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The Surgical Treatment of Congenital Heart Disease

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I. THE SURGICAL TREATMENT OF CONGENITAL HEART DISEASE -- Analysis of the Results in 388 Cases*

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This report is a summary of the frustrations, failures, and accomplishments in the surgical management of all of the congenital cardiac disease thus treated at University Hospitals during the 13 years from September 1, 1939 to October 1, 1952. (Table I)

Any discussion of surgical experiences with these problems at the University of Minnesota Hospitals may appropriately begin by the specific reference to the contributions of Drs. Owen H. Wangensteen and John R. Paine. Dr. Wangensteen initiated surgical interest locally by the successful ligation of a patent ductus arteriosus in this hospital Sept. 1, 1939. The slowness with which momentum in any new field is generated is indicated by the fact that during the next four years only 16 operations were performed for congenital heart disease. Dr. John Paine, upon his return from the armed forces in 1945, guided the expansion of these operations into the field of pulmonic stenosis. The thoughts and ideas of these men have been written into the final record. Moreover, these results represent the aggregate contributions of 12 different staff surgeons in this department who have performed varying numbers of the operations herein enumerated. In addition, a far greater number of surgery fellows and internes have rendered valuable assistance in these efforts.

Etiology:

Congenital anomalies of the heart and great vessels are due to arrested or defective prenatal development. But it is uncertain whether intrinsic factors in the germ plasm or extrinsic factors in the embryo's environment are of major importance. The association of virus and other infections in the early months of pregnancy with a significant incidence of cardiac anomalies has been recorded[1] and is frequently noted in the histories of our patients. This observation together with recent animal experiments suggesting the importance of such factors as vitamin deficiencies,[2] brief periods of anoxia,[3] and cortisone[4] offer hope for future progress in the prevention of the occurrence of these defects. In this respect, a likely productive approach for investigation would seem to be the peculiar sex incidence of many of these defects. Thus, coarctation of the aorta (28 males to 5 females in our series) and patent ductus arteriosus (147 females to 48 males in our series) are examples of sex preponderances which can scarcely be coincidental.

Incidence:

The true incidence of congenital cardiovascular disease is not known and is difficult to ascertain. However, the attitude that these lesions are rare should be dispelled from the minds of all physicians for two reasons; first, because congenital cardiac disease is not particularly unusual and experience has repeatedly demonstrated that the more we learn about these lesions the more frequently do we find them; secondly, because such an attitude fixed in the mind of the general practitioner precludes the early diagnosis of these cases in his office where it must of necessity be made. Statistics now available from large series of unselected autopsies (34,023 in Boston,[5] 30,036 in Minnesota[6] indicate that congenital cardiac lesions are responsible for about 1% of all deaths.

However, an even more impressive indication of the scope of this problem has been the findings in a statewide survey of Colorado School Children[7]. In that state, there was found by use of only cursory screening techniques 2.7 cases of congenital heart disease per

* Supported by Research Grants from Minnesota Heart Association and the U. S. Public Health Service.

TABLE I

CLASSIFICATION AND MORTALITY OF CONGENITAL CARDIAC LESIONS
TREATED BY SURGERY AT UNIVERSITY HOSPITALS
September 1, 1939 to October 1, 1952

CONDITION	NO. PATIENTS			OPERATIVE DEATHS	LATE* DEATHS
I. EXTRACARDIAC					
1. Patent Ductus Arteriosus			195	6 (3%)	0
2. Aortic Pulmonary Window			2	1	
3. Coarctation of Aorta			33	3 (9%)	1 (3%)
II. INTRACARDIAC					
A. Pulmonary Stenosis			143		
1. Valvular Type Treated by Valvulotomy		44			
a. Isolated Pulmonary ("Pure")	33			2 (6%)	0
(1) Without Interatrial Defect	15				
(2) With " "	18				
b. With Other Defects	11			4	0
(1) Tetralogy of Fallot	8				
(2) Pentalogy of Fallot	3				
2. Cases of Pulmonic Stenosis Treated by Systemic Pulmonary Artery Anastomosis (Blalock Procedure)		99		9 (9%)	6 (6%)
B. Complete Transposition of Great Vessels			4	2	0
C. High Interventricular Septal Defect (Eisenmenger)			5	4	0
D. Interatrial Septal Defects			3	2	0
E. Miscellaneous (Truncus Arteriosus)			3	1	0
TOTALS			388	34 (9%)	7 (2%)

* Includes all deaths following discharge from the hospital regardless of the postoperative interval.

1000 sixth grade children and 15.2 cases per 1000 in preschool age children. Since many congenital heart cases do not reach school age, much less the sixth grade; the incidence obtained in the preschool age group is no doubt closer to the true incidence. In the survey in question, there was probably some element of selection in the higher incidence observed in this preschool age group. This factor of selection, however, is in part offset by the fact that admittedly many acyanotic types of congenital heart disease could be missed in such a survey type of physical examination. At any rate, on the basis of these figures, it is possible to make a rough computation that there are born in the United States* each year from 25,000 to 50,000 children with congenital heart defects. Most of these children are potential candidates for corrective surgery. Thus, it is quite likely that more cases of congenital heart disease are being born each year than have ever been operated upon in all of the surgical clinics of this country since 1939. Therefore, we believe that every doctor's office must become a detection center for these defects if we are to make any inroads into this tremendous backlog from other years of unrecognized and untreated cases. Further, it is perhaps obvious that the group of defects most difficult to diagnose (acyanotic) are the most amenable to surgery that is completely curative by present techniques.

PATENT DUCTUS ARTERIOSUS:

The contributions of Gross[8,9] in this field are well known. Less generally recognized is the earlier attempt of Graybiel, Strieder, and Boyer[10] to interrupt the ductus in a 30 year old woman with bacterial endocarditis--a type of case often laden with disaster even with present knowledge and experience. Moreover, in this regard one cannot help but admire the prescience of Dr. John C. Munro[11] who on May 6, 1907 in a paper before the Philadelphia Academy of Surgery proposed ligation of patent ductus arteriosus. He described lucidly both his observations extending over the preceding 19 years, outlined a surgical technique based on dissections made on cadavers of newborn infants; and in addition noted the diagnostic features of patent ductus and aortic pulmonary septal defects, besides suggesting the feasibility of ligation of the shunt in order to increase the patient's life expectancy. So far as can be ascertained, there is no record of his ever having attempted the operation upon the living individual.

