Intraoperative Regional Cerebral Blood Flow During Carotid Endarterectomy

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SUMMARY: *Regional cerebral blood flow (rCBF) during internal carotid artery (ICA) occlusion for endarterectomy can be measured without inconvenience using the probe holder illustrated.*

When mean ipsilateral hemispheric CBF exceeds 20 ml/100 gm/min, an intraluminal bypass is not necessary (63% of patients), except in patients with extensive cerebrovascular disease in whom rCBF should also exceed 20 ml/100 gm/min in all areas. ICA "stump" pressure is falsely high in about 20% of patients, and is therefore not a dependable criterion for selecting patients who need shunting.

While intraoperative shunting is capable of restoring pre-occlusion CBF levels, it does not eliminate the risk of intraoperative ischemic neurological deficit of probable embolic origin.

RÉSUMÉ: *Le flot sanguin cérébral régional (r CBF) peut être mesuré sans danger lors d'une occlusion de l'artère carotide interne (ICA) pour endarterectomie en employant le porteur illustré.*

Lorsque le CBF hémisphérique ipsilatéral dépasse 20 ml/100 gr/min., il n'est pas nécessaire d'utiliser une dérivation intraluminaire (63% des patients), sauf chez les patients avec maladie cérébrovasculaire étendue dont le rCBF dépasse 20 ml/100 gr/min. dans toutes les régions. La pression dans l'artère carotide interne est faussement élevée chez 20% des patients, et par le fait même ne constitue pas un critère fiable pour la sélection des patients à opérer.

Alors que le "shunting" operatoire peut restaurer les niveaux de CBF pre-occlusion, il n'élimine pas le risque peropératoire de troubles neurologiques ischémiques d'origine embolique probable.

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INTRODUCTION

Since reconstruction of cervical internal carotid artery was first advocated for stroke prevention (Eastcott, et al., 1954) there has been increasing recognition of its effectiveness in long-term stroke prevention. (Fields, et al, 1970). As perioperative morbidity and mortality have decreased (Sundt et al., 1975; West et al., 1979), carotid endarterectomy has seemed increasingly attractive as a means of preventing stroke and has been employed with increasing frequency.

Ensuring adequate cerebral perfusion during the mandatory period of internal carotid artery occlusion associated with endarterectomy, however brief, has been a persistent concern. Use of intraoperative intraluminal shunts to carry blood from the common to the internal carotid artery during endarterectomy has remained controversial (Benoit and Nabavi, 1978; Ferguson et al., 1978), and appears to be unnecessary in the majority of patients (Hays et al., 1972). Intraoperative shunting does not absolutely prevent the occurrence of postoperative neurological deficits (Boysen, 1973). In patients with high-lying carotid bifurcations, particularly in association with obesity and a short neck, operation time may be prolonged and technical difficulty is slightly increased if an intraluminal shunt is employed. Moreover, shunt placement, associated with the use of umbilical tapes, has produced injury to the arterial wall in at least three instances in this Centre. Intraoperative cerebral blood flow (Boysen, 1973; Sundt et al., 1974), intraoperative electroencephalogram (Sundt et al., 1974; Chiappa et al., 1979) and internal carotid artery "stump pressure" (Hays et al., 1972; Sublett et al., 1974) have been used to assess the adequacy of cerebral perfusion during carotid occlusion, thereby determining the need for intraoperative shunting. Our experience with intraoperative regional cerebral blood flow (rCBF) in thirty-eight patients undergoing carotid endarterectomy is reported.

PATIENTS AND METHODS

Regional cerebral blood flow was measured over the ipsilateral cerebral hemisphere using an array of sixteen collimated scintillation detectors mounted on a probe holder (Fig. 1) devised and fabricated by one of the authors (M.I.V.). The series is not consecutive. Cases were usually omitted because of the logistic difficulties associated with arranging for isotope and personnel to be simultaneously available on short notice. It is possible that some inadvertent case selection occurred, but no conscious attempt was made to deliberately select patients whose cerebral perfusion appeared to be vulnerable to internal carotid artery occlusion for inclusion in the series.

