Effect of Prazosin on Dynamic Cerebral Autoregulation During Acute Hypotension in Healthy Human Subjects

To the Editor:

Cerebral blood flow is excellently autoregulated when mean arterial pressure is between 65 and 140 mm Hg.1 In the study by Ogoh et al,2 although the decrement in mean arterial pressure (MAP) in the 2 groups were similar, the MAP fell below the lower limits of cerebral autoregulation to an average of 57 mm Hg when subjects took prazosin, and this would be sufficient to explain the slower rate of autoregulation in the prazosin group in phase I. However, the authors interpret this observation as indicating that prazosin directly impaired cerebral autoregulation (I would suggest it is the effect of the lower MAP per se rather than the drug). In contrast, when MAP falls suddenly and significantly as happened during the thigh cuff release, the immediate decrease in cerebral blood flow would be minimized by cerebral vasodilation—an effect prazosin (an α-adrenergic receptor blocker) is not known to interfere with. Thus, the interpretation that the rate of dynamic cerebral autoregulation (in phase I) is impaired by prazosin is difficult to explain on the basis of the known actions of prazosin.

The other possibility which might explain the observations made by Ogoh et al (during phase I) is that α-adrenergic blockade of cerebral vessels with prazosin at baseline might have reduced the ability of cerebral vessels to dilate further (as is required to maintain cerebral blood flow in response to acute hypotension). If this were true, one would expect differences in baseline cerebral blood flow (or middle cerebral artery flow velocity) between the 2 groups but the data do not support this possibility.

On the other hand, vasodilators like angiotensin-converting enzyme inhibitors are known to directly interfere with sympathetically mediated vasoconstrictive cerebral autoregulatory responses when MAP is raised beyond the upper limits of cerebral autoregulation;1 and the vasodilatory effects of captopril have been reported to reset lower limits of cerebral autoregulation to lower values in normotensive and hypertensive rats3 as well as human subjects.4

Disclosures

None.

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Stroke. 2008;39:e167; originally published online October 2, 2008;
doi: 10.1161/STROKEAHA.108.529859
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/39/11/e167

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