

Anesthesia for Cardiovascular Surgery in Infancy

M. Jerome Strong, M.D., Arthur S. Keats, M.D., Denton A. Cooley, M.D.

Since 1954, approximately 700 infants less than one year of age have been treated surgically in this institution for congenital malformations of the heart and great vessels. The details of anesthetic management of the last 100 infants have been summarized and analyzed with special attention to the relationship between the respiratory and circulatory abnormalities of congenital heart disease and the anesthetic agents and techniques. Problems considered in the management of these infants include the emergent nature of operation, preoperative state of the patients, cardiac arrhythmias, blood and fluid replacement, cardiopulmonary bypass in infants, and problems posed by the unique pathophysiology of specific cardiac malformations. Respiratory insufficiency from a variety of causes and central nervous system damage were particular problems in the early postoperative period.

SINCE 1954, approximately 700 infants less than one year of age have been treated surgically in this institution for congenital malformations of the heart and great vessels.¹ In the course of this experience, anesthetic techniques were devised and frequently modified in response to new knowledge of the pathophysiology of these diseases, new surgical techniques, and new anesthetic agents. During this time, there was a notable paucity of published experience for guidance on anesthetic management of this type of patient. Only McQuiston,^{2, 4} Smith,³ Harris,⁵ Smith,⁶ and Bergner⁷ have written on anesthesia for congenital heart disease. Three other reports considered the management of patients with specific cardiac anomalies.^{8, 9, 10} Few attempts were made to relate the circulatory and respiratory abnormalities of congenital cardiovascular disease to the anesthetic agents and

techniques used, particularly in the infant. This then is the primary purpose of this report, which will summarize and analyze in these terms our successes and failures in the management of the last 100 consecutive patients, less than 1 year of age, with congenital heart disease.

General Considerations

The Patients. Surgical treatment of congenital heart disease in infants less than one year of age was usually an emergency undertaken as a result of failure of medical therapy. The usual indications were progressive or intractable heart failure and severe hypoxemia in infants with lesions amenable to surgical therapy. Operation was undertaken as a desperation measure in 16 infants, *in extremis*. Three infants were admitted to the hospital in frank pulmonary edema. Endotracheal intubation without anesthesia or relaxant, positive pressure respiration and rapid digitalization were carried out just before operation. Five infants were operated upon in a moribund state immediately following cardiac catheterization during which circulatory collapse had occurred. Ten of 34 patients with cyanotic heart disease suffered paroxysmal hypoxemic spells (tachypnea, intense cyanosis, syncope and convulsions) in the hospital. Eight were operated upon shortly after a severe spell and were flaccid and unresponsive when anesthesia was induced.

The cardiac defects in these 100 patients are shown in table 1 and are listed according to curative or palliative operations performed. The surgical procedures carried out for the specific cardiac defects are listed in table 2. Since failure to thrive is common with congenital heart disease, most of these infants were small. Seventy-eight weighed less than 10 pounds and 34 less than 7 pounds at the time of operation. Sixty-three infants were less than 2 months and 40 less than one month of age (table 3).

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TABLE 1

Anomaly	Number of Patients	Number of Patients Living
Treated by complete correction		
Patent ductus arteriosus	9	7
Coartation of aorta	9*	7
Total anomalous venous return	5	2
Pulmonary stenosis	6	4
Aortic stenosis	5	3
Atrial and/or ventricular septal defect	5†	4
Vascular ring	1	1
Palliative treatment		
Transposition of great vessels	16	12
Tetralogy of fallot	10	8
Ventricular septal defect	11‡	8
Tricuspid atresia	8	7
Miscellaneous	15§	8
Total	100	71

* Includes 3 patients with preductal coartation and multiple intracardiac defects.
† 2 patients had ASD of ostium primum type, 1 patient had VSD, 2 patients had both an ASD and VSD.
‡ Includes 9 patients with associated PDA and coartation with VSD.
§ Includes single ventricle, atrioventricularis communis, truncus arteriosus and multiple complex defects.

Ten infants were born of mothers who had had rubella during the first trimester of pregnancy. Congenital anomalies following maternal rubella constitute a distinct disease syndrome which includes cataracts, deafness and a variety of cardiac malformations with or without thrombocytopenic purpura.¹¹ The commonest cardiac defect in the maternal rubella syndrome is patent ductus arteriosus, which was present in 5 of these 10 infants. Three infants had thrombocytopenic purpura, one with an anemia of 7 g./100 ml.

