Original Article

Moderate Hypothermia with Low Flow Rate Cardiopulmonary Bypass in Congenital Heart Defect Surgery

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ABSTRACT

Low flow rate perfusion has been recommended in profound hypothermic cardiopulmonary bypass (CPB) in recent years, but has not been used in moderate hypothermic CPB. In this report, 30 patients with congenital heart defects, from 2 to 11 years old and weighing 11.5 to 25 kg. were selected to be the subjects of moderate hypothermia with low flow rate perfusion. Once on CPB, a high flow rate of 2.27 ± 0.36 L/min/m^2 was used to cool the patient to 25.6 ± 0.84°C rectal, 24.1 ± 1.32°C esophageal, and 23.8 ± 1.4°C tympanic temperature, followed by a low flow rate of 1.23 ± 0.09 L/min/m^2 until the main intracardiac repair was completed. Rewarming to a rectal temperature of 34.5°–35.0°C was accomplished with a high flow rate of 2.70 ± 0.22 L/min/m^2 until weaning. The total CPB, cross clamp, and low flow rate perfusion times were 95.4 ± 34.6 min, 51.4 ± 20.2 min, and 45.7 ± 22.4 min respectively. A second group of five patients from 1.5 to 4 years old and from 6 to 11 kg were operated on with profound hypothermic circulatory arrest. A high flow rate of 2.35 ± 0.43 L/min/m^2 was used to cool the temperature to 19.3 ± 0.8°C rectal, 17.5 ± 2.2°C esophageal, and 17.8 ± 1.5°C tympanic, and then the circulation was temporarily arrested. The CPB and arrest time were 55.0 ± 10.7 min and 44.7 ± 3.8 min respectively. Among the patients under moderate hyperthermia with low flow rate perfusion, only one showed metabolic acidosis during cardiopulmonary bypass and received an extra 12 mEq sodium bicarbonate. After 27 to 99 min low flow rate perfusion, the venous oxygen saturation was still greater than 80% for each patient and lactate concentration did not increase. In contrast, among those cases using profound hypothermic circulatory arrest, the blood gas analysis after two min of rewarming demonstrated an obvious metabolic acidosis and increase in lactate concentration. An extra 9 to 24 mEq sodium bicarbonate was needed in each of five patients for acidosis correction. After the sodium bicarbonate administration, the blood gases returned to normal while the lactate concentration still increased progressively. The data from this study suggest that low flow rate perfusion may safely be used in moderate hyperthermic CPB as long as we monitor the oxygen saturation of returned venous blood, keeping it above 80%.

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INTRODUCTION

Profound hypothermia with circulatory arrest has been used for decades in cardiac surgery for neonates and small infants, especially those with complicated heart defects (1, 2). Although 60 minutes of arrest time has been considered tolerable, reperfusion injury and subclinical pathophysiological changes are evident. Neurological complications have been reported by several authors (3, 4, 5). Currently, some authors recommend profound hypothermia with low flow rate perfusion, and it is considered beneficial to the patient's recovery (6, 7, 8), but in moderate hypothermic cardiopulmonary bypass (CPB), high flow rates are still suggested. We undertook this clinical study to determine the possibility of using low flow rate perfusion in moderate hypothermic CPB.

MATERIALS AND METHODS

PATIENTS:

Thirty patients with an age range from 2 to 11 years (5.1 ± 2.6 years), and a weight range from 11.5 to 25 kg (15.6 ± 3.2 kg) were the subjects of this study of moderate hypothermia with low flow rate perfusion. The cardiac defects were: Tetralogy of Fallot (n=22), Total Anomalous Pulmonary Venous Connection (n=3), Levotransposition of Great Vessels (n=1), Double Outlet Right Ventricle (n=2), and Ventricular Septal Defect with pulmonary hypertension (n=2).

Another five patients with a weight less than 11 kg (9.8 ± 2.2 kg) were selected for profound hypothermic circulatory arrest. Their ages were from 1.5 to 3.6 years (2.4 ± 0.7 years). All five patients were diagnosed with Tetralogy of Fallot.

