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Systemic Fibrinoid Diseases

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I. SYSTEMIC FIBRINOID DISEASES: SIMILARITY TO EXPERIMENTAL LESIONS IN RABBITS**

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INTRODUCTION

In recent years renewed interest has been shown in certain diseases which, although varying in their clinical manifestations, have been grouped together as "connective tissue diseases." As proposed by Klinge in 1933,[1] the group included rheumatic fever, rheumatoid arthritis, periarteritis nodosa, and dermatomyositis. In 1938 Masugi and Yä-Shu[2] added scleroderma and in 1942 Klemperer et al.[3] placed lupus erythematosus in this category. The latter authors also introduced the term "diffuse collagen disease" to emphasize the "systemic involvement of the entire connective tissue of the human body" in each of these diseases. Each of the above writers also stressed the presence of "fibrinoid degeneration" in these diseases and considered this material to be the most prominent alteration in the connective tissue. Klemperer[3] described "fibrinoid degeneration" as a "descriptive morphologic term indicating certain well defined optical and tinctorial alterations in the collagenous fibers and ground substance," and he considered "fibrinoid degeneration and collagen sclerosis" as the "morphologic expression of different phases of a disturbed colloidal collagen system."

The concept of these diseases therefore is based largely on the occurrence of fibrinoid in lesions of the cardiovascular system and connective tissue, and it is generally agreed that any of these diseases may be associated with the development of widespread lesions affecting many organs and tissues.[4] For reasons to be discussed later, the disease known as thrombotic thrombocytopenic purpura should also be classified in this group.

Altshuler and Angevine[5] performed histochemical studies on the fibrinoid material occurring in these diseases and found it to have similar tinctorial properties in each type. They suggested that it represented an alteration in the "ground substance." The similarity of its morphologic and tinctorial properties suggests, in addition, that its chemical composition in each case may be similar or identical.

Clinically, these diseases can usually be separated, but the wide variation in the lesions occasionally creates difficulty in recognition of any one specific entity. It is perhaps a less well known fact that these diseases also overlap morphologically and, indeed, occasionally give rise to alterations which suggest an entirely different diagnosis.

As indicated in previous papers[6,7,8,9] the generalized Shwartzman phenomenon is characterized pathologically by the occurrence of widespread fibrinoid lesions affecting the cardiovascular system, and this phenomenon provides a useful model for studying the origin of this material and the development of lesions associated with its presence. The purpose of this paper is therefore two fold: first, to point out the basic morphologic similarities between the "collagen" diseases and second, to illustrate the similarity between the lesions in these diseases and those occurring in the generalized Shwartzman phenomenon.

MATERIALS AND METHODS

The material to be presented consists of six recent cases of fibrinoid disease studied in the Department of Pathology and of material obtained from experimental procedures carried out in rabbits. The latter consists of selected slides from animals given

** These studies were supported by grants from the Minnesota Heart Association, the American Heart Association, and the Youngstown Area Heart Association.

meningococcal endotoxin, alone or in conjunction with certain acidic polymers such as sodium polyanetholsulfonate (Liquoid-Roche), the sodium salt of the ester of polyvinyl alcohol and polysulfonic acid (PVAS-Roche), or dextran sulfate. The methods of preparation and administration of these materials and a detailed account of the morphologic changes which occur following their administration have been described elsewhere.[6,7,8]

PRESENTATION OF MATERIAL

Case I. 3 year old white male.

Admission 4/17/54. Expired 4/19/54.

Complaints: Fever, anorexia, skin rash, heart murmur: 3 weeks duration.

History: Onset with respiratory infection, one month prior; duration 4 days. Week later developed urticarial rash, fever, abdominal and leg pain. 3 days later developed painful left knee. No murmur at this time. Hb 11 Gm.%, WBC 13,700 with 60% N, 5% E, 2% Ba.

Physical: T 102^{6} P 120 R 32 BP 110/65. Acutely ill. Urticarial rash over abdomen, back, chin, arms, upper thighs. Lungs clear. Sinus tachycardia. Cardiomegaly with grade III systolic, grade I diastolic apical murmurs transmitted throughout chest. Apical systolic thrill. Liver 2 FB below RCM. Left wrist painful on motion.

Laboratory: Hb 10.6 Gm%; WBC 11,650 with 80% N. Sedimentation rate 101 mm. ASO titer 625 u/cc. Blood cultures sterile. Urine albumin and sugar 1+.

Course: Digitalized. Begun on ACTH 75 mgm every twelve hours; ascorbic acid 500 mgm daily. Expired 2 days after admission.

Autopsy: Bilateral pulmonary edema and generalized cardiac enlargement (140 grams). Anterior pericardium showed areas of fibrinous pericarditis. The free margins of the tricuspid and mitral valves showed firm, irregular nodules approximately 1 mm. in size. Liver weighed 520 grams and showed passive congestion.

Microscopically there was a severe pneumonitis characterized by edema, polymorphonuclear infiltration, alveolar hyaline membrane formation, and occasional arteries showed endothelial proliferation. The heart showed diffuse acute rheumatic changes with numerous Aschoff bodies and acute mitral and tricuspid valvulitis with fibrinoid deposition. The liver and spleen showed passive congestion.

Case II. 40 year old white female.

1st Admission: Feb., 1953: Discharged Oct., 1953. 3 year history lupus erythematosus.

