Arrhythmia,
An Indication for Valvotomy in Mitral Stenosis;
Concomitant Lobectomy for Bronchiectasis

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Though surgical treatment for mitral stenosis was first suggested in 1902 and some operations were performed on mitral valves of patients as early as 1923, serious and widespread concern with criteria of selection of patients with mitral stenosis for surgical treatment really began with the first successful mitral operations by Bailey and his group in Philadelphia, and Harken and his group in Boston, and very shortly thereafter by others throughout the world.

Methods for selection of cases for mitral commissurotomy have been reported by many. While there is not unanimity of opinion on all factors concerned, and indeed there are still some dissidents to the operation itself, there is a major agreement on many features.

In general, patients with mitral stenosis who are in Class I (functional classification of the New York Heart Association) do not need the operation. Patients in Class IV, especially if well advanced, have passed the optimum time for surgery, though many can be saved and some substantially improved. Class II is a debatable area in selection. Class III is at present the one of choice.

Selection of cases now is usually based on clinical, radiological, electrocardiographic, and hemodynamic findings. The mitral stenosis should be tight and pure if possible. Associated minor cardiac involvement such as mild mitral insufficiency, slight aortic valvular lesions, with no or only minimal left ventricular enlargement, do not contraindicate the operation. Associated tricuspid insufficiency often makes the indication for surgical treatment more urgent. Symptoms or signs of significant disturbance of cardiac function are a usual requirement, and if these are not severe, they should show recent progression, not due to intercurrent complications. Auscultatory findings are usually the most helpful in the diagnosis of the valve lesions. At the apex, the fout-ta-ta-rou of Duroziez, with a loud slappy first sound, a loud opening snap followed by a long rumbling diastolic murmur, with a crescendo presystolic murmur if sinus rhythm is present, and, a loud P2, form the characteristic pattern. Radio-
logical examinations may aid, especially in locating significant left ventricular enlargement, an important contraindication, or a giant left auricle, to suggest the probability of significant mitral insufficiency. Moderate systolic pulsation of the left atrial border, on the barium filled esophagus, or elicited by other methods of examination, is a variable and unreliable sign of significant and prohibitive mitral insufficiency. Calcification of the mitral valve is often readily determined. Its presence, with other indications, may make surgery more urgent, because of the probability of its increasing, and then corrective surgery would be more difficult, dangerous, or impossible. On the other hand its presence may suggest a more careful search for mitral insufficiency. The use of angiocardiography as an aid in the selection of cases for surgical treatment is being developed.

Electrocardiographic criteria, so far, have been concerned mainly with evidence of left or right ventricular hypertrophy. Measurements of the hemodynamics formerly seemed necessary in the selection of cases for surgery. Now these are performed necessarily, in only a small number of candidates, about 5 per cent at one large center. Pulmonary resistance, pulmonary hypertension, and pulmonary capillary pressure curves have been variously assessed as aids in the differential diagnosis and in the selection of cases. Size of the mitral valve orifice is often estimated, but calculation may be erroneous as in a recent case where the orifice was estimated as 0.6 cm. and at surgery this mitral valve was found to be perfectly normal.

Little has been reported on arrhythmias in criteria for selection of cases for surgical treatment. The presence of auricular fibrillation is not considered a contraindication to valvotomy and, in one large series, 42 per cent had had this arrhythmia pre-operatively. While it occurs post-operatively, usually ephemerally, it is of interest that many patients lose their atrial fibrillation after surgery, with or without the use of anti-arrhythmic medication. Nevertheless, atrial fibrillation has not been a consideration in the appraisal of the patient's condition for mitral commissurotomy. Atrial fibrillation is known to be detrimental to cardiac function and occasionally seriously so when it is responsible for a rapid ventricular rate or for the production of thrombi and emboli. Surgery of mitral stenosis now has a low mortality rate and morbidity. Atrial fibrillation with uncontrollable ventricular rate has been considered a contraindication to commissurotomy. We believe the presence of arrhythmias, such as atrial fibrillation, flutter or tachycardia may be of some importance as criteria for selection of patients with mitral stenosis for surgical treatment. We wish to describe a patient with mitral stenosis, in whom the presence of an uncontrollable, atrial flutter was a major reason for valvotomy and concomitant lobectomy was performed because of associated bronchiectasis.

