

Correlation of Regional Cerebral Blood Flow (rCBF) with EEG Changes During Isoflurane Anesthesia for Carotid Endarterectomy: Critical rCBF

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A prospective evaluation of regional cerebral blood flow (rCBF) (ipsilateral middle cerebral artery distribution) was determined using a ^{133}Xe clearance technique in 31 ASA P.S. II-III patients anesthetized with isoflurane-50% N_2O in O_2 for carotid endarterectomy. Each patient was monitored with 16-channel EEG throughout anesthesia and surgery. Critical rCBF was defined as that flow below which EEG signs of ischemia occurred. Critical rCBF ($T_{1/2}$ method of analysis) was $<10 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$ (mean \pm SE 5.9 ± 1.2) in the six patients in whom transient EEG changes occurred at the time of temporary surgical carotid artery occlusion. No EEG changes occurred with occlusion in the other 25 patients; mean (\pm SE) occlusion rCBF in this group was $18.9 \pm 1.3 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$ ($P < 0.001$). Preocclusion flows were not significantly different in the two groups. Critical rCBF during isoflurane anesthesia was less than that previously determined during halothane anesthesia ($18-20 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$), and is compatible with the effects of isoflurane on CMR_{O_2} and CBF. (Key words: Anesthesia: cardiovascular; neurosurgical. Anesthetics, volatile: isoflurane. Brain: blood flow; electroencephalogram; ischemia. Surgery: vascular; carotid.)

IN OUR INSTITUTION, patients requiring carotid endarterectomy usually have presented with a history of transient episodes of cerebral ischemia (e.g., transient ischemic attacks). A primary goal of the intraoperative management of these patients is prevention of cerebral ischemia. We routinely monitor the EEG throughout the patient's anesthesia and surgery to detect cortical ischemia, and measure regional cerebral blood flow (rCBF) to evaluate flow to the ipsilateral middle cerebral artery (MCA) distribution.¹ Using these methods, an rCBF of $18-20 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$ was previously determined in normocapnic patients anesthetized with halothane- N_2O - O_2 to be that flow below which EEG signs of cortical ischemia occurred (critical rCBF).² After several months, during which, for a variety of clinical reasons, we chose isoflurane as the volatile anesthetic for patients undergoing carotid endarterectomy, our clinical impression was that the critical rCBF seemed less during isoflurane than during halo-

thane anesthesia. This led to the current prospective study designed to determine the critical rCBF in normocapnic patients anesthetized with isoflurane-50% N_2O in O_2 .

Methods

The protocol, requiring only clinical monitoring and anesthetic techniques already established in our practice, was approved by the Research Committee of the Department of Anesthesiology, and required no other institutional committee approval or informed consent.

Regional cerebral blood flow was determined in 31 ASA II or III patients 45-79 yr of age (mean 65.5 yr) undergoing carotid endarterectomy. Twenty-four patients received no premedication. Of the seven patients who had their preoperative angiogram on the day of surgery, four received pentobarbital (50-70 mg), two received meperidine (50 mg), and one received diazepam (5 mg), all administered more than 3.5 h prior to the first rCBF determination. All patients taking oral cardiac, antihypertensive, or bronchodilator medications had received these medications more than 4.5 h prior to the first rCBF determination.

Following pre-anesthetic discussion and a brief neurologic exam, baseline ECG (7-lead) and 16-channel EEG recordings were obtained. The 16-channel EEG and the V_5 lead of the ECG were monitored throughout the intraoperative course until the patient emerged from anesthesia and responded to commands in the operating room. Following preoxygenation, anesthesia was induced using incremental doses of thiopental, with isoflurane added to the inspired gas (O_2) when appropriate. Lidocaine 1.0-1.5 mg/kg was administered intravenously prior to laryngoscopy. Orotracheal intubation was facilitated with a nondepolarizing muscle relaxant: pancuronium 0.06-0.1 mg/kg (23 patients); metocurine 0.3-0.4 mg/kg (3 patients); or vecuronium 0.1 mg/kg (5 patients). Ventilation was controlled to maintain arterial carbon dioxide partial pressure (Pa_{CO_2}) at 37-42 mmHg. Total fresh gas flow was equal to or greater than 5 l/min to eliminate or minimize any rebreathing of ^{133}Xe . A level of anesthesia appropriate for hemodynamic stability and continuous EEG monitoring was established using isoflurane-50% N_2O in O_2 . Patients received neither narcotic nor sedative supplements.

