Response to Letter by Muscari and Zoli

Response:

We thank Drs Muscari and Zoli for calling attention to their recent study of plasma levels of the N-terminal portion of BNP precursor (NT-proBNP) in elderly subjects.1 They reported that NT-proBNP levels showed a modest inverse correlation (Spearman $\rho=-0.24$ and $-0.22$, respectively) with hematocrit and hemoglobin, respectively (their Table 3). Similarly, multiple linear regression of log-transformed NT-proBNP levels yielded a partial correlation coefficient $r=-0.22$ ($P<0.0001$) in their large sample ($n=713$ subjects).

These results prompted us to conduct a similar analysis of our ALIAS Pilot Trial data, in the 42 subjects in whom plasma BNP levels were measured.2 Spearman rank-order correlation analysis revealed no significant relationship between baseline hematocrit and baseline BNP level ($\rho=-0.272$, $P=0.081$); no relationship between 24-hour (post-ALB treatment) hematocrit and post-ALB BNP level ($\rho=-0.011$, $P=0.95$); and no relationship between the ALB-induced decrease in hematocrit and increase in BNP level at 24 hours after ALB treatment ($\rho=-0.051$, $P=0.76$).

We also conducted a multiple linear regression analysis incorporating both age and hematocrit level. Both initial and 24-hour BNP levels were highly correlated to the subjects’ age ($P<0.001$) but were not correlated to the corresponding hematocrit value ($P=0.31$ and 0.17, respectively). Similarly, the rise in BNP at 24 hours was highly correlated to age ($P<0.001$) but not to the change in hematocrit ($P=0.6$). A regression plot of the latter data is shown below (Figure).

Thus, we are unable to confirm the conclusions of Drs Muscari and Zoli. It should be noted that, whereas they assessed the BNP precursor, we assayed BNP itself. This might have some bearing on the discrepancy.

Disclosures

None.

Myron D. Ginsberg, MD
Department of Neurology (D4–5)
University of Miami Miller School of Medicine
Miami, Fla

Michael D. Hill, MD, MSc
Calgary Stroke Program
Department of Clinical Neurosciences
University of Calgary
Foothills Medical Centre
Calgary, Alberta, Canada

Yuko Y. Palesch, PhD
Data Coordination Unit
Department of Biostatistics, Bioinformatics and Epidemiology
Medical University of South Carolina
Charleston, SC


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Myron D. Ginsberg, Michael D. Hill and Yuko Y. Palesch

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