for prompt treatment of sore throats or recurrences of limb or joint pains and on the importance of penicillin cover for dental extractions if heart lesions persist. If sulphonamide prophylaxis has been started in hospital it should be continued for at least another two years. Patients should attend after-care clinics at regular intervals. Our own are seen at the third, sixth and twelfth month after discharge and then once each year. These examinations are of value for the following reasons. First, to establish as far as possible whether there has been any fresh rheumatic activity; the occurrence of sore throats or limb and joint pains is noted; the haemoglobin, blood sedimentation rate and weight are recorded and a search is made for nodules. If there is any question of present activity the patients are re-admitted for assessment and treatment.

Secondly, to ensure an adequate check on any change in the cardiac status they may have occurred; at follow-up clinical and radiological signs in the heart may be either more or less marked than they were in hospital and further advice on physical activity must be given. Thirdly, to re-emphasize to the patients or their parents the continued need for sulphonamide prophylaxis and prompt treatment of sore throats or limb or joint pains should they occur.

MITRAL STENOSIS: SELECTION OF CASES FOR MITRAL VALVOTOMY

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When it became apparent that the stenosed mitral valve could be treated by surgery, clinicians immediately were faced with the problem of deciding which patients would benefit by operation. The more obvious indications were predicted from the abnormal anatomy and physiology. Others were arrived at in time by the expedient of trial and error. A number of important points are still sub judice.

It was soon evident that not every person with mitral stenosis was suitable for operation. Some of the earlier cases were failures partly because of the newness of the technique of operating inside the heart and partly because of clinical features which today would have contraindicated operation. In each of the first four cases, all fatal, reported by Bailey and his colleagues (1950), one or more of the following features were present: A very large heart, mitral incompetence, advanced cardiac failure, gross left atrial enlargement and bronchiectasis. The unsuitability of each of these findings will be referred to later.

The broad principles for selection laid down by the earlier workers in this field (Bailey, et al., 1950; Harken, et al., 1950; Baker, et al., 1950) were applied to our first patients (Bedford, et al., 1953). With experience, criteria were modified slightly, mainly towards including patients with features which heretofore would have been regarded as unfavourable or frank contraindications. The current basis for selection, influenced to some extent by discussion with others interested in the subject, but mainly by our observation and experience, is in close accord with the views expressed recently by Baker and his associates (1952). The term 'mitral valvotomy' refers to splitting of the mitral valve commissures by finger or knife; it is synonymous with 'valvulotomy' and 'commissurotomy' used by other writers.

Symptoms

The main indication for mitral valvotomy is breathlessness attributable to mitral stenosis. This fact needs emphasis, for patients with mitral stenosis may be breathless from other causes such as severe associated aortic valve disease or chronic lung disease.
It is not always an easy matter to assess to what extent a person is embarrassed by breathlessness. Many individuals are unable to explain their symptoms in their own words or they omit significant details because they are so familiar with them. However, the majority know how far, or for how long, they can walk at a brisk pace, whether they can keep up with their friends, travel to work or do their shopping or house work. Patients should be encouraged to recount such everyday experiences for they provide a good assessment of the degree of incapacity and of the improvement following operation; for these purposes they are more informative than standard exercise tolerance tests. Orthopnoea, haemoptyses and attacks of pulmonary oedema awakening the patient from sleep or precipitated by effort are common manifesta-

tions of severe mitral stenosis and are signs of pulmonary congestion resulting from obstruction of blood flow from left atrium to left ventricle.

