Original Communications

A CONSIDERATION OF SOME OF THE PROBLEMS IN CARDIOVASCULAR SURGERY

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DURING the last several decades there has been a progressive increase in deaths attributable to cardiovascular disease. It is estimated[1] that 44 per cent of all deaths in the United States in 1948 were due to disorders of the heart and vessels, and that there are at present nine million people in this country with some type of disease of the cardiovascular system. As the length of life increases, derangements of this system assume added importance. Hypertension, arteriosclerosis, and rheumatic fever make up the great bulk of cardiovascular problems, and, unfortunately, much remains to be learned about the etiology as well as the prevention and treatment of these diseases. It is to be hoped in the future that appropriate preventive measures will greatly reduce the mortality and morbidity of cardiovascular disease in general. Even if this hope is realized surgery will still have a role to play, particularly in the treatment of the complications of a mechanical nature.

Excluding peripheral vascular diseases such as arteriosclerosis, Raynaud's disease, Buerger's disease, and varicose veins, the major proportion of cardiovascular cases now amenable to surgery are of congenital or traumatic origin. Trauma, the cradle of surgery, will continue to contribute its quota, even if world conflicts should disappear. Preventive measures such as the possible elimination of rubella may result in a decrease in the incidence of congenital heart disease, but the chances are that congenital disorders will be of fairly frequent occurrence for centuries to come.

It would be unfair to the field of cardiovascular surgery to judge its future potentialities on the basis of present accomplishments, since this field is as much an advancing one as is that of preventive medicine and that of nonoperative therapy. For example, a fundamental advance which would solve the problem of homotransplantation of tissue would open up great potentialities in cardiovascular surgery.

This lecture is not intended to be an all-inclusive consideration of the subject. For example, peripheral vascular disease and portal hypertension will

not be discussed. Greater attention will be devoted to congenital heart disease because of the recent advances in this field. I have arbitrarily divided the disorders to be considered into three groups: (1) those in the treatment of which surgery may bring about good or excellent results, (2) those in which surgical treatment may cause moderate improvement, and (3) those in which surgical treatment is of doubtful value or is as yet undeveloped. There is a justifiable difference of opinion as to the group in which some of the disorders should be placed. The main emphasis will be on unsolved problems rather than on accomplished facts.

The conditions will be discussed in the following order:

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I. DISORDERS IN THE TREATMENT OF WHICH SURGERY MAY ACCOMPLISH GOOD
OR EXCELLENT RESULTS

A. Acquired Lesions.—

1. Constrictive Pericarditis: This is now a well-recognized clinical entity which usually presents few difficulties in diagnosis. It is often impossible to determine the cause. In most such cases it is likely that the tubercle bacillus

is the offender. The use of chemotherapeutic and antibiotic agents in the prevention and control of infections will probably result in a reduction in the incidence of constrictive pericarditis.

The fundamental surgical problem remains the same as that enunciated by Delorme,[2] namely, the removal of the scar which is constricting the heart and in some cases the great blood vessels. It is necessary to remove the scar overlying the epicardium as well as that which lines the pericardium.

Prior to the discovery of streptomycin and similar antibiotic agents, it was held that patients with tuberculous constrictive pericarditis should not be operated upon until the process became inactive. One of the reasons for this viewpoint was that operation subjected the patient to the dangers of a systemic as well as a localized spread of the disease. A second reason given was that it is extremely difficult to remove the shaggy exudate that is present in the subacute stage. It is the opinion of Holman[3] that antibiotic therapy may now allow one to operate with safety during the earlier stages of the disease. Certainly these agents will lessen the chances of spread of the disease, and possibly it will not be necessary to remove the tuberculous granulation tissue covering the surface of the heart. No one has yet had sufficient experience with the combined use of surgery and antibiotic therapy in the treatment of active tuberculous pericarditis to give an authoritative answer.

The types of incision that may be employed in decortication of the heart include a parasternal one with resection of costal cartilages, a median sternotomy, and an intercostal transpleural approach. At the present time I prefer the latter incision for it gives more adequate exposure than the others. A long left anterolateral incision is made overlying the fourth interspace, and the fourth and fifth costal cartilages are divided at their sternal attachments. Through this incision one can expose and decorticate both the left and the right side of the heart, including the superior and inferior vena cava. It makes little difference if the right pleura is torn, provided one recognizes the condition and maintains inflation of the lung. Recent experience has shown that thorough decortication usually causes a prompt decline in the venous pressure and general improvement of the patient. Our results with this incision are much better than those obtained when a parasternal extrapleural incision was used.

2. Wounds of the Heart: As has been true of developments in many fields of surgery, the treatment of wounds of the heart and blood vessels was the springboard for further advances in cardiovascular disorders. It is generally agreed that wounds of the heart should be operated upon if there is continuing bleeding to the outside or into the pleural cavity. Fortunately, various advances in surgery, including improvements in anesthesia, have decreased the mortality rate when operation is necessary. There is some difference of opinion how the patient with uncomplicated acute cardiac tamponade without continuing bleeding should be treated. Dr. Ravitch and I[4] are of the opinion that open operation should be deferred until the effects of at least one pericardial aspiration have been determined. It has been our experience that the majority of patients will respond favorably to one or more aspirations, that

bleeding into the pericardial cavity will not recur, and that an open operation will not be necessary. Elkin and Campbell[5] have recently reported on the effectiveness of aspiration in the treatment of cardiac tamponade.

3. Systemic Arteriovenous Fistula: The majority of such fistulae are traumatic in origin, and hence are considered in this group. The diagnosis and the treatment of traumatic systemic peripheral arteriovenous fistula are clear-cut. The mortality in the many patients operated upon during World War II was amazingly low. It is generally agreed that the best treatment consists of eradication of the fistula with repair of the openings in the artery and vein, and that the second method of choice is quadruple ligation of the artery and vein with excision of the fistula provided the continuity of the artery can be interrupted without the loss of distal parts. There is some difference of opinion as to the choice of time of operation. If the artery involved is one which can be spared if necessary, one may operate as soon as the fistula is discovered. If sacrifice of the involved artery would subject its part to the danger of gangrene, an interval of a few weeks should be allowed to elapse following the injury in order to permit the further development of collaterals.

Most of the congenital peripheral arteriovenous fistulae involve bone, and here the problem of treatment may be an exceedingly difficult one. If the bony involvement is limited,[6] one may excise the segment with or without replacement with a graft. Unfortunately, however, the lesions are usually extensive, and multiple amputation may be necessary.

The subject of intracranial arteriovenous fistulae is a difficult one with many unsolved problems and will not be discussed.

4. Arterial Aneurysm: With better control of syphilis, the incidence of aneurysms should lessen. On the other hand, there will probably be an increase in those due to arteriosclerosis unless preventive and therapeutic measures decrease the incidence of this disease. Unfortunately, the arteriosclerotic process is usually not limited to the aneurysm itself, and this fact renders it important in surgical treatment to interfere as little as possible with the blood supply of parts distal to the aneurysm. The Matas endoaneurysmorrhaphy, the wiring procedure as used by Finney and others and further advanced by Blakemore, and the placing of irritating cellophane around the sac have in common the advantage that they need not interfere unduly with the collateral arterial pathways around the aneurysm. This is particularly true of the wiring procedure. Blakemore[7] now treats some aneurysms by constricting the artery proximal and distal to the aneurysm with polythene tape, thereby reducing the pulse pressure, and by heating wire introduced into the sac. The relief of pain and the abolition of respiratory difficulty that may follow the wiring of a thoracic aneurysm is a dramatic accomplishment. If the results that are being obtained by Gross[8] and others on the homotransplantation of aortic grafts continue to be encouraging, it is likely that the procedure can be used in conjunction with excision of the aneurysmal sac when the neighboring segments of aorta are not grossly diseased. Lam[9] has inserted an

aortic graft without excision of the sac. The development and use of improved plastic materials may be helpful in the treatment of aneurysms.

