Cerebral hyperperfusion syndrome after carotid endarterectomy: Predictive factors and hemodynamic changes

Enrico Ascher, MD, Natalia Markevich, MD, RVT, Richard W. Schutzer, MD, Sreedhar Kallakuri, MD, Theresa Jacob, PhD, and Anil P. Hingorani, MD, Brooklyn, NY

Purpose: It is believed that cerebral hyperperfusion syndrome (CHS) is caused by loss of cerebral autoregulation resulting from chronic cerebral ischemia and that factors including increased intraoperative cerebral blood flow, ipsilateral or contralateral carotid disease, and postoperative hypertension may cause CHS. We describe our experience with CHS, which diverges from published reports.

Materials and methods: From March 2000 to February 2002 we performed 455 carotid endarterectomy (CEA) procedures in 404 patients at our institution. CHS developed 1 to 8 days (mean, 3.2 ± 2.5 days) postoperatively in 9 patients (2%), 6 women and 3 men, whose age ranged from 52 to 84 years (mean, 69 ± 8 years). Indications for surgery in 8 patients without neurologic symptoms were ipsilateral internal carotid artery (ICA) stenoses ranging from 70% to 99% (mean, 80% ± 7%); the remaining patient had an ipsilateral stroke, with good clinical recovery, 7 weeks before CEA. Only 1 patient had significant contralateral ICA stenosis (70%). However, 5 patients had undergone contralateral CEA within the previous 3 months. CHS symptoms were severe headache in 5 patients, seizures in 3 patients (1 stroke), and visual disturbance and ataxia in 1 patient. All 404 patients (455 cases) underwent intraoperative and early (2 weeks) postoperative carotid artery duplex scanning. The 9 patients with CHS also underwent carotid artery duplex scanning at the time of the neurologic event.

Results: Mean intraoperative ICA volume flow (MICAVF) in the 9 CHS cases was not significantly different from that in the other 446 cases (170 ± 47 mL/min and 182 ± 81 mL/min, respectively). However, mean ICA volume flow (481 ± 106 mL/min) and peak systolic velocity (PSV) (108 ± 33 cm/s) for the 9 CHS cases measured at onset of symptoms were higher than those for the remaining 446 cases (267 ± 87 mL/min and 80 ± 26 cm/s, respectively) (P < .01). Of the 9 patients with CHS, only 3 had systolic blood pressures more than 160 mm Hg at onset of symptoms. Severity of ipsilateral and contralateral ICA stenoses was not significantly different between the 9 CHS cases and the remaining 446 cases.

Conclusions: These data do not corroborate the common belief that CHS occurs preferentially in patients with severe ipsilateral or contralateral carotid disease, increased intraoperative cerebral perfusion, or severe hypertension. Recently performed contralateral CEA (<3 months) appears to be predictive of CHS. (J Vasc Surg 2003;37:769-77.)
contralateral ICA, postoperative hypertension, older age, and other factors were analyzed to assess their potential as predictors of CHS. Last, we investigated whether previous contralateral CEA correlated with development of CHS.

MATERIAL AND METHODS

Patient population. From March 2000 to February 2002, 455 consecutive primary CEsAs were performed in 404 patients. Patients included 221 men (55%) and 183 women (45%), with age ranging from 43 to 93 years (mean, 74 ± 9 years). Associated risk factors included hypertension in 275 patients (67%), smoking in 176 patients (44%), diabetes in 122 patients (30%), and coronary artery disease in 98 patients (24%) patients. One hundred twenty-nine primary CEsAs (28%) were performed in patients with neurologic symptoms (stroke, n = 63; transient ischemic attack, n = 53; amaurosis fugax, n = 13) with 60% or greater ipsilateral ICA stenosis at duplex ultrasound scanning. Asymptomatic ICA stenosis (≥60%) was the indication for surgery in the remaining 326 CEsAs (72%). Seventy-six patients (19%) underwent staged bilateral CEA; 51 patients were included in the study, and 25 patients had a history of contralateral CEA performed 1½ to 10S months (mean, 30 ± 23 months) before initiation of the study.

