Effect of Atrial Septal Defects on Hemodynamic Alterations Caused by Acute Partial Obstruction of Pulmonary Artery

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PRIORITY investigations of the hemodynamic effects of progressive constriction of the pulmonary artery have demonstrated the basic principles of right ventricular compensation for an increase in resistance to outflow.1-3

Acute obstruction of the pulmonary artery results initially in an increase in right ventricular diastolic volume and in increased vigor of cardiac contraction, with maintenance of cardiac output and systemic blood pressure. The right ventricular systolic pressure is moderately increased. With increasing degrees of pulmonary obstruction, the right ventricle becomes distended, resulting in a heightened diastolic pressure and further increase in right ventricular systolic pressure. Cardiac output and systemic blood pressures are maintained or slightly reduced. Finally, with even greater degrees of obstruction, one sees pronounced distention of the right ventricle, with further elevation in diastolic pressure, a great reduction in cardiac output, and a decrease in systemic blood pressure.

These studies have been carried out in open-chest dogs, as have other investigations in which graded compression of the main pulmonary artery has been used to reproduce experimentally (a) the physiologic state comparable to massive pulmonary embolism,4,5 (b) the primary effects of reduced cardiac output on renal function,6 and (c) other cardiac dynamic effects.7,8 Under these circumstances, the presence of an acutely produced atrial septal defect has been shown to be beneficial by preventing overdistention of the right ventricle9 and the development of acute failure of the right side of the heart.

The present investigation was carried out to determine the effects on intracardiac and systemic blood pressures and flows of progressive acute partial obstruction of the main pulmonary artery in the more nearly normal physiologic situation pertaining in anesthetized closed-chest dogs studied without thoracotomy. Observations were made on dogs with chronic experimental atrial septal defects and on dogs with intact cardiac septa.

Methods

Ten ordinary-sized mongrel dogs averaging 17.1 (14 to 22) Kg. in weight with experimentally created atrial septal defects were studied anywhere from three weeks to 15 months after the creation of the defects. Because of the desirability of a relatively large shunt in studying the hemodynamic effects in the presence of an atrial septal defect, an effort was made to create large defects by removing the membranous septum of the fossa ovalis and the major portion of the muscular septum. Defects averaging 1.6 by 1 cm. in size were obtained. In addition, four dogs with intact cardiac septa, averaging 17.3 (13 to 20) Kg. in weight, were studied under the same circumstances.

The dogs were anesthetized by the intravenous administration of sodium pentobarbital (25 mg. per Kg. of body weight) plus morphine sulfate (5 mg. per Kg.). The animals breathed 99.5 per cent oxygen via a cuffed endotracheal tube throughout the procedure. Oxygen uptake and pulmonary ventilation were recorded continuously by closed-circuit spirometry.

Measurements of pressure were obtained via cardiac catheters inserted percutaneously through the external jugular veins into the superior vena cava, inferior vena cava, right ventricle, and pulmonary artery.

Central aortic pressure was recorded via a nylon tube 60 cm. long, with internal and external diam-
Thoracic roentgenogram of dog (no. 7) with atrial septal defect showing position of catheters within cardiac silhouette during acute partial obstruction of pulmonary artery. The catheters were introduced percutaneously via the external jugular veins and manipulated so that their tips lay in the superior (S) and inferior (I) venae cavae, right ventricular outflow tract (R), and pulmonary artery (P). The balloon (B) near the tip of the catheter in the main pulmonary artery was inflated with 9 ml. of 70 per cent sodium acetrizoate. The pressures transmitted through the catheters and needles were recorded by means of specially adapted strain gauge manometers. Intravascular pressures, respiration, and electrocardiograms were recorded by means of the photokymographic assembly previously described. In each observation, the zero level for all manometers was set at the mid-thoracic level, and calibrations against a standardized manometer were made over the entire range of pressure during and after each study.

Samples of blood were drawn through a cuvette oximeter for the intermittent determination of oxygen saturation at the femoral artery and at different locations in the cardiac chambers. Samples of blood also were drawn intermittently for the determination of oxygen content and capacity by manometric techniques. These measurements were made by the method of Van Slyke and Neill, as modified by Sendroy.

