PROFOUND HYPOTHERMIA AND THE HELIX RESERVOIR BUBBLE OXYGENATOR

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The success of open intracardiac surgery depends, to a large extent, on whether the technique used allows an accurate, unhurried correction of the lesion, and also on whether the procedure can be completed with a minimum of myocardial damage and without a significant change in the internal environment of the patient.

At normal body temperature this is possible to some extent by employing high-flow perfusion and cardiac asystole — a quiet heart being necessary for the accurate repair of the more complex lesions, and the high flow for normal oxygenation and metabolism — but both these techniques give rise to certain problems which hamper the surgeon in attaining his ultimate goal.

For example, when maintained for any length of time, the cardiac asystole causes myocardial damage, and high-flow perfusion causes excessive blood destruction and often prevents the surgeon from obtaining a bloodless field, especially in cyanotic patients with rich bronchial collateral circulation.

In an attempt to obtain ideal conditions the value of hypothermia used in conjunction with the pump oxygenator was investigated in various centres. The extracorporeal circulation abolishes the dangers of deep hypothermia to the myocardium, while the profound reduction in body temperature, and thus in body metabolism, allows much lower flow rates, resulting in drier operating conditions. It has been shown that hypothermic arrest of the heart is superior to potassium or anoxic arrest in maintaining myocardial energy resources, with the result that myocardial damage is less likely during the period when a quiet heart is necessary for the accurate repair of the defect.

Our experimental studies, and those of other workers, have shown that cold *per se* is not harmful, and that its effects can always be reversed. This paper deals with our clinical experience with hypothermia of 20°C. or lower in conjunction with the bubble oxygenator.

MATERIAL

From July to October 1960, 25 patients suffering from various congenital and acquired heart defects were operated on using this technique. Only patients with the more severe anomalies were selected and therefore atrial septal defects, isolated pulmonary stenoses and uncomplicated ventricular septal defects were not included in this series, since we still prefer to operate on these cases with high-flow normothermic perfusions.

In order to analyse the results and obtain a more accurate impression of the value and dangers of this technique, we have divided the patients into 2 groups. The first includes those patients in whom anatomical correction was possible (Table I), and the second group those in whom anatomical correction was not possible by reason of the nature of the defect (Table II). Thus the second group includes anomalies such as transposition of the great vessels, Ebstein's anomaly, tetralogy of Fallot with pulmonary arterial hypoplasia, and multiple cardiac lesions.

APPARATUS

The extracorporeal circuit is basically that which we have previously described for normothermic perfusions,[1-3] except that the venous blood is drained directly into the

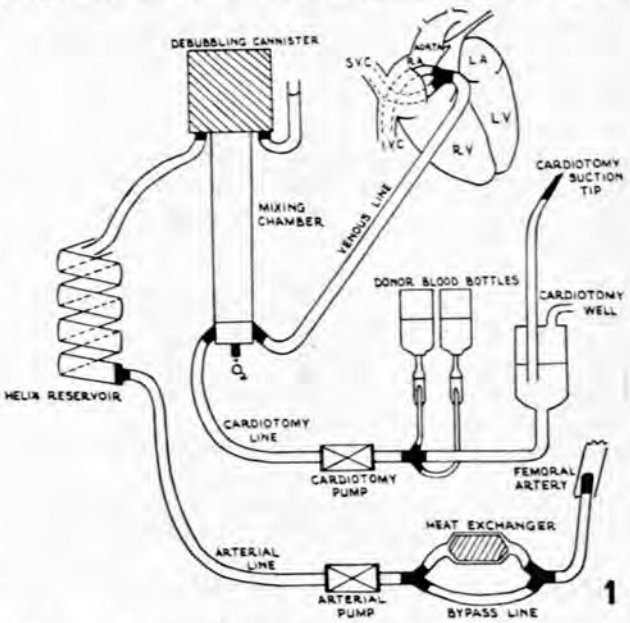

Fig. 1. The extracorporeal circuit employed with profound hypothermia and the bubble oxygenator.

mixing chamber, thus dispensing with the venous pump (Fig. 1). Since the patient's temperature is controlled by means of a heat exchanger in the arterial line, the helix reservoir is not immersed in a water bath.

