AORTIC STENOSIS

AN EVALUATION OF RESULTS OF SURGERY IN 20 CASES

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The surgical relief of aortic stenosis has been associated with far greater difficulties than that of any other stenotic valvular lesion of the heart. The essential problem is to relieve the obstruction and restore valve action without producing significant aortic regurgitation. The first practical surgical method to correct the commissural fusion of aortic stenosis was evolved by Bailey and his associates in 1950.[1] This raised great hopes for the future, but during the past few years these have been considerably dampened by the realization that at least in the earlier series an unusually high operative mortality and morbidity attended this operation.

The indications and contra-indications for aortic commissurotomy have not been clearly defined, and at the present stage of surgical effectiveness it is hard to answer the question how patients should be selected for surgery. The published literature to date still leaves much to be desired in this respect. It was therefore decided to review all the cases operated upon in Johannesburg and try to solve some of these problems.

In the present study an analysis is made of the clinical and operative findings and the subsequent course in 20 patients with aortic stenosis. Of these, 19 patients were operated upon in Johannesburg by three of us (L.F., D.A., G.R.C.) and 1 patient was operated upon by Sir Russell Brock in London.

CASE MATERIAL

Table I summarizes the sex, age, valvular lesions, duration of the follow-up period and clinical features of these 20 cases. There were 11 males and 9 females. The average age was 35·4 years (range 16-60 years). The lesion was pure aortic stenosis in 8 cases, and in 5 an additional element of aortic regurgitation was present which was considered to be haemodynamically insignificant. The remaining 7 patients had combined aortic and mitral stenosis. The aetiology of the valvular lesion was considered to be rheumatic in 18 patients and congenital in 2 cases (cases 6 and 9). The follow-up period in the survivors has ranged from 6 to 28 months (average 19·2 months). Two patients (cases 6 and 8) were considered to have associated severe coronary atherosclerosis. Case 8 had chronic bronchitis and emphysema for many years

before operation. One patient (case 19) had permanent atrial fibrillation, and all the rest were in sinus rhythm.

METHODS

Clinical Aspects

Clinical assessment included a full clinical examination with electrocardiography, radiology including fluoroscopic examination, and phonocardiography. The functional status of each case was based on criteria given by Baker *et al.*[2] Electrocardiographic evidence of left ventricular enlargement was arbitrarily divided into 5 grades based on the changes described by Sokolow and Lyon[3] in the left chest leads or their limb equivalents: Grade 0=Normal. Grade 1=R wave >26 mm. in V5 or V6, or 20 mm. in AVL, or deep S waves in V1 (>15 mm.) and V2 (>26 mm.); ST segment and T wave normal. Grade 2= tall R wave with reduced T wave in left chest leads or their limb equivalents. Grade 3= downward deviation of ST segments. Grade 4=downward sloping ST segments or inverted T waves.

The symptomatology, physical signs, electrocardiographic and radiological findings are set out in Table I. It will be noted that in 4 patients frank congestive failure was present before operation.

Criteria for Diagnosis

The criteria for diagnosis included (1) the presence of a harsh crescendo-decrescendo systolic murmur stopping short of the 2nd heart sound at the base or apex of the heart (confirmed by phonocardiography in most cases), (2) a small volume and plateau quality in the carotid pulse, with a rough systolic thrill, (3) electrocardiographic evidence of left ventricular enlargement, and (4) X-ray evidence of post-stenotic dilatation of the first part of the aorta.

In the group of cases with combined aortic stenosis and incompetence, the regurgitation was considered insignificant by virtue of the absence in each case of (1) a bisferious pulse or a wide pulse pressure, (2) a carotid shudder, (3) a radiologically prominent aortic knuckle, and (4) increased cardiac pulsations on fluoroscopic examination.

The aortic lesion in cases 6 and 9 was considered to be congenital in origin because in the latter case a harsh praecordial systolic murmur had been heard since early infancy, while in the former multiple congenital stigmata were associated with the heart lesion. In neither was there at any time a history suggestive of rheumatic fever.

The severity and operability of the mitral valve lesion was assessed in the usual manner.[4]

The selection of cases for surgery was based on the demonstration of clinically significant aortic stenosis associated with major disability or of progressive electrographic evidence of left ventricular enlargement. Congestive cardiac failure was well controlled in the 4 cases mentioned previously (Table I) before any surgery was undertaken. In the cases where predominant disability was due to the mitral valve lesion, the decision to operate on the aortic stenosis rested on the magnitude of the ventricular-aortic pressure gradient measured after mitral valvotomy was performed.

