ANAESTHETIC CONSIDERATIONS FOR CAROTID THROMBO-ENDARTERECTOMY*

LEONARD C. JENKINS, B.A., M.D., C.M., F.R.C.P.(C),
EVELYN R. ENGELBRECHT, B.A., M.D., AND
W. B. CHUNG, M.D., C.M., F.R.C.S.(C), F.A.C.S.†

SURGICAL PROCEDURES designed to improve precarious brain perfusion demand meticulous supportive anaesthetic management to avoid precipitating additional impairment of circulation. It is worthwhile first reviewing some of the pathophysiological features of carotid artery stenosis and occlusion as a basic guide for approaches to the surgical and anaesthetic management of endarterectomy patients.

PATHOPHYSIOLOGY OF CAROTID ARTERY STENOSIS

Haemodynamic Alterations

The lesion. The usual pathological abnormality of carotid artery stenosis is atherosclerotic narrowing at the origin of the internal carotid artery. Figure 1 shows a carotid arteriogram demonstrating this lesion.¹ Acute thrombosis of the

Figure 1. Carotid arteriogram of patient just showing minimal narrowing of internal carotid artery. (With permission from W. B. Chung, Canadian Journal of Surgery 10: 21-27, January, 1967.)

*Presented at the Annual Meeting, Western Divisions, Canadian Anaesthetists' Society, Saskatoon, Saskatchewan, March 31, 1967.

†From the Department of Surgery and Division of Anaesthesiology, University of British Columbia, Vancouver, B.C.

cervical carotid artery may be superimposed on an atherosclerotic plaque at the carotid bifurcation. Cerebral emboli may occur from the surfaces of ulcerated carotid atheromata and are probably often responsible for hemiplegia associated with carotid arteriograms, especially when the needle is introduced into the region of the carotid bifurcation. Even vigorous palpation of a diseased artery may break fragments loose and result in cerebral infarctions.

Collateral circulation. The circle of Willis, supplied by the bilateral internal carotid and vertebral arteries, potentially allows for adequate anastomotic channels during occlusion of one or more of the inflow vessels. In addition, the external carotid artery supplies anastomotic branches to the middle cerebral artery via the angular branch of the maxillary artery and the ophthalmic branch of the facial artery, while the occipital branch of the facial artery forms an anastomosis with the vertebral artery.² Under normal conditions and with the head maintained in a position of rest there tends to be no mixing of blood in the communicating arteries of the circle of Willis. The opposing streams of arterial blood meet at a "deadpoint" where the pressure of the two is equal, and thus mixing does not occur. If an internal carotid artery is then occluded, the "dead-points" disappear and the anastomotic channels illustrated in Figure 2 become

FIGURE 2. Left carotid arteriogram of patient J.M. showing rapid filling of both left and right anterior and middle cerebral arteries in the presence of a diseased right internal carotid artery. (With permission from W. B. Chung, Canadian Journal of Surgery, 10: 21-27, January, 1967.)

operative. Figure 2¹ is a carotid arteriogram demonstrating collateral circulation, with the normal left carotid artery filling both left and right anterior and middle cerebral arteries, in the presence of a diseased right internal carotid artery. However, the blood flow through anastomotic channels is not always adequate, and 20 to 30 per cent of patients exhibit signs of cerebral anoxia after occlusion of an internal carotid artery.³

Meyer and Denny-Brown⁴ described another important feature in the establishment of a collateral blood flow. They demonstrated that there was a dilatation of the vessel resulting from a localized reduction in intra-luminal pressure. They applied the term "Bayliss effect" to the phenomenon of alteration in vessel diameter mediated by the local head of pressure. It was also demonstrated that when the carotid artery was occluded on one side, the blood flow in the contralateral carotid artery, as well as the vertebral blood flow, showed compensatory collateral increases. With a gradual decrease in blood pressure the collateral contribution progressively fell.⁵

Factors Affecting Cerebral Blood Flow

The cerebral blood flow is dependent upon two main factors: (1) *mean perfusing pressure*, which is related to mean systemic arterial blood pressure, and (2) *cerebrovascular resistance*. Cerebrovascular resistance is dependent upon four main components: (i) blood viscosity, (ii) cerebrospinal fluid pressure, (iii) cerebrovascular diameter, and (iv) body temperature.

