Normality that is Abnormal

A case submitted by:

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CEP 05403-000 - Bairro Cerqueira César - São Paulo – SP

O normal que é anormal

Caso enviado por:

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Case report

Female, 52-year-old patient: mesomorph (BMI 23.7 kg/m), asymptomatic, and not taking any medications. ECG was requested for routine presurgery evaluation.

She presented sinus rhythm, heart rate of 57 bpm, morphology of LBBB. In the frontal plane we observed an early beat, narrow QRS, suggesting supraventricular origin (Figure 1).

In the long II tracing (Figure 2), the second beat corresponds to early supraventricular ectopy, followed by pause with a duration of 1280 ms. Next we show two sinus beats with not conduction disorder and with PR and RR intervals, equal to the other sinus beats (PR = 200 ms and RR = 1070 ms). The last five beats present LBBB morphology.

Question

What is the underlying electrophysiological diagnosis of this ECG?

Portuguese

Relato de caso:

Mulher, 52 anos, normolínea (IMC 23,7 kg/m), assintomática e não fazia uso de medicamentos. Foi solicitado ECG para avaliação pré-operatória de rotina.

Apresentava ritmo sinusal, frequência cardíaca de 57 bpm, morfologia de bloqueio do ramo esquerdo (BRE). No plano frontal observamos um batimento precoce, QRS estreito, sugerindo origem supraventricular (Figura 1).

No traçado do II longo (Figura 2), o segundo batimento corresponde à ectopia supraventricular precoce, seguida de pausa com duração de 1280ms. Em seguida notamos dois batimentos sinusais sem distúrbio da condução e com intervalos PR e RR iguais aos demais batimentos sinusais (PR=200ms e RR=1070ms). Os últimos cinco batimentos finais apresentam morfologia de BRE.

Perguntas

Qual o diagnóstico eletrofisiológico subjacente da arritmia?

Figure 1
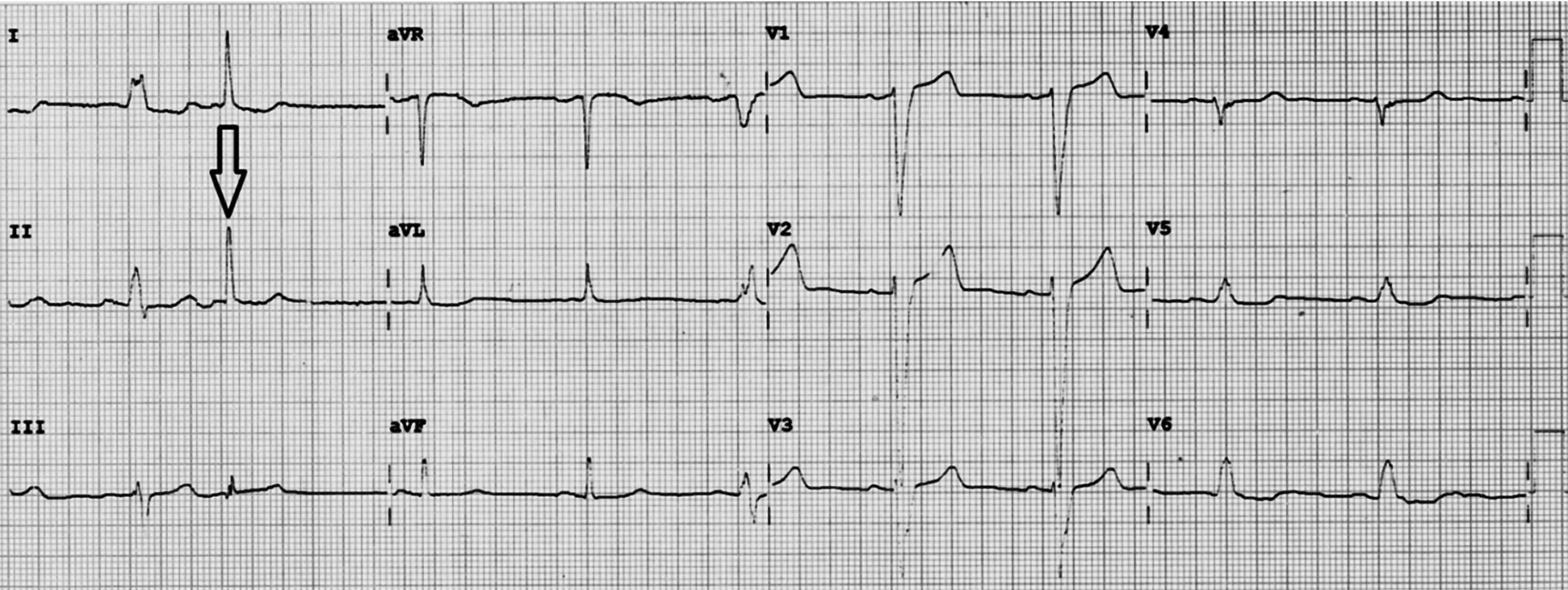
Sinus rhythm, heart rate of 57 bpm, LBBB morphology. In the frontal plane we observe an early beat with narrow QRS(arrow), suggesting supraventricular origin.

Ritmo sinusal, frequência cardíaca de 57 bpm, morfologia de bloqueio do ramo esquerdo (BRE). No plano frontal observamos um batimento precoce com QRS estreito(seta), sugerindo origem supraventricular.

