

Original Articles

A Novel Technique of Antegrade Cerebral Perfusion in the Newborn with Critical Aortic Stenosis

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Abstract: Various methods of cerebral protection have been used during such aortic arch operations as the Norwood Procedure and operations on the interrupted aortic arch in neonates and infants. Deep hypothermia with circulatory arrest is the most common technique, but has a limited safe period for circulatory arrest. Antegrade cerebral perfusion has been introduced to pro-

long this safe period. We reviewed our experience with antegrade cerebral perfusion during surgical repair, in a patient with hypoplastic left heart syndrome in stage 1 palliation. **Keywords:** antegrade cerebral perfusion, neonates, cerebral protection. *JECT. 2004;36:129–132*

Frist et al. and Kazui, et al. (1–3) describe a technique that uses partial brachiocephalic selective cerebral perfusion in adults. A perfusion of both carotids may be necessary only in patients with a compromised circle of Willis or severe carotid artery stenosis.

Frist's use of moderate systemic hypothermia may reduce the risk of excessive bleeding and decrease the complications associated with long CPB times, such as the capillary leak syndrome, by avoiding low temperatures and prolonged cooling and rewarming periods associated with deep hypothermic circulatory arrest.

Aortic arch reconstruction such as the Norwood procedure has commonly been required in deep hypothermic circulatory arrest. Although it is a useful technique, potential risks of such complications as neurological damage cannot be ignored (4). We introduced a cardiopulmonary bypass technique using a double arterial cannulation of the pulmonary truncus and the Blalock–Taussig Shunt (BT-Shunt) through median sternotomy combined with antegrade cerebral perfusion through the right subclavian artery (via BT-Shunt) to avoid circulatory arrest.

Surgical Technique

The surgical technique in a patient with hypoplastic left heart syndrome and antegrade cerebral perfusion is as

follows. After median sternotomy and full heparinization, the ascending aorta, pulmonary trunk, truncus brachiocephalicus, and the right subclavian artery were mobilized. A 3.5 mm BT-Shunt (Gore, Flagstaff, AZ) was constructed end-to-side to the right subclavian artery. After this, the main pulmonary artery and the BT-Shunt were cannulated with an 8 F polyurethane cannula (Stöckert Inst., Munich). The right atrium was cannulated with a 16 F single venous cannula (Terumo Cardiovascular Systems, Ann Arbor, MI).

Before CPB was initiated, the right and the left pulmonary arteries were clamped to improve hemodynamic stability by decreasing pulmonary artery run-off, while maintaining adequate oxygen saturation. Pump flow rate was maintained at 2.8 L/m²/min, the rectal temperature was lowered to 22°C, the hematocrit and the protein were adjusted to 22% and 2.6 g/dL, the colloid osmotic pressure to 14 mmHg, and for the blood gas management, we used the alpha-stat management. At a rectal temperature of 22°C, the pulmonary truncus cannula was clamped, the ductus arteriosus was ligated, the pulmonary truncus was transected, and the distal end was closed. After clamping all the arch vessels and the descending aorta, the pump flow rate was reduced, and the antegrade cerebral perfusion was started with a flow from 30 mL/kg/min, and a cerebral perfusion pressure about 40–80 mmHg (Table 1).

The normal principles of cerebral autoregulation that

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are present during normothermia are maintained during moderate hypothermic CPB. Cerebral blood flow is dependent on brain metabolism. If metabolism is high, the cerebral vascular resistance falls, and the cerebral blood flow increases. This is known as flow/metabolism coupling, and it remains intact during moderate hypothermic CPB. Pressure/flow autoregulation or the ability to maintain a constant CBF despite wide ranges in mean arterial pressure also remains intact during moderate hypothermic CPB. The cerebral vasculature remains capable of dilating during low perfusion pressure and constricts when perfusion pressure is high. In temperatures under 22°C and/or pH-stat management this benefit where lost (5).

A longitudinal incision of the aortic arch was made. The neo-aorta was constructed with a homograft, direct anastomosis of the pulmonary trunk to the ascending aorta and the aortic arch. The perfusion to the BT-Shunt was stopped, and the whole body was perfused with 2.8 L/kg/min by the arterial cannula in the neo-aorta. After reperfusion, CPB was stopped for a few minutes to enlarge the arterial septal defect through the right atriotomy. Modified BT-Shunt was completed by anastomosing the distal end of the graft to the right pulmonary artery. At a temperature about 30°C, the patient parameters were corrected to the following levels pH (7.4), calcium (1.5 mmol/L), calcium (4.5 mmol/L), colloid osmotic pressure (20 mmHg), and hematocrit (34%). The patient was rewarmed to 36°C, with active hemofiltration during the re-warming phase.

Weaning from CPB was accomplished in a slow and controlled manner, balancing the Qp/Qs ratio. Patients with a low pulmonary vascular resistance to systemic vascular resistance ratio are likely to have luxuriant pulmonary blood flow, they are ventilated with a reduced FiO₂ (PaO₂ 100 mmHg) and increased PaCO₂ (50 mmHg). For those with a high pulmonary vascular resistance to systemic vascular resistance ratio it is necessary to hyperventilate them with a higher FiO₂ (PaO₂ 300 mmHg) and a decreased PaCO₂ (25 mmHg).

