~ Back to Basics ~

ECG in Diagnosis, Prevention and Treatment of Sudden Cardiac Death

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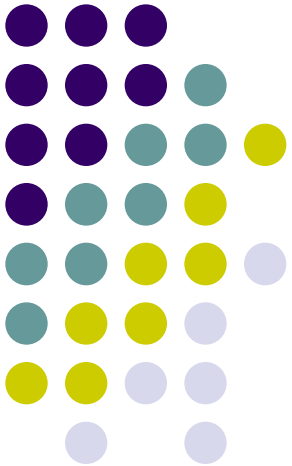


米高積遜猝死 歌迷哭了

今晨一時版

「Though we're far apart, you're always in my heart」（儘管我們分隔，你常在我心……）米高積遜（Michael Jackson）名曲《You are not alone》的歌詞，成為全球樂迷送別他的心聲。被喻為「流行樂之王」（King of Pop）的米高積遜，在美國時間周四下午心跳停頓猝死，終年五十歲。他原定下月舉行「謝幕」巡迴演唱會，惜撒手塵寰，無法向樂迷「謝幕」。

米高積遜於美國西岸時間周四中午十二時在洛杉磯居所中突感不適，不久便昏迷，心跳停頓。私人醫生曾爲他急救，家人報警，救護員抵達時，他已停止呼吸，脈搏亦停止。

家中昏迷 心跳停頓

他送抵醫院後，醫生搶救達一小時，但返魂乏術。其遺體由直升機送往洛杉磯縣法醫事務所，周五驗屍。

■米高積遜在居所昏迷，送院後不治。

家人相信他死於心臟病發，但英國《太陽報》引述加州大學洛杉磯分校醫科中心急症室消息稱，米高助手曾透露，米高曾注射一種名爲「Demerol」的止痛劑後，不久呼吸困難陷入昏迷，最後搶救無效。

米高自八十年代展開事業高峰，無數樂曲成爲經典，包括《Thriller》及《Beat it》等。其唱片在全球總銷量高達七億五千萬張，先後獲得十三項格林美音樂獎，成爲首名紅遍全球的黑人歌手。他近年開始隱居，今年三月曾宣佈復出，準備下月初舉行五十場「謝幕」世界巡迴演唱會，後來演唱會延期至下月十三日，當時傳出其健康出問題。

助手指死於注射止痛劑

全球樂迷知悉噩耗，有人不相信，有人泣不成聲，悲慟不已，自發舉行悼念活動。一代歌神永遠活在歌迷心中。

相關新聞見P2、P24及P26版

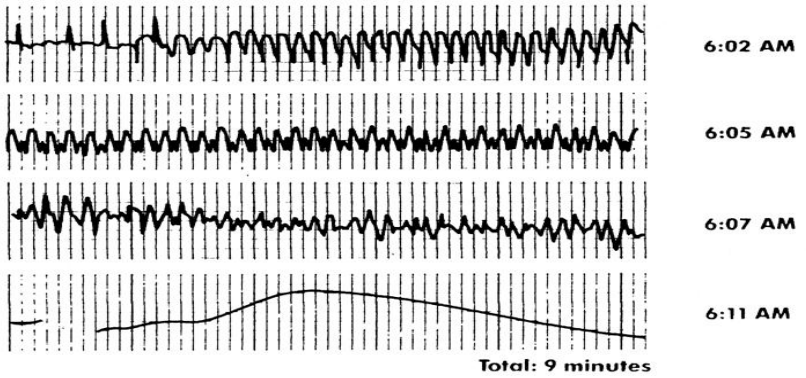
Michael Jackson died of sudden death in 2009

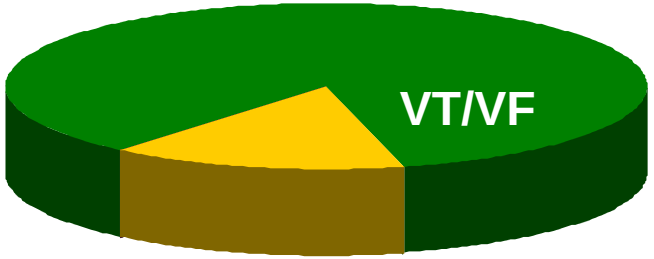
Sudden Cardiac Death (SCD)
心臟猝死

Definition

- Death from **unexpected circulatory arrest**, usually owing to a **cardiac arrhythmia** occurring **within one hour** of the onset of symptoms.

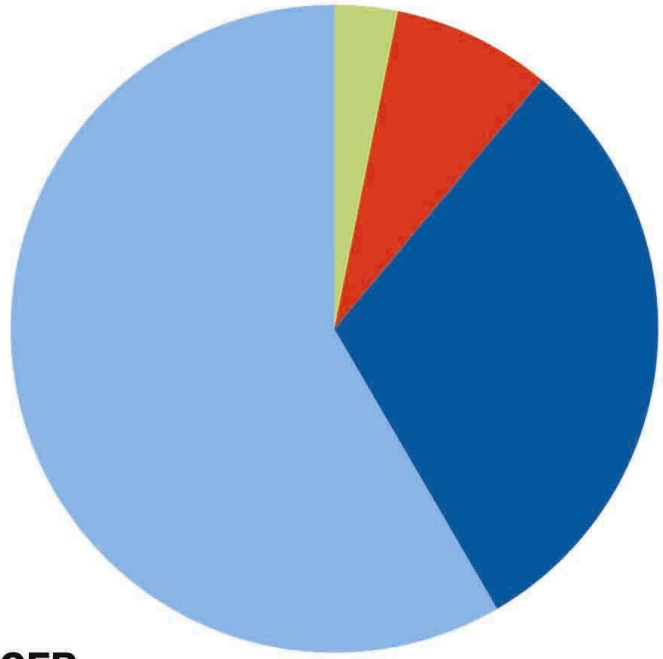
80–90% due to ventricular tachyarrhythmias (VF / VT)





Causes of SCD

More people die from SCD than AIDS, breast cancer and lung cancer combined



SCD
(300,000)

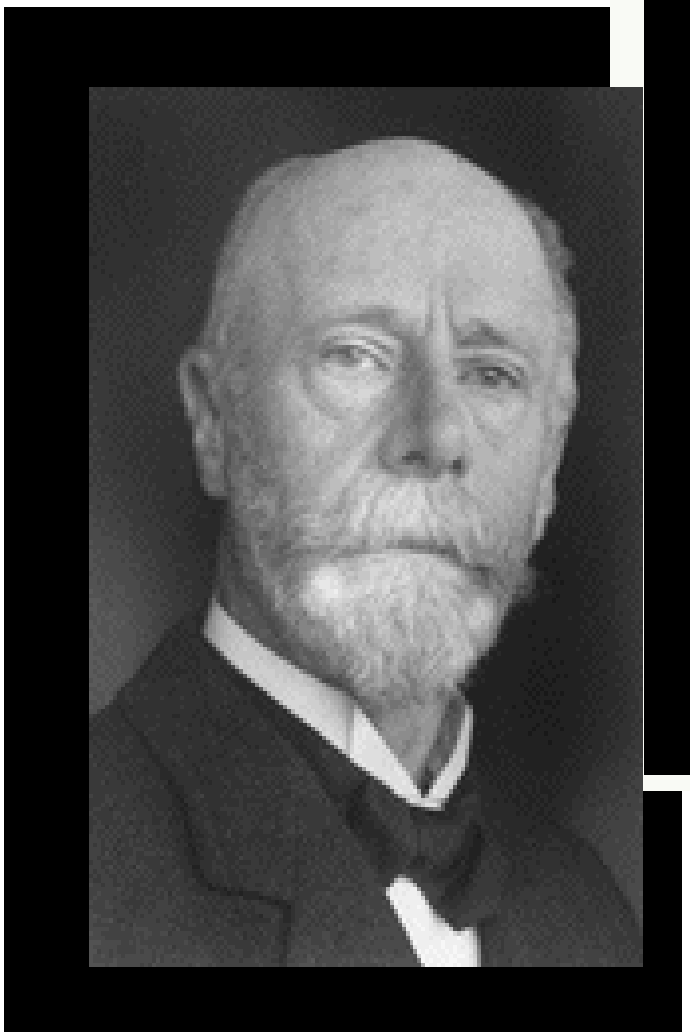
AIDS
(16,000)

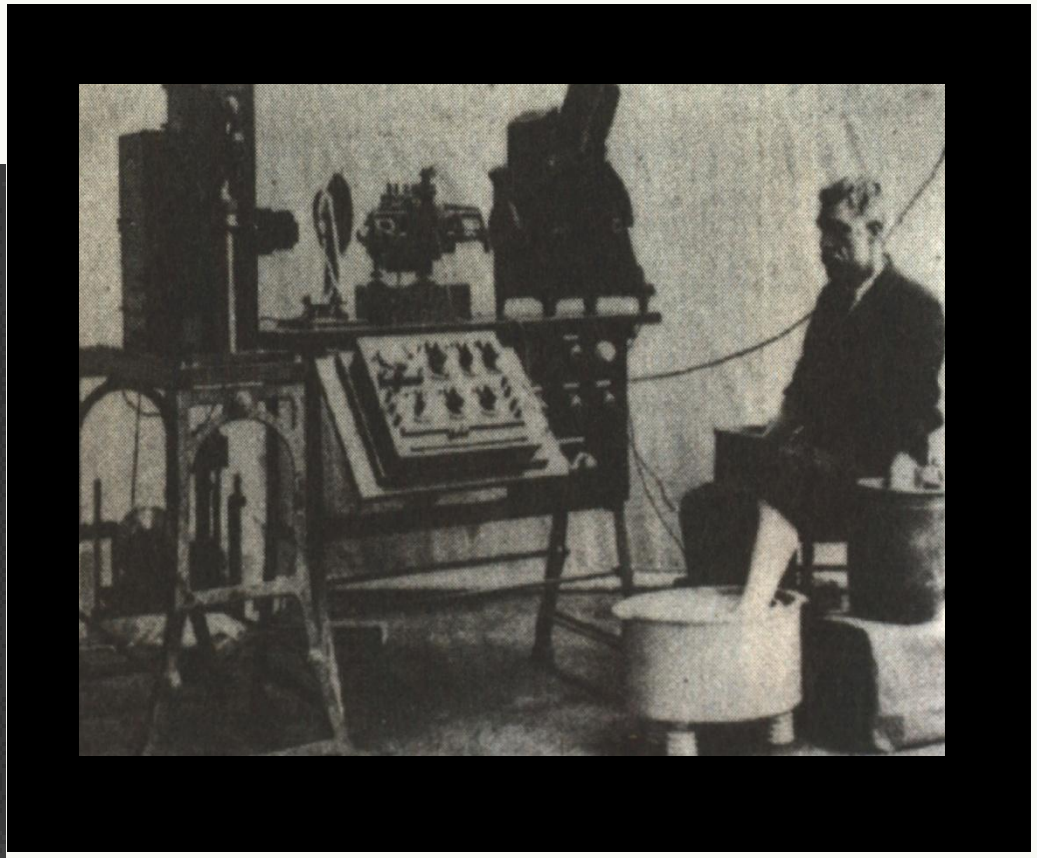
BREAST CANCER
(40,600)

LUNG CANCER
(157,400)

Sources:
NASPE 2001, CDC 2001,
American Cancer Society 2001

String galvanometer

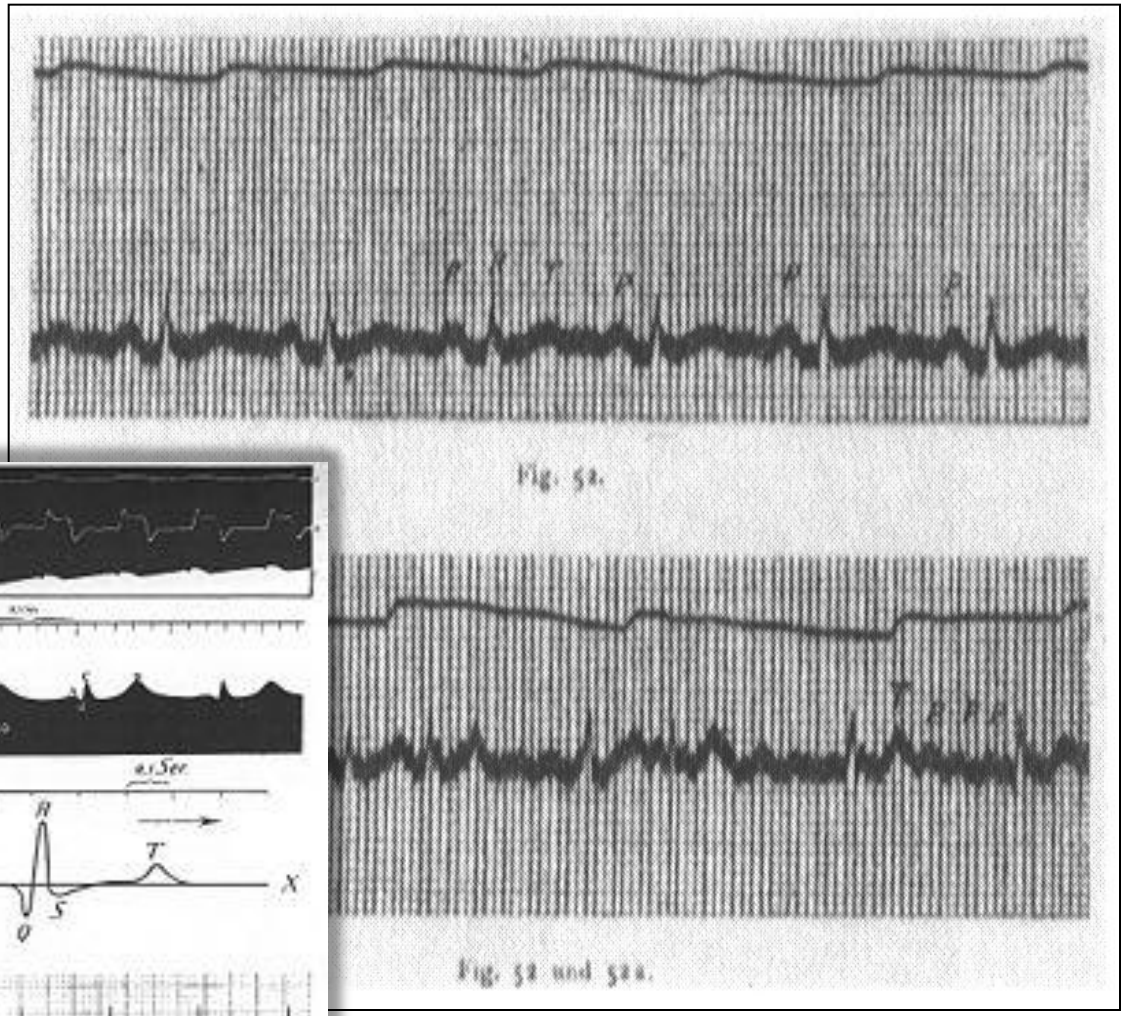




Willem Einthoven (1860-1927)

Nobel Prize Laureate in Year 1924

ECG recorded by Einthoven in Year 1914



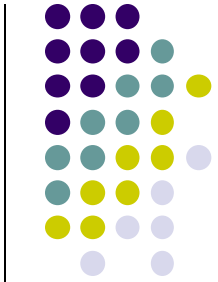
Normal sinus rhythm

Atrial fibrillation

ECG in Sudden Cardiac Death

– The Silent Witness

Different modalities of ECG recording

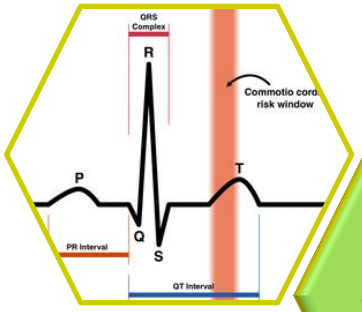


Surface ECG

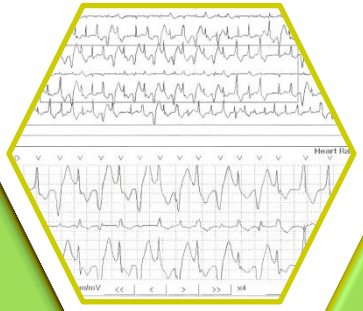
- 12-lead surface ECG
- Signal-averaged ECG
- Microvolt T-wave alternans
- Heart rate variability
- Holter monitoring
- Telemetry
- Implantable loop recorder

Intracardiac ECG

ECG in SCD – Outline



ECG during provocation & stress test



Diagnosis of fatal arrhythmias & underlying heart disease

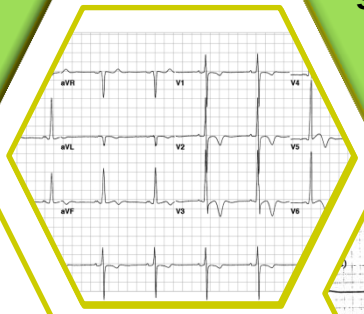
Genotype-phenotype correlation in ion channelopathy

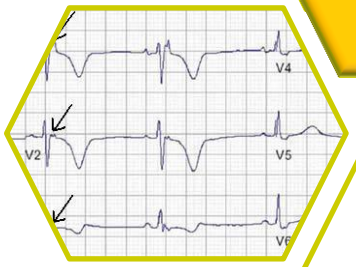
Systemic evaluation of ECG in SCD prevention

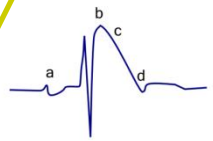
ECG modulation by drugs & environment

ECG in risk stratification

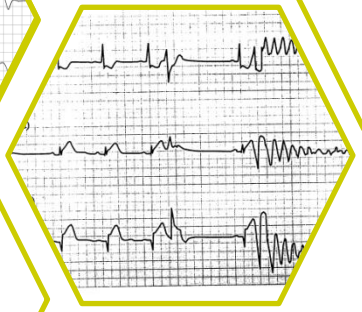
ECG in SR



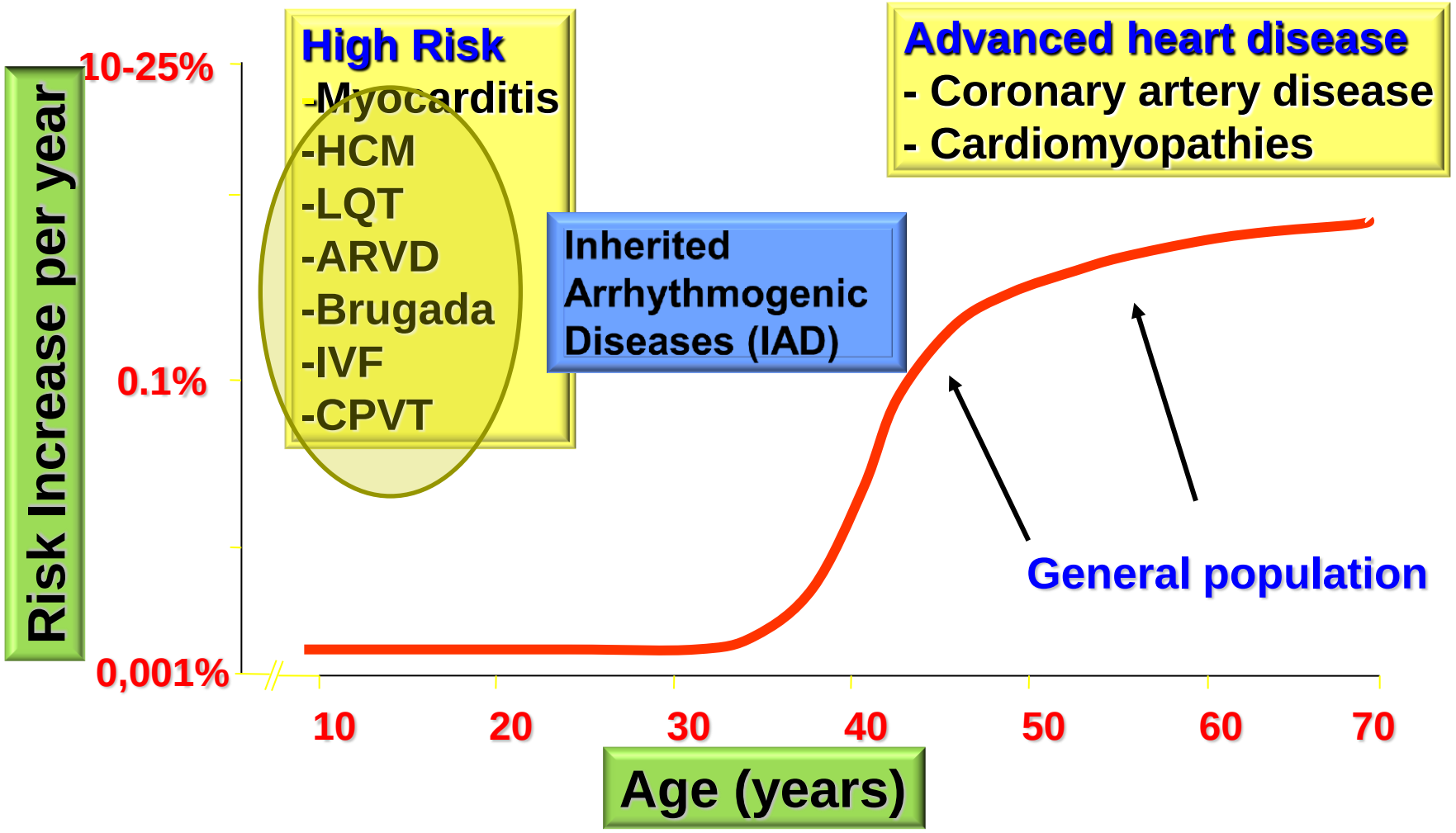




ECG characteristics in Brugada Syndrome
a. Broad P wave with some PQ prolongation
b. J point elevation
c. Coved type ST segment elevation
d. Inverted T wave



Age-specific risk and causes of Sudden Death



Hypertrophic Cardiomyopathy (HCM)

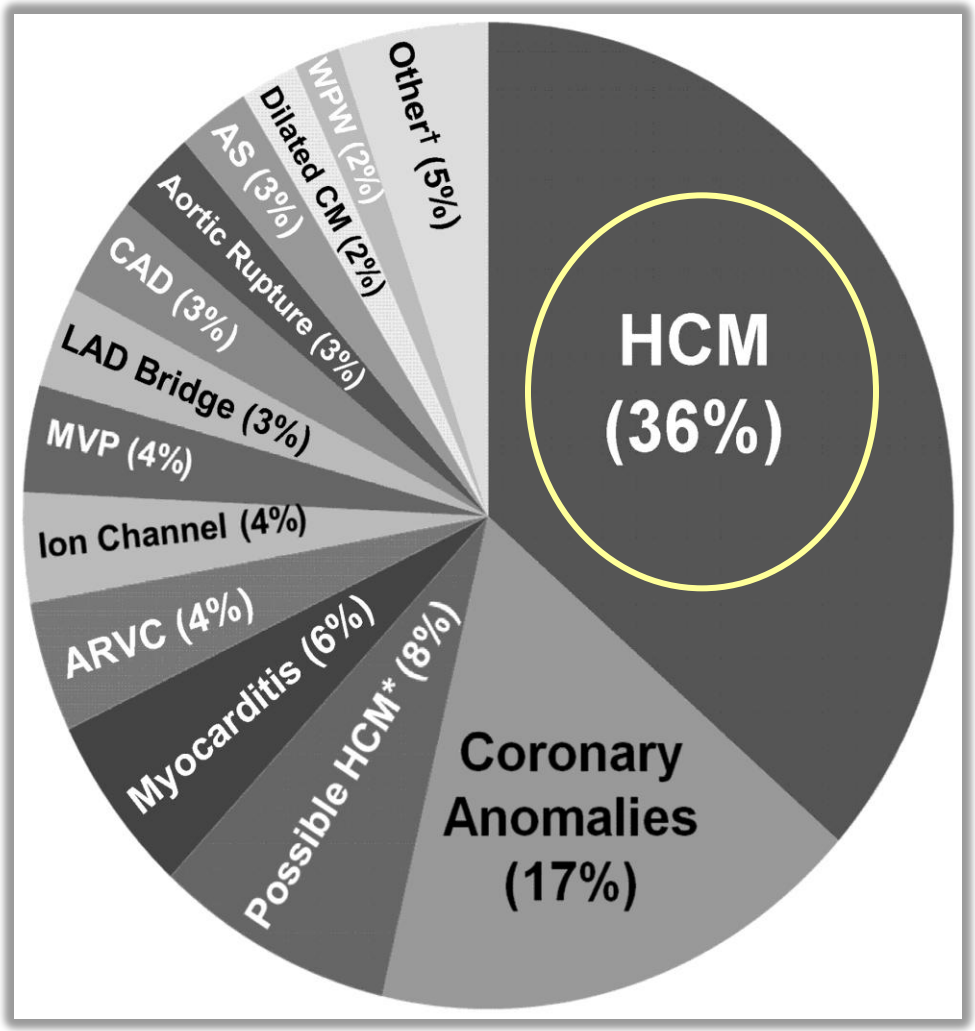
- SCD may be triggered by exercise in high-risk patients
- High-risk ECG features
  - **SMVT**
  - **NSVT on Holter**

FOE of Cameron died of SCD due to HCM

HCM - the commonest cause of SCD in competitive atheletes in a US study

N = 158

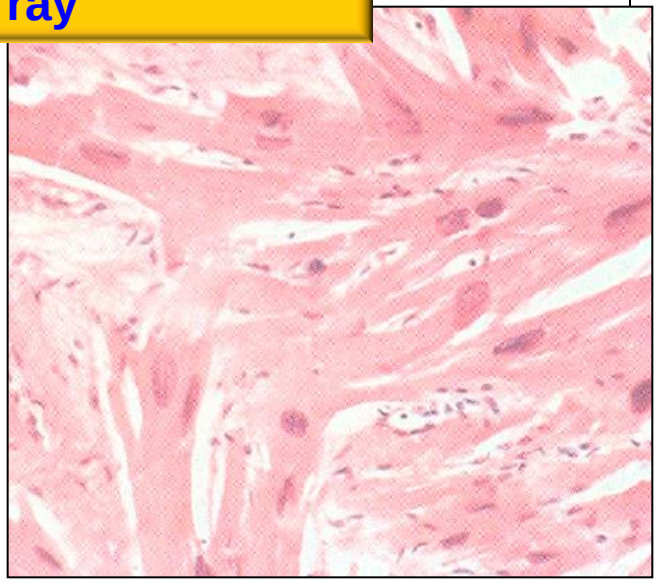
Mean age = 17

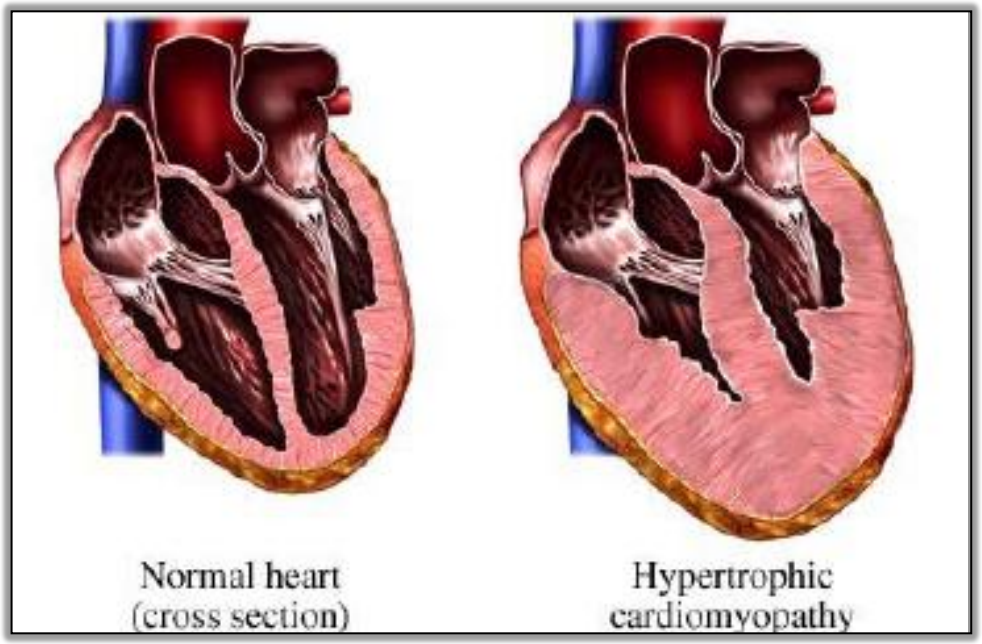


Maron BJ et al, Circulation 1996

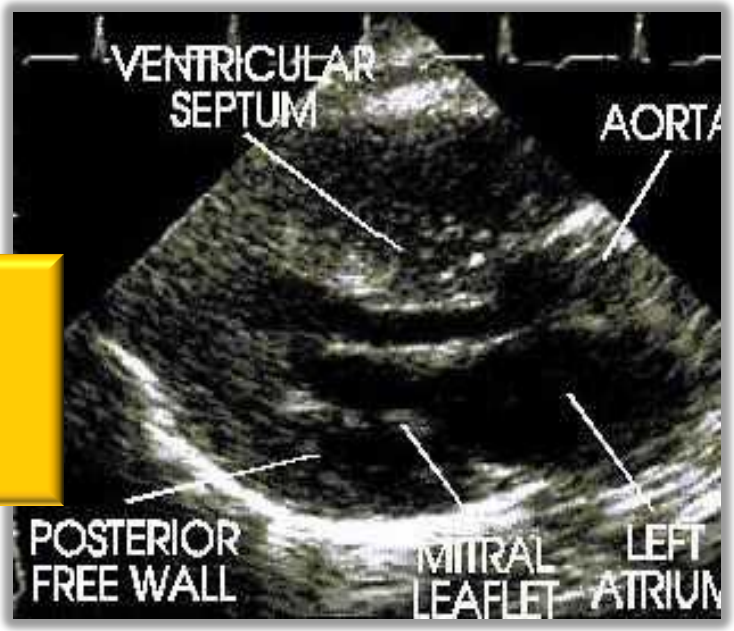
HCM

Myocardial fibre disarray

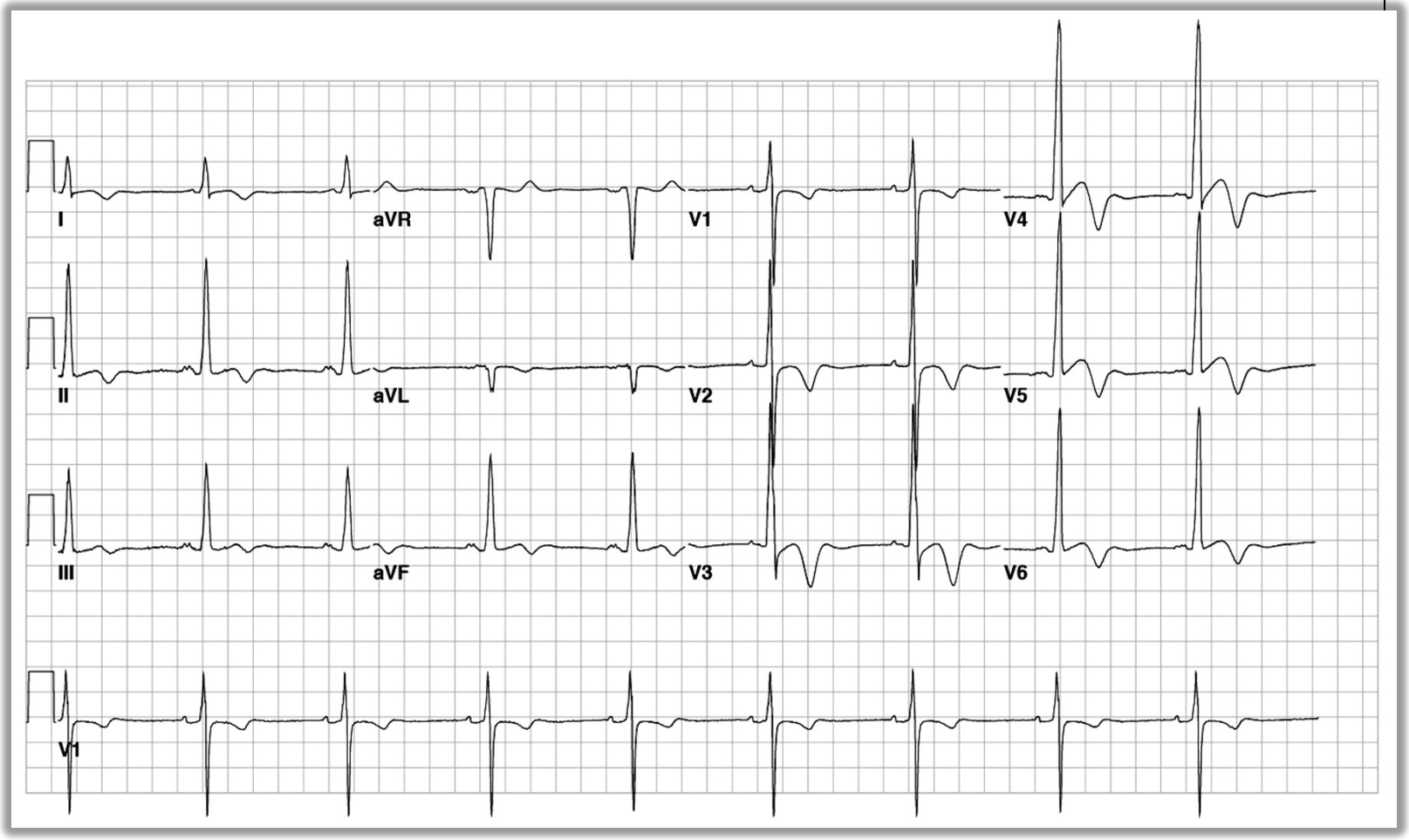




Asymmetrical septal hypertrophy (ASM)

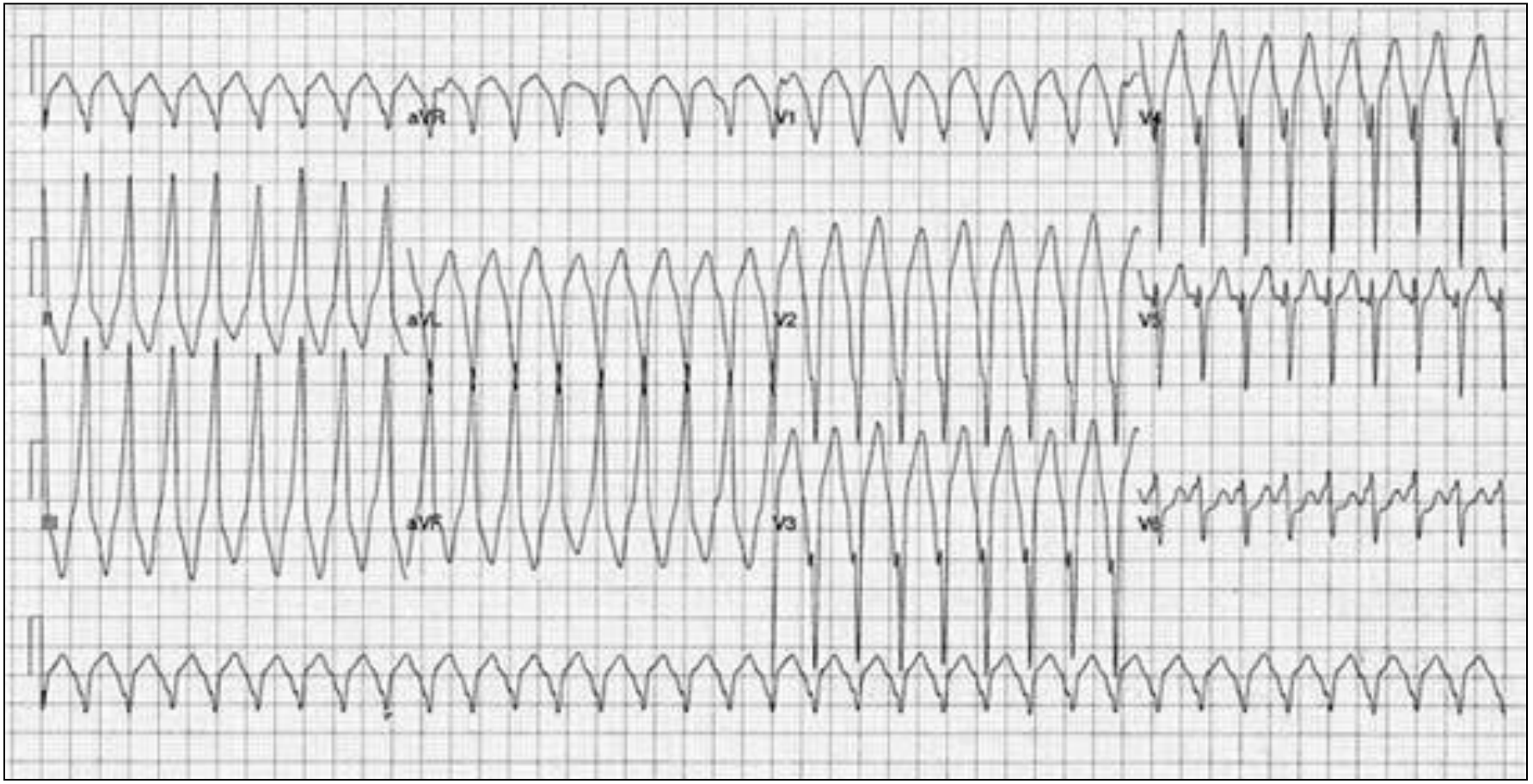


ECG features of HCM



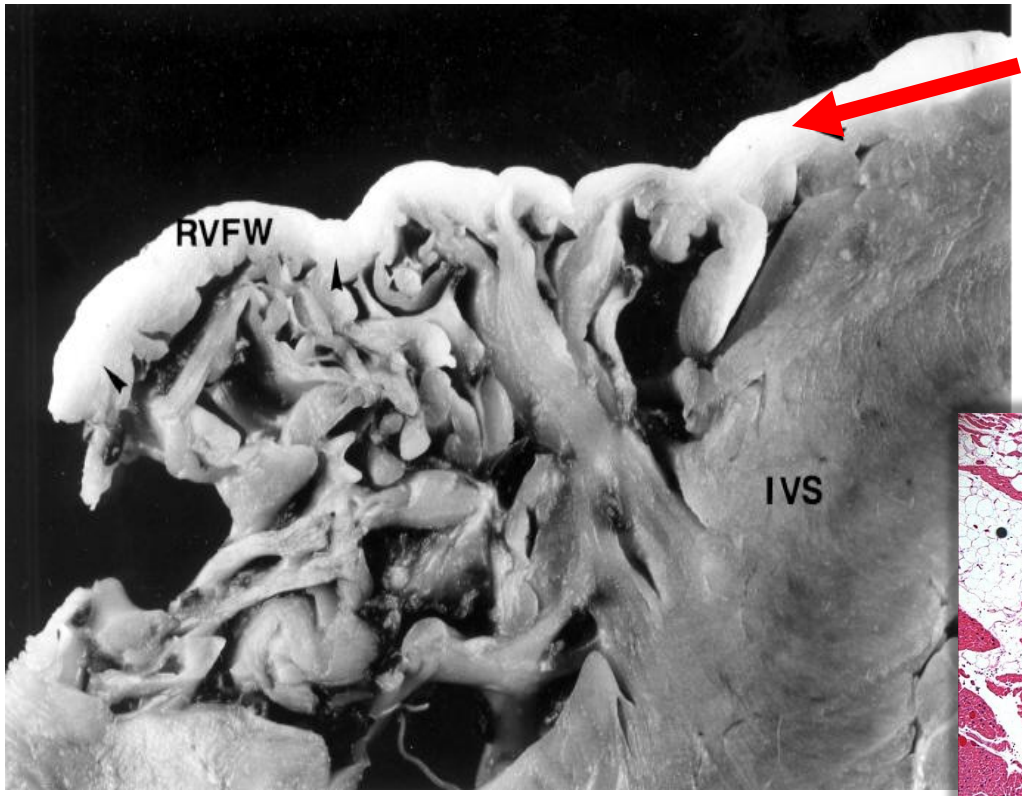
LVH with strain pattern

HCM presenting with SMVT

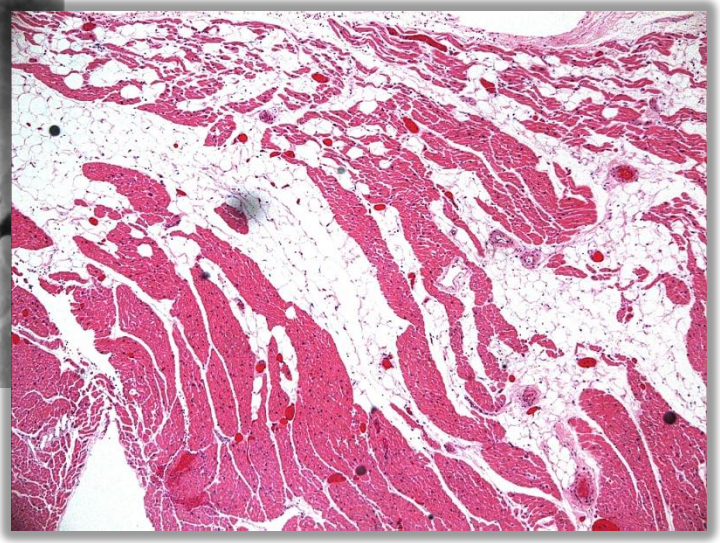


Arrhythmogenic RV Cardiomyopathy (ARVC)

