

Low Flow with Deep Hypothermia

Ronald J. Slauch, C.C.P.

Primary Children's Medical Center
Western Cardiovascular Associates, Inc.
Salt Lake City, Utah

Most congenital cardiac defects are associated with marked bronchial arterial collateral circulation which usually obstructs the vision of the surgeon. The operative field is also very small and the defects are usually difficult to repair. In order to provide the surgeon with the best possible working conditions, we must have a dry, motionless operative field. We can obtain this by using either low flow and deep hypothermia with localized arrest or profound hypothermia with total circulatory arrest. Our present low flow technique leaves us the option of using circulatory arrest if needed. We occasionally use circulatory arrest on patients weighing less than 12 kg (Fig. 1) if the anticipated bypass time is longer than usual. The difficulty of the repair is usually the determining factor. Patients weighing 8–12 kg with extremely complex defects which would require more than 1½ hours of bypass time to repair are considered for total circulatory arrest in order to limit the bypass time to a maximum of 1½ hours. We try to limit the bypass time to one hour in patients weighing less than 8 kg. The other factors which determine whether or not we use circulatory arrest or low flow are controlling collateral circulation and the type of defect involved. The type of defect may involve approaching from the right atrium. In these cases the surgeon's vision and working space are sometimes obstructed by the venous cannulae. The venous cannulae, of course, may be completely removed during periods of circulatory arrest.

Kirklin, et al.,¹ described what he considers to be safe periods of circulatory arrest at given temperatures (Fig. 2). We also use these parameters for low flow; however, we feel safe in extending these time periods when necessary.

Most people will agree that there is a certain amount of blood trauma associated with cardiopulmonary bypass. We feel that a portion of this damage to the blood is directly related to the flow rate. Use of a low perfusion flow rate should decrease the damage. We also feel the possibility exists that higher flow rates contribute to postoperative pulmonary edema, although there are other factors which affect pulmonary function such as alteration of oncotic pressure due to different perfusate composition and increased pulmonary vascular resistance as a result of hypothermia. It is our belief that low flow is safer than total circulatory arrest as far as postoperative cerebral and renal function are concerned, even though there is no firm data to confirm that statement.²

How does hypothermia enable us to use low flow or circulatory arrest? The most obvious answer to that question is that hypothermia causes a significant reduction in O₂ consumption which is directly related to the magnitude of the decrease in temperature. Early studies indicated that the fall in O₂ consumption was linearly related to the decrease in temperature. Later studies have shown that the relationship between O₂ consumption and hypothermia is exponential³ (Fig. 3). In 1961, Fairley and associates found that O₂ consumption decreased approximately 50% at 28°C and about 78% at 18°C. As blood is cooled the oxyhemoglobin dissociation curve shifts to the left which means the hemo-

<u>CIRCULATORY ARREST</u>	<u>LOW FLOW</u>
1. 8-12 Kg A. ANTICIPATED BYPASS TIME LONGER THAN 1½ HOURS	1. 8-12 Kg A. ANTICIPATED BYPASS TIME LESS THAN 1½ HOURS
2. < 8 Kg A. ANTICIPATED BYPASS TIME LONGER THAN 1 HOUR	2. < 8 Kg A. ANTICIPATED BYPASS TIME LESS THAN 1 HOUR
3. IF COLLATERAL CIRCULATION IS NOT CONTROLLED WITH LOW FLOW	3. IF OPERATIVE FIELD IS DRY ENOUGH
4. IF VENOUS CANNULAE ARE OBSTRUCTING SURGEONS VISION AND/OR WORKING SPACE	4. IF VENOUS CANNULAE DO NOT NEED TO BE REMOVED

Figure 1. Parameters for determining the use of low flow or total circulatory arrest.

globin affinity for O₂ is increased, resulting in hemoglobin less willing to give up O₂ to the tissues. However, this effect is counterbalanced by the increased solubility of O₂ in plasma and by the decreased tissue-O₂ demand (Fig. 4).

Metabolic acidosis usually develops during hypothermia. Possible contributing factors include poor peripheral perfusion, unequal cooling, a discrepancy between O₂ availability and tissue requirements, and an increased production of lactate and other organic acids. Hypothermia also decreases the metabolic rate resulting in decreased production of CO₂ which also shifts the curve to the left. The addition of CO₂ decreases the pH, shifting the curve toward the right and increasing O₂ availability.