Case Report: M. G., a 54 year old married white female secretary, born in the United States, was admitted to Beth Israel Hospital July 6, 1954, because of rapid heart rate. This was her fourth admission to this hospital. Her history indicated that she had had no known rheumatic fever in childhood, no joint pains, and had not been restricted in her physical activities. She had had three attacks of pneumonia.
in childhood. At 22, on a medical examination preliminary to taking out life insurance, a heart murmur was first noted. About 1941 she developed a hacking cough and became somewhat short of breath, but was able to continue working regularly. Nasal polyps were removed and she received treatment by an allergist who found her skin sensitive to egg, ragweed and roses. Some improvement followed polypectomy and treatment with injection of allergens. Several electrocardiograms were taken, the last in June, 1947, and all were reported normal. About July 1, 1947, she developed some weakness and stayed at home for a few days. Increased cough with thick mucopurulent expectoration, wheezing, marked shortness of breath, and ortho-
stantly present at both lung bases and about four to five ounces of mucopurulent sputum were coughed up daily.

In June, 1954, after failing to adhere to the salt poor diet and the limitations on physical effort, she experienced an episode of pulmonary edema. This was treated at home. About one month later, she had a recurrence and received cedilanid, intravenously, in addition to morphine, aminophyllin, and her regular digitalis. Her heart beat continued to be 170 to 180 and therefore she was sent to the hospital, July 6, 1954. On physical examination at her fourth admission she appeared moderately dyspneic and orthopneic and had a slight cyanotic tinge to the cheeks. There was

FIGURE 1: (A) Atrial fibrillation; lead CF; Nov. 3, 1950. (B) Regular sinus rhythm, rate 47 per minute; lead 2; Aug. 11, 1954. (C) Atrial tachycardia (flutter) 2:1 and 3:1 A-V block; lead 2; July 31, 1954. (D) Same rhythm, 6:1 A-V block after carotid sinus pressure; lead 3; July 29, 1954. (E) Atrial tachycardia (flutter), rate 115 to 187; lead 3; Oct. 2, 1954, 2 days before surgery. (F) Oct. 4, 1954, during valvotomy, 11:03 a.m., atrial tachycardia, rate 136; lead 3. (G) During valvotomy, 11:22 a.m., regular sinus rhythm, rate 94; lead 3.
cough productive of greenish yellow phlegm. The temperature was 99.8° F. The respirations were 24 per minute. The eyes, ears, nose and throat were essentially normal. She was edentulous. The thyroid was not palpably enlarged. The sublingual and neck veins (patient sitting) were distended. Wheezes and moist rales were heard throughout the lungs, more at the bases. The heart rate was about 170 per minute. When it became slower, about 110, the following heart sounds and murmurs were heard. At the apex the first sound was loud and slappy. An opening snap followed by a moderate, low pitched, rumbling diastolic murmur, louder in presystole, was heard. The second sound was slightly increased in loudness. Later, with regular atrial rhythm and a rate of 70 to 80, the diastolic murmurs were diminished and a Grade II systolic murmur was heard at the apex. The blood pressure was 115/85. The liver was palpated two finger breadths below the costal margin. The spleen was not felt. There was slight pitting edema about the ankles. No clubbing of the toes was found. Dorsalis pedis pulsations were palpable only on the right. The diagnosis was rheumatic heart disease, mitral stenosis, probably tight, with mild mitral insufficiency; supra-ventricular tachycardia, cardiac failure, right and left sided; chronic bronchitis; diabetes mellitus, and cholelithiasis. A urinalysis disclosed a positive test for glucose, specific gravity of 1.010 to 1.015, and 4 to 6 white blood cells in the low power field. The hemogram was normal. Erythrocyte sedimentation rate was 87 mm. (August 9) and 30 mm. (September 8) in one hour (Westregen). The blood was sterile on culture. The electrocardiogram (July 16) revealed the presence of a tachycardia with "P" waves at a rate of 126 per minute, but with the possibility that a second "P" was buried in the T wave in each cycle. "P" waves were not seen in lead 1, inverted 0.5 mm. in leads 2 and 3, upright in aVR, flat in aVL and inverted in aVF. In V1, "P" was upright 0.5 mm., and 0.05 sec. duration, flat in V5, V6, T waves were not visible. The P-R interval was 0.12 sec. (VI). The electrical position was vertical, QRS being almost entirely negative in aVL and positive in aVF. S in V1, R in V5, V6 was only 2 mm.; no evidence of either left or right ventricular hypertrophy. RS-T was depressed about 0.5 mm. in lead 1 and 3, and 0.8 to 1.0 mm. in lead 2, with a configuration similar to that due to digitalis. RS-T was depressed in the left precordial leads, 2.0 to 2.5 mm. in V1, and V5. The T wave was upright and small in the standard limb leads and flat in V1, V5, and V6. The cardiac regime was continued. It included a low sodium diet, 200 mg., and digitoxin, which was increased to 0.2 mg. daily. No insulin was given the first few days since most of the fractional urine specimens were negative for glucose.

