Hello master,

I hope this email finds you well.

I have a very interesting ECG from a friend. I couldn’t find an explanation for this finding. I hope you can help me with that.

If we can find an explanation, other than being a glitch in the ECG machine, I will be very happy to report it.

Here is the case:

A 59 year old female was admitted for possible COVID19 Pneumonia. She is an avid smoker. No other risk factors for CAD. Her chest CT Scan showed hiatus hernia.

An ECG was done and showed a surprising T wave without preceding QRS.

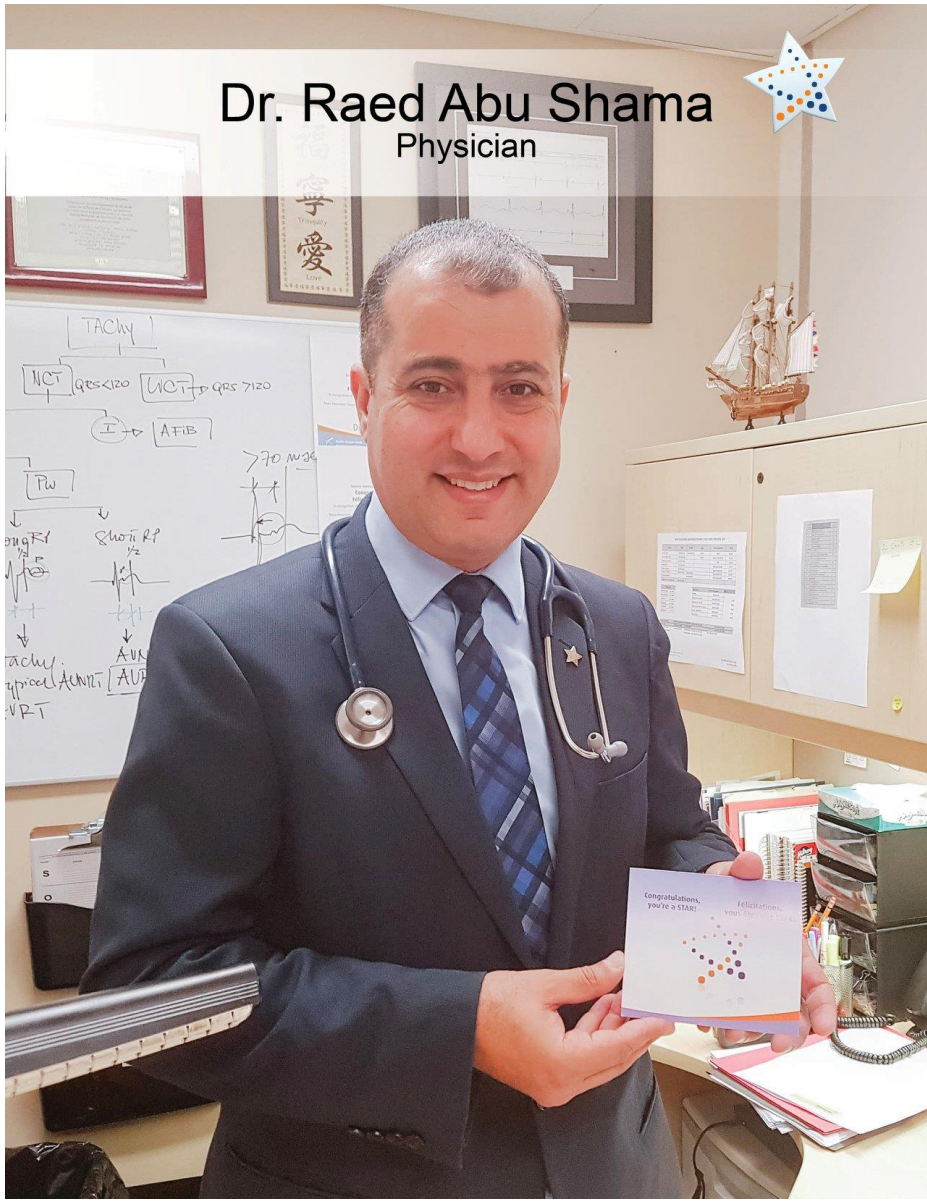
This was noted once. I could not find an explanation for this. The morphology is identical to the other T waves, the timing maps for P wave. There is no change in the preceding T wave to suggest hidden QRS complex there.

I have never seen this before. Do you have any theory or explanation?

Thank you in advance for your help.

Raed Abu Shama

Cardiac Electrophysiologist



Spanish

Hola maestro, espero que este correo electrónico te encuentre bien. Tengo un ECG muy interesante de un amigo. No pude encontrar una explicación para este hallazgo. Espero que puedas ayudarme con eso. Si podemos encontrar una explicación, que no sea un problema técnico en la máquina de ECG, estaré muy feliz de informarlo. Este es el caso: Una mujer de 59 años ingresó por posible neumonía COVID19. Ella es una ávida fumadora. No hay otros factores de riesgo de enfermedad coronaria. Su tomografía computarizada de tórax mostró una hernia de hiato. Se realizó un ECG que mostró una onda T sorprendente sin QRS anterior. Esto se señaló una vez. No pude encontrar una explicación para esto. La morfología es idéntica a las otras ondas T, los mapas de tiempo para la onda P. No hay ningún cambio en la onda T precedente que sugiera un complejo QRS oculto allí. Nunca he visto esto antes. ¿Tienes alguna teoría o explicación?

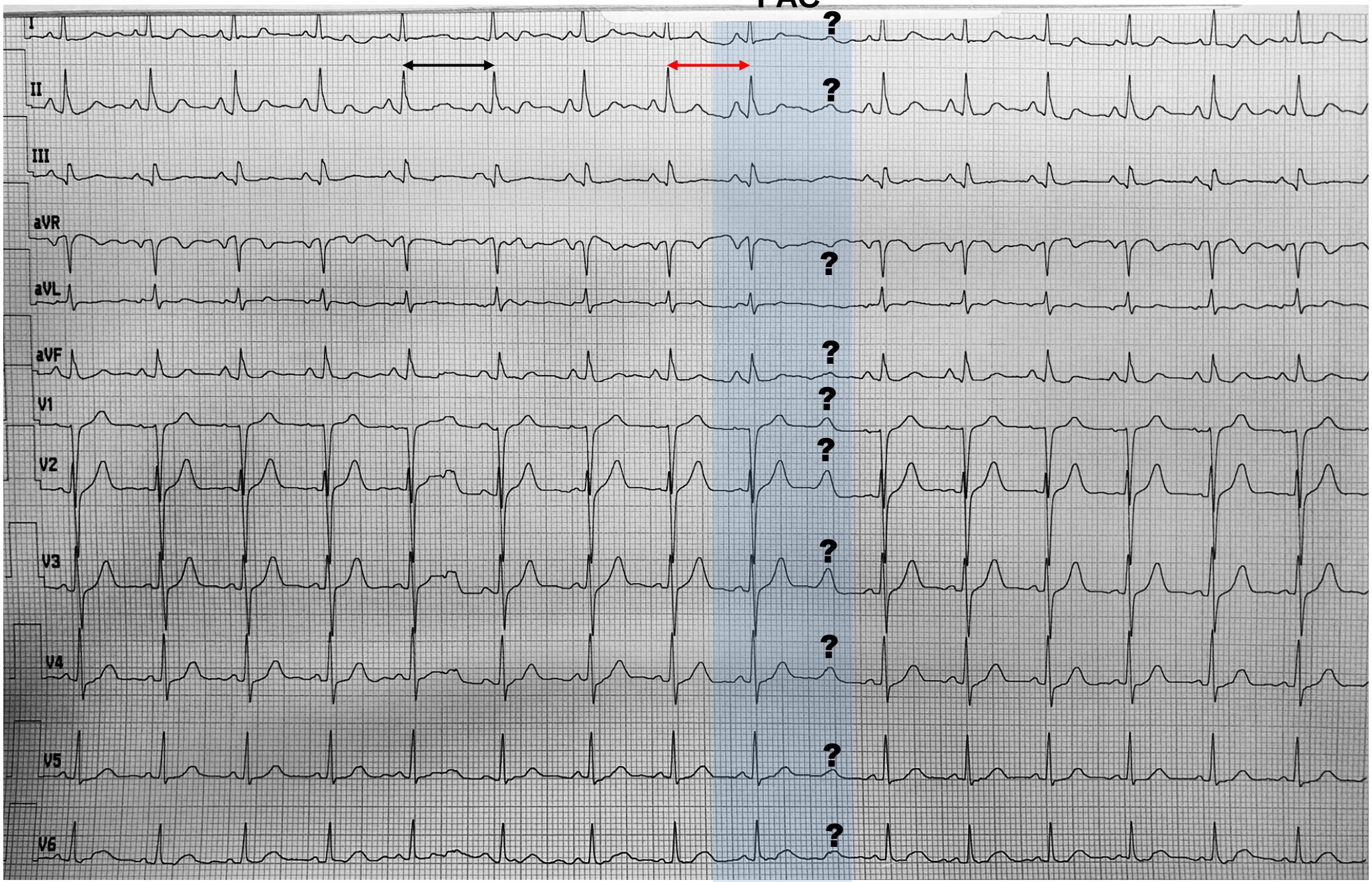
Gracias de antemano por su ayuda.

Portuguese

Olá mestre, Espero que esteja tudo bem com você. Tenho um ECG muito interessante de um amigo. Não consegui encontrar uma explicação para este achado. Espero que você possa me ajudar com isso. Se pudermos encontrar uma explicação, além de ser uma falha na máquina de ECG, terei o maior prazer em relatá-la. Aqui está o caso: Uma mulher de 59 anos foi internada por possível pneumonia COVID19. Ela é uma fumante ávida. Nenhum outro fator de risco para doença arterial coronariana. A tomografia computadorizada de tórax mostrou hérnia de hiato. Um ECG foi feito e mostrou uma onda T surpreendente sem QRS precedente. Isso foi notado uma vez. Não consegui encontrar uma explicação para isso. A morfologia é idêntica às outras ondas T, os mapas de tempo para a onda P. Não há mudança na onda T anterior para sugerir um complexo QRS oculto ali. Eu nunca vi isso antes. Você tem alguma teoria ou explicação?

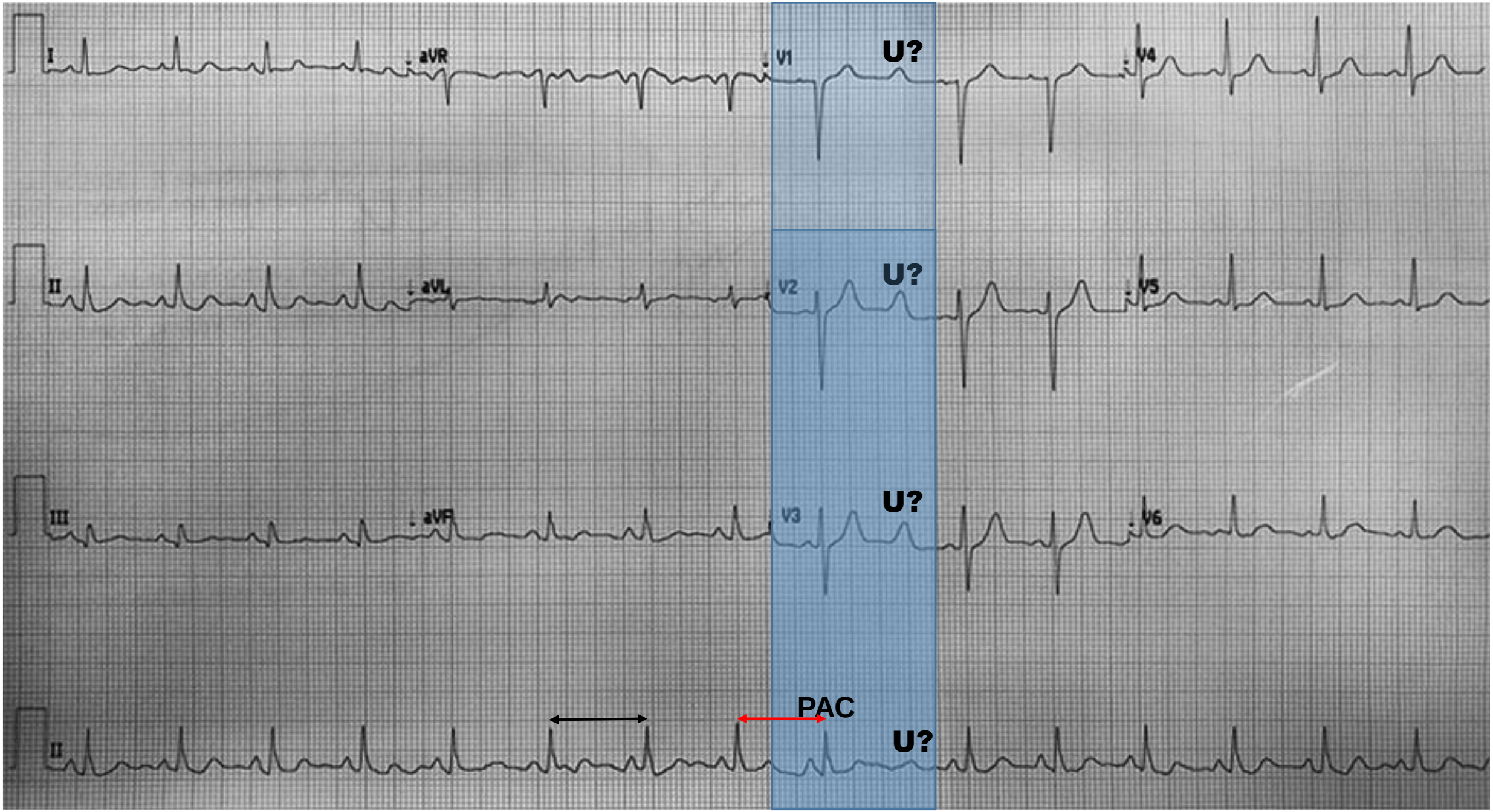
Agradeço antecipadamente por sua ajuda.

