An arrhythmia where the P-loop of the VCG in the Frontal Plane is decisive for the diagnosis

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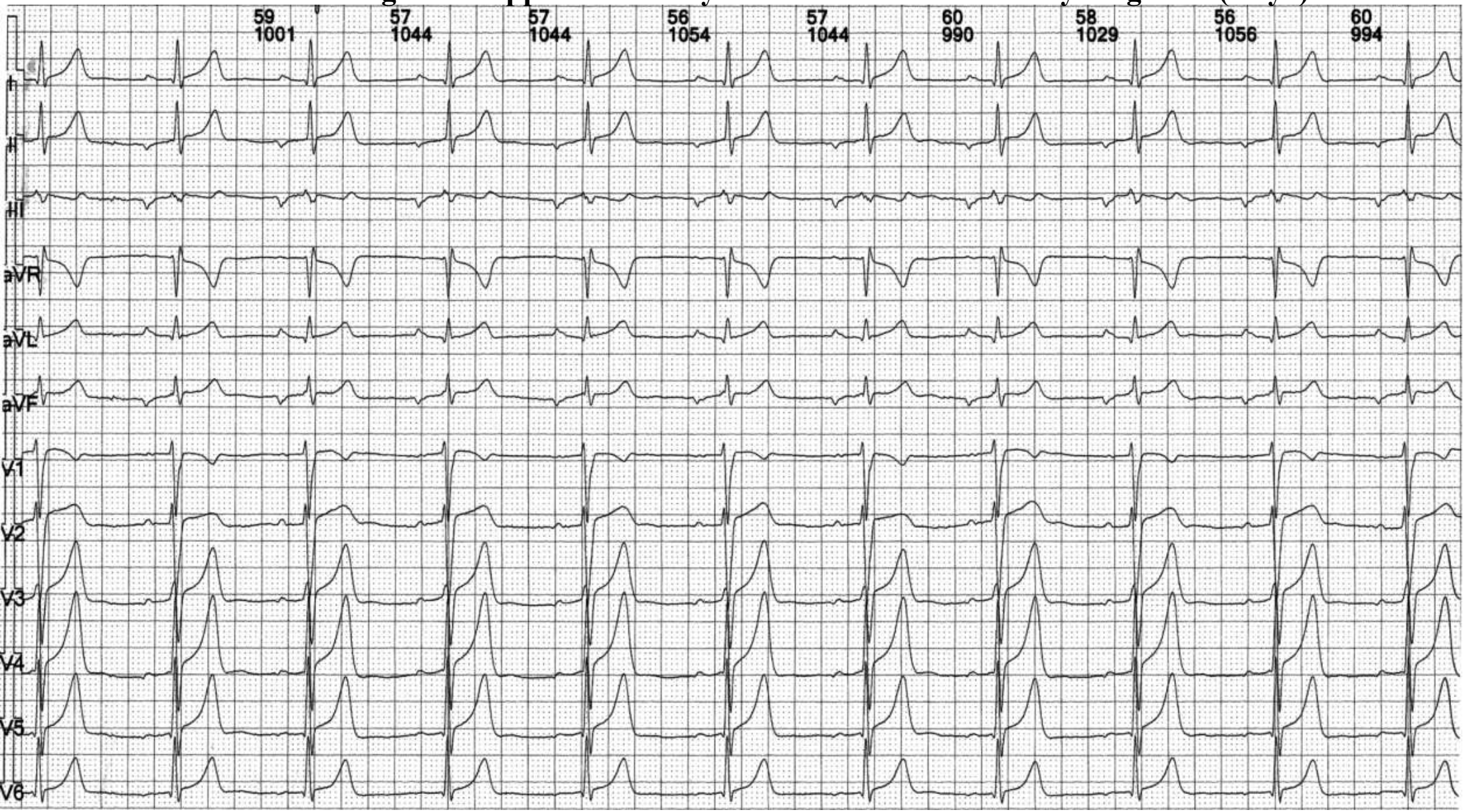
Portuguese

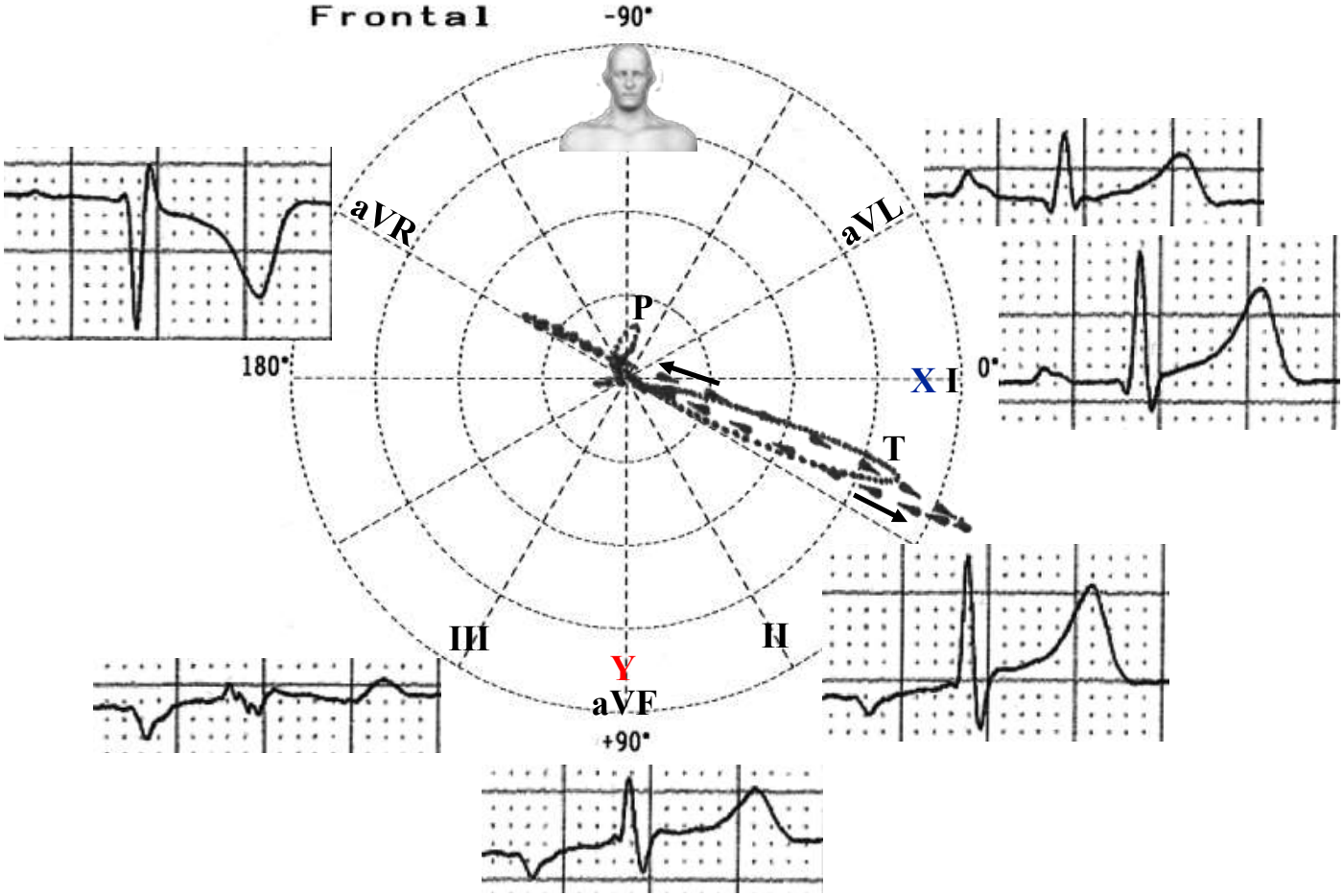
Paciente masculino, adulto jovem (30 anos de idade), raça negra, atleta, veio a nossa consulta para avaiação periódica.
Antecedentes pessoais: NDN. Sem vicios.
Antecedentes familiares nada digno de nota.
Avalição periódica completa realizada há um ano normal.
Exame físico normal PA 120/70mm de Hg FC 55 bpm
ECG e VCG realizado
Qual o diagnóstico da arritmia e porque?

English
Patient male, young adult (30 years old), black race, athlete, came to our consultation for periodic assessment.
Personal Background: NDN. No vices.
Family history nothing worthy of note.
Periodic full assessment performed one year normal.
Normal physical examination. BP: 120 / 70mm Hg; HR 55 bpm
ECG and VCG performed
What is the diagnosis of arrhythmia and why?

The ECG belongs to an apparent healthy athlete Afro descendant young adult(30 yo)

