ANAESTHESIA WITH HYPOTHERMIA FOR OPEN HEART SURGERY IN CHILDREN*

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THE USE OF HYPOTHERMIC ANAESTHESIA to allow "open" heart surgery has become commonplace. The technique is simple and relatively safe and allows direct visual repair of the defect. Its major disadvantage is the limited time available. Therefore, this method is usually restricted to the correction of two types of defects: the atrial septal defect of the secundum type and valvular pulmonary stenosis. The first part of this paper deals with the management of 100 patients with an atrial septal defect, and the second part with 55 patients with valvular pulmonary stenosis. The pathology of each lesion will be briefly reviewed, followed by the anaesthetic management and our results.

ATRIAL SEPTAL DEFECT

Pathological Effects

Figure 1 shows the four main types of defects in the atrial septum viewed from the left side of the heart. Only defects of the ostium secundum type or those involving a patent foramen ovale are suitable for closure using hypothermia alone. Ostium primum defects and atrial ventricular communis defects require too much time for this method. Figure 2 shows a typical secundum type defect.

Figure 3 is a diagrammatic presentation of the haemodynamics resulting from this lesion. With an intact septum the left atrial pressure is 5 mm. higher than the right atrial pressure. With a small A.S.D., there is a pressure gradient of approximately 3 mm. of mercury, producing a left to right shunt. With larger defects the pressure difference is abolished, but a left to right shunt persists. The pulmonary blood flow may increase from two to four times that of the systemic system. The right ventricle adapts well to such a work load over many years and the pulmonary pressures may be only slightly elevated. Ultimately, the right ventricle fails, as a result of either the excessive work load or the development of increased pulmonary vascular resistance. As the right ventricle fails, the right atrial pressure rises and a right to left shunt may develop, producing cyanosis. At this stage the operative risks are very great.

Prognosis

Without operation these patients have an uncertain future. Deaths from A.S.D. in infancy are rare and serious symptoms infrequent before the age of

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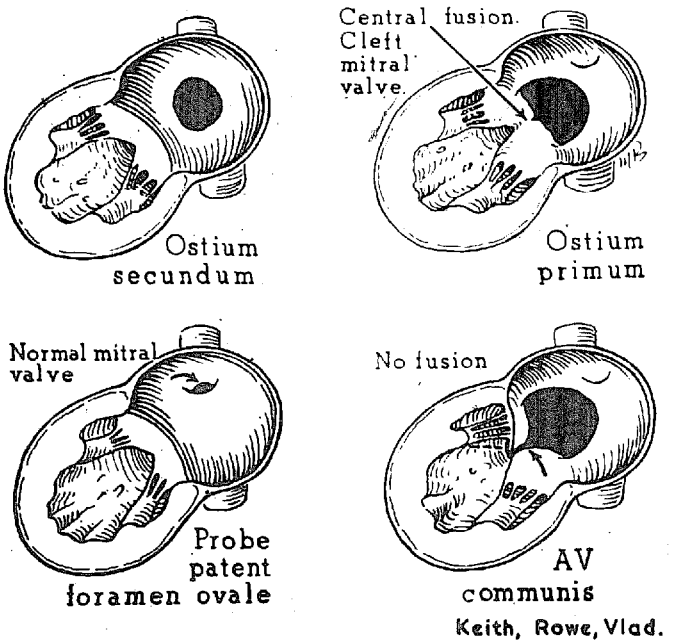