Indicator-dilution curves were recorded simultaneously from the femoral artery and right ventricular outflow tract by means of cuvette oximeters. One such oximeter was placed at the femoral artery, and a second one was so arranged that it could be used interchangeably with any of the venous catheters. The injections of indocyanine green were done at various locations in the heart and great vessels before and during occlusion of the pulmonary artery. These sites included both venae cavae and the main pulmonary artery and its branches. In addition, in dogs with intact cardiac septa, indocyanine green was injected into the right ventricle, with samples being taken simultaneously from the femoral artery and right atrium in order to study the competence of the tricuspid valve.

The presence and magnitude of the venoarterial shunt via the atrial septal defect were determined from the dilution curves recorded at the femoral artery after injection of indicator into the venae cavae of 0.6 and 0.9 mm., respectively; this was introduced through a 19-gauge thin-walled needle inserted percutaneously into a femoral artery. A 19-gauge needle was inserted into the opposite femoral artery to record dilution curves by means of a cuvette oximeter. The pressures transmitted through the catheters and needles were recorded by means of specially adapted strain gauge manometers. Intravascular pressures, respiration, and electrocardiograms were recorded by means of the photokymographic assembly previously described. In each observation, the zero level for all manometers was set at the mid-thoracic level, and calibrations against a standardized manometer were made over the entire range of pressure during and after each study.

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In all of the animals, this shunt was larger from the inferior than from the superior vena cava, as has been reported previously for both experimental and congenital atrial septal defects. The values for right-to-left shunts reported herein are based on arterial dilution curves after injection into the inferior vena cava.

The magnitude of the left-to-right shunts, as well as that of systemic and pulmonary blood flows, was calculated from dilution curves recorded simultaneously from the right side of the heart and the femoral artery after injections of indicators into selected sites in the central circulation.

In dogs with intact cardiac septa, the cardiac output was calculated from the first part of the arterial dilution curves.

The technique designed for occlusion of one branch of the pulmonary artery was applied in the present investigation. Graded degrees of obstruction of the main pulmonary artery were produced by inflating with radiopaque material a rubber balloon placed near the tip of a cardiac catheter. The position of the cardiac catheters within the cardiac silhouette, when the balloon at the tip of the catheter in the pulmonary artery was filled with 9 ml. of sodium acetrizoate (Urokon), is shown in figure 1.

The balloon covered 3 cm. of the shaft of the cardiac catheter and assumed an oval shape with a circular cross section when filled with fluid. The average diameter of the maximal cross section of the balloon associated with various filling volumes is shown in figure 2. As a result of the physical characteristics of those balloons, an increase in filling volume beyond a certain point increases the length but causes minimal increases in the maximal external diameter.

The degree of occlusion was changed stepwise so as to permit stabilization of the circulation from one step to another. Partial obstruction of the main pulmonary artery was maintained for periods longer than 30 minutes in both groups of animals, averaging 98 minutes in dogs with atrial septal defects and 75 minutes in those with intact cardiac septa.

The atrial septal defects were examined at necropsy in three of the animals; particular attention was paid to the location in the atrial septum and the anatomical relationships of the defect to the opening of both venae cavae and the pulmonary veins. The remaining animals with septal defects are still living.

Results

The striking differences between a normal dog and a dog with a chronic atrial septal defect, with regard to the effects on systemic arterial and right atrial pressures produced by acute increases in resistance to right ventricular outflow, causing similar degrees of right ventricular hypertension, are illustrated in figure 3. The continuous recordings of circulatory pressures in the dog with an atrial
Relationship between right ventricular systolic pressure and mean right atrial pressure during acute partial obstruction of pulmonary artery in anesthetized closed-chest dogs breathing 99.5% oxygen. Determinations before the obstruction are indicated by open circles; those during the obstruction are indicated by solid circles. The numerals beside the control values designate individual animals. Values obtained for dogs with atrial septal defects (A.S.D.) are shown in the right panel. Note that pronounced effects on the mean right atrial pressure were not obtained in the dogs with septal defects regardless of the level of right ventricular hypertension, whereas strikingly different effects occurred in dogs with intact cardiac septa (left panel). Pressures returned to control levels rapidly after release of obstruction (dashed lines).

