Bulletin of the

University of Minnesota Hospitals
and
Minnesota Medical Foundation

Valvotomy in Mitral Stenosis

Volume XXIV | Friday, January 30, 1953 | Number 15

BULLETIN OF THE
UNIVERSITY OF MINNESOTA HOSPITALS
and
MINNESOTA MEDICAL FOUNDATION

Volume XXIV | Friday, January 30, 1953 | Number 15

INDEX

Published weekly during the school year, October to June, inclusive

The Bulletin is sent to members of the Minnesota Medical Foundation. Annual membership fee - $10.00

Address communications to: Staff Bulletin, 3330 Powell Hall, University of Minnesota, Minneapolis 14, Minn.

CONTENTS

I. CLINICAL AND PHYSIOLOGIC STUDY OF THE RESULTS OF VALVOTOMY IN PATIENTS WITH MITRAL STENOSIS*

A. M. Richards, M.D.
Ivan D. Baronofsky, M.D.
Craig Borden, M.D.

(I) Introduction

A. General Considerations

Critical evaluation of a new therapeutic procedure is difficult at best. Improvement as judged by symptoms alone is notoriously deceptive, particularly in the realm of cardiovascular disease. The prognosis is often ominous, functional overlay frequent, and methods for quantitation of crucial symptoms such as dyspnea inadequate. Quantitative assay of objective variables, whenever possible, is mandatory.

The application of physiologic methods to diagnosis and the development of the technics of thoracic surgery in the past decade have resulted in great advances in our understanding and treatment of many cardiovascular lesions. In two congenital lesions, viz., patent ductus arteriosus and coarctation of the aorta, the surgical procedure is definitive, the operative mortality low, and the results unequivocal. Inasmuch as a normal circulatory system can be established in these conditions, the diagnosis of either one practically demands immediate correction. On the other hand, definitive therapeutic attack upon more frequent forms of heart disease, viz., acquired cardiac lesions, has progressed of necessity more slowly. Nevertheless, reports from several centers[3,5,27,31] have already established that surgical attack upon the mitral valve stenosed as the result of rheumatic endocarditis is technically sound and reasonably safe. The immediate results are beneficial in carefully selected patients and in certain instances, improvement is dramatic. In contrast to the situation obtaining in patent ductus arteriosus, however, the procedure of mitral valvotomy is palliative and not definitive. Concommitant pathologic changes in the valve leaflet, chordae tendinae, pulmonary circuit and right ventricle are not directly affected although there is reason to believe that some of the lesions in the lung may be partially reversible. The accumulation of hemodynamic data to clarify results in light of the known natural history of the untreated disease is warranted. At the outset, it is obvious that the ultimate effectiveness of valvotomy in the treatment of mitral stenosis can only be ascertained from long term follow-up study.

B. Purpose

It is the purpose of this report to summarize the experience with mitral valvotomy in 52 patients. Forty-six have been followed from 2 months to 2½ years postoperatively. There were 6 deaths. Preoperative cardiac catheterization studies are available on 47 of the 52 patients. Both pre- and postoperative data are complete for 31 patients.* In this group, clinical and physiologic correlations have been carried out in an attempt to assay the results as objectively as possible and evolve sound clinical criteria in selecting candidates for surgical therapy.

C. History of the Development of the Surgical Technic.

The actual surgical technique has been well described in the literature. Actually, historically, it has developed over a long period of time. Lauder Brunton[10] in 1902 was probably the first to suggest that surgery for mitral stenosis is a feasible procedure. Twenty years later Allen and Graham[1] developed a cardiovalvuloscope with a cutting

* This study was partially supported by a grant from the Minnesota Heart Association and the Louis and Maude Hill Family Foundation.

* Data for 4 patients kindly submitted by Drs. Russell Wilson and Ralph Smith from Veterans and Ancker Hospital.

attachment, which was inserted via the auricle rather than the ventricle. About the same time Cutler, Levine, and Beck[16] developed a valvulotome with which pieces of the valve were punched out. Their failure to realize that regurgitation is as deleterious as stenosis, or more so, doomed their procedure to failure. Souttar[35] in 1925 then suggested the insertion of a finger into the atrium with a knife attachment; in fact, he tried this on one patient. With this basis as a background, some 20 years later the problem of the technical approach to mitral stenosis was resurrected. Details of preoperative, operative, and postoperative management were added and with these additions we have the procedure as it stands today. Harken[28] and Bailey[2,4] are to be credited with the idea of relieving stenosis without producing regurgitation.

(II) Pathologic Physiology of Mitral Stenosis.

The mitral valve orifice at the venous end of the pulmonary circuit is normally 4 to 6 sq. cm. in cross-sectional area. It offers little resistance to flow so that the pressure gradient between the left atrium and left ventricle is nil. In contrast to the adult systemic circuit, the pulmonary circuit is a low pressure system. The normal mean pulmonary artery pressure is from 10 to 15 mm. Hg; the normal pulmonary "capillary" or wedge pressure is from 6 to 12 mm. Hg; and the pulmonary venous or left atrial pressure is 4 to 6 mm. Hg. The normal gradient, therefore, or fall in pressure from the pulmonary valve to the mitral valve is quite low, being from 5 to 10 mm. Hg. The normal pulmonary circuit can accommodate large increases in blood flow with little or no alteration in pressure relationships, but when a $2\frac{1}{2}$-fold increase in flow is exceeded, pressure rises have been observed.[14]

When the mitral orifice becomes narrowed or stenosed by disease a resistance is imposed at the critical venous end of the pulmonary circuit. Standard hydraulic formulae for flow through a fixed orifice indicate in simple terms that flow is proportional to the square root of the pressure.[22] Thus as stenosis occurs either flow (cardiac output) must diminish, or the pressure in the left atrium and pulmonary veins must rise proportionally to the square of the resistance; both low cardiac output and high pulmonary pressures are regularly observed in advanced mitral disease.

Application of theoretically constructed pressure-flow curves by Gorlin[22] have indicated that over 2-fold increases in cardiac output (up to 15 liters) can be tolerated with a mitral orifice area of 2.5 sq. cm. before a pulmonary venous pressure of 35 mm. Hg is exceeded. Dexter[17] considers a wedge pressure of 35 mm. Hg as the level at which clinical pulmonary edema occurs, hence the critical valve area of 2.5 sq. cm. Estimation of the valve area by catheterization technics in patients with mitral stenosis have been roughly correlated with disability.[17] Thus major symptoms are usually lacking with valve areas greater than 1.5 sq. cm. and disability invariably severe with areas of 1.0 sq. cm. or less. As suggested by Gorlin,[22] a 2.5 sq. cm. area for the mitral orifice should be the objective of surgery.

The cardinal symptoms of mitral stenosis, i.e., dyspnea, orthopnea, cough, pulmonary edema, hemoptysis, are all pulmonary in origin and have been shown to be related to pressure changes[8] and consequent anatomic alterations in the vascular bed of the lung. Recent work with experimental mitral stenosis, corresponding to the early lesion in man, has shown that pulmonary artery pressure increases but not in proportion to the rise in pulmonary venous pressure.[26] Hence, initially there is a diminution of the normal gradient across the pulmonary bed. (Some gradient of course must be maintained if flow is to continue and in any event, energy for a rise of pressure in any part of the pulmonary circuit must come from the work of the right ventricle.) Under these circumstances, sudden increases in flow in response to exercise can readily precipitate acute pulmonary edema from sudden increases in pulmonary venous and capillary pressure. In the

advanced states of the disease in man, however, severe pulmonary hypertension is usually found which is out of all proportion to estimated increases in pulmonary venous pressure. Calculated resistance at the level of the pulmonary "arterioles" is markedly increased. This increased "arteriolar" resistance may possibly protect the pulmonary capillary bed from sudden increases in cardiac output; indeed, exercise studies have shown that the output is usually "fixed" in advanced mitral stenosis.[23,30] Frank pulmonary edema is seldom observed in the later stages of the disease. Prolonged pulmonary "arteriolar" hypertension ultimately leads to dilatation of the pulmonary artery and enlargement of the right ventricle. Symptoms of right ventricular (congestive) failure then ensue consisting of venous distention, hepatomegaly, peripheral edema, and at times hydrothorax or ascites.

The foregoing conceptions are summarized in table I. Considerable overlap of data is encountered in any attempt at grouping, including calculated valve areas. In lieu of valve area calculation because of technical failure to obtain satisfactory wedge pressures in every case, pulmonary artery pressure can also be correlated with symptomatology and disability in mitral stenosis.

TABLE I.*

CLASS	FUNCTIONAL CAPACITY	MAJOR SYMPTOMS	MITRAL VALVE AREA sq. cm.	MEAN PULMONARY ARTERY PRESSURE mm. Hg
I	Virtually unlimited	None	4 - 2.5	15 - 20
II	Some limitations	Dyspnea Pulmonary edema	1.3 - 1.6	20 - 35
III	Very limited	Hemoptysis	0.6 - 1.1	35 - 60
IV	Bed and chair	Recurrent congestive failure	0.4 - 0.9	45 - 90

*Adapted from Dexter (17). (Functional classifications that of the New York Heart Association.)

(III) Selection of Patients

A. Age and Sex:

Fifty-two patients were subjected to valvotomy from June of 1950 through December of 1952. The procedure was performed by the same surgeon with two exceptions. Five additional patients were explored only; four had prohibitive mitral regurgitation and one extensive atrial thrombosis.

The average age for the group was 37 years. The youngest was a ten-year old girl and the oldest was 54. (A 60-year

old man has been operated upon recently; recovery was uneventful.*) There were 13 men and 39 women, a ratio of 3:1; the average age for men and women was the same. A definite history of rheumatic fever was obtained in 27 patients (52 per cent) occurring from 7 to 43 years previously (average, 23 years).