PERFUSION MANAGEMENT

Currently, there are no established guidelines for the site of cerebral perfusion, flow temperatures, or perfusion pressure. Studies and physiological parameters will help to establish such guidelines (Table 2).

Table 1. Perfusion guidelines for antegrade cerebral perfusion in neonates (24).

Perfusion flow rate:	30 mL/kg/min
Perfusion pressure:	30–80 mmHg
Blood temperature:	22°C
Hematocrit:	22%
Colloid osmotic pressure:	10 mmHg
Alpha-stat blood gas management	

Table 2. Cerebral physiologic parameters in normothermia (15).

Global cerebral blood flow	5–30 mL/100g/min
CMRO ₂	3.5 mL/100g/min
Jugular venous saturation	>60%
Jugular venous PaO ₂	>35%

Cerebral Metabolism

For cerebral ischemia to occur, CBF must be reduced below CMRO₂ either globally or locally; therefore, an understanding of the effect of CPB on CMRO₂ is necessary. During CPB, hypothermia usually constitutes the dominant effect on CMRO₂, although CPB per se also has an effect on CMRO₂ independent of temperature. In clinical (6) and experimental (7) studies, brain metabolism is reduced by 35–50% during normothermic CPB. Why CMRO₂ decrease on CPB is unknown, but the phenomenon may represent depressed neural activity (8) or inadequate capillary perfusion as a consequence of cerebral microembolization (7).

Temperature

Temperature is a major factor directly affecting metabolism by reducing CMRO₂ approximately 7%/°C reduction in temperature (9,10). Hypothermia reduces both the electrophysiologic (60%) and cellular homeostatic (40%) components of brain energy expenditure. Hypothermia protects the brain during ischemia by diminishing high-energy phosphate depletion (11) and inhibiting excitatory neurotransmitter release (12), thereby limiting the extent of ischemia damage. With a temperature from 18–22°C cerebral vascular resistance increases greatly, and pressure–flow autoregulation is impaired, which may be secondary to cold-impaired cerebrovascular relaxation (6,13,14).

Carbon Dioxide Management

Cerebral vascular resistance is markedly affected by PaCO₂. Carbon dioxide diffuses rapidly across the blood–brain barrier, reducing extracellular fluid pH and causing cerebrovasodilatation. Elevating PaCO₂ levels may cause sufficient vasodilatation with a 4% increase in CBF per mmHg, to ablate the normal autoregulatory response to change in perfusion pressure (15). Blood temperature can profoundly alter carbon dioxide solubility, causing a reciprocal change in PaCO₂ of approximately 4.5%/°C. The use of hypothermia during antegrade cerebral perfusion, therefore, influences PaCO₂ and consequently, the cerebral blood flow. The PaCO₂ level during hypothermic antegrade cerebral perfusion is controlled using one of two techniques. The pH-stat and the alpha-stat modes differ in terms of whether PaCO₂ and pH are maintained at normal values at the actual temperature of the patient or at 37°C (of the blood gas analyzer). When the two techniques are

Table 3. Patient demographics.

	Number	Sex	Diagnosis	Age	Weight	Procedure
Group 1	Cases 17	Male 14	Hypoplastic left heart syndrome 13	3–80 days	2.8 ± 4.15 kg	Classic Norwood 14
		Female 3	Critical aortic stenosis 1 Single ventricle 3	15.4 ± 18.9 days	3.38 ± 0.43 kg	Modifies Norwood 3
Group 2	Cases 18	Male 11	Hypoplastic left heart syndrome 15	2–112 days	2.66–4.32 kg	Classic Norwood 16
		Female 7	Shone’s syndrome 1 Single ventricle 2	16.5 ± 26.8 days	3.25 ± 0.49 kg	Modifies Norwood 2

compared during hypothermic CPB at 28°C, a difference in PaCO₂ levels exists between patients managed with the alpha-stat and pH-stat technique. This gradient is sufficient to significantly alter CBF because cerebrovascular carbon dioxide responsiveness is maintained during non-pulsatile CPB (16). Under hypothermic conditions (22°C–28°C) pH-stat management results in pressure-dependent CBF with impaired cerebral autoregulation (6,17) and flow-metabolism coupling.

In contrast, alpha-stat management maintains pressure autoregulation as well as metabolic coupling during hypothermic CPB with greater carbon dioxide responsiveness (5,6). Using alpha-stat management, cerebral autoregulation and PaCO₂ responsiveness have been shown to be preserved 3–8 hours into the post-bypass period (18).

Hemodilution

Blood viscosity normally increases with hypothermia, causing reduced microcirculatory flow at a constant perfusion pressure. This effect may promote sludging of red cells and could cause of cerebral ischemia because of the no-reflow phenomenon.

