PHYSIOLOGIC CONSIDERATIONS OF INTRACARDIAC PRESSURES FOLLOWING CLOSURE OF ATRIAL SEPTAL DEFECTS

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BALTIMORE, Md.

THAT the principal hemodynamic disturbance in patients with atrial septal defect is a left-to-right shunt through the defect is well recognized, but the cause of this shunt has been less clear. In some of our first experiences with closure of atrial defects a prominent thrill was felt as the defect was nearly but not completely closed, and a striking rise in left atrial pressure was noted. As a consequence of this, we have obtained left and right atrial pressures before and after closure of the atrial defect in a number of instances. We wish to present these data with a consideration of the pathogenesis of the left-to-right shunt through the defect and of its surgical significance.

METHOD

Sufficient data for analysis have been obtained on 21 patients undergoing closure of atrial septal defect. Several patients with total anomaly of the pulmonary venous return have been excluded. Those with drainage of the right pulmonary veins into the right atrium were included if repair was modified only by bringing the septum farther to the right in repair than is done in the uncomplicated atrial septal defect. The technique of closure apparently influenced the data little and includes the use of the Gross well, the Sondergaard dissection in the interatrial groove with either interrupted sutures in this groove to grasp the edge of the septum or the purse-string suture described by Björk and Crafoord, and suture under direct vision during circulatory occlusion with hypothermia. Pressures were measured through a 20-gauge thin-wall needle by direct puncture of the various chambers. The needle was connected through a No. 6 cardiac catheter to a Statham strain gauge, and pressure was recorded on a direct writing Sanborn pressure recording system. When care was taken to eliminate all bubbles from the system, satisfactorily undamped pressures were obtained to allow good tracings. Base line pressure was taken with the needle adjacent to the middle of the right atrium. Records of these pressures were reviewed and, in most instances, the mean pressure has been estimated with the use of a compensating planimeter; in a few cases insufficient length of the original tracing has been preserved and only the maximal and minimal pressures are available. Pressures were measured at operation with the thorax open, both before repair of the defect and just before the chest was closed after completion of the repair.

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### Table I

<table>
<thead>
<tr>
<th>CASE</th>
<th>AGE</th>
<th>PULM. FLOW (L/Min./M²)</th>
<th>PULM. RESIST. (MM HG/L/MIN./M²)</th>
<th>LEFT ATRIUM</th>
<th>RIGHT ATRIUM</th>
<th>PULMONARY ARTERY</th>
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<td>BEFORE</td>
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<td>4.6</td>
<td>11.5</td>
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<td>11.2</td>
<td>6/2</td>
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<td>7/6, 6</td>
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<td>12.5</td>
<td>1.39</td>
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<td>25/12</td>
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<td>12/8, 11</td>
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<td>-</td>
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<tr>
<td>Average</td>
<td>24</td>
<td>12.5</td>
<td>0.30</td>
<td>7.1</td>
<td>12.5</td>
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</table>

Pulmonary flows recorded as high when arteriovenous difference of oxygen was tiny or reversed, making calculated flow inestimably high. Pulmonary resistance inestimably low.

Averages based on cases exclusive of those with incomplete data.
Preoperative study of these patients included cardiac catheterization which demonstrated the atrial shunt in all instances. From data thus obtained, pulmonary and systemic flows have been estimated by the Fick principle. In many instances, particularly in young children, the oxygen consumption has not been measured but rather has been estimated as 135 ml. per minute per square meter of surface area. In this laboratory this has been the average oxygen consumption per square meter of surface area, and it is considered that this figure represents as nearly the oxygen consumption at the time of blood sampling, while the child is quiet during fluoroscopy, as measurement from a collection of expired air obtained from a young patient, often anxious concerning the collection of gas. In essence, this means that flows and shunts are estimated on the basis of the arteriovenous oxygen difference and referred to the surface area, the oxygen consumption being considered constant in respect to surface area. An estimate of pulmonary resistance was obtained from cardiac catheterization by dividing the gradient between mean pulmonary artery pressure and left atrial pressure in mm. Hg by the estimated pulmonary flow in liters per minute. In preoperative studies, unless actually measured, mean left atrial pressure was assumed equal to mean right atrial pressure. Normal pulmonary resistance ranges between 1 and 5 mm. Hg per liter per minute per square meter of surface area. (This figure can be readily converted to dynes cm.−2/M2 by multiplying by 60 \times 1.332, or approximately 80.)

