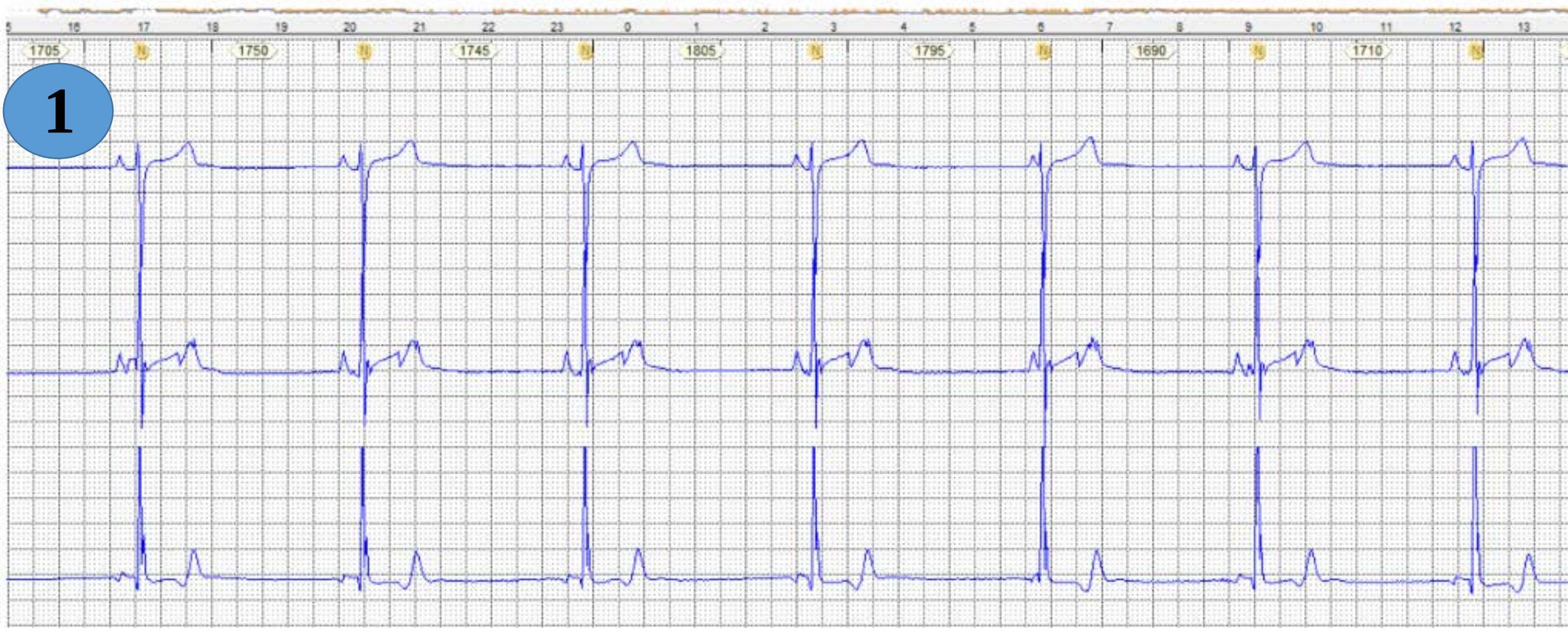
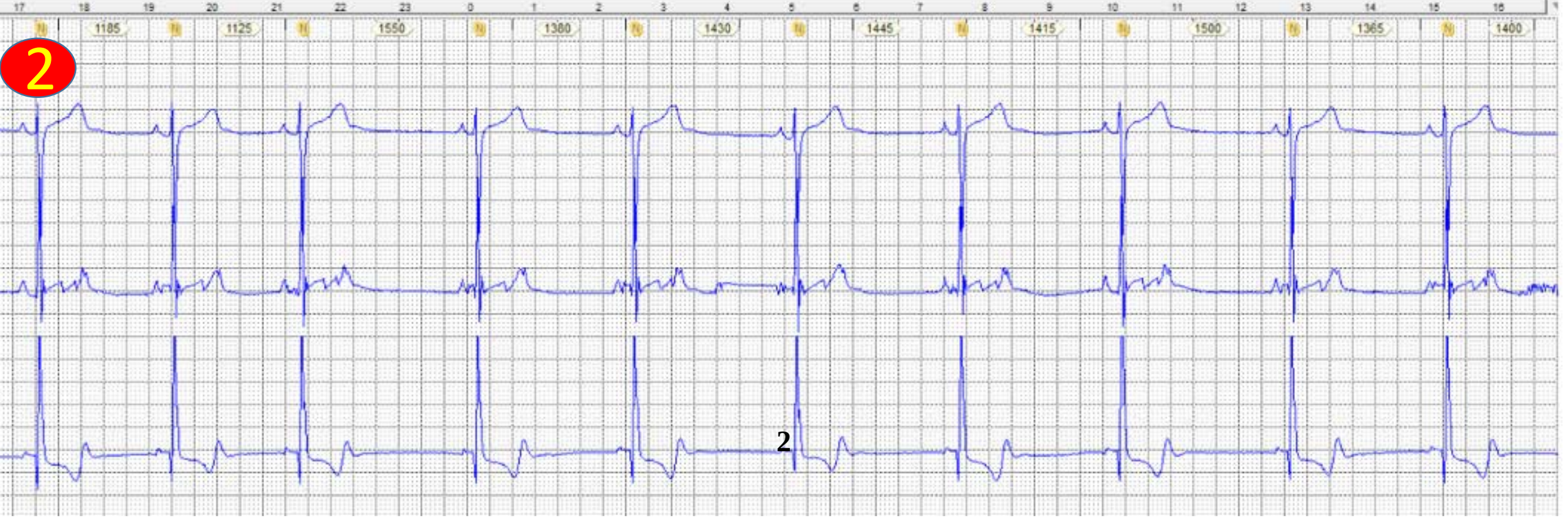
Estimado Andrés,
Gostaria de ouvir as opiniões sobre os traçados do Holter anexo, Se trata de um jovem de 27 anos praticante de triatlo (atleta de alta performance). Sua FC no consultório era 36 bpm e a FC mínima dormindo foi 27 bpm.
Agradeço antecipadamente,
Abraço
Dário C. Sobral Filho
Professor Associado e Livre-Docente de Cardiologia da Universidade de Pernambuco (UPE)
Coordenador de Pós-Graduação e Pesquisa da Faculdade de Ciências Médicas - UPE

Dear Andrés, I would like to hear opinions about the Holter tracings attached, This is a 27-year-old young man who practices triathlons (high-performance athlete). His office HR was 36 bpm and his minimum HR sleeping was 27 bpm.
Thank you in advance,
Hug
Dário C. Sobral Filho Associate Professor and Associate Professor of Cardiology at the University of Pernambuco (UPE)Graduate and Research Coordinator at the Faculty of Medical Sciences – UPE

Estimado Andrés, Me gustaría escuchar opiniones sobre los trazados Holter adjuntos. Se trata de un joven de 27 años practicante de triatlón (deportista de alto rendimiento). Su frecuencia cardíaca en el consultorio era de 36 lpm y su frecuencia cardíaca mínima duralte el sueño era de 27 lpm.
Agradezco anticipadamente,
Abrazo
Darío C. Sobral Filho
Profesor Asociado y Profesor Asociado de Cardiología de la Universidad de Pernambuco (UPE)Coordinador de Posgrado e Investigación de la Facultad de Ciencias Médicas - UPE



HR 33 bpm, We have two T-wave patterns: Notched T-wave and biphasic minus-plus (negative/positive)
Notched T-Wave is normally observed in children an adolescents and in adults with pericarditis



HR 39bpm

Colleages opinions

Opiniões dos colegas

Opinión de los colegas

Dear Andrés

The Holter strips on the triathlete shows the expected vagal induced bradyarrhythmias including marked sinus bradycardia, sinus arrhythmia with some junctional escapes and wandering atrial pacemaker (better seen in the 2nd recording). The middle channels have strange T waves (not sure which lead is represented) but super athletes sometimes have strange T waves. It would be interesting to see the heart rate response to exercise testing. I imagine that he would have a very high V02max and a max heart rate compatible with his age (~190 bpm)

.

Thank you for sharing

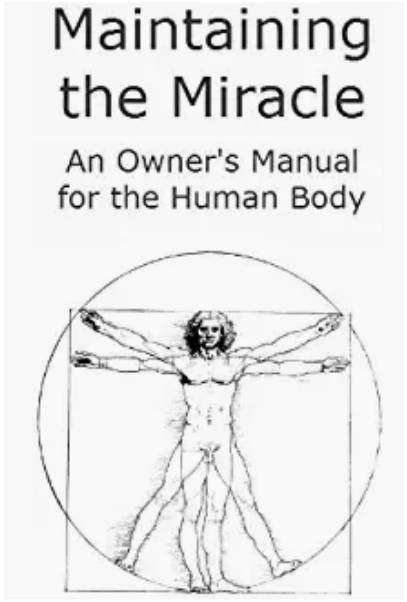
.

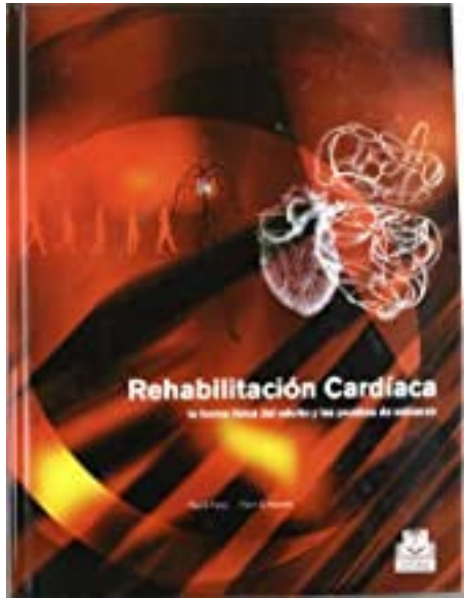
Regards,

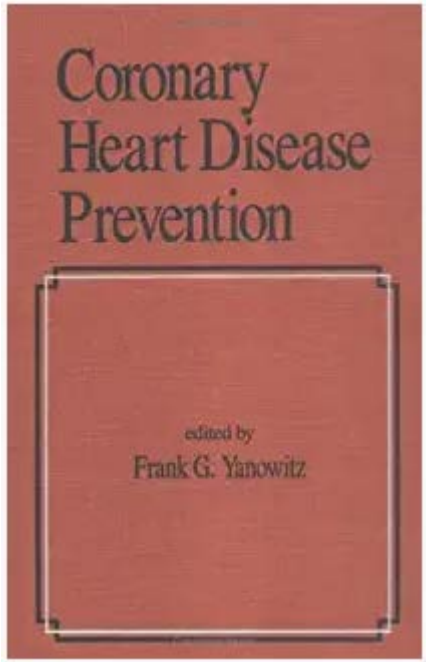
Frank G. Yanowitz MD FACC, FACP Professor of Medicine (Retired) University of Utah School of Medicine. Non-Invasive Cardiology, Preventive Cardiology, Exercise Physiology and Cardiac Rehabilitation

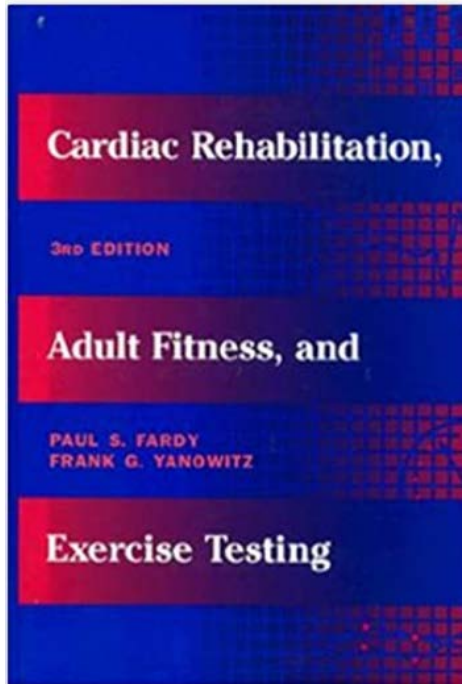
Cardiologist in Salt Lake City, Utah. USA. Books written by Frank: Click on link bellow Amazon

https://www.amazon.com.br/Livros-Frank-G-Yanowitz/s?rh=n%3A6740748011%2Cp_27%3AFrank+G.+Yanowitz









Frank Comments:

For the last 49 years I have been a member of the University of Utah School of Medicine faculty (Cardiology, Geriatrics) and working for Intermountain Healthcare in Salt Lake City. My career began with an interest in computer applications to cardiovascular medicine but later transitioned into an interest in preventive cardiology and preventive geriatrics. I have also had a long interest (since medical school) in electrocardiography, and I have created a very popular internet website on ECG education (http://ecg.utah.edu). Outside of medicine I am an professional jazz pianist and I perform often in the intermountain region around Salt Lake City.

