Brain Perfusion Imaging during Preoperative Temporary Balloon Occlusion of the Internal Carotid Artery

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The main objective of this study was to assess whether ^{99m}Tc-HMPAO brain SPECT imaging can identify patients at high risk of developing an infarct following permanent carotid occlusion in the course of brain surgery. **Methods:** Test balloon occlusion of the internal carotid artery was performed in 44 patients with a variety of head and neck tumors or aneurysms. Technetium-99m-HMPAO was injected intravenously while the balloon was inflated and a SPECT study was obtained 30 min later. Follow-up CT scans were obtained routinely for all patients at 2 wk and 1 mo following surgery, or earlier when necessary. Thirty patients and five normal volunteers had semiquantitative analysis of cerebral perfusion. **Results:** Twenty-six patients demonstrated ipsilateral perfusion abnormalities during trial occlusion. Eight patients in this group underwent bypass grafting prior to sacrifice of the artery: two resulting in infarcts. Eighteen patients had symmetric cerebral perfusion during occlusion and four of these patients underwent permanent therapeutic carotid occlusion; three patients had subsequent infarcts and the fourth patient had an impending stroke. **Conclusion:** Patients with symmetric cerebral perfusion measured by ^{99m}Tc-HMPAO SPECT may still have a high long-term complication rate following carotid sacrifice. The scan findings in these patients were not predictive of the outcome. Patients with asymmetric cerebral perfusion had alternative therapeutic approach to carotid sacrifice and most of them had good surgical outcomes.

Key Words: technetium-99m-HMPAO; cerebral perfusion; carotid artery; test occlusion

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Patients with aneurysms and various tumors at the base of the skull often require temporary or even permanent intraoperative occlusion of the internal carotid artery (ICA), especially if the artery is suspected to be infiltrated or encased by the tumor. The complication rate of permanent carotid occlusion, however, is relatively high. It is therefore essential to identify, preoperatively, those patients who will tolerate the carotid sacrifice with minimal risk of developing neurologic deficits (*1*). Commonly used techniques such as electrophysiological monitoring, measurement of artery stump pressure changes or monitoring the development of neurologic deficits during temporary balloon occlusion of the ICA appear to be inadequate in a significant number of patients (*2–3*).

Technetium-99m-hexamethylpropylene-amine oxime (HMPAO) SPECT imaging is a well-established method for detection of perfusion abnormalities in patients with and without neurologic deficits during balloon test occlusion (*4–5*). Controversy, however, exists regarding the predictive value of this test for patients who undergo permanent carotid occlusion (*6*). We compared the results of cerebral perfusion studies using ^{99m}Tc-HMPAO SPECT and neurologic symptoms during test occlusion of the ICA with prospective clinical evaluation and postoperative follow-up with CT scans. The purpose of the study was to evaluate whether brain SPECT can predict neurologic deficits, following permanent carotid occlusion or other surgical manipulations of the artery.

Semiquantitative analysis of brain SPECT perfusion images was also tested and compared to visual analysis alone for prediction of neurologic events. This approach provides a more objective tool to support the visual impression, especially in mild perfusion abnormalities.

MATERIALS AND METHODS

Patients

Forty-four patients (24 women, 20 men; mean age 49 yr; range 19–79 yr) underwent balloon test occlusion of the ICA for risk evaluation prior to ICA manipulation or permanent ICA occlusion. Five of these patients were included in a smaller series reported previously by Palestro et al. (*5*) but had a longer follow-up period in our study. Fifteen of the 44 patients also had a baseline study performed prior to the occlusion study. A normal occlusion scan or diffusely diminished perfusion in the occluded hemisphere and no prior history of neurologic deficits were usually considered adequate for preoperative evaluation and planning. When a baseline study was obtained before the occlusion study, the time interval between the studies was 24–72 hr.

Patients who underwent surgery also had follow-up for 2 mo to 3 yr after surgery. A CT scan was obtained at 2 wk and 1 mo after surgery. Table 1 describes the diagnoses of these patients, which includes 38 patients with neoplasms at the base of the brain, 4 vascular lesions of the ICA and 2 traumatic lesions.

