THE SURGICAL TREATMENT OF AORTIC STENOSIS*

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In this short presentation I shall discuss the following features of aortic stenosis: (1) The basic indications for operation, (2) certain features of diagnosis, and (3) the type of operation to be performed and the results.

1. INDICATIONS FOR OPERATION

I have written and spoken extensively upon this matter of the need for operation in aortic stenosis[1] but, at the risk of being tedious, I shall make some comments.

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The attitude to aortic stenosis seems still predominantly to be governed by a mixture of complacency and of fear. We still hear the problem presented as that of a patient, usually a man, in the fifties or sixties who, having had a heart murmer for many years with trivial or no symptoms, develops symptoms of varying urgency. From this it is still argued that it is proper, at any age, to procrastinate because the patient may similarly live for many decades.

This is quite false, for aortic stenosis is a crippling and killing disease even in the very young. In children and in

adolescents it is particularly dangerous because of the simple fact that their young, strong heart muscle can over-exert itself abundantly and not show any symptoms of the strain to which it is constantly submitted. In this very tolerance lies the greatest danger, because when the muscle fails it fails rapidly. Sudden death is quite common; it is usually preceded by a period of the urgent features of left ventricular failure, but not always.

Similarly, it is my experience that in the twenties and thirties when clinical symptoms appear the disease is far advanced and if life is to be saved operation must be advised and performed promptly.

The complacency of the past is today often fostered by fear of the dangers of operation. This attitude is quite indefensible, for the medical mortality is very high, sooner or later approaching 100%, whereas operation can be done with low mortality and good results.

I must remind you that the essential fault is obstruction to the emptying of the left ventricle, and that life itself depends on the integrity of the function of this chamber. If relief of the obstruction of any hollow viscus or duct in the body is an indication for surgery then obstruction of the left ventricle is surely a paramount indication! Unfortunately, until relatively recently the clinician was often at a disadvantage in giving advice about the need for operation with any degree of certainty, but today there is little excuse for guessing, for we have methods of investigation which allow us to advise with confidence and accuracy.

I will remind you of the 5 phases into which aortic stenosis may be conveniently divided. They are:

1. No symptoms.
2. An awareness of the heart, e.g. palpitations and some dyspnoea at times.
3. Anginal pains, usually with severer dyspnoea.
4. Features of left ventricular failure as shown by congestive attacks, orthopnoea, angina at rest, syncope, etc.
5. Right-sided heart failure.

There is, of course, a certain amount of overlap in these groups, e.g. a patient may be late in phase 3, tending to spill over into phase 4.

From our experience in correlating these phases with electrocardiographic changes, with left heart catheterization, and with operation findings, we can be fairly dogmatic about the need for operation where the diagnosis of aortic *valvar* disease is certain. Thus in grades 3 and 4, operation is essential and in general no pre-operative left heart catheterization is needed. In grade 5, operation is attended with a very high mortality; most such cases are inoperable unless their failure can first be lessened by medical treatment. It is in grades 1 and 2 that it is usually necessary to apply special methods of investigation to assess the severity of the obstruction.

I must also warn against being too easily deterred from operation by the presence of some aortic regurgitation. If the clinical picture is dominated by regurgitation then operation may not be indicated, but it is much commoner to find cases rejected when the degree of regurgitation is modest and the degree of stenosis is dominant. One should concentrate on the degree of stenosis and tend to ignore the regurgitation whenever possible, certainly not use it as an excuse for avoiding operation.

2. FEATURES OF DIAGNOSIS

The electrocardiogram is invaluable if a strain pattern is shown in the left ventricular leads, but this usually occurs in grades 3, 4 and 5. In grades 1 and 2 there may be only slight changes, and this is especially so in children when, in spite of this, a severe stenosis is already present.

Left ventricular puncture combined with estimation of the cardiac output is invaluable and is now the routine practice in most centres.[2] It is quick, simple and safe and can, if necessary, be repeated on successive occasions. We find in grades 3 and 4, when the left chest leads show a severe strain pattern, that the valve gradient is usually not less than 100 mm. Hg, and this is associated with a low cardiac output. Unless there are unusual features we delay our pressure estimations in these groups until the time of operation. However, we press for their routine employment in groups 1 and 2 and especially in young children.

