THE ECG IN ATRIAL ELARGEMENT AND ATRIAL BLOCK

A.Bayés de Luna, A. Perez-Riera, A. Baranchuk

1. Introduction

For some authors the name "atrial abnormalities" encompass the concepto f atrial enlargement and atrial blocks (1-3). In this paper we will expose the ECG characteristic of both concepts, emphasizing, as happen with ventricular hypertrophy and ventricular blocks that often the ECG pattern of atrial enlargement especially of left atrial enlargement is explained by the coexisting of interatrial block. However as we will see later there are clear evidences that the pattern of interatrial block may exist without the association of atrial hypertrophy. Therefore atrial blocks and atrial enlargement are separate entities that are often associated with each other. Therefore we do not consider it appropriate to use the "umbrella" term atrial abnormalities to include both concepts without distinguishing between them (3), as some authors do (1) (2).

In this paper, we comment separately both copies atrial enlargement and atrial block but we will comment their interactions.

2. Atrial enlargement

2.1 Concept

The ECG expression of atrial enlargement is due more to atrial dilation than atrial hypertrophy because the atrial wall is very thin and when submitted to an increase in pressure it usually dilates before increasing its myocardial mass (4).

The standard techniques to correlate ECG changes with the presence of atrial enlargement were previously based on anatomic, radiologic, and hemodynamic standards (5, 6). The necropsic studies used were feasible only in cases of very advanced heart disease, and thus their utility was limited. For more than 35 years, M-mode echocardiography and especially 2D echocardiography have been considered the "gold standard" (7-9) (Tables 1 and 2).

Cardiovascular magnetic resonance (CMR) is currently the gold standard for atrial volume assessment. It has recently been demonstrated that 2D transthoracic

echocardiography consistently underestimates the left atria (LA) and right atrial (RA) volume compared with CMR imaging (≈15-20%) (10). However, as the volumes assessed by the two techniques have similar slopes, except for the underestimation by 2D echocardiography, the study performed using CMR to evaluate LA volume would likely result in similar resuslts to those obtained using 2D echocardiography (9). The accuracy of ECG criteria for left and right atrial enlargement as detected by 2D Echocrdiography or CMR has recently been shown as is expressed in Table 1 and 2. For full information consult Bayés de Luna (1).

The sensitivity and specificity of different criteria varies with the methodology used, and particularly with the type of population studied. The specificity is usually much higher and the sensitivity lower, but this increases if the population studied presents with a higher degree of atrial enlargement.

2.2 ECG criteria for right atrial enlargement (RAE)

In clinical practice, the most useful criteria for RAE are those that have the highest specificity (≈50%) (see Table 1) (Figure 1).

- Direct P wave criteria are very specific but their sensitivity is very low.
- The QRS criteria with a QRS amplitude in V1 < 4 mm + ratio V2/V1 > 5 are highly specific (> 90 %) with moderate SE (≈ 45 %)
- The combined P + QRS criteria with a P wave amplitude in V2 > 1.5 mm + ÂQRS > 90° + R/S ratio >1 in V1 in the absence of RBBB have 100 % specificity and ≈ 50 % sensitivity.

2.3 False positive and false negative diagnoses of right atrial enlargement

The ECG diagnosis of RAE may be very difficult to reach for the following reasons:

- The voltage of the P wave is strongly influenced by extracardiac factors (Figure 2), which may result in increases (hypoxia, sympathetic overdrive, etc.) (false positive) or decreases in voltage (emphysema, other barrier factors, atrial fibrosis, etc.) (false negative)

- The presence of associated fixed or intermittent atrial block may result in the transient or permanent disappearance of the ECG criteria for right atrial enlargement (false negative) (Figure 3)
- On the other hand, a high-voltage P wave may be seen in patients with exclusively left heart pathology and possible left atrial enlargement (false positive) (pseudo-P-pulmonale) (11)

These are some of the reasons why changes in the atriogram are generally not very sensitive (many false negative) for the diagnosis of RAE. Although there are some factors that increase the incidence of false positives, they are fewer and therefore the specificity of ECG criteria for RAE is much higher.

2.4 ECG criteria for left atrial enlargement (LAE). The most important ECG criteria for LAE are show in Table 2 (1) (Figure 1)

In clinical practice, the most useful criteria for LAE are those that have the highest specificity with the most acceptable sensitivity (see Table 2 and Figure 1, 4 and 5). Based on this principle, we use the following:

- The Morris index (product of negative amplitude of P wave in V1 in mm x duration in ms ≥ - 40 mm/ms)
- P wave duration in leads I, II and/or III ≥ 0.12s + negative mode of P in V1 ≥ 40 ms.

Moreover, AF is present in the majority of cases (>80%) when the left atrial diameter is >60mm. In a cohort of patients consecutively referred for clinical or research CMR studies, those with a history of AF had a significantly higher prevalence of LAE (47%) than patients with sinus rhythm (21%) (1, 11).

2.5 False positive and false negative diagnoses of left atrial enlargement

The diagnosis of LAE may often be difficult to achieve because of the following factors:

- The presence of isolated interatrial block explains the increase in the duration of the P wave, without the presence of evident teminal negative mode of V1 (1). Thus, in contrast the combination of P wave duration in FP + increase of negative mode of P wave in V1 (Morris index) is a good criterion (6) (see before).
- Some patients with isolated non-advanced left heart disease (e.g. mitral stenosis) without evident interatrial block show peaked P waves with no increase in duration (pseudo-P-

pulmonale). In these cases, the presence of a P wave in V1 with a highly negative mode helps in reaching the correct diagnosis (Figure 4 and 5) (see before).

