ANAESTHETIC MANAGEMENT FOR CARDIOPULMONARY BYPASS A REVIEW OF 200 CASES*

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AS EARLY AS 1957, a group of cardiac surgeons and anaesthesiologists at our hospital became interested in undertaking open cardiac surgery using cardiopulmonary bypass. At that time, such procedures were considered possible only in large medical centres. However, we felt that our community hospital and medical staff had the prerequisites necessary for such an undertaking as subsequently listed by Kirklin¹. Thus, a laboratory study was undertaken to establish the proficiency in the surgical and anaesthetic techniques from whence we progressed to clinical management of patients.

The surgical management of congenital and acquired heart lesions is subject to rapid modification. Each new publication and each conference brings forth new surgical techniques and prostheses for the correction of valvular lesions. Concomitantly, our anaesthetic management has changed, based not only upon our own experiences but also upon those of others. We propose here to present the methods, results, and comments on the first 200 consecutive cases. The first six cases² and the first 50³ consecutive cases will be incorporated in the present discussion for completeness even though they have been reported previously.

METHODS

A steady change best describes our anaesthetic management of these patients. This change does not indicate a lack of confidence in the management nor does it reflect unsatisfactory results. On the contrary, we have benefited from our experiences and from the reports of research centres. In addition, trips were made to these medical centres to observe their methods and techniques. However, the methods used were essentially those previously reported³ and are reviewed briefly, mentioning any pertinent changes.

The heart-lung apparatus used in the first 89 cases was a screen oxygenator, the Mark G-K. It was similar in operation to the one previously used by Kirklin as reported by Patrick⁴. Beginning with case 90 we have used the improved Mayo-Gibbon⁵ pump oxygenator, which uses vertical sheets of Mylar-covered aluminium as the oxygenating surface and a mechanical filter. It has a significantly lower priming volume, of great importance when whole blood is used. Arterial output to the patient is maintained equal to the rate of venous return to the machine by means of a servomechanism which uses level-sensing devices in the venous and arterial reservoir and an occluder which controls recirculation of

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oxygenated blood within the machine. Controlled vacuum is used to bring venacaval or right atrial blood to the venous reservoir, to draw blood from the heart to the intracardiac sucker reservoir, and to add donor blood to the filling reservoir. In practice, arterial oxygen saturations exceeding 95 per cent are constantly produced. When long perfusions or aortic cross-clamping are indicated, coronary perfusion is performed by tapping the arterial line. The flow through this coronary system is controlled by screw clamps and measured by a ball-valve mechanism. Occlusive pumps propel the blood through the system with a single roller rotating a full circle on a silastic tube. The rate of each pump is indicated directly on the control panel.

A Brown-Harrison blood heat exchanger is incorporated on the arterial side of the pump. In early cases, it was on the input or venous side. This change was made to effect more rapid temperature changes when hypothermia was elected. Oxygen is supplied to the heart-lung machine from the piped system in the operating room, passing through a Fluotec vaporizer to the screening chamber when halothane is used. When fluroxene is used, the Fluotec vaporizer is set at the 1 per cent marking. Methoxyflurane has not been added to the machine.

The heart-lung apparatus employs the so-called high-flow principle wherein the output on the arterial side, i.e. perfusion index, is set to simulate the calculated basal cardiac output for the particular patient and is based upon the principle that the normal cardiac output is a function of the surface area of the body.

Prior to surgery, all patients are seen in consultation, at which time ideas are exchanged and therapeutic hopes are openly discussed among anaesthesiologist, surgeon, and cardiologist. The surgeon and cardiologist also discuss in detail the patient's condition and prognosis with either the patient or the immediate family. The anaesthesiologist explains to the patient what to expect in the operating room prior to surgery. This explanation is tempered by insight, age, and mentality. Premedication consists of a barbiturate, meperidine, and promethazine, according to usual standards. Belladonna derivatives were not administered to these patients.^{2,5}

Anaesthesia in adults and older children was induced with a hypnotic dose of 2.5 per cent sodium thiopental intravenously and rarely was more than 200 mg required. Oral endotracheal intubation was accomplished by an apnoeic dose of 0.2 to 0.6 per cent succinylcholine chloride administered by continuous intravenous drip. Anaesthesia was maintained by the use of one of the following:

- (a) nitrous oxide-oxygen, muscle relaxant,
- (b) nitrous oxide-oxygen, halothane, muscle relaxant,
- (c) nitrous oxide-oxygen, fluroxene, muscle relaxant,
- (d) nitrous oxide-oxygen, methoxyflurane, muscle relaxant.

