MANAGEMENT OF ANAESTHESIA, PERFUSION AND SUPPORTIVE CARE DURING OPEN INTRACARDIAC OPERATIONS AND EXTRACORPOREAL CIRCULATION

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The well-being of the patient undergoing extracorporeal circulation can best be guarded by an anaesthetist who has thorough knowledge of all facets of the problem. Intimate understanding of the pathophysiology present in the patient, and also that produced by whole-body perfusion, is essential.

ANAESTHESIA

In selecting an anaesthetic agent the goal must be to provide anaesthesia with the least derangement of the normal physiological processes. This is attained by utilizing the lightest levels of anaesthesia consonant with the surgical procedure. Since intracardiac surgical procedures do not require deep anaesthesia, amnesic and analgesic levels of anaesthesia that interfere least with the normal compensatory mechanisms of the patient may be employed. Strict observance of the principles of good anaesthesia is much more important than the selection of agent or technique.

It is advantageous to eliminate spontaneous respiration by manual hyperventilation, by intravenous use of relaxants, or by both means, rather than by deep anaesthesia, since the latter would result in a greater depression of the myocardium and medullary vasomotor centre.

Previous experience should dictate the choice and dosage of premedicant drugs. The advantage of heavy premedication is reduction of the metabolic rate, whereas light premedication preserves homeostatic mechanisms to a greater extent. We take the intermediate position, that of moderate premedication. Pentobarbitone sodium, 1 or 2 mg per pound of body weight, is given orally or rectally respectively 2 hours pre-operatively. Morphine sulphate, 1 mg per 10 pounds, is given intramuscularly 1 hour before operation. Belladonna drugs are not given. This omission has not resulted in an increase of endotracheal secretion or vagal reflexes.

There is ample evidence that a variety of anaesthetic agents and techniques have proved to be satisfactory (Gain, 1957; Matthews et al., 1957; Mendelsohn et al., 1957; Patrick et al., 1957; Bourgeois-Gavardin et al., 1958; Keats et al., 1958; Norlander et al., 1958; Musicant et al., 1959). As a general principle, first choice must be the agent and technique with which the individual anaesthetist is most familiar. Myocardial depression and dilatation of the peripheral vascular bed are pharmacological properties common to all anaesthetic agents. The degree of these effects is proportional to the depth of anaesthesia. While it is of considerable importance that the level of anaesthesia be kept as light as possible, it is probably of no importance which agent is used.

In all our patients anaesthesia is maintained with nitrous oxide and oxygen (in the proportion of 3:2 litres per minute) in a circle, carbon-dioxide-absorption system. Previously, ethyl ether and oxygen were used in a closed-circle system. The change to nitrous oxide and oxygen in a semiclosed system was necessitated by the surgeons’ desire to use diathermy. Seriously ill infants are intubated awake after breathing oxygen. Anaesthesia in children is induced with cyclopropane, and endotracheal intubation is performed after intramuscular injection of suxamethonium chloride, 2 mg per pound. In adults anaesthesia is induced with intravenously administered thiopentone sodium (200 to 300 mg) while oxygen is being breathed, and endotracheal intubation is performed after intravenous injection of suxamethon-
thonium chloride. Two intravenous routes for the administration of blood or drugs are established percutaneously (or by cutting down on veins) in all patients. Intermittent intravenous injections of suxamethonium chloride (2 per cent) are given to those patients in whom control of respiration is difficult. Tubocurarine chloride has been used satisfactorily as a substitute for suxamethonium chloride. Small amounts of pethidine are given intravenously when it appears that the state of analgesia is insufficient.

During total cardiopulmonary bypass, volatile anaesthetic agents can be added to the oxygenator (Mendelsohn et al., 1957; Patrick et al., 1957) or intravenous agents can be given to the patient, or both procedures can be used. Presently, small amounts of thiopentone sodium and suxamethonium chloride are given to the patient as needed. Spontaneous respiration is also influenced by the arterial partial pressure of carbon dioxide, which largely depends on the total flow of gases, and the fractional concentration of carbon dioxide in the gases, which pass through the oxygenator. In our system, ventilation of the oxygenator is arranged to result in an arterial partial pressure of carbon dioxide of approximately 30 mm of mercury.

Management of the lungs during perfusion is a controversial question. In our practice, during this period the lungs are not ventilated but are left slightly inflated with equal parts of helium and oxygen. Some prefer to ventilate the lungs (Bourgeois-Gavardin et al., 1958) while others leave them collapsed and unventilated (Keats et al., 1958).