Septal defect show practically no effects on the mean levels of systemic arterial and central venous pressures as the right ventricular systolic pressure was varied from 95 mm Hg down to normal levels. In contrast, when the dog with an intact atrial septum was subjected to an increase in right ventricular systolic pressure of similar magnitude, great increases in right atrial pressure and pronounced decreases in systemic arterial pressures occurred, resulting in acute cardiac failure when the right ventricular systolic pressure reached a maximal value of approximately 90 mm Hg.

The relationship of right ventricular systolic pressure to the mean right atrial pressure in all of the dogs included in this study is shown in figure 4. In dogs with septal defects prior to occlusion, the right ventricular systolic pressure was less than 40 mm Hg in all instances, averaging 35.1 mm., with a range of 30 to 39 mm. The mean right atrial pressure averaged 4.9 mm., with a range of 2 to 8 mm. Similar values were found for diastolic pressure in the right ventricle. In most of the dogs, an "a" wave was present in the pressure recordings from the right atrium and was associated in some of the animals with a barely discernible "v" wave.

Partial obstruction of the main pulmonary artery in these dogs was followed immediately by an increase in right ventricular systolic pressure. With moderate to severe degrees of obstruction, caused by filling the balloon with 4 to 10 ml. of sodium acetrizoate (fig. 3, lower panel), the right ventricular systolic pressure varied between 50 and 125 mm., averaging 82.5 mm. Increase in right ventricular systolic pressure up to 90 mm. was associated with an average mean right atrial pressure of 5.6 mm. (range of 3 to 9 mm.). With obstructions producing right ventricular systolic pressures of 90 to 125 mm., the average mean right atrial pressure was 8.8 mm. (range of 6 to 12 mm.).

In dogs with intact cardiac septa, pressures during the control studies were within the range for normal animals and were not substantially different from those in the dogs with atrial septal defects (fig. 4). Slight degrees of obstruction (balloon filled with 2 to 4 ml. of sodium acetrizoate) were associated with an increase in right ventricular systolic pressure to levels as high as 60 mm. and a slight average increase in mean right atrial pressure (fig. 3, upper panel). More severe degrees of obstruction were characterized by further increases in systolic pressure and greater elevations in the mean right atrial pressure. At this stage, an increase in right atrial pressure was present at all points on the pressure-pulse complex, with the most pronounced increase at the peak of the "a" wave. With greater degrees of obstruction (balloon filled with 8 to 16 ml. of sodium acetrizoate), the right atrial pressure increased still further, and a predominant "v" wave became evident, as contrasted to the right ventricular systolic pressure, which was
Normal Dog  (17 kg. - Morphine Pentobarbital Anesthesia)

A.S.D. Dog  (17 kg. - Morphine Pentobarbital Anesthesia)

RESPIRATION

SYSTEMIC FLOW: 1.25 L/min.

Figure 5

Original photokymographic record demonstrating typical comparative effects of acute partial obstruction of pulmonary artery on circulatory pressures and flows in normal dog (no. 4) (left panel) and dog (no. 3) with chronic atrial septal defect (right panel). The vertical lines delineating 0.1-second time intervals were 15 mm. apart before photographic reduction. In the dog with the septal defect, no abnormalities in the right atrial and systemic arterial pressure pulses are evident despite severe right ventricular hypertension. However, the normal dog, with comparable right ventricular systolic hypertension, shows a significant decrease in systemic arterial pressure associated with a low systemic flow and an increase in right atrial pressure associated with a prominent "v" wave suggestive of tricuspid regurgitation. The electrocardiogram in the dog with the intact septum exhibits pronounced depression of the S-T segments and T waves. Such transient alterations were not seen in dogs with atrial septal defects.

unchanged or decreased, and the systemic arterial pressure, which decreased, indicating failure of the compensatory mechanisms of the right ventricle (fig. 3). The typical differences in the levels and contours of right atrial and systemic arterial pressure pulses associated with similar degrees of right ventricular hypertension in a normal dog and a dog with an atrial septal defect are illustrated in figure 5. Deflation of the balloon was followed by rapid return of the pressures to approximately the preocclusive levels.