2 cases of arrhythmogenic right ventricular dysplasia presenting with sudden cardiac arrest *Mok NS et al, JHKCC 1997*



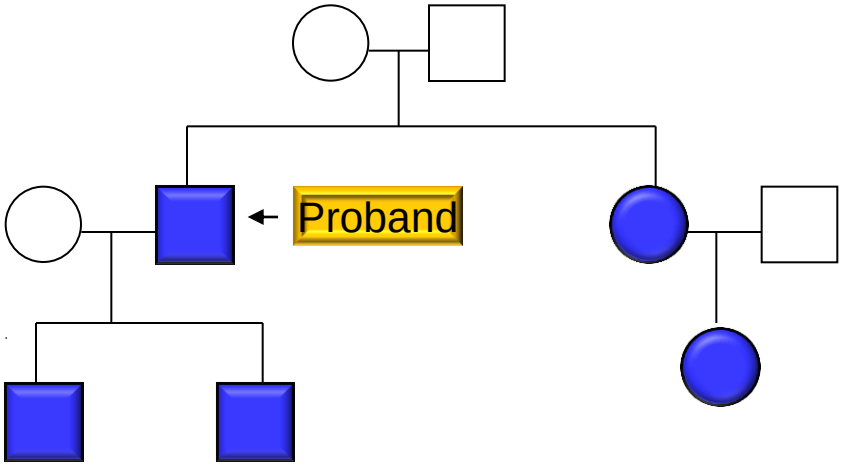
- Fibro-fatty replacement of RV myocardium
- VT/VF/ R heart failure
- Commonest cause of SCD in young athletes in Northern Italy

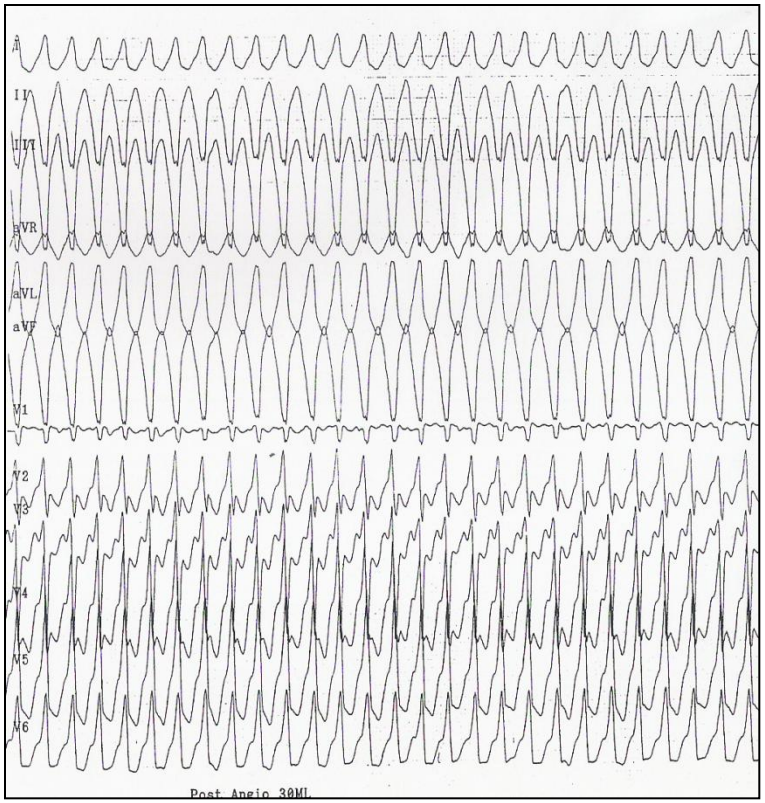


Familial form of arrhythmogenic right ventricular dysplasia presenting with recurrent ventricular tachycardia

Mok NS et al, HKMJ 1999

- **M/50, presenting with VT & syncope**
- **Confirmed ARVC**
- **Mutation in *PKP2* gene in proband and 4 family members**



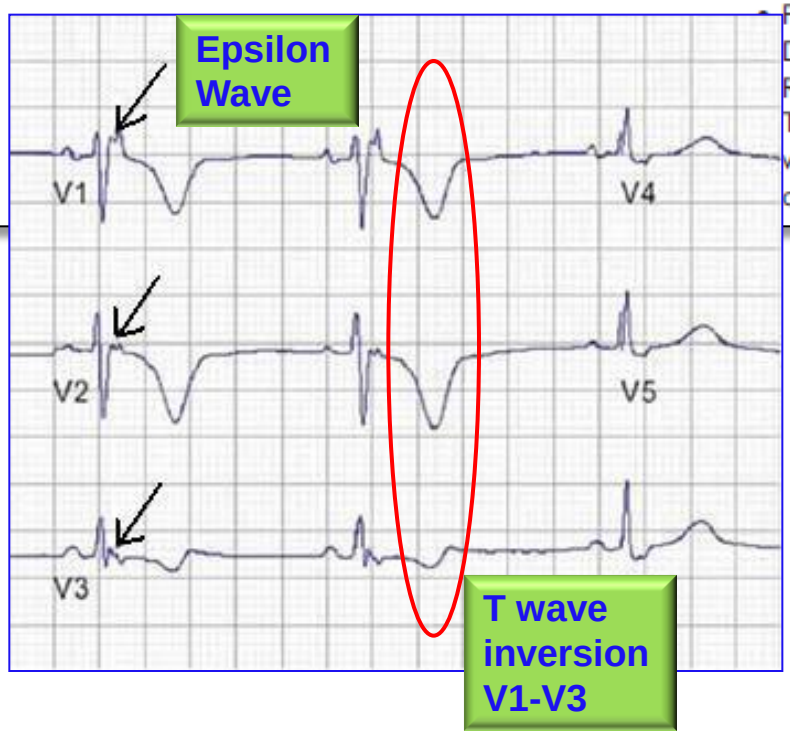


: ***PKP2* gene mutation +ve**

SMVT with LBBB morpholgy

ECG Features of ARVC

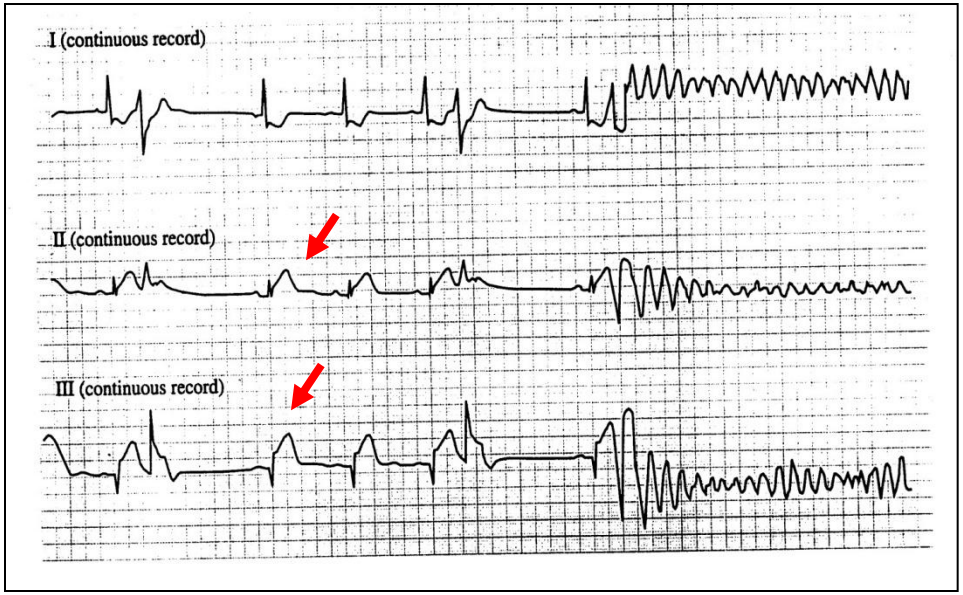
Original task force criteria	Revised task force criteria
IV. Depolarization/conduction abnormalities	
Major	
• Epsilon waves or localized prolongation (>110 ms) of the QRS complex in right precordial leads (V_1 to V_3)	• Epsilon wave (reproducible low-amplitude signals between end of QRS complex to onset of the T wave) in the right precordial leads (V_1 to V_3)
Minor	
• Late potentials (SAECG)	• Late potentials by SAECG in ≥1 of 3 parameters in the absence of a QRS duration of ≥110 ms on the standard ECG • Filtered QRS duration (fQRS) ≥114 ms Duration of terminal QRS <40 μV (low-amplitude signal duration) ≥38 ms Root-mean-square voltage of terminal 40 ms ≤20 μV Terminal activation duration of QRS ≥55 ms measured from the nadir of the S wave to the end of the QRS, including R′, in V_1, V_2, or V_3, in the absence of complete right bundle-branch block



Coronary Artery Disease (CAD)

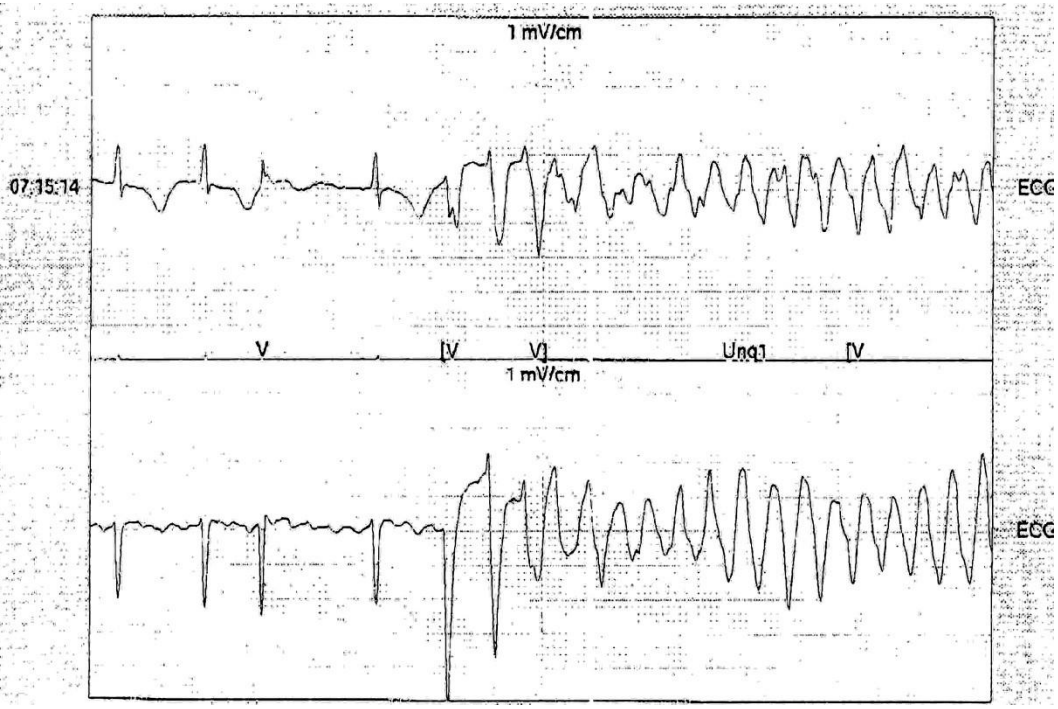
Died of SCD due to AMI

- AMI associated with 15% risk of VF within first 24-48 hours



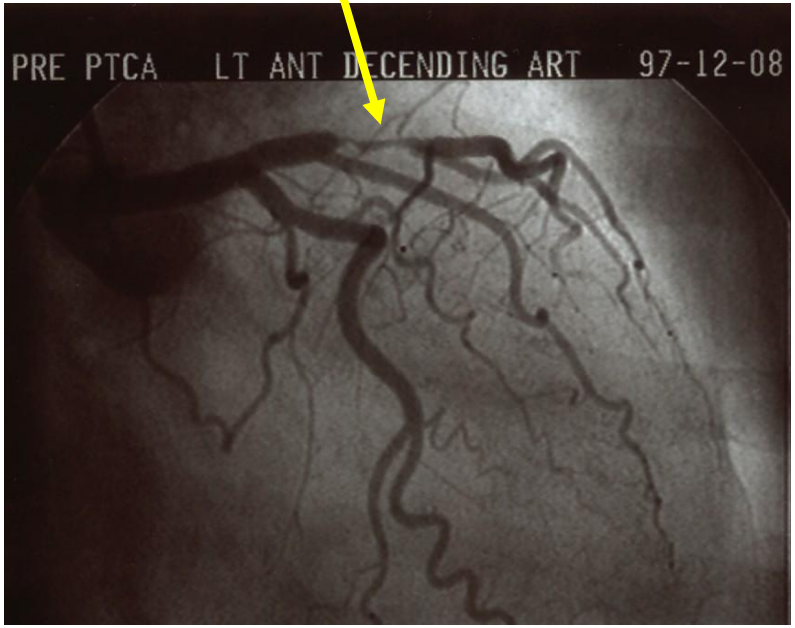
Acute inferior MI presenting with SCD

Polymorphic VT in NSTEMI



Severe CAD leading to polymorphic VT

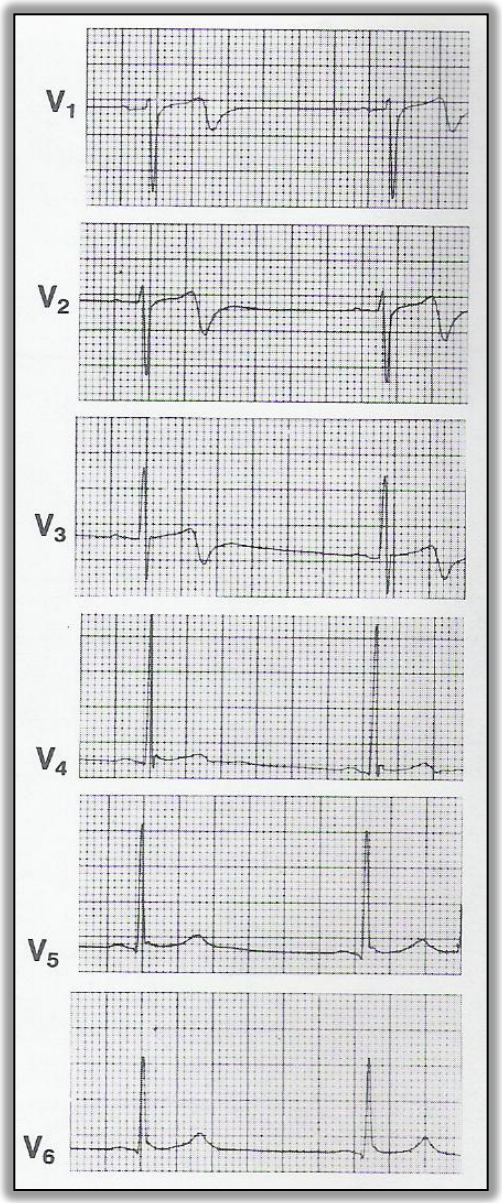
Critical stenosis in LAD artery

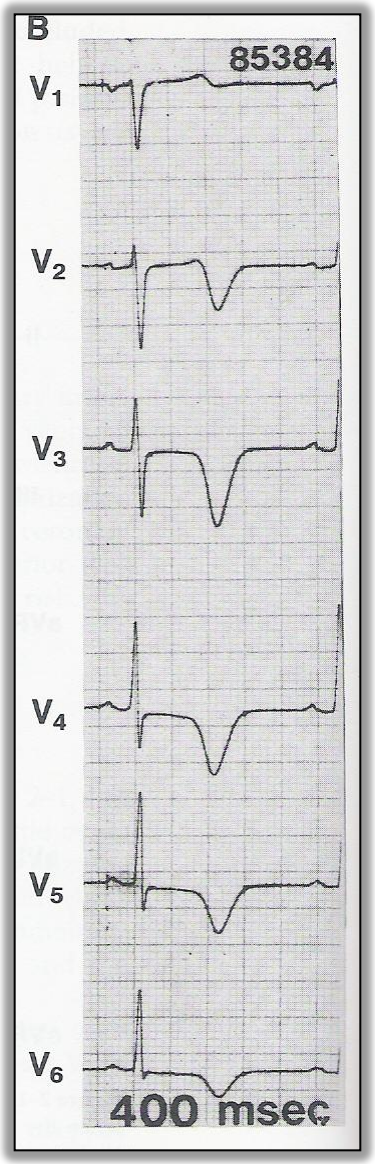


ECG features of critical pLAD stenosis

Wellen's Sign

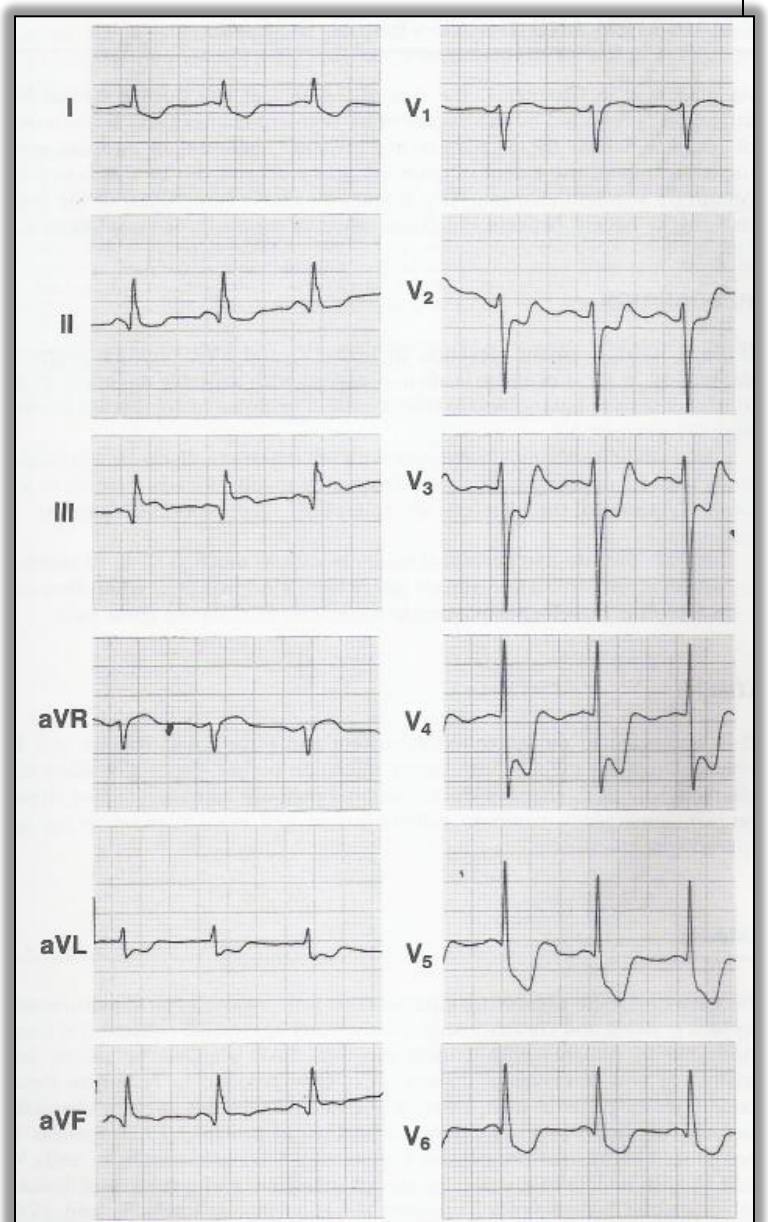
- **Symmetric, deep T Wave inversion or biphasic T waves in V2-V5 or V6 during pain free periods**
- **Minimal (<1mm), if any, ST elevations**



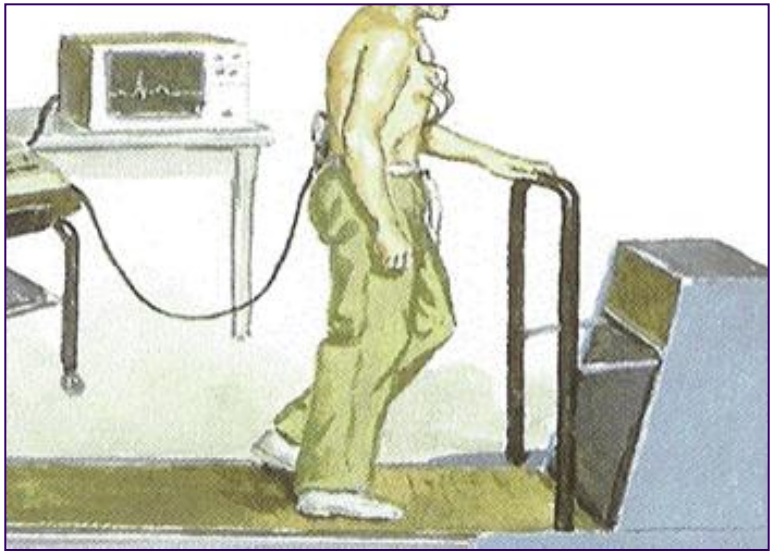


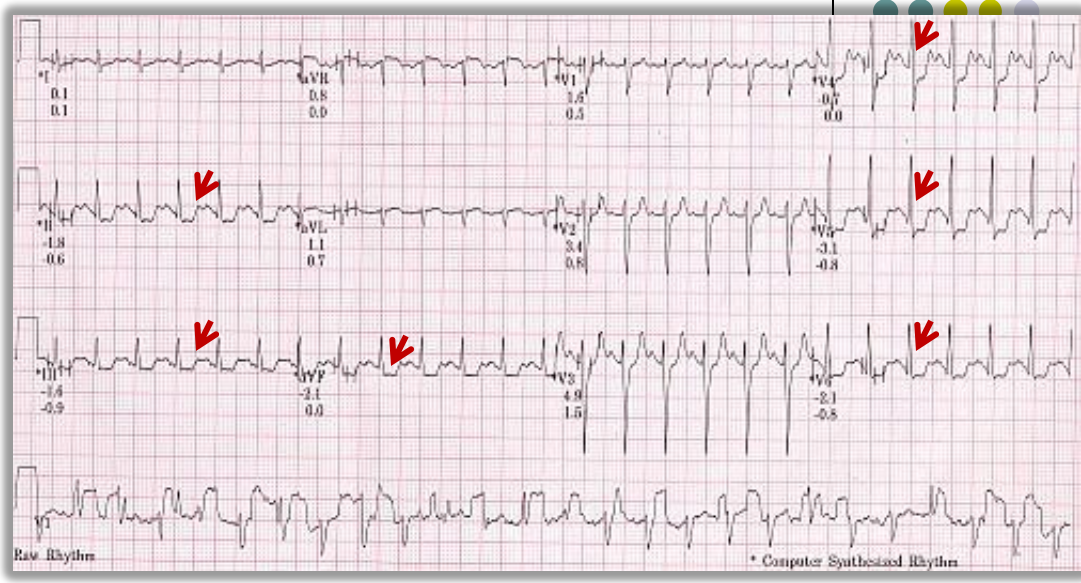
ECG features of L main or 3VD

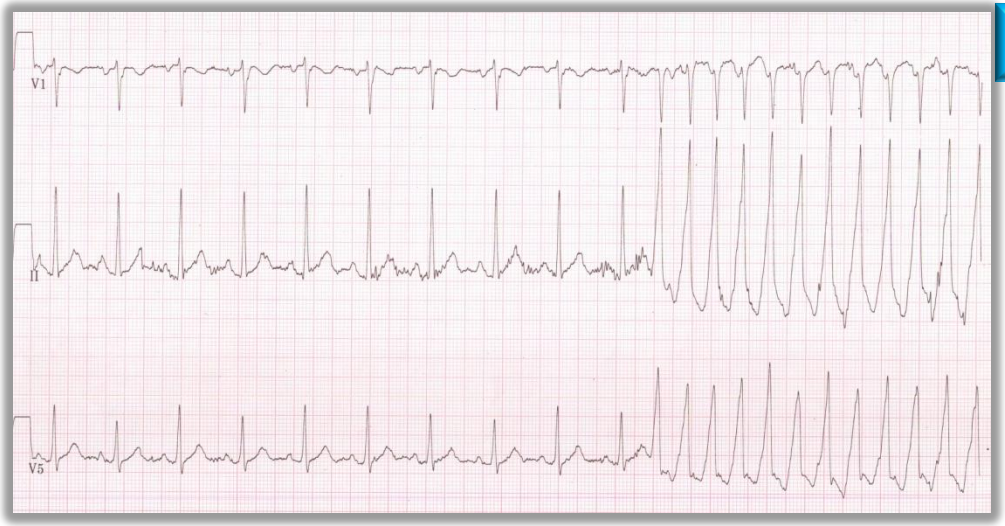
- ECG sign during chest pain
- ST elevation in V1 & aVR
- ST depression in ≥ 8 leads



Exercise stress test







Exercise-induced ST depression

VT induced during exercise test in severe CAD

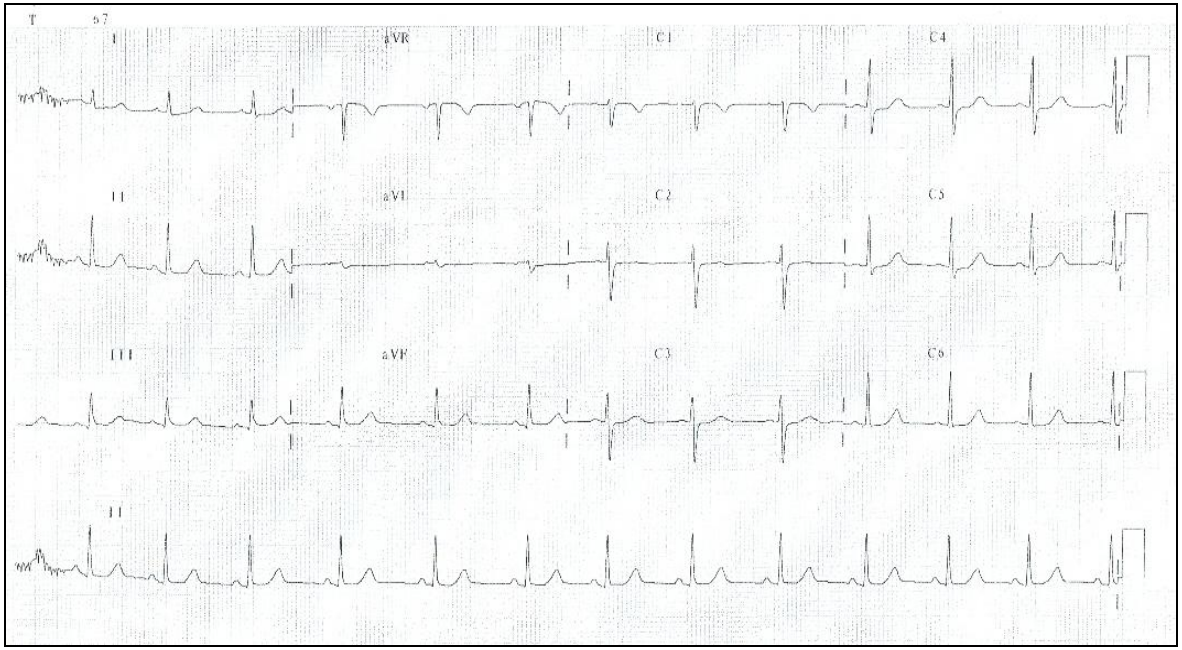
Congenital CAD at risk of SCD

- **Left main atresia**
- **Anomalous origin of coronary arteries with a malignant course**
  - **LCA arising from pulmonary artery**
  - **LCA arising from R Sinus of Valsalva**
  - **RCA arising from L Sinus of Valsalva**
  - **Coronary artery course between aorta & pulmonary trunk**

Congenital left main atresia

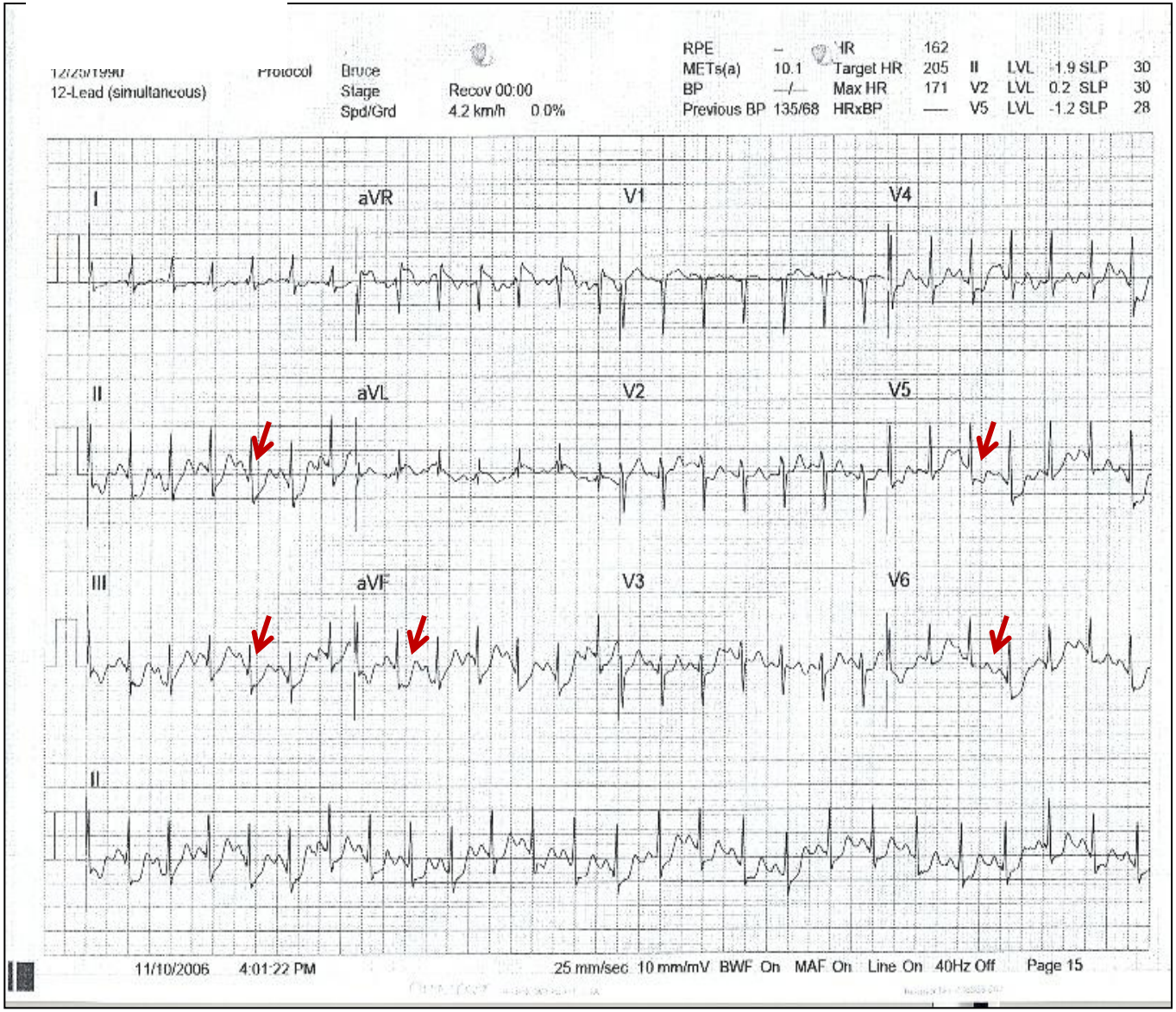
History

- F/15, Chinese, Form 3 student
- Good past health
- August 2006
  - Chest pain followed by syncope after playing badminton for 1 hour



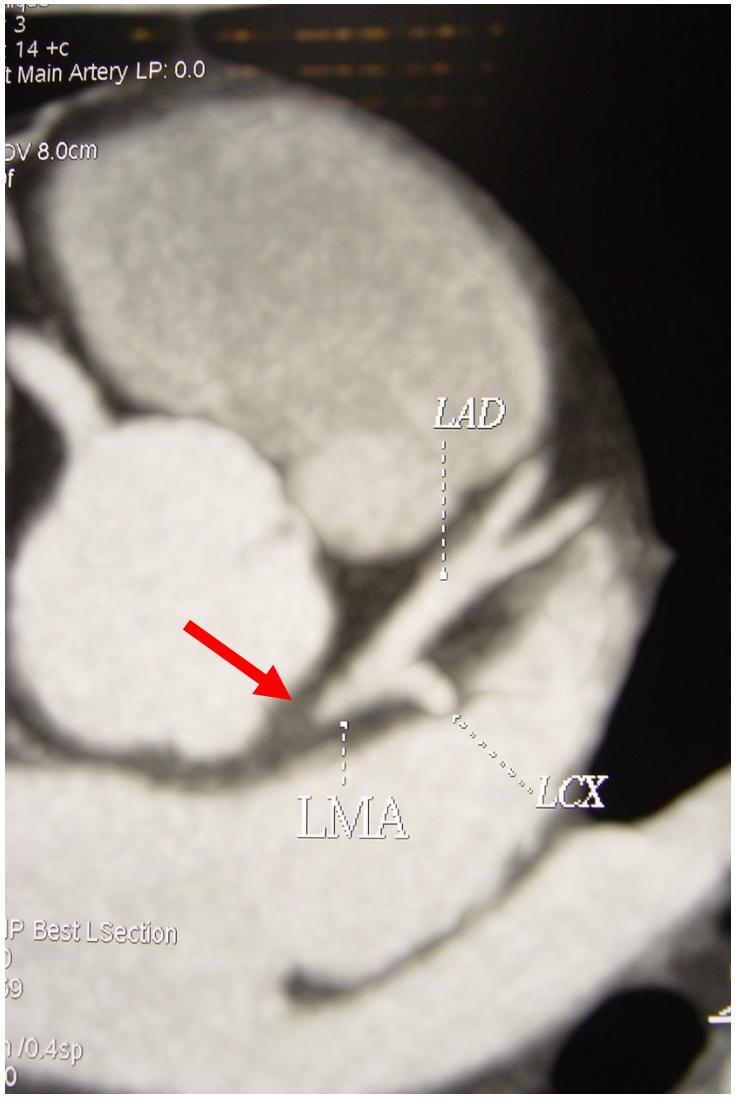
12-lead ECG at baseline

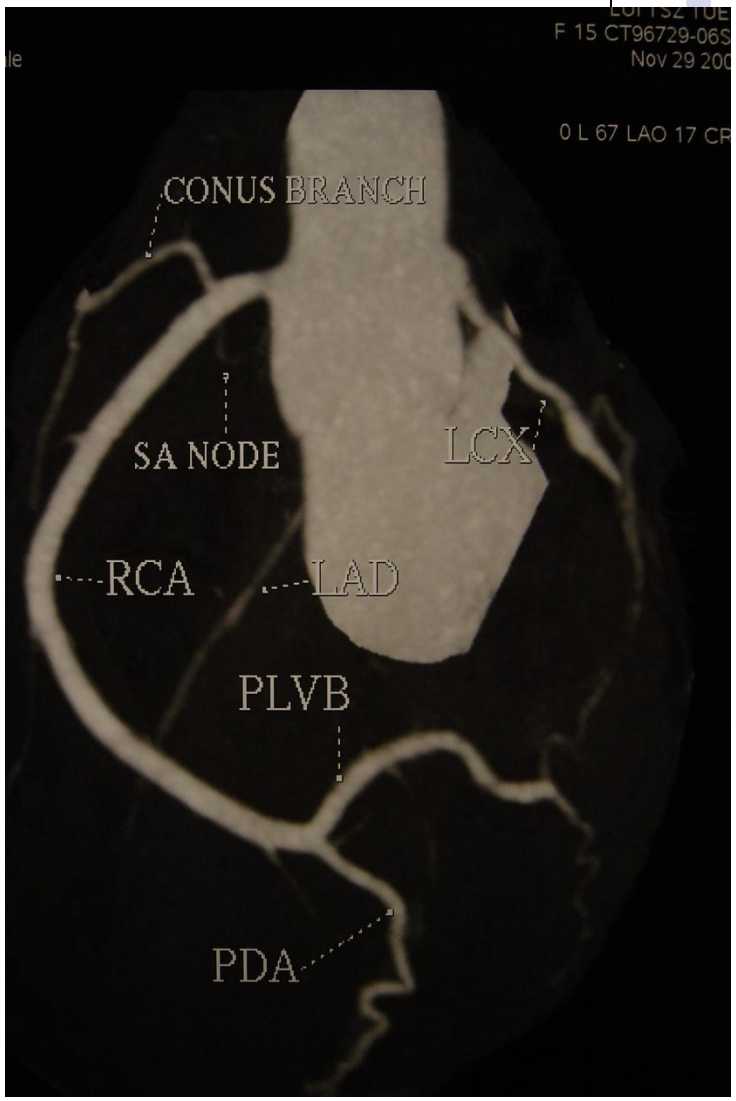
Diffuse ST depression during Treadmill Exercise Test



CT coronary angiograms

L main atresia with collaterals from RCA

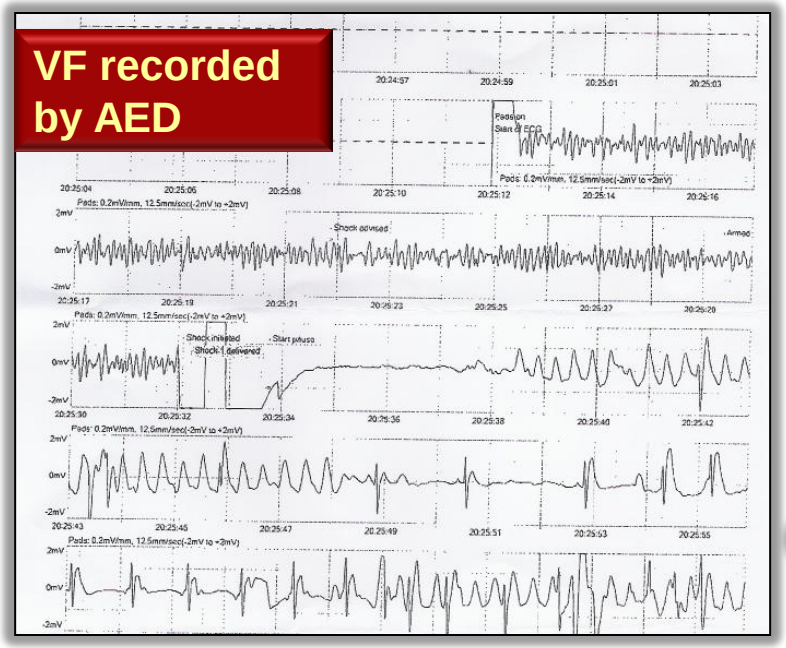




Anomalous RCA with a malignant course

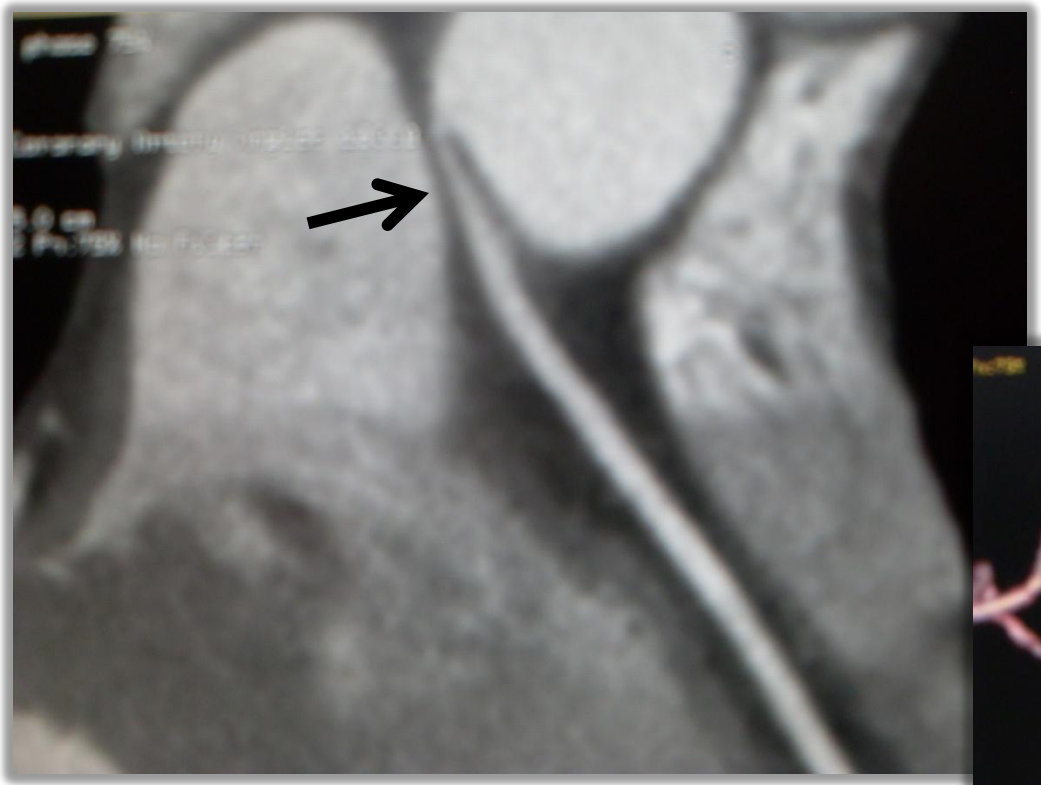
History

- M/36,policeman, good PH
- Sudden collapse with LOC after chest wall hit by a soccer kicked from a close range



