

Staff Meeting Bulletin
Hospitals of the . . .
University of Minnesota

Rheumatic Fever

STAFF MEETING BULLETIN
HOSPITALS OF THE . . .
UNIVERSITY OF MINNESOTA

Volume VIII

Thursday, January 7, 1936

Number 11

INDEX

	<u>PAGE</u>
I. THREE WEEKS AGO	131
II. LAST TWO WEEKS	131
III. MOVIE	131
IV. ABSTRACT	
RHEUMATIC FEVER . . . Paul F. Dwan, and Arild E. Hansen	131 - 141
V. GOSSIP	141

Published for the General Staff Meeting each week
during the school year, October to May, inclusive.

Financed by the Citizens Aid Society

William A. O'Brien, M.D.

I. THREE WEEKS AGO

Date: December 17, 1936

Place: Nurses' Hall
Recreation Room

Time: 12:15 to 1:30 P.M.

Program: Movie: When Fish Fight
Symphony in Sight
Abstract: Borderline
Breast Tumors

Present: 94

Discussion: R. Koucky
W. T. Peyton
C. J. Campbell

II. LAST TWO WEEKS

Christmas Vacation.
No Bulletins issued.

III. MOVIE

Title: Steel

Released by: Audio Productions

IV. ABSTRACTRHEUMATIC FEVER

Paul F. Dwan,
Arild E. Hansen

Definition

Rheumatic fever is an infectious disease manifesting itself in all the tissues of the body, with a strong tendency to carditis and thus in its composite picture giving rise to the manifold condition known as the rheumatic state.

General consideration

Rheumatic fever contracted in childhood accounts for 25% of cardiac deaths up to 50 years. Kaiser in 1930 stated that 8 to 10% of all school children have rheumatic fever at one time or another. In 1935, he states that 1 to 3% of all school children showed evidence of rheumatic heart disease.

Age of onset

The disease has its inception in childhood. The peak of incidence in Minneapolis is between 5 and 6 years of age. This is somewhat younger than the figure given in other parts of the country and abroad.

Sex incidence

The sex distribution seems to be equal between boys and girls. However, most authors feel there is three to four times as much chorea among girls as boys.

Family incidence

It has been found that there is a definite familial incidence of rheumatic fever; there being three times as many cases among families of rheumatic children as among controls.

Geographic distribution

Rheumatic fever is fundamentally a disease of temperate climates. It is most common in cold damp climates, relatively rare in the southern zones, and almost unknown in the tropics.

Seasonal incidence

In the temperate zone, the disease is primarily found during the cold damp months with peaks in the spring and late fall.

Morbidity

Statistics gathered among school children in several large cities in the United States show an incidence of rheumatic fever of 1%.

Social distribution

Rheumatic fever is most frequently found in the urban population. Spot maps of our large cities show the greatest concentration of the disease among the over-crowded, cold, damp tenement districts. Congestion and poor hygienic surroundings seem to foster its spread.

Social aspect

The serious social aspect of rheumatic fever has in the past been unrecognized. Foyton says, "the resigned acceptance of the fact that hundreds of young lives are damaged by rheumatism contrasts vividly with the extraordinary efforts that are made to prevent tuberculosis." The campaign waged against tuberculosis is widespread and is becoming well nigh universal, but Dublin states that a child of 10 years has three times as many chances of dying from heart disease as from tuberculosis. The subject of the prevention and cure of cancer has occupied many institutions established and maintained for this particular purpose. However, cancer is less serious sociologically than rheumatic fever, in that it attacks more frequently the aged, and it does not produce cripples. Yet rheumatic fever, with its resulting carditis, has not been sufficiently considered as a public menace to seriously engage the attention of the medical profession as a whole.

Etiology

The etiology of rheumatic fever is at present generally considered as being the streptococcus. This organism, which is extremely susceptible to environment, may, under changed cultural conditions, vary its characteristics as to morphology, virulence and disease producing ability. By its presence and growth in a tissue,

the organism becomes so attired as to produce the symptom picture we recognize as rheumatic fever.