B. Symptomatology:

In general, patients having relatively "pure" mitral stenosis with pulmonary arterial pressure of 35 mm. Hg or more. Two patients having mean pressures of 21 mm. Hg gave a history of hemoptysis; in both it was minimal in amount and in neither was it observed by a physician. Auricular fibrillation and systemic emboli were not related to pulmonary hypertension, a wide range of pressures being observed in patients with these symptoms. In addition to the symptoms listed, one patient in the series had proven subacute bacterial endocarditis occurring three months prior to surgery.

disabling symptoms and pulmonary hypertension at rest were selected for surgery. All had exertional dyspnea and the mean pulmonary arterial pressure for the group was 47 mm. Hg. Clinical features are tabulated with average mean pulmonary pressure in Table II. Although there is considerable overlap, disabling symptoms are usually associated with marked pulmonary hypertension. For example, frank hemoptysis, a cardinal symptom, occurred with two exceptions, in those patients having a mean

TABLE II

No. Pts.	Clinical Feature	Average Mean PAP
52	Exertional Dyspnea	47
34	Orthopnea	50
26	Congestive Failure	58
23	Pulmonary Edema	52
24	Hemoptysis	50
20	Auricular Fibrillation	45
11	Systemic Emboli	45
7	Calcified Valve (X-ray)	53
3	Chronic Hydrothorax	60

C. Murmurs:

1. Diastolic

a. Apical

All patients had the typical apical rumble of mitral stenosis. In nine instances, there was an accompanying diastolic thrill. In some patients with advanced disease, the mitral stenotic murmur varied considerably, being either difficult to hear or inaudible at times.

b. Left sternal border

The second pulmonic

* Veterans Hospital, Dr. N. K. Jensen

second sound was greater than the aortic in 43 patients and markedly accentuated or split in those with severe pulmonary hypertension. Twenty-one patients in the group had early decrescendo diastolic murmurs along the left sternal border; 11 murmurs were thought to be due to functional pulmonic insufficiency (Graham-Steell)[37] and 10 were attributed to aortic insufficiency. None had an increase in systemic pulse pressure. The range for the systemic pulse pressure was between 38 and 60 mm. Hg, the mean being 45 mm. Hg for each group. The average mean pulmonary artery pressure, on the other hand, was 55 mm. Hg in the group thought to have pulmonary insufficiency as compared to 38 mm. Hg for those diagnosed as having aortic insufficiency. In other words, half of the diastolic murmurs heard along the left sternal border could have resulted from dilatation of the pulmonic valve ring as a consequence of severe pulmonary hypertension.

2. Systolic

a. Apical

An apical systolic murmur of grade II intensity was present in 13 patients. Five patients had apical systolic murmurs of grade III intensity. All of the latter were class IV patients and both groups had "tight" mitral stenosis at surgery. In contrast, grade III apical systolic murmurs were present in 3 of 4 patients who had prohibitive mitral regurgitation at thoracotomy. All were in functional class II-III. Hence, loud apical systolic murmurs point to mitral regurgitation in most ambulatory patients but may be present in class IV patients with "tight" mitral stenosis. Relative tricuspid insufficiency may be the cause of some of the loud apical systolic murmurs in class IV patients with "tight" mitral stenosis.

b. Other locations

Many patients had loud systolic murmurs over the pulmonary area presumably due to dilatation of the pulmonary artery with resultant turbulent flow. Rarely, a loud systolic murmur is heard along the left sternal border in the fourth and fifth interspace. It may not be significant unless associated with an aortic systolic murmur. The electrical axis on the electrocardiogram and the degree of left ventricular enlargement radiologically must be taken into consideration in evaluating aortic systolic murmurs to diagnose dynamically significant aortic stenosis.

D. Roentgenograms:

Findings in the chest roentgenogram are also roughly correlated with disability and the degree of pulmonary hypertension. These consist of a reticular appearance in the lung fields, prominence of the left pulmonary artery segment, and enlargement of the right ventricle. These findings are usually absent in asymptomatic mitral stenosis. All but 2 patients had evidence of some left atrial enlargement.

Correlation of findings with pulmonary hypertension is evident in the accompanying table (Table 3).* The exact cause of the reticular appearance in the lung fields is unknown. Right ventricular size could not be evaluated in five instances because of pleural fluid or chest deformity.

E. Electrocardiogram:

The electrocardiogram aided in the selection of patients by pointing up right ventricular preponderance. The mean electrical axis for the entire group was +95° with a range from +60° to +155°. A right ventricular strain pattern was present in 27; all these had severe pulmonary hypertension, the average mean pulmonary artery pressure being 52 mm. Hg. Although an electrical axis of +95° and a mean pulmonary pressure of 31 mm. Hg was present in one of the four patients with a prohibitive degree of mitral regurgitation, the other three had axes less than +85° and mean pulmonary pressures under 25 mm. Hg.

---

* Roentgenograms were reviewed by Dr. Burt Levin of the Radiology Department.

TABLE III

	No. Pts.	X-ray Finding	Average Mean PAP
Reticulation	18	None	39
	26	Moderate	48
	8	Advanced	69
Pulmonary Artery Segment	13	Normal	32
	36	Straight	40
	3	Prominent	53
Size Right Ventricle	9	Normal	43
	38	Enlarged	52
	5	---	--

(IV) Surgical Management

A. Preoperative preparation

The lessons learned in the past decade about the medical management of cardiac patients, particularly those in congestive failure, have been adapted to the surgical management of patients with mitral stenosis. All patients are admitted to the hospital at least one week prior to surgery for regimentation. A dehydration regimen, when indicated, and digitalis are the principal features of treatment. The cases may be divided into two groups: those with manifest or incipient edema, and those without. A 200 mg. sodium diet and, if needed, diuretics were given to the former group until the patients' weight chart indicated no further loss on continued therapy. Twenty-one of the group had received diuretics in the past. The second group, those without overt edema, were not given diuretics; as a matter of fact, diuretics are contraindicated in this group as it may lead to an electrolyte disturbance. This occurred in one case.

Digitalis was used preoperatively in all cases but 2. Those patients who had auricular fibrillation were digitalized until their apical pulse rate was between 60 to 70 beats per minute. Those patients who had a normal sinus rhythm were given digitalis on the assumption that if fibrillation occurred following surgery it would occur at a slower rate and could be rapidly controlled.[5] When hydrothorax was present, it was tapped until dry as demonstrated by x-ray.

In general, patients with sedimentation rates much over 20 mm. in one hour, were not operated upon in order to avoid surgery in the presence of rheumatic activity.

If dicumarol therapy had been instituted for the prevention of arterial embolization, this was discontinued and the prothrombin time brought to normal.

Antibiotic therapy consisting mainly of aqueous penicillin, 200,000 units, twice daily, was instituted one or two days before surgery. When allergy to one antibiotic was present, another was used.

Every effort was made to keep the patient well sedated the night before and the morning of surgery. Nasal tubes or indwelling urinary catheters were not inserted preoperatively. Intravenous fluids were not used on the morning of

surgery because of fear of inducing pulmonary edema. Atropine sulfate grain 1/300 to grain 1/150 and morphine sulfate grains 1/8 to grains 1/6 subcutaneously were administered one hour prior to the administration of the anesthetic agents.

In patients with severe mitral stenosis, an increase in pulse rate, consequent to anxiety may precipitate pulmonary edema. In hyperexcitable patients, pentothal administered in the patient's room as a "sneak" procedure is often desirable to prevent anxiety. There must be no delay between the time the patient leaves the floor and the induction of anesthesia, as anxiety and its consequences will result.

Lowered vital capacity is not a contraindication to cardiac surgery. This is in contrast to patients who are to undergo pulmonary surgery. The following case illustrates the above point: A 41-year old white female with severe mitral stenosis was presented for the possibility of surgery. She had had a massive pulmonary infarct of the right lower lobe in the past. Her vital capacity was 1.2 liters and bronchospirometric studies showed the right lung oxygen uptake to be 47% and the uptake of the left lung, which is partially collapsed during surgery, to be 53%. This is an exact reversal of normal. Surgery was undertaken, and in spite of not alleviating her stenosis, the patient survived with no complications. A tracheotomy performed two days before the cardiac surgery proved very useful. This was done to allow for easy suctioning of the trachea with a catheter and the prevention of retained secretions. Tracheotomy is of definite aid in those patients who may have trouble postoperatively with retained secretions.

B. Surgical Technique

The anesthetic agent used in all cases has been pentothal-curare solution supplemented with nitrous-oxygen mixture. Whether or not this is the ideal method in the very poor risk patient, remains to be proved. At any rate, every effort should be made to keep the patient as light as possible, thereby avoiding any depression of respiration. In essence, a finger, with or without a knife, is inserted into the atrium via the appendage, the valve is slit in its commissures, to relieve stenosis and prevent regurgitation, and the appendage sewn over. The knife was used in 14 cases in the series, finger-fracture being used in the remainder.

In the event that the appendage is obliterated or small, the atrium may be entered via the left superior pulmonary vein. This approach has been utilized on 2 occasions.

Blood transfusion during surgery is limited to blood-loss replacement. If the loss has been minimal, blood is not given.

C. Postoperative Care and Status

Fluids

Most patients can and should be hydrated orally. The following table summarizes the average intake of the patients in this series for the first 4 postoperative days when recorded.

FLUID INTAKE (cc.)

	Day 1	Day 2	Day 3	Day 4
AVERAGE	1781	1907	1872	1679
RANGE	200-3220	340-2880	580-3950	740-2645
ROUTE ADMINISTRATION OF FLUIDS	11 I.V.O. 5 P.I.V. 33 oral	5 I.V.O. 9 P.I.V. 33 oral	4 I.V.O. 4 P.I.V. 38 oral	3 I.V.O. 2 P.I.V. 35 oral

I.V.O. = intravenously only P.I.V. = part intravenously

Oral = oral route only

When intravenous fluids are given, 2,000 cc. per day should be the limit, unless specific reasons for more exist. At present no patient has been lost because of dehydration. Salt solution is forbidden. As we have gained experience with the operation, more of the patients have been hydrated entirely orally.

Output

In contrast to gastrointestinal surgery, we have not placed great emphasis on urinary output. The following table summarizes the average urinary output for the first 4 postoperative days.

URINE OUTPUT

	Day 1	Day 2	Day 3	Day 4
OUTPUT	684	938	954	1022
RANGE	200-2800	275-4200	200-2475	200-2100

Those patients who have small uninary outputs for the first 2 days present no difficulty. Invariably their output will be normal on the third and following days. This is most likely related to a renal blood flow readjustment following the opening of the valve.

Nasal Suction

Nine patients required nasal suction at some time during the postoperative convalescence. The indications for its use were repeated emesis or acute gastric dilatation. The nasal tube was removed as soon as possible because it interfered with respiration and because it might produce depletion of electrolytes.

