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# Surgical treatment of mitral stenosis : with report of a case

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# THE SURGICAL TREATMENT OF MITRAL STENOSIS WITH REPORT OF A CASE

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## Introduction

The past decade has been marked by increasing interest and progress in the development of surgical procedures designed to correct disabilities of the human heart. Certain congenital anomalies (1-3) and notably the patent ductus (4,, 5), have become amenable to surgical correction. More recently,, attention has been directed toward the many reports concerned with intra-cardiac surgery and particularly with the relief of mitral stenosis by commissurotomy. This technique was developed by Bailey and co-workers in Philadelphia (6) and since its introduction in 1948 has been successfully repeated by many others (7-15).

In January,, 1952,, this operation was successfully performed at the University of Nebraska Hospital by Dr. J. Dewey Bisgard. Because of the interest in this procedure and because of its effectiveness in the treatment of a condition which heretofore led to hopeless disability and early death (16), it seems appropriate and timely that the subject be presented. The purpose of this paper is to present the case referred to above along with a review of the current literature with particular reference to the hemodynamic and functional results of commissurotomy.

## Historical Data

In 1924 Cutler (17) published a comprehensive review of the history of cardiac surgery. In 1929, after ten years of investigation and extensive research, he again published a review (18) of the work done thus far. The results were exceedingly discouraging. Yet, in those ten years, unproductive as they may have seemed, much of what is the basis for modern intra-cardiac surgery was learned.

In his very interesting review, Cutler (17) notes that while ancient people did not appreciate the function of the circulatory system, they were, never the less, convinced that wounds of the heart were necessarily fatal. That this concept prevailed until relatively modern times is evidenced by the following quotation from Billroth, 1883, taken from Bland (19): "Let no man who hopes to retain the respect of his medical breathern dare to operate on the human heart." Since at autopsy healed wounds of the heart containing arrow heads and missiles had been noted, it had been suggested (Fisher, 1867; Blotch, 1882; Rose, 1884) that surgery of cardiac wounds might profitably be undertaken. With specific reference to the treatment of mitral stenosis, Sir Lauder Brunton (20), an internist, wrote

with astonishing prophecy in 1902: "On looking at the contracted mitral valve orifice in a severe case of this disease, one is impressed by the hoplessness of ever finding a remedy which will enable the auricle to drive the blood in a sufficient stream through the small mitral orifice, and the wish unconsciously arises that one could divide the constriction as easily during life as one can after death. The risk which such an operation would entail would naturally make one shrink from it, but in some cases it might be well worth while for the patient to balance the risk of a shortened life against the certainty of a prolonged period of existance which could hardly be called life." Billroth's profound influence upon surgery at this time is well known and this, along with the explosive repercussions from his medical colleagues not only proved the validity of Billroth's earlier statement, but also served notice that the medical profession was not yet ready to open this particular frontier. Never the less, with or without the 'respect of medical brethern', experimental and clinical studies were carried out. Cutler (17) reports that in 1897, Rehn reported the first successful suture of the human heart. With the introduction of intratracheal insuflation by Meltzer

and Aver in 1909,, the possibilities for thoracic surgery were remarkably expanded and with it came increased interest in cardio-valvular surgery.

Doyen,,1913, attempted the surgical relief of congenital pulmonary stenosis in a twenty year old woman by passing a tenotome knife through the right ventricle. The patient died a few hours after operation (17). A year later,, however, Tussier (17) accomplished digital dilation of a stenotic aortic valve.. The patient survived and was reported living and improved ten years later.

Operative attempts to correct mitral and aortic valvular deformities during the nineteen twenties were primarily concerned with efforts to pass a tenotome or cardiovalvulotome through the left ventricle and incise the valve cusps. With two notable exceptions, the results were universally fatal due to severe regurgitation.

In 1923 Cutler and Levine (21) using a tenotome and an approach through the left ventricle incised the mitral valve cusps of an eleven year old girl with severe mitral stenosis. The patient lived four and a half years but remained a semi-invalid. It is well worth noting that at autopsy there was no suggestion

of over growth or scar tissue in the healing process of the valve incision.

In 1925 Sir Henry Sautter (22) suggested a direct surgical attack on the mitral valve by approaching it through the left auricular appendage. He operated on a 15 year old girl and had planned to divide the stenotic valve by passing a thin knife along his finger. Upon exploration however, he felt that division of the anterior leaflet would surely augment her already present regurgitation. He therefore abandoned the idea and was content with a finger dilation of the mitral orifice. The patient lived four years with definite improvement but died of a cerebral embolus. In reporting this operation, Sautter wrote as follows: "It appears to me that the method of digital exploration through the auricular appendage cannot be surpassed for simplicity and directness. Not only is the mitral orifice directly to hand, but the aortic valve itself is almost certainly within reach through the mitral orifice. Owing to the simplicity of the structures and oddly enough to their constant and regular movement, the information given by the finger is exceedingly clear, and personally I felt an appreciation of the mechanical reality of stenosis which I

never before possessed. To hear a murmur is a very different matter from feeling the blood itself pouring back over one's finger. I could not help being impressed by the mechanical nature of these lesions and by the practicability of surgical relief."

Cutler (18) in 1929 reported an overall mortality of 83% with no convincing clinical results. Cutler himself felt that an approach through the left auricular appendage would never be practical and in view of his very determined efforts to solve this problem, it is difficult to imagine why he did not give further consideration to Sautter's work.

Efforts to develop intracardiac surgical techniques were virtually abandoned then until very recent years.

There is perhaps no account which is more interesting than that of Bailey (6) who presented his first successful case at the Annual Meeting, American College of Chest Physicians on June 20, 1948 in Chicago. At that time Bailey reported five attempts to operate on the mitral valve, four of which were followed by the death of the patient. Dr. Bailey first attempted to introduce a trochar through the left auricular appendage and incise the valve cusps. He was unable to locate the mitral valve with the canula so he inserted his

finger and manually dilated the valve and he reported that the valve appeared to tear at both commissures. The patient, whose condition was extremely grave to begin with, showed prompt improvement. Because of her improvement and the desperate nature of the risk, the finger was withdrawn andthe auricular appendage closed with no attempt to incise the valve.. The patient improved remarkably for 30 hours post operatively but died 48 hours after operation. At necropsy the valve was found to have been torn and a fibrin clot had accumulated in the orifice. It was at this autopsy that Dr. Bailey conceived the idea of relieving mitral stenosis by incising the commissures.. There were two more unsuccessful attempts before a satisfactory result was achieved.

On June 10, 1948, he performed an incisional commissurotomy on a 24 year old female. His own account (6) of her post operative course is noteworthy: "Post operatively the previously marked presystolic murmur was absent. A pericardial friction rub was present on the first and second post operative days, but was gone by the third. She was out of bed on the third day and walking the fourth. Her greatest difficulty was inability to void for four days post operatively.

