BRITISH MEDICAL JOURNAL

LONDON SATURDAY NOVEMBER 14 1953

VALVOTOMY IN THE TREATMENT OF MITRAL STENOSIS

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The selection of patients who need surgical treatment for mitral stenosis is a problem which is no longer confined to special centres for cardiac surgery. If patients who are in greatest need of surgical treatment, and who are most likely to benefit from it, are to be given priority, selection must start with the practitioner and in the general medical clinic.

The advent of valvotomy as a method of treatment has stimulated an intensive investigation of the pathological physiology of mitral stenosis by means of cardiac catheterization and related technical procedures. This has led in turn to a renewed interest in the interpretation of clinical signs and symptoms. Many physical signs which have been a source of speculation since the early days of auscultation can now be more precisely interpreted in the light of surgical experience and of recent haemodynamic research. Many of the patients in the present series, and a great many others judged unsuitable for operation, have been investigated by cardiac catheterization, and, at the start, the information so obtained often fortified the decision for or against operation. But with increasing experience we are now seldom influenced by catheter findings in selecting favourable cases for surgery. Admittedly, in extending the scope of surgery to less favourable cases, full investigation is still desirable.

The low mortality and good results of mitral valvotomy are already well known, and it is with the indications for operation that we are now mainly concerned. In this respect we were guided from the start by the pioneer work at Guy's Hospital (Baker, Brock, and Campbell, 1950) and in the United States (Harken *et al.*, 1948; Bailey, 1949), but our present views have been moulded largely by personal experience.

SELECTION OF CASES FOR VALVOTOMY

Basic Indications.—The aim of surgical treatment of mitral stenosis is to remove or reduce valvular obstruction without thereby creating serious valvular incompetence. Provided the fused mitral cusps are split or cut along the line of the commissures, as advocated by Brock, the degree of incompetence need not be increased, and may even be diminished, by valvotomy. The basic

indications for mitral valvotomy may therefore be simply stated as follows: (1) Severe stenosis of the mitral orifice, the anatomical conformation of which must be suitable for surgical correction. (2) The patient's symptoms must be disabling enough to warrant the risks of surgery. (3) We must be satisfied that the symptoms are entirely or mainly due to mitral obstruction, and not to myocardial or other valvular lesions, or to extra-cardiac causes.

Degree of Mitral Stenosis and its Anatomy

If mitral stenosis were a purely mechanical constriction of the valvular orifice, of unvarying anatomical form, and superimposed on an otherwise healthy heart, it would be a relatively simple matter to assess its degree, but in practice the problem is far more complex. We have to take account of the anatomical form of the valvular structures, of secondary changes in the lung vessels, and of complicating factors such as myocardial damage, auricular fibrillation, and associated aortic and tricuspid valve lesions. Nevertheless, in many cases the rheumatic damage falls mainly on the mitral valve cusps.

The ideal type of stenosis for surgical correction is the *diaphragmatic valve* well described by Balfour (1898) as follows: "The condition of the valve generally associated with the presystolic murmur is that usually termed a diaphragmatic valve in which the two segments of the mitral valve are united and stretched like a diaphragm across the auriculo-ventricular opening. This diaphragm, as we may call it, is thickened especially at the edges of the central opening. . . ." Apart from the thickened orifice, the mitral diaphragm as a whole may be relatively pliant, and it can be split so as to re-create mobile and supple valve flaps, and so that obstruction is relieved without incompetence resulting (Type I, Fig. 1). The diaphragmatic valve, which is formed mainly by the large antero-medial or aortic cusp, cannot open or close, but, if still pliant, it balloons like a sail towards the auricle in systole, and billows back towards the ventricle in diastole. This sudden tension of the mitral diaphragm, first in one direction and then in the other, gives rise to important physical signs, which are discussed below.

Sometimes the valve sheet as a whole is indurated and thickened so that ballooning is reduced and at the same time valvotomy made more difficult technically (Type II, Fig. 1). Most valves in this category cannot be completely divided, but an adequate result may be achieved if one commissure is split and the other only partly split. Here the extent of shortening or fibrosis of the chordae tendineae is greater than in Type I, and mobile cusps cannot be reconstructed.

More severe rheumatic damage may fuse the cusps, chordae, and papillary muscles into a rigid, fibrous, funnel-shaped structure directed towards the ventricle (Type III, Fig. 1). If cases for surgery are carefully selected, the surgeon seldom encounters this type of stenosis, which features so prominently in textbooks of pathology. Here little can be done by operation, yet digital dilatation has occasionally produced unexpected clinical improvement.

Deformed and retracted cusps producing a permanently gaping and incompetent orifice, even though some degree of stenosis may be present, are not amenable to surgical reconstruction. Graham Steell (1906) and Carey Coombs (1924) regarded mitral regurgitation as due mainly to dilatation of the left ventricle and of the mitral ring from myocardial damage during the active phase of rheumatic carditis, so that subsequent fibrosis of the cusps failed to constrict the enlarged orifice appreciably. Others regarded the left ventricular enlargement as the result rather than the cause of mitral regurgitation. Whichever view is taken, and both may be correct under different circumstances, the fact remains that left ventricular enlargement and mitral incompetence go together, and both are probably incompatible with a severe degree of mitral stenosis. An enlarged left ventricle should be regarded as evidence of myocardial damage, whether of inflammatory or of mechanical causation.

FIG. 1.—Types of mitral stenosis. The features of each type are described in the text.

Clinical Signs

The clinical signs of a *pliant diaphragmatic valve* are, first and foremost, the snapping first sound, and the

characteristic apex beat described by Broadbent (1897) as "tapping," an adjective originally applied to the first sound by Sansom (1892). Carey Coombs described the apex beat of mitral stenosis as follows: "The hand placed over the praecordium feels the apex bang suddenly and sharply against the chest wall, then sink away from it as suddenly as it arrived." These signs relate to a sudden tension of the pliant mitral diaphragm as it is ballooned upwards into the auricle in systole, and can be felt by the surgeon's finger. We may well speak of this sign as the "closing snap" of the mitral valve. So long as a snapping first sound is audible, and it is best heard with the patient lying and inclined toward his left side, serious mitral incompetence and a hopelessly rigid valve are unlikely, and a systolic murmur may be ignored.