**Physical Signs of Mitral Stenosis and Pulmonary Hypertension**

The importance of distinguishing the types of mitral stenosis likely to benefit by valvotomy has focussed attention on the other palpatory and auscultatory signs which accompany the classical mitral diastolic or presystolic murmur. In the ideal case the first heart sound is abrupt, loud and snapping in quality, features which with the presystolic thrill are responsible for the characteristic apex beat. Direct auscultation of the exposed heart and palpation within the left atrium during operation point to the fact that the first heart sound with

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**FIG. 1.—Diagram of auscultatory findings in mitral stenosis (after Wood).** A complete cardiac cycle is depicted, commencing with the first heart sound and ending with the first heart sound of the succeeding cycle. The second sound is followed by the mitral opening snap, represented arbitrarily by a single line. If the two elements of the second sound are heard, they are shown as two columns in apposition, the heights and grading depending on which element is the louder. A diastolic murmur follows the opening snap, ending with a crescendo pre-systolic murmur which is also shown commencing the cycle. The numerals are grades of loudness, from 1 to 4 (loudest)—N is normal. The diastolic and pre-systolic murmurs are graded separately. The figure below the line represents the duration of the murmur, here grade 4 (from opening snap to first sound). The grading is assessed by auscultating at the apex for the first and third sounds, and pre-systolic and diastolic murmurs, if necessary with the patient lying on the left side; at the third left interspace for the second sound, and at the fourth left interspace close to the sternum for the opening snap. Aortic murmurs are entered verbally below the diagram.

Fig. 1A represents the auscultatory findings in a patient suitable for mitral valvotomy. In Fig. 1B the signs of mitral incompetence are depicted. The third sound, later than the opening snap and without its abrupt, sharp quality, is followed by a short diastolic murmur. Patients with this auscultatory pattern are unsuitable for valvotomy.
these qualities is produced by the anterior leaflet of the stenosed mitral valve when its texture is pliant and supple. Similarly the sharp additional sound which follows the second sound, the so-called mitral opening snap originates from this leaflet early in left ventricular diastole. The evidence is convincing that these two signs, the loud sharp first sound and the opening snap, are accompaniments of a mitral stenosis amenable to valvotomy. When the mitral valve is predominantly incompetent from retraction and deformity of the leaflets and hence unsuitable for valvotomy, the first sound invariably lacks the features mentioned above (Brigden and Leatham, 1953). The auscultatory signs of mitral stenosis can be depicted conveniently by a diagram, a technique which is time-saving and at a glance gives to those familiar with the system a comprehensive idea of the most important auscultatory findings. The symbols and grading used by Paul Wood (Fig. 1) are preferred to the more detailed scheme of Levine (1949).

The signs of mitral stenosis outlined above are not of themselves indications for mitral valvotomy because they can be present without symptoms and may be discovered unexpectedly in the course of a routine medical examination. However, when pulmonary hypertension is present symptoms are seldom absent and then the indication for surgical treatment is more secure.

When the mitral valve is narrowed to a sufficiently severe degree, the blood pressure in the left atrium rises. The elevated pressure is reflected backwards through the pulmonary veins to the peripheral capillary vessels where the pulmonary venous radicles commence and the arterioles end. To maintain blood flow in face of this raised pressure, pulmonary arterial and right ventricular pressure must also increase. This has been called passive pulmonary hypertension (Wood, 1952). By a series of imperfectly understood reflexes, certain patients with severe mitral stenosis develop, in addition, an active constriction of the pulmonary arterioles; the resulting increased resistance to blood flow causes a further elevation of pulmonary blood pressure which may then reach extreme levels.

In the normal person physical exercise raises the pulmonary arterial and venous pressure but the increase is always within fairly well-defined limits. The pulmonary hypertension of mitral stenosis exceeds these limits with exercise and is the most important factor governing the amount of exercise that can be taken without breathlessness. Excessive elevation of the blood pressure in the pulmonary capillaries leads to haemoptysis or pulmonary oedema and severe, often paroxysmal, dyspnoea. The latter distressing symptom is more common with sinus rhythm than auricular fibrillation; it occurs whenever the left atrial pressure exceeds about 35 mm. Hg. Hence, it follows unusually strenuous exercise and may be the initial symptom responsible for drawing attention to the heart disease. Attacks of pulmonary oedema at rest are precipitated by tachycardia, for example with terrifying dreams, or excitement, sexual intercourse, fever, thyrotoxicosis, pregnancy and parturition, and by certain drugs with an atropine-like action.