5. Mitral Stenosis: It may seem unduly optimistic to place this disorder in this group, but I do so because of the surprisingly good early results that are being obtained at present. Most cases of mitral stenosis are due to rheumatic fever, and it is probable that great advances will be made in the prevention or treatment of rheumatic infections. Even without further advances, it is likely that the use of antibiotic and other agents which are available will reduce the repeated attacks of infection and thereby lessen the incidence of mitral stenosis. On the other hand, many attacks of acute rheumatic fever are unsuspected or undiagnosed and the later development of some chronic disorder may give the first unmistakable clue to an earlier attack of an acute infection. For this reason it is likely that there will continue to be a moderate number of cases of mitral stenosis despite preventive and therapeutic advances.

Following the discouraging attempts[10, 11] which were made to treat mitral stenosis by surgical methods in the third decade of the present century, it is largely to the credit of the late Horace Smithy,[12] Bailey,[13] and Harken[14] that the problem was revived several years ago. In view of the present encouraging results, in some of which the procedure consists of dilation of the stenotic valve with the finger, it is difficult to understand why the successful use of this method by Souttar[15] in 1925 did not stimulate greater interest at the time. Whereas Cutler and his associates believed that a segment of the stenotic valve should be removed, and a similar view was held by Smithy, both Bailey and Harken are of the opinion that dilation of the valve and division or tearing of the commissures produce an opening of adequate size and that mitral insufficiency does not result. Both Bailey and Harken prefer an approach through the left auricular appendage as was advocated by Allen and Graham, and there is no doubt that this is preferable to that through the left ventricle. Bailey employs a knife which is attached to the index finger for division of the commissure region. Harken attempts to fracture or tear the commissure region with the index finger, and if this fails he divides it with a knife. It is our experience that in most of the cases one or both of the commissure regions can be torn with the index finger and that the use of a knife is generally unnecessary. Bailey does not favor the making of much pressure with the finger because of fear of tearing one of the valve leaflets. We have not as yet had the unfortunate experience of tearing one of these. It should be emphasized that it is not always possible to tear the commissure region and that one should have a proper cutting instrument available.

The early results that are being obtained in the treatment of mitral stenosis by direct attack on the valve are surprisingly good. The subjective improvement of the patient is usually greater than the alteration in the measurable dynamics of the circulation. It is too early as yet to know what the long term results will be, but even the improvements of the duration that have been recorded to date justify the operation. Subsequent observations may show that there is a gradual reformation of the stenosis. If such is the case, it may

be advisable to remove a segment of the valve, the plan of attack which was used by Cutler, Graham, Smithy and their associates. Since there are still unsolved problems, it is important that efforts to produce mitral stenosis in experimental animals be continued.

In addition to recent progress in the direct attack on the stenotic mitral valve, efforts have been made to prevent bouts of pulmonary edema in this condition by the anastomosis of systemic and pulmonary veins as performed by Sweet[16] or the making of an interauricular defect as advised by Harken.[17] It is obvious that these shunting procedures should not be used if there is a very tight stenosis, indicated by small left ventricular output and inability to increase it with exercise, for a further reduction in output caused by the shunt will not be tolerated. There are probably some cases in which some such procedure will be helpful, but in general it would appear that the surgical treatment of mitral stenosis should consist of a direct attack on the stenotic area itself.

Much probably remains to be learned about the choice of patients in whom a direct attack on the mitral valve is indicated. According to Andrus, with whom I have been associated in this work, the indications for a direct attack on the stenotic mitral valve are as follows: Symptoms and signs of pulmonary engorgement—dyspnea and cough on exertion, paroxysmal dyspnea, pulmonary edema, or frank hemoptysis. The most promising cases are those without enlargement of the left ventricle or prominent enlargement of the left auricle; usually these are signs that mitral insufficiency contributes significantly to the patient's disability. In general, there should be predominantly a stenosis, although a small degree of insufficiency is not a contraindication to commissurotomy. If one uses fairly rigid criteria in the selection of patients, the mortality associated with operation is fairly low. One of the twenty-five patients upon whom we performed a commissurotomy died postoperatively. An additional patient who was found at exploratory thoracotomy to have an obliterated auricular appendage, no commissurotomy being performed, succumbed subsequently from a cerebral embolus.

B. Congenital Lesions.—

6. Patent Ductus Arteriosus: Although a procedure for the correction of patent ductus arteriosus had been suggested many years previously by Munro[18] and had been attempted by Strieder,[19] it is to the everlasting credit of Gross[20] that he was the first to close a patent ductus successfully. The typical case is not difficult to diagnose, and technical procedures are available for obtaining a permanent closure of the ductus with a low mortality. In view of these facts, and because of the dangers of allowing a ductus to remain patent, it is my belief that operation is indicated for all children beyond two or three years of age who have an obviously patent ductus arteriosus. However, it is often difficult to be certain of the diagnosis in infants. Furthermore, Dammann[21] and Keith[22] have recently called attention to the fact that some patients with a very large patent ductus and associated pulmonary hypertension

may not have a typical machinery-like murmur. In some of these patients the presence of a large patent ductus has been confirmed by thoracotomy. There is some difference of opinion whether one should divide the ductus and close the two ends as preferred by Gross[23] and others, or whether one should close the undivided ductus by the use of multiple suture ligatures placed as far apart as possible. My associates and I prefer the multiple suture technique[24] because it seems to us that it is easier and safer. The desideratum is permanent closure of the ductus, and we are aware of only one instance of recanalization in the approximately 300 patients in whom we have used this method.[25] The exception was a child of five who had a ductus which measured 16 mm. in diameter.

Whereas the operation for closure of a patent ductus is not a very dangerous one in most children and young adults, the procedure becomes more difficult and dangerous in the older age groups. Pulmonary hypertension with disease of the walls of the pulmonary artery and ductus increases the likelihood of uncontrollable bleeding from these structures. In the older patients with an enlarged heart and pulmonary hypertension we use a long posterolateral incision instead of the anterolateral incision which is preferred in the less complicated cases. In the occasional patient with extreme pulmonary hypertension in association with a patent ductus there is some flow of blood from the pulmonary artery to the aorta. This reverse flow of blood is particularly apt to occur if the patent ductus is attached to the aorta distal to a coarctation of the aorta. Closure of the ductus is especially dangerous in patients with severe pulmonary hypertension and a reversal of flow through the ductus. In addition to the hazards of hemorrhage, there is the possibility that closure of the ductus will result in a sudden increase in the diastolic volume of the right ventricle and undue distention of that chamber.

Now that the diagnosis of the patent ductus can be made in most instances with accuracy and the operative technique is perfected, it is to be hoped that the condition will be recognized and treated in childhood and that in the future fewer cases will be encountered in adults.

An aortic septal defect providing a communication between the aorta and the pulmonary artery may give signs and symptoms similar to those of the usual patent ductus. A small aortic septal defect may be closed by existing techniques, but the large one extending to the base of the heart presents problems that have not been satisfactorily solved.

7. Coarctation of the Aorta: Fortunately there is usually no difficulty in the diagnosis of coarctation of the aorta. If there is any reason to suspect that the coarctation is at an unusual site, angiocardiography or aortography should be used in demonstrating the lesion. It is not necessary to use these radiological methods in the majority of cases.

