SIMPLE DEEP HYPOTHERMIA FOR OPEN HEART SURGERY IN INFANCY

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TOTAL BODY HYPOTHERMIA made direct surgical repair of intracardiac lesions possible; however, due to complications, some of which were fatal, extracorporeal circulation utilizing cardiopulmonary bypass has been extensively investigated and utilized for open heart surgery.¹⁻⁴ Direct intracardiac surgery in newborns and small infants pose various problems to bypass techniques and attention has been redirected to deep hypothermia and circulatory arrest techniques either by simple surface cooling or by combined surface plus limited supplementary bypass cooling techniques.⁵⁻¹⁰

We have been interested in the experimental and clinical development of simpler and safer techniques for surface cooling and have utilized it exclusively for the surgical repair of various cardiac lesions in over 1,000 patients since 1959. The results have been satisfactory.⁸⁻¹² The present report concerns infants of less than 10 kg body weight with congenital cardiac lesions, in whom open heart surgery was performed utilizing simple surface cooling alone.

CLINICAL MATERIAL

From January 1968, to April 1976, 121 infants weighing less than 10 kg underwent open heart surgery under simple deep hypothermic arrest conditions. Of the 121 infants, results of 62 cases from January 1968 to December 1973 were reported in the *Japan Journal of Thoracic Surgery* in 1974.¹² In these infants, the mean age was 12.3 (± 5.6) months; the mean body weight was 7.8 (± 1.7) kg. Most of these infants were severely incapacitated and belonged to Class III-IV in ASA classification¹⁷ (Table I).

ANAESTHETIC MANAGEMENT

Hypothermia and rewarming were induced by immersion techniques under deep ether anaesthesia combined with the use of large quantities of ganglion blocking agents both as premedication and as supplement to ether anaesthesia.¹¹⁻¹² Haemoconcentration and sludging caused by hypothermia were prevented or at least minimized by the administration of 10% low molecular weight dextran (10 to 15 ml/kg), lactated Ringer's solution (10 ml/kg) and heparin (100 μ /kg). The objective temperature was 20° C in the oesophagus to allow one hour of circulatory occlusion time.

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TABLE I
OPEN-HEART SURGERY USING SIMPLE DEEP HYPOTHERMIA

Cardiac Defect	Age (months)		Total
	0 - 12	13 - 24	
A S D		3	3
V S D	24 (1)	9	33 (1)
V S D + P H	31 (3)	11	42 (3)
V S D + A S D	1	1	2
V S D + P D A + P H	1	1	2
V S D + P S		1	1
Aort. Pul. Sept. Defect	1		1
T O F	9 (1)	8 (3)	17 (4)
E C D (III)	1 (1)	3 (2)	4 (3)
T A P V D	6 (3)		6 (3)
T G A	2 (2)		2 (2)
Double Outlet rt. Ventricle	1 (1)	1	2 (1)
P S	2	1	3
Two Chambered rt. Ventricle	1	1	2
Persist. Trun. Art.	1		1
Total	81 (12)	40 (5)	121 (17)

() : Dead

A S D : Atrial Septal Defect. P D A : Patent Ductus Arteriosus.
V S D : Ventricular Septal Defect. P H : Pulmonary Hypertension.
P S : Pulmonary Stenosis. Aort. Pul. Sept. Defect : Aortic Pulmonary Septal Defect. T O F : Tetralogy of Fallot. E C D (III) : Endocardial Cushion Defect Complete. T A P V D : Total Anomalous Pulmonary Venous Drainage. T G A : Transposition of the Great Arteries. Persist. Trun. Art. : Persistent Truncus Arteriosus.

Cooling and rewarming

Anaesthesia was induced with intravenous thiamylal and maintained by closed circuit ether. As soon as routine ECG, EEG, direct intra-arterial catheter, oesophageal and rectal temperature monitoring devices were installed, slow intravenous administration of triflupromazine (3 mg/kg) was followed by immersion in ice water.