Physiology:

The patent ductus arteriosus is a vasuclar shunt between the aortic arch and the pulmonary artery which serves its function during fetal life by shunting blood from the pulmonary artery directly into the aortic thus by-passing the lungs. Coincident with the expansion of the lungs at birth, the peripheral resistance in the pulmonary circuit falls to the extent that the pulmonary arterial pressure likewise falls below that of the aorta and the direction of the shunt is reversed unless closure of the ductus takes place as it customarily does by the age of 3 months.

Physiologically a patent ductus is of the utmost importance because from 1/4 to 3/4 of the total output of the left ventricle may be lost through the ductal shunt into the pulmonary artery. The consequences of this arteriovenous shunt would be even more disasterous to life expectancy were it not for the fact that the interposition of the pulmonary capillary resistance between the shunt and the heart offers some succor to the latter. However, the constant handicap that this ever present parasite imposes on the heart can easily be appreciated by its documented effects upon life expectancy and growth as indicated below.

Consequences:

The hazards of a patent ductus arteriosus come under two general headings: Poor physical development, and shortened life expectancy. This latter being due

* 3,500,000 (1950) live births per year

to the frequent occurrence in these patients of bacterial endocarditis, congestive heart failure, rupture of ductus, or chronic pulmonary infection.

Adams[12] has analyzed this series of patients and has demonstrated the deleterious effects of a patent ductus upon body development and growth. Moreover, he found that in instances in which surgery was performed late in childhood, loss in physique was frequently not regained.

Many confirmatory surveys[13,14] have been reported since M. Abbott[15] first emphasized the shortened life expectancy associated with left to right shunts. In her series of 92 primary or uncomplicated cases of patent ductus arteriosus, 23% died of bacterial endocarditis and 43% from heart failure. The average age at death in her series was 24 years. Keys and Shapiro[14] found that the average age at death was 39 years for men and 36 years for women.

Experimentally in dogs, the profound increase in susceptibility of the heart valves to infection engendered by left to right shunts has been demonstrated[16]. These effects are thought to depend in part upon the chemical and endocrine changes secondary to the large shunts. The mechanisms demonstrated in these experiments provide an adequate basis for the explanation of this increased susceptibility to infection and further corroborate the clinical observations of the extremely rapid clearing (within ten minutes in one recorded instance[17]) of the blood stream of a persistent septicemia once the abnormal shunt is interrupted.

Surgical Technique:

The patent ductus arteriosus is an abnormally thin-walled conduit for a large volume of blood and one whose walls are not nearly as tough as the fibrous layer of pericardium from which it must be separated. This circumstance together with its location deep in the chest between the aorta and pulmonary artery constitutes an adequate implication of the hazards that may beset the operator.

The preferred operation in this clinic at present is division of the ductus between clamps oversewing the divided ends with fine silk, (6-0) and the placement of a proximal (3-0) stick tie which is tied as the clamps are removed as described by Wangensteen[18]. Potts[19] has introduced a special clamp which greatly facilitates the operation of division.

The location of the incision has undergone variations with time. The majority of the patients in this series have been operated upon through an incision in the second or third interspace anteriorly beginning just to the left of the sternum. However, our present preference is for a lateral or posterolateral approach through the fourth interspace by removal of the fourth rib. Catheter drainage of the chest is instituted routinely.

Ligation or Division:

The operative therapy of patent ductus arteriosus has undergone a steady evolution since the original description of simple ligation. Gross[9] early expressed dissatisfaction with simple or multiple ligature of the ductus due to the fact that recanalization or incomplete obliteration occurred in about 20% of his first 43 cases; thus, he was persuaded to divide the ductus routinely. Our own experience in this clinic during the period when ligation was done routinely (Sept. 1939 - Nov. 1943) is in accord with this suggestion. Among 16 patients in which simple ligature of the ductus was done, recanalization occurred in two. The universally recognized difficulties and hazards associated with reoperation after recanalization makes the operation of division mandatory wherever possible in our opinion.

An early case in our ligated series ( ) illustrates one of the additional disadvantages of ligation procedures. In this patient, cardiac failure occurred nine years following an apparently

successful ligation of his patent ductus. It was then presumed that the ductus had recanalized in this patient and exploration was again carried out. No ductus was found, and at autopsy 9 days later a high interventricular septal defect with pulmonary sclerosis was the only defect present. (Eisenmenger Syndrome)

Age and Limitation in Exercise Tolerance:

The age of our patients varied from five months to 48 years. One hundred and seven (54%) of these cases had definite symptoms and signs of limitations in their exercise tolerance preoperatively.

Murmur:

This is the classical sign for diagnosis, and consists of continuous systolic and diastolic components heard with greatest intensity to the left of the sternum in the second and third interspaces. This characteristic machinery type murmur was present in 190 of our 197 proved cases (96%). In the seven remaining patients the murmur was atypical in that there was only a harsh systolic component. This finding of an atypical murmur is somewhat more common in the younger age group particularly under one year.

Postoperative Murmurs:

These are of importance because of their frequency. The characteristic murmur should disappear immediately following surgery.

A soft systolic murmur in the pulmonic area to the left of the sternum, was present postoperatively in 23 of 159 (14%) cases in which there were adequate follow up examinations. In these 23 cases, the character of the murmur, its location, and the absence of other signs of organic heart disease suggested a functional origin.

An additional 24 patients (14%) in the adequatley followed group of 159 had a harsh systolic murmur present following surgery. The systolic murmur in this group of patients although quite variable in location was usually grade 2 to 4 and was thought to be organic. In most such instances it was thought to be due to such other associated anomalies as interventricular septal defects, pulmonary stenosis, subaortic stenosis, or interatrial septal defects. In many of these cases the presence of this additional defect had been suspected preoperatively, and in a few demonstrated preoperatively by cardiac catheterization or at operation by the persistence of a systolic thrill over the heart after division of the ductus. Earlier in our experience, we undoubtedly missed some cases of pulmonary valvular stenosis associated with a patent ductus, since in the past year two such patients have been detected and both defects treated successfully at the same operation.

Endocarditis:

Finally, there is the problem of the ductus with superimposed bacterial endocarditis. Touroff[20] demonstrated that simple interruption of the ducts without antibacterial therapy resulted in cure on some occasions. Such an outcome, however, is not universally achieved and the preferred contemporary treatment is a period of preoperative treatment of 4 to 6 weeks with heavy dosages of antibiotics followed by surgical division and then continuance of antibiotic therapy for another two to three weeks.

