ANAESTHESIA WITH HYPOTHERMIA FOR CLOSURE OF ATRIAL SEPTAL DEFECTS IN CHILDREN

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THE ATRIAL SEPTAL DEFECT is a lesion which can now be operated on with great success. It has a high incidence in congenital heart disease forming 17 per cent of one large series (1), and 7 per cent of a large paediatric group (2).

The lesion arises from embryological failure of apposition of parts of the intratrial septum. There are four main types of intratrial openings, each with many sub-groups, and any combination may occur (3). These may be described as follows:

(*a*) Probe-patent foramen ovale. this opening is usually closed functionally but may have an inadequate valve.

(*b*) Ostium secundum defect: an opening is present usually in the central portion of the septum, a common variant is the "high" secundum defect which occurs close to the superior vena cava and is accompanied by partial anomalous pulmonary venous drainage (4).

(*c*) Ostium primum defect: an opening is present in the lower portion of the atrial septum with the lower rim formed by the junction of mitral and tricuspid valves (usually abnormal).

(*d*) Atrio-ventricularis communis defect: this defect is similar to ostium primum defect but is accompanied by failure of fusion of the valve tissue and an underlying ventricular septal defect.

The first two groups are the only lesions suitable for direct closure under hypothermia. The time limitation imposed by hypothermia is approximately 8 minutes at 30°C, and the second two groups usually require a longer period.

PATHOLOGICAL PHYSIOLOGY

The atrial septal defect (ASD) produces its deleterious effects by altering the normal circulatory haemodynamics. With an intact atrial septum, the left atrial pressure is 5 mm. Hg higher than right atrial pressure (5). With a small ASD there is a pressure gradient of approximately 3 mm Hg producing a left to right shunt. With a larger defect (2 sq. cm. or greater) the pressure difference is practically abolished and the two atria function as one. With equal filling pressure the right ventricle receives considerably more blood than the left ventricle owing to its greater distensibility in diastole. The flow through the pulmonary circulation may be two to four times that of the systemic circulation, but the right ventricle adapts well to such volume loads in contrast to the left ventricle (6). Usually the pulmonary pressure remains normal or only slightly elevated for many years, illustrating the capaciousness of the pulmonary vascular bed.

Ultimately, right ventricular failure occurs, either directly, or secondary to pulmonary vascular changes producing pulmonary hypertension. As the ventricle

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fails, the atrial shunt may reverse and produce cyanosis Atrial fibrillation develops in approximately 10 per cent of cases (8).

Prognosis

Deaths from this lesion are very rare in infancy (1, 7). Serious symptoms or signs are rare at age 20, but gross cardiac enlargement occurs in half the cases by the age of 40 (8). Campbell *et al.* (9) report that 95 per cent are doing well at age 20, and 85 per cent are doing well at age 30, but from then on deterioration is rapid. The average age at death from uncomplicated ASD is 49 years (10). The great variability in the natural history of this disease makes it impossible to predict which patients are destined for longevity and which will develop pulmonary hypertension (11). The only contra-indication to operation may be those cases with moderate to high pulmonary hypertension associated with a high diastolic pressure and normal or slightly elevated pulmonary flow. Most patients under 45 years of age should have surgery undertaken as soon as the diagnosis is made (12, 13). The age group 2 to 10 years carries the minimum risk.

Anaesthesia

The first successful closure of an atrial septal defect using direct vision, with the aid of hypothermia, was reported by Lewis in 1953 (14). Since that time this method has become increasingly popular because it is simpler than using a cardiac by-pass, more effective than "blind" procedures, and is very safe (1, 12, 15–18). This paper deals with 63 paediatric cases which were diagnosed as having uncomplicated atrial septal defects of the secundum type and came to operation. The ratio of males to females was 23 to 40 which tends to confirm a slightly increased incidence in females. The ages range from 4 to 16 years and are subdivided as follows:

	4–7 years	*8–11 years*	*12–16 years*
Male	7	11	5
Female	18	15	7

Preoperative Visit

The patient must be thoroughly examined preoperatively by the anaesthetist. Any complicating illness results in cancellation of the operation because the procedure is elective in children. The cardiovascular system is examined in detail. A precordial "heave" due to right ventricular hypertrophy and a "thrill" in the pulmonary area may be palpated. An e ection-type systolic murmur in the second left interspace, due to relative pulmonary valvular stenosis with an accentuated, widely split pulmonary second sound, confirms the presence of increased pulmonary flow. Jugular venous distension or hepatomegaly due to right ventricular failure must be ruled out. The electrocardiogram reveals right axis deviation, right ventricular hypertrophy, and right bundle branch block. An X-ray photograph of the chest shows a prominent pulmonary artery bulge and increased vascular markings. A point of importance is the haemoglobin level, which must be over 10 gm. per cent as considerable blood loss can occur.

