Section of Medicine

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DISCUSSION ON AORTIC STENOSIS

Dr. Raymond Daley:

Little advance has been made in the basic understanding of naturally acquired valvular disease since the classical observations of Corrigan in 1832, who described the dynamics of regurgitation, and of Hope in 1835, who noted "the small, weak arterial pulse of aortic stenosis while the heart is giving a violent impulse".

The ætiology of aortic stenosis is often not clear either on clinical or histological grounds. It is a pity that some writings in the past have been so dogmatic in stating that juvenile rheumatism is the cause of the lesion in almost every patient. Congenital aortic stenosis is an important disease with considerable possibilities of surgical improvement. I believe that a number of patients suffer from arteriosclerotic stenosis and there are, of course, a few rare causes such as fibro-elastosis. The historical evidence of rheumatism or chorea in 50 of our patients is less than 50% but a history of rheumatism is a little more common in females than in males. At post-mortem 20 patients had rheumatic aortic stenosis, 6 had congenital lesions, but in 24 it was not possible to give a firm ætiological diagnosis.

Leonardo da Vinci (1513) was the first to show the normal aortic valve opened to give a triangular orifice.

McMillan (1955), working in our Department at St. Thomas's Hospital, has studied a number of fresh post-mortem aortic valves by cinematography. He has shown that as long as the free borders of the cusps are mobile and shut well there may be considerable calcification in the peripheral parts of the cusps and walls of the sinuses of Valsalva without limitation of flow in the valve area. Some fusion of the peripheral parts of the commissures can occur without causing symptoms. Out of 30 specimens studied the shape of the orifice could be roughly divided into certain groups which are:

(i) Early peripheral fusion with good function centrally (3 cases).
(ii) Peripheral calcification not affecting function (1 case).
(iii) Fusion of one commissure (14 cases).
(iv) Partial or complete fusion of two commissures (5 cases).
(v) Partial or complete fusion of three commissures, often with incompetence, and
(vi) Cone-shaped valve with an ellipsoid orifice at the apex, and no sign of former commissures.

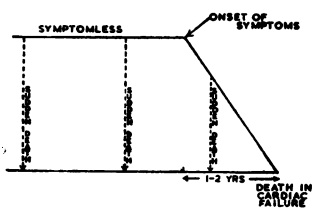

FIG. 1A.—Aortic stenosis.

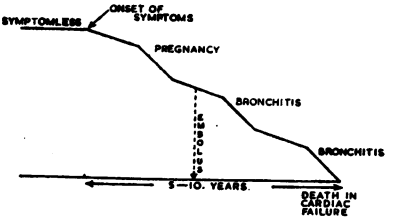

FIG. 1B.—Mitral stenosis.

The life-history of the disease differs significantly from that in patients suffering from mitral stenosis (Fig. 1). The patient is more likely to be male, and in his late forties. He is unlikely to have had serious symptoms for a long time. Once they have developed the first hospital admission may well be the last. Grant (1933) has studied the after-histories

of 43 men with aortic stenosis and incompetence for ten years. 15 died during this time. Two of the most striking features are the considerable liability to sudden death and the comparative rarity of auricular fibrillation.

Apart from left ventricular failure the two most interesting symptoms of aortic stenosis are effort syncope and angina pectoris. Angina occurs in about half the patients and is particularly prevalent in young women. The cause of the pain seems to be quite clear. Left ventricular work is greatly increased, the mean coronary filling pressure reduced, the cardiac output fairly small and fixed, and diastole is shortened. I think that it is a little unnecessary to invoke a sucking action at the coronary ostia due to the jet of blood entering a dilated ascending aorta.

The mechanism of effort syncope is not clear. I doubt if it is due to an arrhythmia, although presumably ventricular fibrillation is the cause of sudden death. Syncope can be initiated by emotion, effort and heat in anyone with a fixed cardiac output. Although the blood flow tends to be low and rather fixed, effort syncope is uncommon in mitral stenosis and hypertensive heart failure, because the blood pressure does not fall. In the former, chronic pulmonary capillary congestion exists, leading either to immediate disabling pulmonary symptoms or to reflex systemic vasoconstriction. In the latter the systemic vasoconstriction is not readily released on effort. Effort syncope, however, is not uncommon in primary pulmonary hypertension. The minute output again tends to be fixed but the blood pressure falls on effort, presumably when muscle vasodilatation occurs, because there is no pulmonary capillary congestion to inhibit its occurrence. Perhaps dilatation can be similarly initiated in aortic stenosis because of the normal and only slowly rising pulmonary capillary pressures. In 2 patients suffering from aortic stenosis whom I have observed fainting there was a bradycardia and the episode appeared to have the quality of a vasovagal attack.