Past History: Butterfly rash 1950. Treated with bismuth, some improvement. Fall, 1951: Rash reappeared, general malaise developed. Spring, 1952: Placed on bed rest 3 months. Summer, 1952: Swelling and aching of hands, mild migratory arthalgia. Begun on cortisone 25 mgms. daily; increased to 300 mgms. daily.

Physical: Scaling, crusty dermatitis face, ears, neck, palms. Deep tendon reflexes hyperactive.

Laboratory: Constant 1-2+ albuminuria, leukopenia (850-8,200), anemia (Hb 3.9-11.9 Gm.%). Sedimentation rate 52 to 162 mm. 3 positive L.E. tests.

Course: Cortisone 150-200 mgms, daily. 1 month later developed psychotic reaction; gradually improved and was discharged 10/7/53.

2nd Admission: 12/30/53; expired 1/2/54.

Complaint: 4 week history productive cough, weakness, weight loss, some dyspnea.

Physical: T 102^{6} P 132, irregular R 24 BP 130/100. Left ventricular enlargement grade II precordial systolic murmur. Inspiratory wheezes right

lung. Liver 3-4 cm. below RCM. Spleen questionably palpable; R and LUQ tenderness.

Laboratory: Urine 1+ albumin. Hb 8 Gm.%. WBC 4,950 with 88% N. Venous pressure 8.5 cm. citrate, circulation time arm to tongue 30 sec. BUN 31 mgm.%; total serum proteins 5.5 Gm% with globulin 4.0 GM.%. EKG - premature ventricular contractions. X-ray - atelectasis RUL.

Autopsy: Bilateral pleural effusion (500 cc), pericardial effusion (450 cc), and ascites (150 cc). Heart enlarged (360 grams) with minimal mitral valve thickening. The lungs showed areas of bronchopneumonia and atelectasis. The liver (1550 Gms.) showed mild fatty changes. The spleen was enlarged (190 Gms.) and fibrous. Kidneys weighed 150 Gms. and showed subcapsular petechiae.

Significant microscopic findings: Renal lesions typical of lupus erythematosus; areas of hemorrhage and necrosis in lymph nodes and spleen; myocardial fibrosis with some thickening of mitral valve, and areas of pulmonary fibrosis and bronchopneumonia with "hyaline" thrombi in the smaller arteries. No periarterial fibrosis of the splenic vessels and no definite "hemotoxylin bodies" were seen.

Case III. 76 year old white female.

Admitted for 6th time 1/25/54. Expired 2/9/54.

Complaints: Dysphagia, sensation of choking, poor vision, exertional dyspnea with dry cough, anorexia, and occasional stiffness and tenderness of fingers.

Past History: 3 previous admissions for bleeding duodenal ulcer - medical treatment. 2 previous admissions for HCVD (BP 180/80, 198/84). Congestive heart failure with no marked improvement following digitalization.

Physical: T 98^{6} P 88 R 32 BP 170/95. Scattered rales, cardiomegaly with occasional premature ventricular contractions. Left lens opacity. Thickening of skin and subcutaneous tissue of fingers and toes.

Laboratory: Urine 2+ albumin. Hb 11.7 Gm.%; WBC 13,250 to 23,100 with 78 to 89% N. Stool 4+ guaiac. Blood culture sterile. Prothrombin time 14.7 sec. (control 12.5). X-rays: Pulmonary fibrosis; lack of esophageal tone with narrowing of distal end; deformed duodenal bulb.

Course: Developed bronchopneumonia 2/6/54. Begun on achromycin. 2/9/54 developed pulmonary edema and expired.

Autopsy: Diffuse pulmonary fibrosis and bronchopneumonia. Pericardial effusion (150 cc), heart normal size. Severe coronary atherosclerosis with myocardial fibrosis. Postmortem rupture of esophagus with liberation of gastric contents into left pleural cavity. Multiple small ulcerations of duodenum. Liver and spleen normal. Kidneys small (90 Gms.) and pitted. Skin appeared generally thickened over abdomen and anterior chest. Microscopic findings included renal lesions similar to those of lupus erythematosus and accelerated hypertension with severe hyaline arteriolosclerosis, and focal areas of hepatic necrosis. The mitral valve was thickened but showed no acute changes. There was diffuse myocardial scarring with occasional areas of perivascular cellular reaction, and severe coronary atherosclerosis. The duodenum showed healed ulcerations; the esophagus a postmortem rupture. Extensive pulmonary fibrosis with some bone formation, along with acute bronchopneumonia, was also observed. The skin showed changes typical of scleroderma.

Case IV. 11 year old white female.

Admitted 2/5/54. Expired 2/6/54.

History: Onset October 1953 with rash on bridge of nose. Two weeks later developed weakness and drowsiness. Hospitalized Nov. 1953 with diagnosis lupus erythematosus (bone marrow).

Treated with cortisone and potassium chloride. Subsequently developed edema of extremities and hypertension (to 190 systolic). Four days prior to admission developed facial edema, lethargy, and vomiting.

Physical: T 99^2 P 120 BP 150/110. Maculopapular rash over cheeks, upper eyelids. Lungs clear. Left cardiac enlargement with rough, grade II apical systolic murmur. Ascites and abdominal tenderness.

Laboratory: Urine 2+ albumin and red cells. WBC 11,450 with 87% N. Hb 8.9 Gms.%. Sedimentation rate 131 mm.

Course: Pulmonary edema developed and patient expired.