Premedication consists of meperidine (0.75 mg./lb.) and atropine (.012 mg /lb.). Morphine is used in older patients in a dosage of 1 mg./10 lb. body weight. Sedation is minimal, as apprehension is rarely a prominent feature and drug depression must be avoided.

Induction

The patient is carried into the operating room while a cheerful conversation is maintained to provide distraction. Using 2½ per cent thiopentone, a "sleep" dose is administered very slowly intravenously, followed by succinylcholine (1 mg./3 lb.). With the onset of complete relaxation and following adequate ventilation, the larynx is sprayed with 5 per cent hexylcaine. Unhurriedly, the patient is intubated using a Portex Magill tube, and its position is checked by stethoscope. The patient is artificially ventilated with a nitrous oxide–oxygen mixture (60:40) until spontaneous respirations return, and then Halothane is added. An 18-gauge needle with stylet is inserted in the right saphenous vein at the ankle to serve as a "spare" entrance to a vein. A cut-down is started in the left saphenous vein and a blood pump administration set[2] is connected. Intravenous extension tubing[3] is used to allow the blood pump to remain at the head of the table. Thus, fluid balance is easily controlled by the anaesthetist. After the various monitoring devices are connected, cooling is started.

Maintenance

The key-note of cardiovascular anaesthesia is the maintenance of the lightest possible plane at all times. Early in this series, nitrous oxide–oxygen anaesthesia was enhanced by "light" ether or cyclopropane during cooling. At the suggestion of one author (D A.) Halothane was substituted and it now used throughout the operation, replacing nitrous oxide/oxygen/relaxant combinations. However, a syringe connected to the intravenous tubing and containing succinylcholine (1 mg./10 lb./cc.) is retained for occasional use. Owing to the presence of electrical apparatus and the use of the cautery, a non-explosive anaesthetic is necessary. Halothane is administered using a Fluotec Vaporizer, and it has been found that adequate anaesthesia with controlled respirations and minimal cardiac depression can be obtained with concentrations of 0.5 to 1.0 per cent. A spring-loaded expiratory valve with a unidirectional valve added is situated adjacent to the endotracheal tube, producing a non-return system but requiring high gaseous flow.

Respiratory control is instituted as soon as the incision is made, and hyperventilation is maintained until the chest is closed. This method produces adequate oxygenation, avoids carbon dioxide retention, enables a lighter plane of anaesthesia to be maintained, and inhibits increased ventricular irritability (19). To follow the positive pressures in the airway, the suction end of a Magill Endotracheal Connector is connected to a gauge calibrated in centimetres of water.

[2]Abbott Laboratories, North Chicago, Ill.

[3]Baxter Laboratories Incorporated, Moreton Grove, Ill.

In keeping with the pharmacological principle of titration, drugs are administered in repeated small doses until the desired effect is produced, or side-effects result. For example, atropine is given intravenously in doses of 0.3 mg. when bradycardia occurs and Prostigmine in doses of 0.1 mg. when tachycardia occurs.

Blood loss is carefully calculated and accurate replacement is made. Blood-soaked sponges are weighed, blood loss through suction is measured using a calibrated glass flask fastened to intravenous pole (kept at eye level), and blood loss on drapes is estimated. Both loss and replacement are charted on the operating room blackboard.

Following closure of the defect, a good nitrous oxide anaesthetic has been established and other agents are no longer required.

With the incision closed, hyperventilation is discontinued and spontaneous respiration commences. When the oesophageal temperature is 33°C., nitrous oxide is discontinued and consciousness returns promptly.

The patient is transferred to the recovery room where the anaesthetist has complete charge of the patient for the initial four hours. The anaesthetist must *not* be a mere technician but a physician who accepts responsibility for all his actions. Only thus does he become a colleague of equal status on a team. The stage may be set in the operating room for many of the immediate postoperative complications, such as, shock from inadequate replacement, cardiac failure from overload, atelectasis from inadequate bronchial suction or ventilation, and pneumothorax or haemothorax.

Monitoring

A cardinal principle of cardiovascular anaesthesia is to ascertain the patient's reaction to stress in every possible way. An oesophageal stethoscope is inserted immediately after induction to follow cardiac rate and rhythm as well as air entry. Blood pressure is taken with a Collens Oscillometer, which enables both systolic and diastolic pressures to be determined. Even with this sensitive instrument, it may be difficult to determine the blood pressure due to peripheral vasospasm and low stroke volumes; the result of hypovolemia and/or hypothermia. Palpation of the carotid vessels and the loudness of the heart sounds provide a temporary index of the patient's condition until the blood pressure can be determined.