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The patient's ECG; EEG; direct arterial pressure (radial artery cannula); inspired concentration (F_I) and expired partial pressure (P_E) of O_2 , N_2O , N_2 , CO_2 , and isoflurane (mass spectrometer); and heart sounds, breath sounds, and temperature (esophageal stethoscope/temperature probe) were monitored throughout anesthesia. Fluids were warmed prior to intravenous administration, and an in-line Siemens Servo Humidifier® model 150 ("artificial nose") was utilized to minimize heat loss from the airway. Mean arterial pressure (MAP) was maintained within the patient's preoperative range. Appropriate adjustments in inspired isoflurane concentration, and/or intravenous propranolol or phenylephrine infusion (0.004%), were utilized when indicated to maintain stable MAP and heart rate. Arterial samples for blood gas analysis were collected after a stable level of anesthesia (as defined by hemodynamic parameters, EEG pattern, $P_{E_{\text{isoflurane}}}$) had been achieved (baseline), and with each of three rCBF determinations (preocclusion, occlusion, and post-repair flows). Blood pressure and expired gas partial pressure were recorded at 2-min intervals during rCBF determinations, and at 5-min intervals otherwise. Using a single extracranial collimated scintillation detector over the posterior parietal boss (MCA distribution), rCBF was determined from analysis of clearance curves of ^{133}Xe (200 μCi) injected through a 27-gauge needle into the internal carotid artery.^{1,3} Counts were recorded on an analogue chart recorder, and each washout curve was analyzed in its intraoperative format and again when replotted on semi-logarithmic scale paper. To optimize the baseline activity for the occlusion of rCBF measurement, the time interval between preocclusion and occlusion flows was at least 5 min, and the beta slope of the preocclusion flow in the semi-logarithmic plot was extrapolated** and used as the baseline for the occlusion rCBF. When a temporary shunt was used,^{1,3} the occlusion rCBF curve was extrapolated for calculation using the $T_{1/2}$ method of analysis.

Flows were calculated using initial slope,⁴⁻⁶ $T_{1/2}$,⁷ and $rCBF_{10}$ ⁸ techniques. According to the $T_{1/2}$ method,

$$rCBF = \frac{0.87 \times 0.693 \times 60}{T_{1/2}} \times 100,$$

** Remaining ^{133}Xe activity from the preocclusion rCBF would result in a gradually decreasing baseline during the occlusion rCBF clearance curve. If the first rCBF clearance curve has not reached zero by the time the second rCBF measurement is initiated, two errors can result. To assume that the baseline for the second curve remains constant at that level of counts evidenced by the first clearance curve at the time the second curve is initiated will result in an over-estimate of flow, while to assume the baseline for the second curve is zero counts will result in underestimation of flow. To minimize this error, we utilized the extrapolated tail of the preocclusion rCBF clearance curve (semi-log plot) as the baseline for the occlusion rCBF curve. Although not without problems, this technique seemed to offer the best practical correction.⁶

TABLE 1. Occlusion rCBF ($T_{1/2}$)

No Patients	rCBF ($T_{1/2}$) ($\text{ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$)	EEG Change
6	<10	Yes
15	≥ 16	No
8	10-15	No
1	9	No
1	8.5	No

where 0.87 is the tissue/blood partition coefficient for Xe in the rapidly perfused tissue (assumed to be gray matter), 0.693 is the \ln of 2, $T_{1/2}$ is the time in seconds required for the counts to decrease to $1/2$ the maximum, 60 converts seconds to minutes, and 100 converts to flow per 100 gram of brain per minute. This method was used for all clearance curves replotted on semi-log paper, and for the 27 intraoperative ("raw") preocclusion clearance curves where the curve had not reached zero baseline by the time the occlusion rCBF was initiated.¹ The ratio of raw to semi-log blood flows was 1.0, suggesting that curve analysis was consistent. Results will be presented using the $T_{1/2}$ method, as in previous reports from our institution.^{1,2,9}

Values for rCBF, hemodynamic variables, respiratory gases, and temperature were compared by Student's *t* test for unpaired data in the patients who demonstrated EEG changes with carotid occlusion versus those who did not. A *P* of less than 0.05 was considered significant.

