Atrial Septal Defect

Clinical and Physiologic Response to Complete Closure in Five Patients

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Complete surgical closure of an atrial septal defect was accomplished under direct vision in five patients. Postoperative catheterization studies demonstrated complete obliteration of the previously existing left-to-right shunt and a reduction of the pulmonary artery blood pressure in all patients. The clinical and hemodynamic status returned to normal limits with but few exceptions.

The ATRIAL septal defect is one of the most common, if not the commonest, of all congenital heart anomalies.\(^1\)\(^2\) The clinical features of the atrial septal defect have been evaluated more precisely in recent years so that the clinical diagnosis may now be established with reasonable accuracy.\(^3\)\(^4\) However, the differential diagnosis between the atrial septal defect and anomalies of pulmonary venous connections, as a sole defect or together with an atrial septal defect, remains most difficult if not impossible.\(^5\)

The natural history of patients with an atrial septal defect shows wide variation, and it is well recognized that in many patients this defect is compatible with relative longevity and an active life. Nevertheless, the mean duration of life in patients with this defect falls in the middle to late thirties, and many patients die during the early years of life.\(^6\)\(^7\) Large defects may lead to death in early life due to the development of congestive heart failure secondary to the great increase in right ventricular work, while in other patients the development of pulmonary artery hypertension leads to disability and finally death from cardiac decompensation.

Considering the variation in the natural history of this anomaly, and, therefore, realizing that many of these patients will benefit from closure of their atrial septal defects, an attempt was made to determine which patients might be expected to develop pulmonary hypertension. This attempt was made by an evaluation of the clinical status and the data derived from the catheterization of 25 patients considered to have an isolated atrial septal defect.

The present studies also offered the opportunity for the evaluation of certain concepts that have become associated with the atrial septal defect, in particular the presence of mitral stenosis resulting in the entity known as Lutembacher's syndrome.

Five patients were selected for surgical therapy from the group of 25 patients with established atrial septal defects. The main purpose of this paper is to present the clinical and physiologic changes following the complete closure of the atrial septal defect in these five patients.

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MATERIAL AND METHODS

The five patients presented at this time have been selected from a group of 49 patients observed during the past three years in whom a clinical diagnosis of an atrial septal defect was entertained. Twenty-five of these patients have been adequately studied by physiologic methods, verifying the presence of a left-to-right shunt at the atrial level, and thereby strongly suggesting the diagnosis of an uncomplicated atrial septal defect. From this group of 25 patients, five were selected for closure of their atrial septal defects. Cardiac catheterization was performed by the usual technic, with pressures determined by means of Statham strain gauges and a Hathaway recording apparatus. The zero reference point was 10 cm. above the patient's back in adults, and 5 cm. above the back in patients weighing less than 20 Kg. Blood gas determinations were made by the Van Slyke-Neill manometric method, and respiratory gas determinations were carried out according to the Scholander micro technic. The phonocardiogram was obtained by the use of a Cambridge cardioscope and recorded through a Tektronix pre-amplifier and a Hathaway galvanometer with a frequency response of 1200 cycles per second.

RESULTS

Physiologic Data Evaluated in an Attempt to Select Patients for Surgery

Twenty-five patients with a clinical diagnosis of an atrial septal defect have been evaluated by means of cardiac catheterization. These patients were divided into two groups on the basis of the level of their pulmonary artery blood pressure. This method of division was selected because it is considered that the development of pulmonary hypertension is the major factor leading to progressive disability and death.

The pertinent data obtained by catheterization in these two groups is depicted in table 1. Group A consists of 13 patients with a normal level of the pulmonary artery pressure, and group B is composed of 12 patients with elevated pulmonary artery blood pressure. A mean pulmonary artery pressure of 20 mm. Hg was considered as the upper limit of normal.