PAC



PAC: Premature Atrial Contraction

ECG performed on Dec 01, 2021 at 04:48:56 P.M. ECG 25 mm/s 40Hz



Dear Andres, This 'strange' ECG reminds me of the famous song melody, "There's No Business Like Show Business", except now we can sing "There's No Repolarization Without Depolarization"….I forgot the rest of the words. It's like a "Ghost In The Machine". I think this mystery T wave is a machine artifact of some kind. The interval from the onset of the 'ghost' T wave to the next sinus P wave is the same as that during the regular rhythm. I have no other explanation.

Regards,

Frank G Yanowitz MD Salt Lake City, UT

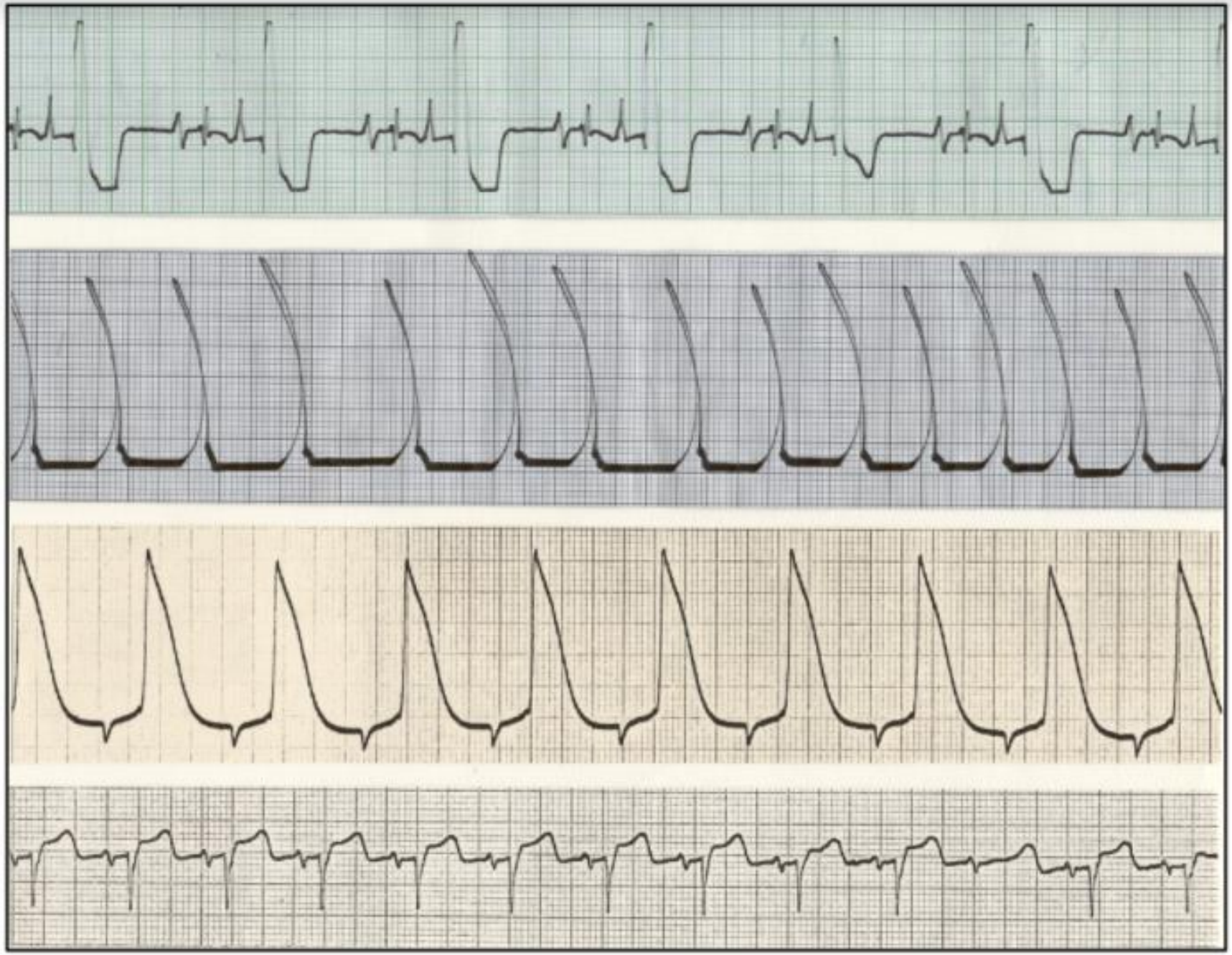
Português

Caro Andrés, Este "estranho" ECG me lembra a famosa melodia da música "Não há negócios como o show business", exceto que agora podemos cantar "Não há repolarização sem despolarização" ... esqueci o resto das palavras. É como um "Ghost In The Machine"(um fantasma dentro da máquina). Acho que essa onda T misteriosa é um artefato de máquina. O intervalo entre o início da onda T "fantasma" e a próxima onda P sinusal é o mesmo que durante a onda normal ritmo. Não tenho outra explicação.

Cumprimentos,

Frank

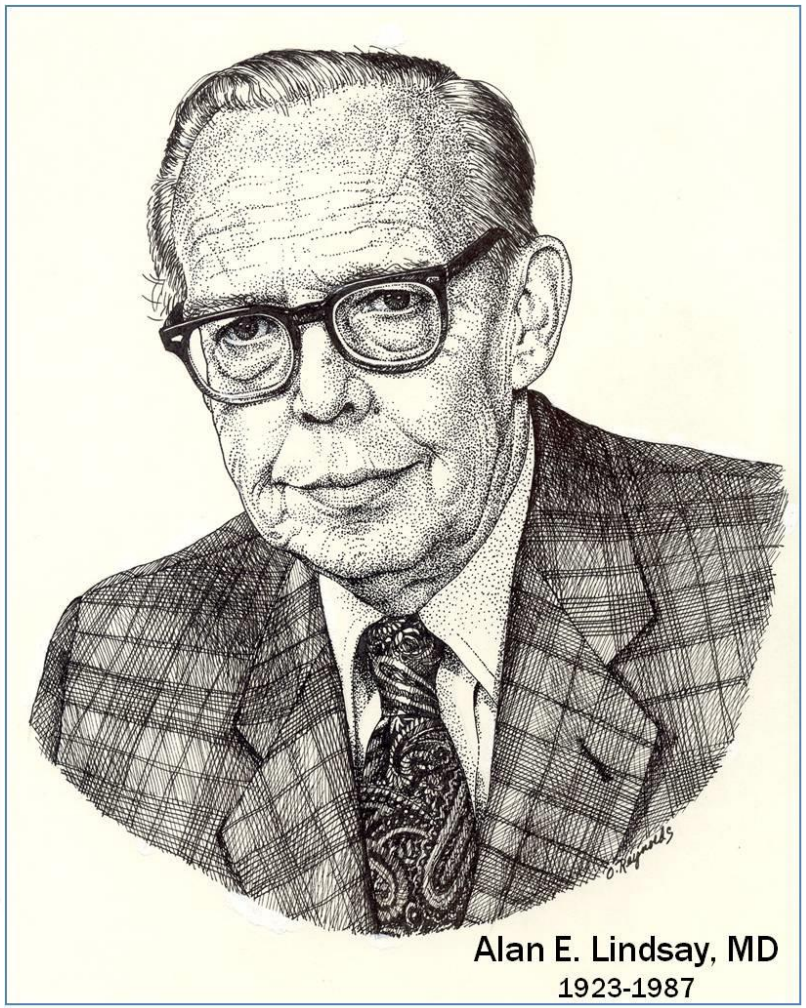
https://www.youtube.com/watch?v=PIiQMsDQ0Uo



…and finally, let it never be said that ECG's lack“ a sense of humor.”

Dear Andres, Regarding the last ECG a slide composed by my dear past friend Alan Lindsay, MD. The ECG at the bottom is interesting.

Frank G Yanowitz MD



Alan E. Lindsay, MD
1923-1987

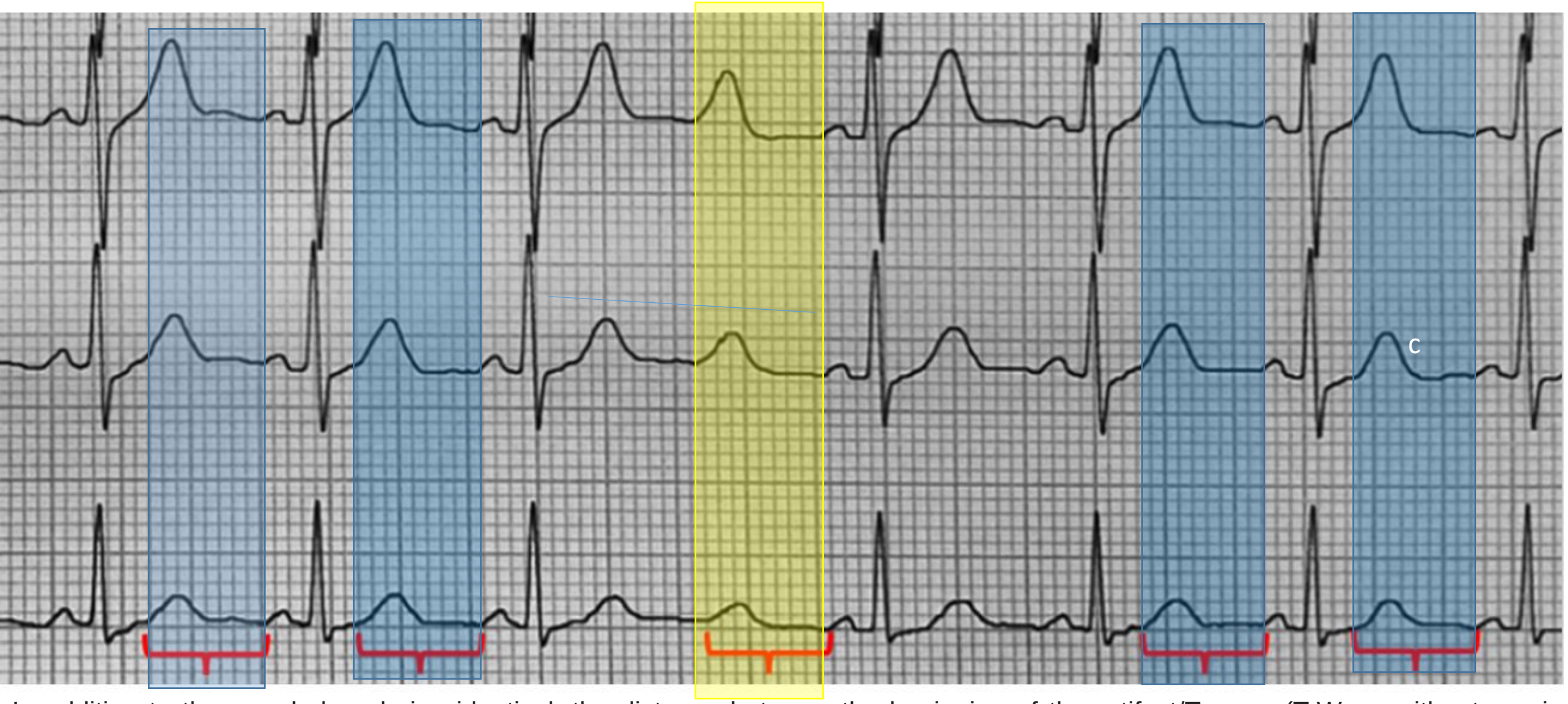
Dr. Alan Lindsay:

"A teacher of substance and style"

I've seen a description of a similar phenomenon. I believe the only possible explanation is that the P-wave and QRS were deleted from the record by a machine failure. In favor of this mechanisms, in addition to the morphology being identical, the distance between the beginning of the artifact/T wave and the beginning of the next P wave remains identical to the others complexes. What would hardly happen if the T wave were an ectopic beat, either ventricular or supraventricular. Below a publication of a similar case. Best Regards,

Antonio Thomas de Andrade,MD

Dr Antonio Thomas de Andrade measurements,



In addition to the morphology being identical, the distance between the beginning of the artifact/T wave (T-Wave without previous QRS) and the beginning of the next P wave remains identical to the others complexes. What would hardly happen if the T wave were an ectopic beat, either ventricular or supraventricular. **Dear Thomas: The T waves preceded by QRSs complexes have greater amplitude than T (or U) waves not preceded by QRS complexes differently Dr Leung L et al.case**

Leung L, Evranos B, Sohal M, Gallagher MM. An isolated T wave. Eur Heart J Case Rep. 2017 Dec 22;1(2):ytx017. doi: 10.1093/ehjcr/ytx017. PMID: 31020075 Free PMC article.

Autors informatinoa

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Corresponding author. Tel: +44 20 8725 5578, Fax: +44 020 8725 3328, Email: ku.gro.srotcod@gnuelasil.

This case report was reviewed by Bastiaan J. Boukens and Jelena Kornej.

