Induced hypertension for vasospasm

One of the heralding signs of vasospasm is often elevated blood pressure, with whatever is left of cerebral autoregulation attempting to increase cerebral blood flow by increasing systemic pressure. Whether induced hypertension is useful in preventing arterial vasospasm is another question altogether.

One study showed that induced hypertension was able to achieve higher flow in ischemic, but not infarcted territories, despite no change in global CBF [1)].

No study of induced hypertension in isolation, though, has shown a decrease in the development of angiographic vasospasm. Thus, it is likely that hypertension may be useful in reversing neurologic deficits that develop from vasospasm, but not as a preventive mechanism by itself [2)].

Induced hypertension (IH) appears to raise CBF most consistently in previous studies, but giving a fluid bolus has also been shown to improve CBF (and thereby DO2) to regions with low baseline flow.

Dhar et al. were able to broadly compare the efficacy of transfusion to a fluid bolus and induced hypertension for augmenting cerebral oxygen delivery to vulnerable brain regions in patients with SAH. They demonstrated that the transfusion of RBCs may be an equally or even more potent intervention than the traditional measures of treating DCI, such as raising blood pressure or giving volume, especially in more anemic patients or to vulnerable brain region at highest risk for ischemia. Any potential benefits must be weighed against the known risks of excessive or unnecessary blood transfusion. Direct comparative studies including those with clinical outcomes are required to define this relative efficacy and risk-benefit ratio. However, for the first time they have provided data that suggests transfusion may be a meaningful alternative or adjunct to hemodynamic augmentation [3)].

Complications

Global edema is an independent risk factor for mortality and poor outcome after SAH. Loss of consciousness, which may reflect ictal circulatory brain arrest, is a risk factor for admission global edema, and vasopressor-induced hypertension is associated with the development of delayed global edema. Critical care management strategies that minimize edema formation after SAH may improve outcome [4)].

Immediate induction of hypertension with higher pressure targets did not result in a lower rate of Delayed Cerebral Ischemia-related infarctions but was not associated with a higher complication rate compared with an incremental approach. Future tailored blood pressure management based on patient- and time-point-specific needs will hopefully better balance the neurological advantages versus the systemic complications of induced hypertension [5)].

1)

Darby JM, Yonas H, Marks EC, Durham S, Snyder RW, Nemoto EM. Acute cerebral blood flow response to dopamine-induced hypertension after subarachnoid hemorrhage. J Neurosurg. 1994 May;80(5):857-64. PubMed PMID: 8169626.
2)

Muizelaar JP, Becker DP. Induced hypertension for the treatment of cerebral ischemia after subarachnoid hemorrhage. Direct effect on cerebral blood flow. Surg Neurol. 1986 Apr;25(4):317-25. PubMed PMID: 3952624.
3)

Dhar R, Scalfani MT, Zazulia AR, Videen TO, Derdeyn CP, Diringer MN. Comparison of induced hypertension, fluid bolus, and blood transfusion to augment cerebral oxygen delivery after subarachnoid hemorrhage. J Neurosurg. 2012 Mar;116(3):648-56. doi: 10.3171/2011.9.JNS11691. Epub 2011 Nov 18. PubMed PMID: 22098203; PubMed Central PMCID: PMC3763719.
4)

Claassen J, Carhuapoma JR, Kreiter KT, Du EY, Connolly ES, Mayer SA. Global cerebral edema after subarachnoid hemorrhage: frequency, predictors, and impact on outcome. Stroke. 2002 May;33(5):1225-32. doi: 10.1161/01.str.0000015624.29071.1f. PMID: 11988595.
5)

Veldeman M, Weiss M, Albanna W, Nikoubashman O, Schulze-Steinen H, Clusmann H, Hoellig A, Schubert GA. Incremental Versus Immediate Induction of Hypertension in the Treatment of Delayed Cerebral Ischemia After Subarachnoid Hemorrhage. Neurocrit Care. 2022 Mar 8. doi: 10.1007/s12028-022-01466-7. Epub ahead of print. PMID: 35260962.