ECG SYNDROMES

“ SIGNS “

Romel Wrenn MD, FACC

21years
Male Caucasian

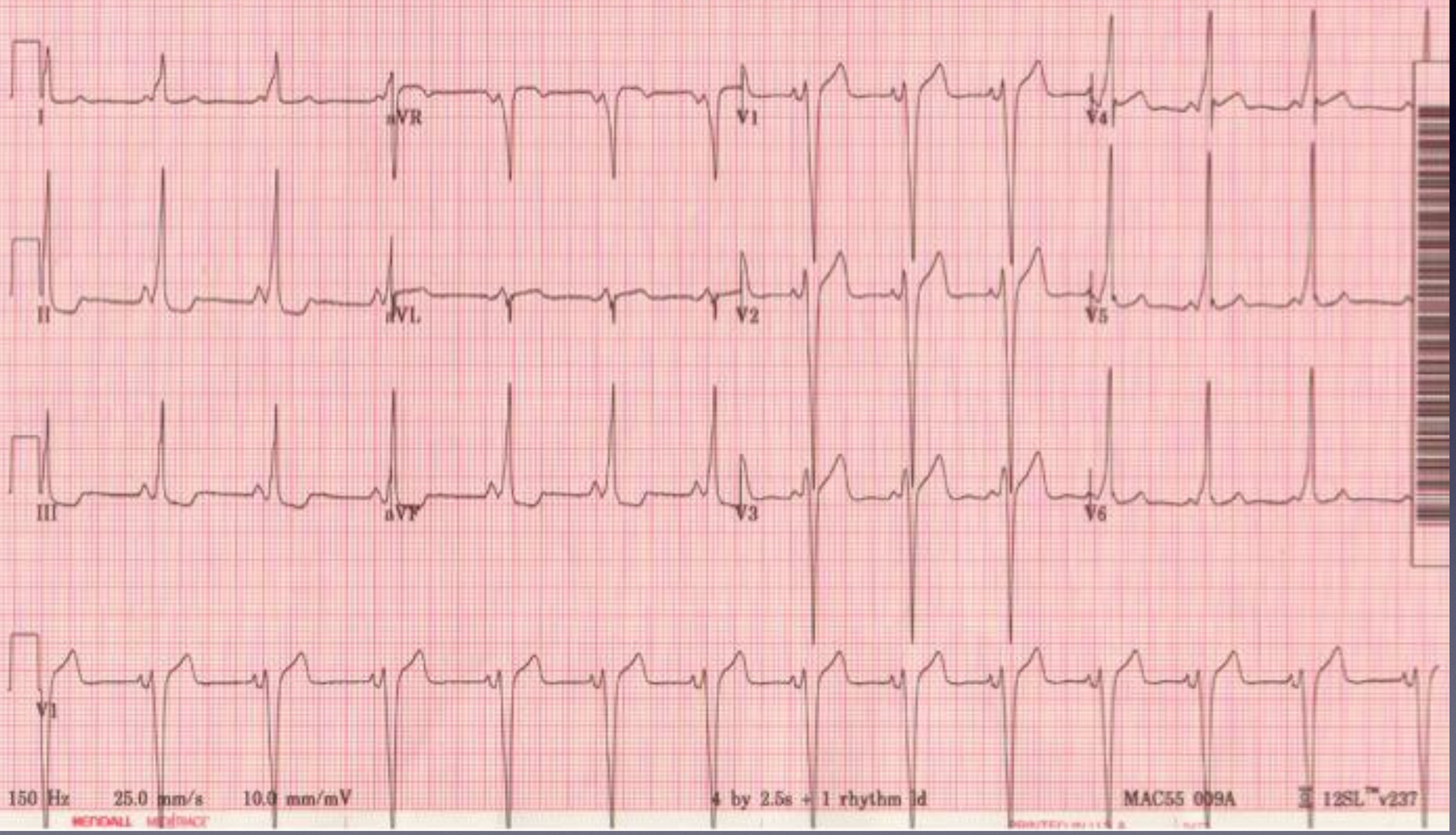
Loc: 1

Technician: 5

Vent. rate	80 bpm
PR interval	128 ms
QRS duration	146 ms
QT/QTc	390/449 ms
P-R-T axes	82 68 -89

Referred by: ELLIS, CHRIS

Unconfirmed



150 Hz 25.0 mm/s 10.0 mm/mV 4 by 2.5s + 1 rhythm ld MAC55 009A 12SL™v237

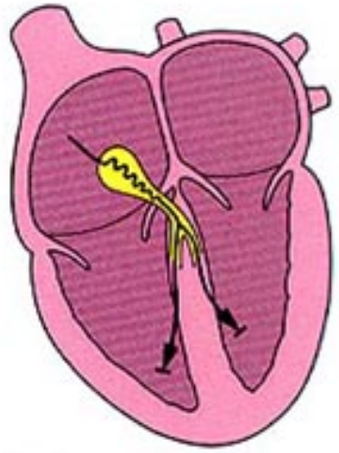
MEDWALL MEDITRACE

Wolff-White-Parkinson

- Wide QRS
  - not due to a delay in depolarization
  - due to an early depolarization
- Shortened PR interval
- Upstroke QRS complex is slurred; *delta* wave

Atrioventricular Bypass Tract

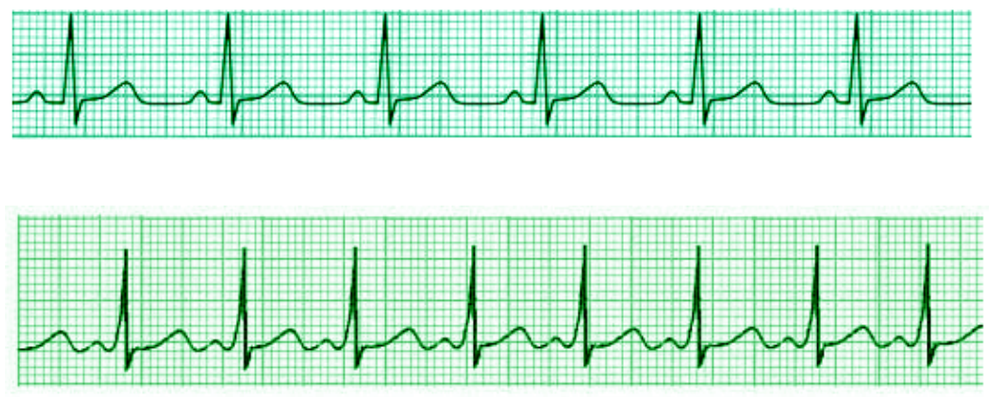
- Accessory pathways form and fail to disappear during fetal development
- Formed near the mitral or tricuspid valves or interventricular septum
- An AV bypass tract is sometimes referred to as the bundle of Kent

	Action	ECG	Comments
	Concealed AV connection (no antegrade conduction)		Ventricles activated by normal H-P system (QRS narrow)

Wolff-Parkinson-White Syndrome

Normally, the AV node is the only conduction pathway for impulses from the atria to the ventricles. Wolff-Parkinson-White syndrome is characterized by the presence of an accessory atrioventicular pathway located between the wall of the right or left atria and the ventricles, known as the Bundle of Kent. This pathway allows the impulse to bypass the AV node and activate the ventricles prematurely. Consequently, an initial slur to the QRS complex, known as a delta wave may be observed. The QRS complexes are wide, more than 0.11 sec, indicating that the impulse did not travel through the normal conducting system. The PR is shortened, to less than 0.12 sec, because the delay at the AV node is bypassed.

The EKG on the top shows normal sinus rhythm. The EKG at the bottom showsWolff-Parkinson-White Syndrome



The accessory pathway can cause a reentry circuit to be established. Reentry is initiated by a premature atrial or ventricular beat coupled with a unidirectional block in one of the pathways (because the normal impulse gets to pathway when it is refractory after the premature beat). The result is a continuous impulse conduction. Reentry causes two kinds of tachycardia.

1. Orthodromic AV reentrant tachycardia which occurs when the impulse is conducted through the AV node with retrograde return to the atria via the Bundle of Kent. The heart rate is usually 140-250 BPM. The QRS complexes are narrow and delta waves are not observed.
2. Antidromic AV reentrant tachycardia which occurs when the impulse is conducted through the Bundle of Kent with retrograde return to the atria via the AV node. The QRS complexes are wide.

Wolff-Parkinson-White syndrome is commonly associated with congenital heart abnormalities like Tetrology of Fallot, coarctation of the aorta, tricuspid atresia and transposition of the great vessels. In severe cases, treatment would involve surgical removal or ablation of one of the pathways.

WPW

In 1930, Louis Wolff, Sir John Parkinson, and Paul Dudley White published article describing 11 patients

Attacks of tachycardia associated with a sinus rhythm electrocardiographic (ECG) pattern of bundle branch block with a short PR interval

Subsequently termed the Wolff-Parkinson-White (WPW) syndrome

Earlier isolated case reports describing similar patients had been published.

In 1943, the ECG features of preexcitation were correlated with anatomic evidence for the existence of anomalous bundles of conducting tissue that bypassed all or part of the normal atrioventricular (AV) conduction system

Genetics of WPW

In most cases, the cause of Wolff-Parkinson-White syndrome is unknown.

A small percentage of all cases are caused by mutations in the *PRKAG2* gene. **PRKAG2 (protein kinase AMP-activated non-catalytic subunit gamma 2)**

Some people with these mutations also have features of hypertrophic cardiomyopathy

The *PRKAG2* gene provides instructions for making a protein that is part of an enzyme called AMP-activated protein kinase (AMPK).

This enzyme helps sense and respond to energy demands within cells. It is likely involved in the development of the heart before birth, although its role in this process is unclear.

Genetics of WPW

Ann Pediatr Cardiol. 2015 May-Aug; 8(2): 153–156.
doi: 10.4103/0974-2069.154149

PMCID: PMC4453188
PMID:26085771

PRKAG2 mutation: An easily missed cardiac specific non-lysosomal glycogenosis

Varun Aggarwal, Nancy Dobrolet,[1] Steven Fishberger,[1] Jenny Zablah, Parul Jayakar,[2] and Zineb Ammous[2]

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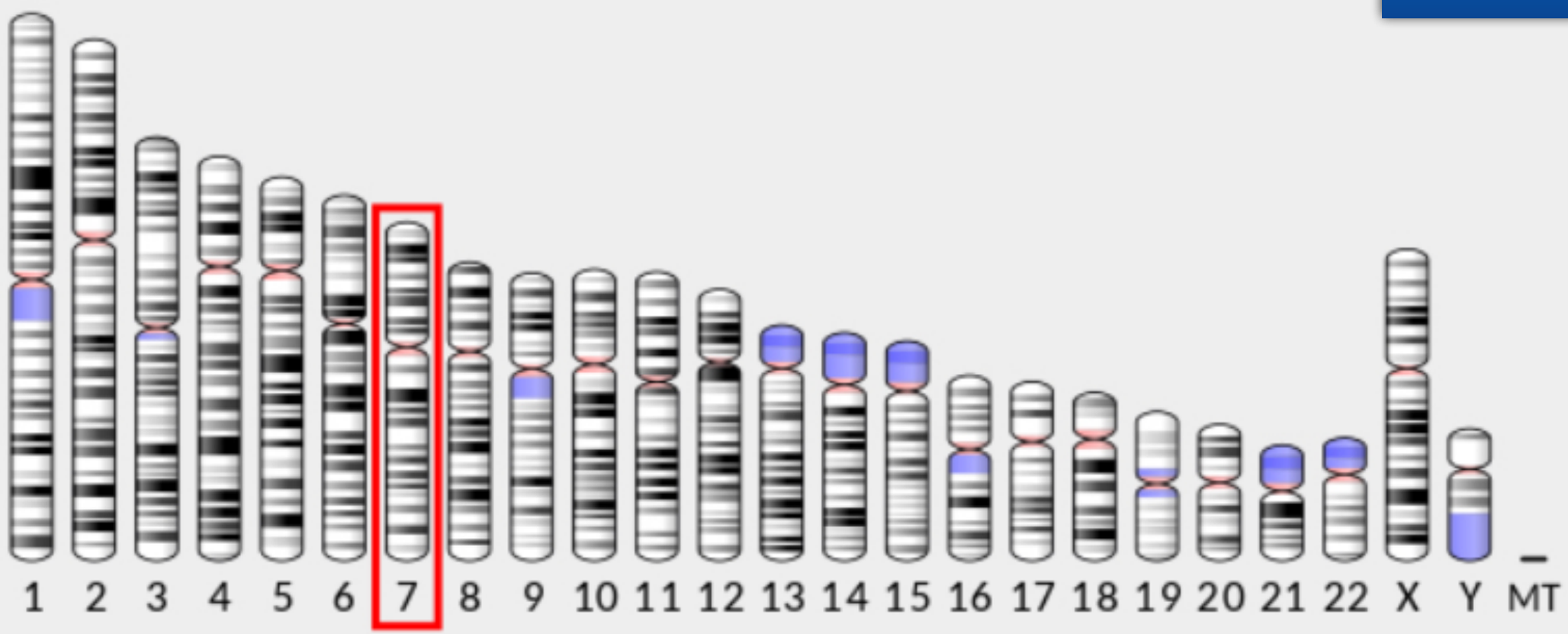
Abstract

Go to:

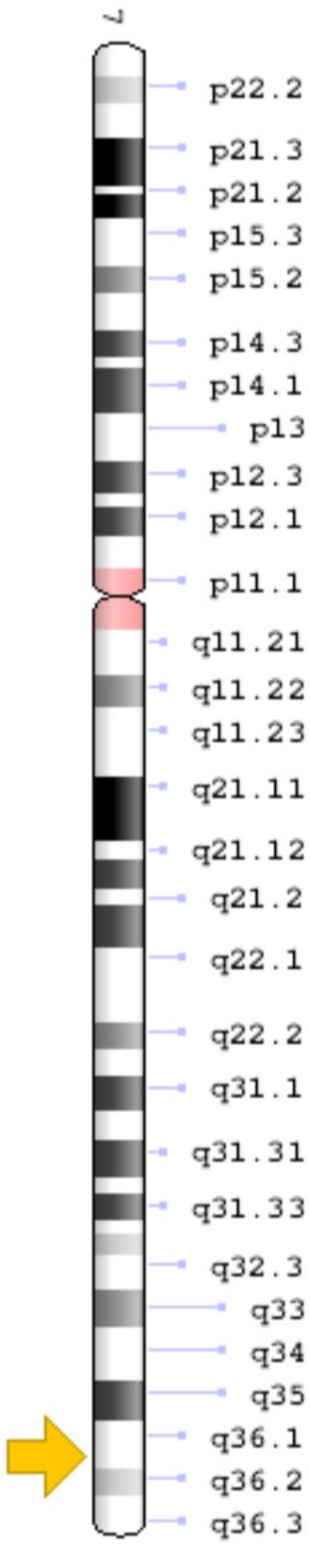
Mutations in PRKAG2 gene that regulates the γ2 subunit of the adenosine monophosphate (AMP) dependent protein kinase have been associated with the development of atrioventricular (AV) accessory pathways, cardiac hypertrophy, and conduction system abnormalities. These patients can potentially be misdiagnosed as hypertrophic cardiomyopathy (HOCM) and/or Wolf-Parkinson White (WPW) syndrome due to similar clinical phenotype. Early recognition of this disease entity is very important as ablation of suspected accessory pathways is not effective and the natural history of the disease is very different from HOCM and WPW syndrome.

Gene location (Human)



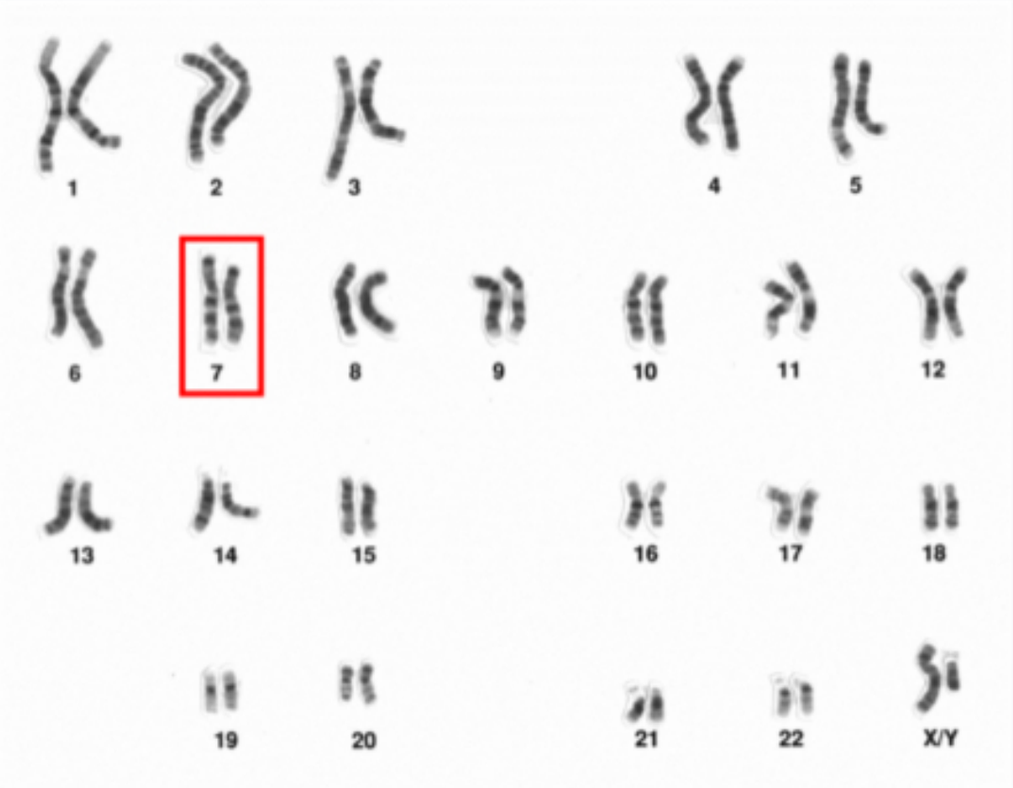
Cytogenetic Location: 7q36.1, which is the long (q) arm of chromosome 7 at position 36.1

Molecular Location: base pairs 151,556,114 to 151,877,231 on chromosome 7 (Homo sapiens Annotation Release 108, GRCh38.p7) (NCBI)



Chromosome 7

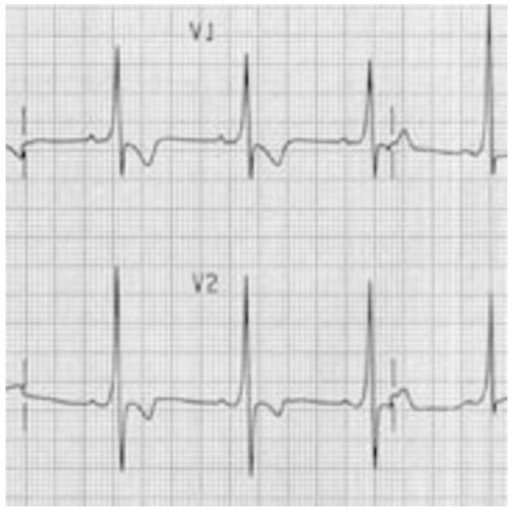
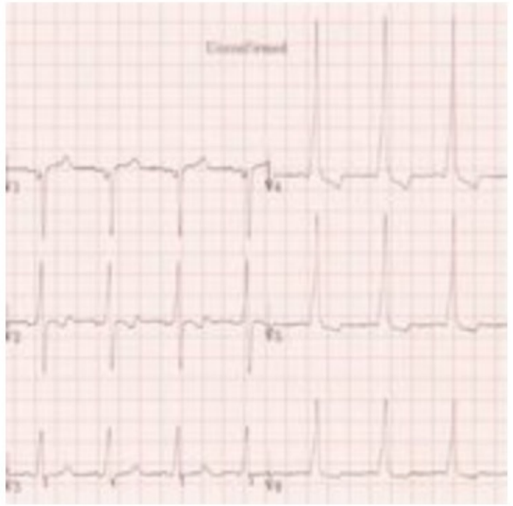
Human chromosome 7 pair after G-banding.
One is from mother, one is from father.

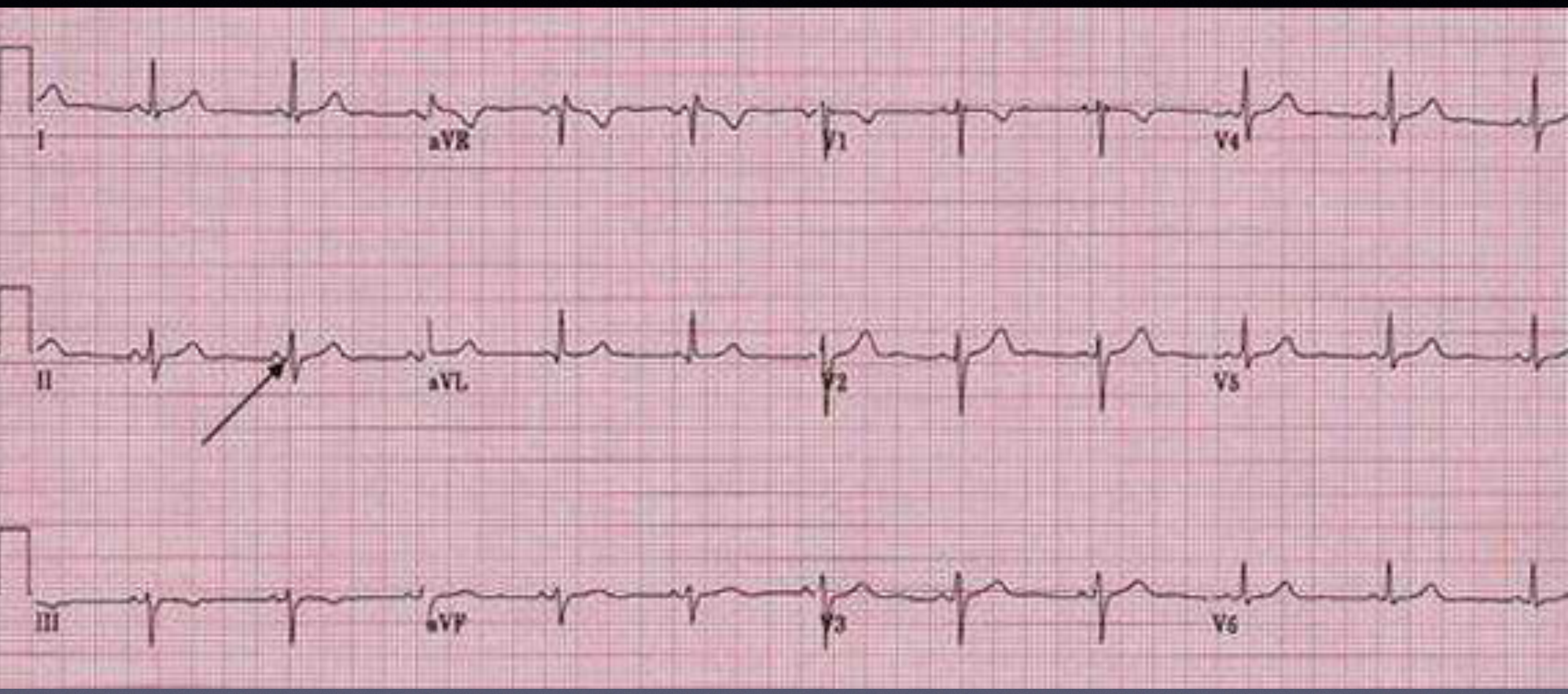


Chromosome 7 pair
in human male karyogram.

Note the mtDNA row - this represents the small amount of DNA found in the mitochondria.

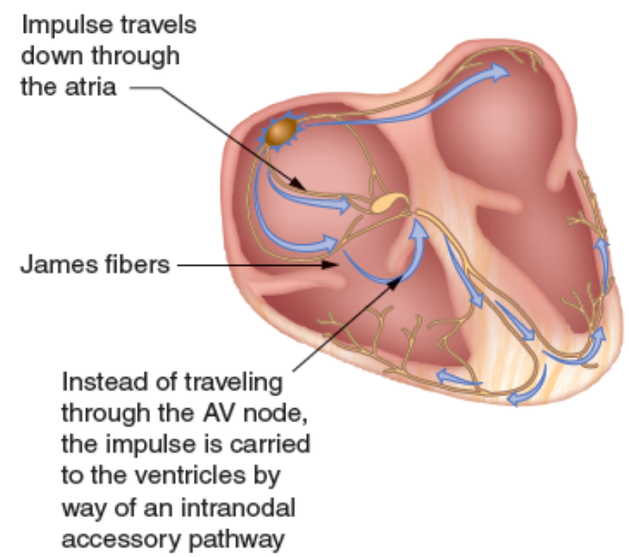
Chromosome	Length (mm)	Base pairs
1	85	249,250,621
2	83	243,199,373
3	67	198,022,430
4	65	191,154,276
5	62	180,915,260
6	58	171,115,067
7	54	159,138,663
8	50	146,364,022
9	48	141,213,431
10	46	135,534,747
11	46	135,006,516
12	45	133,851,895
13	39	115,169,878
14	36	107,349,540
15	35	102,531,392
16	31	90,354,753
17	28	81,195,210
18	27	78,077,248
19	20	59,128,983
20	21	63,025,520
21	16	48,129,895
22	17	51,304,566
X	53	155,270,560
Y	20	59,373,566
mtDNA	0.0054	16,569

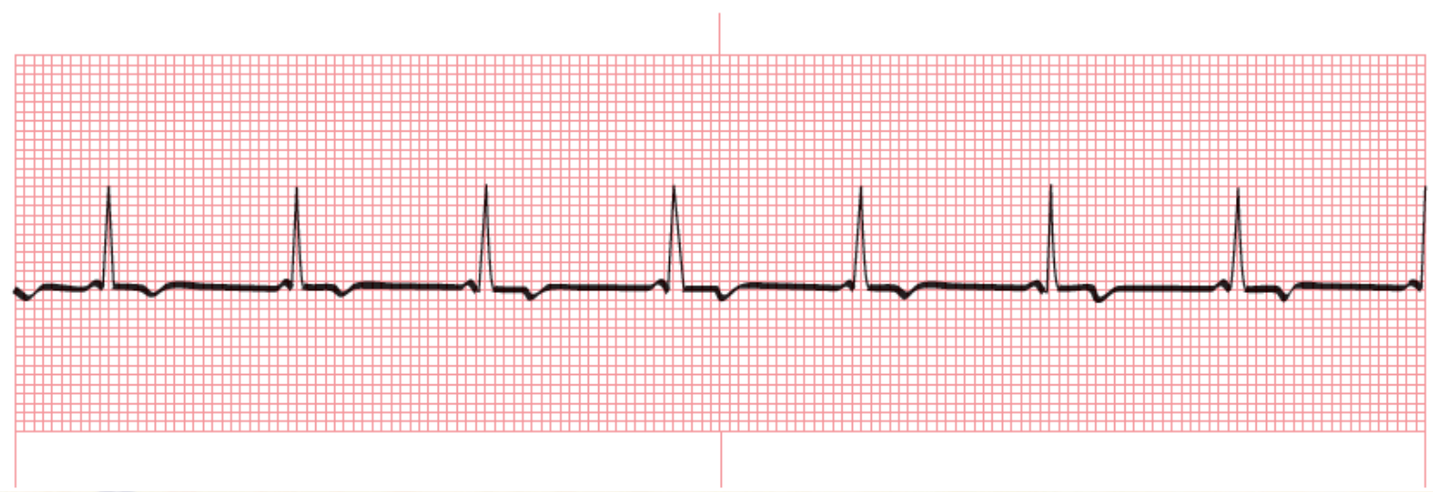
Syndrome	Pathology	ECG	Clinical Significance	Images / References
Wolff-Parkinson White	Pre-excitation syndrome.Re-entrant bundle of AV tissue distant to AV node.	Short PR interval; Prolonged QRS and slurred upstroke of QRS complex (Delta wave).“Type A” shows positive QRS in V1 (LV accessory). Upright positive delta wave in all precordial leads with a resultant R greater than S amplitude in lead V1“Type B” shows negative QRS in V1(RV accessory). Predominantly negative delta wave and QRS complex in leads V1 and V2 and becomes positive in transition to the lateral leads resembling LBBB.	Risk of Atrial arrhythmias being transmitted abherrantly causing VF or VT.Requires ablation of pathwayClinical Case WPW 001	 


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

Preexcitation

- Another type of preexcitation is Lown-Ganong-Levine (LGL) Syndrome
- In LGL syndrome, the accessory pathway, referred to as the James fibers, is within the AV node
- This accessory pathway bypasses the normal delay within the AV node but ventricular conduction occurs through the usual ventricular conduction pathways





LGL

In 1938, Clerc, Levy, and Critesco first described the occurrence of frequent paroxysms of tachycardia in patients with a short PR interval and normal QRS duration

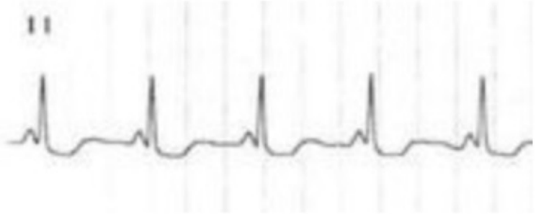
This syndrome was again described in 1952 by Lown, Ganong

