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Role of venous drainage in cerebral arteriovenous malformation surgery, as related to the development of postoperative hyperperfusion injury.

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OBJECTIVE: To elucidate the role of venous drainage in cerebral arteriovenous malformation (AVM) surgery, with respect to the development of postoperative hyperperfusion injury. **METHODS:** For 52 patients with supratentorial AVMs, cortical capillary oxygenation (SaO₂) was assessed intraoperatively, before and after resection, in the vicinity of the AVMs, by using a microspectrophotometric method. Assessed areas were defined as being related to feeding arteries or draining veins or as distant areas. Patients were divided into three groups on the basis of postoperative angiographic findings, as follows: Group 1, all former draining veins preserved (8 patients); Group 2, > or =1 former draining vein visible (12 patients); Group 3, no former draining veins visible (32 patients). Patients and SaO₂ values were pooled and compared by using paired and unpaired t tests (P < 0.05). Venous circulation times were calculated from digital subtraction angiography films. **RESULTS:** The postresectional relative increases in SaO₂ values were highest in draining vein areas (+40.8%, compared with +25% in feeder areas and +25.5% in distant areas). Five postoperative hyperemic complications occurred (9.6%), none in Group 1 (with all draining veins preserved), two (16.7%) in Group 2, and three (9.4%) in Group 3 (with all draining veins occluded). The lowest preresectional SaO₂ values (31.7 +/- 6.2%) were measured in the drainer areas of the five patients who subsequently developed hyperperfusion injuries. Among those patients, postresectional increases in SaO₂ values were significantly greater in drainer areas (+167.8%) than in feeder areas (+28.3%) or distant areas (+25.8%). Postoperative venous circulation times in former draining veins in Group 2 were significantly greater than those in Group 1 (8.9 +/- 1.5 s versus 6.3 +/- 0.6 s). Circulation times in normal veins in the five patients with hyperperfusion injury increased from 5.6 +/- 1.0 seconds (preoperatively) to 8.4 +/- 1.9 seconds (postoperatively). **CONCLUSION:** Postoperative hyperperfusion injury after resection of cerebral AVMs can be explained on the basis of unconstrained arterial inflow into cortical areas, which are rendered hypoxic/ischemic by longstanding preoperative venous hypertension. The risk for postoperative breakthrough complications seems higher in the presence of multiple draining veins, which also participate in the physiological venous drainage system of the ipsilateral hemisphere.

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