To the Editor: In reference to a recent paper “Is Pulse Pressure an Independent Risk Factor for Incident Stroke, Reasons for Geographic and Racial Differences in Stroke” by Glasser et al. in the American Journal of Hypertension,1 we wish to call your attention to past publications from the Framingham Heart Study that would help to further clarify the value of pulse pressure (PP) in diagnosing cardiovascular disease (CVD) events in general, and incident stroke in particular. Importantly, unlike Framingham, which examined Caucasians almost exclusively, the REGARDS trial recruited both African Americans and Caucasians in the United States Stroke Belt; however, the REGARDS trial investigators concluded that their findings were independent of racial differences.

First, consider 2 elderly persons with the same elevated PP: person 1 has a blood pressure (BP) of 165/90 mm Hg, a PP of 75 mm Hg, and an elevated mean arterial pressure (MAP) of 115 mm Hg. Person 2 has a BP of 140/65 mm Hg with the same PP of 75 mm Hg as with person 1, and a low MAP of 90 mm Hg. Indeed, the REGARDS authors did a model with PP and MAP together and both components predicted risk. Placing too much emphasis on systolic BP (SBP) or PP alone does not clarify which physiologic component of BP is contributing to risk and therefore what approach should be taken to reduce risk, i.e., reducing MAP in person 1 and reducing arterial stiffness in person 2.

Thus, as shown in our 1999 Framingham publication,2 in middle-aged and older participants, coronary heart disease (CHD) risk increased with lower diastolic BP (DBP) at any level of SBP ≥ 120 mm Hg, suggesting that higher PP was an important component of risk.

Next, as shown in our 2009 Framingham publication,3 PP as a single component of CVD risk (combined CHD, heart failure, and stroke risk) was inferior to the 2-component model of elevated SBP together with low DBP. By placing both SBP and DBP in the same model, one could measure both arterial resistance and stiffness—the 2 hemodynamic components of hypertensive CVD risk; a single BP component of CVD risk—SBP, DBP, PP, or MAP—could not do that!

Finally, as illustrated in our 2015 Framingham publication,4 the combination of (i) elevated SBP and DBP < 90 mm Hg that defines isolated systolic hypertension and (2) low DBP < 70 (vs. DBP 70–89 mm Hg) that defines low perfusion pressure, predicted increased CVD event risk collectively and increased CHD, heart failure, and stroke risk, individually. Note that Domanski et al., reference 24 in the REGARDS paper, strongly confirm the value of PP in predicting stroke risk in direct contradiction to the conclusion of REGARDS authors. Our hypothesis is that stiff central arteries (wide PP) and low perfusion pressure (low MAP) in this older age population not only contribute to ischemic heart disease, but also to heart failure and stroke, suggesting a generalized cardiovascular low perfusion state beyond the coronary arteries.

DISCLOSURE
The author declared no conflict of interest.

REFERENCES