Hypotension. An autoregulatory mechanism has been demonstrated⁵ in the internal carotid artery during hypotension which is not present in the external carotid artery. When the blood pressure was gradually decreased by induced hypovolaemic hypotension, the internal carotid artery blood flow showed a minimal decrease until a critical blood pressure of 60 to 70 mm. Hg was reached. At this point it fell sharply. On the other hand, the external carotid artery blood flow fell progressively in a linear relation to the decreasing blood pressure. There was no critical blood pressure seen. It is suggested that one of the factors responsible for the difference between the two arteries is the rich sympathetic innervation of the external carotid system, since, following sympathectomy, the external carotid flow reacts like the internal carotid arterial pattern. The sensitive response of the cerebral vessel musculature to stretch reflexes and chemical stimuli, which is not controlled by the autonomic nervous system, seems to be the basis of the autoregulatory mechanism. Thus the following factors have been suggested as being responsible for the autoregulation in the presence of hypotension.

1. When the intraluminal pressure of the cerebral vessels is reduced the tonic constriction of these vessels decreases, their resistance decreases, and flow is maintained until pressure drops below a critical level. Conversely, a rise in blood pressure gives rise to vasoconstriction and thereby a decreased perfusion.

2. A biochemical explanation appears possible. A decreased blood pressure would result in decreased oxygen and increased carbon dioxide tension. These local factors tend to cause vasodilation with a decreased cerebral vascular resistance and therefore an increased flow.

Hyperventilation.^{6,7} The hypocapnia which accompanies hyperventilation of a significant degree (P_{CO_2} of 20 to 25 mm. Hg) will cause the following changes.

1. *Metabolic acidosis.* This not only is produced as a compensatory effect of respiratory alkalosis but results directly through an effect on the intracellular metabolism which in turn is probably secondary to peripheral vasoconstriction and a decrease in cardiac output.

2. *A shift to the left of the haemoglobin dissociation curve.* This may result in tissue hypoxia, particularly if there is a coexistent anaemia or hypoxaemia.

3. *A shift towards anerobiosis in cerebral carbohydrate metabolism.*⁸ This is due to the decreased blood flow, the decreased oxygen transfer in respiratory alkalosis, and the specific effect of decreased carbon dioxide tension on metabolic pathways.

On the basis of these findings it was suggested,^{6,7} that hyperventilation be used with caution in patients with decreased cerebral perfusion or increased cerebral metabolic requirements. Thus it should be avoided in patients with carotid stenosis or occlusion.

Hypercapnia. Increased carbon dioxide tension, by producing cerebral vasodilatation, is a significant factor in the regulation of cerebral blood flow. Inhalation of 5 per cent carbon dioxide can increase the blood flow by 75 per cent in normal man. Therefore it has been suggested as a way of protecting against iatrogenic interference with cerebral blood flow.^{9,10} However, a significant study of the effectiveness of carbon dioxide in producing vasodilatation in the presence of cerebral atherosclerosis¹¹ has concluded that: (1) cerebral vascular reactivity to carbon dioxide is high in normal adults of all ages with only gradual decreases with the normal aging process; (2) cerebral vascular reactivity to carbon dioxide is markedly reduced in severe cerebral vascular disease; (3) a normal response to carbon dioxide inhalation makes the diagnosis of cerebral atherosclerosis unlikely. Therefore, the use of hypercarbia during anaesthesia for carotid artery surgery would appear to be of little value if generalized cerebral atherosclerosis is present.

Other factors. Endogenous adrenaline plays a dual role in affecting the cerebral circulation. The local action of adrenaline produces vasoconstriction, but this is overshadowed by the rise in systolic blood pressure which results in an actual increase in flow. The over-all effect of *noradrenaline*, on the other hand, is one of vasoconstriction.^{11,12}

Thiopentone and most other anaesthetic agents and sedatives decrease the cerebral blood flow along with depression of central nervous system function and cerebral metabolism. The exceptions are diethyl ether, which is believed to have specific vasodilator properties, and chloroform, which has been found to increase cerebral blood flow. Halothane in the concentration of 1.2 per cent in oxygen was demonstrated in man¹³ to be a mild cerebral vasodilator.