On the seventh post operative day the patient had no cardiac murmur audible to the author; the pulmonic second sound remained accentuated. She was asymptomatic and felt she was better than she had been for years. Because of her evident good condition, she was transported without incident by train to a 1000 mile distant medical convention for presentation in person. No murmur is now present. The pulmonic second sound is still loud. She is ambulant and comfortable. In this moment of triumph, achieved amidst a series of grim disappointments, one must appreciate Dr. Bailey's enthusiasm, even if one cannot justify his conclusions --- for he concluded that "surgical technique and methods in anesthesia have now advanced to the point where intracrdiac manipulation may be undertaken with reasonable saftey". The mortality was still 80% !

## Case Report

R.V., a twenty one year old white female, para II, gravida III, entered the University of Nebraska Hospital for the second time on 1/17/52. Her first admission to this hospital was in August, 1951 at which time she was referred by her local physician because of threatened cardiac decompensation complicating her third

pregnancy. She had been followed in Heart Clinic (UND) since September 1951. Her present admission was for the purpose of observation and evaluation for commissurotomy.

Chief Complaints:

1. Shortness of breath and dyspnea on mild exertion
2. Cough with blood-flecked sputum
3. Orthopnea

All four years duration.

Present Illness:

The patient was well until four years ago. About that time she noticed infrequent episodes of shortness of breath and dyspnea on exertion. These attacks were at first mild, were unaccompanied by pain or other symptoms and were rather promptly relieved by rest. In April 1948 she became pregnant for the first time. Shortly thereafter, her previous symptoms became more marked. After approximately the sixteenth week she began to have slight swelling and edema of her hands and feet and developed a cough which was frequently productive of a copious amount of thick, white,, blood-specked sputum. She delivered a living child spontaneously at seven months gestation. Following this pregnancy, her previous symptoms, while still present, persisted only

to a limited degree but in addition she felt weak,, tired and fatigued and was less able to tolerate ordinary activities. Fourteen months later she became pregnant for the second time. The shortness of breath, dyspnea, intolerance to activity, cough, swelling and edema of hands and feet became much more pronounced. As the pregnancy progressed, orthopnea became a prominent part of the picture.. She delivered a living child spontaneously at forty weeks gestation. There was some tendency toward abatement of symptoms following this delivery but they continued to be more severe than after her first pregnancy.. More particularly,, she noted an increased frequency of shortness of breath and orthopnea, the persistance of cough and characteristic sputum and a gradually increasing feeling of tiredness,, fatigue and exhaustion.

In May, 1951,,the patient became pregnant for the third time. Almost immediately thereafter there was a marked and severe exacerbation of all symptoms. There was some imporvement following digitalization, but because of the continued threat of cardiac decompensation the pregnancy was terminated in August by hysterotomy and a bilateral tubal ligation was done. She decompensated post operatively but responded well to digital-

is and continued to progress without further complications.

History by Systems:

Except for the cardio-pulmonary symptoms, the systematic history was essentially negative.

Past Medical History:

The patient had measles, pertussis, diphtheria and mumps in early childhood. There is no history of other acute febrile illness, scarlet fever, acute rheumatic fever, chorea or other prolonged illness.

Operations included a tonsillectomy at age eight; an appendectomy at age fifteen and a hysterotomy and tubal ligation at age twenty (1951) for reasons noted above.

Family history was essentially non-contributory.

Physical Examination:

Examination revealed an alert, well developed, thin, white female noted particularly because of a semi-recumbent position due to orthopnea and moderate dyspnea. Except for cyanosis of the nails and lips there were no significant abnormalities noted in the eyes,, ears, nose,, throat or extremities. Respirations were eighty per minute and regular except for episodes of dyspnea and coughing. The lungs were clear to auscul-

tation and percussion.. Examination of the heart revealed that the PMI was not visible but was palpated in the fifth interspace in the mid-clavicular line. The rate was 88 and regular. There was an apical diastolic thrill. The left heart border was percussed in the fifth interspace in the mid-clavicular line. There was a loud, rough, rumbling diastolic murmur heard best in the 5 interspace just inside the mid-clavicular line and was transmitted to the axilla. The murmur was heard as a quick crescendo which obscured the second sound, followed in early diastole by a rapid, brief decrescendo and continued through mid-diastole as a low pitched rumble rising in a loud harsh crescendo in the presystolic interval and terminated in a snapping first sound. No other murmurs were present. The pulmonic second sound was increased. The was no evidence of venous engorgement, hepatomegaly or peripheral edema. The remainder of the examination was not significant except for a suspicious cervical lesion,, which on biopsy proved to be a carcinoma in situ.

The clinical impression was mitral stenosis without other significant valvular lesions. Roentgenographic examination revealed left auricular enlarge-

ment,, evidences of pulmonary congestion and an increase in density of the pulmonary artery shadow consistant with pulmonary hypertention. The EKG showed right axis deviation. These findings tend to confirm the clinical diagnosis. The blood and urine studies were within normal limits as were the NPN, sedimentation rate and total serum protein.

It was felt that this patient was an ideal candidate for mitral commissurotomy and accordingly this procedure was carried out on January 31,, 1952. The mitral valve was found to be very small, admitting only the tip of the operator's index finger. No other valvular lesions were noted. The operative procedure was without incident. The post operative condition of the patient was good and was noted by a dramatic decrease in pulmonary symptoms. Whereas prior to suggery she had been dyspnic at rest and had not slept without benefit of at least two pillows for over three years, she now had no dyspnea and could lie flat without discomfort.. Whereas she had decompensated following her hysterotomy, she did, following commissurotomy, progress without evidences of decompensation although this was possibly because digitalis was administered..

Her recovery was uneventful except for the col-

lection of some fluid in the left chest which was aspirated on two occasions.

Following operation there was a marked change in the intensity of the diastolic murmur. This had decreased from a grad IV pre-operatively to a grade I to II immediately post operatively and was not more than a grade I within a week.

The patient's improvement in functional capacity has persisted to the present time.

This case represents a 21 year old patient who was dyspnic at rest,, orthopnic and who had decompensated during pregnancy as the result of a severe mitral stenosis.. Her last pregnancy was interrupted and the tubes ligated because of her cardiac state. Subsequently she was treated by mitral commissurotomy and has shown marked regression of symptoms and a definite increase in functional capacity.

Since this patient was operated,, several cases similar to this one in that cardiac decompensation due to mitral stenosis complicated pregnancy, have been reported.. Baker (15) reports several cases in which commissurotomy has been done as late as the 28th week of gestation. The patients so treated had normal subsequent courses and uncomplicated deliveries. Logan

(14) reports two cases of mitral stenosis in pregnancy in which cardiac decompensation appeared to make therapeutic abortion neeessary. Mitral commissurotomy was done in the fourth month of gestation as an alternative procedure. Both patients continued uneventfully to a normal delivery. One of these patients developed a cerebral embolus five days post partum. This catastrophe might well have been averted by left auricular amputation which is now employed routinely by some groups (23).