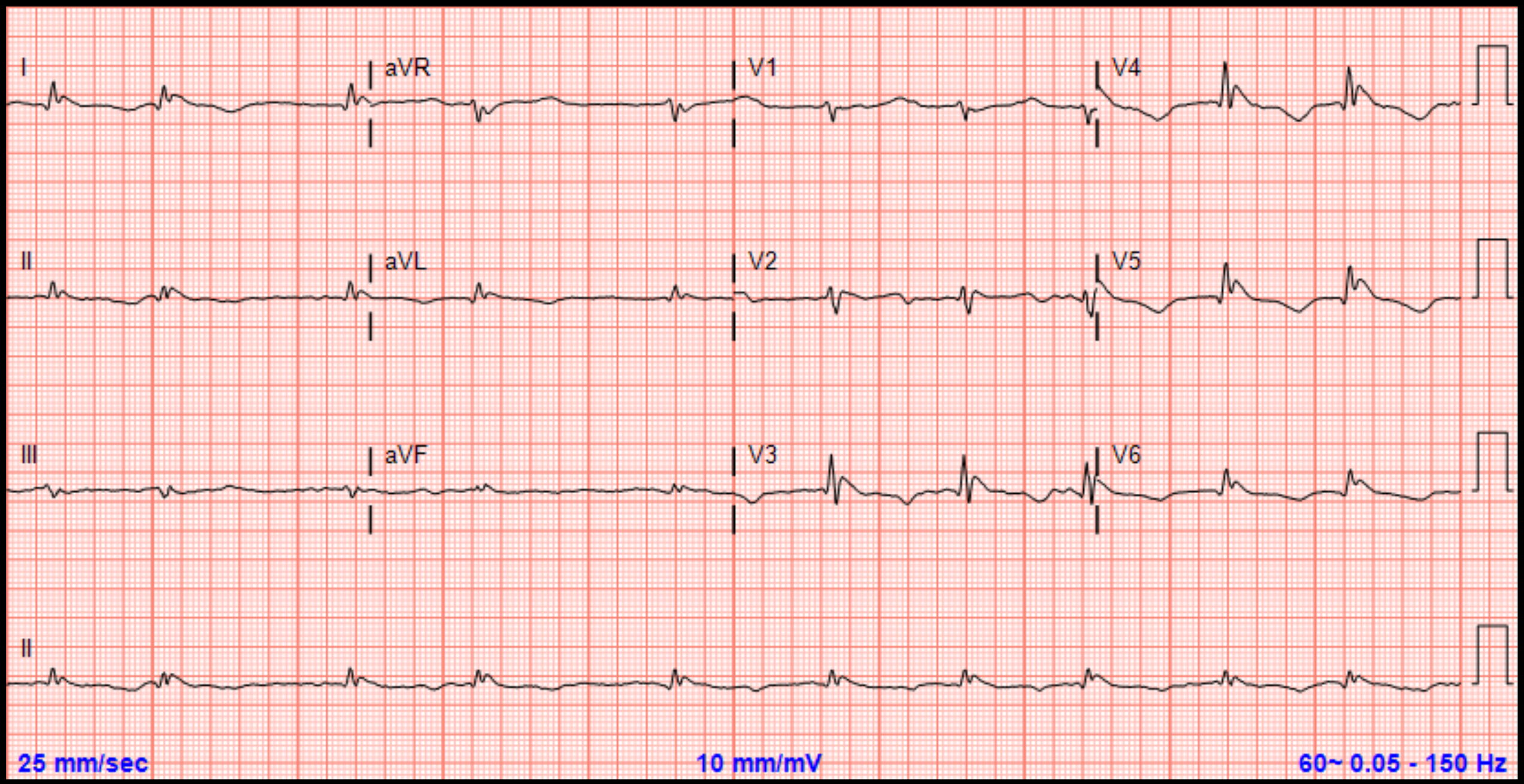
In 1946, Burch and Kimball proposed that an atrio-Hisian (AH) pathway might explain the findings of the syndrome, although no such pathway had yet been identified anatomically. [4]

In 1961, James described fibers that originate in the low atrium and terminate low in the AV node. [5]

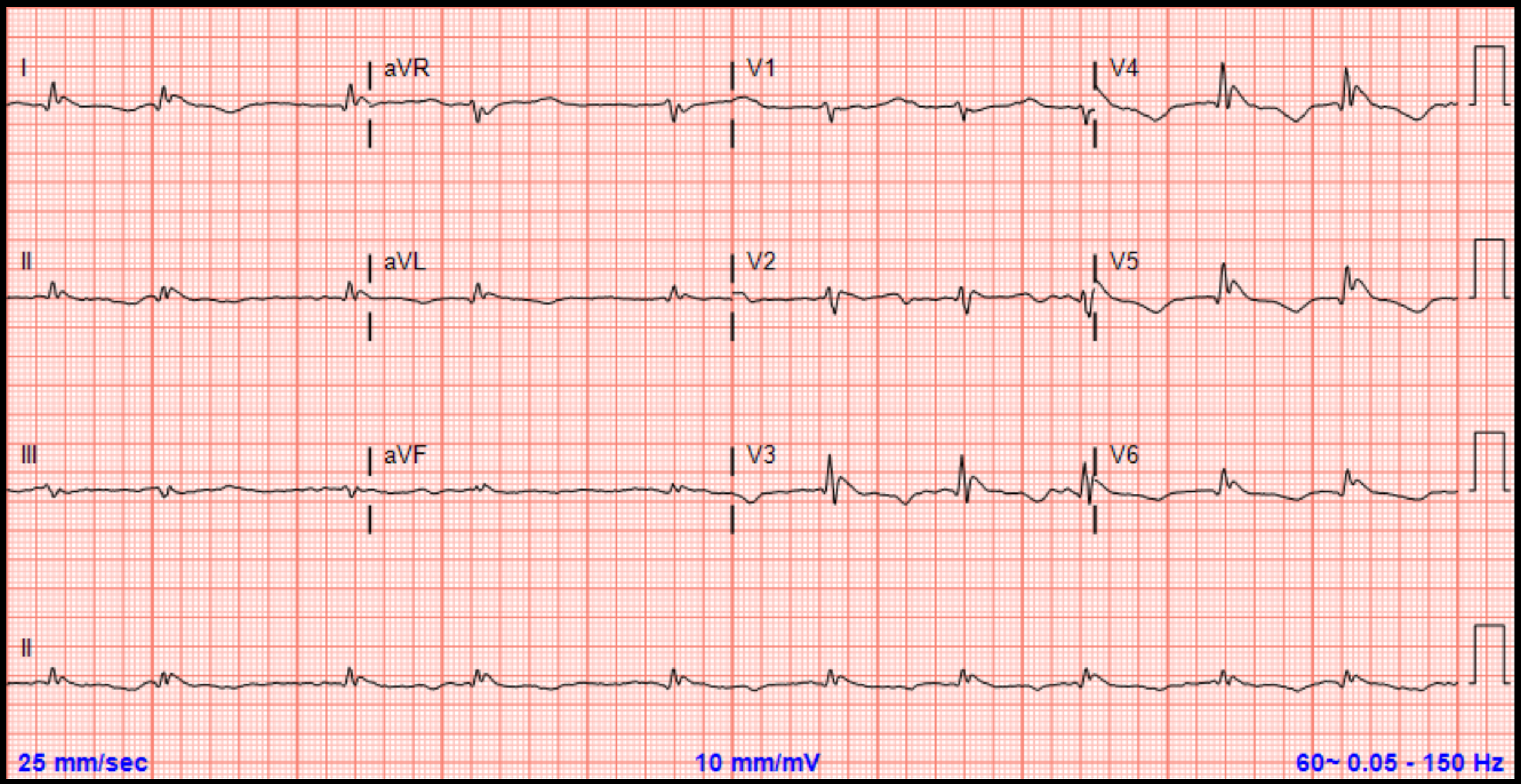
Brechenmacher et al reported anatomic findings of an AH bundle in 1974. [6]

Subsequent investigations into the origin of LGL have largely involved invasive electrophysiologic studies that have sought to identify structural and functional anomalies that might explain the findings of LGL. [7, 8]

Syndrome	Pathology	ECG	Clinical Significance	Images / References
Lown-Ganong-Levine	Pre- excitation syndromeRe-entrant bundle of AV tissue close to AV node .	Accessory pathway is down James fibres.No Delta wave as conduction normal down Bundle of His	Risk of AF being transmitted abherrantly causing VF or VT.Requires ablation of pathway as WPW	


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
25 mm/sec
10 mm/mV
60~ 0.05 - 150 Hz

77 Degrees F
25 Degrees C



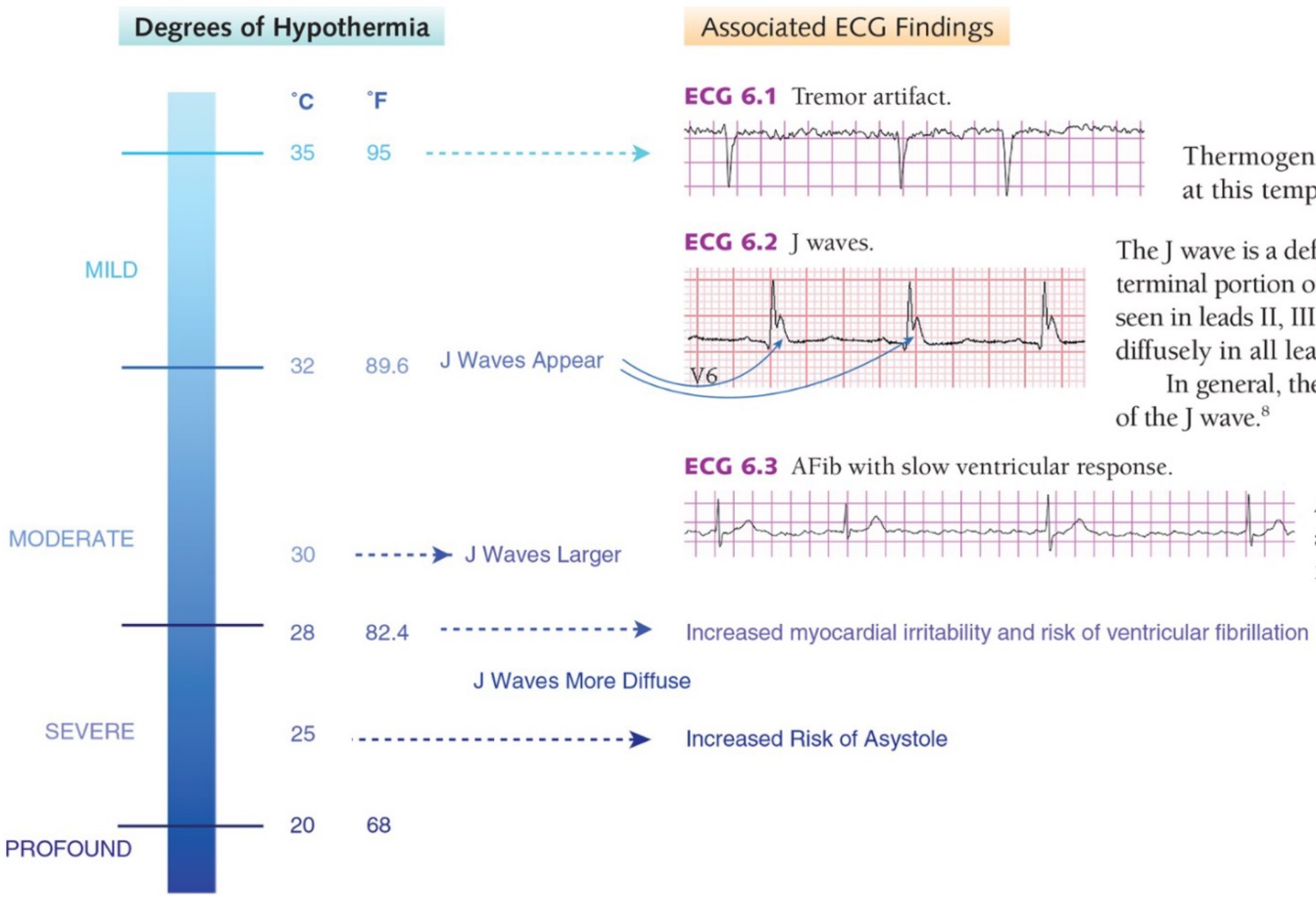
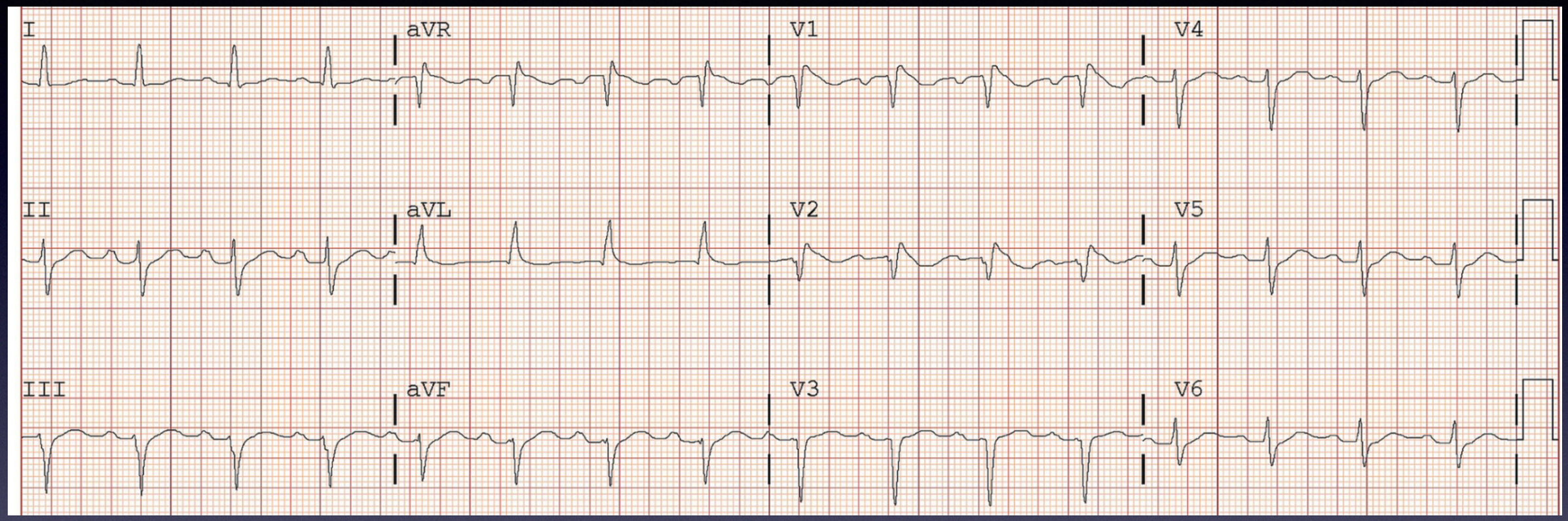
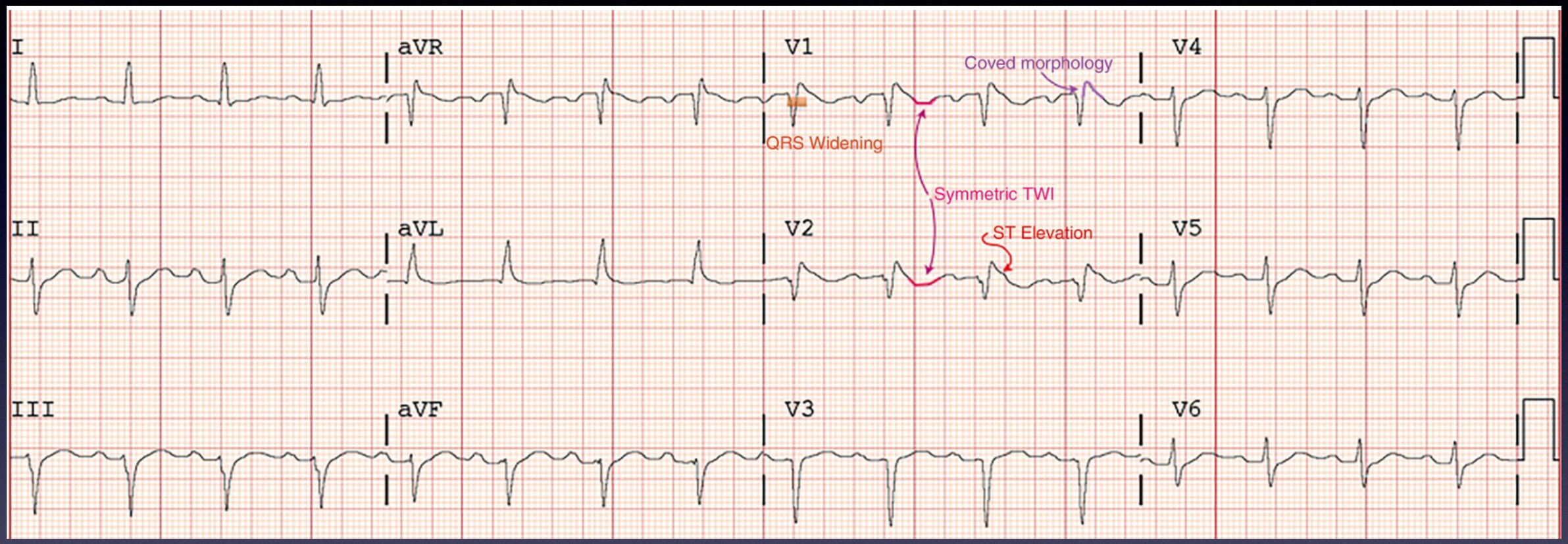


FIGURE 6.5 Scale depicting different degrees of hypothermia.

Conditions with predominant J-waves
Hypothermia
Hypercalcaemia
Hyperkalaemia
Vasospastic angina
Brugada syndrome
Early repolarization syndrome
Short QT syndrome
Hypoxia
Acidosis
Pulmonary embolism
Arrhythmogenic right ventricular cardiomyopathy
Subarachnoid haemorrhage


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


I
aVR
V1
V4
Coved morphology
QRS Widening
Symmetric TWI
II
aVL
V2
ST Elevation
V5
III
aVF
V3
V6

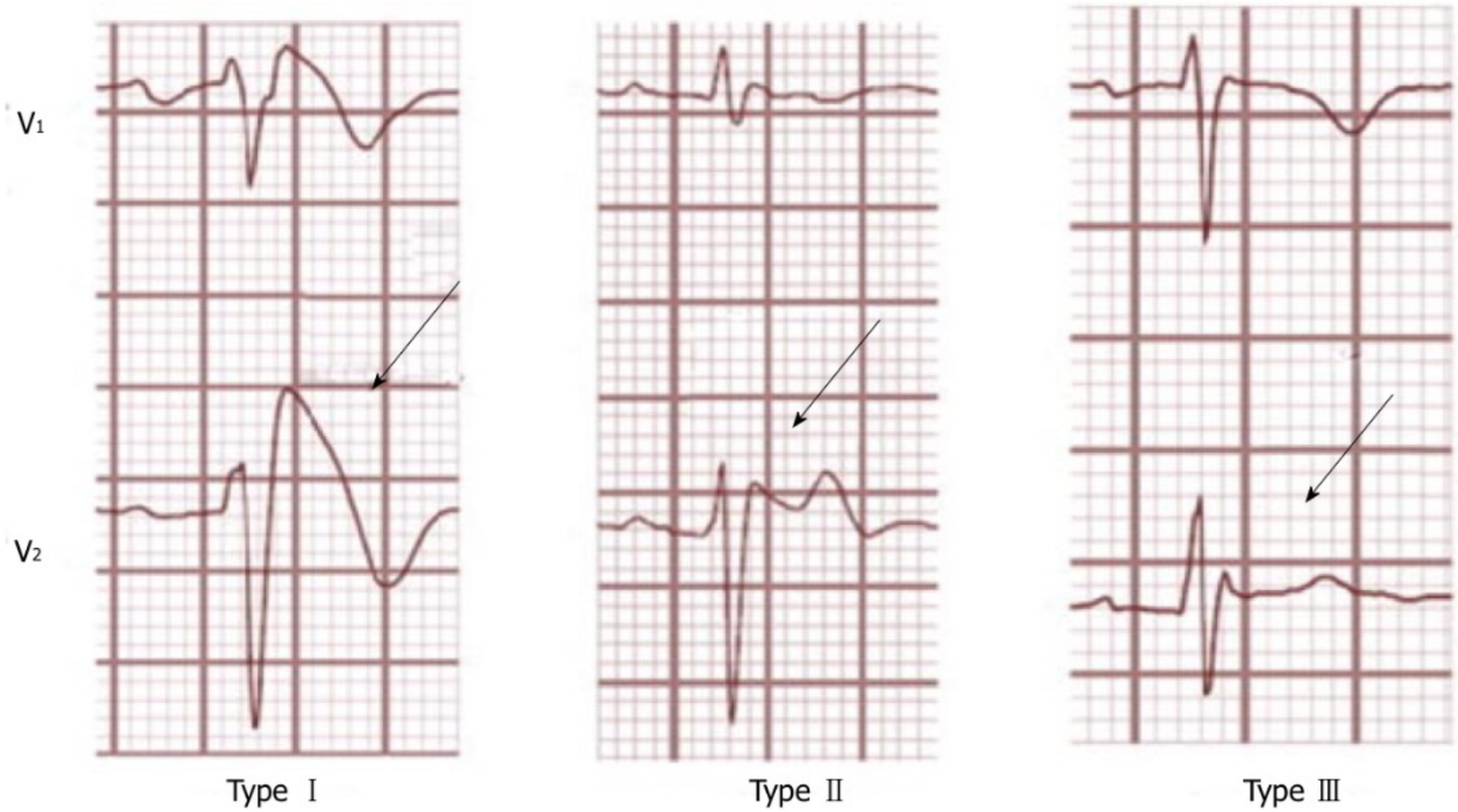
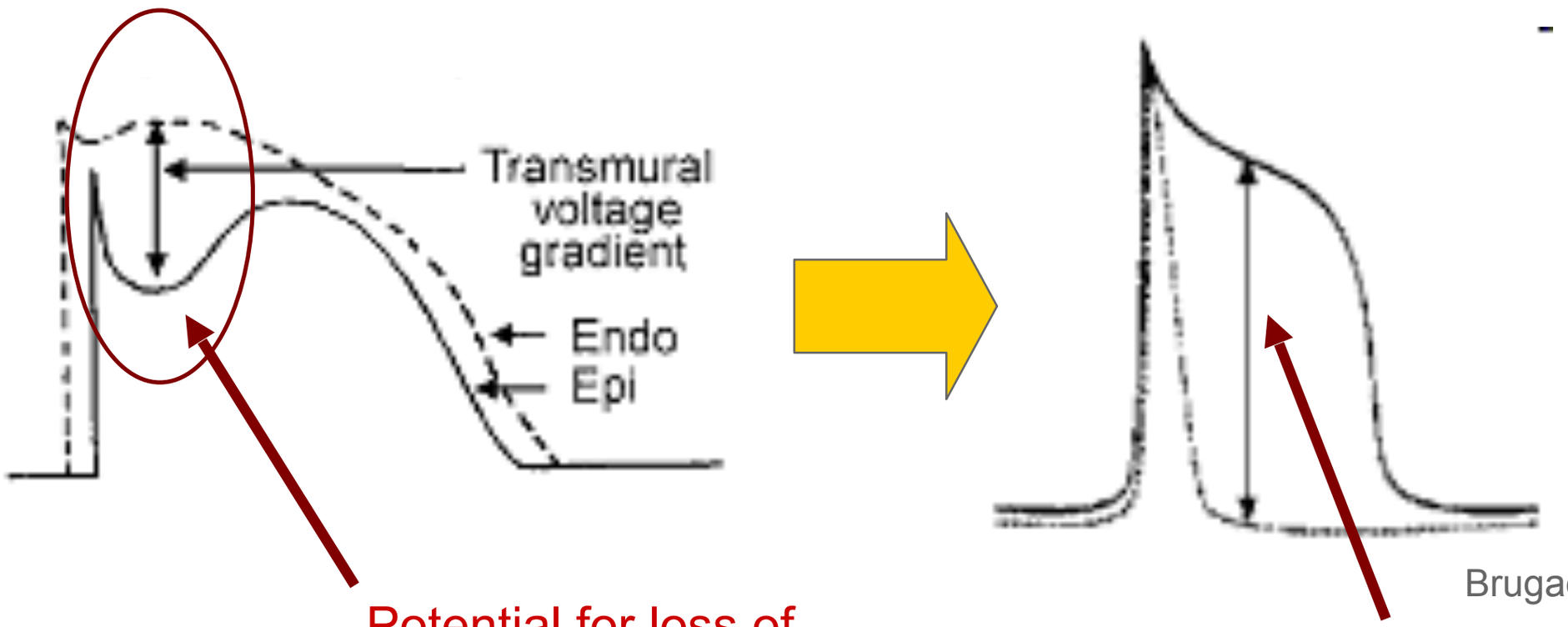


Figure 5 Brugada electrocardiogram-types. Type-1 is characterized by a complete or incomplete right bundle-branch block pattern with a coved morphology ST-segment elevation of ⩾ 2 mm in the right precordial leads (V1-V3) followed by a negative T-wave. In type-2, ST-segment elevation has a saddleback appearance with a high takeoff ST-segment elevation of > 2 mm, a trough displaying > 1-mm ST-elevation followed by a positive or biphasic T-wave. Type-3 has an ST-segment morphology that is either saddleback or coved with an ST-segment elevation of < 1 mm. Reproduced with permission, from ref.[69].

Brugada Syndrome

- Increased Phase 1 repolarization
- Shortened action potential → re-entry



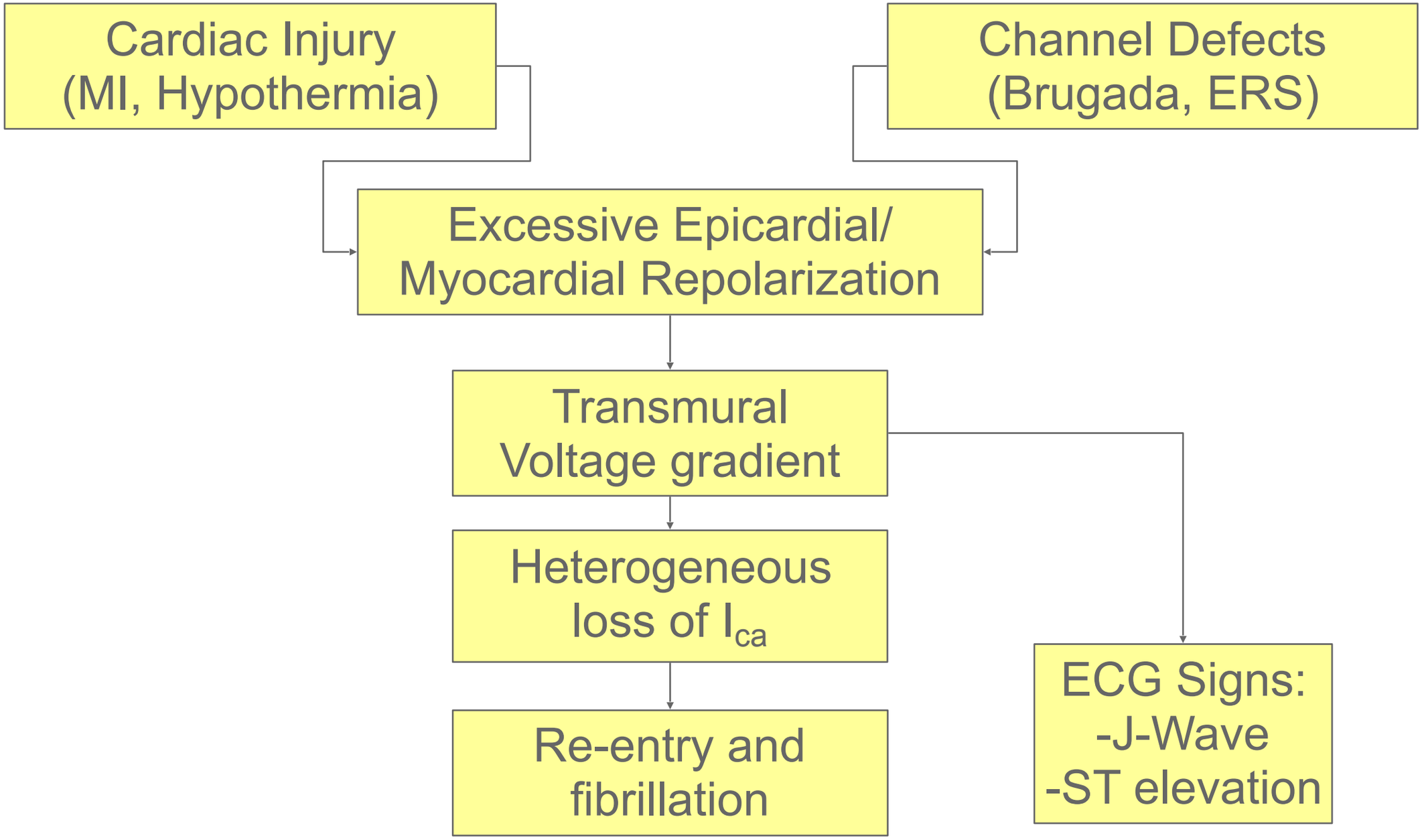
Brugada.org

Potential for loss of Phase 2 Ca^{+2} current

Heterogeneous Repolarization and risk of re-entry

Gussak and Antzelevitch 2000

Summary


Cardiac Injury
(MI, Hypothermia)
Channel Defects
(Brugada, ERS)
Excessive Epicardial/
Myocardial Repolarization
Transmural
Voltage gradient
Heterogeneous
loss of I_{ca}
Re-entry and
fibrillation
ECG Signs:
-J-Wave
-ST elevation

BRUGADA SYNDROME

- RBBB, ST-segment elevation and T inversion.
- mutations in at least 8 separate genes encoding the sodium, potassium, and calcium channels involved in the generation of cardiac myocyte action potential
- sudden cardiac death in up to 20%
- (12/10,000) in Southeast Asia and less frequent cases (5/10,000) in the Western hemisphere
- bangungut in Philippines, lai tai in Thailand, most common cause of SCD in young males in night

