

Postcarotid Endarterectomy Hyperperfusion or Reperfusion Syndrome

Theodoros Karapanayiotides, MD; Reto Meuli, PhD; Gerald Devuyst, MD;
Bartłomiej Piechowski-Jozwiak, MD; Annelise Dewarrat, MD; Patrick Ruchat, MD;
Ludwig Von Segesser, MD; Julien Bogousslavsky, MD

Background and Purpose—Hyperperfusion syndrome (HS) after carotid endarterectomy (CEA) has been related to impaired cerebrovascular autoregulation in a chronically hypoperfused hemisphere. Our aim was to provide new insight into the pathophysiology of the HS using magnetic resonance imaging (MRI) studies with diffusion-weighted imaging (DWI) and perfusion-weighted imaging (PWI).

Methods—Five out of 388 consecutive patients presented 2 to 7 days after CEA, partial seizures (n=5), focal deficits (n=5), and intracerebral hemorrhage (n=3). In 4 patients, using sequential examinations, we identified vasogenic or cytotoxic edema by DWI; we assessed relative interhemispheric difference (RID) of cerebral blood flow (CBF) by PWI; and we measured middle cerebral artery mean flow velocities (MCA Vm) by transcranial Doppler (TCD).

Results—None of the patients presented pathological DWI hyperintensities, consistent with the absence of acute ischemia or cytotoxic edema. In 2 patients, we found an MRI pattern of reversible vasogenic edema similar to that observed in the posterior leukoencephalopathy syndrome. Middle cerebral artery (MCA) mean flow velocities (Vm) were not abnormally increased at any time. PWI documented a 20% to 44% RID of CBF in favor of the ipsilateral to CEA hemisphere.

Conclusions—HS can occur in the presence of moderate relative hyperperfusion of the ipsilateral hemisphere. MCA Vm values may not accurately reflect RID of CBF over the cortical convexity. We suggest that the hemodynamic pathogenetic mechanisms of the HS are more complicated than hitherto believed and that they may be more accurately described by the term “reperfusion syndrome.” (*Stroke*. 2005;36:21-26.)

Key Words: carotid endarterectomy ■ reperfusion ■ ultrasonography, Doppler, transcranial

Hyperperfusion syndrome (HS) after carotid endarterectomy (CEA) consists of a classical clinical triad: transient focal deficits associated with ipsilateral migraine-like headache, seizures, and intracerebral hemorrhage (ICH).¹ The pathophysiology of the syndrome is still obscure, being probably related to the preoperative loss of vascular autoregulatory mechanisms in a chronically hypoperfused hemisphere. Although a degree of hyperperfusion seems to occur in most patients in the immediate postoperative period, florid HS develops in only a few.²⁻⁴ Reversible ipsilateral brain edema evolving in patients after CEA has only recently been recognized,⁵ and its pathophysiology is indeterminate. Hitherto, diffusion-weighted imaging (DWI) has been used in very few patients with HS after CEA,⁶ and perfusion-weighted imaging (PWI) has not been applied to the investigation of the HS. A study⁷ evaluated CEA with sequential PWI, but none of the patients had HS.

We now report 5 patients with HS, 4 of whom were systematically studied with sequential computed tomography

(CT), standard magnetic resonance imaging (MRI), DWI, and PWI, and transcranial Doppler (TCD). We suggest that this post-CEA complication, at least in some patients, may result from interhemispheric asymmetries of perfusion and in the absence of striking absolute hyperperfusion.

Subjects and Methods

Patients

Three-hundred eighty-eight consecutive patients underwent CEA at our center by 2 experienced vascular surgeons. CEAs were routinely performed under loco-regional anesthesia without shunting. If neurological deterioration was observed during clamping of the carotid, shunting was performed (Figures 1-5 and Figure I available online only at <http://www.strokeaha.org>).

Data Acquisition and Analysis

TCD was performed using a Multi-Dop T2 instrument (DWL) with a 2-Mhz pulsed-wave transducer. The middle cerebral arteries (MCAs) were insonated using the temporal bone window at a depth of 50 to 60 mm, where the Doppler signal was mostly accurate and audible. The Pulsating Index (PI) quantifies the shape of the TCD

Received September 2, 2004; final revision received September 13, 2004; accepted October 7, 2004.

From the Department of Neurology (T.K., G.D., B.P.-J., A.D., J.B.), Centre Hospitalier Universitaire Vaudois, Lausanne, Switzerland; Department of Diagnostic and Interventional Radiology (R.M.) and Department of Cardiovascular Surgery (P.R., L.V.S.), CHUV, Lausanne, Switzerland.