These reports substanstiate the view that in cases where disabling pulmonary edema and cardiac decompensation secondary to mitral stenosis complicates pregnancy,, that commissurotomy should be considered as an alternative to therapeutic abortion and that evaluation for sterilization should be done after commissruotomy.

## Pathology of Rheumatic Valvulitis; Pathogenesis and Morbid Anatomy of Mitral Stenosis

The etiology of mitral stenosis is rheumatic valvulitis and this is the most common characteristic cardiac lesion of acute rheumatic fever. This process involves swelling of the collagenous ground sub-

stance of fibrous tissues and is associated with the infiltration of a fibrin deposit throughout the valve. Early in the disease, small verrucae, 1-4 mm. in diameter, consisting of platelets overlaid with fibrin, appear along the line of closure of the valve cusps. These deposits are soon invaded by connective tissue cells followed by organization and replacement by connective tissue. It is Magarey's (24) opinion that repeated fibrin deposits followed by replacement with fibrous connective tissue leads not only to thickening of the valve cusps, but also to gradual narrowing of the valve orifice by a silting-up process. The commissures represent the line of contact between the two valve cusps. As Bailey (25) has pointed out, normally the commissures exist as anatomical entities only dur- the fleeting period of ventricular systole. One does then, wonder by what mechanism they become permanent in the stenotic valve. Magarey (24) has demonstrated adhesions, in all stages of organization, between the adjacent surfaces of the valve cusps. He interprets these to be the result of fibrin clot formation on the endocardium of the valves as a result of valvulitis. The fibrin clots are followed by organisation. This process, often repeated during the onward progression of

the disease,, begins at the periphery of the valve cusps and advances centrally, fusing the valve margins and thereby establishing the commissures as permanent structures which further decrease the size of the valve orifice. Adhesions, fibrous connective tissue replacement,, scarring and fibrous contraction along with varying degrees of calcification produce a stenotic, deformed valve which normally admits three fingers, but which now is a rigid orifice often admitting barely the tip of the index finger.

Essentially the same process of fibrin deposition, adhesions,, replacement by fibrous connective tissue followed by scarring and contraction frequently occurs in the chordae tendonae; they become adherent to one another, shortened and thickened.(26).

An interesting and important factor in the pathology of mitral stenosis and one upon which a portion of the rationale for its surgical management is based,, is Bailey's (6) observation that the plaque of thickened, fibrotic, calcified tissue is primarily at the margin of the valve and that it is surrounded by a zone of relatively normal tissues. In this connection he reports that "It occurred to the author that it might be possible to cut such a plaque completely by

two incisions at the commissures of the valve opening. These incisions should be extended well into the normal valve tissue margin, so that the two halves of the plaque could separate freely, being hinged by the soft valvular tissue at either extremity. It seemed that such an incision could relieve a mitral stenosis without increasing the amount of regurgitation which already existed because of the rigid valve opening."

Physiological Considerations in Mitral Stenosis

An understanding of the clinical manifestations of this disease can be derived only by an appreciation of the altered hemodynamics and pressure changes which occur as a result of the narrowing of the mitral valve orifice. That the primary problem was a mechanical obstruction of the valve orifice and that the symptoms and findings came about as a result of this obstruction, was recognized by such pioneers as Cutler (17), Souttar (22), Brunton (20) as well as others. This concept stood in sharp contrast to that of MacKenzie (27) who, at the time, taught that the difficulty was due to myocardial impairment.

Before turning our attention to the altered circulatory patterns, it is well to understand the pro-

cedures in the study of cardio-pulmonary dynamics.

Methods of Studying Circulatory Dynamics:

1. Cardiac Catheterization:

A detailed discussion of this subject is outside the scope of this paper and may be found elsewhere (28-31). However, a brief review of the procedure and the type of data which may be obtained is pertinent to the understanding of the hemodynamic changes about to be discussed.

A venotomy of the median basilic vein is done under local anesthesia and a specially designed catheter is introduced. It is passed successively through the median basilic, axillary, subclavian and innominate veins, through the superior vena cava, right auricle, right ventricle, pulmonary artery and into the smaller pulmonary radicles while its course is followed with fluoroscopy. As the catheter is then withdrawn, blood samples are collected from the following points: the main branches of the pulmonary artery, the main trunk of the pulmonary artery, various points in the right heart and from the superior vena cava. Arterial blood (femoral artery) samples are also collected and all samples are analysed for oxygen content using the Van

Slyke apparatus.. The arterio-venous oxygen difference can then be determined:

> A-V $O_2$ difference = $O_2$ content in femoral artery blood minus $O_2$ content in pulmonary artery blood..

In addition, pressure tracings are made from the various pulmonary and cardiac structures. Thus it is possible to measure pulmonary "capillary" pressure (32) which in turn has been shown to be equivalent to left auricular pressure (33,34) and to determine the pressure in the pulmonary artery.

With the recognition of the significance of the pulmonary vascular changes (35,, 36, 37) which are reflected physiologically in terms of increased pulmonary vascular resistance, came the need to determine the extent of those changes and to differentiate between the effects produced in the lungs and those attributable to the stenosis of the mitral valve per se.. Gorlin (38, 39) and others (40, 41) have applied hydraulic formulae to this and other data so that the following calculations can be made:

Mitral Valve Area:

$$MVA = \frac{MVF}{31 \quad \text{"PC"} - 5}$$

| | |
|---|---|
| MVA | mitral valve area in $cm.^2$ |
| MVF | mitral valve flow in cc./diastolic second |
| "PC" | pulmonary "capillary" mean pressure in mm. Hg. |
| 5 | assumed left ventricular diastolic pressure |
| 31 | empirical constant |

Pulmonary Arteriolar Resistance:

$$R = \frac{PAm - \text{"PC"}m}{CO} \times 1{,}332$$

Total Pulmonary Resistance:

$$R = \frac{PAm}{CO} \times 1{,}332$$

| | |
|---|---|
| R | resistance in dynes seconds $cm.^{-5}$ |
| PAm | pulmonary arteriolar mean pressure in mm. Hg. |
| "PC" | pulmonary "capillary" mean pressure in mm. Hg. |
| 1,332 | constant for conversion to absolute units |
| CO | cardiac output in cc. per second |

The total pulmonary resistance represents the pulmonary arteriolar resistance plus the resistance of the stenosis of the mitral valve. This relationship affords ready determination of the resistance offered by the stenotic valve itself.

2. Oxygen Consumption Determination:

A sample of the patient's expired air is collected and its oxygen content as well as that of the room air is determined by means of the Haldane apparatus.