Another important sign of a diaphragmatic valve is the opening snap, a sharp click which follows closely after the second sound and which is best heard over the mesocardium often internal to or above the apex beat. The opening snap, formerly described as a double second sound at the apex, was first recognized by Sansom as originating at the mitral valve, and the term "claquement d'ouverture" is generally attributed by French writers to Potain. It may be supposed that the opening snap is caused by sudden tension of the pliant mitral diaphragm in a reversed direction to that responsible for the snapping first sound—namely, towards the ventricle at the start of diastole. We believe that a loud opening snap of clicking quality is a reliable sign of mitral stenosis with a pliant diaphragmatic valve

and that it constitutes important evidence of operability. The opening snap is absent in predominant mitral regurgitation, and is uncommon or lacks a loud clicking quality in severe mitral calcification (Mounsey, 1953; Wynn, 1953).

In regard to murmurs, loud rumbling diastolic and pre-systolic murmurs completely filling diastole constitute reliable evidence of considerable mitral stenosis, though this does not necessarily imply an operable valve. The absence of an impressive mitral diastolic rumble does not exclude severe stenosis, and Levine and Love (1952) have drawn attention to the occasional absence of all diastolic murmurs in this condition. The intensity of the diastolic murmur depends not only on the degree of stenosis but also on the flow through the mitral orifice, and active pulmonary vasoconstriction acts as a barrier to mitral flow. In cases with

severe pulmonary hypertension the mitral diastolic murmur may be quite insignificant, though the opening snap tends to persist.

A mitral systolic murmur need not imply serious incompetence in the presence of a snapping first sound and an opening snap; it may be ignored unless there is evidence of left ventricular hypertrophy in the form of an apex beat with a localized and sustained thrust, the "*choc en dôme*" of Bard. If the first sound is diminished or replaced by a systolic murmur, and the left ventricle is enlarged, the case is probably unsuitable for valvotomy. The electrocardiogram and radiograph are valuable in identifying left ventricular hypertrophy. In evaluating left ventricular enlargement *vis-à-vis* the mitral valve, due regard must be given to the presence of aortic incompetence.

The pulse in severe mitral stenosis is of small volume and sustained in character, and, like the small aorta, is evidence of a restricted output from the left ventricle. It is especially small in cases with active pulmonary hypertension, when the cardiac output is further reduced by a second mechanical barrier in the lungs. In predominant mitral regurgitation the pulse has a more jerky character.

A small hypoplastic aorta is the rule in severe mitral stenosis developing before adult life, and the aortic knuckle is small in the radiograph, situated well below the clavicles, and sometimes invisible in the anterior view.

To sum up, we regard the following clinical signs of mitral stenosis as indicative of a pliant diaphragmatic valve amenable to valvotomy: a snapping apex beat; a snapping first sound (closing snap); and a loud opening snap of clicking quality. Additional evidence of severe mitral stenosis is provided by intense diastolic and presystolic murmurs; a small sustained pulse; and a small hypoplastic aorta in the radiograph.

Signs of Right Ventricular Enlargement

When the right ventricle enlarges, the infundibulum is first involved, causing a palpable systolic impulse on the left of the sternum, usually in the third interspace and well above the apex beat. A diastolic shock due to forcible closure of the pulmonary valves is also felt over the enlarged infundibulum.

The main cavity of the right ventricle enlarges towards the left, displacing the left ventricle posteriorly away from the chest wall. In pure mitral stenosis, therefore, an apex beat displaced to the left is evidence of right ventricular enlargement. The right ventricle seems to strike the chest broadside, so that the apex beat resembles a blow with the open hand, compared with a blow by the closed fist from the left ventricle, which strikes the chest head on. Instead of a localized thrust, the enlarged right ventricle causes a diffuse and less-sustained pulsation between sternum and apex beat which, in mitral stenosis, also has a snapping quality.

In radiographs, right ventricular enlargement gives the heart a square shape with blunted apex, compared with the pointed oval of left ventricular enlargement. Right heart enlargement may also be identified in the left oblique view.

Symptoms

Breathlessness is the earliest symptom in the majority of cases and its onset is usually gradual. Patients notice that they can no longer keep pace with their friends, carry their shopping, or perform their usual domestic duties without distress, and their disability is best assessed in terms of their capacity for these simple everyday tasks. Sometimes attention is first called to the heart by more dramatic symptoms of a characteristic kind.

In 1921, and more fully in 1934, Gallavardin described the occurrence of paroxysmal pulmonary oedema in certain cases of mitral stenosis, which he called "*le rétrécissement mitral œdémateux*," and this particular syndrome was also

described by Oppenheimer and Schwartz (1933) and by McGinn and White (1934). It is encountered especially in women aged 25 to 35 years with long-standing mitral stenosis. The paroxysms of dyspnoea are often provoked by effort or emotion, but may also occur spontaneously at night, and tend to be aggravated during the menstrual period, during pregnancy, and in cold weather. Gallavardin noted that in such patients the heart was little if at all enlarged, the rhythm was regular, and digitalis was ineffective in controlling the breathlessness.

Careful interrogation is necessary to identify minor paroxysms provoked by effort; once dyspnoea attains a certain degree the following symptoms appear—oppression in the chest, severe palpitation, tracheal bubbling and wheezing, cough, and blood-stained expectoration. These symptoms subside gradually with rest. Spontaneous or nocturnal attacks tend to be more severe, may be accompanied by circulatory collapse, and may prove mortal. Sooner or later these patients become orthopnoeic, require extra pillows for sleep, and exhibit some degree of persistent pulmonary congestion. The patient with "oedematous mitral stenosis" is severely incapacitated and offers a marked contrast to the patient with a huge heart, auricular fibrillation, and congestive failure reasonably controlled by digitalis, who may lead a relatively comfortable though restricted life.