RESULTS

Intracardiac pressures taken during operation and before and after repair of the auricular defect are listed in Table I, along with pulmonary blood flow and resistance determined by preoperative cardiac catheterization. Considerable variation of pressures is evident and is not surprising in view of the known changes which occur during operation with the administration and loss of blood, depth and type of anesthesia, and ventilation. Three patients have been listed first in whom the pulmonary artery pressure was high, the pulmonary flow low, and it was felt that the pulmonary hypertension was probably fixed; changes in pressures were least striking in these 3 patients. In all but 2 of the remaining 18 patients the left atrial pressure rose following repair of the defect, as might be expected in view of the normal condition. The tendency was for right atrial and pulmonary artery pressure to fall. A slight rise of left atrial pressure to 5 to 10 mm. Hg was not alarming but, of the 18 patients, 8 had a mean left atrial pressure of more than 15 mm. Hg, ranging up to 28 mm. Hg. Because this striking increase was noted in early cases, we have kept the blood replacement during operation at a less complete level, usually simply matching the suction drainage and excluding the loss of blood on the drapes and sponges. It is probable that if replacement more nearly matched the actual blood loss, left atrial pressures would be recorded higher. In several individuals, and perhaps most strikingly in Patient O. C., a thrill could be felt as the purse-string suture was constricted about the atrial defect. Without further constriction but after inadvertent loss of a moderate amount of blood the thrill disappeared and was not subsequently felt.
The change in left atrial pressure is at the present time unpredictable, as there was no significant correlation with the volume of the pulmonary or systemic flow, vascular resistance, ratio of systemic to pulmonary resistance, or the level of the atrial pressure preoperatively.

All but 2 of these patients are now alive and improved. Patient M. S. developed a supraventricular tachycardia which was present intermittently but frequently for approximately a year but was finally reverted with quinidine; she died the night the rhythm was reverted, apparently of an arrhythmia. The other was Patient J. R. who was felt at operation to have a large insufficient jet through the mitral valve. She developed cardiac asthma on the third postoperative day for about 24 hours and subsequently developed progressive chronic passive congestion. At autopsy, the defect had partially reopened, and there was an old rupture of several of the chordae tendineae with slight prolapse of the mitral valve into the atrium. It was considered that the mitral insufficiency became intolerable when the septal defect, which had served to relieve left atrial pressure, was closed. This may have been one of the relatively minor lesions which impose an insuperable burden on the left ventricle of patients with atrial septal defect as described by Dexter.

**DISCUSSION**

There is ample evidence that the principal hemodynamic disturbance in patients with auricular septal defect is the increased pulmonary blood flow resulting from a left-to-right shunt through the defect. Several groups have demonstrated, by cardiac catheterization, a slightly higher pressure in the left auricle than the right auricle in patients both with and without an auricular septal defect. Little demonstrated, experimentally, a greater pressure rise per unit increase in volume of the left atrium of the dog as compared with the right and cited the observation at human autopsy that the left auricle normally is slightly thicker than the right and presumably has a greater pressure-volume coefficient. Opdyke and Brecher measured pressures continuously in the two atria of dogs and demonstrated a slightly higher pressure in the left atrium during various periods of the cardiac respiratory cycle. This gradient was less in dogs with a surgically created interatrial septal defect. Physical laws of fluid dynamics indicate and measurements confirm, however, that in the presence of a sizable atrial defect no pressure gradient exists. Uhley postulated that the shunt occurs because of a positional difference, the right atrium being below the left atrium in the upright position. The absence of a significant change in the shunt with change of position makes acceptance of this explanation unwise.

