COMPLETE REPLACEMENT OF BOTH MITRAL AND AORTIC VALVES

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Complete replacement of the mitral or of the aortic valve with a prosthetic valve is now a widely recognized procedure undertaken in most cardio-surgical centres.[1-4] In our unit, single valve replacement carries with it a low operative risk and morbidity,[5-8] but replacement of both aortic and mitral valves at one procedure is a far more hazardous and difficult undertaking.[9] The combined replacement of both valves presents a perplexing problem for cardiac surgeons, so much so that experienced surgical teams refrain from attempting multiple valve replacements,[10] and others report high mortality.[1] It is our belief that double valve replacement can be done with a low mortality, provided that patients are fully investigated before operation and the surgical programme is carefully planned. In this communication we report 2 successful surgical results in consecutive cases of severe mitral and aortic valve disease.

CASE REPORTS

Case 1

E.J.B., a White male bricklayer aged 30, was completely asymptomatic until the age of 26, when he suddenly had an attack of acute pulmonary oedema in the early hours of the morning. Pulmonary embolism appeared to have been the precipitating factor, since the acute episode was followed by chest pain and haemoptysis for which he was hospitalized elsewhere for a week. A month after discharge, on discontinuation of all cardiac therapy, he awoke one morning with a recurrence of acute paroxysmal dyspnoea, cough and haemoptysis, requiring urgent readmission and treatment. After this discharge, he developed typical angina pectoris and effort dyspnoea, forcing him to change his occupation to that of barman. From that time his symptoms progressed steadily and relentlessly until he was orthopnoeic, had recurrent attacks of paroxysmal dyspnoea, lost weight and was completely disabled. At the time of examination in the Cardiac Clinic, his effort tolerance was extremely restricted by angina and dyspnoea, and despite digitalis and diuretics, he was virtually a total invalid.

On examination, the pulse was stenotic, blood pressure was 100/65 mm.Hg, and the apex beat was slightly displaced, left ventricular in type and associated with a diastolic thrill. There was a marked lift over the right ventricle, suggestive of pulmonary hypertension. At the apex, a pansystolic murmur of mitral incompetence and a diastolic rumble were present; at the base, the systolic and early diastolic murmur of aortic stenosis and incompetence could be heard (Fig. 1). Right heart failure was absent. The ECG was normal apart from left atrial hypertrophy. X-ray and screening showed cardiomegaly with left atrial enlargement, enlargement of both right and left ventricles, and gross pulmonary venous congestion. The clinical impression was that of severe stenosis and incompetence of both mitral and aortic valves, both valves being calcified.

Cardiac catheterization showed severe pulmonary hypertension with a pulmonary-artery pressure of 110/60 and a brachial-artery pressure of 150/80 mm.Hg. The pulmonary-wedge pressure was 56/42 with a mean of 37 mm.Hg, and simultaneous left ventricular and wedge tracings showed a mean diastolic gradient of 35 mm.Hg (Fig. 2a). A withdrawal tracing from left ventricle to aorta showed severe aortic stenosis with a peak systolic gradient of 90 mm.Hg (Fig. 2b). The cardiac index was 3·9 l./min. with a pulmonary vascular resistance of 9 units.

Cine-angiocardiography showed gross stenosis with slight incompetence of both mitral and aortic valves. The catheter diagnosis was severe aortic and mitral stenosis with slight incompetence of both valves and gross pulmonary hypertension, in part caused by increased peripheral vascular resistance and in part by elevated pulmonary venous pressure. Surgery was advised with a view to replacement of both valves.

Operation was performed on 14 November 1963. The aortic valve was approached through the ascending aorta and the mitral through the left atrium from the left. A bilateral thoracotomy with transverse sternotomy above the 5th rib on the left, and above the 4th rib on the right, were performed, adequately exposing these structures.