SUPPORTIVE CARE PRIOR TO AND FOLLOWING PERFUSION

Supportive care is based on maintenance of adequate systemic blood flow. Prior to and following perfusion the effective cardiac output determines systemic flow. The diminished cardiac reserve of these patients results in less tolerance than usual of physiologic trespass. Consequently, the margin for error is narrowed. Possible causes of reduced effective cardiac output in these patients may be hypovolaemia of unreplaceable blood loss, myocardial and medullary vasomotor depression from deep anaesthesia, mechanical interference with filling or ejection caused by inflow or outflow obstruction of the heart, and inability of the heart to compensate for shunts or valvular stenosis or incompetence.

Diagnosis of reduced effective cardiac output is indirect and is based upon the changes in haemodynamics that result from a reduction in systemic flow. When a group begins work in extracorporeal circulation, a large number of physiological variables should probably be monitored, in order to gain adequate information as to what is happening in the patient. As experience increases, monitoring devices can be gradually eliminated. At the Mayo Clinic, arterial pressure, peripheral pulse and venous pressure are at present monitored and blood volume is estimated.

Arterial blood pressure can be determined directly via an indwelling catheter or indirectly by cuff. In all but very small infants a snugly fitting cuff provides a satisfactory means of detecting blood pressure. Frequent determinations of the blood pressure by this indirect method and continuous palpation of the peripheral pulse (superficial temporal) have proved to represent a more satisfactory approach to assessment of the effective cardiac output than continuous observation of the blood pressure by a direct method. Venous pressure is determined directly by means of a plastic cannula in a peripheral vein of the superior vena caval drainage system. Changes in venous pressure are informative, while the absolute value may be misleading because of deficiencies in recording technique. Relative blood volume is followed by tallying total blood loss and total blood replacement on a balance sheet (Patrick et al., 1957). Direct observation of the heart has superseded monitoring of an electrocardiogram as a means of detecting the presence of cardiac arrhythmias.

A systolic blood pressure of less than 70 to 80 mm of mercury or an undetectable peripheral pulse, or both, indicate that the effective cardiac output is inadequate. If hypovolaemia is the cause, arterial and venous pressures are decreased, pulse rate is increased and the blood volume balance shows unreplaced loss. The treatment is transfusion of whole blood. Heparinized blood is given from the machine while the arterial cannula is in place, and citrated blood is given at all other times. When deep anaesthesia has reduced the effective cardiac output, arterial pressure is decreased, venous pressure and heart rate are increased and the blood volume balance is even.
The treatment is ventilation with oxygen. Mechanical obstruction of blood flow into and out of the heart, based upon heart displacement; a cannula in a vena cava or a finger in a valvular orifice, may result in arterial hypotension. The treatment is removal of the obstruction, thereby allowing the heart to increase its effective output. If these diagnoses and therapeutic measures prove to be inadequate, heart failure must be suspected. This diagnosis is confirmed by the presence of decreased arterial pressure, increased venous pressure and a dilated ineffectual heart. Those patients not previously digitalized are rapidly digitalized. Digoxin, or lanatoside C, 1 mg per square meter of surface area, is given intravenously. Two thirds of the dose is given initially, and the remainder 20 minutes later.

Cardiac arrhythmias are diagnosed by direct observation of the heart and by palpation of the peripheral pulse. They are usually innocuous and disappear spontaneously. Occasionally a persistent arrhythmia that results in a reduction in effective cardiac output requires treatment. Sinus tachycardia, auricular fibrillation and auricular flutter are treated by rapid digitalization. Sinus bradycardia is reversed by small doses of atropine sulfate given intravenously. Ventricular tachycardia and ventricular fibrillation are treated by cardiac massage and electric shock. In atrioventricular dissociation a lead is always sewed to the right ventricular wall. Slow ventricular rates are increased by means of an external, portable pacemaker connected to this lead. This arrhythmia can be managed, although less conveniently, with drugs such as isopropyl noradrenaline or adrenaline.

When effective cardiac output is not promptly increased to satisfactory levels by these therapeutic measures, adrenaline is given intravenously. Cardiac output and arterial pressure are increased by this drug, despite a fall in systemic vascular resistance which is chiefly a result of vasodilatation (Aviado, 1959). Ventricular arrhythmias have not been a problem. A single dose (1 to 10 ml of a 1:10,000 solution) is given initially. If necessary a continuous drip (1:100,000) is instituted.

