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SURGICAL CLOSURE OF ATRIAL SEPTAL DEFECT: THE RESPONSE IN A PATIENT WITH SEVERE PULMONARY HYPERTENSION

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Atrial septal defect is one of the common congenital malformations of the heart. The lesion usually occurs in two locations depending upon the nature of the embryologic arrest in the formation of the septum. The most frequent location is in the area of the foramen ovale, thus a central location; less commonly the defect lies low in the septal wall in close proximity to the atrioventricular valves. Occasionally there are multiple openings; and in addition, frequently are associated anomalies of the pulmonary venous return. When the septal defect occurs low, immediately above the atrioventricular valves, the technical difficulties of closure are greater than when the defect occurs higher in the septum. Regardless of location the defect gives rise to a left-to-right shunt of blood which results in an elevated pulmonary blood flow. Depending upon the magnitude of this pulmonary blood flow varying degrees of enlargement of the right atrium, right ventricle and pulmonary vasculature result. Correspondingly the natural history of patients with this defect shows wide variation in symptomatology and in their life span.

It is recognized that many patients with smaller defects and lesser magnitudes of pulmonary blood flow may lead relatively normal lives and live to middle age or beyond. Large defects may result in pulmonary flows of such a magnitude that congestive failure develops early in life. In other instances the development of pulmonary hypertension is the limiting factor as regards the natural history of patients with this defect.

The factors leading to the development of pulmonary hypertension are not clearly understood, but may include the volume of pulmonary flow and the development of obliterative pulmonary vascular changes.

Therefore, since this lesion is common and frequently results in disability and premature death it constitutes an important challenge for the surgeon to develop safe and adequate technics for its closure.

SURGICAL BACKGROUND

The experimental approach to the solution of the problem of adequate closure of atrial septal defects was initiated by Cohn[3] in 1947. Since that time many

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ingenious but indirect technics have been evolved to accomplish this purpose. These recently have been reviewed by Bailey and associates[1] and by Swan.[5]

Experience in this institution with several types of indirect technics for the closure of atrial defects led to the realization that, despite good results in the experimental laboratory, when applied to very large defects found in the clinical patient, indirect methods did not afford complete closure of the defect. Accordingly, we have recently turned to the method first described by Lewis and Taufic[4] of direct vision closure with the aid of hypothermia and cardiac inflow occlusion. The technical aspects of this procedure have recently been reported by Swan and associates.[6]

Briefly, the body temperature of the anesthetized patient is lowered to approximately 30 C. in a tub of ice water. The patient is then removed to the operating room with a continued fall in body temperature to the neighborhood of 24 C. An anterior transverse sternum splitting bilateral thoracotomy is then done. At this temperature it is considered safe to totally occlude circulation for periods up to 10 minutes. The right auricle is then opened and the septal defect closed under direct vision with interrupted silk sutures. It is of the utmost importance that measures be taken to prevent coronary air embolism at the time of closure of the right atrium.

CASE REPORT

G. H., a 26 year old white woman had experienced ease of fatigue and exertional dyspnea since early childhood. A cardiac murmur was first detected at 5 years of age and bed rest was instituted for one year. Dyspnea and fatigue progressed until at the age of 15 years the exercise tolerance was limited to three blocks. At age 20 years a diagnosis of congenital heart disease was made and the patient was advised to avoid exertion and pregnancy. At age 26 years the patient was unable to walk one block without discomfort, and was forced to sleep with three pillows because of shortness of breath.

The physical examination revealed enlargement of the heart to the left anterior axillary line. There was an over active precordium with a forceful cardiac impulse palpable in the left third and fourth intercostal spaces between the midsternal and midclavicular lines. A systolic thrust followed by a shock was palpable in the left second intercostal space. Auscultation revealed a marked increase in the intensity of the second heart sound in the left second interspace with fine reduplication. A grade III systolic murmur was present along the left sternal border with maximum intensity in the left third intercostal space. This murmur was high pitched and rough in quality and was transmitted toward the apex. The blood pressure in the arm was 105/75 mm. Hg. The lungs were clear and the liver was not enlarged. There was no cyanosis or clubbing, and the femoral arterial pulsations were normal.

