Letters to the Editor


Maintainance of sinus rhythm after electrical cardioversion of persistent atrial fibrillation

I read with interest the article by Plewan et al. [1] that compared head-to-head the efficacy of sotalol vs bisoprolol in the maintenance of sinus rhythm after electrical cardioversion of persistent atrial fibrillation. Their study failed to show any benefit of sotalol over bisoprolol during a follow-up period of 1 year, and showed more adverse effects with sotalol, principally due to proarrhythmia. Several considerations have to be made, however, before banning sotalol indefinitely from our therapeutic arsenal against atrial fibrillation.

The first point concerns efficacy. The dose of sotalol was 80 mg b.i.d., which has been shown to be no more effective than placebo for maintaining sinus rhythm in a randomized placebo-controlled dose-response study [2]. In that study, the optimal dose of sotalol was suggested to be 120 mg b.i.d. Therefore the dose of sotalol may have been too low to show any benefit over bisoprolol.

The second issue concerns proarrhythmia with sotalol, with an incidence of torsades de pointes of 3.1%. The majority of patients had structural heart disease, with a mean left ventricular ejection fraction of 40%. About a third of the patients had coronary artery disease. In a trial using d-sotalol in patients with a left ventricular ejection fraction of 40% or less and with either a recent myocardial infarction or symptomatic heart failure with a remote myocardial infarction, administration of d-sotalol was associated with increased mortality as compared to placebo, presumably due to arrhythmias [3]. Therefore proarrhythmia in the study may have been reduced by excluding patients with a depressed left ventricular function and ischaemic heart disease.

A reply

We are very grateful for the interest in our study [1] and the discussion following publication.

We do not agree with Dr Burri’s suggestion that an 80 mg dose of sotalol b.i.d. would be too low to show any benefit over a pure beta-blocker for maintenance of sinus rhythm after cardioversion of atrial fibrillation. In our trial we demonstrated a significant prolongation of QT and QTc intervals (Table 2; Fig. 2) as a variable of the class-III properties of sotalol at a daily dosage of 160 mg [1]. Furthermore, another placebo-controlled study showed a significant benefit for maintaining sinus rhythm after paroxysmal supraventricular arrhythmias, starting at 80 mg b.i.d. [2]. At a first glance the trial of Benditt et al. [3] seems to be in contrast to these findings. But there was a dose reduction in 20% of all patients in this study to a single 80, 120 and 160 mg daily dosage of sotalol. Taking this fact into account, sotalol was efficient at a mean daily dosage of approximately 200 mg. With respect to this mean, the sotalol dosage given to the patients of Benditt et al. might be in accordance with our results.

We agree with Dr Burri, that an underlying structural heart disease with depressed left ventricular function increases the risk of proarrhythmia under sotalol. We found an incidence for torsade de pointes tachycardia of 3.1%, in accordance with other publications, with a range from 2–5% depending on the severity of concomitant heart disease [4]. Astonishingly both patients in our trial with torsade de pointes tachycardia suffered from arterial hypertension, but had normal left ventricular function. Therefore, excluding ‘high-risk’ patients with proarrrhythmia would not have reduced the incidence of adverse effects under sotalol in our study.

Finally our findings are supported by a study of Steeds et al. [5] who found no difference in control of paroxysmal atrial fibrillation between a dosage of sotalol 160 mg and atenolol 50 mg, although there was improvement compared to baseline prior to treatment with either substance.

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References