Figure 2
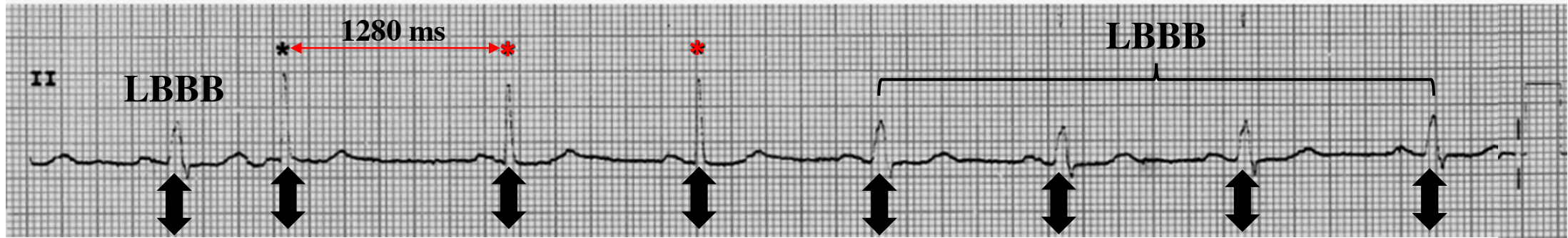
Long II tracing, the second beat (*) corresponds to **APB**, followed by pause with duration of 1280 ms. Next, two sinus beats (*) with no conduction disorder and with PR and RR intervals equal to the other sinus beats (PR = 200 ms and RR = 1070 ms). The last fouth beats present LBBB morphology.

Colleagues opinions

Dear Andres,

This is most likely phase-4 or bradycardia dependent LBBB. Less likely is phase-3 LBBB with supernormal conduction over the left bundle.

 Conduction in the left bundle improves at short a cycle length (640 ms). At cycle lengths between 1040 and 1060, LBBB is observed. There is an overlap of normal QRS complexes at cycle lengths between 1080 and 1280 ms. This overlap can be explained by the linking phenomenon (concealed retrograde conduction through the contralateral bundle, modifying antegrade conduction).

To have a thorough evaluation of conduction over the left bundle at different cycle lengths, you can do vagal maneuvers and exercise to have a wider range of cycle lengths.

Thank you,
Dr Mario Gonzalez MD PhD.
Penn State Hershey Heart and Vascular Institute
500 University Drive
Hershey, PA 17033Tel: 800-243-1455Fax: 717-531-4077

Queridos colegas! El caso presentado muestra un bloqueo de rama izquierda intermitente en fase 3 (periodo refractario prolongado a un poco mas de 1000 mseg). Precozmente, en aproximadamente 600 mseg presenta una fase supernormal de la conducción que surge de la diferencia de potencial entre el musculo cardiaco normal (-90mV) y la repolarizacion incompleta de la rama izquierda (quizás en -60mV). Esto genera potenciales electrotónicos que llevan transitoriamente el potencial de la mencionada rama a la normalidad (cerca de -90mV) lo que suele durar unos 30 a 50 mseg, luego la rama izquierda, al ceder las corrientes electrotónicas vuelve a hipopolarizarse y recién alcanza definitivamente los -90mV después de 1000 mseg. Existe literatura sobre esta situación pero no la tengo al alcance en este momento.

Saludos afectuosos

Gerardo Juan Naun MD Buenos Aires Argentina

English

Dear colleagues! The presented case shows intermittent left bundle branch block in phase 3 (prolonged refractory period, a little beyond 1000 ms). Early, at approximately 600 ms, the patient presents a supernormal phase in conduction, which arises from the difference in potential between the normal cardiac muscle (-90 mV) and the incomplete repolarization of the left bundle branch (maybe -60 mV). This generates electronic potentials that temporarily lead the potential of the mentioned branch to normality (near -90 mV) which usually lasts 30 to 50 ms, then the left bundle branch, when the electronic currents yield, hypopolarizes again and only reaches definitely -90 mV after 1000 ms. There is literature on this situation, but I don't have it close by now.

Warm regards,

Totalmente de acuerdo con la opinión del Dr Nau. Para mostrarlo gráficamente, esta ilustración de un capítulo clásico del cual el Dr Nau es coautor

Cordialmente

Totally agree with the opinion of Dr Nau. To show it graphically, attached this illustration of a classic chapter which Dr. Nau is co-author.
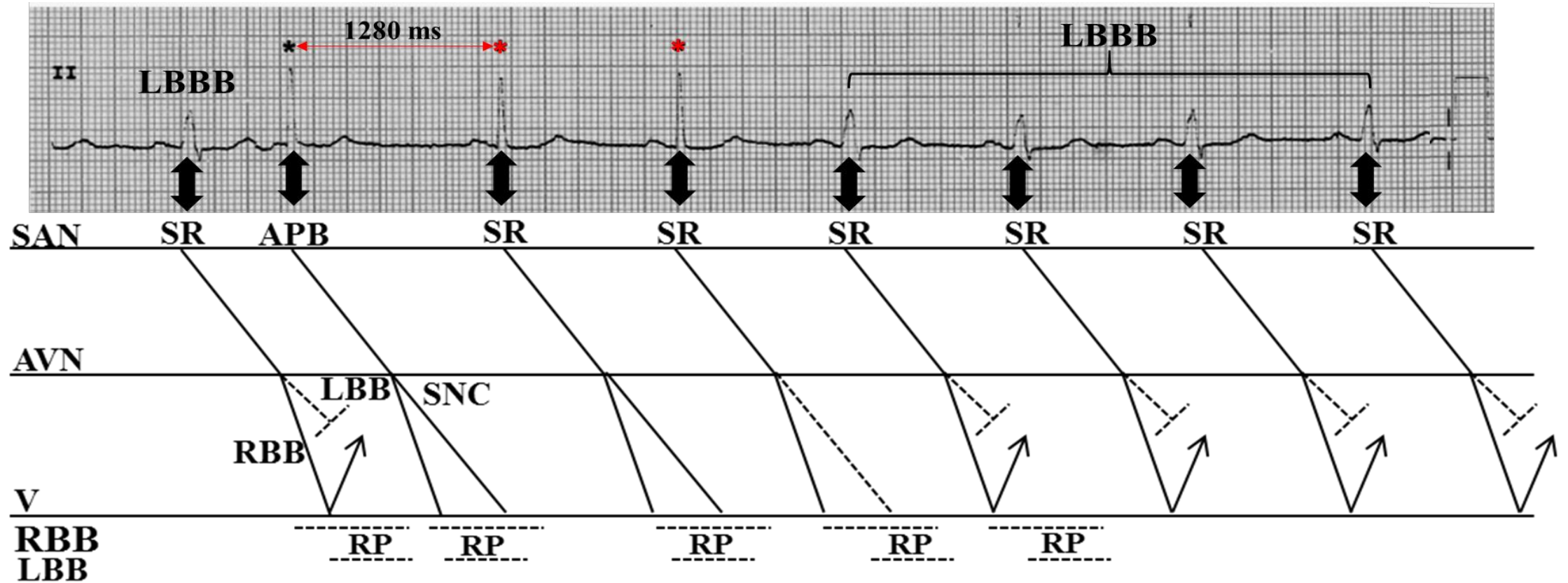
Cordially