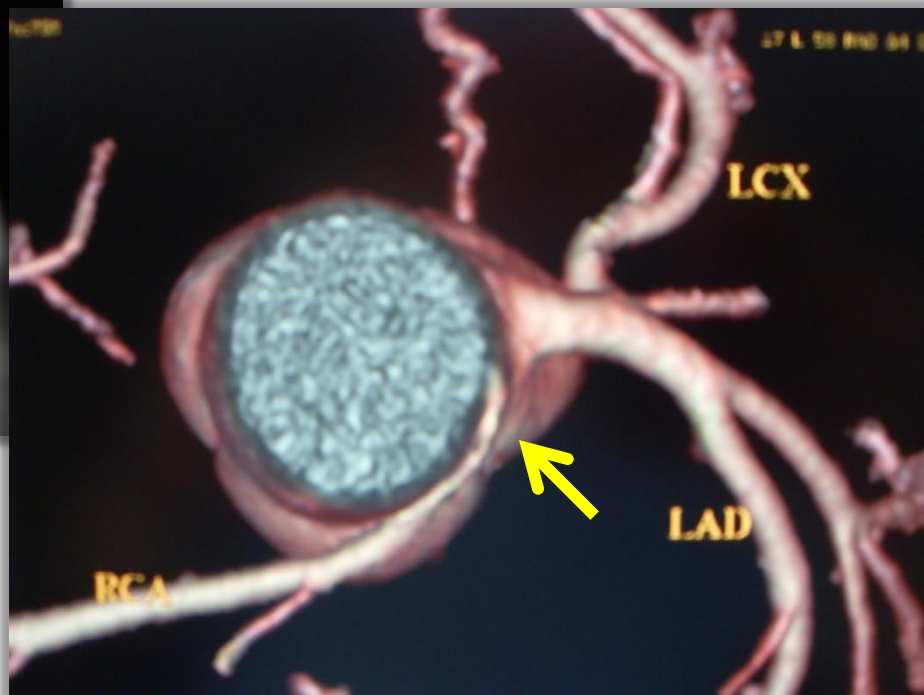
Commotio Cordis

Anomalous RCA with a malignant course in a survivor of Commotio Cordis



pRCA compressed between aorta & RVOT

RCA arising from L sinus of Valsalva



Pathophysiology of Commotio Cordis

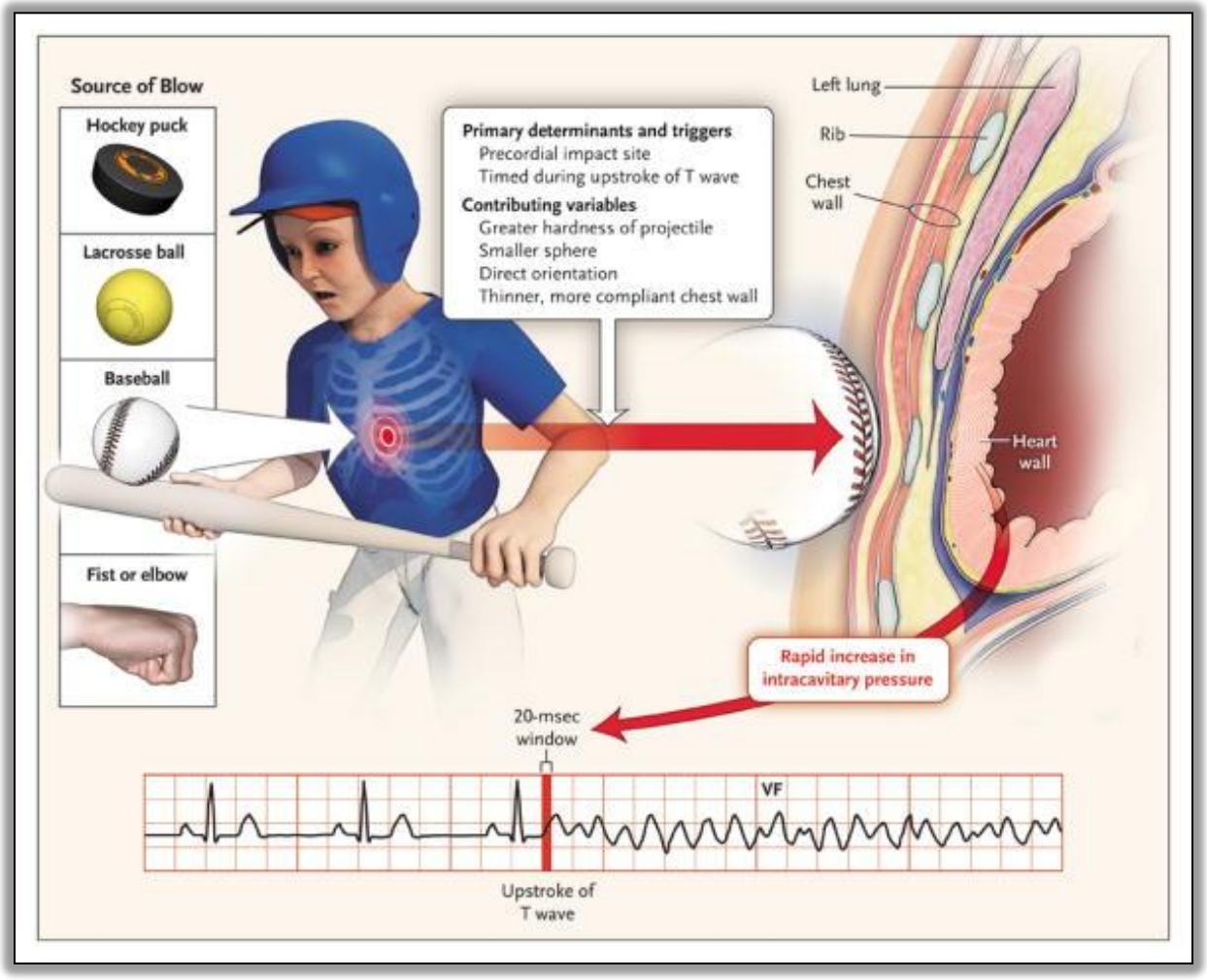
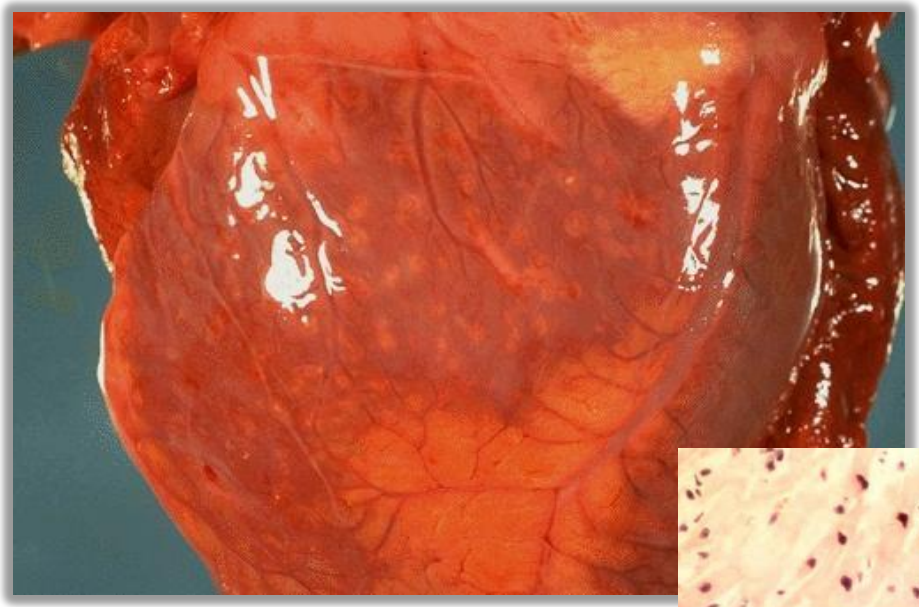


Figure 2 Pathophysiology of Commotio Cordis. In recreational and competitive sports, chest blows may involve balls or pucks or may be inflicted through bodily contact. The location of th blow on the chest and its timing relative to the cardiac cycle are the primary determinants of commotio cordis. Other factors that may contribute to the risk of an event include the density, size, and orientation of the projectile and the shape of the thorax; younger people are the most vulnerable because of their thinner, less developed rib cage and musculature.

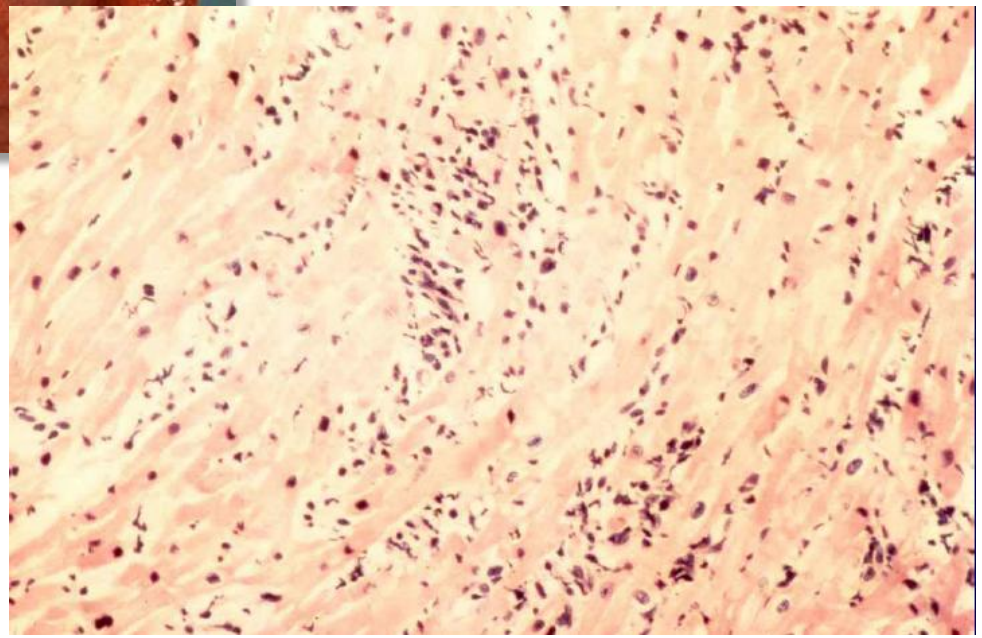
Commotio Cordis.
Maron, Barry; Estes, Mark

New England Journal of Medicine. 362(10):917-927, March 11, 2010.

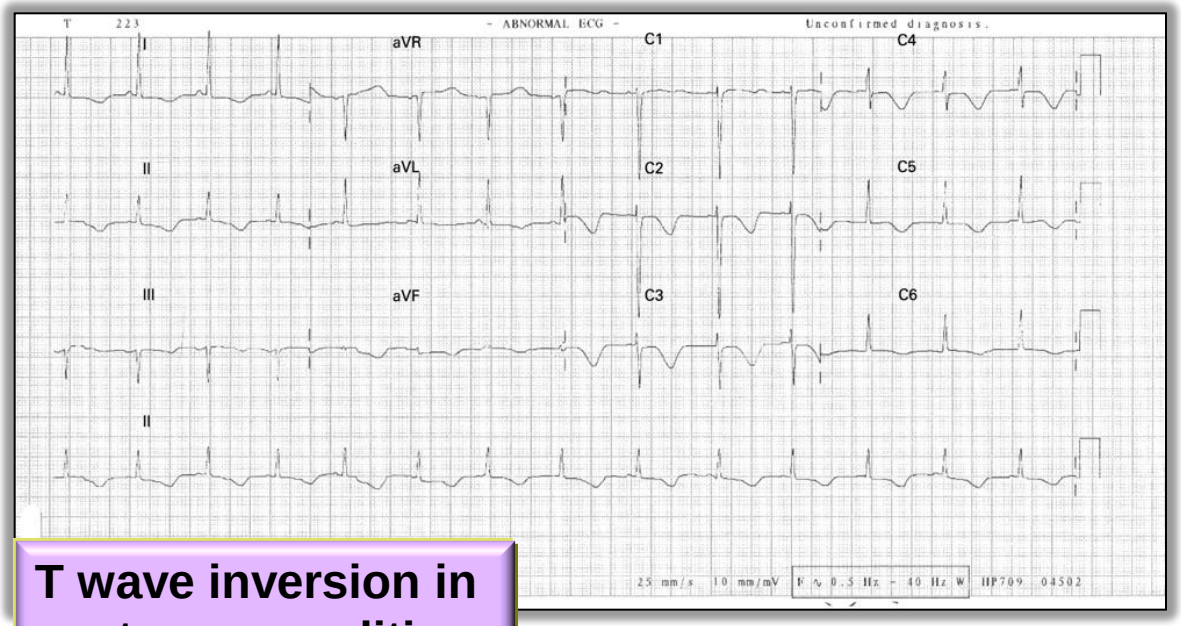
Myocarditis



- Viral / Bacterial / Fungal / Toxin
- Most cases mild
- May present with acute HF or arrhythmia causing SCD



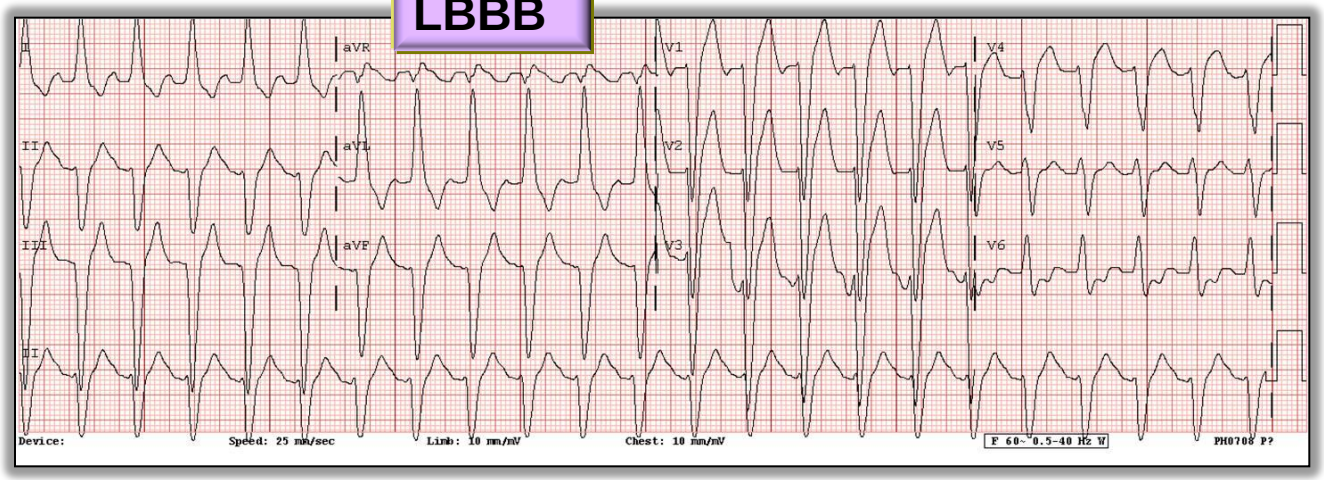
ECG features of myocarditis



T wave inversion in acute myocarditis

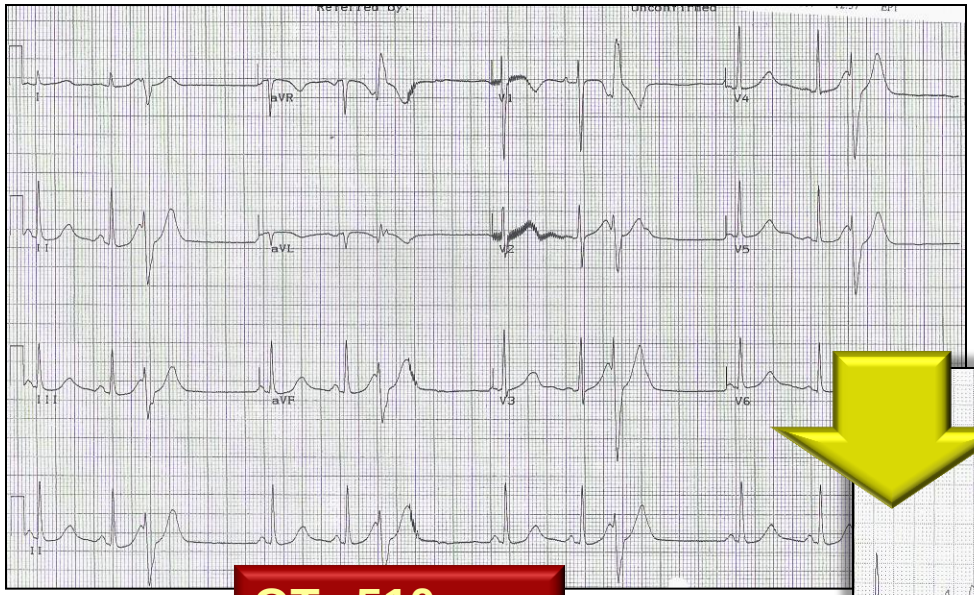
- T wave inversion
- Q wave
- ST segment elevation
- Heart block (AV block or LBBB)
- Prolonged QT interval
- Ventricular arrhythmia

LBBB

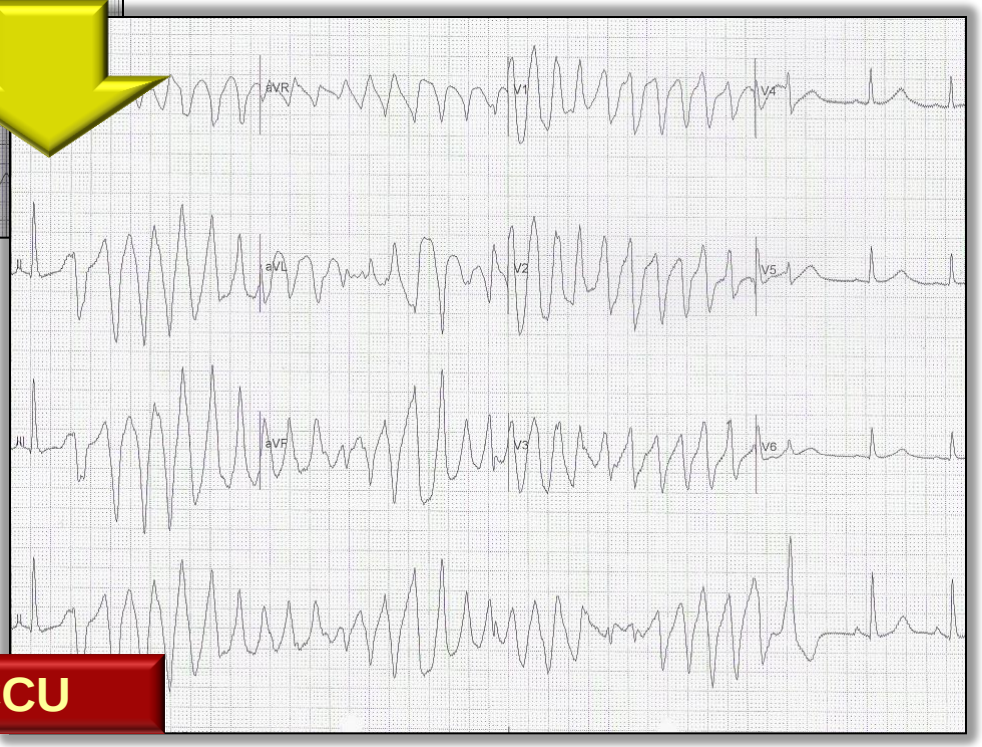


A Chinese lady presenting with long QTc & TdP

- F/ 35, presented with syncope
- QTc prolonged with PVCs
- Torsades de Pointes (TdP) in CCU
- Viral study –
  - Coxsackie B myocarditis



QTc 510ms



TdP in CCU

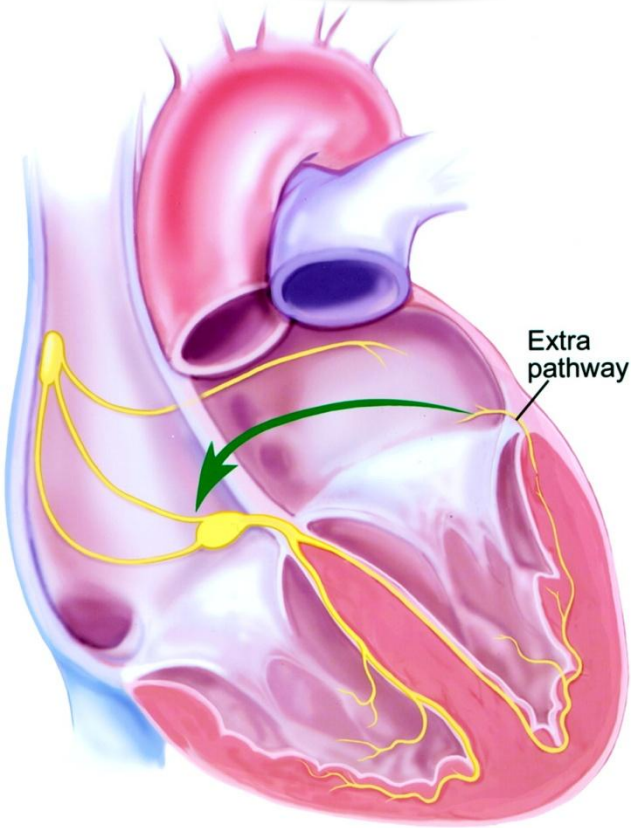
Louis **WOLFF**
John **PARKINSON**
Paul **WHITE**

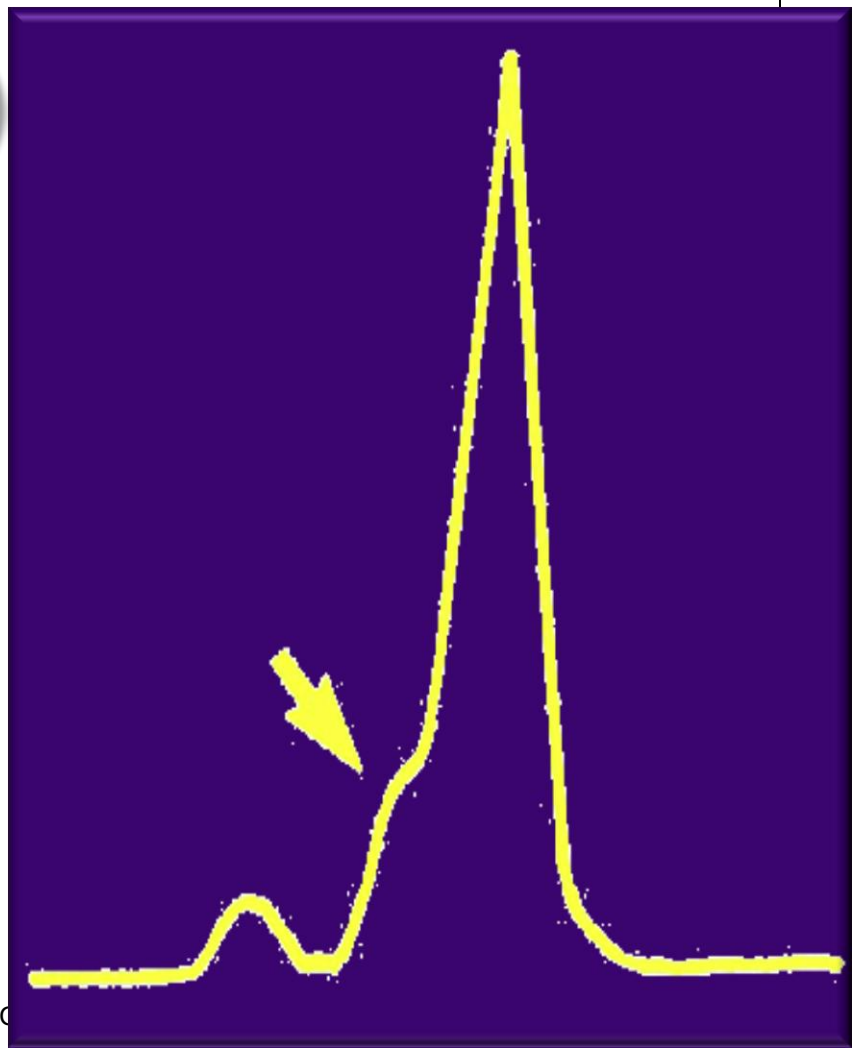
The WPW Syndrome

NS Mok

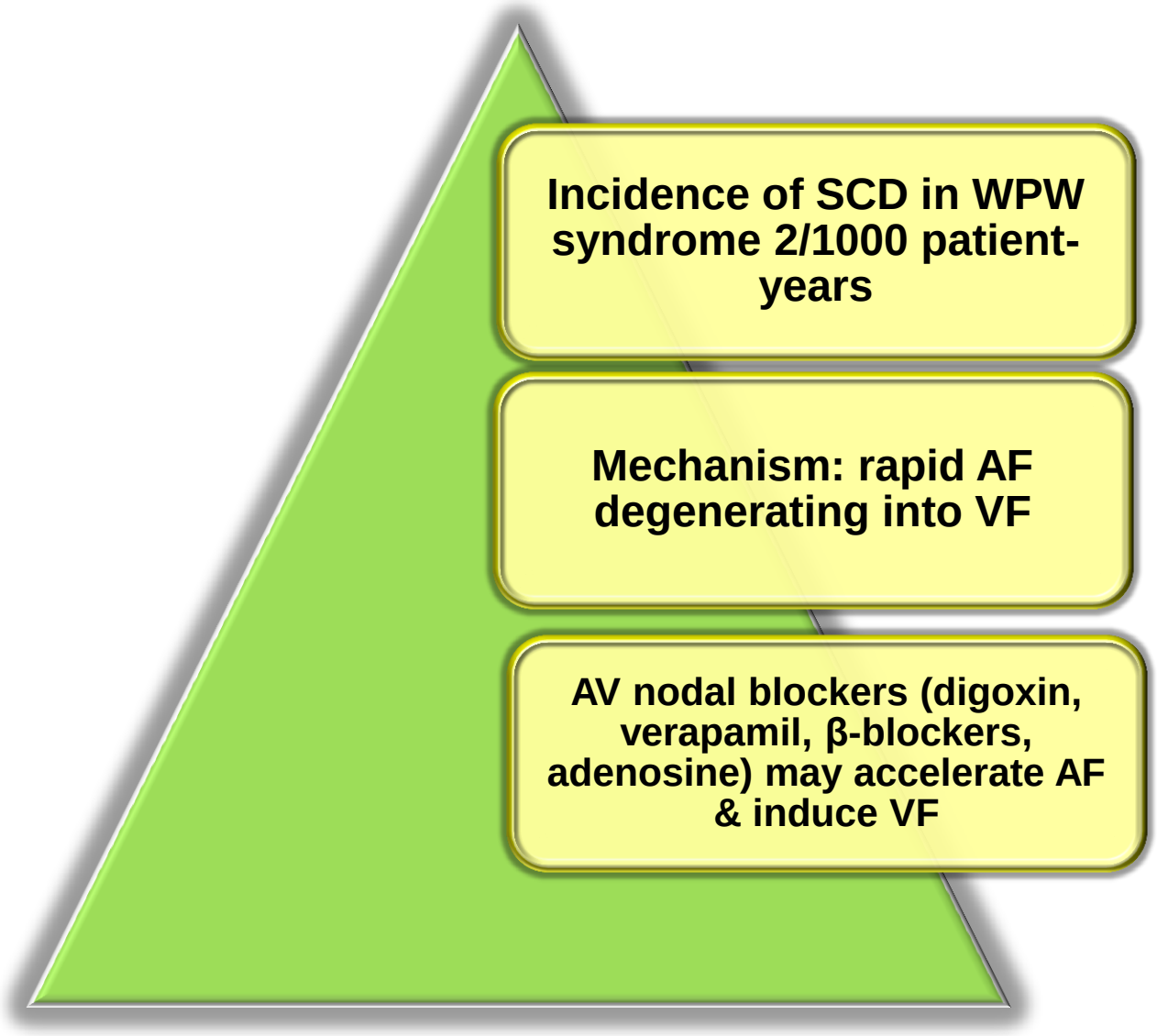
WPW Syndrome

- Short PR Interval
- Wide QRS
- Delta Wave (arrow)





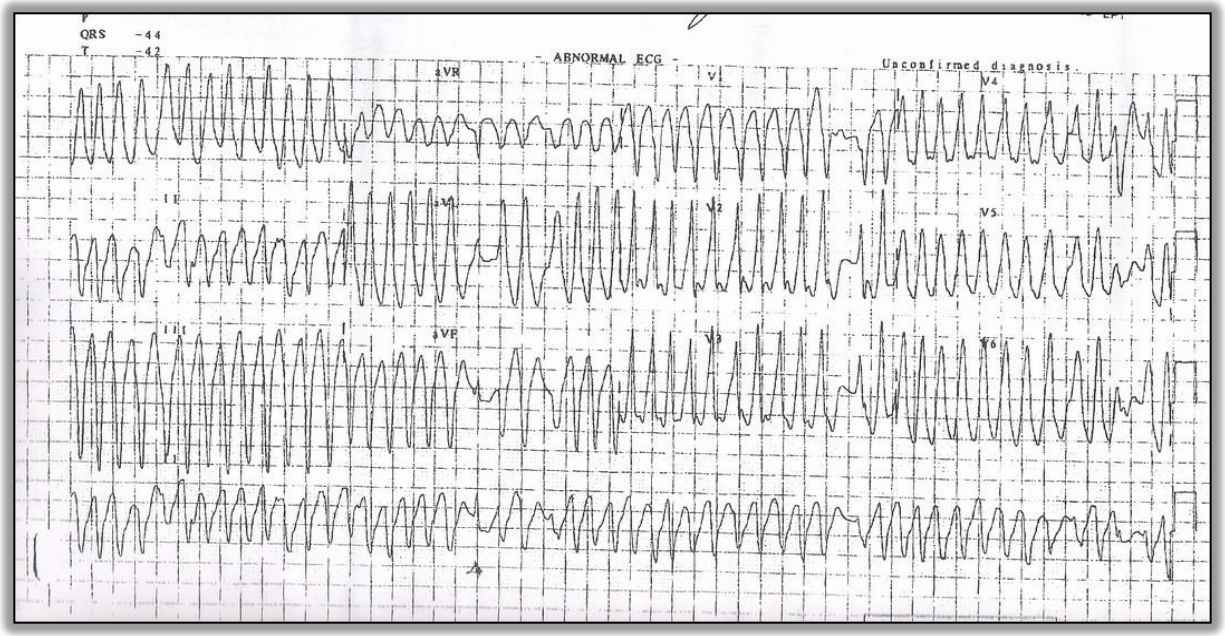
AF in WPW – Risk of SCD



Preexcited AF in WPW syndrome

F : Fast **B**: Broad **I**: Irregular

Preexcited AF with rapid ventricular response

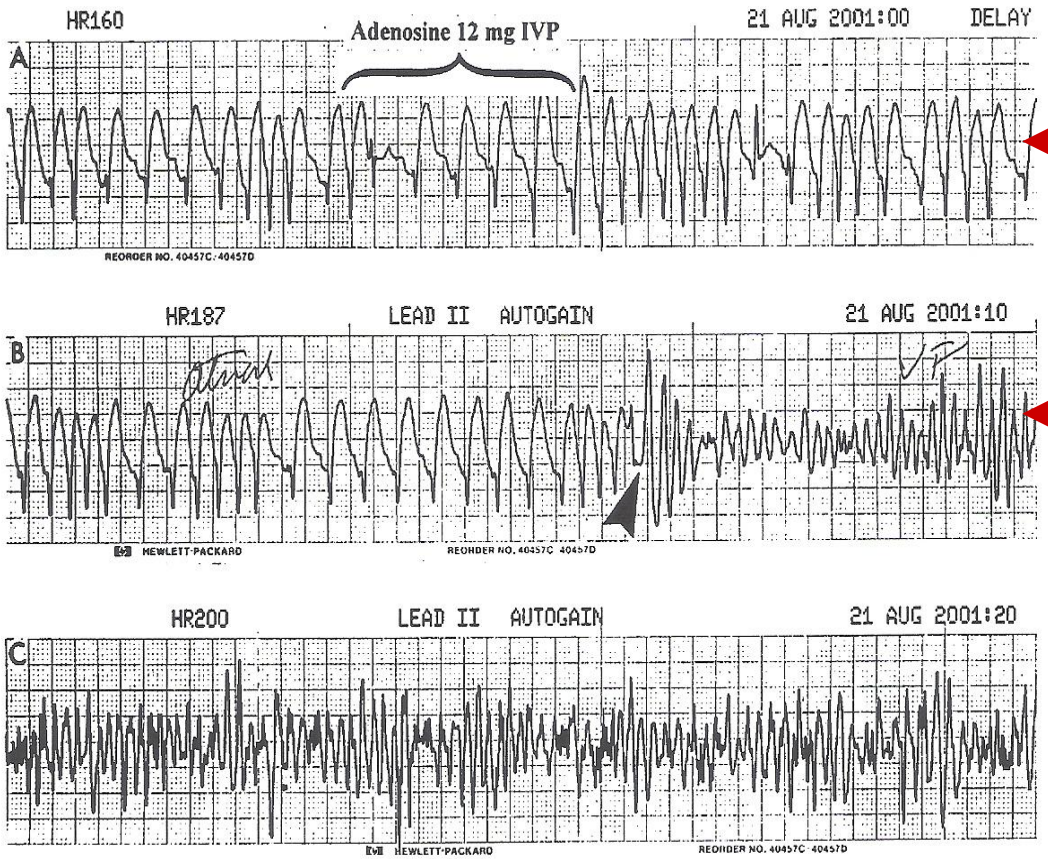


Adenosine Induced Ventricular Fibrillation in Wolff-Parkinson-White Syndrome

ANOOP K. GUPTA, CHETAN P. SHAH, ALOK MAHESHWARI, RANJAN K. THAKUR, OLIVER W. HAYES, and YASH Y. LOKHANDWALA

From the Thoracic and Cardiovascular Institute, Michigan State University and Sparrow Health System, Lansing, Michigan

PACE April 2002

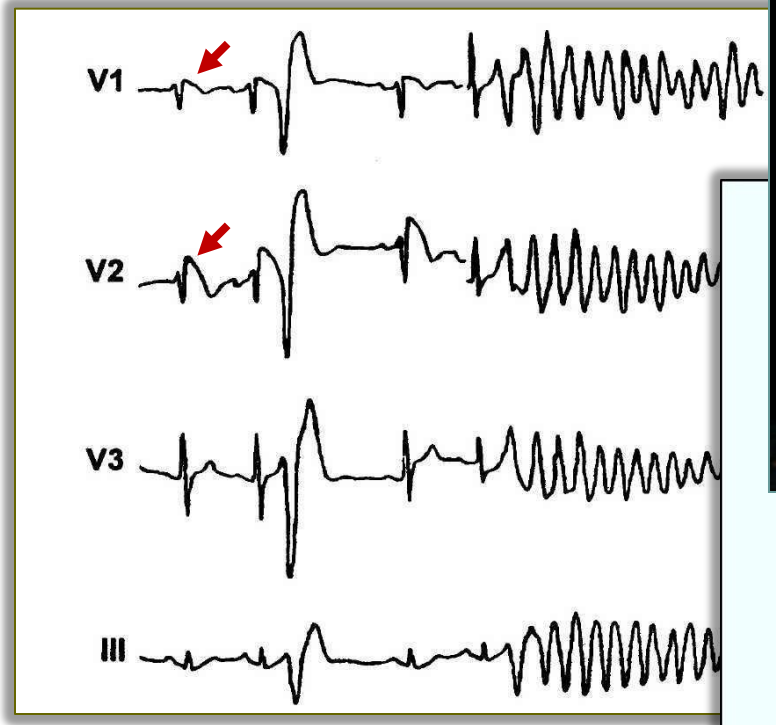


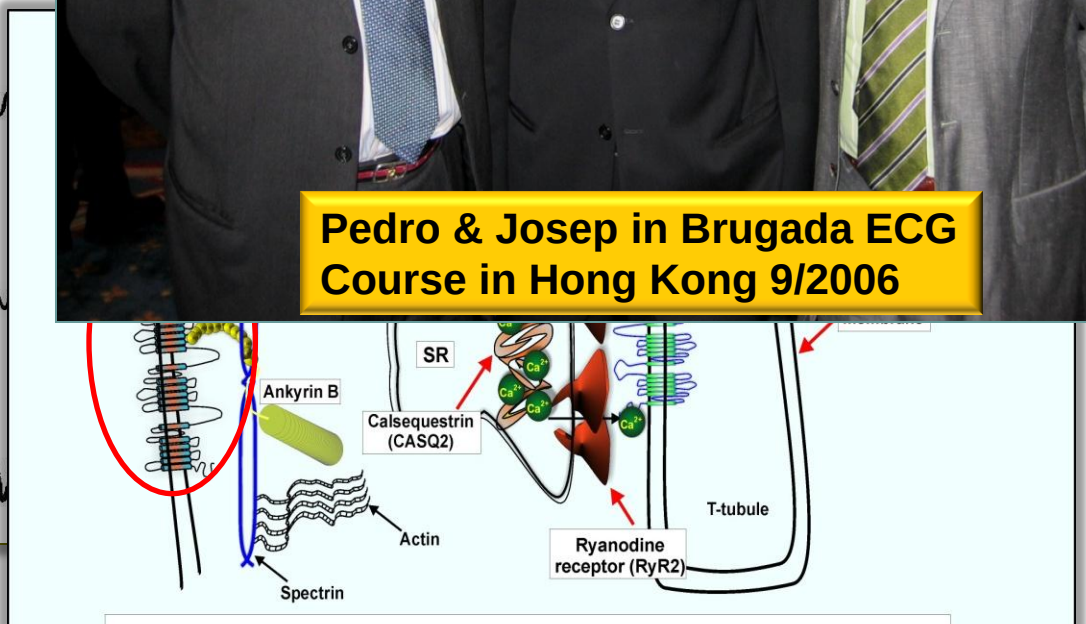
Pre-excited AF

Adenosine-induced VF

Brugada Syndrome (布嘉達綜合症)