Cerebral hyperperfusion syndrome. CHS was defined as severe unilateral postoperative headache ipsilateral to the site of endarterectomy, seizures, or stroke, accompanied by increased ipsilateral ICA volume flow (>100%) compared with intraoperative values.

Surgical technique. All operations were performed with the patient under general anesthesia. Attempts were made to keep systolic blood pressure around 140 mm Hg during carotid cross-clamping, with pharmacologic manipulation when necessary. Intravenous heparin was administered before carotid artery clamping in all cases, and activated clotting time was maintained at more than 300 units throughout the procedure. Systolic ICA backpressure <50 mm Hg or history of acute neurologic event was indication for indwelling shunt placement in 152 cases (33%). External carotid artery disease was undisturbed, and plaque with its adjacent media was routinely transected at the origin of this artery.17 All carotid arteriotomies were closed with patches, of which 449 (98.7%) were synthetic (Sulzer Vascutek, Austin, Tex) and the remaining 6 (1.3%) were fashioned from a neck vein (1 facial vein; 5 external jugular veins). Heparinization was reversed with protamine sulfate in most patients at the end of the procedure, to minimize postoperative hematomas. Exceptions were made in patients with a history of insulin-dependent diabetes and in those with mean ICA volume flow less than 100 mL/min.3 Redundancy or kinking of the ICA was corrected with posterior transverse plication in 99 cases (22%) and resection in 6 cases (1.3%).18

Intraoperative duplex scanning. Completion intraoperative duplex scan was performed in all 455 consecutive CEsAs with an ATL HDI 5000 scanner operated by a single registered vascular technologist. Intraoperative CL10-5 MHz or CL15-7 MHz "hockey stick" transducers inserted in a sterile cover filled with acoustic gel were used by the vascular surgeon to insonate the common carotid artery (CCA) and its branches immediately after cross-clamp release. Our protocol included acquisition of real-time B-mode imaging in sagittal and transverse planes. In addition, all three arterial segments were subjected to color flow imaging, power Doppler scanning, and spectral waveform analysis. Indications for repeat intervention based on B-mode abnormalities were a mobile flap 2 mm or more in length in the ICA and 3 mm or more in length in the CCA, or any other technical defect that caused more than 30% reduction in luminal diameter. Residual CCA disease was an exception because of extensive atherosclerosis in many of these patients. Flaps and residual disease were undisturbed in the external carotid artery. Peak systolic velocity (PSV), end-diastolic velocity (EDV), resistive index (RI), and MICAVF were measured routinely in all segments of the carotid artery. ICA RI was automatically calculated with the duplex scanner with the formula ICA RI = (ICA PSV - ICA EDV)/ICA PSV. We elected to use MICAVF values as indicators of cerebral perfusion. MICAVF was averaged from ICA VF measurements registered three times for more than three consecutive equal cardiac cycles. Each measurement was automatically calculated with the duplex scanner with the formula ICA VF = mean ICA PSV × (ICA diameter/2) × π.19 These measurements are reproducible and correlate significantly with values registered with standard intraoperative flow meters. However, to obtain accurate and reliable volume flow rates one must use color duplex scanning, insonate a straight portion of the vessel, adjust the ultrasound beam to an approximately 60-degree angle, and use arteries 3 mm or greater in luminal diameter with sample volume size matching the lumen. Equally important is to record three to five uniform pulse cycles and calculate the mean of at least three readings. Accuracy of intraoperator and interoperator repeatability was better than 85%.20–23 In addition, Hosoda et al19 showed a significant linear correlation between ICA flow increase and cerebral blood flow in patients with decreased cerebral reactivity. Distal ICA lumen diameter was also routinely measured, and data were analyzed and compared.

Postoperative follow-up. Results of 2-week postoperative follow-up carotid duplex scanning were available for 437 of the 455 cases (96%).