Results

EEG activity was comparable in all patients prior to carotid occlusion.

Transient EEG signs of cerebral ischemia (when less severe, reduction in amplitude of faster frequency activity and increase in amplitude and wave length of slower frequency activity; but when more severe, reduction in amplitude of both faster and slower frequency activity) at the time of temporary carotid occlusion occurred in six patients, each of whom had an occlusion rCBF less than $10 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$ (8.2, 2.3, 6.3, 3.3, 5.6, and $9.5 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$) (tables 1, 2). No EEG signs of ischemia occurred at the time of carotid occlusion in the other 25 patients (tables 1, 2); occlusion flows ($\text{ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$) were 8.5 and 9 in two, 10-15 in eight, and ≥ 16 in 15 patients (table 1). There was no significant difference in preocclusion rCBF between those patients that subsequently showed an EEG change with occlusion ($28.8 \pm 1.3 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$) and those that did not ($31.4 \pm 1.6 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$) (table 2). Mean occlusion rCBF ($T_{1/2}$) was significantly less for the six patients who demonstrated an EEG change ($5.9 \pm 1.2 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$) than in those who exhibited no EEG changes ($18.9 \pm 1.3 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$) (table 2).

TABLE 2. rCBF ($T_{1/2}$) ($\text{ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$)

	EEG Change (6) (Mean \pm SE)	No EEG Change (25) (Mean \pm SE)
Pre-occl.	28.8 \pm 1.3	31.4 \pm 1.6
Occl.	5.9 \pm 1.2*	18.9 \pm 1.3

* Significantly different from group without EEG change, $P < 0.001$.

A temporary shunt was utilized in the six patients who demonstrated an EEG change at the time of carotid occlusion and in the ten other patients with flows less than $16 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$.

No patient awakened with a new neurologic deficit.

Critical rCBF did not correlate significantly with patient temperature, expired isoflurane or N_2O partial pressures, premedication, PaCO_2 , change in MAP between preocclusion and occlusion rCBF (table 3), or the use of propranolol or neosynephrine.

At low flows, the rCBF₁₀ method may slightly overestimate flow; while the $T_{1/2}$ method, as with other initial slope techniques, may slightly underestimate flow.^{4,7,10} When the $T_{1/2}$ method was compared with the rCBF₁₀ method in these patients, the ratio $\frac{\text{rCBF}_{10}}{T_{1/2}}$ was 1.1 for preocclusion and 1.3 for occlusion flows, which is compatible with a tendency for the $T_{1/2}$ method to underestimate and the rCBF₁₀ method to overestimate low flows.^{4,7,10}

The 10-min height/area technique, used to confirm the appropriateness of our technique of plotting the curves on semi-log paper, indicated that the curves were plotted and analyzed appropriately.

Discussion

CRITICAL RCBF

Based upon review and evaluation of our previous experience and that of others, we believe that our definition

TABLE 3. PaCO_2 , MAP, $P_{\text{Eisoflurane}}$, and Temperature in 31 Patients

	EEG Change (6) (Mean \pm SE)	No EEG Change (25) (Mean \pm SE)
PaCO_2 (mmHg)		
Preoccl.	39.7 \pm 0.8	39.8 \pm 0.4
Occl.	39.7 \pm 0.7	40.1 \pm 0.4
MAP (mmHg)		
Preoccl.	99 \pm 4	105 \pm 3
Occl.	95 \pm 6	96 \pm 3
$P_{\text{Eisoflurane}}$ (mmHg)		
Preoccl.	3.82 \pm 0.36	4.43 \pm 0.24
Occl.	3.85 \pm 0.34	4.34 \pm 0.23
Temp ($^{\circ}\text{C}$)		
Preoccl.	35.5 \pm 0.2	35.7 \pm 0.1
Occl.	35.5 \pm 0.2	35.7 \pm 0.1

