THE PRE-OPERATIVE ASSESSMENT OF MITRAL-VALVE DISEASE*

V. SCHRIRE, M.Sc., PH.D., M.D., F.R.C.P.E., F.R.C.P. AND C. N. BARNARD, M.D., M.MED., M.S., PH.D., F.A.C.S.,
Departments of Medicine and Surgery, University of Cape Town, CSIR Cardiovascular Pulmonary Research Group, and the Cardiac Clinic, Groote Schuur Hospital, Cape Town

The diagnosis of mitral-valve disease can readily be made at the bedside. Careful palpation and auscultation, together with the electrocardiogram and roentgenogram, generally provide such precise information that an accurate estimate of the haemodynamic state can usually be deduced without recourse to cardiac catheterization and angiocardiography. The latter procedures, however, are of particular value in quantitating the *degree* of stenosis and incompetence, and in measuring the pulmonary resistance. Furthermore, in the presence of multiple valve involvement they provide, in our experience, the only reliable data.

From 1951 until April 1963, 450 patients were seen and studied in the Cardiac Clinic, Groote Schuur Hospital, and referred for mitral-valve surgery. There were 441 closed-heart and 48 open-heart procedures; several patients requiring two or more operations. In this paper we wish to report the pre-operative clinical data and their relation to the findings at surgery. The study is concerned with the problem of mitral-valve disease without significant aortic valve involvement, particularly aortic stenosis.

MATERIAL AND METHODS

The 450 patients in this series have been selected from 2,500 rheumatic patients referred to the Cardiac Clinic for assessment and treatment. All patients were seen by one of us (V.S.) before operation. With few exceptions, surgery was performed at Groote Schuur Hospital. 14% of the patients were catheterized, but the findings are not discussed in this communication.

With the development of cardiac surgery, and increased experience, there has been a rise in the number of patients on whom surgery has been performed (Table I).

TABLE I. MITRAL VALVE SURGERY

	Closed heart	*Open heart*
1951/1952	4	
1953	30	
1954	40	
1955	22	
1956	39	
1957	16	
1958	25	
1959	46	6
1960	68	2
1961	61	14
1962	71	19
1963	65	23
Total	487	64

As cardiac-bypass procedures have improved in our unit[1-5] and prosthetic valves have been developed,[4-6] more patients are now being submitted to mitral-valve surgery. At present practically no patient is too ill for surgery, provided renal function is satisfactory, and any type of mitral-valve deformity can be improved or corrected. The present limitation is not the number of patients suitable for corrective surgery, but the fact that the medical staff, surgical beds and operating time has not kept pace with the development and demand for cardiac surgery.

A full history and examination was performed in every patient, usually repeated on several occasions. Phonocardiography was used in almost every patient for record purposes and for study, but did not influence the selection of cases in any way. It was invaluable in promoting accuracy of auscultation, and was constantly used to check the clinical findings. Electrocardiography included the standard, the unipolar-limb and the praecordial leads V1 to V7. Most of the patients (75%) were screened by one of us (V.S.) in the Cardiac Clinic and a barium swallow was routine. Roentgenograms were taken independently in the radiological department.

The haemodynamic disturbance was classified into four groups according to the findings at surgery:

A. *Pure mitral stenosis* (356 patients) meant narrowing of the mitral valve, without any palpable evidence of mitral incompetence. In 95% of the patients the valve area measured less than 2 sq. cm.[7]

B. *Trivial mitral incompetence* (38 patients), indicated a slight jet felt during systole and regarded by the surgeon to be of no haemodynamic consequence, often described as a 'purr' rather than as a jet. In 72% the valve area was less than 2 sq. cm.

C. *Mixed stenosis and incompetence* (34 patients), implied a significant degree of both stenosis and incompetence; each component varied considerably from case to case, no attempt being made to assess which was dominant. Attempts at quantitation were not possible at surgery. 89% had valve areas between 1 and 3 sq. cm.

D. *Pure mitral incompetence*. In 21 of the 22 patients in whom the valves were inspected during bypass surgery, no stenosis was present.

RESULTS

The patients were analyzed according to age, race and sex, in Table II. There was no racial difference, the incidence of rheumatic heart disease being proportionate to the relative number of patients of each racial group attending the hospital.[8] The youngest patient in the series was 10 and the oldest 62 years. The female dominance of almost 3:1 is in keeping with the world experience. Severe rheumatic-valve disease appears to manifest itself at an earlier age in the non-White races and is probably more lethal. Most patients over the age of 40 were White.

The diagnosis made on clinical grounds and the findings at surgery are shown in Table III.

Of the 356 patients with pure mitral stenosis (group A) the diagnosis was correctly made in 345 (97%), which is in keeping with the experience of others.[9] Errors were made in both directions. Of 8 patients regarded as having pure mitral stenosis, 7 had trivial mitral incompetence (B) and 1 mixed mitral stenosis and incompetence (C). Ten patients regarded as having trivial mitral incompetence

*Presented at the 44th South African Medical Congress, Johannesburg, July 1963.

TABLE II. RACE, AGE AND SEX OF 450 PATIENTS WITH MITRAL-VALVE DISEASE SUBMITTED TO SURGERY

Age (years)	Total ABCD			Group A*						Group B						Group C						Group D					
				Sex			Race			Sex			Race			Sex			Race			Sex			Race		
	No.	M	F	No.	M	F	W	C	B	No.	M	F	W	C	B	No.	M	F	W	C	B	No.	M	F	W	C	B
10—19	53	14	39	43	11	32	6	21	16	5	2	3	—	1	4	2	1	1	2	—	—	3	—	3	1	2	—
20—29	140	41	99	112	27	85	31	64	17	10	8	2	1	7	2	9	4	5	3	3	3	9	2	7	6	1	1
30—39	130	40	90	106	30	76	54	40	12	8	4	4	4	3	1	9	5	4	4	5	—	7	1	6	7	—	—
40—49	89	28	61	65	21	44	45	14	6	12	3	9	8	2	2	10	3	7	8	1	1	2	1	1	2	—	—
50—59	36	9	27	28	8	20	24	2	2	3	—	3	1	2	—	4	1	3	4	—	—	1	—	1	1	—	—
60+69	2	1	1	2	1	1	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Total	450	133	317	356	98	258	161	142	53	38	17	21	14	15	9	34	14	20	21	9	4	22	4	18	17	3	1

* For classification of groups see text

and 1 as having mixed stenosis and incompetence, had pure stenosis.

Of the 38 patients with trivial mitral incompetence (group B), the diagnosis was correctly made in 29 (80%). There were 12 errors. Ten had pure stenosis and 2 mixed

TABLE III. CLINICAL DIAGNOSIS AND SURGICAL FINDINGS

	Group A	Group B	Group C	Group D
	pure MS	MS + triv. MI	MS + MI	pure MI
Surgically proven ..	356	38	34	22
Clinical diagnosis ..	353	41	35	21
Clinically correct ..	345	29	31	21
Clinical errors	B-7 C-1	A-10 C-2	A-1 B-2	—

stenosis and incompetence. For practical purposes groups A and B (both of whom are suitable for closed-heart surgery) can be taken together, i.e. there were 394 such patients, the clinical diagnosis being correct in 391.

