

Role of Interatrial Block Recognition: A closer look to the Bayés Syndrome

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Interatrial block (IAB) refers to conduction disorders located between the right and the left atrium, and it was found to be a substrate for the development of atrial fibrillation (AF). The pathophysiology of IAB is directly related to a block in the Bachmann's bundle area. IAB has a prevalence of 1% in the global population of middle age people, and 2% among patients with valvular heart disease and cardiomyopathies. IAB was found to be an independent predictor of AF in different clinical entities [1-6]. It was demonstrated that advanced IAB was strongly associated with a higher risk of AF recurrence one year following pharmacological cardioversion, independent of the antiarrhythmic drug utilized [3]. In addition, the presence of pre-existent advanced IAB was associated with a higher risk of AF recurrence post catheter ablation for paroxysmal AF [7], and the presence of advanced IAB predicts new-onset AF after successful cavo-tricuspid isthmus ablation in patients with typical atrial flutter and no history of AF [8].

In this issue of the Journal of Atrial fibrillation, Bazan V, et al. [9] reported an interesting study investigating the role of IAB in enhancing the yield of 24 hour Holter ECG monitoring for the prediction of atrial arrhythmias. The authors should be congratulated for presenting the largest unrestricted series of patients undergoing 24 hour Holter monitoring in the literature. The authors retrospectively analyzed 1017 consecutive 24 hour Holter monitoring recordings performed in a Multidisciplinary Integrated Health Care Institution. A univariate and multivariate regression analysis served to determine the variables associated with a higher 24 hour Holter's yield. The mean age of their population was 62±17 years (55% males). The overall yield was 12.8%, higher for the assessment of the integrity of the electrical conduction system (26.1%) and lower for the

assessment of syncope (3.2%) and cryptogenic stroke (4.6%). The variables associated with higher diagnostic performance were indication from Cardiology ($p < 0.001$), IAB ($p = 0.004$), structural heart disease ($p = 0.008$) and chronic renal failure ($p = 0.009$). Patients less than 50 years of age only retrieved a 7% yield. In the multivariate analysis, indication from Cardiology and IAB remained significant predictors of higher 24 hour Holter's yield. However, in a secondary analysis including echocardiographic data, only identification of IAB remained statistically significant. Therefore, the authors concluded that the recognition of IAB and the type of indication are major determinants of a higher 24 hour Holter's diagnostic yield and may help to optimize the selection of candidates [9].

Of interest, among 212 patients undergoing a complete cardiologic assessment, only 9 of them (4%) had documented AF relapses leading to anticoagulant and/or anti-arrhythmic drug therapy initiation [9]. Seven out of the 9 episodes corresponded to newly diagnosed AF relapses. Interestingly, 7 out of these 9 patients (78%) had IAB. The recognition of IAB yielded a sensitivity of 78%, a specificity of 73%, a positive predictive value of 17%, and a negative predictive value of 98% in the identification of AF relapse prompting anticoagulant and/or anti-arrhythmic drug therapy initiation [9]. As the authors mentioned, the positive predictive value was very low probably because of the low prevalence of IAB and, specially, the very low incidence of "de novo" AF documentation by means of 24 hour Holter monitoring in their population. Although 78% of their patients with AF documentation had underlying IAB, the authors could not perform an adequate correlation analysis between IAB and AF documentation because of the very low incidence of AF during the 24 hour Holter monitor recording.

Key Words

Interatrial Block Recognition, Atrial Fibrillation (AF), Pathophysiology, Interatrial Block (IAB).

