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The INTERNATIONAL CONFERENCE OF PHYSICIANS was held in London from September 8 to September 12. The five sessions of the section of Cardiology were attended by four or five hundred physicians among whom were welcomed many distinguished visitors from overseas. Two of the sessions were joint meetings with the section of Pædiatrics and the section of Disorders of the Chest, respectively.

THE SURGERY OF CONGENITAL HEART DISEASE

September 9, 1947. Joint Meeting with Section of Pædiatrics

MALFORMATIONS OF THE HEART AMENABLE TO THE BLALOCK-TAUSSIG OPERATION

BY HELEN B. TAUSSIG,* Baltimore, Maryland

There are three types of malformation of the heart that cause persistent cyanosis. In the first group there is lack of adequate pulmonary blood flow, as for example in pulmonary stenosis and pulmonary atresia. In the second group there is difficulty in the direction of oxygenated blood to the systemic circulation, as in complete transposition of the great vessels. In the third group there is failure of the blood to be fully oxygenated in its passage through the lungs, as in the Eisenmenger complex. It is only the first of these three groups that is amenable to the Blalock-Taussig operation, and hence only this group is discussed in this paper.

The operation developed by Dr. Alfred Blalock is designed to increase the circulation to the lungs. This is accomplished by the anastomosis of the proximal end of one of the systemic arteries to the side, or distal end, of the right or left pulmonary artery. In order that the blood should flow from a systemic artery to a pulmonary artery the pressure in the systemic circulation must be higher than that in the pulmonary circulation. This is true in all cases of pulmonary stenosis or atresia. In such malformations a Blalock-Taussig operation virtually restores the condition to that existing before the closure of the ductus arteriosus. When there is pulmonary atresia, only rarely does the collateral

circulation develop with sufficient rapidity to enable the infant to survive the closure of the ductus arteriosus. Consequently, in such instances, if operation is to be life-saving, it must usually be performed before the ductus arteriosus undergoes obliteration. When, however, there is pulmonary stenosis, operation can usually be postponed until childhood.

The diagnosis of pulmonary stenosis or atresia is based upon the history, combined with certain clinical and radiological findings. The history of the development of cyanosis at an early age is a characteristic of this malformation. The position which the child assumes when tired is a diagnostic aid because most children who suffer from a reduction in pulmonary blood flow squat when tired.

The outstanding findings on physical examination are persistent cyanosis combined with a heart that is little, if at all, enlarged, a basal systolic murmur and a clear second sound. The electrocardiogram shows a right axis deviation. X-ray of the heart shows an absence of the normal pulmonary conus and upon fluoroscopy there is absence of pulsations of the hilar shadows.

The degree of cyanosis is subject to great variation. Most children are intensely cyanotic. There are, however, a few with a tetralogy of Fallot but with lips

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of normal colour and only slight cyanosis of the fingernails, who are extremely incapacitated. Upon exercise such children usually suffer from a marked fall in the oxygen saturation of the arterial blood.

The cardiac findings are also variable. A basal systolic murmur and a clear second sound are the rule. However, with extreme pulmonary stenosis there may be no murmur. Usually diastole is clear.

The exact diagnosis of the anatomical abnormality is not as important as it is to have the malformation amenable to surgery. The essential criteria for successful operation are: (1) the lack of adequate circulation to the lungs; (2) the availability of a systemic artery; (3) the existence of a pulmonary artery; (4) a pressure greater in the systemic than in the pulmonary artery; (5) the structure of the lungs such that the patient can survive the collapse of one lung and the occlusion of one pulmonary artery; and (6) a heart of such size and structure that it can adjust to the altered circulation.*

Although the existence of a pulmonary artery and of low pulmonary pressure are essential for successful operation, these facts are not always easy to determine prior to operation.

Fluoroscopy is of prime importance both for the visualization of the pulmonary artery and for determining the presence or absence of hilar pulsations. In infants who suffer from lack of adequate pulmonary blood flow, the lungs are usually exceptionally clear. In adults with a large pulmonary artery and high pulmonary pressure, the visualization of hilar pulsations when present is usually not difficult. In children, however, the vessels are smaller and the pulsations are correspondingly more difficult to see. In the rare instances of complete transposition of the great vessels, the pulmonary artery lies posterior to the aorta and there is a considerable amount of lung tissue overlying the pulmonary artery. Under such circumstances the pulsations in the pulmonary artery are difficult, if not impossible, to demonstrate.

Furthermore, the hilar shadows increase as the collateral circulation develops through the posterior mediastinal vessels. These shadows are caused by an aggregation of small vessels and when the observer's eyes are fully dilated appear as an aggregation of minute shadows, which never show expansile pulsations. Absence of hilar pulsations is a pre-requisite for the operation, as their presence is a strong indication of adequate or excessive pulmonary blood flow and is usually indicative of high

pulmonary pressure. Nevertheless, in the absence of hilar pulsations, the existence of a pulmonary artery cannot be determined with certainty prior to operation.

The occurrence of a continuous murmur calls for careful consideration. Such a murmur may be caused by the persistent patency of the ductus arteriosus or by the flow of blood through large vessels of the collateral circulation. The differentiation is important because in the former condition there is certainly a pulmonary artery and therefore operation is possible; whereas in the latter there may be no pulmonary artery in which case operation will be impossible.

As regards the size and structure of the heart, great cardiac enlargement is a contraindication for operation because should the operation cause further enlargement the patient would be unable to maintain compensation.

An over-riding aorta or some pathway for the direction of venous blood into the systemic circulation is essential because it is the direction of venous blood to the lungs that is of benefit to the patient. Indeed, the fundamental difference between the artificial ductus arteriosus in a tetralogy of Fallot and an uncomplicated ductus arteriosus or an arteriovenous aneurysm is that in the former the dextroposed aorta permits the direction of mixed venous blood to the lungs; whereas in the latter conditions only fully oxygenated blood is redirected to the lungs. In a patient with a tetralogy of Fallot, although operation may lead to a marked rise in the oxygen saturation of the arterial blood, the oxygen content of the arterial blood never reaches normal because of the over-riding of the aorta. The saturation of the arterial blood remains between 75 and 85 per cent, and upon exercise it drops to 70 per cent or less. Consequently the blood directed through the artificial pathway to the lungs is essentially that normally directed through the pulmonary artery to the lungs.

The simplest method for the demonstration of the dextroposition of the aorta is by the determination of the circulation time. A short circulation time, arm to tongue, of less than 10 seconds is strong presumptive evidence that venous blood is being pumped directly into the systemic circulation. In doubtful cases, angiocardiology may be necessary to determine the time at which the aorta is visualized.

Cardiac arrhythmias are dangerous. Partial heart block of such a nature that the heart slows with exercise is especially dangerous, because at the end of the operation the heart normally accelerates to adjust to the increased pulmonary blood flow. Slowing of the heart rate at this time is liable to lead to acute cardiac dilatation and standstill.