A Sanborn (150 M. series) six-channel recorder with a four-channel visoscope is used. The electrocardiogram is used to determine cardiac rate and any type of arrhythmia which may occur. Needle electrodes are used to avoid constricting bands on the extremities. The electroencephalogram is also followed, but interpretation is more difficult. E.E.G. tracings are completely *flattened* within 15 to 20 seconds following application of the caval tourniquets. Tracings return to their previous pattern within 2 to 4 minutes after release of tourniquets if the circulation is adequate. Transducers are immediately available for direct pressure recordings when desired. Dye studies with Coomassie Blue[4] using an oxygen cuvette and a Waters Oximeter Control Box can be set up to check on the closure of the defect on the table.

HYPOTHERMIA

Cooling

The patient is placed on the operating room table on a Thermo-O-Rite[5] blanket covered by a cotton sheet. After induction of anaesthesia, ice bags are placed alongside the patient, and over the axillary and inguinal areas. The refrigerant (at a temperature of 32°F.) is then circulated and the patient enclosed in the mattress. Ice is placed on the skin of patients over 75 lb. in weight, but only in small amounts. The use of ice and ice baths in infants has been discontinued because of the difficulty in reversing the rapid falls in tem-

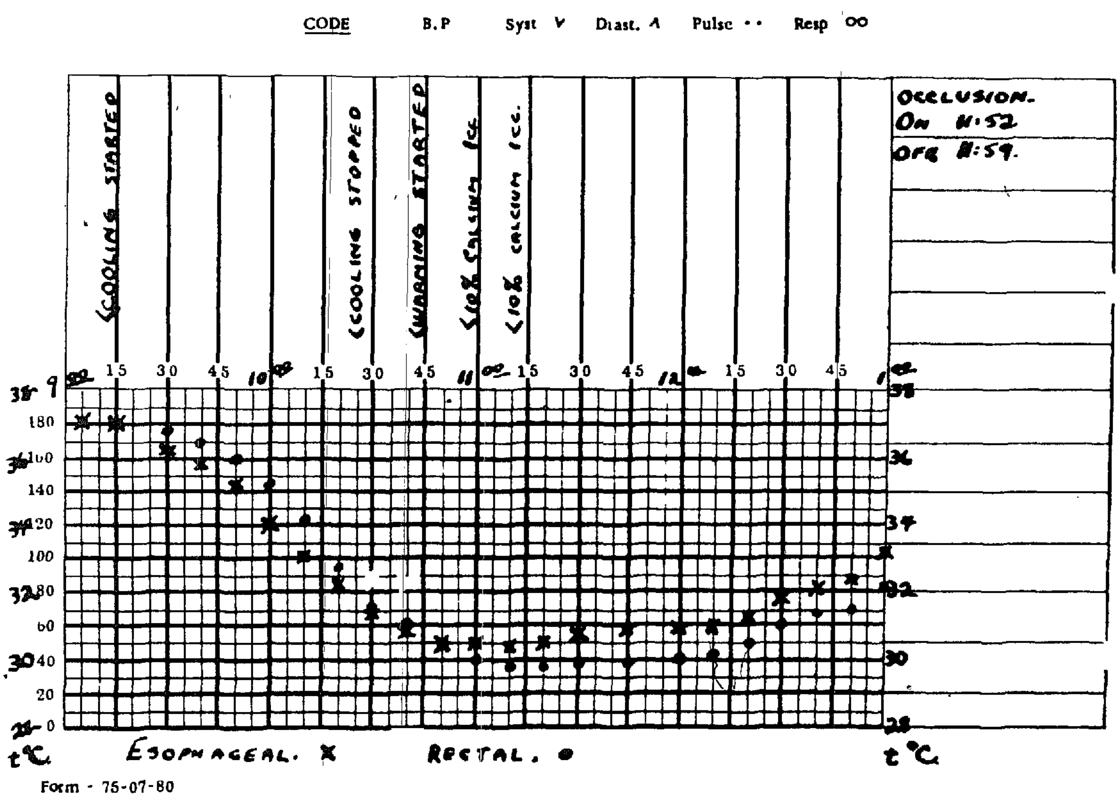

FIGURE 1

peratures that ensue. As an example, one infant, removed from an ice bath at 35°C., cooled to 28°C. despite all efforts at rewarming. At temperatures below 33°C., movement of the patient should be minimized to avoid undue stimulation of a hypothermic heart. Both oesophageal and rectal temperatures are taken and recorded. It is unfortunate that no simple, inexpensive, automatic recording device is available (20). An actual temperature chart is illustrated in Figure 1.

