

1:1 Atrial Flutter After Vernakalant Administration for Atrial Fibrillation Cardioversion

Flutter auricular 1:1 tras la administración de vernakalant para cardioversión de fibrilación auricular

To the Editor,

Vernakalant is a novel antiarrhythmic drug that has proved its efficacy at restoring sinus rhythm in recent-onset atrial fibrillation (AF). Its mechanism of action is based on selective partial blocking of potassium currents in the atrial myocardium, prolonging the atrial refractory period without significantly affecting ventricular refractoriness. This makes it potentially beneficial as a low-effect ventricular proarrhythmogenic, even in patients with structural heart disease.

Class IC drugs—used to restore sinus rhythm and prevent AF recurrence—favor the appearance of flutter, often with 1:1 ventricular conduction. Vernakalant also appears to favor the development of flutter, although 1:1 atrioventricular conduction has not been reported to date.

We present the case of a 77-year-old man with coronary disease without infarction, with hypertensive cardiopathy, normal ventricular function, and 1 previous episode of persistent AF requiring electrical cardioversion. Treatment was with acenocoumarol, enalapril, and simvastatin.

The patient was referred for electrophysiologic study for typical paroxysmic atrial flutter with 240 ms cycle length in the electrocardiogram.

Initially in sinus rhythm, during placement of the circular multipolar catheter used to record electrocardiograms and for

stimulation he developed sustained AF after a 10-min observation period.

We decided to use intravenous vernakalant for cardioversion. After a first 3 mg/kg infusion, the AF organized into flutter with 320 ms cycle length, descending atrial activation sequence in the anterior right atrium, and exact return cycle in the cavotricuspid isthmus, compatible with typical flutter (Fig. 1).

Atrioventricular conduction was initially variable, later stabilizing to 1:1 with right bundle branch block (Fig. 2).

Given good tolerance despite rapid ventricular frequency, we decided to interrupt the drug infusion and perform radiofrequency ablation of the cavotricuspid isthmus.

The Class IC antiarrhythmic drugs flecainide and propafenone slow atrial conduction by blocking voltage-dependent rapid sodium channels, favoring the stability of macro re-entry circuits in anatomic regions with predisposed structures (IC flutter).¹

In the right atrium, they condition the slowing of atrial conduction and limit transversal conduction through the crista terminalis, facilitating the appearance of flutter circuits around the tricuspid annulus.

Due to atrial conduction slowing, IC flutter is usually slow and can be led 1:1 to the ventricles. This greatly accelerates ventricular frequency that is often accompanied by aberrant conduction, which can condition poor hemodynamic tolerance of the arrhythmia.

Although experience in the clinical use of vernakalant is very limited, data on its efficacy and safety in 4 controlled clinical trials has been published.²⁻⁴

Vernakalant has demonstrated efficacy superior to a placebo plus amiodarone in cardioversion of recent-onset AF (52% vs 4%-5% at 90-min observation) with 8% post-dose incidence of atrial flutter—which is far superior to amiodarone (0.9%), above all in patients receiving antiarrhythmic drugs.⁴