The proper treatment of the adult type of coarctation, the common form in which the constricted segment is short, is essentially that which was first employed successfully by Crafoord,[26] and consists of excision of the stenotic or atretic segment and an end-to-end anastomosis of the aorta. It is desirable

to preserve the dilated intercostal arteries. There is some difference of opinion as to the type of suture that should be used in making the anastomosis. We usually employ a continuous everting suture of silk which is interrupted at several points. An overhand noneverting suture also gives satisfactory results. Interrupted sutures probably cause the least disturbance of growth of the anastomotic site and should be used in young children. Several recent experimental studies indicate that the size of the lumen at the site of the anastomosis increases somewhat with the growth of the subject even though a continuous suture is used. This brings up the question as to the age period in which the operation should be performed. I think the ideal period is from six to fifteen years of age. Complications in connection with coarctation rarely occur before the age of six, although in one of our patients hemiplegia developed at the age of two years. The aorta of a child of six is usually of sufficient size to provide a good lumen following anastomosis even though subsequent growth of this site may not be great. The employment of interrupted sutures will increase the likelihood of growth. I have no strong convictions as to the upper age limit at which operation should be advised. We have operated upon a number of patients in their thirties and one in the early forties with good results. On the other hand, I am inclined to think that a patient past forty who has no serious symptoms or complications save for the hypertension accompanying the coarctation should not be operated upon.

The treatment of the infantile type of coarctation in which the constricted segment is long presents a more difficult problem. The same may be said for the occasional case of the adult type when the proximal or distal segment is hypoplastic for a short distance. I think a good result will be obtained in most cases by the use of the left subclavian artery[27] to bridge the narrowed segment if the procedure is performed properly. This means that the constricted segment should be excised in toto, and the proximal end of the aortic segment should be closed with an arterial suture; the left subclavian artery should be cut across proximal to its initial branching, and should then be rotated downwards in such manner that there is no sharp twisting or angulation, and its end should be anastomosed to the distal end of the aorta. If one performs the operation without removing the constricted aortic segment and closing the proximal end of the aorta and without proper rotation of the subclavian, a good straight channel for the flow of blood is not obtained. Usually the subclavian artery is almost as large as the distal aorta, and there is little difficulty in performing a good union. The other alternative is the employment of a homograft of aorta as used by Gross[8] to replace the constricted segment of the aorta. The early results in the experience of Gross have been amazingly good. It is too early as yet to know how the homograft will weather the passing of time. The chances are that it is replaced by the recipient's own tissues. Disadvantages of this procedure are that it is difficult to obtain proper grafts and that two arterial anastomoses have to be performed. The severe constriction of the aorta that is present in the infantile type of coarctation should usually be discovered in childhood, and the use of

either the transposed subclavian artery as a substitute for the segment or the employment of homografts should give fair results. Unfortunately, the stenosis may be proximal to the origin of the left subclavian artery and may be associated with other congenital defects.

The question remains unsettled why some patients who are operated upon for the relief of coarctation do not have the desired decline in blood pressure. Fortunately, this is the exception rather than the rule. Among the possibilities is the fact that even after operation the aortic lumen in some patients is still not as large as it should be. This is particularly likely to be the case when the lumen of the proximal aortic segment is smaller than normal. Another possibility, if the kidney is involved in this type of hypertension, is that renal changes may persist after operation. Still another possibility is that irreversible alterations in the arterioles may have occurred in connection with the hypertension. I think that the number of disappointing results will decrease as additional experience is gained by the performance of larger and more perfect anastomoses. We are fairly well pleased with the results of operations on 104 patients with coarctation.

8. Pulmonary Stenosis and Atresia: The most frequent example of this type is the typical tetralogy of Fallot.[28] Other types of malformations which may be associated with pulmonary stenosis or atresia include tricuspid atresia, single ventricle, pseudotruncus arteriosus, dextrocardia or dextrorotation of the heart, situs inversus viscerum with levorotation, and valvular pulmonary stenosis with intact ventricular septum.

The typical case of *tetralogy of Fallot* presents few difficulties in diagnosis. There is need for more accurate appraisal of the degree of overriding of the aorta, the length of the infundibular stenosis, and the size of the infundibular chamber since this information may alter treatment, as will be discussed later. Patients with excessive dextroposition of the aorta may not do well following the creation of an artificial ductus. With the increase in the blood flow to the lungs there will obviously be an increase in the amount of blood returning to the left side of the heart. Since in cases of extreme dextroposition the aorta receives blood mainly from the right ventricle unless the interventricular defect is large, the left ventricle may be unable to empty itself properly and pulmonary engorgement and edema may develop. One possible means whereby the degree of overriding might be determined more accurately is by the injection of radiopaque material through a catheter placed in the right ventricle and the taking of repeated roentgenograms. The possible value of this procedure will be determined only by multiple trials. If an extreme degree of overriding were proved to be present, one would certainly not wish to create a large artificial ductus. Perhaps some day a safe means will be available for increasing the size of the interventricular defect. This could be done at the same time that the ductus is made.

It is largely as a result of the observations of Brock[29] that the efforts toward a direct attack on infundibular stenosis rather than the creation of an artificial ductus have been stimulated. The direct attack is preferable to the

anastomotic procedure, on theoretical grounds at least, because it reduces the shunt through the interventricular defect at the same time that it increases the pulmonary flow, and it does not add another anomaly to those already present.

Some idea as to the size of the infundibular chamber and the length of the stenosis may be obtained by angiocardiography. The information might be more exact if the diodrast were injected through a catheter introduced into the right ventricle. In addition to the information gained in preoperative studies, one can obtain some idea as to the size of the infundibular chamber by exposing this region at the time of operation. There are certainly many cases of the tetralogy of Fallot in which there is a long infundibular stenosis and in which a direct attack on the stenosis is not feasible. There are probably a moderate number of cases with a short diaphragm-like zone of stenosis and a large infundibular chamber in which a direct attack is possible. As indicated previously, there is need for more accurate means for determining the exact condition of the lesion. In some patients with a short infundibular stenosis it is not difficult to introduce dilating and cutting instruments through an incision in the right ventricular wall and through the stenotic area, as we have done in a few cases following the lead of Brock. It is much more difficult and dangerous to excise part of the stenotic area as performed by Brock[30] and Glover,[31] and others. Technical improvements are needed in this respect. Furthermore, the question remains unsolved whether such a stenotic area in the heart muscle which is treated by dilation and incision or excision will subsequently reform. It is my opinion that improvements in accuracy of anatomic diagnosis and in the technique of intracardiac procedures will result in an increasing number of direct attacks on infundibular stenosis. At the same time Dr. Taussig and I believe that the majority of these patients will have to be treated by the creation of an artificial ductus. Certainly this latter procedure rather than a direct attack offers hope of improving the patient with pulmonary atresia. In this connection it should be mentioned that simple exploratory thoracotomy with the subsequent formation of adhesions and the resulting additional collateral blood supply to the lungs seems to improve the condition of some patients in whom the pulmonary artery is not of suitable size for an anastomosis. The number of collateral channels is probably increased by removing the parietal pleura as recommended by Barrett.[32]

The techniques that are available for the creation of an artificial ductus are well developed, and the results are amazingly good in the typical tetralogy of Fallot. Dr. Taussig[33] is of the opinion that the occasional patient who does not do well following a satisfactory anastomosis does not have an uncomplicated tetralogy of Fallot. We perform an end-to-side, an end-to-end or a side-to-side anastomosis between a systemic and a pulmonary artery, the choice depending upon the age and size of the patient and the observations at the time of operation. This choice has been considered in detail in a previous publication.[34] In the 20 odd per cent of patients in whom the aorta descends on the right, the incision is almost invariably made on the left, and an anastomosis performed between the left subclavian and the left pulmonary artery.

In patients in whom the aorta descends on the left, the incision is usually made on the left in infants, in adolescents, and in adults, and it is usually made on the right in children between the ages of two and twelve years. In patients in whom there is considerable doubt as to the diagnosis, for example when valvular pulmonic stenosis is suspected, the incision is made on the left in order that one may examine the structures at the base of the heart. When the incision is made on the left in the presence of a left descending aorta, the left subclavian artery is used for the anastomosis if it is of adequate size and length; otherwise the aortic side-to-side anastomosis as devised by Potts[35] is employed. We do not hesitate to use an end-to-end anastomosis between the subclavian artery and the divided distal end of one of the pulmonary arteries if the pulmonary artery is unusually small or if the condition of the patient is poor and one wishes to perform the procedure speedily and without traction on the mediastinum. Dr. Taussig and I consider the optimal time for operation to be between the ages of five and ten years. A second operation at a later period is less apt to be necessary if the child has reached the age of five or more before the initial operation is performed. Unfortunately, the condition of many infants and young children is such that earlier operation is required. The general philosophy of Dr. Taussig's cardiac clinic is, "The sicker the sooner."

