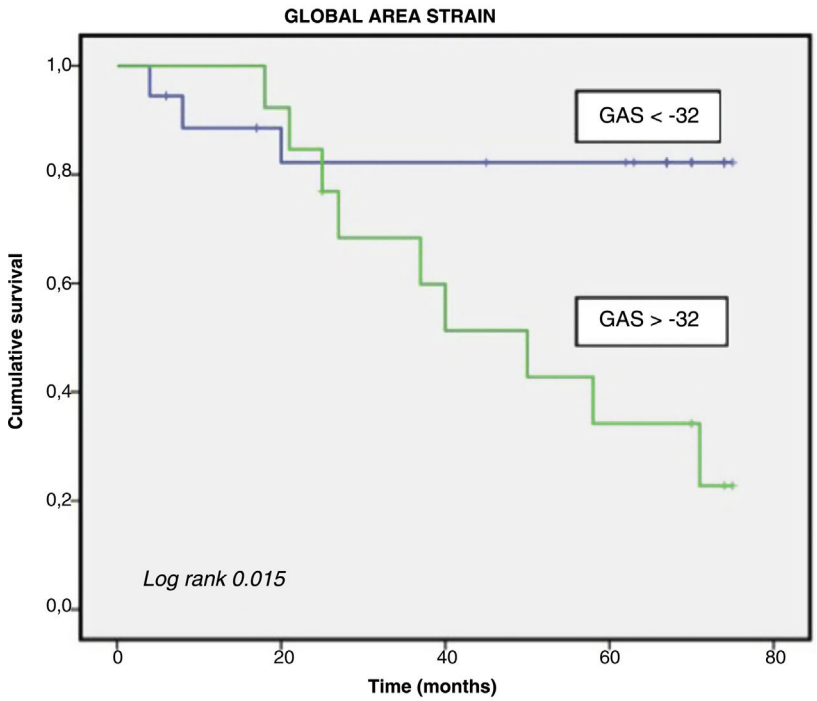

Figure 1. Kaplan-Meier survival curve according to GAS. A GAS > –32 was associated with a significantly lower event-free survival in patients with asymptomatic grade > III aortic regurgitation and LVEF > 55%. GAS, global area strain; LVEF, left ventricular ejection fraction.

parameter that is usually used, LVEF. Determination of GAS does not vary during follow-up of patients who remain asymptomatic and do not meet criteria for surgery.

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ECG patterns of typical and atypical advanced interatrial block: prevalence and clinical relevance

Prevalencia y relevancia clínica de los tipos de bloqueo interauricular avanzado típico y atípico

To the Editor,

The concept of interatrial block (IAB), established by consensus in 2012, is defined as prolonged P wave duration (≥ 120 ms) due to delayed transmission of the sinus impulse through the region of the Bachmann bundle.[1] IAB is classified as partial (P wave ≥ 120 ms) or advanced (P wave ≥ 120 ms with biphasic morphology in the II-III-aVF leads). Atypical patterns of advanced IAB have been described, but their frequency and clinical significance are unknown.[2] The aims of this study were to determine the prevalence of the various IAB patterns (partial, typical advanced, and atypical advanced) in the general population,[3] in centenarians,[4] in patients with heart failure,[5] and in

patients undergoing cavotricuspid isthmus ablation for atrial flutter,[6] and to evaluate associations between these patterns and atrial fibrillation (AF) or stroke.

Data from 4 studies[3–6] were evaluated. These included 240 individuals from the general population who participated in a case-control study (n = 80 and n = 160, respectively) to analyze the relationship between IAB and AF,[3] 60 centenarians,[4] 464 patients with heart failure,[5] and 110 patients undergoing cavotricuspid isthmus ablation to treat atrial flutter.[6] All 4 studies had been approved by their respective local ethics committees and the participants signed an informed consent form.

In each study, researchers performed a standardized analysis of the patients' electrocardiograms. The following IAB patterns were defined[2] (figure 1):

1. Normal P wave: duration < 120 ms and normal morphology.
2. Partial IAB: duration ≥ 120 ms and absence of the final negative component of the P wave in the inferior leads.
3. Advanced IAB:

- Typical: duration ≥ 120 ms and P wave with biphasic morphology in II-III-aVF.
- Atypical by morphology:[2]

- Type 1: duration ≥ 120 ms and P +/– in III-aVF with an isodiphasic component in lead II in the terminal portion (+/0). This occurs because the final part of atrial activation in the P loop is around –30°, at the limit of the positive or negative hemifield of lead II.
- Type 2: duration ≥ 120 ms and P +/– in III-aVF with P +/–/+ in II. Similar to type 1. It occurs because the final part of atrial activation in the P loop is around –30°, but at the end it rotates clockwise and passes from the negative to the positive hemifield of II.
- Type 3: duration ≥ 120 ms and P +/– in II and P negative in III and aVF, but with an isodiphasic component in III-aVF in the first part (0/–). This occurs because the onset of atrial activation has an axis between 0 and –30°.

