

# Trastorno de conducción intraventricular evolutivo muy inusual

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## Case report

Senior, Caucasian, 77-year-old, retired man. He was known to be a carrier of longstanding mitral-aortic rheumatic valvular heart disease.

He complained of dyspnea on moderate effort (NYHAI) since several months ago.

A week ago he had one syncopal episode, not associated with exercise that repeated at rest yesterday. He denied having angina pectoris precipitated by exertion or at rest.

### **Physical examination:**

**Inspection:** Jugular Venous Pulse (JVP) assessed with the patient reclined at a 45° angle, 3.5 centimeters above the sternal angle (normal <4 cm).

**Palpation:** Point of maximum impulse is better felt 1 cm at the left of the mid-clavicular line, roughly at the 6th intercostal space with increased vigor. Absence of thrills. BP 135/85 bpm. HR 60 bpm and regular.

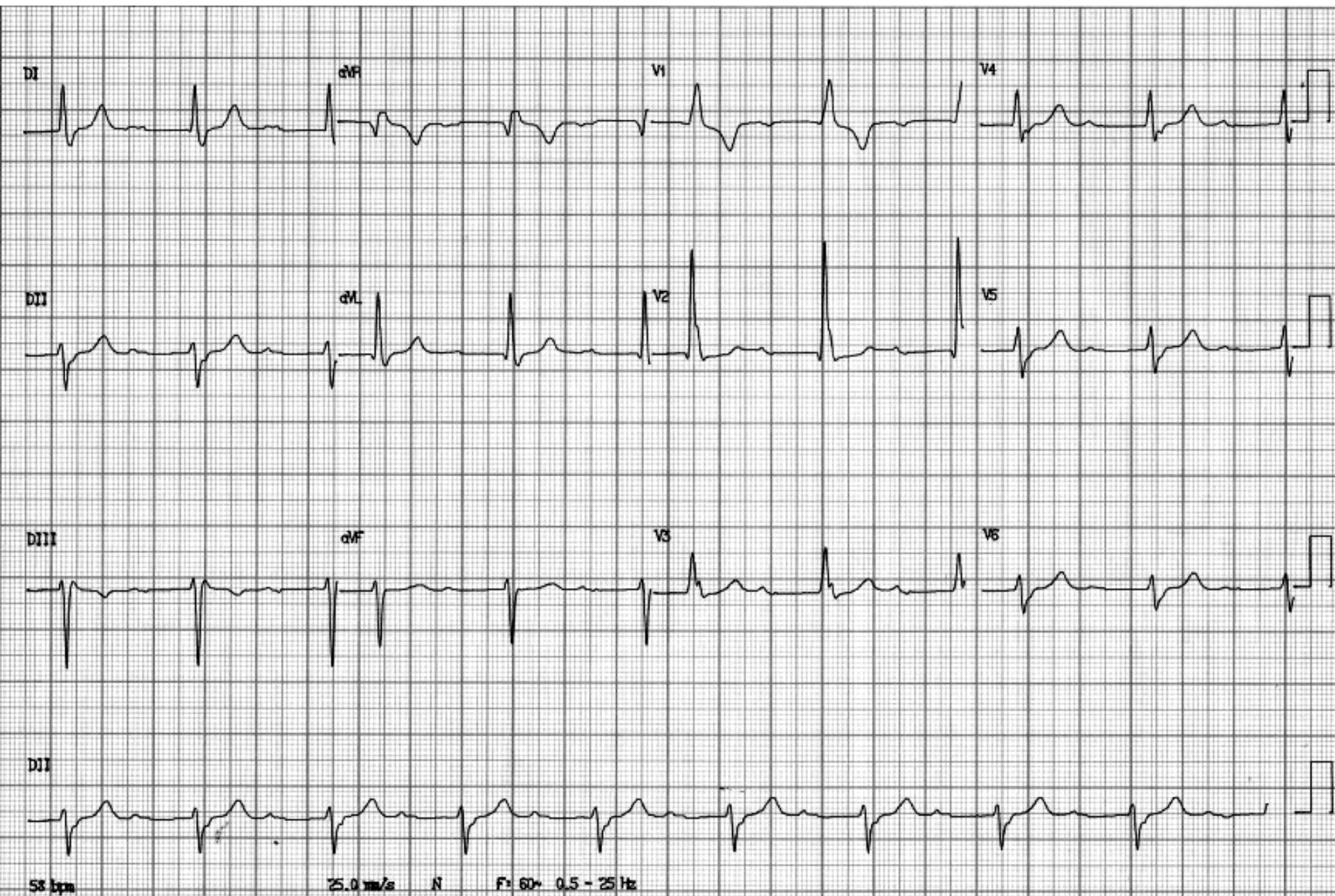
**Auscultation:** In mitral area, S1 with decreased intensity; a soft, low-pitched sound occurring after the second heart sound and preceding the murmur (diastolic opening snap), then diamond shaped low-pitched murmur and S3 gallop. Rectangular mid-frequency systolic murmur.

Over the 2nd right intercostal space (aortic area), systolic click followed by crescendo-decrescendo murmur (diamond shaped murmur), ending just before the second heart sound with radiation into the right neck. The S2 is diminished in intensity (A2).

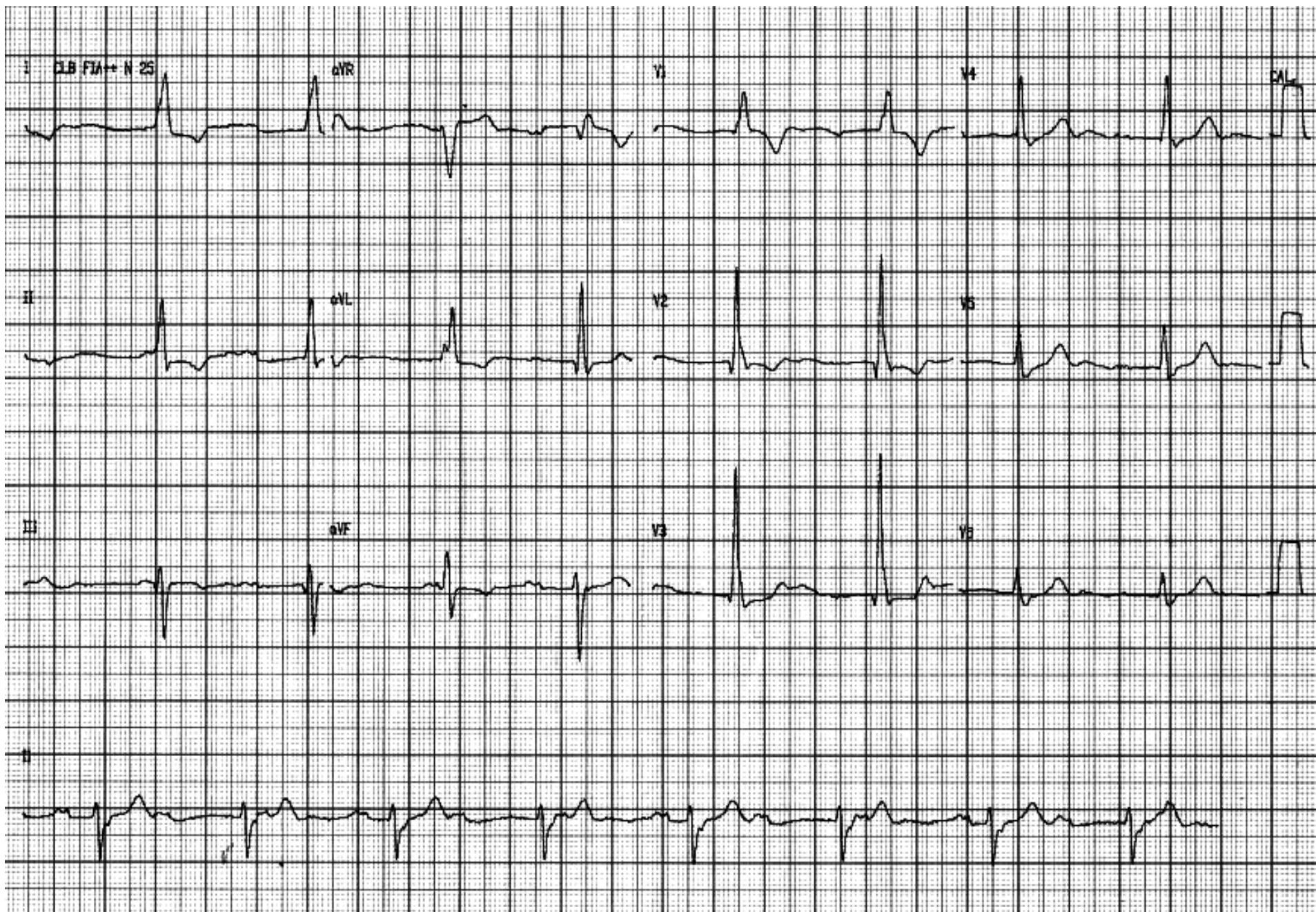
A diastolic high-pitched, musical decrescendo murmur is better heard in the third left intercostal space and irradiates widely along the left sternal border.

