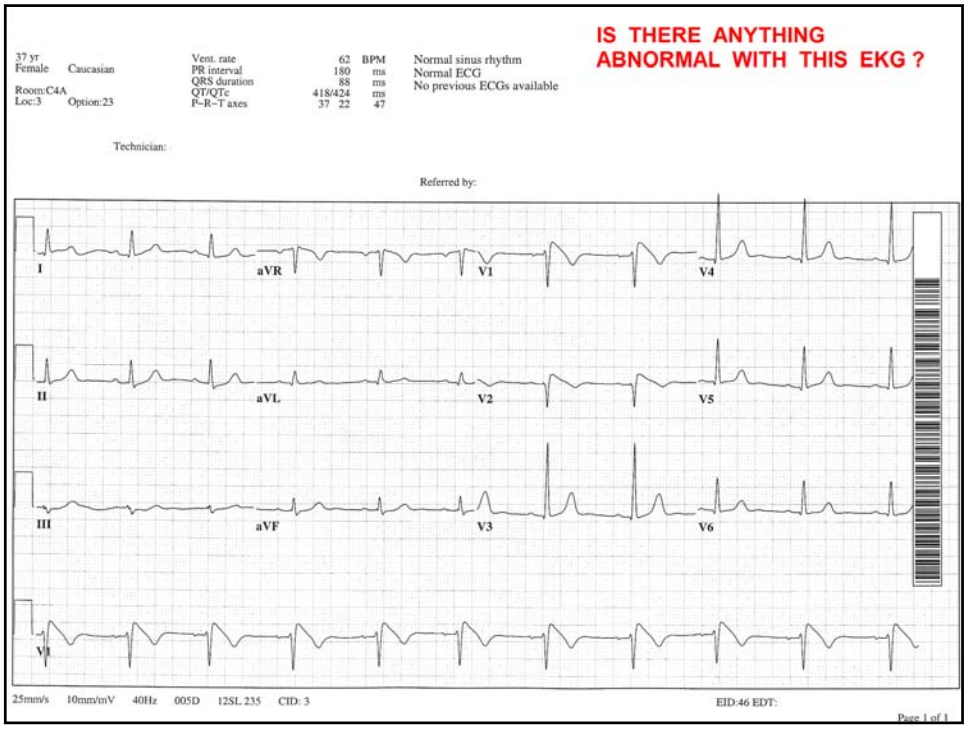
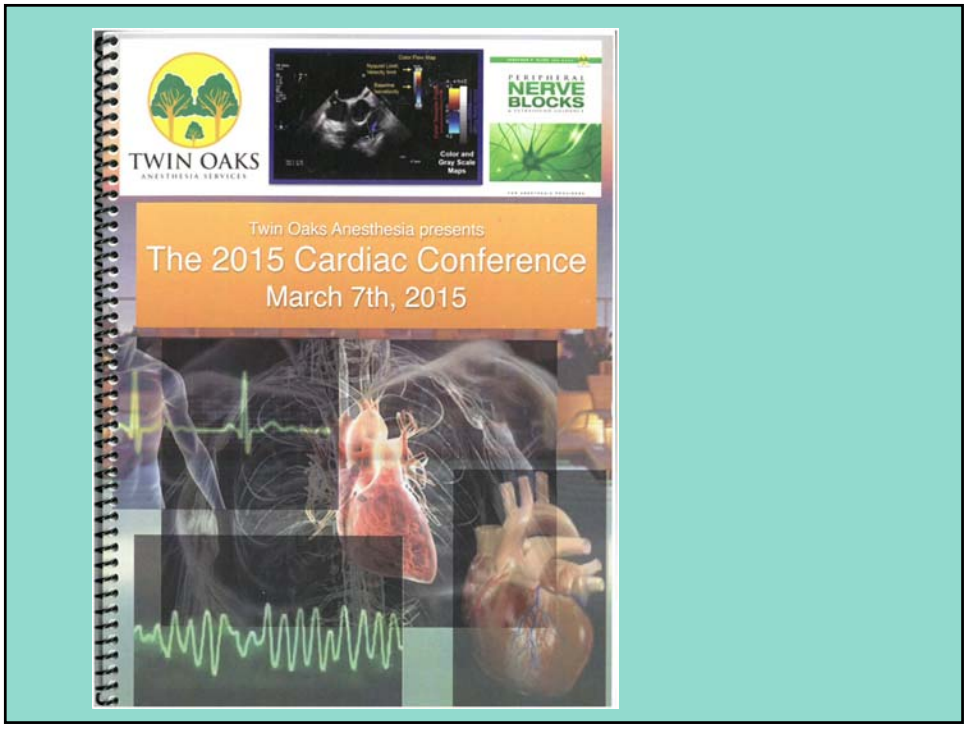
Don't take a chance. No excuses. PLEASE get an AED in your child's school and on the athletic field.

*Let's intervene BEFORE they have the need for an AED! . . . . . Let's support ECG and Echocardiographic screening of our kids . . . . . and as Health Care Professionals: **"Let's ALL be AWARE of the ECG Indicators of the CONDITIONS THAT CAUSE SUDDEN CARDIAC DEATH !!"***


IS THERE ANYTHING ABNORMAL WITH THIS EKG ?
37 yr
Female Caucasian
Room:C4A
Loc:3 Option:23
Vent. rate 62 BPM
PR interval 180 ms
QRS duration 88 ms
QT/QTc 418/424 ms
P-R-T axes 37 22 47
Normal sinus rhythm
Normal ECG
No previous ECGs available
Technician:
Referred by:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
VI
25mm/s 10mm/mV 40Hz 005D 12SL 235 CID: 3
EID:46 EDT:
Page 1 of 1


TWIN OAKS
ANESTHESIA SERVICES
Color and Gray Scale Maps
PERIPHERAL
NERVE
BLOCKS
Twin Oaks Anesthesia presents
The 2015 Cardiac Conference
March 7th, 2015

Course Syllabus:

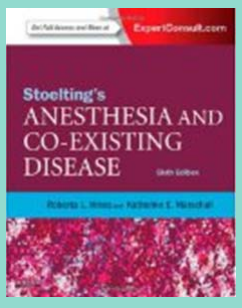
- The Normal ECG
- Brugada Syndrome
- Long QT Syndrome (LQTS)
- Wolff-Parkinson-White Syndrome (WPW)
- Arrhythmogenic Right Ventricular Dysplasia (ARVD)
- Pericarditis / Myocarditis
- Hypertrophy
  - Hypertrophic Cardiomyopathy (HCM)
  - Valvular Disorders
  - Cor Pulmonale

Course Reference Sources:

JACC Journals

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

J Am Coll Cardiol. 2014;64(22):e77-e137. doi:10.1016/j.jacc.2014.07.944



Stoelting's Anesthesia and Co-Existing Disease, 5th Edition

EXPERT CONSULT - ONLINE AND PRINT
By Roberta L. Hines, MD and Katherine E. Marschall, MD

History

PATIENT or FAMILY HISTORY of:

- Sudden Death of apparently healthy individuals?
- Abnormal ECG
- Long QT or Brugada Syndrome
- Hypertrophic Cardiomyopathy
- Coronary Artery Disease

DOES PATIENT HAVE or EXPERIENCE:

- Chest Pain / Pressure / Tightness
- Abnormal shortness of breath ?
- Palpitations, “heart racing – skipping beats” ??
- Syncope, lightheadedness, passing out ?

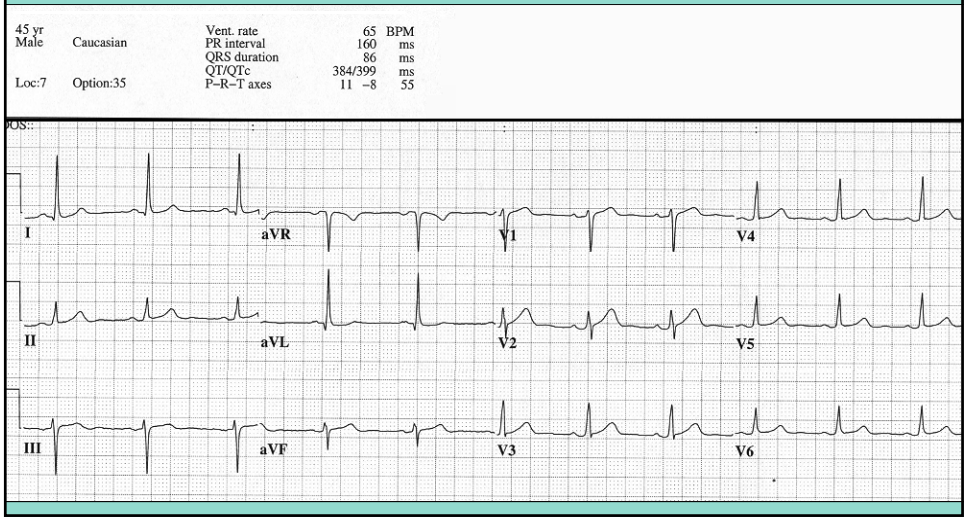
CV Diagnostic Tests:

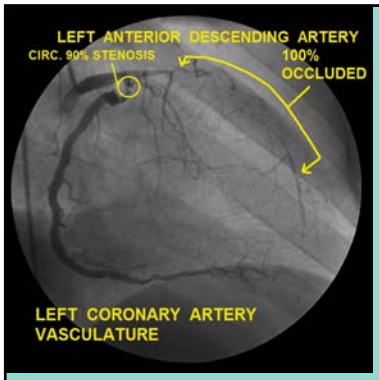
- 12 Lead ECG
- Echocardiogram
- Stress Test
- CT / MRI
- Cardiac Catheterization

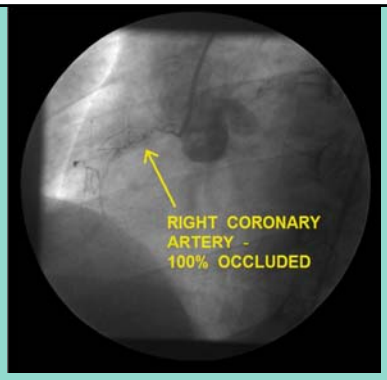
3 QUICK SLIDES ON CORONARY ARTERY DISEASE

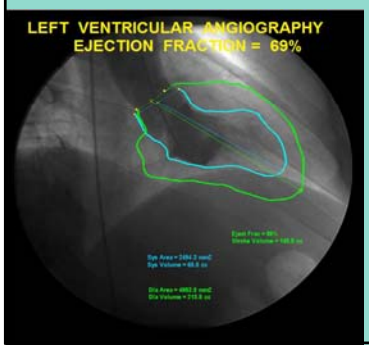
The need for PREOPERATIVE CAD Risk Factor Assessment . . . .

Does the NORMAL ECG rule out OBSTRUCTIVE Coronary Artery Disease ??





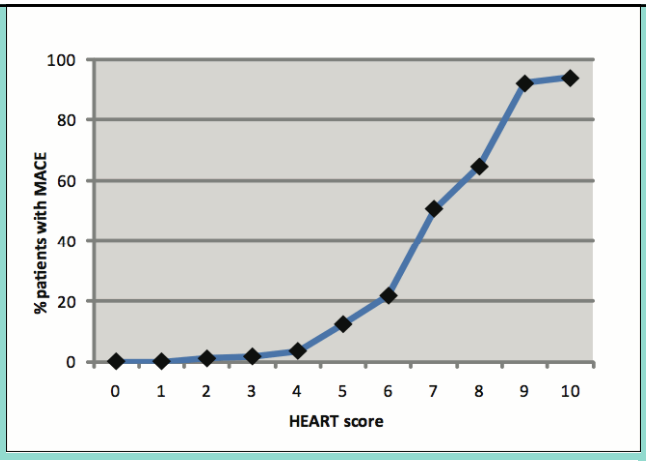




"The WORST coronary vasculature I have seen in nearly 20 years -- an estimated 12,000 cases -- in the CATH LAB . . . And this patient's 12 Lead ECG was essentially normal !!"

ACS Risk Stratification: HEART Score

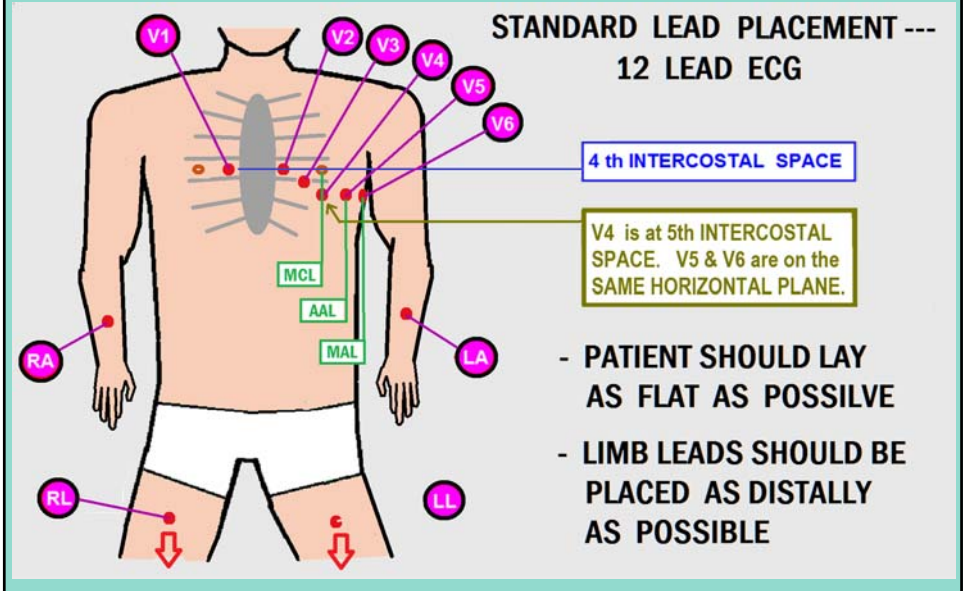
History	Highly suspicious	2
	Moderately suspicious	1
	Slightly/not suspicious	0
ECG	ST Depression	2
	Non-specific repol. Abnormality	1
	Normal	0
Age	65 +	2
	45-65	1
	< 45	0
Risk Factors	3 + Risk Factors –or-- known CAD	2
	1 or 2 Risk Factors	1
	No known Risk Factors	0
Troponin	3X or more normal limit	2
	2 x normal limit	1
	Normal	0



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

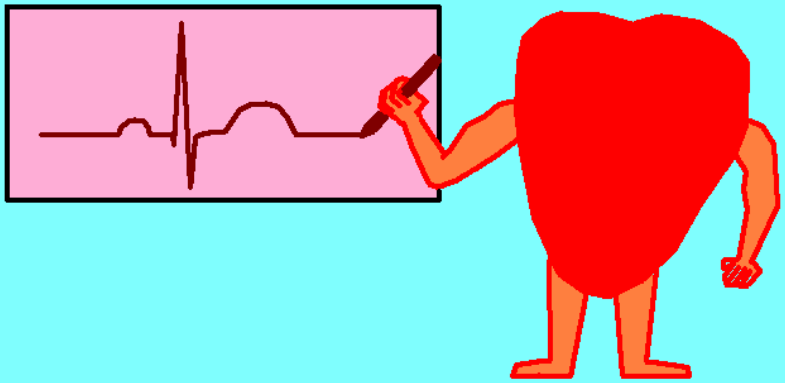
CLASS IIa

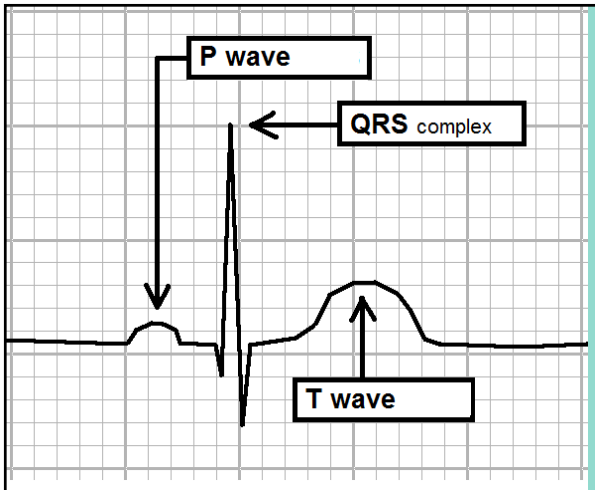
1. A validated risk-prediction tool can be useful in predicting the risk of perioperative MACE in patients undergoing noncardiac surgery (37,114,115). (Level of Evidence: B)



PUTTING IT ALL ON PAPER . . .

WAVEFORMS and INTERVALS . . .





P WAVE =
ATRIAL DEPOLARIZATION

QRS COMPLEX =
VENTRICULAR DEPOLARIZATION
(contracting)

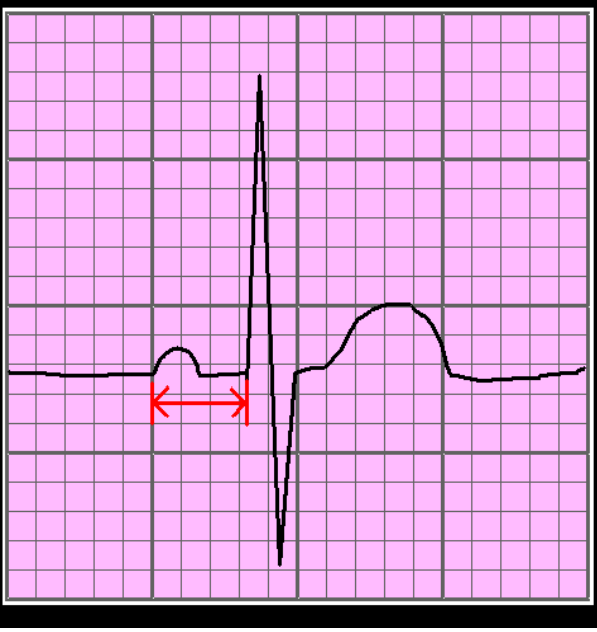
T WAVE =
VENTRICULAR REPOLARIZATION
(recharging)

NORMAL P-R INTERVAL

.12 - .20 SEC

or

120 - 200 mSEC

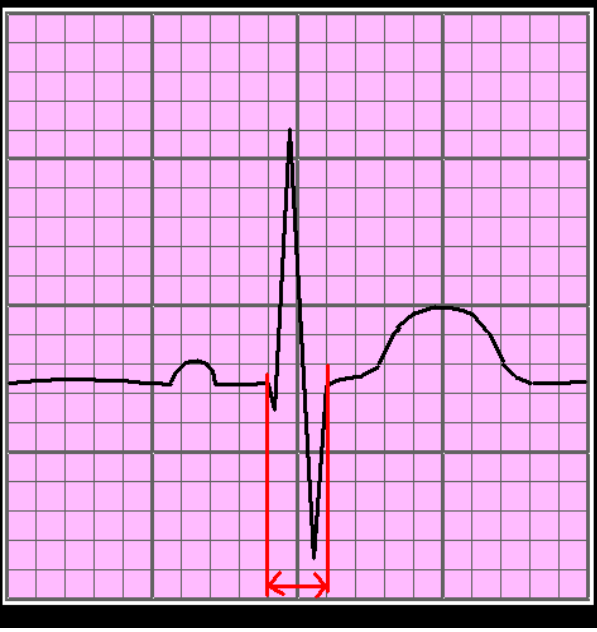


QRS INTERVAL

LESS THAN .12

OR

120 mSEC

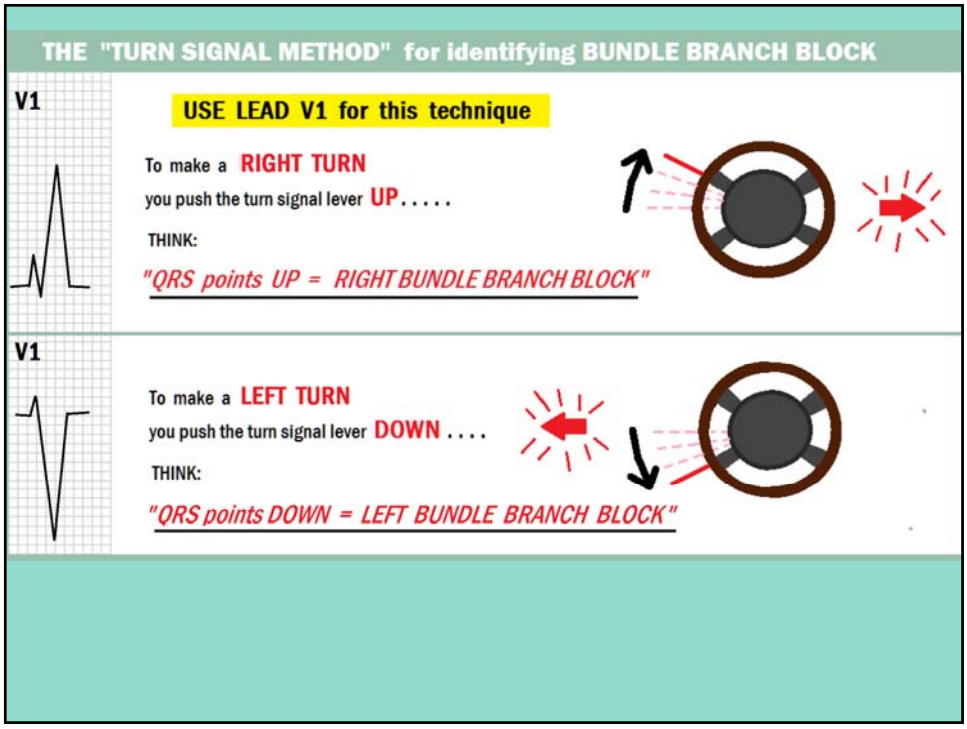


QRS COMPLEX TOO WIDE

WIDER THAN 120 mSEC

THINK:

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



SOME CAUSES OF LEFT BUNDLE BRANCH BLOCK (LBBB)

- CONDUCTION SYSTEM DISEASE
- OLD ANT./SEPTAL MI (NECROSIS TO LBB)
- CARDIOMYOPATHY
- SEVERE L.V.H.
- ACUTE MYOCARDITIS

SOME CAUSES OF RIGHT BUNDLE BRANCH BLOCK (RBBB)

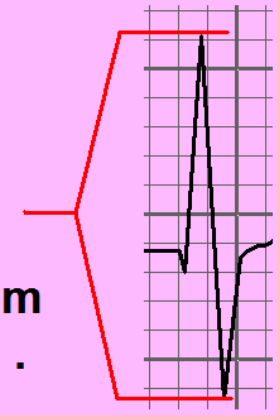
- CONGENITAL VARIATION (IN HEALTHY HEART)
- CONDUCTION SYSTEM DISEASE
- OLD ANT./SEPTAL MI (NECROSIS TO RBB)
- PREVIOUS C.A.B.G. (RBB CUT DURING SURGERY)
- SEVERE R.V.H.
- ACUTE PULMONARY EMBOLUS
- BRUGADA SYNDROME

THE QRS COMPLEX

QRS HEIGHT

is a reflection of the QRS AMPLITUDE.

The NORMAL QRS AMPLITUDE varies from one lead to another . . .