Sergio **Pinskis**

PINSKIS@CCF.ORG

Final conclusions in memory of…

Dr. Pablo Ambrosio Chiale, one of the most notorious members of the so-called "Argentinian Rosenbaum's school of Electrocardiology"

**We had the luck to learn from Pablo who helped us to navigate through this world full of mysteries and treasures**

Long II tracing

SAN = Sinoatrial node; AVN = Atrioventricular node; V = Ventricle; SR = Sinus rhythm; RBB = Right bundle branch; LBB = Left bundle branch; RP = Refractory period; SNC = Supernormal conduction; APB = Atrial premature beat

The second beat (*) corresponds to an APB, followed by a pause with duration of 1280 ms. The next two beats are sinus beats (*) with no conduction disorder and with PR and RR intervals equal to the other sinus beats (PR = 200 ms and RR = 1070 ms). The last four beats have LBBB morphology. Supernormal conduction (SNC) period occurs during a small window in the cardiac cycle. Only a very early APB of the second beat interrupts the functional LBBB, thus allowing conduction through both branches. After two sinus beats with normal intraventricular conduction (narrow QRS complexes), there is gradual slowing of conduction through the LBB, which causes the return of the LBBB pattern. The APB necessarily must fall at relatively constant positions within the cardiac cycle close to the end of the T wave (See next slide).

Conclusion: SNC as the underlying mechanism by "cardiac small window" (change in myocardial excitability) in the cardiac cycle.
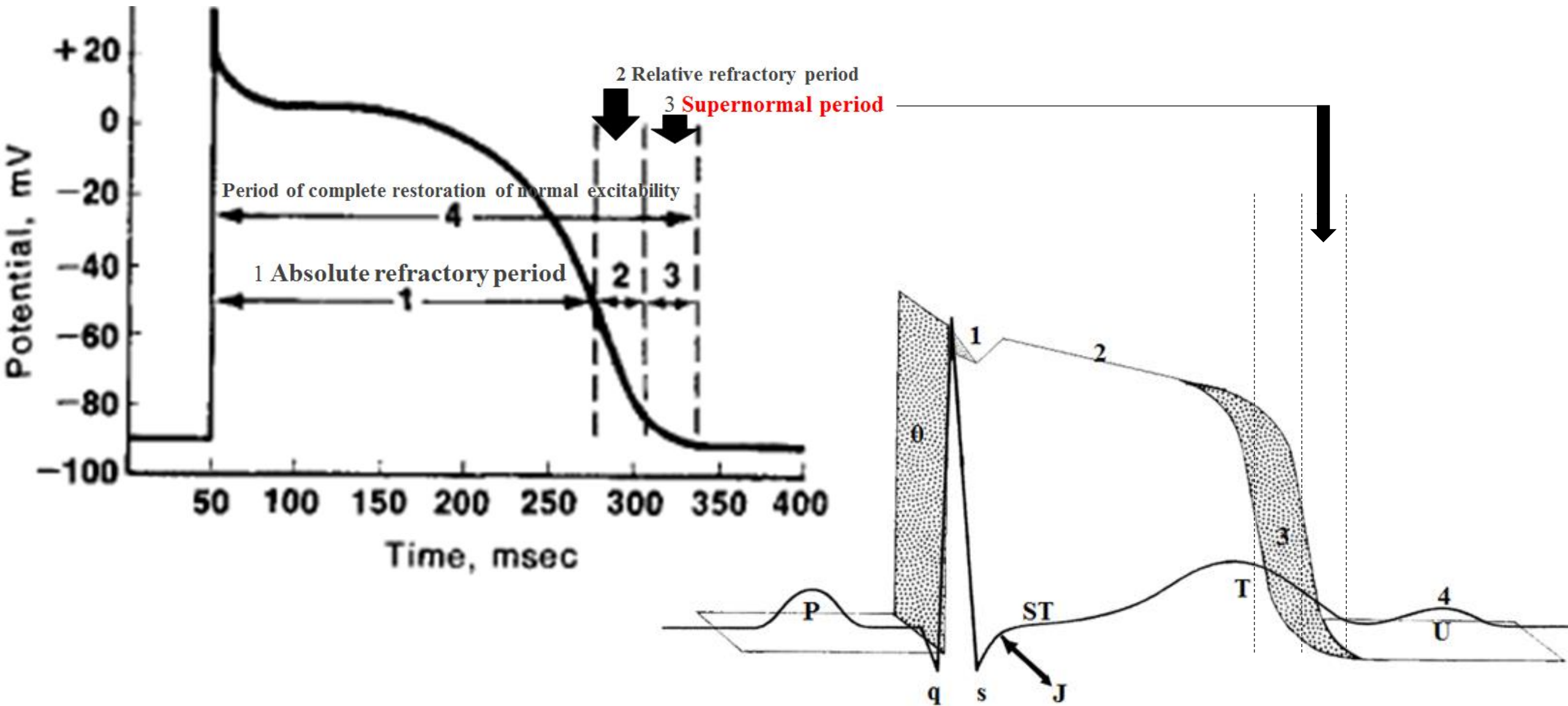
Relationship between action potential and changes in myocardial excitability: 1) Absolute refractory period, (2) Relative refractory period, 3) Supernormal period coincident with the end of T wave and (4) Period of complete restoration of normal excitability.

