Table 2
Exercise test variables

Atropine	27 (13.5)
Positive echocardiography test	30 (15)
Positive echocardiography test with previous CAD	5 (17.2)
Positive electrocardiography test	12 (6)
MET	7.3 ± 2.4

CAD, coronary artery disease; MET, metabolic equivalents.
Data are expressed as no. (%) or mean ± standard deviation.

All tests were performed on a treadmill using the Bruce method or the modified Bruce method. To increase diagnostic efficiency, a 1 mg intravenous dose of atropine was given to patients with no contraindications and with a very low estimated probability of achieving submaximal heart rate due to low functional capacity or nonwithdrawal of rate-reducing medication.[3] Contrast echocardiography was used in all patients with a suboptimal acoustic window.

Baseline patient characteristics are summarized in table 1. Mean pretest probability according to the 2019 ESC guidelines[1] was 23.63% ± 14.31%. Of the patients studied, 127 (63.5%) had a pretest probability > 15%. No adverse events were recorded during the tests.

The stress echocardiography results are summarized in table 2. Test results were positive in 15% of patients (n = 30). Of these patients, only 40% (n = 12) had positive results in the exercise electrocardiography test. Of the positive echocardiography tests, 4 could not be evaluated electrocardiographically due to the presence of underlying conduction disorders.

Atropine was given to 27 patients (13.5%) in accordance with the safety warnings for this drug. The indications for atropine were as follows: 22 patients took beta-blockers on the day of the test or the day before, 1 was under treatment with more than 1 rate-reducing drug, and 4 were not taking rate-reducing medication but had low functional capacity. No adverse effects were observed.

In Spain, stress echocardiography is the most widely available imaging test for the detection of ischemia. Restricting this test to the hospital setting limits the capacity of health services to respond to the growing demand for this procedure.

In our experience, the use of stress echocardiography in a specialized nonhospital center staffed with trained personnel is safe, allows health services to respond to the growing demand for this test, and increases diagnostic efficiency.

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Endocardial autonomic denervation in a patient with neurally mediated syncope and severe cardioinhibitory response

Denervación autonómica endocárdica en paciente con síncope neuromediado y respuesta cardioinhibitoria grave

To the Editor,

We present the case of a male patient, a 45-year-old cattle farmer, with presyncopal and syncopal episodes; the last of these events resulted in head trauma. In addition, he had paroxysmal atrioventricular block (AVB) with prolonged pauses, always at rest (figure 1, asterisk). Given his age and the high number of sudden-onset syncopal episodes, we considered that echocardiography and magnetic resonance imaging would be valuable (despite not being specifically recommended in clinical practice guidelines). These techniques ruled out structural heart disease and signs of sarcoidosis. Due to his occupation, we also tested for Lyme disease, which was negative. Adequate tachycardization was seen with exercise (during telemetry monitoring), as well as a syncopal (cardioinhibitory) response in the tilt table test. The proposed management was autonomic modulation via radiofrequency ablation of the ganglionated plexi.

The procedure was performed under mild sedation. Electroanatomical mapping (Biosense Webster, United States) of the left and right atrium (RA) performed with a multipolar catheter (figure 2A-D) failed to identify any notable low-voltage (< 0.5 mV) areas. The patient had a baseline AH interval of 120 milliseconds (figure 2E). The multipolar catheter was then replaced with an ablation catheter, used to apply high-frequency pacing (10-second bursts at 20 Hz for 25 mA/ms) in the region of the ganglionated plexi to identify vagal responses. Because this procedure was sometimes complicated by the induction of atrial fibrillation, anatomical sites of possible relevance were ablated (in the RA, the junction of the superior vena cava [SVC] with the RA in the posterior region and the coronary sinus [CS] ostium [figure 2A]; in the left atrium, the anterosuperior antral region of the right superior pulmonary vein [contralateral to the SVC application] and the left superior pulmonary vein [figure 2B], the posteroinferior region of the left inferior vein [figure 2C], and the area of the ligament of Marshall at its insertion into the CS [figure 2D]). During the radiofrequency ablation, prolonged asystoles were sometimes obtained, up to 9.4 seconds (figure 2F). The AH interval after the ablation was