**Transthoracic echocardiogram:** Severely abnormal left atrium diameter/body surface area (BSA), cm/m<sup>2</sup>: 56.7 ml/m<sup>2</sup>. Left ventricular diastolic diameter mildly abnormal: 6.2. Eccentric left ventricular hypertrophy. Normal left ventricular ejection fraction = 63%. Diastolic dysfunction of left ventricle. Mitral valve disease: severe stenosis with moderate insufficiency - valve area = 0.9 cm<sup>2</sup> (severe < 1 cm<sup>2</sup>); maximal gradient = 23.5 mmHg; mean gradient = 13.5 mmHg (severe > 10 mmHg). Aortic valve disease with moderate-severe stenosis and moderate insufficiency. Normal tricuspid valve. Normal pulmonary systolic pressure = 30 mmHg. Normal right chambers.

## ECG-1 – At admission



## ECG-2 – A few hours later



**Colleagues' opinions**

## Spanish / English

Querido Andrés, No me lo he mirado con gran detalle, pero el QRS del primer día es  $> 120\text{ms}$  (quizás  $150\text{ms}$ ). Por qué dices incompleto RBBB? El segundo ECG es ciertamente un ritmo de escape ancho ( $\approx 150\text{ms}$ ) del V.I. situado probablemente entre los dos grandes fascículos pero con seguridad que hay conducción alterada por la RD. Es un ritmo idioventricular porque aunque el PH se parece mucho el PF es distinto. Sin embargo, porqué dices que el ritmo de escape ventricular muestra LAF y LSF y no hay conducción alterada en la parte más o menos distal de la RD. Aunque no haya R en VR esto se debe a que domina la activación 21 pero no significa que el estímulo no llega a la RD. Además el 2º complejo de VR es conducido, lo que demuestra una disociación AV incompleta.

Es un caso interesante. Yo querido Andrés lo veo así. Como decimos en España “a vuela pluma”. Me puedo equivocar.

Ya me dirás,

**Antonio Bayés de Luna**

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Respuesta: Querido maestro estoy de acuerdo con su primera apreciación que el QRS tiene duración de  $> 120\text{ms}$ . Lo considero incompleto RBBB por tener 2 condiciones asociadas: 1) transitoriedad o intermitencia (**Bayes 1997**) y 2) asociación con 2 bloqueos fasciculares o divisionales lo que le añade  $20\text{ms}$  cada (ver mas explicaciones en la diapositiva 22). Yo soy de los que piensa que el valor de  $120\text{ms}$  para considerar un bloqueo de rama completo es arbitrario a pesar de consagrado. Mas tarde, en esta presentación, le mostraré el diagnostico mas “masticado”. Espero su respuesta.

Muchas gracias por nos ayudar a pensar maestro querido.

Andrés.

## English

Dear Andrés, I did not check this thoroughly, but QRS the first day is  $>120$  ms (maybe 150 ms). Why do you think about IRBBB? The second ECG is certainly LV wide escape rhythm ( $\approx 150$  ms), probably located between the two large fascicles, but certainly there is no altered conduction by the RB. It is idioventricular rhythm, because although the HP is very similar to the FP, there are differences. However, why do you say the ventricular escape rhythm shows LAF and LSF and there is no altered conduction in the more or less distal part of the RB. Although there is no R in VR, this is because activation 21 is dominant, but it does not mean that the stimulus does not reach the RB. Moreover, the 2nd complex of VR is conducted, which shows incomplete AV dissociation. It is an interesting case. Dear Andrés, this is my point of view. As we say in Spain “a vuela pluma” (in a hurry). I could be wrong.

You let me know,

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**Reply by Andrés to Professor Bayés de Luna:** Dear Master, I agree with your first reflection, that QRS duration  $\geq 120$  ms. You ask me why I mention IRBBB (incomplete). I consider it IRBBB due to 2 associated conditions: 1) transitoriness or intermittence (**Bayes 1997**) and 2) the association with 2 divisional or fascicular blocks, which add 20 ms each. An IRBBB presents up to 110 ms + 2 ms of LPFB and +2 ms of LSFB, which could reach close to 160 ms (please see more explanation on slide 22) I am one of those who consider that the value of 120 ms is arbitrary for the complete one, although it is quite established. Luckily, this paradigm was already broken by Strauss et al in 2011 for LBBB cases (**Strauss 2011**). Now, I am sending you a more thorough diagnosis. I remain waiting for your reply.

Thank you very much for helping us to think, my dear Master.

8Andrés.

Spanish: Querido Potro(Andrés) excelente caso, intentare describir lo que observo.

1. Por la descripción se trata de una paciente con una valvulopatía mitral y aórtica asociada.
2. En el primer ECG RS 110 por min, Con signos de crecimiento biauricular y un BAV de 1er grado de alto grado con una PR de 380 seg. Bloqueo del fascículo anterior izquierdo, en precordiales presencia de fuerzas anteriores prominentes. Dado que presenta retraso en la conducción en las fuerzas finales del QRS en AVR y V5 y V6, lo atribuiría a dilatación del ventrículo derecho, no me impresiona un bloqueo del fascículo medio septal izquierdo.
3. En el segundo ECG presenta BAV de alto grado con ritmo idioventricular que alterna con latidos sinusales conducidos. 3er latido de AVR, AVL y AVL con un PR similar al ECG previo. al igual que V1 a V3 con un PR levemente incrementado de duración de 420 meg. En conclusión presenta un severo trastorno de la conducción AV con episodios de disociación AV y un bloqueo del fascículo anterior izquierdo y signos crecimiento VD, no me impresiona un BAV 2:1 ni tampoco que presenta por las FAP un bloqueo del fascículo medioseptal izquierdo. Un cordial saludo.

Martin Ibarrola

Dear Andrés ( Nick name Potro) Excellent case. I will try to describe what I see.

