The Relief of Acute Right Ventricular Strain by the Production of an Interatrial Septal Defect

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Since sudden resistance increase in the pulmonary circulation leads to right ventricular overdistension and subsequent failure, the question was studied whether or not the existence of an experimentally produced interatrial septal defect could prevent an overstretching of the right myocardium in acute pulmonary stenosis. It was found that myocardial overdistention could be prevented by a small interatrial septal perforation without essentially changing normal interatrial pressure relations. A large defect, which also protected the right heart muscle from overstretching, changed atrial pressure relations and cardiac dynamics entirely.

In patients with a patent foramen ovale unoxygenated blood may enter the systemic circulation when the usual left to right interatrial pressure gradient is reversed. In animals with experimental interatrial septal defects, right atrial pressure exceeded left (a) occasionally during atrial systole, (b) consistently during inspiration and (c) for a longer period during right ventricular failure due to infusion of large amounts of fluid. The frequent clinical occurrence of right to left shunts and right heart strain due to increased pulmonary resistance warranted further investigation of the basic dynamic changes in conditions which bring about a reversal of the interatrial pressure gradient. Pulmonary stenosis, known to increase right atrial pressure and to produce right ventricular failure was therefore used to study left and right atrial, right intraventricular and aortic pressures in one and the same animal first before and then after the production of an interatrial septal defect.

Method

Eleven dogs weighing between 11 and 18 Kg. were anesthetized with 3 mg. per Kg. of morphine sulfate followed by intravenous injection of 200 mg. per Kg. sodium barbital. The chest was opened through the anterior midline. Adequate artificial respiration was maintained, and was interrupted for brief periods when records were taken. The pulmonary artery was dissected free at the conus and a metal clamp applied. The clamp had a lower stationary arm upon which the vessel rested and an upper movable arm for finely graded compression by means of a micrometer screw arrangement on the handle. The clamp was firmly fixed so as to maintain its position for accurately reproducible degrees of constriction before and after the production of an interatrial septal defect. The degree of constriction was changed stepwise, thus always permitting a stabilization of the circulation from one step to the next. To prevent clotting of cannulas, 20 mg. of heparin were administered prior to cannulation.

Aortic pressure pulses were obtained by introducing a rigid cannula through the left carotid artery. Left atrial pressures were recorded through a short catheter of 2.5 mm. lumen through the largest pulmonary vein of the left upper lobe and right atrial pressures were obtained through a 5 mm. wide sound passed through the right jugular vein. In two experiments right intraventricular pressure was recorded additionally with a 15 gage needle thrust through the right ventricular wall. All cannulas were fixed to reduce extraneous vibrations.

The pressure pulses were recorded with Gregg optical manometers and each beam was calibrated at the end of each record with a known static pressure in relation to a base line. A complete calibration of all manometers over the entire pressure range was made after each experiment. The atrial manometers were calibrated in millimeters of saline and the aortic and right ventricular manometer in millimeters of mercury. The common reference point for all pressures, the zero level, was set at the level of the animal board. Therefore all pressures are relative.

Interatrial septal defects were produced by two different methods. In some experiments a modified long hemostat was introduced through the tip of the
left auricular appendage and thrust through the fibrous septum followed by a digital widening of the lesion by exerting pressure on the defect from the right atrial wall. In other experiments the defect was directly produced digitally by entering the left atrium through the left auricular appendage. Larger defects were produced by the latter method. Following the production of the lesion by either method the animals remained in good condition, as indicated by normal contours of the aortic pressure pulses. The postmortem examination and measurement of the defects showed that their sizes varied from 4 to 24 mm. in diameter. The lesions were round or oval and located in the fibrous part and, in the case of larger defects, also in the bordering muscular portion of the interatrial septum.

Both atrial pressure curves were analyzed at four points of special significance, the beginning of atrial systole, peak of atrial systole, end of atrial systole (Z point) which furnishes an index of initial tension within the ventricle, and finally the point just prior to the opening of the atrioventricular valves (V point) which serves as an index of atrial filling during ventricular systole.4

RESULTS

Figure 1 shows two sections of a typical record of left and right atrial and aortic pressure pulses in an animal with an intact septum (A) before and (B) during moderate pulmonary artery occlusion. It is noted that left atrial pressure decreased with pulmonary stenosis and flattened out its pulse contour. Simultaneously right atrial pressure increased and its pulse contour became more prominent. Left-right atrial pressure relations were particularly determined by the alterations of the pulse contour.