Pulmonary hypertension in mitral stenosis may be inferred from the physical sign of right ventricular enlargement, a visible and palpable pulsation over the third and fourth left interspaces close to the sternum. Rarely the enlarged pulmonary artery can be felt in the second left interspace, and just below it, the pulmonary valves produce a palpable shock when closing, heard through the stethoscope as a loud second sound. The Graham Steell murmur of pulmonary incompetence may be present when the pulmonary arterial pressure is very high. The electrocardiogram (Fig. 2) shows right ventricular hypertrophy and, with sinus rhythm, the characteristic widened, notched Pmitrale. The radiological features are referred to below. Pulmonary hypertension is confirmed and measured by cardiac catheterization.

Cardiac catheterization has played an invaluable role in the investigation and explanation of the haemodynamics of mitral stenosis. It has allowed pulmonary arterial and capillary hypertension to be interpreted in terms of symptoms and physical signs. However, it is not an essential part of the investigation of mitral stenosis, and when the indications for operation are clear-cut, we no longer catheterize the patient. We restrict it to doubtful cases or where a discrepancy exists between symptoms and physical signs. Sometimes the physical signs ordinarily found with pulmonary hypertension are masked or attenuated by obesity, chest deformity or other causes not always identifiable. In two instances cardiac catheterization demonstrated high pulmonary pressure in the absence of physical signs or electrocardiographic changes.

Age

The danger of activating the acute rheumatic state by mitral valvotomy in young persons has fixed the lower age limit as 20, although occasionally younger patients have severe mitral stenosis for which operation cannot be delayed. Suitable cases are uncommon over the age of 50.

Associated Valve Disease

Severe aortic valve disease or gross mitral incompetence disqualifies a patient for mitral
valvotomy. These lesions may be regarded as contraindications when they are responsible for clinical, radiological or electrocardiographic evidence of moderate to severe left ventricular enlargement. Mitral incompetence excludes the patient when it causes a mitral systolic thrill and murmur, left ventricular enlargement and great enlargement of the left atrium with or without expansion during ventricular systole.

Mild aortic valve disease with little or no left ventricular enlargement, or a mitral systolic murmur without the other associations of mitral incompetence, are not contraindications.

Tricuspid incompetence may be an additional sign of high pressure in the right ventricle and pulmonary artery. Then it is functional and reversible and a reflection of the severity of the disease, not a contraindication. This type of tricuspid incompetence may improve or disappear with the pre-operative medical treatment. Organic tricuspid disease, stenosis and incompetence, may be disclosed by longstanding, intractable signs of incompetence (prominent systolic pulsation in the neck veins and liver) and can be confirmed by pressure tracings in the right atrium and right ventricle. A limited personal experience suggests that patients with this complication do poorly after mitral valvotomy.

Pulmonary incompetence (the Graham Steell murmur) is the result of high pulmonary artery pressure and is appraised accordingly.

Heart Rhythm

When the patient is suitable otherwise, the heart rhythm whether regular or irregular from auricular fibrillation appears to have no bearing on the suitability for operation. Auricular fibrillation in a patient in the early 20's or younger bespeaks severe rheumatic damage to the heart, a fact which must be borne in mind when considering surgical treatment. Older persons with auricular fibrillation have shown satisfactory improvement after valvotomy. The arrhythmia persists; we are not aware of an instance of established auricular fibrillation reverting to sinus rhythm after operation.

Emboli

Previous emboli, systemic or pulmonary, have been encountered in many patients who have later undergone a successful valvotomy. The evidence is strong, in fact, that operation diminishes or abolishes the tendency towards embolus formation.

Cardiac Failure

Paroxysmal dyspnoea is one of the cardinal features of severe mitral stenosis and one of the urgent indications for operation. The ease with which pulmonary congestion can be induced by tachycardia has been referred to above. A patient in frank congestive (right ventricular) failure should not be submitted for operation; he may respond well to medical treatment, however, and the case should be reassessed in his improved state. It has been widely taught that intractable heart failure is evidence of advanced heart disease with widespread myocardial damage. Recent observations have shown that this is not always so
and that active pulmonary hypertension which can be relieved by valvotomy is often the causative factor (Wood, 1953).