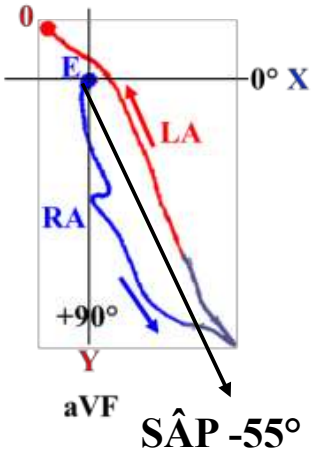


ECG/VCG correlation in the frontal plane



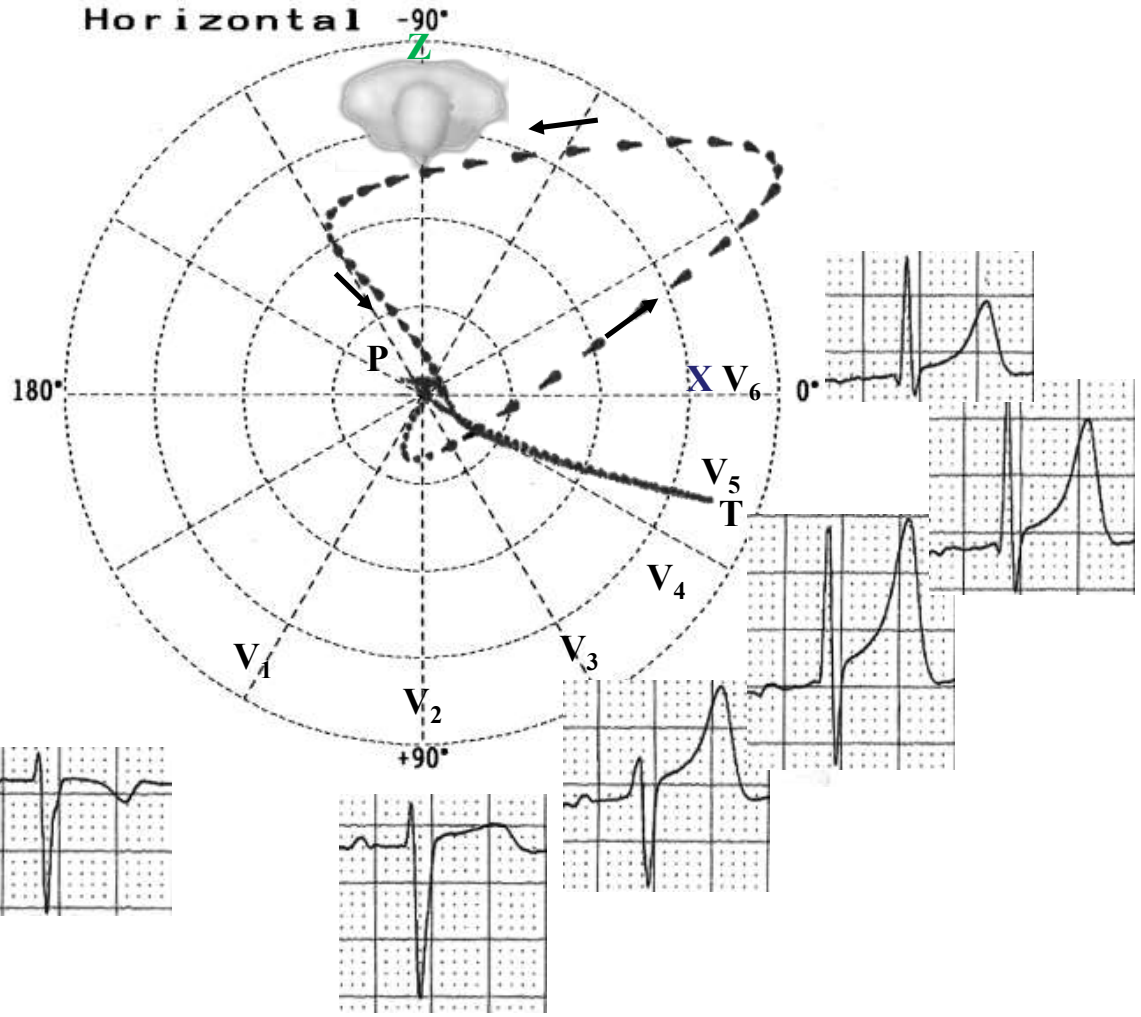
Magnified P-loop

Normal P-loop



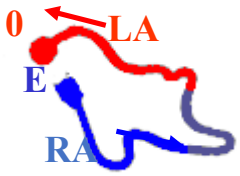
SÂP -55°

ECG/VCG correlation in the horizontal plane

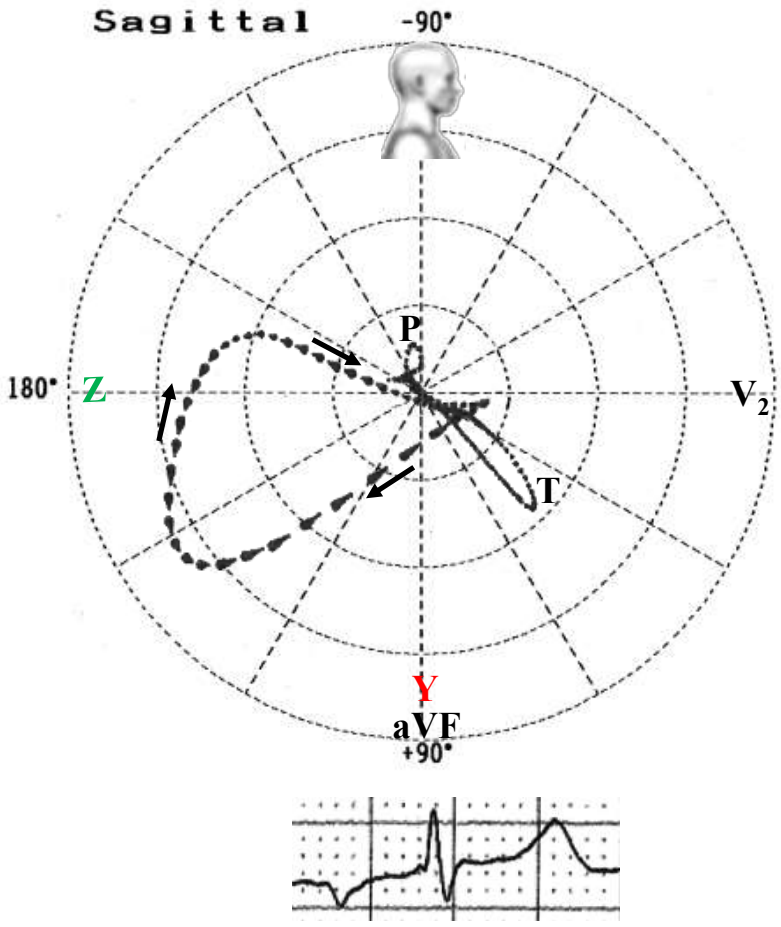


Magnified P-loop

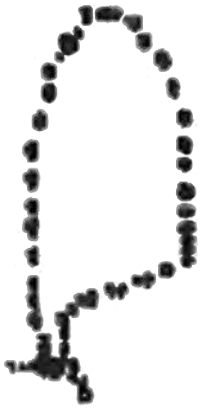
Normal P-loop



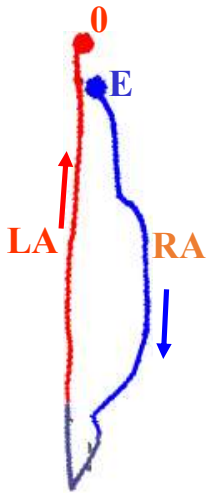
ECG/VCG correlation in the right sagittal plane



Magnified P-loop



Normal P-loop



Colleagues Opinions

Dear Raimundo and Andrés,

In this well trained asymptomatic athlete, a high vagal tone is likely. With a depressed sinus node (down regulation of If or up regulation of Ik Ach, see Mangoni ME et al, Nattel S et al) a rhythm from the coronary sinus arises, a benign condition (normal automaticity). The left atria however seems dilated, from an electrical point of view. An atrial neuromyopathy? (see atrial autonomic nervous system remodeling associated with AF).

What about the echocardiography ?

Is this patient suffering from high blood pressure?

Kind regards,

Philippe Chevalier MD PhD.
philippe.chevalier@chu-lyon.fr

Dear Philippe:
Thank very much for your fast and cleaver answer.
Echo: Normal
Absence of apparent structural heart disease.
Negative familial background
Normal blood pressure
Normal Glycated hemoglobin (HbA1C or A1C), formerly known as glycosylated hemoglobin
Non suspected autonomic neuropathy
In summary young man with apparent good health
Thank in advance
Andrés and Raimundo

Portuguese

Ritmo sinusal, distúrbio de condução intra-atrial, bloqueio AV de primeiro grau

Dr. José Grindler, Dr. Acácio Cardoso, Dr. Gabriel C. De Paula, Dr Marco Bustamante

Serviço de Eletrocardiologia- HC FMUSP

English

Sinus rhythm, intra-atrial conduction disorder, first-degree AV block.

Dr. José Grindler,

Dr. Acácio Cardoso

Dr. Gabriel C. De Paula,

Dr. Marco Bustamante

Electrocardiology Service- HC FMUSP

Dear Andrés

In my opinion we have:
1. Non-sinus atrial ectopic rhythm originating in the floor of the right atrium in the vicinity of the coronary sinus ostium
2. There is slurring of the final portion of the P wave loop in the frontal plane suggesting some conduction delay in the left atrium. P wave loop also shows clockwise rotation in the frontal plane. P wave duration is 124 ms and the axis is in the left upper quadrant towards minus 60 degrees. In the left precordial leads the P waves show minus-plus morphology , frequently observed when the atrial rhythm originates close to the coronary sinus.
3. Long PR interval (.24 sec)
4. Left ventricular activation time is 52 ms (V6) and 56 ms in V1 (little bit prolonged)
4. There is some slurring in the ascendant portion of the S wave in V1 (>40 ms)
5. Tpeak-Tend interval in V5 is 100 ms suggesting some left ventricular remodeling
6. QTc interval 432 ms

Final impression
Right ectopic atrial rhythm
Left atrial enlargement
First degree AV block
Some degree of left ventricular remodeling (electric left ventricular hypertrophy).

Thanks a lot

Dalmo Moreira MD, PhD
Electrophysiology and Electrocardiography Section
Dante Pazzanese Institute of Cardiology, São Paulo, Brazil

Spanish

Hola queridos Andrés y Raimundo:
Se trata de un ritmo auricular bajo en paciente con aumento del tono vagal asociado a un retraso de la conducción nodal por esto la duración del PR que presenta.
Porque ritmo auricular bajo?
La activación auricular la realiza en sentido antihorario comenzando de izquierda a derecha en el plano frontal. En el plano horizontal lo realiza en sentido inverso en forma de bucle. En el plano sagitario la activación caudo craneal.
Todo esto muestra que la activación auricular comienza en la región inferior auricular y activa las aurículas en forma retrógrada en sentido antihorario y de abajo hacia arriba.
Por esto es que mi interpretación es la de un ritmo auricular bajo en un paciente entrenado, la frecuencia del NS se encuentra por debajo del ritmo auricular bajo por hipertono vagal.
Así que no presenta patología ni arritmias a mi criterio.
Un cordial saludo
Martín **Ibarrola MD** Provincia de Buenos Aires Argentina

English

Hello dear Andrés and Raimundo:
It is a low atrial rhythm in a patient with increased vagal tone associated with delayed nodal conduction. For this reason the PR interval duration is prolonged and has low atrial rate.
The atrial activation is processed counterclockwise and from left to right in the frontal plane.
In the horizontal plane, I observe a reversal P-loop.
In the sagittal plane the caudo-cranial activation is evident.
All this shows that atrial activation begins in the lower atrial region and activates the atria in a retrograde fashion counterclockwise and from the bottom up.
This is why my interpretation is that of a low atrial rate in a trained patient, the sinus node frequency is below the low atrial rate by vagal hypertonus.So it does not present pathology or arrhythmias in my opinion.
Kind regards
Martín **Ibarrola MD** Province of Buenos Aires Argentina

Spanish

Estimados Andrés y Raimundo:
En mi opinión es un ritmo auricular que no es sinusal, sino que se origina en el ostium del Seno Coronario, o en su defecto podría ser del anillo tricuspídio bajo. No tengo experiencia con VCG.
Espero con ansia tu análisis!
Un abrazo,
Daniel Banina-Aguerre MD
Montevideo – Uruguay
Director de Electrofisiología Cardíaca - Hospital Militar - Uruguay -
Hospital Central de las Fuerzas Armadas

English
Dear Andrés and Raimundo:
In my opinion it is an atrial rhythm that is not sinusal, but originates in the coronary sinus ostium(CSO), or in its defect could be of the ring tricuspid valve.
I have no experience with VCG.
I look forward to your analysis!
A hug
Daniel **Banina-Aguerre MD Montevideo – Uruguay**
Observation: The coronary sinus ostium (CSO) is the main cardiac vein and it has become a clinically important structure especially through its role in providing access for different cardiac procedures. Considerable variations in the diameter of the CSO are observed. The mean craniocaudal diameter of the CSO is 8.1 ± 1.51 mm, and the mean transverse diameter is 7.67 ± 1.72 mm. Heart specimens without Thebesian valve tended to have larger ostia. The mean craniocaudal diameter and the mean transverse diameter of the CSO is statistically larger in the specimens without Thebesian valves which have a wide variety of their morphology. The majority of the Thebesian valves are semilunar in shape (74.42%). The extent to which the valve covered the ostium is variable, including remnant valves that covered < 15% of the CSO (35%), and valves that are large and covered at least 75% of the CSO (22.09%). In 3% the Thebesian valve completely occluded the ostium.(**Zhivadinovik 2016**)

Spanish

Querido Andrés y Raimundo:

El asa de la onda P en el plano frontal se dirige de abajo hacia arriba y de derecha a izquierda. Lo cual implica que el ritmo es auricular bajo. Si bien como se ha mencionado la duración de la onda P que se observa en el ECG es de 124 mseg, la activación auricular no se encauza en forma normal; por lo cual yo no se si ese factor no es el que esta originando el aumento de su duración. Con respecto al enlentecimiento final (porque las comas están más juntas) yo no puedo afirmar que sea debido a un trastorno de conducción en la auricular izquierda, sino que se debe a que el frente de activación se orienta hacia la misma zona sin tener fuerzas opositoras.

Con los otros dos planos tengo problemas: El horizontal me muestra que las fuerzas iniciales se dirigen levemente hacia atrás, luego en forma horaria hacia adelante para rotar en 8 y terminar en el punto 0. En el sagital derecho me muestra que se dirigen hacia adelante y sin ese entrecruzamiento y sin distancia entre el punto E y el punto 0. Por lo cual es muy difícil saber la secuencia en la activación auricular.

Afectuosamente Isabel

English

Dear Andres and Raimundo:

The P wave/ loop in the frontal plane is heading upward and from right to left. This indicates that this is a low atrial rhythm. Although the duration of the P wave in the ECG is prolonged (124 ms) as mentioned before, and atrial activation does not run a normal course; therefore, maybe this factor is responsible for the increase in P wave duration in the ECG. As to the final slowing (because the dashes are closer to each other), I cannot state that this is due to a conduction disorder in the left atrium, but rather to the activation front heading to the same area with no opposite forces.

The other two planes are problematic for interpretation: the horizontal plane shows the initial forces heading slightly backward, then clockwise and forward to rotate in 8, and end in the 0 point.

The right sagittal plane shows me they are heading forward and not intertwining, and with no distance between the E point and the 0 point. Thus, it is very difficult to know what the atrial activation sequence is.

Affectionately,

Isabel **Victoria Konopka** Hospital Argerich, Buenos Aires · Department of Cardiology

Cardiology, Cardiothoracic Surgery, Internal Medicine (General Medicine)

Buenos Aires Argentina

Spanish
Estimado Andrés y Raimundo:
Veo una onda biauricular probable síndrome de Bayés.
Cordialmente

Dr. Eduardo Quiñones Córdoba Argentina

English
Dear Andrés and Raimundo:
I see a binaural wave likely Bayes´s syndrome.
Sincerely

Dr. Eduardo Quiñones Córdoba Argentina

Universidad Nacional de Córdoba, Argentina

https://www.unc.edu.ar/english/

Spanish

Hola Andrés y Raimundo
Mi opinión
El ECG revela un ritmo auricular bajo con eje de P localizado aproximadamente - 60° y demora en la condución AV
Que nos aporta el VCG?
En plano frontal el bucle de P tiene sus porciones iniciales (correspondiente a despolarización de aurícula derecha), con rotación horaria y localización en cuadrante superior izquierdo. Esto se debería a un foco automático de origen localizado en porción baja de aurícula derecha, posiblemente porción media o baja de la crista terminalis. Si asumimos esto, puede ser que presente una taquicardia auricular en algún momento evolutivo. Seria interesante realizar un Holter o una prueba ergométrica graduada
El bucle P no me sugiere bloqueo interatrial avanzado/completo o sindrome de Bayés.
Saludos
Juan José Sirena MD Santiago del Estero Argentina

English

Hello Andrés and Raimundo
My opinion
The ECG reveals a low atrial rate with a P axis located approximately - 60 ° and delayed AV conduction. What does the VCG provide us?
In the frontal plane the P loop has its initial portions (corresponding to depolarization of the right atrium), with Clock Wise rotation and location in the upper left quadrant. This would be due to an automatic focus origin located in the lower portion of the right atrium, possibly middle or lower portion of the terminal crest. If we assume this, it may present atrial tachycardia at some evolutionary stage. It would be interesting to perform a Holter or a graded exercise test
The P loop does not suggest complete interatrial block or Bayé´s syndrome.
Thank you!

