# Cerebral hyperperfusion after revascularization inhibits development of cerebral ischemic lesions due to artery-to-artery emboli during carotid exposure in endarterectomy for patients with preoperative cerebral hemodynamic insufficiency: revisiting the "impaired clearance of emboli" concept

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## Abstract

The purpose of the present study was to determine whether cerebral hyperperfusion after revascularization inhibits development of cerebral ischemic lesions due to artery-to-artery emboli during exposure of the carotid arteries in carotid endarterectomy (CEA). In patients undergoing CEA for internal carotid artery stenosis (≥70%), cerebral blood flow (CBF) was measured using single-photon emission computed tomography (SPECT) before and immediately after CEA. Microembolic signals (MES) were identified using transcranial Doppler during carotid exposure. Diffusion-weighted magnetic resonance imaging (DWI) was performed within 24 h after surgery. Of 32 patients with a combination of reduced cerebrovascular reactivity to acetazolamide on preoperative brain perfusion SPECT and MES during carotid exposure, 14 (44%) showed cerebral hyperperfusion (defined as postoperative CBF increase ≥100% compared with preoperative values), and 16 (50%) developed DWI-characterized postoperative cerebral ischemic lesions. Postoperative cerebral hyperperfusion was significantly associated with the absence of DWI-characterized postoperative cerebral ischemic lesions (95% confidence interval, 0.001-0.179; p = 0.0009). These data suggest that cerebral hyperperfusion after revascularization inhibits development of cerebral ischemic lesions due to artery-to-artery emboli during carotid exposure in CEA, supporting the "impaired clearance of emboli" concept. Blood pressure elevation following carotid declamping would be effective when embolism not accompanied by cerebral hyperperfusion occurs during CEA.

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