Diagnosis of Patent Foramen Ovale: Concordance between Transthoracic and Transesophageal Echocardiography Using Agitated Saline

Patent foramen ovale (PFO) is found in up to 25% of adults in the general population. (1) Most PFOs are discovered incidentally and have no significant clinical consequences. However, association of PFO has been reported with various clinical conditions, such as cryptogenic stroke, migraine, decompression sickness and platypnea–orthodeoxia syndrome. In turn, cerebral ischemic events represent 70% to 80% of cerebrovascular disease. However, in approximately 40% of cases these conditions have no identifiable cause. (1) It is likely that POF is responsible in these cases, since in those subjects diagnosed with cryptogenic stroke, the probability of having POF is 4 times higher than in control subjects. (2) There are several studies and maneuvers to improve the diagnostic results of this condition. Transesophageal echocardiography (TEE) has been considered the method of reference. (2) However, the results from recent studies show false negative results secondary to intubation and to the difficulty in performing the Valsalva maneuver under moderate sedation. (3) Moreover, Marriott et al. reported that transthoracic echocardiography (TTE) can be used as a diagnostic tool for PFO associated with agitated saline contrast, with sensitivity and specificity similar to those of TEE. (4) These arguments led us to study the concordance between TTE and TEE for the diagnosis of PFO.

A total of 45 consecutive patients were included, who consulted the Neurology Unit at our center due to stroke of unknown origin. Among these patients, 22 (49%) were men. Transthoracic and transesophageal echocardiography was performed, sensitized with agitated saline and Valsalva maneuver.

A right-to-left shunt attributed to PFO was found in 42% of the patients at rest and in 53% with Valsalva maneuver. When evaluating the concordance among the four study variants, TTE and TEE, with and without Valsalva maneuver, a kappa value of 0.64 (Fleiss' kappa) was found. The best correlation between both studies was observed with the Valsalva maneuver (kappa 0.73), the worst being between TTE with Valsalva and TEE at rest (kappa 0.42) (Table 1).

Table 1. One-to-one concordance analysis

Concordance	Kappa	95% CI	p
TTE at rest and TTE with Valsalva	0.69	0.48-0.87	< 0.001
TTE at rest and TEE at rest	0.73	0.49-0.91	< 0.001
TTE at rest and TEE with Valsalva	0.60	0.35-0.82	< 0.001
TTE with Valsalva and TEE at rest	0.43	0.14-0.69	0.003
TTE with Valsalva and TEE with Valsalva	0.73	0.50-0.91	< 0.001
TEE at rest and TEE with Valsalva	0.69	0.45-0.91	< 0.00

TTE: Transthoracic echocardiography. TEE: Transesophageal echocardiography.

Table 2. Severity of right-to-left shunt

PFO	TTEr (n)	%	TTEv (n)	%	TEEr (n)	%	TEEv (n)	%
Absent	25	55.6	20	44.4	25	55.6	21	46.7
Mild	8	17.8	6	13.3	10	22.2	13	28.9
Moderate	3	6.7	4	8.9	3	6.7	3	6.7
Severe	9	20.0	15	33.3	7	15.6	8	17.8

PFO: Patent foramen ovale. TTEr: Transthoracic echocardiography at rest. TTEv: Transthoracic echocardiography with Valsalva. TEEr: Transesophageal echocardiography at rest. TEEv: Transesophageal echocardiography with Valsalva.

When the degree of shunt severity with both methods was analyzed, it was indeed low with TEE (severe TTE 33.3% versus 17.8% with TEE) (Table 2).

Some authors suggest that this discrepancy may be due to the difficulty in performing the Valsalva maneuver when the patient is under sedation. (3) The fact that our patients were not sedated during the study did not affect shunt quantification. It suggests that other mechanisms, such as intubation, may be involved in atrial hemodynamic changes. (5) Atrial septal aneurysm (ASA) was detected in 49% of the study patients with both methods. Sixty-eight percent of patients with ASA were diagnosed PFO with TTE, and 63% with TEE.

Patient tolerance to TEE without sedation was good in 87% of the patients and moderate in 13%. The quality of the acoustic window in TTE was positive in 80% of the cases, and deficient in 20%.

The good concordance between TTE and TEE found in our study confirms similar findings from other authors. (6)

The limitations in our study include the lack of blinded studies compared with other studies, since the physician performing the TTE was the same that then performed the TEE, and was aware of the results of the first study.