- Coved type S
- VF / pc
- *SCN5A* mutatio

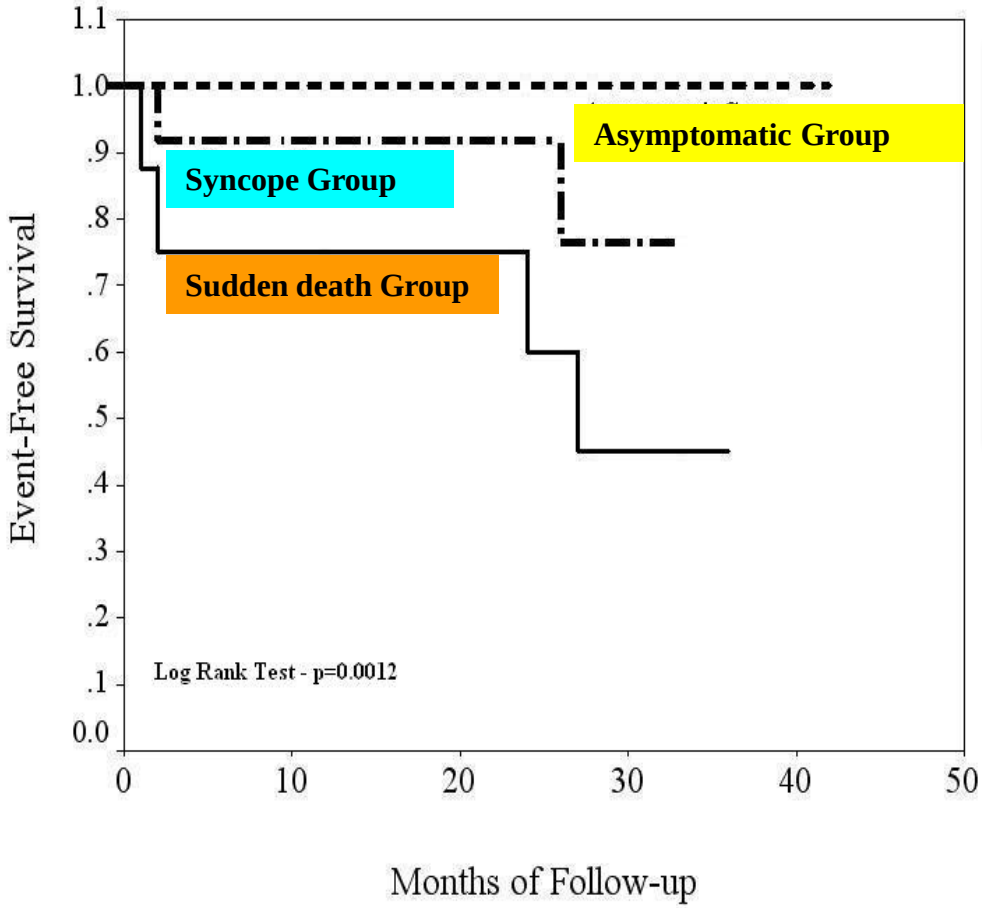




NS Mok 莫毅成
SG Priori
C Napolitano
KK Chan 陳國強
R Bloise
HW Chan 陳漢鏵
WH Fung 馮永康
YS Chan 陳日新

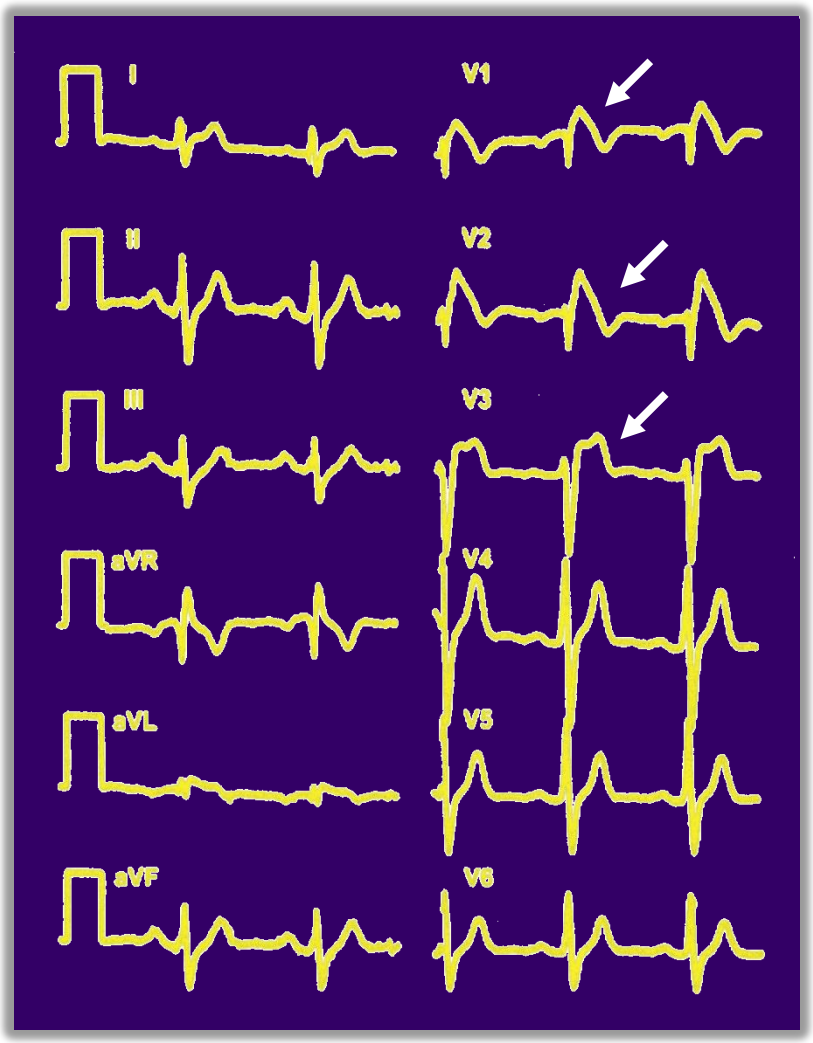
Clinical profile and genetic basis of Brugada syndrome in the Chinese population

Brugada綜合症華裔患者的臨床特點及患病的遺傳基礎

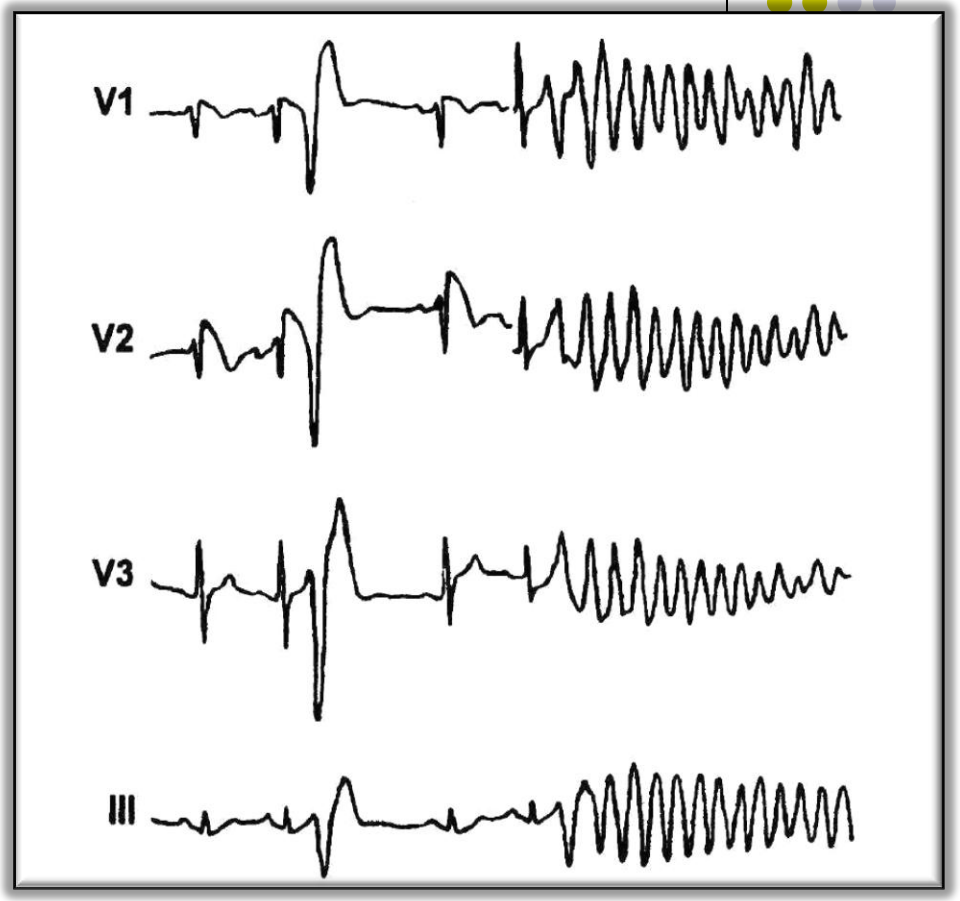


High chance of arrhythmia recurrence in symptomatic subjects with BrS

Mok et al, HKMJ Feb 2004

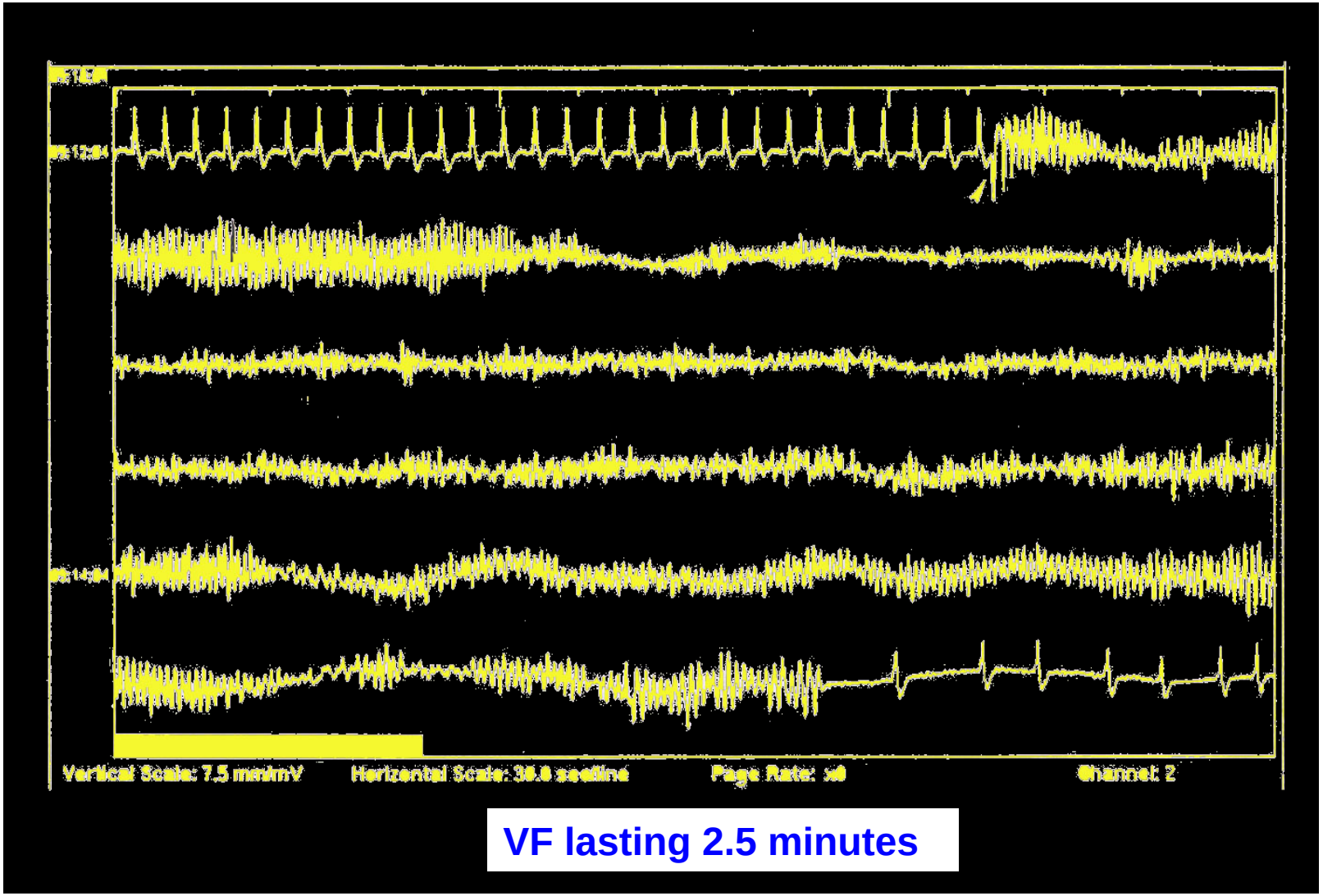


Typical ECG of Brugada Syndrome



Brugada syndrome presenting with VF

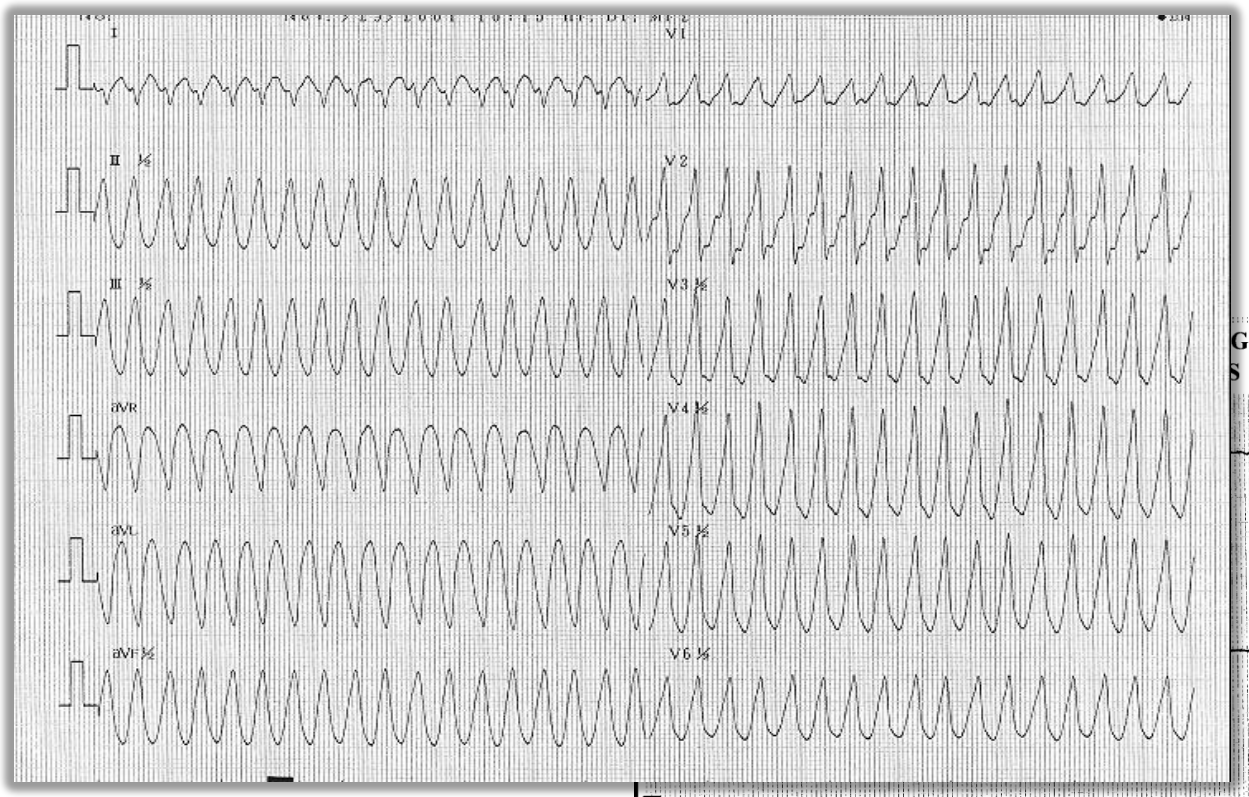
Holter recording of a self-terminating VF during convulsion

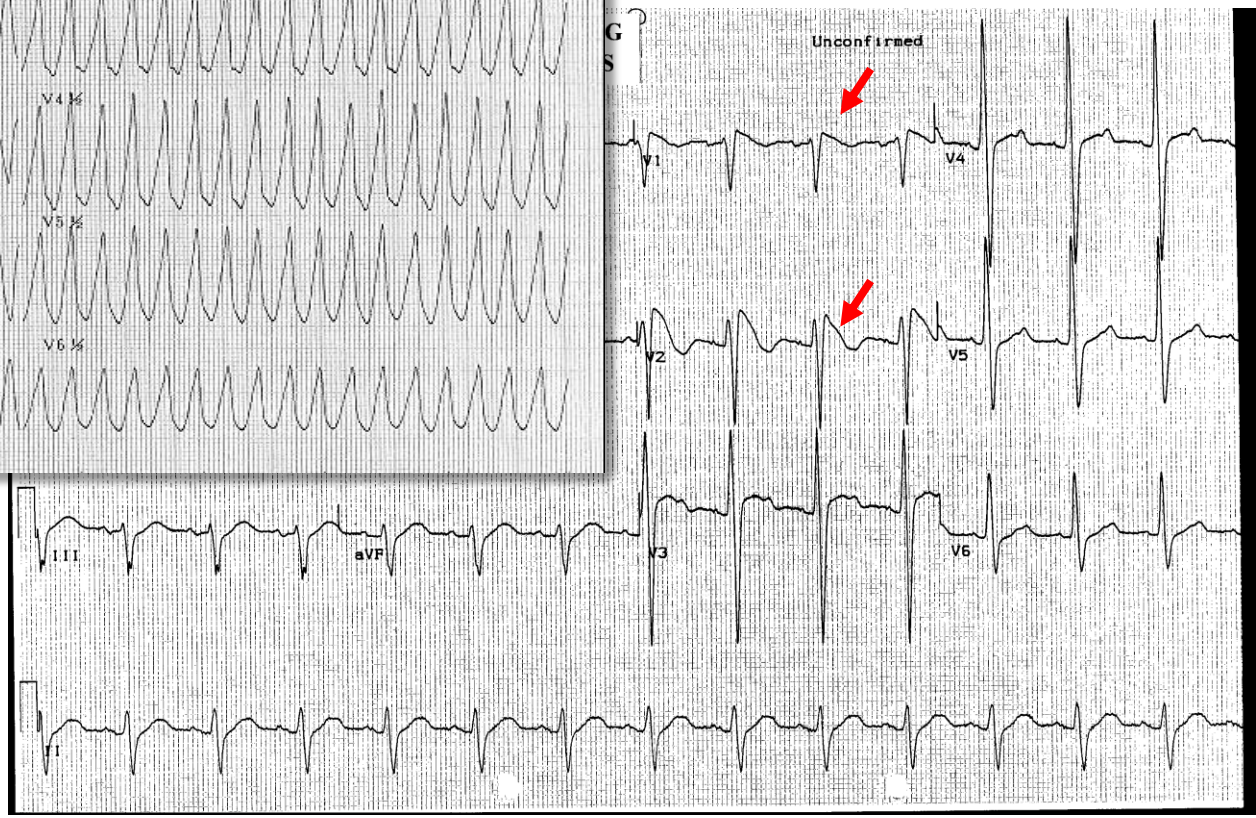


VF lasting 2.5 minutes

Mok NS, et al JHKCC 1999

BrS presenting with SMVT



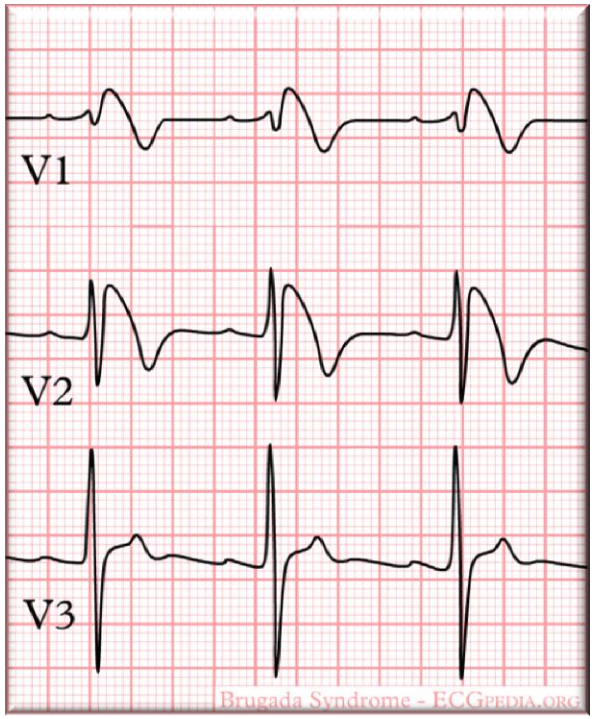


Mok et al, IJC 2003

Consensus on diagnosis of BrS

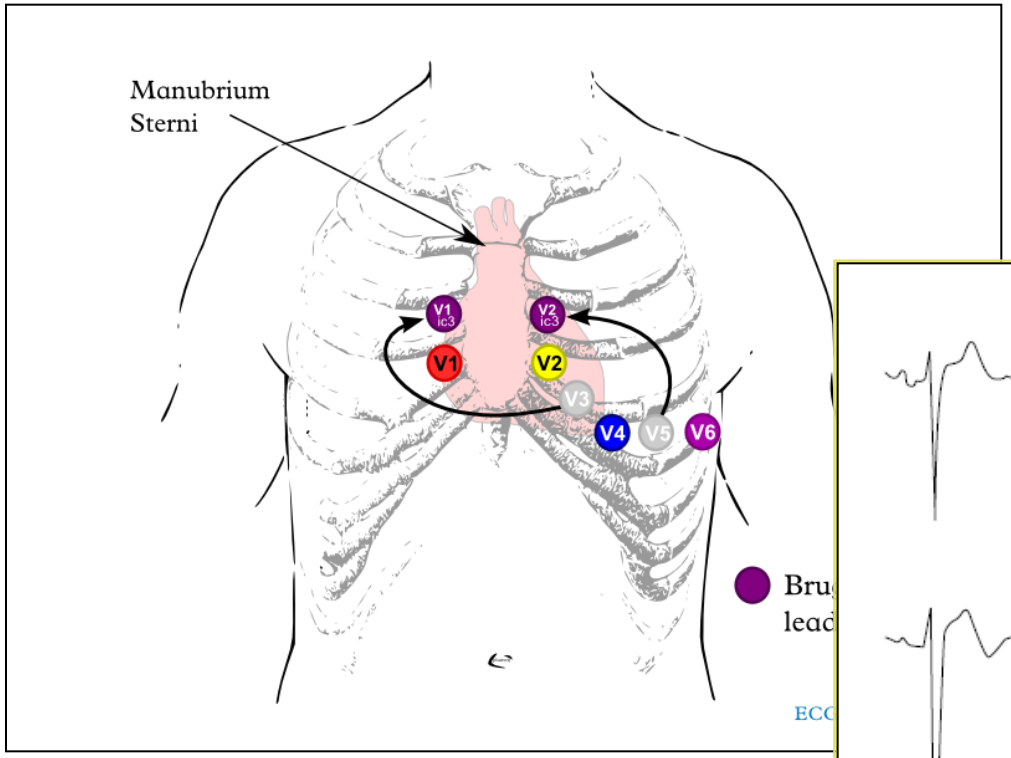
Type 1 Brugada ECG &
at least one of the followings :

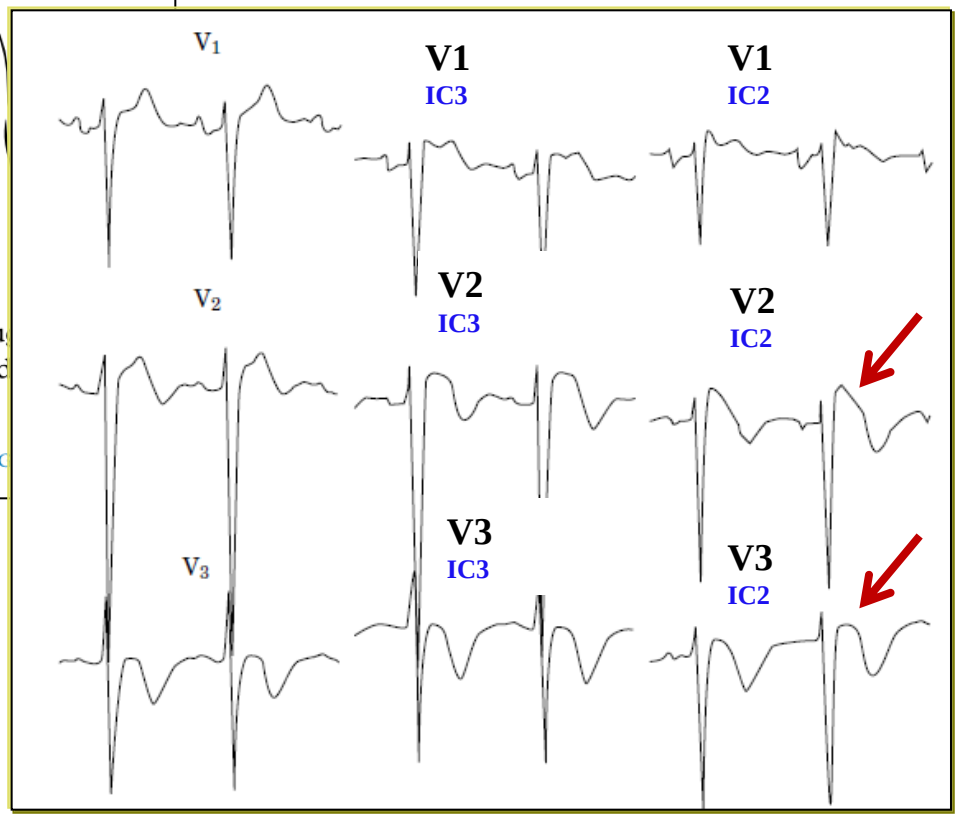
- VF / Polymorphic VT
- Syncope
- Type 1 Brugada ECG in family members
- Family history of SCD <45 years old
- Nocturnal agonal respiration



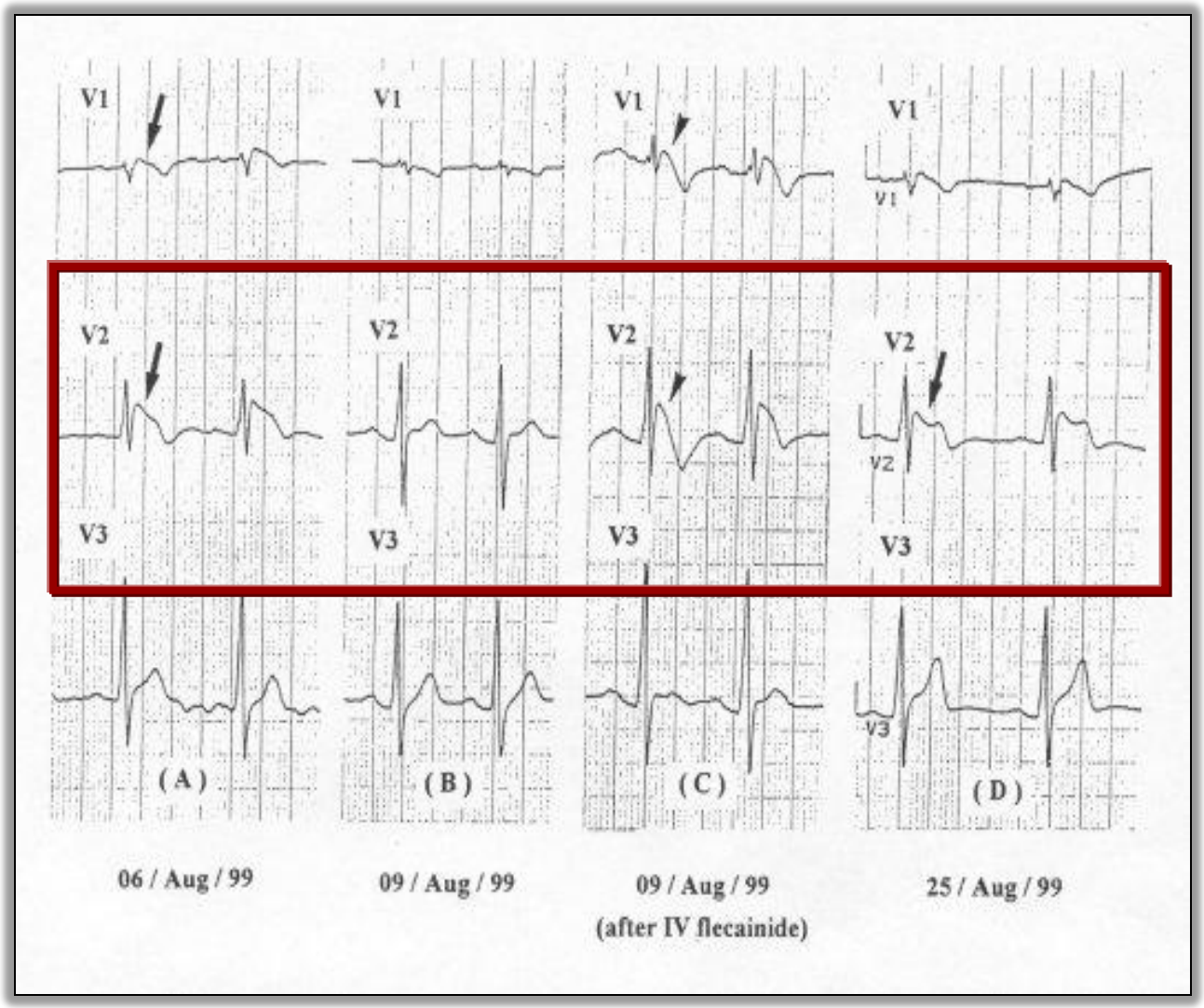
Type 1 Brugada ECG

Placement of V1 & V2 in 3rd or 2nd ICS improved diagnostic sensitivity in BrS





Brugada ECG with dynamic changes

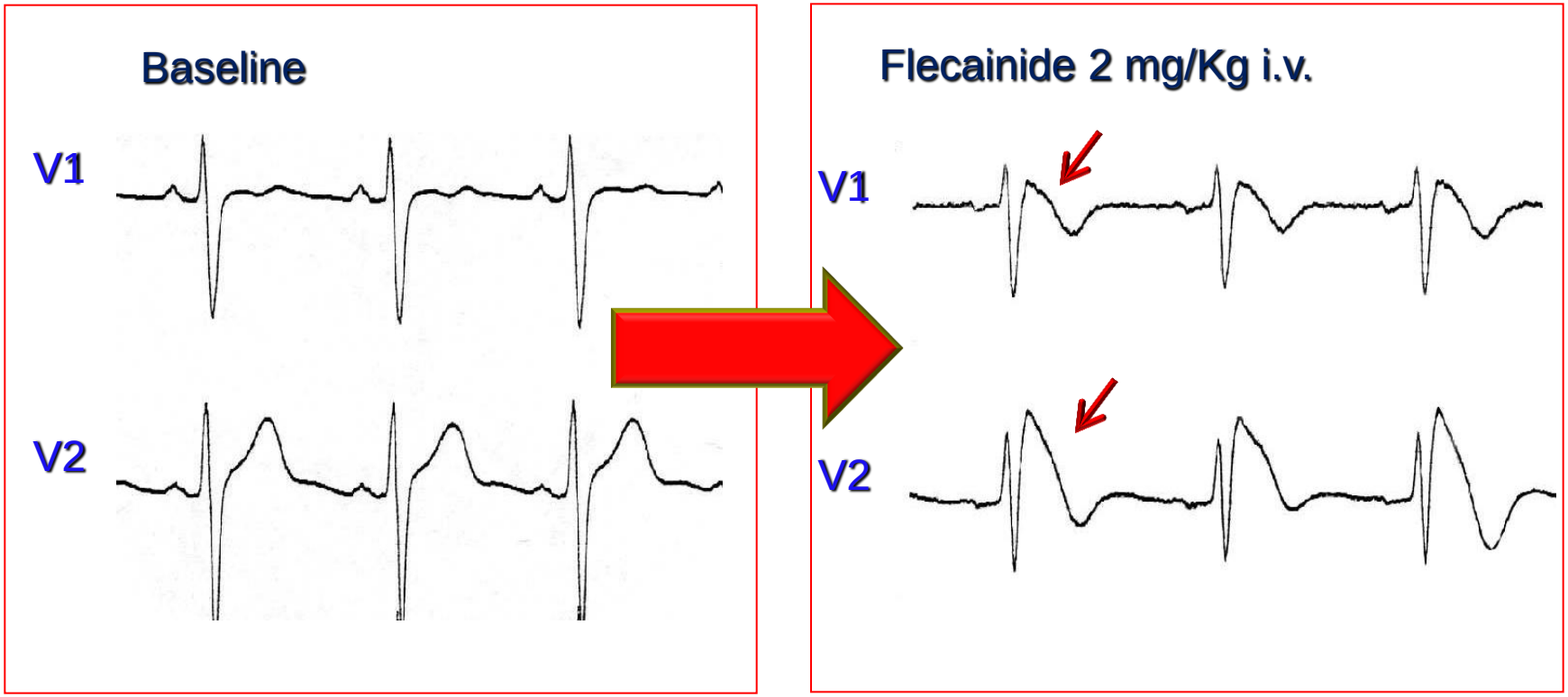


Drug provocation test in BrS

TABLE 2. Drugs Used to Unmask Brugada Syndrome

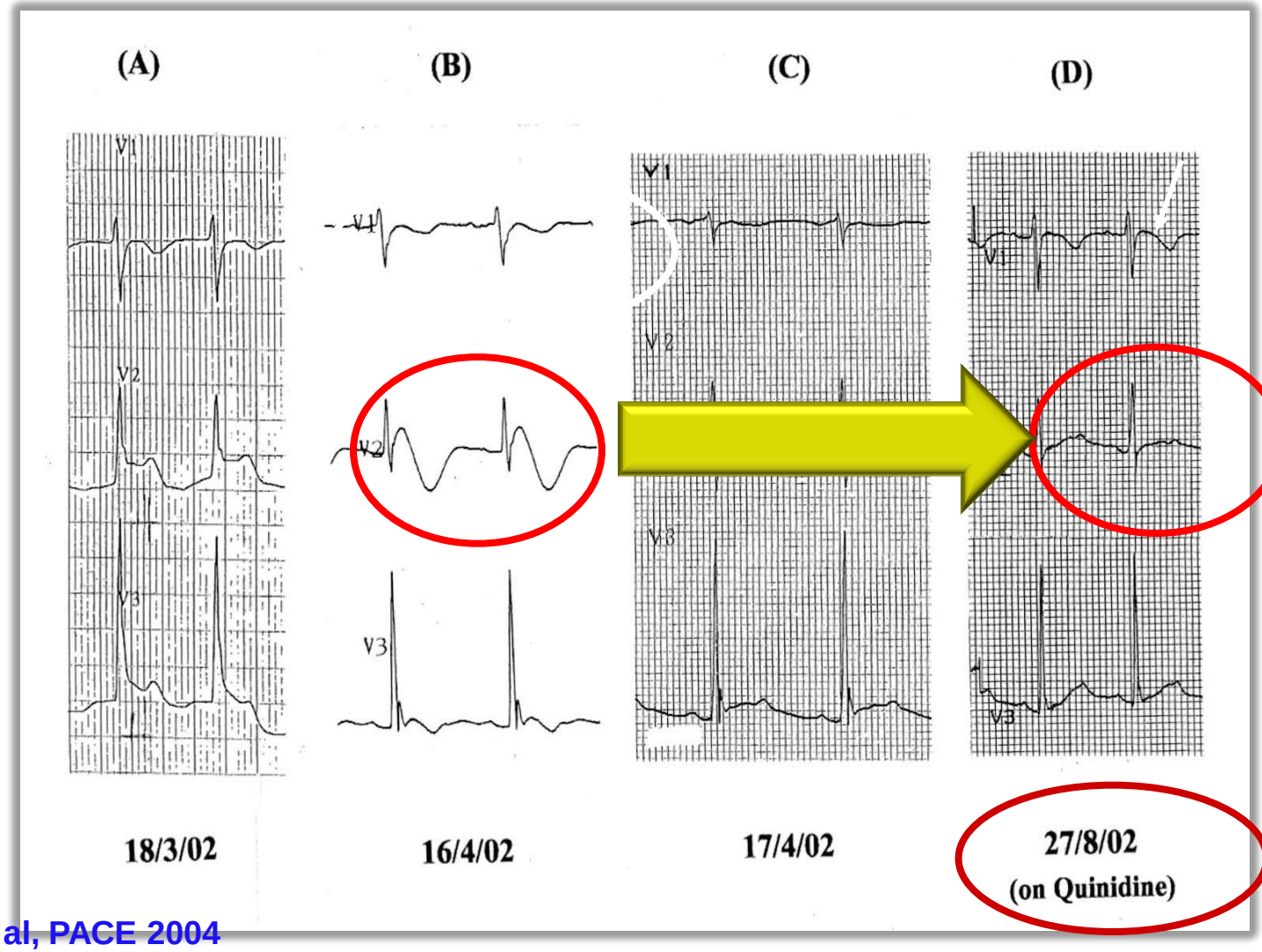
Drug	Dosage and Administration
Ajmaline	1 mg/kg over 5 min, IV
Flecainide	2 mg/kg over 10 min, IV (400 mg, PO)
Procainamide	10 mg/kg over 10 min, IV
Pilsicainide	1 mg/kg over 10 min, IV