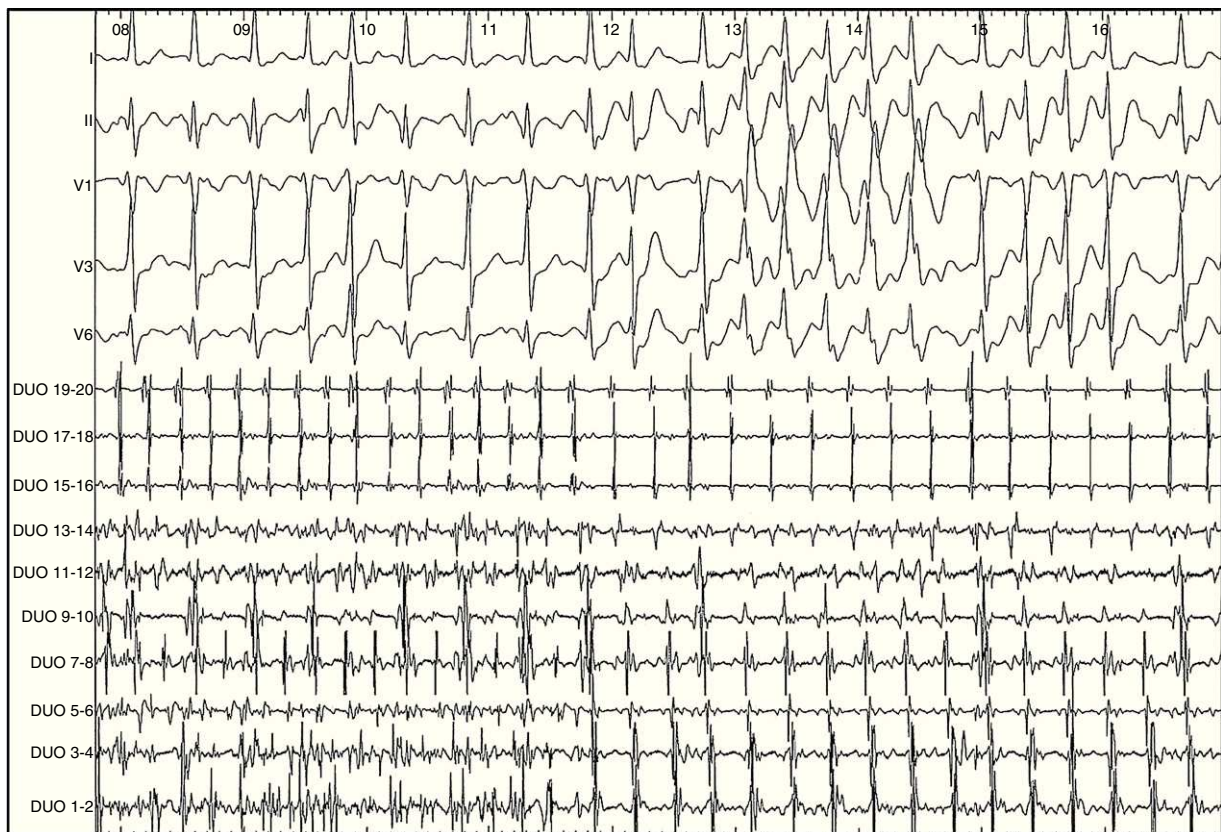


Figure 1. Surface and intracavity electrocardiogram of 12-pole catheter showing the change from atrial fibrillation to flutter.

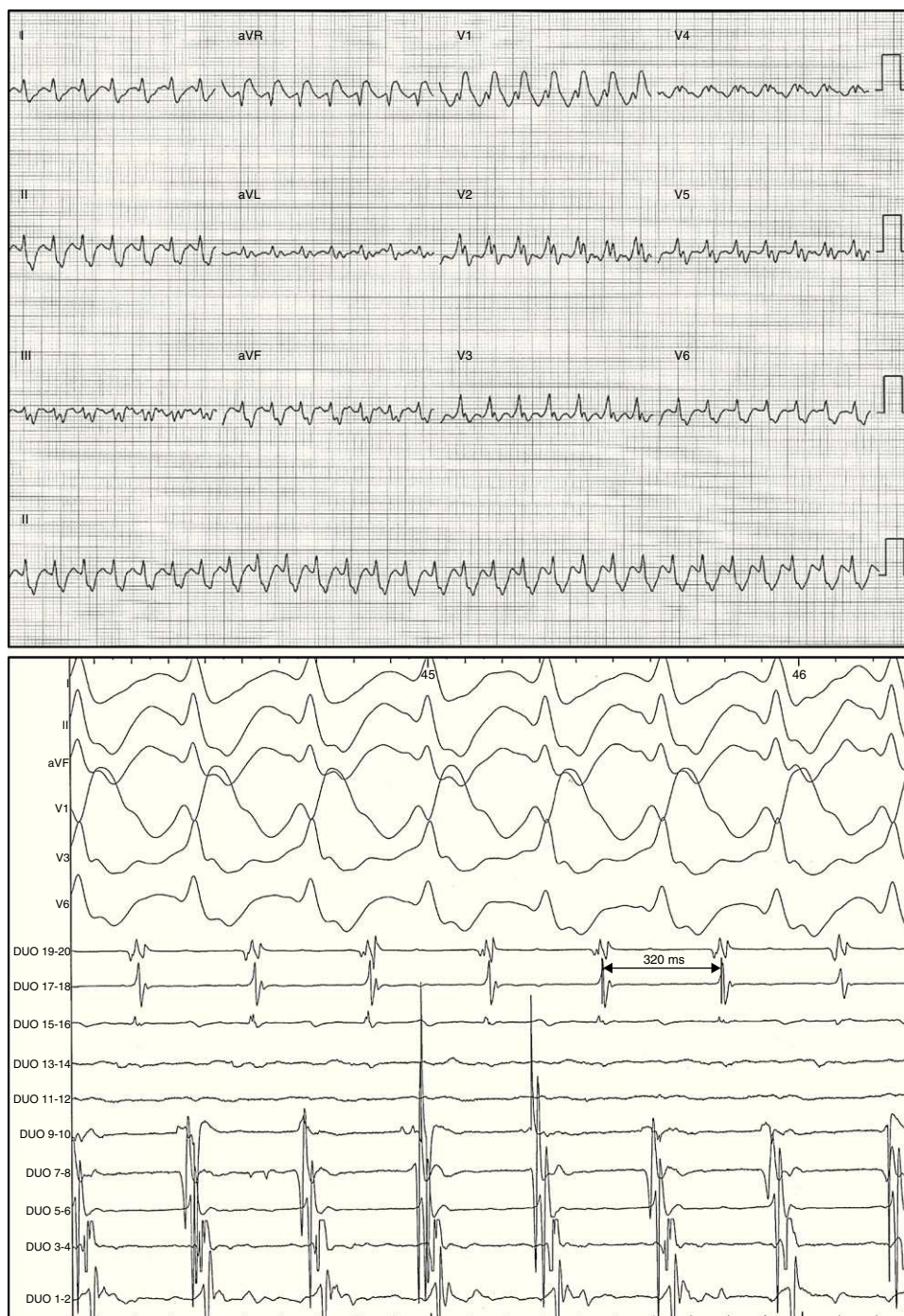


Figure 2. Electrocardiogram and intracavity images of flutter with descending atrial activation sequence in anterior surface, 320 ms cycle length, 1:1 atrioventricular conduction, and right bundle branch block.