Of the 34 patients with mixed stenosis and incompetence, 31 (91%) were confirmed at surgery. There were 4 errors, one patient having pure stenosis, one pure incompetence, and 2 trivial incompetence. All 21 patients diagnosed as pure mitral incompetence had this lesion at surgery.

DISCUSSION

In the following analyses groups A to D are separately considered retrospectively, i.e. on the basis of surgical and not on the clinical diagnoses.

Pre-operative Symptoms

The relation of symptoms to the type of valve disease is shown in Tables IV and V. Careful enquiry was always made

TABLE IV. RELATION OF SYMPTOMS TO TYPE OF MITRAL-VALVE DISEASE PROVEN AT SURGERY

	Group A tight MS	Group B dom. MS + trivial MI	Group C mixed MS + MI	Group D pure MI
	(356)	(38)	(34)	(22)
Rheumatic fever	44	31	61	87
Chorea	6	3	6	0
Emboli	14	13	18	5
AP	14	13	3	9
Left chest pain ..	15	8	9	18
Haemoptysis	49	47	56	36
Fatigue	8	5	6	32
Palpitations	29	16	47	50
Syncope on effort ..	1	0	0	0
SBE	0·3	0	0	27
Pregnancy	18	0	0	0

Figures expressed as percentages.

TABLE V. RELATION OF DYSPNOEA AND DEGREE OF DISABILITY TO TYPE OF MITRAL-VALVE DISEASE PROVEN AT OPERATION

	Group A tight MS	Group B dom. MS + trivial MI	Group C mixed MS + MI	Group D pure MI
	(356)	(38)	(34)	(22)
Orthopnoea	40	42	70	73
PCD	64	60	65	68
Oedema	22	15	60	59
Mild Dyspnoea ..	1	2	3	0
Moderate dyspnoea ..	36	40	19	4
Severe dyspnoea ..	63	58	88	96
Grade 1 disability ..	0·5	0	0	0
Grade 2 disability ..	37	15	17	4
Grade 3 disability ..	57·5	50	35	36
Grade 4 disability ..	5	35	45	60

Figures expressed as percentages

for a history of rheumatic fever, chorea, or equivalents, for emboli, anginal pain, haemoptysis, fatigue, palpitations, syncope on effort, swelling of feet, and for bacterial endocarditis. The effect of pregnancy on the symptomatology was also always noted.

A. *Rheumatic fever.* A history of rheumatic fever was present in a little more than a third of patients with mitral stenosis (groups A and B). A far higher incidence was noted in patients with mixed stenosis and incompetence. In pure mitral incompetence, most patients (87%) had a history of rheumatic fever.

It is worth noting that chorea never occurred with pure mitral incompetence, whereas an incidence of 3·6% was noted in other forms of mitral-valve disease. The severity of the rheumatic process could be assessed by the age at which the patients became aware that they had murmurs, or suffered from valve disease. Most patients with pure mitral incompetence were aware of heart murmurs or heart disease in their 'teens' (average 16 years, range 3 - 45) and symptoms generally dated from an early age. Patients with pure mitral stenosis, on the other hand, were often unaware of any heart disease until severe symptoms requiring surgery had developed, (average age when murmurs were discovered 28, range 6 - 61). The figures obtained for dominant stenosis and trivial incompetence were 27 (6 - 46) and for mixed stenosis and incompetence 30 (6 - 46) respectively.

B. *Emboli.* The incidence of systemic embolism is shown in Table IV. Of the 50 patients with systemic embolism, cerebral involvement was present in 35. Embolism alone was not an indication for surgery in any patient. In some subjects there was a clear association of embolism with the onset of an arrhythmia. Atrial fibrillation was present in 30 of the 50 patients with systemic embolism.

C. *Angina pectoris.* The incidence of chest pain is shown in Table IV. Usually the story was quite typical of angina pectoris in distribution and relation to exertion. Sometimes the pain was atypical but was sufficiently suggestive to be included in this analysis. The pain usually disappeared after valvotomy and was therefore attributed to the valve lesion. It was less common in pure mitral incompetence than in pure mitral stenosis. Left-mammary pain was fairly frequent in all four groups.

D. *Subacute bacterial endocarditis.* This was very uncommon in pure mitral stenosis but occurred in 27% of patients who had pure mitral incompetence.

E. *Pregnancy.* Symptoms occurred in 76 patients during pregnancy, attention often being drawn to the heart for the first time during this state. In 18 patients with pure stenosis, surgery was performed during the pregnant state.

F. *Dyspnoea and oedema.* The incidence of dyspnoea and oedema, and the degree of disability is shown in Table V. Dyspnoea on effort was present in almost all patients, progressive in most, and has been graded as mild, moderate and severe. Patients with orthopnoea and paroxysmal cardiac dyspnoea were classified as severe dyspnoea. In assessing the disability, the method previously described[7] was used. For grade 1 disability, patients were dyspnoeic with strenuous effort, hurrying, going uphill or up steps, but apart from this led reasonably normal lives. Patients with grade 2 disability could not run or hurry. On the level their effort tolerance was reasonable; they had to take hills or steps slowly; intercourse was associated with moderate dyspnoea; they could

cope with the ordinary activities of life, but lived restricted lives. Patients with grade 3 disability could not do their own housework, shopping or indulge in ordinary activity. Stairs were impossible, intercourse very trying. Cases of congestive cardiac failure, in accordance with custom, were put into grade 4. In addition, patients who were totally incapacitated but were not necessarily in congestive cardiac failure at the time of surgery, were also regarded as grade 4. Special attention was paid to the presence of paroxysmal nocturnal dyspnoea. The presence of this symptom put the patient into grade 3, even if the disability during the day was grade 2.

The increased incidence of severe disability, severe effort dyspnoea, paroxysmal nocturnal dyspnoea, orthopnoea and oedema in patients with pure mitral incompetence and mixed stenosis and incompetence is a reflection of case selection rather than the nature of the disease. It has been our practice to select patients for plastic repair or mitral-valve replacement only when severe disability or advanced heart failure was present. Subjects who were thought to be suitable for closed-heart surgery were submitted to operation far earlier, preferably before the development of systemic congestion. No significance can thus be attached to the relative severity of the symptoms in groups C and D in differentiating the 4 types of valve disease.

Conclusion. It can be concluded that the history and the symptoms have little value in deciding which type of mitral-valve disease is present. Certain generalizations, however, can be made. Patients with pure, tight or dominant mitral stenosis have a less frequent history of rheumatic fever, less severe valve damage and more angina pectoris. Patients with significant or gross mitral incompetence complain far more commonly of fatigue and palpitations. They are also more subject to subacute bacterial endocarditis. All patients were limited by dyspnoea. Orthopnoea, paroxysmal cardiac dyspnoea and oedema related more to the stage of the disease than the nature of the valve lesion.