PERFUSION

Survival of the patient following perfusion depends ultimately upon his having been supplied with properly prepared blood at suitable pressures, flow rates and temperatures. The search for a single, reliable means of estimating the adequacy of a perfusion started with the introduction of the technique and has not ended. "Bypassing is a complex clinical art and the manner in which the conduct of a perfusion affects survival and health is probably statistical rather than absolute.

Historically, both the magnitude and the manner of setting of the rate of perfusion have been subjects of controversy. It is now generally agreed that a flow rate of 2.3 l./sq.m/minute or more, or its equivalent in millilitres per kilogram, is adequate (Gibbon, 1959). It is of academic interest to decide whether this flow is fixed and preset or variable and governed by venous return. Suitable equipment and experience allow adequate perfusion in either case. The system for collection of venous blood determines if venous return at the rate of 2.3 l./sq.m/minute can be obtained in the presence of normal venous pressure and normal blood volume. Venous return to the extracorporeal apparatus is determined by the pressure gradient across, and the resistance to flow within, the collection system between the venae cavae and the venous reservoir. Vena caval pressure varies with caval blood volume, but the pressure gradient can be widened by vacuum or siphonage. Resistance to flow is fixed by the inner cross-sectional area and length of caval cannulas, connecting tubing and adapting orifices. At a flow rate of 2.3 l./sq.m/minute increased venous pressure, increased patient blood volume, and venous pooling are observed in man only in the presence of inadequate venous cannulation, or a defective system for collection of venous blood, or both.

Mortality attributable to the perfusion is not apparent in current experience at the Mayo Clinic. A few measurements now make possible the accurate estimate of many of the physiological variables believed basic to an adequate perfusion. Knowledge of oxygenator performance (Levin et al., 1959) and the measurement of total flow rate allow prediction of the rate of oxygen uptake, the partial pressure of oxygen and the percentage saturation of haemoglobin with oxygen, in arterial and mixed venous blood. The measurement of flow rate and venous pressure permit prediction
of arterial pressure since total peripheral systemic resistance is known (Moffitt et al., 1959; McGoon et al., 1959). In this connection, aortic clamping and extracardiac shunts, that is, patent ductus or bronchial flow in tetralogy of Fallot, must be noted and taken into consideration. Previous studies during perfusion (McGoon et al., 1959) permit prediction of pH, carbon dioxide tension, and buffer base. While little information on regional blood flow has been acquired, qualitative changes in the cerebral blood flow are predictable (Thye et al., 1957) from measurement of total flow and venous pressure, and from surgical exclusion of the presence of large extracardiac shunts. The guarantee of normothermia provided by the Brown-Emmons heat exchanger has enhanced considerably the predictability of these physiological events.

Total flow and venous pressure are at present measured by us during clinical perfusions. Measurement of arterial flow rate is indirect and is based upon a calibration curve relating pump revolutions per minute and output. The output of the arterial pump of the Mayo-Gibbon pump-oxygenator is regulated by the rate of venous return but cannot exceed the output of the pump delivering blood to the oxygenator (oxygenator pump).

In practice the oxygenator pump is preset at the flow predicted to be adequate for the perfusion (Kirklin et al., 1957). Partial by-pass is initiated by establishment of continuity between the collecting system and the venae cavae, and application of negative pressure. This results in delivery of blood from the patient to the machine. The arterial pump meanwhile has delivered blood at an equal rate to the patient. Any large extracardiac shunt (for example, patent ductus arteriosus) is ligated at this time. Total by-pass of the heart and lungs is accomplished by occluding the venae cavae around the cannulas and by delivering the intracardiac return to the venous reservoir. At this time systemic flow is equal to the output of the arterial pump which is, in turn, equal to the venous return. Ordinarily the flow obtained is steady and equal to that predicted to be adequate. Flows less than predicted are based upon hypovolaemia or a defective venous collecting system.

Hypovolaemia is due to unreplaced blood loss which is confirmed by a low venous pressure and a negative value on the blood volume balance sheet. Treatment consists of transfusion of heparinized blood in adequate amounts. A defective venous collecting system results in an increased gradient between the venous system of the patient and the venous reservoir. The measured venous pressure will increase if this defect is proximal to the vein in which the pressure is being measured. Common causes include small caval cannulas, placement of cannulas in caval tributaries (for example, innominate and hepatic veins), slippage of occluding tapes over cannular tips, and kinking of connecting lines. Treatment consists in removal of the cause of the impeded venous return.