Prior to the balloon occlusion, each patient underwent three- or four-vessel cerebral angiography through a femoral puncture to evaluate the patency of the circle of Willis. A loading dose of 5000 units of heparin was given intravenously. A 5 french polyethylene balloon catheter was advanced into the petrous portion of the ICA and inflated until total occlusion (left side occlusion, n = 19 and right side occlusion, n = 25). Clin[illegible] examination was performed repeatedly during the 20 min in [illegible] the balloon was occluding the artery. The neurosurgeons evaluated cognitive skills representative of the occluded side, cranial nerves, motor and sensory skills and any change in mental status. Each patient was injected intravenously with approximately 740 MBq (20 mCi) ^{99m}Tc-HMPAO 10 min after the arterial occlusion which was maintained for an additional 10 min after tracer injection. Imaging was accomplished after deflation of the balloon, usually within 30–40 min following the injection. Prior to October 1993, brain perfusion SPECT imaging was performed with a Tomomatic 564 scanner (Medimatic Corp., Denmark) using a high-resolution collimator obtaining eight transaxial slices. Each projection was filtered and backprojected into an image matrix of 64 × 64 pixels. The pixel

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TABLE 1
Diagnoses of Patients Undergoing Balloon Test Occlusion of the Internal Carotid Artery

Diagnosis	No. of patients
Neoplastic lesions	n = 38
Neuroblastoma	1
Squamous-cell carcinoma	4
Pituitary adenoma	7
Spindle-cell tumor	1
Cholesteatoma	1
Meningioma	9
Chondrosarcoma	1
Rhabdomyosarcoma	1
Chondroid chondroma	6
Adenoid cystic carcinoma	1
Mucoepidermoid carcinoma	1
Cylindroma	1
Plasmacytoma	1
Schwanoma	1
Malignant salivary gland carcinoma	1
Histiocytic granuloma	1
Vascular lesions	n = 4
ICA aneurysm	3
Traumatic pseudoaneurysm	1
Others	n = 2
Gun shot injury	2
Total	n = 44

size was 3.2 mm. The convolution filter was a Gaussian-shaped amplitude function. Detector sensitivity correction was also performed. Attenuation correction was performed based on an automated ellipse determination with a constant linear attenuation coefficient of 0.11 cm^{-1}. After October 1993, SPECT was performed using a dual-head gamma camera (Vision T22, Summit Nuclear, Twinsburg, OH) equipped with a low-energy, high-resolution collimator. A 20% window was centered on the 140 keV photopeak of ^{99m}Tc. Ninety-six frames of 30 sec were acquired using elliptical contour rotation mode into a 64 × 64 image matrix. The images were prefiltered using a Butterworth filter (cutoff frequency = 0.2 cycles/cm; power factor = 5). Transaxial, coronal and sagittal slices 1 pixel thick (3.5 mm) were reconstructed.

The SPECT images were analyzed both visually and quantitatively. For visual analysis, two experienced nuclear physicians ranked the SPECT abnormalities as mild, moderate or severe based on the color codes of the computer. Cerebral perfusion abnormalities were classified as focal or diffuse. In case of asymmetric perfusion, the activity in the normal hemisphere was first normalized to the maximal intensity color code and then compared to the contralateral side. Mild abnormality was considered when the color code was one level below the normal area. Moderate abnormality—two levels below the normal and severe abnormality—three levels below normal (an image display with four major color codes was selected for evaluation).