We have been unable to set a valid pattern to quantify shunt severity. In our study, we have assumed the severity according to the number of bubbles counted. We have used a subjective value for tolerance assessment.

In conclusion, and based on our findings and their limitations, the use of TTE to identify PFO, a potential source of stroke of unknown origin, provides diagnostic results that are very similar to those of TEE. The use of agitated saline and Valsalva maneuver facilitate diagnosis. If these findings are confirmed with studies including a greater number of patients and more statistical power, transesophageal echocardiography, a semi-invasive method, would be restricted to patients with bad acoustic window limiting transthoracic echo-

cardiography, to define the anatomy of the atrial septum when planning a percutaneous closure, or as a complementary test in case of atrial fibrillation.

Conflicts of interest

None declared.

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REV ARGENT CARDIOL 2016;84:472-473. http://dx.doi.org/10.7775/rac.v84.i5.7611

Percutaneous Mitral Valve Repair with MitraClip™ in a Patient with Ischemic Mitral Regurgitation

Treating severe or secondary ischemic mitral regurgitation (MR) is a challenge, since surgical valve repair has not shown conclusive benefits and is associated with high rate of recurrence and need for another surgical repair. (1) While international guidelines suggest a surgical approach when combined with coronary bypass surgery, valve repair in isolation is not recommended, particularly for patients with high perioperative risk. (2, 3) Thus, a significant number of patients with severe functional MR do not undergo surgery, resulting in an ominous prognosis. We report the case of an 83-year-old male patient with history of coronary artery disease (coronary bypass surgery in 1999 and percutaneous intervention in 2009) and renal failure (dialysis since 2009), who was referred to our department due to MR associated with Class III dyspnea and 3-month history of Class IV episodes. Baseline transthoracic echocardiography showed severe central jet (segment A2-P2) (3+), left atrial enlargement (62 mm), preserved left ventricular systolic and diastolic diameters (51 and 32 mm) with impaired inferior wall motion and left ventricular ejection fraction of 54% (Figure 1 A). A non-invasive functional study ruled out myocardial ischemia. In order to repair the valve disease, the case was raised to the surgeons, who ruled out the intervention due to its high risk (Society of Thoracic Surgeons [STS] Score 25.2; logistic EuroSCORE 32.7%). Given the unlikely good patient progress if valve regurgitation was not corrected, MitraClip™ (Abbott Vascular) was implanted to repair the mitral valve. The device consists of a cobalt-chromium clip covered with polyester, with two arms that are opened and closed simultaneously to grasp the leaflets of the mitral valve, mimicking the open chest edge-to-edge technique developed by Alfieri et al. (4) Under general anesthesia, venous (right femoral vein with 8-Fr introducer) and arterial (left femoral with 6-Fr introducer) access was performed, followed by anticoagulation with heparin sodium. A 0.035-inch super stiff guidewire was advanced to the right atrium followed by the Mullins set, placing a pigtail catheter at the level of the aortic valve as reference. Transseptal puncture was guided with transesophageal echocardiography (TEE) to ensure the puncture site was distant enough from the mitral valve plane to facilitate manipulation of the device in the left atrium. Immediately following puncture, the guidewire was advanced to the pulmonary vein and the interatrial septum was dilated with a 5.0×40 mm balloon. The transseptal catheter was then removed and subsequently exchanged for a 24-Fr delivery catheter (22 Fr at its distal end) with the clip attached to the distal end. The clip was then orientated to the left atrium through the mitral valve, and was positioned in the ventricle. With the help of TEE, the device was carefully pulled back, and both leaflets were grasped (Figure 2 A & B). Once in place, the clip was closed. Finally, after confirming its adequate positioning, absence of significant atrioventricular gradient (should

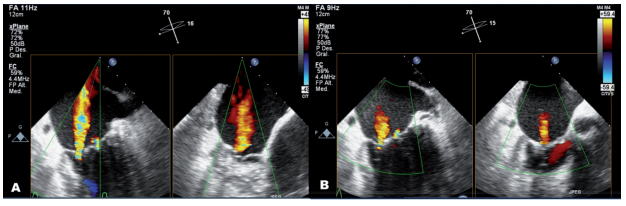

Fig. 1. Mid esophageal four chamber TEE view (X-plane) showing severe mitral regurgitant jet (**A**). After the implantation, reduction of the jet to a mild degree is observed (**B**).

be ≤5 mm Hg), and adequate reduction of regurgitation, the clip was deployed (Figure 1 and Figure 2 C and D).

