Middle Cerebral Artery Perfusion Pressure in Cerebrovascular Occlusive Disease

ROBERT F. SPETZLER, M.D., RICHARD A. ROSKI, M.D.,* AND JOSEPH ZABRAMSKI, M.D.†

SUMMARY We measured the MCAP (middle cerebral artery pressure)/MSBP (mean systemic blood pressure) ratio in 76 patients who underwent an EIAB (extracranial-intracranial arterial bypass). Patients were divided into groups on the basis of angiographic findings. We found a definite correlation between increasing angiographic cerebral vascular occlusive disease and lower MCAP/MSBP ratios. Six of 32 patients with a preoperative neurologic deficit demonstrated mild but definite postoperative neurologic improvement. The mean MCAP/MSBP ratio in these six patients was significantly lower than that for the remainder of this group (p < .05). Finally, postoperative filling of the middle cerebral artery territory through the graft was found to correlate in an inverse linear relationship with MCAP/MSBP. Evidence is presented that hypoperfusion produced by occlusion of major cranial vessels plays an important role in temporary and permanent neurological deficits seen in patients with cerebrovascular disease.

Clinical Material

Our results are based on a series of 76 patients who underwent cerebral revascularization. The group is comprised of 31 female and 45 male patients, whose ages ranged from 21 to 74 years with a mean age of 54 years. Five patients underwent an EIAB (extracranial-intracranial arterial bypass) and ligation of their ICA for the treatment of a giant internal carotid artery aneurysm. The remaining 71 patients were treated with an EIAB for symptomatic cerebrovascular occlusive disease.

Methods

The MCAP was measured by placing a thirty gauge needle, connected to a Bell and Howell transducer, into a cortical branch of the MCA (middle cerebral artery) at the point of intended anastomosis (fig. 1). Although the small needle will blunt the systolic and diastolic spikes and waves it is perfectly accurate in obtaining mean pressures. The mean systemic blood pressure was measured, using the same transducer, through a twenty gauge catheter placed in the radial artery. All procedures were performed under balanced general endotracheal anesthesia with controlled ventilation. Arterial PCO₂ was maintained at 35 torr and PO₂ at approximately 120 torr.

The patient's charts were reviewed to evaluate the patient's presenting complaints, neurologic deficit and to document which hemisphere was involved. For the 76 patients the presenting neurologic symptoms were: 1) TIA's in 39, 2) completed stroke in 7, 3) completed stroke with continuing TIA's in 25, and 4) no ischemic episodes in the 5 patients with an intracranial aneurysm.

The group was separated according to their angiographic findings into one of the following seven categories: 1) intracranial aneurysm without other vascular disease, 2) severe internal carotid artery stenosis (> 85% estimated by a neuroradiologist by comparing measurements from the nonstenotic to stenotic segment of the artery), 3) severe middle cerebral artery stenosis (> 85%), 4) internal carotid artery occlusion, 5) common carotid artery occlusion, 6) bilateral internal carotid artery occlusion, and 7) middle cerebral occlusion.

The patients with a chronic (greater than 3 months), fixed neurological deficit preoperatively were examined following surgery and any change in the examination recorded.

The post-operative angiograms were reviewed independently and the degree of MCA filling through the EIAB graded on a scale from 0 to 5 (for explanation of grading scale see fig. 2).

Results

For each operated cerebral hemisphere, a ratio of the mean middle cerebral artery pressure to mean systemic blood pressure (MCAP/MSBP ratio) was calculated. Initially the superficial temporal artery pressure was measured with the same needle used to measure the cortical MCAP, but found to be the same as the MSBP. The MCAP/MSBP ratio in the five patients who were being treated for an intracranial aneurysm, and without any other vascular disease, averaged one (1.00). Figure 3 lists the average MCAP/MSBP ratios for each of
Using the two tailed t-test, we compared MCAP/MSBP ratios of patients when divided into groups on the basis of presenting symptoms and postoperative results. When the group of 6 patients who presented with a fixed neurological deficit and showed post-operative improvement was compared to the remainder of patients presenting with fixed deficits, the difference in pressure ratios was significant to the level of \( p = 0.039 \) (fig. 5). When the group with only transient symptoms was compared with the group of patients presenting with fixed neurological deficits, the level of significance is 0.015 (fig. 5).

