Baroreflex Regulation of Sympathetic Nerve Activity in Patients With Vasovagal Syncope

To the Editor:

In their study, “Dysfunctional Baroreflex Regulation of Sympathetic Nerve Activity in Patients With Vasovagal Syncope,” Béchir et al implied a unilateral causal relationship between reduction of arterial baroreflex control of heart rate and increase of muscle sympathetic nerve activity (MSNA) in patients with syncope. The authors concluded that dysfunctional baroreflex regulation of sympathetic activity provides “new insights into the mechanisms of vasovagal syncope.”

We have two comments.

Baroreflex function can be depressed by suprabulbar central influences and also by vagal, somatic, or sympathetic afferents. This experimental evidence has provided strong support for the concept that baroreceptive input should be considered as one of the neural mechanisms underlying cardiovascular regulation and not the only mechanism, as suggested by the Béchir et al study. The observation of enhanced MSNA values in patients with syncope both at rest and during lower body negative pressure is in contrast to the reduced MSNA already reported and raises the possibility of an alternative interpretation of the results. Indeed, the depressed baroreflex function observed in patients with syncope may be the consequence of an increase in central sympathetic drive.

In the Béchir et al study, baroreflex inhibitory modulation of MSNA was inferred by using the alpha index that only assesses arterial baroreflex control of heart rate.

A comprehensive approach to arterial baroreflex modulation of heart rate and MSNA in patients with syncope has been previously published in a study misquoted in the Béchir et al paper. An exhaustive assessment of baroreflex function should consider several aspects besides alpha index, including the definition of the entire sigmoidal baroreflex curve during drug infusion and the evaluation of spontaneous reciprocal changes of heart rate, arterial pressure, central venous pressure, and MSNA during progressive tilt (or lower body negative pressure).

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Response

We thank Dr Furlan and colleagues for their comments concerning our study.

First, there is no doubt that there are many contributing cofactors in the pathophysiology of vasovagal syncope. Thus, a baroreceptor dysfunction is, indeed, only one underlying factor, but probably an important one. Many other studies (even one of the respondents’) have shown such an impairment. Indeed, in our study, we did not suggest impaired baroreceptor sensitivity as the only pathophysiological factor in the genesis of vasovagal syncope.

Second, there are stimulus-dependent and -independent baroreceptor testing methods. We used only a stimulus-independent one, because the infusion of vasodilators may lead to syncope, which was not the aim of our study. Furthermore, many substances can interfere pharmacologically with the endings of the baroneurons in the vasculature. This, in turn, might lead to a change in baroreceptor sensitivity.

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