of critical rCBF (*i.e.*, that blood flow below which EEG signs of ischemia occur) is appropriate and valid.^{1,2,9,11} As defined, the critical rCBF ($T_{1/2}$) during normocapnia and isoflurane-50% N_2O in O_2 anesthesia approximated $8\text{--}10 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$ in this prospective evaluation in 31 patients undergoing carotid endarterectomy. This critical rCBF is similar to the $8 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$ suggested by Gibson *et al.* during normocapnia and 0.25–0.5% (expired) isoflurane in 50% N_2O - O_2 in their evaluation of transconjunctival O_2 monitoring in 18 patients undergoing carotid endarterectomy at our institution.¹² It also is compatible with the critical rCBF suggested by a retrospective analysis of occlusion flows with and without EEG changes during isoflurane- N_2O - O_2 anesthesia (with small doses of fentanyl supplementation in some patients) in 317 patients who were part of a review of 1,722 carotid endarterectomies in 1,628 patients at our institution.¹³

In a previous retrospective study, critical rCBF during normocapnia and halothane-50% N_2O in O_2 approximated $18\text{--}20 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$, with no EEG signs of ischemia occurring at occlusion flows greater than $24 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$, and consistent EEG changes occurring at flows less than $18 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$.² Using the same ¹³³Xe rCBF measurement technique, McKay *et al.*⁹ reported that, of 13 normocapnic patients anesthetized with 0.4–0.8% (inspired) enflurane-50% N_2O in O_2 who had occlusion flows less than $18 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$, 11 developed EEG changes consistent with ischemia, while only two demonstrated no EEG signs of ischemia. These authors suggested that the critical rCBF during enflurane anesthesia might be lower than that during halothane, but noted that their data were not sufficient for “meaningful speculation.” Since, in both of those studies,^{2,9} the method of curve analysis assumed the baseline for the occlusion washout curve was zero counts and, thus, would have tended to underestimate flow, the critical rCBF with halothane is at least $18\text{--}20 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$, and, perhaps, greater.

Critical rCBF represents a point along the continuum of the relationship between neuronal O_2 demand and O_2 supply. Critical rCBF neither is defined as a fraction of, nor is necessarily a function of, the preocclusion flow. That, in the patients in the present report, EEG signs of cortical ischemia were absent with rCBF values as low as $10 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$, and no patient awoke with a new neurologic deficit, suggests that O_2 delivery remained adequate for O_2 demand.

CEREBRAL METABOLISM

The results of this study are compatible with previous work addressing the effect of isoflurane on cerebral metabolism in animals, although higher concentrations of isoflurane were used in some of these studies.^{14–18} In cats,

isoflurane (with 75% N₂O) was a more potent cerebral metabolic depressant than halothane at concentrations up to 1.5 minimum alveolar concentration (MAC).¹⁷ In dogs, 1.4–3.0% end-expired isoflurane (in N₂ and O₂) produced a dose-related decline in CMRO₂, with no direct toxic effects on cerebral metabolic pathways at isoflurane concentrations which suppress cortical electrical activity (3% end-expired).¹⁴ In dogs, 0.5 MAC isoflurane is a more potent cerebral metabolic depressant than is 0.5 MAC halothane.¹⁸ Measurement of local CBF and local cerebral glucose uptake (l-CMRg) in rats revealed a dose-dependent l-CMRg reduction in cortical areas with isoflurane, and that the metabolic decrease with isoflurane is more potent than that with halothane and enflurane and reaches a maximal effect at EEG isoelectricity.¹⁹

CEREBRAL "PROTECTION"

In animal studies of hypoxia or global cerebral ischemia, isoflurane has been reported to provide a degree of protection similar to the barbiturates. Compared to a control group, survival time in mice breathing 5% O₂ was increased significantly at inspired isoflurane concentrations of 1.0 and 1.4%.¹⁵ In dogs, global ischemia produced by acute hemorrhagic hypotension had a less detrimental effect on the cerebral energy state in animals given isoflurane as compared to those given N₂O.¹⁵ Similarly, profound hypotension (MAP ~ 30 mmHg) produced by isoflurane for 1 h had no effect on the cerebral energy state, while that produced by halothane, nitroprusside, or trimethaphan was associated with significant perturbation of the cerebral energy state.²⁰