Introduction

Supernormal conduction (SNC): Electrocardiographic manifestation of "supernormal" conduction is defined as conduction that is more rapid than expected or presence of conduction when block is anticipated. It is not supernormal in the sense of being more rapid than normal. Therefore, the term relative supernormality or "supernormality" is more appropriate. SNC was defined by Pick (**Pick 1962**) as better than expected conduction in patients with depressed conduction during a short interval in the ventricular cycle. It is mainly observed in long-duration ECG assessments.

SNC is much more common than previously thought, particularly in the presence of certain clinical conditions. This phenomenon is not rare in clinical arrhythmology (**Constantini 2016; Rosenbaum1971**), little observed in conventional ECG, which is associated with disorders that affect the conduction system. The facilitation of AV conduction is related to electrophysiological phenomena difficult to understand. Its understanding requires the interpretation of unusual situations and little developed during the evaluation of the electrocardiographic tracing. By reporting this case, we aim to create a reasoning sequence that should be considered facing an ECG with the same characteristics, facilitating interpretation and allowing a more accurate diagnosis. Its occurrence during 12-lead ECG is uncommon and its interpretation demands knowledge on electrophysiological alterations that are hard to understand. The most of the cases of so-called SNC described in humans have been associated with baseline disturbance on the His-Purkinje system or AV accessory pathways (AVAPs). Therefore, the term supernormal has been referred to improved conduction but not conduction that is better than normal. In man, "supernormal" conduction is recorded only in abnormally functioning cardiac tissue with a prolonged refractory period in intraventricular conduction system and/or AVAP (**Constantini 2016**). A prolonged refractory period, appears to be one of the prerequisite requirements for its occurrence, Additionally, occurs at relatively constant position within the cardiac cycle close to the end of the T wave (See next slide). SNC is in relation with the presence of a phase of supernormal excitability experimentally demonstrated in the late phase of repolarization of cardiomyocytes. Additionally, both supernormal and alternating conduction are related phenomena through a pathological bundle branch or an AVAP considered as a marker of the presence of SNC through these structures. Finally, the phenomenon occurs earlier at faster heart rates and later in longer cycle lengths/slower heart rates. Its identification requires the interpretation of rare and little elaborate situations during the ECG tracing assessment. By reporting this case we aim to propose a rationale sequence that should be considered when facing an ECG with these same features, which would enable a greater accuracy to make a definitive diagnosis.

Discussion

AV conduction normalization after atrial premature beat (APB) is an event not often observed in ECG tracings. This improvement in conduction by the His-Purkinje system is called SNC. It does not necessarily mean better than normal, but less abnormal than what is expected for the basal conduction presented.

The possible criteria for SNC are: (I) improvement in beats presenting conduction block or delay, (II) early impulses that conduct better than the previous impulses, (III) QRS narrow beats are proven to be of supraventricular origin, and (IV) the phenomenon cannot be explained by mechanisms other than SNC (**Oreto 1994; Klein 1982**).

The table 1 summarizes the possible mechanisms that may explain SNC and similar phenomena.

In the ECG case presented, a link with bradycardia-dependent block (phase 4) was ruled out, since after the early beat starting the phenomenon, conduction normalization remained for another two cycles, with RR intervals above the basal rhythm intervals.

A link with tachycardia-dependent block (phase 3) When an impulse is conducted to the ventricles beyond 720ms after a QRS complex of RBBB configuration, the impulse falls after the abnormally long effective refractor period of the RBB and passes through the right bundle branch (RBB). When the conducted impulse occurs within 720ms after a QRS complex of RBBB configuration, the impulse usually falls in the refractory period and is blocked in the RBB; however, only when the impulse occurs very early 480ms after that does it fall in the supernormal period and passes through the RBB. I was not observed in the present case. The presence of SNC plays an important role in the initiation of reentrant ventricular tachycardia.

SNC was observed in case of extremely late APBs during phase 3 RBBB (**Luzza 2015**) and in the left bundle branch block (LBBB) revealed by relatively late APBs.

The third hypothesis (worsening in conduction in the unaffected branch, leading to conduction times' equalization in both branches) was also ruled out, since before conduction equalization by the branches, a PR interval prolongation would be expected, which was not observed in this case.

Another explanation would be supraventricular and ventricular conductions fusion, which would lead into a QRS with intermediate morphology (hybrid QRS complex) and a possible AV dissociation. In the ECG, narrow QRS morphology repeats twice, being preceded by sinus rhythm with PR interval maintenance, thus ruling out this hypothesis.

Another theory would be the linking phenomenon. This is characterized by retrograde activation of one of the branches by the contralateral branch (concealed transseptal conduction), preventing anterograde activation of the affected branch during sinus rhythm (**Lehmann 1985**). The conduction aberration in this phenomenon perpetuates after APB. In our case, AV conduction normalization occurs after APB, making this possibility also unlikely.

Finally, during the initial stage of left bundle branch recovery, at the end of the action potential, a small window in the cardiac cycle may lead to a shift from the sick area in the blocked branch, where the tissue has a SNC capacity (the greater the heart rate, the earlier the window for SNC). Because of the short period in this phase, only a very early APB could exceed it and allow conduction through the affected branch. This hypothesis is the one closest to an explanation of the ECG findings.

