Association Between Pulse Pressure Values During the Acute Stroke Stage and Stroke Outcome

To the Editor:

We read with great interest the recent Research Report by Aslanyan et al.1 The authors reported that elevated weighted average pulse pressure (PP) during the first 60 hours of ischemic stroke was independently associated with poor outcome assessed by mortality, Barthel index, National Institutes of Health Stroke Score and modified Rankin Scale score. Elevated baseline PP was associated with Barthel Index and Rankin score but not with mortality. Weighted average PP was the only blood pressure (BP) component to be consistently associated with all outcome measures.

We have also evaluated the prognostic value of the different BP components in 198 patients with acute stroke (146 cases with cerebral infarction and 42 cases with intracerebral hemorrhage) by means of 24-hour BP-monitoring.2 Our results indicated an independent association between increasing 24-hour PP levels and 1-year mortality after correcting for stroke risk factors, stroke subtypes, clinical (stroke severity and level of consciousness) and radiological characteristics (brain edema, mass effect and hemorrhagic transformation). Higher PP levels on hospital admission were related to an increased risk of 1-year mortality on univariate analysis, but in the multivariate Cox-regression model this association did not retain its statistical significance. This finding underlines the superiority of 24-hour BP variables over admission BP measurements in predicting stroke outcome. It is in keeping with the results of Aslanyan et al and other investigators, which indicate that variables describing BP course during the acute stroke period, such as 24-hour,2,3,4 beat-to-beat5 and weighted average6 BP recordings correlate more strongly and independently with stroke outcome.

On the other hand, other studies failed to document any association between PP levels in acute stroke and early3 or late outcome.4 Since, antihypertensive medication have been shown to have a differential effect on conduit vessel stiffness and to selectively alter the different BP components,6–8 we believe that the prognostic impact of PP levels at baseline and especially during the first hours of ictus on stroke outcome needs further clarification. An increasing body of evidence suggests that raised BP levels following acute stroke may not be a benign phenomenon and are associated with adverse prognosis.9 However, before embarking in a large random-ized, placebo-controlled trial the question of whether the lowering of the pulsatile, the steady or both BP components in acute stroke might improve outcome remains to be answered.

Konstantinos N. Vemmos, MD
Acute Stroke Unit
Department of Clinical Therapeutics
University of Athens
Athens, Greece

Georgios Tsigougli, MD
Konstantinos Spengos, MD
Department of Neurology
University of Athens
Athens, Greece

analysis in this particular study to suggest that stroke increases the incidence of AD or precipitates AD in a vulnerable individual; ie, it is possible that the stroke patients with AD would have developed AD even if the stroke were prevented. Perhaps a study to compare AD patients with and without a history of stroke could look into the question of how the pathology in AD is influenced by the occurrence of a stroke.

Nandini Chakraborty, MD, DNB
Ailsa Hospital
Ayr, United Kingdom


Response:
We thank Dr Chakraborty for her interest in our study and for raising an important issue. We agree that our study does not (and as currently designed can not) address “the question of how the pathology of Alzheimer disease (AD) is influenced by the occurrence of a stroke.” However, previous studies have suggested that ischemia may increase β-amyloid deposition in mouse models of AD and that the severity of cognitive impairment detected prior to death was greater in subjects who had brain infarcts at autopsy as compared with infarct-free subjects with equivalent degrees of Alzheimer pathology. In our study, we observed that the overall risk of developing a dementia (regardless of type; ie, “all dementia”) doubled in subjects who had sustained a stroke as compared with those who had not sustained a stroke; we thus concluded that “prevention of stroke should significantly reduce the risk of all dementia.” We chose not to report the relative risks of developing individual subtypes of dementia because we did not have a sufficient number of cases to permit such analyses. When we compared the relative risk (RR) of developing either AD or mixed dementia in stroke cases and controls, there was no difference between the groups, but the wide confidence intervals do not permit a definite conclusion. The crude RR was 1.0 (CI, 0.6 to 1.9), the RR adjusted for age, sex, and education was 1.0 (CI, 0.6 to 1.7). These compare to the RRs of 2.2 and 2.0 for all dementias. Our data can thus neither confirm nor refute Dr Chakraborty’s suggestion that those who developed AD after a stroke would have done so even in the absence of a stroke. As more of these subjects donate their brains for post-mortem studies we hope to address this interesting question in future studies.

We also thank Dr Chakraborty for detecting a typographical error in Table 1 of our article. Both the table and the text correctly state the actual number of subjects (cases and controls) with at least one apolipoprotein E (ApoE) e4 allele as 22/127 (17%) subjects with a stroke and 153/694 (22%) of subjects without a stroke. The equivalent percentage was incorrectly stated in Table 1 (although it was correctly noted in the text).

We acknowledge the absence of baseline covariate data in some subjects, and have listed in Table 2 the actual denominator used for each subgroup analysis. ApoE genotype data were missing in 37% of subjects; many of these subjects were enrolled (and died) prior to initiation of ApoE genotyping in the Framingham study population. However, we consider it unlikely that the missing data affect the validity of our results because the unadjusted analysis, the analysis adjusting for age, sex, and education alone (data available in 97% of the subjects), and the analysis adjusting for stroke type and presence or absence of second stroke (data available in 97%) were all significant and these relative risks were also ≥2.0.

Sudha Seshadri, MD
Alexa Beiser, PhD
Cristina S. Ivan, MD
Rhoda Au, PhD
Carlos S. Kase, MD
Margaret Kelly-Hayes, RN, EdD
Philip A. Wolf, MD
Boston University School of Medicine
Boston, Mass


Does Preventing Stroke Prevent All Kinds of Dementia?
Nandini Chakraborty

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