Swift and others offer the suggestion that the cause of rheumatic fever is a filterable virus. This virus produces an allergic situation which responds to thermal, chemical, bacterial or traumatic insults which then produce the clinical picture of the rheumatic state. This hypothesis would explain many of the bizarre manifestations of rheumatic fever.

Vitamin "C" deficiency has been studied as a cause of rheumatic fever. A wealth of material has been gathered but so far no definite conclusion can be drawn.

Clinical manifestations

Leseque' states that rheumatic fever licks the joints and bites the heart. Our present conception of the manifold nature of the disease is due largely to Cheadle, who in 1889 was the first to point out the wide variety of manifestations that may occur in the rheumatic state. Following an acute infection which serves as the portal of entry, the character of the disease is dependent upon those tissues involved. These may be manifested in such constitutional symptoms as malaise, chill, headache, general soreness, fever, anemia, anorexia and malnutrition. There also are the specific symptoms dependent upon the involvement of joints, central nervous system, skin, lungs, pleura, kidneys, muscle, peritoneum, and heart. This acute stage usually lasts from 2 to 4 weeks. After this comes a period of indefinite length with low grade fever and rapid pulse. The transition from this stage of active infection into the stage of convalescence is gradual and variable. One of the greatest difficulties in the determination of the end of the particular attack is due to the marked tendency toward recrudescence.

Recurrences

Shapiro studied 342 cases over a vary-

ing number of years. He found that 178 or 52.1% had only the one attack so that more than one-half of them gave no history of recurrences. Below is given his table showing the incidence of recurrences.

Incidence of Recurrences
in Juvenile Rheumatism

Total number of rheumatic children examined	342
Children with one attack only	178 (52.1%)
Children with recurrent attacks	164 (47.9%)

Recurrences within	No.	%	Total	%
1 year	44	26.8	44	26.8
2 years	44	26.8	88	53.6
3 years	28	17.1	116	70.7
4 years	20	12.2	141	86.0
5 years	5	3.1	141	86.0
6 years	8	4.9	149	90.0
7 years	4	2.4	153	93.3
8 years	5	3.1	158	96.4
9 years or more	6	3.6	164	100.0

Association with chorea

Sydenham's chorea has long been considered as part of the clinical picture of the rheumatic state. It is thought to be due to the action of both the infectious agent and its toxins. The former is thought to produce perivascular inflammatory changes and the latter to produce toxic changes in the cells of the cerebral cortex. The clinical picture of chorea with its purposeless movements, muscle weakness and mental irritability can be passed over.

Kuser, in 1,181 cases of rheumatic infection, found chorea in 28% of his cases. This rheumatic manifestation seems to have a predilection for girls. Keschner found a sex distribution of three females to every male with chorea. There also seems to be a racial distribution in that a large number of cases are found in Jews.

Chorea has an interesting effect upon the prognosis of rheumatic fever, according to Jones and Bland. For some reason, it seems to lessen the degree of carditis and the mortality. Jones and Bland give the following figures for this condition:

Incidence of Rheumatic Heart Disease

Chorea alone	3%
Chorea with arthritis	73%
Arthritis without chorea	86%

Mode of onset: Shapiro

Onset preceded by:	No.	%
Cold	27	13.5
Sore throat	9	4.5
Pneumonia	4	2.0
Measles	2	1.0
Scarlet fever	12	5.9
Chorea	20	9.9
Other	9	4.5
Gradual onset	90	44.8
Not obtained	28	13.9
Total	201	100.0

Carditis itself has been often found to be the presenting symptom. Smith and Sutton state that 78 of 427 cases of heart disease gave no history of poly-arthritis, chorea, joint, or growing pains. The occurrence of heart disease as the only manifestation of rheumatic fever varies with the climate as McLean points out that cardiac disease alone appears 52% more frequently in Alabama than in New York City.

Incidence of carditis

Rheumatic fever has a strong tendency to carditis with permanent damage. Herein lies the real danger of this disease. The incidence of carditis found by five writers is given below.