The results of operation were judged mainly by subjective improvement in the patient's functional capacity and amelioration of such symptoms as syncope and angina. The results were classified into 4 subdivisions—Markedly improved, Improved, Same and Worse. Objective criteria of improvement were gauged largely on physical signs, electro-

TABLE I. ANALYSIS OF PRE OPERATIVE CLINICAL DATA IN 20 CASES

					Symptoms				Signs								ECG	X-Rays			
CN	S	A	L	FU	Sy	An	PND	FC	BP	RPC	CT	CPV	SSB	SSM	EDM	CCF	LVH	CTR	LV+	PDA	AK	AVC
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23
1	F	18	AS	14	+	+		II	100/70	SR	+	S	Sc	4	Ab		3	42			S	
2	F	29	AS	26	+	+		II	88/64	SR	+	S	Loud	4	Ab		1	42			Z	
3	M	34	AS	11		+		II	106/72	SR	+	S	Soft	3	Ab		2	45		+	Z	
4	F	37	AS	Died		+		III	110/90	SR		S	N	3	Ab			45			Z	
5	F	39	AS	Died		+	+	III	85/50	N	+	S	Loud	5	Ab		3	58	++	+	S	
6	M	44	AS, C	8		+		II	125/100	SR		S	Soft	3	Ab		1	60	++		Z	
7	M	48	AS	Died		+	+	II	120/95	SR	+	S	Soft	3	Ab		3	48	++	+	Z	
8	M	48	AS	8		+	+	II	110/80	SR		S	N	5	Ab		4	43	++		S	
9	M	16	AS, AI, C	Died		+	+	I	90/70	SR	+	S	Soft	4	2		4	51·5		+	Z	
10	M	16	AS, AI	6		+	+	II	110/90	SR		S	N	4	2		4				S	
11	M	30	AS, AI	Died		+		II	130/68	N	+	S	Soft	3	3		4	43	++		Z	
12	M	43	AS, AI	24		+		I	110/95	SR	+	S	N	5	3		4				S	
13	F	60	AS, AI	Died		+		II	100/80	SR		S	N	3	1		4				S	
14	M	37	AS, MS	18		+		II	110/70	N		S	Z	3	Ab	+	1	50	+	+	Z	
15	F	24	AS, MS, AI	14		+		II	130/60	Col		S	N	3	2		1	50			Z	
16	F	31	AS, MS, AI	24		+		II	130/105	SR	Sh	S	N	2	2		2	50			Z	
17	F	32	AS, MS, AI	9		+		II	130/100		+	S	Loud	4	2		3	39	+		Z	
18	F	36	AS, MS, AI	28		+	+	II	130/85	N		S	Z					58				
19	F	43	AS, MS, AI	24		+		III	110/70	N	+	S	Loud	3	1		4	55	++		N	
20	M	48	AS, MS, AI	Died		+		II	90/60	SR		S	N	2	2		1	57			S	

1: CN=case number. 2: S=sex (M=male, F=female). 3: A=age in years. 4: L=lesion (AS=aortic stenosis, AI=aortic incompetence, MS=mitral stenosis, C=congenital). 5: FU=duration of follow-up in months (D=died). 6: SY=syncope. 7: An=angina. 8: PND=paroxysmal nocturnal dyspnoea. 9: FC=functional classification. 10: BP=blood pressure in mm. Hg. 11: RCP=character of radial pulse (SR=slow rising, Col=collapsing, N=normal). 12: CT=carotid thrill (Sh=shudder). 13: CPV=carotid pulse volume (S=soft, N=normal). 14: SSB=second heart sound at base (SC=soft ejection click, N=normal). 15: SSM=stenotic systolic murmur-grade. 16: EDM=early diastolic murmur-grade (Ab=absent). 17: CCF=congestive cardiac failure. 18: ECG, LVH=electrocardiogram, left ventricular hypertrophy-grades 0-5. 19: CTR=cardio-thoracic ratio (%). 20: LV+=left ventricle enlarged. 21: PDA=post-stenotic dilatation of the aorta. 22: AK=aortic knob (S=soft, N=normal). 23: AVC=calcification of aortic valve.

cardiographic changes of diminished left ventricular enlargement and significant radiological reduction in cardiac size. A standardized exercise-tolerance test was not performed.