Total cardiopulmonary bypass was established by draining the venous blood through a catheter in the right atrium and another in the pulmonary artery. The oxygenated blood was returned from the oxygenator through a catheter in the common femoral artery. Haemodilution and moderate hypothermia with high-flow perfusion were used. The aorta was opened transversely about $\frac{1}{4}$ inch above the right coronary ostium. Both coronary arteries were cannulated and perfused throughout the procedure with cold, oxygenated blood. The aortic valve was stenosed and incompetent from fibrosis and calcification of all 3 leaflets. The cusps were excised down to the annulus and the valve replaced with a size 3 (19 mm. diameter) UCT aortic prosthesis.[6] Once this was in place, the ascending aorta was allowed to fill with blood to dispel all air, and the aortotomy was closed with interrupted 4-0 silk sutures. The aortic clamp was released and the heart rewarmed. The left atrium was opened and the mitral valve found to be stenosed and incompetent, from calcification and fibrosis of both anterior and posterior leaflets, shortening of the chordae and hypertrophy of the papillary muscles. The leaflets and chordae, and portion of the papillary muscles, were excised. A medium

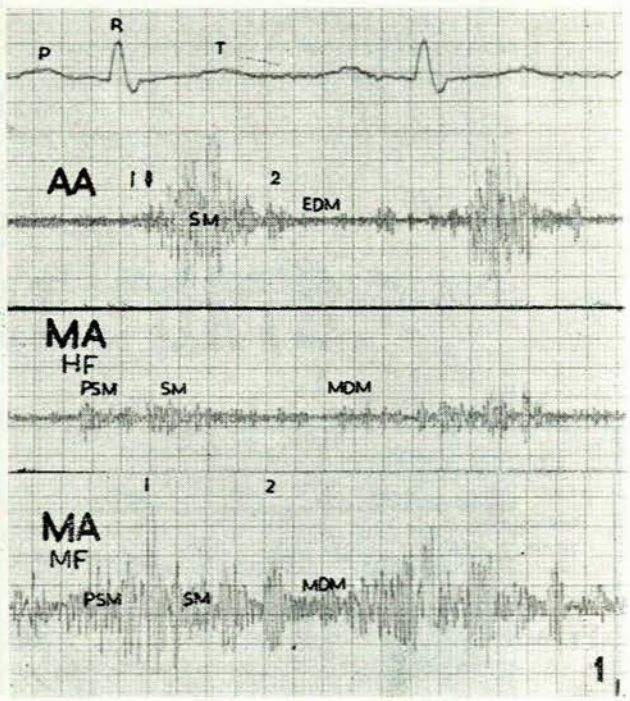

Fig. 1. At the aortic area (AA) the aortic systolic murmur and early diastolic murmur of aortic stenosis and incompetence are shown. Synchronous high frequency tracings at the mitral area (MA) show the pansystolic murmur of mitral incompetence. The low-pitched mid-diastolic and presystolic murmurs are brought out in the medium frequency recording (MAMP).

size (3·80 sq.cm. area) UCT lenticular prosthesis was inserted with interrupted 2-0 silk mattress sutures. Once in place, the left heart was filled with blood, the prosthesis being

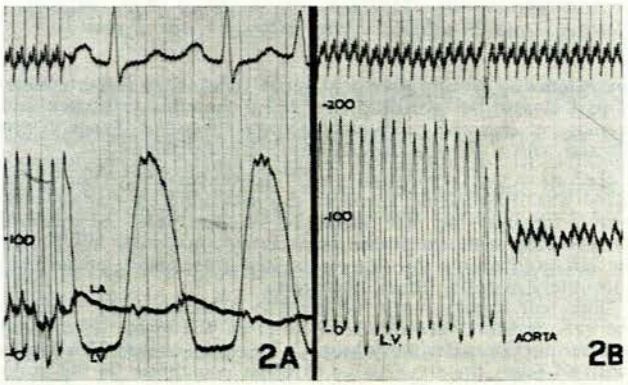

Fig. 2(a). The marked gradient between left ventricle and left atrium indicating severe mitral stenosis is shown. *Fig. 2(b)* shows gradient between left ventricle and aorta indicating severe aortic stenosis.

kept incompetent. The atriotomy was closed with interrupted 3-0 silk mattress sutures. The catheter in the pulmonary artery was removed and the opening closed with a purse-string suture. The heart was defibrillated with an electric shock and bypass discontinued. Duration of bypass was 215 minutes and recovery from the anaesthetic was prompt.

The patient's postoperative course was extremely smooth from the cardiac point of view and the patient was discharged from hospital 21 days after surgery. Postoperative neuritic pain in the right leg was the main disability. Three months after surgery the patient had no cardiac symptoms and exercise tolerance was completely normal. The major complaint was still pain down the right leg.