A Bennington heat-exchange unit is used. This works on the principle that the blood entering it is spread in a thin film over a highly-polished inner component and so comes into contact with the similarly polished inner wall of the jacket carrying the heat exchange fluid. Water from a tank containing melting ice is used for cooling, and water between 40° and 45°C. for rewarming. The water is pumped through the jacket at a rate of 12 gallons per minute.

The number of heat-exchange units employed depends on the weight of the patient. Thus, for patients weighing up to 20 kg., 1 unit is adequate, for patients between 20 and 40 kg., 2 units, and for those over 40 kg., 3 units are used. When more than 1 unit is employed they are placed in parallel, not in series, thus reducing the resistance in the arterial line. In order to keep the patient's temperature

constant at the desired level, a line bypassing the heat exchanger is also incorporated in the arterial line.

TECHNIQUE

Anaesthesia, except for the hypothermic stage, is similar to that for operations involving normothermic perfusions.

During the hypothermic stage anaesthetic drugs are not needed, since 'cold narcosis'—as shown by complete electro-encephalographic (EEG) inactivity—is present. It is only during the precooling and warming periods of the bypass, when EEG activity is present, that drugs may be necessary.

Exposure for the operation is obtained by means of a median sternotomy. The preparations for cardiopulmonary bypass are as described previously,[1-3] except that both venae cavae are catheterized through the right atrial appendage, and the catheters are secured by means of a Rommel clamp. The arterial line is taken back to the patient through a stainless steel catheter in the right common femoral artery, except in children under the age of 18 months. In these small babies we find the common femoral, external iliac or common iliac arteries to be too small to take a catheter with an adequate internal diameter and therefore the arterial catheter is inserted through the ascending aorta with the tip pointing towards the aortic valve.

As soon as bypass is begun, the tapes round the venae cavae are tightened so that the patient's heart is completely bypassed. The flow rate during the period of cooling is estimated on the basis of 2·4 litres per square metre per minute for patients with a body surface up to 1 square metre and, for those with a greater surface area, 2·1 litres per square metre per minute. Unlike previous reports,[4,5] we cool on complete bypass with high flow rates. This has the advantage that the heart can be opened immediately and also that cooling is more rapid.

When the mid-oesophageal temperature reaches 20°C. the flow rate is reduced to one-third of the initial flow and maintained there throughout the cooled phase, unless for technical reasons the flow is stopped completely. When the desired temperature is reached, it is kept constant by allowing blood to flow through either the heat exchanger or the bypass line, as required.

Rewarming is commenced about 10 minutes before completion of the intracardiac repair, and the heart beat is restarted before the cardiotomy is closed, to permit decompression of the non-beating heart with the cardiotomy suckers, thus avoiding over-distension of the relaxed myocardium.

Since the flow rate is low during the cooled phase, the blood returning through the coronary sinus does not obscure the operative field and we therefore allow flow through the coronary vessels throughout the operation, except when tying sutures in a ventricular septal defect. For this, a completely relaxed heart is necessary and this is only possible when the aorta is cross-clamped and the myocardium is completely free of blood.

During rewarming the flow rate is rapidly increased until full flow is reached, thus shortening the rewarming time.

Bypass is discontinued as previously described.[1-3]

RESULTS

Survival

In Group I (Table I), 3 of the 18 patients did not survive. In case 10 a tracheotomy was required early in the postoperative phase to deal with excessive bronchial secretions; on the tenth postoperative day a fistula developed between the mediastinum and the tracheotomy wound and the patient died 24 hours later from an undetected air tamponade. Case 9 succumbed to a pulmonary embolus on the fifth postoperative day and case 13 died during surgery from aortic incompetence, following an attempt to correct a calcific aortic stenosis plus incompetence. None of these deaths can thus be blamed on the hypothermic technique.