$O_2$ consumption = $O_2$ content room air minus $O_2$ content of patient's expired air.

From the oxygen consumption and the arteriovenous oxygen difference the cardiac output can be determined by means of the Fick principle:

Cardiac output in liters per minute

$$\frac{O_2 \text{ consumption in cc. per minute}}{\text{A-V } O_2 \text{ Differenc in cc per liter}}$$

For comparative purposes a standard index, known as the cardiac index, is necessary. This is determined as follows:

Cardiac index =

$$\frac{\text{cardiac output in liters per minute}}{\text{Body surface area in square meters}}$$

## Physiological Changes in Mitral Stenosis

Using the methods just described, Lewis (42),

Dexter (43), Draper (44) and others, (45-49) have investigated the pressure flow relationships across the mitral valve and have demonstrated that as the valve orifice becomes smaller, certain compensatory adjustments occur which alter the pattern of circulatory dynamics. The pathogenesis of these changes is not clearly understood but three definite alterations occur and these can be correlated with the clinical manifestations of the disease.

Lewis and co-workers (42) have shown that with a normal mitral valve area of 4.0 cm. a normal valve flow of 150 cc. per diastolic second can be obtained with a relatively low head of pressure i.e., 6 mm, Hg. in the left auricle. When tissue oxygen demands are greater, as during exercise, this flow can be greatly increased with only a small increase in the pressure head (50). Thus it is apparant that the volume of blood passing through the mitral valve orifice is a function of the size of the orifice and the head of pressure behind the valve and that normally these relations are such that the flow can be increased without developing left auricular or pulmonary hypertension.

In diastole from 40 to 60 cc. of blood must flow through the orifice from the auricle to the ventricle

in about a half second or less. With a narrowed valve orifice, this may not occur with a normal left auricular pressure. The diminution of the mitral valve area will result in a decrease in flow through the valve if the left auricular pressure remains the same. Conversely, if the left auricular pressure is raised, a normal valve flow can be maintained, even though the valve orifice be narrowed (39, 50). The latter is the initial compensatory adjustment which takes place.

The stenotic valve offers marked obstruction to auricular emptying and in the presence of any amount of regurgitation there is reflux of blood during ventricular systole, the total effect of which is to increase left auricular pressure. With only a mild stenosis (MVA greater than 2.5 $cm.^2$), this adjustment may suffice for some time and the patient have little or no difficulty since a cardiac output sufficient to meet ordinary tissue requirements can be maintained without the development of undue pulmonary hypertension (42). As the valve area approaches 1 $cm.^2$, that is one fourth its normal area, greater and greater pressures are necessary in order to maintain a normal valve flow and auricular pressures of from 30 to 40 mm. Hg. may occur (51). Since the pulmonary veins are not equipped with

valves,, this pressure is transmitted via these veins to the capillary bed of the lungs,. When the pressure in the pulmonary capillaries exceeds the osmotic pressure of plasma, approximately 25 mm. Hg.,, there is exudation of fluid into the alveoli and pulmonary edema occurs (51).. This is a critical point in the course of the disease because a tolerable left auricular pressure and a normal cardiac output can no longer exist together.. As will be pointed out later, these patients have severe pulmonary symptoms including dyspnea, orthopnea, and attacks of pulmonary edema..

In summary then, as the valve area becomes smaller a greater head of pressure, represented by pulmonary "capillary" pressure, is required for normal valve flow. With a mild stenosis this can be accomplished without difficulty when only usual oxygen requirements are necessary because up to a valve area of 1.0 $cm.^2$ and increase in flow can be accomplished with a small increase in left auricular pressure. Such an increase in left auricular pressure is not sufficient to raise the pulmonary "capillary" pressure to the level of the 'threshold' for pulmonary edema to occur.. However, a critical valve area (1.0 $cm.^2$) is eventually reached which,, even though there is a marked increase in the pressure head, results

in a very small increase in mitral valve flow. This is because the dynamic effect of stenosis tends to be curvilinear (42) meaning that each increment of stenosis has a greater and greater effect. Such a pressure increase approaches the plasma osmotic pressure and if maintained, results in pulmonary edema (39). This is avoided by a second physiological change, a decrease in cardiac output.

Lewis (42) has demonstrated that the cardiac index decreases in linear fashion as the mitral valve area decreases. There are presumably two main factors at operation in the production of this decrease in cardiac output. The first is the narrowed mitral valve. As noted above, each increment in stenosis has a greater dynamic effect than had the preceeding increment. This means that an equivalent pressure increase will have less and less effect on the mitral valve flow until ultimately the effect is negligible and cardiac output will be reduced. The second factor is a change in the pulmonary arterioles which will be discussed presently. This decrease in cardiac output has the effect of reducing the pressure head required in the left atrium and pulmonary "capillaries" thereby averting the threat of pulmonary edema. Compensation in this direction is limited of

course by the tissue demands of oxygen and the decreased cardiac output is readily reflected by an increased oxygen uptake in the tissue capillaries. This is manifest by an abnormally high arterio-venous oxygen difference (47).

It is well to note that at this point the stenosis is already well advanced (MVA of 1.0 $cm.^2$ or less) (42) and that even at rest these patients may have a reduced cardiac output (47). It is at this point that the third compensatory adjustment occurs. Physiologically this change is an increase in pulmonary arteriolar resistance and is the physiologic counterpart of the anatomical narrowing of the arterioles and small arteries in the lungs (35-37), augmented, apparently, by some degree of vaso constriction (43). The stimulus for its appearance is prsumably a resting pulmonary "capillary" pressure close to the pulmonary edema 'threshold' of 30 mm. Hg. (42).

Physiologically the effect of this change is to reduce the cardiac output (because the right ventricle must work against greater resistance) and thereby shift the pressure flow equilibrium towards a lower left auricular pressure. This favors a lower pulmonary "capillary" pressure. In addition it prevents the right

ventricle from flooding the pulmonary capillaries with the precipitation of attacks of pulmonary edema. It becomes apparent that with increased arteriolar resistance in the lung, that the pulmonary artery pressure will rise (52, 53). This pressure may reach values approaching systemic arterial pressure (54). This compensatory adjustment, then, is both advantageous and disadvantageous in its threefold effect. It allows some degree of protection to the pulmonary capillaries from a strong right ventricle. It decreases the cardiac output in favor of a lower left auricular pressure and a lower pressure in the pulmonary capillaries. It is a constant force against which the right ventricle must work and as will be seen, leads to right ventricular failure.