It is noted that little difference exists as regards age in groups A and B. In group A the mean age was 17.6 years, while in group B the mean age was 21.1 years. Moreover, the age spectrum was roughly similar in each group. In group A, the normotensive group, there were patients aged 50, 47, and 33 years, as well as younger patients; while in group B there were also older patients aged 56, 49, 30 years, as well as patients in the younger age group, namely 5, 6 and 7 years. Thus it would not appear that age, as reflecting duration of pulmonary blood flow, in itself is a major factor in the development of pulmonary hypertension. One patient in group A, 47 years of age, had a pulmonary index of 12 liters and a pulmonary artery pressure of 30/12 mm. Hg: while one patient in group B, although only 9 years of age, had a pulmonary index of but 4.8 liters and a pulmonary artery pressure of 83/56 mm. Hg. These two patients demonstrate the apparent lack of relationship between duration of flow as reflected in the age of the patient, magnitude of the flow, and the development of pulmonary hypertension.

The patients in the normotensive group revealed evidence of large left to right blood flows, with the oxygen saturation of the pulmonary artery blood samples averaging 85.5 per cent, and the mean pulmonary index being 13.2 liters. Patients comprising the hypertensive group, however, revealed considerable variation in the pulmonary artery blood oxygen saturation, with levels ranging from as low as 23 per cent to a high of 89.4 per cent. The mean saturation level was, therefore, considerably less than that of the normotensive group, being 70.9 per cent.

Table 1.—Physiologic Data on 25 Patients with an Atrial Septal Defect. Group A, 13 Patients with Normal Pulmonary Artery Pressure; Group B, 12 Patients with Pulmonary Artery Pressure Elevation.

<table>
<thead>
<tr>
<th>Group</th>
<th>Age</th>
<th>Pulm. Artery Press. mm. Hg</th>
<th>Pulm. Artery Mean Press. mm. Hg</th>
<th>Pulm. Artery Saturation %</th>
<th>Pulm. Index L./min./M.²</th>
<th>Total Pulm. Artery Press. Dynes/sec./cm.⁻²</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>17.6</td>
<td>25/10</td>
<td>15</td>
<td>85.8</td>
<td>13.2</td>
<td>122</td>
</tr>
<tr>
<td>B</td>
<td>21.1</td>
<td>63/31</td>
<td>42.0</td>
<td>70.9</td>
<td>8.0</td>
<td>770</td>
</tr>
</tbody>
</table>
pulmonary artery hypertension with a level of 138/76 mm. Hg. This 30 year old patient had been cyanotic for several years and presented in right heart failure. Necropsy examination a few weeks following catheterization demonstrated a large atrial septal defect as the sole anomaly. There was also present marked dilation of the right atrium, and great dilation and hypertrophy of the right ventricle, together with widespread pulmonary occlusive disease.

The total pulmonary artery resistance was low in the patients in group A, the mean being 122 dynes, second, cm.\(^{-5}\). The patients in the hypertensive group revealed an elevated total pulmonary artery resistance, the mean being 770 dynes, second, cm.\(^{-5}\).

**CASE REPORTS OF FIVE PATIENTS WITH CLOSURE OF AN ATRIAL DEFECT**

**Case 1**, C. C., a 6 year old white girl, was found to have a cardiac murmur shortly after birth. Growth and development were normal, and there was no limitation of the exercise tolerance at any time. From the age of 4 to 6 years there was an increased frequency of upper respiratory infections.

Physical examination revealed slight asymmetry of the thorax with prominence of the precordium. The heart was not enlarged and no thrills or shocks were palpable. The second heart sound in the second left intercostal space was widely reduplicated but was normal in intensity. A grade II systolic murmur, rough and blowing in quality, was audible along the left sternal border with maximum intensity in the second left intercostal space.

The electrocardiogram demonstrated an RsR' type of QRS complex in lead V\(_1\) with a delayed intrinsicoid deflection time. The tracing was interpreted as indicating incomplete right bundle branch block.