Flecainide provocation test in BrS



IV flecainide unmasked Type 1 Brugada ECG pattern

Quinidine normalized the ST-segment elevation in BrS



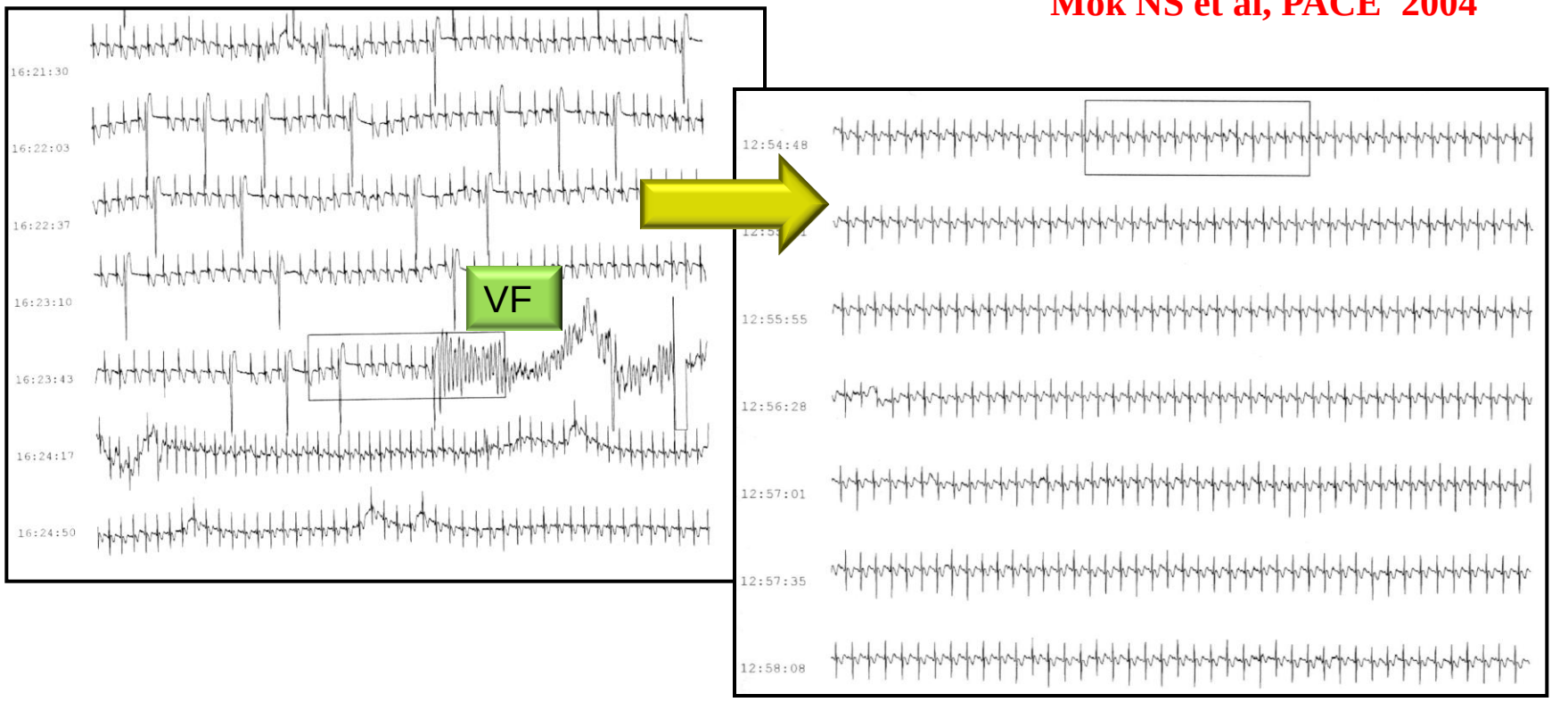
Mok et al, PACE 2004

Successful Use of Quinidine in Treatment of Electrical Storm in Brugada Syndrome

NGAI-SHING MOK, NGAI-YIN CHAN, and ALEX CHI-SUEN CHIU*

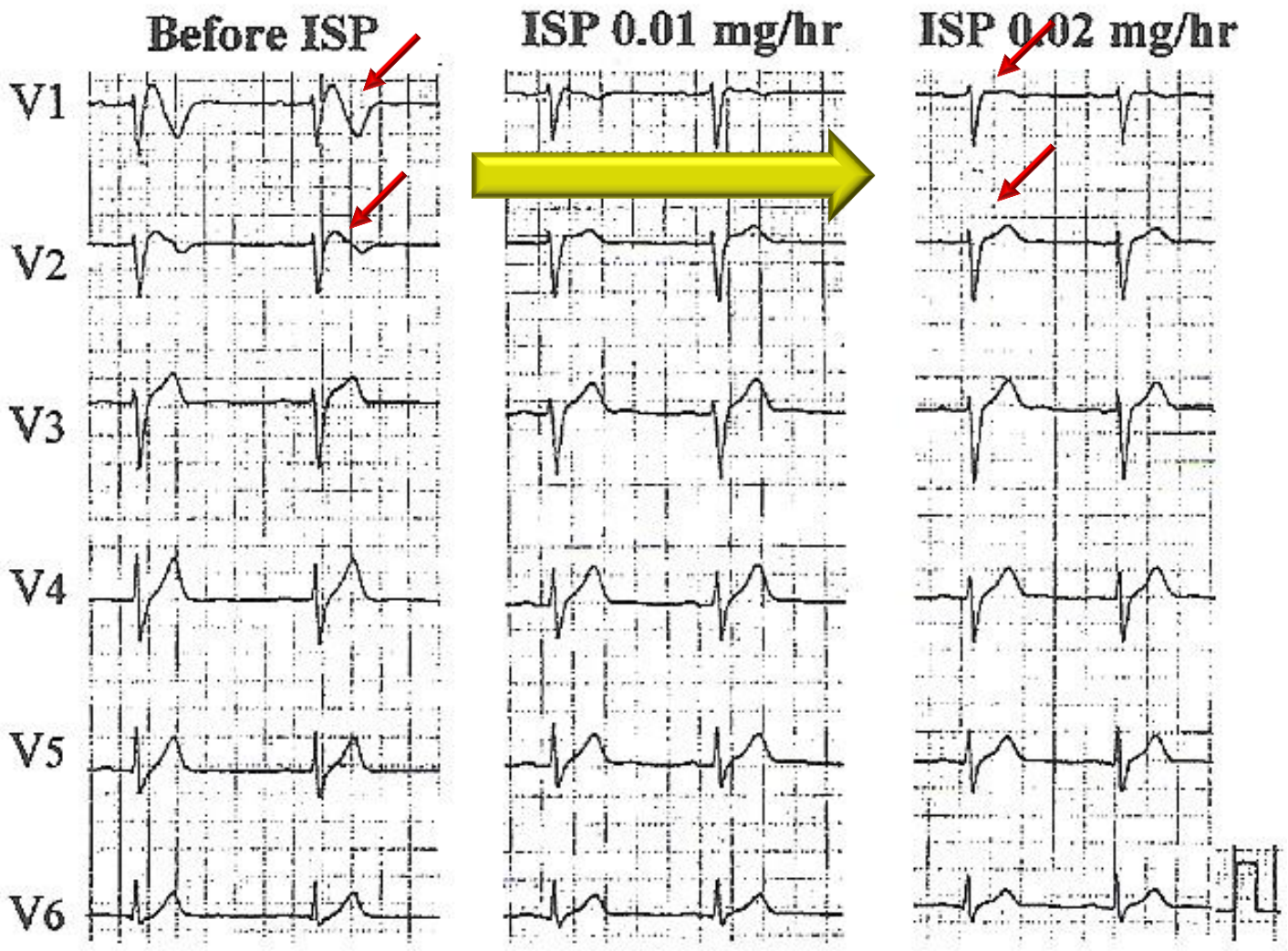
From the Cardiology Team, Department of Medicine and Geriatrics, Princess Margaret Hospital, Hong Kong, and the *Department of Medicine and Geriatrics, Caritas Medical Center, Hong Kong, China

Mok NS et al, PACE 2004



Quinidine suppressed VF recurrences

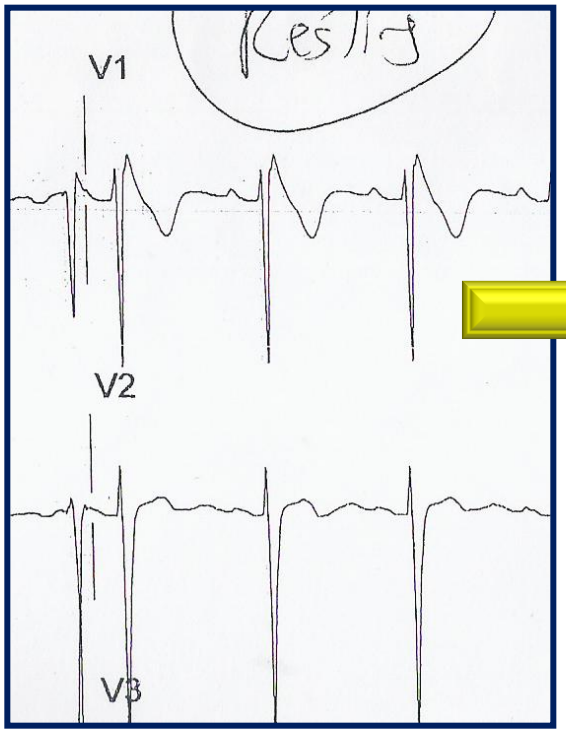
Isoprenaline normalized ST elevation in BrS

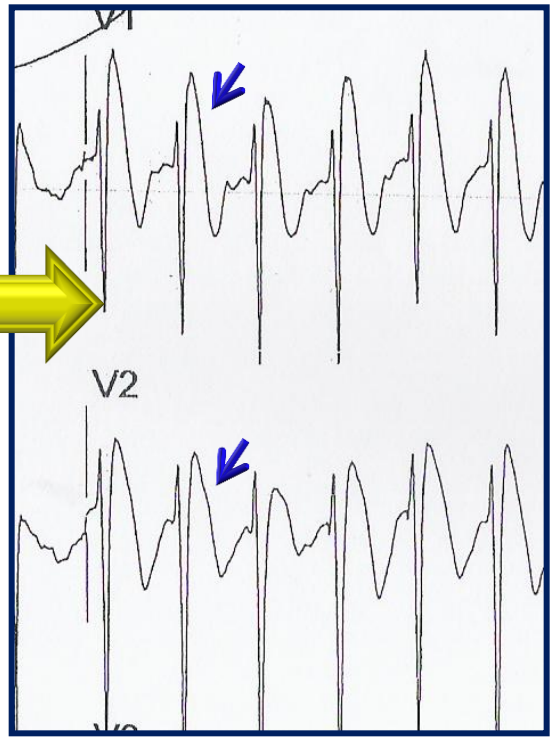


Exercise unmasked BrS in a patient with *SCN5A* p.Arg1897Trp mutation

- M/17, father died of SD at 49
- Treadmill exercise test -Type 1 Brugada ECG
- Genetic study in PMH - ***SCN5A* p.Arg1897Trp** mutation

Resting





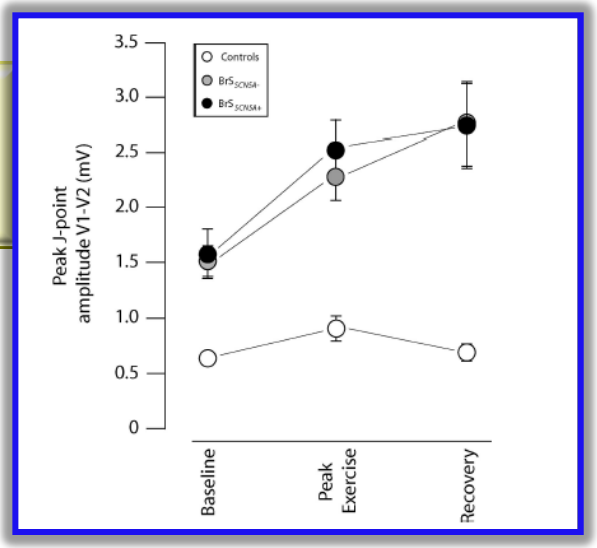
Post Exercise

Exercise augments ST elevation in BrS

Exercise-Induced ECG Changes in Brugada Syndrome

Amin AS et al, Circulation 2009

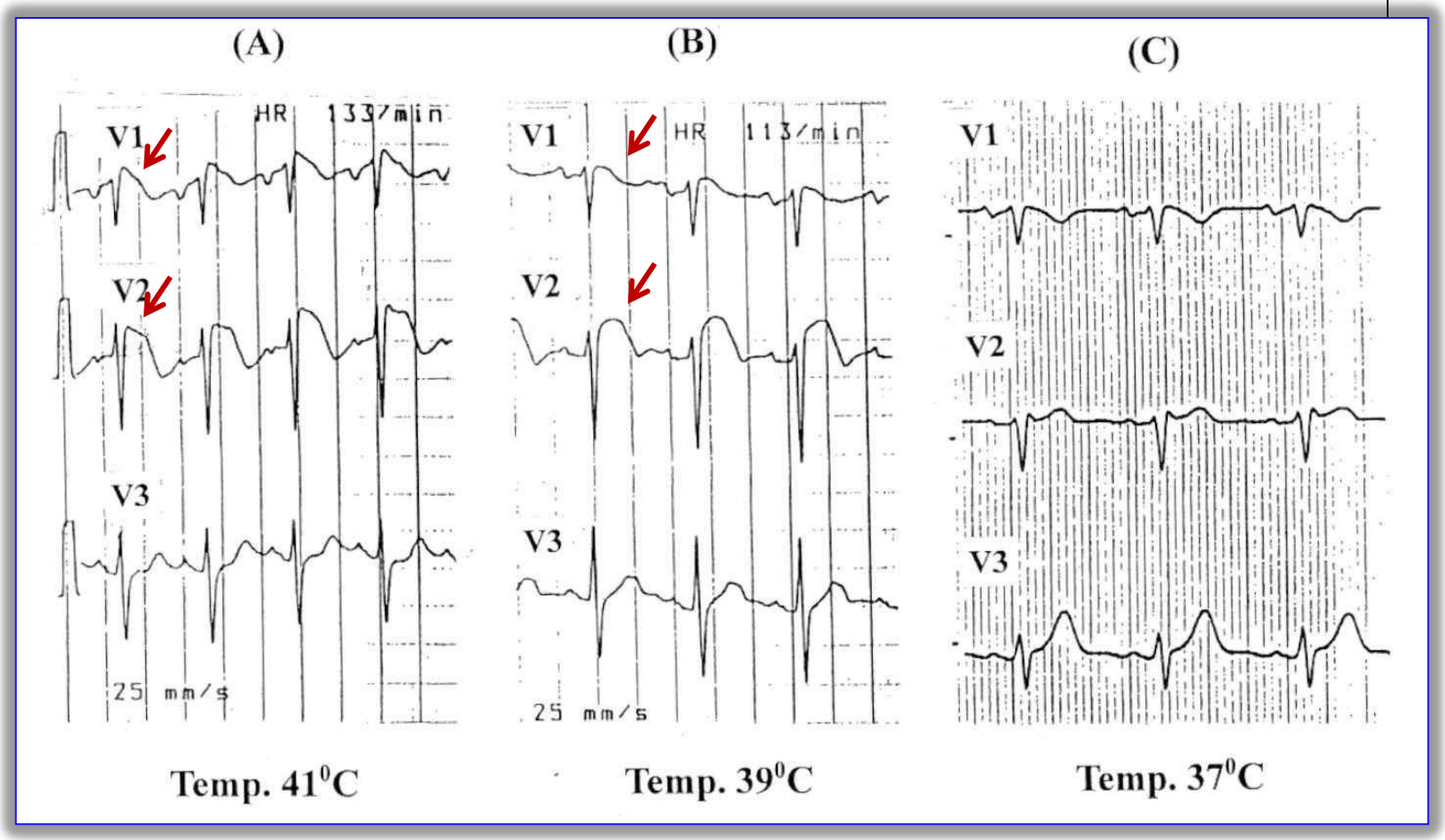
- 50 Brugada patients + 35 controls
- Exercise aggravated the ECG phenotype in BrS



Augmented ST-Segment Elevation During Recovery From Exercise Predicts Cardiac Events in Patients With Brugada Syndrome

Makimoto H et al, JACC 2010

Fever unmasked Brugada ECG in a patient with *SCN5A* H681P mutation



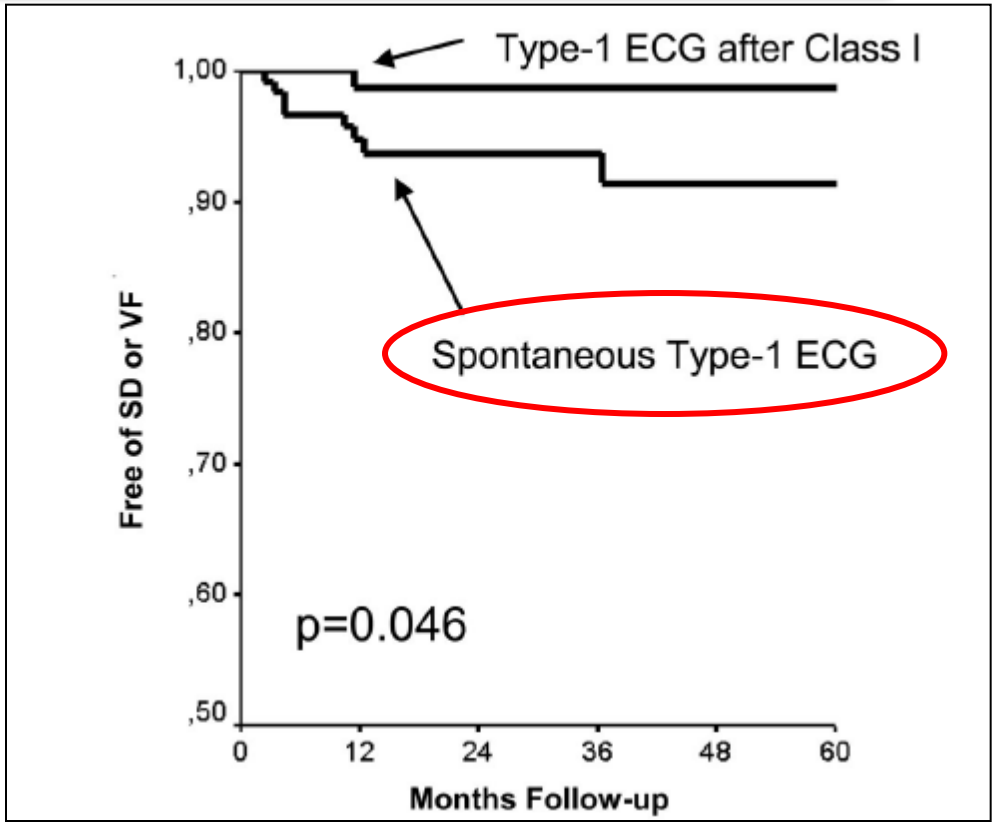
Mok et al, JCE 2003

Spontaneous Type 1 Brugada ECG predicts cardiac events

Long-Term Prognosis of Individuals With Right Precordial ST-Segment–Elevation Brugada Syndrome

Lars Eckardt, MD*; Vincent Probst, MD*; Jeroen P.P. Smits, MD*; Eric Schulze Bahr, MD; Christian Wolpert, MD; Rainer Schimpf, MD; Thomas Wichter, MD, FESC; Pierre Boisseau, PhD; Achim Heinecke, PhD; Günter Breithardt, MD, FESC; Martin Borggrefe, MD; Herve LeMarec, MD, PhD; Dirk Böcker, MD; Arthur A.M. Wilde, MD, FESC

Circulation 2005



Early Repolarization Syndrome (ERS)

Early repolarization (ER) manifesting as

- J wave
- a notch or slur on terminal part of QRS complex
- ST elevation

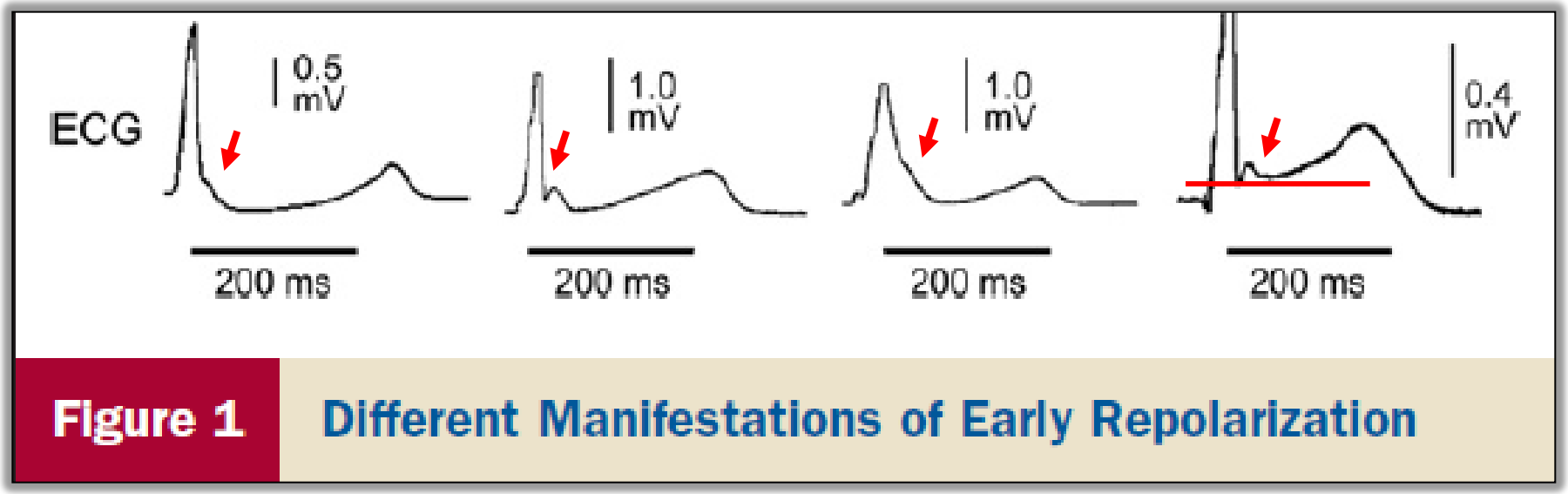


Figure 1 Different Manifestations of Early Repolarization

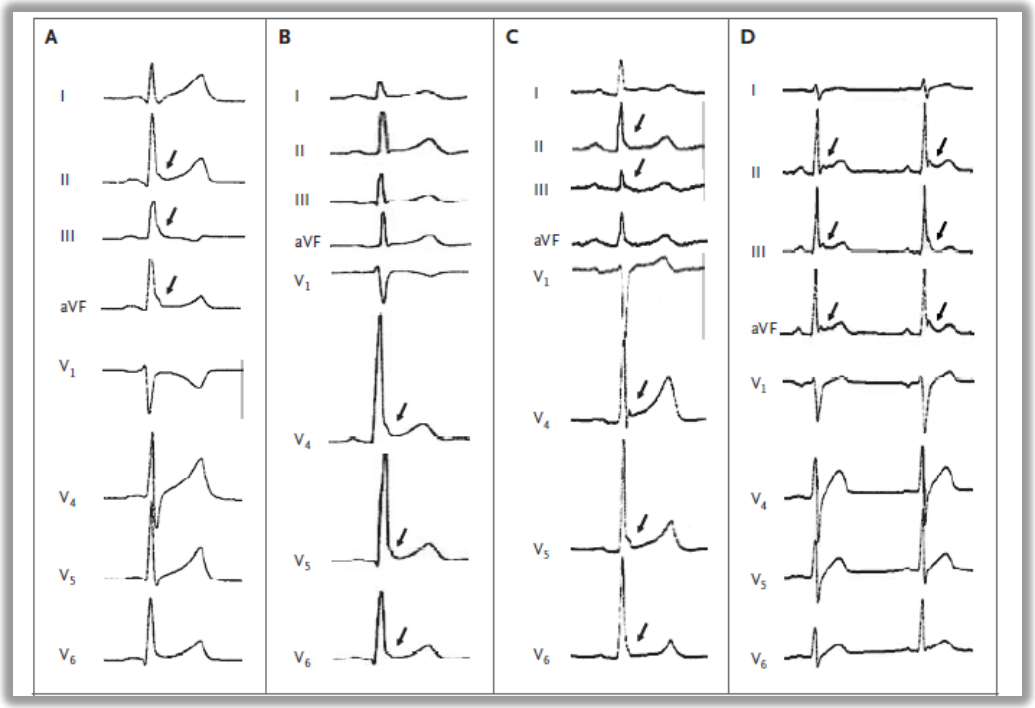
Early Repolarization Syndrome (ERS)

Sudden Cardiac Arrest Associated with Early Repolarization

Michel Haïssaguerre, M.D., Nicolas Derval, M.D., Frederic Sacher, M.D.,

Increased prevalence of early repolarization among 206 patients with idiopathic VF

(31% vs 5% in control subjects)



Haissaguerre et al NEJM 2008

ER predicts higher VF recurrence in pts with idiopathic VF

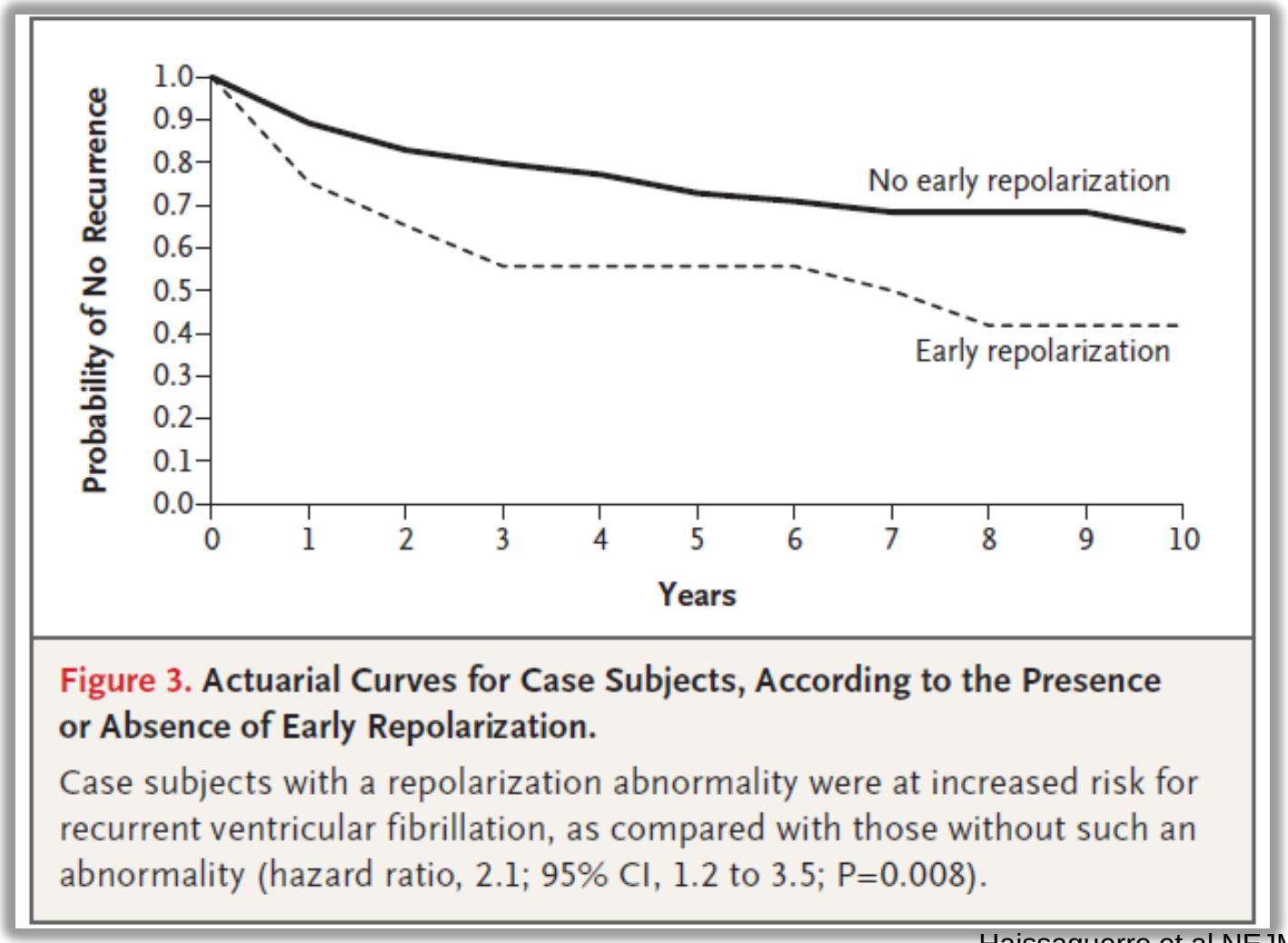
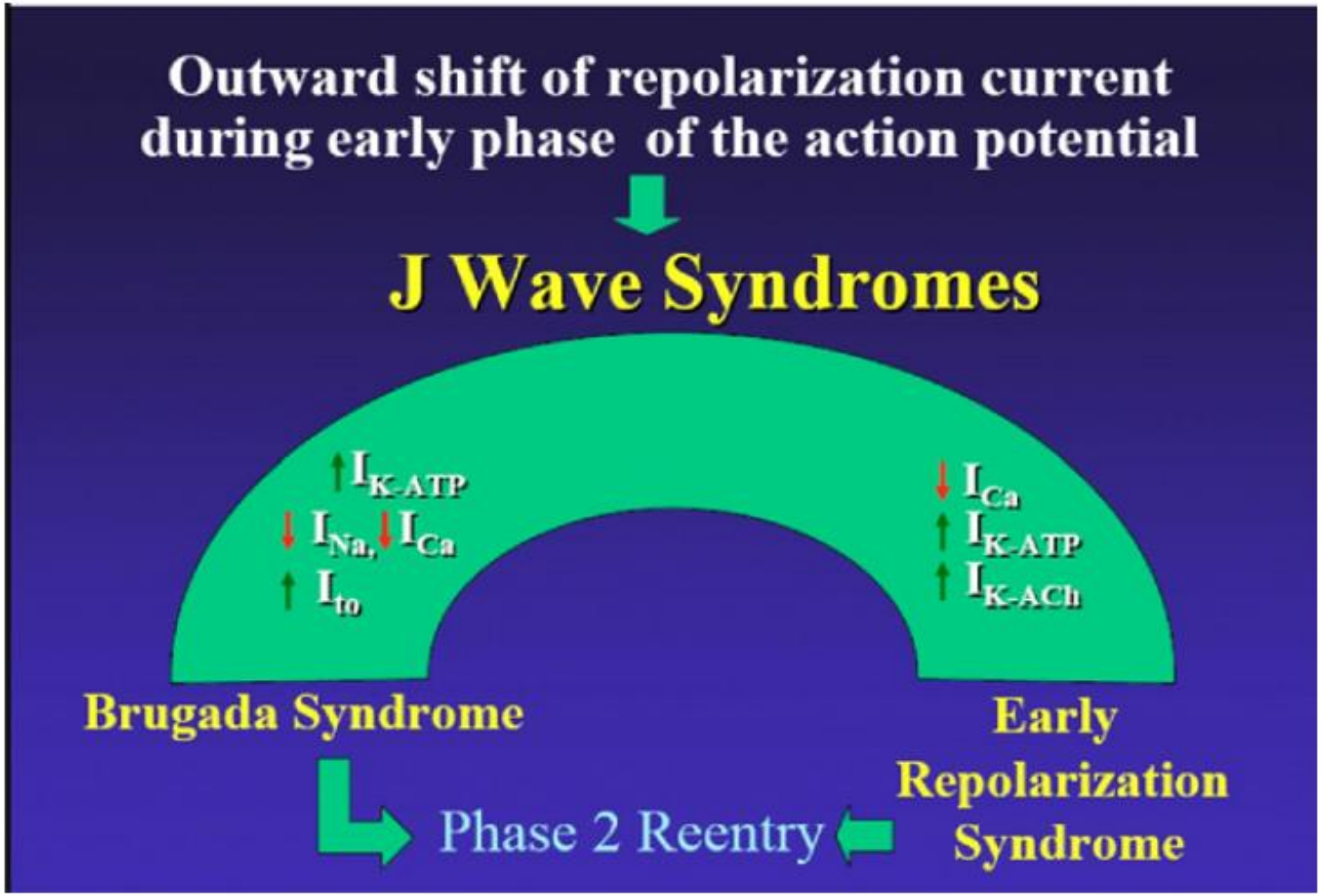


Figure 3. Actuarial Curves for Case Subjects, According to the Presence or Absence of Early Repolarization.

Case subjects with a repolarization abnormality were at increased risk for recurrent ventricular fibrillation, as compared with those without such an abnormality (hazard ratio, 2.1; 95% CI, 1.2 to 3.5; P=0.008).

Haissaguerre et al NEJM 2008

J Wave Syndromes



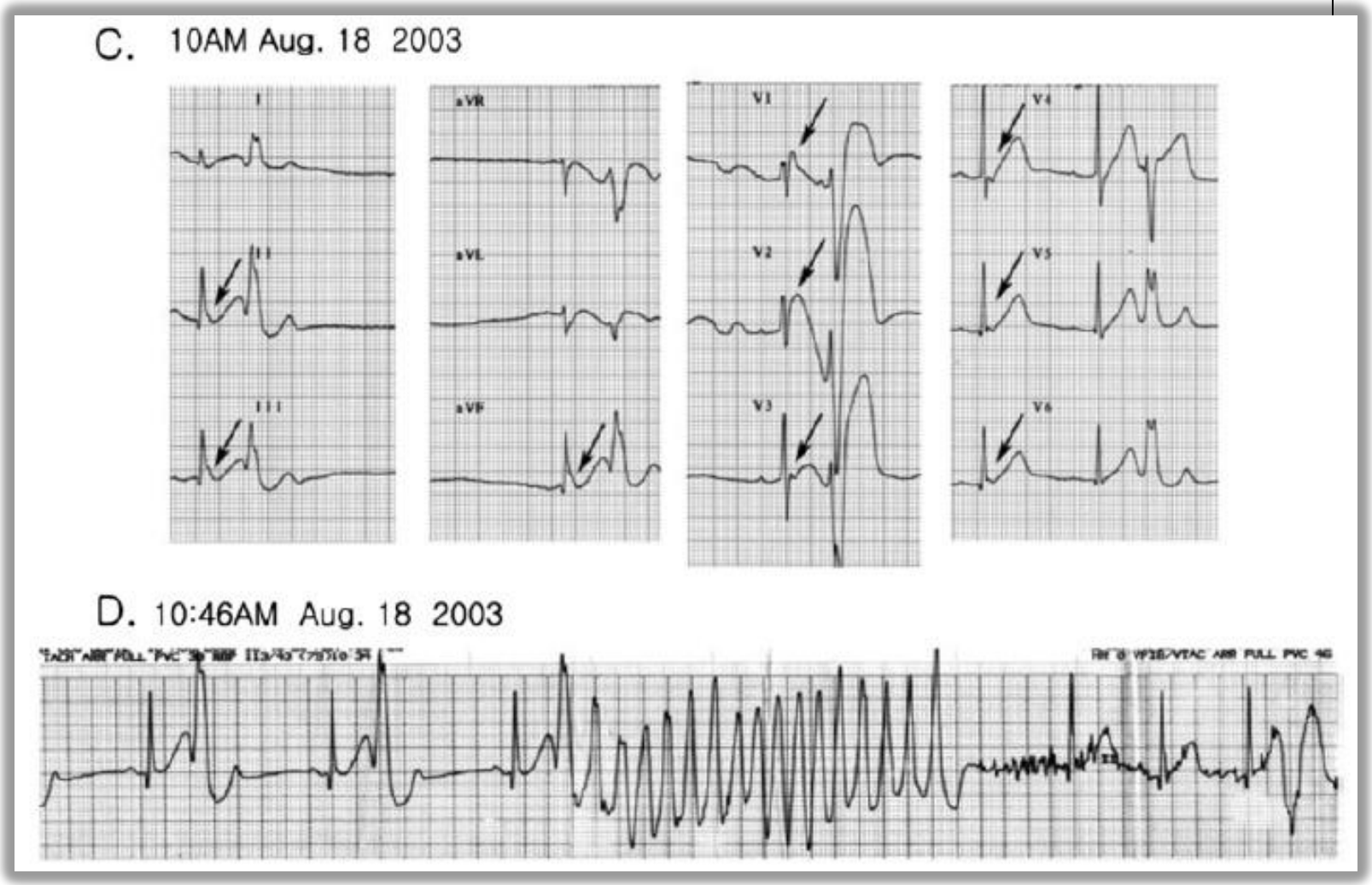
Antzelevitch, et al Heart Rhythm 2010

J Wave syndromes - classification

Table 1 J-wave syndromes: Similarities and differences

	ERS Type 1	ERS Type 2	ERS Type 3	Brugada syndrome
Anatomic location responsible for chief electrophysiologic manifestations	Anterolateral left ventricle	Inferior left ventricle	Left and right ventricles	Right ventricle
Leads displaying J point/ J-wave abnormalities	I, V_4–V_6	II, III, aVF	Global	V_1–V_3
Response of J-wave amplitude/ST elevation to:	Lateral leads	Inferior or Inferolateral leads	Global	R precordial leadS
Bradycardia or pause	Increase	Increase	Increase	Increase
Sodium channel blockers	Little or no change	Little or no change	Little or no change	Increase
Gender dominance	Male	Male	Male	Male

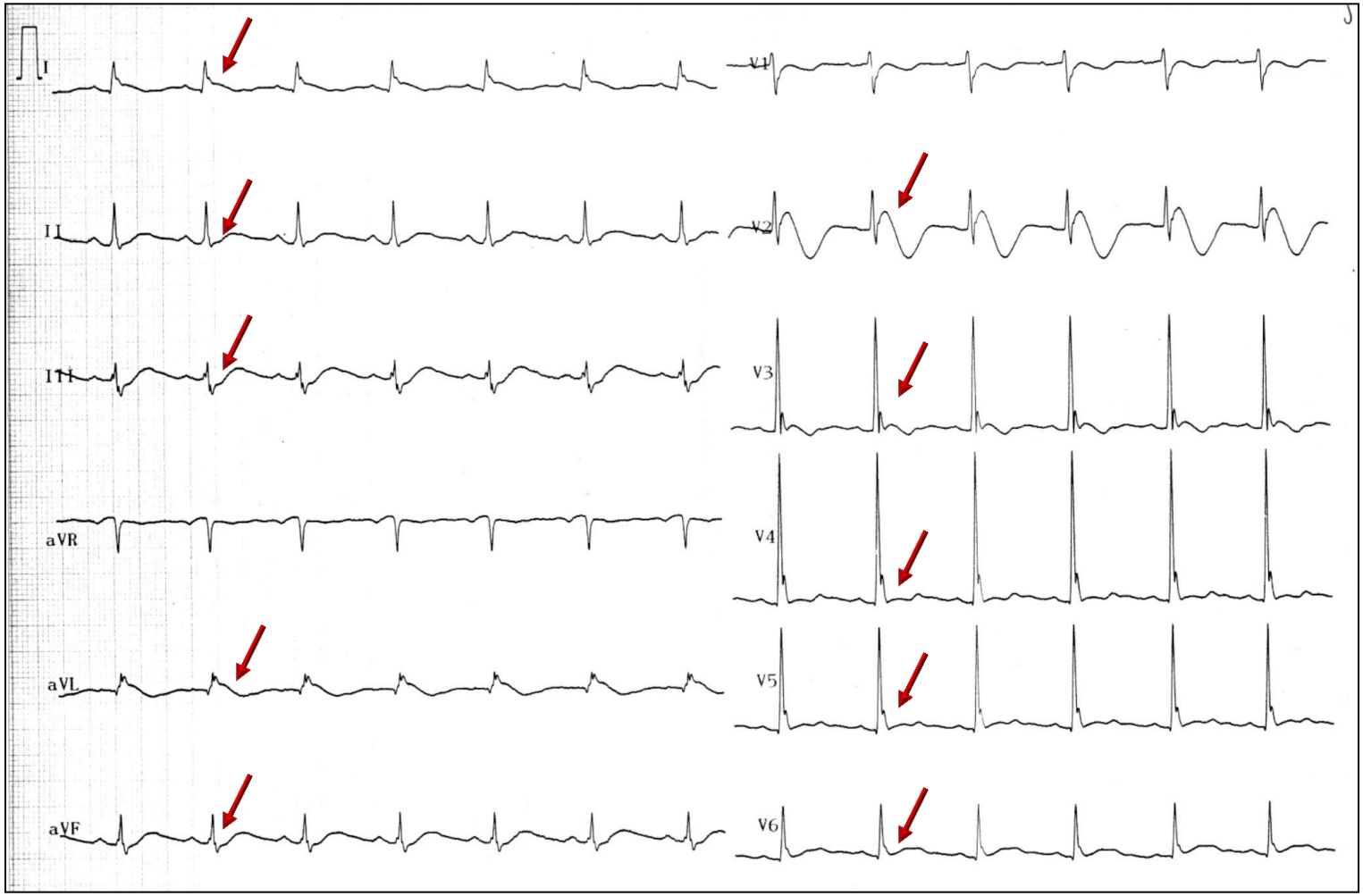
J Wave syndrome – ERS Type 3



J Wave syndrome – ERS Type 3 in a HK Chinese

M/17 Survivor of a VF storm

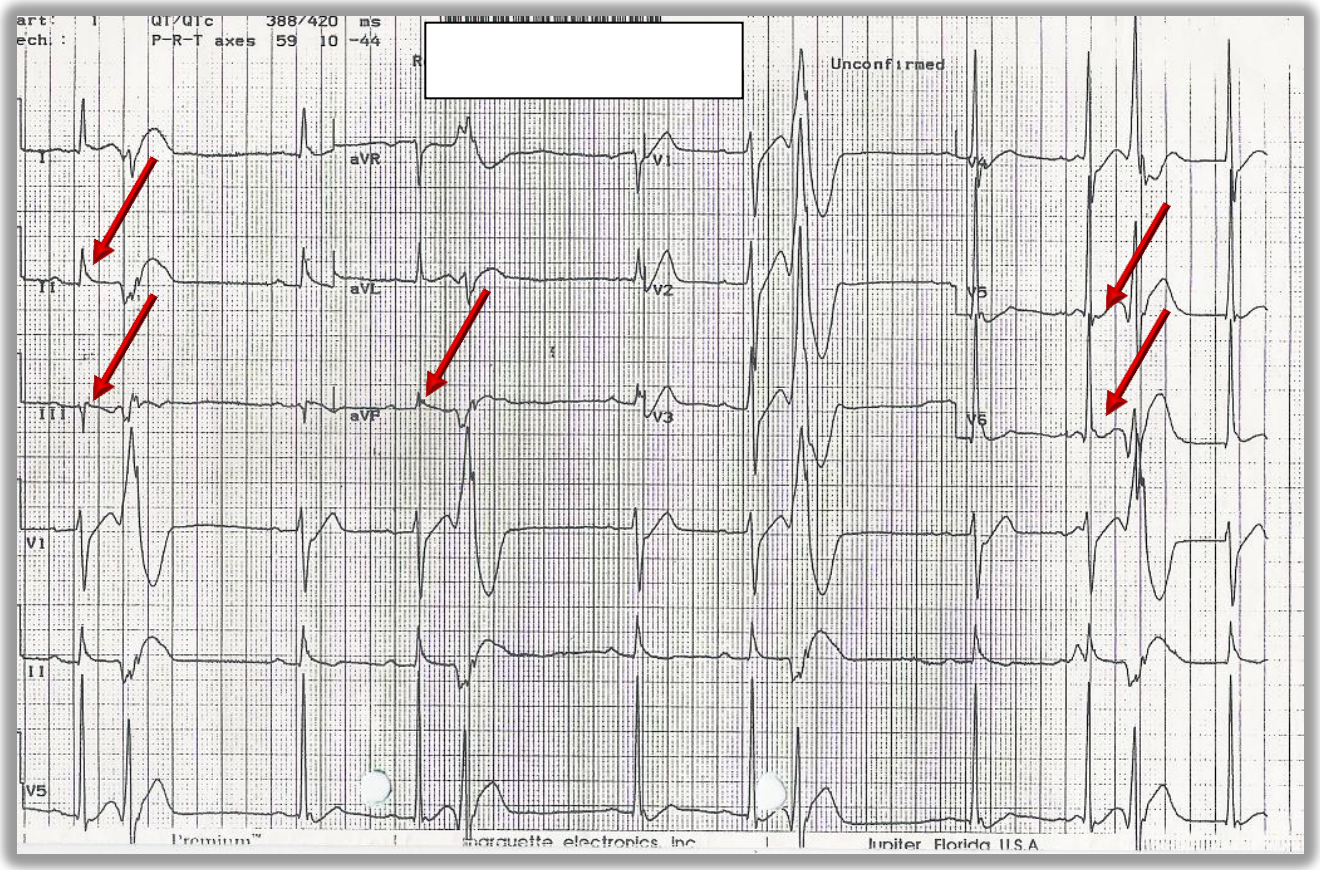
J waves over limb and precordial leads



ERS Type 2 (ER in infero-lateral leads) in a patient with idiopathic VF

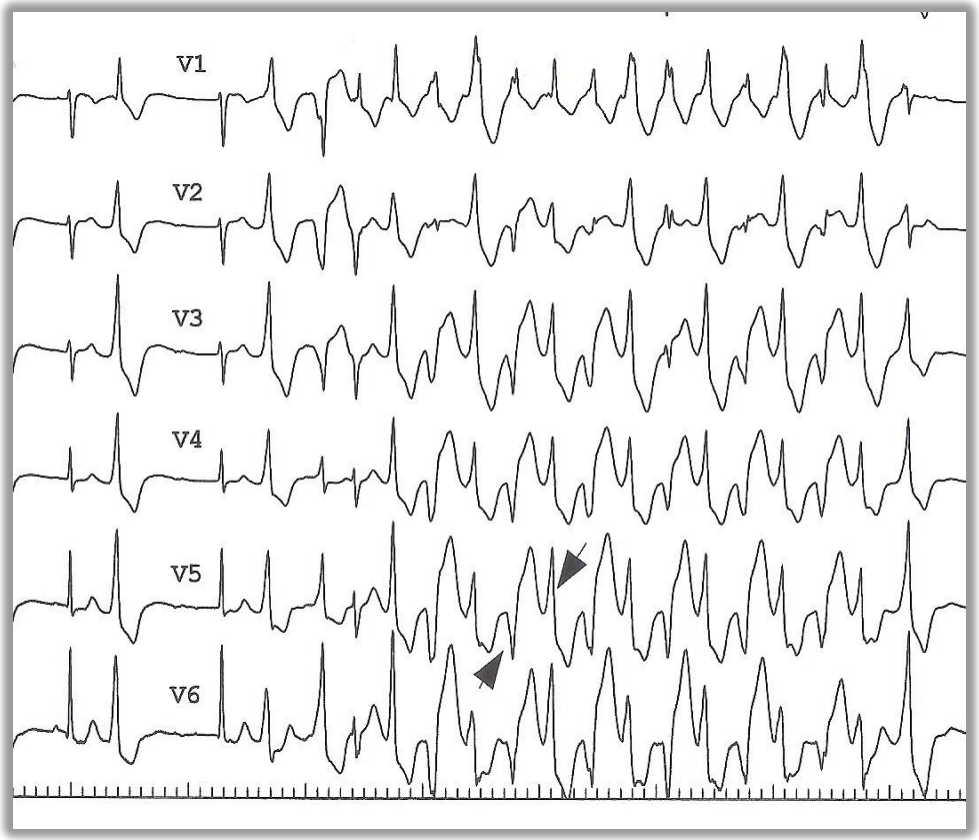
M/ 55

Recurrent VF cardiac arrest survivor



Catecholaminergic Polymorphic VT (CPVT)