Juan José Sirena MD Santiago del Estero Argentina

Final comments by

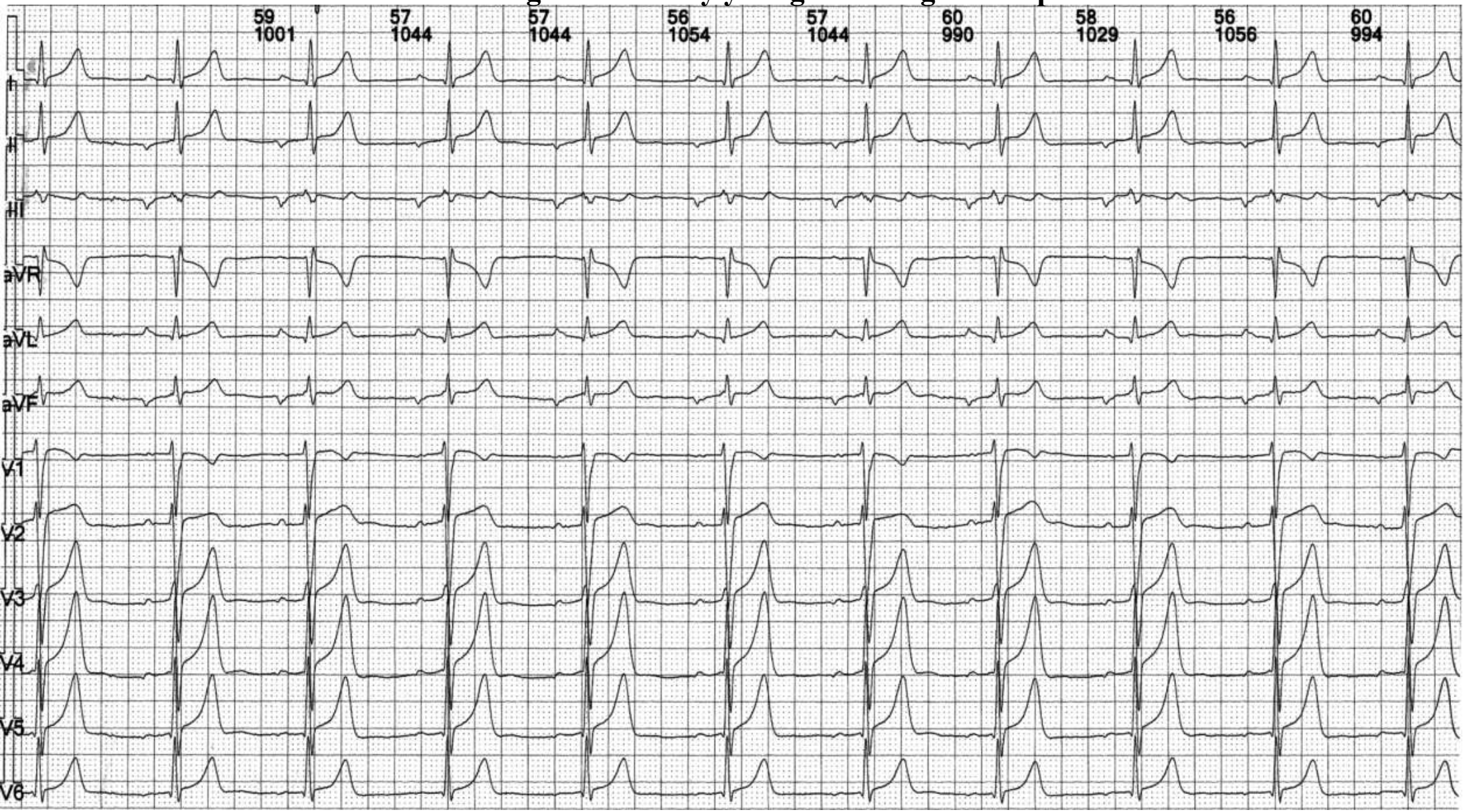
Andrés Ricardo **Pérez-Riera, M.D. Ph.D.**
Design of Studies and Scientific Writing Laboratory in the ABC School of Medicine, Santo André, São Paulo, Brazil
https://ekgvcg.wordpress.com

Raimundo **Barbosa-Barros**, MD
Chief of the Coronary Center of the Hospital de Messejana Dr. Carlos Alberto Studart Gomes. Fortaleza – CE- Brazil

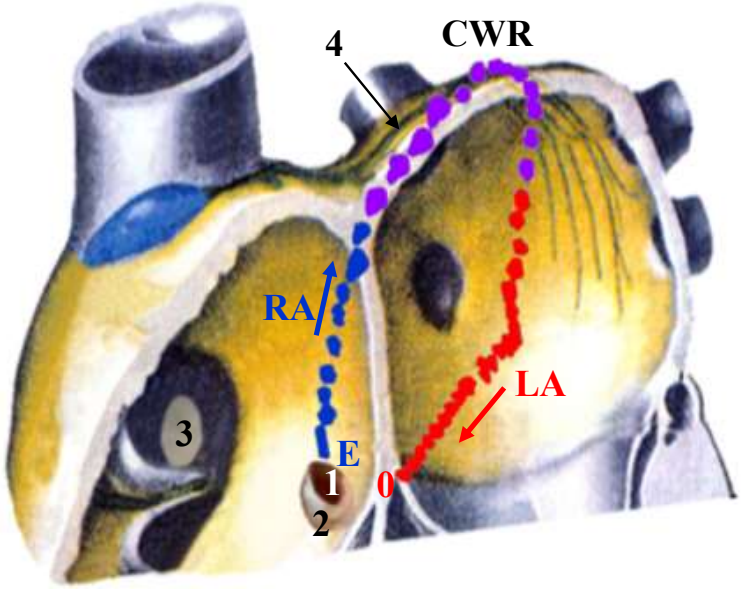
“Só existe uma coisa pior do que falarem mal de mim. É não falarem” (Oscar Wilde)

"There is only one thing worse than saying bad things about me. Do not talk about me"(Oscar Wilde)

The ECG belongs to a healthy young adult vagotonic sportsman



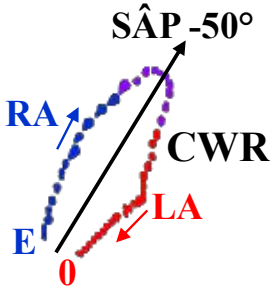
ECG/VCG correlation in the frontal plane

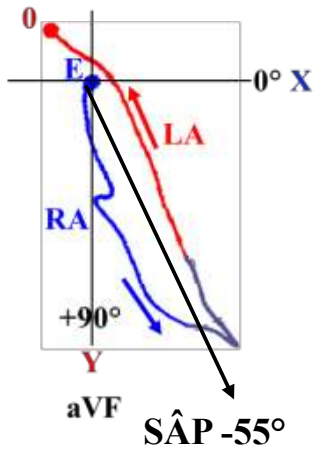


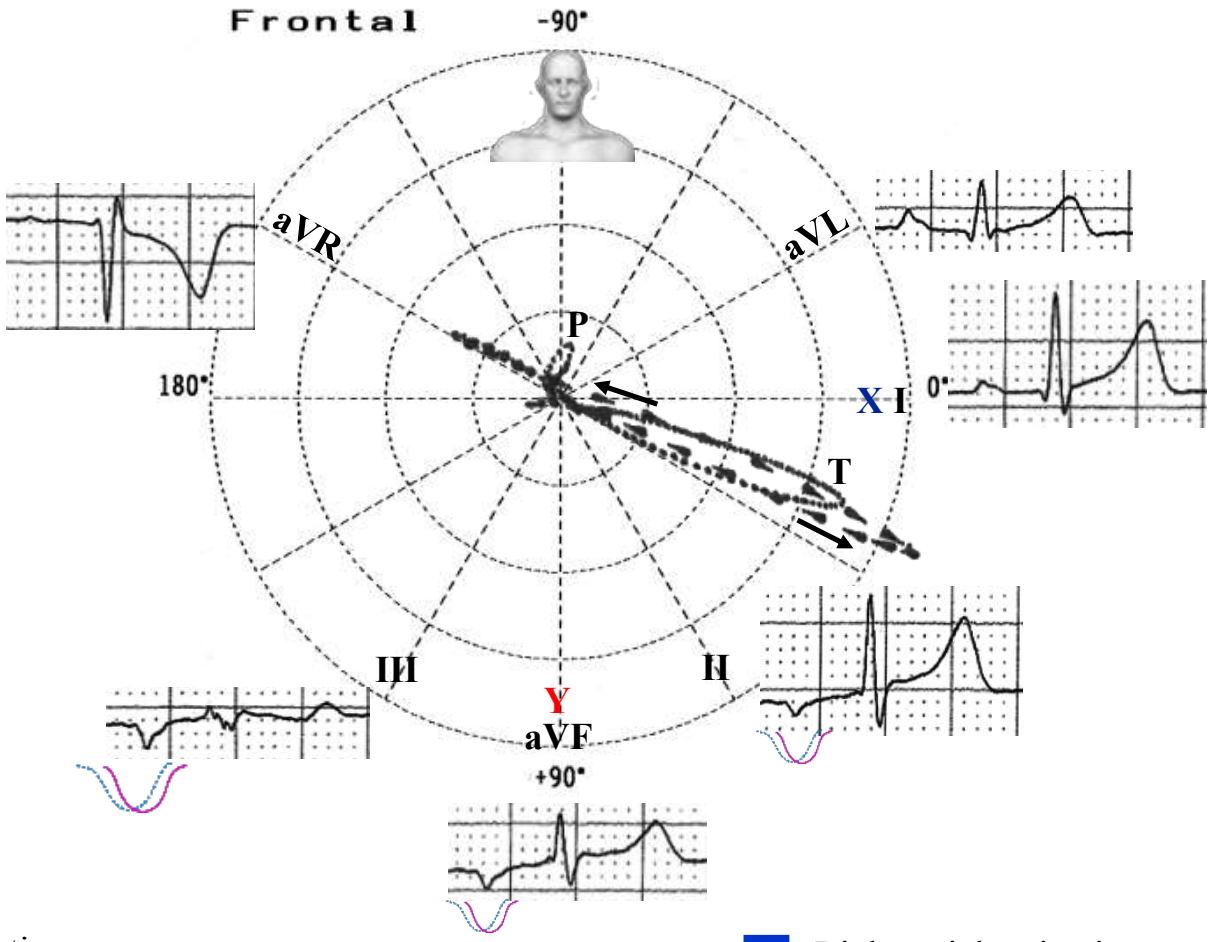
Normal P-loop

The present case

Magnified P-loop







1. Coronary sinus ostium
2. Thebesian valve
3. Fossa ovalis
4. Bachman's region (BR)

CWR: P-loop with Clock Wise Rotation

Right atrial activation

Biatrial activation

Left atrial activation

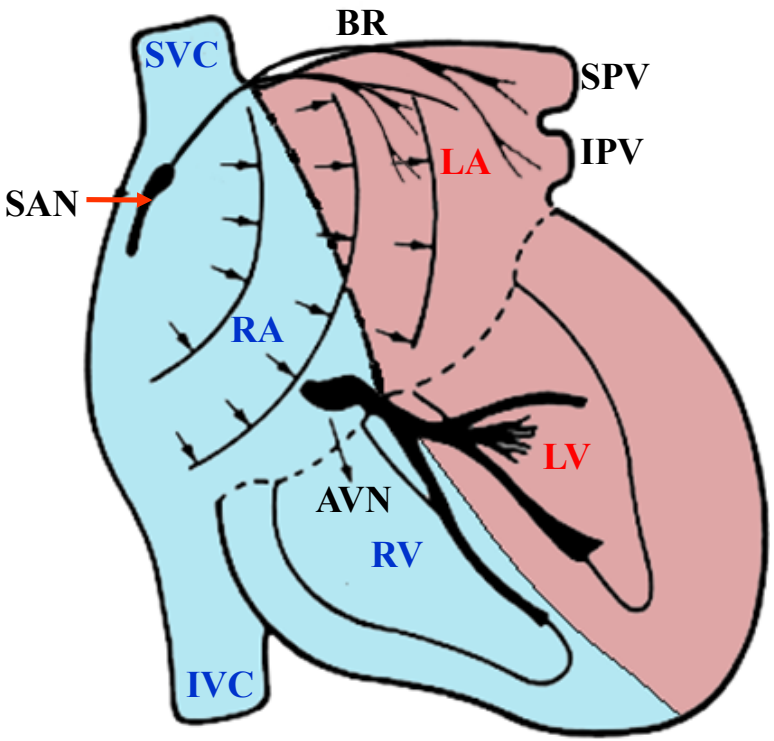
The polemic atrial depolarization pathways

The sinoatrial node (SAN) is located in the upper part of the right atrium (**RA**) in the healthy heart, and serves as the natural pacemaker. These nodal cells manifest spontaneous depolarizations and are thus responsible for generating the normal cardiac rhythm; such a heart rate can also be described as intrinsic or automatic. Importantly, the frequency of this earliest cardiac depolarization is well modulated by both sympathetic and parasympathetic efferent innervation. In addition, the nodal rate can also be modulated by local changes within perfusion and/or the chemical environment (i.e., neurohormonal, nutritional, oxygenation, etc.). Although the atrial rhythms normally emanate from the SAN, variations in the initiation site of atrial depolarization have been documented outside of the histological nodal tissues, particularly when high atrial rates are elicited, and may include paranodal tissue (**Yamamoto 2006; Betts 2003; Boineau 197; 1980; Lee 1995**). One of the most conspicuous features of SAN cells or P cells is that they possess poorly developed contractile apparatus (a common feature to all myocytes specialized for conduction), comprising only about 50% of the intracellular volume (**Anderson 2009;.Yamamoto2006; Tranum-Jensen1976**). In general, although it typically cannot be seen grossly, the location of the SAN is on the roof of the **RA** at the approximate junction of the superior vena cava, the **RA** appendage, and the sulcus terminalis. In the adult human, the node is approximately 1 mm below the epicardium, 10-20 mm long, and up to 5 mm thick (**Anderson 2009; Waller 1993)**. After initial SAN excitation, depolarization spreads throughout the atria.

