Q2 Does fluid therapy in the management of cerebral ischemia influence outcome (CBF or clinical)?

Studies were too heterogeneous to be combined in an overall body of evidence. Their individual grading (reported in the quality assessment forms at the end of this document) hence corresponds to the body of evidence grading.

We only retrieved observational studies, which did not perform any statistical adjustment for confounders. Studies were small and those with a before-after design that may provide some interesting information were too heterogeneous in terms of design to be combined with a meta-analytical approach. Moreover, studies did not always provide consistent findings. We briefly summarise some of these studies that, at the best, may only provide hypotheses.

Normal saline bolus in six patients with vasospasm determined a significant CBF increase in areas with low perfusion [1]. In 35 patients with vasospasm receiving hypertonic saline boluses also experienced an increase of CBF [2]. Finally, hypervolemia obtained with albumin, low-molecular-weight dextran, and 10% glicerol was associated with normalization of CBF in the cerebral emisphere where it was reduced by vasospasm [3].

In two studies volemia expansion with hetastarch and albumin, or isovolemic hemodilution obtained by venisection and infusion of albumin and dextran 70, respectively, did not increase CBF [4, 5].

Two studies treated new neurological symptoms in SAH with hypervolemia using albumin, glicerol, dextran, or plasma, monitoring part of these patients with a Swan-Ganz catheter. Neurologic improvement and absence of progression to infarction in most cases led the authors to conclude that hypervolemic therapy was effective [6, 7]. The two studies, however, had very serious limitations connected to the small sample size, to the absence of a instrumental diagnosis of vasospasm, no specific definition of treatment, and lack of adjustment for confounding factor.


