

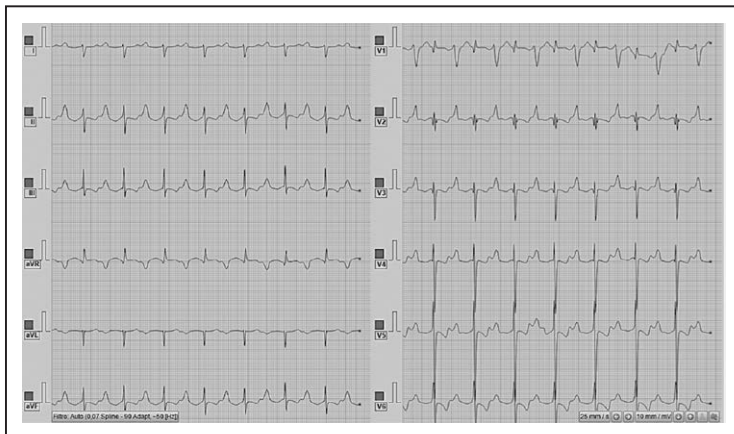
# What Is the Structural Abnormality in This Patient?

## ECG CHALLENGE

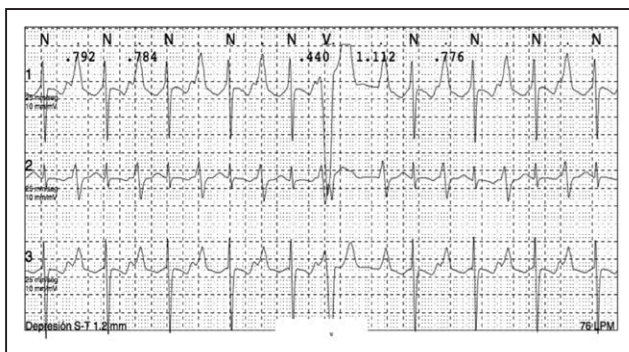
A 59-year-old man presented to our hospital reporting 3 months of exertional dyspnea, ortopnea, paroxysmal nocturnal dyspnea, and lower leg edema. On physical examination, he had jugular venous distention, bibasilar rales, large tender liver, and peripheral edema. On cardiac auscultation, he had a regular heart rate with premature ventricular contractions, systolic ejection grade III/VI cardiac murmur in pulmonic area, and louder  $P_2$  than  $A_2$ . Chest x-ray showed cardiac enlargement, and an ECG (Figure 1) was recorded; because of premature heartbeats on admission, a 24-hour Holter ECG was obtained (Figure 2). On the basis of ECG findings, what is the rhythm of the patient and what is the structural abnormality?

Please turn the page to read the diagnosis.

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**Figure 1.** ECG obtained during emergency department admission.



**Figure 2.** Twenty-four-hour Holter ECG showing 1 premature ventricular contraction.

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## RESPONSE TO ECG CHALLENGE