Surface induced hypothermia in combination with controlled ventilation can cause a decrease in serum potassium. Respiratory alkalosis has also been known to cause a decrease in serum potassium. There are conflicting reports on the hypothermic affects on serum calcium levels. Some reports indicate increases in total serum calcium levels. Since we have abandoned the use of surface induced hypothermia we no longer experience

**SAFE DURATION OF
TOTAL CIRCULATORY ARREST**

TEMPERATURE (NASOPHARANGEAL)	DURATION (MINUTES)	DECREASE IN O ₂ CONSUMPTION (APPROX.)	
28°C	20		
26°C	30	28°C	50%
22°C	45	18°C	78%

Kirklin, et al., 1973

Fairly, H.B., 1961

Figure 2. Safe periods of circulatory arrest (which we feel safe in extending) when using low flow.

Figure 3. Decrease in O₂ consumption with decreased temperatures.

AMOUNT OF DISSOLVED O₂

0.3 VOL.%	AT	38°C
1.5 VOL.%	AT	30°C
2 VOL.%	AT	25°C
2.5 VOL.%	AT	10°C

Figure 4. Increase of dissolved O₂ in plasma with decreased temperatures.

as many problems with cardiac irritability or arrhythmias before bypass which we attributed to electrolyte imbalances as well as certain anesthetic drugs. Any electrolyte imbalances which we experience now are usually related to the prime in the extra-corporeal circuit.

ORGAN PROTECTION

Of course, the main reason we use hypothermia during low flow or circulatory arrest is to protect the vital organs and tissues which, without hypothermia, would not receive adequate gas transfer. The two most important organs are the brain and the heart. Since we are operating on the heart and the aorta is cross-clamped, it is necessary to have some type of myocardial protection whether or not hypothermia or low flow is used.

THE BRAIN

Hypothermia reduces cerebral O₂ consumption. This enables us to use circulatory arrest or low flow, which most people believe necessary, in order to repair difficult congenital cardiac defects. Belsey and associates⁴ suggested that much of the cerebral damage seen in patients undergoing surgery utilizing profound hypothermia and circulatory arrest was secondary to low cerebral CO₂ tensions which resulted in vasoconstriction. Therefore, it is our belief that the use of deep hypothermia and low flow, which still provides a dry, motionless operative field, we can perfuse the brain with a normal or close to normal pCO₂. It also allows us to perfuse (limited though it may be) other vital organs such as the kidneys and liver.

TECHNIQUE

In preparation of the patient in the operating room we try to maintain normal body temperature. Since we do not use surface cooling, we have no desire for the temperature to drop until we are ready to go on bypass. Arterial and venous pressures are monitored at all times as well as the patient's temperature. We have a slave scope as well as a master so everyone in the room can see all temperatures and pressures at all times.

Inhalation anesthetics are most often used in induction; however, the type of cardiac defect should be considered to determine the most effective method. For example, an IV injection would be more effective with a right to left shunt, and an inhalation anesthetic

would act quicker with a left to right shunt because of the blood flow pattern. Halothane is quite popular because it is easily controlled and has few side effects. It also induces vasodilatation which is extremely helpful in the cooling and rewarming phases. It does, however, have a cardiac depressant effect.

PRIME

We use different degrees of hemodilution in our prime. Due to increased viscosity of blood at cooler temperatures, we dilute the patient to a hematocrit close to the temperature we are taking the patient. We never use a crystalloid solution in the prime when operating on infants because of oncotic pressure alteration. We do, however, use a balanced electrolyte solution in the prime for larger children and adults. To achieve a particular degree of hemodilution for patients under 10 kg we add fresh frozen plasma to the blood in hopes of restoring otherwise depleted coagulation factors. Fresh blood should be used to avoid electrolyte or acid base imbalances.

The circuit is primed with a recirculation line in the event that circulatory arrest is needed. The recirculation line allows us to adjust the hematocrit or the temperature of the prime while the venous and arterial cannulae are clamped and/or removed during the circulatory arrest period.

We are firm believers in arterial line filtration when using bubble oxygenators, especially with radical temperature changes in the perfusate. We incorporate a bypass line around the filter in the event of problems developing with the filter. However, we have never experienced any apparent problems with an arterial line filter.

On bypass, we immediately begin to cool. The heater-cooler allows extreme temperature ranges, yet it is easily controlled, maintaining safe gradients between water, perfusate, and patient temperatures. We monitor nasopharyngeal, rectal and perfusate, as well as the water temperatures. Myocardial temperature is also monitored during cross-clamping.