[4]Courtesy of Ayerst, McKenna and Harrison, Limited, Montreal, P.Q.

[5]Thermo-O-Rite Company, Limited, Buffalo, N.Y.

Drift

Drift depends on (i) the size of the patient, (ii) degree of muscle tone, (iii) the amount of peripheral vasodilation, and (iv) ambient temperature and humidity (21). Excessive subcutaneous fat appears to be an important factor as it hinders rewarming by providing a layer of insulation. Considerable individual variation exists and the best indication of subsequent temperatures is the slope of the temperature curve As the slope steepens, cooling should cease at higher temperatures because the drift will be greater. It appears wiser to cool a patient more slowly and thus retain more control over the temperature.

Closure was performed in our early cases at rectal temperatures of 28° to 29°C. The oesophageal temperatures were likely much lower, and ventricular fibrillation was common. With experience and shortened occlusion periods, rectal temperatures of 31° ± 1°C. or oesophageal temperatures of 30° ± 1°C. are perfectly adequate and allow time for unforseen difficulties.

Rewarming

Rewarming is instituted according to the temperature and the stage of surgery. The latest model of Thermo-O-Rite machine has one solution at 32°F. and a second separate solution at 105°F. By turning a valve one can alternate the circulating fluid as desired. Formerly, the rewarming solution was kept at 110°F., but one patient received a slight first degree burn. Rewarming is hastened by the use of hot water bottles. When the temperature reaches 33°C., the patient is awakened and returned to the recovery room.

Complications

The serious complication of ventricular fibrillation is related to the increased irritability of the heart due to hypothermia. This complication occurred in eleven patients and invariably happened immediately after the occlusion. "The asphyxiated, manipulated, cold abnormal heart is particularly prone to enter ventricular fibrillation at a time when an attempt is being made to restore the circulation" (19). It is obvious that numerous factors summate to trigger ventricular fibrillation in a predisposed individual.

An increased bleeding tendency was present in only one patient in whom heparinized blood was not immediately neutralized. Disturbances of acid-base balance akin to "acute acidotic syndrome" (21) have not occurred. Gross shivering is only seen in children over the age of puberty, but increased muscular tone is constant. Mild degrees of metabolic acidosis probably occur even with short occlusion, moderate hypothermia, and light anaesthesia with hyperventilation. Children can apparently compensate for mild degrees of acidosis, but unfortunately no complete biochemical studies have been undertaken in this series.

Occlusion

Before Occlusion

Immediately preceding occlusion of the heart, the anaesthetist has certain responsibilities. (*a*) Blood loss is balanced, using citrated blood. Then, heparinized blood is used to replace blood lost during occlusion. Heparinized blood is preferable to citrated owing to the rapidity of replacement sometimes required, and the difficulty of accurately balancing the citrate with calcium to avoid citrate intoxication. Citrated blood may also be a factor in causing ventricular fibrillation (20) (a minimum of 500 cc. must be on hand in the operating room at this stage). The heparin content of blood used during the occlusion is calculated and neutralized by an equivalent dose of Polybrene.[6] (*b*) A small amount of succinylcholine is injected to reduce diaphragmatic movement during the closure. (*c*) For 30 seconds preceding the occlusion 100 per cent oxygen is administered. (*d*) An ether hook is placed at the edge of the incision and connected to a special carbon dioxide cylinder unrelated to the anaesthetic. A flow of 6 L./min. is used to form a "blanket" of carbon dioxide over the atrial defect. Carbon dioxide is more soluble in blood than air, and it is hoped that by this method carbon dioxide will displace air that inadvertently enters the left atrium. (*e*) The anaesthetist times the duration of application of the caval tourniquets. The average time in our series was 4.6 minutes with a range of 2½ to 7½ minutes. At the end of 5 minutes, the time is called so that the surgeon can decide whether sufficient time remains to complete the procedure. Although the necessity has not arisen, the repair can be done in multiple stages as suggested by Johnson (21).

During Occlusion

Gas flow is cut off and the lungs allowed to collapse during occlusion. When the final suture in the defect is being tightened, the lungs are inflated, forcing out blood which fills the left atrium, and displacing air. If ventilation occurs before this time, the operative site is hidden by blood.

In a child the average blood loss due to emptying of the heart chambers and adjacent vascular beds approximates 200 to 300 cc. This volume is lost in the first 2 minutes of occlusion and should be immediately replaced. If more blood than this is suctioned, then the possibilities of a loose caval tourniquet, a "missed" left superior vena cava, or distortion of the heart with back flow through the aortic valve must be considered. Corrective measures have to be instituted immediately to avoid massive blood loss. It should be obvious that the presence of two or more anaesthetists is obligatory during this stage.

