Atrial Septal Defect: Factors Affecting the Surgical Mortality Rate

By Dwight C. McGoon, M.D., H. J. C. Swan, M.B., Ph.D., Robert O. Brandenburg, M.D., Daniel C. Connolly, M.D., Ph.D., and John W. Kirklin, M.D.

Analysis of accumulated experience with the repair of atrial septal defect in 119 adults has revealed several factors determinable preoperatively that appear to have a strong influence on the operative risk. These factors are related to the presence of pulmonary vascular disease or heart failure and contribute importantly to the selection of patients for operation.

Surgical treatment for atrial septal defect has been practiced for approximately 5 years. The selection of patients for operation should be based upon a number of considerations, including knowledge of the operative mortality rate and factors influencing it. This study was undertaken for the purpose of gathering and interpreting data upon these matters.

Material

The records of patients 15 years of age or older who were operated upon by us before January 1958 for repair of an atrial septal defect have been analyzed. The first such operation in this institution was performed in February 1953. Patients having partial persistent common atrioventricular canal (so-called ostium primum defect) or the complete form of this malformation were not included herein; these were reported on previously.1 Patients with associated abnormalities such as valvular pulmonary stenosis, ventricular septal defect, common atrium, or total anomalous pulmonary venous connection likewise were not included. Patients having partial anomalous venous connection of the right lung were considered to have a variant of simple atrial septal defect and were included. A total of 119 cases fulfilled these qualifications.

The notes concerning clinical findings were reviewed. Cardiac catheterization had been performed2 preoperatively at the Mayo Clinic in 83 per cent of the cases and elsewhere in an additional 4 per cent. The observations at operation had been recorded in detailed notes, which were reviewed. All deaths that occurred during operation or during the period of postoperative hospitalization were considered operative deaths.

One hundred and eight of the patients had been operated upon by the atrial-well technic.3 In 7 patients repair was made through open cardiomyotomy with extracorporeal circulation,4 in most instances because of very high right atrial pressure. Four patients were operated upon by the Bailey technic5 for miscellaneous reasons.

Results

Fourteen patients did not survive the operation or postoperative period in the hospital, for a total mortality rate of 12 per cent. In 2 of the nonsurvivors, repair was not carried out because a decrease in systemic arterial blood pressure and an increase in pulmonary arterial pressure occurred when the defect was temporarily occluded. In only 2 of the 14 cases were there obvious surgical technical factors that may have contributed significantly to the death. In 1 patient, operated upon by the atrial-well technic, there was severe postoperative hemorrhage into the thorax, probably causally related to the patient's death with hyperpyrexia and oliguria 48 hours after operation. In a second patient, the quantity of heparin administered during operation was inadequate.

The operative mortality in relation to the age of the patients is shown in table 1. The mortality rates in the various age groups are relatively uniform.

A decision as to whether or not congestive heart failure was present or had previously been present was usually made by the examining physician. It is fully realized that rigid
definition of heart failure from clinical criteria is impossible. If congestive heart failure had occurred, the mortality rate was 39 per cent whereas in its absence the mortality rate was 7 per cent (table 2).

Patients with a peak right atrial pressure of 15 mm. Hg or more, 10 in number, experienced a hospital mortality rate of 50 per cent (table 2). It was assumed that patients not catheterized had a peak right atrial pressure of less than 15 mm. Hg, an assumption which seems justified since all patients not catheterized had clinically and surgically uncomplicated septal defects and showed no evidence of heart failure or pulmonary hypertension.

The magnitude and direction of the shunt were determined in patients catheterized in our institution. For all patients not catheterized it is assumed that the right-to-left shunt was less than 10 per cent, an assumption that seems justified for the reasons already noted. This is the basis for the data concerning the magnitude of right-to-left shunt recorded in table 2. A striking difference exists between these 2 groups as regards operative mortality. In patients with a right-to-left shunt of 10 per cent or more, the hospital mortality rate was 50 per cent.

Fifty-three per cent of the patients with a systolic pressure of 75 mm. or more in the pulmonary artery were nonsurvivors, in contrast to a mortality rate of 4 per cent in patients with pulmonary arterial pressure of less than 75 mm. Hg. It was assumed that in patients not catheterized the systolic pressure in the pulmonary artery was 75 mm. or less, for the reason already noted.

In 45 patients data were available for calculations of total pulmonary resistance and total systemic resistance. The hospital mortality rate was 71 per cent for the 7 patients in whom the ratio of total pulmonary resistance to systemic resistance equaled or exceeded 0.5 (table 2). The use of this resistance ratio rather than the pulmonary resistance itself serves to minimize the effect of nonspecific factors, such as body size, and to allow a more accurate comparison of such data.