Peanesthetic Drugs. The 16 infants mentioned above received no preanesthetic medication because of the emergent nature of the operation. The remaining 84 infants received only atropine 0.1 mg./101 pounds, intramuscularly, 45 minutes before operation. Atropine was used not primarily for its drying effect but rather to prevent the bradycardia of cyclopropane and succinylcholine. Since most in-

fants were hypoxic preoperatively, even brief periods of breath-holding or respiratory obstruction during induction, especially with cyclopropane, produced prompt bradycardia (less than 100 beats per minute). Atropine delayed the onset of this bradycardia.

Morphine in doses of 0.25-0.5 mg./10 pounds of body weight had been given therapeutically to all infants with hypoxemic spells. Morphine was used to decrease oxygen demand by decreasing motor activity, especially the tachypnea. At times morphine dramatically decreased cyanosis and prevented further hypoxemic spells.

The 5 infants brought directly from the cardiac catheterization laboratory in circulatory collapse had received a variety of drugs for sedation (pentobarbital, meperidine) and for resuscitation (procaine amide, atropine, calcium, isoproterenol, sodium bicarbonate).

Infants with signs of congestive heart failure were digitalized prior to operation. None were digitalized prophylactically. A deliberate attempt was made to achieve less than maximum digitalization if immediate operation was anticipated in order to decrease the likelihood of ventricular arrhythmias, atrioventricular dissociation or heart block as a result of

TABLE 2

Malformation	Surgical Procedures Performed
Patent ductus arteriosus	Ligation or division
Coartation of aorta	Resection and anastomosis
Total anomalous pulmonary venous return	Complete correction with cardiopulmonary bypass
Pulmonary valve stenosis	Closed or open valvulotomy with or without cardiopulmonary bypass
Atrial septal defect and/or ventricular septal defect	Pulmonary banding or closure of defect with cardiopulmonary bypass
Aortic valvular stenosis	Aortic valvulotomy with cardiopulmonary bypass
Transposition of the great vessels	Creation of atrial septal defect (Blalock-Hanlon operation)
Tetralogy of Fallot	Systemic to pulmonary artery anastomosis (Potts or Blalock operation)
Tricuspid atresia	Systemic to pulmonary artery anastomosis

TABLE 3

Age Range in Months	Number of Patients	Weight Distribution (pounds)				
		3-5	5-7	7-10	10-15	15-20
0-1	40	10	17	13		
1-2	23		5	18		
2-3	9			5	4	
3-6	13		2	5	4	2
6-12	15			3	6	6
Total	100	10	24	44	14	8

digitalis toxicity. Even in the absence of digitalis, these arrhythmias tend to occur in hypoxic infants with anesthesia and cardiac manipulation. In addition, it was anticipated that the cardiac status of the infants would be improved by operation and maximal digitalization in the preoperative period may represent digitalis intoxication in the postoperative period as a result of the improved functional state. Digitalized infants were still able to respond to atropine with an increased heart rate.

Induction and Maintenance. Except for infants already intubated, anesthesia was induced with 50 per cent cyclopropane in oxygen using an infant to-and-fro system with carbon dioxide absorption. As soon as consciousness was lost, succinylcholine (15-30 mg.) with atropine (0.1-0.15 mg.) in the same syringe was injected intramuscularly. Injections were given into the deltoid muscle and rubbed vigorously. Complete muscle paralysis usually appeared within one minute, but at times was delayed as long as three minutes in infants with marked circulatory failure. Early in this experience when succinylcholine was administered in this manner without atropine, severe bradycardia usually with nodal rhythm occurred in 20-30 per cent of patients and was responsible for cardiac standstill in two infants. For this reason, succinylcholine was always given with atropine. Although these arrhythmias might be expected more frequently in digitalized infants,¹² we could not relate its incidence to the use of digitalis.

Following complete paralysis, tracheal intubation was performed with the largest tube possible. Since an Ayre's insufflation system was used for maintenance, a large tube minimized the air leak so that sufficient intrapul-

monary pressure for controlled respiration with an open chest and for re-expansion of atelectatic lungs could be attained. Since a larger tube could be inserted in the paralyzed infants compared to those awake, the endotracheal tube of infants who were intubated preoperatively was changed to a larger one after induction of anesthesia and paralysis.

