Surgical Treatment of Mitral Incompetence

ANDREW LOGAN, R. W. D. TURNER, AND A. H. KITCHIN

With the assistance of J. C. J. L. Bath, R. J. M. McCormack, J. D. Wade, and P. R. Walbaum

From the Cardiac Department, The Western General Hospital and the Thoracic Surgical Department, The Royal Infirmary, Edinburgh

Between October 1962 and March 1966, 101 patients with mitral disease from the Cardiac Department of the Western General Hospital in Edinburgh were treated by repair or replacement of the valve under cardiopulmonary bypass. Fifty patients had heavily sclerosed or calcified valves with pure or dominant stenosis and were treated by valve replacement. They presented a hæmodynamic and technical problem different from that of the repair group and will not be discussed here. This paper is concerned with 51 patients treated by repair of the valve and followed for 4 to 45 months.

THE PATIENTS

The principal clinical features are shown in Table I and summarized in Table II. The proportion of women to men was similar to that in patients treated by valvotomy for stenosis. As with mitral stenosis, it is clear that age itself is no barrier to successful treatment. The high proportion of patients with atrial fibrillation and large hearts indicates the severity of myocardial damage as discussed below.

CLINICAL FEATURES

Dyspnoea. All patients complained of undue breathlessness on exertion. The increased work of breathing responsible for the subjective sensation of dyspnoea is principally related to the level of pulmonary venous hypertension determined by left atrial pressure. This is related to mitral incompetence, with any degree of stenosis which may accompany it, and also to atrial myocardial damage and left ventricular contractility, both of which

affect pressure-volume relations in the left atrium. The situation is, therefore, complex, quite apart from the fact that breathlessness may also be influenced by pulmonary disease, obesity, anæmia, and lack of physical fitness. Although dyspnoea is sometimes a measure of left ventricular failure, it is not necessarily a good guide to the severity of valvular incompetence and objective findings are frequently more important than the subjective assessment.

Fatigue. In patients with dominant mitral incompetence fatigue may be a more important symptom than dyspnoea. This may occur when there is a low left atrial and therefore pulmonary capillary pressure and a low cardiac output, in comparison with most patients with pure or dominant mitral stenosis.

Embolism. Seven patients gave a history of systemic embolism. The incidence in those with dominant incompetence is lower than in patients with dominant stenosis.

Physical Signs. As with the introduction of each new surgical procedure, the need for an accurate pre-operative diagnosis has been the stimulus to greater precision in investigation and a particular search for each relevant clinical sign, in the hope that the correlation between signs elicited by simple methods and the results of elaborate techniques may lead to a confident diagnosis without need for the latter.

Palpation. An apical thrust from left ventricular hypertrophy in the absence of associated aortic valvular disease or systemic hypertension usually correlates well with other evidence of severe mitral incompetence, but may be absent if the chest is

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TABLE I

CLINICAL DATA BEFORE AND AFTER OPERATION ON 51 PATIENTS OPERATED UPON FOR REPAIR OF MITRAL VALVE

Case No., sex, and age (yr.)	Atrial fibrillation	Congestive failure	Pre-operative							Post-operative					Remarks
			Mitral systolic murmur	Mitral diastolic murmur†	Third sound	Aortic stenosis Aortic incompetence	CTR %	ECG*	Grade of severity†	Follow-up (mth.)	Mitral systolic murmur	CTR %	ECG*	Result	
1 F 34	0	0	44443	S	+	++	58	46	M	41	00000	58	36	Good	
2 F 31	0	0	55555	S	+	0+	54	26	M	42	12100	48	26	Good	
3 F 42	+	+	44444	M	0	0+	60	49	M	43	32100	53	RB-BB	Good	
4 M 32	+	+	44432	S	+	++	62	65	S	37	43210	62	58	Fair	Subsequent death Subsequent embolism
5 F 30	+	0	55433	M	+	++	65	30	M	39	23210	55	25	Good	
6 F 29	+	0	45555	M	+	00	75	47	S	40	24310	63	35	Good	
7 F 34	+	+	43210	S	+	00	55	42	M	34	10000	52	35	Good	Operative death
8 F 27	+	0	44444	M	0	++	70	45	VS	—	—	—	—	—	
9 F 33	+	0	44444	S	+	00	63	34	M	36	22100	58	24	Good	
10 F 43	+	+	44444	M	+	0+	73	50	VS	31	32100	70	44	Good	Subsequent embolism Operative death
11 F 49	+	0	54322	M	+	++	58	53	M	31	00000	53	42	Good	
12 F 28	+	0	44444	S	+	++	67	40	VS	—	+	—	—	—	
13 F 30	+	0	44444	M	+	++	55	55	M	31	21000	48	45	Good	Operative death
14 F 55	+	+	44444	S	+	0+	80	34	VS	—	—	—	—	—	
15 F 42	+	+	43222	M	+	++	65	34	M	30	23320	67	35	Good	
16 M 58	+	+	54444	S	+	++	65	45	S	30	44333	65	60	Poor	Re-operation Subsequent death
17 F 29	+	+	44444	S	+	++	80	47	S	31	23211	68	35	Good	
18 F 45	+	+	44444	S	+	++	70	50	S	32	32000	56	37	Good	
19 F 51	+	+	54432	M	0	++	60	48	M	32	32000	60	30	Good	
20 F 39	+	+	43322	M	+	++	67	48	S	30	00000	60	42	Good	
21 M 21	+	+	43222	S	0	++	77	67	VS	30	32100	67	55	Good	
22 F 56	0	0	44444	O	0	0+	66	39	M	26	44444	58	38	Poor	Re-operation
23 F 38	+	+	43322	M	+	0+	56	36	M	25	00000	50	40	Good	
24 M 38	+	0	45311	M	+	0+	66	55	M	27	00000	58	41	Good	
25 F 18	0	0	55555	S	+	0+	63	55	M	25	43200	54	30	Good	Re-operation
26 F 43	+	0	54322	S	+	++	68	50	S	27	43100	60	50	Poor	
27 F 31	0	+	23322	S	+	0+	50	38	M	23	01000	50	21	Good	
28 M 54	+	0	43211	S	+	00	57	42	S	22	12100	50	40	Good	Operative embolism
29 F 34	+	+	55433	S	+	00	66	65	VS	24	45322	66	29	Good	
30 F 49	+	+	45322	S	+	++	62	30	S	—	—	—	—	—	
31 M 31	0	0	54321	O	+	0	50	65	M	24	23100	50	43	Good	Operative death Operative embolism
32 F 38	0	0	43221	S	+	++	56	46	M	23	32100	48	24	Good	
33 F 29	+	0	54322	S	+	0+	65	49	S	22	21000	54	—	Good	
34 M 44	+	+	44321	M	+	0+	55	45	S	20	12100	59	40	Good	
35 F 18	+	0	44332	S	+	++	70	60	S	20	12100	58	47	Good	
36 F 41	+	+	44321	M	0	++	70	31	VS	20	33222	70	30	Poor	
37 F 54	+	+	34421	M	0	00	70	15	S	—	—	—	—	—	Operative death Operative death
38 F 47	+	0	44432	S	+	00	80	38	VS	—	—	—	—	—	
39 F 47	+	+	44510	M	+	00	69	45	M	21	00000	61	33	Good	
40 F 47	+	+	34420	S	+	++	55	50	S	20	00000	55	45	Good	
41 F 35	0	0	55544	S	+	++	57	63	M	19	44322	48	39	Good	
42 F 40	+	0	45432	S	+	00	78	38	S	18	22100	70	29	Good	
43 F 27	+	+	45531	S	+	00	80	15	VS	18	33200	71	15	Good	Operative death
44 F 42	+	0	34221	M	0	0+	57	27	M	—	—	—	—	—	
45 F 34	+	0	12100	M	+	++	72	26	S	11	10000	72	26	Good	
46 F 47	0	+	34554	S	0	00	70	80	VS	11	34321	63	70	Poor	Re-operation
47 F 45	+	+	33221	M	0	00	80	54	VS	11	23100	77	47	Good	
48 M 53	0	+	34432	O	+	00	50	53	M	6	34322	62	64	Poor	
49 F 22	+	0	34554	S	+	00	67	53	M	5	23221	76	50	Good	
50 M 38	+	0	43210	M	0	00	70	40	M	4	20000	—	30	Good	
51 F 24	+	+	34433	M	+	00	63	37	S	4	10000	64	—	Good	

*ECG, sum of deepest S in right præcordial leads + highest R in left præcordial leads (mm.).