A 25-year-old woman was referred for evaluation because of occasional episodes of palpitations. A routine ECG was performed using a GE MAC 5500 ECG machine. The ECG showed a T wave that was not preceded by a P wave or QRS complex (Figure*1*).*next slide*). The ECG was otherwise normal.

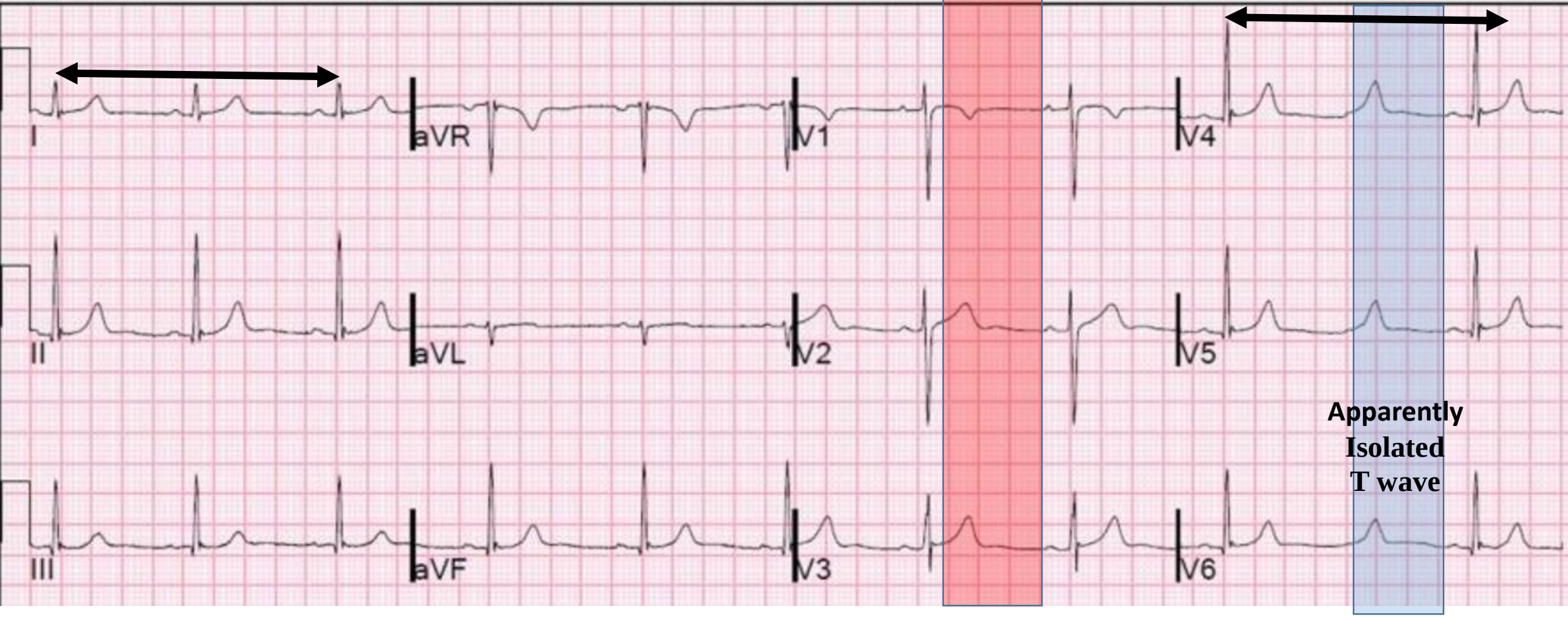
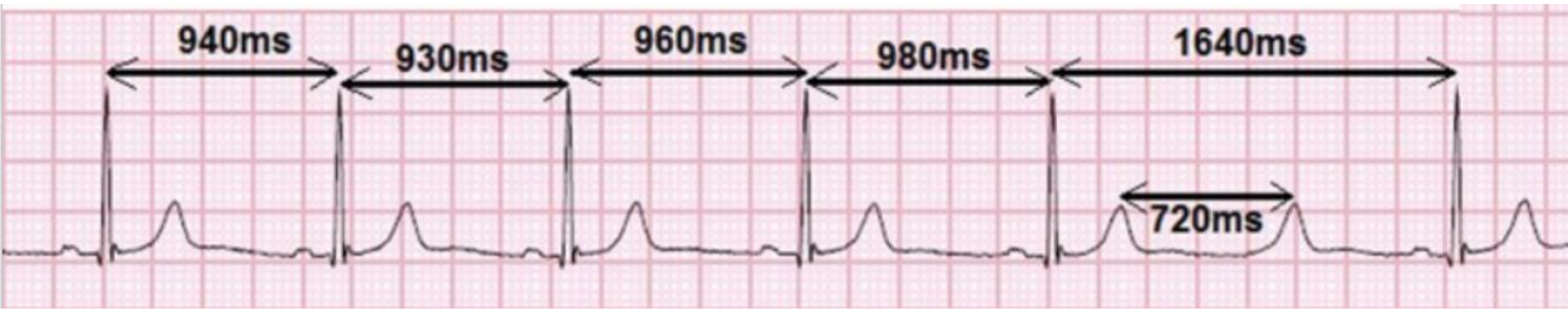


Figure 1: 12-lead ECG showing the isolated T wave (light blue).

Measurement of the RR intervals showed that the interval encompassing the isolated T wave was approximately 250 ms shorter than twice the mean of the four previous RR intervals (Figure*2*).). Measurement of the T–T interval showed that the missing 250 ms belonged to the interval preceding the apparently isolated T wave.



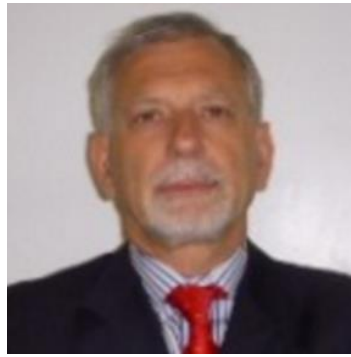
Ventricular repolarisation must be preceded by depolarisation. As the T wave is the surface ECG manifestation of ventricular repolarisation, its occurrence without a preceding QRS complex can only mean that the ECG machine has failed to register this. We conclude that the ECG machine omitted to record a period of 250 ms that encompasses the P wave and QRS complex, which should have accompanied the isolated T wave. A well-maintained digital ECG machine failed to register the P wave and QRS complex of one cardiac cycle. No explanation other than artefact could explain the findings. This ECG artefact phenomenon has not been previously described. A similar transient failure of data collection falling at a different part of the cardiac cycle could produce an impression of atrioventricular block or shortening of the PR or QT interval, with potential clinical consequences. Observation of any discrepancy in the RR interval could be easily dismissed as sinus arrhythmia. Non-physiological artefacts of the surface ECG were more common in the era of analogue recording equipment but in the digital era, an understanding of how artefacts can still be produced on an ECG is important as well as the fact that all machines are subject to error.[1,2]

References

1. Stevenson W, Maisel W.. Electrocardiography artifact: what you do not know, you do not recognize. Am J Med 2001;110:402–403.10.1016/S0002-9343(01)00637-4 [PubMed] [CrossRef] [Google Scholar]
2. Knight BP, Pelosi F, Michaud GF, Strickberger SA, Morady F.. Physician interpretation of electrocardiographic artifact that mimics ventricular tachycardia. Am J Med 2001;110:335–338. [PubMed] [Google Scholar]

Un ECG muy interesante. La repolarización de algunos latidos (no todos) esta deformada, particularmente en el plano frontal. Es posible que sean extrasístoles auriculares.
Onda U, sola, aislada y muy separada de la onda T. Nunca vi.
El diagnóstico podría ser esclarecido con registro intra-esofágico.
Espero ver las observaciones y explicaciones de los expertos.
Saludos.

Oscar Pellizón MD Rosario National University CATEDRA DE CARDIOLOGIA



A very interesting ECG. The repolarization of some beats (not all) is deformed, particularly in the frontal plane. It is possible that they are atrial premature contractions .U wave, alone, isolated and very separate from the T wave. I never saw. The diagnosis could be clarified with intra-esophageal recording. I look forward to seeing the comments and explanations of the experts. Greetings.
Oscar

Querido Oscar muy interesante tu comentario de realizar registro intraesofágico con el objetivo de esclarecer el diagnóstico. Por otra parte, Parece ser una T y não onda U. El problema como dice Frank "no existe onda T sin QRS precedente"

Dear Oscar: Your comment about performing intra-esophageal electrocardiographic registration in order to clarify the diagnosis is very interesting. On the other hand, it seems to be a T and not a U wave. The problem is as Frank says:

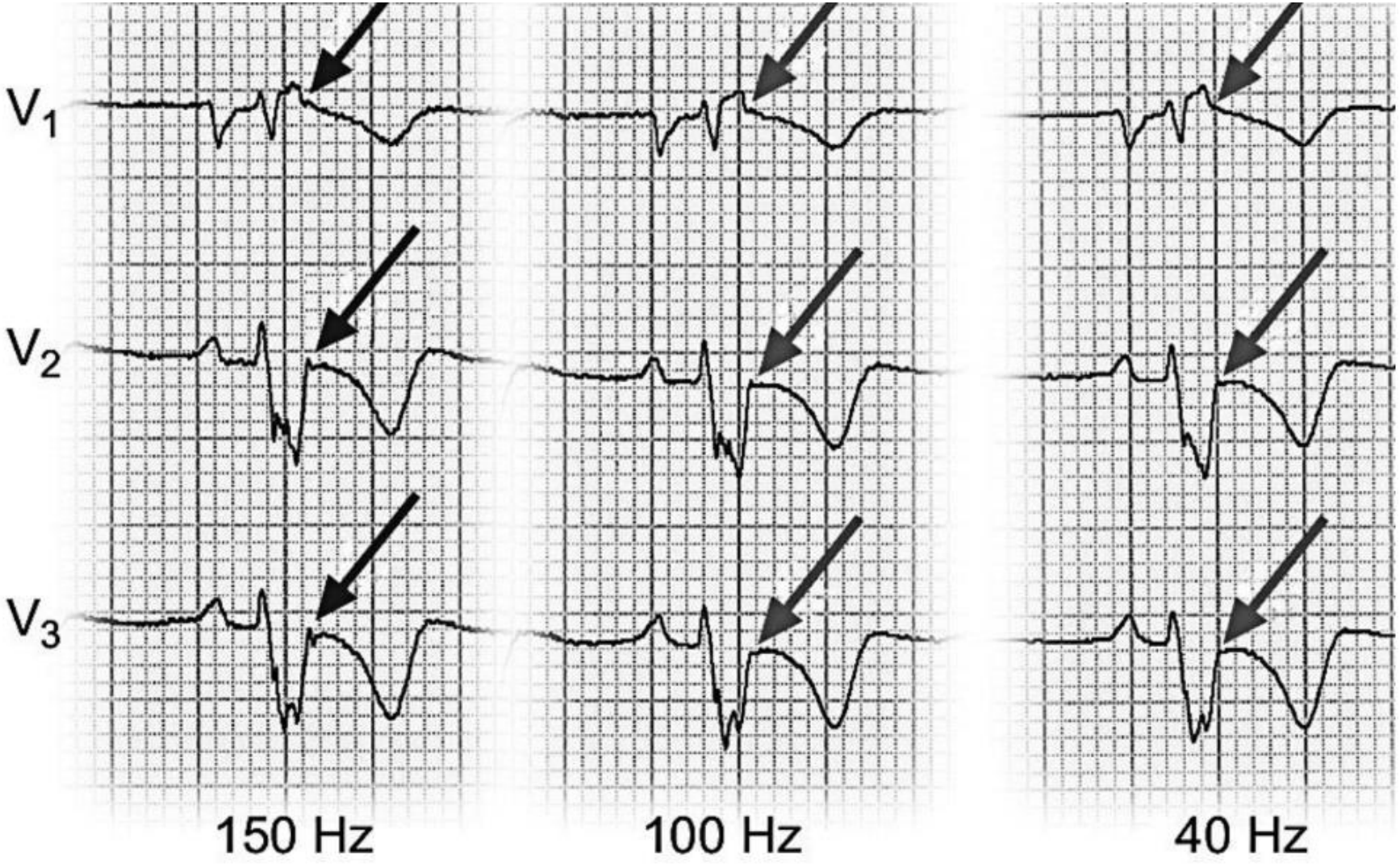
"There is no T wave without a corresponding preceding QRS complex".

Andres

Andrés R. Pérez-Riera comments

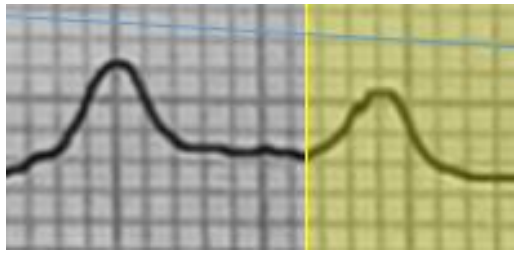
My new “daughter” Bella

Your ECG used 40 Hz filter which is inappropriate because has low resolution.



Example: Low-pass filter cutoff frequency influences the detection of the ε wave in AC: at the recommended 150 Hz cutoff frequency, the ε wave is detected in leads V1-V3. At a 100 Hz cutoff frequency, the ε wave is attenuated in V1-V2 and absent in V3. At 40 Hz, the ε wave disappears from leads V1-V3 (Garcia-Niebla J, Baranchuk A, Bayes de Luna A. Epsilon Wave in the 12-Lead Electrocardiogram: Is Its Frequency Underestimated? Rev Esp Cardiol (Engl Ed). 2016;69:438).