Case 2

J.v.R., a White female of 38 years, was first seen at the age of 28 with a story of severe rheumatic fever as a child of 12 years. She was left with valve lesions and moderate disability. At the age of 24 she married, fell pregnant, and was able to go through pregnancy without disability, following which she was thought to have had a recurrence of rheumatic fever for which she received steroids and salicylates. Palpitations and slight effort dyspnoea developed and, since she wanted to have more issue, she was referred for assessment. On examination she was found to have severe mixed aortic and mitral valve disease with considerable cardiomegaly and sinus rhythm.

When she was 37, atrial fibrillation developed, precipitating her into heart failure. From then her course was one of progressive disability with marked effort dyspnoea, orthopnoea, paroxysmal cardiac dyspnoea and finally congestive cardiac failure not responding to digitalis and diuretics. During this illness, she lost 28 lb. in weight.

On examination, there was no evidence of right heart failure. The pulse was bisferriens in type with a blood pressure of 130/60 mm.Hg. Considerable cardiomegaly was present with an apex in the 5th space beyond the midclavicular line. A marked lift over the right ventricle was present and pulsation beyond the right sternal border suggested a left atrial aneurysm, which was confirmed on screening. A pansystolic murmur of mitral incompetence with a short mid-diastolic murmur

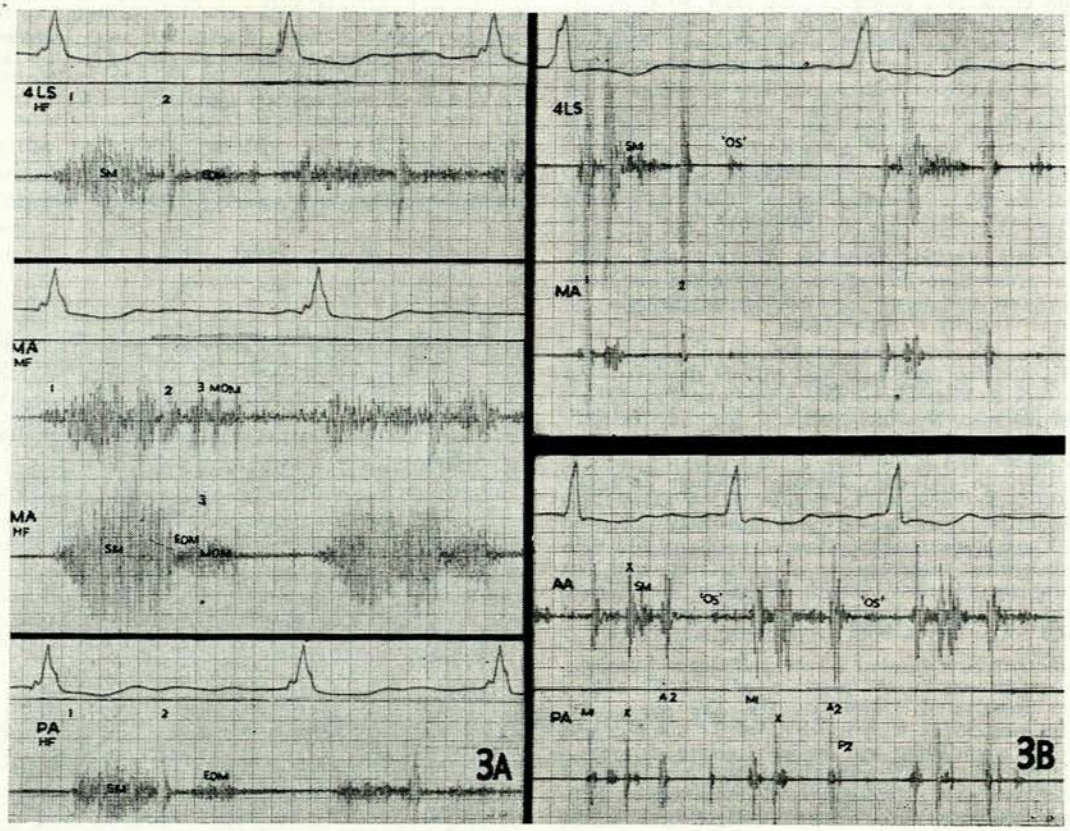

Fig. 3(a). Before surgery. The pansystolic murmur of mitral incompetence and the mid-diastolic murmur of mitral stenosis are recorded at the mitral area. The aortic systolic and early diastolic murmurs are shown at the pulmonary area (PA) and tricuspid area (4LS). A third sound is recorded and atrial fibrillation is present. *Fig. 3(b). After surgery.* The click produced by the aortic prosthesis when fully open is shown in systole. The loud second sound is due to closure of the aortic prosthesis. The sound in diastole 'OS' is due to the opening of the mitral prosthesis. There are no diastolic murmurs, but a short aortic systolic murmur persists.

and no opening snap were heard at the apex (Fig. 3a), and at the base, systolic and early diastolic murmurs of aortic valve disease were present. The ECG showed marked digitalis effect, probably superimposed on left ventricular hypertrophy. On X-ray, moderate cardiomegaly with a large left atrium and slight pulmonary venous congestion were present (Fig. 4a).