OPERATION

One of us explored the mitral valve by auricular cardiotomy in all cases, and performed mitral commissurotomy if necessary. The finger was then passed into the left ventricle, and an area of the anterior ventricular wall was selected which was free from papillary muscles. The corresponding point on the surface of the heart was noted, a place being chosen where there were not many coronary vessels. The finger was removed from inside the heart and the auricle was clamped and sewn up in the usual way.

A purse-string was inserted around the chosen area and a small stab-wound made through it into the ventricle. Hegar's, or better Brock's, dilators were used to dilate the hole gradually and gently, and to find the outflow tract and the aortic valve. The septum was apt to produce extrasystoles if prodded, and ventricular fibrillation might occur. A good idea of the shape, size and position of the aortic orifice could be obtained by gently inserting the small dilator into the aorta and feeling it there with the free hand. Complete obstruction must not be permitted for more than one beat, but a fair idea of the size was obtained by feeling the variations in the aortic-systolic thrill. The pressure gradient could be obtained before and after dilating the valve by means of a malleable silver cannula attached to the lead tube of the recording apparatus (a Sanborn electromanometer was used in most cases, but a strain-gauge replaced it in the last few), which was passed into the ventricle and then through the valve. At other times a needle was pushed first into the ventricle and then into the aorta to record the magnitude of the pressures. A pressure gradient of 20 mm.Hg across the aortic valve was taken as the critical level for dilatation. In those cases where double valvotomy was performed the final systolic pressure gradient from ventricle to aorta was gauged after the mitral valve was opened.

The dilatation itself was done first by the Hegar's or Brock's fenestrated dilators, the sizes being gradually increased up to the one corresponding to the tri-fin expanding dilator, which completed the stretching of the valve. It is probably this instrument, if used too violently, that is most likely to produce an incompetence. The fingers of the free hand are placed around the aorta at the level of the valve to check any tendency of the dilator to overdo the stretching or slip too far into the aorta. There is usually no difficulty, after passing the first small dilator, in finding the way up the outflow tract; during the actual stretching the anaesthetist closes both carotid arteries in the neck and this has been enough to prevent embolism in the cerebral vessels.

In 2 cases obstruction was found not only at the valve but also ½-1 inch below it, apparently produced by the ventricular wall. Brock's punch was used to nibble away the anterior wall of the outflow tract, which on histological section was found to be lined by thick endocardium and rather fibrous myocardium. In both of these cases the valve itself was not stretched to the full; one has been improved and the other much improved. The former went into ventricular fibrillation at the end of the punch-dilatation, and required three shocks of the defibrillator to restore normal rhythm. In neither of these two cases was the gradient brought down to normal; but the results have justified the caution and perhaps even the incompleteness of the dilatation.

The cardiotomy opening in the ventricle is closed temporarily by the purse-string, and permanently by 2 or 3 non-absorbable mattress sutures.

RESULTS

Pressures. The pre- and post-valvotomy systolic pressure gradients across the aortic valve with operative results are shown in Table II. Post-valvotomy pressures were not recorded in those surviving cases who developed episodes of ventricular fibrillation on the table. The pre-valvotomy systolic pressure gradients ranged from 25-140 mm. Hg. Of the 11 cases in which post-operative pressures were recorded a marked reduction of the gradient occurred in only 6. It will be noted that the highest pressure gradients were present in those cases that died.

TABLE II. PRE- AND POST-OPERATIVE VENTRICULAR-AORTIC PRESSURE GRADIENTS AND RESULTS

Case No.	*Pre-*	*Post-*	*Result*
1	48	—	Improved
2	90	55	M.* Improved
3	58	10	M. Improved
4	140	—	Died
5	—	—	—
6	72	22	Died
7	70	54	M. Improved
8	35	10	Died
9	—	—	—
10	—	—	—
11	40	—	Worse
12	120	—	Died
13	95	57	Died
14	40	10	Improved
15	25	0	Improved
16	25	16	Worse
17	—	47	Worse
18	40	—	Improved
19	32	10	Same
20			

* M = Markedly.

Deaths. The causes of death, with post-mortem valvular findings, are listed in Table III. Four patients (cases 4, 5,

TABLE III. TIME AND CAUSE OF DEATH WITH POST MORTEM VALVULAR FINDINGS

Case No.	*Time of Death*	*Cause of Death*
4	During Op.	Ventric. Fibrillation
5	During Op.	Ventric Fibrillation
10	During Op.	Ventric. Fibrillation
12	During Op.	Ventric. Fibrillation
20	1 day post-op.	Post-op. Haemorrhage
8	2 weeks post-op.	Acute L.V.F.
6	5 months post-op.	Pulm. Infarction
13	9 months post-op.	Gross C.C.F.