THE QRS COMPLEX

QRS AMPLITUDE

is influenced by:

- age
- physical fitness
- body size
- conduction system disorders
- chamber hypertrophy

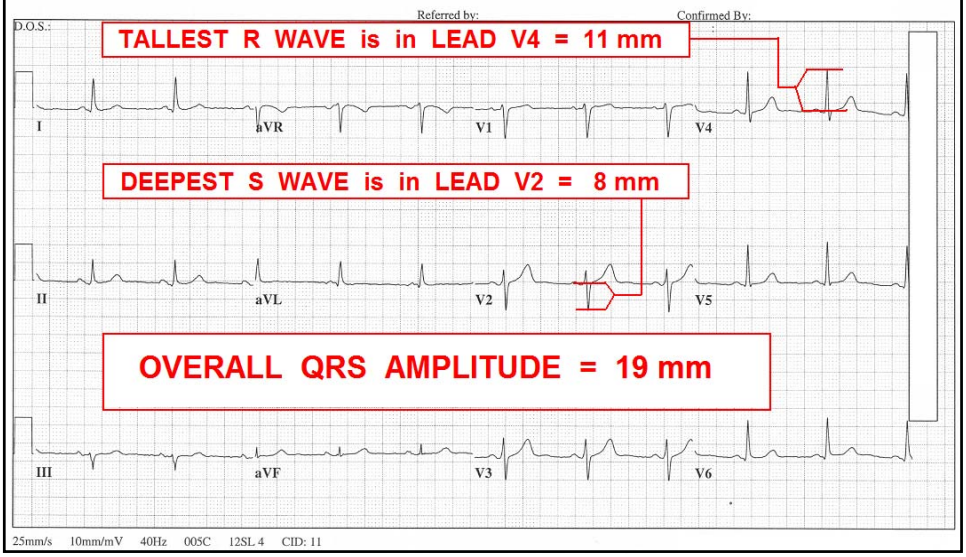
THE QRS COMPLEX

QRS AMPLITUDE

is measured by finding the TALLEST POSITIVE DEFLECTION (R WAVE) and the DEEPEST NEGATIVE DEFLECTION (S WAVE) on the 12 LEAD EKG and ADDING THE VALUES TOGETHER

MEASURING THE "OVERALL QRS AMPLITUDE"

Add the SIZE of the TALLEST R WAVE to the SIZE of the DEEPEST S WAVE . . . .



THE QRS COMPLEX

QRS AMPLITUDE

MAXIMUM NORMAL VALUES are difficult to define due to differences in PATIENT AGE, BODY SIZE, and FITNESS.

HOWEVER A GENERAL VALUE GUIDELINE IS: 3.0 mV
(30 mm on normally calibrated EKG)

OVERALL QRS AMPLITUDE TOO HIGH:

(GREATER THAN 3.0 mV / 30 mm)

THINK:

- VENTRICULAR HYPERTROPHY

THE QRS COMPLEX

QRS AMPLITUDE

CRITERIA FOR MINIMUM AMPLITUDE:

Abnormally LOW QRS VOLTAGE occurs when the OVERALL QRS is:

≤ 0.5 mV IN ANY LIMB LEAD

– and –

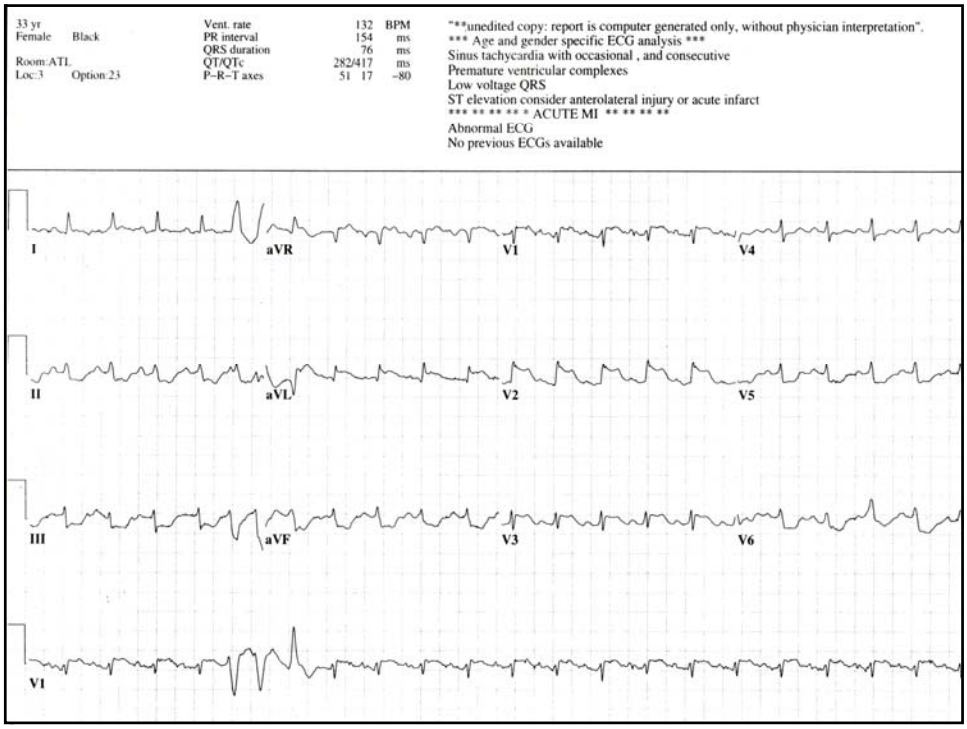
≤ 1.0 mV IN ANY PRECORDIAL LEAD

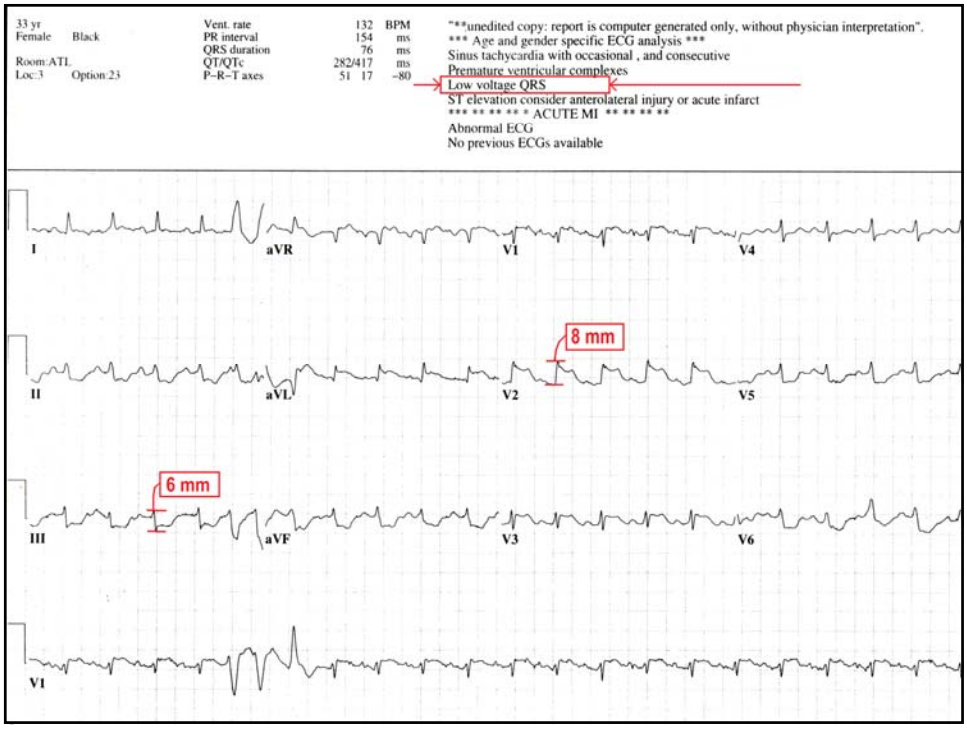
OVERALL QRS AMPLITUDE TOO LOW:

(VERTICAL QRS SIZE)

THINK (in absence of obvious OBESITY):

- MYOCARDITIS / CONSTRICTIVE PERICARDITIS
- EFFUSIONS / TAMPONADE
- COPD c HYPERINFLATION
- AMYLOIDOSIS (abnormal protein accumulation in organs)
- SCLERODERMA (abnormal hardening of skin)
- HEMACHROMOTOSIS (excessive iron buildup in blood / organs)
- MYXEDEMA (thyroid disorder)


33 yr
Female Black
Room:ATL
Loc:3 Option:23
Vent. rate 132 BPM
PR interval 154 ms
QRS duration 76 ms
QT/QTc 282/417 ms
P-R-T axes 51 17 -80
"**unedited copy: report is computer generated only, without physician interpretation".
*** Age and gender specific ECG analysis ***
Sinus tachycardia with occasional , and consecutive
Premature ventricular complexes
Low voltage QRS
ST elevation consider anterolateral injury or acute infarct
*** ** ** ** * ACUTE MI ** ** ** **
Abnormal ECG
No previous ECGs available
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1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Abnormal ECG
No previous ECGs available
I
aVR
V1
V4
8 mm
II
aVL
V2
V5
6 mm
III
aVF
V3
V6
V1

BRUGADA SYNDROME
A DEADLY STEMI Mimic.....

CASE STUDY 18 -- BRUGADA SYNDROME

CHIEF COMPLAINT and SIGNIFICANT HISTORY:
37 y/o FEMALE patient arrives via EMS after being involved in a low speed motor vehicle accident. Per EMS crew, patient was the driver and sole occupant of a car that struck a tree. Patient does not recall accident. Upon further questioning, patient admits to other episodes of syncope and near-syncope. Patient denies feeling any chest pain / pressure or shortness of breath. She states she "felt great" today, until just before the the accident, when she "suddenly felt lightheaded and must have blacked-out."

RISK FACTOR PROFILE:

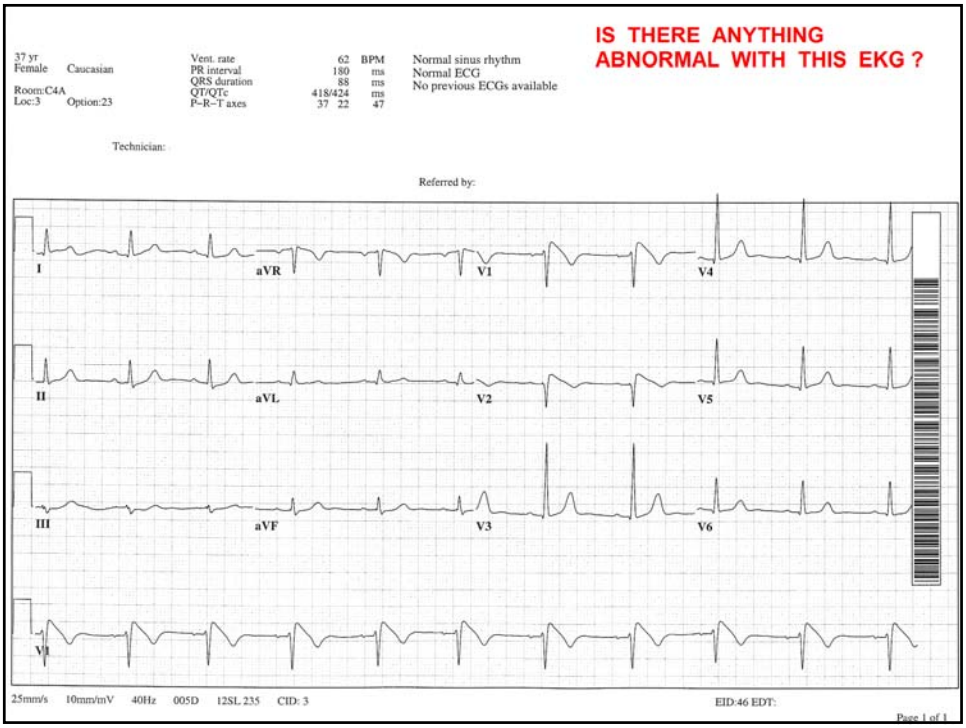
FAMILY HISTORY: MATERNAL AUNT DIED AT AGE 31, UNEXPECTEDLY. WAS RULED AS A "HEART ATTACK." THERE WAS NO PRIOR KNOWN HISTORY OF CAD.

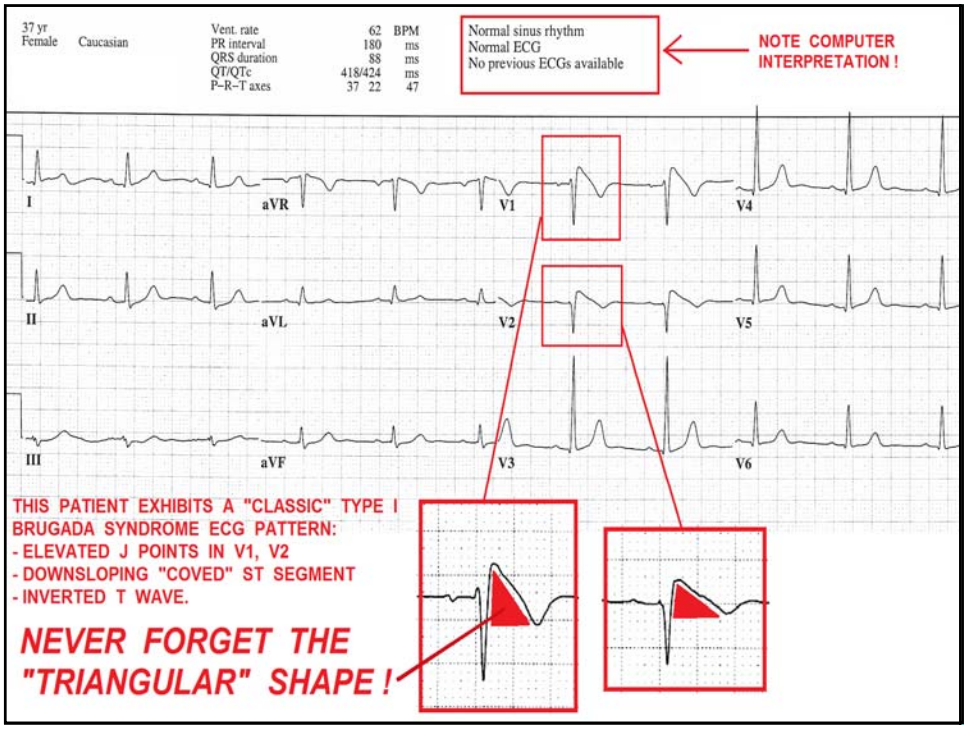
PHYSICAL EXAM: Pt. CAO x 3, skin warm, dry, color normal. Abrasions /contusions on face (airbag deployment). Patient appears to be in excellent physical condition, states she exercises several times per week (aerobics, weight training, swimming).

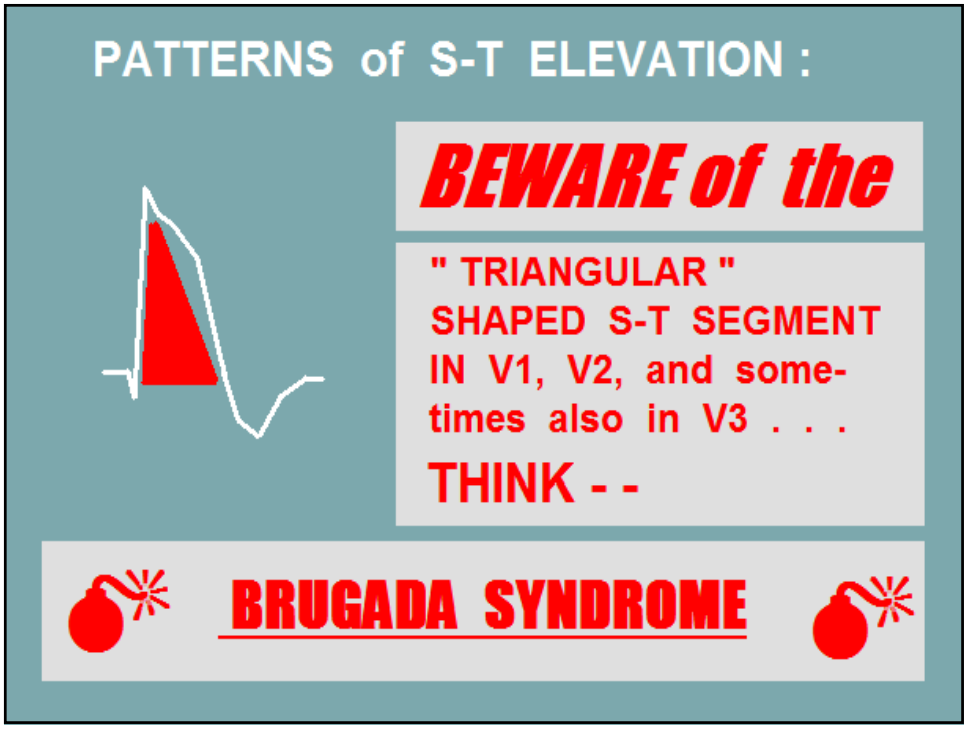
VITAL SIGNS: BP: 112/66, P: , R: 20, SAO2: 100% on room air.

LABS: TROPONIN: < .04 BMP and CBC: all values within normal limits.

IS THERE ANYTHING ABNORMAL WITH THIS EKG ?

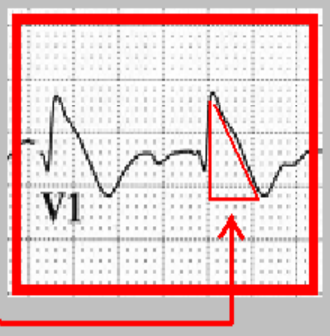



37 yr
Female Caucasian
Vent. rate 62 BPM
PR interval 180 ms
QRS duration 88 ms
QT/QTc 418/424 ms
P-R-T axes 37 22 47
Normal sinus rhythm
Normal ECG
No previous ECGs available
NOTE COMPUTER INTERPRETATION !
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
THIS PATIENT EXHIBITS A "CLASSIC" TYPE I BRUGADA SYNDROME ECG PATTERN:
- ELEVATED J POINTS IN V1, V2
- DOWNSLOPING "COVED" ST SEGMENT
- INVERTED T WAVE.
NEVER FORGET THE "TRIANGULAR" SHAPE !

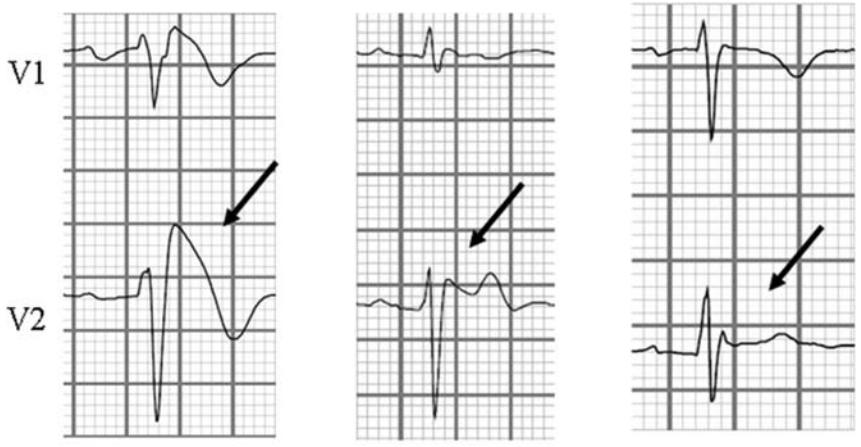

PATTERNS of S-T ELEVATION :
BEWARE of the
" TRIANGULAR " SHAPED S-T SEGMENT IN V1, V2, and some-times also in V3 . . .
THINK - -
BRUGADA SYNDROME

BRUGADA SYNDROME

1. RBBB PATTERN
2. S-T ELEVATION
   V1, V2, possibly V3
3. ATYPICAL "TRIANGLE" SHAPED S-T SEGMENT
4. USUALLY EFFECTS YOUNG, HEALTHY PEOPLE
5. CAUSES SUDDEN DEATH by TORSADES



Brugada Syndrome -- 3 ECG Patterns:

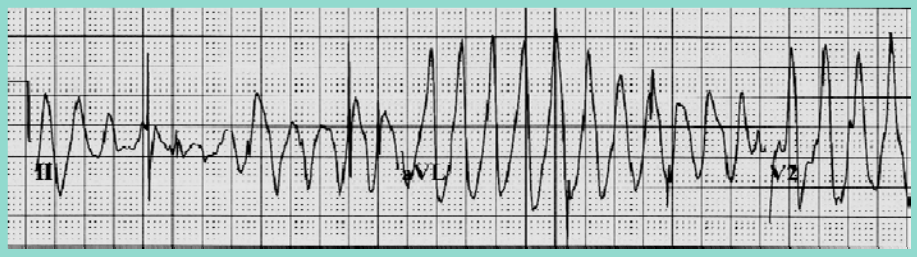


Type 1: Coved type ST-segment elevation

Type 2: saddle-back type ST-segment elevation

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

Brugada / Long QT Syndromes cause:

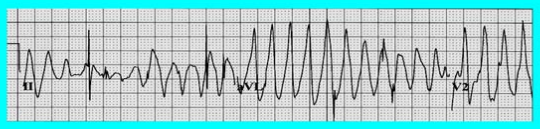


Torsades de Pointes:

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

WIDE COMPLEX TACHYCARDIA
TORSADES de POINTES

(QRS > 120 ms)



ABC s

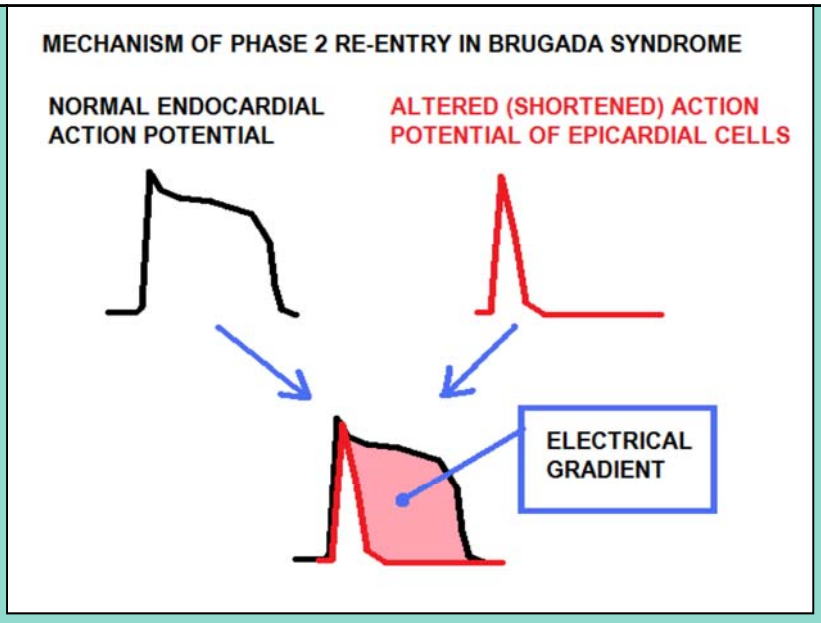
NO PULSE	PULSE - UNSTABLE	PULSE - STABLE
GO TO V - FIB ALGORITHM !	▪ IMMEDIATE DEFIBRILLATION START IV CONSIDER SEDATION	▪ O2 / IV / EKG ▪ MAGNESIUM INFUSION 1 - 2 gm OVER 5 - 60 min.

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

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

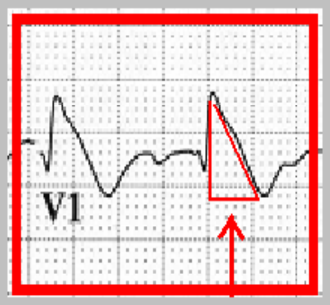
REPORT ANY ABNORMAL FINDINGS TO PHYSICIAN.



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

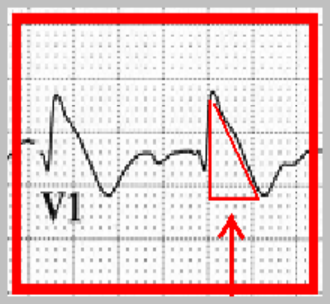
Brugada & Long QT Syndrome: Causes Torsades de Pointes – (TdP) A LETHAL DYSRHYTHMIA which Causes immediate syncope (no cardiac output). TDP may Degenerate into VENTRICULAR FIBRILLAION, resulting in CARDIAC ARREST and DEATH.

