

PROFOUND HYPOTHERMIA*

EDWARD F DAW, M D , EMERSON A MOFFITT, M D , JOHN D MICHENFELDER, M D ,
AND HOWARD R TERRY, JR , M D †

OF THOSE PATIENTS who have an intracranial haemorrhage from an aneurysm, approximately half survive. Of the survivors, approximately one third are neurologically well. It is always possible, and indeed probable, that haemorrhage from the aneurysm will recur in a survivor. It is for patients who have survived their initial intracranial haemorrhage with little or no neurological deficit, and for those in whom an aneurysm is discovered incidentally during a complete neurological examination, that advanced measures of treatment have been devised.

Many neurosurgeons prefer to wrap the aneurysm in muscle, plastics, or fascia, some place a clip precisely about the base, and others attempt permanent trapping or reduction of the flow by carotid ligation. Many surgeons use a technique of induced hypotension for the actual period of repair, but this technique is hazardous because of poor tissue perfusion due to hypotension. Some think that, during the period of induced hypotension, the body should be protected against reduced tissue perfusion by application of moderate surface hypothermia.

Of the many methods used in the treatment of aneurysms of cerebral vessels, none is as controversial as hypothermia. The primary objective of hypothermia is protection of vital cerebral structures from hypoxia during the period of repair of the aneurysm. By reducing their oxygen consumption, viability of tissues is preserved during a period of diminished oxygen supply. The choice of technique of hypothermia must be predicated on the skill and dexterity of the surgeon. The surgeon should utilize that technique which best serves his surgical skill for the treatment of a specific aneurysm at a specific location considered in the light of the patient's neurological status and prognosis.

In the early 1950's, when surface hypothermia or hypotension, or both, were utilized at the Mayo Clinic, it became evident that, in a number of patients, neurological deficits developed as a result of the operative procedure. The time required to repair the aneurysm often exceeded the safe period of 8 to 12 minutes without circulation afforded by surface hypothermia of 29° to 30° C. With this in mind, we began to look for other methods of treatment of these patients.

TECHNIQUES OF PROFOUND HYPOTHERMIA

The primary objective of profound hypothermia is the reduction of oxygen consumption to a level such that prolonged periods—30 minutes or more—of

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†Section of Anesthesiology, Mayo Clinic and Mayo Foundation, Rochester, Minnesota.

complete circulatory arrest may be imposed safely. Indications for the use of profound hypothermia in the treatment of intracranial aneurysms vary with each surgeon. In general, they are¹ (1) multiple aneurysms with involvement of one or both carotid arteries, (2) large, broad-based aneurysms which appear to be repairable easily only by collapsing the sac, (3) anticipated difficult exposure and increased risk of rupture, (4) anomalous vascular configuration in the central nervous system.

At the Mayo Clinic, since 1960, three methods have been used for the production of profound hypothermia. These are the Drew technique, the closed-chest technique, and isolated cerebral perfusion.

Drew Method

The Drew technique, or open-chest technique,² consists of substituting two pumps for the heart while the patient's lungs are acting as the oxygenator. During 1960 and 1961, we utilized the Drew technique in 18 surgical procedures.³

Anaesthesia usually was induced by means of a fast acting inhalation agent such as halothane or cyclopropane. Parenterally administered agents were not used because we did not know the fate and duration of action of intravenous drugs in the presence of profound hypothermia. No premedication was given for the same reason. Everything possible was done to minimize coughing and straining during induction of anaesthesia.

After induction of anaesthesia and positioning of the patient on the operating table, devices to monitor arterial pressure, venous pressure, oesophageal temperature, and electroencephalogram were attached and protective devices to guard against dislodging of the endotracheal tube and monitoring equipment were set up. Anaesthesia was maintained with 0.5 to 1 per cent halothane in a mixture of nitrous oxide and oxygen (3:2) flowing at 5 litres per minute in a semi-closed-circle absorbing system. Respiration was controlled by the depth of anaesthesia and hyperventilation by means of a mechanical ventilator.

In this double operation, while the craniotomy was being performed by the neurosurgical team, the cardiovascular team performed the median sternotomy and prepared the inguinal region for the arterial cannulation. Then the patient was heparinized with 90 mg of heparin per square metre of body surface, and cannulation was completed.

