ELECTIVE CIRCULATORY ARREST IN NEUROSURGICAL OPERATIONS

BY

T. V. CAMPKIN AND S. H. DALLAS

SUMMARY

Elective peripheral circulatory arrest under moderate hypothermia in neurosurgical operations on intracranial aneurysms and angiomata gives improved operating conditions and a lower mortality and morbidity than previous methods. Circulatory arrest can be achieved by means of intracardiac pacing and placement of a balloon catheter in the ascending aorta, at the same time allowing adequate coronary artery perfusion. The management and complications in a series of thirty-seven consecutive cases are described and discussed.

It has been established that temporary circulatory arrest at moderate levels of hypothermia is of great value during certain operations on intracranial aneurysms and angiomata (Small and Stephenson, 1966). When the cerebral blood flow is arrested, exposure and manipulation of the lesion is safer since the risk of rupture of an aneurysmal sac or of serious haemorrhage from an angioma is greatly reduced.

In this unit an open-chest method has been employed involving simultaneous craniotomy and median sternotomy. Short periods of circulatory arrest of up to 8 minutes at 31°C are provided for the surgeon by aortic occlusion using soft clamps. This technique, however, inflicts considerable extra trauma and inevitably prolongs the period of postoperative recovery (Campkin, 1965).

In order to avoid the morbidity associated with thoracotomy, a closed-chest technique of obtaining circulatory arrest has been developed over the last two years. This method has been employed mainly in the operative treatment of anterior cerebral aneurysms (particularly those arising on the anterior communicating artery) and middle cerebral lesions. It is in these groups that the highest operative mortality was incurred prior to the introduction of a technique utilizing circulatory arrest (Campkin and McNeil, 1964).

Under moderate hypothermia (30.5–31.5°C) a balloon catheter is introduced into the ascending aorta. An endocardial pacemaking electrode is also introduced into the right ventricle. When circulatory arrest is required the systolic blood pressure is lowered by employing a high pacing rate. The balloon is then inflated with saline in order to occlude the proximal ascending aorta. The cardiac rate can now be reduced allowing the pressure on the proximal side of the balloon to rise, hence ensuring an adequate coronary artery perfusion pressure, while maintaining complete circulatory arrest distal to the inflated balloon.

This paper describes the anaesthetic management of these patients. The cardiological aspects are described in a communication elsewhere (Davison et al., 1968).

METHOD

Premedication usually consists of atropine 0.6 mg and promazine 25 mg given intramuscularly 45 minutes before the start of anaesthesia. Induction is achieved using thiopentone and is followed by suxamethonium. The larynx and trachea are sprayed with 4 per cent lignocaine and intubated with a cuffed nylon reinforced latex tube. Midoesophageal and rectal thermocouples are inserted and an intravenous infusion of dextrose-saline set up in an ankle vein. Spontaneous ventilation with nitrous oxide, oxygen and halothane (1–2 per cent) is allowed until the patient is placed on the operating table. Electrocardiographic leads are strapped on the limbs and the electrocardiogram monitored continuously using an oscilloscope (Cardiorater, Cardiac Recorders Ltd.). Active surface cooling is not used at this stage but the patient is completely exposed and the body temperature starts to fall slowly.

Using image intensifier control (fig. 1) a transvenous endocardial pacemaking electrode is
introduced into the apex of the right ventricle. When it is in the correct position the cardiac rhythm can be captured at 1 volt.

A right femoral arteriotomy is now made and a modified Mahajan-Clifton triple lumen catheter (United States Catheter Corporation) inserted and passed up the aorta under fluoroscopic control until the balloon lies in the proximal part of the ascending aorta. The position of the balloon may be checked by distending it with radiopaque dye. The openings in the catheter on either side of the balloon are connected via strain-gauge transducers (Devices) to a four-channel oscilloscope. The aortic pressure is thus continuously monitored via two transducers and before inflation of the balloon the two pressures are, of course, identical. After distension of the balloon the proximal opening measures coronary artery pressure and the distal opening measures the peripheral arterial pressure which will be at or near zero. The electrocardiograph and sometimes the central venous pressure are also continuously displayed.

The induction of anaesthesia and catheterization of the aorta occupy 1-2 hours and during this time the patient's oesophageal temperature falls to approximately 33-34°C. The patient is now transferred to the operating table (fig. 2), placed between tubulated blankets and further surface cooling maintained using Selectotherm Hypothermia apparatus. Shoulder-blade defibrillating electrodes are strapped to the chest so that DC shock can be applied in the event of ventricular fibrillation.