These alterations were analyzed in all experiments by measuring instantaneous left and right atrial pressures at the beginning, peak

![Diagram](https://example.com/diagram.png)

Fig. 1. Changes of left and right atrial pressures during progressive pulmonary artery constriction. A, control; B, moderate constriction. Calibrations in mm. Hg for aortic, in mm. saline for atrial pressure; zero point 60 mm. below level of atria.

and end of atrial systole and the V point. Data of two experiments are shown as examples in the four left columns (A) of tables 1 and 2. With pulmonary stenosis left atrial pressure declined more at the V point and peak of atrial systole than at the beginning and end of atrial systole (tables 1A and 2A, lines 1 and 4). On the other hand, right atrial pressure increased more at the V point than at the beginning of atrial systole (tables 1A and 2A, lines 2 and 5). These changes determined the left to right atrial pressure differences at different parts of the atrial cycle. The difference was calculated by subtracting right from left atrial pressures. During control the left to right atrial pressure difference was greatest at the V point and smallest at the peak of atrial systole (tables
RIGHT VENTRICULAR STRAIN AND INTERATRIAL SEPTAL DEFECT

1A and 2A, line 3). With pulmonary artery constriction right atrial pressure exceeded left during part of the atrial cycle with a maximal pressure reversal at the V point, while right pressure remained below left at the beginning (tables 1A and 2A, line 6), and in some experi-
tirely obliterated, right contours indicated tri-
cuspid regurgitation and right pressures ex-
ceeded left at all points of the atrial cycle.

When a small interatrial septal defect of 4 to 10 mm. in diameter was created in four dogs left atrial pressure decreased and right in-
ments also at the end of atrial systole (table 1A, line 6). This pattern of a phasic pressure re-
versal in each atrial cycle was found in all experiments when the pulmonary artery was constricted by about 65 to 80 per cent of its lumen. With still greater degrees of pulmonary stenosis left atrial pulse contours became en-
creased but slightly. As an example, the atrial pressure measurements of one of these experi-
ments are tabulated in the four right columns of table 1B. When identical degrees of pul-
monary artery constriction were applied as before the perforation, left atrial pressure de-
creased, right increased and both atrial pulse

Table 1.—Influence of Moderate Pulmonary Stenosis on Interatrial Pressure Relations (A) before and (B) after the Creation of a Small Interatrial Septal Defect 5 Mm. in Diameter

<table>
<thead>
<tr>
<th>Point of measurement</th>
<th>With intact septum</th>
<th>With perforated septum</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BAS</td>
<td>PAS</td>
</tr>
<tr>
<td><strong>Without pulmonary artery obstruction</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(1) Left atrial pressure</td>
<td>148</td>
<td>162</td>
</tr>
<tr>
<td>(2) Right atrial pressure</td>
<td>112</td>
<td>130</td>
</tr>
<tr>
<td>(3) Left/right pressure difference</td>
<td>+36</td>
<td>+32</td>
</tr>
<tr>
<td><strong>With identical degrees of pulmonary artery constriction</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(4) Left atrial pressure</td>
<td>139</td>
<td>146</td>
</tr>
<tr>
<td>(5) Right atrial pressure</td>
<td>130</td>
<td>160</td>
</tr>
<tr>
<td>(6) Left/right pressure difference</td>
<td>+9</td>
<td>-14</td>
</tr>
</tbody>
</table>

BAS = beginning of atrial systole; PAS = peak of atrial systole; Z = end of atrial systole; V = point before opening of ativoventricular valves. Left to right pressure difference is labeled plus and right to left minus.

