cope. A typical history makes the diagnosis, and no further evaluations are needed excepted a clinical examination, an electrocardiogram, and supine and standing blood pressure measurements.[2] Study of vasovagal syncope should include patients with no doubt about the diagnosis. A positive outcome to a tilt test coming with a typical history allows the clinician to confirm the diagnosis and permits inclusion of the patient in a study. The group with a negative outcome to a tilt test, but a typical history of syncope, is certainly not a control group. It is hard to conceive a true control group for comparison with patients with vasovagal symptoms.[2] A clinical examination, and even further investigations performed between episodes, cannot make the difference between a group of normal subjects and the patients.[2] The incidence of unexplained syncope is so large (one in 6) that anybody may experience vasovagal symptoms at least once in a whole life.[3] It is why regular designs for clinical investigations (controlled studies) are probably not appropriate to study vasovagal syncope. However, it is difficult to draw a clear conclusion from uncontrolled studies.

Another difficulty of the studies using the tilt test is the timing of blood withdrawals during the test. Hormone blood levels have slow dynamics compared with reflex controls, and, therefore, hormone blood levels may result from the summation of serial events. Hormone blood levels obtained immediately before syncope probably reflect this very pathologic state, but also includes the remaining amount of hormones released in response to all the previous events: standing duration, shift from supine to upright posture, and stress of previous blood withdrawals. A set of hormone levels obtained right before syncope in a group of patients may be homogenous in terms of hormone response to this state, but would be scattered by the individual differences in standing duration and delay between the position shift (supine to standing) and the blood withdrawal.

Unfortunately, there are no solutions to these problems. They probably explain why our understanding of vasovagal symptoms is so limited. It is why studies like the one of Ermis et al[1] are needed despite their limitations. Such studies allow one to improve in the knowledge and understanding of the vasovagal syncope, and they are a step to better management of the symptoms.

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1. Ermis C, Samniah N, Sakaguchi S, Lurie KG, Pham S, Lu F, Benditt DG. Comparison of catecholamine response during tilt-table-induced vasovagal syncope in patients <35 to those >65 years of age. *Am J Cardiol* 2004;93:225–227.
2. Brignole M, Alboni P, Benditt D, Bergfeldt L, Blanc JJ, Bloch Thomsen PE, van Dijk JG, Fitzpatrick A, Hohnloser S, Janousek J, et al, and the Task Force on Syncope, European Society of Cardiology. Guidelines on management (diagnosis and treatment) of syncope. *Eur Heart J* 2001;22:1256–1306.
3. Shen WK, Gersh BJ. Fainting: approach to management. In: Low PA, ed. Clinical Autonomic Disorders. Philadelphia: Lippincott-Raven, 1997:649–679.

doi:10.1016/j.amjcard.2004.02.077

Correction

In the April 15, 2004, issue of the *AJC*, there is an error in Table 5 of the article, "Comparison of Directional Coronary Atherectomy and Stenting Versus Stenting Alone for the Treatment of De Novo and Restenotic Coronary Artery Narrowing" by Stankovic et al (page 957). In Table 5, "target lesion revascularization" is listed twice. The second line from the bottom should read: "target **vessel** revascularization."

Correction

There is an error in the February 1, 2004, issue of the *AJC*. In the abstract of the article "Comparison of Reperfusion Regimens With or Without Tirofiban in ST-Elevation Acute Myocardial Infarction by Martinez-Rios et al, page 280, the incorrect trifiban bolus is given as "4 μ/kg/30 min." The correct dosage should read **"0.4 μg/kg/30 min."**