1. By the description, this is a patient with mitral valve disease and associated aortic valvular disease.; 2. In the first ECG, SR 110 bpm, with signs of biatrial enlargement and 1st degree AVB, with PR of 380 sec. LAFB; in precordial leads the presence of prominent anterior forces. Since there is conduction delay in the final forces of QRS in AVR and V5 and V6, I would attribute it to RV dilatation. It doesn't seem to be left septal fascicular block. 3. In the second ECG, there is AVB of a high degree with idioventricular rhythm, alternating with conducted sinus beats. 3rd beat of AVR, AVL and AVL with a pr similar to the previous ECG. Just as in V1 to V3 with slightly increased PR of 420 meg duration. To conclude, he presents severe disorder in AV conduction with episodes of AV dissociation, LAFB and signs of RV enlargement. It seems there is no 2:1 AVB or LSFB by PAF.

Regards,Martin

Ibarrola



Dear friends,

Very nice description of the physical exam and echocardiogram!!!

The first ECG shows a sinus cycle length of 1040 msec, prolonged PR interval of 480 msec, right bundle branch block, left anterior fascicular block, and left septal fascicular block. Most likely, A-V conduction is solely dependent on the left posterior division of the left bundle.

The second ECG shows A-V dissociation. The sinus cycle length is 800 msec, which is shorter than that in the first ECG, and most likely explains the transition from sinus rhythm to A-V dissociation. The ventricular escape cycle length is 1120 msec. The QRS of the escape rhythm has a different morphology than the QRS complexes when A-V conduction was present, which implies a lower origin in relation to the sites of block. The second QRS in leads aVR, aVL, and aVF is similar to those of the first ECG when conduction was present. I assume there is sporadic A-V conduction.

Thank you again for the interesting cases you share with us.

Best regards,

Mario **Gonzalez** M.D.

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# Final comments

**Spanish- Portuguese - English**

**“Leer mucho es uno de los caminos para la originalidad. Una persona será tan mas original y peculiar cuanto más conozca lo que los otros dicen”**

**Miguel de Unamuno ( 1864-1936) pensador español**

**“Ler muito é um dos caminhos para a originalidade. Uma pessoa será tão mais original e peculiar quanto mais conhecer o que disserem os outros”**

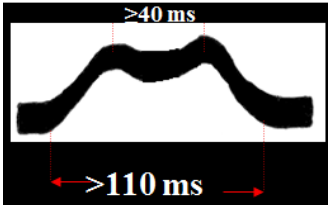
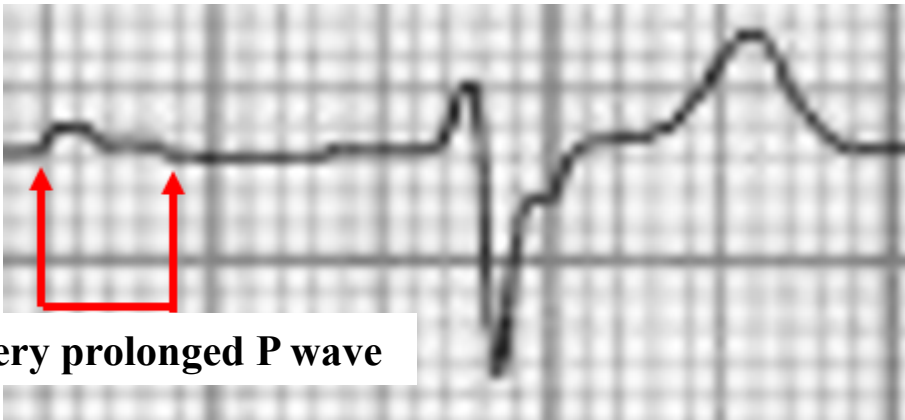
**Miguel de Unamuno ( 1864-1936) pensador espanhol**

**"Reading a lot is one way to originality. People will be so much more original and distinctive, the more they know what others say."**

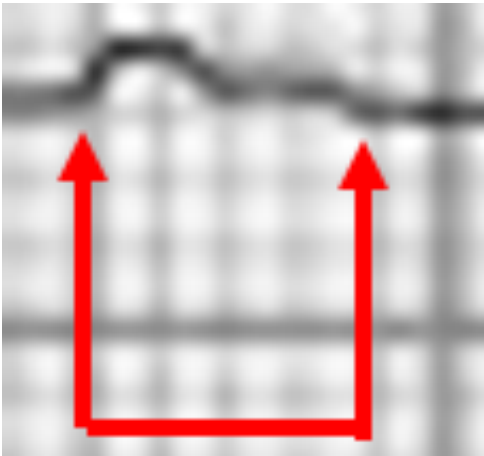
**Miguel de Unamuno (1864-1936) Spanish thinker.**

The first ECG shows association of:

**I. Left atrial enlargement and possible some degree of interatrial block**

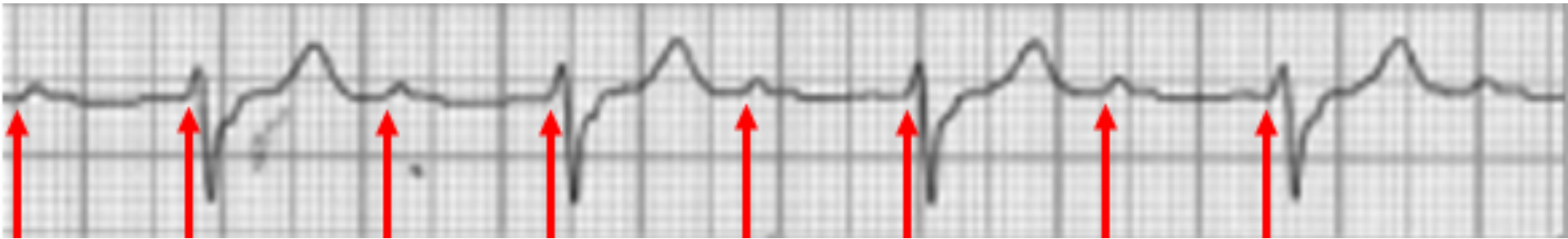


**Abnormal bifid or bimodal very broad P wave**



**P duration = 160 ms!!**

**II. Very prolonged PR interval (PRi = 480ms) or first-degree atrioventricular block (suggesting incomplete left posterior fascicular block)**



In old persons, the maximal normal superior limit for PR interval is 220 ms. Recently, findings from the Framingham Heart Study population have challenged this long-standing perception by demonstrating, that prolonged PR interval is associated with an increased risk of atrial fibrillation (AF) and pacemaker implantation (Schnabel 2009; Cheng 2009). Moreover, in contrast with previous studies, prolongation of the PR interval was also associated with all-cause mortality in a community-based sample (Cheng 2009; Magnani 2011).

