

Cerebral Hyperperfusion Syndrome by the Numbers: Transient Focal Neurological Deficit, Imaging-Proven Focal Hyperperfusion, and High Graft Flow Rate Following Superficial Temporal Artery–Middle Cerebral Artery bypass in a Patient With Symptomatic Carotid Occlusion—Case Report

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BACKGROUND AND IMPORTANCE: Cerebral hyperperfusion syndrome (CHS) is a well-known complication of superficial temporal artery (STA) to middle cerebral artery (MCA) bypass for ischemic cerebrovascular disease. While this argues against “low flow” in the bypass construct, flow rates in the graft have not been previously quantified in the setting of CHS.

CLINICAL PRESENTATION: A 58-yr-old man presented with recurrent left hemispheric ischemic strokes and fluctuating right hemiparesis and aphasia. Vascular imaging revealed left cervical internal carotid artery occlusion and perfusion imaging confirmed left hemispheric hypoperfusion. After failing to respond to maximal medical therapy, the patient underwent single-barrel STA-MCA bypass. Postoperatively, his symptoms resolved and blood pressure (BP) was strictly controlled within normal range. However, 2 d later, he developed severe expressive aphasia. CTA demonstrated a patent bypass graft and SPECT showed focal hyperperfusion in Broca’s speech area. Seizure activity was ruled out. A high graft flow rate of 52 mL/min was documented by quantitative magnetic resonance angiography (MRA). Thus, the diagnosis of CHS was made and managed with strict BP control. The patient exhibited complete recovery of speech over a period of days and was discharged home. Repeat SPECT at 4 mo showed resolution of hyperperfusion and quantitative MRA demonstrated reduction of graft flow rate to 34 mL/min.

CONCLUSION: This is the first case of perfusion imaging-proven CHS after STA-MCA bypass, where high graft flow rates are objectively documented. Our observations constitute irrefutable evidence challenging the classic belief that the STA-MCA bypass is a low-flow construct.

KEY WORDS: Cerebral blood flow, Cerebral ischemia, Extracranial–intracranial bypass, Hyperperfusion, Quantitative magnetic resonance angiography