Thirteen cases in this series had active bacterial endocarditis prior to surgery. One patient also had active glomerulonephritis. In the cases with bacterial endocarditis, the dissection is considerably more hazardous due to the periductal inflammation. In our series, 10 of the patients with endocarditis were dealt with satisfactorily. In the other three, hemorrhage occurred which terminated fatally.

At least six additional patients had good evidence of having had rheumatic fever prior to surgery and two others had had probable attacks. The fact that none of these patients have had any recurrent attacks after therapy seems significant in this regard.

Results:

During the period of this study, 203 patients* were subjected to thoracotomy with the preoperative diagnosis of a patent ductus arteriosus as the primary defect. In 197 (97%) of these the diagnosis was correct in that a patent ductus arteriosus was found and either ligated in continuity or divided in 195 patients with 6 deaths, an operative mortality of 3.0% for the entire group. (Table II) Three of these six deaths occurred in the first 16 patients and three in the last 179 patients (1.6% mortality), indicating that with the present knowledge and techniques, this operation is a safe one. Gross[9] has reported 3 deaths in 43 patients with ligation and 4 deaths in 180 divisions for a total operative mortality of 3.1%.

TABLE II

PATENT DUCTUS ARTERIOSUS--THERAPY AND MORTALITY
(197 Consecutive Cases)

Type of Treatment	Number of Patients	Deaths	Recurrences
Routine ligation (1939-1943)	16	3*	2
Routine division (1943-1952)	169	3	0
Cases ligated (1943-1952) for technical reasons	10	0	1
Thoracotomy only (due to cardiac arrest)	2	0	
TOTALS	197	6 (3.0%)	3

*One death occurred at second operation in a recanalized case with bacterial endocarditis.

Two patients in our series had both pulmonary valvulotomy and division of a patent ductus arteriosus performed at the same operation without incident. There were two patients in whom the operation was not completed. Cardiac arrest occurred twice in the first patient shortly after the chest was opened, and in the second patient recurring severe episodes of bradycardia and cardiac irregularity during the dissection of the ductus forced abandonment of the procedure. Both patients recovered, but neither has been operated upon again.

The 97% accuracy in diagnosis of this condition is a tribute to the acumen of our associates in the Department of Pediatrics and Medicine. Of the six

* does not include the patients with coarctation of aorta and an associated patent ductus.

patients who did not have a patent ductus, two had aortic pulmonary defects, (both catheterized preoperatively) two had high interventricular septal defects, (Eisenmenger type, both catheterized preoperatively), one had a ruptured sinus of Valsalva due to subacute bacterial endocarditis, and one patient is still undiagnosed. All of these defects simulate the physiologic defects of a patent ductus very closely as is indicated by the fact that preoperative cardiac catheterization failed to give an absolute diagnosis in three of these six patients.

The ideal time for operation in patent ductus arteriosus deserves some mention. If the diagnosis can be accurately made in infancy, operation should be advised at that time since serious complications may and do develop frequently during periods of delay for observation. Such periods of procrastination until the patient becomes somewhat older or until obvious complications occur may not be the wisest course in view of the low mortality and morbidity now associated with operative correction. Further, operation is easier and safer in the younger age groups. Mortality and morbidity statistics will always be lower in operations done during childhood because cardiac reserve has not been depleted and in children the ductus is technically easier to dissect due to a relatively greater length and elasticity. Operations performed in children avoid the risks of stunted growth, cardiac failure, bacterial endocarditis, or sclerotic changes[21] in the pulmonary circulation, all of which render the operation more hazardous and the end results less certain.

Therefore, in the absence of compelling contraindications, the diagnosis of patent ductus arteriosus, we believe, should be followed promptly by operative correction.

AORTIC-PULMONARY SEPTAL DEFECTS:

The basic circulatory fault in aortic-pulmonary fistula, or window as it is sometimes called, is essentially the same as in a patent ductus arteriosus; and the physical findings usually closely simulate the latter disease although invariably are of a more severe degree. Developmentally, an aortic-pulmonary fistula is the result of incomplete separation of the truncus arteriosus into the aorta and pulmonary artery leaving a fistulous communication between these vessels usually beginning a few millimeters distal to the valves.

Previously this defect was thought to be extremely rare since only 13 cases has been reported in the literature up until 1950[22] and 10 of these were in Maude Abbott's collection.[15]

However, in our relatively small experience, we have encountered 7 of these lesions. In two of these patients a determined attempt has been made (R.L.V.) to close the abnormal fistula by multiple ligations with one success (., age 7) and one failure due to hemorrhage. Gross[23] has reported one successful ligation (also the first) and Scott[24] recently described the initial successful division of this short treacherous shunt.

COARCTATION OF THE AORTA

Coarctation of the aorta is generally classified in two groups, the so-called "infantile" type and the "adult" types. These terms are somewhat confusing because the adult type occurs in infants and the infantile type may be found in adults. In the former or "infantile type" the constricted aortic segment usually diffusely involves the entire region of the fetal isthmus (from the origin of the left subclavian artery to the site of insertion of the ductus arteriosus). In addition, this defect is commonly accompanied by other major congenital cardiac anomalies which preclude the infant surviving more than days or weeks.

The "adult" type occurs as a more focal area of constriction, usually 1 cm. or less in length, most frequently at or just below (rarely above) the

insertion of the ligamentum arteriosum. Frequently associated anomalies in these cases are the finding of patent ductus arteriosus (24% is this series) or bicuspid aortic valves. However, the coarctation is often the major sole anomaly and the effect upon the heart and circulation is the result of the aortic obstruction.

Reifenstein, Levine, and Gross[25] have reviewed a series of 104 autopsied cases of moderate, extreme, or complete degrees of the "adult" type of coarctation of the aorta in patients two years of age or older, which have been reported since Abbott's review in 1928. They found that although coarctation occasionally was compatible with long life, 25% of patients with this condition died before their 20th birthday and that 61% of patients died before or during their fortieth year of life of complications of the aortic obstruction. The average age at death was only 35.0 years. These death-dealing complications are approximately equally divided between bacterial endocarditis, rupture of the aorta, cardiac failure, and cerebral hemorrhage subsequent to rupture of an aneurysm in the Circle of Willis.

Our own experience has done nothing to dispel this grim prognosis for our first two patients died suddenly in the hospital while awaiting surgery. Cerebrovascular accidents accounted for both deaths.