The development of a high-amplitude "v" wave in association with severe degrees of right ventricular hypertension in the normal dogs suggests the presence of tricuspid regurgitation under these circumstances. This was confirmed by means of dilution curves recorded simultaneously from the right atrium and femoral artery after injection of the indicator into the right ventricle. The effects of progressively increased resistance to right ventricular outflow on these curves and the estimated degree of tricuspid regurgitation estimated therefrom, along with other physiological variables in a dog with an intact cardiac septum, are shown in figure 6. A significant degree of tricuspid regurgitation was detected when the right ventricular systolic pressure was increased to 80 mm. Hg, with a progressive increase in severity as the obstruction to right ventricular outflow was increased to the point at which acute failure of the right side of the heart appeared.

The relationships between systemic arterial pressure and right ventricular systolic pressure during progressive occlusion of the pulmonary artery in all of the dogs studied are shown in figure 7. In dogs with atrial septal defects, the control determinations of mean aortic pressure averaged 137 mm. (range of 109 to 159 mm.). Regardless of the duration of the obstruction (up to 160 minutes) and

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Effect of acute progressive partial obstruction of pulmonary artery on competency of tricuspid valve as determined by dilution curves recorded simultaneously from right atrium and femoral artery after right ventricular injection in normal dog (no. 4). Dilution curves were recorded at successive stages of obstruction produced by distention of a balloon in the main pulmonary artery (P.A.) with the volumes of sodium acetrizoate (Urokon) indicated. The cardiac output, degree of tricuspid regurgitation, and the right atrial, right ventricular, and aortic pressures associated with each degree of obstruction are indicated. Rapidly appearing dye was not detected in the right atrium until the right ventricular systolic pressure was 80 mm. Hg (right upper panel). At this level of obstruction to right ventricular outflow, the estimated degree of regurgitation at the tricuspid valve was 45 per cent of the stroke volume; this was associated with a significant increase in right atrial pressure and a slight decrease in aortic pressure and cardiac output. At the maximal right ventricular pressure attained (90 mm.), these effects were still more striking (left lower panel). Further obstruction then produced severe acute right-heart failure (right lower panel), recovery from which was complete and rapid after deflation of the balloon.

The level of right ventricular systolic hypertension, systematic changes in the systemic arterial pressure were not observed (figs. 3 and 7).

When a mild degree of obstruction was produced in normal dogs (figs. 3 and 7), no real effect on the systemic arterial pressure could be seen. However, beyond a critical level of restriction of the cross-sectional area of the pulmonary artery, a considerable decrease in systemic arterial pressure occurred (fig. 3). These pressure changes were immediately reversed after the obstruction was released (fig. 7).

From the data on oxygen saturation for the determination of shunts, it was found that all the dogs with experimental atrial septal defects had shunts in the left-to-right direction (fig. 8), averaging 34 per cent of the pulmonary flow (range of 6 to 70 per cent). Preferential shunting of blood from the right lung was demonstrated after the injection of indicator into the right and left pulmonary arteries as described previously. In the control studies, no correlation was apparent between the magnitude of the shunts and the pressures recorded in the right side of the heart or in the systemic circulation.

Small right-to-left shunts of less than 10 per cent of the systemic flow were demonstrated by indicator-dilution methods before partial obstruction of the pulmonary artery in seven of the 10 dogs with atrial septal defects (fig. 8). One dog had a relatively large
right-to-left shunt representing 29 per cent of the systemic flow.

Partial obstruction of the pulmonary artery of the dogs with septal defects caused alterations in the direction and magnitude of the shunts across the defect. Progressively increased elevation of right ventricular pressure was followed by a progressive decrease in the left-to-right shunts that averaged 50 per cent of the control determinations and was associated with a large right-to-left shunt of as much as 55 per cent of the systemic blood flow (fig. 8).

The relationship of right ventricular pressure to the ratio between the pulmonary and the systemic blood flow in dogs with septal defects is shown in figure 9. Prior to partial obstruction, this ratio was greater than 1.0 in all of the animals, averaging 1.6. During the period of obstruction, however, the ratio was reduced to an average of 0.79, indicating that under these circumstances, the shunt was reversed from a predominantly left-to-right direction to a predominantly right-to-left direction.