Statistical analysis. Instat (GraphPad, San Diego, Calif) was used to compare degree of preoperative ICA stenosis, mean arterial blood pressure, MICAVF, ICA PSV, ICA EDV, ICA RI, and distal ICA lumen diameter between patients with CHS and those with an uneventful postoperative period. The same program was used for comparison of MICAVF, ICA PSV, ICA EDV, ICA RI, and distal ICA lumen diameter obtained intraoperatively, during CHS, after symptom relief, and 2 weeks after surgery. We reported these values as mean ± 1 SD. The Fischer exact test was used to compare incidence of CHS in patients with and without a history of contralateral CEA and with the time interval between the two operations.
Correlation between degree of ipsilateral or contralateral ICA stenosis with ipsilateral intraoperative mean ICA flow volume was performed with SPSS version 8.0 (SPSS, Chicago, Ill). The same program was used to correlate ipsilateral MICAVF with ipsilateral ICA stenosis in the presence of contralateral ICA occlusion.

RESULTS

Cerebral hyperperfusion syndrome. CHS developed in 9 patients (2%) (group 1) 1 to 8 days postoperatively (mean, 3.2 ± 2.5 days). Six patients were women and 3 patients were men, whose ages ranged from 52 to 84 years (mean, 69 ± 8 years). Female gender was not predictive of CHS (P = .3). History of hypertension was reported in 8 of these patients (89%). However, only 3 patients (33%) had measured systolic blood pressure 160 mm Hg or greater at the time of symptoms. In fact, 1 patient was receiving intravenous neosynephrine to treat systemic hypertension when severe headache developed. History of hypertension was not predictive for CHS (P = .2). Five patients had sudden onset of severe unilateral headache. No headache was associated with severe hypertension during the symptomatic phase (blood pressure 174/84, 165/76, 160/84, 156/65, and 107/47 mm Hg, respectively), photophobia, or vomiting. Nausea was reported by 3 of 5 patients. Intensity of headache was such that it led us to obtain additional hemodynamic carotid duplex scans. Clearly these were not migraine or cluster headaches. Indications for surgery in the 8 neurologically asymptomatic patients were ipsilateral ICA stenosis ranging from 70% to 99% (mean, 80% ± 7%). The remaining patient had an ipsilateral stroke, with good clinical recovery, 7 weeks before CEA. Severity of ipsilateral ICA stenosis in group 1 was not significantly different from that recorded for group 2 (remaining 446 cases), and ranged from 55% to 99% (mean, 81% ± 10%) (P = .8). Only 1 case (11%) in group 1 had contralateral ICA stenosis (70%), whereas 95 cases (21%) in group 2 had 70% or greater stenosis (P = .7). Hyperperfusion signs and symptoms included severe unilateral headache ipsilateral to the endarterectomy, which prolonged hospital stay in 4 of 5 patients; seizures in 3 patients; and ataxia with visual disturbance in 1 patient. One of the 3 patients with seizures also had stroke within the first postoperative month.

Intraoperative hemodynamic findings. Intraoperative MICAVF measurements in groups 1 and 2 (non-CHS) were not significantly different (Table I). In addition, there were no significant differences between the 2 groups relative to intraoperative ICA values for PSV, EDV, RI, and luminal diameter. CHS did not develop in any of the 23 cases with the highest mean ICA flow volume measured intraoperatively (337 to 610 mL/min; mean, 405 ± 64 mL/min).

Postoperative hemodynamic duplex findings in CHS. MICAVF, ICA PSV, and ICA luminal diameter increased significantly during symptoms of CHS and decreased significantly after symptoms subsided in the 9 patients with CHS (Table II). However, ICA RI and ICA EDV were not significantly different in these patients. Only MICAVF obtained after symptoms subsided in group 1 patients were significantly lower then values measured in group 2 in the second postoperative week (Table III).