Computer-assisted sector analysis of brain perfusion was performed on 30 of the 44 patients and on 5 normal volunteers. The operators performing the quantitation were blinded to the reading of the visual analysis. Five scans were performed on normal volunteers with no prior history of neurologic abnormalities and used for comparison. Six representative transaxial slices parallel to the orbitomeatal line were selected and six symmetric pairs of regions of interest (ROIs) were placed on both sides of the cerebral cortices, based on 30° angular separation (Fig. 1). The ROIs for the outer edge of the cortex were defined using a threshold of a 45% change in the maximal ^{99m}Tc-HMPAO counts between the cortex and the surrounding tissue (*7*). The inner edge of the cortex was defined as a concentric ellipse with axis lengths that were 67% of the distance from the center of the slice to its outer edge. Manually drawn ROIs were used to define the head of the caudate nuclei and the thalami. The ratio of the average number of counts per pixel in the lesion to the average number of counts per pixel in the contralateral nonaffected area was obtained for all patients and compared to the corresponding mean segmental ratio in the normal volunteers, after normalizing to the maximum cerebeller activity. The significance of the difference was tested by Student's t-test.

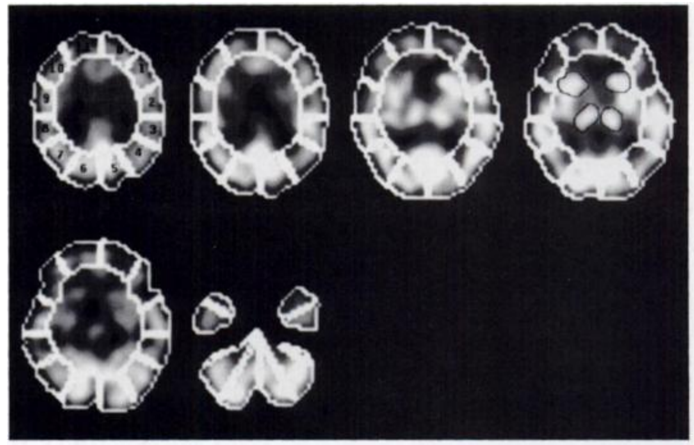

FIGURE 1. Six representative transaxial slices parallel to the orbitomeatal line with placement of ROIs and numbered sectors for semiquantitative analysis.

No rigid quantitative criteria are available to define the degree of perfusion abnormalities (*8–9*). It is generally accepted that areas of diminished perfusion are abnormal when the interhemispheric differential activity is greater than 10% (*8*). We therefore defined mild hypoperfusion as an area with ^{99m}Tc-HMPAO uptake of 10%–19% less than the contralateral identical ROI whereas moderate and severe hypoperfusion was defined as diminished perfusion of 20%–29% and ≥30% compared to the contralateral ROI, respectively.

RESULTS

All the baseline studies (15/44 patients) were performed prior to the occlusion test and showed normal symmetric perfusion pattern. Twenty-six of the 44 patients demonstrated ipsilateral perfusion abnormalities during trial occlusion. Seven of them were categorized by visual analysis as having mild perfusion abnormalities, 14 as moderate abnormalities and 5 as severe abnormalities (Table 2). Sixteen of the 26 patients (62%) showed diffuse abnormalities in the involved hemisphere while 10 patients (38%) had more focal abnormalities. Only 3 of the 26 patients (11.5%) developed neurologic symptoms during occlusion (aphasia and change in mental status in two patients

TABLE 2
Correlation of SPECT, Occlusion Symptoms and Surgical Outcome

SPECT (n = 44)	Occlusion symptoms (n = 5)	Ligation (n = 4)	Bypass (n = 10)	Resection* (n = 30)	Infarct (n = 6)
Abnormal (n = 26)	3		8 (2)†	18	2
Mild (n = 7)				7	
Moderate (n = 14)	1		5 (1)	9	1
Severe (n = 5)	2		3 (1)	2	1
Normal (n = 18)	2	4 (3)	2 (1)	12	4

*Resection of tumor without ICA sacrifice.
†Numbers in parentheses indicate infarcts.

