ANAESTHESIA FOR CAROTID ARTERY SURGERY

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Carotid endarterectomy has assumed an increasingly important role in the definitive management of those patients with cerebrovascular insufficiency whose lesions are located in the extracranial vasculature. Of the extracranial arterial lesions which cause symptoms of transient cerebral ischaemia, the most common are internal carotid artery stenosis, vertebrobasilar insufficiency and lesions of the first part of the subclavian artery associated with the subclavian steal syndrome (Editorial, 1974). The first reported successful carotid reconstruction was achieved in 1954 (Eastcott, Pickering and Rob, 1954) and since that time endarterectomy of a stenosed segment of the carotid artery has become a standard and successful method of treatment.

The objectives of the operation are to remove the atheromatous plaque without leaving loose intima distally, to leave a smooth arterial wall and to achieve a blood-tight closure (Smellie, 1972). As a result, the operation entails clamping of the common, internal and external carotid arteries during the actual removal of the plaque and during the arterial reconstruction. As with every operation, it is important that the benefits from surgery exceed the risks inherent in the procedure. If carotid endarterectomy is to be performed on patients with extracranial occlusive cerebrovascular disease who have not suffered a stroke, then every effort must be made to prevent ischaemic brain damage during the period of operation.

GENERAL CONSIDERATIONS

Experimentally, it has been established clearly that obstruction of a carotid artery does not decrease its blood flow until the cross-sectional area of the vessel has been decreased to less than 5 mm² (that is, an 80–90% reduction) (Brice, Dowsett and Lowe, 1964). Even when carotid obliteration has become complete, the change in cerebral blood flow may be remarkably slight. Thus, in a study of cerebral arterial pressures in the monkey, Symon (1967) demonstrated that complete occlusion of one internal carotid artery would cause a decrease of only 14% in the pressure in the ipsilateral middle cerebral artery. Occlusion of the contralateral carotid artery had virtually the same effect. Bilateral carotid occlusion produced a decrease of 50% in the middle cerebral artery pressure since the monkey, like man, receives the greater part of its cerebral blood flow via the carotid arteries. However, the margin of safety is very wide, since cerebral blood flow has to be decreased to 15% of normal before cerebral necrosis occurs (Zulch and Behrend, 1961).

Nevertheless, despite the foregoing experimental evidence, cerebral infarction can occur not infrequently in man after unilateral carotid obliteration. This is a result of the individual variability in the effectiveness of the circle of Willis, which is frequently the site of abnormalities (Riggs and Rupp, 1963). Unfortunately, no readily available reliable test has been devised which will predict whether or not the clamping which occurs during surgery will be tolerated. As a result, most surgeons make use of a bypass shunt to ensure adequate cerebral perfusion during the disobliteration of the artery (Smellie, 1972; Sharbrough, Messick and Sundt, 1973; Howe and Kindt, 1974). There are surgeons, however, who prefer to avoid a shunt, considering that it impedes the operator and that during its insertion intimal damage may occur. Thus, although endarterectomy can be performed with low morbidity and with an operative mortality approaching 1%, the incidence of sequelae directly attributable to surgery cannot be ignored. In a recent series, De Weese and colleagues (1973) described the results from 103 patients. There was one death during operation and six strokes following surgery, a 6.8% incidence of events directly caused by surgery. Nonetheless, operative hazards are acceptable if the later prognosis of patients can be shown to have been improved significantly by the procedure. Evidence of the value of surgery in extracranial arterial lesions has been provided by the Joint Study of Extracranial Arterial Occlusion. In 1970, Fields and colleagues randomly allocated 316 patients to surgical treatment (169 patients) and to non-operative treatment (147 patients). In the surgical
group there was an 11.2% incidence of serious complications (3.5% mortality; 7.7% postoperative strokes) compared with a 1.4% incidence (one death and one major stroke) in the non-operated group. Despite this high incidence of early problems, however, the number of patients remaining symptom-free within the average follow-up period of 42 months was overwhelmingly in favour of the surgical group. Furthermore, Bauer and colleagues (1969) indicated that long-term survival in patients with transient cerebral ischaemia resulting from carotid stenosis was better following surgery than was obtained by non-operative treatment.