Temperature

Temperature elevation over 100° F. rectally was obtained in 48 cases on the first day, 46, 37, 24, and 17 cases on the second, third, fourth, and fifth day respectively.

Coughing

In order to prevent atelectasis and its attendant consequences, great attention is paid to the simple reflex of coughing. As previously mentioned, tracheotomy has been performed on those patients who were believed to be unlikely to respond well to demands to cough. We recognize that thoracic surgical patients have difficulty in coughing because of pain. However, for the most part, patients will comply with the demand of the physician or nurse to cough. Instruction to the patient at the bedside on the act of coughing is very helpful. Whenever the patient is seen by nurse or physician, coughing is insisted upon.

In addition to the above points, the customary care is given as to any patient with a thoracotomy. Antibiotics are given routinely. Ambulation is started as soon as the chest catheters are removed (usually on the third or fourth day) and food intake encouraged slowly. Low sodium diets are given to those individuals who were on these diets preoperatively.

(V) Clinical Results

A. Symptomatic:

To simplify presentation of the overall clinical results, patients were grouped according to the classification of the New York Heart Association.[15] This classification is based on symptoms and considerable difficulty is encountered in evaluating symptoms and disability in patients with chronic rheumatic heart disease. Not only is the natural course of the disease variable,[24,33] but some patients have few symptoms because they voluntarily restrict their activity, while others greatly exaggerate their symptoms.

Nevertheless, in the preoperative evaluation, it was possible to separate

15 patients limited to a bed-chair existence by severe dyspnea and recurrent congestive failure (class IV), Table IV. Thirty-six patients were placed in a combined group (classes II-III). One patient fell into class I because of a

TABLE IV

CLINICAL RESULTS OF VALVOTOMY				
Preoperative		Deaths	Postoperative	
Class	No. of patients	No. of patients	Class	No. of patients
IV	15	4	IV	1
II-III	36	2	III	2
I	1	0	I-II	43

lack of symptoms referable to the pulmonary circuit; the indication for surgery in this patient was a recent embolus to the brain manifested by left hemiplegia.

In the postoperative evaluation, it was possible to separate the obvious therapeutic failures (Table IV); all 3 had previously been in class II-III. The one now falling into class IV had associated aortic stenosis and insufficiency. Although initially improved, this patient subsequently developed left ventricular enlargement and recurrent congestive failure. One of the 2 in class III was a technical failure; a cut in the valve leaflet during surgery resulted in dynamic mitral insufficiency. The other failure is unexplained. All 3 patients require salt restriction and periodic mercurial diuretics.

Forty-three of the 46 survivors (85 per cent of the original group) were without doubt improved, having minor or relatively few symptoms to date. All claim subjective improvement as does one of those considered to be a therapeutic failure.

Objective evidence for improvement is apparent in the 11 former class IV patients of this group, all of whom have become ambulatory and capable of activity involved in ordinary living. They are free of edema without salt restriction or mercurials. Hepatomegaly and venous distention disappeared within the first 3 weeks after surgery. One with chronic hydrothorax has required thoracentesis once in the 4 months which have elapsed since valvotomy. Previously thoracentesis was needed once every 3 to 4 weeks. Two episodes of transient congestive failure occurred in one girl in the first year postoperatively. She has now been followed $2\frac{1}{2}$ years and swam without symptoms this past summer. An illness consistent with rheumatic fever occurred this fall but she is again asymptomatic. Two of the 11 patients are in the immediate postoperative period. Eight of the remaining 9 are women and all are doing their own housework. One boy, aged 19, is working full time as a grocery stock clerk.

In contrast, objective evidence for clinical improvement for the remaining 32 patients formerly in class II-III is less certain and usually lacking in those whose only symptom was exertional dyspnea. Nevertheless, none have had a recurrence of orthopnea, frank pulmonary edema, or systemic emboli. Only one has had recurrent hemoptysis and this occurred in a man following the stress of playing 40

minutes of sandlot football. Four had a transient episode of mild congestive failure in the first 2 months postoperatively. None are now on salt restriction or mecurials, although 10 had required one or the other preoperatively.

Of 23 women, formerly in class II-III, 20 were married. They have returned to their housework, which in several instances includes the care of small children. One has gone through two pregnancies and another one pregnancy. Acute pulmonary edema as the result of circulatory overload from blood transfusion occurred in one patient following delivery. The other 2 pregnancies and deliveries were without event. One patient, 7 weeks pregnant, underwent valvotomy and is now doing well in the 12th week of pregnancy. The remaining 3 women in the group were unmarried. One is teaching, another clerking in a department store, and the other working as a telephone operator.

All of the 9 men have returned to some gainful work. Full time occupations include those of a city bus driver, electrician, and garage maintenance supervisor.

B. Murmurs

Most of the murmurs noted preoperatively have been little changed by valvotomy. Apical diastolic murmurs are less intense in about half the cases. An apical diastolic thrill presists in 3 patients. Most patients have grade II plus, high-pitched, short systolic murmurs at the apex after valvotomy. Only one has a grade III, apical, systolic murmur and this is the patient in whom mitral insufficiency was inadvertently produced. Two of the Graham-Steell murmurs have disappeared. The apical diastolic murmur disappeared completely in 3 patients. In one, the postoperative valve area was calculated, and a value of 2.6 sqm. was obtained. In the other, the diastolic murmur recurred during the fourth postoperative month. Change in murmurs is a poor index to the clinical-physiologic improvement except in those rare instances where murmurs disappear.

C. Roentgenograms

Changes in the x-ray of the chest were of little aid in evaluating improvement, no change being detected in 36 instances. There was decrease in size of the pulmonary artery segment in 10 patients, decrease in size of the left atrium in 7, and decrease in size of the right ventricle in 4. All those with decrease in size of the right ventricle had postoperative pulmonary pressures under 27 mm. Hg. There was some clearing of the lung fields in one instance but no correlation between reticular appearance and lung biopsy could be found.

D. Mortality

There were 6 deaths, giving an overall mortality rate of 12%. Four deaths were in class IV patients and 2 were in class II-III patients, a mortality for each group of 27 and 5%, respectively. One death was the result of ventricular fibrillation occurring during induction of anesthesia in a class IV patient. Another was associated with unexplained hypotension in a class IV patient and was presumably the result of low cardiac output. One was technical. Fracture of the commissure resulted in extensive laceration of the atrial-ventricular wall. The remainder were caused by cerebral embolization, death occurring on the second or third day. In only one was a dislodged thrombus clearly the cause of the cerebral embolus. The other 2 patients presented evidence of diffuse cortical damage suggesting the possibility of multiple air emboli.

A high mortality in class IV cardiac patients has been reported in other series.[27,31] Without active intervention in this group, the outcome is certain in a matter of months. Because of the obvious dramatic improvement in class IV patients successfully treated, these poor risk patients have been accepted for surgery despite a high mortality.

E. Complications

There were relatively few com-

plications. The occurrence of transient mild congestive failure in 5 patients early in the postoperative period was noted (Section IV, A.). One patient had postoperative atelectasis and pneumonitis. Two developed atelectasis secondary to phrenic injury. One developed a hemothorax.

A pericardial friction rub can be heard between the second and fourth postoperative day in most patients. In only one was there radiologic evidence of effusion.

1. Rheumatic Activity

The patient with probable pericardial effusion ran a febrile course for 25 days and had a markedly elevated sedimentation rate. Convalescence was protracted but recovery with improvement was evident in 6 months. The appearance of an aortic diastolic murmur 3 months following the febrile illness supported the diagnosis of rheumatic activity. In another patient, fever, pleuritic pain, cardiac enlargement, and dyspnea coming on one month following valvotomy also suggested rheumatic activity. There was elevation of the sedimentation rate, a C-reactive protein reaction of 4+, and an anti-streptolysin titer of 317 U/cc., all of which subsequently returned to normal. A similar febrile illness in a patient, occurring 2 years postoperatively, was initiated by rapid auricular fibrillation. Sedimentation rate and anti-streptolysin O titer were elevated, returning to normal in one month. Auricular biopsies from each patient were examined for Aschoff nodules. These were present in only one instance, a much lower percentage than reported by Janton et al.[31]

2. Arrhythmias

Eleven patients that had had normal sinus rhythm before surgery developed auricular fibrillation afterwards. Fibrillation occurred most frequently from 1 to 5 days postoperatively. The rate of fibrillation varied from 90 to 176. In those patients whose apical rates were above 100, from 0.2 mg. to 3.2 mg. digitoxin intravenously given within 36 hours, was necessary to bring the rate down to 80.

Because of the ever present threat of pulmonary edema resulting from rapid rate, every effort must be made at early diagnosis of changes in rate and rhythm of the apical beat. Nurses must be taught to take apical pulses and the responsible physicians must examine the patient frequently. Whenever a rapid rate or arrhythmia is detected, an electrocardiogram is mandatory for accurate diagnosis. Therapy should be instituted and maintained under electrocardiographic control. Arrhythmias, other than auricular fibrillation, have not been observed, although these have been reported by Dexter.[36]

Conversion to normal sinus rhythm was accomplished in 9 of the above 11 patients. Four patients converted in 1 to 4 days following the onset of the fibrillation on digitalis alone. Four patients converted in one day with the use of quinidine grains III to grains VI, 3 times a day. One patient, who fibrillated at a slow rate (96), and whose dose of digitalis was not increased, converted spontaneously. Two patients failed to convert on quinidine. Conversion to normal sinus rhythm can, therefore, be accomplished in the great majority of instances in which auricular fibrillation occurs following surgery. None of the patients were converted to normal sinus rhythm who had established auricular fibrillation prior to surgery.

(VI) Clinical and Physiologic Correlations

A. Methods

Preoperative studies for 31 patients were repeated postoperatively in approximately 6 weeks and in 5 instances 1 to 2 years following surgery. Right heart catheterization was performed using techniques previously described.[6,13] Pulmonary "capillary" or wedge pressures were measured in 14 patients by pushing the catheter tip into a small branch of the pulmonary artery as tightly as possible.[29] Integrated mean pressures were used in the comparative analyses. Using

a modification of Stewart's method,[6,7,38] the volume of blood contained in the lungs, the left heart and great vessels, the so-called central blood volume was determined in 13 patients. The total blood volume was also determined. The total lung volume was measured in 18 patients. The functional residual volume was determined by the open circuit method.[12] The vital capacity and its subdivisions were recorded with a spirometer.