Correspondence to Theodoros Karapanayiotides, 90-92 Agias Paraskevis Str, 111 44 Athens, Greece. E-mail theoneu@otenet.gr

© 2004 American Heart Association, Inc.

Stroke is available at <http://www.strokeaha.org>

DOI: 10.1161/01.STR.0000149946.86087.e5

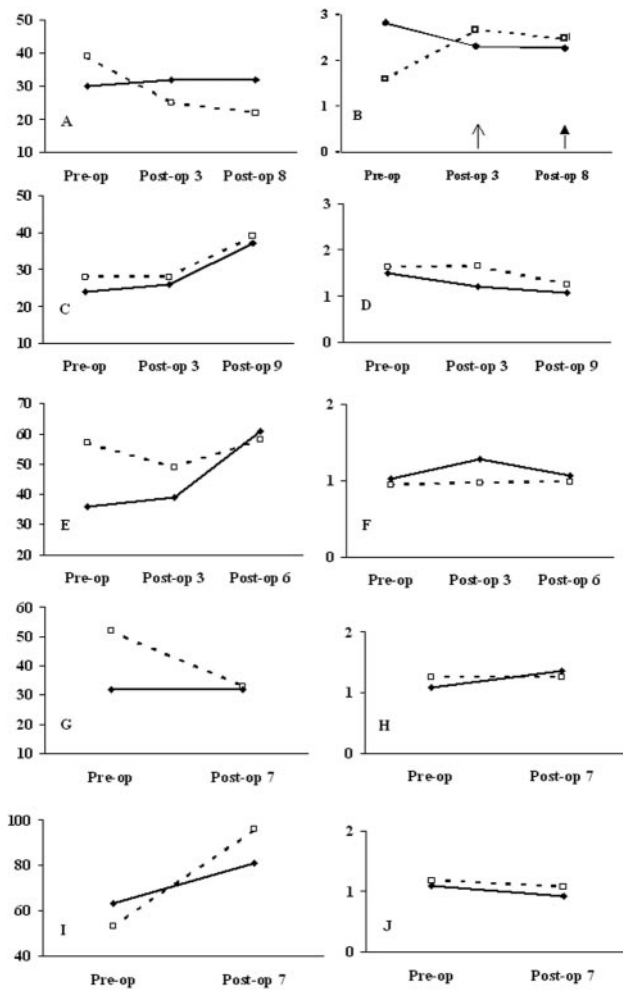


Figure 1. Baseline (preoperative) and post-CEA (postoperative) values of TCD-measured MCA Vm (left column; cm/sec) and MCA PI (right column) in patients 1 to 5. Light arrows indicate day of onset of “hyperperfusion” symptoms. Dark arrows indicate day at which the first PWI study was performed. Solid lines indicate hemisphere ipsilateral to CEA (IH); dotted lines indicate contralateral hemisphere (CH). A and B, Patient 1. Three days before surgery, IH MCA Vm is moderately reduced. After the appearance of “hyperperfusion” symptoms, RID is reversed in favor of the IH (from -23% to $+28\%$); however, right MCA Vm is normal. The left MCA PI increases by 67%, whereas the right MCA PI decreases moderately. By postoperative day 8, interhemispheric asymmetry increases (RID: 45%) as CH MCA Vm reaches lower normal values, whereas IH MCA Vm remains constant. C and D, Patient 2. Preoperatively, MCA Vm values on both sides are within the inferior or normal limits. Postoperatively, the low baseline velocities increase almost symmetrically to values well below the upper normal limits. PI in the IH decreases earlier and to a greater extent than the PI in the CH. However, all PI values are within normal limits. E and F, Patient 3. The preoperative relative hypoperfusion of the IH (RID: -33%) finally resolves on postoperative day 6 as a consequence of a 69% increase of the baseline MCA Vm, not exceeding upper normal limits. At the onset of “hyperperfusion” symptoms, the baseline IH PI is increased by 25%. G and H, Patient 4. The baseline relative hypoperfusion of the IH resolves because of a 37% decrease of the contralateral MCA velocity. Right MCA PI increases by 25%. I and J, Patient 5. This was the only patient in which postoperative MCA Vm were bilaterally above the normal limits because of an 81% increase in the IH and a 29% increase in the CH. PI slightly decreased in both hemispheres.

waveform and is inversely correlated to cerebral vascular resistance. Using MCA mean flow velocity (Vm), we evaluated the relative

interhemispheric difference (RID) as follows: $RID (\%) = 100 \times (Vm \text{ MCA ipsilateral to CEA} - Vm \text{ MCA contralateral to CEA}) / Vm \text{ MCA contralateral to CEA}$.

