report revision. In the HG registry, the median time requested for each revision ranged from 1.0 to 2.2 min, depending on device type and event actionability (unpublished data). That is significantly shorter than the time measured in the Evolvo trial (7 min), commented on in the letter by Locati, in which another proprietary system had been used. Time for transmission revision in the Evolvo trial looks particularly long, if we consider that in the pilot study testing the same technology, in spite of centre inexperience, the mean time for revision was 5 ± 2 min, and it decreased from 6 ± 2 to 4 ± 1 min, after the first two sessions in each centre. In the HG model, continuous updating and filtering of remote alerts allowed nurses and physicians to focus their attention on critical cases and to disregard insignificant reports. In fact, it is a common experience that the majority of daily transmissions show no alerts. This aspect may explain why the manpower measured in the HG registry is four times shorter than that we had measured in our pilot study 5 years earlier, when little experience, if any, was available about the right strategy for remote monitoring. Similar findings have been reported in the MoniC (Model Project Monitor Centre) prospective multicentre study in which a centralized Home Monitoring model, consisting of one monitor centre and nine satellite clinics, was tested. The investigators found a higher daily workload in MoniC than in HG, probably due to the activation of nearly all types of event notifications, the use of more complex decision-tree algorithms, and the multicentric approach in particular with no access to the patients’ clinical charts. This last point drives the attention on the role of the organizational model applied in the HG registry, which in our opinion is pivotal to explain the very low manpower we measured. This model is based on a tight interaction between the roles of an expert responsible nurse and a responsible physician. The former controls Home Monitoring data flow on a daily basis, filtering critical events or unclear interpretations to the physician. In the HG registry, 85% of reports were directly acknowledged by the nurse and only 15% were submitted to the physician. In the HG registry, such as the one already ongoing, could be more useful. As the rate of clinical events is low, this subgroup will need to be medicated lifelong; thus, a possible medical strategy could be to start with low doses to avoid side-effects and only increase dosage (or change to an ICD) based on clinical evolution. For gaining insight into the safety and efficacy of quinidine in asymptomatic BrS, a registry, such as the one already ongoing, would certainly be useful to increase our understanding of the real place of quinidine in (asymptomatic) BrS.

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Adverse effects of long-term therapeutic doses of quinidine in asymptomatic Brugada patients: should low doses be used first?

Pharmacological therapy is controversial in Brugada syndrome (BrS), but quinidine, an old anti-arrhythmic drug initially proposed by Belhassen and coworkers in this indication, is gaining support from different groups. Bouzeman et al. 1 nicely present in this Journal a large series of asymptomatic subjects with BrS who received hydroquinidine and had an electro-physiology study on treatment. The study confirms the low rate of ventricular arrhythmias (VAs) or clinical events (syncope) in this subgroup of BrS. A finding that we would like to highlight is the high rate of adverse events that occur under therapeutic doses of hydroquinidine (600–1200 mg/day). The authors report three patients who developed major adverse events (6.8%) and eight mild-to-moderate (18.1%), for a total of 24.9%. This included a pro-arrhythmic effect in one patient; two other patients had an implantable cardioverter-defibrillator (ICD) implanted due to major intolerance. Recently, we described a series of symptomatic patients in whom quinidine was used for secondary prevention of VA and made an extensive review of the literature. In this analysis, low doses of quinidine (<600 mg) were well tolerated as only 1 out of 20 subjects presented side-effects (5%) with an 85% effectiveness in preventing the recurrence of VA. Our data supports the concept that doses of quinidine as low as 200 mg/day (of quinidine sulfate) may be an option when adverse events occur with high doses. We emphasize this because it is probably in asymptomatic subjects that low doses of quinidine could be more useful. As the rate of clinical events is low, this subgroup will need to be medicated lifelong; thus, a possible medical strategy could be to start with low doses to avoid side-effects and only increase dosage (or change to an ICD) based on clinical evolution. For gaining insight into the safety and efficacy of quinidine in asymptomatic BrS, a registry, such as the one already ongoing, would certainly be useful to increase our understanding of the real place of quinidine in (asymptomatic) BrS.

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References


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Letters to the Editor

Side effects could certainly be decreased with lower dose of quinidine in asymptomatic Brugada patients, but what about efficacy? Author reply

We appreciate the letter from Márquez et al. regarding our article concerning the interest of hydroquinidine (HQ) in preventing ventricular