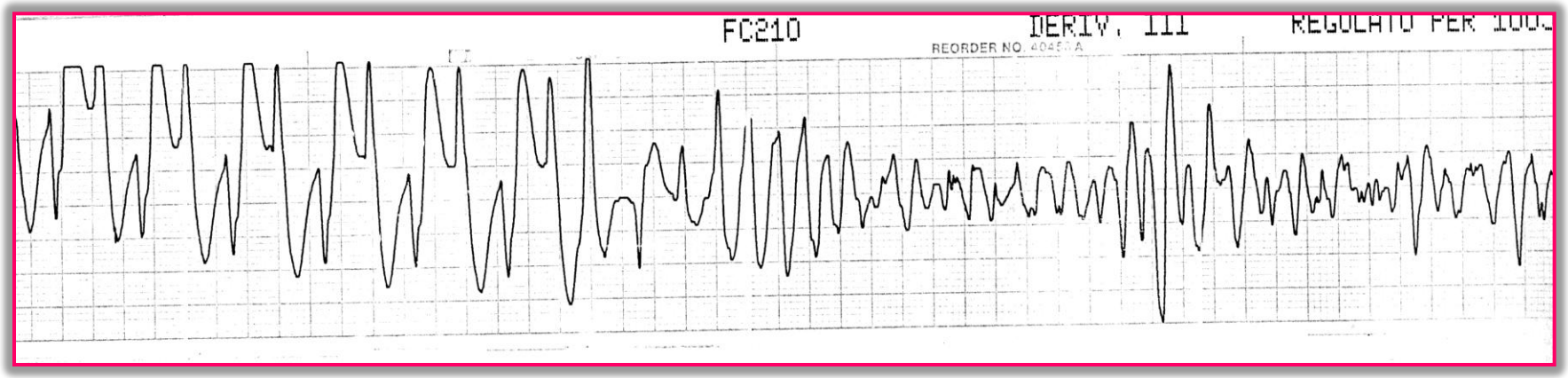
Stress or exercise-induced VT / VF causing SCD



Bi-directional VT with a RBBB pattern & alternating QRS axis

Catecholaminergic Bi-directional VT degenerating into VF

P.G, female, 9yrs

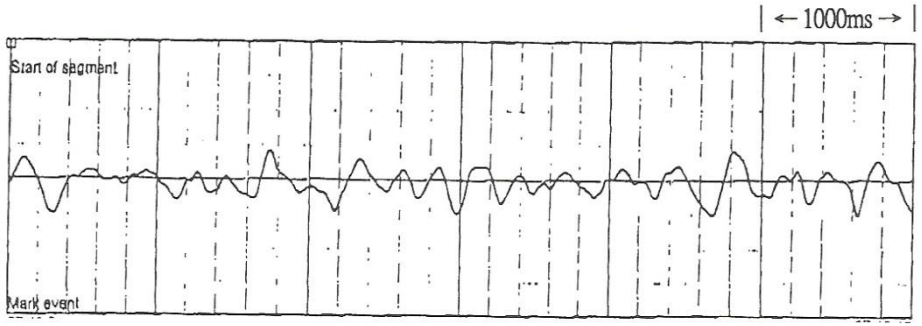


CPVT- A newly-recognized cause of SCD and syncope in Hong Kong Chinese

Mok NS et al , CMJ 2006

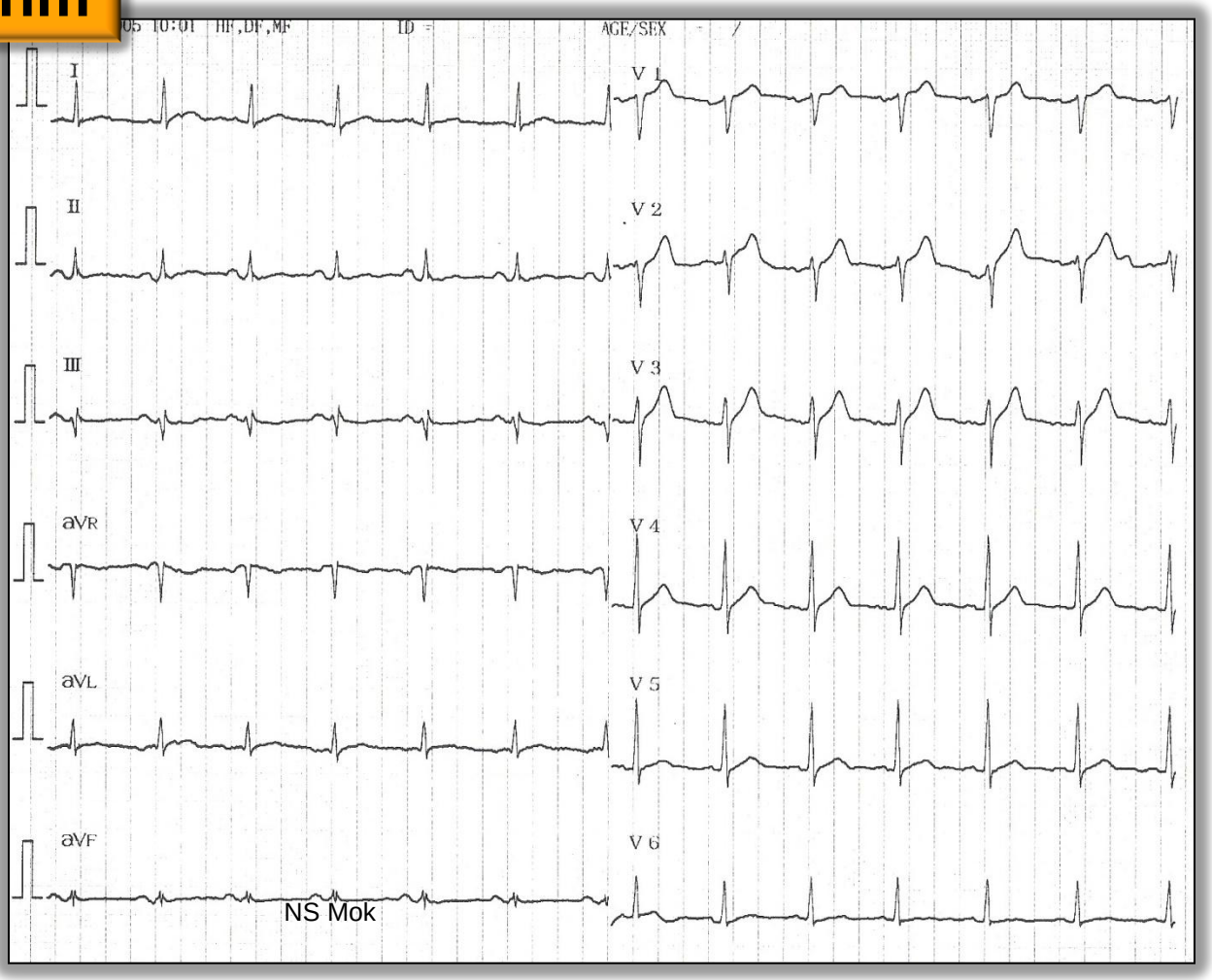
Shing Po Daily News 10/2005

- F/14, Chinese
- Hx of syncope after quarrelling with friend
- Collapsed after catching a bus

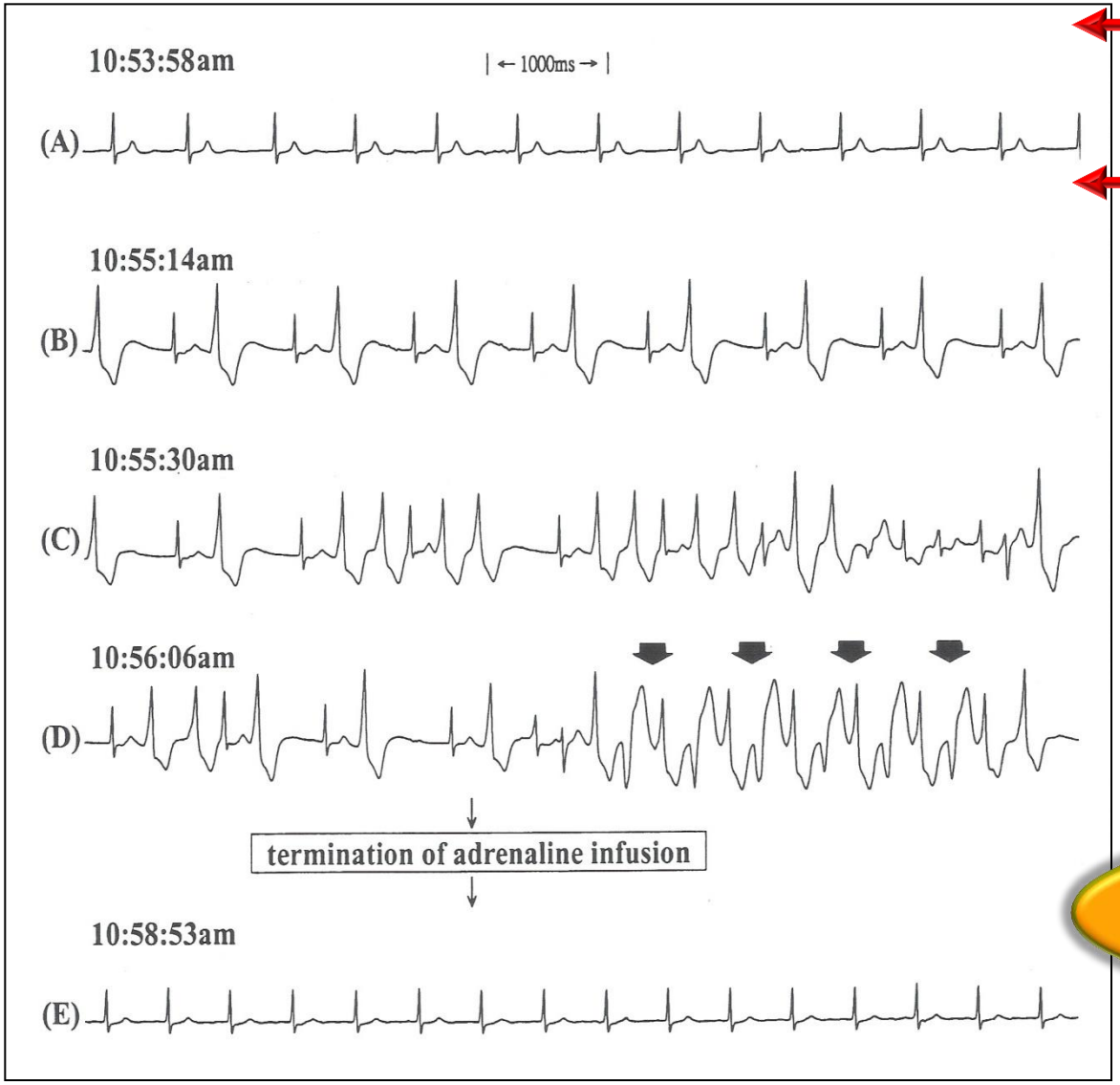


VF recorded on AED

Normal 12-lead ECG recorded in sinus rhythm



Adrenaline provocation test in CPVT



Monomorphic PVCs

Polymorphic VT

Bi-directional VT

CPVT confirmed by *hRyR2* mutation

Mok NS et al CMJ 2006

Congenital Long QT Syndrome (LQTS)
長QT綜合症

怪病「長QT綜合症」 聲浪刺激心亂跳

鬧鐘響 電話鈴 嚇死人

鬧鐘或電話突然響起會隨時「嚇到冇命」？瑪嘉烈醫院心臟科專科醫生指出，由基因病變引起的罕見疾病「長 QT 綜合症」，患者「唔嚇得」，否則會突然心跳紊亂，該院十年來僅發現六宗個案。此症更有五成機會遺傳給家人，有女患者由母親至姨甥女共三代齊中招，其母當年被鬧鐘響聲刺激至嚴重休克。母親去世後，整家接受基因測試才揭發多名家族成員受遺傳。

張女士憶述，其母自二十多歲起已間中突然暈低，僅靠掃鼻油解決問題，一直不知病因。

三代中招歸喪命

至〇五年晚上，張母在家中突然被鬧鐘聲嚇至暈倒送院，醫生發現她心跳不正常，腦部功能僅餘約百分之一，猶如「植物人」。張母其後轉送至療養院，半年後因併發肺炎去世。

張女士說，當時醫生已安排她及妹妹抽血及做心電圖測試，結果發現兩妹妹的心跳 QT 波偏長，是長 QT 綜合症的症狀，需服用減慢心跳的藥物。

張女士擔心此症遺傳給下一代，多次查問醫生如何檢測，去年獲轉介至瑪嘉烈醫院進行基因測試，其子幸無被遺傳，但妹妹腹中懷有一對孖囡都「中招」。早產出生的細孖，在三十二周時已發病，心跳加速，服藥也未能控制病情，要安裝心臟起搏器，病情始受控。

患病率五千分一

瑪嘉烈醫院心臟科專科醫生莫毅成表示，約每五千人才有一人患長 QT 綜合症，他們不能受刺激，常被誤會性格「腩癌」膽小，外國有一成猝死嬰兒與長 QT 綜合症有關，他指，患者的心臟結構正常，但心電圖的 QT 波較常人長，做運動、受驚嚇或情緒激動時最易病發。該院十年內曾進行二十三宗遺傳性心律不齊病症的基因研究，當中六宗屬長 QT 症，病人分別曾出現兩次家族猝死病史及致命性心律不齊。

QT 是心電圖中顯示的兩種心電波「Q 波」及「T 波」。香港先天及結構性心臟病學會會長鄭長華解釋，心臟中的傳電細胞負責傳遞電流訊號，讓心臟得以規律跳動。每次細胞通電及傳電，均在心電圖中呈現一個 QT 波，若每次 QT 波之間相隔過長，即所謂「長 QT」，可致心跳電流紊亂，容易造成心房顫動甚至猝死。

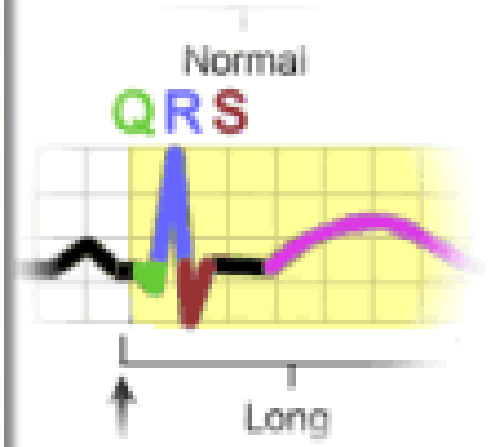
正常人 QT 波間隔不多於零點四六秒，高於此數即屬異常。由先天性遺傳基因變異引起的長 QT 症十分罕見，絕大部分病例由後天因素引發，例如體內電解質或酵質膠降等。另外，不少藥物也可致長 QT，如心律不整藥物、精神科藥物，同時服用某胃藥及抗生素等。若醫生發現病人出現長 QT，一般先仔細詢問病人服藥狀況及家族病史，認為極可能屬遺傳性個案才轉介基因測試。

鄭長華又指，常用的一分鐘心電圖檢查未必可準確檢測長 QT 綜合症，部分先天性遺傳個案或在特定環境才會病發，例如突然受驚或極度緊張等。醫生會在嚴密監察及十分安全的病房環境中，向病人靜脈注射「緊張質爾蒙」，模擬受驚狀態，再同步觀察 QT 波有否異常。

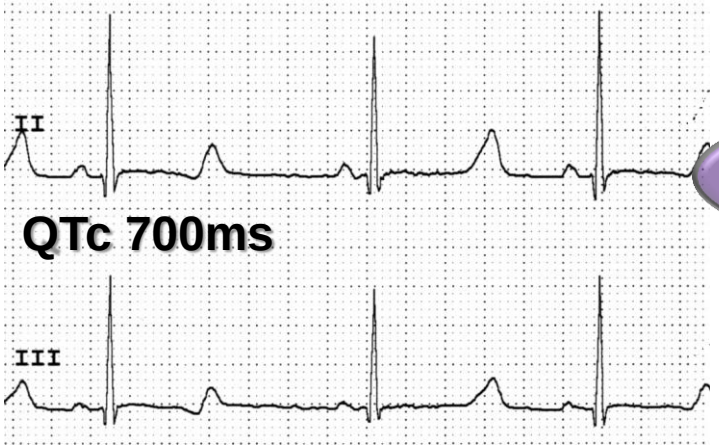
●參加基因測試很重要，測試結果必須準確無誤。

●張女士與家人接受基因測試後，發現三代均有家人遺傳長 QT 綜合症。

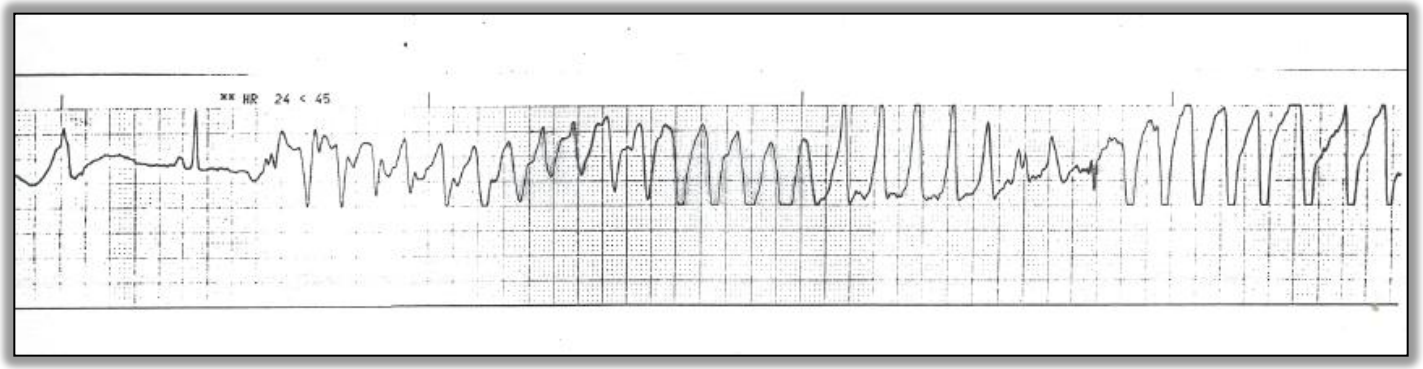
Auditory stimuli triggers SCD in a long QT family



Congenital Long QT Syndrome (LQTS)
長QT綜合症

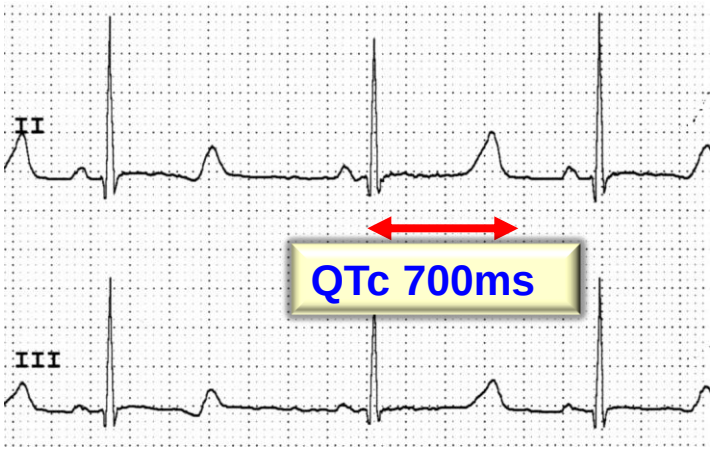


Prolonged QT interval on ECG

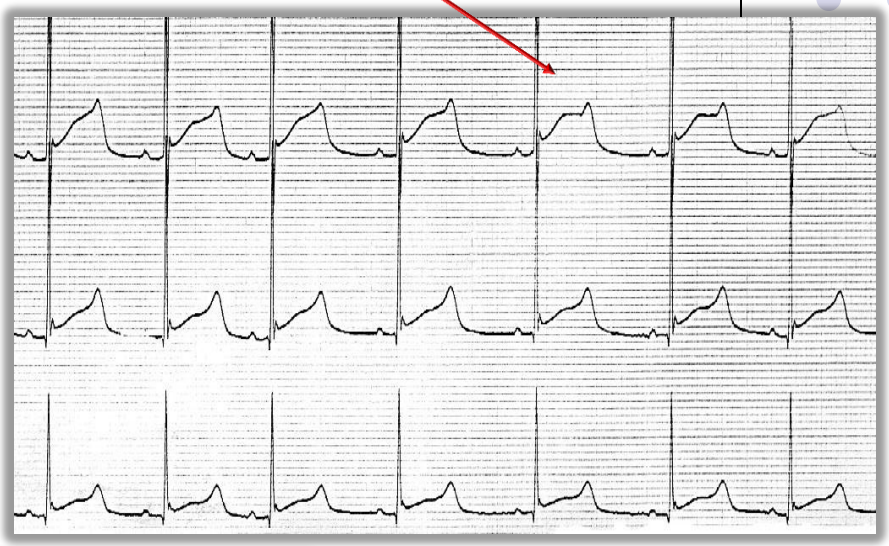


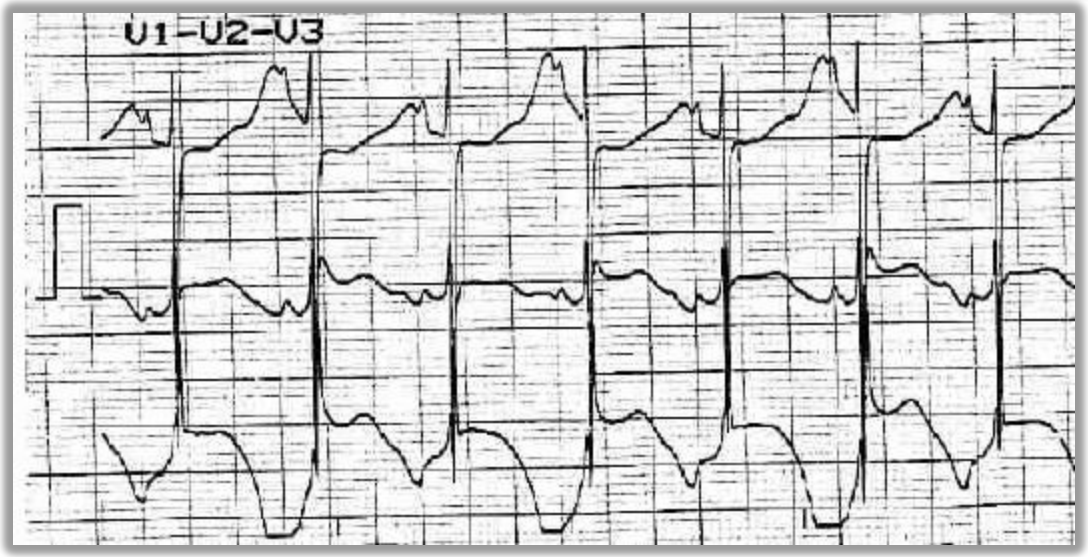
Torsade de pointes

ECG abnormalities in LQTS



Notched T waves





T wave alternans

Diagnostic criteria of LQTS

Schwartz Score

≤ 1 point – low probability

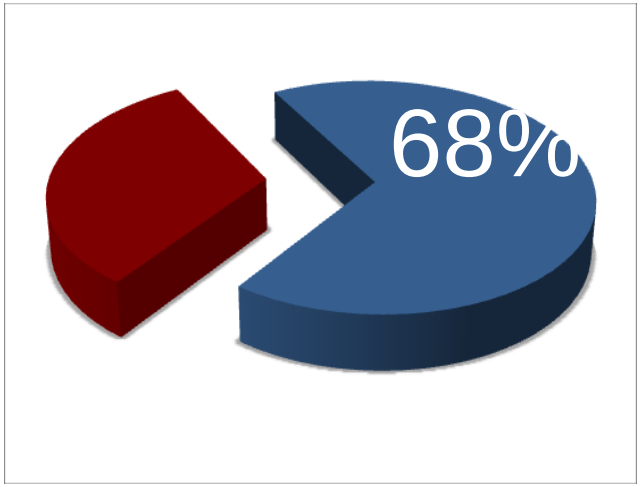
>1-3 points – intermediate proba

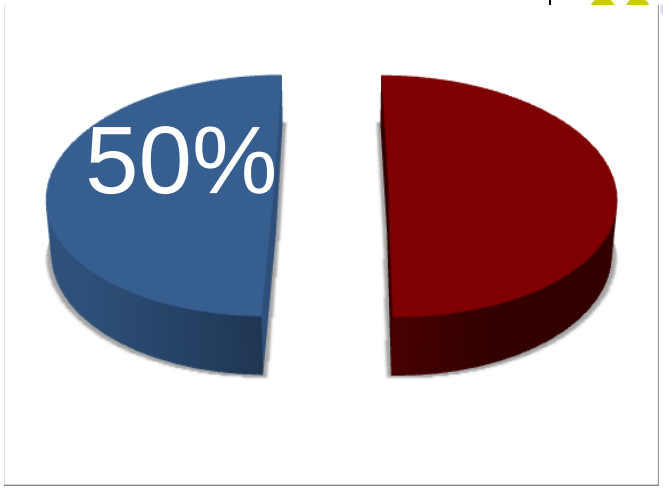
≥ 3.5 points – high probability of LQT (revised 2006)

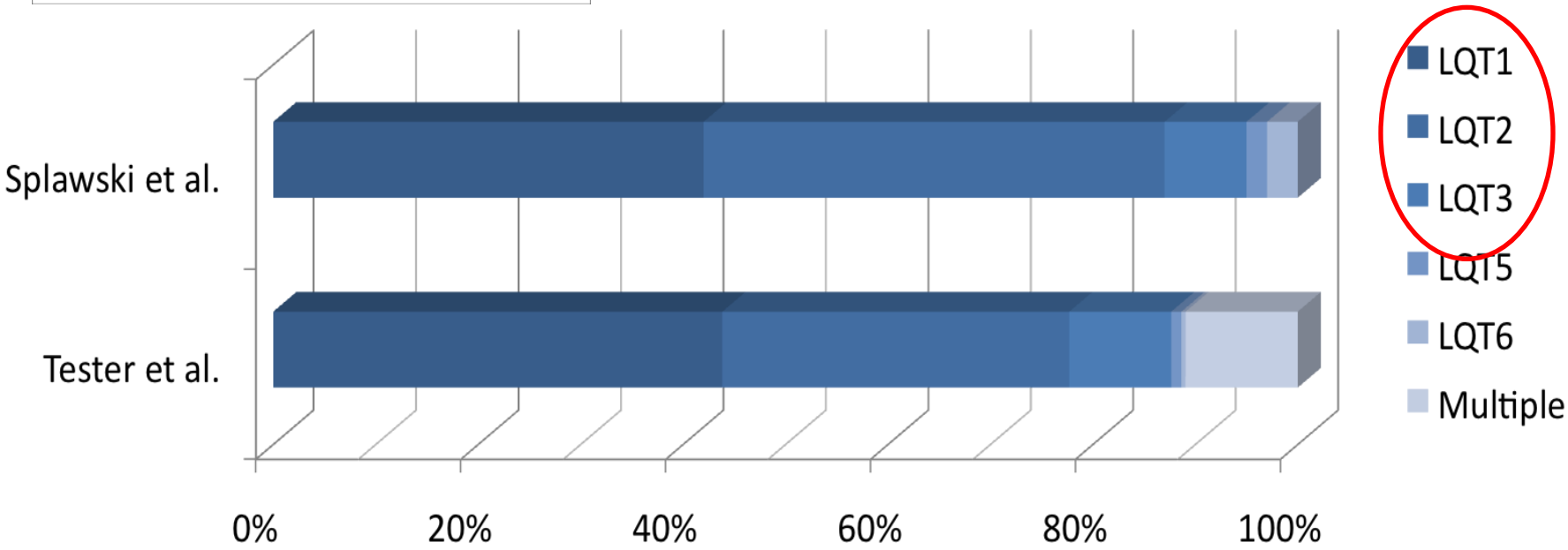
Dr. Peter Schwartz visiting Hong Kong in 2011

Schwartz PJ 1993

Genotypes in index patients with LQTS



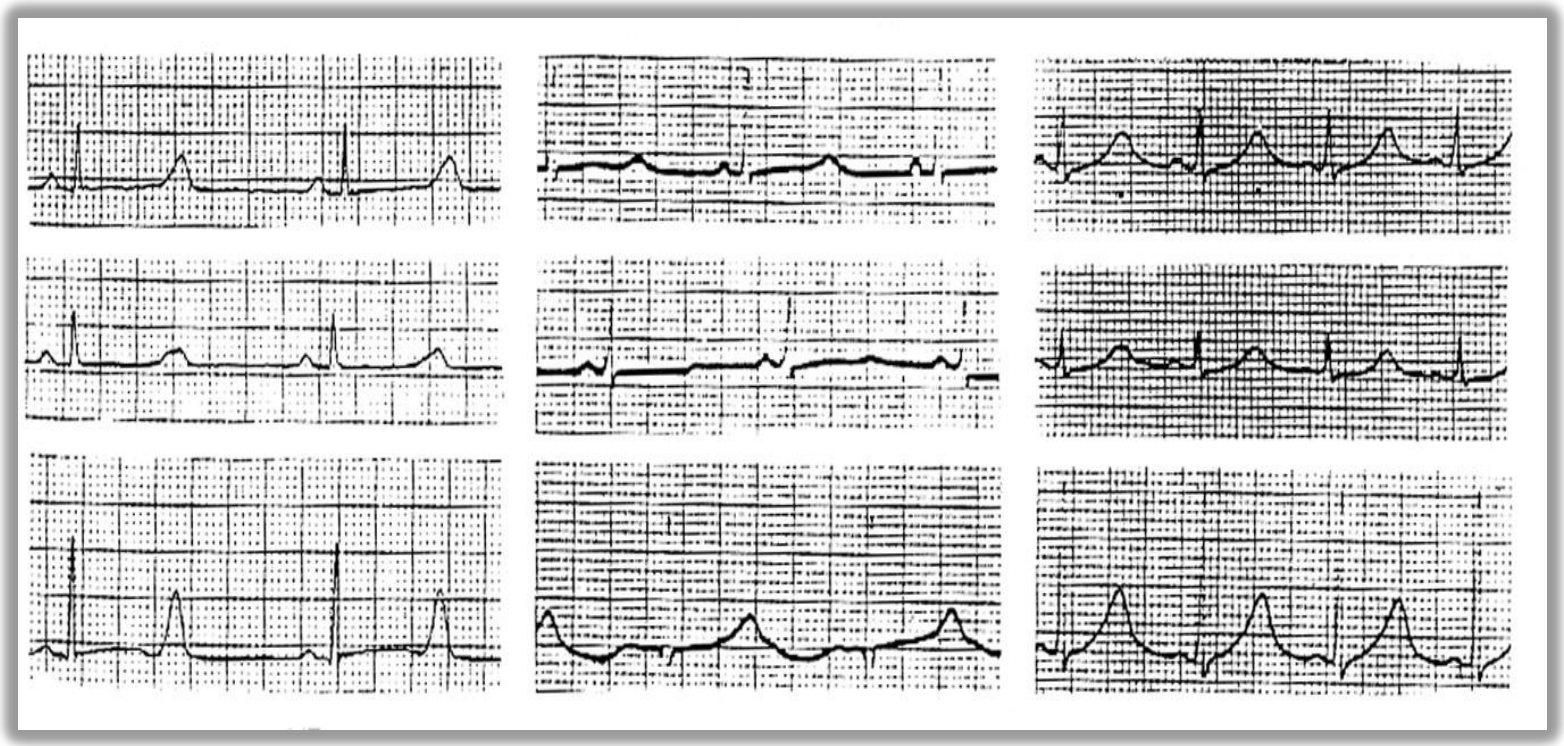




Splawski et al. Circ 2000; Tester et al. JACC 2006

Genotype-Phenotype correlation - ECG T-wave patterns

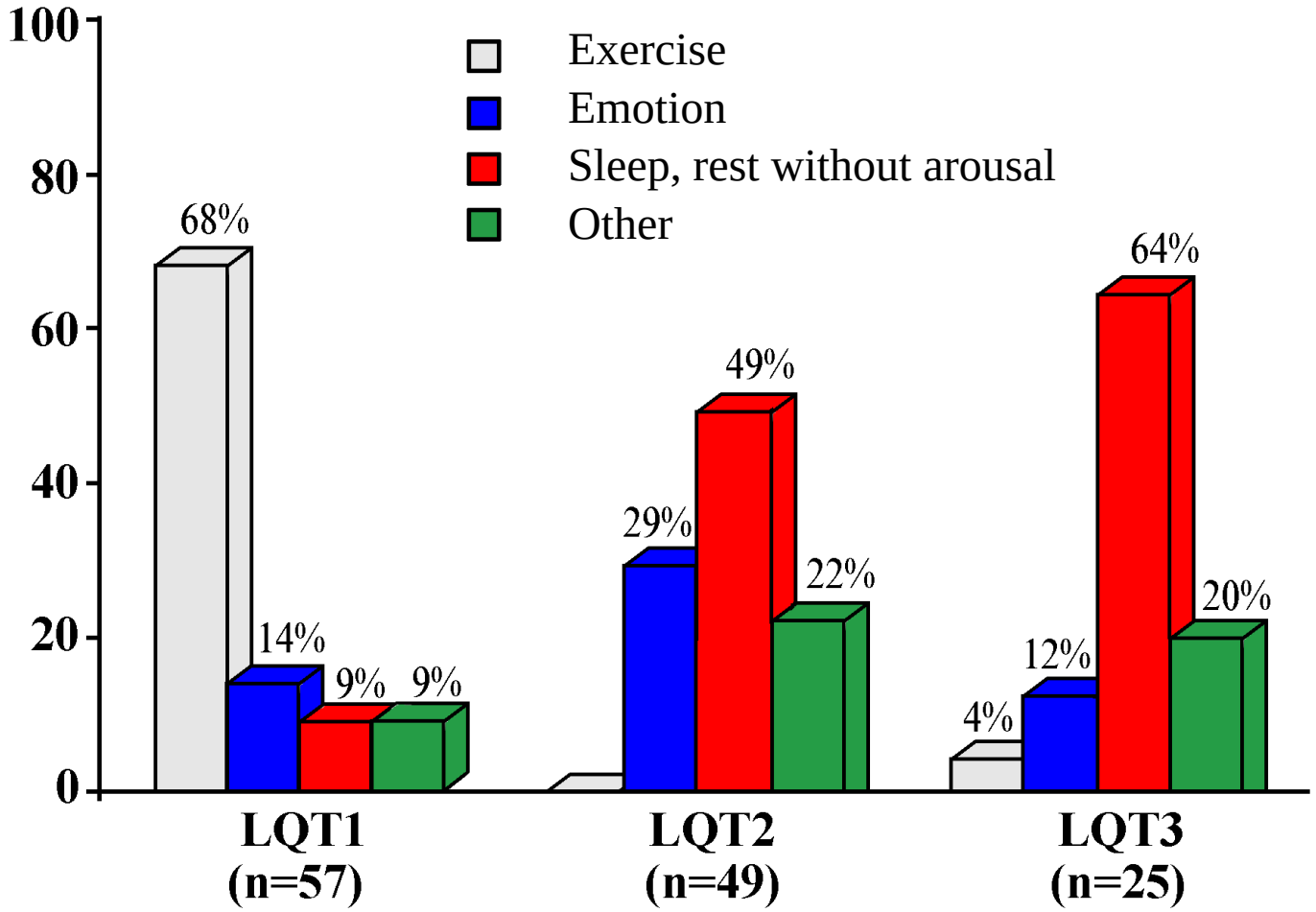
LQT3	LQT2	LQT1
SCN5A	***KCNH2***	***KCNQ1***
Chromosome 3	Chromosome 7	Chromosome 11