* These criteria were discussed in detail in the Brown Memorial Lecture given by the author in Atlantic City, June 1947, entitled "An Analysis of Malformations of the Heart Amenable to a Blalock-Taussig Operation," to be published in the *American Heart Journal*.

Rotation of the heart upon its axis calls for careful investigation, as this condition is frequently associated with a complicated malformation.

The over-all mortality rate of the first 350 patients* submitted to a Blalock-Taussig operation is shown in Table I. Although not shown in this

TABLE I
RESULTS OF THE FIRST 350 BLALOCK-TAUSSIG OPERATIONS

	Total No. of Cases	Percentage
Deaths	60	17.0
Exploratory	16	4.5
Unimproved	8	2.5
Slight improvement	18	5.0
Excellent	248	71.0

table, the mortality rate in infants and in adults has been considerably higher than in children. The mortality rate in infants has been approximately 30 per cent and that in children approximately 10 per cent and in adults approximately 18 per cent. The 16 instances in which only exploratory operation was performed include patients in whom no pulmonary artery was found, patients in whom the pulmonary pressure was abnormally high, and patients in whom the heart action was so poor that Dr. Blalock was forced to close the chest even though the condition appeared to be amenable to surgery. The eight cases (or 2.5 per cent) in which there was no improvement indicate how seldom a thrombosis occurred at the site of the anastomosis.

The amount of the benefit derived from operation depends upon the size of vessel used for the anastomosis, the extent of the over-riding of the aorta, and the existence of other malformations in addition to a tetralogy of Fallot. The beneficial effects of operation are measured by the changes in the blood, by the changes in the heart size, and by the increase in the exercise tolerance of the individual. The oxygen saturation of the arterial blood rises to between 75 and 90 per cent and the red blood cell count, the hemoglobin level, and the hematocrit reading return to normal values.

The changes in the oxygen saturation of the arterial blood in four typical cases are shown in Table II. The first column gives the pre-operative values; the second column shows the rise in the oxygen saturation approximately three weeks after operation; the subsequent columns give the values at six months, one year, and two years after opera-

* Since the paper was read, Dr. Blalock and his assistants have operated on more than 250 additional patients with an operative mortality rate of less than 10 per cent.

TABLE II
THE PERCENTAGE OF OXYGEN SATURATION OF THE ARTERIAL BLOOD BEFORE AND AFTER A BLALOCK-TAUSSIG OPERATION

	Name	Before oper.	After operation			
			Immed.	6 mo.	1 yr.	2 yr.
1.	R. S.	78.0	85.0	86.9	88.4	88.4
2.	J. S.	65.8	79.5	84.0	83.0	68.0
3.	H. C.	65.9	81.4	82.0	—	83.0
4.	M. C.	20.6	47.9	58.9	59.0	64.0

TABLE III
RED BLOOD CELL COUNT IN MILLIONS AND HEMATOCRIT READING IN FOUR CASES OF A TETRALOGY OF FALLOT BEFORE AND AFTER A BLALOCK-TAUSSIG OPERATION

	Name	Before oper.	After operation			
			Immed.	6 mo.	1 yr.	2 yr.
<i>Red cell count</i>						
1.	R. S.	6.4	5.5	5.5	5.5	5.5
2.	J. S.	9.0	6.9	5.3	5.0	4.8
3.	H. C.	10.5	6.6	5.5	—	5.5
4.	M. C.	5.7	5.2	6.3	6.3	6.4
<i>Hematocrit</i>						
1.	R. S.	59	48	42	46	47
2.	J. S.	71	56	44	47	41
3.	H. C.	86	57	48	—	47
4.	M. C.	48	43	48	47	53

tion. In Case 2 it will be noted that the oxygen saturation after two years had fallen. Table III gives the red blood cell counts and the hematocrit values on the corresponding patients at the corresponding times. In these tables it is seen that in Case 2 the fall in the oxygen saturation was not accompanied by a rise in the red blood cell count or in the hemoglobin level; therefore the lower oxygen saturation of the arterial blood was probably due to the fact that the child was crying (not in a basal state) when the sample was taken. As shown in these tables, in most instances after operation the oxygen saturation of the arterial blood rises to between 75 and 85 per cent and the red blood cell count, the level of the available hemoglobin and the hematocrit reading return to normal values.

Case 4 is from the record of an infant, and as so frequently happens in spite of extreme anoxæmia, he did not have polycythemia. In this instance the oxygen saturation of the arterial blood has risen steadily but has not yet reached the optimum level,

and the red blood cell count and the hematocrit value have also risen. It is to be hoped that when the oxygen saturation of the arterial blood reaches 75 per cent, the red blood cell count and hematocrit reading will again decline.

In every instance the above changes are accompanied by a dramatic change in the patient's physical condition. The lips are usually of normal colour and although the fingernails may show a tinge of cyanosis, the clubbing slowly recedes; furthermore the patient's exercise tolerance is greatly improved.

The ability of the heart to adjust to the operation is shown by the after course. The degree of cardiac enlargement has varied from patient to patient, and by and large it has varied with the size of the vessel anastomosed to the pulmonary artery. In most instances, however, the heart has been able to adjust to the load and to maintain compensation. Indeed, only 3 of the first 220 patients, operated on for pulmonary stenosis or atresia and discharged improved, have subsequently died of cardiac failure. Two of these three were known not to have a tetralogy of Fallot because they both showed electrocardiographic evidence of a left axis deviation. Two other patients have developed cardiac failure and, in

addition, two children have shown radiological evidence of progressive cardiac enlargement. That progressive cardiac enlargement and cardiac failure are the exception, not the rule, is shown by the fact that approximately 30 per cent of the patients have shown no change in the size of the heart following operation; 30 per cent have shown an increase in size during the first three weeks after operation and thereafter have shown no further increase in heart size; and approximately 30 per cent have shown an increase in heart size between the time of discharge and their return for the six months checkup and thereafter have shown no further cardiac enlargement.

All patients are permitted full activity at the end of the third month after operation. The extent of the improvement in the exercise tolerance has been tremendous. We expect any child with a pulmonary stenosis or atresia to walk more than a mile after a successful operation. Many a child three months after has walked six miles, and more than one twelve-year old boy who estimated his exercise tolerance before operation as three or four city blocks has been on a 10-mile hike within the first six months after operation.

SURGICAL TREATMENT OF PULMONIC STENOSIS

BY ALFRED BLALOCK, Baltimore, Maryland

I wish to thank the members of the Section of Cardiology for the privilege of participating in this symposium.

Six years ago Dr. Edwards A. Park and I carried out experiments in an attempt to devise a treatment for coarctation of the aorta. The thoracic aorta of the experimental animal was cut across, the two ends were closed and the subclavian artery was used to by-pass the point of occlusion. Before this method was used in the treatment of coarctation in man, a better method was developed by Dr. Crafoord of Stockholm: his operation is the preferred procedure but there are some instances in which it is necessary to employ the method which Dr. Park and I described.