Autopsy: Ascites (100 cc), bilateral pleural effusion (75 cc), pericardial effusion (50 cc). Bilateral pulmonary edema. Heart enlarged (340 Gms.), with mitral valve thickening. Liver (1190 Gms.) and spleen (150 Gms.) enlarged; grossly normal. Kidneys enlarged (220 Gms.) with numerous petechial hemorrhages.

Microscopically, a variety of changes was observed. The renal vessels showed the typical changes of periarteritis nodosa while the glomerular changes varied from "wire loop" lesions of lupus erythematosus to changes indistinguishable from subacute glomerulnephritis. Lesions of periarteritis nodosa were observed in other organs and this seemed to be the predominant lesion. However, the mitral valve showed acute cellular reaction without fibrinoid and the myocardium showed numerous areas of focal necrosis and cellularity, some of which resembled Aschoff bodies. The splenic arteries showed no "periarterial fibrosis" and no "hemotoxylin bodies" were observed.

Case V. 32 year old white female.

1st Admission: 4/22/50 for study of liver and kidney malfunction.

History: Easy bruising, multiple food allergies and intermittent eczema since birth. Epistaxis with menses. 1946: Positive Wasserman developed during pregnancy; negative 6 months before. 1948: Developed albuminura with edema in 3rd trimester of 2nd pregnancy. Albuminuria persisted post partum (4+). 1949: 4+ cephalin flocculation with increased prothrombin time.

Physical: Eczematoid eruption on cheeks, nose, forehead. Ecchymoses of lower extremities. Soft apical systolic murmur.

Laboratory: Urine albumin 1-2+. Hb 13.8 Gm.%. WBC 6,800 with 80% N. Platelets 168,000. Bleeding time 3 minutes; capillary clotting time 13 minutes 15 sec. Sedimentation rate 72 mm. Stool guaiac 1+. Lee White clotting time 98 minutes in third tube.

Discharged: 4/28/50.

2nd Admission: 5/18/50. Same complaints and P.E.

Laboratory: Urine albumin 3+. Hb 12.7 Gm.%. WBC 10,500 with 80% N. Sedimentation rate 115 mm. Prothrombin time 26.5 sec. (control 12.1 sec.) Serum proteins 6.1 Gm.% with globulin 3.6 Gm.%. Wasserman 4+, VDRL weakly positive.

Course: ACTH 20 mgm. daily for 8 days. No improvement. Discharged 6/2/50.

3rd Admission: 7/9/51

History: 1st trimester of pregnancy. Weight gain of 20 pounds with nausea and vomiting.

Physical: Essentially as before plus moderate pitting edema of lower extremities.

Laboratory: Urine albumin 2+. Hb 10.6 Gm.%. WBC 4,350. Platelets 144,000. Sedimentation rate 164 mm. Serum proteins 5.4 Gm.% with globulin 3.3 Gm.%.

Discharged 7/21/51 to be followed in OPD.

4th Admission: 6/6/54. Expired

6/13/54.

History: Onset acute abdominal pain followed by tenesmus and diarrhea 6 days prior to admission. Abdomen became distended and patient was hospitalized elsewhere; treated with suction for 5 days.

Physical: T 99 P 100 BP 130/80. Soft apical systolic murmur. Abdomen distended with diffuse and rebound tenderness. Bowel sounds normal on admission but absent 3 hours later. Pelvic and rectal: boggy resistance, slight tenderness in cul de sac.

Laboratory: Urine: albumin 2+, sugar trace, RBC 3+. Hb 11.3 Gms.%. WBC 7,300 with 78% N. BUN 33 mgm%, K 3.7 meq/L. Serum proteins 5.1 Gms%.

Course: Gradually deteriorated, expired 6/13/54.

Autopsy: Numerous ecchymoses. 500 cc. bloody fluid in abdomen; 800 cc. dark fluid each pleural cavity. Heart normal size with areas of fibrosis up to 6 mm. Atelectasis both lower lobes of lungs. Multiple subserosal hemorrhages throughout intestinal tract with fibrinous adhesions between bowel loops. Both adrenals enlarged and replaced almost entirely by hemorrhage. Liver 1790 Gms., several areas of hemorrhage. Spleen enlarged (340 Gms.) and soft. Kidneys enlarged (230 Gms.) with multiple subcapsular hemorrhages. Uterine cavity filled with blood.

Microscopically, the renal changes were typical of lupus erythematosus, with many "wire loop" lesions. The heart showed old scars and also areas of acute necrosis with neutrophilic reaction. The mitral valve showed hyaline thickening but no acute changes. The liver showed diffuse areas of hemorrhage and necrosis and the vessels and sinusoids contained distinctly eosinophilic, homogeneous refractile thrombotic material. In some cases this material was also beneath the endothelium of the vessels. Focal areas of hemorrhage and necrosis were also seen in the spleen. The intestinal lesions consisted of diffuse hemorrhage associated with vascular occlusion by material similar to that described in the liver. In addition occasional arteries showed fibrinoid replacement of most of the vessel wall with acute polymorphonuclear reaction producing a picture identical with periarteritis nodosa.

Case VI. 7 months old white male.

1st Admission: 7/20/53.

Complaint: Intermittent bloody-mucous stools since birth.

Physical: Right cervical lymphadenopathy, palpable spleen tip, macuolpapular rash over neck and abdomen.