Fluoroscopic examination revealed a considerable increase in the vascularity of the lung fields. The main pulmonary artery and the right and left main branches were increased in size and showed an increased amplitude of pulsations. The right atrium and right ventricle were enlarged.

Cardiac catheterization studies (table 2) revealed a rise in the blood oxygen saturation at the right atrial level indicating a left-to-right shunt. There was a considerable increase in the pulmonary blood flow and a slight increase in the right ventricular pressure.

Surgical correction of the defect was performed on May 25, 1953. Ventricular fibrillation occurred during the period of reduction of body temperature and prior to the opening of the chest. The thorax was immediately opened and cardiac massage instituted.

A short period of cardiac standstill next occurred, followed by an idioventricular rhythm. The venae cavae were occluded, the right atrium opened and a large atrial septal defect closed with interrupted sutures. The postoperative course was complicated by the occurrence of atelectasis in the right lower lobe. The patient was discharged on June 8, 1953.

Three months postoperatively the patient was asymptomatic. Examination revealed a persistence of the reduplication of the second heart sound in the second left intercostal space. The systolic murmur along the left sternal border was decreased in intensity. There was no significant change in the electrocardiogram. Fluoroscopic examination showed no change in the vascularity of the lung fields or size of the pulmonary arteries. There was, however, a definite decrease in the amplitude of pulsations in the pulmonary arteries. There was also slight decrease in the size of the right atrium and right ventricle, so that the over-all heart size was within normal limits.

Postoperative cardiac catheterization was performed on Aug. 26, 1953. There was a normal oxygen saturation in the pulmonary artery with no evidence of a left-to-right shunt at the atrial level. The pulmonary artery and right ventricular pressures were slightly decreased (table 2).

**Case 2**, A. C., a 6 year old white boy, was one of nonidentical twin boys delivered without difficulty following a normal pregnancy. A cardiac murmur was detected at birth and has been present since. Frequent respiratory infections and slow weight gain occurred during the first two years of life. Since the age of 2 years the patient has been asymptomatic.

The physical examination revealed an active precordium with an apical thrust. A grade III blowing, systolic murmur was present along the left sternal border with maximum intensity in the second left intercostal space. The second heart sound in the second left intercostal space was increased in intensity and widely reduplicated. A medium pitched, soft, mid-diastolic murmur of short duration was audible along the lower left sternal border with transmission toward the apex (fig. 1).

The electrocardiogram demonstrated an incomplete right bundle branch block pattern. The fluoroscopic examination revealed an increased vascularity of the lung fields. The main pulmonary artery and the left and right pulmonary arteries were increased in size and amplitude of pulsations. The aorta was inconspicuous in all views. The right atrium and right ventricle were slightly enlarged.

Cardiac catheterization revealed a rise in blood oxygen content at the right atrial level indicating a left-to-right shunt (table 2) with a pulmonary blood flow of considerable magnitude. The right ventricular pressure was slightly elevated, whereas the pulmonary artery pressure was normal, resulting in a pattern of functional pulmonic stenosis (fig. 2).

Surgical closure of the atrial defect was accom-
### Table 2—Catheterization Studies in Five Patients before and after Closure of an Atrial Septal Defect.

