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Stroke

LETTER TO THE EDITOR

Letter by van der Jagt and Suarez Regarding Article, "Impact of Goal-Directed Therapy on Delayed Ischemia After Aneurysmal Subarachnoid Hemorrhage: Randomized Controlled Trial"

Mathieu van der Jagt, MD, PhD; Jose I. Suarez, MD, FNCS, FANA

To the Editor:

The clinical trial recently published by Anetsberger et al[1] is an important addition to the literature on aneurysmal subarachnoid hemorrhage, and it is timely, given the increasing evidence indicating the importance of fluid- and hemodynamic management in neurocritical care and its strong potential to improve outcomes. However, we have several observations that we would like the investigators to address, which we believe will enhance interpretation of the results.

First, since both treatment groups had an aim to keep central venous pressure >4 mm Hg: how were central venous pressures in both groups? This is relevant since venous congestion (high central venous pressure) may facilitate cerebral edema (indeed found to be more prevalent in the control group, in which early fluid balance was also higher which may have contributed to higher central venous pressures).[2] Second, authors state that "In the absence of vasospasm, fluid management focused on maintenance of normovolemia": does this indicate that vasospasm implicated other policies than normovolemia? Third, a neurosurgeon evaluated all in hospital outcomes, among which many that are within the realm of intensive care medicine (eg, pneumonia, pulmonary edema, metabolic disturbances, myocardial pathologies etc): were there predefined criteria for the in hospital and secondary outcomes? Fourth, the definition of the primary outcome (DCI) seems ambiguous and not adhering to the 2010 Vergouwen criteria[3]: DCI was defined as a new focal neurological deficit or a cerebral infarction in the presence of vasospasm revealed by radiological imaging or both; do the authors mean that vasospasm was always necessary for DCI diagnosis (this is now not clear from their definition), and why did the authors extend the time frame from 1 hour (according to the consensus definition) to 8 hours? Fifth, arterial hypertension was significantly more often a premorbid item in the Goal-Directed Therapy group, and hypertension is known to affect the cerebral autoregulation capacity, possibly rendering the brain less sensitive to cerebral edema in induced hypertension: the authors did not adjust their analysis for this potentially important difference in the baseline characteristics. Sixth, although the authors hint at using ICP/CPP management routinely, they surprisingly do not provide any numbers. Doing so would further enhance interpretation of the results. Seventh, a conspicuous finding is that in the before-vasospasm period, the Goal-Directed Therapy group achieved a good GEDI (indicating sufficient preloading, and thus volume to the heart) and MAP ≥70 mm Hg more often than the controls but (unexpectedly) with less fluid loading and less norepinephrine; furthermore, after the prevasospasm period, GEDI and cardiac output were (significantly) lower in the Goal-Directed Therapy group, in spite of similar fluid loading and slightly more inotropics used. Could the authors comments on these seemingly contradictory findings? Finally, the authors indicate in their conclusions that their management algorithm, which focused on both normovolemia according to transpulmonary thermodilution parameters and induced hypertension, seems beneficial overall. However, we do not concur with the authors. Although we surely commend the authors for their important trial, as it certainly strengthens the evidence for the potential benefits of hemodynamic monitoring, we think their results do not contribute to the evidence in favor of efficacy of induced hypertension in aneurysmal subarachnoid hemorrhage-related DCI, because the design of the study was not appropriate to address this.

The findings of this study provide support and confirm those of previous studies,[2,4] and pave the way for further, preferably multicenter, investigations on early hemodynamic and fluid management as an essential part of critical care management of aneurysmal subarachnoid hemorrhage.

For Sources of Funding and Disclosures, see page e18.

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ARTICLE INFORMATION

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