SNC occurs during delivery of premature extrastimuli such that conduction is unexpected and/or more rapid than anticipated when block is predicted. The supernormal conduction may masquerade as gap phenomenon, but the mechanism is completely different. Supernormal conduction is due to cellular depolarization during a brief period after cellular repolarization following phase 3 of the action potential. The presence of supernormal excitability has been observed in vitro and in whole animal experiments, but its presence as an important clinical phenomenon is disputable, except perhaps, in ischemic myocardium. Instead, what is suspected to be supernormal conduction clinically is likely, in most cases, due to another mechanism such as the gap phenomenon or the peeling back of the refractory period. Other potential explanations for apparent supernormal conduction are summation of subthreshold responses and phasic autonomic influences whereby increased sympathetic tone may be responsible for rapid tissue conduction where block existed previously.

Historical aspects The earliest observation of "supernormal' conduction in a patient with complete heart block was Sir Thomas Lewis (1881-1945). The great master wrote: *"There is a case recently reported from my laboratory, in which an auricular rhythm and a much slower ventricular rhythm interplay and produce almost accurate coupling.... In the whole series of curves, response of the ventricle is invariably to any auricular systole which falls between the summit of T and its end-point, there is no response to an auricular systole falling in any other part of the ventricular cycle. ... In other words, over this phase there has been an overswing in the recovery curve of responsiveness., reminiscent of or identical with the "supernormal" phase of recovery described by Adrian and Lucas"* (*Lewis T 1911;1913*).


Lewis suffered a myocardial infarction at the age of 45 and gave up his 70-cigarette-a-day habit, being one of the first to realize that smoking damaged the blood vessels. He died from coronary heart disease at his home
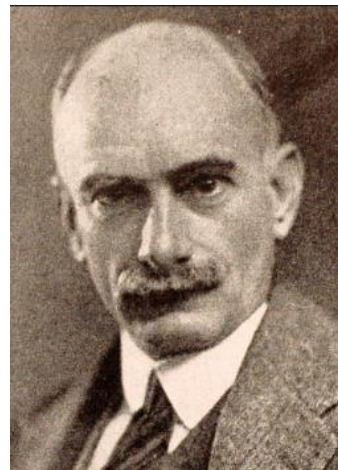
26 December 1881 (Taffs Well, Cardiff, Wales) – 17 March 1945 (Loudwater, Hertfordshire)

Sir James Mackenzie' published tracings that were recorded in 1913 in a patient with right bundle branch block and paroxysmal AV block. Resumption of the sinus rhythm was possibly due to "supernormal" conduction induced by the retrograde conduction of the escape impulse. The relatively frequent clinical reports of purported AV nodal "supernormality" that followed the original description by Lewis contrasted with the relative paucity of observations of "supernormality" of the His-Purkinje tissue. In the clinical setting, "supernormality" of conduction is manifested by better than expected, but not more rapid than normal, conduction, thus, the term relative supenormality or "supernormality" is more appropriate when applied to the ECG. In man, "supernormal" conduction is recorded only in abnormally functioning cardiac tissue. It has been demonstrated in the His-Purkinje fibers, but not in the AV node, the His bundle, or the atrial or ventricular myocardium. In fact, its existence in tissue other than the His-Purkinje is denied by most investigators. The paradox of unexpected improved AV conduction ascribed to "supernormal" AV nodal conduction can be explained by a number of alternate mechanisms such as the "gap" phenomenon, "peeling," or dual AV nodal conduction.

Sir James Mackenzie, MD FRS FRCP
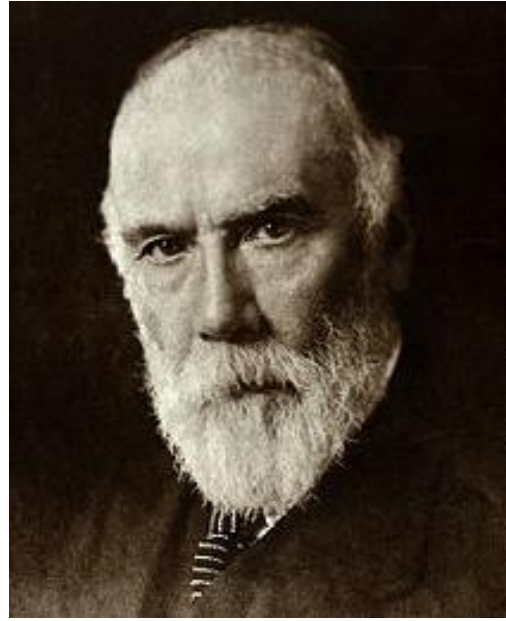
12 April 1853 (Scone, Scotland) – 26 January 1925 (London, England)