MRI was performed on a 1.5-T Magnetom Vision MRI unit (Siemens). After routine T1 (repetition time [TR]=510, echo time [TE]=12) and fast spin-echo T2 (TR=3800, TE=90) sequences, we obtained an echoplanar single-shot spin-echo DWI trace sequence (TR=5000 ms, TE=100 ms, $b=1000 \text{ sec/mm}^2$, 20 5-mm-thick slices with 1.5-mm gap, matrix size 128×128). PWI series was performed immediately afterward (T2* echoplanar gradient echo, TR=120 ms, TE=45 ms, 5 5-mm-thick slices with 1.5-mm gap, matrix size 128×128 ; and 40 acquisitions obtained every 2 seconds during intravenous administration of 0.2 mmol/kg Gd-DTPA; Omniscan, Nycomed Amersham), followed by a 15-mL saline flush at a rate of 5 mL/second into an antecubital vein with a power injector (Spectris MR Injection System; Medrad). Perfusion maps were constructed by a pixel-by-pixel analysis of the time series. The maximum slope of decrease, which is associated with the relative cerebral blood flow (rCBF), was computed. Regions of interest were chosen in comparable contralateral territories of the MCA and signal-to-time change curves were plotted. The RID of rCBF in the regions of interest was calculated according to the formula $RID (\%) = 100 \times (rCBF \text{ ipsilateral to CEA} - rCBF \text{ contralateral to CEA}) / rCBF \text{ contralateral to CEA}$. Values of rCBF in the specified regions of interest were measured in arbitrary MRI units.

Results

Two to 7 days postoperatively, focal neurological deficits developed in 5 patients, accompanied in all by partial seizures that were secondarily generalized in patients 3 and 4. Severe to mild headache ipsilateral to the operated carotid preceded the onset of symptoms by 3 hours to 6 days in patients 4 and 5. Five to 8 days post-CEA, a >2 -cm in diameter ICH evolved in patients 1, 3, and 5, whereas cortical petechial hemorrhages evolved in patient 4. The study of patient 1 had been published before as a case report.⁸ All 5 patients had preoperative intra-arterial 4-vessel angiography and brain CT. Extracranial Doppler and TCD were performed within 1 week before surgery in all patients. Preoperative MRI was available in patients 1 through 3. Within 24 hours after the onset of symptoms of “hyperperfusion,” we performed brain CT, electroencephalogram, extracranial Doppler, and TCD in all patients. Patients 1 through 4 had sequential MRI studies with DWI and magnetic resonance angiography (Table 1). Sequential PWI studies were performed in patients 1, 2, and 4. The baseline characteristics of the patients in whom HS developed compared with those who did not are presented in Table I (available at <http://www.strokeaha.org>).

Discussion

Hyperperfusion after CEA is classically defined as $>100\%$ increase in CBF compared with preoperative values.^{1,4,9} However, in a study using the intraoperative ^{133}Xe technique,¹⁰ one-third of the patients who presented headache and seizures postoperatively had only modest CBF increases (27% to 36%). Using the same technique, another study⁹ could not document striking hyperperfusion in 5 of 14 patients in whom ICH developed after CEA. In a ^{133}Xe study,¹¹ half of the patients in whom ICH developed after CEA had $<30\%$ increase in CBF compared with preoperative values. Conversely, in recent prospective SPECT studies,^{4,12} all 4 patients who presented clinically HS showed $>100\%$ CBF increase.