ECG Image Index

The following ECG categories contain hundreds of ECGs that range from the sublime to the ridiculous, from simplicity to complexity, and from boring to fascinating. Many of the ECG rhythm strips come from the collection of the late Dr. Alan Lindsay, master teacher of electrocardiography. Most of the 12- and 6-lead ECGs were recorded at LDS Hospital in Salt Lake City, Utah. Marquette Electronics has also given permission to use ECG rhythms and diagrams from their educational posters. Each of the ECGs has an interpretation and many have additional explanations that help explain the diagnosis. Feedback is encouraged using the feedback form provided with this website. Fanntastic ECGs dear Frank I learn a lot with you!! Thaks dear friend Andrés

https://ecg.utah.edu/img_index

Les comparto mi interpretación

- Ritmo de bradicardia sinusal con frecuencia promedio de 34 LPM
- El quinto latido del trazado marcado como (1) – Diapositiva 2- es automatismo sinusal lento permite el escape de un ritmo nodal (la onda P antecede el QRS pero sin onda de pre-exitación)
- Se observa algo similar en los latidos 4 y 5 del trazada marcado como (2)-Diapositiva 3-

Por tanto creo que se trata de una bradicardia sinusal con una disociación isorritmica.

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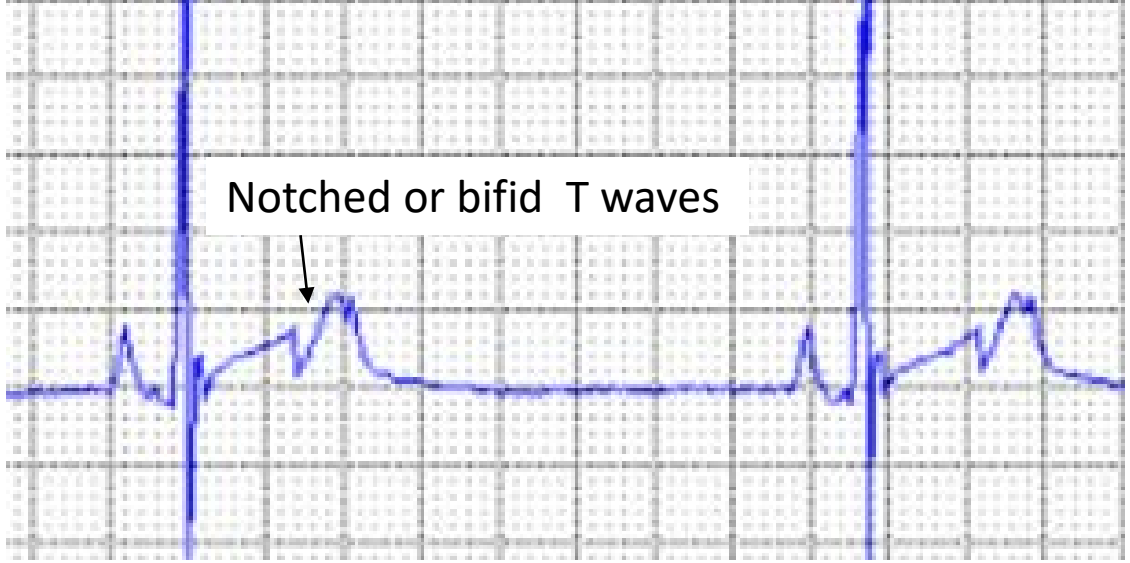
Médico Universidad Nacional de Tucuman-Cardiólogo de la Federación Argentina de cardiología
Epidemiólogo. Master en imagen cardiaca. Master en Insuficiencia Cardiaca
PhD en Ciencias Biomédicas. Expresidente Capitulo de falla cardiaca e http
Extesorero Capitulo central SCC
Miembro de junta directiva nacional Sociedad Colombiana de Cardiología 2018-2020,2020-2022

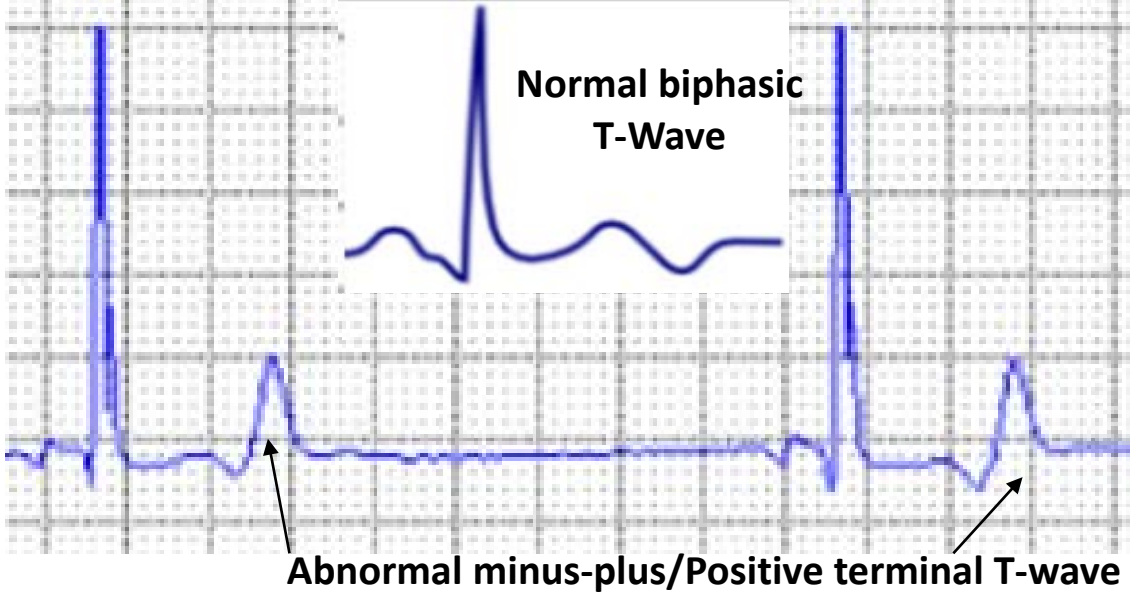
I share my interpretation- Sinus bradycardia rhythm with average rate of 34 bpm- The fifth beat of the tracing marked as (1) – Slide 2- is slow sinus automatism allows escape from a nodal rhythm (the P wave precedes the QRS but without pre-excitation wave)- Something similar is observed in beats 4 and 5 of the line marked as (2)-Slide 3-So I think it is sinus bradycardia with isorhythmic dissociation.

Final Comments

Andrés Ricardo **Pérez-Riera M.D. Ph.D.**

Internal Medicine Professor. Uninove University campus Mauá, São Paulo, Brazil.
Postgraduate advisor at Centro Universitário Saúde ABC, Santo André, SP, Brazil.





We have voltage of P-waves (P-Amplitude = 4 mm) and two T-wave patterns: notched T-wave and minus-plus (negative/positive) biphasic T-waves. **A negative-positive biphasic T wave SUCH AS THE PRESENT CASE is abnormal and often is seen in patients with RVH.**

The biphasic T waves are known for **dynamic change in polarity** . It may either pull down the or pull up the adjacent ST segment . Prolonged QT interval is a closely related to the biphasic T wave. Some times a U wave can be inscribed in such a way it may mimic a biphasic T wave.

The T wave is the positive deflection after each QRS complex. Normally, the T wave is formed at the end of the last phase of ventricular repolarization. The T wave represents the repolarization (or recovery) of the ventricles. The interval from the beginning of the QRS complex to the apex of the T wave is referred to as the absolute refractory period. The last half of the T wave is referred to as the relative refractory period (or vulnerable period).Ventricular repolarization is the process by which the ventricular myocytes return to their negative resting potential so they can depolarize again. While this phase of the cardiac cycle is rapid, an upright low amplitude broad hump following the QRS complex is seen in normal T wave morphology. Changes in T wave morphology can be indicative of various benign or pathologic conditions affecting the myocardium. Proper knowledge of T wave morphology is essential to successful evaluation and management of several conditions.

T waves are the most enigmatic waves in clinical electrocardiography . This is not a surprise , when you consider a tall T wave and a markedly inverted T wave both can be normal in at least in 6 leads out of 12 lead standard electrocardiogram (V1 V2 V3 , 2 ,3 aVF, of course the aVR) Common T wave patterns that can either be physiological or pathological: Tall T wave, Inverted T wave, Notched or Bifid T wave Biphasic T wave

* T wave polarity is strongly determined by the direction of QRS vector. Generally it should be on the same direction as QRS. In the presence of conduction defect or chamber hypertrophy this gets altered and is refered to a secondary repolarization changes . This has to be differentiated from primary biphasic T waves.

What is a biphasic T wave ? (The present case)

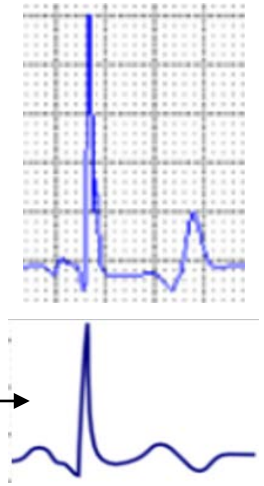
A T wave which is inscribed on either side of baseline is called biphasic T wave .

Many of the normal persons can have a biphasic T wave.

A typical biphasic wave can be two types

Terminal positivity

Terminal negativity



Terminal negativity is more significant than terminal positivity , especially in CAD.

A terminal negativity especially in mid precardial leads would suggest ongoing ischemia in LAD territory .

This happens due to dispersion of repolarization between endocardium and epicardium.

The other mechanism could be the altered ventricular gradient between QRS vector and T wave vector.

Why biphasic T waves are important ?

The biphasic T waves are known for dynamic change in polarity . It may either pull down the or pull up the adjacent ST segment . Prolonged QT interval is a closely related to the biphasic T wave.

Some times a U wave can be inscribed in such a way it may mimic a biphasic T wave. This is especially common in baseline bradycardia.

LVH is one of the common cause of biphasic T wave (Usually terminal positivity)

Biphasic T wave as mode of presentation of NSTEMI

Even though , ST depression is considered the dominant and classical theme of NSTEMI , It is now recognised NSTEMI has another mode of common presentation as biphasic T waves.