B. Pulmonary Artery Pressures

The average mean pulmonary artery pressure for the 31 patients decreased from 45 mm. Hg to 32 mm. Hg postoperatively, a significant fall of 13 mm. Hg (P=.01). The results are presented in Table V for the two groups of patients, 8 in class IV, and 23 in class II-III.

TABLE V

Physiologic Measurements in 31 Patients Subjected to Valvotomy

Number of Patients	Functional Class		Average Mean P.A.P.	Average Right Ventricular Diastolic Pressure mm. Hg	Resting Cardiac Output L/Min.
8	IV	Before After	65 47	9 5	3.07 4.21
23	II-III	Before After	38 26	5 4	4.52 4.15
Predicted Normal			11-15	0-4	5.85

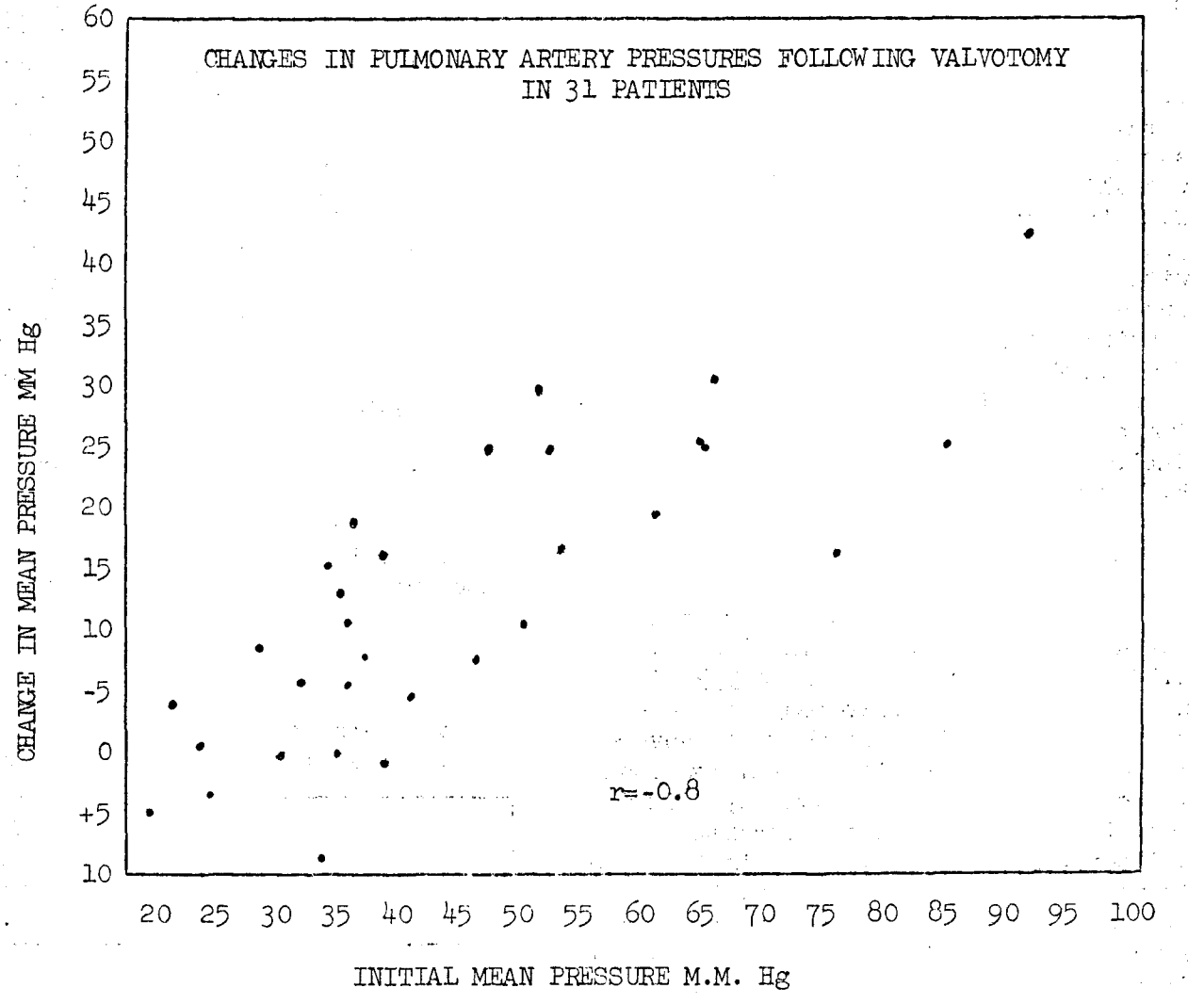
The average initial pressure for the class IV patients was higher than for the others and had fallen from 65 mm. Hg to 47 mm. Hg. This average fall in pressure was significant (P=.03). The pressure fell in 6 of the 8 patients, the largest decline being 43 mm. Hg and the smallest being 17. In 2 patients a small rise was obtained, 7 and 9 mm. Hg., respectively. In one patient the accuracy of the initial measurement was doubtful. In the other, extreme anxiety during the repeat catheterization resulted in an increase of cardiac output from 2.02 to 6.23 L./min. without evidence of dyspnea. The ability to increase cardiac output over threefold, notwithstanding the small rise in pressure, is an excellent index of improved circulatory function. As noted previously, all patients in this group were greatly benefited and the evidence for the clinical improvement was objective. Continued clinical improvement occurred despite the fact that all of them still had abnormally high pulmonary artery pressures (from 39 to 49 mm. Hg).

The 23 patients in class II-III had an average pressure of 38 mm. Hg which fell to 26, postoperatively. This fall in pressure though essentially the same as that of class IV patients, was of more statistical significance because of the larger number of patients (P<.001). The pressure decreased in 18 of the 23 patients, the decrease varying from 4 to 41 mm. Hg. Postoperative mean pressures of 15 to 19 mm. Hg were obtained in 5 patients and 6 others had pressures under 25 mm. Hg. This finding shows that the pulmonary hypertension of mitral stenosis can be almost completely alleviated. The pressure was essentially unchanged in 3 patients and it increased slightly in 2 patients. In 4 of these 5 patients, ob-

jective evidence for clinical improvement was lacking; in the one obvious therapeutic failure, a low salt regimen is needed to prevent edema. Two others, who were obvious therapeutic failures, had a fall in pressure, one of 8 mm. Hg and the other 17 mm. Hg.

The fall in mean pulmonary arterial pressure for the 31 individual patients is plotted against the initial level of pulmonary arterial pressure in Figure 1.

Figure 1



A highly significant negative correlation was obtained ($r=-0.8$ $P<.01$). Thus the higher the initial pressure, the greater the fall, and the lower the pressure the less the fall. A similar correlation has been described by Janton and co-workers.[31] Since the amount of disability is roughly proportional to the degree of pulmonary hypertension, the reduction of pulmonary pressures, in general, is greater in those patients with more advanced symptoms. These results support the contention that pulmonary hypertension is one of the most important criteria for surgery and that valvotomy is effective in lowering high pulmonary pressure.

C. Right Ventricular Filling Pressures

The average right ventricular diastolic pressure before valvotomy was 9 mm. in the eight class IV patients (see Table V). Postoperatively, the pressure fell to a normal value of 5. The average

difference is not of statistical significance, in part, perhaps, because of the small number of cases. Since increased filling pressure is a measure of right ventricular failure, the fall to normal probably has clinical significance. As a group, these patients were in borderline compensation before surgery and well compensated without mercurials or salt restriction after surgery. In 4 of the 8 patients the ventricular filling pressure fell from 14 mm. Hg to a normal of 6 mm. Hg, an average fall of 8 mm. Hg. In 3 with nearly normal pressures it did not change, and in one it was slightly increased. The striking fall in some of the most severely ill patients of the group fitted closely with the clinical improvement as manifested by disappearance of venous distention, hepatomegaly, and postoperative freedom from edema. In contrast, the 23 class II-III patients had normal filling pressures before and after surgery. Similar observations have been reported by Dexter.[17]

D. Resting Cardiac Output

The average resting cardiac output of the 8 patients in class IV was 3.07 L/min., a value which is 55% of normal. Following valvotomy, it rose to 4.21 L/min. Thus in the most severely incapacitated patients with low resting cardiac outputs, presumably "fixed", the blood flow increased approximately a liter per minute. Again this increase is not statistically significant but strongly indicates that better myocardial function is contributing to the clinical improvement. In contrast, the initial output of 4.52 L/min. for the 23 class II-III patients did not change appreciably. This group includes 3 patients who were therapeutic failures; their cardiac output was low before surgery (3.33 L/min.) and did not change.

E. The Mean Circulation Time

The mean circulation time from the pulmonary to the femoral artery[7] in the 6 class IV patients studied was 20.8 seconds initially and fell to 17.8 seconds. The values for 10 class II-III patients were 14.8 and 14.9 seconds. These changes are not significant but again the differences between the two groups (class IV vs. class II-III) is apparent.

F. The Arterial Oxygen Saturation

The arterial oxygen saturation showed a probably significant increase from 92 to 95% for the group of 31 patients (P=.05). It is possible that more sensitive methods of analysis in which oxygen tension was measured directly would show more striking results. Occasional patients have very marked unsaturation, values of 78% and 80% being obtained. This finding is most likely related to increased pulmonary capillary pressure causing interstitial alveolar edema with resultant increase in alveolar-blood oxygen gradient. With reduction in pressure and edema, the diffusion gradient is less.

G. Total and Central Blood Volume

Table VI presents values for the total blood volume and the central blood volume as determined in 13 cases.

TABLE VI

Total and Central Blood Volume for 13 Patients Undergoing Valvotomy

	Central Blood Volume c.c.	Total Blood Volume c.c.
Before	1035	5225
After	1001	4835
Predicted Normal	837	4292

The average total blood volume was increased to 5225 cc. initially (predicted normal 4292 cc.) and fell to 4835, a probably significant decrease of 390 cc. (P=.06). In the 3 class IV patients studied there was a decrease of approxi-

mately one liter pointing to the loss of occult edema. The average central blood volume was 1035 cc., 198 cc. larger than the predicted normal for the group. This did not change after surgery. Although objections have been raised as to the sensitivity of the technic for measuring central blood volume,[21] it may be concluded that there was no large decrease in intrathoracic blood volume to account for the relief of dyspnea experienced by these patients. Furthermore, the data lends support to the thesis that the pulmonary vascular tree is a relatively rigid system, particularly in patients with symptomatic mitral stenosis so that small changes in volume can lead to large changes in pressure.