- If important atrial fibrosis exists, small and even unapparent P waves (concealed sinus rhythm) may be seen, even in the presence of evident left atrial or bi-atrial enlargement (12). This problem increases the number of false negatives (low SE)
- Many patients with COPD or thoracic abnormalities, pectus excavatum, and those with straight-back syndrome present with short but evident negative P waves in V1, a morphology that may be confused with LAE (1).

2.6 Bi-atrial enlargement

The most important diagnostic criteria are as follows (1) (Figure 6):

- The P wave in lead II is taller (≥2.5mm) and wider (≥0.12s) than normal.
- The first part of P wave is positive and peaked in V1-V2 (positive mode > 1.5mm) with a slow negative node (width ≥1mm)
- Signs of left atrial enlargement with right ÂP. The opposite case is not valid because the ÂP can be on the left side in isolated RAE of patients with congenital heart diseases.
- The presence of atrial fibrillation along with QRS changes suggestive of RAE.

Frequently, more then one criterion is found (Figure 6) (P ≥ 120 ms in FP + P ± in V1 with first part peaked and alow negative mode).

1. ATRIAL BLOCKS

Probably atrial blocks are not considered as an individual pattern in the majority of books on ECG, because the ECG patterns found in atrial enlargement, especially in LAE, are in many cases influenced by the presence of interatrial blocks. The term “atrial abnormalities”, that we don no support, has been coined to encompass both atrial enlargement and atrial block (2,3). While it is true that atrial blocks are often associated with atrial enlargements (sometimes this also happens at the ventricular level), the ECG patterns of atrial block are independent entities because there are 3 criteria that define an ECG pattern as being caused by a block or deterioration of conduction (1) (13) (14).

a) The ECG pattern may appear transiently, and the pattern may change abruptly and progressively to more advanced forms;

b) The ECG pattern may appear without associated other processes such as cardiac chamber enlargement or ischemia, although in many cases, one or more of these conditions may coexist; and
c) Similar ECG pattern may be reproduced experimentally (15) (Figure 7)

Various studies performed by Bayés de Luna (15-18) and others, such as Spodick (19, 20), Holmqvist et al. (21), and Platonov (22), have re-evaluated the concept that atrial blocks and atrial enlargement are separate entities, although in many cases, they are associated.
The interatrial block that occurs between the right and left atriaum is well detected by surface ECG and will be discussed in depth. Figure 8, shows the schematic diagnosis of different types of atrial block.
Interatrial blocks, like other types of block, (sinoatrial, atrioventricular, and ventricular) may be first, second (transient block, atrial aberrancy), or third degree.
The evolution from first-degree interatrial block to third-degree (advandec) interatrial block may be seen, as also happens in other types of block.
It is very difficult to identify the presence of a block within a single atrium (intra-atrial block) using surface ECG. We have seen that occasionally apparently normal P waves ($\leq$ 0.12s with some small notches) present with total or near total clockwise rotation of the loop in FP, and we hypothesize that this is due to the presence of a localized block in some part of the right atrium (Figure 8 D and 9). However, the exact diagnosis of right intra-atrial block can only be performed by intracavitary ECG (long high right atrium to low right atrium (HRA-LRA) interval) (Figure 9). In could be say important to perform a new study on that addition, the atrial delay may also be located in the left atrium in some cases of very long and notched P waves, especially if large slurrings are present in the second part of the P wave.

Firs-degree (partial) interatrial block (Figure 8 B and 11 B)

The electrical impulse is conducted normally from the right atrium to the left atrium, through Bachmann's bundle, but with a delay. Therefore, the ECG shows a P wave of $\geq$ 0.12s in leads I, II, or III. This ECG parameter has also been used for diagnosis of LAE. In fact, the delay in atrial conduction, rather than the increased atrial mass, explains the increase of P wave duration.

The P wave morphology in V1 usually presents with a negative mode of the P wave that is less evident than in cases of LAE because the P loop is derected in a less backward direction in isolated first-degree interatrial block.
The intracavitary recording shows that the HRA-coronary sinus distance is increased.
Finally, the prevalence of first-degree interatrial block in the general population is very high . Recently, it has been shown to be associated with atrial fibrillation , and cardiovascular and all-cause mortality.

Third-degree (advanded) interatrial block (Figure 8 C, 10 and 11 C)

Electrical impulse is blocked in the upper and middle part of the interatrial septum in the Bachmann's bundle zone and a little lower, and a retrograde left atrial activation occurs (16). In rare situations, there can be atrial dissociation. Waldo et al (14) demonstrated that cutting the Bachmann's bundle in dogs causes a similar morphology, with P wave + - in inferior leads.
The ECG shows a P wave of ≥ 0.12s and ± morphology in II, III, and VF due to caudocrenial activation of the left atrium . Bayés de Luna defined the ECG-VCG criteria (15) and demonstrated that this type of block is a very specific marker for LAE (SP ≈ 100%). The positive mode of P in II, III, and VF is at times not well seen, probably due to fibrosis, and the diagnosis of junctional rhythm due to an apparently negative P wave in II, III, and VF may be made. We also demonstrated that it is frequently accompanied by paroxysmal atrial arrhythmia and atypical atrial flutter in particular, during follow-up (16-18), especially in valvular heart diseases and cardimyopathies. Therefore, this association is considered an electrocardiographic clinical syndrome (23,24) that has been named Bayés Syndrome (25,26).

Second-degree atrial block

Second-degree interatrial block appears transiently and corresponds to a type of atrial aberrancy . It manifests as a sinus P waves with morphology of first or third degree of interatrial block that vary its morphology:

- Induced by atrial or ventricular premature complexes
- Appear and disappear suddenly and transiently in one ECG
- Show a P wave that changes morphology transiently in successive ECGs, leading to misdiagnosis.