†M, moderate; S, severe; VS, very severe.

broad or the chest wall is thick. In many cases of severe mitral incompetence an unequivocal left ventricular thrust may be present without electrocardiographic evidence of left ventricular hypertrophy. A systolic thrill is not a good guide to severity of incompetence.

Mitral Systolic Murmur. In general, the louder and harsher a mitral systolic murmur and the better it is

propagated towards the axilla and left lung base, the more likely it is to signify important mitral incompetence. In the individual case, however, intensity alone is an unreliable guide to severity, as is that of any murmur in relation to the corresponding valvular defect. Turbulence is related not to the volume flow of blood from left ventricle to left atrium but to the velocity which is influenced by the size and shape of the valve orifice and the qualities

of the cusps' margins. The valve may be very sclerotic, even calcified, or soft and supple.

In order to encourage precision and to facilitate the comparison between pre- and post-operative findings, it is advisable that the loudness of a murmur be recorded according to conventional grading (1-5) in at least five positions, at the mid-clavicular line, at the anterior, middle, and posterior axillary lines, and at the left lung base. Following operation there may be little change at the apex but considerably reduced propagation to the left axilla and lung base and unequivocal objective evidence of improvement on other grounds. For this reason it is best to record the intensity of the murmur in all these situations. The maximal intensity was graded as 4-5 out of 5 grades in 49 cases.

Third Heart Sound. A third heart sound, coinciding with the phase of rapid ventricular filling, is an important physical sign whatever its precise pathogenesis, and when loud it is unlikely to be missed. Yet it can readily be overlooked by casual auscultation, without the deliberate noting and recording of its presence or absence in each case. As is well known, a physiological third heart sound is frequently present in youth and is heard early in diastole during the normal phase of rapid ventricular filling. It is enhanced by a hyperdynamic circulation from any cause. A similar pathological sound may be associated with increased ventricular filling secondary to valvular incompetence. It may also occur with myocardial damage from any cause with normal ventricular filling and in the absence of a systolic murmur.

A third heart sound was heard or recorded in 43 of the 51 patients in this series.

Mid-diastolic Murmur. A short mid-diastolic murmur at the apex was present in all but three cases. It is impossible to tell before operation whether this is caused by a mild degree of associated stenosis or by increased blood flow through the mitral valve in diastole due to the incompetence.

Opening Snap. It has been known for some time that a clear opening snap may be present despite dominant mitral incompetence. In the present series a snap was heard or recorded in 13 of the cases, but in no instance was dominant stenosis present, and in all there was severe incompetence. This fact is emphasized because it is still frequently assumed that an opening snap is a sign of severe mitral stenosis, whereas its presence bears no close relation to the degree of stenosis or incompetence. In most cases the defect observed by the surgeon under direct vision was an elevation of the middle third

of the aortic cusp of the mitral valve above the edge of the mural cusp in ventricular systole. There was no correlation between the presence or absence of an opening snap and the site of incompetence.

Cardiac Rhythm. Most of the patients had atrial fibrillation. Only 9 were in sinus rhythm. One patient in this latter group had ruptured chordæ tendineæ in an otherwise apparently normal heart, and one patient was treated for repair of traumatic mitral incompetence produced at a previous operation for the relief of stenosis. A third, aged 18, was the youngest patient in the series: she developed atrial fibrillation 10 days after operation, but after quinidine had failed sinus rhythm was restored electrically.

TABLE II
SUMMARY OF CLINICAL FEATURES

No. of cases	..	..	..	..	..	51
Men	..	..	..	..	..	9
Women	..	..	..	..	..	42
Sinus rhythm	..	..	..	..	..	9
Atrial fibrillation	..	..	..	..	..	42
Ages						
18-19	..	..	..	..	..	2
20-29	..	..	..	..	..	9
30-39	..	..	..	..	..	17
40-49	..	..	..	..	..	15
50-62	..	..	..	..	..	8
Mitral systolic murmur						
Grade 4-5/5	..	..	..	..	..	49
Third heart sound	..	..	..	..	..	43
Cardiothoracic ratio (%)						
70-82	..	..	..	..	..	19
60-69	..	..	..	..	..	17
55-59	..	..	..	..	..	11
50-54	..	..	..	..	..	4
Clinical assessment						
Very severe	..	..	..	..	..	13
Severe	..	..	..	..	..	15
Moderately severe	..	..	..	..	..	23
Mild	..	..	..	..	..	0

Cardiac Failure. In patients with cardiac failure which is not attributable to some recent factor such as the onset of atrial fibrillation, a respiratory infection, pregnancy, or anæmia, severe myocardial impairment must be presumed and the operative risk assessed accordingly.

Congestive failure and its treatment may give rise to important electrolyte changes which will affect the risk of perfusion and must be first corrected. In some patients with long-standing failure there is clear evidence of cardiac cirrhosis of the liver, but in others with hepatic enlargement it can be assumed, despite normal tests of liver function, that some degree of damage will have resulted, and this, too,

adds to the operative risk. In our experience hepatic enlargement with systolic expansion from tricuspid incompetence is an important guide to severity and operative risk.

Renal function may be impaired by congestive failure with a consequent rise in the level of blood urea.

The term "congestive cardiac failure" is conventionally limited to peripheral signs such as distension of the jugular veins, engorgement of the liver, and œdema of the limbs, but, of course, congestion of the lungs is just as much cardiac failure and responds to similar treatment. In the present context left ventricular failure with dyspnoea, pulmonary œdema, septal lines on the radiograph, or a raised end-diastolic pressure in the left ventricle was usually the first evidence of decompensation. Fifteen patients gave a history of cardiac failure or had evidence of failure on admission, and in 10 there was evidence of considerable tricuspid incompetence.

RADIOGRAPHY

Cardiothoracic Ratio. This was used as an index of generalized cardiac enlargement before and after operation but is an unreliable guide to left ventricular enlargement since it includes portions of three chambers. In 36 of the 51 cases gross enlargement (greater than 60%) was present. Cardiac enlargement appears to correlate with myocardial damage rather than with the severity or duration of mitral incompetence.

Individual Chamber Enlargement. This is difficult to measure on the ordinary radiograph. All cases in this series had apparent enlargement of the left atrium and auricle but to a greatly variable degree from moderate to aneurysmal. Although gross enlargement of these chambers is most often associated with pure or dominant mitral incompetence, there are many exceptions and any degree of enlargement may be found with any degree of mitral incompetence or stenosis or combination of these defects.

Left ventricular enlargement as judged by rounding and displacement of the apex downwards and outwards in the anterior and left oblique views was present in all cases, again to a variable degree. Although gross enlargement of the left ventricle in patients with isolated mitral disease usually occurs with severe incompetence, it may also be present in uncomplicated mitral stenosis, presumably from rheumatic myocardial damage. It is noteworthy that considerable dilatation of the right atrium may be found without tricuspid disease and is not nec-

essarily in proportion to the pressure within (Kitchin and Turner, 1964).

Enlargement of the right ventricle is difficult to judge when associated with that of the left ventricle and may contribute to increase in the cardiothoracic ratio or to prominence of the right heart border or to backward displacement of the left ventricle in the left oblique view.

Pulmonary Artery Enlargement. Enlargement of the pulmonary artery correlates well with pulmonary arterial hypertension. It was present in varying degree in 21 cases.