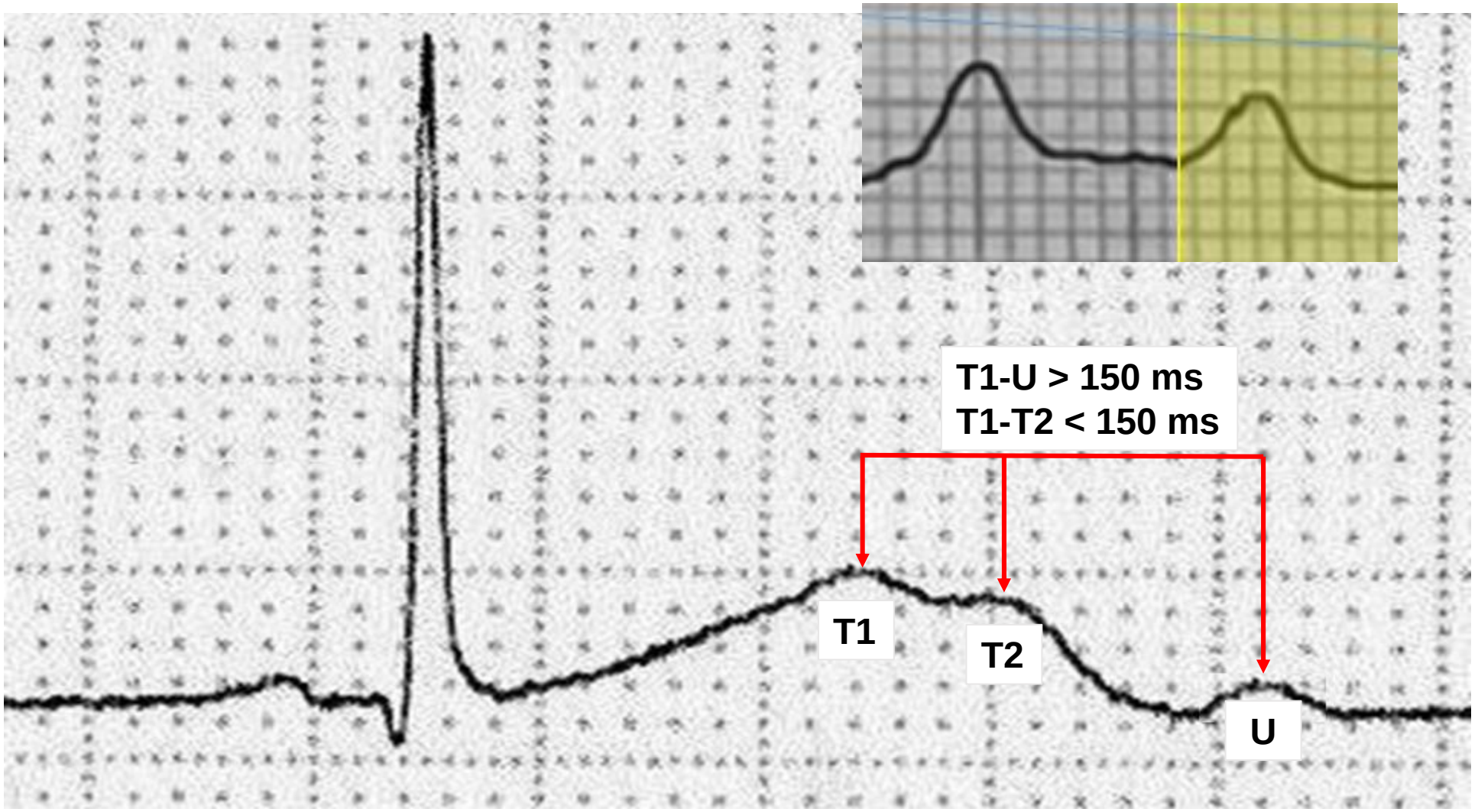
Dear Raed, I think that the hypotheses of Frank and Thomas are more likely to occur in the present case, therefore, there are differences in voltages between the T waves preceded by the corresponding QRS complex **There's No Repolarization Without Depolarization**.



Viskin et al attempted to determine the correlation between the presence of post- extrasystolic changes in the STU segment and a history of sustained VT. The authors compared the configuration of the STU segment of the post-extrasystolic beat (the sinus beat after a PVC) with the STU configuration during sinus rhythm in three patient groups: 1) 41 patients with spontaneous VT/VF (VT/VF group), 2) 63 patients with heart disease and high grade ventricular arrhythmias (control group), and 3) 29 patients with high grade ventricular arrhythmias without structural heart disease (reference group). Post--extrasystolic T wave changes did not correlate with a history of VTs. However, post-extrasystolic U wave changes were more common among the patients with VT/VF than among control subjects. By logistic multiple regression analysis, a low LVEF and post-extrasystolic U wave changes were independent predictors of VTs. They concluded that post-extrasystolic T wave changes are common and lack predictive value. Post-extrasystolic U wave changes may be a specific marker of a tendency to the development of spontaneous VTs. Prospective studies should be performed to confirm this association.

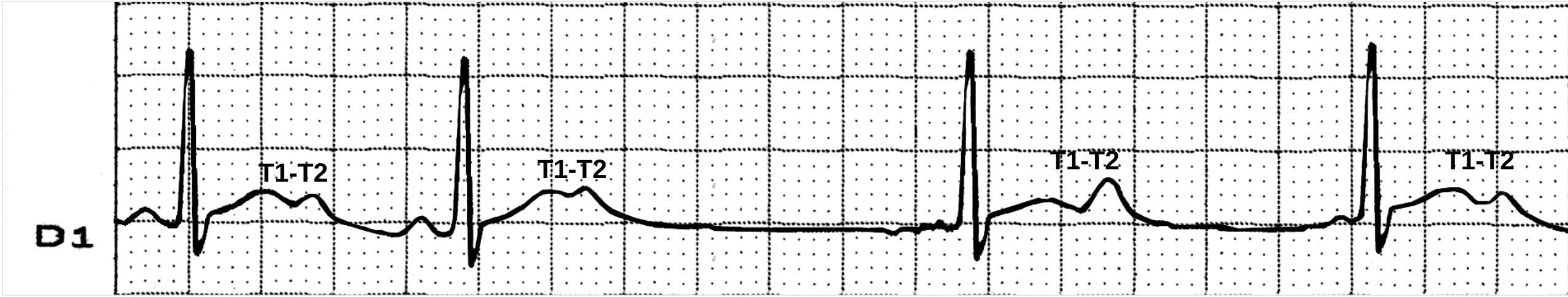
S Viskin 1, K Heller, H V Barron, I Kitzis, M Hamdan, J E Olgin, M J Wong, S E Grant, M D Lesh. Postextrasystolic U Wave Augmentation, a New Marker of Increased Arrhythmic Risk in Patients Without the Long QT Syndrome. J Am Coll Cardiol. 1996 Dec;28(7):1746-52. doi: 10.1016/S0735-1097(96)00382-8.

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



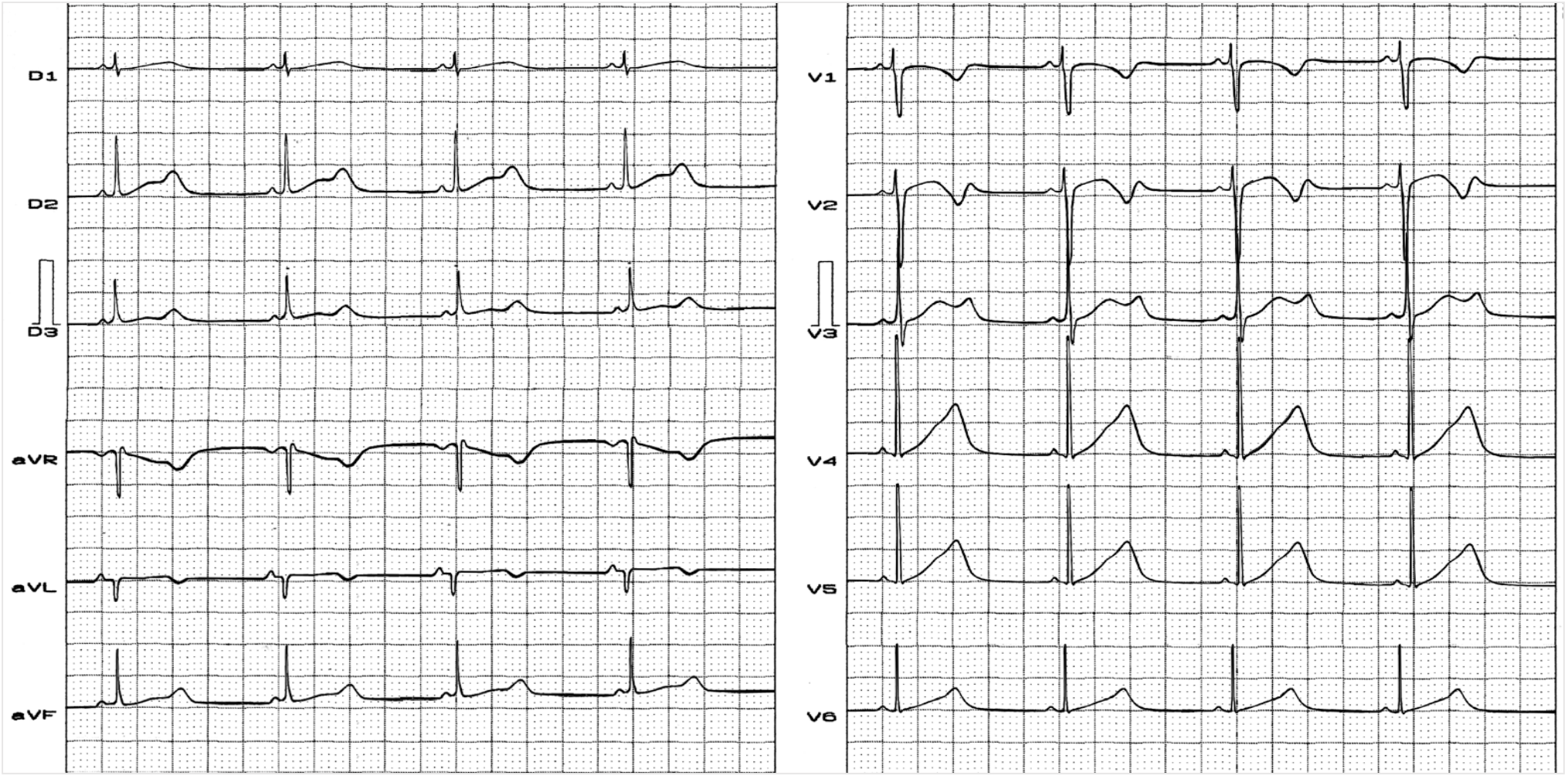
Characteristics of the HERG LQT2 variant (**Lepeschkin E.:The U wave of the electrocardiogram. Mod Concepts Cardiovasc Dis 1969;38:39; Lepeschkin E.: Physiologic basic of the U wave. In Advances in Electrocardiography. Edited by Schlant RC, and Hurst JW. New York, Grune & Stratton 1972;pp 431-447**).

Bimodal T wave (T1-T2 pseudo U-wave dependent on bradyarrhythmic pause). Is this mechanism possible in your case?



T2 voltage wave increases in voltage after pauses (Roden DM, Spooner PM. Inherited long QT syndromes: a paradigm for understanding arrhythmogenesis. J Cardiovasc Electrophysiol. 1999 Dec;10(12):1664-83).

Name: D.S.F; **Age:** 11 years old; **Sex:** Fem. **Weight:** 38 kg; **Height:** 1.45 m; **Race:** white; **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.

Conclusion As the T wave is the surface ECG manifestation of ventricular repolarization, its occurrence without a preceding QRS complex can only mean that the ECG machine has failed to register (Lisa Leung, Banu Evranos, Manav Sohal, Mark M Gallagher. An isolated T wave. European Heart Journal - Case Reports, Volume 1, Issue 2, December 2017, ytx017, https://doi.org/10.1093/ehjcr/ytx017)

T Wave

The T wave represents ventricular repolarization. T waves are normally positive, but negative T waves are normal findings in leads aVR and V_1 (and in young people, in V_2). The causes of pathologic T-wave inversion include myocardial ischemia and infarction, ventricular strain, and treatment with digoxin. Following a myocardial infarction, T-wave inversion develops within 12 to 48 hours and is usually permanent. There is a wide variation in both the duration and the amplitude of the T wave. Flattening T waves are seen with hypokalemia, and peaked T waves are seen with hyperkalemia. The T wave represents the mid-latter part of ventricular repolarization. A normal T wave has an asymmetrical shape; that is, its peak is closer to the end of the wave than to the beginning. When the T wave is positive, it normally rises slowly and then abruptly returns to the baseline. When it is negative, it descends slowly and abruptly rises to the baseline. The *asymmetry* of the normal T wave contrasts with the symmetry of abnormal T waves in certain conditions, such as MI and a high serum potassium level. The exact point at which the ST segment ends and the T wave begins is somewhat arbitrary and usually impossible to pinpoint precisely. However, for clinical purposes accuracy within 40 msec (0.04 sec) is usually acceptable.

