Propafenone, a class IC antidysrhythmic drug, is frequently used for pharmacological cardioversion of atrial fibrillation. Using IC antidysrhythmics to reverse recurrent atrial fibrillation is associated with a 3.5–20% incidence of atrial flutter with either “classical” or atypical patterns of atrial activation [1–3]. On the contrary, the appearance of peaked alternating with normal P waves immediately after successful cardioversion of atrial fibrillation with propafenone has not been described.

A 59-year-old woman was admitted to our Cardiology unit for a recent onset (2 h) atrial fibrillation. Four years before she underwent a pharmacological cardioversion of atrial fibrillation, and at the time of admission she was not taking drugs. Routine laboratory analyses were normal, and echocardiography showed normal dimension of both atria and normal ejection fraction (60%). The patient underwent pharmacological cardioversion (propafenone 2 mg/kg bolus i.v. followed by 560 mg i.v. slow infusion over 24 h) with rapid restoration of sinus rhythm. After cardioversion the electrocardiogram showed the rapid alternation of a normal and a “right” P wave for about 20 h (Fig. 1), together with ST-T changes that were more evident in leads with the tallest P waves (D2, D3, AVF) (Fig. 1b). P-P interval ranged between 560 ms, corresponding to the highest voltage (0.4 mV) P waves (Fig. 1b), and 800 ms after the restoration of normal voltage (0.1 mV) P waves (Fig. 1f). No sign of atrial stunning was evident at an echocardiography performed immediately after cardioversion (E wave 7 cm/s; A wave 6 cm/s). Six months later the patient was still in sinus rhythm, and the echocardiographic parameters were unchanged.

A peaked P wave is consistent either with increased right atrium mass or with verticalization of the heart, both conditions frequently associated with severe COPD or pulmonary hypertension [4, 5]. However, the patient was free from pulmonary diseases. Furthermore, the P wave changes secondary to any of these conditions should be stable. Even the possibility that propafenone activated an ectopic pacemaker, probably located near the superior vena cava, is unlikely because IC antidysrhythmics are known to depress ectopic pace-makers [6, 7]. Analogously, the possibility that the giant P wave reflected atrial stunning, a mechanical dysfunction that has been described following both electrical and pharmacological cardioversion, [8, 9], conflicts with post-cardioversion echocardiography. However, even a brief period of high atrial frequency can induce an electrical remodeling [10], both of sinus node and of atrial electrophysiologic substrate [11, 12], without an evident atrial mechanic dysfunction. In fact temporal patterns of progression and regression of electrical and mechanic remodeling of the atrium are different [13]. Thus, it can be hypothesized that cardioversion could have determined a progressively reversible electrophysiologic remodeling [11]. Finally, the observed changes in P waves morphology could reflect the shifting of pacemaker, a phenomenon that could be determined or influenced by...
sympathovagal balance [14]. Indeed, propafenone could have conditioned P wave changes through its beta-blocking properties [15]. Accordingly, the different P–P intervals for the different P wave morphologies likely indicate changing sympathovagal balance.

Whichever is the pathogenesis of the observed transient peaked P wave, it seems of interest to assess the prevalence of this finding after cardioversion as well as to verify whether it heralds further atrial dysrhythmias or, else, marks a procoagulatory condition.

**Conflict of interest statement** The authors declare that they have no conflict of interest related to the publication of this manuscript.

**References**