Mitral stenosis, then, imposes a number of modifications on the pulmonary circulation. The first of these is an increase in left auricular pressure made necessary in order to insure adequate left ventricular filling in the presence of a smaller mitral valve. Further narrowing is attended by a greater left auricular pressure which is transmitted to the pulmonary capillaries. This adjustment is limited on the one hand by systemic oxygen requirements and on the other by the osmotic pressure of

plasma which, when exceeded, results in pulmonary edema. When the valve area approaches a critical value of approximately one fourth normal size, organic narrowing and apparently vasoconstriction of pulmonary arterioles occur which, while preserving the pulmonary capillary bed, offers tremendous resistance against which the right ventricle must work.

The mitral valve area and the pulmonary arteriolar changes are emphasized because they represent two points of increased resistance interposed in the circulatory pathway and the equilibrium maintained between them has a profound effect upon the symptomatology of this disease.

## Pathologic-Physiologic-Clinical Correlation

A discussion of only the clinical aspects of mitral stenosis has not been included as a part of this paper because such a description by itself would leave much to be desired. It is only by correlating the pathologic and physiologic changes with the symptomatology that the variable clinical picture can be appreciated.

Dexter (43), Andrus (55), Ellis (46), Lukas (54) and others (56, 57) have investigated the hemodynamic

changes before and after operation in patients with mitral stenosis and have correlated these findings with the clinical manifestations of the disease. The appreciation of mitral stenosis as a mechanical disease which imposes alterations in the pressure flow equilibrium across the mitral valve along with an understanding of the role of the pulmonary vascular changes does to some extent clearify the variability of the symptomatology seen in these patients.

With a mild stenosis (MVA greater than 1.5 $cm.^2$) and a relatively insignificant rise in pulmonary arteriolar resistance (less than 145 dynes seconds $cm.^{-5}$) there will be very little symptomatology (42). A murmur may be present because there is some obstruction to the flow of blood at the valve (58, 59) but the hemodynamic effect of the stenosis at this point is negligible. This is explained the basis of pressure flow relationships and is attributable to the fact that until the valve approaches a critical area of 1.0 $cm.^2$ adequate ventricular filling may be obtained with only a slight increase in left auricular pressure (42). This increase in pressure is not sufficient to cause more than slight exertional dyspnea.

As the valve area reaches its critical value of

1.0 $cm.^2$ ), the symptomatology becomes more marked and the character of the symptom complex appears to be related to the development of pulmonary vascular changes (35) which, as noted earlier, is reflected physiologically intterms of increased pulmonary arteriolar resistance.

Patients having a stenosis of this degree with only slightly elevated pulmonary arteriolar resistance have severe disabling dyspnea, orthopnea, paroxysmal nocturnal dyspnea, attacks of pulmonary ddema and hemoptysis. The valve area has now reached a point where, in order to maintain an adequate cardiac output,, a high auricular pressure is required and this pressure is transmitted directly to the capillary bed of the lungs. The ultimate pathogenesis of dyspnea is not clearly understood, but it appears to be related to reflex stimulation brought about by congestion in the pulmonary vascular bed. Two factors are at work in the production of this congestion. The first is the high pressure in the pulmonary capillaries required by a high pressure gradient needed for adequate mitral valve flow. The second is related to the pulmonary arteriolar resistance and the strength of the right ventricle.. Since at this stage of the disease the pulmonary arteriolar resistance is still low (150 to 569

dynes seconds cm.$^{-5}$) it is easily exceeded by the force of right ventricular contraction. The output of the right ventricle, then, contributes to the pressure rise in the pulmonary capillaries. It is this pressure rise which apparently causes dyspnea by reflex stimulation. When the rise in pulmonary capillary pressure approaches the value of the osmostic pressure of plasma, pulmonary edema occurs (51).

Two clinical types of hemoptysis occur in patients with mitral stenosis and they stem from different causes. (60). One is characterized by a foamy pink sputum which is related either to local pulmonary congestion or to pulmonary infarction following an embolus. The latter is usually a late manifestation and is associated with auricular fibrillation. The second type is a massive hemoptysis, the source of which is evidently one or more varicosities of bronchial veins which afford collateral channels between the pulmonary and systemic venous systems (61). It is significant to note that patients in this group, i.e., those with severe stenosis and only a slightly elevated pulmonary arteriolar resistance do not have marked symptoms or signs of right ventricular failure (42). An efficient right ventricle can, by a small increase in output, produce a critical rise in

pulmonary venous pressure with the resultant rupture of bronchial varicosities (50). This mechanism may also be important in averting pulmonary edema since it would tend to lower pulmonary "capillary" pressure. In time a point is reached where the left auricular pressure required to maintain an adequate mitral valve flow is incompatible with plasma osmotic pressure in the pulmonary capillaries even when the patient is at rest. This appears to be the stimulus for the development of increased vascular resistance in the lungs (35). The symptoms will now depend upon the magnitude of the pulmonary arteriolar resistance and the competence of the right ventricle. In fact, there may be clinical improvement. If the pulmonary resistance is only moderate, these patients may have exertional dyspnea but are not disabled as in the preceeding group. This is because the pulmonary resistance is sufficient to protect the capillary bed in the lungs, yet not high enough at this point to result in right ventricular failure. As Dexter (43) points out, this is a late development and such clinical improvement means only that the disease has progressed.

When pulmonary arteriolar resistance reaches a high value (700 to 1600 dynes seconds $cm.^{-5}$ ) and the

mitral valve area is 1.0 cm.$^2$ or less, the pressure in the pulmonary artery rises sharply (42) the clinical evidence of which is accentuation of the pulmonic second sound, the Graham-Steell murmur and radiographic evidence of dilated pulmonary arteries (62). Pulmonary symptoms are not conspicuous among patients in this stage of the disease. Instead they show evidences of low cardiac output characterized by fatigue, weakness, exhaustion and an abnormally high arterio-venous difference because of the greater oxygen uptake from tissue capillaries (47). Although the evidence provided by clinical correlation appears to be lacking, this could also account in part at least for the cyanosis. The high resistance in the pulmonary circuit represents a constant obstacle against which the right ventricle must wor and eventually signs of right ventricular failure appear, manifest by cardiomegaly, venous engorgment, hepatomegaly, and peripheral edema.

The additional findings in patients with mitral stenosis are brought about secondarily to the chain of events just described. With a rise of pressure behind the stenotic valve, there is dilation of the left auricle which predisposes to auricular fibrillation and stagnation of blood in the auricular appendage. This

leads to thrombus formation and subsequent cerebral, pulmonary and peripheral emboli..

## Diagnostic Criteria For Mitral Stenosis

The diagnosis of mitral stenosis is based upon the functional status of the patient,, ausculatory evidence,, fluoroscopic and X-ray findings and electrocardiographic observations. Cardiac catheterization gives valuable physiological data, but is not essential for the diagnosis..

The functional capacity of the patient and the correlation with the size of the mitral valve has been discussed in the preceeding section. Since the best surgical results are obtained in those patients whose functional capacity is not markedly limited and whose symptoms are of recent origin, one should not wait until disabling changes occur before making this diagnosis.