A fall in body temperature leads to a significant decrease of cerebral blood flow and mean arterial pressure. Cerebral metabolism is also reduced to a point where the cells can survive for a short period without any oxygen at all. There appears to be a direct linear relationship between body temperature and cerebral metabolism, but fortunately, in the early stages of cooling, the reduction in cerebral blood flow tends to lag behind the fall in temperature unless the patient is inadvertently allowed to shiver.⁷

A rise in oxygen tension (particularly above atmospheric pressure) leads to vasoconstriction of cerebral vessels. However, with the inhalation of 80 per cent oxygen or with arterial oxygen tensions as high as 400 mm. Hg, cerebral blood

flow has remained unchanged, provided that the carbon dioxide tension remains normal.¹⁴

Clinical Presentation

Presenting signs and symptoms. Clinical manifestations produced by internal carotid artery disease are usually those of unilateral focal cerebral ischaemia, monocular visual disturbances, and contralateral sensory and motor deficits. However, it is important to appreciate that almost any neurological deficit may be produced by extracranial cerebrovascular disease.¹⁵

Palpation of the carotid artery in the neck or in the tonsillar fossa is often misleading. A bruit heard in the neck is unquestionably the most important physical finding, suggesting significant extracranial vascular disease.¹⁵

Investigative Procedures. Arteriographic studies are essential to confirm the diagnosis. The two most popular techniques currently employed for carotid arteriography are percutaneous needle puncture of the common carotid artery and retrograde subclavian arteriograms by means of percutaneous insertion of a catheter into the brachial or axillary artery.¹⁵

Ophthalmodynamometry has been used recently to evaluate the status of the carotid circulation.¹⁶ It is based on the finding that the pressure in the ophthalmic artery is the same as that immediately distal to the carotid block. The ophthalmodynamometer is a means of applying a variable measured pressure against the sclera. While this pressure is being applied, the fundus is observed with an ophthalmoscope. The range of pressure through which the retinal arteries pulsate determines the systolic and diastolic pressure readings.

The use of radioactive isotopes to determine cerebral blood flow is of value in detecting stenosis or obstruction but is of no value in localizing the site of the obstruction.¹⁴

Electroencephalograms have been of little value in the investigation of the condition, but are of value in detecting acute hypoxic cerebral changes which may occur when the carotid artery is clamped at surgery.¹⁷

Aspects of Medical Management Pertaining to Anaesthesia

Prior to surgical consultation, endarterectomy patients may have had medical management with a variety of drug preparations. The anaesthetist should be aware of them, as they may influence the conduct of the anaesthetic.

Vasopressors, which are given with the intent of increasing blood pressure and thereby cerebral perfusion, cause a rise in cerebral vascular resistance to the point where the total cerebral arterial flow may actually decrease.¹⁵

The use of carbon dioxide and carbonic anhydrase inhibitors (Diamox) has been demonstrated to increase oxygen delivery by active dilatation of the cerebral arteries. However, it is of little value in dilating the arteriosclerotic lesion.¹³

Anticoagulation remains as the only widely accepted form of medical management. It has been used in many cases where the level of the block in the carotid system is unknown, whereas surgery is limited to blocks at the extracranial level. Anticoagulants appear to exert their greatest benefit in reducing the incidence of

transient ischaemic attacks. The mechanism of action is believed to be the reduction of thrombo-embolism, since there is no change in the rate of cerebral blood flow or blood viscosity. The benefits gained, however, are often more than nullified by the complications of severe or fatal haemorrhage.^{15,18}

ANAESTHETIC MANAGEMENT FOR CAROTID THROMBO-ENDARTERECTOMY

The Surgical Procedure at the Vancouver General Hospital

Three basic types of surgical approach have been evolved to restore brain circulation: (1) excision with graft replacement, (2) graft bypass, (3) endarterectomy. Endarterectomy is particularly suitable for discrete atherosclerotic lesions and has been the method used for 74 of the 78 lesions corrected surgically between 1961 and 1966 at the Vancouver General Hospital.¹ The carotid vessels are exposed. A longitudinal arteriotomy is made and an internal polyethylene shunt is established. The intraluminal shunt is used in preference to the external type of shunt because it is less cumbersome and more easily inserted. Systemic and local heparin are used in all cases.¹ The endarterectomy is then performed.