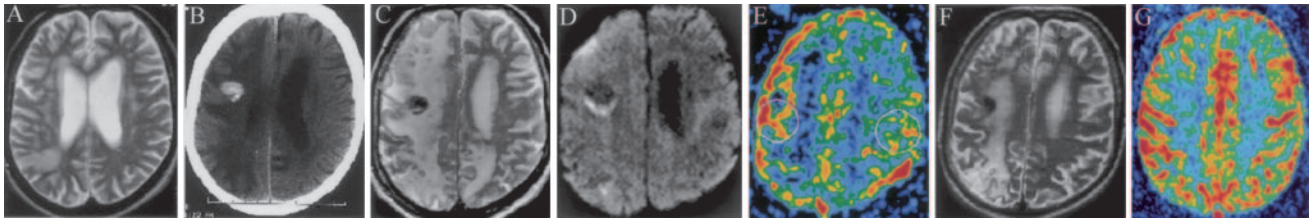


Figure 2. Patient 1. T2-weighted MRI 11 weeks before CEA demonstrates a right parietal infarct (A). Nonenhanced axial CT obtained on postoperative day 8, soon after the onset of neurological deterioration, demonstrates diffuse hypodensity in the right frontal and parietal lobes and a left posterior paraventricular hypodensity. The hypodensity predominantly involves the white matter and produces a mass effect on the body of the right lateral ventricle. A small subcortical frontal hyperdensity corresponds to hemorrhage (B). T2-weighted MR images 12 hours later show hyperintensity in the regions demonstrating hypodensity on CT. The old right parietal infarction is also evident (C). On axial DWI at a similar level, these same regions appear isointense, consistent with the absence of cytotoxic edema (D). PWI demonstrates a relative hyperperfusion in the right frontal and parietal lobes and in the left inferior parietal lobule. By calculating the arbitrary MRI units in the regions of interest (white circles), we found a 20% relative interhemispheric difference (RID) in favor of the right hemisphere (E). A repeat MRI after 4 months demonstrates regression of the extensive right hemispheric edema, ipsilateral periventricular leukoencephalopathy, and inferior parietal gliosis. A T2 solid hypointensity in the region of the right frontal hematoma corresponds to hemosiderin deposits (F). PWI documented isoperfusion of the hemispheres (G).

To the best of our knowledge, this is the first PWI study in patients presenting symptoms of “hyperperfusion” after CEA. A previous MRI perfusion study⁷ found early bolus arrival time on the ipsilateral hemisphere in 4 of 7 patients who were asymptomatic after CEA, confirming the common occurrence of the reperfusion hyperemic response. In the present study, the relative hemispheric hyperperfusion ipsilateral to CEA in patients 1 to 4, as evidenced by PWI, was only moderate, ranging from 20% to 44%. Because PWI is not a quantitative method and preoperative PWI studies were not performed, we cannot draw conclusions about absolute CBF differences. However, these 4 patients had relatively unchanged and normal ipsilateral MCA Vm values early after the onset of symptoms. This observation raises the question whether the so-called hyperperfusion symptoms can occur in the absence of excessively increased absolute CBF values. Even if relative CBF increases are more important, the values of reference remain to be defined: preoperative and ipsilateral to the CEA, postoperative and contralateral to the CEA, or both?

TCD has been extensively used to investigate the time course of cerebral hemodynamics during and after CEA³ but has a major limitation. Because TCD measures velocity and not flow, there exists a good correlation between alterations in measured velocity and changes in regional CBF, provided only that the diameter of the studied artery remains constant.¹³ However, changes in MCA diameter during the

course of the HS are unknown. A single study¹⁴ has addressed the issue of regional CBF during CEA, correlating TCD-measured velocities with the intra-arterial ¹³³Xe technique. The authors proposed that MCA Vm reflects hemodynamic events in the territory of the lenticulostriate arteries far better than in the cerebral cortex. Our results suggest that MCA Vm values either underestimate or overestimate the relative interhemispheric CBF differences over the cortical convexity.

Focal neurological deficits in patients who are initially well after CEA may result from brain ischemia caused by carotid occlusion or embolization from the endarterectomy site. DWI can differentiate cytotoxic from vasogenic edema. The former is characterized by DWI hyperintensity and a decline of the apparent diffusion coefficient (ADC) of water, whereas the latter is characterized by relative increase in the diffusion of water molecules and only a variable weak change in DWI signal intensity.^{6,15} In our study, the absence of pathological DWI hyperintensity in the hemisphere ipsilateral to the operated carotid, at least 6 hours after the onset of the hyperperfusion symptoms, suggests a nonsignificantly lowered ADC, consistent with the absence of acute cerebral infarction or cytotoxic edema. Furthermore, PWI did not demonstrate a perfusion deficit corresponding to the recent T2 hyperintensities, but on the contrary documented a mild to moderate relative hyperperfusion. Therefore, in patients 1 to 4, we cannot attribute the development of focal neurological