Anesthesia was maintained with 0.5-1.0 per cent halothane in oxygen administered through a Y insufflation system (modified Ayre) with a 5-8 liter gas flow. Respiration was controlled by intermittent occlusion of the expiratory limb of the Y, aided by intermittent administration of 0.2 per cent succinylcholine infusion.¹³ Usually succinylcholine was administered only until respiratory control could be gained and during the critical period of the operative procedure to insure immobility of the mediastinum, *e.g.*, during vascular anastomosis.

The Ayre's system, because of its simplicity and low resistance during spontaneous respiration, has continued to be highly satisfactory for maintenance of anesthesia. The major disadvantage has been the drying effect on the tracheobronchial tree and the tendency for mucus plugs to form in the small endotracheal tube. Since drying is a function of time, this has been a problem only in the postoperative period.

Halothane was used because it is nonexplosive and unshielded electronic monitors were used nearby. In addition, its potency permitted use of high concentrations of oxygen. Since only light planes of general anesthesia were required, bradycardia and ventricular arrhythmias from halothane alone did not occur. Nitrous oxide was not used with halothane because of the respiratory abnormalities associated with congenital heart dis-

ease. Decreased pulmonary compliance has been demonstrated in all congenital cardiac anomalies regardless of pulmonary blood flow or degree of shunt.¹⁴ Those patients with increased pulmonary blood flow have a significant decrease in pulmonary diffusing capacity as well.¹⁵ In addition, 52 patients were in the lateral decubitus position during operation and disturbance in the ventilation-perfusion ratio is well known to occur in this position.¹⁶ Finally, in order to provide adequate surgical exposure in operations such as resection of coarctation, it was necessary to retract the left lung in such a manner that it could not be ventilated at all. Oxygenation was therefore maintained by the dependent lung only. For these several reasons nitrous oxide was not added to halothane.

A naso-gastric tube was inserted in all infants following endotracheal intubation and left in place for several hours postoperatively. This insured removal of air and gastric secretions the accumulation of which can lead to inadequate tidal exchange in infants.¹⁷

Seventy five per cent of infants were admitted to the recovery room with the endotracheal tube in place, even though many were awake or reactive at this time. Extubation was carried out only when the infant vigorously objected to the endotracheal tube. Following cessation of surgical stimuli at the end of operation, some infants who appear awake and active return to deep sleep in the recovery room. The endotracheal tube therefore insured an airway for treatment of hypoventilation from this cause.

Sixteen infants required total cardiopulmonary bypass for correction of their defects. Their anesthetic management differed only in that *d*-tubocurarine (0.3 mg./10 pounds) was given just prior to bypass, to insure immobility since no anesthetics were administered through the oxygenator. The 14 infants who survived bypass breathed adequately at the end of operation and neostigmine was not required.

Cardiac Arrhythmias. These occurred at some time in all patients whose hearts were manipulated. However, the severity, duration, and reversibility of arrhythmias seemed to some degree related to depth of anesthesia. Cessation of manipulation and withdrawal of the anesthetic were the most effective remedies

for arrhythmias, particularly of ventricular origin. When these measures were not successful, intravenous atropine (0.05–0.1 mg.) was effective for nodal bradycardia and phenylephrine (0.05–0.1 mg.) or neostigmine (0.15 to 0.25 mg.) in repeated doses, when necessary, was effective for supraventricular tachycardia. Since most arrhythmias were precipitated acutely by manipulation of the heart every effort was made to avoid treatment by drugs such as digitalis, quinidine and procaine amide whose action persist into the postoperative period. No patient in this group received any of these drugs during anesthesia. Arrhythmias primarily the result of hypoventilation, hypotension, or excessive anesthesia did not occur in this group.

We were unable to define the role of digitalis in contributing to ventricular arrhythmias during anesthesia. In one infant who had been rapidly digitalized during the hour prior to operation, induction of anesthesia with cyclopropane promptly led to ventricular fibrillation which was successfully converted by a single external d.c. shock. Two other infants in this group were anesthetized with cyclopropane under identical circumstances without difficulty.

Two infants developed complete heart block; one secondary to surgical closure of a ventricular septal defect and one followed aortic valvulotomy. Repeated intravenous doses of isoproterenol (10–20 μ g.) together with an internal pacemaker were ineffective in providing adequate circulation and both expired.