Deep ether anaesthesia is an important coadjutant for safe induction of hypothermia. The administration of ether requires careful attention to the various monitoring parameters and is given gradually as the body temperature decreases. In general, we have recommended administration of 1.5 ml/kg of ether, which will provide a blood level of 120 mg ether/dl by the time 30° C oesophageal temperature is reached, and 2.5 to 3.0 ml ether/kg that will give a blood level of 180 mg ether/dl of blood at temperatures below 25° C. Oesophageal and rectal temperatures follow a parallel course, the oesophageal being 1° to 2° C lower than the rectal temperature during the cooling phase. When the temperature gradient between these two sites reaches 4° C or more, it is indicative of inadequate peripheral perfusion.¹⁸

FIGURE 1

The active immersion cooling was discontinued at 1° or 2° C above the planned temperature. The oesophageal temperature continued to drop one or 2° C before it reached a plateau. The rectal temperature continued to fall until rewarming was started (Figure 1).

The mean oesophageal temperature was 20.8° C ($\pm 1.5^{\circ}$ C), and the mean rectal temperature 18.9° C ($\pm 2.1^{\circ}$ C). In 40 cases, the oesophageal temperature fell below 20° C and most were patients with complex cardiac defects. Shortly before the end of the intracardiac procedure the patient was reimmersed in water warmed to 43° C to 45° C to facilitate cardiac resuscitation. Rewarming was discontinued when the oesophageal temperature recovered to 35° C.

Haemodynamic changes

The blood pressure measured through the radial artery catheter fell as the body temperature decreased and the systolic pressure at 20.8° C oesophageal temperature, just prior to induction of circulatory arrest, was about 40 per cent of the precooling figures. As the body temperature rose the blood pressure recovered and

HAEMODYNAMIC CHANGES DURING HYPOTHERMIA

FIGURE 2

at 35° C, when rewarming was discontinued, it had returned to 86.5 per cent of precooling pressures.

The mean cardiac rate of 156/min before cooling represents tachycardia probably related to the premedication. The rate fell as body temperature decreased, the pre-occlusion rate being only 33.6 per cent of the precooling rate. At the termination of rewarming (35° C) the heart rate was 101.5 per cent of the initial value (Figure 2).

The most remarkable change in the ECG was the prolongation of P-R, QRS and R-R intervals caused by bradycardia. As the body temperature decreased, the QT interval¹⁹ or electric systole increased up to 164 per cent of the precooling level just prior to induction of circulatory arrest. As rewarming progressed it shortened to 0.425 sec. or control values (Figure 3).

Arrhythmias were rarely noticed during induction of hypothermia. They were observed only in two cases of ventricular septal defect and pulmonary hypertension (No. 600 and No. 674), occurring transiently at 26° C and 23° C respectively. Sinus rhythm was usually present until arrest was induced. During rewarming, just after the re-establishment of the cardiac beat and before the contraction had

CORRECTED Q-T INTERVAL (Q-Tc) DURING HYPOTHERMIA

FIGURE 3

recovered sufficiently, various types of arrhythmias were observed, but past this period, sinus rhythm was again usual.

Respiratory management and acid-base balance

Ether, given by closed system was the anaesthetic agent of choice. Spontaneous ventilation was maintained until respiratory depression became severe enough to require controlled ventilation (28–30° C). Since the same minute volume as that of normothermia was maintained, hypocarbia occurred as the temperature decreased.

During hypothermia some degree of anaerobic metabolism was observed with accumulation of fixed acids, mainly lactic acid, probably secondary to decreased liver function as has been reported in the past²¹⁻²⁴ (Figure 4).

Circulatory arrest

After the desired temperature was attained, circulatory arrest was obtained by inflow occlusion and cardioplegia induced by Young's solution²⁰ constituted of 0.3 per cent potassium citrate, 2.46 per cent magnesium sulfate and 0.001 per cent neostigmine, which was injected into the coronary arteries transaortically. The intracardiac procedure was performed in a completely flaccid heart. The mean circulatory arrest time was 40'20" and in 16 cases it extended to more than 60 minutes. These 16 cases were infants with complex cardiac malformations and their lowest temperatures, excepting four cases, reached figures below 20° C. The longest circulatory arrest was in an infant with transposition of the great arteries (No. 807), who underwent Mustard's operation during an arrest time of 77'20" (Table II).