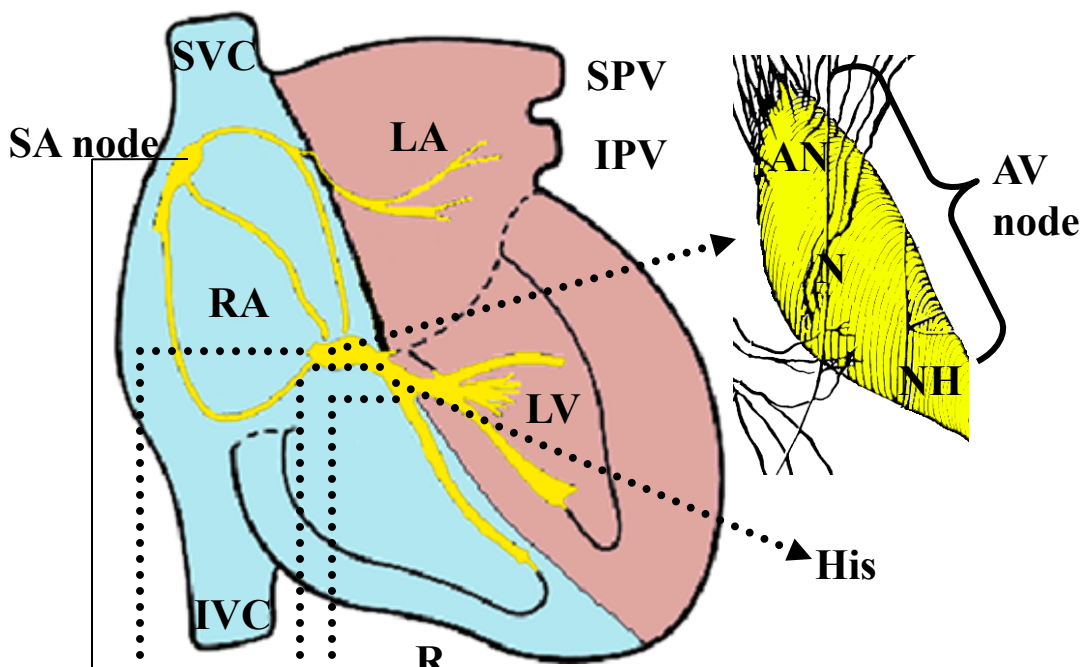
## First degree AV block

Concept: it is the prolongation of the PR interval (PRI)  $>200$  ms in adults,  $>180$  ms for adolescents between 14 and 17 years of age and  $>160$  ms in children, by conduction slowing in the atria (PA), AV node (AH), His bundle and its branches (HV) or association of the former, where each atrial depolarization (P wave) is followed by the corresponding ventricular depolarization (QRS), thus maintaining a 1:1 AV ratio. The PR interval is defined as the time elapsed between the onset of the P wave and the onset of the QRS complex (beginning of q or R wave), called PR, PRI or PQ interval, and it translates the time it takes the stimulus to go from the SA node until the onset of ventricular depolarization, in the middle third of the left septal surface. Normally, PR interval should be between 120 and 200 ms in the adult population.  $<120$  ms is associated with ventricular preexcitation. The PR interval represents the time needed for an electrical impulse from the sinoatrial (SA) node to conduct through the atria, the AVN, the bundle of His, the bundle branches, and the Purkinje fibers. Thus, as shown in electrophysiology studies, PR interval prolongation (ie, first-degree AV block) may be due to conduction delay within the right atrium, the AVN, the His-Purkinje system, or a combination of these.

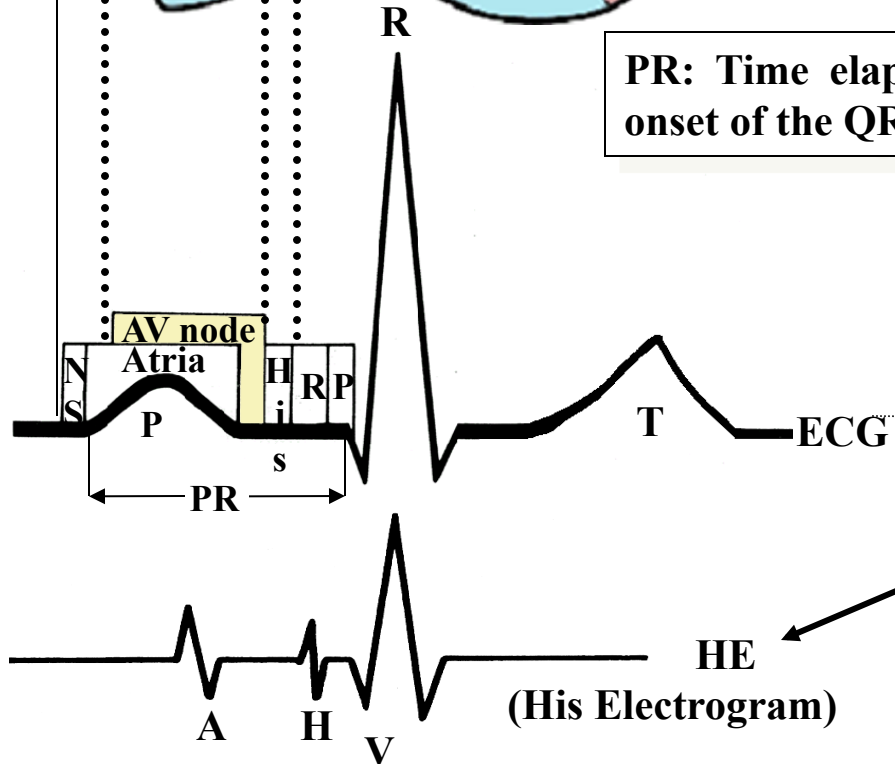
The duration of the PR interval depends, essentially, on three factors:

- 1) Age: directly proportional, i.e., the greater the age, the more prolonged. Elderly people with heart rate  $<70$  bpm may present PR intervals of 210 ms without considering there is first degree AV block. The prevalence of first-degree AV block increases with advancing age; first-degree AV block is reported in 5% of men older than 60 years (Upshaw 2004). The overall prevalence is 1.13 cases per 1000 lives.
- 2) Heart rate: inversely proportional, i.e. the lower it is, the shorter the PR interval and vice-versa.
- 3) Ethnic group: In a study of 2,123 patients aged 20-99 years, first-degree AV block was more prevalent among African-Americans than among Caucasians in all age groups except for those in the 8th decade of life. The peak in African-American patients occurred in the 10th decade of life, whereas the peak in Caucasian patients was in the 9th decade of life (Upshaw 2004).

