

POST-OPERATIVE MANAGEMENT AND COMPLICATIONS FOLLOWING PROFOUND HYPOTHERMIC SURGERY

By J. M. Neutze, Marie M. Simpson, Eve R. Seelye and B. G. Barratt-Boyes

The development of surgical techniques for the correction of congenital cardiac lesions in infancy has introduced new problems in post-operative care. The major change in surgical practice has been the introduction of the use of profound hypothermia in the infant¹⁻⁴ but there have been no particular post-operative complications resulting directly from this technique. Rather, the problems are those of management of small, sick infants who have undergone major surgery to the heart. As most difficulties occur in infants undergoing surgery in the first year of life, we have recently reviewed the management of 82 infants under one year of age, operated on at Green Lane Hospital up to December 1971,⁵ and this paper is largely based on that experience. Full surgical details have been published elsewhere³ and the present discussion is restricted to post-operative management in the intensive care unit.

Four of the infants were less than one week of age, 38 were aged 1 week to 3 months and 40 were between 3 and 12 months. Diagnoses are shown in Table I. Post-operative care is primarily concerned with replacement of blood loss, administration of appropriate fluids and electrolytes and the provision of circulatory and respiratory support.

BLOOD LOSS

Although 3 infants lost about 70 ml. of blood/kg. body weight in the first 24 hours, losses in most cases were between 20-25 ml./kg./24 hours. When this was replaced with whole blood, post-operative haemoglobin levels were sometimes as high as 18 g/100 ml. This was probably related to the use of undiluted, fresh, heparinized blood in the heart lung machine. Replacement in the intensive care unit is therefore now carried out with varying proportions of fresh, citrated blood and freeze-dried plasma. Where blood loss exceeds 100 ml. 50 mg. of calcium chloride are given. Although one patient was explored for suspected, but unconfirmed tamponade, no reoperations were carried out for bleeding. Unsuspected, localised tamponade caused the death of one patient with complex transposition of the great vessels (TGA) and a second case of tamponade was relieved by shortening of the intrapericardial drainage tube. In general, however, bleeding has not been a major problem.*

FLUIDS AND ELECTROLYTES

In the first 24 hours intravenous fluid was given at a rate of 2 ml/kg. body weight/hour, initially as N/5 saline with 4.2% dextrose but more recently as 5% dextrose in water. When higher infusion rates were used, the volume load frequently proved excessive. The 100 mg/kg/hour of glucose given with this regimen appears adequate to prevent hypoglycaemia. It is noteworthy that Hamilton, giving 500 mg./kg./hr. noted hypoglycaemia in some patients up to 5 days post-operatively.⁶ Baum⁷ has recorded high Insulin levels following hypothermia in infants infused with glucose at this rate and the higher infusion rate of glucose may actually increase the risk of hypoglycaemia as it is difficult to avoid some fluctuation in the rate of administration.

Urinary output proved difficult to measure accurately but recorded volumes were frequently low in the first 24 hours (Table II) although infants with an uncomplicated course usually passed 0.5-1 ml./kg./hr. In all surviving cases, urinary output increased in the second 24 hours and there was no evidence of the development of renal damage in the post-operative period. The urinary output of sodium and potassium showed wide variation (Table III) but in general, urinary losses of sodium were low and of potassium, moderate. Losses of urinary sodium and potassium were not clearly related to urinary volume.

Oral or naso-gastric fluids were usually started at 30-36 hours.

With the exception of patients judged clinically to have a low cardiac output, severe metabolic acidosis was not a feature, the base deficit reaching 5 mEq./litre in only 10% of those patients who had a straight forward post-operative course. However 31 of the 82 patients received bicarbonate in the first 24 hours. In 75% of cases serum potassium fell or remained at early post-operative levels. Potassium supplements were given to 42 patients in the first 24 hours, usually in the first 8-10 hours. The average requirement for patients who had received Digoxin and diuretics pre-operatively was 0.63 mEq./kg. and for those who had not received Digoxin and diuretics was 0.33 mEq./kg.

ARRHYTHMIAS

Significant arrhythmias occurred in 40 cases, some patients showing more than 1 type (Table IV). Thirteen of 26 patients with simple transposition of the great vessels (TGA) had nodal arrhythmias, 4 of 5 patients with endocardial cushion defect had nodal arrhythmias and 3 showed A-V dissociation. Most arrhythmias settled within a few hours. Pacing wires were inserted in 25 patients and used in 12 cases. As pacing might have been useful in 8 other patients, temporary ventricular pacing wires are now inserted routinely at operation.