We cool as quickly as possible, but in hopes of attaining more even cooling, we maintain cold perfusion for 12–15 minutes; longer if it is needed to reach a nasopharyngeal temperature of at least 20°C. The perfusate temperature is stabilized at 20°C after the cross-clamp is placed on the aorta. At the time of the cross-clamp, a cold cardioplegic myocardial preservation solution is infused into the aortic root. We infuse 10 cc/kg which usually brings the temperature of the myocardium down to less than 15°C. At this time the perfusion flow is reduced to provide the surgeon with a bloodless operative field.

Blood gases are routinely drawn every 15 minutes. Venous gases are drawn at the beginning of the cooling phase and during the rewarming phase as an indication of adequate perfusion. Low flow with poor peripheral perfusion can cause metabolic acidosis and we make no attempt to correct this acidotic state until the rewarming phase has begun. Our main concern is to keep the arterial pO₂ and pCO₂ within normal limits. During the cross-clamp, cold cardioplegic solution is re-infused at 20-minute intervals, or sooner if the myocardial temperature rises above 20°C.

When repair of the defect nears completion the rewarming phase begins. Just before the cross-clamp is removed room temperature cardioplegic solution is infused in order to bring the temperature of the myocardium closer to that of the perfusate. Mannitol is also given at this time in hopes of preventing post-ischemic subendocardial edema. We

SUMMARY

1. DRY, MOTIONLESS OPERATIVE FIELD
2. HYPOTHERMIA
3. TECHNIQUE:
 - MYOCARDIAL PROTECTION
 - PRIME/PERFUSATE
 - HEMATOCRIT
 - ACID BASE
 - ELECTROLYTES
 - COOLING/WARMING
 - SAFE TEMPERATURE GRADIENTS
 - SAFE PRESSURE GRADIENTS
4. OFF BYPASS:
 - NORMOTHERMIA
 - NORMAL HcT.
 - NORMAL ACID BASE/ELECTROLYTES

Figure 5. Summary.

try to warm the patient as quickly as possible, yet maintaining safe gradients between water, perfusate and patient temperatures. As the patient rewarms, the flow is increased, sometimes as high as $3.2 \text{ l/m}^2/\text{min}$. However, we do not allow the arterial line pressure to rise above 300 mmHg.

By the time the patient reaches a temperature of 32°C the hematocrit should be between 30 and 35%. This is achieved by draining some of the perfusate from the circuit and replacing it with packed cells until we have obtained the hematocrit desired. This is easily accomplished, when total circulatory arrest is used, by completely draining the extracorporeal circuit while the pump is stopped and recirculating a new prime to obtain the desired hematocrit. One disadvantage of this technique, in my opinion, is that when bypass is reinstated you must perfuse a cold patient with a high viscosity perfusate which will restrict the flow thus impeding warming time.

Our next concern is to correct any acid base or electrolyte imbalances. We very rarely experience electrolyte imbalances. However, we often find the need to correct certain degrees of metabolic acidosis. Respiratory alkalosis, sometimes associated with hypothermia, usually corrects itself by the time the patient reaches normothermia.

At the completion of bypass, we try to have the patient's temperature above 34°C , the hematocrit above 30%, and any acid base or electrolyte imbalances corrected. The left atrial pressure is monitored along with the systemic while coming off bypass to prevent over filling of the patient. Inotropic or chronotropic drugs are given when needed, and if urine output has been inadequate a diuretic is given.

SUMMARY

Hypothermia, which decreases tissue O_2 consumption, enables us to use low flow and/or total circulatory arrest. By using low flow or circulatory arrest, the surgeon is

provided with much better exposure and working conditions, and the patient is protected from the dangers of high flow cardiopulmonary bypass (Fig. 5).

The proper priming solution used for pediatric perfusion is a very important consideration. Electrolyte and acid base imbalances as well as oncotic pressure regulation and viscosity should be carefully observed.

During the procedure all pressures and temperatures should be monitored and safe gradients should be adhered to. At termination of bypass, the perfusionist should strive for normal acid base and electrolytes, a normal hematocrit and a normal body temperature.

REFERENCES

1. Kirklin, J. W., et al., Cardiopulmonary Bypass for Cardiac Surgery. *Surgery of the Chest*, 3rd Ed., Ch. 29: 846-66, 1976.
2. *Ibid.*
3. Fairley, H. B. Metabolism in Hypothermia. *Brit. Med. Bull.* 17: 52, 1961.
4. Belsey, R. H. R., et al., Profound Hypothermia in Cardiac Surgery. *J. Thorac. Cardiovasc. Surg.* 56: 497, 1968.