Treatment directed at the tachycardia included administration of quinidine up to 0.4 Gm. every two hours for five or six doses daily, omission of digitalis for 12 days, redigitalization with digitoxin 0.9 mg. followed by 0.2 mg. daily, the use of cedilanid 1.6 mg. intravenously, given in nine hours, prosteny up to 5 Gm. daily, and potassium chloride 4.6 Gm. daily. Strips of electrocardiogram were recorded many times each day. During most of the period to August 17 the atrial tachycardia was present with R and P inverted. Ventricular rates were between 100 and 150, and during several short episodes 180 to 200.

The blood serum potassium, July 28 was 4.7 mEq. per liter; on August 2 the NPN was 31 mg. per 100 cc., sodium 138.4 mEq. and potassium 5 mEq. per liter. On August 25 the atrial tachycardia had been present continuously for the previous eight days, and rales had not been given a good time. In test again for the possible presence of digitalis toxicity as a cause of the atrial tachycardia. The acetyl-strophantidin test for digitalis toxicity was performed. Just prior to the test the electrocardiogram revealed the presence of the atrial tachycardia with "P" inverted in leads 2 and 3, atrial rate 200, ventricular rate 120 to 135. The drug was administered intravenously 1.2 mg., over a period of 12 minutes, with monitoring by the continuous electrocardiogram. Except for the appearance of three scattered premature ventricular beats (at two to three minutes and 0.45 mg.) no other variations were noted. Added carotid sinus massage was ineffectual. Two Gm. of quinidine had been given during the day. At 8:00 p.m. the apical rate was 100 and fairly regular. On August 26 the acetyl-strophantidin test was performed with a double dose; 2.4 mg. were given intravenously over a period of 27 minutes. There was no effect on rate or rhythm on the continuous electrocardiogram. The ventricular rate was about 140 to 170. Atrial waves were not distinguishable in the electrocardiogram before, during and just after the test. No untoward effect was observed. The results of the two tests were interpreted as failing to indicate the presence of digitalis toxicity, or that the atrial tachycardia was digitalis induced. Nevertheless treatment was continued with prostenyl 3 to 5 Gm. orally and potassium chloride 4.5 Gm., per day. The atrial tachycardia persisted, with the ventricular rate between 105 and 120. By September 13 she had been in the hospital 69 days. During this time the atrial tachycardia had been continuous with the exception of about six days in the previous month. Frequent and sudden elevation of the ventricular rate to 170 to 200
was a serious threat to life. Moderately large doses of proestynl with potassium chloride, often difficult to take, were required to reduce the ventricular rate. These drugs, also quinidine, digitalis, and prostigmine, as used, were unable to break the arrhythmia permanently or to control the ventricular rate adequately. On search for other therapy, it was thought that possibly the left atrial distention due to tachycardia, or a predisposed chamber of the mitral orifice, might be an important factor contributing to the disturbance of cardiac rate and rhythm. Its relief by surgery then seemed rational. After a day at home she returned to the hospital September 14 to be evaluated more completely for mitral commissurotomy, and prepared for the surgery, if feasible. By fluoroscopy and x-ray film the cardiac silhouette showed prominence of the pulmonary artery segment. On the barium filled esophagus the impression of the left atrium indicated moderate enlargement. There was only slight systolic pulsation of the left atrial border. The right ventricle was thought to be enlarged. The auscultatory findings were as previously described and diagnostic of mitral stenosis. The cardiac symptoms even prior to the tachycardia, if due to the mitral stenosis, strongly suggested the presence of marked narrowing of the mitral orifice. The diabetes required 20 units of insulin before and in the early part of her present hospital stay. On the hospital diet, and with her loss of weight, the insulin requirement became less and from August 22 on, insulin was omitted. Sugar in the urine was recorded as a trace or zero, and acetone was never found. The patient's temperature did not rise above 99.8° F. with the exception of one brief episode of fever, July 17 and July 18, which disappeared after several injections of Dicrysticin. About five ounces of greenish yellow mucopurulent sputum continued to be produced daily. Culture of this showed occasional streptococcus viridans, non-hemolytic streptococcus and micrococccus catarrhalis. Active rheumatic fever was a possible cause for the cardiac arrhythmia which had been carefully considered. The antistreptolysin titer was elevated; 100 units on September 20, 1000 units on September 24 and 700 units on September 30. As a therapeutic test and for possible beneficial effect, cortisone, 75 mg. daily for two weeks, was given (September 22 to October 4) but failed, at this dosage, to influence the auricular tachycardia. A bronchogram, on September 30, with Dionsil, revealed the presence of extensive saccular bronchiectasis in all segments of both lower lobes, more severe on the left. The lingular bronchus of the left upper lobe and the right middle lobe bronchus did not fill completely. Because of the pulmonary findings it was thought that left lower lobectomy might be done at the time of commissurotomy. Consent for this additional procedure was obtained from the patient. From September 14 to 25, while being prepared for surgery, the patient continued to receive proestyn, 3.75 to 5.0 Gm. by mouth daily, except on September 20, when only 2 Gm. were taken; also potassium chloride 4.5 Gm. daily. There were frequent rises in the ventricular rate to about 140 per minute and on September 25 the apical rate rose to 200. Digitoxin was then restarted; none had been given for one month, since August 26; 1.2 mg. were given orally in 48 hours, then 0.15 mg. daily; proestyn 5 Gm. daily and, because of some dyspnea, the mercuhydrin, 1 cc., was increased in frequency to daily for three days. In six hours the ventricular rate slowed to 108 to 120. However on October 2 it was 200 once more. Prostigmine in doses of 0.25 mg. and 0.6 mg. was given to a total of 3 mg., but the apical rate remained between 130 and 180. On October 4, the morning on which the operation was scheduled, her cardiac rate, even after proestyn 750 mg., prostigmine 0.5 mg. and secoral 0.2 Gm., was 140 to 150. The anesthesiologist was reluctant to begin. However, since a rapid heart rate (160 to 200) had been present for two days almost continuously, and since it was for the elimination of this tachycardia that the surgery was in large part being urged at this time, since cardiac failure was still minimal with the probability of increase, and since there had been several days of considerable surgical preparation, it was decided to start the anesthesia and proceed with the operation. The heart rate and electrocardiogram were continuously observed on the cardio scope.