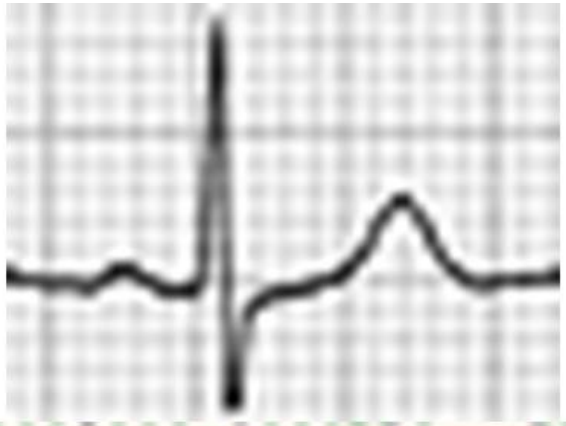
Long and Short QT Syndromes

T Wave Morphology The T wave is often biphasic or notched. These abnormalities are particularly evident in the precordial leads and contribute to the diagnosis of LQTS; they often are more immediately striking than the sheer prolongation of the QT interval. Following cessation of exercise, major repolarization changes often appear, and they are useful for the diagnosis.

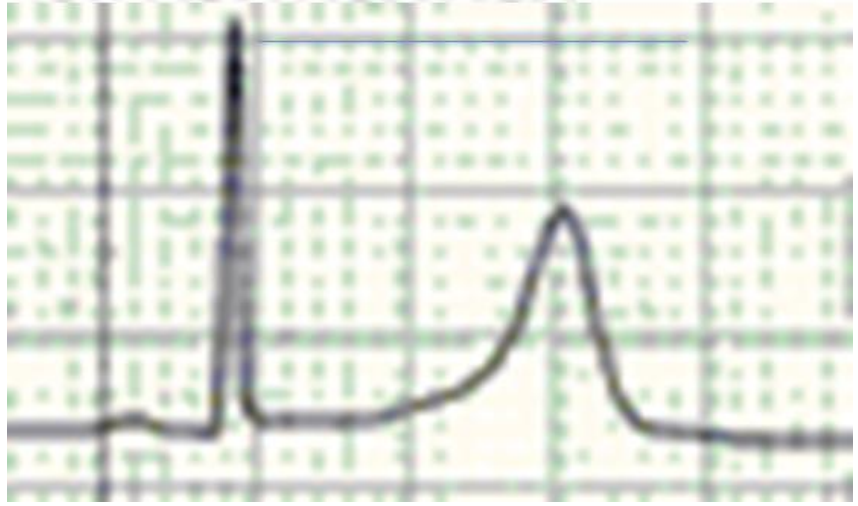
Notched T waves are more frequent in symptomatic patients (81% vs. 19%). They probably reflect the presence of subthreshold early afterdepolarizations (EADs). Their appearance following exercise is markedly more frequent (85% vs. 3%) among LQTS patients than among healthy controls. Among children, notched T waves are not necessarily abnormal.

Gene-specific ECG patterns do exist. LQT1 patients tend to have smooth, broad-based T waves, whereas LQT2 patients frequently have low-amplitude and notched T waves; LQT3 patients have a more distinctive pattern characterized by a late onset of the T wave. However, even within families, extreme heterogeneity of T wave morphology may be observed.

Characteristics of LQT1 variant or kvLQT1 defect



Normal (QT = 390 ms)

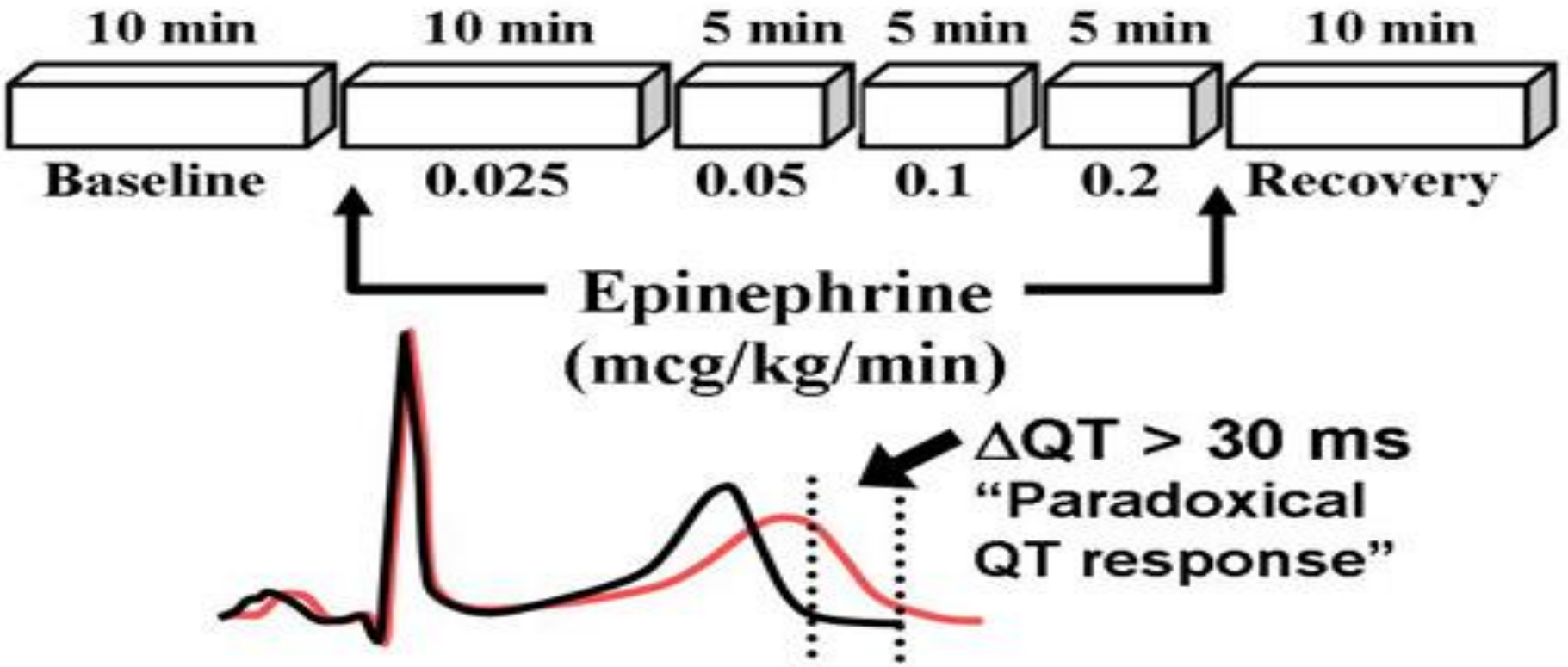


LQT1 60% of total

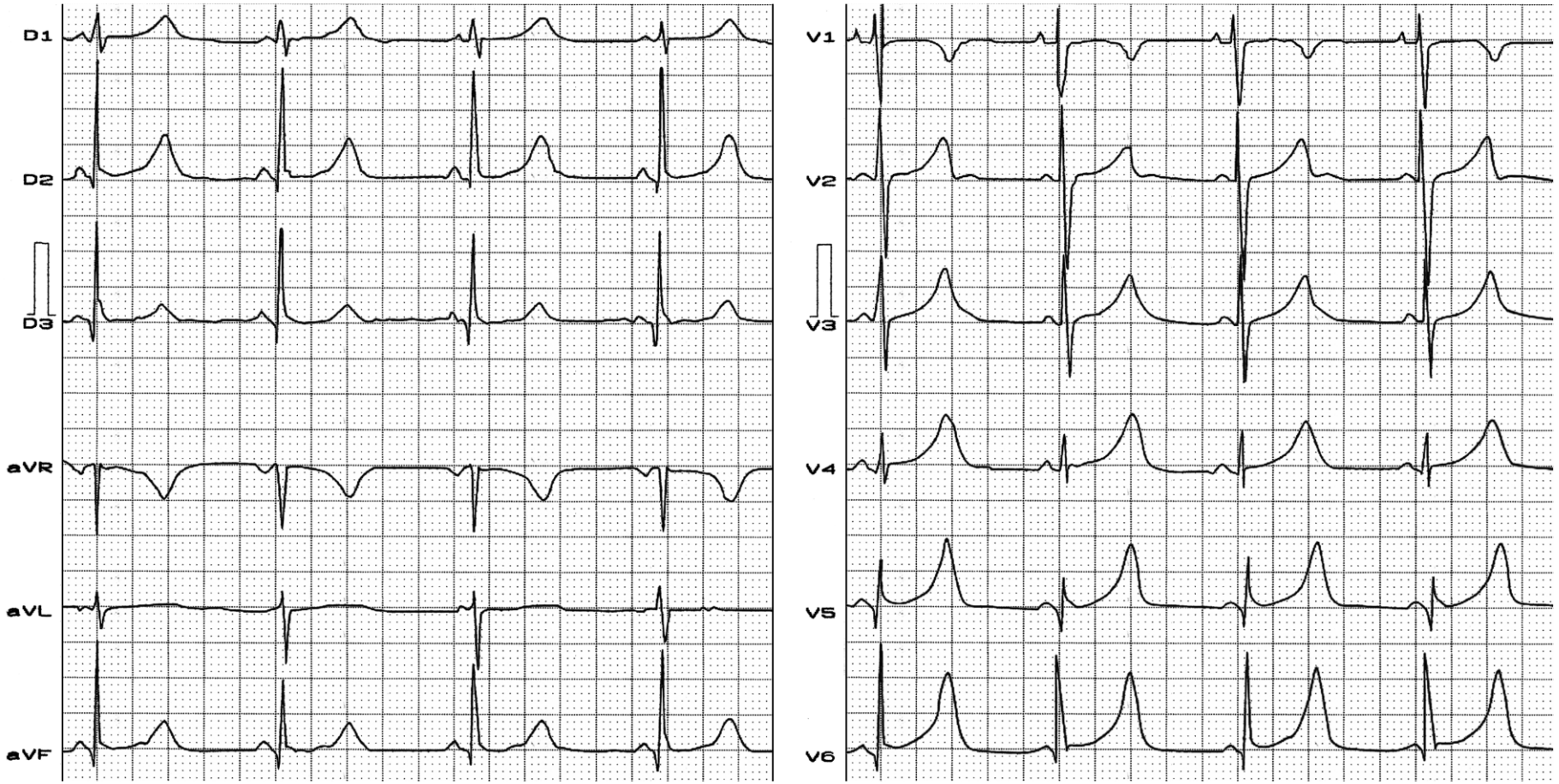
Event triggers: *Exercise, especially swimming*

- Broad-based prolonged T waves (QT = 580 ms).
- Moderate HR dependence of QT interval.
- Short arm of chromosome 11.

- Mutation: 11p15.5.
- Affected channel in TAP: I_{ks} delayed rectifier potassium current.
- Single variant with high % of events during exercise or swimming.

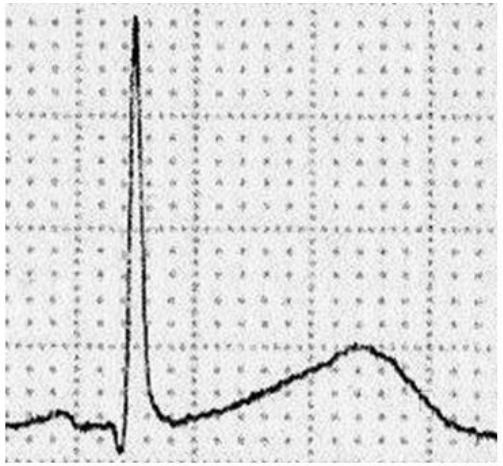


The Mayo Epinephrine QT Stress Test (Mayo Clinic Proceedings 2002) demonstrated that paradoxical lengthening of the absolute QT interval during low-dose epinephrine infusion has 75% positive predictive value and 96% negative predictive value with respect to LQT1. This clinical diagnostic test is now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

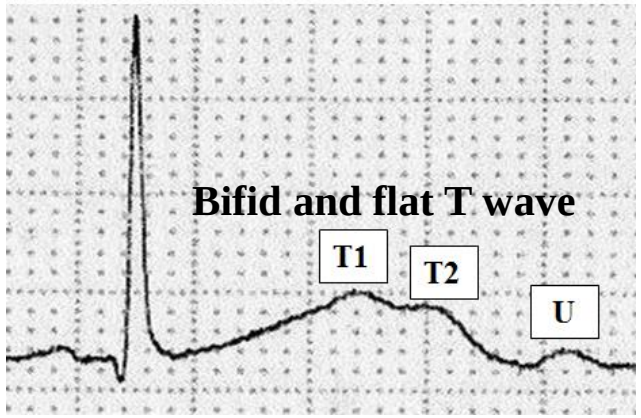


ECG from a patient with LQT1. Typical wide-based T-waves with a large amplitude are observed.