Barger, Edwards, Parker, and Dry and Hull suggested that the shunt is due to relative resistance to filling of the right and left ventricles. Dow and Maloney amplified these observations following studies in dogs with experimentally created auricular septal defects, in which they could demonstrate no measurable difference in pressure between the right and left atria. Dow and Dexter investigated this concept in patients with atrial septal defects and...
correlated ventricular output with atrial pressure. With elevated atrial pressure, output of the right ventricle increased while that of the left decreased, and the common atrial pressure appeared to depend more on left than on right ventricular function.

Our concept of the mechanism of shunt in atrial septal defect is graphically illustrated in Fig. 1. In the normal subject, the left atrial pressure has been measured and found to be 1 to 5 mm. Hg higher than the right. Consequently in the normal heart the right and left ventricles fill to the same volume (in the presence of the balanced circulation which must exist normally) under different pressures. In the presence of a large atrial septal defect the pressure in the two auricles is equal and, under such circumstances, the ventricles fill differently with a similar pressure, the right filling more than the left in the uncomplicated case.

This is probably the explanation why pulmonary hypertension is the less frequent accompaniment of atrial septal defect than other communications between the right and left sides of the heart, for as pulmonary resistance increases with associated right ventricular hypertrophy, resistance to filling of the right ventricle increases or the right ventricle fails, and the atrial shunt diminishes. In infants, the thickness of the walls of the two sides of the heart, and hence, probably the resistance to filling, is approximately equal so that in infants the shunt is relatively less than in adults. Only rarely does uncomplicated atrial septal defect cause significant embarrassment to the very young patient.
The observed rise in pressure in the left atrium while pressures in other chambers fell, following closure of atrial septal defect, is further support of this mechanism of the atrial shunt. Of some surgical significance is the fact that left atrial pressure may rise precipitously and strikingly. In 8 of the 21 patients, the mean left atrial pressure rose to 15 mm. Hg or higher. Persistence of this pressure in the left atrium in itself would lead to pulmonary engorgement and edema, and compensation evidently occurs in the postclosure period.

We have been struck by the manner in which intracardiac pressures in patients with an open chest may vary depending upon the condition of the patient’s blood loss and replacement. Estimated blood volumes in patients with atrial septal defect have shown an increased volume in the usual instance with a decrease in this volume after closure. Because of this and because of the observed increase in left atrial pressure we have been less vigorous in blood replacement during operation in these than in other patients being treated with cardiovascular disease.

Finally, in contrast to patients with patent ductus arteriosus or ventricular septal defect, the closure of an atrial defect with a large left-to-right shunt may not be entirely innocuous. We have attributed dyspnea (and in one case cardiac asthma) and periods of arrhythmia after repair of atrial defects to left ventricular decompensation, of which we have indication from increased left atrial pressure at operation. Watkins and Gross noted in some patients a transient left ventricular failure which they attributed to a greater ventricular filling pressure after septal repair. The primary defect appears to be in the left ventricle, which in some cases is hypoplastic, and should be treated by restriction of fluid, diuretics, digitalization, and, on some occasions, venesection. Fortunately, in only the occasional patient has this been a significant problem in the postoperative period. Similar studies and conclusions have recently been reported by Pemberton, Kirklin, and Wood.

CONCLUSION

Intracardiac pressures taken during operation for closure of atrial septal defect in 21 patients have been analyzed. In 3 patients with pulmonary hypertension, increased pulmonary resistance and only slight increase in pulmonary blood flow, there was the least change in these pressures. In the remaining cases, the left atrial pressure tended to rise while pressure in other chambers fell following repair of the defect. In 8 of these patients the mean pressure rose to more than 15 mm. Hg. These data support the concept that flow through the defect is due to decreased resistance to filling of the right as compared with the left ventricle.

This elevation of left atrial pressure following repair of the defect seems to be well tolerated by the otherwise normal heart and compensation proceeds rapidly. In some instances subjection of the left ventricle to an unaccustomed high filling pressure may lead to decompensation, especially in the presence of left-sided valvular or myocardial abnormalities.