To begin the perfusion, blood from the left atrium was pumped through a heat exchanger into the external iliac artery (Fig 1, right). Perfusion and cooling was accomplished utilizing flows from the perfusion apparatus of 2.5 litres per minute per square metre of body surface. When the right atrial pressure increased, indicating right ventricular failure in response to the cold, blood was taken from the right atrium and pumped into the pulmonary artery (Fig 1, left). When an oesophageal temperature of 15° C was reached after periods of perfusion ranging from 13 to 28 minutes,⁴ periods of complete circulatory arrest or periods of low flow (less than 1.0 L per min per M²) were induced, during which the aneurysm was repaired. After testing the repair at varying rates of flow of blood, rewarming was instituted by heating the blood passing through the heat exchanger. A gradient of 12° C was always maintained between the blood leaving the

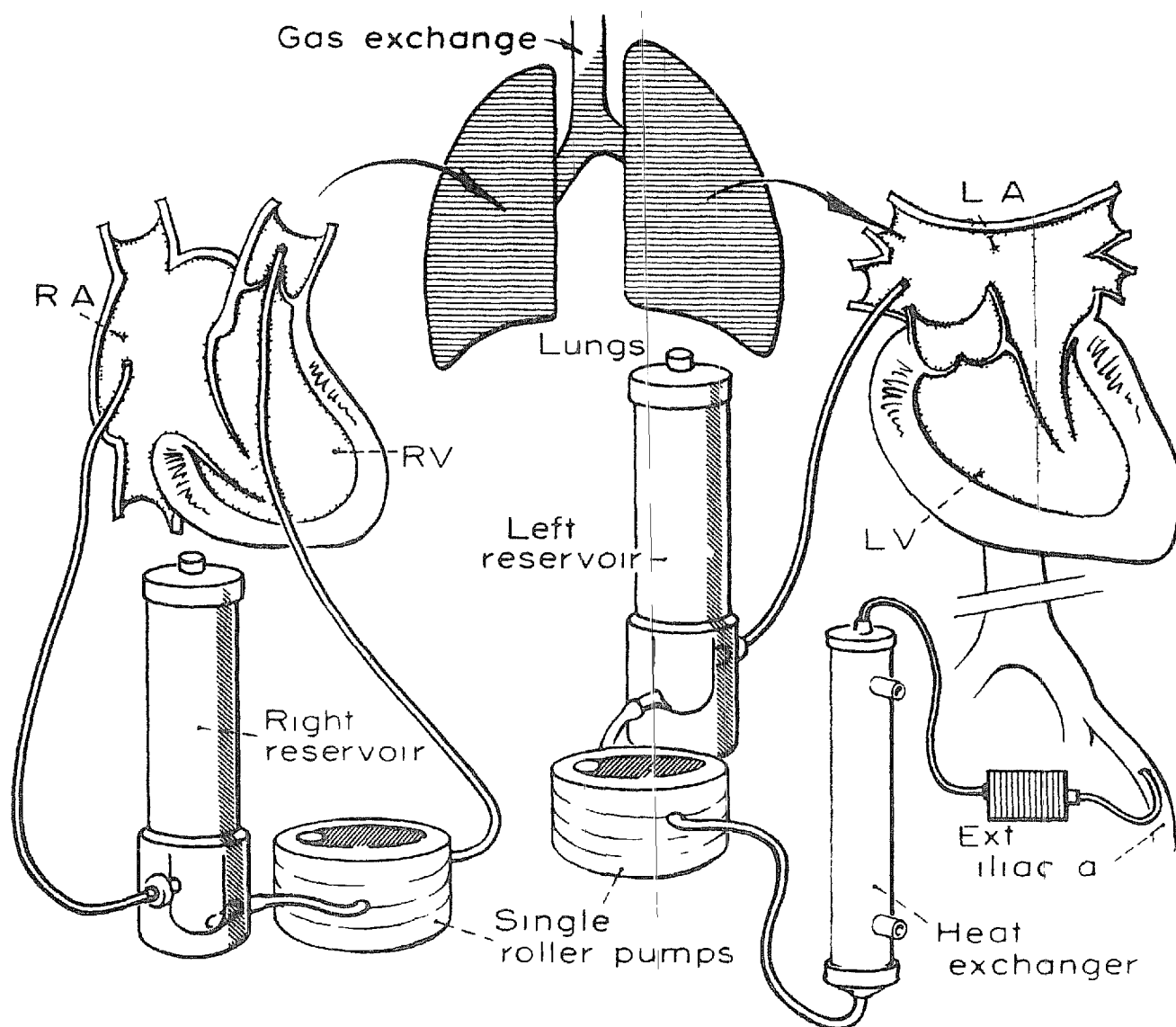


FIGURE 1 Circulation in the Drew (open-chest) perfusion for profound hypothermia (Reproduced with permission from TERRY, H R, JR, DAW, E F, & MICHENFELDER, J D Hypothermia by Extracorporeal Circulation for Neurosurgery An Anesthetic Technic *Anesth & Analag* 41 241 (1962))