- Atypical by duration:[2] duration < 120 ms but P +/– in II-III-aVF.

The mean age of the participants was 74 years in the general population group, 101 years in the centenarian group, and 65 to 67 years in the heart failure and atrial flutter ablation groups. Men accounted for 70% to 78% of participants in all groups except the centenarian group, in which 70% were women.

In the series studied, 40% to 50% of participants had a normal P wave, except the centenarians (32%). Atypical advanced IAB occurred in 5.6% (95% confidence interval [95%CI], 3.0%-10.4%) of the general population controls, 25.0% (95%CI, 15.8%-37.2%) of the centenarians, 16.8% (95%CI, 13.7%-20.5%) of heart failure patients, and 6.4% (95%CI, 3.1%-12.6%) of patients with atrial flutter (table 1). These prevalence values were similar to those of typical

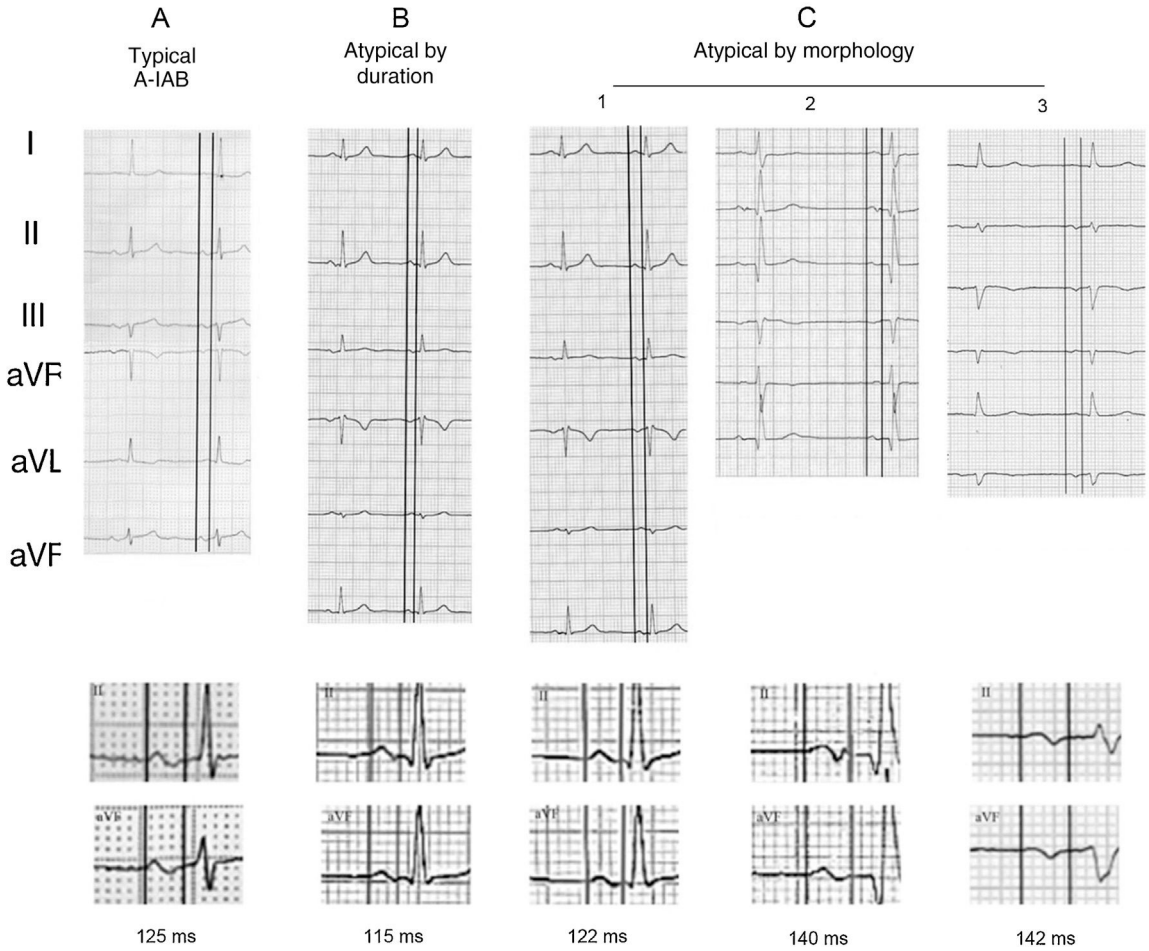

Figure 1. Typical and atypical advanced interatrial block patterns: A, typical advanced interatrial block; B, atypical advanced interatrial block by duration; C, atypical advanced interatrial block by morphology. ms, P wave duration in milliseconds.