A murmur may be the earliest sign of mitral stenosis (63).. It is heard best with the patient recumbent in the left lateral position. The murmur is characteristically a low pitched diastolic rumble, usually well localized at the apex and may be transmitted to the axilla. The murmur may be of varying intensity,, depending upon the degree of stenosis and the presence or absence of mitral

regurgitation. Fluoroscopic and roentgenographic studies reveal enlargement of the left auricle, right ventricle and pulmonary artery. The lung parenchyma may show evidences of pulmonary engorgement. The electrocardiographic changes are those of right ventricular hypertrophy.

## Commissurotomy
## Operative and Post Operative Considerations

While there have been other surgical approaches to this problem such as pulmonary-azygos vein anastomosis (73, 74); the use of mechanical hearts, which may eventually prove to be well worth while, (75); and valve reconstructions (76, 77), it is conceded that, at the present time, a direct attack upon the mitral valve by commissurotomy (or valvulotomy) is the most practical and the most physiologically sound procedure available to us today. It is also conceded that, at the present time, an approach to the mitral valve through the left auricular appendage as originally suggested by Sautter (22) is the most feasible. There are, however, two points of view with regard to the method by which the commissures should be split. Bailey and co-workers prefere to incise the commissures with a guillotine. (6).

Harken et. al. (8) feel that an adequate mitral valve orifice can be established by splitting the commissures with the index finger,, hence the term finger fracture valvulotomy. On the basis of these two methods,, other workers have tended to proceed with the finger fracture method, then incise the commissures only if an adequate orifice cannot otherwise be obtained.

The details of the operative procedure have been described by those who developed the technique,. Bailey (25, 64), Harken (8), Brock (15) and it will be only briefly reviewed here.

The chest is opened through an antero-lateral incision in the fourth left interspace and after proper retraction of the ribs and lungs, the pericardial sac and heart are exposed. Palpation of the heart at this point will give considerable information concerning the nature of valvular lesions (25). A localized diastolic thrill palpable at or near the apex of the left ventricle is pathognomonic of mitral stenosis. A diastolic thrill palpable over the base of the left ventricle close to the atrio-ventricular ring indicates aortic regurgitation. A localized diastolic thrill palpable over the right ventricle indicates severe tricuspid stenosis. A systolic thrill over the root of the aorta indicates aortic

stenosis and a systolic thrill over the left atrium is pathognomonic of mitral insufficiency. After having evaluated the heart by palpation, the pericardium is opened parallel and posterior to the left phrenic nerve and the left auricular appendage is identified. A purse string suture is placed so as to circumscribe the base of the appendage about 0.5 cm. distal to its junction with the atrium and a non crushing clamp is applied proximal to the suture across the base of the appendage. The distal portion of the auricular appendage is then opened by an incision just large enough to admit the surgeon's index finger. The surgeon's index finger is then inserted through the incision into the auricular appendage and as the purse-string is drawn up about his finger,, the clamp is removed. Blood loss attended by this procedure is minimal if present at all.

The surgeon now directs his finger toward the ventricle until it contacts the mitral valve. Internal palpation is now possible. Previously it has been felt that all mitral stenosis was accompanied by some degree of regurgitation. However a regurgitant jet is felt in less than 50% of the patients (25). The index finger is then inserted through the stenotic valve orifice and upward and forward pressure is exerted against the ant-

ero-lateral commissure. This maneuver tends to separate the valve cusps along their line of closure. Similarly,, pressure against the poster-medial extremity opens the posterior commissure.

The guillotine is a thin,, sheathed, blade with a trigger mechanism which is passed between the two glove layers of a doubly gloved index finger (7,, 65). By engaging the blade upon the commissures,, the valve cusps may be separated by incision.

Actually the commissures are separated a little at a time, the surgeon intermittently removing his finger from the valve orifice. This avoids the production of anoxia and its serious consequences as well as allows him to detect the first evidences of regurgitation if this should occur.

Having completed this procedure, the finger is withdrawn from the mitral orifice and with it still in within the atrial lumen, the clamp is reapplied. The purse-string is then drawn up tightly and the surgeon withdraws his finger from the auricular appendage. The left auricular appendage is then ligated near its base and a left auricular appendectomy is done. This procedure has significantly reduced the incidence of post operative emboli (23). The pericardium is closed with interrupted sutures and the chest wall closed by the

usual method.

Keown et. al. (66) and Pender (67) have studidd and reported the methods in anesthesiology for this procedure.

The post operative course of these patients is in general marked by dramatic improvement and an uncomplicated recovery.

The ausculatory changes post operatively have been reported by Spiegl (68) and in his opinion,, the intensity of the diastolic murmur tends to parallel the severity of the stenosis in its evolution and a post operative dicrease tends to parallel the clinical improvement in the patient. The diastolic rumble tends to diminish during the first 7 to 10 days,, gradually becoming inaudible. The Graham-Steell murmur,, when present preoperatively, disappears post operatively. This is consistent with the physiologic finding of a decrease in pulmonary arterial pressure (63,, 49, 57). A grade I to II mitral systolic murmur is reported by nearly all investigators but from clinical,, physiological,, X-ray and electrocardiographic studies,, it is felt that it does not represent a significant degree of insufficiency. The appearance post operatively of an apical systolic murmur of grade III or more suggests

mitral insufficiency produced at operation and is a poor prognostic sign (57).

Arrhythmia is not a common post operative complication and when it occurs, it is usually reversible (57). Pericardial effusion occurs in about half the cases and is treated by pericardial aspiration. Hemiplegia from cerebral emboli is much less common since the introduction of left auricular appendage amputation. There have been no instances of reactivation of rheumatic infection (57) even though two thirds of the auricular appendages show histologic evidence of rheumatic activity. (69, 70).

Mortality: The overall mortality as determined from recent reports of several groups whose series is now significantly large, ranges from ten percent to thirteen percent (15,, 57, 71). It is to be noted that these figures include the first attempts made and as such represent a period when the operative technique was being developed. The patients were those whose stenosis was of long standing and whose prognosis was already grave. Baker, Brock,, Campbell (15) report that of 8 deaths in the first 50 cases,, all occured within the first 22 operative attempts. Of these 8 deaths, all had had auricular fibrillation andthe causes of death were cerebral embolism

and right heart failure. These are late manifestations of this disease. Janton, Glover and O'Neill (71), in reporting 400 consecutive cases admit an over all mortality of 10%. However, over half of the patients in this series were treated at an early or moderately advanced stage and in this group the mortality has been 5%. It is evident that, with the operative procedure better developed and with better selection of patients, a lower mortality can be expected. In addition, these figures tend to point out the significance of early operative intervention.