Athlete's Heart

The term athlete's heart refers to a constellation of electrical and structural cardiac adaptations in response to intensive and long-term athletic training. Such physiological cardiac remodeling allows the generation of a large and sustained cardiac output even at rapid heart rates. Although athletic adaptation of the left side of the heart has been well recognized, recent evidence revealed that the athlete's RV undergoes structural and functional adaptation in synergy with the LV. Physiological remodeling of the RV can manifest electrical and structural changes that mimic those observed in ARVC, including RV enlargement (typically in conjunction with LV enlargement and normal ventricular wall motion), anterior

precordial T wave inversion (typically biphasic T wave morphology with preceding convex ST segment elevation), and PVCs of RV origin.
Distinguishing physiological remodeling of the athlete's RV (generally considered benign and non arrhythmogenic) from ARVC (which is responsible for as many as 22% of SCDs in young athletes in Europe) has important management and prognostic implications. A false-positive diagnosis can potentially lead to erroneous disqualification from competition, whereas a false-negative diagnosis can result in devastating SCD.
RV dilatation in conjunction with convex ST segment elevation and biphasic anterior T wave inversion, and concomitant enlargement of the left and right sides of the heart with normal wall motion, appear to be benign findings and should not trigger further evaluation for ARVC in asymptomatic athletes without an adverse family history. Conversely, the presence of symmetrical anterior T wave inversion that extends beyond lead V1, preceded by isoelectric or downsloping ST segments, depolarization abnormalities (e.g., Epsilon waves or terminal QRS activation delay), reduced limb lead voltages, PVCs with LBBB morphology and superior axis, and global RV systolic dysfunction or regional wall motion abnormalities, should prompt careful investigation to exclude ARVC.
Normal T wave characteristics: Upright in all leads except aVR and V_1, Amplitude < 5mm in limb leads, < 10mm in precordial leads (10mm males, 8mm females) and duration relates to QT interval. The normal T wave in V1 is inverted. An upright T wave in V1 is considered abnormal — especially if it is tall (TTV1), and especially if it is new (NTTV1). Normal T waves are upright in leads I, II, and V3-V6,. T-wave voltage have less than 5 mm in limb leads, less than10 mm in precordial leads, and variable presentations in III, aVL, aVF, and V1-V2.(**Becker DE. Fundamentals of electrocardiography interpretation. Anesth Prog. 2006 Summer;53(2):53-63**.) This graphical depiction on ECG is associated with lead placement and the electrical pathways of the heart.

Normal Orientation

General

Normally upright in leads 1 and 2 and in the chest leads over the left ventricle.

Precordial Leads

Lead V1 may have a positive, negative, or biphasic T wave.

The T wave in V1 may be inverted at any age (is more often inverted than upright) and the T in V2 can normally be inverted.

When the T in V1 is upright, it is almost never as tall as the T in V6.

In infants and young children precordial T waves may be inverted.

In adult males it is considered abnormal if the T waves are inverted as far to the left as lead V3.

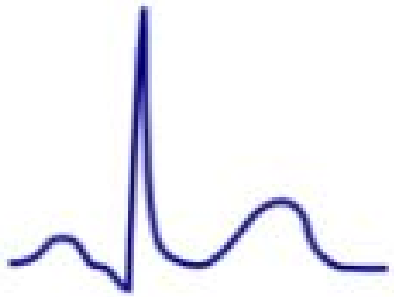
In adult females the T in V3 may be shallowly inverted.

aVF
Normally upright in aVL and aVF if the QRS is > 5 mm tall but may be inverted if the R waves are smaller.
It is not uncommon to have an isolated negative T wave in lead III, aVL, or aVF. Cardiologists are often asked to consult pre-operativley on the patient with the isolated flipped T in lead III.

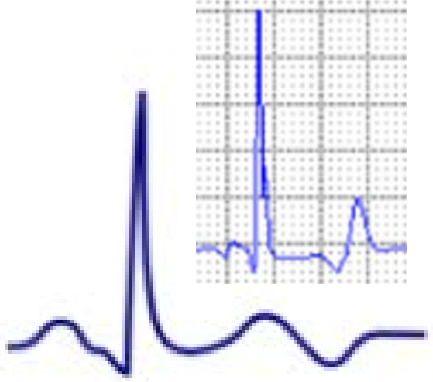
aVR
Normally inverted in aVR.

In The Presence of Conduction Delay
When a conduction abnormality (e.g., LBBB, RBBB, or a paced rhythm) is present, the T wave should be deflected opposite the terminal deflection of the QRS complex. This is known as appropriate T wave discordance. If the T waves are oriented in the same direction as the QRS complex, this is termed T wave concordance, and may be a sign of ischemia in the presence of left bundle branch block.

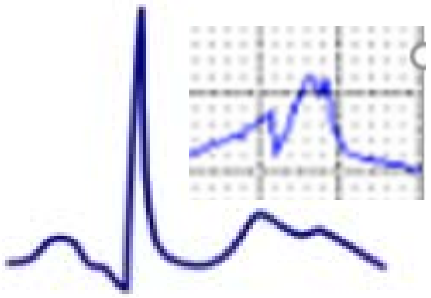
The present case



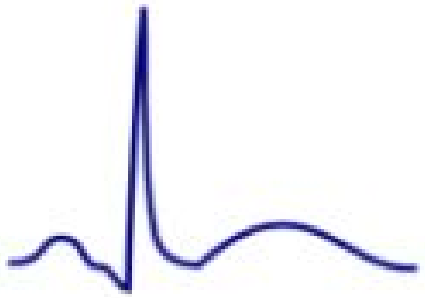
Normal



Biphasic



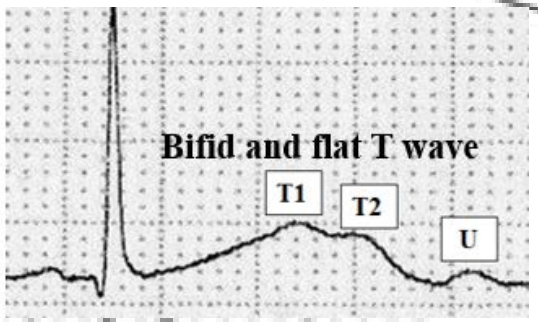
Bifid / notched

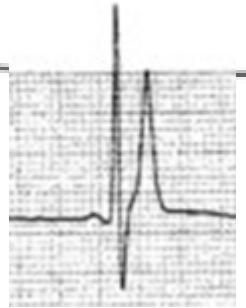


Broad / slow

Flat

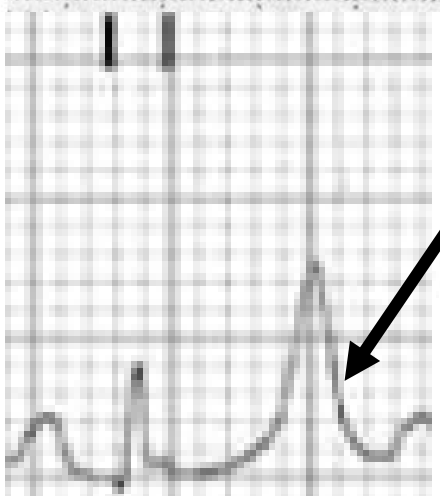
Nonspecific ST-T wave abnormalities



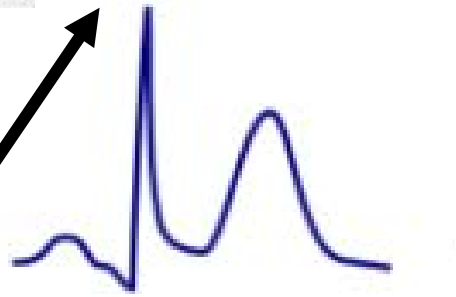


Congenital SQTS

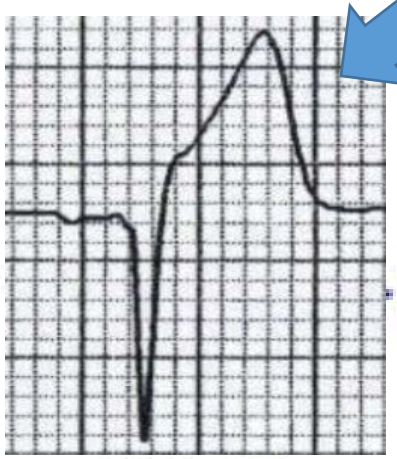
Broad, asymmetrically peaked or '*hyperacute*' T-waves (HATW) are seen in the early stages of ST-elevation MI (STEMI),