Thirty-eight carotid endarterectomies were carried out on thirty-five patients, ranging in age from 44 to 71 years. Twenty-three (61%) were males and fourteen were females. Twenty-two patients (58%) presented with a history of cerebral infarction, which was, in most instances, confirmed by CT scan. Ten patients (26%) presented with transient cerebral ischemic attacks. (TIAs). A further six patients (16%) were subjected to endarterectomy on the basis of stenosis of greater than eighty percent of the luminal diameter discovered at the time of angiography

https://doi.org/10.1017/S0317167100043262 Published online by Cambridge University Press

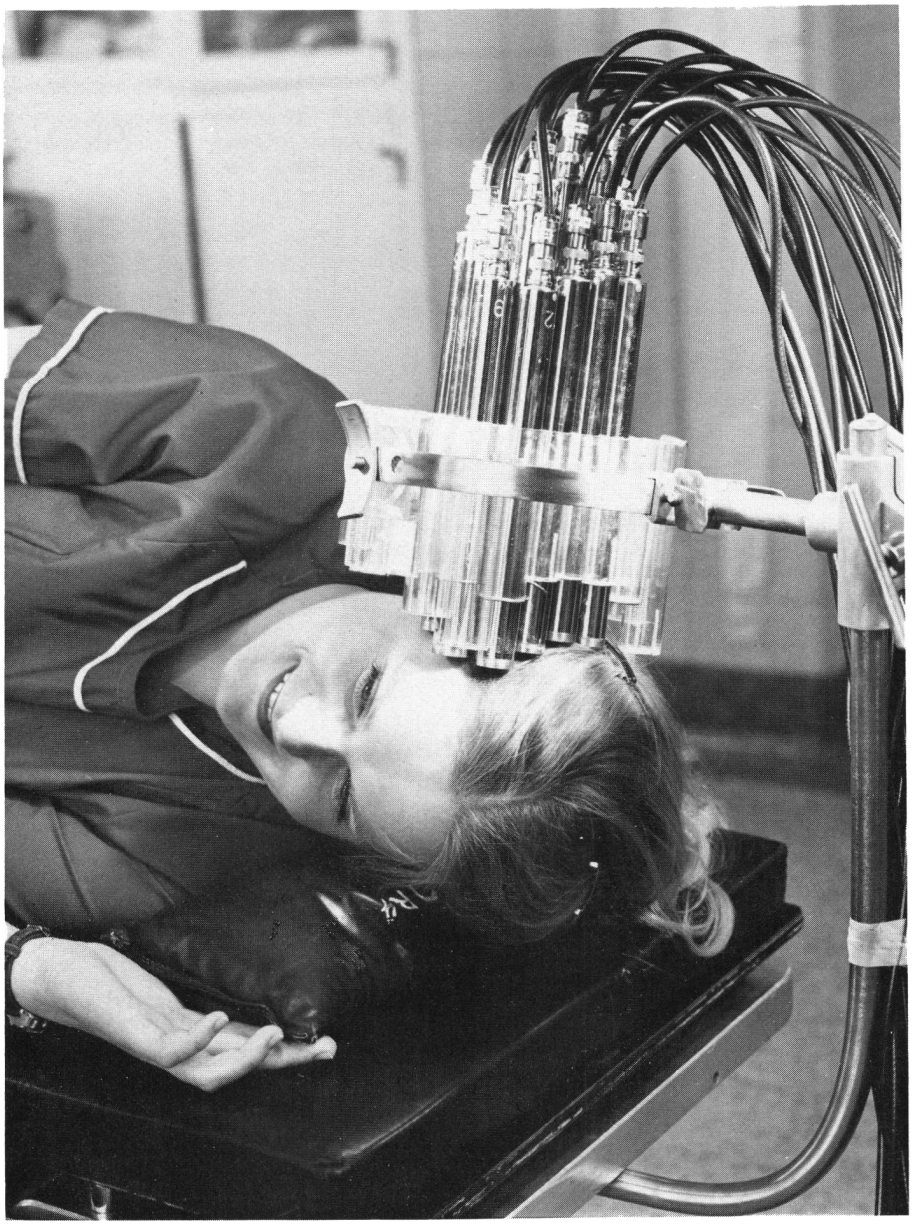

Figure 1 — Probe holder containing 16 collimated scintillation detectors mounted on operating table.

carried out for symptomatic carotid bifurcation plaque on the opposite side.