Diagnosis:

Diagnosis in most cases can be easily made by physical examination alone provided there is an awareness of the existence of this condition. The blood pressure in the arms is elevated above normal and the femoral and abdominal pulsations are faint or absent. Collateral arterial anastamoses (with rib erosion by X-ray) may be seen. In contrast to the patients with a patent ductus, physical development is often better than average probably as a consequence of the collateral arterial circulation through the muscles of the trunk. Conceivably, the augmented perfusion of the pituitary gland could also account for some measure of this sturdy musculoskeletal growth so commonly noted.

The X-ray finding of notched ribs in the older individuals is strongly suggestive of coarctation. It is regretable that frequently this sign is absent. The cardiac silhouette usually exhibits evidence of left ventricular hypertrophy together with a lack of prominence of the aortic knob and an increased prominence along the left sternal border ascribed to enlargement of the left subclavian arterial segment.

The diagnosis in infants is worthy of special mention because the only findings may be evidence of cardiac decompensation and an inconsequential murmur or even normal heart sounds. The blood pressure in the arms may be normal. In such cases the diagnosis can be suspected by the impalpable femoral pulsations. In the past such infants have invariably died without the diagnosis being made or an attempt has been made to tide these children over by hospitalization, digitalization, and other measures because it was felt that such children were too young to operate upon. After losing two infants with a completely correctable coarctation during such periods of procrastination, we are of the opinion that once an infant has exhibited the signs and symptoms of cardiac decompensation due to an aortic block, active measures should be instituted to regain cardiac compensation and the child then operated upon without delay. Our youngest case ( ) operated upon at the age of 43 days illustrates this concept. This infant had diffuse cardiac enlargement filling almost the entire thorax not typical of any particular lesion; murmurs and cyanosis were absent. The absence of femoral pulses together with some elevation of the blood pressure in the arms (140/90) were the diagnostic findings. The child was extremely lethargic, dyspneic, and appeared to be in very bad condition at the time surgery was undertaken in spite of digitalization and other measures. A large patent ductus with a coarctation proximal was found. The ductus was

divided and the coarctated segment of aorta resected with end to end anastomoses made. There was an immediate and remarkable change in the child's condition postoperatively, and the child now appears normal. It is possible, but not altogether certain, that operation done at such a young age may even require a second resection in later years because of relative growth differential at the anastomatic site. Even if this be true, the risks of two procedures seems justified in tiny tots desperately ill before the initial procedure. In this regard, it should be emphasized that the cross sectional area of the aorta must be reduced by more than 75% before any signs or symptoms of obstruction will develop.[26]

Results:

In the period ending October 1, 1952, 35 patients have been operated upon with a diagnosis of coarctation of the aorta. Two patients merit special mention. In the first patient (a man of 31 years), there was no coarctation found but a severe acquired atherosclerosis of the entire thoracic and abdominal aorta with a complete thrombotic occlusion of the abdominal aorta at its bifurcation was present. In the other case (., age 11) there was a mild degree of a coarctation present but it appeared of secondary importance to the primary lesion which was obviously an Eisenmenger complex with a tremendously enlarged pulmonary artery and pulmonary hypertension. In these two cases only exploration was done and both died postoperatively of cardiac failure. In the remaining 33 patients, the operative mortality was 9% (3 deaths).

Twenty-six patients had resection of the coarctated area with end to end anastomosis of the aorta. One operative death occurred in this group in a patient who developed a septicemia and aortic aneurysm at the point of application of the proximal aortic clamp. At autopsy in this patient, the anastomotic line was well healed, but the aneurysm had ruptured two months postoperatively.

Three patients had resection of the coarcted area with restoration of continuity by an end to end left subclavian-aortic anastomosis. There were no deaths in this group.

In one patient, once the obliterated ductus was cut the tented —up aorta assumed a normal configuration. Clamps were applied while the interior of the aorta was explored for a diaphragm, but none was found. Simple closure of the aortic incision was done and this patient has had a good result.

The second death occurred in the oldest patient in the group (age 39 with severe preoperative angina) who suffered a fatal coronary occlusion with ventricular fibrillation during induction of the anesthesia.

The third death was in a patient who died of cardiac arrest during the dissection preliminary to resection. Blood pressure cuffs had been applied to both arms of the patient and during the dissection both were inflated at the same time. Immediately the heart went into an extremely dilated state and stopped. The heart was so tight against the pericardium that it was difficult to open the latter for massage. The difficulty was recognized almost immediately, but it was impossible to restart this acutely dilated heart. At autopsy, a complete coarctation was present making it apparent that at the moment both blood pressure cuffs were inflated, the entire cardiac output had to be received into the two carotid arteries, an intolerable obstacle for this small child.

No arterial grafts were used, no patients were refused exploration, nor were any patients explored and not resected because of the length or character of the aortic constriction.

Gross[27] has reported an operative mortality of 11% for his first 100 cases of coarctation treated surgically.

Postoperative Results:

Blood Pressure:

The effects upon blood pressure have been gratifying. Preoperatively, only 13% of these patients had a normal blood pressure whereas postoperatively 83% are apparently cured of their hypertension and an additional 17% have achieved some reduction in blood pressure. These results are summarized in relation to the type of anastomosis in Table III. It will be noted that the reduction in blood pressure to normal levels has been best with an end-to-end anastomosis of

TABLE III

COARCTATION OF AORTA

TYPES OF OPERATION
AND RESPECTIVE RESULTS IN SURVIVING PATIENTS

Type of Operation	No Relief of Hypertension	Fair* Relief of Hypertension	Cure of Hypertension	Total Number of Cases
Excision of coarctation of aorta; subclavian to aorta anastomosis.		3		3
Division of Ligamentum Arteriosum only.			1	1
Excision of coarctation, with end-to-end anastomosis of aorta.		2	24	26

* Systolic still above 140 but diastolic below 90 mm.Hg.

the aorta following the resection of the obstructing segment. Thus, this operation is the one of choice whenever possible. Even though the reduction in blood pressure has not been as complete following a subclavian-aorta anastomosis, the subjective relief of symptoms has been just as real; and we favor this procedure in preference to homologous aortic grafts because of the fact that the latter, as has been amply demonstrated, do not survive. We fear that such grafts put into young people with life expectancies approaching 50 years may ultimately degenerate with arteriosclerosis, thrombosis, or aneurysm formation. However, these objections are not compelling enough to preclude the use of homologous grafts when no other method is feasible.

