Neurocardiogenic, vasovagal syncope

See page 1745 for the article to which this Editorial refers

Dual chamber pacing with rate-drop response is one of the latest therapeutic innovations. It significantly reduces the likelihood of syncope but not the occurrence of presyncope[1]. We concur with Benditt that it seems prudent to be cautious about the present status of cardiac pacing in vasovagal syncope[2]. One of the most extensive overviews on pharmacological treatment is by Atiga et al.[3]. The agents most likely to be effective include beta-blockers, fludrocortisone, and alpha-adrenergic agonists. Education on avoiding trigger situations, increasing salt and fluid intake, regular meals, sitting or lying down quickly if warning symptoms occur, are other general recommendations[3].

For unproved treatments, it is a standard requirement that a properly designed randomized clinical trial be performed[4]. Unfortunately, not every question in medicine can be solved by this approach. This applies to the syndrome of neurocardiogenic syncope. In this field, large controlled studies are difficult to undertake due to the variable frequency of spontaneous symptoms and apparent long periods of remission. This contrasts with the urgency of decision making after frequent or dramatic syncopal attacks. The clinical spectrum varies widely between ‘malignant’ and ‘benign’[5]. In their paper in this issue Santini et al. suggest coupling the best available cardiac pacing mode with the possibility of delivering an active drug[6]. New sophisticated devices are supposed to have: (i) a diagnostic element; (ii) a patient activated intervention algorithm; (iii) an automatic drug-delivery element; (iv) a dual chamber pacemaker; (v) a data-storage unit. In this respect, atropine proved to be effective in the prevention of the cardio-inhibitory type of syncope. For the vasodepressor type, there was a statistically non-significant trend in favour of atropine. So, even when the ‘pharmaco-pacemaker’ is available, one drug would probably not suffice in every incident in every patient. Moreover, the type of syncope will vary from one patient to another and from one moment to another. So, more than one ‘ideal’ drug would have to be available in the automatic pharmacopacemaker, delivering medication after the appropriate selection of the most adequate drug. This is a very complex situation.

So, let us return to reality. Can reality lead to a simple solution? For many patients it has! Morillo et al. were probably the first to recognize the therapeutic value of serial tilting[7]. Neurocardiogenic syncope is the result of an abnormal autonomic reflex. It is also the outcome of an imbalance between orthostatic tolerance and gravitational stress. The baroreceptor response may be conditioned by serial tilt table tests. This has also been our experience. When heavily symptomatic patients were subject to serial tilt testing, the first negative tilt test result was obtained at the second session in 18 cases, at the third in nine, at the fourth in eight, at the fifth in three, at the sixth in three, at the eighth in one (mean 3·24, median 3, SD 1·46). To retain a long-lasting effect, tilt training or standing training had to be continued at home on a daily basis: two sessions of 30 min upright standing against a vertical wall[8,9]. This remedy can, of course, be combined easily with the general recommendations as mentioned before.

We admit today that the optimal treatment for patients with vasovagal syncope remains uncertain. Neurocardiogenic syncope is not a disease but a functional disorder with a spurious and intermittent nature. As Benditt pertinently summarizes, we must be cognizant of the fact that, while vasovagal faints are most often ‘benign’, recurrent vasovagal symptoms may cause unwanted lifestyle changes (e.g. loss of independence, excessive health insurance premiums, restriction of driving privilege) and predispose them to injury or accidents. Additionally, prevention of even infrequent faints may be essential for certain occupations (e.g. airline pilots, commercial vehicle operators, critical-care medical personnel) or sporting activities (e.g. mountaineers, skiers)[2].

To restore a maximum of physical integrity in the syncopal patients, a stepwise approach seems to be wise: (i) general counselling; (ii) tilt training; (iii) medication; (iv) pacing; (v) automatic drug (e.g. atropine etc.) delivery, combined with pacing.

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New approach to the diagnosis of myocardial bridging by intracoronary ultrasound and Doppler

Myocardial bridging is anatomically defined by the intramyocardial course of portions of the coronary arteries, mainly the midportion of the left anterior descending coronary artery[1]. Its typical angiographic presentation is the systolic ‘milking’ effect due to transient myocardial vessel compression[2]. Since Portmann and Iwig’s[3] description of angiographically visualized systolic narrowing of the left anterior descending coronary artery, many subsequent reports have described the milking effect of this artery. A large discrepancy exists between pathological series, in which the incidence has varied from 15% to 85%/4, and angiographic series, in which it is reported as being between 0-51% and 2-5%/5. There is also considerable controversy regarding the clinical, haemodynamic and prognostic significance of myocardial muscle bridges. Although various reports are available describing myocardial ischaemia, myocardial infarction[6], conduction disturbances[7] and sudden death[8] in association with this anatomical variation and otherwise normal coronary arteries, myocardial bridges are generally considered harmless clinical anomalies.

Ge et al. were pioneers in the potential clinical application of intravascular ultrasound to detect myocardial bridging[9], and provided unique information concerning wall morphology in this condition. Intravascular ultrasound is a unique technique that visualizes the coronary wall and therefore enables changes in its shape and structure with particular reference to coronary segments with myocardial bridging, to be analysed. This analysis demonstrated that vessel compression within the bridge is not a purely systolic event, but persists throughout large periods of diastole.

Ge et al.[10] present a new description of morphological signs characteristic of myocardial bridging by intravascular ultrasound. For the first time, they describe a specific morphological sign, a ‘half-moon’ like echolucent area surrounding the bridge segment. The presence of this sign seems to be highly specific for the existence of myocardial bridging, as it can only be found in the bridge segment, and not in the proximal and distal segments and other coronary arteries. It is important to note that when the specific half moon phenomenon is demonstrated by intravascular ultrasound, the milking effect can be provoked by intracoronary administration of nitroglycerin, although the milking effect was not initially revealed by angiography.

In the present study Ge et al.[10] nicely highlight the potential use of intravascular ultrasound in the assessment of myocardial bridge and its clinical