PROFOUND HYPOTHERMIA—TECHNICAL ASPECTS

By Brian Barratt-Boyes

We became interested in the use of profound hypothermia with circulatory arrest for the correction of congenital heart defects in infancy because the results using extracorporeal circulation were unsatisfactory. The chief reason for this, in our experience, was poor surgical exposure which led far too often to surgical error or, at best, to prolongation of the perfusion time beyond acceptable limits in the infant group. Surgical exposure was poor when working inside the posteriorly positioned atria because of the relatively bulky semi-rigid double caval cannulae and excessive intracardiac blood, despite aortic crossclamping; and in the ventricles because of incomplete myocardial relaxation. A technique which would provide a completely relaxed empty heart in which to perform the intracardiac repair under conditions of total circulatory arrest, seemed the only satisfactory answer to this problem. To achieve these ends, the method of temperature manipulation used effectively by the Kyoto group was chosen because of its theoretical advantages. Over the three years since the introduction of this technique at Green Lane Hospital, in July 1969, we have been so satisfied with the ease of surgical exposure and in the results achieved in 140 infants in the first two years of life, that we now undertake intracardiac repair in all infants with potentially correctable lesions who demand relief, regardless of size. Palliative surgery is used only in uncorrectable lesions. The conditions that have been treated in this way are listed in the Table in order of frequency.

**TABLE 1**

<table>
<thead>
<tr>
<th>PROFOUND HYPOTHERMIA IN INFANCY</th>
<th>ACYANOTIC</th>
<th>CYANOTIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricular septal defect</td>
<td>Simple transposition</td>
<td></td>
</tr>
<tr>
<td>Partial AV canal</td>
<td>Tetralogy of Fallot</td>
<td></td>
</tr>
<tr>
<td>Truncus arteriosus</td>
<td>Complex transposition*</td>
<td></td>
</tr>
<tr>
<td>Aortic stenosis*</td>
<td>Total anomalous</td>
<td></td>
</tr>
<tr>
<td>Pulmonary stenosis</td>
<td>pulmonary venous</td>
<td></td>
</tr>
<tr>
<td>Total AV canal</td>
<td>connection</td>
<td></td>
</tr>
<tr>
<td>Atrial septal defect</td>
<td></td>
<td></td>
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<tr>
<td>Mitral stenosis</td>
<td></td>
<td></td>
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<tr>
<td>Anomalous coronary artery*</td>
<td></td>
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</tbody>
</table>

**TECHNIQUE**

Premedication consists of 0.1 ml/kg bodyweight of a mixture of pethidine 25 mg, chlorpromazine 6.25 mg and promethazine 6.25 mg/ml. Anaesthesia is induced by mask, using equal parts of nitrous oxide and oxygen plus 0.5 % halothane. Intravenous d-tubocurarine (1 mg/kg) is given and followed by insertion of an orotracheal airway and nasopharyngeal and rectal temperature probes. Ventilation is continued with nitrous oxide and oxygen and 0.5% halothane. Carbon dioxide (2.5%) is added to anesthetic and oxygenator gasses at temperatures below 30°C.

The infant is placed on a cooling blanket and covered with ice bags. Pressure monitoring lines are sited in the abdominal aorta and inferior vena cava via a left groin incision. The ice bags are removed at a nasopharyngeal temperature of 24°C to 25°C and a sternal splitting incision is made.

The child is heparinised (3 mg/kg) after taping both cavae and a single venous cannula is inserted into the right atrial appendage followed by a 10F to 12F catheter into the ascending aorta for arterial return. The simplified infant perfusion circuit to which these lines are connected (Fig. 1) consists of a 9" Kay Cross disc oxygenator with a filter on the arterial line, a single pump for arterial return and an efficient heat exchanger. Arterial line temperature and pressure are both monitored. There is no venous return reservoir. A defoaming chamber of open-heart return pump, as the small amount of open-heart blood is discarded. The circuit is primed with 1L of fresh heparinised whole blood, drawn on the morning of operation, to which 2 mEq of potassium chloride and 5 mEq of sodium bicarbonate are added. Phenindamine is therefore not used, to avoid the risk of additional fluid load, although each 450 ml of blood contains 50 ml of 5% glucose as a base for the heparin.

When cannulation has been completed the temperature may have fallen to 22°C and under these circumstances, in simple cyanotic conditions, such as aortic stenosis, pulmonary stenosis, aortic septal defect and ventricular septal defect, cooling bypass is not instituted, but blood is drained through the venous line into the machine to provide a dry heart. The aorta and cavae are then occluded and the intracardiac repair begun. In all cyanotic and complex conditions, however, a short 5 to 10 minute period of total body perfusion is required, prior to exanguination to fully oxygenate the infant and to lower the temperature further. The perfusion flow rate is 100ml/kg/min. and the temperature difference between nasopharynx and main arterial blood does not exceed 8°C. The safe circulatory arrest time appears to be 60 minutes at 22°C nasopharyngeal and 70 to 75 minutes at 18°C. In some complex conditions it may not be possible to complete the repair within the permissible circulatory arrest time and a further short period of cold arrest is then added to reoxygenate the tissues. If this is done, either both cavae must be cannulated, or the cardiotomy incision closed and air evacuated from the heart. An example where two additional periods of cold perfusion were required is depicted in Fig. 2.