Paroxysmal pulmonary oedema is explained by a sudden increase in pulmonary capillary pressure above the level of the colloid osmotic pressure of the plasma. Opposed by severe mitral obstruction, any increase in the output of the right ventricle—for example, on exertion—must elevate the pressure in the pulmonary veins and capillaries. In the past it was difficult to explain why pulmonary oedema should occur in certain cases of severe mitral stenosis and not in others, often with far larger hearts; the probable explanation is that in some cases the pulmonary arterioles are constricted, thus limiting the output of the right ventricle and so protecting the lung capillaries from any sudden overdistension. In mitral stenosis with pulmonary oedema, pulmonary hypertension is a purely passive process, due to a rise in pressure behind the obstructed mitral valve, and the absence of cardiac enlargement and of auricular fibrillation indicates that the myocardium is relatively intact. This combination of severe mechanical obstruction and a sound myocardium represents the ideal condition for surgical intervention.

Classification of Cases for Surgical Purposes

For the purpose of deciding suitability for valvotomy and for assessing its results, a classification based purely on symptoms or the degree of incapacity is unsatisfactory. A patient with symptoms of great severity may be most eligible for surgical relief, whereas another far less troubled may be entirely beyond it.

The pulmonary arterial pressure at a given moment is not a reliable basis for deciding on surgical treatment unless it is interpreted in relation to the clinical signs and symptoms, and a complete haemodynamic investigation is not always practicable even in expert hands. We believe that, eventually, suitability for valvotomy will usually be decided on the basis of routine cardiological examination rather than on catheter findings. The primary indication for valvotomy is severe mitral obstruction with an operable valve, and in this respect the clinical signs of a pliant diaphragmatic valve are significant. Next in importance is the size of the right ventricle. If tricuspid disease is excluded, right ventricular enlargement provides the best measure of the degree of permanent damage resulting from mitral stenosis, and thus it is the best guide in regard to the prospect of surgical relief. At the Middlesex, having selected the cases with operable valves, we graded them for surgical purposes into three classes, identifiable by routine cardiological examination:—Class I: cases without or with minimal signs of right ventricular enlargement; class II: cases with con-

FIG. 2.—Mitral stenosis with clinical signs of slight right ventricular enlargement (class I). The patient, a woman aged 36, in sinus rhythm, had several attacks of pulmonary oedema. Valvotomy was performed with a good symptomatic result (Case 55). FIG. 3.—Mitral stenosis with clinical and electrocardiographic evidence of right ventricular enlargement (class II). The pulmonary artery and its hilar branches were dilated. Mean pulmonary arterial pressure was 54 mm. Hg. The patient, a woman aged 28, was considerably restricted by breathlessness. A good result followed valvotomy. FIG. 4.—Mitral stenosis with considerable cardiac enlargement (class III). Cardio-thoracic ratio 68%. Mean pulmonary arterial pressure was 48 mm. Hg. The patient, a woman aged 48 with auricular fibrillation, had recurrent attacks of congestive failure which improved with treatment.

siderable right ventricular enlargement, but without congestive failure; class III: cases with gross right ventricular enlargement and congestive failure.

Class I.—Regular rhythm was more common than auricular fibrillation, the pattern of right ventricular hypertrophy was seldom present in the electrocardiogram, and radiography showed a mitral configuration without much increase in transverse diameter of the heart. There was often perihilar congestion, but without great dilatation of the pulmonary artery (Fig. 2). The mean cardio-thoracic ratio in this group was 51%. Paroxysmal pulmonary oedema was common. The mean pulmonary arterial pressure averaged 35 mm. Hg.

Class II.—Right ventricular enlargement was evident clinically, radiologically, and in the electrocardiogram. Normal rhythm was more common than auricular fibrillation, and paroxysmal pulmonary oedema was less frequent than in class I. In some cases, episodes of congestive failure occurred, due to the onset of fibrillation or to pulmonary infarction, but failure was not persistent. Dilatation of the pulmonary artery and its hilar branches was evident in radiographs, but perihilar oedema was often absent (Fig. 3). The mean cardio-thoracic ratio was 55%. The mean pulmonary arterial pressure averaged over 50 mm. Hg, indicating active pulmonary hypertension. These patients have severe mitral obstruction with some myocardial damage.

Class III.—The heart was considerably enlarged, the cardio-thoracic ratio exceeding 65% (Fig. 4). Aortic valve

disease was present in some cases, and often tricuspid incompetence. Auricular fibrillation was invariable, and congestive failure was recurrent or could not be completely relieved by treatment. The liver was permanently enlarged and presumably damaged. Patients in this class posed the problem of whether the mitral obstruction played a sufficiently important part in the overall picture to justify mitral valvotomy. If the signs of a pliant diaphragmatic valve were present it was thought that some measure of surgical relief was at least possible.

Table I presents an analysis of the first 72 cases operated on at the Middlesex Hospital, classified as described.

Results

Mitral valvotomy has been performed on 150 patients—87 at the Middlesex Hospital and 63 at Harefield Hospital and the London Chest Hospital. There were 31 men and 119 women, a ratio of 1 to 4. The youngest patient was 19 and the oldest 57; 78% were between 30 and 49. The heart rhythm was regular in 88 patients (59%), and auricular fibrillation was present in the remaining 62 (41%). The incidence of certain other clinical features is mentioned below.

Four of the 150 patients operated on died as a direct result of the operation—that is, within four days—an immediate operative mortality of 2.7%. Four others died after an interval of one month or longer. Details of these fatalities are given later.