heat exchanger and that entering the body to prevent the possible development of gas emboli. After rewarming, the perfusion was stopped and the heparinized state was reversed by administration of protamine or hexadimethrine bromide (Polybrene), 135 mg per square metre of body surface.

It is interesting to note that, in the 18 operations with the Drew technique, only three repairs were completed in the time usually considered to be safe at temperatures used in surface hypothermia. The great disadvantage of the Drew technique has been the large volume of fresh whole blood needed to prime the perfusing apparatus and to maintain the blood volume of the patient after perfusion. The average amount administered to these patients during the operative and immediate postoperative period was approximately 10 litres. In the postoperative period, pain and discomfort at the site of the median sternotomy and pulmonary complications following thoracotomy added to the over-all morbidity and mortality.

Of the 18 surgical procedures performed utilizing the Drew technique, the over-all mortality was 27.7 per cent. Mean arrest time was 25 minutes. Of the 18 patients, 11 had no deficit or minimal deficit in the postoperative period.⁴

Closed-Chest Technique

Because of the disadvantages of the Drew technique (the large amounts of blood required and the morbidity due to intracardiac cannulation and median sternotomy), a closed-chest technique was developed similar to the method of Patterson and Ray.⁵ In this method, a Mayo-Gibbons vertical-sheet pump-oxygenator provides the perfusion and extracorporeal cooling without requiring thoracotomy.

Anaesthesia is induced with either cyclopropane, halothane, or small intravenous doses of thiopental or methohexital. Intubation is accomplished after administration of large doses of a muscle relaxant to prevent coughing and straining which might rupture the aneurysm. As in the Drew technique, anaesthesia is maintained with halothane in a nitrous oxide-oxygen mixture. Respiration is controlled by means of a Bird Anesthesia Assistant-Controller. An inspiratory pressure of approximately 18 to 20 cm of water and expiratory pressure of -4 to -6 cm of water are utilized.

After induction of anaesthesia and intubation, electrocardiographic electrodes are placed. A venous catheter is passed through an antecubital vein into the region of the right atrium. A Cournand needle or Rochester plastic needle no. 16 or 17 is placed in the radial artery, percutaneously or through a small cutdown. Primary monitoring of these patients has been based on the electrocardiogram and on central venous pressure and arterial pressure measured directly by strain-gauge manometers. As with the Drew technique, protective devices are used to ensure that intravenous tubings, corrugated rebreathing tubings, and monitoring devices are not disturbed while the patient is being positioned on the operating table. During the craniotomy the patient's temperature is reduced to approximately 30° C by surface cooling with the Therm-O-Rite blanket.

While the aneurysm is being exposed, the pump-oxygenator is primed with 1500 ml of blood, 1500 ml of 5 per cent glucose in 0.2 per cent saline, and 240 ml of a serum albumin solution. As in the open-chest method, total body heparinization is accomplished by giving 90 mg of heparin per square metre of body surface. The venous lines from the patient to the perfusion apparatus are inserted through the femoral vein and are threaded up into the vena cava to the level of the diaphragm (Fig. 2). An arterial cannula is placed in the femoral artery. Perfusion is instituted by drawing venous blood from the patient and supplying an equal amount of arterial blood to him. After adequate perfusion is established, cooling begins. The temperature gradient (12° C) is maintained between the heat exchanger and the patient to prevent bubble formation and gas emboli or induction of premature ventricular fibrillation by the cold blood.