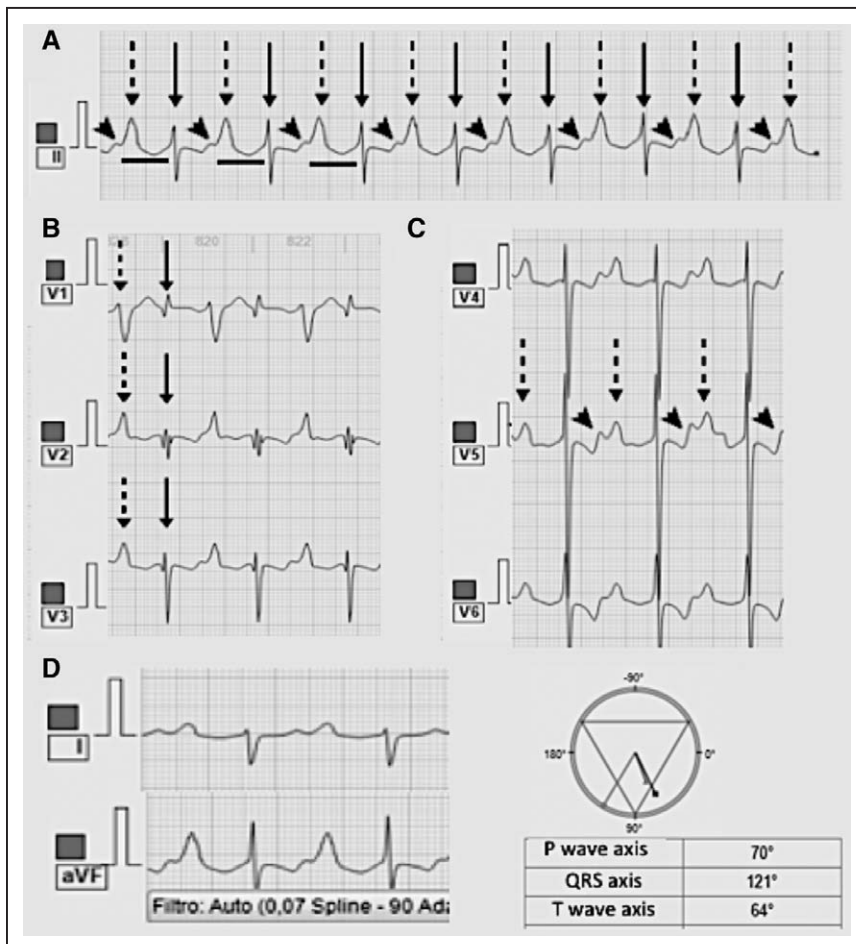
To interpret the ECG and determine the anatomic abnormality in this case, it is necessary to take into account the clinical and ECG findings in combination, which are remarkable for right ventricle (RV) failure as demonstrated by the findings on physical examination of jugular venous distention, large tender liver, and peripheral lower leg edema. In addition, there is a systolic murmur, fixed splitting of  $S_2$  and louder  $P_2$ , thus suggesting an interatrial septal defect.

The ECG shows a sinus rhythm with an extremely tall P wave in inferior and precordial leads and notable increased duration and depth of terminal-negative portion of P wave in V1 (mimicking rS morphology of a QRS). Other ECG findings include first-degree atrioventricular conduction block, RV hypertrophy, and nonspecific repolarization changes (Figure 3). The high-voltage P waves are observed in the 24-hour Holter ECG as well, where they can even generate confusion with the QRS morphology, suggesting ventricular bigeminy, but following them through the other channels clarifies that they represent P waves, which in second channel have even higher voltage than QRS (Figure 4).

Cardiac magnetic resonance imaging confirmed an ostium secundum atrial septal defect (ASD) of 13 mm,