The effects of partial occlusion of the pulmonary artery on the magnitude of flows determined by the direct Fick method in both groups of animals are shown in figure 10. In the dogs with septal defects, the pulmonary blood flow prior to obstruction averaged 4.6 L./min., with a range of 1.8 to 9.0 L./min., as compared to the systemic blood flow, which averaged 2.8 L./min. (range of 1.5 to 6.0 L./min.). When right ventricular hypertension was produced, the pulmonary flow was reduced to levels comparable to the range for normal animals, averaging 2.4 L./min. Only minor changes in the levels of systemic blood flow occurred despite the high right ventricular systolic pressures obtained.

In the dogs with intact cardiac septa, the control cardiac output averaged 2.5 L./min. (range of 1.6 to 3.7 L./min.). During the obstruction, despite the lesser degree of right ventricular hypertension obtained, a considerable decrease in cardiac output invariably occurred. Although the most pronounced occlusion produced the greatest decreases in systemic arterial pressure and cardiac output,
Figure 10
Relationship between right ventricular pressure and systemic and pulmonary blood flow in normal dogs and those with atrial septal defects during acute partial obstruction of pulmonary artery. The blood flows were determined by the Fick method. When the right ventricular systolic pressure was increased to more than 60 mm. Hg in dogs with defects, a reduction of pulmonary blood flow to levels comparable with the range for normal animals occurred, and only minor effects on the magnitude of the systemic flow were detected. In normal dogs, however, despite the lesser degree of right ventricular hypertension, a considerable decrease in cardiac output invariably occurred.

A close correlation was not apparent between the degree of obstruction, the fall in blood pressure, and the decrease in cardiac output.

Discussion
The balloon-catheter technique for producing graded obstruction of the main pulmonary artery in closed-chest dogs is a valuable tool for studying the hemodynamic effects of increased resistance to right ventricular outflow in animals without thoracotomy; such animals presumably are in a more nearly normal hemodynamic state than are open-chest animals.

Unfortunately, differences in the shape and length of the balloon when it is inflated, as well as in its exact position in the pulmonary artery, prevent accurately reproducible degrees of obstruction by inflation of the balloon to a given volume. Consequently, no attempt has been made to quantitate the reduction in cross-sectional area of the pulmonary artery attained in these studies. However, the critical level of occlusion at which circulatory failure is produced has been demonstrated in open-chest animals by other investigators to be approximately 60 per cent of the cross-sectional area of the pulmonary artery.

The results of the present study clearly demonstrate that dogs with chronic atrial septal defects have a considerably greater ability to withstand the effects of acute partial obstruction of the pulmonary artery than do dogs with intact cardiac septa. These findings confirm the acute studies of Brecher and Opdyke in open-chest animals.

In normal closed-chest dogs, increase of right ventricular systolic pressure in excess of 60 mm. Hg significantly affected right atrial and systemic arterial pressures. Additional obstruction so that the right ventricular systolic pressure was increased to 90 mm. produced acute cardiac failure in these animals, characterized by a decrease in systemic arterial pressure and cardiac output. In contrast, dogs with atrial septal defects maintained right ventricular systolic pressures at a level greater than 90 mm. for the duration of the observations (20 to 97 minutes) without apparent evidence of cardiac failure.

The basis for the increased ability of these dogs with atrial septal defects to maintain the high right ventricular systolic pressure required to overcome an acute increase in resistance to right ventricular outflow probably is mainly the result of the "safety-valve" action of the atrial septal defect, which, by permitting a right-to-left shunt, prevents overdistention of the right ventricle and also allows maintenance of normal volume flow and pressure in the systemic circulation, despite a decrease in pulmonary flow.