In discussing the experimental work on coarctation with Dr. Helen Taussig, she suggested that I try to find a method for increasing the blood flow to the lungs in cyanotic patients with pulmonic stenosis. As you know, the most common abnormality of this type is the tetralogy of Fallot. This problem was investigated in the experimental laboratory and I found that the cyanosis produced by an operative procedure which I shall not go into at this time could be partially alleviated by the creation of an artificial ductus. After the experimental work was

completed, the problem was returned to the clinic and work on patients was begun.

As Dr. Taussig has said, the major difficulty in the tetralogy of Fallot is the pulmonic stenosis. Fortunately the stenosis is usually limited to a single point (pulmonary conus region), and the pulmonary artery distal to this point is usually a fair sized vessel. Since this is true, the pulmonary artery in most cases is suitable for anastomotic purposes.

I would like to review for a moment the anatomy of this region. As you know, the arch of the aorta usually gives rise to the innominate artery, the left carotid artery, and the left subclavian artery. When the aortic arch descends to the left, which is usually the case, the innominate arises on the right side. When the aortic arch descends to the right, which is observed in one-fourth of our cyanotic patients, the innominate arises on the left. I prefer to use the subclavian branch of the innominate for the anastomosis, and hence the position of the aorta should be determined pre-operatively. This can be done with great accuracy by the use of the method of Bedford and Parkinson of London. The incision is usually made on the side opposite that on which the aorta descends. The operative procedure consists of anastomosing a branch of the

aorta or the aorta itself (as recommended recently by Potts) to one of the two pulmonary arteries. As you know, the pressure in the pulmonary artery is low, the pressure in the aorta and its branches is high, and a large quantity of blood will flow from the systemic to the pulmonary circuit after such an anastomosis is made. The incision is made with the patient lying on his back but with the side to be operated upon at a slightly higher level. This leaves the opposite lung in good condition for carrying out respiratory functions during the operative procedure. After the pleural cavity has been entered, the azygos vein is doubly ligated and divided. The pulmonary artery is then exposed and it is dissected free of the surrounding tissues. The innominate with its branches is then exposed and in the usual operation the subclavian branch of the innominate is ligated distally. It is occluded proximally with a bulldog clamp and is cut across proximal to the ligature. The pulmonary artery is then occluded proximally with a special rubber-shod instrument and distally with a bulldog clamp, and a transverse opening is made on its upper surface between the two points of occlusion. Using fine non-absorbable suture material an anastomosis is then made between the end of the subclavian and the side of the pulmonary artery. The subclavian branch of the innominate makes a good angle with its parent vessel and stenosis at the point of origin is not nearly so apt to occur as when the subclavian artery that arises directly from the aorta is used.

As stated previously, the end-to-side type of anastomosis is preferred. In some instances, however, it is necessary to do an end-to-end anastomosis after having ligated and divided one of the pulmonary arteries. One of the advantages of the end-to-side anastomosis is that it allows the shunted blood to go to both lungs.

The motion picture, a portion of which I will show now, illustrates the operation on a five-year-old boy who had a moderate degree of cyanosis. His disability was fairly marked. The anæsthetic agent in this case was cyclopropane with a high concentration of oxygen. The exposure of the right pulmonary artery is shown. The pressure in the artery was measured by the use of a needle and a manometer. In this patient the pressure was about 200 mm. of water (15 mm. Hg.) which is about the average pressure that we have obtained in our cases. If the pressure is higher than 375 mm. of water, we hesitate to carry out the procedure.

You will note in the picture that there is no difficulty in this case in approximating the subclavian artery to the pulmonary artery. Difficulty is encountered in some of the older, larger patients in approximating these vessels. In some of these

the incision is made on the opposite side and the subclavian branch of the aorta is used. You will note that the transverse opening in the upper surface of the pulmonary artery is slightly larger than the end of the subclavian artery. You will note also that the suture everts the intima of the vessels.

I now show you a picture of this boy 18 days after the operation. You will observe that his cyanosis is much lessened. It takes a longer period of time for the characteristic clubbing of the fingers to disappear. More important, however, is the lessening or the disappearance of the disability. Thus far some of the patients seem to be able to lead normal lives.

A few comments might be of interest regarding the various vascular anomalies we have found. In the first 475 patients there were 121 in whom the aortic arch was on the right and in the majority of these there was an obliterated ductus which joined the subclavian branch of the innominate. In at least 14 of these patients there was an anomaly in the position of the superior or inferior vena cava or both; the number is probably considerably in excess of this. In at least 5 the superior pulmonary vein entered the superior vena cava. In 15 patients or more there was rotation of the heart to the right. Various other arterial and venous anomalies were encountered.

The mortality rate has been high but not discouragingly so. When the last analysis was made, it was found that 474 patients who were thought pre-operatively to have the tetralogy of Fallot or one of its variations had been operated upon. An end-to-side anastomosis between the subclavian artery and the pulmonary artery was performed in 331 of these and among these there were 37 deaths, or 11 per cent. Among the patients who succumbed in this group there were three with transposition of the aorta and the pulmonary artery and one with truncus arteriosus. Several of the deaths occurred a number of months following the operation.

An end-to-end anastomosis between a branch of the aorta and the divided distal end of one of the pulmonary arteries was performed in 23 cases with 4 deaths. An end-to-side anastomosis between one of the carotid arteries and a pulmonary artery was performed in 30 cases with 9 deaths. An end-to-side anastomosis between the innominate artery and a pulmonary artery was carried out in 47 cases with 13 deaths. A side-to-side anastomosis between the aorta and one of the pulmonary arteries was performed in 2 cases with 1 death. In 37 of the total number of cases only an exploratory thoracotomy was performed; in most of these a pulmonary artery was not present or at least could not be found. Among these patients who

had an exploratory thoracotomy without an anastomosis there were 18 deaths. In the entire total of 474 cases there were 86 deaths, a mortality of 18 per cent. This includes all patients who have died, whether in hospital or after leaving hospital, and whether or not an anastomosis was performed.

There has been no serious disturbance of the circulation of the arm, the subclavian of which was ligated, in any of the patients and the function of the arm is excellent. There have been approximately 400 such patients in whom the subclavian artery was ligated and divided.

The age of patients operated upon has varied

from two months to 44 years. We consider the ideal age for operation to be from three to nine years. All of the patients received penicillin. There have been no instances of empyema or pericarditis. Heparin and dicumarol have been used very rarely.

In conclusion I would like to say that it is too early to evaluate the ultimate effects of this operative procedure since it has been less than three years since the first such operation was performed. We can state that the results thus far are quite encouraging.

By J. W. BROWN, Grimsby

Ample justification for operation is found in the relatively bad prognosis of cyanotic congenital heart disease. Observation of over 100 cases has shown that many die before the age of three, and about a quarter of the cases survive the school age. Most of the children can be educated if given the opportunity and they should always be taught to read and write. Between 60 and 70 per cent of the cases fall into the group of the tetralogy of Fallot where pulmonary stenosis is combined with a large interventricular septal defect, an over-riding dextro-positioned aorta, and right ventricular hypertrophy.