Case No.	*Calcification of value*	*Pliability of value*	*Approx. Size of Orifice (sq. in.)*
4	Gross	Restricted	¼
5	Moderate	Fixed	¾
10	Moderate	Restricted	¾
12	Gross	Fixed	¼
20	Gross	Fixed	¼
8	Mild	Restricted	¼
6	—	—	—
13	Gross	Fixed	—

10 and 12) died of ventricular fibrillation on the table. One patient (case 20) died of shock due to persistent uncontrollable loss of blood from his thoracotomy wound, on the day after operation; autopsy did not establish the site of bleeding. Aortic valvotomy appeared to be successful in case 8, but on the day after the operation he developed an attack of acute left ventricular failure with electrocardiographic evidence of an anterior myocardial infarct. The attacks of pulmonary oedema persistently recurred and the patient finally succumbed after 2 weeks. The necropsy revealed an infarcted area of myocardium around the cardiotomy site, but no thrombus was found in the coronary vessels. Furthermore, inspection of the aortic valve showed a large laceration produced by the Brock's dilator, on the periphery of two grossly thickened, distorted, fused aortic cusps (Fig. 1).

Fig. 1. Post-mortem appearance of aortic valve in case 8. Note the laceration of fused aortic cusps (A B). Normal valve orifice (X Y).

Marked coronary atherosclerosis and pulmonary emphysema (diagnosed pre-operatively) was also present. A successful aortic dilatation was performed on case 6, but on the 15th post-operative day he developed a right-sided hemiplegia, presumably due to a cerebral embolus from the heart. This neurological complication improved sufficiently for the patient to be discharged from hospital 2 months after operation. His cardiac status at the time was felt to be satisfactory. Three months later he died after a massive pulmonary embolus from the lower limb. Permission for necropsy was refused. Case 13 had been in frank congestive cardiac failure on several occasions before she was operated on. After surgery cardiac failure returned and her condition remained virtually unchanged until the time of her death some 9 months after valvotomy.

Survivors

The results of surgery in the 20 cases are summarized in Table IV. Of the 12 surviving patients 8 were improved

TABLE IV. OPERATIVE RESULTS IN 20 CASES

Deaths	Operative deaths	5 (25%)	
	Late deaths	3 (15%)	
	Total	8 (40%)	
Survivors	Marked improvement	3 (25%)	8 (66·7%)
	Improved	5 (41·7%)	
	Same	1 (8·3%)	4 (33·3%)
	Worse	3 (25%)	
	Total	12 (100%)	
Total		20	

(66·6%), 3 markedly so. Four patients (33·3%) remained the same or became worse after operation.

Subjective changes, based on functional grading as noted above, revealed that of the 8 improved cases 3 improved from Grade II to 0, 3 from Grade II to I, one from Grade III to II, and one case, although he has remained Grade I, feels that he can do more than he could before the operation. In the group with poor operative results, 2 cases have regressed from Grade II to III, one to Grade IV, and one case has remained unchanged—Grade III (Table V). Symptoms of angina and syncope have disappeared completely in all three cases designated 'markedly improved'—they were present in all before operation (Table I)—and are considerably reduced in the other five cases designated 'improved'.

TABLE V. ANALYSIS OF PERTINENT PRE- AND POST-OPERATIVE CLINICAL FINDINGS IN SURVIVORS

CN	A	L	FU	FC		RP				EDM		DAI		ECG LVH		CTR		Result
						P		Ch										
				Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1	18	AS	14	II	I	30	90	SR	Col	Ab	4	—	+++	3	4	42	42	Improved
2	29	AS	26	II	0	24	40	SR	N	Ab	—	—	—	1		42	43	Markedly improved
3	34	AS	11	II	0	34	46	N	N	Ab	—	—	—	2	2	45	41	Markedly improved
7	48	AS	8	II	0	25	40	SR	N	Ab	2	—	+	3		48	—	Markedly improved
9	16	AS, AI, C	6	I	I	20	100	SR	Col	2	5	+	+++	4	4	51·5	58	Improved
11	30	AS, AI	24	II	III	62	100	N	Col	2	4	+	+++	4	4	43	54	Worse
14	37	AS, MS	18	II	I	40	40	N	N	Ab	3	—	+	1	2	50	57	Improved
15	24	AS, MS, AI	14	II	I	70	52	Col	Col	Ab	3	—	++	1	4	50	47	Improved
16	31	AS, MS, AI	24	II	III	25	120	SR	Col	2	4	+	+++	2	4	39	55	Worse
17	32	AS, MS, AI	9	II	IV	30	70	N	Col	2	3	+	+++	3	4	58	72	Worse
18	36	AS, MS AI	28	III	II	45	140	N	Col	2	3	+	+++	3	4	47	51	Improved
19	43	AS, MS, AI	24	III	III	40	60	N	Col	1	3	+	++	4	4	55	66	Same