Atrial aberrancy may also present as a transient bizarre P wave without the morphology of first or third degree interatrial block. In figure 12 may be seen two examples of atrial aberrancy one (1) that corresponds to a case of second degree interatrial block and the other.

These changes in P wave morphology are caused by variations in the atrial path of the sinus impulse through the atria. They should be differentiated from changes induced by breathing, atrial fusion beats, and artifacts, including diaphragmatic contraction.

TABLE 1 Right atrial enlargement. ECG criteria with high specificity (1)

ECG criteria	SE %	SP %
A. QRS criteria		
1. QR or qR in V1	≈ 15	> 95
2. QRS V1 ≤ 4 mm + QRS V2/V1 ≥ 5	46	93
3. R/S > 1 in V1	≈ 25	> 95
4. ÂQRS > 90°	34	> 95
B. P criteria		
1. P wave in inferior leads < 2.5mm	7	100
2. Positive part of P wave V1 > 1.5mm	17	100
3. Positive part of P wave V2 > 1.5mm	33	100
C. Combined		
1. Positive part of P wave in B2>1.5mm+ÂQRS>+90°+R/S>1 in V1	49	100

TABLE 2 Left atrial enlargement. ECG criteria based on P wave changes with high specificity (1)

ECG criteria	SE %	SP %
1. Morris index (Morris et al.1964) (P terminal force in V1 mm/ s) (0.04mm/s)	69	93

2. NYAC score P ≥ 120ms in I or II + Morris index (>0.04mm/s) + ÂP ≈ 0°	15	98
3. P ≥ 120ms in lead II + Terminal mode negarive of P in V1 > 40ms	50	87
4. P ≥ 120 ms in lead II + Morris index (>0.04mm/s)	69%	49%
5. Interpeaks of P wave >40ms	15	100
6. P ± in II, III, VF	5	100
7. ÂP beyond + 30°	8	90
8. P wave duration in II or III leads ≥ 0.12s	33%	88%

FIGURES

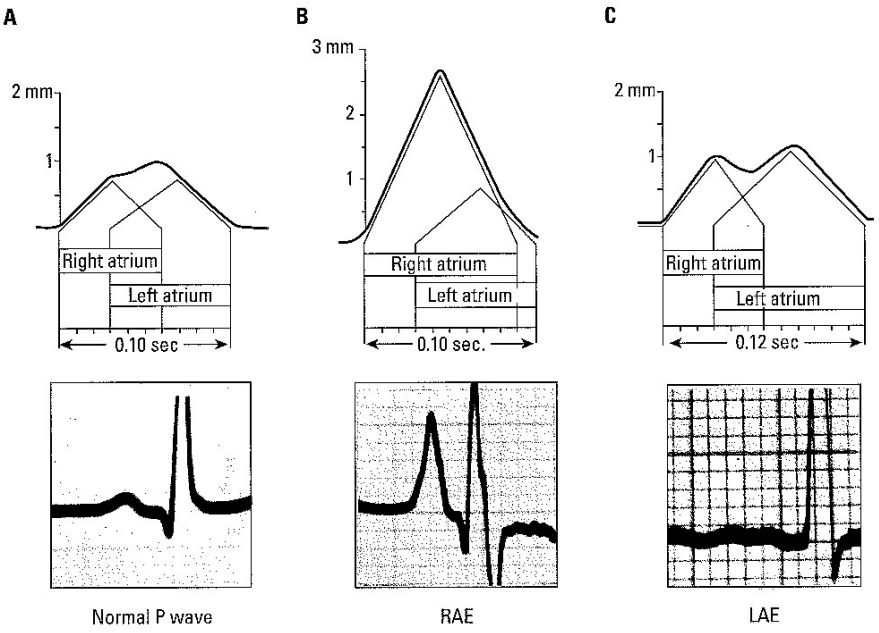

Figure 1 Above: diagram of atrial depolarization in a normal P wave (A), right atrial enlargement (RAE) (B), and left atrial enlargement (LAE) (C). Below: examples of the three types of P wave.

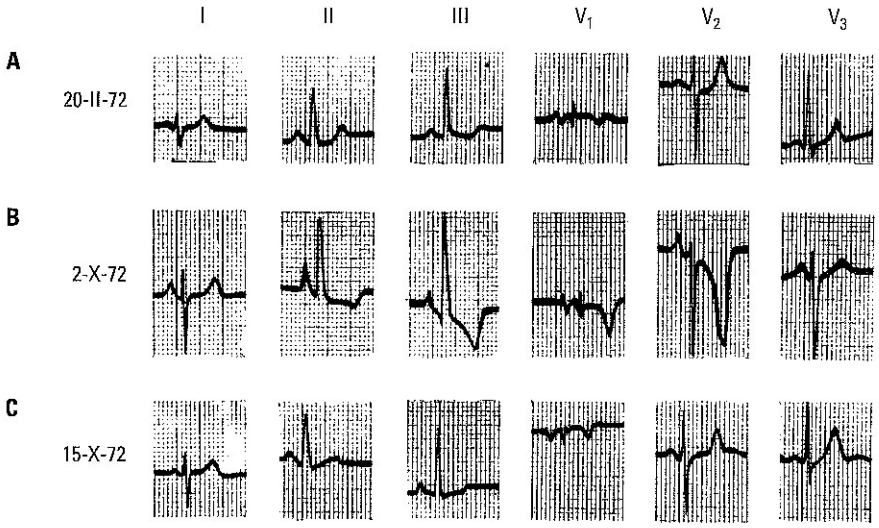

Figure 2 A patient with chronic cor pulmonale and an acute respiratory affection. A tall, peaked P wave (B) that did not previously exist (A) appeared, disappearing a few days later (C). Observe how the negativity of the T wave increases in V1 and V2 in the B tracing.