Cyclopropane-oxygen or nitrous oxide-halogenated agent-oxygen mixtures were used to induce anaesthesia in small children or adults in whom venipuncture was difficult. The maintenance period was similar to one of the previously described combinations.

Throughout anaesthesia and surgery the electroencephalogram, the electrocardiogram, and the rectal temperature were monitored. Venous pressures were

monitored constantly throughout surgery and into the postoperative period either by external jugular catheterization or by passing a catheter through the brachial vein into the superior vena cava or innominate vein. A Mayo pressure monitor system was used for these determinations together with Statham transducers. The arterial pressures were taken by the cuff method. Further assessment of the patient's general condition was made by noting colour, general appearance, fullness of pulse, and pupil size.

Before cannulation in preparation for cardiopulmonary bypass, heparinization was accomplished by administering 90 mg/m^2 body surface area of heparin. After each 90 minutes of bypass, one half the original dose of heparin was repeated. Heparin was neutralized by administering 140 mg/m^2 body surface area of protamine sulphate. Prior to removal from the market, hexadimethrine bromide (Polybrene) was equally effective. Frequently, an additional 50 to 100 mg of either drug was required to bring about adequate clotting of blood.

Once cardiopulmonary bypass was instituted, 4 litres/min of carbon dioxide was allowed to flow into the open pericardium. As the result of the work of Cagin,⁷ any blood which collected in the pericardium was taken away by the high-speed sucker so that it was not returned to the pump.

Throughout surgery, blood loss was continuously measured by weighing sponges and determining the quantity of blood which collected in the suction bottle. Blood replacement was governed by venous pressure, arterial pressure, and measured blood loss. While the chest was open, great vessel or heart tension was determined by "the educated hand" of the surgeon, and this further helped in assessing the need for blood replacement.

Once cardiopulmonary bypass was instituted, small doses of sodium thiopental were occasionally administered if the patient showed signs of awakening. As previously stated, halothane or fluroxene was added to the heart-lung machine at the discretion of the anaesthesiologist. Methoxyflurane was never added to the machine. Small doses of either *d*-tubocurarine or succinylcholine chloride were administered as needed to maintain a quiet patient.

Cardioplegia was attained by a variety of methods. Early in the series, potassium citrate was employed following the work of Kolff,⁸ but this was subsequently abandoned. This was followed by hypoxic cardiac arrest at 30°C for varying periods of time. The hypoxia was brought about by cross-clamping the aorta. Saline slush or cold saline solution at 4°C was later used followed by electrically induced ventricular fibrillation at 30°C . Currently, coronary perfusion is preferred when the aorta must be cross-clamped to obtain a relatively bloodless field. The latter may also be combined with electrically induced ventricular fibrillation to obtain cardioplegia.

Direct current has replaced alternating current as the method of choice in terminating ventricular fibrillation in accordance with the work of Lown.⁹ Direct current has not been used to terminate any adverse cardiac arrhythmias during or following cardiopulmonary bypass other than ventricular fibrillation. Small doses of succinylcholine chloride are given prior to application of the electrical shock in order to diminish the intensity of the skeletal muscle response.

RESULTS

All procedures are tabulated (Table I) according to the primary anaesthetic and the type of cardiac lesion or operative procedure. There were 46 in-hospital deaths during or following the 200 operations, a mortality rate of 23 per cent.

