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CONTINUED MORBIDITY AFTER MITRAL VALVE SURGERY

M. M. ZION, M.B., B.CH. (RAND), M.R.C.P. and J. L. BRAUDO, M.B., B.CH. (RAND), M.R.C.P.E.

From the Cardiac Clinic, Johannesburg General Hospital, and the Cardio-Pulmonary Research Unit of the Council for Scientific and Industrial Research

There is today fairly general agreement on the indications for surgery in mitral stenosis and results that have much in common have been obtained in most centres. Approximately 70% of patients obtain a good result, and in the great majority of these the benefit obtained is lasting. What of the 30% in whom there is an immediate poor result or who actually regress after initial improvement? In view of the large number of patients who have undergone mitral valvotomy, the absolute number of those with poor results is not insignificant. It is the purpose of this report to discuss some of the problems presented by such patients.

Material

In April 1956, a clinic was opened under the aegis of the Cardiac Clinic at the Johannesburg General Hospital for the purpose of following up patients who had undergone mitral valvotomy. In spite of efforts to see all such patients, it soon became obvious that the great majority of the patients attending the clinic were in fact those with continuing ill-health. Very few of those with good results were seen and we were not able to trace such cases, not having the help of a social worker.

Thus, of over 300 patients subjected to mitral-valve surgery, we were personally able to follow up only 98, of whom 42 (43%) had good results, 16 (16%) fair results and 40 (41%) poor results. The result in each case was assessed by the effect of the operation on disability as graded by Wood,[1] as follows: An improvement by 2 grades or more constituted a good result, by 1 grade a fair result, and no change or deterioration, a poor result. A proportion of 'good result' cases attending the clinic were troubled by temporary complications, and they often ceased attending when the complications abated. Figures for all our cases can therefore not be given. However, the problems presented by patients who continued to attend could be divided into the following clear-cut groups:

1. 'Post-commissurotomy syndrome'.
2. Inadequate enlargement of the mitral valve orifice.
3. Late re-stenosis of the mitral valve.
4. Repeat mitral valvotomy.
5. Persistent right heart failure and/or tricuspid incompetence.
6. Combined mitral stenosis and mitral incompetence.
7. A miscellaneous group with poor results despite adequate operative enlargement of the mitral valve.

'Post-Commissurotomy Syndrome'

Recurrent bouts of this syndrome, as described by Papp and Zion[2], troubled some of our patients, irrespective of whether the functional result of surgery was good or poor. The clinical picture consists of recurrent bouts of fever with chest pain and, in some, evidence of pericarditis and left pleural effusion. Each episode is rapidly aborted by steroid therapy, the pyrexia settling within 2 or 3 days.

Case 1

A 50-year-old male, had a successful mitral valvotomy in April 1956. From 1 month after operation, he experienced recurrent bouts of substernal pain, aggravated by respiration and associated with fever. Each bout of illness was rapidly relieved by prednisone, 5 mg. q.i.d., and, in the intervals, he was asymptomatic, with a normal effort tolerance. These attacks occurred at intervals of 2-4 months, the most recent being in August 1957, 16 months after operation.

Inadequate Enlargement of the Mitral Valve Orifice

It is the aim of the thoracic surgeons operating on cases from the clinic to split stenosed mitral valves to the valve ring, or as near thereto as possible. In their earlier experience, the surgeons were often content with much less extensive splits and, even at the present time, technical difficulties occasionally preclude an adequate split. While it cannot be denied that there are instances of continued good results among subjects in whom the split was considered adequate, it is clear that a number of our patients with persistent disability were in fact those with inadequate splits. It may well be that these latter cases have re-stenosed soon after surgery, as suggested by Wood[1] (case 3). However, it must be remembered that the surgeon's description of the size of the valve orifice after surgery is only a rough indication of the residual degree of obstruction to blood flow, and the actual obstruction may be significantly different in cases with apparently equal 'long diameters' of valve orifice as described by the surgeon. Late re-stenosis may occur with a recurrence of symptoms after improvement lasting 1-2 years (case 5).

In assessing these subjects, we have appreciated that the patients' description of improvement in the first 6-9 post-operative months may be due to causes other than those

associated with the relief of obstruction at the mitral valve. It is common experience that patients may mention improvement even when only cardiotomy without valvotomy has been performed. This improvement may be psychogenic or, more important, may be due to the fact that in many subjects the operation has allowed time for intensive cardiac and respiratory medical therapy. Respiratory therapy consists of breathing exercises, postural drainage, etc., which may benefit those with chronic bronchitis, a not infrequent complication. In these subjects improvement is usually temporary and not noted for longer than 6-9 months.