TABLE I.—Mitral Valvotomy: Analysis of the First 72 Cases Operated on at the Middlesex Hospital. Results According to Classification

Class	No. of Cases	Duration of Symptoms (Years)	Orthopnoea	Paroxysmal Dyspnoea	Haemoptysis	Pulsus Parvus	R.V. Pulsation	Pulmonary Diastolic Shock	Auricular Fibrillation	E.C.G. Signs of R.V. Enlargement	Pul. Arterial Pressure* (mm. Hg.)	Pul. Capillary Pressure* (mm. Hg.)	Cardio-thoracic Ratio (%)	Results†	
														Good	Fair or Poor
I	33	6	21 (64%)	20 (61%)	25 (76%)	9 (27%)	23 (70%)	14 (42%)	10 (31%)	0 (0%)	35	20	51	73%	23% 1 late death
II	30	7	17 (57%)	9 (30%)	20 (67%)	14 (47%)	25 (83%)	22 (73%)	6 (20%)	30 (100%)	54	25	55	69%	19% 1 operative death, 1 late death
III	9	12	8 (89%)	6 (67%)	4 (44%)	1 (11%)	5 (56%)	2 (22%)	8 (89%)	1 (11%)	28 } 55 }	19 } 22 }	63	40%	20% 2 operative deaths
Total	72		46 (64%)	35 (49%)	49 (68%)	24 (33%)	53 (74%)	38 (53%)	24 (33%)	31 (43%)					

* In class III there were two results only.

† Results apply to 61 cases adequately followed up.

One hundred and eleven patients have been followed up for periods of five months to two and a half years. The results are given in Table II. The remaining 39 have been

TABLE II.—*Mitral Valvotomy: Results*

Result in 111 cases:			
Good	81	(74%)	
Fair	9	(20%)	
Poor	13		
Too early for assessment	39		
Operative deaths	4	(2.7% of whole group)	
Later deaths	4		
150			

operated on within the past three months. All have made satisfactory progress to date, but it is too early to assess the results.

In each case the result was assessed in relation to the pre-operative disability. A good result indicates relief from symptoms such as paroxysmal dyspnoea, orthopnoea, and haemoptysis, and a considerably increased capacity for effort. For example, a more or less bedridden patient is able to sleep flat and to get about in comfort; an ambulant patient whose activities had been much restricted is able to lead a reasonably normal life or to return to work. A fair result implies an obvious and worth-while relief of disability, but short of the optimum improvement. A poor result means slight, doubtful, or no benefit.

Results in Relation to Classification of Cases.—The number of patients in each class is too small to allow of any far-reaching conclusions, but the results indicate a trend. Provided that clinical signs of a pliant diaphragmatic valve are present, the outlook after operation differs little in classes I and II, though in class II the right ventricle is larger and the pulmonary arterial pressure is correspondingly higher. Those in class III have the largest hearts and there is usually a history of recurrent congestive failure; they fare less well and carry a higher operative risk than cases in classes I and II.

SPECIAL FEATURES

Pulmonary Hypertension

The pulmonary arterial pressure by itself does not provide an indication of the degree of mitral stenosis. In cases with paroxysmal pulmonary oedema the mean pulmonary arterial pressure at rest may be little raised, and again in the later stages of mitral disease, when auricular fibrillation and failure have supervened, it need not be greatly elevated. On the other hand, the size of the right ventricle reflects the cumulative effect of pulmonary hypertension on the heart, and is more informative than the level of the pulmonary arterial pressure at a given moment.

The factors responsible for pulmonary hypertension complicating mitral stenosis are beyond the scope of this paper. We were concerned, however, in observing whether pulmonary hypertension influenced the result of mitral valvotomy. The good and poor results in classes I and II were of much the same order, though the degree of pulmonary hypertension was far higher in class II. The outcome of operation was equally favourable in the six patients with

the highest pulmonary arterial pressures and greatly increased pulmonary vascular resistance, and in the six with the lowest pressures recorded in this series who had only slightly raised pulmonary resistance (Table III).

Pulmonary arterial pressure has been measured approximately one year after operation in two patients (Nos. 9 and 1) included in Table III. In both, the mean pressure had fallen, the cardiac output increased, and the total pulmonary vascular resistance lowered by 50% and 60% respectively of their original values. The final figures were still well above normal, yet symptomatic improvement was satisfactory. Similar findings have been reported by other authors (Holling, 1952; Baker, Brock, Campbell, and Wood, 1952).

In mitral stenosis with gross right ventricular enlargement other factors than pulmonary hypertension have to be considered—namely, rheumatic myocardial damage, the effects of auricular fibrillation, and the possibility of associated tricuspid valvular disease.

Rheumatic Myocardial Damage

The relative importance of a defective valve and of rheumatic myocardial damage in the causation of cardiac enlargement and failure is an old-standing problem. Experience of the results of valvotomy has proved that the valvular factor is often far more important than Mackenzie and others believed and taught.

Nevertheless, a grossly enlarged right ventricle implies a damaged myocardium, and whether the damage is rheumatic or whether it is due purely to mechanical stress, it is unlikely to be reversible. The degree of benefit to be expected from valvotomy when the myocardium is permanently damaged is always problematical and the operative risks must be increased. Provided that tricuspid disease is excluded and there are signs of severe mitral stenosis with an operable valve, surgery need not be contraindicated if the age and other circumstances are favourable.

Auricular Fibrillation

So long as the operation was indicated on other grounds, we have disregarded auricular fibrillation entirely in selecting our cases. Indeed, fibrillation offers the advantage of a heart rate controlled by digitalis, whereas in normal rhythm tachycardia may be uncontrollable. In so far as fibrillation is commoner in the advanced than in the early stages of the disease, so those with fibrillation are more often unsuitable for valvotomy than those with regular rhythm. For the same reason the operative mortality would be expected to be higher amongst the fibrillators. Of the entire series of 150 patients, 41% had auricular fibrillation, including three of the four patients who died at operation or within four days of it. Four fatalities occurred later; one of these was fibrillating at the time of operation and three were in sinus rhythm. Otherwise the functional results were as good in those with fibrillation as in those with sinus rhythm.

During the actual valvotomy, bizarre auricular and ventricular arrhythmias were recorded as often in sinus rhythm as in auricular fibrillation.