The results of the creation of an artificial ductus in the treatment of pulmonary stenosis or atresia are not so good when there are associated malformations such as tricuspid atresia, single ventricle, dextrocardia or dextrorotation, or situs inversus with levorotation, as they are in the typical uncomplicated tetralogy. Tricuspid atresia is usually associated with a diminutive or absent right ventricle and some abnormality of the pulmonary orifice. Some patients with this condition are improved by the creation of an artificial ductus. Others do poorly, due in part at least to a resulting increase in strain on the left ventricle in the absence of an interventricular defect. Patients with tricuspid atresia and with only a small opening between the two auricles may be improved by the creation of a large interauricular defect. If one employs the method which Hanlon and I[36] devised for this purpose, one has to be very careful during the procedure not to occlude entirely the existing communication between the two auricles for more than a few seconds. Patients with a single ventricle respond very poorly to the creation of an artificial ductus, and the operation should not be advised if the diagnosis can be made and if the patient is getting along moderately well. Patients with dextrocardia, or dextrorotation of the heart, or situs inversus viscerum with levocardia are likely to have a variety of malformations and are likely to respond unfavorably to the creation of an artificial ductus.[37]

Valvular pulmonary stenosis with or without a communication between the two auricles and with an intact ventricular septum occurs approximately 10 per cent as often as the tetralogy of Fallot. The work of Brock,[29] of Sellors,[38] and of others has added materially to the diagnosis and treatment of this condition. Cyanosis is present in about 70 per cent of the cases and is due to a right to left

shunt through a patent foramen ovale or an auricular septal defect. The pooling of diodrast as observed on angiocardiography in the pulmonary artery distal to the valvular stenosis is an important clue in diagnosis. The pressure in the right ventricle is significantly elevated and may exceed that in the left ventricle. Bing recorded a right ventricular pressure of 223 mm. Hg in one of our patients. The conditions observed at the time of operation are characteristic. There is definite right ventricular hypertrophy, usually a poststenotic dilatation of the pulmonary artery, a jet of blood can be felt striking the wall of the pulmonary artery, and usually the dome-shaped pulmonary valve can be palpated. Patients with valvular pulmonary stenosis are not improved by the creation of an artificial ductus. It is true that cyanosis if present is lessened, but these patients subsequently have right-sided heart failure.

We have followed essentially the technique of Brock in treating valvular pulmonary stenosis.[39] The approach is through an incision in the right ventricle, and the valve is cut and dilated. The Brock flat valvulotome with a probelike end and two sharp cutting edges is a very satisfactory instrument. Even so, the umbrellalike instruments recently developed by Potts present some advantages. In general we have been greatly pleased with the results obtained but a number of questions are as yet unanswered. Among these are the following: (1) Why is it that the blood pressure usually declines and the heart may fail shortly after opening the chest and exposing the heart? (2) Why is it that the pressure in the right ventricle may not fall to normal following division and dilation of the valve and in the presence of definite improvement in the condition of the patient? (3) Should operation be advised prior to the development of great incapacity and of right ventricular hypertrophy and dilatation? (4) Will extreme right ventricular hypertrophy, once developed, ever disappear entirely following operation? (5) Should the stenotic valve simply be incised and dilated as is done at present, or should a segment of the valve be excised? (6) When the valve is incised and dilated, do the incisions in the leaflets remain open or do they partially grow back together?

A longer postoperative follow-up of the patients and a repetition of the studies will probable supply an answer to some of the above questions. It is known that patients with great enlargement of the heart are those in whom difficulty is most likely to be encountered during the operation, and I believe that patients should be operated upon in advance of this enlargement if the condition is diagnosed. It has been observed in some of our patients that the size of the right side of the heart has decreased postoperatively, but this may be due to a disappearance of the dilatation rather than to a regression of the hypertrophy. The finding of Dr. Bing[40] that the pressure in the right ventricle usually does not fall to normal in the first few months after operation may be explained on the basis of inadequate division and dilation of the valve or possibly on the basis of partial obstruction at the pulmonary orifice by the greatly hypertrophied right ventricular muscle. I have already commented on the fact that patients show decided improvement postoperatively despite the persistence of some right ventricular hypertension. The valve in congenital

pulmonic stenosis is only moderately thickened, and I believe that incision and dilation will result in a permanent increase in the size of the opening. It appears likely that endothelium grows over the incised area and that the opening remains patent. If such should prove not to be the case, it will be advisable to remove part of the valve as has been performed by Varco.[41] No ill effects attributable to pulmonary insufficiency have been observed in any of our patients subjected to pulmonary valvulotomy, and it would seem that patients should withstand removal of part of the valve without ill effects.

9. Pulmonary Arteriovenous Fistula: This condition is being diagnosed with increased frequency and should be suspected in cyanotic patients in whom there is no evidence of disease of the heart itself. In doubtful cases, angiocardiography is helpful in diagnosis. Evidence of abnormal communications between pulmonary arteries and veins may be late in developing. Unfortunately, the communications are usually multiple and parts of both lungs may be affected. Treatment consists of excision of the diseased areas; in some instances a wedge-shaped resection will suffice. If a lobe is extensively involved it should be removed. One should not, however, remove an entire lung unless there are no normal segments present for fear that evidence of abnormal arteriovenous communications will subsequently appear in the remaining lung. One of the most dramatic results which I have witnessed in surgery followed the removal of a lobe of the lung containing multiple arteriovenous communications from a 28-year-old woman. Total incapacity was replaced by perfect health. The saturation of the arterial blood with oxygen is now normal and there has been a gain of 36 pounds in weight.

10. Anomalies of the Aortic Arch: Anomalies of the aortic arch and its major branches occur frequently in patients with congenital heart disease, but only a few of these in themselves cause any difficulty. A small proportion of these anomalies interfere with breathing or swallowing or both due to pressure on the trachea and esophagus. This difficulty is particularly likely to be present if there is a bifid aorta, although the same symptoms may be caused occasionally by a retro-esophageal artery such as an aberrant subclavian. Neuhauser[42] has made important contributions to the diagnosis of this condition. Gross[43] and others have shown that the treatment consists of the division of the offending blood vessel. In the case of the bifid aorta, the smaller of the two arches should be divided and the two ends closed. The most interesting patient with a bifid aorta in our experience is a boy who had tetralogy of Fallot as well. Treatment consisted of division of the smaller arch and an anastomosis of its distal end to the left pulmonary artery. In general, surgical treatment of anomalies of the arch is satisfactory.

II. DISORDERS IN THE TREATMENT OF WHICH SURGERY MAY BRING ABOUT
MODERATE IMPROVEMENT

A. Acquired Lesions.—

11. Essential Hypertension: Whereas there is a high percentage of good results in the treatment of hypertension associated with coarctation of the

aorta, pheochromocytoma and unilateral renal disease, the outlook in essential hypertension is less promising. It is true that some patients with essential hypertension obtain an excellent result after sympathectomy, and one may therefore criticize the placing of this disorder in this group. I do so because it would seem, in view of follow-up studies, that the ultimate treatment of hypertension will not be by surgical sympathectomy. A recent report[44] of a thirteen-year period showed that 21 per cent of the patients were improved, but only 8 per cent had a normal blood pressure five years after their operation. At the same time, if medical measures fail, sympathectomy is the best that we have to offer at present. It is possible that when more is known about the cause of essential hypertension, some operation on the central nervous system will be helpful. It is more likely that ultimately a successful nonoperative form of treatment will be found.