BRUGADA SYNDROME



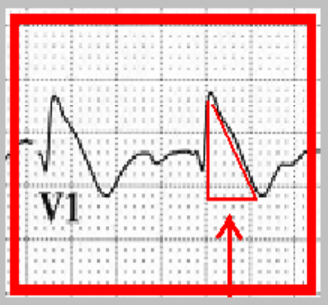
- GENETIC DISORDER - GENE SCN5A, which encodes CARDIAC SODIUM CHANNELS.
- CAUSES EARLY RIGHT VENTRICULAR SUB-EPICARDIAL REPOLARIZATION
- CAUSES RUNS OF TORSADES de POINTES, and SUDDEN DEATH from TORSADES and V-FIB.
- IS BELIEVED TO CAUSE 4 - 12 % of ALL SUDDEN DEATHS, and 50 % of ALL CARDIAC DEATHS where pt. has a STRUCTUALLY NORMAL HEART.

BRUGADA SYNDROME



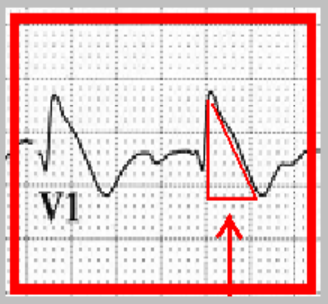
- SEVERAL VARIATIONS of this disorder are known to exist.
- CONCEALED and NON-CONCEALED.
- The NON-CONCEALED version HAS THE V1-V3 abnormality VISIBLE at all times.
- The CONCEALED version - pt. has a NORMAL EKG at most times - a DRUG STUDY, an EP STUDY, and / or GENETIC TESTING must be done to rule out or confirm diagnosis.

BRUGADA SYNDROME



- YOUNG MALES of SOUTHEAST ASIAN DESCENT are in HIGH RISK GROUP, however this disorder affects ANY RACE or GENDER.
- BRUGADA SYNDROME is HEREDITARY.
- SUSPECT BRUGADA SYNDROME in patients with FAMILY HISTORY of BRUGADA / SUDDEN DEATH, and/or TORSADES.

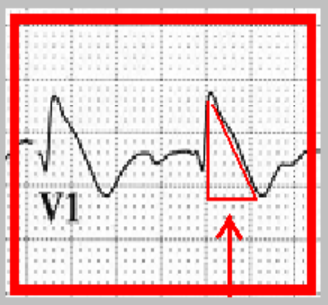
BRUGADA SYNDROME - TESTING



- For CONCEALED cases, a drug study of AJMALINE, FLECAINIDE, or PROCAINAMIDE can UNMASK the "tell-tale" TRIANGULAR COMPLEXES of V1 and V2.
- IN EP STUDIES, a PROLONGED H-V INTERVAL may be observed.
- GENETIC TESTING is performed by THE RAMON A. BRUGADA FOUNDATION.

BRUGADA SYNDROME - TREATMENT

ICD implantation is the only known effective treatment to date.



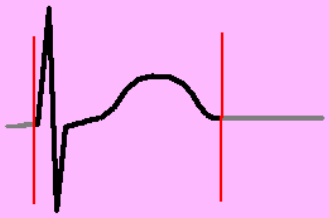
www.brugada.org

www.sads.org

www.QTsyndrome.ch

www.crediblemeds.org

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, *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.

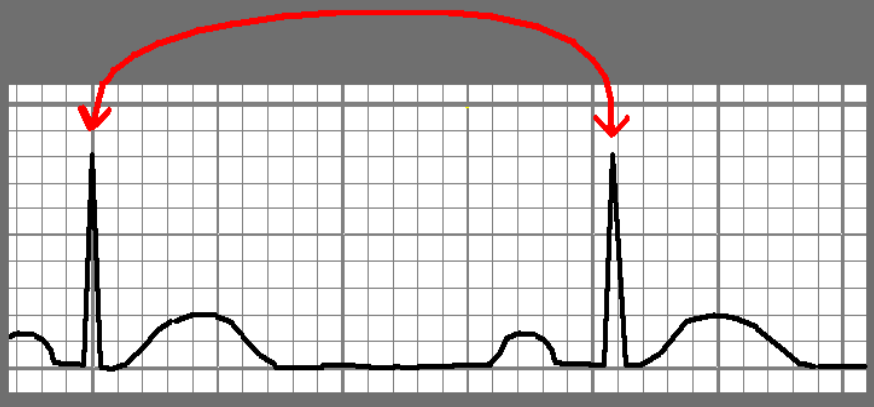
QT CORRECTION FORMULAS:

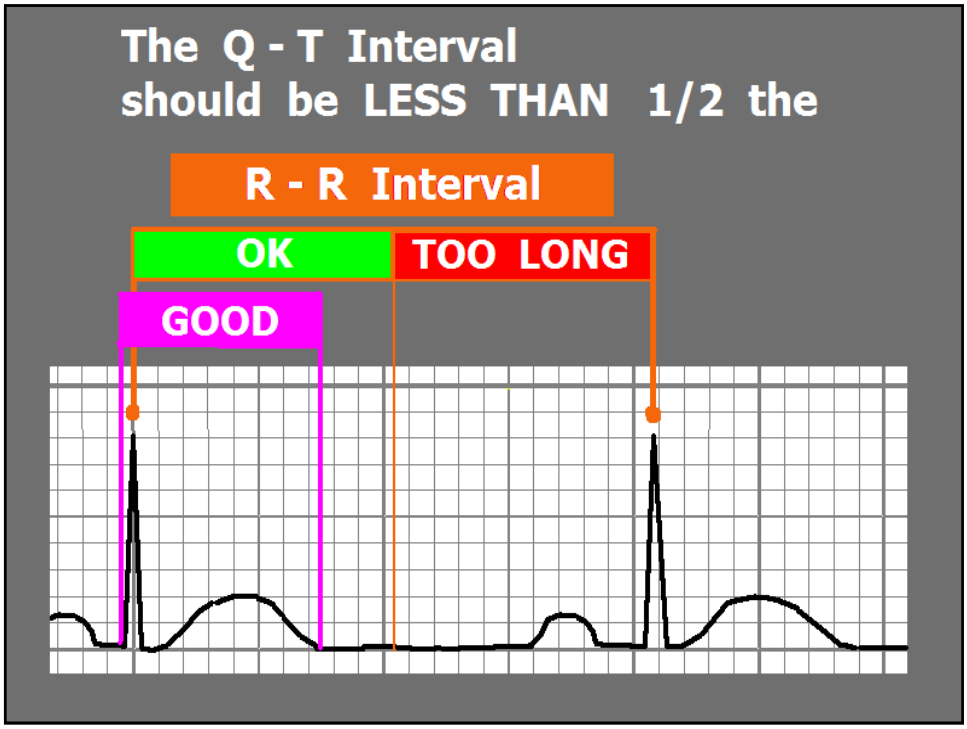
Bazett's	QTc=QT/√ RR
Fredericia	QTc=QT/(RR)1/3
Framingham	QTc=QT+0.154(1-RR)
Rautaharju	QTc=656/(1+HR/100)

DETERMINING Q-T INTERVAL LIMITS

THE "QUICK PEEK" METHOD

(for Heart Rates 60 - 100)



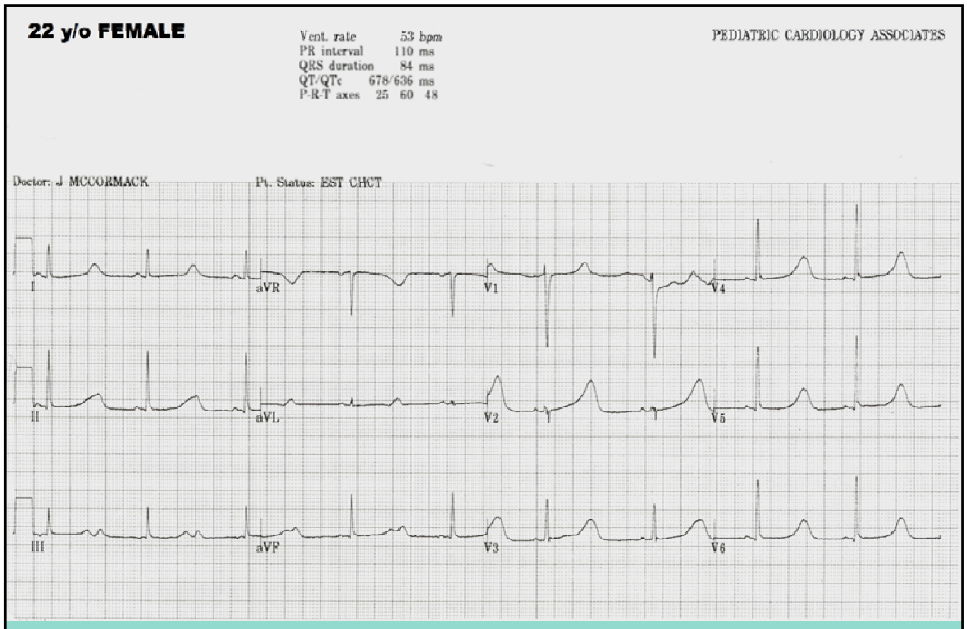


ECG Indicators of Long QT Syndrome:

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

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

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



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

CASE PROGRESSION - 22 YEAR OLD FEMALE:

DIAGNOSED WITH "EPILEPSY." All anticonvulsant medications were INEFFECTIVE at Controling grand-mal seizure activity.

During visit with Electrophysiologist, patient exhibited Torsades de Pointes during EST, collapsed. DURING TDP EPISODE patient experienced "grand mal Seizure."

ICD Implanted. ECG finding also discovered in patient's infant son. Received ICD at age 5.

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

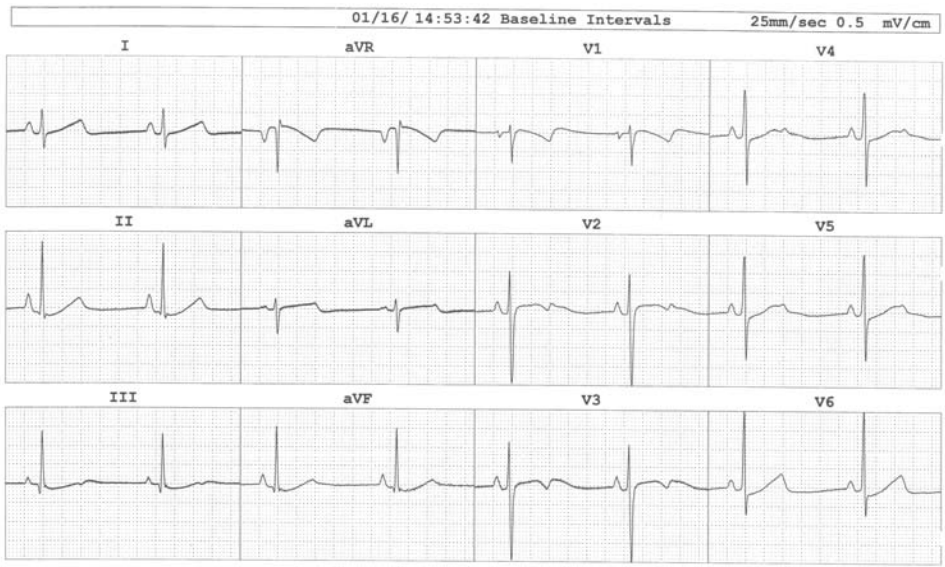
Miscellaneous

- Coronary Artery Disease
- Mitral Valve Prolapse

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

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

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



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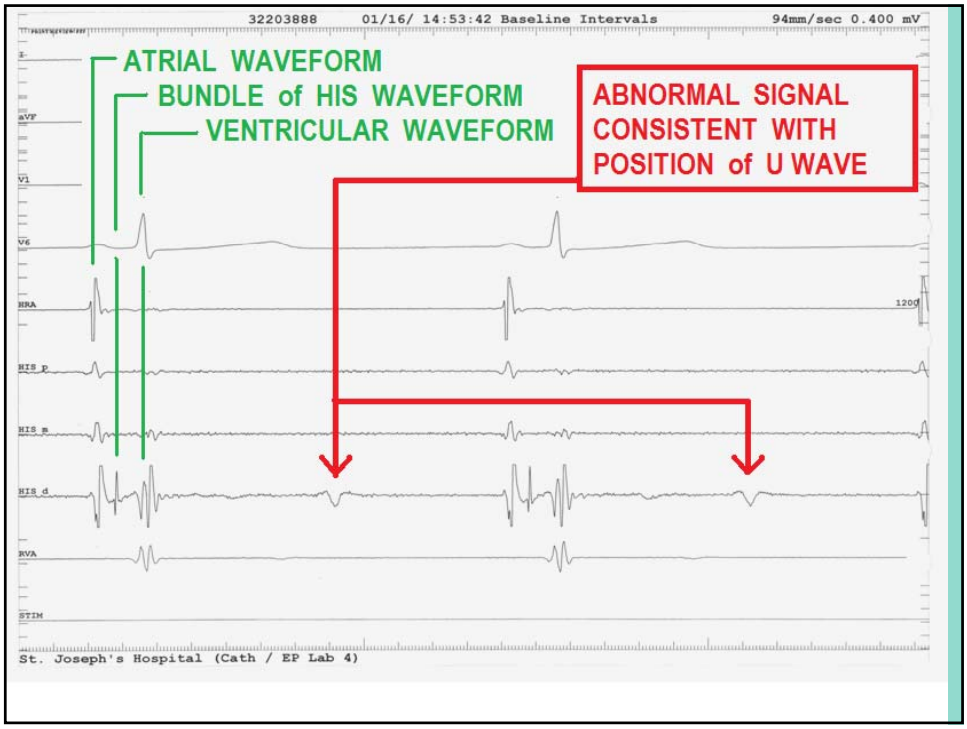
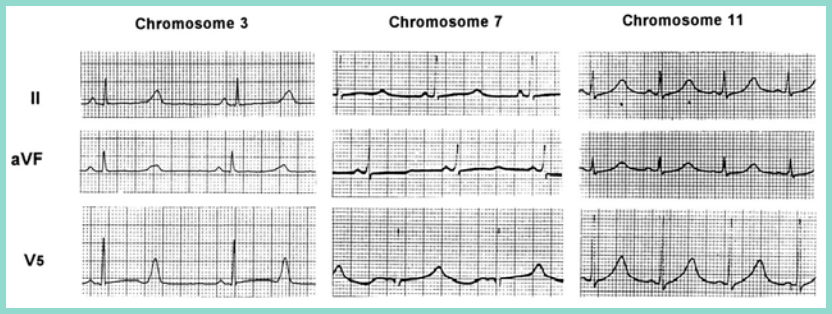
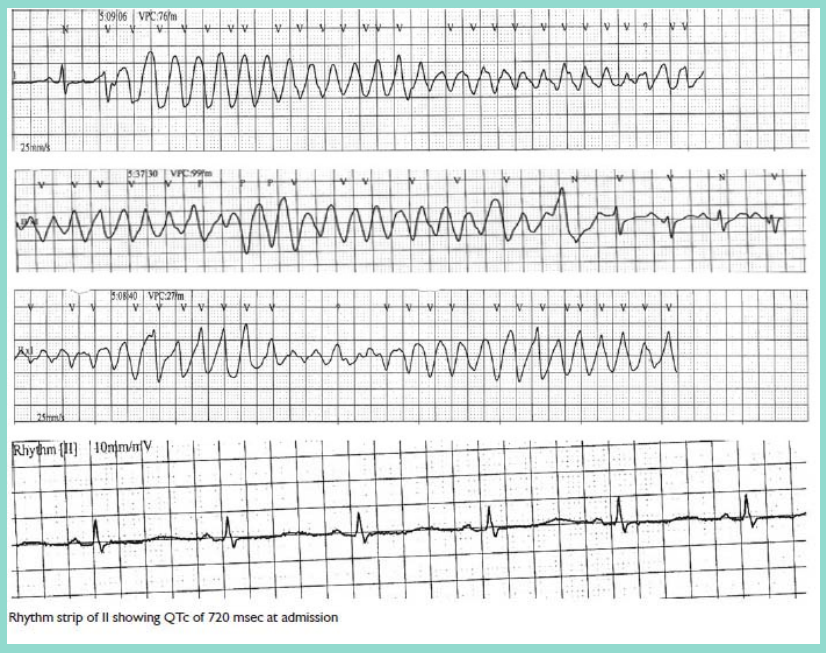


ECG recordings from leads II, aVF, and V5 in three patients from families with long QT syndrome linked to genetic markers on chromosomes 3, 7, and 11.

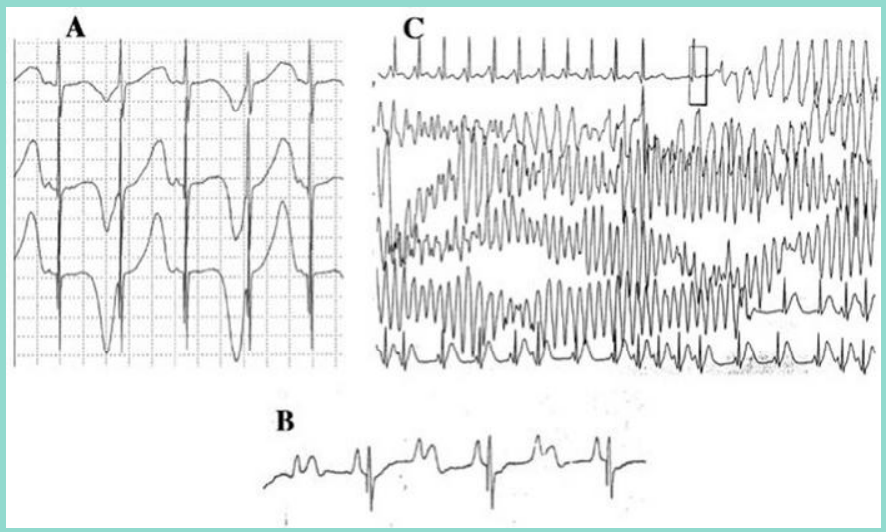


Moss A et al. Circulation 1995;92:2929-2934



Rhythm strip of II showing QTc of 720 msec at admission

A. T WAVE ALTERANS
B. LQTS TYPE 2 (NOTCHED T WAVES)
C. PATIENT WITH LQTS ecg = run of Torsades, reverts to NSR



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Acquired

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

Miscellaneous

- Coronary Artery Disease
- Mitral Valve Prolapse

If patient has a PROLONGED Q-T INTERVAL, AVOID DRUGS THAT LENGTHEN THE Q-T.

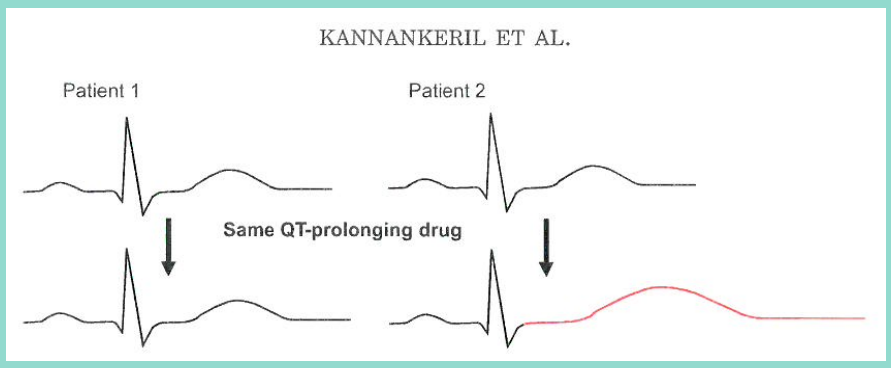
Such drugs include:

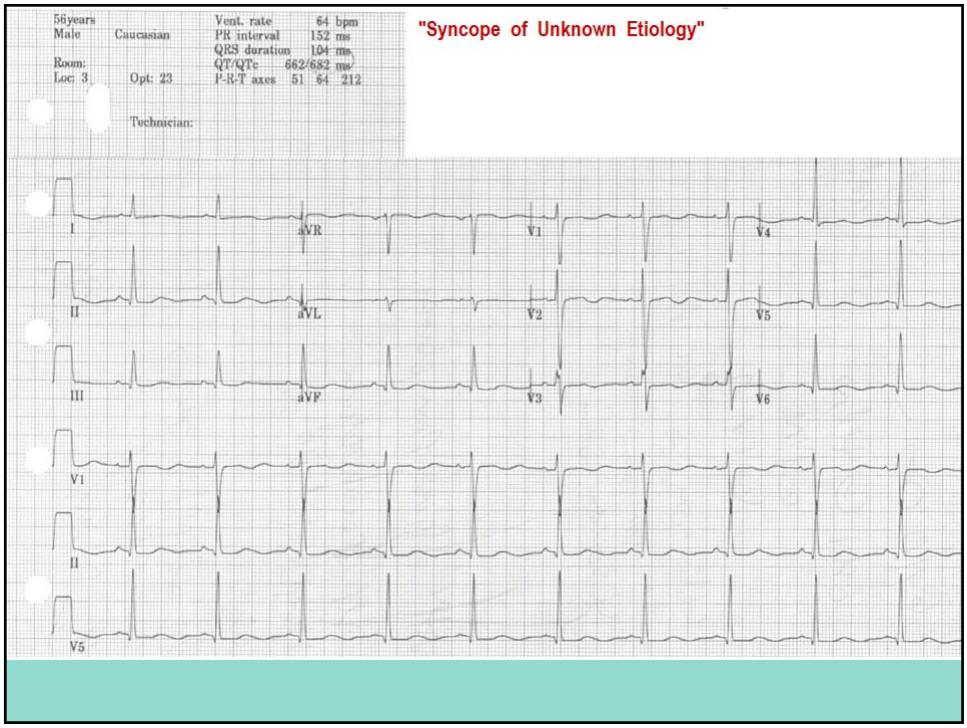
-Amiodarone	-Ritalin
-Procainamide	-Benadryl
-Levaquin	-Haloperidol
-Erythromycin	-Thorazine
-Norpace	-Propulcid
-Tequin	..... AND MANY MORE .....

www.torsades.org , & www.azcert.org

PATIENT 1: NORMAL

PATIENT 2: GENETIC REPOLARIZATION ABNORMALITIES and/or HYPOKALEMIA is PRESENT.