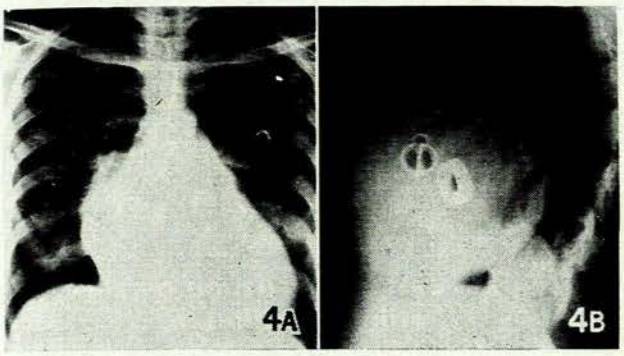

Fig. 4(a). The cardiomegaly, large left atrium and hilar congestion are shown. *Fig 4(b).* The two prosthetic valves are shown.

At cardiac catheterization, the right ventricular pressure was 64/6, left atrial pressure 33/11 with a mean of 18, left ventricular pressure 160/5 and right radial-artery pressure 160/77 mm.Hg. Synchronous left atrial and left ventricular pressures showed a mean diastolic gradient of 14 mm.Hg (Fig. 5) and a pull-through from left ventricle to aorta showed no systolic gradient. The cardiac output was 2·4 l./min. with a cardiac index of 1·65 l./min./metre^2 body surface area, and a pulmonary vascular resistance of 10 units.

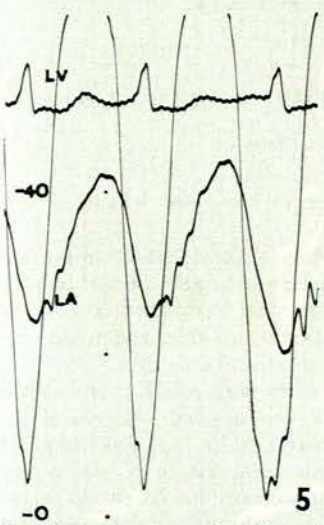

Fig. 5. Synchronous left-atrial and left-ventricular tracings show the marked gradient across the mitral valve and the large CV wave of mitral incompetence pre-operatively.

Cine-angiocardiography confirmed the presence of gross mitral incompetence with calcified mitral valve and fairly marked aortic incompetence. Surgical repair was advised.

Operation was performed on 24 February 1964. Once again, the aortic valve was approached through the ascending aorta and the mitral through the left atrium from the left. A median sternotomy was employed to expose the ascending aorta and a left anterolateral thoracotomy through the bed of the 4th rib was used as the approach to the left atrium. Total cardiopulmonary bypass was instituted by draining the venous blood by means of a single catheter in the right atrium, the arterial blood being returned from the pump oxygenator through a catheter in the common femoral artery. Haemodilution, moderate hypothermia and high perfusion were used. The aortic valve was exposed through a transverse aortotomy. Both coronary arteries were cannulated and perfused with oxygenated cold blood. The aortic valve was severely incompetent owing to fibrosis and shortening of all 3 cusps. These were excised down to the annulus and the valve replaced with a No. 4 (22 mm. diameter) UCT aortic prosthesis and secured with interrupted 2-0 silk mattress sutures. The ascending aorta was filled with blood and the aortotomy closed with interrupted 4-0 silk sutures. The aortic clamp was released and the heart rewarmed. The left atrium was opened and the mitral valve found to be incompetent owing to fibrosis and shortening of both cusps, and shortening of the chordae and papillary muscles. Some calcification was observed in the anterior leaflet. Both leaflets with chordae and part of the papillary muscles were excised, and a No. M/23 (3·80 sq.cm. area) UCT mitral prosthesis was inserted and secured with interrupted 1-0 and 2-0 silk mattress sutures. Once in place the valve was kept incompetent and the left heart filled with blood. The left atriotomy was closed and the heart defibrillated with a single electric shock. The patient had an uneventful postoperative course.