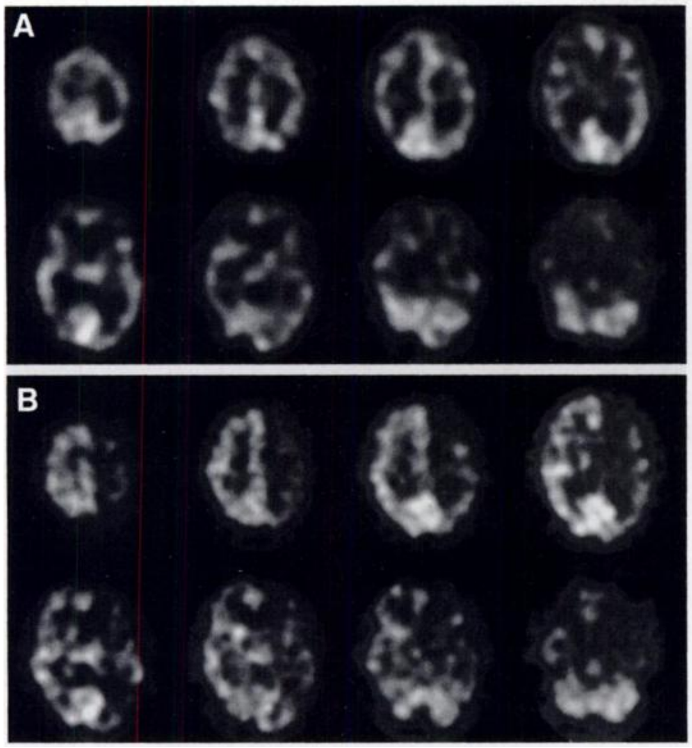

FIGURE 2. A patient with a right ICA aneurysm. Baseline study (A) shows symmetric perfusion to the brain. Images obtained during balloon test occlusion of the right ICA (B) show decreased perfusion in the entire right hemisphere. The patient underwent carotid bypass surgery without complications. (The right side of the brain corresponds to viewer's right.)

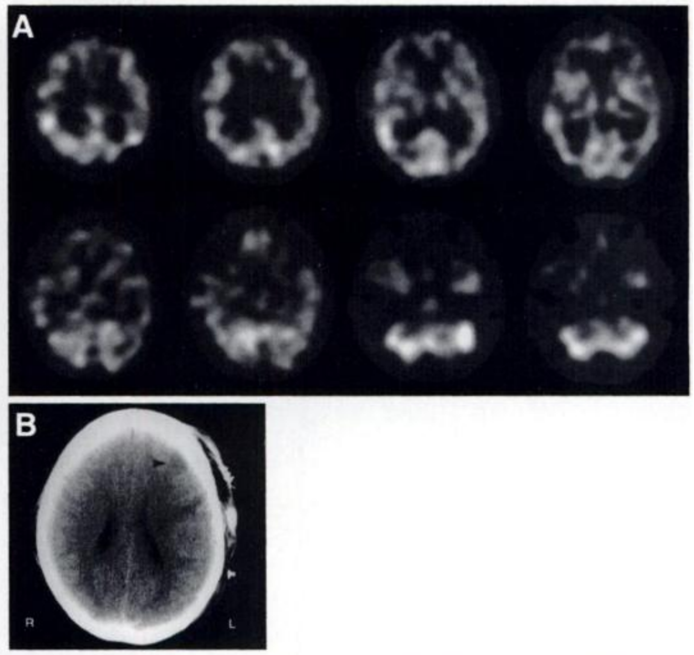

FIGURE 3. A 25-yr-old woman with rhabdomyosarcoma in the right infratemporal fossa. The occlusion study shows symmetric perfusion in both hemispheres (A). CT scan performed 1 wk after ICA ligation (B) shows an infarct in the left frontal watershed area (arrow). The patient remained asymptomatic.

with diffuse left-sided perfusion abnormalities and visual changes in a patient with diffuse abnormalities in the right hemisphere). Symptoms occurred only in patients with moderate to severe perfusion abnormalities. The treatment plan was modified in all the patients with abnormal perfusion. Eight patients underwent extracranial to intracranial (ECIC) vascular bypass grafting prior to sacrifice of the artery: two resulting in infarcts and hypodense lesions on the corresponding CT scans, due to occlusion of the graft, and the other six had no complications (Fig. 2). Eighteen patients underwent only carotid exposure and resection of the tumor without complications.