Comparison of mean ICA flow volume and other hemodynamic parameters between patients with severe headache alone and those with other neurologic symptoms. The 5 patients with severe ipsilateral headache alone had significantly lower MICAVF during symptoms (mean, 423 ± 64 mL/min) compared with the 4 remaining patients with neurologic symptoms (mean, 557 ± 99 mL/min; P < .05). However, no statistically significant differences were found between these 2 groups of patients for ICA PSV (104 ± 26 cm/s vs 115 ± 39 cm/s), ICA EDV (25 ± 6 cm/s vs 34 ± 11 cm/s), ICA RI (0.75 ± 0.06 vs 0.7 ± 0.04), or mean blood pressure (99 ± 18 mm Hg vs 100 ± 11 mm Hg) (all P > .1).

Conversely, MICAVF results for the severe headache group were significantly higher than values obtained for the remaining patients who did not develop signs and symptoms of CHS (mean, 267 ± 87 mL/min) (P < .0001).

Correlation of MICAVF with ipsilateral and contralateral ICA disease. There was an inverse relationship between severity of ipsilateral ICA stenosis and postendarterectomy MICAVF in all 455 cases (correlation coefficient, 0.15 at 0.01 level) (Fig 1). Also, correlation between amount of ipsilateral flow volume in the ICA and severity of contralateral disease (up to 99%) was not significant (Fig 2). Furthermore, inverse correlation was observed in 29 patients with contralateral ICA occlusion between severity of ipsilateral ICA stenosis and MICAVF (coefficient, 0.44 at 0.05 level) (Fig 3).

CHS and blood pressure. Intraoperative mean systemic blood pressure for group 1 (87 ± 8 mm Hg) and group 2 (89 ± 13 mm Hg) was not significantly different (P = .65). Mean systemic blood pressure within 2 weeks after CHS symptoms had subsided in group 1 was significantly lower then that for group 2 at 2-week follow-up duplex scanning (95 ± 7 mm Hg and 102 ± 17 mm Hg, respectively; P < .02). Incidence of postoperative systolic blood pressure more than 160 mm Hg in the CHS group (22%) was not statistically different from that in the non-CHS group (34%) (P = .7). Patients with CHS had lower
Table II. Comparison of duplex findings in 9 patients with CHS obtained intraoperatively and during symptoms

<table>
<thead>
<tr>
<th>ICA parameter</th>
<th>Intraoperative</th>
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<th>After symptoms</th>
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<tr>
<td></td>
<td>Mean</td>
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<tr>
<td>Lumen (mm)</td>
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<td>0.66</td>
<td>.001</td>
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*MVF, Mean volume flow; PSV, peak systolic velocity; EDV, end-diastolic velocity; SD, standard deviation.

Table III. Comparison of postoperative duplex measurements for 9 patients with CHS after symptom relief (group 1) and 428 without CHS at 2-week follow-up examination (group 2)

<table>
<thead>
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<th>Group 2</th>
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<tr>
<td></td>
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<td>Mean</td>
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<td>RI</td>
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<td>Lumen (mm)</td>
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<td>0.6</td>
<td>.1</td>
<td>5.1</td>
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</table>

*MVF, Mean volume flow; PSV, peak systolic velocity; EDV, end-diastolic velocity; SD, standard deviation.

intraoperative systolic pressure (mean, 132 ± 11 mm Hg) compared with preoperative values (147 ± 16 mm Hg) and values obtained during hyperperfusion symptoms (148 ± 19 mm Hg) (P = .04 and P = .05, respectively). There were no significant differences in CHS patients regarding preoperative mean systemic pressure (94 ± 8 mm Hg) and systolic blood pressure (147 ± 16 mm Hg) with values obtained during hyperperfusion symptoms (100 ± 15 mm Hg and 148 ± 19 mm Hg, respectively) (P = .9).