Septal Lines. Septal lines from pulmonary venous hypertension are relatively uncommon in mitral incompetence unless associated with significant stenosis, because the mean left atrial pressure tends to be less raised: they were present in 12 patients.

ELECTROCARDIOGRAPHY

The electrocardiogram is often of little value in pre-operative assessment as regards the severity of incompetence, because the pattern of left ventricular hypertrophy is not always present despite other evidence of hypertrophy or dilatation. It is well known that considerable left ventricular hypertrophy must exist before it is evident electrocardiographically, and in some cases it may be that concomitant right ventricular hypertrophy neutralizes the potentials due to left ventricular hypertrophy. In other cases, as a result of rheumatic myocardial damage together with the direct effects of mitral incompetence, there is dilatation with thinning of the left ventricular wall and consequently a reduction in voltage.

The pattern of biventricular hypertrophy may be present particularly in patients with large hearts.

In patients with dominant mitral incompetence and cardiographic evidence only of right ventricular hypertrophy, there is likely to be considerable pulmonary hypertension. Severe pulmonary hypertension is rare in patients with dominant mitral incompetence and is a serious manifestation, but there were four such cases in the present series, all associated with some degree of mitral stenosis.

Table I gives the cardiographic findings as regards combined height of R wave in left præcordial leads and depth of S wave in right præcordial leads. S-T changes are difficult to interpret because the majority of patients had atrial fibrillation and therefore were on digitalis.

Hæmodynamic and Angiographic Studies are described in a separate section.

SURGICAL PROCEDURE

Surgical correction of mitral incompetence was attempted in 1938 by the use of venous homografts (Murray, Wilkinson, and MacKenzie, 1938). Thereafter, many other closed methods were tried including introduction of a pericardial band across the ventricular aspect of the mitral valve (Bailey *et al.*, 1951; Logan and Turner, 1952), placing of a baffle at the mitral orifice (Harken *et al.*, 1954), circumferential suture of the mitral annulus (Glover and Davila, 1957), and polar fusion of the mitral cusps by externally applied mattress sutures (Kay and Cross, 1955; Nichols, 1957). In general, the results of closed methods have been unsatisfactory. Our own experience (Logan and Turner, 1952) was limited to the use of pericardial bands below the mitral orifice in 19 cases. In the survivors there was evidence of reduction of incompetence and most maintained improvement for many years. Attempts to adjust the bands more precisely and under greater tension led to death from hæmorrhage and, in the climate of opinion which then obtained, the procedure was abandoned.

Establishment of the technique of cardiopulmonary bypass led to a renewal of interest in the surgical treatment of mitral incompetence (Merendino and Bruce, 1957; Lillehei *et al.*, 1958). In certain cases of traumatic mitral incompetence repair of cusps or replacement of chordæ with pericardial grafts proved satisfactory (McGoon, 1960). Substitution of defective cusps with ivalon or teflon was less rewarding because of the tendency of these fabrics to become rigid in the blood stream and to fragment (Kay *et al.*, 1958). Plication of the mitral annulus has been one of the more widely used methods of correction in non-traumatic cases and in those in which the cusps remained mobile (Effler *et al.*, 1958; Guidry *et al.*, 1958; Wooler *et al.*, 1962). The ball-valve prosthesis (Starr and Edwards, 1961) has a wider application including the more numerous cases in which the valve is rigid and incompetence is combined with stenosis.

There are few reports on the late results of annuloplasty. Bigelow (1962) studied 11 patients 1–2½ years after operation. Of these, 9 showed on catheterization a fall in atrial systolic pressure and reduction or absence of evidence of mitral incompetence: all had been in grade 3 or 4 before operation; 6 reached grade 1 and 4 grade 2 after operation. He concluded that repair by annuloplasty was usually durable. Morris *et al.* (1962) reported good short-term results in 16 patients with pure mitral insufficiency and in 14 with combined stenosis and insufficiency. They emphasized the low mortality of this operation except in those patients who had sufficient associated aortic incompetence to

produce a technical problem during perfusion. They stated: "The fear voiced by some that annuloplasty does not offer permanent benefit, because the sutures may cut through, has not been borne out in our experience." Anderson *et al.* (1962) in a study of 11 patients from 4 to 41 months after mitral annuloplasty, using clinical and hæmodynamic methods of evaluation, found that 10 showed objective improvement in one or more parameters. There was in all some decrease in heart size, and in two it was considerable. Two had developed significant mitral stenosis and in one, after initial improvement, mitral incompetence had recurred. In the experience of Ellis *et al.* (1963) and in our own experience, the mortality of annuloplasty is lower than that of mitral valve replacement.

Before the development of confidence in the efficiency of annuloplasty we tried several of the procedures mentioned above. Teflon or ivalon extensions of the mural cusp large enough to affect reflux seemed in addition to offer some obstruction to the normal flow of blood. Thereafter, it was observed that a single suture reducing the size of the annulus could completely abolish reflux, and from that time, with the three exceptions mentioned below, no procedure other than plication of the annulus was used, provided that the cusps were not so sclerosed or calcified as to cause important valvular obstruction, in which case the valve was replaced with a Starr-Edwards prosthesis. This paper is concerned only with the cases treated by annuloplasty, together with one case of ruptured chordæ treated by chordal replacement and two cases of traumatic mitral incompetence due to a torn cusp which was repaired.

Principles of Annuloplasty. Mitral annuloplasty is applicable only to valves in which the cusps are neither calcified nor heavily sclerosed, are not fused or, if fused, can readily be separated, and are not fixed by adhesion or shortening of the chordæ. The operation is based on two anatomical features—the tough, fibrous atrioventricular ring, and the shallow funnel-shape of the mitral valve. The diameter of the ring is larger than the orifice itself, and one can therefore narrow the ring by folding without causing stenosis of the orifice. Sutures round or through the fibrous ring to secure the fold have less likelihood of cutting through than if they were in myocardium. Plication alters the shape of the valve as well as its size, and with a view to reducing that diameter which is at right angles to the long axis of the valve orifice, the plication is made adjacent to the commissures. In order to avoid distortion of the aortic valve, no suture encroaches on the base of the aortic cusp of the mitral valve (Fig. 1). One likely

FIG. 1.—Relation of aortic or anterior cusp of the mitral valve to the aortic valve.

cause of recurrence of incompetence is the tenuous nature of the annulus in some cases. The fibrous tissue composing it may be so dense as to cause difficulty in the passage of a round-bodied needle, or so soft as to offer barely appreciable resistance. Slackening of the sutures may also occur when atrial muscle is included in them. Mattress sutures with their longer thread may become looser than simple sutures if any myocardium is included. We, therefore, believe that the annuloplasty should be done with simple strong non-absorbable sutures, taking the whole thickness of the atrio-ventricular ring, avoiding the cusp, and including as little muscle as possible.

Operative Technique. Initially our approach for direct exposure of the mitral valve was by a right thoractomy because we were concerned to be able to expose the

FIG. 2.—Diagram showing atrial side of incompetent mitral valve; the middle third of the anterior cusp rises into the atrium above the posterior cusp.

aortic and tricuspid valves. It had the advantages of easy access to the right atrium for withdrawal of blood to the oxygenator and of avoidance of an obliterated left pleural space in those patients who had previously undergone closed mitral surgery through a left approach. However, from the right side the mitral valve was more distant and its anatomy less familiar. These considerations led us to use a left thoractomy which we immediately preferred for most cases. It allows through the left atrium a wide exposure of the valve, orientated as we have been accustomed to feel it. The left ventricular apex is readily accessible for introduction of a venting catheter.

A Melrose pump oxygenator was used before 1964 but thereafter we have employed an NEP rotor pump and disposable plastic oxygenator primed with 5 per cent dextrose, 20 ml./kg. body weight. ACD blood is added to make up the required priming volume. The operation is done at normal body temperature, and co-ordinated ventricular action is maintained throughout so that the degree of mitral incompetence can be observed. The outflow of the right ventricle is cannulated for withdrawal of blood to the oxygenator. A vent is placed in the apex of the left ventricle and the left atrium is widely opened with an incision parallel to the coronary sulcus and passing between it and the left lower pulmonary vein. Retraction forwards of the anterior edge of this incision gives a clear view of the mitral valve and the degree and nature of the incompetence can be seen.