Cyanosis is present and depends upon the oxygen unsaturation of the blood and the polycythæmia, which in their turn are dependent upon the degree of pulmonary stenosis and upon the amount of the pulmonary blood supply. Cyanosis may vary from day to day and definitely with the season. The factor of the over-riding aorta cannot be of enormous importance when the figures of oxygen saturation after operation are considered, but it is of importance in relation to paradoxical embolism and cerebral abscess. In this series thrombotic incidents due to polycythæmia have not been frequent. Commonly there is a basal systolic murmur and a clear second sound. Absence of a murmur does not invalidate diagnosis of the tetralogy.

Diagnosis is essentially radiological, and the picture of a small pulmonary artery with a concave pulmonary arc and absence of pulmonary congestion and of hilar pulsation are convincing. A right aortic arch is frequently present, and a cœur-en-sabot may be seen in the older children. The electrocardiogram shows a right axis, but abnormalities of the ventricular complexes and conduction defects do not appear to be so common as in auricular septal defect.

There are other conditions that may be confused with the tetralogy. Tricuspid atresia is distinguished by the presence of a left axis in the electro-

cardiogram, a pathognomonic finding in a cyanotic case. Transposition of the great vessels usually causes gross cardiac enlargement over a short period of time, and survival is generally short. There is a narrow vascular pedicle which broadens in the oblique view, for then the aorta comes directly in front of the pulmonary artery.

The evil prognostic signs are a rising red cell count, a high hematocrit reading, syncopal attacks, and thrombosis. Many of the cases exist rather than live in the fullest sense.

The point before us is that operation is not a cure, but a physiological adjustment that allows a greater circulation to the lungs through an artificially created ductus arteriosus. The subject still remains liable to the inherent risks of the patent ductus, and the mixed blood in his aorta is little different from venous blood. The immediate results of operation are so beneficial to the patient that we must critically study every case of cyanotic heart disease so that those types of abnormality likely to be helped by operation may be recognized. Where clinical and ordinary laboratory investigation fails to make a satisfactory diagnosis, some help may be had by intracardiac catheterization and by angiocardiography.

Of the operation, one is entitled to ask at this stage whether the calibre of the anastomosis created in infancy will increase *pari passu* with the growth of the child. There might well be the risk of contraction and ultimate obliteration at the suture line in the same way as the small ventricular septal defect of the *maladie de Roger* may spontaneously close by contraction of its fibrous margins. We know too that in the cyanotic case a patent ductus fulfilling an obviously physiological purpose in shunting blood between two pressure systems may quite suddenly close and bring about the death of the patient, showing that the mere fact of a shunt does not necessarily keep open an abnormal com-

munication. The Potts operation of direct anastomosis between aorta and pulmonary artery may have something to offer in allowing the surgeon to determine the size of orifice that he may make.

If prognosis is to be the basis on which surgical treatment of congenital heart disease is to be founded, the time has come when this problem should be seriously and collectively examined. Our present ideas are based upon statistics that contain many neonatal cases. Statistics depend upon the interest shown by clinicians and pathologists, and naturally the highest incidence of congenital heart disease is in children's hospitals, rather than the general

hospital. Only the interesting case is reported, and no account is taken of the living because we tend to publish only the case with post-mortem control. As an example where is the adult with patent ductus arteriosus? We certainly do not see him in our clinics. He has not been cured in childhood by the surgeon as yet. Do we fail to recognize him when he is in failure, or when he has infective endocarditis? He is not seen by the pathologist who perhaps practises section of the great vessels within the pericardium. Does the ductus after all close spontaneously in adolescence or early adult life?

COARCTATION OF THE AORTA

By C. CRAFOORD, Stockholm

First of all I want to say that I feel it as a great honour that I have been invited to read this paper. We have heard an excellent review by Dr. Taussig and Dr. Blalock of the work they have done on the treatment of the blue babies. There are, however, two more congenital heart malformations that have been more closely studied than ever before because experience has shown that by surgery these two can be completely cured—patent ductus and coarctation of the aorta. I am going to deal with coarctation, which has been thoroughly studied in my hospital where the first operations for this disease were performed, and to give you only a résumé of my results in patent ductus as this is going to be covered by the following speakers.

Coarctation of the aorta was first described by Morgagni in 1760. Wadstein in 1897 collected 103 reported cases. Maud Abbott (1931) described among 1000 cases with congenital cardiovascular defects 142 cases of coarctation, which indicates that about 15 per cent of all congenital lesions in this group are coarctation.

The incidence of the adult form of this malformation is about 1 per 1500–2000 when estimated from large post-mortem series. The juvenile form has no surgical interest as these cases die very early. The adult form is usually confined to the area of the ductus arteriosus. From the United States Pearlman, when examining men between 18 to 35 years of age for army service found only 1 in about 10,000. This discrepancy depends on the fact that by rapid clinical examination quite a number of coarctation cases are never detected. Both clinical and post-mortem findings show that the incidence of this congenital lesion is much more common than was previously believed and more common among men than among women. Maud Abbott gives this proportion as 3 to 1. Among my 22 resected cases 7 were females.

Other developmental disturbances may be combined with this form of coarctation: the bifid aortic cusp, estimated by Abbott to occur in 25 per cent of the cases; secondly hypoplasia of the aortic wall, according to Abbott in 10 per cent of cases, this is naturally of surgical importance and must always be borne in mind as a pronounced hypoplasia makes a resection and suture impossible to perform; thirdly patency of the ductus arteriosus; and finally there is a small group of cases in which the points of origin of the great vessels leaving the aorta are abnormally situated, the coarctation itself may be of considerable length, and defects of the ventricular or atrial septum may be present. Some of these are of importance in diagnosis because their existence may influence the decision to operate or not.

Early atherosclerosis in arteries proximal to the narrowed segment seems to be very common. The oldest patient I have resected was 27 years of age and some have been around 20: in these cases we could find very slight or no atherosclerotic changes.

The prognosis regarding length of life is of special interest. Ask Upmark (1942) found that about 25 per cent of all cases died before they reached 20, 50 per cent before 40, and 90 per cent before 50 years of age. The commonest causes of death according to Abbott are in order of frequency: congestive heart failure, rupture of the aorta or the heart, cerebral hæmorrhage, and bacterial endocarditis.

All my coarctation cases have been studied in the heart clinic of Prof. Nylin before operation, a collaboration that I consider to be of greatest importance. Coarctation seems always to be accompanied by increased blood pressure above the level of the coarctation, and decreased pressure below. It is not known how early in infancy these changes in the blood pressure are present. In

a 3-year-old child reported by Bodlander (1946) the blood pressure was 130/60 which is to be considered as hypertension at this age; erosion of the ribs was seen in the radiographs.

We are of the opinion that a higher blood pressure in the legs than in the arms practically excludes coarctation. In our material the blood pressure has been high in the arms and low in the legs in all patients. In the two youngest, both children of 11 years of age, we found systolic blood pressures ranging in the arms in one between 150 and 190, and in the other between 140 and 150 mm.