FIGURE 1

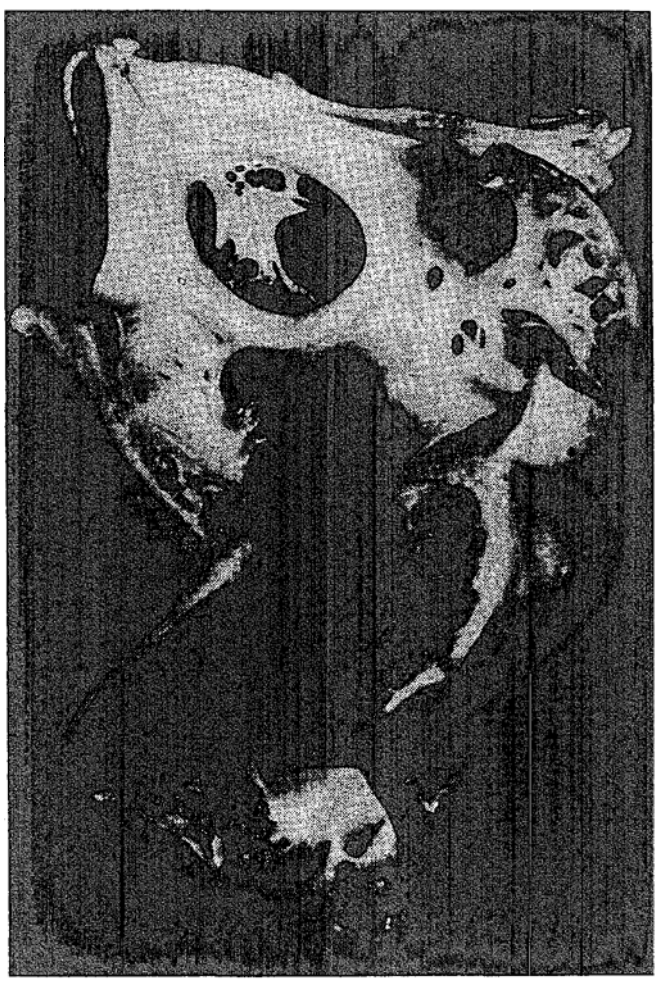

FIGURE 2

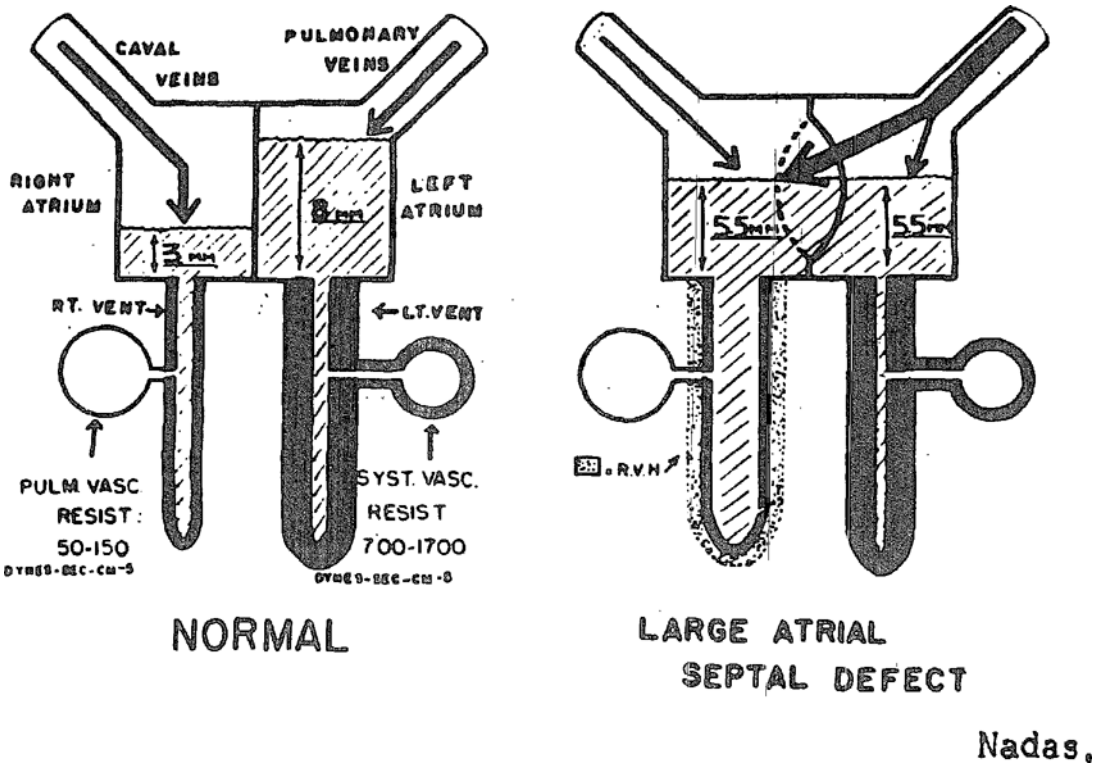

FIGURE 3

20 years. However, by 40 years of age, half the patients have gross cardiac enlargement and the average age at death is 49 years. Therefore, it is advisable to repair the defect at any early age. The age group two to ten years carries the least risk because this group have not had time to develop serious complications and the defect is still small and readily repaired.

Anaesthetic Management

The anaesthetic management begins with the preoperative visit.[1] Because this procedure is always elective in children, any complicating illness results in cancellation of the operation. The patient is thoroughly examined with special attention to the cardiovascular system. If no contraindications exist, premedication is ordered one hour preoperatively as follows:

1. Under 20 lb. atropine (0.012 mg./lb.) only.
2. 20–80 lb. atropine (0.012 mg./lb.) and meperidine (0.75 mg./lb.) and pentobarbital (1 mg./lb.).
3. Over 80 lb. atropine (0.012 mg./lb.) and morphine (0.1 mg./lb.).

The methods of induction of anaesthesia vary with the size of the patient and the severity of the cardiovascular lesion. In infants, cyclopropane and oxygen may be used followed by intramuscular succinylcholine (2 mg./lb.). If very seriously ill, succinylcholine with oxygen only is to be preferred. For the induction of babies and children, 2½ per cent thiopentone ("sleep" dose) followed by intravenous succinylcholine (1 mg./lb.) is satisfactory. After oxygenation, intubation is performed with a Portex Magill tube. After intubation anaesthesia is

maintained with nitrous oxide-oxygen until spontaneous respirations return. During cooling, nitrous oxide-oxygen-halothane (0.5 to 1 per cent) is used. During surgery, until the occlusion period, hyperventilation is maintained and nitrous oxide-oxygen-halothane is supplemented by additional succinylcholine as needed. After occlusion, hyperventilation is maintained with nitrous oxide-oxygen only.

