Clinical, electrical, and echocardiographic characteristics of patients with advanced interatrial block

Características clínicas, eléctricas y ecocardiográficas de los pacientes con bloqueo interauricular avanzado

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Bachmann's bundle is integrated with a set of specialized parallel myocardial fibers and accounts for 80 - 85% of interatrial conduction. It runs along the anterosuperior atrial walls and its compromise leads to interatrial block (IAB), which can be: (a) partial (p-IAB) if conduction is delayed (on ECG, it produces a P-wave ≥ 120 ms) or (b) advanced (a-IAB) if conduction is completely interrupted and left atrial (LA) depolarization occurs in the retrograde caudocranial direction (P wave is ≥ 120 ms and biphasic $\pm$ in lower leads II, III, and VF)¹. a-IAB causes electrical and mechanical atrial dysfunction, which favors the development of "atrial fibrotic myopathy"^{2,3}; fibrosis favors blood stasis and intra-atrial thrombosis (especially in the appendage)⁴, as well as the development of supraventricular arrhythmias such as flutter (typical and atypical), and especially atrial fibrillation (AF): the so-called Bayés syndrome³⁻⁸. There is evidence that a-IAB is an atrial morphofunctional dysfunction marker and a predictor of events such as AF or flutter (*de novo* or relapses after cardioversion or ablation), embolic cerebrovascular accident (CVA), and heart failure³⁻¹⁰. IAB pathogenesis is not fully known, but its genesis involves degenerative, ischemic, infiltrative, and inflammatory factors (age is the most determining factor); consequently, it may appear that an underlying heart disease exists or not, regardless of the LA size, although it

usually occurs in dilated LAs; it can be intermittent and frequency dependent (second-degree IAB) and even reversible, as well as atrial electrical remodeling. The prevalence of IAB increases exponentially with age, and it is high in the general population (59% in those older than 60 years) and in different patient populations (41-48%).

The purpose of this study was to clinically, electrically, and echocardiographically characterize patients with IAB, and analyze its relationship with AF/flutter and CVA. A descriptive, observational, retrospective protocol was designed that included consecutive patients, of legal age, who attended a high-resolution cardiology clinic (visits with electrocardiogram and echocardiogram) over a 3-year period; all of them were in normal sinus rhythm during the visit. Sixty-three cases of a-IAB were identified on ECG and, therefore, a similar sample of consecutive patients with p-IAB (Fig. 1) and without IAB was selected, which yielded a total sample of 165 patients for the study. The analyzed variables were obtained from the computerized medical records and from patient visits (echocardiographic and echocardiographic parameters). Using SPSS 19.0, a descriptive analysis was carried out (which expressed the results of quantitative variables as the mean and standard deviation, and those of qualitative variables as percentage), as well as a bivariate (using the Chi-square test,

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Table 1. Characteristics of the patients without IAB, with partial IAB, and with advanced IAB

	Normal patients (n = 52)	Partial IAB (n = 50)	Advanced IAB (n = 63)	p-value
Age (years), $\bar{X} \pm SD$	59.8 $\pm$ 13.3	66.9 $\pm$ 11.4	76.4 $\pm$ 8.2	< 0.0001
Age $\geq$ 65 years n (%)	22 (42.3)	33 (66.0)	57 (90.5)	< 0.0001
Male gender, n (%)	25 (48.1)	32 (64.0)	41 (65.1)	0.132
Medical history				
Hypertension, n (%)	28 (53.8)	33 (66.0)	55 (87.3)	< 0.0001
Diabetes mellitus, n (%)	7 (13.5)	5 (10.0)	24 (38.1)	< 0.0001
Dyslipidemia, n (%)	31 (59.6)	33 (66.0)	47 (74.6)	0.228
Smoking and former smoking, n (%)	19 (36.5)	24 (48.0)	21 (33.3)	0.261
Body mass index (kg/m ²), $\bar{X} \pm SD$	27.9 $\pm$ 4.7	29.0 $\pm$ 4.3	30.2 $\pm$ 5.2	0.045
Obesity (BMI $\geq$ 30 kg/m ²), n (%)	18 (34.6)	21 (42.0)	31 (52.5)	0.159
Glomerular filtration (mL/min), $\bar{X} \pm SD$	88.7 $\pm$ 23.3	79.2 $\pm$ 19.1	70.5 $\pm$ 23.2	< 0.0001
Stage $\geq$ 2 chronic kidney disease (GF < 90 mL/min), n (%)	22 (43.1)	34 (68.0)	50 (79.4)	< 0.0001
Stage $\geq$ 3 chronic kidney disease (GF < 60 mL/min), n (%)	4 (7.7)	6 (12.0)	20 (32.3)	0.001
COPD, n (%)	5 (9.6)	2 (4.0)	9 (14.3)	0.186
Confirmed OSAHS or snoring + apneas	8 (15.4)	6 (12.0)	8 (13.6)	0.883
Ischemic heart disease, n (%)	5 (9.6)	10 (20.4)	14 (22.2)	0.177
AF/flutter, n (%)	22 (42.3)	15 (30.0)	17 (27.0)	0.194
Cerebrovascular accident, n (%)	0 (0.0)	1 (2.0)	10 (15.9)	na
Stroke, n (%)	0 (0.0)	1 (2.0)	8 (12.7)	na
Transient ischemic attack, n (%)	0 (0.0)	0 (0.0)	2 (3.2)	na
Electrocardiogram				
Atrioventricular block, n (%)	2 (3.8)	3 (6.0)	20 (31.7)	< 0.0001
Complete bundle branch block, n (%)	3 (5.8)	10 (20.0)	13 (20.6)	0.057
Anterior or posterior fascicular block, n (%)	5 (9.6)	9 (18.0)	18 (29.5)	0.225
Positive Cornell index, n (%)	2 (3.8)	6 (12.0)	8 (12.7)	0.225
Positive Sokolow-Lyon index, n (%)	1 (1.9)	3 (6.0)	4 (6.3)	0.493
Echocardiogram				
LV hypertrophy, n (%)	7 (13.7)	10 (20.0)	16 (28.6)	0.166
Sigmoid interventricular septum ($\geq$ 13 mm), n (%)	7 (13.5)	10 (20.0)	16 (25.4)	0.281
Sigmoid interventricular septum $\bar{X} \pm SD$	13.6 $\pm$ 2.1	15.0 $\pm$ 2.6	16.3 $\pm$ 2.5	0.058
LV ejection fraction, $\bar{X} \pm SD$	61.75 $\pm$ 7.06	57.5 $\pm$ 2.2	60.7 $\pm$ 11.1	0.103
$\leq$ 51% LV ejection fraction, n (%)	2 (3.8)	11 (22.0)	7 (12.5)	0.022
< 40% LV ejection fraction, n (%)	0 (0.0)	3 (6.0)	4 (7.1)	0.159
Diastolic dysfunction, n (%)	15 (30.0)	19 (40.4)	38 (71.7)	< 0.0001
Diastolic dysfunction, n (%)				
No	35 (70.0)	28 (59.6)	15 (28.3)	< 0.0001
Mild	11 (22.0)	14 (29.8)	25 (47.2)	
$\geq$ Moderate	4 (8.0)	5 (10.6)	13 (24.5)	
LV 4C area (cm ²), $\bar{X} \pm SD$	18.6 $\pm$ 4.0	21.5 $\pm$ 4.9	23.3 $\pm$ 4.7	< 0.0001
LA indexed biplane volume (mL/cm ²), $\bar{X} \pm SD$	30.0 $\pm$ 8.5	37.8 $\pm$ 11.0	39.5 $\pm$ 11.8	0.002
Dilated LA, n (%)	16 (30.8)	33 (66.0)	44 (78.6)	< 0.0001
Mitral annulus calcification, n (%)	3 (5.8)	3 (6.0)	2 (21.8)	0.011
$\geq$ Moderate left valve disease, n (%)	8 (15.4)	10 (20.0)	12 (19.0)	0.812
Relevant mitral valve disease ($\geq$ moderate regurgitation or stenosis), n (%)	5 (9.6)	6 (12.0)	7 (11.1)	0.926