MYOCARDIAL SUPPORT

Although cardiac output has not been measured routinely many infants were judged clinically to pass through a period with a low cardiac output. The basis of this diagnosis was the presence of at least 2 of the following signs; peripheral vasoconstriction, low urinary output, a rise in the base deficit and a rise in the serum potassium. Digoxin was given to 37 infants in the first 24 hours and to a further 12 in the second 24 hours. A diuretic was given to 8 patients on day 1 and 14 on day 2. Sixty-three patients received digitalis and diuretics pre-operatively and although Digoxin was stopped 36 to 48 hours before operation, we were reluctant to reintroduce the drug post-operatively until potassium levels were stabilised. For this reason the initial treatment for a "low output state" was often the infusion of Isoprenaline. This was rarely required in the treatment of infants with ventricular septal defect (VSD) or simple TGA but was required in over half the patients in other groups. Isoprenaline was given by a constant infusion pump to 36 patients for periods varying from a few hours to several days, the average maximal dose being 0.18 ug/kg/min. Three examples will illustrate management in this important area.

Case 1

G.F. had TGA, PDA and a large VSD and underwent balloon septostomy at 6 days of age. Despite intensive therapy with Digoxin and diuretics he declined rapidly with congestive heart failure and correction was attempted at 11 days. The procedure was uneventful although a small coronary branch was sacrificed with the ventriculotomy. The heart took over well but was paced at a rate of 120 because of the presence of a slow nodal rhythm. Intermittent positive pressure respiration (IPPR) was provided throughout the post-operative course. On return to the intensive care unit (Fig. 1) the blood pressure was satisfactorily maintained for 6 hours but the infant was peripherally constricted and no urine was produced. Even more disconcerting was a sharp rise in base deficit and serum potassium, only partly relieved by the administration of base, Digoxin and Isoprenaline. A total of 50 mEq. of base (given as bicarbonate and THAM) and increasing dosage of Isoprena-

TABLE I
DIAGNOSIS IN 82 PATIENTS

Endocardial cushion defect	-	-	-	-	5
Ventricular septal defect	-	-	-	-	18
Total anomalous pulmonary venous connection	-	-	-	-	10
Persistent truncus arteriosus	-	-	-	-	3
Pulmonary stenosis or atresia	-	-	-	-	7
Tetralogy of Fallot	-	-	-	-	12
Transposition of the great arteries (simple)	-	-	-	-	16
Transposition of the great arteries (complex)	-	-	-	-	10
Aortic stenosis	-	-	-	-	1
TOTAL					82

TABLE II
URINARY VOLUME POST-OPERATIVELY
(ml/kg. body weight/day)

	First 24 hours		Second 24 hours	
	No.	Volume	No.	Volume
All patients	40	18	21	31
"Normal Cardiac Output"	22	24 (10 - 68)	7	29 (20 - 46)
"Low Cardiac Output"	18	10 (0 - 32)	14	33 (0 - 73)

TABLE III
URINARY OUTPUT OF SODIUM AND POTASSIUM POST-OPERATIVELY

	First 24 hours		Second 24 hours	
	mEq/L urine	mEq/kg/day	mEq/L urine	mEq/kg/day
SODIUM	29 (5 - 93)	0.6 (0.03 - 1.7)	19 (8 - 50)	0.7 (0.1 - 1.7)
POTASSIUM	99 (15 - 186)	1.5 (0.1 - 4.0)	75 (21 - 136)	2.9 (0.2 - 5.2)

First 24 hours : 15 patients, average urinary volume 15 ml/kg/day
 Second 24 hours : 9 patients, average urinary volume 36 ml/kg/day