If there is no significant change in MR, the clip can be opened and repositioned as many times as necessary. If, despite good positioning, mitral regurgitation persists, a second and even a third clip can be deployed in order to reach the lowest possible MR.

The course was asymptomatic during the 30-day follow up, showing mild MR and reduction in the left atrial diameter (50 mm).

Percutaneous mitral valve repair with MitraClip™ implantation is a therapeutic option to repair surgery or valve replacement in selected patients who are not candidates for surgery or have very high perioperative risk. At present, this is the only device applied clinically in Argentina, with over 25,000 cases worldwide. It has been approved by the European Community (EC Mark) for the treatment of primary and secondary MR, and by the United States of America (Food and Drug Administration) for primary MR, in all cases in patients with high perioperative risk. The initial clinical experience with this technique in patients with functional MR has shown its short-term efficacy and safety; however, the stability of long-term results is still unknown. Three multicenter, randomized trials (COAPT, RESHAPE-HF, and MITRA-FR) are currently evaluating the role of this percutaneous technique in the context of functional MR, and their results will be available next year. Meanwhile, a minimally invasive approach with this technique in high risk patients with no other effective therapeutic option appears to be an adequate approach, as was the case in this report in which a reduction of MR from severe to mild was achieved, resulting in significant symptomatic improvement.

The implantation of these devices not only improves the functional class, but considerably reduces readmissions, and probably increases survival rate compared with the conservative approach (only therapeutic option for the majority of these high risk patients).

Adequate patient selection is essential to ensure success; for that purpose, a multidisciplinary approach that includes clinical and interventional cardiologists, cardiac surgeons, imaging specialists and anesthesiologists is necessary. In this regard, the Heart Team concept gains strength: a team that evaluates each patient comprehensively and decides on the best therapeutic strategy. The utilization of TEE, especially 3D TEE, is of vital importance to visualize the anatomy of the mitral valve (and subvalvular) apparatus, and to guide the procedure while angiographic images are not as relevant as in other percutaneous interventions.

In conclusion, a new device –among many others currently being developed– emerges as an alternative for patients with severe MR and no other effective treatment option, repairing the MR and improving symptoms. Further studies comparing this technique with valve surgery or simply versus medical treatment will shed light on its role in the management of this valve condition.

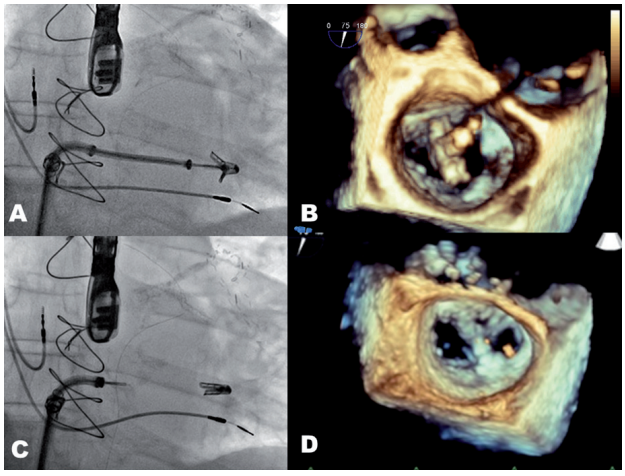

Fig. 2. Position of the clip guided by angiography (**A**) and 3D echocardiography (atrial view), showing the clip at the level of segments A2-P2 (**B**). Correct clip deployment is confirmed (**C** and **D**).

Conflicts of interest

None declared.

(See authors' conflicts of interest forms on the website/Supplementary material).

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REV ARGENT CARDIOL 2016;84:473-474. http://dx.doi.org/10.7775/rac.v84.i5.8956

Spontaneous Echo Contrast in the Right Heart Chambers of a Patient with Autoimmune Hemolytic Anemia

Spontaneous echo contrast (SEC) is an echocardiographic phenomenon characterized by slow intracavitary contrast movement, like a "smoke cloud". It is caused by the aggregation of red blood cells, which provides blood with echogenic capacity.