The electrocardiogram (fig. 1) demonstrated an r s R′ pattern in precordial position V-1. The R′ wave was 18 mm. in amplitude and showed a delayed intrinsicoid deflection time of .05 seconds. The total QRS duration was .10 seconds. There were prominent upright P waves in the right sided precordial leads. The tracing was interpreted as indicating right ventricular hypertrophy with incomplete right bundle branch block and probable right atrial enlargement.

The fluoroscopic examination showed a considerable increase in the vascularity of the lung fields. The main pulmonary artery and the left and right pulmonary arteries were greatly increased in size and showed significant increase in the amplitude of pulsations. The right atrium was greatly enlarged. The ventricular area was considerably enlarged with the configuration suggesting right ventricular enlargement (fig. 2).

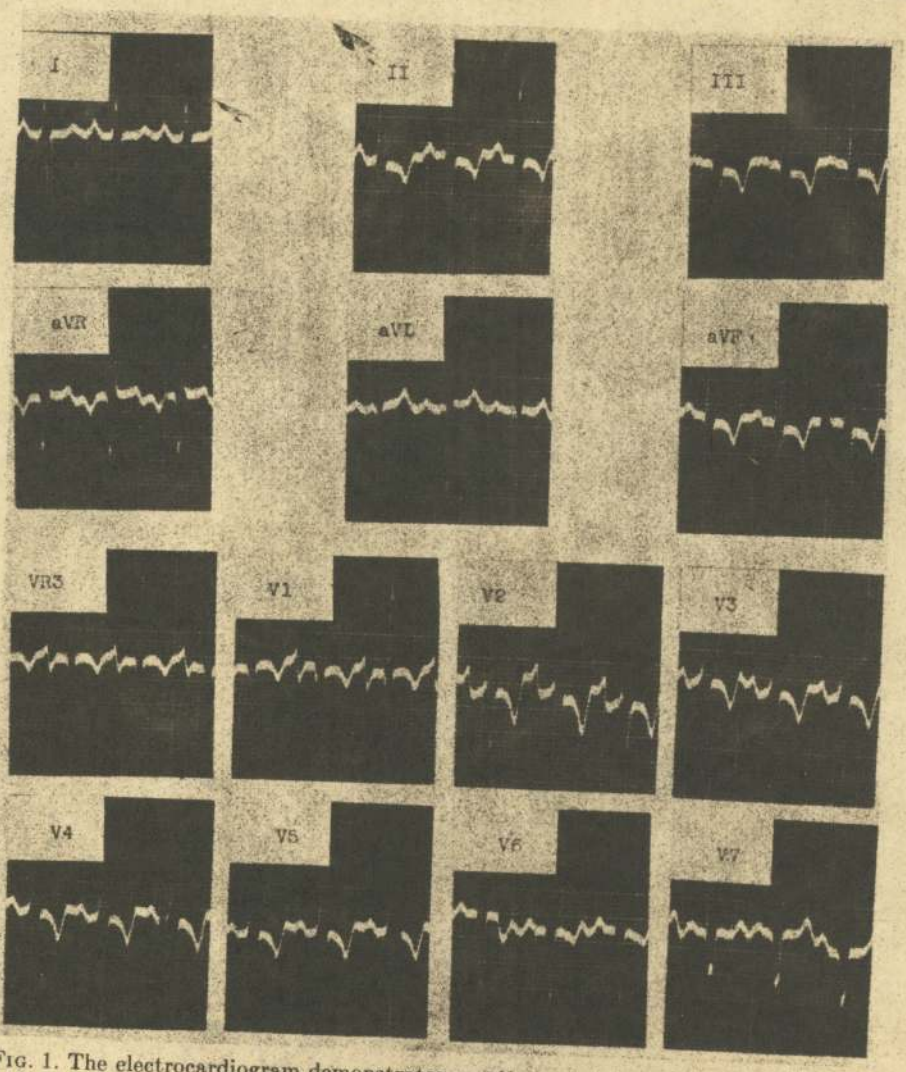

FIG. 1. The electrocardiogram demonstrates a pattern of right ventricular hypertrophy with incomplete right bundle branch block, and right atrial enlargement.