CHS and contralateral CEA. Of the 76 bilateral CEAs, CHS developed in 5 cases (6.6%) compared with 4 of the 379 remaining cases (1.1%) (P < .01). Contralateral CEA had been performed in 5 of the 9 patients with CHS (56%) from 3 days to 67 days (mean, 39 ± 23 days). In none of the 5 patients with CHS who underwent contralateral CEA did restenosis develop during follow-up of 7 to 29 months (mean, 17 months). Of the 76 patients who underwent bilateral CEA, intraoperative duplex scans and hemodynamic data for both operations were available for review for 51. The remaining 25 patients underwent the first CEA before commencement of the present study, and no hemodynamic data were recorded. There were no significant differences in intraoperative MCAVF, ICA PSV, ICA EDV, and ICA RI values between the first and second CEA in the 51 available cases (Table IV). Of these 51 patients, 47 did not have CHS and 4 did; the remaining patient with CHS underwent the initial CEA before onset of this study. No significant intraoperative hemodynamic differences were observed for the first and second CEA between the latter two groups (Tables V and VI). In addition, we performed an independent analysis of data for the 35 patients who underwent bilateral CEA within 3 months. Intraoperative hemodynamic data were available for 33 patients because 2 patients underwent the initial CEA before the start of this study. Similar intraoperative hemodynamic findings were observed for the first CEA for the 29 patients without CHS and the 4 patients with CHS (Table VII) and for the second CEA for the 30 patients without CHS and the 5 patients with CHS (Table VIII).

Patients who underwent contralateral CEA within the previous 3 months were at a significantly higher risk for CHS after the second operation (14.3%) than were patients with longer intervals (>3 months) between CEAs (0%) (P < .02).

Technical defects. No technical defects were identified at B-mode imaging or waveform analysis in group 1. Group 2 patients underwent 9 intraoperative revisions (2%). Three partially thrombosed patches, 3 mobile flaps in the CCA longer than 5 mm that were causing more than 60% diameter reduction, and 1 ICA periadventitial intraluminal flap were repaired with patch incision and flap removal. Also, 2 ICA stenoses were repaired with a larger patch. Three tacking sutures used to repair an intimal flap caused one of these defects, and the other occurred in a redundant ICA treated with posterior transverse plication. All 9 technical defects were successfully revised, and B-mode images and spectral waveform analysis at completion duplex studies were normal.

Postoperative neurologic morbidity and mortality. One stroke (13%) occurred in group 1 patients, and 3 strokes were documented in group 2 patients. The patient in group 1 had seizures 1 week postoperatively, with no duplex scan evidence of technical defects at the operative site and with no computed tomography or magnetic resonance imaging findings suggestive of stroke. However, edema and signs of hyperperfusion were noted on the magnetic resonance images (Fig 4). The symptoms subsided in 2 days, and the patient was discharged home. Intraoperative MCAVF of 178 mL/min increased to 632 mL/min during the period of seizures, and decreased to 141 mL/min after symptoms subsided. No technical defects were observed on the postoperative duplex scans. One week later this patient was readmitted to a nearby hospital with stroke. Of the 4 strokes in this series, 3 were not accompanied by increased ICA flow volume. One of these
patients had had a recent stroke (<2 weeks) and critical ipsilateral ICA stenosis, and neurologic symptoms worsened postoperatively without evidence of CHS or intracerebral hemorrhage. The 2 additional ischemic strokes occurred in patients with low intraoperative mean ICA flow volume (<100 mL/min) in whom protamine sulphate was given to reverse the heparin effect. Neither patient had clinical or hemodynamic evidence of CHS. All 4 postoperative strokes (≤30 days) contributed to a 0.9% overall stroke rate for the 455 CEAs. The overall 30-day mortality rate was 1% (4 patients). The combined stroke and mortality rate was 1.7% (7 of 404 patients).
DISCUSSION

CHS occurs in approximately 0.4% to 7.7% of patients undergoing CEA. This wide range reflects, in part, differences in study inclusion clinical criteria and sample size. For example, headache occurs in as many as 62% of patients. However, these headaches are classified as mild to moderate in most patients (78%). Sudden onset of severe unilateral headache ipsilateral to the endarterectomy site was documented in only 5 patients in this study. In addition, most studies have not confirmed the diagnosis of CHS with cerebral hemodynamic data. Thus the true incidence of CHS remains elusive, inasmuch as other causes may be involved in development of postoperative headache, seizures, and hemorrhagic stroke. In the present series we matched clinical symptoms with hemodynamic evidence of cerebral hyperperfusion, and incidence of CHS was 2%.