B. Congenital Lesions.—

12. *Transposition of the Aorta and Pulmonary Artery:* This type of congenital malformation is rather common if one includes those instances in which death occurs shortly after birth. Since the aorta and the pulmonary artery are transposed but the systemic and pulmonary veins occupy their normal positions, there has to be some type of communication between the two sides of the heart if there is to be any chance of survival. Usually there is an interventricular defect, an interauricular defect, a patent ductus, or various combinations of these lesions.

Adequate means are available for the diagnosis of transposition of the great arteries. The problem of surgical therapy is an exceedingly difficult one and is far from solution. Not only is there an inadequate mixing of blood of the two circulations but there is also severe pulmonary hypertension. We have been fairly well pleased with the results that have been obtained in some of our patients in whom a large interauricular defect was created.[45] In some instances this was accompanied by the making of an artificial ductus of the end-to-end type. The results have been fair in children and very poor in infants. Some of our best results have been obtained in patients who had pulmonic stenosis as well as transposition of the great arteries and also in those in whom the transposition was of the Taussig-Bing type[46] in which the pulmonary artery overrides an interventricular defect. A smaller percentage of those children with a typical transposition have been definitely improved.

The problem of therapy has been made more difficult by inability to reproduce transposition in toto in the experimental animal. Actually Hanlon and I have produced by a multiple stage procedure what amounts to a total transposition, but the right ventricle was unable to carry on the systemic circulation effectively. These experiments are being continued.

It appears on first thought that one's efforts should be directed toward the development of methods which would allow one to cut across the pulmonary artery and aorta of the patient with transposition and to transpose or correct the position of these vessels, using a suture anastomosis. It is possible that such a procedure may be carried out after perfection of the artificial

heart and lung. Even if this proves to be feasible ultimately, there is still the problem that the coronary arteries could not be transposed and that they would continue to receive venous blood from the right ventricle. Almost certainly the oxygen content of this blood would be higher than before the positions of the aorta and pulmonary artery were corrected, and it is possible that it would suffice for the nourishment of the myocardium. On the other hand, it is known that infants with an anomalous origin of coronary arteries from the pulmonary artery usually do poorly. However, the pulmonary arterial pressure in these patients is low whereas it is high in those with transposition. Even if it becomes technically feasible to correct the position of the aorta and the pulmonary artery except for their orifices, and even if the myocardium should receive adequate oxygen from venous blood, there still remains the problem of the pulmonary hypertension. It is possible but not at all certain that it would disappear after correction of the position of the great arteries.

Since one could not alter the position of the coronary arteries even if one were able to correct the positions of the aorta and the pulmonary artery, perhaps the ideal would be the development of methods whereby the systemic and pulmonary veins could be transposed, leaving the aorta and the pulmonary artery in their transposed positions. If total transposition of the venous return could be accomplished successfully it would probably result in great improvement in the condition of the patient. The coronary arteries would receive arterial blood, and one's greatest concern would be with what would happen to the pulmonary hypertension. Unfortunately, an experimental complete transposition of the systemic and pulmonary veins is difficult if not impossible with existing techniques. Brown and I[47] have accomplished in the experimental animal a transposition of the pulmonary venous return, but this required the removal of most of the left lung and the making of a large interauricular defect. Anastomoses of veins to the auricular appendage or auricle are very likely to become occluded. Anatomic relationships add to the difficulties of producing complete transposition of veins, such as, for example, the fact that the inferior vena cava is some distance from the left auricular appendage. Transposition of some of the pulmonary veins can be produced without too much difficulty, and we used this method to a limited extent in our early efforts to treat transposition in patients. Unfortunately patients with transposition of the great arteries tolerate a partial alteration in venous return very poorly since it causes an imbalance between the two sides of the heart. The situation is totally different from that existing in the normal heart where the blood leaves from one side and returns to the other.

After this lengthy discussion it should be repeated that our best results in the treatment of transposition of the aorta and pulmonary artery have followed the creation of a large interauricular defect. We are encouraged but are not satisfied with the results, and better methods should be devised.

13. Anomalies of Venous Return: These anomalies may occur in either the pulmonary or the systemic circulation. In other words, some or all of the pulmonary veins may empty into the right auricle or into veins that drain into the

right auricle, and part or all of the blood returning through the venae cavae may empty into the left auricle.

If all the pulmonary veins drain into the right auricle or its tributaries, there must be an interauricular defect to make the condition compatible with life. In most patients in whom several but not all of the pulmonary veins drain into the right auricle or its tributaries there are associated malformations. In one of the patients studied by Dr. Bing[48] in whom several but not all of the pulmonary veins emptied into the superior vena cava but in whom there were no associated malformations, there was a great increase in pulmonary blood flow without an associated increase in the pulmonary pressure. The ideal in surgical therapy in such a case would be the anastomosis of the aberrant veins to the left auricular appendage or one of its tributaries. Almost equally good results would probably be accomplished by the less difficult procedure of making a large interauricular defect. Muller[49] has recently anastomosed the side of an anomalous left pulmonary vein to the left auricular appendage with a good result. Dr. Bing has found pronounced pulmonary arterial hypertension with strain on the right ventricle in two patients in whom all of the pulmonary veins drained into the right auricle through the superior vena cava. The systemic arterial oxygen saturation in these patients was only moderately depressed, and the main problem was the burden on the right ventricle. An attempt was made in one of these patients to correct the venous return but without success. The creation of a very large interauricular defect would probably have relieved the strain on the right ventricle. The larger the auricular defect the greater will be the opportunity for a right to left shunt.

As pointed out earlier, there is danger of spontaneous closure of an anastomosis between a vein and an auricle or auricular appendage. The anatomic structure is such that it is difficult if not impossible to anastomose the right pulmonary veins to the left side of the heart unless they actually pass across the midline and into the left chest. In some instances all of the pulmonary veins, right and left, do come together in the left side of the chest and enter the innominate vein or the superior vena cava by a common trunk. Under such circumstances it is important to realize that one is dealing with a common trunk and that total occlusion while one is performing an anastomosis to the auricular appendage would result fatally. If the trunk is large, it may be partially occluded while the anastomosis is being performed; otherwise one of its branches may be used for this purpose. As indicated previously, the implantation of the anomalous vein into the correct side of the heart is the ideal procedure, but the dangers of subsequent closure of the anastomosis and the difficulties of determining the exact anatomic picture may influence one to make a large interauricular defect if one is not already present.

Important anomalies of the venae cavae are less frequently observed. If an anomaly is discovered that is producing symptoms, the problems in treatment are similar to those that have been discussed in connection with the pulmonary veins; that is, transplantation into the proper side of the heart or the making of a large interauricular defect. Bilateral superior venae cavae occur

fairly frequently and are usually associated with other malformations. Usually both of the superior venae cavae enter the right side of the heart, but in some instances the left one drains into the left side. If this latter condition as well as the presence of a normal right superior vena cava is determined preoperatively, one may ligate the left superior vena cava in the course of attacking the associated congenital malformation.

III. DISORDERS IN THE TREATMENT OF WHICH SURGERY IS OF DOUBTFUL VALUE OR SUITABLE METHODS ARE NOT YET FULLY DEVELOPED

A. Acquired.—

14. Coronary Artery Disease: I hope that I am in error in placing this disorder in this group because if it is proved that many cases of coronary arterial disease are amenable to surgical therapy, it will constitute one of the greatest of all advances in medicine. However, it would appear that our chief hope lies in the prevention of arteriosclerosis, possibly by dietary measures.

It is largely due to the ingenuity and tenacity of Dr. Claude Beck[50, 51] that some surgical progress has been made in this most difficult field. In many experiments and some clinical trials, efforts have been made to protect the heart against coronary occlusion by the establishment of an extracoronary bed and of communications between the three main coronary arteries. The methods which have been employed include the grafting of adjacent tissues such as skeletal muscle, lung,[52] pericardium, and omentum onto the surface of the heart in an effort to supply blood to the ischemic myocardium from an outside source; the use of chemical or physical inflammatory agents applied directly to the heart in an effort to stimulate intercoronary communications; and more recently attempts to cause a backward flow of blood through the coronary veins and capillaries by a direct anastomosis between a systemic artery and the coronary sinus of the heart.[51, 53] Other efforts which have been made to improve the condition of the patient with coronary arterial disease and angina pectoris include thoracic sympathectomy for the relief of pain, ligation of the great cardiac veins, which may cause stasis and a better distribution of blood in the myocardium, total thyroidectomy, the implantation of a systemic artery into the myocardium, and the excision of infarcts of the heart.

