Presented by the Society of Cardiovascular Patient Care
19th CONGRESS MIAMI
May 25-27,2016
scpc.org/Congress

Stayin' Alive: Sudden Cardiac Death Athletic Pre-participation Screening

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DOWNLOAD ACS & STEMI - St Joseph's Hospital Presentation

Download 2011 HRS Perioperative Management of CIEDs

Wayne Ruppert: Bio

- Developed curriculum for and instruct "ECG Identification of Sudden Arrhythmia Death Syndromes," 4 hour course (approved for 4.0 CE credits FL Board of Nursing)
- Interventional Electrophysiology Technologist, St Joseph's Hospital Pediatric Cardiology Program, 1999-2009
- State of Florida Board of Nursing approved CE Provider (CE Broker #50-12998)

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

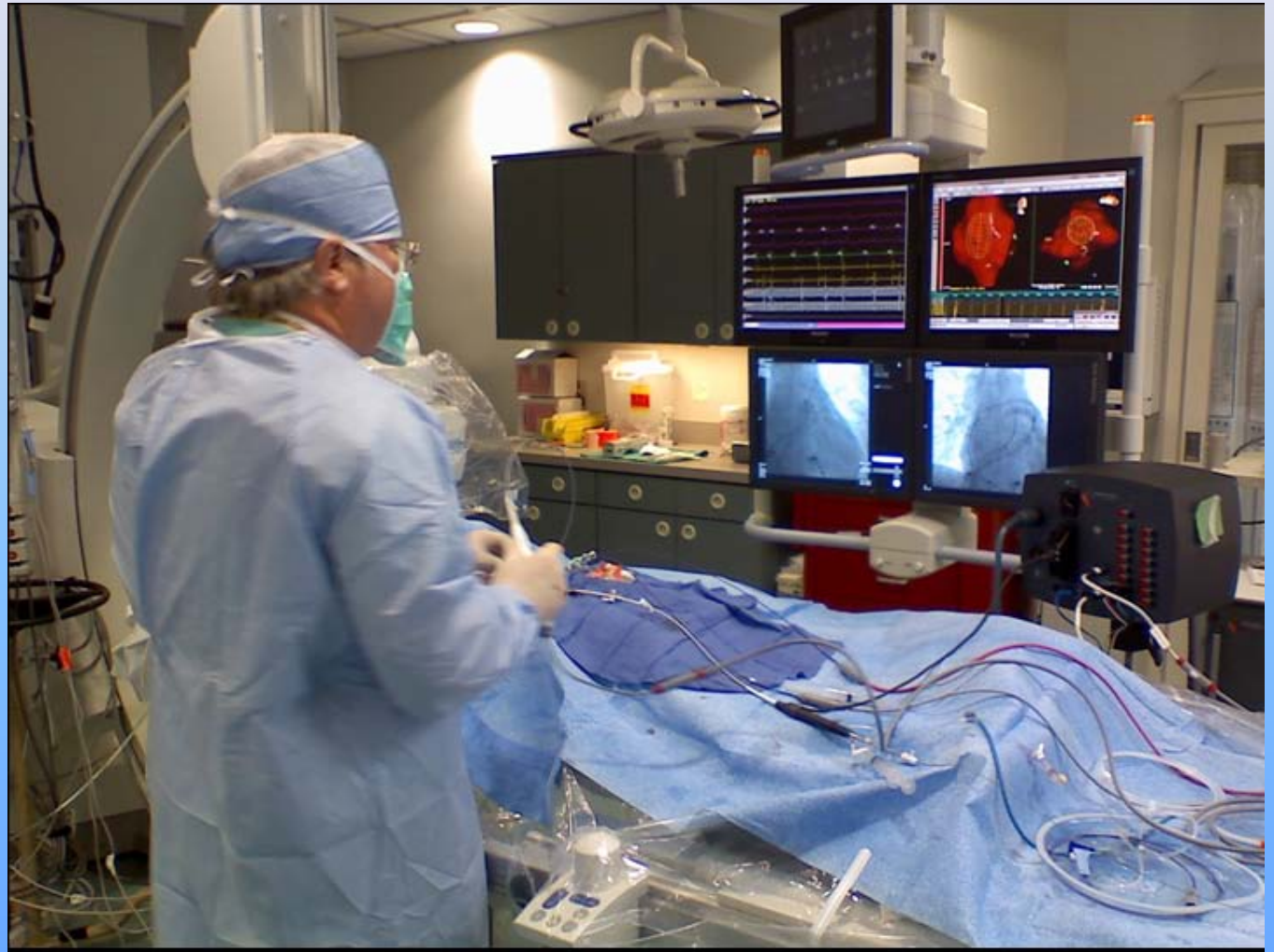
Other Reference Sources:

www.JACC.org

http://circ.ahajournals.org/

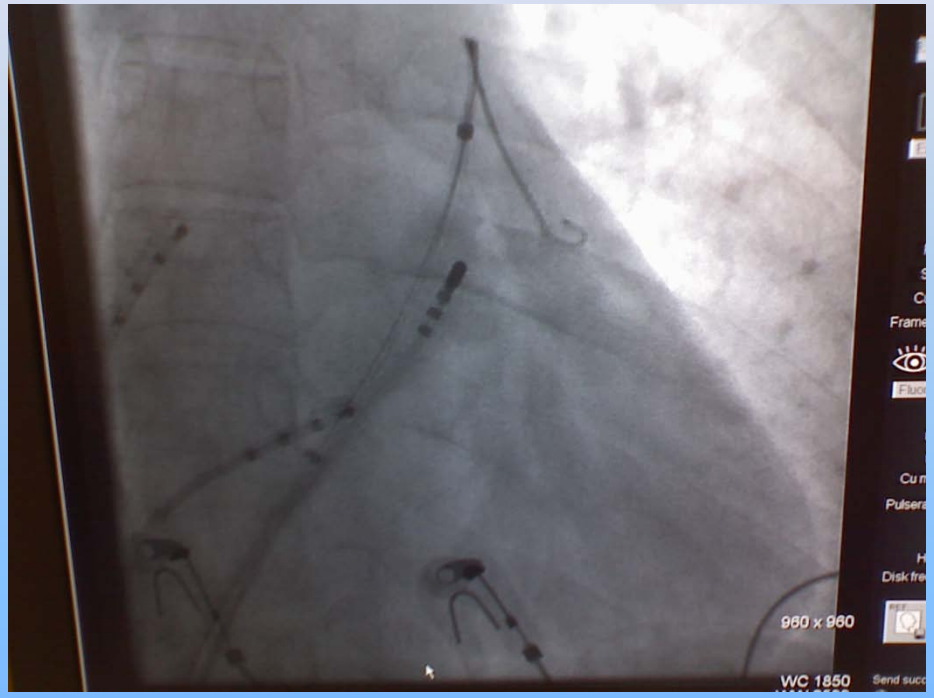
www.SADS.org

Other Reference Sources:
Electrophysiology (EP) Lab Case Studies

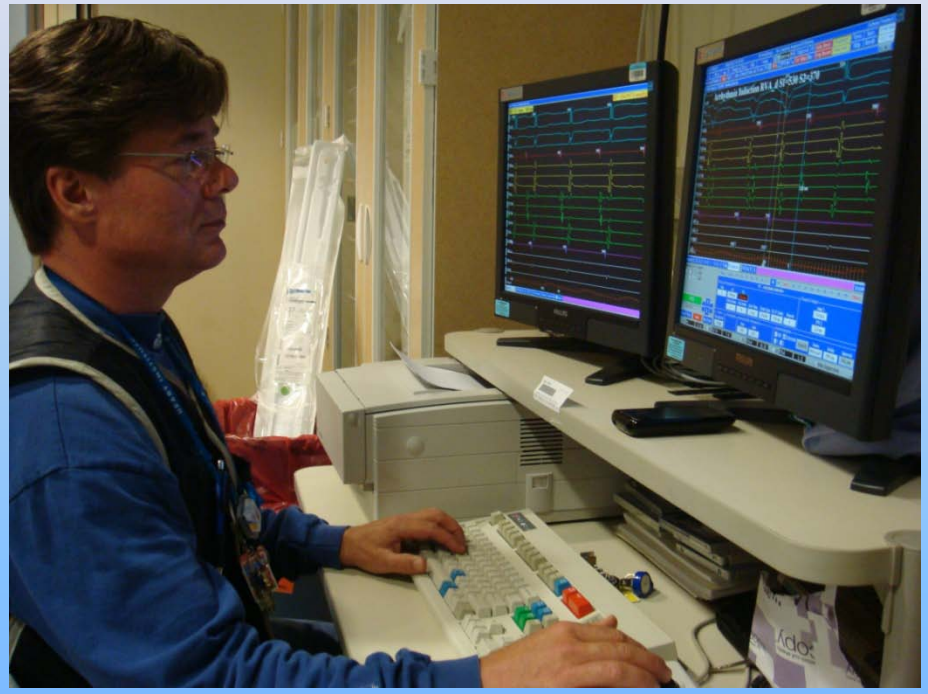


James Irwin, MD, FACC, FHRS performs an EP study and Ablation at St. Joseph's Hospital in Tampa, FL, in 2008

Electrophysiology Lab Case Studies



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



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

Sudden Arrhythmia Death Syndromes (SADS)

CARDIAC ARREST PREVENTION:

The identification and management of conditions associated with Sudden Cardiac Death in the pediatric and young adult population

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*

Prevalence

Sudden Deaths in Young Competitive Athletes

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

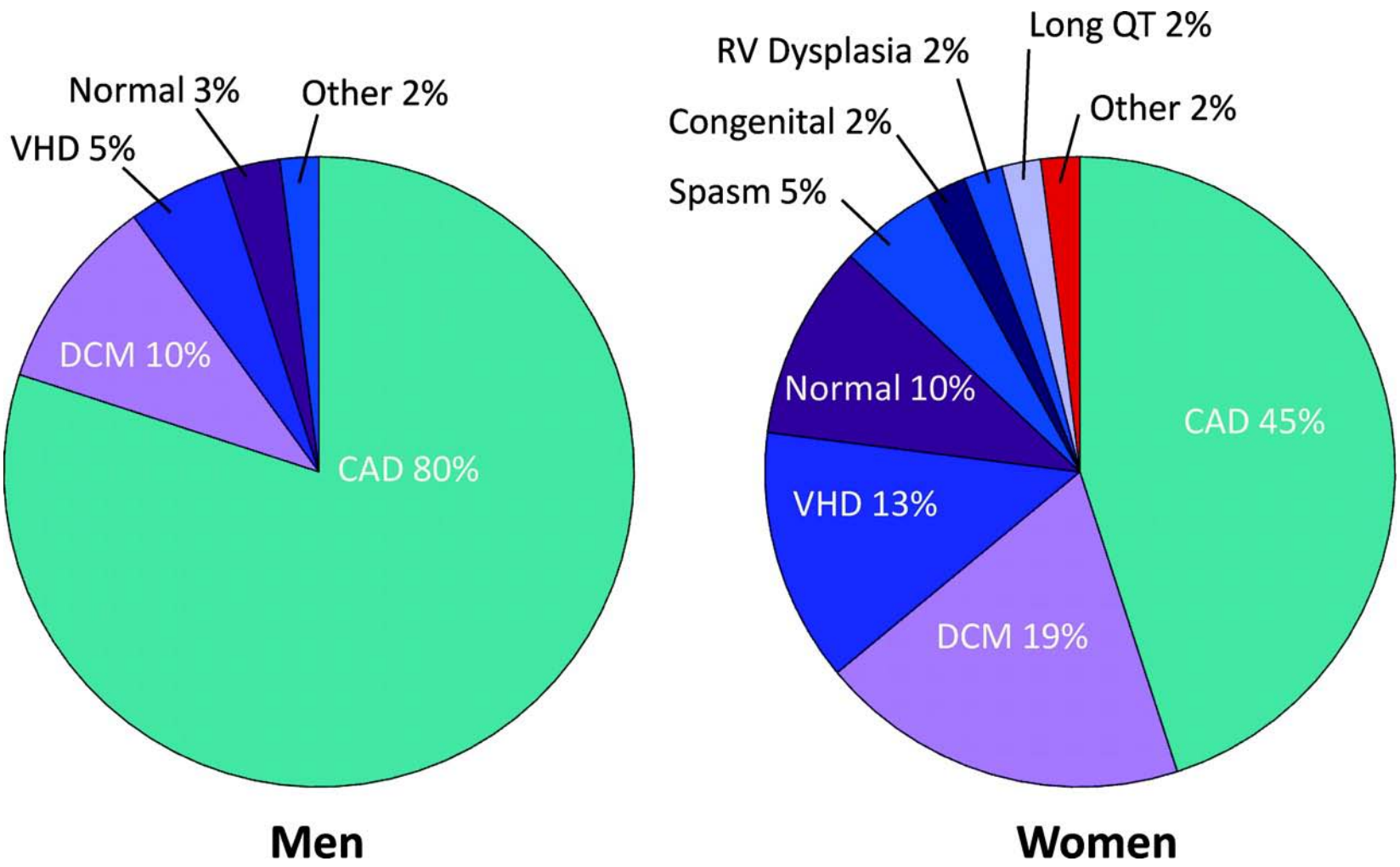
Analysis, causes of 1866 Deaths in the US, 1980 –2006:

- **Cardiovascular: 56%**
- **Traumatic: 22%**
- **Commotio Cordis: 3%**
- **Heat Stroke: 2%**
- **Other: 17%**

Estimated SADS Prevalence in US Population:

- HCM: 1/500 *J Am Coll Cardiol*. 2014;64
- BrS: 1/2,500 SADS Foundation
- LQTS: 1/2,500 Lenhart,SE 2007 AHA Circ
- ARVD: 1/10,000 SADS Foundation
- CPVT: 1/10,000 US Nat'l Library of Medicine
- WPW: 1/1,000 Circulation.2011; 124: 746-757

Structural heart disease in cardiac arrest survivors.



Rajat Deo, and Christine M. Albert Circulation. 2012;125:620-637

Prevalence

Adverse Drug Reactions: Torsades de Pointes secondary to QT prolonging medications:

- Occur in and out of hospital
- Underreported
- Medical community undereducated
- 7,000 in-hospital ADRs / year (all cause)
- Major issue with pharmaceutical industry, many drugs removed from market due to high incidence of TdP and TdP associated mortality

Compared to sudden death from CAD, SADS mortality prevalence is low, HOWEVER . . . .

- Nearly EVERY SADS death is a NEEDLESS TRAGEDY that could have been AVOIDED with appropriate screening and management.
- Many SADS victims are infants, children and young adults who are otherwise healthy.
- Sudden death is often the first symptom of SADS
- Diagnosed and managed properly, SADS patients can live long, productive and happy lives

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. Marfans, Ehlers-Danlos, Loeys-Dietz Syndromes)

SADS Conditions
Etiology and Mechanisms:

- **Genetic (Inherited and New Mutation)** (LQTS, SQTS, Brugada, HCM, CPVT)
- **Acquired (Medication Induced and Other Causes)** (LQTS)
- **Structural** (HCM, ARVD, WPW)
- **Ion Channelopathies (Ion exchange at Cellular Level, effects Depolarization / Repolarization)** (LQTS, Brugada, CPVT)

Dysrhythmias Associated with Mortality in SADS Conditions:

- **Torsades de Pointes** (LQTS, Brugada)
- **Monomorphic VT, deteriorates to VF** (CPVT, ARVD, SQTS, HCM)
- **Bi-Directional Polymorphic VT, deteriorates to multifocal Polymorphic VT then VF**: (CPVT)
- **Ventricular Fibrillation** (any SADS condition)
- **Supraventricular and Wide QRS Tachycardias deteriorating to VF** (HCM, WPW)

Common SADS Dysrhythmia Triggers:

- Physical Exertion
  - Athletes
  - Military Personnel
  - Anyone who engages in physically demanding activity
- Emotional Duress
- Adrenergic Stimulation
- QT Prolonging Medications / Substances

Gaining Support and Recruiting Volunteers for your SCDP Program;

The emotional aspect of presenting the raw truth:

From Headline News, the Victims of SADS Conditions . . . .

High School Athlete Dies After Collapsing At Practice

August 15, 2011 11:28 PM

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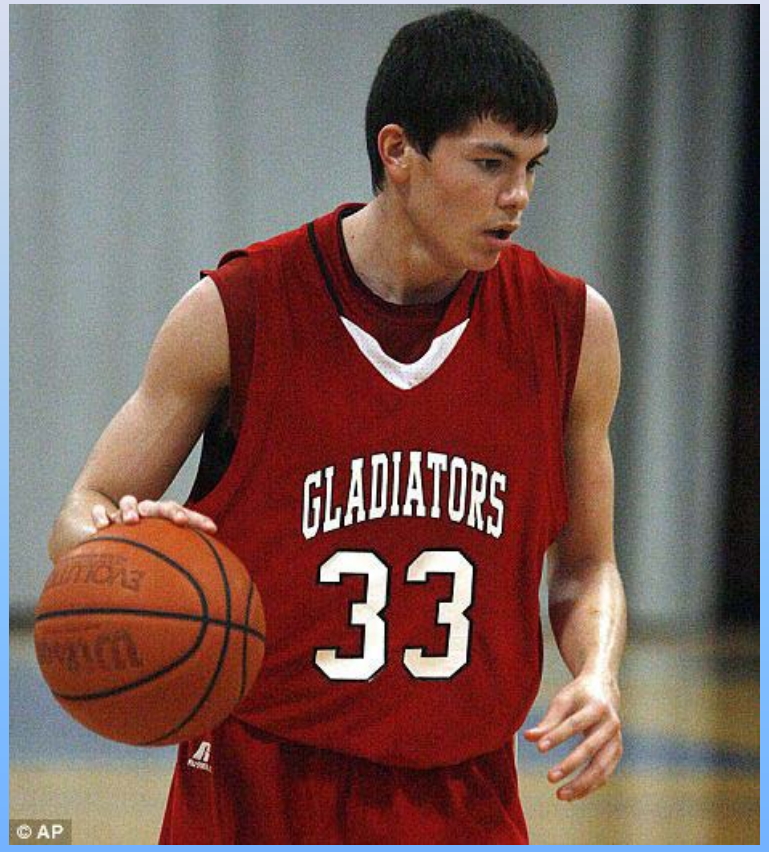
Teen basketball player collapses and dies on court - third school boy sportsman to do so in less than a month



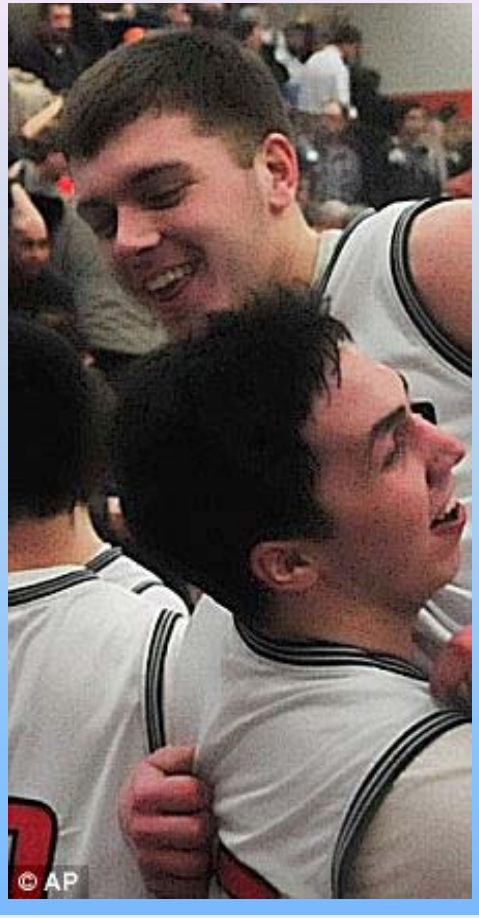
By DAILY MAIL REPORTER
UPDATED: 12:03 EST, 14 March 2011

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.

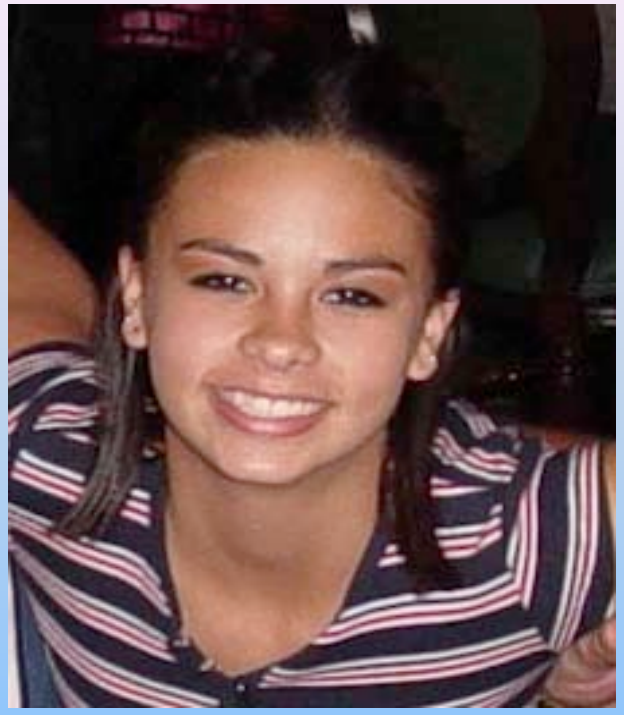
Greg Moyer, 15

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."***

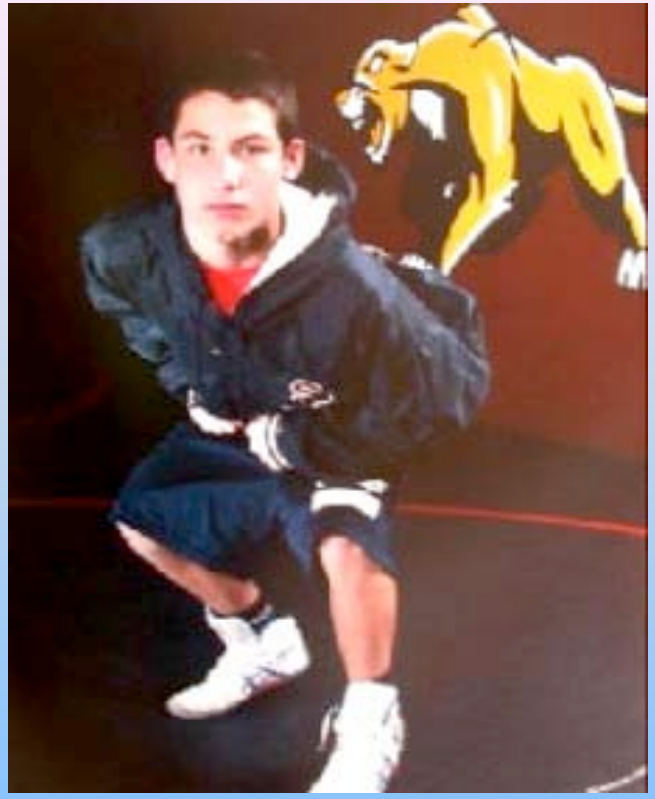
High school quarterback Reggie Garrett threw his second touchdown pass of the night, walked off the field, and collapsed from sudden cardiac arrest. He died in the ambulance on the way to the hospital in West Orange, Texas.

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 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 free electrocardiograms (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.*

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 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.

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

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)

A Scientific Statement From the American Heart Association and the American College of Cardiology

Endorsed by the Pediatric and Congenital Electrophysiology Society and American College of Sports Medicine

"It is underscored that the present report is not limited in scope to universal mass screening for athlete populations but importantly includes considerations for screening large, young, and truly general populations (school-aged, 12–25 years old, of both sexes)."

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)

A Scientific Statement From the American Heart Association and the American College of Cardiology

Endorsed by the Pediatric and Congenital Electrophysiology Society and American College of Sports Medicine

"It appears only logical and fair that when relatively small athlete populations are targeted for screening, at least some consideration should be given to extending this screening to nonathletes in the same jurisdiction and venue."

In consideration of the message from the 2014 AHA/ACC Scientific Statement conveyed in the last two slides, we are presenting the blueprint for a program that extends BEYOND the athletic field to an entire community . . . .

Elements of Community Based

Sudden Cardiac Death Prevention (SCDP) Programs

&

SCDP Medical Centers of Excellence

Wayne W Ruppert, CVT, CCCC, NREMT-P
Cardiovascular Coordinator
Bayfront Health Dade City

SCDP Program Development Strategy:

National level sponsorship (e.g.: ACC/SCPC, AHA, HRS) would charter a committee whose mission is to:

- Develop the structure of a community model for SCDP programs
- Develop evidence-based best practices for SCDP programs
- Promote professional and public awareness of SADS Conditions
- Monitor, measure and provide feedback as to the effectiveness of SCDP programs at local and national levels.

SCDP Program Development Strategy:

SCDP National Committee membership (partial list):

- Industry leading physician(s) in SADS research, etiologies, diagnosis, treatment, prevention
- ACC / AHA / HRS /
- Hospital industry leadership
- National EMS subject matter experts (SMEs)
- Government
- Pharmaceutical industry
- Colleges / Schools
- Sports Medicine

SCDP Program Development Strategy:

National level SCDP program sponsorship, continued:

- Provide communication network to facilitate collaboration between local community programs.
- Utilize a national data sharing network for the purpose of refining current best practices and research. This could be achieved by:
  - Merging with NCDR registries
  - Solicit input from regional and local program coordinators gain insight into most efficient and effective methods for data collection and management.

SCDP Program Development Strategy:

- **Local Level:** SCDP programs should be a collaborative community partnership consisting of:
  - Physician Medical Director
  - Hospital
  - Cardiologists / Electrophysiologists
  - Schools, Colleges, Athletic Programs
  - EMS (new “Community EMS Model” ideal)
  - Local Government
  - News Media

SCDP Program Development Strategy:

- **Recruit Volunteers**
  - Physicians
  - Cardiovascular and Echo Technologists
  - Nurses
  - School Administrators / College Deans
  - School Teachers / College Professors
  - Coaches
  - Civilians

Your SCDP Program; Elements for Success:

- Stick to Science: use evidence-based criteria
- Use available resources (e.g. “this presentation”)!!
- Establish collaborative partnerships with others in your community.
- Select SCDP Team members who SHARE the PASSION for this subject
- Use established models (e.g. Johns Hopkins Heart Hype program).

Key Elements of a comprehensive SCDP Program:

- **Coordinated Community SADS Screening Programs** that include:
  - A collaborative partnership between Schools, Colleges, Athletic Organizations, Doctor's Offices, Fire/EMS Stations and Hospitals to conduct Athletic Pre-Participation and NON-athletic screening at select locations.