COPD: Chronic Obstructive Pulmonary Disease; IAB: interatrial block; LA: left atrium; LV: left ventricle; na: not applicable; OSAHS: Obstructive Sleep Apnea-Hypopnea Syndrome.

Fisher's exact test, Student's t-test, and Mann-Whitney's U-test) and a multivariate analysis; the level of significance was established at $p < 0.05$.

Bivariate analysis between the three groups of patients yielded the results shown in [Table 1](#). The strong relationship detected between IAB and age,

Figure 1. Left panel: ECG horizontal leads of a patient with partial IAB. Right panel: ECG horizontal leads of two patients with advanced IAB.

Table 2. Variables related to the risk for developing advanced IAB

	OR	IC 95%	p-value
Age $\geq$ 65 years	6.1	1.6-23.7	0.009
Diabetes mellitus	6.1	2.1-17.8	0.001
LA dilation	6.0	2.0-17.4	0.001
Diastolic dysfunction	3.1	1.3- 7.7	0.013

CI: confidence interval; IAB: interatrial block; LA: left atrium; OR: odds ratio.

high blood pressure, diabetes mellitus, chronic kidney disease, first-degree atrioventricular block, diastolic dysfunction (particularly if it involved intra-atrial pressure elevation), and LA dilation (the presence of a-IAB was more common in patients with larger atria) stands out. However, a-IAB relationship with a “documented” history of AF/flutter was not demonstrated, probably due to the diagnostic difficulty these arrhythmias entail if they are not permanent (although most part of persistent AFs, which accounted for 55% of all of them, and 100% of flutters belonged to group a-IAB). The fact that 90.9% (10 cases) of ischemic CVAs in the series (cardioembolic or of unknown cause) occurred in patients who had a-IAB and barely in 9.1% of those who had p-IAB also stands out; of these 10 cases in the a-IAB group, 80% corresponded to stroke and 20% to transient ischemic attacks and, of all of them, only 40% had a previous history of documented AF or flutter. When the groups with p-IAB and a-IAB were

compared using a bivariate analysis, only a-IAB was found to be related to a history of CVA ($p = 0.022$), especially of stroke ($p = 0.04$).

Finally, a multivariate analysis was carried out (which included all three groups of patients and all variables shown in Table 1 with a $p < 0.1$), which determined that the only variables related to the risk for developing a-IAB were being ≥ 65 years of age (odds ratio [OR] = 6.1), diabetes mellitus (OR = 6.1), diastolic dysfunction (OR = 6.0), and left atrial dilation (OR = 3.1) (Table 2).

The obtained results lead to conclude that the factors associated with the risk of suffering a-IAB are being ≥ 65 years old, diabetes, ventricular diastolic dysfunction, and dilated left atrium. Patients with a-IAB have a higher rate of ischemic CVA, especially stroke, which appears together with a history of AF or without it (either due to underdiagnosed arrhythmia or underlying atrial myopathy). These results highlight the possibility of AF screening and prevention and the need to specify whether patients with a-IAB should be anticoagulated to particularly prevent CVAs.

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Conflicts of interest

The authors declare that they have no conflicts of interest.

Ethical disclosures

Protection of human and animal subjects. The authors declare that no experiments were performed on humans or animals for this research.

Confidentiality of data. The authors declare that no patient data appear in this article.

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