H. Lung Volume

Table VII shows the mean total lung volume for 18 patients.

TABLE VII

	Total Lung Volume	Residual Volume	Ratio of Total Lung Volume to Residual Air %	Alveolar Nitrogen Retention Vol. %
Before	4475	2027	45	2.23
After	4114	1879	46	2.36
Predicted Normal	5183	1579	30	1.14

The average total lung volume was 4475 cc. This value is 14% smaller than the predicted normal for the group (5183 cc.). There was little change postoperatively. The residual volume was 2027 cc., a value 28% above the predicted normal for the group (1579 cc.). The alveolar nitrogen concentration was 2.23 vols. % and did not change. These data are slightly different from that of previous workers[9] who report normal or slightly reduced residual volume and a normal alveolar nitrogen concentration in patients with cardiac disease as compared to normals. Two of the patients were found to have emphysematous blebs on examination of the lung at surgery and one had known bronchial asthma. The measurements probably reflect a minimal degree of pulmonary emphysema in some of the patients. The relief of dyspnea experienced by all of these patients cannot be directly related to changes in lung volume or intra-alveolar mixing of gases as measured by this technic.

I. Calculated Mitral Valve Area and Pulmonary Resistance

Gorlin's formula[22] for calculation of the effective area of the stenotic mitral valve without regurgitation is:

$$MVA = \frac{MVF}{31 \sqrt{PC-5}}$$

where MVA = mitral valve area in sq. cm.

MVF = mitral valve flow in cc/sec.

$$= \frac{\text{cardiac output cc/min.}}{\text{diastolic filling period sec/min.}}$$

PC = pulmonary "capillary" or wedge pressure

5 = assumed left ventricular diastolic filling pressure

31 = empirical constant

Although theoretically valid, the applicability of the formula has been questioned since it depends upon the following assumptions:

1) The absence of mitral regurgitation.

2) That wedge pressures equal left atrial pressure.

3) A normal left ventricular diastolic filling pressure.

4) The constancy of a correction factor for such variables as turbulence and pulsatile flow.

The wedge pressure has been found to equal the left atrial pressure in certain cases of atrial septal defect,[18,19,29] and not in others.[11] Recent work in dogs has failed to correlate closely wedge pressure with pulmonary vein pressure when the latter was below 17 mm. Hg.[26] In our experience, satisfactory wedge pressures are seldom obtained in those cases where the mean pulmonary artery pressure is over 60 mm. Hg (see case M.L. Table VIII). Satisfactory wedge pressures permitting calculation of valve areas have been obtained in 14 patients preoperatively and 11 postoperatively. Correlation of calculated valve area with clinical status agrees with the pattern described by Dexter[17] (see Section II). "Arteriolar" and total pulmonary resistance roughly follow the same pattern. In two fatal cases, measurement of valve area in the postmortem specimen as compared to calculated valve area in sq. cm. gave the following results: 0.4 actual, 0.9 calculated and 0.1 actual, 0.6 calculated.

Data for 6 patients whose valve areas were calculated both before and after valvotomy are presented in Table VIII along with functional classification.

TABLE VIII

Valve Area, Wedge Pressure and Pulmonary Resistance Before and After Commissuratomy

		Class	Calculated Mitral Valve Area	Mean Pressure		Resistance	
				Wedge	P.A.	Pulmonary "Arteriolar"	Total Pulmonary
T. J.	Before	III	0.7	32	66	847	1638
	After	I	2.6	11	25	251	449
L. C.	Before	III	1.1	38	52	230	855
	After	I	1.3	15	22	139	437
T. W.	Before	III	0.9	23	35	238	391
	After	II	1.9	20	35	240	315
P. R.	Before	III	1.1	16	37	485	854
	After	I	2.4	9	30	361	516
M. M.	Before	III	1.1	31	43	178	637
	After	II	1.6	14	26	215	466
M. L.	Before	IV	.3	65	92	884	3019
	After	I	.5	44	49	130	1272
Average	Before	III-IV	.8	34	54	477	1232
	After	I-II	1.7	19	31	223	576
Normal			4-6 cm.	6-12	11-13	67	166

The measurements indicate the effectiveness of surgery in doubling the effective mitral valve area and halving the wedge pressures and resistances. In one patient (T.J.) the theoretically desirable calculated valve area of 2.6 sq. cm., was obtained and was borne out by the clinical result. An excellent clinical result was also obtained in case M.L. but calculated valve areas do not reflect much change probably because of unreliable wedge pressures. Subjective improvement was obtained by case T.W. and even though pulmonary artery pressure did not change, calculated valve area doubled. Biopsy specimen revealed severe medial hypertrophy of the small pulmonary arteries, and in this case "arteriolar" resistance did not change. Pulmonary arteriolar resistance, however, did decrease in all but 2 patients. The fall in "arteriolar" resistance within the short postoperative period suggests the presence of a vaso-constrictor mechanism at the level of the pulmonary "arterioles" which is relieved by surgery. If valid, these criteria are objective evidence of the value of surgery not only in increasing the effective valve area but also in reducing a portion of the pulmonary "arteriolar" resistance.

J. Electrocardiogram

The average frontal plane electrical axis for this group was +95°. Following surgery there was a definite shift to the left---the average axis being +78°. Ten patients with electrical axes of less than +90° had an average pressure of 33 mm. Hg. The 21 patients having electrical axes greater than +90° or a pattern of right ventricular preponderance had higher pressures and a greater fall (Table IX). There was no correlation in individual cases between the initial pressures and initial axis or change in pressure and change in axis. Nevertheless, the axis shift probably reflects the reduction of pulmonary hypertension, even though some positional changes may have resulted from surgery.

TABLE IX

	Before		After	
ECG	Number of Patients	Average Mean P.A.P.	Number of Patients	Average Mean P.A.P.
Axis $\angle$ 45°---89°	10	33	20	27
Axis $\geq$ 90° or greater	21	50	11	38
R.V.P.	19	50	17	37

K. Lung Biopsy

In an attempt to evaluate pulmonary pathology found in mitral stenosis,[32,34] lung biopsies have been studied in 28 of the 31 patients. Seven showed an abnormal degree of arteriosclerosis and 4 of the 7 were in class IV. The average pulmonary artery pressure of 61 mm. Hg was a probably significant elevation above the mean of the entire group. It would seem that hypertension of the lesser circulation accelerates arteriosclerosis of the smaller pulmonary arteries in a manner similar to that seen in systemic hypertension. Eleven cases showed chronic passive congestion and the remaining 10 had no significant

changes. Mean pulmonary artery pressures for each group before and after surgery are given in Table X. Postoperatively, the average pulmonary

TABLE X

Changes in Pressure Related to Lung Biopsy Findings

Biopsy		Arteriosclerosis	Chronic Passive Congestion	No Significant Changes
Number of Patients		7	11	10
Mean Pulmonary Artery Pressure	Before	61	45	47
	After	33	33	36

artery pressures for the three different groups had fallen to the same level. Clinical improvement was if anything more remarkable in the group with arteriosclerotic changes. In this series, then, the presence of arteriosclerosis of the smaller pulmonary arteries in no way prevented the relief of pulmonary hypertension or symptomatic improvement.

L. Preliminary Long Term Follow-Up Studies

Five of the 31 patients have been studied from 13 to 21 months postoperatively. Three of the patients had measurements recorded 1 to 3 months after surgery as well. With the excep-

TABLE XI

LONG TERM FOLLOW-UP - Preliminary Results

Date of Study	Date of Surgery	Pulmonary Artery Pressures		
		Systolic	Diastolic	Mean
25 F 2/13/51	3/27/51	99	46	62
J.B. 4/17/51		64	---	--
9/22/52		56	27	43
48 F 8/23/51	8/30/51	43	24	30
D.W. 10/6/52		38	22	30
16 M 12/5/50	12/26/50	54	22	33
C.D. 4/12/51		46	37	42
10/2/52		43	--	--
29 M 7/16/51	8/1/51	40	18	21
J.C. 10/9/51		29	12	17
10/2/52		31	13	17
46 F 6/19/51	7/17/51	17	9	12
F.K. 10/16/52		22	11	17

tion of one patient (F.K.), none of the group showed a significant increase in pressure. The greatest fall in pressure in these 3 patients occurred in the immediate postoperative period, although clinical improvement continued for many months. These data, though meager, suggest that the greatest fall in pressure occurs within 6 to 8 weeks postoperatively and that the pulmonary artery pressure is then stable for one to 2 years.

(VII) Discussion

The results of this study indicate that the majority (over 80%) of selected patients undergoing successful fracture of a stenosed mitral valve are improved. Not only is there subjective improvement in dyspnea and increase in exercise tolerance but symptoms known to be related to pulmonary hypertension, such as hemoptysis, are alleviated. Those previously needing regimentation to prevent the recurrence of right heart failure are now free of edema on a normal salt intake without diuretics.

Many discrepancies, however, are found in attempting to correlate the degree of clinical improvement to changes in measurable physiologic functions in the individual patient. In many instances, the immediate clinical improvement appears to be out of proportion to changes in hemodynamics that can be measured by present methods. The importance of psychologic factors bearing on the therapeutic result is well known and cannot be minimized. An attack at the source of the disorder is now offered to victims of a disease that can be progressively disabling and that formerly held no real hope for therapy. For example, one very disabled patient upon being informed that cardiac catheterization was scheduled in order to evaluate the desirability of valvotomy had a complete change of personality and a spontaneous diuresis with a weight loss of 8 pounds.

Nevertheless, the postoperative studies show that a significant reduction of pulmonary artery pressure, an average decrease of 13 mm. Hg for the group, was achieved. Normal or near normal pulmonary artery pressures were obtained in about 12% of the patients, all of whom had had progressive symptoms but none of whom were in the advanced stages of the disease. This finding is most encouraging and validates the soundness of the surgical procedure. The data at hand, unfortunately, do not enable the clinician to select with certainty those particular patients who will benefit to the ideal extent of being left with a nearly normal pulmonary circulation. In general, these patients were in their thirties, gave a history of 5 years' duration or less, had only a moderate degree of pulmonary hypertension and right ventricular enlargement, and relatively pliable, easily fractured valves at surgery.

