SURGERY FOR MITRAL STENOSIS

Part II. Mortality in Mitral Commissurotomy

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EVALUATION of a new surgical procedure such as mitral commissurotomy must depend upon consideration of its total cost as compared to the benefits obtained by its use. Functional improvement due to mitral commissurotomy can now be assessed only on a short-term basis. A number of years must pass before the ultimate results of combined surgical and medical management can be ascertained. At present, the value of surgical intervention in properly selected patients with mitral stenosis appears to be clearly established.

In assessing the cost of the procedure, the mortality and morbidity involved are the primary considerations. Of these, mortality is more important and more easily defined. The first mitral commissurotomy at the Cleveland Clinic was performed in May 1951, and the 100th operation of the consecutive series in December 1953. Of the 100 patients, 92 survive. The purpose of this report is to analyze the causes of death in the early postoperative period with the aim of determining such measures as may make the operation safer in the future.

Of the eight patients, six died in the hospital after operation; two died of their disease after discharge from the hospital, one approximately two months after discharge and the other more than a year later. The cause of death in each of the six who died during postoperative hospitalization was determined by postmortem examination (table 1). The two patients who died elsewhere after discharge were not observed during the terminal phases of their illnesses. Postmortem examinations were obtained in both cases. Although the complete findings are not yet available to us, the two patients are presumed to have died of their disease.

Before this operation was undertaken, an attempt was made to anticipate the complications that might be encountered. Ventricular arrhythmias and uncontrolled hemorrhage at the time of surgery were the most feared complications but did not materialize. Death in the operating room did not occur. There was no instance of cardiac arrest or ventricular fibrillation that required cardiac massage or electroshock defibrillation. The causes of the six early postoperative fatalities can be grouped under three categories: (1) cerebral

TABLE 1

Analysis of Factors in 6 Hospital Deaths after Commissurotomy

Case No.	Sex	Age (Yr.)	Preoperative Evaluation	Operative Findings	Time of Death	Cause of Death	Postmortem Findings	Preventable or Not-Preventable	Comment
1	F.	37	Pure stenosis, fibrillation, cong. failure.	Pure stenosis. Good valvulotomy.	2nd p.o. day.	Cerebral embolus.	Embolus rt. middle cerebral artery.	Not preventable.	Occurred several hr. p.o. Occluding vessels would not have helped.
2	F.	26	Pure stenosis.	Pure stenosis. Good valvulotomy.	4th p.o. day.	Shock and tamponade, pericardial bleeding.	Pulm. edema cardiac hypertrophy.	Preventable.	Considered an error of technic.
3	F.	41	Pure severe stenosis, marked cardiomegaly, fibrillation, pulm. vascular disease.	Pure stenosis, fresh thrombus in auricular appendage. Good valvulotomy.	1st p.o. day.	Cerebral embolus.	Large mural thrombus, embolus left middle cerebral artery.	Not preventable.	Vessels occluded during surgery. No neurologic signs until several hours postoperative.

4	M.	42	Pure stenosis, left pleural effusion.	Pure stenosis. Good valvulotomy. Old pleural thickening.	10th p.o. day.	Probable cardiac arrhythmia, ? quinidine toxicity.	No significant findings.	Probably preventable.	Quinidine Rx for postoperative fibrillation.
5	M.	47	Stenosis and insufficiency, severe pulm. hypertension, auricular fibrillation, diffuse pulmonary fibrosis.	Markedly calcified valve, predominant stenosis, severe pulmonary disease. Fair valvulotomy.	11th p.o. day.	Cardio-pulmonary failure.	Severe unclassified pulmonary fibrosis.	Not preventable.	Calculated risk, pulmonary insufficiency probably chief cause of death.
6	M.	56	Pure stenosis, cong. failure, marked myocardial insufficiency.	Pure stenosis. Good valvulotomy.	15th p.o. day.	Cardio-pulmonary failure.	Purulent bronchitis, broncho-pneumonia, multiple thromboses, arterioles of lung. Empyema.	Not preventable.	Calculated risk.

embolization, (2) error in technical management, and (3) the bad-risk patient. Appreciation of the significant factors in each category may lead to a reduction in the current rate. If so, mitral commissurotomy performed under proper circumstances will have a mortality rate of perhaps 2 to 3 per cent.

