ECG Interpretation for Patients Presenting with Sudden Cardiac Death

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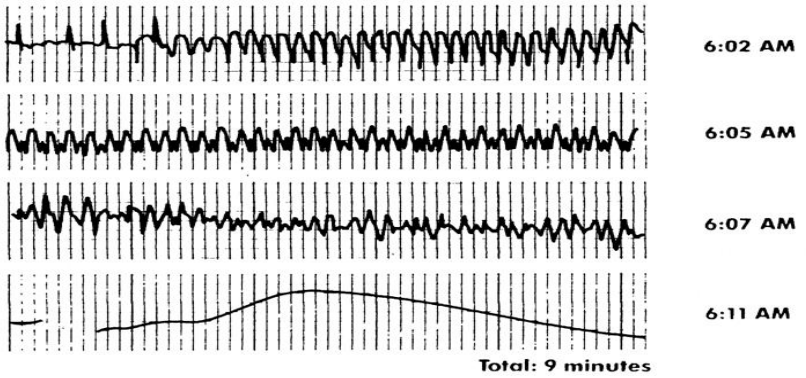
Dr. Ngai-Shing Mok
Dept of Medicine & Geriatrics
Princess Margaret Hospital
Hong Kong

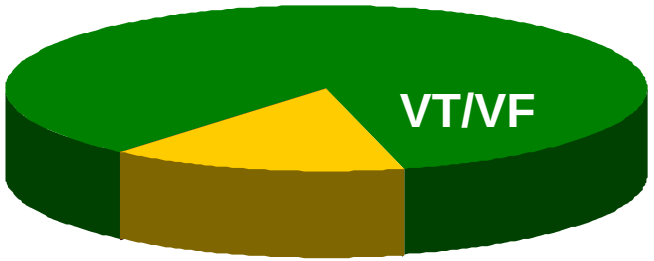
Sudden cardiac death (SCD)

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)



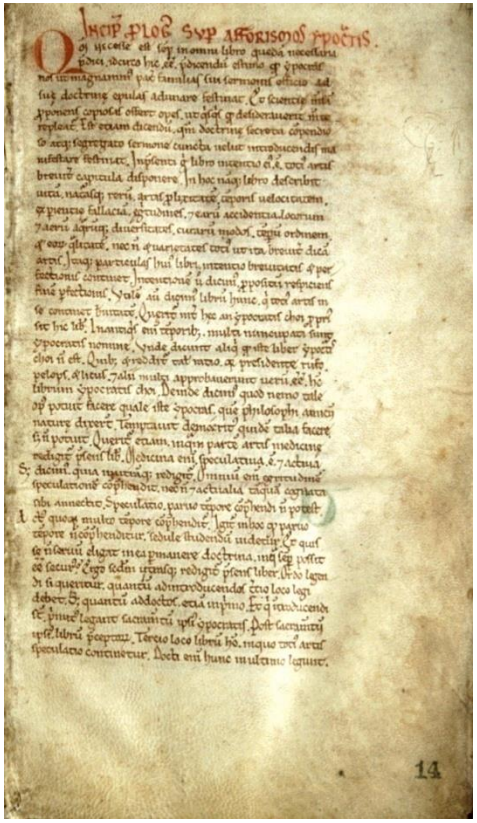


Causes of SCD

History of Sudden Cardiac Death

Sudden cardiac death ; a 2400-year-old diagnosis ?

Int J Cardiol July 2003



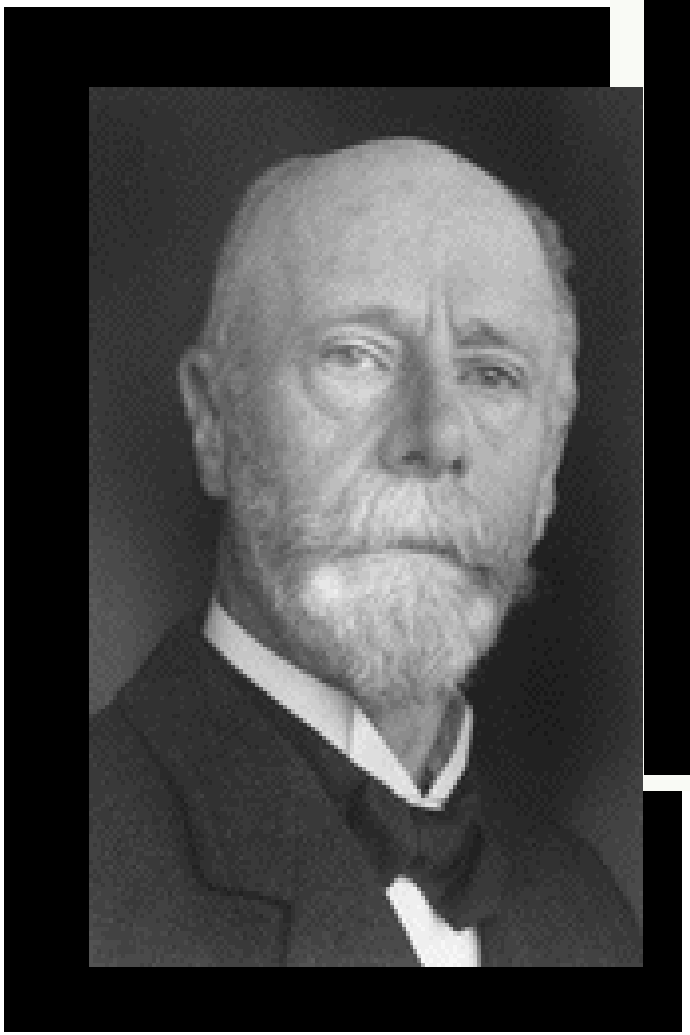
Aphorisms

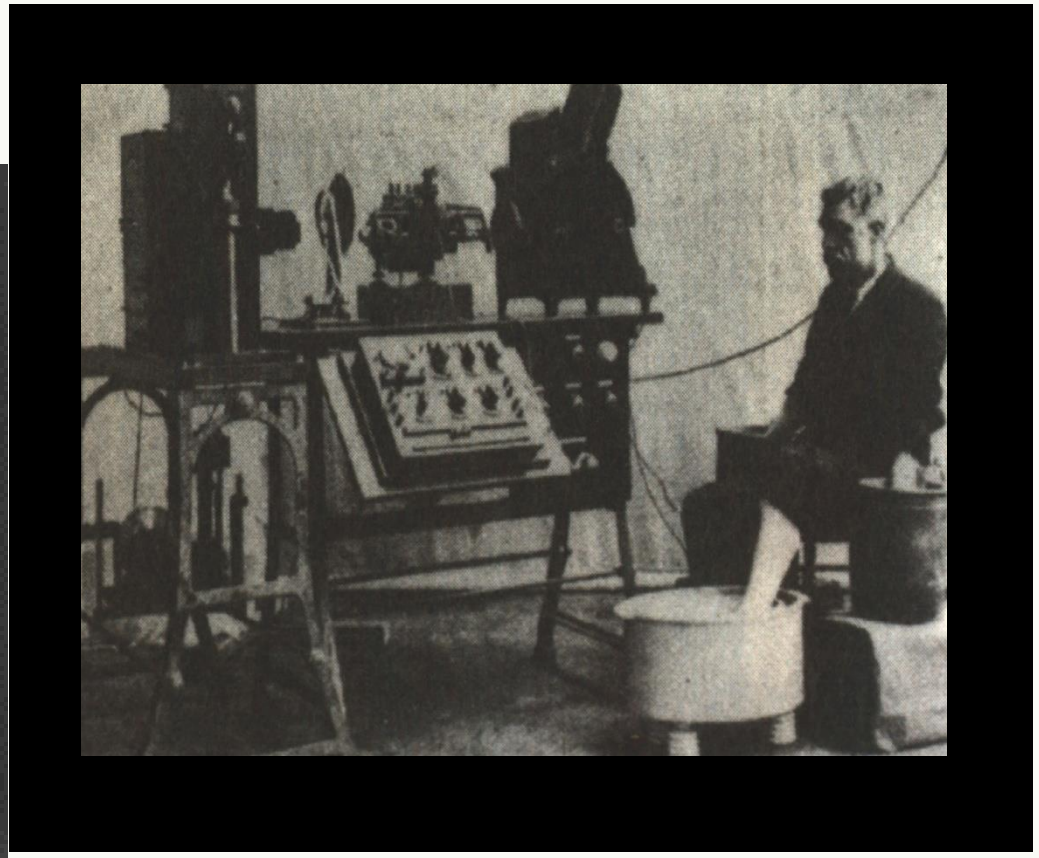
Aphorisms II, 41:

"Those who are subject to frequent and severe fainting attacks without obvious cause die suddenly"

Hippocrates

String galvanometer

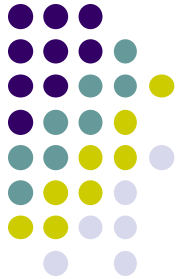




Willem Einthoven (1860-1927)

Nobel Prize Laureate in Year 1924

Different modalities of ECG recording

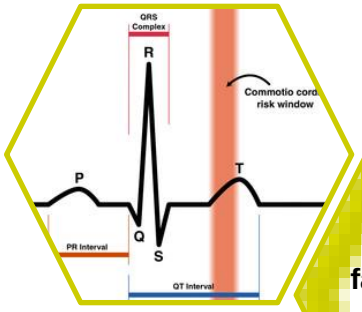


Surface ECG

- 12-lead surface ECG
- Automatic external defibrillator (AED)
- Signal-averaged ECG
- Microvolt T-wave alternans
- Heart rate variability
- Holter monitoring
- Telemetry
- Implantable loop recorder

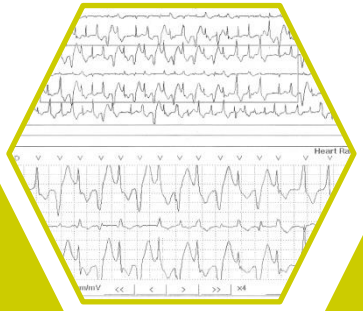
Intracardiac ECG

ECG in SCD – Outline



Diagnosis of fatal arrhythmias & underlying heart disease

ECG during provocation & stress test



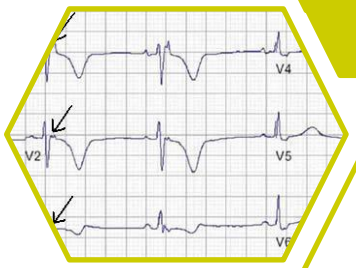
Genotype-phenotype correlation in ion channelopathies

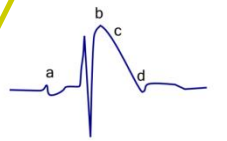
Systemic evaluation of ECG in SCD

ECG modulation by drugs

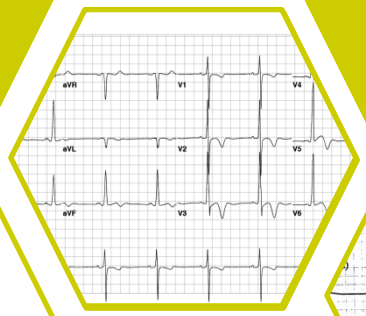
ECG in risk stratification

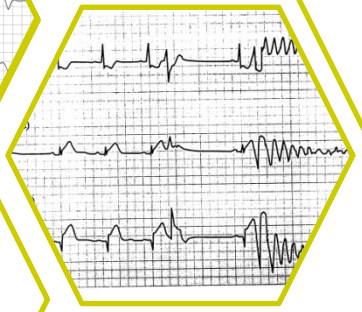
ECG in SR



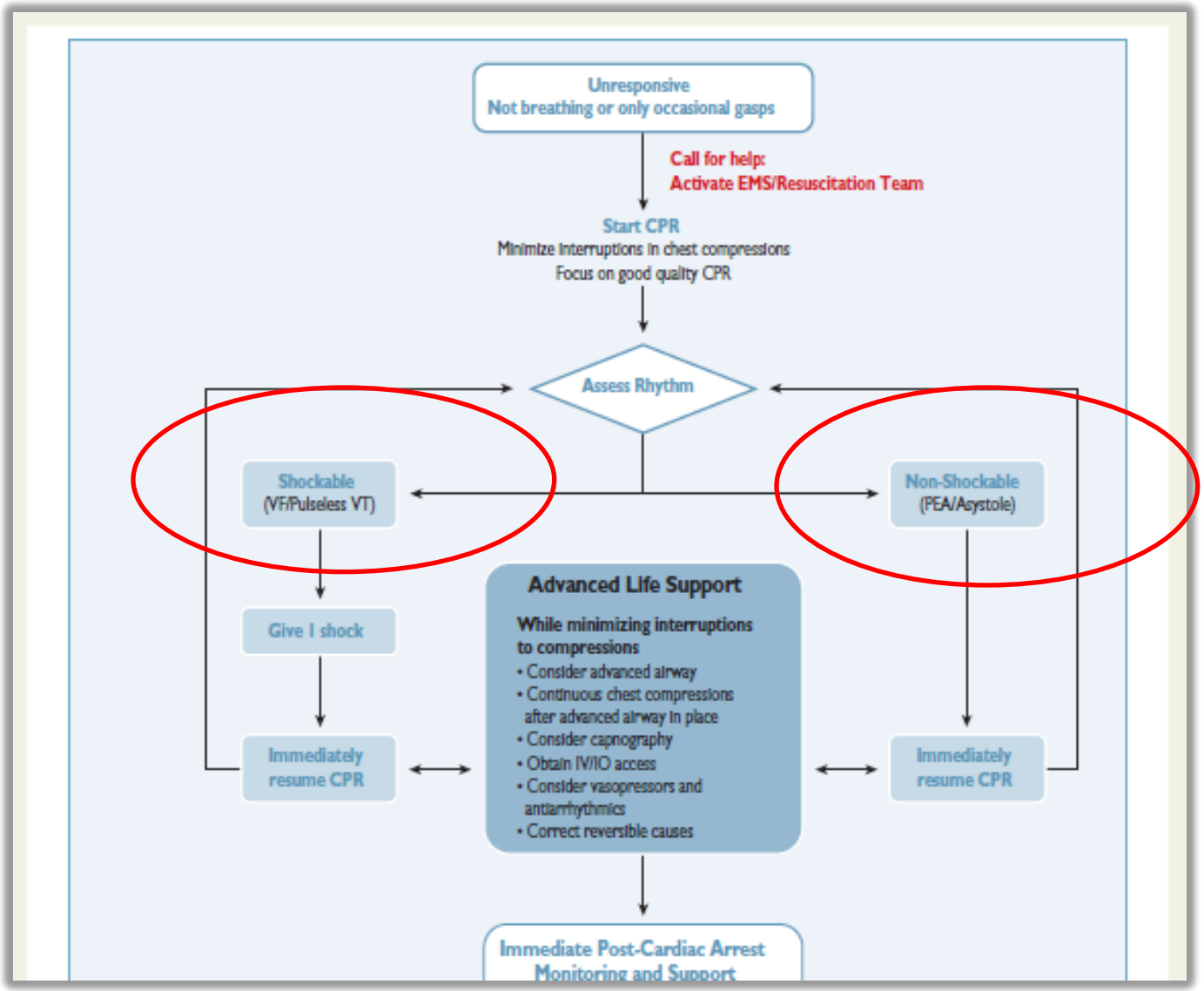


ECG characteristics in Brugada Syndrome
a. Broad P wave with some PQ prolongation
b. J point elevation
c. Coved type ST segment elevation
d. Inverted T wave



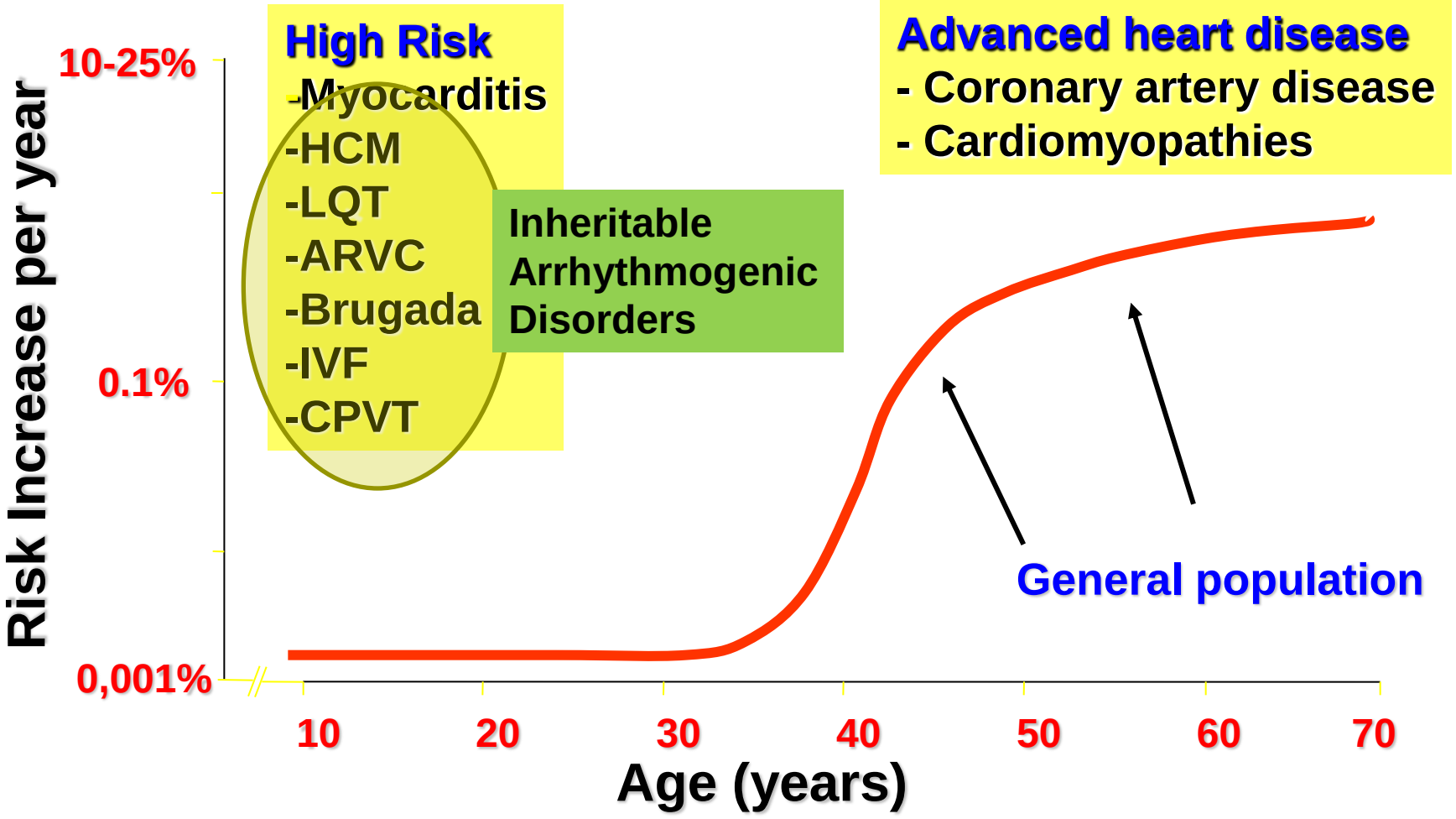


Recognition of shockable rhythm during SCA - VF or pulseless VT



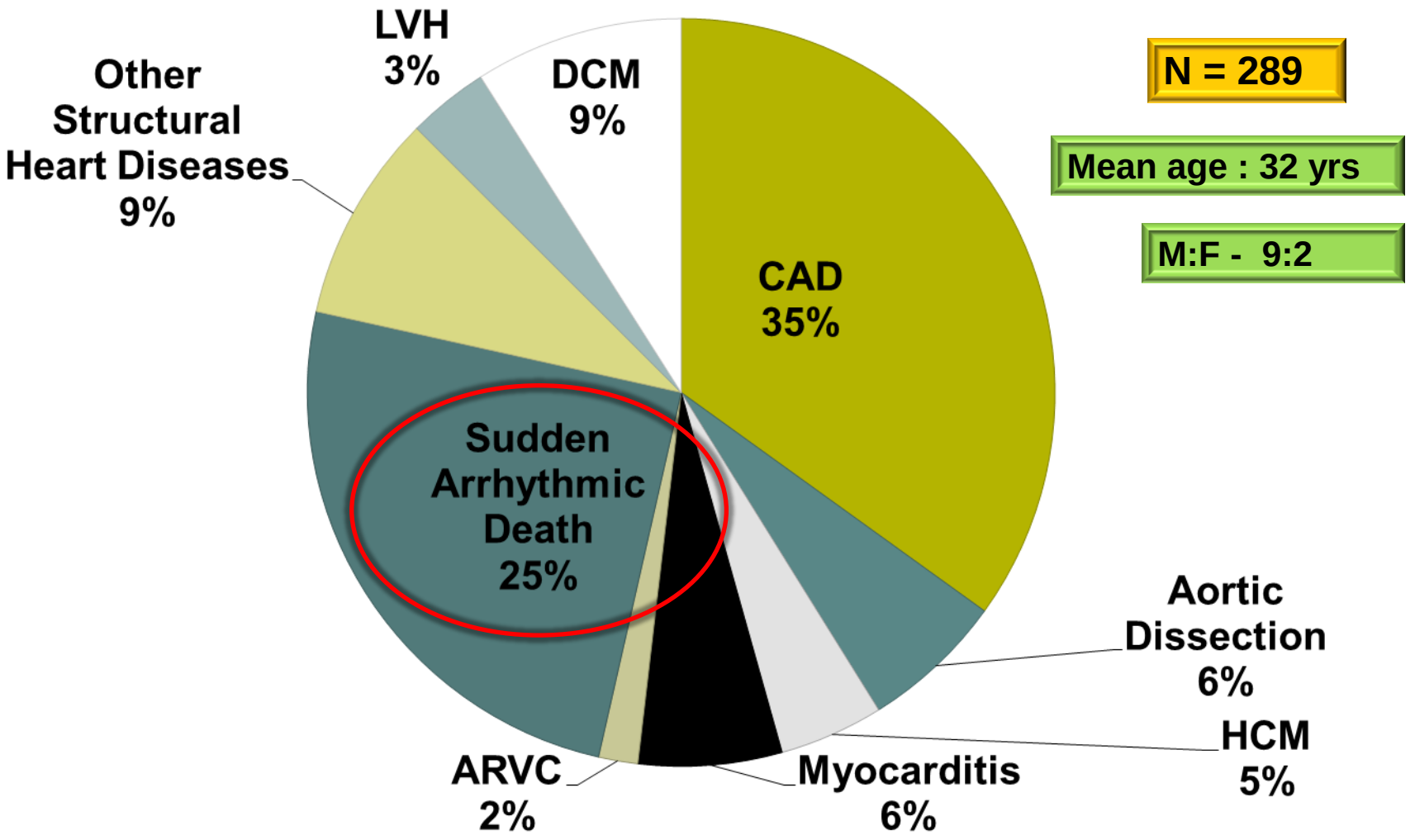
ESC Guidelines 2015

Age-specific risk and causes of SCD



Causes of Sudden Cardiac Death Among Young Victims – A 5-Year Review of Autopsies Done in Hong Kong

Mok et al, APHRS 2013

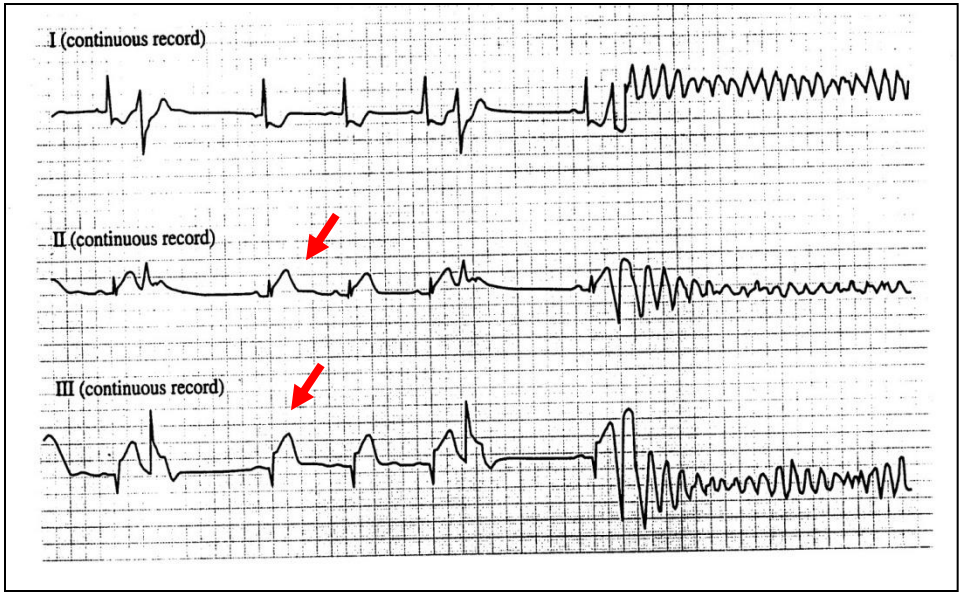


Coronary artery disease (CAD)

Sir Edward Youde 1924-1986

Died of SCD due to AMI

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



Acute inferior MI presenting with SCD

ECG features of critical pLAD stenosis

Winter Is Coming After a Cardiac Arrest

Circulation. 2017;135:1977–1978.

- Upsloping ST depression V2-V4 (up to V6) continue to tall positive and symmetrical T waves
- 1-2 mm ST elevation in lead aVR

De Winter Syndrome

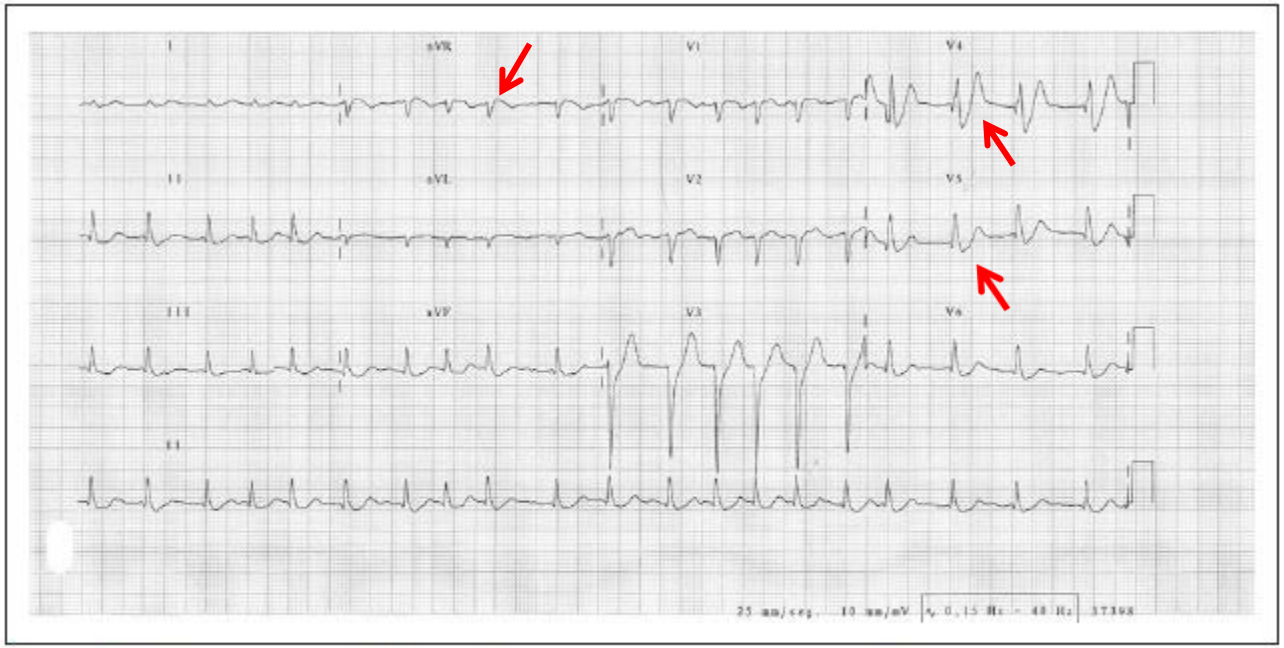


Figure 1. Twelve-lead ECG performed on patient admission to the emergency department.