Functional Capacity: Evaluation of treatment in terms of functional capacity is both difficult and hazzardous. Functional capacity is determined largely by subjective signs and symptoms which are in themselves difficult to evaluate. Secondly, there is no universal standard for classification of either previous status or post operative results. Finally spontaneous changes occur which alter the clinical course of these patients. For example pulmonary vascular changes and right ventricular failure may result in symptomatic improvement by relieving pulmonary congestion, yet in terms of pathology, the disease is progressing.

Janton, Glover and O'Neill (71) using as their

criteria for good or excellent results, the return to normal or nearly normal activity with or without medication, report 78% of their 400 patients to be in this catagory. Baker, Brock and Campbell,, (15) using a similar criteria report good or excellent results in 65 to 70% of their first 50 cases.

## Physiological Effects of Operation

The determination of cardiac output at rest and during exercise,, of arterio-venous oxygen differences and of pressures within the pulmonary circuit before and after commissurotomy and their correlation with clinical results offer a more objective basis upon which to evaluate this procedure.

Pulmonary "Capillary" Pressure: In eleven of twelve patients studied by Dexter (43),, the immediate result was a marked fall in pulmonary "capillary" pressure resulting in relief of symptoms of congestion. In one patient,, the pulmonary "capillary" pressure was little changed but cardiac output rose significantly. In 5 cases observed by Werko (49) a similar fall in pulmonary "capillary" pressure was observed in 4 patients. In one of this series,, pressures in the pulmonary circuit

rose post operatively due to surgically induced mitral incompetance.

Cardiac Output: Gerbade et. al. (56) report a rise of 15 to 30% in resting cardiac output and a rise of 20 to 29% above the resting level following exercise in 10 of 12 patients. Two patients showed no change which was due to persistant pulmonary arterial hypertension resulting in right ventricular depression. It is suggested that in these two patients the surgical procedure had not accomplished and adequate valve orifice. Arterio-venous oxygen differences at rest and during exercise were lowered by 5 to 29%.

In Dexters series (43) and in that of Worko (49) the immediate effect was to lower pressure in the pulmonary circuit rather than to increase cardiac output. In view of the pressure flow equilibrium across the mitral valve (5) and increase in valve area could result in either an increase in cardiac output or a decrease in pulmonary "capillary" pressure depending on the physiological state of the patient at the time (42). In one patient in Dexter's series, the pulmonary "capillary" pressure did not fall, but the cardiac output rose.

With an increase in mitral valve area , less pressure is required for a given mitral valve flow (42) and

and a drop in pulmonary "capillary" pressure is expected. By virture of the same reason, the pulmonary "capillary" pressure may remain constant and with an increase in valve area, cardiac output may rise provided the right ventricle is not depressed by extremes of pulmonary hypertension. Either and increase in cardiac output (if surgically induced mitral regurgitation can be ruled out) or a decrease in pulmonary "capillary" pressure represents a return toward normal physiologic adjustment.

Pulmonary Artery Pressure: Gerbade et. al. (56), Baker et. al. (15), Janton (71) and Werko (49) all report that in a technically adequate commissurotomy, pulmonary artery pressures tend to fall.

Pulmonary Arteriolar Resistance: Dexter (43) and Werko (49) have observed post operative changes in pulmonary arteriolar resistance.

Post operative pulmonary artery pressure and pulmonary arteriolar resistance tend to follow two interesting patterns. The one may be an abrupt, dramatic drop, the other a gradual decrease over a period of six months or more. The very early and abrupt drop suggests that the preoperative elevation may have been due to vaso constriction of the pulmonary arterioles. The prolonged

elevation post operatively would appear to be related the organic narrowing of arterioles. A progressive decline in pulmonary arteriolar resistance raises the question of the reversibility of vascular changes in the lung. A longer period of follow-up and further study must be done before this can be answered.

Right Ventricular Function and Peripheral Venous Pressure: Gerbade (53) and Dexter (43) have shown that right ventricular pressure as determined by diastolic filling pressure tends to approach or reach normal values post operatively. The peripheral venous pressure follows the same course. The increase in right ventricular filling pressure produced by exercise pre-operatively is abolished or diminished post operatively.

Valve Patency: It is natural to question whether or not the mitral orifice will remain patent once commissurotomy has been done. Clinically a recurrence of stenosis might be detected by a return of symptoms, by X-ray and electrocardiographic findings along with long term physiologic studies. In following over 400 case for six months to four years, Janton, Glover and O'Neill (71) report they have found no evidence of recurrence of a stenotic valve. This is significant in view of the fact that about half of the auricular appendages resected

at surgery showed evidence of rheumatic activity although active rheumatic acrditis had not been demonstrable by clinical or laborotorymeans (69,, 70).

Muller (72) reported the post mortem findings in two patients who died three weeks and seven months respectively following commissurotomy. He found no evidence of healing together of the incised borders of the commissures and where there was no calcification, endothelialization had occured. It is also noteworthy that Culter's patient, age 11, lived 4½ years after operation and that autopsy showed no evidence of recurrence of stenosis (21).

These findings suggest that stenosis probably doesn't recurr, but the final answer must await a longer period of evaluation.

## The Factors To Be Considered and Their Significance In Selecting Patients For Commissurotomy

History: There is ample evidence (60, 55, 43, 57) to indicate that the best surgical results are obtained in those patients with mitral stenosis whose symptoms are those of early, progressive cardio-pulmonary dysfunction namely, fatigue, exertional dyspnea and cough. Paroxysmal docturnal dyspnea, dyspnea at rest, pulmonary edema

or right heart failure imply a more advanced stage of the disease (42). With the onset of disabling symptoms, the valve has already become small (1.0 cm. or less). Further progression of the disease may well be that of an increasing symptomatic series of events, the results of gradual myocardial breakdown and mechanical strain, rather than any further contraction of the valve orifice. Age: Bailey (25) reports the youngest patient to have been four and a quarter years, the oldest fifty-nine. Earlier, it was believed that active carditis was more apt to be present in younger persons and that caution should be used up to the age of twenty. While there is no reason to believe that commissurotomy per se in any way prevents recurrence of rheumatic carditis, there have been no cases reported where this has followed surgery. However, the ability to detect a smoldering rheumatic process by clinical means is admittedly inadequate (69) and a higher mortality is present where such is found to be present (57, 71). This would suggest that in those age groups which are more likely to harbor a sub-clinical rheumatic process, caustion might still be used.