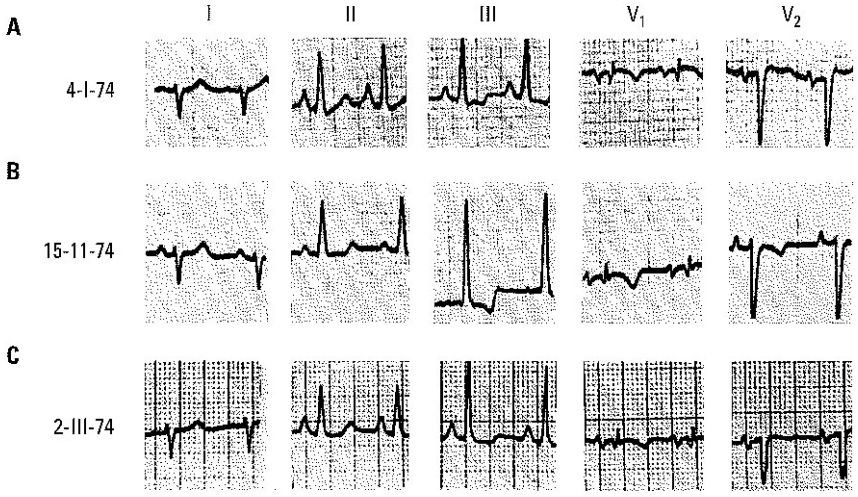

Figure 3 (A) A 45-year-old patient with subacute cor pulmonale. Note the right ÂP. Some days later (B), the ÂP was left, returning to the right in a third ECG (C) recorded at 15 days. This example shows how P waves that fail to suggest right atrial enlargement can be seen in cases where the right cavities are affected by right atrial enlargement due to atrial aberration.

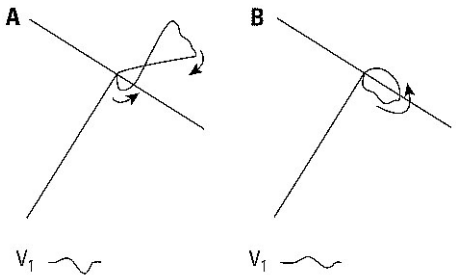

Figure 4 (A and B) P wave morphology in V1 and P loop in a case of isolated partial interatrial block (B) associated with left atrial enlargement (A).

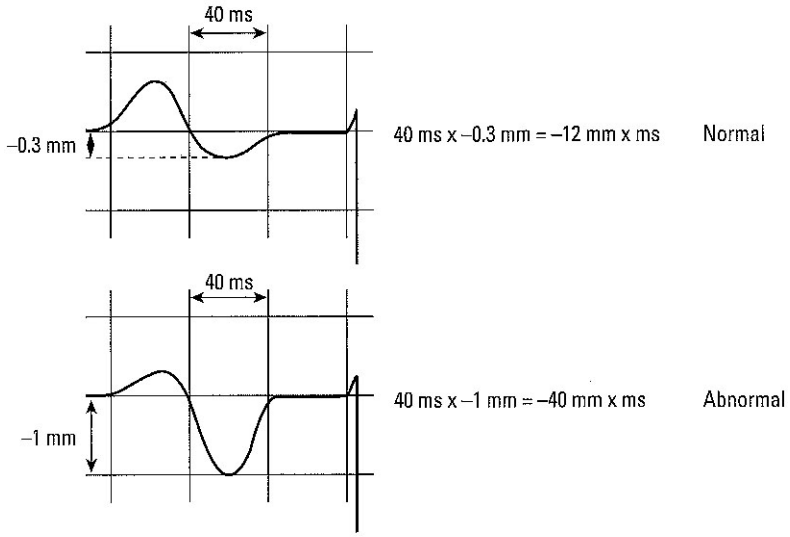

Figure 5 Diagram contrasting normal and abnormal negative components of the P wave in V1. When the value calculated using the width in seconds and the height in millimetres of the negative mode exceeds 40mm + ms, it is considered abnormal.

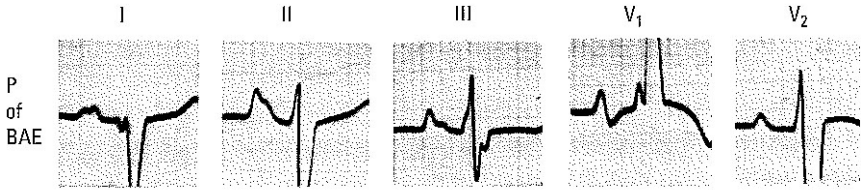

Figure 6 Example of P wave and loop morphology in bi-atrial enlargement (BAE).

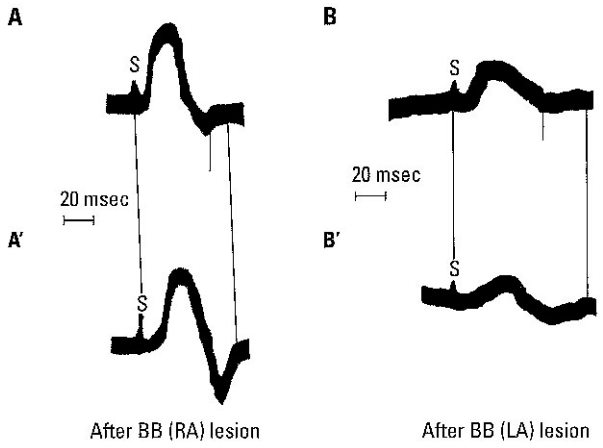

Figure 7 Adapted from experimental Bachmann's bundle block (Waldo et al. 1971). (A) Control P wave recorded in ECG lead II when the atrial were paced from the right atrium. See the change of morphology after Bachmann's bundle lesion in the right side. (B) P wave recorded in lead II after the creation of a lesion in the left atrial (LA) portion of Bachmann's bundle (BB). In both cases the changes in conduction time and morphology after block are shown. (Adapted with permission from Waldo(1971)).