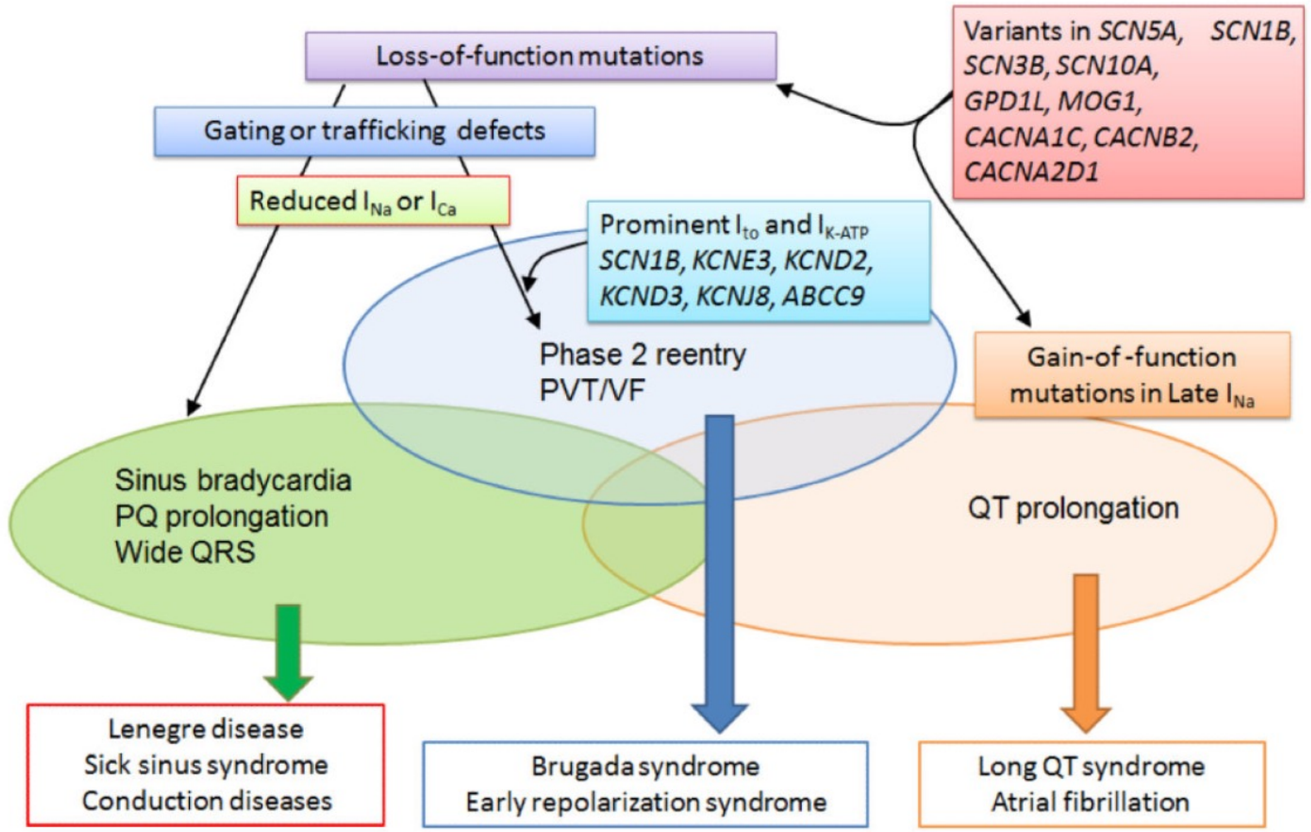
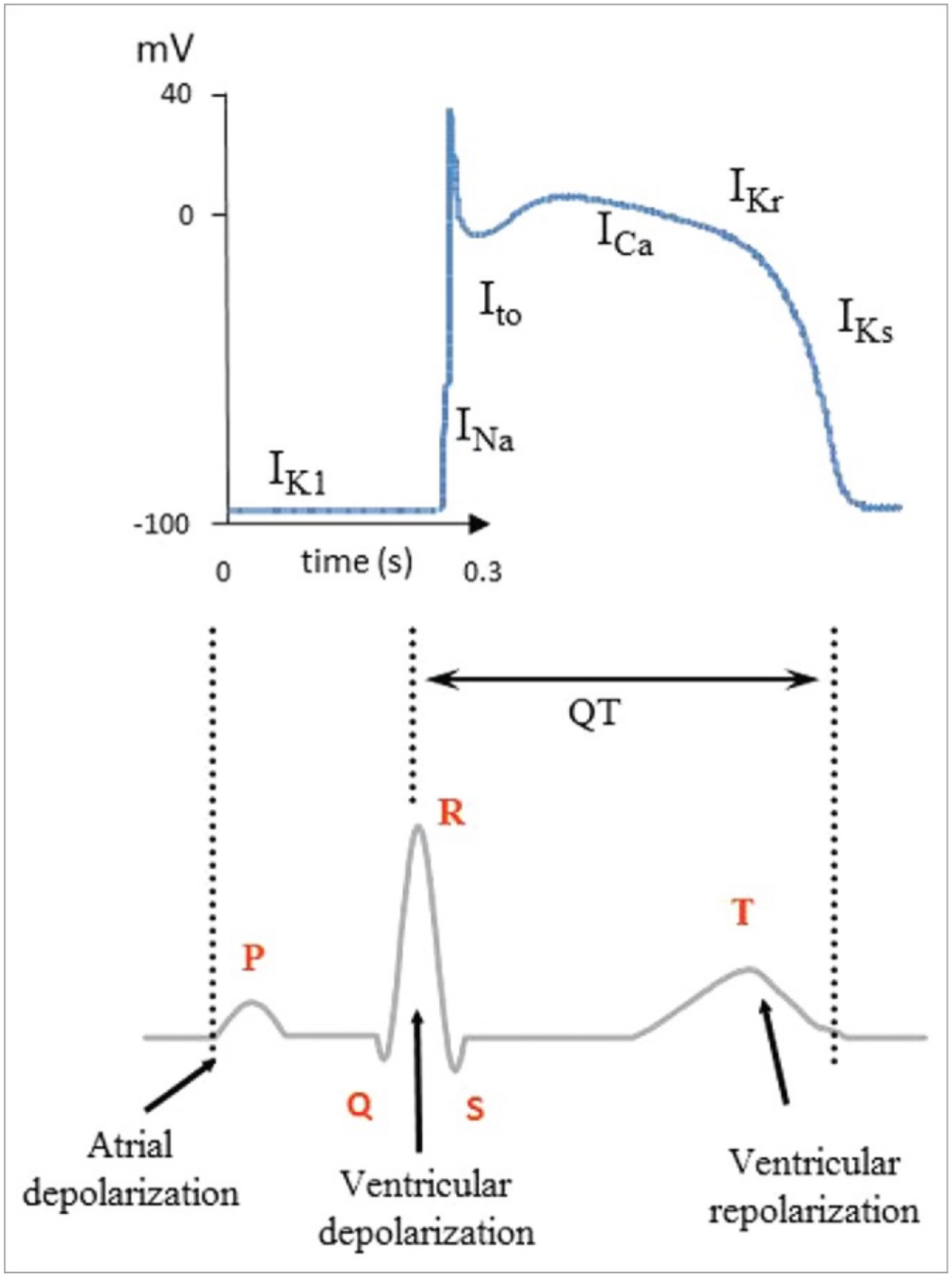
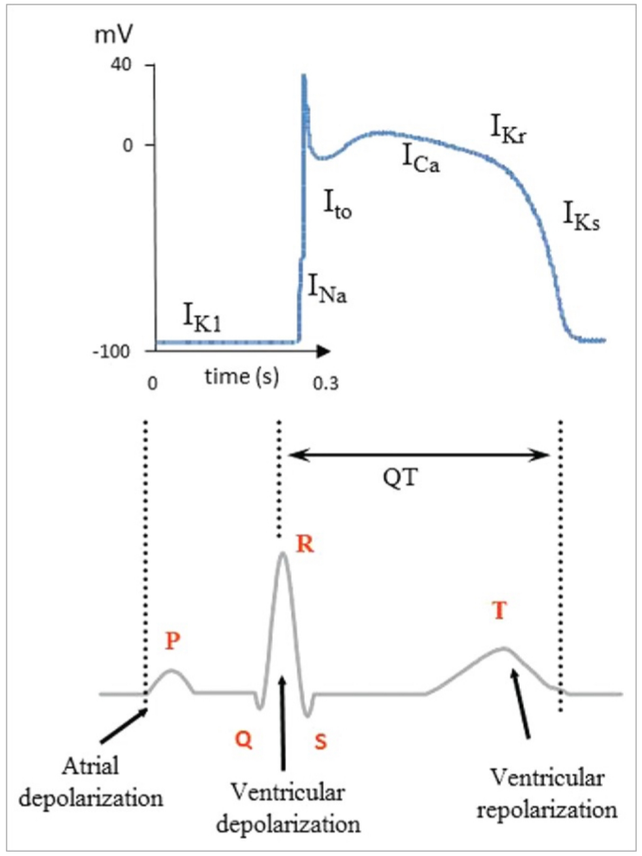


Figure 2.
Schematic showing the three categories of overlap syndromes resulting from genetic defects resulting in loss of function of sodium (I_{Na}) and/or calcium (I_{Ca}) channel current. In the absence of prominent I_{to} or $I_{K\text{-}ATP}$, loss-of-function mutations in the inward currents result in various manifestations of conduction disease. In the presence of prominent I_{to} or $I_{K\text{-}ATP}$, loss-of-function mutations in inward currents cause conduction disease as well as the J wave syndromes (Brugada and Early Repolarization Syndromes). Early Repolarization Syndrome is believed to be caused by loss-of-function mutations of inward current in the presence of prominent I_{to} in certain regions of the left ventricle (LV), particularly the inferior wall of the LV. The genetic defects that contribute to BrS and ERS can also contribute to the development of long QT and conduction system disease, in some cases causing multiple expressions of these overlap syndromes. In some cases, structural defects contribute to the phenotype.



The top panel shows the ventricular action potential and the currents contributing to each phase. The bottom panel depicts the features and intervals seen in a typical electrocardiogram (e.g., the PR interval is the time between the P wave and the R wave).

CHANNEL	CHARACTERISTICS
Sodium Channels	
Fast Na^+	Phase 0 depolarization of non-pacemaker cardiac action potentials
Slow Na^+	"Funny" pacemaker current (I_f) in cardiac nodal tissue
Potassium Channels	
Inward rectifier (I_{ir} or I_{K1})	Contributes to late phase 3 repolarization; maintains phase 4 negative potential
Transient outward (I_{to})	Contributes to phase 1 of non-pacemaker cardiac action potentials
Delayed rectifiers (I_{Kr} and I_{Ks})	Phase 3 repolarization of cardiac action potentials
ATP-sensitive ($I_{K, ATP}$)	K_{ATP} channels; inhibited by ATP; therefore, open when ATP decreases during hypoxia; in vascular smooth muscle, adenosine removes the ATP inhibition and opens these channels, producing hyperpolariation and vasodilation.
Acetylcholine-activated ($I_{K, ACh}$)	Activated by acetylcholine; Gi-protein coupled
Calcium-activated ($I_{K, Ca}$ or BK_{Ca})	Open in response to Ca^{++} influx in vascular smooth muscle
Calcium Channels	
L-type (I_{Ca-L})	Slow inward, long-lasting current; phase 2 non-pacemaker cardiac action potentials and late phase 4 and phase 0 of SA and AV nodal cells; important in vascular smooth muscle contraction
T-type (I_{Ca-T})	Transient current that contributes to early phase 4 pacemaker currents in SA and AV nodal cells



The top panel shows the ventricular action potential and the currents contributing to each phase. The bottom panel depicts the features in a typical electrocardiogram (e.g., the PR interval is the time between the P wave and the R wave).

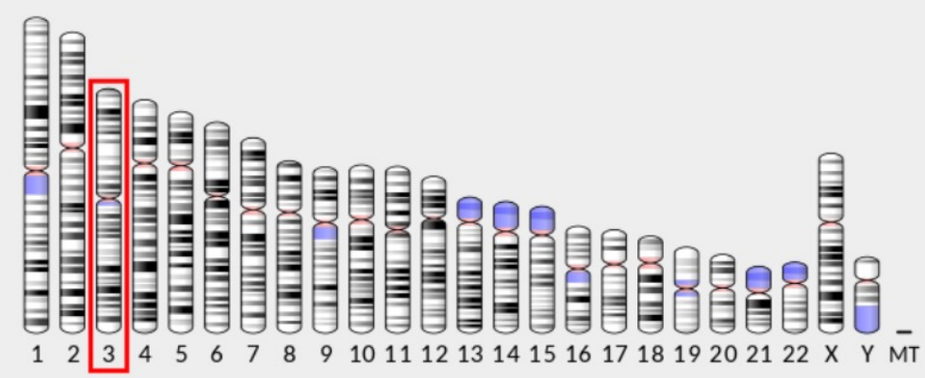
Many of the antiarrhythmic drugs that are used to treat cardiac arrhythmias have their action on sodium, calcium and potassium channels.

SCN5A gene
sodium voltage-gated channel alpha subunit 5

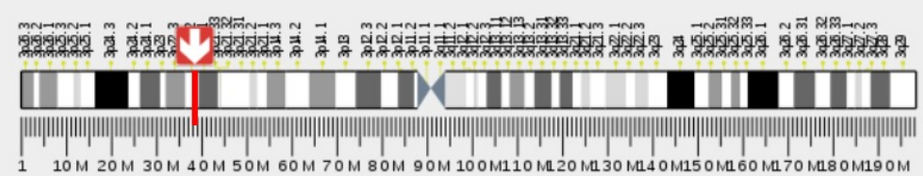
- Instructions for producing sodium channels
- >400 mutations in patients with Brugada Syndrome
- (SUNDS) Southeast Asia
- 10-30% mutations in SCN5A
- Loss of function mutation
- Reduction of sodium current during phase 0 (early upstroke) and phase 1 (early repolarization)

	sodium voltage-gated channel alpha subunit 5
External IDs	OMIM: 600163 MGI: 98251 HomoloGene: 22738 GeneCards: SCN5A

Gene location (Human)



Chr.	Chromosome 3 (human)[1]		



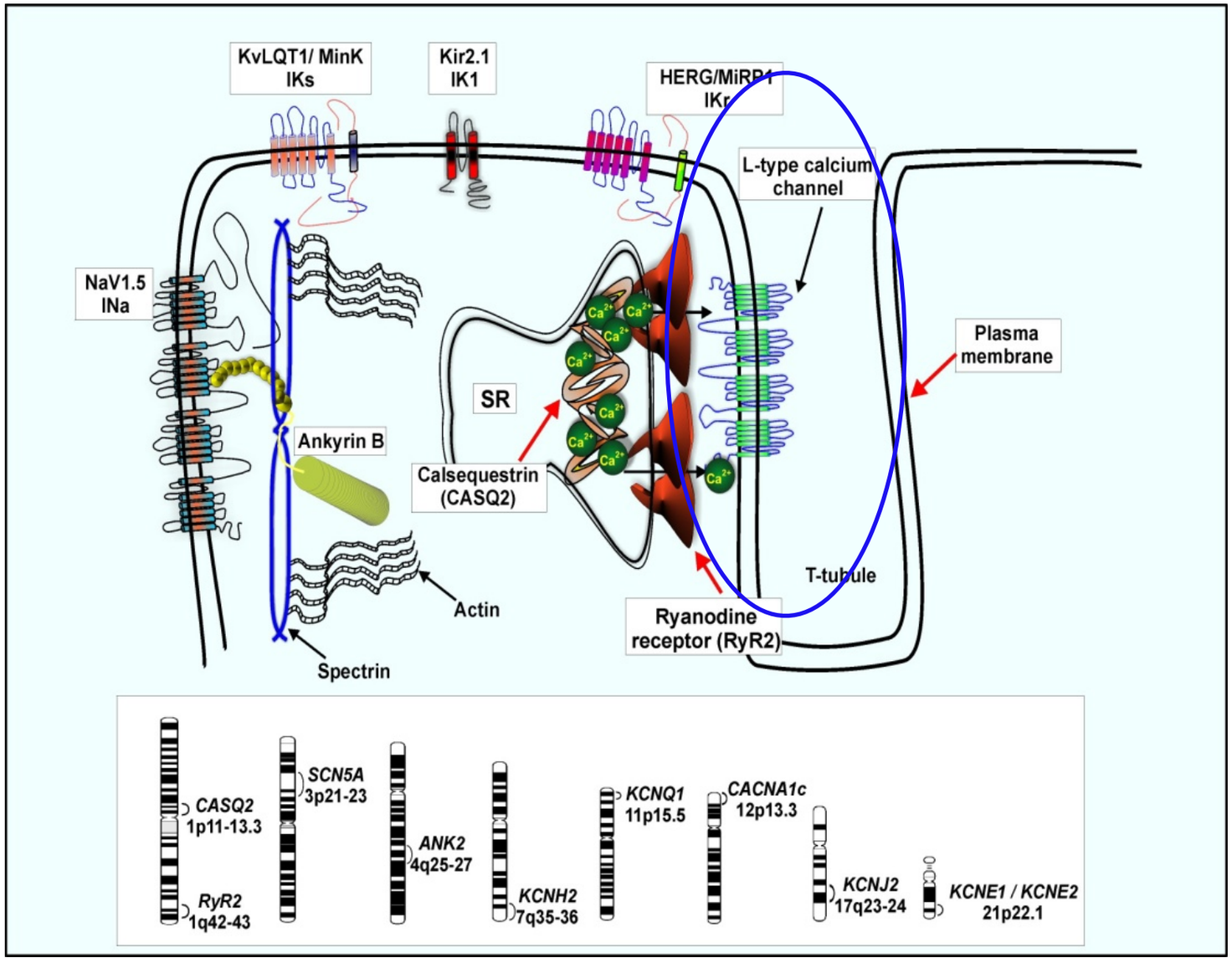
Band	3p22.2	**Start**	38,548,057 bp[1]
		End	38,649,673 bp[1]

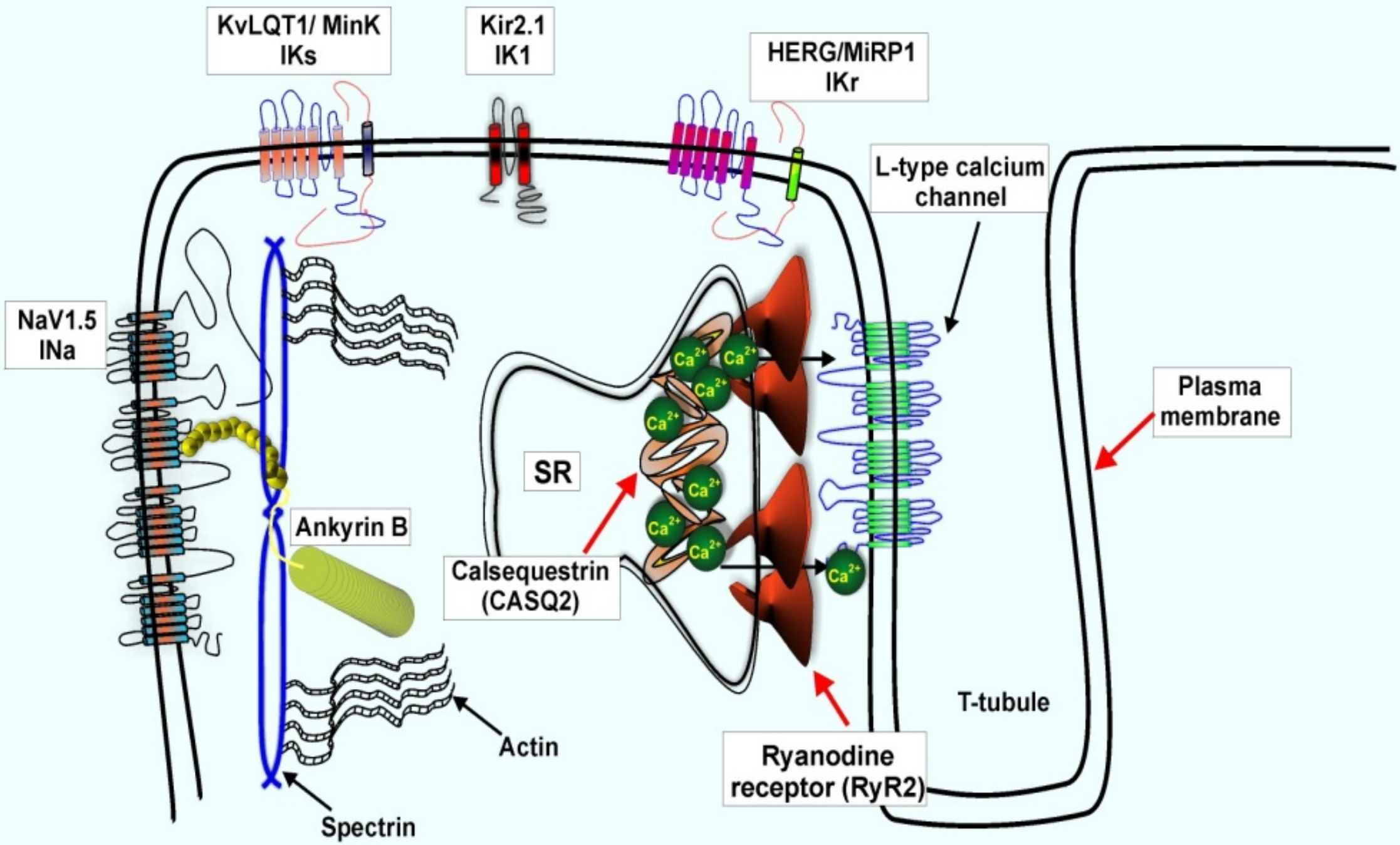
2013

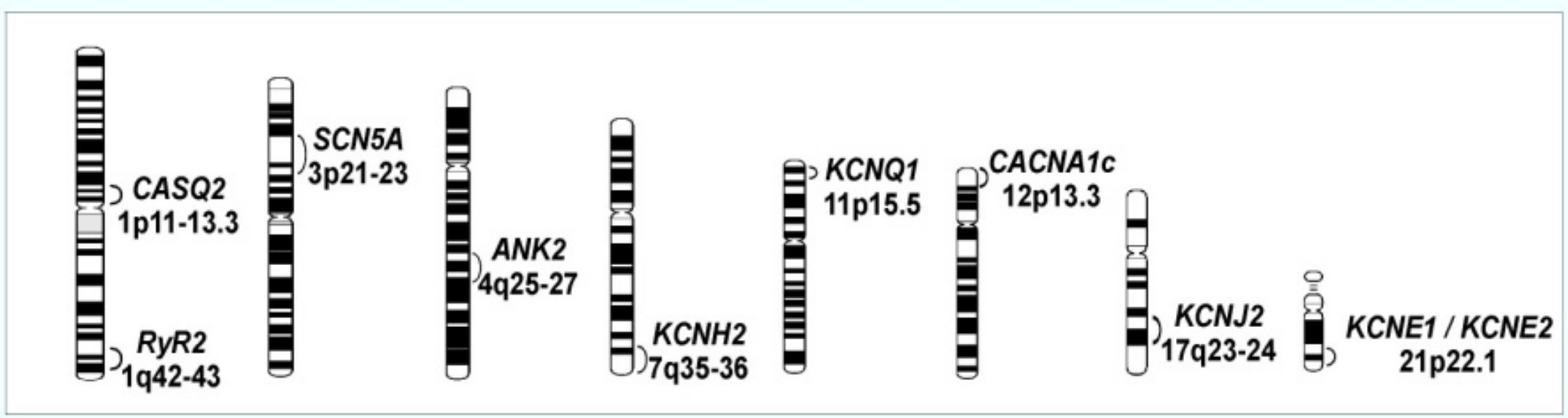
Cardiac L-type calcium channel (LTCC)

- Cardiac LTCC plays a pivotal role to regulate heart rhythm and contractility
- Mutations in LTCC reported to be associated with inherited arrhythmogenic diseases

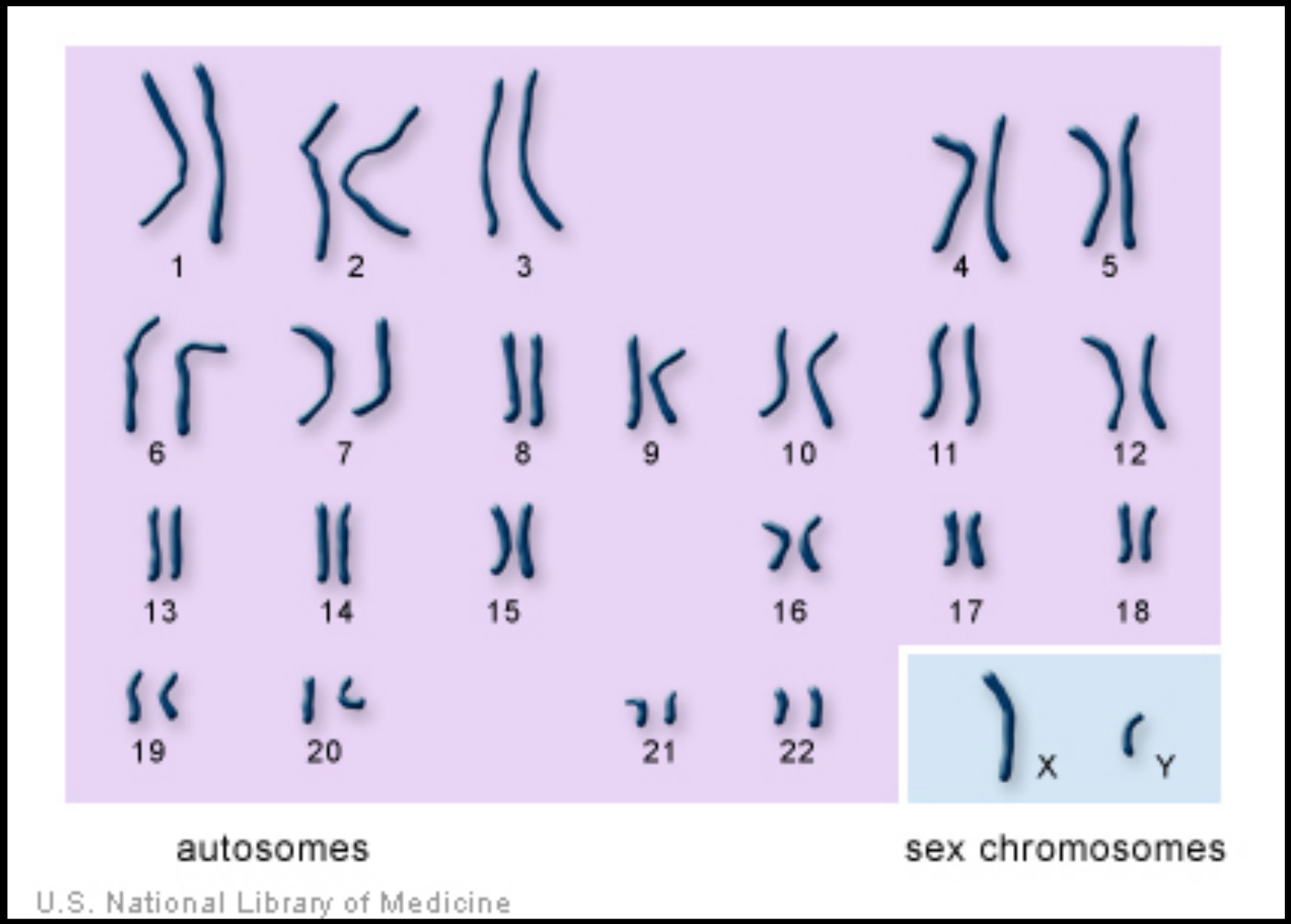
- **Brugada syndrome (BrS)**
- **Long & short QT syndrome (LQTS, SQTS)**
- **Idiopathic VF (IVF)**
- **Early repolarization syndrome (ERS)**

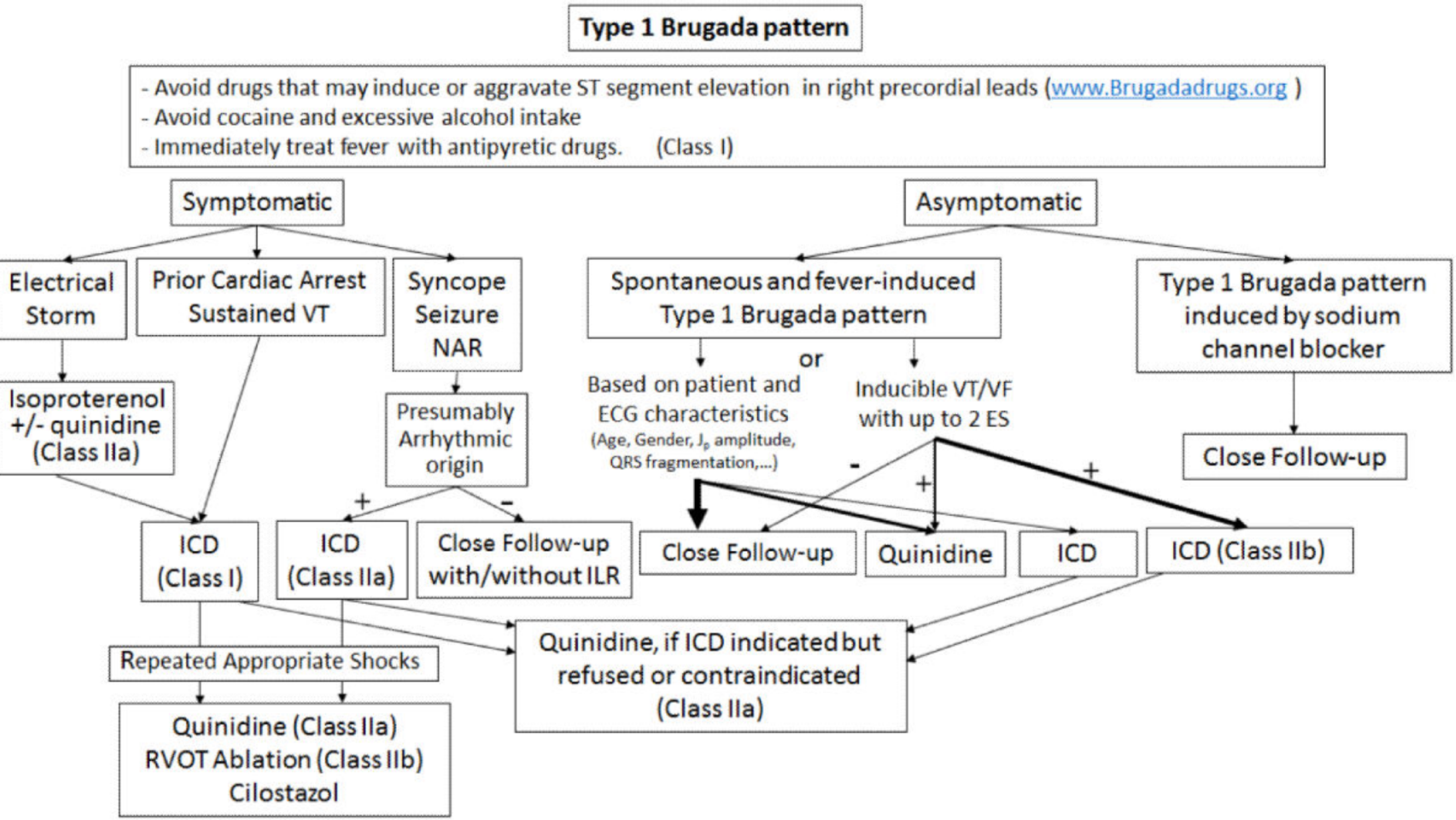



KvLQT1/ MinK
IKs
Kir2.1
IK1
HERG/MiRP1
IKr
L-type calcium
channel
NaV1.5
INa
Plasma
membrane
SR
Ankyrin B
Calsequestrin
(CASQ2)
T-tubule
Actin
Ryanodine
receptor (RyR2)
Spectrin


CASQ2
1p11-13.3
RyR2
1q42-43
SCN5A
3p21-23
ANK2
4q25-27
KCNH2
7q35-36
KCNQ1
11p15.5
CACNA1c
12p13.3
KCNJ2
17q23-24
KCNE1 / KCNE2
21p22.1

