Asians. It may suggest that PPR would be 1 of numerous risk factors of atherothrombosis and that the role of it may not explain the whole clinical events after percutaneous coronary intervention.

Previous studies reported that high PPR is associated with poor clinical outcomes in patients with coronary artery disease (3,4). However, it is not clear that tailored dosing or choice of antiplatelet agents based on platelet function test can improve the prognosis of patients with high PPR. The GRAVITAS (Gauging Responsiveness With A VerifyNow Assay—Impact on Thrombosis and Safety) trial could not show the benefit of high-dose clopidogrel therapy in patients with high PPR (5). The TRIGGER-PCI (Testing Platelet Reactivity in Patients Undergoing Elective Stent Placement on Clopidogrel to Guide Alternative Therapy With Prasugrel) study was stopped early after an interim analysis revealed a primary event rate too low to allow for any meaningful comparison between clopidogrel and prasugrel (6). The results of 2 major trials make us question whether PPR would be a modifiable factor of thrombosis or simply an intrinsic property reflecting one’s thrombotic status. Further studies are warranted to investigate these unsolved problems, especially regarding the ethnic difference of response to antiplatelet regimen and the necessities of personalized antiplatelet therapies.

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REFERENCES


Vasovagal Syncope as a Cause of Syncope in Long-QT Syndrome

The report by Liu et al. (1) containing data from the International Long QT Syndrome Registry regarding the risk factors for recurrent syncope and subsequent fatal or near fatal events in children and adolescents with long-QT syndrome (LQTS) who present for evaluation after episodes of syncope is of great interest. The registry includes all sorts of syncopal episodes, and in their discussion, Liu et al. aptly emphasize that risk stratification in these patients requires careful examination to distinguish whether a syncopal episode stemmed from LQTS-associated dysrhythmia or whether it was simply a vasovagal mediated episode occurring in an LQTS host.

In the general population, a vasovagal faint is by far the most frequent cause of syncope (2). It refers to a syncopal episode induced by a fall in blood pressure and cerebral hypoperfusion due to reflex response upon various triggering factors (standing up, prolonged orthostatic stress, reaction to blood taking, and so on). The underlying mechanism is a loss of vasoconstrictor tone associated with relative or absolute bradycardia. Although some patients present with clear and prolonged premonitory symptoms, acute onset has also been reported (3). In general, the overall prognosis in patients with vasovagal syncope is excellent (4).

The lifetime cumulative incidence of ≥1 syncopal episode in teenagers in the general population is high, with about 40% by age 21 years (2,5). This is almost identical to the cumulative prevalence of 41% in children and adolescents with prolonged corrected QT intervals or young carriers of LQTS-causing mutations in the study by Liu et al. (1). There is no reason to assume that vasovagal syncope has a lower prevalence in patients with LQTS than in the general population. Thus, whereas syncope in the setting of LQTS is definitively a red flag, given the risk for fatal arrhythmic events (6), the epidemiological data on the frequency of vasovagal syncope in the general population suggest that the vast majority of syncopal episodes in patients with LQTS are caused by vasovagal syncope.

The obvious challenge for clinicians is to identify the patients who are at risk. With an additional positive family history of syncope or sudden death (3), the identification of high-risk patients would possibly be more accurate.

Thus, although the risk for an aborted cardiac arrest or LQTS-related sudden cardiac death rises with the occurrence of syncopal episodes, syncope does not equate to torsades de pointes, especially not in the young, and careful history taking remains a cornerstone of diagnosis and treatment in these patients.

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We agree with Dr. Olde Nordkamp and colleagues that careful history taking remains crucial for appropriate risk stratification and management of patients with long-QT syndrome (LQTS) (as well as other subjects with unexplained syncope). As Dr. Olde Nordkamp and colleagues note, this point was also emphasized in our report in our discussion of the clinical implications of our findings (1). However, we think that the comment regarding the fact that the vast majority of syncopal episodes in patients with LQTS are associated with a vasovagal mechanism may mislead readers to underestimate the clinical importance of the development of syncope in patients with LQTS. In this regard, it is important to note that the sources Dr. Olde Nordkamp and colleagues cite regarding a high frequency (41%) of syncopal episodes in general population (obtained from questionnaire data in a relatively small sample of medical students) may be an overestimation, as data from other studies suggest that the frequency of syncope before age 18 years is much lower (approximately 15%) (2,3). Furthermore, estimation of the frequency of vasovagal syncope in nonselected patients is difficult, because in most studies, these events are not thoroughly investigated, and this information may not take into account the true penetrance of LQTS (or other arrhythmogenic disorders) in the general population (4).

Information regarding the circumstances of syncopal events in patients with LQTS in the registry was obtained prospectively and carefully validated by study specialists. Our data consistently show that among patients with LQTS, syncope is the most powerful predictor of subsequent fatal or near-fatal events. Importantly, the present study shows that children and adolescents with LQTS who experienced syncope had a pronounced 7- to 15-fold increase in the risk for subsequent aborted cardiac arrest or sudden cardiac death (1). These findings contrast those in the general population, in which vasovagal events were shown to be associated with a benign clinical course (3), and strongly suggest that the majority of syncopal episodes in patients with LQTS should be considered arrhythmic in nature until proven otherwise.

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Surgical Ablation for Atrial Fibrillation

Not All Energy Sources Are Created Equal

We read the recent report by Onorati et al. (1) with great interest. We agree with the investigators that the effect of surgical lesion pattern has been poorly investigated and had hoped that this report would clarify the success of different lesion patterns in patients undergoing concomitant surgery for atrial fibrillation. Unfortunately, we believe that the report has several inherent flaws that make the results inconclusive.

Most important, the left atrial group combined patients whose lesions were created using different types of energy sources. Myriad new ablation technologies have been introduced in the past decade, and the ability of each to create transmural linear lesions varies. Bipolar radiofrequency (RF) ablation appears more reliable in creating transmural lesions than monopolar RF ablation (2). The most likely reason that a change from monopolar RF to bipolar RF ablation occurred after December 2003 was the improvement in technology and thus outcomes. Fourteen of 32 patients undergoing the limited approach had lesions created using monopolar RF ablation. The “extensive” approach began in August 2004, after bipolar RF ablation was introduced. This suggests that the outcomes were inherently biased toward the “extensive group,” the group with the better energy source. Even in the 18 patients who underwent left atrial with bipolar RF ablation, the freedom from atrial fibrillation in this series was 53%. We recently reported our results in 175 patients using a left atrial-only approach, with freedom from atrial fibrillation of 81% (3). It is difficult to account for this disparity in results.

We congratulate Onorati et al. (1) for attempting to address a critical question: Does a more extended lesion set improve sinus