The operation which appears to me to have the most likely possibilities for improving the circulation of the myocardium is the original one proposed by Beck[50] in which some tissue adjacent to the heart is placed against the heart and an inflammatory reaction is provoked by an agent such as talc. In addition to the fact that this is a less dangerous procedure than that in which an anastomosis between a systemic artery and the coronary sinus is created, it would seem that the chances for prolonged improvement in the circulation are greater. Whether one uses total, partial, or no occlusion of the coronary sinus at its ostium in conjunction with its anastomosis to a systemic artery, it is likely that the shunted arterial blood will ultimately find its way into the heart chambers through dilated venous channels in which the pressure is lower than that in the capillaries.[54, 55] I am skeptical of the value of the systemic artery

to the coronary sinus operation, despite the fact that Heimbecker and I[56] have shown recently by observation of capillaries and determinations of the oxygen content of blood that arterial blood can be made to flow backward through the veins and capillaries of the intestinal tract, for a brief period at least, under conditions which will not be detailed here. Fluids seek a low pressure route and blood will not flow through a high pressure zone for an extended period when small veins exist which are capable of dilation when subjected to increased pressure.

Beck has emphasized his belief that the operations which he has described will not eradicate the disease and that a complete cure is not to be expected. Since the average duration of life of patients who survive the first attack of coronary occlusion is approximately five years, it is extremely difficult to evaluate the effects of a procedure which is not curative, and thousands of trials will be required before a definite answer can be supplied. Even though it would appear that the possibilities of making real progress in the treatment of coronary disease by existing techniques are not great, the importance of the problem demands that experimentation be continued.

15. Aortic Valvular Stenosis: Aortic stenosis as an isolated lesion is rare and is more often acquired than congenital. Rheumatic fever is the most frequent causative agent. It is fortunate that the lesion is less common than mitral stenosis, for the surgical problem is thus far a more difficult one. Our efforts to treat aortic stenosis are limited to two cases and both resulted fatally from ventricular fibrillation. The first patient (who was to be operated upon by the late Horace Smithy) died as the incision was being made in the chest wall, and the second died shortly after the stenotic valve had been incised and dilated through an incision in the left ventricle. Bailey[31] has attempted to stretch the stenotic orifice by the use of a dilator introduced through the carotid artery and down the aorta, but he has now abandoned this method in favor of the ventricular approach.

Subaortic stenosis,[57] which is also rare, is usually a congenital lesion and is caused by the persistence of a band or membrane of connective tissue which lies immediately below the aortic valves. The problem of surgical therapy should be easier in this condition than in rheumatic aortic stenosis.

16. Insufficiency of Heart Valves: The problem of the surgical treatment of insufficiency of the various valves of the heart is still decidedly in the experimental stage and will probably remain there for some time. One could probably anchor in place an artificial mechanical valve by the use of existing techniques, but there is great doubt whether such a valve would function for an extended period. If one is to place accurately a graft of viable tissue such as pericardium, one should be able to suture with care in a dry field under direct vision, which means that an extracorporeal circulation is required. Furthermore, it remains to be determined whether such valves will live and function properly. This latter problem may present greater difficulties than those of artificial circulation. Gordon Murray and his associates[58] used vein segments, and Templeton and Gibbon[59] employed vein and pericardial grafts in

efforts to repair experimental defects in valves. The results are moderately encouraging, particularly with the use of pericardium, but the observation period is too short for critical assessment. Even if the results in partial replacement of the normal valve by a graft were practically perfect, the question is still unanswered as to the fate of a graft placed in a scarred area such as exists in an acquired insufficiency. The functioning of heart valves is an extremely complicated mechanism and is dependent upon the attachment of the chordae tendinae as well as other factors. At any rate, the problem of valvular insufficiency is an important one which it will be very difficult to solve.

I am aware of the fact that Bailey and his associates are placing a pedicled flap of pericardium across the left ventricle just below the mitral valve in the treatment of mitral insufficiency, and it is said that the early results are encouraging. A long observation period will be required for proper evaluation.

17. Intracardiac Tumors: Existing surgical methods are suitable for the excision of tumors of the surface of the heart. Unfortunately these lesions are usually metastatic. The proper treatment of tumors of the interior of the heart awaits the development of more accurate means of diagnosis as well as means either for good visualization with instrumentation of the interior of the beating heart, or perfection of the artificial circulation which will permit an open operation in a bloodless chamber of the heart.

B. Congenital.—

18. Anomalous Origin of Left Coronary Artery: In the rare anomaly in which the left coronary artery arises from the pulmonary artery the infants usually die of cardiac failure during the early months of life. This condition may occur as the sole anomaly. Bland, White, and Garland[60] have shown that the condition can sometimes be diagnosed by the finding by electrocardiography of inversion of the T waves in all three leads combined with low voltage curves.

There is a possibility but not a great likelihood that this condition may be treated surgically by severing the connection of the left coronary with the pulmonary artery and anastomosing its distal end to a systemic artery such as the subclavian. It would be necessary to perfuse the coronary artery with blood while the procedure is being performed. Smith and I have made a few attempts to carry out a similar procedure in experimental animals but thus far without success. The small size of the coronary artery in an infant renders perfusion and simultaneous anastomosis a difficult and dangerous undertaking.

19. Auricular Septal Defects: A defect of the auricular septum is a common lesion and frequently an isolated one. These defects vary greatly in size and position. The shunt of blood is usually from the left to the right auricle and hence there is usually no cyanosis. The shunt imposes an added load on the right side of the heart, and there is generally dilatation of the right atrium and ventricle and right ventricular hypertrophy. Failure of the right side of the heart may occur. In some cases the shunt is reversed, that is, from right to left, and cyanosis is present. In the absence of pulmonary hypertension

it seems obvious that a more balanced circulatory pattern would result from closure of an isolated auricular septal defect.

When pulmonary hypertension is present it may very well be that closure of an interauricular defect is contraindicated. The answer probably hinges on the question whether the pulmonary hypertension is primary or secondary, and if secondary, whether the hypertension would be lessened by closure of the defect. In the presence of marked arteriolar disease in the lungs the right ventricular pressure and consequently the right auricular pressure are elevated. As a result the shunt in the two auricles may be directed from right to left. Under these circumstances repair of the auricular defect would close the opening which may be preventing an excessive increase in pressure in the right auricle and may transform the condition into one resembling Ayerza's disease. Since the cause of pulmonary hypertension in association with an auricular septal defect is obscure and may be due to primary changes in the pulmonary arteries, I simply wish to emphasize the possibility that closure of the defect may be contraindicated. It is possible that closure of the defect combined with denervation of the lungs may be the treatment of choice. Denervation of the lungs without closure of the defect might make the condition worse.

Since there are many auricular septal defects unassociated with complicating factors, it is important that a satisfactory method for closing them be developed. Much experimental work along this line is being performed, such as that of Cohn[61] on the use of part of the wall of the auricle, and that of Swan[62] on the employment of the auricular appendages. One of the difficulties of the problem is that experimentally produced defects are prone to spontaneous closure, and it is difficult to gauge the effectiveness of various methods of repair. The method which Hanlon and I[36] devised for creating an interauricular defect usually results in one which does not close spontaneously, but its position is not the same as that of naturally occurring interauricular defects. Gordon Murray[63] made a preliminary report on his attempts to close interauricular defects by a blind method in which coarse suture material is passed through the heart in the region of the septum and is tied loosely. A somewhat similar method has been reported by Perianes,[64] I doubt whether any of the clinical or experimental methods which have been reported thus far will prove to be the answer to this problem. Good visualization of the interior of the auricles is desirable since the defects vary so greatly in size and position, and a wholly satisfactory method may have to await the perfection of the extracorporeal circulation.