The exact mechanisms involved in the spread of impulses (dromotropism) from the SAN across the atria are still today, somewhat controversial (**Anderson 2009; Boyett 2007**). However, it is generally accepted that: 1) the spread of depolarizations from nodal cells can go directly to adjacent myocardial cells; and 2) preferentially ordered myofibril pathways allow this excitation to rapidly transverse the **RA** to both the left atrium(**LA**) and the atrioventricular node (AVN). It is believed by many that there are three preferential anatomic conduction pathways from the SAN to the AVN (**Anderson 2009; Garson 1998**). In general, these can be considered as the shortest electrical routes between the nodes. Note that there are microscopically identifiable structures, appearing to be preferentially oriented fibers, that provide a direct node-to-node pathway. In some hearts, pale staining Purkinje-like fibers have also been reported in these regions. More specifically, the anterior tract is described as extending from the anterior part of the SAN, bifurcating into the so-called Bachmann's bundle which importantly delivers impulses to the **LA** and with a second tract that descends along the interatrial septum that connects to the anterior part of the AVN. The middle (or Wenckebach's pathway) extends from the superior part of the SAN, runs posteriorly to the superior vena cava, then descends within the atrial septum, and may join the anterior bundle as it enters the AVN. The third pathway is described as being posterior (Thorel's) which, in general, is considered to extend from the inferior part of the SAN, passing through the crista terminalis and the Eustachian valve past the coronary sinus to enter the posterior portion of the AVN. In addition to excitation along these preferential conduction pathways, general excitation spreads from cell to cell throughout the entire atrial myocardium via the specialized connections between cells, the gap junctions, that typically exist between all myocardial cell types.

Biatrial chamber activation without the polemic preferential pathways

BR: Bachmann's region



SPV: superior vena cava
IPV: inferior vena cava
SPV: superior pulmonary vein
IPV: inferior pulmonary vein
LA: left atrium
RA: right atrium
LV: left ventricle
RV: right ventricle
AVN: AV-node

Bachman's region, in the upper part of the interatrial zone, also know as the anterior interatrial band is the only track that conveys impulses to the **LA** (**Bachman 1916**). Specialized conduction cells in human interatrial septum (IAS) have been identified, specifically in the fossa ovalis (FO) and its flap valve. The cells are aggregated in a structure, which is surrounded by fibrous and fatty tissue. Further investigations are warranted to explore electrophysiological characteristics of this structure (**Mitrofanova 2014**) .

It then follows that towards the end of atrial depolarization, the excitation reaches AVN via the aforementioned atrial routes, with the final result being excitation of the AVN. Further, these routes are known as the slow or fast pathways, which are considered to be functionally and anatomically distinct. The slow pathway typically crosses the isthmus between the coronary sinus and the tricuspid annulus; it has a longer conduction time, but a shorter effective refractory period. The fast pathway is commonly a superior route, emanating from the interatrial septum, and has a faster conduction rate but, in turn, a longer effective refractory period. Normal conduction during sinus rhythm occurs along the fast pathway, but higher heart rates and/or premature beats are often conducted through the slow pathway, since the fast pathway may be refractory at these rates. Though the primary function of the AVN may seem simple, that is to relay conduction between the atria and ventricles, its structure is very complex (**Anderson 2009**). As a means to describe these complexities, mathematical arrays and finite element analysis models have been constructed to elucidate the underlying structure-function relationship of the node (**Li 2008**). After leaving the bundle of His, the normal wave of cardiac depolarization spreads first to both the left and right bundle branches; these pathways rapidly and simultaneously carry depolarization to the apical regions of both the left and right ventricles. Finally, the signal broadly travels through the remainder of the Purkinje fibers and ventricular myocardial depolarization spreads. In certain pathological conditions, direct accessory connections from the AVN and the penetrating portion of the bundle of His to the ventricular myocardium have been described (**Becker 1976**). Yet, the function and prevalence of these connections, termed Mahaim fibers, is poorly understood. A rare bundle of Kent, an additional aberrant pathway when present, exists between the atria and ventricles and is associated with the clinical manifestation of ventricular tachycardias (also known as Wolff-Parkinson-White syndrome). Therapeutically, this accessory pathway is electrically identified and then commonly ablated as a curative procedure.

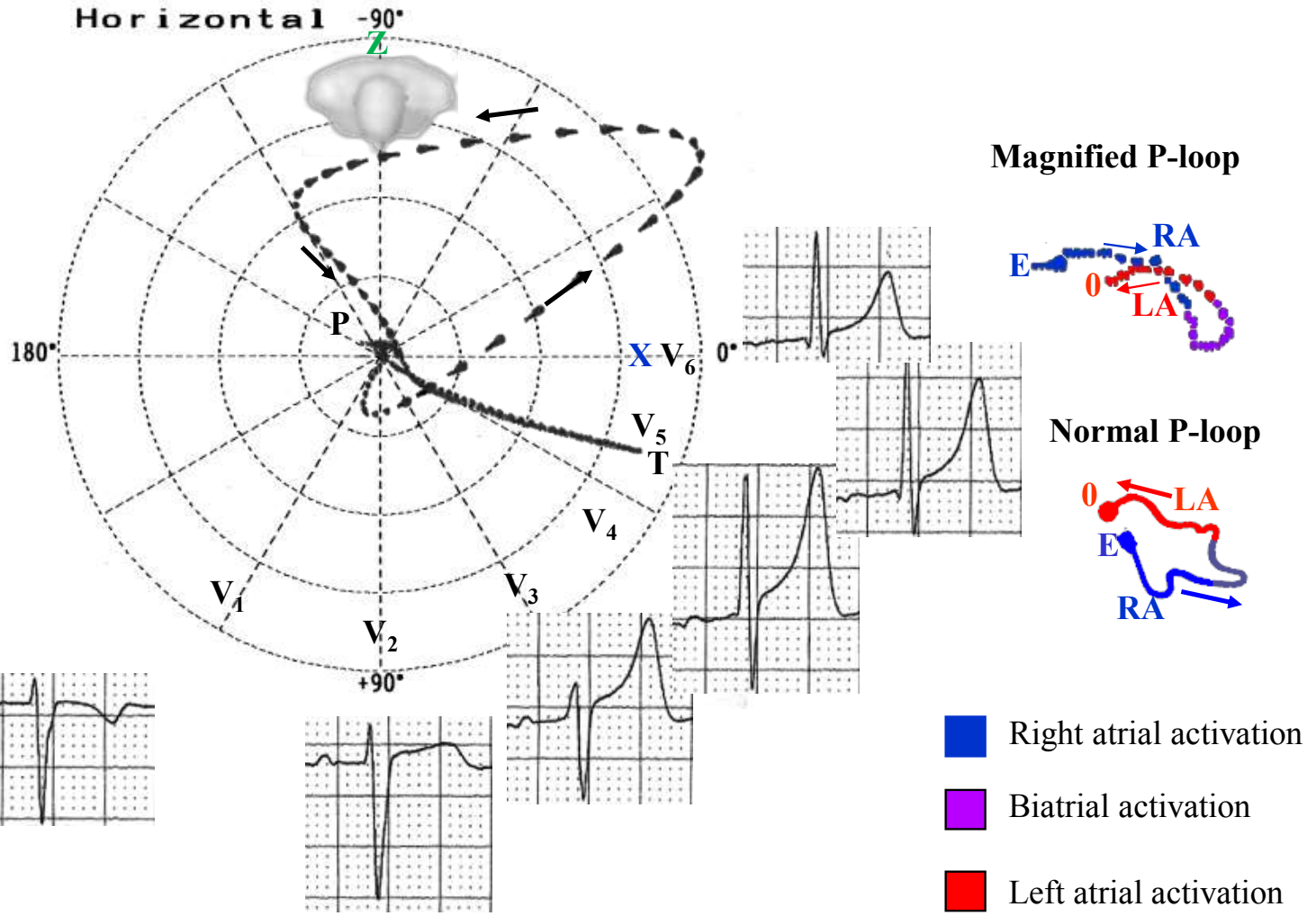
ECG/VCG correlation in the horizontal plane

P-polarity negative P-wave in the inferior leads

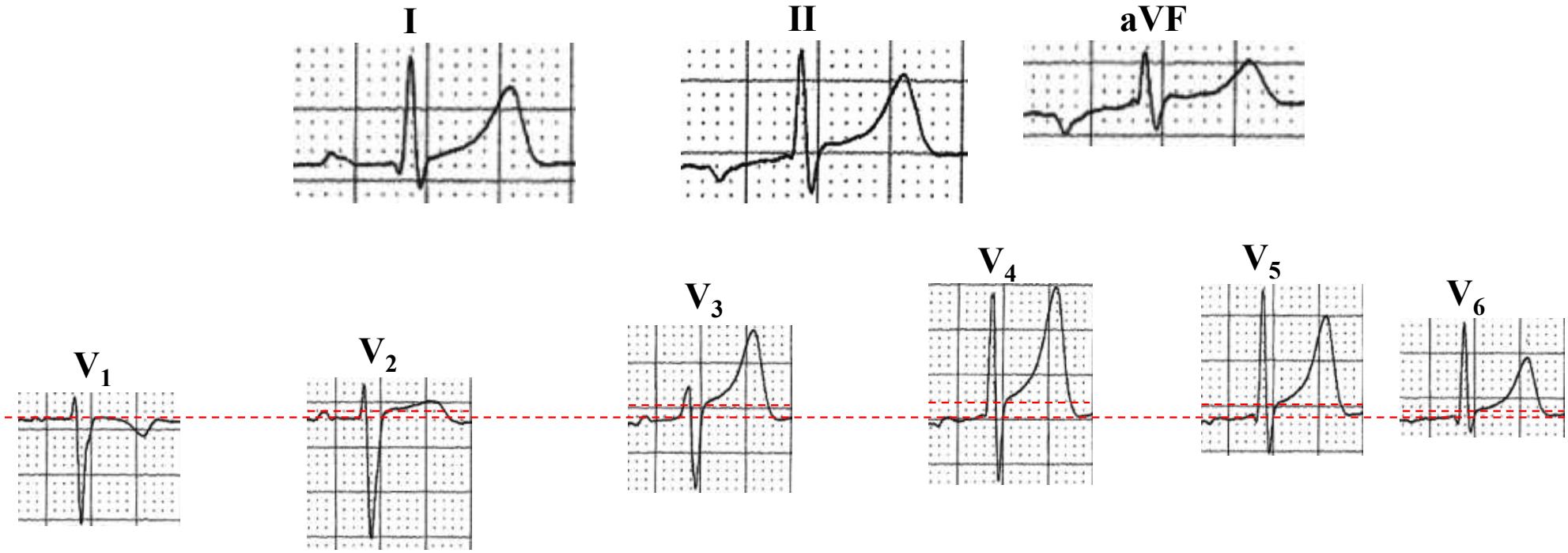
E: it constitutes the zero point of VCG and it remains stationary before the onset of the P loop. It corresponds to the isoelectric line between the T wave and the P wave of ECG. The **E** letter corresponds to the cardiac dipole.

0: it corresponds to the end of the P-loop.

The P loop begins in the **E** point and ends in the so-called **0** point. The former has an anterior and inferior location in relation to the latter. On the other hand, in this case **E** point is posterior related **0** point.