TABLE I
SUMMARY OF NUMBER AND TYPE OF SURGICAL CONDITIONS OR OPERATIONS AND
PRIMARY ANAESTHETIC AGENTS USED

Operation or lesion		Nitrous oxide	Halothane	Fluroxene	Methoxy- flurane	Total
Mitral Valvuloplasty or annuloplasty	Number	4	50	16	11	81
	Deaths	1	12	2	2	17
	Per cent	25	24	12.5	18.1	20.9
Mitral Starr valves	Number	0	1	8	7	16
	Deaths	0	0	2	3	5
	Per cent	0	0	25	42.8	31.2
Aortic Valvuloplasty	Number	3	24	6	4	37
	Deaths	0	6	3	2	11
	Per cent	0	25	50	50	27
Aortic Starr valves	Number	0	2	2	2	6
	Deaths	0	2	1	1	4
	Per cent	0	100	50	50	66
IASD	Number	10	9	6	1	26
	Deaths	1	1	0	0	2
	Per cent	10	11	0	0	7.6
IVSD	Number	2	5	2	0	9
	Deaths	2	1	0	0	3
	Per cent	100	20	0	0	33
PS	Number	5	5	1	1	12
	Deaths	0	0	0	0	0
	Per cent	0	0	0	0	0
Others	Number	4	7	1	1	13
	Deaths	2	2	0	0	4
	Per cent	50	28	0	0	31
Total	Number	28	103	42	27	200
	Deaths	6	24	8	8	46
	Per cent	21.4	23.3	19.0	29.6	23.0

Table II lists the primary anaesthetic agents with respective in-hospital mortalities. Twenty-eight patients received a combination of sodium thiopental-nitrous oxide-muscle relaxant with 6 deaths occurring in the hospital, a mortality rate of 21.4 per cent. In none of these cases was the anaesthetic agent even remotely implicated. One hundred and three patients received a combination of sodium thiopental-nitrous oxide-halothane-muscle relaxant with 24 in-hospital deaths, a rate of 23.3 per cent. In only 8 of these cases could the anaesthetic agent be even remotely implicated. Thus, we say that in 8 of these 103 patients, halothane

TABLE II
SUMMARY OF IN-HOSPITAL DEATHS AND PRIMARY ANAESTHETICS WHERE ANAESTHESIA
WAS POSSIBLY A CONTRIBUTORY CAUSE

	Nitrous oxide	Halothane	Fluroxene	Methoxy- flurane	Total or average
Total cases	28	103	42	27	200
Total in-hospital deaths	6	24	8	8	46
Per cent	21 4	23 3	19	29 6	23
Cases anaesthetic possibly contributory to death	0	8	1	6	15
Per cent anaesthetic possibly contributory to death	0	7 7	2	22	7 5
Bypass time of fatalities in minutes	63	110	160	114	125
Bypass time of survivors in minutes	83	72	86	88	82

anaesthesia might have played a part, with a mortality rate of 7 7 per cent for this series. Forty-two cases were anaesthetized by a sodium thiopental-nitrous oxide-fluroxene-muscle relaxant combination with 8 in hospital deaths, a mortality rate of 19 per cent. However, in only one of these deaths could the anaesthetic agent be remotely implicated, a mortality rate in the fluroxene cases of 2 per cent. Finally, 27 patients were anaesthetized with a sodium thiopental-nitrous oxide-methoxyflurane-muscle relaxant combination with 8 deaths occurring in the hospital with a mortality rate of 29 6 per cent. Six of these cases *might* (and we emphasize the word *might*) have been in part due to the anaesthetic agent, a mortality rate of 22 per cent for the methoxyflurane group.

The average time on bypass for the 154 patients with an in-hospital survival was 82 minutes, while that of the 46 patients who represented in-hospital deaths was 125 minutes. There was no correlation of deaths with the primary anaesthetic agent (nitrous oxide, halothane, etc.) and bypass time (Table II). The time for

TABLE III
CAUSES OF IN-HOSPITAL DEATHS IN 200
CONSECUTIVE OPEN-HEART OPERATIONS
USING CARDIOPULMONARY BYPASS

1	Anaesthesia possibly contributing	
	Nitrous oxide	0
	Halothane	8
	Fluroxene	1
	Methoxyflurane	6
2	Inadequate surgical procedure	11
3	Deficient cardiac output	11
4	Postoperative haemorrhage	4
5	Pulmonary complications	2
6	Coronary artery disease	2
7	Miscellaneous	1

fluroxene cases and methoxyflurane cases was essentially equal, while for nitrous oxide and halothane it was somewhat shorter

Cardioplegia was produced most frequently for surgery upon the aortic valve. Saline slush was used in 28 cases with 14 in-hospital fatalities or a 50 per cent mortality rate. However, only 8 of these deaths could possibly be related directly to the use of slush and thus a mortality rate of 33 per cent was attributed to the use of slush. Aortic valve surgery of a similar nature was carried out under coronary perfusion in 13 patients with 7 in-hospital fatalities, a mortality rate of 54 per cent. Only 3 of these latter could be related to coronary perfusion, and thus there was a mortality rate of 23 per cent for which the perfusion could be implicated.