Controlled pulmonary ventilation using a Blease Pulmoflator is instituted after the injection of tubocurarine 15-20 mg. Anaesthesia is maintained with 50 per cent nitrous oxide, 50 per cent oxygen and 1 per cent halothane. Tidal volume is adjusted to 500-600 ml and a subatmospheric phase of -5 cm H$_2$O provided. Deliberate hyperventilation is not employed and the authors consider it desirable to maintain Pco$_2$ at normal levels (after suitable correction for the patient's temperature).

Rewarming is started at approximately 33°C (oesophageal) because an after-drop in temperature always occurs. At this level of hypothermia and with the form of anaesthesia employed, the aortic systolic blood pressure usually stabilizes at about 80 mm Hg.

Shortly before circulatory arrest is required, propranolol 2-3 mg is injected slowly intravenously. Propranolol slows the cardiac rate and usually produces a slight fall in systolic arterial pressure to 50-70 mm Hg. It also reduces the incidence of ectopic ventricular disturbances during and immediately after the arrest period. Larger doses of propranolol are unnecessary and dangerous because a marked fall in blood pressure and cardiac arrest may occur (Johnstone, 1966).

Circulatory arrest is produced when the neurosurgeon requires a bloodless field, usually for the application of a clip to an aneurysmal sac. The patient's oesophageal temperature at this stage is between 30.5 and 31.5°C. The anaesthesia is discontinued and controlled ventilation stopped. The heart is now paced artificially at a rate varying from 120 to 180 beats/min. Inadequate diastolic filling occurs, the cardiac output falls and the aortic pressure drops to 20-30 mm Hg. The balloon is now rapidly inflated with isotonic saline. When the aorta is occluded the pacing rate is reduced and the pressure on the cardiac side of the balloon is allowed to rise, so ensuring adequate coronary artery perfusion. Circulatory arrest may be maintained for periods of up to 8 minutes at 31.5°C and may safely be repeated after an interval during which the cardiovascular system is allowed to return to normal. In the majority of cases, however, an arrest period of less than 4 minutes was required.

Fifty ml of 8.4 per cent sodium bicarbonate (50 m.equiv) is infused during each arrest period to compensate for the metabolic acidosis which occurs. Low molecular weight dextran is also given intravenously (200-500 ml) with the object of preventing sludging in small cerebral vessels. Blood is warmed to 40°C before transfusion because the rapid infusion of cold blood will precipitate a further fall in temperature and marked electrocardiographic changes may be seen.

Anaesthesia is recommenced immediately after the arrest period and there is always a marked post-arrest rise in blood pressure when the circulation is re-established. This is due to the release of pressor amines and can be reduced by an increase in the halothane concentration and a further small dose of propranolol.

The catheters are withdrawn as soon as the need for circulatory control has passed and the femoral
FIG. 1
The patient in the X-ray department prepared for the insertion of the aortic catheter, showing the image intensifier and monitors and the cardio-oscilloscope, pacemaker and defibrillator.

FIG. 2
The patient in position on the operating table, showing the tubulated blanket, the Blease Pulmo-illator, the cardiac monitor, pacemaker and defibrillator, and the four-channel Devices recording apparatus.
arteriotomy is repaired. Rewarming of the patient is continued until the oesophageal temperature has risen to 33–34°C. Halothane is discontinued and atropine 0.6 mg and neostigmine 2.5 mg given to restore spontaneous ventilation. The patient returns to the ward with a temperature of 34°C and promazine is given intramuscularly in small doses to prevent shivering.

RESULTS
Forty-six periods of circulatory arrest have been achieved in 37 patients, 15 male and 22 female, aged 12 to 62 years (average 42). The pathological conditions and durations of arrest are summarized in table I. In 24 patients, including 5 who underwent two periods of arrest there were no postoperative complications attributable to the method.

<table>
<thead>
<tr>
<th>Operations performed with circulatory arrest at 30.5-31.5°C.</th>
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<td>No. of lesions</td>
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<tr>
<td>Anterior cerebral and anterior communicating aneurysms</td>
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<td>Posterior communicating aneurysms</td>
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One patient had both an anterior communicating and an internal carotid aneurysm operated on at the same operation. The average duration of arrest was 3 minutes 10 seconds.

There were two deaths.

CASE 1.
A female aged 49 years was operated on for a large carotid bifurcation aneurysm. She was hypertensive (180/100 mm Hg) and there was cardiac enlargement. Control of bleeding when the balloon was inflated was not successful and serious haemorrhage occurred which was eventually controlled.