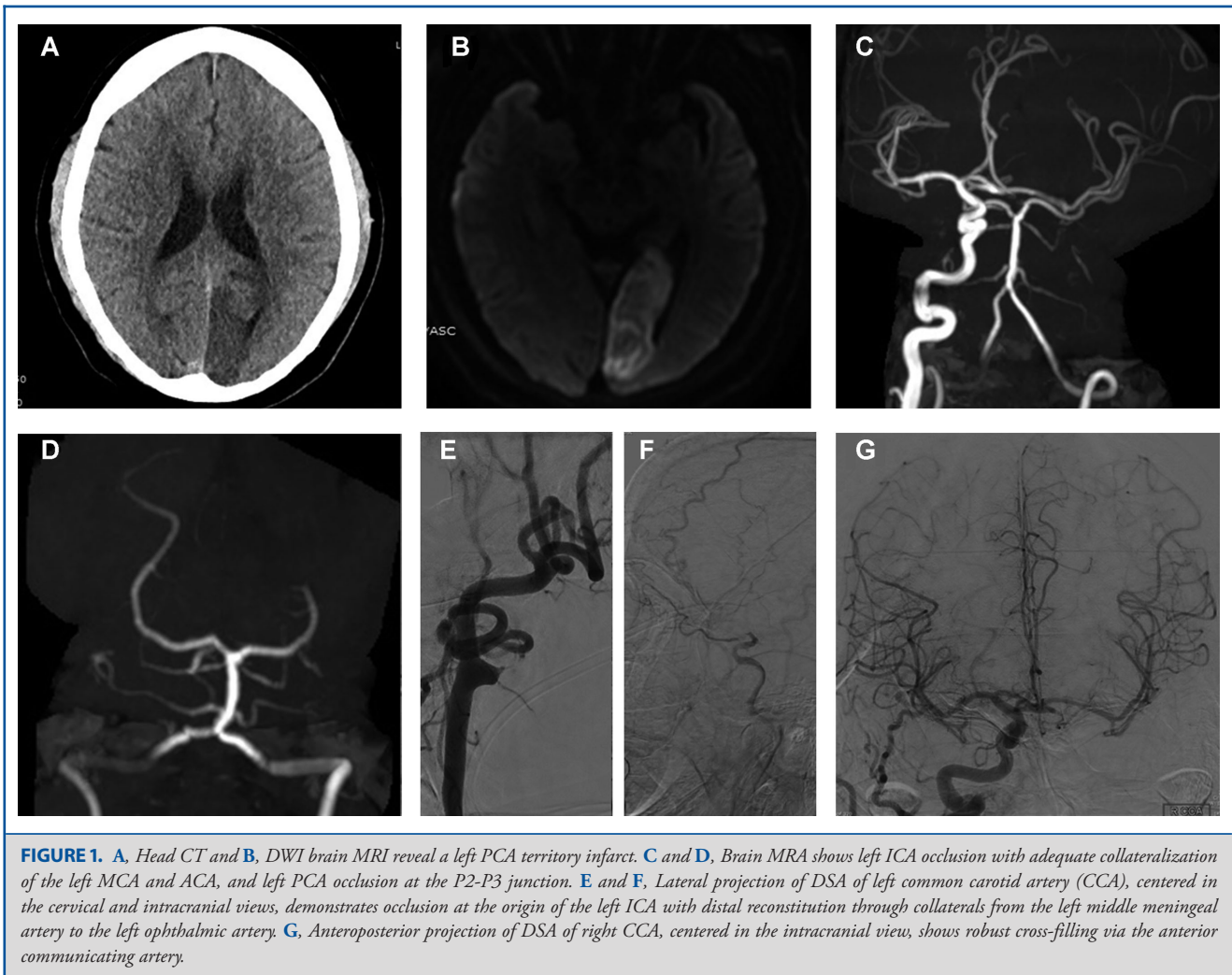
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Extracranial–intracranial (EC-IC) bypass surgery is commonly performed to supplement cerebral blood flow (CBF) for secondary stroke prevention in patients with

ischemic cerebrovascular disease (ICVD).^{1–6} Given its efficacy and simplicity, the “low-flow” superficial temporal artery (STA) to middle cerebral artery (MCA) bypass

ABBREVIATIONS: **AcomA**, anterior communicating artery; **BP**, blood pressure; **CBF**, cerebral blood flow; **CHS**, cerebral hyperperfusion syndrome; **EC-IC**, Extracranial–intracranial; **EEG**, electroencephalogram; **ICA**, internal carotid artery; **ICVD**, ischemic cerebrovascular disease; **MCA**, middle cerebral artery; **MRA**, magnetic resonance angiography; **PCA**, posterior cerebral artery; **qMRA**, quantitative magnetic resonance angiography; **SBP**, systolic blood pressure; **SPECT**, single photon emission computed tomography; **STA**, superficial temporal artery.