Operation (L. A. S.): The patient was placed on the table in the left lateral position with the head and chest somewhat elevated. The chest was opened through an incision in the periosteal bed of the fifth rib. The anesthesiologist had been having difficulty because of the large amount of sputum emanating from the left lower lobe. The left lower lobe bronchus was exposed promptly and clamped after ligation and division of the pulmonary artery branches to the lobe. Following this there was no difficulty with secretions. On the cardio scope the ventricular rate was recorded as 140 to 150 per minute, with atrial tachycardia, and P, and P, inverted. The blood pressure was between 80 and 100 systolic. At about 11:20 a.m. the phrenic nerve and vessels were mobilized and the pericardium incised. There was a sudden reversion to regular sinus rhythm with a drop in rate to 95; the blood systolic with concurrent improvement in the color of the tissues. There was a marked diastolic thrill at the apex of the left ventricle. The left atrium was greatly enlarged. Old organized clot was present in the atrial appendage. A systolic regurgitation jet of moderate degree was present and palpable. The posterior leaflet (mural) of the mitral valve contained much calcium in its edge and was thickened and shrunken. The mitral orifice was
a slit like structure which could be opened easily and admitted one and a half fingers. But the super position of the edge of the posterior leaflet caused an increase in the stenosis functionally. The anterior lateral commissure was cut and then digitally fractured to the muscular wall. The short posterior medial commissure was not cut. At this point the diastolic thrill at the apex of the heart had disappeared. The left lower lobectomy was then completed. Except for one minute, about 13 minutes after its cessation, the atrial tachycardia did not recur throughout the rest of the operation, a period of about 90 minutes, and the cardiac rate did not exceed 120. The postoperative reaction was not unduly severe. The patient received oxygen by nasal catheter; 1000 cc. of blood by slow intravenous infusion were given over a period of