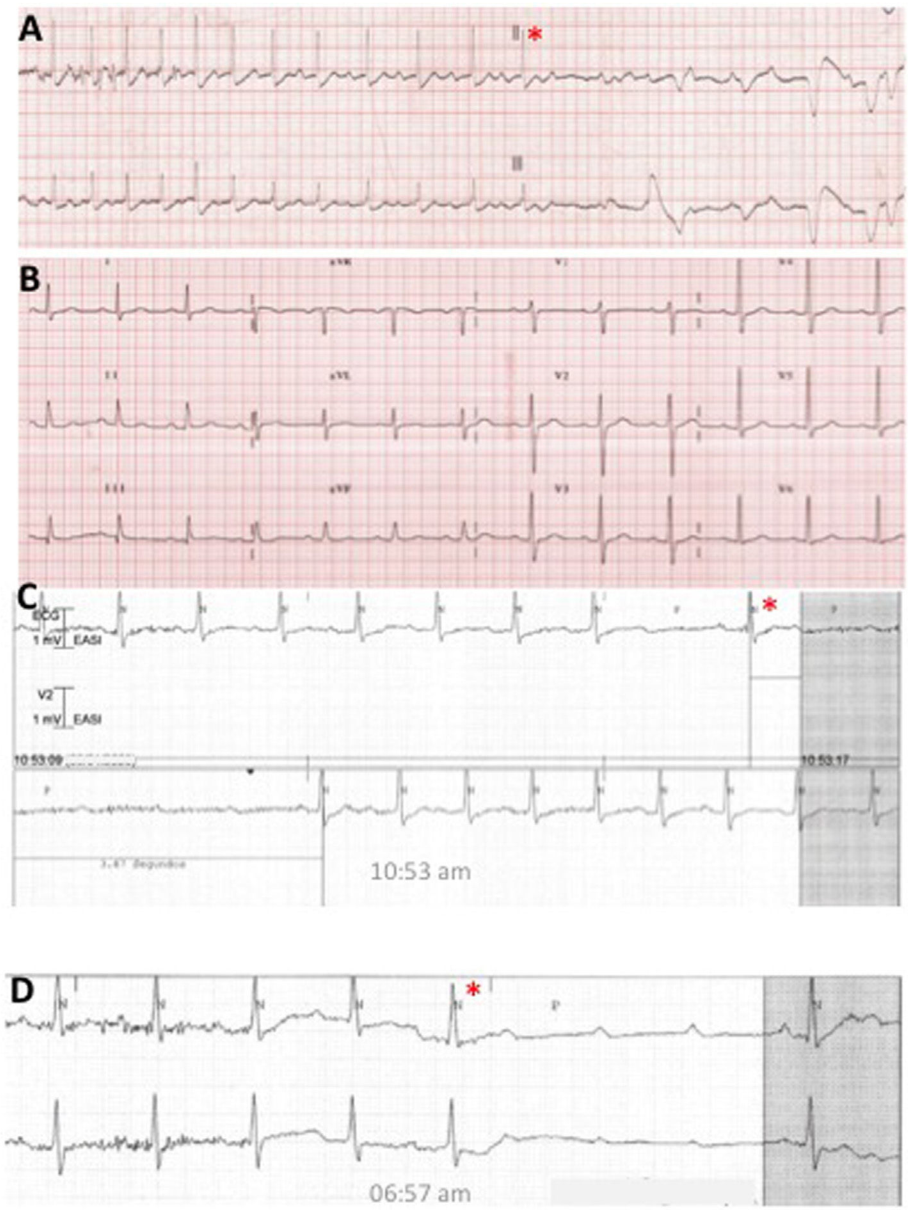

Figure 1. A: rhythm strip obtained by the emergency healthcare service for one of the syncopal episodes. B: 12-lead electrocardiogram at hospitalization. C and D: telemetry recordings during the patient's admission.

90 milliseconds (figure 2G) and there was no notable change in the PP interval (or in the Wenckebach point). After the procedure, the patient remained hospitalized with electrocardiographic monitoring (for 72 hours) and no AVB episodes were recorded. At 1 month, there were still no blocks in Holter monitoring (but a slight variability in heart rate [HR], with a minimum rate of 66 bpm) (figure 2H) and an atropine test showed appropriate denervation, given the absence of tachycardization. Holter ECG results at 4 months were almost identical to the previous test results (minimum HR of 62 bpm, 0 pauses $<$ 2.5 s) (figure 2I). The patient also showed considerable clinical improvement, with no presyncope or syncope and an adequate response to physical exercise.

In this case, endocardial ablation of the ganglionated plexi is presented as an alternative to pacemaker implantation. Various groups have reported the results of biatrial ablation[1] or even unifocal ablation in the RA[2] in patients with neurally mediated syncope and severe cardioinhibitory response. Although numerous doubts remain regarding the long-term outcomes (eg, only 48 patients in the series reported by Qin et al.[1] and 9 in that reported by Debruyne et al.[2] completed 12 months of follow-up) and the optimal strategy, this approach currently represents a promising alternative for highly selected patients such as ours. Although permanent pacemaker implantation would have helped to control the AVB episodes in our patient, given the positive response to the tilt table test, we believe that there was a high probability of syncopal recurrence due to the persistence of the vasodepressive response.[3,4] Theoretically, the interruption not only of the efferent pathway with endocardial ablation, but also that of the afferent pathway might underlie the amelioration of both the hypotensive and cardioinhibitory responses. In addition, the slight increase in HR after the cardiomodulation might increase cardiac output, which, as previously described,[4] represents a more relevant mechanism to the syncopal response than the decrease in peripheral vascular resistance.

