Letter by Saji and Kimura Regarding Article, “Arterial Stiffness and Cerebral Small Vessel Disease: The Rotterdam Scan Study”

To the Editor:

We read with great interest the article by Mariëlle et al that was recently published in Stroke. In their population-based study that included 1460 participants, increased arterial stiffness indicated by aortic pulse wave velocity (PWV), a conventional measurement of arterial stiffness, was associated with a larger volume of white matter lesions. Furthermore, lacunar infarcts and deep or infratentorial microbleeds were associated with a higher aortic PWV in persons with uncontrolled hypertension.

We applaud their results, because we also showed an independent association between cerebral small vessel disease and increased arterial stiffness indicated by brachial-ankle PWV, a convenient measurement of arterial stiffness. In our studies, both asymptomatic lacunar infarcts and white matter disease were independently associated with a high brachial-ankle PWV. Although Mariëlle et al did not show an independent association between lacunar infarcts and increased arterial stiffness, a previous study using aortic PWV showed this association. Moreover, the cardioankle vascular index, a measure of arterial stiffness independent of blood pressure similar to brachial-ankle PWV, was associated with asymptomatic lacunar infarcts. Hence, the association between lacunar infarcts and increased arterial stiffness has been validated by multiple methods to assess arterial stiffness.

In contrast, there was no significant association between the cardioankle vascular index and white matter disease in multivariate analysis in our previous study. Because cardioankle vascular index is calculated using the stiffness parameter, which reflects local stiffness of a blood vessel, vessel stiffness per se, may not be associated with white matter disease. These findings indicate that highly pulsatile pressure and flow in the cerebral small vessels could contribute to more progressive white matter disease than lacunar infarcts.

As for microbleeds, Henskens et al did not show an independent association between microbleeds and increased arterial stiffness. This result is opposite to that of the recent report by Mariëlle et al. This discrepancy may depend on a difference in the definition of microbleeds. Mariëlle et al classified the location of microbleeds as lobar, deep, or infratentorial. Conversely, Henskens et al did not classify microbleeds by location. Because lobar microbleeds are thought to be associated with amyloid angiopathy, this mixture could have affected the results of Henskens et al.

Although small numbers of hypertensive subjects were enrolled in the previous studies, these independent associations between cerebral small vessel disease and arterial stiffness are important, because cerebral small vessel disease is still a major risk for stroke.

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Disclosures
None.

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