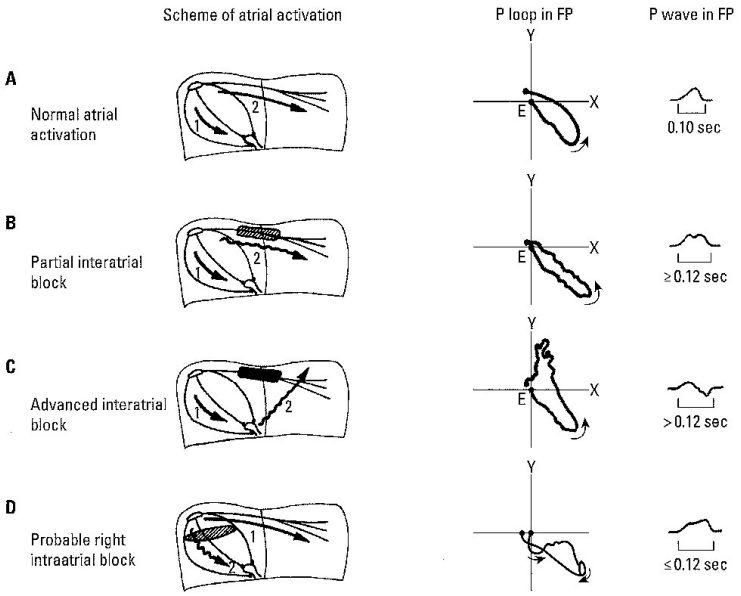

Figure 8 Diagram of atrial conduction under normal circumstances (A), partial interatrial block (B), advanced interatrial block with left atrial retrograde activation (AIB with LARA) (C) and probable right intra-atrial block (D).

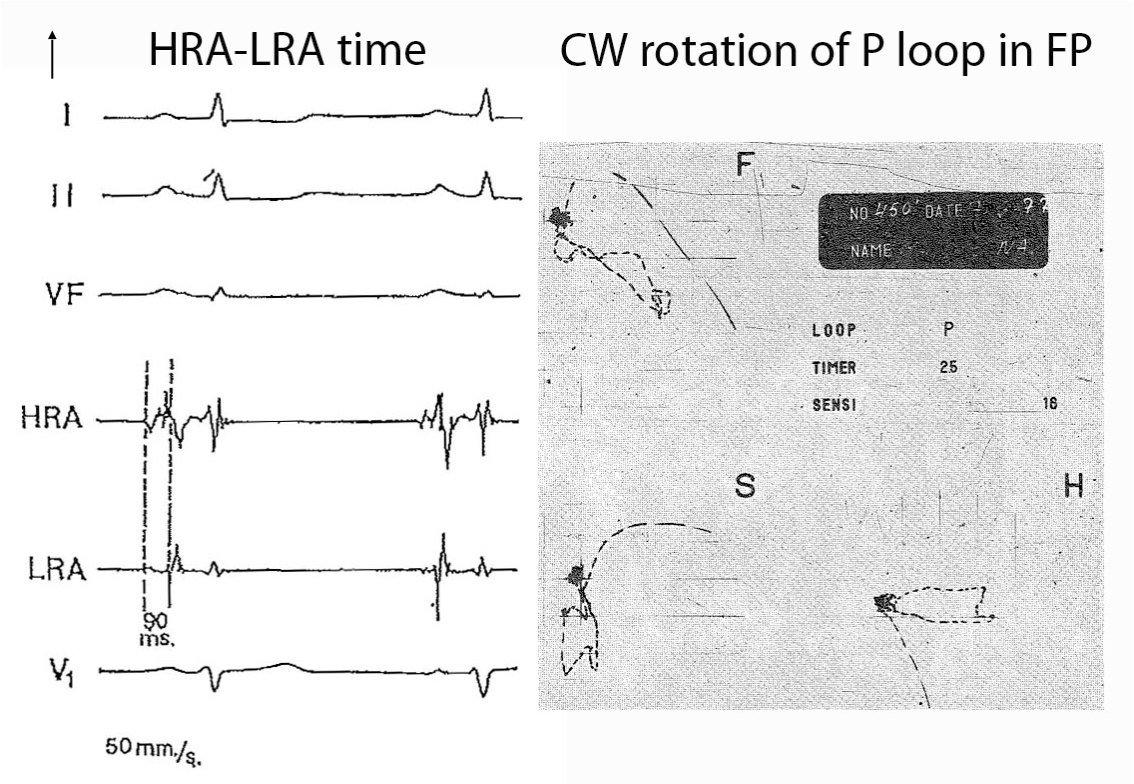

Figure 9 One case of nearly total clockwise rotation of the P loop in frontal plane (FP) that show a normal P wave (morphology and duration) in the ECG. In A the loop also rotates clockwise in horizontal plane (HP), suggesting that the right atrial block is located in the anterior part or the right

atrium. See the increase of time between HRA-LRA that assure the presence of block in the right atrium.

