The Hemodynamic Effect of STA-MCA Bypass

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SUMMARY  rCBF was measured by 133Xenon inhalation at rest and during pCO2 manipulation in 19 patients prior to and at various times after STA-MCA bypass surgery. The resting flow increased gradually postoperatively, seemingly more due to progressive clinical recovery from the original neurologic disability than due to the surgical intervention. CO2 reactivity increased in some patients, more in those who preoperatively had the poorest reactivity. The negative relationship between preoperative reactivity and postoperative change in reactivity was highly significant. These observations are consistent with the interpretation that the surgery did not significantly affect the resting flow level but did augment the collateral reserve in those cases in which it had been most severely impaired by the arterial lesion.

A BASIC ARTICLE of faith held by most of us who sometimes recommend STA-MCA bypass to patients is that it may help some of them by increasing the blood flow to the brain, or if not that, it at least increases the number of blood vessels by which blood can flow into the brain. The clinical experience of cessation of recurrent TIA following a bypass operation in a patient with carotid artery occlusion is a gratifying one, though for us it has been relatively rare.1-6

In this paper, we wish to present our initial experience with the serial measurement of rCBF and CO2 reactivity in patients undergoing STA-MCA bypass surgery. It will be seen that the putative benefits of the procedure do not appear to be mediated by a significant sustained effect on the resting rCBF, though a trend toward enhancement of CO2 reactivity in some patients does appear, lending some support to the hypothesis that the collateral circulation reserve may be enhanced. To the extent that this result may be beneficial, it may become possible to use rCBF measurements as an aid to the primarily clinical decision to perform this surgery.

In the context of this clinical study we make comparisons of two commonly used rCBF indices, the f1 and the ISI and find, again, some reason for preference for the latter.7

Methods

Patient Selection

19 patients aged 46-68 (mean 62), with internal carotid occlusion (11), carotid siphon stenosis (5), middle cerebral artery occlusion (2), and middle cerebral artery stenosis (1) were selected for STA-MCA bypass surgery on the basis of clinical and angiographic data, without regard to the rCBF measurements. Clinical disability was mild in all cases: all cases in which it had been most severely impaired by the arterial lesion.

cases the MABP was maintained less than 120 mmHg with appropriate therapy. None had diabetes. Criteria for the latter.4

Summary of rCBF

rCBF was determined by the 133Xenon inhalation method as described in several previous papers, using the two compartment mathematical model perfected by Obrist.8-10 Flow determinations were available from 7 homologous regions of each hemisphere derived from separate scintillation detectors mounted in parallel array. The fast compartment flow (f1) and the initial slope index (ISI) were determined for each region. For this paper, the 7 regional values were averaged as the hemispheric mean. For comparison purposes among patients, the hemisphere in which the bypass operation was performed was compared with the unoperated hemisphere.

Study Protocol

In all cases the rCBF was measured within 2 weeks prior to surgery and at various times postoperatively, the interval between measurements increasing with time from the operation, constrained to some extent by the inconvenience to the patient of traveling great distances for the follow-up evaluation. On the occasion of each measurement, rCBF was determined at least once at rest, and at least once more during voluntary hyperventilation or during inhalation of 2% or 3% CO2 added to room air. In all cases, the end-tidal ex-
TABLE Effect of Surgery on CO₂ Reactivity

<table>
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<th>Time</th>
<th># pts.</th>
<th># runs</th>
<th>Slope</th>
<th>Intercept</th>
<th>Correl coeff.</th>
<th>Slope</th>
<th>Int.</th>
<th>CC</th>
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<td>.974</td>
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<td>.449</td>
<td>.65*</td>
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<td>.547</td>
<td>.72‡</td>
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<td>11</td>
<td>18</td>
<td>.929</td>
<td>.565</td>
<td>.80‡</td>
<td>.932</td>
<td>.601</td>
<td>.79‡</td>
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</table>

Slope, intercept, correlation coefficients for the linear regression equation expressing post-op CO₂ reactivity change as a function of pre-op reactivity.

* p < 0.05. † p < 0.01. ‡ p < 0.001.

pired air pCO₂ was made to change at least 6 torr. When 2 rest determinations were made on one occasion, they were averaged. CO₂ reactivity was calculated from the flows resulting from the highest and lowest pCO₂ of the session as flow change ÷ pCO₂ change. Postoperative reactivity change was calculated as postoperative reactivity minus preoperative reactivity.

Results

For the purpose of analysis and illustration the postoperative measurements were assembled into four arbitrary time intervals: 0–2 weeks (22 measurements in 14 patients), 2–8 weeks (14 measurements in 12 patients), 8–26 weeks (36 measurements in 15 patients), and more than 26 weeks (18 measurements in 11 patients). None of the results more than a year postoperatively were different from those in the interval 26 weeks–1 year, so they were grouped together.