The surgical significance of these observations would seem to be that such patients should not be overtransfused during operative repair of the defect and
that the occasional patient who shows signs of left ventricular failure postoperatively should be treated with digitalis and measures directed at reducing circulating plasma volume.

REFERENCES


DISCUSSION

(PAPERS BY SHUMWAY [PAGE 643], JULIAN [PAGE 654], GLOVER [PAGE 661], BENJAMIN [PAGE 679], WILLIAMS [PAGE 685], SAYEIN [PAGE 692] AND THEIR ASSOCIATES)

DR. ARTHUR M. VINEBERG, Montreal, Quebece.—(This discussion was not returned. —EDITOR.)

DR. SAMUEL A. THOMPSON, New York, N. Y.—The proof of the pudding is in the eating. Our clinical experience with this procedure is limited to 19 cases. In 8 cases it was used as a single procedure and in 11 cases it was combined with cardioplexy. The immediate postoperative reaction is very satisfactory so far as relief of pain is concerned, but we have no long-term follow-up. We believe that there is an additional shunt of blood with
increased vascularity to the pericardium following ligation of the internal mammary artery.
We believe, however, that the full value of this increased vascularity cannot be realized unless there is some connection, such as intimate contact of the pericardium to the myocardium beyond the point of coronary obstruction. In the light of our very limited experience, we believe that mammary ligation may be useful in the very early cases of coronary disease, or in the very late cases with severe complications where major surgery cannot be tolerated. In the moderately advanced coronary cases, as well as those of myocardial ischemia, we believe the best results will be obtained when mammary ligation is used in combination with some other procedure such as cardiectomy. Cardiectomy furnishes the necessary vascular bridge between the myocardium and the pericardium. Our present experience leads us to believe that the procedure of internal mammary artery ligation does have value, and we plan to continue its use unless later experience gives some contrary indication.

DR. WILLIAM H. MULLER, JR., Charlottesville, Va.—I should like to discuss the very nice work presented by Drs. Williams and Bahnsen. We have carried out similar determinations on some 17 patients undergoing closure of atrial septal defects, except that we have not correlated these determinations with the degree of shunt flow.

(slides) This slide shows the determinations which we obtained in our cases. In most instances, the right auricular pressure diminishes after the shunt has been obliterated and this pressure likewise diminishes in the right ventricle. We found a significant increase in left auricular pressure in some instances after obliterating the shunt, but we have not attached the same significance to this finding as have Drs. Williams and Bahnsen.

(slides) This slide shows that there is usually a significant decrease in right auricular and right ventricular oxygen saturation after the defect is closed. There is no significant change in the left auricular oxygen saturation.

(slides) This slide demonstrates that the blood volume usually diminishes by about 20 percent after the defect is closed. These measurements were obtained one day before and 3 weeks after the defect was closed, utilizing the T-1824 Evans blue dye method. For this reason we have been somewhat reluctant to transfuse these patients up to their preoperative circulating blood volume after the defect is closed.

DR. MOORE.—(This discussion was not returned.—Editor.)

DR. ADRIAN KANTROWITZ, Brooklyn, N. Y.—I rise to discuss Dr. Glover’s paper. Since this procedure was suggested, we have been interested in studying some of the possible mechanisms for increased coronary blood flow by ligation of the internal mammary artery.

(slides) As Dr. Glover has pointed out, presumably the pathway for improved flow of blood to the myocardium would be by way of the pericardiophrenic arteries which arise from the internal mammary arteries and thence by anastomoses to the coronary arteries. This is essentially a Y arrangement and if you would ligate the internal mammary artery at this point this would presumably improve the blood flow to the pericardium and collaterals.