Twenty-one (55%) of the atheromatous plaques narrowed the internal carotid artery lumen by less than eighty percent of its premorbid diameter, as judged by biplane cerebral angiography and Doppler imaging. The remaining forty-five percent reduced the luminal diameter by eighty percent or more.

Degree of narrowing of the contralateral internal carotid artery was assessed from the biplane angiograms as shown in Tables 2 & 3. Potential collateral circulation to the intracranial arterial territory of the intraoperatively occluded internal carotid artery via either anterior or posterior communicating arteries or both was assessed on a three point scale as shown in Tables 2 & 3.

Nineteen patients (50%) were hypertensive (ie. recorded blood pressures consistently exceeded 180/100). Most of these patients had been receiving antihypertensive medication for many years.

Anesthesia throughout the series consisted of induction with thiopental sodium followed by maintenance with either halothane 0.5% or enflurane 0.5 - 1.0% plus nitrous oxide, supplemented by fentanyl. Halothane was employed in patients with bronchospasm, renal disease or a history of angina pectoris. Otherwise enflurane was employed because of its greater predictability and lessened recovery time.

$PaCO_2$ was maintained at or near 40 torr. Arterial samples were taken from a radial artery cannula simultaneously with each ^{133}Xe injection. When $PaCO_2$ differed by more than 5 torr from that value, cerebral blood flow was corrected to normocapnic values. (Olesen, 1974).

Mean arterial blood pressure (MABP) was monitored with an arterial cannula, usually radial, and was maintained at not less than 100 torr.

Three measurements of CBF were carried out routinely. The initial or baseline determination was carried out as soon as the proximal external carotid artery (ECA) had been exposed. An encircling rubber ligature was placed about the proximal ECA and drawn up through a red rubber catheter sleeve to prevent extracerebral isotope "contamination". A bolus of one to three ml. of physiologic saline solution containing two to three mCi. of ^{133}Xe was injected as quickly as possible through a 25 ga. needle inserted into the common carotid artery. The ^{133}Xe clearance curves were displayed on a chart recorder and the data was stored on magnetic tape. (Meditronic Cerebrograph Model RCBF 165-Meditronic, DK9560, Hadsund, Denmark) Cerebral blood flows were determined by the initial slope method, deriving the mean blood flow for the ipsilateral cerebral hemisphere, and noting any areas of lesser perfusion (Fig. 2). After allowing a minimum of twelve minutes for isotope clearance, a second determination of CBF during trial occlusion was made. On this occasion the common carotid artery was occluded in a similar fashion to the external as soon as the ^{133}Xe bolus

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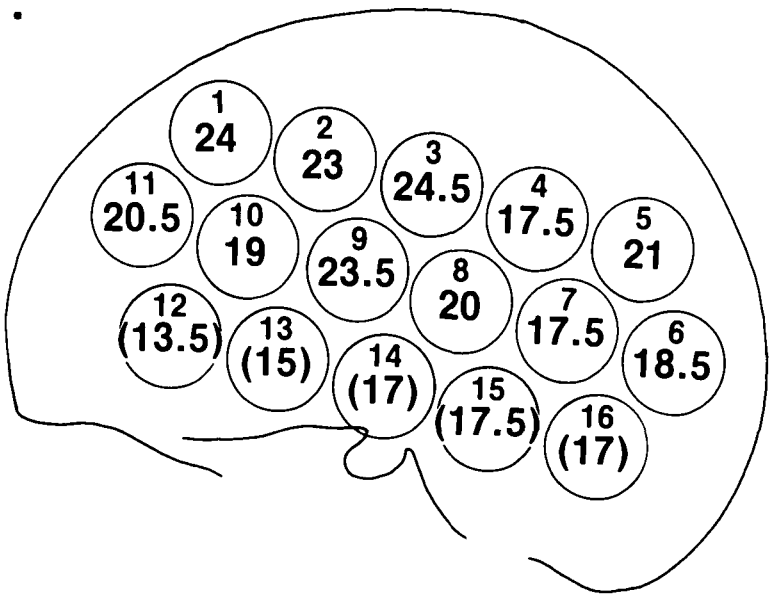