Characteristics of the HERG LQT2 variant



Normal



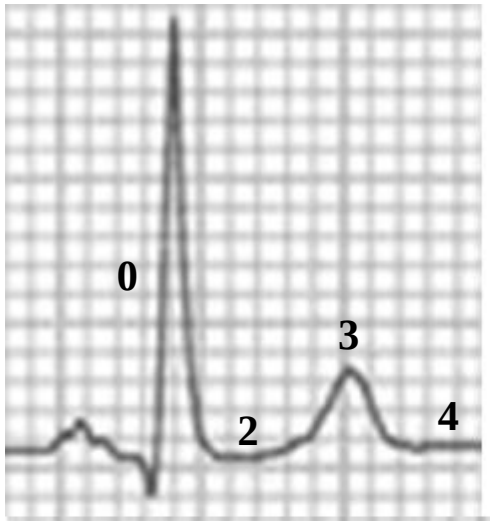
LQT2 ≈35% of the total

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

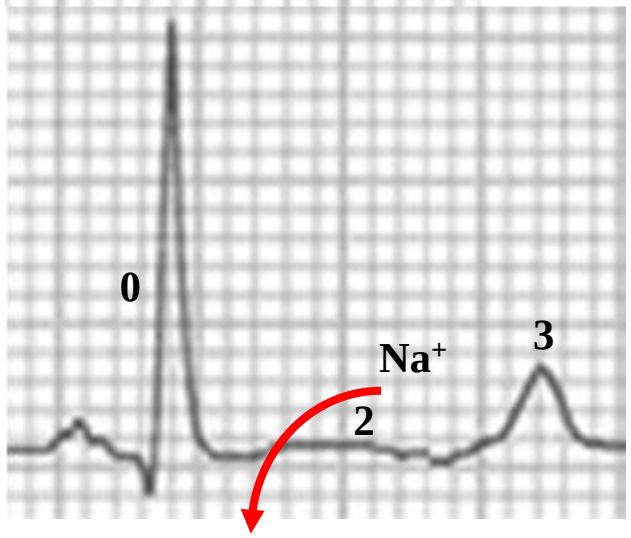
LQT2: OMIM 152437. Mutation: alpha subunit of the rapid delayed rectifier potassium channel (hERG = MiRP1). The current through this channel is known as I_{Kr}. This phenotype is also probably caused by a reduction in the repolarizing current.

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



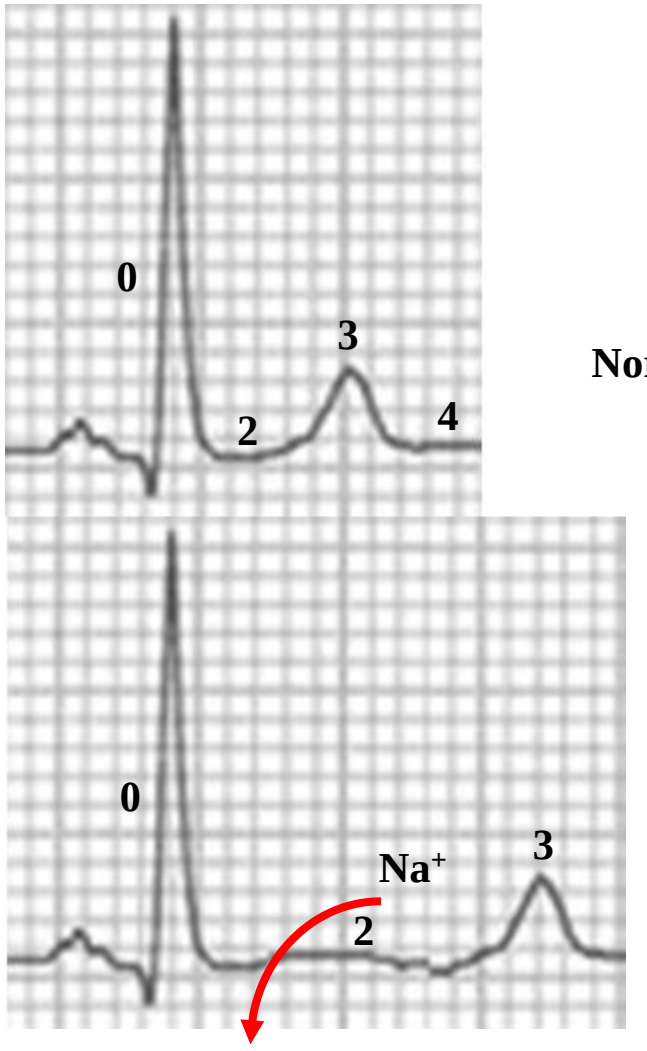
≈ 1% of the total

Delayed appearance of T wave

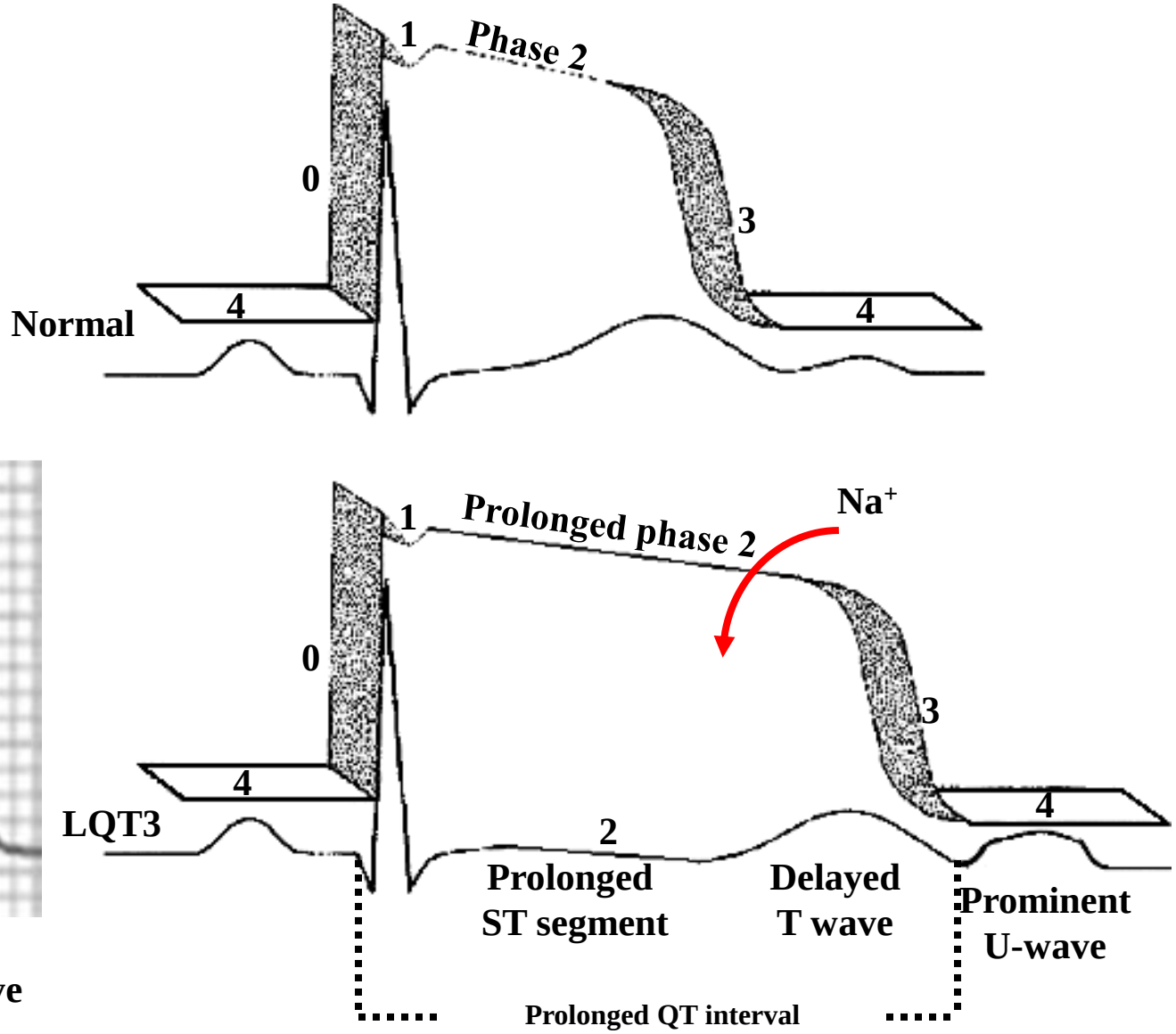
≈ 80% of events during sleeping or at rest