I do not wish to dwell on the physical signs but I should like to make a plea for the abandonment of attaching diagnostic importance to the presence or absence of a systolic thrill in the aortic area. It is surely more sensible to feel the carotid arteries or the aortic arch at the root of the neck. Coarse vibrations felt there are diagnostic of aortic stenosis and are quite unlike the smooth thrill felt in an aortic to-and-fro murmur due to predominant aortic incompetence. The hearing of the murmur over the olecranon processes is no more than a clinical *tour de force*. If the murmur is loud enough it will have wide radiation. An important point to recognize is that if aortic stenosis is very severe blood flow may be insufficient to produce a murmur. This is sometimes seen in the operating theatre, when pressures are simultaneously measured in the left ventricle and aorta. If the cardiac output is sufficiently reduced there may be no pressure gradient across an aortic stenotic valve. A useful sign is an early systolic click associated with post-stenotic dilatation of the ascending aorta.

Valve calcification (Fig. 2) is very common although I know of no means of assessing

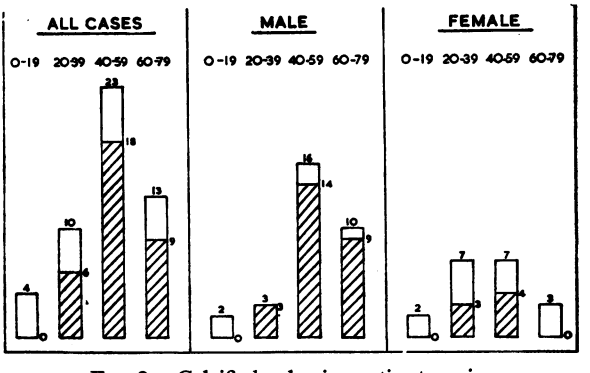

FIG. 2.—Calcified valve in aortic stenosis.

whether it is in the valve or the valve ring. It is best seen with the image intensifier but ordinary fluoroscopy is so satisfactory that if it had been invented after tomography it would have been hailed as an advance.

A great deal has been said about pulsus bisferiens in combined aortic stenosis and incompetence. It may be a very useful sign but varying pressure of the palpating finger may give the impression of pulsus bisferiens where none exists. The most important

measurement in the peripheral pulse is delay in the systolic upstroke time. This is prolonged to between 0·175 and 0·3 second in five cases of aortic stenosis (Fig. 3) and may revert to normal after successful valvotomy.

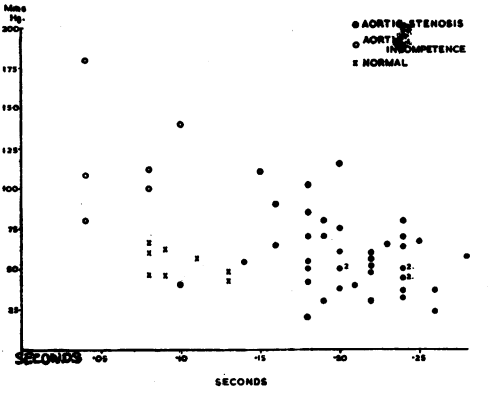

FIG. 3.—Brachial artery pulse pressure and systolic upstroke time.

In 1955 Gorlin joined us at St. Thomas's Hospital in a hæmodynamic study of a number of patients with aortic valve disease. Some of the deductions were made by a combination of cardiac catheterization and study of other hearts in McMillan's artificial perfusion system. With the advent of direct left ventricular puncture either through the chest wall as Brock practises or via a bronchoscope and the insertion of a fine polythene tube as we have been doing, these deductions have been found to be substantially correct. They are that:

The "critical" orifice area in pure aortic stenosis is 0·5 sq. cm., in mixed stenosis and incompetence less than 1·5 sq. cm. With these respective degrees of stenosis left ventricular systolic pressure is elevated to 200–250 mm.Hg with transvalvular pressure gradients of 100–150 mm.Hg. Because of the inability of the coronary flow to increase in proportion to the demands of ventricular work, due to low coronary perfusion pressure, the ventricular systolic pressure seldom exceeds 250 mm.Hg when no further stroke work can be done. In view of the large gradient between the aorta and ventricle in diastole, regurgitant orifice areas no greater than 0·5 sq. cm. were capable of more than doubling total ventricular output and work. Although left ventricular stroke work is increased at rest (two to four times normal) the pulmonary capillary diastolic pressure is near normal in 80%.