Femoral Pulses:

In all 30 surviving patients, there has been an immediate return of the femoral pulses with subjective relief of claudication and muscle cramps in all of the cases in which those symptoms were present.

Other Complications:

Coronary Occlusion:

One patient (, U.H., age 31) developed severe pain in chest, hypotension, and electrocardiographic changes considered diagnostic of coronary occlusion a few hours following the conclusion of surgery. Fearful of the effect of hypotension upon the volume of coronary blood flow, we immediately instituted a continuous intravenous drip of arterenol with almost immediate relief of the chest pain although the electrocardiographic

changes persisted for some days. We believe that this type of treatment for coronary occlusion, which was successful in this case, merits more trials.

In older patients with considerable hypertension and evidence of coronary sclerosis preoperatively, we now believe it desirable to administer enough arterenol prophylactically in the postoperative interval in order to maintain the hypertension for a few days; and then gradually adjust the arterenol drip to allow the pressure to fall to its own baseline in order to prevent the development of acute coronary insufficiency.

There has been one late death (., age 34) in this group of thirty patients who survived the operative correction of their coarctation. This man had a resection of his coarctation with end-to-end anastomosis performed on 9/15/50. Preoperatively his arm blood pressure was 150/118. On February 1951, he was seen in the clinic and was noted to have had a considerable improvement postoperatively. His blood pressure on that date was 146/88. On April 13, 1951 (7 months postoperatively) he dropped dead when told his father had died. Autopsy disclosed a well-healed aortic anastomosis, and severe coronary atherosclerosis with a fresh occlusion as the cause of death. Ironically, the father had not died but had a cerebral accident from which he recovered. This early and severe degree of sclerosis of the coronary circulation frequently seen in patients with coarctation deserves further investigation.

PULMONIC STENOSIS

The effective surgical management of patients with certain types of cyanotic heart disease dates from that technique first successfully managed and described by Blalock and Taussig in 1945[28]. Their imaginative venture, wherein the total pulmonary blood flow is augmented through the construction of a subclavian-pulmonary artery anastomosis, promptly won medical and lay recognition for the clear-cut relief from exertional dyspnea afforded to these severely crippled individuals. Coincidentally in these successful cases, the convalescent lost much of the cyanotic suffusion of the visible mucous membranes, as well as the plethoric congested capillary networks characteristically contributing to the "blue baby's" facial features. In 1946 Potts et al[29] reported a technique, and the surgical instruments necessary for its performance, which was both ingenious and helpful in these newborn and other tiny-sized tots critically afflicted by certain cardiac lesions with pulmonary arterial insufficiency and with cyanosis. Their operation was particularly applicable to children with systemic vessels too small for the usual type of anastomosis. In their procedure a short slit in the aorta was joined side to side with the pulmonary artery, and also effectively increased the pulmonary artery flow. It has the disadvantage of sometimes increasing the heart strain unduly on the left ventricle. The most recent and the next significant landmark in the surgical history of this condition was created by Sellors[30] and by Brock[31]. To them, after studying pathological specimens with the tetrad of Fallot, it seemed easier to incise the diaphragm-like barrier often existing in the pulmonary artery valvular area. They attacked directly, therefore, this unnatural barrier to normal blood flow out of the heart and to the lungs. Once an adequate lumen had been created, the patient was better clinically because of the increased volume delivered to the lungs; too, theoretically his outlook was superior to that possible in the patient with a surgically created ductus of the Blalock or Potts type, since the overall cardiac workload would seem to be less. Unfortunately, for scientific publications, most papers dealing with the surgical management of the various kinds of congenital heart defects associated with cyanosis, have inclined to lump the treatment of many lesions together under the rather broad, and at times quite inaccurate, approximate title of "Surgery for Pulmonic Stenosis." This term is admittedly an awkward one to work with in a paper, yet recourse to more precisely descriptive nomenclature leaves a complex wordy residue hardly less confusing to

the reader. There appears to be no handy, simplified correct fashion for titling this particular problem and hence much of the literature, which is now already abundant and will not be reviewed, can be found listed under that phrase. This aggregate of abnormal cardiac states consists of obstruction to right ventricular inflow at the tricuspid area (atresia or stenosis), or to outflow (infundibular or valvular stenosis, or pulmonary arterial atresia). In addition, these individual sites of interference with free flow may have associated septal defects of ventricle and/or atrium. Although certain rarer combinations of these forms have been recognized at this clinic, the preponderance of cases consists of patients with the characteristics of Fallot's tetralogy (infundibular and/or pulmonary valvular stenosis plus an interventricular septal defect with overriding of the aorta). The next largest fraction is made up of individuals believed to have pulmonary valvular stenosis with or without a functionally patent interatrial septum. Historically, this group has been dissociated from those with the tetralogy of defects, and the term "pure" or isolated pulmonary stenosis used to characterize it. In reality, about one-half to three-fourths of these cases of valvular pulmonic stenosis with an intact ventricular septum have an identifiable interatrial septal defect.

The frequency of pulmonic stenosis among all congenital cardiac problems is an almost indeterminable statistic, since any one clinic's experience is quite reasonably a reflection of the magnitude of the drawing power generated by the special surgical contributions of those persons working therein. It might be of interest to note that roughly equivalent members of cases were seen and operated upon in our clinic in both the patent ductus group and in those with pulmonic stenosis during that period between the first Blalock procedure (1945) and the present. This value is in agreement with Maude Abbott's proportions for 1,000 collected cases of congenital malformations of the heart[15]. In her group of collected cases there were 110 patients with various types of pulmonic stenosis as compared to 92 patients with patent ductus arteriosus, and 70 with aortic coarctation.

On the basis of our observations, it now seems reasonable to make certain inferences which are somewhat at variance with earlier teachings and writings. First, pulmonic stenosis, in some variant, is not a rare congenital cardiac condition. It is at least as frequent as patent ductus arteriosus. Secondly, among individuals with pulmonic stenosis and an interventricular septal defect (Fallot's tetrad) a significant degree of pulmonary valvular stenosis is present in at least 1/3 of the cases. Among the more current papers, Brock[32], Sellors[33] and Brown[34], with much larger series of comparable type cases carefully studied for this relationship, offer support for this belief. Third, pulmonary valvular stenosis with an intact ventricular septum is not nearly the uncommon lesion it was once considered to be. Approximately one-fourth (33/143) of all the patients with some form of pulmonic stenosis in our series had no demonstrable interventricular septal defect.