Intracranial Pressure

Intracranial pressure (ICP) can directly affect the level of cerebral perfusion pressure (CPP) and possibly cerebral blood flow (CBF) (midaortic pressure—ICP = CCP). Intracranial pressure progressively increases in experimental animals during hypothermic and normothermic CPB (19,20). The cause of increased intracranial pressure is unknown, but it could represent the development of cerebral edema (21). However, studies show that the blood-brain barrier remains intact during hypothermic CPB and rewarming (22), and brain water content and intracranial

compliance after CPB are unaltered (20). Another mechanism of increased intracranial pressure could be dilation of cerebral venules secondary to loss of pulsatile flow (23). Experimental studies have found that increasing the tonicity of the pump priming solution significantly reduces the magnitude of intracranial pressure change (19).

PATIENTS AND METHODS

Operative Technique

Group 1 DHCA was used in all patients for systemic outflow tract reconstruction and excision of the arterial septum. DHCA period: 41–77 min (mean 62.05 ± 9.9 min).

Group 2 Hypothermic circulatory arrest was restricted to excision of the atrial septum. The systemic outflow tract reconstruction was done in antegrade cerebral perfusion via a modified BT-Shunt at a flow rate of 30 mL/kg/min. The HCA period was 0–9 min (mean 3.7 ± 2.7 min). The period of antegrade cerebral perfusion was 34–79 min (mean 53 ± 12.5 min).

RESULTS

DISCUSSION

From 1997 until 2001, 35 unselected consecutive patients underwent a Norwood procedure in our unit. Twenty-eight patients had a hypoplastic left heart syndrome, one patient had Shone’s syndrome with critical mitral valve stenosis, one patient had a critical aortic valve stenosis with hypoplastic left ventricle, and five patients had a single ventricle with L-transposition of the great arteries and systemic outflow tract stenosis. According to a change in our operative technique for systemic outflow

Table 4. Operative variables.

	Group 1	Group 2	P
Survival Stage 1	10 (59%)	14 (78%)	0.053 Binominal test
Anatomic lesions	0	0	
Neurologic event	1 Discrete hemiparesis	0	
CPB time mean ± SD	153–288 min 201.5 ± 39.3 min	150–297 min 193.3 ± 33.1 min	0.257 (t-test)
Serum lactate levels	6.8 ± 4.2 mmol/L	5.9 ± 2.7 mmol/L	0.56 (t-test)
ICU-Stay			
Mean ± SD	7.15 d 11.7 ± 2.83 d	6–49 d 17.2 ± 10.28 d	0.048 (t-test)
Total hospital stay			
Mean ± SD	21 ± 43 d 30.7 ± 6.5 d	15–76 d 33 ± 14.5 d	0.31 (t-test)

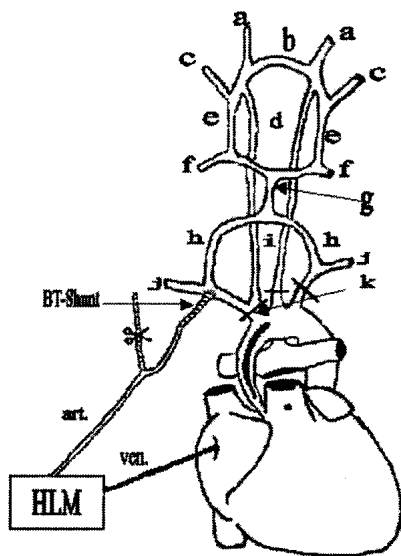


Figure 1. Antegrade cerebral perfusion by a BT-Shunt via the circle of Willis: (a) A.cerebri ant.dext/sin; (b) A.communicans ant; (c) A.cerebri media dext/sin; (d) A.carotis interna dext/sin; (e) A.communicans ant. dext/sin; (f) A.cerebri post.dext/sin; (g) A.basilaris; (h) A.vertebralis dext/sin; (i) A.carotis com.dext/sin; (j) A.subclavia dext/sin; (k) Truncus brachiocephalicus.

tract reconstruction from DHCA toward antegrade cerebral perfusion in 1999, two groups of patients resulted (Table 3).

Perfusion via a modified BT-shunt under exclusion of the aortic arch provides continuous oxygen supply to the supra-aortic region and, via collaterals, even to the lower part of the body. Flow studies demonstrated a significant amount of blood returning to the heart via the inferior vena cava. Because of the risk of cerebral hyperperfusion, we use pressure control and do not exceed a flow rate of 30 mL/kg/min. The observation that no neurological abnormalities occurred in our small series supports the utility of this procedure to avoid DHCA. During antegrade cerebral perfusion, the subaortic vessels and the descending aorta must be occluded to avoid return of blood into the operative field. Nevertheless, total bypass time under antegrade cerebral perfusion was not significantly different from bypass time under DHCA. The assumption that serum lactate levels should decrease significantly in Group 2 because of continuous perfusion could not be proved. We partially ascribe the significant improvement in survival in Group 2 to the minimization of DHCA. The longer ICU period in Group 2 can be explained by the extraordinarily long stay of the patient with Shonés syndrome attributable to her pulmonary vascular disease (Table 4).

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