TABLE IV
POSTOPERATIVE COMPLICATIONS

1 Alopecia	8
2 Jaundice	
Homologous jaundice	2
Clinically jaundiced-haemolytic (1 Plasma Hb to 600 mg %, no jaundice)	6
3 Infarcts of organs	
Cerebral infarct, non-fatal	4
Pulmonary infarct, non-fatal	1
Hemiparesis from CVA, hypotensive in origin, non-fatal	1
Fatal renal infarct	1
Burns of myocardium	4
4 Emboli	
Femoral in postoperative period	2
5 Heart blocks	
Complete, required pacing	2
6 Phlebitis	
Moderately severe	6
7 Anuria	
Fatal	3
Lower nephron	1
Anuria that cleared, apparently not lower nephron	3
8 Slough	
From levophed	1
9 Post-pericardiotomy syndrome	10
10 Psychiatric problem	
Temporary psychosis, aetiology not clear	3
Psychosis existed preoperatively	1
11 Atelectasis	
Problem case	9

Table III lists the causes of death and Table IV lists some of the more outstanding complications, some of which were fatal and hence are included in Table III. The cases of alopecia³ and fat emboli⁷ have been reported previously. Table V lists those cases in which we thought anaesthesia could be a contributory cause to the fatal outcome.

TABLE V
CAUSES OF DEATH WHERE ANAESTHESIA WAS POSSIBLY IMPLICATED

Agent	Lesion	Time of Death	Terminal Event	Post mortem
Nitrous oxide	None	None	None	None
Halothane	(MI)MS	On table	Irrev ventric fibrill at end of bypass	None
	AS	On table	Irrev ventric fibrill	Burn or infarct of myocardium
	IVSD	On table	Irrev ventric fibrill	Repaired IVSD, otherwise neg
	AS	On table	Saline slush used, irrev ventric fibrill	Non-contributory
	MS	On table	Irrev ventric fibrill	Old and new renal infarcts
	AS	On table	Saline slush used, irrev ventric fibrill	Burn or infarct of myocardium
	AS	On table	Saline slush used, irrev ventric fibrill	None
	MS(AS)	On table	Sudden collapse after turned from lateral to supine position	Caseous tuberculosis, undiagnosed
Fluroxene	AI	On table	Saline slush used, irrev ventric fibrill	Burn or infarct of myocardium
Methoxyflurane	AS	On table	Saline slush used, irrev ventric fibrill	None
	AI	On table	Saline slush, arrest after slush irrev	Non-contributory
	MI + AI	3 hr postop	Never regained consciousness, irrev heart failure	Non-contributory
	MS Starr valve	3½ hr postop	Never regained consciousness, irrev heart failure	Right renal infarct, recent
	AI Starr valve	3 hr postop	Never regained consciousness, irrev heart failure	None
	MS	12 hr postop	Slowly regained con- sciousness, irrev failure, profound hypotension before bypass	Non-contributory

AS = Aortic stenosis

MS = Mitral stenosis

IVSD = Interventricular septal defect

MI + AI = Mitral insufficiency and aortic insufficiency

Irrev ventric fibrill = Irreversible ventricular fibrillation

COMMENTS

The patients in this series were unselected. The decision to operate was based on anatomical and/or haemodynamic operability. Many of the patients had been refused surgery in other centres. Some of these latter patients survived while some did not. It was felt by the surgeon, cardiologist, and anaesthesiologist that something could and should be offered a patient in a positive manner rather than in a negative, hopeless attitude. This we feel in part explains the high mortality rate, particularly in the patients with acquired mitral and aortic valvular disease.

In a review of this type, hospital mortality statistics may be without meaning

unless there is some standardization of the variables. In a clinical situation, this would appear to be almost impossible to attain where several different anaesthesiologists are involved. The surgical experience was standardized to the extent that only two surgeons were involved and one of them did well over 90 per cent of the cases. However, as relates to the anaesthetic agents alone, certain trends are apparent in analysing these 200 cases.