Hypothermia. Deliberate hypothermia was not used in any patient and every effort was made to prevent inadvertent hypothermia. Hypothermia complicated unnecessarily the care of these patients by contributing to postoperative respiratory insufficiency, bleeding, and difficulties in assessing the circulatory status. Despite the routine use of a heating pad during anesthesia, 67 infants had rectal temperatures less than 97° F. and 35 less than 94° F. on admission to the recovery room. The effectiveness of a heating pad was obviously limited in the 52 infants operated on in the lateral decubitus position.

Monitoring During Anesthesia. A precordial stethoscope was used during induction of anesthesia. An esophageal stethoscope and

the ECG, were used during maintenance. Systolic blood pressure was determined with a cuff by palpation of the radial or ulnar artery. In aortic stenosis, transposition of the great vessels, and in malformations with severe heart failure, the radial, ulnar and brachial pulses are usually so weak they cannot be used for monitoring. In such patients palpation of a carotid or axillary artery can, with experience, be an acceptable substitute. Direct intra-arterial and venous pressure measurements were not used in this age group.

During extracorporeal circulation, the electroencephalogram (EEG) proved useful in assessing the adequacy of cerebral perfusion. Even though newborns and young infants have a slow wave pattern while awake and depth of anesthesia is difficult to assess by EEG, cerebral hypoxia from inadequate perfusion promptly produced an isoelectric pattern.

Blood Replacement. Since 60 per cent of the infants suffered from heart failure or were polythemic before operation, blood transfusion was used sparingly. Although hemoglobin was less than 10 g./100 ml. in 7 infants, only 2 were given packed cells before operation. Sixteen infants received blood as part of cardiopulmonary bypass since the oxygenator was primed with heparinized blood. Of the remaining 84 infants only 19 received any blood during their entire hospital stay. Two received packed cells preoperatively, 9 were transfused with whole blood during operation and 8 in the postoperative period to replace blood loss. No polycythemic infant was transfused. Had it been necessary, whole blood rather than plasma would have been used. Blood loss during operation was estimated visually. Sponges were not weighed and blood was not replaced as lost. Blood was given only when definite signs of hypovolemia appeared (hypotension, pallor, or a fall in previously elevated venous pressure). Palpation of the right atrium or at times the external jugular vein provided a good index of venous pressure for this purpose. In patients with right sided heart failure, *e.g.*, anomalies associated with pulmonary stenosis, the size of the liver was a good index of venous pressure responding promptly by a change in size to hemorrhage and transfusion. In contrast to adults, tachycardia was not use-

ful as an index of blood loss in these infants, since normal heart rates were so high.

A dilemma frequently arose as to the wisdom of transfusion in an infant with congestive failure who had bled much but who remained in congestive failure with elevated venous pressure or pulmonary edema. We have not transfused these infants until signs of congestive failure disappeared as a result of therapy of myocardial failure or as a result of arterial hypotension. Vasopressors have not been useful in this situation.

Fluid Therapy. A micro-drip infusion of 5 per cent dextrose in 0.25 normal saline was started in the operating room after induction of anesthesia. In almost 60 per cent of patients it was possible to insert an 18 gauge Rochester needle into a vein on the dorsum of the hand or less commonly the ankle, thus avoiding a cutdown. The hand was preferred to the ankle since infiltration or obstruction could be noted promptly and corrected by the anesthesiologist. Scalp vein infusions have not been satisfactory because of the ease with which they become dislodged during and after operation. A secure route for intravenous medications was essential for the survival of many patients.

Despite efforts to administer a minimum volume of intravenous fluid all patients received 50–75 ml. during operation as the volume necessary to maintain patency of the needle and as a vehicle for drugs. Postoperatively this same solution, 5 per cent dextrose in 0.25 normal saline was administered by microdrip at 25 ml./pound/day or 750 ml./m.²/day.