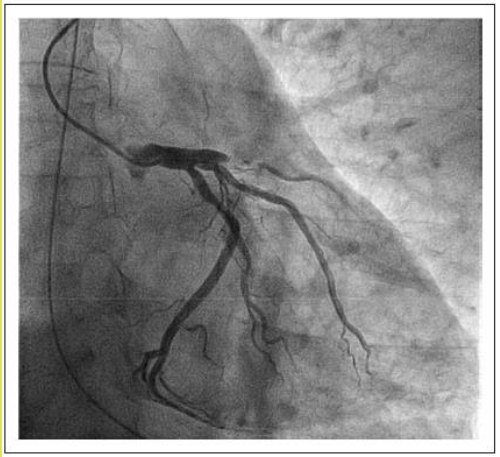
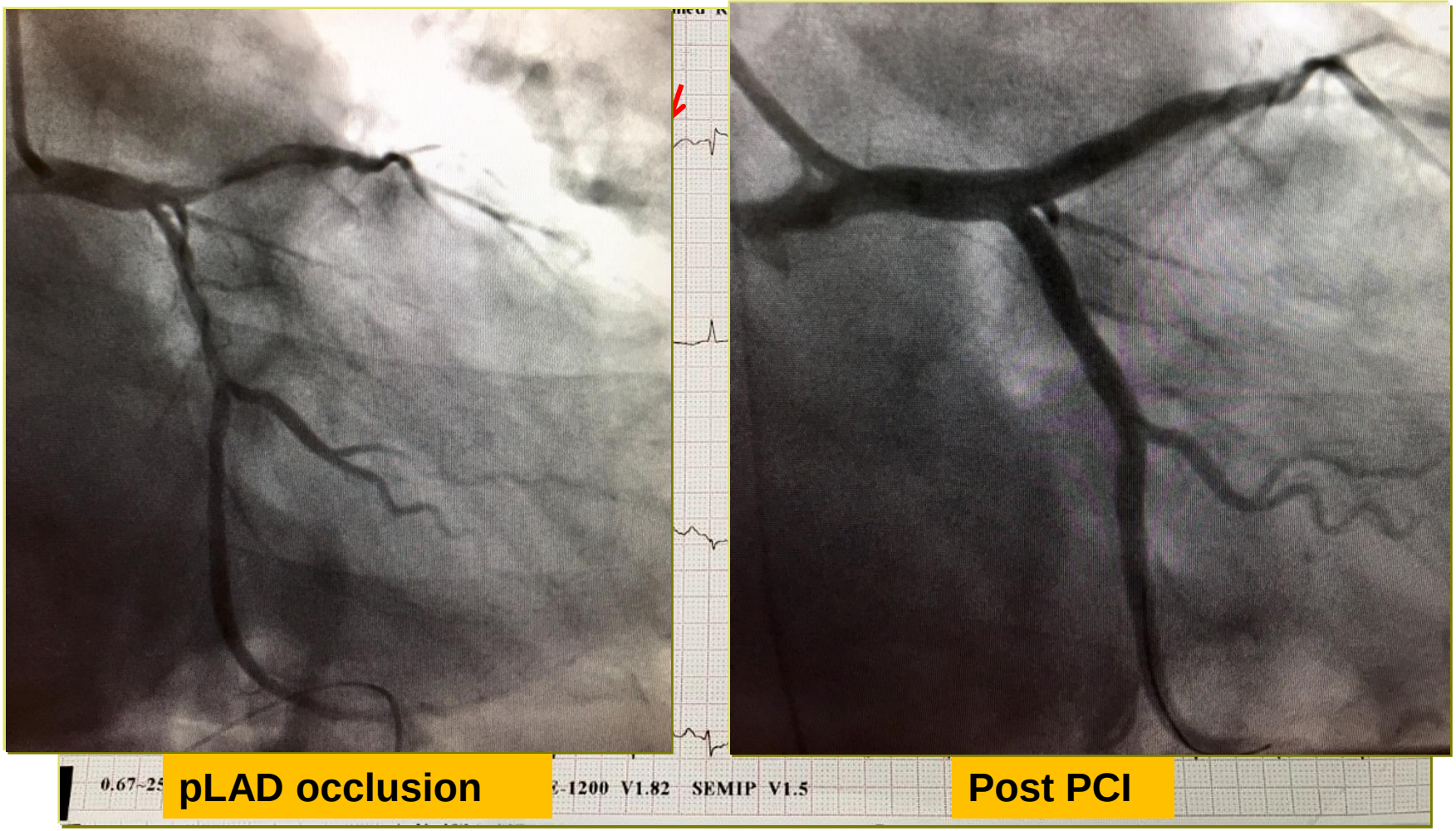


Figure 2. Coronary angiography demonstrates an acute coronary occlusion of proximal left anterior descending artery.

- M/51
- Sudden cardiac arrest while cooking
- Immediate CPR
- VF successfully defibrillated by AED



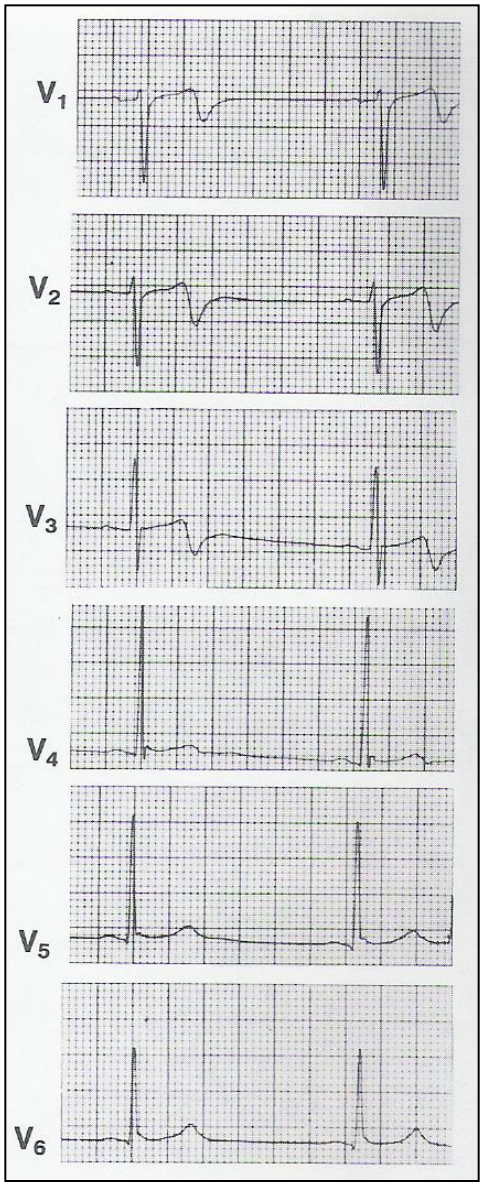
ECG features of critical pLAD stenosis

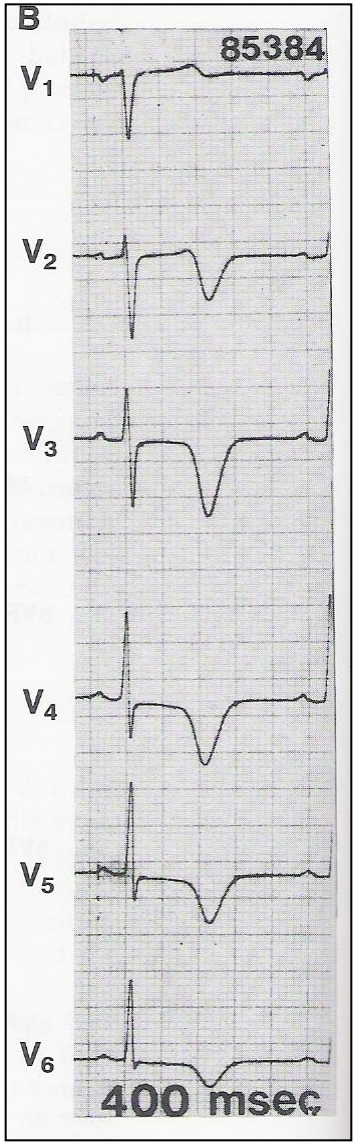
Wellen's Syndrome

- Symmetric, deep T Wave inversion or biphasic T waves in V2-V3 (up to V6) during pain free periods

- Minimal (<1mm), if any, ST elevations

HJJ Wellens, et al. AHJ 1982

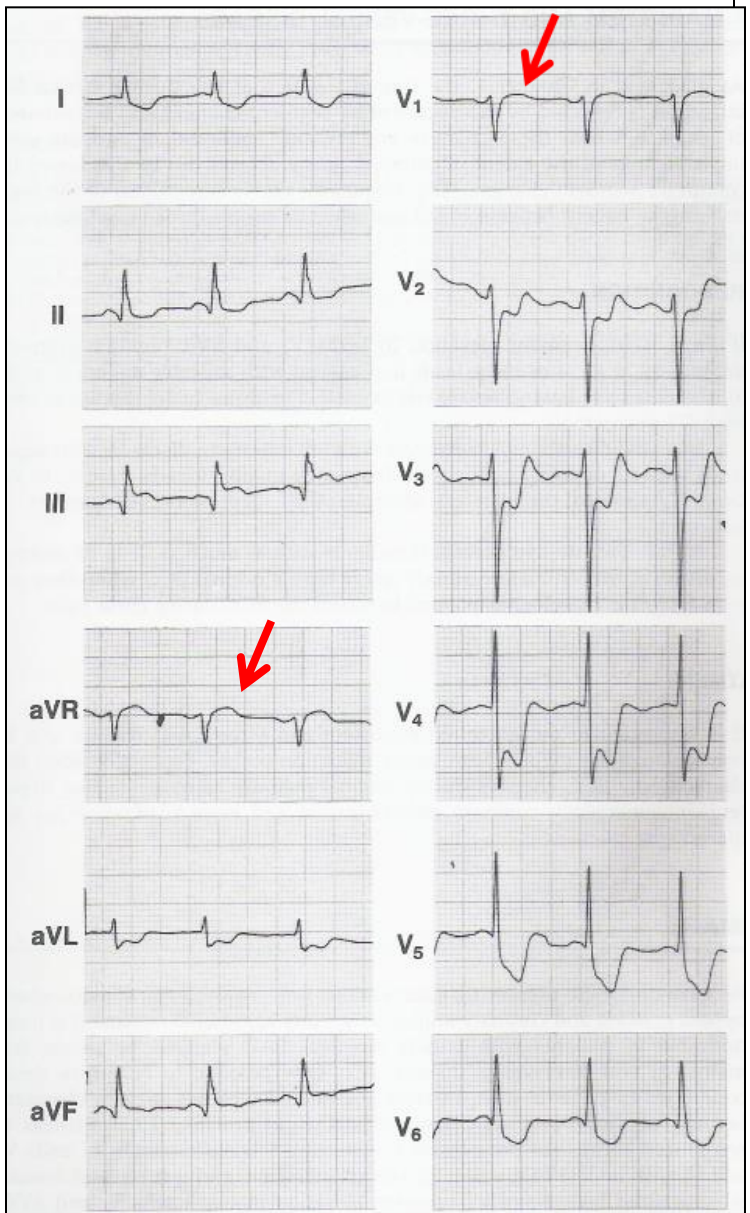




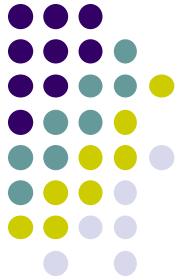
ECG features of L main or 3VD

- ECG sign during chest pain
- ST elevation in V1 & aVR
- ST depression in ≥ 8 leads (esp V3-V5, V4 greatest STD)
- 71% sensitivity

Atie J , et al. EHJ 1991



Congenital CAD at risk of SCD

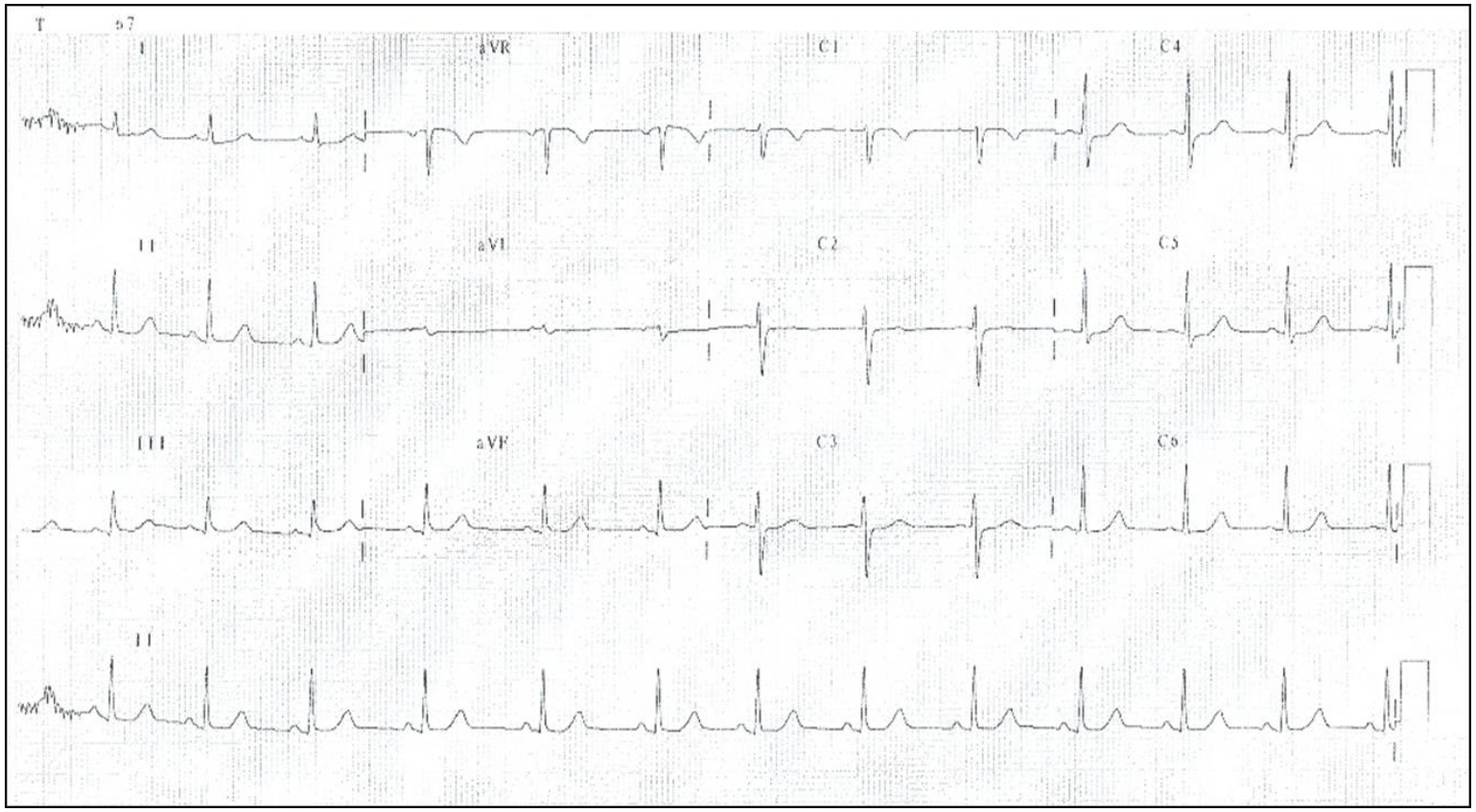


- Left main atresia
- Anomalous origin of coronary arteries with a malignant course
  - LCA arising from pulmonary artery
  - LCA arising from R Sinus of Valsalva
  - RCA arising from L Sinus of Valsalva
  - Coronary artery course between aorta & pulmonary trunk

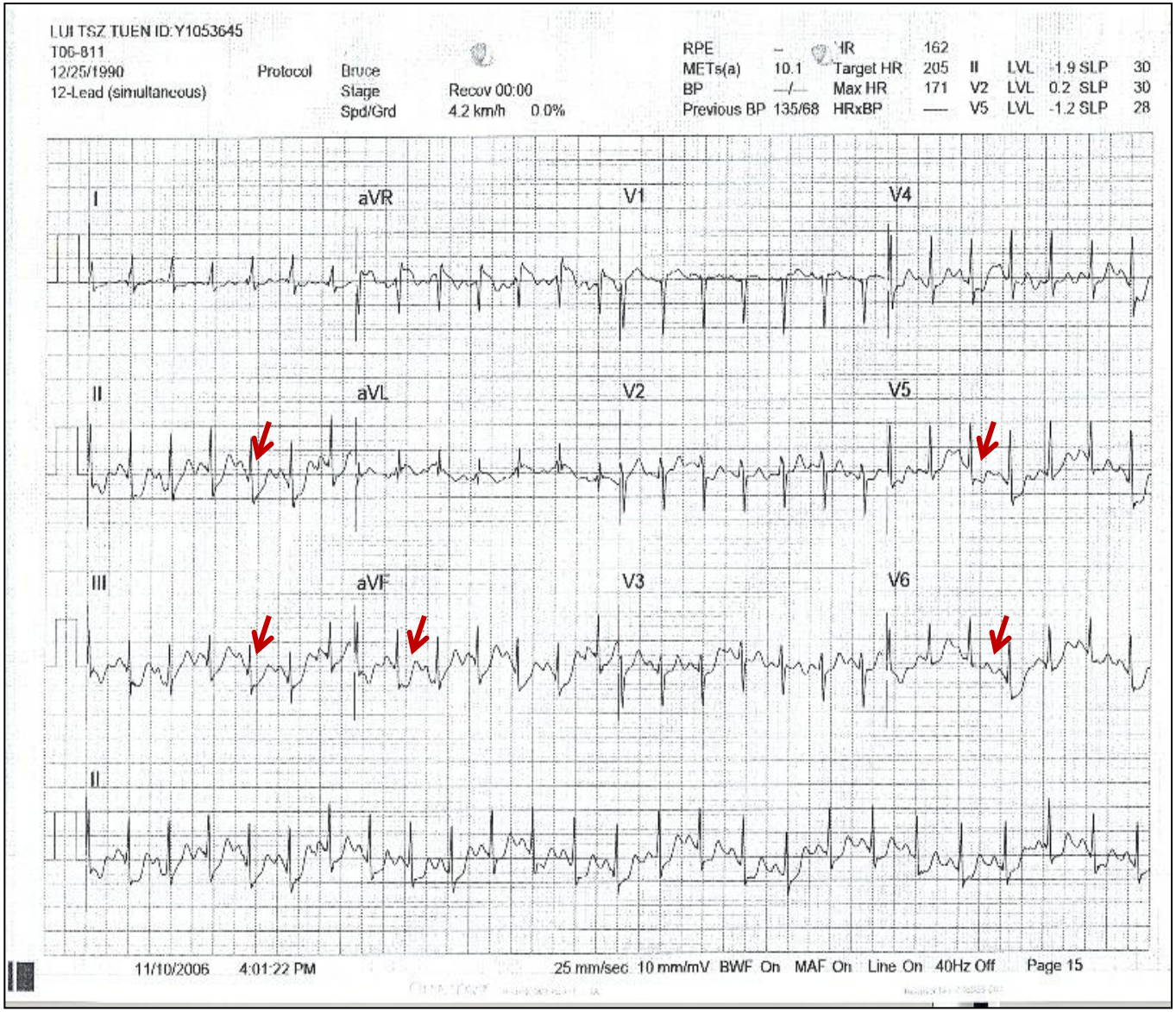
Congenital left main atresia

History

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

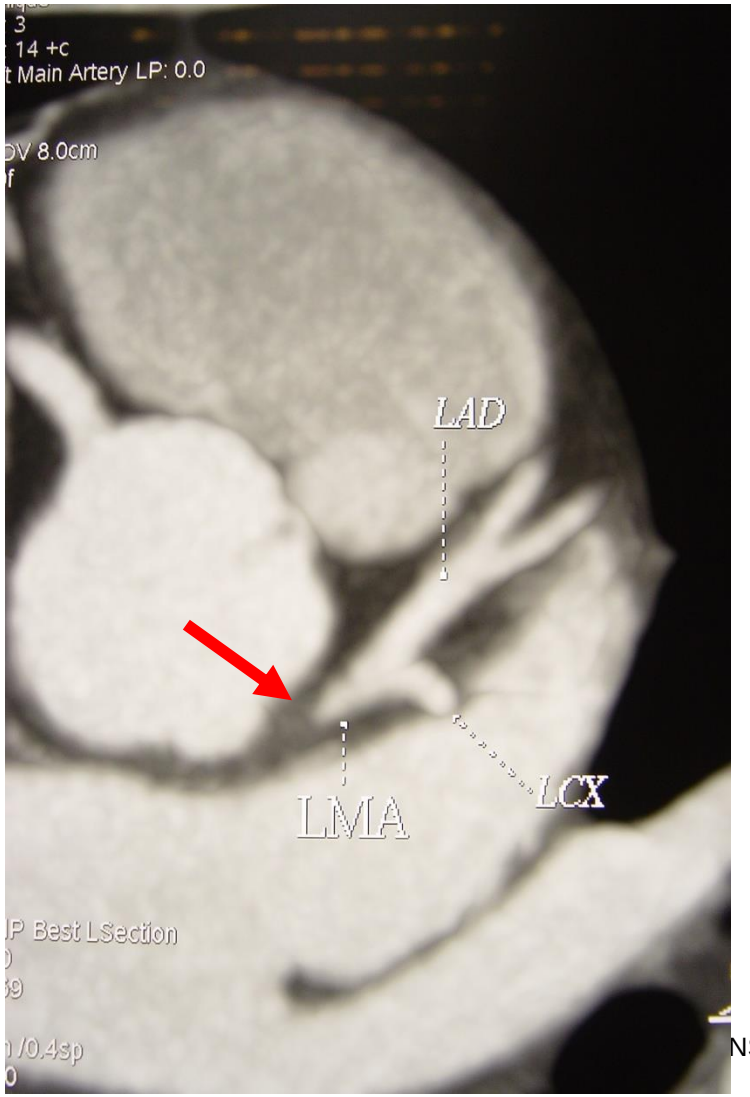


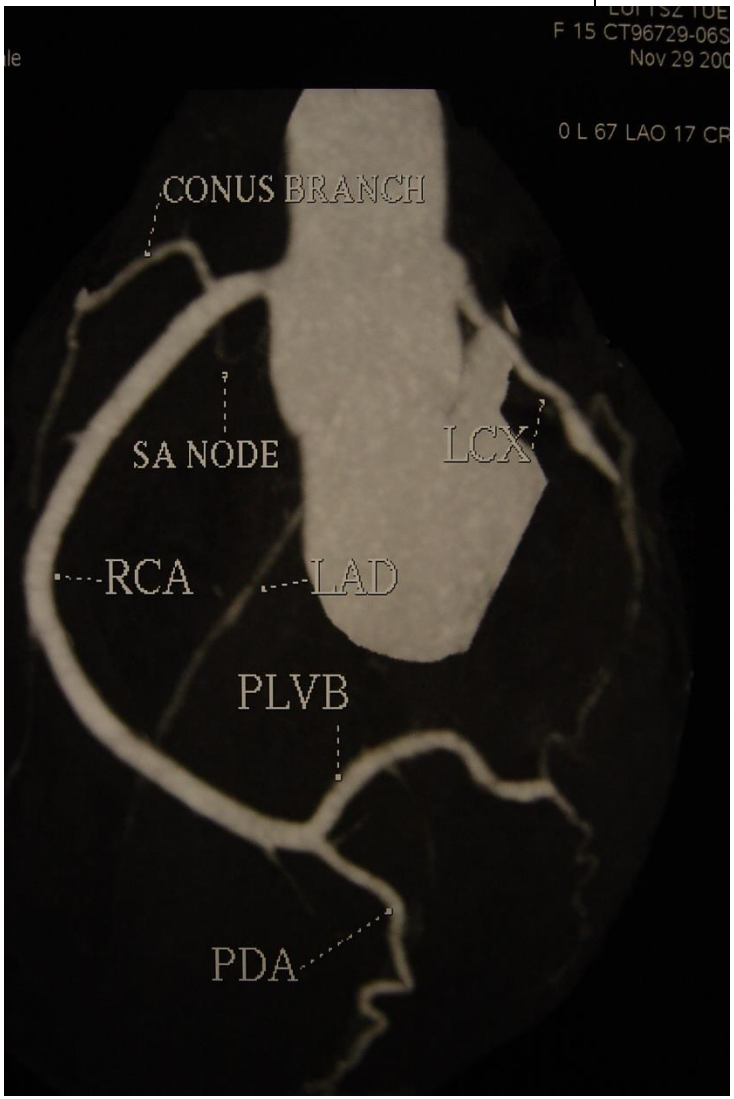
Diffuse ST depression during Treadmill Exercise Test



CT coronary angiograms

- L main atresia with collaterals from RCA



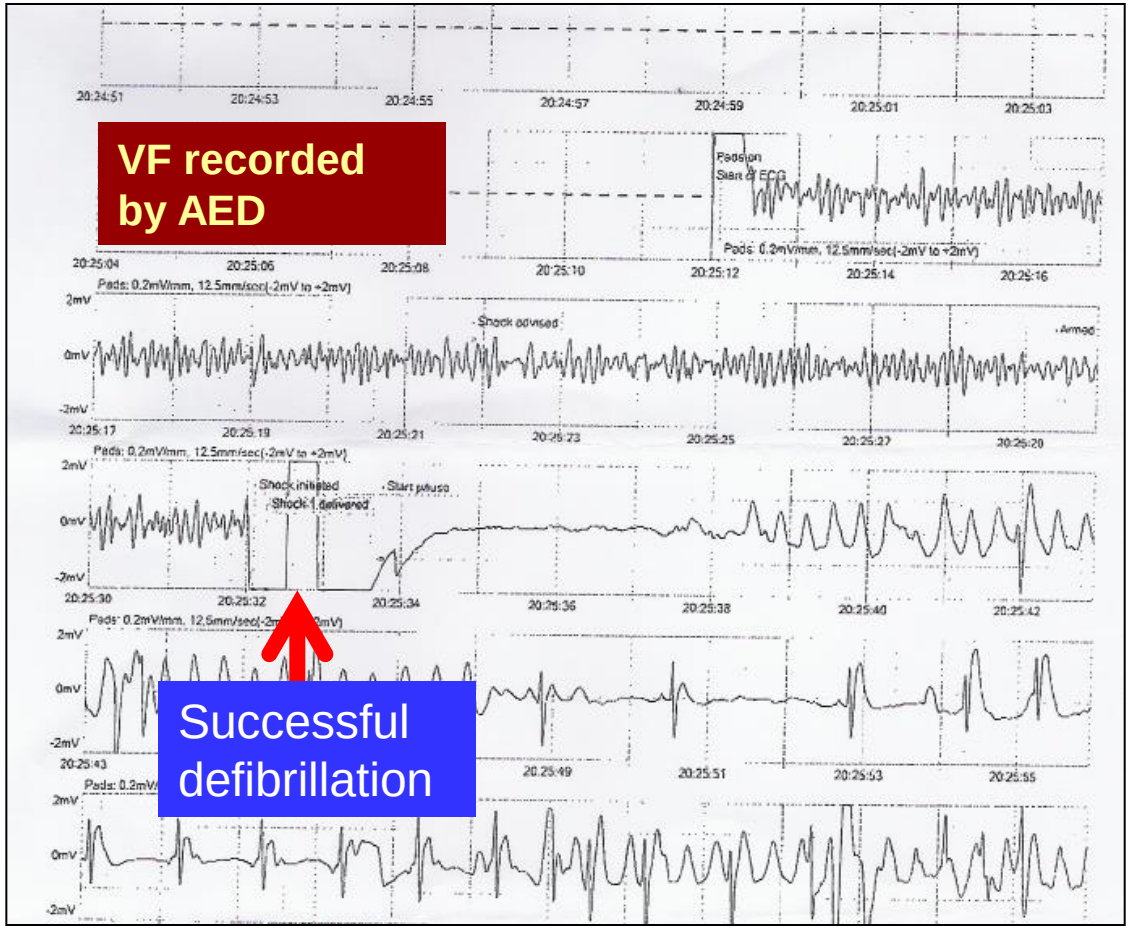


NS Mok

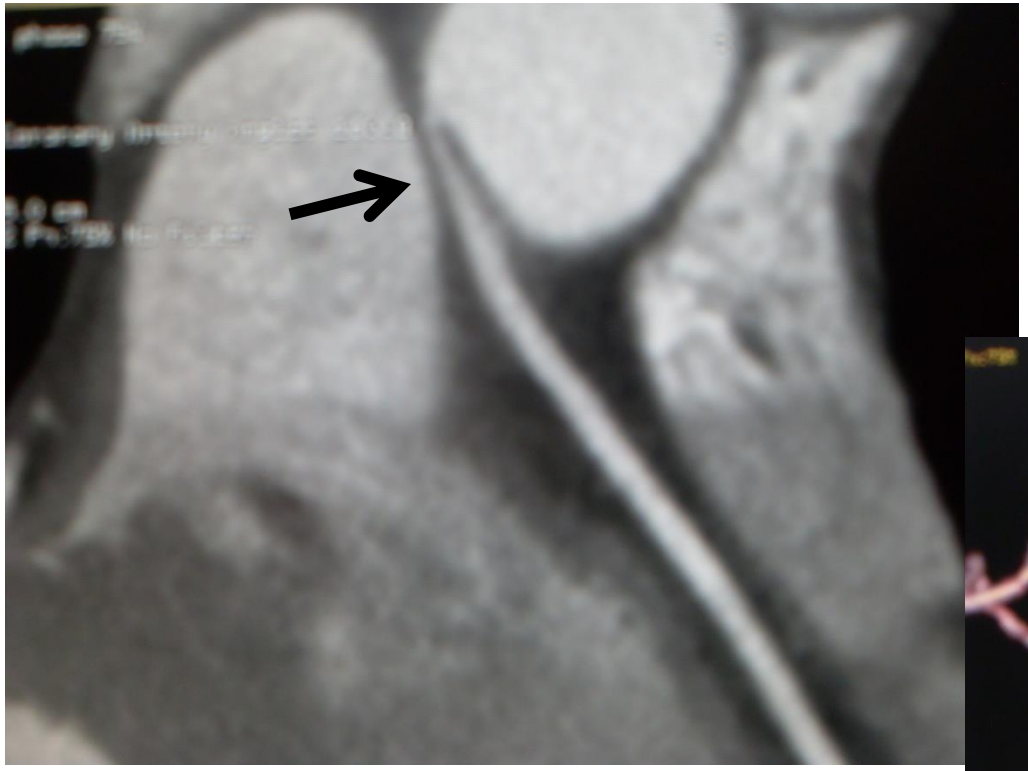
Anomalous RCA with a malignant course

History

- M/36,policeman, good PH
- Sudden collapse with LOC while playing soccer with firemen