The immediate cause of incompetence most frequently encountered was that the edge of the aortic cusp rose into the atrium above the edge of the mural cusp with ventricular systole (Fig. 2).

This failure of apposition affected the middle third or more of the cusps. The consequent reflux was directed laterally to strike the atrial wall just below the left lower pulmonary vein, a position recognized by external palpation of the heart during operation for mitral stenosis as the usual site of the thrill associated with minor degrees of mitral incompetence. The reason for this failure of the cusps to meet was not in any case confidently recognized. Possible causes were shrinkage of the mural cusp, rupture of chordæ from the aortic cusp, or enlargement of the valve ring, but there were reasons against accepting any of these explanations. A ruptured chorda of the aortic cusp was found in two cases, but in both the separation of the cusp margins was greater than could have been caused by the loss of a single chorda, and reconstitution of the chorda with pericardium failed to correct the incompetence; in several patients the mural cusp was shrunken and immobile but in others it was well formed, and moreover we have palpated numerous valves in which the mural cusp was a mere fixed narrow ridge and yet the valve was competent. There was considerable variation in the size of the valve ring associated with incompetence but in no instance was it considered that incompetence was due principally to enlargement of the ring.

After visual assessment of the degree of incompetence it is confirmed that the valve is suitable for annuloplasty in that the cusps are not calcified or very fibrosed

and the chordæ not grossly shortened. The annuloplasty sutures are then placed—one anteriorly and one posteriorly—and each takes a bite of the valve ring at the corresponding commissure and again a short distance away along the attachment of the mural cusp (Fig. 3). One correctly placed suture, its proper position perhaps ascertained by trial, at each commissure will completely abolish the incompetence, and initially we relied on these single unsupported sutures until late recurrence of incompetence in a few cases was observed. We therefore now support this basic repair with further sutures. After competence has been obtained, the left ventricle is kept empty by gentle suction on the apical vent. The folds in the ring made by the two sutures are each secured by two or three more sutures according to the length of the fold and finally the knots of all the sutures are covered with atrial wall which is bound down by a single light suture. It is probable that the ring should be narrowed a little more than is necessary for immediate competence to allow for slight loosening of sutures. On completion of the repair the left ventricular vent is temporarily clamped to allow the left ventricle to fill with blood, thereby permitting a final check on the competence of the valve.

The left atrial incision is now closed and when cardiac action is satisfactory, which is usually immediately, the left ventricular vent is removed and bypass is ended. The venous line is removed, the right ventricular incision is closed, and the pericardium is loosely sutured. The chest is closed in layers leaving an intercostal drain attached to a water seal.

In the 48 patients who underwent mitral repair of this kind we were struck with the frequency of pure mitral incompetence, the cusps being no more than slightly sclerosed. Only four had sufficient fusion of the cusps to require valvulotomy. In addition, one patient had ruptured chordæ of the aortic cusp which were repaired and one had an ostium secundum type of atrial septal defect which was closed.

In the case of mitral incompetence due to spontaneous rupture of chordæ a different technique was employed. An unusually strong, coarse, systolic thrill was felt over the ascending aorta but not over any part of the left atrium, and caused momentary doubt about the diagnosis which, however, had been confirmed by angiocardiography. When the left atrium was opened, all the chordæ which should have secured the mural cusp to the papillary muscles were found to be broken, leaving the longer parts of the chordæ attached to the muscles and short tags on the cusp. No abnormality of the cusps or of the intact chordæ was recognized. The chordæ which were broken looked otherwise normal. The flail cusp fluttered up into the atrium during ventricular systole and directed a gush of blood to the right against the aortic root (Fig. 4). To replace the broken chordæ a single band of pericardium was used. One end was sutured along the margin of the mural cusp where it had lost its chordæ. The other end was passed through the ventricular wall at the base of the anterior papillary muscle and drawn outwards until the band had the degree of tension required to prevent prolapse of the mural cusp. The external end of the band was then

FIG. 3.—Annuloplasty—placing of sutures in the mitral ring.

sutured to the anterior ventricular wall. The left atrium was closed and the operation was ended.

It is possible that adjustment of the autogenous pericardial graft was made when the systolic pressure in the left ventricle was rather low, because, though the patient became asymptomatic and there was no residual sign of severe mitral incompetence, there was still an apical systolic murmur. From our experience of the use of pericardium in the chambers of the heart it seemed unlikely that the band would lengthen with age and permit recurrence of prolapse of the cusp. Shortening and sclerosis seemed more probable changes. In the event the pericardium was dragged through the ventricular wall with recurrence of incompetence.

In the two cases of mitral incompetence consequent upon valvulotomy for stenosis, incompetence was due to a tear of the posterior attachment of the aortic cusp. The tear was repaired with interrupted 4×0 silk sutures.

Post-operative Management. Venous and arterial pressures and the biochemical state are monitored for at least 24 hours after operation and for longer in those patients causing anxiety. At the end of the operation and before removal of the arterial perfusion cannula, blood is transfused from the oxygenator to the patient until the central venous pressure rises to approximately 20 mm. Hg. This pressure falls within a few hours, but usually enough blood is transfused over the night of operation to

FIG. 4.—Case 22. Incompetence produced by ruptured chordæ of posterior cusp.

keep the venous pressure at about 10 mm. All the patients are digitalized over the period of operation and many require diuretics for mild degrees of heart failure in the early post-operative days. An endotracheal tube is kept in position for 24 hours after operation, and respiration is assisted by a triggered Bird ventilator. It is our impression that this practice has resulted in less exhaustion for the patient and a lower incidence of respiratory complications. Prophylactic chemotherapy is not used but tracheal aspirate, pleural drains, and sputum are examined bacteriologically, and if a pathogenic organism is isolated, the appropriate antibiotic is given.

Operative Deaths. The syndrome of low cardiac output occurred in five patients, three of whom had severe aortic incompetence. The features of the syndrome were exhaustion, dyspnoea, cold blue extremities, oliguria, and desaturation of the venous blood. These patients were treated for heart failure, and intermittent positive pressure respiration was maintained through a tracheostomy. Despite these measures, four patients died as a direct result of the continuing low cardiac output, and the fifth died after an epileptiform seizure on the second post-operative day when it appeared that his condition had started to improve. Necropsy in this patient showed generalized anoxic brain damage; necropsies in the other two patients dying

with low cardiac output showed in one a large infarct of the left ventricular wall and in the other a partial obstruction of the mitral valve by clot.

A sixth patient failed to recover consciousness after operation and death was due to cerebral embolism. At operation there was a large friable thrombus in the left atrium and a piece of this was known to have escaped in the left ventricle. A seventh patient died after operation from a nursing error in relation to blood transfusion.

Five of these seven deaths were in the very severe group.

Operative Complications in Survivors. Three patients sustained cerebral damage at the time of operation but recovered. Micro-embolism, with air or particles of clot or other substances, and inadequate perfusion were considered possible causes but in none was the precise cause established. Cerebral damage of uncertain aetiology is a recognized complication of open-heart surgery with the bypass technique.

One patient had a series of epileptiform convulsions during the first few post-operative hours and thereafter made a normal convalescence. One appeared initially to have severe cerebral damage but over the next few months proceeded to make a steady recovery and now has no residual disability. The third patient also had a severe initial disability

with hemiparesis and partial aphasia, but made slow progress over the next few months and now has a mild disability and has returned to his previous occupation which involves driving a car.

LONG-TERM RESULTS

Of the 44 patients who survived operation, 36 are much improved as far as the heart is concerned, but 2 of them are handicapped in some degree by the effects of cerebral damage, though they are able to live normally active lives (Table III). Two patients died suddenly, one while swimming, 24 and 34 months after operation.