# PR interval and its correlation with His Electrogram (HE) intervals



**PR:** Time elapsed between the onset of P wave and the onset of the QRS complex. VN: 120 to 200 ms

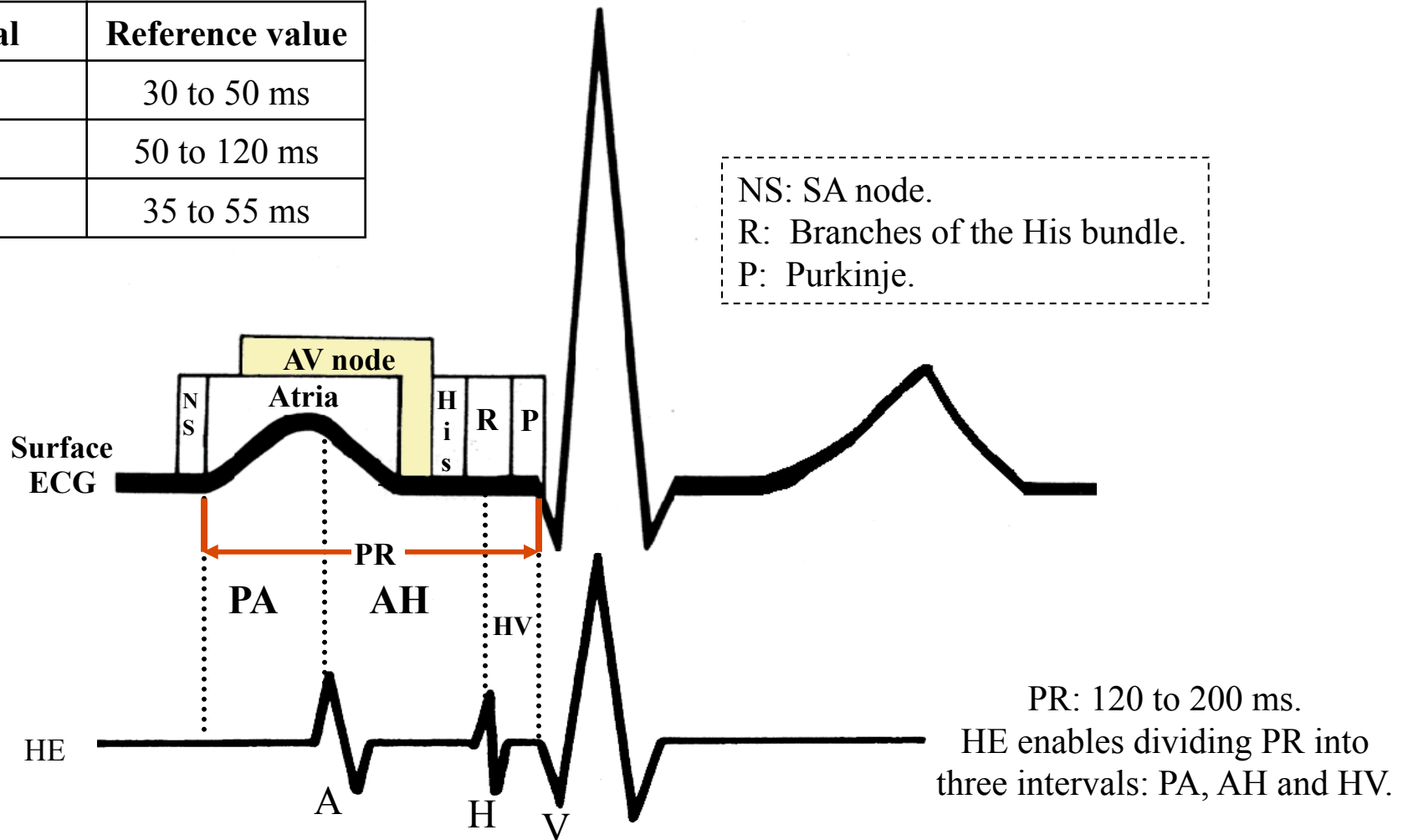


Interval	Reference value
PA	30 to 50 ms
AH	50 to 120 ms
HV	35 to 55 ms

**A:** First deflection of HE corresponding to inferior RA;  
**H:** Electrical activity of His bundle;  
**V:** Ventricular activation.

# Correlation of PR interval with electrogram (HE)

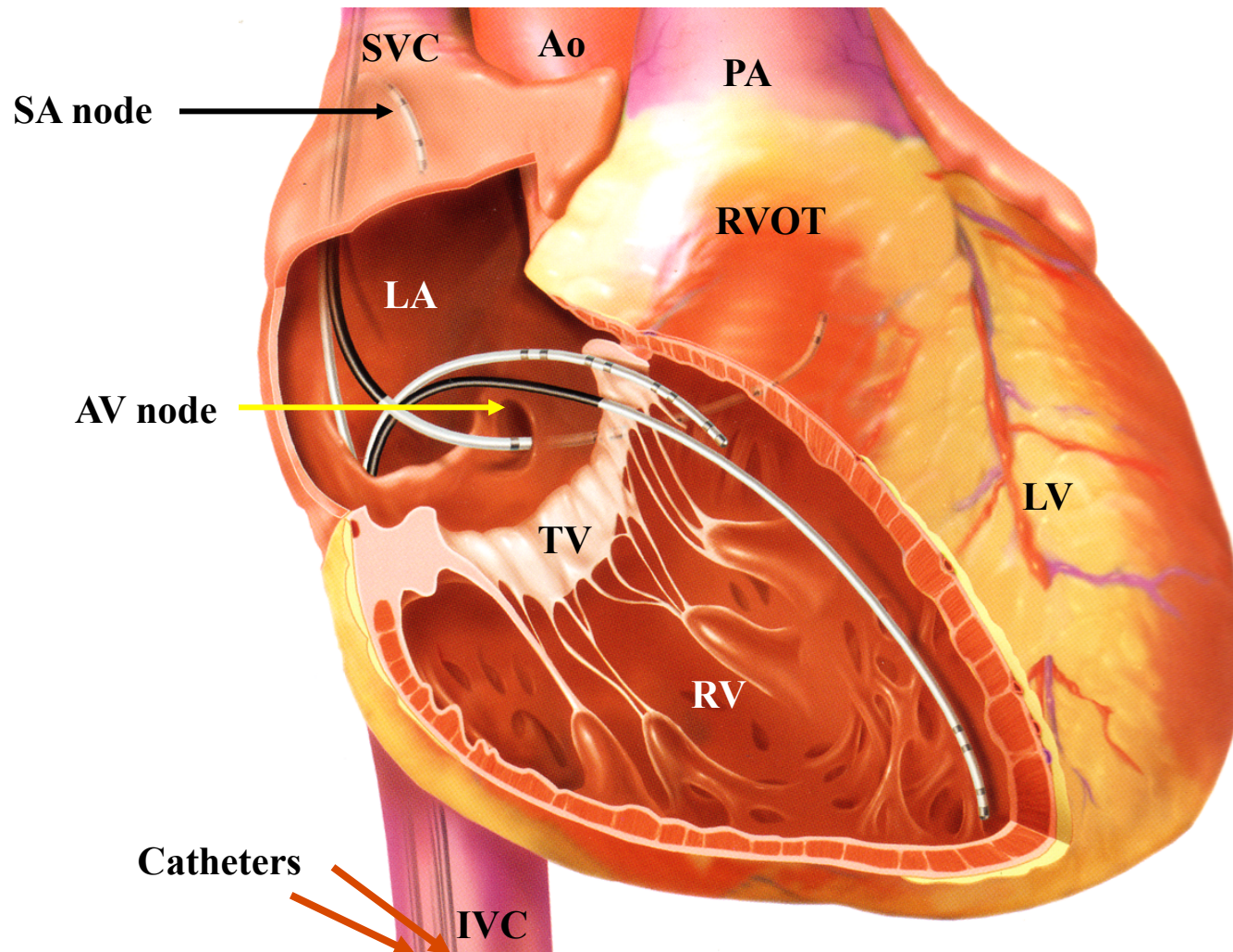
Interval	Reference value
PA	30 to 50 ms
AH	50 to 120 ms
HV	35 to 55 ms



PA: approximation of intraatrial conduction time;  
AH;  
HV: His-Purkinje conduction time;

A: first deflection of HE that corresponds to inferior RA;  
H: electrical activity of His bundle;  
V: ventricular activation.