There are four stages of the disease:

Stage I: The valve orifice area is greater than 0·5 sq. cm. in pure cases and greater than 1·5 sq. cm. in mixed cases. The only physiological deviation will be a prolonged systolic upstroke time. The patient is free of symptoms.

Stage II: The valve orifice area is 0·5 sq. cm. or less in pure cases and less than 1·5 sq. cm. in mixed cases. Resting left ventricular diastolic pressure and blood flows are within normal limits, but on effort the diastolic pressure rises. Stroke output and work are fixed. These patients are free of symptoms except with unusual but varying degrees of effort. The limitations here are mainly hydraulic in nature.

Stage III: Orifice areas are the same but the myocardium usually shows signs of generalized patchy fibrosis from years of impaired coronary flow. The huge load borne well in Stage II is no longer tolerated on slight exercise. The resting left ventricular diastolic pressure is within normal limits or elevated slightly and blood flow is invariably decreased. Effort causes elevation in diastolic pressure, a fixed or falling output, and often acute right ventricular failure as well. These patients are limited by symptoms, first of left- and soon of right-sided failure. Thus it is not until late in the disease when the muscular component fails that symptoms become severe and the disease runs an accelerated course.

Stage IV: Terminal bilateral cardiac failure.

I should like to submit that patients should be operated upon while still in Stage II, before serious symptoms and myocardial fibrosis have appeared. This means that the majority of patients will be children and young people in whom gross valvular calcification has not yet occurred. The best operative results appear to be in young people with non-calcific congenital aortic stenosis. The worst results are in middle-aged patients in whom the valves are usually heavily calcified. It must be stressed again that any blind procedure

which leads to increased incompetence even with some lessening of stenosis is a disservice to the patient.

If operation is decided against, apart from the usual medical measures thyroid ablation is often worth while. It is best carried out with radio-iodine. Following treatment, angina is usually much improved, dyspnœa is less and sometimes the heart size decreases.

I have deliberately avoided discussing subaortic stenosis because, despite what people tell me, I believe it is a very rare disease and its diagnosis inaccurate.

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Mr. Andrew Logan (Department of Surgery, University of Edinburgh):

The Surgery of Aortic Stenosis

The views which I shall express on the surgery of aortic stenosis are based on an experience which is small in relation to some of the published figures. For example, Bailey (1956) quoted 450 cases of aortic valvotomy and Harken (1956) 70 cases. I have performed aortic valvotomy in only 31 cases. 7 of the patients had congenital aortic stenosis; 17 had acquired aortic stenosis without significant incompetence or other valvular lesion; and 7 had aortic stenosis combined with mitral stenosis.

The records of the first 6 patients with congenital stenosis have already been published (Marquis and Logan, 1955). Each underwent pre-operative angiocardiography in an attempt to exclude subvalvular stenosis since it seemed inappropriate to attempt enlargement of a subaortic ring by the same technique as was used for the separation of fused cusps. All were operated upon more than three years ago. None has died. In all, the signs of aortic stenosis were reduced. In 5, however, aortic incompetence was produced or increased, and in 2 the incompetence was severe. The single patient who had no reflux after operation still has none and is well. The result in her case is good enough to indicate that the aortic cusps were flexible and well-formed and that valvotomy achieved extensive separation of the cusps accurately in the line of the commissures. The occurrence of slight to severe, but not lethal, aortic incompetence in the other 5 cases, along with a reduction of stenosis, suggests that in these, too, considerable separation of the cusps in the correct line was obtained, but that the cusps were ill-formed rather than that a cusp was divided at operation. The most severe incompetence occurred in a boy of 13. He still has severe incompetence and the heart is larger than it was 6 months after operation, but whereas he was, at 13, a weedy boy 14 lb. underweight he is now, at 16, 3 st. heavier, up to normal weight, asymptomatic and leading an energetic life. In all, an improvement in cardiac function and in general health has lasted for three years after valvotomy. It remains to be seen how long the heart will compensate for reflux before a comparison can be made with the calculated effect of unrelieved stenosis. Meantime, there is no reason to change the opinion that valvotomy for congenital aortic valvular stenosis should be reserved for those patients who are in danger of sudden death or of deterioration which might prejudice the outcome of a later operation. The risk of incompetence does not justify withholding operation in growing children who show the electrocardiographic pattern of extreme left ventricular hypertrophy and in adults who show progressive left ventricular hypertrophy with increasing cardiac size.