After the completion of repair, the heart is closed and air is flushed out by syringing isotonic saline solution through a fine catheter introduced into the apex of the left ventricle. As the aortic crossclamp has been applied above (distal to) the arterial cannula, this solution is conveniently aspirated through a connector in the arterial line and the heart is gently massaged to exclude all the air during this manoeuvre. The pulmonary artery is also needed for air. In patients who have had an atrial baffle repair for transposition, the air is flushed from the systematic right ventricle via the right atrial appendage and the venous return line is moved to the left atrial appendage for the rewarming perfusion.

The infant is now rapidly rewarmed by a further 20 to 30 minute period of total body perfusion at a flow rate of 100 to 120 ml/kg/min. A further 2 mEq of potassium and 10 mEq of sodium bicarbonate are now added and the temperature of the machine blood is rapidly raised to 30°C and within a few minutes to 37°C. The heart usually begins beating almost immediately, but if not it is electrically defibrillated. The central venous pressure is maintained at 5 to 10 mm. Hg to allow the vigorous beating heart to contract against the normal arterial pulse (Fig. 3). Perfusion is stopped at 35°C and rewarmed completed with the water blanket during wound toilet and closure. A pressure mon-
Fig. 1. Photograph of perfusion circuit used in profound hypothermia. The heat exchanger is mounted vertically on the right of the machine.


Fig. 3. The aortic pulse contour recorded 4 minutes after commencing rewarming bypass at a flow rate of 100 ml/kg/min. The central venous pressure (CVP) at this time was 8 mm. Hg. From Barratt-Boytes, B. G., Neutze, J. M., Seely, E. R. and Simpson, M. Progress Cardiovasc. Dis. 1972 (in press). Reproduced with permission.

Fig. 4. Temperature graph during operation in an infant with transposition. c = cooling bypass; w = warming bypass. From Barratt-Boytes, B. G., Simpson, M. and Neutze, J. M. Circulation Suppl. 1, 43, 25, 1971. Reproduced with permission.

Fig. 5. Graph showing average changes in temperature (upper = nasopharyngeal, lower = rectal) heart rate and blood pressure during profound hypothermia with surface cooling followed by a 5 minute period of cooling bypass (BP), a one hour circulatory arrest period and a 20 minute period of rewarming bypass. From Barratt-Boytes, B. G., Neutze, J. M., Seely, E. R. and Simpson, M. Progress Cardiovasc. Dis. 1972 (in press). Reproduced with permission.
itoring catheter is placed in the left atrium and temporary pacemaker wires on to the right ventricle. The pericardium is not sutured. At the conclusion, the tubocurarine is reversed with an appropriate intravenous dose of a mixture of atropine 0.15 mg and neostigmine 0.3 mg.

The sequence described is shown in graphic form in Fig. 4 and Fig. 5 depicts the average changes in nasopharyngeal and rectal temperatures, blood pressure and pulse rate.

COMMENT

In addition to the technique described, there are two other methods of temperature manipulation available, namely, cooling and rewarming entirely by surface means or cooling and rewarming entirely by total-body perfusion. The former technique eliminates the hazards of perfusion and heparinisation but is time-consuming. It has the major disadvantage that recovery of myocardial function during surface rewarming is slow and cardiac massage is required and may damage the myocardium. Both as a result of poor cardiac output and slow rewarming of the liver, metabolic acidosis is greater than with other techniques. Core cooling and rewarming is attractive because it conserves total anaesthetic time, but it prolongs the perfusion time for at least one hour. Moreover, body cooling is uneven and oxygen consumption at the time of arrest is twice as high as with surface cooling. The circulatory arrest time is therefore probably significantly shorter at any given temperature than when surface cooling is used. Core cooling is unwise when there is any aortic incompetence present, as the left ventricle rapidly becomes ineffective.

There are a number of theoretical advantages in combining surface cooling and core rewarming. With surface means, the muscle mass is evenly and effectively cooled, while all the core organs, including the heart and liver, maintain effective function for a longer time. This process is reversed with core rewarming, as the heart and liver warm faster than the periphery and both cardiac output and lactate metabolism improve rapidly. The duration of bypass is not, of course, related to the complexity of the lesion and true pulsatile flow is maintained by the beating heart.

The fear that circulatory arrest at low temperatures will be associated with brain damage in these infants has not been supported by our experience,8,9 or that of others.8,9 Only one infant has suffered brain damage attributable either to the duration of circulatory arrest or air embolisation and in this instance the arrest time was prolonged beyond the safe limit (70 minutes at 22°C). In this eight-month-old child the damage was minor and progress now appears to be normal.

SUMMARY

A technique employing surface cooling followed by circulatory arrest and a short period of core rewarming is described and recommended for use in infants requiring repair of congenital heart defects. This technique provides ideal operating conditions and enables intracardiac repair to be undertaken safely, even in complex conditions, in the early months of life. Alternative methods of temperature manipulation are briefly described and discussed.

REFERENCES