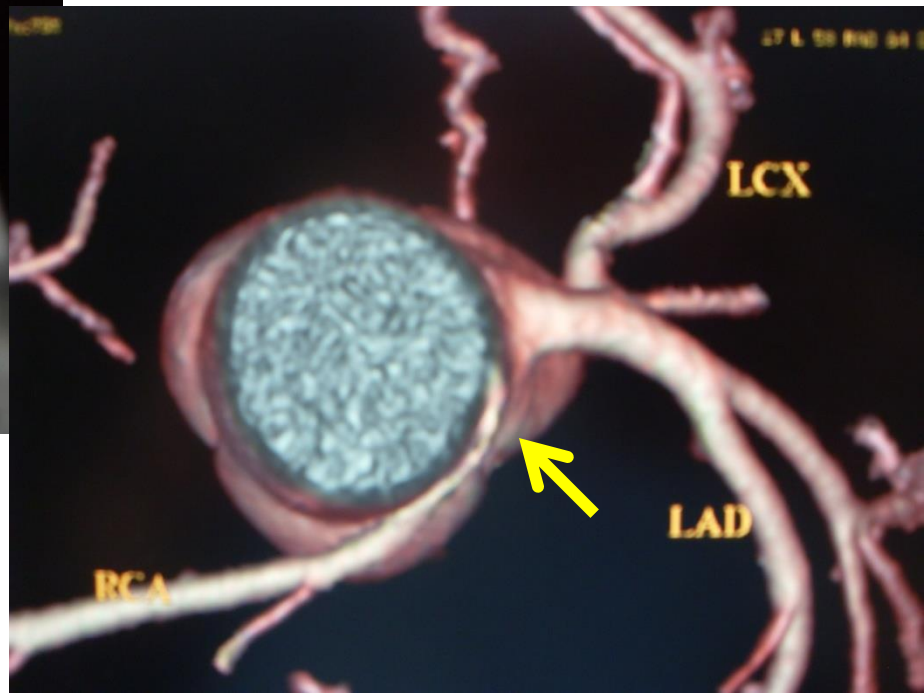


Anomalous RCA with a malignant course in a survivor of Commotio Cordis

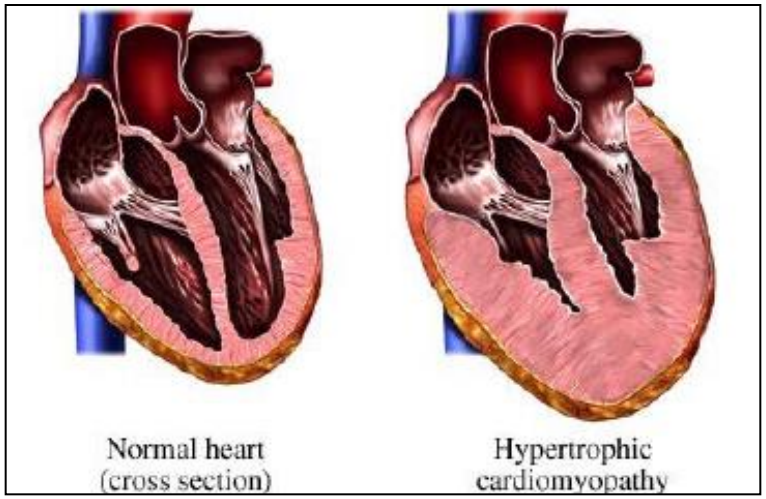


pRCA compressed between aorta & RVOT

RCA arising from L sinus of Valsalva

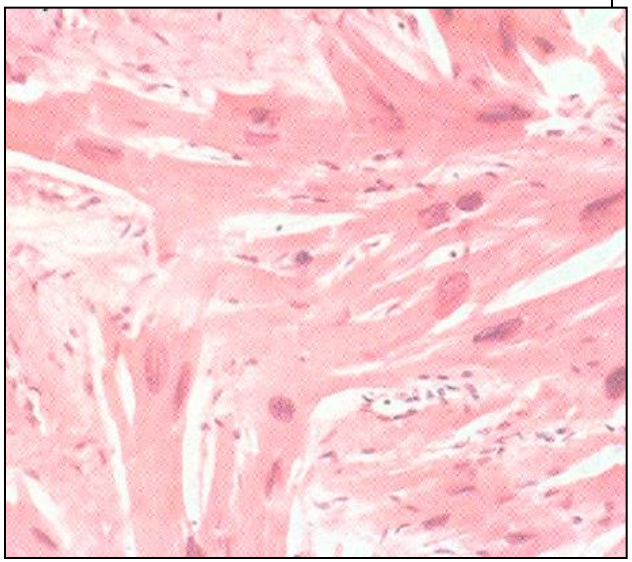


Hypertrophic Cardiomyopathy (HCM)



Asymmetrical septal hypertrophy (ASM)

Myocardial fibre disarray

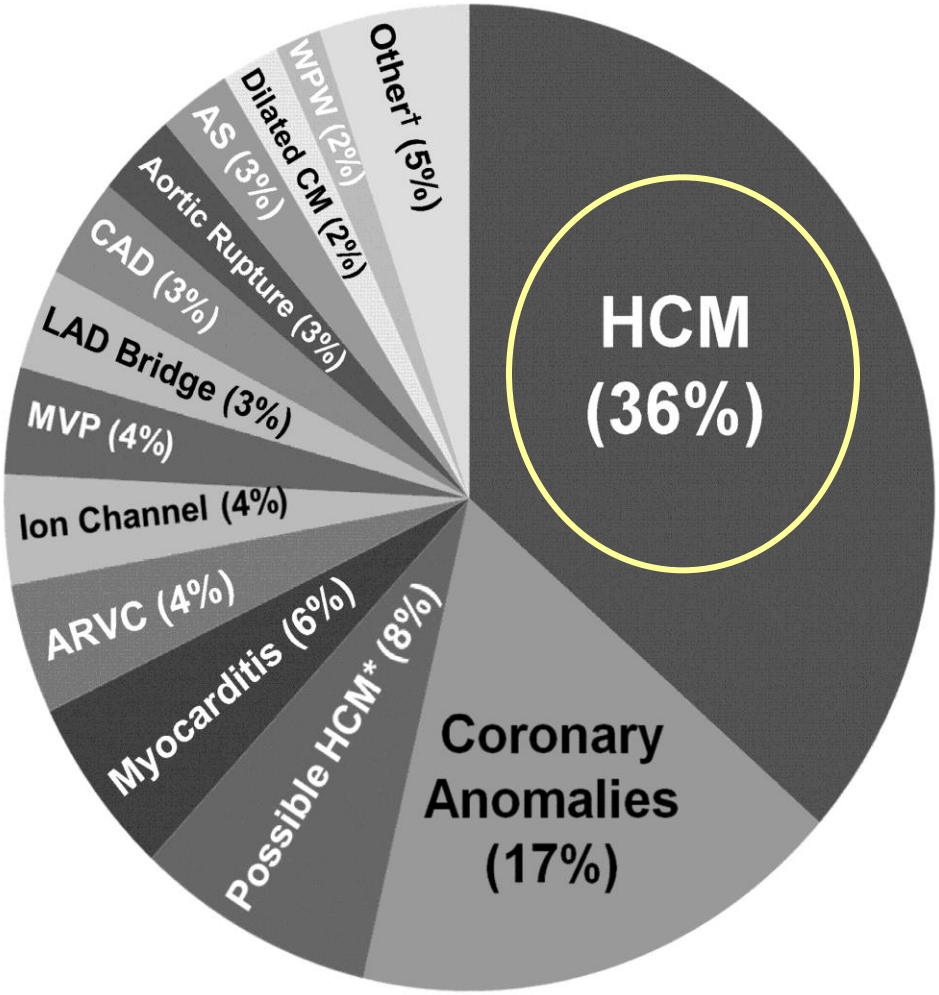


- **SCD may be triggered by exercise in high-risk patients**
- **High-risk ECG features**
  - SMVT
  - NSVT on Holter

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

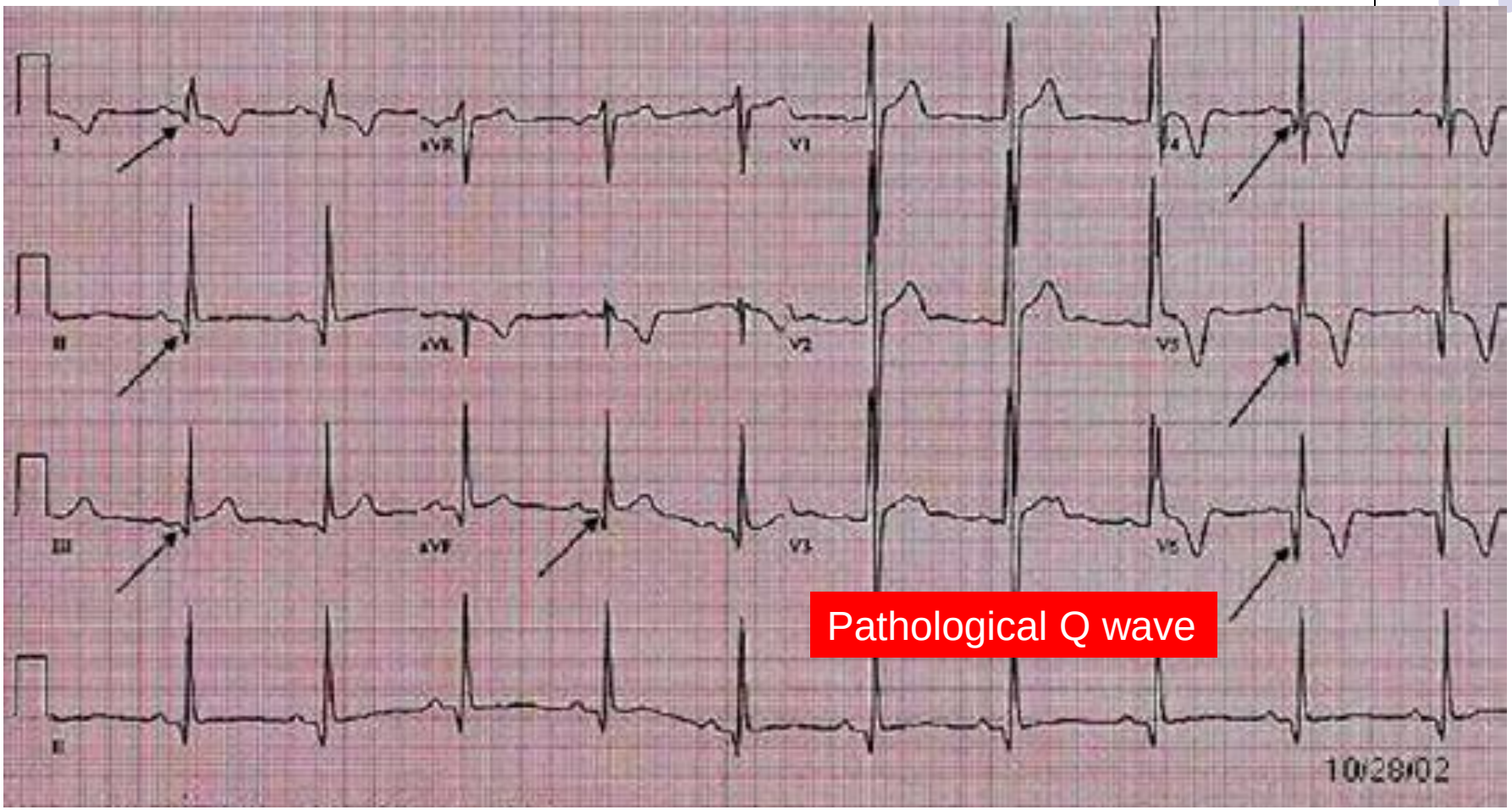
N = 158

Mean age = 17



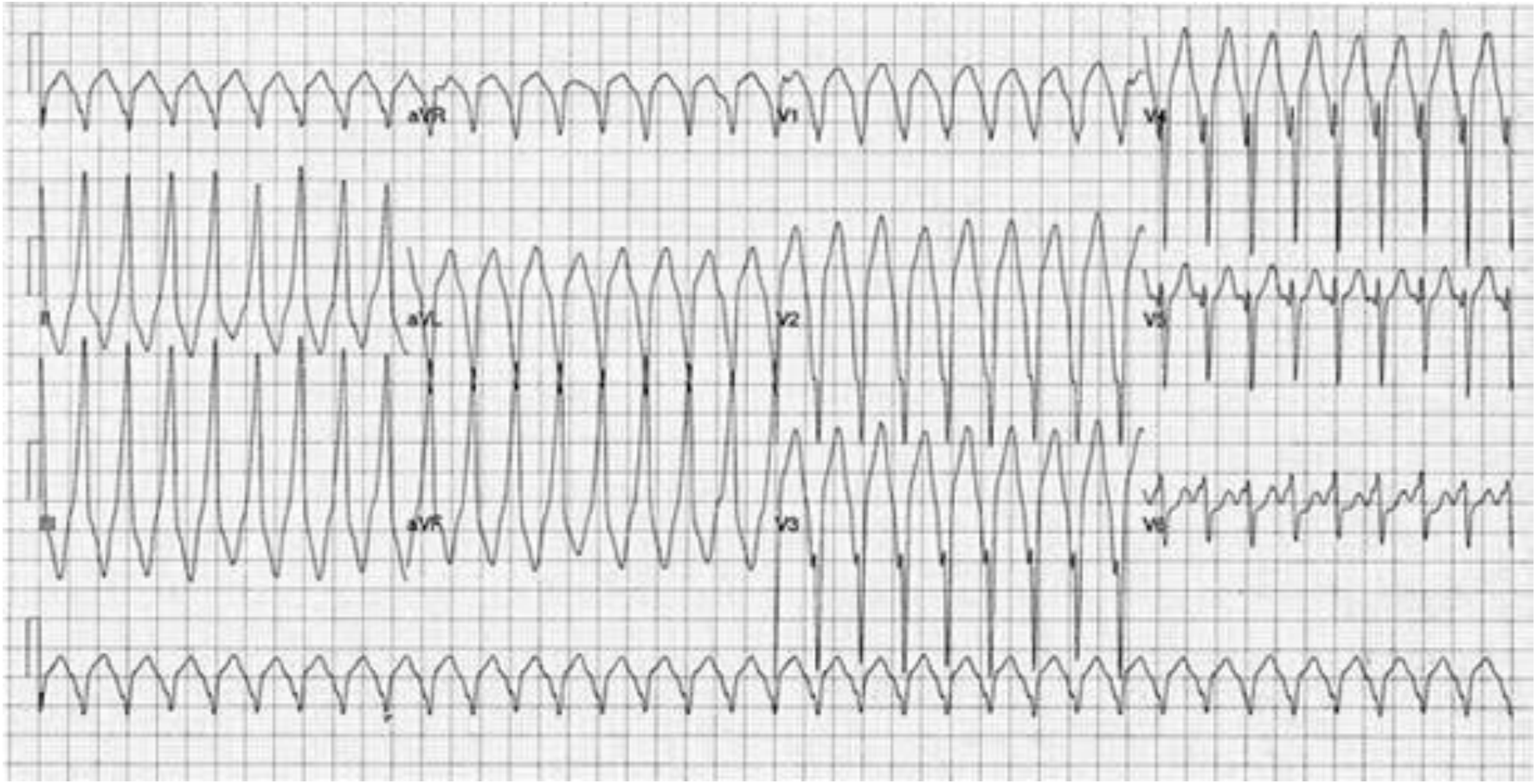
Maron BJ et al, Circulation 1996

ECG features of HCM



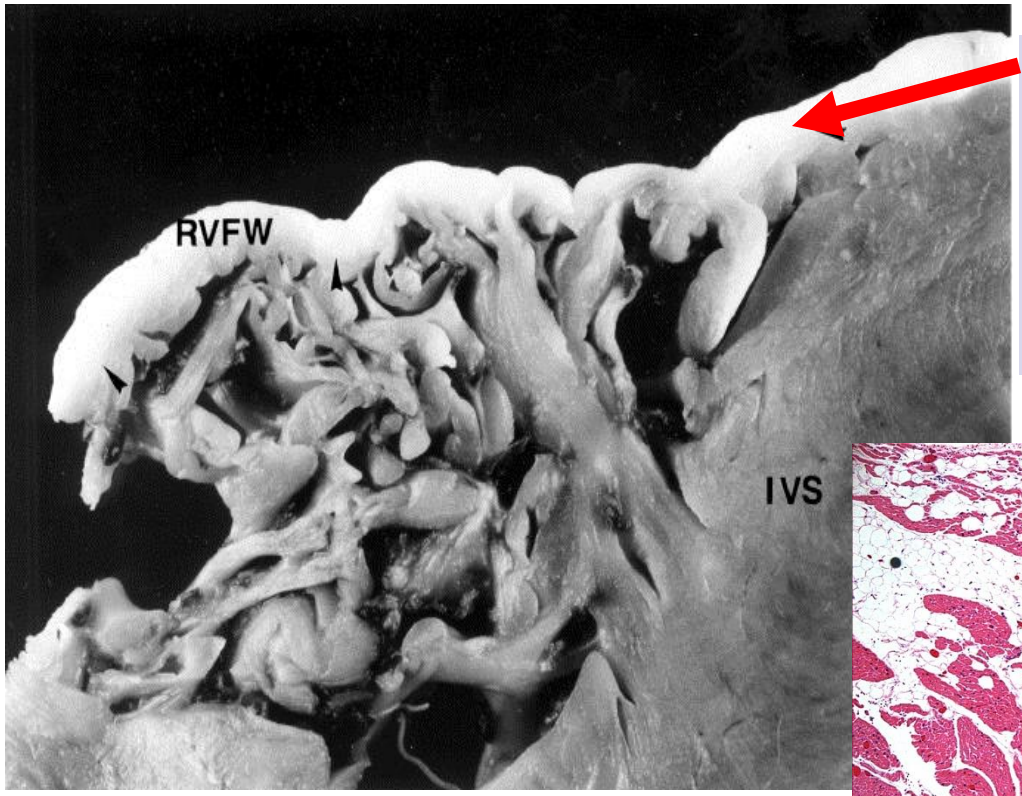
LVH with strain & pseudoinfarction pattern

HCM presenting with SMVT

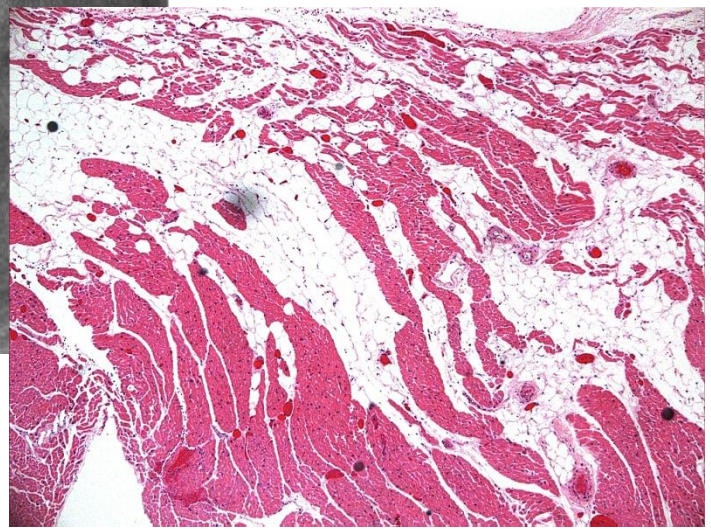


Arrhythmogenic RV Cardiomyopathy (ARVC)

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



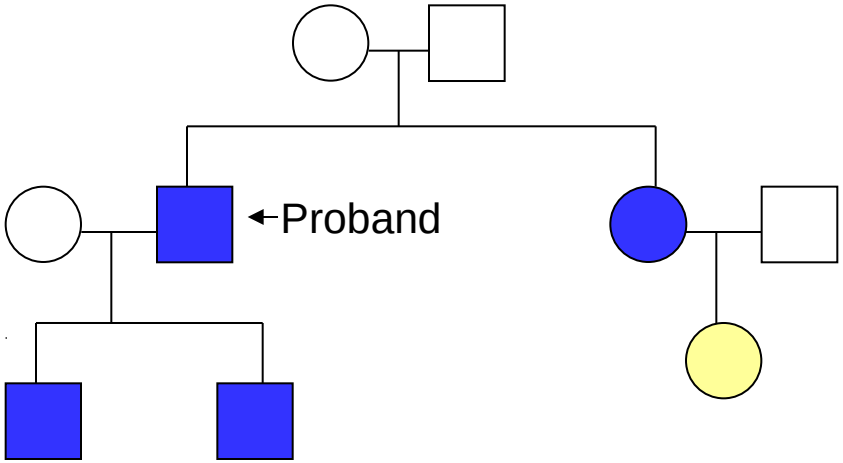
- Fibro-fatty replacement of RV myocardium
- VT/VF/ R heart failure
- Commonest cause of SCD in young athletes in Northern Italy



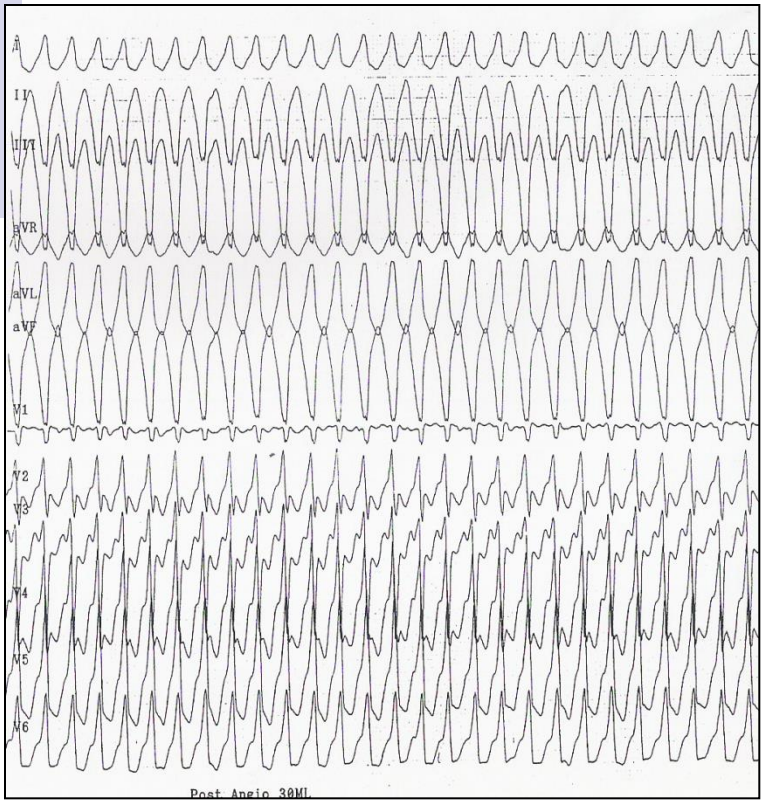
Familial form of arrhythmogenic right ventricular dysplasia presenting with recurrent ventricular tachycardia

Mok NS et al, HKMJ 1999

- M/50, presenting with VT & syncope
- Confirmed ARVC
- Genetic study found mutation in *PKP2* gene in proband and his sister & 2 sons



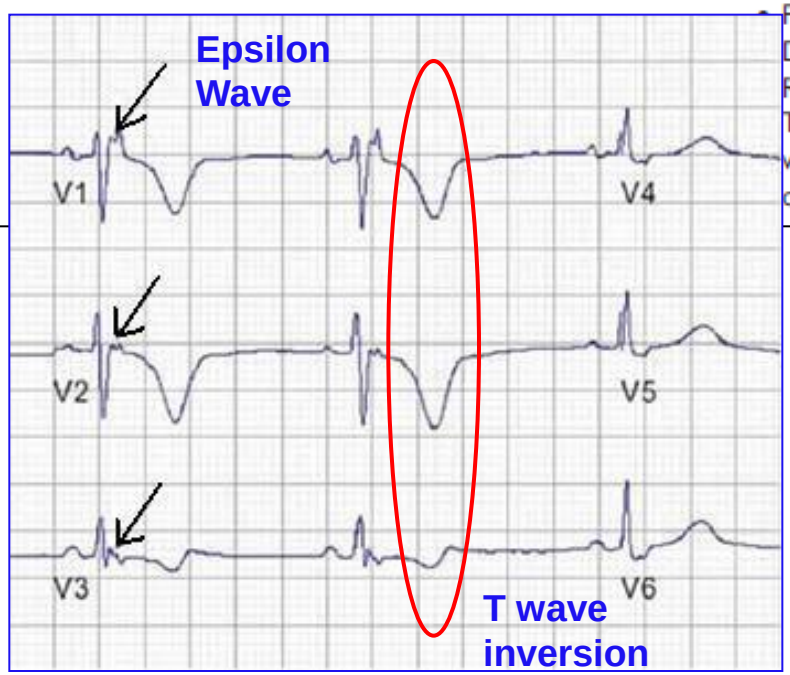
: **PKP2 gene mutation +ve**

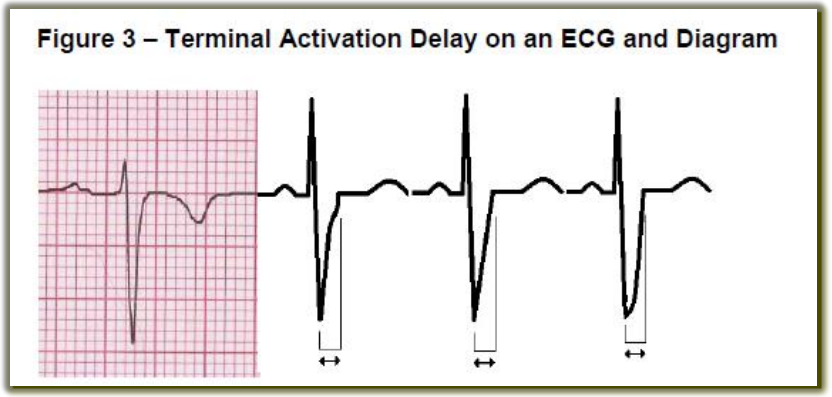


SMVT with LBBB morpholgy

ECG Features of ARVC

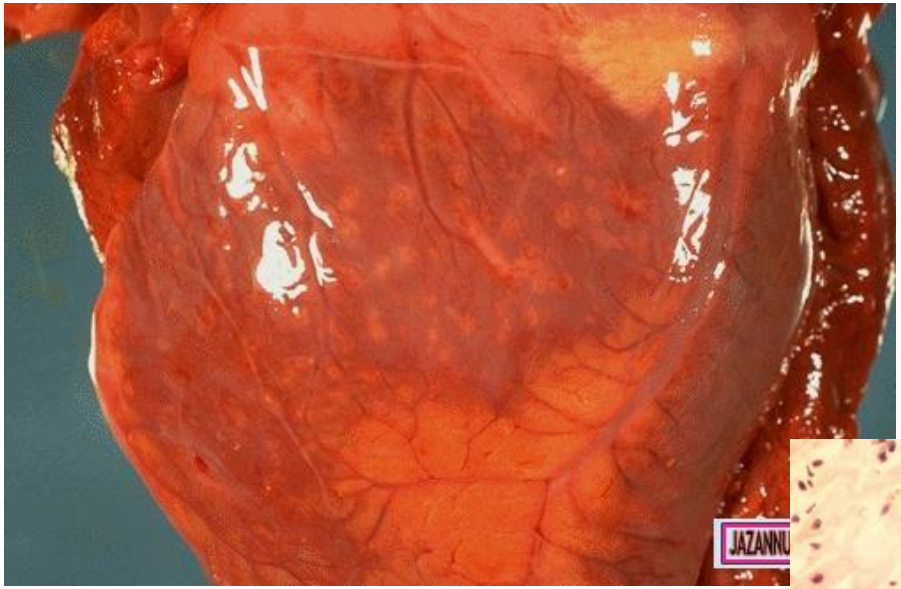
Original task force criteria	Revised task force criteria
IV. Depolarization/conduction abnormalities	
Major	
• Epsilon waves or localized prolongation (>110 ms) of the QRS complex in right precordial leads (V_1 to V_3)	• Epsilon wave (reproducible low-amplitude signals between end of QRS complex to onset 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