The seventh patient in the congenital group was not investigated by angiocardiography. At that stage it was assumed that severe aortic stenosis was almost certainly valvular and not subvalvular. Inspection of the heart at thoracotomy raised no suspicion that the stenosis was subvalvular. The same blind transventricular approach was used. It was immediately obvious that a different condition had been encountered because the obstruction, instead of being disrupted with ease, strongly resisted the opening of the dilator. I exerted a degree of force quite inappropriate to valvotomy and succeeded in widely opening the dilator. There was no thrill of reflux or evidence of other damage and the heart's action remained good. The state of the child gave no cause for anxiety until sudden death occurred five hours later. At autopsy a fibrous ring was found 0·7 cm. below the lowest part of the aortic cusps, which were normal. The ring had been ruptured posteriorly and

the tear which extended into the ventricular wall passed upwards in the aortic wall between the right and left posterior cusps, then above the left coronary orifice. None of the cusps was damaged. Death may have been the result of obstruction of the mobilized left coronary orifice although there was no pathological evidence that obstruction had occurred.

The 17 patients with acquired aortic stenosis and no other major valvular disease were selected in the belief that the duration of their survival without operation was likely to be less than eighteen months. The records of some of these also have been published (Logan and Turner, 1954). All had more than one of the triad, syncope, angina, and congestive heart failure. Only 2 had uncalcified valves.

Of these 17, 9 are dead, 1 during operation, 3 within three weeks of operation, and 5 from six to thirty months after operation. The immediate death was the consequence of neglect of the elementary precaution of controlling the ventricular incision with a tourniquet. The search for the orifice among the calcific masses of the valve was more prolonged than usual, blood leaked continuously, ventricular fibrillation supervened and normal rhythm could not be restored. Of the 3 early deaths, 1 was from pulmonary infarction three days after operation. The patient had intractable cardiac failure for which a year before I had ligated the inferior vena cava. She had a severe degree of hepatic cirrhosis. One patient died nine days later from coronary thrombosis with a large cardiac infarct. 2 of the patients who died later had been temporarily improved and the others followed the uninterrupted course of severe aortic stenosis. 2 were examined *post mortem.* Both had rigid valves, one without evidence of division. One heart weighed 900 grams and the other 1,100 grams.

Of the 8 survivors, 1 did not have a valvotomy because I was unable with the maximum permissible force to rupture a calcific ring surrounding the valve orifice. All the others have some increase of aortic reflux. One has a left homonymous hemianopia, the result of calcific embolism. Because of a technical difficulty the head vessels had not been occluded during valvotomy. One is not obviously improved. 5 have lost their important symptoms and follow normal activities. One is asymptomatic. Pre-operatively he was dyspnœic and suffered from angina and syncope. He had been in congestive heart failure. For five weeks after operation he remained in heart failure but three months later returned to office work and has remained well for three years.

That is, in 17 cases of acquired aortic stenosis there were 4 early deaths, 5 late deaths, 2 unimproved and 6 with improved cardiac function, one of them much improved. None had a severe degree of aortic reflux.

The 7 patients with combined aortic and mitral stenosis were all initially improved. None died and none had severe aortic reflux. Since the gradient across the aortic valve was not measured, I have no means of apportioning the benefit to the two operations or of indicating how the aortic valve was affected.

If the selection of cases of aortic stenosis for operation is restricted to those in which cardiac impairment is so severe that death is imminent, the question arises whether surgical intervention by blind valvotomy is profitable. The opinion has been given that we merely deduct a few months from one patient's life and add them to that of another without a net gain. Taking the worst group, that of pure acquired aortic stenosis, that is clearly not so. In the whole group there were few years to come and all of them bad. There have already been three good years and ten fair years, which compares well with the results of the surgery of carcinoma.

The technique used in all cases was the same. Through a left thoracotomy a two-bladed expanding dilator with a spread of 4 cm. was introduced through an incision in the anterior wall of the left ventricle and placed in the aortic valve under the guidance of external palpation of the origin of the aorta. The dilator was opened as far as that could be done with a reasonable amount of force or until the aortic wall was stretched over it. Then the dilator was withdrawn and the ventricular wall was repaired. This is an exceedingly crude method of valvotomy but I can say that by the use of a similar technique in mitral stenosis the commissures are more often and more completely divided, and with less damage, than I can achieve by any other way. The dilator sometimes selects a commissure when I am unable to recognize it by digital palpation. It is at least possible that the same would be true in aortic stenosis. The absence of sudden severe post-operative aortic incompetence suggests that in none was a cusp completely divided. I am unconvinced of the need for a three-bladed dilator or of the likelihood that the blades will occupy the commissures. Again comparing the mitral operation, the valvotomy is simply a matter of tearing by stretching and, whatever the direction of the stretch, the tear occurs in the weakest line which is fortunately usually the commissure. The amount of force required to tear a cusp is so great as to be easily recognized and avoided. If the aortic cusps are malformed or destroyed no technique at present available will make their function good

If the cusps are well formed the dilator will probably separate them as effectively as any other instrument (Fig. 1A and B).