TABLE III
RESULTS IN 43 PATIENTS

Status	No. of patients
Operative deaths	7
Subsequent deaths	2
Improved	36
Good	36
Fair	0
Not improved	6
Recurrence of incompetence	3
Persistence of incompetence	3
Subsequent embolism	3
Subsequent re-operation	4

Six are classified as having poor results. One had a very severe disability with gross congestive failure and ascites before operation and though no worse is not really improved. Four with recurrence of the mitral systolic murmur to an intensity almost as great as before operation and with recurrence of symptoms have been treated surgically a second time but with valve replacement.

In some previously very severely disabled patients post-operative improvement has been remarkable. Two have achieved successful pregnancies without difficulty. One woman aged 28 with an enormous heart who was unable to undertake any form of physical activity, and had been largely confined to bed for 15 years, is now leading an almost normal life and recently went on a walking tour in the Lake District, climbing hills with little dyspnoea. Some other results have been almost as dramatic and most patients have returned to work or normal household activities.

It may be that with correctly placed and adequately supported plicating sutures recurrence will be less frequent, but it is too early to be sure of this and it seems likely that in a few cases, especially with large hearts from myocardial damage, further stretching of the valve ring will occur. We are, however, sufficiently encouraged to persevere with this technique which on general principles seems preferable to the insertion of a prosthesis. These

results are gratifying, but we are aware that irreversible myocardial damage as shown by considerable cardiac enlargement persists in some patients and in some there is aortic valvular disease.

We are uncertain about the long-term significance of the mitral systolic murmur which has persisted or recurred in some patients, but it is clear that the post-operative loudness of the apical systolic murmur does not always bear a close relation to the relief of dyspnoea and objective evidence of improvement.

Mitral Systolic Murmur. In the 44 survivors of operation the mitral systolic murmur was abolished in 8, much reduced in 16, reduced in 16, and unchanged in 4 patients (Table IV).

TABLE IV
RESULTS IN 44 SURVIVORS OF OPERATION

Mitral systolic murmur	Abolished ..	8
	Much reduced ..	16
	Reduced ..	16
	Unchanged ..	4
Third heart sound	Abolished ..	41
	Unchanged ..	3
Cardiac enlargement	Much reduced ..	23
	Reduced ..	9
	Unchanged ..	12

One important observation has been that in many cases there is little change in the loudness of the murmur at the apex but a considerable reduction in the loudness in the axilla and left lung base with considerable subjective improvement and objective improvement on other grounds. It is for this reason, as emphasized above, that the intensity of the systolic murmur should be recorded on a five-point scale in at least five positions from the mid-sternal border to the left lung base (Fig. 5).

Third Heart Sound. A previously present third heart sound was abolished in all but 3 patients. It is clear that occasionally a third heart sound may persist despite abolition or great reduction in the loudness of the systolic murmur.

Cardiac Enlargement. Cardiac enlargement as judged by the cardiothoracic ratio was much reduced in 23, reduced in 9, and unchanged in 12 patients (Fig. 6).

In some cases radiographic evidence of improvement has been striking (Fig. 7-9) even in those with very large hearts, while in others, despite great functional improvement, there has been relatively little change. Presumably in such cases there has been long-standing dilatation of the atrium and

FIG. 5.—Mitral systolic murmur graded in two positions before and after mitral annuloplasty.

ventricle with severe rheumatic myocardial damage and the cardiac muscle has lost its elasticity.

The disappearance of septal lines caused by distended lymphatics in the interlobular septa pro-

vides reliable evidence of a fall in pulmonary venous pressure secondary to that in left atrial pressure. Septal lines disappeared in all patients in whom they were previously present and who obtained a satisfactory functional result.

FIG. 6.—Cardiothoracic ratios before and after mitral annuloplasty.

Electrocardiography. The electrocardiogram even in severe mitral incompetence frequently shows no evidence of left ventricular hypertrophy, and post-operative changes have been a poor guide to improvement. Nevertheless, taking the maximum height of R and depth of S in præcordial leads as some index of left ventricular potentials, there was generally, as shown in Fig. 10, a reduction in this index after operation. There was no consistent change in the mean QRS axis, and since, because of atrial fibrillation, most patients were on digitalis, changes in the ST-T portion of the electrocardiogram were impossible to analyse.

Systemic Embolism. In only one patient in this series was clot observed in the left atrium at the time of operation, though in other cases clot may have been present in the left auricle which was not explored. This was the only patient in whom it was considered that embolism occurred during the operation and was responsible for cerebral damage and death. In addition to clot, she had a moderate degree of stenosis with calcification of the valve and in these combined respects was different from all other patients in the series treated by repair.

Three patients sustained systemic embolism 2 weeks, 4 weeks, and 10 months, respectively, after the operation. In none had atrial clot been seen and

Fig. 7.—A and B, showing reduction in heart size after annuloplasty.

none had been treated with long-term anticoagulants. The first, with a mild hemiparesis, made a rapid and complete recovery. The second is well except for a speech defect. In these two patients time relationships suggest that embolism was due to thrombus laid down at the time of operation or soon

after. The third patient, who had a mild hemiparesis 10 months after the operation, had twice experienced systemic embolism before operation. There is no reason to relate recurrence to the operative procedure. After these complications all patients were treated with long-term anticoagulants, but we are

Fig. 8.—A and B, showing reduction in heart size after annuloplasty.

FIG. 9.—A and B, and C and D, showing reduction in heart size after annuloplasty.

uncertain how long this treatment should be continued.

HÆMODYNAMIC STUDIES

In most cases uncomplicated mitral incompetence can be diagnosed and its severity can be estimated clinically. When in addition there is mitral stenosis or aortic or tricuspid disease, the assessment of the relative severity of mitral incompe-

tence may be impossible without cardiac catheterization and angiography. In some cases of mitral incompetence the auscultatory signs are misleading (e.g. Case 22).

In the present series catheterization was carried out in most cases, not usually to establish the diagnosis but in order to obtain data on intracardiac pressures and cardiac output to correlate with other indices of severity, and in order to evaluate the hæmodynamic effects of annuloplasty.

FIG. 10.—Electrocardiographic potentials: sum of maximal S and maximal R in præcordial leads before and after mitral annuloplasty.

Number of Cases Catheterized. Of the 51 cases, 31 were catheterized before operation. Those who were not catheterized include 3 patients at the beginning of the series before the study was planned and a group of patients previously assessed in another centre. In general, they did not differ in any respect from the rest of the series, and the mode of selection of catheterized cases, therefore, did not affect the representative nature of the findings.

Post-operative studies were carried out 1 to 12 months after operation in 20 cases. In 16 of these, data were complete enough for a valid comparison with the pre-operative study. In 3 of the remaining 4 cases pre-operative catheterization had not been carried out. With the exception of 3 cases re-catheterized because of suspected deterioration after operation, the 16 cases who had full pre- and post-operative studies were unselected on clinical grounds.

Techniques. Catheterization was carried out percutaneously through the right femoral vein using the Seldinger technique. Pressures were measured using Statham strain gauges. The reference level was 10 cm. above the table top. Right atrial, right ventricular, left atrial, and left ventricular pressures were recorded, the left heart pressures being

obtained by transseptal puncture (Brockenbrough, Braunwald, and Ross, 1962) with a No. 7 teflon catheter.

Cardiac output was measured by the Fick principle with simultaneous expired air collection and right ventricular mixed sampling over two-minute periods. Air was analysed by the Lloyd-Haldane method and oxygen saturation of the blood was measured by the Brinkmann hæmoreflector. In most cases the arterial sample was obtained from the left atrium after transseptal puncture.

Indicator dilution curves (indocyanine green) were recorded with an earpiece densitometer from left atrial injections.

The aorta and left ventricle were catheterized retrogradely from the femoral artery for angiocardiology. A single plane vertical beam was employed and the most useful projection was found to be right anterior oblique in which the mitral valve is seen in profile.