Table 3 lists individually the pertinent information on each of the 14 nonsurviving patients, which has already been assimilated in the figures presented in table 2.

**TABLE 1.—Age and Hospital Mortality Rate**

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Total patients</th>
<th>Hospital deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>15-29</td>
<td>47</td>
<td>4 9</td>
</tr>
<tr>
<td>30-39</td>
<td>34</td>
<td>4 12</td>
</tr>
<tr>
<td>40-49</td>
<td>28</td>
<td>5 18</td>
</tr>
<tr>
<td>50 or more</td>
<td>10</td>
<td>1 10</td>
</tr>
<tr>
<td>Total</td>
<td>119</td>
<td>14 12</td>
</tr>
</tbody>
</table>

*The percentage figure refers to that proportion of blood passing through the systemic circulation which is shunted blood.*

Comment

The operative mortality rate encountered in this series for the repair of an uncomplicated atrial septal defect is less than 2 per cent, a risk so favorable that it approximates that of closure of an uncomplicated patent ductus arteriosus. In any condition the advisability of surgical treatment can be deduced only after careful consideration of the comparative risks of operative and nonoperative treatment. Because of the low operative risk established in this large group of patients operated upon by the atrial-well technic, we feel justified in advising that an atrial septal defect of significant size be repaired by this method even in the asymptomatic or minimally symptomatic patient.

The evidence at present available appears to indicate that closure of atrial septal defects in patients in whom pulmonary blood flow is clearly less than systemic blood flow will almost always result in death. The same appears true for patients with patent ductus arteriosus, aortico pulmonar fistula, common atrioventricular canal, and ventricular septal defect. Such patients are not accepted by us for closure of their intracardiac defects.

Only 1 of these 119 patients, who was operated upon early in the series, was inoperable by this criterion, and he failed to survive. Thirteen additional patients failed to survive. Since actual errors of surgical tech-
nic were rare, an inquiry into the factors that appear to have increased the risk of repair of atrial septal defect in the nonsurviving but theoretically operable patients is of importance. Better treatment of patients with these factors present should reduce the risk of repair for them in the future.

Age is the one factor so studied which does not appear per se to influence operative mortality strongly (table 1). It is amply demonstrated in table 2 that each of the factors listed there has a significant influence on the risk of the operation.

Rigid rules for the selection of patients for operation are seldom wise, for individual details must be appraised in each patient. The experience reported here, however, suggests that a clinical history of congestive heart failure, a markedly elevated right atrial pressure, a large right-to-left shunt, severely elevated pressure in the pulmonary artery, and markedly increased pulmonary vascular resistance adversely affect the operative risk in the group as a whole.

Furthermore, examination of each individual patient in an effort to determine the influence of abnormal values for these 5 factors that might affect the outcome of operation has yielded information of great practical clinical value. As recorded in table 4, if none or only 1 of these unfavorable factors exists in a given patient, the risk of operation is highly acceptable. The risk is very high when 2 such factors are present, and appears extreme in this series if 3 or more coexist.

Attention must of course be given to the probability that improvement in operative management or in preparation of the patient will reduce the mortality for the various categories of patients.

Two of the 5 pertinent factors are directly related to heart failure; these are a history of congestive heart failure and a markedly elevated right atrial pressure. Also, a large right-to-left shunt is probably indirectly related in most instances to right ventricular failure. Very high pulmonary arterial pressure is usually related to greatly increased pulmonary vascular resistance. Thus, there

### Table 2

<table>
<thead>
<tr>
<th>Factor</th>
<th>Total no. of patients</th>
<th>Hospital deaths No.</th>
<th>Per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>History or evidence of congestive heart failure&lt;br&gt;Present</td>
<td>18</td>
<td>7</td>
<td>39</td>
</tr>
<tr>
<td>Absent</td>
<td>101</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Peak right atrial pressure&lt;br&gt;15 mm. Hg or more</td>
<td>10</td>
<td>5</td>
<td>50</td>
</tr>
<tr>
<td>Less than 15 mm. Hg</td>
<td>109</td>
<td>9</td>
<td>8</td>
</tr>
<tr>
<td>Right-to-left shunt&lt;br&gt;10 per cent or more</td>
<td>14*</td>
<td>7</td>
<td>50</td>
</tr>
<tr>
<td>Less than 10 per cent</td>
<td>105</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Pulmonary hypertension&lt;br&gt;75 mm. Hg or more, systolic</td>
<td>19</td>
<td>10</td>
<td>53</td>
</tr>
<tr>
<td>Clinically absent or less&lt;br&gt;than 75 mm. Hg, systolic</td>
<td>100</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Ratio of total pulmonary resistance to systemic resistance†&lt;br&gt;0.5 or more</td>
<td>7</td>
<td>5</td>
<td>71</td>
</tr>
<tr>
<td>Less than 0.5</td>
<td>38</td>
<td>7</td>
<td>18</td>
</tr>
</tbody>
</table>