Figure 2— Patient 18, W.T. - rCBF following ICA occlusion. The lower row of probes probably does not represent pure CBF. Note that probes 4, 6, 7, and 10 reveal areas of inadequate cerebral perfusion, while probes 5, 8 and 11 reveal areas of borderline CBF.

reached the brain. Occlusion was maintained for a period of one to two minutes, while CBF was observed and the encircling ligatures were then released. While time was allowed for isotope clearance in each case, the dissection and preparation for endarterectomy proceeded. After the second CBF measurement, the patient was heparinized, and the common, external, and internal carotid arteries were occluded. Arteriotomy of the common and internal carotid arteries was carried out in the fashion customary for carotid endarterectomy. An 18 ga. plastic cannula (Deseret Angiocath. The Deseret Co., Sandy, Utah) was then advanced through the arteriotomy into the internal carotid artery, ensuring that the endothelium appeared normal at the distal end of the arteriotomy prior to cannula insertion and that no intraluminal debris was pushed into the proximal internal carotid artery (ICA). The encircling rubber ligature about the ICA was then drawn down just tightly enough to prevent backflow of blood from the ICA. The arterial cannula was connected to an extension tubing which was completely filled with heparinized physiologic saline. The latter, in turn, was connected to a calibrated pressure transducer and mean ICA "stump" pressure was determined.

Carotid endarterectomy was then carried out with or without employing an intraluminal bypass depending on the measured CBF.

When shunting was deemed necessary, a Javid carotid bypass shunt (C.R. Bard, Inc., Santa Anna, Calif.) was employed. The shunt tubing was previously trimmed to desired length, totally filled with heparinized physiologic saline, and cross-clamped. The proximal end was inserted first and after drawing down the encircling rubber ligature about the common carotid artery the cross clamp was released to clear the shunt of any potential intraluminal debris. The distal end of the shunt was inserted, after removing the "stump" pressure cannula, again taking care not to introduce any intraluminal debris into the proximal ICA. The cross clamp was then released and flow was reestablished. Time required for shunt placement varied slightly but was usually about three minutes or slightly less.

After carotid endarterectomy had been carried out and the arteriotomy had been closed, a further CBF measurement was carried out in identical fashion to the initial determination. On several occasions an additional CBF measurement was made with injection into the common carotid artery with the intraluminal shunt in place and functioning.

Intraoperative shunting was employed throughout the series when mean CBF for the ipsilateral hemisphere was less than 20 ml/100 gm/min. Others have suggested a slightly higher value for the critical lower limit of intraoperative CBF. (Boysen, 1973; Sundt et al., 1974).

In the series reported by Sundt et al., (1974), however, EEG changes did not occur until the occlusion CBF was less than 18 ml/100 gm/min. The significance of the EEG changes described as predictors of postoperative neurological deficit has been questioned by other investigators (Ferguson et al., 1978).

Because of concern about the lack of correlation between CBF and "stump" pressure, it seemed best to adopt a criterion of 50 torr mean as indicative of adequate cerebral perfusion. This is consistent with the methods employed by Boysen et al. (1973), though other investigators (Sublett et al., 1974) believe that lower mean "stump" pressures are consistent with adequate intraoperative cerebral perfusion.

RESULTS

The values of mean ipsilateral CBF, mean ICA "stump" pressure, and postoperative clinical status of the thirty-eight patients are summarized in Table 1. Twenty-four patients (63%) showed mean occlusion CBF values in excess of 20 ml/100 gm/min and the remaining fourteen patients (37%) showed blood flows below that level. The distribution of patients showing blood flows greater than 20 ml/100 gm/min tended to be bimodal, with some patients clustering just above 20 and the remainder showing blood flows well above that level. (Table 1)