At the other extreme of life are those who, as a rule, have other cardiac complications which would mitigate against the performance of successful or profitable

Multiple Valvular Lesions: This question is decidedly in a state of flux. Previously it was felt that either mitral insufficiency or aortic stenosis or both were contraindications if they were of sufficient dynamic significance to have expressed themselves by appreciable left ventricular enlargement or by a wide pulse pressure. Recently Bailey (25) has reported successful treatment of a tight tricuspid stenosis and 28 cases of severe mitral regurgitation. Follow up studies on the treatment of multiple valvular lesions will be awaited with interest. For the present, at least, it seems advisable to select for surgery those patients whose mitral stenosis is the predominant lesion as evidenced by the following: left ventricle normal size; left atrium, right ventricle and pulmonary artery enlarged as evidenced by fluoroscopy and X-ray; right ventricular preponderance or no axis deviation, certainly never left axis deviation as determined by electrocardiographic studies.

Hemoptysis: This represents an urgent indication for surgery becaus it implies the presence of severe pulmonary capillary hypertension.

Auricular Fibrillation and Emboli: These represent complications of mitral stenosis and their appearance suggests a late stage of the disease. Both have con-

tributed heavily to a high overall operative mortality. However, with the development of a technique designed to prevent emboli during and after surgery, it is advisable that these patients be operated (23). Auricular fibrillation has been treated satisfactorily post operatively with quinidine. There is no increased tendency for it to occur in patients in whom it had not been present pre-operatively. Any tendency toward ventricular fibrillation should be brought under control before operation by medical management (56).

A criteria which has been found useful in selecting patients for surgery (57) is given on the following page.

Criteria For Selecting Patients For Commissurotomy

| Grade | Functional Capacity | Candidacy for Surgery |
|---|---|---|
| 0 | No disability | No surgery |
| I | Little or no difficulty on usual activity; lead relatively normal lives | Most would not do surgery in this group |
| II | Progressive exertional dyspnea | Ideal |
| III | Seriously incapacitated; unable to do usual activity | Acceptable |
| IV | Completely incapacitated | Debatable |
| History: | | |
| a. | Early, progressive cardio-pulmonary dysfunction | Ideal |
| b. | Marked dyspnea, hemoptysis, congestive failure | Acceptable |
| X-ray: | | |
| a. | Left atrium and right ventricle, minimally enlarged | Ideal |
| b. | Left ventricle, minimally enlarged | Acceptable |
| EKG: | | |
| a. | Normal electrical axis or right ventricular strain | Acceptable |
| b. | Left ventricular strain | Never acceptable |
| Multiple Valvular Lesions: | | |
| a. | Mitral Stenosis only | Ideal |
| b. | Mitral Stenosis with mitral insufficiency and/or aortic valve lesion in presence of normal left ventricle. | Acceptable |

Contraindications to Commissurotomy: Active rheumatic carditis, subacute bacterial endocarditis, dynamically significant additional valvular lesions, especially if manifest by left ventricular enlargement or hypertrophy, and other serious associated disease such as nephritis or tuberculosis constitute definite contraindications to surgery.

## Summary

In 1948 Dr. Charles Bailey described the technique of commissurotomy for the surgical treatment of mitral stenosis. Since then the procedure has been given an extensive clinical trial by Bailey and co-workers along with others including Baker and Brock (15), Janton et. al. (57, 70), and Harken (8) to mention only a few and a host of favorable reports indicate it has proved its value in the treatment of this disorder. Something like 1100 to 1200 cases have been reported to date. While there have been other surgical approaches to this problem, such as pulmonary-azygos vein anastomosis (73, 74), the use of mechanical hearts which allow direct visualization of the valve (75) and valve reconstructions (76, 77) it is conceded that, at the present time, commissurotomy (or valvulotomy) is the most practical

and the most physiologically sound method available to us today.

Recently, January 31, 1952, this operation was performed for the first time at the University of Nebraska Hospital on a young woman who, five months previously, had undergone a therapeutic hysterotomy and tubal ligation because of cardiac decompensation secondary to mitral stenosis which had complicated her pregnancy. The patient showed a dramatic response post operatively and has maintained good functional status to date.

The pathologic physiology and the physiologic response to commissurotomy as determined from clinical and circulatory studies conducted from six months to four years after operation are herein reviewed. These studies tend to show that as a result of narrowing of the mitral valve certain hemodynamic changes occur resulting first in an increase in left auricular pressure which is transmitted directly to the pulmonary capillaries in the lungs and as this pressure approaches the osmotic pressure of plasma, pulmonary edema occurs (51). Eventually a critical valve area (1.0 cm. ) is reached at which point a further rise in pressure no longer significantly increases the flow of blood across the valve (42). This represents a

late change in the course of the disease and is accompanied by organic narrowing of the pulmonary arterioles (37, 78) and probably some degree of vasoconstriction (29,, 43). The pulmonary arterial pressure rises and the total pulmonary pressure represents a high level of resistance against which the right ventricle must work and which leads to eventual right ventricular failure. Long term post operative studies indicate that these dynamic consequences are favorably altered by surgical separation of the mitral valve cusps along the line of their commissures. Continued improvement in these patients over a period of months to two years (43, 57, 72) suggests that there may be some tendency toward reversal of pulmonary vascular changes,, particularly vaso constriction, but such changes must necessarily await further evaluation.

The procedure itself has been developed beyond the point of grave risk and prohibitive mortality. If one may justifiably exclude those very early cases who were grave risks with or without surgery,, and consider that certain of those deaths might have been obviated with present experience,. the mortality is less than 5%. Indeed the over all mortality including those earlier

cases was only 10-13 %. Bailey (25) reports that in patients whose symptoms are of short duration and who do not have other significant valvular lesions, the mortality is 1%.

From the foregoing statement it is obvious that these are the patients who are best benefitted by surgical intervention. Reports from overall studies suggest that about 70 to 75% of patients surgically treated are able to resume normal or relatively normal lives. There is a significant increase in surgical mortality and decrease in clinical results in those patients whose symptoms are of long duration or whose history reveals such complications as auricular fibrillation, pulmonary or cerebral emboli or long standing cardiac decompensation.

Since the patient herein reported was operated several reports (14, 15) have appeared in the literature stating that commissurotomy has been done as an alternative procedure to therapeutic abortion where pulmonary edema and cardiac decompensation due to mitral stenosis have complicated pregnancy. The patients so treated continued their pregnancies uneventfully and had normal uncomplicated deliveries. They advise the procedure in thissituation and suggest that where mitral stenosis tends to contradict further pregnancy, evaluation for

sterilization be done after commissurotomy (15).

## Conclusions

1. Commissurotomy is a relatively safe procedure and has been shown to favorably alter the hemodynamic changes in the pulmonary circuit which result from mitral stenosis.
2. Surgical intervention should be considered in those patients with mitral stenosis who show signs of early progressive cardio-pulmonary dysfunction. To delay treatment is to invite the development of changes within the pulmonary vascular system and myocardium which may well be irreversible.
3. This procedure may be considered an alternative to therapeutic abortion or sterilization in women with mitral stenosis whose cardiac status contradicts pregnancy.

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