If hypertension is present, the possibility of coarctation should always be borne in mind. The subjective symptoms resulting from the hypertension in the upper part of the body vary and may include general weakness, palpitation, vertigo, throbbing headache, and a feeling of heaviness in the head, and visual troubles.

The low blood pressure in the lower half of the body, which never occurs in hypertension from other causes, not infrequently produces vague or even quite pronounced symptoms suggestive of intermittent claudication, but may cause no symptoms at all.

The majority of patients seek medical advice because of one or more of the above symptoms. The clinical findings on examination are mainly as follows:

1. By inspection and palpation abnormal pulsation is found, both in collaterals (most often in the axillæ, the supraspinous fossæ, and close to the lower borders of one or more ribs), and also in dilated arteries in the neck and head, central to the stenosis, and finally absence of pulsations in the arteries in the legs.

2. By percussion and auscultation one finds hypertrophy of the heart mainly on the left and a harsh systolic murmur over the base of the heart also at the back especially in the interscapular area to the left. Probably the murmur originates from collaterals and not from the stenosed part of the aorta. In one case of mine, in which the stenosis was complete, this systolic murmur was present before operation and vanished afterwards, which shows that the murmur could not have been due to the stenosis itself. A systolic murmur over large collaterals can also be heard and recorded phonocardiographically.

3. *Oscillographic findings.* This examination has been used with special interest in our hospital and has been carried out with a new apparatus designed by Dr. Ejrup in the heart clinic of Prof. Nylin. The recordings can be calibrated and are thus directly comparable at different examinations. The auscultatory method is adequate in studying blood

pressure changes in the arms, but in the legs it is often difficult to hear the sounds when auscultating in the popliteal fossæ. The oscillogram gives a clear and visible record of the blood pressure and pulsations in the legs even in coarctation cases, and is of great interest because, as shown by Ejrup in 1945 and 1946, there is a special reaction to exercise. If in a normal case an oscillogram is taken on the upper or the lower limbs after exercise, the blood pressure is higher and the pulsations are greater than in the oscillograms taken during the rest.

In organic obstruction of the arteries and in coarctation an "inverse reaction" with a diminished blood pressure and a decrease of pulsations appears after exercise. This reaction has been found in patients with thrombo-angiitis obliterans, severe arteriosclerosis, and arterial embolism. It is not influenced by the presence or absence of intermittent claudication. In obstruction of the arteries of the lower extremities intermittent claudication will sooner or later develop if the exercise is great enough, but the abnormality is shown by oscillography long before clinical symptoms appear.

In coarctation cases at rest the oscillographic tracings show big pulsations and an elevated blood pressure in the arms and small pulsations and low blood pressure in the legs. After work test the tracings show further increase of the pulsations and the blood pressure in the arms but in the legs a paradoxical or pathological reaction is demonstrated by either a decrease of the blood pressure and the pulsations or absence of increase in blood pressure compared with the tracings at rest.

4. *X-ray examination.* We have found (A) and (B) constant findings; (C), (D), and (E) inconstant findings.

- (A) Widening of the left subclavian artery which shows up as an S-curved outline to the left of the superior mediastinum, an impression on the œsophagus, and an indentation to the left in the posterior mediastinum.

- (B) Dislocation and shortness of the aortic arch with absence of the aortic knuckle.

These constant findings are easy to understand. A frontal section from a frozen body in the post-mortem room makes it easy to understand that a dilatation of this left subclavian artery will produce a convex shadow against air-containing lung and an impression on the œsophagus.

The indentation we believe is most often formed between the entrance of the subclavian artery in the aorta and the aortic wall above the coarctation, and not due to the constricted area itself. The dense fibrous tissue which can be dissected when the aorta is freed at operation often gives by itself such a shadow that the actual coarctation is difficult to

see as an indentation in the X-ray film. I have, however, a picture that shows two different indentations: one cranial formed by the subclavian artery and the aorta, and one caudal due to the coarctation itself.

(C) Erosions of the ribs.

(D) Enlargement of the left ventricle and increased curve to the right of the ascending aorta.

(E) Enlargement of the left auricle.

In experimental studies on dogs in 1935-6, I demonstrated that the flow of blood to all the organs could remain suspended for as long as 25 minutes without there being any subsequent signs of organic damage, provided an adequate flow to the brain was secured. This circulation to the brain was maintained by creating anastomosis between the carotid and jugular vessels in a dog of the same size lying beside it. On the strength of this observation I took, in certain patients with a patent ductus, the risk of placing clamp forceps on the aorta above and below the point of entry of the duct, leaving them in place during the time necessary to divide the duct and suture the aorta. In one of the patients this part of the operation took no less than 27 minutes: I was forced to it because an attempted ligature cut the ductus completely with bleeding somewhat difficult to control. In spite of this no noticeable disturbances were observed.

Because of repeated experiences of clamping of the aorta in cases suitable for division of the ductus I began in 1943 to discuss whether it might not be possible to treat congenital coarctation of the aortic isthmus by resection.

In the light of the results obtained in the experiments and of the clinical experiences it seemed that in patients with congenital isthmus stenosis, who already possessed a well-developed collateral system between the large arteries arising central to the coarctation and the arteries in the lower half of the body, the aorta could be kept closed for considerably longer than 27 minutes without danger to the patient.

As far as I had been able to ascertain at that time the only other investigator who had considered the possibility of relieving coarctation surgically was Blalock, who, however, attacked the problem from a different standpoint, having considered some form of anastomosing operation or plastic reconstruction as the only way of improving the circulation peripheral to the stricture.

I, therefore, decided that the bad prognosis of aortic coarctation was a sufficient justification for attempting surgical treatment which was tried for the first time on two cases in October, 1944.

Resection of the constricted part of the aorta was

carried out and the aorta was sutured end to end with oiled silk with a technique similar to the one described by Carrell. Both cases healed and normal blood pressure conditions were re-established.

Until the end of July, 1947, 22 cases have been resected with 2 deaths, one due to hæmorrhage from a divided intercostal artery from cutting through of the central ligature, and one (about a month after the operation) due to formation of a false dissecting aneurysm from insufficiency of the aortic suture. Three more cases have been explored without any attempt at resection. In one the constriction of the aorta extended from the origin of the innominate artery to well below the left subclavian artery. In the second the aortic wall was extremely thin and hypoplastic. In the third, the oldest in my series, aged 35 years, there was extensive atherosclerosis. The two first mentioned of these cases died of post-operative complications. The first had bilateral pneumonia, insensitive to penicillin and sulphonamides; he had been treated for months before operation with a combination of penicillin up to a million unites a day and sulpham compounds because of bacterial endocarditis and with healing of this complication. The second died from extensive uncontrollable oozing from the wound, and into the pleural cavity; he had been operated on many years before for appendicitis, that time also with severe after-bleeding from the wound. The third of these patients healed without complications.

Despite this experience I would like to stress that an ordinary exploratory thoracotomy cannot be considered more dangerous than an exploratory laparotomy and is certainly justified in a great variety of cases and also in order to determine whether or not resection can be performed in those cases of coarctation that are deemed suitable for operation.