<table>
<thead>
<tr>
<th>Name, Age and Sex</th>
<th>Time of Studies</th>
<th>Pressures, mm. Hg</th>
<th>Oxygen Content, Per Cent Saturation</th>
<th>Cardiac Output</th>
<th>Cardiac Index</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean</td>
<td>Mean</td>
<td>Mean</td>
<td>Mean</td>
</tr>
<tr>
<td>1. C.C. Age 6, female</td>
<td>Preop.</td>
<td>6</td>
<td>32/4</td>
<td>27/16</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>Postop. 3 mos.</td>
<td>1</td>
<td>25/2</td>
<td>20/10</td>
<td>13</td>
</tr>
<tr>
<td>2. A.C. Age 6, male</td>
<td>Preop.</td>
<td>1</td>
<td>35/3</td>
<td>20/4</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>Postop. 3 mos.</td>
<td>2</td>
<td>28/1</td>
<td>29/13</td>
<td>18</td>
</tr>
<tr>
<td>3. J.G. 4 mos., female</td>
<td>Preop.</td>
<td>3</td>
<td>26/4</td>
<td>18</td>
<td>101/11</td>
</tr>
<tr>
<td></td>
<td>Postop. 7 days</td>
<td>5</td>
<td>15/3</td>
<td>15/7</td>
<td>10</td>
</tr>
<tr>
<td>4. G.H. Age 26, female</td>
<td>Preop.</td>
<td>7</td>
<td>100/1</td>
<td>100/30</td>
<td>53</td>
</tr>
<tr>
<td></td>
<td>Postop. 4 mos.</td>
<td>1</td>
<td>30/0</td>
<td>30/17</td>
<td>21</td>
</tr>
<tr>
<td>5. E.M. Age 19, female</td>
<td>Preop.</td>
<td>1</td>
<td>56/0</td>
<td>55/12</td>
<td>27</td>
</tr>
<tr>
<td></td>
<td>Postop.</td>
<td>1</td>
<td>25/2</td>
<td>24/3</td>
<td>10</td>
</tr>
</tbody>
</table>

**Note:** All pressures are measured in mm. Hg. Values are mean pressures.
plished on June 26, 1953. The postoperative course was uneventful.

Examination three months postoperatively revealed no cardiac murmurs. The second heart sound in the left second intercostal space continued to be increased in intensity and reduplicated. There was no significant change in the electrocardiogram. Fluoroscopic examination revealed a decrease in the heart size. There was continued enlargement of the pulmonary arteries; however, the amplitude of pulsations of these vessels had decreased to normal. Recatheterization (table 2) revealed no evidence of a left-to-right shunt at the atrial level and normal pressures in the right ventricle and pulmonary arteries (fig. 2).

Case 3, J. G., a 4 year old girl, was known to have a cardiac murmur from the age of 3 months. The child developed normally and has been asymptomatic.

Physical examination revealed a normal heart size and no palpable thrills or shocks. The second heart sound in the second left intercostal space was increased in intensity and was widely reduplicated. A grade III blowing systolic murmur was audible along the left sternal border with maximum intensity in the second left intercostal space. A short medium pitched mid-diastolic murmur was audible along the lower left sternal border with transmission to the apex.

The electrocardiogram revealed a pattern of right ventricular hypertrophy. Fluoroscopic examination demonstrated increased vascularity of the lung fields. The main pulmonary artery and the right and left pulmonary arteries were increased in size and showed an increased amplitude of pulsations. The right atrium and right ventricle were enlarged.

Cardiac catheterization was performed on July 21, 1953, and revealed (table 2) a left-to-right shunt at the atrial level.

A thoracotomy was performed on Dec. 1, 1953 under hypothermia. The atrial septal defect was closed under direct vision with the aid of occlusion of the circulation for five minutes.

The postoperative course was uneventful. Results of postoperative cardiac catheterization on Dec. 8, 1953, showed no left-to-right shunt at the atrial level. Physical examination revealed that the previously described systolic and diastolic murmurs were no longer present. Fluoscopy demonstrated a decrease in the heart size to within normal limits. There was a persistence of the enlargement of the pulmonary arteries but normal pulsations were present in these vessels. There was no significant change in the electrocardiogram.

Case 4, G. H., a 26 year old, white woman, had noted limitation of exercise tolerance since childhood. A cardiac murmur was first detected when the patient was 5 years old. A progressive decrease in exercise tolerance with severe dyspnea had occurred so that by the time of the preoperative examination walking tolerance was limited to one block.

The physical examination revealed enlargement
of the heart to the left. A systolic thrust followed by a shock was palpable in the second left intercostal space. A tapping impulse was palpable along the lower left sternal border. The second heart sound in the second left intercostal space was slightly reduplicated. A grade III rough systolic murmur was audible along the left sternal border with maximum intensity in the third intercostal space.