During 1958, at Guy's Hospital, we had 3 examples of children in whom symptoms were absent and the electrocardiographic changes were slight, and yet there was a pressure gradient across the aortic valve of over 100 mm. Hg. One of these children actually developed angina and syncope 3 months later while awaiting admission for open operation. It is not possible to give intelligent and confident advice in these cases without left heart catheterization.

Valvar and Subvalvar Aortic Stenosis

In all young patients and in patients of any age in whom the valve is not demonstrably calcified we demand actual catheterization of the left ventricle so as to obtain a pressure withdrawal record.[3] In this way we can assess whether the stenosis is valvar or subvalvar. In one patient we have even recognized a supravalvar aortic stenosis, confirmed it by a retrograde aortogram, and then operated on it successfully under bypass.

A congenital subvalvar obstruction has been met with in 16 cases and has been made the subject of a special report.[4] In most of these the diagnosis was made by left ventricular puncture, but occasionally, when the stenosis is very near the valve, differentiation of the exact level is not possible and the true diagnosis can then only be made at open operation. I would refer you to the above article[4] for critical discussion of the various technical features; but I would state that at present I prefer a closed transventricular dilatation for most cases of congenital subvalvar stenosis and consider the pre-operative recognition important because one may then be able to avoid the open operation under bypass which is needed when a non-calcified stenosis exists.

Functional Aortic Subvalvar Stenosis

The estimation of the exact level of the stenosis is also essential in recognizing the important condition of functional aortic subvalvar stenosis. This condition has been the subject of two communications[5,6] and I refer you to these for fuller details. Not only is there still wide ignorance of this disease, but even when acquainted with the account of it many clinicians are frankly sceptical. This is unfortunate, because the severity or urgency of symptoms often indicates need for relief by operation whereas operation cannot give relief and in most cases an ill-advised cardiotomy is fatal. I have now met 7 cases of this disease in quite a short time and must emphasize that its recognition is an important

practical matter in aortic stenosis in adults and *in every case* it must be thought of and deliberately excluded.

The most important single observation is the recognition that the valve is not calcified. In aortic valvar stenosis in patients over the age of 30 there is usually little difficulty in demonstrating calcification radiologically. Unless this can be done then the diagnosis of valvar stenosis must be suspect. Occasionally the valve is not calcified in cases of rheumatic disease but in almost every such case the presence of mitral valve disease supplied the true explanation. In all other cases of isolated aortic stenosis it is essential to *prove* the presence of calcification before the diagnosis of valvar stenosis is accepted. Personally, I do not rely upon screening reports, nor upon negative tomography, but insist that plain radiological confirmation be obtained.

If calcification is not demonstrable then it is folly to proceed to operation without a pressure withdrawal record obtained by left ventricular puncture. In this way a diagnosis can be made in a few moments and was, indeed, so made in all our patients. For one reason or another 4 of these were operated on and 3 died at once. The exception was a man aged 32 in whom a lesion was first noticed when he was aged 16. Exploration was done because a congenital fibrous stenosis could not be excluded. Operation revealed a functional obstruction, and fortunately, he did not die. Other surgeons have encountered such cases in the early twenties; so it must be thought of and if possible excluded, even at this young age.

In one of my patients the predisposing factor was left ventricular hypertrophy from previous systemic hypertension. This was not a factor in the remainder, in whom we have to fall back upon the explanation of an obscure cardiomyopathy. I do urge you not to ignore the great importance of this new disease of the left ventricle—functional aortic subvalvar stenosis—hitherto unrecognized until revealed by left ventricular pressure withdrawal records.

3. TYPES OF OPERATION

Much of the time that is spent arguing about the exact type of operation to be done for aortic stenosis could be better spent in emphasizing the basic need for an operation of any sort. More patients are dying or continuing unrelieved of their disability without any operation than are being submitted to open or closed procedures. I have visited clinics and hospitals in many parts of the world and am impressed with the continuing reluctance to advise and to use surgery in this serious disease. Even when operation is used it is commonly only for the advanced or desperate cases, rarely at the proper time when there is the greatest chance of a good result with a low mortality.

Non-calcified Aortic Valvar Stenosis

I have already mentioned that I prefer a closed transventricular operation, but that an open operation is essential for a non-calcified valvar stenosis. Those of us who have tried closed dilatation on such cases know that the smooth, high, dome-shaped valve is not amenable to safe splitting with an instrument. The valve cone is torn and damaged so that severe regurgitation follows. The result is either fatal or an aggravation of the clinical state. Visual division of the fibrous valve cone is essential.