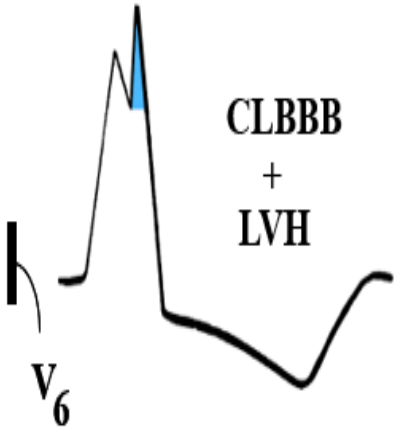
Hyperkalemic T-wave



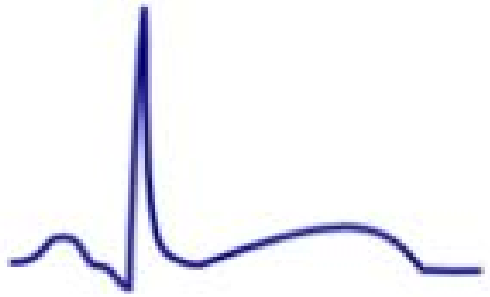
Repolarization Variant



Ischemia



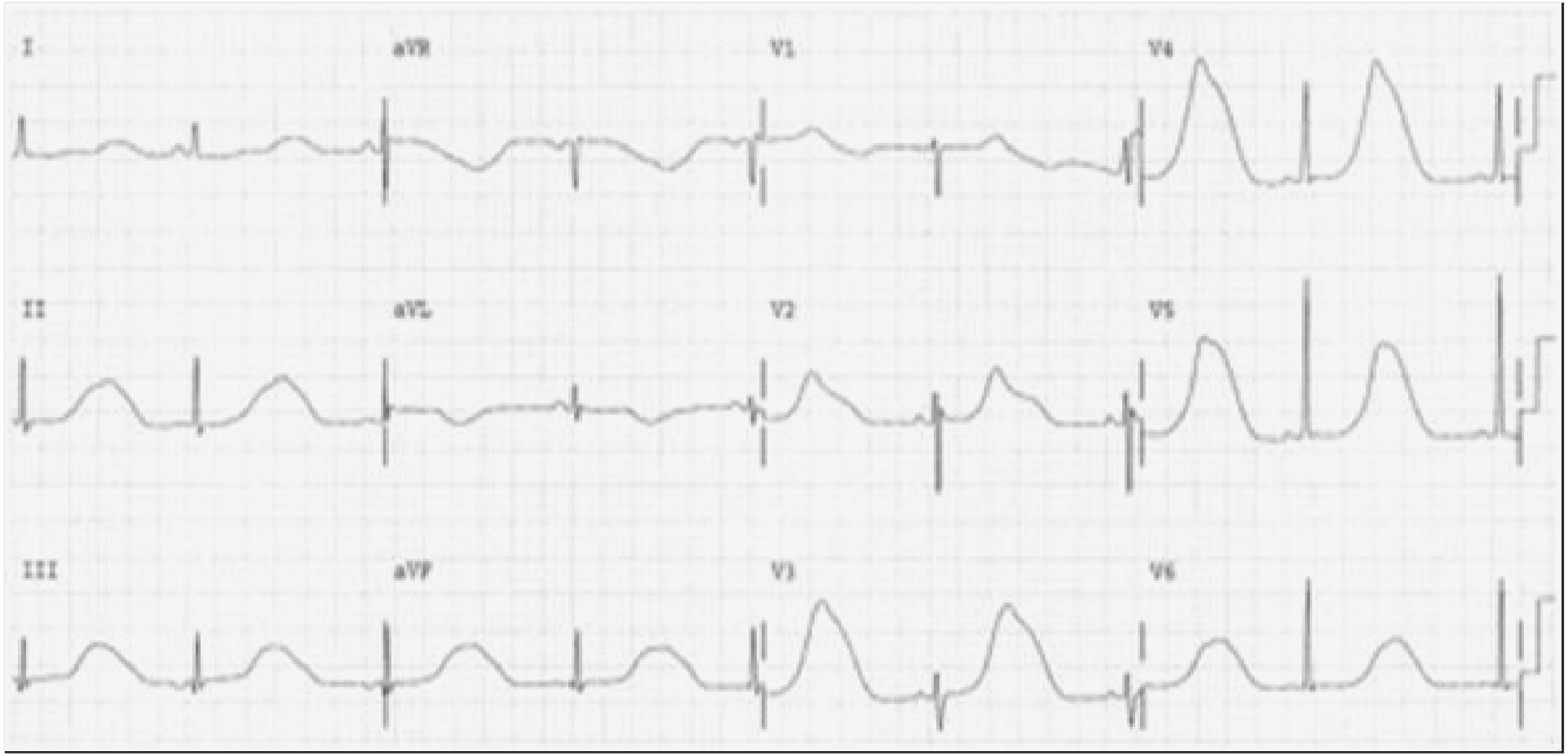
Strain pattern



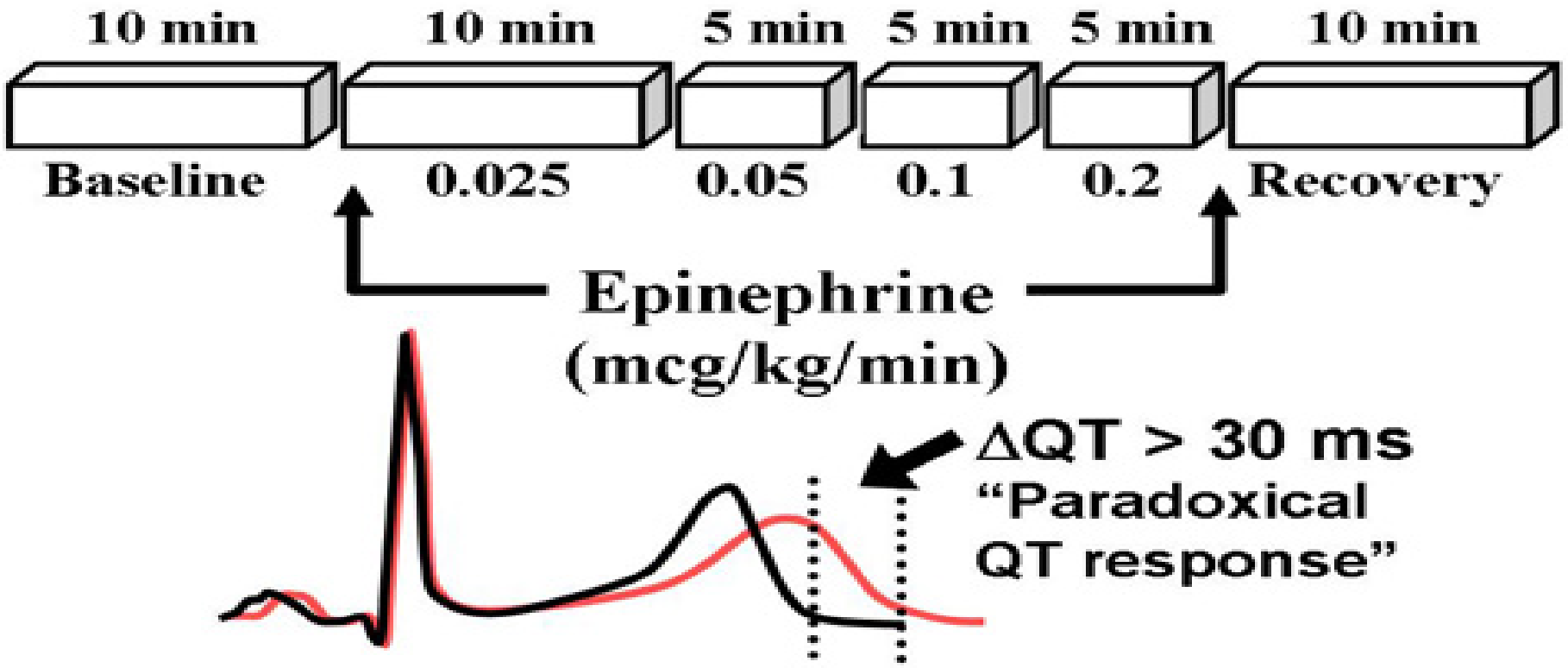
Prolonged QT interval

See next slides

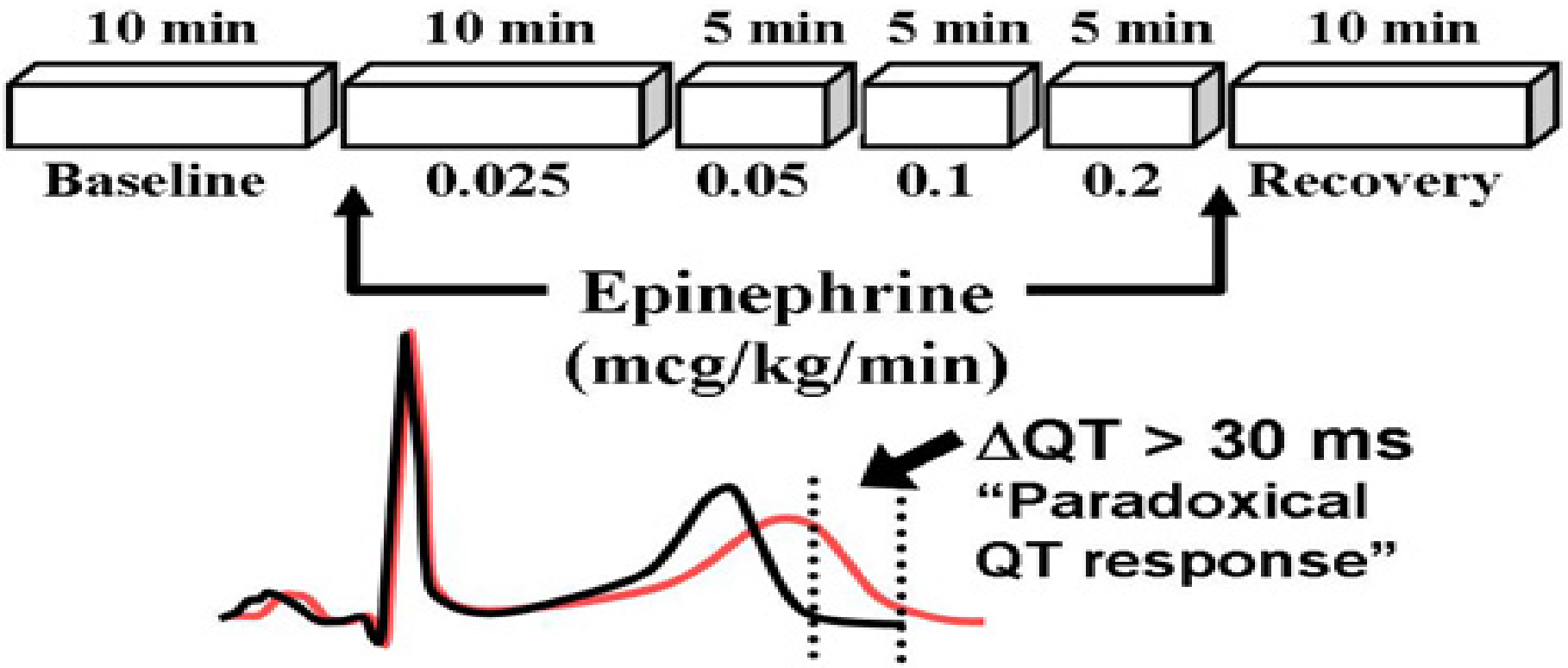
ECG example of Jervell and Lange-Nielsen syndrome with giant T waves ("Himalayan")



Marked QT interval prolongation and TU complexes with remarkable great width in patients with Jervell-Lange-Nielsen syndrome. The diagnosis of such syndrome, the recessive autosomal form of long QT syndrome, associated with congenital deafness, was confirmed by the identification of 2 different mutations in the KCNQ1 (KvLQT1) gene of potassium channel, which results in A341V and K362R; one was a de novo mutation and the other one inherited from his father. In the peripartum period, there is increased risk of arrhythmia in this syndrome. Marked QT prolongation is characteristic, but the T waves of remarkable great width that are shown here are unusual (Darbar 2005).



The Mayo Epinephrine QT Stress Test (Mayo Clinic Proceedings 2002) demonstrated that paradoxical lengthening of the absolute QT interval during low-dose epinephrine infusion has 75% positive predictive value and 96% negative predictive value with respect to LQT1. This clinical diagnostic test is now used in heart rhythm centers throughout the world in an effort to unmask patients with concealed LQT1.



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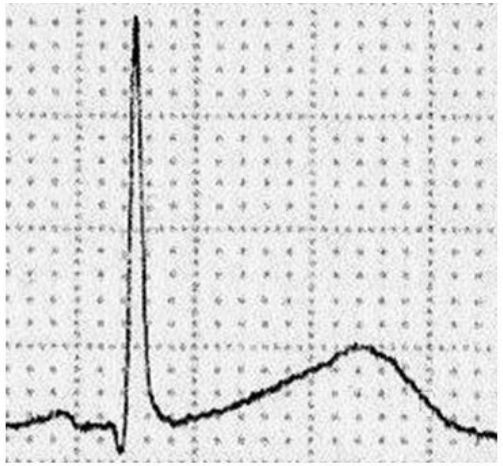
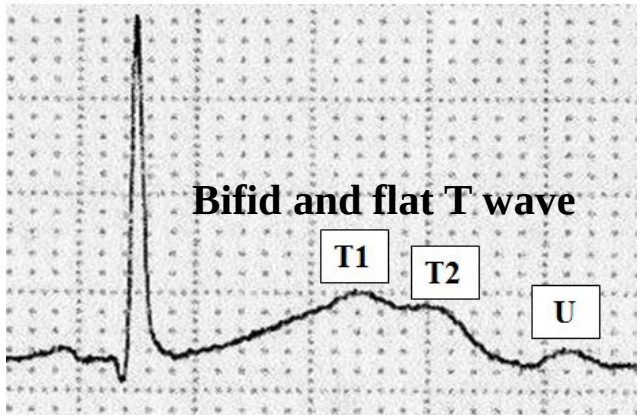
Characteristics of the HERG LQT2 variant