The electrocardiogram demonstrated a pattern of right ventricular hypertrophy and partial right bundle-branch block. The fluoroscopic examination revealed an increased vascularity of the lung fields. The main pulmonary artery and the right and left main branches were considerably dilated and showed an increased amplitude of pulsations. The over-all heart size was increased with marked enlargement of the right atrium and right ventricle.

Cardiac catheterization was performed on April 14, 1953, and demonstrated a marked pulmonary artery pressure elevation and the presence of an atrial septal defect (table 2) with a left-to-right shunt at this level.

Direct surgical closure of a large atrial defect was accomplished on April 15, 1953, with hypothermia and cardiac inflow stasis. The postoperative course was complicated by the occurrence of supraventricular arrhythmias.

A considerable increase in the patient’s exercise tolerance occurred postoperatively. Four months following surgery the patient was able to walk half a mile.

Physical examination revealed a decrease in the intensity of the second heart sound in the second left intercostal space and a decrease in the intensity of the systolic murmur from grade III to grade I. There was a definite decrease in the heart size by roentgenologic examination (fig. 3). There was a decrease in the vascularity of the peripheral lung fields and a considerable decrease in the amplitude of pulsations over the main pulmonary artery and the right and left main branches.

The electrocardiographic studies postoperatively showed a decrease in the amplitude of the R wave in lead V₁ and a decrease in the depth of the S wave in lead V₆. Postoperative physiologic studies demonstrated a dramatic decrease in the previously ele-
Fig. 3. Case 4, G. H. The chest roentgenogram preoperatively (A) and four months postoperatively (B).

Fig. 4. Case 4, G. H. The preoperative pulmonary artery pressure contour is shown on the left in the lower of the two pressure tracings. A marked elevation to average levels of 100/30 mm. Hg is noted. The postoperative pulmonary artery pressure on the right shows a marked decrease in pulmonary artery pressure to average levels of 30/15 mm. Hg. The upper pressure tracing in both panels represents the brachial artery pressure.

Physical examination revealed asymmetry of the thorax with prominence of the precordium. Palpation demonstrated an overly active precordium with a thrill in the epigastrium and along the lower left sternal border. A shock was palpable in the second left intercostal space. Auscultation revealed an accentuated, widely reduplicated second heart sound in the second left intercostal space. A grade III rough blowing systolic murmur was present along the left sternal border with maximum intensity in the

vated pulmonary artery pressure (fig. 4) and showed no evidence of a left-to-right shunt at the atrial level.

Case 5, M. E., a 19 year old white woman, was thin and easily fatigued during childhood. Respiratory infections occurred frequently during childhood and adolescence, with episodes of pneumonia. A diagnosis of heart disease was made at age 10 years, when a murmur was first detected. Preoperatively the patient complained of dyspnea and ease of fatigue.
third left intercostal space. A low-pitched, short, mid-diastolic murmur was audible between the lower left sternal border and the midclavicular line.

The electrocardiogram revealed a pattern of right bundle-branch block and right ventricular hypertrophy.

Fluoroscopy showed an increased vascularity of the lung fields. The main and right and left pulmonary arteries were greatly increased in size and showed an increased amplitude of pulsations. The right atrium and right ventricle were considerably enlarged.

Cardiac catheterization data demonstrated a moderately severe pulmonary hypertension (table 2) and an increased pulmonary blood flow with a left-to-right shunt at the atrial level.

On Aug. 28, 1953, a large atrial septal defect was closed under direct vision with the aid of hypothermia and inflow and outflow stasis.

