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 Introduction {#s0005} ========== Ebstein's anomaly is an uncommon congenital anomaly of the tricuspid valve characterized by the presence of a marked apical displacement of the atrialized portion of the right ventricle with a cuspidial-free wall that opens to the left atrium. This leads to an increase in ventricular interatrial shunt and causes progressive ventricular dysfunction, pulmonary artery hypertension, and failure. The syndrome is the result of arrested pulmonary venous return, which occurs between 2 and 6 months of gestation. At presentation, these patients are cyanotic and have a murmur of pulmonic insufficiency. However, the murmur usually becomes silent within the first months of life, and patients show a satisfactory exercise tolerance with no clinical or hemodynamic signs of right ventricular failure. Echocardiography is the primary tool for diagnosing Ebstein's anomaly and for monitoring clinical outcome in these patients. Because there is no medical therapy proven to reduce morbidity and mortality, surgery is the only effective therapeutic modality. Case report {#s0010} ========== An 18-year-old man presented to the emergency room with a 6-month history of palpitations and orthopnea. He was admitted to the hospital after having been asymptomatic for 2 days. Physical examination revealed a blood pressure of 130/90 mmHg, heart rate of 88 beats per minute, a regular heart rhythm, no edema, no jugular venous distension, and a normal precordium. Chest auscultation revealed a holosystolic murmur in the right infrascapular region. The blood sample showed a normal serum level of creatinine, but the levels of sodium, potassium, and urea were low. Electrocardiography revealed normal sinus rhythm with a ventricular rate of 60 beats per minute. A transthoracic echocardiogram revealed the presence of a thin, markedly hypertrophied interatrial septum and a markedly enlarged right ventricle with an end-diastolic diameter of 74 mm. There was no significant tricuspid regurgitation and no pericardial effusion. There was poor acoustic enhancement of the interatrial septum ([Figure 1A](#f0005){ref-type="fig"} and [Video 1 520fdb1ae7