1. Cerebral Embolization. Embolization has long been recognized as a frequent complication or valvular heart disease, especially in the presence of auricular fibrillation or congestive heart failure. Observations during operation have shown that thrombi commonly occur in the left auricular appendage and within the atrium and that thrombi within the right auricle and systemic veins are also common. Hence, embolization is a frequent hazard and probably will remain an unpredictable factor in mitral valve surgery. Two of our patients died of cerebral infarction due to emboli which were dislodged during or shortly after operation. Both of these patients were young women with pure mitral stenosis in whom good surgical outcomes were anticipated. Postmortem examination in each case revealed the source of the embolus to be within the left auricle at the junction of the auricular appendage.

Embolization following mitral commissurotomy is by no means confined to the brain and is not always fatal. Clinical evidence of embolization occurred in six other patients, all but one of whom have recovered without residual effects. Three of these were probably spared cerebral emboli by the technic of intermittent occlusion of the innominate and left carotid artery, suggested by Bailey.[1] This measure is designed to divert a fragment of clot or calcium to the descending aorta, and was employed as a routine measure in most patients of this series. Two emboli to the lower extremities, and one embolus to the mesentery occurred when this technic was employed and all patients recovered without further surgical intervention. In spite of all present precautions, embolism will continue to be a definite factor in operative mortality.

2. Errors in Management. The surgical care of the patient with mitral stenosis has become a well-regulated hospital procedure. Nevertheless, there are numerous details that apply solely to individual cases and offer unsuspected pitfalls. Two hospital deaths were attributable to errors in technical management. The loss of these two patients was particularly tragic, as both had had valvulotomies that promised excellent clinical results; in retrospect both deaths were avoidable. One patient, a 26 year old woman (case 2), was re-explored on the third postoperative day because of cardiac tamponade. It was thought that there was a leak in the auricular appendiceal stump. The bleeding, however, was from a small, pericardial vessel at the base of the superior pulmonary vein. It was easily ligated, but the patient did not recover. Failure to insure adequate hemostasis at the time of valvulotomy is a surgical error.

The second patient, a 42 year old man (case 4), had a technically satisfactory operative procedure. On the fifth postoperative day, he developed auricular fibrillation. After his ventricular rate was controlled with digitalis, gradually increasing doses of quinidine were employed in an attempt to restore sinus rhythm. On the ninth and tenth days after operation, he received 0.6 Gm. of

quinidine sulfate every four hours. He died unexpectedly in his sleep during the night of the tenth postoperative day, and postmortem examination demonstrated no apparent cause. It is assumed that his death was due to ventricular fibrillation, precipitated by quinidine. The incrimination of quinidine in this case is purely presumptive as no evidences of quinidine intoxication were recognized. The drug had been used frequently before and has been used since without difficulty. Of 49 patients with sinus rhythm before operation, 25 have developed auricular fibrillation during the early postoperative period. It has been possible to restore normal sinus rhythm in 22 of these patients. In every instance, successful treatment was accomplished by the use of procaine amide, quinidine, or a combination of the two drugs.

3. The Bad-Risk Patient. Selection of the surgical candidate may be hazardous in any new surgical procedure. There are seriously ill patients with rheumatic heart disease to whom all medical therapy had been offered and has failed and whose surgical indication is desperation itself. In most of these, disability is due to a combination of factors rather than to mitral stenosis alone. Myocardial damage, associated valve deformities, and superimposed pulmonary disease are formidable in the surgical candidate. Obesity, malnutrition, and arteriosclerosis also are important factors in estimating the individual's ability to recover from operation. When adequate and prolonged medical management is incapable of resolving all objective manifestations of congestive heart failure, at least temporarily, the patient is not considered a candidate for surgery.[2]

There are, however, other patients who are not obviously disqualified. These may show 40 to 60 per cent cardiac enlargement, very severe pulmonary hypertension, and pulmonary fibrosis and emphysema, but respond to medical measures by resolution of the signs of heart failure. When such patients have mechanically severe mitral stenosis, they may be greatly benefited by mitral commissurotomy. This series includes a number of these, of whom two died. Most of those who survived have shown surprising recovery and effective rehabilitation. These two deaths occurred in men, aged 47 (case 5) and 56 years (case 6), who were accepted for surgery as calculated poor risks. The hazard was explained in detail to the patients and to their families. Both patients were given prolonged preparation in the hospital before surgery, and both demonstrated sufficient improvement in their over-all status to brighten an otherwise gloomy prognosis. In each case, the immediate postoperative course was difficult, but serious trouble was not encountered until the end of the first week.