Key Elements of a comprehensive SCDP Program:

- **Community Education and Integration:**
  - Public awareness of SADS conditions:
    - Symptoms of SADS Conditions
    - Etiologies (i.e.: genetics, acquired)
    - Exacerbation factors (i.e.: medications, physical activities)
    - Medical Centers of Excellence for SADS diagnosis and treatment
  - CPR/AED training programs
  - Community based CPR/AED Responder teams
  - AEDs and chest protection in schools/colleges, and at all sporting events)

"Neighbors-Saving-Neighbors"

CPR + AED Responder Program

Innovative program has saved over a dozen lives in Florida:

- Community responders taught hands-on CPR and AED operations (download curriculum)
- AED located centrally in community
- Responders are dispatched by County 911 for cardiac arrests

- ***Responders on scene, start CPR and administer AED therapy; average response time < 2 minutes.***

Innovative CPR Training:
"CPR-Training-on-the-Go" machines, DFW airport

Key Elements of a comprehensive SCDP Program:

- **Patient Education:**
  - Education specific to their SADS diagnosis
  - Importance of taking meds prescribed for their SADS condition
  - Medications to avoid
    - Prescription
    - Over-the-Counter
    - Illegal drugs
  - Activities to avoid
  - Other diagnosis-specific considerations
  - Support organizations and community resources

Key Elements of a comprehensive SCDP Program

- **Athletic Programs; Colleges, Schools, Athletic Organizations:**
  - **Education for Administrators, Teachers, Coaches**
  - **Screening** (Athletic Pre-Participation and NON-athletic screening in schools, doctor's offices, hospitals, fire / EMS stations)
  - AEDs in all athletic areas
  - Chest protection for baseball and other sporting events with high probability of chest trauma
  - "Safety Baseballs," especially for younger athletes

Key Elements of a comprehensive SCDP Program

- **Education for Physicians, Pharmacists, ARNPs/PAs, Paramedics, EMTs** and other Healthcare providers in:
  - SADS Awareness
  - ECG Indicators of SADS conditions:
    - LQTS, SQTS (including U wave criteria)
    - Hypertrophy (including typical HCM presentation)
    - Brugada Syndrome (3 types and concealed)
    - WPW (Delta wave, short P-R, QRS wide base, narrow apex)
    - ARVD (Epsilon's wave and typical ECG presentation)
  - QT prolonging medications

Key Elements of a comprehensive SCDP Program

- **Hospitals: Physician Order Sets, Policies and Procedures**
  - Implementation of QT Interval Monitoring protocols (both ECG and QT medications).
  - Emergency Department and Admission order sets for Syncope, near-syncope, Palpitations and Seizures that include diagnostic pathways for ruling out SADS conditions
  - Flowcharts that illustrate the process for evaluating patients with syncope and seizures of unknown etiology

Bayfront Health Dade City

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:											
A	389	400									
B	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				
H			390	420							
I			416	430							
J			400	400							
K			435								
L			410	400	430	410	440	420	478	430	
M					510						
N					480						
O					470						
P					500						
Q						400	420	400	413		
R						440					
S						430	440	460			
T							400	480			
U								430			
V									491		
W									441	440	440
X											530
Y											460
Z											390

QTc	Men	Women
Abnormal	>450	>460
Panic	500+	500+

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):1639-1650

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"

Key Elements of a comprehensive SCDP Program

- **Hospitals: Quality Improvement Processes**
  - Registry/database/spreadsheet for tracking patients presenting to the ED with SADS conditions:
    - Specific SADS diagnosis
    - Previously known or unknown condition
    - Patient presentation: symptoms, ECG rhythm, whether cardiac arrest occurred
    - Hospital course of treatment
    - Patient outcome
    - Follow-up care

Key Elements of a comprehensive SCDP Program

- **Hospitals: Quality Improvement Processes**
  - Flowcharts that illustrate the process for evaluating patients with syncope and seizures of unknown etiology
  - Monitoring and tracking incidence of medication induced QT prolongation
  - Monitoring and tracking incidence of in-house cardiac dysrhythmias and cardiac arrest secondary to medication induced QT prolongation

Key Elements of a comprehensive SCDP Program

- **Hospitals: Cardiac Diagnostic and Interventional Services**
  - Qualified Electrophysiologists and Cardiologists on staff
  - Common diagnostics: MRI, EST, Echo, CT
  - EST protocol for LQTS (Schwartz)
  - Cardiac Catheterization and Electrophysiology Labs (on-site or transfer agreements)
  - Ablation procedures for Wolff-Parkinson-White Syndrome patients

Key Elements of a comprehensive SCDP Program

- **Hospitals: Cardiac Diagnostic and Interventional Services, Continued:**
  - Alcohol Septal Ablation
  - Ablation procedures: Brugada Syndrome
  - ICD implantation
  - Left cardiac sympathetic denervation (LCSD)
  - Cardiac biopsy
  - Surgical Myectomy
  - Genetic testing

Resources for a comprehensive SCDP Program

- Link to SADS Foundation – list of multiple Genetic Testing organizations.

www.SADS.org

Click here for SADS Foundation resources for dealing with insurance companies in paying for GENETIC TESTING.

SADS Foundation's total resources for Schools. Includes screening, care plans for students with SADS Conditions and emergency care.

Blue Cross Blue Shield TEC Advisory letter; regional BCBS to

Genetic Testing for LQTS SADS Foundation Resources:

- **Blue Cross Blue Shield TEC Advisory letter; regional BCBS to**

Key Elements of a comprehensive SCDP Program

- **Hospitals; Resuscitation Services:**
  - Therapeutic Hypothermia for Out of Hospital Cardiac Arrest with ROSC

Athletic Pre-Participation Screening

As per:

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)

A Scientific Statement From the American Heart Association and the American College of Cardiology

Endorsed by the Pediatric and Congenital Electrophysiology Society and American College of Sports Medicine

2016 Evidence Based

Athletic Pre-Participation Screening

Prepared by: Wayne W Ruppert, CVT, CCCC, NREMT-P

1. **History and Physical Evaluation:**

   Use AHA/ACC approved format:

   - 14 Element AHA Prescreening Tool
   - AAP Pre-participation Physical Evaluation Monograph

Bayfront Health
Dade City

The 14 Element AHA Cardiovascular Screening Checklist for Congenital and Genetic Heart Disease (Recommended for Pre-Participation Screening of Competitive Athletes)

Personal History

Yes	No	
☐	☐	1. Chest pain/discomfort/tightness/pressure related to exertion
☐	☐	2. Unexplained syncope/near-syncope*
☐	☐	3. Excessive exertional and unexplained dyspnea/fatigue or palpitations, associated with exercise
☐	☐	4. Prior recognition of a heart murmur
☐	☐	5. Elevated systemic blood pressure
☐	☐	6. Prior restriction from participation in sports
☐	☐	7. Prior testing for the heart, ordered by a physician

* When determined to be not of neurcardiogenic (vasovagal) in origin. Of particular concern is syncope post-strenous activity.

Family History

Yes	No	
☐	☐	8. Premature death (sudden and unexpected, or otherwise) before age 50 attributable to heart disease in ≥1 relative
☐	☐	9. Disability from heart disease in close relative <50 y of age
☐	☐	10. Hypertrophic or dilated cardiomyopathy, long-QT syndrome, or other ion channelopathies, Marfan syndrome, or clinically significant arrhythmias; specific knowledge of certain cardiac conditions in family members

Physical Examination

Yes	No	
☐	☐	11. Heart murmur**
☐	☐	12. Femoral pulses to exclude aortic coarctation
☐	☐	13. Physical stigmata of Marfan syndrome
☐	☐	14. Brachial artery blood pressure (sitting position)***

** Auscultation should be performed in both sitting and standing positions (or with Valsalva maneuver). Objective is to identify murmurs of dynamic LV outflow tract obstruction.

*** Should be taken in both arms.

Pre-participation Screening Forms

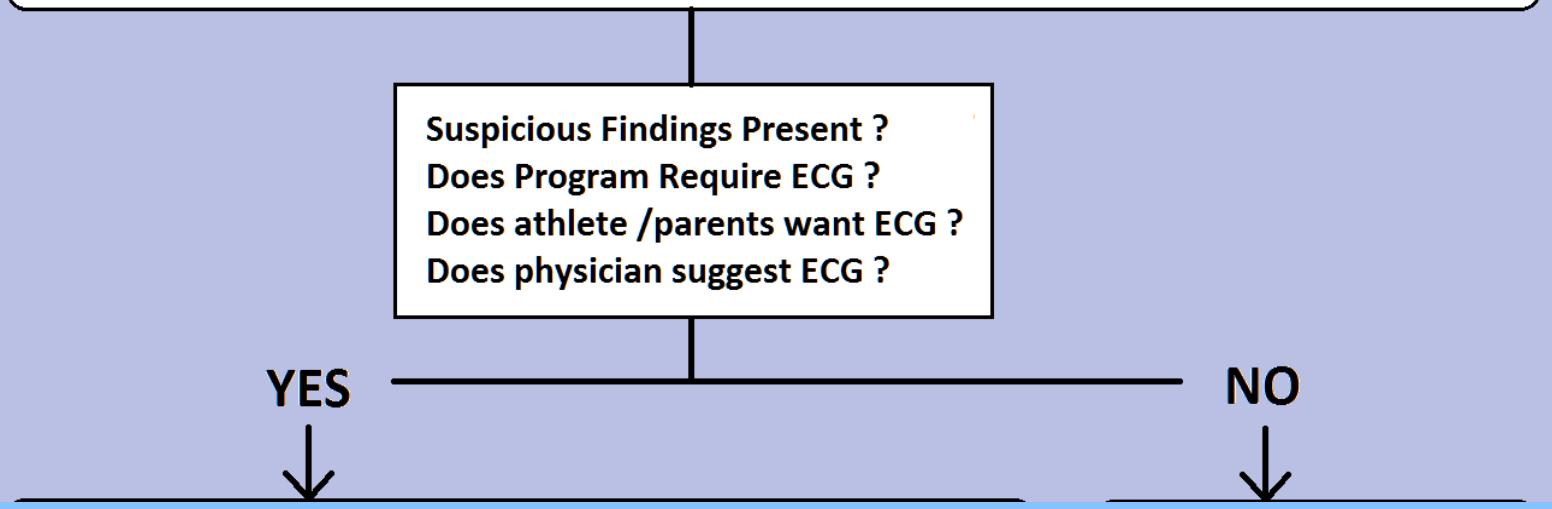
CLICK HERE to Download 14 Element AHA Pre-Participation Cardiovascular Screening of Competitive Athletes Form

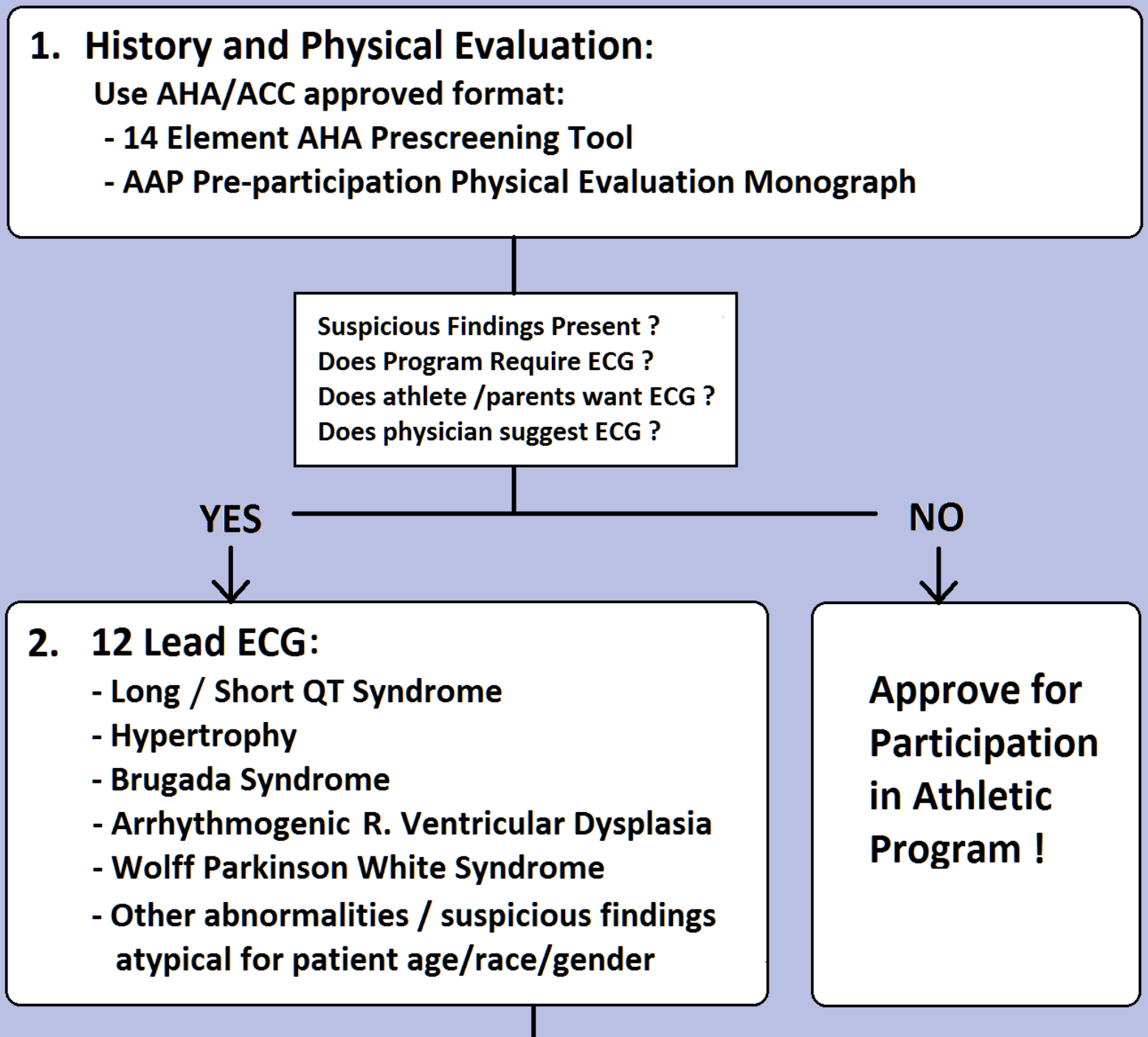
Click here for the American Academy of Pediatrics Athletics Pre-Participation Screening Form.

1. **History and Physical Evaluation:**

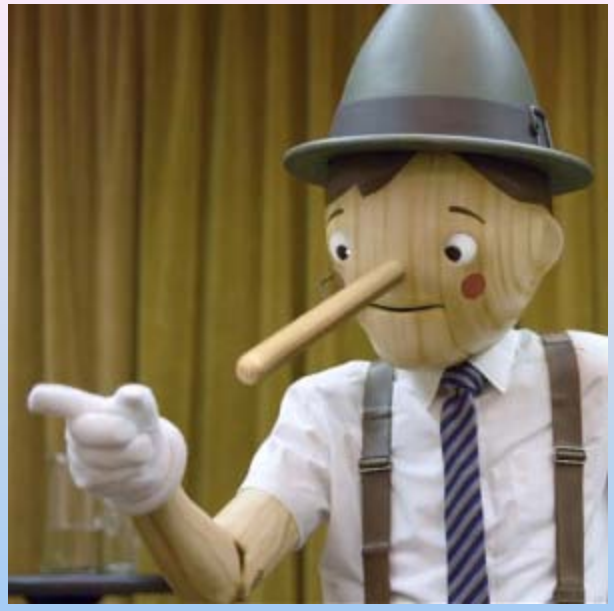
   Use AHA/ACC approved format:

   - **14 Element AHA Prescreening Tool**
   - **AAP Pre-participation Physical Evaluation Monograph**




1. History and Physical Evaluation:
Use AHA/ACC approved format:
- 14 Element AHA Prescreening Tool
- AAP Pre-participation Physical Evaluation Monograph
Suspicious Findings Present ?
Does Program Require ECG ?
Does athlete /parents want ECG ?
Does physician suggest ECG ?
YES
NO
2. 12 Lead ECG:
- Long / Short QT Syndrome
- Hypertrophy
- Brugada Syndrome
- Arrhythmogenic R. Ventricular Dysplasia
- Wolff Parkinson White Syndrome
- Other abnormalities / suspicious findings atypical for patient age/race/gender
Approve for Participation in Athletic Program !

EKGs DON'T ALWAYS TELL THE TRUTH. . .



"As is true when evaluating the ECG for Acute Coronary Syndrome, there is always an undesirable degree of LACK OF SENSITIVITY ("false negatives") and LACK OF SPECIFICITY ("false positives").

ECG Evaluation – Considerations:

- **Sensitivity and Specificity issues with the ECG:**
  - **A "negative ECG" does not rule out SADS conditions**
  - **The following SADS conditions typically have "normal" ECG presentations:**
    - Catecholeminertic Polymorphic Ventricular Tachycardia
    - Concealed Brugada Syndrome
    - Concealed bypass tracts (e.g. retrograde conducting only)
  - **HCM and other cardiomyopathies do not always present with ECG abnormalities.**

ECG Evaluation – Considerations:

- **Sensitivity and Specificity issues with the ECG:**
  - **A "positive ECG" warrants additional investigation**
  - **A positive ECG may result from:**
    - Another (unrelated) condition
    - A "False Positive" – no abnormality is present
    - An ECG pattern associated with athletic training; It may resemble those seen in patients with incomplete or mild expressions of primary cardiomyopathies and ion channelopathies.

Leave the detailed ECG diagnosis to the cardiologist.

Leave the detailed ECG diagnosis to the cardiologist.

However every critical care nurse, paramedic or other professional who reads an ECG should be aware of some important clues . . .

In the next section we will review important ECG features associated with common SADS conditions . . .

For more SPECIFIC information about ECG indicators for the following conditions, click on the associated link:

- Long QT Syndrome
- Short QT Syndrome
- Hypertrophic Cardiomyopathy
- Brugada Syndrome
- Arrythmogenic Right Ventricular Dysplasia
- Wolff-Parkinson-White Syndrome

Long QT Syndrome

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

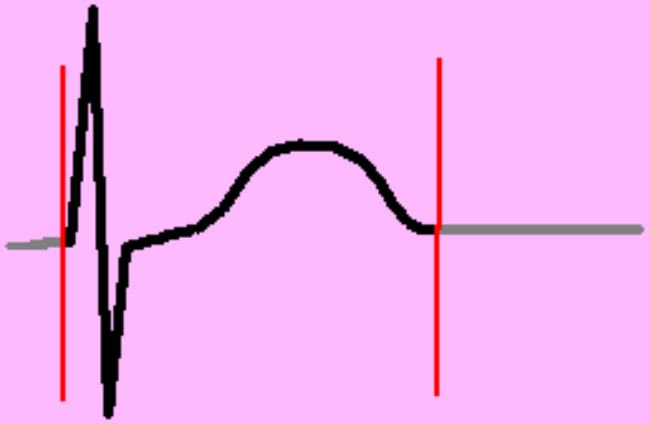
CNS & Autonomic nervous system disorders

Miscellaneous

Coronary Artery Disease

Mitral Valve Prolapse

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.

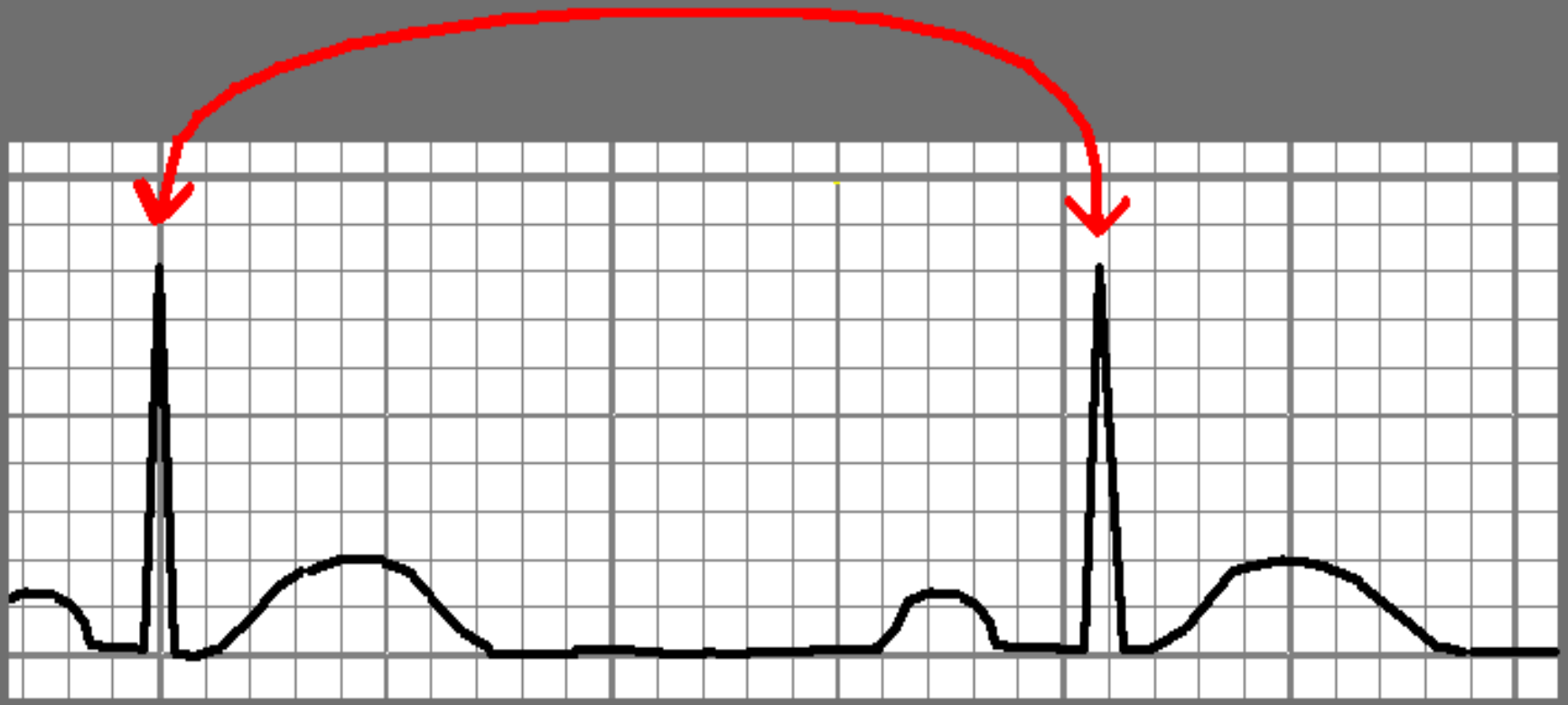
QT CORRECTION FORMULAS:

Bazett's	$QTc=QT/\sqrt{RR}$
Fredericia	$QTc=QT/(RR)1/3$
Framingham	$QTc=QT+0.154(1-RR)$
Rautaharju	$QTp=656/(1+HR/100)$

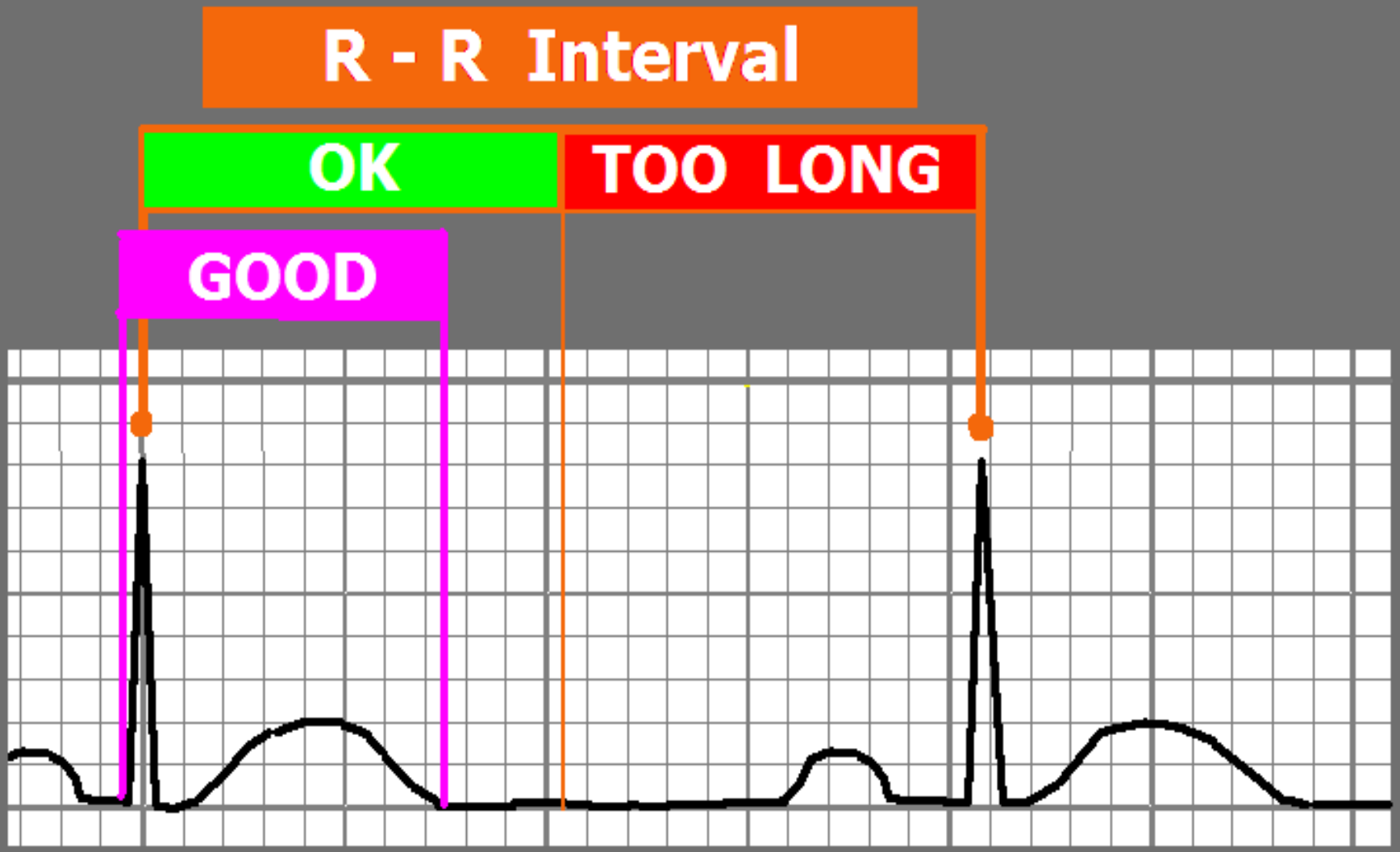
DETERMINING Q-T INTERVAL LIMITS

THE "QUICK PEEK" METHOD

(for Heart Rates 60 - 100)



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



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 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**

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

Suspected LQTS Considerations include:

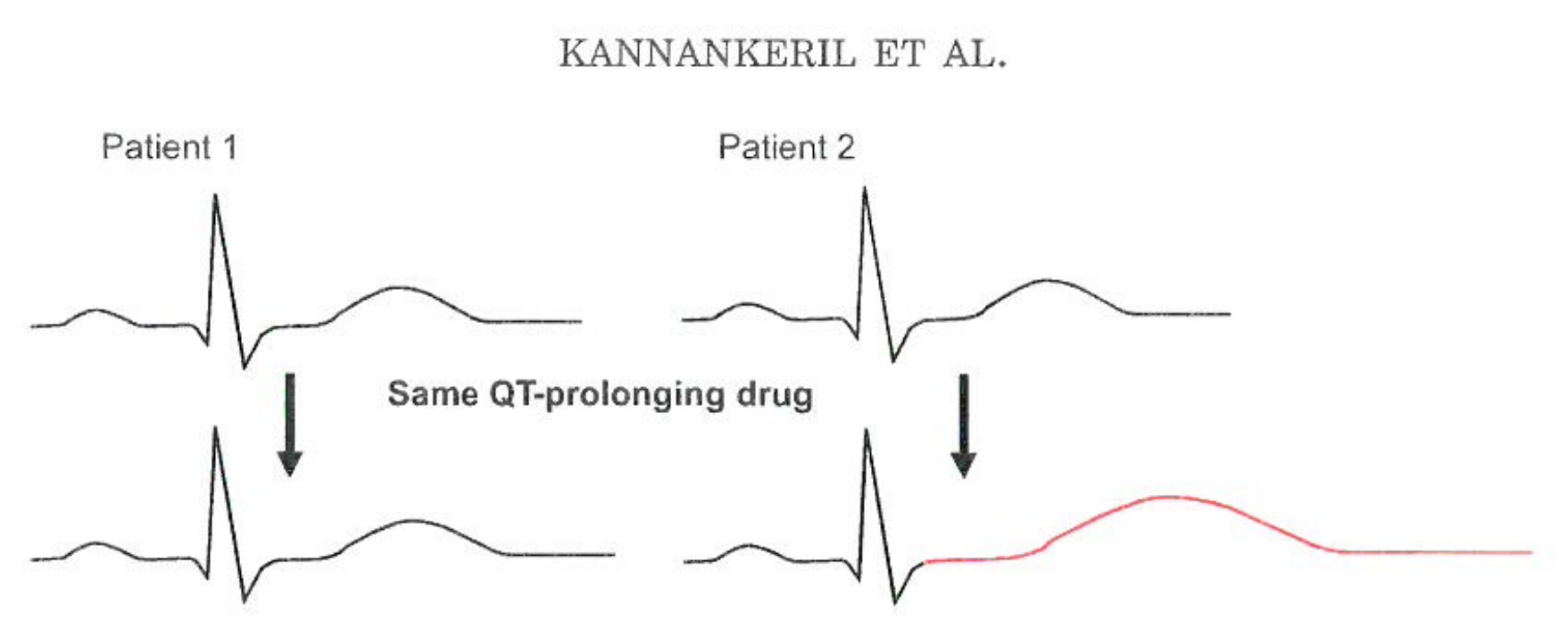
- ***Avoidance of Meds that are known to prolong the QT Interval. Click here for current list from CREDIBLEMEDS.ORG***

Commonly used QT prolonging meds include:

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

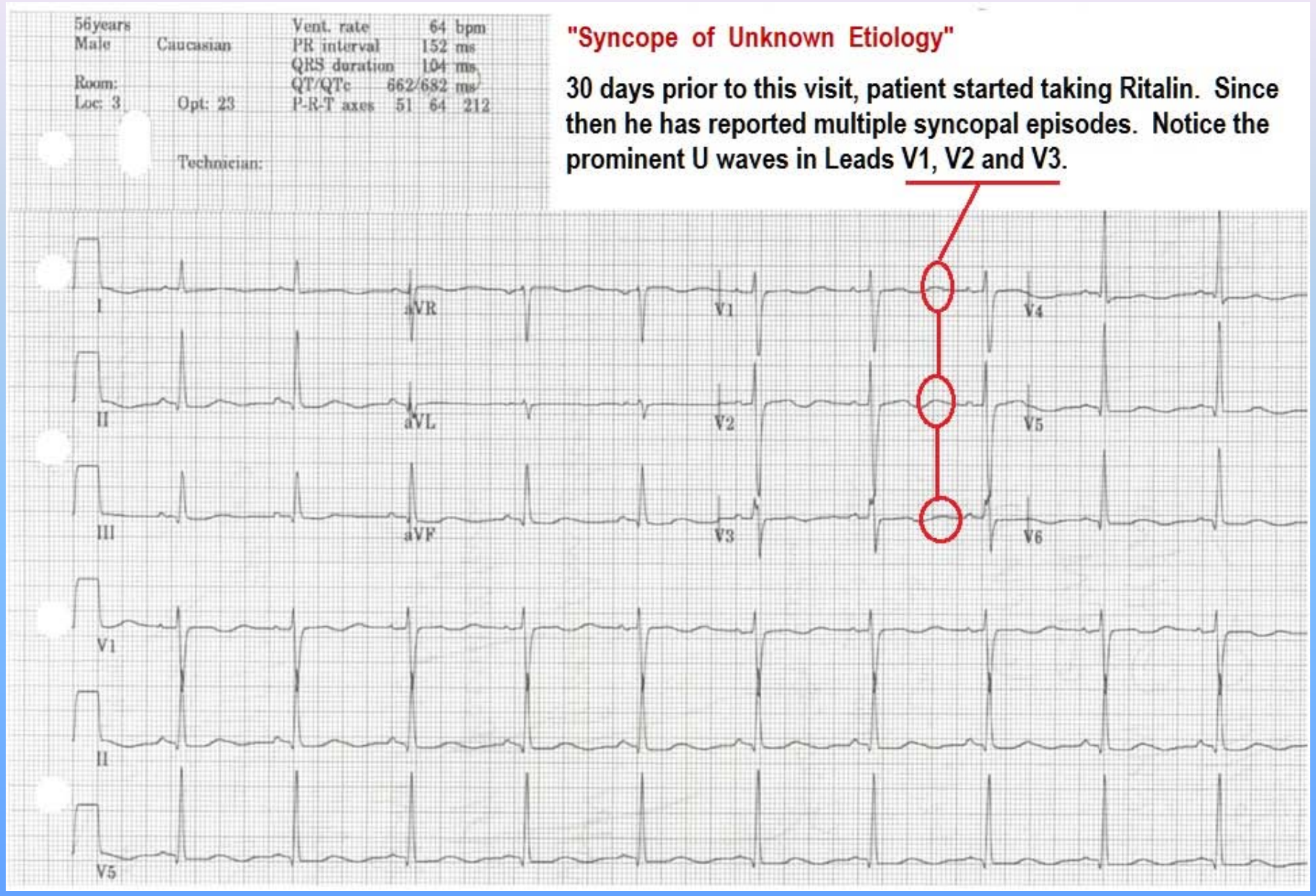
PATIENT 1: NORMAL

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



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

Medication induced LQTS Case Study: 56 year old male



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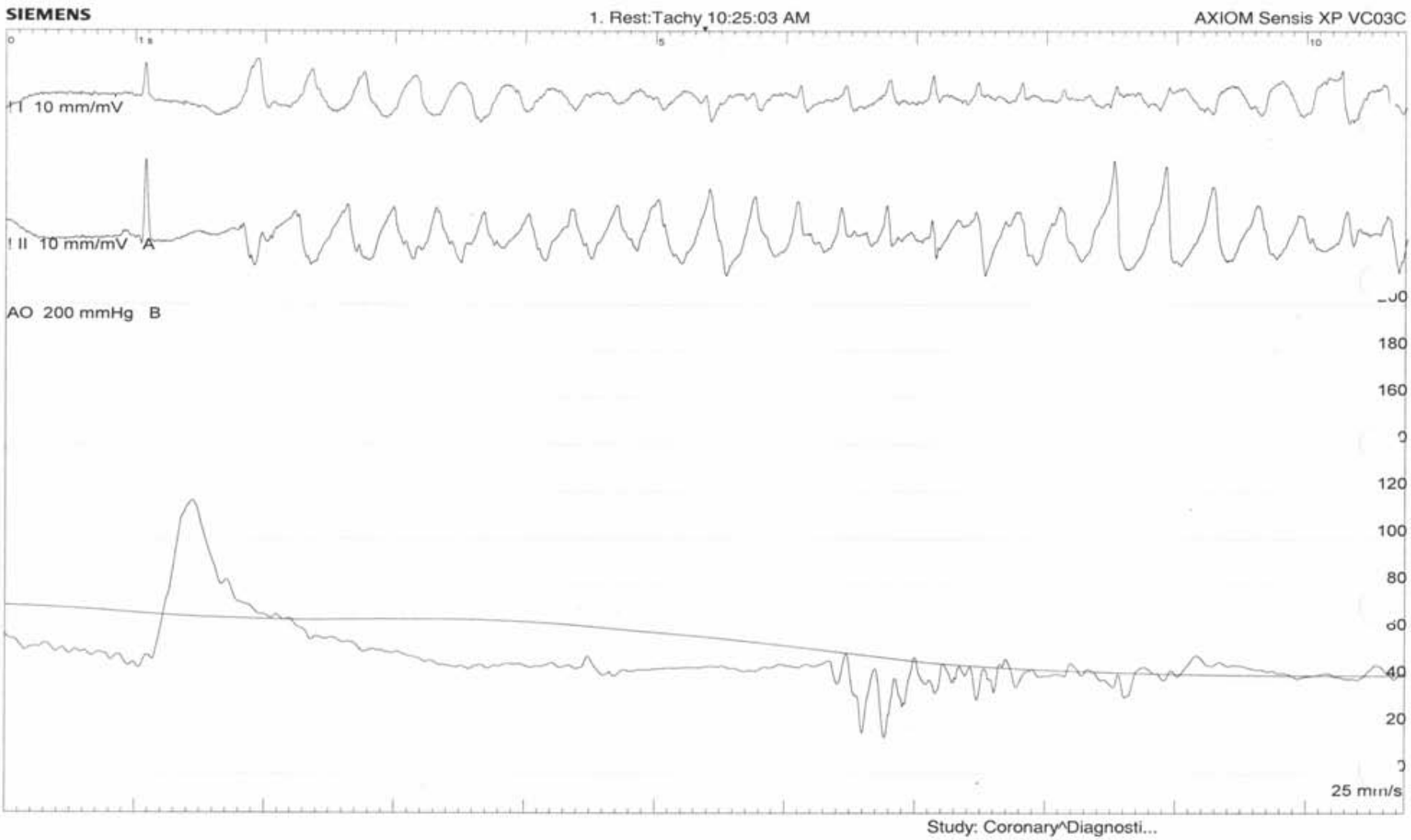
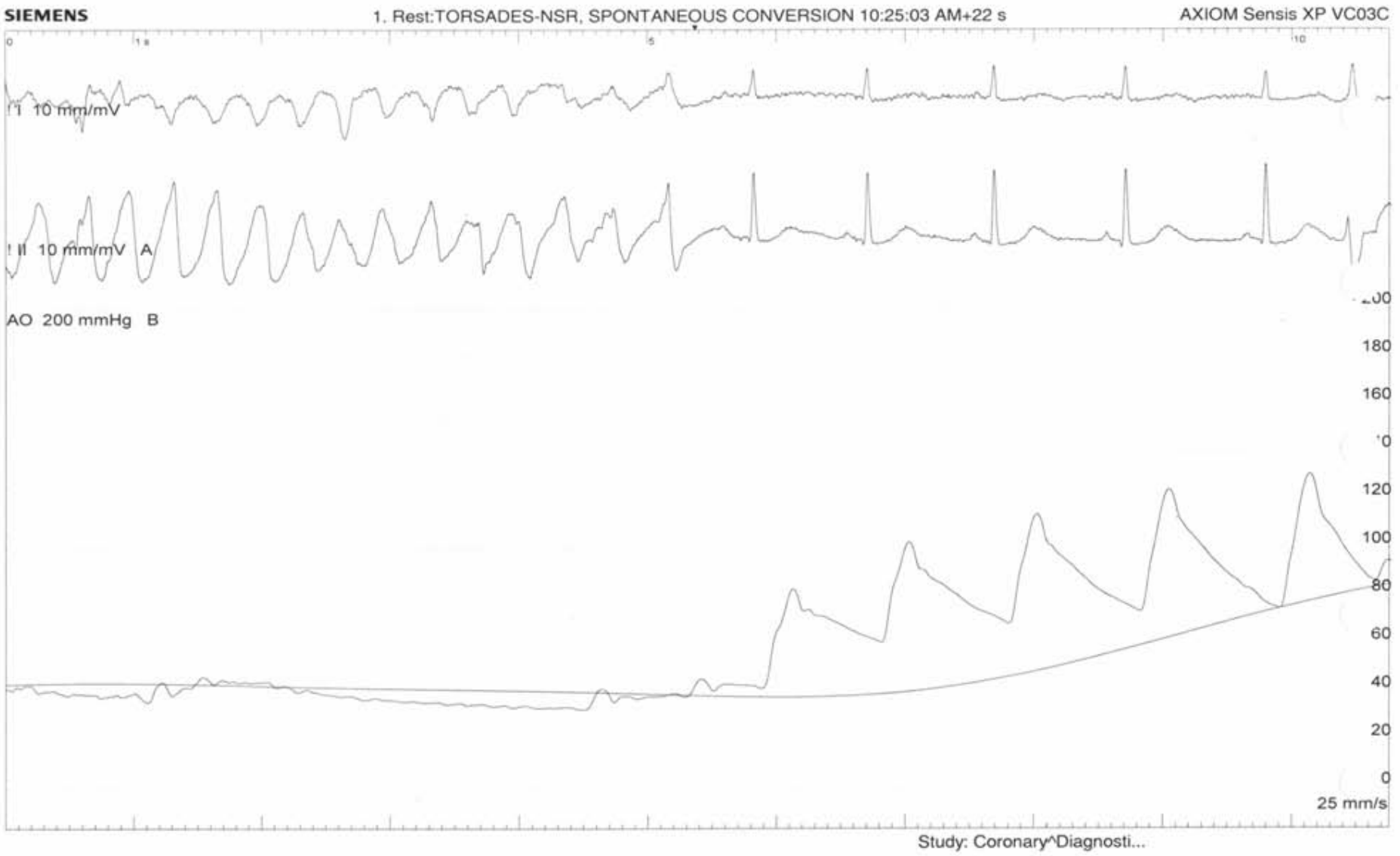
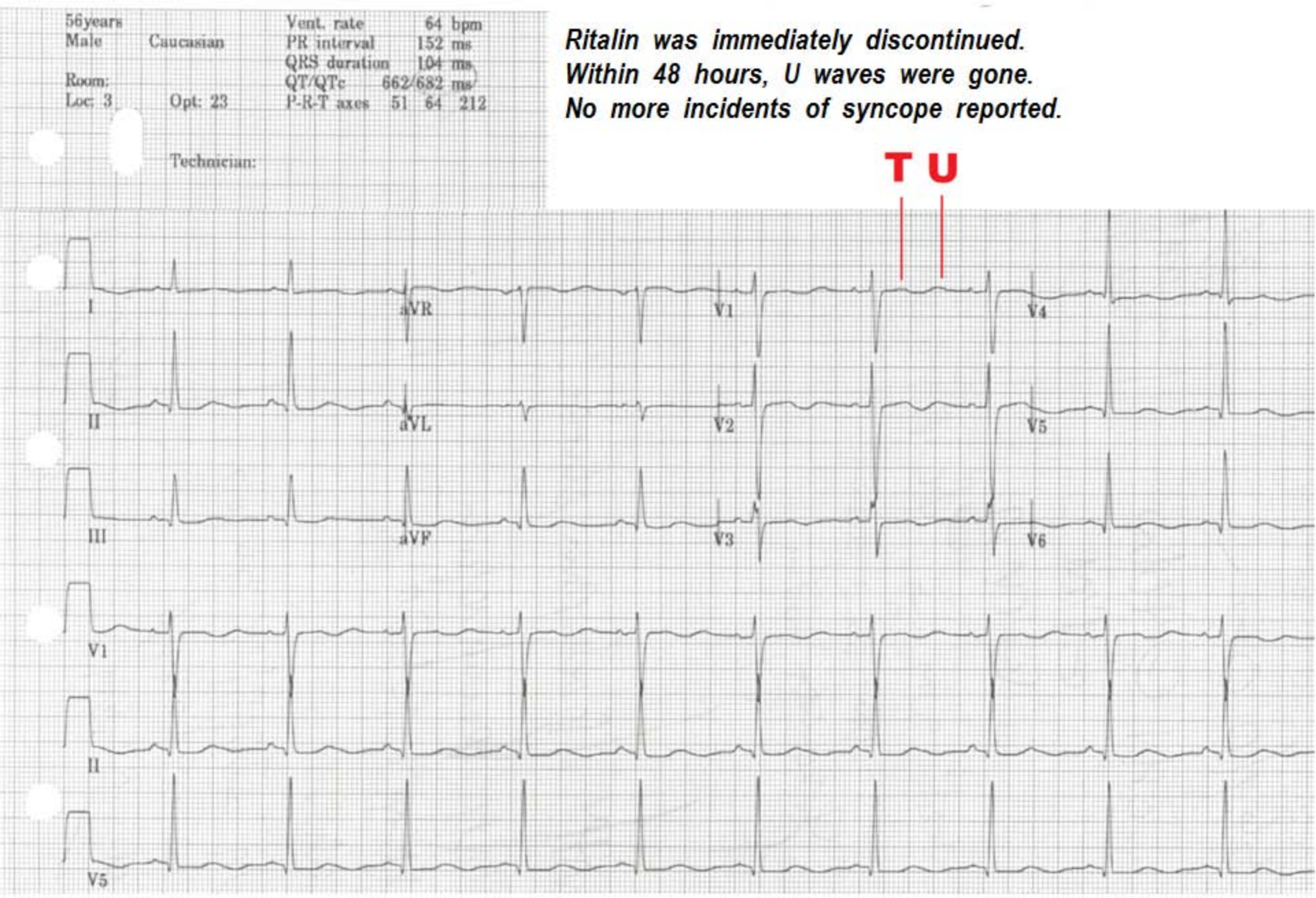


Run of Torsades de Pointes occurred during Cardiac Catheterization . . .

Medication induced LQTS Case Study: 56 year old male



Torsades de Pointes self-terminates just before aborted Defibrillation


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

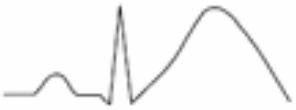
TREATMENT OF TORSADES de POINTES per AHA ACLS 2015:

-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.

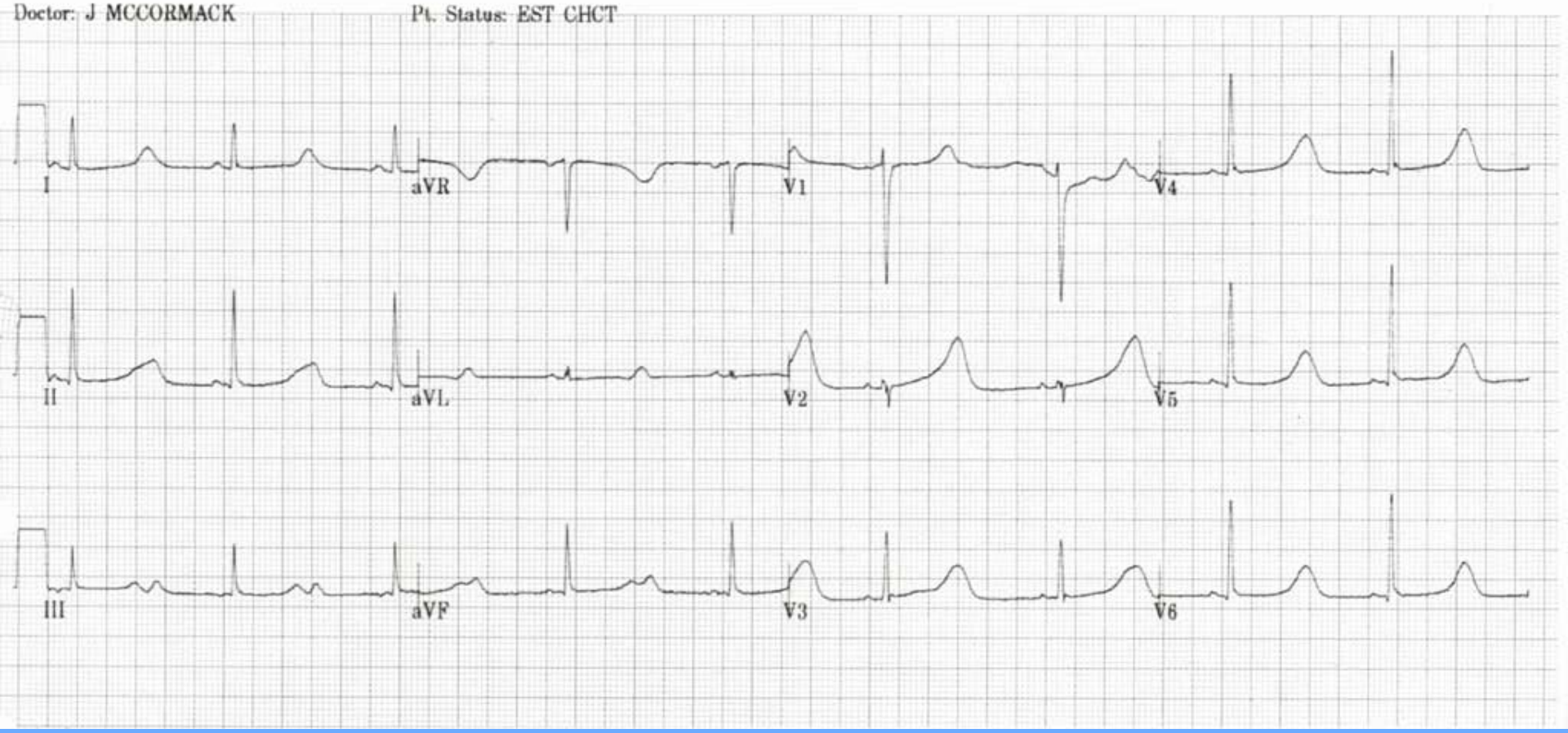
GENETICALLY ACQUIRED LONG QT SYNDROMES:
ECG PATTERNS of 3 MOST COMMON VARIATIONS:

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%

22 y/o FEMALE

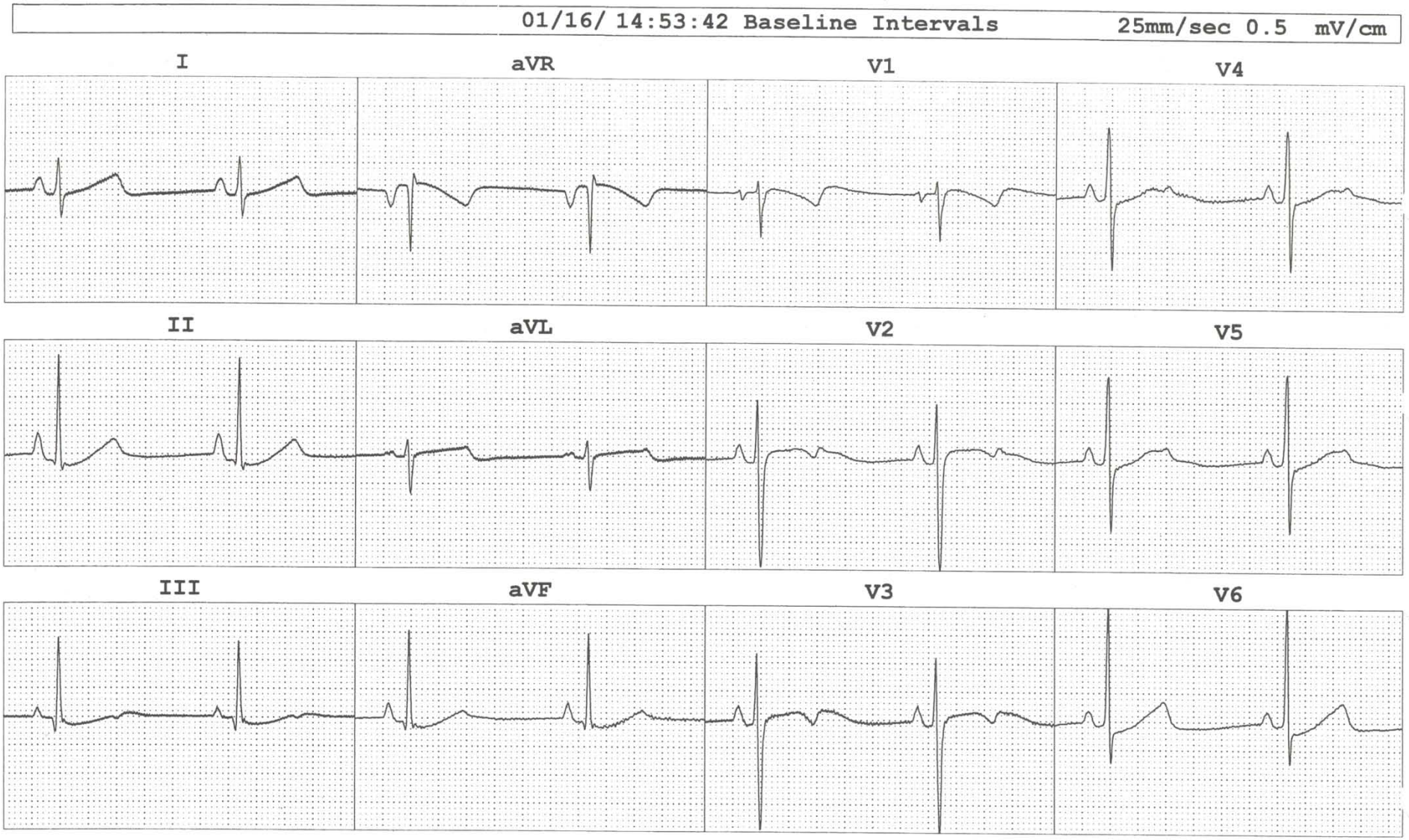
Vent. rate	53 bpm
PR interval	110 ms
QRS duration	84 ms
QT/QTc	678/636 ms
P-R-T axes	25 60 48

PEDIATRIC CARDIOLOGY ASSOCIATES



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

15 year old male with undiagnosed LQTS. He suffered out-of-hospital sudden cardiac arrest; it was the first indication of his condition. His ECG is shown below. T waves consistent with LQTS Type 2 can be seen in Leads V2 and V3:



The young man whose ECG is featured on the previous page had a complete recovery from his out-of-hospital cardiac arrest, thanks to immediate bystander CPR and use of an AED by mall security. I had the pleasure of meeting him when we implanted his ICD.

LQTS Risk Assessment Score

Developed by Peter Schwartz, MD

	Points
Electrocardiographic findings	
A QTc	
≥480 ms	3
460–479 ms	2
450–459 ms (in males)	1
B QTc 4th minute of recovery from exercise stress test ≥480 ms	1
C Torsade de pointes	2
D T wave alternans	1
E Notched T wave in 3 leads	1
F Low heart rate for age	0.5
Clinical history	
A Syncope	
With stress	2
Without stress	1
B Congenital deafness	0.5
Family history	
A Family members with definite LQTS	1
B Unexplained sudden cardiac death below age 30 among immediate family members	0.5

SCORE: ≤1 point: low probability of LQTS.