Case 2

A 30-year-old female was subjected to cardiotomy in May 1954. Owing to the elastic quality of the valve, the surgeon was unable to achieve a split, but he stretched the valve orifice from a size admitting the finger to the base of the nail, to one admitting the finger to the proximal interphalangeal joint. For 6 months after operation she felt much better, her effort tolerance improving from Grade IIb to Grade I[1]. Thereafter, she rapidly deteriorated to her pre-operative status and she has experienced gradually increasing disability since then.

Late Re-Stenosis of the Mitral Valve

The question of early re-stenosis has already been mentioned. Late re-stenosis in subjects who have had *adequate* valvotomy is rare and, in our experience, is usually attributable to recurrent rheumatic activity. It is clear that rheumatic activity may occur in adults many years after the last recognised attack of rheumatic fever. Such rheumatic activity may manifest itself either by arthralgia without fever, or by overt acute rheumatic fever. As the cardiac penalty for rheumatic activity may be high and as such activity is largely preventable, it is our present routine to place all subjects undergoing valvotomy on permanent penicillin prophylaxis, either in the oral form (Phenoxymethylpenicillin—penicillin V), 120 to 240 mg. daily or Benzathine penicillin 1,200,000 U. intramuscularly once a month. In those subjects sensitive to penicillin, we have used sulphonamides and occasionally broad spectrum antibiotics.

In some of these patients with late re-stenosis, the symptoms have recommenced soon after the rheumatic activity. By contrast, symptoms attributable to mitral stenosis commence only several years after an initial attack of rheumatic fever. The development of tight stenosis in a previously normal valve would appear to be a slower process than in a previously structurally abnormal valve. However, tight stenosis may develop fairly rapidly in both groups, but the development of symptoms due to the stenosis may be related both to the degree of elevation of left atrial pressure and to its *duration.*

Case 3

A 17-year-old female with Grade II disability[6] underwent mitral valvotomy on 4 September 1953. The valve was tightly stenosed and a moderately good split to a diameter of 1 inch was performed. Thereafter she felt normal and returned to full activity. She was not on any anti-rheumatism prophylaxis. About 1 year after the operation she began to experience generalized joint pains, but did not consult a doctor. A few months later, effort dyspnoea recommenced and steadily progressed, culminating in an attack of acute pulmonary oedema during the 3rd month of a pregnancy, nearly 4 years after operation. On clinical examination, there were all the signs of tight mitral stenosis with radiological and electrocardiographic evidence of marked right ventricular and left atrial enlargement. These signs had been present before the operation, but owing to the patient's failure to attend for follow-up it is not known whether they changed during the early post-operative course. Pregnancy was terminated and repeat valvotomy advised. At operation a tight stenosis was found, with firm adhesion of the commissures, which were cut with a knife and split to the valve ring. A moderate degree of mitral incompetence was produced.

Case 4

A 27-year-old female was first seen in 1953, when she gave an 8-year history of dyspnoea, which had progressed to Grade III.[6] Clinically, there was evidence of tight mitral stenosis with electrocardiographic and radiological features of left atrial and right ventricular enlargement. At operation on 14 September 1953, a tight stenosis was found and a 'moderately good' split was performed. The excised left atrial appendage was found, on histological examination, to contain Aschoff nodes. She received oral penicillin for only 6 weeks post-operatively and remained extremely well for 2 years. Thereafter, she was assessed as having mild rheumatic activity in December 1955, and during 1956 breathlessness gradually returned and steadily progressed to Grade III disability. She experienced paroxysmal atrial fibrillation. Her electrocardiogram (Fig. 1), which after the first operation had shown marked diminution of right ventricular hypertrophy, now showed a return to the pre-operative picture, and her heart had enlarged slightly. Re-stenosis was diagnosed and repeat valvotomy advised. At operation a tight stenosis was found and digital splitting could not be performed. The anterolateral commissure was cut with a knife, and the final diameter of the mitral valve was 1½ inches.

Repeat Mitral Valvotomy

Patients of the previous 2 groups may be subjected to repeat valvotomy. This may be technically more difficult than the initial operation, partly because of more difficult entry into the previously opened left atrium. We feel that results of repeat mitral valvotomy should be considered separately from the results of the initial valvotomy. Our results in patients subjected to 2nd and 3rd operations will be presented in a separate publication.