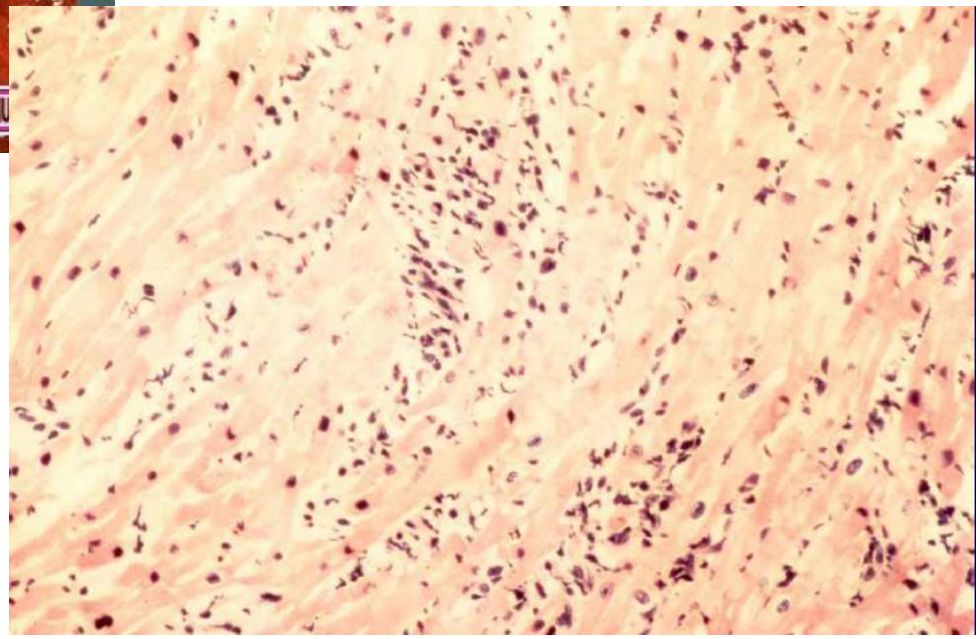


TAD >55ms

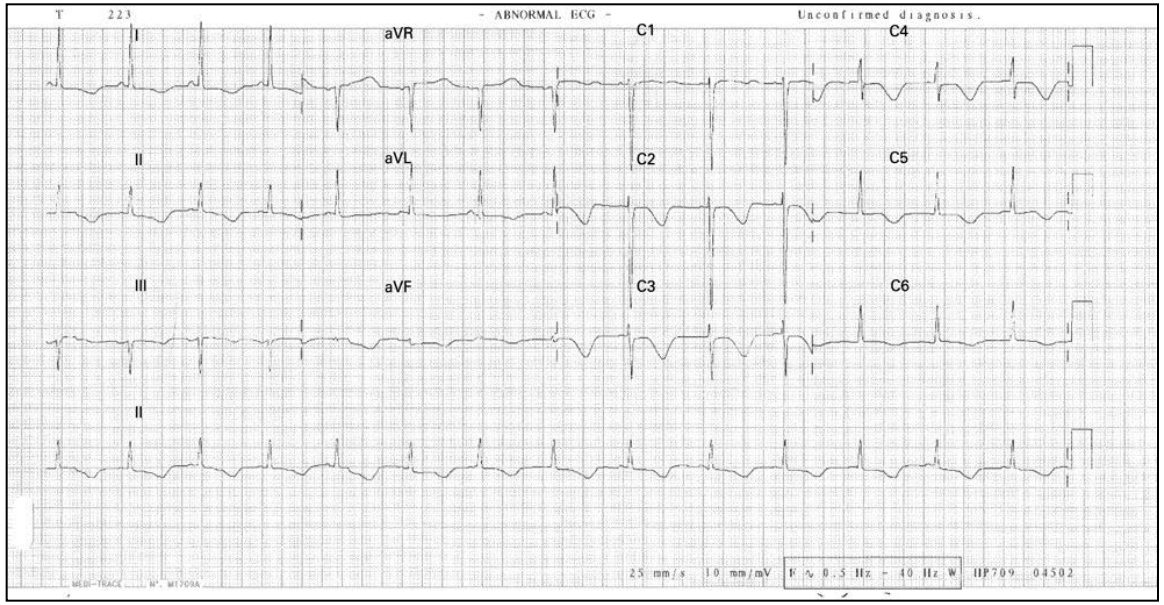
Myocarditis



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



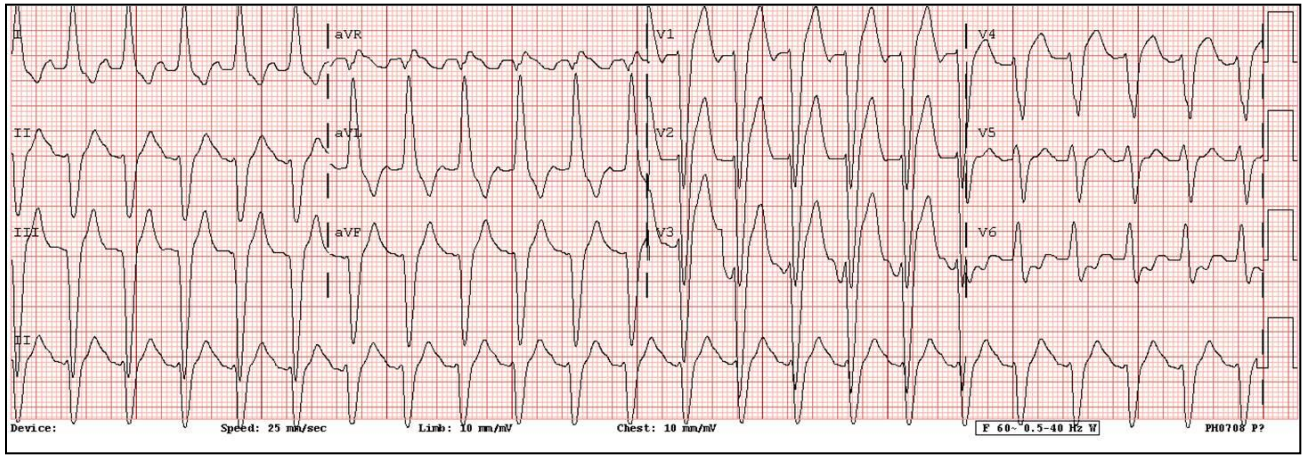
ECG features of myocarditis



T wave inversion in acute myocarditis

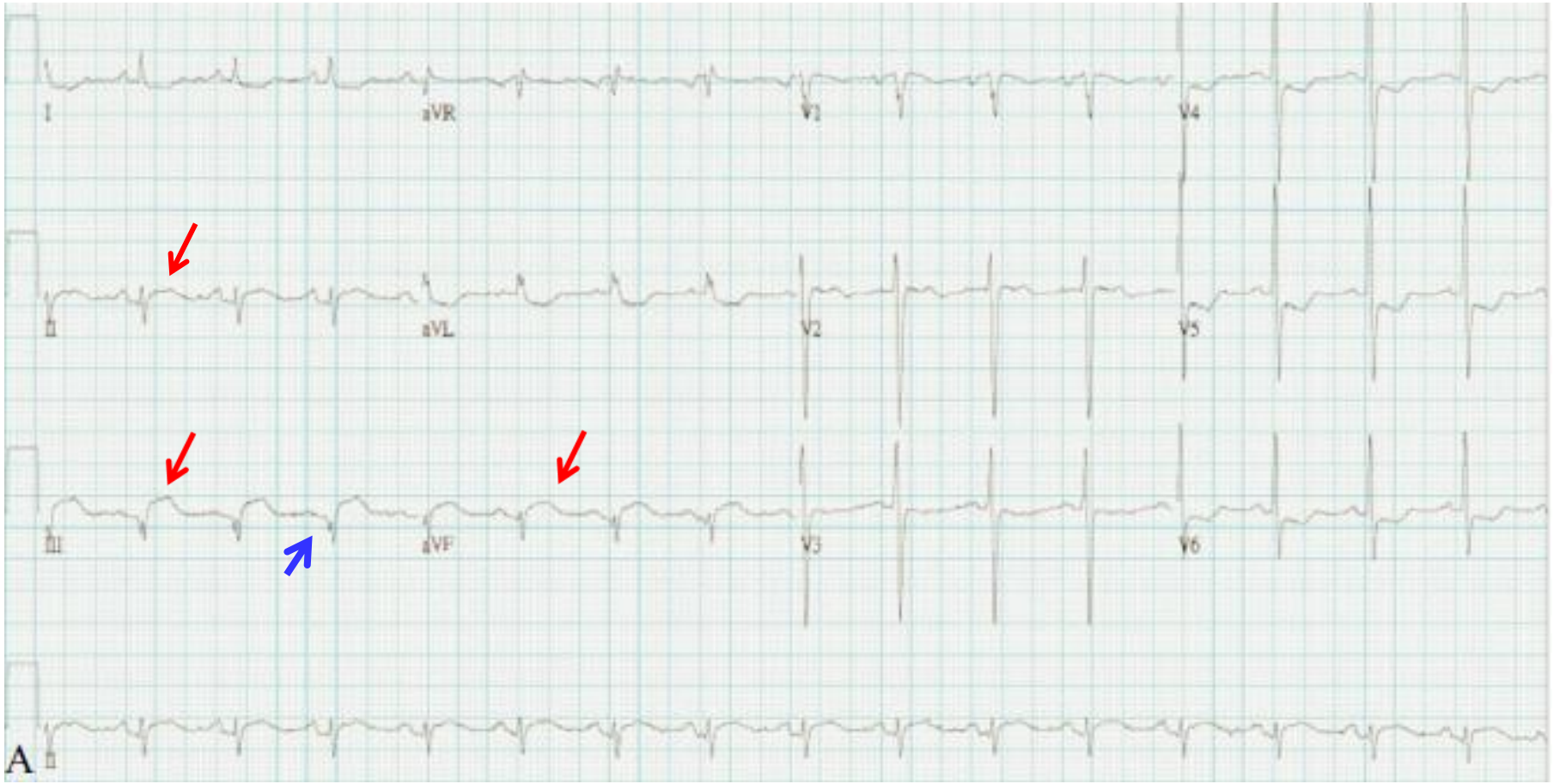
- T wave inversion
- Heart block (AV block or LBBB)
- Q wave
- ST segment elevation
- Prolonged QT interval
- Ventricular arrhythmia

LBBB

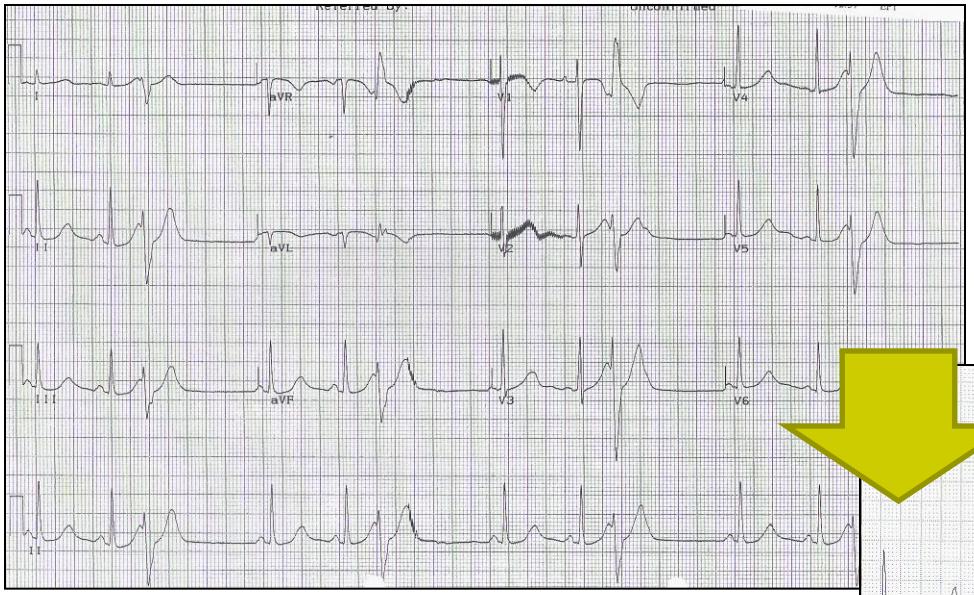


Acute Viral Myocarditis Mimicking ST Elevation Myocardial Infarction: Manifestation on Cardiac Magnetic Resonance

Acta Cardiol Sin 2010;26:44–7

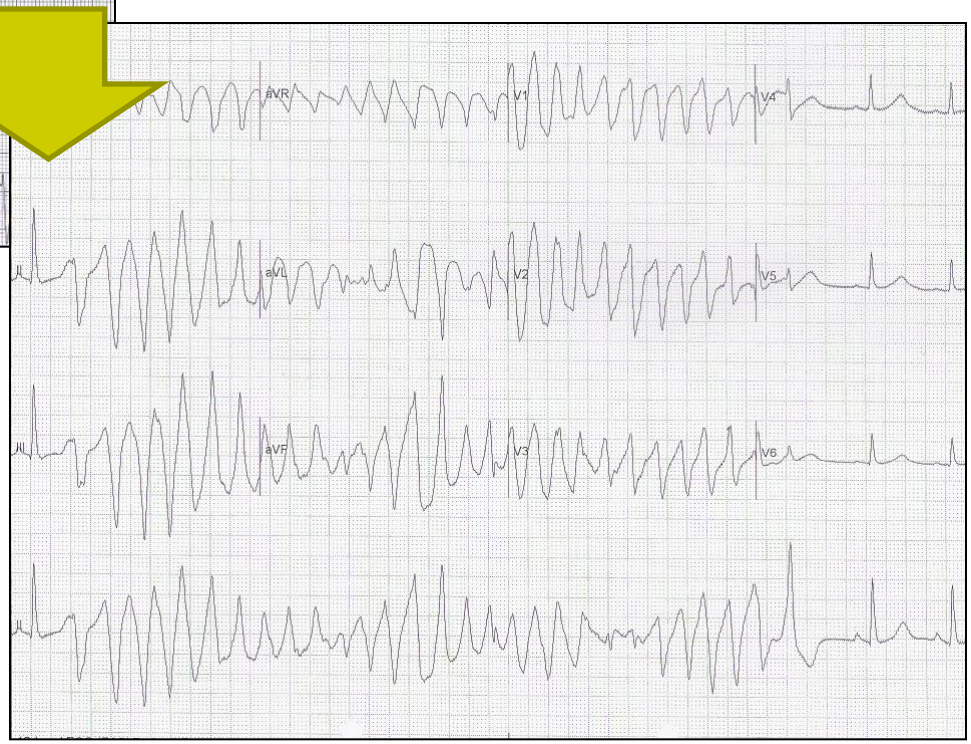


A Chinese lady presenting with long QTc & TdP



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

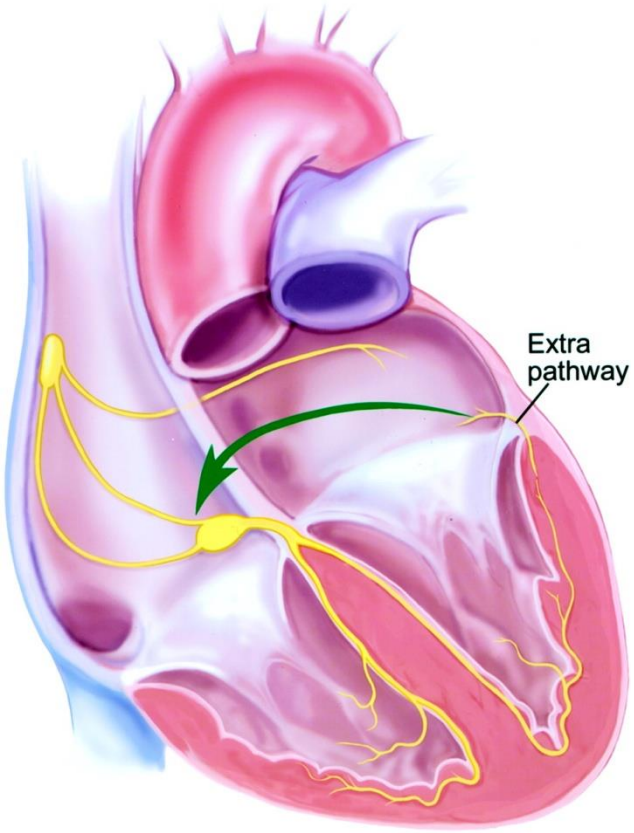
QTc 510ms

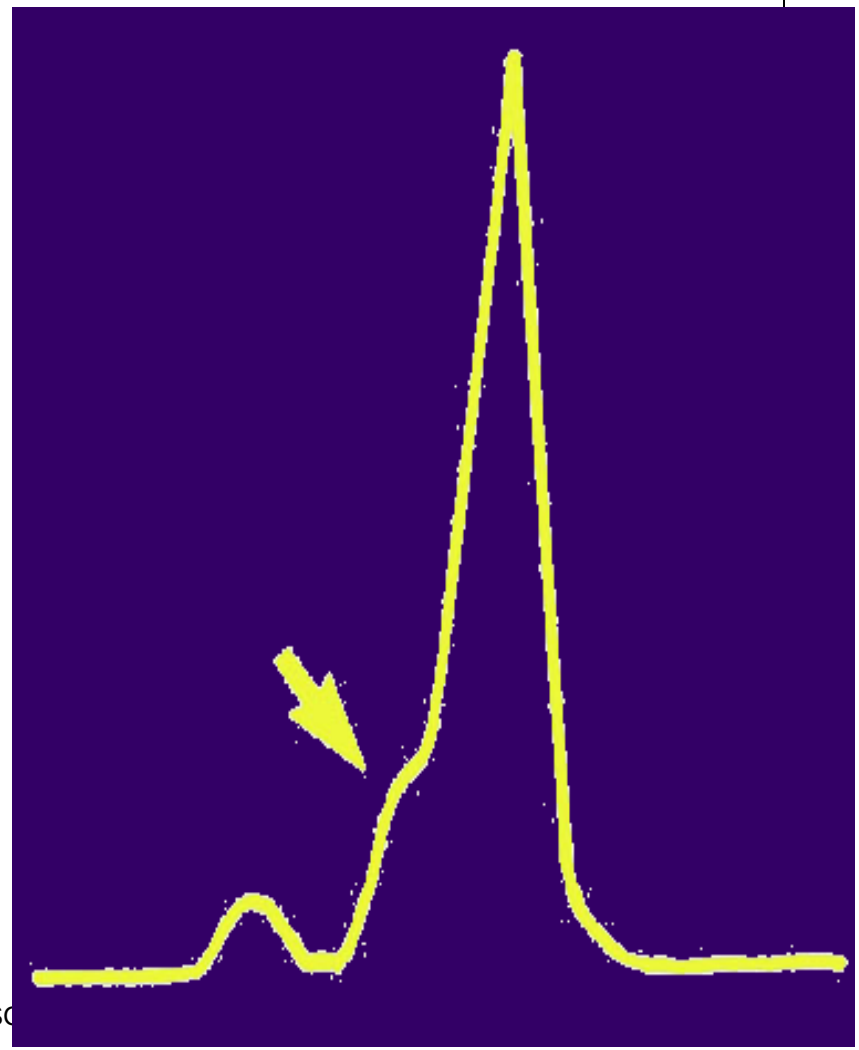


TdP in CCU

WPW syndrome

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





AF in WPW – Risk of SCD

A case with Wolf-Parkinson-White syndrome first presented with a devastating event: aborted sudden cardiac death

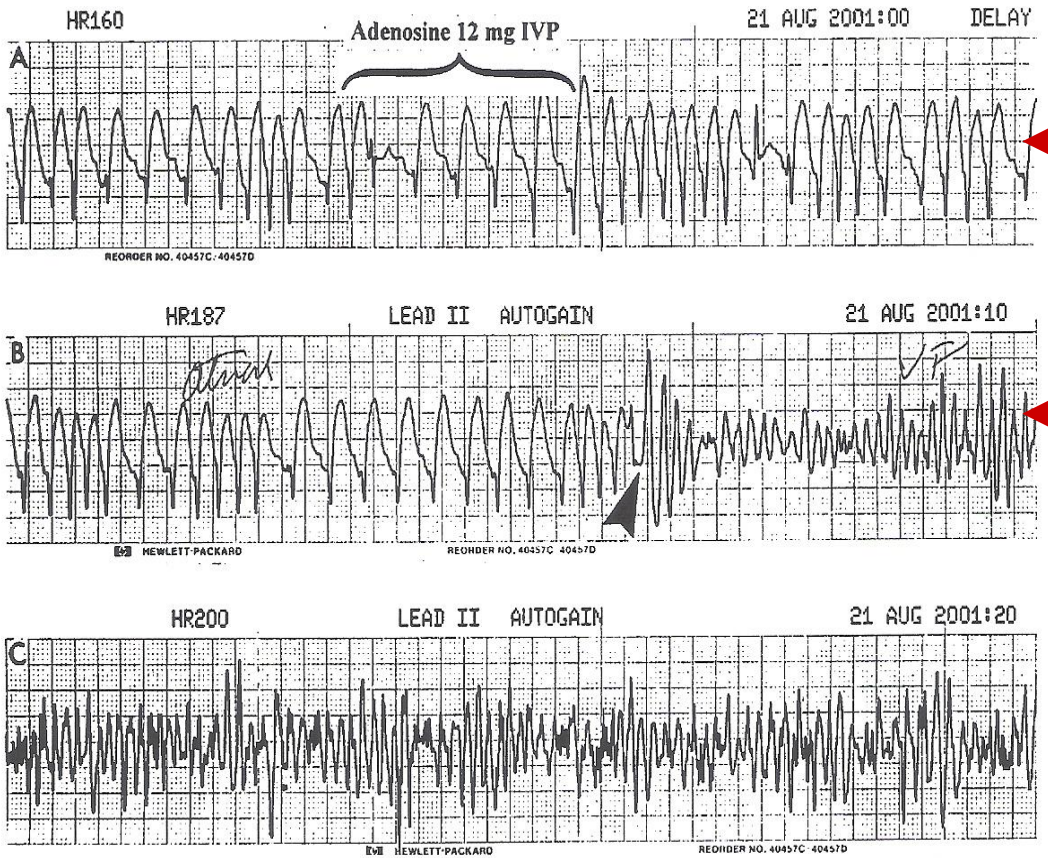
- Incidence of SCD in WPW syndrome 2/1000 patient-years
- Mechanism: rapid AF degenerating into VF
- AV nodal blockers (digoxin, verapamil, β-blockers, adenosine) may accelerate AF & induce VF

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



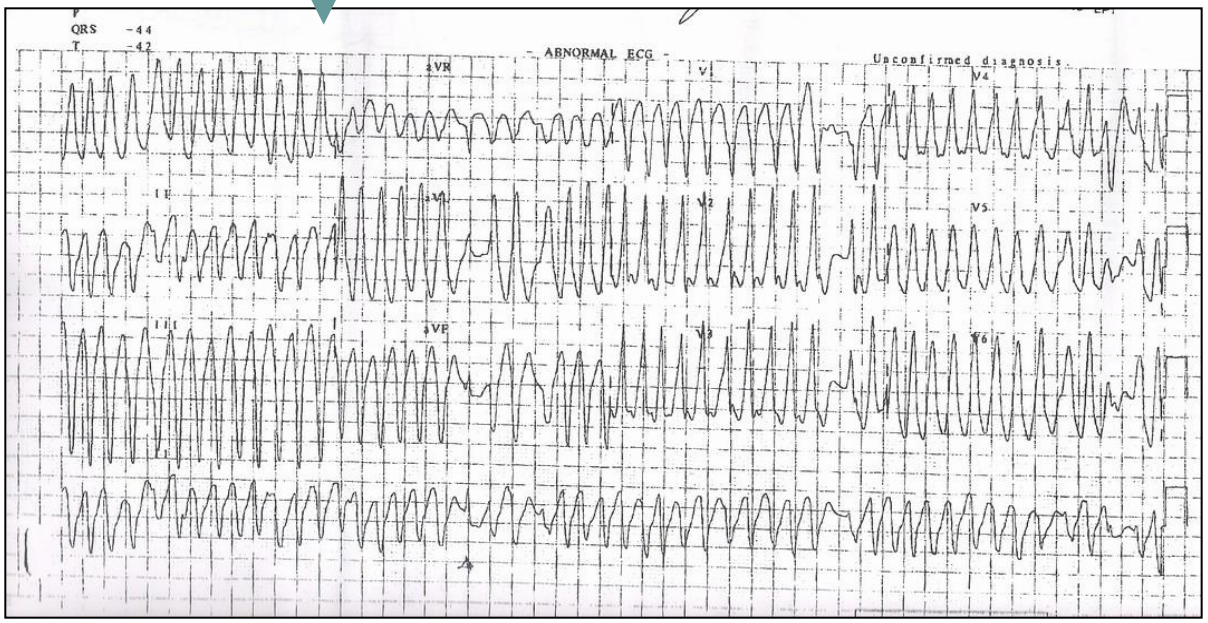
Pre-excited AF

Adenosine-induced VF

Preexcited AF in WPW syndrome

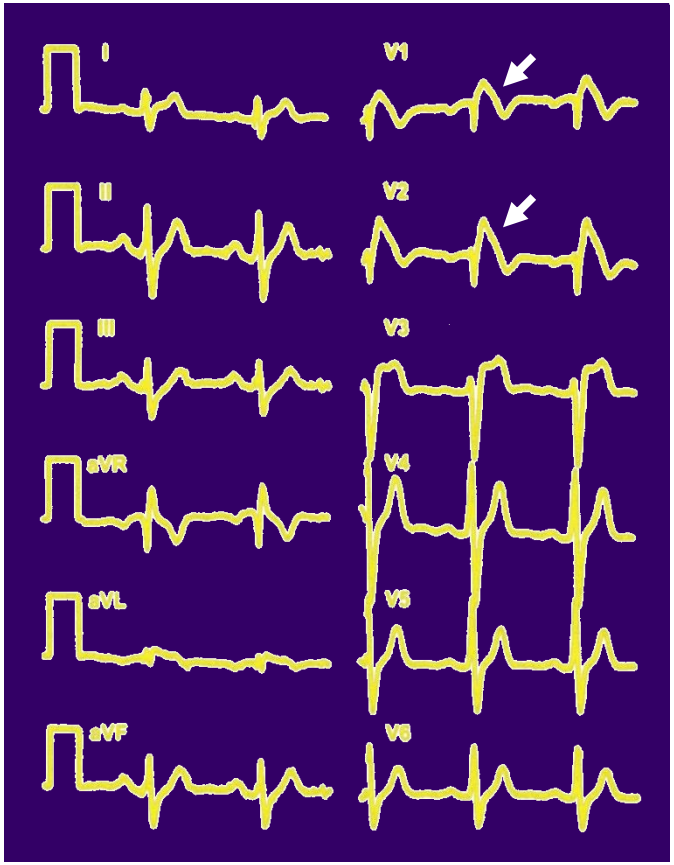
F : **Fast** **B**: Broad **I**: Irregular