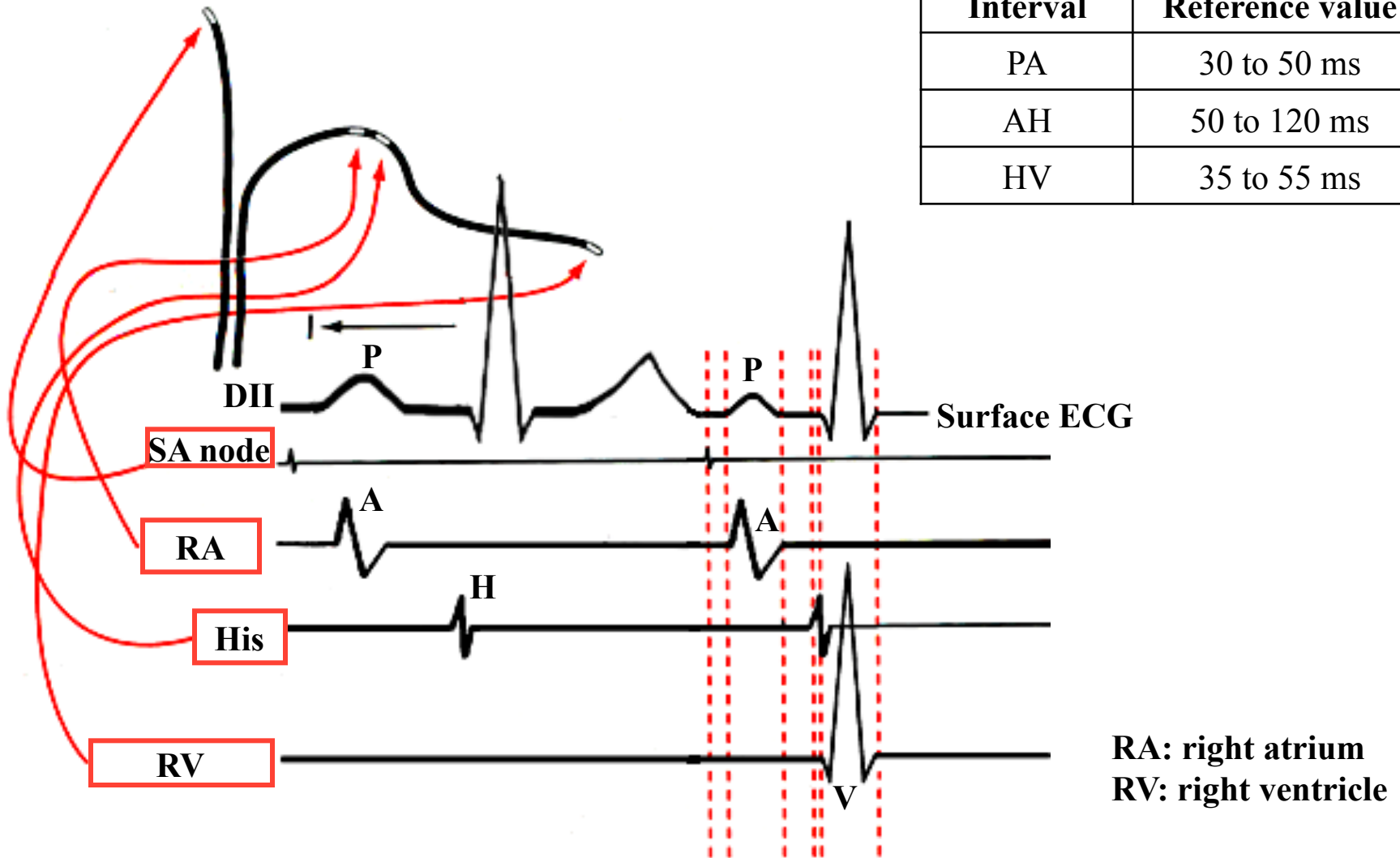
# Intracavitary electrogram



Location of catheters for intracavitary electrogram study during HE.

# Intracavitary electrogram and correlation with surface ECG

Interval	Reference value
PA	30 to 50 ms
AH	50 to 120 ms
HV	35 to 55 ms



Intracavitary electrogram with catheters placed on the SA node, the RA, the His bundle, and the RV.

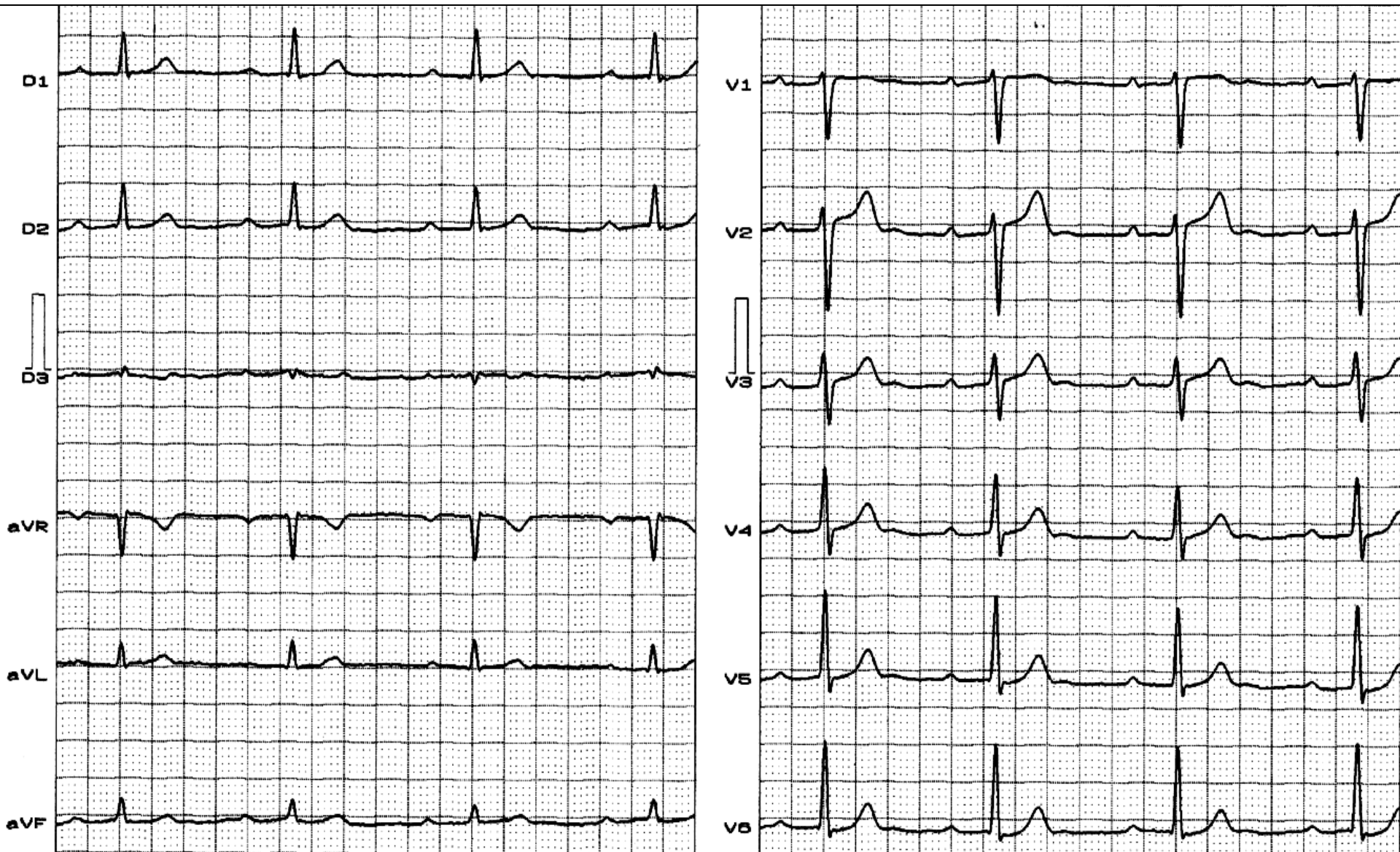
## Times of His electrogram

It is divided into three successive times:

- I. PA interval:** this is an approximation of intraatrial conduction time. It is measured from the onset of the P wave in ECG up to the apex of the 1st rapid deflection of the a wave in HE (inferior right atrium). Normal value: 30 ms to 50 ms. Intraatrial block extends the PA interval and favors the appearance of AF and atrial flutter.
- II. AH interval:** conduction time through the AV node (AVN). It is measured from the onset of the A wave in the HE (inferior right atrium) up to the 1st rapid deflection of the His bundle potential (H). Normal value: 50 ms to 120 ms. It is considered 1st degree AV block when the AH interval is  $\geq 120$  ms. The most frequent location of 1st degree AV block is the AV node region: 80% of the cases. The AV blocks located in the PA and AH regions are called supra-hisian and are considered to have a good prognosis. The AVN is the only normal electrical connection between the atria and the ventricles. It is an oval or elliptical structure, measuring 7-8 mm in its longest (anteroposterior) axis, 3 mm in its vertical axis, and 1 mm transversely. The AVN is located beneath the right atrial endocardium, dorsal to the septal leaflet of the tricuspid valve, and about 1 cm superior to the orifice of the coronary sinus. Blood supply to the AVN is provided by the AVN artery, a branch of the right coronary artery in 90% of individuals and of the left circumflex coronary artery in the remaining 10%. The His bundle has a dual blood supply from branches of anterior and posterior descending coronary arteries. Likewise, the bundle branches are supplied by both left and right coronary arteries. The AVN has a rich autonomic innervation and is supplied by both sympathetic and parasympathetic nerve fibers. This autonomic innervation has a major role in the time required for the impulse to pass through the AVN.
- III. HV interval:** time of His-Purkinje conduction (intraventricular). It is measured since the onset of H deflection up to the earliest onset of ventricular activation in surface ECG. Normal value: 35 ms to 55 ms. The time the stimulus stays in the His bundle and its branches is very short (30 ms maximum), as a consequence of the greater conduction velocity in this area (4000 mm/s), which explains that the His AV block, when there is increase of the HV interval  $< 50\%$ , co-exists with a normal PR interval in surface ECG ( $< 200$  ms). This condition is known as concealed first degree AV block (PR interval  $< 200$  ms and HV  $> 55$  ms).