After Occlusion

Commencing within 1 minute of the release of the caval tourniquets there is a rapid rise of systolic and diastolic blood pressures, occasionally reaching levels of 230/130 within 5 minutes. During the subsequent 20 to 40 minutes a gradual fall in the blood pressure occurs until it reaches normal levels. The cause of this hypertensive phase is not certain. The characteristics of the blood pressure changes suggest a sympathomimetic source. Some rise in blood pressure usually occurs, but *not* if marked hypovolemia is present, if an associated pulmonary stenosis exists, or if the heart is beating inadequately for any reason.

Blood loss is again calculated and oxygen is administered until fluid balance is

[6]Supplied by Abbott Laboratories, Montreal, P.Q.

restored. Nitrous oxide anaesthesia is maintained until surgery is completed. Suction is applied to chest drains during closure of the thorax so that no pools of blood accumulate.

Complications

The most serious complications of closure of atrial septal defects under hypothermia occur in conjunction with the occlusion period.

Air emboli Two patients had air bubbles evident in the coronary vessels after occlusion and both hearts fibrillated and required massage. Two others with cerebral disturbances postoperatively were probably due to air emboli Recovery was complete in all cases. To avoid this complication, carbon dioxide is used, the lungs are inflated for the final suture, and the table is tilted so the defect is at the uppermost part of the left atrium.

Ventricular fibrillation. This complication occurred in eleven patients. Two were associated with air emboli, already mentioned, two with marked blood loss, one with the use of citrated blood for replacement, and one with inferior vena caval obstruction. In the other five patients, the causes were not evident, but there was no direct relation to duration of occlusion All recovered completely.

Blood loss. Blood loss exceeded 1,000 cc. in five patients for reasons already discussed. Successful replacement was accomplished.

Anatomical difficulties. (*a*) *Diagnostic error*: two patients had an ASD with partially anomalous venous return—closure was successfully accomplished in 6 and 5 minutes respectively; one patient had an ostium primum defect and was closed successfully in 7½ minutes. (*b*) *Multiple defects*: two patients had three defects each, requiring 4 and 7½ minutes respectively and one had two defects requiring 5½ minutes. (*c*) *Dextro-position*: one patient with dextro-position of the heart was technically difficult and required 7½ minutes to repair. (*d*) *Large defect*: in one patient early in the series, digital exploration of the right atrium revealed no lower rim; a repair was not attempted but one year later closure of a secundum defect was successfully accomplished using the cardiac by-pass method; subsequent to this case, all ASD's were visualized and then closed. (*e*). *Incomplete closure*: in only one patient has an incomplete closure been discovered.

Severe arrhythmias occurred in two patients. One patient developed auricular tachycardia 25 minutes after induction while at a temperature of 37.4°C. The heart rate exceeded 260 per minute and the blood pressure fell to 40/0. Deep cyanosis occurred despite full oxygenation, owing to a reversal of the atrial shunt. The tachycardia gradually responded to five doses of 0.1 mg. of Prostigmine over a period of 20 minutes. Operation was cancelled. This patient had had a "blind" Sondergaard procedure two years previously with incomplete closure.

A second patient had a transient period of auricular fibrillation with a ventricular rate of only 40, but recovered promptly. Transient arrhythmias of all types may occur but are especially common when the cavaltapes are placed in position or when the heart is manipulated.

Postoperative complications consisted of cerebral disturbance, in two patients, one lasting 10 days and the other a few hours. One patient developed a small hydropneumothorax, and another developed a mild pneumonia. A late complication occurred in a patient who developed a staphylococcal pericarditis one month postoperatively.

SUMMARY

This paper deals with a total of 63 cases of atrial septal defect with which one author (A.W C) was associated. All patients were diagnosed preoperatively as atrial septal defect of the secundum type and surgery was undertaken for direct closure of the defects with the aid of hypothermia. No deaths occurred in the series. The physiopathological disturbances, anaesthetic and hypothermic techniques, and complications are discussed.

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RÉSUMÉ

Ce travail contient l'etude de 63 cas de communications interauriculaires auxquels l'auteur prit une part active. Tous les cas furent diagnostiqués préoperativement comme étant des communications interauriculaires de type secondaire et furent dirigés vers la chirurgée pour une fermeture directe de la communication avec l'aide de l'hypothermie. Il n'y a eu aucun décès dans cette série Les effets physio-pathologiques de la communication, la technique anesthésique, l'hypothermie et les complications sont discutées.

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