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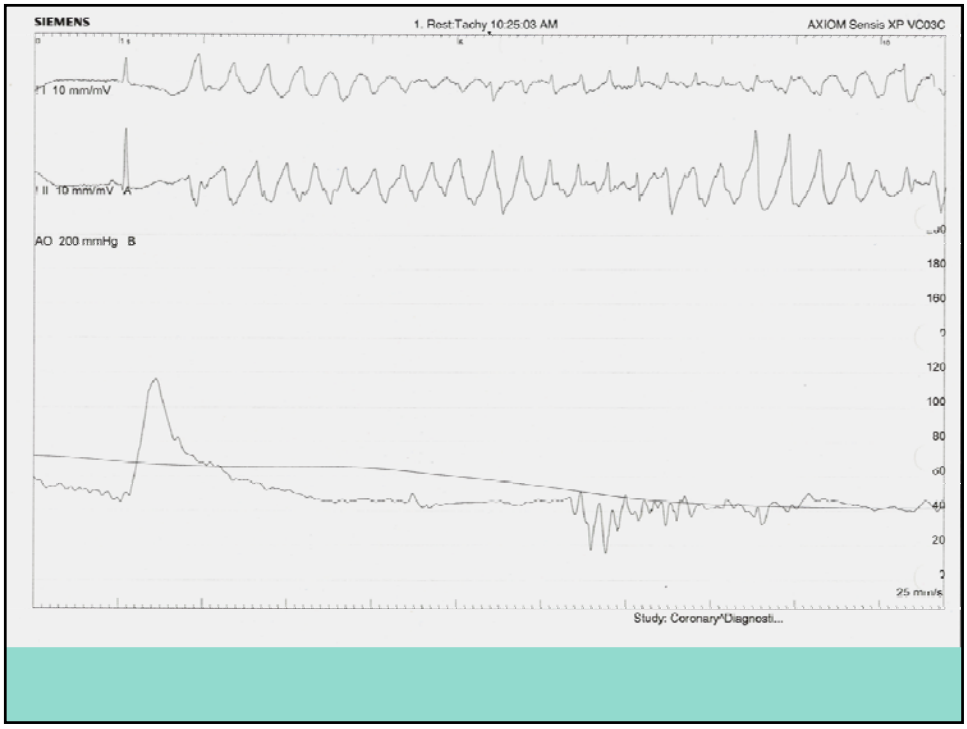
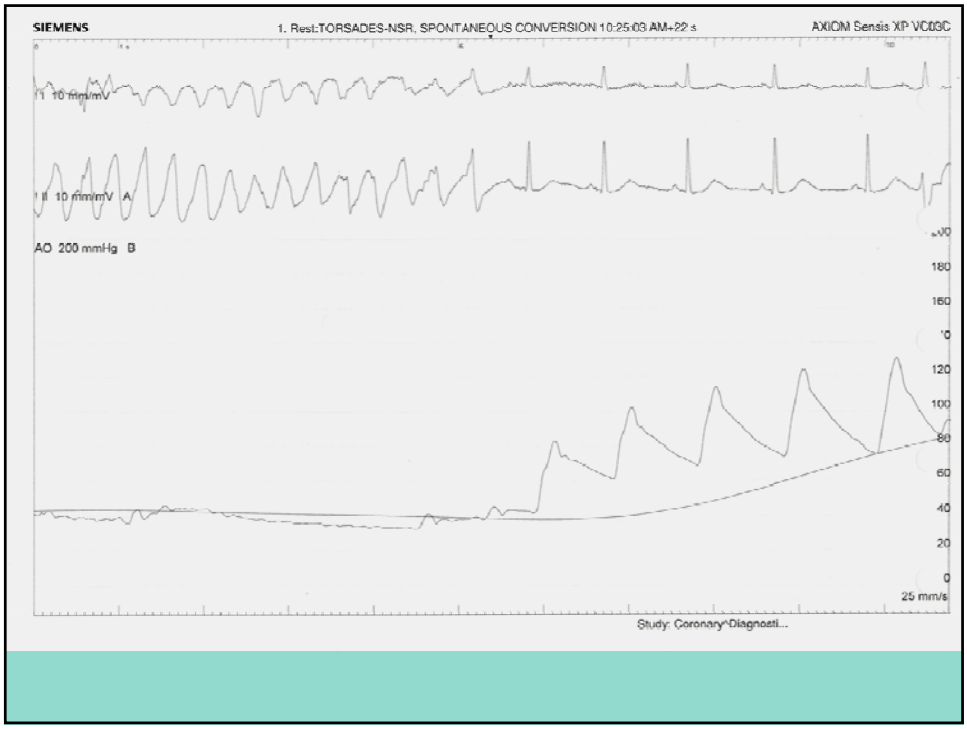
-Erythromycin	-Thorazine
-Norpace	-Propulcid
-Tequin	

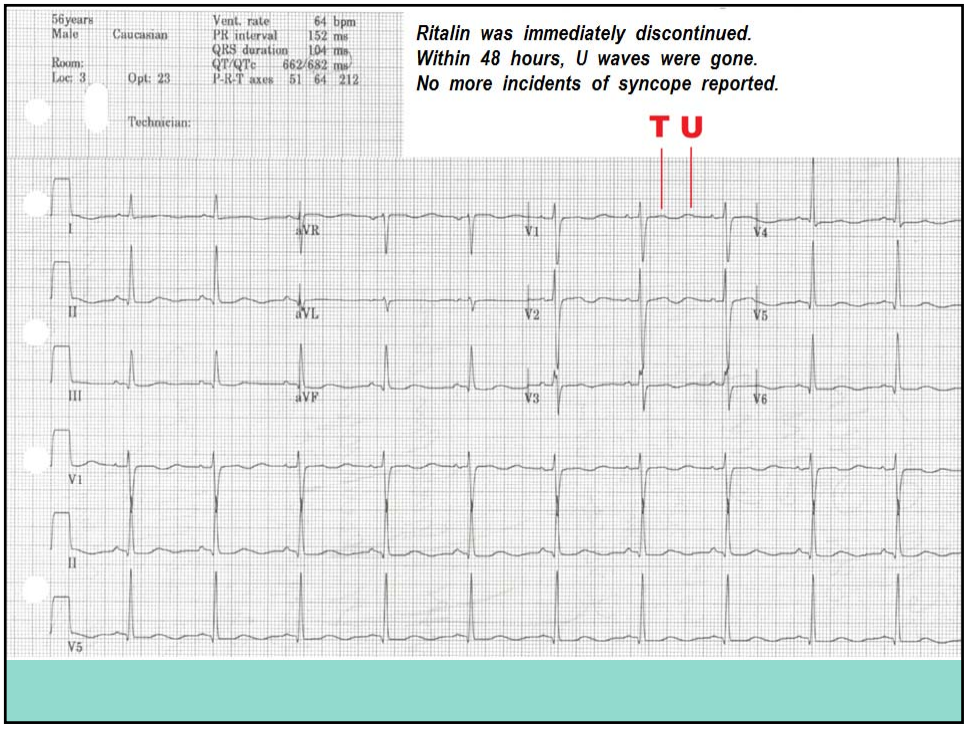
. . . . . AND MANY MORE . . . . . .

AND MANY MORE . . .

See: www.torsades.org / JAMA




SIEMENS
1. Rest:TORSADES-NSR, SPONTANEOUS CONVERSION 10:25:03 AM+22 s
AXIOM Sensis XP VC03C
I 10 mm/mV
II 10 mm/mV A
AO 200 mmHg B
180
160
120
100
60
40
20
0
25 mm/s
Study: Coronary^Diagnosti...


56years
Male Caucasian
Room:
Loc: 3 Opt: 23
Technician:
Vent. rate 64 bpm
PR interval 152 ms
QRS duration 104 ms
QT/QTc 662/682 ms
P-R-T axes 51 64 212
Ritalin was immediately discontinued.
Within 48 hours, U waves were gone.
No more incidents of syncope reported.
T U
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
V1
II
V5

Q: What is the ideal medication to treat Torsades?

Q: What is the ideal medication to treat Torsades?

A: Magnesium Sulfate, 1 – 2 grams over 5 – 60 minutes (AHA ACLS)

ABSOLUTELY NO DRUGS THAT PROLONG THE Q-T INTERVAL ! !

ECG Indicators of Long QT Syndrome:

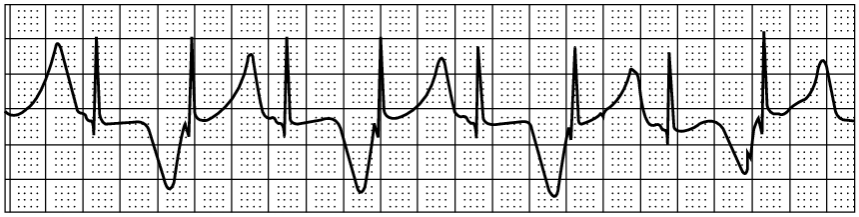
- QTc 460ms or longer in females*
- QTc 450ms or longer in males*
- T wave alterans
- U waves >100% of the T wave
- U waves merged with T waves
- U waves >0.1mv (1mm on standard calibrated ECG)

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

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

Long QT Syndrome: T wave Alterans:

ECG Indicating T Wave Alternans



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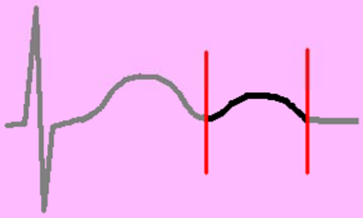
☞ **WHEN LQTS IS SUSPECTED, TAKE THE FOLLOWING PRECAUTIONS . . . .**

THE U WAVE

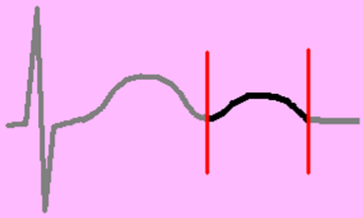


- SEEN INFREQUENTLY

U WAVES PANIC VALUES (CONSIDERED INDICATOR OF LONG QT SYNDROME) WHEN:

- 100% or more SIZE OF T WAVE
- MERGED WITH T WAVE
- MORE THAN 1mm IN HEIGHT

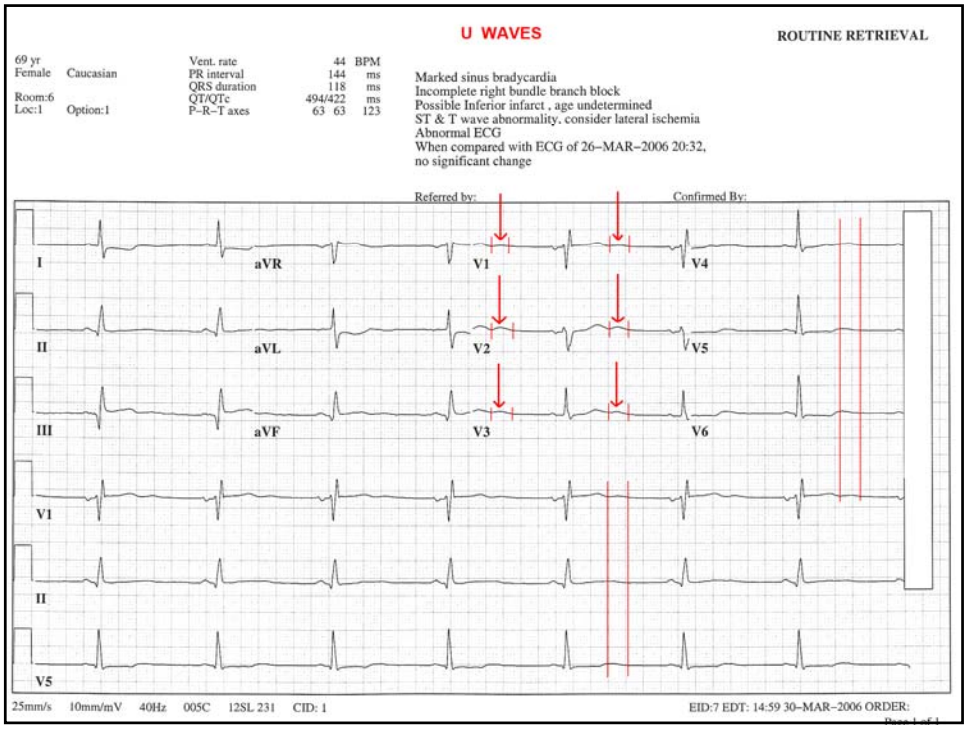
THE U WAVE

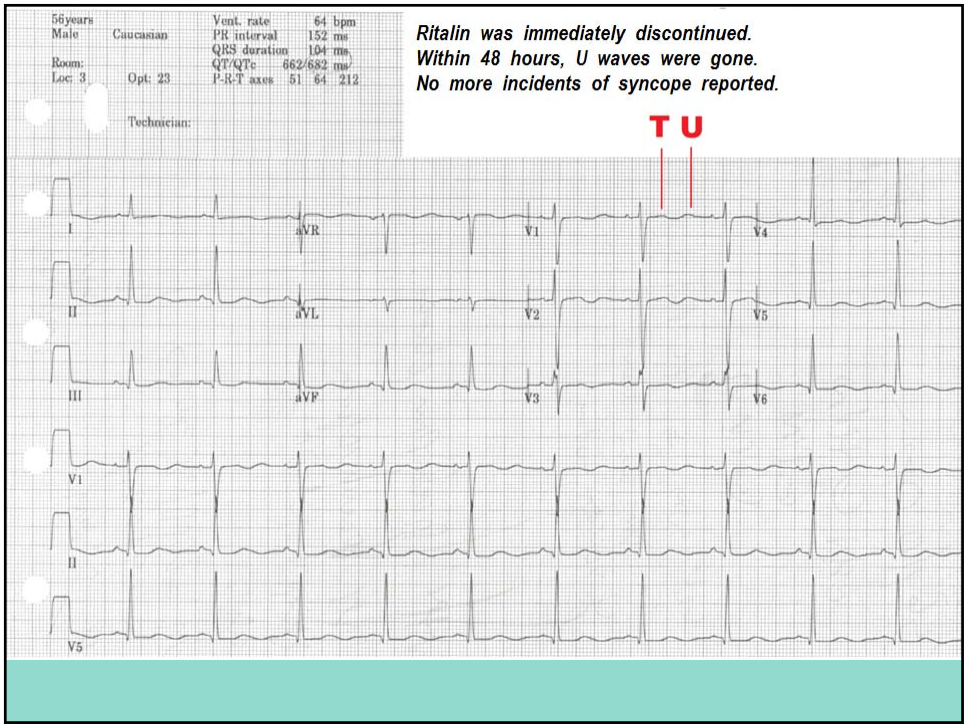


- SEEN INFREQUENTLY

- MOST VISIBLE IN LEADS V2 & V3

- OFTEN NOT PRESENT IN LEADS II, III, AVF


U WAVES
ROUTINE RETRIEVAL
69 yr
Female Caucasian
Room:6
Loc:1 Option:1
Vent. rate 44 BPM
PR interval 144 ms
QRS duration 118 ms
QT/QTc 494/422 ms
P-R-T axes 63 63 123
Marked sinus bradycardia
Incomplete right bundle branch block
Possible Inferior infarct , age undetermined
ST & T wave abnormality, consider lateral ischemia
Abnormal ECG
When compared with ECG of 26-MAR-2006 20:32,
no significant change
Referred by:
Confirmed By:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 40Hz 005C 12SL 231 CID: 1
EID:7 EDT: 14:59 30-MAR-2006 ORDER:


56years
Male Caucasian
Room:
Loc: 3 Opt: 23
Technician:
Vent. rate 64 bpm
PR interval 152 ms
QRS duration 104 ms
QT/QTc 662/682 ms
P-R-T axes 51 64 212
Ritalin was immediately discontinued.
Within 48 hours, U waves were gone.
No more incidents of syncope reported.
T U
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

When ECG Indicators of Long QT Synrome are present:

- Obtain a thorough patient history, to rule out incidence of syncope and family history of sudden death/ near sudden death.
- Evaluate patient's meds list for meds that prolong the QT Interval.
- Rule out hypothermia
- Rule out CVA
- Evaluate the patient's electrolyte levels, and
- MONITOR PATIENT'S ECG FOR RUNS OF TORSADES
- Consider "expert consult" (electrophysiologist) to rule out LQTS

When ECG Indicators of Long QT Synrome are present:

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- MONITOR PATIENT'S ECG FOR RUNS OF TORSADES
- Consider "expert consult" (electrophysiologist) to rule out LQTS

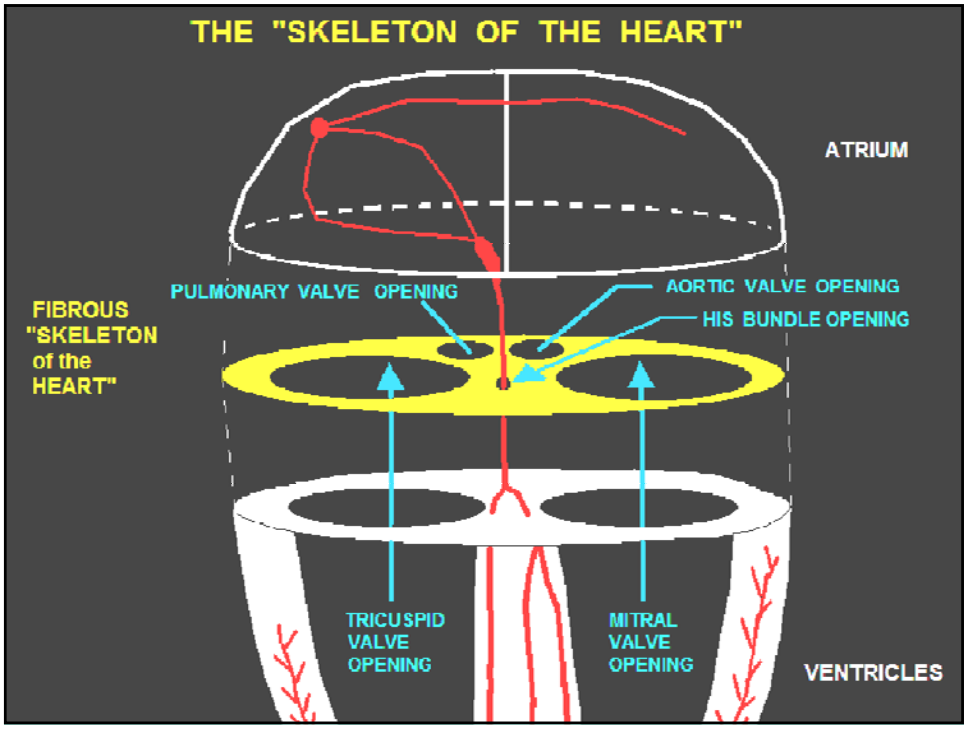
☞ ***LQTS + ECTOPY =*** ⇧ ***Risk of Torsades de Pointes!***

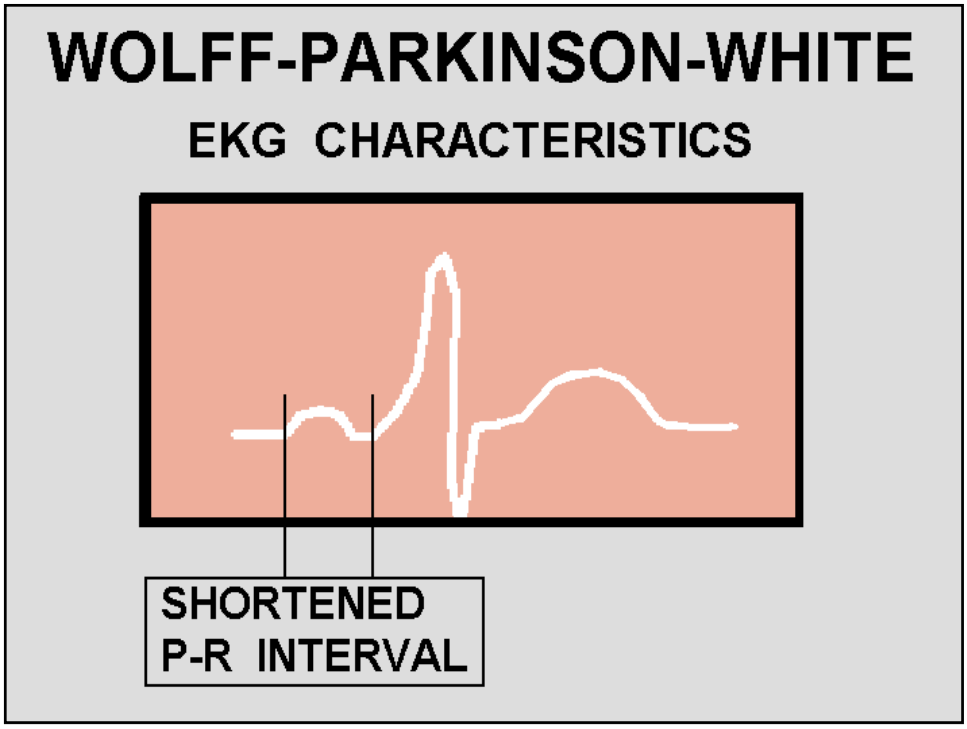
Suspected LQTS Considerations include:

- ***Avoidance of Meds that are known to prolong the QT Interval.***
  (refer to LIST OF MEDS KNOWN TO PROLONG THE QT INTERVAL).

TREATMENT OF TORSADES de POINTES per AHA ACLS:

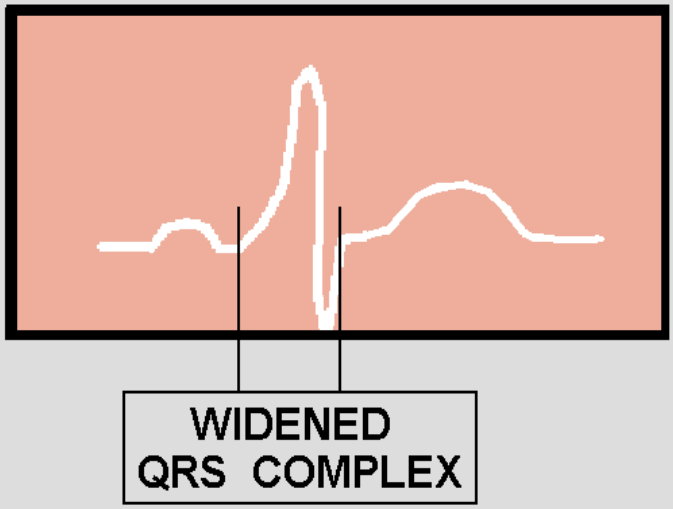
- TRANSIENT: MAGNESIUM SULFATE 1 – 2 gm IV infusion over 5 – 60 minutes.
- PERSISTENT, PATIENT UNSTABLE: DEFIBRILLATION (prior to 2010: Synchronized Cardioversion)
- CARDIAC ARREST: FOLLOW Ventricular Fibrillation Algorithm.