PHYSICAL SIGNS

The examination of the venous pulse was of no value in differentiating the type of valve disease present. Neither the height of the jugular venous pressure nor the type of pulsations were of any help. The peripheral pulse likewise gave no special information, being recorded as small in many patients and normal in the majority. The blood pressure was of no value in diagnosis. The findings are shown in Tables VI and VII.

TABLE VI. RELATION OF FINDINGS ON PALPATION TO TYPE OF MITRAL-VALVE LESION PROVEN AT OPERATION

	Group A tight MS	*Group B dom. MS + trivial MI*	*Group C mixed MS + MI*	*Group D pure MI*
	(*356*)	(*38*)	(*34*)	(*22*)
RV apex	44	34	15	0
Palp. mitral	36	13	3	0
RV lift	70	63	88	70
Palp. P2	33	21	24	45
LV apex	12	34	65	100
LV enlargement	6	10	64	100
Clinical cardiomegaly	14	26	50	100
Systolic thrill	0·3	3	6	27
Diastolic thrill	15	27	35	18
AF	22	40	42	90

Figures expressed as percentages.

Palpation

The relation of findings on palpation to the type of mitral-valve lesion proven at operation, are shown in Table VI. In pure mitral stenosis the apex was most commonly formed by an enlarged right ventricle, which imparted a heaving sensation to the palpating hand. Alternatively the beat was impalpable, being replaced by a 'tap' produced by closure of the mitral valve. In 13% it was regarded as normal in character. The heart was generally not enlarged, and when enlargement was clearly present, it was usually associated with severe pulmonary hypertension, tricuspid incompetence, atrial fibrillation and systemic venous hypertension or with aortic incompetence.

An undue lift in the parasternal area over the outflow tract of the right ventricle was interpreted as right ventricular hypertrophy and was often associated with palpable closure of the pulmonary valves. These findings were regarded as a reflection of significant pulmonary hypertension. The apex beat was thought to be thrusting and left ventricular in type, suggestive of left ventricular hypertrophy, in 12% of the patients. This was generally attributed to displacement of the apex beat by the hypertrophied right ventricle. In half these patients there was associated aortic-valve disease or hypertension. However, occasionally left ventricular hypertrophy, confirmed at surgery, was present for no clear reason. It is possible that such patients had passed from mitral incompetence to mitral stenosis during the natural course of the disease. However, in our experience with a large number of patients observed over many years, conversion of mitral incompetence to pure mitral stenosis has been rare. Alternatively, left ventricular disease, e.g. rheumatic myocarditis, may be responsible.

The sole reason for diagnosing trivial incompetence associated with mitral stenosis (group B) was the presence of a mitral systolic murmur (see below). In other respects the two conditions were indistinguishable.

In pure mitral incompetence on the other hand, the heart was always enlarged, the apex beat being formed by the hyperkinetic, thrusting, over-filled left ventricle, which was readily recognized. In the presence of considerable left ventricular enlargement, however, parasternal pulsation is more difficult to interpret, since it may be produced by displacement forwards of the right ventricle by the enlarged left ventricle. Furthermore, giant left atrial enlargement, which was sometimes associated with mitral incompetence, resulted in pulsation both to the right and to the left of the sternum. Right ventricular enlargement, however, was recorded in most patients.

Where both mitral stenosis and incompetence were present palpation was often rewarding. The heart was enlarged in half the patients; the apex being left ventricular in type in two-thirds of cases. Unfortunately, palpation was not infallible, significant mitral incompetence being found without a thrusting left ventricle.

Conclusion. It can be concluded that palpation is often of great value in determining what type of mitral-valve disease is present. In pure or dominant mitral stenosis the apex beat has a heaving or 'tapping' quality, the heart is usually not enlarged and signs of pulmonary hypertension are often present. A diastolic thrill is common and left ventricular enlargement rare. In mitral incompetence, a thrusting over-filled, over-active left ventricle forms the apex, and the heart is considerably enlarged; thrills are usually systolic and not diastolic. Where mitral stenosis and incompetence are combined, the diagnosis is often difficult. Left ventricular enlargement, however, suggests significant incompetence and right ventricular enlargement, significant stenosis.

Palpation, unfortunately, may be misleading. Significant incompetence may be found in the presence of pure right ventricular enlargement and pure mitral stenosis in the presence of left ventricular enlargement. Furthermore, when tricuspid incompetence and heart failure are present, or aortic incompetence coexists, considerable cardiomegaly may be present in the absence of any mitral incompetence.

TABLE VII. RELATION OF AUSCULTATORY FINDINGS TO TYPE OF MITRAL-VALVE LESION PROVEN AT OPERATION

	Group A tight MS	*Group B dom. MS + trivial MI*	*Group C mixed MS + MI*	*Group D pure MI*
	(*356*)	(*38*)	(*34*)	(*22*)
Apical SM	36	100	100	100
MDM	95	90	87	87
PSM	74 (22)	51 (38)	19 (42)	0 (90)
Opening sound	85	58	26	33
Closing sound	50	37	10	5
3 HS	5	10	20	87
Pulmonary ejection sound	25	18	8	0

Figures expressed as percentages
Figures in parentheses = patients with atrial fibrillation.

AUSCULTATION

(a) *Signs of mitral-valve disease.* Auscultation played the major role in diagnosis. The relation of the auscultatory findings to the type of mitral-valve lesion proven at operation are shown in Table VII. If, at first, attention is confined to the

apex, a good idea of the state of the mitral valve could usually be obtained. The importance of systole has been described elsewhere.[10] In brief, systole was silent in 286 of the 450 patients (Table VIII). Pure mitral stenosis was found in 280, trivial stenosis in 5, therefore only one important error in diagnosis was made. When a systolic murmur was heard at the apex it could generally be divided into one of three groups.