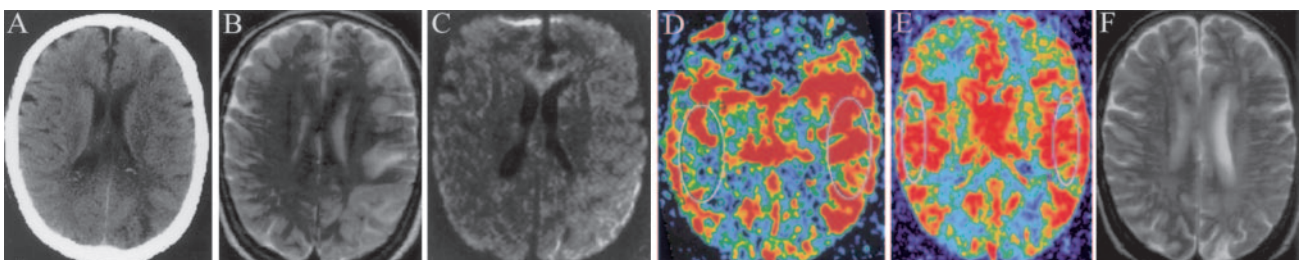


Figure 3. Patient 2. Nonenhanced axial CT, 7 hours after the onset of symptoms, shows left frontal, parietal, and occipital hypodensities involving predominantly the white matter, and some involvement of the overlying cortex with sulcal effacement (A). A few hours later, axial T2-weighted MR images demonstrate diffuse edema of the left hemisphere (B). On axial DWI images at the same level, these regions are isointense to slightly hyperintense, consistent with the absence of acute brain ischemia. The slight diffuse DWI hyperintensity is most probably caused by the T2 component of DW images, known as the T2 shine-through effect. (C). PWI documents a relative hyperperfusion (RID=44%) of the left hemisphere (D). A repeat PWI 6 days later demonstrates complete resolution of the previous relative interhemispheric difference (E). Two months later, the extensive T2 hyperintensities present in B have resolved (F).

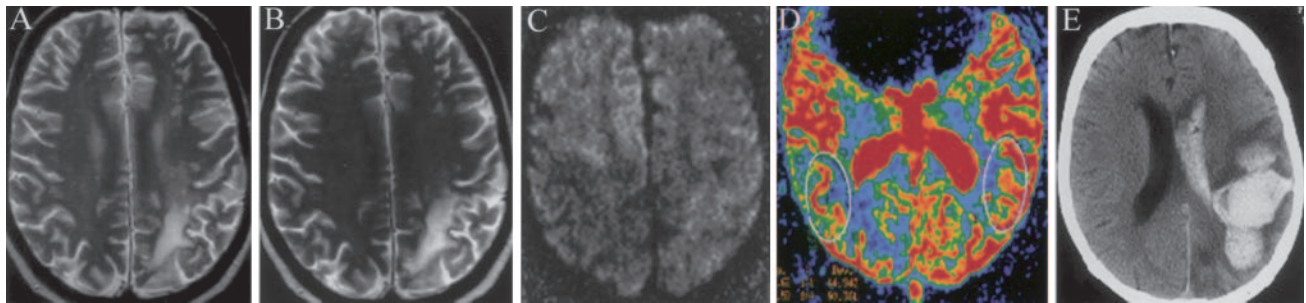


Figure 4. Patient 3. Axial T2-weighted MR image 2 months before surgery shows hyperintensity in the left inferior parietal lobule, consistent with the parietal infarction that complicated the cerebral intra-arterial angiography 1 month earlier (A). MR image 18 hours after the onset of partial seizures reveals only the old left parietal infarction (B). On axial DWI this region is isointense, consistent with a chronic infarction. Other changes in signal intensity are not identified in the left hemisphere (C). PWI reveals a relatively increased CBF (RID=23%) in the left hemisphere (D). Two days later, the patient's condition deteriorates and CT demonstrates extensive hemorrhage with perilesional edema and intraventricular extension (E).

deficits and seizures after CEA to cerebral ischemia. The extended subcortical T2 hyperintensities in patients 1 and 2, which implied white matter edema, were characterized by isointensity on DWI, suggesting normal or increased diffusion of water, that is, vasogenic edema in accordance with previous studies.⁶ Unfortunately, ADC maps were not technically available during the period of our study to document increased ADC in the edematous brain regions.

A DWI study of moderately hypertensive patients with posterior leukoencephalopathy syndrome (PLES) showed a similar to our study pattern of vasogenic edema.¹⁵ In patient 1, extracellular edema also affected the contralateral to CEA occipital white matter, documenting that an identical MRI pattern may be encountered in both the HS and the PLES, and confirming that CEA influences bilaterally cerebral hemodynamics.^{3,12} Some authors suggest that blood pressure (BP) elevations and hyperperfusion are interlinked and may act synergistically as triggers for a subsequent pathogenetic cascade.¹⁶ All our patients in whom HS developed had a history of hypertension, and all had higher than normal BP during the symptoms of hyperperfusion, although in patients 2 and 4 BP increases were not striking. It is known that cerebral hyperperfusion can occur even in patients with stable and normalized blood pressure after surgery;^{1,4,17} however, cerebral hyperperfusion in the presence of BP elevations is the rule. Continuous postoperative vigilance and meticulous BP control, even in normotensive patients, is of utmost importance because CBF is pressure-dependent during the period of postoperative hyperperfusion caused by defective