Acid-Base Balance During Hypothermia

FIGURE 4

Cardiac resuscitation was accomplished by transaortic coronary artery reperfusion with 10 ml of a "cocktail" consisting of 2 per cent CaCl_2 10 ml, 20 per cent glucose 10 ml and 0.1 per cent noradrenaline 1 ml or by manual cardiac massage. The mean time required to resuscitate the heart was 4'47". There was no special correlation between resuscitative time and circulatory arrest time, or between resuscitative time and the lowest oesophageal temperature.

In 16 infants, resuscitation required more than 10 minutes. This difficulty may have been secondary to depression of cardiac function caused by operative manipulation immediately before induction of circulatory arrest, air embolization to the coronary arteries, A-V block, imperfect surgical repair, and excessive use of cardioplegic solution (Table III).

Morbidity and mortality

Ventricular fibrillation is the most serious reported complication of hypothermia. However, none of the patients in our study group developed it. Cardiac standstill was observed in seven infants. In five, it occurred concomitant to surgical manipulation of the heart for exploration and in preparation to induce circulatory arrest;

TABLE II
CIRCULATORY OCCLUSION TIME

Average 40'20" ($\pm 15'10''$ S.D.) (77'20" - 14'40")								
Cases over 60 min. of Occlusion Time								
No	Age (months)	Weight (kg)	Cardiac defect	Esopha. temper.	Rectal temper.	Occlu. time	Resusci. time	Outcome
727	22	7.5	T O F	17.7°C	15.0°C	63'35"	14'30"	Alive
733	16	9.5	T O F	19.0	17.0	75 45	7 45	Dead (1)
839	6	6.5	T O F	19.2	22.4	60 00	8 00	Dead (7)
849	15	9.5	T O F	17.9	14.0	60 00	30 00	Dead (6)
979	23	9.7	T O F	20.9	16.9	64 00	25	Alive
1001	9	7.3	T O F	18.5	15.0	76 40	9 00	Alive
821	11	6.0	V S D	19.2	17.2	60 00	14 20	Alive
932	12	7.5	V S D + P H	19.0	18.0	64 30	2 00	Alive
962	9	7.6	V S D + P H	21.5	19.2	66 00	3 00	Alive
757	7	4.8	E C D (III)	19.8	14.6	61 15	21 45	Dead (1)
868	17	7.0	E C D (III)	17.9	15.9	62 04	2 36	Dead (1)
876	18	6.8	E C D (III)	20.0	20.0	73 30	9 30	Dead (6)
807	9	6.1	T G A	17.7	13.2	77 20	18 35	Dead (3)
863	8	6.2	T G A	17.9	15.3	75 30	55 00	Dead (2)
859	6	5.1	A S D + V S D	20.1	18.5	67 00	16 00	Alive
777	17	8.0	Double Outlet	18.8	15.7	65 00	9 50	Alive

TABLE III
RESUSCITATION TIME

Average 4'47" ($\pm 8'53''$ S.D.) Cases over 10 min. of Resuscitation Time								
No	Age (months)	Weight (kg)	Cardiac defect	Esopha. temper.	Rectal temper.	Occlu. time	Resusci. time	Outcome
647	13	9.0	V S D	21.8°C	20.1°C	36 00	35 00	Alive
821	11	6.0	V S D	19.2	17.2	60 00	14 20	Alive
918	19	11.0	V S D + P S	19.8	17.7	38 05	30 00	Alive
959	10	5.8	V S D + P H	17.6	15.6	54 40	12 10	Alvie
727	22	7.5	T O F	17.7	15.0	63 35	14 30	Alvie
849	15	9.5	T O F	17.9	14.0	60 00	30 00	Dead (6)
967	10	8.5	T O F	20.8	18.7	47 30	16 13	Alvie
973	24	10.0	P S	19.4	14.9	49 50	12 30	Alive
863	8	6.2	T G A	17.9	15.3	75 30	55 00	Dead (2)
807	9	6.1	T G A	17.7	13.2	77 20	18 25	Dead (3)
758	10	8.4	T A P V D	21.3	17.5	57 30	19 50	Alvie
775	2	4.5	T A P V D	20.0	18.2	39 05	34 55	Dead (1)
890	10	6.9	T A P V D	18.1	16.6	38 30	19 00	Dead (1)
859	6	5.1	A S D + V S D	20.1	18.5	67 00	16 00	Alive
757	7	4.8	E C D (III)	19.8	14.6	61 15	21 45	Dead (1)
752	17	9.2	Two Cham. rt. Ventri.	19.0	19.2	41 15	11 15	Alive