The postoperative course was complicated by recurring episodes of chest pain and fever. Cardiac catheterization studies postoperatively on Oct. 15, 1953, showed a normal pulmonary artery pressure and no evidence of a left-to-right shunt at the atrial level. Three months after surgery the patient was asymptomatic, performing all household activities, and had walked one mile without fatigue. Physical examination revealed that the previously described systolic and diastolic murmurs were no longer present. The intensity of the second heart sound in the second left intercostal space was reduced, but continued to be reduplicated. There was a decrease in the size of the right atrium and right ventricle by fluoroscopic examination. There were no significant alterations in the electrocardiogram.

**DISCUSSION**

**Selection of Patients for Closure**

Now that it has been established that atrial septal defects can be successfully closed, the problem of the selection of candidates for the procedure arises. The great variability in the natural history of patients with this defect has long been recognized. Thus at this stage in the development of the operative procedure, it would be of great value could one differentiate those patients who will enjoy relative longevity from those who will develop pulmonary artery hypertension leading to serious disability and possibly death 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. An evaluation of this group of 25 patients does not suggest that age in itself is a significant factor in the development of pulmonary hypertension. Thus, it is noted that there is no significant difference between the average ages in the two groups that were formed on the basis of the level of their pulmonary artery pressure. Also, it may be noted that the extremes of age in the two groups show no great difference.

The patients with normal pulmonary artery pressure had pulmonary artery blood flows of the greater magnitude. Thus, it would appear that neither the duration nor the magnitude of the pulmonary artery blood flow in themselves are of critical significance in the development of pulmonary artery hypertension. There are factors other than age and magnitude of pulmonary blood flow to be considered in the development of pulmonary hypertension in patients with atrial septal defects. An evaluation of these factors is beyond the scope of this paper.

The total pulmonary artery resistance was within normal limits in the patients with normal pulmonary artery blood pressure despite the presence of greatly increased pulmonary artery blood flows. This reflects the capaciousness and great distensibility of the normal pulmonary vascular bed. The group of patients with pulmonary artery hypertension, however, revealed a considerable increase in the total pulmonary artery resistance. The development of pulmonary artery hypertension with the resulting decrease in the magnitude of the pulmonary blood flow, suggests a loss of the normal distensibility of the pulmonary vascular bed and the development of occlusive changes in the smaller pulmonary arteries and arterioles. Currently there would not appear to be any recognized factor that portends the development of pulmonary hypertension in patients with atrial septal defects.

At the present time closure of atrial septal defects under direct vision is being advised in patients having a pulmonary blood flow three times the value of the systemic flow. This is purely an arbitrary level and will doubtlessly be altered as experience dictates. The presence of pulmonary hypertension does
not in itself negate the possibility of successful operative therapy. Patient G. H. is an excellent example of the dramatic benefits, both physiologic and clinical, that may result from closure of an atrial septal defect even in the presence of marked pulmonary hypertension. However, the presence of pulmonary hypertension does call for careful evaluation of the patient. When there is moderate to marked pulmonary artery hypertension with a high diastolic pressure level, associated with a normal or slightly elevated pulmonary artery blood flow then it is considered inadvisable to recommend surgical closure of the defect.

It is anticipated that with increasing experience the present criteria will be altered and the indication for surgical therapy in patients with an atrial septal defect will be the diagnosis of the uncomplicated anomaly.

Operative Technic of Closure

Experience in this institution with several types of indirect techniques for the closure of atrial defects led to the realization that, despite good results in the experimental laboratory, indirect methods did not secure complete closure of the large defects frequently encountered clinically. Accordingly, we have recently turned to the method first described by Lewis and Taufic10 of closure under direct vision with the aid of hypothermia and cardiac inflow occlusion. The technical aspects of this procedure have recently been reported by Swan and associates.11

Briefly, the body temperature is lowered to approximately 30 C. by placing the anesthetized patient 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 performed. At this temperature it is considered safe to totally occlude circulation for periods up to 10 minutes. The right atrium 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.