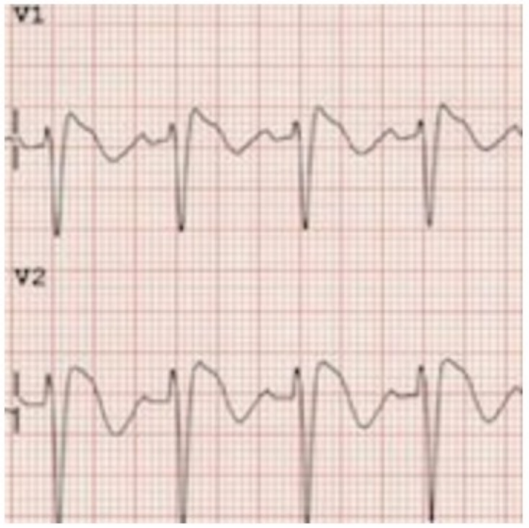
Homo sapiens sapiens

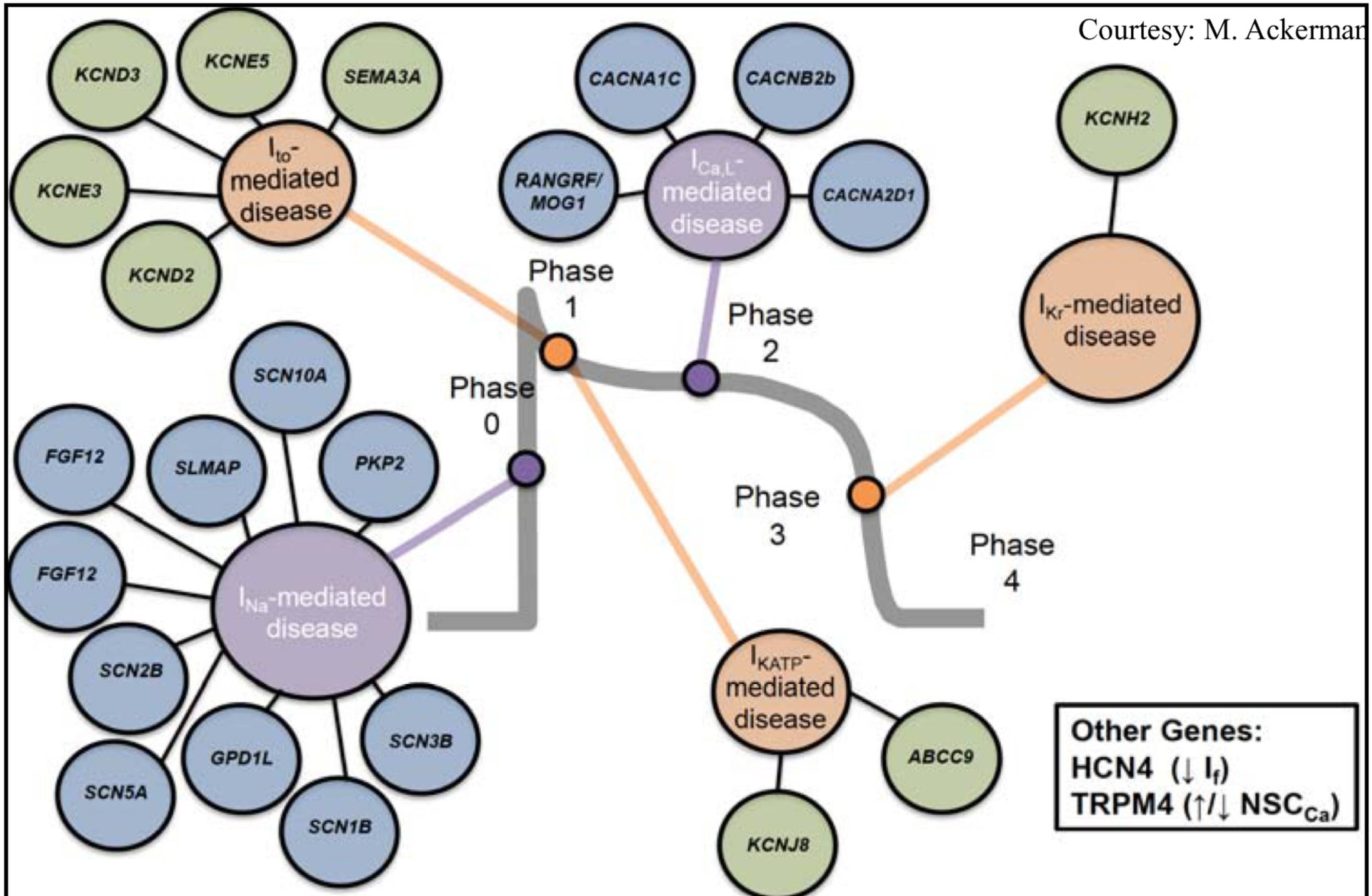


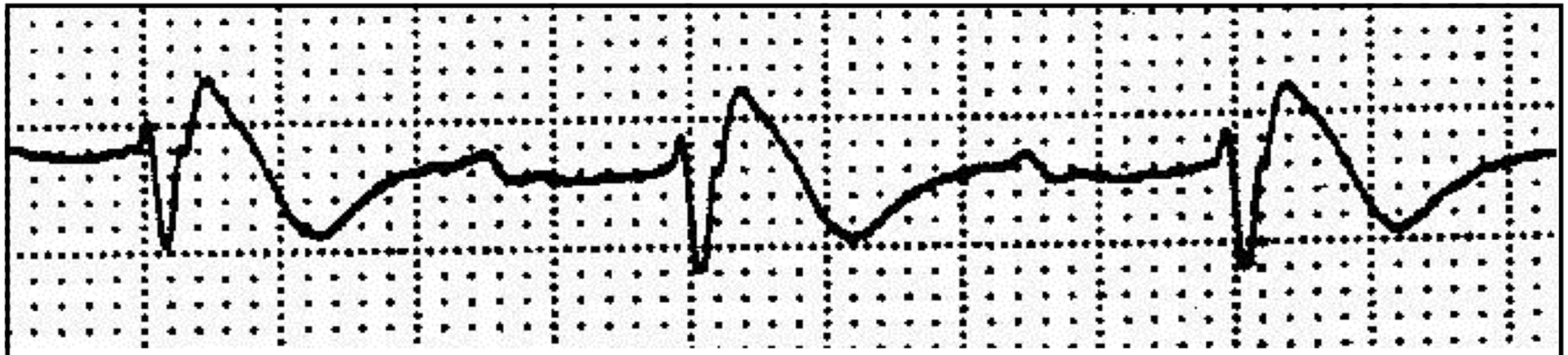

Type 1 Brugada pattern
- Avoid drugs that may induce or aggravate ST segment elevation in right precordial leads (www.Brugadadrugs.org)
- Avoid cocaine and excessive alcohol intake
- Immediately treat fever with antipyretic drugs. (Class I)
Symptomatic
Asymptomatic
Electrical Storm
Prior Cardiac Arrest Sustained VT
Syncope Seizure NAR
Spontaneous and fever-induced Type 1 Brugada pattern
Type 1 Brugada pattern induced by sodium channel blocker
Isoproterenol +/- quinidine (Class IIa)
Presumably Arrhythmic origin
or
Based on patient and ECG characteristics (Age, Gender, Jp amplitude, QRS fragmentation,...)
Inducible VT/VF with up to 2 ES
Close Follow-up
+
-
+
-
+
ICD (Class I)
ICD (Class IIa)
Close Follow-up with/without ILR
Close Follow-up
Quinidine
ICD
ICD (Class IIb)
Repeated Appropriate Shocks
Quinidine, if ICD indicated but refused or contraindicated (Class IIa)
Quinidine (Class IIa)
RVOT Ablation (Class IIb)
Cilostazol

Brugada Syndrome

- first described in 1992 in 8 patients with aborted sudden cardiac death – Brugada brothers

Syndrome	Pathology	ECG	Clinical Significance	Images / References
Brugada	Sodium channelopathy.40% familial (autosomal dominant)	RBBB with ST elevation in V1-3. Convex and concave ST variants	Risk of sudden death, mandates urgent ICD	


Courtesy: M. Ackerman
KCND3
KCNE5
SEMA3A
KCNE3
I_{to}-mediated disease
KCND2
CACNA1C
CACNB2b
RANGRF/ MOG1
$I_{Ca,L}$-mediated disease
CACNA2D1
KCNH2
I_{Kr}-mediated disease
Phase 1
Phase 2
Phase 0
Phase 3
Phase 4
SCN10A
FGF12
SLMAP
PKP2
FGF12
I_{Na}-mediated disease
SCN2B
SCN5A
GPD1L
SCN1B
SCN3B
I_{KATP}-mediated disease
ABCC9
KCNJ8
Other Genes:
HCN4 (↓ I_f)
TRPM4 (↑/↓ NSC_{Ca})



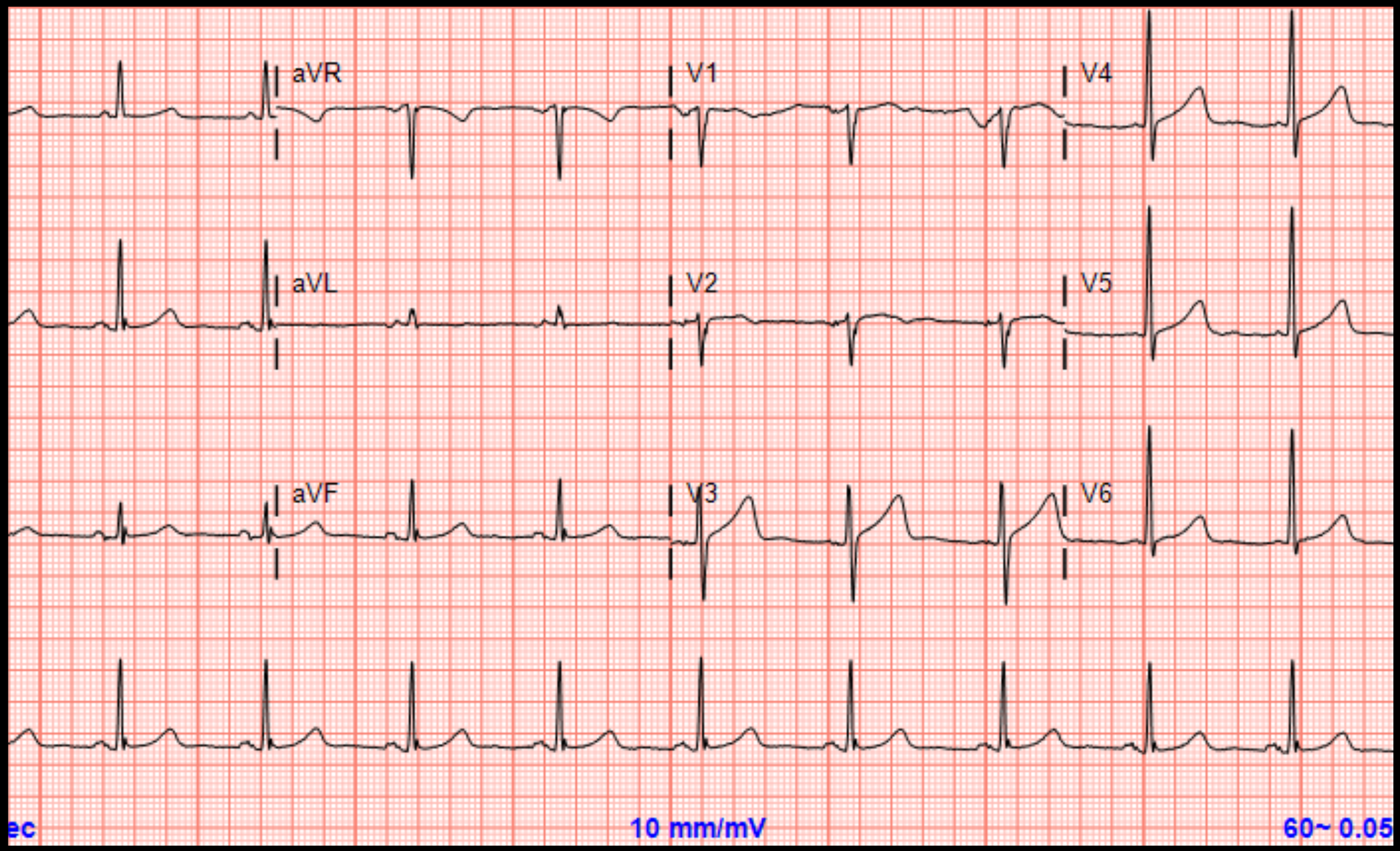
Conclusions:

- still much to learn!
- expanding genetics (pathophys.), role SCN5a
- symptomatic patients are at risk, ICD!
- asymptomatic patients, risk ill defined.
- plea for large registries!!!!

Brugada syndrome, Genetic testing

Why genetic testing?:

- ♥ **Reaching a diagnosis** -
- ♥ **Presymptomatic treatment** -
- ♥ **Risk stratification** ±
- ♥ **Gene-specific treatment** -



ERS

? Malignant

Ali A *et al*. Early repolarization syndrome

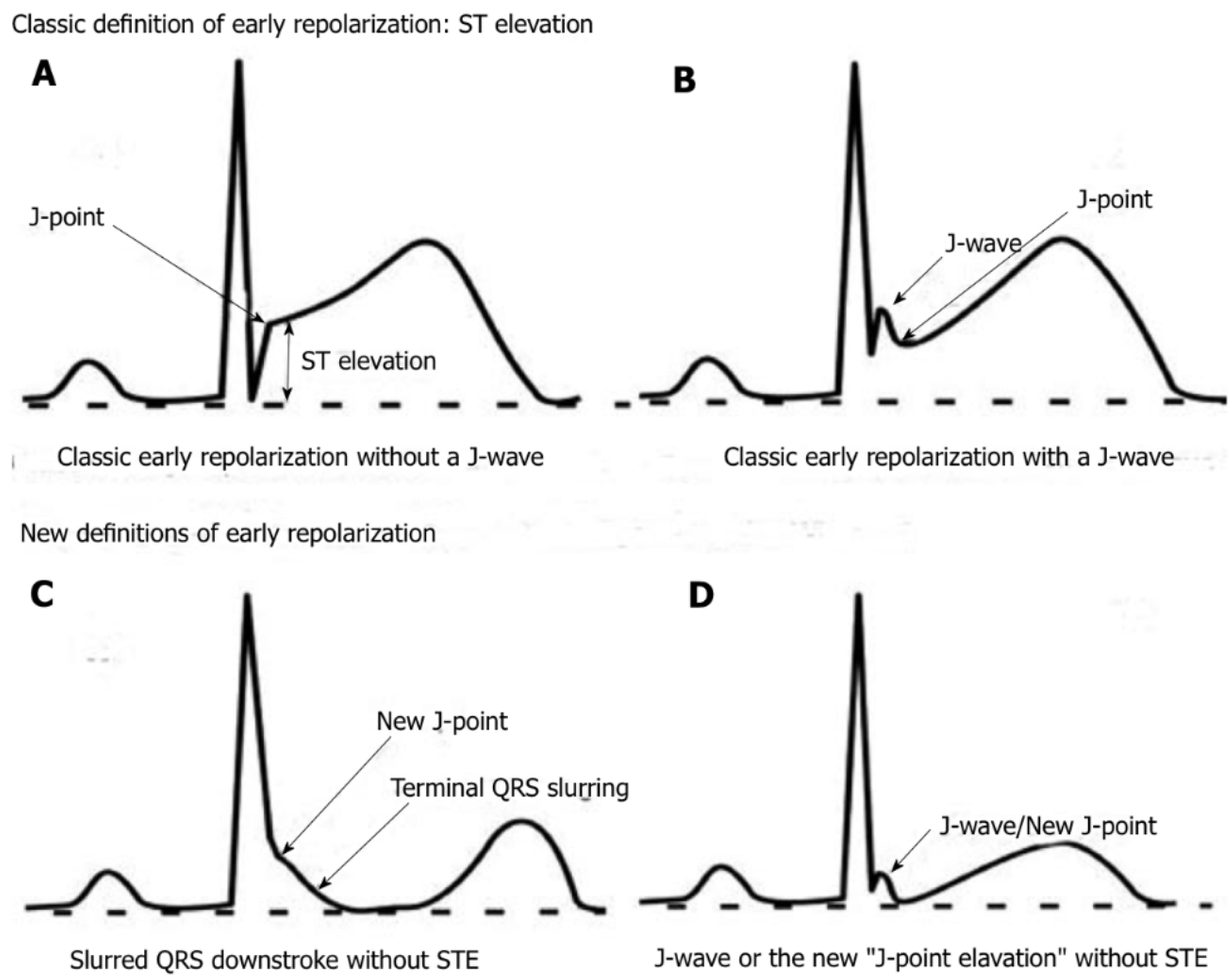
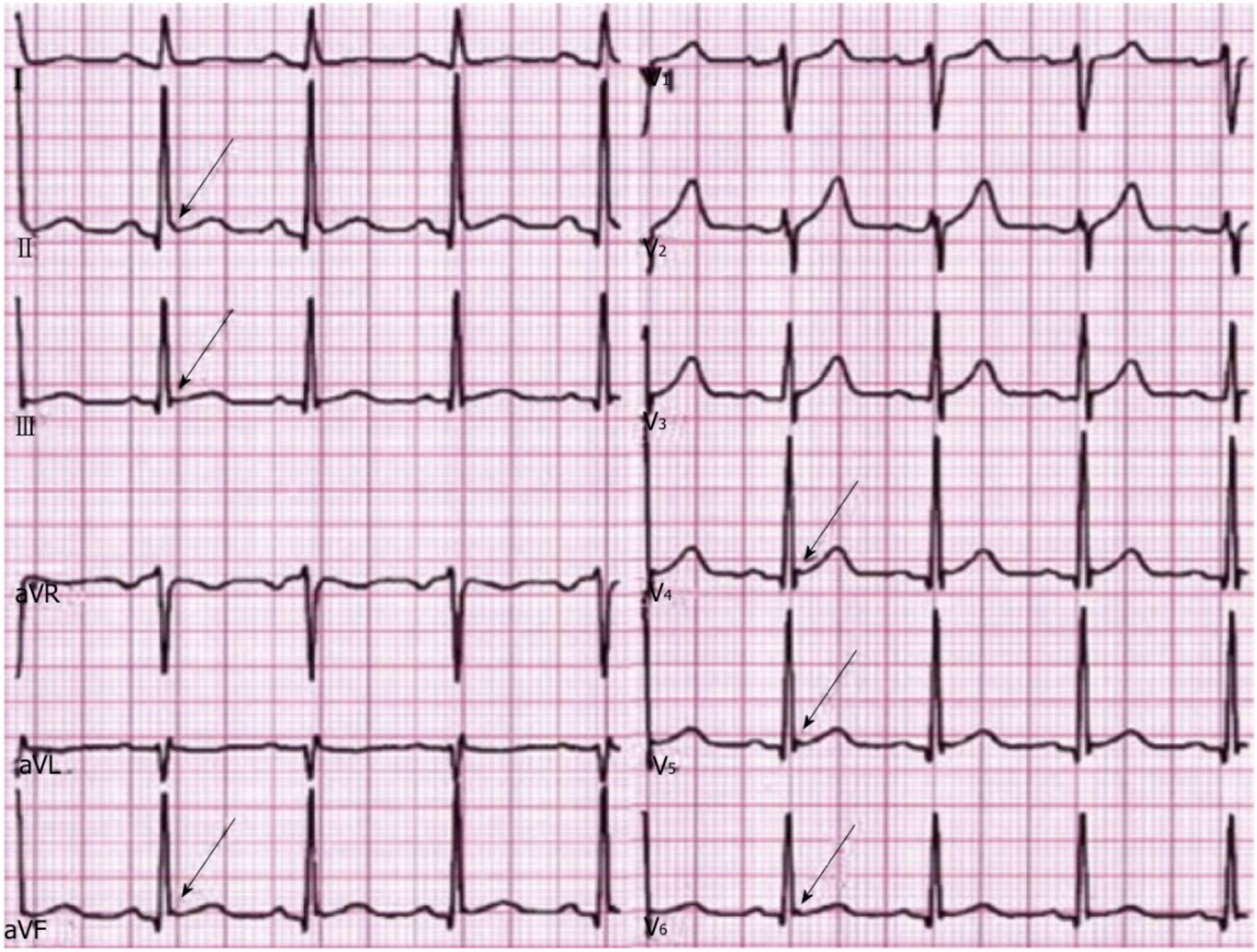


Figure 1 Examples of the classic and new definitions of early repolarization. Examples of the original (classic) and emerging (new) definitions of early repolarization (ER). A and B show the classic form of STE-type ER, which is the form identified by ECG software algorithms. Notice the presence of a J wave in (B), followed by an ascending/upsloping ST segment. Both forms are considered benign; C and D show the malignant form of ER demonstrated as slurring at the end of QRS complex (C) or a discrete notch/J wave (D) followed by a horizontal/downslopping ST segment (no ST elevation). Reproduced from ref.[49], with permission from the publisher. STE: ST elevation type ER; ECG: Electrocardiographic.

Ali A *et al*. Early repolarization syndrome



Malignant early repolarization: J-wave elevation (arrows) as slurring (lead II) and notching in the inferior and lateral leads and ascending ST segment in most leads.

- Three Subtypes of ERS

- Antzelevitch et al

- (1) type 1: It shows ER in the lateral precordial leads that is seen in healthy male athletes and has the lowest risk of malignant arrhythmias

- (2) type 2: It shows ER in the inferior and inferolateral leads and is associated with a greater risk of malignant arrhythmias

- (3) type 3: It shows ER pattern in all ECG leads and has the highest risk of malignant arrhythmias and electrical storms

Table 1

J-wave Syndromes: Similarities and Differences

	J Wave Syndromes					
	Inherited				Acquired	
	ER in lateral leads ERS Type 1	**ER in inferior or infero-lateral leads ERS Type 2**	**Global ER ERS Type 3**	**Brugada Syndrome**	**Ischemia-mediated VT/VF**	**Hypothermia-mediated VT/VF**
Anatomic Location responsible for chief EP manifestations	Antero-lateral left ventricle	Inferior left ventricle	Left and right ventricles	Right ventricle	Left and right ventricles	Left and right ventricles
Leads Displaying J point/ J-wave abnormalities	I, V4–V6	II, III, aVF	Global	V1–V3	Any of 12 leads	Any of the 12 leads
Response of J wave amplitude/ST Elevation to: Bradycardia or pause Na+ channel blockers	Increase Little or no change	Increase Little or no change	Increase Little or no change	Increase Increase	N/A N/A	N/A N/A
Sex Dominance	Male	Male	Male	Male	Male[70,71]	Either gender
VF	Rare Commonly seen in healthy men and athletes[11,31,51]	Yes[21,23]	Yes, Electrical Storms[7, 27]	Yes	Yes	Yes
Response to Quinidine	Normalization of J point elevation and inhibition of VT/VF	Normalization of J point elevation and inhibition of VT/VF	Limited data; Normalization of J point elevation and inhibition of VT/VF	Normalization of J point elevation and inhibition of VT/VF	Limited data	Inhibition of VT/VF[59]
Response to Isoproterenol	Normalization of J point elevation and inhibition of VT/VF	Normalization of J point elevation and inhibition of VT/VF	Limited data;	Normalization of J point elevation and inhibition of VT/VF	N/A	N/A
Gene Mutations	*CACNA1C, CACNB2B*[57]	*KCNJ8*[56], *CACNA1C, CACNB2B*[57]	*CACNA1C*[57]	*SCN5A, CACNA1C, CACNB2B, GPD1-L, SCN1B, KCNE3, SCN3B, KCNJ8*	SCN5A[72]	N/A

EP=electrophysiology; N/A=not available; ERS=early repolarization syndrome; VT=ventricular tachycardia; VF=ventricular fibrillation

Table 3 Heart Rhythm Society/European Heart Rhythm Association/Asia Pacific Heart Rhythm Society consensus statement on the diagnosis and management of primary inherited arrhythmia syndromes recommendations for therapeutic interventions in early repolarization syndrome

Expert consensus recommendations on early repolarization therapeutic interventions		
Class Ⅰ	1	ICD implantation is recommended in patients with a diagnosis of ER syndrome who have survived a cardiac arrest
Class Ⅱa	2	Isoproterenol infusion can be useful in suppressing electrical storms in patients with a diagnosis of ER syndrome
	3	Quinidine in addition to an ICD can be useful for secondary prevention of VF in patients with a diagnosis of ER syndrome
Class Ⅱb	4	ICD implantation may be considered in symptomatic family members of ER syndrome patients with a history of syncope in the presence of ST-segment elevation > 1 mm in 2 or more inferior or lateral leads
	5	ICD implantation may be considered in asymptomatic individuals who demonstrate a high-risk ER ECG pattern (high J-wave amplitude, horizontal/descending ST segment) in the presence of a strong family history of juvenile unexplained sudden death with or without a pathogenic mutation
Class Ⅲ	6	ICD implantation is not recommended in asymptomatic patients with an isolated ER ECG pattern

ER: Early repolarization; ECG: Electrocardiogram; ICD: Implantable cardioverter-defibrillator.

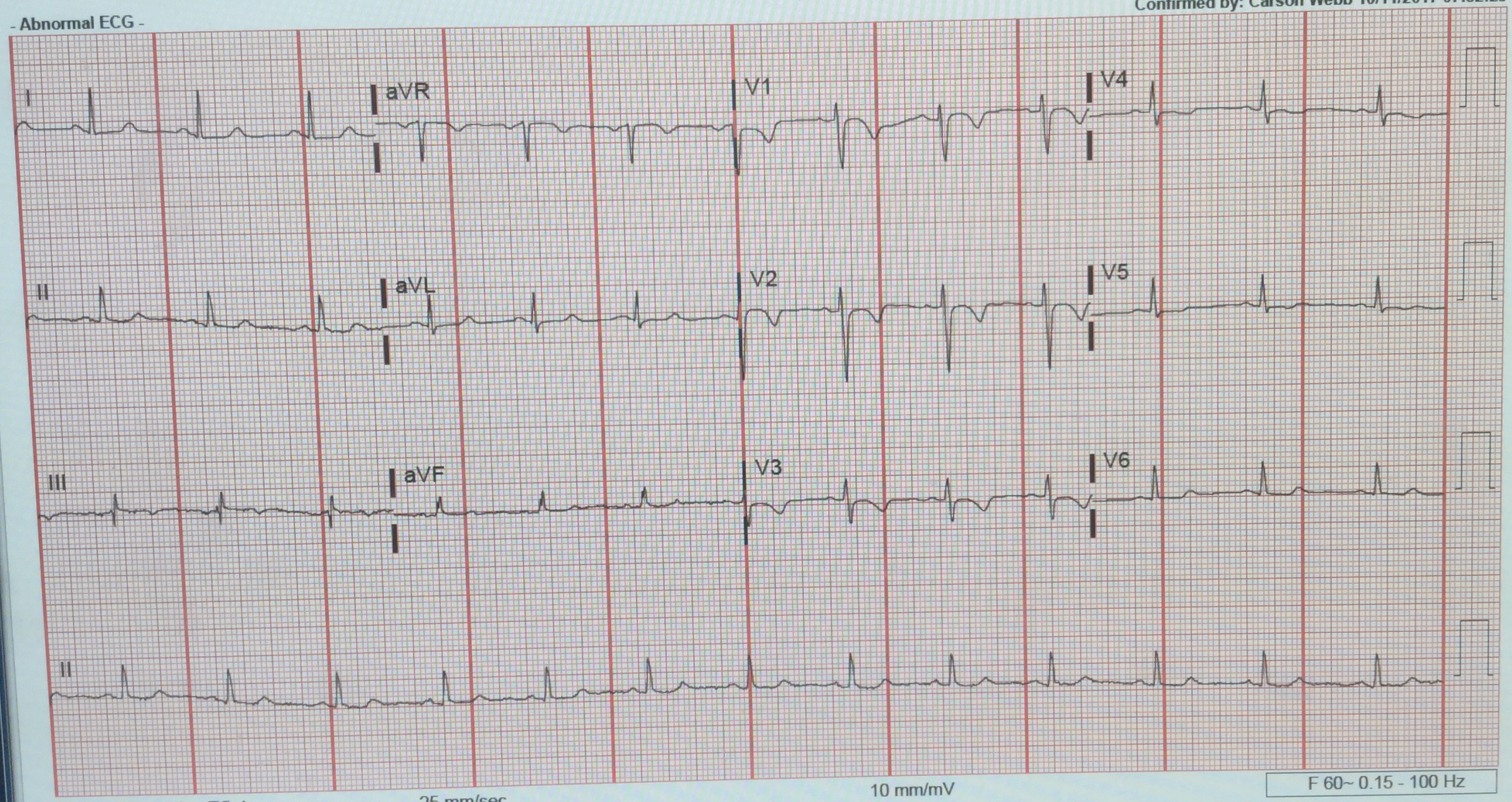
Rx:
Dx:

Rate	80	Sinus rhythm
PR	125	Borderline intraventricular conduction delay
QRSD	114	T-wave inversions in the anterior leads, consider ischemia, clinical correlation is necessary.
QT	371	Electronically signed on 10-11-17 07:32:23 AKDT by Carson Webb
QTc	428	
--AXIS--		
P	14	
QRS	40	
T	12	