Hagge	50%
Gibson	74
Church	57.5
Kemp	80
Jones	86

With these figures representing the general statement of carditis, we can consider individually the types of involvement. Church finds endocarditis in 50% of his cases of rheumatic fever. Gibson finds pericarditis in 50% of his cases. Coombs, Swift and Less find some myocardial damage to be an almost constant finding.

Pericarditis

Coombs cites figures from the post-mortem records of the Bristol General Hospital. He found the following incidence of pericarditis:

	<u>Pericarditis</u>
1. Patients dying in 1st decade	100.00%
2. " " " 2nd "	83.3 %
3. " " " 3rd "	41.6 %
4. " " " 4th "	23.0 %
5. " " after 40 years	26.0 %
6. Taking all ages together	53.0 %

Gibson in a recent study found that only 10 of 73 cases examined at necropsy showed no pericarditis.

Myocarditis

Tony feels that myocardial involvement is, as a rule, the most important feature of the rheumatic disease, for it is the condition of the myocardium, more than any other single factor, that in most cases determines the prognosis, and it is the myocardial damage which is responsible for death in most of the rapidly fatal cases. Coombs, from a study of the hearts of fatal cases of rheumatic fever, concludes, "probably then, death in rheumatic heart disease occurring before the age of 16 is reached is more often due to acute inflammation of the myocardium than to anything else." Symptoms of cardiac insufficiency with dyspnea and usually with precordial pain are present, and the signs of early decompensation--pulmonary congestion, cyanosis of the lips and fingers, and enlargement and tenderness of the liver appear.

Endocarditis and valvulitis

The importance of disease of the

endocardium and heart valves is second only to that of myocarditis. Active inflammatory and destructive changes, probably toxic in origin, are serious and the fibrosis of repair may be just as severe in its effects on valve function as are the active changes. The mitral valve is the most frequent seat of extensive trouble, the aortic next in frequency, while the valves of the right side show a low incidence of serious involvement.

Active disease may promptly disappear, leaving a minimal degree of fibrosis limited to the valve orifice. With no further activity, this orifice is sufficient for the needs of the child's heart, but, as the heart chambers and valves reach adult size, the aperture at the mitral tip may remain in the original dimensions fixed by the fibrosis which followed the childhood attack. The stenosis is relative, develops gradually and insidiously, and in the absence of further active phases of disease may not give definite signs or symptoms until approaching maturity brings a rapid growth of the heart.

Course

The course of rheumatic fever varies tremendously. It may run its course in a month and leave behind no evidence of damage. It may invade the body and kill rapidly by the overwhelming nature of the infection. It may present the picture of one acute illness with recovery but frequent recurrences until the ultimate damage is great. It may simulate such a chronic disease as tuberculosis in which the disease process slowly burns, manifesting itself only slightly but producing more damage slowly but inevitably.

The following cases will illustrate the variation in the course of rheumatic fever:

Case I: Hospital No. 1

Onset of disease November 1935, at age of 12. He made a gradual recovery and returned to school in

February 1936. A few days later, he had a recurrence of symptoms, developed pleurisy, evidence of mitral heart disease, became progressively worse and died in April 1936.

Case II: Hospital No.

Onset of disease February 1932, at age of 8. Her presenting picture was that of chorea. She showed evidence of early mitral disease as evidenced by a systolic murmur at the apex and x-ray findings of right ventricular enlargement and dilatation of her left auricle. She had two recurrences of chorea but now is doing well and shows no evidence of mitral disease.

Case III: Hospital No. 1

Onset of disease in 1927, at age of 5. He had a violent illness and was left a cardiac cripple with mitral disease and aortic regurgitation. His course was that of continued invalidism with frequent breaks in compensation. Finally, he died of heart failure in February 1935.

Case IV: Hospital No.

Onset of disease January 1935, at age of 15. She had a relatively severe illness and emerged after 6 months with early mitral disease and delay in her conduction time. She gradually improved, had no recurrences, and now is subjected to no restrictions. She still has evidence of mitral disease upon auscultation but from year to year she has shown no ill effects from her mode of living.