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

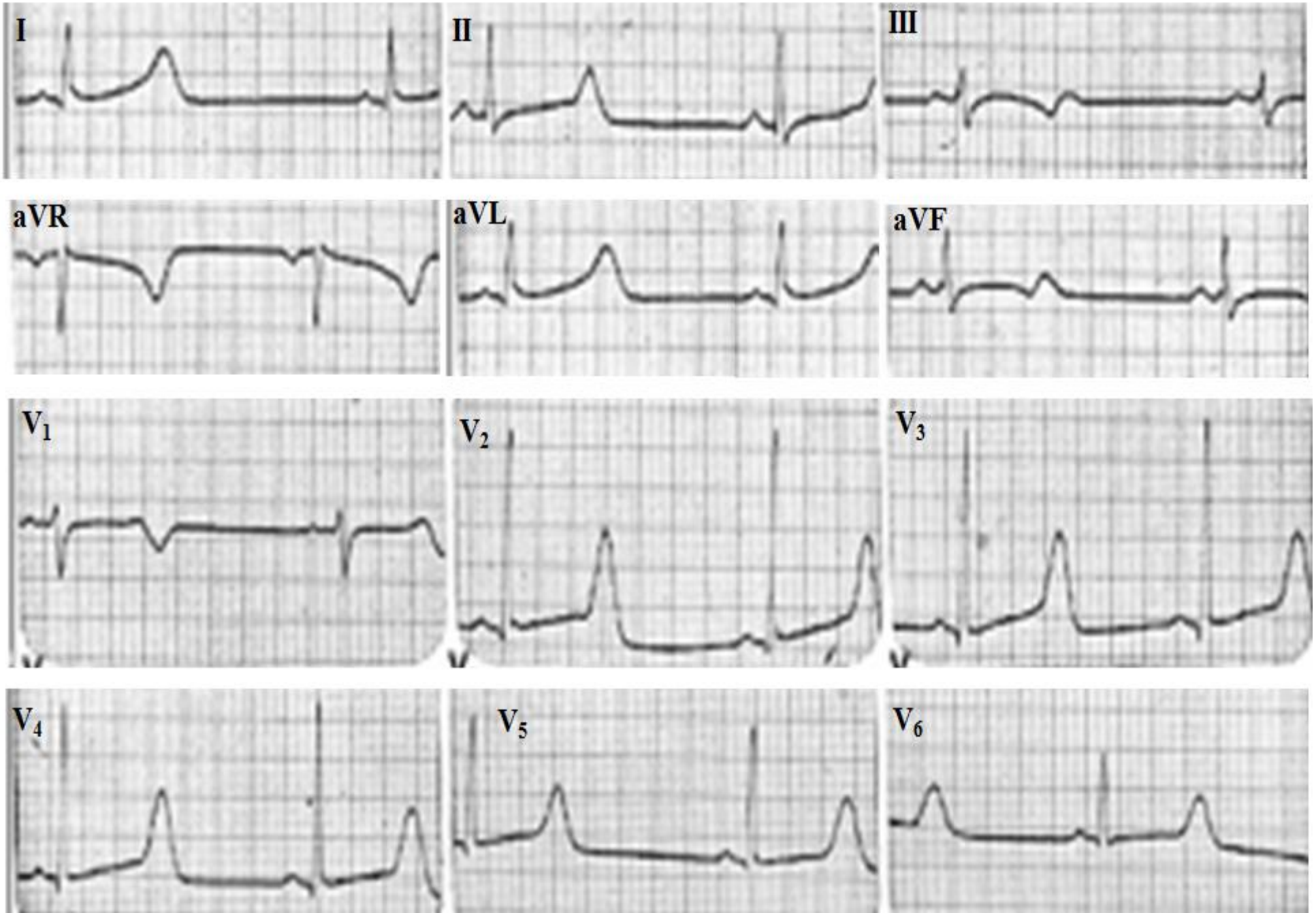


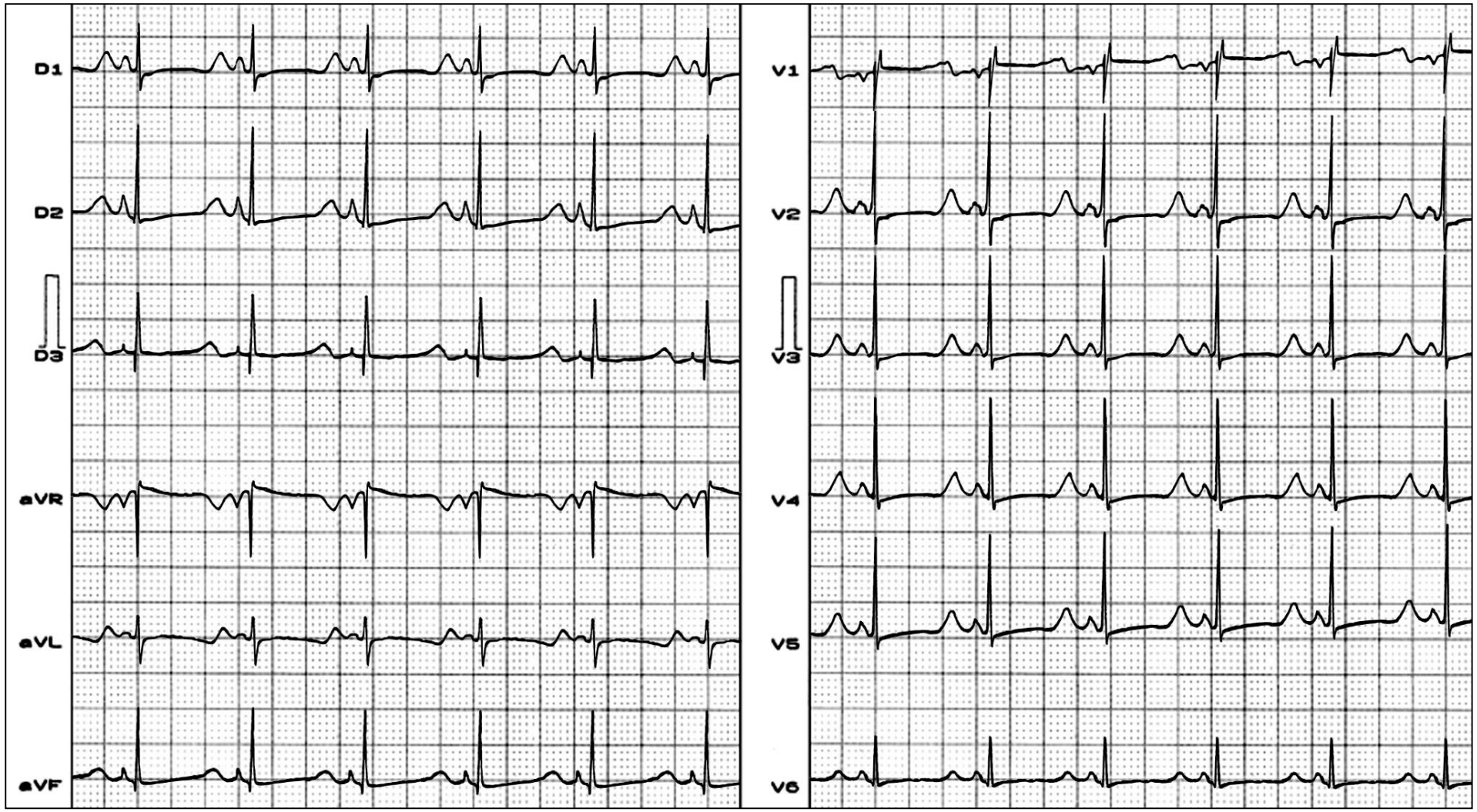
Delayed appearance of T wave



Characteristics of the LQT3 variant, SCN5A mutation

LQT3 ECG

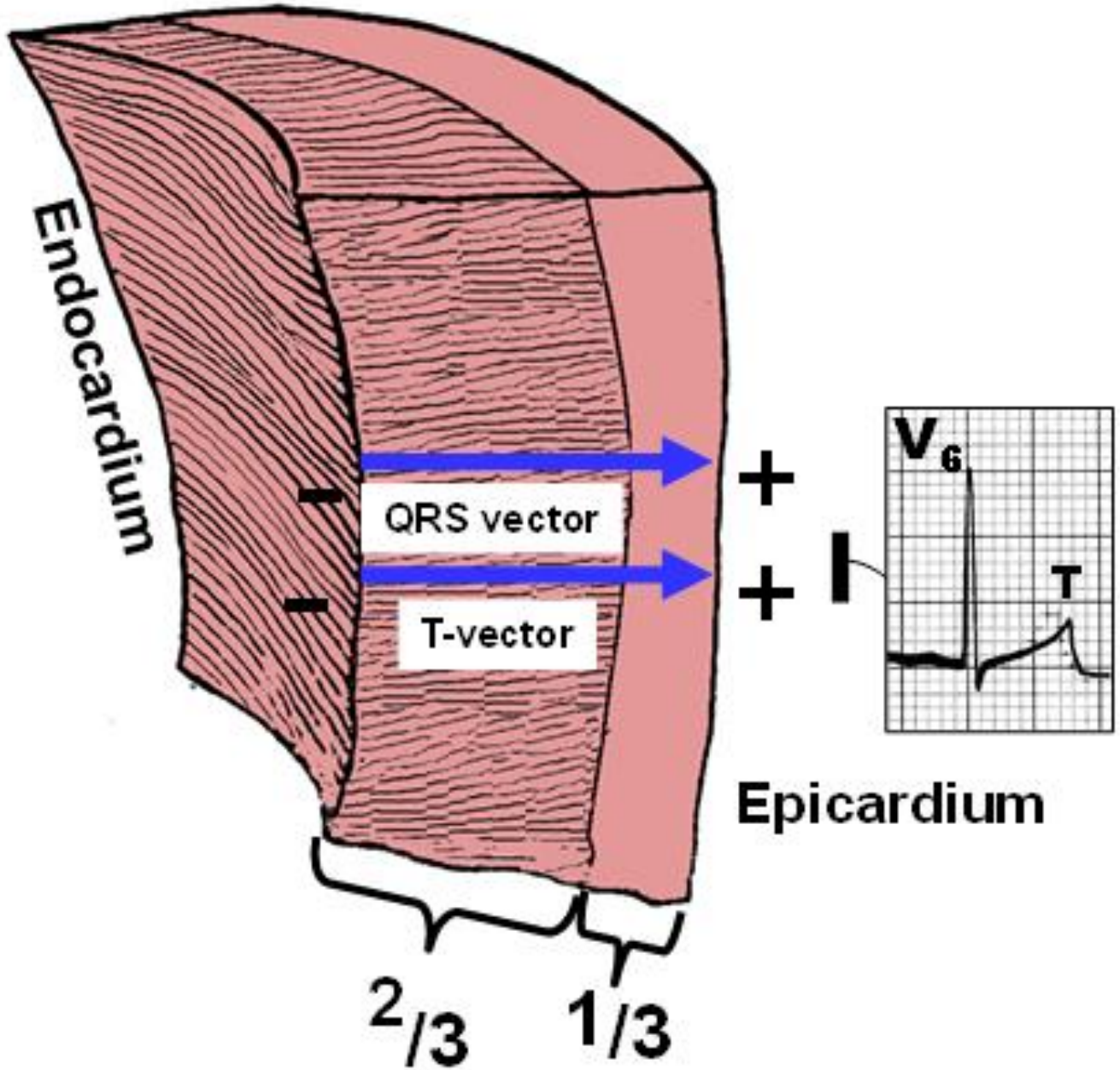




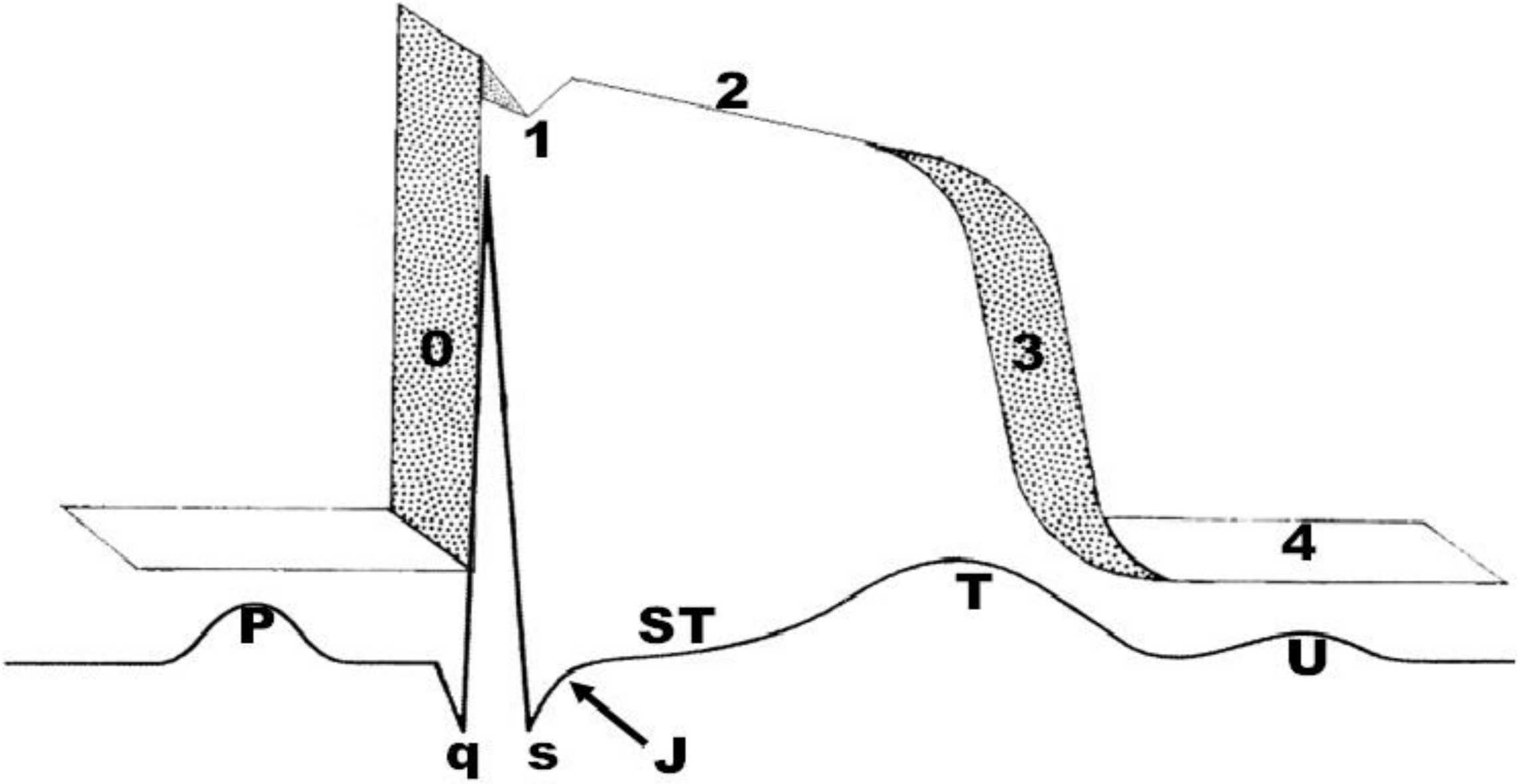
This ECG belongs to a newborn baby with the LQT3 variant. Clear ST segment prolongation and delayed appearance of T wave. Affected gene: SCN5A, p21-24 mutation in chromosome 3, AP phase: plateau, dome or phase 2 by persistent sodium inflow.

Primary T Wave Abnormalities caused by myocardial ischemia or other factors are concordant (i.e., negative T wave in the leads with a dominant S wave or RS pattern). The significance of concordant T wave changes in the leads with a dominant R wave (i.e., upright T wave) is less certain. In the study by Sgarbossa et al. upright T waves in leads V_5 and V_6 had a sensitivity of 26 percent for the diagnosis of acute MI, but it is not clear whether this T wave pattern resulted from MI. Upright T waves in the leads with a dominant R wave (i.e., leads I, aV_L, V_5, V_6) are frequently present in subjects with LBBB without MI or other structural myocardial disease. This may occur when the secondary repolarization change caused by LBBB fails to produce T wave inversion but lowers the amplitude of the upright T wave. It may also occur when the location of the transition zone of the QRS complex differs from that of the T wave. This situation can be recognized when the T wave remains upright in leads V_5 and V_6 but is negative in lead aV_L. In many cases, however, the mechanism of a persistent concordant T wave in the leads with an R wave is unexplained by these mechanisms

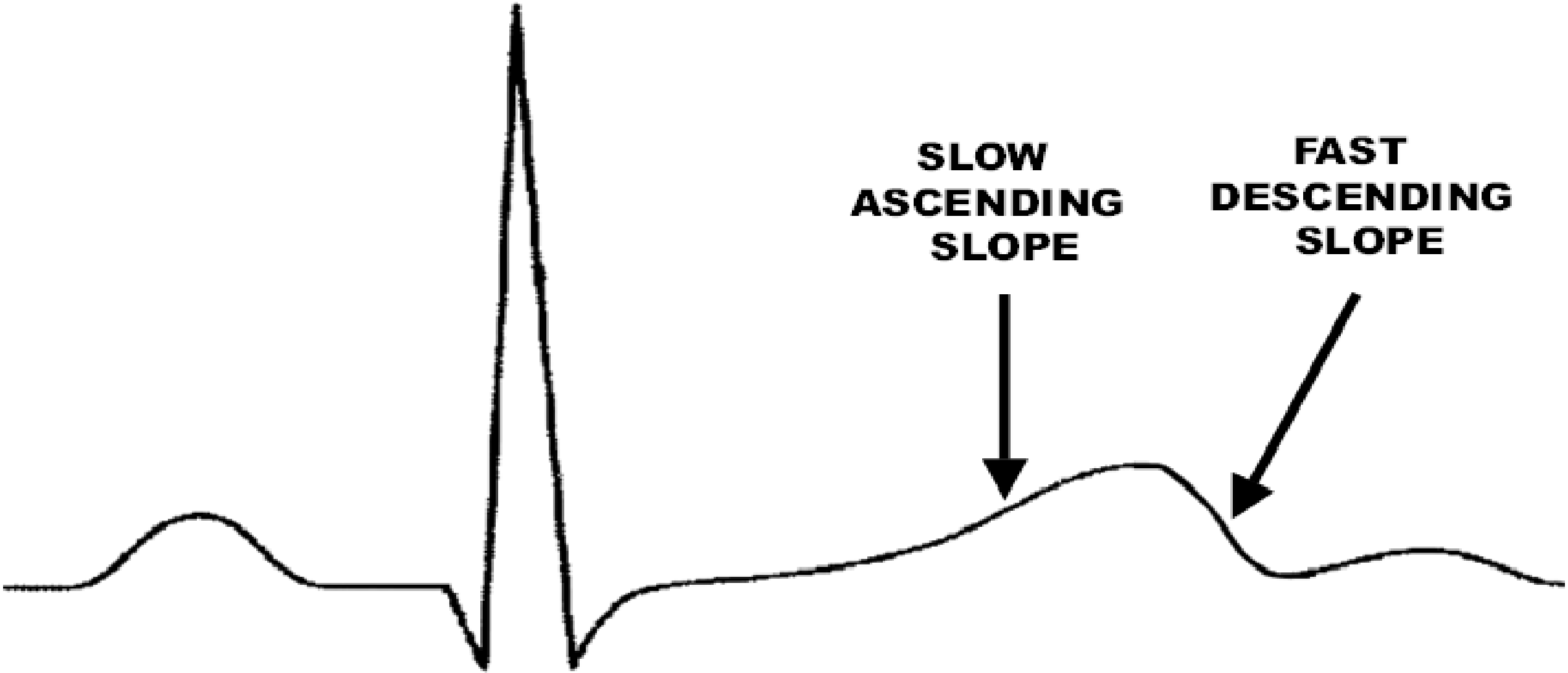
T Wave Abnormalities LBBB and Ventricular Preexcitation Transient T wave abnormalities have been observed for variable periods of time after the disappearance of LBBB or ventricular preexcitation. In patients with preexcitation, T wave abnormalities persisted for days, weeks, and sometimes up to 3 months. The incidence of transient T wave abnormalities and the magnitude of the T wave abnormalities increase with increasing degree of preexcitation (i.e., the duration of the preexcited QRS complex. No T wave abnormalities occurred in patients with concealed AV bypass conducting only in the retrograde direction. The pattern of primary T wave changes produced by ventricular preexcitation depends on the location of the accessory pathway connections. The septal and posterior AP are associated with more prominent anteriorly directed T wave deflections (i.e., increased amplitude of the T wave in the right and mid-precordial leads) and deviation of the T wave vector superiorly (i.e., negative T waves in leads II, III, and aV_F). The left lateral APs are associated with rightward deviation of the T wave in the frontal plane (i.e., T wave inversion or flattening in the left “lateral” leads). The distribution of body surface potentials studied before and 1 day and 1 week after ablation of APs strongly suggests that the transient T wave abnormalities after cessation of preexcitation are caused by AP prolongation over the preexcited area and that substantial recovery of AP duration takes place within 1 week after the ablation. The gradual disappearance of primary T wave abnormalities following normalization of intraventricular conduction has been considered a possible manifestation of repolarization “memory.” This implies that the abnormal sequence of activation is “remembered” for some time after return to a normal activation pattern. Another term applied to describe such a phenomenon is “electrical remodeling.” It is of interest that similar abnormalities have not been recorded in patients with intermittent RBBB.