Internal carotid artery mean "stump" pressure was 50 torr. or greater in

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TABLE I

No.	Patient	Mean ICA Stump Pressure (torr)	Mean CBF* During ICA Occlusion (ml 100 gm/min)	Intraoperative Shunt	Immediate Postoperative Clinical Status
1	J.B.	60	14.4 ± 2.7	+	NC
2	R.O.	35	16.4 ± 2.5	+	NC
3	J.O.	48	30.1 ± 4		NC
4	I.R.	N.A.	31.3 ± 8.9		NC
5	W.P.	65	36.2 ± 6.7		NC
6	G.M.	30	23.6 ± 5.1		NC
7	W.S.	N.A.	29.7 ± 6.6		NC
8	C.H.	30	21.5 ± 2	+	NC
9	E.P.	60	14.8 ± 4.3	+	NC
10	I.M.	80	45.5 ± 7.1	+	NC
11	N.V.	60	53.2 ± 7.3		M
12	B.N.	50	27.4 ± 4.3		NC
13	S.V.	23	54.6 ± 6		NC
14	B.L.	65	26.1 ± 8.9		NC
15	M.F.	50	16.4 ± 3.3	+	M
16	M.F.	2	30.7 ± 4.1		NC
17	C.E.	40	46.4 ± 16		NC
18	W.T.	45	20.9 ± 4.2		S
19	V.P.	77	29.3 ± 5.1		NC
20	H.S.	22	11.8 ± 2.6	+	NC
21	C.T.	60	49.8 ± 5.2		NC
22	W.E.	N.A.	7.3 ± 1.8	+	NC
23	M.K.	66	14.3 ± 2.3	+	NC
24	H.N.	54	23.8 ± 3.8		NC
25	K.G.	30	25 ± 5.9		NC
26	J.G.	42	23.6 ± 3.7		NC
27	M.P.	10	5.7 ± 0.9	+	NC
28	S.M.	65	22 ± 5.6	+	M
29	E.D.	47	8.9 ± 1.3		NC
30	C.W.	37	27.5 ± 4.3	+	NC
31	L.B.	50	8.8 ± 1.8	+	NC
32	D.D.	70	12.3 ± 2.5	+	S
33	E.D.	N.A.	22.7 ± 4.1	+	NC
34	E.L.	45	64.8 ± 13.5		NC
35	D.C.	35	29.9 ± 4.1		NC
36	F.T.	N.A.	14.7 ± 3.3		NC
37	W.F.	17	5.1 ± 1.8	+	M
38	N.B.	47	13.3 ± 4.4	+	NC

N.A. Mean ICA "stump" pressure measurement not available
NC No change in immediate postop neurological status
M Mild postoperative neurological deficit
S Severe postoperative neurological deficit
\+ Intraoperative shunt was used
* Mean CBF ± one standard deviation

fifteen patients (45%) and less than 50 torr in eighteen patients (55%). ("Stump" pressure was not recorded in 5 patients).

The ICA "stump" pressure and mean CBF agreed in seventeen (52%) of the patients in whom both determinations were recorded: The numerical values for both parameters were either above or below the stated criteria indicative of adequate intraoperative cerebral perfusion. The ICA "stump" pressure was *falsely low* in ten patients (30%): The "stump" pressure fell below 50 torr when recorded mean CBF exceeded 20 ml/100 gm/min. Similarly the ICA "stump" pressure was *falsely high* in six patients (18%).

The clinical neurological status of thirty-two of the patients (84%) was unchanged in the immediate postoperative period. Four patients (11%) showed mild increases in neurological deficit which cleared completely within two to seven days. In three of these four patients an intraoperative shunt was employed. Two additional patients (5%) showed more major increases in neurological deficit which had not completely cleared by the time they left hospital. In one of these patients, an intraoperative shunt was employed (32-D.D.), and in the other (18-W.T.) no shunt was used. Those patients who developed postoperative neurological deficits are shown in Table 2, along with the features of the clinical neurological deficit and the intraoperative CBF. The number is small and, as noted above, only two of the increases in neurological deficit were major and persistent. In cases 16, 32, and 37 intraoperative shunts were employed. One of the severe neurological deficits occurred in a shunted patient (No. 32) and the other occurred in a patient in whom an intraoperative shunt was not employed (No. 18). All of the patients possessed some pathways for potential collateral circulation to the cerebral hemisphere which was susceptible to intraoperative ischemia, but one notes the discrepancy between angiographic characteristics and measured CBF, for example, between cases 11 and 32 where potential collateral pathways appeared to be similar but with the blood flow in one of these patients being only approximately a quarter of that in the other. Similarly one notes that in patient No. 18, despite contralateral ICA occlusion, the intraoperative CBF remained comparable to or higher than that in cases 16, 28, 32, and 37 in which there was no haemodynamically significant stenosis of the contralateral ICA.