cerebrovascular autoregulation.^{2,12} There is evidence that BP reduction can control symptoms and decrease MCA Vm ipsilateral to CEA, although the interaction between BP and MCA Vm is complex.² In a series,² there were patients with episodes of hypertension (>170 mm Hg systolic BP) but without symptoms of hyperperfusion and other patients with less elevated, yet abnormal BP values, in whom symptoms developed. Pharmacological control of BP did not influence MCA Vm in the former, but decreased ipsilateral MCA Vm in the latter. Strict BP control in selected patients using intra-operative TCD criteria has been associated with decreased rate of ICH after CEA.¹⁸ The "normal perfusion pressure breakthrough" hypothesis¹⁹ could explain the development of a bilateral (PLES) or unilateral (HS) "hypertensive encephalopathy" in the absence of severe hypertension. In a rat model of chronic cerebral hypoperfusion,²⁰ it has been documented a >100% increase in capillary density, with some of these capillaries becoming structurally defective in their walls. These capillaries are mechanically weaker and consequently more vulnerable to breakthrough by the distending forces that occur on reperfusion. Furthermore, using the same experimental model, it has been shown that chronic hypoperfusion may be responsible for a reduction in the threshold for hypertensive breakthrough.²¹ Whether these changes occur in humans is unknown. However, if these were true, and in combination with the loss of the appropriate vasoconstrictive response, they would account for the post-CEA vasogenic edema even in patients without severe hypertension but with relative PWI-documented hyperperfusion.

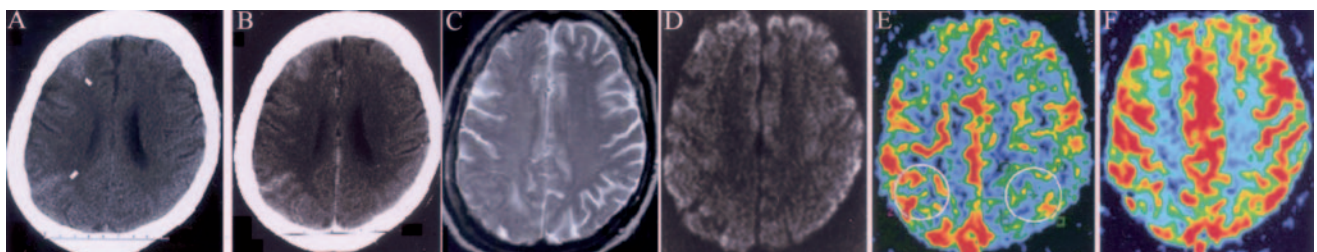


Figure 5. Patient 4. Axial nonenhanced CT, obtained 2 hours after the onset of seizures, shows right frontal and parietal sulcal effacement (edema) along with some cortical hyperdensities corresponding to petechial hemorrhages (A, white bars) and gyriform contrast enhancement (B). On T2-weighted MRI on postoperative day 15, the right hemispheric edema has mostly resolved (C). In DWI at the same level, there are no identifiable changes in signal intensity over the right hemisphere (D). PWI demonstrates a relative increase of the CBF in the right parietal lobe (RID=16%) (E). On a repeat PWI 1 month later, the previous RID has completely resolved (F).

