ANAESTHESIA FOR CAROTID ARTERIAL SURGERY

BY

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The surgical correction of stenosis and other lesions of the carotid arteries is now increasingly common. This is due in part to wider recognition of the frequency of these conditions and to improved radiological techniques in diagnosis; but mainly to advances in anaesthesia and surgery which have made possible procedures formerly prevented by associated tissue damage due to prolonged ischaemia.

THE PROBLEMS

The problems of anaesthesia in these cases are twofold: primarily, that of preventing anoxic brain damage during periods of arterial occlusion; and secondly, that of the management of a group of patients of advancing years, who in the majority of instances are suffering from generalized degenerative arterial disease and often from its accompaniments of ischaemic cardiac disease or cerebrovascular lesions.

Carotid and vertebral angiography, used in the diagnosis and pre-operative assessment of these cases, reveal a considerable variation in the degree of pre-existing encroachment on the normal cerebral arterial inflow with which patients may be presented for surgery. This may range from a partial stenosis of one carotid artery only, to cases in which there is involvement in some degree of both carotids and perhaps of the vertebral arteries in addition. Rob and Wheeler (1957) have emphasized that those cases involving clamping of a partially stenosed internal carotid artery, or those in which the common carotid is clamped below a total block of the internal carotid (thus interfering with collateral circulation), will suffer brain damage unless hypothermia is employed. Cases in which only a vessel already totally occluded by disease is to be clamped obviously undergo no reduction in cerebral blood flow and do not merit hypothermia. Thus careful assessment of the type and site of the lesion influences the decision to employ this technique.

Bering et al. (1956), using monkeys, showed that the relationship of brain oxygen consumption to temperature was not linear but S-shaped, and suggested that for reasonable benefit during neurosurgery the temperature should be lowered to at least 30°C, since little reduction in brain metabolism occurs above 31°C, while oxygen consumption at 30°C drops to 50 per cent of normal. Thus hypothermia to 30°C is aimed at in these patients, and levels below 28°C are avoided in view of the increased liability to cardiac arrhythmia below this temperature.

The use of hypothermia probably has a secondary protective function in preventing intracerebral thrombosis during periods of arterial occlusion, by way of its effect on coagulation and the level of circulating platelets (Macfarlane, 1948).

Stone et al. (1956) demonstrated that the onset of shivering during hypothermia could raise brain oxygen consumption by over 100 per cent and it is essential that this is not allowed to occur, particularly during periods of carotid clamping.

METHOD OF HYPOTHERMIA

The method of hypothermia employed has been that of surface cooling, using either icebags and fans, or a collapsible canvas bath for total immersion of the patient. This method, though slower than pervascular techniques, has the great virtue of simplicity and there is a relative absence of morbidity due to the technique alone. Temperature is recorded by a direct reading thermistor thermometer with leads inserted a measured distance into the oesophagus (thus lying closely related to the heart) and into the rectum. The oesophageal lead has been shown by Cooper and Kenyon (1957) to provide a reliable index of blood temperature during cooling, and the rectal lead, though slower and less accurate in recording temperature trends during cooling, has proved
useful in following temperature changes at the slower rate of rewarming in the ward.

Electrocardiographic monitoring at frequent intervals during cooling is desirable, particularly in view of the high incidence of ischaemic heart disease in these patients, and the fluctuations of blood pressure that may occur during manipulation of the carotid sinus. The electrocardiographic record has also proved a useful early indication of shivering.