**ANAESTHETIC CONSIDERATIONS**

Patients about to undergo carotid endarterectomy are frequently elderly and hypertensive, and may have generalized arterial disease. If a temporary bypass shunt is not employed and arterial occlusion occurs, it is obvious that the remaining cerebral arteries, which may be diseased themselves, must supply the tissue nourished previously by the occluded vessel. As a result, local anaesthetic techniques such as cervical plexus block have been advocated (Sublett, Seidenberg and Hobson, 1974). The theoretical advantages of local anaesthesia are minimal alterations in the normal cardiovascular and respiratory functions of the patient along with the ability to monitor the patient’s level of consciousness and neurological state. In this way, immediate warning of cerebral ischaemia may be achieved. However, in many ways, general anaesthesia has proved to be more suitable. Incidental advantages include the ability to control the patient’s respiration and so maintain arterial $P_{O_2}$ and $P_{CO_2}$ at optimal values. The potential hazards of airway obstruction and of agitation in the uncooperative patient are avoided. Undoubtedly, general anaesthesia is much more pleasant for the patient. In addition, the anaesthetist can attempt in various ways to provide additional protection to the brain during surgery by his ability to

1. control the carbon dioxide tension and hence either increase or decrease cerebral blood flow,
2. increase cerebral perfusion through an increase in arterial pressure,
3. produce deep anaesthesia usually with halothane or barbiturates,
4. induce general hypothermia,
5. use hyperbaric oxygenation.

Unfortunately there is no general agreement as to the best combination of these variables (Smith and Wollman, 1972); thus it is probably worth while considering the advantages and disadvantages of each before delineating in more detail possible techniques of general anaesthesia.

**Hypercapnia.** The evidence in favour of hypercapnia is straightforward—it increases cerebral blood flow (Kety and Schmidt, 1948; Sokoloff, 1960; Reivich, 1964). However, this concept can be criticized on the grounds that it may decrease merely the cerebrovascular resistance on the side of the patent carotid artery and so “steal” blood flow which would otherwise have crossed to supply the compromised hemisphere. For example, it has been demonstrated in animals that, following surgical ligation of the middle cerebral artery, higher arterial $P_{CO_2}$ tensions were associated with larger infarcts (Soloway et al., 1968; Battistini et al., 1969). Using a similar preparation, Symon (1970) found that arteriolar pressure distal to the arterial occlusion was decreased by the administration of carbon dioxide. In patients, Boysen and colleagues (1971) demonstrated that hypercapnia, in contrast to normocapnia and hypocapnia, was associated with decreases in both internal carotid artery occlusion pressure and the efficiency of autoregulation. Similar findings have been noted by Ehrenfeld and colleagues (1970) and Fourcade and colleagues (1970). In addition, Pistolese and colleagues (1971) demonstrated that, although hypercapnia would increase cerebral blood flow when the carotid artery was patent, it had no effect on cerebral blood flow once the artery had been occluded. The constant finding of higher internal carotid artery occlusion pressures during moderate hypocapnia than during hypercapnia (Ehrenfeld et al., 1970; Fourcade et al., 1970; Boysen et al., 1971) speaks in favour of using the former since, other factors being equal, a higher pressure distal to the occlusion will result in an increased blood flow to regions with local vasodilatation. In contrast, laboratory investigations of squirrel monkeys have indicated a detrimental effect from a decreased arterial $P_{CO_2}$ tension (Michenfelder and Sundt, 1973). It would seem reasonable, therefore, in the clinical situation to maintain arterial $P_{CO_2}$ near to the normal value for the particular patient undergoing the operative procedure. This means that, in those patients with chronic obstructive pulmonary disease with an increased preoperative or baseline arterial $P_{CO_2}$, a greater than normal $P_{CO_2}$ will be required.

**Deliberate hypertension.** Deliberately increasing the
arterial pressure would seem to be a more hopeful procedure, in that it could be anticipated that the normal areas of brain would autoregulate to hold flow constant, while those areas in which local perfusion pressure had decreased below the autoregulatory limits would benefit. In patients subjected to ligation of the middle cerebral artery, hypertension decreased markedly the area of ischaemia (Barbistini et al., 1969). Hypertension has been observed also to reverse neurological symptoms in patients with cerebrovascular insufficiency (Farhat and Schneider, 1967). However, drug-induced hypertension may not be the whole answer. If the patient has a good collateral circulation, the pressure distal to a carotid occlusion may be increased by increasing the arterial pressure, but such patients are much less at risk anyway. In those patients with poor collateral circulations, an increase in arterial pressure will influence minimally the carotid artery occlusion pressure (Boysen, Engell and Henriksen, 1972). Thus local perfusion pressure cannot be improved significantly when it is most needed. However, although agreement on the use of induced hypertension is not complete, certainly no one advocates hypotension. It will be appreciated that if a patient with a complete carotid occlusion is rendered hypotensive, cerebral infarction may occur because at a low arterial pressure a marginally adequate anastomotic flow may become inadequate (Adams, 1967). In addition, it must be remembered that many of these patients are hypertensive normally so that an apparently "normal" arterial pressure during anaesthesia may be inadequate to maintain cerebral perfusion. The other danger which arises from acute hypotension in a patient with a partial carotid stenosis is that the slowing of the blood flow may initiate thrombus formation at the site of the stenosis.