Laboratory: Hb 6.2 to 10.3 Gms.%. WBC 5,720 to 17,200 with persistent neutrophilia (to 68%) and eosinophilia (to 16%). Sedimentation rate 111 to 128 mm. Platelets 28,000 to 110,000. Strongly positive cuff test. Prolonged bleeding time (10 minutes 30 seconds).

Discharged 9/9/53 - no definite diagnosis.

2nd Admission: 11/2/53.

Complaint: Occasional bleeding episodes from skin rash.

Physical: Open areas of eczema. Mild generalized lymphadenopathy. Grade I precordial systolic murmur. Spleen tip palpable. Liver 4 cm. below RCM.

Laboratory: Findings essentially as before. Skin biopsy negative.

Discharged 11/28/53.

3rd Admission: 12/8/53. Expired 12/8/53.

Complaint: Two day history of vomiting.

Physical: T 102^4 R 64. Old and fresh

petechiae over entire body. Moderate resistance to neck flexion. Spleen palpable; liver 6-8 cm. below RCM. Patient expired shortly after admission.

Autopsy: Thymus hemorrhagic. Spleen slightly enlarged (25 Gms.) and liver enlarged (360 Gms.). The kidneys showed petechial hemorrhages in the pelves and were enlarged (30 Gms.).

Microscopically the small arteries and capillaries in almost every organ showed occlusive material which was usually homogeneous, refractile, and deeply eosinophilic. Occasionally it appeared somewhat granular or laminated and was noted to contain red cells and leukocytes. Material of similar appearance was also noted in several vessels to lie beneath the endothelium and occasionally to protrude into the lumen of the vessel. These "hyaline thrombi" had the usual tinctorial features of fibrinoid. They were most numerous in the heart section. but were also present in almost every other organ including the lungs and the liver sinusoids. The presence of this material was often associated with a marked endothelial proliferation but there was a striking absence of inflammatory reaction.

Table I summarizes the incidence of fibrinoid lesions in rabbits following the administration of meningococcal endotoxin alone or in conjunction with the acidic polymer Liquoid. In addition to these regularly occurring lesions, hemorrhage and necrosis of the thymus and the adrenals also occurs. The latter lesion, however, has been observed only when endotoxin has been given in association with Liquoid, and in a higher percentage of the animals when this polymer is given simultaneously with endotoxin. The occurrence of thymic hemorrhage and necrosis is often associated with more severe renal, splenic, and cardiac lesions, but adrenal hemorrhage has been observed in occasional animals in which other lesions were absent.

The similarity of the renal lesion produced by Liquoid to the "wire loops" of lupus erythematosus has been observed previously by Hausman and Dreyfus[10] who gave large amounts of the polymer intravenously to rabbits. Of interest in this connection are the reports by Inderbitzen[11,12] concerning the production of lupus erythematosus cells by the addition of Liquoid or PVAS to suspensions of normal human

Table I

THE INCIDENCE OF FIBRINOID FOLLOWING I.V. MENINGOCOCCAL TOXIN OR MENINGOCOCCAL TOXIN PLUS LIQUOID

Method	Number of animals	Presence of fibrinoid				
		Kidney	Spleen	Heart	Liver	Lung
2cc of 1:80 toxin x 1	50	4%	10%	12%	16%	16%
2cc of 1:80 toxin x 2	27	63%	67%	60%	74%	33%
2cc of 1:80 or 1:1000 toxin and 8mg liquoid	40	60%	70%	55%	28%	75%

leukocytes. Hausman and Dreyfus, in attempting to confirm this observation, noted the occurrence of the renal glomerular capillary lesions and confirmed "in part" Inderbitzen's observations, but we have been unable to produce a "typical" LE cell by the methods outlined in Inderbitzen's reports.

DISCUSSION

A detailed study of the lesions in each of these cases shows vascular alterations characterized by fibrinoid which is frequently associated with areas of hemorrhage and necrosis. The severity of these lesions and the organ or system involved is so variable that in some cases the morphologic diagnosis would depend upon which particular slide one examined. In case I, for example, the fibrinoid is confined to the heart and is associated with the typical alterations of acute rheumatic carditis. In the case of scleroderma, however, even though the fibrinoid is largely confined to the renal afferent arterioles and glomerular capillaries, the manner of its deposition is such as to be compatible with either lupus erythematosus or accelerated hypertension, as well as scleroderma. In each of the other cases the lesions are so widespread and variable that changes characteristic of more than one type may be found.

Thrombotic thrombocytopenic purpura also appears to fall into this group of diseases. Since the report by Baehr, Klemperer and Schifrin[13] it has been assumed by most investigators that the occlusive material in this disease is composed of platelets, but the studies by Gore[14] showed a "prethrombotic" lesion consisting of the presence of a homogeneous acidophilic material beneath the endothelium of the vessels. This material, assumed to be the initial lesion of the disease, gave the usual staining reactions of fibrinoid. Orbison[15] has since shown that primary vascular damage characterized by the presence of fibrinoid and the formation of aneurysms occurs at the arteriocapillary junction in thrombotic thrombocytopenic purpura. In addition to these observations, it should also be emphasized that no specific tissue stain for platelets exists at present time, and the assumption that this material is platelets therefore appears to be unjustified, although it is not improbable that platelets might become enmeshed in the fibrinoid material within the vessels.