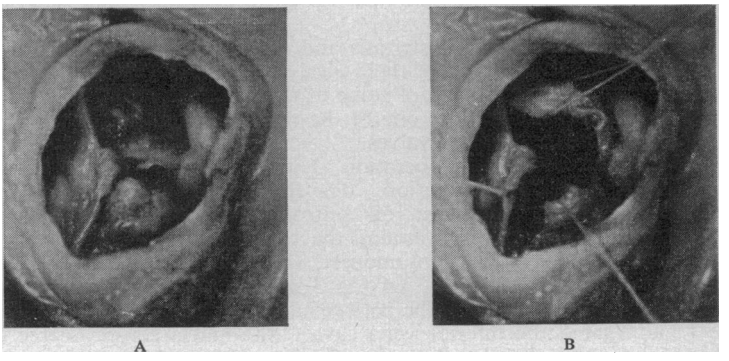

FIG. 1.—Acquired aortic stenosis without calcification after valvotomy. A, Valve closed; B, Open. The appearance of rigidity is the consequence of fixation. In the fresh state the cusps had almost normal mobility.

Bailey (Bailey *et al.*, 1956) began to approach the aortic valve through the ascending aorta for two reasons: because he had encountered ventricular fibrillation with the trans-ventricular route, and the thick friable ventricular wall had sometimes been difficult to repair. He did not by the change eliminate trouble from ventricular fibrillation and bleeding but continued to use the trans-aortic route for the sake of avoiding injury to the ventricular muscle and of using the finger both for the performance of the valvotomy in some cases and the assessment of the quality of the valvotomy in all. I believe that the expanding dilator usually makes a less traumatic valvotomy than the finger because each blade provides counter-pressure for the other, whereas the finger must pull upon the pedicle of the heart or against externally applied pressure. Digital palpation of the valve after valvotomy would certainly be helpful in prognosis and might sometimes lead to modification of the state of a commissure after the first dilatation. Whatever route is used, ventricular fibrillation is likely to occur in some cases. The massive thickness of the left ventricle and the small left ventricular cavity make effective cardiac massage, as judged by the presence of a radial pulse, difficult or impossible and electrical defibrillation must be used without delay.

With the increasing use of refrigeration and of pump-oxygenators to permit open cardiotomy in other conditions it is to be expected that they should be used for aortic valvotomy under vision. In the United States, Lewis *et al.* (1956) and Swan (1956) have used refrigeration and Lillehei *et al.* (1956) the pump. In the short time afforded by refrigeration I doubt that with knife or scissors I could make as accurate a dissection of the commissures as competence of the valve demands, even in an uncalcified valve with well-formed cusps. Where the cusps are deformed or the commissures obscured or calcified I should prefer to rely on the dilator for their separation. For the division of a subaortic stricture, however, a visual approach may be desirable and the three to five minutes afforded by refrigeration may be adequate for this. Probably neither refrigeration nor the pump-oxygenator is necessary for the correction of aortic valvular stenosis so far as that can be done now, just as they are unnecessary for the correction of mitral stenosis. But the visual approach through the aorta with the pump-oxygenator has importance for the future since it will be by that method that the aortic valve will be assessed and repaired or replaced by a prosthesis when an efficient and durable one can be constructed.

It is clearly important in cases of congenital aortic stenosis to know pre-operatively the site of stenosis. For this purpose some reliance has been placed on angiocardiography although evidence that it would demonstrate a subaortic stricture is lacking. Some help might be derived from manometry of the left side of the heart by left ventricular puncture as practised by Brock *et al.* (1956). In manifestly severe acquired aortic stenosis measurement of the systolic gradient across the aortic valve before operation cannot contribute much. If, as is likely, the maximum opening of the valve is completed with one dilatation, measurement of the gradient during operation, before and after valvotomy may aid prognosis but nothing else. When aortic stenosis complicates mitral stenosis, however, measurement of the gradient across the aortic valve after mitral valvotomy is required for the decision on proceeding to aortic valvotomy. For lack of that investigation I have on two occasions