Anesthetic Problems in Specific Cardiac Malformations

Tetralogy of Fallot and Pulmonary Stenosis. Both these anomalies are characterized by obstruction to outflow of the right ventricle and a low pulmonary blood flow. Deep cyanosis with polycythemia is common in patients with tetralogy due to the large right to left shunt through a ventricular septal defect and to the chronic hypoxemia with compensatory polycythemia. However, cyanosis is not common in infants with pulmonary stenosis with an intact ventricular septum. Because of the critically low pulmonary blood

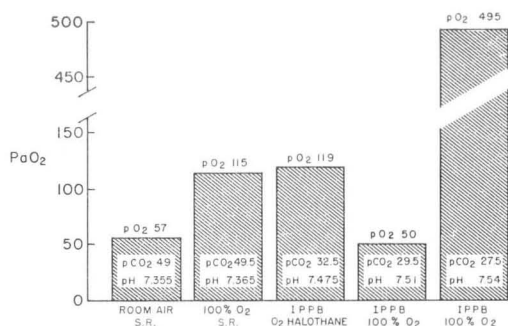


FIG. 1. Blood gas studies during complete surgical correction of tetralogy of Fallot in a 6 year old girl weighing 20 kg. who had had a Potts anastomosis 4 years previously. Arterial blood was sampled at 5 periods: (1) spontaneous respiration with room air (Room Air S.R.), (2) spontaneous respiration with 100 per cent oxygen (100% O₂ S.R.), (3) controlled respiration (+5 to +15 cm. of water) with 0.5 per cent halothane in oxygen with closed pleura (IPPB O₂—halothane) and (4) controlled respiration (+5 to +15 cm. of water) with 0.5 per cent halothane in oxygen with open pleura (IPPB 100% O₂) and immediately following correction of the defect with pleura open and controlled respiration with 100 per cent O₂. The patient who was heavily premedicated with pentobarbital, meperidine and scopolamine slept during the first two sampling periods and moderate respiratory depression was evident (Pco₂ 49 mm. of mercury). Administration of 100 per cent oxygen for 5 minutes increased Pao₂ from 57 to 115 mm. of mercury. After intubation and hyperventilation with halothane oxygen no additional increase in Po₂ occurred. When the pleura was opened, cyanosis increased and Po₂ fell to 50 mm. of mercury despite continued hyperventilation with halothane oxygen and without atelectasis or systemic hypotension. The loss of the negative intrapleural pressure combined with positive pressure ventilation increased pulmonary vascular resistance leading to increased right to left shunting through the VSD and increased cyanosis. After complete repair of the malformation, Po₂ increased to 495 mm. of mercury during oxygen breathing with an open pleura.

flow, situations which cause pulmonary vasoconstriction (hypoxia and hypercarbia) or increased pulmonary vascular resistance (excessive positive airway pressure) produce marked hemodynamic effects. In pulmonary stenosis high airway pressure leads to prompt circulatory collapse from decreased left heart filling. In tetralogy of Fallot, high airway pressure, by increasing resistance to right heart outflow, increases the right to left shunt leading to an increase in cyanosis despite hyperventilation with oxygen. This has been noted previously

by Harmel and Lamont¹⁰ and Shields* has documented this by recording a fall in oxygen saturation by ear oximeter, when the pleura was opened and controlled respiration instituted. We have frequently observed a progressive increase in cyanosis with increasing duration of positive pressure respiration during open pleura in patients with tetralogy. Cyanosis increased despite high inspired oxygen mixtures and without collapse of any lung tissue (fig. 1).¹⁸ On the other hand, patients with high pulmonary blood flow (atrial and ventricular septal defects, patent ductus arteriosus) not only tolerate elevated airway pressures well, but often require high pressure to provide adequate ventilation.

The polycythemia of patients with tetralogy is associated with thrombocytopenia and hypofibrinogenemia which accounts for their poor clotting and tendency to bleed postoperatively.¹⁹ Paradoxically this polycythemia and increased blood viscosity also predispose to the intravascular thrombosis, particularly cerebral thrombosis which occurs not rarely in such patients.²⁰ Six polycythemic infants in this series required a second operation because of the thrombosis of a previously performed systemic to pulmonary artery shunt. Three polycythemic infants had transient convulsions in the postoperative period. Since all 3 recovered without neurologic deficit, it was postulated that increased blood viscosity possibly associated with dehydration may have led to cerebral thrombosis.

Total Anomalous Venous Return (TAVR). In the common form of the anomaly all pulmonary veins empty into the right atrium either directly or via a left superior vena cava. Oxygenated blood reaches the left heart through an interarterial septal defect without which survival is not possible. For this reason operative manipulation or distortion of the right atrium which would be well tolerated by other patients may obstruct flow to the left heart in these patients causing prompt hypotension and bradycardia. Transfusion is particularly hazardous in this anomaly because any increase in right atrial pressure is promptly transmitted to the pulmonary veins with con-

* Personal Communication from J. R. S. Shields, University of Washington, St. Louis, Missouri.

sequent pulmonary edema. Bloody pulmonary edema may also appear immediately after repair of this defect if any obstruction to pulmonary venous return has been produced by the repair. Four of the 5 patients in this group had pulmonary edema in the early post-operative period and 3 expired within six hours of operation from this cause.

