Cerebral Vasoconstriction in Vasovagal Syncope: Any Link With Symptoms? A Transcranial Doppler Study

To the Editor:

We welcome the confirmation by Lagi et al1 of our finding that progressive hypocapnia occurs during tilt-induced presyncope in patients with recurrent vasovagal syncope (VVS).2 In a larger series of patients and control subjects, we also demonstrated that similar changes occur in normal subjects (with no previous history of syncope) who undergo tilt-induced VVS.2

There is little doubt that cerebral autoregulation fails to preserve cerebral blood flow during presyncope,3 but the conclusion that this is due to cerebral vasoconstriction, paradoxical or not, is controversial and does not reflect the complexity of events that take place before VVS. This conclusion is based predominantly on the consistent, undisputed reports1,2 of a markedly rising pulsatility index before syncope, but such a conclusion ignores at least 3 important points.

(1) Pulsatility index is an unreliable index of cerebrovascular resistance because it does not take systemic blood pressure or cerebrovascular critical closing pressure into account.2,4,5 Changes in pulsatility index during presyncope may even, in fact, be inversely related to changes in cerebrovascular resistance.2

(2) Other, probably more reliable, indices of cerebrovascular resistance, such as classical cerebrovascular resistance (arterial blood pressure/cerebral blood flow velocity) and resistance-area product,2 consistently show declines in cerebrovascular resistance during presyncope, suggesting that cerebral vasodilatation is taking place.2

(3) The relative preservation of systolic cerebral blood flow velocity during presyncope despite markedly impaired diastolic flow has been consistently demonstrated by most groups, including Lagi et al.1,2 Maintenance of systolic flow, especially in the face of precipitously falling systemic blood pressure, is inconsistent with the hypothesis of simple cerebral vasoconstriction and supports the hypothesis of active vasodilatation. The dilemma as to why diastolic flow is impaired if cerebral vasodilatation is taking place has been potentially answered by our demonstration of rising cerebrovascular critical closing pressure during presyncope,2 possibly due to the progressive hypocapnia demonstrated by Lagi and us.1,2

In conclusion, we agree with Lagi et al that hyperventilation-induced hypocapnia forms an important part of the extremely complex pathophysiology of VVS. We believe, however, that hypocapnia contributes to the impairment of cerebral autoregulation during presyncope not through simple cerebral vasoconstriction, but by elevating cerebrovascular critical closing pressure and selectively impairing diastolic cerebral blood flow, thereby counteracting the effects of active cerebral vasodilatation.2

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*Circulation.* 2002;106:e54
doi: 10.1161/01.CIR.000031829.09784.82
*Circulation* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/106/13/e54

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