**Mitral Calcification**

Calcification of the mitral valve is best detected by radioscopy with the patient turned slightly towards the right oblique position. Extensive calcification is seen without much searching, for it stands out under the fluoroscope as a dense mobile mass at the site of the mitral valve. Formerly it was our opinion that calcification of this degree would be an insuperable obstacle to valvotomy, apart from the hazard of detaching a fragment into the circulation. However, Mr. T. Holmes Sellors has operated on a patient with gross calcification of this type who was otherwise suitable. The valve was split easily and the functional result was gratifying. No emboli were detached. Nevertheless, extensive mitral calcification is usually a feature of severe, longstanding disease with incompetence, cardiac enlargement and other unfavourable factors which contraindicate operation. The decision against operation seldom depends on calcification alone.

Less degrees of calcification in the form of discrete, opaque flecks which can be discovered often after more or less diligent fluoroscopic search are inconsequential and have no bearing on selection for operation.

**Radiography**

From what has been said about the clinical indications and physical signs of the suitable case for mitral valvotomy it should be possible to picture the radiographic features of the ideal case. The cardiac shadow should be no more than moderately enlarged; the cardio-thoracic ratio, despite its obvious shortcomings as an accurate measure of heart size, allows this permissible degree of enlargement to be expressed as not greater than 60 per cent. The left atrial appendix is visible on the left cardiac border above the ventricular curve, by virtue of the left atrial enlargement and of the adjacent infundibular (right ventricular) enlargement. The pulmonary artery curve is more or less prominent; when the pulmonary arterial pressure is greatly elevated this curve is, as a rule, conspicuous. The aortic knuckle is small or absent. Pulmonary venous congestion causes a characteristic fan-shaped shadow radiating outwards from the hilar regions. The primary subdivisions of the right and left pulmonary arteries may be dilated and superimposed on the pulmonary venous shadows.

Pulmonary haemosiderosis does not appear to influence the operative result over a period of at least 18 months. Unless longer follow-up studies alter this impression, haemosiderosis should not be regarded as a drawback to surgical treatment.
Fig. 3a illustrates the typical radiographic appearances of a patient considered to be ideal for valvotomy. A good functional result was achieved. Fig. 3b was taken one year after operation.

**Associated Diseases**

Patients with mitral stenosis are often affected by attacks of bronchitis from which permanent residual changes in the lungs such as bronchiectasis and emphysema may entail a reduced effort tolerance. Therefore breathlessness in these patients calls for careful appraisal, for when lung disease is the dominant factor it will not be improved by valvotomy. On the other hand, if the lungs have escaped detectable damage surgical relief of mitral stenosis seems to leave the patient less susceptible to recurrences of lung infections.

Pulmonary tuberculosis is seldom found with severe mitral stenosis, but if the indications for valvotomy are otherwise clear-cut, there is no reason why it should not be performed. Recently Mr. Holmes Sellors has operated successfully on a girl with the tetralogy of Fallot and left upper lobe tuberculous cavitation, a left upper lobectomy being followed immediately by a Blalock anastomosis. It would appear that equally encouraging results may be expected from surgical treatment for co-existing mitral stenosis and pulmonary tuberculosis (Hill, 1952).

Angina pectoris occasionally complicates mitral stenosis, but there is little information on the effect of valvotomy on this symptom. In two instances where it was present no attacks have occurred since operation, two to six months ago. A third patient, a woman aged 49, had angina of effort for a year and a cardiac infarction five months before operation. Since valvotomy two months ago she has had one attack of chest pain at rest.

The aggravating effect of uncontrolled thyrotoxicosis on mitral stenosis has been referred to earlier. The severity of the cardiac symptoms cannot be assessed until the thyrotoxicosis is treated. A course of thiouracil may alter the complexion of the heart disease in dramatic fashion; thyroidectomy, if preferred can then be carried out with safety. If mitral stenosis can still be blamed for symptoms and the indications outlined above are present, valvotomy should be performed.