a short period of cardiac massage sufficed to re-establish the heart beat. The remaining two infants (No. 600, ventricular septal defect with pulmonary hypertension and No. 1022, aortic pulmonary septal defect) developed severe bradycardia followed by cardiac standstill at 25° C and 27.5° C respectively while still

TABLE IV
POST-OPERATIVE RESPIRATOR MANAGEMENT

No	Age (months)	Weight (kg)	Cardiac Defect	Controlled Ventilation (days)	Outcome	Remark
821	11	6.0	V S D	53	Alive	A-V Block
844	10	7.0	V S D	23	Alive	
851	10	7.0	V S D	11	Alive	
755	9	5.2	V S D + P H	7	Dead (7)	Air Way Obstruction
869	4	4.5	V S D + P H	28	Alive	
877	9	7.0	V S D + P H	3	Alive	
880	18	9.5	V S D + P H	30	Alive	
883	6	6.6	V S D + P H	8	Alive	
922	12	6.5	V S D + P H	5	Alive	Pneumothorax
959	10	5.8	V S D + P H	8	Alive	
849	15	9.5	T O F	6	Dead (6)	Resp. Insuf. & L O S *
866	16	10.0	T O F	11	Alive	
878	22	9.0	T O F	5	Alive	
919	9	6.5	T O F	10	Alive	
967	10	8.5	T O F	6	Alive	Hemothorax
1001	9	7.3	T O F	3	Alive	
863	8	6.2	T G A	2	Dead (2)	Air Way Obstruction
859	6	5.1	V S D + A S D	11	Alive	
876	18	6.8	E C D (III)	6	Dead (6)	Renal Insuf.
777	17	8.0	Double Outlet	2	Alive	
890	10	6.9	T A P V D	3	Dead (3)	A-V Block & L O S *
991	12	7.8	Persist. Trun. Art.	13	Alive	
1022	6	7.0	Aort. Pul. Sept. Defect	3	Alive	

* L O S : Low Output Syndrom.

TABLE V
POST-OPERATIVE CNS DYSFUNCTION

No	Age (months)	Cardiac defect	Esopha. temper.	Rectal temper.	Occlu. time	Dysfunction	Outcome
755	9	V.S.D. + P.H.	21.6°C	18.5°C	39'50"	VII	Dead (7)
800	9	V.S.D.	20.8	18.0	51 40	III	Alive
807	9	T.G.A.	17.7	13.2	77 20	III	Dead (3)
876	18	E.C.D. (III)	20.0	20.0	73 30	III	Dead (6)
890	10	T.A.P.V.D.	18.1	16.6	38 30	III	Dead (3)

immersed in ice water. The operations were performed in the usual manner; once the hearts were exposed, they were resuscitated easily about two minutes' massage. The surgical repairs were completed during induced circulatory arrests of 36'45" at 23.6° C oesophageal temperature and 44'00" at 20.5° C. Post-operative courses were uneventful and left no sequelae.

Other reported complications of hypothermia, such as haemorrhagic tendency²⁵⁻²⁶ and "rewarming shock",²⁷⁻²⁸ were not observed in our patients.