FIGURE 2: (A) Oct. 6, 1954, 48 hours after operation; episode of atrial tachycardia, P waves upright, lead 3; no subsequent episodes with such rapid atrial rate. (B) from left to right, leads 1, 2 and 3. Sinus tachycardia, rate 125, upright P in lead 3. (C) Nov. 23, 1954, 60 days after operation. From left to right, leads 1, 2, 3, aVR, aVL, aVF, V1, V2, V3, V4, V5 and V6. Regular sinus rhythm, rate 70; digitalis effects.
six hours. There was some difficulty in expectorating and coughing, and large amounts of purulent material were obtained at each suctioning. At 3 hours after operation the heart rate was 112. Twenty four hours later she "dangled" off the edge of the bed for 10 minutes; the heart rate was 120, respirations 20, and blood pressure 114/80. Forty-eight hours after operation there was an episode of tachycardia with an atrial and ventricular rate of 160 to 170 per minute. The electrocardiogram (Fig. 2A) showed this to be different from the preoperative tachycardia in that P1 and R, now were upright and not inverted. Medication administered had to be given parenterally. Acetyl-strophanthidin, quinidine gluconate, benedryl and amytal were given over a period of several hours. Slowing of the heart rate occurred during the night, and the next day there was a regular sinus rhythm with a rate of 84 per minute. By October 19 (15 days after operation) the patient showed signs of considerable improvement; the cyanotic tinge to the cheeks was no longer present; the cardiac rate was 80 and regular; the cardiac sounds had changed from before surgery. The first sound at the apex was less accentuated; the opening snap and diastolic murmur were not heard. A grade 1 to 2 (6 maximum) systolic murmur was present; P1 was not accentuated; there was no evidence of venous hypertension, sublingual and neck veins were not unduly filled. Rales were present at the right lung base, and about ½ to 1 ounce of sputum was expectorated daily. For the three weeks up to the time of her discharge from the hospital (November 17), her temperature was normal, though the ESR on November 3 was still elevated, 57 mm. For a few hours on October 11 and October 28 there was an increase in cardiac rate to 130 and 170 per minute, respectively, but the electrocardiogram revealed P waves in lead 2 and 3 to be upright. After her return home the digitoxin was continued at 0.1 mg. daily, quinidine 0.2 Gm., three to four times daily; the diet was moderately restricted in sodium. Restrictions on physical activity were slowly removed. There were several episodes, each of a few minutes to about one hour, of increase in cardiac rate, which the patient felt and made note of. The heart rates at the apex taken then by the patient and doctor were about 100 to 120. In two of these periods the electrocardiogram was recorded and revealed a regular sinus rhythm with upright P waves in all standard limb leads. She returned to her regular work on January 13, 1955, 100 days after the comissurotomy and lobectomy, and up to the time of writing, two months, has been working regularly. During this period there has been no recurrence of the atrial tachycardia chiefly for which valvotomy was undertaken.

Pathological Findings

The specimen of atrial appendage (posterior) was 2 x 2 x 0.7 cm. The endocardial surface was smooth and glistening. No Aschoff bodies were seen. Left lower lobe of lung with 2 cm. of main stem bronchus, measured 18 x 7 x 2.5 cm. and weighed 150 Gm. The pleural surfaces were smooth and glistening over the entire posterior surface except where there were fibrous strands adherent to the pleural surface; this was most marked over the posterior basal surfaces. The anterior, posterior and lateral basal branch bronchi were all dilated and filled with viscid white secretions. They could be traced to the periphery. The pulmonary arterioles showed considerable thickening of the musculature and some narrowing of the lumen. The smaller arteries also had considerable hypertrophy of the muscular layer and subintimal deposition of lipid material. There was some thickening of the alveolar septa.

Discussion

There were several unusual problems in diagnosis and treatment of mitral stenosis in the case described above:
1) separation of certain symptoms and signs of mitral stenosis and cardiac failure, from those of chronic bronchiectasis,
2) estimation of the tightness of mitral stenosis,
3) the nature and cause of arrhythmia,
4) the treatment of the cardiac and pulmonary conditions.

The patient had been observed over a period of seven and a half years,
during which time increase of the cardiac and pulmonary symptoms and signs were noted. Progression of the cardiac disease was seen in the gradual increase in the shortness of breath and orthopnea, the development of paroxysmal nocturnal dyspnea, venous hypertension, enlargement of the liver and slight edema about the ankles. Treatment with digitalis, periodic mercurial diuretics and sodium poor diet was necessary and helpful during the last few years. Bronchitis is common in tight mitral stenosis. The diagnosis of chronic bronchiectasis, however, was based on several features: history of several attacks of pneumonia during childhood, and also later; chronic hacking cough since childhood, with the production of four to five ounces of mucopurulent sputum daily for many years. This diagnosis was established by the bronchogram, corroborated at surgery, and finally by the gross and microscopic examination of the lung removed at operation. Chronic bronchiectasis undoubtedly was responsible for much of the cough, the expectoration, and some dyspnea; also for most of the rales heard over both lungs many years, for in spite of the many rales there was often little or no dyspnea and the patient was able to work regularly. The bout of fever early in the last hospital stay subsided promptly with antibiotic therapy, as did several such previous bouts, and therefore seemed caused by the pulmonary infection. Elevated sedimentation rate, antistreptolysin titer and fever, need not be due to rheumatic activity.