1: CN=case number. 2: A=age in years. 3: L=lesion (AS=aortic stenosis, AI=aortic incompetence, MS=mitral stenosis, C=congenital). 4: FU=duration of follow-up in months. 5-6: FC=functional classification (5-18: Pre=pre-operative, Post=post-operative). 7-10: RP=radial pulse, P=pressure, Ch=character (SR=slow-rising, Col=collapsing, N=normal). 11-12: EDM=early diastolic murmur-grade (Ab=absent). 13-14: DAI=degree of aortic incompetence. 15-16: ECG, LVH=electrocardiogram, left ventricular hypertrophy-grade 0-5. 17-18: CTR=cardio-thoracic ratio (%).

Objective changes. The pertinent pre- and post-operative clinical findings have been listed in Table V. From purely objective criteria, i.e. changes in pulse pressure and character, the intensity of the early diastolic murmur and changes of left ventricular enlargement on electrocardiogram, it will be noted that before surgery 5 cases had no clinical aortic regurgitant lesion while in the remaining cases the aortic incompetence was considered to be minor. As a direct result of operation, 8 cases (66·6%)—see also Table VI—developed

TABLE VI. POST-OPERATIVE COMPLICATIONS IN SURVIVORS

Number of Cases	*Complication*	*Time of Onset**
8 (66·6%)	Gross aortic incompetence	Immediate
2	Mild aortic incompetence	Immediate
1	Sub-acute bacterial endocarditis	2
3	Post-valvulotomy syndrome	1–2
2	Left ventricular aneurysm	?–24
1	Antero-lateral infarct (ECG)	Immediate

* Months after operation.

gross aortic regurgitation and 2 (in whom no aortic incompetence was present before operation) developed mild aortic incompetence. Only 2 of the 12 survivors have remained after operation without clinical evidence of any aortic regurgitation.

In no case has there been any electrocardiographic evidence of diminution of left ventricular enlargement. Six patients showed a higher left ventricular grading after operation and 4 have remained unchanged (2 cases not recorded).

Heart size as evidenced by the cardio-thoracic index, showed slight reduction in 2 cases, no change in 2 cases, and moderate to marked increase in 7 cases.

Complications. Table VI lists the post-operative complications in the survivors. One case with congenital aortic stenosis (case 9) developed sub-scute bacterial endocarditis 2 months after aortic dilatation. Three cases showed evidence of a post-valvotomy syndrome with fever, chest pains of a pleuro-pericardial nature and arthralgias shortly after operation. One case (case 16) had repeated attacks of this syndrome during the first year after operation. Two cases have developed left ventricular aneurysms as revealed by X-ray (one of them also by electrocardiographic changes). The electrocardiogram of another patient (case 3), taken soon after operation, revealed the pattern of a large antero-lateral infarct; this has since remained unchanged, but clinically the patient has markedly improved.

DISCUSSION

Because of the small number of cases and the co-existence in some of multivalvular lesions which have added certain variables difficult to interpret, any deductions made from this report should be no more than tentative. However, the total number of cases of aortic valvotomy carefully followed up is small, and consideration of the cases in this report has some value when compared with other series.

The results of aortic valvotomy (Table IV) in this series compares favourably with those reported in other series where the valvulotomy was performed *via* a transventricular approach.[5-8] The immediate operative mortality in these reports varied between 17 and 31·8% and the over-all mortality between 25 and 45%. The 'good' results in survivors ranged between 50 and 88% and the 'poor' results from 7·6 to 50%. The 8 (66·6%) good results in this series shows that reasonable success can be achieved by this operation despite valvular deformity. The beneficial results of surgery in the improved cases have, to date, been maintained and emphasize the value of successfully relieving the mechanical obstruction.