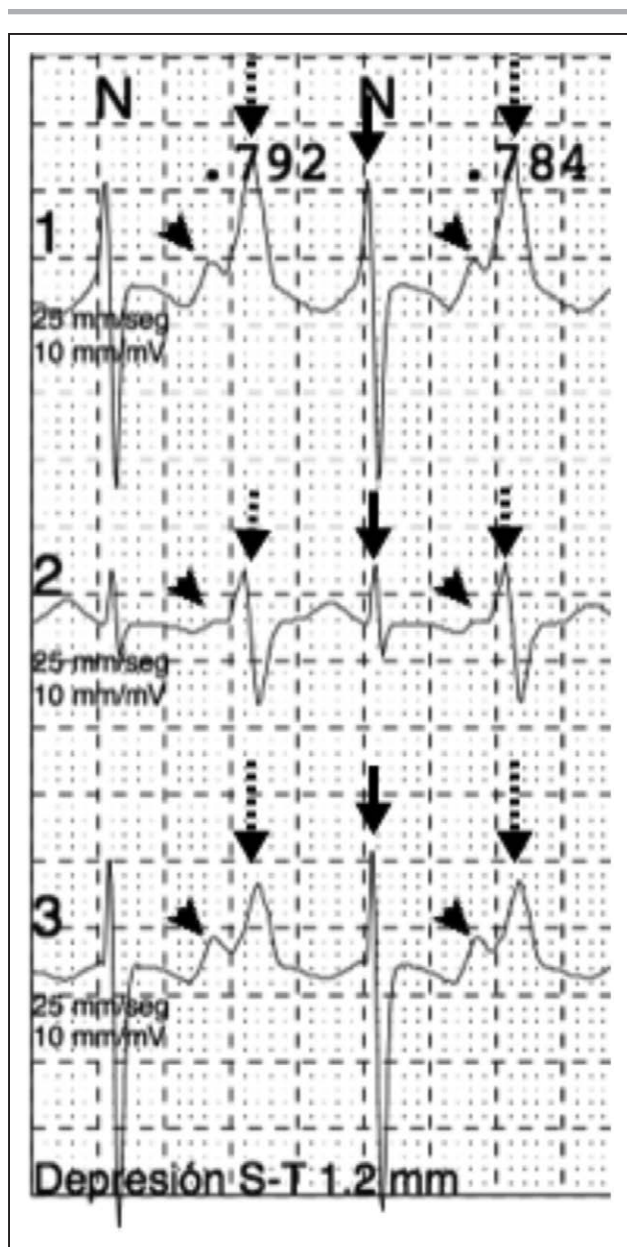
left-to-right shunt,  $Q_p/Q_s=2.1$ , giant right atrium (end-systole area of  $63\text{ cm}^2$ ; normal reference,  $<18\text{ cm}^2$ ), and signs of volume and pressure overload to the RV with severe dilation (diastolic diameter, 59 mm), mild hypertrophy, TAPSE (Tricuspid Annular Plane Systolic Excursion): 6 mm, and a RV ejection fraction of 16% (Figure 5). The patient also had a severe impairment of left ventricle function with an ejection fraction of 15%; however, no explanation was found for this latest finding after the diagnostic workup.

ASDs are among the most common presentations of congenital heart diseases in adults. ECG characteristics of ASD include right bundle-branch block, which could be incomplete because of RV hypertrophy. Right QRS axis deviation is associated with ostium secundum ASD, the QRS is leftward in ostium primum defects, and inverted P waves in inferior leads may be seen in sinus venosus ASD. Carmichael et al<sup>1</sup> described that, in patients with ASD, right bundle-branch block pattern and RV hypertrophy were seen in 66% and 25% of cases, respectively. Patients with normal pulmonary pressure more commonly have right bundle-branch block, whereas patients with higher pulmonary pressure develop qR morphology in V1 with incomplete right bundle-branch block. As an explanation to this, they argued that the higher the pressure in the RV, the higher the prob-



**Figure 3. ECG findings.**

Sinus rhythm, atrioventricular delay with a PR segment of 400 ms (**A**, solid line), high-voltage P waves are identified (dotted arrow in **A**, **B**, and **C**), and they precede every regular QRS complex (solid arrow in **A** and **B**), P waves have higher amplitude than QRS in V1 (**B**); QRS axis deviated to the right ( $121^\circ$ , **D**), and morphology is compatible with incomplete RBBB because of qR in V1 (**B**) and right ventricle hypertrophy based on  $R/S < 1$  and  $S > 7$  mm in lead V6 (**C**),  $R$  in V1 +  $S$  in V6  $> 10.5$  mm and deep S waves in V4 to V6 (**B** and **C**). T waves are represented by arrow head in DII and V5 (**A** and **C**). RBBB indicates right bundle-branch block.