Normal

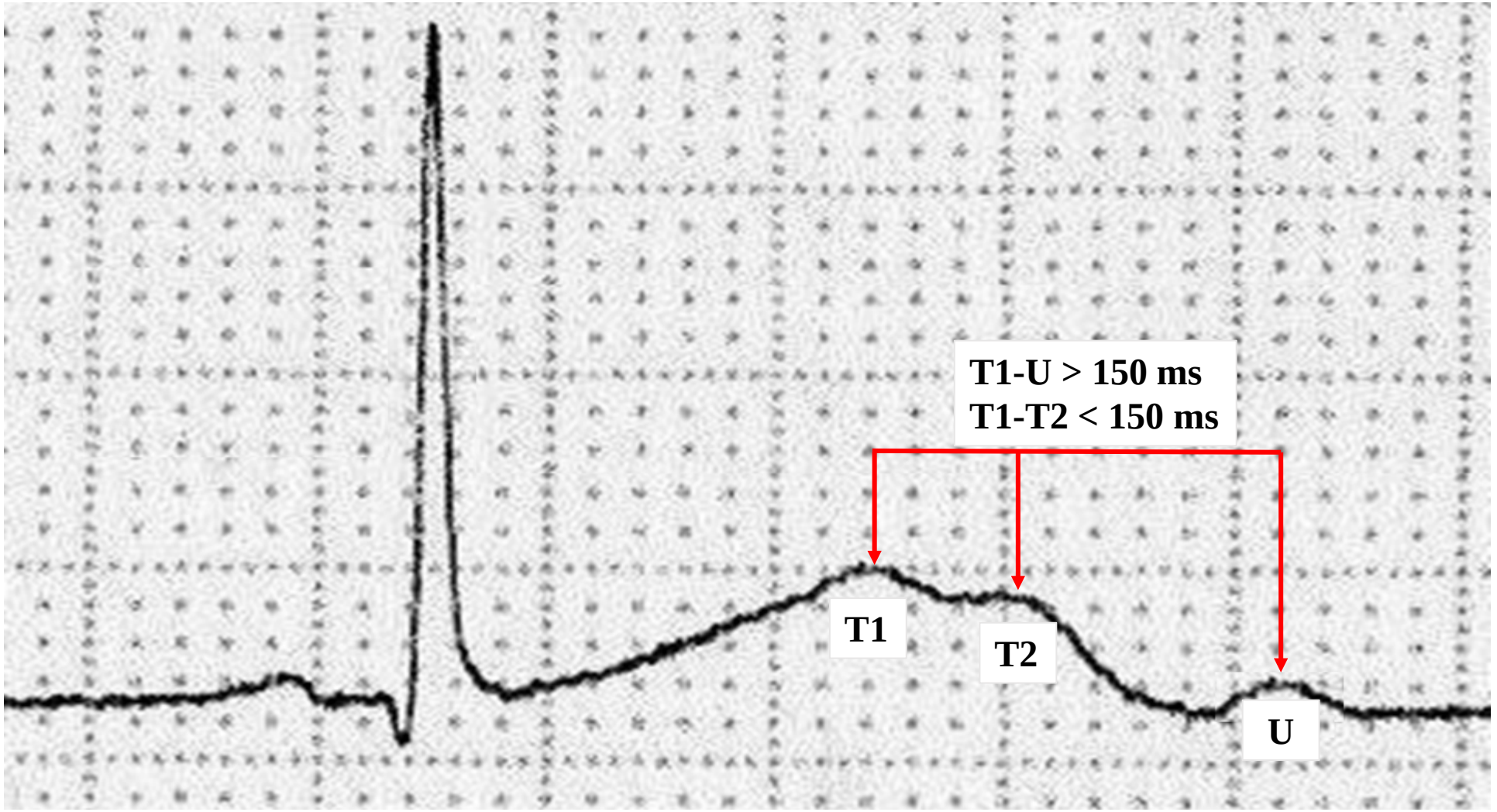


LQT2 ≈35% of the total

Event triggers: *Emotion or stress and noises*: LQT2.
Auditory arousal.

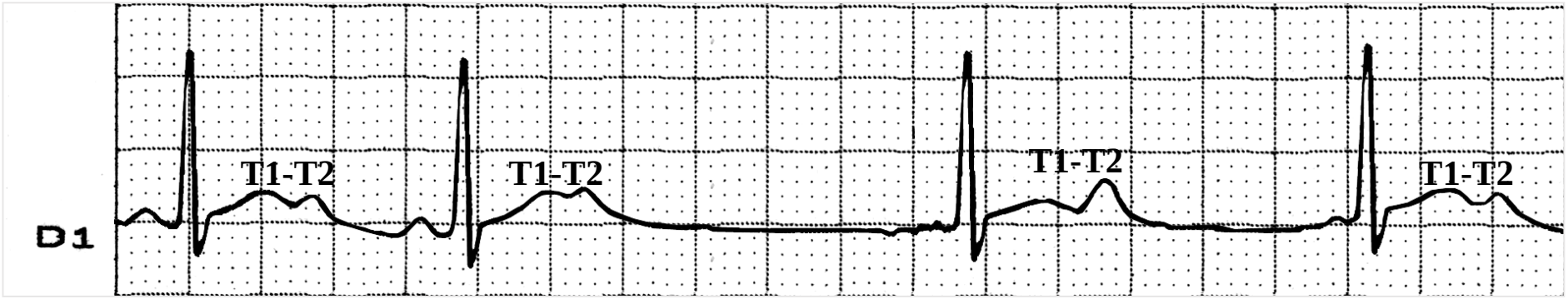
LQT2: OMIM 152437. Mutation: alpha subunit of the rapid delayed rectifier potassium channel (hERG = MiRP1). The current through this channel is known as I_{Kr}. This phenotype is also probably caused by a reduction in the repolarizing current.

Differentiation between bimodal T waves of LQT2 from the T-U interval



Characteristics of the HERG LQT2 variant (Lepeschkin 1969; 1972)

Bimodal T wave (T1-T2 pseudo U-wave dependent on bradyarrhythmic pause)

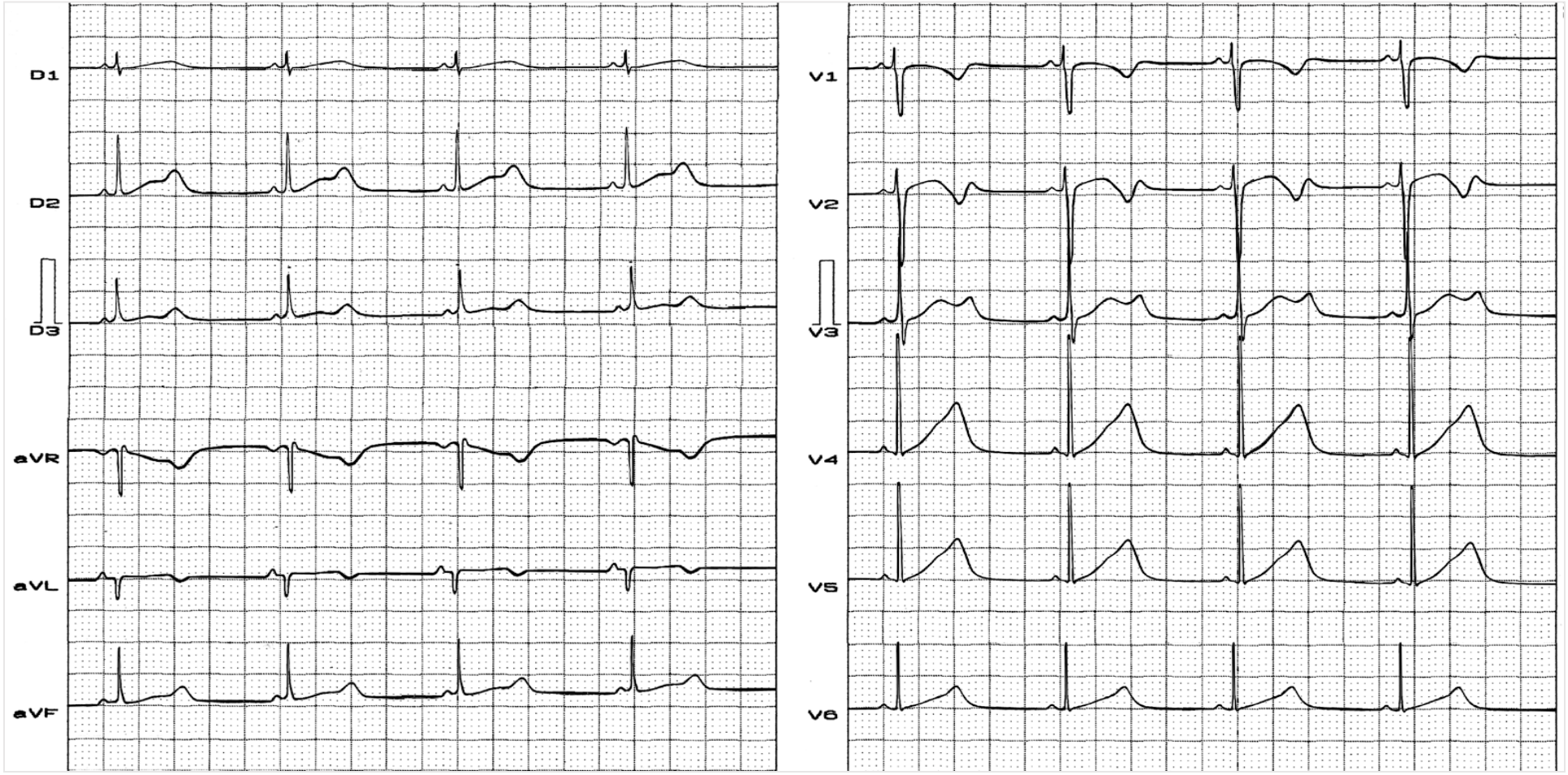


Prominent U wave that increases voltage in pauses (Roden 1999).

Congenital long QT syndrome with high-risk:

- Congenital deafness (Jervell-Lange-Nielsen syndrome).
- Recurrent syncope due to malignant ventricular tachyarrhythmia.
- Family history of sudden death.
- QTc > 500 ms.
- 2:1 atrioventricular block.
- T wave electric alternans.
- LQTS3 genotype.

Name: D.S.F; **Age:** 11 years old; **Sex:** Fem. **Weight:** 38 kg; **Height:** 1.45 m; **Race:** white; **Date:** 09/18/2001
Medication in use: Propanol 240 mg.

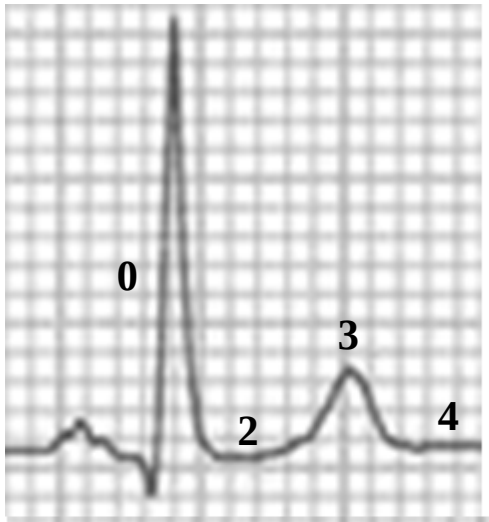


Clinical diagnosis: heredofamilial long QT syndrome without deafness. Tracing performed moments after episode of syncope. Marked increase of T-U wave is observed.
ECG diagnosis: sinus rhythm, HR: 63 bpm, long QT interval 500 ms (normal maximal value: 430 ms); very evident prominent U waves in DII and V3.

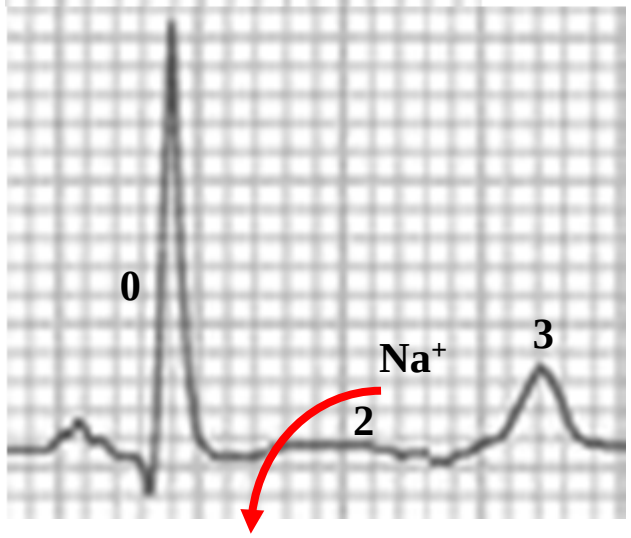
ECG of a heredo-familial long QT syndrome case without deafness. Tracing performed moments after syncope episode. Marked T-U wave increase observed.

Characteristics of the LQT3 variant, SCN5A mutation

Long QT interval by ST segment prolongation.
Delayed appearance of T wave, significant dependence on heart rate of QT interval, affected gene: SCN5A, 3p21-24 mutation in chromosome 3, TAP phase: plateau, dome or phase 2 by persistent sodium inflow.