Clinical Data of Patients 1 Through 4

Patients, Sex, Age (y)	Risk Factors	Index Stroke	Imaging (Timing From HS Onset)	HS Manifestations, BP Fluctuations, Follow-up
Patient 1, M, 76	Smoking, hypertension (140/70 mm Hg)	Right parietal infarction, subocclusive stenosis right ICA CEA after 10 wk	CT (2 h): old parietal infarction CDFI*: patent right ICA TCD (12 h): Fig. 1A, 1B CT (30 min): Fig. 2B CDFI: patent right ICA TCD (1 h): Fig. 1A, 1B MRI, MRA, DWI, PWI (12 h): Fig. 2C–2E, Fig. 1A MRI, PWI, DWI: Fig. 2F, 2G	BP soon after CEA: 150–160/80–90 mm Hg under amlodipine, enalapril 36 h postoperative: left hemianopsia, left hemiparesis, epilepsy partialis continua BP: 190–210/90–105 mm Hg. Clonazepam, labetalol. Thereafter, 145–160/60–80 mm Hg Post-operative day 8: focal seizures, left hemiplegia, stupor. BP: 210/105 mm Hg. Labetalol
Patient 2, F, 77	Hypertension (150/90)	Left posterior borderzone infarction, >95% stenosis left ICA CEA after 3 mo	TCD (6 h): Fig. 1C, 1D CT (7 h): Fig. 3A MRI, MRA, DWI, PWI (15 h): Fig. 3B–3D, Fig. 1B TCD: Fig. 1C, 1D MRI, DWI, PWI: Fig. 3E MRI, DWI, PWI: Fig. 3F	BP intraoperatively: 220/90 mm Hg, nitroprusside; thereafter, 140–155/70–80 mm Hg 48 h postoperative: simple partial seizures, right hemiplegia, coma BP: 140–155/70–80 mm Hg; clonazepam, phenytoin Postoperative day 7: unchanged neurological status, BP 130/85 mm Hg 2 mo: mild right hemiparesis, mild dysphasia, BP: 140/85 mm Hg BP soon after CEA: 150/90 mm Hg
Patient 3, F, 64	Hypertension (135–145/85–95 mm Hg), diabetes, smoking	Left posterior borderzone infarction, subocclusive stenosis left ICA Intra-arterial angiography complicated by left occipitoparietal infarction CEA after 3 mo	CT (2 h): old occipitoparietal infarction TCD (40 min): Fig. 1E, 1F MRI, MRA, DWI, PWI (6 h): Fig. 4B–4D, Fig. 1C CT (45 min): Fig. 4E TCD (12 h): Fig. 1E, 1F — —	36 h postoperative: partial seizures, global aphasia; BP: 200/110 mm Hg spontaneously normalized (140/90 mm Hg); clonazepam 48 h postoperative: recurrent seizure, no postictal deficit, BP: 130–145/80–90 mm Hg Postoperative day 5: somnolence, right hemiplegia, global aphasia, partial seizures; BP: 145/90 mm Hg; amlodipine 2 mo: mild aphasia, mild right arm paresis, BP: 140/90 mm Hg BP soon after CEA: 130–150/80–90 mm Hg
Patient 4, F, 64	Hypertension (135/80 mm Hg), obstructive cardiomyopathy, hypercholesterolemia	Right hemispheric TIA, 95% right ICA stenosis CEA after 48 h	— — CT (2 h): Fig. 5A, 5B CDFI: patent right ICA TCD (4 h): Fig. 1G, 1H MRI, DWI, PWI (7 d): fig 5C–5E, Fig. 1D MRI, DWI, PWI: Fig. 5F	24 h postoperative: mild right frontal headache; BP: 155–165/95–105 mm Hg Postoperative day 5: discharged; BP: 145/85 mm Hg Postoperative day 7: left hemiparesis, seizures; BP (on admission): 135/90 mm Hg; clonazepam, carbamazepine 1.5 mo: mild left facio-brachial paresis, BP: 130/80 mm Hg

*Color Doppler flow imaging.
M indicates male; F, female.

Summary

After CEA, partial seizures, focal neurological deficits with or without headache, and intracerebral hematomas can occur in the presence of relative hyperperfusion of the ipsilateral hemisphere, ranging from 20% to 44%. In some patients, this is accompanied by TCD evidence not suggesting absolute hyperperfusion. MCA Vm values may not accurately reflect relative interhemispheric CBF differences over the cortical convexity. Strict postoperative BP control is of paramount importance. The edema associated with the HS is reversible and demonstrates a vasogenic MRI pattern similar to that of the PLES. Finally, we suggest that reperfusion, rather than hyperperfusion, is the term that may better-encompass the whole of the hemodynamic and neuroeffector pathogenetic mechanisms implicated in this rare but always intriguing post-CEA complication.

Acknowledgments

T.K. received a grant from the "Empeirikion Foundation," Athens, Greece.