Case 5

A 36-year-old female was first seen in 1951 with a history of progressive dyspnoea for 5 years. In May 1950 she coughed up small amounts of blood on 3 occasions. Orthopnoea and paroxysmal attacks of palpitations had been present for one year. Her disability when seen was Grade III.[6] All the clinical features of tight mitral stenosis were present. The electrocradiogram showed marked right ventricular hypertrophy and the radiological features of left atrial, pulmonary artery and right ventricular enlargement were present. The cardio-thoracic ratio was 45% and septal lines were present at the lung bases. One month before her first cardiotomy, congestive cardiac failure appeared. On 11 May 1951 cardiac catheterization revealed a pulmonary artery pressure of 92/48 mm. Hg. Pulmonary capillary pressure was not obtainable. Mitral valvotomy was attempted on 26 May 1951. The mitral orifice measured 1/8th inch in diameter with tough thickened edges. A split of the lateral commissure was obtained with difficulty. The medial commissure was partially cut with a Bailey knife. The final valve opening was 5/8ths inch in diameter. She improved considerably for 1½ years and cardiac catheterization 10 months after surgery revealed a pulmonary artery pressure of 40/18 mm. Hg and a pulmonary capillary mean pressure at rest of 12 mm. Hg. In January 1953 dyspnoea reappeared and by May 1954 she was as incapacitated as she had been before her first operation. An attempt at repeat cardiac catheterization was unsuccessful and on 20 August 1954 a second thoracotomy was performed. The mitral valve orifice was ¼ inch in diameter with considerably deformed and thick tough fibrous edges. The appendage tore during attempts at valvotomy, she bled profusely, and surgery was abandoned. Over the past 3 years she has become more incapacitated with severe dyspnoea on the slightest exertion, mild congestive cardiac failure, angina pectoris and permanent atrial fibrillation. The physical signs are unchanged. The electrocardiogram shows an increase of right ventricular hypertrophy and the X-ray findings are unchanged. Valvotomy is in our opinion still indicated in this patient and the use of newer techniques, with possible open cardiotomy, will be necessary.

Persistent Right Heart Failure and/or Tricuspid Incompetence

An interesting finding in 8 patients has been the persistence of right heart failure and/or tricuspid incompetence in

patients who, after a satisfactory valvotomy, have experienced considerable improvement in exercise tolerance. It seems reasonable to assume that after valvotomy the pressure in the pulmonary venous circulation decreases, with consequent diminished turgidity of the lungs and relief of dyspnoea. The cause of the persistence of signs of right heart failure is not clear and detailed haemodynamic investigation of these patients is planned with a view to elucidating the problem. The possibilities which suggest themselves are:

1. *Persistence of increased pulmonary arterial resistance.* It has been established[3] that in a proportion of patients who have had a successful valvotomy the pulmonary arterial resistance does not fall, but it is not clear from the reports whether such patients are symptomatically improved. Of the 8 patients we have seen with persistent right heart failure and/or tricuspid incompetence, 6 had definite electrocardiographic evidence of right ventricular hypertrophy pre-operatively and in 4 this diminished or disappeared. Thus it would seem that in at least some of these cases persistence of the increased pulmonary arterial resistance is not responsible for the clinical state.

2. *Irreversible myocardial damage.* In recent years, a great deal of attention has been focused on the mechanical obstruction to the flow of blood in patients with rheumatic heart disease. However, it is well to remember that earlier observers[4] often stressed the importance of myocardial damage in heart failure. Whether such damage may or may not be irreversible in some cases, is not known. It may be of significance that 4 of the 8 patients had moderately enlarged hearts (cardiothoracic ratio greater than 55% pre-operatively) with further enlargement post-operatively.

3. *Organic tricuspid disease* probably explains some of these cases, for it is accepted that the proportion of cases of tricuspid disease diagnosed clinically falls short of that found at necropsy.[7] Whatever the explanation, these patients continue to require medical therapy for control of their right heart failure. How their ultimate prognosis will compare with those with good results from valvotomy is not known, although it would seem that it should be significantly poorer. It is of interest that of our 8 patients, 2 had their operations more than 3 years ago, and they continue to remain subjectively well.