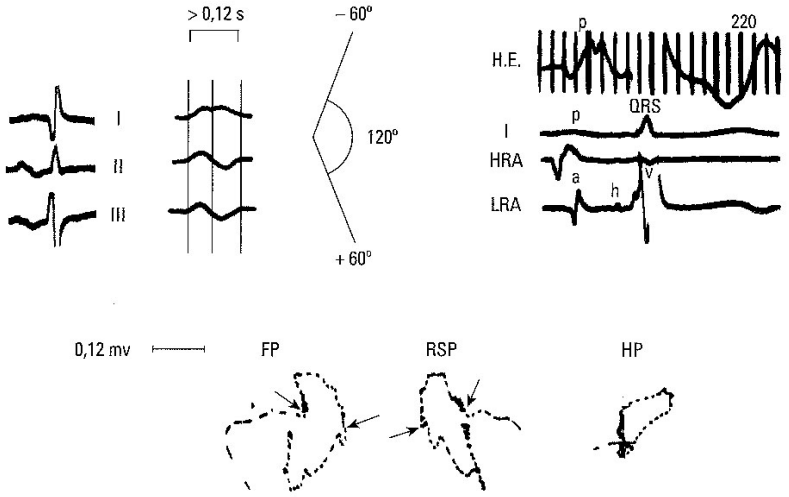

Figure 10 Above: P wave ± morphology in I, II, and III typical of advanced interatrial block with retrograde conduction to the left atrium. Observe how the ÂP and the angle between the direction of the activation in the first and second parts of the P wave are measured. To the right, intra-esophageal ECG (HE) and endocavitary registrations (HRA: high right atrium; LRA: low right atrium) demonstrate that the electrical stimulus moves first downwards (HRA-LRA) and then upwards (LRA-HE). Below: P loop morphology in the three planes with the inscription of the second part moving upwards.

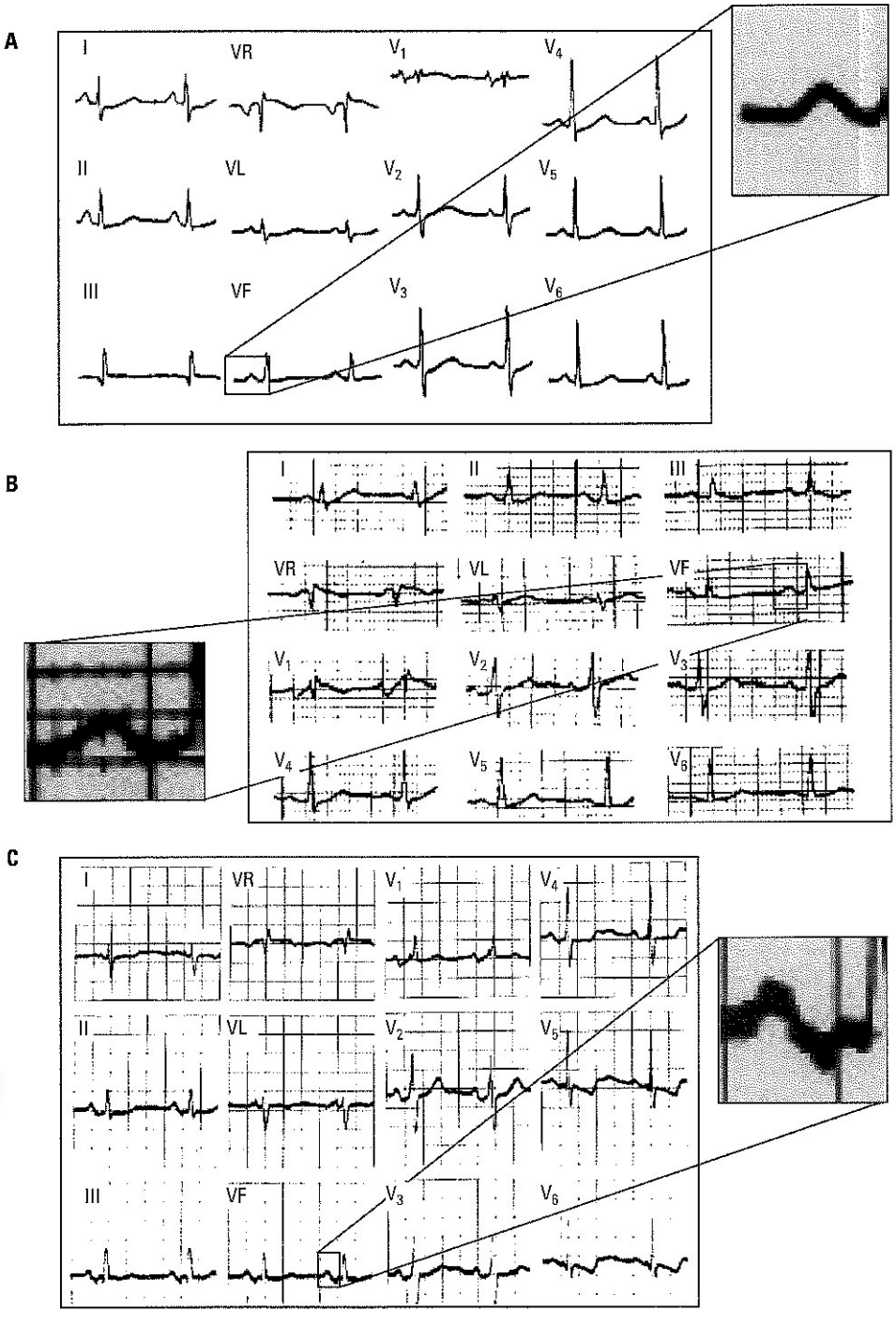

Figure 11 Probressive interatrial block: Three ECGs from a patient with mitro-aortic valve disease. (A) P wave in II, III, and VF with normal atrial duration (P=105ms) and P wave of pseudo P pulmonary type. (B) An intermediate morphology that corresponds to a first-degree interatrial block (P=135ms). (C) Advanced interatrial block appearing after 5 years with P ± morphology in II, III, and VF (P=145ms).