1.5 to 3 points: intermediate probability of LQTS.

≥3.5 points high probability.

Short QT Syndrome

- Normal QTc for males is 350-450ms
- Normal QTc for females is 360-460ms
- Therefore any QTc <350 in males and <360 in females is considered short.
- Typical QT intervals for patients with diagnosed SQTS is between 220-350ms.
- Many ECG feature little or no ST Segment; the T wave originates at the termination of the S wave.

Short QT Syndrome

- **Most Common Dysrythmias:**
  - **Atrial Fibrillation**
  - **Ventricular Tachycardia**
  - **Ventricular Fibrillation**

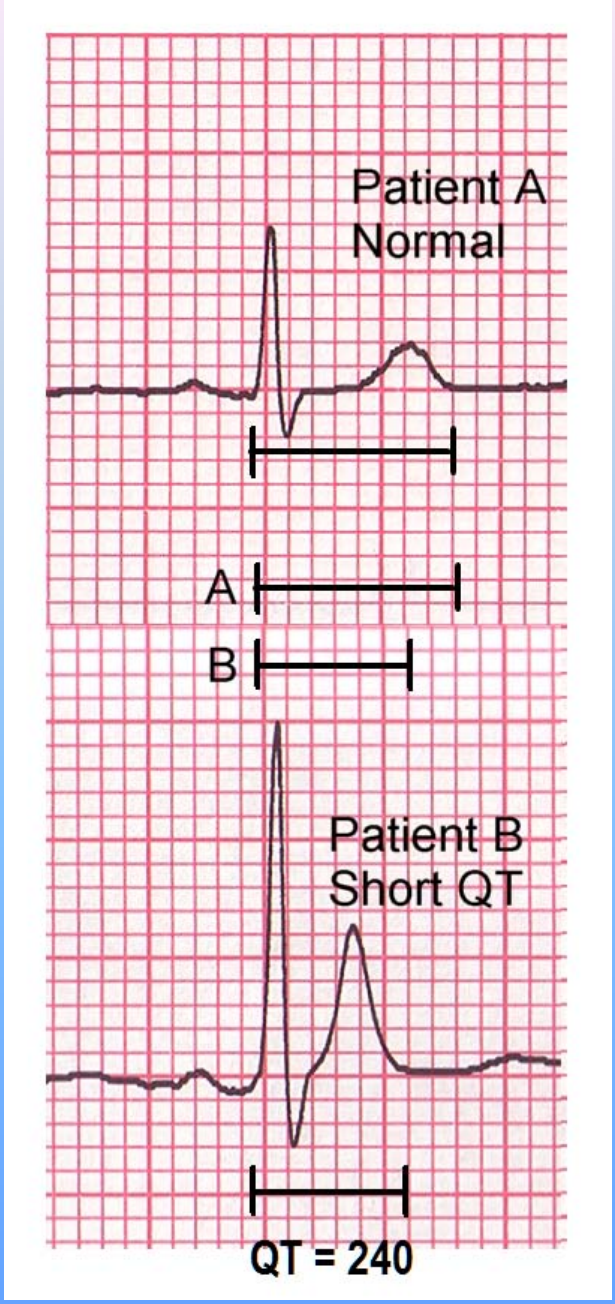
Short QT Syndrome

- When an abnormal (Short OR Long) QT interval is noted, rule out SECONDARY causes first:
  - hyperkalemia
  - Acidosis
  - Hypercalcemia
  - Hyperthermia
  - effects of drugs like digitalis
  - effect of acetylcholine or catecholamine

Short QT Syndrome

- The Rautaharju formula for calculating the QTp (predicted QT interval) is proclaimed as being a more accurate measurement for SQTS ECG evaluation.
- The Rautaharju QTp formula:

(QTp)=656/(1+heart rate/100)



SQTS

www.shortqtsyndrome.org

Short QT Syndrome

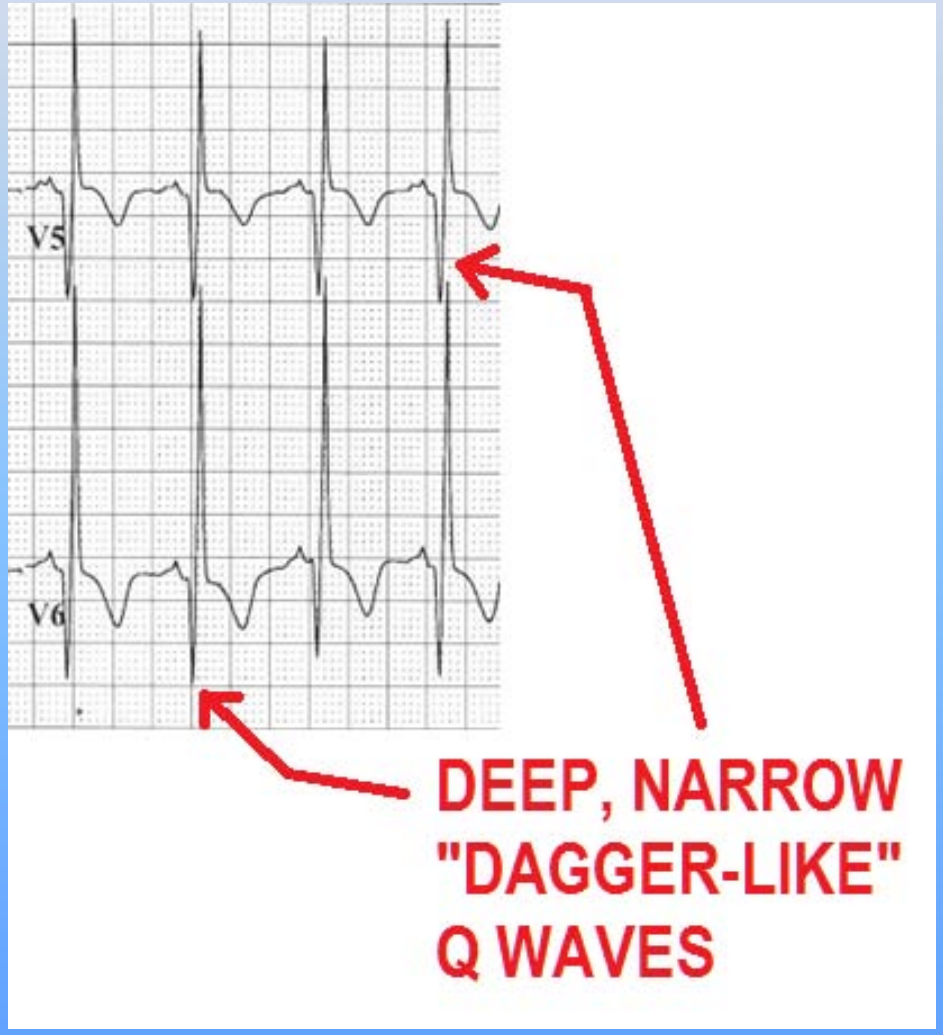
Management

- Medications: Quinidine
- ICD

ECG Indicators: Hypertrophic Cardiomyopathy

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

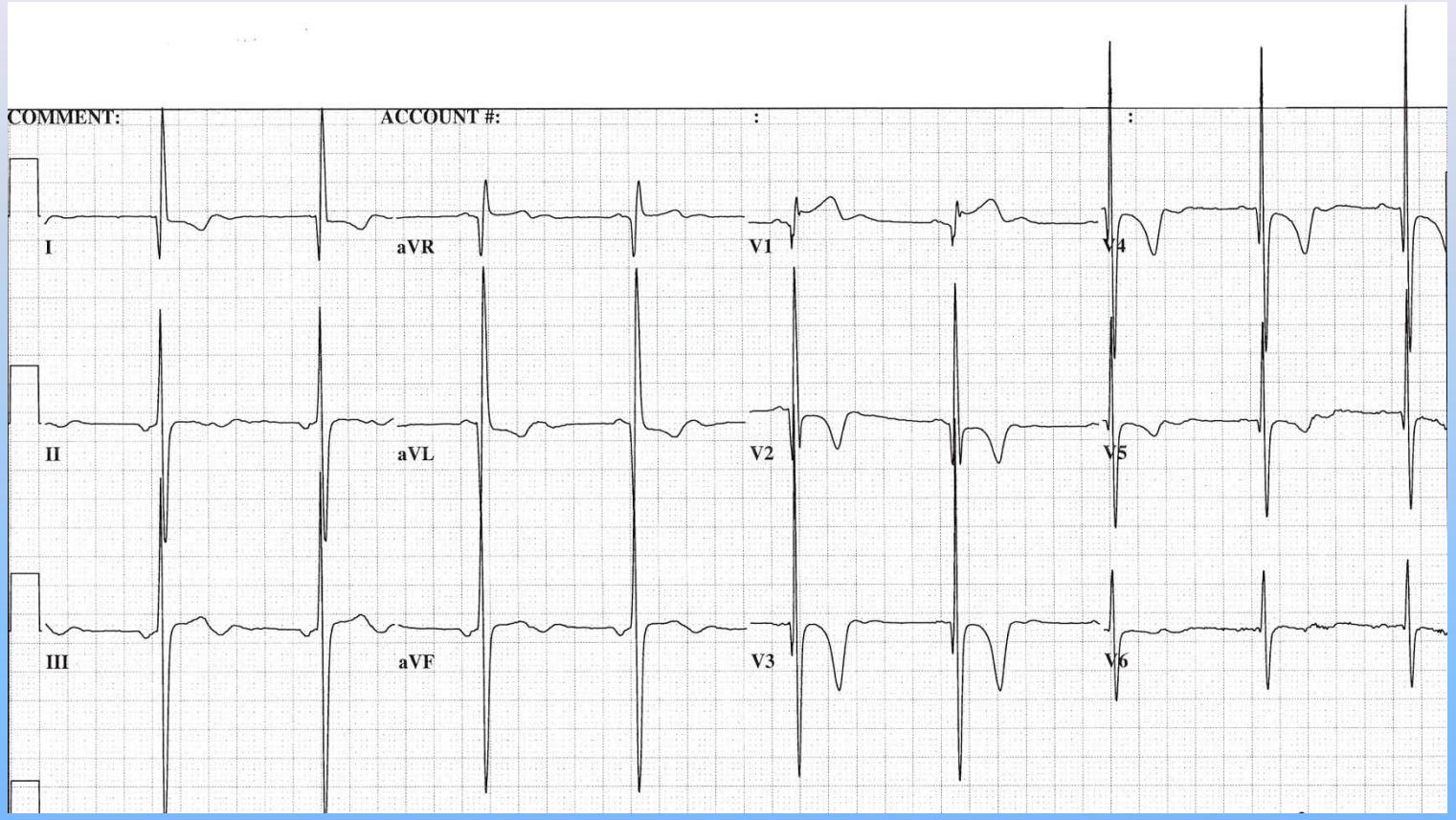
ECG Indicators:
Hypertrophic Cardiomyopathy



ECG Indicators: Hypertrophic Cardiomyopathy

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

Hypertrophic Cardiomyopathy (HCM)



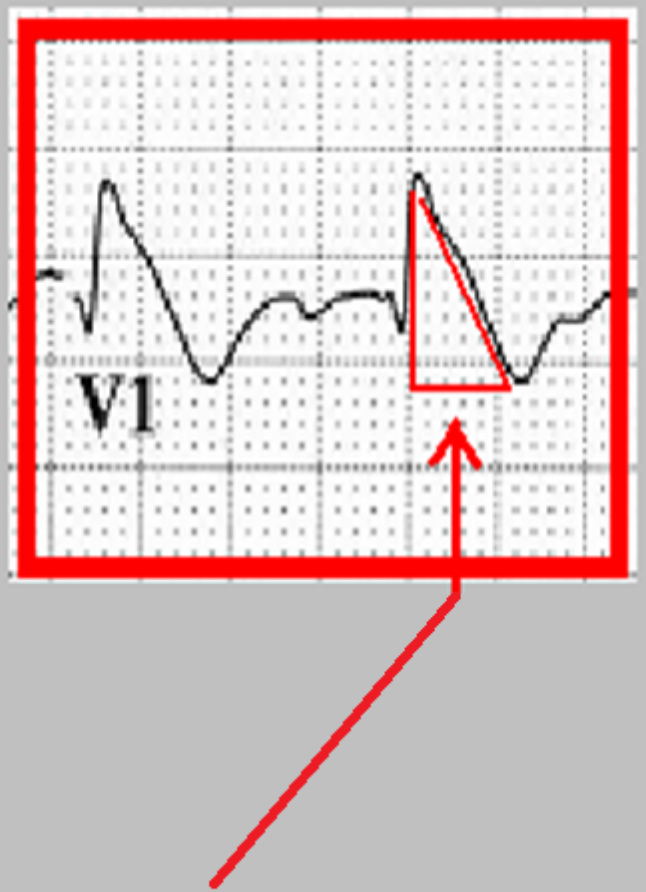
12 Lead ECG Traits:

- **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

BRUGADA SYNDROME

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



IS THERE ANYTHING ABNORMAL WITH THIS EKG ?

37 yr
Female Caucasian

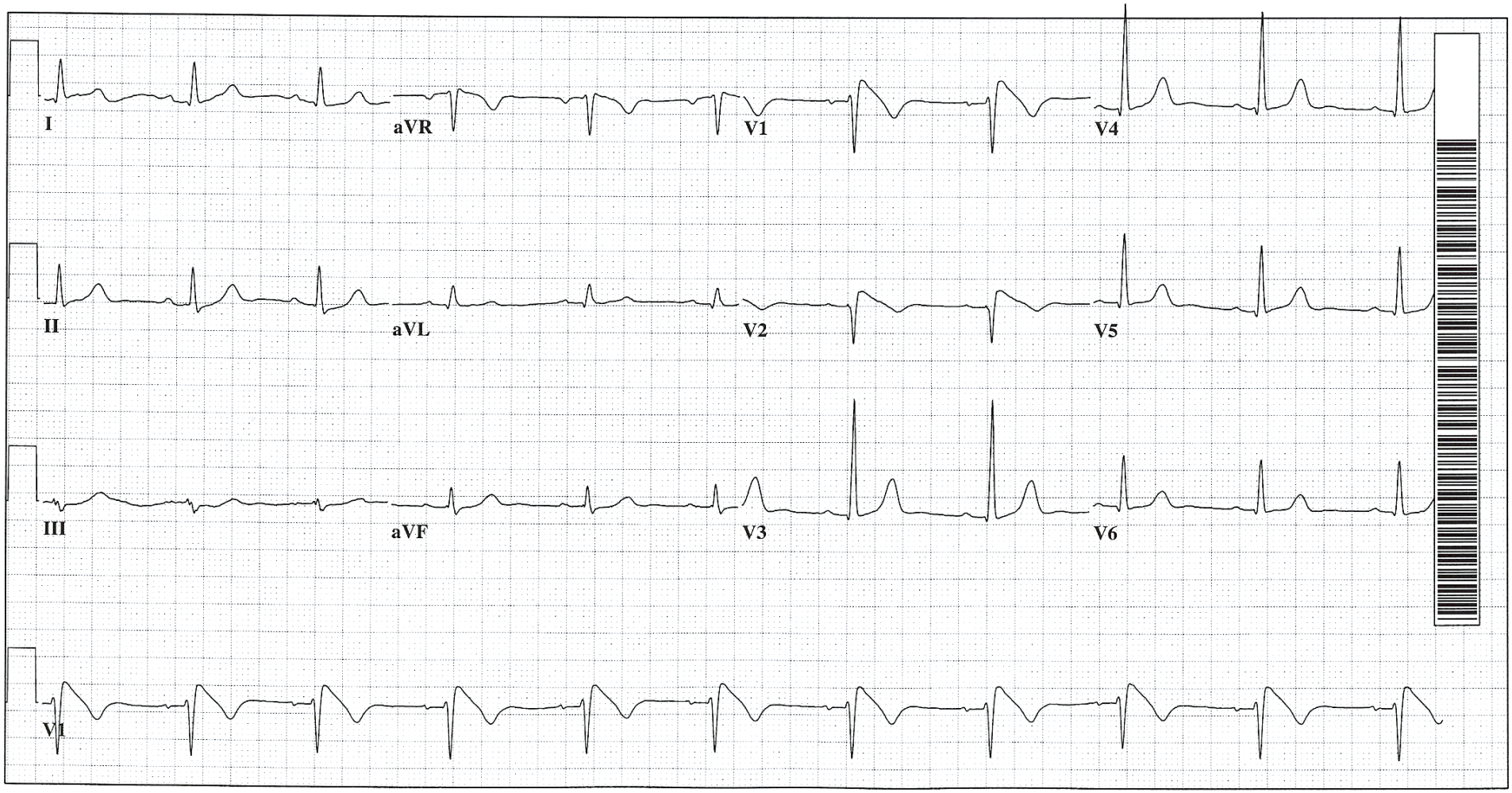
Room:C4A
Loc:3 Option:23

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:



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

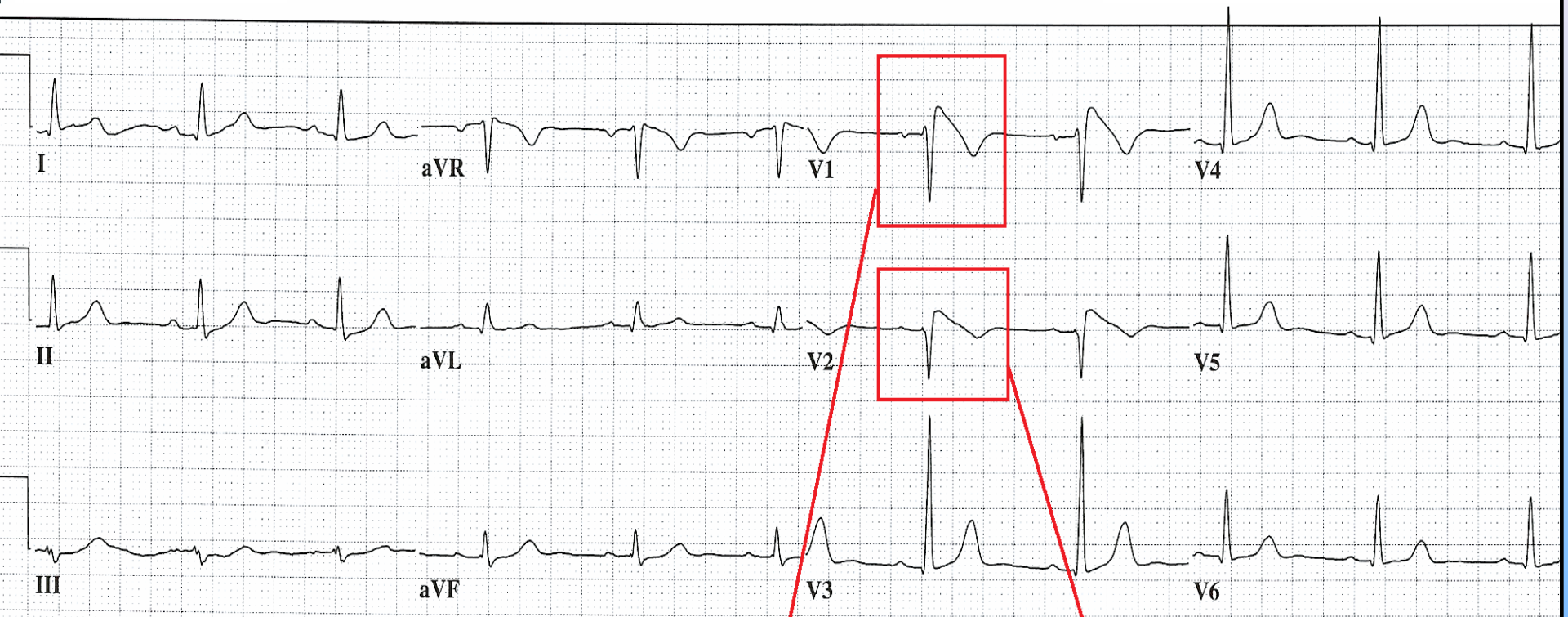
EID:46 EDT:

37 yr
Female Caucasian

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

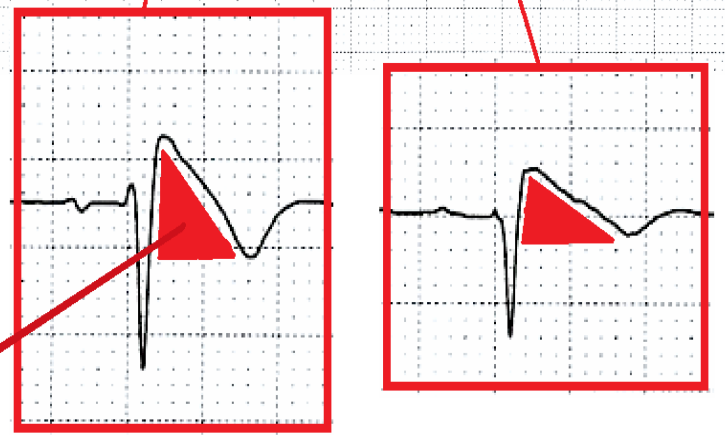
Normal sinus rhythm
Normal ECG
No previous ECGs available

NOTE COMPUTER INTERPRETATION !



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

NEVER FORGET THE "TRIANGULAR" SHAPE !



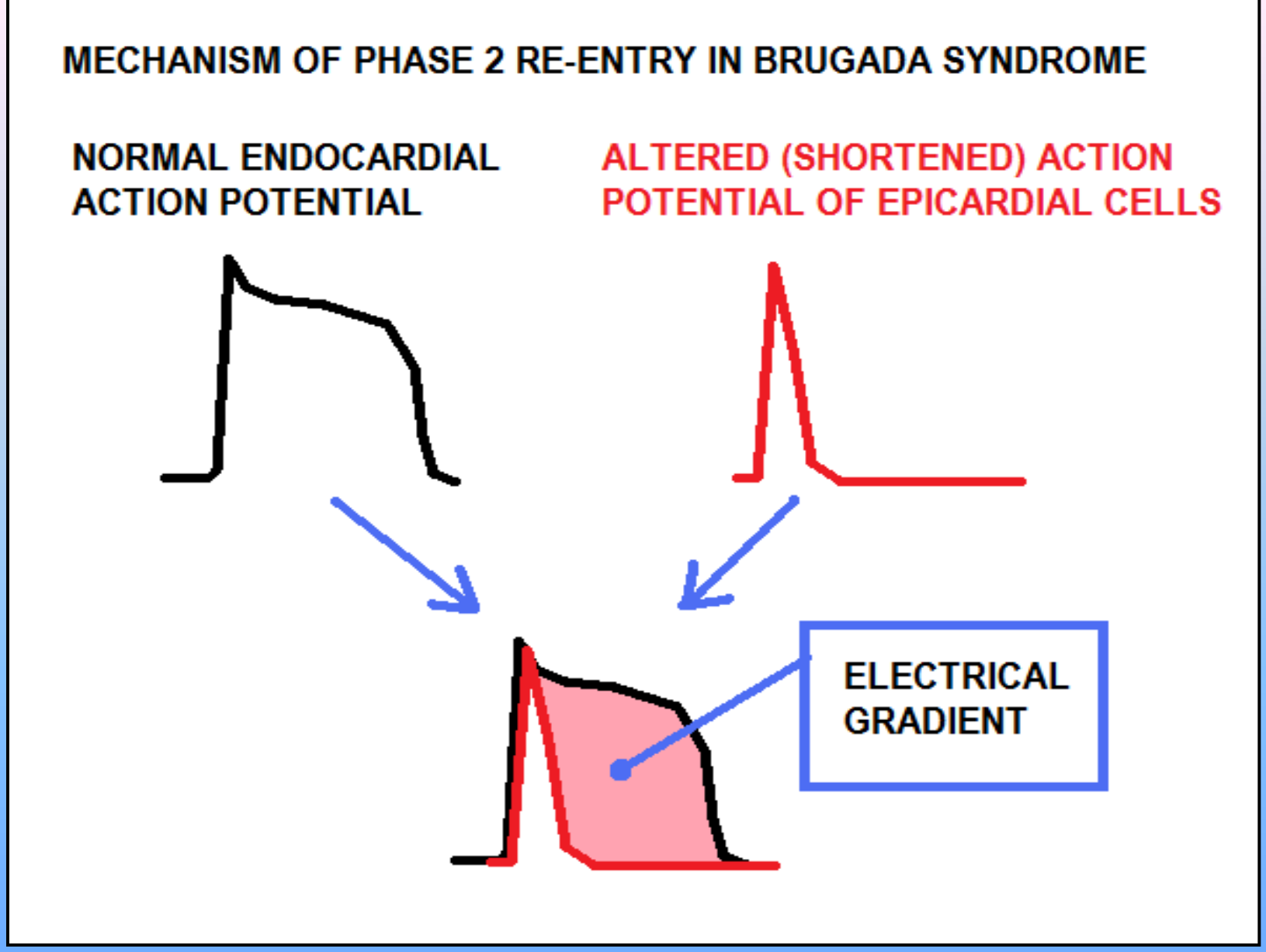
PATTERNS of S-T ELEVATION :

BEWARE of the

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

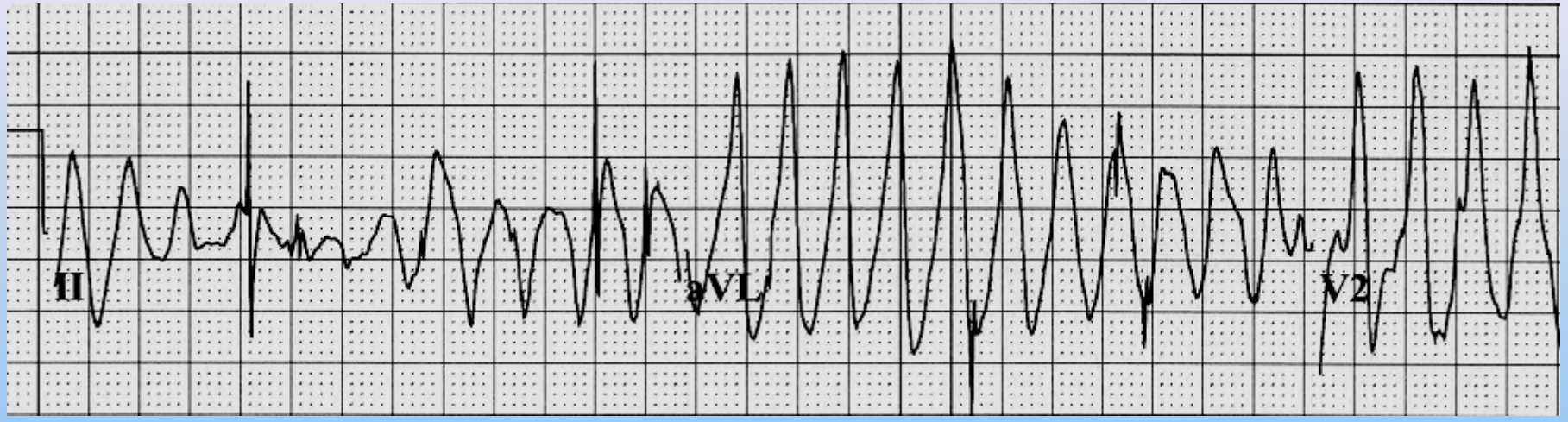
THINK - -

BRUGADA SYNDROME



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

Brugada / Long QT Syndromes cause:

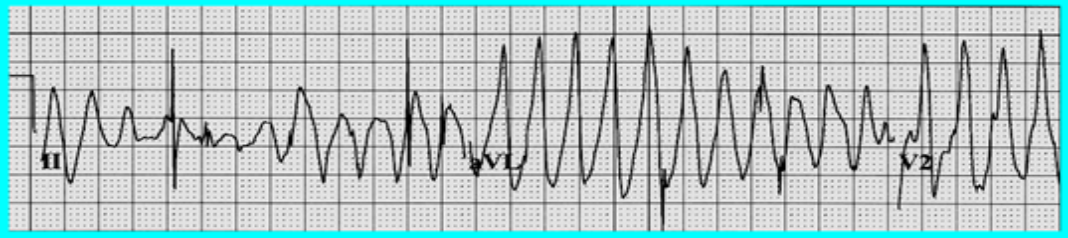


Torsades de Pointes:

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

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,

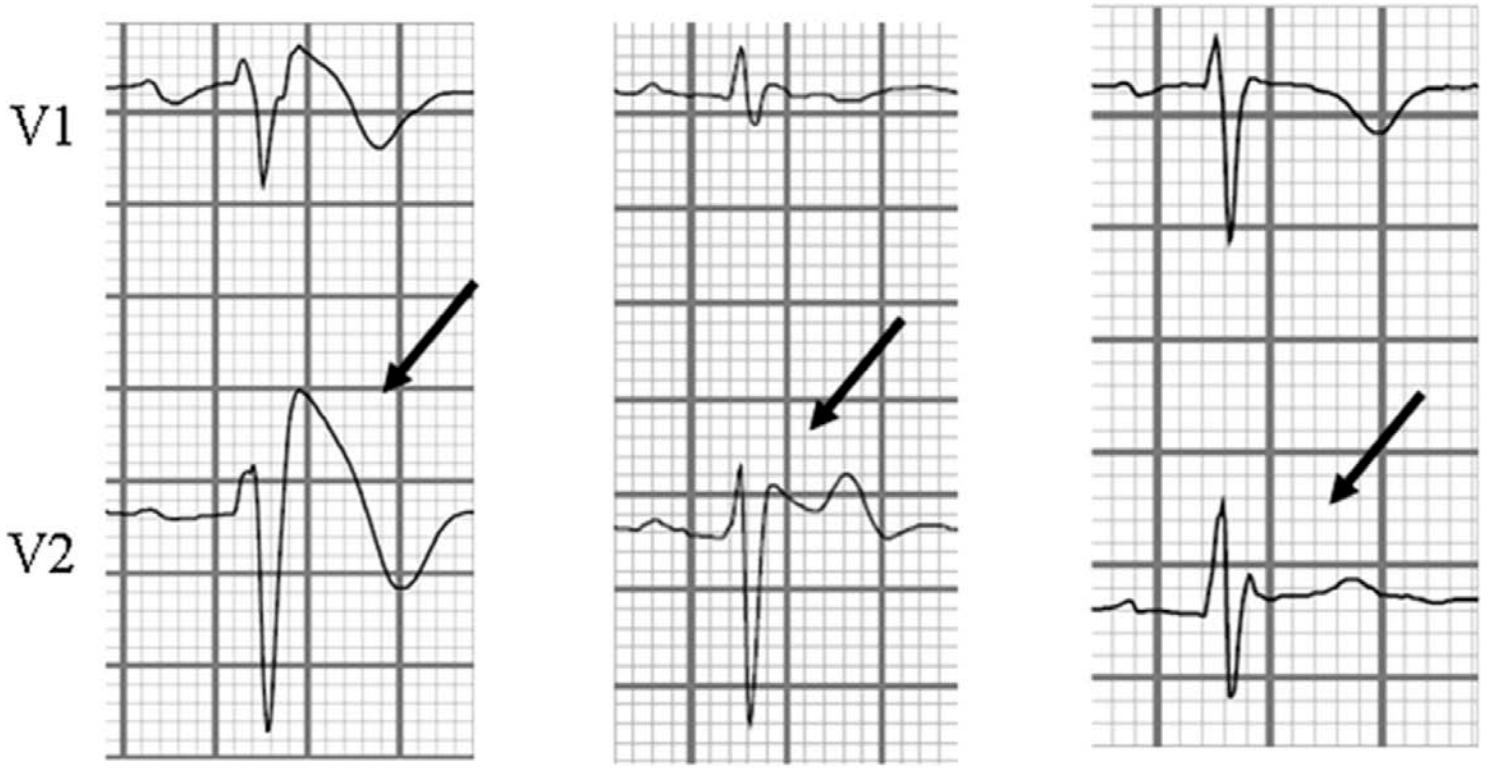
DO NOT give PROCAINAMIDE, AMIODARONE, or SOTALOL to patients with TORSADES or POLYMORPHIC VT !!!

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.

ECG abnormality diagnostic or suspected of Brugada syndrome.



Type 1:
Coved type
ST-segment elevation

Type 2:
saddle-back type
ST-segment elevation

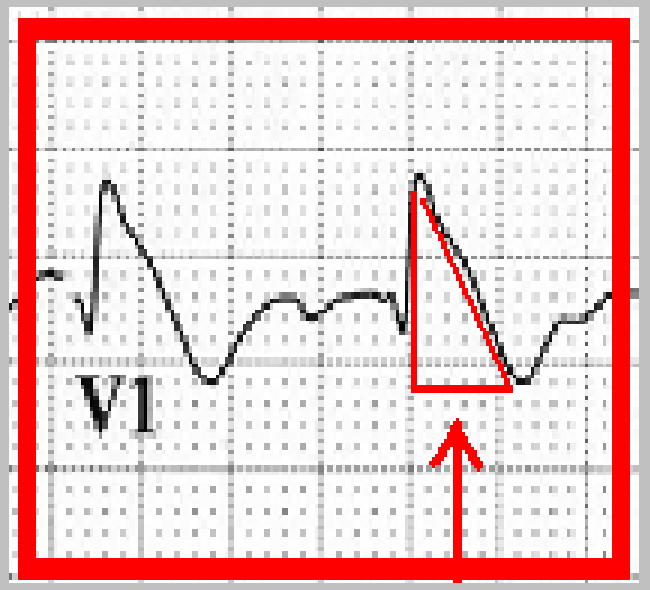
Type 3:
Saddle-back type
"ST-segment elevation"

Yuka Mizusawa, and Arthur A.M. Wilde Circ Arrhythm Electrophysiol. 2012;5:606-616

American Heart Association.

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

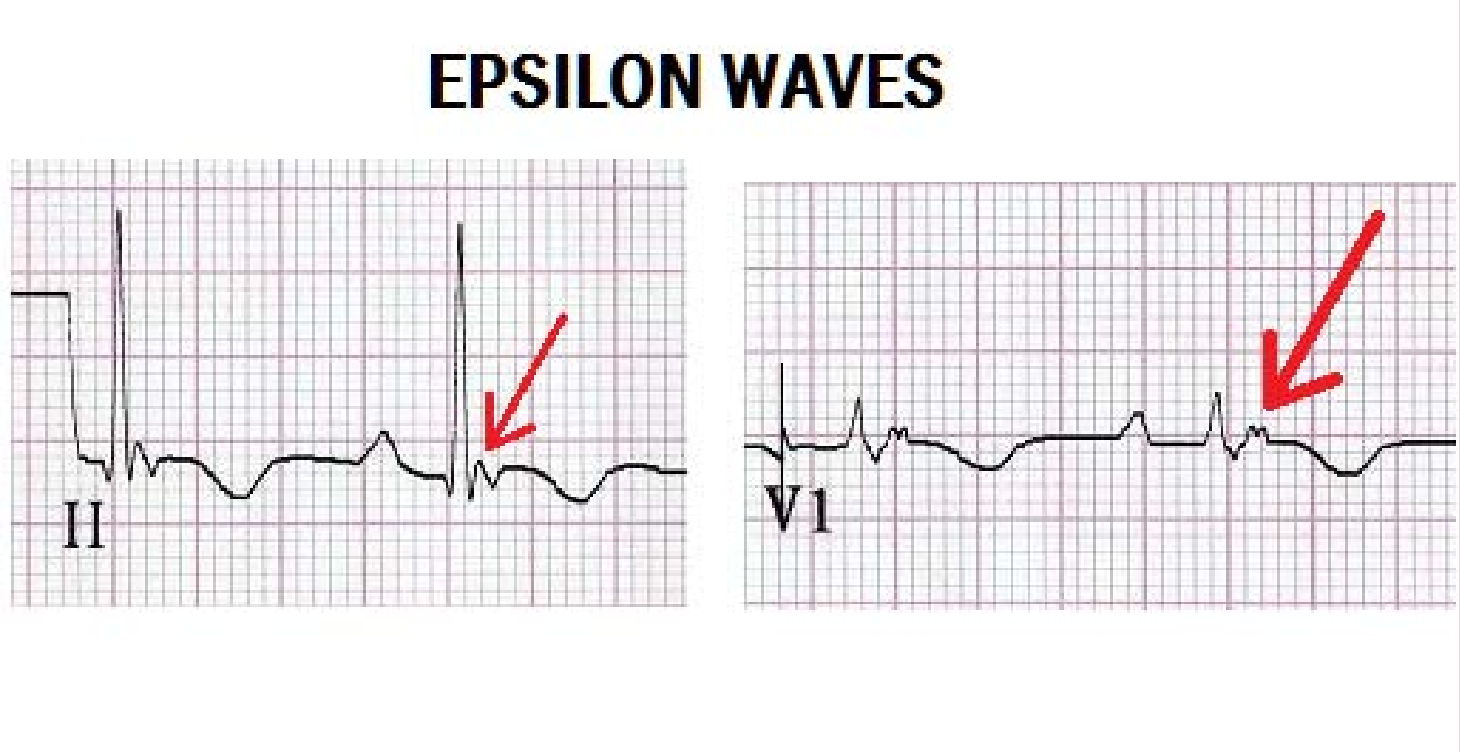
- **A genetically acquired myocardial disease associated with paroxysmal ventricular arrhythmias and sudden cardiac death.**
- **Characterized pathologically by fibro-fatty replacement of the right ventricular myocardium.**
- **The second most common cause of sudden cardiac death in young people (after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

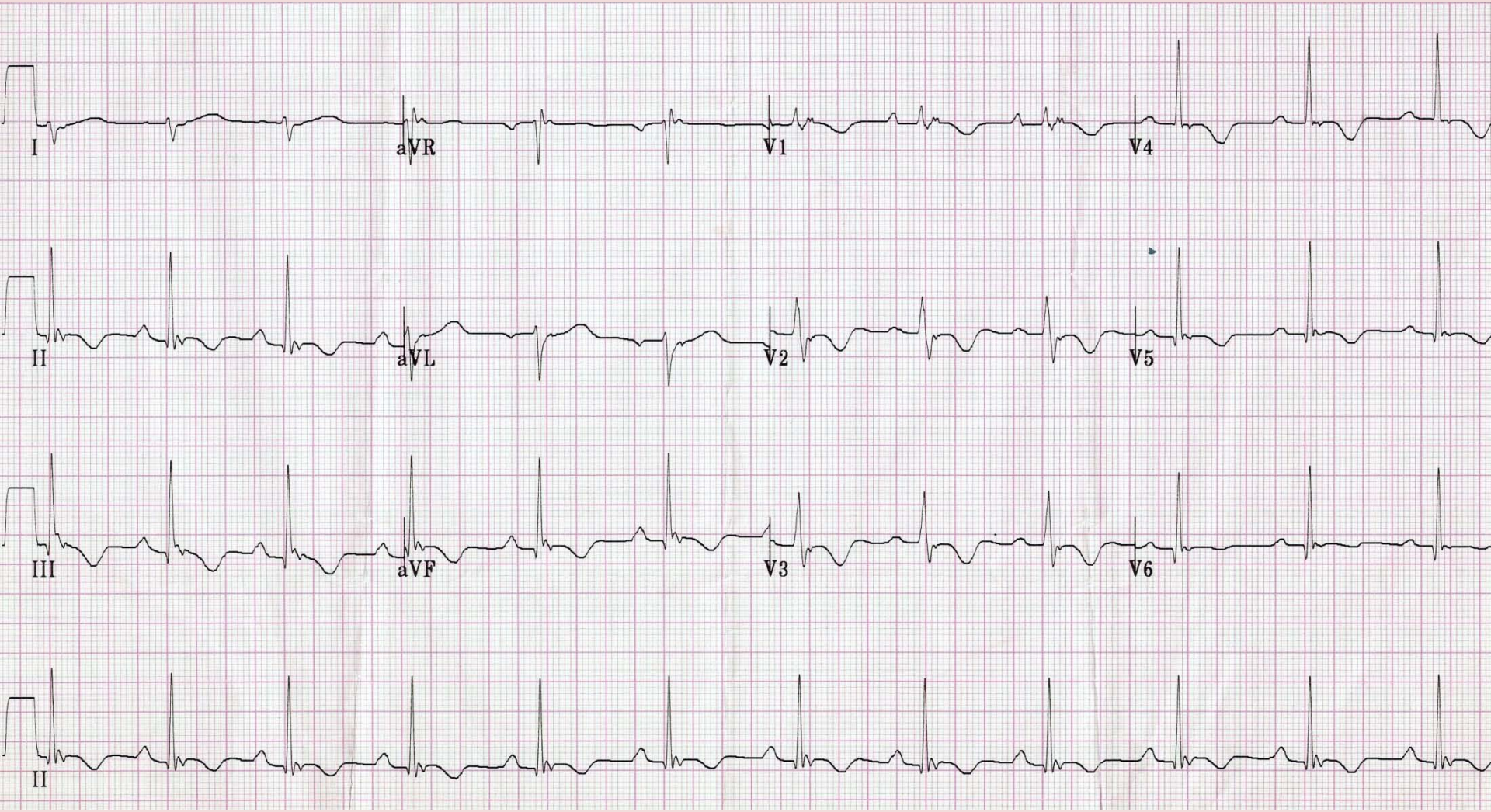
Arrhythmogenic Right Ventricular (RV) Cardiomyopathy and/or Dysplasia:

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

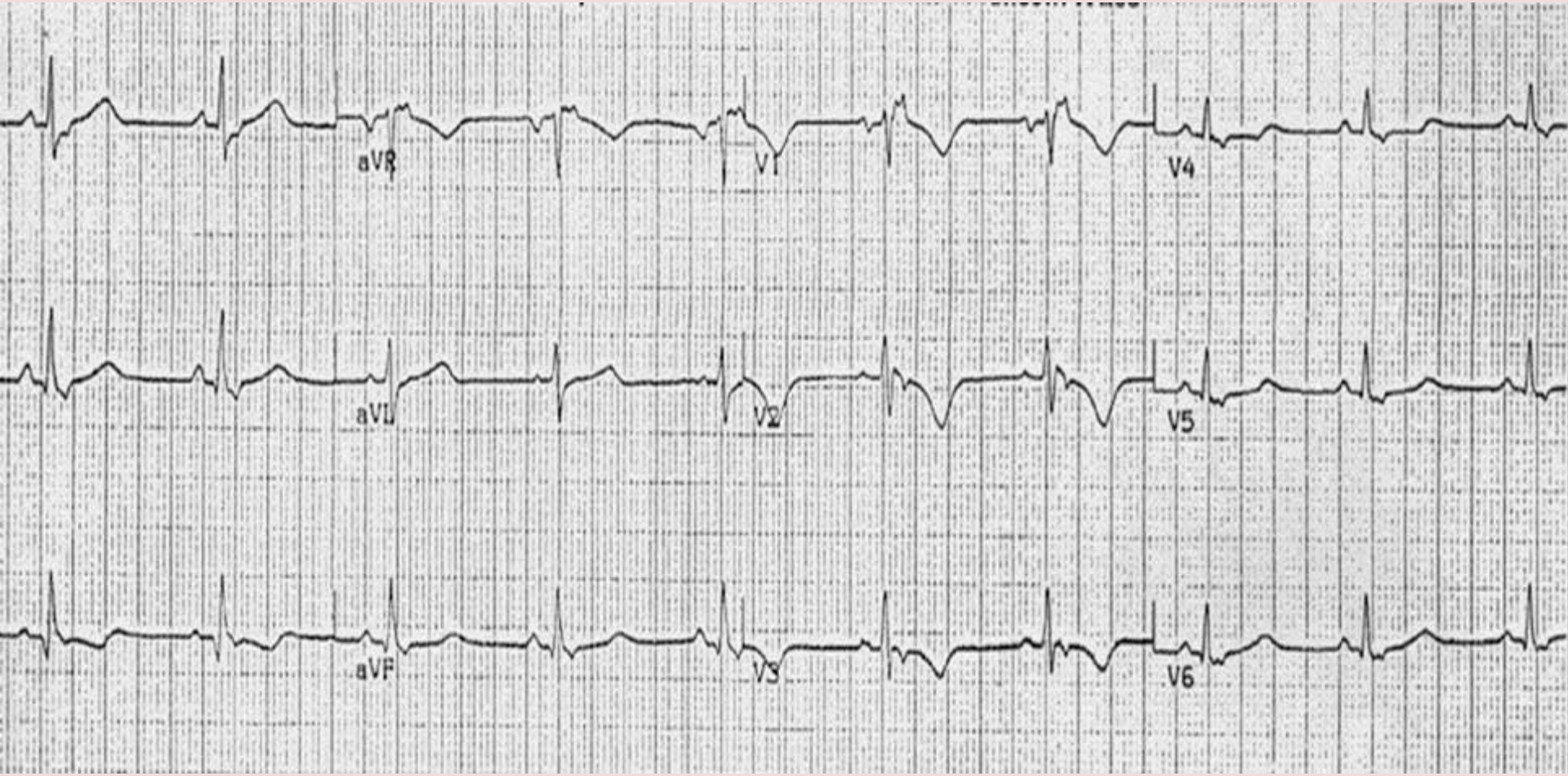


ARVD ECG 1

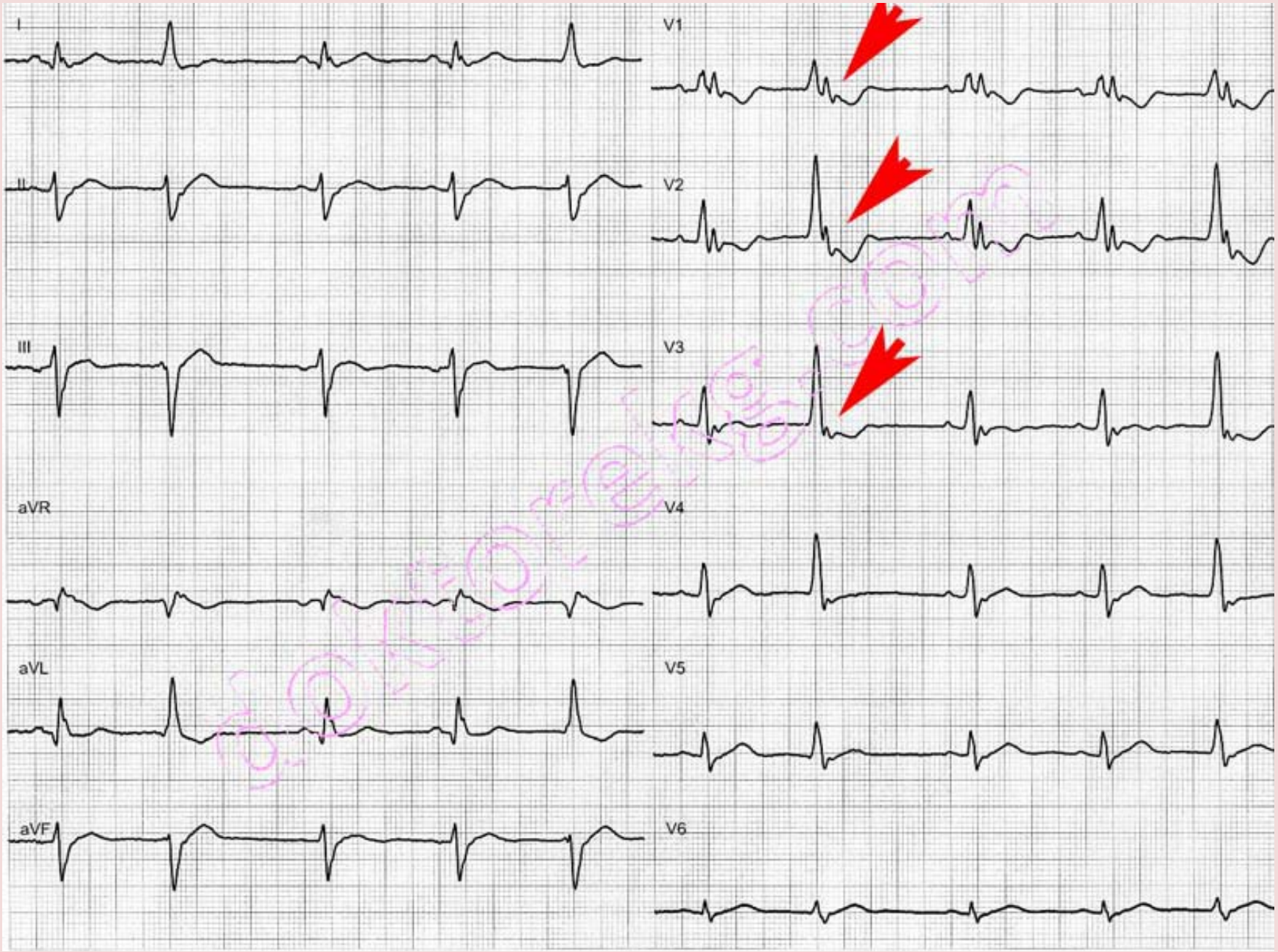


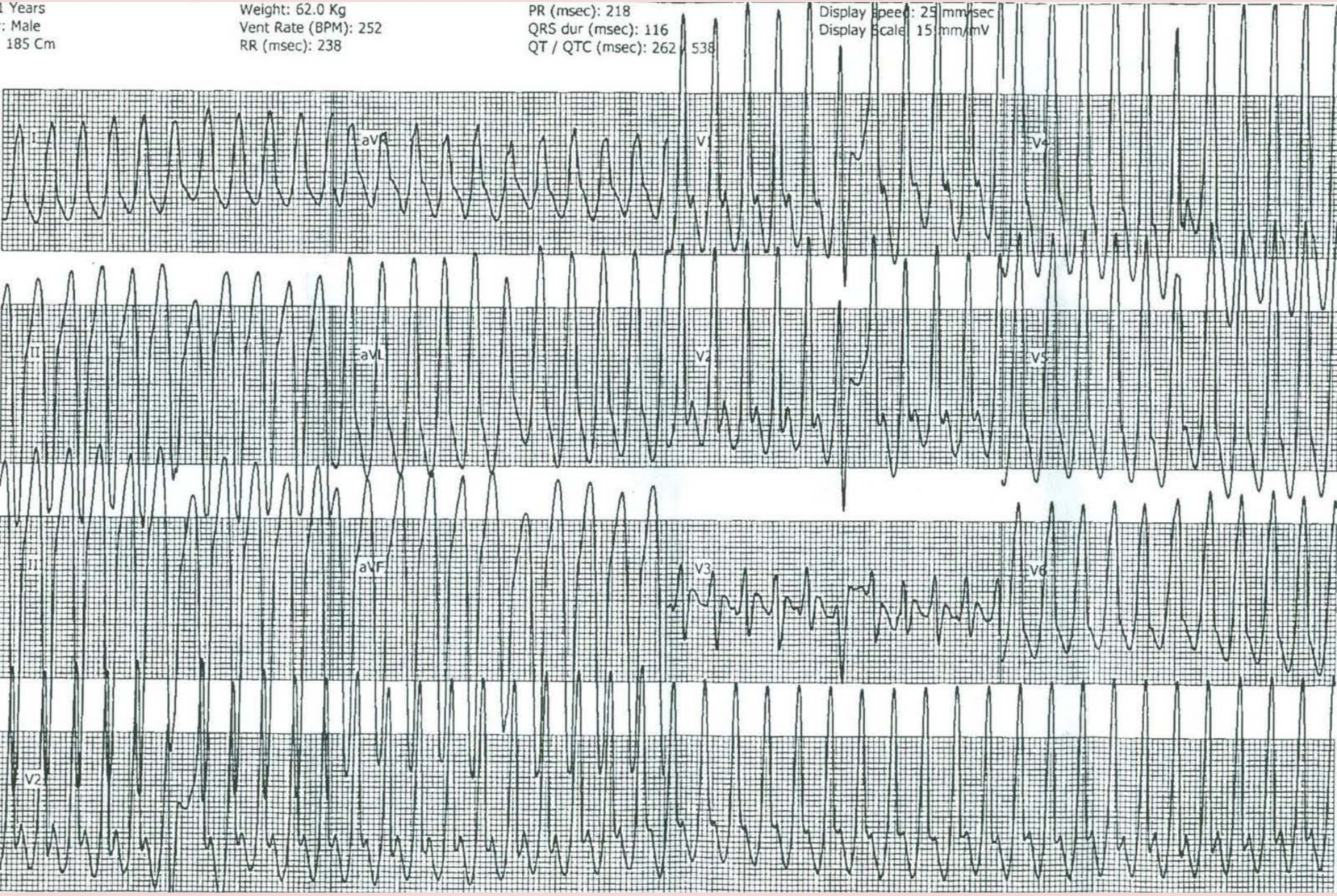
1. **“Incomplete RBBB” Pattern**
2. **V1, V2 Rs pattern**
3. **Inverted T waves, symmetrical, - Global**
4. **Epsilon’s waves**