Principles of Anaesthetic Management for Carotid Thrombo-endarterectomy

Prevention of ischaemic brain damage during surgery. The danger of ischaemic brain damage, ever present with a stenotic or occluded carotid artery, is increased during surgery not only by the period of total occlusion necessary prior to the establishment of the internal shunt, but also by the possible fall of blood pressure which may occur during anaesthesia and surgery. Recently, internal jugular venous oxygen tension monitoring has been advocated as an index of adequacy of cerebral oxygenation and perfusion.^{19,20} We are currently evaluating this type of monitoring in our series of patients.

Prompt recognition of early thrombosis of the operated artery in the post-anaesthetic recovery room. Rethrombosis is one of the major postoperative hazards following carotid thrombo-endarterectomy. As it is recognizable only by the development of contralateral neurological signs or ipsilateral visual disturbances, the patient must be co-operative as soon as possible after the end of the operation. For this reason and also to avoid hypotension, premedication and anaesthesia are kept as light as possible. The anaesthetic administration is judged so that the patient will be conscious very early in the immediate postoperative period.

Current Anaesthetic Approach for Carotid Thrombo-endarterectomy

The current anaesthetic approach at the Vancouver General Hospital, advocated to protect the brain during the usual 8 to 35 minutes (mean of 16 minutes) required for an adequate endarterectomy, has evolved through a series of stages¹ from (1) general anaesthesia and hypothermia (30° C) by surface cooling (10 procedures), to (2) local anaesthesia (17 procedures), to (3) general anaesthesia with external or internal shunt (51 procedures). These methods have developed from experience over the past five years¹ during which time 60 patients with 78 carotid artery lesions were treated surgically. Of the ten patients initially

operated upon under general anaesthesia and hypothermia, three had external and two had internal shunts. Because of the prolonged anaesthetic recovery time associated with hypothermia, the next phase was the use, in 17 cases, of local anaesthesia, which had the advantage of providing constant monitoring of the patient's neurological status. Only one of the 17 patients required insertion of a shunt to offset hypoxic changes. The disadvantage of local anaesthesia was the great strain placed on the surgeon by the necessity of speed, and the potentially detrimental effects of hurrying, associated with unexpected jeopardizing movements of the patient.

TABLE I
PREOPERATIVE MEASURES TO MAINTAIN CEREBRAL PERFUSION

Measure	Results
Light premedication	early awakening cardiovascular stability
Careful induction	cardiovascular stability no cough or strain
Endotracheal tube (Saunders)	patent airway

The present anaesthetic approach for carotid thrombo-endarterectomy at the Vancouver General Hospital is summarized in Tables I through IV. Table I deals with the preoperative management. This entails careful assessment of the patient and the ordering of a light preanaesthetic medication consisting of a small amount of analgesic or tranquillizer and a belladonna agent. Over-sedation must be avoided to prevent hypotension and prolonged emergence from anaesthesia. On arrival of the patient in the operating room and after a reliable intravenous route is established (central venous pressure catheter), a sleep dose of thiopentone is given. Following intravenous succinylcholine and ventilation with oxygen, an armoured endotracheal tube is placed. This type of tube prevents kinking, which can be associated with carotid artery surgery following positioning and retracting manoeuvres.

Halothane is chosen for maintenance of anaesthesia (Table II). It is a mild cerebral vasodilator. It allows a non-explosive technique and a relatively rapid