Case V: Hospital No. 1

Onset of disease in August 1932, at age of 8. He had a recurrence in November and another one in December of the same year. Upon a third recurrence in February 1932, he was found to be developing mitral disease. Since then, he has had 3 recurrences of his disease but now is doing well. He

has a mild mitral disease which does not impair his ability to attend school. A sedentary life seems to add no further strain on his heart.

Case VI: Hospital No.

Onset of disease in May 1933, at age of 13. He emerged from his first attack without evidence of heart disease. However, a year later, he had a recurrence of his disease and developed aortic regurgitation. Since then, he had done well and now has aortic regurgitation with no functional impairment of his heart.

Diagnosis

Of Rheumatic State:

The principal problems in dealing with rheumatic fever are two. First, the accurate diagnosis of the condition so as to be able to firmly go forward with the treatment. Second, the management of each case as an individual. No rules can be laid down and each case must be handled on its merits. Unless a definite diagnosis is made, there is a tendency to allow the child up after the subsidence of the initial febrile rise. It must be remembered that the patient then enters a prolonged period of convalescence in which the disease process is still present but manifesting itself only by a low grade fever. It is the care during this stage that determines the patient's outcome.

The most confusing differential is between the mild symptoms of rheumatic fever and the non-rheumatic pains of childhood. Shapiro has tabulated the salient features of these two conditions as given in the table below.

The Differences Between Non-Rheumatic "Growing Pains"
and Joint Pains of Subacute Rheumatic Fever

	<u>Non-Rheumatic "Growing Pains"</u>	<u>Joint Pains of Subacute Rheumatic Fever</u>
<u>Time of pain:</u>	Soon after going to bed. Pain gone in morning. No pain on motion.	Worse on arising. Exaggerated motion. Difficulty in walking; may cause limp. Pain present during most of day; disappears on getting warm in bed.
<u>Location of pain:</u>	In muscles of thighs and legs. Child vague in pointing out site of pain.	In joints themselves. Child points to knees or ankles. Often complains of pain in joints of upper extremities also.
<u>General Health:</u>	Usually good.	Usually poor.
<u>Other signs of rheumatic activity:</u>	Usually none.	Common; may have frequent nose-bleeds, unexplained fever, pallor, abdominal cramps, undernourished. Evidence of carditis.
<u>Objective findings in joints:</u>	None.	Often joints are slightly swollen and hot.
<u>Family history of juvenile rheumatism:</u>	Uncommon.	Very common.

Another difficult point is the evaluation of cardiac findings. Frequently, the question of congenital heart disease arises. The table below presents the characteristic features of each.

Differential Diagnosis of Congenital and Acquired Heart Disease

	<u>Congenital</u>	<u>Acquired</u>
<u>Age:</u>	Usually discovered in early infancy.	May develop any time in life but usually after infancy.
<u>History:</u>	Beginning usually in infancy and referable to circulatory changes.	Rheumatic fever or other infectious process.
<u>Symptoms:</u>	Lack of aortation: 1. Cyanosis 2. Clubbing	Passive congestion: 1. Edema 2. Enlargement of liver and spleen