*In 3 of the 7 who survived, the pulmonary arterial systolic pressure was 40 mm. Hg or less, suggesting that the increased right-to-left shunt was due to some other factor than failure of the right side of the heart. If these 3 patients are excluded, the mortality rate in the group of 11 remaining patients with a right-to-left shunt of 10 per cent or more is 64 per cent.

†Sufficient data to allow calculation of resistances were available for only 45 patients. This is therefore a somewhat selected group, weighted with those patients for whom the advisability of operation was questioned.

appear to be 2 basically important and closely interrelated determinants of the factors which influence survival of the patient who undergoes operative closure of an atrial septal defect; namely, the presence or absence of excessively high pulmonary vascular resistance and the presence or absence of heart failure.

### Increased Pulmonary Vascular Resistance

When prolonged excessive blood flow through the lungs occurs, resulting from a left-to-right shunt of blood, obstructive changes in the
TABLE 3.—Five Factors Related to Operative Mortality in Patients Representing Surgical Deaths

<table>
<thead>
<tr>
<th>Death</th>
<th>History of presence of congestive heart failure</th>
<th>Right-to-left shunt</th>
<th>Systemic blood flow</th>
<th>Pulmonary arterial systolic pressure (mm. Hg)</th>
<th>Ratio of total pulmonary vascular resistance to that of systemic vessels (resisting air)</th>
<th>Right atrial pressure (mm. Hg)</th>
<th>Left-to-right shunt, pulmonary vascular flow (ml.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Yes</td>
<td>38</td>
<td>86</td>
<td>.77</td>
<td>27/12</td>
<td>33</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Yes</td>
<td>19</td>
<td>99</td>
<td>.73</td>
<td>35/23</td>
<td>21</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>No</td>
<td>47</td>
<td>138</td>
<td>&gt;1.0</td>
<td>6/4</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>No</td>
<td>19</td>
<td>86</td>
<td>.55</td>
<td>4/3</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>No</td>
<td>31</td>
<td>116</td>
<td>.7</td>
<td>10/4</td>
<td>28</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>No</td>
<td>16</td>
<td>107</td>
<td>.57</td>
<td>9/5</td>
<td>42</td>
<td></td>
</tr>
<tr>
<td>7†</td>
<td>No</td>
<td>8</td>
<td>94</td>
<td>.39</td>
<td>?</td>
<td>?</td>
<td>50</td>
</tr>
<tr>
<td>8</td>
<td>Yes</td>
<td>8</td>
<td>94</td>
<td>.39</td>
<td>9/4</td>
<td>57</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Yes</td>
<td>0</td>
<td>114</td>
<td>.22</td>
<td>20/8</td>
<td>75</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>Yes</td>
<td>12</td>
<td>44</td>
<td>.1</td>
<td>12/4</td>
<td>80</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>Yes</td>
<td>0</td>
<td>50</td>
<td>.16</td>
<td>26/15</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>Yes</td>
<td>0</td>
<td>40</td>
<td>.12</td>
<td>25-30/7</td>
<td>68</td>
<td></td>
</tr>
<tr>
<td>13‡</td>
<td>No</td>
<td>0</td>
<td>76</td>
<td>.2</td>
<td>12/4</td>
<td>53</td>
<td></td>
</tr>
<tr>
<td>14‡</td>
<td>No</td>
<td>0</td>
<td>34</td>
<td>.1</td>
<td>8/1</td>
<td>63</td>
<td></td>
</tr>
</tbody>
</table>

*Catheterization done elsewhere—data incomplete.
††Reading of ear oximeter dropped from 93 at rest to 88 with exercise.
‡Death apparently due to technical factors.

pulmonary vasculature develop in some patients. The effect of increased pulmonary vascular resistance on hemodynamics and cardiac function differs significantly in the various congenital cardiac anomalies that permit a left-to-right shunt.