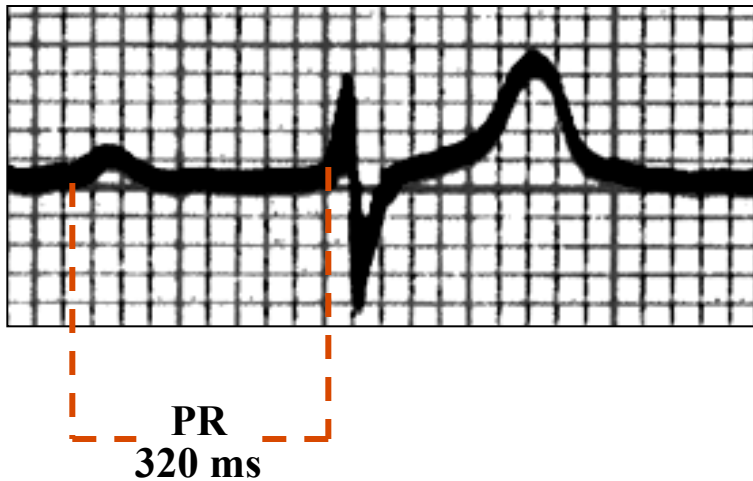
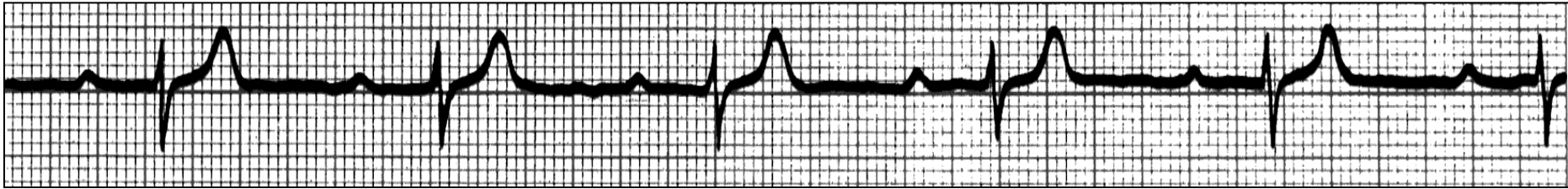
# ECG with 1st degree AV block

Male patient, 26 y/o, rheumatic fever still active



**ECG diagnosis:** First degree AV block: all atrial stimuli are conducted to the ventricles: 1:1 AV ratio. Prolonged PR interval (30 ms) even with heart rate of 55 bpm.

# First degree AV block



First degree AV block: all atrial stimuli are conducted to the ventricles: 1:1 AV ratio.

Prolonged PR interval (320 ms) even with sinus bradycardia of 52 bpm.

First-degree AV block is considered “marked” when the PR interval exceeds 300 ms (**Barold 2001; 2006**).

## Possible locations of first degree AV block

	Supra-hisian or pre-hisian	Hisian and infra-hisian
Location	AV node	His bundle or divisions
Percentage	75%	5% and 20%
QRS duration	Up to 100 ms	120 ms or more: branch block morphology
Electrogram	Prolongation of AH or PA intervals	Infrahisian: splitting of H deflection with progressive distancing: H1-H2
Autonomic influence	Important	Less
Prolonged Wenckebach cycles	Frequent	Rare
Prognosis	Better	Worse: it may evolve abruptly into advanced block. Risk of SCD

A. Supra-hisian or pre-hisian: 75%. Associated to narrow QRS.

B. Hisian: 5%.

C. Infra-hisian, fascicular or divisional: 20%. B and C associated to wide qrs.

Overall, dysfunction at the AVN is much more common than dysfunction at the His-Purkinje system. If the QRS complex is of normal width and morphology on the ECG, then the conduction delay is almost always at the level of the AVN. If, however, the QRS demonstrates a bundle-branch morphology, then the level of the conduction delay is often localized to the His-Purkinje system.

Occasionally, the conduction delay can be the result of an intra-atrial conduction defect. Some causes of atrial disease resulting in a prolonged PR interval include endocardial cushion defects and Ebstein anomaly (**Sherron 1985**).

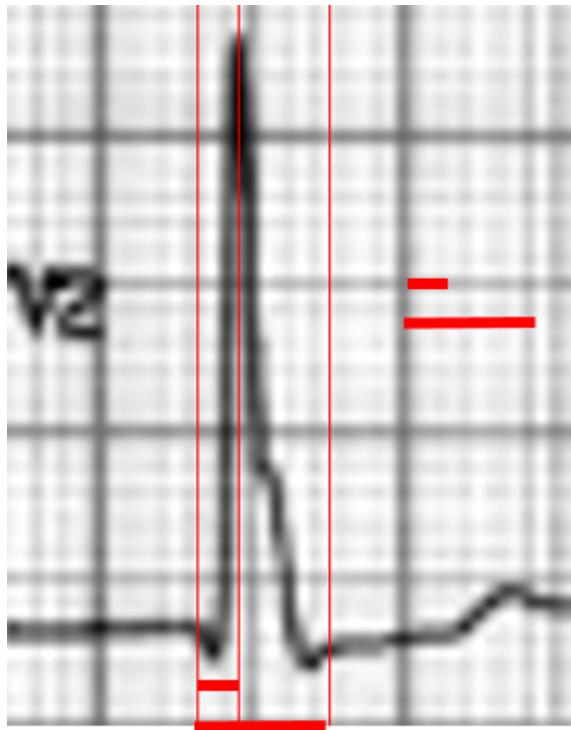
Locations of 1st degree AV block and differences between supra-hisian, hisian and infra-hisian topography.