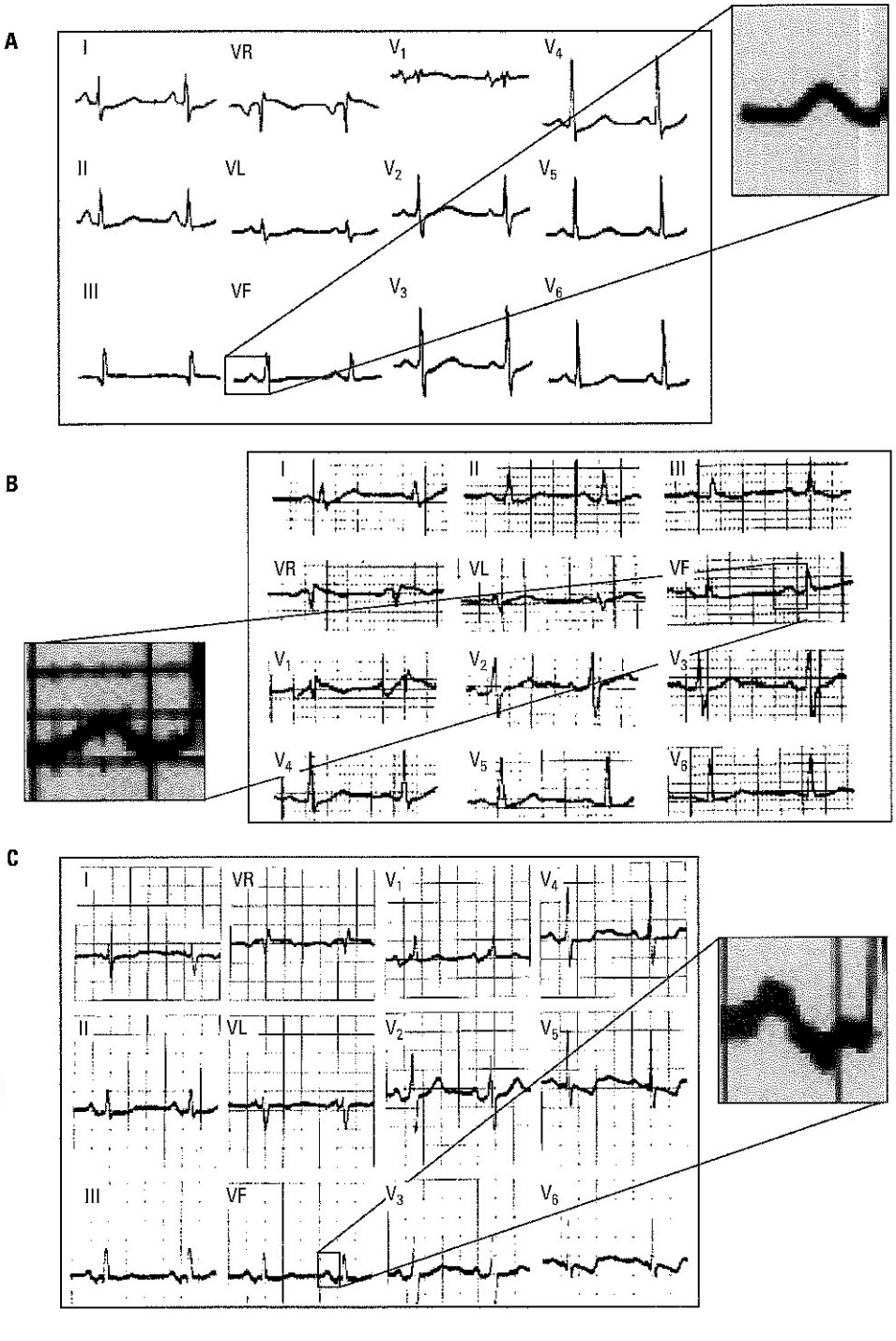

Figure 11 Probressive interatrial block: Three ECGs from a patient with mitro-aortic valve disease. (A) P wave in II, III, and VF with normal atrial duration (P=105ms) and P wave of pseudo P pulmonary type. (B) An intermediate morphology that corresponds to a first-degree interatrial block (P=135ms). (C) Advanced interatrial block appearing after 5 years with P ± morphology in II, III, and VF (P=145ms).