Finally, we divided the patients into groups on the basis of their post-operative angiographic bypass scores. Postoperation angiography was routinely obtained at 3–4 days following surgery. Blood pressure and \( \text{PCO}_2 \) were not controlled. When the MCAP/MSBP ratios for these patients were plotted against the angiographic score, an inverse linear relationship became apparent (fig. 6). Patients with the most depressed pressure ratios consistently demonstrated the greatest degree of filling via the bypass.

Discussion

We have attempted to clarify the role of cerebral hypoperfusion in patients with symptomatic cerebrovascular disease by measuring the cortical middle cerebral artery pressures. The mean middle cerebral artery and systemic blood pressures were measured in a total of 76 patients and a MCAP/MSBP ratio calculated for each. Five patients had an EIAB for treatment of an aneurysm; none of these five had evidence of occlusive cerebrovascular disease. The average MCAP/MSBP ratio for this group was one (1.0), verifying the observation of Bakay, et al.\(^1\) that the MCA pressure is normally very close to radial artery pressure. In the other cases, the MCAP/MSBP ratios correlated inversely with the severity of the angiographic findings (figs. 2 and 3), demonstrating progressive cerebral arterial pressure with progressive occlusive cerebrovascular disease.

The role of hypoperfusion in producing ischemic symptoms in cerebrovascular disease in man has long
been recognized. Animal models have demonstrated that occlusion of vessels to the brain will result in a neurologic deficit correlating with the amount of cerebral ischemia after a certain threshold has been reached and that early deocclusion will result in reversal of the neurologic deficit.\(^2\)

Decreased arterial pressure secondary to carotid artery occlusion has been widely used to treat intracranial aneurysms. Bakay and Sweet documented the considerable fall in middle cerebral artery pressure that followed ipsilateral carotid artery occlusion in the management of intracranial aneurysms.\(^1\) It was rationalized that reducing pressure would decrease the risk of aneurysmal rupture. Unfortunately, however, reducing perfusion pressure increases the risk of ischemic complications; the acute risk of stroke following elective carotid occlusion is greater than ten percent.\(^3,4\) Furthermore, there is an increased incidence of late ischemic complications in these patients ipsilateral to the occluded carotid artery.\(^4\) The risk of stroke can virtually be eliminated by selection of patients whose cerebral blood flow falls less than 25% with carotid occlusion,\(^3\) or by performing a EIA and staged occlusion.\(^6\)

In our series of aneurysm patients the MCAP fell to an average ratio of 0.76 when the ICA was temporarily occluded. This is a considerably higher ratio than the 0.58 MCAP/MSBP ratio recorded from the unilateral ICA occlusion group secondary to cerebrovascular disease. This difference is likely due to the extensive disease in the collateral vessels in the latter group of patients making the available collateral blood supply less efficient. That hypoperfusion may be the primary cause in producing symptoms in cerebrovascular dis-

**Figure 3.** Histogram of mean MCAP/MSBP ratios plotted for groups of patients divided on the basis of angiographic appearance preoperatively. Group I - nonocclusive vascular lesions (patients with an aneurysm); Group II - ICA (internal carotid artery) stenosis (severe - greater than 85% of vessel diameter); Group III - MCA (middle cerebral artery) stenosis (severe); Group IV - ICA occlusion; Group V - CCA (common carotid artery) occlusion; Group VI - ICA occlusion and Group VII - MCA (middle cerebral artery) occlusion.

**Figure 4.** Histogram of mean MCAP/MSBP ratio plotted for the group of patients with nonocclusive vascular lesions (i.e., aneurysms), the group of patients with stenotic vascular lesions (Group II and III from Figure 2) and the group of combined occlusive vascular lesions (Groups IV, V, VI, and VII from Figure 2).

**Figure 5.** Histogram of mean MCAP/MSBP ratios plotted for the patients with preoperative neurological deficits showing improvement following bypass (Group I), for the patients with preoperative deficits unimproved by bypass (Group II), for all patients with preoperative neurological deficits (Group I and II), and for all those with only transient symptoms prior to surgery (Group III).