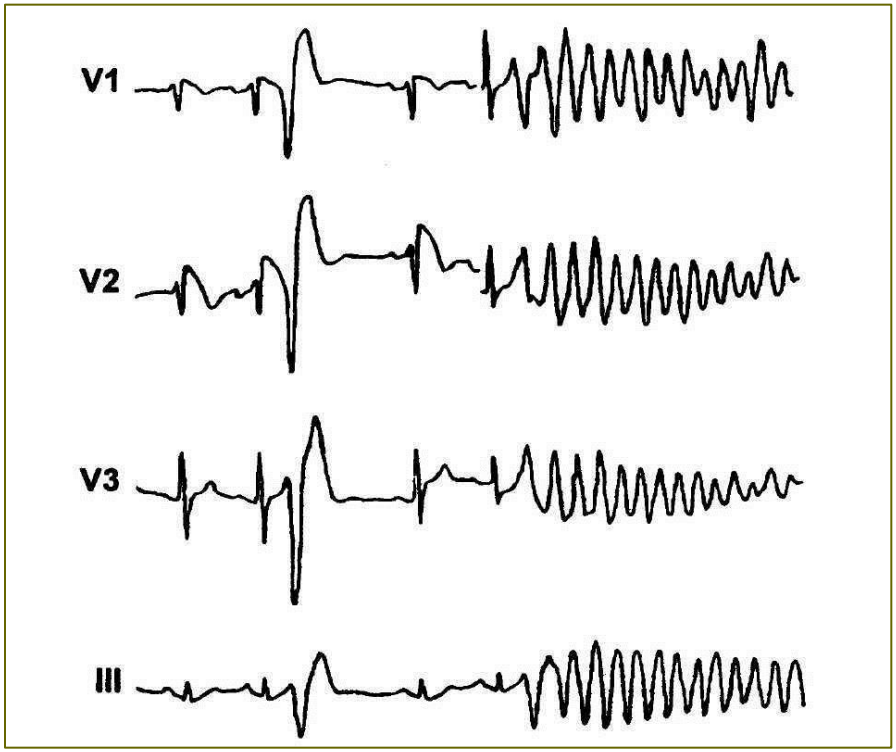
Preexcited AF with
rapid ventricular response



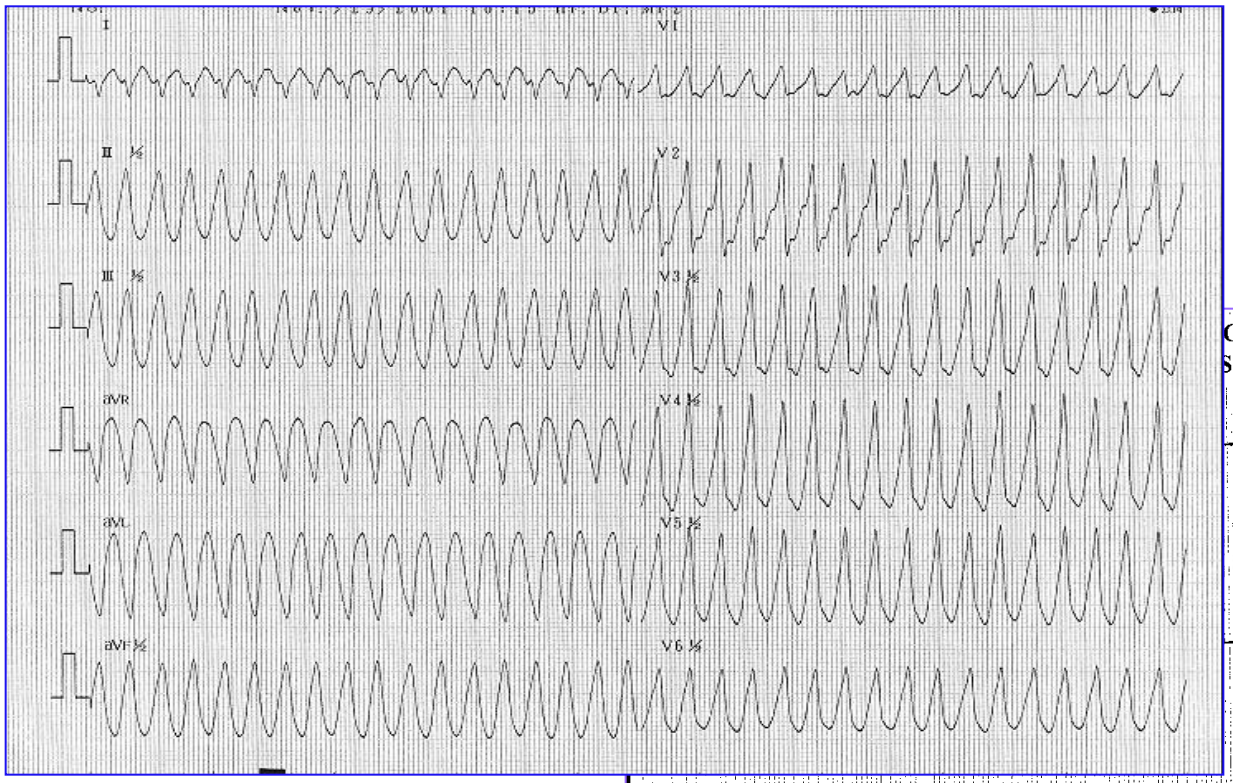
Brugada Syndrome

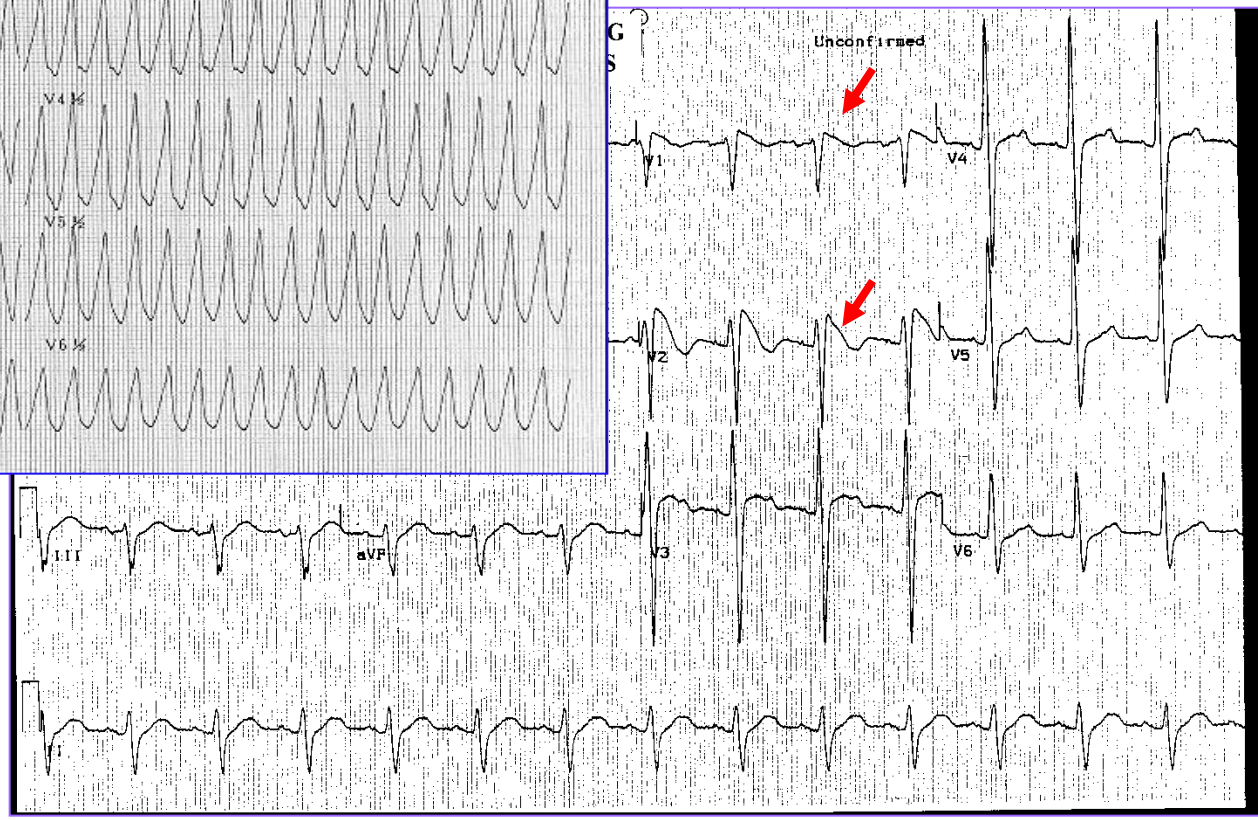
- In 1992, Pedro and Josep Brugada first reported a new syndrome in 8 **SCD** patients with **ST segment elevation in V1-V3 but no structural heart diease**
- Known as "**Brugada syndrome**" since 1996
- Prone to develop **VF / PVT** leading to SCD





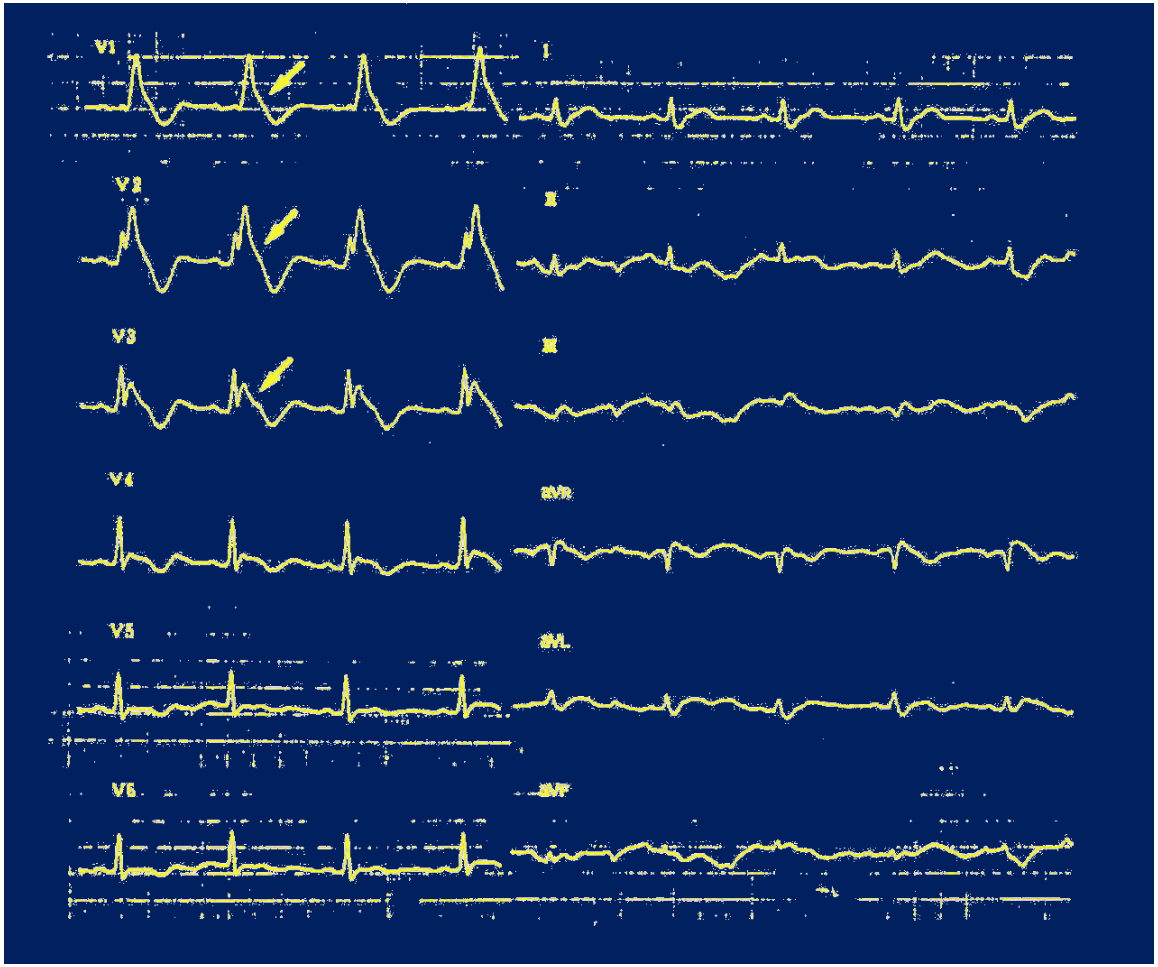
BrS presenting with SMVT





Mok NS, et al. IJC 2003

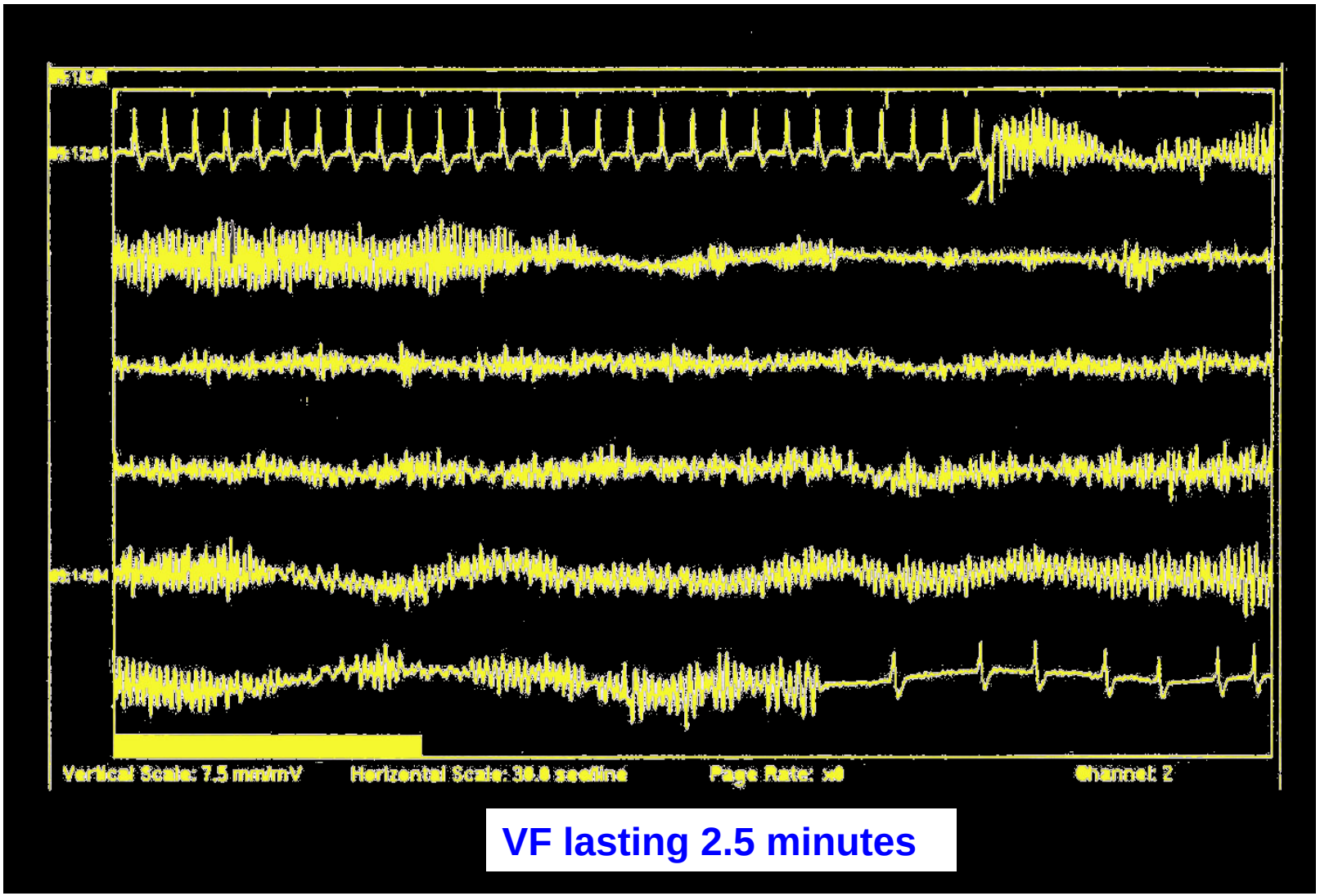
1st HK Chinese with Brugada syndrome -



12-lead ECG showing RBBB pattern & ST elevation in V1-V3

Mok NS, et al. JHKCC 1999

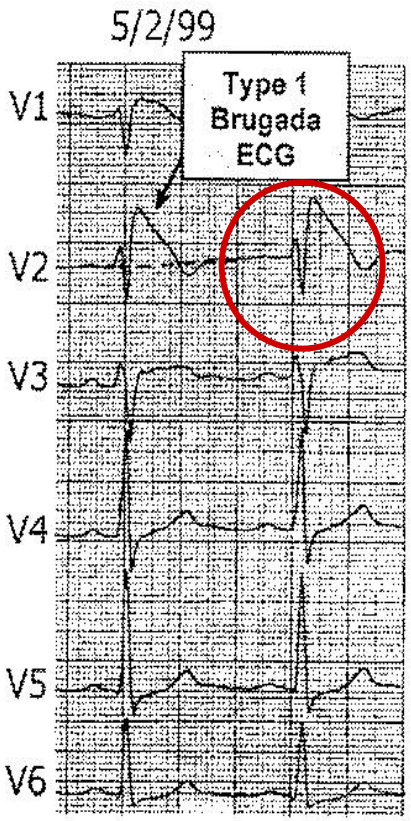
Holter recording of a self-terminating VF during convulsion



VF lasting 2.5 minutes

Mok NS, et al JHKCC 1999

Diagnosis of BrS

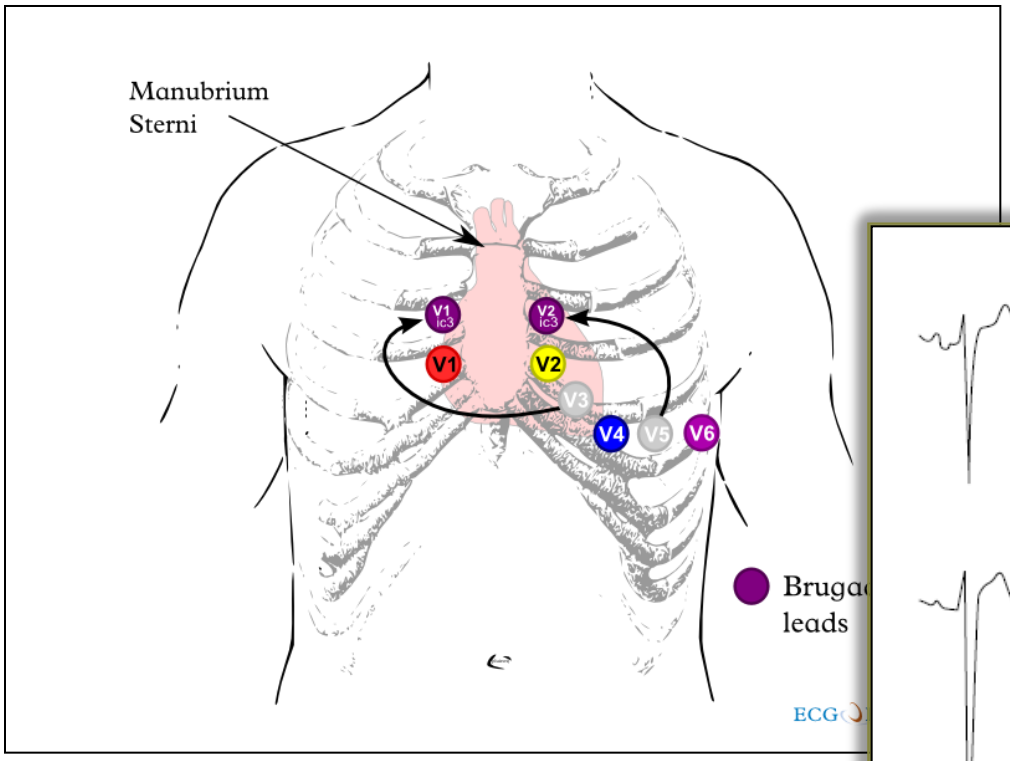


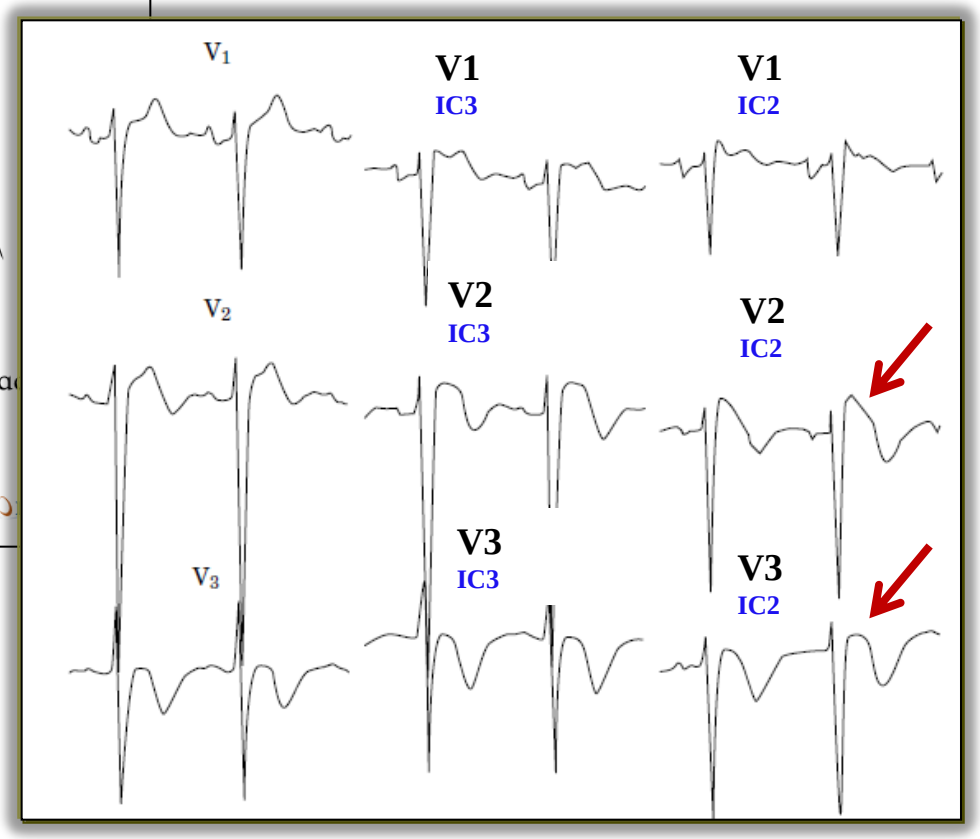
Diagnosis of Brugada Syndrome

Recommendations	Class[a]	Level[b]	Ref.[c]
Brugada syndrome is diagnosed in patients with ST-segment elevation with type 1 morphology ≥ 2 mm in one or more leads among the right precordial leads V1 and/or V2 positioned in the second, third, or fourth intercostal space, occurring either spontaneously or after provocative drug test with intravenous administration of sodium channel blockers (such as ajmaline, flecainide, procainamide or pilsicainide).	I	C	This panel of experts

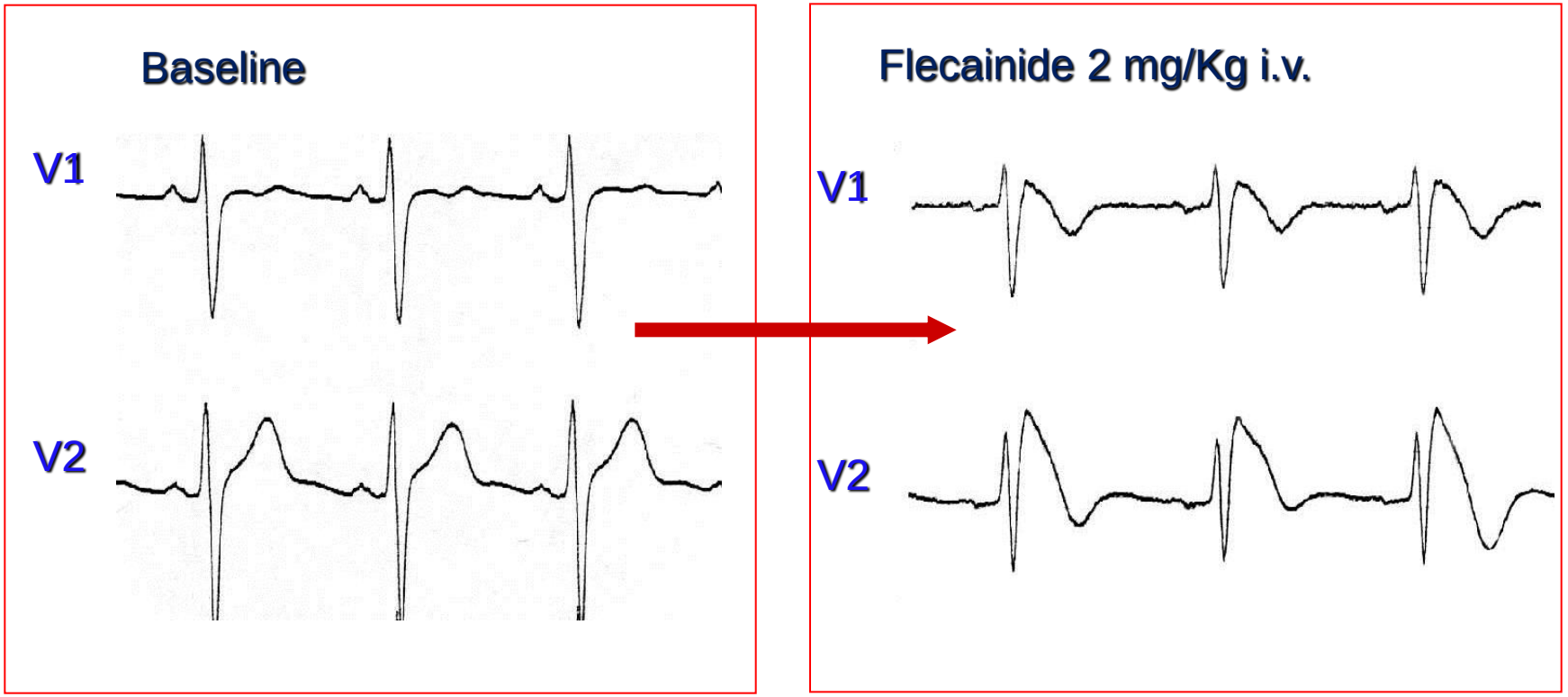
ESC Guidelines 2015

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





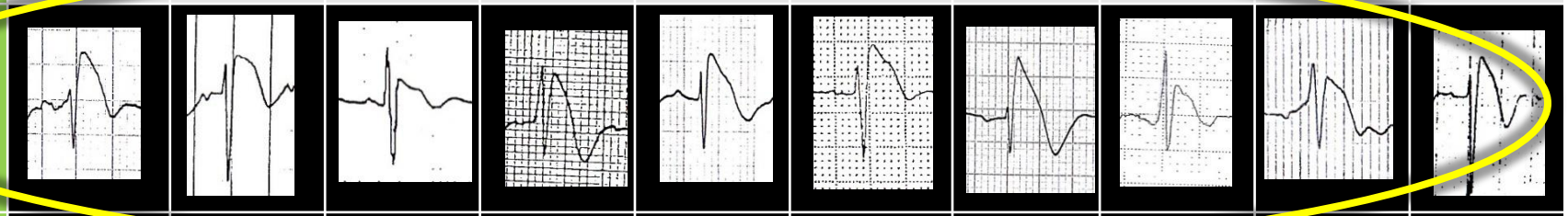
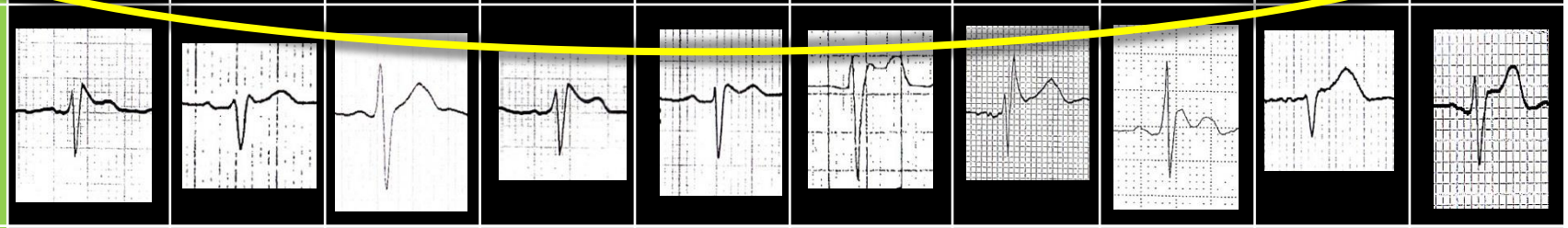
Flecainide provocation test in BrS