Example of Supernormal conduction
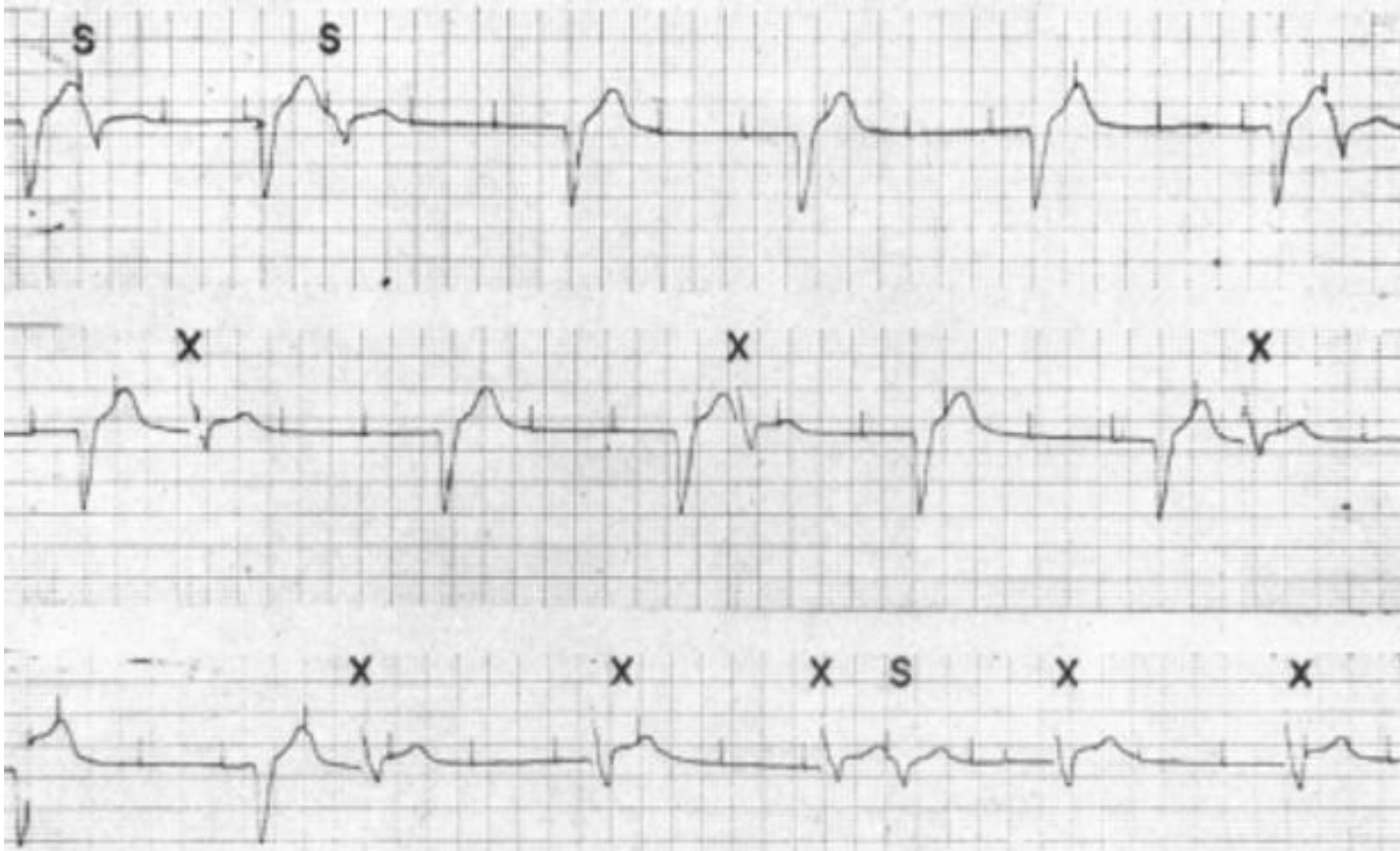
Supernormal phase of excitability exposed by intracardiac stimulation in a patient with apparent complete heart block. The top strip, obtained during idioventricular bearing, shows ventricular responses following only those stimulus artefacts occurring during the final portion of the T wave or slightly after. Stimuli of high intensity (X) were applied in the middle strip. No after-effects were noted. The lower' strip shows how subthreshold responses become supra threshold during the supernormal phase (terminal portions of the T wave) of the "strong" stimuli.

Table 1

Supernormal conduction mechanisms
• Resolution of bradycardia-dependent block (phase 4 block)
• Tachycardia-dependent right bundle-branch block (RBBB) associated with SNC (Katoh 2000)
• Worsening in conduction in the unaffected branch, leading to conduction times' equalization in both branches
• Fusion beat (Gomes 1975)
• Linking phenomenon (Luzza 2015a,b)
• SNC window (Lehmann 1985)
Similar phenomena or pseudo supernormal conduction
• Wedensky effect (Oreto 1892)
• The Gap phenomenon (Wu 1974)
• The Wenckebach phenomenon in the bundle branches permitting normalization of aberrant intraventricular conduction (Gallagher 1973)
• Summation of sub threshold impulses (Lukas 1989)
• Dual atrioventricular nodal pathways causing longitudinal dissociation of the AV node (Moe 1968: Denes 1975)
• Longitudinal dissociation in the RBB (Walston 1976)
• Peeling back refractoriness (Suzuki 1989)
• The shortening of refractoriness by changing the preceding cycle length (Denker 1984)
• Pulsatile changes in vagal discharge (Moe 1968; Jedlicka 1987)
• Facilitation of conduction by ectopic beats (Gallagher 1973)
• Bilateral bundle branch block (Childers 1978)
• Ventriculophasic arrhythmia (Moe 1968; Childers 1978)

The phenomenon of the "gap in atrioventricular conduction"

The gap phenomenon, sometimes confused with supernormal conduction, was first described in 1965 by Moe in dogs and later by Ahktar in humans as a condition in which premature impulses fail to conduct but conduction resumes with even earlier premature extrastimuli. The mechanism has been well studied and is related to the inherent electrophysiological properties of conduction tissue responsible for functional and effective refractory periods of the tissues involved. The effective refractory period is the longest premature coupling interval during fixed rate pacing that fails to activate tissue. The functional refractory period is the shortest coupling interval that can result in conduction after delivery of premature extrastimuli during a fixed rate pacing.

