Letters to the editor

Sir,—We offer an alternative interpretation of the evidence presently available regarding the use of medical treatment for the management of neurally mediated syncope. Hargreaves et al suggested that “on the present evidence it seems reasonable to start treatment with β blockers or disopyramide and assess the response by repeating the tilt test.”1 This statement is supported by several uncontrolled trials.2 However, the few controlled trials reported to date do not seem to support such a view.1-4 Our group reported no benefit when intravenous disopyramide was compared with placebo. Furthermore, when patients were retested after one week of placebo given by mouth, 82% had a negative head-up tilt.5 This finding strongly argues against the use of head-up tilt as an indicator of therapeutic efficacy. Similar results have recently been reported in a etilefrine-placebo crossover controlled trial.7 In this study, 50% of patients had a negative repeat tilt on placebo compared with 43% receiving etilefrine. Likewise, evidence regarding the efficacy of β blockers for the prevention of neurally mediated syncope has been obtained from small uncontrolled trials.2-4 The only two randomised trials addressing this issue were unable to prove any beneficial effect with atenolol.6 Besides, preliminary data from different laboratories suggest that treatment with β blockers may be potentially prophylactic.1-10 However, the major limitation of these studies is the lack of a placebo controlled group. These preliminary data indicate that the routine recommendation of pharmacological treatment of patients with neurally mediated syncope and a positive head-up tilt test should be viewed with caution. Similarly, recent reports of the natural history of patients with neurally mediated syncope diagnosed by head-up tilt indicate that syncope recurs in 10-30%.1-14 The only variable that identifies patients at risk of recurrence of syncope is the frequency of syncope episodes.14 Several questions regarding the management of patients with neurally mediated syncope remain unanswered. Do all patients with unexplained syncope and a positive head-up tilt test need pharmacological treatment? If so, how long should treatment be maintained? Should repeated head-up tilt testing be used as an indicator of therapeutic efficacy? Most patients with neurally mediated syncope are young, physically active, and generally have no evidence of structural heart disease. The side effects caused by β blockers and disopyramide may outweigh the benefit of treatment in this group. Furthermore, given the low recurrence rate in previous studies previously unexplained syncope and a positive head-up tilt test, many patients would need to be treated in order to prevent one syncope episode. We believe this approach is not cost effective, may be hazardous, and should be discouraged. Interestingly, two preliminary reports have documented a decreased incidence in the frequency of syncopal episodes after neurally mediated syncope has been diagnosed.13-14 Patients with recurrent unexplained syncope have generally undergone multiple consultations and a wide variety of expensive diagnostic tests that have been unable to establish the cause of the episode. This creates great frustration for both patient and physician. Inducing a vasodilator response during head-up tilt that clearly resembles the clinical presentation reassures the patient with a definitive diagnosis. Early recognition of prodromal symptoms provoked during head-up tilt should alert the patient of impending syncope and may influence the patient’s awareness of neurally mediated syncope. This factor may be related to the significant decrease in frequency of syncopal episodes seen in patients with a positive head-up tilt who have not been pharmacologically treated.

We recommend that for most patients with neurally mediated syncope, reassurance, increased salt intake, and close follow up may be a reasonable approach. Only patients with frequent syncopal episodes (> 6 episodes/year) and those who have been injured during a faint should be treated. Perhaps patients who do not have prodromal symptoms may be at a higher risk of recurrence and require treatment. The efficacy of therapy should be evaluated by long-term follow-up and should not rely solely on the outcome of a repeated head-up tilt. Large multicentre controlled trials are needed to define the risks and benefits of treatment in patients with neurally mediated syncope. Furthermore, we should provide this information and help to establish treatment guidelines.

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This letter was shown to the authors, who reply as follows:

Sir,—We thank Dr Morillo and colleagues for their interest and well written letter. We find ourselves in broad agreement with their comments and accept that most people with infrequent episodes of syncope do not require pharmacological intervention or pacing. As we stated in the editorial, those people who avoid prolonged standing and lie down at the onset of premonitory symptoms may not need specific treatment if they have infrequent syncope and adequate warning symptoms. Indeed as Morillo et al point out, the incidence of recurrent syncope after diagnosis may be ≤ 30%.

Some patients are troubled by frequent episodes. Because the best management protocol for these people has not been defined, properly constructed large multicentre placebo controlled trials are needed. Nevertheless on the basis of present evidence, we believe that drug treatment should be reserved for those frequent episodes and that efficacy should be established by clinical follow up as well as repeat head-up tilt testing.

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