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Also, in large series of in-hospital population, Asad N, and Spodick DH [10] identified a prevalence of IAB in 47% in their screened population, with a higher prevalence in the subgroup above 60 years of age [10]. Bayés de Luna A, et al. [4] reported a series of patients with similar echocardiographic parameters and with long-term follow-up

to analyze the incidence of atrial tachyarrhythmias in 16 patients with advanced IAB, and compared them with 22 patients with partial IAB. At one year of follow-up, the incidence of arrhythmias was 80% in the advanced IAB group and, 20% in the partial IAB group. At 30 months of follow-up, the advanced IAB group presented a higher incidence of atrial flutter/fibrillation (15/16, 93.7%), compared with the control group with partial IAB (6/22, 27.7%) ($p < 0.0001$). Moreover, the 24 hour Holter monitoring showed that the prevalence of frequent premature atrial contractions was much more frequent in advanced than in partial IAB patients (75% versus 25%, respectively). These patients should be closely followed using long-term monitoring in order to capture a first episode of AF to proceed with further therapeutic management.

Cosio FG et al performed an interesting study in patients with IAB using intracardiac mapping, demonstrating the retrograde activation of the left atrium in these patients with block in the Bachmann's bundle area [11]. Holmqvist F et al. [12] studied the characteristics of the P-wave morphology according to the way of atrial activation and the relation of this pattern with AF. Indeed, the P wave of the electrocardiogram may show alterations that can be associated with atrial arrhythmias. Hordof AJ et al. [13] found a statistical association between the low resting membrane potential and a prolonged P wave duration. Josephson ME et al. [14] reported that a prolonged interatrial conduction time was significantly related to abnormal P wave morphology. Interesting to note that neither left atrial size nor atrial pressure overload was found to correlate well with abnormal P wave morphology [14]. We have previously demonstrated that patients with a predisposition to develop AF have significantly longer P wave duration, PA intervals, inter and intra-atrial intervals, and atrial conduction delays [15]. We observed that the P wave duration was significantly longer in patients who had abnormal atrial endocardial electrograms (137 ± 17 ms) than in those who did not (125 ± 15 ms, $P < 0.02$). Both the intraatrial (54 ± 12 ms) and interatrial (101 ± 14 ms, $P < 0.001$) conduction times were also significantly longer in patients who had abnormal atrial endocardial electrograms [15]. An abnormally prolonged and fractionated atrial electrogram may reflect inhomogeneous local electrical activity related to a delayed and non-uniform anisotropic conduction through fibrotic atrial myocardium, and was closely related to the vulnerability of the atrial muscle to develop AF [15-18].

Therefore, in the evaluation of patients with altered P wave morphology in the electrocardiogram, it is very important to keep in mind that patients who have a greater susceptibility to develop AF possess abnormally prolonged and fractionated atrial endocardial electrograms, a significantly longer P wave duration, a significantly longer intra-atrial and inter-atrial conduction time of sinus impulses; and a significantly higher incidence of induction of sustained AF [16-18]. Awareness of this strong association in IAB patients may lead to better therapeutic management in individual patients. Due to this strong association of IAB, atrial conduction defects, and abnormal atrial endocardial electrograms with AF, there is a necessity of further studies to shed more light in characterizing the Bayés syndrome in different clinical scenarios, and to better understand the substrate of atrial fibrosis, along with the probability of earlier institution of anticoagulation and antiarrhythmic drugs.