Deep anaesthesia. Deep anaesthesia, by decreasing cerebral oxygen requirements, is theoretically attractive. The total energy requirements of the brain are the resultant of two major factors: the energy required for the maintenance of cellular integrity and that required for the maintenance of cerebral function (Michenfelder and Theye, 1970). The mechanism whereby anaesthetic agents alter cerebral metabolic activity is not known, although they are thought primarily to alter cerebral function and this, in turn, determines alterations in energy requirements (Theye and Michenfelder, 1968). Anaesthetic agents alter that aspect of cerebral function responsible for the maintenance of the conscious state and since other aspects of cerebral function need not be affected to a similar degree, the anaesthetic state produced by different agents may be associated with different cerebral metabolic rates (Michenfelder and Theye, 1970) and so could, in theory, induce different degrees of cerebral protection. For example, Smith and colleagues (1975) studied the effects of seven anaesthetic techniques on dogs in which the right middle cerebral and internal carotid arteries had been ligated. They found that most animals in the "awake" and "light halothane" groups developed unilateral weakness and had infarction of approximately 10% of the hemisphere. Deep halothane anaesthesia, even with normal arterial pressures, was associated with massive infarctions and severe hemiplegia in almost all animals. In contrast, only one of the 18 animals given barbiturates developed any neurological lesion—a mild transient hemiparesis. In addition, those animals given barbiturates (pentobarbitone 25 mg/kg or thiopentone 20 mg/kg) were observed to have small or absent infarcts. In a later study from the same unit it was demonstrated that barbiturates or narcotic anaesthesia would protect against the development of cerebral oedema (Smith and Marque, 1975). Moreover, Michenfelder and Sundt (1975) have observed that there was significantly better survival and significantly less neurological deficit in primates with permanent middle cerebral artery occlusion given barbiturates, compared with similar animals not given barbiturates. It is possible that one factor involved in this protective action of barbiturates is the well-known decrease in intracranial pressure produced by them which would result in an increase in cerebral perfusion pressure. The most obvious reason, however, would be that the decreased cerebral oxygen requirements under barbiturate anaesthesia had protected areas of brain which were partially ischaemic. Nonetheless, one must recognize that deep halothane anaesthesia produces also fairly major decreases in cerebral oxygen uptake, although these do not equal the decreases produced by the barbiturates (2% halothane decreases cerebral metabolic rate for oxygen by approximately one-third, while deep barbiturate anaesthesia causes a decrease of about one-half).

Hypothermia. Hypothermia is another technique which may be useful as an adjunct to carotid surgery on account of its metabolic effects. Hypothermia decreases cerebral metabolic rates, presumably by the direct depressant action of decreased temperature on the rates of all enzymatic reactions. Accordingly, both major determinants of cerebral energy requirements (see above) would be affected equally. Rosomoff (1959) has shown that hypothermia, as compared
with normothermia, decreased the size of infarcts in dogs with middle cerebral artery ligations. It has been shown also that the duration of circulatory arrest required to produce permanent neurological deficits in animals is increased from 4-6 min (normothermia) to 20-29 min under hypothermic conditions (Anabtawi and Brockman, 1962). Although hypothermia to 30 °C was used originally to protect the brain during operations on the carotid bifurcation, at this temperature the theoretical anoxic period should not exceed 8 min, which is insufficient time for most carotid artery reconstructions. The disadvantages of hypothermia are that it is time-consuming and may significantly prolong the anaesthetic. In addition it is not without morbidity, cardiac dysrhythmias and damage to the skin and subcutaneous tissue being documented. Although few centres advocate the use of hypothermia, it may play a part in the patient with severe disease who will not have a shunt placed during the operation (Smith and Wollman, 1972).

Hyperbaric oxygenation. The use of hyperbaric oxygen to improve the oxygen delivery to the brain during carotid endarterectomy has been recommended by Homi and colleagues (1966) and by Jennett and colleagues (1969). Although it is obvious that this technique cannot be of use in areas of total ischaemia (Jennett et al., 1969), it has the two advantages of increasing cerebral oxygenation and constricting cerebral blood vessels in normal brain (Jacobson, Harper and McDowall, 1963) but not those in ischaemic brain (Harper, Ledingham and McDowall, 1965). It is evident, however, that this form of treatment is limited by the availability of walk-in hyperbaric chambers.

In assessing these methods of cerebral protection, measurements have been made of jugular venous oxygen tension (Viancos et al., 1966; Galbraith, 1967; Jenkins and Chung, 1969). This may be useful in so far as it is known that jugular venous \( P_{O_2} \) of less than 20 mm Hg would be unacceptable. However, the jugular venous oxygen tension is an average of the whole hemisphere and a normal value is no guarantee that areas of severe hypoxia do not exist within the wide drainage area of the jugular bulb (Meyer et al., 1962). Thus, assessment of the jugular venous oxygen tension has not proved to be of particular help in predicting the occurrence of focal neurological deficits (Larsen et al., 1967).