Most important, however, is the finding of a highly significant correlation between the degree of the pulmonary hypertension and the fall in pressure following surgery (Figure 1). Those patients most in need of symptomatic relief and having the greatest degree of pulmonary hypertension are the ones most obviously benefited clinically, paralleling the reduction in pulmonary pressure. This reduction in pressure appears to be independent of other known variables contributing to the disabling symptomatology including, with rare exception, sclerotic changes in the pulmonary vessels as observed in biopsy specimens. Calculation of the valve orifice areas, when feasible, indicates that on the average it is doubled by valvotomy and may approach the theoretically ideal area of 2.5 sq. cm. Left atrial pressure, when estimated by satisfactory wedge (pulmonary "capillary") pressure, is reduced well below the colloid osmotic pressure of the blood. Pulmonary "arteriolar" resistance as well as overall resistance is halved (Table VIII). Hence, in addition to the lessened resistance achieved directly by increasing the mitral orifice, there is a release of a portion of the "arteriolar" spasm contributing to the pulmonary hypertension. This reduction of pulmonary pressure would logically lessen the work of the right ventricle. Except for 6 out of 8 patients falling into the class IV cate-

gory, however, improvement could not be related to increase in resting cardiac output, which showed no consistent changes in the group as a whole. Nor could relief of dyspnea be related to changes in the volume of blood in the lungs or to changes in any of the subdivisions of the total lung volume. Both disability and clinical improvement, with few exceptions, is best described in terms of pulmonary vascular pressure change. It follows then that since the most consistently measurable result of valvotomy is a reduction of pulmonary pressures, the procedure should be reserved for those patients having pulmonary hypertension.

Patients in functional class IV constitute a special group worthy of comment. Not only is the evidence for clinical benefit more objective but these patients have higher pulmonary artery pressures to begin with and obtain a greater fall. Most of them obtain an increase in resting cardiac output amounting, on the average, to a liter a minute as well as a reduction in total blood volume. The latter is of interest since no patient was operated with obvious clinical edema. It points to improved renal hemodynamics leading to the loss of occult edema and is well correlated clinically with decrease in venous distention and hepatomegaly. Although the operative mortality in this group of class IV patients is over 25%, it is not high in view of the poor general status of these patients and in view of the fact that the untreated disease in this stage is usually fatal within a year. The dramatic results obtained in the survivors justify the risk.

The ultimate effectiveness of valvotomy on the morbidity and mortality of patients with mitral stenosis will require long term evaluation of a large number of treated cases from large centers. Preliminary observations in this study indicate continued clinical improvement in 2 class IV patients followed over a 2 year period even though maximum reduction of pulmonary pressure occurred in the first 2 months. The significant point is that not only was progression of pulmonary hypertension arrested, but life span certainly was prolonged in a state of relative good health. The condition of the valve at the time of surgery will have considerable bearing on the duration of benefit.[24] Rigid, thickened or extensively calcified valves logically would prevent a long term good result, whereas pliable, easily fractured ones favor it. Calcification as detected fluoroscopically is a poor guide to actual state of the valve. The evaluation of long term results in mild to moderately disabled patients, on the other hand, will be more difficult. Cognizance must be taken of the fact that a certain number of such patients may have disabling symptoms, such as pulmonary edema and rarely hemoptysis, then stabilize to get along well with only moderate restriction of activity for long periods of time up to 10 or even 20 years.[24,33]

The results of this study and the foregoing discussion point up 2 major indications for surgery: (1) disabling symptoms associated with established pulmonary hypertension and (2) systemic embolization. Factors initially thought to constitute contraindications including age, calcification of the valve, minimal aortic insufficiency, auricular fibrillation, congestive failure, previous systemic emboli, previous pulmonary infarction, low vital capacity, essential systemic hypertension, healed bacterial endocarditis, loud apical systolic murmurs, in and of themselves, appear to have no influence on a favorable operative result. Dynamic aortic lesions with left ventricular enlargement prevent a favorable result although insufficient experience is not yet available to define exact limits. Congestive failure per se is no contraindication providing clinical compensation can be achieved. This may require several weeks of hospital regimen. Only a few cases of mitral stenosis will prove to have intractable congestive failure under these conditions. Subacute bacterial endocarditis and obvious rheumatic activity are definite though temporary contraindications. Predominate mitral insufficiency is a contraindication. Diagnosis is often difficult and depends largely on evaluation of apical systolic

murmurs. Wedge pressure curves may help in diagnosis, but are not uniformly obtainable or reliable.

Selection of ideal candidates for valvotomy on clinical grounds alone is possible in many patients between the ages of 20 - 40, giving a history of progressive dyspnea with hemoptysis or transient congestive failure in whom the murmur is typical, the radiologic picture distinctive, and the electrical axis +90°. Nevertheless, in our opinion, quantitive control over selection of patients by cardiac catheterization data is desirable if not mandatory. Although the examiner with broad experience in cardiac catheterization can predict the range of findings, performing the test will prevent many an error in judgment by both expert and novice, particularly in patients with atypical features. Atrial septal defects, for example, have been encountered that resembled mitral stenosis in all its clinical aspects, including the apical diastolic murmur.

Surgery has not been performed on asymptomatic patients or those with mean pulmonary artery pressures under 20 mm. Hg in line with the policy of Dexter[17] and Harken.[27] We are strongly opposed to the stand taken by Bailey and his group[3] in advocating surgery as soon as the mitral stenotic murmur is diagnosed. Not only is minimal mitral stenosis compatible with longevity and ordinary activity, but as pointed out in section II a critical reduction in valve orifice area must occur before hemodynamic changes ensue to produce symptoms. Patients in this category undergoing surgery would probably obtain a good result, but may have been asymptomatic for years without surgery. Moreover, the surgical procedure is palliative and not definitive. How long palliation will last and whether or not stenosis will recur are as yet unanswered questions. There is no reason to believe that recurrent rheumatic activity or subacute bacterial endocarditis is prevented.

Occasional patients may be encountered who have had pulmonary edema or severe exertion, but are asymptomatic and have normal hemodynamics at rest. Exercise tests at the time of catheterization may reveal an abnormal pattern and a critically small calculated valve orifice area. Data on such a case has recently been published by Cournand.[21] The only patient in this series with a similar history had a resting pulmonary artery pressure of 30 mm. Hg. Our material to date supports the view that most patients with disabling symptoms due to mitral stenosis have some degree of pulmonary hypertension at rest. Surgery should be postponed in those patients having normal or near normal pulmonary pressures as determined by cardiac catheterization.

Systemic embolization constitutes a special indication for valvotomy whether or not there are accompanying symptoms referrable to the pulmonary circuit. Chances of a recurrence to vital areas are good and the results of conservative therapy poor so that an aggressive approach is warranted. Eleven patients in this series had evidence for systemic emboli; two had complete hemiplegia. In only two of the total group was there evidence of atrial thrombosis at surgery. One other was explored, but massive atrial thrombosis denied access to the valve. None have had a recurrant embolus to date, nor have systemic emboli been reported so far in patients following recovery from valvotomy. Final judgment is reserved, but it appears that valvotomy sufficiently alters pressure-flow relationships in the left atrium to prevent thrombus formation as evidenced by systemic embolization.

Summary

1. An analysis of 52 patients with mitral stenosis selected for valvotomy is presented.

2. Correct therapeutic management of these patients is a team venture requiring the combined efforts of cardiologist, thoracic surgeon and anesthesiologist.

3. Overall clinical and physiologic results indicate that over 80% of carefully selected patients will obtain immediate benefit from mitral valvotomy.

1. Allen, D. S., and Graham, E. A.
Intracardiac surgery a new method.
J.A.M.A. 79:1028, 1922.

2. Bailey, C. P.
The surgical treatment of mitral stenosis.
Dis. of Chest. 15:377-397 (April) 1949.

3. Bailey, C. P., Bolton, H. E., Redondo - Ramirez, H. P.
Surgery of the mitral valve.
Surg. Clin. N. Am. P1807-1847 (Dec.) 1952.

4. Bailey, C. P., Glover, R. P., and O'Neill, T. J. E.
Surgery of mitral stenosis.
J. Thoracic Surg. 19:16, 1950.

5. Baker, C., Brock, R. C., Campbell, M., and Wood, P.
Valvotomy for mitral stenosis.
Brit M. J. 4767:1043-1055 (17 May) 1952.

6. Borden, C. W., Ebert, R. V., Wilson, R. H., Wells, H. S.
"Studies of the pulmonary circulation. I. The circulation time from the pulmonary artery to the femoral artery and the quantity of blood in the lungs in normal individuals."
J. Clin. Investigation 28: 1134-1137, (Sept.) 1949.

7. Borden, C. W., Ebert, R. V., Wilson, R. H., Wells, H. S.
"Studies of the pulmonary circulation. II. The circulation time from the pulmonary artery to the femoral artery and the quantity of blood in the lungs in patients with mitral stenosis and in patients with left ventricular failure."
J. Clin. Investigation 28:1138-1142 (Sept.) 1949.

8. Borden, C. W., Ebert, R. V., Wilson, R. H., and Wells, H. S.
Pulmonary hypertension in heart disease.
New England J. Med. 242:529-534 (Apr.) 1950.

9. Borden, C. W., Ebert, R. V., Wilson, R. H. and Wells, H. S.
"A comparison of the effect of voluntary hyperventilation in normal persons, patients with pulmonary emphysema and patients with cardiac disease."
J. Lab. & Clin. Med. 36:119-126 (July) 1950.

10. Brunton, L.
Preliminary note on the possibility of treating mitral stenosis by surgical methods.
Lancet 1:352, 1902.

11. Calazel, T., Guand, R., Daley, R., Draper, A., Foster, G. and Bing, R. J.
Comparison of right auricular, left auricular, and the pulmonary arterial pressures in cases of atrial septal defects.
Bull. Johns Hopkins Hosp., 88:20, 1951.

12. Cournand, A., Baldwin, E. D., Darling, R. C., Richards, D. W.
Studies on intrapulmonary mixture of gases: IV significance of pulmonary emptying rate and modified open circuit measurement of residual air.
J. Clin. Investigation, 20:681-684 (Oct.) 1941.