The angiographic characteristics of the patients with low intraoperative CBF (less than 20 ml/100 gm/min) are shown in Table 3. In only three instances in the group was there a haemodynamically significant stenosis or complete occlusion of the opposite ICA and in all instances except one, some potential channels for collateral circulation were visualized angiographically. In most of the cases shown one would have angiographically judged the potential collateral circulation to be adequate in the face of measured cerebral blood flows which were

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TABLE 2

Patients Developing Postoperative Neurological Deficits

Patient	Preoperative Risk Factors	Degree and Localization of Clinical Deficit	Stenosis of Contra-lateral ICA*	Intraoperative CBF with Ipsilateral ICA Occlusion**	Collateral Circulation of Intraoperatively Occluded ICA	
					Anterior***	Posterior****
11. N.V.	Hypercholesterol-emia	M Ipsilateral Cerebral Hemisphere	0.0	53.2 ± 7.3	0	2
16. M.F.	Hypertension, Heavy Smoking	M. Ipsilateral Cerebral Hemisphere	0.6	16.4 ± 3.3	1	0
18. W.T.	Hypertension, Diabetes	S. Bilateral Cerebral Hemispheres	1.0	20.9 ± 4.2	1	2
28. S.M.	Hypertension, Heavy Smoking	M. Ipsilateral Cerebral Hemisphere	0.0	22.0 ± 5.6	1	0
32. D.D.	Hypertension	S Ipsilateral Cerebral Hemisphere	0.2	12.3 ± 2.5	0	2
37. W.F.	Hypertension	M. Ipsilateral Cerebral Hemisphere	0.0	5.1 ± 1.8	1	0

M - Mild postoperative neurological deficit

S - Severe postoperative neurological deficit

* - Amount of reduction of diameter of ICA at point of maximal stenosis expressed as a fraction of diameter of ICA immediately superior to the carotid sinus. All measurements were made on whichever of the biplane projections showed the greatest reduction in diameter. If intracranial and extracranial ICA stenoses were both present, the diameter reduction indicated is that of the site of greater stenosis.

*** - Anterior communicating artery

**** - Posterior communicating artery

0 - No collateral circulation visualized

1 - Relevant vessels of circle of Willis patent with some visualization of the intracranial territory of the intraoperatively occluded ICA.

2 - Relevant vessels of circle of Willis large and widely patent with good visualization of the intracranial territory of the intraoperatively occluded ICA.

judged too low to permit endarterectomy without the use of an intraoperative intraluminal shunt. Intraoperative shunts were employed in all of the patients shown in Table 3 with the exception of patient 36 (FT) in whom a shunt was not employed for technical reasons.

DISCUSSION

Boysen, (1973) has reviewed the literature extensively with regard to establishing a critical lower level of CBF during carotid clamping. Based on correlation with the EEG, for example, slowing of EEG frequency was found at CBF levels of 16-22 ml/100 gm/min, while a decrease in amplitude occurred at CBF levels of 11-18 ml/100 gm/min. Electroencephalographic changes occurred within twenty to forty seconds of clamping, and the mean CBF for the group of patients developing EEG changes was 16 ml/100 gm/min. Similarly, Sundt et al., (1974) found that CBF, as determined by the initial slope method, must exceed 18 ml/100 gm/min at an arterial carbon dioxide tension ($PaCO_2$) of 40 torr to sustain a normal EEG with an anesthetic technique similar to that employed in the present study. Chiappa et al., (1979) support a similar critical lower limit for occlusion CBF. Both Chiappa et al., (1979) and Ferguson et al., (1978) have demonstrated that appearance of intraoperative EEG changes indicative of ischemia is not always associated with a postoperative neurological deficit. The evidence suggests that the critical lower value of cerebral blood flow necessary to prevent ischemic damage during carotid clamping is 18-21 ml/100 gm/min (Boysen, 1973).