Auricular fibrillation developed within one to three days of operation in 13 out of 48 patients in sinus rhythm who were operated on at the Middlesex Hospital. In anticipation of this, pre-operative digitalization was carried out as a routine in all cases irrespective of rhythm. In three instances the arrhythmia was related to a small area of pulmonary atelectasis, and sinus rhythm returned promptly when the lung re-expanded. A fourth patient reverted spontaneously before quinidine was given. In seven of the remaining nine cases quinidine restored sinus rhythm, usually after 10 to 15 gr. (0.65 to 1 g.) by mouth; the largest daily dose needed was 30 gr. (2 g.). Post-operative auricular fibrillation was irreversible in two patients. In addition, we have encountered one instance of established auricular fibrillation initiated by cardiac catheterization. Heparin

TABLE III.—*Mitral Valvotomy: Results in Patients with Highest and Lowest Pulmonary Arterial Pressures*

Series No. of Patient	Mean Pressure (mm. Hg)		Resistance (Dynes/sec./cm. ⁻⁵)		Result
	Pul. Art.	Pu. Capill.	Total Pul.	Pul. Arteriole	
9	80	—	2,000	—	Good
1	73	—	2,336	—	"
34	65	30	1,304	704	"
5	64	25	—	—	"
53	60	32	—	—	"
18	59	—	2,048	—	"
21	32	21	640	216	"
19	29	—	208	—	"
44	23	19	376	656	"
32	22	18	336	92	"
51	20	12	664	264	"
26	17	8	416	216	"

was given before and during quinidine therapy in two cases, but it is not used as a routine. There has been no instance of embolism related to quinidine therapy. Attempts to abolish post-operative fibrillation with quinidine during the first 10 days after operation were unsatisfactory and resulted in sinus rhythm vacillating with fibrillation. Quinidine is more likely to restore permanent sinus rhythm after the fourteenth day. When auricular fibrillation was established before operation we did not attempt to abolish it, and we have not encountered spontaneous reversion to normal rhythm in such cases.

Embolism

There was a history of embolism in 14 (9%) of the entire series of 150 cases. At operation a thrombus in the left auricle was encountered in only one of these cases (No. 22—auricular fibrillation); an old adherent clot was dissected away from the appendix with some difficulty. The patient died 12 hours after operation from cerebral embolism. In another case a fatal pulmonary embolism occurred one month after operation.

Post-operative embolism occurred altogether in nine patients; the details are set down in Table IV. Left auricular thrombi were noted at operation in 14 cases; in 11 of these the thrombus was in the appendix and in three in the body of the auricle.

TABLE IV.—*Mitral Valvotomy: Details of Post-operative Emboli in Nine Patients*

Case No.	History of Previous Embolus	Clot in Left Auricle	Site of Post-op. Embolus	Rhythm	Interval after Op.	Clinical Course
17	Yes	No	Pulmonary (multiple)	Recent auric. fib.	1 month	Died
22	"	Yes—in appendix	Cerebral	Auric. fib.	During operation	Died 12 hours later
42	No	No	Pulmonary (multiple)	" "	2 months	Died 5 months after op.
J.O. 46	"	?	"	" "	1 month	Died
60	"	No	Cerebral	Sinus	During operation	"
58	"	"	Cerebral popliteal	Auric. fib.	"	Permanent hemiplegia
62	"	"	Cerebral	Sinus	"	Recovery
87	"	"	Renal	Sinus	3 days	"
	"	"	Cerebral	Auric. fib.	During operation	Permanent hemiplegia

Two patients (Cases 17 and 42) died some months after operation from multiple pulmonary emboli under circumstances which deserve comment. In both patients the operation was technically satisfactory and a good result was anticipated on discharge from hospital. One and three months respectively after operation, during unusually severe foggy weather, they developed bronchitis and rapid auricular fibrillation, complicated by phlebothrombosis of the legs and repeated pulmonary emboli; both developed intractable cardiac failure. The third patient encountered a similar train of misfortunes one month after operation. Death in these three cases followed massive pulmonary embolism. Since this experience we have taken special care to warn the practitioner of the dangers of bronchitis complicating the early post-operative period and of the importance of detecting and treating phlebothrombosis in such patients.

Congestive Cardiac Failure

Congestive (right ventricular) failure had been present at one time or another in 29 of the complete series of 150 patients. Operation was undertaken in these cases only after long deliberation, since factors other than mitral stenosis, such as myocardial damage, might have contributed to the failure to a predominant degree. The majority of them responded favourably to medical treatment, but in a minority (class III) failure was recurrent or a state of borderline failure persisted. The deciding factors in favour of valvotomy were unquestionable signs of a diaphragmatic valve.

The Middlesex Hospital series of 87 cases included 19 with a history of right ventricular failure in whom valvotomy was deemed worth while. In eight of them failure had been related to the onset of auricular fibrillation or an intercurrent lung infection. When the exciting cause was treated the signs of failure disappeared and the heart became smaller, so that these patients were then included in classes I or II. The remaining 11 belonged to class III.

Three of the 19 patients died, one at operation from a tear of the auricular wall and two within a few days of operation. The result of valvotomy was good in four patients and poor in three. The remaining nine have been operated on within the past few months; all have survived, but the interval is too short to allow evaluation of the results. In a few cases the venous pressure increased and oedema developed within 10 days of operation. No cause was discovered, and the signs of failure disappeared spontaneously.

Calcification of the Mitral Valve

A grossly calcified mitral valve is likely to be rigid and is often incompetent. Yet the signs of a pliant diaphragmatic valve may exist with calcification which stands out conspicuously on radiology. Calcification was obvious in 9 of the 87 patients treated at the Middlesex Hospital. Small flecks of calcium perceived only after long scrutiny were disregarded. In one instance of gross calcification it appeared that valvotomy would be impossible; yet, because the signs of an operable valve were present, operation was proceeded with. Massive calcification involved the valve ring and invaded the cusps, which, however, were mobile. The commissures were split with ease and the functional result has been gratifying.

In addition to the nine instances of calcification discovered by radiology, a further 18 of varying degree were noted at operation, making a total of 27, or 31% of the Middlesex Hospital series. So far calcification by itself has not prevented operation, although often a knife had to be used to split the commissures. Emboli occurred in two of these cases; in one, transient hemiplegia developed after successful splitting of a slightly calcified valve; in the other it followed unsatisfactory widening of a heavily calcified valve.