Methods of investigations taken up since a year ago, which we hope will diminish the number of explorations in coarctation cases, are cardio-angiography and heart catheterization.

By the latter method all sorts of shunts such as ventricular or atrial defects or patent ductus can be diagnosed by taking blood samples simultaneously from different parts of the heart and a peripheral artery. Cardio-angiography I believe will be of utmost importance. By this method the site and the extent of the constriction can be determined and unnecessary explorations can be avoided; it also makes it possible to study the anatomical result of the operation.

We intend now if possible to take cardio-angiograms as a routine after our operations. In 5 of 6 cases we have found normal conditions. In the

sixth there was a small vague rounded shadow close to the aorta at the calculated site of the anastomoses indicating the possibility of formation of a small aneurysm at this point. This case will be subjected to further cardio-angiograms in order to see what happens. Clinically he is in very good condition.

As mentioned we have now resected the aorta in 22 patients. Two of these died. The 20 surviving have healed without or with minor post-operative complications of no importance to the subsequent course. They have all been followed up: in all the subjective symptoms that the patients had before the operation have vanished.

In most cases the blood pressure in the arms has returned to low normal values. In a few it is still at or slightly above the normal upper limits. In all cases the blood pressure in the legs has been elevated—in some up to 10–20 mm. higher than in the arms which is to be considered as normal: in the others up to the same or slightly below the blood pressure in the arms. All the blood pressure readings I am now discussing are at rest.

In all cases the oscillograms from the lower limbs after work have gone back to normal showing much increased oscillations and an increasing blood pressure instead of the pathological or paradoxical reaction before operation.

Cardiac tolerance tests as used by Nylin have been made in all cases. In some before operation this test has indicated a certain amount of cardiac insufficiency after work. In all when these tests were repeated after operation a normal function was found. This correlates with our finding that the cardiac volume has returned to normal in all cases with pathological findings in that respect before operation. In some cases in which the heart volume has been within calculated normal values we have, however, also found a diminution after operation.

Patent Ductus Arteriosus

I want to give a brief résumé of my results in 101 operations for patent ductus. I have done double ligature with injection of 50 per cent glucose solution as a sclerosing agent between the ligature in 69 of the cases, and in the remaining 32 I have divided the ductus. In most of these I have done a clean arterial suture of the wound both in the aorta and the pulmonary artery.

All the cases have been correctly diagnosed and there was no discussion of the certainty of the diagnosis. All had the typical continuous murmur with typical localization which could be demonstrated by auscultation and verified by phono-cardiographic registration.

In all surviving cases this murmur disappeared and has not reappeared in any case.

I have divided the 101 cases into 95 non-infected cases and 6 cases with infection of the ductus and septicæmia.

In the first group 2 cases died. The first could have been avoided. Everything was quite normal until the catheter was passed through the intratracheal tube for post-operative aspiration. It was not observed that the suction catheter practically filled the whole lumen of the small intratracheal tube, and in consequence the endobronchial pressure was rendered highly negative. The same effect was produced as was described in this country from the effect of the negative pressure wave in blast injuries. There was a sudden heart stop. The wound was immediately reopened and the heart action revived by direct heart massage. The time of complete heart arrest was, however, too long, about 5 minutes, and the patient died about 48 hours later from the result of severe cerebral anoxæmia.

In the second case there was a very marked hypoplasia of the aortic wall around the ductus. This was not observed before the ductus was divided after clamping of the aorta above and below. Because of the hypoplasia of the aortic wall it was extremely difficult to obtain a sutureline without leak and the aorta had to be clamped for a little more than 45 minutes. In this case post-operative anoxæmic damage of the spinal cord developed. Because of this and other complications the patient died three weeks after operation.

This gives a mortality of just over 2 per cent in this group, with complete healing without recurrences in the surviving cases.

In the second group of 6 cases 5 healed with no complications and 1 died. This was the first in the group before we had penicillin available in Sweden. With only sulpha-drugs it was impossible to get negative blood cultures, which we have been able to obtain in the rest of the cases before operation. This fatal case seemed to heal quite all right in the beginning. She left hospital about 6 weeks after operation with negative blood cultures, normal temperature and only slightly elevated sedimentation rate. However, she came back to hospital a couple of months afterwards with all clinical signs of a recurrence of her septicæmia and also a recurrence of her ductus arteriosus. It was difficult to declare how this recurrence might have occurred as this patient was treated with complete division of the ductus and suturing of both the aorta and the pulmonary artery. I believe that an infected hæmatoma must have formed between the pulmonary artery and the aorta and that this hæmatoma secondarily must have broken into both the pulmonary artery and the aorta. The patient was submitted to a re-operation at which was found a

big false aneurysm between the aorta and the pulmonary artery with communication between the aneurysm and both the big vessels. An attempt was made to resect the aneurysm and to close the aorta and the pulmonary artery. This was, how-

ever, technically impossible to perform. After a partial resection of the aneurysm this was sutured and the chest wound closed. The patient died shortly after the operation was finished.

PATENT DUCTUS ARTERIOSUS

BY RAE GILCHRIST, Edinburgh

Notable contributions have been made to this subject in recent years. I shall confine my remarks to the diagnosis of the *patent ductus* and the results of surgical occlusion. Nearly 70 cases have been under observation and I am greatly indebted to my surgical colleagues, Sir John Fraser and Mr. Walter Mercer, who have kindly placed their operative notes at my disposal.

In diagnosis the hallmark is the continuous murmur described by Gibson, heard best below the inner end of the left clavicle and almost invariably accompanied by an accentuated second pulmonary sound. Difficulties arise in infancy before the murmur has developed its full continuous quality, and also in later life, when, in the presence of congestive heart failure, it may at times be inaudible so that the lesion escapes clinical recognition. By the time school age is reached the murmur has usually developed its distinctive continuous quality. When the channel is large, the ductus of wide bore, 1 cm. or more in diameter, the leak from the aorta to the pulmonary artery is considerable. The physical signs are then well marked, the murmur loud and roaring, with a thrill coarse and widespread. The heart is larger than when the murmur is less intense and an isolated finding. On cardioscopy a fast, vigorous over-active heart with an increased and flicking type of pulsation in the aorta and pulmonary artery is very characteristic. In my experience the dilated left auricle and a hilar-dance in the smaller vessels of the lung roots are exceptional. They do not occur in the average case, but only when the ductus is of wide bore. On the other hand, in the pulmonary artery itself the vigorous, forceful pulsation—what we call in Edinburgh a Highland fling—is very characteristic.

Bohn's exercise test in our hands has not come up to expectations. When the physical signs are doubtful in a child, that is before the Gibson murmur has developed, the response of the blood pressure to exercise has seldom been of help in reaching a diagnosis. In the majority of children the electrocardiogram is of normal form. After the age of 10 years the proportion of patients who show a minor grade of left axis deviation tends steadily to increase.