The flowmeter we have available to us in the laboratory was not small enough to insert into either the internal mammary artery or the pericardiophrenic artery; so we chose an anatomic situation which was approximately the same. This is, the bifurcation of the aorta, which again is a Y arrangement. According to the thinking presented by the speaker, clamping one iliac artery should shunt more blood to the other iliac artery. We inserted our instruments to measure blood pressure and flow to the left iliac artery, and recorded these variables while we clamped and unclamped the right iliac artery. It should be remembered that there is a rich anastomosis between these two vessels, below the point of clamping through the hypogastric arteries.

(slides) The upper tracing is the recording of left iliac blood pressures, the lower tracing is the recording of blood flow through this vessel. At this point the contralateral iliac artery is clamped. There is a momentary rise in blood pressure for one or two heartbeats and an associated momentary increase in blood flow. Then there is a slight drop in blood pressure for perhaps 15 or 20 seconds, again associated with a small decrease in blood flow.

As you see, however, both tracings very quickly stabilize at approximately the preclamping control levels.
When the contralateral artery is unclamped there does not seem to be any appreciable change, in pressure or flow.

We have concluded from this experiment, then, that for this situation we have no evidence that would indicate that occluding one artery would significantly alter pressure or flow in the other.

DR. ROBERT KLOPSTOCK, Brooklyn, N. Y.—(This discussion was not returned.—Ed.)

DR. ANDREW G. MORROW, Bethesda, Md.—We have also been interested in the changes in left and right heart pressure following septal defect closure as described by Dr. Williams and Dr. Bahnson. However, we have collected our data in a somewhat different fashion.

In patients being operated upon with hypothermia or extracorporeal circulation, a catheter is passed into the right ventricle before operation. As soon as the chest is opened another catheter is passed via a segmental pulmonary vein into the left atrium. Thus, the arterial pressure, right ventricular and left atrial pressures can be recorded for 24 hours. Here are such measurements in a patient with an atrial septal defect operated on with hypothermia. You will see that as the patient cools there is a fall in ventricular filling pressure. With manipulation of the heart and passing the caval ligatures the pressures rose strikingly. In the immediate postoperative period there are striking elevations in the left atrial pressure, but in the course of 6 or 8 hours it gradually returns to normal, confirming what Dr. Williams suggested.

I would be particularly interested to know if data concerning digitalis are available, and if their patients who showed these elevations were digitalized.

DR. B. L. BROFMAN, Cleveland, Ohio.—As a so-called cardiologist, I rise primarily to applaud the intensification and expansion of the surgical onslaught against coronary heart disease, with particular reference to the resourcefulness and ingenuity of Dr. Julian’s group and others. The catastrophic consequences of this relentless occlusive process in the coronary arteries, as manifested by increasing invalidism and exorbitant mortality, have attained such proportions that relative medical complacency should be replaced by generalized alarm to stimulate far greater efforts as warranted by the extent of the devastation associated with this disease. By its very nature, coronary heart disease defies “objective” evaluation, and hence the validity of any medical or surgical treatment is necessarily difficult to establish, keeping in mind that controversy has continued to rage for years over many fundamental concepts in coronary disease.

In our desire to achieve the “direct” surgical approach to this disease, we must not disregard certain fundamental (but neglected) considerations with regard to the pathophysiology of coronary heart disease. Unfortunately, the remarkable technical accomplishment of coronary arterial anastomosis and of coronary endarterectomy is of relatively little significance as compared to the more formidable problems inherent in the “direct” approach. Presumably, contrast visualization of the coronary arteries in man will eventually be feasible (if somewhat dangerous); thus, in a patient with clinical symptoms of coronary heart disease, “localization” of obstructions in the coronary circulation will allow the surgeon to select at least the most “significant” diseased segment for the “direct” approach. (I do not believe we can anticipate the application of this procedure to more than one or perhaps two separate obstructions in a given patient.) Unfortunately, it is axiomatic that there is no obligate relationship between the extent and location of the occlusive process in the coronary arteries and the clinical consequences thereof. Only “moderate” stenosis of one artery may result in death; on the other hand, chronic multiple complete occlusions of two or more arteries may produce no symptoms and no myocardial damage. In a fundamental sense, the patient’s symptoms are not due to the actual occlusive process in the coronary arteries, but to the response of the myocardium.