Eighteen patients had symmetric cerebral perfusion during occlusion. One of them developed a headache during the balloon test occlusion, which was not attributed to the occlusion. Four patients underwent permanent therapeutic carotid occlusion without revascularization; one patient developed a right middle cerebral artery infarct which was evident on the CT scan on the fifth postoperative day (although the patient did not develop hemiparesis). Another patient developed an acute left hemiparesis 24 hr postligation and required an emergency ICA bypass with a vein graft. Consequently, the hemiparesis resolved and no infarct was detected on follow-up CT scans. The other two patients developed a watershed infarct (Fig. 3) and a lacunar infarct 1 wk and 1 mo after ICA sacrifice, respectively. Two patients underwent revascularization with a vascular graft prior to carotid ligation, one without complications and the other resulting in an infarct, most likely related to an hypercoagulable state. The other twelve patients underwent tumor resection without sacrifice of the artery and had no complications. The routine follow-up CT scans were normal in those patients without infarcts after surgery.

Semiquantitative Analysis. The visual analysis classified 15 of the SPECT images as abnormal, and 15 of the images were normal. All patients with perfusion abnormalities (15/15) showed significantly reduced perfusion in the affected segments (>10% reduction in flow compared to the mean normal values in the controls) ($p < 0.01$). The severity of perfusion abnormalities was congruent with the percent reduction in flow in 12/15 patients, using the previously described scale of abnormalities (Table 3). In three patients the visual analysis mildly overestimated the reduction in flow compared to the quantitative analysis. Nevertheless, one of the fifteen scans read as normal by visual analysis showed retrospectively a 13% reduction in flow to the right temporal area. Although this patient underwent bypass surgery prior to carotid resection, he sustained a large right temporal infarct following surgery.

In summary, six patients developed an infarct after surgery (after ligation of the ICA in three and after bypass graft surgery in the remainder). In the first group, one infarct was related to surgical complications, and in the latter group two infarcts were related to surgical complications and one attributed to a hypercoagulable state. Four of the six patients with infarcts had a normal occlusion scan. The other two patients had moderate and severe perfusion abnormalities during occlusion.

TABLE 3
Comparison of Visual and Semiquantitative Analysis of Perfusion Abnormalities on Brain SPECT

		Visual analysis (n = 30)			
		Symmetric perfusion (n = 15)	Asymmetric perfusion: Mild (n = 5)	Moderate (n = 7)	Severe (n = 3)
Semiquantitative analysis (n = 30)	Symmetric perfusion	14			
	Asymmetry*				
	Mild	1	5	1	
	Moderate			6	2
	Severe				1

*$p < 0.01$.

DISCUSSION

Patients with tumors at the base of the skull or aneurysms of the carotid artery undergo surgical interventions that may require permanent sacrifice of the ICA. Inadequate collateral circulation at the circle of Willis, after permanent carotid occlusion, may lead to infarction due to hypoperfusion and ischemia.

Resting regional cerebral blood flow has limited value in the assessment of cerebral vascular disease or cerebral vascular reserve. Stressing the vascular flow capacity of the brain circulation by temporary occlusion of the ICA may identify patients at high risk of developing neurological complications following carotid ligation.

Stable xenon CT can measure absolute regional cerebral blood flow (*10*). The patient, however, needs to be transported to the CT scanner with the catheter in place followed by reinflation of the balloon without angiographic guidance. Overinflation of the balloon may cause intimal injury and carotid dissection (*11*). PET with ^{15}O-water is also capable of measuring absolute regional cerebral blood flow, but this technique is not widely available and is also difficult to perform in neurosurgical patients.

At the beginning of the century, Matas (*12*) used manual compression of the carotid artery with clinical monitoring to predict the risk of carotid ligation. The duration and efficiency of compression were probably inadequate to produce a reliable response. Furthermore, monitoring the neurological status of the patient is unreliable in predicting the tolerance to carotid ligation, as was noted in our series and others (*13*). Twenty-two of the 39 asymptomatic patients in our study had an abnormal perfusion scan. Since a significant decrease in blood flow is required before a neurological deficit develops, the reduction in blood flow during temporary carotid occlusion may be below the threshold for clinical symptoms. By contrast, it was found in baboons (*14*) that following carotid ligation there is marked reduction of the responsiveness and tolerance of the carotid circulation to changes in blood oxygen saturation and blood pressure. This may explain transient signs of cerebral ischemia postligation or even delayed neurological deficit following changes in blood gases or blood pressure postoperatively.