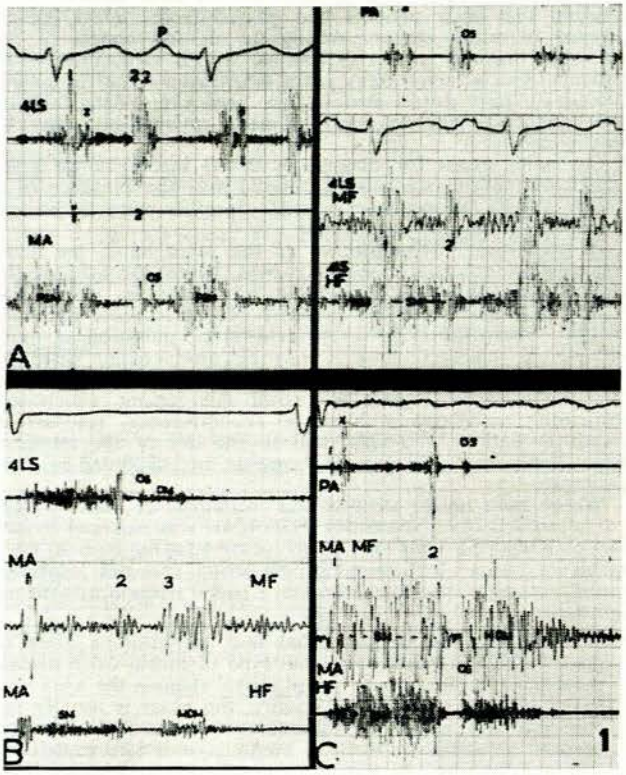


Fig. 1 (*A*). Tracing from a patient with tight mitral stenosis, tricuspid incompetence and pulmonary hypertension. At the mitral area (MA) the presystolic murmur (psm) ends at the accentuated and delayed first sound (1). The opening snap (os) is separated from the second sound by 0·05 sec. and there is a distinct interval between it and the onset of the diastolic murmur. At the tricuspid area (4LS) both the high frequency (HF) and medium frequency (MF) tracings show the pansystolic murmur of tricuspid incompetence and the mitral diastolic murmur which has radiated to the tricuspid area. The pulmonary ejection sound (x) is best shown at the pulmonary area. (*B*). Tracing from a patient with mitral stenosis and incompetence, tricuspid stenosis and incompetence. The pansystolic murmur of mitral incompetence is shown at the mitral area and that of tricuspid incompetence at the tricuspid area. The tricuspid diastolic murmur commences after the opening snap and distinctly before the mitral diastolic murmur, which commences with the left ventricular filling sound (3). Atrial fibrillation is present. (*C*). Tracing from a patient with mitral incompetence. The pansystolic murmur of mitral incompetence extends a little beyond the aortic second sound. The mid-diastolic murmur is relatively short. Atrial fibrillation is present. The pulmonary ejection click and opening snap are shown at the pulmonary area.

Firstly, an insignificant or immaterial systolic murmur, often short and soft (grade 1 to 2/6 in intensity[11]) was present in 30 patients, 28 of whom had pure mitral stenosis and 2 trivial incompetence. Secondly, tricuspid systolic murmurs radiating to the apex (see below) could usually be readily differentiated from mitral incompetence. This was present in 37 patients. Thirdly, a pansystolic murmur of mitral incompetence was present in 97 patients. Severe mitral incompetence was recognized by a loud (grade 4 or more) systolic murmur radiating widely both medially and laterally and into the back. Where mitral stenosis and incompetence were combined, the diagnosis of the severity of each component was extremely difficult and the greatest errors occurred in this group. In general, loud murmurs were associated with severe incompetence and soft murmurs with slight or no incompetence. However, too much overlap occurred to make this a reliable basis for diagnosis (see Table VIII).

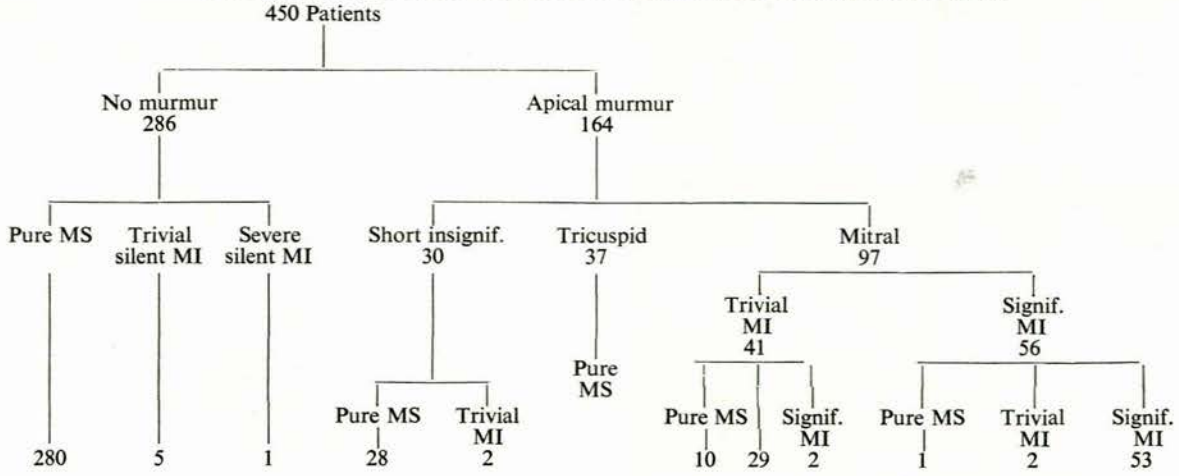
An apical mid-diastolic murmur is one of the most constant findings in mitral-valve disease and was rarely absent, whatever the pathology, whether pure mitral stenosis, pure mitral incompetence or combined stenosis and incompetence were present. In pure and dominant mitral stenosis, however, it was generally loud (grade 1-6%, grade 2-47%, grade 3-46% and grade 4-1%) whereas with pure mitral incompetence it was soft (26% grade 1, 74% grade 2), and in mixed mitral stenosis and incompetence it was 6%, 38% and 56% respectively. A presystolic murmur was not heard in pure mitral incompetence but only two patients were in sinus rhythm.

An opening snap reflects the mobility of the anterior leaflet of the mitral valve. Since, in pure mitral stenosis, gross mitral-valve deformity and valve destruction is not the rule, an opening snap is frequently present (Table VII). Mixed mitral stenosis and incompetence usually implies more severe valve destruction, fibrosis and immobility so that an opening snap is usually absent. Only one-third of the patients with mitral incompetence had clear opening snaps. Similarly, a loud first sound occurred in at least half the patients with pure mitral stenosis, whereas in incompetence or dominant stenosis and incompetence, the loud systolic murmur started with the first heart sound and often replaced it. A third heart sound was heard in only 5% of the patients with mitral stenosis, whereas it was far more frequent in pure incompetence and in mixed stenosis and incompetence. In the group with pure mitral stenosis the third sound was always due to right ventricular filling, whereas in mitral incompetence it was due to left ventricular filling. Differentiation was made clinically by the situation of the third heart sound. When the third heart sound is right ventricular in origin it is heard maximally or solely at the fourth left space. When it arises in the left ventricle it is heard at the apex and radiates both medially and laterally.

(b) *Signs of pulmonary hypertension* (Fig. 1A). Some degree of pulmonary hypertension is nearly always present in patients with mitral-valve disease bad enough to come to surgery. This is usually passive, i.e. left-atrial, pulmonary-venous, pulmonary-capillary and pulmonary-arterial pressures are all proportionately elevated. In an appreciable but relatively small number of patients, a disproportionate elevation in pulmonary-arterial pressure develops owing to changes in the pulmonary resistance vessels. This protects the pulmonary capillaries from sudden surges in pressure but throws additional strain on the right ventricle. Severe pulmonary hypertension is reflected clinically by the marked lift over the outflow tract of the right ventricle and the palpable pulmonary-valve closure. On auscultation it is recognized by the accentuated pulmonary second sound and the pulmonary ejection click. Severe pulmonary hypertension reduces flow across the mitral valve so that the mitral diastolic murmur is often soft or absent. The opening snap may be similarly affected. An early diastolic murmur (Graham-Steell murmur) owing to pulmonary incompetence may be heard, and was found in 14 patients. We are aware that the existence of such a murmur has been questioned,[12] but we have no doubt that it occurs. We have even diagnosed a patient as having aortic incompetence and found no aortic incompetence at bypass surgery, with disappearance of the murmur after correction of the mitral incompetence. Pulmonary incompetence was diagnosed only when the early diastolic murmur was localized to the pulmonary area with a palpable second sound (diastolic shock) and all the clinical, electrocardiographic and radiological hallmarks of pulmonary hypertension were present. Severe pulmonary hypertension (pulmonary-arterial pressure more or less at systemic level) was found at cardiac catheterization in patients with pure stenosis or mixed stenosis and incompetence, but not in pure mitral incompetence. A pulmonary ejection click was likewise not heard in mitral incompetence but was common in mitral stenosis (Table VII and Fig. 1).