CARDIOPULMONARY BYPASS:

Moderate hypothermia with low flow rate: CPB was established as previously described (9). Anesthesia was induced with fentanyl citrate (7.5 ug/kg). Heparin was administered at an initial concentration of 2 mg/kg into the right atrial appendage and 20 to 30 mg in the prime. Activated clotting time was maintained at greater than 450 seconds throughout CPB. A membrane oxygenator[a,b] and roller pump[c] were used in the extracorporeal circuit. Crystalloid solution and packed red blood cells, if necessary, were used for the prime. A high flow rate of 2.27 ± 0.36 L/min/m^2 was used to cool the patients to 25.6 ± 0.84°C rectal, 24.1 ± 1.32°C esophageal, and 23.8 ± 1°C tympanic temperature, followed by a low flow rate of 1.23 ± 0.09 L/min/m^2 until the main intracardiac repair was completed and rewarming started with a high flow rate of 2.70 ± 0.22 L/min/m^2. The blood pressures were from 30 to 50 mmHg during low flow rate perfusion. The patients were weaned from CPB when the rectal temperature was at 34.5°C to 35.0°C. The hematocrit during CPB was 21.4 ± 4.2%. After discontinuation of CPB, heparin was neutralized with protamine sulfate. The remaining blood in the bypass circuit was returned to the patient through the external jugular vein. The total CPB time was 95.4 ± 34.6 min, cross clamp time was 51.4 ± 20.2 min, and low flow rate perfusion lasted 45.7 ± 22.4 min.

Profound hypothermia with circulatory arrest: The anesthesia and CPB circuit were the same as that for moderate hypothermia with low flow rate. A high flow rate of 2.35 ± 0.43 L/min/m^2 was used to cool the patients to 19.3 ± 0.8°C rectal, 17.5 ± 2.2°C esophageal, and 17.8 ± 1.5°C tympanic temperature, respectively. CPB was temporarily stopped after aortic cross clamping. After the completion of intracardiac repair, CPB was restored, and the temperature was rewarmed with a high flow rate to 34.5°C to 35.0°C rectal temperature. Hematocrit during CPB was 22.7 ± 2.1%. CPB lasted 55.0 ± 10.7 min, and arrest time was 44.7 ± 3.8 min.

SAMPLING:

In cases performed with moderate hypothermia with low flow rate, arterial and venous blood samples were drawn from the pump circuit at 2 min after the initiation of CPB, the start of low flow rate perfusion, at the beginning of rewarming, and just before weaning from CPB.

In cases performed with profound hypothermia with circulatory arrest, sampling was done at 2 min after the initiation of CPB, at the beginning of circulatory arrest, 2 min after rewarming, and just before weaning.

Immediately after sampling, all the samples were sent to the laboratory for blood gas analysis with a blood gas analyzer[d] and lactate concentration with a lactate analyzer[e].

RESULTS

Among 30 cases under moderate hypothermia with low flow rate perfusion, only one patient showed metabolic acidosis during CPB and required an extra 12 mEq sodium bicarbonate. After 27 to 99 min low flow rate perfusion, venous oxygen saturations remained above 80% for each case, and lactate concentrations did not increase. All the patients except one had adequate urine output from 50 to 600 ml (199 ± 155 ml) during CPB. The results are listed in Table 1.

In those cases using profound hypothermic circulatory arrest, all the samples drawn 2 min after rewarming showed obvious metabolic acidosis. Blood pH decreased from a pre-arrest value of 7.39 ± 0.02 to a post-arrest value of 7.26 ± 0.04, and the base deficit from 2.11 ± 0.52 mmol/L to -4.24 ± 2.61 mmol/L. Lactate concentration increased from 3.56 ± 0.21 mmol/L to 4.82 ± 1.10 mmol/L. An additional 9 to 24 mEq sodium bicarbonate was needed for each of the 5 patients for acidosis correction. After the bicarbonate administration, the blood gas re-

a Minimax, Medtronic, Inc., Anaheim, CA
b Baxter Healthcare Corp., Irvine, CA
c Sarns 7400, Sarns, Inc./3M Health Care, Ann Arbor, MI
d Model 990, AVL Corp., Graz, Austria
e Model 23L YSI Corp, Ohio, USA

turned to normal, while the lactate concentration still increased progressively to 6.32 ± 1.31 mmol/L just before weaning (Table 2).