ARVD ECG 2

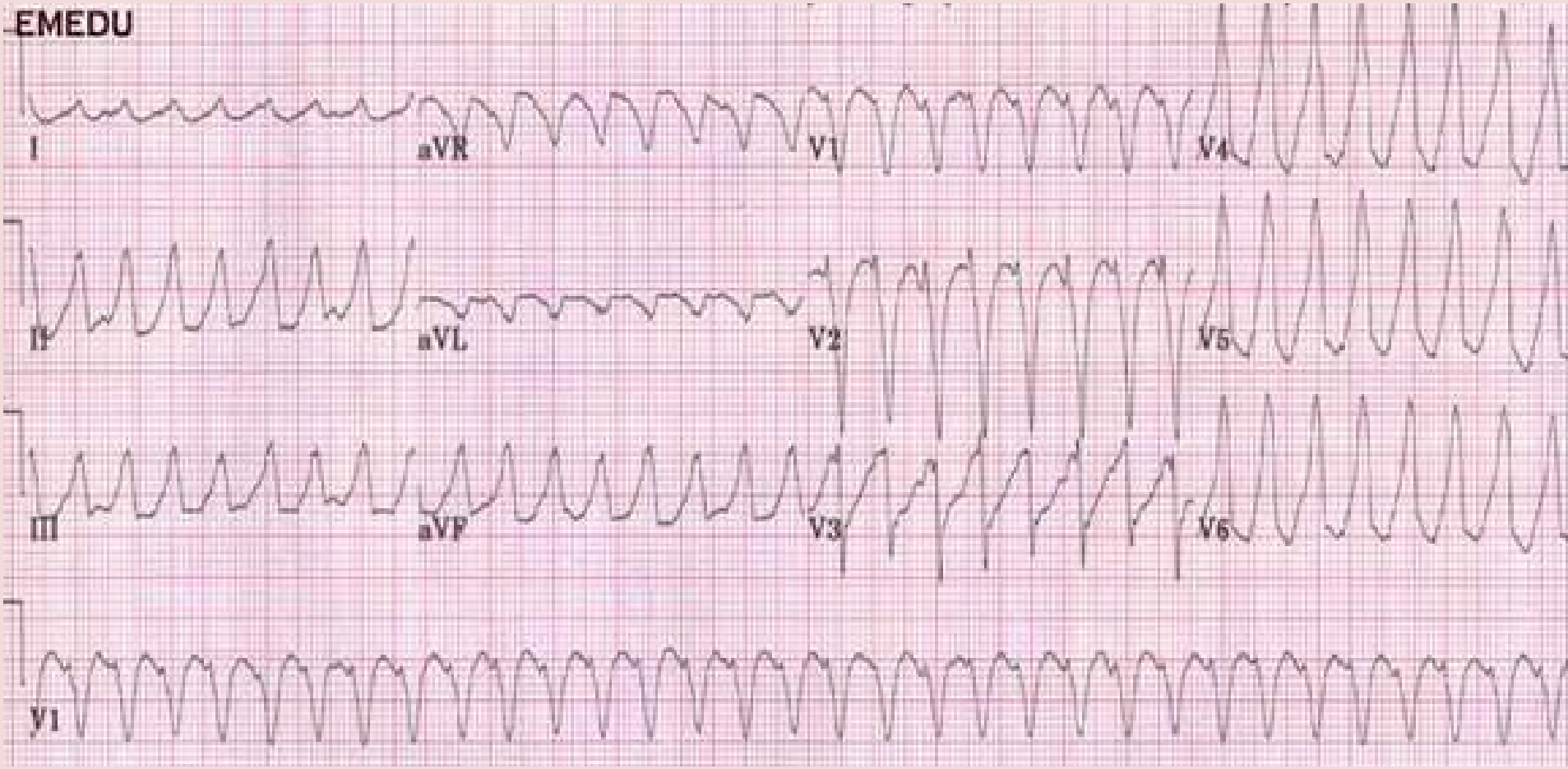


1. **"Incomplete RBBB" Pattern**
2. **V1, V2 Rs pattern**
3. **Inverted T waves, symmetrical, - Global**
4. **Epsilon's waves**


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

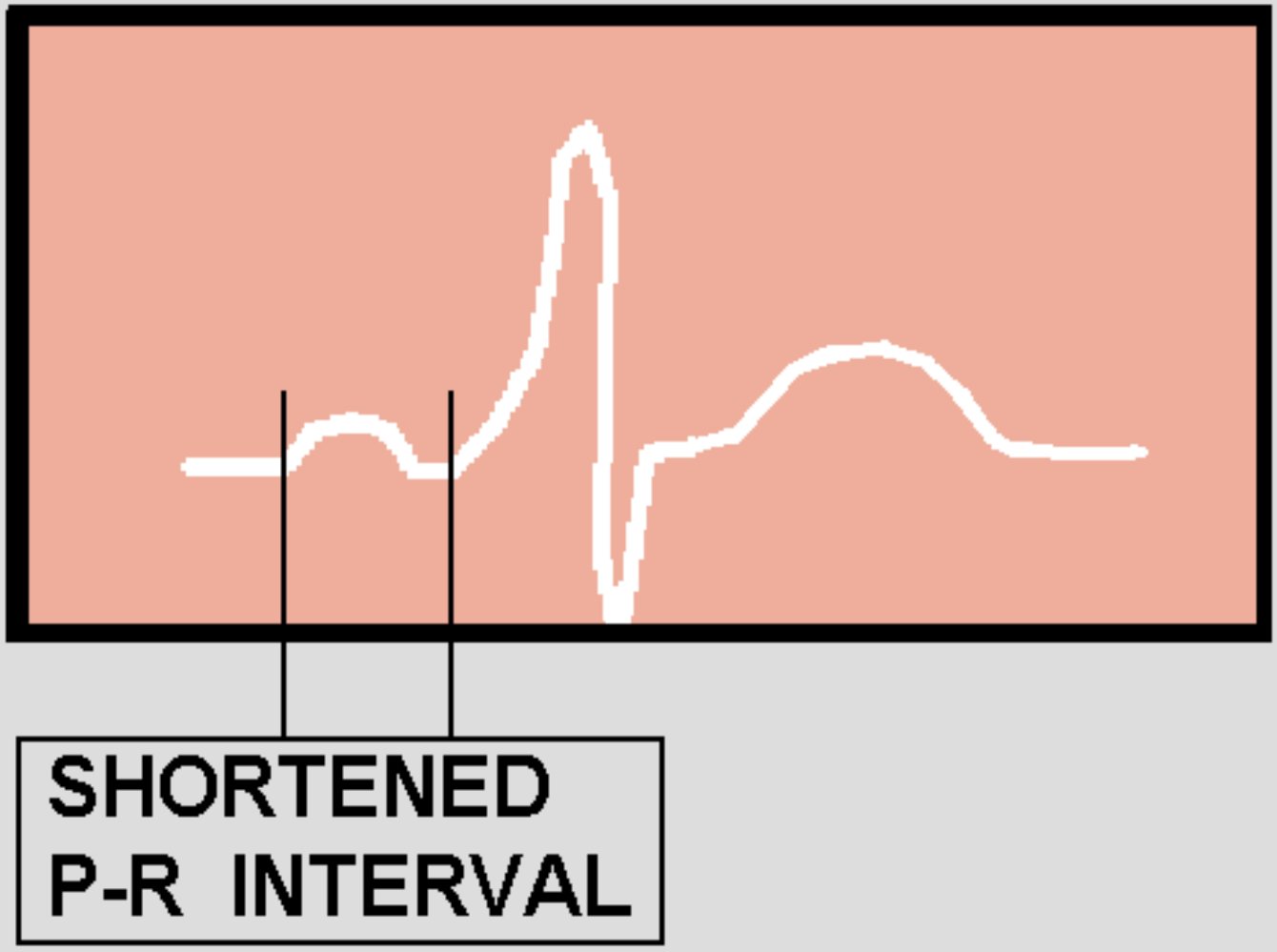

1 Years
: Male
185 Cm
Weight: 62.0 Kg
Vent Rate (BPM): 252
RR (msec): 238
PR (msec): 218
QRS dur (msec): 116
QT / QTC (msec): 262 / 538
Display Speed: 25 mm/sec
Display Scale: 15 mm/mV
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V2

ARVD INDUCED VT



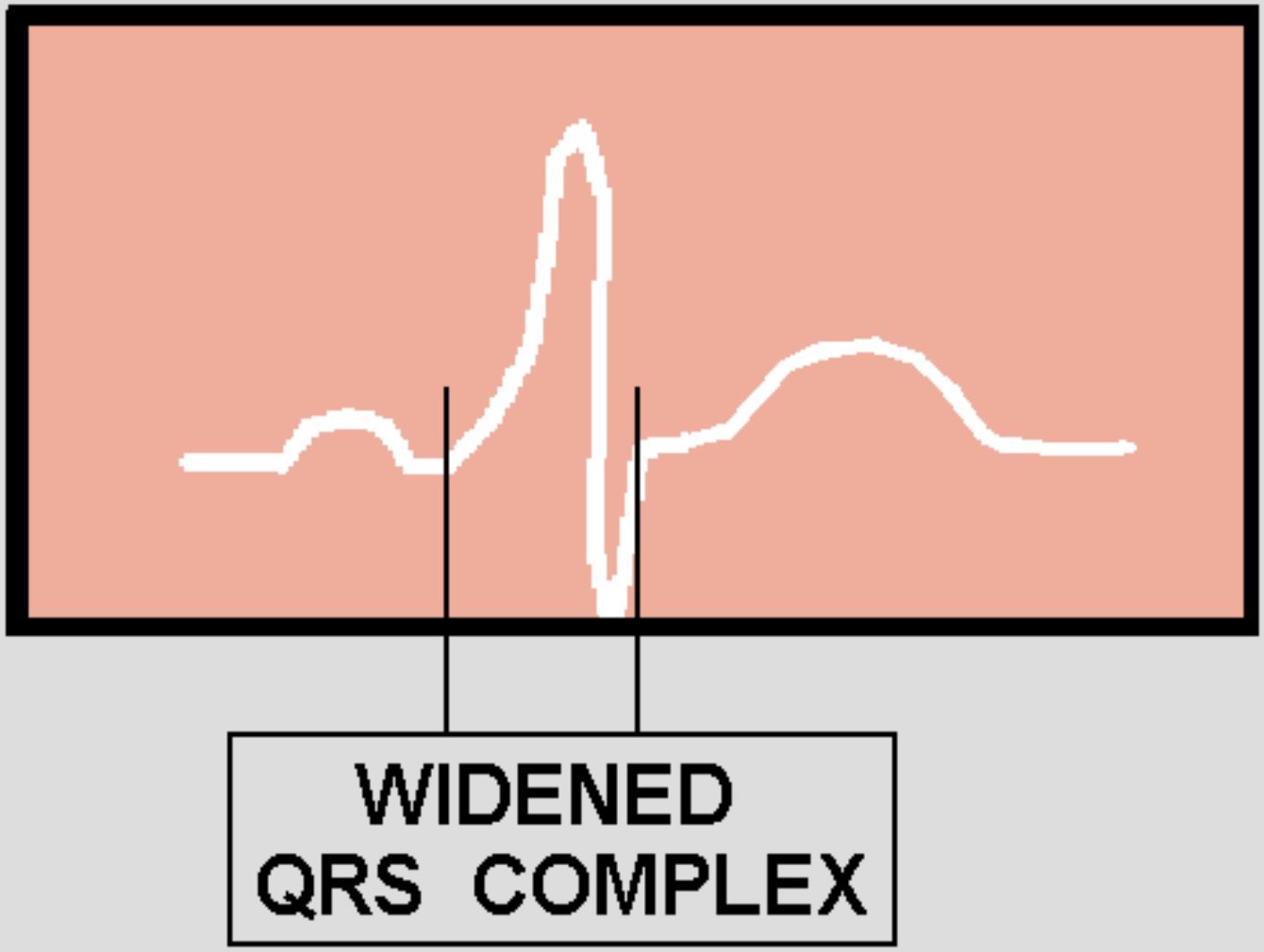
WOLFF-PARKINSON-WHITE

EKG CHARACTERISTICS



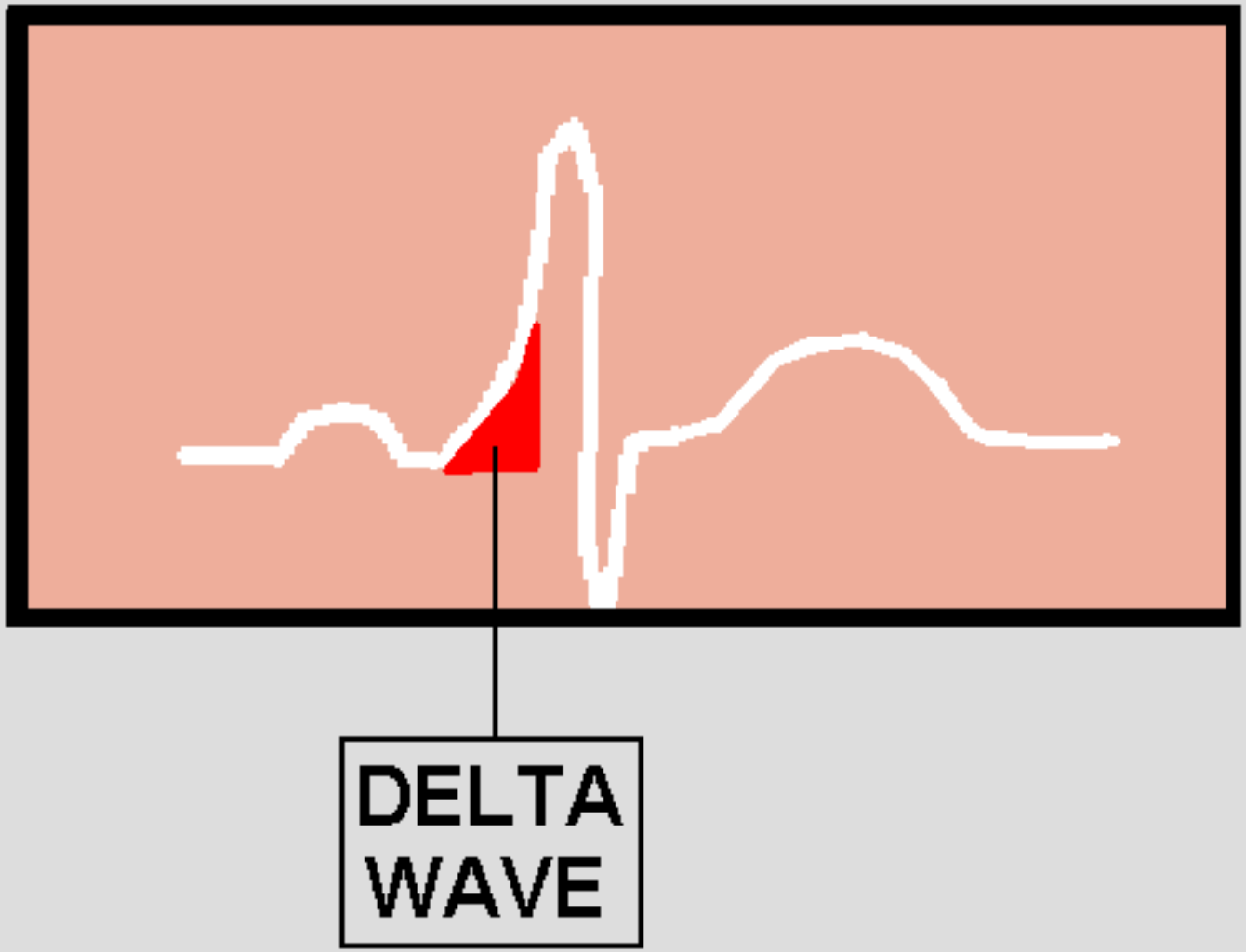
WOLFF-PARKINSON-WHITE

EKG CHARACTERISTICS



WOLFF-PARKINSON-WHITE

EKG CHARACTERISTICS



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

16 yr
Female Caucasian

Room:REC
Loc:20 Option:50

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

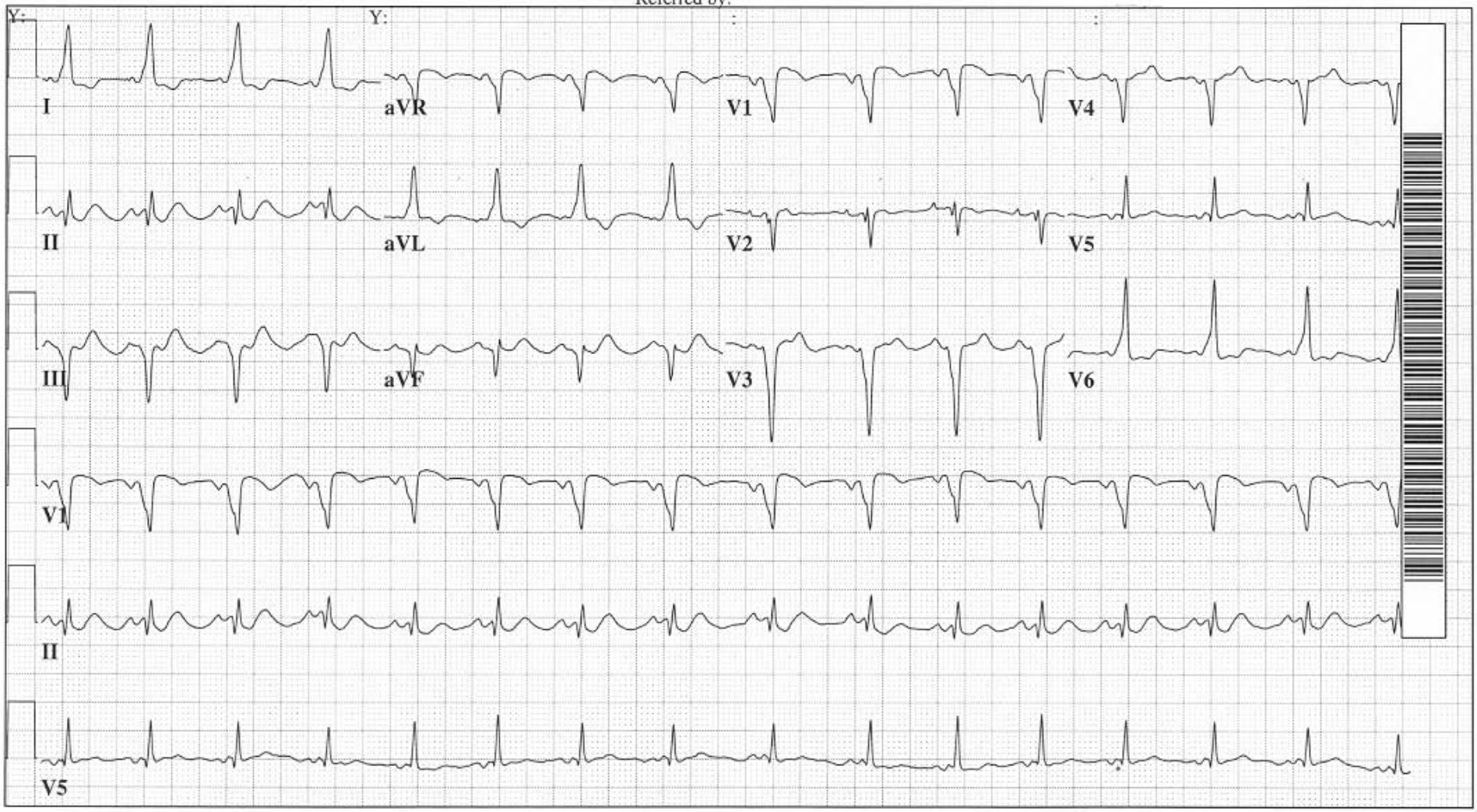
Normal sinus rhythm with sinus arrhythmia
Left atrial enlargement
Anterior infarct , age undetermined
Inferior infarct , age undetermined
ST & T wave abnormality, consider lateral ischemia
Wolff–Parkinson–White
Abnormal ECG
No previous ECGs available

History:Unknown
Technician: DP
Test ind:EKG

EKG CLASS #WR030100
60783

WOLFF-PARKINSON-WHITE TYPE B

Referred by:



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

EID:18 EDT: 16:01 17–MAY–1997 ORDER:

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

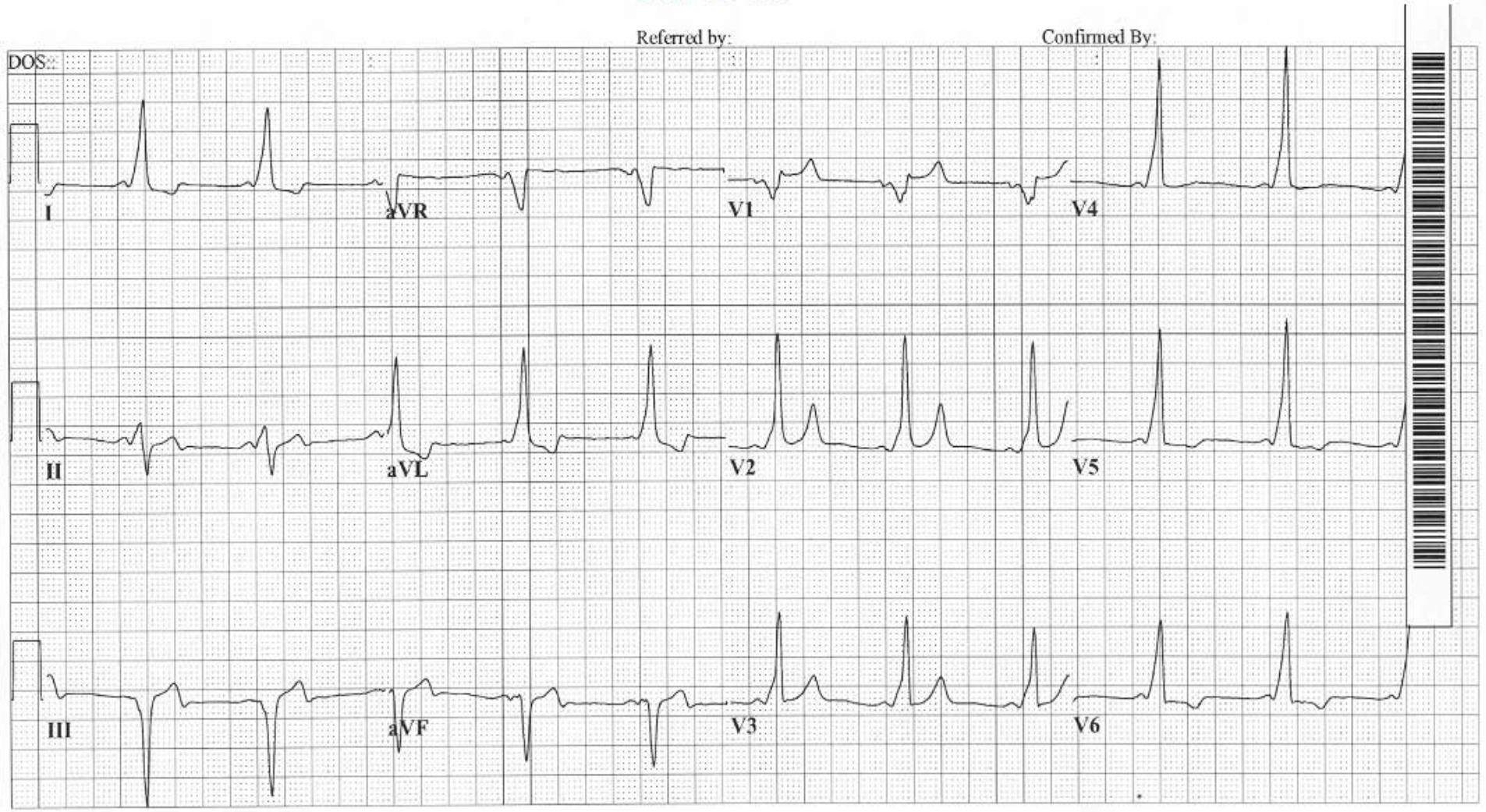
Normal sinus rhythm
Wolff-Parkinson-White
Abnormal ECG
No previous ECGs available

PRE-ABLATION

WOLFF-PARKINSON-WHITE TYPE A

Technician EKG CLASS #WR03696205

Referred by: Confirmed By:



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

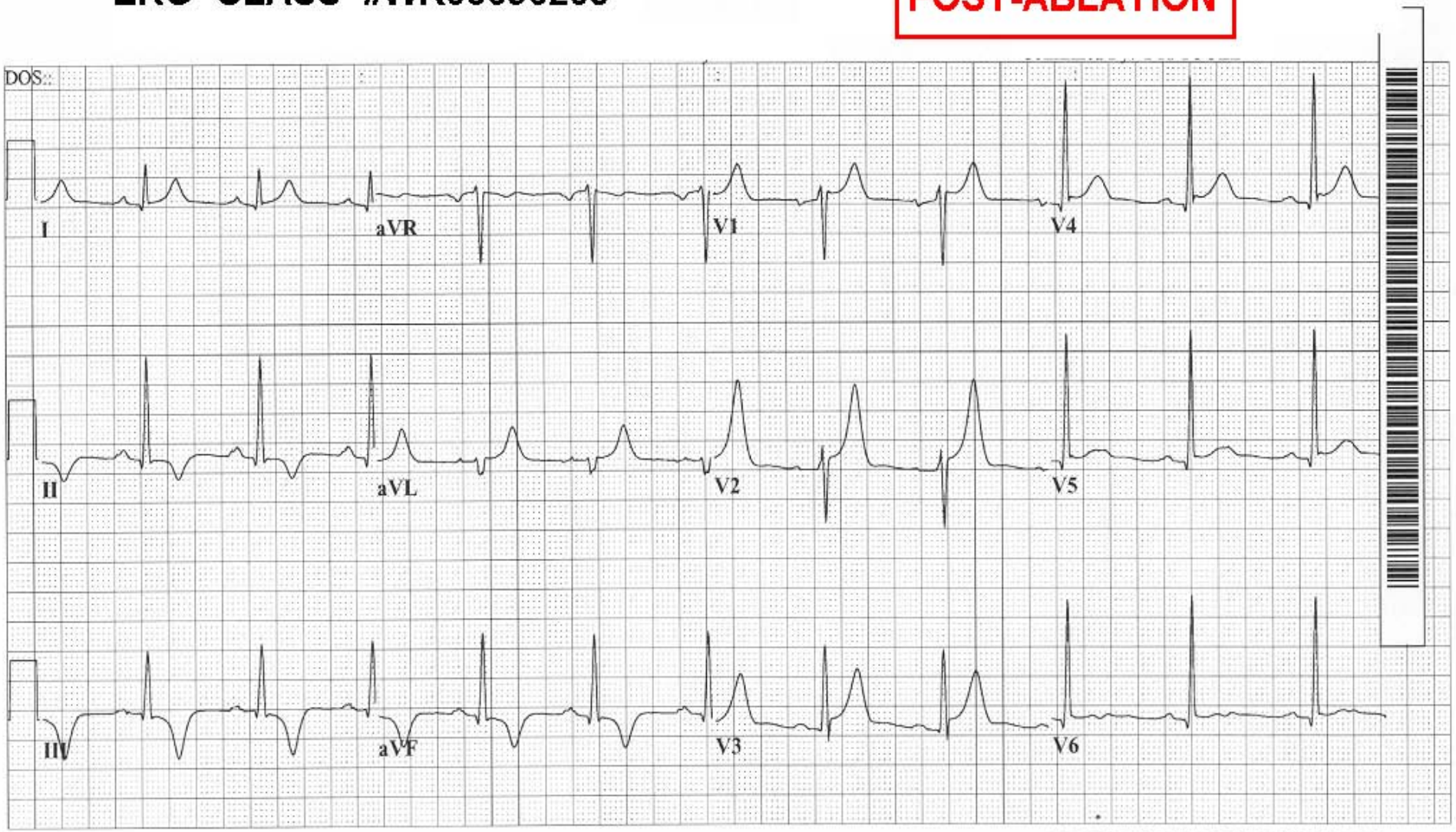
Room:426
Loc:5 Option:28

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

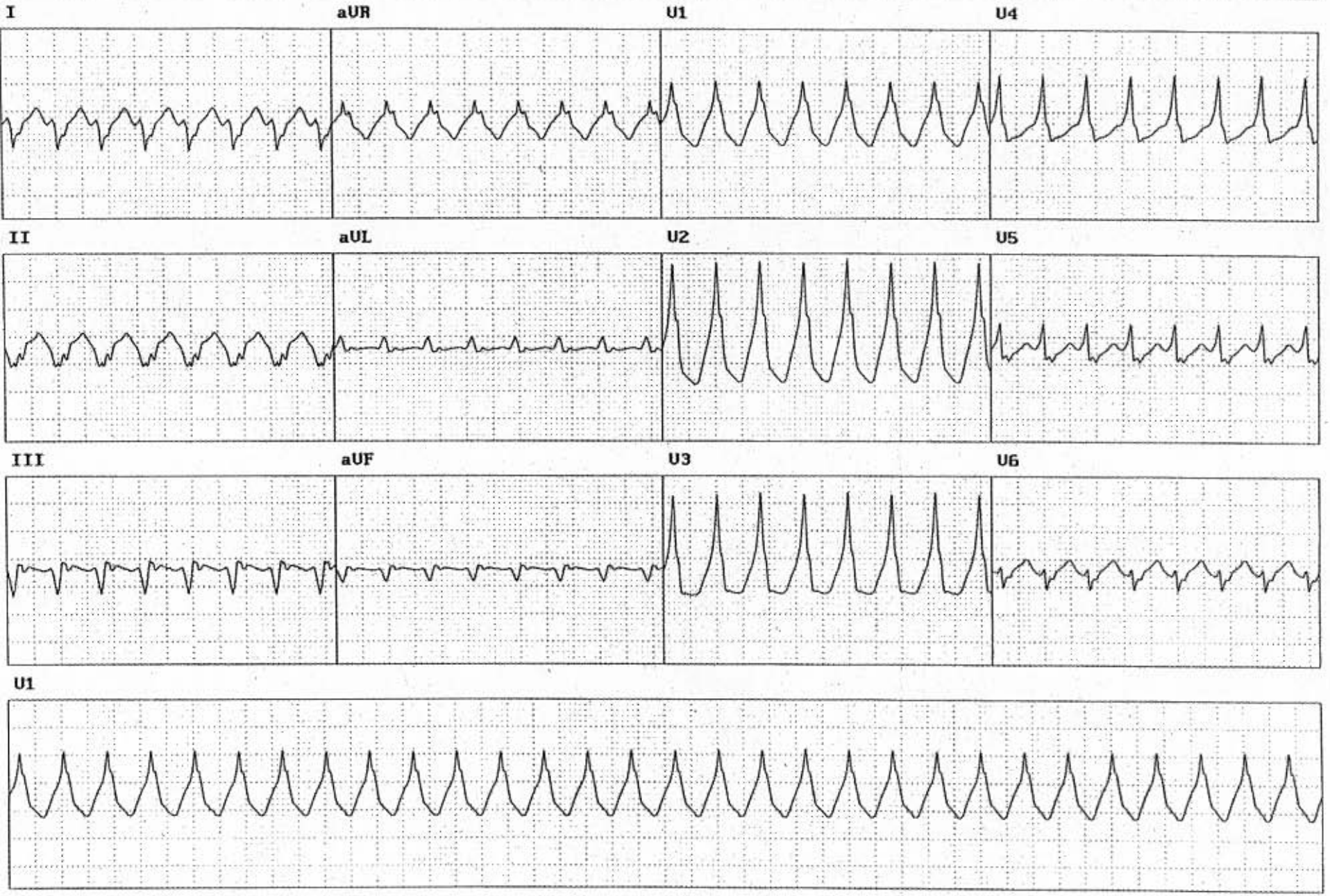
POST-ABLATION



25mm/s 10mm/mV 40Hz 005C 12SL 4 CID: 12

EID:17 EDT: 09:08 04-MAY-1999 ORDER:

05/04/99 14:16:00 25mm/sec 4.2mm/mU



ST. JOSEPH'S HOSPITAL W-P-W DOUBLE BY-PASS TRACT

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%

ST. JOSEPH'S HOSPITAL-

37 yr
Male Caucasian

Room:OP
Loc:8 Option:16

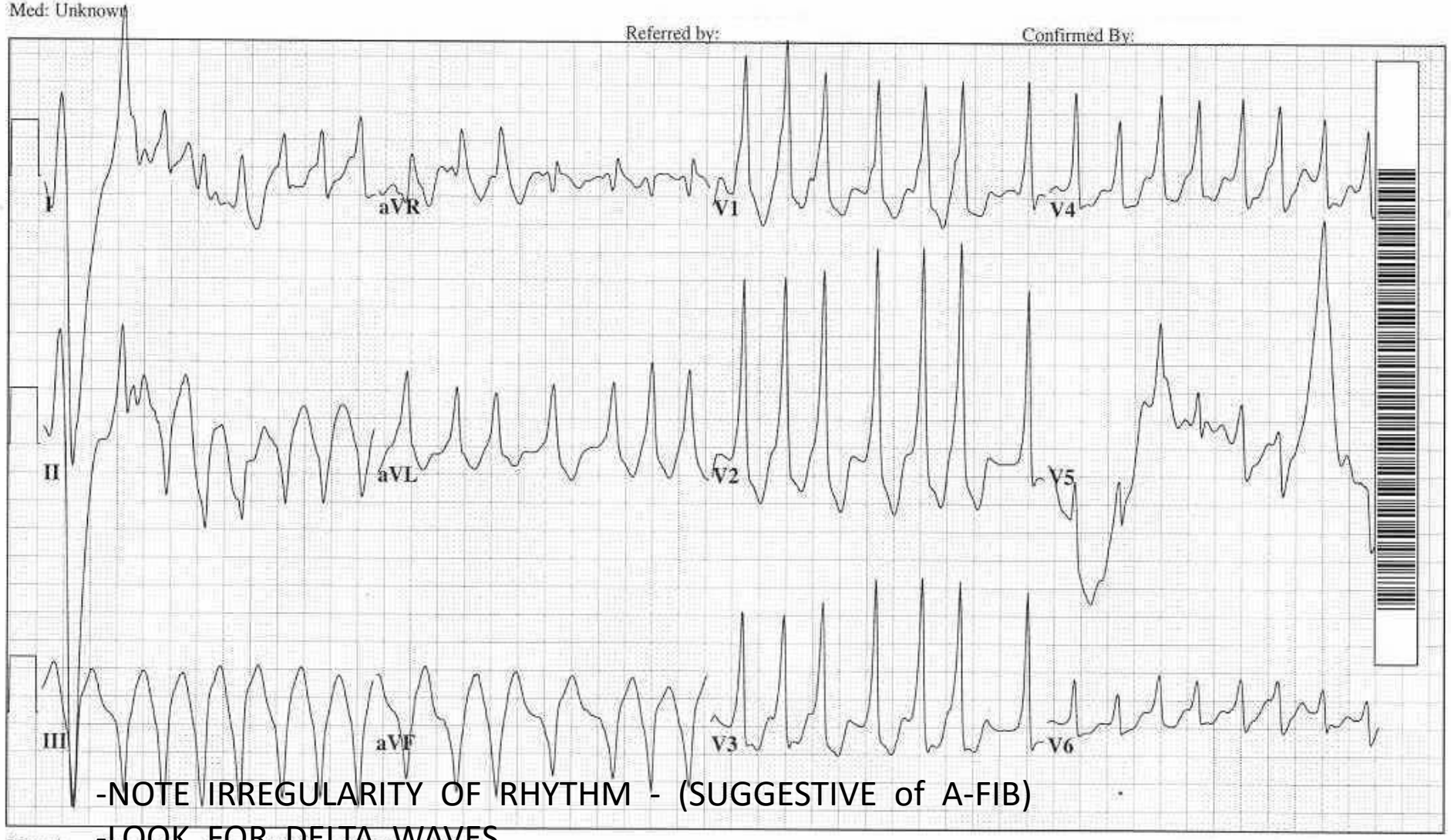
Vent. rate	180	BPM
PR interval	*	ms
QRS duration	148	ms
QT/QTc	284/491	ms
P-R-T axes	* -77	103

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

Med: Unknown

Referred by:

Confirmed By:



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

25mm/s 10mm/mV 40Hz 005C 125L 72 CID: 6

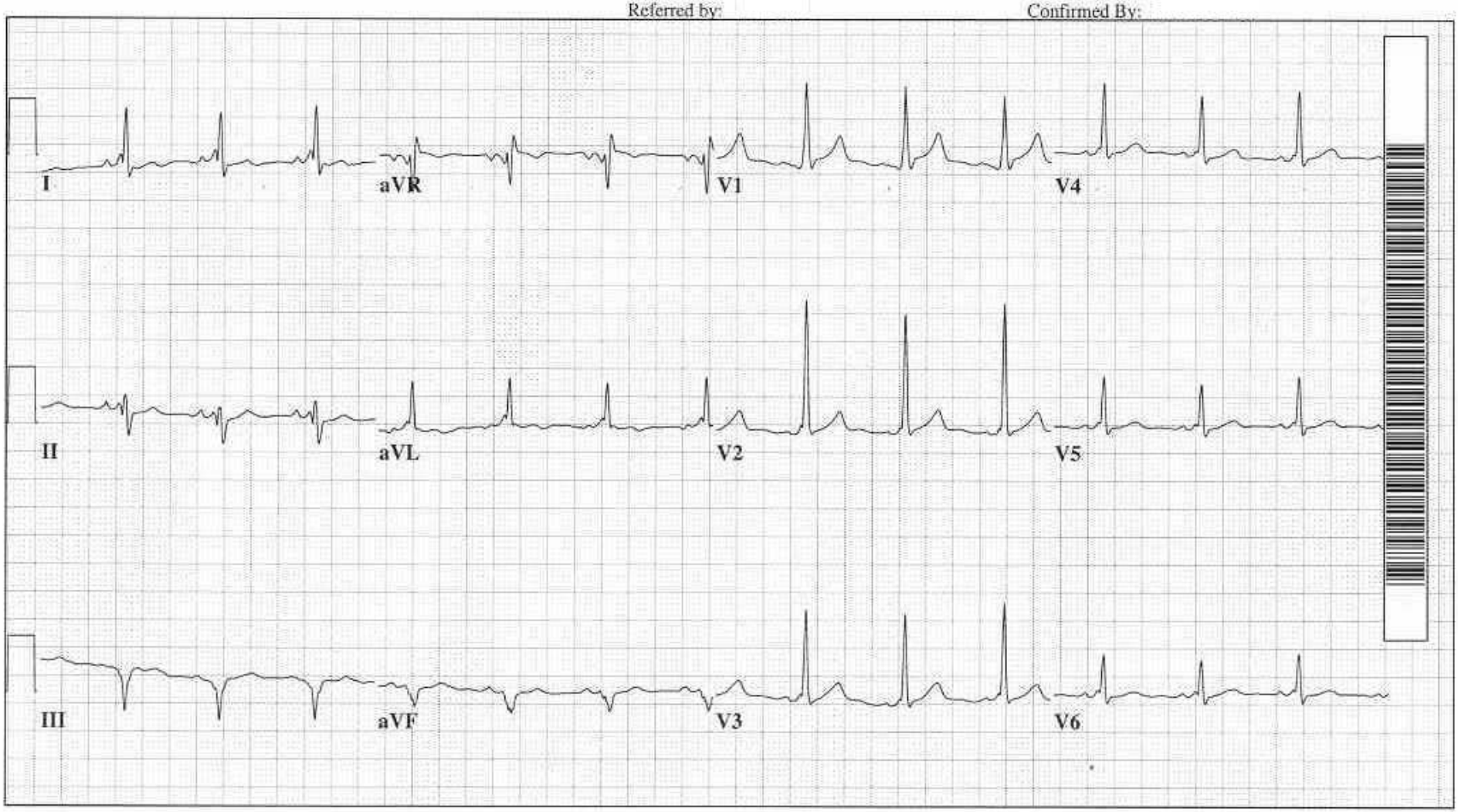
37 yr
Male Caucasian

Room:OP
Loc:8 Option:19

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

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

Referred by: Confirmed By:



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

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.***

WIDE COMPLEX TACHYCARDIA (QRS > 120 ms)

MONOPHASIC

ABC s

NO PULSE

GO TO
V - FIB
ALGORITHM !

PULSE - UNSTABLE

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

PULSE - STABLE

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

WIDE COMPLEX TACHYCARDIA (QRS > 120 ms)

MONOPHASIC

ABC s

NO PULSE

GO TO
V - FIB
ALGORITHM !

PULSE - UNSTABLE

- IMMEDIATE SYNC. CARDIOVERSION:
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  - -AMIODARONE

PULSE - STABLE

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

ST. JOSEPH'S HOSPITAL-

37 yr
Male Caucasian

Room:OP
Loc:8 Option:16

Vent. rate	180	BPM
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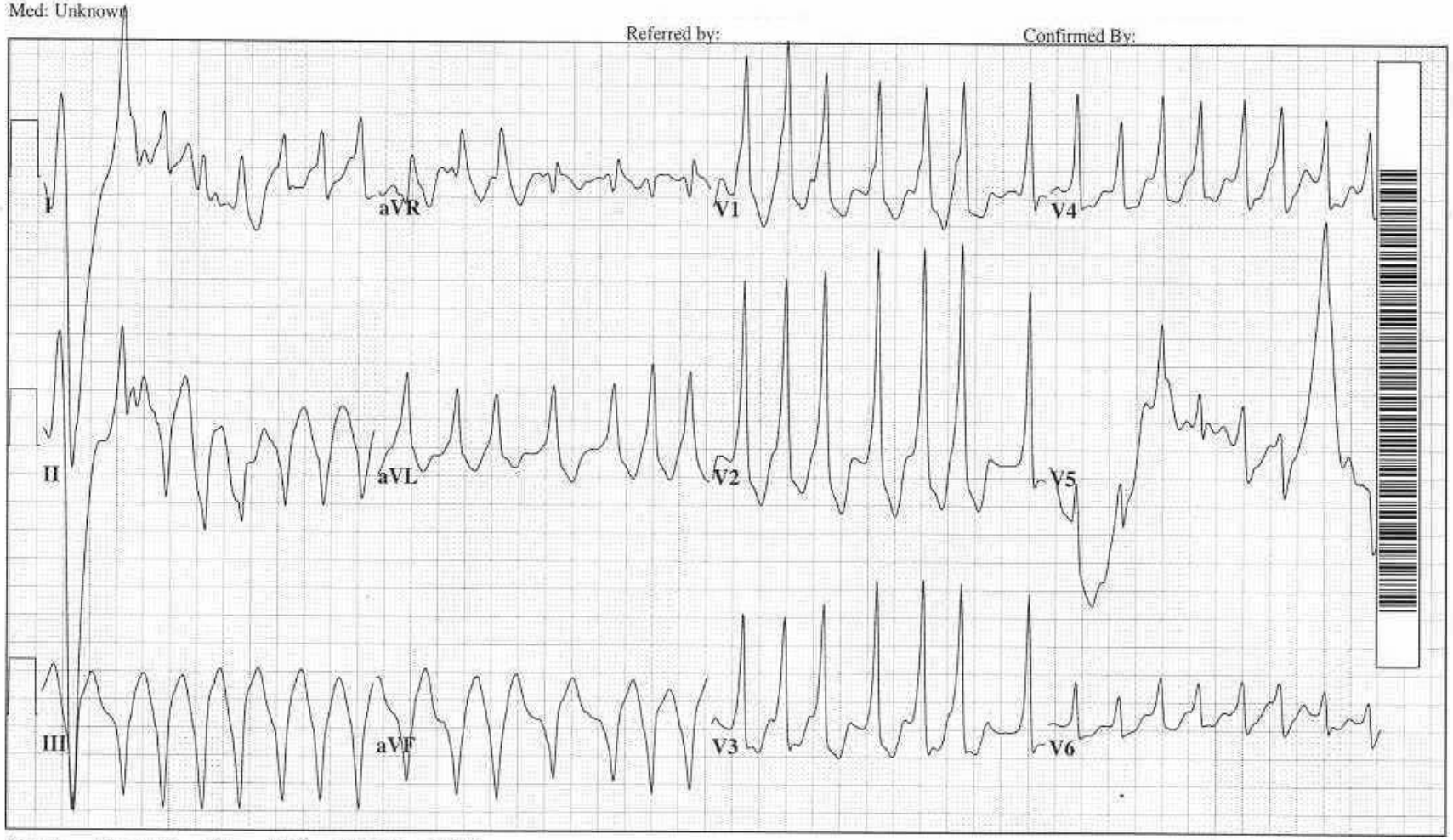
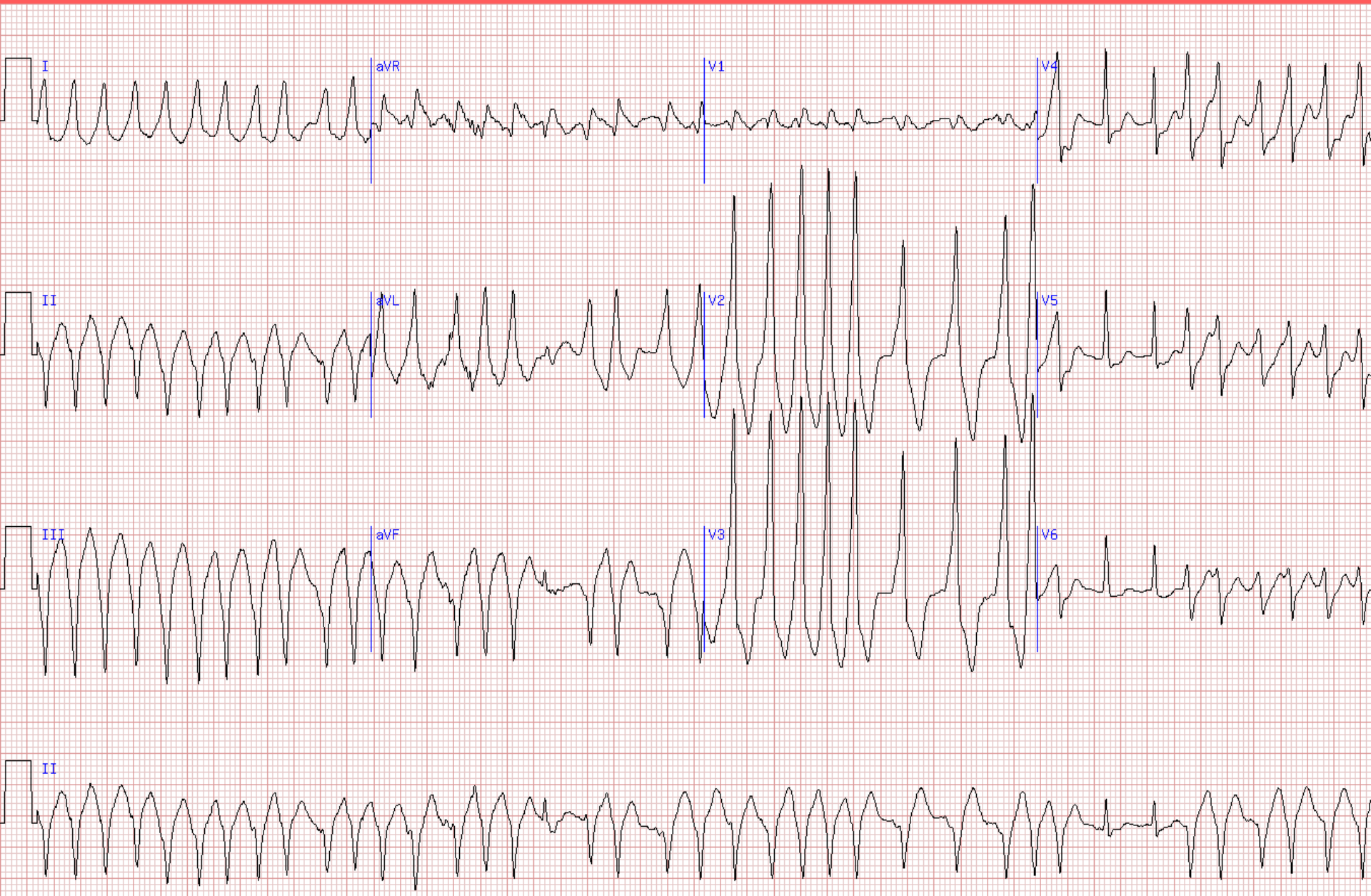


25mm/s 10mm/mV 40Hz 005C 12SL 72 CID: 0

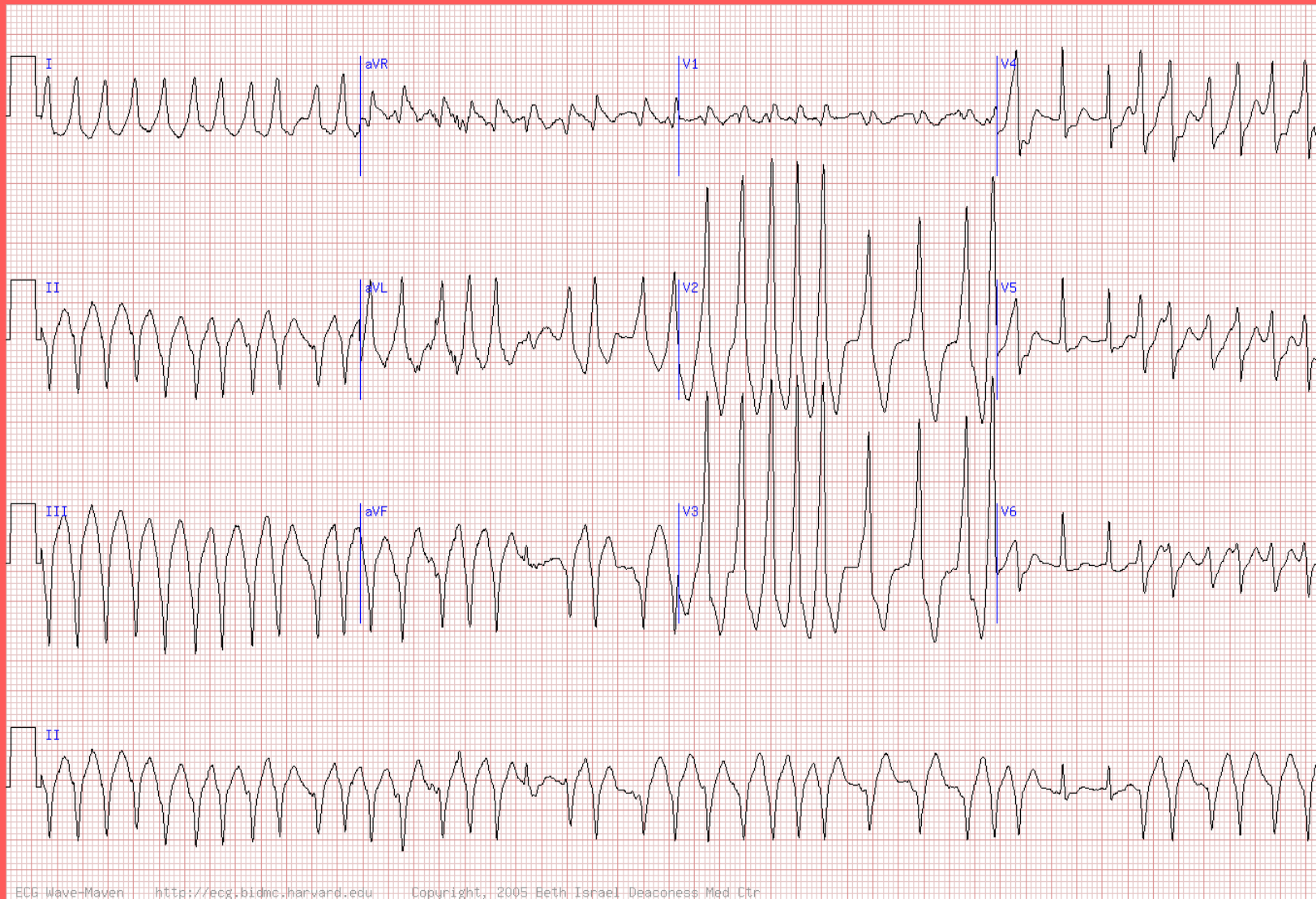
-NOTE IRREGULARITY OF RHYTHM - (SUGGESTIVE of A-FIB)
-LOOK FOR DELTA WAVES

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



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

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



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)

Are any abnormalities present on the 12 Lead ECG ?

Does patient have any abnormal or suspicious findings from the History/Physical/Screening form, including family histories suggestive of inherited cardiac disorders ?

YES

NO

3. Additional Testing as Indicated:
   - Echocardiogram
   - Exercise Stress Test
   - Electrophysiology Study
   - Cardiac Catheterization
   - Genetic Testing
   - CT/MRI/Nuclear Imaging
   - Other diagnostic procedures, as indicated
   - Other Consults, as indicated

Approve for Participation in Athletic Program !

Thank you !!

Questions ??

Appendix:

- The Mandatory ECG Screening Debate
- Understanding Genetic Transmission
- Marfan's Syndrome
- Correlation SIDS and LQTS
- Additional LQTS slides
- Additional Web-based Resources

Mandatory ECG Screening; The National Debate:

The ECG Screening Debate: some of the issues . . . .

PROS:

- Identify many SADS Conditions
- Quick and inexpensive Procedure
- Despite expenses associated with running a large-scale screening program, saving the life of one or a few patients who are otherwise "young and healthy" is worth it.

CONS:

- ECG's inherent issues with Sensitivity and Specificity (false negatives and positives)
- "False Negative" results – if taken at face value and patient is given "clean bill of health," could have catastrophic results
- "False Positive" results may delay or preclude healthy athletes from sports participation
- Large expense of such a program to net very few cases

Mandatory ECG Pre-participation Screening: The Italian Experience

A nationwide mandatory pre-participation athletic screening was introduced in Italy in 1982.

Mandatory ECG Pre-participation Screening: The Italian Experience

Corrado et al compared mortality rates in athletes before the introduction of mandatory screening ECGs versus mortality rates after implementation of the program

Mandatory ECG Pre-participation Screening: The Italian Experience

The study concluded that after implementation of mandatory pre-participation ECG screening of athletes in Italy, ***the annual incidence of sudden cardiovascular death in athletes decreased by 89%.*** *JAMA.* 2006;296(13):1593-1601

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)

A Scientific Statement From the American Heart Association and the American College of Cardiology

Endorsed by the Pediatric and Congenital Electrophysiology Society and American College of Sports Medicine

"A number of educational institutions have screened, or are currently systematically screening, prospective athletes for heart disease with a variety protocols that include echocardiograms or ECGs: Harvard University, University of Wisconsin, Howard University, University of Virginia, Georgetown University, Stanford University, University of Washington, and other institutions with programs in selected sports. . . ."