Case 6

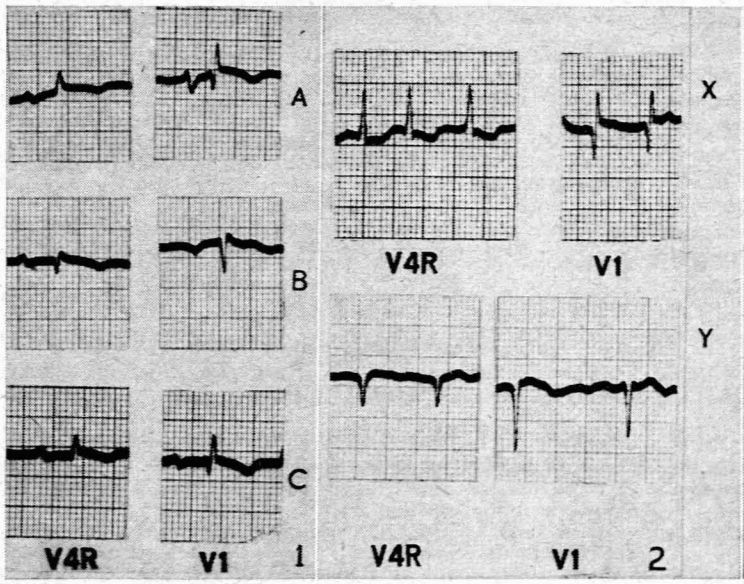
A 30-year-old female was first seen in 1953, with dyspnoea Grade III-IV[6] and orthopnoea, but no paroxysmal nocturnal dyspnoea. Reversible congestive cardiac failure with evidence of tricuspid incompetence had been present off and on for 1 year. There was clinical evidence of tight mitral stenosis with atrial fibrillation. The electrocardiogram (Fig. 2) showed right ventricular hypertrophy and radiology revealed a large heart (C.T.R. 60%) with enlargement of right ventricle, pulmonary artery and both atria, and evidence of pulmonary hypertension (Fig. 3). At operation in July 1953, the valve was found to have a diameter of $\frac{3}{8}$ths inch and was split to over 1 inch. Since operation the dyspnoea is Grade I and there is no orthopnoea. Tricuspid incompetence persists and she requires diuretics to keep her free from oedema. The electrocardiogram (Fig. 2) now shows absence of right ventricular hypertrophy and the radiological appearances are unchanged except for further cardiac enlargement (C.T.R. 64%).

Combined Mitral Stenosis and Mitral Incompetence

We are in general agreement with Belcher[5] that good results are obtained by valvotomy in a small number of patients with stenosis plus incompetence of greater than 'insignificant degree'. Nevertheless, the proportion of good results falls short of that obtained in patients without incompetence and, even though the surgeon has reported 'no increase of regurgitation' at operation, the clinical course may continue steadily downhill. It would seem that these patients are following the natural history of mitral incompetence and that their stenosis has not been a factor in causing symptoms.

Case 7

A 22-year-old female gave a history of breathlessness progressing over several years to Grade III[6] with associated orthop-



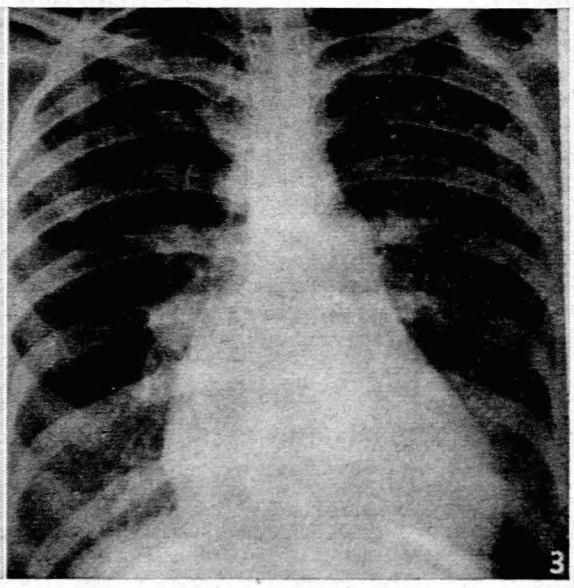


Fig. 1. A=August 1953; B=November 1955; C=August 1957. Electrocardiogram in Case 4, showing right ventricular hypertrophy present in August 1953, absent in November 1955 after successful valvotomy, and again present in August 1957 with the development of re-stenosis.

Fig. 2. X=pre-operative; Y=post-operative. Electrocardiogram in Case 6 showing right ventricular hypertrophy pre-operatively, but no right ventricular hypertrophy post-operatively.