For example, in the presence of a large ventricular septal defect or a large aorticopulmonary communication the volume of blood shunted is a function of the relative pulmonary and systemic resistances. As the pulmonary resistance increases, a progressively smaller proportion of the pulmonary blood flow originates from the left ventricle.

In contrast, the volume of blood shunted through an atrial septal defect is only indirectly related to pulmonary vascular resistance, but rather is directly related to the relative volumes of blood that the right and left ventricles will accept from the atria during diastole. The atria can probably be regarded as a single reservoir for filling of the ventricle in the presence of an atrial septal defect of a size that does not materially restrict free flow of blood between the atria. The volume of blood entering either ventricle during diastole is dependent upon many factors, such as the relative impedance to the flow of blood offered at the mitral and tricuspid valves, and the completeness of emptying of each ventricle during systole. Probably most important are the relative volume distensibilities of the left and right ventricles, which are in part determined by the thickness and pliability of the ventricular walls. In the absence of markedly increased pulmonary vascular resistance and associated pulmonary hypertension the wall of the right ventricle is thinner and more distensible than that of the left ventricle, resulting in a large left-to-right shunt. Probably for the same reason, in the normal heart the end-diastolic pressure in the left ventricle is significantly higher than in the right. Thus, in the presence of an atrial septal defect, the hypertrophy of the right ventricle associated with the development of severe pulmonary hypertension may induce distensibility characteristics that more closely approximate those of the left ventricle, with concomitant reduction of the left-to-right shunt and the development of a right-to-left shunt.

There is a second contrast between the hemodynamic consequence of pulmonary hypertension in the presence of a ventricular septal defect and in the presence of an atrial septal defect. In the former, when pulmonary resistance is still low, the left ventricle has a large volume load imposed upon it, but as pulmonary resistance increases, the left ventricular volume load diminishes toward normal without a concomitant increase in its pressure load. It is never called upon to expel its blood against a resistance greater than that of the systemic vascular bed. Indeed, as pulmonary resistance climbs to excessive levels and a predominant right-to-left shunt appears, the strain imposed by the hemodynamic derangement shifts from the previously burdened left ventricle to the right ventricle.

However, in the case of an atrial septal defect, when the progressive increase in pul-
monary resistance is associated with a reduction in the volume of left-to-right shunt, the volume of blood pumped by the right ventricle is thereby lessened, but the total work load of the right ventricle remains increased above the normal level. Thus, in contrast to ventricular septal defect, the development of pulmonary hypertension in a patient with an atrial septal defect may not significantly lessen the encumbrance of the initially overburdened right ventricle.

Heart Failure. The data presented demonstrate that strong evidence for the presence of heart failure indicates a high operative risk for closure of an atrial septal defect. Therefore, it may be well to consider the mechanisms that result in heart failure in the presence of an atrial septal defect.\(^7\)

Of nonsurvivors in this series, 4 of 14 patients exhibited signs of heart failure in association with severe pulmonary hypertension, and in each of these the volume load on the right ventricle had been much reduced by an increasing right-to-left shunt as well as by the reduction in volume of blood shunted from left to right.

Three patients exhibited strong evidence of heart failure in the absence of severe pulmonary hypertension, with large pulmonary blood flows and decreased pulmonary resistances. It may be significant that in 2 of these patients, marked tricuspid insufficiency was noted at operation, more severe than was noted in other patients in the series. Tricuspid insufficiency, of course, imposes a still greater volume load on the right ventricle.

Once chronic heart failure, from whatever cause, has occurred, the ability of that heart to regain compensation is apparently hampered, since any tendency to do so again increases the volume of the left-to-right shunt and thus the work of the right ventricle. Therefore, in certain patients it would seem inordinately difficult for the right ventricle to recover from a state of chronic failure, for not only is it unable to escape the burden of any existing pulmonary hypertension, but also, as it might tend to regain compensation, the volume load imposed upon it is increased.

### Table 4.—Relation of Over-all Operative Mortality Rate to Number of “Specific Factors” Present in Individual Patients

<table>
<thead>
<tr>
<th>Factors* present in patient</th>
<th>No. of patients</th>
<th>Mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>80</td>
<td>1.3</td>
</tr>
<tr>
<td>1</td>
<td>22</td>
<td>4.5</td>
</tr>
<tr>
<td>2</td>
<td>9</td>
<td>56</td>
</tr>
<tr>
<td>3</td>
<td>6</td>
<td>83</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>5</td>
<td>2</td>
<td>100</td>
</tr>
</tbody>
</table>

*The 5 adverse factors listed in table 2.