With completion of the surgical repair and closure of the ventricular or atrial incision, intracardiac return fills the heart and is no longer diverted to the extracorporeal apparatus. Right ventricular ejection of this blood results in pulmonary flow and thereby concludes total by-pass. Left ventricular ejection may result in a carotid pulse. The presence of a carotid pulse at this time is considered to be a favourable prognostic sign. Release of the caval tapes, reduction of vacuum, removal of the superior vena caval cannula, and withdrawal of the inferior vena caval cannula to the right atrium are carried out stepwise. Each step results in an increased opportunity for venous return to enter the heart and thereby augment cardiac output. The degree to which this occurs is carefully assessed at each successive stage. The central circulating blood volume is being restored at this point and care must be taken to preserve normovolaemia. An active, contracting heart, a palpable peripheral pulse, an arterial blood pressure of 80 mm of mercury or more, and a great reduction in the rate of venous return to the machine indicate that venous return is entering the heart preferentially and increasing cardiac output. In this circumstance the perfusion is discontinued. A dilated, flabby heart, absent peripheral pulse, and maintenance of venous return to the extracorporeal circuit indicate that the heart has not been able to respond to the work of providing for adequate systemic flow. In this case the perfusion is continued until the basis for the heart failure has been ascertained and, if possible,
eliminated. This is ordinarily a surgical consideration.

**BODY TEMPERATURE DURING PERFUSION**

The goal of normothermia was approached from many angles but never realized during the first 3 years of open heart surgery at the Mayo Clinic. Initially the patient was warmed with a heating blanket. This proved to be inadequate. Next, the saline solution used for filming the oxygenator and the gases entering the oxygenator were heated to 40°C. The donor blood was kept at 38°C. Mild hypothermia continued to occur in all patients. The degree of hypothermia depended on body size, length of perfusion and rate of flow (Moffitt et al., 1959).

Incorporation of a Brown-Emmons heat exchanger (Brown et al., 1958) into the extracorporeal circuit has resulted in satisfactory control of body temperature. While this apparatus is routinely used for the maintenance of normothermia, it also provides a method for the rapid induction and reversal of hypothermia. Since the solubility of oxygen in blood is inversely proportional to the temperature of the blood (Sendroy et al., 1934), changing the temperature of the perfusing blood from that of the patient introduces the possible danger of gas emboli. The critical factor is the degree and direction of difference between the temperature at which blood-gas equilibrium takes place in the oxygenator and the temperature of the patient.

The combination of mild hypothermia (30°C) and perfusion (Sealey et al., 1958), or profound hypothermia (15°C) without perfusion (Gollan, 1954; Drew and Anderson, 1959), has been suggested. These approaches warrant interest. A limited clinical experience with both approaches over the past year suggests several applications. Mild hypothermia and reduction of arterial flow are advantageous when increased intracardiac return complicates the surgical repair and when normal flows cannot be returned adequately through the caval cannulas in small infants. Profound hypothermia has been useful when a period of circulatory arrest facilitates or makes possible a difficult surgical procedure.

**SUMMARY**

Light anaesthesia with respiration controlled by hyperventilation is ideal for patients undergoing open intracardiac operations and extracorporeal circulation. Careful, intelligent management of the anaesthetic procedure is more important than the specific premedication, agent or technique. In essence, supportive care includes all efforts required to maintain satisfactory systemic blood flow. Systemic blood flow before and after perfusion depends upon the effective cardiac output. The factors that decrease the effective output of the heart are haemorrhage, deep anesthesia, mechanical obstruction of blood flow through the heart and the pathophysiology of the cardiac defect. Before and after perfusion, information on the effective cardiac output is obtained indirectly by checking the arterial blood pressure, peripheral pulse, venous pressure and relative blood volume. When the arterial pressure falls to less than 70 to 80 mm of mercury and the peripheral pulse cannot be palpated, or with either event, the effective cardiac output is inadequate and must be actively treated. Treatment is directed at removal of the cause. During perfusion, systemic blood flow is determined by the output of the arterial pump. The rate of flow, the venous pressure and the relative blood volume are monitored. A steady arterial flow of 2.3 l./sq.m/min or more is adequate for perfusion of the whole body. Venous return at this same rate of flow, with normal venous pressure and blood volume, can be obtained by a satisfactory collecting system for venous blood. The factors that decrease venous return and arterial flow during perfusion are hypovolaemia and a defective collecting system for venous blood. The Brown-Emmons heat exchanger provides a satisfactory means for control of the temperature of the patient during perfusion.

**REFERENCES**