Normal



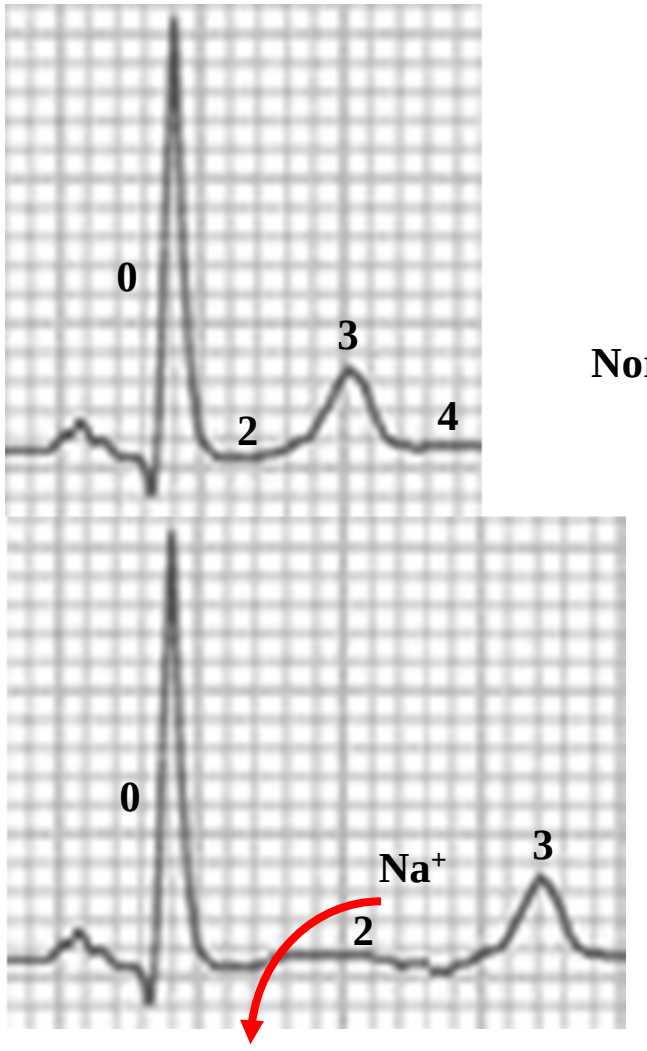
≈ 1% of the total

Delayed appearance of T wave

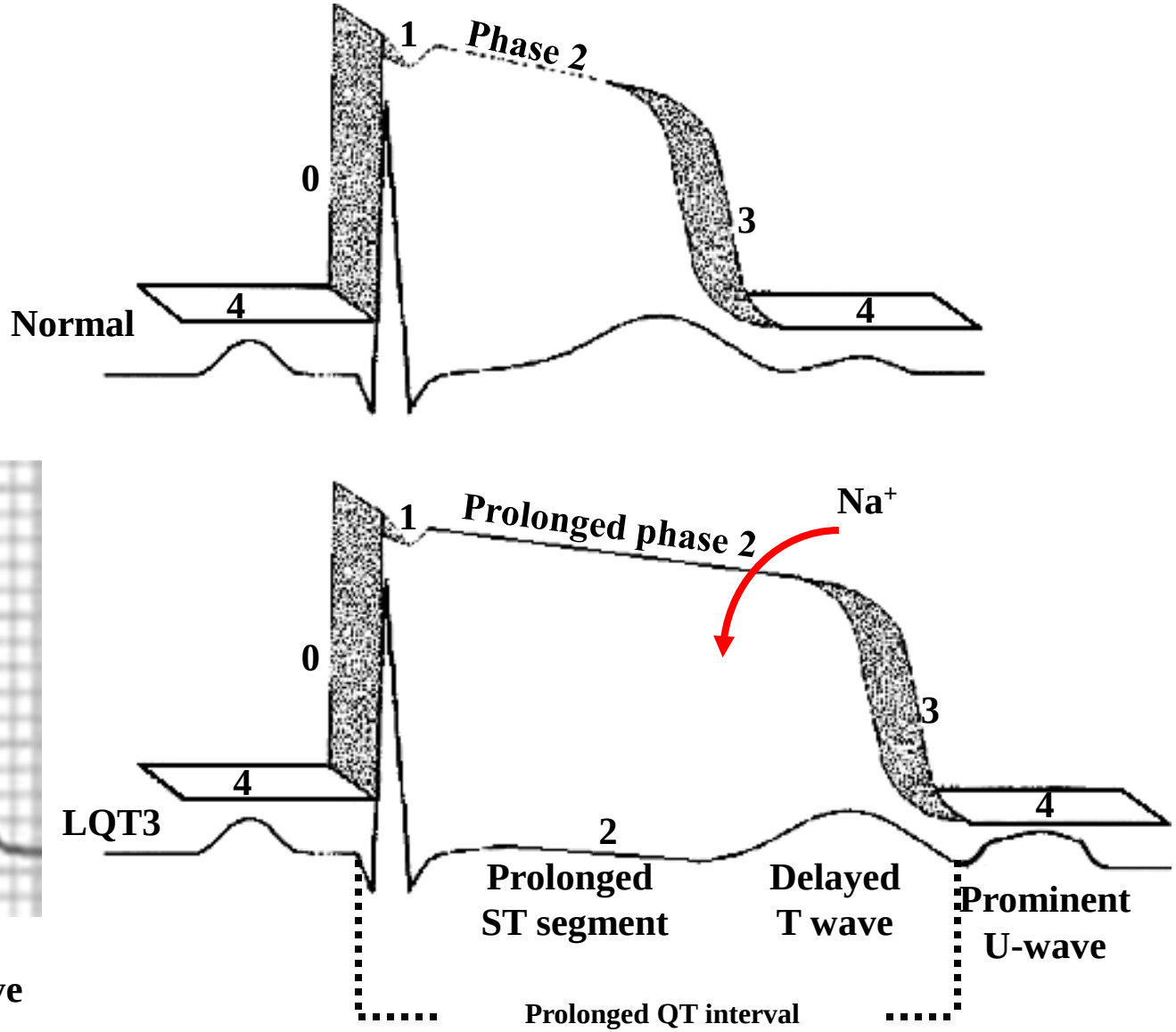
≈ 80% of events during sleeping or at rest