In this case it is possible that the balloon had slipped down the aorta so that the innominate artery was still perfused. The operation ended uneventfully but sudden death occurred 3 hours postoperatively. Postmortem examination showed gross coronary atheroma but there was no obvious occlusion and no other obvious cause for death.

CASE 2.
A female aged 39 years died 8 weeks after operation. She had suffered a subarachnoid haemorrhage 10 weeks previously, was aphasic and a left hemiparesis was present. Postoperatively she never regained consciousness fully and had developed a right hemiplegia. A femoral thrombosis was treated with anticoagulants but emboli were thought to have lodged in the reticular activating system and in the cerebral hemisphere on the side opposite to the middle cerebral aneurysm undergoing operation. In addition the groin wound was infected.

In another patient femoral artery thrombosis was successfully treated with anticoagulants, and in a further five the groin wounds became infected. One patient who suffered from chronic bronchitis developed a chest infection after operation. This responded satisfactorily to treatment with tetracycline. Another patient who suffered from angina of effort showed evidence of cardiac irregularities and T-wave inversion following arrest. These changes were assumed to indicate a relative anterior ischaemia. They disappeared without treatment within 30 minutes.

CASE 3.
A fit man aged 19 years developed ventricular fibrillation at the end of 8 minutes arrest. Ten attempts at external defibrillation during external cardiac massage were unsuccessful. Thoracotomy was then performed and internal defibrillation was also unsuccessful until the heart was doused with warm saline when spontaneous rhythm was immediately restored.

Failure to defibrillate was attributed to a long period of hypothermia (nearly 1½ hours at 31°C) before arrest and too much propranolol (7.5 mg instead of the recommended maximum of 5 mg).

In two cases the balloon burst on inflation.

CASE 4.
In this case adequate hypotension was obtained for 2 minutes by pacing the heart as in the method of Small and associates (1966). Ventricular fibrillation then occurred but external cardiac massage and external defibrillation restored the circulation to normal; the total period of hypotension was 6½ minutes. There were no subsequent ill effects.

CASE 5.
An initial arrest of 5½ minutes was followed by rebound hypertension, the pressure reaching 140 mm Hg. Ten minutes later a further period of arrest was necessary to control bleeding and after 3 minutes the balloon burst. The blood pressure was kept down to 30 mm Hg by pacing, and bilateral carotid artery compression in the neck enabled the aneurysmal sac to be clipped.
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DISCUSSION

In a previous communication the advantages of circulatory arrest during operations on intracranial aneurysms and angiomata were discussed (Campkin, 1965). The majority of these operations are undertaken for the treatment of anterior, anterior communicating and middle cerebral artery aneurysms, but the technique may be indicated in other cases because of the size or anatomical localization of the lesion. Rupture of an aneurysm during manipulation or exposure may produce disastrous haemorrhage and attempts to control the bleeding, combined with excessive retractor pressure, may damage vessels supplying the hypothalamus or other vital areas.

The technique described has been developed after 8 years experience utilizing both profound hypothermia and moderate hypothermia combined with circulatory arrest. Profound hypothermia has serious disadvantages and it is of interest that most neurosurgical units have now abandoned its use. Cardiac bypass requires heparinization, and its reversal and the problem of postoperative oozing after neurosurgical operations when this technique is used have never been satisfactorily overcome. Furthermore, although profound hypothermia will allow periods of circulatory arrest of up to 45 minutes, this duration is not required. In the majority of the cases in this series the arrest period was less than 4 minutes.

The technique previously employed in this centre involving median sternotomy and aortic occlusion proved a considerable advance on methods hitherto used. The operative mortality associated with lesions of the anterior and middle cerebral vessels was reduced and the technique was used in 38 patients. It is still suitable for emergency cases when the services of a cardiologist are not available to carry out the closed-chest technique. There is, however, an increased postoperative morbidity associated with lesions of the anterior and middle cerebral vessels was reduced and the technique was used in 38 patients. It is still suitable for emergency cases when the services of a cardiologist are not available to carry out the closed-chest technique. There is, however, an increased postoperative morbidity associated with the open-chest method and the technique is no longer used routinely.

The method described in this communication has been used in 37 cases and there has been one death in the immediate postoperative period, the cause of which is obscure but probably not attributable to the technique. One death occurred 8 weeks postoperatively and was probably due to thrombo-embolism from blood clot on the catheter.