Thirty-seven infants had prolonged post-operative apnoea or hypoventilation and required artificial respiration. Of these, 14 were extubated within 12 hours, the

TABLE VI
POST-OPERATIVE DEATH

No.	Age (months)	Death (days)	Cardiac defect	Respi. fail.	Circulatory failure			Cardiac Tamponade	Renal failure	Op. techni. failure	Brain damage
					L.O.S.	A-V Block	Corona. Thrombo.				
912	11	3	V.S.D.	○						○	
755	9	7	V.S.D. + P.H.	○*							○
913	9	2	V.S.D. + P.H.	○							
943	6	1	V.S.D. + P.H.					○			
713	19	8	T.O.F.		○						
733	16	1	T.O.F.	○!						○	
839	6	7	T.O.F.				○ ‡				
849	15	6	T.O.F.	○	○		○ ‡				
757	7	1	E.C.D.	○ #							
868	17	1	E.C.D.	○	○						
876	18	6	E.C.D.						○ §		
775	2	1	T.A.P.V.D. ‡	○							
890	10	3	T.A.P.V.D.		○	○					○
923	3	1	T.A.P.V.D.	○	○						
807	9	3	T.G.A.	○ **	○						○
863	8	2	T.G.A.	○ #							
953	12	1	Double Outlet rt. Ventri.		○						

* Respirator trouble. ** Hasty weaning of respirator. # Endotracheal tube trouble.
! Lungedema. ‡ Sudden death. § Disturbance of body fluid. ‡ Emergency cases.

apnoea being secondary to muscle relaxants and anaesthetics. The authors prefer artificial respiration to muscle relaxant antagonists. Five of the remaining 23 patients requiring more than 12 hours of ventilatory support died. In case ventilatory support is needed for more than 12 hours, controlled ventilation should be used until the weaning is adapted for better cardiopulmonary function. To prevent fighting the ventilator morphine (0.4 mg/kg) and diazepam (5 mg) may be used intravenously (Table IV).

Post-operative central nervous system dysfunction, transient or permanent, was observed in five patients. The infant with ventricular septal defect (No. 800) had mental disturbance for four days but recovered without any sequelae. The infant with endocardial cushion defect (No. 876) regained normal level of consciousness on the fourth day but succumbed on the sixth post-operative day secondary to fluid and electrolyte difficulties. The remaining three infants died without recovering consciousness (Table V).

Seventeen infants (17/121 = 14 per cent) died post-operatively. The cause of death was difficult to determine clearly, but the single most important cause was respiratory failure. Some of the deaths could have been prevented, such as in Case No. 757, an infant with endocardial cushion defect, on whom the tracheal tube slipped out of the trachea inadvertently. In Case No. 755 (ventricular septal defect with pulmonary hypertension), power failure of the respirator occurred, and in Case No. 863 (transposition of great arteries) cardiac arrest took place while changing tracheal tubes. The infant with transposition of the great arteries (No. 876) was weaned off the respirator too soon. Two infants with tetralogy of Fallot (No. 837 and No. 849) had sudden cardiac arrest on the sixth and seventh

post-operative day respectively and could not be resuscitated; autopsy revealed findings compatible with coronary artery embolization. The infant with total anomalous pulmonary venous drainage (No. 775) was operated upon in emergency, with active pneumonia prior to operation and succumbed one week post-operatively from progression of the pulmonary infection. Case No. 876, an infant with endocardial cushion defect, died from water and electrolyte imbalance secondary to profuse polyuria of obscure origin (Table VI).

DISCUSSION

Simple deep hypothermia lacks the stressful effects of non-physiological extracorporeal circulation and has a number of advantages for infant open heart surgery, such as reduced need of blood and a completely still and bloodless operative field unobscured by perfusion catheters and tubes that may constitute great obstacles in the exposure of intracardiac defects, especially in small hearts.