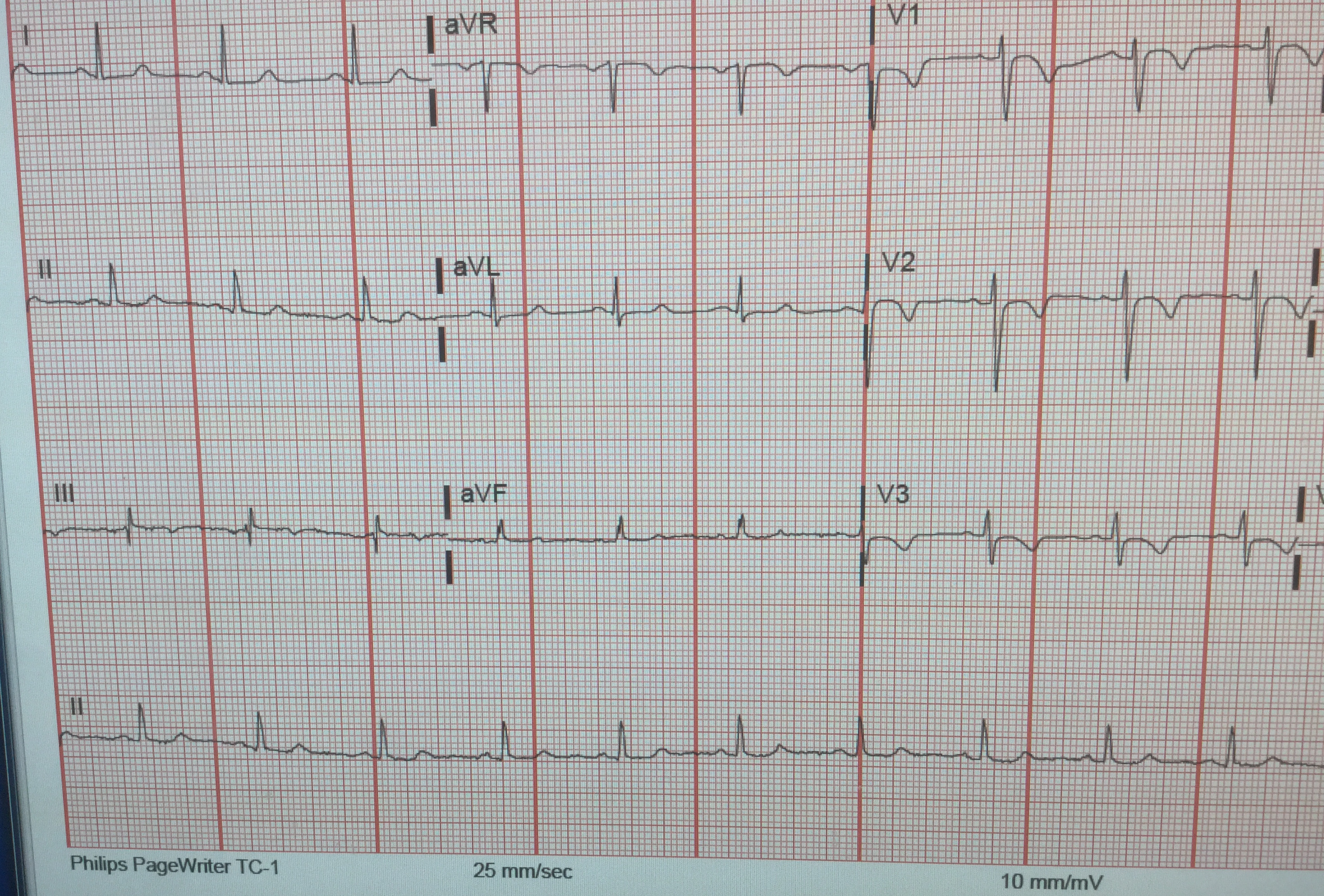
Req MD: Carson Webb
FIN# 3013791821
Field2:
Field3:
Field4:

Confirmed by: Carson Webb 10/11/2017 07:32:23

- Abnormal ECG -



Philips PageWriter TC-1 25 mm/sec 10 mm/mV F 60~ 0.15 - 100 Hz


I
aVR
V1
II
aVL
V2
III
aVF
V3
II
Philips PageWriter TC-1
25 mm/sec
10 mm/mV

ECG - ARVD

Epsilon Wave (Most Specific Finding - 30%)

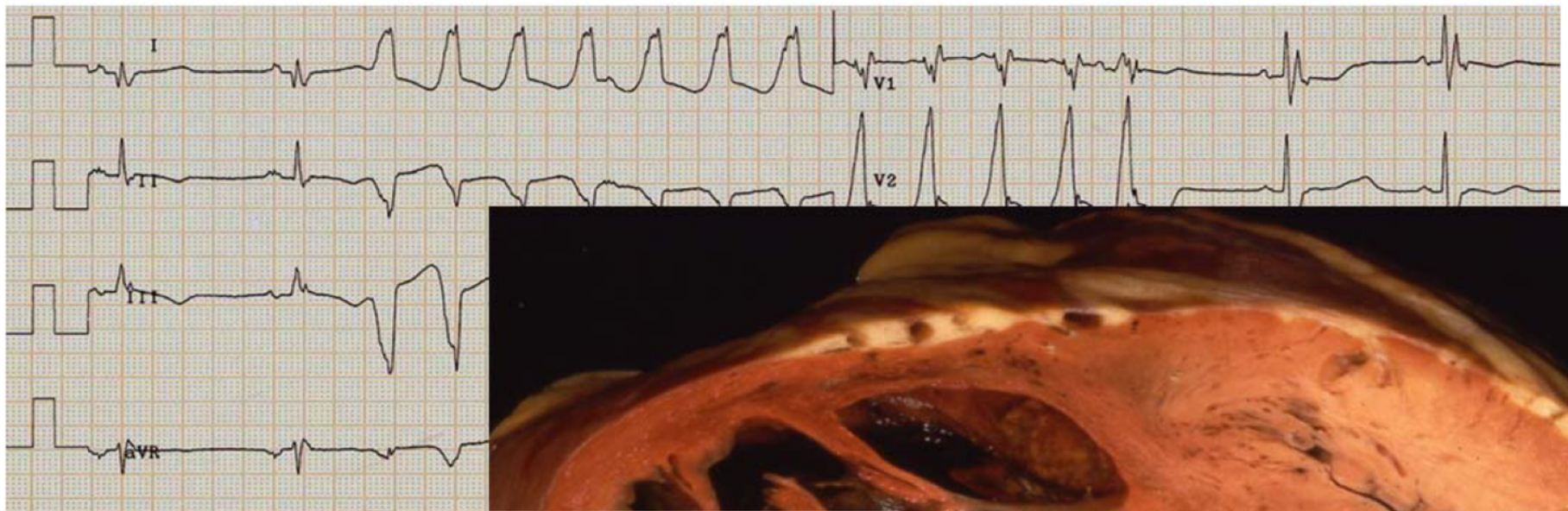
T Wave inversions in V1 - V3 (85%)

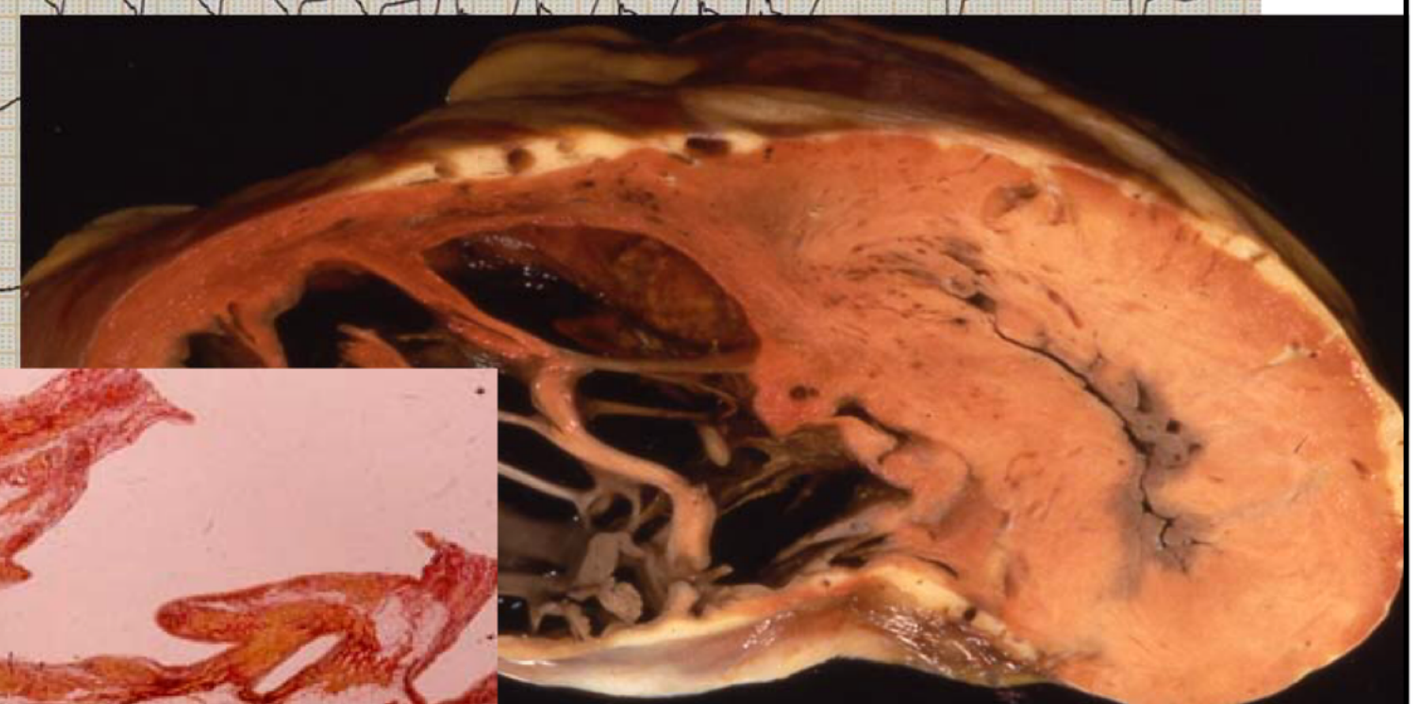
Prolonged S Wave Upstroke of 55ms in V1-V3 (95%)

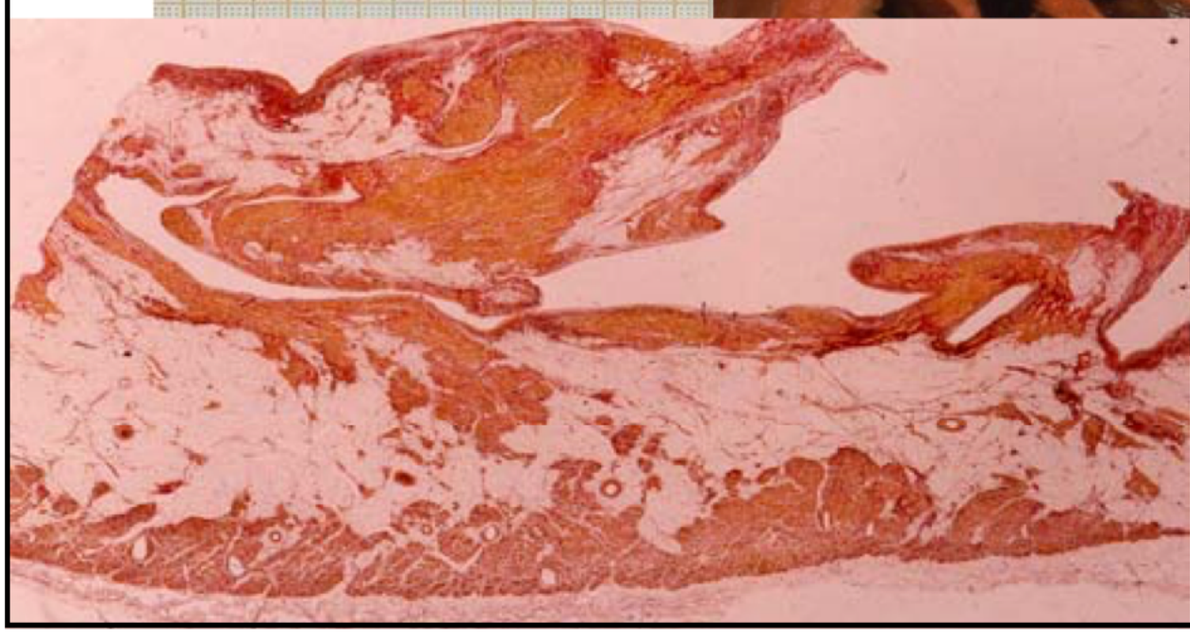
QRS Widening of 110ms in V1 - V3

Paroxysmal VT with LBBB Morphology

Arrhythmogenic RV cardiomyopathy







LBBB arrhythmias
Palpitations, dizziness
Sudden cardiac death

Section # – Disease	Diagnostic	Prognostic	Therapeutic
Section I – LQTS	+++	+++	++
Section II – CPVT	+++	+	-
Section III – BrS	+	+	-
Section IV – CCD	+	+	+
Section V – SQTS	+/-	-	-
Section VI – AF	-	-	-
Section VII – HCM	+++	++	+
Section VIII – ACM/ARVC	+	+/-	-
Section IX – DCM	+/-	-	-
Section IX – DCM + CCD	++	++	+
Section X – LVNC	+	-	-
Section XI – RCM	+	+	+

2010 revision (Table 1)..

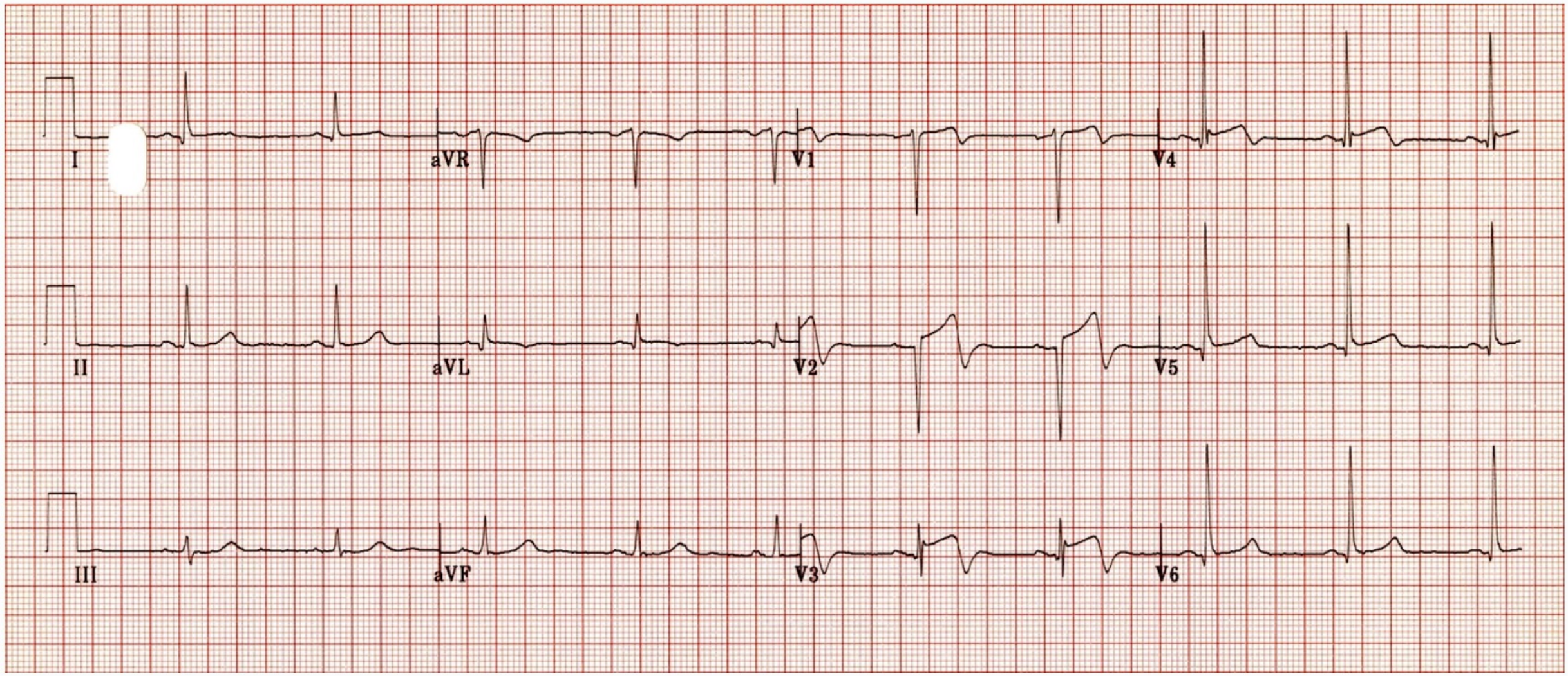
Table 1. 2010 Task Force Criteria for the Diagnosis of Arrhythmogenic Right Ventricular Cardiomyopathy

I. Global and/or regional dysfunction and structural alterations	
Major	By 2-dimensional echocardiogram: regional RV akinesia, dyskinesia, or aneurysm and 1 of the following (end diastole):• PLAX RVOT≥32mm (corrected for body size [PLAX/BSA]≥19 mm/m^2)• PSAX RVOT≥ 36mm (corrected for body size [PSAX/BSA]≥21 mm/m^2)• O fractional area change≤33%
	By MRI: regional RV akinesia or dyskinesia or dyssynchronous RV contraction and 1 of the following:• Ratio of RV end-diastolic volume to BSA≥110 mL/m^2 (male) or ≥100 mL/m^2 (female)• O RV ejection fraction≤40%
	By RV angiography: regional RV akinesia, dyskinesia, or aneurysm
Minor	By 2-dimensional echocardiogram: regional RV akinesia or dyskinesia and 1 of the following (end diastole):• PLAX RVOT≥29 to <32mm (corrected for body size [PLAX/BSA]≥16 to <19 mm/m^2)• PSAX RVOT≥32 to <36mm (corrected for body size [PSAX/BSA]≥18 to <21 mm/m^2)• O fractional area change >33% to ≤40%
	By MRI: regional RV akinesia or dyskinesia or dyssynchronous RV contraction and 1 of the following:• Ratio of RV end-diastolic volume to BSA≥100 to < 110 mL/m^2 (male) or ≥90 to <100 mL/m^2 (female)• Or RV ejection fraction >40% to ≤45%

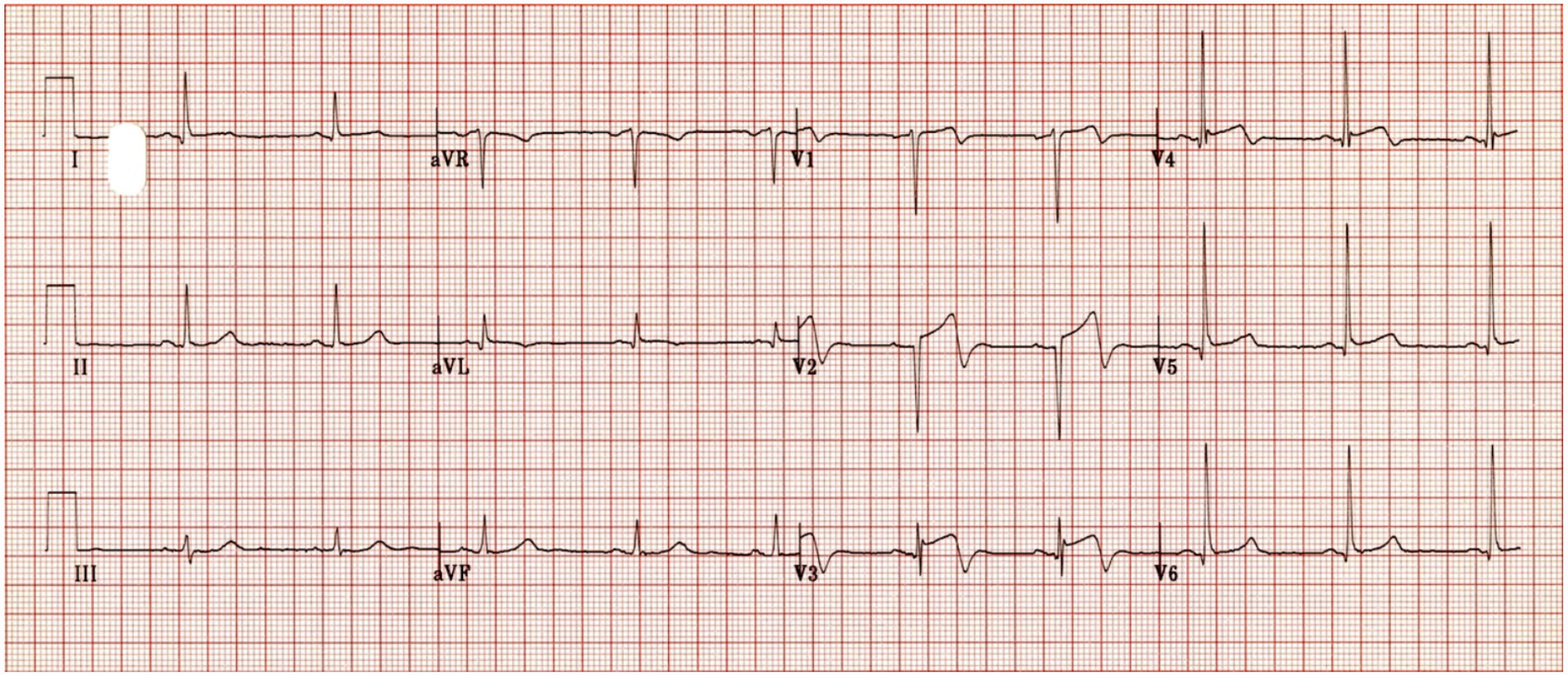
II. Tissue characterisation of the wall	
Major	Residual myocytes<60% by morphometric analysis (or <50% if estimated), with fibrous replacement of the RV free wall myocardium in ≥1 sample, with or without fatty replacement of tissue on endomyocardial biopsy
Minor	Residual myocytes 60% to 75% by morphometric analysis (or 50% to 65% if estimated), with fibrous replacement of the RV free wall myocardium in ≥1 sample, with or without fatty replacement of tissue on endomyocardial biopsy
III. Repolarisation abnormalities	
Major	Inverted T waves in right precordial leads (V_1, V_2, and V_3) or beyond in individuals >14 years of age (in the absence of complete RBBB QRS≥120 ms)
Minor	Inverted T waves in leads V_1 and V_2 in individuals>14 years of age (in the absence of complete RBBB) or in V_4, V_5, or V_6Inverted T waves in leads V_1, V_2, V_3, and V_4 in individuals>14 years of age in the presence of complete RBBB
IV. Depolarisation/conduction abnormalities	

IV. Depolarisation/conduction abnormalities	
Major	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 by SAECG in ≥1 of 3 parameters in the absence of a QRS duration of ≥110ms on the standard ECG: filtered QRS duration (fQRS)≥114 ms; duration of terminal QRS<40μV (low-amplitude signal duration)≥38 ms; root-mean-square voltage of terminal 40 ms≤20μVTerminal activation duration of QRS≥55ms 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 RBBB
V. Arrhythmias	
Major	Nonsustained or sustained ventricular tachycardia of left bundle-branch morphology with superior axis (negative or indeterminate QRS in leads II, III, and aVF and positive in lead aVL)
Minor	Nonsustained or sustained ventricular tachycardia of RVOT configuration, LBBB morphology with inferior axis (positive QRS in leads II, III, and aVF and negative in lead aVL) or of unknown axis>500 ventricular extrasystoles per 24h (Holter)

V. Arrhythmias	
Major	Nonsustained or sustained ventricular tachycardia of left bundle-branch morphology with superior axis (negative or indeterminate QRS in leads II, III, and aVF and positive in lead aVL)
Minor	Nonsustained or sustained ventricular tachycardia of RVOT configuration, LBBB morphology with inferior axis (positive QRS in leads II, III, and aVF and negative in lead aVL) or of unknown axis>500 ventricular extrasystoles per 24h (Holter)
VI. Family history	
Major	ARVC/D confirmed in a first-degree relative who meets current Task Force criteriaARVC/D confirmed pathologically at autopsy or surgery in a first-degree relativeIdentification of a pathogenic mutation * categorized as associated or probably associated with ARVC/D in the patient under evaluation
Minor	History of ARVC/D in a first-degree relative in whom it is not possible or practical to determine whether the family member meets current Task Force criteriaPremature sudden death (<35 years of age) due to suspected ARVC/D in a first-degree relativeARVC/D confirmed pathologically or by current Task Force criteria in second-degree relative

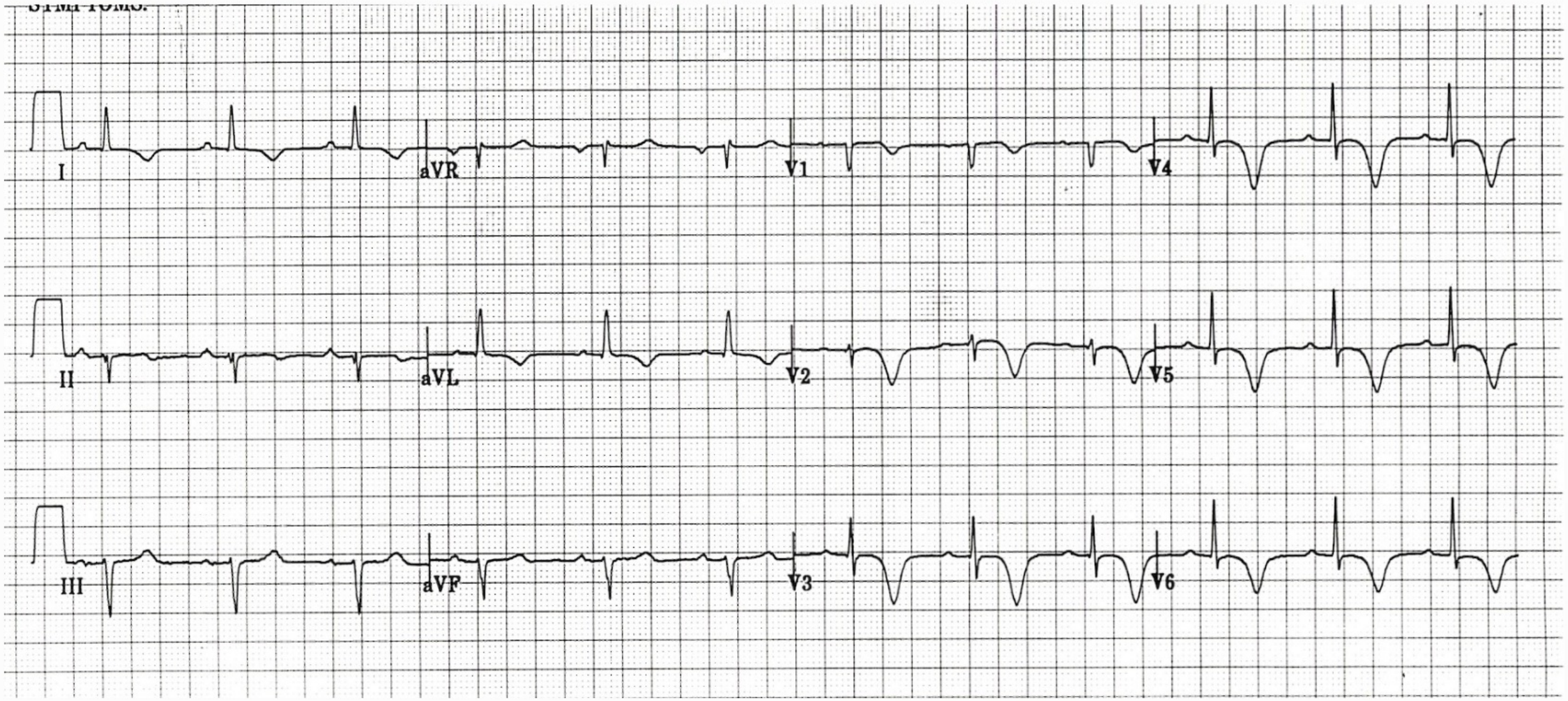


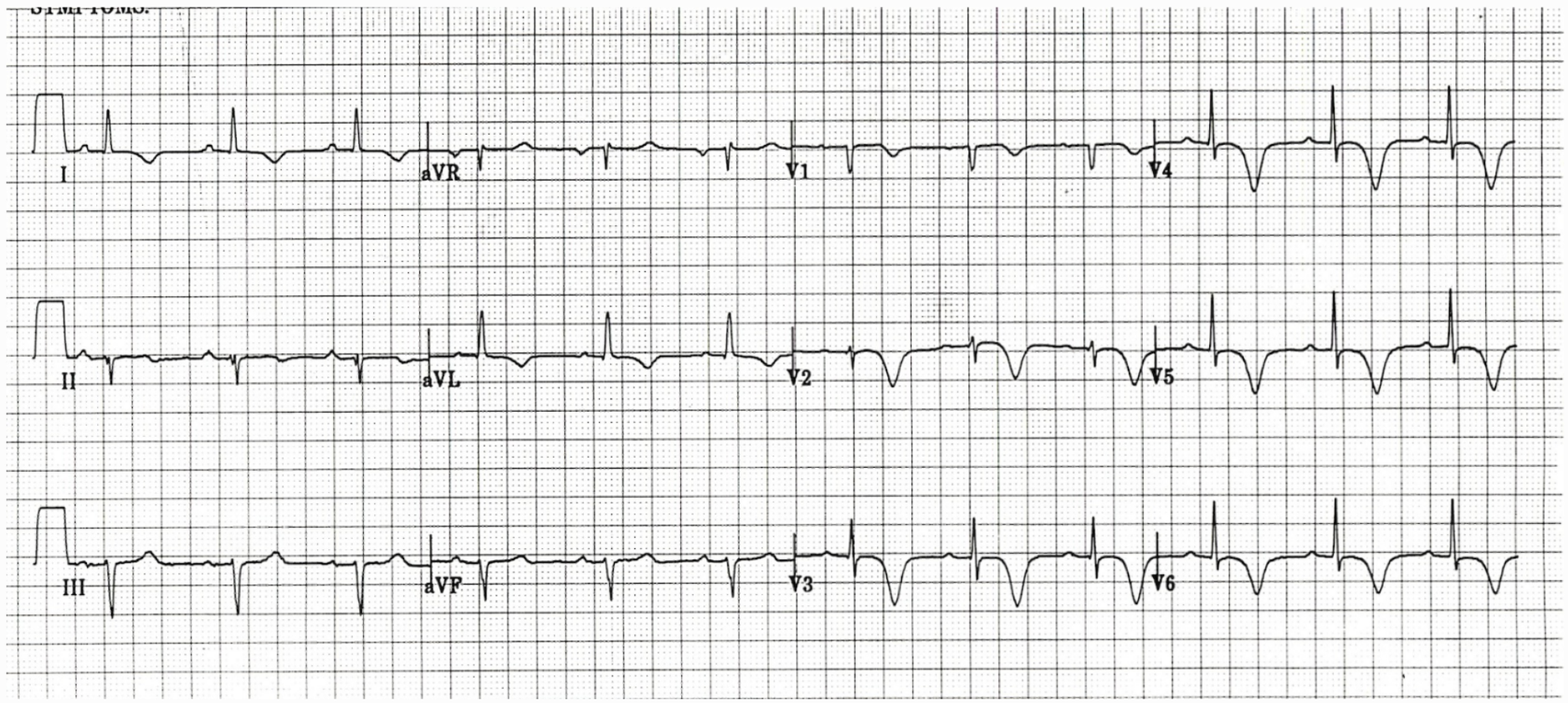
Romel Wrenn MD, FACC



Wellens Syndrome (Type A Pattern)

- Biphasic precordial T waves with terminal negativity, most prominent in V2-3.
- Minor precordial ST elevation.
- Preserved R wave progression (R wave in V3 > 3mm)