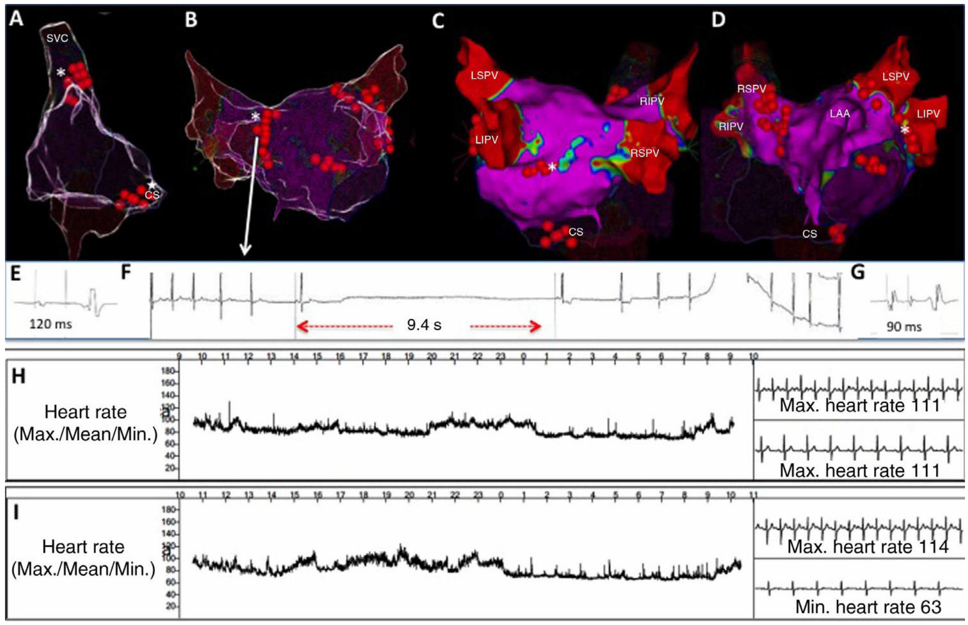

Figure 2. A: anatomical reconstruction of the right atrium (RA) showing the radiofrequency applications at the junction of the superior vena cava (SVC) with the RA (asterisk) and at the coronary sinus (CS) ostium (star). B: anterior view of the left atrium mainly showing the lesions contralateral to the SVC in the anterosuperior region of the right pulmonary vein (asterisk). C: posterior view of the left atrium showing the 4 pulmonary veins (PVs): left superior (LSPV), left inferior (LIPV), right superior (RSPV), and right inferior (RIPV). D: anterior view (slightly oriented toward the left) showing the applications in the region of the ridge between the left atrial appendage (LAA) and the left PVs (asterisk). E: AH interval before ablation. F: asystole provoked by radiofrequency application in the anterosuperior region of the RSPV. G: AH interval after ablation. H and I: Holter-ECG frequency histograms obtained 1 and 4 months after ablation.

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Left ventricle myocardial deformation pattern in severe aortic valve stenosis without cardiac amyloidosis. The AMY-TAVI trial

Patrón de deformación miocárdica del ventrículo izquierdo en la estenosis aórtica grave sin amiloidosis cardiaca. Estudio AMY-TAVI

To the Editor,

Cardiac amyloidosis (CA) is characterized by extracellular deposition of amyloid fibrils in the myocardium and other cardiac structures. Although its actual prevalence is unknown, transthyretin-related CA is thought to be present in 15% to 30% of patients with aortic stenosis (AS) treated by transcatheter aortic valve implantation (TAVI), possibly identifying a patient subgroup with a poorer prognosis. Echocardiography is an essential tool used to establish the initial diagnostic suspicion. However, the coexistence of AS and CA could mask the diagnosis of the latter, as they share common features.[1]

Several publications report on advanced echocardiographic indices based on left ventricular longitudinal myocardial strain, which could differentiate CA from other forms of hypertrophy. These indices include RELAPS (relative apical sparing of longitudinal strain [LS]),[2] septal apical to basal LS ratio (SAB),[3] or left ventricular ejection fraction (LVEF) to global longitudinal strain (GLS) ratio (EFSR).[4]

Our aim was to assess the diagnostic utility of applying these LS-based echocardiographic criteria described for suspected CA in patients with severe AS without amyloidosis.

As part of the AMY-TAVI (NCT03984877) trial to study the prognostic impact of CA in patients with severe AS who