Associated Valvular Disease

Mitral Incompetence.—Free mitral regurgitation is almost always a contraindication to valvotomy, but we believe that serious incompetence is incompatible with severe stenosis presenting signs of a pliant diaphragmatic valve. In such cases a systolic murmur may be ignored unless there is left ventricular enlargement. Expansile pulsation of the left auricle visible in the anterior view on radiology, and evident on both right and left heart borders, should be taken as evidence of significant mitral incompetence. We have seldom found the diagnosis of mitral incompetence a difficult problem except in cases with gross right ventricular enlargement as already discussed.

Aortic Valve Disease.—In the presence of signs of tight mitral stenosis, we have ignored slight and occasionally moderate aortic valve disease. One-third of the Middlesex Hospital cases had aortic incompetence and three of them also had aortic stenosis. However, mitral stenosis was always the dominant lesion. Free aortic incompetence with a thrusting apex beat, a jerky pulse, and left ventricular enlargement contraindicates mitral valvotomy. The result has been good in 15 of the 24 patients with slight aortic incompetence and fair in seven; two have died.

Tricuspid Valve Disease.—The possibility of organic tricuspid disease must always be considered when the right ventricle is greatly enlarged. It should be suspected when the signs of tricuspid regurgitation occur in the absence of congestive failure, when there is a noticeable tricuspid systolic murmur or thrill, and when the right auricle is unduly large. Tricuspid stenosis may sometimes be demonstrated by cardiac catheterization. The absence of pulmon-

any congestion and relative freedom from dyspnoea and orthopnoea, in the presence of gross right-sided cardiac enlargement, especially if associated with liver swelling and ascites, is characteristic of tricuspid disease. We have regarded this condition as a contraindication to valvotomy. One patient with proved tricuspid stenosis and incompetence was submitted to mitral valvotomy on grounds of her hopeless disability and at her express wish, but unfortunately she died from post-operative embolism, so that the experiment was inconclusive. Functional tricuspid incompetence which disappears when failure responds to treatment is no bar to operation.

Pulmonary Complications

When mitral stenosis is complicated by chronic bronchitis and emphysema it is difficult to decide whether the symptoms are mainly cardiac or pulmonary, and long and careful observation may be necessary. We have performed mitral valvotomy on nine patients with well-marked chronic bronchitis and emphysema; the result was good in two and fair or poor in four. Two died and one case has not been followed up. Two out of three patients with extensive pulmonary haemosiderosis have done well after operation, and the radiological appearance of the lungs has shown a tendency to improve.

Pregnancy

Valvotomy was successfully performed during pregnancy in two patients who developed repetitive paroxysms of pulmonary oedema starting at the third and fourth months. The operation was done in one case at the fourth month, and in the other at the 32nd week, shortly after the patient first came under our care. Great improvement resulted and pregnancy continued normally to term.

Acute pulmonary oedema complicating mitral stenosis is the most important cardiac cause of death actually during pregnancy, and when it occurs after the third month a situation of the utmost gravity arises. Digitalis and other medical measures are unlikely to prevent a recurrence of the attacks if pregnancy continues, so that the choice lies between mitral valvotomy and termination of pregnancy by abdominal hysterotomy. Valvotomy has the double advantage of affording immediate relief to the underlying cause of the pulmonary oedema and of a good chance of preserving the life of the child. Late in pregnancy, however, it must carry a risk of precipitating premature labour in the post-operative period.

We have little doubt that immediate valvotomy is the method of choice in dealing with the emergency which arises when a patient with mitral stenosis develops pulmonary oedema after the third month of pregnancy. This does not imply that, in general, valvotomy renders those with severe mitral stenosis capable of undergoing pregnancy with safety, or that we should encourage such a belief in these patients.

Contraindications to Mitral Valvotomy

The current contraindications, in summary, are as follows:

Age.—Few patients under the age of 20 present with the indications for valvotomy. Because of the danger of activating the rheumatic state, operation should be postponed in these cases unless the symptoms compel immediate surgical relief. We have generally regarded 50 as the upper limit, although six of our patients had passed this age.

Acute Rheumatism.—We have never attempted valvotomy if clinical evidence of acute carditis was present.

Associated Valve Disease.—Predominant aortic valve disease, free mitral incompetence with left ventricular enlargement, and organic tricuspid disease are contraindications to valvotomy.

Myocardial Damage.—This is assessed from the size of the heart and the presence or absence of congestive failure. Gross right ventricular enlargement is a relative contraindication and gross left ventricular enlargement an absolute contraindication to valvotomy. One of our patients who had a coronary occlusion and left bundle-branch block in the electrocardiogram was nevertheless operated on successfully and with benefit.

Pulmonary Disease.—Severe emphysema, chronic bronchitis, and bronchiectasis have proved to be unfavourable.

Congestive Cardiac Failure.—If right ventricular failure cannot be relieved by treatment, mitral valvotomy is unlikely to be successful.

Giant Left Auricle.—Aneurysmal dilatation of the left auricle is often associated with mitral incompetence and with pericardial adhesions. In spite of the huge size of the heart, these patients may remain free from congestive failure for years on digitalis, and their disability is often far less than in cases of severe mitral stenosis with smaller hearts. Valvotomy is contraindicated in such cases.

Anaesthesia for Mitral Valvotomy

Non-explosive agents have been used throughout. Induction and intubation are performed with minimal doses of thiopentone and curare, after which narcosis is maintained by endotracheal gas-oxygen. Approximately 1 g. of procaine hydrochloride in saline is given over the period of an hour by intravenous drip. Topical local analgesic agents are not used. Further small doses of barbiturate and curare are added as required.

Cardiac irregularities of serious import were uncommon and demanded urgent attention on only two occasions. Continuous electrocardiograms, which were taken in the first 30 cases, are now recorded only for special reasons.

Technique of Mitral Valvotomy

The postero-lateral fifth-rib incision is preferred to the antero-lateral, the patient being tilted somewhat towards the supine position. The pericardium is freely opened parallel to the phrenic nerve to expose the lateral aspect of the heart.