**Figure 6.** Plot of mean MCAP/MSBP ratios against postoperative angiographic scores (based on MCA filling through the bypass - Figure 1).
ease in man can be appreciated from the following case presentations.

Case 1
A patient with bilateral ICA occlusion frequently developed weakness of his right arm and aphasia particularly when he assumed the erect posture. These changes were associated with only a small drop in systemic blood pressure. He also developed the same symptoms whenever he ambulated briskly. These symptoms were present and progressive for a period of three months up to the day of surgery. It would seem reasonable to assume that these simple activities altered the delicate balance of collateral flow in this patient resulting in a temporary ischemic episode. Or as Grubb has suggested, that the metabolic demand outstrips the available blood flow. The frequency of the symptoms, the low MCAP/MSBP perfusion ratio of 0.26 in this patient, and the complete relief of symptoms following a patent EIAB during a one year follow-up, support this concept.

Case 2
A patient with a known internal carotid artery occlusion entered the hospital with hypertension of 210/150 which was treated aggressively. The blood pressure fell unexpectedly during treatment to 90/60, during which period she developed a hemiparesis appropriate to the ICA occlusion. With resumption of normal blood pressure her deficit cleared. The same symptoms occurred sporadically at other times, but the blood pressure was not monitored. In this patient, a relatively low MCAP/MSBP ratio of 0.46 in the affected MCA territory led to focal symptoms when a general blood pressure drop occurred. In most patients the first symptom of a significant blood pressure drop is syncpe. Only when a significant focal low perfusion area exists can a generalized blood pressure drop or increased metabolic demand be expected to produce focal rather than generalized symptoms. No further symptoms occurred during eighteen months follow-up after an EIAB.

Of particular clinical interest in this series is the fact that six of the patients demonstrated immediate improvement in their previous neurologic deficit following surgery, and that the six had a mean MCAP/MSBP ratio of 0.32. Each patient had an occlusive as opposed to stenotic vascular lesion. The finding of an immediate reversible neurologic deficit is not new. Reports of improvement in aphasia, homonymous hemianopsia, and other neurologic deficits have been documented. This is the first time that these types of reversible neurologic deficits have been directly correlated with low cerebral arterial pressures. The fact that these six patients as a group have a significantly lower MCAP/MSBP ratio than similar patients whose deficit did not improve, suggests that there may be a pool of viable but non-functioning neurons in those with low perfusion pressures.

Astrup et al. have demonstrated in baboons that neuronal electrophysiological function, as evidenced by normal evoked potentials, is present with cerebral blood flows from 50 to 20 ml/100 gm/min. Evoked potentials diminish with flows below 20 mg/100 gm/min. and finally disappear at 15 ml/100 gm/min. It is not until flows of 12 ml/100 gm/min. are reached that cell death becomes imminent. They have coined the phrase "ischemic penumbra" to describe the hypoperfused zone around an infarct that may still contain the non-functioning, though potentially viable neurons. These results are supported by Morawetz who found that recovery without infarction only occurred when blood flow was above 12 ml/100 gm/min. Presumably flow greater than this value was above the threshold for infarction. Our results, which show that patients with reversible neurologic function have very low perfusion pressures suggest that this ischemic penumbra may be present chronically in some patients with severe cerebrovascular disease.

It is reassuring that there is indeed a relationship between the post-operative extent of angiographic filling of the MCA territory and the MCAP/MSBP ratio (fig. 5). This confirms the belief that the EIAB may dilate in response to need. These findings do not dispute the important role of emboli as the major cause of TIA's. In fact, by the relatively minimal MCA perfusion pressure drops observed in patients with severe stenotic lesions, the embolic theory for producing TIA's in this patient population is supported. Our results provide further evidence for the role of hypoperfusion in the pathophysiology of transient and permanent ischemic neurologic deficits in patients with occlusion of major cerebral vessels.

References
Middle cerebral artery perfusion pressure in cerebrovascular occlusive disease.
R F Spetzler, R A Roski and J Zabramski

*Stroke*. 1983;14:552-555
doi: 10.1161/01.STR.14.4.552

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1983 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/14/4/552

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/