Flutter usually appears in the transition to sinus rhythm. To date, neither poor tolerance nor 1:1 atrioventricular conduction have been reported; hence, suspending drug administration when flutter appears seems unnecessary.

One recent study did not demonstrate the efficacy of vernakalant in the cardioversion of flutter.⁵ The present study has identified a significantly prolonged flutter cycle length (mean, 55 ms), accompanied by the slight slowing of atrioventricular

conduction, which explains why—at the time of writing—1:1 atrioventricular conduction has not been reported.

The principle mechanism of action of vernakalant is based on blocking potassium currents present in the atrium (I_{Kur} and $I_{K,ACh}$), prolonging the refractory period and potential for atrial action, which produces antifibrillatory action.⁶ Moreover, in vitro studies have proven vernakalant blocks voltage-dependent sodium channels and that inhibition is greater at higher heart rates—as in AF.

In this patient, slowing atrial conduction—possibly due to sodium channel blocking—could have favored the appearance of slow flutter, which permitted 1:1 conduction to the ventricles.

Although infrequent, this pattern of proarrhythmia should be borne in mind given the foreseeable expansion in the use of vernakalant even though, in our opinion, it does not limit the drug's clinical usefulness.

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Late Development of Pulmonary Arterial Hypertension After Arterial Switch for Transposition of the Great Arteries

Desarrollo tardío de hipertensión arterial pulmonar en paciente con transposición de grandes arterias sometido a switch arterial

To the Editor,

Pulmonary hypertension rarely develops in patients with transposition of the great arteries (TGA) who undergo surgical correction with no residual defect.

We report the case of a male patient diagnosed with TGA associated with an intact ventricular septum at birth. On the first day of life, he underwent cardiac catheterization for Rashkind balloon atrial septostomy. Pulmonary artery pressure was above systemic pressures. When the patient was 11 days old, he underwent definitive correction by means of an arterial switch procedure and closure of the atrial septal defect. He was followed-up in the pediatric cardiology department, and remained asymptomatic with no clinical signs or results in the complementary tests (including echocardiography) that might have led to suspicion of pulmonary hypertension. When aged 9 years, he started to feel tired after increasingly less effort (New York Heart Association functional class III). In the 6-min walk test, he covered

480 m and his heart rate increased from 90 to 120 bpm. In the tests performed, the electrocardiogram showed right-sided predominance. Echocardiography showed good ventricular contractility, and a ventricular eccentricity index of 1 (Fig. 1). No other abnormalities were suggestive of pulmonary hypertension. In view of the discrepancy between the clinical symptoms and the complementary tests, we decided to perform cardiac catheterization, which showed pulmonary arterial hypertension above the systemic blood pressure (Fig. 2) with high pulmonary artery resistance (11.7 UW), normal pulmonary capillary wedge pressure, and limited response to pulmonary vasodilators. In view of this diagnosis, we decided to initiate treatment with bosentan 62.5 mg/12 h and sildenafil 2 mg/kg/day. After 1 year, clinical improvement was seen in the 6-min walk test, in which the patient covered 616 m with a lower increase in heart rate (65 to 81 bpm). Cardiac catheterization was repeated, and a substantial decrease in pulmonary pressures to 50% of the systemic pressures was observed, along with a decrease in pulmonary artery resistance to 5.4 UW.

Pulmonary arterial hypertension associated with congenital heart disease is included in the first group of the clinical classification of pulmonary hypertension of the Dana Point Meeting, the group of pulmonary arterial hypertension. Our patient belonged to the fourth subgroup of pulmonary arterial hypertension associated with congenital heart disease,

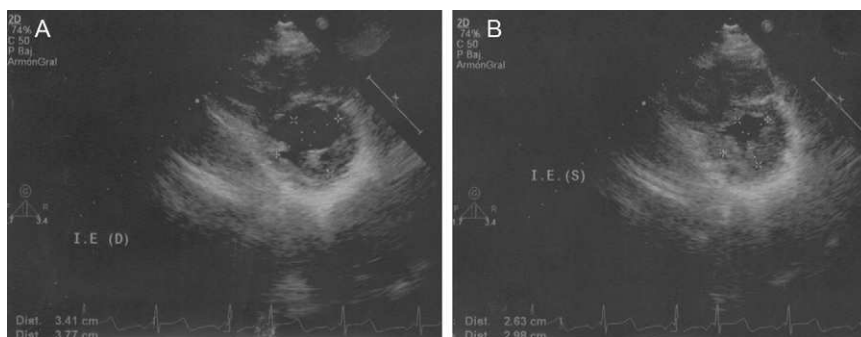


Figure 1. Transthoracic echocardiography in the parasternal short-axis plane. Measurement of the eccentricity index of the left ventricle in diastole (A) and systole (B).