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Distributions of local oxygen saturation and its response to changes of mean arterial blood pressure in the cerebral cortex adjacent to arteriovenous malformations.

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BACKGROUND AND PURPOSE: To test the hypothesis that neither "steal" as cortical ischemia caused by reduced perfusion pressure nor "breakthrough" on the grounds of loss of pressure autoregulation exist in brain tissue surrounding arteriovenous malformations (AVMs), we established patterns of cortical oxygen saturation (SO(2)) adjacent to AVMs and its behavior after alterations of mean arterial blood pressure. METHODS: With a microspectrophotometer, SO(2) was scanned in the cortex around AVMs of 44 patients before and after resection and in that of a non-AVM group (n=42) before transsylvian dissection. Autoregulation was evaluated by linear regression analysis after elevation of mean arterial blood pressure (5 microg/min IV noradrenaline). SO(2) values were calculated as medians, percentage of critical values (<25% SO(2)), and coefficients of variance (approximate heterogeneity of SO(2) distributions). All values are given as mean+/-SD. RESULTS: Forty patients with AVM had an uneventful postoperative course (group A). Four hyperemic complications ("breakthrough") occurred (group B). Autoregulation was tested intact in all groups at all times. Preoperative SO(2) distributions in groups A and C (non-AVMs) were identical. In group B, significantly ($P<0.05$) lower medians (group A, 52.9+/-16.3%; group B, 44.2+/-17.1%; group C, 51.9+/-11.5% SO(2)), more critical values (group A, 6.5+/-5.1%; group B, 14.7+/-11.1%; group C, 7.1+/-4.9%), and heterogeneous SO(2) distributions (group A, 20.2+/-12.7%; group B, 27.9+/-12.4%; group C, 26.8+/-10.9%) were seen. Increase of median values was significantly higher in group B (76.3+/-10.4% SO(2)) than in group A (65.9+/-13.4% SO(2)) after resection. CONCLUSIONS: Severely hypoxic areas are uncommon in the cortex adjacent to AVMs and occur predominantly in patients prone to hyperemic complications. Reduced perfusion pressure is compensated in most cases, and moderate hyperemia prevails after excision. Reperfusion into unprotected capillaries of severely hypoxic cortical areas results in "breakthrough," for which vasoparalysis appears not to be the underlying mechanism.

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