On the other hand, a number of shortcomings may be mentioned. These include the reported danger of ventricular fibrillation,¹⁻⁴ time limitations of circulatory arrest,¹⁰ anxiety about cardiac resuscitation,²⁹⁻³¹ haemorrhagic tendency,²⁵⁻²⁶ and "rewarming shock".²⁷⁻²⁸

To eliminate these disadvantages, especially ventricular fibrillation, many studies have been reported by various authors. Ōta⁵ reported the usefulness of the powerful peripheral vasodilating effects of phenoxibenzamine at the dose of 1 mg/kg. Barratt-Boyes and others^{6,15} combined surface cooling to 25° to 26° C and core cooling to 20° C by a short supplementary bypass prior to induction of circulatory arrest, with bypass rewarming. Mori¹⁵ reported the applicability of halothane anaesthesia for the induction of hypothermia with this combined method. Steward¹⁴ observed ventricular fibrillation in six of the 16 patients cooled below 26° C by surface cooling alone. One of these six patients was anaesthetized with halothane; the other five were under nitrous-oxide-narcotic curare anaesthesia. Steward stressed the necessity of using assisted circulation at temperatures below 30° C. Mohri³² compared ether and halothane anaesthesia in animal studies and pointed out the strong tendency toward hypotension during rewarming, and a significant incidence of post-operative gait disturbance when hypothermia was induced under halothane anaesthesia.

We have reported that deep ether anaesthesia is the ideal agent for induction of hypothermia by surface cooling, especially when combined with ganglion blocking agents in large quantities to eliminate some of the undesirable effects of ether, thus improving and maintaining good peripheral perfusion. Temperatures below 20° C can be safely and predictably obtained by this method.⁸⁻¹² In our 121 patients, arrhythmia related to hypothermia was seen in only two patients. Frank cardiac standstill occurred in only two cases, since in five others it was precipitated by surgical manipulation of the heart. Ventricular fibrillation, the most dangerous complication, could be prevented completely by this method. Advantages of ether anaesthesia for induction of hypothermia has been reported by many others such as Mohri,³² Okamura,³³ and Okada.³⁴

Regarding restriction of circulatory arrest time and the consequent anxiety about

resuscitation, our results in cases requiring more than 60 minutes of circulatory arrest were not good, but exceeding of the accepted safe limits could not be ascribed as the only cause of death. The majority of these cases had complex anomalies and were in poor clinical condition and, in spite of this, all hearts could be resuscitated after completion of the surgical repair. We would predict safe arrest times of 90 to 120 minutes if temperature could be lowered adequately. Our clinical and experimental studies indicate safe circulatory arrest times of eight minutes at 30° C body temperature, 30 minutes at 25° C, 60 minutes at 20° C, and 120 minutes at 15° C. We believe that almost all operable congenital diseases of the heart can be corrected within this time frame.

Many studies directed to facilitating resuscitation of the heart have been done and reported: coronary perfusion,²⁹ supplementary extracorporeal circulation,^{6,15,30} cardiac pacing,³¹ and cardiac massage.⁸⁻¹⁰ We have adopted the cardiac massage or manual coronary perfusion. Some of the cases requiring long periods of circulatory arrest also required longer times to resuscitate the heart, but pre-operative myocardial damage and severe depression of cardiac function immediately before arrest might have played an important role besides the arrest time. It stresses the need to maintain optimal haemodynamic conditions prior to induction of circulatory arrest.

The so-called haemorrhagic tendency^{25,26} and rewarming shock^{27,28} have been reported as complications during hypothermia. In addition to the known changes of coagulation function with hypothermia and the use of anticoagulant agents during surgery, the normally occurring hypotension under hypothermic conditions may mask true bleeding points; thus the possibility of post-operative bleeding is undeniable. However, careful surgical haemostasis combined with the trans and post-operative use of haemostatic agents, mainly antiplasminic drugs, have allowed us to prevent re-operation or death from haemorrhage.

Though ether has the possibility of explosion as a hazard if the electric cautery is used for surgical haemostasis, a complete closed circuit method prevents this hazard. In over 1,000 cases we have not experienced an explosion.

The "rewarming shock" is probably caused by the "wash-out acidosis" in conjunction with the inadequate recovery of cardiac function to meet the increased body oxygen demand that accompanies the rise of temperature, and usually occurs above 28° C. With our technique, the recovery of cardiac function parallels the increasing metabolic needs of rewarming.³⁵ This complication was never observed.