Fig. 3. Teleradiogram of chest in Case 6, showing enlargement of the heart, with prominence of the right ventricle, both atria and the pulmonary artery. The lungs show features of pulmonary hypertension, and 'septal' lines.

noea and paroxysmal nocturnal dyspnoea. Clinically there was evidence of combined mitral stenosis and incompetence. At operation in February 1955, a mobile valve was found with a transverse diameter of 6/8ths inch and a regurgitant stream along the whole length of the valve orifice. The valve was split to 1½ inch diameter and the surgeon noted no increase of regurgitation. Her effort tolerance has not altered at all since operation, though clinically, the mid-diastolic murmur previously present has disappeared.

Other Causes of Poor Results Despite Adequate Post-Operative Size of the Mitral Valve

In some subjects, adequate surgery is performed but the patients experience little or no improvement. Possible explanations for these cases are:

1. Associated valvular defects, which pre-operatively were considered insignificant or were not diagnosed (e.g. tricuspid stenosis), but were in fact significant.

2. The presence or development of essential hypertension or ischaemic heart disease.

3. Unrecognized rheumatic activity.

4. Persistence of chronic bronchial infections in patients in whom bronchial disease has contributed largely to their disability.

5. The development of permanent atrial fibrillation, not present pre-operatively. We concur with other workers, that atrial fibrillation developing immediately after the operation should be terminated, if possible, by quinidine. In those cases where this is not possible, we feel that 3 repeated attempts at intervals of 3-4 months should be instituted, but that anticoagulant therapy should be given for 10-14 days before each attempt, to diminish the risk of embolization.

6. Persistent pulmonary hypertension. There is no unanimity of opinion about the frequency of persistent pulmonary hypertension after adequate valvotomy. That such a state may prevail in some subjects is agreed. Do such subjects remain incapacitated in terms of limitation of effort tolerance? In our series, pre-operative cardiac catheterization was performed on few cases and post-operative catheterization on an even smaller number. Evans and Short[3] emphasized the importance of right ventricular hypertrophy on the electrocardiogram as a sign of pulmonary hypertension in mitral stenosis. In our series there were 34 patients with electrocardiographic evidence of right ventricular hypertrophy pre-operatively. Of these, 15 had complete regression and 8 had considerable diminution of electrocardiographic evidence of right ventricular hypertrophy. Of the 11 patients in whom right ventricular hypertrophy did not regress, only 4 had valvular splits which would at present be regarded as good and, of these 4, 3 have been much improved, with increase of effort tolerance, the 4th patient having a fair result. The remaining 7 patients had poor splits (4 cases), fair splits (2 cases) and grossly induced mitral incompetence (1 case). In these 7 subjects, 6 had poor results and one, with a fair split, had a good result. It would appear, therefore, that persistent pulmonary hypertension may not be common after a good split and, moreover, that even if present it does not necessarily prevent alleviation of symptoms. Its effect on ultimate prognosis cannot as yet be assessed.

There remains, however, a very small group in whom failure to improve cannot be explained. This group is difficult to investigate without accurate objective methods of assessment of disability.

SUMMARY AND CONCLUSIONS

Mitral valvotomy is an established surgical procedure of low mortality and with a high proportion of good and apparently lasting benefits. However, about 30% of subjects have persistent or recurrent symptoms and the assessment and management of these patients becomes a problem, which is discussed in this article. These patients can be placed in several categories with differing requirements for management. Several of the problems posed by these patients are as yet unanswered. In attempting to assess the reason for continued morbidity, the following questions need to be answered:

1. Is the patient having recurrent bouts of post-commissurotomy syndrome?

2. Was the surgical procedure adequate?

3. Has there been recurrent or continued rheumatic activity?

4. Has re-stenosis occurred?

5. Is there evidence of persistent right heart failure and/or tricuspid incompetence?

6. Was significant regurgitation found at operation?

7. Are associated valve defects, or hypertension, or coronary artery disease responsible for symptoms?

8. Is there persistent chronic bronchial disease?

9. Is atrial fibrillation, not previously present, responsible for disability?

10. Is there persistent pulmonary hypertension?

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REFERENCES

1. Wood, P. (1954): Brit. Med. J., **1**, 1051 and 1113.
2. Papp, C. and Zion, M. M. (1956): Brit Heart J., **18**, 153.
3. Evans, W. and Short, D. S. (1957): *Ibid.*, **19**, 457.
4. Lewis, T. (1944): *Diseases of the Heart.* London: Macmillan.
5. Belcher, J. R. (1956): Lancet, **2**, 7.
6. Baker, C., Brock, R. C., Campbell, M. and Wood, P. (1952): Brit. Med. J., **1**, 1043.
7. Smith, J. A. and Levine, S. A. (1942): Amer. Heart J., **23**, 739.