(c) *Signs of tricuspid-valve disease* (Figs. 1A, 1B and 2). It may not be appreciated how frequently the tricuspid valve is involved in rheumatic heart disease. Necropsy study shows at least a 25% incidence. Tricuspid incompetence is far commoner than stenosis. Tricuspid incompetence is certainly commoner clinically than pathologically, because of the frequency of functional incompetence, i.e. incompetence that is transient and disappears with bed-rest and therapy. It is freely admitted

TABLE VIII. THE RELATION OF THE APICAL SYSTOLIC MURMUR TO MITRAL VALVE DISEASE



that the distinction between functional and organic tricuspid incompetence is artificial, and in Table IX we have analyzed the incidence of tricuspid incompetence (transient and constant) in our series.

Tricuspid incompetence can be recognized by the systolic pulsation in the jugular veins and liver. Although occasionally there may be no murmurs, this is not common and a tricuspid

TABLE IX. ASSOCIATED VALVE DISEASE

	Group A tight MS	*Group B dom. MS + trivial MI*	*Group C mixed MS + MI*	*Group D pure MI*
	(356)	*(38)*	*(34)*	*(22)*
TI	24	26	12	18
TS	5	9	3	4
AI	37	31	35	23
AS	4	5	6	0

Figures expressed as percentages

systolic murmur was found in 105 patients. The murmur is quite characteristic, being pansystolic, best heard in the fourth left intercostal space and xiphisternal area, and increasing in intensity on inspiration. Sometimes it has a peculiar 'honking' character[13] (Fig. 2). With marked right ventricular dilatation the murmur becomes widespread and easily heard at the apex, so that it may be confused with mitral incompetence. One of

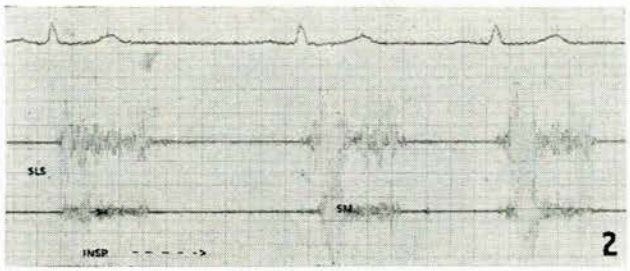


Fig. 2. The effect of inspiration on the systolic murmur of tricuspid incompetence is shown. The high frequency ('honking') component which occupies the first third of systole is markedly accentuated.

our patients masqueraded as gross mitral incompetence for years before it was appreciated how widely the murmur could radiate and how loud it could be at the apex. The murmur can be differentiated from that of mitral incompetence by the fact that it is loudest at the tricuspid area and tails off rapidly as the axilla is reached, being rarely heard posteriorly. Tricuspid murmurs are particulaly common in advanced heart disease, in patients with marked cardiomegaly, pulmonary hypertension, atrial fibrillation and congestive cardiac failure. After energetic treatment for heart failure, the murmurs frequently disappear and are attributed to functional incompetence. Tricuspid stenosis was encountered in 8 patients, requiring both tricuspid and mitral valvotomy. The incidence of associated valve disease is shown in Table IX.

(d) *Signs of aortic-valve disease.* The diagnosis of aortic incompetence is usually made on murmurs and a full pulse pressure. The incidence of an early diastolic murmur of aortic incompetence is shown in Table IX. The murmur is high-pitched, best heard in the third and fourth left spaces and radiates along the left sternal border to the apex. In 90% of the patients the murmur was unassociated with any change in pulse pressure. Before the advent of open-heart surgery, closed-heart valvotomy was advised even in the presence of aortic incompetence producing a blood pressure of 95/30 mm.Hg. Nowadays, however, patients with gross aortic incompetence would be advised bypass surgery.

Aortic stenotic murmurs can be recognized because they are short, and stop before the second sound. The murmur is rough, and is best heard to the left and right of the sternum radiating to the neck and apex, but occasionally it is loudest at the apex, and it may in fact be confined to the apex. It can usually be differentiated from mitral incompetence but in cases of mixed aortic and mitral-valve disease differentiation of murmurs is extremely difficult. These cases have not been included in this analysis.

Electrocardiography

The electrocardiograms (Figs. 3, 4) were all analyzed for rate, rhythm, PR and QRS intervals, P and QRS complexes, mean electrical axis in the frontal plane and the direction of the loop. The chest leads were especially examined for the heights of the Q, R and S waves in V1 and V6. Using these data electrocardiographic diagnoses were made as follows: For left-atrial hypertrophy there had to be a P wave of 3 mm. or more in height in any lead (usually lead 2) or more than 0.10 second in duration, or when bifid, more than 0.04 second between peaks; for right atrial hypertrophy[14] a tall peaked P wave 2·5 mm. or more in height in the limb leads, 3 mm. or more in the chest leads; for left ventricular hypertrophy voltage change (R in leads V5 or V6 and S in V1 exceeding 35 mm., R in V5 or V6 exceeding 26 mm., R in aVL more than 11 mm. and R in aVF more than 20 mm.), ST-segment depression, T or U-wave inversion; and for right ventricular hypertrophy, right axis deviation, an R/S ratio in V1 exceeding 1, an R/S ratio in V6 less than 1, ST-segment depression and/or T-wave inversion beyond V1 and delayed onset of the ventricular activation time in V1 to 0·03 second or more.[13]

Rhythm. The incidence of atrial fibrillation (Table VI) has already been discussed. If ECGs were available before the onset of the arrhythmia these were analyzed in preference, hence the discrepancy between Tables VI and X. Other rhythm disturbances encountered were atrial and ventricular ectopics, supraventricular tachycardia, a-v dissociation and nodal rhythm. Atrial flutter was included in atrial fibrillation.