EEG is performed frequently to extend the clinical evaluation. Abnormal clinical examination may occur in the absence of EEG changes and EEG abnormalities may occur in the absence of ischemia (*1–2*). Another parameter to monitor is ICA stump pressure. Barker et al. (*15*) measured ICA stump pressure changes during temporary ICA occlusion and found a significant overlap among the groups studied. Transcranial doppler can evaluate cerebral artery velocity and pulsatility but requires a high degree of skill and cannot be applied to all patients (*16*).

Technetium-99m-HMPAO is a widely available radiotracer that has been used extensively for evaluation of regional cerebral blood flow (*17–18*). It shows rapid brain uptake and distributes proportionally to regional blood flow (*19–20*). The tracer is converted intracellularly to a hydrophilic compound and remains fixed in the brain for a prolonged time, allowing delayed imaging after injection. Technetium-99m-HMPAO scintigraphy reflects blood flow during the first few minutes of test occlusion. The timing of the injection may affect the development of perfusion abnormalities significantly. Measurements of mean arterial pressures in the distal occluded carotid artery suggests that it may take time for collateral perfusion to develop and immediate injection after occlusion is probably not advisable (*13*).

SPECT perfusion imaging was shown in our study to be more sensitive than occlusion symptoms alone. The use of two different types of camera systems had no significant influence on the results since both systems have similarly high resolution and identical quantitation software was applied to all images. Nevertheless, four patients with symmetric cerebral perfusion during balloon test occlusion developed early or delayed infarcts. Although several studies reported a strong predictive value for balloon test occlusion with SPECT (*4,13*), other reports showed 20%–22% of infarction following carotid ligation in patients with normal occlusion SPECT (*1–2*). Palestro et al. (*5*) found a 100% predictive value of a negative study in 14 patients who underwent carotid occlusion. Nevertheless, 11 of the patients only had temporary occlusion of the artery during surgery and lack of symptoms in these patients would not necessarily predict the absence of long term complications following permanent occlusion. In addition, the follow-up of the three patients who underwent permanent carotid occlusion was too short (24–48 hr postligation) to reveal delayed neurological effects. Peterman et al. (*21*) and Simonson et al. (*13*) reported one watershed infarct among five patients and one hemiparesis among eight patients with symmetric occlusion scan following permanent carotid occlusion, respectively. Origitano et al. (*22*) used a combination of perfusion SPECT and ^{133}Xe cerebral blood flow studies and found delayed ischemic complications in 4 of 18 patients who underwent permanent carotid occlusion.

Symmetric cerebral perfusion usually indicates adequate functional reserve at the time of occlusion. After complicated carotid surgery, the cerebral circulation becomes more vulnerable to changes in blood pressure and arterial oxygen tension which may develop in the postoperative period and lead to hypoperfusion and ischemia. Another mechanism is ischemia secondary to an embolus originating from the stump of the occluded artery, or at a site distal to the occlusion. The injection of heparin after balloon test occlusion may reduce the risk of emboli. Test occlusion cannot predict this late occurrence of thromboembolism, which is a significant cause of delayed cerebral ischemia after ICA sacrifice (*23*), and may explain the delayed infarcts that occurred in our patients. Our results also show that revascularization procedures carry a certain risk. Graft occlusion is the most common complication with patency rates reported from 66% to 95% (*24*). Most of the causes of graft occlusion are technical and can be improved by experience and careful intraoperative monitoring.