SURGICAL MANAGEMENT:

The clinical management of patients with pulmonic stenosis has undergone much change since the first hesitant gropings in 1946 by our diagnosticians and surgeons. It is to the credit of the former (pediatrician and internist) that their errors have been substantially less consequential and less frequent than those of the operating teams. All, however, have gained confidence and more frequent successes by the empiricism of trial and error. Currently, most individuals suspected of having some type of pulmonic stenosis undergo cardiac catheterization and often angiocardiography in addition to history and physical examination and the standardized techniques of electrocardiography, fluoroscopy, roentgenography, and circulation times.

Tetralogy of Fallot:

Those cases diagnosed as having pulmonic stenosis and a ventricular septal defect (Fallot's tetrad) are considered candidates for early surgery only if there is definite evidence of deterioration. This state may be represented either by recurrent bouts of heart failure, or decompensation of the pulmonary collateral channels (fainting episodes, extremes of dyspnea on slight effort), or a sustained rate of increase in the hemoglobin, red blood cell, or hematocrit values.* We feel that the diagnosis of tetralogy of Fallot need not signify early surgical treatment unless one or more of these disturbing developments appear. This withholding of surgical palliation in the cases with tetralogy does not deny the knowledge of the startlingly high death rate in this group without surgical treatment. In this respect, Campbell[35] has pointed out that untreated, only one-half of the tetrads will live to age seven, four out of five will have succumbed by age 14, and but one in ten ever lives to age 21. In the face of this grim prognosis the reasons that we have tended to withhold operative treatment from those living reasonably well with their ailment are twofold. First, we are in firm belief that completely curative cardioplastic procedures will be available within the next few years or sooner. A number of times at the autopsy table we have, under direct vision through an incision in the outflow tract of the right ventricle, completely converted a heart with the defects of a tetrad into an anatomically normal one by two or three appropriately placed stitches. To meddle for small gains in exercise tolerance and cosmetic improvement, if the child is tolerably well off, we feel is unwise at this stage. Therefore on the basis of this reasoning, now, when relief does become necessary in any given child we are inclined to favor the anastomosis of a systemic vessel to the pulmonary artery so as to disturb least those structures within the pericardium unto that time when definitive and corrective operations on the heart are at hand. However, in those cases of tetrad with a valvular type pulmonic stenosis (11 cases in our series) we have usually preferred to do a valvulotomy, rather than an anastomosis, considering it the first stage of a completely corrective plan. Unless therefore a shunt operation or a valvulotomy is required in order to avert some cardiovascular disaster, we recommend continued careful observation and follow-up.

* Often precursors of cerebral infarcts and abscesses

Pulmonic Stenosis With Intact Ventricular Septum:

For those with pulmonic stenosis and an intact ventricular septum although the diagnosis may be quite clear-cut, the true identity is regularly sought by means of cardiac catheterization often supplemented by angiocardiography. The direct measurement via the cardiac catheter of right ventricular (and pulmonary artery whenever possible) pressures as well as the detection of any intracardiac shunts contributes valuably to the appraisal of a need for surgery.

Much information remains to be accumulated in the documentation of the fate of these persons with pulmonic stenosis and an intact ventricular septum. Maude Abbott[15] has suggested that the life expectancy is 22 years for those without an associated interatrial defect, and 18 years for those with this additional anomaly. Greene[36] has confirmed these approximate values; wherein for 63 cases the life expectancy was 26 years. For those cases with minimal degrees of pulmonary valvular stenosis as affirmed by the absence of right strain pattern on the electrocardiogram and with right ventricular pressures under 60 mm. of mercury, the need for valvulotomy seems quite questionable. Our inability to obtain consistently an intraventricular tension much lower than the latter figure after valvulotomy, as measured by cardiac catheterization at periods up to one year postoperatively, buttresses this nonoperative viewpoint. For those with definitely elevated right ventricular pressures

(above 60 mm. Hg.), abnormal ECG patterns, moderate to very severe dyspnea on exertion, varying degrees of cyanosis (related to a shunt through an interatrial septal defect), repeated bouts of right heart failure; pulmonary valvulotomy via the right ventricle offers a safe operation with an almost certain hope of nearly complete subjective relief. This procedure is virtually corrective surgery. There have been but two deaths in the 33 consecutive cases of this type operated upon to date.

Results of Valvulotomy for Isolated Pulmonic Stenosis:

In general the results following valvulotomy have been very good (Table IV). Most of the patients have been operated upon too recently to predict long term results, but the immediate improvement has been gratifying. Almost one-half of the patients have had a result classified as excellent. The most striking of these various improvements have been the reduction in dyspnea and the increase in exercise tolerance.

TABLE IV

RESULTS AND MORTALITY OF VALVULOTOMY
IN CASES WITH ISOLATED PULMONARY STENOSIS

	Isolated without Interatrial	Isolated with Interatrial	Total
Number of Patients	15(a)	18(b)	33
Results			
Excellent*	6	10	16 (48%)
Improved	7	7	14 (43%)
Unimproved but living	0	1	1 (3%)
Died	2	0	2 (6%)

(a) One patient also had an associated patent ductus divided and another had an associated aortic-pulmonary window which was not treated.

(b) One patient had an associated patent ductus divided.

* Excellent=no cyanosis and tolerance for strenuous exercise indistinguishable from normal.

An example of this was patient S.K., a 9 year old non-cyanotic boy with isolated valvular pulmonary stenosis, who before surgery was unable to walk more than one to two blocks without having to rest. Following a valvulotomy under direct vision, the boy's exercise tolerance increased remarkably to the extent that he was able to perform as a normal child to the casual observer, this included swimming, bike riding, and gymnasium exercises. Preoperatively the pressure in his right ventricle was 260/0 mm. of Hg., and in the pulmonary artery was 12/10 mm. of Hg.; and his cardiac output was 1 liter per minute. Two weeks postoperatively, the right ventricular pressure had dropped to 60/0 mm. of Hg., the pulmonary artery pressure was 14/8 mm. of Hg. and the cardiac output at rest

had increased to 2.1 liters per minute.

In addition to the marked reduction in dyspnea, in most patients there was usually a moderate to marked increase in the exercise tolerance, greater than that seen in patients who had had a Blalock type of procedure for Tetralogy of Fallot. Those patients who were cyanotic due to a coexistant interatrial communication experienced after valvulotomy either a disappearance or a reduction in their cyanosis with a concomitant decrease or disappearance of clubbing and "watch crystal" formation of the digits.