The apparent protection provided by isoflurane in the above circumstances may not apply to regional ischemia. In a primate study, isoflurane concentrations sufficient to produce a deep burst suppression EEG pattern (~2 MAC) were not protective compared to thiopental following 6 h of middle cerebral artery occlusion in normothermic, paralyzed, hypocapnic (PaCO₂ ~ 30 mmHg) adult baboons.²¹ Noting that the reason for the lack of cerebral protection with isoflurane compared to that provided by thiopental is not known, the authors suggested that protection may not result entirely from metabolic depression, but may involve other factors, such as flow distribution. In this study, although all animals in the thiopental group received nitroprusside and all animals in the isoflurane group received phenylephrine to control mean arterial pressure (MAP), MAP was significantly lower in the isoflurane group.

CBF

At concentrations less than 1.5 MAC, isoflurane produces less cerebral vasodilation than does halothane.¹⁷ In man, CBF is not increased significantly at 1 MAC isoflu-

rane, but is at higher isoflurane concentrations.†† One MAC isoflurane (plus 75% N₂O) produced no significant change in CBF in normocarbic cats.¹⁷ That, in our patients, pre-occlusion cerebral blood flows generally were less during isoflurane than they had been during halothane anesthesia is compatible with this difference in the effects on blood flow of the two agents.

Preocclusion blood flows measured by the ¹³³Xe technique in our patients with cerebrovascular disease were lower than those reported in man during isoflurane anesthesia by Eintrei *et al.*,²² Newman *et al.*,²³ or Murkin.²⁴ We believe a review of these reports lends credence to the importance of comparing comparable patient populations and methodologies when addressing the effects of a specific anesthetic agent. In the Eintrei report,²² data were collected from 24 adult (22–69 yr of age) premedicated patients with brain tumors anesthetized with 1.5 MAC isoflurane (corrected for 70% N₂O) and 0.1–0.2 mg · h⁻¹ fentanyl, and ventilated to 26–33 mmHg PaCO₂. "Mannitol 300–500 mg · kg⁻¹ was given IV at the start of surgery . . ." Regional CBF was measured by placing topically 0.6–1.3 mCi ¹³³Xe in 0.1 ml saline on cortex under a 1 cm² 20-μm-thick Mylar® film and plotting a 10-min washout curve from a single external detector. Mean (±SD) rCBF was 67 ± 27 ml · 100 g⁻¹ · min⁻¹ during N₂O-isoflurane anesthesia at MAP 74 ± 13 mmHg. Newman *et al.*²³ studied 12 patients (29–70 yr of age) undergoing cerebral aneurysm clipping. The unpremedicated patients received 2–6 μg/kg of fentanyl as part of the induction sequence. Anesthesia was maintained with 1.0 ± 0.25% inspired isoflurane in O₂-air (no N₂O) for at least 1 h prior to CBF measurement. Mean (±SD) PaCO₂ was 33 ± 4 mmHg. Patients received 1–1.5 mg/kg mannitol at least 20 min prior to craniotomy. Using a Kety-Schmidt technique involving 10% N₂O, with cerebral venous blood aspirated from the jugular bulb and a 15-min time to N₂O saturation, mean (±SD) global CBF was 49 ± 14 ml · 100 g⁻¹ · min⁻¹, with a range of 31.9–85.7 ml · 100 g⁻¹ · min⁻¹. Murkin *et al.*²⁴ studied seven patients (mean age 30 ± 9 yr) undergoing closed heart surgery. The patients received narcotic premedication 90 min prior to surgery. Anesthesia was maintained with 2.5% (inspired) isoflurane in O₂ (no N₂O, no air) for at least 30 min prior to CBF measurements using ". . . an insert gas washout technique utilizing ¹³³Xe and an array of 10 scintillation detectors, 5 over each cerebral hemisphere." At a mean (±SD) PaCO₂ of 38 ± 6 mmHg and a mean (±SD) cerebral

†† Murphy FL Jr, Kennell EM, Johnstone RE, Lief PL, Jobs DR, Thompkins BM, Gurtsche BB, Behar MG, Wollman H: The effects of enflurane, isoflurane and halothane on cerebral blood flow and metabolism in man. Abstracts of Scientific Papers, Annual Meeting of the American Society of Anesthesiologists, 1974, pp 61–62.

perfusion pressure of 69 ± 15 mmHg, CBF was 44.5 ± 6.8 $\text{ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$. Thus, these three studies differ from each other and from the present report with regard to patient populations, maintenance anesthetic regimens (including isoflurane concentrations), techniques of measuring CBF, and reported CBF values. The three reports do not address the question of critical CBF.