Since the work load of the right ventricle of dogs with such defects was increased over a period of several months because of the left-to-right shunt, it is possible that the compensatory changes in the right ventricle induced by these shunts played a significant role in the greater ability of these dogs to maintain sharply increased levels of systolic pressure. However, the pulmonary arterial pressures of all the dogs with defects were within the range for normal animals before obstruction of the pulmonary artery. In addition, gross evidence of right ventricular...
hypertrophy could not be found in the three animals examined at necropsy. It has been postulated that right ventricular failure in the presence of right ventricular hypertension is produced or accentuated by a reduction in coronary flow to the myocardium, caused by the increased resistance to drainage of coronary venous blood into the right side of the heart. However, Gregg has indicated that an increase of right ventricular pressure by constriction of the pulmonary artery was accompanied by augmentation of coronary inflow in both systole and diastole. If, however, the pulmonary constriction was abrupt, in the presence of an adequately maintained central coronary pressure, a transient period of reduced coronary flow occurred that was ascribed to the mechanical effects of extravascular compression. The decrease in systemic arterial pressure during partial obstruction of the pulmonary artery in the normal dogs would accentuate the effects tending to decrease coronary flow in contrast to the dogs with defects, which maintained normal systemic arterial pressure under these circumstances. In addition, in the dogs with intact cardiac septa, increase in right ventricular systolic pressure to between 75 and 90 mm. Hg was associated with a decrease in pulmonary blood flow, which averaged 83 ml./min./Kg. of body weight, whereas in the dogs with defects, the pulmonary blood flow averaged 120 ml./min./Kg. at right ventricular systolic pressures in excess of 90 mm.

That myocardial ischemia played a significant role in right ventricular failure in the normal dogs is suggested by the pronounced depression of the S-T segments and the T waves in the electrocardiograms (fig. 5). Such transient alterations were not seen in animals with atrial septal defects. Similar electrocardiographic abnormalities, presumably from myocardial ischemia, have been described during acute pulmonary embolism in humans.

While it appears certain that a right-to-left shunt via an atrial septal defect is beneficial to an animal faced with an acute increase in resistance to right ventricular outflow, it is not certain that the arterial hypoxemia caused by such a shunt might not be sufficiently deleterious over a long period to negate the immediate beneficial effects conferred by the septal defects. This question is of considerable practical importance in the problem of closure of septal defects or aortic-pulmonary communications in patients who have severe pulmonary hypertension caused by greatly increased pulmonary vascular resistance. It is well documented that a high incidence of sudden death occurs after surgical closure of the defect in such patients presumably because of acute right-heart failure caused by the high resistance to right ventricular outflow. The present studies support this interpretation and the suggestion that the presence of a unidirectional right-to-left interatrial communication would be beneficial in such patients in the immediate postoperative period. However, the long-term effects of such a communication under this circumstance have not been demonstrated.

Summary

Systemic and pulmonary blood flows and pressures were determined in anesthetized closed-chest dogs with chronic atrial septal defects and in dogs with intact cardiac septa. In the former animals, graded obstruction of the main pulmonary artery associated with right ventricular systolic pressures of more than 80 mm. Hg was produced without evidence of failure of the right side of the heart. A considerable decrease in the magnitude of the left-to-right shunts and an increase in the size of the right-to-left shunts occurred; these changes were associated with only minor changes in filling pressure of the right ventricle, and systematic changes in systemic arterial pressure did not appear. Analysis of right atrial pressure pulses suggested the development of an increased force of contraction of the right atrium and no evidence of tricuspid regurgitation under these circumstances.

In contrast, partial obstruction of the main
pulmonary artery in the dogs with intact cardiac septa, so as to increase the right ventricular systolic pressure to more than 60 mm Hg, was associated with pronounced increases in mean right atrial pressure and striking decreases in cardiac output and systemic arterial pressure. The pressure recordings from the right atrium and indicator-dilution curves demonstrated the development of significant tricuspid regurgitation.

The deterioration of right ventricular function in dogs with intact cardiac septa appears to be related to overdistention of the right ventricle and possibly also to a disproportionate reduction in coronary blood flow to the myocardium caused by a decrease in systemic arterial pressure and the concomitant increase in resistance to coronary venous drainage from the right side of the heart due to the increased right atrial and right ventricular pressures.

It is concluded that under the conditions of these studies, an atrial septal defect provides a considerable increase in resistance to the hemodynamic effects associated with right ventricular hypertension caused by acute partial obstruction of the main pulmonary artery. The possible duration of this beneficial effect beyond the 33 to 160 minutes of observation in these studies has not been explored.

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