A finding of some interest and perhaps of significance in helping to differentiate these cases of pulmonic stenosis with intact ventricular septum from those with a septal defect is the finding that if the right ventricular pressure approximates or exceeds that in the systemic arteries, the ventricualr septum is likely to be intact. On the other hand, in those cases with a ventricular septal defect, the right ventricular pressure is considerably lower (rarely above 80 mm. Hg. systolic).

Results of Valvulotomy for Tetralogy of Fallot:

The results and mortality for this group is summarized in Table V. Analysis of the four deaths occurring in this group has emphasized to us the importance of careful selection for this procedure of those cases in which the obstructive component is primarily valvular and not infundibular.

TABLE V

RESULTS AND MORTALITY OF VALVULOTOMY IN PATIENTS WITH TETRALOGY

Number of Patients	11
Results	
Improved	7 (64%)
Unimproved but living	0
Died	4*(36%)

* Includes all deaths early and late.

In those cases which are improved by valvulotomy, the benefits are as substantial as those seen following the Blalock procedure.

Technique for Valvulotomy:

Five of the 44 patients in which valvulotomy was performed were operated upon by cutting the pulmonary valve stenosis under direct vision after occluding both venae cavae temporarily. In the remaining 39 patients, the Brock transventricular technique was used.

Results of Systemic Pulmonary Artery Shunts for Tetralogy of Fallot:

The results of systemic pulmonary artery shunt procedures performed in the 97 patients with pulmonic stenosis are summarized in Table VI. To date, 106 operations have been performed upon 97 patients with 15 deaths, a mortality of 15%. This mortality figure includes the death of everyone who has ever been operated upon and subsequently died in or outside of the hospital. It also includes those patients subjected to thoracotomy and in which for various reasons it was not possible to make the shunt. As indicated in Table VI, 75% of this entire group have derived substantial benefit from their shunt operation in the form of lessened cyanosis, increased exercise tolerance, and in some cases increased growth. All patients in the improved group are capable of ordinary daily activities such as attending school, riding bicycles and moderately strenous play without limitations.

TRANSPOSITION OF THE GREAT VESSELS

In this severe anomaly the great veins (vena cavae and pulmonary veins) enter the heart into their respective chambers in normal relationship. However, the aorta with its coronary arteries arises from

TABLE VI

PULMONARY STENOSIS

CASES IN WHICH A SYSTEMIC PULMONARY SHUNT WAS ATTEMPTED OR COMPLETED

RESULTS AND MORTALITY

Total Patients			97*
Total Operations			106
Total Deaths			15**
A. Vascular Anastomoses Completed		86	
Patients improved	67		
Patients unimproved but living	8		
Died**	11		
B. Thoracotomies with Exploration without Anastomoses		11	
Died**	3		
C. Re-operations		9	
Improved	6		
Unimproved	2		
Died**	1		
Total all Patients			
Improved			75%
Unimproved			10%
Mortality**			15%

*Two additional patients were explored with the diagnosis of pulmonary stenosis but were found to have an Eisenmenger Heart. No shunts were attempted in these two patients.

**All postoperative deaths in or out of hospital regardless of postoperative interval are included.

the right ventricle and the pulmonary artery originates from the left ventricle. Obviously such a defect is incompatible with life unless there is one or more additional defects (atrial or ventricular septal defects, patent ductus) which permit some arterialized blood to reach the aorta. Even with these compensating defects, these children infrequently survive beyond infancy.

Attempts to correct the positions of the great arteries are prevented by the position of the coronary arteries. One of us, (R.L.V.) has conceived the idea of leaving the arteries in their respective anamalous positions and transplanting the great veins to the opposite side of the heart. This concept carried out to its conclusion would result in a normal circulation except that it will be

reversed as to right and left.

As the first stage, 4 patients have been subjected to anastomosis of the right pulmonary veins to the right atrium. Two are definitely improved and are now awaiting the next stage, and the other two have died of cardiac decompensation.

EISENMENGER SYNDROME
(High interventricular septal defect and pulmonary hypertension)

In the period of this report, five patients with Eisenmenger hearts have been subjected to thoracotomy under a mistaken diagnosis. Preoperative diagnoses were as follows: patent ductus arteriosus, two; pulmonic stenosis, two; and aortic coarctation, one.

In the first 4 patients nothing was done except an exploratory thoracotomy and three (75%) died immediately postoperatively usually in the recovery room. This result is invariable in patients with low cardiac reserve who are subjected to operation and in whom nothing can be done to improve the situation.

Thus, when the fifth case, an 18 month old infant with cardiac decompensation and pulmonary congestion was explored recently, and an Eisenmenger syndrome identified as the defect present rather than a patent ductus as preoperatively diagnosed, an unsuccessful attempt was made to close this interventricular defect under direct vision by opening the right ventricle. Despite the immediate tragedy of this failure, our group is encouraged sufficiently by the measure of actual knowledge derivable in no other way, as to make another attempt at a curative procedure, should we face the reality of a previously unsuspected Eisenmenger's type of heart at an operation initiated for some other lesion.

INTERATRIAL SEPTAL DEFECTS

Three patients have been operated upon for closure of interatrial septal defects. The approach in all three was to open the right atrium and under direct vision sew up the defect.

In the first two cases the pump-oxygenator developed by Dennis was used. Both of these patients succumbed due to technical errors which would be unlikely to recur, but which are inevitably a part of operating in unfamilar territory.

The third case was successfully operated upon recently by Lewis using vena caval occlusion with hypothermia to reduce the tissue oxygen requirements.

SUMMARY

We have presented a review of the 388 cases of congenital heart disease treated surgically in this hospital during the past 13 years.

It is perhaps apparent from this review that a considerable variety of both the ordinary and bizarre anatomical types of congenital heart disease can be substantially benefited by judiciously selected surgical procedures.

One of the most gratifying aspects of the study has been to note that 266 of these patients, or 68% of the entire group, have had curative or virtually curative surgery with a total mortality (early and late) of only 6%. These patients make up the groups with patent ductus arteriosus, aortic pulmonic septal defects, coarctations of the aorta, isolated pulmonic stenosis, and interatrial septal defects. All of these patients may be expected to have a normal or near normal life expectancy following successful surgery.

The fact that curative surgical techniques are not available for all congenital cardiac lesions is the stimulus that motivates the research activities of our group.