For maximum safety during this type of surgery, various physiological functions must be monitored. In particular, the cardiovascular system must be closely checked. In addition to an oesophageal stethoscope and an oscillometer, we use a six-channel Sanborn machine with visoscopes for obtaining an electrocardiogram, electrocephalogram, and direct pressure recordings. Blood loss is measured by using calibrated suction flasks, weighing of sponges, and estimating the blood loss on drapes. Administered intravenous fluid is accurately measured. All data is charted concurrently on a blackboard to maintain an exact report on blood balance.

Hypothermia

Patients are surface cooled by using a "Therm-O-Rite" blanket with fluid circulating at 0° C., and ice-bags are placed over the axillary and inguinal areas. Patients over 75 lb. may have small amounts of ice placed directly on the skin to increase the rate of cooling. Surface ice should be used cautiously as it greatly increases subsequent drift of temperature. When temperature is below 33° C., movements of the patient should be minimal to avoid undue stimulation of the hypothermic heart. Oesophageal, rectal, and muscle temperatures are recorded every ten minutes.

The rate of cooling cannot be accurately predicted because of the numerous factors involved, such as size of patient, degree of muscle tone, amount of peripheral vasoconstriction or dilatation, amount of adipose tissue, the ambient temperature and humidity including the use of ice, and individual variation. The most reliable guide to subsequent temperatures is indicated by the slope of the temperature curve. As the slope steepens, cooling must cease somewhat earlier because of increased drift. When cooling is rapid, the oesophageal and rectal temperatures may differ by several degrees, with the oesophageal temperature usually being lower.