Evaluation of direct intracardiac pressures at open-chest surgery is of limited value, for they are influenced by many factors—exposure of the intrathoracic structures to atmospheric pressure, positive pressure, ventilation, anaesthesia, loss of blood and manipulation of the heart during the operation. Nevertheless, such pressure measurements probably do indicate approximately the degree of severity of the valvular obstruction. If this assumption is valid it would appear that the aortic stenosis was most severe in those cases who died, was moderate in the improved group, and was mild in the cases with poor results (Fig. 2). This suggests that in patients with severe grades of aortic stenosis the operative mortality is extremely high; and the patients with mild stenosis, judged by pressure gradient, presumably had poor results because aortic stenosis was not a major lesion (they all had mitral disease as well), and surgery tended to replace an insignificant stenosis with a significant aortic regurgitation.

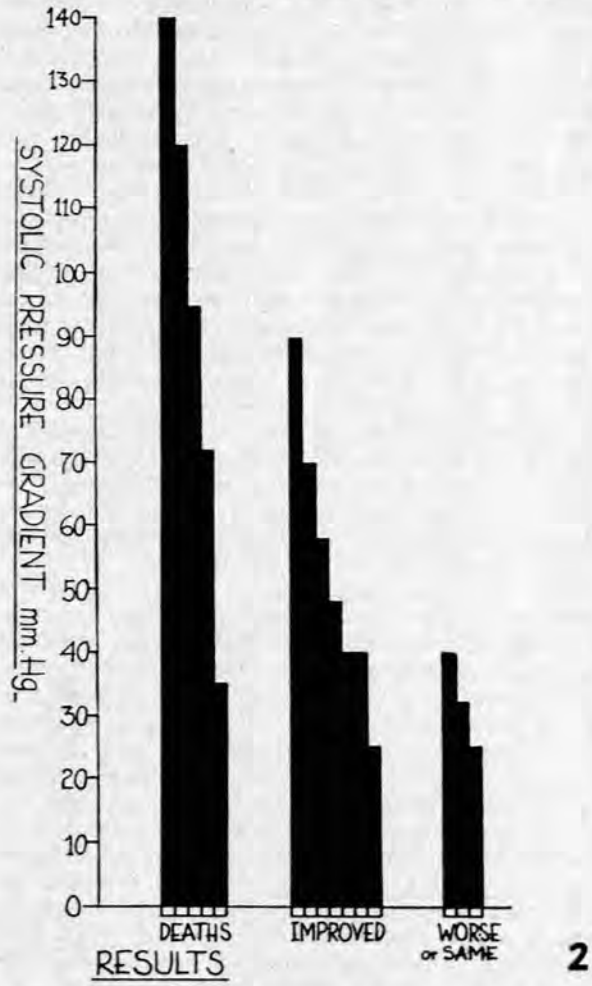

Fig. 2. Comparison of pre-operative aortic systolic pressure gradients with results.

Cerebral embolization during or soon after aortic valvotomy is a well recognized hazard of this operation[5, 6] despite carotid compression during dilatation of the valve. In this series only one case (case 6) sustained a cerebral embolus. He developed hemiplegia (from which he recovered) 15 days after the operation. There were no embolic phenomena after operation in the rest of the survivors.

In none of the reported series so far has mention been made of the development of left ventricular aneurysm after aortic commissurotomy *via* the transventricular approach. Two such cases (cases 11 and 19) occurred in the present series. This is not unexpected when one considers that aneurysms of the right ventricle have occurred after pulmonary valvotomy for isolated pulmonic stenosis.* A third patient (case 3), despite his excellent functional improvement, has an extensive anterolateral infarct pattern on electrocardiogram since operation. Presumably the pathogenesis of the aneurysm is the same as that occurring after myocardial infarction; the site of cardiotomy being the weak point (infarcted area) in the left ventricular wall. If this complication is to be averted it is possible that the usual 10-day post-operative period of bed rest may be insufficient.

A major factor in relieving stenosis is the production of regurgitation. How often this is produced cannot be accurately concluded from the literature. Muller and Hyman[9] report that significant aortic incompetence did not occur post-operatively in any of their 13 cases. Likoff *et al.*[6] state that the risk of producing aortic insufficiency where none existed before is 5% and that of creating or increasing an early diastolic murmur is 13%. In the largest series published to date by Bailey *et al.*[5] there is no indication of the incidence of post-operative regurgitation. In the present series the incidence of aortic incompetence developing after surgery is alarmingly high. Whereas before operation 5 of the surviving cases had no clinical evidence of incompetence and the remaining 7 had only insignificant regurgitation, post-operatively 66·6% developed gross and 16·6% mild aortic insufficiency. Only 2 of the 12 surviving patients escaped this complication completely and in the cases with gross aortic incompetence there has been associated evidence of increase in the size of the left ventricle, both electrocardiographical and radiological (Table V). The creation or exaggeration of aortic incompetence is a serious development and its possibility should be a prime consideration in deciding to send any patient for aortic valvotomy.