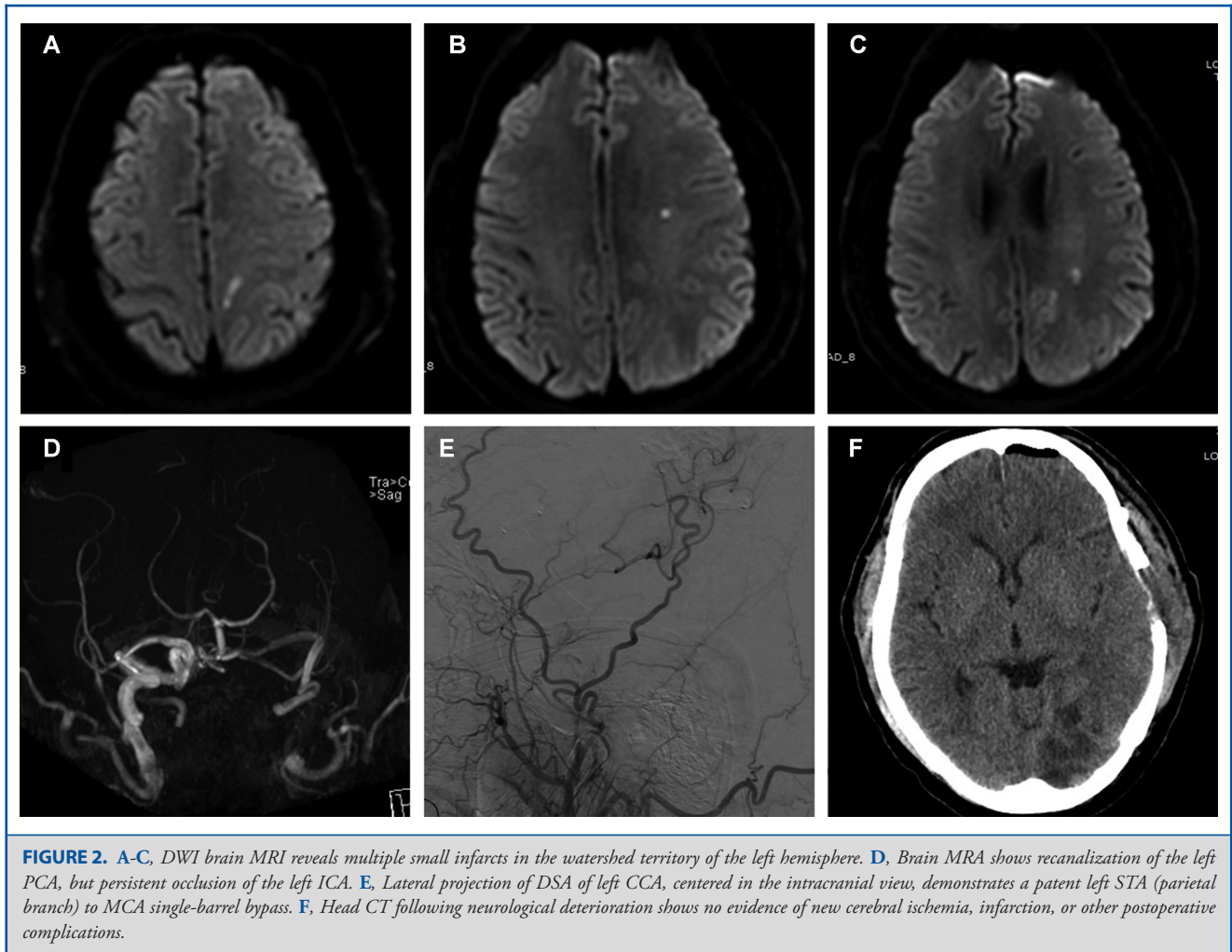


constitutes the workforce of cerebral revascularization in the anterior circulation. Immediate graft flow rates of 15 to 35 mL/min are typically observed after single-barrel STA-MCA bypass.⁷⁻⁹ However, using quantitative magnetic resonance angiography (qMRA) via noninvasive optimal vessel analysis (NOVA qMRA®, VasSol Inc, River Forest, Illinois),¹⁰⁻¹² we have frequently measured postoperative graft flow rates of 50 to 100 mL/min after single-barrel STA-MCA bypass. Consistent with our observations, cerebral hyperperfusion syndrome (CHS) occurs after single-barrel STA-MCA bypass in up to 45% of patients with ICVD.¹⁻⁶ Notwithstanding, no prior studies provided quantitative graft flow assessments in this setting. In this report, we present incontestable evidence that augmentative STA-MCA bypass grafts for ICVD may carry high flow rates, leading to postoperative CHS in some patients.

CLINICAL PRESENTATION

A 58-yr old diabetic and hypertensive man presented with sudden-onset visual blurriness and a systolic blood pressure (SBP) of 215 mm Hg. Neurological exam revealed right homonymous hemianopsia. Head CT and brain MRI showed left posterior cerebral artery (PCA) distribution infarct. Head and neck magnetic resonance angiography (MRA) and cerebral angiography demonstrated occlusions at the left internal carotid artery (ICA) origin and P2-P3 junction of the left PCA, with a robust anterior communicating artery (AcomA; Figure 1). Echocardiography was unremarkable. The etiology of the PCA stroke remained unclear. The patient was discharged home 2 wk later on blood pressure (BP) medications and warfarin.