Phonocardiograms at the apex and apex cardiograms were recorded.

Pre-operative Data. The range of values found for the main parameters is shown in Fig. 11.

Cardiac Index and Stroke Index. A low forward cardiac output was almost invariable. The mean

FIG. 11.—Distribution of intracardiac pressures and cardiac output in patients catheterized before operation.

cardiac index was $2.64 \text{ l./min./sq. m.} \pm 0.12$ (standard error of mean). The forward stroke index was $33.5 \text{ ml./sq. m.} \pm 2.07$. Normal values for these parameters are $3.8 \text{ l./min./sq. m.}$ (range $2.5-5.1$) for cardiac index and 47 ml./sq. m. for stroke index (Donald *et al.*, 1955). Exercise was studied in 5 patients. In these the rise in output was sub-normal.

Mixed Venous Oxygen Saturation. Depression of this value is a feature of poor systemic perfusion. In general it is related to cardiac output unless there is significant arterial undersaturation. The normal resting mean value is about 70 per cent. In the present series the values ranged from 39 to 75 per cent (mean $59.9\% \pm 1.60$). On mild exercise in 5 patients falls of 20 per cent were usual and values as low as 27 per cent were found.

Left Atrial Pressure. All patients were in atrial fibrillation and the form of the pressure wave in the

left atrium generally showed the pattern of a late systolic peak wave with a rapid descent following ventricular relaxation. Examination of the wave form alone, however, was of little use in determining the degree of regurgitation. Atrial size and presumably compliance are very large uncontrolled factors in such estimates. Some cases with gross reflux and aneurysmal dilatation of the left atrium had normal left atrial pressures, as has been noted previously (Braunwald and Awe, 1963). Other cases with relatively small atria could generate peak left atrial pressure of 40 or 50 mm. Hg, with apparently no greater degree of reflux and less disability. In some cases the peak left atrial pressure equalled the right ventricular systolic pressure. The average peak pressure was $30.8 \text{ mm. Hg} \pm 2.01$ and mean left atrial pressure $18.6 \text{ mm. Hg} \pm 1.26$. On exercise in 5 patients both mean and peak atrial pressures rose. In one the peak pressure exceeded 90 mm. Hg.

Left Ventricular Pressure. Slight increases in the left ventricular end-diastolic pressure were common (over 10 mm. Hg in 12 out of 31 patients) and are related to the large total stroke volume (forward and backward) and increased ventricular filling which accompanies mitral reflux. In a few instances it was considerable (over 15 mm. Hg), and because of the presence of other features (in particular a slow rate of pressure generation by the ventricle and a particularly low cardiac output), was thought to indicate some degree of myocardial "failure".

A diastolic gradient across the mitral valve was the rule when left atrial pressure was raised. The mean gradient was 4 mm. Hg, but gradients as high as 15 mm. Hg were present with dominant reflux. In 5 instances a significant degree of mitral stenosis was present and these patients tended to have the highest pressure gradients.

Pulmonary Hypertension. Some passive increase in the pulmonary artery pressure, due to the rise in left atrial pressure, was the rule. The mean right ventricular systolic pressure was $42.9 \text{ mm. Hg} \pm 2.62 \text{ mm. Hg}$. In these cases the calculated pulmonary vascular resistance was low (mean 2.1 units or $170 \text{ dynes-sec.-cm.}^{-5}$). In 5 patients, however, the pulmonary artery systolic pressure was high (over 65 mm. Hg): 4 of them had some mitral stenosis as well as the predominant incompetence; and the fifth had in addition an ostium secundum atrial septal defect with a large shunt and a high pulse pressure in the pulmonary artery, though little increase in pulmonary vascular resistance.

In general the results confirmed the low pulmonary vascular resistance in mitral incompetence unaccompanied by significant stenosis.

Tricuspid Incompetence. Exaggerated systolic waves in the right atrial pressure pulse associated with pulsating neck veins and indicating tricuspid incompetence were present in 5 instances.

Apex Cardiogram. The excursion of the apical impulse recorded by low frequency pressure transducer showed a typical pattern with an abrupt rise at the onset of ventricular systole, a rather unsustained plateau during systole, and a rapid indrawing at the onset of ventricular relaxation. During diastole there was a secondary thrust in the position of the third sound, representing rapid ventricular filling. Many patients with gross mitral incompetence and large atria had a parasternal lift which might have been mistaken for a right ventricular thrust. The form of the impulse, however, is quite different, with a late systolic peak corresponding closely to the left atrial pressure pulse, and in the absence of pulmonary hypertension there is no doubt that this parasternal impulse is due to left atrial expansion in ventricular systole.

Phonocardiogram. This proved useful in confirming the presence of a third heart sound in some cases.

Indicator Dilution Curves. The presence of valvular regurgitation between the injection and sampling sites distorts the normal indicator dilution curve to produce slowing of the disappearance slope, and obscures the recirculation peak. At one time it was believed that a quantitative estimate of regurgitant flow could be obtained from analysis of the dye curves (Korner and Shillingford, 1955), but this has not been borne out in practice (Marshall, Woodward, and Wood, 1958).

Angiocardiogram. Angiocardiography with injection of contrast material into the left ventricle was carried out in 23 instances. Several early angiograms were technically unsatisfactory. It was found that a single plane vertical projection with the patient in the right anterior oblique position gave the best demonstration of valve size and function and of the degree of regurgitation. Retrograde catheterization was preferred to the trans-septal route for the angiocardiogram, not because the presence of the catheter in the valve might produce spurious incompetence—this was thought unlikely and in fact did not occur in normal mitral valves—but because of the tendency of the trans-septal catheter to flick back into the atrium during injection. A possible fallacy is the production of incompetence by extrasystoles which are difficult to avoid during left ventricular injections.

FIG. 12.—Left ventricular angiocardiogram: no mitral reflux.

With experience it becomes possible to gain from the angiographic appearances a fairly accurate impression of the degree of regurgitation found at operation. The relative opacification of atrium and aorta, bearing in mind the site of the atrium, and the appearance of the regurgitation contrast medium are important.

Regurgitation through a predominantly stenotic valve appears as a fine jet; that through a grossly incompetent valve appears as a broad wave of dye rapidly filling the atrium (Fig. 12–14).

The angiographic method of assessing regurgitation is clearly not quantitative, and its reliability is a function of the experience of the investigator. Nevertheless it yielded crucial information in certain cases, particularly Case 22: the physical signs simulated aortic stenosis, with a murmur and thrill maximal in the aortic area and transmitted to the carotid arteries; however, the aortic valve was shown to be normal, and the murmur was produced by initial incompetence from ruptured chordæ of the posterior cusp. Similar cases have been reported (Osmundson, Callahan, and Edwards, 1958).

Patient Groups and Correlations. With the above data it was possible to divide the 31 patients into various subgroups as follows. (1) Five patients had functional tricuspid regurgitation; (2) five patients had severe pulmonary hypertension; (3) in 8, the left ventricular end-diastolic pressure was con-

Fig. 13.—Left ventricular angiogram, showing moderate mitral reflux through a narrowed valve. 13B is taken 0.4 sec. after 13A.

siderably raised; and (4) in 7, the cardiac output was very low.

Although numbers are small, an attempt to distinguish probable correlations among the various parameters measured was made using rank correlation methods. Contingency tables were prepared for the correlations which appeared relevant and the

significance of apparent associations tested by the χ^2 test.

The only correlations which were significant ($\chi^2 > 1.6$) were those between severity and the intensity and propagation of the mitral systolic murmur, and between severity and calculated pulmonary vascular resistance. A history of oedema or diuretic requirement was associated with an increase in the left ventricular diastolic pressure. Cardiomegaly was associated with a widely propagated mitral systolic murmur, an increase in the left ventricular diastolic pressure, and raised left atrial pressures. The higher the mean left atrial pressure, the higher the peak systolic pressure in the atrium. Notably, cardiac index and clinical severity or heart size were not significantly correlated.