There were no significant correlations between postoperative flow change and such individual variables as nature of the arterial lesion, severity or duration of preoperative disability, or whether the patient did or did not have recurrent preoperative TIA, nor whether these did or did not cease postoperatively. There were no significant postoperative changes in blood pressure. Therefore, all patients are considered together. Although there was regional variability, this did not yield a meaningful pattern. Qualitatively, the time courses of rCBF and CO₂ reactivity changes were not significantly different among the seven regions of each hemisphere. All of the data are therefore expressed as the mean value for the 7 regions of the operated and unoperated hemispheres.

Postoperative Angiography

The postoperative angiogram revealed perfusion via the graft of variable numbers of middle cerebral artery branches. This included only 1 or 2 branches in 4 cases while it was more extensive, sometimes including the entire Sylvian triangle in the rest. In none did contrast extend to the anterior or middle cerebral artery of the operated hemisphere nor to the opposite hemisphere. There was no correlation between the postoperative increase in diameter of the STA and the number of MCA branches filled, nor between either of these angiographic measurements and any of the changes in rCBF and CO₂ reactivity whether considered as single regions or hemispheric means.

![Figure 1. Time course of resting f, hemispheric mean, operated side, nonoperated side. Asterisks indicate measurement is significantly greater than preoperative value (p < 0.05).](http://stroke.ahajournals.org/)

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The Resting Blood Flow

The time courses of the $f_i$ (fig. 1) and ISI (fig. 2) are illustrated. Both indices showed the same general pattern. For both indices the flow was increased postoperatively, the difference between the preoperative and the late postoperative measurements being statistically significant ($p < .05$) for the operated hemisphere.

Small but significant differences between the two hemispheres were present preoperatively. Though these persisted postoperatively, they became smaller with time, i.e. the flow increased more in the operated hemisphere than in the nonoperated hemisphere. It is important, we believe, to note that the progressive increase was not fully evident immediately postoperatively, but developed over time. This trend was more clearly evident with the ISI.

CO$_2$ Reactivity

There was no systematic change in the group means postoperatively compared with the preoperative measurements. However, when these data were analyzed individually, comparing the preoperative reactivity with the change in reactivity postoperatively, a significant negative correlation emerged, i.e. the poorer the preoperative reactivity, the more likely it was to have increased postoperatively while the more nearly normal (higher) it was preoperatively, the less likely it was to have changed postoperatively (fig. 3). These correlations appeared to strengthen with time, and were statistically significant only with the ISI. There was not much difference in these correlations between the operated and nonoperated hemispheres (table).

Analogous correlations between preoperative resting flow and postoperative flow change, between preoperative flow and postoperative reactivity change, and between preoperative reactivity and postoperative flow change were analyzed for, and not found to be present with more than random statistical significance.

Discussion

Seemingly differing from previous reports, the absence here of correlation between angiography and rCBF reflects no more than that the pre- and postoperative angiograms were never done on the same day, and often not in the same week as the rCBF, as well as the poorer regional sensitivity of the inhalation method compared with the intracarotid method.

The main contribution of this paper is the observation of bilateral hemispheric measurements of blood flow made serially over a long time.

Thus, after patients undergo STA-MCA bypass, there is a gradual progressive increase in flow in both hemispheres, more on the operated side, which probably takes six months to become completed. This increase is ultimately somewhat greater in the operated hemisphere. One interpretation would be that the flow, preoperatively constrained by the arterial lesion (most commonly occlusion of the internal carotid artery), continues to be somewhat constrained postoperatively by the maximum capacity of the graft which, thereafter, progressively enlarges, gradually constraining the flow less and less, allowing it eventually to rise to meet the static metabolic requirement of the brain with the eventual benefit of improved clinical function.

The trouble with this interpretation is that the rest-
As we have looked at these data, and those of our other patients, we have noted much more variation in average resting flow level from one patient to another, than between hemispheres with and without arterial lesions, or even within regions of a hemisphere containing an arterial occlusion.

From these and other observations we have been driven to conclude that the bypass surgery does not substantially affect the resting flow level in most cases; that the resting flow preoperatively is adequate for the brain's metabolic needs, while continuing deficiency of it is not the cause of the patient's static or intermittent symptoms. The resting flow progressively increases postoperatively, we believe, not due to the new supply of blood via the anastomosis, but due rather to the normal course of recovery of brain function from the minor cerebral infarction or TIAs which led originally to our having recommended the surgery.