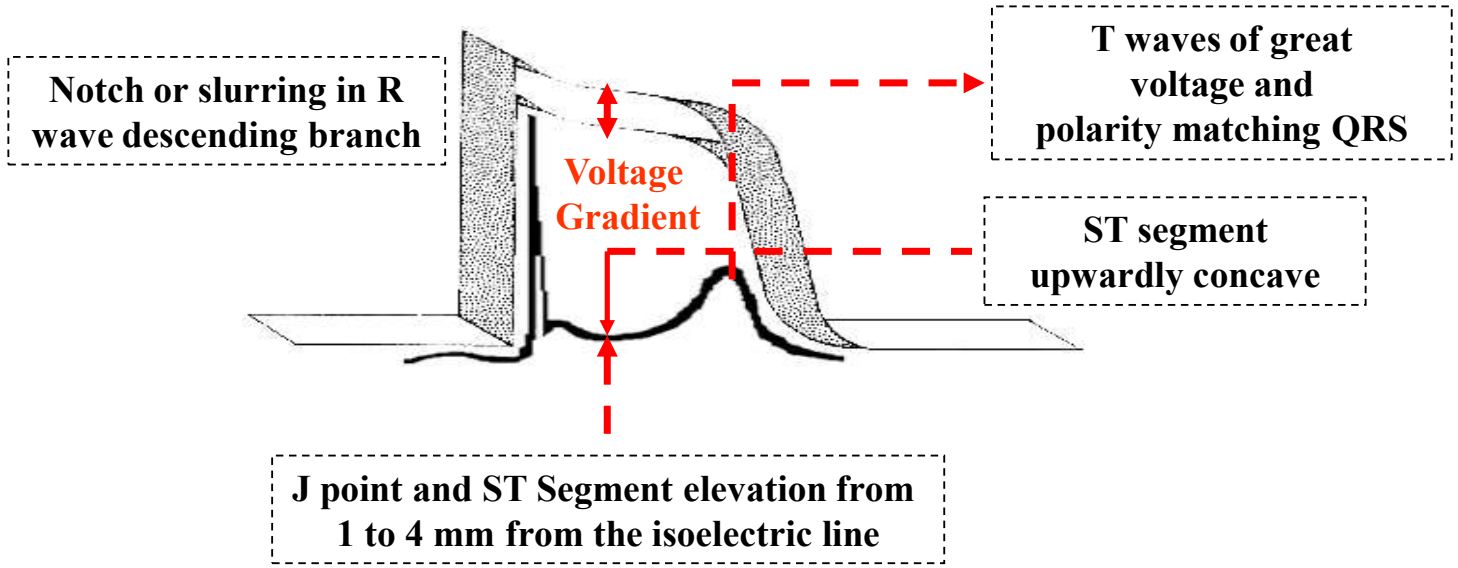
Benign Early Repolarization Pattern in the lateral precordial leads (V4-V6, II, aVF and minimal in I)



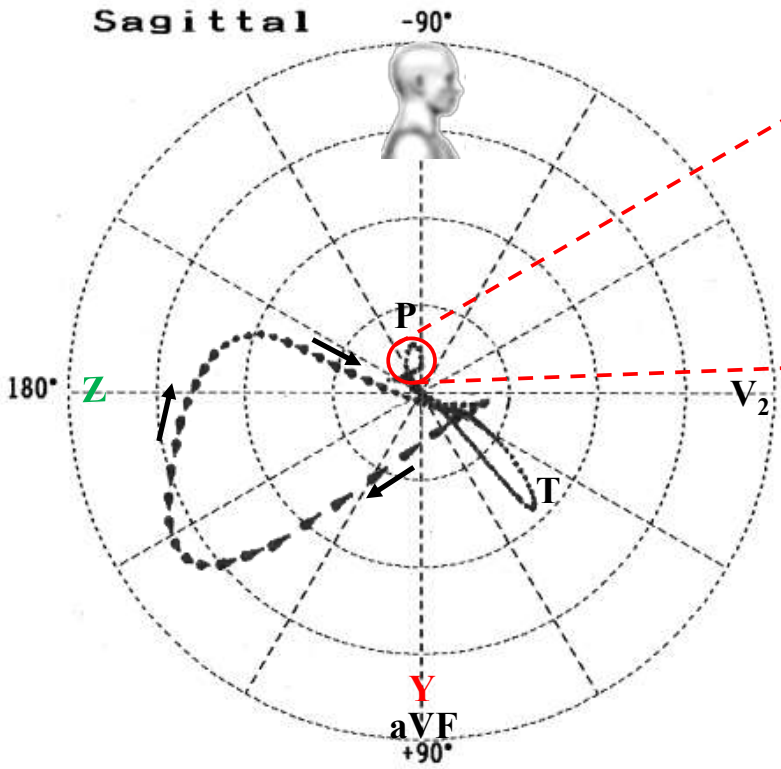
J point and ST segment elevation, usually < 2 mm (exceptionally it may be > 5 mm) of superior concavity in middle and/or left precordial leads and possibly in inferior leads, followed by pseudo symmetrical T waves, with great width and polarity matching QRS. Transition area in precordial leads of sudden occurrence.

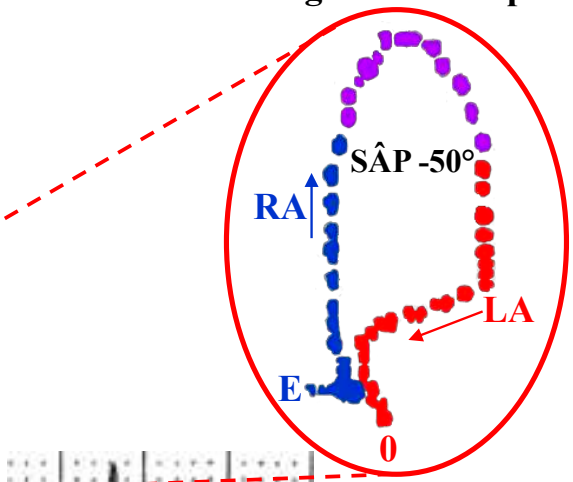
ECG criteria that suggest benign Early Repolarization Pattern (ERP) (Pérez-Riera 2012)

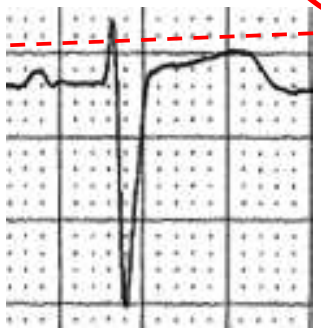
- HR: sinus bradycardia is frequent;
- Axes of QRS, ST segment and T wave, are oriented in the same direction in the FP;
- Deep and narrow Q waves followed by R wave of great voltage in left precordial leads;
- Notch or slurring of R wave descending branch;
- Transition area in precordial leads of sudden occurrence;
- J point and ST segment elevation, usually < 2 mm (exceptionally it may be > 5 mm) of superior concavity in middle and/or left precordial leads and possibly in inferior leads;
- Possible reduction in J point and ST segment elevation by sympathetic action and sympathomimetic drugs;
- Absence of reciprocal or mirror image (exception in VR lead);
- Symmetrical T waves, with great width and polarity matching QRS;

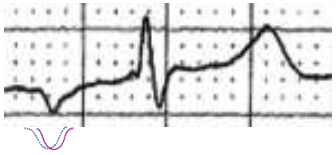


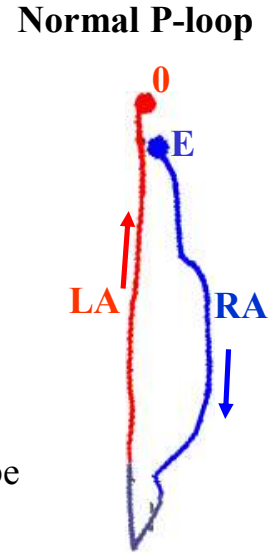
ECG/VCG correlation in the right sagittal plane