Table 1
Prevalence of the various types of interatrial block in the studies analyzed

Study	Participants	Normal P wave	Partial IAB	Advanced typical IAB	Advanced atypical IAB			
					By morphology			By duration
					Atypical 1	Atypical 2	Atypical 3	
General population (REGICOR) Age: 74 y Men: 70.0%	n = 80 Atrial fibrillation	26.3% (17.9-36.8) (n = 21)	62.5% (51.6-73.3) (n = 50)	7.5% (3.5-15.4) (n = 6)	2.5% (0.7-8.7) (n = 2)	1.25% (0.2-6.8) (n = 1)	0% (0-4.6) (n = 0)	0% (0-4.6) (n = 0)
	n = 160 Controls	44.4% (36.9-52.1) (n = 71)	46.9% (39.3-54.6) (n = 75)	3.1% (1.4-7.1) (n = 5)	5.0% (2.6-9.6) (n = 8)	0.6% (0.1-3.5) (n = 1)	0% (0-2.3) (n = 0)	0% (0-2.3) (n = 0)
General population (centenarians) Age: 101 y Men: 30.0%	n = 60	31.7% (21.3-44.2) (n = 19)	26.7% (17.1-39.0) (n = 16)	16.7% (9.3-28.0) (n = 10)	15.0% (8.1-26.1) (n = 9)	1.7% (0.3-8.9) (n = 1)	1.7% (0.3-8.9) (n = 1)	6.7% (2.6-15.9) (n = 4)
Heart failure Age: 65 y Men: 70.9%	n = 464	50.6% (46.1-55.2) (n = 235)	20.5% (17.1-24.4) (n = 95)	12.1% (9.4-15.4) (n = 56)	10.1% (7.7-13.2) (n = 47)	0.9% (0.3-2.2) (n = 4)	0.4% (0.1-1.6) (n = 2)	5.4% (3.7-7.8) (n = 25)
Atrial flutter ablation Age: 67 y Men: 78.1%	n = 110	38.2% (29.7-47.5) (n = 42)	50.0% (40.8-59.2) (n = 55)	5.5% (2.5-11.4) (n = 6)	3.6% (1.4-9.0) (n = 4)	0.9% (0.2-5.0) (n = 1)	0.9% (0.2-5.0) (n = 1)	0.9% (0.2-5.0) (n = 1)

IAB, interatrial block

advanced IAB in all the groups. The most common form of atypical advanced IAB was type 1 morphology.

The relationship between these IAB patterns and the risk of AF or stroke was analyzed in patients with heart failure. The various types of atypical block by morphology were grouped into a single category because of the small number of cases. In heart failure patients, the presence of typical advanced IAB or atypical advanced IAB by morphology was associated with a similarly increased risk of AF (respectively, hazard ratio [HR], 2.06; 95%CI, 0.96-4.43, and HR, 1.96; 95%CI, 0.96-3.92) and stroke (HR, 2.96; 95%CI, 0.67-13.01, and HR, 2.82; 95%CI, 0.74-10.79). These results indicate that atypical advanced IAB is a risk factor for AF or stroke in these patients in the same manner as typical advanced IAB.

Among the limitations of this analysis (in addition to those of each study), we mention the heterogeneity of the study designs, the interobserver variability in determining atypical IAB patterns, and the differences in the follow-up times and methods used in the studies.

In conclusion, atypical advanced IAB is as frequent as typical advanced IAB, and it occurs most often as type 1 by morphology. Our results indicate that the prevalence of this condition increases with age and the presence of heart failure. In heart failure patients, atypical advanced IAB by morphology is associated with an increased risk of AF and stroke, similar to that of typical advanced IAB.

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AUTHORS' CONTRIBUTIONS

Definition of the objectives and study design (R. Elosua, M. Martínez-Sellés, A. Baranchuk, A. Bayés-de-Luna), data collection (LA Escobar-Robledo, A. Massó-van Roessel), data analysis (R. Elosua), interpretation of the results (R. Elosua, L.A. Escobar-Robledo, A. Massó-van Roessel, M. Martínez-Sellés, A. Baranchuk, A. Bayés-de-Luna), writing of the first draft (R. Elosua, A. Bayés-de-Luna), critical review of the manuscript and approval of the final version, as well as availability to answer questions related to the validity and integrity of the data and the study (R. Elosua, L.A. Escobar-Robledo, A. Massó-van Roessel, M. Martínez-Sellés, A. Baranchuk, A. Bayés-de-Luna).