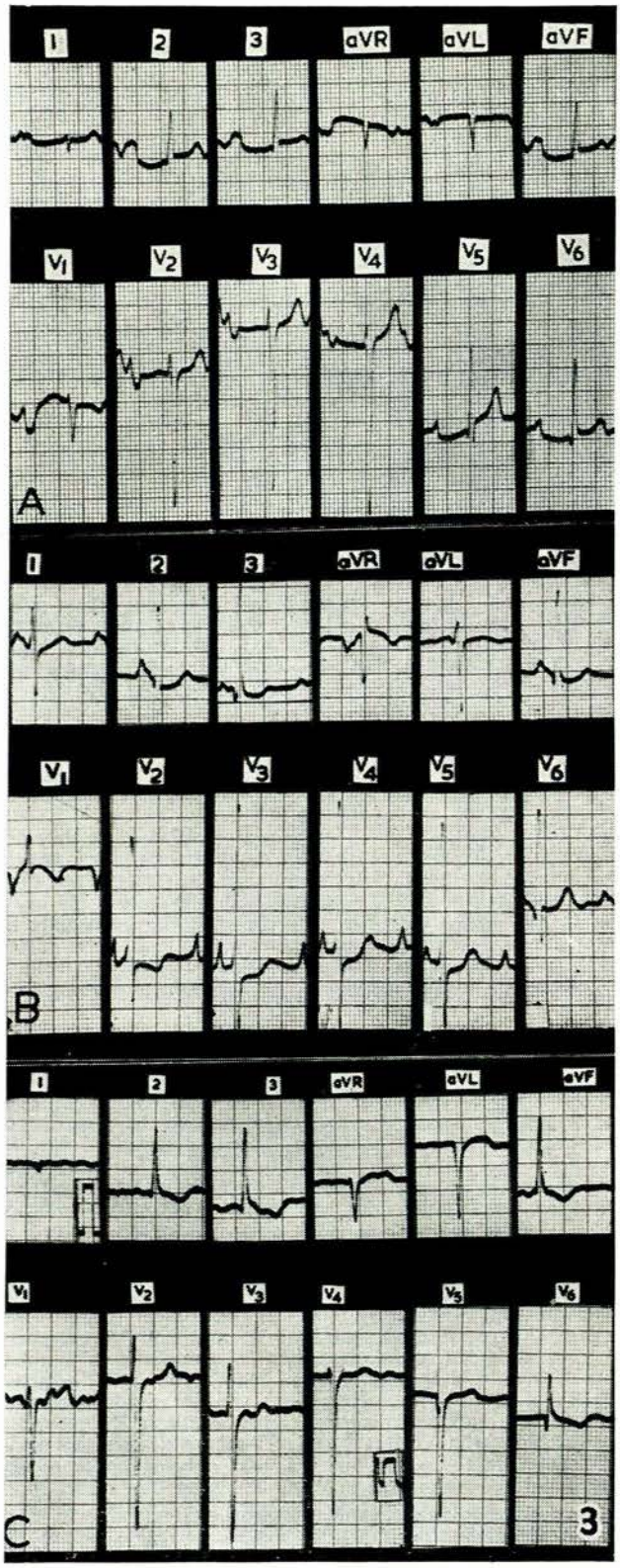


Fig. 3 (*A*). 'Mitral' P waves owing to left-atrial hypertrophy are well shown in a young boy with mitral stenosis. The P-R interval is prolonged to 0.38 sec. Right axis deviation is present but there is no abnormal ventricular dominance. The high voltage in the chest leads is attributed to a thin chest wall. (*B*). Severe pulmonary hypertension was present responsible for the right-ventricular hypertrophy. Biatrial hypertrophy is present (*C*). Atrial fibrillation, digitalis effect and left ventricular hypertrophy are present in this tracing from a patient with pure mitral incompetence.

P-R Interval. This was usually normal. It was prolonged beyond 0·21 second in 23 patients, some of whom had had no digitalis.

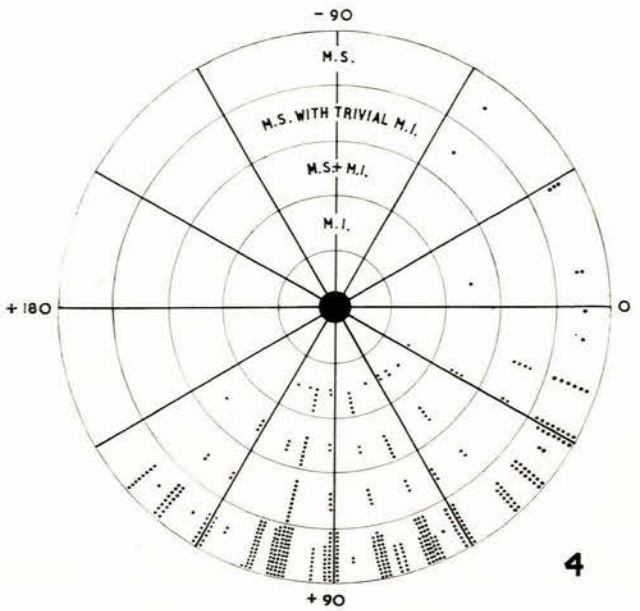


Fig. 4. The mean axes in the frontal plane have been plotted in patients with pure mitral stenosis, stenosis with trivial incompetence, mixed stenosis and incompetence and pure incompetence. A wide range is shown in all groups; most falling between +20° and +140°.

P Waves. The incidence of left atrial hypertrophy is shown in Table X. In the absence of atrial fibrillation, atrial hypertrophy was usually present, whatever the nature of the valve lesion. Usually the left atrium was enlarged, occasionally the right (Fig. 3). An appreciable number of patients in all 4 groups had no evidence of atrial hypertrophy.

TABLE X. ANALYSIS OF ELECTROCARDIOGRAPHIC DIAGNOSES IN MITRAL-VALVE DISEASE

	Group A tight MS	*Group B dom. MS + trivial MI*	*Group C mixed MS + MI*	*Group D pure MI*
	(*356*)	(*38*)	(*34*)	(*22*)
AF	17	40	35	80
Left atrial hypertrophy	60	53	26	14
Right atrial hypertrophy	5	8	6	0
Mild right ventricular hypertrophy	18	21	27	0
Severe right ventricular hypertrophy	14	18	12	4
Incomplete RBBB ..	8	8	12	4
Left ventricular hypertrophy	0·6	2	15	40
Digitalis	—	—	18	60
Normal	10	5	3	0

Figures expressed as percentages.

QRS. The QRS-interval varied from 0·03 to 0·10 seconds with a mean of 0·06 seconds, and no significant difference occurred in the 4 groups. The mean electrical axis in the frontal plane in all patients is shown in Fig. 4. A wide scatter was found in all types of mitral-valve disease. The loop was clockwise in 89 - 95% of the patients.

The incidence of right and left ventricular hypertrophy is shown in Tables X and XI. In Table X the interpretation of the tracings are analyzed using the above criteria. In the presence of digitalis, however, ST-segment depression in the left ventricular surface leads must be interpreted with care. The incidence of left ventricular hypertrophy is thus recorded as 40% in group D. In all probability, almost all these patients had left ventricular hypertrophy.

In Table XI the actual ratio of the Q, R and S waves in V1 and V6 are shown. An rS or QS wave in V1 and an R or qR in V6 can be regarded as normal. An R, rR or qR wave in V1 has been regarded as severe right ventricular hypertrophy. A dominant R (Rs) or equal R and S waves (RS) in V1 has been regarded as mild right ventricular hypertrophy.

Similarly an R or qR complex in V6 is normal, an rS or RS is usually encountered in right ventricular hypertrophy and an Rs or qRs is probably normal. Incomplete right bundle-branch block probably indicates right ventricular hypertrophy and not a conduction disturbance.