Significant increase in MICAVF (mean, 209%) was documented in all of our patients with CHS during the symptomatic period compared with intraoperative values ($P < .02$). The former values significantly decreased after symptoms subsided. MICAVF also correlated with intensity of postoperative symptoms; patients with severe headache had lower MICAVF than did those with more severe neurologic symptoms. Although we recognize that a potential weakness of this study is absence of cerebral blood flow (CBF) measurements, it is logical to assume they should correlate with MICAVF. If total intracerebral blood flow rates are increased, all components of the circle of Willis may be challenged. However, if only a segment of the brain requires increased blood flow, the ipsilateral feeding vessels would be primarily responsible for this demand. To our knowledge, CBF measurements were never recorded in patients with CHS during the symptomatic phase. Rather, CBF values have been used as predictors of CHS. Reigel et al., using intraoperative xenon-labeled CBF measurements, demonstrated that patients with increased intraop-

Fig 3. Correlation between severity of ipsilateral ICA stenosis and intraoperative mean internal carotid artery flow volume (MICAVF) in presence of contralateral ICA occlusion (29 cases) ($R = -0.44$ at 0.05 level).

| Table IV. Comparison of intraoperative hemodynamic duplex measurements for first and second surgeries in 51 patients who underwent bilateral CEA |
|---------------------------------|------------------|------------------|------------------|------------------|
|                                 | First CEA | Mean | SD | P | Second CEA | Mean | SD | P |
| MVF (mL/min)                   | 183      | 66   | 1.0| 183 | 72         |
| PSV (cm/s)                     | 68       | 31   | .06| 81  | 37         |
| EDV (cm/s)                     | 22       | 12   | .1 | 26  | 14         |
| RI                             | 0.67     | 0.1  | 1.0| 0.67| 0.1        |

$MVF$, Mean volume flow; $PSV$, peak systolic velocity; $EDV$, end-diastolic velocity; $SD$, standard deviation.

| Table V. Comparison of intraoperative hemodynamic duplex measurements during first CEA for available 47 patients without CHS and 4 patients with CHS who underwent bilateral CEA |
|---------------------------------|------------------|------------------|------------------|------------------|
|                                 | Non-CHS | Mean | SD | P | CHS | Mean | SD | P |
| MVF (mL/min)                   | 179     | 73   | .2 | 178 | 39     |
| PSV (cm/s)                     | 66      | 34   | .5 | 62  | 7      |
| EDV (cm/s)                     | 22      | 12   | .5 | 18  | 6      |
| RI                             | 0.66    | 0.1  | .3 | 0.71| 0.1    |

$MVF$, Mean volume flow; $PSV$, peak systolic velocity; $EDV$, end-diastolic velocity; $SD$, standard deviation.
operative CBF ratio (before and after CEA) were more likely to have CHS. More recently, other authors from the same institution reported a significant correlation between intracerebral hemorrhage after CEA and the difference in CBF ratios. These are interesting observations that should be further investigated, because if confirmed they may be used in selection of patients who require closer postoperative follow-up and possibly longer hospitalization. However, correlation of increased CBF in patients with CHS remains to be proved.

Intraoperative ICA flow volume in patients with contralateral ICA occlusion may be influenced by collateral flow and pressure originating from the vertebral arteries or even from the contralateral external carotid artery. In these patients we recorded lower intraoperative mean ICA flow volume values and a negative correlation with ICA back-pressure. However, collateral flow is not a stable phenomenon and may vary as flow is restored via the repaired principal channel. As expected, the direction of flow reverses and the collateral branches tend to diminish in size and number. It is probable that such changes occur over hours, thus eliminating backflow and pressure as a permanent source of resistance to ICA flow volume.