The lesions of the generalized Shwartzman phenomenon appear similar, in many respects, to those which have been described in the cases summarized above. In the rabbit, however, the fibrinoid material is more often associated with gross areas of hemorrhage and necrosis, which are particularly prominent in the spleen and kidneys. This difference is probably related to the rapidity of development of the experimental lesions in contrast to the slower, progressive nature of the human disease processes. One other point of difference is the lack of acute cellular reaction about most fibrinoid lesions involving the arteries of the experimental animals. In the human material which has been presented, however, finrinoid lesions with and without cellular reaction have been demonstrated in the same case, suggesting that this factor may be of secondary importance. The basic morphologic similarity of the lesions and the identical staining features of the fibrinoid material suggest a similar mode of origin of the fibrinoid in both the human and experimental lesions, and it is the additional factors such as the presence or absence of cellular reaction and the major localization of the lesions which permit their separation into individual disease processes. These factors also suggest that, although the derivation of the fibrinoid material may be the same, different etiologic agents may be involved in its production and deposition.

Studies of the generalized Shwartzman phenomenon have indicated that the fibrinoid which accumulates within various organs and tissues is probably

derived from the circulating blood[7,8,9] and that fibrinogen may be involved in its formation.[8,16] Its morphologic and tinctorial features are identical to those described by Altshuler and Angevine for fibrinoid occurring in the so-called "collagen diseases." These observations strongly suggest that the basic alteration in these diseases is not a change in collagen or connective tissue. Therefore, it seems more reasonable to refer to them as systemic fibrinoid diseases, at least until more specific details of the various etiologic factors concerned are known.

The factors necessary for the production and localization of this material in the human are now known at present. Experimentally, however, changes in the circulating blood appear to be involved in its production, and structural or functional vascular alterations appear to be important in its localization. These factors, and the close similarity between the human and experimental lesions suggest that a more detailed investigation of abnormalities in blood coagulation and serum proteins might provide further information concerning the pathogenesis of these diseases.

SUMMARY AND CONCLUSIONS

Six cases of systemic fibrinoid disease have been presented. The varied lesions occurring in these diseases, and their morphologic similarity to one another and to the lesions occurring in the generalized Shwartzman phenomenon have been discussed and illustrated. Factors important in the experimental production and localization of fibrinoid lesions appear to be an alteration in or production of abnormal serum proteins and structural or functional alterations in the endothelium of the cardiovascular system. It is suggested that similar mechanisms may operate in the pathogenesis of the human diseases which have been discussed.

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II. MEDICAL SCHOOL NEWS

Coming Events

February 7 - 12	Continuation Course in Neurology for General Physicians and Specialists
February 9	J. B. Johnston Lectureship; "Cerebral Circulation and Oxygen Consumption;" Dr. Seymour S. Kety, National Institute of Neurological Diseases and Blindness, U. S. Public Health Service, Bethesda, Maryland; Mayo Auditorium; 8:15 p.m.
February 14 - 16	Continuation Course in Internal Medicine for Internists
February 17	Special Lecture; "The Effects of Disease on History;" Professor John Fulton, Yale University; Weyerhaeuser Room, Minnesota Historical Society Building, St. Paul; 2:00 p.m. (Tea hour following lecture)
February 17 - 19	Continuation Course in Cancer Detection for General Physicians
Feb. 28 - March 2	Continuation Course in Clinical Hematology for General Physicians and Internists

* * *

Continuation Course

The University of Minnesota and the Minnesota Division of the American Cancer Society will join in the presentation of a continuation course in Cancer Detection for General Physicians from February 17 to 19, 1955, at the Center for Continuation Study. This year's program will consist of a series of lectures and informal discussions concerning the cancer problem and cancer detection techniques. Examination procedures will be taken up in detail.

* * *

Faculty News

Dr. F. H. Van Bergen, Associate Professor and Director, Division of Anesthesiology, attended the annual meeting Association of University Anesthetists which was held at Duke University, Durham, North Carolina, on January 22 and 23. He participated in a panel discussion on "Muscle Relaxants and the Role They Play in Anesthetic Mortality and Morbidity." Next year's meeting of this association will be held at the University of Minnesota and the Mayo Clinic.

Dr. Raymond N. Bieter, Professor and Head, Department of Pharmacology, attended a meeting of the Committee on Drug Addiction and Narcotics of the National Research Council which was held at the U.S.P.H.S. Hospital, Lexington, Kentucky, on January 21 and 22.

Dr. K. W. Stenstrom, Professor of Biophysics and Director of Radiation Therapy, has recently received a letter from Dr. Donn Mosser, Instructor of Radiation Therapy, on leave under the auspices of the American Cancer Society for study. Dr. Mosser is currently in Manchester, England, and will continue to study there until April 1. He will spend April in Edinburgh, Scotland, and in May he will go to Paris and later to Copenhagen. He plans to return next fall.

Several members of the faculty of the Department of Bacteriology and Immunology attended the recent Biology of Poliomyelitis Conference which was held at the New York Academy of Sciences from January 19 to 21. Dr. J. T. Syverton, Professor and

Head, was a Session Chairman and Doctors W. F. Scherer, K. T. Brunner, J. O'H. Tobin, and George Gifford also attended. The Department of Bacteriology was recently host to Dr. Jean Vieuchange, Pasteur Institute, Paris, France, and Dr. Carl Eklund, Rocky Mountain Laboratory, Hamilton, Montana.