In formulating this hypothesis, we recognize the absence of critical control data which would be needed to sustain it, i.e. serial measurements in similarly affected patients not operated on. These are being collected and will be reported subsequently. We recognize further the unhappy state of being unable to explain the preoperative sympotms in the absence of a simple chronic deficiency of blood flow, but can only suggest that it is intermittently or occasionally transiently insufficient.

Is there then no purpose in the surgery? We think there is. Although the resting flow is apparently not constrained by preoperative blood vessel anatomy, it does appear that the maximum flow is constrained at least in some patients. This is reflected in the observation that the greatest postoperative reactivity increases are in those patients with the poorest reactivity preoperatively. The effect of the surgery is thus not to augment the resting flow, but the capacity for acute further vasodilation. Within the limits of the scatter of the data (fig. 3) one could see the reactivity measurement preoperatively used as a possible criterion in helping to select candidates for the surgery.

The hypothesis is already widely held that in health the principle determinant of the cerebral blood flow is the metabolic need of the brain. We suggest further, reflecting on these limited data, that this determinism also holds true in disease. That is, except during acute failure of the cerebral circulation, resulting in a TIA or stroke, the brain finds a way to get a blood supply adequate to its chronic needs; if a major artery is occluded, a collateral will develop to continue to resupply it adequately, or an acute infarction will intervene, incidentally reducing the demand thereafter.

In this context, the reduction in blood flow following an infarction is due to, rather than the cause of, the reduced function of the brain generally, and of the marginal zone of reduced neuronal excitability focally around the infarct. The flow gradually increases in both hemispheres following the infarction as neurophysiologic recovery (i.e. from "diasciesis") proceeds unrelated to the effects of the STA-MCA bypass.

There are enough clinical and experimental data to document the presence of a range of flow within which function is acutely progressively impaired: EEG change, about 19 cc/100 gram/min., loss of evoked potential, about 16 cc/100 gram/min., K⁺ flux about 12 cc/100 gram/min., and cell death at about the same level. It is important to realize, however, that these functional impairments can be documented for only the acute state. Both we, and many others, have had anecdotal experiences of substantial improvements in brain function from a seemingly stable chronic state, following carotid endarterectomy, and more recently occasionally following STA-MCA bypass. But having witnessed similar improvements in individuals following cholecystectomy and prostatec-

![Figure 3. Relationship between preoperative CO₂ reactivity and postoperative change in reactivity, operated hemispheres 26 weeks. For 18 measurements in 11 patients this relationship is significant p < .0001. See also Table 1.](http://stroke.ahajournals.org/)

Figure 3: Relationship between preoperative CO₂ reactivity and postoperative change in reactivity, operated hemispheres 26 weeks. For 18 measurements in 11 patients this relationship is significant p < .0001. See also Table 1.
HEMATOCRIT AND EXPERIMENTAL ISCHEMIA/Pollock et al.

the importance of haematocrit as a risk factor in cerebral vascular accidents in polycythaemia has long been recognised, and several studies have demonstrated the importance of haematocrit as a risk factor in cerebral infarction.1 In a study of experimental middle cerebral artery occlusion in cats, Sundt et al showed that

The Effect of Haematocrit on Cerebral Perfusion and Clinical Status Following Carotid Occlusion in the Gerbil


SUMMARY The effect of haematocrit on the sequelae of carotid occlusion has been studied in the gerbil stroke model. In 72 animals one carotid artery was clipped. After 30 minutes, the area of cerebral hemisphere filling with a carbon suspension administered intravenously was measured on coronal brain slices and related to the haematocrit. Exchange transfusion of autologous packed red blood cells or plasma produced a haematocrit range of 26% to 59%. At high haematocrit a larger area of non perfusion was more commonly encountered, and the anterior cerebral artery territory was more frequently affected along with that of the middle cerebral. Fifty-seven animals had temporary occlusion of both carotid arteries. Survival was adversely affected at high haematocrit. The role of haematocrit in affecting the pathophysiology of cerebral ischaemia is discussed. Stroke, Vol 13, No 2, 1982

THE INCREASED PREVALENCE of cerebrovascular accidents in polycythaemia has long been recognised, and several studies have demonstrated the importance of haematocrit as a risk factor in cerebral infarction.1 In a study of experimental middle cerebral artery occlusion in cats, Sundt et al showed that injections of autologous packed red blood cells increased both the mortality and the volume of the resultant infarct.2 Haemodilution with albumin resulted in a smaller mean infarct size though the effect was not statistically significant. The numbers of animals in the study was small however and haematocrit levels not given. We have therefore used the gerbil stroke model to study in more detail the effect of varying the haematocrit level on the results of carotid occlusion.

References

Reprints: Dr. Steven S. Pollock, The Reta Lila Weston Institute of Neurological Studies, The Middlesex Medical School, London, WIN-8AA.
The hemodynamic effect of STA-MCA bypass.
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