Direct auscultation of the heart is carried out with a sterile stethoscope. The auricle and its appendix are gently palpated for thrombi, and if these are absent a light clamp is placed across the base of the appendix. Then, without using any sutures, the tip of the appendix is cut off and the incision enlarged to leave an opening one and a half times the diameter of the surgeon's index finger.

As the clamp is removed the finger is inserted gently. Sometimes the isthmus between the appendix and body of the auricle is unduly narrow and has to be stretched gradually. With the finger now well in the auricle, the tip can be swept over the aortic cusp and carried laterally until the roughened edges of the stenosis are felt. More posteriorly and laterally the wide mouth of the pulmonary vein can be identified. Attention is paid to the following points: ballooning of the antero-medial mitral cusp in ventricular systole, the size of the stenotic opening, the presence of calcification, and the existence of a regurgitant jet.

The actual valvotomy has often been described; it consists in engaging the side of the finger-tip in the opening and trying to fracture the commissures antero-laterally and postero-medially as far as the valve ring, a structure whose presence is inferred rather than felt (Fig. 5).

With the diaphragmatic type of valve, the posterior commissure, which is usually shorter, should be ruptured first; if the anterior commissure is split first, the resulting loss of tension makes the posterior commissure more difficult to deal with. When both commissures have been split (and considerable force may have to be used) the valve cusps are palpated for mobility, the finger is passed down the fan of the chordae tendineae on to the papillary muscles to exclude any cross-fusion, and, finally, a regurgitant jet is again sought for.

The finger is then withdrawn as the auricular clamp is reapplied, and the incision sutured with fine thread. The heart is auscultated again. The pericardium is loosely closed after flushing out with saline, and the chest closed. Temporary water-seal drainage of the chest is maintained for 24 to 48 hours to avoid the effects of a sudden accumulation of pleural fluid.

Matters are not always so straightforward, and the following complications may be encountered:

Auricular Thrombosis.—The incidence of thrombosis in the left auricle or its appendix in this series was 9%. Great caution should be exercised if the auricle does not pulsate and clot is felt within it. The tip of the appendix can be incised and the resultant gush of blood may sweep out a recent clot. Organized clot may be dissected out of the appendix if a light clamp is applied to the base. The danger lies in separating a piece of organized clot as the clamp is applied. If the thrombus is firmly attached, the operating finger may be worked carefully around without disturbing it. A small or thrombosed appendix or a thrombosed auricle may preclude the normal entry, and, if so, access may be gained through the pulmonary vein. The distal branches are held in check ligatures while a clamp is placed at the base of the vein. An oblique incision over the main trunk allows the finger to be introduced, and at the com-

routine intravenous drip may be adequate for a moderate haemorrhage, but transfusion direct into the left ventricle or aorta is needed if a pint (570 ml.) or more is suddenly lost. This had to be undertaken on one occasion in our series.

Influence of Technical Result of Operation on Functional Result

An assessment has been made of the technical result of each valvotomy performed. When free splitting of the commissures was achieved without producing incompetence, 84% of the patients obtained a good functional result, compared with only 46% when the valvotomy was less complete. If the tiny slit of the stenosis is enlarged even by a few square millimetres, the volume of blood passing through the valve may be considerably increased. This may account for the early benefit experienced by certain patients in whom the technical result of the operation was poor in so far as the commissures were split to only a limited degree or a funnel-shaped valve could be merely dilated by the finger.

Summary and Conclusions

The basic indication for mitral valvotomy is disability from mitral stenosis of an anatomical form which is amenable to surgical correction. This type of stenosis, in which there is a *pliant diaphragmatic valve*, gives rise to characteristic clinical signs by which it may be recognized and which are described.

The most important consequence of pulmonary hypertension is right ventricular enlargement, and the size of the right ventricle, as determined by clinical, radiological, and electrocardiographic findings, provides a more valuable index to the degree of pathological change

due to mitral stenosis than does the level of the pulmonary arterial pressure at a given moment. Cardiac catheterization is no longer required as a routine measure in the selection of cases suitable for mitral valvotomy. Selection may now be made on a basis of the symptoms and of the data provided by routine cardiological examination.

Mitral valvotomy has been performed on 150 patients, of whom 31 were men and 119 women. Their ages ranged from 19 to 57 years. Four patients died as a direct result of the operation, an immediate mortality of 2.7%. Four died from various causes some time after the operation.

The results in 111 patients followed up for five months to two and a half years have been good in 74% and only fair or poor in 20%. Patients with auricular fibrillation (41% of the series) did as well as those in sinus rhythm, but three of the four operative fatalities were in fibrillation.

Cases selected for operation on grounds of an operable valve were classified according to the size of the right ventricle and the presence of congestive failure. Patients with large hearts, and congestive failure which failed to respond to treatment, benefited least from valvotomy. When free splitting of the commissures was achieved, 84% of the patients obtained good symptomatic relief, whereas when valvotomy was judged at operation to be less satisfactory only 46% of good results were obtained. Nevertheless, partial splitting of the valve does not preclude satisfactory clinical improvement.

FIG. 5.—Necropsy specimen of mitral stenosis after valvotomy with (left) assumed appearance before operation. The patient (J. O.) died one month after operation from multiple pulmonary emboli.

pletion of the operation simple suture is used to close the incised vein. In one case the auricular thrombosis was so extensive that it was impossible to introduce a finger or instrument into the heart. The pulmonary vein was too small, and direct incision through the auricular wall showed a thick layer of clot which could not be penetrated.

Embolism.—The separation of clot or calcified fragments may produce embolism in the cerebral vessels, but this is seldom recognized until the patient recovers consciousness. Where a thrombus is palpable in the auricle, the anaesthetist compresses the carotid arteries in the neck during the actual valvotomy. This temporarily arrests the flow into these vessels, and any emboli may be swept along with the active blood flow and avoid the brain. This method is more simple than temporary occlusion of the innominate and left carotid arteries within the thorax.