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

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Common arterial trunk type I in a 15-year-old boy with grade I pulmonary hypertension

Tronco arterioso tipo I en adolescente de 15 años con hipertensión pulmonar de grado I

To the Editor,

Common arterial trunk is a truncoconal congenital heart disease that represents up to 4% of all congenital heart disease and is characterized by a single arterial trunk from which the coronary, pulmonary, and systemic arteries originate. The most widely-used classification is that of Collett and Edwards, based on the anatomic origin of the pulmonary arteries. Without surgical treatment, 80% of patients die prematurely, and survival to adolescence or adulthood is rare; almost all survivors develop severe vascular obstructive disease or Eisenmenger syndrome.[1,2]

With prior ethics committee authorization and consent from the patient's parents, we present the case of a 15-year-old male patient—diagnosed at 2 months of age with common arterial trunk type 1 that was not surgically treated based on the parents' decision—who was reassessed due to onset of dyspnea. On admission, pulse oximetry was 85% and there was a left parasternal systolic murmur. Transthoracic echocardiography confirmed the prior diagnosis, showing a tri-leaflet truncal valve with moderate regurgitation and a ventricular septal defect measuring 20 × 26 mm. This was followed by computed tomography which showed abnormal origin of the left coronary artery (figure 1A). Catheterization showed systemic pulmonary pressure, with end-diastolic pressure of the left and right ventricles of 15 mmHg. On calculation using the Fick method with an assumed oxygen consumption of 132 mL/min/m^2, Qp/Qs was 4.7, with a pulmonary vascular resistance index of 5.45 UW/m^2 and PVR/SVR ratio = 0.19; when oxygen was given, the Qp/Qs ratio increased to 5.08 and the pulmonary vascular resistance index decreased to 4.49 UW/m^2 (table 1); a homogenous right capillary blush was observed (figure 1B). Karyotype study and fluorescent in situ hybridization (FISH) were reported as normal.

It was decided to perform surgical correction, with connection of the pulmonary ventricle using a 22-mm woven Dacron tube, implantation of a 21-mm bovine aortic valve prosthesis in the pulmonary position, closure of the ventricular septal defect with a bovine pericardial patch, and aortic valvuloplasty. Right and left pulmonary biopsy reported muscularization of the tunica media of the intralobular and centrilobular arteries, with no intimal reaction, small vessels with fibrin clots, and hemosiderin-containing alveolar macrophages (figure 1C,D).

The patient progressed well, in NYHA functional class I. Follow-up catheterization at 23 months found a mean pulmonary pressure of 28 mmHg, pulmonary vascular resistance index of 4.92 UW/m^2, and PVR/SVR ratio = 0.19.

Left to the natural history of the disease, it is rare for patients to survive to adolescence and adulthood; almost all develop severe pulmonary vascular obstructive disease or Eisenmenger syndrome.[2] The pathophysiological mechanisms that determine the reversible or irreversible nature of pulmonary hypertension in congenital heart disease remain unclear. Blood flow and pressure are key triggers for pulmonary vascular remodeling in congenital heart disease: with greater flow and pressure, the blood flow in the whole pulmonary arterial bed is disrupted, causing inflammation and proliferation.[3]

Genetic susceptibility factors have been identified that may predispose to or accelerate pulmonary vascular remodeling: the most noteworthy are bone morphogenetic protein receptor type 2 (*BMPR2*) and transcription factor Sox17.[4] Roberts et al.[5] identified mutations in 6% of patients with congenital heart disease-associated pulmonary hypertension. In patients with congenital heart disease, Liu et al.[6] found a significant difference in *BMPR2* mutation between those with and those without pulmonary vascular disease.

The case presented here had an unusual natural history. The patient did not have protective anatomical abnormalities (pulmonary branch hypoplasia/stenosis) or pulmonary banding; furthermore, he lived in a city that has an altitude of 2240 m, and did not develop early irreversible pulmonary hypertension despite a large shunt—this could be related to physiological and genetic adaptation processes that have been described in animals living at high altitude. It is unknown if protective genetic factors exist that would explain why some patients, despite having congenital heart disease with a large shunt, develop pulmonary hypertension later and to a lesser severity.

In conclusion, patients should be assessed thoroughly as candidates for surgical correction without using age as an exclusion criterion for corrective treatment; it is important to understand the mechanisms that allow some patients to be protected and not develop pulmonary vascular disease.