We report a case of intense SEC in a rare location. The case corresponds to a 24-year-old male patient with history of systemic lupus erythematosus (SLE), who consulted for a 48-hour history of arthritis in the left knee. On admission, his blood pressure was 95/60 mmHg, his heart rate 120 bpm, and axillary temperature 38 °C.

Lab tests: Hematocrit 25%, hemoglobin 8 g/dl; WBC 12,000/mm3; platelets 120.000/mm3; total bilirubin 1 mg/dl; positive direct and indirect Coombs tests with cold agglutinins in serum.

Streptococcus agalactiae was isolated from blood cultures and synovial fluid. The patient was hospitalized with diagnosis of septic arthritis associated with hemolytic anemia. Antibiotic therapy with imipenem/vancomycin was started. The patient progressed to septic shock, requiring mechanical ventilation. Hydrocortisone and IV gamma globulin therapy was initiated.

Transthoracic echocardiography showed left heart chambers with normal diameters and left ventricular systolic function and presence of dense SEC in non-enlarged right chambers, inferior vena cava and suprahepatic veins (Figure 1A and B). Moderate pericardial effusion and mild mitral, aortic, and tricuspid regurgitation was also observed. Echocardiography was performed without echographic contrast, and no other clear bubble source was found.

An Echo-Doppler of the lower limbs showed no signs of venous thrombosis, and a chest CT scan with intravenous contrast ruled out pulmonary thromboembolism (PTE).

Shock improved and the hematocrit was stabilized without transfusion or anticoagulant therapy.

Transthoracic echocardiography was repeated one week after the first study showing SEC absence (Figure 2A and B). Subsequently, a transesophageal echocardiography confirmed disappearance of SEC and absence of vegetation.

Normally, during echocardiography, cardiac chambers are free from echoes because ultrasound reflection on blood is not intense enough to generate images. However, under blood congestion, red cells stack together (rouleaux), which provides echogenic capacity that can be observed as a "smoke cloud". (1)

Spontaneous echo contrast depends on the concentration of red blood cells, blood velocity, and presence of plasma proteins. (2) Individual red blood cells are normally prevented from aggregating by the repulsive electrostatic effects of their negatively charged surface caused by plasma proteins, particularly fibrinogen. (3) Spontaneous echo contrast generation is more common with increased fibrinogen, which would act as a screen on which erythrocyte aggregation occurs more easily. (2, 4)

Spontaneous echo contrast is observed by transthoracic echocardiography in left chambers in 0.1% to 3.5% of studies in patients with conditions causing blood stasis, such as chronic atrial fibrillation, severe mitral stenosis, and dilated cardiomyopathy with low cardiac output. It is considered to predict embolism, so patients should be considered for anticoagulant therapy. (2)

Although most often detected in the left atrium and left atrial appendage, SEC has also been described within the left ventricle, right heart chambers, descending aorta, pulmonary artery, and inferior vena cava. (1) Cases of SEC in the right atrium and ventricle have been reported in patients with autoimmune hemolytic anemia and erythrocyte aggregation mediated by auto-antibodies. (5, 6)

Miller et al. reported the case of a 9-year-old girl with a history of Evans syndrome (thrombocytopenia and autoimmune hemolytic anemia), who was admitted due to fulminant disseminated aspergillosis. During hospitalization, the echocardiogram showed SEC in the right heart chambers (not in the left chambers) resulting from red blood cell agglutination mediated by 'warm' IgM antibodies. Autopsy revealed pulmonary thromboembolism (PTE) and infarction in lungs, brain, liver, and kidneys. (5)

Similarly, Dogan et al. reported the case of a 67-year-old male patient with SEC in the right heart

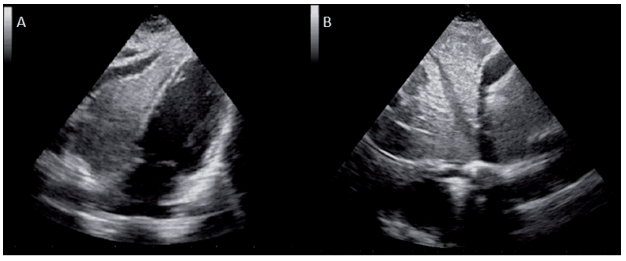

Fig. 1. Transthoracic echocardiography, subcostal view. Spontaneous echo contrast in the right chambers (**A**) and in the inferior vena cava and suprahepatic veins (**B**).