Two weeks after surgery atrial fibrillation was successfully converted to sinus rhythm with quinidine, only to recur 2 days later. A further trial failed, so quinidine was abandoned and digitalis continued. The patient was much improved when discharged 3 weeks after operation (Figs. 3b and 4b).

DISCUSSION

Experience with valve surgery[7] has shown a wide spectrum of both mitral and aortic valve disease, varying from pure stenosis to pure incompetence. Between these 2 extremes, all combinations of both stenosis and incompetence occur. As far as mitral valve surgery is concerned, a wide choice of procedures exists.[11] For pure stenosis, closed valvotomy is still the method of choice, whereas in pure incompetence either annuloplasty, a baffle procedure or pericardial grafting, may be selected. When stenosis and incompetence co-exist, valve damage is usually more severe and generally speaking nothing short of valve excision and replacement is effective. In the case of aortic valve disease, on the other hand, we have concluded that valve excision and replacement is the only regularly successful procedure at present, whatever the haemodynamic disturbance.

Both the mitral and aortic valves are involved in about one-third of patients presenting with rheumatic-valve disease. The cardiac surgeon is thus frequently presented with the problem of repairing both valves, and bi-valve replacement may be the only way in which this can be effectively accomplished.

It has been our experience that repair of both aortic and mitral valves should be done at one operation[8] and should not be staged.[10] This can be achieved with a low mortality, provided that (1) adequate exposure of both valves is obtained, (2) optimal total body perfusion is maintained, and (3) the myocardium is carefully protected throughout.

1. *Adequate Exposure of both Valves*

Replacement of both aortic and mitral valves entails a long period of total cardiopulmonary bypass. A good exposure of the valves to be replaced will reduce the duration of the procedure to the minimum. The aortic valve is best exposed through the root of the ascending aorta, and the mitral valve through a left atriotomy. A median sternotomy, left thoracotomy or right thoracotomy alone usually do not provide ready access to both these structures. For bi-valve replacement, therefore, a wider exposure is necessary—such as a bilateral thoracotomy with transverse sternotomy, or a median sternotomy with either a right or a left thoracotomy, depending on whether the left atrium is approached from the right or the left side.

An accurate pre-operative assessment is essential to ensure that the surgeon knows beforehand that he has to deal with both valves and can plan his approach accordingly. Our most common error has been the underassess-

ment of the severity of the aortic valve lesion in the presence of severe mitral valve dysfunction.

2. *Optimal Total Body Perfusion*

The technique used is basically similar to that described previously.[12] High flow rates at slightly reduced body temperatures, with haemodilution, appear to be more satisfactory, however.[13, 14] The bubble oxygenator having been primed with one-third of 5% dextrose and water, and two-thirds of fresh, whole, heparinized blood, flow rates of 2·2 l./min./metre^2 body-surface area, were used. The duration of perfusion was 215 minutes in case 1 and 192 minutes in case 2.

3. *Careful Protection of the Myocardium*

Coronary blood flow is maintained throughout the procedure. Both coronary arteries are cannulated and perfused via a separate system with cold, oxygenated blood.[6] Another important point is the prevention of forceful retraction of the heart muscle, and decompression of the left ventricle as soon as bypass is begun to avoid overdistension.

SUMMARY

Severe disease of both mitral and aortic valves is a common clinical condition resulting in considerable disability, frequently ending in congestive cardiac failure and death. Prosthetic mitral and aortic valves which provide adequate function have been developed and can be used to replace diseased valves with a low operative morbidity and mortality. Replacement of both valves at one procedure presents a severe challenge to the cardiac surgeon.

We report the successful replacement of both aortic and mitral valves in 2 patients.

We wish to thank our surgical and medical colleagues in the Cardiothoracic Surgical Unit and Cardiac Clinic, Groote Schuur Hospital, for their cooperation and assistance, and in particular Mr. C. C. Goosen for the large part he played in the development of the UCT prosthetic valves. We also thank Dr. J. G. Burger, Medical Superintendent of Groote Schuur Hospital, for permission to publish details of these cases. Finally, we are grateful to the CSIR, the City Council of Cape Town and the University of Cape Town for financial assistance. As always, Prof. J. H. Louw of the Department of Surgery, University of Cape Town, receives our appreciative thanks for his constant support.