Hemodynamic Changes Following Closure

The striking feature of the postoperative catheterization data (table 2) is the evidence of complete closure of the atrial septal defect in all patients. The mixed venous blood samples as obtained from the venae cavae and right heart chambers revealed no significant rise at any level. Moreover, the oxygen content of the blood samples at the pulmonary arterial level was within normal limits. An inferior vena caval sample was not obtained in patient G. H. during the postoperative catheterization, thus making it impossible to accurately evaluate the significance of the oxygen content at the atrial level. The fact that the oxygen saturation of the pulmonary artery blood sample was normal compared with the elevated preoperative level indicates the absence of a left-to-right shunt.

An equally striking change was the decrease in pulmonary artery blood pressure noted postoperatively in the two patients with significant pulmonary artery hypertension. In patient G. H. the preoperative pulmonary artery pressure was 100/30, whereas the pressure four months postoperative was 30/17 (fig. 4). A significant decrease in the pulmonary artery pressure occurred postoperatively in patients J. G. and C. C. although the preoperative pulmonary artery pressures were within normal limits. This decrease in the pulmonary artery pressures indicates the presence of a normal pulmonary vasculature and suggests that the slight preoperative elevations were a result of a disproportion between the greatly elevated pulmonary blood flow and the capacity of a normal pulmonary vascular bed. The persistence of slight pulmonary artery hypertension postoperatively in patient, G. H., suggests the possibility of structural changes in the pulmonary vascular bed.

The preoperative pressure gradient recorded between the right ventricle and pulmonary artery in patient A. C. represents functional pulmonic stenosis. This phenomenon reflects a greatly elevated pulmonary artery blood flow, and does not indicate anatomic stenosis of the pulmonary valve. This interpretation of the pressures is clearly substantiated by the
postoperative pressure tracing demonstrating complete disappearance of the gradient (fig. 2). This phenomenon of functional pulmonic stenosis should be considered when entertaining a diagnosis of mild, pure pulmonic stenosis.

The total pulmonary artery resistance was moderately elevated postoperatively in three patients. This finding again suggests residual structural changes at the level of the small pulmonary arteries and arterioles.

Preoperatively the systemic blood flow was considered to be within normal limits in all five cases. This has been the usual finding in our patients with atrial septal defects, and it is not thought that these patients have decreased systemic blood flows. Postoperatively no consistent change in the systemic blood flow was observed. Preoperatively the pulmonary blood flow was significantly increased in all patients, ranging from a calculated pulmonary index of 7 liters to 40 liters. Postoperatively there was a marked reduction in the pulmonary artery blood flow to levels equalling the systemic blood flow.

The physiologic data obtained postoperatively in these five patients reveals complete closure of the atrial septal defect in all patients. The hemodynamic picture has returned to normal with the exception in some instances of possible residual structural changes in the smaller pulmonary arteries and arterioles.

Clinical Changes Following Closure

Dyspnea and ease of fatigue were prominent features of the preoperative course in patients G. H. and M. E. Postoperatively, there was great amelioration of these symptoms. The mechanisms resulting in dyspnea in patients with atrial defects are not well established. The resting systemic blood flow and the peripheral arterial oxygen saturation were within normal ranges in these patients, thus tending to minimize the role of systemic hypoxia as a significant factor in the genesis of these symptoms. A more important mechanism may be the decrease in resiliency and elasticity of the lungs of patients with large pulmonary blood flows due to the engorgement of the pulmonary vasculature. Dyspnea may thus result from the increased respiratory effort necessitated in the presence of these "stiffened" lungs, and by the continued stimulation of the Hering-Breuer reflex.

The changes in the cardiac murmurs noted postoperatively are of interest in evaluating their origin. The systolic murmur present in all patients over the pulmonic area had disappeared in two patients and had greatly diminished in intensity in the remaining three patients (fig. 1). This would tend to substantiate the opinion that this murmur arises as the result of the increased blood flow through the pulmonary artery, and is in effect the murmur of functional pulmonic stenosis. The persistence of the murmur postoperatively in three patients in all probability reflects the continued presence of turbulent blood flow in the dilated pulmonary artery and establishes the fact that this murmur is not a result of flow through the atrial septal defect itself.