The maximal perfusing rate of blood flow has been 2.5 to 3.0 litres per minute or about 1.3 to 1.8 litres per minute per square metre of body surface for the average adult patient. This flow is inadequate for whole body perfusion, but the patient is protected by prior reduction of body temperature to 30° C by his own cardiac output. Thus, initially, tissue is perfused by the patient's cardiac output and by the machine. During this period, cooling is slow because perfusion from the pump-oxygenator works against the cardiac output. When ventricular fibrillation of an ineffective heartbeat occurs, perfusion is entirely from the

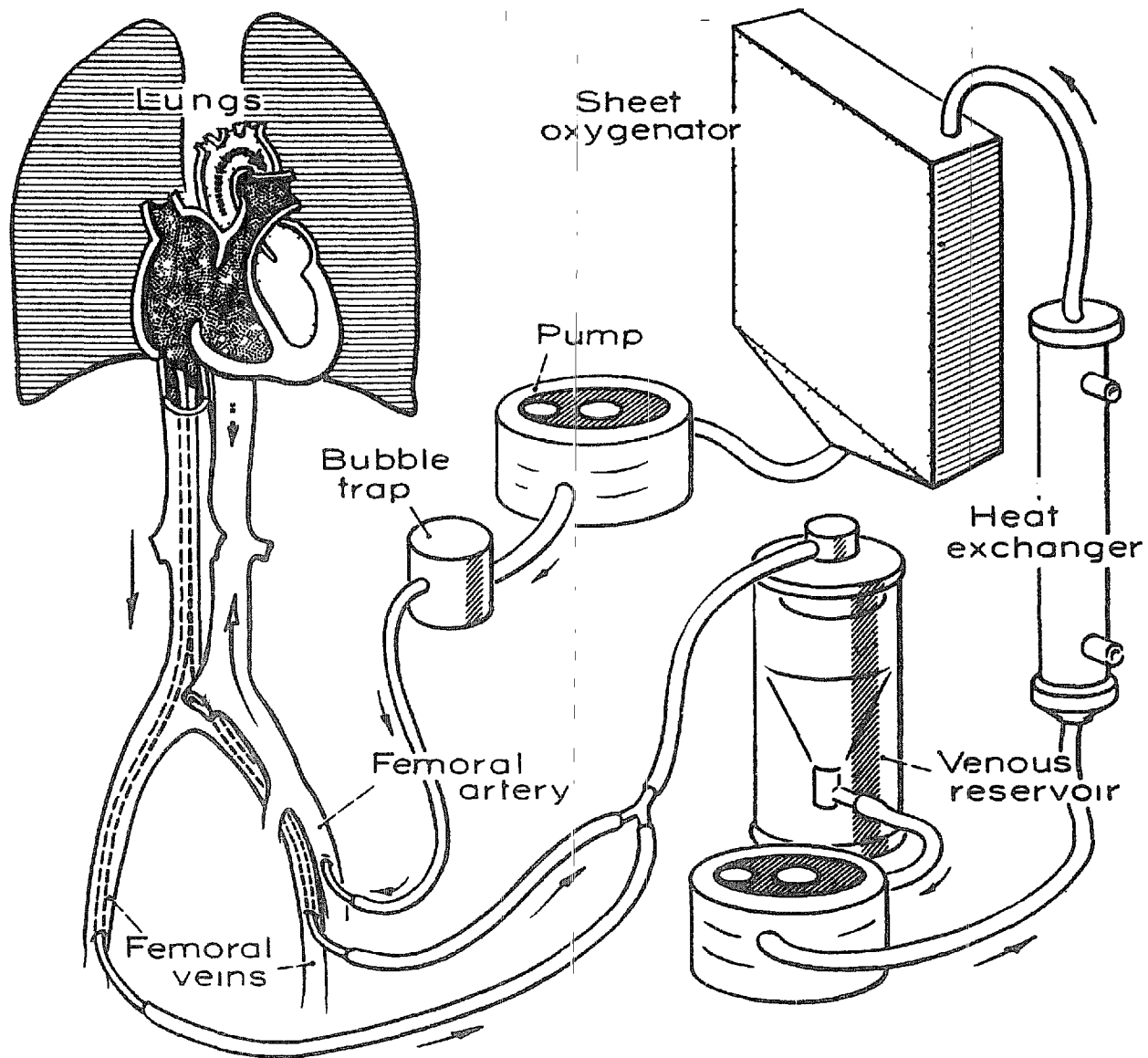


FIGURE 2 Circulation in closed-chest technique with Mayo-Gibbon vertical-sheet pump-oxygenator and peripheral cannulation (Reproduced with permission from Michenfelder, Terry, Daw, MacCarty, and Uihlein⁴)

machine, and the patient's temperature is such that the oxygen demand is satisfied by perfusion at 1.3 to 1.8 litres per minute per square metre. After ventricular fibrillation, pulmonary ventilation is terminated and cooling progresses rapidly to the desired oesophageal temperature of 13° to 15° C. Circulatory arrest or low flows are then produced to allow repair of the aneurysm.