Representation of ventricular repolarization (vector T), where we observe that depolarization (QRS) and repolarization (T wave) have the same direction, and therefore, similar polarities of QRS and T wave.



Normal profile of T wave with slow ascending ramp and faster descending ramp.


SLOW
ASCENDING
SLOPE
FAST
DESCENDING
SLOPE

Dr. Alan Lindsay:

"A teacher of substance and style"

Distinguished physician and violinist. Argentine doctor Pablo Chiale, a prominent member of the FAC Arrhythmias Committee, world-renowned in cardiac arrhythmias with a long history and a pioneer in electrophysiology, died on September 20, 2014.

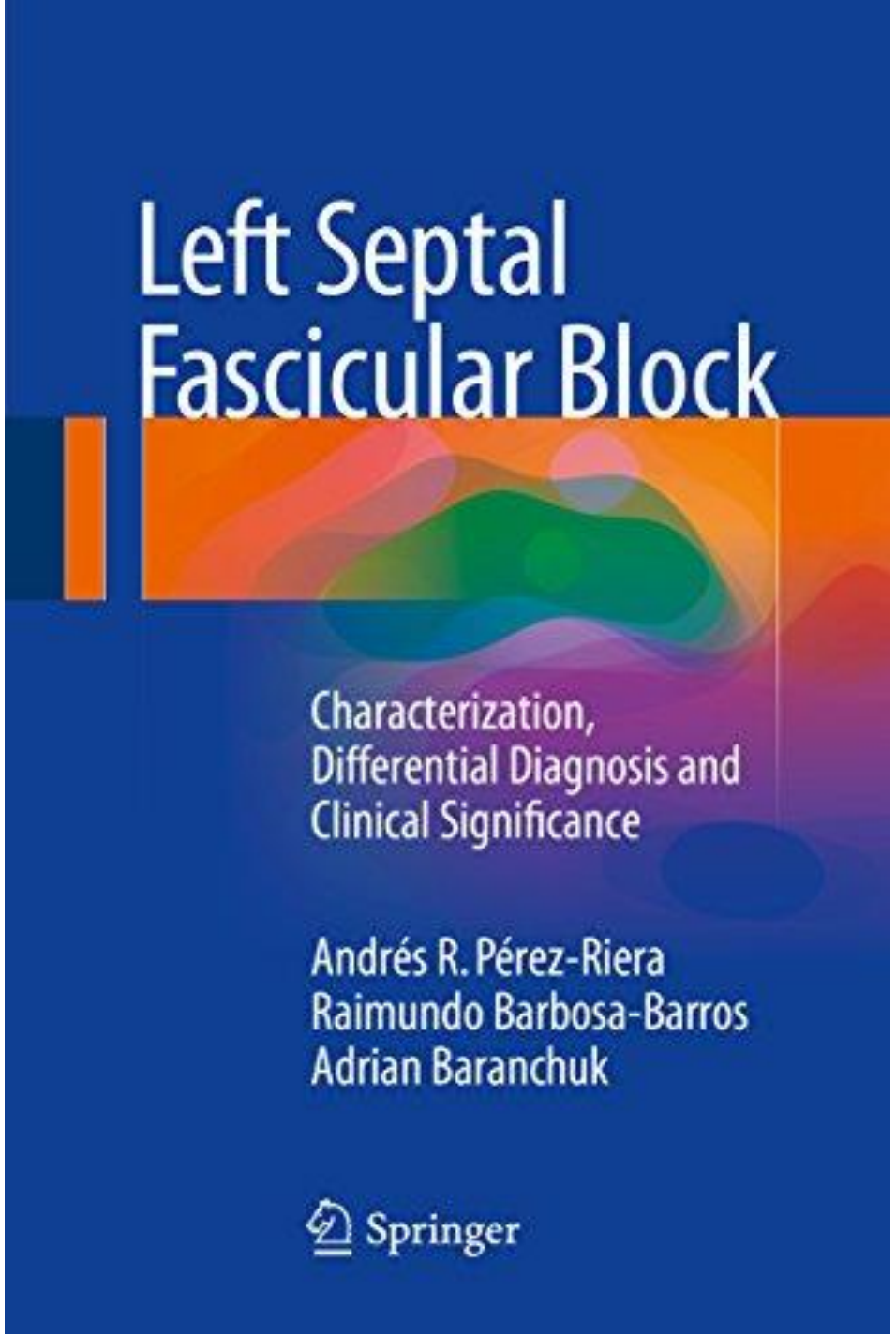
Dedication to Pablo Ambrosio Chiale

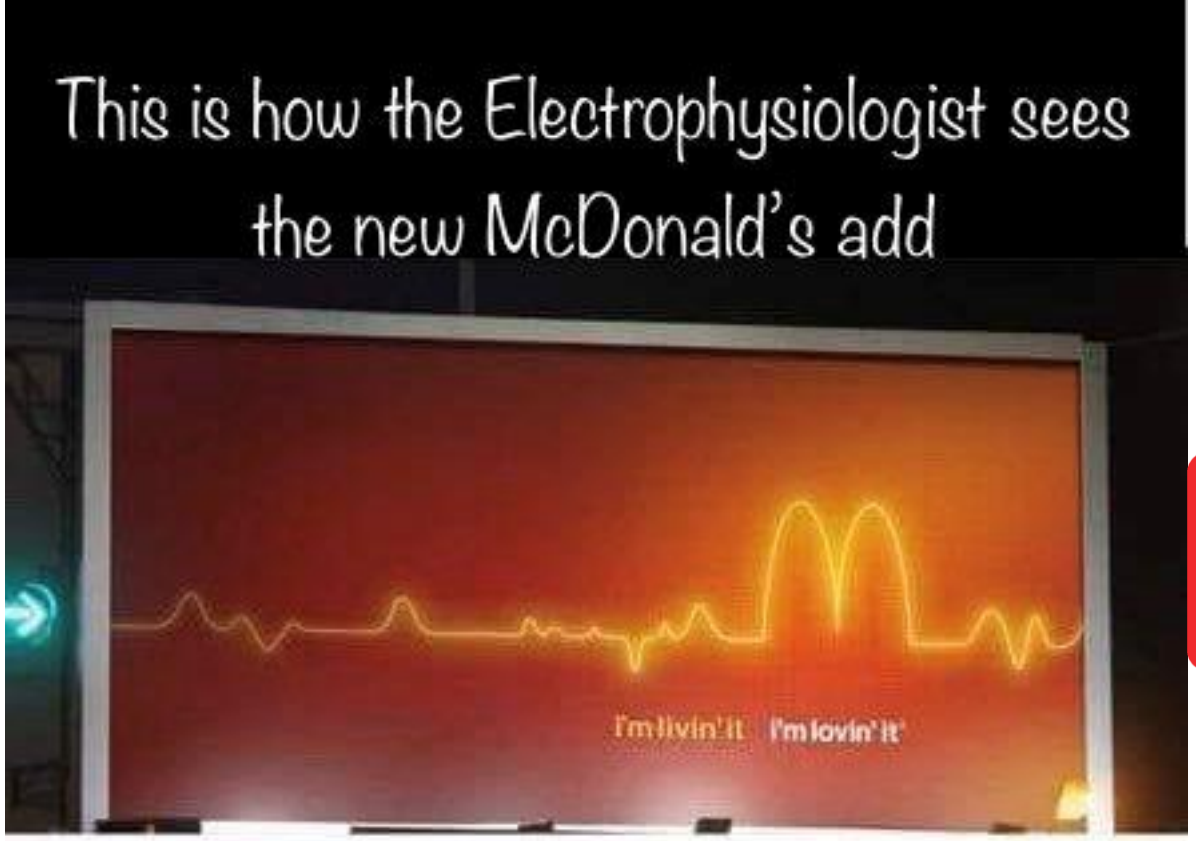
The editors of this book want to dedicate this work to the memory of one of our most brilliant colleagues and an amazing human being. Pablo A. Chiale was one of the most notorious members of the so-called "Rosenbaum's school of Electrocardiology". Dr. Chiale's scientific production is extensive and difficult to quote completely, but he described the electrophysiological properties of accessory pathways, the autoimmune condition of Chagas' disease, the pathophysiology of inappropriate sinus tachycardia, lidocaine-sensitive atrial tachycardias, electrotonic modulation of cardiac repolarization and more recently, a maneuver to unmask Brugada patterns in patients with advanced right bundle branch block; a maneuver that is known today as "Chiale's maneuver".

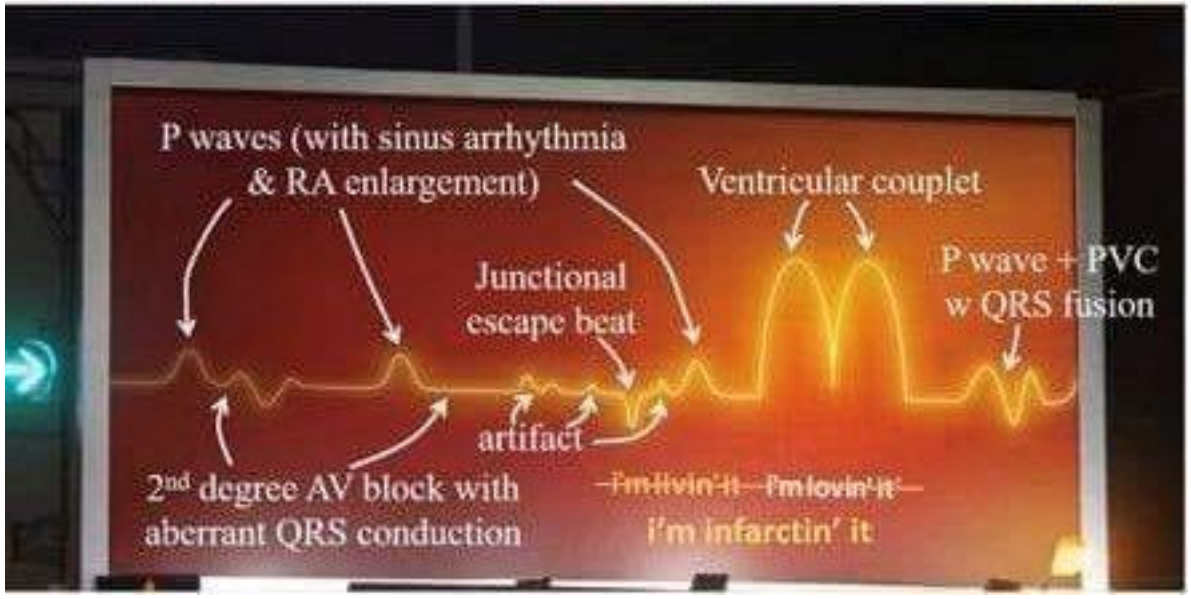
Dr Chiale was a passionate individual who made his life count to the last day. His legacy, undoubtedly, subsists in his disciples around the world.

We had the fortune to learn from Pablo; and this book is dedicated to the "Master" who helped us to navigate through this world full of mysteries and treasures.

The authors: Andrés, Adrián and Raimundo.




This is how the Electrophysiologist sees the new McDonald's add
I'm livin' it
I'm lovin' it


P waves (with sinus arrhythmia & RA enlargement)
Ventricular couplet
Junctional escape beat
P wave + PVC w QRS fusion
artifact
2nd degree AV block with aberrant QRS conduction
I'm livin' it
I'm lovin' it
I'm infarctin' it