The diagnosis of tight mitral stenosis was based upon several features. Important in this diagnosis was the onset of pulmonary symptoms due to the cardiac lesion. The pulmonary manifestations of "tight, non-regurgitant" mitral stenosis were reported from this hospital almost 25 years ago by Held and associates. Frequent pulmonary infections, bronchopneumonia, also chronic progressive pulmonary symptoms may be prominent, and in the past have led to the diagnosis of tuberculosis and pulmonary neoplasm. Similar pulmonary manifestations were present in our case over the period of a few years but were difficult to separate from those of the chronic bronchiectasis. The acute pulmonary symptoms of tight mitral stenosis were clearly described and explained by McGinn and White more than 20 years ago. These acute pulmonary symptoms, pulmonary edema, cardiac asthma or hemoptysis, occurring with physical effort or its equivalent, and due to acute left atrial failure behind a tight and obstructive mitral orifice, therefore have been referred to by us for years as the "McGinn and White" syndrome. Asthmatic-like attacks and pulmonary edema associated with effort or tachycardia, did occur in our patient. Thus the presence of both these chronic and acute pulmonary manifestations strongly suggested that if due to mitral stenosis the orifice was narrow. The auscultatory "ffout-ta-ta-rou" rhythm was considered pathognomonic of the presence of mitral stenosis by Duroziez. With the loud and slappy apical first sound, with a moderately loud opening snap, a long loud mid-diastolic and a "presystolic" murmur it further suggested that the stenosis was marked. The absence of a loud P2 usually associated with pulmonary congestion or pulmonary hypertension has been
noted, P was only mildly to moderately accentuated, grade 2 (6 maximum). The frequently noted absence of a systolic murmur at the apex together with absence of evidence of notable enlargement of the left ventricle in the electrocardiographic and roentgenographic studies, and the absence of marked ventricular systolic pulsation of the posterior left atrial border in the fluoroscopic examination all pointed to the presence of little or no mitral insufficiency. Mild to moderate ventricular systolic pulsation of the posterior left atrial border, we and others have found when no systolic jet was noted at surgery. We believe mild to moderate systolic pulsations of the left atrium may result from the ambient ventricular systolic commotion of the heart and large blood vessels, even in the absence of valvular lesions, especially with the vertically positioned heart. Bramwell has emphasized the importance of distinguishing expansile pulsation of the left atrium from backward displacement of the left atrium by an hypertrophied right ventricle, which he says may occur in pure mitral stenosis. Bulging of the mitral valve with ventricular systole has also been mentioned as a cause of left atrial pulsations. The significance of the absence of large ventricular systolic pulsations of the posterior left atrial border in ruling out marked mitral insufficiency remains to be better evaluated. Esophageal piezocardiograms have been considered helpful in differential diagnosis. The arrhythmia described above, commonly called atrial flutter, because of the A-V block, is closely related to atrial fibrillation and therefore the latter has also been considered in the discussion. It can also be classified as atrial tachycardia, with a rate of about 200, a caudal pacemaker, without T waves and with frequent 2:1 and greater A-V block, and occasional 1:1 response. Recently it has been shown that atrial tachycardias are commonly due to digitalis toxicity, and that concomitant disturbances of the body electrolytes, potassium and sodium, may play a role in their production. Several attempts at evaluation of such an etiology were undertaken and included abstinence from digitalis for long periods, administration of potassium and pronestyl and finally the tests with acetyl strophanthinid. The patient's type of atrial tachycardia of long duration, with the caudal focus and almost no extrasystoles, was not the one usually found in the Lown and Levine group due to digitalis. The results in our trials failed to establish digitalis as the cause of the arrhythmia in our case. The sudden cessation of the arrhythmia during surgery and its continued absence thereafter despite the administration of full doses of digitalis also favor a non-digitalis origin.