**Figure 4.** Twenty-four-hour Holter where dotted arrow indicates P wave, solid arrow is pointing to the QRS complex, and the arrow head is marking T wave. See the amplitude of P wave in channel 2 being much higher than QRS resembling ventricular bigeminy.

ability of developing RV hypertrophy, which generates an electric vector that counterbalances the left ventricle depolarization in V1; therefore, a predominantly positive deflection is seen in this lead. These findings are consistent with those in our patient (see Figure 3B); he had right-axis deviation and qR morphology in V1 that was consistent with his severe pulmonary artery hypertension, thus explaining clinical and ECG changes of RV hypertrophy and failure.

The most noteworthy abnormality in our ECG was the high-voltage P waves shown in the emergency department ECG and 24-hour Holter strips. P-wave abnor-

malities in patients with ASD are secondary to the atrial enlargement related to RV volume overload as we have shown in our case. P-wave enlargement has been described in up to 90% of patients with ASD, being more common than typical QRS findings of ASD such as axis deviation.<sup>2</sup> Criteria for right atrial abnormality are met by our patient; these include amplitude in lead II >2.5 mm and prominent initial positive in V1 and V2, findings that have a specificity of 100% but low sensibility.<sup>3</sup> However, we were highly impressed with 2 aspects regarding the P wave of our case: first, the surprisingly high voltage in inferior and precordial leads, and, second, the marked increase in duration and depth of terminal-negative portion of P wave in V1. We did not find similar descriptions that linked such P-wave changes with ASD or other kinds of congenital heart disease in adults. We hypothesize that those changes are related with the severe right atrium dilation, increased atrial mass, and changes in the depolarization vector. The MRI findings are consistent with giant atrium, which may or may not be related to his ASD.

In summary, the ECG presented in this case shows changes consistent with secundum ASD, which include rightward axis deviation and incomplete right bundle-branch morphology. In addition, the ECG also showed extremely high-voltage P waves that correlated with a large dilated right atrium on cardiac MRI. The MRI fits criteria for giant atrium. The presence of such P waves is not commonly described on ECGs of patients with ASD, and this finding made the approach to our patient difficult at the beginning because the P waves were misinterpreted as bigeminy rhythm on ECG and 24-hour Holter monitoring.

## ACKNOWLEDGMENTS

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## DISCLOSURES

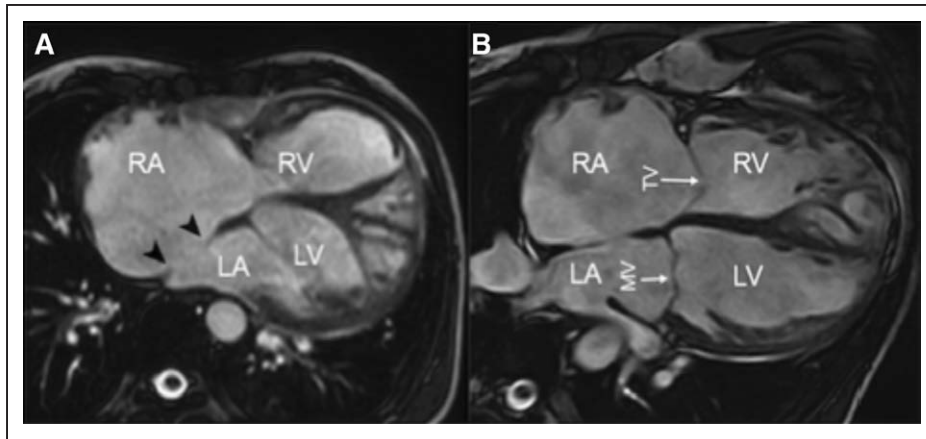
None.

## FOOTNOTES

*Circulation* is available at <http://circ.ahajournals.org>.

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**Figure 5. Cardiovascular magnetic resonance.**

Axial (A) and 4-chamber (B) steady-state with free precession (SSFP) images correlating with physical and ECG findings. See the huge right atrium and the atrial septal defect (ASD) with black arrow heads pointing to the tips of the ASD. LA indicates left atrium; LV, left ventricle; MV, mitral valve; RA, right atrium; RV, right ventricle; and TV, tricuspid valve.

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