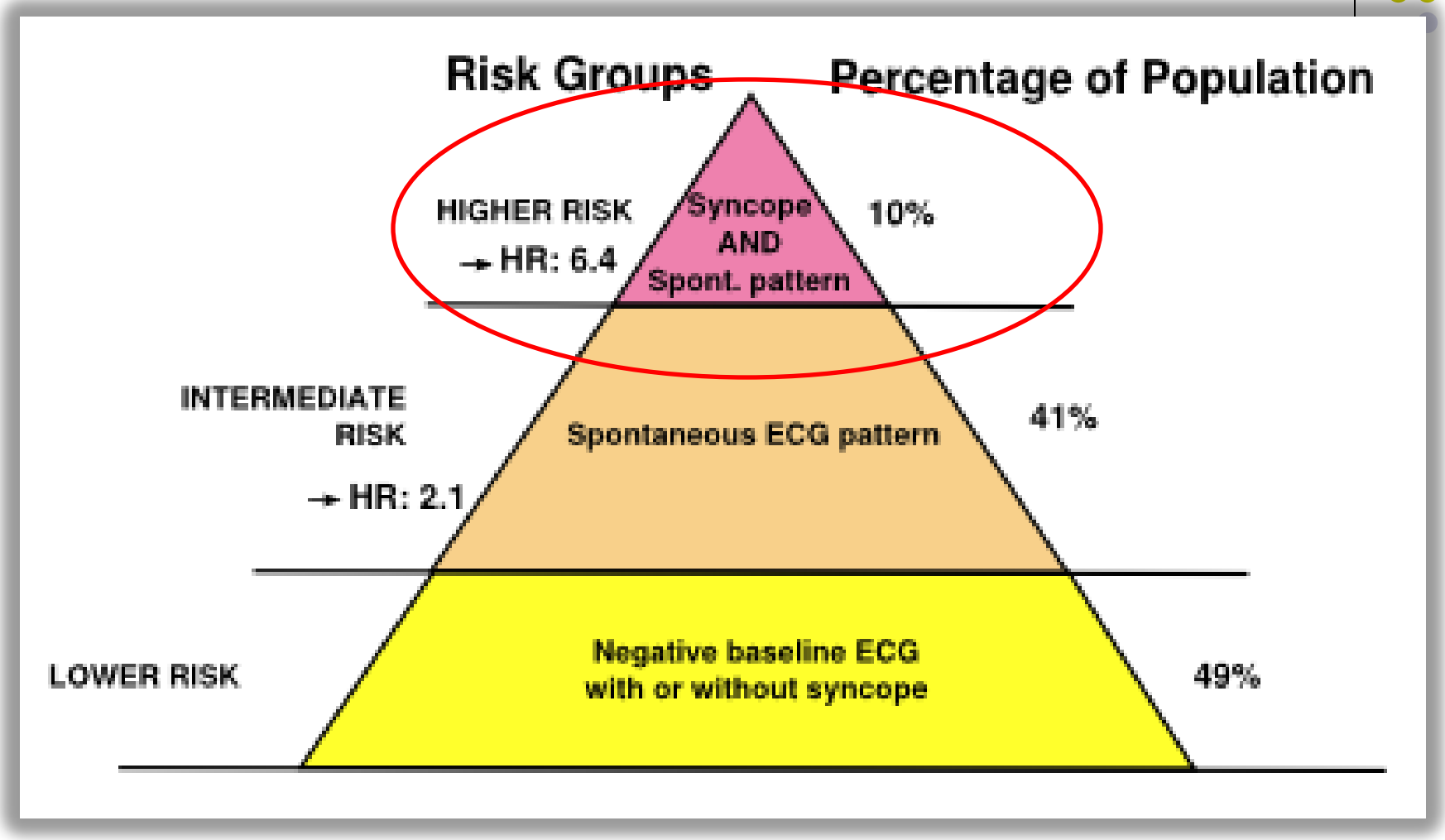
Type 1 Brugada ECG pattern unmasked by IV flecainide during family screening

Fever unmasking Type 1 Brugada ECG in 10 patients – A single centre experience in Hong Kong

Mok NS, et al, APHRS 2013

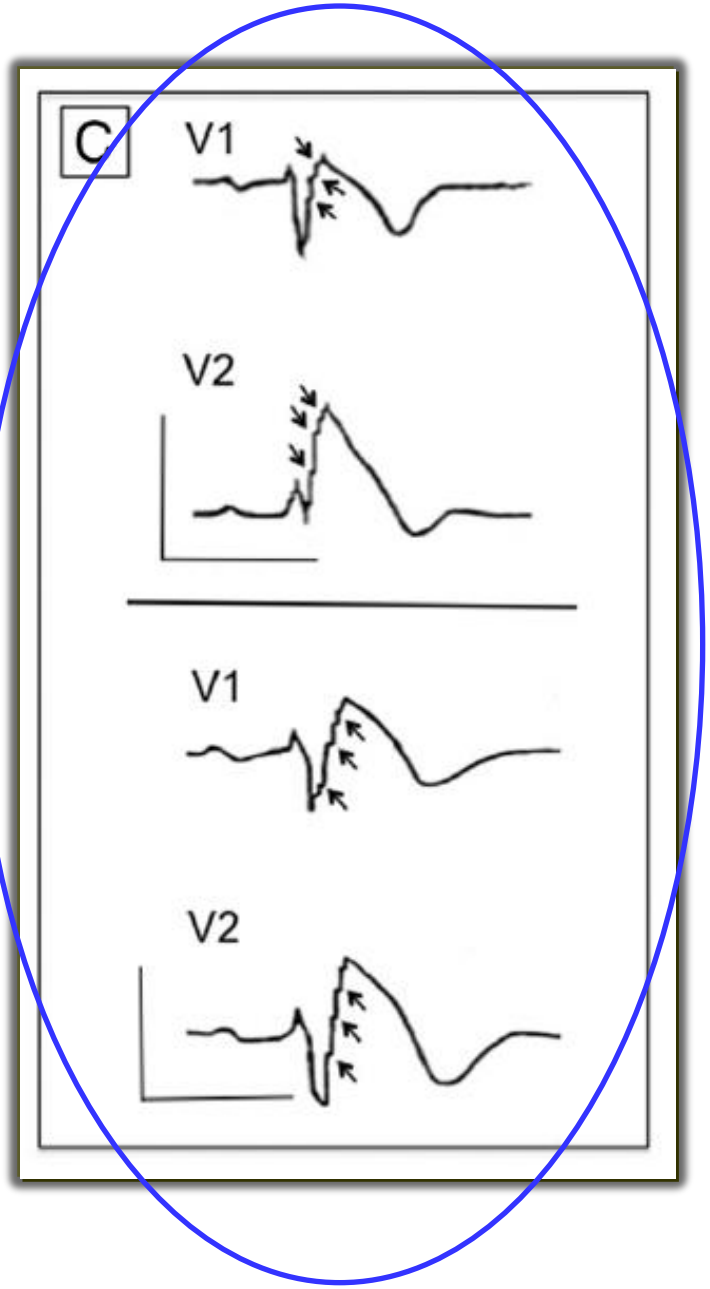
Patient	Ho TK	Wong KM	Chan TC	Lam Y	Wong HM	Cheng KB	Poon KB	Tsang MF	Liang LF	Choi PC
Max Temp (0C)	39.1	41.0	38.3	37.5	39.0	38.7	38.9	38.7	38.7	38.6
ECG V2 (Fever)										
ECG V2 (NO Fever)										
Δ J point (mm)	-4	-4	-4	-4	-4	-5	0	-3	-3	-5

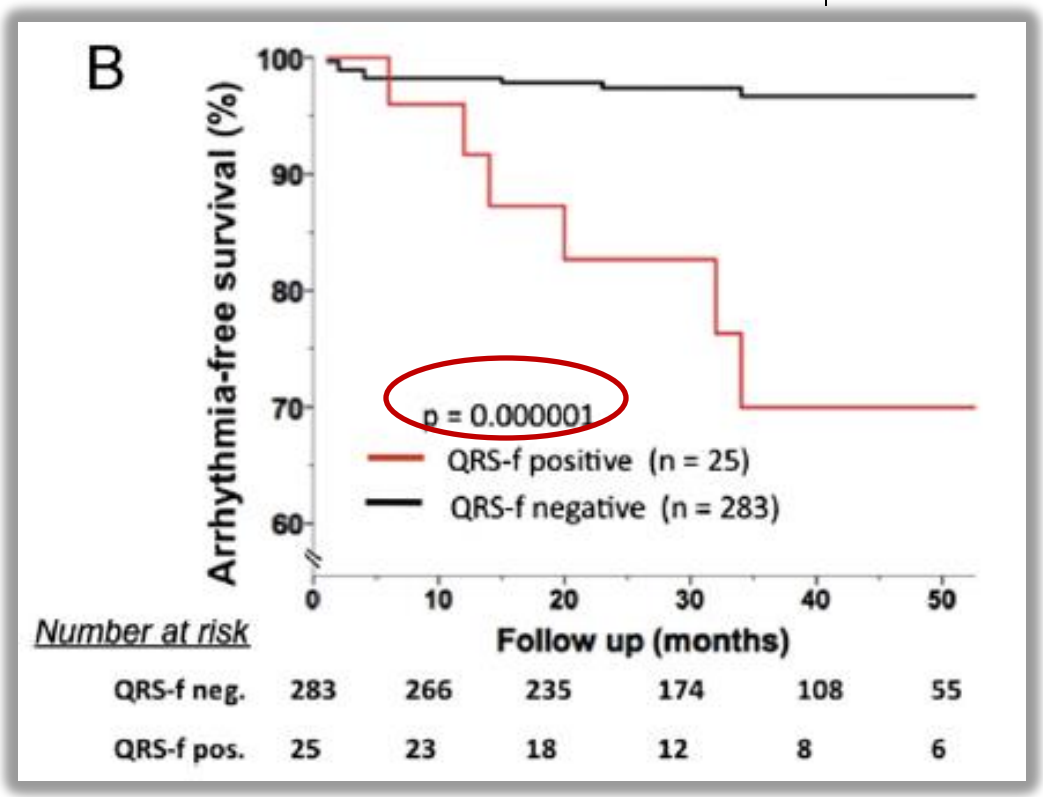
Risk factors and prognosis of BrS



Priori et al. Circulation 2002

Fragmentation of QRS predicts high risk in BrS





Priori et al. JACC 2012

Early Repolarization Syndrome (ERS)

Early repolarization (ER) manifesting as

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

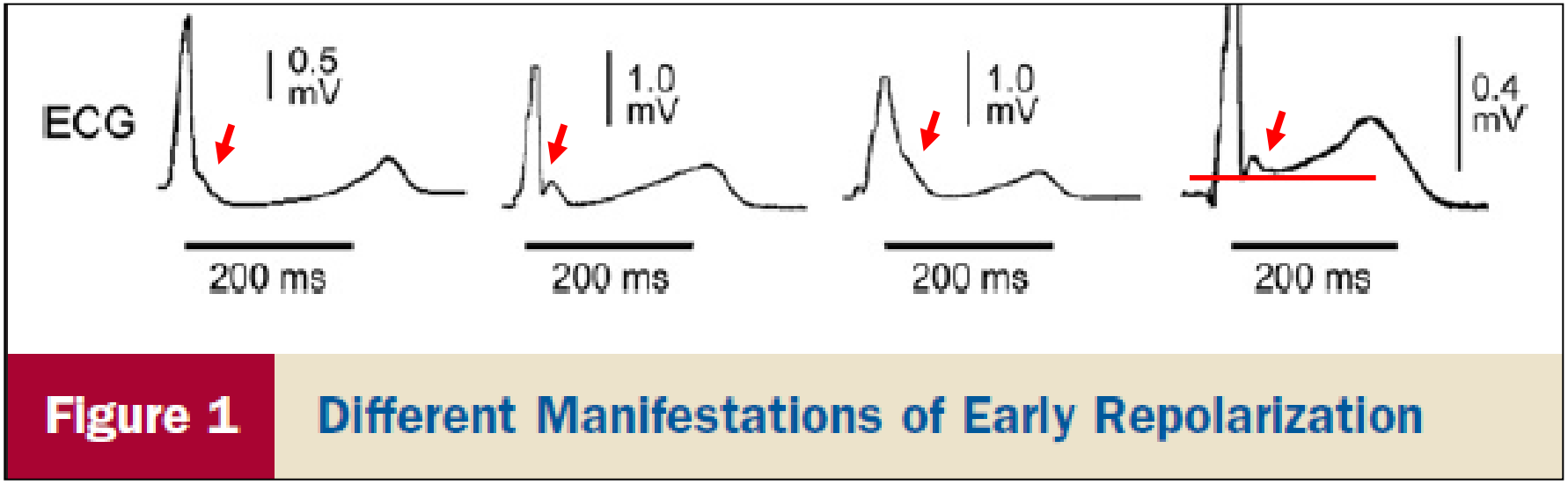


Figure 1 Different Manifestations of Early Repolarization

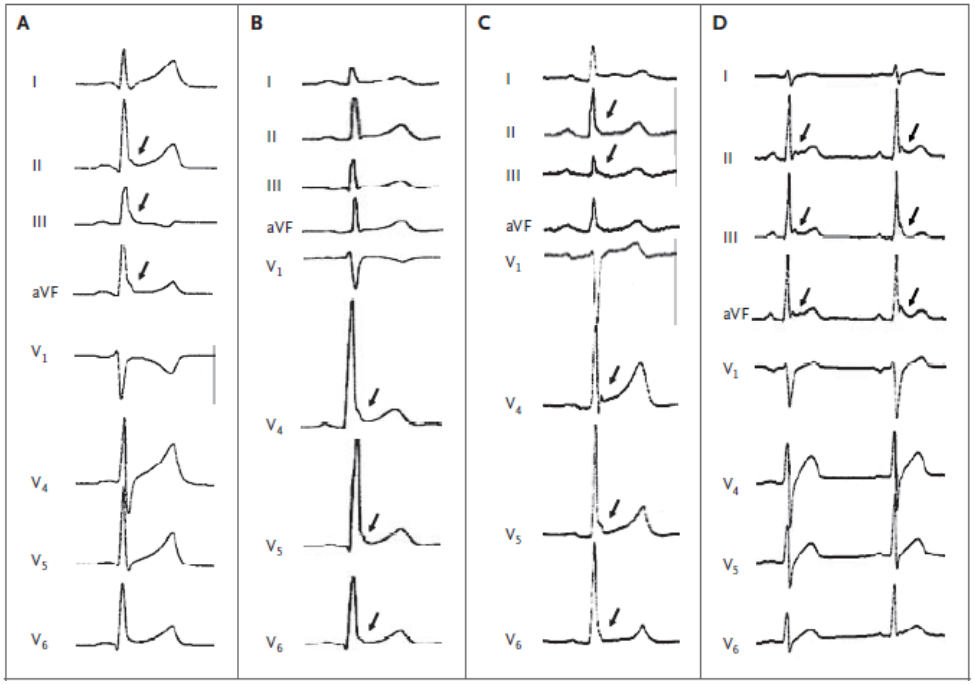
Early Repolarization Syndrome (ERS)

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

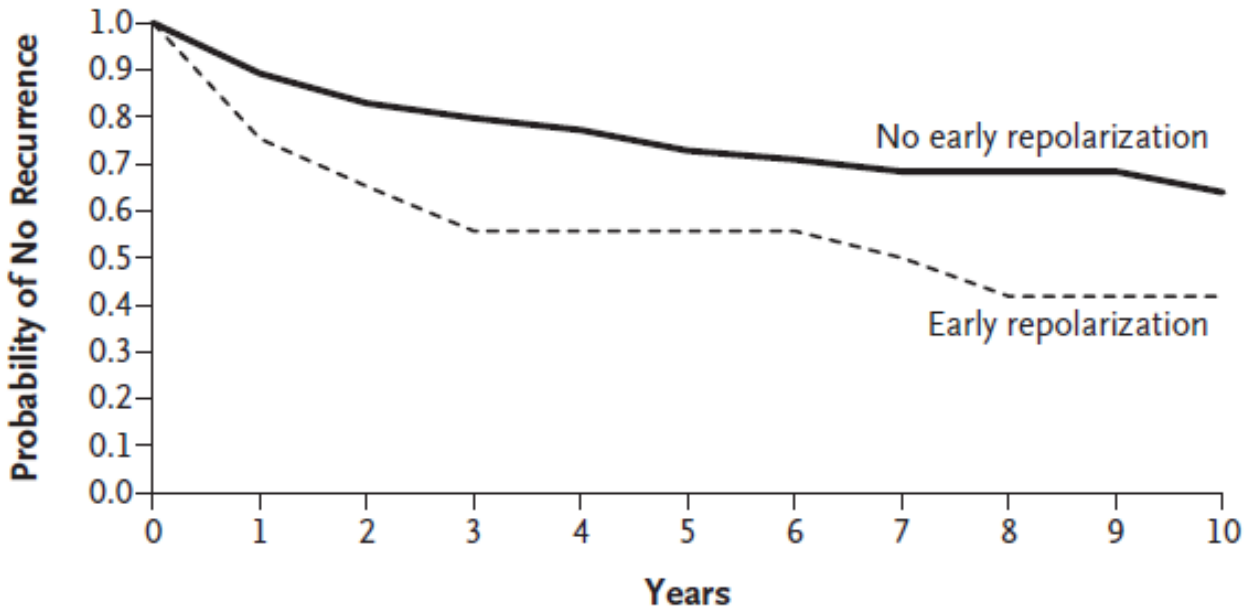
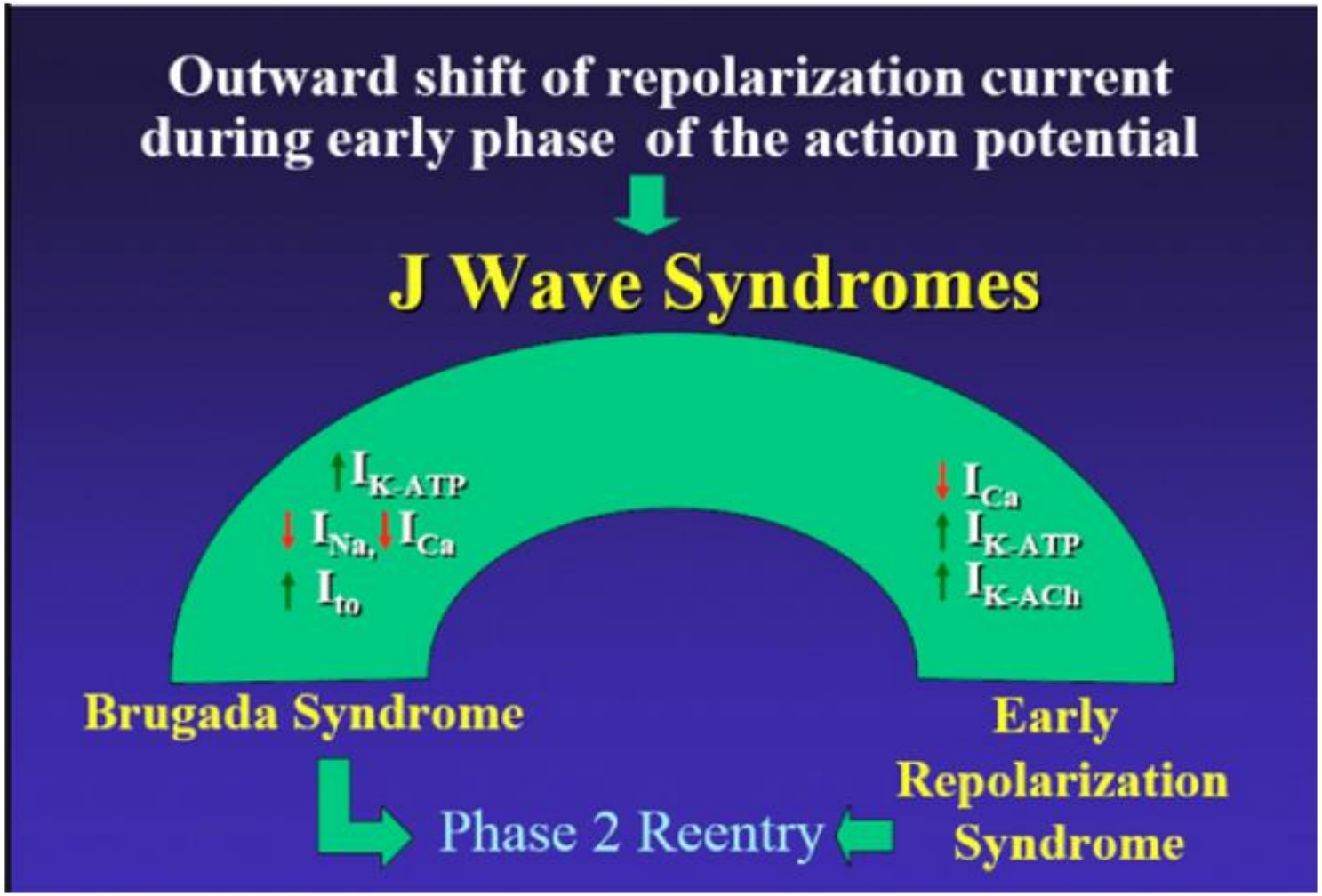


Figure 3. Actuarial Curves for Case Subjects, According to the Presence or Absence of Early Repolarization.

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

rly repolarization syndrome.

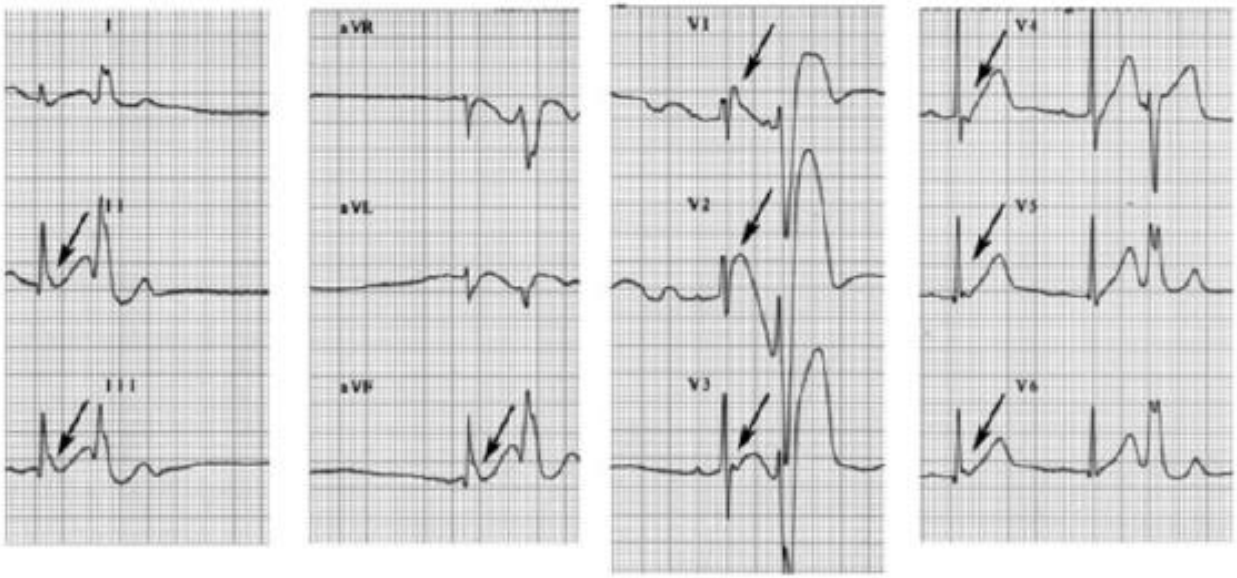
	Points
polymorphic VT	3
	2
/	1
is category	
leads with hor-	2
) in ≥ 2 inferior and/	1.5
and/or lateral ECG	1
is category	
or peak of T wave	2
	2
m	2
	1
a first- or second-	0.5
is category	
n	0.5
ding)	
RS	

ndromes: Similarities and differences

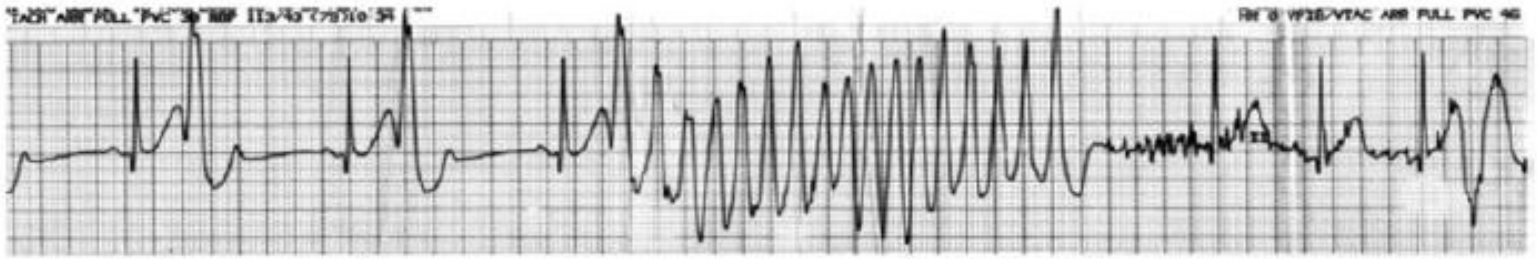
	Inherited			
	Early repolarization in lateral leads (ERS type 1)	Early repolarization in inferior or inferolateral leads (ERS type 2)	Global early repolarization (ERS type 3)	Brugada syndrome
hief ic	Anterolateral left ventricle	Inferior left ventricle	Left and right ventricles	Right ventricle
oint/ lities	I, V_4–V_6	II, III, aVF	Global	V_1–V_3
ıse	Increase	Increase	Increase	Increase
	Little or no change	Little or no change	Little or no change	Increase
	Male	Male	Male	Male

