

ANAESTHESIA WITH PROFOUND HYPOTHERMIA IN A CASE OF TRANSPOSITION OF THE GREAT VESSELS WITH RENAL FAILURE

K.W. TURNBULL, B.A.SC., M.D., F.R.C.P.(C), W.A. DODDS, M.D., F.R.C.P.(C),
AND G.R. AVERY, M.B.

THE USE OF PROFOUND HYPOTHERMIA for intra-cardiac surgery in infants and neonates has resulted in improved surgical conditions¹ and decreased paediatric mortality. Many of the candidates for profound hypothermia are in the high-risk categories. This report concerns a four-month-old girl weighing 5 kg who had pre-operative renal failure requiring peritoneal dialysis.

The cardiac output of this infant had progressively fallen. The increased demands of growth precipitated heart failure in a heart already compromised by transposition of the great vessels. A previous balloon septostomy (at age two weeks) had resulted in short-term improvement. Therapeutic manoeuvres including digitalis, diuretics and supplementary oxygen did not help. A repeat heart catheterization showed equal atrial pressures and eliminated any chance of improvement from further septostomies.

Increasingly severe renal failure as indicated by anuria, hyperkalaemia, urea retention and increasing creatinine levels resulted from the inadequate renal perfusion. Surgical correction of the lesion was considered the only possible way to improve tissue perfusion. Pre-existing severe renal failure suggested profound hypothermia might be contra-indicated as the exsanguination technique may cause renal tubular damage.² On the other hand Moyer³ and Collins⁴ suggest that hypothermia affords moderate protection against severe renal damage due to ischaemia. Despite this and the risk of the postoperative low cardiac output syndrome,¹ the decision was made to proceed with the operation.

ANAESTHETIC MANAGEMENT

Pre-operative preparation focused on proper fluid, electrolyte and caloric balance. Peritoneal dialysis was used to modify the hyperkalaemia. The serum potassium was reduced from 6.7 meq/L to 4.4 meq/L with a normal pH.

No premedication was used. Induction was with oxygen-halothane. The initial halothane concentration of 0.5 per cent was increased gradually to 1.25 per cent as tolerated. Oro-tracheal intubation was performed during spontaneous respiration. Controlled ventilation was then established using nitrous oxide-oxygen (5:3), halothane (0.75–1.0 per cent) and d-tubocurarine. The Jackson-Rees modification of the Ayre's T-piece was employed. The electrocardiogram was monitored; arterial and venous catheters were inserted for sampling and pressure monitoring. Temperature was measured in both oesophagus and rectum and recorded.

The infant was cooled on a cooling blanket. The body surface was covered with

From the Department of Anaesthesia, the Vancouver General Hospital and the University of British Columbia.

plastic bags filled with melting crushed ice. Five per cent CO₂ was added during the surface cooling when oesophageal temperature was 34° C, and cooling was stopped at an oesophageal temperature of 26° C (rectal temperature 27° C). The addition of CO₂ resulted in acceptable carbon dioxide tensions (P CO₂ 35.0–40.0 mm Hg).¹ The chest was opened and further cooling commenced on full cardiopulmonary bypass after placement of the cardiac catheters. A haemodilution technique similar to that of Johnston *et al.*⁵ was used resulting in a haematocrit of 22.5 per cent. Bypass was stopped when the oesophageal temperature had fallen to 15.5° C (rectal temperature 21° C). Total exsanguination into the bypass pump oxygenator was then carried out. Circulation was arrested for one hour and twenty minutes during which time a Mustard procedure was done. PEEP of 5–7 cm H₂O was maintained, using 50:50 nitrous oxide and oxygen. Resumption of perfusion and weaning from bypass were assisted by 100 mg of calcium chloride. Cardiac dysrhythmias (2:1 block and A-V dissociation) cleared as the infant was rewarmed. The oro-tracheal tube was left in place. On arrival in the post-anaesthetic room the patient was reacting to painful stimuli, had an oesophageal temperature of 32° C (rectal 27° C) and tissue perfusion and peripheral cyanosis improved as the temperature returned to normal.

POSTOPERATIVE CARE

Elective controlled ventilation was maintained using a volume ventilator (Bennett MA-1). Despite one episode of bradycardia and hypercarbia she was successfully weaned from the respirator six days postoperatively.

Peritoneal dialysis was continued daily to control hyperkalaemia while urine output and urine osmolality gradually increased. Urine output and renal function were acceptable by the seventh postoperative day. Her general condition improved markedly, congestive heart failure disappeared and she was discharged from the hospital.

CONCLUSION

Any low perfusion state may precipitate renal failure. The response of this infant to surgical improvement of cardiac performance and the resultant improved tissue perfusion suggest pre-existing renal failure is not a contra-indication to hypothermia when hypoperfusion is present. Further research and clinical experience should help to clarify the significance of preoperative renal failure in candidates for profound hypothermia. It is hoped that the success of this case will encourage others to use similar techniques to save infants with congenital heart defects in the presence of renal failure.

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REFERENCES

1. STEWARD, D.J., JOHNSTON, A.E., & SLOAN, I.A. Anaesthetic management of infants undergoing profound hypothermia for surgical correction of congenital heart defects. *Can. Anaesth. Soc. J.* 21: 15-22 (1974).
2. MOYER, J.H., MORRIS, G., & DEBUKEY, M.E. Hypothermia, I. Effect on renal hemodynamics and on excretion of water and electrolytes in dog and man. *Ann. Surg.* 145: 26 (1957).
3. MOYER, J.H., HEIDER, C., MORRIS, G.C., & HANDLEY, C. Hypothermia III. The effect of hypothermia on renal damage resulting from ischemia. *Ann. Surg.* 146: 152 (1957).
4. COLLINS, V.J. Principles of anesthesiology, 2nd ed., Lea & Febiger, Philadelphia, p. 611 (1966).
5. JOHNSTON, A.E., RADDE, I.C., STEWARD, D.J., & TAYLOR, J. Acid-base and electrolyte changes in infants undergoing profound hypothermia for surgical correction of congenital heart defects. *Can. Anaesth. Soc. J.* 21: 23-45 (1974).