Dr. Robert I. Wise, Director of the Bacteriology Laboratory and Assistant Professor of Medicine, participated in a postgraduate course in Obstetrics and Gynecology which was given at the School of Medicine, University of Nebraska, in Omaha on January 20 and 21. The course was given under the direction of Dr. Roy G. Holly, a former member of our faculty in obstetrics and gynecology who now heads that department at the Nebraska institution.

Dr. Newell R. Ziegler, Director of the Blood Bank, was recently guest speaker at the meeting of the Renville-Redwood County Medical Society which was held at Redwood Falls on December 21.

* * *

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III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

February 7 - 12, 1955

Monday, February 7

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 - 12:30 Physical Medicine and Rehabilitation Staff Seminar; Speech Problems of the Cerebral Palsied; Gertrude Eggert; Heart Hospital Theater.

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

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

12:30 - 1:30 Physiology Seminar; Ureteral Response to Specific Pharmacologic Agents; Donald Bravick; 214 Millard Hall.

1:00 - 2:00 Roentgenology-Surgical-Pathological Conference; Paul Lober and L. G. Rigler; Todd Amphitheater, U. H.

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

1:30 - 3:30 Dermatology Hospital Rounds; H. E. Michelson and Staff; Dermatology-Histopathology Room, C-394 Mayo Memorial.

4:00 - 6:00 Anesthesiology Conference; F. H. Van Bergen and Staff; Todd Amphitheater, U. H.

4:30 - Public Health Seminar; Opportunities in Medicine for Parent Education; Reynold A. Jensen; Room 100, Mayo Memorial.

4:30 - Pediatric-Medicine Infectious Disease Rounds; Station 33, U. H.

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

Ancker Hospital

8:00 - 9:00 Pediatrics Contagion Rounds; Richard Lein; Contagion 5.

8:30 - 10:30 Medical and Surgical Chest Conference; Dr. Gehlen and Staff; Auditorium.

9:30 - 12:00 Visiting Staff Rounds.

10:00 - 12:00 Surgery Grand Ward Rounds; Begin Floor E4.

11:00 - 12:00 Pediatric Rounds; Harry Orme; Contagion 1.

12:30 - 2:30 Surgery Out-Patient Clinic; Room 8.

2:00 - 3:00 Routine EKG Interpretation; Dr. Sommers and House Staff; Medical Record Library.

Monday, February 7 (Cont.)

Ancker Hospital (Cont.)

2:30 - 3:00 Discussion of Problem Case; Auditorium.

3:00 - 4:00 Surgery Journal Club; Classroom.

3:00 - 4:00 Lectures on Electrocardiography; Ben Sommers; Auditorium.

4:00 - 5:00 Medical Clerk Journal Club; Auditorium.

Minneapolis General Hospital

10:30 - 12:00 Medicine Rounds; Thomas Lowry; Station 31.

11:00 - Pediatric Case Discussions; Erling Platou; Station 4.

11:00 - Orthopedic and Fracture Rounds; Drs. John Moe and Arthur Zierold; Station 20.

12:30 - Surgery Grand Rounds; Dr. Zierold, Station 21.

1:30 - 2:30 Tuberculosis Conference; J. A. Myers; Station 8.

2:00 - Pediatrics Rounds; William Krivit; Stations 4,5, & 6.

Veterans Administration Hospital

9:30 - Infectious Disease Rounds; Drs. Hall, Zinnemann, and J. Brown.

1:30 - Cardiac Conference; Drs. Smith, Berman, Hoseth, Simonson, Tamlyn, and Farquhar; Conference Room, Bldg. I; Rounds immediately following conference.

Tuesday, February 8

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Pediatric Conference; Samuel Feinberg, Irvine McQuarrie and Staffs; Eustis Amphitheater, U. H.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 104 Jackson Hall.

12:30 - Physiological Chemistry Seminar; Aldosterone; Lester Lansky; 214 Millard Hall.

12:30 - Bacteriology and Immunology Seminar; Bacterial Mutation, Spontaneous and Induced. Experimental Methods of Luria and Delbrück, Newcombe, and Lederberg Used to Demonstrate the Occurrence of Mutation; J. Melnykowycz; 1050 Mayo Memorial.

12:30 - Anatomy Seminar; The Enzymatic Machinery of the Thyroid Hormone; Carl Friz; 226 Jackson Hall.

3:30 - General Physiology Seminar; 323 Zoology Building.

3:30 - Pediatric Seminar; Persistent Common Atrioventricular Canal; Brian Kiely; 1450 Mayo Memorial.

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

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

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

5:00 - 6:00 X-ray Conference; Presentation of Cases from Mount Sinai Hospital; Drs. Westley and Springer; Eustis Amphitheater, U. H.

Tuesday, February 8 (Cont.)

Ancker Hospital

8:00 - 9:00 Pediatric Rounds; Dale Cumming; Contagion 1.
9:00 - 10:30 Visiting Staff Rounds.
9:00 - 12:00 Practical Diagnostic Clinic; Harry Orme; Out-Patient Department.
11:00 - 12:00 Medical X-ray Conference; J. R. Aurelius; Auditorium.
2:30 - 4:00 Routine EKG Interpretations; Resident Staff.
4:00 - 5:00 Medical-Pathological Conference; W. F. Mazzitello, Auditorium.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Elizabeth Lowry and A. Bridge; Station 5.
10:00 - Psychiatry Grand Rounds; R. W. Anderson, Station 3.
10:00 - Cardiac Rounds; Paul F. Dwan; Classroom, Station 4.
11:00 - 12:00 Medicine-Surgery Conference; Classroom, Station 8.
12:30 - 2:30 Dermatology Rounds on Clinic; Carl W. Laymon and Staff.
12:30 - ECG Conference; Boyd Thomes and Staff; 302 Harrington Hall.
1:00 - Tumor Clinic; Drs. Eder, Coe, and Lipschultz; Classroom.
3:30 - Pediatric-Psychiatry Rounds; Jack Wallinga; Station 4.