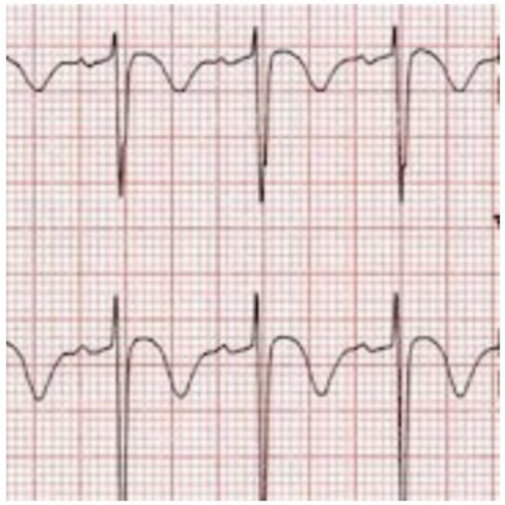

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6



Wellens Syndrome (Type B Pattern)

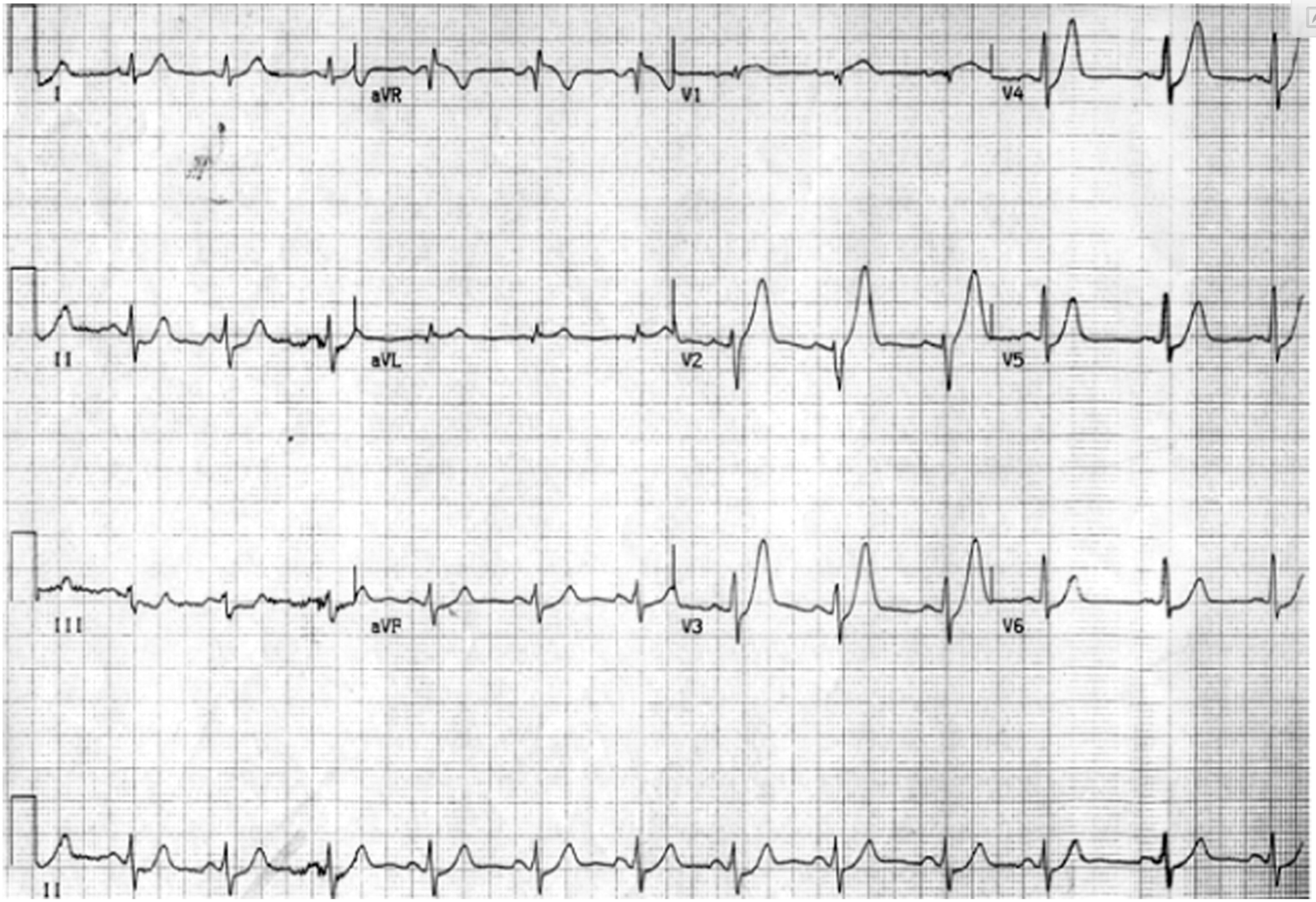
- There are deep, symmetrical T wave inversions throughout the anterolateral leads (V1-6, I, aVL).

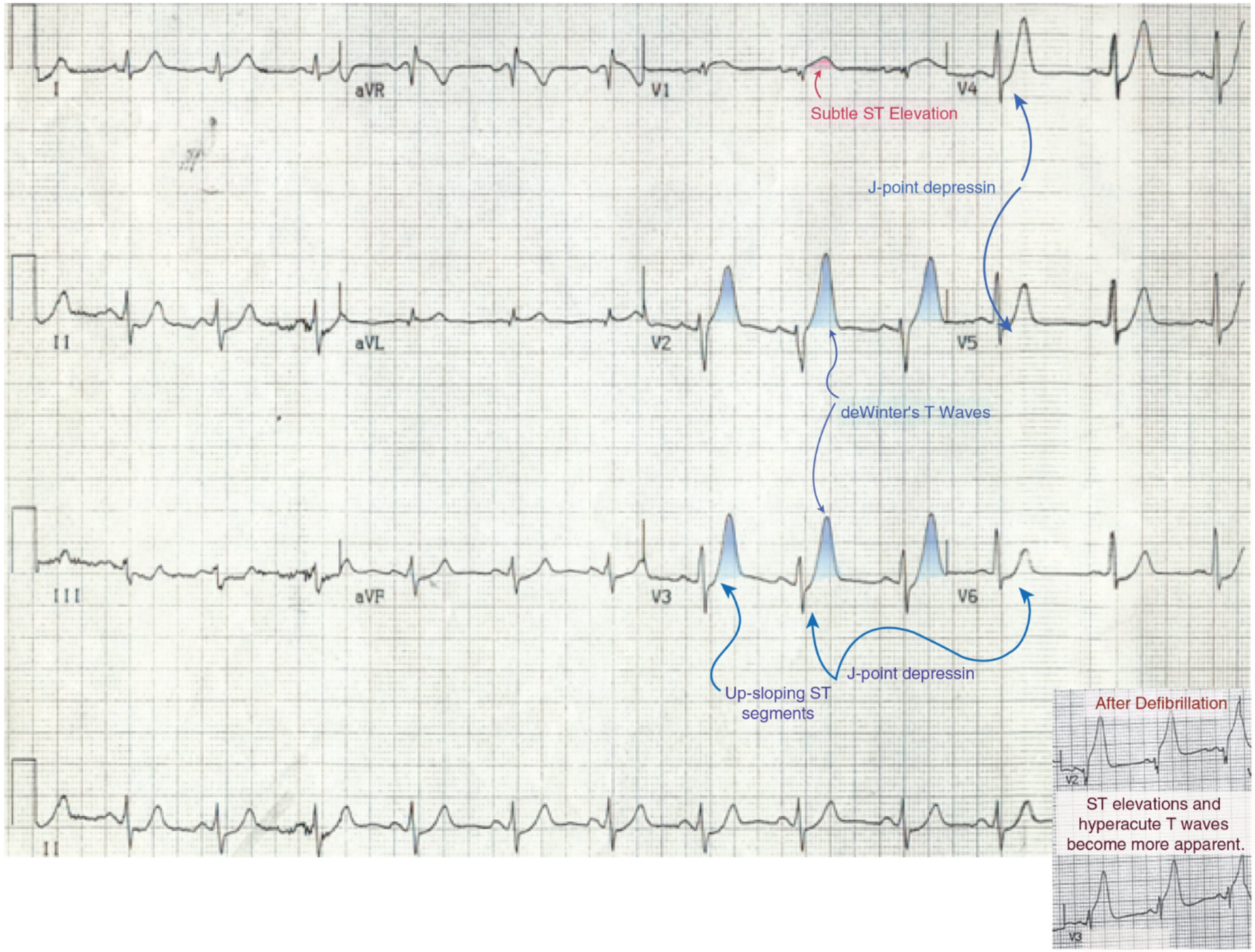
Syndrome	Pathology	ECG	Clinical Significance	Images / References

Syndrome	Pathology	ECG	Clinical Significance	Images / References
Wellens	Proximal critical stenosis of LAD artery	Symmetrical deeply inverted T waves in V2-3 or Biphasic in V2-3 with minimal ST elevation.Changes occur in pain free state and normalise when pain	Acute anterior MI if untreated	

Wellen's Syndrome

- Am Heart J 1982; 103:730-736
- Critical Stenosis in Proximal LAD
- 75% AMI Despite Medical Rx
- ACS Requiring Urgent Cardiac intervention despite negative biomarkers.




I
aVR
V1
V4
Subtle ST Elevation
J-point depressin
II
aVL
V2
V5
deWinter's T Waves
III
aVF
V3
V6
J-point depressin
Up-sloping ST segments
II
After Defibrillation
V2
ST elevations and hyperacute T waves become more apparent.
V3

The NEW ENGLAND JOURNAL of MEDICINE

CORRESPONDENCE

A New ECG Sign of Proximal LAD Occlusion

N Engl J Med 2008; 359:2071-2073 | November 6, 2008 | DOI: 10.1056/NEJMc0804737

To the Editor:

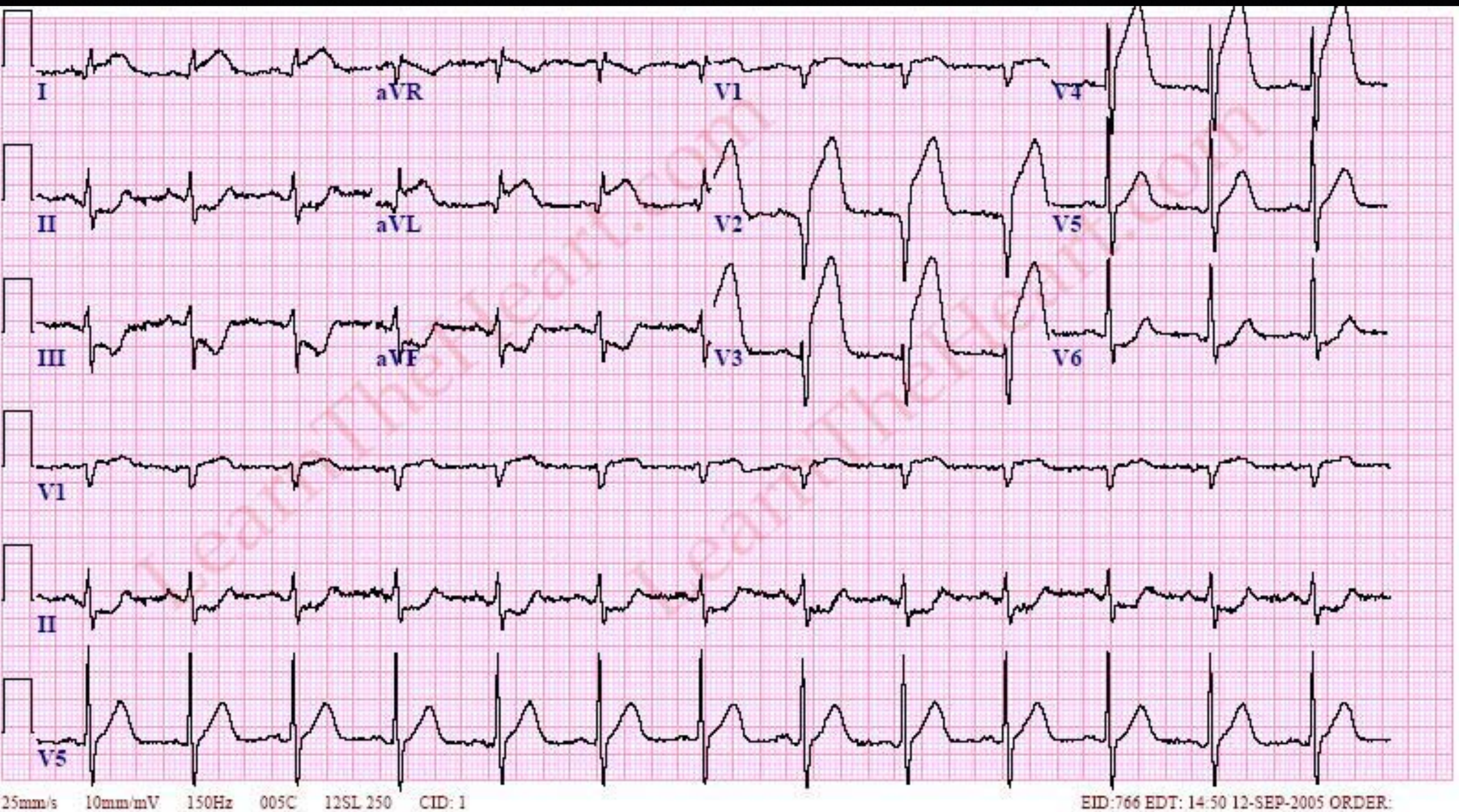
Recognition of characteristic changes in an electrocardiogram (ECG) that are associated with acute occlusion of a coronary artery guides decisions regarding immediate reperfusion therapy.[1-3] Working from our primary database of percutaneous coronary interventions, which includes records of the ambulance, or admission, ECG (performed on first medical contact with the patient), the preprocedural ECG, and the coronary angiogram, we describe a new ECG pattern without ST-segment elevation that signifies occlusion of the proximal left anterior descending coronary artery (LAD). Instead of the signature ST-segment elevation, the ST segment showed a 1- to 3-mm upsloping ST-segment depression at the J point in leads V_1 to V_6 that continued into tall, positive symmetrical T waves. The QRS complexes were usually not widened or were only slightly widened, and in some there was a loss of precordial R-wave progression. In most patients there was a 1- to 2-mm ST-elevation in lead aVR (see Figure 1 for representative examples of this ECG pattern). We recognized this characteristic ECG pattern in 30 of 1532 patients with anterior myocardial infarction (2.0%).

FIGURE 1

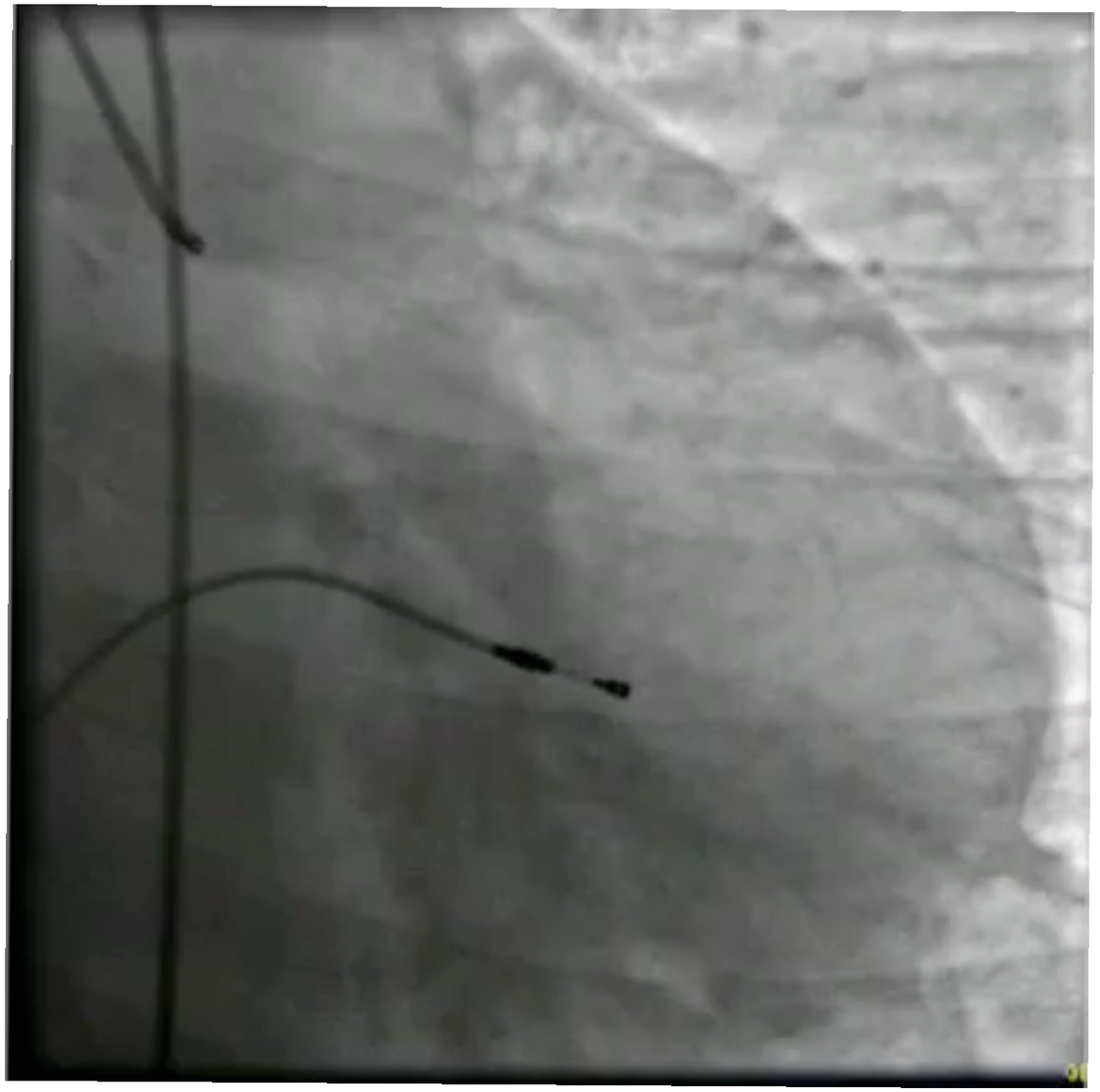
Unique Precordial ST-Segment and T-Wave Morphology.

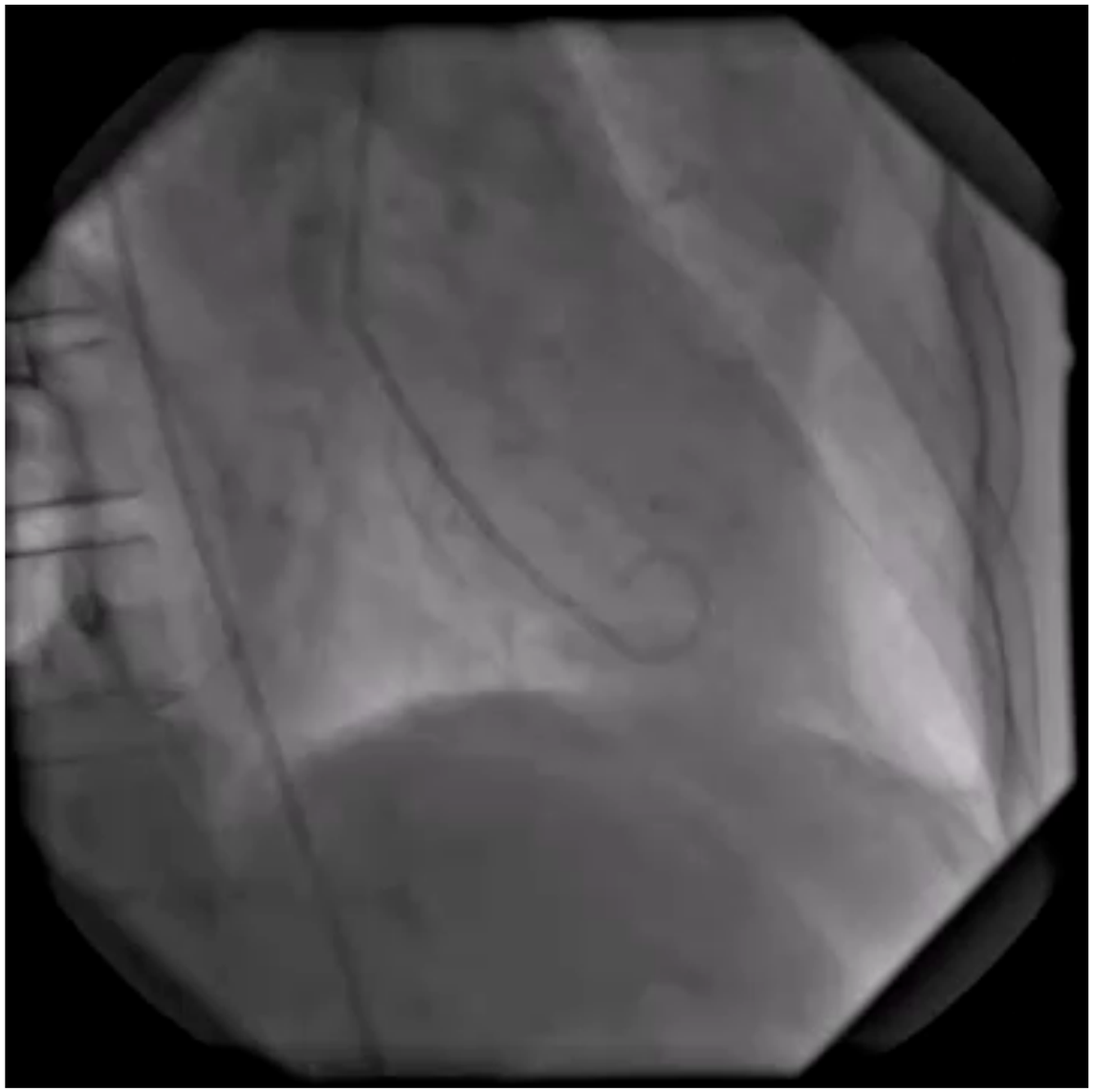
Although tall symmetrical T waves have been recognized as a transient early feature that changes into overt ST elevation in the precordial leads, in these patients this pattern was static, persisting from the time of first ECG until the preprocedural ECG was performed and angiographic evidence of an occluded LAD was obtained (i.e., 30 to 50 minutes). The ECGs with this pattern were on average recorded 1.5 hours after symptom onset. Collateral filling of the LAD ranged from Rentrop class 0 to class 3, and a wraparound LAD was present in 50% of patients. There was no evidence of involvement of the left main stem of the coronary artery, nor was there evidence of significant disease in the coronary arteries supplying the posterior or posterolateral myocardial territories. Potassium levels on admission were normal (3.9±0.5 mmol per liter). Despite successful procedures in all cases, there was considerable loss of myocardium, with a median creatine kinase MB peak of 342 μg per liter.

The electrophysiological explanation of the observed ECG pattern remains elusive. We could not establish patient characteristics, nor could we identify coronary angiographic characteristics that were unequivocally associated with the ECG pattern described as compared with a pattern of anterior ST elevation. Theoretically, an anatomical variant of the Purkinje fibers, with endocardial conduction delay, could be present. Alternatively, the absence of ST elevation may be related to the lack of activation of sarcolemmal ATP-sensitive potassium (K_{ATP}) channels by ischemic ATP depletion, as has been shown in K_{ATP} knockout animal models of acute ischemia.[4] It is of great importance for physicians and paramedics involved in the triage of patients with chest pain for reperfusion therapy to recognize this ECG pattern.

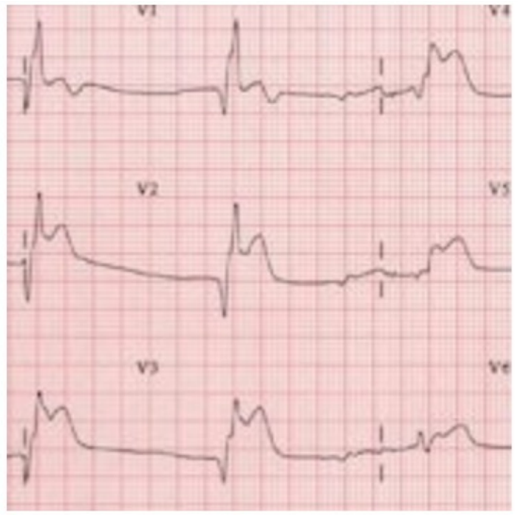

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 150Hz 005C 12SL 250 CID: 1
EID:766 EDT: 14:50 12-SEP-2005 ORDER:

TAKOTSUBO CARDIOMYOPATHY





- This entity was introduced as "tako-tsubo-like left ventricular dysfunction" in 1990 by Satoh.
- "Tako-tsubo" is a fishing pot with a round bottom and narrow neck, which is used for trapping octopuses in Japan.

Tako Tsubo	Cardiomyopathy with hypertrophic LV inferior and Hypotrophic superior wall (Octopus jar heart)	ECG changes as MI but usually brought on by stressful event	Normal angiogram but require cardiology follow up	

Stress Induced Cardiomyopathy

Sharkey SW, Lesser JR, Zenovich AG, et al. *Acute and reversible cardiomyopathy provoked by stress in women from the United States*. Circulation 2005;111:472–9.

- Women 95% cases
- Mean age 68
- Not just emotional stress
  - Intense physical stress
  - Acute medical illness (ICU)
  - No stress in 10%
- Probably 2% of ACS cases

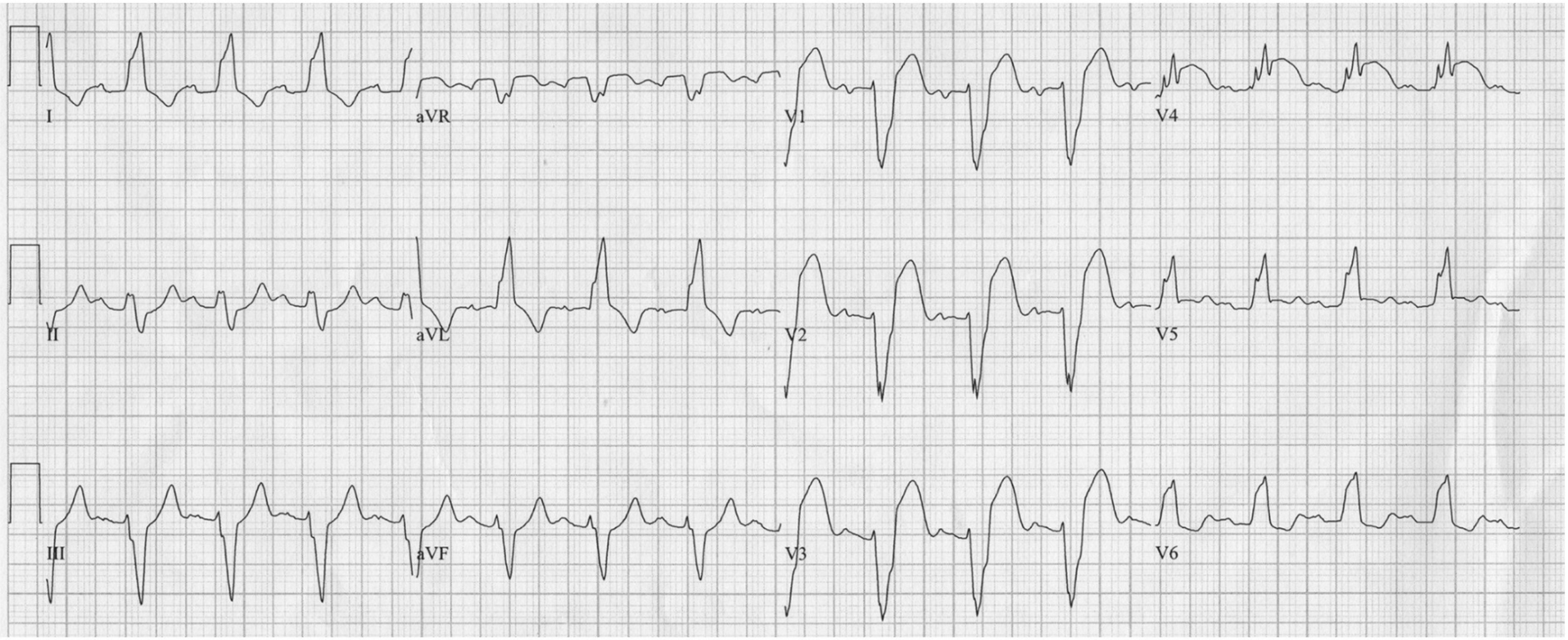
Etiology ???

- Wall motion abnormality doesn't correlate to single coronary distribution
- Catecholamine induced vascular spasm?
- Catecholamine induced reversible myocyte injury?
  - Is LV apex more sensitive to injury?