TABLE II
OPERATIVE MEASURES TO MAINTAIN CEREBRAL PERFUSION

Measure	Results
N ₂ O, O ₂ , halothane	cautery early awakening cerebral vasodilation
Gallamine	light anaesthesia reduction of carotid sinus reflex decreased cerebral venous pressure
Controlled ventilation (PCO ₂ 40-45 mm. Hg)	increased cerebral blood flow
Horizontal position	increased cerebral blood flow
Surgical shunt, with heparinization	increased cerebral blood flow

emergence. The use of a muscle relaxant permits a lighter plane of anaesthesia, and allows controlled ventilation with a tendency to hypercarbia (P_{CO_2} of 40–45 mm. Hg) to be conducted more precisely. Gallamine is preferred because of its atropine-like effect in reducing the carotid sinus reflex, which may be stimulated during carotid artery manipulation. It also tends to offset the bradycardia associated with halothane. It tends to prevent abdominal muscle straining associated with spontaneous respiration, which may create increased central venous pressure and a subsequent rise in cerebral venous pressure. The Wright respirometer and Astrup blood-gas analysis are utilized in monitoring the carbon dioxide tension, which is maintained just at or above normal levels. A bypass shunt is used routinely with local and systemic heparinization.

Several other monitors are used (Table III) in addition to frequent determinations of the blood pressure. The electroencephalogram is of value in assessing the

TABLE III
SPECIAL MONITORING

Procedure	Results
E.E.G.	gross evaluation of cortical activity
E.C.G.	useful with associated atherosclerotic heart disease
Systemic arterial and internal jugular PO_2 , PCO_2 , pH, HCO_3^- , base excess	index of cerebral blood flow
Central venous pressure	indirect indicator of cerebral venous pressure blood volume cardiac pump capacity monitor transfusion route
Esophageal temperature	check on stability
Wright respirometer	prevention of hyperventilation maintenance of adequate ventilation

adequacy of cerebral blood flow, especially in the critical period of clamping of the artery just prior to insertion of the shunt, and also in detecting possible kinking of the shunt. The electrocardiogram is of value especially in patients with generalized atherosclerosis and associated heart disease.

Determination of the internal jugular venous oxygen tension has been used as an aid in assessing the adequacy of cerebral blood flow by us and others.^{19,20} The central venous pressure is useful in assessing stability of the cardiovascular system. Oesophageal temperature readings aid in avoiding hyperthermia or hypothermia, and in establishing normothermia by the end of the procedure.

Special medications have been advocated to increase cerebral blood flow and perfusion during surgery (Table IV):

TABLE IV
SPECIAL MEDICATIONS TO MAINTAIN CEREBRAL PERFUSION

Medication	Results
Low molecular-weight dextran	increased cerebral blood flow decreased blood viscosity
Vasopressors	avoidance of hypotension

1. Low molecular-weight dextran reduces blood viscosity and thereby improves flow and probably reduces the incidence of early postoperative thrombosis.^{21,22}

2. *Vasopressors* are used only when hypotension cannot readily be corrected by the usual attention to blood volume, carotid sinus reflex, depth of anaesthesia, and ventilatory effects on central venous filling. They are used intermittently and for as short a period as possible.

RESULTS OF CAROTID THROMBO-ENDARTERECTOMY AT THE VANCOUVER GENERAL HOSPITAL¹

Of the 60 patients, there was clinical improvement in 47, or 78 per cent (Table V). There were three deaths, giving a mortality rate of 5 per cent. One

TABLE V
SUMMARY, 60 PATIENTS, 78 PROCEDURES

	Incidence/total	Per cent
Re-establishment of flow (78 lesions)	63/78	87
partial occlusion	57/57	100
complete occlusion	6/21	29
Clinical improvement (60 patients)	47/60	78
flow re-established	45/53	85
flow not re-established	2/7	28
Increased neurological deficit	6/60	10
temporary	3/60	5
permanent	3/60	5
Deaths	3/60	5

of the deaths occurred one month postoperatively, owing to a myocardial infarction. A second death was due to rethrombosis at the operative site (the right carotid artery), with cerebral infarction in the right middle cerebral distribution. In this instance general anaesthesia and hypothermia had been used. The blood pressure fell to 70/50 mm. Hg very briefly near the end of the surgical procedure, and again within fifteen minutes after arrival in the post-anaesthetic room. The patient was unconscious on arrival in the postanesthetic room, but was "responding to some commands" eight hours later. Fifteen hours later she was conscious, but her left arm and leg were paralysed. The complicating factor of the prolonged emergence from hypothermia appeared to be of significance here, in that rethrombosis might have been detected earlier had hypothermia not been used.