In earlier patients the method of anaesthesia employed during hypothermia involved the use of the conventional sequence of nitrous oxide and oxygen with pethidine and small increments of d-tubocurarine chloride to prevent shivering, and the use of controlled respiration through an absorber—essentially a modified form of the technique described by Cheatle (1957) and Thornton (1957) for aortic reconstruction under hypothermia in this Unit. It was felt, however, that for carotid surgery, with no necessity for either profound relaxation or controlled respiration, this sequence held certain disadvantages and was open to certain criticism. Firstly, the use of nonvolatile agents such as pethidine and relaxants is associated with delayed excretion of these drugs due to reduced metabolism (Delorme, 1955). Prolonged recovery periods may occur and difficulty may be experienced in instituting adequate spontaneous respiration. Secondly, the reversal of d-tubocurarine with neostigmine at low temperatures may be incomplete due to delayed excretion and depressed acetylcholine formation (Brown, 1954) and may be hazardous in the presence of the bradycardia seen at these temperatures, despite the use of atropine. Recurarization may be seen as rewarming occurs (Zaimis et al., 1958). Thirdly, vaso-dilatation is not marked and hence the rate of cooling may be slow, with a severe after-fall and a danger of overshooting the desired temperature level (Scurr, 1955), due to the existence of a “cold shell”.

These considerations pointed to the desirability of an inhalation agent and led to the use of halothane, the properties of which, by avoiding the drawbacks mentioned, appear to offer certain advantages in hypothermia over the previous technique. In a small number of cases this drug has proved very satisfactory.

**DETAIL OF TECHNIQUE WITH HALOTHANE**

All patients have routine pre-operative chest radiographs and electrocardiograms and estimations of haemoglobin, electrolytes and blood urea are performed. Premedication consists of pethidine 100 mg and atropine 0.65 mg, given 1 hour preoperatively. Anaesthesia is induced with a sleep dose of 5 per cent thiopentone (varying in this series from 200 to 400 mg) followed by suxamethonium 80 mg and atropine 0.65 mg. After inflation of the lungs the larynx is visualized and sprayed with 4 per cent lignocaine and intubation is performed with the largest possible cuffed oral Magill tube. Anaesthesia is maintained using nitrous oxide 5 l. and oxygen 3 l./min, 1 per cent halothane being delivered into an M.I.E. circle absorber from a Fluotec vaporizer. The lungs are ventilated until respiration returns and breathing is then normally unassisted throughout cooling, with the absorber in circuit and with a controlled leak. An infusion of half normal saline is set up and electrocardiographic leads are attached. The thermometer leads are then inserted, the patient stripped and then either packed with ice bags from head to feet or placed in a canvas bath which is filled with iced water at 7 to 9°C. Changes in ambient temperature have little effect on the rate of cooling, which depends primarily on the body weight and on the degree of obesity.

During cooling, recordings are made every 15 minutes of pulse rate, blood pressure, and oesophageal and rectal temperature and the three standard electrocardiograph leads. In order to achieve the desired level of 30°C at the commencement of operation, the ice bags are removed when a temperature of 30.5°C is attained or, when the immersion method is used, the bath is emptied at 33°C. The patient is then dried and covered with a prewarmed electric or circulating blanket which is heated or removed according to temperature trends during the operation in order to minimize after-fall. Over-rapid rewarming during surgery of both carotid arteries has not been encountered.

On completion of the operation the patient is allowed to rewarm at the rate of about 1 to 1.5°C per hour and is kept in the operating theatre until a definite upward trend in temperature is noted and a temperature of 30.5°C has been exceeded. Light anaesthesia is maintained during this period.
with halothane 0.5 per cent in order to prevent severe shivering with its attendant circulatory strain.

Patients are then returned to the ward with the rectal thermometer lead in situ, and breathing oxygen. The progress of rewarming is controlled by covering with blankets and intermittent use of the electric blanket, if necessary, while shivering is prevented by the use of pethidine.

A chart of the course of cooling, with blood pressure and pulse trends during this procedure in a typical case of this series is shown in figure 1.

Instead of assuming the cyanotic tinge of cold stasis so often seen during hypothermia. After-falls in temperature are lessened—presumably because of the absence of a thick cold shell with its associated temperature gradients. In addition the danger of vascular collapse due to dilatation replacing vasoconstriction during rewarming would seem to be reduced. The lessened hazard of cold necrosis in well dilated extremities was noted by Gray (1955) and, in fact, no complications of this sort were experienced.