Three days later, he returned with dizziness and headache. Neurological exam revealed mild right hemiparesis and expressive



aphasia. Head CT was unchanged. Brain MRI demonstrated multiple left hemispheric watershed infarcts (Figure 2). The patient had been compliant with antihypertensive medications and international normalized ratio was therapeutic. Head and neck MRA showed left PCA recanalization, but persistent left ICA occlusion (Figure 2). Cerebral hemodynamic failure was suspected. Technetium-99m hexamethylpropyleneamine oxime (^{99m}Tc HMPAO) single photon emission computed tomography (SPECT) demonstrated marked CBF asymmetry favoring the right hemisphere, unaffected by acetazolamide challenge (Figure 3). Despite maximal medical treatment, including permissive hypertension, the patient failed to improve clinically, with fluctuating right hemiparesis and expressive aphasia. Decision was made to surgically revascularize the left cerebral hemisphere via a single-barrel STA-MCA bypass, using the parietal branch of the STA and a frontal M4 branch of the MCA. However, intraoperative flow measurements in the graft and recipient were not performed.

Following an uneventful bypass procedure, the patient's motor and speech deficits resolved immediately. However, on the second postoperative day, he developed worsening expressive aphasia. His SBP had been maintained in the range of 100 to 150 mm Hg postoperatively. Cerebral hypoperfusion was suspected. However, head CT was unchanged and CTA showed a patent bypass graft. A 24-h electroencephalogram (EEG) demonstrated no epileptiform activity, thus ruling out the possibility of seizures. SPECT confirmed globally improved left hemispheric perfusion, with focal hyperperfusion in the region of the bypass, around Broca's area. qMRA demonstrated a flow rate of 52 mL/min in the bypass graft (Figure 3). A diagnosis of postoperative CHS was made and strict BP control (100-120 mm Hg) was implemented with intravenous nicardipine. Aphasia resolved completely over a few days and the patient was discharged home 2 wk later.

At his 4-mo follow-up, the patient had no new cerebrovascular events. Repeat SPECT revealed symmetric hemispheric