omitted aortic valvotomy at the time of the mitral operation and been obliged to repeat the thoracotomy a few months later. The principal value of pre-operative measurement of a pressure gradient and blood flow at the aortic valve is probably in the assessment of patients who, having undoubted aortic stenosis and left ventricular hypertrophy are yet asymptomatic and have little valvular calcification. It is in this group that the best result from valvotomy is to be expected but the prognosis is so uncertain that the achievement by operation cannot be estimated. There is no obvious way of overcoming this difficulty but to find a large enough number of these cases by routine cardiac examination and, having overcome hesitation to use ventricular puncture in asymptomatic patients, to select an arbitrary degree of severity of stenosis as indicated by the integration of pressure gradient and blood flow at the aortic valve; then, rather than operate on all with more severe stenosis (a course which could give information only about the undesirable consequences of valvotomy) or continue to observe the natural course in all, to make control series for operative and conservative management.

SUMMARY

(1) Although the results of aortic valvotomy in patients who have been gravely damaged by aortic stenosis are poor they are worth while.

(2) The blind transventricular operation probably gives a valvotomy as accurate as other methods but the trans-aortic route must be expected to be required in due course for replacement of the valve.

(3) In symptomatic pure aortic stenosis, measurement of the pressure gradient across the aortic valve during thoracotomy can contribute to prognosis but in combined aortic and mitral stenosis it may be necessary for a decision on the need for aortic valvotomy.

(4) The use of aortic valvotomy should be extended to asymptomatic patients investigated by measurement of the pressure gradient and blood flow across the aortic valve and known to have severe stenosis, but an attempt should be made to compare the prognosis with and without valvotomy.

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Dr. J. F. Goodwin (speaking at short notice in place of Professor J. B. Duguid):

I propose to discuss the study of aortic stenosis by means of the cardiogram and arterial pulse tracing, and wish to present the results of some work (with which I have been closely associated) by my colleagues, Drs. Abdin, Doyle and Neilson, at the Postgraduate Medical School.

Abdin (1957) has studied the cardiograms of 53 cases of aortic stenosis (25 of whom are living), and compared them with 19 cases of coarctation of the aorta, 15 cases of hypertension, and 4 cases of hypertension associated with ischæmic heart disease. Deep T-wave inversion (greater than 5 mm.) in left præcordial leads was found in 26 of the cases with aortic stenosis, but in only one of the hypertensive and coarctation cases respectively. By contrast, it was found in 3 of the 4 cases with ischæmic heart disease and hypertension. T-wave inversion was found to be appreciably greater in cases of aortic stenosis than in those with hypertension who had equal R-wave voltage, and equivalent degrees of left ventricular hypertrophy at autopsy. This disproportionate T-wave inversion suggested that some factor other than myocardial hypertrophy was responsible, and the finding of ischæmic lesions in the myocardium in the absence of coronary artery disease in 13 cases of aortic stenosis at autopsy suggested that this factor was cardiac ischæmia, which results from deficient coronary blood flow due to the obstruction to the outflow of the left ventricle. The presence of very similar degrees of T-wave inversion in the cases with ischæmic heart disease and the known association of anginal pain with aortic stenosis harmonize with

this view. In 6 cases of aortic stenosis and 3 of hypertension an RS pattern was found in V_5 in association with signs of left ventricular hypertrophy. All had pathological evidence of right as well as left ventricular hypertrophy, supporting the findings of Pagnoni and Goodwin (1952).

Doyle and Neilson (1957) have studied the relation of the systolic upstroke time of the peripheral arterial pulse to the pulse pressure in aortic stenosis with and without incompetence. In aortic stenosis the systolic upstroke is usually prolonged and the pulse pressure usually low, but close correlation with the severity of the stenosis is often not obtained. When the pulse pressure was related to the upstroke time during changes induced by the Valsalva manœuvre, or during atrial fibrillation, cases of severe stenosis showed a considerable reduction in upstroke time for a given change in pulse pressure. In mild cases and in those with significant associated incompetence, there was little change in upstroke time for an equivalent change in pulse pressure. The reduction in upstroke time in relation to fall in pulse pressure was considerably less after successful aortic valvotomy. Expressed in another way, patients with severe aortic stenosis showed little alteration in rate of rise of the systolic upstroke during the Valsalva manœuvre, while those with incompetence showed considerable alteration. The fall in pulse pressure during the Valsalva manœuvre is probably due to a fall in stroke volume, and hence the marked reduction in upstroke time when the pulse pressure falls suggests that the systolic upstroke time is a resultant of the stroke volume and resistance at the aortic valve. The form of the arterial pulse in aortic stenosis is mediated, at least partly, by changes in peripheral resistance, since pulses of equal size recorded at various stages during the Valsalva manœuvre were often of different contour.