Transposition of the Great Vessels. In this defect, blood flows from the right ventricle directly to aorta and from the left ventricle to the pulmonary artery. Thus two separate circulations exist and survival is possible only because of mixing between these circulations through a patent foramen ovale and/or patent ductus arteriosus or VSD. Some oxygenated pulmonary venous blood passes through the atrial communication to the right atrium and thence to the aorta. The palliative procedure performed (Blalock-Hanlon operation) consists of creation or enlargement of an existing atrial defect to increase the admixture of pulmonary venous blood to the systemic circulation or to relieve the high left atrial pressure. Cardiopulmonary bypass is not required.

Because most pulmonary venous blood is pumped back through the pulmonary artery, induction of inhalation anesthesia is always prolonged. Only a small portion of the anesthetic taken up in the lungs reaches the systemic circulation and brain, and high concentrations of inhalation agents are well tolerated during induction. For the same reason, the administration of oxygen results in little improvement in cyanosis. This is in contrast to cyanotic patients with tetralogy of Fallot (fig. 1) in whom administration of oxygen decreases pulmonary vascular resistance and increases the oxygen content of whatever blood flows through the lungs with improvement of cyanosis.

To create a septal defect, it is necessary to occlude temporarily the right pulmonary artery and veins. This maneuver increases the hypoxemia and may lead to bradycardia, hypotension, and finally cardiac arrest. Severe bradycardia and hypotension occurred in 7 of 16 patients with TGV and was treated successfully in all with atropine and phenylephrine. Operative speed to minimize the duration of

occlusion of one half the pulmonary circulation is vital to the success of this operation.

Because of its low blood gas solubility and consequent rapid uptake and elimination, cyclopropane has been a useful agent for these infants. Cyclopropane in 10–20 per cent concentration with the Ayre's insufflation technique was used in 6 of 16 patients. More recently halothane-oxygen has been used because of the explosion hazard of open cyclopropane.

Atrial and Ventricular Septal Defect (ASD and VSD): The surgical repair of atrial septal defects was limited to two infants with *ostium primum* defects associated with cleft mitral valve, mitral insufficiency, progressive cardiac enlargement and intractable failure.²¹ Because of the close anatomic association of the mitral valve with the bundle of His, atrioventricular block may result from the repair. Although neither infant suffered this complication, both had transient pulmonary edema in the operating room following repair.

Because of the high mortality (42 per cent)¹ associated with complete repair of ventricular septal defects in infants, banding of the pulmonary artery was the procedure of choice in infancy. Pulmonary artery banding reduces the heart failure and may prevent permanent pulmonary vascular changes secondary to high pulmonary blood flow and pulmonary hypertension. The indication for pulmonary banding was a large left to right shunt with pulmonary hypertension.¹ Of 14 infants with ventricular septal defects, 11 had a banding of the pulmonary artery and 3 infants with both ASD and VSD and severe congestive failure underwent complete repair with one death due to underdevelopment of the left ventricle.

The Postoperative Period

In addition to the postanesthetic problems which may occur in any infant such as residual effects of muscle relaxants or anesthetics, airway obstruction, and hypothermia, those following cardiovascular surgery include cardiac failure with or without arrhythmias, pneumothorax, hemothorax, cardiac tamponade, pulmonary hypertension with systemic hypotension and central nervous system damage. In this group of patients the postoperative prob-

lems were limited to respiratory insufficiency and central nervous system damage.

Respiratory Insufficiency. It was often difficult in these patients to distinguish between respiratory insufficiency of pulmonary origin and insufficiency secondary to cardiac failure. Tachypnea, sternal and intercostal retraction, flaring of the alae nasi, grunting respiration, and diminished breath sounds were characteristic not only of lower respiratory tract obstruction from secretions or diffuse atelectasis, but also of pulmonary edema without rales and pulmonary hypertension.