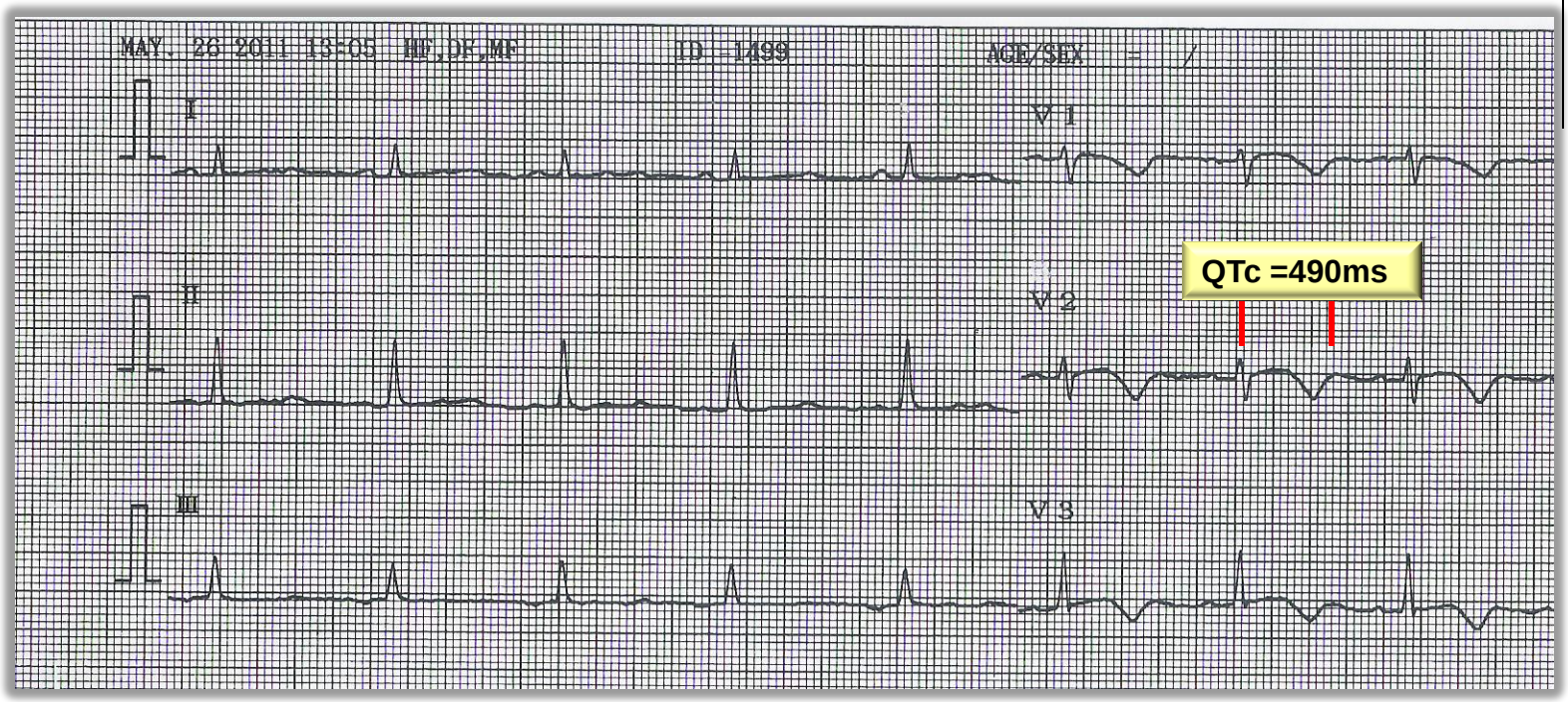
Moss AJ et al. Circulation 1995;92:2929-2934

Genotype-Phenotype Correlation

- Triggers for Lethal Cardiac Events in Different Genotypes



F/30, asymptomatic
Mother with confirmed LQTS
QTc 490ms



Schwartz Score

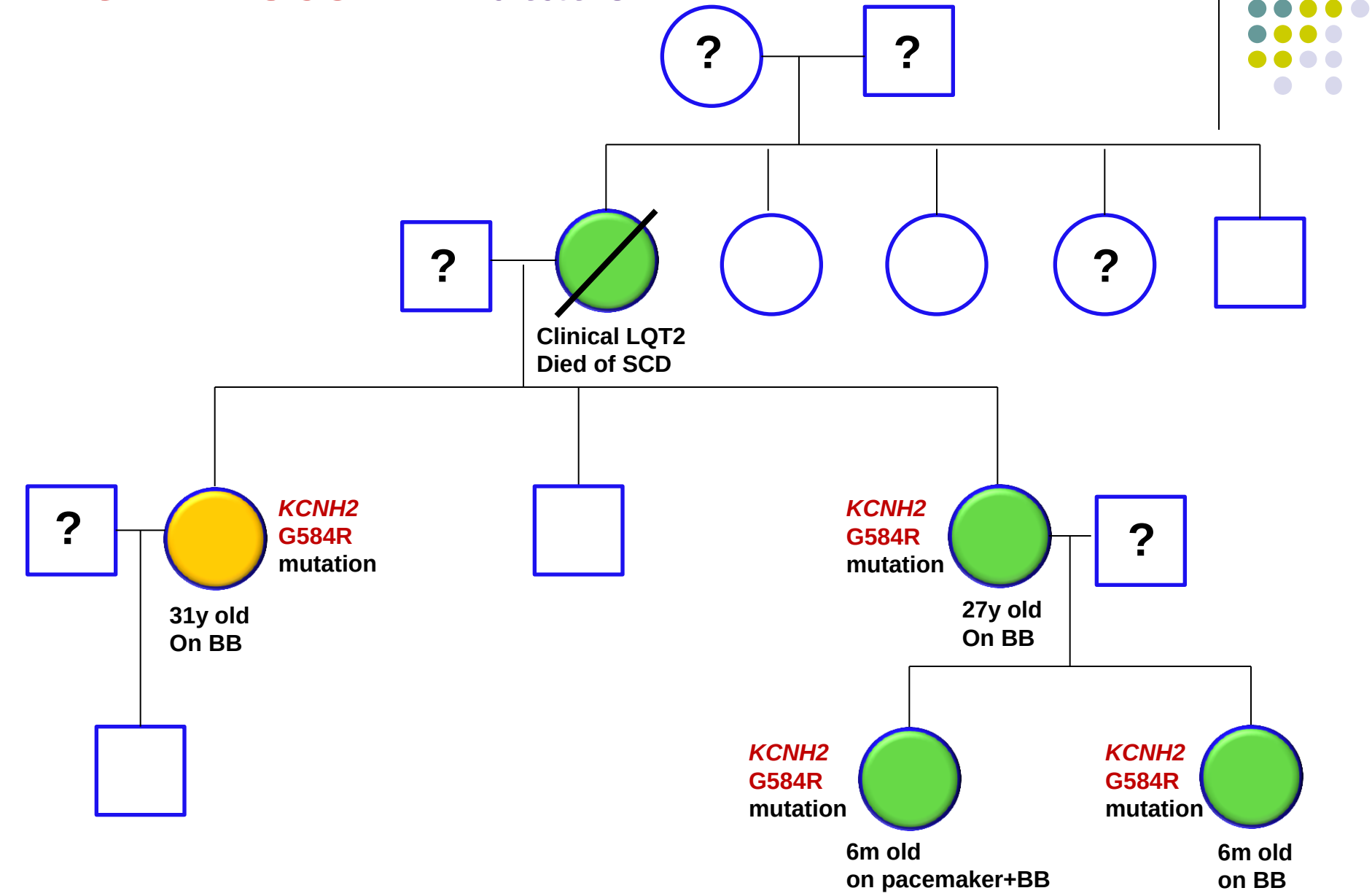
QTc 490ms = **3**

Family hx of LQTS = **1**

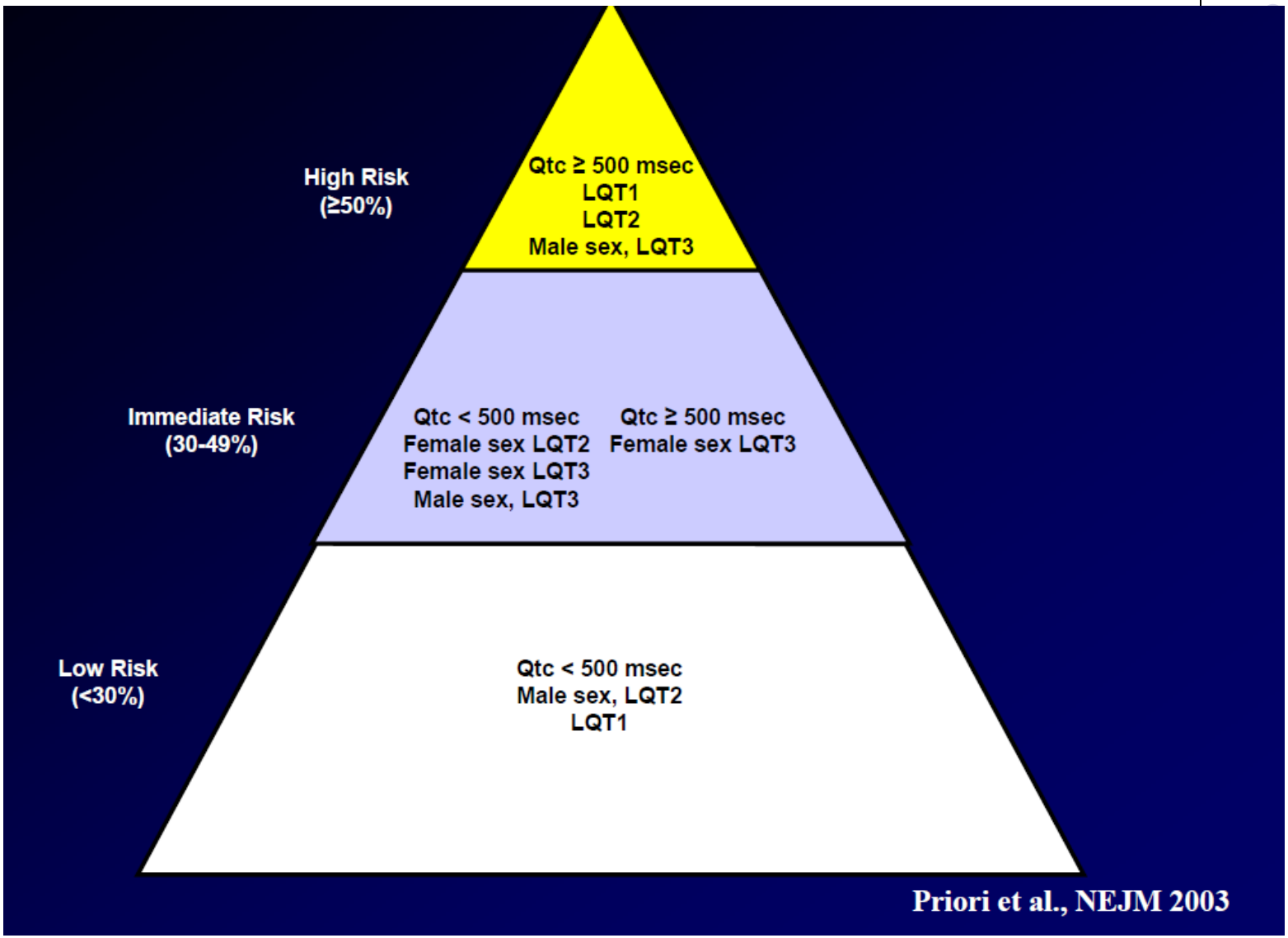
Total = **4** (> 3.5 - high probability of LQTS)

Genetic study : ***KCNH2*** G584R mutation (LQT2)

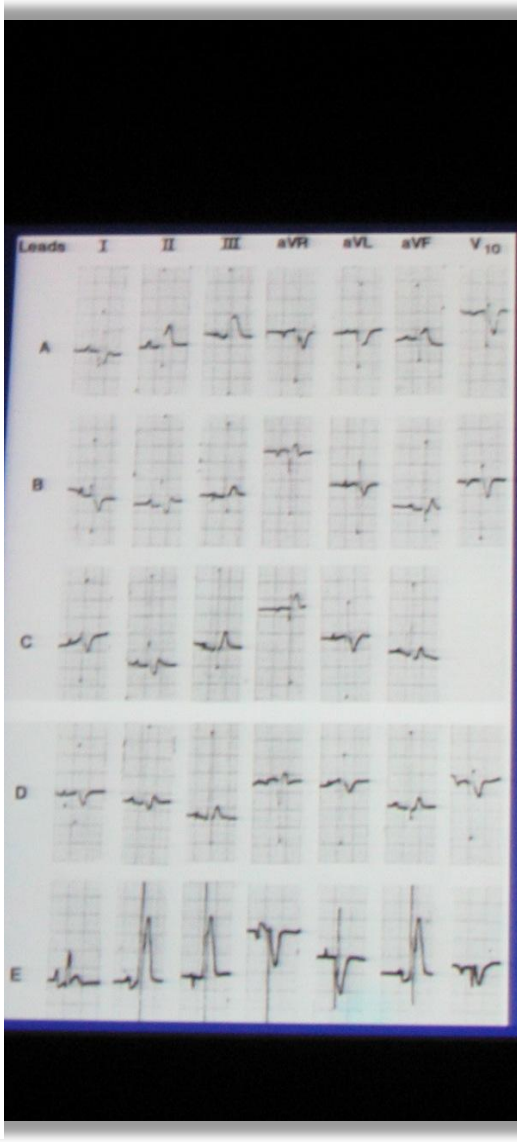
Genetic testing for family screening of *KCNH2* G584R mutation



Long QTc & LQT3 genotype – Bad prognostic indicators in LQTS

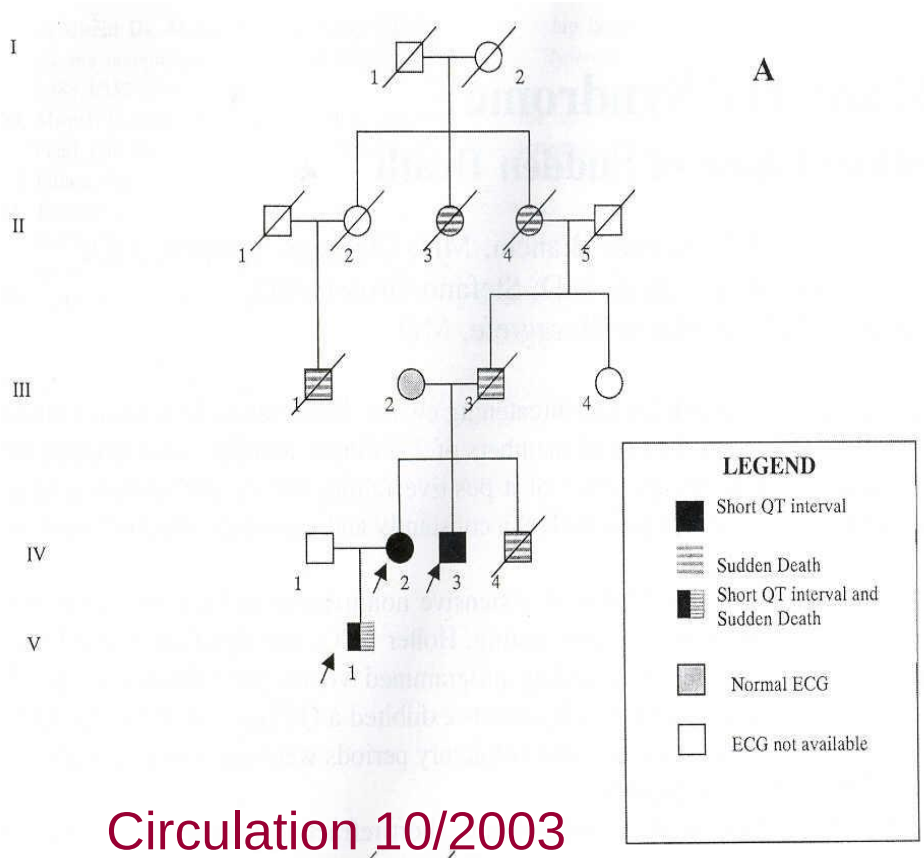


Short QT interval in Eastern Grey Kangaroos



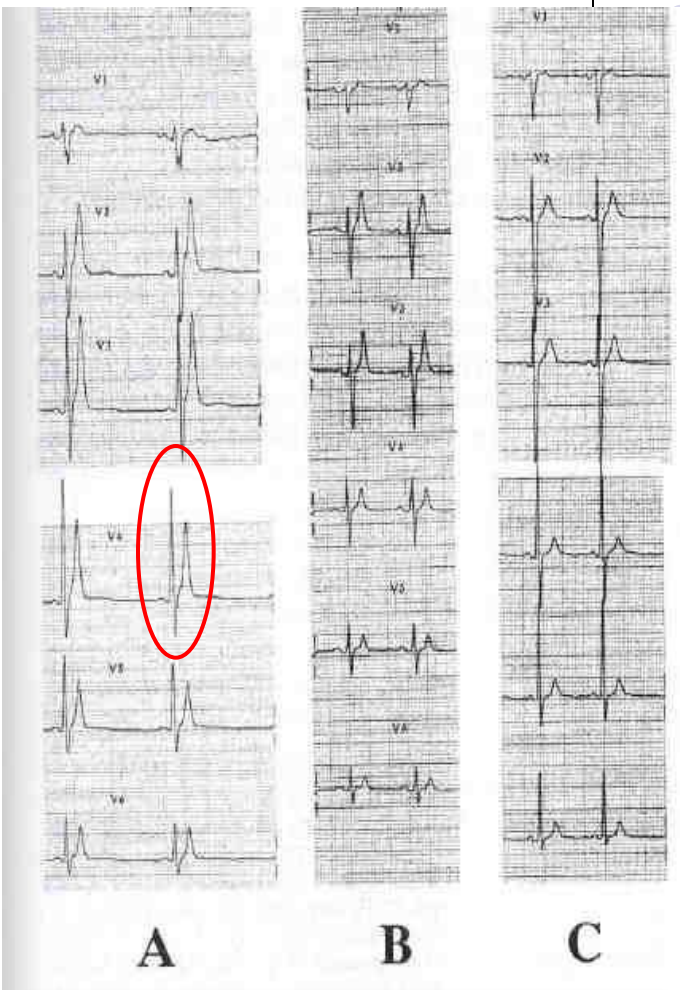
Rezakhani et al
Austr Vet J 1986

Short QT Syndrome (SQTS)



Circulation 10/2003

Family pedigree – SCD associated with Short QT interval on ECG



QTc < 300ms, QT < 280ms

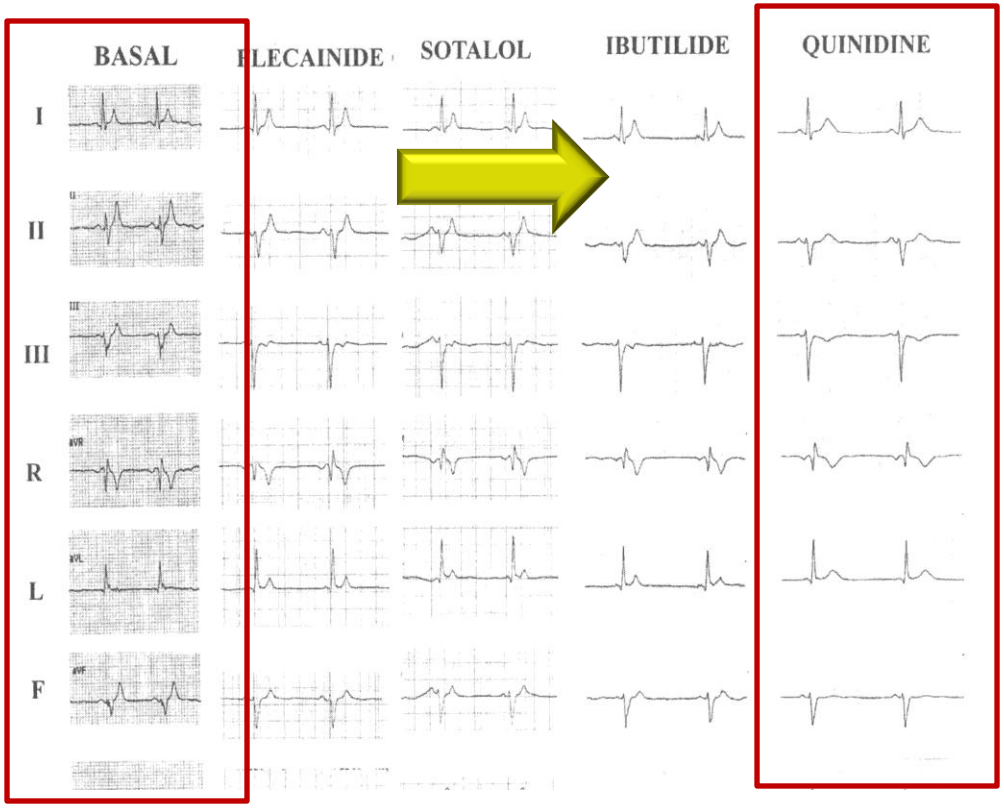
Quinidine in SQTS

Short QT Syndrome: Pharmacological Treatment

JACC April 2004

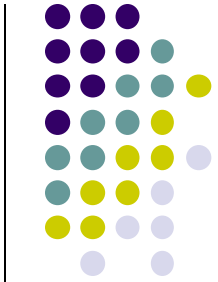
Fiorenzo Gaita, MD,* Carla Giustetto, MD,* Francesca Bianchi, MD,* Rainer Schimpf, MD,†
Michel Haissaguerre, MD,‡ Leonardo Calò, MD,§ Ramon Brugada, MD,‖ Charles Antzelevitch, PHD,¶
Martin Borggrefe, MD,† Christian Wolpert, MD†

Asti and Rome, Italy; Mannheim, Germany; Bordeaux-Pessac, France; and Utica, New York



Quinidine prolonged QT interval in patients with SQTS & rendered VF non-inducible

ECG evaluation in SCD prevention



Secondary Prevention

- Survivor of SCD

Primary Prevention

- Family hx of SCD / premature death / inherited arrhythmogenic diseases
- Unexplained syncope / convulsion
- Consistent or unusual chest pain / SOB during exercise

? Pre-participation ECG screening for athletes

ECG preparticipation screening for competitive athletes

- Italian study found 89% reduction of SCD in screened athletes from 1979 to 2004
- Recommended by ESC, International Olympic Committee, most European Cardiologic Societies (but not AHA)
- Universal pre-participation ECG screening of young competitive athletes still controversial

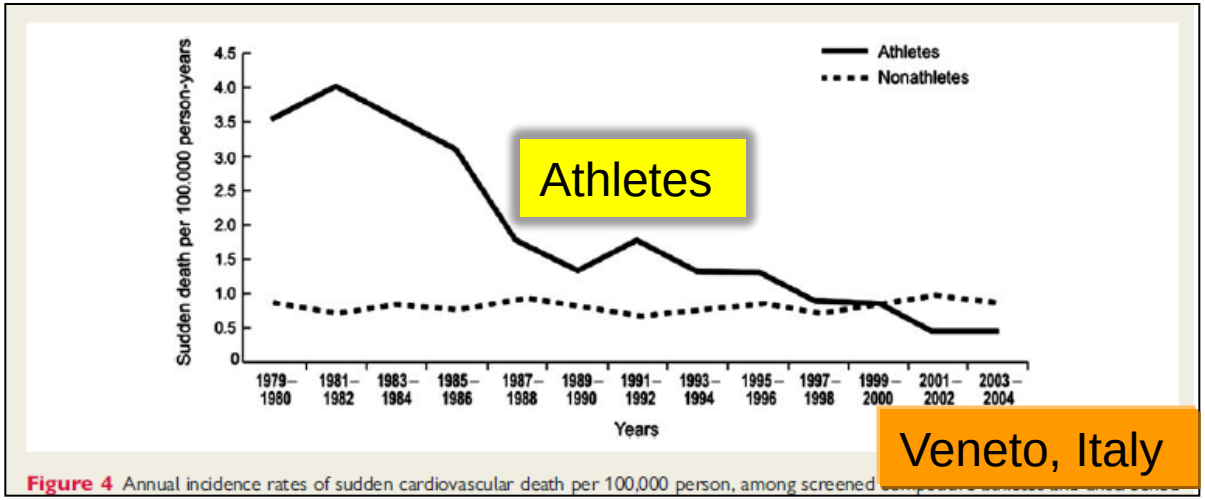
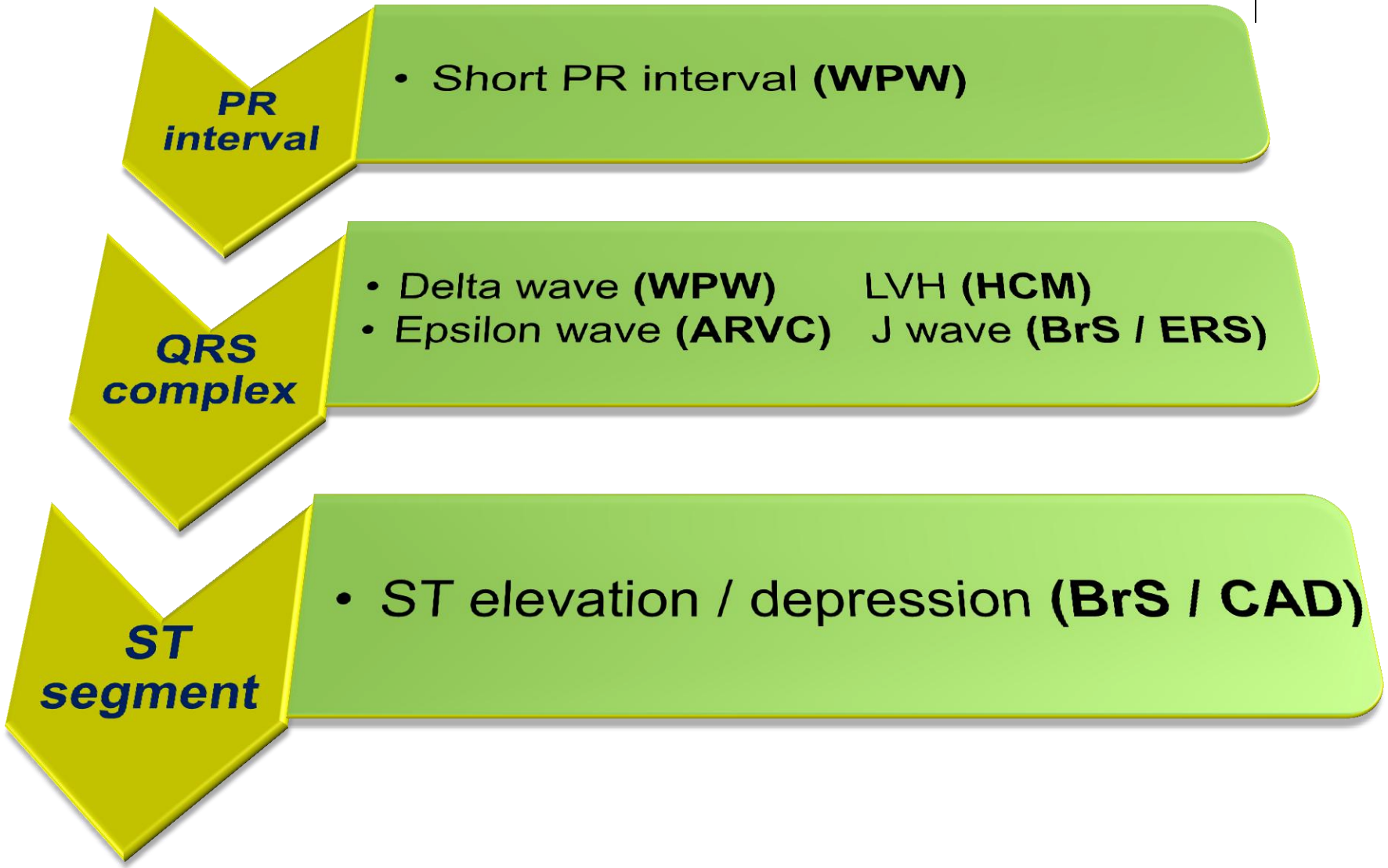


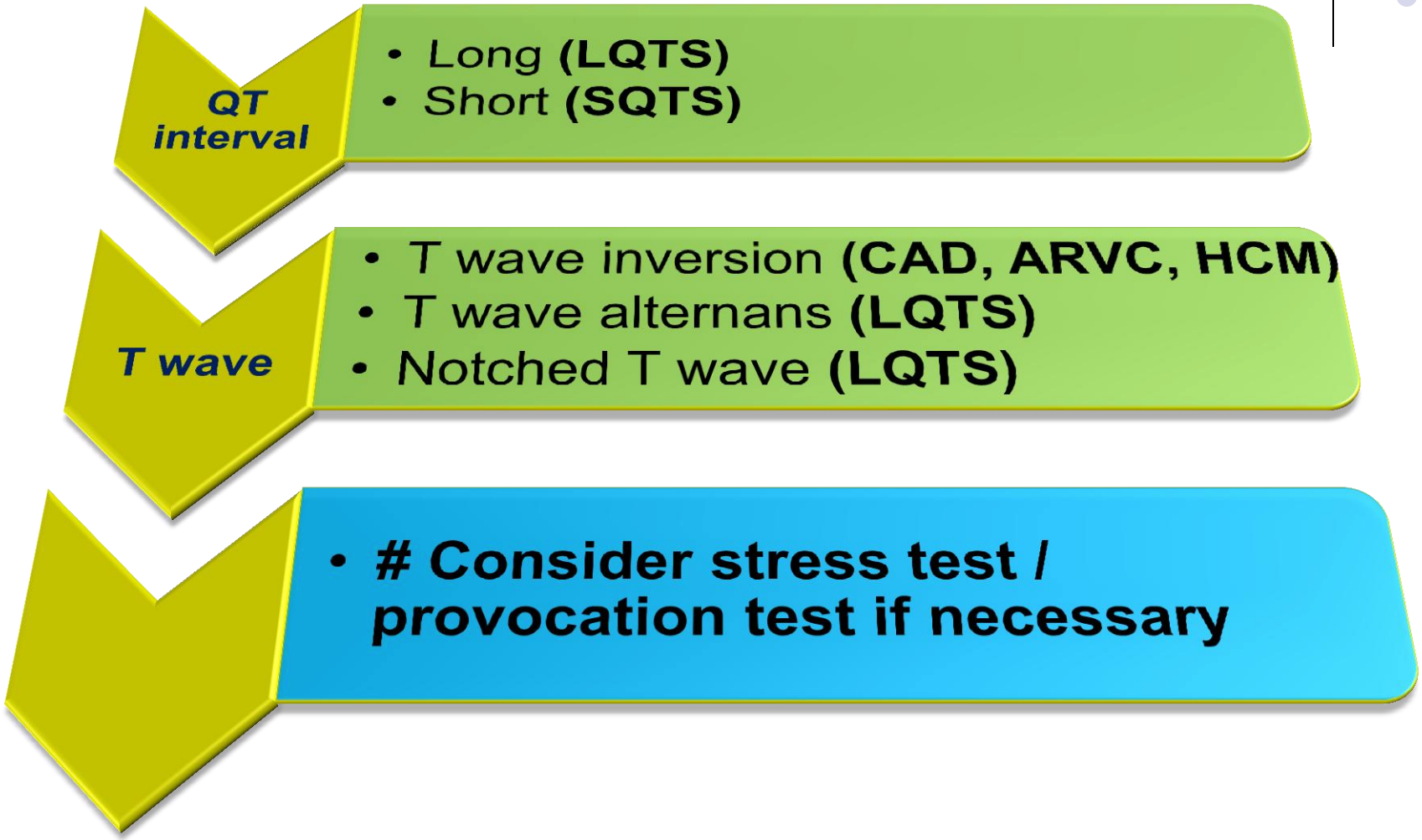
Figure 4 Annual incidence rates of sudden cardiovascular death per 100,000 person, among screened

Corrado M et al, EHJ, April 2011

Systematic evaluation of 12-lead ECG in subjects at risk of SCD

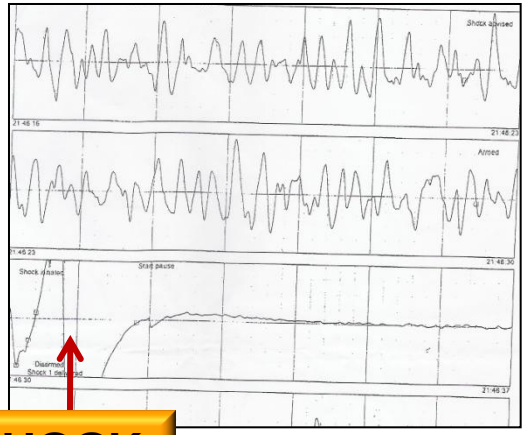


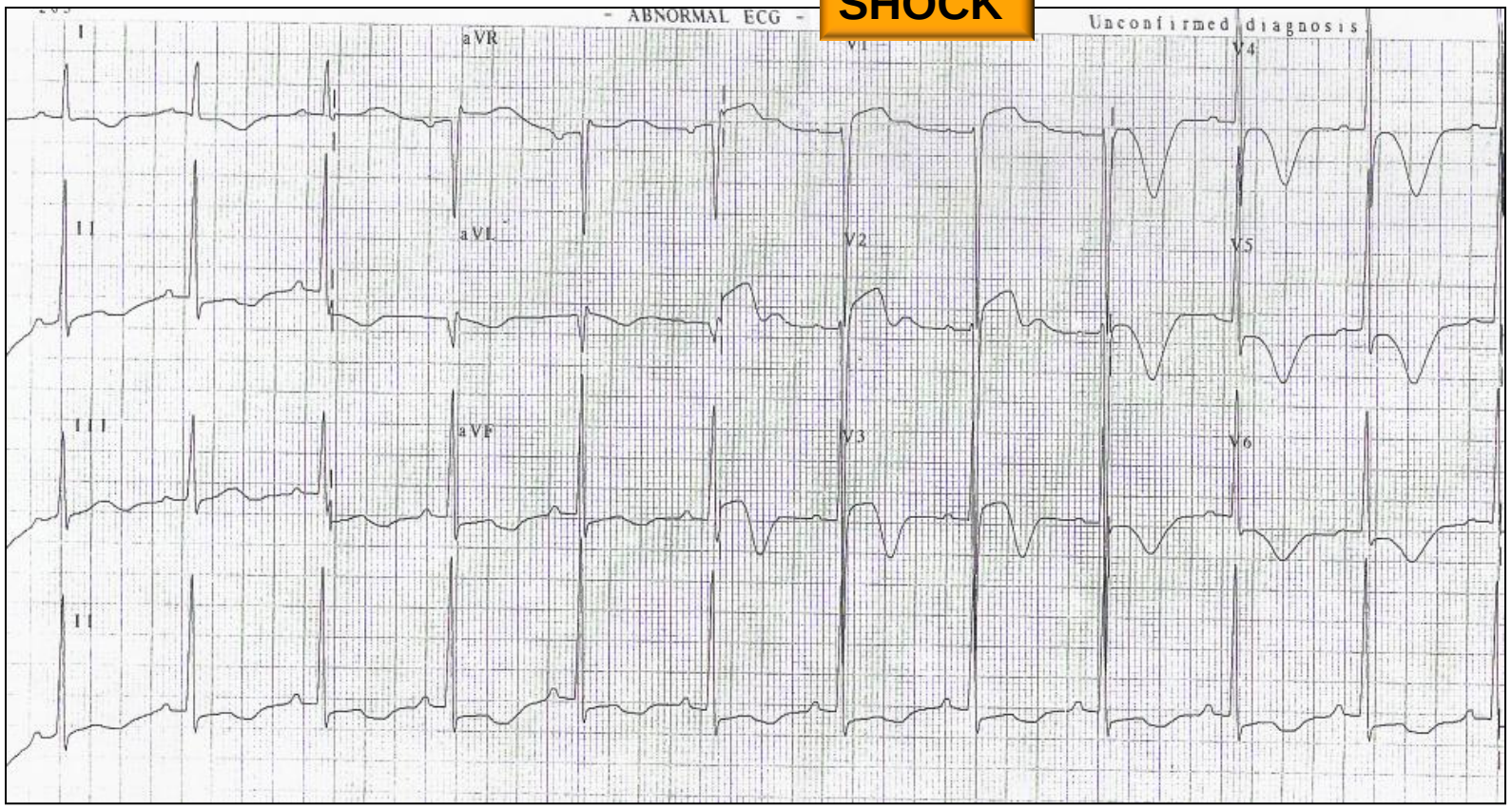
Systematic evaluation of 12-lead ECG



Case Scenario (1)

- M/18, professional soccer player
- Sudden collapse in a soccer match
- Documented VF, successfully defibrillated by AED





What is the most likely cause of his cardiac arrest ?