J Wave syndrome – ERS Type 3

C. 10AM Aug. 18 2003



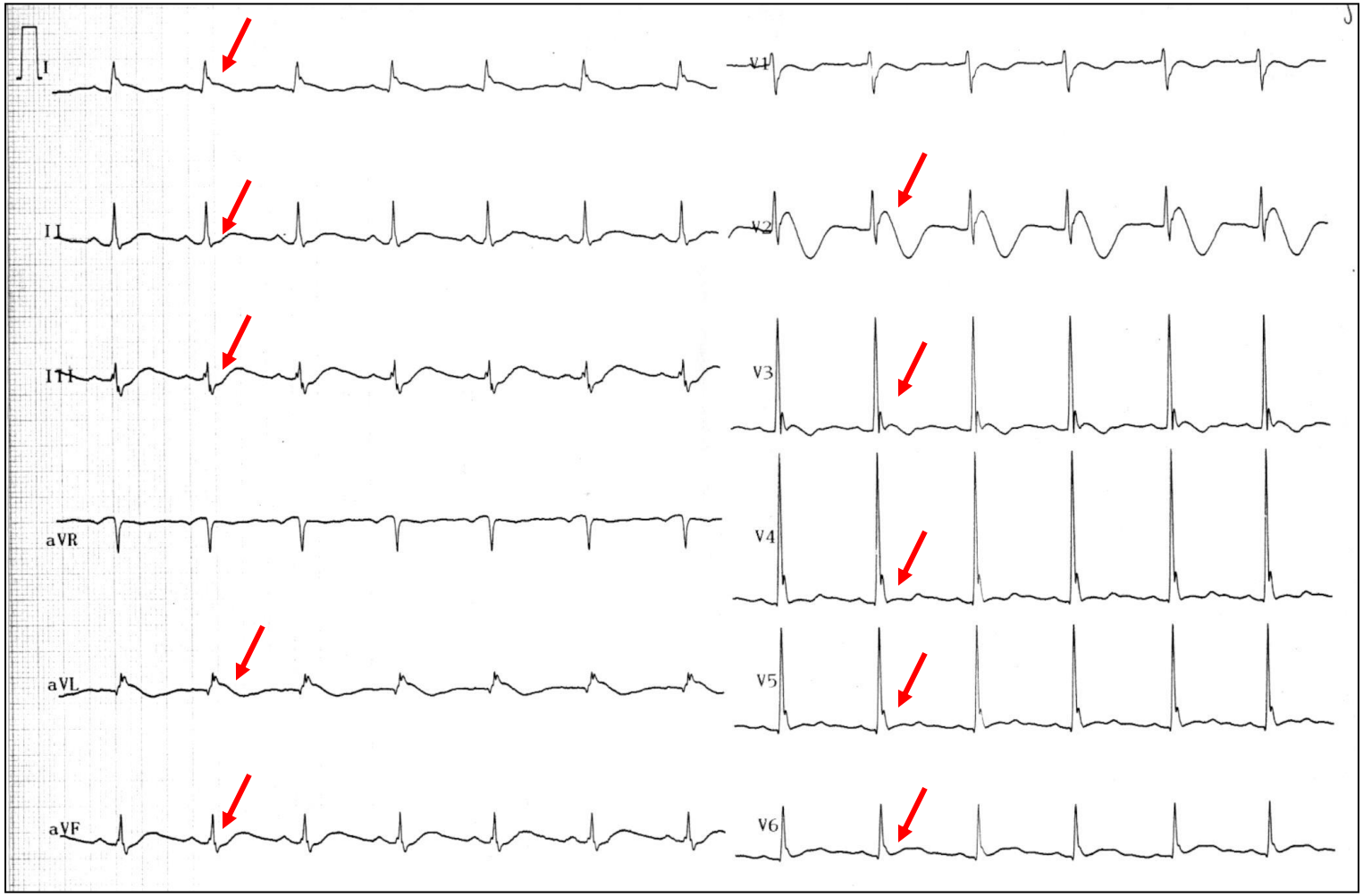
D. 10:46AM Aug. 18 2003



J Wave syndrome – ERS Type 3 in a HK Chinese

M/17 Survivor of a VF storm

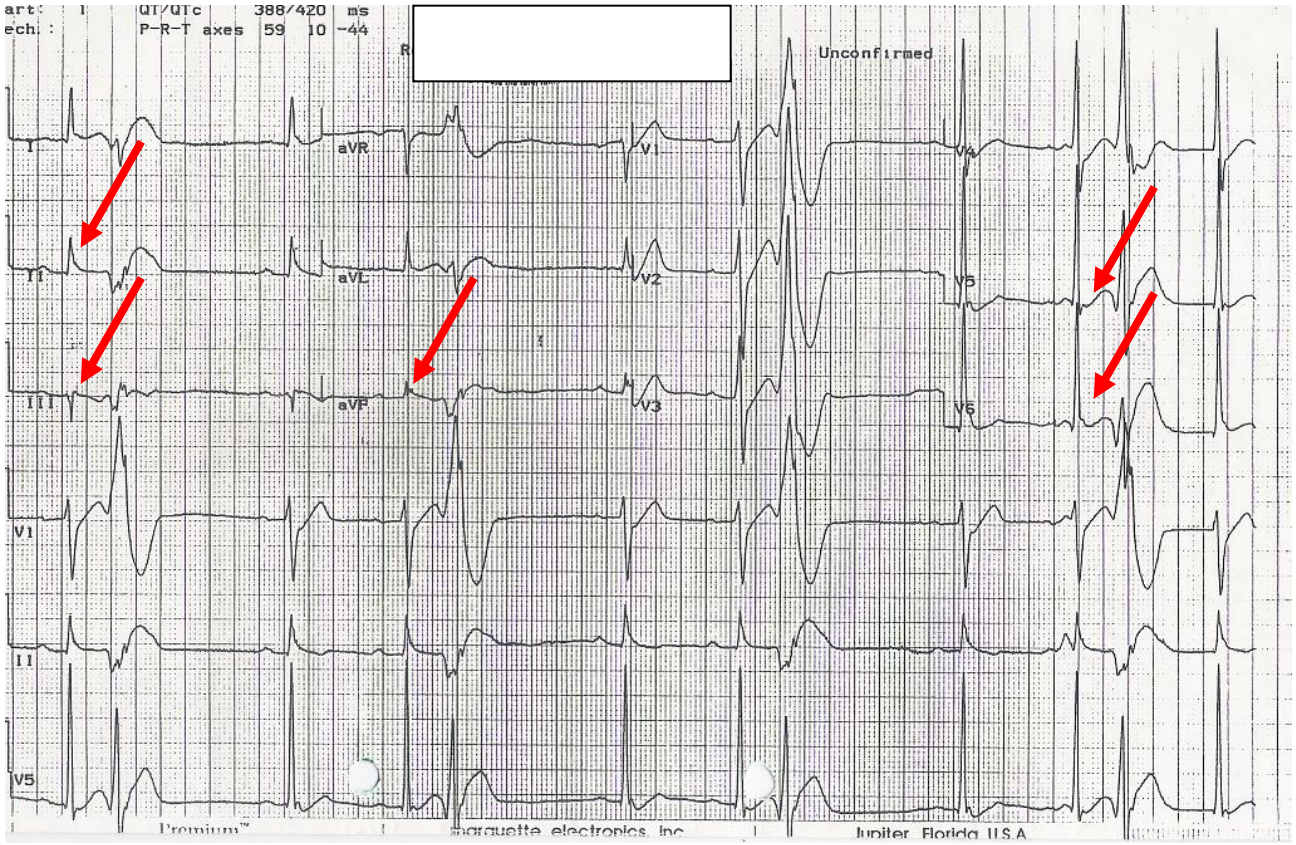
J waves over limb and precordial leads



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

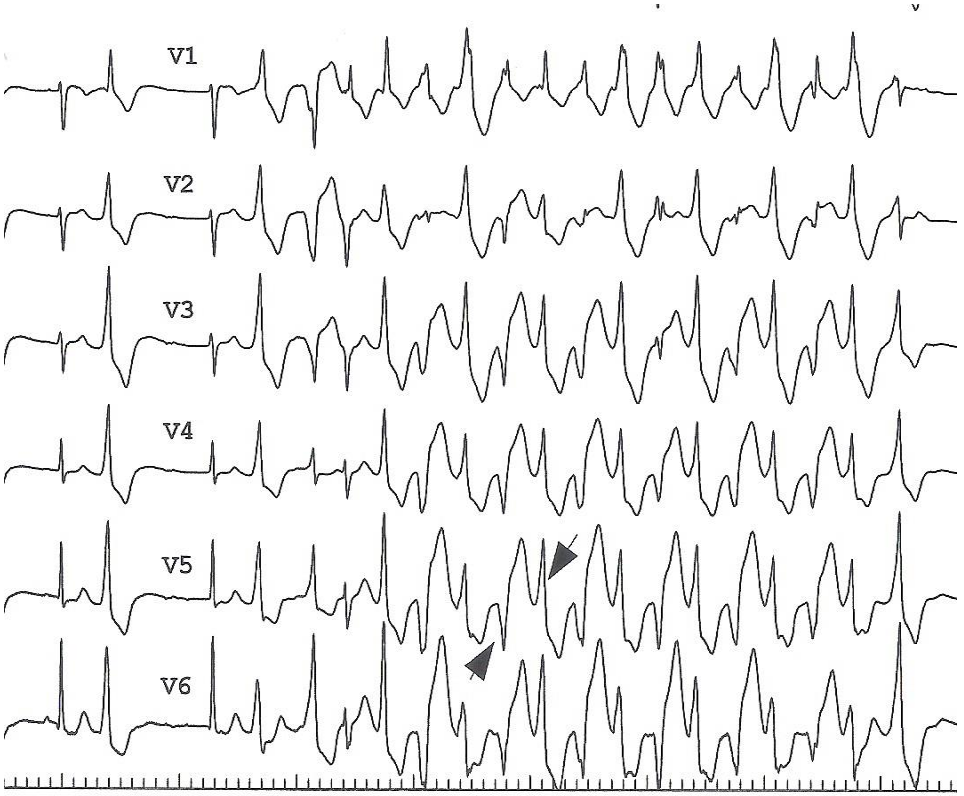
M/ 55

Recurrent VF cardiac arrest survivor



Catecholaminergic polymorphic VT (CPVT)

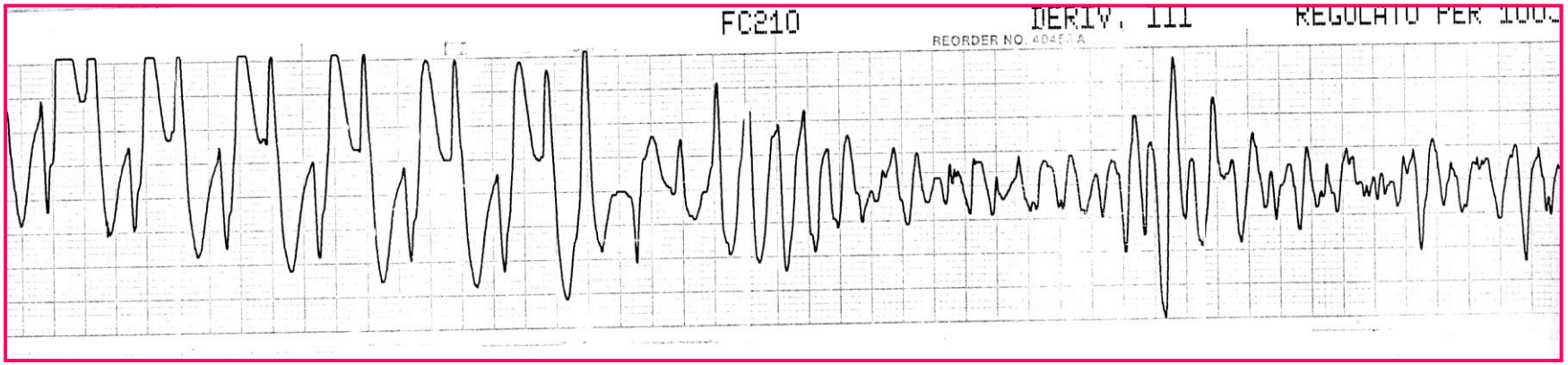
- **Stress or exercise-induced biVT / pVT / VF causing SCD**
- **Manifest in childhood or adolescence with 30-50% mortality by age 30**



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

Catecholaminergic Bi-directional VT degenerating into VF

P.G, female, 9yrs

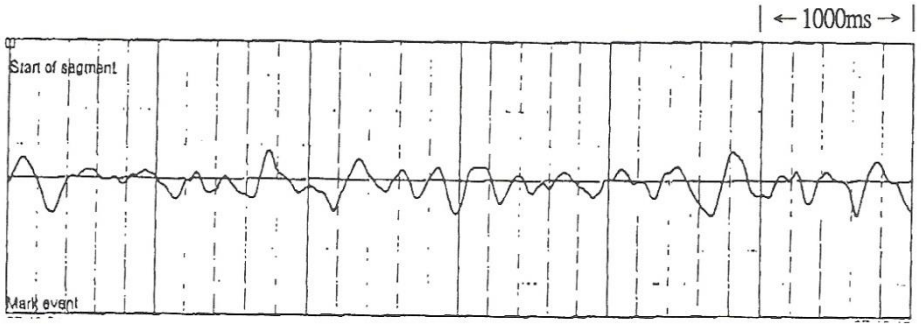


CPVT- A newly-recognized cause of SCD and syncope in Hong Kong Chinese

Mok NS et al , CMJ 2006

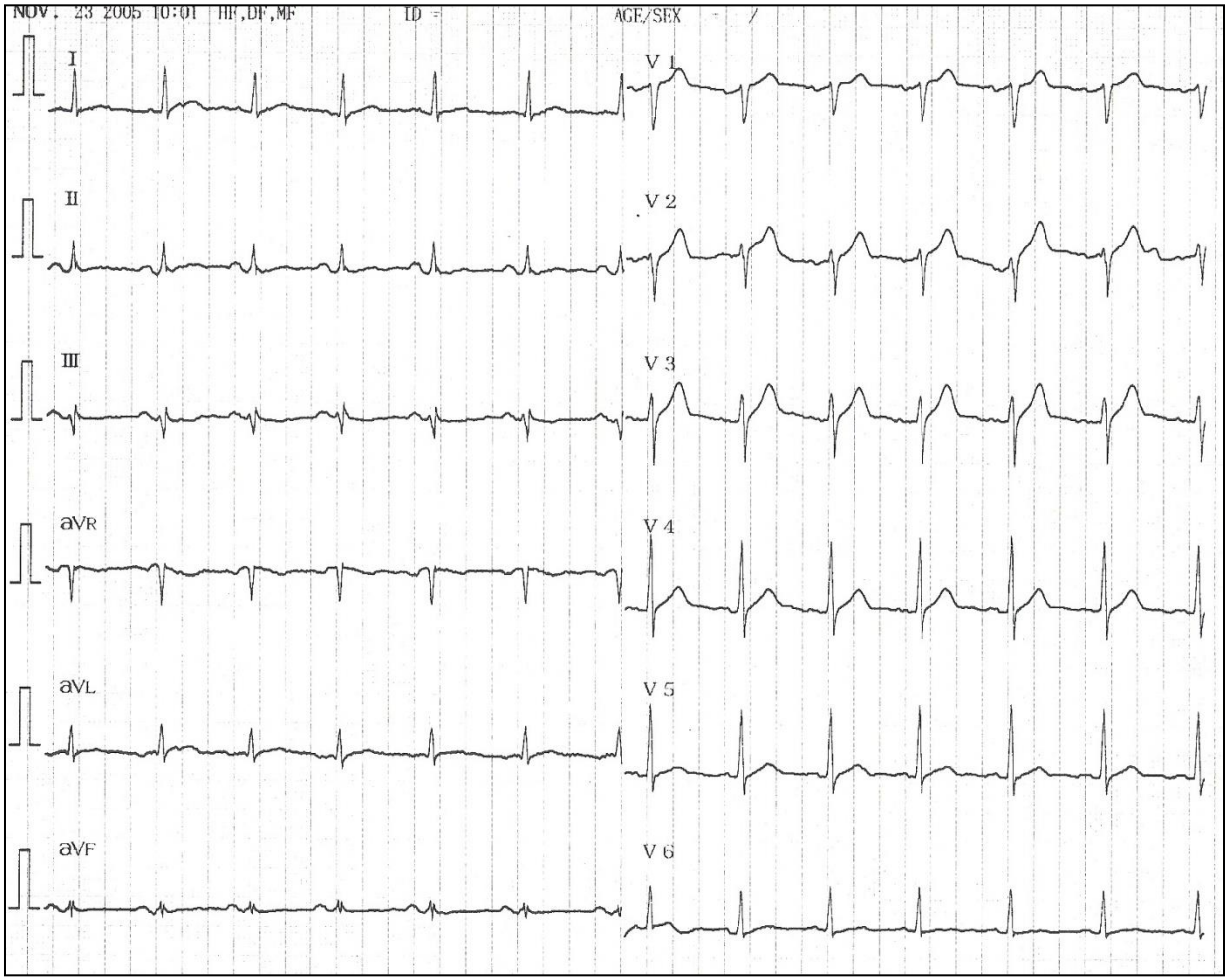
Shing Po Daily News 10/2005

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

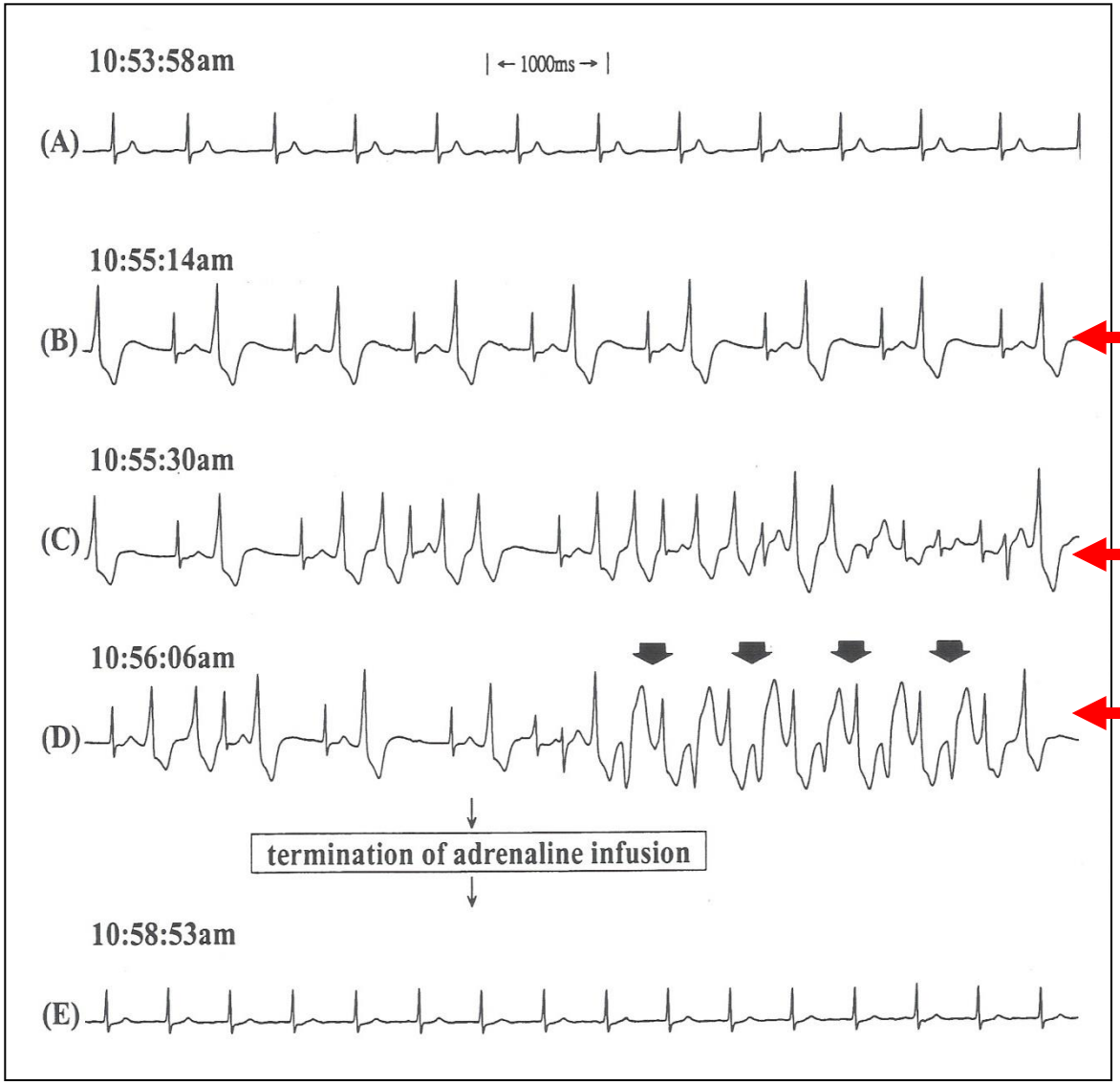


VF recorded on AED

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

Diagnosis of CPVT

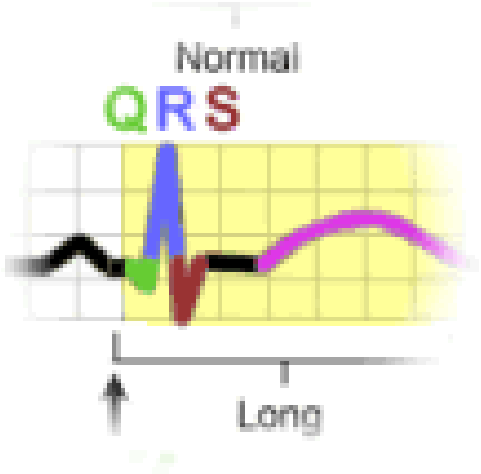
Diagnosis of catecholaminergic polymorphic ventricular tachycardia

Recommendations	Class[a]	Level[b]	Ref.[c]
CPVT is diagnosed in the presence of a structurally normal heart, normal ECG and exercise- or emotion-induced bidirectional or polymorphic VT.	I	C	14,52, 457
CPVT is diagnosed in patients who are carriers of a pathogenic mutation(s) in the genes *RyR2* or *CASQ2*.	I	C	14,52

ESC Guidelines 2015

Congenital Long QT syndrome (LQTS)

- A rare inherited cardiac arrhythmia syndrome characterized by
  - Prolonged QT intervals
  - Torsades de pointes (TdP)
  - Syncope, seizure and sudden cardiac death (SCD)
- +ve family hx in 60%
- Usually present at very young age



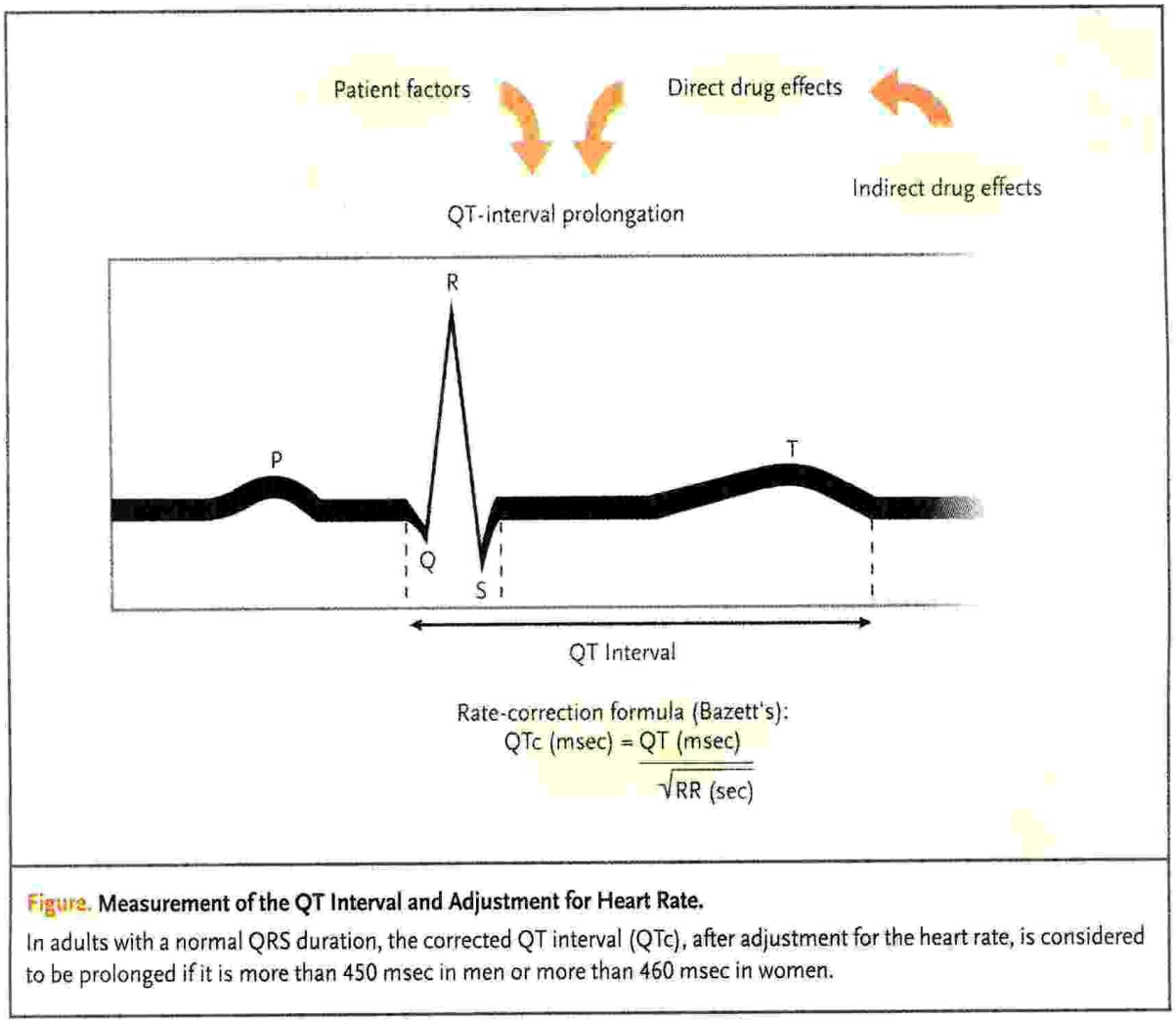
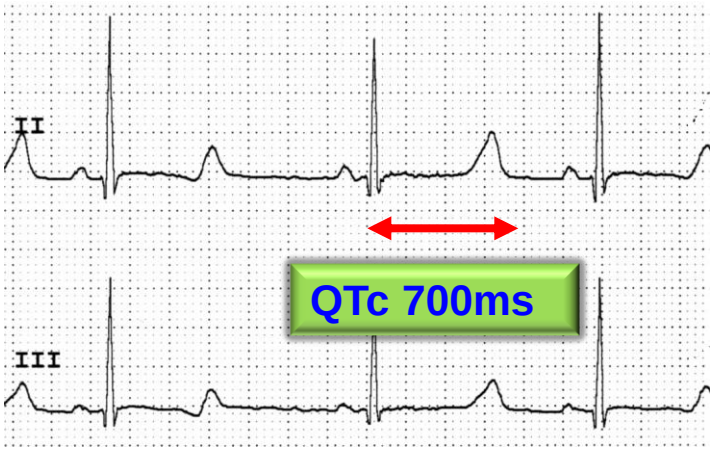


Figure. Measurement of the QT Interval and Adjustment for Heart Rate.

In adults with a normal QRS duration, the corrected QT interval (QTc), after adjustment for the heart rate, is considered to be prolonged if it is more than 450 msec in men or more than 460 msec in women.