The deformity and calcification of the aortic valves is considerable and it would appear impossible from the post-mortem motion pictures (McMillan[10]) that the relief of aortic stenosis could be achieved without the production of significant aortic incompetence in a high proportion of cases. On the other hand this complication of the operation should not obscure the record of success obtained in the cases with good results. The most disturbing problem, however, is the inability to predict before operation which cases will develop gross, and which mild or no aortic insufficiency.

Of the 6 surviving cases with isolated aortic lesions, good results were obtained in 5, while in the combined group the results were half good and half poor. This suggests that those cases with isolated aortic lesions who survive operation have a much better improvement clinically than those with combined lesions. A possible explanation for the lesser improvement in the latter group might be the superimposition on an already damaged mitral valve of an operatively produced aortic regurgitation.

In Table VII the pre-operative clinical data in survivors and deaths have been reviewed to see which factors might have a bearing on the selection of future cases for aortic valvotomy. The following observations may be made:

TABLE VII. COMPARISON OF PRE-OPERATIVE CLINICAL DATA IN SURVIVORS AND DEATHS

	Survivors	*Deaths*
No. of cases	12	8
Sex:		
Males	6	5
Females	6	3
Lesion:		
Isolated aortic stenosis	4	4
Aortic stenosis and incompetence	2	3
Isolated aortic lesion	6	7
Aortic and mitral lesion	6	1
Symptoms and Signs:		
Syncope	6	4
Angina	7	7
Paroxysmal noctural dyspnoea	5	5
Congestive cardiac failure	1	3
ECG:		
Left ventr. hypertrophy, grade 1	3	3
Left ventr. hypertrophy, grade 2	2	—
Left ventr. hypertrophy, grade 3	3	1
Left ventr. hypertrophy, grade 4	3	3
X-ray:		
Cardio-thoracic ratio 40-50%	8	2
Cardio-thoracic ratio 51-60%	3	3
Aortic valve calcification	1	4
Mean age (years)	31·5	42
Mean duration (years)	3·8	5

1. *Age.* The mean age of the survivors (31·5 years) was considerably lower than the mean age of the fatal cases (42 years). In 10 cases under 35 years of age there was only one fatality, while in 7 cases over 40 years there were only 2 survivors (Table I). This conforms with the results published by Likoff *et al.*[6] showing that the operative mortality for aortic commissurotomy is 4 times greater after the age of 50 years than before.

2. *Combined lesions.* This report further conspicuously bears out the observation of Bailey *et al.*[5] that patients with combined aortic and mitral stenotic lesions simultaneously corrected are attended with a much lower operative mortality than those with essentially pure isolated aortic stenosis in whom the aortic valve alone was operated upon. Likoff *et al.*[6] state that the mortality rate is 3 times greater for aortic valvotomy alone than it is for combined aortic and mitral valvotomy. Of the 8 deaths in this series, 7 cases had isolated aortic lesions, as compared to only 1 with combined aortic and mitral stenosis. Two possible explanations for this difference in mortality are as follows: (1) That a severe grade of mitral stenosis may apparently 'protect' the left ventricle from excessive hypertrophy while in isolated aortic stenosis the left ventricle, by contrast, is thickened and seems more likely to fibrillate during surgery. Furthermore, the enlarged and thickened ventricle is more difficult to pump manually in maintaining the circulation during ventricular fibrillation and the increased muscle mass makes electrical defibrillation more difficult (Zinsser[11]). (2) In the group with multi-valvular lesions, it is possible that the mitral stenosis is the predominant cardiac disability while the aortic one is relatively mild or

* Unpublished data from the Cardiac Clinic, Johannesburg General Hospital.

insignificant. Hence the operative mortality would tend to approximate to that for mitral valvotomy alone. A study of symptomatology before valvotomy may serve to substantiate further the dominance of mitral stenosis in the combined group. The subjective pattern in combined stenosis is made up of symptoms which characterize the individual lesions. Where the manifestations of a particular involvement are predominant it most likely follows that this is in fact the predominant lesion. Angina and syncope reflect aortic valve involvement while in mitral stenosis these symptoms are most uncommon. Hence, in the 13 cases with isolated aortic lesions, syncope and angina were present in severe degrees in 7 and 11 patients respectively, whereas in the 7 patients with dual valvular lesions these two symptoms were present (in a mild form) in only 3 and 3 cases respectively. It may be argued correctly therefore that aortic stenosis was not the predominant lesion in the majority of patients with combined mitral and aortic valve disease.