References

1. Bayés de LA, Guindo J, Viñolas X, Martínez-Rubio A, Oter R, Bayés-Genís A. Third-degree inter-atrial block and supraventricular tachyarrhythmias. *Europace*. 1999;1 (1):43-6.
2. Baranchuk A, Enriquez A, Antiperovitch P, Alexander B, Çinier G. Advanced interatrial block as a key marker for atrial fibrillation recurrence: Bayés' syndrome. *J Geriatr Cardiol*. 2017;14 (3):169-173.
3. Enriquez A, Conde D, Hopman W, Mondragon I, Chiale PA, de Luna AB, Baranchuk A. Advanced interatrial block is associated with recurrence of atrial fibrillation post pharmacological cardioversion. *Cardiovasc Ther*. 2014;32 (2):52-6.
4. Bayés de LA, Cladellas M, Oter R, Torner P, Guindo J, Martí V, Rivera I, Iturralde P. Interatrial conduction block and retrograde activation of the left atrium and paroxysmal supraventricular tachyarrhythmia. *Eur. Heart J*. 1988;9 (10):1112-8.
5. Conde D, Baranchuk A. [Interatrial block as anatomical-electrical substrate for supraventricular arrhythmias: Bayés syndrome]. *Arch Cardiol Mex*. 2014;84 (1):32-40.
6. Bayes de LA, Fort de RR, Trilla E, Julia J, Garcia J, Sadurni J, Riba J, Sagues F. Electrocardiographic and vectorcardiographic study of interatrial conduction disturbances with left atrial retrograde activation. *J Electrocardiol*. 1985;18 (1):1-13.
7. Caldwell J, Koppikar S, Barake W. Advanced interatrial block is associated with atrial fibrillation recurrence after successful pulmonary vein isolation for paroxysmal atrial fibrillation. *J Electrocardiol*. 2013.
8. Enriquez A, Sarrias A, Villuendas R, Ali FS, Conde D, Hopman WM, Redfearn DP, Michael K, Simpson C, De Luna AB, Bayés-Genís A, Baranchuk A. New-onset atrial fibrillation after cavotricuspid isthmus ablation: identification of advanced interatrial block is key. *Europace*. 2015;17 (8):1289-93.
9. Bazan A, Cediel G, Llibre C, Sarrias A, Romeo I, Ibars S. Contemporary Yield of 24-hour Holter Monitoring: Role of Inter-Atrial Block Recognition. *J Atr Fibrillation*. 2019;
10. Asad N, Spodick DH. Prevalence of interatrial block in a general hospital population. *Am. J. Cardiol*. 2003;91 (5):609-10.
11. Cosío FG, Martín-Peñato A, Pastor A, Núñez A, Montero MA, Cantale CP, Schames S. Atrial activation mapping in sinus rhythm in the clinical electrophysiology laboratory: observations during Bachmann's bundle block. *J. Cardiovasc. Electrophysiol*. 2004;15 (5):524-31.
12. Holmqvist F, Platonov PG, Mc NS, Polonsky S, Carlson J, Zareba W, Moss AJ. Abnormal P-wave morphology is a predictor of atrial fibrillation development and cardiac death in MADIT II patients. *Ann Noninvasive Electrocardiol*. 2010;15 (1):63-72.
13. Hordof AJ, Edie R, Malm JR, Hoffman BF, Rosen MR. Electrophysiologic properties and response to pharmacologic agents of fibers from diseased human atria. *Circulation*. 1976;54 (5):774-9.
14. Josephson ME, Kastor JA, Morganroth J. Electrocardiographic left atrial enlargement. Electrophysiologic, echocardiographic and hemodynamic correlates. *Am. J. Cardiol*. 1977;39 (7):967-71.
15. Centurion OA, Isomoto S, Fukatani M, Shimizu A, Konoe A, Tanigawa M, Kaibara M, Sakamoto R, Hano O, Hirata T. Relationship between atrial conduction defects and fractionated atrial endocardial electrograms in patients with sick sinus syndrome. *Pacing Clin Electrophysiol*. 1993;16 (10):2022-33.
16. Centurión OA. Clinical implications of the P wave duration and dispersion: relationship between atrial conduction defects and abnormally prolonged and fractionated atrial endocardial electrograms. *Int. J. Cardiol*. 2009;134 (1):6-8.
17. Centurión OA, Shimizu A, Isomoto S, Konoe A, Kaibara M, Hayano M, Yano K. Influence of advancing age on fractionated right atrial endocardial electrograms. *Am. J. Cardiol*. 2005;96 (2):239-42.

18. Centuri3n OA, Shimizu A, Isomoto S, Konoe A. Mechanisms for the genesis of paroxysmal atrial fibrillation in the Wolff Parkinson-White syndrome: intrinsic atrial muscle vulnerability vs. electrophysiological properties of the accessory pathway. *Europace*. 2008;10 (3):294–302.