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)

A Scientific Statement From the American Heart Association and the American College of Cardiology

Endorsed by the Pediatric and Congenital Electrophysiology Society and American College of Sports Medicine

"Such screening efforts have benefited some young individuals through identification of potentially life-threatening cardiovascular disorders. Such initiatives have been supported consistently by the 1996 and 2007 AHA Scientific Statements, as well as the present document."

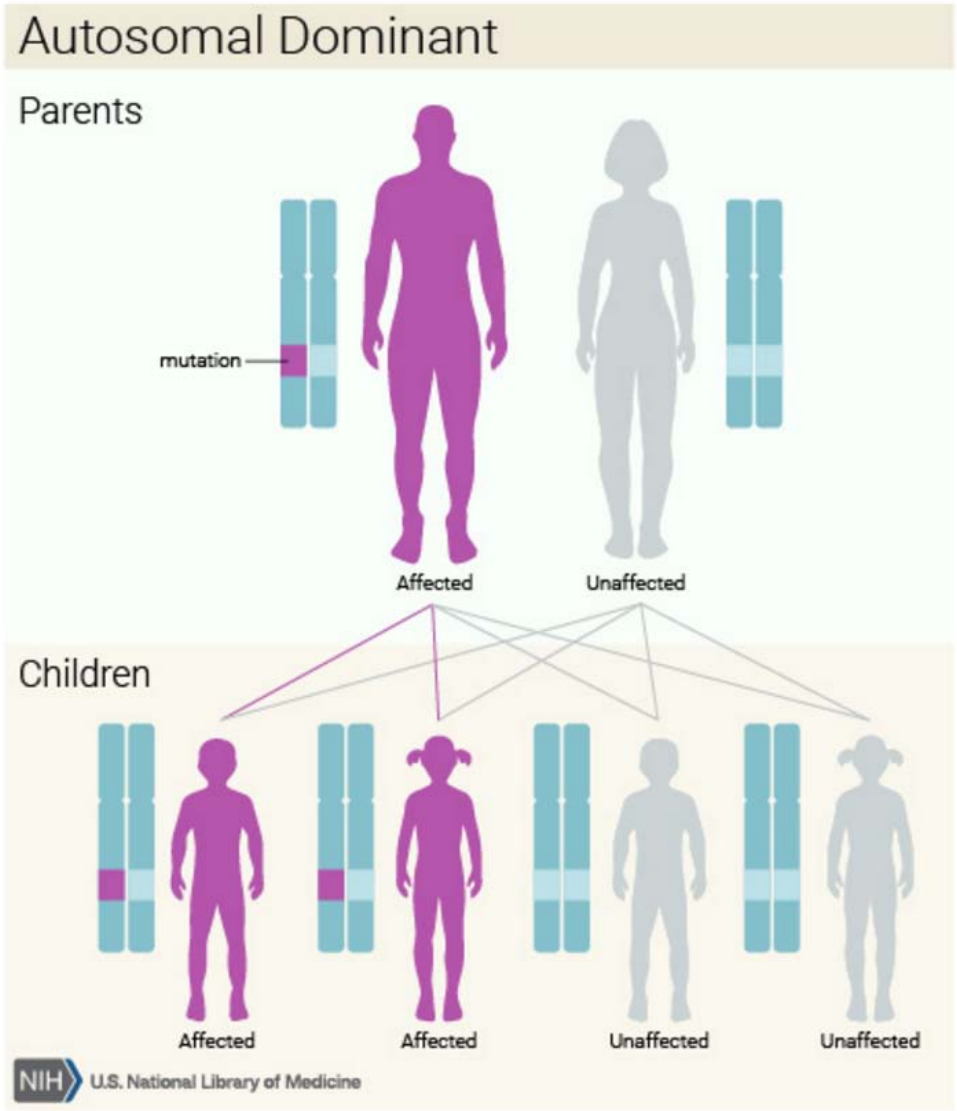
The ECG Screening Debate

Scientific Journal Article in favor of ECG screening for all infants, children and young Adults:

"If one uses just a H&P, only the small number with positive symptoms or history, and an abnormal physical examination will be identified. Unfortunately, only 50% of youth who have experienced Sudden Cardiac Arrest describe antecedent symptoms and only 16% know of a positive family history."

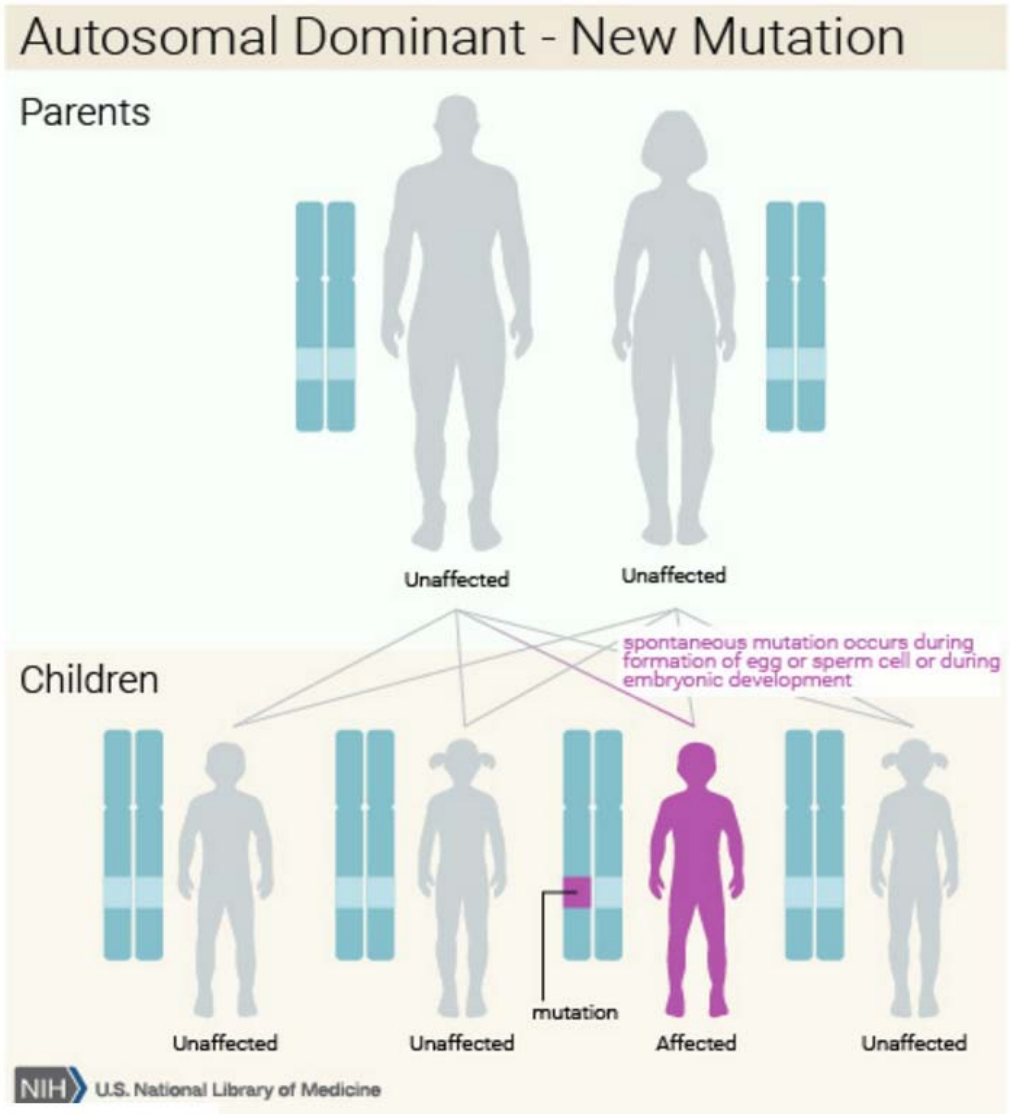
Circulation 2014; 130: 688-697

Understanding Genetic Transmission



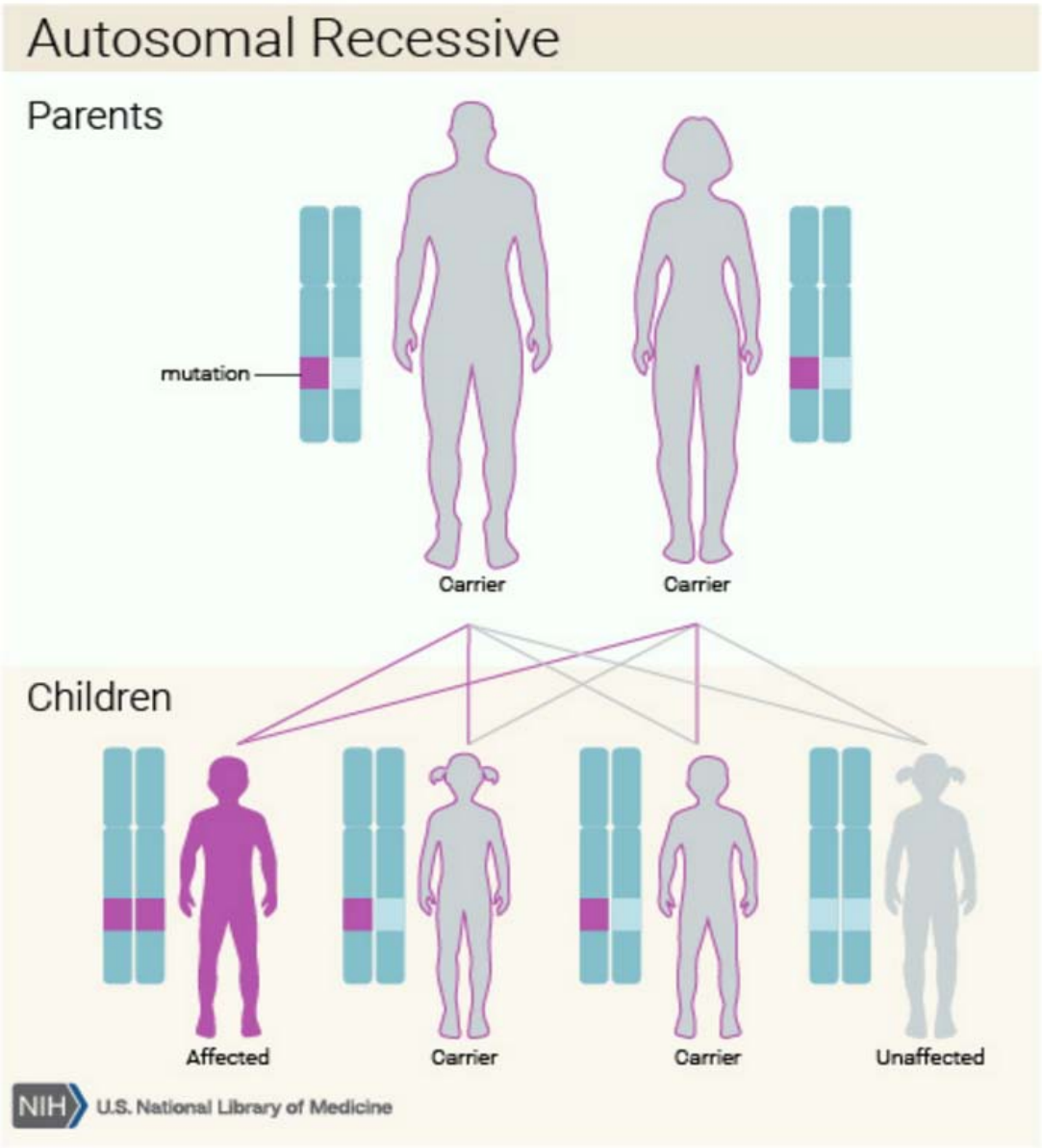
Credit: U.S. National Library of Medicine

Understanding Genetic Transmission



Credit: U.S. National Library of Medicine

Understanding Genetic Transmission



Marfan's Syndrome

- **Marfan syndrome is a genetic disorder that affects the body's connective tissue.**
- **Body systems affected:**
  - **skeleton,**
  - **eyes,**
  - **heart,**
  - **lungs and**
  - **blood vessels**

- **For more information:**

The Marfan Foundation

Physicial Characteristics of Marfan's Syndrome

- **Long arms, legs and fingers**
- **Tall and thin body type**
- **Curved spine**
- **Chest sinks in or sticks out**
- **Flexible joints**
- **Flat feet**
- **Crowded teeth**
- **Stretch marks on the skin that are not related to weight gain or loss**

Marfans- Cardiovascular Abnormalities:

- Valvular disorders: Mitral Regurgitation / Prolapse.
- Septal defects
- Aortic Root Dilitation
- Wolff-Parkinson-White syndrome (wide and narrow QRS tachycardias)
- Atrial Fibrillation
- Ventricular Dysrhythmias and Sudden Death

Interesting AHA Circulation Journal from 1955

Additional Marfan's Syndrome Resources

- Marfan's syndrome: natural history and long-term follow-up of cardiovascular involvement, Marsalese et al, JACC 1989; 422-428

SIDS Correlation to LQTS

Schwartz et al instituted ambitious screening studies with 12-lead ECGs of >30,000 healthy neonates in the first week of life, who were followed up for 1 year and subsequently a study of almost 45 000 neonates at 15 to 25 days of life (1). A prolonged QT interval was strongly associated with SIDS, and LQTS mutations in the sodium channel (2).

1. Schwartz PJ, Priori SG, Dumaine R, et al. A molecular link between the sudden infant death syndrome and the long-QT syndrome. N Engl J Med. 2000;343:262–7.
2. Arnestad M, Crotti L, Rognum TO, et al. Prevalence of long-QT syndrome gene variants in sudden infant death syndrome. Circulation.2007;115:361–7

SIDS Correlation to LQTS

- Stillbirths, Sudden Infant Deaths, and Long-QT Syndrome AHA Circulation, 2004: Schwartz, Peter
- A Molecular Link between the Sudden Infant Death Syndrome and the Long-QT Syndrome, P Schwartz et al, NEJM 2000; 343:262-267

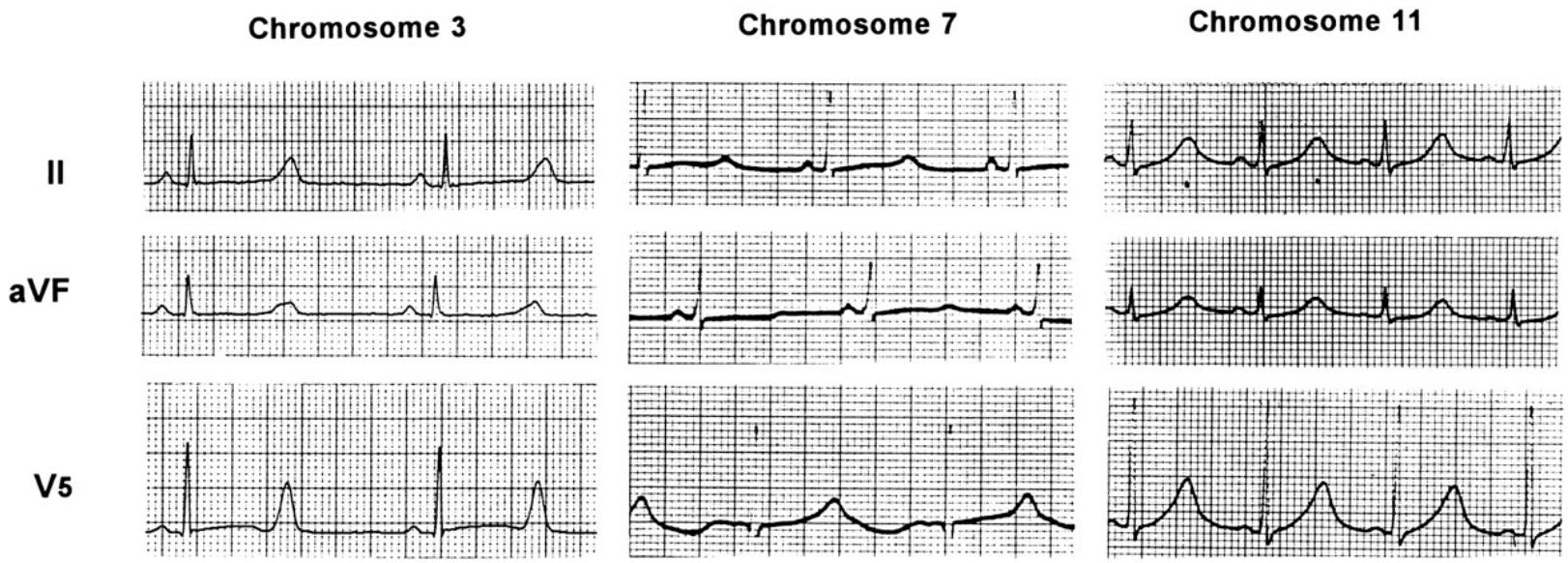
- Click here for PDF version of above article

CAUSES OF PROLONGED QT INTERVAL:

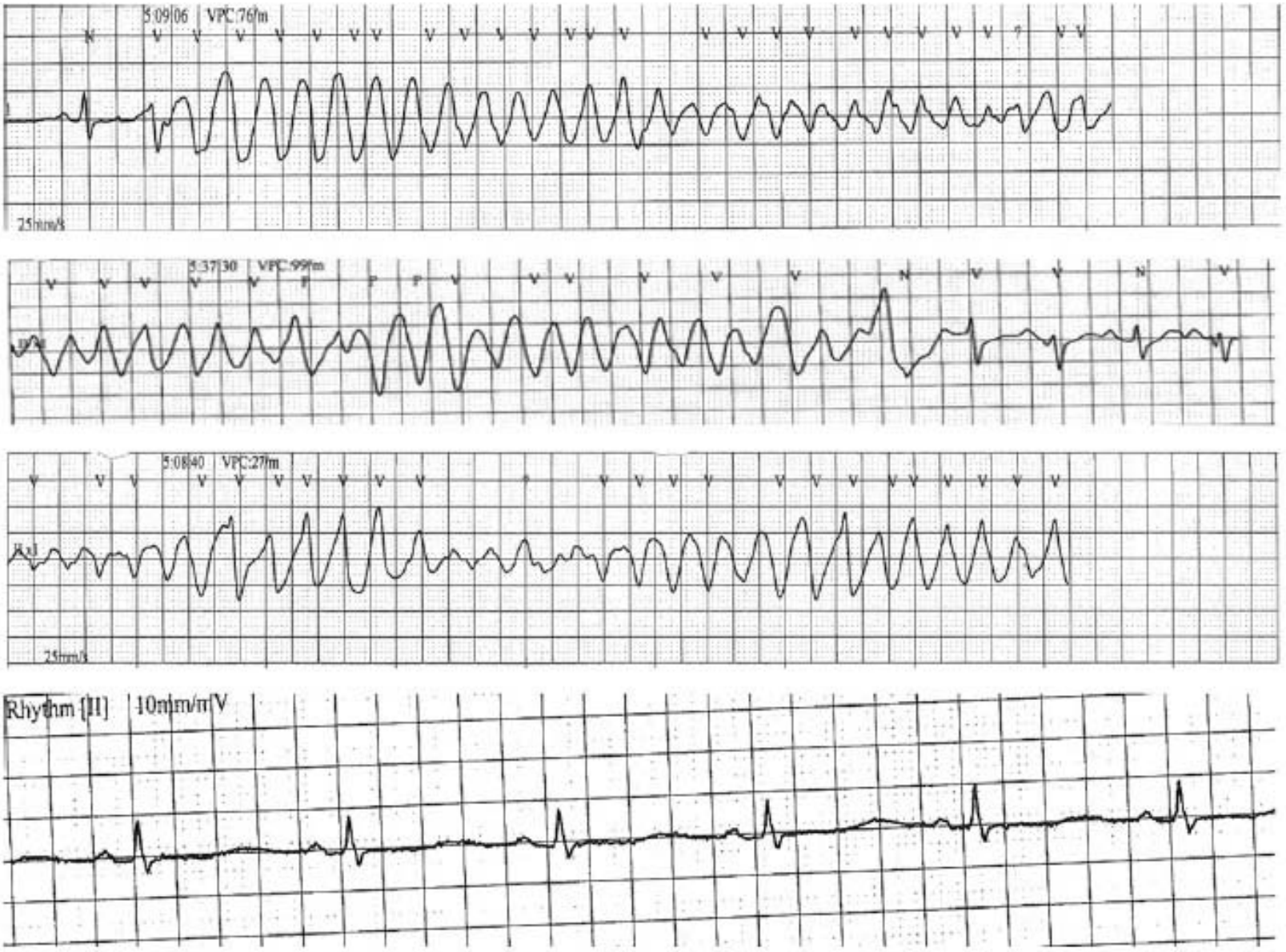
- PHARMACOLOGICAL (see www.longQT.org)
- ELECTROLYTE ABNORMALITIES. most common: HYPOKALEMIA, HYPOMAGESEMIA, HYPOCALCEMIA
- GENETICALLY ACQUIRED
- FEMALE GENDER
- DIABETES MELLITUS
- ANOREXIA
- HEPATIC DISORDERS
- INTRACRANIAL HEMORRHAGE
- HYPOTHYROIDISM
- LIQUID PROTEIN DIET

SOURCE: "ACQUIRED LONG QT SYNDROME," A. CAMM, et. al, 2004, BLACKWELL PUBLISHING, p. 21

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.

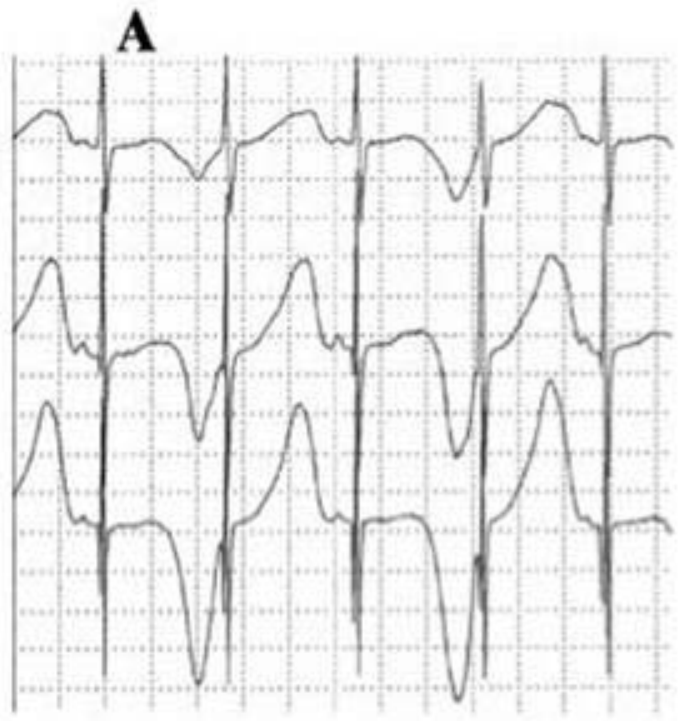


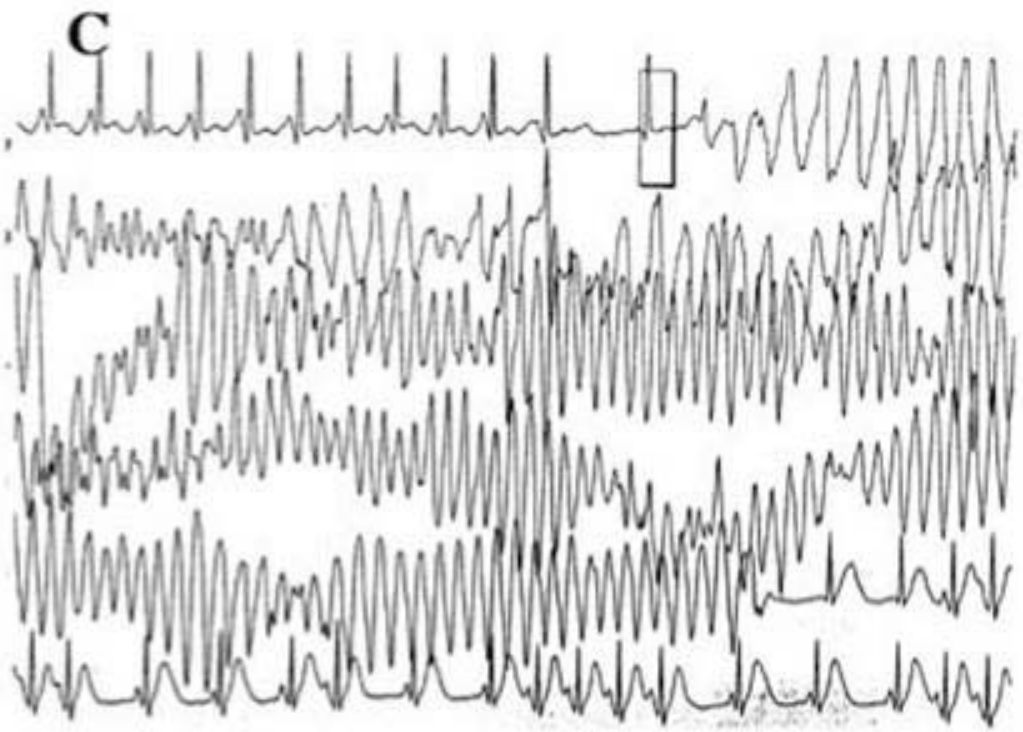
Moss A et al. Circulation 1995;92:2929-2934

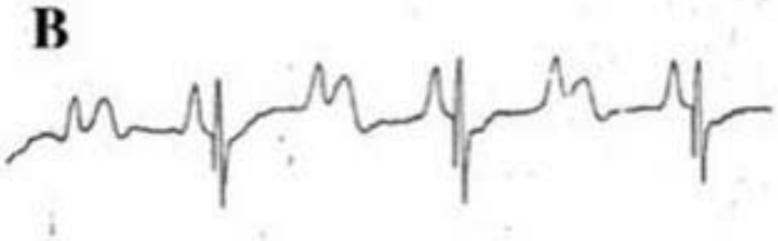


Rhythm strip of II showing QTc of 720 msec at admission

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







QT Prolonging Meds: Resources

http://www.sads.org/living-with-sads/Drugs-to-Avoid#.Vwm1yqQrI2w

http://www.brugadadrugs.org/

https://crediblemeds.org/pdftemp/pdf/DrugsToAvoidList.pdf

- 2014 AHA ACC Assessment of 12 Lead ECG as Screening for SADS

Additional Resources

- AHA Circulation: Impact of Laboratory Molecular Diagnosis on Contemporary Diagnostic Criteria for Genetically Transmitted Cardiovascular Diseases: Hypertrophic Cardiomyopathy, Long-QT Syndrome, and Marfan Syndrome

- Trends in Sudden Cardiovascular Death in Young Competitive Athletes After Implementation of a Preparticipation Screening Program

This presentation has been prepared by:

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For the

Society of Cardiovascular Patient Care's

19th Annual Congress

May 27, 2016

Miami, Florida

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