TABLE V
DEATHS IN THE INTENSIVE CARE UNIT

1. Respiratory	-	-	-	-	-	3
Iatrogenic pneumothorax (TAPVC)	-	-	-	-	-	1
Collapsed lung (Complex TGA)	-	-	-	-	-	1
Sub-glottic stenosis—late death (TAPVC)	-	-	-	-	-	1
2. Low Cardiac Output, Incompletely Corrected Lesions						5
P.S. or atresia with underdeveloped right ventricle	-	-	-	-	-	3
Complete AV canal with inadequate A-V valve	-	-	-	-	-	1
Aortic stenosis with rudimentary aortic valve	-	-	-	-	-	1
3. Low Cardiac Output, Treatment Satisfactory						5
TGA, extreme acidosis pre-operatively	-	-	-	-	-	1
Complex TGA	-	-	-	-	-	3
T/F with myocardial fibrosis	-	-	-	-	-	1
4. Low Cardiac Output, Treatment Inadequate						5
Sudden arrest, (AV canal, VSD + Co. Ao., TGA, complex TGA)	-	-	-	-	-	4
Tamponade (Complex TGA)	-	-	-	-	-	1

Subscript: TAPVC — Total anomalous pulmonary venous connection
 TGA — Transposition of the great arteries
 T/F — Tetralogy of Fallot
 VSD — Ventricular septal defect
 Co. Ao. — Coarctation of the aorta
 P.S. — Pulmonary stenosis

TABLE IV
POST-OPERATIVE ARRHYTHMIAS

Sinus bradycardia	6
Nodal arrhythmia	22 (6 slow)
A-V dissociation	11
Supraventricular tachycardia	7 (1 DC conversion)
Ventricular premature beats	3
Complete heart block	1

line to 2 ug/kg/min failed to improve the situation and the baby arrested and died at 12 hours.

Comment

Rising base deficit and serum potassium were associated with a declining clinical state. The development of a metabolic acidosis may be aggravated by the presence of hypoxia. Serum potassium levels usually fall post-operatively but, of course, this change is modified by the administration of potassium. When due allowance is made for these factors, rising serum potassium and base deficit, combined with low urinary volume, appear, in the absence of cardiac output measurements, the most reliable indicators of a low output state.

Case 2

C.B. had simple TGA and underwent septostomy at two days. Because of progressive hypoxia a baffle repair was carried out at 4 months. Initial progress was uneventful (Fig. 2) but urinary output averaged just under 0.5 ml/kg./hr. and at 17 hours there was a brief episode of cyanosis. At this time, the base deficit had risen to 12 mEq./litre and the serum potassium to 6 mEq./litre. With the administration of base, Isoprenaline and Digoxin, these values improved and urinary output increased to over 1 ml./kg./hr. Subsequent progress was uneventful.

Comment

Because of the apparent well being of this infant, recordings of serum potassium and blood gases were neglected and it is suspected that the cardiac output had been low for some time before measurements were made at 17 hours. Earlier in our experience, another infant with TGA who showed a similar clinical picture, arrested unexpectedly and died 26 hours post-operatively.

Case 3

J.M.* had a ventricular septal defect, patent ductus arteriosus and patent foramen ovale. Despite vigorous treatment with Digoxin and diuretics she remained in severe congestive heart failure and when surgery was undertaken at 2 months she was just over her birth weight. Post-operatively she was paced for 32 hours because of sinus bradycardia and ventilation was assisted either by continuous positive air way pressure (CPAP) or IPPR. Initial progress was uneventful (Fig. 3) although the infant was hypoxic, probably because of right to left shunting through the patent foramen ovale as well as through the lungs. Urine volume averaged just under 1 ml./kg./hr. but by 12 hours, the infant appeared peripherally constricted. Despite the administration of Isoprenaline, the base deficit rose to a peak of 8 mEq./litre and serum potassium to a peak of 5.8 mEq./litre. Phenoxybenzamine was therefore given (1 mg./kg. over 1 hour), Isoprenaline dosage was increased, bicarbonate was given and Digoxin was recommended. Following a further peak in serum potassium and base deficit, these values improved, signs of peripheral vasoconstriction disappeared and from 23 hours the urinary output increased to 6 ml./kg./hr. A second dose of Phenoxybenzamine was given at 40 hours. Further intravenous potassium was required from 40 hours, Isoprenaline dosage was tailed off from 44 hours and stopped at 72 hours and CPAP was discontinued at 69 hours. Further progress was uneventful.

Comment

In addition to the hypoxia it is suspected that this infant had a low cardiac output in the hours preceding intensification of therapy. The rise of serum potassium and base deficit following the administration of Phenoxybenzamine are compatible with improved perfusion of regions where the circulation was inadequate but the relative contributions of Phenoxybenzamine, Digoxin, Isoprenaline and base to the improved condition of the infant cannot be distinguished.