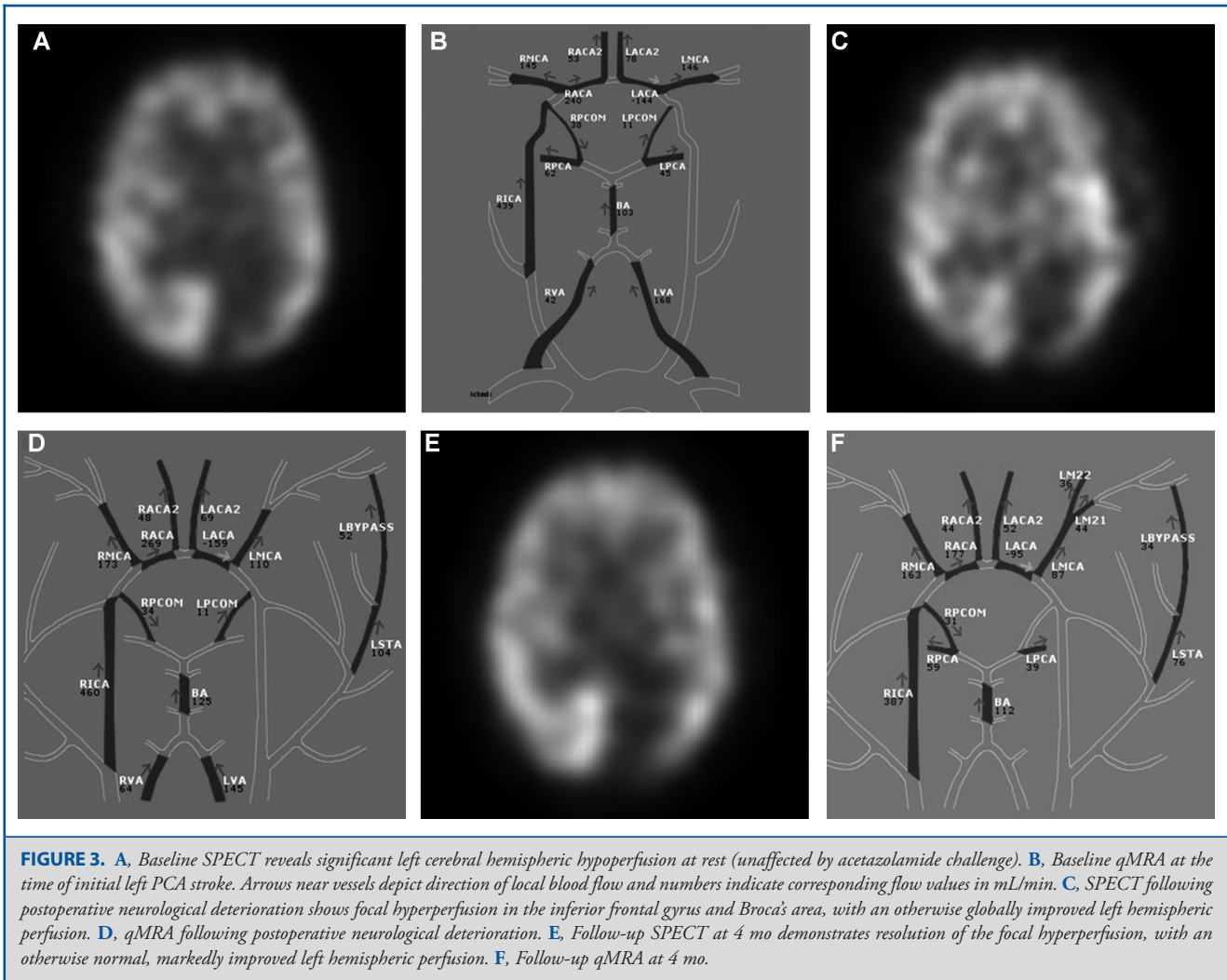


FIGURE 3. **A,** Baseline SPECT reveals significant left cerebral hemispheric hypoperfusion at rest (unaffected by acetazolamide challenge). **B,** Baseline qMRA at the time of initial left PCA stroke. Arrows near vessels depict direction of local blood flow and numbers indicate corresponding flow values in mL/min. **C,** SPECT following postoperative neurological deterioration shows focal hyperperfusion in the inferior frontal gyrus and Broca's area, with an otherwise globally improved left hemispheric perfusion. **D,** qMRA following postoperative neurological deterioration. **E,** Follow-up SPECT at 4 mo demonstrates resolution of the focal hyperperfusion, with an otherwise normal, markedly improved left hemispheric perfusion. **F,** Follow-up qMRA at 4 mo.

perfusion, with no evidence of residual hyperperfusion. qMRA showed reduced flow rate of 34 mL/min in the bypass graft (Figure 3). Cerebral angiography demonstrated a patent and maturing graft, with reduced interhemispheric collateral flow across the AComA.

IRB Approval and Patient Consent

IRB approval was not obtained, since it is not required for case reports in our institution. The patient gave verbal consent.

DISCUSSION

This report demonstrates that CHS is a real complication of STA-MCA bypass in patients with ICVD, its occurrence depending on the flow rate carried by the graft. Using the combination of SPECT and qMRA, we found that symptomatic CHS occurred in the early postoperative period when flow in the graft surpassed 50 mL/min. We also observed that recovery from

CHS was associated with significant reduction in graft flow, from 52 mL/min to 34 mL/min. These findings strongly challenge the classic belief that an augmentative STA-MCA bypass is a low-flow construct. This is the first time high flow rates are quantitatively documented in postoperative CHS following STA-MCA bypass.