All the patients in both groups recovered well after the operation. No surgical deaths or neurological complications occurred.

DISCUSSION

Perfusion flow rate is an extremely important parameter for CPB. Maintenance of adequate perfusion flow rate is necessary to meet tissue metabolic needs. Previous studies have demonstrated that the metabolic rate decreases to about 60% of baseline at 28°C and to 40% at 25°C body temperature. Furthermore, the metabolic rate decreases another 15% under anesthesia (10). So, during CPB with cooling to 25°C to 28°C, which is considered moderate hypothermia, the metabolic rate is about 35 to 50% of the basal metabolic rate measured at physiological body temperature.

Generally, the tissue extracts oxygen mainly from hemoglobin bound oxygen for metabolic needs. Free oxygen accounts for only a minimal portion of tissue needs. In CPB, the blood is hemodiluted and thus the bound oxygen decreases in accordance with the degree of hemodilution. Fortunately, the partial pressure of oxygen during CPB generally increases because of the higher inspiratory oxygen fraction (FiO_2), which means the extractable free oxygen for metabolism increases and partially compensates for the reduced bound oxygen. It is estimated that the total extractable oxygen is about 75% of that presurgically if the hemoglobin concentration is half the presurgical value, arterial oxygen partial pressure is controlled at 300 mmHg, and venous oxygen saturation is maintained at 75%.

Metabolic acidosis exists whenever the peripheral perfusion flow rate is not adequate (6). In this circumstance, the blood pH and base deficit will decrease and lactate concentration will increase. If the oxygen deficiency is slight, the acidosis can be compensated for by the blood buffer system and blood gas analysis remains normal. But increased glycolysis under oxygen deficiency will lead to increased blood lactate concentration. So the lactate is a more sensitive indicator of early oxygen deficiency than blood gas analysis (3). The accumulation of acid materials in the presence of severe oxygen deficiency will exhaust the compensatory ability of the blood buffer system, and the results of blood gas analysis will demonstrate an abnormal pH and base deficit. Surely, the blood lactate concentration will increase progressively. Administration of bicarbonate

Table 1: Results of blood gas analysis and lactate measurement in moderate hypothermia with low flow rate.

		2 min on CPB	Start Low Flow	Rewarming	Before Weaning
pH	arterial	7.40 ± 0.07	7.37 ± 0.07	7.42 ± 0.07	7.43 ± 0.08
	venous	7.37 ± 0.07	7.34 ± 0.06	7.38 ± 0.06	7.37 ± 0.07
pO_2 (mmHg)	arterial	328.7 ± 212.4	406.4 ± 140.4	412.9 ± 153.5	300.1 ± 131.1
	venous	71.9 ± 71.7	130.4 ± 121.8*	55.5 ± 15.1	47.4 ± 12.3
pCO_2 (mmHg)	arterial	41.8 ± 7.8	44.8 ± 8.5	39.6 ± 9.3	37.9 ± 9.7
	venous	42.8 ± 10.8	49.2 ± 8.0	46.0 ± 9.6	44.9 ± 9.3
O_2sat (%)	arterial	98.8 ± 1.9	99.8 ± 0.1	99.8 ± 0.2	99.7 ± 0.3
	venous	83.4 ± 11.9	94.5 ± 4.9*	85.2 ± 7.6	75.1 ± 17.9
BE (mmol/L)	arterial	0.47 ± 2.99	0.69 ± 2.89	1.05 ± 2.87	0.66 ± 3.06
	venous	0.96 ± 3.07	-0.03 ± 3.05	0.98 ± 3.05	0.54 ± 3.20
Lact (mmol/L)	arterial	3.76 ± 1.32	3.97 ± 1.15	3.72 ± 0.89	3.73 ± 1.04
	venous	3.71 ± 1.18	3.90 ± 1.01	3.63 ± 0.95	3.90 ± 1.04

* $p < 0.05$, compared with 2 min on CPB

Table 2: Results of blood gas analysis and lactate measurement in profound hypothermic circulatory arrest.