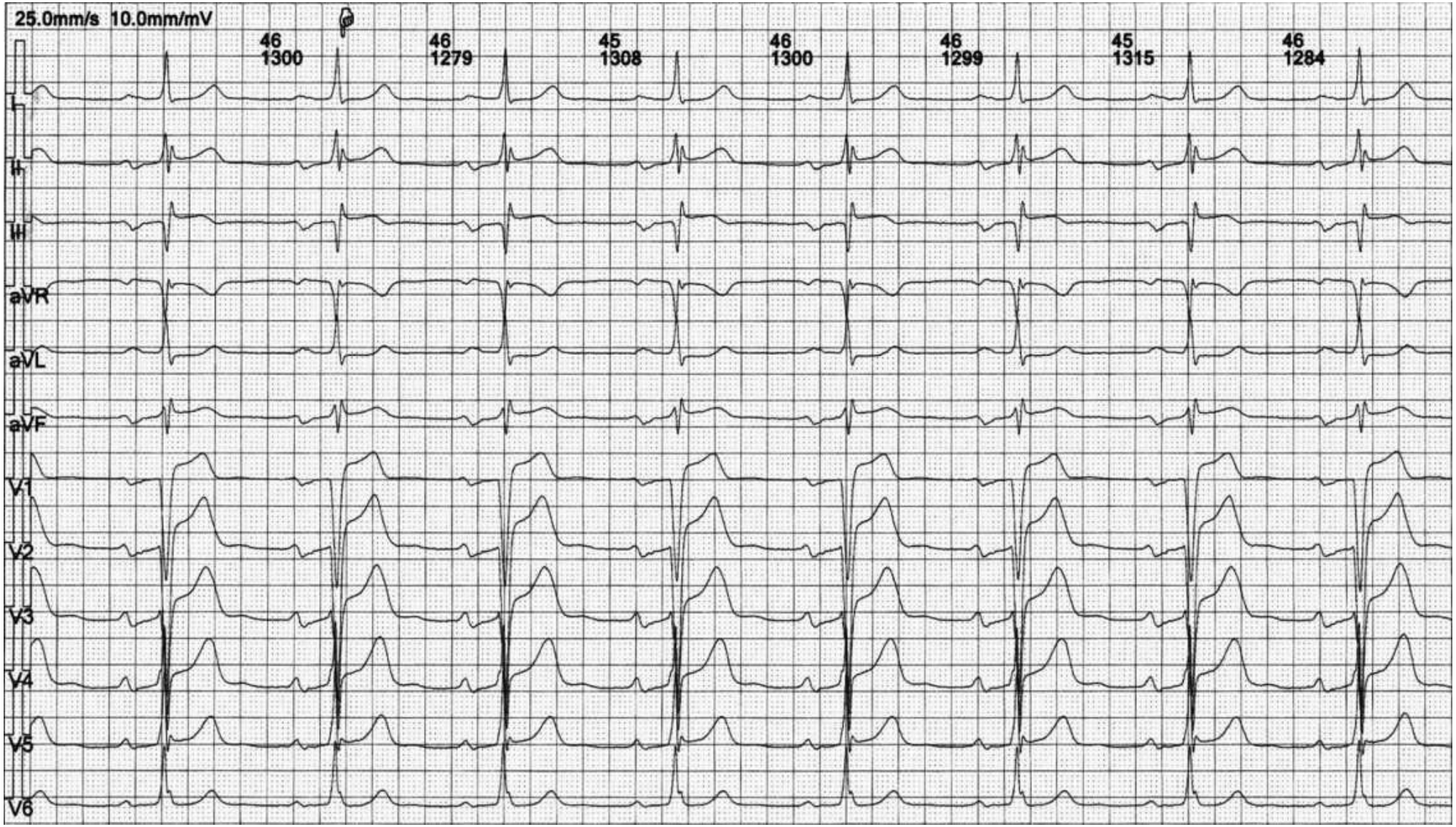
Right atrial activation

Biatrial activation

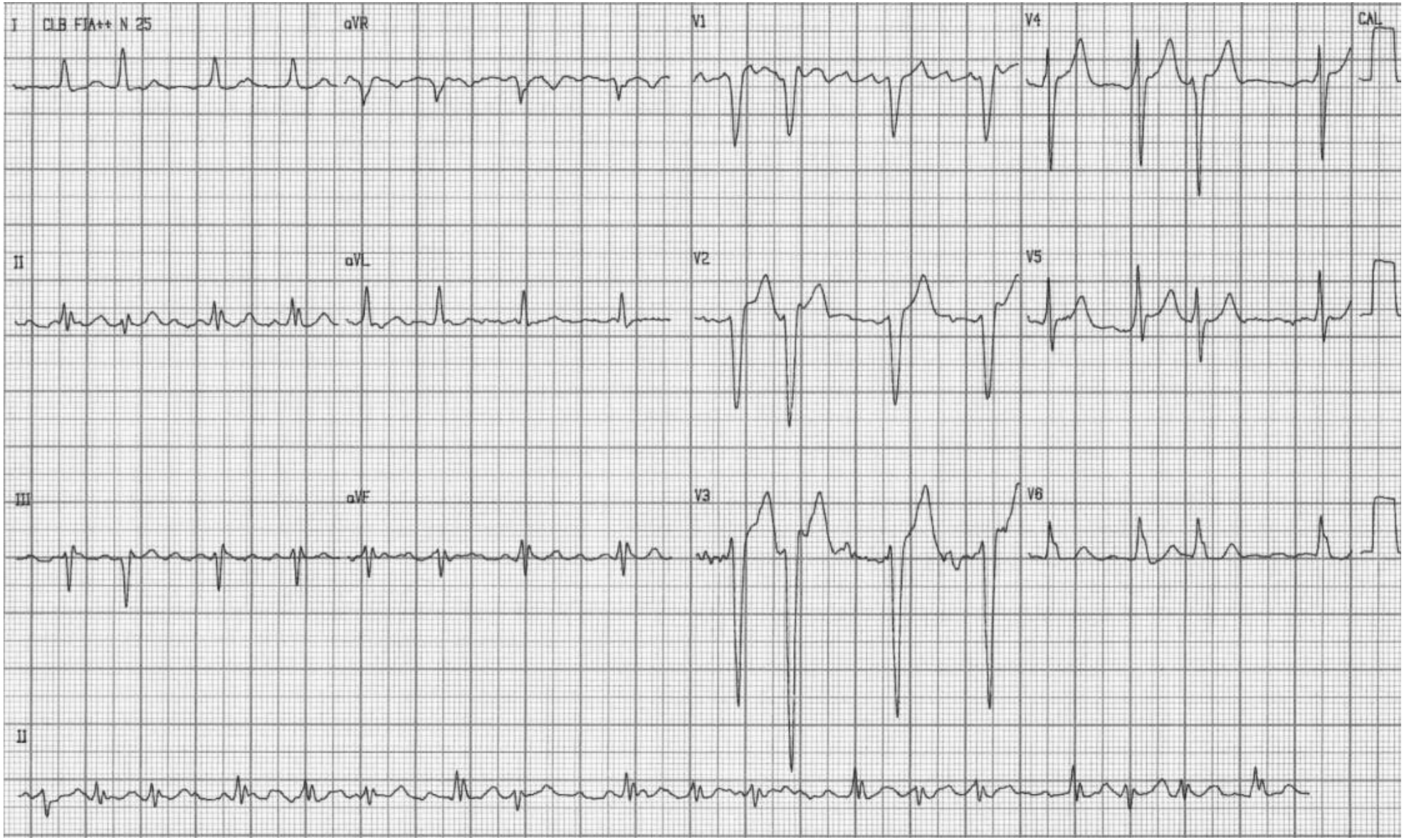
Left atrial activation

Conclusion: Low ectopic atrial rhythm with origin focus near the coronary sinus ostium. The differential diagnosis should be made with advanced or complete interatrial block (Bayés' syndrome). Explanation in the next slides.

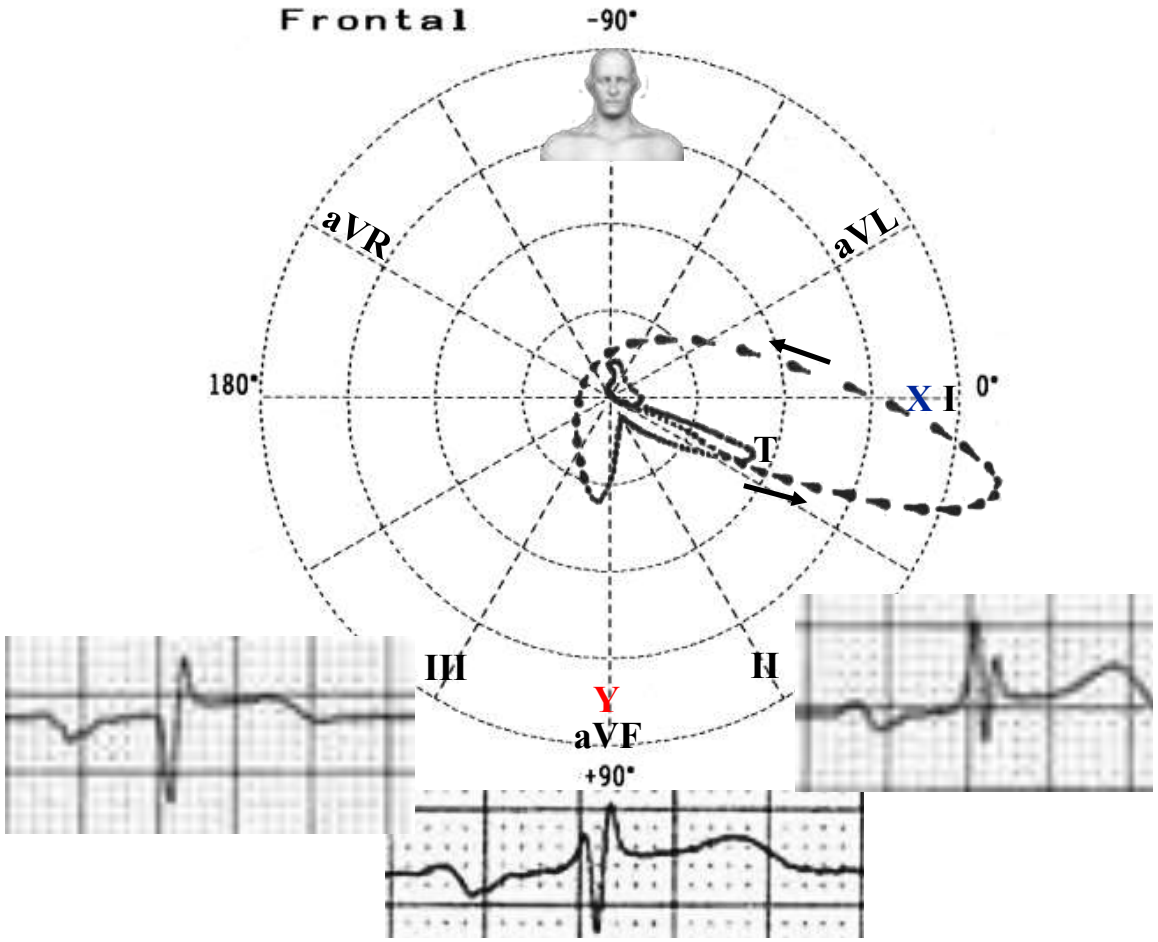
Example of complete or advanced interatrial block (Bayés de Luna 1979) in a patient with Hypertrophic Cardiomyopathy (ECG performed on 2014)

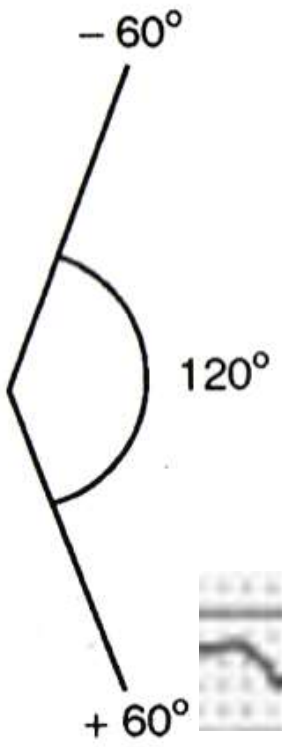


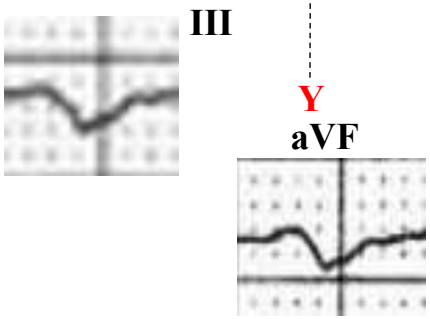
ECG performed on 2015, one year after the same patient in atrial fibrillation

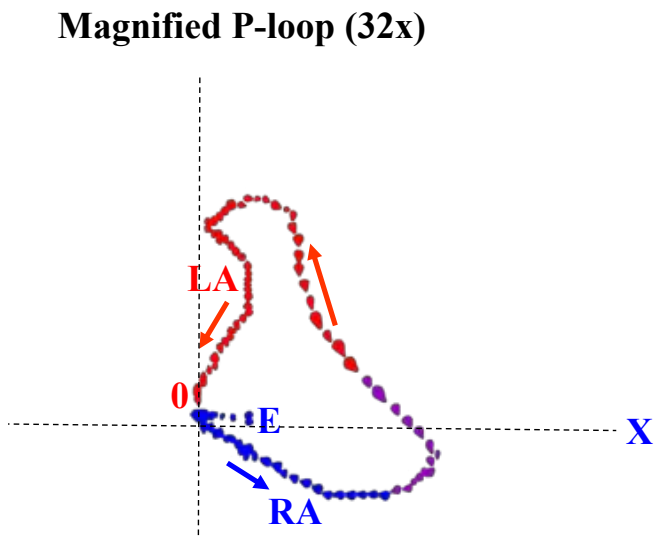


ECG/VC correlation in the frontal plane



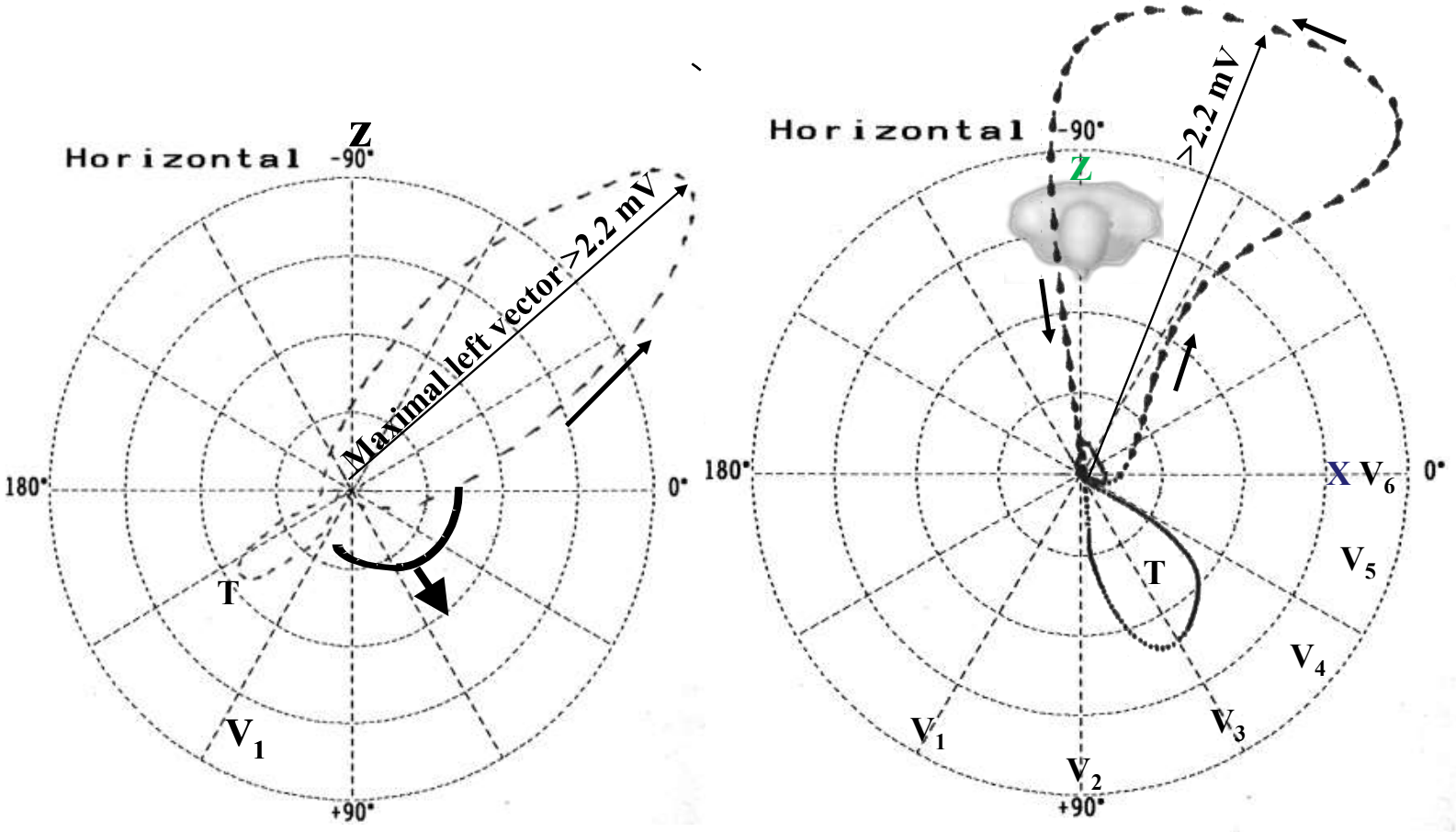




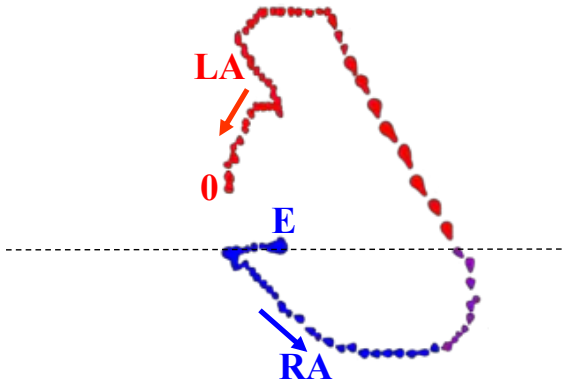


Electrical impulse is blocked/delayed in Bachmann's region (BR), in the upper part of the interatrial zone but retrograde left atrial activation usually occurs (**Ariyarajah 2005**). Note the existence of an open angle between the vector of the first portion of the P-wave (**RA**) and the last one (**LA**). Electrophysiological study demonstrates retrograde activation of the **LA**. Consequently, P loop/wave in orthogonal lead "Y", aVF and III is biphasic "plus-minus" ±. **LA** activation occurs by an alternate route (caudo rather than proceeding from right to left via the BB (**Spodick 2007**).