Table 2.—Influence of Moderate Pulmonary Stenosis on Interatrial Pressure Relations (A) before and (B) after the Creation of a Large Interatrial Septal Defect 24 Mm. in Diameter

<table>
<thead>
<tr>
<th>Point of measurement</th>
<th>With intact septum</th>
<th>With perforated septum</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BAS</td>
<td>PAS</td>
</tr>
<tr>
<td><strong>Without pulmonary artery obstruction</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(1) Left atrial pressure</td>
<td>120</td>
<td>128</td>
</tr>
<tr>
<td>(2) Right atrial pressure</td>
<td>98</td>
<td>114</td>
</tr>
<tr>
<td>(3) Left/right pressure difference</td>
<td>+22</td>
<td>+14</td>
</tr>
<tr>
<td><strong>With identical degrees of pulmonary artery constriction</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(4) Left atrial pressure</td>
<td>105</td>
<td>110</td>
</tr>
<tr>
<td>(5) Right atrial pressure</td>
<td>101</td>
<td>138</td>
</tr>
<tr>
<td>(6) Left/right pressure difference</td>
<td>+4</td>
<td>-28</td>
</tr>
</tbody>
</table>

BAS = beginning of atrial systole; PAS = peak of atrial systole; Z = end of atrial systole; V = point before opening of ativoventricular valves. Left to right pressure difference is labeled plus, and right to left minus.
contours changed about in the same manner but not quite to the same extent as with the intact septum. Thus the magnitude of the interatrial pressure gradient reversal was reduced by the small shunt (tables 1A and B, lines 4 to 6). The pattern of the phasic pressure reversal obtained with 65 to 80 per cent pulmonary artery constriction was, however, essentially the same as before the septum was perforated. According to the prevailing pressure gradient the flow through the shunt was, therefore, directed from left to right during some parts of the atrial cycle and reversed during other parts of the cycle. Greater pulmonary stenosis caused a complete interatrial pressure gradient reversal at all points of the atrial cycle. Even with severe degrees of pulmonary artery constriction left atrial pulse contours were, however, not quite obliterated (see also fig. 3B).

The influence of a small defect on directional atrial pressure gradients in another experiment is illustrated in the upper graphs of figure 2 by plotting left (solid line) and right (dotted lines) atrial pressures, measured at the V point, with the same degree of pulmonary stenosis before (A) and after (B) the defect was made. It is obvious from the crossing of lines that with the presence of a defect a greater degree of pulmonary artery constriction had to be applied to reverse the interatrial pressure gradient.

Figure 3 illustrates the typical difference of the response of the right heart when the septum was intact (A) and perforated (B) by a small fenestra 5 mm. in diameter. In the records of section A presystolic pressure in the right ventricle rose to 13 mm. Hg and the pressure rose very slowly to a systolic peak of only 27 mm. Hg. Right heart failure due to myocardial overdistention was obviously imminent. The records of section B show a reduction in presystolic pressure to 4 mm. Hg which was followed by a sharp elevation of pressure to a systolic peak of 58 mm. Hg. Duration of systole also increased. Obviously the presence of a small interatrial perforation served to relieve the right myocardium from augmented pulmonary resistance and aided in the prevention of right heart failure. The beneficial effect of such a shunt is also shown by the improvement of left ventricular output as evidenced by an increase in aortic pressure from 20/18 to 63/42 mm. Hg under identical degrees of pulmonary artery constriction.

Quite different results were obtained in those experiments in which a large interatrial septal defect was created. In seven dogs perforations varying from 14 to 24 mm. in diameter were made. Left atrial pressure decreased and right increased after the operation, thus reducing the right-left interatrial pressure difference (table 2A and B, lines 1 to 3). With identical degrees of pulmonary artery resistance, left atrial pressure did not decline but even increased to almost the same level as the moderately rising right atrial pressures (compare table 2A and B, lines 4 to 6). In the lower graphs of figure 2 data of another experiment with a large defect were plotted. Here also left atrial pressure, measured at the V point (solid line), increased almost to the same extent as right (dotted line). Even with complete pulmonary artery occlusion (at stage 5 in figure 2) the magnitude of the interatrial pressure gradient reversal remained small. The data of table 2B and the lower right graph in figure 3 show that a large perforation tends to equalize both atrial
pressures at normal as well as with increased pulmonary resistance. This tendency is also illustrated in figure 4A and B by the records of (fig. 4A). Then the pulmonary artery was completely occluded for half a minute and the records reproduced in the right section of figure 4 were

still another experiment. As soon as the large defect was created right and left atrial pulse contours became almost identical in appearance obtained. Right and left atrial pressures both increased and almost retained their previous contours while aortic pressures were barely