The technique has two disadvantages, viz. thrombo-embolic complications and the occasional occurrence of serious ventricular dysrhythmias.

Cerebral thrombo-embolic complications developed in two cases postoperatively and four cases developed symptoms of ischaemia in the right leg. With one exception these occurred early in the series and attempts to reduce the time that the aortic catheter is in situ, combined with meticulous attention to removal of clot from the femoral artery, has reduced the incidence of this complication.

Ventricular arrhythmias are less serious. Ventricular fibrillation occurred on six occasions in four patients and in three patients defibrillation was immediately successful. In one man open-chest cardiac massage was required and normal rhythm was only restored after the heart had been warmed with saline. In this patient prolonged hypothermia and an excess of propranolol were probably the cause of the ventricular fibrillation.

In many operations on intracranial aneurysms a complicated technique of this kind is not required and in these patients moderate hypothermia and controlled hypotension provide satisfactory operating conditions for treatment of the lesion. The technique described in this paper has been evolved over several years to deal with certain groups of aneurysms previously associated with a high operative mortality. This mortality has been reduced and although the method is not required in every case it has proved a valuable advance in the treatment of these dangerous lesions.

REFERENCES


ARRET CIRCULATOIRE SELECTIF DANS LES INTERVENTIONS NEUROCHIRURGICALES

SOMMAIRE
L'arrêt circulatoire périphérique sélectif sous hypothermie modérée a amélioré, lors des interventions neurochirurgicales sur des anévrismes et des angiomes intracraniens, les conditions opératoires et a réduit la mortalité et la morbidité, en comparaison aux méthodes préalables. L'arrêt circulatoire peut être obtenu à l'aide d'un pacing intracardiaque et de la mise en place d'un cathéter à ballon dans l'aorte ascendante, permettant simultanément une perfusion suffisante des artères coronaires. La conduite et les complications dans une série de trente-sept cas consécutifs sont décrites et discutées.

BOOK REVIEW


In a meeting sponsored by a firm of drug manufacturers to stimulate interest in their product, it would be unrealistic to expect the sponsors to exclude papers which were generally complimentary to their product merely because some of the statements contained therein owed more to enthusiasm than to statistical analysis. However, although some of the papers in this report describe clinical impressions rather than controlled trials, others are of real value in establishing the place of diazepam in anaesthesia.

Diazepam (Valium, Roche) is a tranquilizer of the benzodiazepine class, a group which also contains chlordiazepoxide (Librium) and nitrazepam (Mogadon). In a useful introductory paper on the pharmacology of the group Dr. Parkes quotes evidence that the muscle relaxant properties of diazepam are due to interference with interneuronal conduction at spinal levels, and that the tranquillity produced by the drug is due to interruption of conductivity in the limbic system of the cerebral cortex. Although diazepam prevents the rise in blood pressure which usually follows stimulation of the hypothalamus of the cat, the author concludes that diazepam has, in animals and probably in man also, no effects on arterial pressure or respiration beyond those which result from the reduced demand of the tranquilized state, while leaving the respiratory response to carbon dioxide unimpaired. This remarkable lack of toxicity, which is emphasized by the Symposium chairman, Professor Dundee, in his summary, obviously makes diazepam a drug of great interest. The main body of the report is devoted to sections describing its use in premedication, as an induction agent, and after operation.

As premedication, diazepam 10 or 20 mg caused a considerable degree of drowsiness in two-thirds of patients, but was less effective than 100 mg chlordiazepoxide in relieving apprehension. Its lack of antispasmodic activity has been a disadvantage when it is used alone. It is neither analgesic nor antianalgesic and does not cause nausea or vomiting. In his paediatric practice, Bush found that it compared unfavourably with an opiate phenothiazine mixture.

As an induction agent it appears very safe but perhaps is a little unreliable. Possibly due to over-cautious dosage there appears to have been some difficulty in ensuring an adequate depth of sleep in some patients. An interesting paper by Main describes the use of the drug in dentistry in the abnormally apprehensive patient; an injection of local anaesthetic is given under cover of a brief anaesthetic induced by methohexitone. The operation is then completed under sedation provided by intravenous injections of diazepam. Although the use of potent anaesthetic drugs in the absence of a second qualified person must, of course, be condemned, a reliable, safe, intravenous sedative may be, as Professor Dundee described it, "a Godsend to dentists".

This report contains much information of use to anyone considering the inclusion of diazepam in his anaesthetic armamentarium, and is very reasonably priced. The many "clinical impressions" must be taken for what they are worth.

A. A. Gilbertson