20. The Lutembacher Syndrome: This syndrome consists of an auricular septal defect combined with congenital or acquired mitral stenosis and great dilatation of the pulmonary artery. It is well known that patients with auricular septal defects are prone to have rheumatic fever and mitral stenosis. The greatest strain in the Lutembacher syndrome is probably on the right auricle, the right ventricle, and the pulmonary artery, whereas the left ventricle and the aorta are spared except in the later stages. Because of the fact that the shunt is from left to right, there is usually no cyanosis. White[65] has pointed out that

some patients with this condition survive to old age, but this is the exception rather than the rule.

If surgical therapy is indicated, it would appear that the attack should be on the stenotic mitral valve rather than upon the auricular septal defect. If the flow of blood through the stenosed mitral valve is increased by commissurotomy, the left to right shunt would decrease, thus lessening the burden on the right side of the heart. If the mitral stenosis can be dealt with effectively, and if pulmonary arterial disease is not present, it would probably be desirable to close the interauricular defect if a safe and suitable method were available. In the presence of pulmonary arteriolar disease the defect should not be closed even if the mitral stenosis has been alleviated since the defect probably serves as a safety outlet in permitting some right to left shunt and prevents an excessive rise in pressure in the right auricle. As stated previously, the pulmonary artery in the Lutembacher syndrome is greatly enlarged. Even if one could reduce the size of this vessel by surrounding it with an irritating material such as cellophane, it is doubtful whether the patient would be benefited.

Approximately one year ago I performed a mitral commissurotomy on a patient ill with the Lutembacher syndrome. She survived the procedure, but her condition is little if any improved at the present time.

21. Ventricular Septal Defects: Defects of the ventricular septum are frequently observed lesions, they are difficult to diagnose with certainty, they vary greatly as to size, location, and associated symptoms if any, and many of them are compatible with long life. The shunt of blood is usually from left to right. Since this shunted blood returns to the left side of the heart, both ventricles take part in transporting it. In this respect the situation is different from that in interauricular defects with a left to right shunt in that the right ventricle has the entire burden of pumping the shunted blood.

The prognosis in patients with small ventricular septal defects is excellent, and surgical therapy is not indicated. It is in patients with large high defects in whom the large shunt causes changes in the pulmonary blood vessels and in those in whom for some reason a right to left shunt develops that closure of the defect might be considered. In patients in whom a large shunt is predominantly left to right, closure of the shunt would undoubtedly benefit the patient by protecting the pulmonary vessels and reducing the strain on the heart. On the other hand, if pulmonary hypertension and a right to left shunt are present, closure of the defect might result in an increase in the residual volume of the right ventricle, a rise in pressure in the right ventricle and pulmonary artery, and in right-sided heart failure.

Ventricular septal defects are hard to produce experimentally and are likely to close spontaneously, which makes difficult the problems of surgical methods of closure. As far as I know, no satisfactory method has been developed. Murray[63] made a preliminary report of his efforts to close ventricular septal defects in patients by a blind suture method. Because of the fact that the exact position of the defect varies widely from patient to patient and that some of the

defects are in close proximity to heart valves and to the orifices of major arteries, I do not believe that a blind method of suture is the answer to this surgical problem. The defects which produce symptoms are those that are closest to the heart valves and great arteries, and these present the chief difficulty in closure. As indicated previously, the low ventricular defect is a relatively benign lesion and closure is rarely if ever indicated. I am fairly certain that the proper surgical closure of ventricular septal defects that are producing symptoms will have to await the further development of methods of carrying on the circulation artificially which will allow under direct vision the careful placing and suturing of a graft of tissue such as pericardium in a position where there will be no interference with the action of heart valves or blockage of the orifices of important vessels. These technical difficulties added to those of accurate anatomic diagnosis indicate that it will be some time before this problem is solved.

   22. The Eisenmenger Complex: In the Eisenmenger complex there is a high ventricular septal defect; the aorta overrides this defect and thus receives blood from both the right and the left ventricle. It differs from the tetralogy of Fallot mainly in that there is no pulmonic stenosis. The pulmonary artery is usually very large, and pressure within it is high. This condition, which fortunately is not common, may not be detected in childhood since cyanosis may not appear until the time of adolescence or later.

   Serious changes in the pulmonary arterioles have been described in the Eisenmenger complex. Old and Russel[66] found the following: (1) medial hypertrophy; (2) an increase in elastic tissue with respect to both thickening of the pre-existing fibers and the presence of new fibers between the elements of the media; and (3) nonatheromatous intimal thickening with proliferation of fibrous tissue. These changes result in varying degrees of narrowing of the smaller pulmonary vessels. It has not been determined with certainly whether the pulmonary hypertension is primary or secondary, but it is very likely that it is secondary to the large high ventricular defect. If it were not for this increase in the pulmonary vascular resistance, most of the output of both ventricles would go to the lungs and the systemic flow would be inadequate. According to Bing,[67] the blood flow through the lungs in the Eisenmenger complex is essentially normal in quantity.

   The ideal operation for the treatment of the Eisenmenger complex would be the closure of the high defect before pronounced and irreversible alterations in the pulmonary arterioles occur. As indicated in the discussion of the treatment of other high interventricular defects, this operation should be performed in a dry field under direct vision, and a graft of some tissue such as pericardium should be used. The proximity of the aorta to the defect renders an attempt at blind closure too dangerous. After definite pulmonary hypertension and visible cyanosis have developed, closure of the defect might result in failure of the right side of the heart. Since the pulmonary blood flow in this condition is said to be normal, and since there is severe constriction of the smaller

pulmonary vessels, it is doubtful whether an artificial reduction in the size of the pulmonary artery would accomplish any good, and it might make the condition worse. This point, however, has not been determined. A significant reduction in the size of the main pulmonary artery combined with sympathectomy might result in improvement. Unfortunately, an entirely satisfactory method for the partial closure of large diseased arteries has not been developed, and there is always the danger of subsequent hemorrhage at the site if a sizable reduction in caliber is caused. All in all, the problem of the surgical treatment of the Eisenmenger complex and of high ventricular septal defects in general is a most difficult one. Its solution probably will have to await further developments in intracardiac surgery.

23. *Pulmonary Hypertension in Association with Congenital Heart Disease:* Pulmonary hypertension in association with a number of the congenital cardiovascular defects has already been discussed briefly. The question arises whether the alterations in the pulmonary arterioles are primary[68] or whether they are secondary to the congenital defect in the heart or great blood vessels. The future role of surgical therapy in the treatment of pulmonary hypertension in association with congenital heart disease is bound up so closely with this unsettled question of etiology that no dogmatic statements seem warranted. Even if it be granted that the pulmonary hypertension in the majority of cases is due directly to the alteration in circulatory dynamics caused by the cardiovascular defect, there is still the problem whether these changes in the pulmonary vessels will be reversed if the defect is corrected. These alterations in the pulmonary vessels are usually anatomic rather than vasospastic. It has been stated previously that in many cases the increase in the resistance in the pulmonary vascular bed serves as a safety mechanism in preventing an unbalancing of the systemic and pulmonary vascular systems. This is probably the case in the Eisenmenger complex, transposition of the great vessels, isolated ventricular septal defects, aortic atresia with patent ductus, the single ventricle in which both great arteries or the aorta alone arises from the rudimentary outflow chamber, and in some instances of truncus arteriosus. Even if the nature of the pulmonary vascular resistance were such that it could be lowered by sympathectomy without correction of the defect, severe circulatory disturbances would likely ensue. I am of the opinion that the pulmonary vascular changes are usually secondary to the cardiovascular defect and that the treatment should consist of a direct attack on the defect when and if a method is available. Naturally the best results will be obtained in those patients in whom vascular alterations in the lungs are not far advanced. If the primary defect can be remedied, it is possible that an additional procedure such as pulmonary sympathectomy will be helpful in reducing the pulmonary resistance, but there is no convincing evidence that the pulmonary vessels are under autonomic control. Unfortunately, surgical means are as yet unavailable for dealing with many of these intracardiac defects, and therapy will have to await further advances.