## First-degree AV block prognosis

First-degree AV block has been considered a benign condition in healthy individuals, mainly among athletes. However, in a large cohort of patients with stable coronary artery disease, first-degree AV block is associated with heart failure and death (**Crisel 2011**). Progression from isolated first-degree heart block to high-degree block is rare (**Mymin 1986**). Patients with first-degree AV block and infranodal blocks, however, are at increased risk for progression to complete AV block. Heart block in children with Lyme carditis tends to resolve spontaneously, with median recovery in 3 days (range, 1-7 days) (**Costello 2009**). The Framingham Study have shown that first-degree AV block is associated with increased risk of all-cause mortality in the general population. Compared with individuals whose PR intervals were  $\leq 200$  ms, those with first-degree AV block had a 2-fold adjusted risk of AF, a 3-fold adjusted risk of pacemaker implantation, and a 1.4-fold adjusted risk of all-cause mortality (**Cheng 2009**). Each 20-ms increment in PR intervals was associated with an adjusted hazard ratio of 1.11 for AF, 1.22 for pacemaker implantation, and 1.08 for all-cause mortality. The Korean Heart Failure registry selected 1,986 patients with acute HF in sinus rhythm and divided them into 4 groups, depending on the presence of first-degree AV block and/or QRS prolongation. During the median follow-up of 18.2 months, overall death rate was highest in patients who had both first-degree AV block and prolonged QRS. This group also showed worst outcomes regarding the requirement of invasive managements during the index admission, in-hospital mortality, post discharge death/rehospitalization, and cardiac device implantation (**Park 2013**). Patients with stable CAD who had a PR  $\geq 220$  ms had a significantly higher risk of reaching the combined end point of HF or cardiovascular death over a follow-up of 5 years (**Crisel 2011**). In the COMPANION Trial, 1520 patients fulfilling criteria for cardiac resynchronization therapy (CRT) implant were assigned to normal (PR  $< 200$  ms) or prolonged (PR  $\geq 200$  ms) AV delay and cohorts were compared within the optimal pharmacologic therapy and CRT groups regarding an endpoint of all-cause mortality or HF hospitalization. CRT was compared with optimal pharmacological therapy in normal and prolonged PR interval groups. Randomization to CRT was associated with a reduction in the endpoint in all patients; the strength of the association was greater for those with first-degree AV block versus normal PR intervals. This analysis demonstrated that the deleterious effect of first-degree AV block in patients with systolic dysfunction, HF, and wide QRS complexes could be attenuated by CRT (**Olshansky**

2012). These studies suggest that first-degree AV block is not necessarily a benign condition; in patients with chronic systolic HF and wide QRS, CRT may attenuate its deleterious effect.

**III. Probable Left Septal Fascicular Block (LSFB):** prominent QRS anterior forces(PAF): R wave voltage of V1  $\geq 5$  mm; R wave of V2  $> 15$  mm; R/S ratio in V1  $> 2$ ; R/S ratio in V2  $> 2$ ; R wave "in crescendo" from V1 through V3 and decreasing from V5 to V6; increased ventricular activation time or intrinsic deflection V1 and V2:  $\geq 35$  ms (**Pérez-Riera 2016**); embryonic initial q wave in V1-V2; absence of q wave in left precordial leads V5, V6 and I (by absence of the first septal vector or  $1_{AM}$ ).



**R wave of V2  $> 15$  mm**

“All these changes occur with a QRS that widens no more than 0.02 seconds in pure and uncomplicated LAH” (**Elizari 2007**).

**Embryonic initial q wave in V2**

**— Prolonged Ventricular Activation Time (VAT) = 45 ms. Normal value  $\leq 35$ ms**

**— Prolonged QRS duration = 162ms ( prolonged): IRBBB 110ms +  $\approx$  2ms LAFB (**Elizari 2007**) +  $\approx$  + 2ms LSFB**

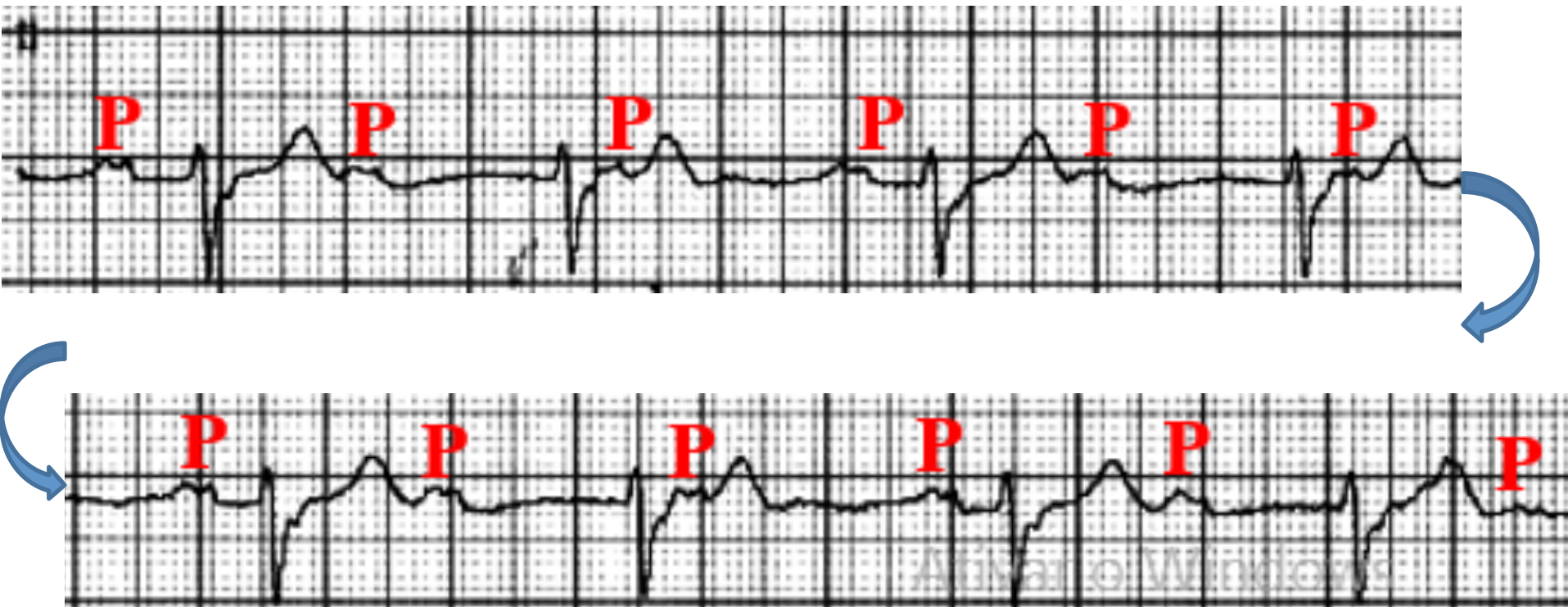
- IV. LAFB:** Extreme shift of SÂQRS in the left superior quadrant in the frontal plane (QRS axis  $-70^\circ$ ). rS in II, III and aVF, SIII > SII, R-peak time in aVL  $\geq 45$  ms. In precordial leads, voltage decrease of R wave and concomitant increase in S wave depth in V5 and V6, as a consequence of the superior dislocation of the forces.
- V. Incomplete right bundle branch block:** Supraventricular cardiac command, broad final S wave in left leads, qR pattern in aVR lead with wide final R wave followed by negative T wave, monophasic R wave in V1 followed by negative ST-T.

### ECG-1 Conclusions:

- *Left atrial enlargement and probable some degree of interatrial block: bifid and very prolonged P wave: P wave duration = 160 ms!!*
- *Very prolonged PR interval ( $PR_i = 480$  ms) or first-degree atrioventricular block (suggesting incomplete left posterior fascicular block)*
- *Probable Left Septal Fascicular Block (LSFB)*
- *Left Anterior Fascicular Block (LAFB)*
- *Incomplete right bundle branch block (IRBBB)*
- *Tetra fascicular block? : incomplete LPFB+LAFB+LSFB+ IRBBB*

The second ECG shows advanced AV block with AV dissociation. This is a form of complete or 3<sup>rd</sup> degree AV block with features of bifascicular block. P heart rate = 75 bpm. Ventricular command or the escape rhythm shows heart rate = 53 bpm and only conduction disturbance by the Left Septal Fascicle and Left Anterior Fascicle without any degree of right bundle branch block.

Conclusion: probable tetrafascicular block not described in the literature yet.



P-P = 75 bpm

R-R = 53 bpm

**Approach:** Dual-chamber pacemaker (DDD) implantation. In patients who have intermittent third-degree AV block and syncope not attributable to other causes, and marked prolonged HV interval  $\geq 100$  ms, permanent pacing is warranted. Class Ia.

Andrés

## References

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