There were no deaths in which anaesthesia could be implicated in those patients receiving nitrous oxide as the primary anaesthetic agent. In only one death in 42 anaesthetics could fluroxene be implicated (Table V), and here we are perhaps being overcritical of this agent because saline slush and long perfusions are capable of contributing significantly to an increased mortality and morbidity rate. Possibly, the combination of halogenated agent (fluroxene), saline slush, and prolonged perfusion so altered or disturbed the homoeostatic mechanisms within the myocardium that nothing but a fatal outcome was possible. The 8 fatalities, in 103 anaesthetics, possibly attributed to the presence of halothane might likewise be due to the use of this agent in the presence of disturbed myocardial homeostatic mechanisms. The pattern in each one of these fatalities was very similar to that with fluroxene. The much higher mortality for patients receiving methoxyflurane (Table II) is disturbing. All patients were carried in plane one of surgical anaesthesia or lighter as indicated by the anaesthetic records. The type of surgery for each of the four primary anaesthetic agents, i.e. nitrous oxide, halothane, etc., carried the same risk. The age and physical status of the patients were equally distributed for all the primary anaesthetic agents. Perhaps the higher incidence of fatal outcome when methoxyflurane was used indicates a greater toxicity upon the open heart using cardiopulmonary bypass techniques. This series would justify such a conclusion, however, a hasty conclusion may not be warranted in retrospect after a larger number of cases are done with as much standardization as the clinical situation permits. "Casual coincidences are not uncommon in medicine and it is a useful axiom that etiological factors and pathological happenings should not be regarded as cause and effect unless their relationship can be established beyond a reasonable doubt"¹⁰ This higher mortality certainly has not been our experience using methoxyflurane on other surgical patients. There are many factors to be considered in evaluating fatalities in the light of anaesthesia in this or any series of cases. In reviewing these 15 cases in which we say anaesthesia could be implicated, it would have been possible to incriminate the surgical procedure or deficient cardiac output. However, as anaesthesiologists we often overlook that the potent and perhaps toxic qualities of our anaesthetic agents might be contributory to the demise of surgical patients when the proper set of circumstances exists.¹¹

Other causes of in-hospital mortality are listed in Table III, following the classification of McGoon.¹² These cases, all surgical mortalities, reflect limitations in judgment and procedure on the date of operation. Many of the surgical conditions encountered a few short years ago could be adequately corrected today with improved techniques and prostheses. Some of the patients who died in the immediate postoperative period would be salvageable today in the light of recent improvements.

The aetiology of 8 cases of alopecia remains obscure. Perhaps heparin played a part, though previously³ we ascribed this condition to a possible electrolysis resulting from faulty grounding between electrocautery and electroencephalogram leads. Pressure as an aetiological factor did not seem to apply as the alopecia did not always occur on the pressure points of the scalp.¹³

Thirteen patients experienced fat embolization⁷ manifested by lipuria and various central nervous system phenomena. This has not occurred since we abandoned the practice of returning extra-pericardial blood to the heart-lung machine by means of suction. Fat globules are often seen floating in this blood and could well be the source of emboli.

Most patients had a significant rise in serum bilirubin. However, only 8 patients became jaundiced to a degree detectable with the naked eye. This no doubt represents a haemolytic reaction from the physical trauma inflicted upon the red blood cells during the period of bypass. Many of these patients have compromised liver function due to chronic congestive failure as a result of their heart disease. The latter results in a more sluggish removal of plasma haemoglobin and further adds to the rise in serum bilirubin. The bilirubin in all cases returned to normal in a matter of days, justifying a conclusion that hepatocellular damage, if any, was minimal. Two of these patients developed homologous serum jaundice and subsequently went on to an uneventful recovery, an incidence of 0.5 per cent. This is somewhat lower than the 1.86 per cent described by Adashek¹⁴ as occurring after open-heart surgery employing cardiopulmonary bypass.

Infarcts of distant organs are a surgical complication, however, the 4 cases of myocardial burns require some explanation. All 4 patients (fatalities) were treated for ventricular fibrillation by the use of alternating 60-cycle current at 0.1 second using voltage from 110 to 115 volts. Burns of the myocardium have long been a complication of repeated alternating current shocks applied to the heart.¹⁵ We have not seen this complication since using direct current according to the methods of Lown.⁹ Some of the persisting and fatal cases of ventricular fibrillation early in our series may have been due to the faulty grounding of electrical equipment as described by Whalen.¹⁶ There has been, more recently, a definite reduction in the incidence of irreversible ventricular fibrillation as a result of the installation of a new and modern electrical system in our operating suite.