During the period of perfusion, a mixture containing 7 per cent CO₂ and 93 per cent O₂ is metered into the oxygenator. While on bypass, but before ventricular fibrillation occurs, the same gas mixture is administered to the patient from the gas machine. The purpose of the carbon dioxide is to protect the brain from massive vasoconstriction due to the cold perfusate, at the 7 per cent concentration it has not caused visible increase in brain size.

During periods of circulatory arrest, the venous lines are left open to drain blood from the patient to the machine. This reduces venous pressure and thus greatly reduces bleeding at the operative site. The volume of this blood is usually 1000 to 1500 ml and frequently is in excess of 1500 ml. This volume of blood is rapidly retransfused to the patient at the onset of initiating bypass.

After the aneurysm is repaired and tested at various rates of flow, rewarming is begun, the 12° temperature gradient previously mentioned is maintained. When

the oesophageal temperature is 28° to 30° C, the heart is electrically defibrillated through the chest wall with the Morris external defibrillator. Rewarming is continued to 32° to 34° C, at which time the perfusion is ended. As the patient's own circulation takes over, the blood volume is increased by arterial transfusion from the pump-oxygenator. Arterial transfusion from the machine is continued until arterial pressure is adequate or until central venous pressure is approximately 15 to 18 cm of water. The heparinized state is reversed as in the Drew method.

Up to the end of January, 1964, 40 surgical procedures have been performed by the closed-chest technique. The over-all mortality was 20 per cent. Of the 40 patients, 26 had no or minimal neurological deficit. Three had significant deficits in the postoperative period, two of these had aphasia but were improving in the late postoperative period.

The mean arrest time was 13.7 minutes with a range of 0 to 50 minutes, periods of low-flow perfusion (0.6 to 1.0 L per min per M²) ranged from 8 to 55 minutes with a mean of 29 minutes. The mean cooling time for the closed-chest technique was 32.8 minutes, and the mean rewarming time was 38.8 minutes.

Cerebral Perfusion

A third method for the production of profound hypothermia in the brain has been used in our laboratory and on one patient in the operating room. This is a method of isolated cerebral perfusion after the technique of Kristiansen and associates,⁶ and consists of taking blood from an artery, usually the common carotid, pumping it through a heat exchanger, and returning it into another artery, usually the internal carotid. By proper placement of catheters and by cross-clamping the isolated internal carotid, cooling of one hemisphere can be achieved. The rate of cerebral perfusion (150 to 250 ml/min) can be regulated by controlling the speed of the pump. Circulatory arrest to the perfused hemisphere can be accomplished by stopping the pump, during which time bleeding from collateral vessels is minimal. Cerebral temperatures of 15° C have been achieved. The venous drainage of the cooled hemisphere is through the usual channels into the superior vena cava and right atrium. Inadvertent whole body hypothermia to temperatures of 30° to 32° has been common, but this has not caused any difficulties.

During the period of isolated cerebral perfusion in the operating room, the patient was placed on a Therm-O-Rite blanket warmed to 40° C to minimize the inadvertent whole body hypothermia.

The problems of total body heparinization in operating under these conditions are similar to those in the Drew and the closed-chest techniques. Meticulous care in haemostasis must be exerted. It remains to be seen whether this more simplified method of achieving profound hypothermia of the brain for repair of aneurysms will achieve results similar to those of the two previously described methods.

COMPLICATIONS AND CONTRAINDICATIONS

Cooling takes a greater length of time with the closed-chest technique, in our experience, the shortest interval was 18 minutes and the longest was 72 minutes. Rewarming time has been comparable to the cooling time.

There have been no significant disturbances in acid-base balance in these patients, and no drugs have been given for adjustment of acid-base balance. It is thought that the development of metabolic acidosis in hypothermic perfusion is not due to the hypothermia per se but to inadequate perfusing flow rates. Acidosis is correlated with the duration of circulatory arrest and not with the duration of perfusion when perfusion is two or more litres per minute per square metre.⁷

Mild metabolic acidosis has occurred in the evening of the day of operation and was characterized by low or low normal pH values associated with low or low normal buffer base concentration. The degree of acidosis was related to duration of perfusion and periods of low flows. This would indicate that total body perfusion of 1.3 to 1.8 L per min per M² was not totally adequate. Usually, by the second day the pH and buffer base concentration have returned to normal without treatment.⁸

Uihlem and associates⁹ performed preoperative and postoperative psychometric studies on patients before and after profound hypothermia. These studies showed that, if no psychometric abnormality existed in the preoperative period, none appeared in the postoperative period. If the patients exhibited a preoperative psychometric abnormality, this defect persisted in the immediate postoperative period and, in some cases, was more severe. All of these abnormal psychometric findings tended to revert to the preoperative status in the late postoperative period.