TABLE I. LESIONS ANATOMICALLY REPAIRABLE BY PRESENT TECHNIQUES (GROUP I)

No.	*Case*	*Race/Sex/Age*	*Weight in kg.*	*Lesion*	*Flow rates (ml. per min.)*		*Lowest mid-oesophageal temp. in °C.*	*Duration of bypass in mins.*	*Outcome*
					Min.	*Max.*			
1	J.P.	EM 5½ years	15	Tetralogy of Fallot	880	1,536	15	113	Survived
2	D.M.	EM 15 years	50	VSD + pulm. infund. stenosis	1,000	3,250	18	72	Survived
3	I.P.	EM 21 years	64	VSD + pulm. infund. stenosis	1,000	3,822	18	85	Survived
4	P.N.	EF 4 years	12	VSD	800	1,320	19	92½	Survived
5	M.M.	CM 14 years	45	VSD + pulm. infund. stenosis	1,672	2,940	20	93	Survived
6	B.B.	CM 22 years	65	VSD	1,760	3,700	17	67	Survived
7	M.N.	AM 23 years	50	Tetralogy of Fallot	1,760	3,255	17	126	Survived
8	B.S.	EM 15 months	2·3	Tetralogy of Fallot	600	1,150	17	41	Survived
9	D.duP.	EM 30 years	82	VSD + pulm. infund. stenosis	1,200	3,870	19	131	Died
10	G.E.	EM 4½ years	16	VSD + pulmonary hypertension	880	1,800	16	73	Died
11	F.M.	EF 31 years	53	Acquired AS + AI	1,000	2,700	14·9	97	Survived
12	A.S.	EM 8 years	18	Tetralogy—re-operation on VSD	1,000	2,100	14·4	57	Survived
13	G.T.	EM 31 years	?	Calcific AS + AI	1,269	3,710	14·8	240	Died
14	J.B.	EM 27 years	59	VSD + AI	1,800	3,650	15	120	Survived
15	A.v.R.	EF 20 months	8	Idiopathic pulmonary hypertension	700	960	24	70	Survived
16	V.M.	CM 3 years	9	Tetralogy of Fallot	440	1,200	17	117	Survived
17	M.S.	EF 9½ years	23	VSD + PDA	800	2,118	17	130	Survived
18	C.McL.	CF 30 years	50	AS + MI	1,000	3,150	19·1	128	Survived
19	A.C.	CM 10 years	23	Tetralogy of Fallot	616	2,000	15·3	101	Survived

VSD=ventricular septal defect, pulm. infund. stenosis=pulmonary infundibular stenosis, AS=aortic stenosis, AI=aortic incompetence, PDA=patent ductus arteriosus, MI=mitral incompetence, E=European, C=Coloured, A=African.

The mortality in Group II (Table II), where an anatomical correction was not possible, was 100%. This also cannot be attributed to the perfusion technique. Three patients with tetralogy of Fallot malformation and hypoplasia of the main pulmonary artery are included in this group, since complete correction in one operation is not possible. We agree with Brock[9] that these defects should be corrected in two stages: at the first stage the right ventricular outflow obstruction is relieved and, after several months, when the pulmonary vessels have had time to develop due to the increased flow through the pulmonary circuit, the ventricular septal defect can be closed, at a second operation. In cases 8 and 16 in Group I, this was done with excellent results, after the first stage operation. The second operation, of course, has not yet been performed.

ADVANTAGES

1. *Hypothermic Arrest of the Heart*

In all the patients in this series ventricular fibrillation ensued when the myocardial temperature dropped below 25°C. The fibrillation became less vigorous as the temperature dropped, but continued unless the aorta was cross-clamped or bypass was discontinued. A quiet operative field was thus obtained by hypothermic arrest without the use of anoxia or drugs, thus avoiding the deleterious effects of those techniques.[7]

2. *Bloodless Operative Field*

This, to our minds, is one of the greatest advantages of profound hypothermia. The low flow rate reduces the amount of blood that rushes to the heart *via* the coronary sinus and the bronchial collateral vessels. This small return can easily be taken care of by the intracardiotomy suckers. Furthermore, bypass can be stopped completely, thus producing a dry heart, if desired.

3. *Diminished Danger of Metabolic Acidosis in Long Perfusions*

During normothermic bypass with high-flow perfusions the pH is maintained at a fairly constant level by the efficient elimination of CO_2 by the oxygenator. There appears to be a decrease in this function during prolonged perfusions. Base bicarbonate falls in a fairly regular fashion and it must be assumed that fixed acid excess is the cause of the base deficiency. Eventually there is a drop in the pH.

In profound hypothermia our experience has shown that in all the patients perfused for more than 1 hour, the pH remained constant and the base bicarbonate only dropped below 20 mEq. per litre in 2 patients out of 21.

It appears that profound hypothermia, even at the low flow rates employed during the cold phase, prevents accumulation of acid metabolites and decreases the danger of metabolic acidosis.