Tearing of the Auricle.—The incision in the appendix sometimes splits, particularly if the auricular wall is thin. This should be recognized before the finger is withdrawn, as it permits insertion of control sutures before the clamp is applied. The loss of blood from an unsuspected tear can be catastrophic.

Rigidity of the Stenosis.—The finger may fail to rupture the commissure despite the application of considerable force; if so, the greatest care must be exercised to avoid tearing into the aortic cusp or pulling off a papillary muscle. Either of these events may produce a severe regurgitation. If the finger fails, a knife-like instrument attached to the finger can be used, care being taken to avoid injury to surrounding structures.

Haemorrhage.—In a straightforward operation the loss of blood does not exceed 1–2 oz. (28–57 ml.). If much manipulation is required or if the auricular wall is torn, urgent measures are necessary to replace the blood loss. A

Good results were obtained alike in those with greatly elevated pulmonary arterial pressures and in those with a slight or moderate elevation.

The following are regarded as contraindications to operation: active rheumatic carditis, severe myocardial damage as revealed by gross right ventricular enlargement and intractable congestive failure, predominant aortic valve disease, organic tricuspid valve disease, mitral incompetence associated with left ventricular enlargement, and a giant left auricle. Chronic bronchitis, emphysema, and other pulmonary complications prejudice the success of the operation.

The technical details of mitral valvotomy and the operative complications are briefly described.

We wish to thank many colleagues in hospital and in practice, particularly Dr. K. Shirley Smith and others at the London Chest Hospital, who have kindly referred patients needing valvotomy. We are especially indebted to Mr. J. R. Belcher, who has taken an active part in the surgical management of the Middlesex Hospital patients.

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CHLORAMPHENICOL TREATMENT OF BRONCHIECTASIS IN CHILDREN

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Although most acute bacterial infections are now readily amenable to chemotherapy the problem of chronic infection based on structural changes remains unsolved. Whatever the prime cause or causes of bronchiectasis, the main symptoms in childhood result from chronic or recurrent infection of damaged lung. The damage is almost certainly permanent, yet follow-up studies suggest that the infection, as evidenced by clinical signs and symptoms, may die out towards adolescence. Cultures of sputum show the frequency of infection with *H. influenzae*, and, as this is an organism highly susceptible to chloramphenicol, a study has been made of the effect of this drug on the course of bronchiectasis. The study has been brought to an end by the recognition of the possible dangerous action of chloramphenicol on bone marrow. Sufficient knowledge had already been gathered to shed light on two problems—the nature of the infection, and the response, bacteriological and clinical, to chemotherapy; and these observations are reported here.

Clinical Material

Thirty-six cases of bronchiectasis in all, aged 3 to 15 years, have been treated with chloramphenicol. Judged by the quantity of sputum obtained while the patients were being treated by physiotherapy, 22 were severe and 14 were mild cases. Fifteen cases were able to produce an indefinite amount of sputum, 10 with involvement of at least four lobes. Among eight unilobar cases, only two are included in the severe group.

The first observations concern 15 cases, aged 5 to 10 years, in the Meath School of Recovery at Ottershaw (Invalid Children's Aid Association). In 11 the sputum grew *H. influenzae* alone, and in four *H. influenzae* with pneumococci. All had first been treated by a physiotherapist for many weeks, and eight of the cases could cough up a limitless quantity of purulent sputum at any time during the day. The dose of chloramphenicol was 500 mg. eight-hourly in capsules by mouth, equivalent to between 20 and 30 mg. per lb. (44 and 66 mg. per kg.) body weight daily. The quantity of sputum lessened dramatically, and by the third or fourth day of treatment it became difficult to obtain a specimen; cultures were sterile.* After seven days the drug was stopped in 10 and continued in five of the cases. The mildest case had no more sputum after this week of treatment and remained well and symptom-free for a whole year following discharge home. In the other nine cases sputum cultivated on the sixth day after the end of treatment showed a return of the original organism, while sputum from the five cases still under treatment remained sterile in four and grew pneumococci in one. Two cases continued treatment at half the original dose for two further weeks, the sputum remaining sterile, while two very severe cases, treated on half the original dose for five and six months, later increased the quantity of sputum to $\frac{1}{2}$ to 1 oz. (14 to 28 ml.) daily (compared with 6 to 8 oz. (170 to 230 ml.) collected easily before treatment) and the sputa grew bacteria from time to time, but never *H. influenzae*. In all the severe cases *H. influenzae* regained its ascendancy in the sputum within 10 days of the end of treatment, though cough and the quantity of sputum were less than before treatment began.

Three cases had 250 mg. of chloramphenicol twice a day for one, two, and five months (approximately 10 mg. per lb. (22 mg. per kg.) daily), and pneumococci, streptococci, and staphylococci appeared from time to time without *H. influenzae*. In seven cases three doses of 250 mg. eight-hourly for one day of each week were given for one month in one case, for three months in five cases, and for five months in one case (12 to 24 mg. per lb.: 26 to 53 mg. per kg.). Cultures of sputum were sterile on the day after treatment, but grew various bacteria, including *H. influenzae*, by the sixth day, so that weekly treatment failed to produce constant sterility of the sputum. Assessment of the 15 cases one year after starting treatment showed a maintained improvement in seven; two are somewhat better, and in the six clinically most severe cases lessening of the sputum occurred only while chloramphenicol was actually being given.

A second observation at the Meath Home concerned eight patients from 3 to 8 years old. Six sputa grew *H. influenzae* and one sputum grew *H. influenzae* with *N. catarrhalis*. In one case there was a pure growth of pneumococci, and this is the only case in the study from whose sputum *H. influenzae* was not grown. Six cases were given 250 mg. eight-hourly for 18 days (13 to 21 mg. per lb. (29 to 46 mg. per kg.) daily). On this dose, two-thirds of that given in the previous test, there was the same dramatic change in the quantity and character of the sputum from the third or fourth day, with the same return of organisms within a few days of the end of treatment. Sputum from

*This term is used for the sake of brevity to describe cultures containing no colonies of any pathogen and very few colonies of commensals.