		2 min on CPB	**Start Arrest**	**Rewarming**	**Before Weaning**
pH	arterial	7.39 ± 0.02	7.36 ± 0.05	7.26 ± 0.04*	7.48 ± 0.05
	venous	7.38 ± 0.03	7.36 ± 0.06	7.18 ± 0.01*	7.39 ± 0.08
pO_2 (mmHg)	arterial	396.2 ± 40.9	356.4 ± 66.8	344.7 ± 137.2	287.2 ± 171.1
	venous	76.1 ± 42.5	133.5 ± 105.2*	108.5 ± 88.0*	90.5 ± 51.5
pCO_2 (mmHg)	arterial	44.2 ± 1.9	44.4 ± 1.2	53.4 ± 8.9*	34.8 ± 5.9
	venous	46.9 ± 7.4	47.4 ± 6.9	66.5 ± 7.5*	43.3 ± 10.2
O_2sat (%)	arterial	99.9 ± 0.1	99.8 ± 0.1	99.7 ± 0.3	99.1 ± 1.4
	venous	87.4 ± 14.6	99.6 ± 0.1*	85.6 ± 14.5	89.7 ± 8.0
BE (mmol/L)	arterial	2.11 ± 0.52	-0.12 ± 0.92	-4.24 ± 2.61*	3.30 ± 1.41
	venous	1.12 ± 2.59	-0.59 ± 2.07	-5.42 ± 1.51*	1.42 ± 0.61
Lact (mmol/L)	arterial	3.56 ± 0.21	3.82 ± 0.60	4.82 ± 1.10*	6.32 ± 1.31**
	venous	3.54 ± 0.31	3.64 ± 0.89	4.34 ± 1.21*	5.80 ± 1.42**

* $p < 0.05$, compared with 2 min on CPB; ** $p < 0.01$ compared with 2 min on CPB

will adjust the pH and base deficit, but it cannot make the increased lactate return to normal over a short period. Lactate levels are only normalized after a long time of oxidative metabolism.

Also, the tissue oxygen extraction rate increases in the presence of poor perfusion, leading to a greater arterial-venous oxygen difference and lower venous oxygen saturation. Like the lactate concentration, the venous oxygen saturation is another sensitive indicator of the adequacy of perfusion.

In this report, 30 patients were operated on using moderate hypothermia with low flow rate perfusion. The results showed that after 27 to 99 minutes low flow rate perfusion, the venous oxygen saturation did not decrease to below the normal limit, and in each case, remained higher than 80%. The other parameters of blood gas analysis were also within normal limits. Similarly, the lactate did not increase. All but one of the patients did not require extra sodium bicarbonate during CPB. In contrast, in those patients operated on under profound hypothermia, obvious metabolic acidosis occurred in all 5 cases after 39 to 47 min of circulatory arrest. The lactate also increased. After the addition of bicarbonate, the blood gas analysis returned to normal, but the lactate did not decrease to normal, instead increasing progressively because of further influx of lactate into the circulation following the opening of microcirculation. The average increase in lactate was about 80% compared to the prearrest value.

Although profound hypothermia with circulatory arrest is a valuable method of cardiopulmonary bypass in cardiac surgery for neonates and small infants with complex congenital heart defects, there still exists some degree of oxygen deficiency and acidosis after arrest, which may be important factors associated with postoperative reperfusion injury and subclinical pathophysiological changes. In recent years, profound hypothermia with low flow rate was recommended by some authors (6-8), but high flow rate is still used in those patients with moderate hypothermia. Extended high flow rate perfusion will result in severe damage to blood components and increase the quantity of returned blood from collateral vessels to the heart, which also influences the surgical procedure and cardiac preservation. The results from our study demonstrate that low flow rate perfusion can be used during moderate hypothermia, as long as the oxygen saturation of returned venous blood is kept over 80%, and tissue oxygen insufficiency and metabolic acidosis will be unlikely to occur.

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