13. Cournand, A., Ranges, H. A.
Catheterization of right auricle in man.
Proc. Soc. Exp. Biol. & Med. 46:452, 1941.

14. Cournand, A., Riley, R. L., Himmelstein, A., and Austrian, R.
Pulmonary circulation and alveolar ventilation - perfusion relationships after pneumonectomy.
J. Thoracic Surg. 19:80, 1950.

15. Criteria Committee of the New York Heart Association.
Nomenclature and criteria for diagnosis of diseases of the heart.
ed. 4 New York, J. J. Little & Ives, 1945 P. 72-3.

16. Cutler, E. C., Levine, S. A., and Beck, C. S.
Surg. treat. of mitral stenosis: Exp. and clinical studies,
Arch. Surg. 9:689, 1924.

17. Dexter, L.
Pathologic physiology of mitral stenosis and its surgical implications.
Bull. New York Acad. Med. 28:90-105 (Feb.), 1952.

18. Dexter, L., Dow, J. W., Haynes, F. W., Whittenberger, J. L., Ferrier, B. G., Goodale, W. T., Hellems, H. K.
Studies of pulmonary circulation in man at rest; normal variation and the interrelation between increased pulmonary blood flow, elevated pulmonary arterial pressure and high pulmonary capillary pressure.
J. Clin. Investigation, 29:602, 1950.

19. Dow, J. W., and Gorlin, R.
Pulmonary capillary pressure as index of left atrial mean pressure in dogs.
Federation Proc. 9:33, 1950.

20. Edwards, J. E., Tompkins, R. G., Hood, R. T., Kirklin, J. W. and Burchell, H. B.
Biopsy of the lung and cardiac catheterization studies in patients treated surgically for mitral stenosis.
Proc. Central Soc. Clin. Research, 25:26, 1952.

21. Ferrer, M. I., Harvey, R. M., Cathcart, R. T., Cournand, A., Richards, D. W. Jr.
Hemodynamic studies in rheumatic heart disease.
Circ. 6:688-710 (Nov.), 1952.

22. Gorlin, R. and Gorlin, S. G.
Hydraulic formula for calculation of the area of the stenotic mitral valve, other cardiac valves, and central circulatory shunts. I.
Am. Heart J. 41:1-29 (Jan.), 1951.

23. Gorlin, R., Sawyer, C. G., Haynes, F. W., Goodale, W. T., and Dexter, L.
Effects of exercise on circulatory dynamics in mitral stenosis. III.
Am. Heart J. 41:192-203 (Feb.) 1951.

24. Graham, G. K., Taylor, J. H., Ellis, L. B., Greenberg, D. J., Robbins, S. L.
Studies in mitral stenosis II. A correlation of post mortem findings with the clinical course of the disease in 101 cases.
Arch. Int. Med. 88:532-547 (Oct.) 1951.

25. Griffith, G. C., Miller, H., Cosby, R. S., Levinson, D. C., Dimitroff, S. P., Zinn, W. J., Oblath, R. W., Herman, L. M., Johns, V. J. Jr., Meyer, B. W. and Jones, J. C.
The selection and medical management of patients with mitral stenosis treated by mitral commissurotomy.
Circ. 7:30-36 (Jan.) 1953.

26. Haddy, F. J., Ferrier, A. L., Hannon, D. W., Alden, J. F., Adams, W. L., Baronofsky, I. V.
An evaluation of wedge pressures in dogs under conditions of normal and elevated pulmonary vascular pressures.
(In Press)

27. Harken, D. E., Dexter, L., Ellis, L. B., Ferrand, R. E. & Dickson, J. F.
Surg. of mitral stenosis III. Finger-fracture valvuloplasty.
Ann. Surg. 134:722-742, 1951.

28. Harken, D. E., Ellis, L. B., Ware, P. F., and Norman, L. R.
Surg. treat. of mitral stenosis.
New Eng. J. Med., 239:801, 1948.

29. Hellems, H. K., Haynes, F. W., Dexter, L., and Kinney, T. D.
Pulmonary capillary pressures in animals estimated by venous and arterial catheterization.
Am. J. Physiol. 155:98, 1948.

30. Hickam, J. B. and Cargill, W. H.
Effects of exercise on cardiac output and pulmonary artorial pressure in normal persons and in patients with cardiovascular disease and pulmonary emphysema.
J. Clin. Investigation 27:10, 1948.

31. Janton, O. H., Glover, R. P., O'Neill T. J. E., Gregory, J. E., and Froio, G. F.
Results of the surgical treatment for mitral stenosis. Analysis of one hundred consecutive cases.
Circ. 6:321-333 (Sept.) 1952.

32. Larrabee, W. F., Parker, R. L., and Edwards, J. E.
Pathology of intrapulmonary arteries and arterioles in mitral stenosis.
Proc. Staff Meet. Mayo Clin. 24:316, 1949.

33. Love, D. E., and Levine, S. A.
Mitral stenosis and long-lasting congestive heart failure or auricular fibrillation.
New England J. Med. 247:917-920 (11 Dec.) 1952.

34. Parker, F. Jr., Weiss, S.
The nature and significance of the structural changes in the lungs in mitral stenosis.
Am. J. Path. 12:533, 1936.

35. Souttar, P. W.
Surg. treat. of mitral stenosis.
Brit. M. J. 2:603, 1925.

36. Spiegl, R. J., Long, J. B., & Dexter, L.
Clinical observations in patients undergoing finger fracture mitral valvuloplasty: II. Electrocardiographic observations.
Am. J. Med. 12:631-638 (June) 1952.

37. Steell, G.
Murmur of high pressure in pulmonary artery.
M. Chron. 9:182-188, 1888.

38. Stewart, G. N.
Pulmonary circulation time, quantity of blood in the lungs and output of the heart.
Am. J. Physiol. 58:20, 1921.

II. MEDICAL SCHOOL NEWS

Coming Events

February 2-4	Continuation Course in Clinical Chemistry for General Physicians
February 5-7	Continuation Course in Cancer Detection for General Physicians
February 12-14	Continuation Course in Cardiovascular Diseases for General Physicians
February 16-18	Continuation Course in Recent Advances in Diagnosis for Internists
February 17	Phi Delta Epsilon Lecture; "Iron Metabolism and Iron Deficiency Anemia"; Dr. Carl V. Moore, Professor, Department of Medicine, Washington University School of Medicine, St. Louis; Owre Amphitheater; 8:00 p.m.
March 2-4	Continuation Course in Clinical Dietetics

* * *

Continuation Course

The University of Minnesota, in conjunction with the Minnesota Heart Association, will present a continuation course in Cardiovascular Diseases at the Center for Continuation Study on February 12 to 14, 1953. Intended primarily for physicians engaged in general practice, the course will emphasize treatment of the more common cardiovascular disorders. A discussion of peripheral vascular disease will be included. The program will also feature a clinical correlation session. The guest faculty will include the eminent cardiologist, Dr. Arthur M. Master, Associate Professor of Clinical Medicine, Columbia University College of Physicians and Surgeons, New York City. Clinical and full-time members of the staff of the University of Minnesota Medical School and the Mayo Foundation will complete the faculty.

* * *

Faculty News

Dr. James Rogers Fox, Student Health Service physician and Instructor in Public Health, recently spoke at the annual meeting of the Renville County Medical Society and at a meeting of the Minneapolis Department of Health, discussing his experiences of last summer when he participated in the British National Health Plan. He will speak on the same subject at a meeting of the Ramsey County Medical Auxiliary in the near future. Dr. Fox has recently opened an office in the Northwestern Bank Building in Minneapolis for the practice of occupational medicine. He is devoting half-time to his office, the remainder to the Student Health Service.

* * *

New Minnesota Medical Foundation Members

Mr. W. C. MacFarlane, Minneapolis
Mr. Joseph Paper, St. Paul
Miss Mary Paper, St. Paul
Mr. K. W. McKee, St. Paul
Mr. E. W. Wylie, Los Angeles, California
Mrs. D. B. Rosenblatt, Minneapolis
Mr. Herman Rosenblatt, Minneapolis
Mr. Bruce Boynton, Minneapolis
Mr. Elmer H. Smith, Minneapolis
Mrs. Albert G. Minda, Minneapolis
Carroll D. Burk, M.D., Denver, Colorado
H. R. Leland, M.D., Minneapolis
Mr. Sam Mairs, Minneapolis
Mr. R. B. Rathbun, Minneapolis
Mr. Jack Phelan, Minneapolis
Mr. Frank A. Nelson, Minneapolis

III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

February 2 - 7, 1953

Monday, February 2

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and - Staff; Todd Amphitheater, U. H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; W-612, U. H.

10:00 - 12:00 Neurology Rounds; A. B. Baker and Staff; Station 50, U. H.

11:30 - Tumor Conference; Doctors Kremen, Moore, and Stenstrom; Todd Amphitheater, U. H.

11:30 - 12:30 Physical Medicine Staff Seminar; Functional Results of Femoral Head Arthroplastics or Prostheses; A. B. Quiggle; Heart Hospital Auditorium.

12:15 - Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.

12:30 - 1:30 Physiology Seminar; Acetate Metabolism in the Mammary Gland; William E. Peterson; 214 Millard Hall.

1:30 - 2:30 Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; U. H.

4:00 - Pediatric Seminar; Lead Poisoning; Margaret Bailly; Sixth Floor West, U. H.

4:00 - 5:30 Seminar on Fluid and Electrolyte Balance; Gerald T. Evans; Todd Amphitheater, U. H.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

4:30 - Public Health Seminar; 15 Owre Hall.

4:30 - 6:00 Physiology 114A and Cancer Biology 140 -- Research Conference on Cancer, Nutrition, and Endocrinology; Drs. Visscher, Bittner, and King; 129 Millard Hall.

5:00 - 6:00 Urology-Roentgenology Conference; C. D. Creevy, O. J. Baggenstoss, and Staff; Eustis Amphitheater.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Eldon Berglund; Newborn Nursery, Station C.

10:30 - 12:00 Tuberculosis and Contagion Rounds; Thomas Lowry; Station M.

11:00 - Pediatric Rounds; Erling Platou; Station K.

12:30 - Surgery Grand Rounds; Dr. Zierold; Sta. A.

1:00 - X-ray Conference; Classroom, 4th Floor.

2:00 - Pediatric Rounds; Robert A. Ulstrom; Stations I and J.

Monday, February 2 (Cont.)