This does not pertain in the case of acute heart failure due to severe arrhythmia, to which these hearts are prone, and which may quickly subside with the return of normal rhythm.

### Summary

The data on 119 adult patients who had been operated upon by us for closure of an atrial septal defect were studied to determine what features determinable preoperatively bear upon the patient’s ability to survive the operative period. Of the several factors so analyzed, 5 are significantly related to survival, namely, a history of clinical congestive heart failure, a markedly elevated right atrial pressure, a large right-to-left shunt, severely elevated pressure in the pulmonary artery, and markedly increased pulmonary vascular resistance. Each of these pertains to the presence of heart failure or excessive pulmonary vascular resistance. The mortality rate for repair of uncomplicated atrial septal defects by the atrial-well technic was less than 2 percent.

### Summario in Interlingua

Datos colligite in le casos de 119 patientes adulte operate per nos clauder un defecto del septo atrial esseva studiate con le objectivo de determinar qual factores de determinabili-te pre-operatori affice le capacitate del patientes de superviver al periodo del operation. Inter le varie factores analyosate ab iste puncto de vista, 5 es significativemente relationate al question del supervivencia. Illos es (1) un historia de clinice congestive disfallimento car-
diate, (2) un marcamente elevate pression
dextero-atrial, (3) un grande derivation dex-
tero-sinistre, (4) un severmente elevate pres-
ion in le arteria pulmonar, e (5) marcate
augmentos del resistentia pulmmono-vascular.
Omne iste factores es relacionate al presentia
de insufficiencia cardiac o de excesso del re-
sistentia pulmmono-vascular. Le mortalitate in
le reparo de non-complicate defectos del septo
atrial, effectuate per medio del technica a pu-
teo atrial, essea quasi 2 pro cento.

REFERENCES

1. Cooley, J. C., and Kirklin, J. W.: The sur-
gical treatment of persistent common atrio-

2. Wood, E. H.: Symposium on cardiac catheter-
ization. Special technics of value in cardiac ca-
theterization laboratory. Proc. Staff Meet.

3. Gross, R. E., Watkins, E., Jr., Pomeranz,
surgical closure of interauricular septal de-

4. Kirklin, J. W., Patrick, R. T., and Theve,
R. A.: Theory and practice in the use of a
pump-oxygenator for open intracardiac

5. Bailey, C. P., Downing, D. F., Geckeler,
G. D., Likoff, W., Goldberg, H., Scott,
J. C., Jantion, O., and Redondo-Ramirez,
H. P.: Congenital interatrial communica-
tions: Clinical and surgical considerations
with a description of a new surgical tech-
37: 888, 1952.

6. Swan, H. J. C., Burchell, H. B., and Wood,
E. H.: The presence of venoarterial shunts
in patients with interatrial communications.


Brofman, B. L., Charms, B. L., Kohn, P. M., Elder, J., Newman, R., and Rizika, M.:
Unilateral Pulmonary Artery Occlusion in Man. Control Studies. J. Thoracic

Temporary occlusion of the right or left main pulmonary artery was performed in
over 100 patients by means of an especially designed triple-lumen cardiac catheter
with an inflatable cuff. Pulmonary arterial flow to 1 lung was shut off for as long
as 2 hours with no ill effects. In the control subjects there was no significant electro-
cardiographic change nor any important alteration in systemic blood pressure, oxygen
saturation, cardiac output, or oxygen consumption. The main pulmonary artery
pressure rose about 30 per cent but exhibited a slight fall on breathing 100 per cent
oxygen, indicating further expansibility of the vascular tree. Apparently marked
increases in unilateral flow (even to 5 times normal) are attended by significant de-
creases in pulmonary vascular resistance. Distal to the occlusion the pulmonary
arterial pressure fell to about 8 mm. Hg, approximating pulmonary wedge pressure.
Another indication of retrograde flow from the left atrium was the finding of high
blood oxygen saturation. There was evidence of bronchial artery-pulmonary vascular
communications in the control subjects. X-ray studies showed no change in vascular
pattern during the period of main unilateral pulmonary artery occlusion.

Enselberg
Atrial Septal Defect: Factors Affecting the Surgical Mortality Rate
DWIGHT C. MCGOON, H. J. C. SWAN, ROBERT O. BRANDENBURG, DANIEL C. CONNOLLY and JOHN W. KIRKLIN

_Circulation_. 1959;19:195-200
doi: 10.1161/01.CIR.19.2.195
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1959 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/19/2/195

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org/subscriptions/