THE "SKELETON OF THE HEART"
ATRIUM
PULMONARY VALVE OPENING
AORTIC VALVE OPENING
HIS BUNDLE OPENING
FIBROUS "SKELETON of the HEART"
TRICUSPID VALVE OPENING
MITRAL VALVE OPENING
VENTRICLES


WOLFF-PARKINSON-WHITE
EKG CHARACTERISTICS
SHORTENED
P-R INTERVAL

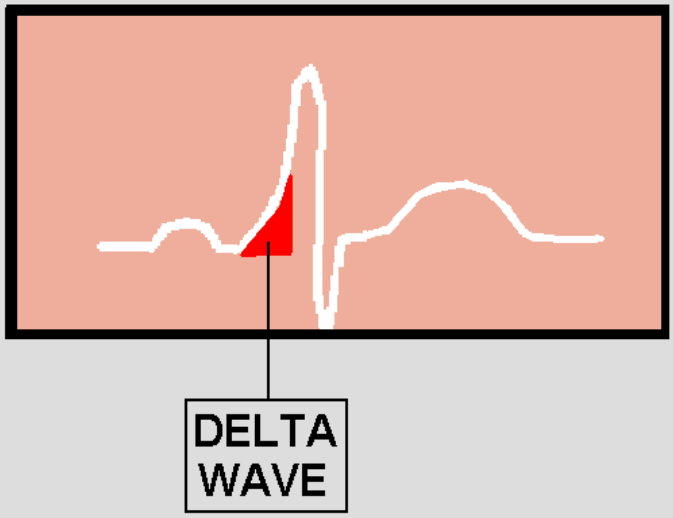
WOLFF-PARKINSON-WHITE

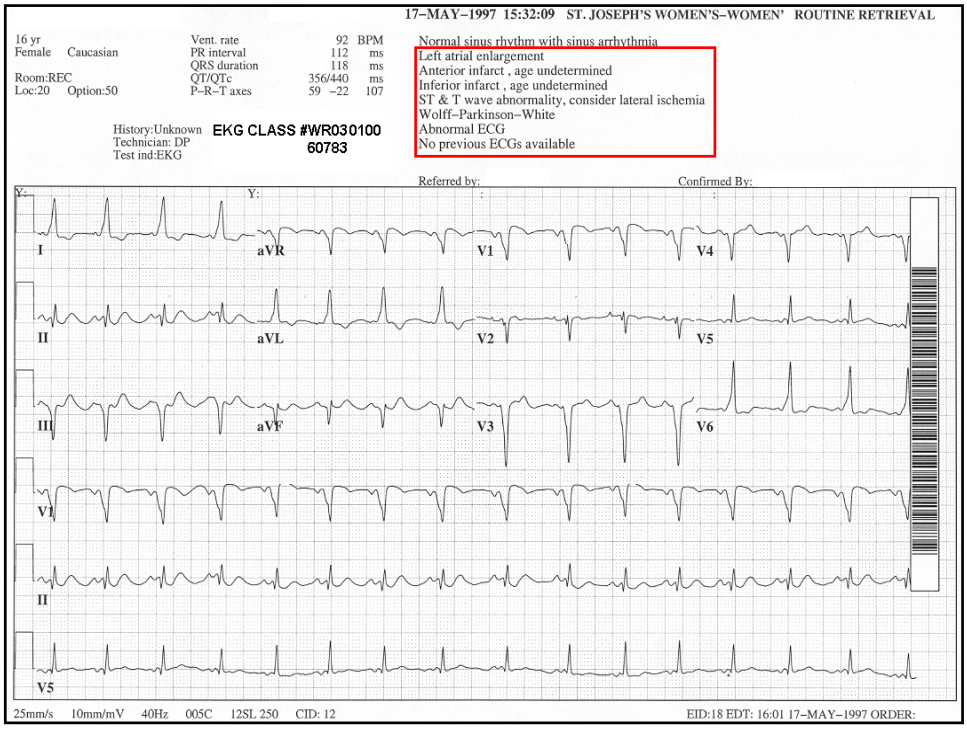
EKG CHARACTERISTICS

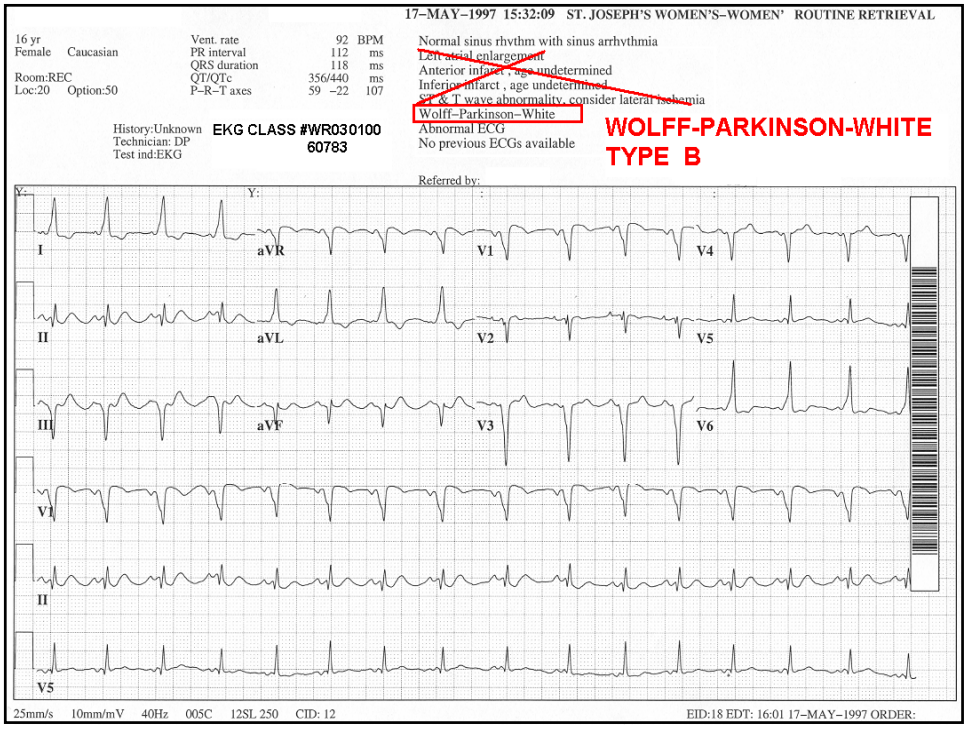


WOLFF-PARKINSON-WHITE

EKG CHARACTERISTICS




17–MAY–1997 15:32:09 ST. JOSEPH'S WOMEN'S–WOMEN' ROUTINE RETRIEVAL
16 yr
Female Caucasian
Room:REC
Loc:20 Option:50
Vent. rate 92 BPM
PR interval 112 ms
QRS duration 118 ms
QT/QTc 356/440 ms
P–R–T axes 59 –22 107
Normal sinus rhythm with sinus arrhythmia
Left atrial enlargement
Anterior infarct , age undetermined
Inferior infarct , age undetermined
ST & T wave abnormality, consider lateral ischemia
Wolff–Parkinson–White
Abnormal ECG
No previous ECGs available
History:Unknown
Technician: DP
Test ind:EKG
EKG CLASS #WR030100
60783
Referred by:
Confirmed By:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 40Hz 005C 12SL 250 CID: 12
EID:18 EDT: 16:01 17–MAY–1997 ORDER: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PR interval 112 ms
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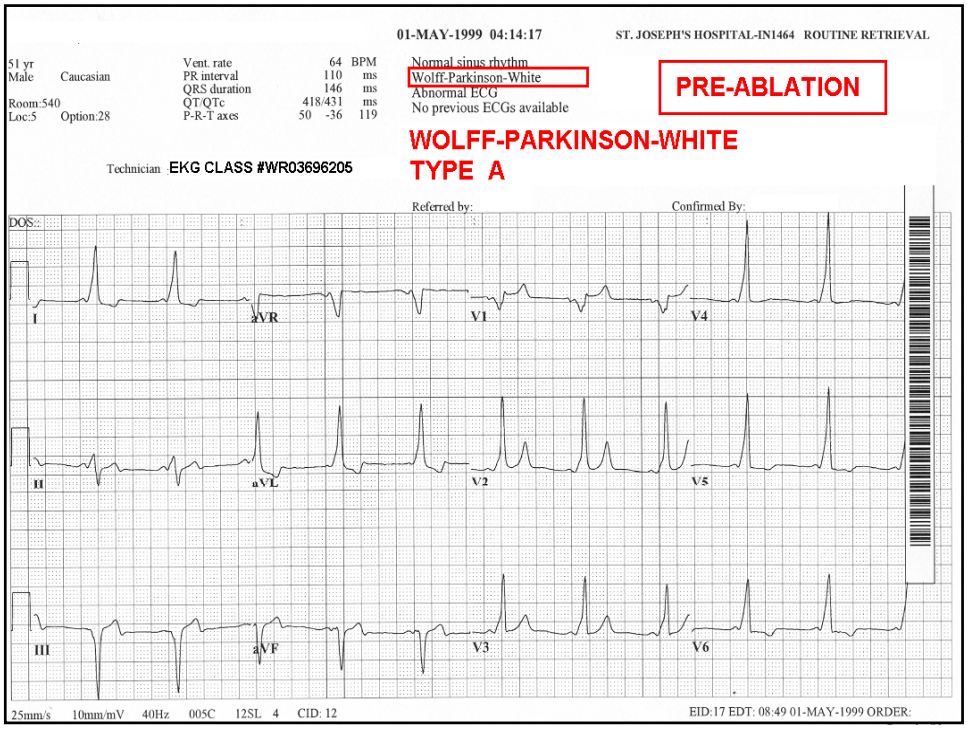
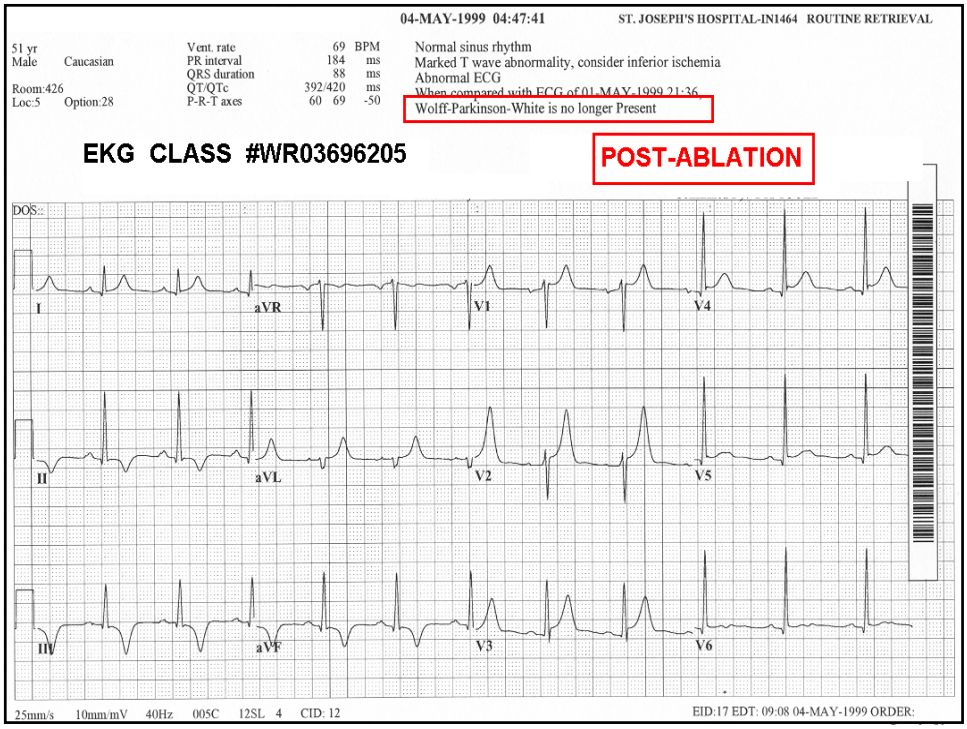
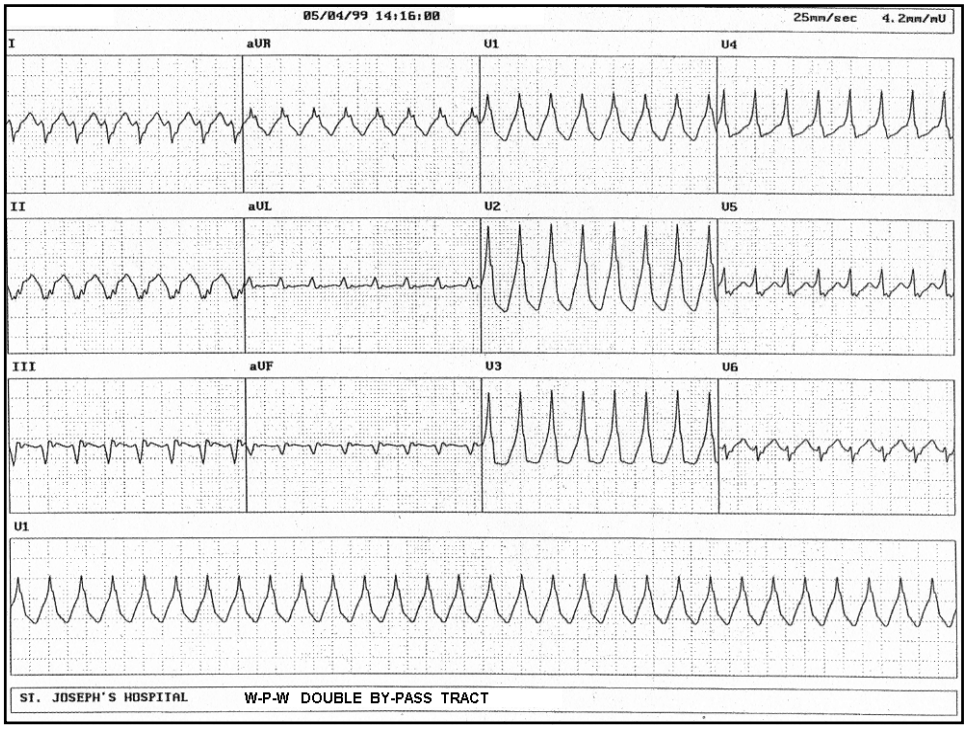
Inferior infarct , age undetermined
ST & T wave abnormality, consider lateral ischemia
Wolff–Parkinson–White
Abnormal ECG
No previous ECGs available
WOLFF-PARKINSON-WHITE
TYPE B
History:Unknown
Technician: DP
Test ind:EKG
EKG CLASS #WR030100
60783
Referred by:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 40Hz 005C 12SL 250 CID: 12
EID:18 EDT: 16:01 17–MAY–1997 ORDER:


01-MAY-1999 04:14:17
ST. JOSEPH'S HOSPITAL-IN1464 ROUTINE RETRIEVAL
51 yr
Male Caucasian
Room:540
Loc:5 Option:28
Vent. rate 64 BPM
PR interval 110 ms
QRS duration 146 ms
QT/QTc 418/431 ms
P-R-T axes 50 -36 119
Normal sinus rhythm
Wolff-Parkinson-White
Abnormal ECG
No previous ECGs available
PRE-ABLATION
WOLFF-PARKINSON-WHITE
TYPE A
Technician EKG CLASS #WR03696205
Referred by:
Confirmed By:
DOS::
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 12
EID:17 EDT: 08:49 01-MAY-1999 ORDER:


04-MAY-1999 04:47:41
ST. JOSEPH'S HOSPITAL-IN1464 ROUTINE RETRIEVAL
51 yr
Male Caucasian
Room:426
Loc:5 Option:28
Vent. rate 69 BPM
PR interval 184 ms
QRS duration 88 ms
QT/QTc 392/420 ms
P-R-T axes 60 69 -50
Normal sinus rhythm
Marked T wave abnormality, consider inferior ischemia
Abnormal ECG
When compared with ECG of 01-MAY-1999 21:36,
Wolff-Parkinson-White is no longer Present
EKG CLASS #WR03696205
POST-ABLATION
DOS::
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 12
EID:17 EDT: 09:08 04-MAY-1999 ORDER:



Wolff-Parkinson-White + A-fib = DISASTER

37 y/o male

Chief Complaint: Lightheadedness, Palpitations, Shortness of Breath

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

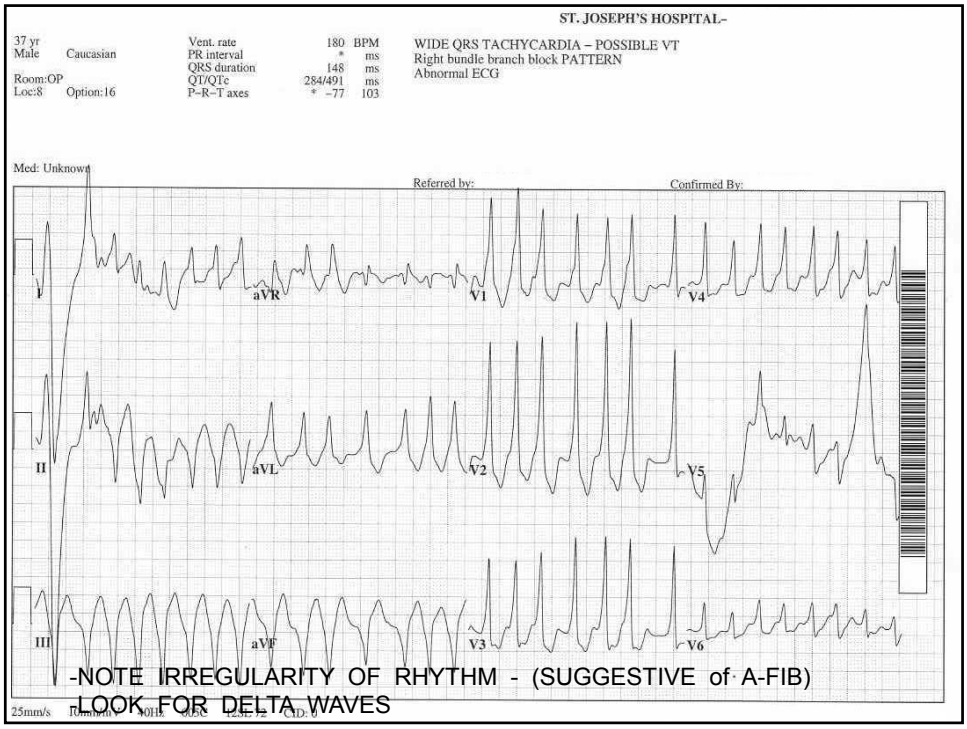
PMH: HTN (non-compliant)

37 y/o male

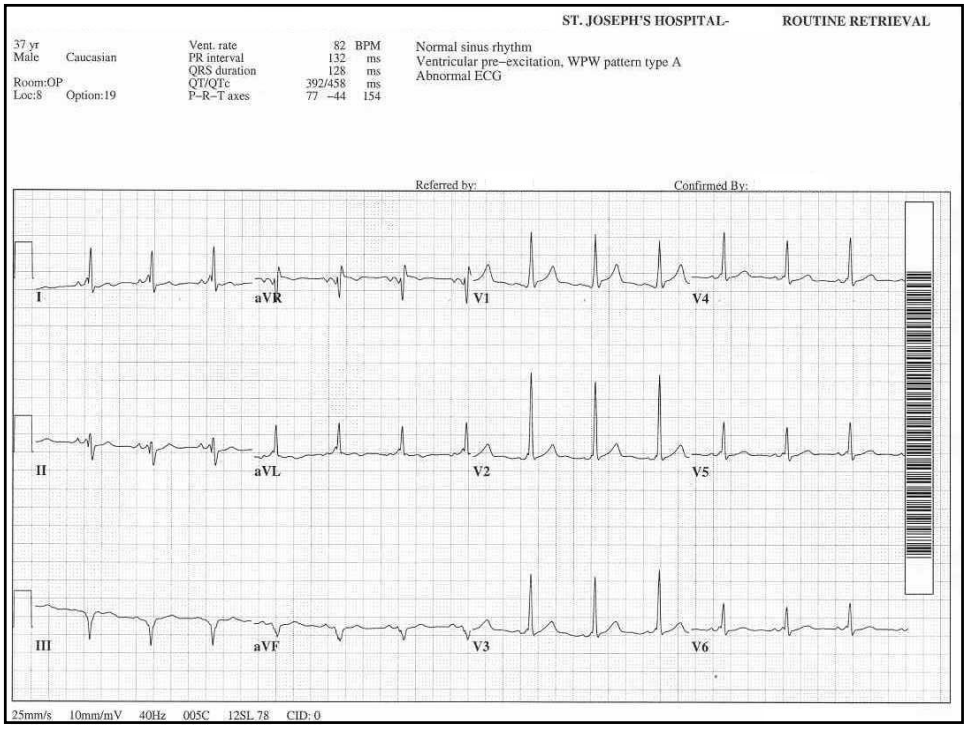
PE: Alert, oriented, restless, cool, pale, dry skin. PERL, No JVD, Lungs clear. Abd soft non tender, Extremities: WNL, no edema

Meds: None, NKDA

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



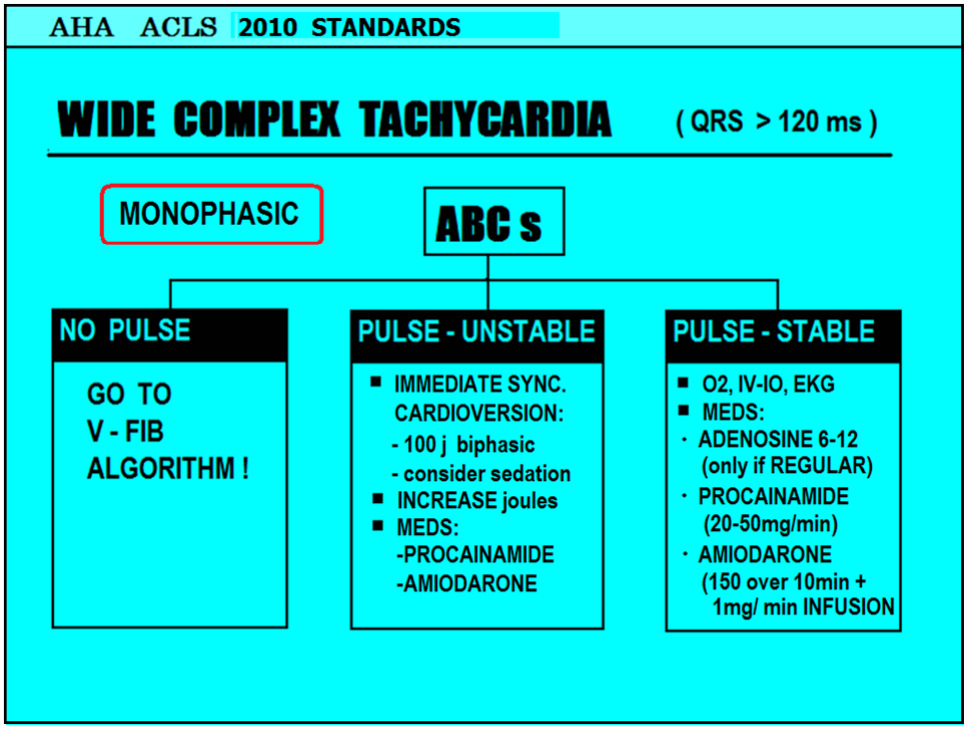
-NOTE IRREGULARITY OF RHYTHM - (SUGGESTIVE of A-FIB)
-LOOK FOR DELTA WAVES



Physician correctly identified Atrial Fibrillation with Rapid Ventricular Response.

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

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



AHA ACLS 2010 STANDARDS

WIDE COMPLEX TACHYCARDIA (QRS > 120 ms)

MONOPHASIC

ABC s

NO PULSE

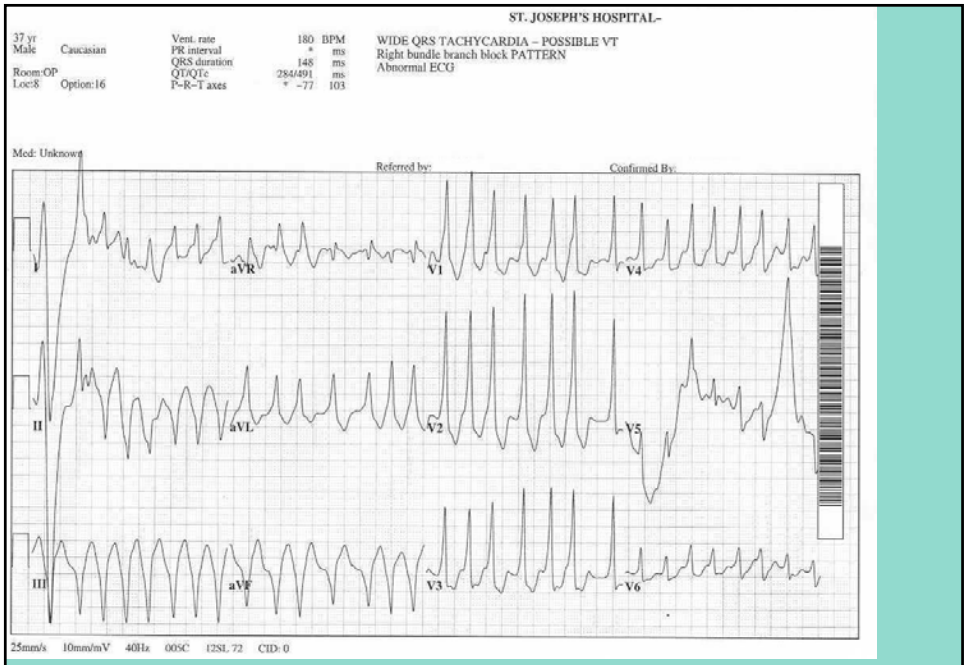
GO TO
V - FIB
ALGORITHM !