Some have suggested that an intravascular agglutination due to profound hypothermia may be responsible for neurological deficits. There is evidence that this intravascular agglutination or sludge formation may in fact be due to low flow rates of perfusion and not due to the low temperature.¹⁰

One absolute contraindication to the closed chest technique is aortic insufficiency. After fibrillation occurs, blood can flow into the left ventricle in a retrograde fashion and cause acute ventricular dilation. Diseases which would restrict retrograde aortic flow also are contraindications. A patent ductus would allow aortic flow and pressure to be transmitted to the pulmonary bed. All of our patients are examined preoperatively by a cardiologist to rule out contraindicating cardiac disease.

Relative contraindications are severe pulmonary, renal, or hepatic disease which may be affected adversely by perfusion and levels of hypothermia and circulatory arrest.

In general, cardiac defibrillation has not been difficult. Defibrillation usually occurs after one or two impulses of 450 volts for 0.25 second. On several occasions we have had to use eight to ten shocks of 450 volts and several shocks at 750 volts. With proper placement of the paddles, use of moderate pressure in applying the paddles, and application of electrode jelly between the paddles and the chest wall, burns have not occurred. Of the 40 patients undergoing closed-chest perfusion, 36 required defibrillation, three defibrillated spontaneously on reaching temperatures of 26° to 28° C, and one required open-chest defibrillation.

During closed-chest perfusion, there is a potential hazard of pulmonary vascular damage. If the flow in the bronchial arteries is great, some blood returns to

the left atrium and could result in high pressures in the pulmonary vascular bed with membrane damage and pulmonary oedema. No such problem occurred in the 40 patients. We believe that the period of perfusion, from fibrillation to defibrillation, should be as short as possible.

Control of bleeding in these patients in the immediate postperfusion and postoperative period is very difficult. Meticulous haemostasis is needed to avoid the development of intracerebral haemorrhages, subdural haematomas, and epidural clots. Profound hypothermia, extracorporeal circulation, and prolonged heparinization do alter the coagulation mechanism in a manner which is not entirely understood.

There is evidence that excessive formation of fibrinolysin can occur as a consequence of perfusion.¹¹ The reasoning is that some of the original heparin is destroyed and microclots of fibrin form in the body and in the perfusion apparatus. This presence of fibrin stimulates production of fibrinolysin and elevated levels of fibrinolysin persist into the postperfusion period, causing excessive bleeding. We attempt to retard this by the administration of a second dose of heparin (one half of the original dose) 1 hour after perfusion is instituted.

In addition to meticulous attention to haemostasis during operation, our management of postoperative bleeding has included the administration of (1) epsilon-aminocaproic acid, (2) antihæmophilic plasma which contains the antihæmophilic factor (Factor VIII), and, occasionally, (3) fibrinogen.

OTHER APPLICATIONS OF PROFOUND HYPOTHERMIA

The technique of profound hypothermia deserves consideration for procedures other than the repair of intracranial aneurysm.

Profound hypothermia has been a valuable adjunct to whole body perfusion for certain types of cardiac defects which cannot be repaired even with whole body perfusion. The patient's temperature is reduced to 15° to 20° C and the repair is effected during circulatory arrest. Examples are (1) complete transposition of the great vessels, (2) aortopulmonary fistula, and (3) closure of a Potts anastomosis as part of the complete correction of tetralogy of Fallot. Other types of cardiac surgery are possible, but difficult, with high-flow whole body perfusion. In severe tetralogy of Fallot, the greatly increased bronchial flow complicates visualization and repair of the defects. Profound hypothermia is induced to allow safe reduction of perfusing flow, or periods of circulatory arrest, during which the intracardiac procedure is done.

Carotid insufficiency, although a neurological diagnosis, is sometimes amenable to surgical treatment. In many cases, a localized plaque or stenosis may produce sufficient neurological disturbances to be a severe handicap. Carotid endarterectomy or bypass graft may permit these patients to return to an active life. Often it is impossible to insert a graft or bypass at the distal end of the carotid because of its proximity to the foramen lacerum. A short period of circulatory arrest would allow ample time for anastomosis of the graft.