In a patient with coronary heart disease, the unwarranted assumption that the functional disturbance is due to a given obstruction of a coronary artery is most ill-advised, and
may have disastrous consequences. Restoration of flow into a given area of myocardium may not only have no beneficial effect, but, if flow is restored to a scar in a previously infarcted region, preferential perfusion of a low-resistance area may deprive viable myocardium of critically needed blood, so that a deleterious effect is produced.

The patient with coronary heart disease has a precariously balanced circulation; adequate intercoronary collaterals may maintain the dynamic integrity of the heart almost indefinitely. Any procedure which enhances the production of collaterals in advance of the occlusive process will be remarkably effective. On the other hand, a "direct" approach, which may actually disrupt this balance, may have catastrophic consequences.

DR. FRANK GERBODE, San Francisco, Calif.—I want to discuss Dr. Bahnson’s paper. We have not routinely measured intra-atrial pressures during operation for the closure of atrial septal defects, but I believe this may be quite important in some cases. We have had at least three instances of left ventricular failure in our atrial closures. Two patients developed failure after closure of rather large septal defects with low pulmonary vascular resistance. Both required hospitalization for some weeks after closure of the intra-atrial septal defect, but fully recovered. This means, therefore, that one must be on the lookout for this later, after the immediate postoperative course. We have been very careful about transfusing patients during and immediately following operation for those reasons. The one death in our last 35 atrial closures was in a poorly developed child who had an atrophic left ventricle and a considerably smaller mitral than tricuspid valve.

DR. ORMAND C. JULIAN, Chicago, III.—I should like to make two comments, one on Dr. Moore’s mention of thrombosis. One of the results of our work seems to indicate that, if the long mammary artery was used, thrombosis is a complication, whereas if the small homogeneous graft, larger in diameter, is used, thrombosis did not occur. Dr. Brofman has raised the problem of possible clinical use of this technique if we could once get it to work in 100 per cent experiments. Where would the end of the graft be implanted? There would be two possibilities, one being that one should implant the graft distal to the obstructed area, just as we do in the case of lower extremity arteriosclerosis; the other might be that one should use the graft to increase the pressure proximal to the obstruction wherever it may be, perhaps even the left coronary artery in toto being the recipient of this grafted blood supply.

DR. ROBERT P. GLOVER, Philadelphia, Pa.—I should like to thank the several discussants. It must be obvious that in the early stages of this program of internal mammary artery ligation for increasing myocardial blood flow, neither authors nor discussants have much experience to fall back upon. The paper was given not only to present our initial attempts to corroborate the findings of the Italian workers but also to stimulate interest and thinking in the complex field of coronary arterial disease. It is quite understandable that Dr. Viseberg might find this procedure objectionable on the surface it seems to be diametrically opposed to his procedure of implantation of the very same artery into the myocardium. I must emphasize the fact that I am not promoting this procedure, as he has implantation of the mammary artery, but merely trying to find out whether it has value. Undoubtedly in a condition so insidious and variable as coronary artery disease evaluation will take years.

Dr. Thompson is to be particularly commended for his attitude and well-spoken comments. His procedure of pericardial poudrage has been used by him and others for years. Undoubtedly it has merit yet he has taken the trouble to try this newer and even more innocuous method. I agree with his observation that, ultimately, mammary artery ligation as presented may find its proper place as an adjunct to other procedures. In the meantime, however, it is essential to evaluate it extensively, both experimentally and clinically, on its own merits as a single operative procedure.

DR. G. RAINNEY WILLIAMS, Baltimore, Md.—In answer to Dr. Morrow’s question with regard to digitalization of patients with elevated left atrial pressures at the time of operation, we have not digitalized such patients unless further clinical evidence of failure was present. Patients with atrial septal defects, without failure, have not been digitalized preoperatively as digitalis is said to increase the left-to-right shunt.

We should like to thank the other discussants for their comments.