DISCUSSION

Many significant advances in cardiovascular surgery can be and will be made by the employment of existing methods and techniques, but many others will have to await the further development of methods for carrying on the circulation artificially in order that one may operate under direct vision in a bloodless field. Still others are dependent upon the solution of the problem of homotransplantation of tissues. This latter problem is more baffling than the former, and no real strides have been made. It is to be hoped that some agent will be found that will so alter the donor-recipient relationships that transplanted tissues will survive and function permanently. The work with cortisone and ACTH along these lines is not very encouraging.

Important advances have been and are being made in studies on methods for carrying on part or all of the circulation artificially. Many of the needs for and future applications of these methods have been mentioned in this paper in the discussion of individual lesions.

Kolff and others who have worked on the artificial kidney have demonstrated conclusively that it is possible to withdraw blood continuously, to pass it through an artificial circuit, and to return it to the body without harm to the patient. The blood is removed from an artery of the patient, who has received heparin, is passed through the artificial kidney, and is returned to one of the patient's veins. The problem is more complicated if one wishes to take over the cardiorespiratory functions because the use of the artificial lung presents greater complexities than does that of the artificial kidney. In order to take over the cardiorespiratory functions, it is necessary to heparinize the patient, to withdraw blood from a vein, to expose it to oxygen and allow carbon dioxide to escape, and then to pump the oxygenated blood into a peripheral artery. As a result of the work of Gibbon and many others, oxygenators that function with remarkable efficiency have been constructed.

PARTIAL CIRCULATION

The mechanical lung and pump as partial replacement for the functions of the heart and lungs should be helpful to patients having difficulty in oxygenating adequately the blood which is to enter the systemic arterial system. Among other conditions, the procedure should prove helpful in the treatment of patients with severe pulmonary edema which cannot be relieved by conventional methods of treatment.

The remaining remarks on partial circulation will deal only with replacement of the function of the right or the left side of the heart, the subject's own lungs being used for the oxygenation of the blood. If and when the artificial lung is perfected and proved to be safe, it is likely that the use of methods for replacement of the function of one of the two sides of the heart will be limited to experimental work, since the cannulation of the great blood vessels is in itself an extensive and a dangerous procedure. Furthermore, there are many intracardiac and extracardiac procedures in which it is desirable to have both sides of the heart bloodless.

Leeds, Gray, and Cook,[69] Sewell and Glenn,[70] and others have rendered the *right side* of the heart bloodless, except for coronary venous return, by diverting the blood from the superior and inferior venae cavae to a pump and then through a cannula to the pulmonary artery. As indicated previously, this required the insertion of at least two large cannulae. Since the coronary blood flow equals 5 per cent or more of the cardiac output, difficulties are presented in operations on the right side of the heart from the viewpoints of visibility and blood loss. If the outflow from the coronary sinus and thebesian vessels is to be collected and returned to the circulation, it should be debubbled. Sufficient experimental evidence has been accumulated to demonstrate that a unilateral pump can be opened and closed with recovery of the animal.

A unilateral pump to replace the function of the right side of the heart temporarily should theoretically allow one to operate on lesions of the tricuspid and pulmonary valves, including stenosis, atresia, and insufficiency, on Ebstein's disease, on the rare infundibular stenosis unaccompanied by a septal defect, and on some intracardiac tumors. The unilateral pump would not suffice for the closure of septal defects or for operations for other lesions associated with septal defects. The pump will probably find its greatest usefulness in permitting one to create various experimental lesions of structures of the right side of the heart.

Zollinger,[71] Kantrowitz,[72] and others have used a unilateral pump to substitute for the *left side* of the heart. The blood from the left auricle or the pulmonary veins is withdrawn through a cannula and it is then pumped through another cannula into a systemic artery such as the subclavian. It is more difficult to substitute for the left side of the heart than the right. One of the reasons for this is the likelihood of arterial air embolism associated with opening the left side of the heart. Furthermore, the coronary arterial flow is dependent, when the left heart pump is used, upon the competence of the aortic valve. Nevertheless, Kantrowitz has reported survival of cats following opening and closing of the bloodless left ventricle.

A unilateral pump to replace temporarily the function of the left side of the heart should theoretically allow one to operate on lesions of the mitral valve and on intracardiac tumors. The coronary arteries would probably have to be cannulated and perfused if one were operating for aortic stenosis or insufficiency. The unilateral pump would not suffice for the treatment of septal defects. The present results on the treatment of mitral stenosis are so encouraging that the employment of a pump in this connection seems unlikely. Because of the dangers accompanying the use of the left heart substitute, and because of its limited clinical usefulness as visualized at present, it would appear that this method will be employed mainly in the creation of experimental lesions.

TOTAL CIRCULATION

Wesolowski[73] and others have shown that *both sides* of the heart may be rendered relatively bloodless by using two pumps, one for the right and another for the left. The connections made would be similar to those described

under partial circulation; namely, one apparatus to withdraw blood from the venae cavae and propel it to the pulmonary arterial circulation, and another to withdraw blood from the left auricle or pulmonary veins and pump it to a systemic artery. Under these conditions the entire heart is deprived of blood except for the coronary flow, and various intracardiac procedures would be possible. It seems doubtful, however, whether the use of two pumps to replace the heart's functions will ever be a practical procedure in patients because the connections necessary between the two pumps and the patient make it too formidable. The method should continue to serve a useful purpose in experimental work.

Another method by which the function of the heart and lungs can be supplanted is by *cross circulation* between the recipient and a donor. This can be accomplished by having the donor's heart serving as the pumping mechanism, or mechanical pumps can be used. In either case, the donor's lungs would oxygenate the blood. Experiments indicate that the dangers to the donor are about as great as those to the recipient, and it would appear that this method will not have clinical application.

The last and best method is that in which an *artificial oxygenator* is used. The method involves cannulation of the venae cavae and passing of the venous blood through an oxygenator or artificial lung. The blood which has taken up oxygen and has lost carbon dioxide is then pumped to a systemic artery. Both sides of the heart are rendered relatively bloodless (if the aortic valve is competent) and intracardiac procedures such as operations on heart valves, septal defects, and intracardiac tumors are possible. The cannulation is simple as compared to that which is required when an oxygenator is not used. Those who are working in this field have had many problems to solve such as the prevention of damage to the blood and the prevention of air embolism. The two major problems remaining are the construction of a simple suitable oxygenator and the solving of the difficulties and dangers of operating on heparinized patients. Under the leadership of investigators such as Gibbon, Crafoord, Bjork, Kolff, Dennis, Glenn, Jongblood, and others, it is very likely that these problems will be solved. Only when this time arrives will it be possible to determine how well the diseased human heart will withstand having its functions taken over temporarily and incisions made into its chambers. It is probably fortunate that the dog's heart, because of its great tendency to fibrillate, presents a rigid test for the artificial circulation; the human heart presents less of a problem in this respect. It would seem that the initial clinical employment of the artificial heart and lungs for total replacement should be on patients with a usually fatal disorder such as congenital transposition of the aorta and pulmonary artery. The hearts of these patients withstand operative procedures remarkably well, and the use of the artificial circulation should give one an opportunity to divide and transpose the aorta and the pulmonary artery.

I hope it is obvious from my remarks that there are many unsolved problems in cardiovascular surgery. Many of these will be solved by modifications

in existing techniques. Others will have to await further fundamental developments. With the interest and enthusiasm that are being shown by competent investigators, the future development of this field of surgery seems assured.

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