Three other patients suffered rethrombosis of the operated artery with increased neurological deficit which was detected immediately. Re-exploration and removal of the thrombus gave good results, with clinical improvement.

The third fatality, and the three patients who had permanent increases in neurological deficit, did not show extracranial thrombosis of the vessels at re-exploration, or, in the case of death, at autopsy. The cerebral infarction was therefore attributed to intracranial thrombosis. Review of the preoperative

arteriograms of this group of patients showed that they had diffuse atherosclerotic changes of the cerebral vessels. Such abnormal arteriograms were seen in only six of the sixty patients in this series, four of whom developed neurological complications. It is therefore concluded that patients with extensive intracranial vascular disease should not be chosen for carotid endarterectomy.¹

Our results compare favourably with those of other centres,²³⁻²⁶ particularly for the last 27 procedures done (and not reported in this series), in addition to the initial 78, where the surgical and anaesthetic methods outlined have been used. There have been no deaths, and the rate of clinical improvement has been higher (85%).

SUMMARY

Patients presenting for carotid thrombo-endarterectomy have precarious brain perfusion. The anaesthetic approach must not be detrimental to adequate cerebral blood flow. Systemic arterial blood pressure must be maintained. Agents or techniques are avoided which may increase cerebrovascular resistance by affecting blood viscosity, cerebrospinal fluid pressure, cerebrovascular diameter, or body temperature changes.

In the preoperative period, a light premedication is advocated to promote early awakening and to maintain a stable respiratory and cardiovascular status. Induction is smooth, with no straining or coughing. An armoured oro-endotracheal tube is used to ensure a patent airway at all times. Maintenance anaesthesia is nitrous oxide, oxygen, and halothane. Thus, cautery may be used. Cerebral vasodilatation tends to occur with halothane. The respiratory tract is not irritated. Early awakening is possible. Gallamine is used to ensure a light plane of anaesthesia and to offset the bradycardia possibly produced by halothane. Also, the carotid sinus reflex, which may be stimulated during surgery, is blocked with gallamine. Use of a muscle relaxant prevents abdominal muscle contractions, which inevitably occur during spontaneous respiration and which may increase central venous pressure and, consequently, cerebral venous pressure. Gallamine facilitates the use of controlled ventilation. By monitoring systemic arterial and internal jugular venous Po_2 , Pco_2 , pH, bicarbonate and base excess (Astrup), precise regulation of tidal volume, minute volume (Wright respirometer) and Pco_2 at 40-45 mm. Hg is achieved. Moderate hypercarbia tends to increase cerebral blood flow and hyperventilation; cerebral vasoconstriction and cerebral hypoxia are avoided. A polyethylene surgical shunt bypass is used during the thrombo-endarterectomy. Additional special monitors used are the electrocardiogram, electroencephalogram, central venous pressure, and oesophageal temperature. Low molecular-weight dextran is advocated to decrease viscosity and thus increase cerebral blood flow.

Results of carotid thrombo-endarterectomy in the past five years in 60 patients undergoing 78 procedures showed 47, or 78 per cent, with clinical improvement. There were three deaths, giving a mortality rate of 5 per cent. One death was due to a myocardial infarct one month postoperatively. Another patient died from rethrombosis of the internal carotid artery in the early postoperative period. The

third death resulted from intracranial rethrombosis with infarction, superimposed on a setting of severe generalized cerebral atherosclerosis.