The massive hæmorrhage usually encountered in hepatectomy or in creation of splenorenal shunts suggests the possible application of profound hypothermia to

these surgical procedures. Another possible application is in the problem of transplantation of organs. Profound hypothermia and circulatory arrest may simplify the manipulations of transplantation and anastomosis.

Total body perfusion with profound hypothermia and periods of circulatory arrest combined with administration of cytotoxic agents may be useful in the treatment of malignant diseases. Dr Temple Fay showed, years ago, that cold had an adverse effect on malignant tissues, possibly a technique utilizing profound hypothermia to protect normal tissue and potentiate the effect of a toxic substance on abnormal tissue might be developed.

RÉSUMÉ

Parmi les nombreuses techniques utilisées dans le traitement des anévrismes des vaisseaux cérébraux, aucune n'est plus discutée que l'hypothermie. Le but principal de l'hypothermie est de protéger de l'hypoxie les tissus cérébraux durant la réparation de l'anévrisme. Le choix de la technique de l'hypothermie doit s'appuyer sur l'habileté du chirurgien. On a associé l'usage de l'hypothermie (par refroidissement, par hypotension, ou par les deux à la fois) aux troubles neurologiques qu'un certain nombre de malades ont présentés à la suite de l'opération. La durée de la réparation de l'anévrisme a souvent dépassé la période de sécurité sans circulation produite par le refroidissement à 29-30 degrés C.

L'objectif principal de l'hypothermie profonde est de réduire à un tel point la consommation d'oxygène que le sujet peut subir, sans inconvénient, des périodes assez prolongées d'arrêt circulatoire complet. Ces périodes peuvent durer trente minutes et même davantage. En général, les indications de l'hypothermie profonde sont les suivantes:

- 1) Les anévrismes multiples intéressant une artère carotide ou les deux
- 2) Les gros anévrismes, à large base, qui semblent n'être réparables facilement qu'en vidant le sac
- 3) La difficulté probable de visualiser l'anévrisme, et le risque de le rupturer
- 4) Les anomalies vasculaires du système nerveux central

Depuis 1963, à la clinique Mayo, on a utilisé diverses techniques pour produire l'hypothermie profonde. Ce sont la technique de Drew, la technique à thorax fermé, et la perfusion cérébrale isolée.

La méthode de Drew est une technique à thorax ouvert et elle consiste à substituer au cœur deux pompes, alors que les poumons du malade servent d'oxygénateur. En utilisant cette technique, on a pratiqué l'induction de l'anesthésie à l'aide d'agents à action rapide par voie respiratoire, tels que l'halothane ou le cyclopropane, on a évité la voie veineuse pour la prémédication et pour l'anesthésie, parce qu'on ignorait le sort et la durée d'action des agents intraveineux chez les sujets soumis à l'hypothermie profonde. On a maintenu l'anesthésie à l'aide de l'halothane à 0.5 à 1 pour cent dans un mélange de protoxyde d'azote et d'oxygène, on a fait la respiration contrôlée à cause de la profondeur de l'anesthésie, et un ventilateur mécanique a servi à l'hyperventilation. Lors de cette double opération, l'équipe neurochirurgicale pratiquait la craniotomie, pendant que l'équipe cardio-vasculaire pratiquait une sternotomie.

médiane et préparait la région inguinale pour la canulation artérielle. Alors, on administrait de l'héparine au malade. Pour commencer la perfusion, on pompait le sang de oreillette gauche vers l'artère iliaque externe, en le faisant passer par l'appareil qui en variait la température. Lorsque la pression de l'oreillette droite augmentait, ce qui signifiait une défaillance du ventricule droit à cause du refroidissement, on pompait le sang de l'oreillette droite vers l'artère pulmonaire. Lorsqu'on atteignait une température œsophagienne de 15 degrés C, on commençait à ralentir ou à arrêter complètement la circulation pendant certaines périodes au cours desquelles se pratiquait la réparation de l'anévrisme. Durant la période de réchauffement, on gardait toujours une différence de 12 degrés C entre le sang qui sortait de l'appareil refroidisseur et celui qui entrait dans la circulation du malade, ceci avait pour objet de prévenir les embolies gazeuses. Nous avons trouvé que le grand désavantage de la technique de Drew est qu'elle nécessite une quantité considérable de sang frais pour amorcer l'appareil à transfusion et pour maintenir le volume sanguin du malade après la transfusion. La douleur et les malaises consécutifs à la sternotomie médiane, de même que les complications de la thoracotomie ont joué un rôle dans la morbidité et la mortalité moyennes.