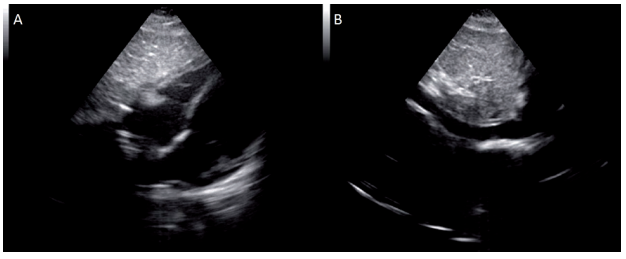

Fig. 2. Transthoracic echocardiography, subcostal view. Disappearance of spontaneous echo contrast in the right chambers (**A**) and in the inferior vena cava (**B**).

chambers and chronic obstructive pulmonary disease, who was admitted due to pneumonia and autoimmune hemolytic anemia. In addition to PTE, this patient had pulmonary hypertension and right ventricular enlargement. Spontaneous echo contrast in the right heart chambers disappeared after a week with corticoid therapy and anticoagulation with low-molecular-weight heparin and warfarin. (6)

We report the case of a patient with SLE, septic shock, and autoimmune hemolytic anemia, with intense SEC detected in a rare location (right heart chambers, inferior vena cava, and suprahepatic veins). Different from the cases described above, no signs of PTE were found in our patient. Moreover, SEC disappeared with the remission of shock and "hemolytic crisis" under corticoid and gamma globulin therapy, and without anticoagulant therapy. Therefore, it would be reasonable to assume that the major SEC mechanism was erythrocyte aggregation caused by the interaction between red blood cells and auto-antibodies. The lower shear stress in the venous circulation (especially in septic shock) would contribute to SEC production in the right heart circulation. The absence of SEC in the left heart chambers would occur because the erythrocyte aggregation in "stacks of coins" or rouleaux disintegrate in the pulmonary microcirculation or are retained by the pulmonary capillaries acting as filter.

According to our review of the literature, this would be the first report of spontaneous echo contrast caused by this mechanism, in the absence of PTE, and disappearing without anticoagulant therapy.

Conflicts of interest

None declared.

(See authors' conflicts of interest forms on the website/Supplementary material).

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REV ARGENT CARDIOL 2016;84:475-476. http://dx.doi.org/10.7775/rac.v84.i5.9067

Recurrent Syncope in Patient with Multinodular Goiter

Syncope is a rare clinical manifestation of cervical tumors. (1) Its pathophysiology lies in the mechanical compression or irritation caused by the tumor on the carotid sinus and the IX cranial nerve (glossopharyngeal nerve). As a result, an exaggerated baroreflex response is triggered as the precursor of the so called neuromediated syncope. (2) We report a rare case of cervical goiter first manifested as syncope.

The case corresponds to a 67-year-old hypertensive and diabetic female patient, with chronic anemia secondary to atrophic gastritis. She had daily syncopal episodes of 2-month evolution, associated with movements of the neck and upper limbs. Physical examination revealed a palpable cervical tumor. Baseline ECG showed a sinus rhythm of 65 bpm, first-degree atrioventricular (AV) block (PR interval 220 ms), narrow QRS, and no other abnormalities (Figure 1A). Electrocardiographic monitoring during syncopes revealed a paroxysmal AV block with QRS intervals >3.5 seconds (Figure 1B and C). A thyroid ultrasound followed by a thorax CT scan confirmed the presence of a large left-sided multinodular goiter with bilateral