Male sex has a higher risk. This is the mirror image of Brugada syndrome

Normal ECG and action potential versus LQT3 ECG and action potential

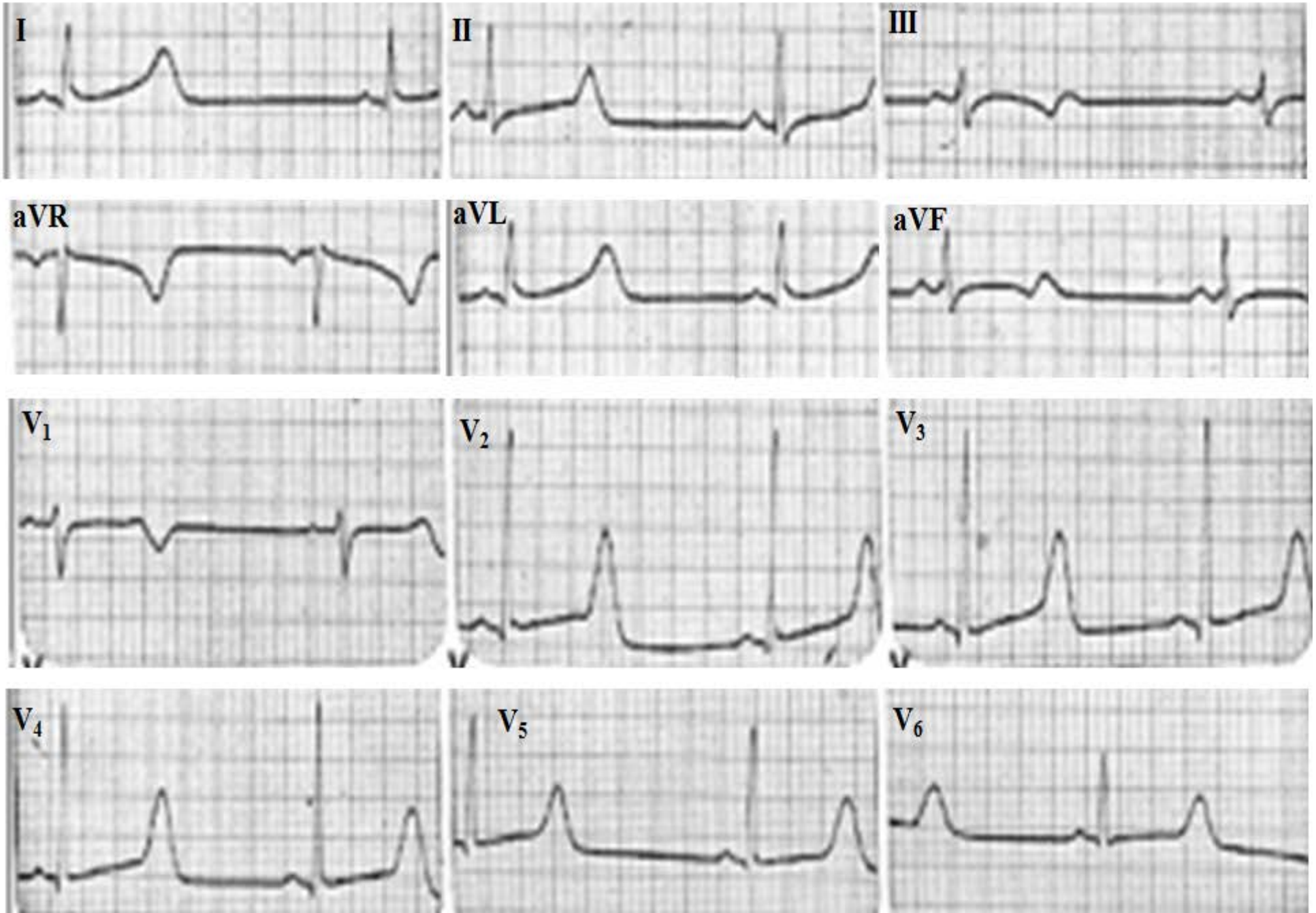


Delayed appearance of T wave

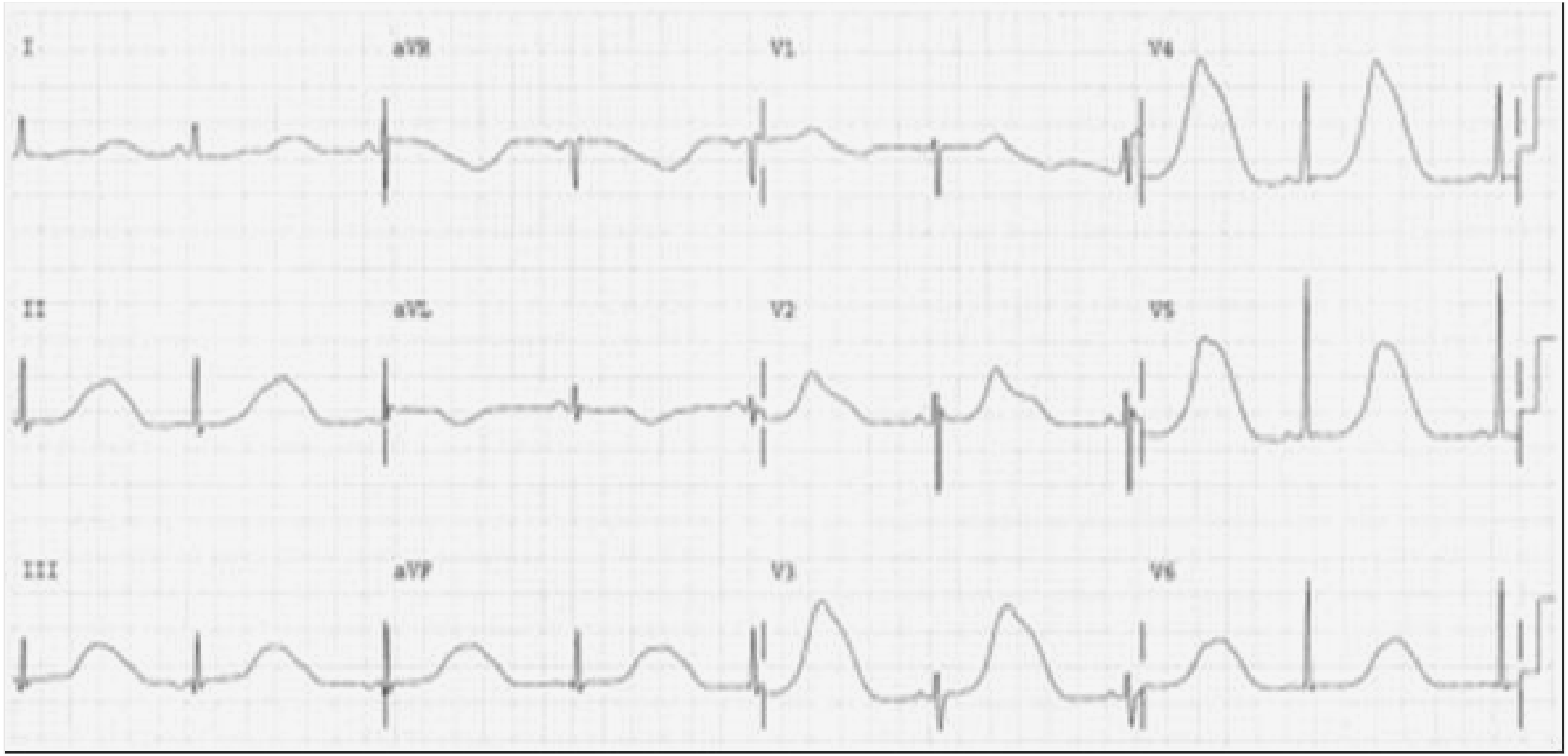


Characteristics of the LQT3 variant, SCN5A mutation

LQT3 ECG

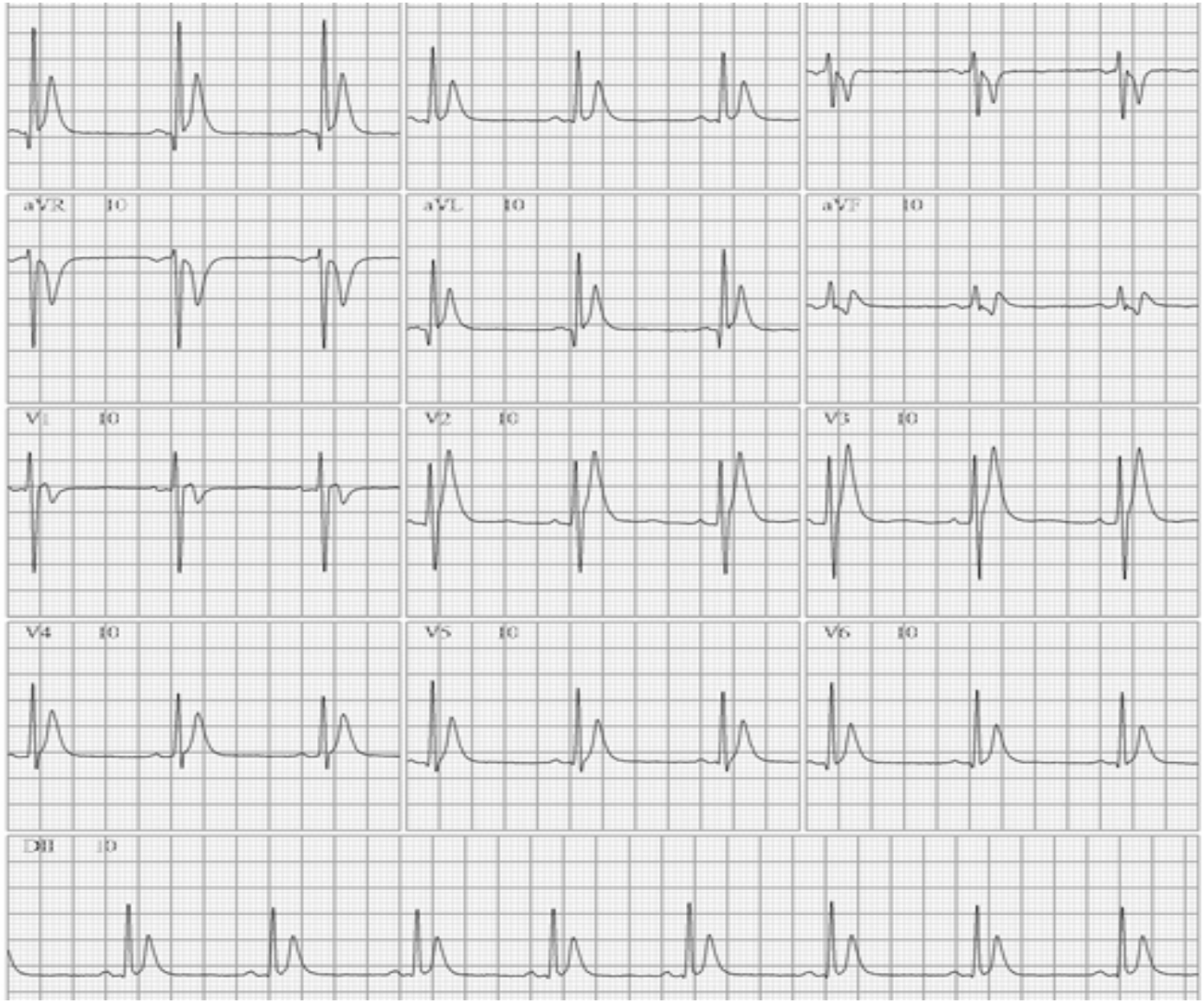


ECG example of Jervell and Lange-Nielsen syndrome with giant T waves ("Himalayan")

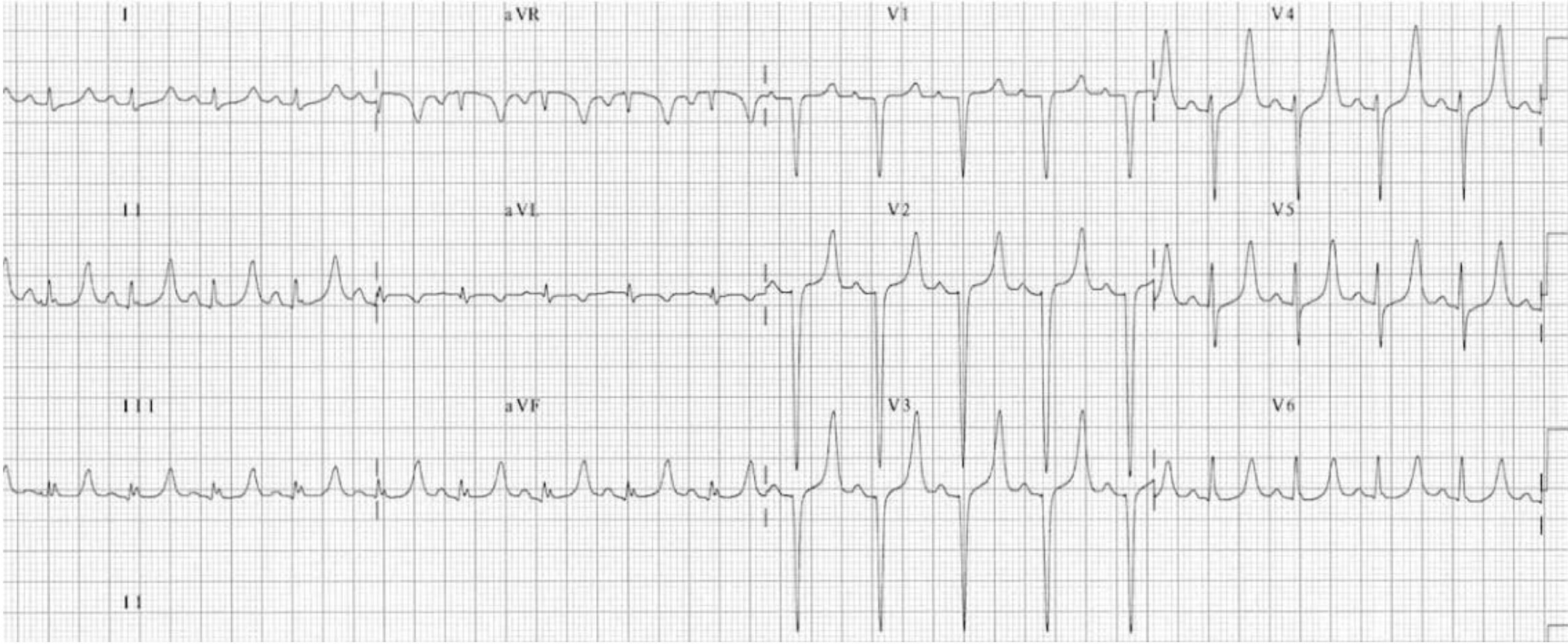


Marked QT interval prolongation and TU complexes with remarkable great width in patients with Jervell-Lange-Nielsen syndrome. The diagnosis of such syndrome, the recessive autosomal form of long QT syndrome, associated with congenital deafness, was confirmed by the identification of 2 different mutations in the KCNQ1 (KvLQT1) gene of potassium channel, which results in A341V and K362R; one was a de novo mutation and the other one inherited from his father. In the peripartum period, there is increased risk of arrhythmia in this syndrome. Marked QT prolongation is characteristic, but the T waves of remarkable great width that are shown here are unusual (Darbar 2005).

Name: MTC; **Sex**: F; **Age**: 54 y/o; **Date**: March 20, 2014; **Ethnic group**: Caucasian. ECG of one sister of the proband.



Clinical diagnosis: Congenital short QT syndrome with mutation in Caveolin-3 (SQT7).
ECG diagnosis: Sinus rhythm, HR = 68 bpm; P wave: ; SÂP + 32º, PR interval duration: 120 ms, PR segment depression (>0.5 mm) in II and V5, absence of ST segment, positive-negative T wave or “minus-plus T wave sign” in aVF, and QT = 280 ms; QTc = 295 ms.



Tall, narrow, symmetrically peaked T-waves are characteristically seen in hyperkalaemia.

T wave abnormalities include Peaked T waves, Broad, asymmetrically peaked or "hyperacute" T-waves (HATW), Inverted T waves, Biphasic T waves 'Camel Hump' T waves and Flattened T waves .