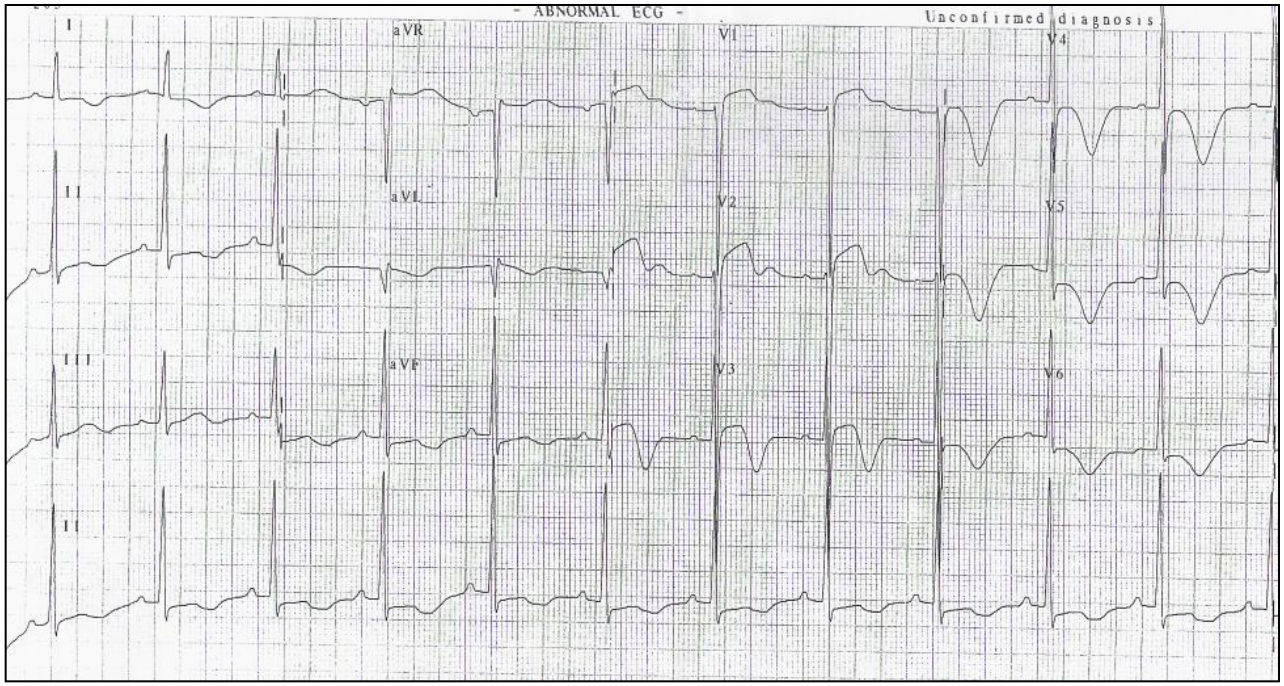
(A) HOCM

(B) LQT1

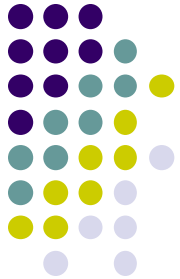
(C) CAD (congenital)

(D) ARVC

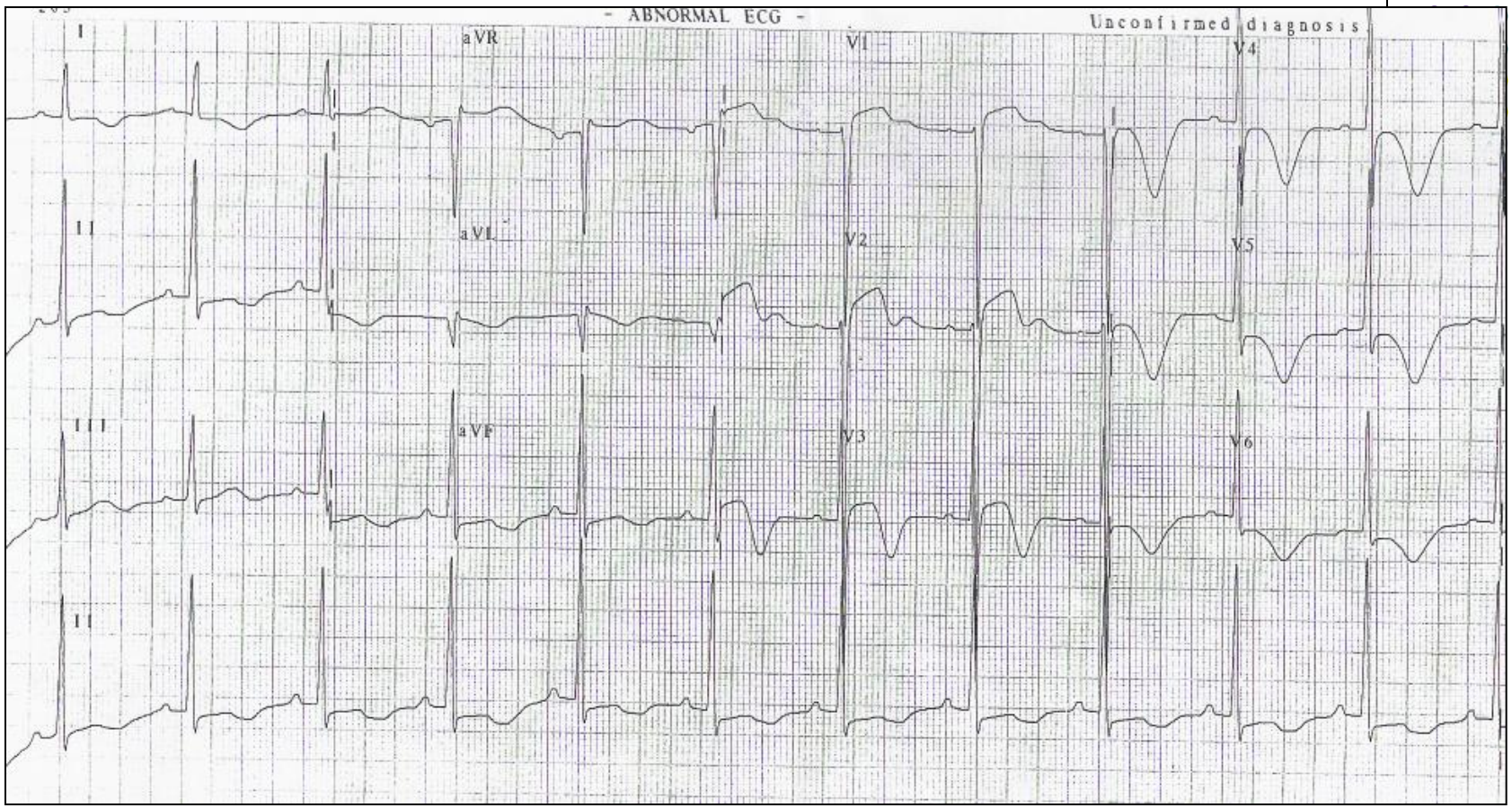
(E) CPVT



Cardiac arrest provoked by exercise in an adolescent



- HOCM (LVH with strain pattern)
- LQT1 (long QT interval + T wave changes)
- Congenital CAD (Normal ECG in SR)
- ARVC (Epsilon wave + T wave inversion on V1-3)
- CPVT (Normal ECG in SR)



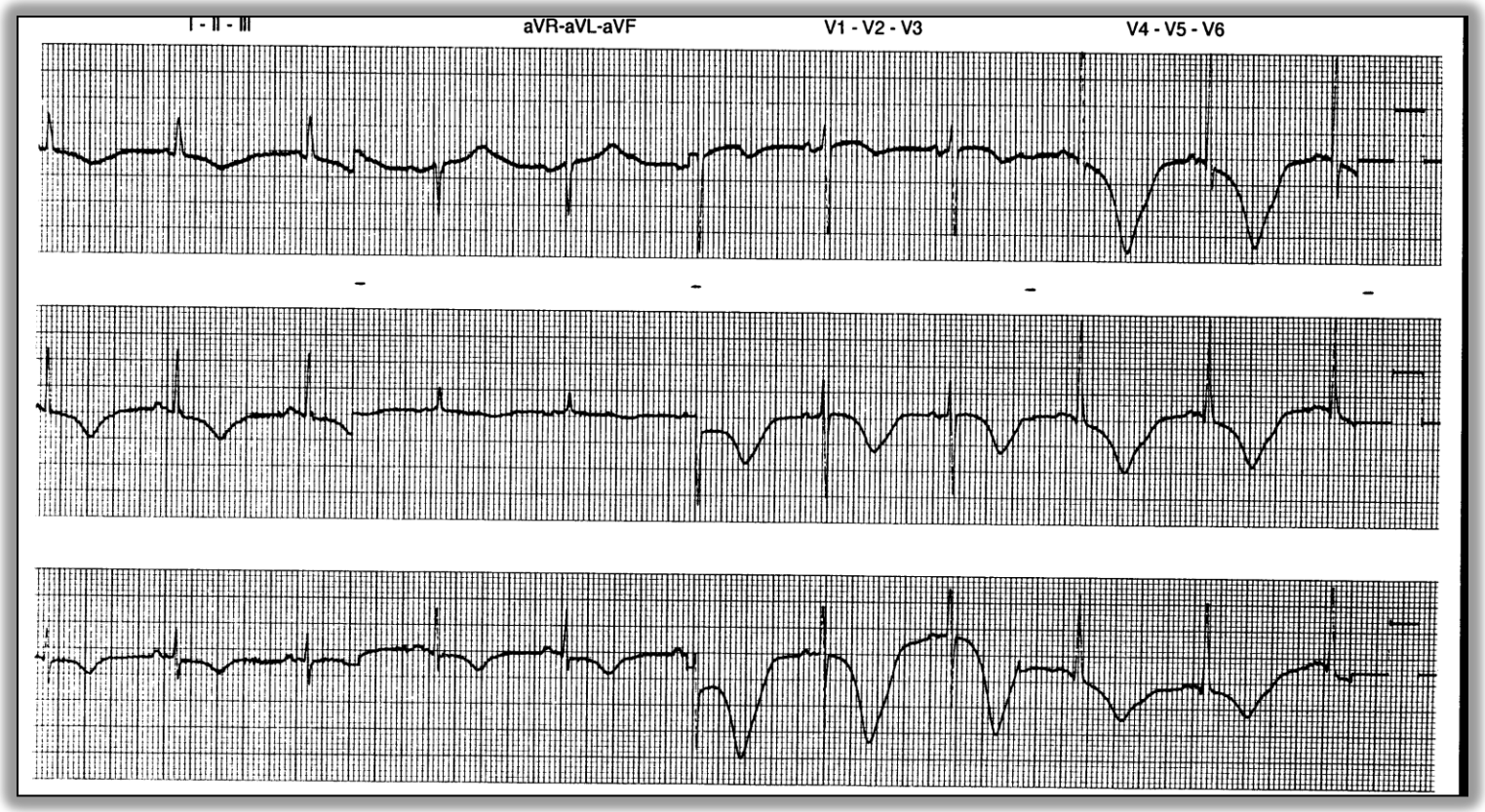
LVH with strain pattern

- SV1 + RV5 >35mm
- T wave inversion on precordial leads

Answer : A (HOCM)

Case Scenario (2)

- F/74, hx of HT
- Sudden collapse at home , remained comatose on admission



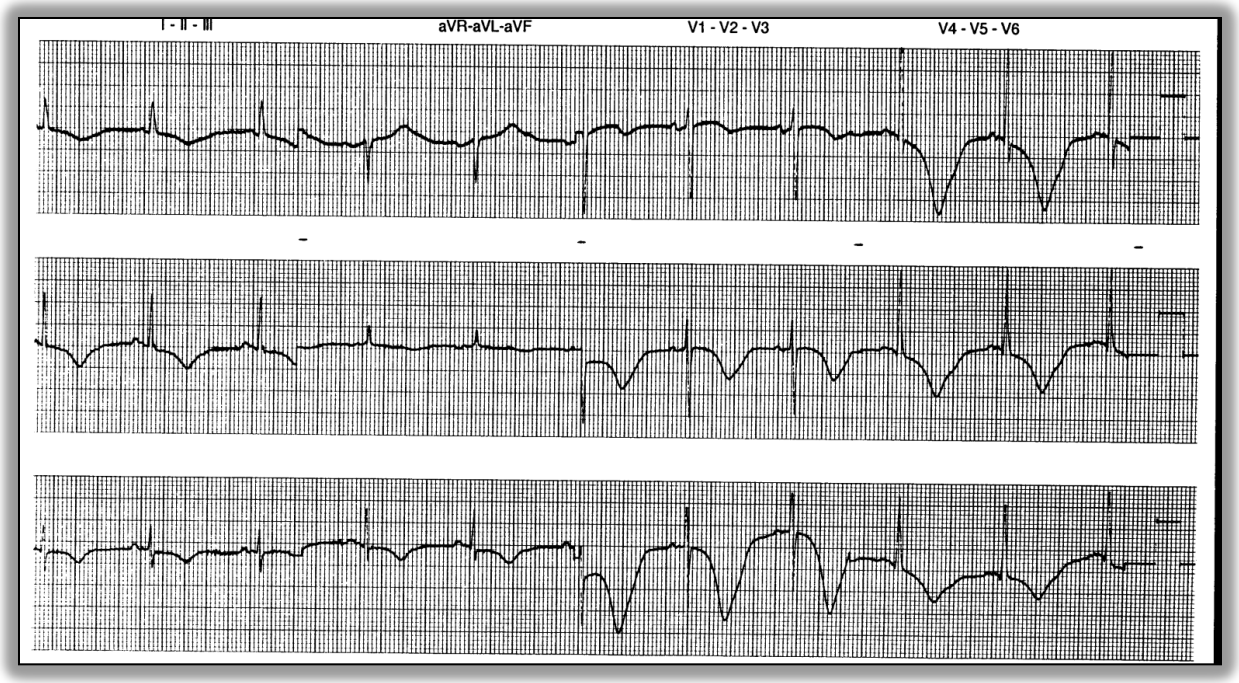
What is your next investigation ?

(A) Coronary Angiogram

(B) Echocardiogram

(C) CT Brain

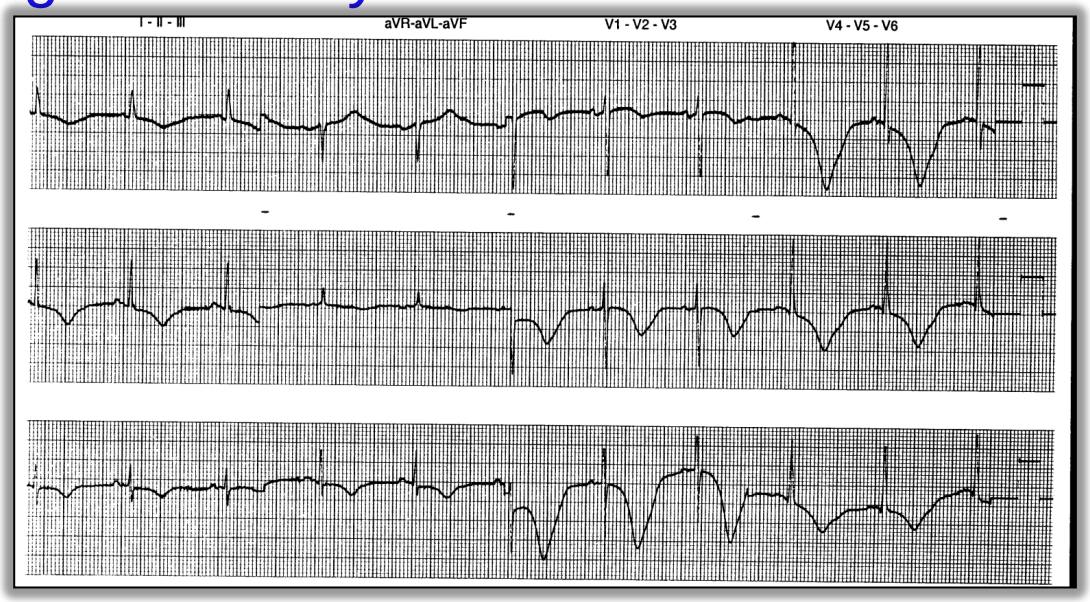
(D) Holter monitoring

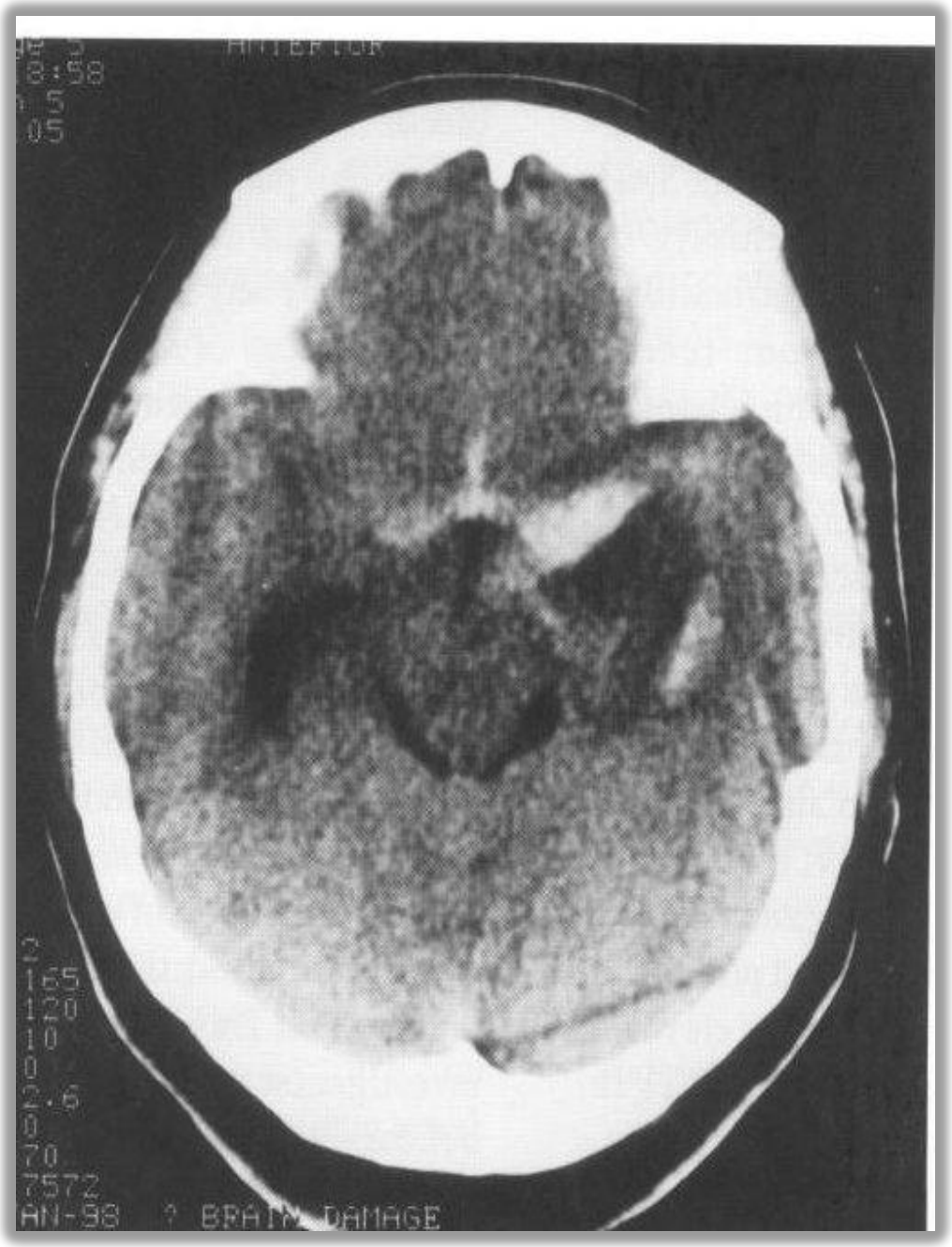


Subarachnoid Haemorrhage

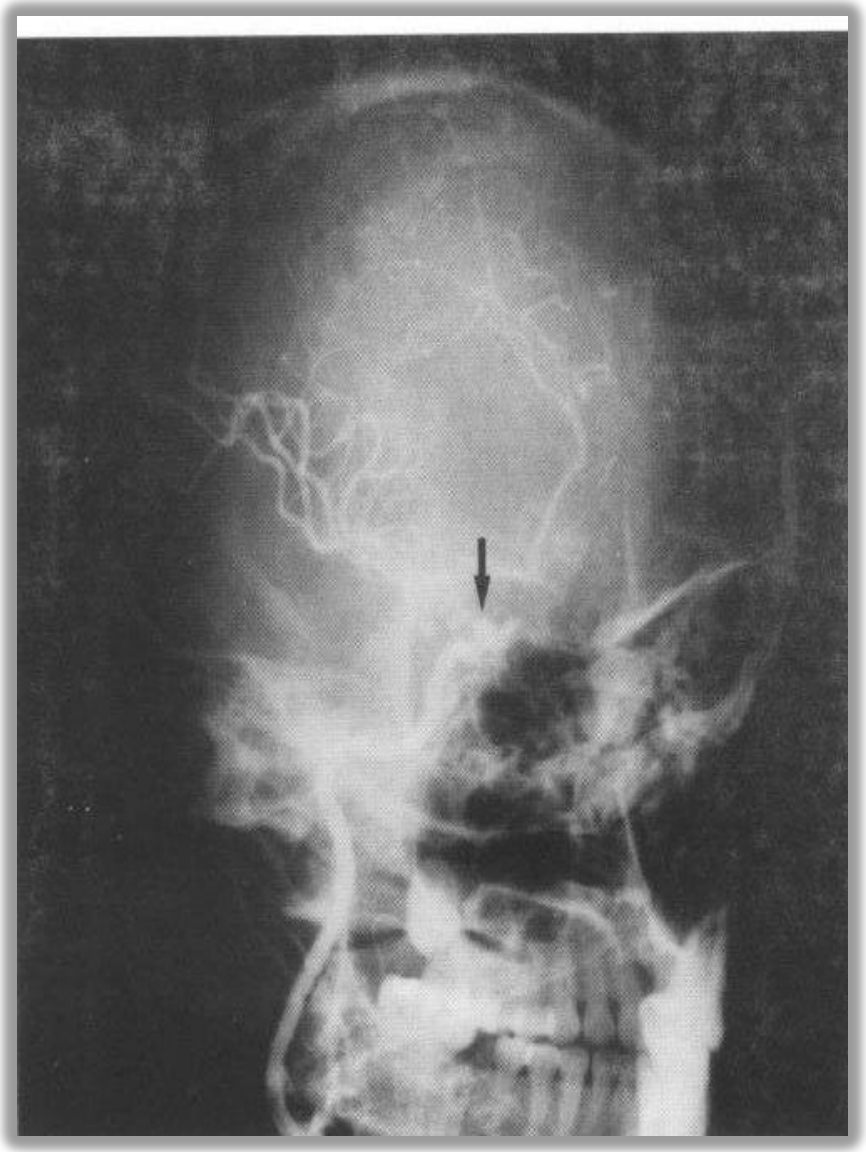
- SAH may result in abnormal ECG known as the ***CNS - T wave pattern*** (**or neurogenic pattern**)
  - Prolonged QT interval
  - Giant inverted T waves
- May present with VT / TdP leading to sudden death
  - Cardiac monitoring mandatory within first 48-72 hours

Answer : A (CT Brain)

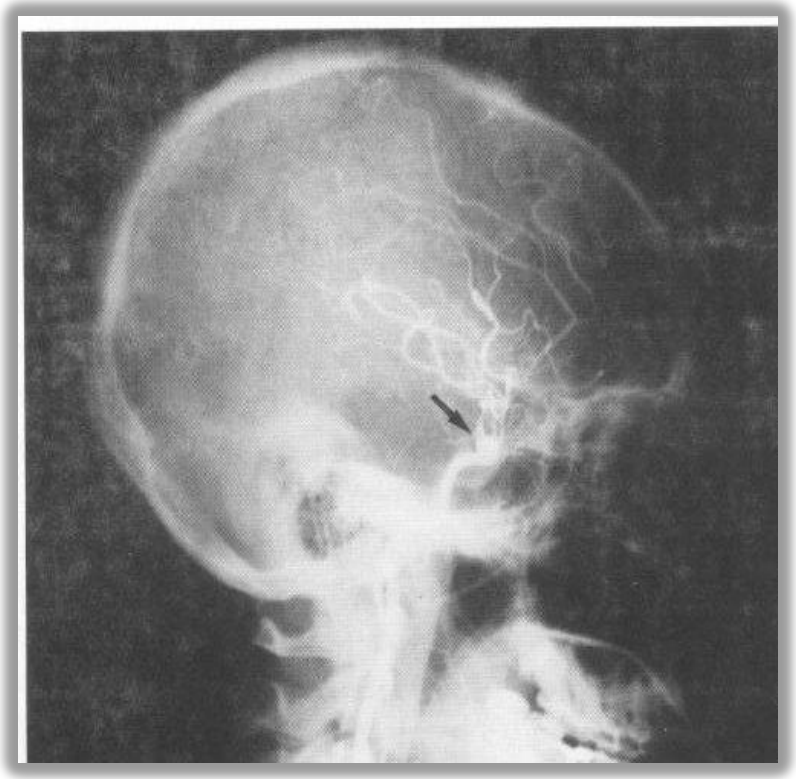




Subarachnoid Haemorrhage



Cerebral Aneurysm



VT recorded in ICU

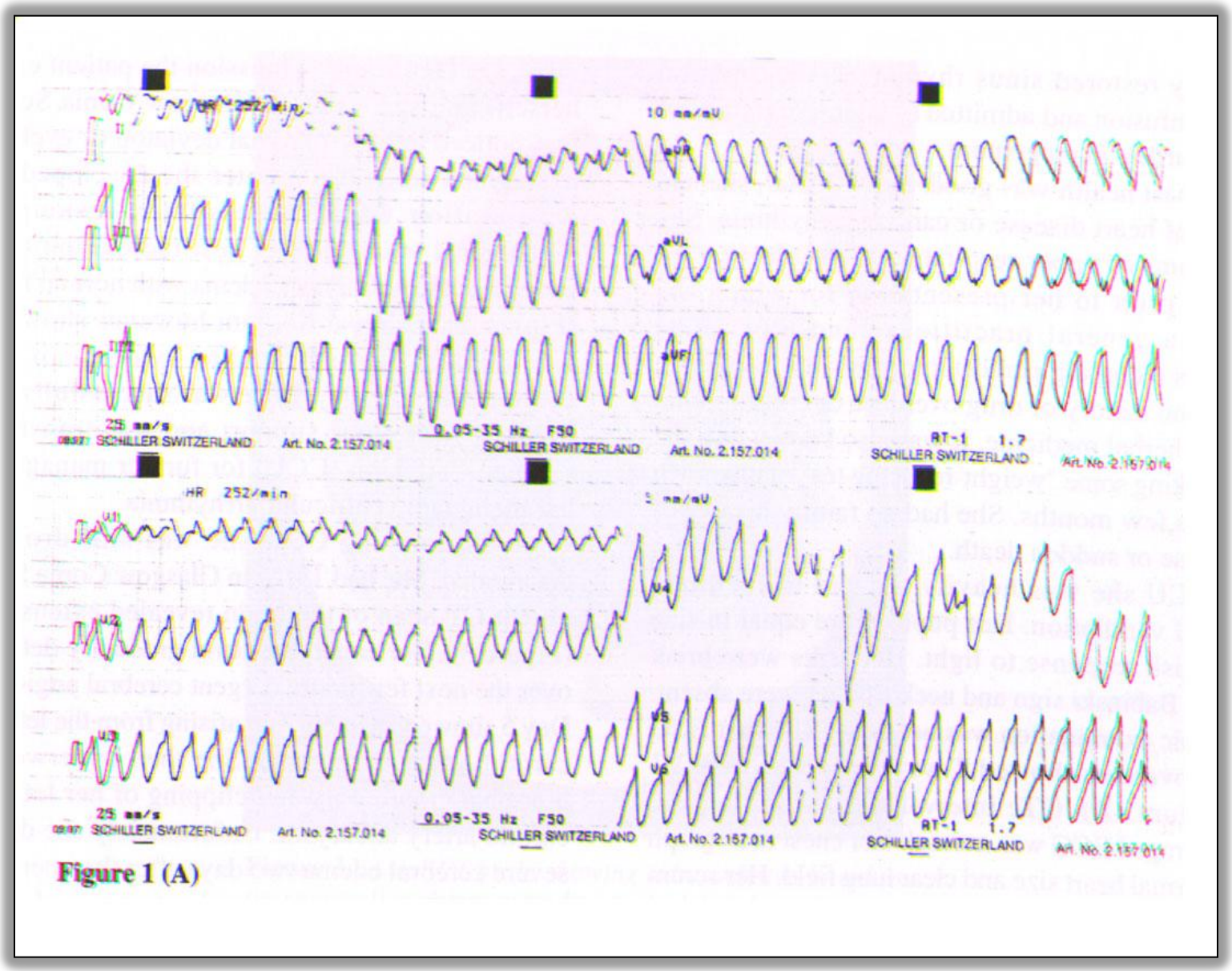
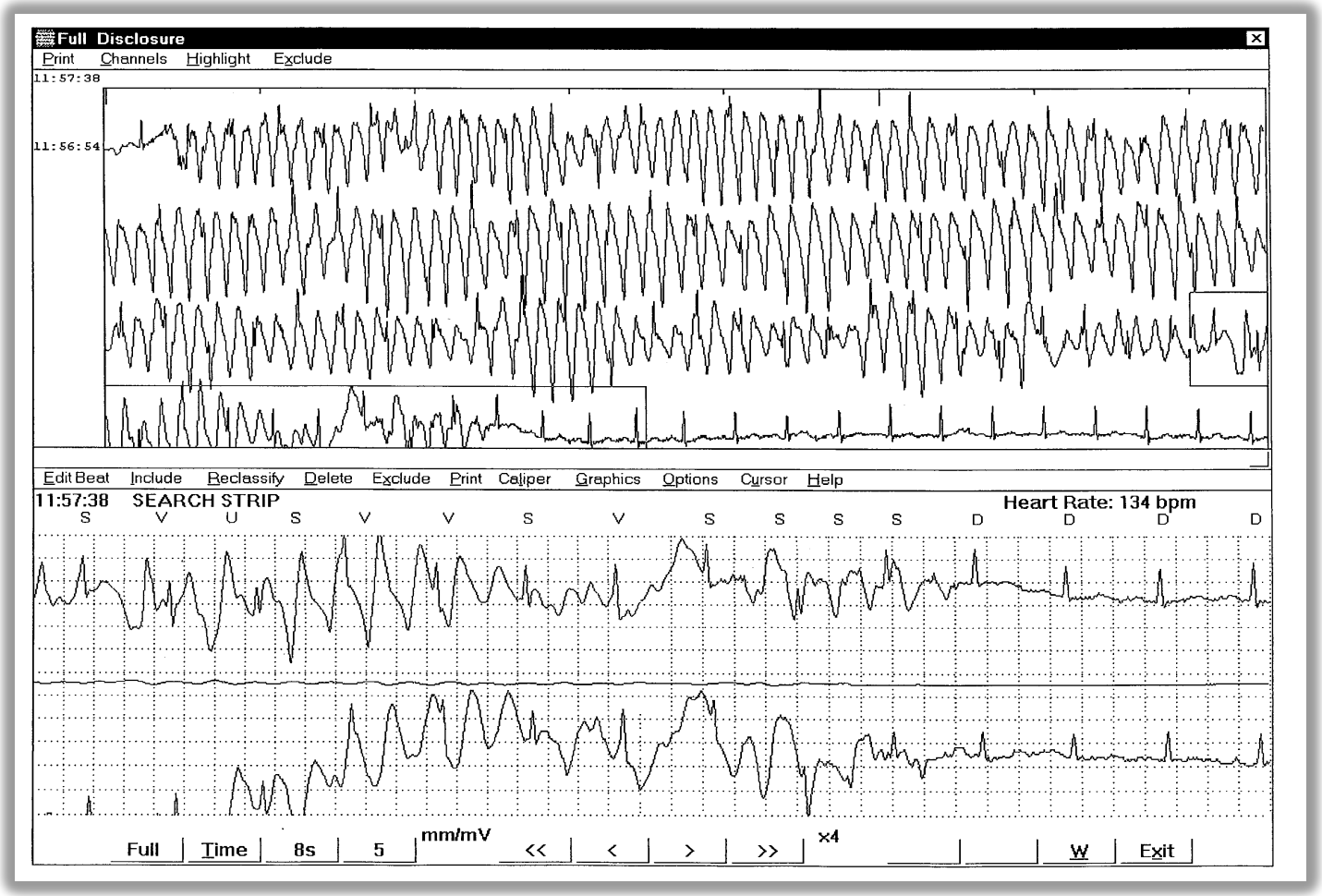


Figure 1 (A)

Case Scenario (3)

- A lady with suspected polymorphic VT on Holter monitoring



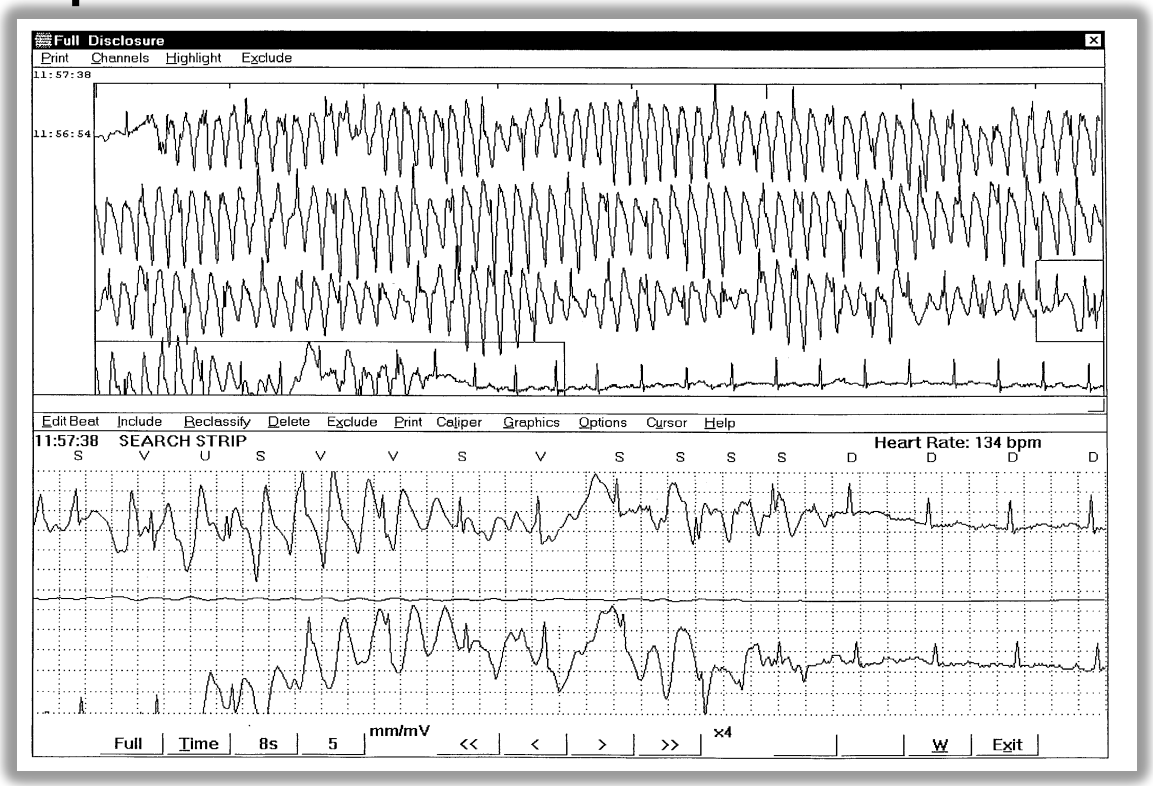
What is the your ECG diagnosis ?

(A) Polymophic VT

(B) Preexcited AF

(C) Torsades de pointes

(D) Others



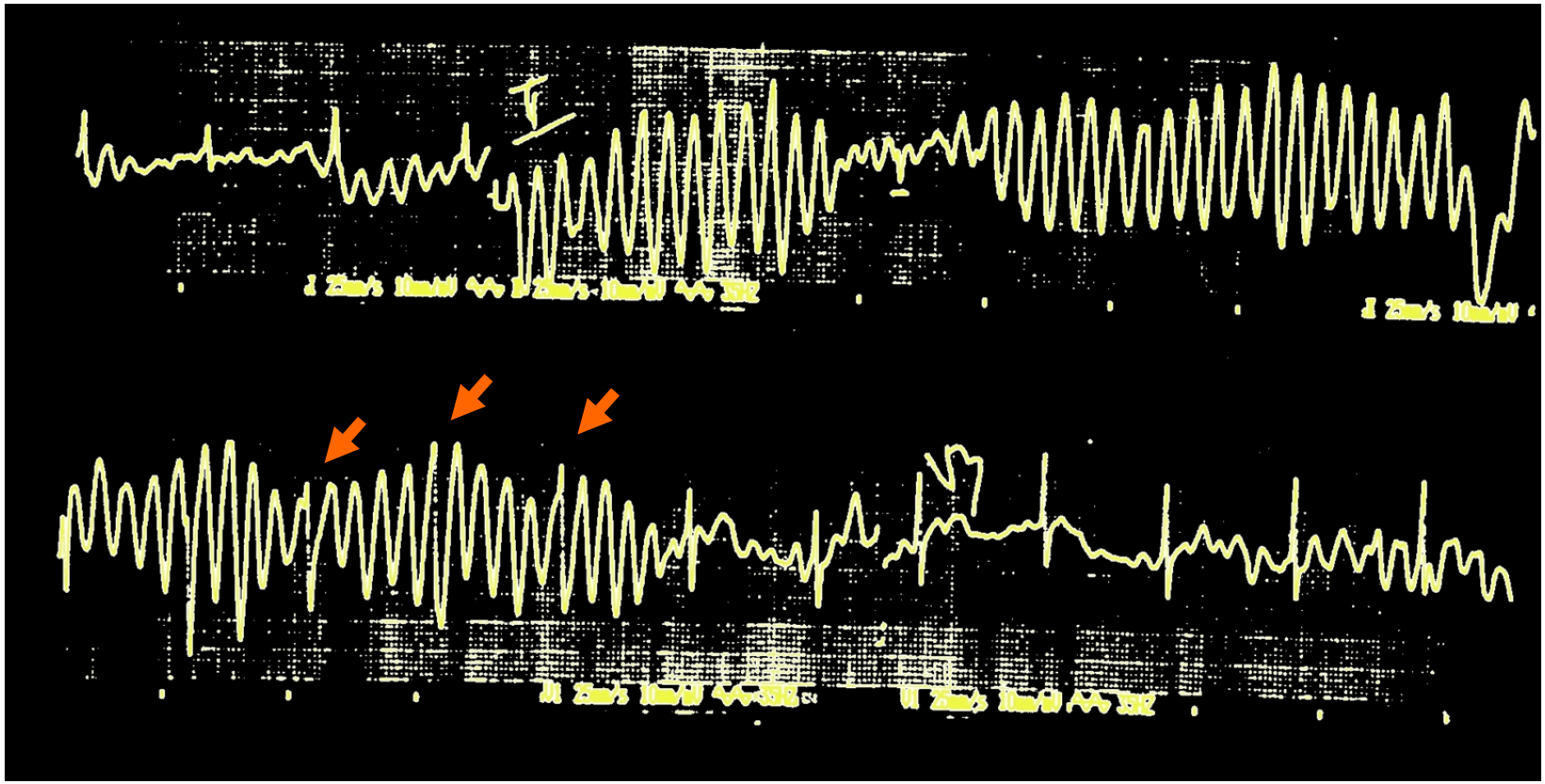
ECG artifacts mimicking VT / VF

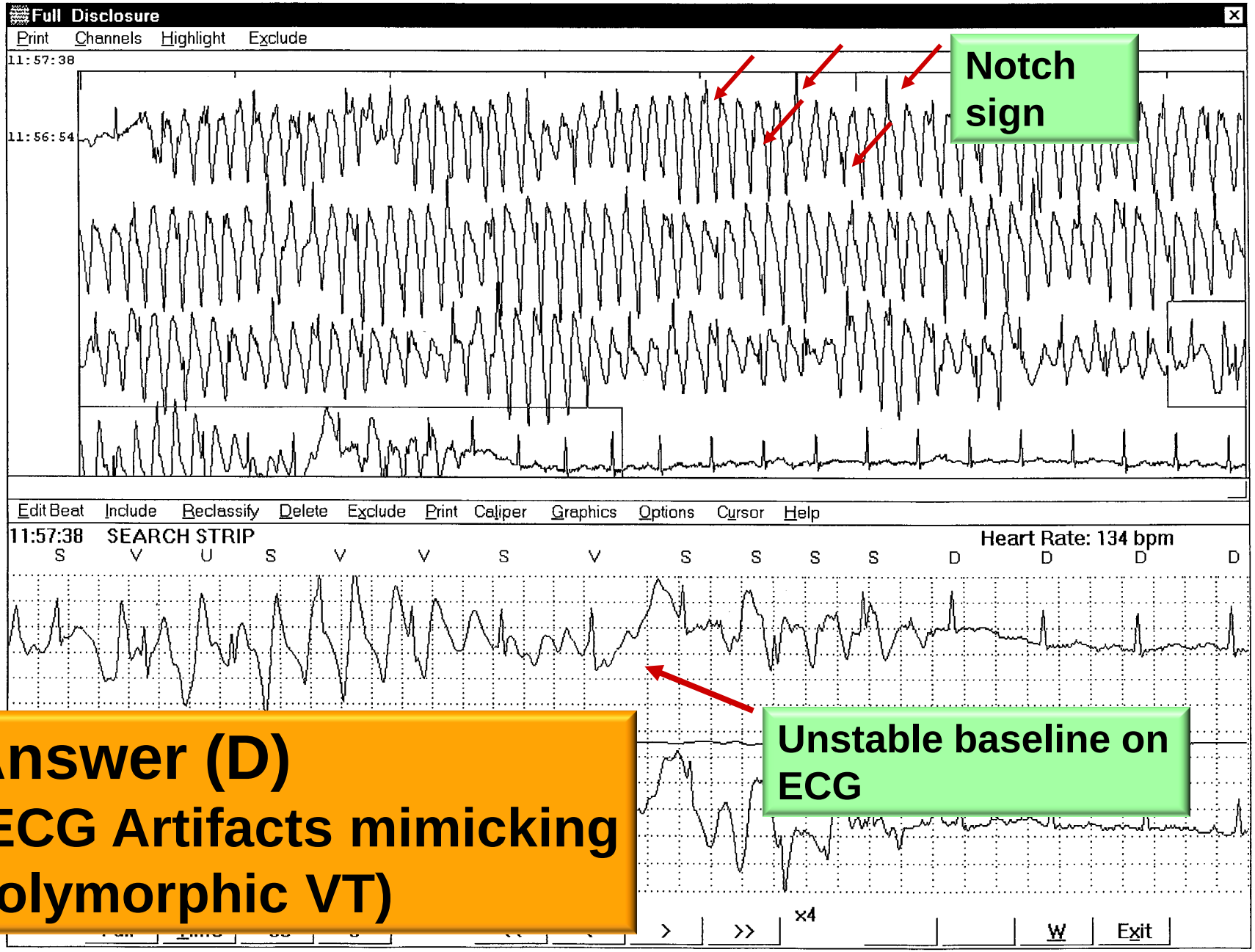
- ECG artifacts may mimic VT / VF leading to unnecessary treatment (eg. ICD) – ***NEJM Oct 1999***

- Clues to recognizing ECG artifacts during ? VT / VF

  - Normal QRS complexes at the cycle length of base-line rhythm within the artifact (the notch sign)
  - Unstable base line on ECG before the event, after the event or both
  - Association with body movement (eg hand tremor)
  - Patient asymptomatic
  - No haemodynamic disturbance during the event (feel the pulse & check BP)

Artifacts mimicking polymorphic VT in a patient with Parkinsonism





Answer (D)
(ECG Artifacts mimicking polymorphic VT)

Conclusions

1. SCD is a common cause of death due to cardiac arrhythmias
2. 12-lead ECG remains an important tool in *diagnosis* of underlying heart disease and arrhythmias, *risk stratification* and *guiding Ix & treatment* for prevention of SCD
3. 12-lead ECG during sinus rhythm should be systematically evaluated in SCD survivor and individuals who are/may be at risk of SCD
4. Stress test and/or provocation tests may unmask diagnostic ECG features of underlying heart disease to improve diagnostic accuracy

Sherlock Holmes


Thank You !
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