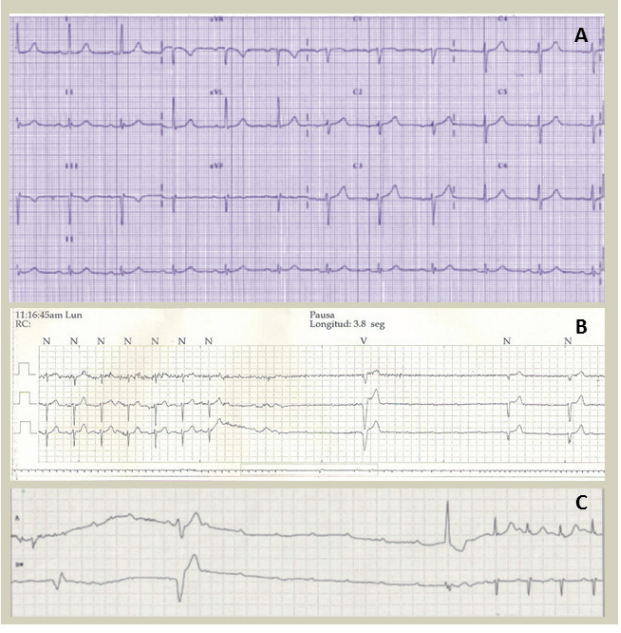

Fig. 1. A. Baseline ECG showing sinus rhythm with prolonged PR interval. **B** and **C**. Complete paroxysmal AV block during two syncopal episodes.

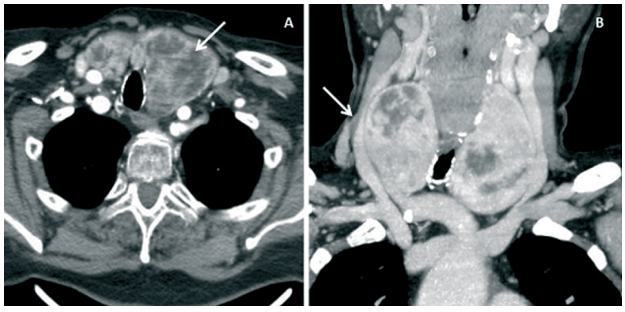

Fig. 2. Intrathoracic multinodular goiter. **A.** Axial plane computed tomography scan with contrast: large, solid, heterogeneous, well-defined tumor (arrow) that shifts the tracheal lumen to the right. **B.** Coronal multiplanar reconstruction showing the mass extending into the superior mediastinum to both carotid spaces.

extension to the carotid axes (Figure 2). No significant angiographic stenoses were observed in the carotid axes. The thyroid function test was compatible with normality. The echocardiography and electroencephalogram showed no pathological findings.

Placement of temporary pacemaker followed by surgery of the cervical goiter was performed. During postoperative hospitalization, the patient did not experience syncopes or dizziness, and no rhythm disturbances were monitored, so a permanent pacemaker implantation was unnecessary. The patient remained asymptomatic for a year. Thirteen months later, the patient consulted for lightheadedness. Electrocardiographic monitoring registered a second-degree AV block without cervical goiter recurrence; therefore, a dual-chamber permanent pacemaker was implanted.

Neurally mediated or reflex syncope derived from hypersensitivity of the carotid sinus is part of the "carotid sinus syndrome" (CSS). Typically, this syndrome has been divided into three types: cardioinhibitory, vasodepressor, and mixed type. The cardioinhibitory type progresses with sinus bradycardia or asystole, and rarely causes atrioventricular conduction disorders. (3) In our case, the patient had symptomatic complete AV block without sinus node dysfunction. Space occupying lesions in the head and neck commonly associated with CSS are glottis and nasopharyngeal squamous carcinomas, metastatic lymph nodes, parotid tumors and abscesses, among others. (4) This association between cervical mass and CSS has been described as "parapharyngeal space syndrome". In our case, the goiter extending into the supra-aortic trunks led us to consider the compression or irritation of the carotid sinus or glossopharyngeal pathway. After mass removal, the patient remained asymptomatic during hospitalization, and no rhythm disturbances were monitored. However, a year later the patient presented with lightheadedness and evidence of advanced AV block, so a permanent pacemaker was placed. On admission, the ECG showed a first-degree AV block with not very prolonged PR interval (220 ms) and narrow QRS, with no other abnormalities. We therefore believe that the cervical goiter was the extrinsic precipitating cause of the syncope. A limitation of this case was the inability to determine whether it was an infra-Hisian block, because a His bundle electrogram was unavailable; however, it is likely that, based on the patient's ECG and paroxysmal AV block, the electrophysiological study would not have been conclusive, and conduction intervals most likely would have been normal. Recurrence after a year suggests that, in cases of CSS with atrioventricular conduction disorders, instead of sinus dysfunction, the syncope etiology can be mixed and with an intrinsic conduction disorder, requiring longer follow-up.

Conflicts of interest

None declared.

(See authors' conflicts of interest forms on the website/Supplementary material).

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