PULSE - UNSTABLE

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

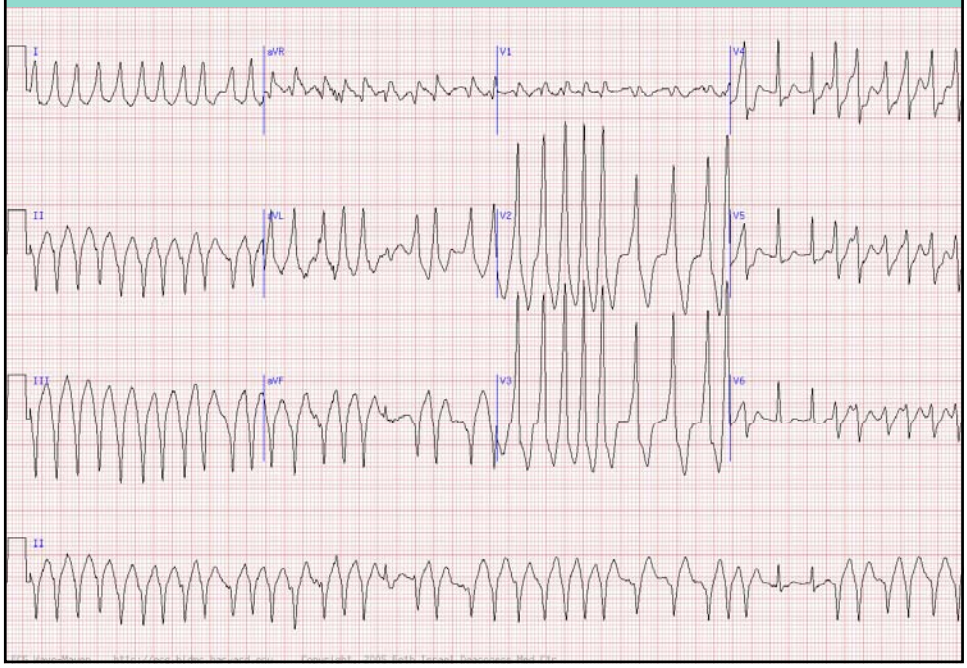
PULSE - STABLE

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



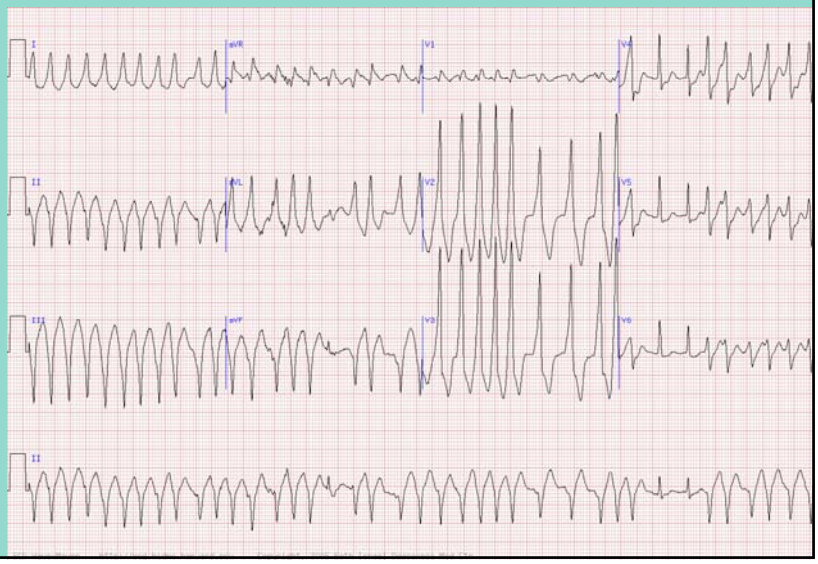
-NOTE IRREGULARITY OF RHYTHM - (SUGGESTIVE of A-FIB)
-LOOK FOR DELTA WAVES

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



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

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



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

Arrhythmogenic Right Ventricular Dysplasia

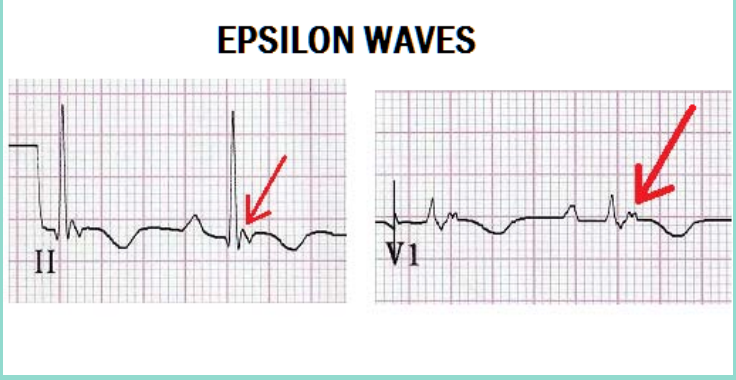
- A genetically acquired myocardial disease associated with paroxysmal ventricular arrhythmias and sudden cardiac death.
- Characterized pathologically by fibro-fatty replacement of the right ventricular myocardium.
- The second most common cause of sudden cardiac death in young people (after HOCM), 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

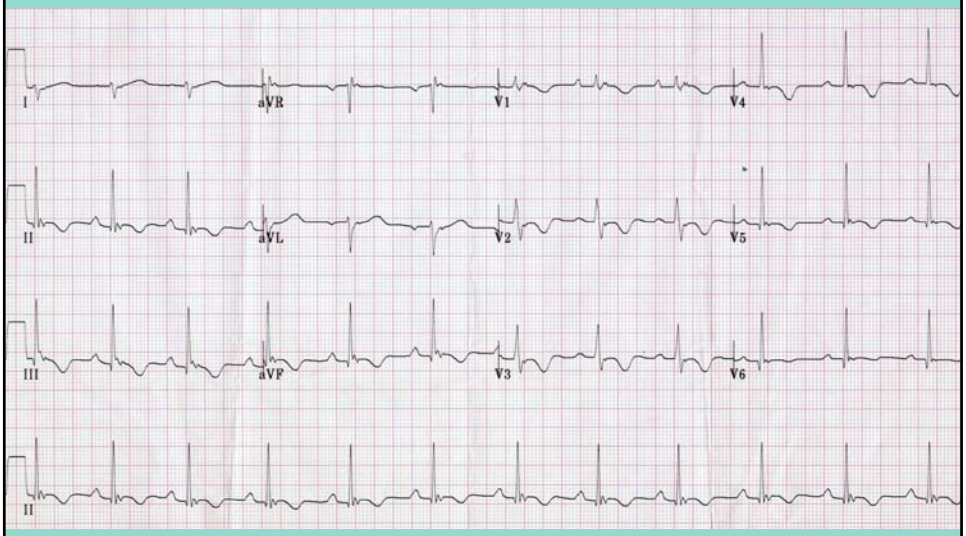
Arrhythmogenic Right Ventricular (RV) Cardiomyopathy and/or Dysplasia:

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

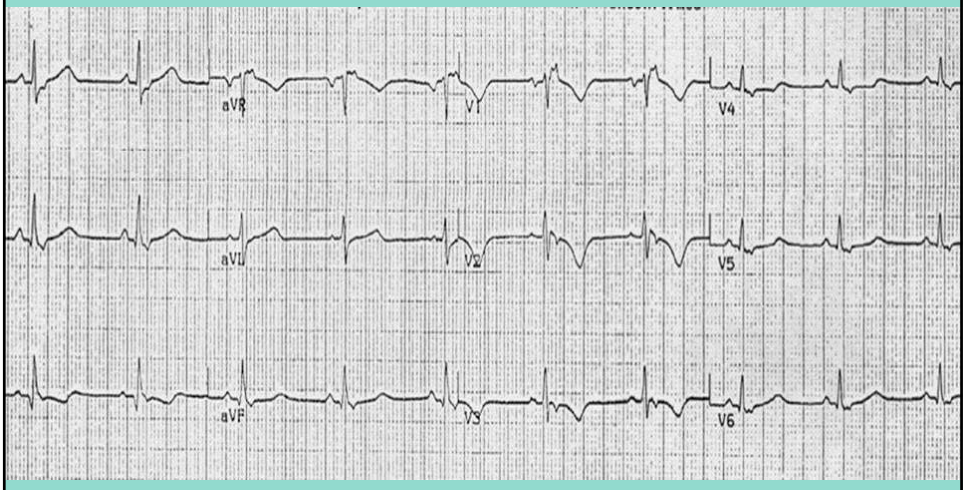


ARVD ECG 1



1. "Incomplete RBBB" Pattern
2. V1, V2 Rs pattern
3. Inverted T waves, symmetrical, - Global
4. Epsilon's waves

ARVD ECG 2



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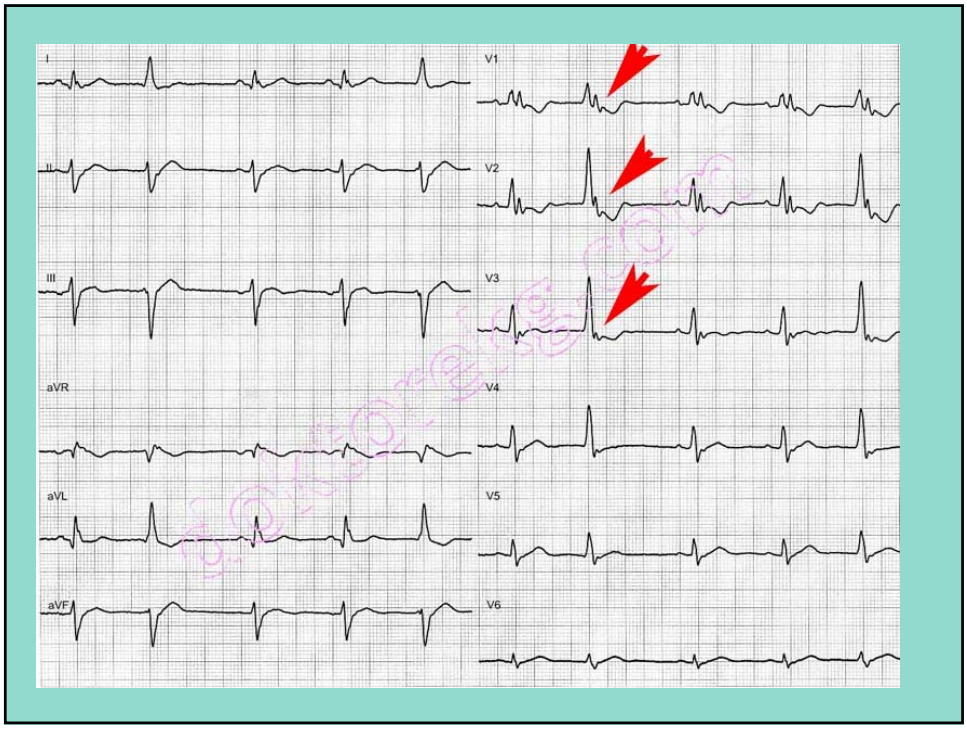
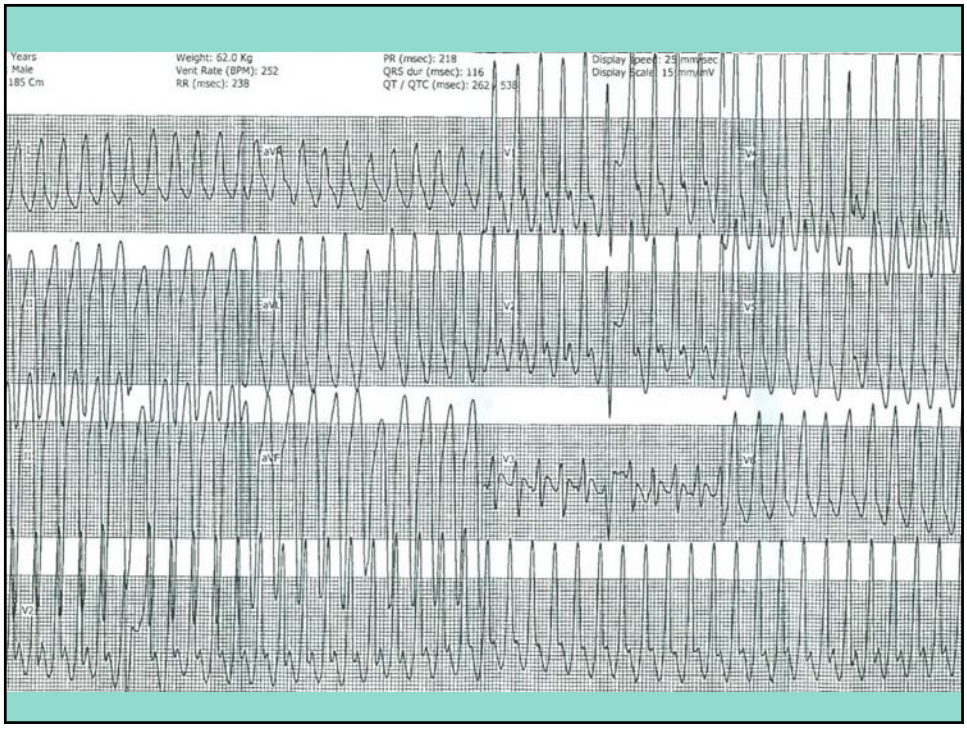
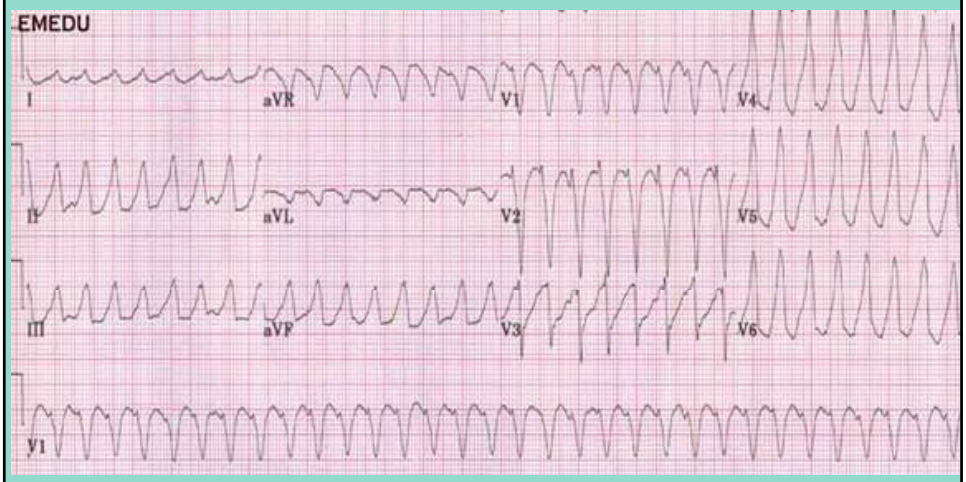

I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6


Years
Male
185 Cm
Weight: 62.0 Kg
Vent Rate (BPM): 252
RR (msec): 238
PR (msec): 218
QRS dur (msec): 116
QT / QTC (msec): 262 / 538
Display Speed: 25 mm/sec
Display Scale: 15 mm/mV

ARVD INDUCED VT



THE CARDIOMYOPATHIES

CARDIOMYOPATHY -

DAMAGE or DEATH OF CARDIAC MUSCLE CELLS, USUALLY THE RESULT OF A PATHOLOGICAL PROCESS, RESULTING IN A GLOBAL DECREASE OF VENTRICULAR FUNCTION.

THE CARDIOMYOPATHIES

ISCHEMIC CARDIOMYOPATHY:

CAUSED BY OR RESULTING FROM ISCHEMIC CORONARY ARTERY DISEASE.

- REVERSIBLE: WHEN CAUSED BY ISCHEMIA WITHOUT NECROSIS
- IRREVERSIBLE: WHEN CAUSED BY NECROTIC TISSUE

THE CARDIOMYOPATHIES

NON-ISCHEMIC CARDIOMYOPATHY:

1. DILATED
2. HYPERTROPHIC
3. RESTRICTIVE
4. SPECIFIC (OTHER)

THE CARDIOMYOPATHIES

ETIOLOGY (NON-ISCHEMIC):

- IDIOPATHIC
- INFECTIOUS
- AUTOIMMUNE
- GENETIC
- ALCOHOLIC / TOXIC
- CARDIOVASCULAR DISEASE / CHRONIC HYPERTENSION

THE CARDIOMYOPATHIES

INFECTIOUS ETIOLOGIES:

VIRAL:
- COXSACKIE A & B
- ECHO
- INFLUENZA
- POLIO
- HERPES
- ADENOVIRUS
- MUMPS
- RUBELLA / RUBEOLA
- HEPATITIS B & C
- HIV

SPIROCHETAL:
- LYME'S DISEASE

BACTERIAL:
- SALMONELLA
- LEGIONELLA
- CLOSTRIDIUM

RICKETTSIAL

FUNGAL:
- CRYPTOCOCCUS

PROTOZOAN:
- TOXOPLASMOSIS GONDI
- TYPANOSOMIASIS CRUZI

THE CARDIOMYOPATHIES

SPECTRUM OF CLINICAL PRESENTATIONS:

- WEAKNESS
- DYSPNEA (often exertional)
- CONGESTIVE HEART FAILURE
- ANGINA / CHEST DISCOMFORT
- MIMIC ACUTE MI
- SYMPTOMS OF PERICARDITIS
- CONGESTIVE HEART FAILURE
- PALPITATIONS
- SUDDEN DEATH

THE CARDIOMYOPATHIES

EKG FINDINGS MAY INCLUDE:

- PERCARDITIS CHANGES
- CHAMBER HYPERTROPHY (A / V)
- Q / QS COMPLEXES
- POOR R WAVE PROGRESSION
- AV NODAL / BBB (LBBB common)
- ATRIAL FIBRILLATION
- VENTRICULAR COMPLEXES

THE CARDIOMYOPATHIES

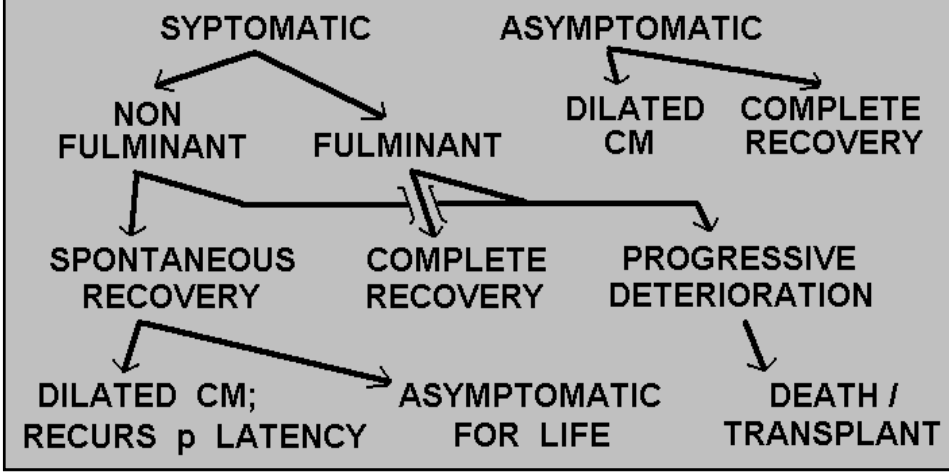
EKG FINDINGS, con't:

- Normal EKG

- ACUTE MI (S-T ELEVATION > 1mm IN TWO or more consecutive leads)

THE CARDIOMYOPATHIES

CARDIOTROPIC VIRAL INFECTION DISEASE PROGRESSION and PROGNOSES:



THE CARDIOMYOPATHIES

CASE STUDY: 19 y/o Female presents to ER via EMS, C/O shortness of breath. Her skin is pale, clammy, and diaphoretic. EMS states they found her lethargic, with a BP of 66/38. Her O2 SAT was 79. They placed her on O2 15 LPM via NRB mask, and started IV NS KVO, then bolused her with a 250cc fluid challenge. Currently she is awake, C/O DIB, weakness and nausea. She's "had the flu" for the last 10 days.

THE CARDIOMYOPATHIES

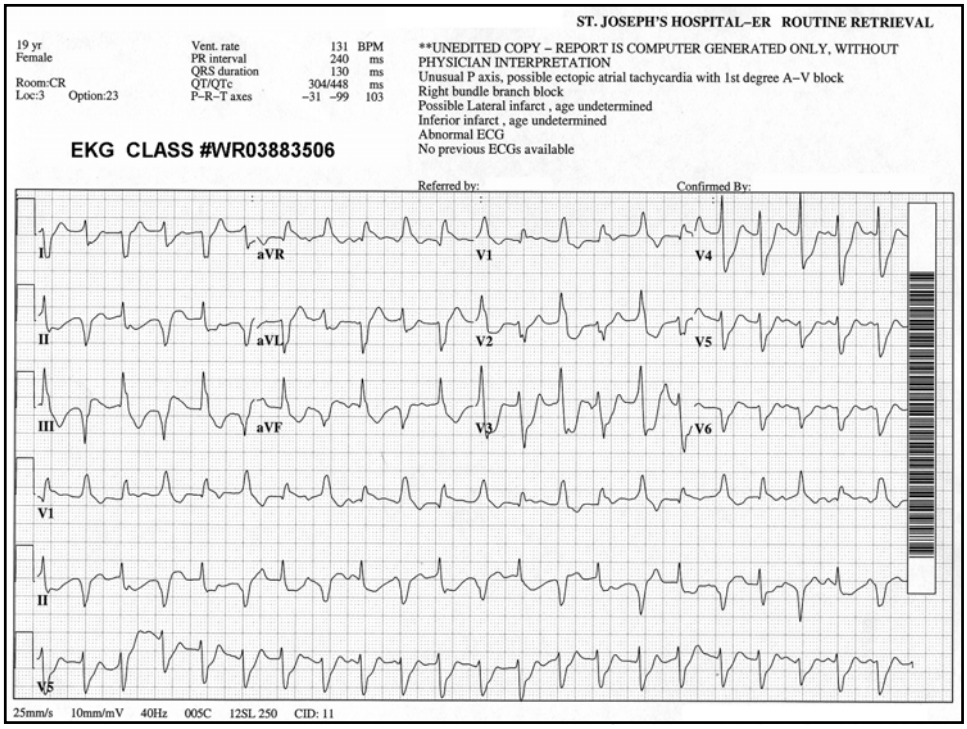
CASE STUDY: PHYSICAL EXAM reveals JVD, BBS= Coarse Crackles in bases and mid fields, rales in the upper fields. Pt is becoming increasingly anxious by the minute, C/O increased DIB. Her family states she has been "too weak to get out of bed for the last few days."
Repeat BP = 56 / 30, HR = 134, R = 36, SAO2 = 88% on 15 LPM O2 via NRB.

YOUR COURSE OF ACTION IS ?