Veterans Administration Hospital

7:30 - Anesthesiology Conference; Surgical Conference Room, Bldg. 43.
8:30 - Hematology Rounds; Drs. Hagen and Wexler.
8:30 - Surgery Journal Club; Conference Room, Bldg. I.
9:30 - Surgery-Pathology Conference; Conference Room, Bldg. I.
10:30 - Surgery-Tumor Conference; D. Ferguson and J. Jorgens.
1:00 - Review of Pathology, Pulmonary Tuberculosis; Conference Room, Bldg. I.
1:30 - Combined Medical-Surgical Chest Conference; Conference Room, Bldg. I.
2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III.
4:00 - Thoracic Surgical Problems; Conference Room, Bldg. I.
5:30 - Physiology Seminar; Surgical Conference Room, Bldg. 43.

Wednesday, February 9

Medical School and University Hospitals

11:00 - 12:00 Pathology-Medicine-Surgery-Pediatrics Conference; Todd Amphitheater, U. H.
12:30 - 1:20 Radio-Isotope Seminar; Betatron Room in Cobalt Underground Section, U. H.
1:00 - 2:00 Dermatology Clinical Seminar; F. W. Lynch; 300 North Clinic.

Wednesday, February 9 (Cont.)

Medical School and University Hospitals (Cont.)

1:30 - 3:00 Pediatrics Allergy Clinic; Albert V. Stoesser and Lloyd Nelson; W-211, U. H.

3:30 - 4:30 Dermatology-Pharmacology Seminar; 3rd Floor Conference Room, Heart Hospital.

4:30 - 5:50 Dermatology-Infectious Disease Seminar; 3rd Floor, Conference Room, Heart Hospital.

5:00 - 6:00 Radiology Residents Lectures; Fluoroscopy of Pharynx and Larynx; Kent F. Westley; Todd Amphitheater, U. H.

5:00 - 5:50 Urological-Pathological Conference; C. D. Creevy and Staff; A503, Mayo Memorial.

5:10 - 6:10 Endocrine Seminar; 271 Lyon Laboratories.

5:30 - 7:30 Dermatology Journal Club and Discussion Group; Hospital Dining Room.

7:30 - 9:30 Dermatology Seminar; Review of Interesting Slides of the Week; Robert W. Goltz; Todd Amphitheater, U. H.

*8:15 p.m. J. B. Johnston Lectureship; "Cerebral Circulation and Oxygen Consumption;" Dr. Seymour S. Kety, National Institute of Neurological Diseases and Blindness, U. S. Public Health Service, Bethesda, Maryland; Mayo Auditorium.

Ancker Hospital

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

11:00 - 12:00 Pediatric and Contagion Rounds; Harry Orme; Contagion 1.

11:00 - 12:00 Medicine Resident Rounds; W. F. Mazzitello.

3:00 - 5:00 Infectious Disease Rounds; Auditorium.

Minneapolis General Hospital

8:30 - 9:30 Obstetrical and Gynecological Grand Rounds; William P. Sadler and Staff; Station 30.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station 11.

11:00 - Pediatric Rounds; Erling Platou and Richard Raile; Station 6.

12:30 - Pediatrics Staff Meeting; Classroom, Station 4.

Veterans Administration Hospital

8:30 - 10:00 Orthopedic X-ray Conference; E. T. Evans and Staff; Surgical Conference Room, Bldg. 43.

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

9:00 - Gastro-Intestinal Rounds; Drs. Wilson, Zieve, Ferguson, Brakel, Swenson, Nesbitt and Sadoff.

10:30 - Psychosomatic Conference; C. K. Aldrich; 7th Floor, Bldg. 43.

12:30 - Medical Journal Club; Doctors' Dining Room.

12:30 - X-ray Conference; J. Jorgens; Conference Room, Bldg. I.

Wednesday, February 9 (Cont.)

Veterans Administration Hospital (Cont.)

1:30 - 3:00 Metabolic Disease Conference; Drs. Flink and Williams.

3:30 - Urology Pathology Slide Conference; Dr. Gleason; Conference Room, Bldg. I.

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

Thursday, February 10

Medical School and University Hospitals

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; Room 3.148 Mayo Memorial.

11:00 - 12:00 Cancer Clinic; K. Stenstrom, B. Zimmermann; Todd Amphitheater, U. H.

12:30 - 1:55 Physiology Seminar 210; Transport; Selected Topics in Permeability; Nathan Lifson; 214 Millard Hall.

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

4:00 - 5:00 Anesthesiology Seminar; F. H. Van Bergen and Staff; Room 100, Mayo Memorial.

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

7:30 - 9:30 Physiology 211 Seminar; Selected Topics in Heart and Circulation: Hemodynamics; M. B. Visscher and Robert Evans; 271 Lyon Laboratories.

Ancker Hospital

9:00 - 10:00 Pediatric Contagion Rounds; Alexander Stewart; Contagion 5.

9:30 - 10:30 Medical Grand Rounds; Auditorium; Visiting Staff Rounds immediately following Grand Rounds.