In the present series all patients with mean ipsilateral hemispheric CBF of

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TABLE 3

*Patients with Low Intraoperative CBF (< 20 ml/100gm/min)**

Patient	Mean Intraoperative CBF (ml/100gm/min)*	Degree of Stenosis of Contralateral I.C.A.**	Collateral Circulation of Intraoperatively Occluded ICA	
			Anterior***	Posterior****
1. J.B.	14.4 ± 2.7	0.0	2	2
2. R.O.	16.4 ± 2.5	0.0	2	2
9. E.P.	14.8 ± 4.3	0.9	2	2
20. H.S.	11.8 ± 2.6	0.0	0	2
22. W.E.	7.3 ± 1.8	1.0	2	1
23. M.K.	14.3 ± 2.3	0.4	2	2
27. M.P.	5.7 ± 0.9	0.3	0	2
29. E.D.	8.9 ± 1.3	0.2	0	0
31. L.B.	8.8 ± 1.8	0.4	0	2
36. F.T.	14.7 ± 3.3	0.0	2	0
38. N.B.	13.3 ± 4.4	1.0	2	0

* CBF with ipsilateral CCA & ECA occluded

** Degree of Stenosis = $\frac{ICAn - ICAs}{ICAn}$ when ICAn = normal diamater of ICA. ICAs = diameter iof ICA at point of maximal narrowing.
"Normal" diameter was measured above the sinus or the siphon, depending on whether the more severe stenosis was intracranial or extracranial. The biplane projection showing the greater degree of narrowing was selected for measurement.

*** Anterior - Potential collateral circulation via the anterior communicating artery, as determined angiographically.

0 - No potential collateral pathway visualized

1 - Anterior communicating artery and both anterior cerebral arteries patent.

2 - Anterior communicating artery and both initial portions of the anterior cerebral arteries of large diameter with spontaneous contrast cross-filling of at least the anterior cerebral circulation on the side of intraoperative ICA occlusion.

**** Posterior - Angiographically demonstrated potential collateral circulation via the posterior communicating artery ipsilateral to the intraoperatively occluded ICA.

0 - No potential collateral pathway visualized

1 - Ipsilateral posterior communicating artery patent (seen from contrast injection in carotid or vertebral injection, or from both routes).

2 - Ipsilateral posterior communicating artery of large diameter with spontaneous passage of contrast into the posterior cerebral circulation from carotid injection or into middle cerebral circulation from vertebral injection, or in both directions.

less than 21 ml/100 mg/min were shunted, with the exception of three cases (18, 29, 36). In cases 29 and 36 intraoperative shunts were not employed for technical reasons. Neither of these patients developed an intraoperative neurological deficit, despite mean ipsilateral hemispheric blood flows that we would consider potentially capable of producing ischemic damage. In patient 18, the CBF was considered borderline but adequate and an intraoperative shunt was not employed. This was perhaps injudicious and, accordingly, the case is worthy of more detailed consideration. The patient is a long standing hypertensive and a mild diabetic. The clinical history included two previous left hemispheric cerebral infarctions. The first of these had occurred four years prior to operation and the second occurred three weeks prior to operation. He had made a good recovery from both, with little residual neurological deficit. Investigation revealed a total occlusion of the right ICA and an irregular plaque at the common carotid bifurcation on the left side. The latter was narrowing the origin of the ICA by approximately seventy percent. There was some passage of contrast into the right middle cerebral arterial distribution via the anterior circle of Willis. Postoperatively, after left carotid endarterectomy, the patient demonstrated a moderately severe neurological deficit reflecting new ischemic damage to both cerebral hemispheres, but affecting the left hemisphere more than the right. Postoperative CT scan confirmed bilateral hemispheric cerebral infarction which suggests a haemodynamic rather than an embolic etiology. If one examines the regional cerebral blood flow in this patient, as illustrated in Fig. 2 one notes that while the mean hemispheric cerebral blood flow (Table 1) met our criteria for adequate cerebral perfusion, several of the regional blood flow values (probes 4, 6, 7, 10) did not. This suggests that in similar patients with contralateral ICA occlusion, particularly if associated with multiple risk factors, no rCBF values should be below 21ml/100 gm/min, if cerebral perfusion is to be considered adequate, and that the critical lower limit for occluded mean CBF should perhaps be revised upward to 25-30 ml/100 gm/min.