Consideration of rheumatic activity as the cause of the arrhythmia was oblige. The nature of this primary disease, the elevated sedimentation rate, cardiac failure and also the high antistreptolysin titer were in its favor. It has been mentioned previously that an elevated erythrocyte sedimentation rate and antistreptolysin titer need not be regarded as positive evidence of rheumatic activity. Many features of the case made one doubt active rheumatic fever the cause of the arrhythmia: the patient's age, 55 years; the long afebrile period during
this arrhythmia; the absence of joint symptoms; the periods with sinus rhythm and normal or slow cardiac rate, both atrial and ventricular, without digitalis; the normal Q-T interval. A therapeutic trial with cortisone, 75 mg. a day for 10 days also failed to influence the arrhythmia. Additional evidence later was the absence of Aschoff bodies or other anatomical evidence of fresh rheumatic activity in the biopsied atrial appendage and in the lung removed. The sudden cessation of the atrial tachycardia immediately with the cardiac surgery, and its continued absence from then on to the time of writing, five months later, also seemed evidence against the presence of fresh rheumatic activity as the cause of the arrhythmia. The mechanism and cause of the arrhythmia were not clear. As a contributory cause, mechanical distention of the left atrium and pulmonary veins was thought of. It had been a common belief that many irregularities of the heart were due to overdistention of its chambers, especially that fibrillation of the atria may so be produced. It was postulated that in a predisposed rheumatic left atrium, distention of the left atrium and pulmonary veins, directly, or through presso-receptors here with efferent connections to right and left atria, might be responsible. On the grounds that by lessening of atrial distention there would be reduction of stimuli through such a reflex nervous mechanism and so possible cessation of the arrhythmia, and also by improvement of cardiac function after reduction of obstruction of the tight mitral valve by valvotomy, there might be improvement of circulation to the atrium, this operation was recommended.

The reasons for starting with the surgical procedure in spite of the presence of the arrhythmia with an uncontrollable, rapid ventricular rate are mentioned above. The ventricular rate of 136 to 150 continued during the induction of the anesthesia, chest incision, and opening of the chest wall and deflation of the lung. A growing trend toward less fear of arrhythmias occurring during surgery encouraged continuation of the operation with careful cardiographic monitoring. When the left phrenic nerve was displaced and the pericardium over the left atrium and in the proximity of the superficial cardiac plexus manipulated, cardiac rhythm suddenly reverted to normal with an atrial and ventricular rate of 95 per minute. The atrial appendage was promptly opened and the orifice of the tight mitral valve was enlarged to two finger-breadths by finger fracture of the anterior lateral commissure. After presence of the arrhythmia for three months in spite of considerable medical treatment for its elimination, its cessation during cardiac surgery directed at its removal, and its continued absence for five months, up to the time of writing, strongly suggest a causal relation between the surgery and this result. The exact mechanism of termination of the arrhythmia is not clear. Its sudden end during operative manipulation in the region of the phrenic and cardiac nerves, and its absence thereafter, suggested reflex action through direct stimulation of the cardiac nervous apparatus aided by reduction of stimulation due to lessening of left auricular distention. Ligation of the left lower bronchus with division of pulmonary artery branches.
may have contributed to the conversion of rhythm through reflex action via the vagus.\textsuperscript{52} It is well known that vagal stimulation may convert atrial flutter to normal action.\textsuperscript{53} In addition to these more direct vagal actions, indirect vagal stimulation through stretch receptors of the atria may be concerned. The atrial presso-receptors have recently received careful study by Whitteredge\textsuperscript{52} and Paintal\textsuperscript{54} and increase of pressure within or distention of the atria was shown to send stimuli over the cervical vagus nerve. Paintal has located receptors for stretch stimuli in both atria; in the left posteriorly, some near the opening of the veins.\textsuperscript{54} Though their function was obscure, he found the impulses were carried through the vagus nerve. Scherf has recently reported\textsuperscript{55} production of atrial fibrillation in one animal experiment when the atrial (right) wall was stretched after cooling, though cooling alone in this one experiment did not cause the arrhythmia. While the explanation of the exact mechanism of the arrhythmia in our case and of the manner of its cessation are speculative, it is factual that it did stop with the cardiac surgery and thereafter. Of considerable importance, is the concept that an uncontrollable arrhythmia with a rapid cardiac rate need not be a contraindication to valvular surgery. But, it appears, as in this case, mitral valvotomy may even be effective in helping to terminate the arrhythmia and so be an additional indication for such surgery, in the presence of other suitable conditions. After conversion of the arrhythmia there was improvement in the patient's general condition, ventricular rate and blood pressure. The lobectomy was then completed without difficulty. Combination of left lobectomy and valvotomy has been reported recently by Bailey\textsuperscript{56}; also right lobectomy for carcinoma of the lung combined with valvotomy for mitral stenosis, has been reported by Shaw,\textsuperscript{57} and Bailey.\textsuperscript{56}

**SUMMARY AND CONCLUSION**

1. A 55 year old woman with rheumatic mitral stenosis and chronic bronchiectasis developed atrial tachycardia (flutter) with rapid ventricular rate. This arrhythmia was uncontrollable though considerable treatment with antiarrhythmics, other cardiac drugs, therapy for digitalis intoxication, and antirheumatic treatment had been tried, over a period of three months.