Stress Induced Cardiomyopathy

Diagnostic criteria:

- Transient akinesis or dyskinesis of apex-distal LV
- New ST elevation or T wave inversion
- No obstructive coronary artery disease
- Absence of other causes of transient myopathy
  - Head trauma
  - Intracranial bleed
  - Pheochromocytoma
  - Myocarditis


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

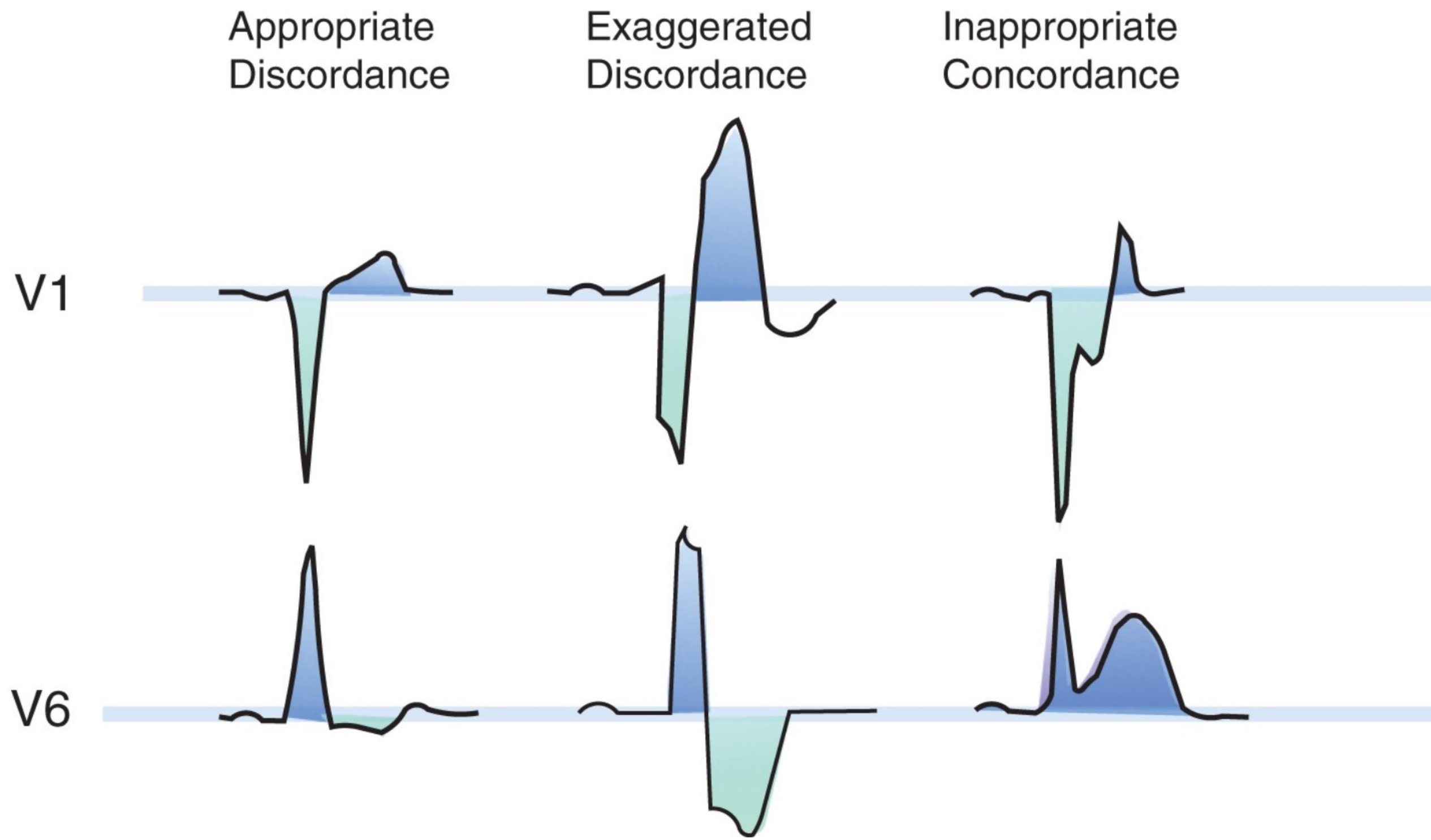
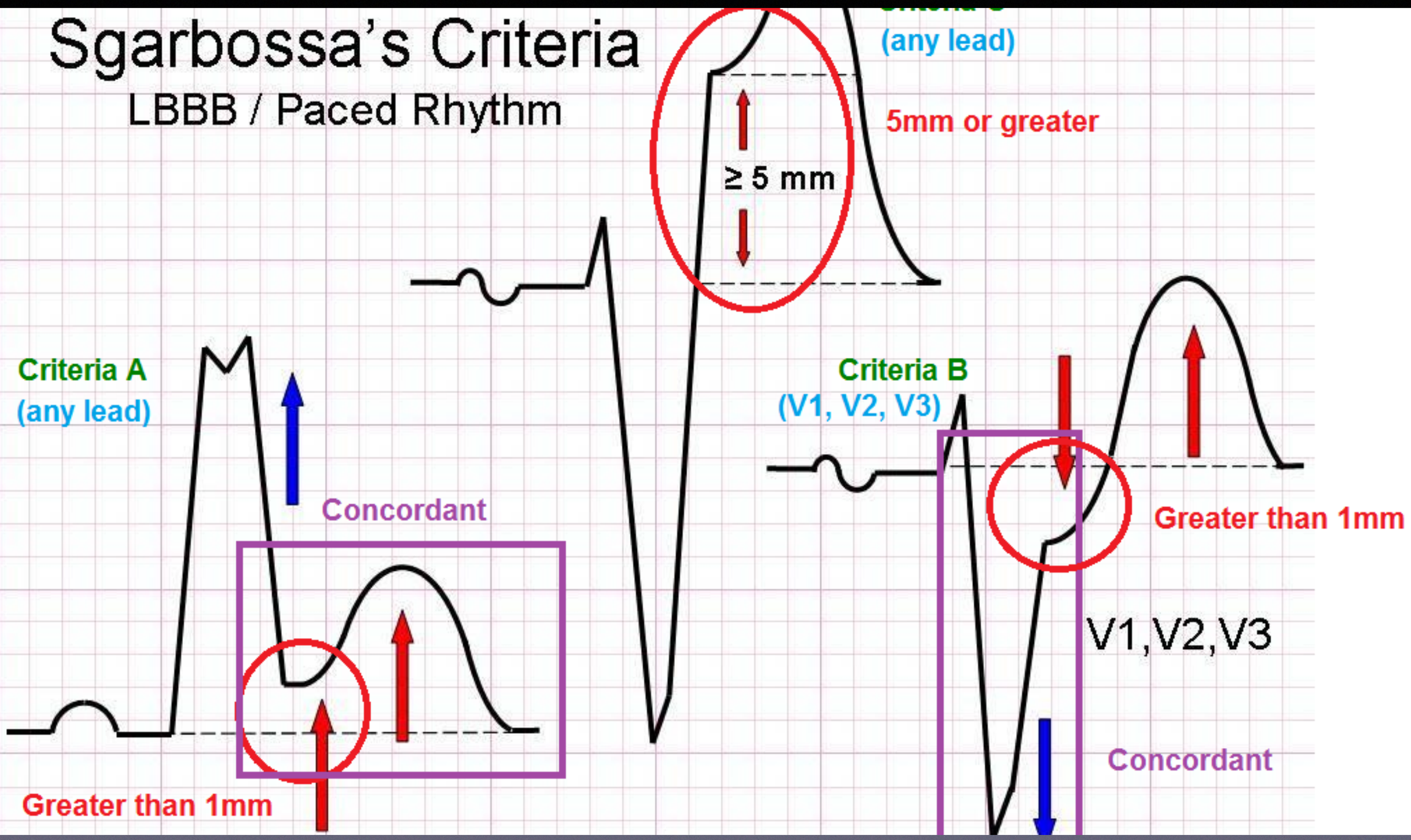


Figure 12.24 Appearance of discordance and concordance in left bundle branch block.

Sgarbossa's Criteria

LBBB / Paced Rhythm



131patients withLBBB/26003 STEMI Patients December 27, 1990, and was completed on February 22, 1993.

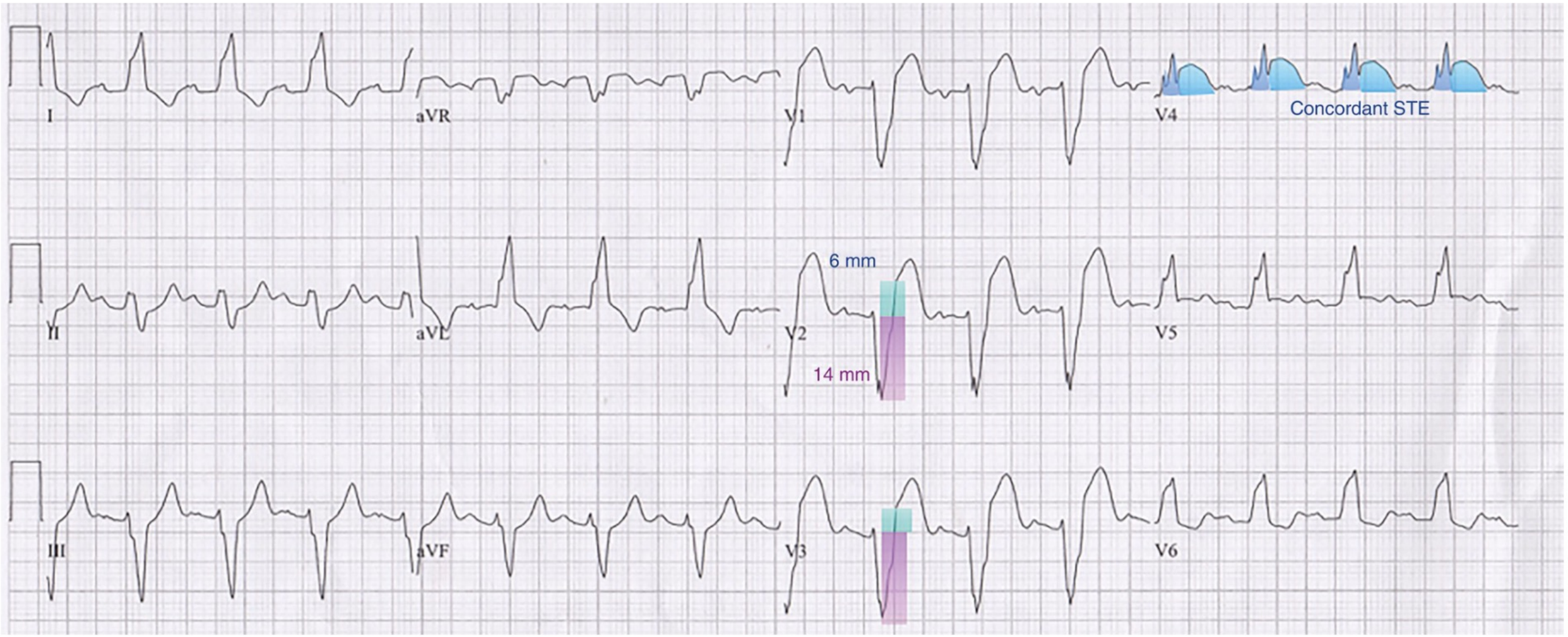
- Sgarbossa, Elena B.; Pinski, Sergio L.; Barbagelata, Alejandro; Underwood, Donald A.; Gates, Kathy B.; Topol, Eric J.; Califf, Robert M.; Wagner, Galen S. (1996). "Electrocardiographic **Diagnosis of Evolving Acute Myocardial Infarction in the Presence of Left Bundle-Branch Block"**. New England Journal of Medicine. 334 (8): 481–487.

Sgarbossa's criteria

Three criteria are included in Sgarbossa's criteria:[2]

- ST elevation ≥1 mm in a lead with a positive QRS complex (ie: concordance) - 5 points
- ST depression ≥1 mm in lead V1, V2, or V3 - 3 points
- ST elevation ≥5 mm in a lead with a negative (discordant) QRS complex - 2 points

≥3 points = 90% specificity of STEMI (sensitivity of 36%)[2]


I
aVR
V1
V4
Concordant STE
II
aVL
V2
6 mm
14 mm
V5
III
aVF
V3
V6

	Sgarbossa Criteria	Smith-Modified Sgarbossa Criteria (Unweighted)
Inappropriate Concordance	Concordant ST elevation ≥ 1 mm in any lead (5 points) Concordant ST depression ≥ 1 mm in leads V1-V3 (3 points)	Concordant ST elevation ≥ 1 mm in any lead Concordant ST depression ≥ 1 mm in leads V1-V3
Exaggerated Discordance	Absolute Discordance: Discordant STE ≥ 5 mm in any lead (2 points)	Discordance out of proportion: $\frac{\text{ST-segment elevation}}{\text{Depth of S wave}} > 0.25$

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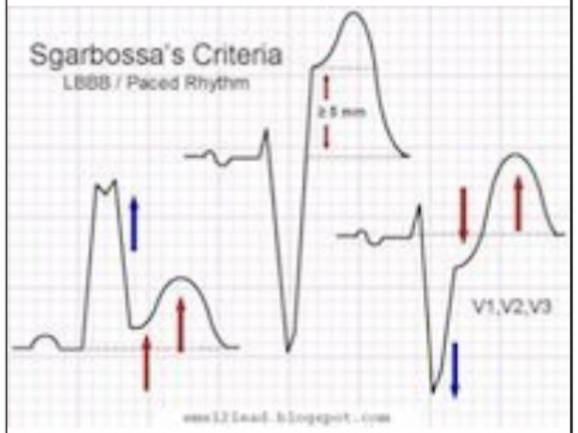
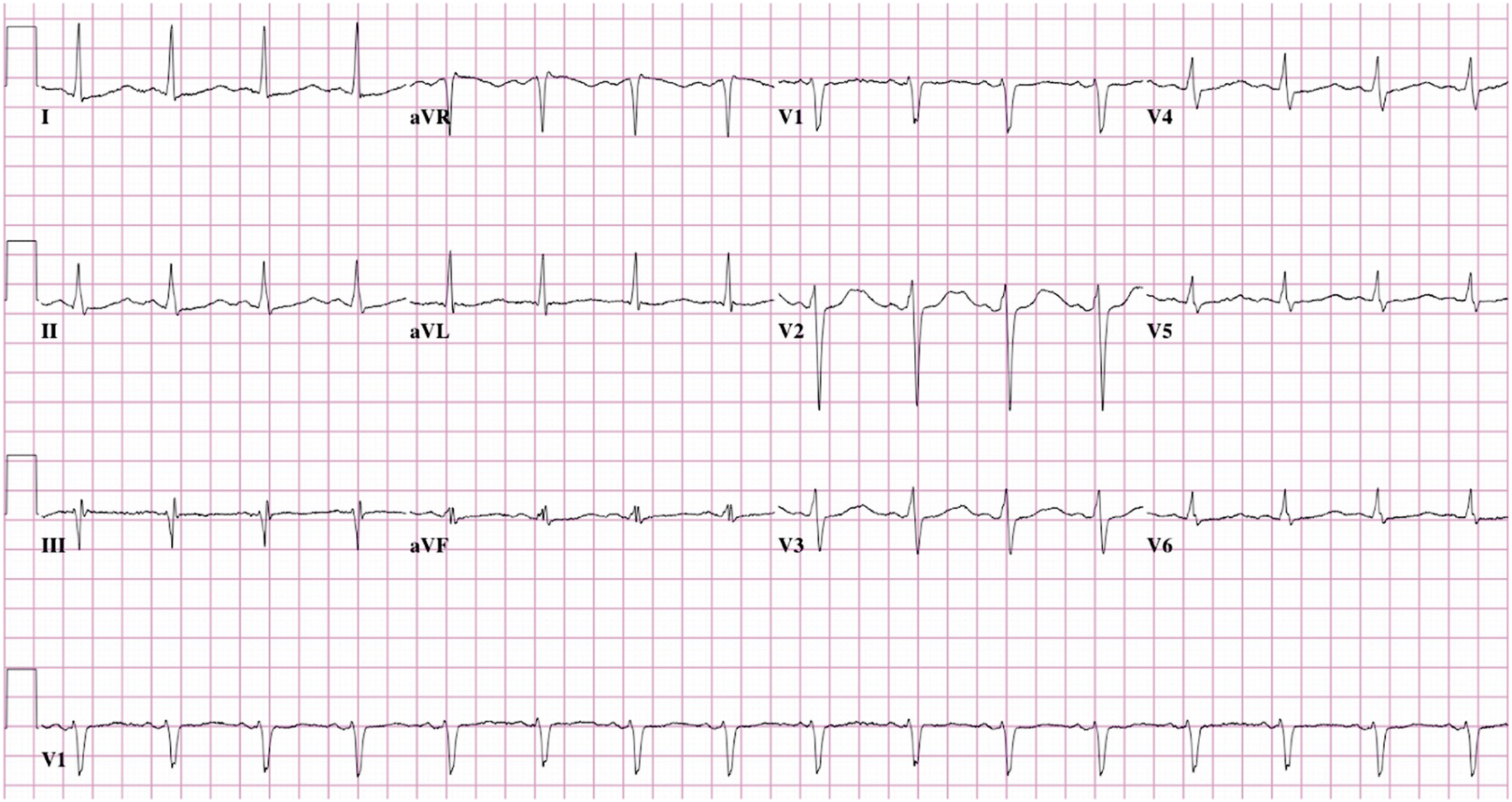
Sgarbossa criteria	Derived from GUSTO 1 trial to identify MI in paced or LBBB patients	ST elevation >1mm concordant with QRS complex (5pts), ST depression >1mm in V1-3 (3pts), ST elevation >5mm discordant with QRS 2 points. >3 points consistent with MI	Help risk stratify patients with chronic LBBB presenting with ACS symptoms	



ECG 8.18A

LONG QT INTERVAL

Causes:

- Myocardial infarction, myocarditis, diffuse myocardial disease
- Hypocalcemia, hypothyrodism
- Subarachnoid hemorrhage, intracerebral hemorrhage
- Drugs (e.g. Sotalol, Amiodarone)
- Heredity

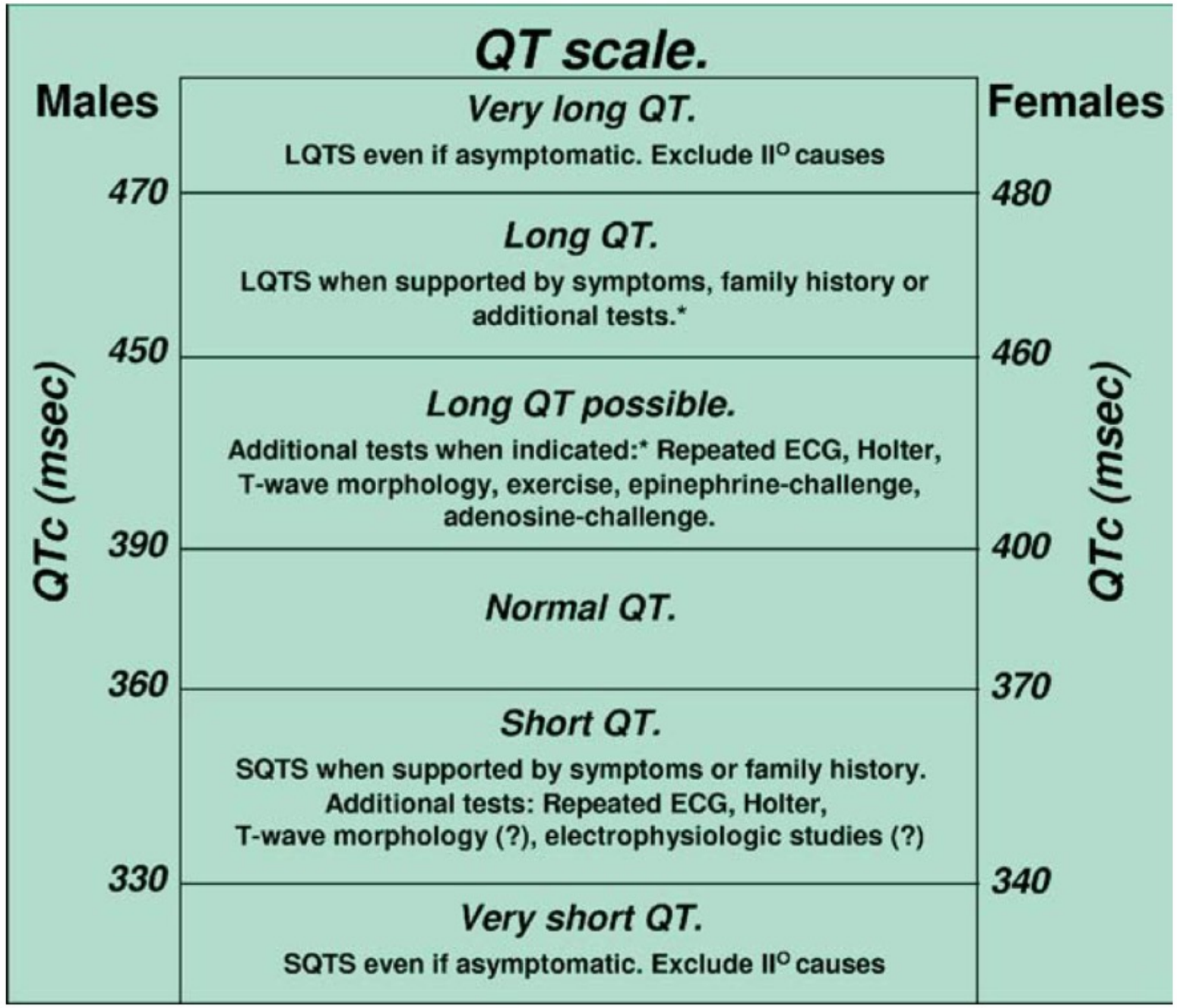
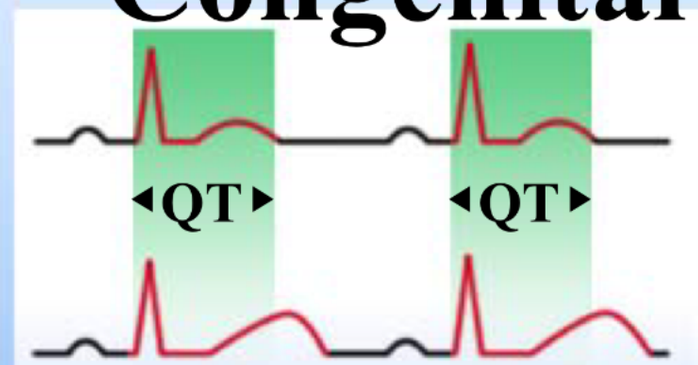


Figure 3 Proposed "QT scale" for defining the spectrum of QT intervals, ranging from very short to very long. See text for definitions and additional tests. This is a modification of the algorithm proposed by Vincent[41] for long QT syndrome (LQTS). SQTS = short QT syndrome.

12. Viskin S. The QT interval: Too long, too short or just right. Heart Rhythm 2009; 6: 711–715.

Congenital Long QT Syndrome



Normal QT interval

Prolonged QT

1. Syncope
2. Seizures
3. Sudden death

Torsades de pointes

LQTS Genetic Testing

LQT1
Ch 11p15.5
KCNQ1
30-35%

LQT2
Ch 7q35-36
KCNH2
25-30%

LQT3
Ch 3p21-24
SCN5A
5-10%

MAYO CLINIC

SCN4B-LQT
(**LQT10**)
Ch 11q23.3
NaV1.5 β4
< 1%

AKAP9-LQT
(**LQT11**)
IKs ChIP
< 1%

SNTA1-LQT
(**LQT12**)
NaV1.5 ChIP
< 1%

KCNJ5-LQT
(**LQT13**)
Kir3.4
< 1%

LQT1
Ch 11p15.5
KCNQ1
30-35%

CALM1-LQT
(**LQT14**)
CaV1.2 ChIP
< 1%

CALM2-LQT
(**LQT15**)
CaV1.2 ChIP
< 1%

LQT2
Ch 7q35-36
KCNH2
25-30%

CAV3-LQT
(**LQT9**)
Ch 3p25
Caveolin-3
< 1%

CALM3-LQT
(**LQT16**)
CaV1.2 ChIP
< 1%

TRDN-LQT
(**LQT17**)
< 1%

LQT3
Ch 3p21-24
SCN5A
5-10%

TS1
(**LQT8**)
Ch 1q42
CACNA1C
<<1%

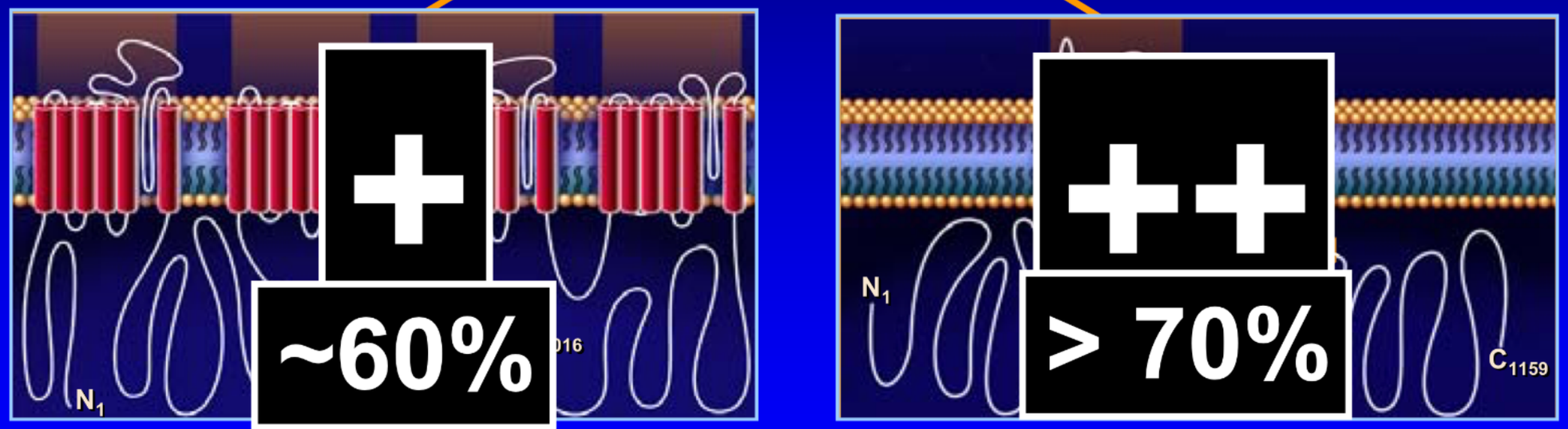
ATS1
(**LQT7**)
Ch 17q23
KCNJ2
<1%a

LQT6
Ch 21q22
KCNE2
<1%

LQT5
Ch 21q22
KCNE1
1%

LQT4
Ch 4q25-27
ANK2
< 1%

Efficacy of Beta Blocker Therapy



Moss et al. *Circulation* 101:616-623, 2000
Villain et al. *European Heart Journal* 25:1405-1411, 2004
Wilde … Ackerman. *Circulation* 2016 (LQT3)
Ackerman, Priori, Schwartz, Wilde. Personal LQTS Clinics, 2017

Diagnostic, Prognostic, and Therapeutic Implications of Genetic Testing for LQTS

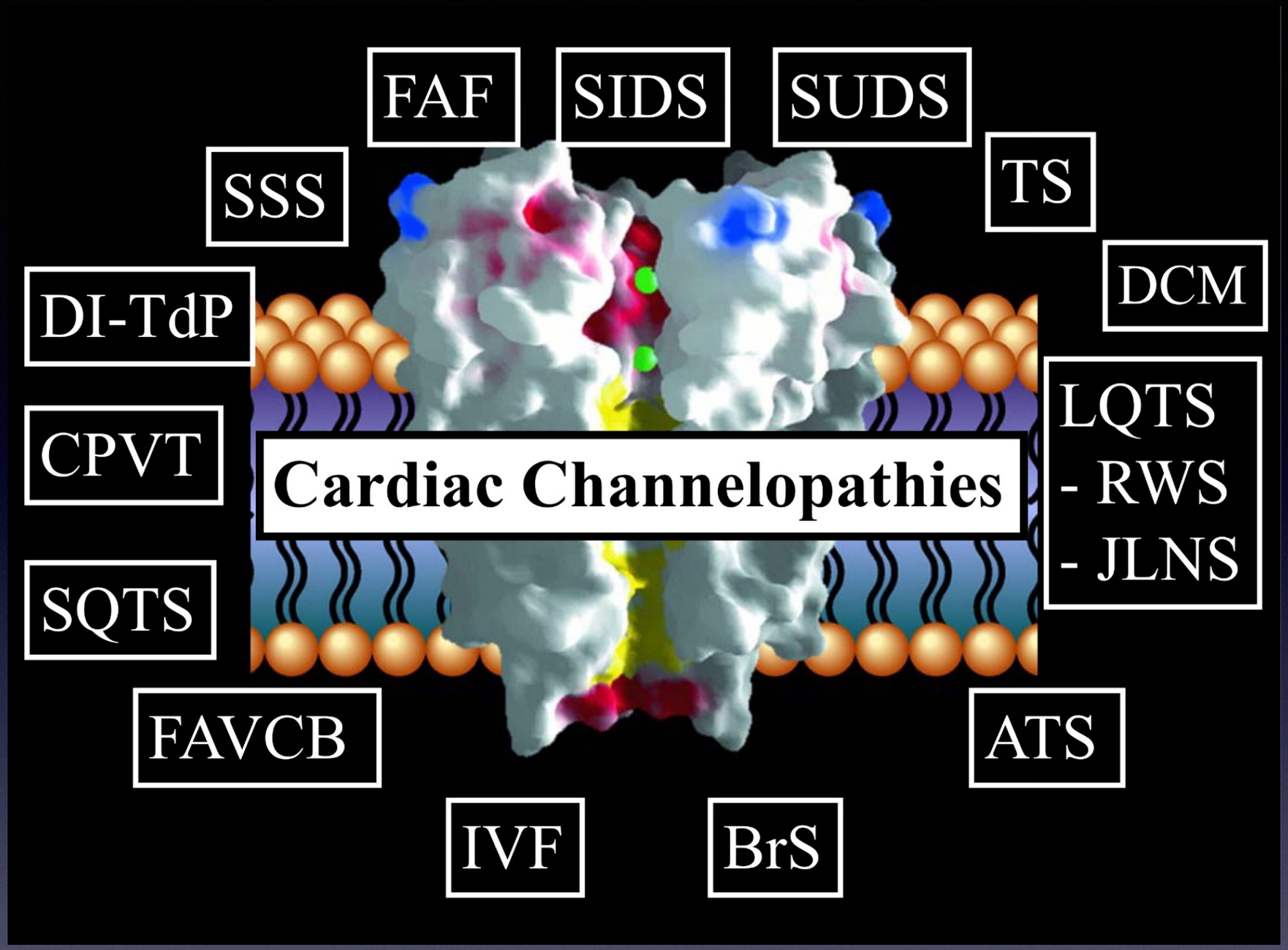
Disease	Diagnostic	Prognostic	Therapeutic
LQTS	+++	+++	++
CPVT	+++	+	-
BrS	+	+	-
CCD	+	+	+
SQTS	+/-	-	-
AF	-	-	-
HCM	+++	++	+
ARVC	+	+/-	-
DCM	+/-	-	-
LVNC	+	-	-
RCM	+	+	+

Ackerman, Priori, et al. *Heart Rhythm* 8:1308-1339, 2011

Indications for ICD Therapy in LQTS

Primary Prevention

- QTc > 550 ms and not LQT1
- LQT2 women, QTc > 500 ms, +/- Sx
- Infants with 2:1 AV block?
- JLNS (LQTS w/ deafness)?