On the eighth postoperative day, the one patient (case 5) became progressively dyspneic with prolongation of the expiratory phase of respiration and the development of moderate cyanosis. He presented the clinical picture of intractable bronchial asthma with progressive anoxia. Death occurred on the eleventh postoperative day. There had been no evidence of pulmonary edema or of increasing systemic congestive changes. There had been no major bronchial

obstruction by mucous plugs, such as is frequently encountered in patients who are unable to cough up bronchial secretions in the first few postoperative days. Postmortem examination revealed extremely severe chronic pulmonary disease, characterized by diffuse focal fibrosis of alveolar membranes, interlobular fibrosis, diffuse occlusion of bronchioles by mucous plugs, and severe generalized pulmonary vascular disease involving both the major vessels and the precapillary arterioles. We consider this pulmonary disease to be irreversible.

The sixth patient was the oldest in the entire series. In addition to long-standing congestive heart failure, which had been temporarily cleared by medical management, there was evidence of moderately severe, generalized arteriosclerosis. Postoperatively, wound healing was delayed, and on the eleventh postoperative day, he suddenly developed a pressure pneumothorax on the left. This was complicated by the development of empyema, and death occurred on the fifteenth postoperative day.

Timely recognition of the patient who will not be able to tolerate the postoperative course of mitral commissurotomy will always be a matter of individual judgment and experience. It is now obvious that neither of these two patients were suitable candidates for surgery; nevertheless, a large number of similar poor-risk patients have survived the operation and have demonstrated appreciable improvement.

SUMMARY AND CONCLUSIONS

Surgery for mitral stenosis is now an established procedure. Although its long-term benefits have yet to be ascertained, the mortality rate of this operation compares favorably to mortality rates of other types of elective major surgery. The technic is standardized, and deaths during operation rarely should occur.

In a series of 100 mitral commissurotomies performed at the Clinic between May 1951 and December 1953, eight deaths occurred, six in the early postoperative period. Causes of death in the six are analyzed and categorized.

1. Cerebral embolization. In the good-risk patient with symptomatic mitral stenosis, the greatest danger is embolization. Two patients in whom a good surgical outcome was anticipated died of cerebral infarction due to emboli which were dislodged during or shortly after operation. Three of six additional patients who manifested clinical evidences of embolization were probably spared the development of cerebral emboli by the Bailey technic of intermittent occlusion of the innominate and left carotid artery.

2. Errors in management. Although the surgical care of these patients is a well-regulated hospital procedure, two deaths were attributable to avoidable errors in technical management. One of these patients was re-explored because of cardiac tamponade. A tiny vessel in the pericardium at the base of the superior pulmonary vein was easily ligated but the patient failed to regain consciousness. The second death occurred after a technically satisfactory procedure and was probably due to ventricular fibrillation, attributable to the use of quinidine. The incrimination of quinidine is presumptive. Anticipated fatali-

ties from cardiac arrest and uncontrolled hemorrhage did not occur in this series. This may be ascribed to careful preoperative preparation, light anesthesia, and continual electrocardiographic monitoring during operation. This last precaution has made it possible to recognize potentially dangerous alterations in heart rhythm before they become apparent in the surgical field.

3. The bad-risk patient. It is not possible to be dogmatic about the selection for surgery of patients with very severe rheumatic heart disease and mitral stenosis. There are several factors, however, which influence the final decision to select or to reject the individual candidate for commissurotomy. It must be emphasized that the mechanical effect of mitral stenosis is only one factor in the production of symptoms in many patients with rheumatic heart disease. Intractable myocardial insufficiency may preclude any benefit from surgery. The patient with more than 40 per cent cardiac enlargement presents a greatly increased surgical risk, and desperation alone is not an indication for surgical intervention. The importance of an adequate pulmonary reserve has been stressed. Irreversible changes may exist in the lung even though myocardial function is adequate. Two patients in this series who were calculated poor risks died during postoperative hospitalization.

Although an early postoperative mortality of 6 per cent may be considered reasonable, this figure should be, and can be reduced. We believe that, with judicious consideration of the foregoing categories, mitral commissurotomy can be carried out under proper circumstances with an expected mortality rate of 2 to 3 per cent. A mortality rate of less than 2 per cent should be expected if only young patients with uncomplicated mitral stenosis were to be selected for surgery, but such a timorous policy would reflect little credit on the surgeon and do a grave injustice to many patients.

References

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