Post-bypass haemorrhage has presented a problem at times. Thrombocytopenia has not occurred, though platelet counts have been borderline. The problem most frequently seems to be related to inadequate surgical haemostasis or inadequate neutralization of heparin. Fibrinolytics to date have not been a problem. Four patients exsanguinated or expired as a result of post-bypass or postoperative haemorrhage. One patient exsanguinated from a lacerated pulmonary artery. One had cardiac tamponade from unknown bleeding points, and two patients had fatal haemorrhage from unknown bleeding points.

Seven patients experienced significant anuria in the postoperative period. Three cases were fatal. One was of the lower-nephron nephrosis type, three lasted 2 to 3 days and cleared spontaneously. These latter did not appear to be of the lower-nephron nephrosis type. The factors often leading to renal shutdown were generally found to be absent in our patients.¹⁷ The heart-lung machine in the

present series is a high-flow machine and only moderate, generalized body hypothermia (30°C) is used. Haemolysis was not excessive, and our patients generally were not acidotic at the conclusion of bypass.^{3, 17} There was no correlation between length of bypass and the appearance of renal complications. It was not felt that hexadimethrine bromide (Polybrene) played any role in the aetiology of renal shutdown as the dosage to produce renal shutdown is generally much larger than that used in this series. In each instance of renal failure, low cardiac output existed at some point before, during, or at the conclusion of bypass, and this was probably the underlying aetiological factor.

The post-pericardiotomy syndrome was diagnosed in 10 patients in this series and did not appear to contribute to in-hospital mortality.

The absence of belladonna derivatives in the premedication did not appear to influence the conduct or the outcome of the operation or anaesthesia adversely. Vagal reflexes and secretions were never a problem, and dangerous tachycardias as previously reported³ were notably absent. This again raises the possibility that all patients who are to receive general anaesthesia may not require a belladonna derivative.¹⁸

Succinylcholine chloride has been adequate for the control of any patient movement that appeared before bypass, during bypass, or at the time of ventricular defibrillation. Concentrated solutions of 0.4 per cent to 0.6 per cent succinylcholine chloride were used to reduce the total volume of fluid administered to these cardiac patients. Some patients received *d*-tubocurarine chloride. Postoperative apnoea has not been a problem.

The entire operative course was smoother when fluroxene or halothane was used in the pre-bypass, bypass, and post-bypass period. Frequently, large amounts of these two agents were vaporized from the Fluotec vaporizer, but the superior anaesthetic course seemed to justify this added expense. In view of our mortality figures (Table V), and the work of Miller,¹⁹ we would conclude that fluroxene is the anaesthetic agent of choice to fortify nitrous oxide in the management of patients undergoing cardiopulmonary bypass for the correction of acquired and congenital heart lesions.

Those patients in the present series anaesthetized with nitrous oxide and methoxyflurane appeared to have a more stormy course while on bypass. This could be due to a lack of potency in nitrous oxide. Methoxyflurane was not administered during the bypass period and possibly blood levels were not high enough to keep the patient adequately anaesthetized. However, the concentration of methoxyflurane might have been sufficient to affect the cardiovascular system adversely and might have resulted in the higher mortality than with halothane and fluroxene. This conclusion, previously stated above, might not be warranted in a subsequent and larger series.

Carbon dioxide was allowed to flow slowly into the pericardial sac at 4 litres/minute during bypass so as to displace the air and have only gaseous carbon dioxide present. If gas embolization did occur it would be a carbon dioxide embolus, and any adverse results from such an embolus would be of shorter duration because of the greater solubility of carbon dioxide than air. We have no substantiation for this except on a theoretical basis.

Finally, as Kirklin¹ has stated, undertakings of this magnitude are possible in a community hospital only when certain prerequisites have been met. It is not for physicians in these hospitals, in the vast majority of instances, to do original work in this extensive and important field, but rather it is their purpose to apply what has been well established by large research centres. Thus, such community hospitals and their medical staff fulfill their purpose to the community. The future of open-heart surgery using cardiopulmonary bypass is approached with optimism and a sense that surgery of the heart will soon have morbidity and mortality rates comparable with those for other organ systems.