	<u>Congenital</u>	<u>Acquired</u>
<u>Symptoms:</u> (Cont.)	3. Delayed mental growth 4. Delayed physical growth 5. Dyspnea due to poor oxygenation	3. Dyspnea due to edema. 4. No cyanosis. 5. Clubbing and late event.
<u>Progression of findings:</u>	1. Cardiac findings fail to progress.	1. Progressive changes in findings.
<u>Cardiac hypertrophy:</u>	Right-sided or not at all.	Left-sided in aortic disease. Right-sided in mitral disease.
<u>X-ray changes:</u>	Enlargement of right ventricle. Lack of enlargement of left auricle as seen in esophagogram.	Enlargement of both ventricles. Enlargement of left auricle, as seen in esophagogram.
<u>Apex beat:</u>	Feeble or unchanged.	Accentuated.
<u>Murmur:</u>	Character: Loud, harsh, musical Location: At base.	Character: Soft blowing. Location: At apex.
<u>Blood changes:</u>	Polycythemia. Increase in hemoglobin.	Anemia. Leucocytosis.
<u>Vascular changes in eye grounds:</u>	With cyanosis: 1. Irregularities in lumina of the vessels, which become tortuous like large angle worms. Knapp, 1861. 2. Vessels become darker. 3. Peripheral twigs distended. 4. Small hemorrhages.	No characteristic changes.
<u>Electrocardiograph:</u>	1. Right axis deviation. 2. Great amplitude to complexes. 3. Great variation in the height of the complexes.	Changes of conduction chiefly. Secondary alteration of axis deviation. Mitral - right axis deviation. Aortic - left axis deviation.

There are many conditions which must be considered in the complete differential diagnosis of the rheumatic state. Among those which are not usually considered in a discussion of the subject but have impressed us of being important are acute lymphatic leukemia, acute appendicitis, and meningococcemia.

Laboratory findings:

The blood shows an increase in sedimentation rate, a polymorphonuclear leucocytosis, and a secondary anemia. Urinalysis allows detection of renal damage on a rheumatic basis. Other laboratory data have been gathered but

have not aided appreciably in the diagnosis of rheumatic fever.

Electrocardiogram

Electrocardiographic study is becoming an important adjunct in the diagnosis of active carditis. The usual manifestations in the conventional leads are conduction delays, abnormalities of the QRS complex and S-T interval as described by Swift, partial or complete heart block, arrhythmias and abnormalities of the axis deviation. The highest incidence of these changes in acute rheumatic fever are recorded by Swift who found electrocardiographic evidence of myocardial damage to be almost a constant finding. Most authors do not agree entirely with Swift but his work was done by serial study of acutely ill children.

The fourth or chest lead is proving of value in the diagnosis of acute carditis. It is felt that the fourth lead elicits changes by reflection or damage in heart areas which are "silent" to the conventional leads.

X-ray

Roentgenologic investigation is warranted at the onset of the disease and periodically throughout the life of the

patient who suffers heart damage. The x-ray is invaluable in following the effect of this disease on the heart.

Pathology

With the exception of the small sessile vegetations on the cardiac valves, the exudate in the pericardium, and the subcutaneous nodules, the principal morbid changes in rheumatic fever are of microscopic dimensions. These microscopic lesions occur largely in connective tissue, particularly in and around small vascular branches and are known as Aschoff bodies.

Prognosis

The prognosis of rheumatic fever depends to a large extent upon the carditis resulting from the infection. The incidence of carditis has been discussed above. The prognosis of rheumatic heart disease may be considered below. Stroud states that of 307 cases of rheumatic heart disease 40% were totally disabled or dead at the end of 10 years.

Prognosis of Rheumatic Infection During a Ten-Year Period

<u>Author</u>	<u>Period</u>	<u>No. of Patients Traced</u>	<u>Average Age At Onset</u>	<u>Average Duration of Infection: Years</u>	<u>Deaths %</u>	<u>Valvular Heart Disease %</u>
Ash	1922-32	416	6.8	7.5	22.3	66.1
Jones & Bland	1921-32	1,000		8.0	21.7	70.6
Kaiser		564		5.8	8.2	64.0
Findlay	1920-29		7.0	5.0+	19.9	60.9
Stroud	1922-31	307	7.3		31.9	
Wilson	1916-27	355	7.3		10.8	79.5

The prognosis of rheumatic fever has been reported by the six authors given above. It will be seen that their mortality varies from 10.8 to 31.9%.

John Lovett Morse reviewed 100 cases of heart disease which had been seen in his office and followed from 10 to 30 years.