What is the optimal temperature for occlusion and repair of atrial septal defects? There are numerous points to consider but the time factor is the most vital one. In our series, the average time for repair of the first 75 cases was 4.8 min., but in the last 25 cases was 3.9 min. However, the occlusion period ranged from 2 to 7½ min. It is necessary to allow a maximum period of occlusion even if operative time is somewhat increased. Thus the optimal temperature is 30° to 31° C.

Rewarming is instituted according to the slope of the temperature curve and the stage of the procedure. The warming solution is circulated at 40° C. and hot water bottles may be added. When the rectal temperature reaches 33° C., the patient may be awakened and returned to the recovery room.

Within the moderate range of hypothermia used in this series, complications ascribed to hypothermia were rare. These may be mentioned as follows.

(1) Increased myocardial irritability. Eleven cases developed ventricular fibrillation but all of these occurred immediately after the occlusion period and four cases had visible coronary air emboli. Many factors other than hypothermia were involved.

(2) Alterations in bleeding and clotting times. No haematological investigations were performed in this series but haemorrhage was not a troublesome feature, except during the occlusion period.

(3) Metabolic acidosis. In adults undergoing hypothermia, an "acute acidotic syndrome" has been described during rewarming. None of our patients developed this complication. A study of the acid-base and electrolyte changes in some of our patients showed only minor alterations.

Occlusion Period

Before the heart is occluded, several steps must be taken.

(1) Blood loss must be determined and balanced. Then a 500 cc. bottle of heparinized blood must be started intravenously. This bood is administered to replace losses occuring during the occlusion.

(2) A flow of CO_2 (6 L/min) is started and connected to an "ether hook" at the operative site. This gas blankets the left atrium to avoid air emboli.

(3) Succinylcholine is injected to prolong the apnoeic phase.

(4) 100 per cent oxygen is administered for 30 sec. preceding occlusion.

During the occlusion period, additional steps must be taken.

(1) Gas flow is cut off and the lungs are allowed to collapse.

(2) The duration of occlusion is announced at two-minute intervals.

(3) As the final suture in the defect is tied, the lungs are inflated, forcing out blood which fills the left atrium and displaces any air present.

(4) Blood lost is replaced but if the loss exceeds 300 cc. a search must be made for an unrecognized left superior vena cava, a loose caval tourniquet, or a distortion of the heart producing aortic valve insufficiency.

Within one minute of the restoration of the circulation, there is a marked rise in the systolic and diastolic blood pressures, sometimes reaching 230/130. During the subsequent 10 to 40 min., there is a gradual fall to normotensive levels. This hypertensive phase always occurs to some degree unless extreme hypovolaemia exists or gross myocardial failure is present. This has been called the post-occlusion hypertensive phase and has been studied in conjunction with Dr. R. A. Millar of Montreal. He determined the catecholamine levels present during this phase and they exactly parallelled the hypertensive phase.[2] It appears that the adrenal glands are stimulated to secrete epinephrine and norepinephrine during this phase, but the exact mechanism has not yet been determined.

Results

This paper deals with 100 consecutive paediatric cases which were diagnosed as having uncomplicated atrial septal defects of the secundum type. They came to operation during the period January 1957 to April 1960. The ages ranged from 4 to 16 years and are subdivided according to Table I.

The ratio of females to males was 60/40 which tends to confirm a slightly increased incidence in females.

TABLE I

	4–7 yrs.	8–11 yrs.	12–16 yrs.	Total
Male	15	15	10	40
Female	28	19	13	60

Complications (deaths: 2 per 100 cases).

(1) September, 1957, an eight-year-old patient, 24 hours postoperatively, developed mediastinal tamponade followed by a cardiac arrest. Resuscitation was unsuccessful; lungs had gross intrabronchial and intra-alveolar haemorrhage.