SUMMARY

Two hundred, unselected, consecutive open-heart operations using cardiopulmonary bypass are reviewed with particular reference to methods and results. An attempt was made to correlate primary anaesthetic agents used and in-hospital mortality. Nitrous oxide was never implicated in any of the deaths where it was used as the primary anaesthetic agent. In those cases where halogenated agents were used, methoxyflurane had the highest in-hospital mortality rate, halothane was next, and fluroxene had the least number of deaths where anaesthesia could be implicated. On the basis of our results and interpretations, fluroxene would appear to be the anesthetic agent of choice in managing patients for open-heart surgery using cardiopulmonary bypass.

RÉSUMÉ

Depuis 1957, nous avons fait 200 cas de chirurgie à cœur ouvert. Pour les 89 premiers cas, nous avons employé un oxygénateur à écran, l'appareil Mark G-K, pour les autres cas, nous avons employé la pompe oxygénatrice améliorée Mayo-Gibbon. Du côté artériel de la pompe, nous avons intercalé un "blood heart exchanger" de Brown-Harrison. L'oxygène pour la machine cœur-poumon était amené du système de tuyaux et passait dans un fluotec. Nous avons employé de grands débits.

En prémédication, nous avons employé un barbiturique, de la mépéridine et de la prométhazine, nous n'avons pas employé de belladone. L'induction a été faite avec une dose hypnotique de thiopental à 2.5 pour cent. Nous avons pratiqué l'intubation endotrachéale par la bouche après avoir donné une dose suffisante de chlorure de succinylcholine de 0.2 à 0.6 pour cent pour produire l'apnée chez le malade. Nous avons continué l'anesthésie avec du protoxyde d'azote et des myorésolutifs seuls, ou en ajoutant de l'halothane, du fluroxène ou du méthoxyflurane.

Les tracés électrocardiographiques et encéphalographiques, la température rectale, la pression veineuse étaient continuellement indiqués par des appareils. Nous avons réalisé l'héparinisation en donnant 90 mg/m² de surface corporelle d'héparine, à toutes les 90 minutes de circulation extracorporelle, nous donnions la moitié de la dose originale d'héparine. Nous avons neutralisé l'héparine en administrant 140 mg/m² de surface corporelle de sulfate de protamine. Une fois

la circulation extracorporelle établie, nous installons dans le péricarde ouvert un courant de 4 litres/min de CO₂. L'arrêt du cœur a été réalisé de façons diverses. Au début, nous avons employé du citrate de potassium et puis, plus tard, nous l'avons abandonné. Nous avons aussi pratiqué l'arrêt cardiaque par l'hypoxie à 30° C. Par la suite, nous avons fait l'arrosage au sérum physiologique salé à 40° C. De façon courante, nous préférons la perfusion des coronaires.

Comme méthode de choix pour défibriller un ventricule, nous avons remplacé le courant alternatif par du courant direct.

A la suite de ces 200 opérations, il est survenu 46 décès à l'hôpital, ce qui donne un taux de mortalité de 23 pour cent. Si l'on considère l'agent anesthésique principal comme facteur additionnel, le taux de mortalité serait comme suit : avec le N₂O aucun cas, avec l'halothane 8 cas, avec le fluroxène 1 cas, avec le méthoxyflurane 6 cas. Les autres causes de mortalité étaient : opération inadéquate 11 cas, débit cardiaque déficient 11 cas, hémorragie post-opératoire 4 cas, complications pulmonaires, 2 cas, maladie de l'artère coronaire 2 cas, divers 1 cas.

La durée moyenne de circulation extracorporelle pour les 154 malades qui ont survécu a été de 82 minutes, la durée de circulation extracorporelle des 46 malades qui sont décédés a été de 125 minutes.

Les complications post-opératoires ont été : l'alopecie, 8 cas, l'ictère, 8 cas, l'infarctus des organes, 11 cas, des embolies, 2 cas, des blocages cardiaques, 2 cas, des phlébites, 6 cas, de l'anurie, 7 cas, le syndrome postpéricardiotomie, 10 cas, des problèmes psychiatriques, 4 cas, et l'atélectasie, 9 cas.

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