We were surprised to find that intraoperative hemodynamic parameters were not significantly different between patients in whom CHS developed and those who remained asymptomatic. This observation casts doubt on the perceived complete loss of autoregulatory mechanisms as the etiologic factor in CHS. If the intracerebral vessels were maximally vasodilated, higher intraoperative MICAVF would be expected in patients with CHS compared with those without CHS. In addition, this potential autoregulatory vasoparalysis precipitated by the presence of chronic low flow, as in severe ipsilateral ICA stenosis or severe contralateral ICA disease, was not confirmed. Our data show an inverse relationship between severity of ipsilateral ICA stenosis and postendarterectomy MICAVF. Conversely, none of the 23 patients with the highest intraoperative MICAVF (mean, 405 mL/min) in the present study had CHS.

Control of postoperative hypertension is widely believed to be the mainstay for prevention of CHS. When the reflex contraction capability of intracerebral arteries is lost, increased systemic blood pressure may place undue burden on these dilated vessels that may lead to hemorrhage. However, Pomposelli et al documented postoperative blood pressure greater than 180 mm Hg in only 5 of 11 patients with intracranial hemorrhage after CEA. Haisa et al reported one case of hyperperfusion leading to intracerebral bleed despite strictly controlled blood pressure. Bernstein et al also reported their experience with one normotensive patient who had CHS and intracranial hemorrhage while maintaining normal systemic blood pressure after endarterectomy. In our series, only 3 of 9 patients with CHS had postoperative systolic blood pressure 160 mm Hg or greater. Thus, not only are we unable to support the concept that hypertension is a major contributor to development of CHS, but there is no conclusive evidence in the literature to link these two conditions.

In this study we analyzed several other potential risk factors for CHS. One predictive factor was history of contralateral CEA. Of the 76 cases with bilateral CEA, CHS developed in 6.6% compared with only 1.1% of the 379 remaining cases (P < .01). Moreover, the interval between the two operations was also a significant predictor of CHS. Incidence of CHS was 14.3% when the interval between operations was less than 3 months, and was 0% with longer intervals between operations (P > .02).

Ille et al described 1 patient who experienced CHS after bilateral CEAs performed 3 weeks apart. They believe that severe hypertension with blood pressure reaching
230/120 mm Hg was the leading cause of postoperative seizures, and hypothesized that dysfunction of carotid baroreceptors after bilateral endarterectomy precipitated intermittent hypertensive episodes. Bove et al27 showed unusual baroreceptor reactivity as a consequence of previous contralateral CEA. Of their patients who exhibited postoperative hypotension, 29% underwent bilateral CEA, and only 9% of the normotensive group had bilateral operations ($P > .05$). Although inconsistencies in baroreceptor behavior may be the causative factor for CHS, much remains to be studied in this area. The influence of contralateral CEA, particularly when performed within a relatively short interval (3 months), on development of CHS also deserves further investigation. We were unable to identify any significant differences in clinical or hemodynamic parameters that distinguished patients who underwent unilateral versus bilateral CEA and CHS. Conversely, Darling et al28 reported on safety of performing bilateral CEA within a short interval (1-4 days) in 204 patients. However, they fail to mention the incidence of CHS or the number of patients lost to follow-up. Increased awareness of this syndrome may permit more thorough appreciation of CHS and its predictive factors.

Although transitory severe headache may impose limited burden for the postoperative patient, seizures and stroke may not only be associated with significant physical and mental disability but also with death. The relative infrequency of CHS appears to be the main reason why this entity is poorly understood. Consequently, we believe that conclusive data can only be obtained from large multicenter prospective studies, particularly if a treatment protocol is to be evaluated. In the meanwhile, we remain cautious about performing bilateral CEA within a short interval (<3 months).

**REFERENCES**


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