(2) September, 1959, a 14-year-old patient had an incomplete closure of A.S.D. in March, 1957. At second attempt a 3-inch hole was torn in the right atrium and 7 L. of blood was lost. Resuscitation was successful but the operation was postponed. During rewarming, cardiac arrest occurred. When again resuscitated, cooling was reinstituted, and A.S.D. closed. Subsequently, the heart would not maintain its contractility.

Complications (*morbidity*: 29 *per* 100 *cases*). Cardiac arrythmias: 11 patients developed ventricular fibrillation after occlusion, 4 patients developed other serious irregularities; air emboli: 4 patients had visible coronary air emboli; 5 patients had evidence of cerebral disturbances, probably air emboli; blood loss: 10 patients lost over 1,000 cc. during occlusion; infection: 3 mild and 1 severe case; technical difficulties: 4 multiple defects, 3 associated anomalous pulmonary veins, 1 dextroposition, 1 incomplete closure, 1 thoracotomy only.

Valvular Pulmonary Stenosis

This defect is corrected by occluding the heart, opening the pulmonary artery, and incising the pulmonary valve. This procedure is named after Henry Swan who first reported it in 1954.

Congenital stenosis of the pulmonary valve is due to a fusion of the cusps which may be partial or almost complete (Fig. 4). The pathological changes are entirely the result of this obstruction to the outflow of blood from the right ventricle. The right ventricular pressure is greatly increased and may exceed systemic levels. The right ventricle hypertrophies and later undergoes dilatation and failure. The right atrial pressure also rises and in the presence of a patent foramen ovale, a right to left shunt may occur (Fig. 5).

Prognosis

Without valvotomy the prognosis depends entirely on the degree of stenosis. The more complete the stenosis, the earlier cyanosis and R.V. of atrium occur. Infants with a pin-hole orifice may develop cyanosis and failure and die in the first weeks of life. Patients with moderate degrees of stenosis may survive to middle age.

Anaesthetic management

The anaesthetic management for these cases is identical with that for atrial septal defects. The optimal temperature for this procedure is 31° to 32° C,. as the occlusion period is shorter. The average occlusion time in our cases was 3.2 min.