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**Fig. 3.** Effect of a small interatrial septal defect 5 mm. in diameter upon right intraventricular, aortic, left and right atrial pressure when identical degrees of pulmonary artery constriction were applied in the same experiment (A) before and (B) after the perforation was made. Calibrations in mm. Hg for aortic and right intraventricular, in mm. saline for atrial pressures; zero point 60 mm. below level of atria.

**Fig. 4.** Effect of a large interatrial septal defect of 23 mm. in diameter upon aortic, left and right atrial pressures (A) without pulmonary stenosis, and (B) with complete occlusion of the pulmonary artery for half a minute. Calibrations as in figure 1.
reduced. Accordingly the flow through the shunt was large enough to maintain a nearly normal cardiac output.

**Discussion**

The "zone of interatrial pressure reversals" which we were able to produce in our experiments by moderate pulmonary artery constriction emphasizes the importance of the recording of phasic atrial pressure changes for evaluating the interatrial pressure relations. Recording of mean pressures could not reveal these phasic pressure gradient reversals but could create the misleading picture that one atrial pressure was higher than the other during the entire atrial cycle. It ignores the fact that blood could be transferred through the shunt in opposite directions in rapid alternation during each atrial cycle. Phasic reversals of the interatrial pressure gradient and shunt flow of the nature reported in these experiments and such as those caused by respiratory changes are apparently more frequently occurring than the conventional methods of testing reveal.

Comparison of the animals with different sized defects showed that atrial pressure relations were only slightly altered by the presence of a small defect. However, in acute pulmonary stenosis enough blood was shunted through such a small defect from the right to the left atrium to relieve right myocardial overdistention. The small shunt acted, thus, as a "failure-preventing safety valve." The experimental evidence presented here supports recent clinical observations reported by Engle and Taussig that "the patency of the foramen ovale acts for a time as an 'escape valve' and lessens the load on the right side of the heart by the shunt of blood from the right auricle to the left."

A different situation was found in animals with large-sized defects. When the size of the defect exceeded 1.5 or 2 cm. in diameter in a medium-sized dog heart, atrial dynamics were completely altered (lower right plot in fig. 2). Of particular significance was the resemblance of right and left atrial pulse contours (fig. 4), indicating that the normally quite different atrial volume elasticity characteristics were almost lost. We must assume that atria with such extensive septal defects form functionally a more or less common cavity.

Since a small septal perforation did not essentially alter atrial dynamics and limited the shunting of venous blood into the systemic circulation, right ventricular strain may be benefited by such a small shunt. No benefit could be expected, however, from large defects which change atrial dynamics extensively and permit the shunting of such large amounts of blood that cardiac output can be maintained near normal with complete occlusion of the pulmonary artery.

**Summary**

During acute experiments in open chest dogs the pulmonary artery was constricted with increasing severity by identical degrees of occlusion, first before and then after an interatrial septal defect was made. Instantaneous right and left atrial, right intraventricular, and aortic pressures were recorded through cannulas which were not displaced during the defect operation. Progressive pulmonary stenosis reduced left and increased right atrial pressures. Left atrial pulse contours flattened out and right became more pronounced. Moderate stenosis reversed the usual left to right interatrial pressure gradient at the peak of atrial systole and V point, whereas no pressure reversal took place at the beginning of atrial systole. The same type of phasic pressure reversals occurred after the creation of a small interatrial septal defect. Accordingly the shunt flow was directed from left to right during one part of the atrial cycle and in the opposite direction during the other part of the cycle. With marked stenosis right atrial pressures exceeded left at all points of the atrial cycle. Right heart failure produced in the intact heart by severe pulmonary artery constriction was alleviated by the presence of a small interatrial septal defect. The defect reduced excessive right intraventricular diastolic pressures and thus prevented myocardial overstretch. A large defect altered atrial dynamics insofar as left atrial pressures increased together with right when the pulmonary artery was occluded.
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