The overall incidence of intraoperative neurological deficit in the entire series is higher than one would anticipate, based on recently reported experience. (West et al., 1979). As previously noted this is not a consecutive series and, while no deliberate attempt was made to select higher risk patients, one cannot escape the possibility that we may have been more zealous in obtaining intraoperative CBF determinations in those patients who we considered to be at higher risk (Sundt et al., 1975).

Of the six patients who experienced intraoperative neurological deficits, four were operated on using intraoperative shunts and two were operated on without using shunts. This suggests that not all intraoperative deficits develop on a haemodynamic basis and therefore that intraoperative shunting cannot be expected to completely eliminate the risk of ischemic neurological deficit. In all cases in which shunts were placed, the patient was heparinized, the lumen was carefully inspected at both proximal and distal ends of the arteriotomy during shunt placement to ensure no possibility of embolization, and the shunt was flushed through by brief unclamping after the proximal end had been placed, and prior to insertion of the distal end. In spite of this four patients developed postoperative neurological deficits after the use of shunts. In three of these the deficit was mild and

https://doi.org/10.1017/S0317167100043262 Published online by Cambridge University Press

cleared quickly. The fourth patient, however, developed a major cerebral infarction which was hemorrhagic as demonstrated by CT scan, suggesting the likelihood of an embolic etiology.

Our experience reaffirms the fact that ICA "stump" pressure is not a reliable indicator of the adequacy or the inadequacy of ipsilateral cerebral perfusion (Boysen, 1973). In our experience the "stump" pressure reliably predicts CBF in fifty-two percent of patients and indicates a false low pressure in a further thirty percent. Provided careful attention is paid to maintaining a normal Pa CO_2 and MABP at the time of "stump" pressure measurement, one could therefore ensure adequate cerebral perfusion in approximately eighty percent of patients based on the "stump" pressure criterion. In the remaining twenty percent, however, one could experience a false sense of security about CBF when cerebral perfusion is not, in fact, adequate.

Though angiographically demonstrated occlusion of the contralateral ICA appears to show some tendency to be associated with low intraoperative CBF, the data as shown in Tables 2 & 3 do not support the commonly accepted view that the angiographically demonstrated cerebrovascular anatomy is of value in predicting intraoperative blood flow to the potentially ischemic cerebral hemisphere. In this context it should be pointed out that, while not specifically referred to in the Tables, none of the patients in Tables 2 & 3 showed haemodynamically significant arterial stenoses in the vertebrobasilar system. While the total number of cases included in Tables 2 & 3 is small, it seems probable that reliance on preoperative angiography to determine which patients require intraoperative shunting may well be as misleading as reliance on "stump" pressure.

In the three patients in the current series in whom CBF was measured with the intraluminal shunt in place, CBF levels were comparable to the preocclusive values in the same patients in all instances. This confirms that intraluminal shunting restores adequate CBF during endarterectomy.

In summary, it is feasible to measure rCBF of the ipsilateral hemisphere routinely at the time of endarterectomy. Using the probe holder illustrated, this can be done with minimal extension of operating time and with no inconvenience to the operating team or anesthetist. Most patients (at least two-thirds) can tolerate complete occlusion of the internal carotid artery during endarterectomy without sustaining ischemic neurological deficit. In the remainder, cerebral perfusion should probably be maintained by the placement of an intraluminal shunt, though intraoperative shunting in our hands has not been a universally reliable means of preventing intraoperative neurological deficit. These deficits probably occur on an embolic basis. In patients with extensive cerebrovascular disease or with contralateral ICA occlusion, careful attention should be paid to rCBF in determining the need for shunting rather than depending on mean hemispheric CBF. The internal carotid artery "stump" pressure is not a reliable means of predicting intraoperative cerebral ischemia and will lead to a false sense of security in about twenty percent of patients.

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