Familial AV Conduction Block

Drug-Induced Torsades

Andersen Tawil Syndrome

Timothy Syndrome , LQT8

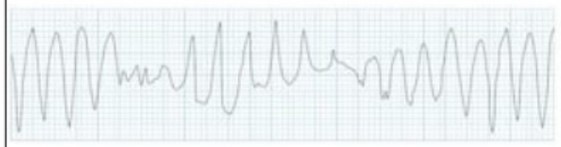
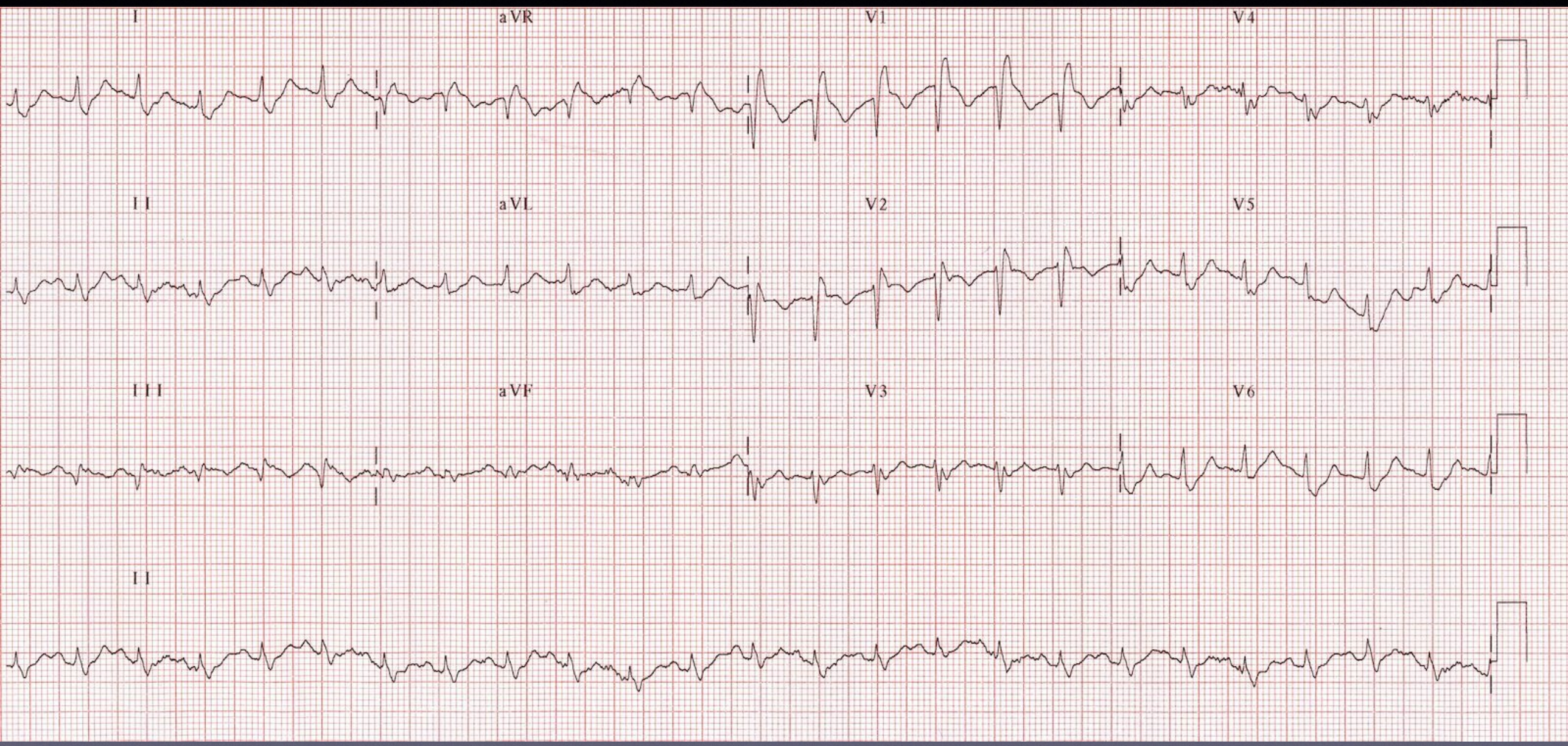
Syndrome	Pathology	ECG	Clinical Significance	Images / References
Romano - Ward	Inherited Long QT syndrome (Autosomal dominant)Defect of Na and K channels.**Not** associated with deafness	Long QT, T wave alternans, notched T wave, R on T phenomenon, Torsades	Risk of Torsades. Electrolyte optimisation, may require ICD	

Table 2. Cardiac channelopathies

Disease	Channel protein	Gene
Atrial standstill	Nav1.5: sodium channel, voltage-gated, type V, α subunit	*SCN5A*
Brugada syndrome type 1	Nav1.5: sodium channel, voltage-gated, type V, α subunit	*SCN5A*
Brugada syndrome type 3 (short QT syndrome type 4)	Cav1.2: calcium channel, voltage-gated, L type, α1C subunit	*CACNA1C*
Brugada syndrome type 4 (short QT syndrome type 5)	Cavβ2: calcium channel, voltage-gated, β2 subunit	*CACNB2*
Brugada syndrome type 5	Navβ1: sodium channel, voltage-gated, type I, β subunit	*SCN1B*
Brugada syndrome type 6	Potassium channel, voltage-gated, Isk-related subfamily, member 3	*KCNE3*
Brugada syndrome type 7	Navβ3: sodium channel, voltage-gated, type III, β subunit	*SCN3B*
Brugada syndrome type 8	Hyperpolarization-activated cyclic nucleotide-gated potassium channel 4	*HCN4*
Catecholaminergic polymorphic ventricular tachycardia type 1	RyR2: ryanodine receptor 2	*RYR2*
Dilated cardiomyopathy type 1E	Nav1.5: sodium channel, voltage-gated, type V, α subunit	*SCN5A*
Dilated cardiomyopathy type 10	ATP-binding cassette, subfamily C, member 9 (sulfonylurea receptor 2)	*ABCC9*
Familial arrhythmogenic right ventricular dysplasia type 2	RyR2: ryanodine receptor 2	*RYR2*
Familial atrial fibrillation type 3	Kv7.1: potassium channel, voltage-gated, KQT-like subfamily, member 1	*KCNQ1*
Familial atrial fibrillation type 4	Potassium channel, voltage-gated, Isk-related subfamily, member 2	*KCNE2*
Familial atrial fibrillation type 7	Kv1.5: potassium channel, voltage-gated, shaker-related subfamily, member 5	*KCNA5*
Familial atrial fibrillation type 9	Kir2.1: potassium channel, inwardly-rectifying, subfamily J, member 2	*KCNJ2*
Familial atrial fibrillation type 10	Nav1.5: sodium channel, voltage-gated, type V, α subunit	*SCN5A*
Familial atrial fibrillation type 12	ATP-binding cassette, subfamily C, member 9	*ABCC9*
Jervell and Lange-Nielsen syndrome type 1	Kv7.1: potassium channel, voltage-gated, KQT-like subfamily, member 1	*KCNQ1*
Jervell and Lange-Nielsen syndrome type 2	Potassium channel, voltage-gated, Isk-related subfamily, member 1	*KCNE1*
Long QT syndrome type 1	Kv7.1: potassium channel, voltage-gated, KQT-like subfamily, member 1	*KCNQ1*
Long QT syndrome type 2	Kv11.1: potassium channel, voltage-gated, subfamily H, member 2	*KCNH2*
Long QT syndrome type 3	Nav1.5: sodium channel, voltage-gated, type V, α subunit	*SCN5A*
Long QT syndrome type 5	Potassium channel, voltage-gated, Isk-related subfamily, member 1	*KCNE1*
Long QT syndrome type 6	Potassium channel, voltage-gated, Isk-related subfamily, member 2	*KCNE2*
Long QT syndrome type 7 (Andersen-Tawil syndrome)	Kir2.1: potassium channel, inwardly-rectifying, subfamily J, member 2	*KCNJ2*
Long QT syndrome type 8 (Timothy syndrome)	Cav1.2: calcium channel, voltage-gated, L type, α1C subunit	*CACNA1C*
Long QT syndrome type 10	Navβ4: sodium channel, voltage-gated, type IV, β subunit	*SCN4B*
Long QT syndrome type 13	Kir3.4: potassium channel, inwardly-rectifying, subfamily J, member 5	*KCNJ5*
Nonprogressive familial heart block	Nav1.5: sodium channel, voltage-gated, type V, α subunit	*SCN5A*
Paroxysmal familial ventricular fibrillation, type 1	Nav1.5: sodium channel, voltage-gated, type V, α subunit	*SCN5A*
Pogressive familial heart block type IA (Lenegre-Lev syndrome)	Nav1.5: sodium channel, voltage-gated, type V, α subunit	*SCN5A*
Pogressive familial heart block type IB	Transient receptor potential cation channel, subfamily M, member 4	*TRPM4*
Short QT syndrome type 1	Kv11.1: potassium channel, voltage-gated, subfamily H, member 2	*KCNH2*
Short QT syndrome type 2	Kv7.1: potassium channel, voltage-gated, KQT-like subfamily, member 1	*KCNQ1*
Short QT syndrome type 3	Kir2.1: potassium channel, inwardly-rectifying, subfamily J, member 2	*KCNJ2*
Short QT syndrome type 4 (Brugada syndrome type 3)	Cav1.2: calcium channel, voltage-gated, L type, α1C subunit	*CACNA1C*
Short QT syndrome type 5 (Brugada syndrome type 4)	Cavβ2: calcium channel, voltage-gated, β2 subunit	*CACNB2*
Short QT syndrome type 6	Cavα2δ1: calcium channel, voltage-gated, α2/δ1 subunit	*CACNA2D1*
Sick sinus syndrome type 1, autosomal-recessive	Nav1.5: sodium channel, voltage-gated, type V, α subunit	*SCN5A*
Sick sinus syndrome type 2, autosomal-dominant	Hyperpolarization-activated cyclic nucleotide-gated potassium channel 4	*HCN4*

Alternative names are in parentheses.



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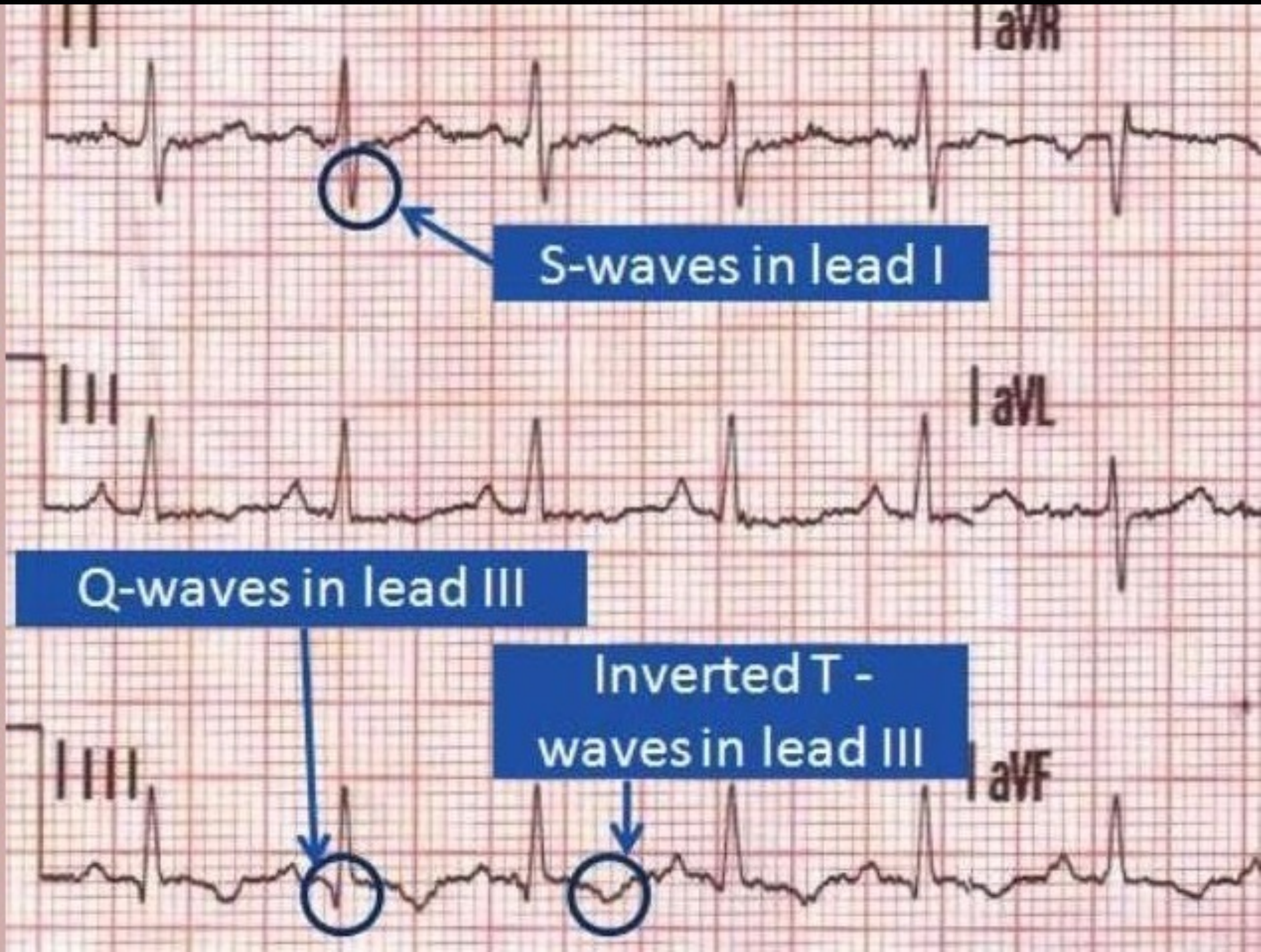
ECG S1Q3T3 Classic Pattern



ST Elevation V1

ST elevations in right-sided leads are more likely to be found when pulmonary embolism is massive and associated with cardiogenic shock.[9]

ST Elevation aVR

Changes in Rhythm

Sinus Tachycardia

Sinus tachycardia is associated with acute pulmonary embolism but is only 28%–38% sensitive.[7,10] The positive likelihood ratio for tachycardia is 1.8 (1.5–2.2).[7]

Atrial Fibrillation

Signs of RV Strain

Right Ventricular Conduction Delay

An R′ may be present in lead V1. Both incomplete and complete right bundle branch blocks are insensitive findings (10%–12% prevalence).[10]

Right Axis Deviation

This may occur when there is severe right heart strain.

S1Q3T3

S and Q waves (1.5-mm deep) associated with a T-wave inversion in lead III is another insensitive finding (found in 8.5% of patients with PE).[7]

ECG Abnormalities Associated with Pulmonary Embolism

p. 171

The electrocardiogram is one of the first diagnostic tests performed in patients suspected of having pulmonary embolism. Many of the classical findings taught to occur in association with acute pulmonary embolism are insensitive. Some depolarization and repolarization abnormalities, however, are specific to pulmonary embolism and predictive of poor prognosis.

T-Wave Inversions

Patients with severe PE often have inverted T waves. T-wave inversions are prognostic of poor outcome.

Anterior Precordial Leads

T-wave inversion in V1 most common.

T-wave inversion in V1-V4 is 98% specific for PE (positive LR 3.7 [2.4–6.1]).[7]

T-wave inversions will be deepest in leads V1-V2 in patients with acute pulmonary embolism and in leads V3-V4 in patients with acute coronary syndrome.[8]

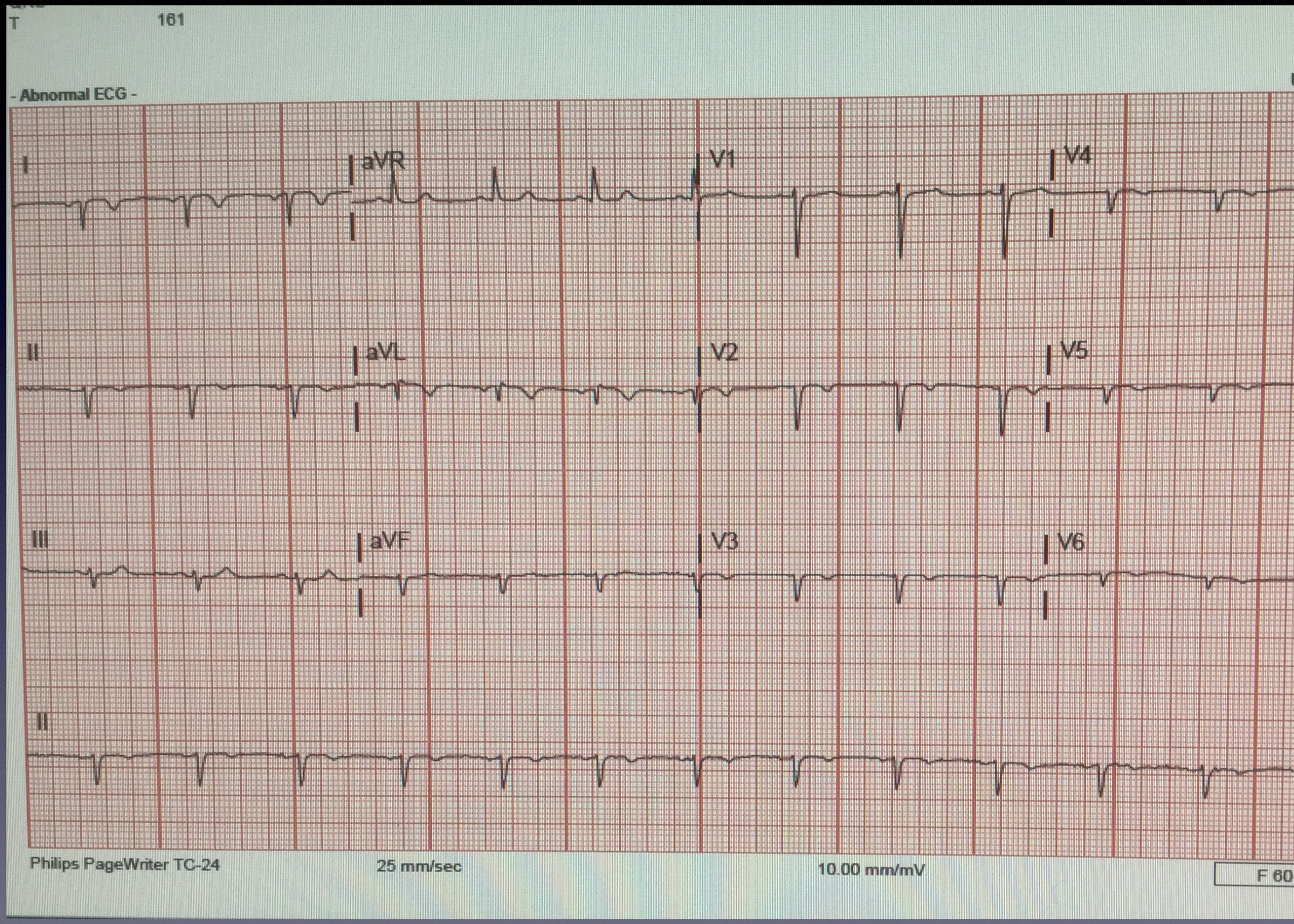
Lead III/Inferior Leads

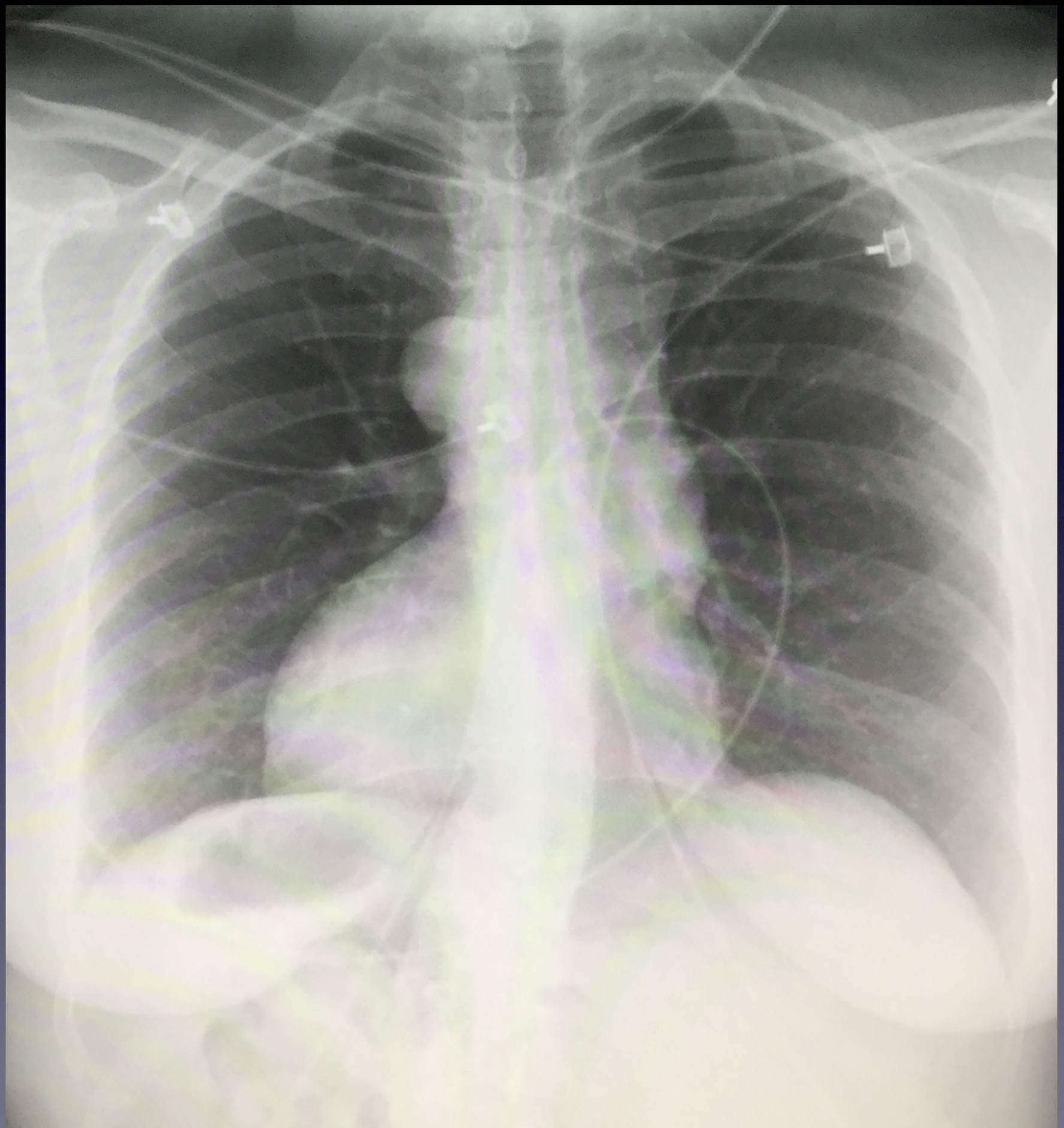
The presence of negative T waves in lead III in conjunction with T-wave inversion in V1 has been shown to be more specific for acute pulmonary embolism, differentiating it from acute coronary syndrome.[8]

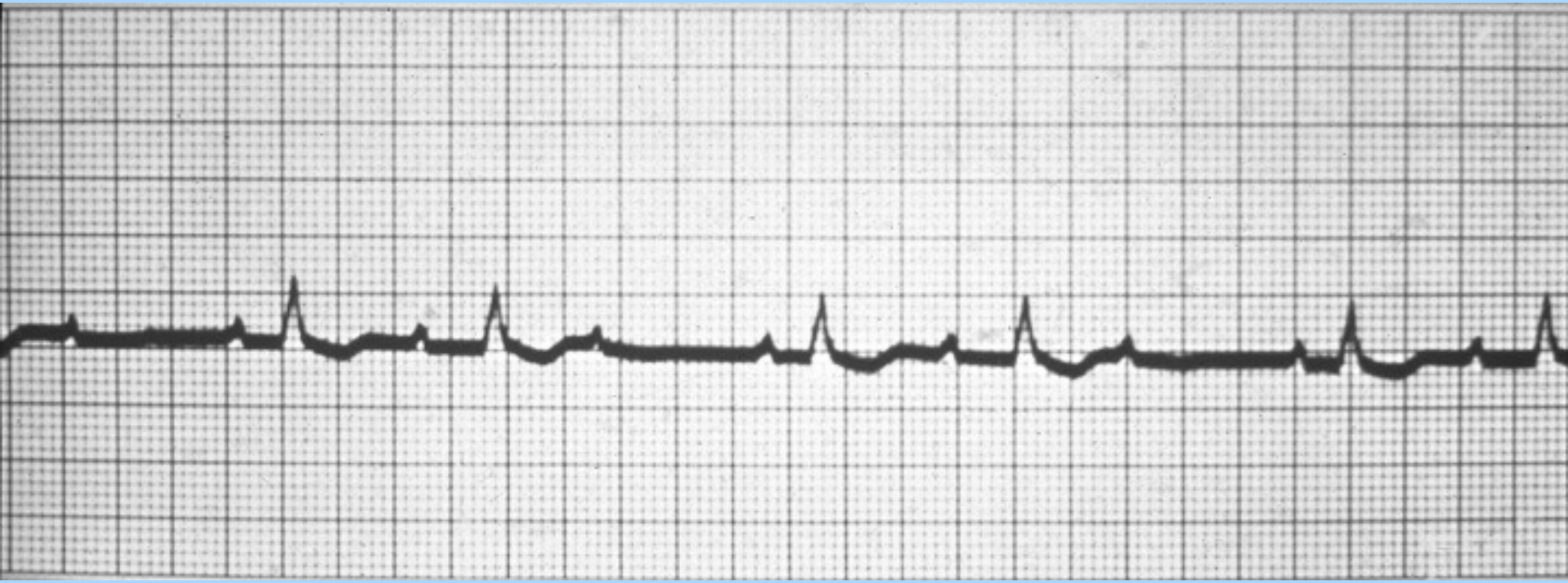
ST Elevation V1

ST elevations in right-sided leads are more likely to be found when pulmonary embolism is massive and associated with cardiogenic shock.[9]

ST Elevation aVR


T
161
- Abnormal ECG -
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
Philips PageWriter TC-24
25 mm/sec
10.00 mm/mV
F 60



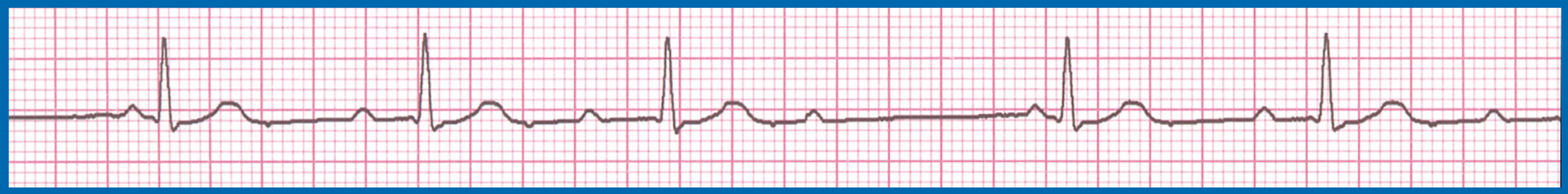


Second Degree A-V Block – Mobitz Type I – Wenckebach AV Block

2nd degree heart block

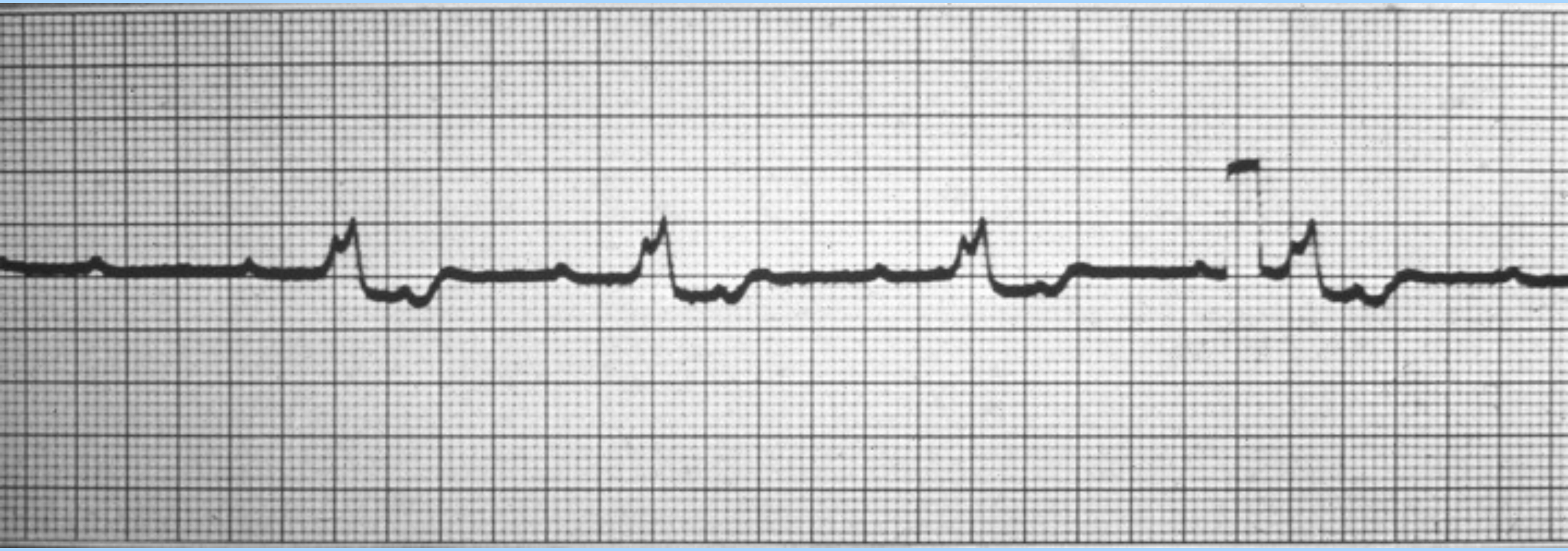
- Divided in to two types
- Type 1 - Wenkeback (Mobitz type I)
- Type 2 - Mobitz type II

2nd Degree AV Block, Type I

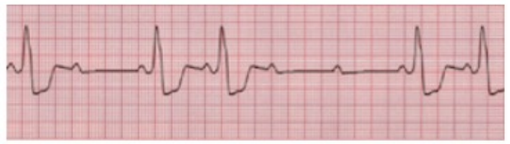


- PR interval progressively lengthens
- One non conducted beat- (P wave not followed by QRS).
- Following beat has shorter PR interval

Etiology: Each successive atrial impulse encounters a longer and longer delay in the AV node until one impulse (usually the 3rd or 4th) fails to make it through the AV node.



Second Degree A-V Block – Mobitz Type II

Mobitz II	A-V blockPR of constant interval	Constant PR interval until P waved dropped with P-P interval twice normal	Likely to progress to CHB	
Wenckebach	A-V blockPR of increasing interval	Lengthening PR interval until P waved dropped with P-P interval variable. Grouped beats	Need follow up but less likely to progress to CHB