Phenoxybenzamine is a useful adjunct to the therapy of a low output state when improvement has not occurred with Digoxin, Isoprenaline and base. Because of marked vaso-

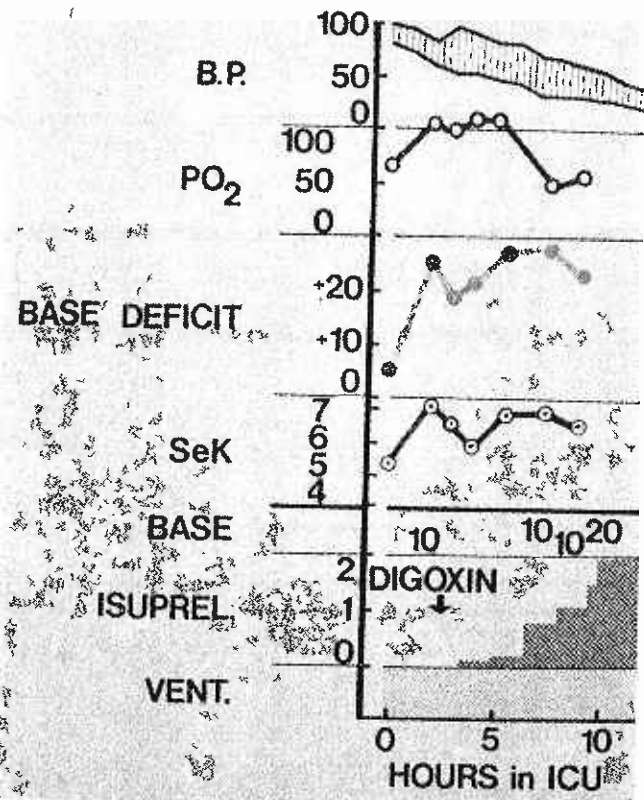


Fig. 1. Case 1. Changes in arterial blood pressure (mm Hg.) Arterial PO_2 (mm Hg.) Base deficit (mEq./L) and Serum Potassium (mEq./L). Treatment with base (mEq.) Isoprenaline (microgram/Min.) and ventilation.

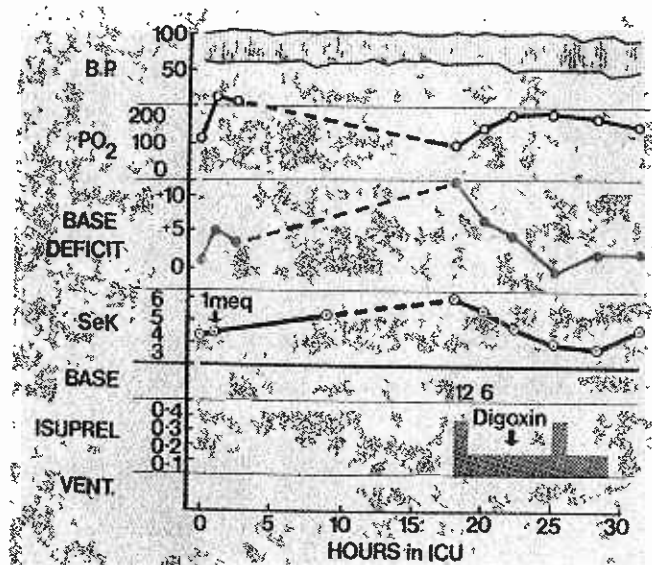


Fig. 2. Case 2. Post-operative progress.

constriction, Phenoxybenzamine was given earlier than usual in this infant but it is important not to delay its introduction unduly as it appears less effective when low cardiac output and intense peripheral vasoconstriction have been present for a prolonged period. Simultaneous transfusion may be required to maintain atrial pressures and Phenoxybenzamine administration may have to be repeated as early as 10 hours after the first dose.

The unreliability of the clinical assessment of a low output state is notorious.⁸ In the 82 patients of the present series, five infants died from a probable low output state which might have responded to more vigorous treatment. In each case, greater attention to urinary output, serum potassium and base deficit might have anticipated the disaster but repeated measurements of cardiac output would

*Footnote: This patient was treated after December 1971.