TABLE XI. ANALYSIS OF Q, R AND S WAVES IN VI AND V6

		Group A tight MS (356)	*Group B dom. MS + trivial MI* (38)	*Group C mixed MS + MI* (34)	*Group D pure MI* (22)
VI	rS or QS	46	47	50	78
	R, rR, qR, Rs, RS	43	40	35	9
	rSr, 'rsR', qRs ..	10	13	15	13
V6	R, qR	40	42	50	73
	rS, RS	29	16	23	9
	qRs, Rs	31	42	26	18

Figures expressed in percentages.

From Tables X and XI it can be seen that ECG evidence of right ventricular hypertrophy is common and more or less the same in groups A, B and C, left ventricular hypertrophy being rare. In group D (pure mitral incompetence) right ventricular hypertrophy is rare and left ventricular hypertrophy common.

Conclusions. The value of electrocardiography can be summarized as follows: The ECG provides evidence of chamber enlargement and of pulmonary hypertension, but is of no value in differentiating mitral stenosis from combined stenosis and incompetence, and only of slight value in differentiating pure mitral incompetence. Left atrial hypertrophy is common to all types of mitral-valve disease. Right ventricular hypertrophy is rare in mitral incompetence and common in all other forms; the reverse being true for left ventricular hypertrophy.

TABLE XII. THE RELATION OF THE ROENTGENOGRAPHIC FINDINGS TO THE TYPE OF VALVE LESION*

	Pure MS Group A	*MS trivial MI* Group B	*mixed MS & MI* Group C	*Pure MI* Group D
Slight to moderate left atrial enlargement ..	96	85	78	47
Severe or gross left atrial enlargement ..	2·4	4	22	53
Calcified valves ..	15	26	30	7
Left ventricular enlargement	1·5	4	21	100

*Expressed as percentages.

Radiology

The value of radiology (Fig. 5) in diagnosis will be reported in a separate study. In Table XII the results obtained on screening are shown. Few patients had no left atrial enlargement; thus normal hearts were rarely found and these were only in patients with pure mitral stenosis. The size of the left atrium tended to vary with the degree of mitral incompetence and severe or gross enlargement occurred mainly with pure or dominant mitral incompetence as did left ventricular enlargement. Calcified mitral valves were uncommon in pure incompetence and commonest in mixed stenosis and incompetence. The heart was normal in size only in pure mitral stenosis.

Conclusions. Pure mitral incompetence and pure mitral stenosis without cardiomegaly, could generally be recognized radiologically. When mitral incompetence was present the

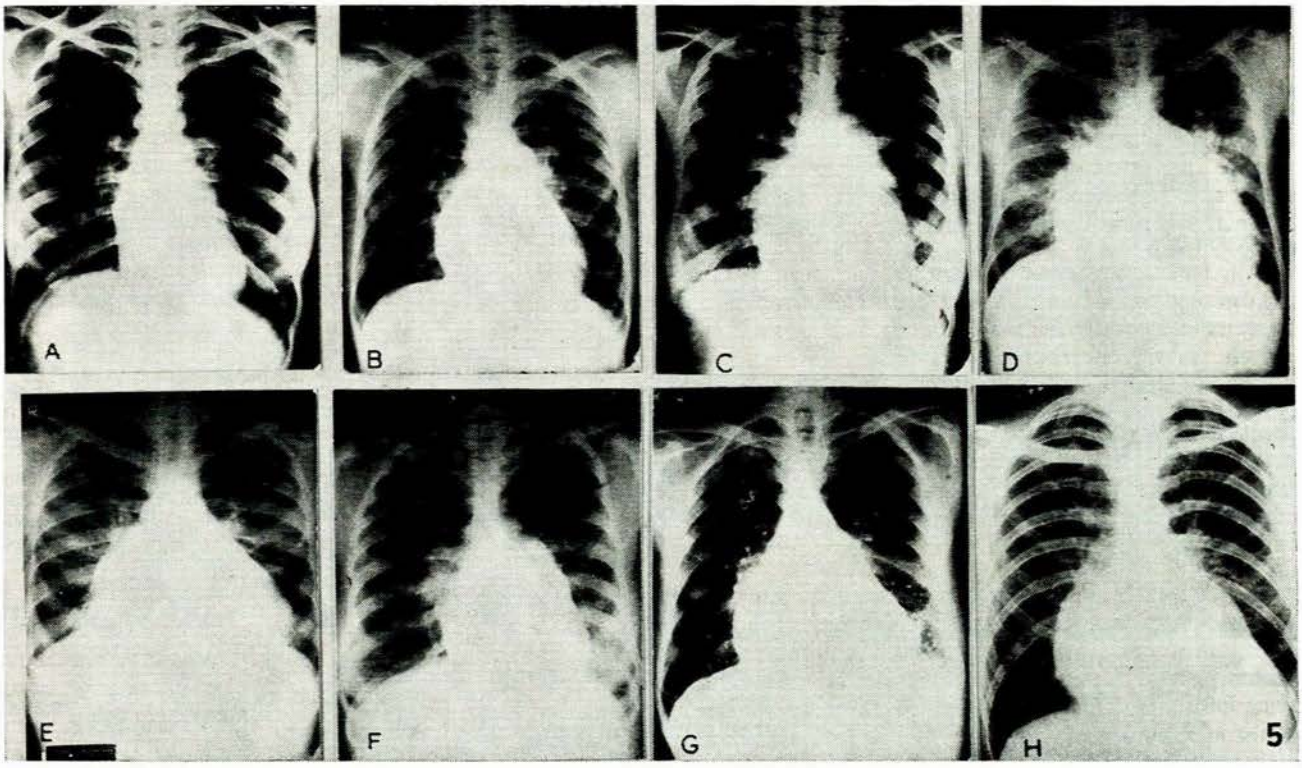


Fig. 5 (*A*). Pure tight mitral stenosis with a normal cardiothoracic ratio but left atrial enlargement and hilar congestion. (*B*). Pure tight mitral stenosis with pulmonary hypertension resulting in pulmonary arterial and right-sided chamber enlargement. (*C*). Pure tight mitral stenosis with pulmonary hypertension and tricuspid incompetence resulting in a hypoplastic aorta, large pulmonary arterial segment, enlargement of the left atrial appendage and the right atrium. (*D*). Pure mitral stenosis with gross organic tricuspid incompetence and congestive failure producing tremendous enlargement of the right atrium and ventricle. Marked hilar congestion is present. (*E*). Dominant mitral stenosis, trivial incompetence, tricuspid stenosis and incompetence and chronic congestive failure in a patient who masqueraded as mixed mitral stenosis and incompetence for years, until cardiac catheterization and angiocardiography revealed that most of the cardiomegaly was right-sided. Hilar congestion is marked. (*F*). Mixed mitral stenosis and incompetence. (*G*). Mixed mitral stenosis and incompetence and tricuspid incompetence with ossified haemosiderosis. (*H*). Pure mitral incompetence with aneurysmal left atrial and marked left ventricular enlargement. Pure mitral stenosis and pure mitral incompetence can readily be recognized. When pulmonary hypertension, tricuspid incompetence and cardiac failure are superimposed, pure mitral stenosis cannot readily be distinguished radiologically from other forms of mitral-valve disease.

degree of incompetence could not be assessed radiologically and this condition could not be differentiated from mitral stenosis particularly when heart failure or associated valve disease was present (Fig. 5).