CHS is thought to result from rapid restoration of regional CBF in a chronically hypoperfused brain, with maximally dilated leptomeningeal arteries and limited capacity for cerebrovascular autoregulation.^{13,14} It is a classic complication of carotid revascularization and EC-IC bypass for moyamoya or atherosclerotic ICVD.^{1-6,13,14} Following a single-barrel STA-MCA bypass, up to 45% of patients with ICVD may develop CHS.¹⁻⁶ It usually develops hours to days after revascularization and, in its most typical form after carotid revascularization, is associated with CBF increases of 100% or more.^{13,14} Risk factors include advanced age (>70 yr), longstanding hypertension, diabetes mellitus, cerebral hypoperfusion with impaired cerebrovascular reserve, and

postoperative hypertension,^{13,14} all of which except age and postoperative hypertension were present in this patient. Whether other yet unidentified risk factors might have also contributed to the development of CHS in this patient remains unknown. Clinically, CHS manifests with headaches, seizures, and focal neurological deficits. Untreated, life-threatening cerebral edema and/or intracerebral hemorrhage may ensue.^{13,14} CHS is a diagnosis of exclusion. In the setting of EC-IC bypass, the possibility of cerebral ischemia secondary to graft thrombosis, nonconvulsive seizures,¹⁵ postoperative hematoma, or other complications should be ruled out first. For this reason, head CT, CTA, and a 24-h EEG were performed in this patient and were unremarkable. Conversely, SPECT and qMRA confirmed the diagnosis of CHS. SPECT has been shown to be a reliable modality for the diagnosis and monitoring of CHS following surgical revascularization.¹⁶ Similarly, qMRA was found to have good-to-excellent correlation with intraoperative flow measurements and postoperative angiographic findings after EC-IC bypass.¹¹ Distinguishing CHS from cerebral ischemia is of paramount importance when managing revascularized patients. Strict BP and hydration control constitute the mainstay of treatment for CHS, resulting in rapid and complete resolution of symptoms within 2 wk in the vast majority of patients.² In contrast, the same measures can exacerbate cerebral ischemia and precipitate cerebral infarction. Failure to understand the importance of BP management in this setting can lead to potentially catastrophic complications and poor outcome.

Finally, it should be noted that, although a high bypass flow rate increases the risk of CHS, its occurrence is likely multifactorial. In fact, we routinely obtain qMRA to assess intracranial hemodynamics after EC-IC bypass and have occasionally observed flow rates as high as 50 to 100 mL/min in otherwise asymptomatic patients. Thus, the diagnosis of CHS cannot be made solely on the basis of qMRA flow rate values. For this reason aggressive antihypertensive therapy should be avoided in patients with overly robust bypass grafts who are otherwise asymptomatic, as it might precipitate bypass graft thrombosis and cerebral ischemia.

CONCLUSION

Augmentative STA-MCA bypass grafts can carry high flow rates in excess of 50 mL/min, which may increase the risk of postoperative CHS. The latter is a diagnosis of exclusion and should be promptly recognized and differentiated from cerebral ischemia, since strict BP and hydration control constitute the mainstay of treatment for the former, while the same measures may exacerbate the latter.

Disclosure

The authors have no personal, financial, or institutional interest in any of the drugs, materials, or devices described in this article.

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COMMENTS

This nice case report is a good addition to the armamentarium of techniques needed to have the job well done with maximal security. As described in detail in this nice paper the authors show that the STA-MCA bypass offers more than enough flow but the early CHS when measured in mL/min in this case report was dramatic. Indeed, one should follow bypass patients with already obstructed vessels carefully and be prepared to manage CHS if clinically suspected necessitating then radiological confirmation and exclusion of other reasons, too. This helps the

neurosurgeon to avoid unnecessary complications and even permanent deficits after a seemingly technically perfect bypass to allow the best recovery for the already sick patients. Furthermore, this study shows also that bypass surgeons are still needed but the demand for 'high-flow' bypasses is decreasing also in the management of complex aneurysms.

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A hyperperfusion syndrome following revascularization for ischemia can be observed in patients after STA-MCA bypass in a few patients. I have seen this in a number of patients after STA-MCA bypass, or after

radial artery to MCA bypass. Sometimes, it can result in a focal brain hemorrhage as well. So, the patient's blood pressure must be carefully controlled in the postoperative period for at least 48 hours.

The authors have documented the hyperperfusion syndrome quite nicely with HMPAO-SPECT scanning. The occurrence of postoperative focal seizures can mimic all of the symptoms, and the blood flow increases, and must be ruled out with electroencephalography, and the patient must be placed on anticonvulsants as well.

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