The preoperative examination in three patients revealed a medium-pitched, short, mid-diastolic murmur along the lower left sternal border. This murmur disappeared postoperatively in all three patients (fig. 1). The disappearance of this murmur postoperatively strongly suggests absence of associated mitral stenosis. Palpation of a normal mitral valve orifice at surgery offers incontestible evidence of the presence of a normal mitral valve. It is emphasized, therefore, that the presence of this diastolic murmur in a patient with an atrial septal defect does not necessarily indicate the association of anatomical mitral valve stenosis. This combination of mitral stenosis and an atrial septal defect, termed Lutembacher's syndrome, is an established entity. However, the present findings emphasize the fact that this lesion is diagnosed on a clinical basis much more frequently than is justified by its rare occurrence. The basis for the murmur in question is considered to be the greatly increased blood flow through the tricuspid valve, thus representing a functional tricuspid stenosis. In support of this explanation is the location of the murmur along the lower left sternal border in contrast to the apical location of the diastolic murmur heard frequently in patients with a patent ductus arteriosus or a large ventricular septal defect in which case.
the murmur results from a functional stenosis of the mitral valve.

Reduplication of the second heart sound in the second left intercostal space persisted postoperatively. The physiologic basis for this finding has not been well established. It is significant in this regard that there was also persistence of incomplete right bundle branch block electrocardiographically. The intensity of the second heart sound was significantly decreased in the two patients who had presented evidence of pulmonary hypertension preoperatively.

Minor changes were noted on comparing the pre- and postoperative electrocardiograms in these five patients. A decrease in the amplitude of the R' wave in lead V1 and a decrease in the depth of the S wave in lead V4 occurred in patients A. C. and G. H., suggesting a decrease in the element of right ventricular hypertrophy. There was a persistence of the partial right bundle-branch block pattern in the four patients revealing this finding preoperatively. The subsequent outcome of the conduction defect in these patients may be helpful in determining the origin of this electrocardiographic abnormality.

The postoperative roentgenographic studies showed a decrease in heart size in all instances. This decrease was most prominent in the two patients who preoperatively showed the greatest increase in heart size and revealed definite evidence of pulmonary hypertension. The early postoperative decrease in heart size may be a reflection of the decrease in the total blood volume which occurs following closure of the atrial septal defect. Further decrease in heart size may be a reflection of the diminished work load of the right heart and may indicate an actual decrease in the size of the right ventricle and right atrium. In contrast to the decrease in the size of the right heart chambers the pulmonary artery remained dilated postoperatively in all patients. There was, however, a decrease to within normal limits of the previously increased amplitude of pulsation in these vessels.

Thus there has been definite evidence of the abolishment or improvement in all signs and symptoms that preoperatively were related to the great increase in the pulmonary artery blood flow.

**Summary and Conclusions**

1. Five patients are presented in whom an atrial septal defect was closed under direct vision under conditions of open heart surgery.

2. Clinical and hemodynamic studies obtained postoperatively are compared with the preoperative findings and their significance discussed.

3. Evaluation of physiologic data did not reveal significant criteria portending the development of pulmonary hypertension in patients with an atrial septal defect.

4. Indications for surgical closure in a patient with an atrial septal defect are presented.

**Acknowledgment**

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**Sumario Español**

1. Cinco pacientes se informan en los cuales un defecto del septo atrial se cerró bajo observación directa en condiciones de cirugía cardíaca abierta.

2. Los estudios clínicos y hemodinámicos postoperatorios obtenidos se comparan con los hallazgos preoperatorios y el significado se discute.

3. Evaluación de los datos fisiológicos no reveló criterios significativos presagiando el desarrollo de hipertensión pulmonar en pacientes con defecto del septo atrial.

4. Las indicaciones para el cierre quirúrgico de defecto del septo atrial se presentan.

**References**


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