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II. MEDICAL SCHOOL NEWS

Coming Events

October 16-18	Continuation Course in Treatment of Diseases of the Chest for General Physicians
October 17	American Trudeau Society Lecture; "The Effects of BCG Vaccination in Silicotic Animals," Dr. Arthur J. Vorwald, Director, The Trudeau Foundation and the Saranac Laboratory, Saranac Lake, New York; The Nicollet Hotel; 8:00 p.m.
October 20-25	Continuation Course in Gastro-Intestinal Roentgenology for Radiologists
October 21	The Minnesota Pathological Society Lecture; "Hypothermia and Cardiac Surgery," Dr. F. John Lewis, Associate Professor, Department of Surgery; Owre Amphitheater; 8:00 p.m.
October 22	Leo G. Rigler Lecture; "X-ray Diagnosis of Diseases of the Gallbladder," Dr. B. R. Kirklin, Professor, Department of Radiology, Mayo Foundation, Rochester, Museum of Natural History Auditorium; 8:15 p.m.
October 30-31	Continuation Course in Medical Economics for Physicians
Oct. 31 - Nov. 1	Special Homecoming Program for Physicians
November 13-15	Continuation Course in Fractures and Surgery of Trauma for General Physicians

* * *

Continuation Course

The University of Minnesota will present a continuation course in Gastro-Intestinal Roentgenology for Radiologists from October 20 to 25, 1952. The course, which will be presented at the Center for Continuation Study, is intended for radiologists and will cover all aspects of roentgenologic examination of the gastro-intestinal tract. The distinguished visiting faculty members will include Dr. Mortimer Lubert, Associate Radiologist, Mount Sinai Hospital, Cleveland; Dr. Olle Olsson, Professor, Department of Diagnostic Roentgenology, University of Lund, Lund, Sweden; Dr. Richard R. Schatzki, Chief, Department of Roentgenology, Mt. Auburn Hospital, Cambridge, Massachusetts; Dr. Paul C. Swenson, Professor, Department of Radiology, Jefferson Medical School, Philadelphia, Pennsylvania; and Dr. Frederic R. Templeton, Professor, Department of Radiology, University of Washington Medical School, Seattle. The course will be presented under the direction of Dr. Leo G. Rigler, Professor and Head, Department of Radiology, and he will be joined by members of the faculty of the University of Minnesota Medical School and the Mayo Foundation.

On Wednesday, October 22, the annual Leo G. Rigler Lecture will be presented at 8:15 p.m. in the Auditorium of the Museum of Natural History. This year the Rigler Lecture will be given by Dr. B. R. Kirklin, Professor, Department of Radiology, Mayo Foundation, Rochester. Dr. Kirklin's subject will be "X-ray Diagnosis of Diseases of the Gallbladder." All physicians and other interested persons are invited to attend.

DON'T FORGET THE HOMECOMING PROGRAM OCTOBER 31-NOVEMBER 1

III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

October 20 - 25, 1952

Monday, October 20

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 Seminar; The Fundamentals of Treatment of Rheumatoid Arthritis; Paul Bilka; Heart Hospital Auditorium.

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

12:30 - 1:30 Physiology Seminar; Some Trends in Histochemistry; David Glick; 214 Millard Hall.

1:30 - 2:30 Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; 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 - 6:00 Physiology 114A and Cancer Biology 140 -- Research Conference on Cancer, Nutrition, and Endocrinology; Drs. Visscher, Bittner, and King; "Cortisone and X ray"; C. Martinez and T. Newberry; 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, October 20 (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.

3:30 - Psychosomatic Rounds; C. K. Aldrich; Bldg. I.

Tuesday, October 21

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 Hosptial.

* 8:00 p.m. Minnesota Pathological Society Meeting; Hypothermia and Cardiac Surgery; F. John Lewis; Owre Amphitheater.

Ancker Hospital

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

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

Minneapolis General Hospital

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

10:00 - Cardiac Rounds; Paul F. Dwan; Station I; Classroom.

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.

Tuesday, October 21 (Cont.)

Minneapolis General Hospital (Cont.)

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

Veterans Administration Hospital

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

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

8:45 - Surgery Journal Club; 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 - Chest Surgery Conference; Drs. Kinsella and Tucker; Conference Room, Bldg. I.

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

3:30 - 4:20 Clinical Pathological Conference; Conference Room, Bldg. I.

Wednesday, October 22

Medical School and University Hospitals

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-109, U. H.

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; Pediatrics Case; O. H. Wangensteen, C. J. Watson and Staff; Todd Amphitheater, U. H.

12:30 - 1:20 Radioisotope seminar; Cosmic Radiation; E. P. Ney; 12 Owre Hall.

1:30 - 3:00 Physiology 114B -- Circulatory and Renal Systems 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.

* 8:15 p.m. Annual Leo G. Rigler Lectureship; X-ray Diagnosis of Diseases of the Gall Bladder; Dr. Byrl R. Kirklin, Professor, Department of Radiology, Mayo Foundation, Rochester; Museum of Natural History Auditorium.

Wednesday, October 22 (Cont.)

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.

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.

12:30 - Pediatric Conference; Menstruation in Adolescent Girls; Nora Winther; Station I, Classroom.

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

2:00 - 4:00 Infectious Disease Rounds; 8th Floor.

4:00 - 5:00 Infectious Disease Conference; Classroom, 8th Floor.

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.

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, October 23

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; Countercurrent Separation of Biological Substances; J. G. Hamilton; 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.

Thursday, October 23 (Cont.)

Medical School and University Hospitals (Cont.)

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

Ancker Hospital

4:00 - Medical Pathological Conference; Auditorium.

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.

11:00 - Pediatric Rounds; Erling S. Platou; 7th Floor.

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

1:00 - House Staff Conference; Station I.

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, October 24

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; Carcinoma in Situ of the Cervix; John L. McKelvey; 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.

Friday, October 24 (Cont.)

Minneapolis General Hospital

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

10:30 - 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 Building.

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, October 25

Medical School and University Hospitals

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

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

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; Heart Hosptial 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; Nucleo-cytoplasmic Interrelationships in the Nerve Cells; J. F. Hartmann; 226 Institute of Anatomy.

Ancker Hospital

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

Minneapolis General Hospital

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

11:00 - Pediatric Clinic; C. D. May and Floyd Denny; Classroom, 4th Floor.

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

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* Indicates special meeting. All other meetings occur regularly each week at the same time on the same day. Meeting place may vary from week to week for some conferences.