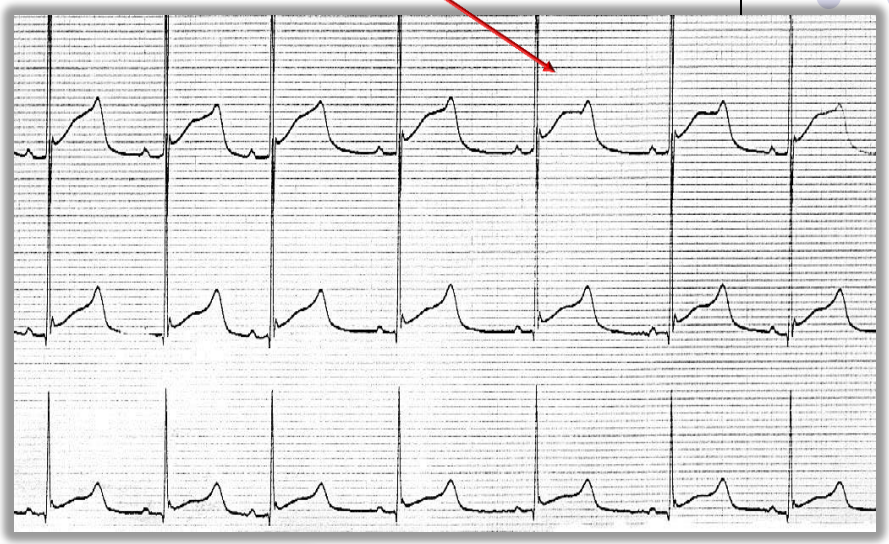
- QT interval is rate-dependent
- QTc – QT interval corrected for heart rate
- QTc prolonged if
  - ➢450ms in M
  - ➢460ms in F

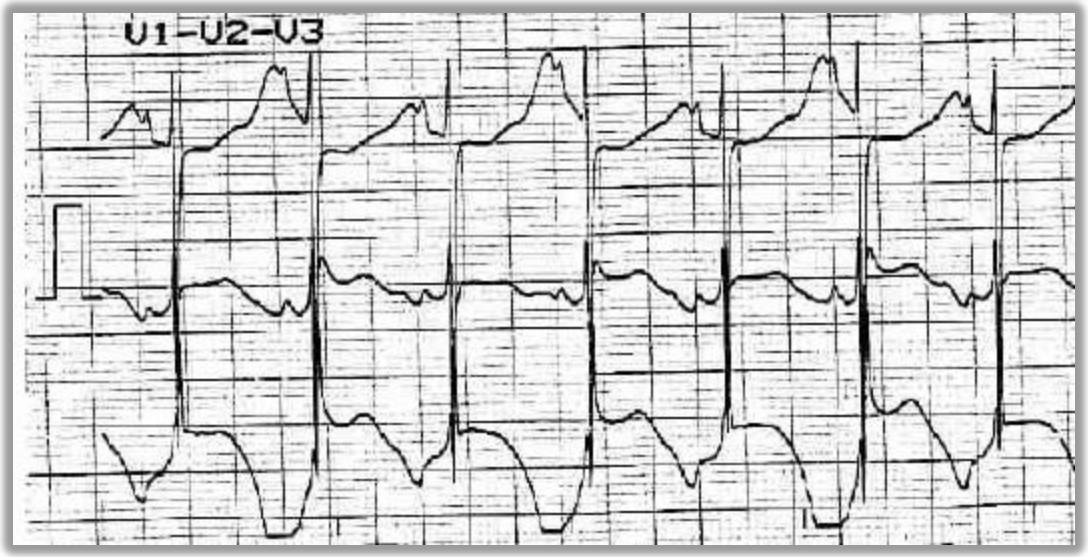
ECG abnormalities in LQTS



QTc 700ms

Notched T waves

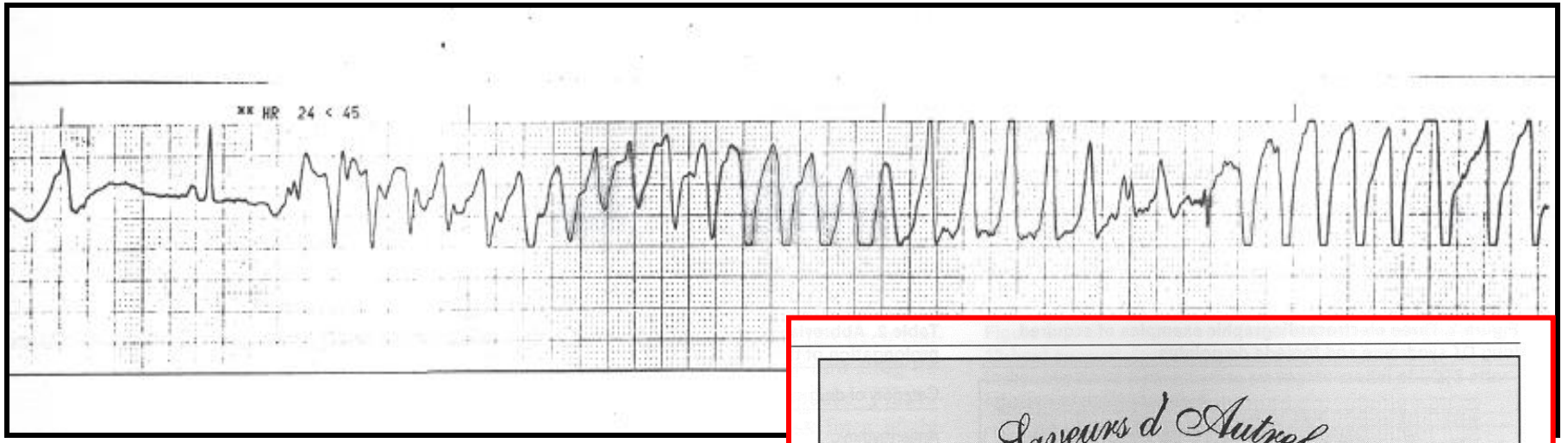




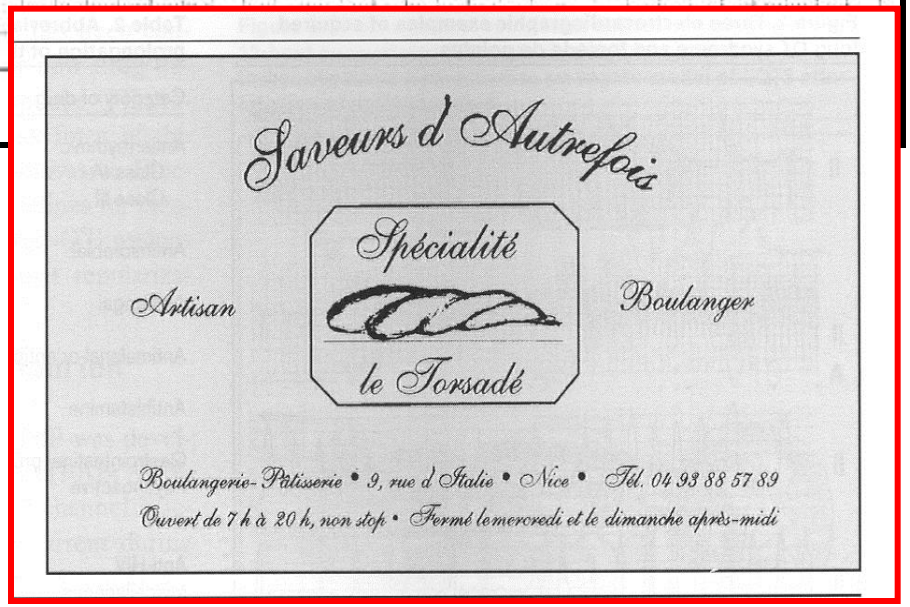
T wave alternans

“Torsade de Pointes (TdP)” – Twising about the baseline

F/13, recurrent syncope



TdP - Polymorphic VT with changing amplitude, morphology & axis



Diagnosis of Long QT Syndrome

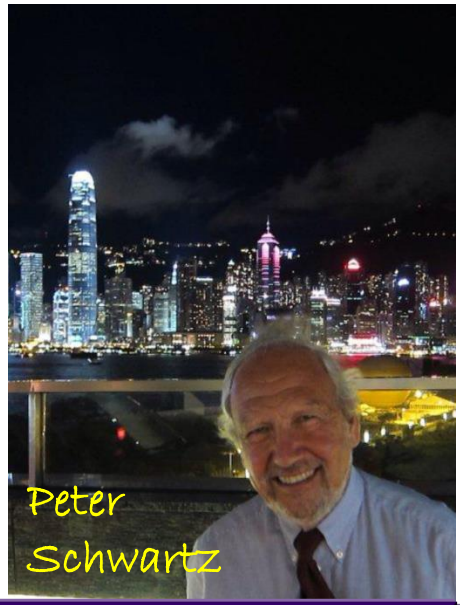
Diagnosis of Long QT Syndrome (in the absence of secondary causes for QT prolongation)

Recommendations	Class[a]	Level[b]	Ref.[c]
LQTS is diagnosed with either – QTc >480 ms in repeated 12-lead ECGs or – LQTS risk score >3.[431]	I	C	This panel of experts

- **Approximately 20-25% of patients with LQTS may have a normal range QTc**
- **Provocative tests for QT measurement may be considered in uncertain cases :**
  - **Recovery phase of Exercise Testing**
  - **During Epinephrine Infusion**

ESC Guidelines 2015

Schawartz Score



Schwartz Score

≤ 1 point – low probability

>1-3 points – intermediate probability

≥ 3 points – high probability of LQTS (revised 2006)

Table. Diagnostic criteria of QT syndrome[6]

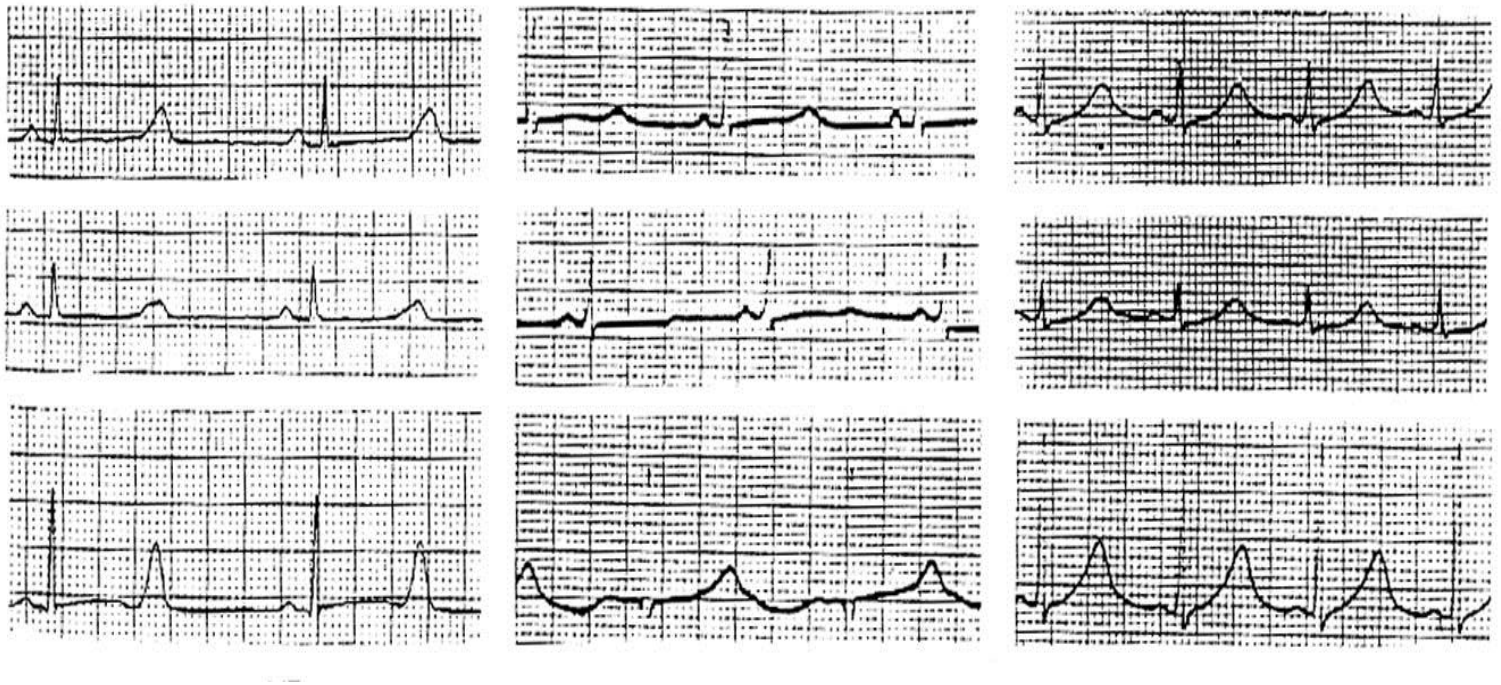
Criteria	Points
ECG* findings†	
QTc‡	
≥ 480 ms	3
460-470 ms	2
450 ms (in males)	1
Torsade de pointes	2
T-wave alternans	1
Notched T-wave in three leads	1
Low heart rate for age (<2nd percentile)	0.5
Clinical features	
Syncope with stress	2
Syncope without stress	1
Congenital deafness	0.5
Family history§	
Family history of definite long QT	1
Unexplained sudden cardiac death at age <30 years among immediate family members	0.5

* ECG electrocardiogram/electrocardiography
† In the absence of drug therapy affecting these features
‡ QTc corrected QT interval
§ Same family member cannot count twice

Schwartz PJ 1993

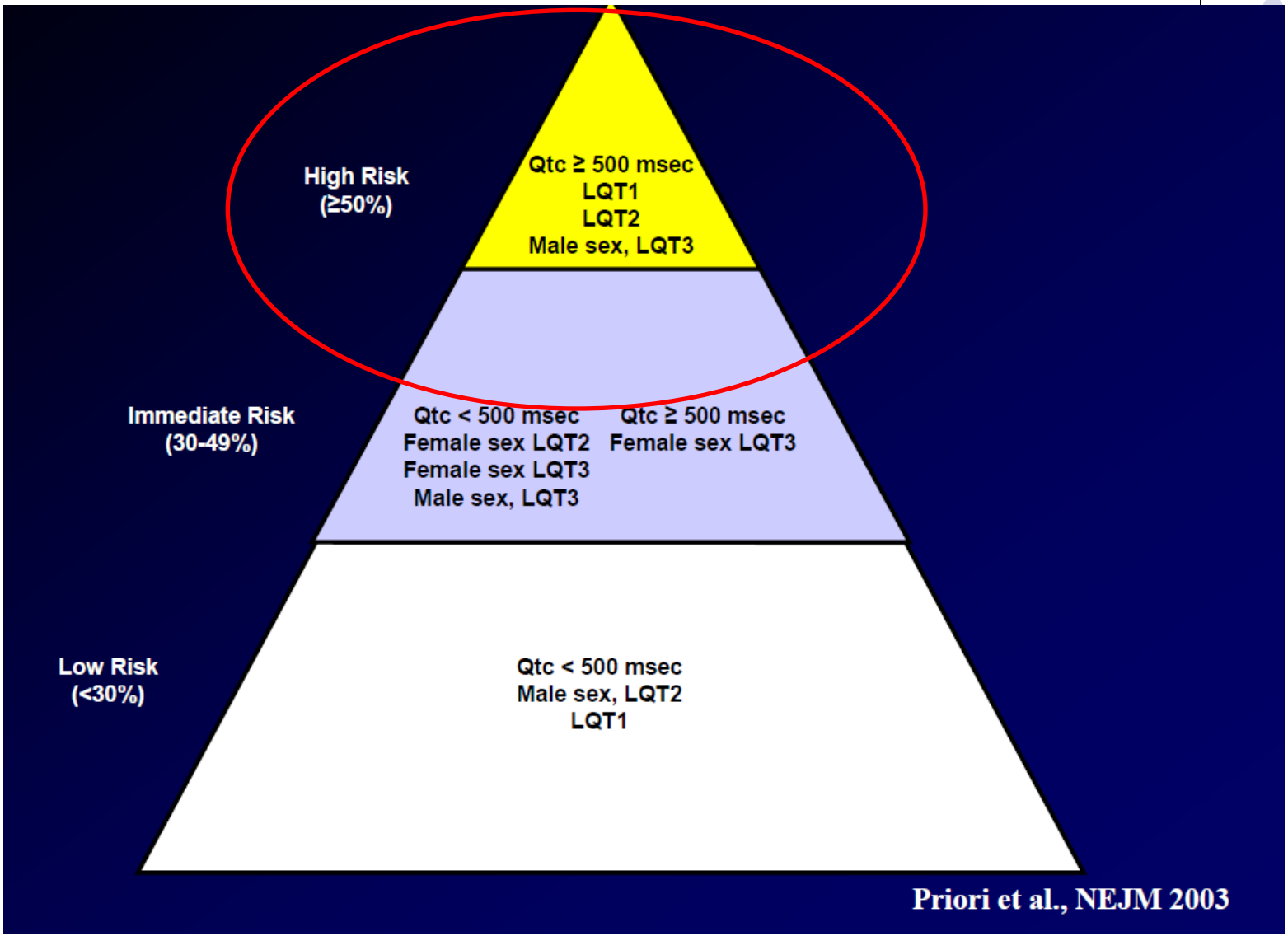
Genotype-Phenotype correlation - ECG T-wave patterns

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

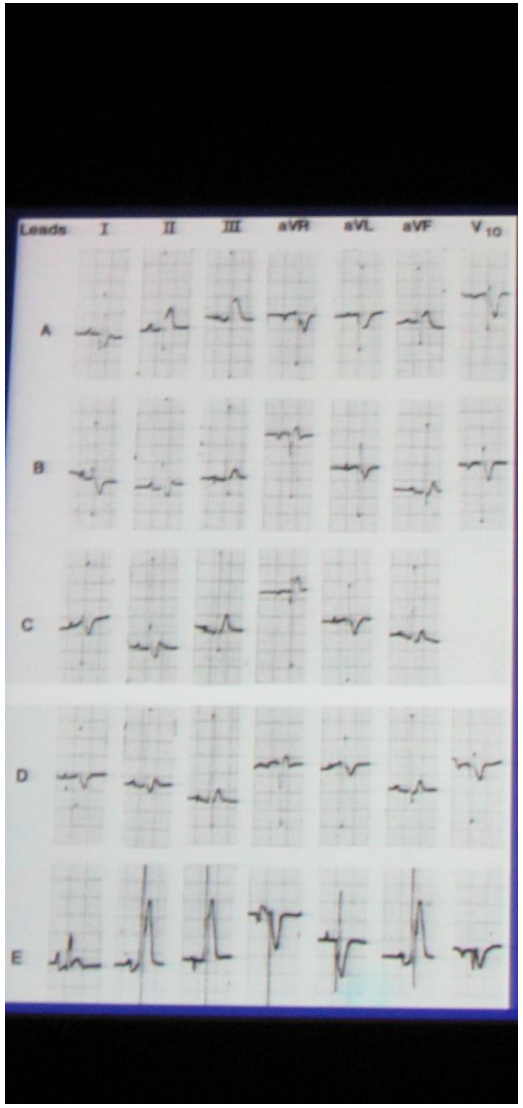


Moss AJ et al. Circulation 1995;92:2929-2934

Long QTc & LQT3 genotype – Bad prognostic indicators in LQTS

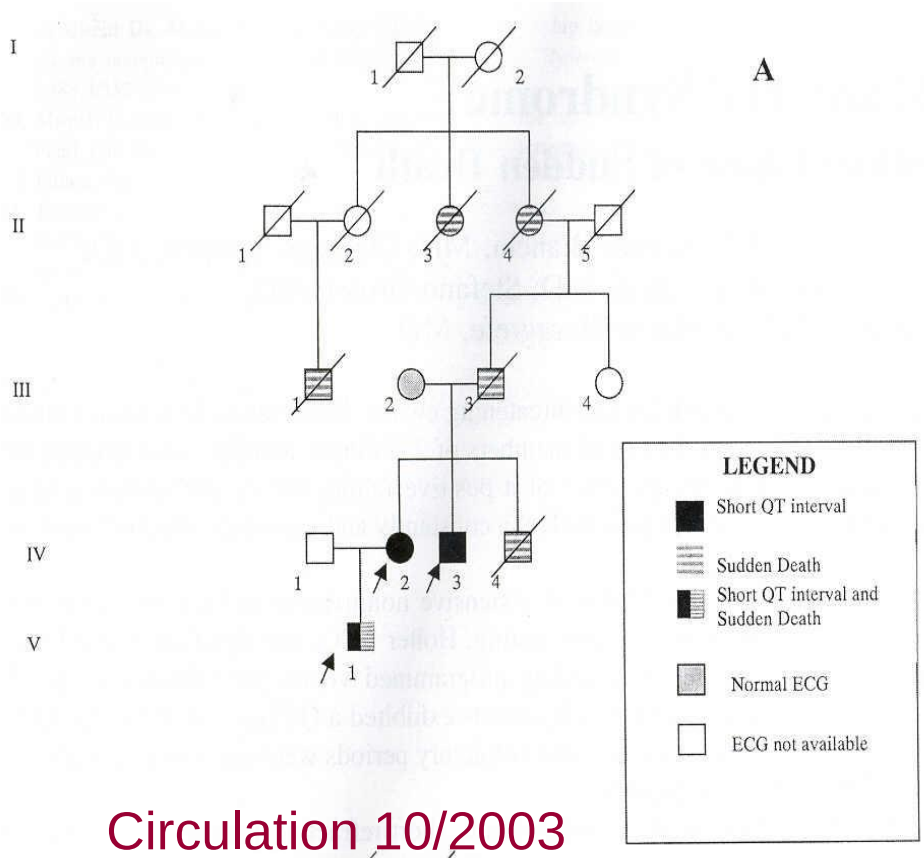


Short QT interval in Eastern Grey Kangaroos



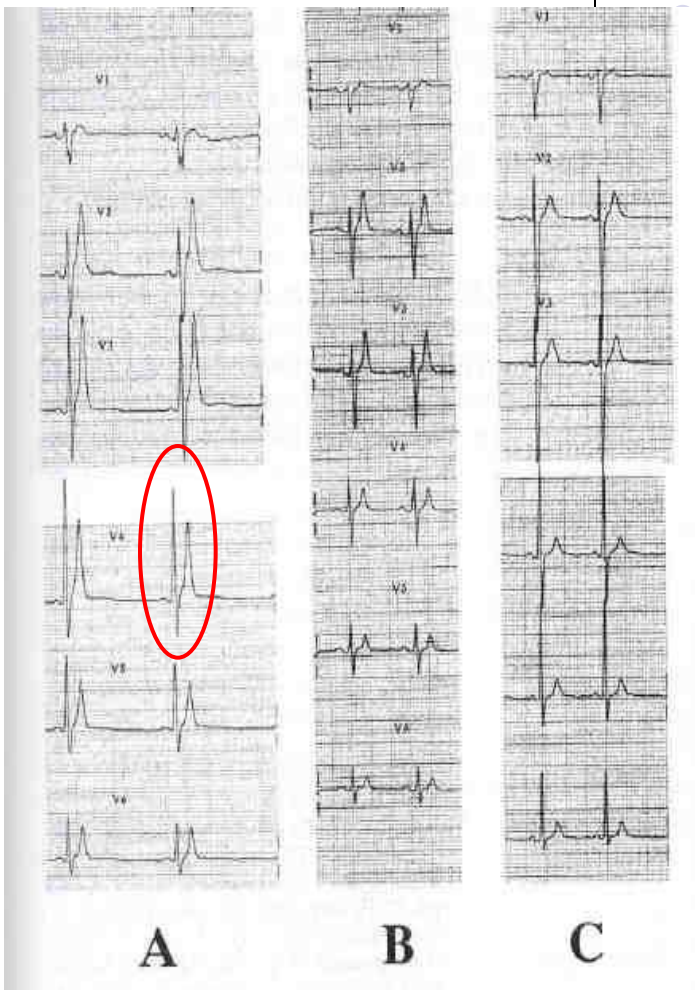
Rezakhani et al
Austr Vet J 1986

Short QT Syndrome (SQTS)



Circulation 10/2003

Family pedigree – SCD associated with Short QT interval on ECG



QTc < 300ms, QT < 280ms

Diagnosis of Short QT Syndrome

Diagnosis of Short QT Syndrome

Recommendations	Class[a]	Level[b]	Ref.[c]
SQTS is diagnosed in the presence of a QTc ≤340 ms.	I	C	This panel of experts
SQTS should be considered in the presence of a QTc ≤360 ms and one or more of the following: (a) A confirmed pathogenic mutation (b) A family history of SQTS (c) A family history of sudden death at age <40 years (d) Survival from a VT/VF episode in the absence of heart disease.	IIa	C	This panel of experts

ESC Guidelines 2015

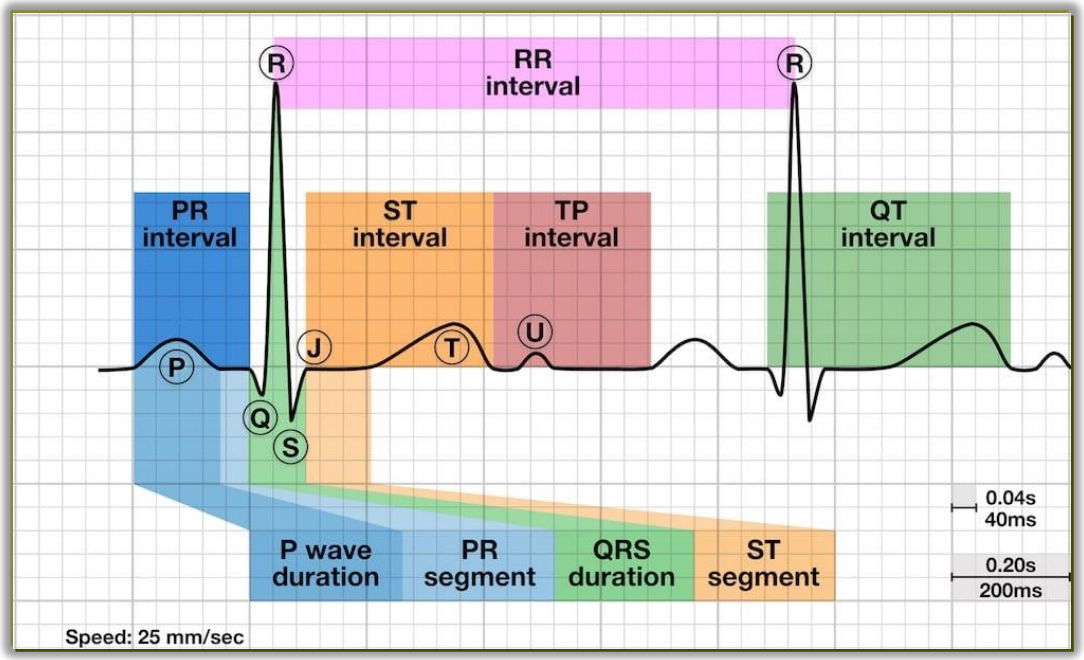
Systematic evaluation of 12-lead ECG for patients presenting with SCD

PR interval

- Short PR interval **(WPW)**
- AV Block / LBBB (**Myocarditis**)

QRS complex

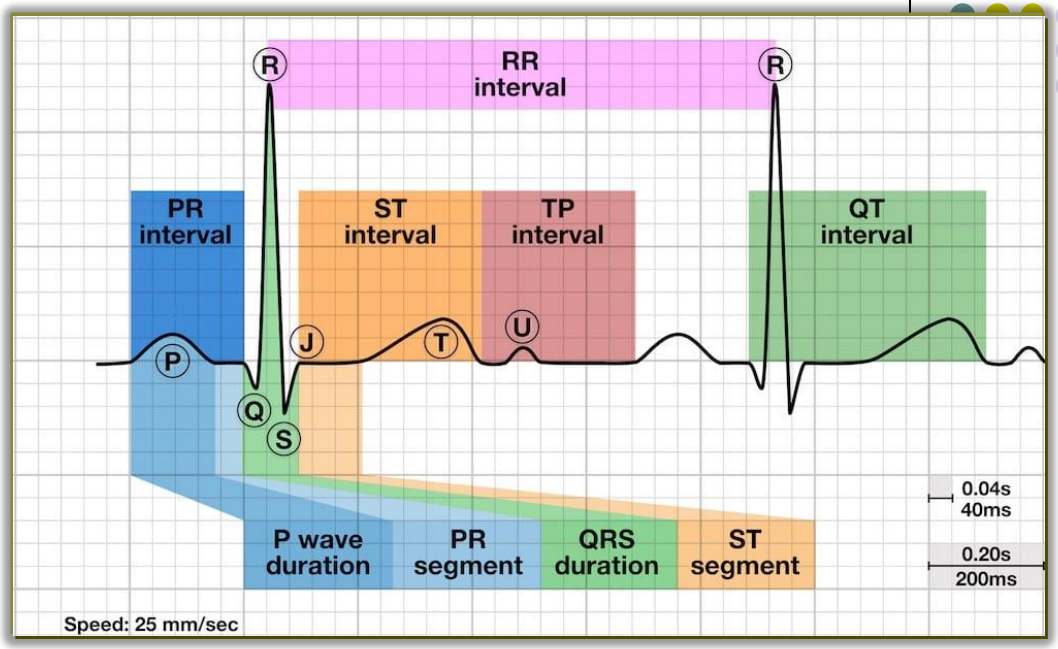
- Delta wave **(WPW)**
- LVH **(HCM)**
- Epsilon wave **(ARVC)**
- J wave **(BrS / ERS)**
- TAD (**ARVC**)



ST segment

- Wellen sign & de Winter sign **(CAD)**
- ST elevation / depression **(BrS / CAD)**

Systematic evaluation of 12-lead ECG



QT interval

- Long **(LQTS)**
- Short **(SQTS)**

T wave

- T wave inversion **(CAD, ARVC, HCM)**
- T wave alternans **(LQTS)**
- Notched T wave **(LQTS)**

 # Consider stress test / drug provocation test if indicated

Conclusions

1. SCD is a common cause of death due to cardiac arrhythmias
2. SCD may be caused by both structural heart and primary electrical diseases which may be inheritable
3. 12-lead ECG remains an important tool in *diagnosis* of arrhythmias & underlying heart disease and *guiding Ix & treatment* for secondry prevention of SCD
4. 12-lead ECG during sinus rhythm should be systematically evaluated in SCD survivors
5. Stress test and/or drug provocation tests may unmask diagnostic ECG features of underlying heart disease to improve diagnostic accuracy

Sherlock Holmes

Thank You !