Measurement of the systolic upstroke time in relation to changes in the pulse pressure in a number of cardiac cycles offers a better guide to the severity of stenosis than measurement of the resting tracing.

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Mr. W. P. Cleland said that he had performed aortic valvotomy on 15 patients during the last three years. In only 1 of these was the ætiology believed to be congenital. In 9 there was pure calcific aortic stenosis, and in 5 there was mitral valve disease in addition to the aortic stenosis. All these patients belonged to Dr. Daley's Stage III.

He would entirely agree with Dr. Daley that his Stage II cases were likely to give better results with surgical treatment, but he disagreed that the Stage III cases should not be submitted to operation, as the follow-up of his own small group indicated.

Of his 15 patients, 3 died either at operation or in the immediate post-operative period—2 from cardiac arrest and 1 from a calcific cerebral embolus.

Of the survivors, only 1 has died. The remainder have been followed-up for periods of six months to three years and all have maintained their original improvement.

The majority of the patients had symptoms of angina, syncopal attacks and dyspnœa, and considerable relief of symptoms was obtained in the majority. This improvement was associated with equivalent objective signs of benefit.

The transventricular approach was employed in all, using a triradiate mechanical dilator. Interference with the myocardium was reduced to a minimum and blood loss from the ventricle was very carefully avoided. Lack of attention to these two details was certainly responsible for two of the operative deaths.

The effects of the operation were determined by measuring the gradient across the aortic valve and by determining the systolic upstroke time of the pulse wave. The measurement of the gradient gives a valuable idea of the degree of obstruction and the amount of relief obtained, provided conditions at the time of successive measurements are as nearly similar as possible.

Mr. Cleland was quite satisfied with a 50% reduction in gradient even though the latter was still high. Repeated attempts at dilatation in an attempt to reduce the gradient often resulted in troublesome or fatal cardiac irregularities.

The follow-up of these cases has suggested that a fairly conservative approach is quite justified. He has not felt the need to approach the valve through the aorta but considers the performance of aortic valvotomy under direct vision in a bloodless field and a motionless heart is probably likely to be the operation of choice in the future.

Dr. Ronald Gibson said that at The Brompton Hospital and at Guy's Hospital, direct puncture of the left ventricle was performed in 60 patients. The results in the first 28 cases had been analysed in detail by Dr. Peter Fleming and himself (1957). The procedure was simple and safe, but the systolic pressure gradient across the aortic valve obtained by this method must be interpreted with caution. This gradient depended not only on the size of the valve orifice, but also on the stroke volume, which is related to cardiac output, heart rate and the degree of aortic regurgitation. For example, he had found that with an estimated valve size of 0·25 sq. cm. at operation the systolic gradient at left ventricular puncture varied between 30 and 210 mm.Hg. These results are considered to be due to differences in stroke volume. A gradient of up to 100 mm.Hg had been found with dominant aortic regurgitation. However, all cases with a systolic gradient of 120 mm.Hg or more had dominant aortic stenosis. There was, therefore, little correlation between height of a pressure gradient and the surgeon's estimate of the valve size. In the absence of significant aortic regurgitation there was a fair correlation between the surgeon's estimate of the valve size and the calculated aortic valve area. The left ventricular ejection period was prolonged in all cases, but degree of prolongation gave no indication of the degree of valve obstruction. Therefore they considered that paradoxical splitting of the second heart sound did not necessarily indicate severe aortic stenosis. On the other hand, it was found that the degree of prolongation of the corrected brachial upstroke time was related to the severity of the stenosis.

A direct relationship had been found between the systolic gradient and the degree of left ventricular hypertrophy on the electrocardiogram in all the cases in this series, irrespective of whether stenosis or regurgitation was the dominant lesion.

REFERENCE

FLEMING, P., and GIBSON, R. (1957) *Thorax*, **12**, 37.

Mr. Ian M. Hill:

I have carried out 12 aortic valvotomies in adults, 2 in isolated aortic stenosis and 10 combined with mitral valvotomy, all by the transventricular approach at normal temperatures.

The 2 isolated cases were severely disabled, 1 with right and left failure and the other with angina. The case with failure had a good result and now two and a half years later is beginning to slip back again. The anginal case died of irreversible ventricular fibrillation at operation, one mass of calcium in the valve producing partial rupture of the aortic wall when pressed against it by the dilator, leading to a hæmatoma about the origin of the left coronary artery.