FIG. 14.—Left ventricular angiogram, showing severe mitral reflux.

Post-operative Data. Comparison of pre- and post-operative data in the 16 patients in whom they are available are shown in Fig. 15. The whole number was divided into 2 groups of 8, shown separately, according to the apparent clinical improvement. In group B, 3 patients obviously did badly after operation and their data show deterioration; the 5 others show improvement in forward output and pressures, but clinically they were less than satisfactory with persistence of a loud systolic murmur. The 8 patients in group A all showed great clinical improvement.

Cardiac Index and Stroke Index. All but 3 patients showed an increase in cardiac index and stroke

FIG. 15.—Comparison of pre- and post-operative catheterization data in 16 patients, divided into two groups according to the degree of clinical improvement.

index. The mean increases were 1.01 ± 0.14 l./sq. m., and 14.8 ± 3.7 ml./sq. m., respectively, and the differences are highly significant ($p < 0.001$).

Pressures. Except in 3 patients, left atrial mean and peak pressures fell after operation. Mean pressure fell by 5.6 ± 1.38 mm. Hg, and peak by 9.3 ± 2.16 mm. Hg ($p < 0.01$ for both). The right ventricular systolic pressure fell also (mean fall 12.1 ± 2.17 mm. Hg), despite the increased pulmonary blood flow.

Although in a group of patients the wave form of the left atrial pressure is not a good guide to the severity of mitral incompetence, in the individual patient, in whom such factors as atrial size and compliance are constant, the height of the systolic regurgitant wave probably follows closely changes in the volume of regurgitant flow. Fig. 16 shows a series of 4 representative examples of the left atrial pressures in the same patients before and after annuloplasty: the upper two examples show considerable reductions of the systolic wave. The third

is a patient in whom there was some associated stenosis of the valve, and the fourth is one with a large atrium in whom the result was unsatisfactory.

Indicator Dilution Curves. Post-operative curves were recorded in 17 of the 19 patients. DT/BT ratios (Broadbent and Wood, 1954) were calculated for each patient and ranged from 1.5 to 3.6. The normal upper limit (2.1) was exceeded in 5 of the 17 patients, indicating persistence of significant regurgitation. The rest, however, had curves of normal contour indicating little or no regurgitant flow.

Pre- and Post-operative Comparisons. These must be viewed critically because of the possible changes in several variables which may affect the results. Post-operative anaemia, with reduced oxygen-carrying capacity of the blood, might account for a rise in resting cardiac output. If this were the explanation, however, intracardiac pressures would tend to rise rather than fall; also the fact that the rise in output is associated with a decreased arterial

LEFT ATRIAL PRESSURE

FIG. 16.—Four examples of direct left atrial pressure recorded before and after mitral annuloplasty.

venous oxygen saturation difference discounts anaemia as a possible explanation. The mean change in oxygen capacity in the group was a fall of 0.71 ml./100 ml., which was not significant ($p > 0.3$).

It is theoretically possible that the output figures, though not the pressure changes, might be affected by the persistence of a small left-to-right shunt through the site of transeptal puncture. To account for the measured rises in pulmonary blood flow, however, there would have to be an atrial septal defect of moderate size. At operation, except in one case where the site of puncture was visible, permitting the escape of a fine jet of blood, there was no evidence of any such shunt.

The conclusion seems justified that the com-

bination of a rise in pulmonary blood flow with a fall in left atrial and right ventricular pressures after annuloplasty points to increased forward flow and decreased mitral regurgitation.

DISCUSSION

In the individual patient the severity of regurgitation cannot be judged solely on the intensity of a systolic murmur, though there is a fairly good correlation in a large group of patients. A loud mitral systolic murmur may be present without significant mitral incompetence, i.e. without relevant symptoms, a third heart sound, pulmonary venous or arterial hypertension, and without left atrial or

left ventricular enlargement or hypertrophy. If there has been little myocardial damage, compensation may last for many years. Since surgical methods are likely to improve, operation should be deferred in such cases.

Severe incompetence may be present without a loud murmur. In one patient not included in the present series there was a barely audible systolic murmur and a long rumbling diastolic murmur with all the customary clinical evidence of mitral stenosis. This patient had, however, a wide valve with scarcely any stenosis and gross incompetence which was subsequently corrected by annuloplasty.

Since it may not be possible to know with certainty the degree of valvular incompetence, its severity should be assessed from time to time by haemodynamic and angiocardiographic methods. Some patients with large hearts from severe myocardial damage have relatively unimportant mitral incompetence, and in them there may be difficulty in assessing the relative significance of these two factors.

A third heart sound may occur with myocardial damage from any cause, when ventricular filling is normal and there is no systolic murmur, but it is not necessarily a sign of severe mitral incompetence but may result from myocardial damage. This sound is important as it is evidence of severe rheumatic heart disease. Its presence, except in the relatively young, suggests important incompetence or myocardial damage, and in its absence the necessity for surgical treatment should be seriously questioned. In two patients in whom the systolic murmur was eliminated by surgical treatment, the third sound persisted.

Post-operative improvement may be assessed by a reduction in the cardiothoracic ratio and in left atrial and left ventricular size, and by disappearance of pulmonary congestion or of septal lines in the costophrenic angles. A reduction in the cardiothoracic ratio is not specific for any one cardiac chamber and probably implies diminished residual blood volume from improved myocardial performance rather than a reduction in hypertrophy.

Alteration in left atrial volume cannot be measured precisely except by simultaneous biplane angiography but may be obvious in the postero-anterior and in the oblique views. A reduction in left ventricular volume is difficult to distinguish from a reduction in over-all cardiac size, and again is largely a subjective assessment by ordinary radiography but is nevertheless often convincing.

Contraindications to Operation. Judgement in the choice of patients for surgical treatment can only be achieved by operating in the different groups and

by correlating surgical findings with adequate pre- and post-operative studies and long-term results. In those patients who have had severe congestive cardiac failure with considerable cardiac enlargement or pulmonary hypertension and in whom secondary changes in lungs, liver, and kidneys can be presumed, the risks of open-heart surgery are considerable. Five patients in this series who died as a direct consequence of the operation were in this group. Nevertheless, other patients of comparable gravity survived, so that the adverse factors referred to above are relative rather than absolute contraindications. In the poor risk group the decision for or against operation must be based not only on the severity of heart disease and the general condition, but also on the individual's attitude to the disability and domestic responsibilities.

Optimal Time for Operation. In this disease there is often progression in left ventricular enlargement with replacement of muscle by fibrous tissue and therefore important and irreversible myocardial damage. In the individual patient it cannot be proved that earlier surgical treatment for mitral incompetence would have arrested this pathological process but it is a reasonable assumption. It is also probable that the operative risk and long-term results will be better if surgical treatment is not delayed. In some cases, followed for years before operation, there has been a progressive increase in size of the heart, and after operation there may be an appreciable reduction. In others, despite correction of the defect, little change results.

Since the operative risk has been shown to be reasonably low, treatment should be advised as soon as it is certain that severe mitral incompetence is present and particularly if there is evidence of progressive left ventricular hypertrophy or enlargement. Improvement in results is likely to follow not only from advances in surgical and perfusion techniques but from the decision to advise surgical treatment early, before irreversible changes have occurred.

Long-term Prognosis. It is too early to express a confident opinion on the lasting benefits to be expected from the surgical repair of the mitral valve but it is probable that long-term results will largely depend on the degree of previous myocardial damage from rheumatic myocarditis and mitral regurgitation. The left ventricle has remarkable powers of compensation by hypertrophy and in non-rheumatic conditions, for example aortic valvular disease, it may not fail for many years. Unfortunately in rheumatic heart disease myocardial

damage is frequently present apart from that due to increased left ventricular work, and when advanced is just as likely to be irreversible. Benefit may still result from reduction in the undue hæmodynamic burden on a damaged myocardium, but in many cases improvement must be limited by the severity of this myocardial damage.