Cardiac catheterization was done via a left basilic vein on March 5, 1953. The catheter pursued an abnormal course indicating the presence of a persistent left superior vena cava entering the right atrium through the coronary sinus. The catheter was advanced into the right ventricle and into the left atrium and left ventricle. Blood samples indicated no anomalous pulmonary venous drainage into this persistent left superior vena cava. A second cardiac catheterization was done on April 14, using the right basilic venous system and the results are shown in table I. The pulmonary artery was intubated without difficulty and the catheter was again passed into the left atrium and thence into a pulmonary vein. The study indicated the presence of a left to right shunt at the atrial level with a pulmonary flow of 8.9 liters/min./M^2. There was a marked elevation of the pulmonary arterial pressure to average levels of 100/30 mm. Hg (table I).

On April 15 a thoracotomy was done using a transternal approach under hypothermia at

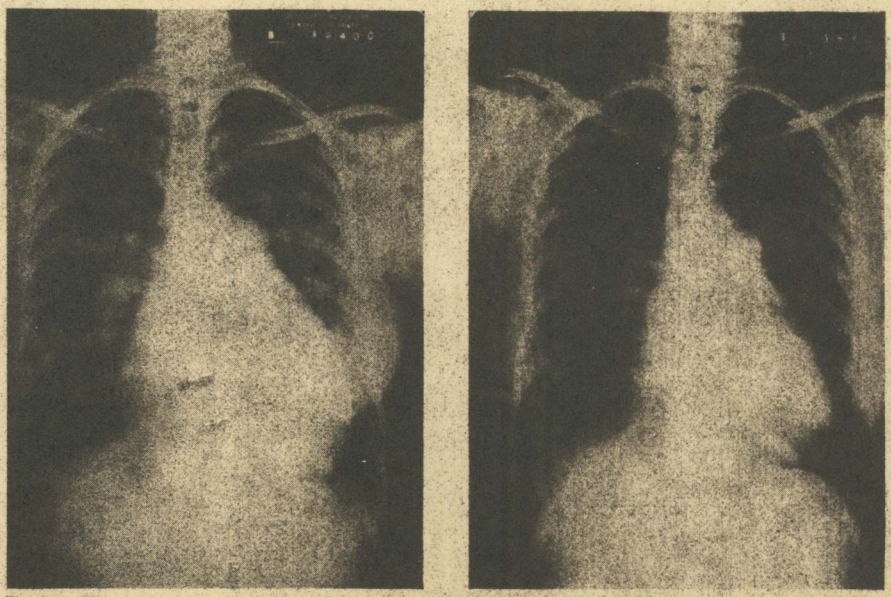

FIG. 2. The preoperative chest roentgenogram is shown on the left and four months postoperative on the right. Decrease in heart size and decreased vascularity of the lung fields is apparent.

21.5 C. Marked enlargement of the pulmonary artery and right atrium was apparent on exposure of the heart. The vena cavae were occluded, the lateral wall of the right atrium was clamped and an incision was made. The right atrium was opened, and a 2.5 by 4.0 cm. atrial defect was closed and circulation re-established after occlusion of the circulation for 7.5 minutes. Following closure of the chest wall and warming to normal body temperature shock presented which responded to blood transfusion and pressor agents.

Postoperatively a rapidly changing succession of supraventricular cardiac rhythms occurred. The postoperative course was further complicated by a transient partial aphasia which disappeared within 48 hours. The patient was discharged on May 18, 1953.

TABLE I

Preoperative physiologic studies

Catheter Position	Pressure Systolic/ Diastolic mm. Hg	Oxygen Content cc./liter	Oxygen Saturation Per cent
Superior vena cava	—	104.0	54.9
Inferior vena cava	—	124.8	65.9
Right atrium*	11/5	161.3	85.1
Right ventricle*	100/8	164.1	86.5
Pulmonary artery*	100/30	163.8	86.4
Left atrium	14/5	177.1	93.4
Pulmonary vein	15/4	178.6	94.3
Pulmonary capillary	12 (mean)	—	—
Brachial artery	113/84	179.5	94.7

Oxygen consumption: 203 cc./min. Systemic blood flow: 2.3 l./min./M^2. Pulmonary blood flow: 8.9 l./min./M^2.