Dead	36%
Cardiac invalids	3%
Hearts normal	37%
Hearts slightly damaged	18%
Hearts not examined	6%
Alive and well	61%

Relation of prognosis to cardiac enlargement, myocarditis and pericarditis. Morse offers the following table:

	<u>Dead</u> 36 Cases	<u>Alive and Well</u> <u>Hearts Normal</u> 37 Cases	<u>Alive and Well</u> <u>Damaged Hearts</u> 18 Cases
<u>Enlargement</u>			
Slight		14)	4)
Moderate		4(-- 23 = 62%	4(-- 10 = 55%
Considerable		5)	2)
<u>Myocarditis</u>	55%	21%	17%
<u>Failure of Compensation</u>	30%		
<u>Pericarditis</u>			
Dry	10) (36%	2) (8%	2 = 11%
Effusion	3)	1)	

Relation of care received to prognosis

Morse has shown that proper treatment does influence the prognosis of rheumatic fever. He stresses the fact that proper treatment is largely a matter of complete rest throughout the duration of the infection. His results are tabulated below:

Care:	Dead within 4 mo.	36 Cases after 4 mo.	Cardiac invalids 3 cases	Alive and well. Condition of heart not known 6 cases	Alive and well. Heart slightly damaged. 18 cases	Alive and well. Heart normal. 37 cases.
Very good)			33%	33%)	11%)	40%)
(48%	55%			(66%	(72%*	(70% ±
Good)			---	33%)	61%)	30%)
Fair	12%	27%	33%	16%	6%	13%
Poor	32%	18%	33%	---	---	5%
?	2%	---	---	1%	4%	4%

* 93% of those in whom care is known.

± 80% of those in whom care is known.

Treatment:

The care of a patient with rheumatic fever depends on an understanding of the acute and chronic aspects of the disease, and the points discussed above. Rheumatic fever has 4 distinct divisions in its clinical course.

1. The initial infection which causes invasion of the body. As was pointed by Shapiro, this is usually some streptococcal infection, usually of the upper respiratory apparatus. This episode is accompanied by local and general response to the infection. The treatment is symptomatic.

2. After a pause of about 2 weeks, the disease strikes, manifesting itself in all the tissues of the body. Treatment is again symptomatic and the patient submits well to care because of he is sick.

3. Following this acute stage of the disease, there follows a long convalescent period in which the disease is slowly but steadily producing permanent damage. Treatment in this stage is difficult. The patient feels pretty well and rebels at enforced rest. The essential care at this time is absolute rest and careful observation. This is the stage where all of our diagnostic acumen is brought into play. Morse has shown in the table given above that proper care in this stage determines the ultimate condition of the patient. Proper care is difficult in the home because of lack of cooperation. Prolonged stay in the ordinary hospital is prohibitive because of the cost. Convalescent homes with proper facilities and a low per diem cost seem to be the best solution to the problem.

4. After the infectious process has burned itself out, we are left with the permanent damage done by the disease. This is likened to the burned over countryside following a forest fire. Treatment now consists of bolstering and protecting the damaged heart so as to bring a maximum of comfort and happiness to these unfortunate children.

Summary

We have attempted to paint a picture of the rheumatic state as it is described in the literature and as we have seen it in this clinic. Years of contact with this disease have added much to our respect for this terrible affliction of childhood. We enter a plea for accuracy of diagnosis and proper care so that within our power we can limit the ravages of this disease.

Bibliography

1. Ash, Rachel
Prognosis of rheumatic infection in childhood.
Am.J.D.Ch.52: 280, 1936.
2. Bass, M. H., Mond, H., Musseloff, C. R., and Oppenheisner, E. T.
Systolic murmurs in children.
J.A.M.A. 101: 17-20, (July 1) 1933.
3. Bland, E. F., Jones, T. D., and White, P. D.
Disappearance of the physical signs of rheumatic heart disease.
J.A.M.A. 107: 569-573, (Aug.) 1936.
4. Coburn, A. F.
The factor of infection in the rheumatic state.
Williams and Wilkins, 1931.
5. Dana, H. W., and Reidy, J. A.
Mitral valve disease; pathological report.
Am. J.M.Sc.191: 109-117 (Jan.) 1936.
6. Dwan, F. F.
Heart disease vs. "heart trouble."
J.Lancet 55: 277-280, (May 1) 1935.
7. Dwan, F. F. and Shapiro, M. J.
The four lead electrocardiograph in children.
In press, Am.J.D.Ch.
8. Jones, T. D. and Bland, E. F.
The clinical significance of chorea as a manifestation of rheumatic fever.
J.A.M.A. 105:571-577 (Aug.) 1935.