RÉSUMÉ

Les patients qui se présentent pour une thrombo-endartériectomie de la carotide ont une circulation cérébrale précaire. L'anesthésie ne doit pas nuire au courant du sang dans le cerveau. La pression artérielle doit être maintenue. Il faut éviter les agents ou les techniques susceptibles d'accroître la résistance vasculaire du cerveau en affectant la viscosité du sang, la pression du liquide céphalo-rachidien, le diamètre des vaisseaux cérébraux et les changements de température du corps. Avant l'opération, on conseille une prémédication légère afin de préparer un réveil précoce et de maintenir un état respiratoire et cardiovasculaire stable. L'induction est douce, sans effort et sans toux. Nous employons un tube oro-trachéal dont la paroi contient une spirale métallique pour qu'en tout temps les voies respiratoires demeurent libres. L'entretien de l'anesthésie se fait au protoxyde, oxygène et halothane. Ainsi on peut employer le cautère. Avec l'halothane, une vasodilatation cérébrale a tendance à se produire. Il n'y a pas d'irritation des voies respiratoires. Il est possible d'obtenir un réveil précoce. On emploie la gallamine dans le but de maintenir un plan superficiel d'anesthésie et de prévenir la bradycardie que l'halothane peut produire. De même, le réflexe du sinus carotidien, susceptible d'être stimulé au cours de la chirurgie, se trouve bloqué par la gallamine. L'usage des myorésolutifs prévient les contractions des muscles abdominaux qui, au cours de la respiration spontanée, surviennent nécessairement; ces contractions peuvent augmenter la pression veineuse centrale et, en conséquence, la pression veineuse cérébrale. La gallamine facilite l'usage de la ventilation contrôlée. En suivant de près la PO_2 et la PCO_2 dans les artères systémiques et dans la veine jugulaire interne, le pH, les bicarbonates et l'excès de bases (Astrup) on peut réaliser une régulation précise de l'air courant, du volume minute (respiromètre de Wright) et une PCO_2 entre 40 à 45 mm. Hg. Une légère hypercarbie a tendance à augmenter la circulation cérébrale et l'hyperventilation, la vasoconstriction et l'hypoxie cérébrales sont évitées. Les moniteurs additionnels spéciaux que nous employons sont l'électrocardiogramme, l'électroencéphalogramme, la tension veineuse centrale, la température dans l'oesophage. On emploie un tube de polyéthylène comme voie d'évitement au cours de la thrombo-endartériectomie. Du dextran à faible poids moléculaire est à conseiller pour diminuer la viscosité sanguine et augmenter ainsi la circulation cérébrale.

Au cours des cinq dernières années, les résultats de la thrombo-endartériectomie carotidienne chez 60 malades qui ont subi 78 thrombo-endartériectomies chirurgicales ont montré de 47 à 78 pour cent d'amélioration clinique. Trois morts établissent le taux de mortalité à 5 pour cent. Une mort est due à un infarctus survenu un mois après l'opération. Un autre malade est mort d'une nouvelle thrombose de la carotide interne au début de la période postopératoire. La troisième fatalité est due à une nouvelle thrombose intracrânienne avec infarctus ajoutée à une athérosclérose cérébrale généralisée et importante.

