
To the Editor:

We read with great interest the article by Ryoo et al.1 We concur with the authors that branch occlusive disease is an important mechanism of stroke typically attributed to small artery disease but pathologically closer to large artery disease. Brain arteries are unique in several aspects compared with systemic arteries because of their histology, the low-resistance flow pattern they carry, the presence of collateral flow through the Circle of Willis, and the ability of the brain to autoregulate the blood flow. All these features may affect the flow pattern and compensatory mechanisms of brain large arteries. Because of this uniqueness, it should not be assumed that brain arteries have similar responses to systemic arteries, and some of these assumed behaviors may require replication based on data from brain arteries. In this context, we would like to point out that the measurement used to define remodeling in the study by Ryoo et al1 is problematic. In this work, as well as in the cited references in the introduction, the referent arterial size used to assess whether positive or negative remodeling occurs is either the homonymous contralateral (presumably) unaffected artery or the proximal or distal segment of the affected artery. This approach may have some limitations. First, it does not take into account that the contralateral homonymous arterial diameter is affected by the collateral flow on that side, that is, if there is a large anterior cerebral artery branching off from the same internal carotid artery, the middle cerebral artery would be expected to be smaller.2 Second, it does not take into account the normal arterial taper in size as the artery extends further into the skull, and thus the use of the proximal arterial segment as a referent may falsely lead to an overestimation of positive remodeling. Using the distal poststenotic segment is also problematic as poststenotic dilatation may occur. Furthermore, because atherosclerosis is rarely a focal disease surrounded by healthy contiguous arterial segments, using neighboring segments may inherently lead to error because these segments may also undergo remodeling. Third, arterial size is a major determinant of lumen-to-wall ratio, so not controlling for arterial size may lead to a false impression of positive remodeling.3 Finally, to further the uncertainty about the true presence of positive remodeling induced by atherosclerotic plaque in cerebral arteries as it occurs in coronary arteries, evidence from our group has shown a lack of plaque-induced outward remodeling in brain arteries using the identical methods described in the landmark article on compensatory outward remodeling in coronary arteries by Glagov et al.4,5 We also found evidence that the association between plaque area and remodeling varies by arterial size, with the smallest brain arteries exhibiting shrinkage rather than outward compensatory enlargement.

We think that these methodological considerations should be systematically taken into account in any work that addresses intracranial remodeling. We hope that with the growing interest in brain arterial remodeling, prospective data using more precise methods to capture well the outer adventitial circumference can more definitively confirm whether brain arteries do accommodate plaque build-up, and if they do, to what extent.6 We enthusiastically embrace the challenge of better understanding the natural history of remodeling in brain arteries, particularly in an era where personalized medicine will demand a personalized approach to primary and secondary stroke prevention according to an individual’s phenotypic expression of brain arterial disease.

Disclosures

Dr Gutierrez reports funding from the American Heart Association (#13CRP14800140). The other authors report no conflicts.

Jose Gutierrez, MD, MPH
Mitchell S.V. Elkind, MD, MS
Randolph S. Marshall, MD, MS
Department of Neurology
Columbia University Medical Center
New York, NY

Letter by Gutierrez et al Regarding Article, "Differential Vascular Pathophysiologic Types of Intracranial Atherosclerotic Stroke: A High-Resolution Wall Magnetic Resonance Imaging Study"
Jose Gutierrez, Mitchell S.V. Elkind and Randolph S. Marshall

Stroke. 2015;46:e260; originally published online October 20, 2015;
doi: 10.1161/STROKEAHA.115.011631
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/46/12/e260

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