11:00 - 12:00 Medicine Resident Rounds; W. F. Mazzitello.

2:00 - 3:00 Routine ECG Interpretation; Ben Sommers; Medical Record Library.

Minneapolis General Hospital

9:30 - Neurology Rounds; Heinz Bruhl; Station 4.

9:30 - Pediatric Contagion Rounds; R. B. Raile; Station 4.

10:00 - Psychiatry Grand Rounds; R. W. Anderson and Staff; Station 3.

11:30 - 12:30 Clinical Pathological Conference; John I. Coe; Classroom.

12:30 - 2:30 Dermatology Rounds and Clinic; Carl W. Laymon and Staff.

1:00 - Fracture X-ray Conference; Drs. Zierold and Moe; Classroom.

1:00 - House Staff Conference; Station 4.

Veterans Administration Hospital

8:00 - Experimental Surgery Laboratory Meeting; Conference Room, Bldg. I.

8:30 - Hematology Rounds; Drs. Hagen and Doe.

Thursday, February 10 (Cont.)

Veterans Administration Hospital (Cont.)

9:00 - Surgery Grand Rounds; Conference Room, Bldg. I.

9:00 - Surgery Ward Rounds; D. Ferguson and Staff; Ward 11.

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

1:00 - Infectious Disease Conference; Conference Room, Bldg. I. (Rounds immediately following conference.)

Friday, February 11

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.

11:00 - 12:00 Vascular Rounds; Davitt Felder and Staff Members from the Departments of Medicine, Surgery, Physical Medicine, and Dermatology; Eustis Amphitheater, U. H.

11:45 - 12:50 University of Minnesota Hospitals Medical Staff Meeting; Nervous System Intoxications; Maynard Cohen and Ian Brown; Powell Hall Amphitheater.

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

1:00 - 2:00 Physiology Seminar 212; Selected Topics in Respiration: Respiratory and Circulatory Effects of Hypothermia; E. B. Brown; 214 Millard Hall.

1:30 - 2:30 Dermatology Grand Rounds; Presentation of Cases from Grouped Hospitals (University, Ancker, General and Veterans) and Private Offices; H. E. Michelson and Staff; Eustis Amphitheater, U. H.

2:30 - 4:00 Dermatology Hospital Rounds; H. E. Michelson and Staff; Begin at Dermatological Histopathology Room, C-394 Mayo Memorial.

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

3:30 - 4:30 Dermatology-Physiology Seminar; 3rd Floor Conference Room, Heart Hospital.

4:00 - 5:00 Physiology Seminar 213; Selected Topics in Advanced Neurophysiology: Role of the Vestibular Apparatus and the Cerebellum in the Extrapyramidal Motor Activity; Werner Koella; 129 Millard Hall.

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

5:00 - Urological Seminar and X-ray Conference; A503, Mayo Memorial.

Ancker Hospital

8:00 - 9:00 Pediatric Rounds; Charles Steinberg; Contagion 1.

10:30 - 11:30 Pediatric Contagion Rounds; Richard Smith; Contagion 1.

11:00 - 12:00 Contagion Rounds; Harry Orme; Contagion 5.

Friday, February 11 (Cont.)

Ancker Hospital (Cont.)

2:00 - 3:00 Routine EKG Interpretation; Resident Staff.

3:00 - 4:00 Medical-Surgical-Pathological Conference; Auditorium.

4:00 - 5:00 Medical Journal Club; Conference Room, E5.

4:00 - 5:00 X-ray Surgery Conference; Auditorium.

Minneapolis General Hospital

10:00 - Otolaryngology Conference; Robert A. Priest; Large Classroom.

10:30 - Pediatric Surgical Conference; Tague Chisholm and B. Spencer; Classroom, Station 4.

12:00 - Surgery-Pathology Conference; Drs. Zierold and Coe; Classroom.

1:00 - 3:00 Clinical-Medical Conference; Thomas Lowry; Classroom, Station 8.

Veterans Administration Hospital

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

11:00 - 12:30 Psychiatry Case Conference; Werner Simon; Psychiatry Department, VA Hospital Annex.

12:30 - Urology X-ray Conference; X-ray Department.

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

2:00 - Pathology Slide Conference; E. T. Bell; Conference Room, Bldg. I.

Saturday, February 12

Medical School and University Hospitals

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

9:00 - 9:30 Pediatric Grand Rounds; Eustis Amphitheater, U. H.

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

9:15 - 10:00 Surgery-Roentgenology Conference; Alexander R. Margulis, 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 Rounds; J. L. McKelvey and Staff; Station 44, U. H.

10:00 - 12:00 Otolaryngology Seminar on Current Literature; L. R. Boies and Staff; Todd Memorial Room, A-675, Mayo Memorial.

Ancker Hospital

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

9:30 - 11:00 Medicine Grand Ward Rounds; W. F. Mazzitello.

11:00 - 12:00 Medical Clerk Case Conference; W. F. Mazzitello.

Saturday, February 12 (Cont.)

Minneapolis General Hospital

8:00 - Urology Staff Conference; T. H. Sweetser; Main Classroom.

9:00 - Psychiatry Grand Rounds; R. W. Anderson; Station 3.

9:30 - Pediatrics Rounds on all Stations; R. B. Raile.

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

Veterans Administration Hospital

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

8:30 - Medical X-ray Conference; Conference Room, Bldg. I.

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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.