Electromanometer pressure readings have been taken in all cases at operation; but the gradient across the aortic valve is not always helpful in assessing the degree of stenosis under these conditions as the operative handling of the ventricle may grossly alter the output and therefore the gradient across the valve. Usually after mitral valvotomy the aortic gradient increases, but where the left ventricle has been manipulated it may even fall despite a good mitral valvotomy. Direct sounding of the aortic valve through the apex of the left ventricle is a more reliable assessment and in the 10 cases mentioned and another 6 in whom the aortic valve was sounded, but not dilated during mitral valvotomy, there was no mortality and no morbidity in this method of handling the problem. The functional results have been good.

Mr. D. N. Ross said that Sir Russell Brock had performed about 130 aortic valvotomies (1957), mostly by the transventricular route with good results. He agreed with Mr. Cleland that there was danger in prolonged manipulations with the dilator in situ while trying to abolish the gradient entirely. This was likely to precipitate ventricular fibrillation. The overall mortality figures were approximately 10%, the figure being higher in the earlier cases and lower now.

In addition, he had operated on about 15 cases of congenital aortic stenosis under direct vision through the aorta with the aid of hypothermia.

There had been a surprising number of cases of subaortic stenosis encountered—about 11 cases or approximately 10%. At present there is no entirely satisfactory method of demonstrating this lesion pre-operatively and attempts had been made to do catheter withdrawal tracings via a LV puncture needle. The present attitude is that calcification of the

valve is so common in isolated aortic valve stenosis in patients of 30 years of age that subvalvar stenosis is suspected in any patient of this age who does not have a calcified valve on screening. At operation, in a suspected case, withdrawal pressure records are taken with a catheter and these show an "infundibular" type of tracing below the valve. Subvalvar stenosis is generally of a diaphragmatic nature—consisting of a ring of puckered endocardium about 1 cm. below the valve and this is split by means of blunt transventricular dilatation.

REFERENCE

BROCK, R. C. (1957) *Brit. med. J.*, i, 1019.

Mr. B. B. Milstein:

I should like to add two points which have emerged from Sir Russell Brock's experience. The first is the hopelessness of operating on patients in intractable cardiac failure from aortic stenosis. Such patients were submitted for operation early in his series and the mortality was extremely high. These cases are now rejected as beyond hope of surgical treatment. Patients who have been in right- or left-sided heart failure but who respond to medical treatment are not subject to the same operative risk and very good results have been obtained in these advanced cases but it is desirable to wait as long as possible, preferably three months after the attack of cardiac failure, before proceeding to operation.

The second point concerns the results of operation. This is not entirely a matter of duration of life after operation. Anginal pain and syncope, for example, can usually be completely relieved. The effect on exertional dyspnœa is variable. Some patients may die subsequently from left ventricular failure and this is not surprising when one sees the gross hypertrophy of the left ventricle and the extreme degree of fibrosis in the muscle which is present in advanced cases. These changes are clearly irreversible. Nevertheless, these patients' lives may be made tolerable by the relief of angina or syncope as a result of operation.

Dr. C. J. Gavey mentioned the value of the symptoms of weakness and ready exhaustion of fairly sudden onset, in drawing attention to the possibility of serious deterioration in the prognosis of aortic stenosis. Such symptoms were so often due to anxiety that there was a risk of overlooking their alternative origin in the falling cardiac output due to aortic stenosis.

Dr. Gavey stressed the difficulty of deciding whether or not to offer surgery in the more advanced valvular lesions, especially since certain mechanical estimations of function, however carefully performed, could sometimes be misleading.

Mr. I. K. R. McMillan said that he had carried on his investigation of the function of stenotic aortic valves beyond the figures quoted by Dr. Daley and that his experience now included some 50 cases. The classification of the type of orifice still fell into six main groups, which were as follows:

(i) Early peripheral fusion with good function centrally (4 cases).
(ii) Peripheral calcification, not affecting function (2 cases).
(iii) Fusion of one commissure (24 cases).
(iv) Partial or complete fusion of two commissures (8 cases).
(v) Partial or complete fusion of three commissures, often with incompetence (8 cases).
(vi) Cone-shaped valve with an ellipsoid orifice at the apex, and no sign of former commissures (3 cases).

He pointed out the risk in group 4 when doing a blind valvotomy of tearing the relatively normal commissure and producing incompetence while not splitting the fused calcified commissures, and suggested that direct vision surgery of the valve would be advantageous in these cases.