The gap phenomenon occurs when the functional refractory period of tissue proximal in the conducting system is shorter than the effective refractory period of distal conducting tissue. The functional refractory period of proximal conducting tissue occurs at longer coupling intervals than the effective refractory period of the same tissue. During delivery of progressively more premature extrastimuli, block first occurs in the distal conduction system, but with progressive prematurity, conduction will resume in the distal tissue due to proximal conduction delay. The conduction delay that occurs in proximal tissue must exceed the refractory period of the distal tissue for conduction to be restored. We show an gap phenomenon is an experimental (**Moe 1968**) and clinical (**Gallagher 1973**) alternative explanation for ECG abnormalities suggestive of SNC. The gap phenomenon could be abolished by decreasing the basic cycle length or by β-receptor blockade, both of which prevented conduction block of A_2 in the His-Purkinje system. Several types of gap phenomenon during anterograde conduction have been described:

I. **Type 1** has a proximal site of block at the AV node and the distal site of block is the His-Purkinje system (HPS): Previous reports have demonstrated that while relatively late atrial premature beats (APBs) are blocked within the His-Purkinje system, earlier APBs may successfully propagate to the ventricle if they encounter sufficient A-V nodal delay to allow recovery of the distal area of refractoriness (Type I "gap").

II. **Type 2** has a proximal site of block at the HPS (proximal) and a distal site of block at the HPS (distal): is due to delay within the HPS (Type II "gap"). Relatively late APBs were noted to block within the HPS, similar to the findings in Type I. In Type II, however, when earlier premature atrial impulses encountered delay in a relatively proximal area of the His-Purkinje system, allowing more complete recovery of the distal area of refractoriness. Both types of gap phenomena represent examples of apparent supernormal conduction (SNC) (**Gallanger 1973**).

III. **Type 3** has a proximal site of block at the His bundle and distal site of block at the HPS.

IV. **Type 4** has a proximal site of block in the atrium and a distal site of block at the HPS or the AV node.

V. **Type 5** gap phenomenon has a proximal site of block at the AV node (proximal) and a distal site of block at the AV node (distal).

VI. **Type 6** gap phenomenon, is similar to type 2 gap phenomenon, there is conduction at a proximal site and block at a distal site. This differs from type 2 as there is no conduction delay or block noted at the proximal site.

The Wedensky effect

Wedensky effect is defined as a "stronger" stimulus, where in the case of AV block, a ventricular premature beat or paced beat, is followed by transient anterograde conduction by decreasing the refractoriness of the AV conduction (**Schamroth 1971**). Its existence in humans remains controversial (**Engel 1977**). Wedensky phenomenon consists in proximal delay of the AV conductions system with recovery of the distal portion. Obviously, the gap phenomenon is an unlikely mechanism in our case. The phenomenon of the "gap in AV conduction" was studied in human hearts by Wit et al (**Wit 1970**), using a catheter technique for recording electrical activity of the His bundle. The authors applied premature atrial stimuli throughout the basic atrial cycle, either during sinus rhythm or atrial pacing. As the coupling interval between the basic (A_1) and premature (A_2) atrial depolarizations was decreased, a point was reached where A_2 was no longer conducted to the ventricles. The region of conduction block was located distal to the His bundle. The interval between basic and premature His bundle depolarizations at which block occurred provided a value for the effective refractory period of the HPS. If A_2 was then made to occur earlier in the basic cycle, a point was reached where conduction of the premature response to the ventricles resumed. When this occurred, conduction delay of A_2 in the AV-N had increased sufficiently to allow for recovery of excitability of the His-Purkinje system (the interval between successive His bundle depolarizations was greater than the effective refractory period of the HPS).

Wedensky effect example
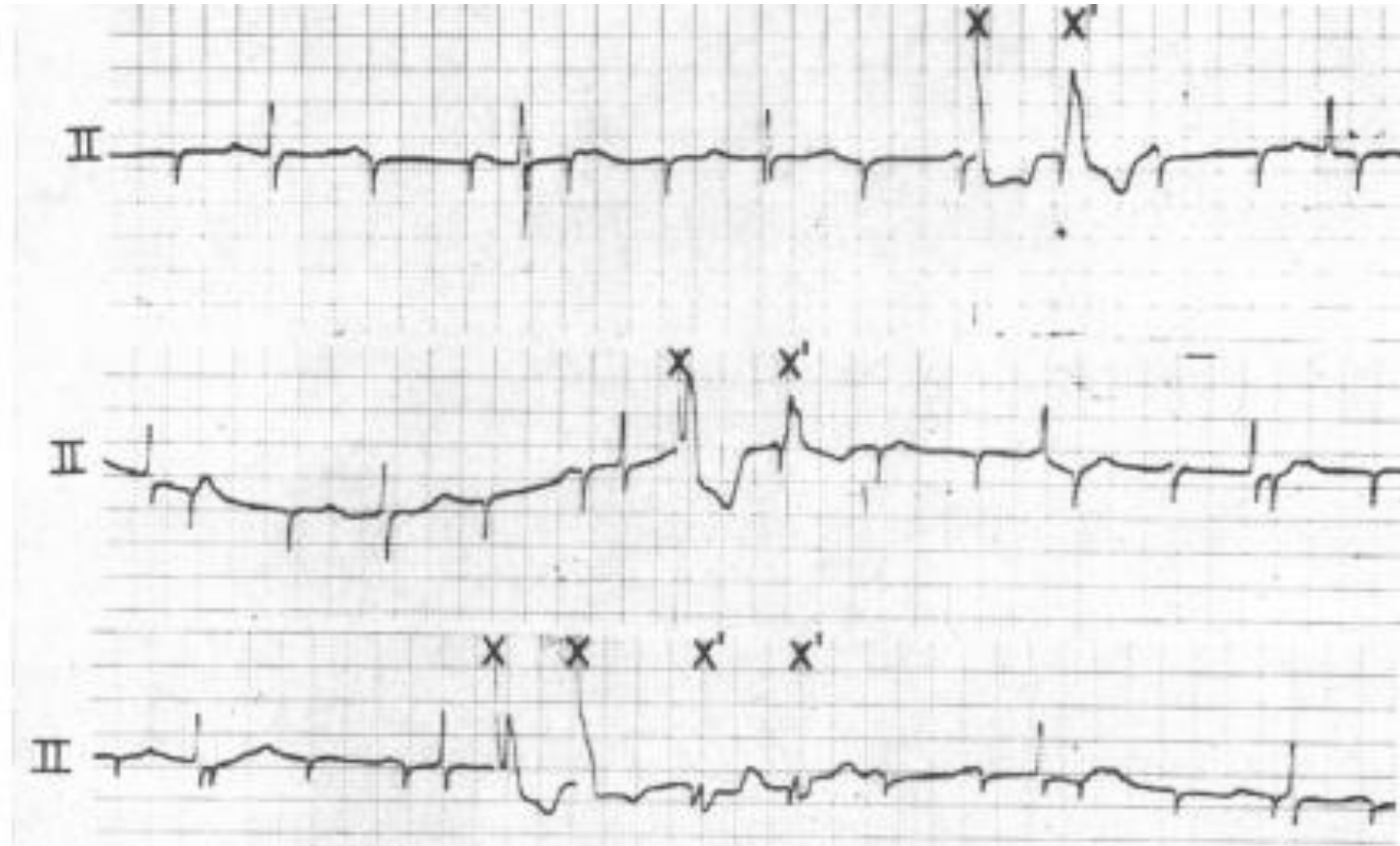
Wedensky effect in the human heart. The upward deflections are the spontaneous QRS complexes, presumably of A-V nodal origin. The downward-directed spikes are the subthreshold stimulus artefacts from the artificial pacemaker. Note that stimuli that are seven times above 'threshold (marked with an X) and that do not occur in the vulnerable phase of natural beats, are able to evoke responses (X') from previously subthreshold stimuli. These responses occur well after the end of the T wave of the strong shock, hence ruling out vulnerability and supernormality as the underlying mechanisms.