In common with many surgeons I have done this under hypothermia and have in fact operated on 10 patients in this way. Three patients died and the result was good in 5 and not so satisfactory in 2, in whom substantial regurgitation occurred. It is now my custom to use total heart-lung bypass for these cases because the longer time available is desirable for 3 reasons. First, one can do the actual valvotomy with greater prevision when unhurried; second, one can spend adequate time on careful suturing of the aortic wall incision; third, the ability to give powerful coronary perfusion is invaluable in avoiding or correcting arrest of ventricular fibrillation when the muscle is in poor condition. I find, in fact, that I use some 30 minutes of bypass for these cases and would hate to go back to the hurried work needed to complete the delicate task in the 10 minutes available with hypothermia. The safe and secure suturing of the aortic wall is especially important. It may be possible to rely upon a lateral clamp and then to complete the suture at leisure, but this may fail and then disaster can follow.

The avoidance of regurgitation is paramount, and when a homogeneous, smooth dome is displayed (as is the case in severe stenosis) I prefer to make only 2 incisions, thus producing a bicuspid valve. However, in certain cases, and especially the less severe ones, the 3 commissures must be cut and then the greatest care must be taken to cut exactly in the line of the fused edges. It is so easy to transgress to one or other side, and regurgitation then follows. This is much more prevalent and much more significant than after open pulmonary valvotomy, presumably because of the higher systemic pressure to which the valve is exposed. It can be a great disappointment when substantial regurgitation follows an open valvotomy, and this is scarcely mitigated by the hope that as the valve scleroses in later years the regurgitation may lessen.

I have not used any form of artificial coronary perfusion, either forward or retrograde; nor have I used potassium arrest in these cases. I have simply clamped the aorta and anoxic arrest has followed in all the 6 cases in which I have used bypass, but there was no difficulty in any of them in starting normal action as soon as the aortic clamp was removed and normal coronary perfusion restored. Unless or until I encounter difficulty with this I do not intend to enter upon the complications of artificial coronary perfusion for periods of half an hour. For longer operations there may be more need for it.

There has been one post-operative death in 6 open cases under bypass.

I have also stated that in some cases of congenital subvalvar stenosis a pre-operative pressure withdrawal record may fail to exclude a valvar stenosis. Also in some severely ill patients we have decided against a pre-operative withdrawal record. Actually today, with our greater experience of this technique, it is doubtful if the examination should ever be omitted. If, however, doubt remains and it is felt that an open operation under bypass, with all that it implies, is to be avoided if possible, a simple policy can be used. For such cases I make a smaller anterior incision along the line of the left sixth rib and expose the left ventricle low down so that a catheter can be introduced easily at the apex to give a straight run along the line of the axis of the outflow tract for a pressure withdrawal record. If this now shows a subvalvar stenosis I insert an expanding dilator and split the fibrous stricture

open. This quick operation is well tolerated and sometimes gives an excellent result with substantial obliteration of the gradient, I am, as yet, unconvinced that a great deal more can be achieved by means of an open operation from above. Access is so restricted that a full and adequate resection of the subvalvar stricture is very difficult. Although surgeons state they can do this, the evidence is as yet slight and the reports in the literature of actual pressure changes are scanty and include no large series. We must await further information before we can decide this point and in the meantime I intend to continue using transventricular dilatation except when a subvalvar stenosis is unwittingly exposed from above.

I must point out that in these cases an element of functional obstruction secondary to left ventricular hypertrophy is a superadded phenomenon, just as occurs in the right ventricle in many cases of pulmonary stenosis. In fact in severe cases it may be impossible to relieve the stenosis substantially either by an open or by a closed procedure. In this event we must await the natural process of recession of the muscular hypertrophy, by which means the high intraventricular pressure will gradually drop to a normal or near-normal figure. It is quite certain that this can occur in the left ventricle for it has been observed and recorded, although as yet not so often as in the right ventricle.

I would mention that congenital subvalvar stenosis is, in my experience, usually a very severe condition and I always think of it as being likely to be present when a young patient has a very severe stenosis.

Calcific Aortic Valvar Stenosis

When we come to calcific aortic valvar stenosis the problem of the best type of operation is more complex. In patients under the age of 45-50 years there is obviously a good case for an open operation, especially when the valve is apparently not heavily calcified. I think one must accept the desirability of an open operation in these patients.