Metabolic acidosis and hypokalaemia are two constant changes observed during hypothermia, and have been described as causes of arrhythmias and ventricular fibrillation. To minimize this complication, manoeuvres directed to alter metabolic changes such as hypercapnoea,^{23,24} hypocapnoea,^{13,32} administration of acid³⁶ or alkaline³⁷ agents, and injection of KCl⁵ have been reported, but our studies³⁸ delineating the significant band of these changes at various body temperatures do not seem to implicate these metabolic alterations as the main cause of ventricular arrhythmias. The implications of metabolic acidosis under hypothermia do not seem to be as significant as in normothermic conditions.³⁸ Hypokalaemia is a physiological change with decreasing temperatures and its degree is well within safe limits, as we have reported in the past.³⁹ Based on these principles, correction

of the metabolic acidosis is restricted only to patients with severe hypotension, marked bradycardia, or difficult cardiac resuscitation. Sodium bicarbonate is the preferred agent and the degree of correction aimed at B.E. of -10 mEq/l only when oesophageal temperature is around 20° C. Hypokalaemia is not actively corrected during the hypothermic state.

To obtain the best operative results, especially in small infants, particular care of the respiratory management is mandatory. The anatomic peculiarity of the respiratory system in infants, the weakness of respiratory muscles, and the relatively large sized intra-abdominal organs makes respiratory failure a constant threat. Downes⁴⁰ reported that 55 per cent of infants undergoing "closed cardiac surgery" required post-operative artificial respiration, and 49 per cent of them died. Barrat-Boyes⁶ reported the use of artificial respiration in seven infants out of 37 cases following open heart surgery. Twenty per cent ($23/121 = 19.0$ per cent) of our patients required artificial respiration post-operatively for more than 12 hours, and five died ($5/23 = 21.7$ per cent). Our current indications for artificial ventilation include not only the obviously hypoxaemic patient secondary to respiratory or circulatory failure, but also the patient that required a circulatory arrest time of over 50 minutes. Although some authors⁴¹ recommend tracheostomy even in small infants we believe that nasal intubation is adequate to provide even long-term respiratory management. In fact we used tracheostomy only in one infant, early in our experience.

SUMMARY

Results of open cardiac surgery under deep simple hypothermia in 121 infants with body weight of less than 10 kg are reported. Deep ether anaesthesia combined with large quantities of ganglion blocking agents (triflupromazine 3 mg/kg) constitutes the anaesthetic management of choice for deep surface-induced hypothermia. The mean lowest oesophageal temperature was 20.8° C, and 18.9° C rectally. The mean circulatory arrest time was 40 minutes. Seventeen infants (14.0 per cent) died post-operatively. There were no operative deaths attributable to failure of cardiac resuscitation.

This technique widens the scope of open heart surgery in small infants. Most of the surgically correctable malformations should be operable by this method. More than the potential hazards of hypothermia, which we believe are solved by our technique, the major problem posed by surgery in these small infants is the trans and post-operative respiratory management.

RÉSUMÉ

Les auteurs rapportent leur expérience de 121 enfants de moins de 10 kilos (poids moyen: 7.8 kilos), opérés à cœur ouvert, sous hypothermie profonde, au cours des huit dernières années. Quatre-vingt-un de ces enfants étaient de moins d'un an (âge moyen: 12.3 mois).

Ces enfants ont été opérés sous C.E.C., en hypothermie profonde permettant des arrêts circulatoires de 14 à 77 minutes (moyenne: 40 minutes 20 secondes).

L'hypothermie était produite par immersion en bain d'eau glacée sous anes-

thésie profonde à l'éther et sous protection de bloqueurs sympathiques à hautes doses. La moyenne des températures œsophagiennes atteintes se situait à 20.8° C, et celle des températures rectales à 18.9° C.

Aucun décès n'est relié à des causes anesthésiques ou à la réanimation du myocarde. Cependant, les soins post-opératoires respiratoires ont influencé significativement le pronostic. Dix-sept enfants (14 pour cent) sont morts en post-opératoire.

Les auteurs concluent que l'hypothermie profonde est une méthode simple pour la chirurgie ouverte des tout jeunes enfants.

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