It will be recalled in this respect that 20 years ago total thyroidectomy was in vogue for the treatment of heart failure (Blumgart, et al., 1933). The results were unpredictable and the method has fallen into disuse. Yet occasionally, the distressing paroxysmal dyspnoea of mitral stenosis can be improved or even abolished by thyroidectomy.

Mrs. A. T. (B 69026), aged 45, admitted to The Middlesex Hospital under Dr. D. Evan Bedford; mitral stenosis with sinus rhythm; for nine months frequent attacks of pulmonary oedema at night and with effort; exercise tolerance reduced to less than 50 yds. walking. Total thyroidectomy (Mr. R. Vaughan Hudson) was followed by complete freedom from symptoms. Pattern of right ventricular enlargement in chest leads VI-6 disappeared. Myxoedema was controlled with small doses of thyroid extract. After three years, heart symptoms returned and mitral valvotomy was performed with satisfactory results.

Pregnancy in patients with mitral stenosis does not call for a different set of criteria for selection for operation. In the majority of instances they proceed to term uneventfully. Yet a sufficiently large number, usually in sinus rhythm, develop acute pulmonary oedema. The physical signs of pulmonary hypertension in a pregnant woman with rapid diminution in effort tolerance are a warning and indicate bed rest. If in spite of routine treatment with digitalis, sodium restriction and mercurial diuretics no improvement is apparent, or a radiograph shows the vascular markings of pulmonary venous congestion, then the danger of pulmonary oedema is great. This hazard may appear as early as the third calendar month or in the eighth month when the burden on the circulation is said to reach its peak. Pulmonary oedema is a specially terrifying experience for the pregnant woman and recurrences are common. Mitral valvotomy may be life-saving under these circumstances, allowing the pregnancy to continue and later making the patient a fitter person to undertake the responsibilities of motherhood.

Until more is known about the long-term results of mitral valvotomy, women who have been operated on successfully should not be encouraged to embark on repeated pregnancies.

**Summary**

The criteria for selection of patients with mitral stenosis for mitral valvotomy are:

1. Breathlessness, pulmonary oedema and haemoptyses resulting from mitral stenosis not from associated valve lesions or complications.
2. Signs of the type of mitral stenosis amenable to surgery, namely a loud, abrupt first heart sound and opening snap together with the murmur of mitral stenosis.
3. Signs of pulmonary hypertension, namely palpable pulsation of the right ventricle, a loud palpable second sound and sometimes the Graham Steell murmur of pulmonary incompetence. Tricuspid incompetence may be an added sign of pulmonary hypertension; it has this significance when gross cardiac enlargement is absent. Cardiac
catheterization confirms pulmonary hypertension. It no longer forms part of the routine investigation and need only be performed when a discrepancy exists between symptoms and signs.

4. The most suitable age range is 20 to 50.

5. Severe aortic valve disease, tricuspid stenosis and mitral incompetence should be absent, although mild degrees of these lesions are permissible.

6. The rhythm may be either regular sinus or auricular fibrillation.

7. Previous emboli, systemic or pulmonary, are not contraindications. Operation may prevent future emboli or lessen their frequency.

8. Right ventricular failure should be absent, although a patient in failure may respond to treatment and eventually become a candidate for operation.

9. Gross cardiac enlargement from cardiac failure, severe associated valvular lesions or irreversible rheumatic muscular damage, contraindicates surgery.

10. Thyrotoxicosis should be controlled with thiouracil before mitral stenosis is assessed for surgery.

11. Repeated chest infections with residual permanent lung damage such as bronchiectasis or emphysema sufficient to produce symptoms, make valvotomy of dubious value.

12. During pregnancy, if symptoms increase and pulmonary oedema develops in a patient otherwise suitable for mitral valvotomy, the operation may be life-saving and allow successful delivery later. The majority of pregnant women with mitral stenosis do not require surgical treatment.

The patients referred to in this paper were under the care of Dr. D. Evan Bedford in conjunction with whom the observations were made. Many of the views expressed were influenced by the teaching of Dr. Paul Wood, whose chief assistant the writer is at the National Heart Hospital. Mitral valvotomy in all cases was performed by Mr. T. Holmes Sellors assisted by Mr. J. R. Belcher.

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