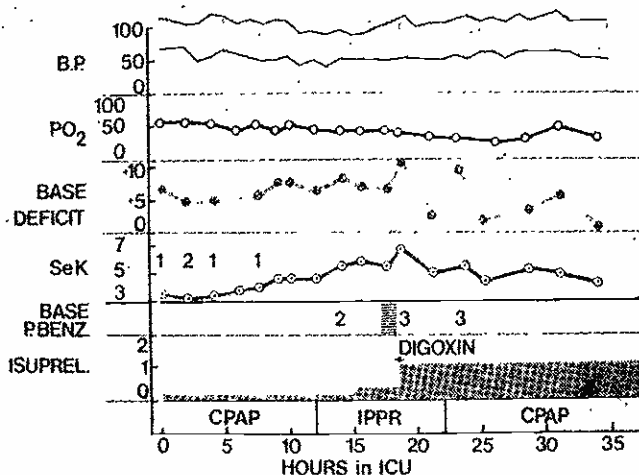


Fig. 3. Case 3. Figures in Serum Potassium section indicate mEq. Potassium given. Hatched area in next section indicates time of administration of Phenoxybenzamine.

clearly be superior. Although measurements of acceptable accuracy can be made by dye dilution, this method is cumbersome in a busy intensive care unit and it is hoped that the thermodilution technique can be adapted for use in infants.

RESPIRATORY SUPPORT

Over the period of this study, respiratory support was given by IPPR using a pressure-cycled patient-triggered ventilator. Intubation was carried out with portex nasotracheal tubes, tracheostomy being required in only 2 infants. IPPR was used in 37 patients, the indications being respiratory problems in 11, circulatory problems in 13 and a combination of the two in 13. It was required most frequently in patients with total anomalous pulmonary venous connection, persistent truncus arteriosus, pulmonary stenosis or atresia and complex transposition. The respiratory indications for IPPR were inadequate ventilation or respiratory arrest, and respiratory complications attributable to the surgical procedure itself were very uncommon. During ventilation, areas of atelectasis occurred in 13 patients of whom 5 developed frank pulmonary infections but the only lethal complication was the late development of sub-glottic stenosis in one of the 2 patients who required tracheostomy.

Recently Kirklin⁸ reported the post-operative use of CPAP as originally described by Gregory *et al.*,⁹ in the treatment of respiratory distress of the newborn. Kirklin now uses this technique in all infants after corrective cardiac surgery, starting with a CPAP of 6 mm.Hg. and an FIO₂ of 0.75. The anaesthetic bag joined to a side arm of the circuit permits intermittent manual ventilation where necessary. Since learning of this application of CPAP we have used the technique in most infants requiring respiratory support, but have still resorted to IPPR on a number of occasions. It appears likely that both techniques will retain a place in management, particularly of the younger patient.

We have not used CPAP as a routine, preferring to remove the endotracheal tube in the operating theatre where possible. On several occasions however, removal of the endotracheal tube has proved premature and the use of CPAP allows for excellent control in weaning the patient off ventilatory support.

DEATHS

Eighteen patients died in the intensive care unit (Table V). It should be possible virtually to eliminate deaths from respiratory causes and the major remaining problem is the management of a low output state. The death of the 5 patients with incompletely corrected lesions was unavoidable. The 5 patients listed as having "satisfactory treatment" were treated intensively but in the absence of records of cardiac output, we could not be certain that management was ideal. In each of the 5 cases listed as having "inadequate treatment", cardiorespiratory collapse occurred suddenly when the infants were thought to be in a satisfactory condition. In retrospect each patient was probably in a low output state, in which minor respiratory problems or other disturbances may trigger abrupt decompensation. This is the area requiring the most meticulous monitoring if post-operative deaths are to be reduced to a minimum.

SUMMARY

Post-operative management is described for 82 patients under 1 year of age after cardiac surgery under profound hypothermia.

In most cases blood loss was readily managed by accurate replacement with whole blood and freeze-dried plasma. Fluid intake was restricted to 2 ml./kg./hr. Oliguria was common in the first 24 hours but no renal damage occurred. Many patients required bicarbonate and potassium supplements. Arrhythmias were common and insertion of pacemaker wires is now routine. Respiratory support was provided by IPPR in 37 patients but more recently the technique of CPAP has been introduced. The major problem in management has been the detection and treatment of lowered cardiac output. Eighteen patients died, most from circulatory causes.

ACKNOWLEDGEMENTS

This study was supported in part by the Medical Research Council of New Zealand.

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