4. *Safety Margin Provided by Profound Hypothermia*

In high-flow normothermic perfusions, little time is allowed during bypass for any mechanical breakdown in the heart-lung machine, e.g. electricity failure, pump failure, etc. With profound hypothermia an interruption of perfusion for 30 minutes or more is tolerated and, during this time, any breakdown can be repaired. In fact, if it came to the worst, there would be enough time to convert the perfusion technique to the Drew technique.[9,10]

If a patent ductus arteriosus is diagnosed when the right ventricle is opened, this troublesome mistake in pre-operative diagnosis does not present any problem. The shunt into the pulmonary artery can be controlled by the surgeon introducing his finger into the pulmonary artery to block the opening of the ductus until profound hypothermic levels are reached. At that stage the circulation can be stopped completely and the ductus dissected and ligated.

DISADVANTAGES

1. *Longer Perfusions*

The time needed to reach and then reverse the profound hypothermia lengthens the total extracorporeal perfusion time. In our experience, profound hypothermia adds between 20 and 30 minutes to the total perfusion time; however, with more experience and better organization this disadvantage could be eliminated. Moreover, the quiet and bloodless operative field facilitates the closure of the ventricular septal defect or the reconstruction of the hypoplastic pulmonary artery in severe tetralogies. This shortens the total time required and is some compensation.

TABLE II. LESIONS ANATOMICALLY IRREPAIRABLE BY PRESENT TECHNIQUES (GROUP II)

No.	*Case*	*Race/Sex/Age*	*Weight in kg.*	*Lesion*	*Flow rates (ml. per min.)*		*Lowest mid-oesophageal temp. in °C.*	*Duration of bypass in mins.*	*Outcome*
					Min.	*Max.*			
1	M.S.	MM 40 years	41	Tetralogy of Fallot. Hypoplastic pulmonary artery	1,320	3,000	18	176	Died
2	M.C.	EF 9 months	2·3	Complete ECD with both pulmonary arteries and aorta arising from one ventricle. Hypoplastic pulmonary artery	300	600	16·1	114	Died
3	S.M.	CF 1 year	2·4	Tetralogy of Fallot. Hypoplastic pulmonary artery	Pump off	480	14	58	Died
4	S.J.	CF 8 years	15	Tetralogy of Fallot with hypoplasia of MPA and branches	600	1,600	16·4	234	Died
5	L.K.	CF 14 days	3	Ebstein's anomaly with 2 ostium secundum atrial septal defects + large PDA	520	700	14	61	Died
6	J.S.	EM 3 months	4	Transposition of great vessels + patent foramen ovale	Pump off	480	10	74	Died

ECD = endocardial cushion defect, MPA = main pulmonary artery, PDA = patent ductus arteriosus, M = Malay, E = European, C = Coloured.

2. *Greater Blood Destruction*

In our experience with the bubble oxygenator the haemolysis depends more on the length of perfusion than on the volume of flow. Therefore, with profound hypothermia — due to the longer period of bypass — the postoperative plasma haemoglobin levels were higher than with normothermic perfusions. This, however, has not been the experience of other workers employing disc and screen oxygenators.

3. *Postoperative Renal Failure*

Two of the patients in this series developed, postoperatively, the signs associated with tubular necrosis. In 120 normothermic perfusions we did not experience this complication and it was therefore decided to study the effects of profound hypothermia on the kidneys. This will be the subject of another report. It appears that kidney function returns to normal as soon as rewarming has taken place.

4. *Postoperative Morbidity*

It has been our experience that postoperative bleeding is more troublesome in the hypothermic cases than in the normothermic ones, and in this series there were 3 patients who had to be taken back to the operating theatre to control the bleeding. The disturbing feature is that the bleeding in all 3 cases started several hours after completion of the operation and the reason for this certainly requires further study.

CONCLUSIONS

Our experimental and clinical experience makes us believe that this technique has a very definite place in the repair of the more complicated cardiac lesions. The justification for its use in the simpler defects will come, if it does, only with more clinical and experimental experience.

In conclusion we would like to reiterate the statement made in a recent publication:[8] 'So far, the number of patients subjected to deep hypothermia is small . . . It may be some years before one can safely steer between the first rush of pioneer enthusiasm and over-biased inhibitory conservatism'.

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