2. On the possibility that distention of the left atrium due to tight mitral stenosis might be contributory in production of arrhythmia, probably through stimulation of stretch receptors and reflex vagal action, reduction of this distention by mitral valvotomy was recommended.

3. The three month old arrhythmia stopped suddenly during surgery, while cardiac neural structures were manipulated just prior to the dilatation of the tight mitral valve.

4. Arrhythmia has remained absent up to the present time, five months after surgery, and the patient improved greatly and has been working regularly for the past two months.

5. Concomitant lobectomy of the left lower lobe, for chronic bronchiectasis, was performed with the valvotomy.
6. This experience would suggest that an uncontrollable arrhythmia need not be a contraindication to mitral valvotomy, and in a suitable case may be an additional indication for it.

**RESUMEN**

1. Una mujer de 55 años con estenosis mitral reumática y bronquiectasia, presentó taquicardia atrial (flutter) con aceleración ventricular. Esta arritmia fue incontrolable aunque se emplearon antiarrítmicos en consideración así como otras drogas cardíacas, tratamiento para la intoxicación digitalica y tratamiento antirreumático por más de tres meses.

2. Ante la posibilidad de que hubiese distensión del atrio izquierdo debida a acentuada estenosis como factor que contribuyese a la arritmia, probablemente por estimulación de los receptores a la distensión y por acción vagal refleja, se recomendó la reducción de esta distensión por valvulotomía mitral.

3. La arritmia de tres meses de duración se detuvo durante la operación mientras se manipulaban las estructuras neurales cardíacas justamente antes de la dilatación de la válvula estrecha.

4. La arritmia no ha vuelto a presentarse hasta ahora, cinco meses después de la operación y la enferma mejoró grandemente y ha estado trabajando regularmente durante los últimos dos meses.

5. Al mismo tiempo que se hizo la valvulotomía, se realizó lobectomía inferior izquierda por bronquiectasia.

6. Esta experiencia sugeriría que una arritmia incontrolable no es necesariamente una contraindicación para la valvulotomía mitral y que en un caso adecuado podría precisamente ser una indicación para ella.

**RESUME**

1. Une femme âgée de 55 ans, atteinte de rétrécissement mitral d'origine rhumatismal associé à une dilatation des bronches chronique, fut atteinte d'un flutter avec accélération considérable du rythme ventriculaire. On ne parvint pas à faire cesser cette arythmie malgré un effort considérable de traitement avec les régulateurs du rythme cardiaque et toutes les médications cardiaques habituelles, le traitement de l'intoxication digitalique, et le traitement anti-infectieux contre le rhumatisme. Ces essais furent poursuivis pendant trois mois.

2. On envisagea alors la possibilité que la distension des cavités droites en rapport avec la sténose mitrale serrée puisse jouer un rôle dans la production de l'arythmie vraisemblablement par une stimulation des terminaisons nerveuses, et par l'action d'un réflexe neurovégétatif. On envisagea alors la distension de l'orifice par une valvulotomie mitrale.

3. Cette arythmie qui durait depuis trois mois s'arrêta soudain au cours de l'intervention, alors que l'on atteignait les centres nerveux cardiaques exactement avant que l'on procédât à la dilatation de la valvule mitrale.

4. Jusqu'à présent, cinq mois après l'intervention, l'arythmie ne se reproduisit pas, la malade est très améliorée, et depuis deux mois a repris un travail régulier.
5. Au cours de cette intervention sur la valve mitrale fut réalisée la lobectomie simultanée du lobe inférieur gauche, qui était atteint de bronchiectasie.

6. Cette expérience permet de penser qu'une arythmie résistante à la thérapeutique ne doit pas être considérée comme une contre-indication à la valvulotomie mitrale. Il est même possible que dans des cas déterminés, elle puisse en réaliser une indication supplémentaire.

**REFERENCES**


*Complete reference list will appear in reprints.*