* Average of two blood samples.

TABLE II

Postoperative physiologic studies

Catheter Position	Pressure Systolic/ Diastolic mm. Hg	Oxygen Content cc./liter	Oxygen Saturation Per cent
Superior vena cava	—	103.9	59.1
Right atrium*	2/1	122.3	69.5
Right ventricle*	30/0	124.8	71.0
Pulmonary artery*	30/17	124.6	69.8
Pulmonary capillary*	9 (mean)	—	—
Brachial artery	104/63	168.9	96.0

Oxygen consumption: 204 cc./min. Systemic and pulmonary blood flow: 3.0 l./min./M^2.
* Average of two blood samples.

Physical activities were progressively increased so that within four months the patient could walk a distance of half a mile without discomfort and was able to attend and participate in dances. The patient has been able to sleep comfortably with one pillow. There has been a 12 pound weight gain in the four months since discharge from the hospital.

Physical examination at four months revealed definite decrease in the heart size with no palpable thrusts or shocks. On auscultation the second heart sound in the left second interspace was reduplicated and only slightly increased in intensity. No murmurs were present along the left sternal border. A grade I systolic murmur was audible at the apex. The electrocardiogram demonstrated atrial flutter with a varying ventricular response.

Fluoroscopic examination revealed a decrease in the vascularity of the lung fields. A marked decrease in the amplitude of pulsations in the pulmonary arteries was noted as compared to those present preoperatively. The right atrium and right ventricle remained enlarged but showed a striking decrease in the size since surgery (fig. 2).

Cardiac catheterization done on Sept. 10, 1953 (table II), revealed a normal oxygen saturation of the mixed venous blood at the pulmonary arterial level of 69.8 per cent indicating an absence of a left to right shunt. There was a dramatic decrease in the pulmonary arterial pressure to average levels of 30/17 mm. Hg.

DISCUSSION

The patient discussed in this paper was a 26 year old woman with a large atrial septal defect and pulmonary hypertension. The physiologic data presented revealed that the defect was completely closed at the time of operation. Thus, this case demonstrated the feasibility of complete closure under direct vision of large atrial septal defects despite the very considerable enlargement of the heart, the age of the patient, and the significant pulmonary hypertension.

Now that it has been established that such defects can successfully be closed it is of the utmost importance that accurate clinical evaluation of these patients be pursued. The clinical diagnosis of atrial septal defect has been discussed in the past by several authors.[1, 2] Once the diagnosis has been established the problem of the selection of suitable candidates for surgical correction arises. The great variability in the natural history of patients with this defect is well known. Thus, at this stage in the development of the operative procedure it would be of great help could one predict those patients who will enjoy relative longevity as compared to those who will develop complications leading to serious disability early in their lives. However, a review of the studies of our patients with atrial

septal defects reveals that such a prediction is difficult, if not impossible to accomplish.

Early in the history of any operative technic it is common to wait until the patient is actually a very poor risk for the operative procedure before advising surgical intervention. The indications for operation are changed as experience grows until finally patients with relatively few or early symptoms are suggested for operation. If the operative risk can be demonstrated to be sufficiently low with this procedure this well may be the case as regards the patient with an atrial septal defect. At the present time we have successfully closed atrial septal defects by the method described in 4 patients with death in a fifth. It is considered by the authors that, in the future, atrial septal defects may be closed merely because of the presence of such a defect regardless of the clinical status of the patient. The criterion for operation will thus be similar to that in patients with a patent ductus arteriosus, namely the diagnosis of the anomaly.

CONCLUSIONS

The clinical course, the operative technic, and physiologic studies of a 26 year old woman with an atrial septal defect are presented.

The postoperative course, the physical and fluoroscopic findings and the physiologic data all indicate complete closure of the defect.

This patient demonstrates that severe pulmonary artery hypertension does not constitute a contraindication to closure of an atrial septal defect provided an increased pulmonary blood flow is present.

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