Peel back effect or peeling back refractoriness

Anterograde concealed conduction into the concealed AVAP has been postulated to be one of the factors preventing the reciprocating process via the AP in patients with the concealed Wolff-Parkinson-White (WPW) syndrome. It is assumed that pre-excitation of the AV node by a ventricular or junctional beat shortens the absolute refractory period of the AV or the His-Purkinje system and allows conduction of a supraventricular impulse (**Luzza 2015**).

The AV simultaneous pacing frequently shortens the refractoriness of the concealed AVAP and such facilitation seems to be well explained by the probable anterograde concealment in it and peeling back of the refractory barrier (**Suzuki 1989**).

Lee et al (**Lee 1997**) observed this mechanism using the combination of low dose of sotalol and a class IA agent greatly prolongs refractoriness. The magnitude of the effect increases at shorter coupling intervals. The peeling back effect is due to pre-excitation of the AV node by a ventricular or junctional beat that shortens the absolute refractory period of the AV node or the HPS and allows conduction of a supraventricular impulse.
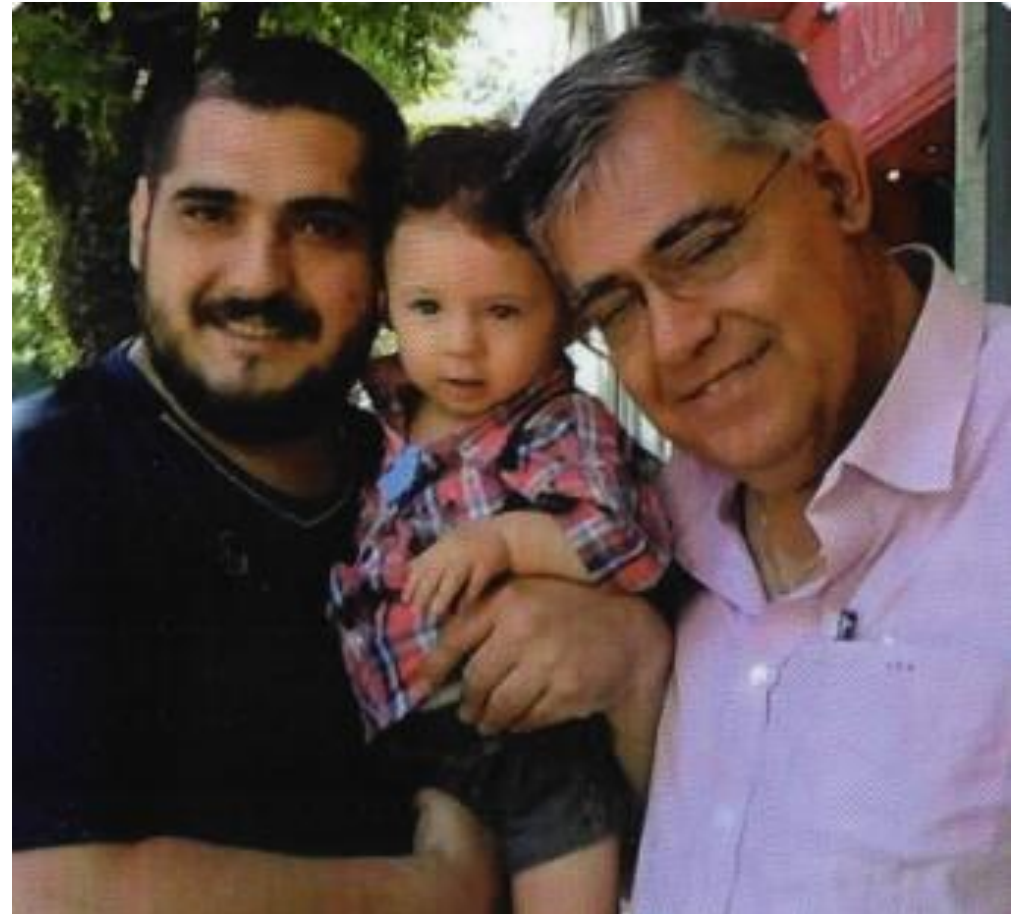
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Thank you for your attention

Three generations