ECG/VC correlation in the horizontal plane

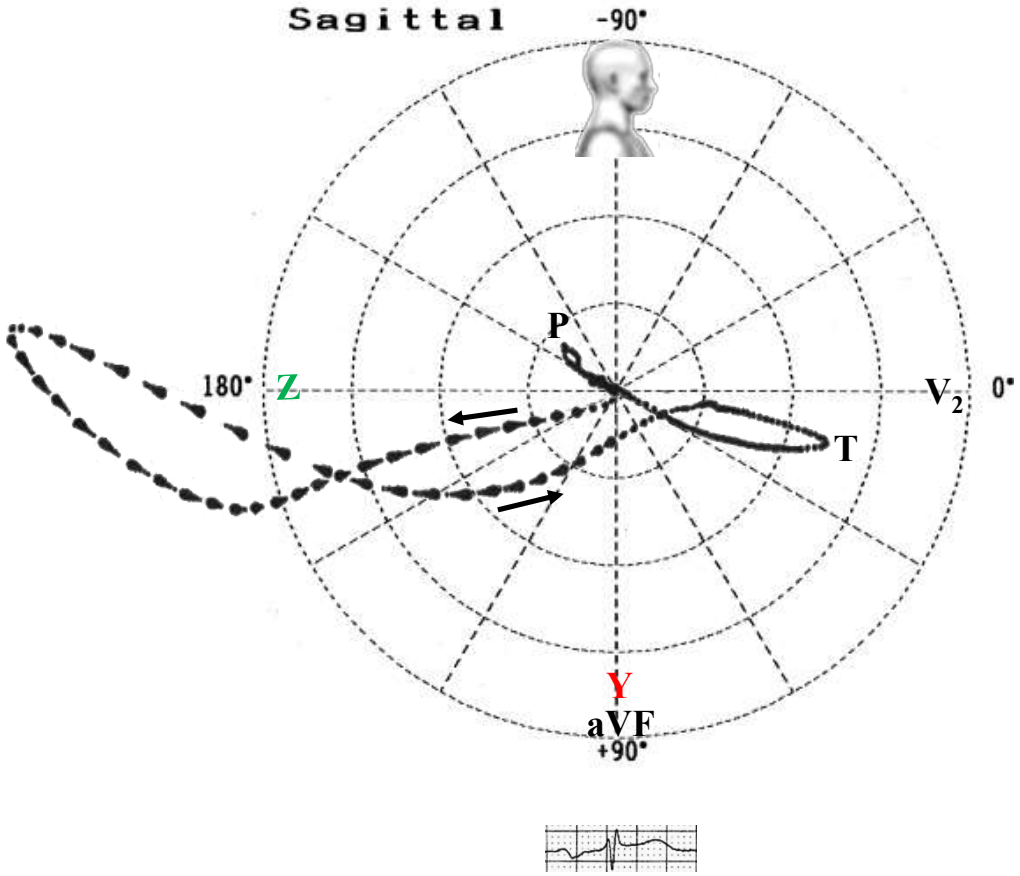


Magnified P-loop (32x)

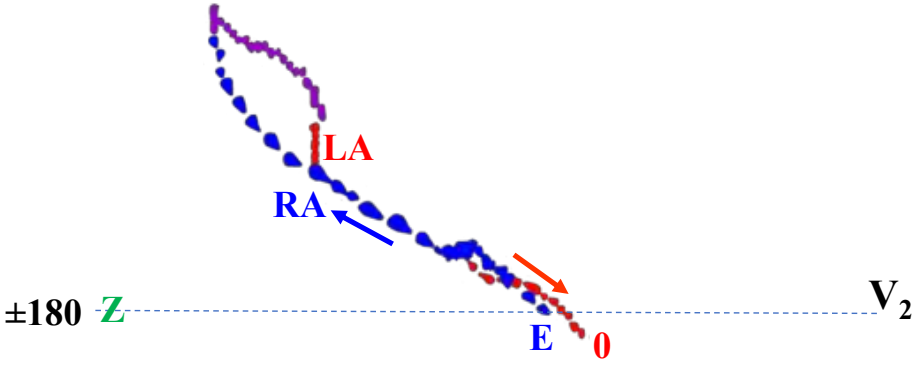


Instantaneous maximal left vector >2.2 mV in LVH Repolarization abnormalities: Deviation of the ST segment and the T wave in the opposite direction to the main QRS loop causes widening QRS amplitude and wide QRS-loop /T angle (QRS/T angle near 160º): LVH with Left Ventricular Strain Pattern.

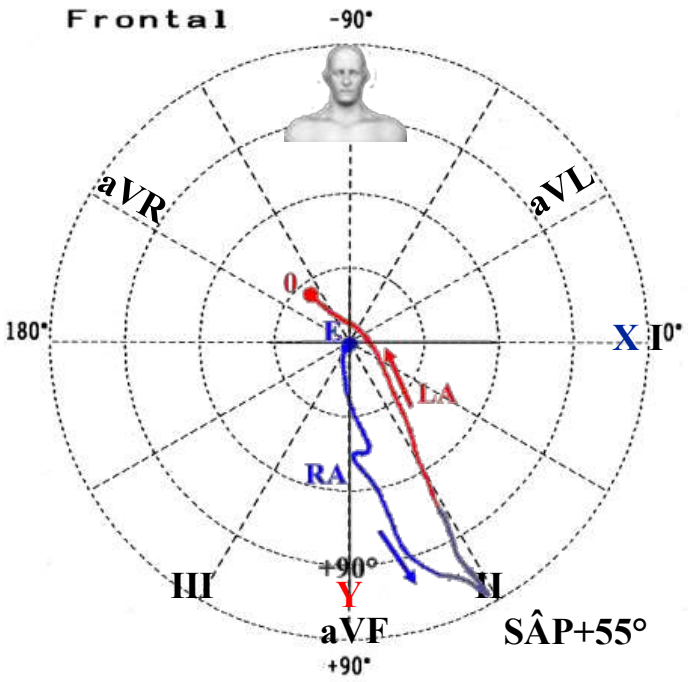
ECG/VC correlation in the right sagittal plane

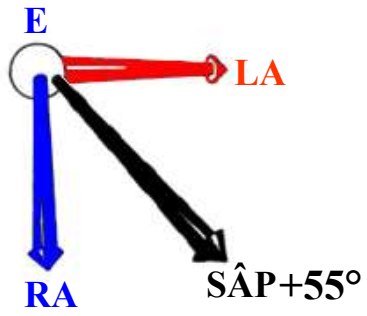


Magnified P-loop (32x)



Normal P-loop in the frontal plane

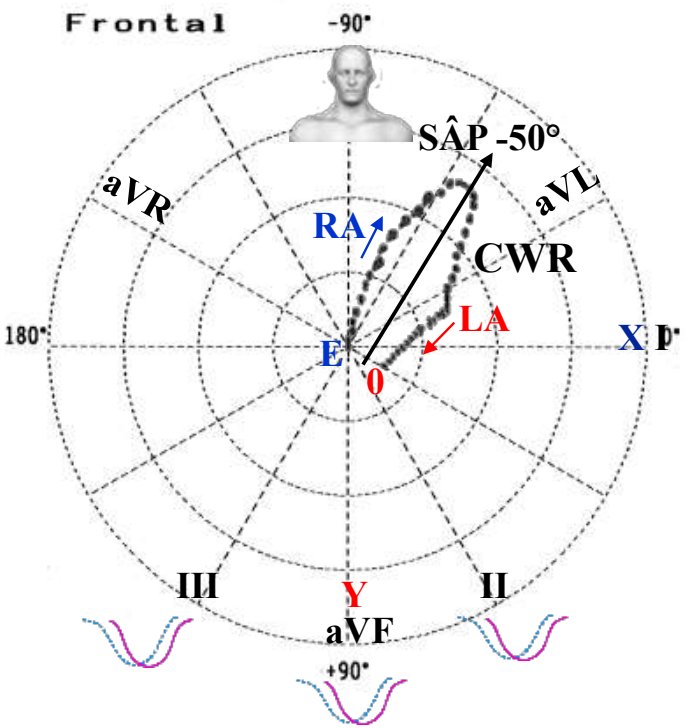




Positive P-wave in II-aVF, and variable polarity in III (plus-minus or positive).

CCWR: Counter Clock Wise Rotation

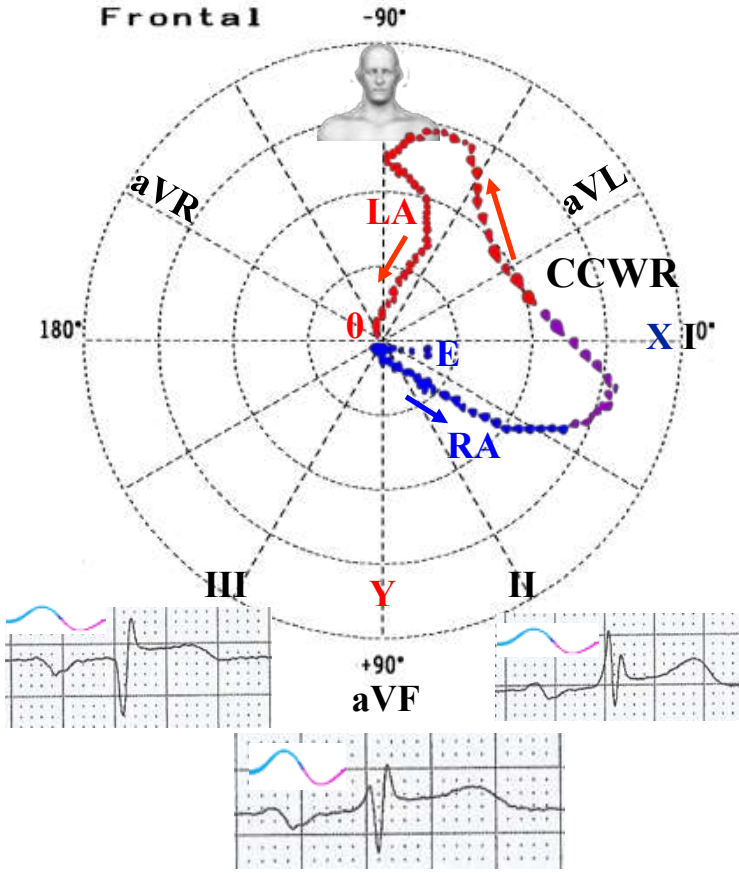
Frontal plane P-loop in Low ectopic atrial rhythm



CWR: Clock Wise Rotation

Negative P-wave in the inferior leads.