9. Katz, L. N. and Kissin, M.
A study of lead IV.
Am.Heart J. 8:595, 1933.
10. Maliner, M. M.
Adrenalin chlorid test for
murmurs in children.
Arch. Ped. 49:305-313 (May) 1932.
11. Martin, A. T. and Ellenberg, S. L.
Rheumatic involvement of the
appendix.
J. of Ped.:9: 234, 1936.
12. Morse, J. L.
Prognosis of acute endocarditis in
childhood; analysis of 100 cases.
Am. J.Dis.Child. 42: 735-748,
(Oct.) 1931.
13. Shapiro, M. J. and Shapiro, G. K.
Clinical studies in juvenile
rheumatism.
Minn. Med. 18: 370-377, (June) 1935.
14. Swift, H. F. and Cohn, A. E.
Electrocardiographic evidence of
myocardial involvement in rheumatic
fever.
J. Exper. Med. 39:1-35, (Jan.) 1924.
15. Wood, F. C. and Wolfert, C. C.
The electrocardiographic diagnosis
of coronary occlusion by the use
of chest leads.
Am.J.Med.Sc.183: 30, 1932.

V. GOSSIP

We wish you all a very happy and successful New Year.....The Center for Continuation Study has just announced the program of the Postgraduate Medical Institute to be held January 18th to February 13th. The first week will be devoted to traumatic surgery, the second to obstetrics and gynecology, the third to pediatrics, and the fourth to internal medicine. Programs will start at 8 and end at 5 daily. In addition, three nights each week will be given over to special lectures. A student may register for one or more weeks and a certificate will be awarded after the successful completion of each course. Students from outside the Twin City area will be expected to

live at the Center. The tuition fee is \$15.00 per week which does not include living expenses. The unique feature of the Institute is the consideration of only one subject a day, as an example: Monday, January 18th, the program will be "Injuries of the Spine, Ribs and Pelvis," by Dr. Wallace H. Cole and associates.....Melville H. Manson left January 3d to assume his new position with the American College of Surgeons. Dr. Manson is one of our own graduates and did all of his graduate work here after completing his internship at the University of Wisconsin General Hospital. "Doc" Manson will be missed by the students who liked him very well as a teacher, by the staff who found in him an able associate, and by his many friends who will miss personal contacts with him. All join in wishing him well in his new work.It seems that I have held off long enough telling you that our new daughter, Kathleen Ann, born the day before Christmas, is doing very well. She is said to look like her father and as someone added, when he had hair and weighed eight pounds.By the way, we are pleased to announce that Alfred Washington Adson, famed Neuro-Surgeon of the Mayo Clinic, will be our guest at a staff meeting later in January. On previous occasions, he has discussed for us the surgical treatment of vascular disease, hypertension, meningiomas, and cord tumors.....M.J.Shapiro, who is so liberally quoted in today's abstract, is none other than Moe Shapiro of our medical staff. Dr. Shapiro has had an unusual opportunity to study rheumatic fever in childhood. He is at present in charge of WPA's star medical project in the northwest at the Lymanhurst School where a group of rheumatic children are hospitalized to continue their school work....Our authors today are from the Pediatrics Staff. Dr. Dwan's contributions in electrocardiographic phases of heart disease in childhood are rapidly gaining. Instead of waiting until the time when men develop an avocation by force of circumstance, he has started his hobby early in life. When you visit his home, it looks like a gangster's arsenal for he is a gun collector...Dr. Hansen has recently recovered from a severe illness and all are glad to see him about again. In spite of his very good nature, he does not appreciate reference to the graduate work he did on a special project at Jail University. Adios