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The prevention of shivering was very effective and halothane concentrations of less than 1.5 per cent were invariably adequate for this purpose. At this light level of anaesthesia the blood pressure reduction was small, provided that additional atropine was given at the time of induction of anaesthesia and respiration appeared clinically adequate, with no necessity for manual assistance.

Elimination was rapid, with short recovery periods. Early recovery of consciousness was seen in several patients at temperatures of 30.5°C, a point of importance in the prophylaxis of postoperative chest complications.
In the majority of these cases blood replacement was unnecessary.

**pH, Pco₂, and electrocardiographic changes**

Figure 2 shows the pH and Pco₂ changes occurring in arterial blood during cooling in four of the patients anaesthetized with halothane. Samples for these estimations were taken from the femoral arteries into heparinized syringes, and pH measured by the method of Astrup and Schrøder (1956), using a direct-reading radiometer pH meter, having restored the samples to 38°C. Results were corrected for temperature (for these purposes no account was taken of variation in haemoglobin, plasma proteins, etc.) and Pco₂ values were obtained from the Singer-Hastings nomogram, having estimated total carbon dioxide values by a modification of the manometric method of Van Slyke. All patients show the expected increasing acidosis as cooling proceeds, yet clinically this produced no adverse signs and prolonged recovery periods or profound blood pressure falls after cessation of anaesthesia were not seen.

Black et al. (1959) demonstrated during halothane anaesthesia at normal temperature a definite Pco₂ threshold in each case above which cardiac arrhythmia occurred. They showed that the magnitude of this figure varied inversely with the systolic pressure while bearing no relationship to actual inspired halothane concentrations. It seems probable that the use of this drug in hypothermia is associated with a lessened incidence of serious cardiac arrhythmia.

Electrocardiograms in these patients show the normal changes of hypothermia, including bradycardia with widening of PR interval QRS complex and ST segment. Ventricular ectopic beats occurred infrequently at temperatures of 30° to 31°C, and were the commonest arrhythmia seen. Auricular fibrillation during rewarming was seen in one patient anaesthetized with halothane but this reverted spontaneously to normal rhythm as the temperature rose. Ventricular fibrillation occurred once only at a temperature of 35°C in a patient aged 54 whose pre-operative electrocardiogram suggested cardiac ischaemia. Anaesthesia in this patient was carried out using nitrous oxide and oxygen, pethidine and d-tubocurarine, with controlled respiration. The cause of fibrillation here is not clear but complete recovery followed successful cardiac massage.

**RESULTS**

Over 100 patients, anaesthetized by various members of this department, have been operated on for lesions of the carotid arteries. In 64 unselected examples there were 59 cases of carotid stenosis due to atheroma or thrombus; 2 carotid aneurysms; 2 carotid body tumours; and 1 periarterial haematoma following angiography. Fifty-three patients were operated on under hypothermia and 20 of these were anaesthetized with halothane.

The average duration of carotid artery clamping for thrombo-endarterectomy was 20 minutes and the longest period recorded in this series was 35 minutes at 29.5°C in a patient with a partial stenosis of the internal carotid artery.

Complications were few and recovery was uneventful in the majority of patients. There were 4 deaths, of which 2 only could be remotely associated with anaesthesia. The first of these was due to pulmonary embolism and myocardial infarction on the second postoperative day and followed uneventful anaesthesia without hypothermia for a total carotid thrombosis. The second occurred from bronchopneumonia after operation under hypothermia in a patient with a pre-
existing hemiplegia and disordered consciousness due to an acute carotid thrombosis. No deaths occurred in the 20 patients anaesthetized with halothane. There were 3 instances of transient auricular fibrillation during rewarming (1 following halothane) and 1 case of left basal pneumonia following hypothermia.

SUMMARY
The problems of anaesthesia for surgery of the carotid artery are discussed. A method of hypothermia with halothane anaesthesia, used in certain of these cases, is described. The advantages of using this drug are enumerated.

It is believed that the properties of halothane render it particularly suitable for use with hypothermia in this type of case.

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REFERENCES