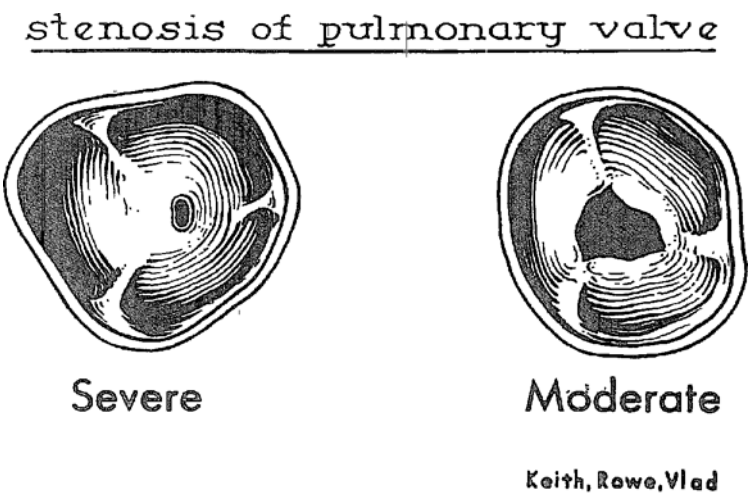

FIGURE 4

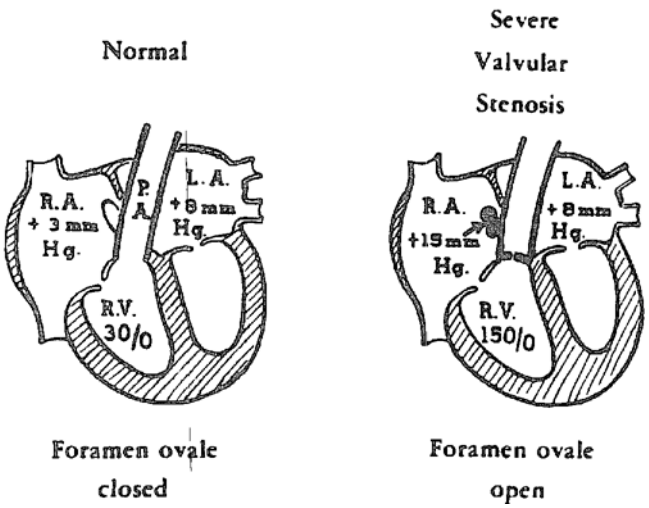

FIGURE 5

Results

TABLE II

	0–6 mos.	6 mos.–7 yrs.	8–16 yrs.	Total
Male	6	12	8	26
Female	7	14	8	29

Complications (deaths: 6 per 55 cases).

Age	*Time of death*	*Post-mortem findings*
20 days	7 hrs. postop.	Pulmonary atresia, small R.V.
10 weeks	8 hrs. postop.	V.S.D., infundibular pulmonary stenosis, small R.V.
8 days	24 hrs. postop.	Infundibular pulmonary stenosis, hypoplastic R.V.
10 days	5 hrs. postop.	Tricuspid hypoplasia, small R.V., mediastinal tamponade
21 days	14 hrs. postop.	Tricuspid hypoplasia, small R.V. pulmonary atresia
4 mos.	D.O.T.	Tricuspid atresia, pulmonary atresia.

These infants all came to operation with cyanosis and cardiac failure and were unresponsive to medical therapy. In some, there was no time for cardiac catheterization and exact diagnosis. The risks of such a major procedure to a moribund infant must be accepted. With more knowledge and experience, and infinite attention to detail, the operative mortality in these tiny infants will be reduced.

Complications (morbidity: 16 per 55 cases). Ventricular fibrillation, 11; arrythmias, 1; blood loss over 1,000 cc., 1; wound infection, 1; haemothorax, 1; atelectasis, 1.

Once the stenosed valve is incised and the circulation restored, these patients are dramatically improved. If no other lesions are still present, morbidity should not exceed that of thoracotomy.

Summary

The pathological features, the anaesthetic management, and the results of operation on 100 children with atrial septal defects and 55 children with valvular pulmonary stenosis have been discussed. These operations were carried out at the Hospital for Sick Children, Toronto, using anaesthesia with hypothermia only. This technique is simple and safe and allows direct visual repair for these defects.

Résumé

L'anesthésie sous hypothermie en chirurgie «à coeur ouvert» est entrée dans la pratique quotidienne. A l'«Hospital for Sick Children» de Toronto, cette méthode est utilisée essentiellement pour la correction de (a) défauts du septum auriculaire d'origine secondaire, et (b) la sténose des valvules des artères pulmonaires.

Cette communication s'appuie sur une série de 100 cas de fermeture de la communication inter-auriculaire, et de 60 cas de correction de la sténose des valvules pulmonaires.

On décrit les aspects anatomiques, physio-pathologiques et pronostiques de ces deux types de lésions cardiaques congénitales.

On discute ensuite de la conduite de l'anesthésie, y compris la préparation pré-opératoire, l'induction, le maintien et le contrôle.

On expose les méthodes utilisées pour refroidir et réchauffer les patients, ainsi que les complications de l'hypothermie.

On décrit et discute les problèmes qui se posent avant, pendant et après la période occlusive.

On expose enfin en détail les complications et les résultats de la correction de ces cas de lésions congénitales cardiaques.

REFERENCES

1. Conn, A. W., Allan, D., & Junkin, C. I. Anaesthesia with Hypothermia for Closure of Atrial Septal Defects in Children. Canad. Anaesth. Soc. J. *6*: 327–336. (1959).
2. Conn, A. W., & Millar, R. A. Post Occlusion Hypertension and Plasma Catecholamine Levels. Canad. Anaesth. Soc. J. *7*: 443–446 (1960).