CONCLUSION

Despite improvement in image resolution and quantitation techniques, prediction of complications in patients undergoing permanent carotid occlusion is still dangerous, secondary to the surgery itself, with or without symmetric perfusion on a brain SPECT. Although the risk related to carotid ligation remains high, even after a negative balloon test occlusion, ^{99m}Tc-HMPAO imaging can show clinically silent areas of decreased perfusion which indicate inadequate cerebral collaterals and mandate bypass surgery prior to permanent occlusion or limit the surgery to temporary occlusion.

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EDITORIAL

Temporary Balloon Occlusion, SPECT and Carotid Artery Sacrifice

One problem that still concerns the practice of medicine is the ability to predict whether or not a patient can safely tolerate sacrifice of an internal carotid artery. Advances in neurosurgical techniques now permit the removal of an increasingly complex array of intracranial neoplasms and vascular abnormalities. Because these procedures often require permanent occlusion or sacrifice of an internal carotid artery, preoperative knowledge of whether or not a patient can safely tolerate such a procedure can have a significant impact on surgical planning.

Lorberboym et al.'s article (*1*) reminds us that the identification of those individuals at risk for neurologic complications following ICA occlusion remains an elusive goal. Part of the problem in identifying high risk patients is that postocclusion complications may be due not only to hemodynamic insufficiency, but also to emboli, vasospasm and even radiation vasculopathy (*2*). There are a plethora of tests currently available that can be used to assess the ability of a given individual to tolerate permanent occlusion of the ICA: carotid artery stump pressures, contrast angiography, xenon (both radionuclide and stable CT) cerebral flow studies, PET and SPECT (*3–15*). These procedures are typically performed in conjunction with temporary balloon occlusion of the ICA and focus on only one aspect of this very complex issue: cerebral vascular reserve. They offer no information about the likelihood that an individual will develop complications secondary to anything other than cerebral vascular insufficiency.

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Normal cerebral blood flow (CBF) is approximately 55 ml/100 g/min. Neuronal dysfunction and cerebral ischemia develop when CBF drops below about 20 ml/100 g/min with cerebral infarction occurring when CBF falls below 10 ml/100 g/min. The appearance of a neurologic deficit during balloon occlusion of the ICA, with simultaneous neurologic examination, indicates that CBF has fallen below the critical threshold necessary to maintain neuronal function and is considered unequivocal evidence that the patient will not tolerate ICA occlusion. Unfortunately the failure to develop neurologic defects during temporary balloon occlusion does not ensure the safety of the procedure and stroke rates of up to 20% following a negative temporary balloon occlusion have been reported (*9,16*). It is generally assumed that the limitation to temporary balloon occlusion with clinical examination is that this procedure fails to identify patients with marginal CBF, i.e., less than 55 ml/100 g/min, but more than 20 ml/100 g/min. Under ordinary steady state conditions in patients with marginal cerebral vascular reserve CBF remains above 20 ml/100 g/min. Any condition that results in a decrease in systolic blood pressure could cause CBF to fall below the critical threshold with subsequent cerebral ischemia or infarction. It is on the identification of those patients with marginal cerebral blood flow that virtually all preoperative investigations currently focus.

Technetium-99m HMPAO cerebral perfusion SPECT imaging with the general availability of both the equipment and the tracer, as well as the fact that a patient need undergo balloon occlusion only once are attractive features of this technique, which according to several reports, may be a useful adjunct to temporary balloon occlusion (*9–15*). The results reported by Lorberboym et al. (*1*) suggest, however, that a reassessment of the procedure is in order. A meaningful comparison of these results to those previously reported would have to focus on the patient population, postoperative follow-up, complications and technique.

PATIENT POPULATION

CBF and cerebral vascular reserve are intimately related to the systemic hemodynamic status of a patient. There are numerous factors that affect systemic hemodynamics directly, and, therefore, cerebral hemodynamics indirectly. For example, age and pre-existing medical conditions are just two factors that can affect a patient's ability to respond to hemodynamic stress. Intraoperative complications, such as, an adverse reaction to anesthesia or profound blood loss can also impede cerebral blood flow. Finally postoperative complications (pulmonary emboli and sepsis) can also lead to cere-