References

- Sundt TM, Sharbrough FW, Piepgras DG, Kearns TP, Messick JM, O'Fallon WM. Correlation of cerebral blood flow and electroencephalographic changes during carotid endarterectomy. With results of surgery and hemodynamics of cerebral ischemia. *Mayo Clin Proc.* 1981;56:533-543.
- Jorgensen LG, Schroeder TV. Defective cerebrovascular autoregulation after carotid endarterectomy. *Eur J Vasc Surg.* 1993;7:370-379.
- Araki CT, Babikian VL, Cantelmo NL, Johnson WC. Cerebrovascular hemodynamic changes associated with carotid endarterectomy. *J Vasc Surg.* 1991;13:854-860.
- Ogasawara K, Yukawa H, Kobayashi M, Mikami C, Konno H, Terasaki K, Inoue T, Ogawa A. Prediction and monitoring of cerebral hyperperfusion after carotid endarterectomy by using single-photon emission computerized tomography scanning. *J Neurosurg.* 2003;99:504-510.
- Breen JC, Caplan LR, DeWitt LD, Belkin M, Mackey WC, O'Donnell TP. Brain edema after carotid surgery. *Neurology.* 1996;46:175-181.
- Schaefer PW, Gonzalez RG, Hunter G, Wang B, Koroshetz WJ, Schwamm LH. Diagnostic value of apparent diffusion coefficient hyperintensity in selected patients with acute neurologic deficits. *J Neuroimaging.* 2001;11:369-380.
- Gilliard JH, Hardingham CR, Kirkpatrick PJ, Antoun NM, Freer CEL, Griffiths PD. Evaluation of carotid endarterectomy with sequential MR perfusion imaging: a preliminary report. *AJNR.* 1998;19:1747-1752.
- Viana Baptista M, Maeder P, Dewarrat A, Bogousslavsky J. Conflicting images. Case report. *Lancet.* 1998;351:414.
- Piepgras DG, Morgan MK, Sundt TM Jr., Yanagihara T, Mussman LM. Intracerebral hemorrhage after carotid endarterectomy. *J Neurosurg.* 1988;68:532-536.
- Reigel MM, Hollier LH, Sundt TM, Piepgras DG, Sharbrough FW, Cherry KJ. Cerebral hyperperfusion syndrome: a cause of neurologic dysfunction after carotid endarterectomy. *J Vasc Surg.* 1987;5:628-634.
- Schroeder T, Sillesen H, Boesen J, Laursen H, Sorensen P. Intracerebral haemorrhage after carotid endarterectomy. *Eur J Vasc Surg.* 1987;1:51-60.
- Hosoda K, Kawaguchi T, Shibata Y, Kamei M, Kidoguchi K, Koyama J, Fujita S, Tamaki N. Cerebral vasoreactivity and internal carotid artery flow help to identify patients at risk for hyperperfusion after carotid endarterectomy. *Stroke.* 2001;32:1567-1573.
- Bishop CC, Powell S, Rutt D, Browse NL. Transcranial Doppler measurement of middle cerebral artery blood flow velocity: a validation study. *Stroke.* 1986;17:913-915.
- Halsey JH, McDowell HA, Gelmon S, Morawetz RB. Blood velocity in the middle cerebral artery and regional cerebral blood flow during carotid endarterectomy. *Stroke.* 1989;20:53-58.
- Ay H, Buonanno FS, Schaefer PW, Le DA, Wang B, Gonzalez RG, Koroshetz WJ. Posterior leukoencephalopathy without severe hypertension: utility of diffusion-weighted MRI. *Neurology.* 1998;51:1369-1376.
- Naylor AR, Evans J, Thompson MM, London NJ, Abbott RJ, Cherryman G, Bell PR. Seizures after carotid endarterectomy: hyperperfusion, dysautoregulation or hypertensive encephalopathy? *Eur J Vasc Endovasc Surg.* 2003;26:39-44.
- Ascher E, Markevich N, Schutzer RW, Kallakuri S, Jacob T, Hingorani AP. Cerebral hyperperfusion syndrome after carotid endarterectomy: predictive factors and hemodynamic changes. *J Vasc Surg.* 2003;37:769-777.
- Dalman JE, Beenackers ICM, Moll FL, Leusink JA, Ackerstaff RGA. Transcranial Doppler monitoring during carotid endarterectomy helps to identify patients at risk of postoperative hyperperfusion. *Eur J Vasc Endovasc Surg.* 1999;18:222-227.
- Spetzler RF, Wilson CB, Weinstein P, Mehdorn M, Townsend J, Telles D. Normal perfusion pressure breakthrough theory. *Clin Neurosurg.* 1978;25:651-672.
- Sekhon LHS, Morgan MK, Spence I. Normal perfusion pressure breakthrough: the role of capillaries. *J Neurosurg.* 1997;86:519-524.
- Morgan MK, Johnston I, Besser M, Baines D. Cerebral arteriovenous malformations, steal, and the hypertensive breakthrough threshold. An experimental study in rats. *J Neurosurg.* 1987;66:563-567.

Postcarotid Endarterectomy Hyperperfusion or Reperfusion Syndrome

Theodoros Karapanayiotides, Reto Meuli, Gerald Devuyst, Bartlomiej Piechowski-Jozwiak, Annelise Dewarrat, Patrick Ruchat, Ludwig Von Segesser and Julien Bogousslavsky

Stroke. 2005;36:21-26; originally published online December 2, 2004;

doi: 10.1161/01.STR.0000149946.86087.e5

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Copyright © 2004 American Heart Association, Inc. All rights reserved.

Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:

<http://stroke.ahajournals.org/content/36/1/21>

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Stroke* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the [Permissions and Rights Question and Answer](#) document.

Reprints: Information about reprints can be found online at:
<http://www.lww.com/reprints>

Subscriptions: Information about subscribing to *Stroke* is online at:
<http://stroke.ahajournals.org/subscriptions/>