THE CARDIOMYOPATHIES

CASE PROGRESSION:

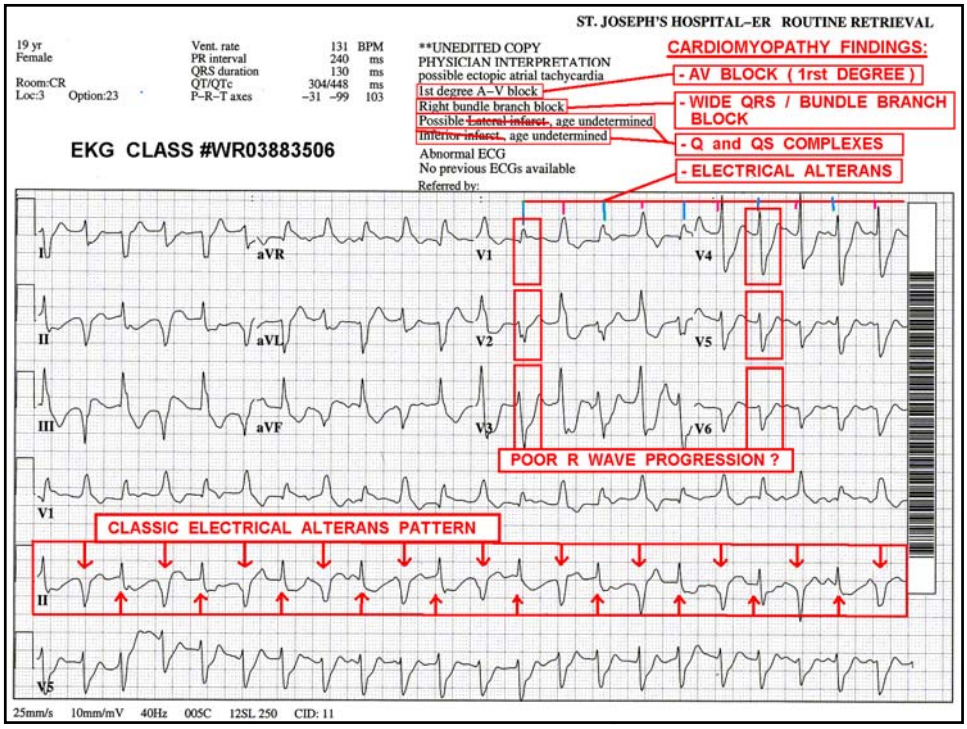
- STAT INTUBATION
- EKG
- DOPAMINE gtt (15 mcg / kg / min)
- CXR
- ECHOCARDIOGRAM
- LABS (CMP, CBC, PT/PTT/INR, ABG, TOXICOLOGY, BLOOD CULTURES, CARDIAC ENZYMES)



THE CARDIOMYOPATHIES

EKG FINDINGS MAY INCLUDE:

- A NORMAL EKG
- PERCARDITIS CHANGES
- CHAMBER HYPERTROPHY (A / V)
- Q / QS COMPLEXES
- POOR R WAVE PROGRESSION
- AV NODAL / BBB (LBBB common)
- ATRIAL FIBRILLATION
- VENTRICULAR COMPLEXES

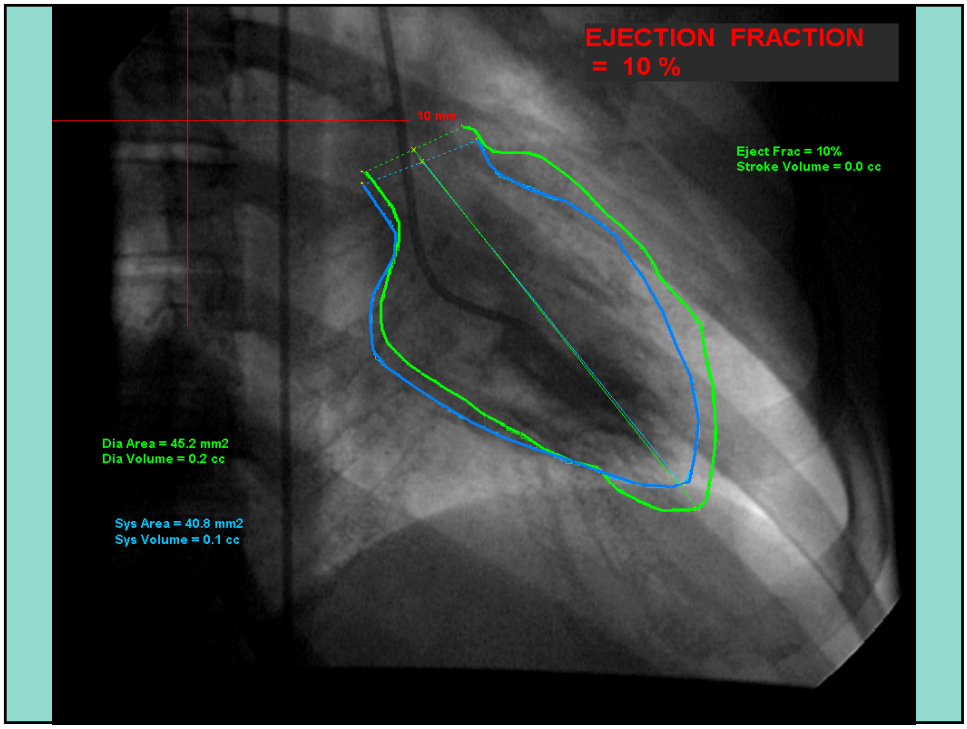


THE CARDIOMYOPATHIES

CASE STUDY :

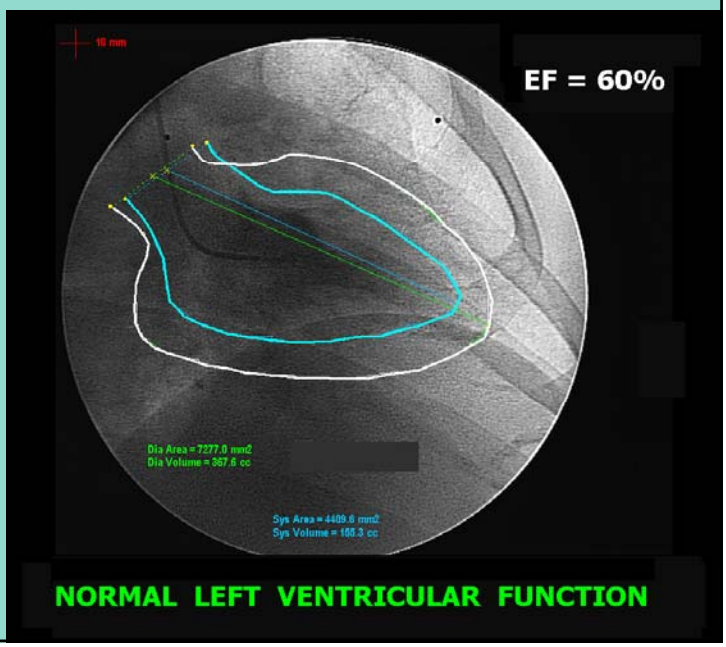
STAT ECHOCARDIOGRAM REVEALED
GLOBAL HYPOKINESIS, EF < 20%.
NO PERICARDIAL EFFUSION NOTED.
NO VALVULAR DYSFUNCTION NOTED.

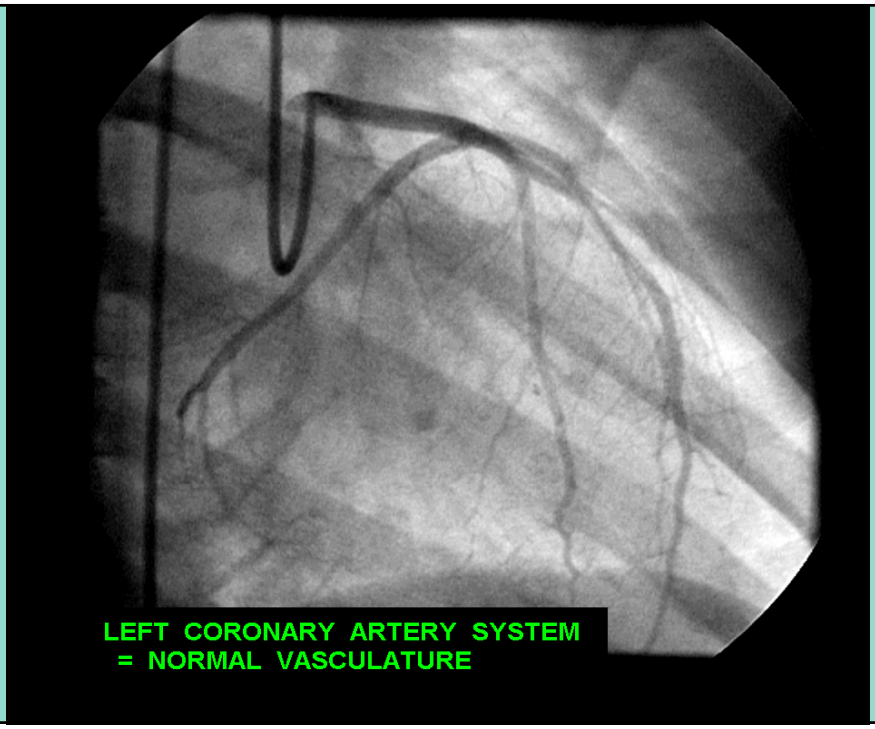
NEXT COURSE OF ACTION ?

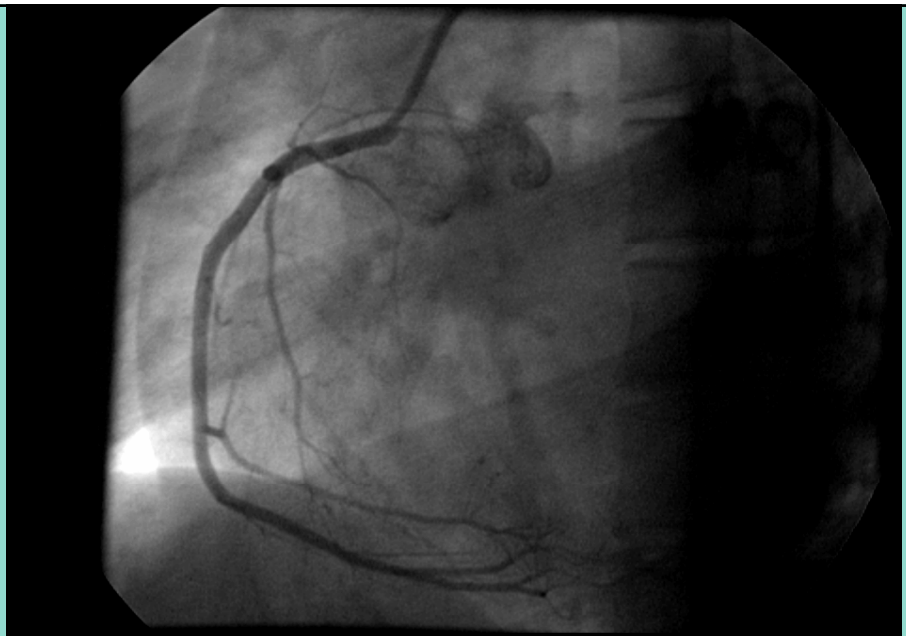


NORMAL EJECTION FRACTION =

55 - 70%







RIGHT CORONARY ARTERY SYSTEM = NORMAL VASCULATURE

THE CARDIOMYOPATHIES

CASE STUDY :

IABP INSERTED DURING CARDIAC CATH.

STAT TRANSFER TO REGIONAL CARDIAC TRANSPLANT FACILITY ORDERED.

PATIENT EXPERIENCED VENTRICULAR TACHYCARDIA and IRREVERSIBLE V-FIB BEFORE HELICOPTER ARRIVAL.

CHAMBER ENLARGEMENT

EKG CHANGES

INCREASE IN CHAMBER SIZE and/or MASS RESULTS IN AN INCREASE IN AMPLITUDE and/or TIME IN ORDER TO ACHIEVE DEPOLARIZATION.

SIMPLY PUT, THE EKG WAVEFORMS ARE BIGGER AND LONGER THAN NORMAL IN CHAMBER ENLARGEMENT.

When ECG Indicators of CHAMBER HYPERTROPHY Are present on the 12 Lead ECG, An ECHOCARDIOGRAM should Be obtained and evaluated to:

- **CONFIRM HYPERTROPHY**
- **DETERMINE ETIOLOGY** (VALVULAR STENOSIS / VALVULAR REGURGITATION vs other etiology) **. . . .**

CHAMBER ENLARGEMENT

SYSTOLIC OVERLOAD

A CONDITION WHERE THE HEART MUST OVERCOME UNUSUAL RESISTANCE TO EJECT BLOOD. THIS RESULTS IN MUSCLE THICKENING, or HYPERTROPHY.

- VALVULAR STENOSIS
- SYSTEMIC HYPERTENSION
- PULMONARY HYPERTENSION
- CONGENITAL ABNORMALITIES

CHAMBER ENLARGEMENT

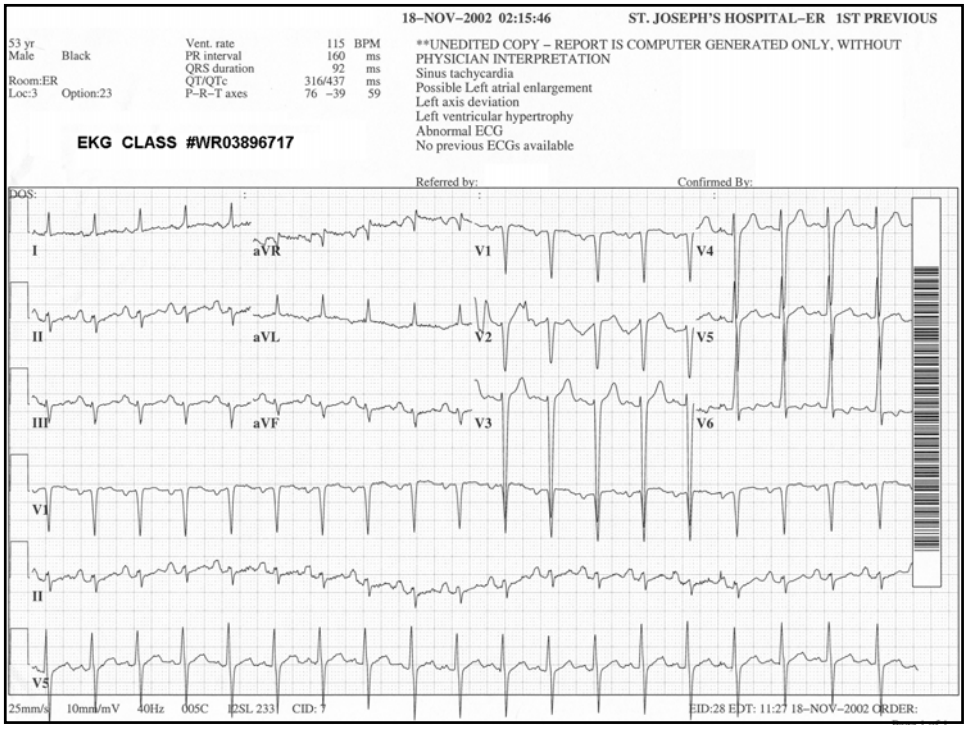
DIASTOLIC OVERLOAD

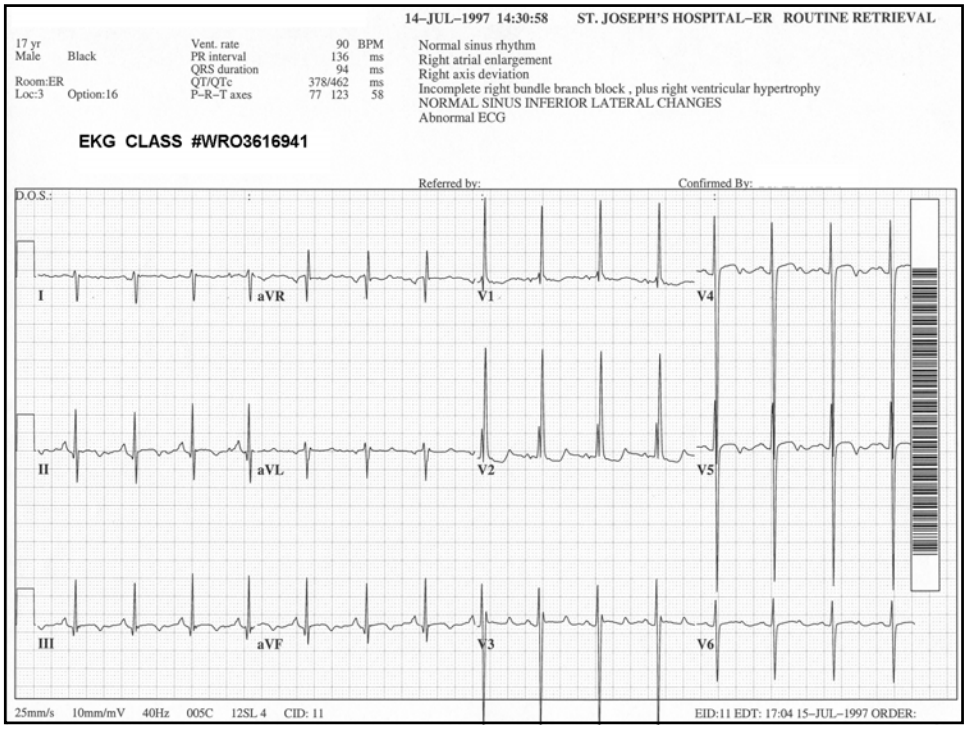
A CONDITION WHERE DURING DIASTOLE, THE CHAMBER IS OVER-ENGORGED BY EXCESSIVE BLOOD VOLUME. THIS RESULTS IN "STRETCHING" or DILATION OF THE CHAMBER.

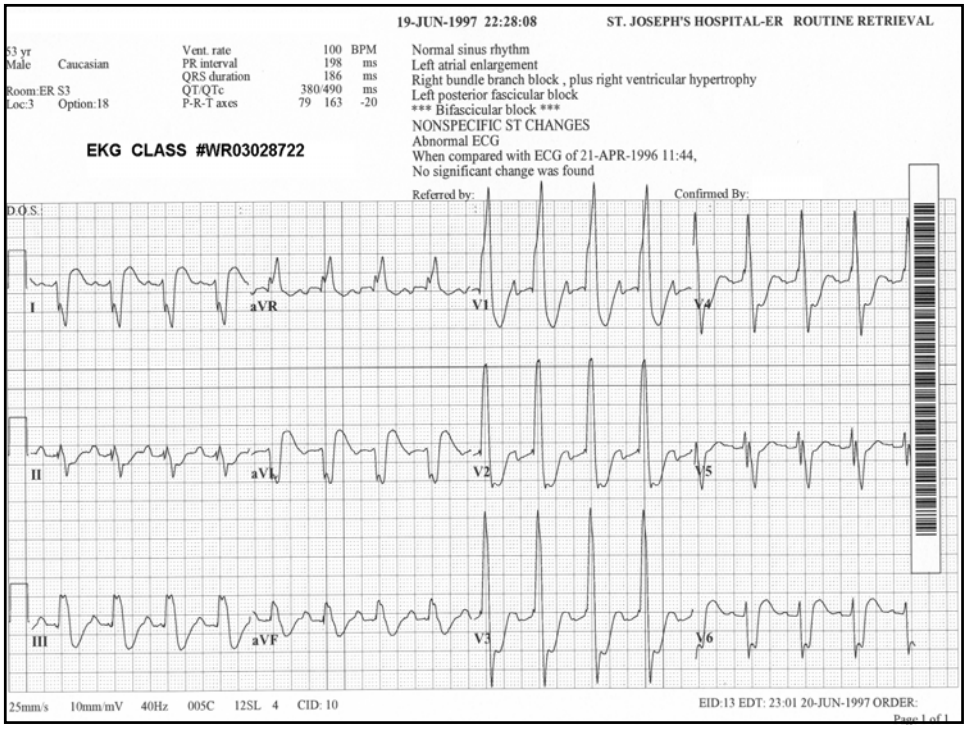
- VALVULAR REGURGITATION
- FLUID VOLUME OVERLOAD

SUSPECT HYPERTROPHY WHEN:

QRS COMPLEXES "SPEAR THROUGH" OTHER LEADS !



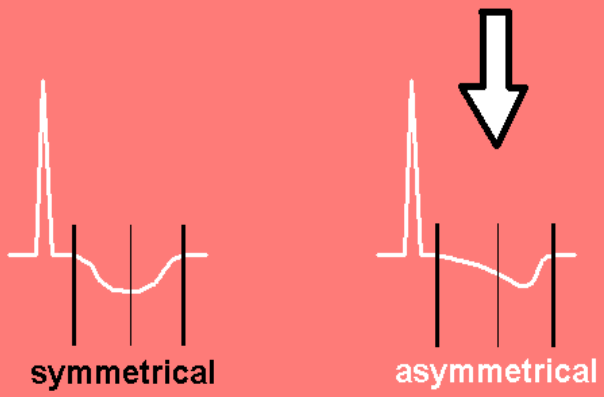

14-JUL-1997 14:30:58 ST. JOSEPH'S HOSPITAL-ER ROUTINE RETRIEVAL
17 yr
Male Black
Room:ER
Loc:3 Option:16
Vent. rate 90 BPM
PR interval 136 ms
QRS duration 94 ms
QT/QTc 378/462 ms
P-R-T axes 77 123 58
Normal sinus rhythm
Right atrial enlargement
Right axis deviation
Incomplete right bundle branch block , plus right ventricular hypertrophy
NORMAL SINUS INFERIOR LATERAL CHANGES
Abnormal ECG
EKG CLASS #WRO3616941
Referred by:
Confirmed By:
D.O.S.:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 11
EID:11 EDT: 17:04 15-JUL-1997 ORDER:


19-JUN-1997 22:28:08 ST. JOSEPH'S HOSPITAL-ER ROUTINE RETRIEVAL
53 yr
Male Caucasian
Room:ER S3
Loc:3 Option:18
Vent. rate 100 BPM
PR interval 198 ms
QRS duration 186 ms
QT/QTc 380/490 ms
P-R-T axes 79 163 -20
Normal sinus rhythm
Left atrial enlargement
Right bundle branch block , plus right ventricular hypertrophy
Left posterior fascicular block
*** Bifascicular block ***
NONSPECIFIC ST CHANGES
Abnormal ECG
When compared with ECG of 21-APR-1996 11:44,
No significant change was found
EKG CLASS #WR03028722
Referred by:
Confirmed By:
D.O.S.:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 10
EID:13 EDT: 23:01 20-JUN-1997 ORDER:
Page 1 of 1

CHAMBER ENLARGEMENT

VENTRICULAR STRAIN PATTERNS

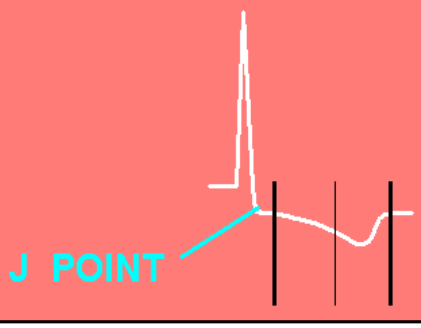
- T-WAVES ARE INVERTED and ASYMMETRICAL



CHAMBER ENLARGEMENT

VENTRICULAR STRAIN PATTERNS

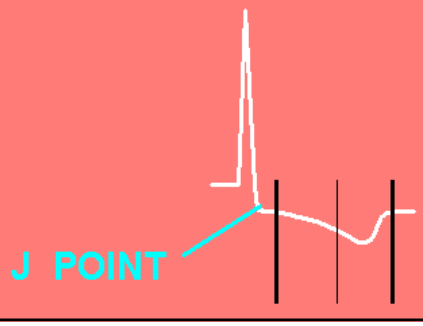
- T WAVES ARE INVERTED AND ASMMETRICAL
- THERE MAY BE S-T SEGMENT DEPRESSION



CHAMBER ENLARGEMENT

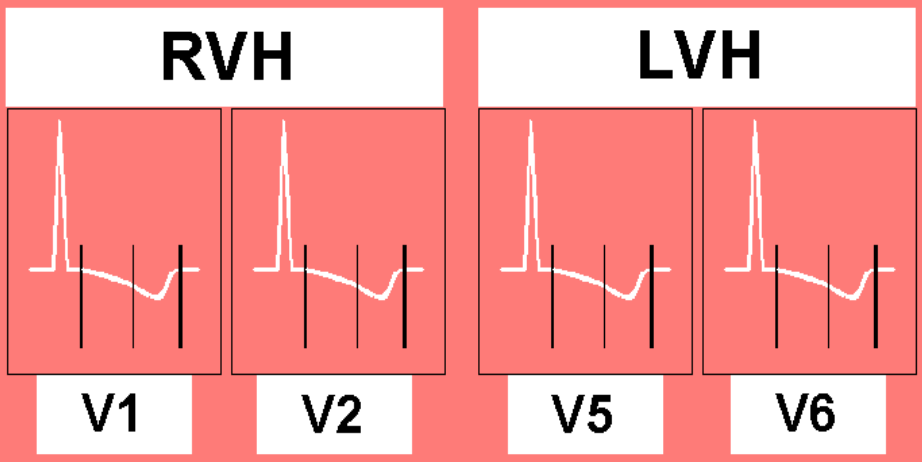
VENTRICULAR STRAIN PATTERNS

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



CHAMBER ENLARGEMENT

VENTRICULAR STRAIN PATTERNS



CHAMBER ENLARGEMENT

VENTRICULAR ENLARGEMENT

I USE SEVERAL TECHNIQUES FOR IDENTIFYING RIGHT AND LEFT VENTRICULAR HYPERTROPHY.