In diagnosis it is important to correlate any general septicæmic signs with the local evidence of a patent ductus, as this at once gives the clue to a bacterial infection restricted at its onset to the pulmonary artery in the immediate neighbourhood of the mouth of the ductus. In the diagnosis of the infected case there is a triad of signs that make a characteristic clinical picture: (1) local evidence of a patent ductus, often overwhelmed by the more dramatic and florid features of (2) a generalized septicæmia and later as the local vegetative process advances in the pulmonary artery, and (3) multiple lung infarcts. It is, of course, desirable to make the diagnosis of endarteritis of the pulmonary artery before infarcts, systemic or pulmonary, occur. Early ligation should be urged.

With the increasing experience of the past seven years I have little hesitation in expressing the view that on the recognition of a patent ductus in childhood, surgical closure should be recommended with few exceptions. The results obtained by closure of the channel are very good. As the ductus may on occasions close spontaneously surgery can be withheld, if the physical signs are not advanced and the heart unembarrassed, perhaps until the age of 7 or 8. After this age if the ductus remains patent the child should be sent to the thoracic surgeon, preferably before the age of 10 is reached. If the signs are gross, nutrition impaired, and the child obviously handicapped, closure of the ductus should be undertaken earlier, even by the age of 3 or 4 years. After the age of 10 the surgical technique is a trifle more hazardous and the structure less resilient and the late Mr. Tudor Edwards showed that the mortality rate for intra-thoracic interference increased with each decade. In my experience the age period 7 to 10 years is for choice the most suitable time to recommend ligation. When recognized first about the age of 20 to 30 there is less justification for recommending surgery. If the general state of health is satisfactory, the response to effort good, and the heart not enlarged, then the ductus must impose no more than a trifling burden on the circulation. In these circumstances I doubt the wisdom of recommending a major

surgical procedure. The development of a fever, or any septicæmic signs demands prompt action and every care should be taken to reduce the risk of a *Streptococcus viridans* infection so commonly derived from oral or dental infection. It is important, for instance, to recommend the administration of sulphonamides over the period of dental extractions if the risks of septicæmia are to be avoided in those known to harbour this and other congenital cardiac defects.

In the immediate post-operative care of these patients measure to reduce the tendency to massive pulmonary collapse are important. A pleural effusion should be tapped even within 24 or 48 hours of operation. Respiratory exercises should be commenced at the first opportunity. In fact it is useful to have the child taught the appropriate exercises in the days before surgery is undertaken.

The results we have obtained in Edinburgh have been very satisfactory. It is seven years since the first ligation was undertaken. Deaths have occurred from massive pulmonary collapse in two infected cases and also from mediastinal infection (before penicillin was available) in one case and from hæmorrhage in another. With the exception of three patients in whom recanalization occurred the remainder have all obtained striking benefit, more

than sufficient to compensate for the bitter disappointments of our earlier experience.

Perhaps one or two case records are more instructive than a statistical analysis. All the patients improved in physical fitness and general nutrition. A decrease in the size of the heart and an improved psychological outlook are the rule. A boy, attending a special school for crippled cardiacs had his ductus ligated 3 years ago; he now plays Rugby football. Only eight months ago a boy of 13 had his ductus ligated: last week his mother sent me his headmaster's report for the term: "He is an entirely different boy since his operation. His thrust and responsiveness are much more positive and sustained. We have seen this at his cricket and at his work. He has lost his diffidence." One other exceptional patient is worthy of mention, as five years have elapsed since this boy's ductus was ligated at the age of 13: within two years he gained 43 lb. in weight and grew 8 inches in stature. Since then he has been accepted as A1 for the Army, and has won a regimental boxing competition, does a 15-mile cross-country run twice a week, and claims to have done 100 yards in 10 seconds. Surgery can save lives from invalidism and prevent premature death from infection. Closure of the ductus is a most notable advance in cardiac therapeutics.

By HOLMES SELLORS, London

I am confining my remarks to *patent ductus arteriosus*, the only form of congenital heart disease with which I have had much experience. Previous speakers have stressed most of the clinical and therapeutic aspects of the condition, and I want to speak of some of the most unusual features. At the outset I would like to stress the view that it should be regarded as a condition requiring surgery unless there are obvious contraindications. Even though it is less than ten years since the first ductus was tied, the minimal risk of operation in practised hands far outweighs the risks that the patient undergoes from being left alone. I have been most impressed by the results, even in patients who had practically no symptoms. Children develop quickly physically and mentally, and young adults are most emphatic in stating how well they feel after the arterio-venous shunt has been relieved.

In all I have now operated on 46 patients with this lesion, with one death. Ten of these patients had infective endocarditis, and 5 were ligated in the active phase of the disease. Since the use of penicillin, we have tended to wait until the infection has been well controlled, and to tie the ductus some weeks later.

Another 10 patients were showing signs of

embarrassment with dyspnoea, considerable enlargement of the heart, and subjective symptoms.

Of the remaining 24 about half complained of some disability or limitation of activities that was cured by ligation of the ductus. The others, mainly young children, were virtually symptomless.

The diagnosis depends on a combination of the classical murmur, the low diastolic pressure, and radiography; and it is fair to say that the murmur occupies most attention. In our 46 cases the murmur and thrill could be regarded as typical in 35. But there were two patients in whom no murmur was heard until infective endocarditis developed—one of these had been fully investigated some months previously, and the evidence for previous absence in the other case was quite strong. In other words, a thrill or murmur can be absent with a patent ductus.

Next there was a group of 9 cases in which the murmur was atypical. In 5 instances a harsh systolic sound only could be detected, and in the remaining 4 the lesion was probably complicated. The diagnosis had to be based on other factors, but in each patient the patent ductus was recognized and occluded. This brings me to an important point in the observation and behaviour of the

murmur at operation. In all cases we have used a stethoscope directly on the heart to establish the point of maximum intensity which ordinarily lies 3.5 cm. below the actual ductus on the conus. Normally when the ductus is compressed the murmur and palpable thrill disappear, but there have been a number of cases in which sounds still persisted. A coarse systolic bruit that disappears in several weeks is fairly common, but there were 4 cases in which the thrill and murmur altered in character and position but still persisted. If we had only recognized this after operation, the possibility of recanalization or failure to close the ductus would have been considered. In these unusual cases complete closure of the ductus was confirmed and additional dissection to exclude other channels was carried out. One of these patients constituted our only fatality: the diagnosis before operation was recorded as a persistent ductus plus a patent auricular septum. The residual murmur, which was that of the patent septum, was heard, and the septum was demonstrated at autopsy. The cause of death was an unrecognized low degree of hæmophilia which led to continued pleural hæmorrhage in spite of the use of anticoagulants and massive blood transfusions. I have emphasized this, because I feel that additional lesions can easily be overlooked, and that our knowledge of the causation of both thrill and murmur is incomplete. On occasions an accidental pressure or kinking of the pulmonary artery has produced unusual sounds, and even mediastinal displacement can add to the confusion. There is also the effects of air or traces of blood in the pericardium to be considered if this sac is nicked during operation.