During administration of the anaesthetic, however, the condition of the brain may be monitored with continuous electroencephalograms (e.e.g.) and by intermittent measurements of regional cerebral blood flow. A good correlation between changes in the e.e.g. and the cerebral blood flow has been shown by Sharbrough, Messick and Sundt (1973) during carotid artery surgery.

Having discussed certain of the theoretical implications affecting anaesthesia for carotid artery surgery, I would like to conclude by describing certain anaesthetic techniques which are in current use and, in this way, it may be possible to see the outworking of the theoretical in the clinical situation. To do this, I have taken the liberty of describing in some detail the anaesthetic technique in use at the Mayo Clinic (Sharbrough, Messick and Sundt, 1963). Variations of this basic technique, in use in other centres, will be considered later.

Premedication consists of atropine with an opiate, diazepam or both. On arrival in the anaesthetic room, a brief neurological examination and a baseline e.e.g. are followed by the induction of anaesthesia with thiopentone, and endotracheal intubation facilitated by suxamethonium administered i.v. Anaesthesia is maintained with halothane in a 50% nitrous oxide/oxygen mixture and ventilation is controlled mechanically. A non-depolarizing muscle relaxant is given to permit the artificial ventilation. Percutaneous cannulation of either a radial or dorsalis pedis artery permits both continuous direct arterial pressure monitoring and intermittent sampling of arterial blood for blood-gas determinations. Arterial \( P_{CO_2} \) is maintained between 35 and 46 mm Hg and the arterial oxygen tension greater than 110 mm Hg. Care is taken to avoid hypotension throughout anaesthesia, with the use of appropriate vasopressor therapy when indicated. Cerebral blood flow is determined intermittently and the e.e.g. is monitored continuously. A bypass shunt is used in all patients. In addition, a continuous oscillographic display of the electrocardiogram is monitored throughout administration of the anaesthetic (patients with carotid artery disease frequently have other cardiovascular pathology). Heparin is given i.v. before the carotid artery is clamped. During occlusion, the arterial pressure is increased deliberately by 15-20% except in those patients with baseline systolic arterial pressures of 175 mm Hg or greater. The additional increases in arterial pressure are achieved by the use of an i.v. infusion of phenylephrine (0.002%).

At the conclusion of the procedure, the arterial cannula is removed, any persistent neuromuscular blockade is reversed and the trachea is extubated. In the recovery room the patient receives 40% oxygen via a close-fitting face mask and is evaluated neuro-
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logically before being transferred to the neurological intensive care unit.

The most widespread modifications of this basic technique are the supplementation of the nitrous oxide/oxygen anaesthesia with either narcotics (fentanyl or pentazocine) or barbiturates (thiopentone or methohexitone) in place of the halothane. Under conditions of narcotic supplementation, decreases in the patient’s arterial pressure are less likely and the drug-induced increase of arterial pressure may be unnecessary.

One must point out also that Kenyon, Thomas and Goodwin (1972) use heparin, administered systemically and regionally, as the sole protection to the cerebral circulation with no restriction placed on the duration of carotid occlusion.

In summary, therefore, although the theoretical implications of anaesthesia for carotid artery surgery are debatable still, it would appear that a regime of:

(i) general anaesthesia at normothermia and supplemented by barbiturates, narcotics or light halothane which
(ii) maintains a normal or slightly increased arterial oxygen tension,
(iii) maintains a normal or slightly below normal arterial carbon dioxide tension,
(iv) maintains the systolic arterial pressure at normal or slightly increased values,
(v) employs a bypass shunt in all cases, and
(vi) makes use of transient systemic heparinization

would afford the best possible protection to the patient during the period of operation. Looking into the future, it seems likely that the particular anaesthetic technique shown eventually to be that best suited to carotid artery surgery will be the technique to adopt for surgery on the smaller and more distal cerebral arteries.

REFERENCES


We analyzed 1,145 consecutive carotid endarterectomies for carotid artery ulcerative stenosis which were categorized into four groups according to preoperative risk factors and monitored with intraoperative cerebral blood flow measurements and electroencephalograms and with postoperative electroencephalograms and retinal artery pressure measurements. Like the earlier multicenter General Anesthesia (GA) versus Local Anaesthesia (LA) for carotid surgery (GALA) trial, a recent independent study showed no difference in transient ischemic attack, stroke, myocardial infarction, and death rates between carotid endarterectomy (CEA) performed under LA compared with GA.