Inverted T waves are seen in the following conditions: Normal finding in children, persistent juvenile T wave pattern, myocardial ischemia and infarction (including Wellens Syndrome), Bundle Branch Block(BBB), Ventricular hypertrophy with 'strain' patterns, pulmonary embolism, hypertrophic cardiomyopathy(HCM), and Cerebrovascular injury consequence of raised intracranial pressure

Abnormalities in the T-wave may represent variations of normal cardiac electrophysiology or signs pathology. Tall T-waves (also called hyper-acute T waves) can be an early sign of STEMI. The morphology of the T waves can begin to broaden and peak within 30 minutes of complete coronary artery occlusion(CAO), and thus may be the earliest sign of MI on the ECG. The T waves will be broadened and peaked in the leads corresponding to the artery occlusion.(**Levis JT. ECG Diagnosis: Hyperacute T Waves. Perm J. 2015 Summer;19(3):79.**)

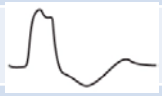
Tall T waves can also be signs of ventricular hypertrophy, depending on the distribution in the precordial leads. Additionally, T waves may be tall as a normal variant. Due to this, it is crucial to compare all ECGs with elevations in T-wave morphology to a prior study. Keep in mind elevated T waves may even occur in as normal variation in young patients and athletes, typically in the precordial V2-V4 leads.(**Hancock EW. Normal ECG or peaked T waves? Hosp Pract (1995). 1998 May 15;33(5):19-20.)**

Inverted T waves are associated with myocardial ischemia. The inversion of a T wave is not specific for ischemia, and the inversion itself does not correlate with a specific prognosis. However, if the clinical history is suggestive of ischemia in the setting of inverted T waves, this is correlative.(**Somers MP, Brady WJ, Perron AD, Mattu A. The prominent T wave: electrocardiographic differential diagnosis. Am J Emerg Med. 2002 May;20(3):243-51.**) Wellen syndrome is symmetrically inverted T waves in anterior precordial leads; these T waves suggest a severe narrowing of the left anterior descendent coronary artery at a proximal location. Recognition of this condition is vital to prevent a large anterior STEMI.(**Miner B, Grigg WS, Hart EH. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Aug 1, 2022. Wellens Syndrome**.) However, Wellens signs can be seen in various other pathologies such as pulmonary disease, so appropriate clinical correlation is imperative.Hyperkalemia is a condition that can cause peaked T waves. Depending on the degree of hyperkalemia, the peaked T-waves may range from a low amplitude to tall peaks to a sinusoidal pattern on ECG. The mechanism of the T-wave morphologies is through inhibition of the positively charged extracellular potassium on repolarization of the myocardium. In initial ECG changes in hyperkalemia, the T waves become narrow, pointed, and tall; these changes will be seen in all leads on the EKG. As the hyperkalemia progresses, other EKG abnormalities may occur: decreased P wave height, a widened QRS, PR prolongation, and eventually, the ECG may become sinusoidal.(**Somers MP, Brady WJ, Perron AD, Mattu A. The prominent T wave: electrocardiographic differential diagnosis. Am J Emerg Med. 2002 May;20(3):243-51**.) (**Levis JT. ECG diagnosis: hyperkalemia. Perm J. 2013 Winter;17(1):69.**)

Several medications are indirectly associated with T wave abnormalities. Medications such as antiarrhythmics, digoxin, and diuretics can cause electrolyte abnormalities leading to changes in T wave appearance. A key to differentiating ischemia/infarction from electrolyte-induced T-wave changes is through the distribution of changes on ECG. Electrolyte abnormalities cause diffuse changes in the T-wave morphology throughout the ECG rather than specific to a coronary artery distribution.

Diffuse, deep, symmetrically inverted T waves may be seen in a severe central nervous system trauma or pathology. These are called cerebral T waves. Conditions associated with cerebral T waves are an ischemic stroke, intracranial bleed, and traumatic brain injury.(**Somers MP, Brady WJ, Perron AD, Mattu A. The prominent T wave: electrocardiographic differential diagnosis. Am J Emerg Med. 2002 May;20(3):243-51.)** Left bundle branch block innately causes T wave to deflect in the opposite of the major deflection of the QRS. Diffuse T wave inversions on an ECG can be associated with pericarditis. The changes on an ECG for pericarditis take place over 2-3 weeks, initially with ST-elevation, then T wave inversion, with eventual resolution of the ST segment.(**Khandaker MH, Espinosa RE, Nishimura RA, Sinak LJ, Hayes SN, Melduni RM, Oh JK. Pericardial disease: diagnosis and management. Mayo Clin Proc. 2010 Jun;85(6):572-93**.) Massive pulmonary embolism can cause right ventricular strain, which can manifest as the classic S1Q3T3 (deep S wave in lead I, Q wave and T wave inversion in lead III).

Differential diagnosis

T-wave Inversion	Peaked T-waves
Normal variant	**The hyperacute phase of MI**
Myocardial ischemia	**Prinzmetal angina**
Ventricular strain	**Normal variant**
Cerebrovascular injury	**Hyperkalemia**
HCM	**LVH**
LBBB 	**LBBB**
RBBB	**Acute pericaritis**
Ventricular beats	**Congenital Short QT syndrome**

Cerebral T waves

In 1954 George Burch described T wave abnormalities as myocardial ischemia mimics in patients with a variety of acute cerebral insults. His classic paper (http://circ.ahajournals.org/content/9/5/719.full.pdf) published in May 1954 popularized the term Cerebral T waves. The T waves were described as large, were similar to those seen in early myocardial isehemia, and were reported to revert to normal with improvement of the clinical condition, or changed to the pattern of any underlying heart disease present prior to the intracranial insult. They usually appear as diffuse giant T-wave inversions or large, upright T-waves or sometimes as flat T-waves.

Etiological Theories

Originally the cause was thought to be preexisting coronary artery disease exacerbated by the physiological demands of the critical illness. However in many cases, the autopsy studies of the heart showed no macroscopic evidence of significant coronary artery stenosis or MI.

Sakamoto Hironosuke et al(**Sakamoto H, Nishimura H, Imataka K, Ieki K, Horie T, Fujii J (1996). "Abnormal Q wave, ST-segment elevation, T-wave inversion, and widespread focal myocytolysis associated with subarachnoid hemorrhage". Japanese Circulation Journal. 60 (4): 254–7.**) proposed widespread focal myocytolysis due to overstimulation of sympathetic centres in the hypothalamus leading to release of catecholamines which could damage myocardial cells

By inducing constriction of the myocardial microcirculation, thus leading to focal ischemia or By a direct toxic effect as the mechanism which result in the ECG changes seen in Subarachnoid hemorrhage. After studying the characteristic pattern of focal myocardial lesions, some researchers proposed that the damaging catecholamines are released from intramyocardial nerve endings rather than from the general (systemic) circulation.(**Baroldi G. Pathologic evidence of myocardial damage following acute brain injuries. In: Di Pasquale G, Pinelli G, eds. Heart-Brain Interactions. New York, NY: Springer-Verlag**) This focal myocytolysis is different from myocardial infarction histologically and seems to have no prelidiction for subendocardial zone which is typical for MI. Rogers et al(**Rogers MC, Abildskov JA, Preston JB (1973). "Neurogenic ECG changes in critically ill patients: an experimental model". Critical Care Medicine. 1 (4): 192–6**.) produced increases and decreases in the amplitude of the T wave in cats by stimulating the right and left sides of the hypothalamus and stellate ganglia respectively. They suggested that the mechanism is unilateral alteration of sympathetic tone to the heart. Some studies proposed the ECG changes in acute cerebral events are due to the stimulation or injury to insular cortex which is proven to have cardiovascular effects on stimulation.[6][7] (**Svigelj V, Grad A, Tekavcic I, Kiauta T (1994). "Cardiac arrhythmia associated with reversible damage to insula in a patients with subarachnoid hemorrhage". Stroke; a Journal of Cerebral Circulation. 25 (5): 1053–5**.)(**Oppenheimer SM, Gelb A, Girvin JP, Hachinski VC (1992). "Cardiovascular effects of human insular cortex stimulation". Neurology. 42 (9): 1727–32**.)The suggestion that cerebral T waves are neurally induced is supported by the observation that inverted T waves may normalize if brain death occurs.

Incidence and Prevalence

According to study on 150 acute stroke patients by David S Goldstein, T-wave inversions (Cerebral T-waves) were noticed in up to 29% of them.(**Goldstein DS (1979). "The electrocardiogram in stroke: relationship to pathophysiological type and comparison with prior tracings". Stroke; a Journal of Cerebral Circulation. 10 (3): 253–9. PMID 462510.**)

In one case series, the ECG pattern of Cerebral T-waves with prolonged QT interval was seen in 72% of patients with subarachnoid hemorrhage and 57% of patients with intraparenchymal hemorrhage.

In a study of 100 consecutive patients with cerebrovascular accident(CVA), it is noted that there is 2 to 4 fold higher incidence of Cerebral T waves when compared to control group.(**Dimant J, Grob D (1977). "Electrocardiographic changes and myocardial damage in patients with acute cerebrovascular accidents". Stroke; a Journal of Cerebral Circulation. 8 (4): 448–55. PMID 898240.**.)

New T wave abnormality, Cerebral or Cardiac???

In the acute setting, it is very significant to accurately interpret new T-wave changes to arrive at a diagnosis and provide timely intervention.

History taking should include questions about past and present history of significant cardiovascular symptoms to rule out underlying heart disease.

If any heart disease is present, the chances of it causing the abnormality should be considered.

Quick correlation should be made with the rest of the ECG and clinical presentation of the patient.

A quick neurological exam can be done to rule out cerebral origin of T-wave abnormality.

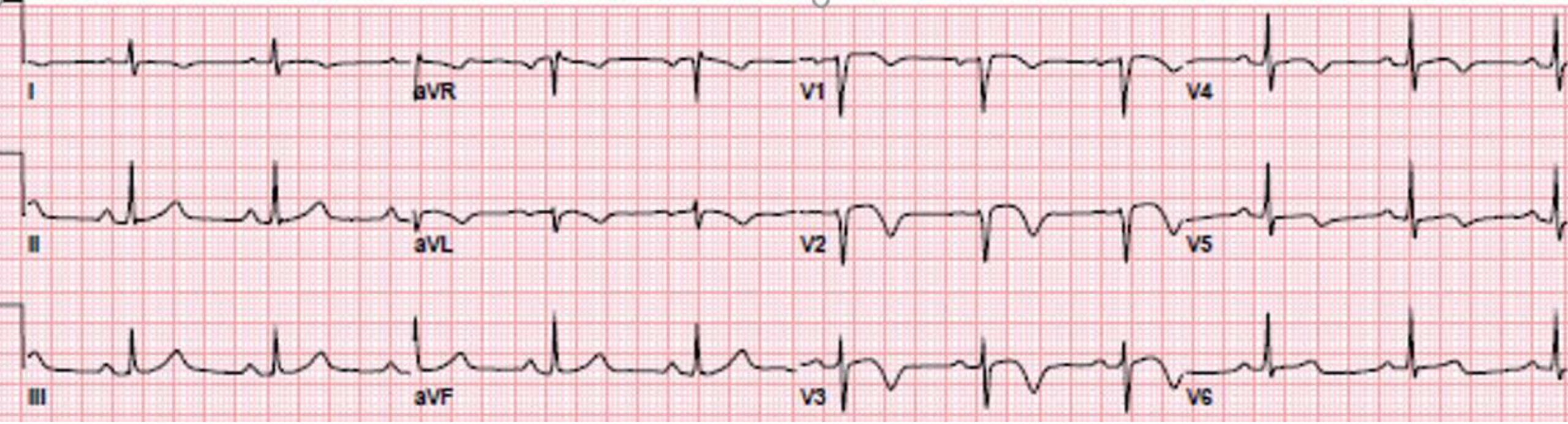
In cases where neurological exam is not possible due to patient condition, QT interval should be evaluated. Usually in cerebral causes, there is associated prolonged QT interval versus normal QT interval seen in myocardial infarction.(**Catanzaro JN, Meraj PM, Zheng S, Bloom G, Roethel M, Makaryus AN (2008). "Electrocardiographic T-wave changes underlying acute cardiac and cerebral events". The American Journal of Emergency Medicine. 26 (6): 716–20. doi:10.1016/j.ajem.2007.10.017.)**

However, to arrive at a definitive diagnosis, methods for diagnosing acute myocardial injury are necessary like

Echocardiography

Lab tests to detect elevated levels of biochemical markers of myocardial injury and

Autopsy findings in case death occurs.



ECG demonstrating the second type of pattern associated with Wellens' syndrome. In leads V2-V3, the T waves are deeply inverted, typical of Type-B T waves. Type-A T waves often evolve into Type-B T waves, which is what occurred in this patient.