Twenty-two infants in this group suffered postoperative respiratory insufficiency requiring special therapy. Eleven were admitted to the recovery room with inadequate respiration: Respiratory insufficiency was secondary to pulmonary edema in 7, to hypothermia (88° F. by rectum) in one, and to anoxic brain damage in 3. Two infants were moribund from cerebral anoxia suffered prior to operation for palliation of TGV. The other infant had been inadequately ventilated during anesthesia with consequent cerebral anoxia. (See below.)

Eleven infants developed respiratory insufficiency after admission to the recovery room with adequate respiration. Six suffered from atelectasis or unilateral massive collapse and were treated by tracheal suction after insertion of an endotracheal tube. Two of these subsequently died and at autopsy had microscopic evidence of underdeveloped lung tissue. Four infants had mechanical interference with respiration. In one complete respiratory obstruction developed due to inspissation of secretions in a endotracheal tube. This was promptly recognized and corrected. The lumen of these small tubes is easily obstructed by inspissated mucus or blood, especially after pulmonary edema when an Ayre's system with dry gases is used. A microdrip of saline into the endotracheal tube at a slow rate prevents this complication. A second infant developed marked gastric distention which interfered with respiratory exchange and which was promptly relieved by a nasogastric tube. A third infant developed pleural effusion 2 days after pulmonary banding and required a thorocentesis to improve ventilation. The fourth infant with a large VSD had compression of a mainstem bronchus by a dilated right atrium which

existed preoperatively. The size of the atrium gradually decreased during the postoperative period with improvement in respiration.

Sequelae

Five patients exhibited signs of central nervous system injury in the postoperative period. Thirty-six hours following open repair of aortic stenosis one infant developed generalized clonic-tonic seizures and left hemiparesis probably secondary to an embolus. Cerebrospinal fluid pressure and composition were normal and recovery was complete in 10 days. Intermittent cerebral edema and finally death followed superior caval-pulmonary artery anastomosis in one patient. At autopsy the shunt was completely occluded by thrombus. The three remaining infants with cyanotic heart disease, polycythemia and cerebral thrombosis were discussed above.

Mortality. Overall hospital mortality in this group was 21 per cent. Mortality by diagnosis is shown in table 1. Errors in anesthetic management contributed to three deaths. Severe hypotension leading to cardiac arrest occurred after injection of protamine in one infant who underwent cardiopulmonary bypass for aortic stenosis. Resuscitation was not successful. Since protamine is always given shortly after bypass and during a period when myocardial failure, hypovolemia, hypervolemia, or pulmonary edema may appear, it is commonly claimed to be responsible for these events. An etiological relation has not been well established. In a second infant, death was due to inadequate blood replacement when the venous cutdown in the ankle became dislodged and was unrecognized. An unrecognized leak in the halothane vaporizer resulted in hypoventilation of a third infant, who never regained consciousness or spontaneous respiration after operation.

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TRANSFUSION HEPATITIS The incidence of hepatitis following transfusion of 1 to 2 units of blood ranges from a few per million to 2 to 3 per cent. After multiple transfusions, the incidence rises even further. Since the hepatitis virus cannot be eliminated by sterilization, selection of donors was carried out as follows: (1) Exclusion of all individuals who give a history of ever having had viral hepatitis or whose blood donation has caused hepatitis in at least two recipients. (2) Careful physical examination of all prospective donors. (3) Biochemical evidence of latent parenchymal liver damage before each donation of blood. The most feasible procedure for screening donors appeared to be the estimation of glutamic-pyruvic transaminase (GPT) activity. In heart surgery requiring total bypass, 1509 blood samples of 925 donors were checked for GPT activity before use. Five and three-tenths per cent of the samples showed elevation of GPT above 20 U corresponding to 8.2 per cent of the donors. Repeated studies of the donors occasionally showed an increase in GPT despite initially normal values, stressing the importance of checking every pint of donated blood. In 43 per cent of the donors with abnormal GPT levels, further liver function tests were performed. In nearly half of them at least two or more tests were abnormal. Where liver biopsy was performed, it confirmed the presence of liver disease. By elimination of abnormal samples and by prophylactic administration of gamma globulin, the incidence of hepatitis in patients undergoing total bypass could be reduced from 13 per cent in the control period to 2.2 per cent during the period of GPT screening. (Walter, S.: *The Value of Transaminase Estimation in the Selection of Blood Donors Particularly for the Purpose of Extracorporeal Perfusion*, *Thoraxchirurgie* 13: 336 (Oct.) 1965.)