3. *Symptomatology.* The pre-operative presence and duration of such symptoms as syncope, angina and dyspnoea appeared to have no prognostic significance on the outcome of surgery in this series. This similar lack of correlation has been reported by others.[6, 12] On the other hand the presence of congestive cardiac failure before operation was highly significant. Of the 4 cases who were in cardiac failure before valvotomy, 2 died, 1 became worse and 1 has remained the same. This would indicate that the presence of pre-operative congestive cardiac failure may be an unfavourable prognostic sign for operative success. The same experience pertained in the series reported by Baker and Campbell.[7]

4. *Electrocardiogram.* The degree of pre-operative left ventricular grading on electrocardiogram in the survivors and deaths was not helpful in deciding on the risks of surgery (Table VII). However, a progressive deterioration of the electrocardiogram has been regarded as a strong indication for operation, provided that no contra-indications are present.

5. *X-rays.* Before surgery the heart size was radiologically normal in 8 of the 12 survivors as compared to only 2 of the 8 fatal cases. Furthermore, the presence of aortic valve calcification on fluoroscopy was found to be of great prognostic significance in this series. This sign was present in 4 (80%) of the deaths as compared to only 1 (20%) of the survivors. Thus, a large heart with valvular calcification seen on fluoroscopy might be expected to be a most unfavourable combination of signs. Logan *et al.*,[12] reporting on a series of 15 cases subjected to aortic valvotomy, noted that aortic valve calcification was present in all the cases that died. Some of these conspicuous clinical differences in the survivors and deaths are shown in the histogram in Fig. 3.

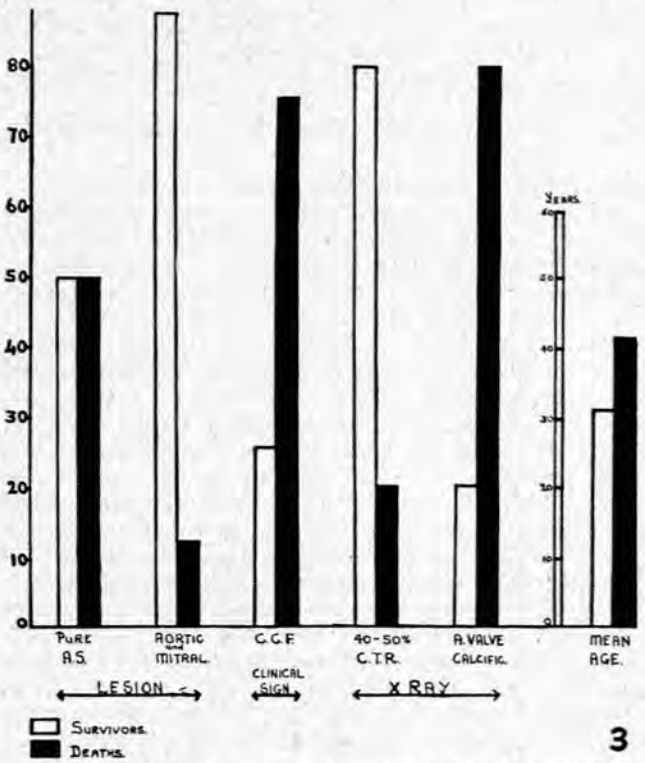

Fig. 3. Significant pre-operative clinical data in survivors and deaths.

SUMMARY AND CONCLUSIONS

The results and follow-up study of 20 patients who underwent transventricular aortic commissurotomy are presented. The operative results in this series compare favourably with those reported in others. The mortality rate is high when the aortic valve alone is diseased (50%) and low (14%) when there is combined aortic and mitral stenosis.

The most serious permanent complication of the operation is the production of aortic incompetence.

The clinical factors which indicate a poor surgical risk are defined. They include (1) increasing age; (2) an isolated aortic valve lesion (worse than combined aortic and mitral stenosis); (3) the presence of congestive cardiac failure before operation; (4) a large heart with fluoroscopic evidence of aortic valve calcification and (5) a transvalvular pressure gradient >100 mm.Hg before aortic valvotomy. Each sign does not necessarily constitute a contra-indication, but when two or more are present, then we believe that such a case should not be submitted to surgery.

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