Ancker Hospital

8:30 - 10:00 Chest Disease Conference

1:00 - 2:00 Medical Grand Rounds.

Veterans Administration Hospital

8:00 - 9:00 Neuroradiology Conference; J. Jorgens, R. C. Gray; 2nd Floor Annex.

9:00 - G.I. Rounds; R. V. Ebert, J. A. Wilson, Norman Shrifter; Bldg. I.

11:30 - X-ray Conference; J. Jorgens; Conference Room, Bldg. I.

2:00 - Psychosomatic Rounds; Bldg. 5.

Tuesday, February 3

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Pediatric Conference; L. G. Rigler, I. McQuarrie and Staff; Eustis Amphitheater, U. H.

9:00 - 12:00 Cardiovascular Rounds; Station 30, U, H.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 102 I. A.

12:30 - 1:30 Physiology 114D -- Current Literature Seminar; 129 Millard Hall.

4:00 - 5:00 Pediatric Rounds on Wards; I. McQuarrie and Staff; U. H.

4:30 - 5:30 Clinical-Medical-Pathological Conference; Todd Amphitheater, U. H.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital

5:00 - 6:00 X-ray Conference; Presentation of Cases by University Hospitals Staff; Eustis Amphitheater, U. H.

Ancker Hospital

8:00 - 9:00 Fracture Conference; Auditorium.

8:30 - 9:30 Medical-Roentgenology Conference; Auditorium.

1:00 - 2:30 X-ray - Surgery Conference; Auditorium.

Minneapolis General Hospital

9:30 - 10:30 Obstetrics and Gynecology Staff Rounds; William P. Sadler and Staff; 301 Harrington Hall.

10:00 - Pediatric Rounds; Spencer F. Brown; Stations I and J.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station F.

12:30 - Grand Rounds; Fractures; Sta. A; Willard White, et al.

12:30 - Neuroroentgenology Conference; O. Lipschultz, J. C. Michael and Staff.

12:30 - EKG Conference; Boyd Thomes and Staff; 302 Harrington Hall.

1:00 - Tumor Clinic; Drs. Eder, Cal, and Lipschultz.

1:00 - Neurology Grand Rounds; J. C. Michael and Staff.

Tuesday, February 3 (Cont.)

Veterans Administration Hospital

7:30 - Anesthesiology Conference; Conference Room, Bldg. I.

8:30 - Infectious Disease Rounds; Dr. Hall.

8:30 - Surgery Staff Seminar; Physiology of Colon; Norton Rogin; Medical Conference Room, Bldg. I.

9:00 - Liver Rounds; Drs. Nesbitt and MacDonald.

9:30 - Surgery-Pathology Conference; Conference Room, Bldg. I.

10:30 - Surgery Tumor Conference; L. J. Hay, J. Jorgens; Conference Room, Bldg. I.

1:00 - Review of Pathology, Pulmonary Tuberculosis; Conference Room, Bldg. I.

1:30 - Combined Medical-Surgical Chest Conference; Conference Room, Bldg. I.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III.

3:30 - 4:20 Autopsy Conference; E. T. Bell and Donald Gleason; Conference Room, Bldg. I.

Wednesday, February 4

Medical School and University Hospitals

8:00 - 9:00 Roentgenology-Surgical-Pathological Conference; Paul Lober and L. G. Rigler; Todd Amphitheater, U. H.

11:00 - 12:00 Pathology-Medicine-Surgery Conference; Medicine Case; O. H. Wangensteen, C. J. Watson and Staff; Todd Amphitheater, U. H.

12:30 - 1:30 Radioisotope Seminar; Film: The Radioisotope; Methodology; Training Film on Planning of the Isotope Experiment; (Part IV in the series); 12 Owre Hall.

1:30 - 3:00 Physiology 114B -- Circulatory and Renal System Problems Seminar; Dr. M. B. Visscher, et al; 214 Millard Hall.

4:00 - 5:30 Physiology 114C -- Permeability and Metabolism Seminar; Nathan Lifson; 214 Millard Hall.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

5:00 - 5:50 Urology-Pathological Conference; C. D. Creevy and Staff; Eustis Amphitheater, U. H.

8:00 - 10:00 Dermatological-Pathology Conference; Review of Histopathology Section; R. Goltz; Todd Amphitheater, U. H.

Ancker Hospital

8:30 - 9:30 Clinico-Pathological Conference; Auditorium.

2:00 - 4:00 Medical Ward Rounds;

3:30 - 4:30 Journal Club; Surgery Office.

Wednesday, February 4 (Cont.)

Minneapolis General Hospital

9:30 - Pediatric Rounds; Max Seham; Stations I and J.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station D.

11:00 - Pediatric Seminar; Arnold Anderson; Classroom, Station I.

11:00 - Pediatric Rounds; Erling S. Platou; Station K.

12:00 - Surgery Seminar; Dr. Zierold; Classroom.

12:15 - Pediatrics Staff Meeting; Classroom, Station I.

1:30 - Visiting Pediatric Staff Case Presentation; Station I, Classroom.

Veterans Administration Hospital

8:30 - 10:00 Orthopedic X-ray Conference; E. T. Evans and Staff; Conference Room; Bldg. I.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker.

2:30 - 4:00 Psychosomatic Rounds; C. K. Aldrich; Conference Room, Bldg. I.

4:00 - Combined Medical-Surgical Conference; Conference Room, Bldg. I.

7:00 p.m. Lectures in Basic Science of Orthopedics; Conference Room, Bldg. I.

Thursday, February 5

Medical School and University Hospitals

8:00 - 9:00 Vascular Rounds; Davitt Felder and Staff Members from the Departments of Medicine, Surgery, Physical Medicine, and Dermatology; Heart Hospital Amphitheater.

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Todd Amphitheater, U. H.

12:30 - Physiological Chemistry Seminar; Effect of Anticoagulants on Electrophoretic Patterns of Serum Proteins; T. Pacholl; 214 Millard Hall.

1:30 - 4:00 Cardiology X-ray Conference; Heart Hospital Theatre.

4:00 - 5:00 Physiology-Surgery Conference; Todd Amphitheater, U. H.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hansen and Staff; E-534, U. H.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital

5:00 - 6:00 Radiology Seminar; Presentation of Cases from Miller Hospital; Drs. Peterson and Corrigan; Eustis Amphitheater, U. H.

7:30 - 9:30 Pediatric Cardiology Conference and Journal Club; Review of Current Literature 1st hour and Review of Patients 2nd hours; 206 Temporary West Hospital.

Ancker Hospital

4:00 - Medical-Pathological Conference; Auditorium.

Thursday, February 5 (Cont.)

Minneapolis General Hospital

9:30 - Neurology Rounds; Heinz Bruhl; Station I.

10:00 - Pediatric Rounds; Spencer F. Brown; Station K.

10:00 - Psychiatry Grand Rounds; J. C. Michael and Staff; Sta. H.

1:00 - Fracture - X-ray Conference; Dr. Zierold; Classroom.

1:00 - House Staff Conference; Station I.

2:00 - 4:00 Infectious Disease Rounds; Classroom.

4:00 - 5:00 Infectious Disease Conference; Wesley W. Spink; Classroom.

Veterans Administration Hospital

8:00 - Surgery Ward Rounds; Lyle Hay and Staff; Ward 11.

8:00 - Surgery Grand Rounds; Conference Room, Bldg. I.

11:00 - Surgery-Roentgen Conference; J. Jorgens; Conference Room, Bldg. I.

Friday, February 6

Medical School and University Hospitals

8:00 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:30 - 11:50 Medicine Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:30 - 11:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

11:45 - 12:50 University of Minnesota Hospitals Staff Meeting; The Pathogenesis of Rheumatic Fever; Lewis Thomas, Floyd Denny, Robert A. Good, Richard T. Smith; Powell Hall Amphitheater.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

3:00 - 4:00 Neuropathological Conference; F. Tichy; Todd Amphitheater, U. H.

4:00 - 5:00 Physiology 124 -- Seminar in Neurophysiology; Ernst Gelhorn; 113 Owre Hall.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

5:00 - Urology Seminar and X-ray Conference; Eustis Amphitheater, U. H.

Ancker Hospital

1:00 - 3:00 Pathology-Surgery Conference; Auditorium.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Wallace Lueck; Station J.

Friday, February 6 (Cont.)

Minneapolis General Hospital (Cont.)

10:30 - Pediatric Surgery Conference; Oswald Wyatt; Tague Chisholm; Station I, Classroom.

12:00 - Surgery-Pathology Conference; Dr. Zierold, Dr. Coe; Classroom.

1:00 - 3:00 Clinical Medical Conference; Thomas Lowry; Classroom, Station M.

1:15 - X-ray Conference; Oscar Lipschultz; Classroom, Main Bldg.

2:00 - Pediatric Rounds; Robert Ulstrom; Stations I and J.

Veterans Administration Hospital

1:00 - Pathology Slide Conference; E. T. Bell; Conference Room, Bldg. I.

10:30 - 11:20 Medicine Grand Rounds; Conference Room, Bldg. I.

Saturday, February 7

Medical School and University Hospitals

7:45 - 8:50 Orthopedic X-ray Conference; W. H. Cole and Staff; M-109, U. H.

9:00 - 10:00 Infertility Conference; Louis L. Friedman, David I. Seibel, and Obstetrics Staff; Station 54.

9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater.

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; Heart Hospital Amphitheater.

9:15 - 10:00 Surgery-Roentgenology Conference; L. G. Rigler, J. Friedman, Owen H. Wangensteen and Staff; Todd Amphitheater, U. H.

10:00 - 11:30 Surgery Conference; Todd Amphitheater, U. H.

10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U. H.

11:30 - Anatomy Seminar; A Study of the Birefringence of Various Mammalian Striated Muscles; Joseph Wethington; 226 Institute of Anatomy.

Ancker Hospital

8:30 - 9:30 Surgery Conference; Auditorium.

Minneapolis General Hospital

11:00 - 12:00 Medical - X-ray Conference; O. Lipschultz, Thomas Lowry, and Staff; Main Classroom.

Veterans Administration Hospital

8:00 - Proctology Rounds; W. C. Bernstein and Staff; Bldg. III.

8:30 - 11:15 Hematology Rounds; Drs. Hagen, Goldish, and Aufderheide.
11:15 - 12:00 Morphology . . . . . Dr. Aufderheide.