increase in hemorrhagic events. Furthermore, this analysis does not address the economic cost-benefit, especially if the new oral anticoagulants are prescribed.

With regard to the recommendations on dronedarone, although the guidelines do not explicitly recommend its use in patients with permanent AF, this is considered reasonable therapy for long-term control of heart rate (IIA, evidence level B), which could lead to confusion. The publication of the PALLAS study and the recent recommendations from medications agencies, compiled in our article, have resolved these questions and clarified, at least for the time being, the role of dronedarone in AF by confirming that the importance given by the guidelines to this drug was hasty and unprecedented in the history of ESC guidelines.

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Ajmaline Test and ESC 2010 Clinical Practice Guidelines on Atrial Fibrillation

Test de ajmalina y guía de práctica clínica sobre fibrilación auricular 2010 de la ESC

To the Editor,

We read with great interest the editorial in your journal entitled “New Evidence, New Controversies: a Critical Review of the European Society of Cardiology 2010 Clinical Practice Guidelines on Atrial Fibrillation.” In this editorial, controversial aspects of the 2010 guidelines are discussed. Of note, obviously, are the new embolic and hemorrhagic risk scales and criteria, with their corresponding therapeutic recommendations and reflections on strategies for rhythm and frequency control. However, no mention was made of the ajmaline or flecainide challenge test as a means of detecting Brugada syndrome (BS) either in the document itself or in the editorial comment.

The association between BS and atrial fibrillation (AF) is well known and represents an added problem in the management of patients with AF. In our hospital, AF was the first clinical manifestation in 35 of 613 patients with BS (in press). Of these, 11 cases were detected after starting treatment with group IC antiarrhythmic drugs and 2 patients arrived at the hospital with an acute arrhythmic event. The first survived sudden death 1 month after starting propafenone. The second case was a 22-year-old woman who came to the emergency room for a fibrillation episode and atrial flutter. In accordance with the guidelines, treatment was initiated with flecainide. Minutes after administration, the patient experienced a type 1 electrocardiographic pattern indicative of BS and subsequent degeneration into ventricular fibrillation.

These are not isolated cases, but rather have been reported numerous times in the literature. Pappone et al. analyzed the presence of latent BS after administration of type IC drugs to 356 individuals attended in the emergency room with new-onset AF and found 11 cases of BS. Three of these had ventricular tachycardia/ventricular fibrillation during follow-up. Juntila et al. reviewed cases in which the typical Brugada pattern was observed in the electrocardiogram after a range of trigger events (fever, propofol, etc.). Of the 9 patients in whom BS was detected after administration of sodium-channel blockers, 1 experienced sudden cardiac death and another ventricular tachycardia.

We therefore believe that this challenge test is of prime importance and should be taken into account in young patients with “isolated” AF and in those with a history of syncope and/or a family history of sudden death despite having normal baseline electrocardiogram because, as is well known, electrocardiograms can undergo changes over time. Although it is true that BS appears in only a small percentage of all patients with AF, it is essential to identify these patients because they are managed differently, given the contraindication of certain drugs, including sodium-channel blockers widely used to treat AF.

We therefore propose a special mention in the current guidelines to enable subsequent application in daily clinical practice and to avoid fatalities.

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**Ajmaline Test and ESC 2010 Clinical Practice Guidelines on Atrial Fibrillation. Response**

To the Editor,

We read with interest the comments on our recent review of the European Society of Cardiology (ESC) 2010 guidelines for the management of atrial fibrillation (AF). As the authors of the letter point out, there have been several reports of Brugada syndrome being unmasked when class IC antiarrhythmic drugs (flecainide, propafenone) are used to treat AF. However, the proportion of cases detected in such circumstances is not high. From a total of 613 patients, we identified 11 patients with Brugada syndrome (1.8% of all cases with AF), while Pappone et al. diagnosed the syndrome in 11 (3%) of 356 cases of new-onset AF. That is presumably why the ESC guidelines did not make any recommendations on this aspect of the condition, and we why we did not do so in our article. However, we agree with the authors that a diagnosis of Brugada syndrome leads to a need for specific treatment alternatives, and that clinicians should be alert to the emergence of typical electrocardiographic changes when treating AF patients with class IC antiarrhythmic drugs.

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