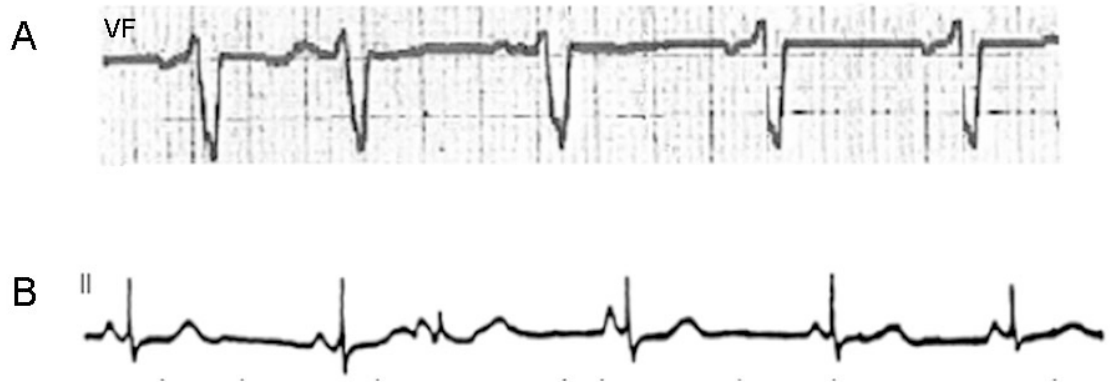

Figure 12 Two cases of atrial aberrancy. The first one A is a case of second degree interatrial block. This is a case of a patient with basal advanced interatrial block (P± with first part isoelectric that mimicks AV junction rhythm) that presents aberrant atrial conduction, ectopically induced by premature atrial complex, with a pattern, in this case, of first degree interatrial block (x). B. A patient with aberrant atrial conduction also ectopically induced by a premature atrial complex. After this premature complex, a transitory P wave with a different morphology, but not with a pattern of first or third interatrial block appears (x). The PR interval is equal to previous PR intervals. Other explanations to this change (atrial escape, artifact, etc.) are unlikely (see Bayés de Luna 2012a and text).

REFERENCES

1. Bayés de Luna A. Clinical Electrocardiography. Wiley-Blacwell 2013
2. Tsao CW, Josphson ME, Hauser TH. Accuracy of electrocardiographic criteria for atrial enlargement: validation with cardiovascular magnetic resonance. J Cardiovasc Magn Reson 2008; 10: 7.
3. Lee K, Appleton C, Lester S, et al. Relation of eCG criteria for left atrial enlargement in two-dimensional echocardiographic left atrial measurements. Am J Cardiol 2007; 9: 113.
4. Ferroglio S, Pham TD, Hordof A, et al. Right atrial ultrastructure in congenital heart disease. Am J Cardiol 1979; 43: 820.
5. Reynolds G. The atrial electrogram in mitral stenosis. Br Heart J 1953; 15: 250.
6. Morris JJ, Estes EH, Whalen RE, et al. P wave analysis in valvular heart disease. Circulation 1964; 29: 242.
7. Reeves WC, Hallahn W, Schwitter EEJ, et al. Two-dimensional echocardiographic assessment of ECG criteria for right atrial enlargement. Circulation 1981; 64: 387.

8. Kaplan J, Evans G, Foster E, et al. Evaluation of ECG criteria for right atrial enlargement by quantitative two-dimensional echocardiography. J Am Coll Cardiol 1994; 23: 747.
9. Rodevan D, Bjornerheim R, Ljosland M, et al. Left atrial volumes assessed by three-and two- dimensional echocardiography compared to MRI estimates. Int J Cardiol Imaging 1999; 15: 397.
10. Whitlock M, Garg A, Gelow J, et al. Comparison of left and right atrial volume by echocardiography versus cardiac magnetic resonance imaging using the area-length method. Am J Cardiol 2010; 1065: 1345.
11. Bayés de Luna A. Clinical Arrhythmology. Wiley-Blackwell, 2011.
12. Bayés de Luna A. et al. Concealed sinus rhythm. Journal Electrocardiology….
13. Bayés de Luna A, Garcia-Cosio F, Platonov P, et al. Interatrial blocks: A separate entity from left atrial enlargement: A consensus report. J of Electrocardiol 2012; 45: 445.
14. Waldo A, Harry L, Bush Jr, et al. Effects on the canine P wave of discrete lesions in the specialized atrial tracts. Circulation Res 1971; 29: 452.
15. Bayés de Luna A, Fort de Ribot R, Trilla E, et al. Electrocardiographic and vectorcardiographic study of interatrial conduction disturbances with left atrial retrograde activation. J Electrocardiol 1985; 18: 1.
16. Bayés de Luna A, Cladellas M, Oter R, et al. Interatrial conduction block and retrograde activation of the Leith Artium and paroxysmal supreventrivular tachyarrhythmias. Eur Heart J 1988; 9: 1112.
17. Bayés de Luna A, Guindo J, Viñolas X, et al. Third-degree inter-atrial block and supraventricular tachyarrhythmias. Europace 1999; 1: 43.
18. Bayés de Luna A, Cladellas M, Oter R, et al. Interatrial block with retrograde activation of the left atrium: influence of preventive antiarrhythmic treatment. Int J Card 1989; 22: 147.
19. Spodick DH, Ariyarajah V. Interatrial block. The pandemic remains poorly perceived. Pacing Clin Electrophysiol 2009; 32: 662.
20. Spodick DH, Ariyarajah V. Interatrial block: a prevalent, widely heglected and portentous abnormality. J Electrocardiol 2008; 41: 61.
21. Doksey JD, Dunan M, Massie E. Clinical vectrocardiography and electrocardiography. Chicago: Year Book Publishers; 1977.
22. Platonov P. Atrial conduction and atrial fibrillation. What can we learn from ECG? Cardiol J 2008; 15: 402.

23. Braunwald E. Foreword to Clinical Electrocardiography. A. Bayés de Luna Wiley-Blackwell 2011.
24. Daubert JC. Atrial flutter. Edited by Waldo……
25. Conde D, Baranchuk A. Bloqueo interauricular como sustrato anatómico-eléctrico de arritmias supraventriculares: Síndrome de Bayés. Archivo de Cardiologia de México 2014; 84: 32-40.
26. Conde D, Baranchuk A. What a Cardiologist must know about Bayes' Syndrome. Rev Argent Cardiol 2014; 82. http://dx.doi.org/10.7775/rac.v82.i2.3996
27. Bayés de Luna A. Bloqueo a nivel auricular. Rev Esp de Cardiologia 1979 vol 32 nº1.
28. Julia J, Bayés de Luna A, Candell J, et al. Aberrancia auricular: A proposito de 21 casos. Rev Esp de Cardiologia 1978 vol 21 nº2.