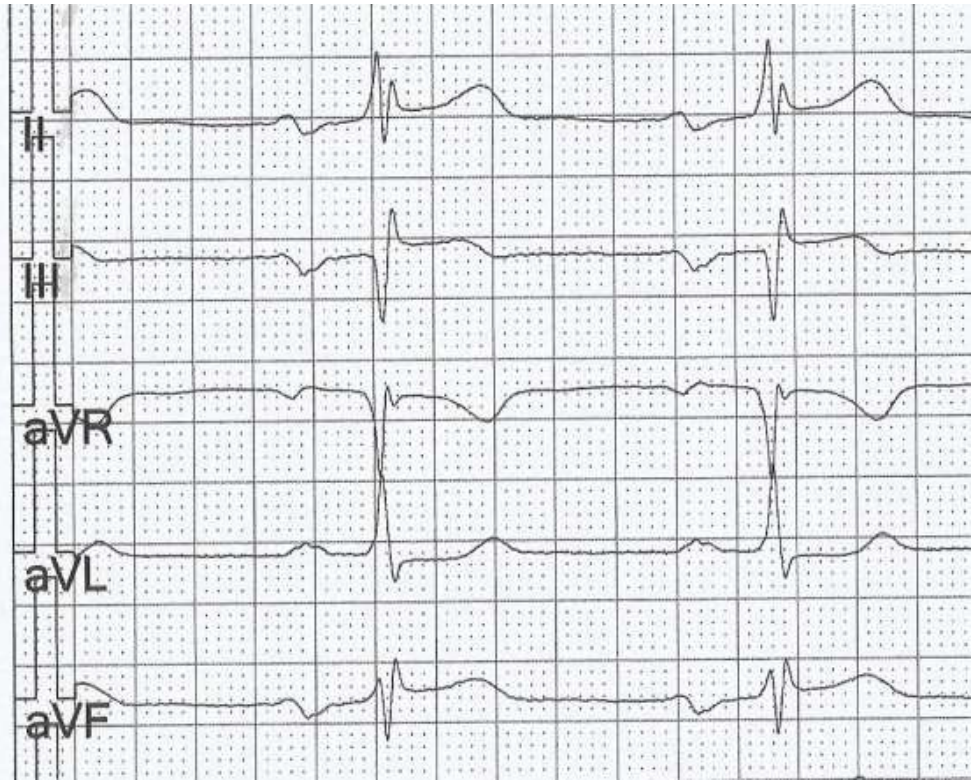
Frontal plane P-loop in the frontal in Advanced or complete interatrial block

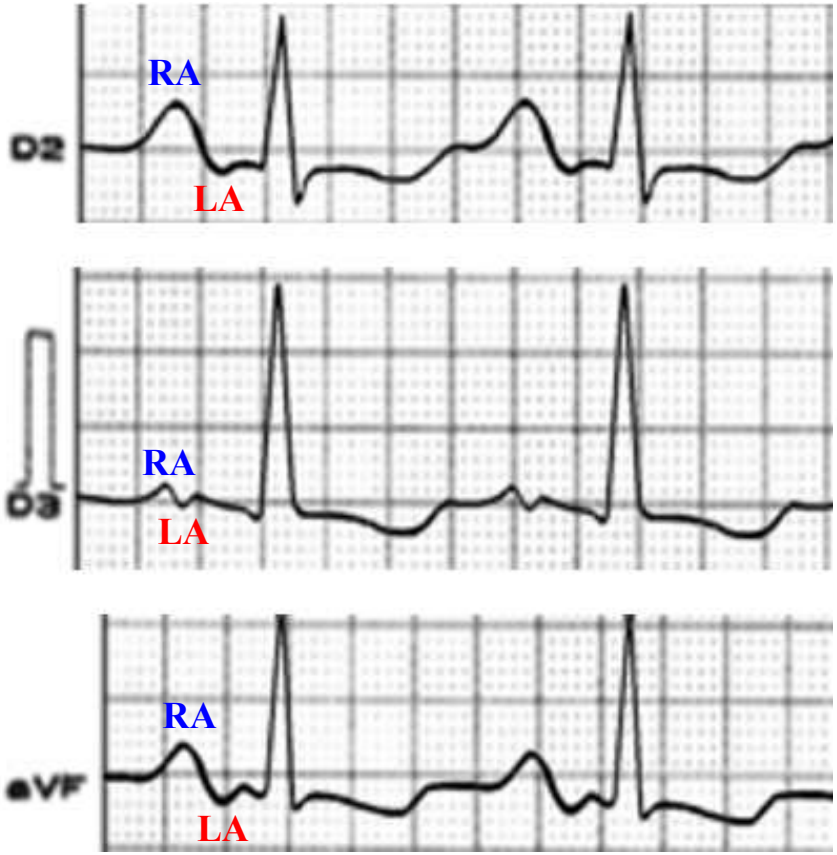


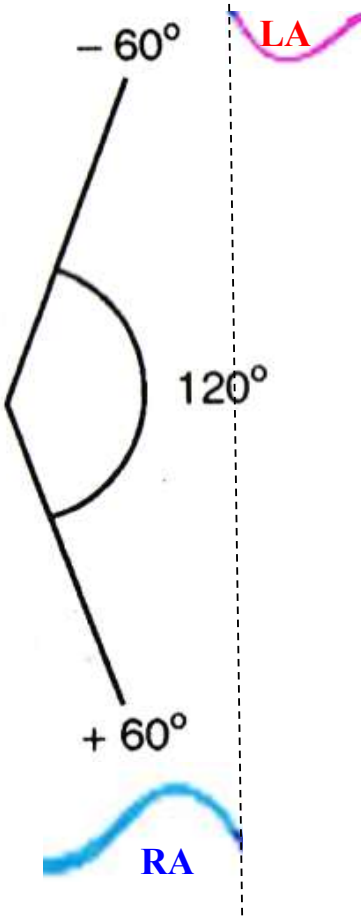
CCWR: Counter Clock Wise Rotation

Open angle >90º between the first and the second part of the P-loop: ≥50 ms above the X or Z axis in its initial portion. biphasic (positive/negative) polarity or "plus-minus" P-loop-wave.

Complete or advanced interatrial block in inferior leads







The diagnostic criteria for Interatrial Conduction Disturbances with Left Atrial Retrograde Activation (IACD-LARA) are (Bayés de Luna 2017; 2012; 1998; 1989;1988; 1985; Conde 2014; Baranchuk 2013):

I. Electrocardiogram

- ECG: biphasic "plus-minus" P +/- in II, III and VF with
- P- wave duration ≥120 ms. Studies published before the 2012 consensus frequently use the cut-off of 110 ms instead of the correct one (120 ms).The measurement methods of P wave length vary between studies (automatic, semiautomatic, manual). Several studies use only one ECG lead (II) or the lead with the longest P wave instead of selecting the first initiation of P wave and the last finish in any lead **(Ninios 2017).**

II. Vectorcardiogram

- Open angle >90º between the first and the second part of the P.
- Orthogonal ECG: P +/- in Y lead with a negative mode greater than 40 m.
- More than 50 ms above the X or Z axis
- Open angle between the two parts of the P loop in both the frontal and right sagittal planes
- Presence of notches and slurring in the last part of the P-loop.
- Activation of the LA have to follow a caudo-cranial activation (through the coronary sinus)

III. Esophageal ECG: High esophageal leads minus-plus with delayed inscription and Low esophageal leads Plus-minus P wave

IV. Intracavitary ECG: craniocaudal sequence in **RA** and caudo cranial sequence in **LA** combined with oseophageal ECG.

V. Magnetic Resonance Image

- Presence of atrial fibrosis detected by the new delayed enhancement magnetic resonance imaging (MRI) techniques **(Lacalzada 2017).**

VI. Non invasive electro-mapping techniques

VII. 3D speckle-tracking echocardiography: that allows to measure the **LA** strain **(Domsik 2014).**

Summary: P-wave polarity, shape, voltage, duration reveals several aspects of the atria: Proper function, fibrosis, desynchrony, and activation paths can be inferred from the surface P-wave analysis. The ECG/VCG can help differentiating atrial enlargements of the atria from conduction defects including intra- and interatrial block.(**Baranchuk 2015**). P-wave morphology does not depend on the size of the atria in young, healthy athletes, and that PTF is not a reliable marker of left atrial enlargement in the current population (**Petersson 2014**).

Differential diagnosis between Low ectopic atrial rhythm and complete interatrial block

	Low ectopic atrial rhythm	Complete interatrial block Bayés´s syndrome
Predominance of parasympathetic activity	Frequent	No
Age	Young adult	Elderly.(**Martínez-Sellés 2016**) high prevalence in the elderly, particularly in those with heart disease. (**Martínez-Sellés 2017**)
Causes	Vagotonia	Mainly delayed conduction between the right and left atrium in the upper part of the interatrial zone around the Bachmann's bundle. Atrial fibrosis increased axial resistance and the gap junctions between cardiomyocites, and decreased maximum upstroke velocity of AP due to reduced Na current density.
Normal heart	Yes.	No
Structural heart disease	Rarely. Endurance male athletes are more likely to develop AF than non-athletes. Structural remodelling of the left atrium(LA), elevated LA pressure, inflammation, myocardial fibrosis, vagal tone, sinus bradycardia and genetic predisposition. (**Flannery 2017**).	Present (**Baranchuk 2017**) coronary artery disease (**Alexander 2017**) The presence of diffuse coronary artery disease defined as >1 significant coronary artery lesion is associated with IAB, patient in hemodialysis **(Marano 2016),** Chagas cardiomyopathy **(Enriquez 2014),** valvular heart disease (**Wu 2016**), heart failure, patients undergoing cardiac resynchronization therapy, Friedreich's ataxia (**Panas 2010**), obstructive sleep apnea (**Baranchuk 2011**), hypertension, stroke (**Ariyarajah 2007**). Brugada syndrome (**Barbosa-Barros 2017**).
P-wave polarity in inferior leads II, III and VF with P	Negative	P +/- "plus-minus". ≥50 ms above the X or Z axis
P-loop/wave axis	Left axis deviation	First portion +30° last portion extreme left axis deviation

	Low ectopic atrial rhythm	Complete interatrial block Bayés´s syndrome
P-loop/wave duration	≤110 ms. Rarely prolonged	≥120 ms
Atrial fibrillation(AF)	Rare. Only elite athletes	High association with atrial arrhythmia, more specifically AF. AF prevalence raises from 6-7% in those aged 65-74years to 13-17% in those >75years.(**Vicent 2017**)
Predictor of complications	No	Supraventricular arrhythmia and cardioembolic cerebrovascular accidents.(**Betancor 2017**) in multiple clinical scenarios.
P-wave polarity in inferior leads II, III and VF with P	Negative	P +/- "plus-minus". ≥50 ms above the X or Z axis
P-loop/wave axis	Left axis deviation	Firth portion + 30° last portion extreme left axis deviation
P-wave polarity in inferior leads II, III and VF with P	Negative	Biphasic P wave +/- "plus-minus". ≥50 ms above the X or Z axis
P-loop/wave axis	Left axis deviation	Firth portion + 30° last portion extreme left axis deviation
P-loop/wave duration	≤110 ms	≥120 ms
P-loop rotation	Clockwise	Counter Clock Wise
CHADS2 score (Congestive Heart failure, hypertension, Age ≥ 75 years (doubled), Diabetes, Stroke (doubled), Vascular disease, Age 65–74 years, Sex category (female sex)	Low CHADS2 score	IAB and a high CHADS2 score independently and synergistically predict new onset AF in patients in sinus rhythm, indicating an approximately 12-fold higher risk in patients with both IAB and a high CHADS2 score (**Wu 2016**). Anticoagulant therapy may be initiated irrespective of documented AF (**Tischer 2014**) anticoagulant drugs could probably be used in the presence of high CHA2DS2VASc, supraventricular ectopic activity, and advanced IAB with high risk of atrial arrhythmias (Bayes syndrome) to prevent cognitive impairment and embolic stroke (**Baranchuk 2014; 2016**)

Reasons why interatrial atrial blocks are little known

1. Most of the literature studies employ only lead II or a combination of 2 or 3 leads
2. Any of the 12 leads of the standard ECG may have the widest P waves, which establish the magnitude of block. Given the high prevalence of IAB in hospital patients and its ominous portents (LA enlargement, thrombosis and embolism, arrhythmias), physicians should be aware of its frequency and computer software should be programmed to recognize it.
3. Much of the literature concerning IAB has loosely named it for one of its correlates in nearly every case; in IAB an abnormally wide P is seen on ECG and IAB should be named by its precise name
4. ECGs encoding systems do not have a code for IAB.

Bayés de Luna A et al. (**Bayés de Luna 1988**) studied 16 patients with ECG evidence of advanced interatrial block with retrograde activation of the left atrium (LA): P duration ≥120 ms, and plus-minus (+/-) biphasic P waves in inferior leads II, III, and VF. Eight patients had valvular heart disease, four had dilated cardiomyopathy and four had other forms of heart disease. Patients with valvular heart disease and cardiomyopathy were compared with a control group of 22 patients with similar clinical and echocardiographic characteristics, but without this type of interatrial block. Patients with advanced interatrial block and retrograde activation of the LA had a much higher incidence of paroxysmal supraventricular tachyarrhythmias (93.7%) during follow-up than did the control group. Eleven of 16 patients (68.7%) with advanced interatrial block and retrograde activation of LA had atrial flutter (atypical in seven cases, typical in two cases, and with two or more morphologies in two cases). Six patients from the control group (27.7%) had sustained atrial tachyarrhythmias (five atrial fibrillation and one typical atrial flutter). The atrial tachyarrhythmias were due more to advanced interatrial block and retrograde activation of LA and frequent PACs than to LAE, because the control group with a LA of the same size, but without advanced interatrial block and retrograde activation of LA and with less incidence of PACs, had a much lower incidence of paroxysmal tachycardia. Bayés de Luna et al.(**Bayés de Luna 1989**) demonstrated the value of preventive antiarrhythmic treatment in patients with advanced interatrial . In this population LAE is present in 90% of cases. Using drugs (amiodarone, quinidine or verapamil) this percentage was greatly lowered (25%). Tachyarrhythmias such as atrial fibrillation and atrial flutter in advanced IAB is observed in >90% of cases. From 81,000 ECGs, Bayes de Luna et al (**Bayes de Luna 1985**) collected 83 cases that fulfilled the criteria of Interatrial Conduction Disturbances with Left Atrial Retrograde Activation (IACD-LARA) (P +/- in II, III and VF with P width ≥120 ms). The authors present the detailed study of 35 cases with surface ECG and VCG and 29 cases with orthogonal ECG leads. The results are then compared against two control groups: with cardiopathy (30 cases) and without cardiopathy (25 cases).The prevalence of IACD-LARA was nearly 1% globally, and 2% among patients with valvular heart disease.

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