SUMMARY AND CONCLUSIONS

The pre-operative findings in 450 patients who were subjected to cardiac surgery have been analyzed in order to correlate the clinical data (including ECG and radiological) with the haemodynamic disturbances found at surgery. In 356 of these subjects pure mitral stenosis was present, in 38 trivial mitral incompetence coexisted, 34 had mixed stenosis and incompetence and 22 pure incompetence.

Females were far more commonly affected than males and the age ranged from 10 to 62 years. Although there was no particular racial predominance, non-Whites appeared to be more severely affected at an earlier age.

The history was of little assistance in separating the type of mitral-valve deformity in any particular case. As a whole, patients with pure or dominant mitral stenosis had a less frequent history of rheumatic fever, less severe valve damage and more angina pectoris. Patients with pure mitral incompetence generally had recurrent attacks of rheumatic fever (not chorea) and were aware of valve defects in their teens. They complained more readily of fatigue and palpitations and were peculiarly liable to attacks of bacterial endocarditis.

Palpation was of great value. A normal-sized heart with palpable mitral-valve closure and a lift over the right ventricle indicated mitral-valve obstruction. A thrusting hyperkinetic left ventricle associated with a large heart indicated mitral regurgitation. However, pure mitral stenosis and mixed stenosis and incompetence could usually not be distinguished when the heart was enlarged, particularly in the presence of atrial fibrillation and heart failure. Occasionally the apex appeared to be left ventricular in type in the absence of mitral incompetence, and it was not exceptional to find no evidence of left ventricular enlargement in cases of mixed stenosis and incompetence.

If an apical systolic murmur was absent, pure mitral stenosis was present with few exceptions. Short, soft apical systolic murmurs were generally of no account. Tricuspid systolic murmurs radiating to the apex could be recognized by their radiation and response to inspiration. A mitral systolic murmur was recognized by its duration, radiation and quality. As a rule, soft murmurs meant trivial incompetence, and loud murmurs significant incompetence, but, if one relied on this, serious errors could arise.

Mitral diastolic murmurs were common, whatever the haemodynamic disturbance, tending to be loudest and longest in pure stenosis. An opening snap was present in most patients with pure stenosis. It tended to disappear where valve deformity and destruction was great, as in mixed disease and pure mitral incompetence. A left ventricular filling sound was heard only in mitral incompetence.

The association of pulmonary hypertension, tricuspid and aortic-valve disease significantly altered the auscultatory signs and this has been discussed. Severe pulmonary hypertension was usually the result of obstruction alone or obstruction with regurgitation, and was rare in pure incompetence. Tricuspid-valve disease occurred in approximately 25% of cases. Aortic incompetence, usually of no haemodynamic significance was frequent and aortic stenosis was rare, chiefly owing to case selection in this series.

Electrocardiography was helpful only insofar as it provided evidence of chamber enlargement and pulmonary hypertension. Left atrial and right ventricular hypertrophy was found in every form of mitral-valve disease. Left-ventricular hypertrophy however was overwhelmingly associated with mitral incompetence. In the presence of digitalis (most patients were on digitalis at this stage of their disease) interpretation of ST-T-wave changes as an expression of left ventricular hypertrophy was difficult.

Radiology was useful in confirming the diagnosis of mitral-valve disease. Left atrial enlargement was generally present, and roughly speaking the larger the chamber the more likely was the diagnosis of incompetence, so that aneurysmal enlargement was almost always associated with gross incompetence. Mitral-valve calcification indicated mitral stenosis or mixed valve disease, being rare in pure incompetence. Radiology did not help in differentiating pure stenosis from stenosis with incompetence.

In conclusion, pure mitral stenosis and pure mitral incompetence can usually be diagnosed pre-operatively with relative ease and a high degree of accuracy. This means that the majority of patients who require surgery can be referred for closed- or open-heart surgery respectively on clinical grounds. In pure mitral incompetence valve replacement is generally not necessary. The signs of mixed stenosis and incompetence, however, may be indistinguishable from those of dominant stenosis. Even when mixed disease is correctly diagnosed it is often impossible to determine which is dominant. This is of practical importance at the present moment, because dominant stenosis can still be dealt with by closed-heart surgery, whereas the open-heart approach is required when incompetence is significant and valve replacement is generally required. It is therefore our current practice to investigate by cardiac catheterization and angiocardiography any patient thought to have mixed stenosis and incompetence in whom surgery is contemplated.

We wish to thank the medical staff of Groote Schuur Hospital and the Cardiac Clinic for their cooperation in referring their patients; and Dr. J. G. Burger, the Medical Superintendent, for permission to publish. Our special thanks are due to our surgical colleagues, Drs. W. L. Phillips, R. P. Hewitson, D. Casserley, R. Frater and G. S. Muller Botha, for their reports on their findings at surgery. The City Council of Cape Town and the Council for Scientific and Industrial Research have supported us financially, for which we express our thanks.

REFERENCES

1. Barnard, C. N., Phillips, W. L., De Villiers, D. R., Casserley, R. D., Hewitson, R. P., Van der Riet, R. L. and McKenzie, M. B. (1959): S. Afr. Med. J., **33**, 789.
2. Barnard, C. N., McKenzie, M.B. and Schrire, V. (1961): Brit. J. Surg., **48**, 655.
3. Barnard, C. N. and Schrire, V. (1961): Postgrad. Med. J., **37**, 666.
4. (a) Barnard, C. N., Goosen, C. C., Holmgren, L. V. and Schrire, V. (1962): Lancet, **2**, 1087.
   (b) *Idem* (1962): S. Afr. Med. J., **37**, 97.
5. Barnard, C. N. and Schrire, V. (1963): Surgery, **54**, 302.
6. Barnard, C. N., Schrire, V. and Goosen, C. C. (1963): Lancet, **2**, 856.
7. Schrire, V., Vogelpoel, L., Phillips, W. L. and Nellen, M. (1955): S. Afr. Med. J., **29**, 1108.
8. Schrire, V. (1964): *Ibid.*, **38**, 488.
9. Lewis, B. M., Gorlin, R., Houssay, H. E. J., Haynes, F. W. and Dexter, L. (1952): Amer. Heart J., **43**, 2.
10. Schrire, V. (1964): *Ibid.* (in the press).
11. Levine, S. A. (1933): J. Amer. Med. Assoc., **101**, 436.
12. Brest, A. N., Udhoji, V. and Likoff, W. (1960): New Engl. J. Med., **263**, 1229.
13. Schrire, V. (1963): *Clinical Cardiology*. London: Staples Press.
14. Kilpatrick, J. L. (1951): Brit. Heart J., **13**, 309.