REFERENCES

1. CHUNG, W. B. Carotid Artery Surgery for Cerebrovascular Insufficiency: Experience with 78 Procedures in 60 Patients. *Canad. J. Surg.* 10: 21 (1967).
2. BRECHNER, V. L.; WALTER, R. D.; & DILLON, J. B. Practical Electroencephalography for the Anaesthesiologist. 1st ed., Springfield: Thomas (1962), p. 61.
3. PILCHER, C. & THUSS, C. Cerebral Blood Flow. *Arch. Surg.* 29: 1024 (1934).
4. MEYER, J. S. & DENNY-BROWN, D. The Cerebral Collateral Circulation: I. Factors Influencing Collateral Blood Flow. *Neurology.* 7: 447 (1957).
5. MEYER, J. S.; HANDA, J.; HUBER, P.; & YOSHIDA, K. Effect of Hypotension on Internal and External Carotid Blood Flow. *J. Neurosurg.* 23: 191 (1965).
6. KEUSKAMP, D. H. G. Hyperventilation and Hypoxia of the Brain: Considerations on Oxygen Dissociation. *Anaesthesist.* 14: 204 (1965).
7. WOLLMAN, H.; ALEXANDER, S. C.; COHEN, P. J.; SMITH, I. C.; CHASE, P. E.; & VAN DER MOLEN, R. A. Cerebral Circulation during General Anaesthesia and Hyperventilation in Man. *Anesthesiology.* 26: 329 (1965).
8. ALEXANDER, S. C.; COHEN, P. J.; WOLLMAN, H.; SMITH, T. C.; REIVICH, M.; & VAN DER MOLEN, R. A. Cerebral Carbohydrate Metabolism during Hypocarbica in Man: Studies during Nitrous Oxide Anesthesia. *Anesthesiology.* 26: 624 (1965).
9. HORMI, J.; HUMPHRIES, A. W.; YOUNG, J. L.; BEVEN, E. G.; & SMART, J. F. Hypercarbic Anesthesia in Cerebrovascular Surgery. *Surgery.* 59: 57 (1966).
10. WELLS, B. A.; KEATS, A. S.; & COOLEY, D. A. Increased Tolerance to Cerebral Ischemia Produced by General Anaesthesia during Temporal Carotid Occlusion. *Surgery.* 54: 216 (1963).
11. SCHIEVE, J. F. & WILSON, W. P. The Influence of Age, Anaesthesia and Cerebral Atherosclerosis on Cerebral Vascular Activity to Carbon Dioxide. *Am. J. Med.* 15: 171 (1953).
12. WYLIE, W. D. & CHURCHILL-DAVIDSON, H. C. A Practice of Anaesthesia. 2nd ed., London; Lloyd Luke (1966).
13. WOLLMAN, H.; ALEXANDER, S. C.; COHEN, P. J.; CHASE, P. E.; MELMAN, E.; & BEHAR, M. C. Cerebral Circulation of Man during Halothane Anesthesia: Effect of Hypocarbica and of D-tubocurarine. *Anesthesiology.* 25: 180 (1964).
14. TURNER, J.; LAMBERTSEN, C. J.; & OWEN, S. G. Effects of 0.08 and 0.8 Atmospheres of Inspired Oxygen Tension on Cerebral Hemodynamics at a "Constant" Alveolar Carbon Dioxide Tension of 43 mm. of Mercury. *Fed. Proc.* 16: 130 (1957).
15. STANSEL, H. C. & CAHOW, C. E. Extracranial Cerebrovascular Disease. *Ann. Rev. Med.* 16: 331 (1965).
16. CALDERON, R. G. & EISENBREY, A. B. Postural Ophthalmodynamometry in the Surgical Management of Occlusive Aortocranial Disease. *J. Neurosurg.* 22: 30 (1965).
17. PEREZ-BORJA, C. & MEYER, J. S. Electroencephalographic Monitoring during Reconstructive Surgery of the Neck Vessels. *Electroencephalog. & Clin. Neurophysiol.* 18: 162 (1965).
18. GONZALEZ, L. L. & LEWIS, C. M. Cerebral Haemorrhage following Successful Endarterectomy of the Internal Carotid Artery. *Surg. Gynec. & Obst.* 122: 773 (1966).
19. LYONS, C.; CLARK, L. C.; McDOWELL, H.; & McARTHUR, K. Cerebral Venous O₂ content during Carotid Thromboendarterectomy. *Ann. Surg.* 160: 561 (1964).
20. CLAUS, R. H.; HASS, W. U.; & RANSOHOFF, J. Simplified Method for Monitoring Adequacy of Brain Oxygenation during Carotid Artery Surgery. *New England J. Med.* 273: 1127 (1965).
21. CHRISTOPHERSON, E. B.; MAY, A. G.; DE LEESE, J. A.; & ROB, C. G. The Hemodynamic Effects of Low Molecular Weight Dextran in Arteries with Critical Stenosis during Acute Hemorrhage. *Surgery.* 60: 402 (1966).
22. MARSHALL, M. Anaesthesia for Carotid Endarterectomy Using Local Brain Perfusion and Cooling. *Anaesthesia.* 20: 207 (1965).
23. EGAN, R. W. & UPSON, J. E. Carotid Artery Surgery for Cerebral Vascular Insufficiency. *Angiology.* 16: 698 (1965).
24. HOHF, R. P.; WATTS, D. R.; TARKINGTON, J. A.; & CRAMPTON, A. R. Late Results of Surgical Treatment for Carotid Insufficiency. *Surg. Gynec. & Obst.* 123: 319 (1966).
25. MURPHY, F. & MACCUBBIN, D. A. Carotid Endarterectomy. A Long-term Follow-up Study. *J. Neurosurg.* 23: 156 (1965).
26. PERDUE, C. D. & LAWRY, K. Surgical Revascularization after Acute Thrombosis of the Carotid Artery. *Am. Surgeon.* 31: 790 (1965).