REFERENCES

1. Ellis, F. H., McGoon, D. C., Brandenburg, R. O. and Kirklin, J. W. (1963): J. Thorac. Cardiovasc. Surg., **46**, 482.
2. Effler, D. B. and Groves, L. K. (1963): Dis. Chest, **43**, 529.
3. Björk, V. O., Cullhed, I. and Lodin, H. (1963): J. Thorac. Cardiovasc. Surg., **45**, 635.
4. Kay, E. B., Zimmerman, H. A. and Mendelsohn, D. (1963): Circulation, **27**, 767.
5. Barnard, C. N., Goosen, C. C., Holmgren, L. V. and Schrire, V. (1962): Lancet, **2**, 1087.
6. Barnard, C. N., Schrire, V. and Goosen, C. C. (1963): *Ibid.*, **2**, 856.
7. Barnard, C. N. and Schrire, V. (1963): Surgery, **54**, 302.
8. Barnard, C. N., Schrire, V., Frater, R. W. M. and Goosen, C. C. (1964): Surgery (in the press).
9. Cartwright, R. S., Giacobine, J. W., Ratan, R. S., Ford, W. B. and Palich, W. E. (1963): J. Thor. Cardiovasc. Surg., 45, 35.
10. Effler, D. B., Groves, L. K. and Favaloro, R. (1964): Arch. Surg., **88**, 145.
11. Frater, R. W. M., Barnard, C. N. and Schrire, V. (1964): S. Afr. Med. J., **38**, 776.
12. Barnard, C. N. and Schrire, V. (1961): Surgery, **49**, 500.
13. DeWall, R. A., Lillehei, R. C. and Sellers, R. D. (1962): New Engl. J. Med., **266**, 1078.
14. Kirklin, J. W. (1962): Surg. Clin. N. Amer., **42**, 1537.

CONSIDERATIONS IN THE PROSTHETIC CONSTRUCTION OF THE AORTIC VALVE

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'I will praise thee; for I am fearfully and wonderfully made; marvellous are thy works; and that my soul knoweth right well.

My substance was not hid from thee, when I was made in secret, and curiously wrought in the lowest parts of the earth'.

Psalm 139: 14, 15.

Diseased aortic valves are at present being replaced by prosthetic valves by cardiac and experimental surgeons in many parts of the world. In the construction of such valves synthetic materials,[1, 2, 6, 7, 9, 10, 14] homologous[3, 11, 12] or autogenous[8, 13] tissues are being employed.

The present-day opinion seems to favour synthetic materials. At the same time, however, homologous-organ transplantation[4, 5, 15, 16] is receiving the serious attention of many workers in this field. It has been well established that only in the case of identical twins will the transplanted organ be tolerated by the host, without the development of immunological reactions. In the case of valve surgery it can therefore be justly assumed that the heart will similarly tolerate its own pericardium as a valve. Whether, however, the healing granulation-tissue reaction at the site of union will be so limited to the vicinity of the suture line as not to cause significant scarring and possibly calcification, even after many years, and whether the relatively avascular pericardium will obtain sufficient nutriment from the blood medium as to prevent significant anoxic degeneration with ultimate fibrosis, are questions which, as yet, cannot be fully answered.

Autogenous pericardium has several other important attributes. It is readily and freely available at the time of operation in a fresh and sterile state and at no cost to the patient or institution. It is strong and thin and when employed as a valve cusp it has great mobility.

In order to manufacture such pericardial cusps at the time of operation, a formula was devised whereby it became possible to determine correctly the size and shape of a cusp which would fit the aorta in question in such a way that neither incompetence nor stenosis would either persist or develop. Thereafter the main objection to pericardium seemed a technical one because pericardium was extremely difficult to handle as a single cusp, while as a whole valve it was virtually impossible to handle. This difficulty was

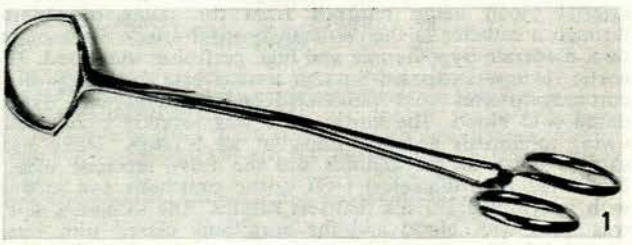

Fig. 1. Aortic pericardial-cusp clamp.