SUMMARY

Fifty-one patients were treated by repair for mitral incompetence and followed thereafter for 4 to 45 months.

Criteria for assessing severity, which is related to valvular incompetence and to the equally important myocardial damage, are described.

Twenty-eight patients were classified as having "severe" or "very severe" rheumatic heart disease, 23 "moderately severe" disease, and none "mild".

Clinical manifestations are discussed in detail.

In order to ensure precision and to facilitate comparison between pre- and post-operative findings it is recommended that the loudness of a mitral systolic murmur should always be recorded, not only according to conventional grading but in at least five positions from the mid-clavicular line to the left lung base. After operation there may be little change at the apex but there may be considerable reduction further to the left.

A third heart sound was present in 43 patients before operation and disappeared after it in most; it persisted in 2 patients, despite disappearance of the murmur. An opening snap was heard or recorded in 13 patients, despite the absence of significant mitral stenosis.

Cardiac enlargement was graded as "gross" in 19, "severe" in 17, "moderate" in 11, and "mild" in 4 cases.

An electrocardiographic pattern of left ventricular hypertrophy was infrequent in comparison with its incidence in patients with aortic valvular disease of comparable severity.

Most patients were investigated by cardiac catheterization and angiocardiology and the findings are described. Post-operative studies were carried out 1-12 months after operation in 20 patients.

Of the 51 patients, 7 died as a result of the operation, one from a post-operative nursing accident. There were 2 late deaths.

Three patients, early in the series, sustained cerebral damage at the time of operation but recovered. There has been no such occurrence in the past 18 months.

One patient with moderate stenosis, calcification of the valve, and clot in the left atrium had a fatal

cerebral embolism. Three patients subsequently sustained systemic embolism, 2 weeks, 4 weeks, and 10 months, respectively, after operation. In none had clot been observed. None was under treatment with anticoagulants at the time. Two have recovered almost completely and the third has residual dysphasia. All patients are now treated with anticoagulants on a long-term basis.

The result has been classified as "good" in 36 patients, and "poor" in 6. One patient in the "poor" group had congestive cardiac failure with ascites before operation. Four patients have subsequently been treated by valve replacement owing to recurrence of incompetence.

It is concluded that in the "good" and "moderately good" risk groups the surgical mortality is sufficiently low for operation to be advised earlier than had previously been the case, that long-term results will largely depend on the degree of previous myocardial damage caused by rheumatic myocarditis as well as mitral regurgitation, that early results in the majority of cases are encouraging, and that at present this technique, in suitable cases, is preferable to valve replacement.

REFERENCES

- Anderson, A. M., Cobb, L. A., Bruce, R. A., and Merendino, K. A. (1962). Evaluation of mitral annuloplasty for mitral regurgitation. Clinical and hemodynamic status four to forty-one months after surgery. *Circulation*, **26**, 26.
- Bailey, C. P., O'Neill, T. J. E., Glover, R. P., Jamison, W. L., and Ramirez, H. P. Redondo (1951). Surgical repair of mitral insufficiency. *Dis. Chest*, **19**, 125.
- Bigelow, W. G. (1962). In discussion following Morris *et al.*: Open mitral valvuloplasty. *J. thorac. cardiovasc. Surg.*, **43**, 17-37, p. (36).
- Braunwald, E., and Awe, W. C. (1963). The syndrome of severe mitral regurgitation with normal left atrial pressure. *Circulation*, **27**, 29.
- Broadbent, J. C., and Wood, E. H. (1954). Indicator-dilution curves in acyanotic congenital heart disease. *Circulation*, **9**, 890.
- Brockenbrough, E. C., Braunwald, E., and Ross, J. (1962). Transseptal left heart catheterization. A review of 450 studies and description of an improved technique. *Circulation*, **25**, 15.
- Donald, K. W., Bishop, J. M., Cumming, G., and Wade, O. L. (1955). The effect of exercise on the cardiac output and circulatory dynamics of normal subjects. *Clin. Sci.*, **14**, 37.
- Effler, D. B., Groves, L. K., Martinez, W. V., and Kolff, W. J. (1958). Open-heart surgery for mitral insufficiency. *J. thorac. cardiovasc. Surg.*, **36**, 665.
- Ellis, F. H., McGoon, D. C., Brandenburg, R. O., and Kirklin, J. W. (1963). Clinical experience with total mitral valve replacement with prosthetic valves. *J. thorac. cardiovasc. Surg.*, **46**, 482.
- Glover, R. P., and Davila, J. C. (1957). The treatment of mitral insufficiency by the purse-string technique. *J. thorac. cardiovasc. Surg.*, **33**, 75.

- Guidry, L. D., Callahan, J. A., Marshall, H. W., and Ellis, F. H. (1958). The surgical treatment of mitral insufficiency by mitral annuloplasty. *Proc. Mayo Clin.*, **33**, 523.
- Harken, D. E., Black, H., Ellis, L. B., and Dexter, L. (1954). The surgical correction of mitral insufficiency. *J. thorac. Surg.*, **28**, 604.
- Kay, E. B., and Cross, F. S. (1955). Surgical treatment of mitral insufficiency. *Surgery*, **37**, 697.
- , Nogueira, C., Head, L. R., Coenen, J. P., and Zimmerman, H. A. (1958). Surgical treatment of mitral insufficiency. *J. thorac. cardiovasc. Surg.*, **36**, 677.
- Kitchin, A., and Turner, R. (1964). Diagnosis and treatment of tricuspid stenosis. *Brit. Heart J.*, **26**, 354.
- Korner, P. I., and Shillingford, J. P. (1955). The quantitative estimation of valvular incompetence by dye dilution curves. *Clin. Sci.*, **14**, 553.
- Lillehei, C. W., Gott, V. L., DeWall, R. A., and Varco, R. L. (1958). The surgical treatment of stenotic or regurgitant lesions of the mitral and aortic valves by direct vision utilizing a pump-oxygenator. *J. thorac. cardiovasc. Surg.*, **35**, 154.
- Logan, A., and Turner, R. (1952). The diagnosis of mitral incompetence accompanying mitral stenosis. Review of eleven cases treated surgically. *Lancet*, **2**, 593.
- Marshall, H. W., Woodward, E., and Wood, E. H. (1958). Hemodynamic methods for differentiation of mitral stenosis and regurgitation. *Amer. J. Cardiol.*, **2**, 24.
- Merendino, K. A., and Bruce, R. A. (1957). One hundred and seventeen surgically treated cases of valvular rheumatic heart disease. *J. Amer. med. Ass.*, **164**, 749.
- McGoon, D. C. (1960). Repair of mitral insufficiency due to ruptured chordæ tendineæ. *J. thorac. cardiovasc. Surg.*, **39**, 357.
- Morris, J. D., Sloan, H., Wilson, W. S., and Brandt, R. L. (1962). An appraisal of clinical results in open mitral valvuloplasty. *J. thorac. cardiovasc. Surg.*, **43**, 17.
- Murray, G., Wilkinson, F. R., and MacKenzie, R. (1938). Reconstruction of the valves of the heart. *Canad. med. Ass. J.*, **38**, 317.
- Nichols, H. T. (1957). Mitral insufficiency: Treatment by Polar cross-fusion of the mitral annulus fibrosus. *J. thorac. Surg.*, **33**, 102.
- Osmundson, P. J., Callahan, J. A., and Edwards, J. E. (1958). Mitral insufficiency from ruptured chordæ tendineæ simulating aortic stenosis. *Proc. Mayo Clin.*, **33**, 235.
- Starr, A., and Edwards, M. L. (1961). Mitral replacement: clinical experience with a ball-valve prosthesis. *Ann. Surg.*, **154**, 726.
- Wooler, G. H., Nixon, P. G. F., Grimshaw, V. A., and Watson, D. A. (1962). Experiences with the repair of the mitral valve in mitral incompetence. *Thorax*, **17**, 49.