A cause des inconvénients de la technique de Drew, on a mis au point une technique à thorax fermé, le tube qui amène le sang veineux du malade à l'appareil est inséré dans la veine fémorale et remonté dans la veine cave jusqu'au niveau du diaphragme, pendant qu'une canule artérielle est placée dans l'artère fémorale. La transfusion est mise en marche en soutirant du sang veineux du malade pour le remplacer par une quantité égale de sang artériel. On n'a pas dépassé la vitesse de 2,5 à 3 litres par minute, c'est-à-dire environ 1,3 à 1,8 litres par mètre carré de surface corporelle de l'adulte moyen.

A cause de l'insuffisance de ce courant sanguin pour une transfusion de tout le corps, on protège le malade en abaissant d'abord sa température à 30 degrés C. Par la suite, il est protégé par son propre débit cardiaque. Lorsque apparaît de la fibrillation ventriculaire ou une insuffisance des battements du cœur, la perfusion s'opère entièrement par l'appareil, et la température du malade est telle que cette perfusion suffit pour assurer l'oxygénation. Après la fibrillation ventriculaire, on cesse la ventilation pulmonaire et le refroidissement progresse rapidement pour atteindre la température œsophagienne de 13 à 15 degrés C. On arrête alors la circulation ou on la diminue considérablement pour permettre la réparation de l'anévrisme. Pendant la transfusion, on administre au malade un mélange constitué de 93 pour cent d'oxygène et de 7 pour cent de CO₂, ce mélange est mesuré à l'oxygénateur. Le rôle de l'acide carbonique est de protéger le cerveau contre une vasoconstriction massive provoquée par la perfusion froide. Durant le réchauffement, on maintient la différence de 12 degrés dont nous avons parlé antérieurement, lorsque la température œsophagienne atteint 28 à 30 degrés C, on défibrille le cœur à l'électricité à travers la paroi thoracique et on continue à réchauffer le malade jusqu'à 32 à 34 degrés C. La transfusion artérielle est continuée au moyen de l'appareil jusqu'à l'obtention d'une pression artérielle suffisante ou d'une pression veineuse centrale d'environ 15 à 18 cm d'eau.

Une troisième technique d'hypothermie profonde du cerveau est la perfusion

cérébrale isolée, elle consiste à prélever le sang d'une artère, généralement la carotide primitive, à le pomper vers l'appareil qui en change la température, et de là à une autre artère, généralement la carotide interne. En plaçant convenablement les conduits et en clampant la carotide interne isolée, on peut refroidir un hémisphère cérébral. On peut régler la vitesse de la perfusion cérébrale par la vitesse de la pompe. On est arrivé à des températures cérébrales de 15 degrés C. L'arrêt circulatoire de l'hémisphère perfusé se produit en arrêtant la pompe. Le drainage veineux des hémisphères refroidis se fait vers la veine cave supérieure par les voies usuelles. Il s'est produite souvent une hypothermie involontaire de tout le corps, mais cette éventualité a été sans conséquence.

L'insuffisance aortique est une contre-indication absolue à la technique à thorax fermé. Dans ce cas, lorsque la fibrillation survient, le sang peut refluer, dans un mouvement rétrograde, vers le ventricule gauche et causer une dilatation ventriculaire aiguë. Les maladies qui peuvent restreindre le courant aortique rétrograde sont aussi des contre-indications. Un canal artériel permettrait au courant et à la pression aortiques de se transmettre au lit pulmonaire.

Les troubles sérieux des poumons, des reins ou du foie sont des contre-indications relatives.

Durant la période immédiate posttransfusionnelle et postopératoire, le contrôle de l'hémorragie est très difficile chez ces malades. Il faut pratiquer une hémostase méticuleuse. Il est évident qu'il peut se former un excès de fibrinolyse à la suite de la perfusion.

On doit penser à la technique d'hypothermie profonde pour la réparation de certaines anomalies cardiaques, pour les endartériectomies de la carotide ou pour les greffes de dérivation, et pour les cas où une hémorragie massive est à craindre, comme dans les hépatectomies ou les anastomoses spléno-rénales. De plus, on pourrait utiliser cette technique associée à l'administration d'agents cytotoxiques dans le traitement du cancer.

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