The preocclusion flows in the present report are similar to those derived from a retrospective evaluation of 317 carotid endarterectomy patients anesthetized with isoflurane-50 $\text{N}_2\text{O}-\text{O}_2$ (with small [$<250 \mu\text{g}$] supplemental doses of fentanyl in some patients) in whom rCBF was measured by the intracarotid ^{133}Xe injection technique outlined in this and other^{9,12} reports.¹³ Mean (\pm SD) pre-occlusion rCBF was 37 ± 15 $\text{ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$ in Grade 1, 37 ± 17 $\text{ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$ in Grade 2, 32 ± 13 $\text{ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$ in Grade 3, and 29 ± 17 $\text{ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$ in Grade 4 patients. ‡‡ That CBF values during isoflurane anesthesia tend to be lower in patients with severe cerebrovascular occlusive disease should not be surprising. This is also reflected by the lower mean flows in our Grade 4 patients.

EEG, SHUNT

The EEG is a reliable indicator of cortical function during general anesthesia.^{1,2} EEG levels prior to carotid occlusion were comparable in all of our patients receiving isoflurane in this prospective evaluation. Furthermore, this general EEG pattern during isoflurane was judged by the same electroencephalographer (FS) to be comparable to that observed in patients receiving halothane anesthesia in the previous retrospective study.² Additional similarities between the two studies include three of the investigators (JMM, JDM, TS), comparable anesthetic and surgical techniques, equipotent concentrations of halothane and isoflurane, and utilization of the $T_{1/2}$ method for analysis of the clearance curves. Differences between the two evaluations, in the present prospective study, include: respiratory gas measurements by the mass spectrometer; analysis of the clearance curves by three techniques, including plotting on semi-log paper, extrapolation

‡‡ System for grading a patient's potential risk for carotid endarterectomy:¹³

Grade 1: Neurologically stable (no neurologic risk factors), no major medical risks, no major angiographically determined risks, unilateral or bilateral ulcerative-stenotic disease.

Grade 2: Neurologically stable, no major medical risks, significant angiographically determined risks.

Grade 3: Neurologically stable, major medical risks, with or without significant angiographically determined risks.

Grade 4: Major neurologic risks, with or without associated major medical or angiographically determined risks.

of the beta slope of the pre-occlusion rCBF curve as baseline for the occlusion rCBF curve; and two additional surgeons.

In our practice, the surgeon's decision to place a temporary shunt is based upon the patient's history and examination, the angiographic findings, the EEG at the time of occlusion, and the rCBF.¹ Of the 31 patients under discussion, temporary shunts were utilized in the six with EEG changes, and in ten others who had occlusion rCBF $< 16 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$ but no EEG indications of ischemia. In these ten patients, the time between carotid occlusion and shunt flow was ≥ 4 min in nine patients and 2 min in one patient (rCBF = $8 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$). We cannot speculate as to possible eventual EEG changes in these patients had the shunts not been utilized. However, since Boysen¹⁰ noted that most EEG changes occurred within 20–40 s after occlusion (halothane anesthesia), Sharbrough *et al.*² reported a 12–180 s interval between occlusion and EEG changes, and our experience has been that EEG changes which are going to occur usually do so within 1 min of occlusion, we conclude that sufficient time for the EEG to indicate ischemia probably had occurred in at least nine of the ten patients.

That the critical rCBF is less during isoflurane anesthesia suggests that this anesthetic may be the preferable currently available volatile anesthetic when neuronal function is at risk from ischemia. That, at comparable levels of cortical activity as defined by EEG, the critical rCBF appears to be different during isoflurane than during halothane prompts questions as to the mechanism(s) by which these agents affect CMRO_2 and CBF.

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