One cannot exclude the possibility of recanalization if simple ligature is used. I have employed no other method in the series, and am left with two cases which would in many ways serve as examples; both were originally infective cases in which the murmur persisted in an altered character; both are

clinically in excellent condition, and in one the diastolic pressure is at normal level; the other maintained a low diastolic all through, even directly after the ductus was tied.

The question of blood pressure has been fully studied, and in children under 14 years the average figure was 106/44 mm. before operation and 104/71 some time after. In adults an average reading of 123/56 went to 128/81, giving a rise in the diastolic level of 25 mm. At the moment of ligature the pressures sometimes rise appreciably and a typical reading was 100/40 before operation, 130/110 immediately after ligature, persisting for 10 to 14 days to stabilize at 96/70. This rise in blood pressure occasionally coincides with a very rapid post-operative pulse rate lasting for a week to a fortnight and not due to any difficulty in lung expansion.

The final point I would make is concerned with the radiographic appearances. On screening the dilated conus and the free active pulsation in the lung root may be recognized, and we have found considerable value in kymography, where we have noted an erratic notching over the conus in a proportion of cases. I am not certain, but feel that this is almost diagnostic, and it has been of help in doubtful cases.

Of other abnormalities that have been encountered at operation, there has been a stricture of the left pulmonary artery, and a large vein resembling a left superior vena cava in size and position which in part entered lung substance.

These points I have brought forward to suggest that diagnosis is not always simple, and that there are an appreciable number of additional complications that may be encountered. Surgery, apart from its therapeutic value, has afforded some light on the pathology of the living, and has certainly led to some doubt as to the reliability of interpretation of classical physical signs.

BY O. S. TUBBS, London

I wish to congratulate Dr. Taussig, Dr. Blalock, and Dr. Crafoord on their remarkable work. My experience is limited to 33 patients operated upon with the diagnosis of patent ductus, and my comments are based on this series.

In 32 of these 33 cases, the diagnosis of patency of the ductus proved correct at operation. An additional lesion was almost certainly present in two; in the rest, the ductus appeared to be the only abnormality. A Gibson murmur was present in all the cases except in one child of 8 in whom the

murmur was confined to systole; the lumen of the ductus in this latter case proved extremely small.

With regard to the X-ray findings, prominence of the pulmonary artery below the aortic knuckle should not be expected in every case, as it was sometimes completely absent. The sex incidence in the series showed the expected preponderance of females (22 females and 10 males).

Indications and optimum time for operation. Starting in 1939 with an infected case, my experience was for a long time confined to those with infection,

but it is now thought that *all* cases in which a patent ductus is diagnosed as the sole lesion, should be treated surgically at about the age of 7, unless there are special indications to operate earlier. Subsequent to the age of 7, secondary changes and complications, including infection are liable to occur in the untreated case, so that operation should not be delayed much beyond this age.

With regard to an upper age limit, the operation does not become more hazardous after the age of 20. There had been 12 patients over the age of 20 with 2 deaths, and 20 patients under 20 with 3 deaths, *all* the deaths being in infected cases.

The higher mortality in the presence of infection, i.e. 5 deaths in 12 cases (40 per cent) had been the general experience before penicillin became available. This raises the question as to the place of operation in the infected case at the present time. The dramatic reduction in fever and rapid removal of bacteria from the blood stream in such patients is well recognized, but although many patients progress to complete recovery, the mortality is considerable. On these grounds a full course of penicillin should be given, and this should be followed by operation to prevent recurrence. In the penicillin-resistant case the ductus should be closed as soon as the diagnosis had been made. Evidence of systemic embolism in addition to pulmonary infarction is not a contraindication to operation as, although the chance of cure is much less when systemic embolism has become apparent, recovery does sometimes follow.

Cyclopropane had been used as the anæsthetic agent in earlier cases, but ether was now preferred,

with curare to diminish the respiratory excursion, as this was less likely to produce cardiac irregularities. With regard to the operation, the posterior approach through the fourth intercostal space was much preferred to the anterior incision, as the cosmetic result was better, and, far more important, it gave a wider exposure so that it would be possible to deal with any accident that might occur.

The best method of closing the ductus remained a subject for discussion as the incidence of recanalization following the various techniques was still argued. The criterion on which recanalization could be diagnosed was also not fully agreed although most workers considered a diastolic murmur as indicative of a leak. The short ductus of large calibre was the type most likely to recanalize following simple ligature, and on these grounds it was probably preferable to divide such cases and suture the two ends.

The changes consequent to closure of the ductus were mentioned, including the immediate rise of diastolic blood pressure, the abolition of the thrill in the pulmonary artery and of the diastolic murmur, and the rapid reduction in the size of the heart, and in the prominence of the pulmonary artery and its branches in the lung field.

Complications were rare. Tachycardia was commonly seen in the first week or ten days after operation, but proved of little consequence. Massive collapse of the left lung or left lower lobe had been seen, but this had not caused serious disturbance. Fluid collection in the pleural sac was occasionally sufficient to require aspiration.

COARCTATION OF THE AORTA

BY CRIGHTON BRAMWELL, Manchester

Sufficient time has not yet elapsed since the introduction of this operation to enable one to tell how much it would add to the span of life, but there is good reason to be optimistic.

In his own series of 36 cases of coarctation, 13 were over 30 years of age at the time they first came under observation. His 3 oldest patients were dead, but all lived to over fifty, and 2 died of conditions unassociated with their coarctation, one of carcinoma of the stomach and the other of lobar pneumonia. Of the other 10 patients over thirty, 9 were still living and many of these were capable of undertaking strenuous physical exertion without any apparent disability. The third decade seemed to be the most dangerous, for many patients were free from symptoms and, being men of good muscular development, were liable to be subjected

to severe physical strain; while in the case of women, their first confinement might prove fatal. There seemed to be a fair chance that those who survived the hazards of the third decade might live another twenty years. Should these older patients be operated on, or should we consider that having come safely through the dangerous period from twenty to thirty implied that their coarctation was sufficiently well compensated to make their further outlook more favourable? Perhaps my patients may have been particularly fortunate, since in Maud Abbott's series there were 23 per cent of deaths in the fourth decade.

The sex incidence is difficult to explain. Women with coarctation are less likely to break down because apart from confinement, they are not subject to the same physical strain as men, and

hence they may never be medically examined. This, however, cannot be the whole explanation for even in Maud Abbott's series, which was based on post-mortem statistics, the number of males was twice as great as the number of females. Necropsies, however, were more frequently performed on males.

Regarding cardiac enlargement my experience differs from Dr. Crafoord's. In only 4 of my 36 cases was the heart much enlarged, and in all four there was some additional lesion that would account

for the enlargement: three had aortic incompetence and one a freely patent ductus.

MAURICE CAMPBELL spoke of the gradual rise of blood pressure observed directly in some cases and deduced from analysis of all reported cases (*Brit. Heart J.*, 1947, 9, 203).

S. SUZMAN described a method by which the collateral circulation in the back could be demonstrated or increased (*Brit. Heart J.*, 1947, 9, 187).