In practice I have found that so many patients in the thirties or forties have such severe features of the disease when they come for operation, often having been on the verge of left ventricular failure, that one fears the greater burden of an open operation and still turns to a quick closed transventricular dilatation, which has given such a high proportion of good results.

Open operation under hypothermia now holds no attractions for me in these patients, I have used it in 6 cases with 3 survivals. The short time at one's disposal limits the time available for safe suture of the aortic incision, and I consider this an important danger in these cases. Even more important is the danger of irreversible ventricular fibrillation, and this was the usual mode of death in my 3 fatal cases. The coronary perfusion provided by the pump oxygenator seems to be the best means of supporting the strained left ventricle.

For the same reasons I am unattracted by the quick in-and-out open operation done under fluothane anaesthesia, which gives even less time than hypothermia.

For patients in the fifties and early sixties (operation is rarely indicated later than the early sixties) I am unattracted by open operation under bypass and unhesitatingly perform a closed transventricular dilatation. This procedure carries a low mortality, as is shown by the fact that in my last consecutive 100 cases there were only 7 deaths. The results are good in some 70% and in many the improvement is dramatic. They include many patients in the most advanced stages of aortic stenosis. The number of relapses in which a second valvotomy is needed has been small, some 5 or 6 patients only. I have in fact done a second operation in only one patient, a man aged 35, and he succumbed to an open procedure under hypothermia.

The incidence of regurgitation, either inflicted or aggravated, has been small, doubtless due to the rigidity of the calcified valve structure. In passing, one must decry the fears so often expressed that heavy calcification is a contra-indication to operation. This is just not so, for many very good results have been obtained in its presence. As a balance to the few cases which have suffered some regurgitation, there are those in whom pre-operative regurgitation has been lessened or virtually obliterated as a result of the improved mobility of the cusps secured.

I hold no brief at all for the various closed transaortic procedures by which instrumental or finger dilatation is used blindly from above. I am totally unconvinced of their supposed advantages and am unattracted by the extra technical hazards involved. The transventricular operation gained disrepute when it was done with a large, clumsy and dangerous type of instrument. If a simple two-bladed dilator is used, only the thickness of an ordinary lead pencil, it causes no damage to the ventricle and no controlling purse-string stitch is needed. It can be slipped in easily and the whole procedure takes barely 60 seconds. During this short time the brain is protected from calcific emboli by temporary occlusion of the head and neck aortic branches. Simple finger pressure controls the small incision while 2 or 3 stitches are inserted.

While continuing to use this simple, quick procedure for most cases I am now gradually building up my own experience with open valvotomy under bypass. Clearly it is essential to prosecute this open method as much as possible until we know how much extra risk it carries. At present the evidence that exists seems to show that it in fact carries a substantial extra mortality. It is difficult to secure precise figures in long enough series to form a final opinion. It would seem that in the best hands the open operation under bypass carries a mortality of some 25% and in average hands the mortality is about twice this.

It is inevitable that one is influenced by this high risk when faced with the individual patient and when one knows that a simple closed operation carries a risk of less than 10%. This would seem to compel caution, at present, in selecting cases for the more severe, even if more exact, open procedure.

It will be observed from my composite figures, given above, of open operations under hypothermia and under bypass, that the total is 22 with 7 deaths; these include both calcified and non-calcified cases.

SUMMARY

The subject is treated under the 3 headings of (1) indications for operation, (2) diagnostic features, and (3) types of operation.

Under (1) stress is laid on the danger of withholding or delaying operation in suitable cases, and on the fact that in such cases the mortality rate is far higher if operation is withheld.

Under (2) reference is made to the diagnostic value of ECG and of pressure gradient. Left ventricular puncture combined with estimation of cardiac output is invaluable. The importance of the differentiation of valvar from subvalvar stenosis is mentioned, and particularly of the newly recognized 'functional aortic subvalvar stenosis'. The diagnosis of the latter condition is discussed and the danger of cardiotomy if it is present. Stress is laid on the importance of demonstrating calcification in excluding this condition.

Under (3) the place of the closed transventricular operation and of the open operation is discussed, and the value of the heart-lung bypass in aortic stenosis. The open operation is preferred in non-calcified aortic valvar stenosis, but in the calcific condition the problem of the best type of operation is more complex and the author discusses the relative merits of the different available procedures in various types of case.

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