1. AXIS OF LEAD I and V1
2. PRESENCE OF ATRIAL HYPERTROPHY
3. R-WAVE PROGRESSION OF V LEADS
4. STRAIN PATTERN OF T WAVES IN V1 - V2 and V5 - V6
5. MATHEMATICAL FORMULAS

CHAMBER ENLARGEMENT

*MATHEMATICAL FORMULAS FOR DETERMINING LVH and RVH

LVH

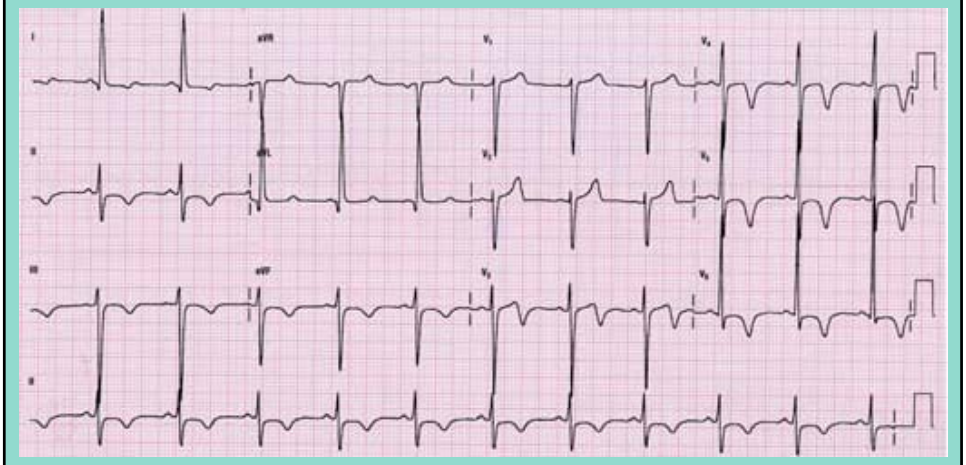
- R-WAVE V1 + S-WAVE LEAD III > 25mm
- R-WAVE V5 or V6 > 26mm
- S-WAVE V1 + R-WAVE V5 or V6 > 35mm
- LARGEST R-WAVE + LARGEST S-WAVE in V-LEADS > 45mm

RVH

- R-WAVE V1 + S-WAVE V5 or V6 > 10.5mm
- rSR' in V1 where R' ≥ 10mm

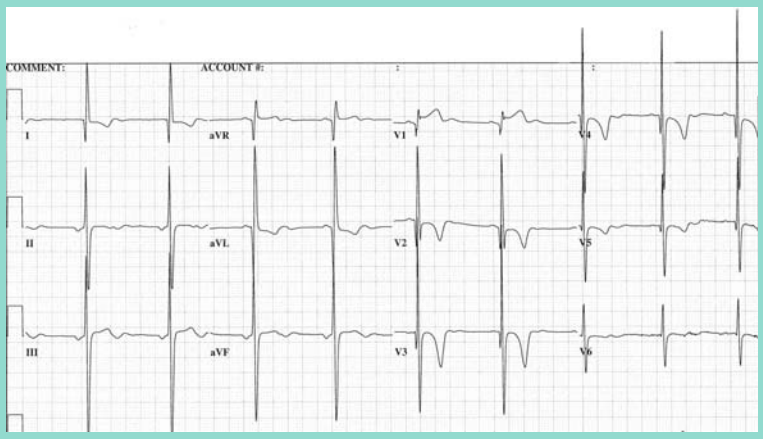
* THIS IS A PARTIAL LIST.

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 (INFERIOR, ANTERIOR, LATERAL)**
- **T WAVES are SYMMETRICAL (No STRAIN PATTERN noted).**

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 (No STRAIN PATTERN noted).**

Typical 12 Lead ECG Indicators Suggestive of HCM:

LVH
Inverted T waves (predom V4-V6, II, aVF, I, L)
ST Depression / Strain pattern
Q waves
Left Axis Dev
LAE

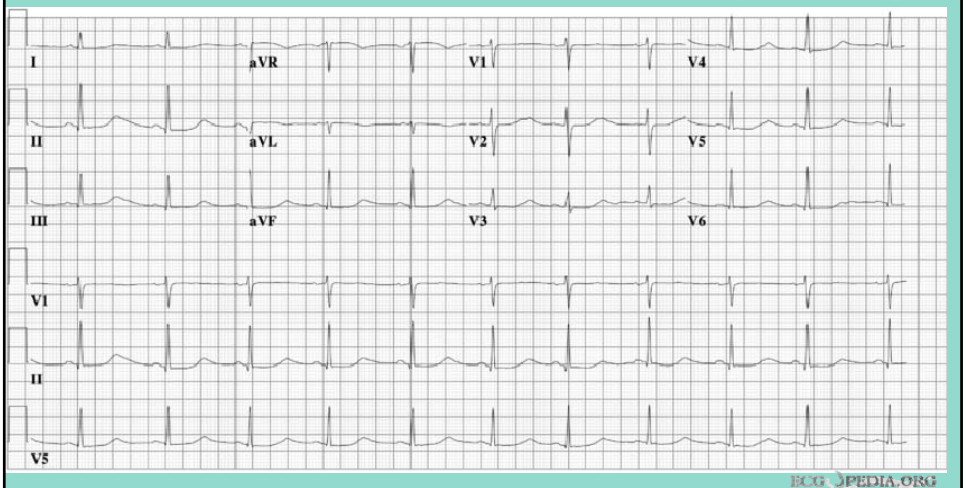
Over 90% of patients with HCM will have an abnormal ECG.[19–21] ECG abnormalities include T wave inversion (TWI), ST segment depression, pathological Q waves, conduction delay, left-axis deviation (LAD) and left atrial enlargement (LAE).

In a patient with KNOWN Hypertrophic Cardiomyopathy, OR a patient with NO PREVIOUS DIAGNOSIS OF HCM who presents with an ECG like the one on the previous slide, what DIAGNOSIS of HCM . . . . . .

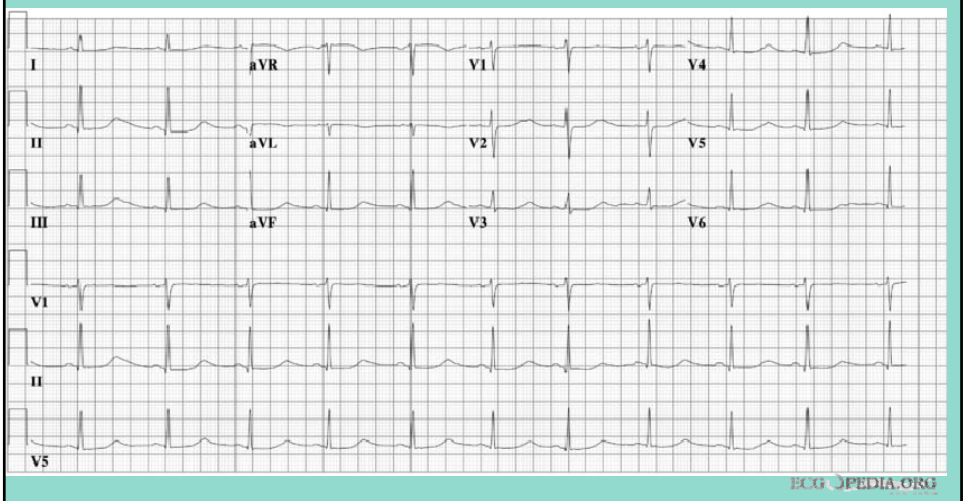
. . . . . WHAT DIAGNOSTIC TEST would be most appropriate to order next ? ? ?

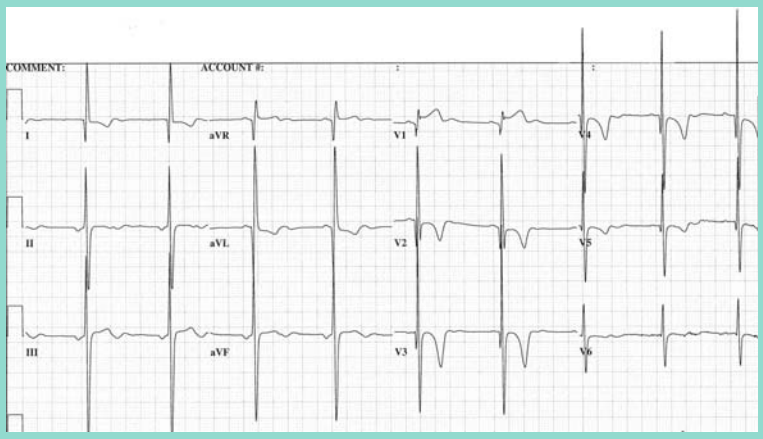
(HINT….we want to assess Left Ventricular function !)

QUIZ TIME !

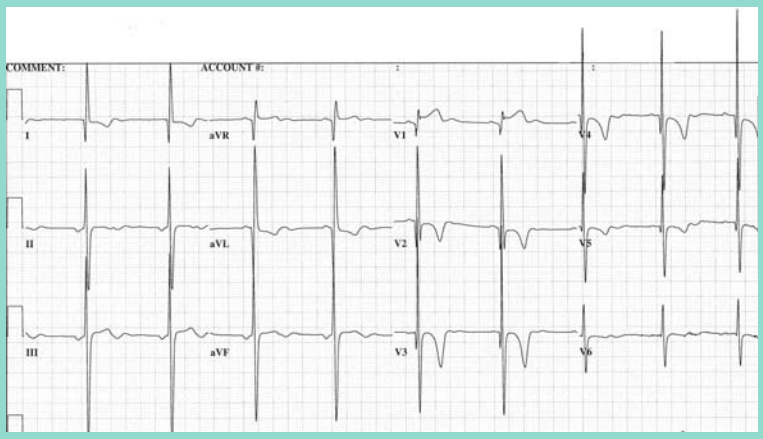


LQTS Type I . . . .what triggers Torsades in patients with this type of LQTS ?





Hypertrophic Cardiomyopathy (HCM)



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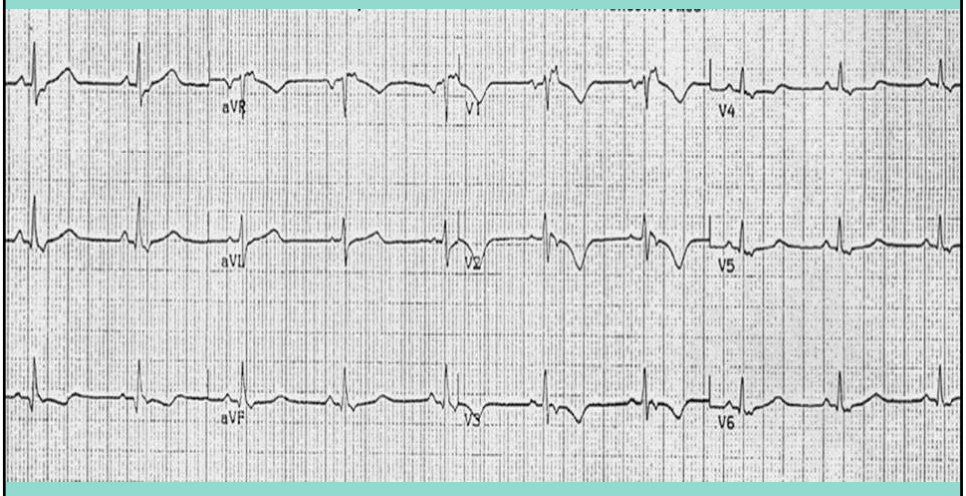
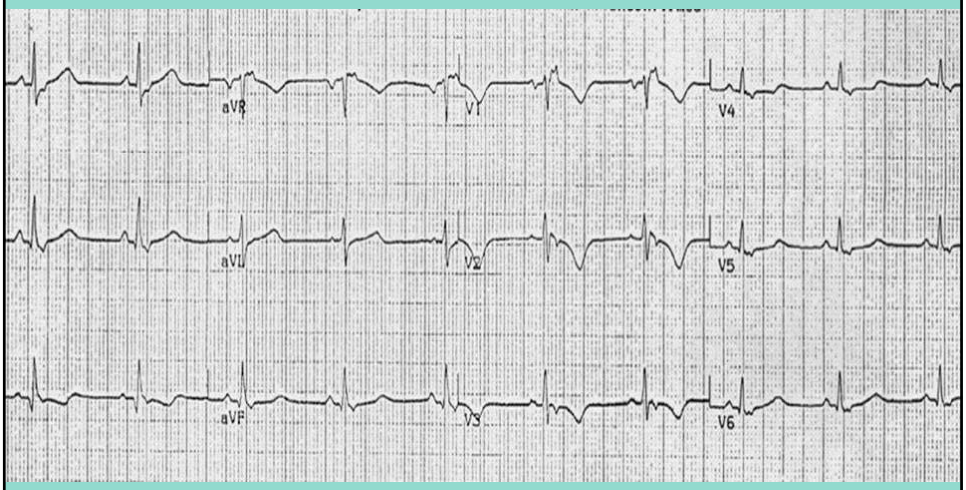


1. **"Incomplete RBBB" Pattern**
2. **V1, V2 Rs pattern**
3. **Inverted T waves, symmetrical, - Global**
4. **Epsilon's waves**

ARVD



1. "Incomplete RBBB" Pattern
2. V1, V2 Rs pattern
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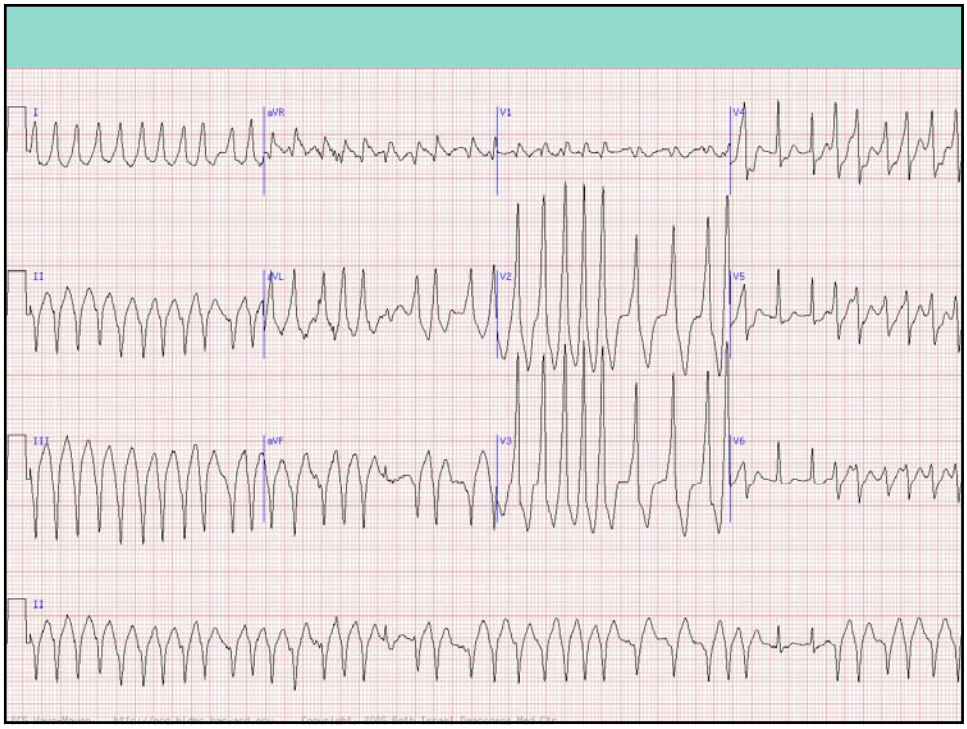
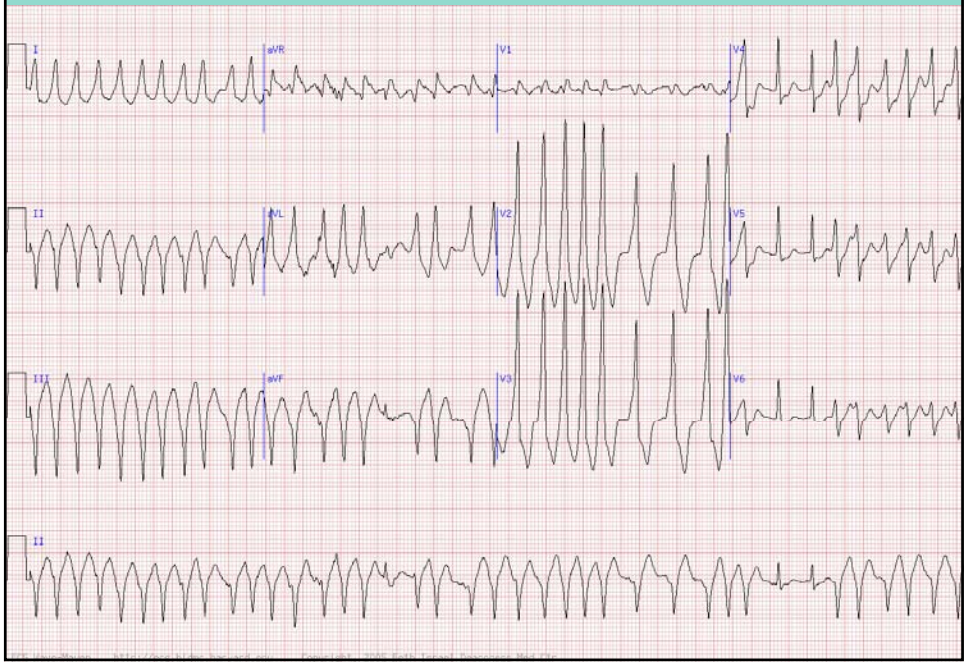
4. Epsilon's waves



W-P-W with Afib & RVR



What CLASSIFICATIONS of MEDS do we NEVER give patients with W-P-W and A-Fib??

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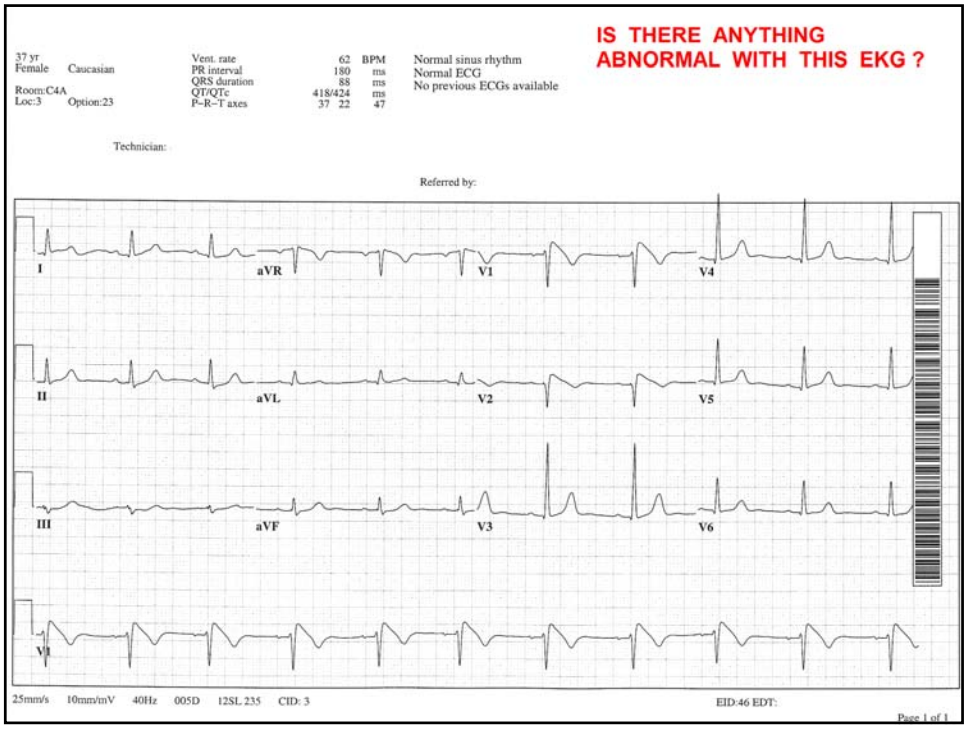
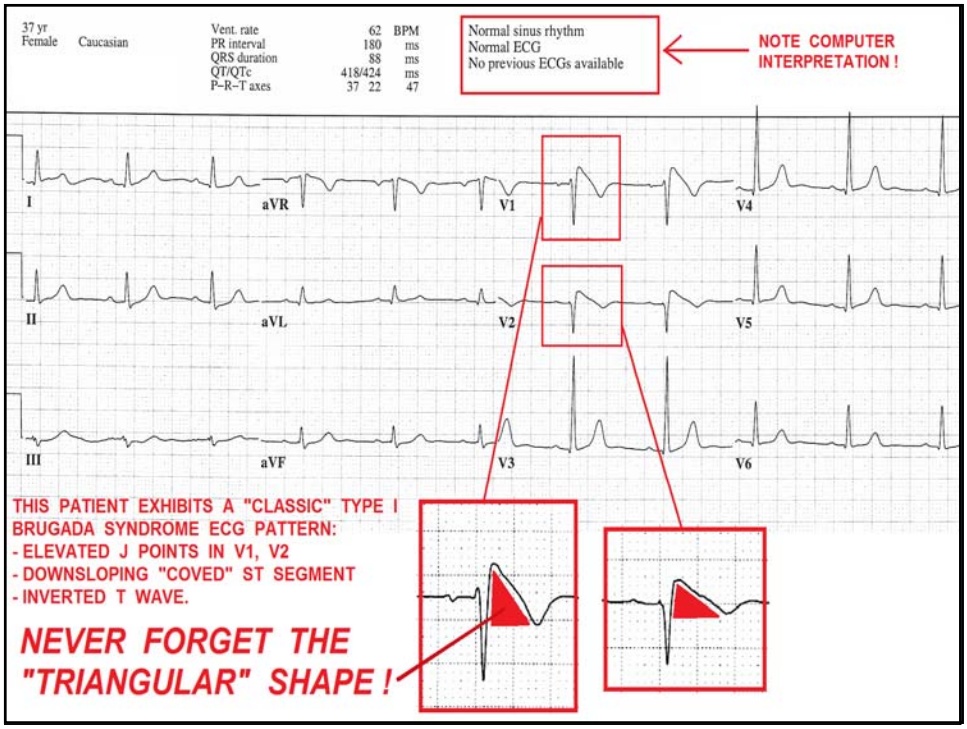
AV Nodal Blocking agents –
-NO Calcium Channel Blockers
-NO Adenosine
-AMIODARONE Contraindicated!

What CLASSIFICATIONS of MEDS do we NEVER give patients with W-P-W and A-Fib??

AV Nodal Blocking agents –
-NO Calcium Channel Blockers
-NO Adenosine
-AMIODARONE Contraindicated!
. . . .and WHATEVER ELSE Will says !!!!!! ☺


IS THERE ANYTHING ABNORMAL WITH THIS EKG ?
37 yr
Female Caucasian
Room:C4A
Loc:3 Option:23
Vent. rate 62 BPM
PR interval 180 ms
QRS duration 88 ms
QT/QTc 418/424 ms
P-R-T axes 37 22 47
Normal sinus rhythm
Normal ECG
No previous ECGs available
Technician:
Referred by:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
25mm/s 10mm/mV 40Hz 005D 12SL 235 CID: 3
EID:46 EDT:
Page 1 of 1


37 yr
Female Caucasian
Vent. rate 62 BPM
PR interval 180 ms
QRS duration 88 ms
QT/QTc 418/424 ms
P-R-T axes 37 22 47
Normal sinus rhythm
Normal ECG
No previous ECGs available
NOTE COMPUTER INTERPRETATION !
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
THIS PATIENT EXHIBITS A "CLASSIC" TYPE I BRUGADA SYNDROME ECG PATTERN:
- ELEVATED J POINTS IN V1, V2
- DOWNSLOPING "COVED" ST SEGMENT
- INVERTED T WAVE.
NEVER FORGET THE "TRIANGULAR" SHAPE !

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

Wayne W Ruppert, 352-521-1544 wayne.ruppert@bayfronthealtth.com