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

Endocarditis

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William A. O'Brien, M.D.

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GREETINGS TO THE HOSPITAL STAFF

With the beginning of another academic year I welcome this opportunity to extend most cordial greetings to the members of our University Hospitals Staff. I wish that it were possible to greet each one of you personally but even more than that, I want to express to the staff my sincere admiration and commendation for the superb service which you have rendered to the hundreds of poliomyelitis patients who have been in this hospital over the past several months.

Your devotion to duty, your ceaseless and tireless efforts to give every patient the best of medical care, your personal sacrifices and your disregard of the dangers of infection when necessary exemplify the ideals of service for which our medical and allied professions have long been esteemed. To the physicians, the nurses, the physical therapy technicians and the administrative and the service staffs who have carried on so nobly during this emergency I extend the heartfelt appreciation of the Medical School and the University. We all take pride in your achievements.

With the opening of this new college year the University has a fantastic enrolment and is faced with an educational task far beyond what anyone anticipated even a few months ago. In the Medical School increased teaching loads are primarily in graduate and postgraduate programs; although the opportunities for professional training in the medical field have been expanded by new courses of instruction in Hospital Administration and in Occupational Therapy.

The result of all this is even greater crowding of facilities which were already inadequate. To carry on effectively under these conditions will require ingenuity, patience and cooperation. We all will welcome the day when the new facilities in the projected Mayo Memorial will become available. Until that time, however, I am confident that we will continue to make progress and conduct the type of program for which our Medical School and University Hospitals are becoming widely and favorably known.

Harold S. Diehl
Dean

October 1, 1946

I:

UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
CALENDAR OF EVENTS
 October 5 - October 11, 1946

No. 127Saturday, October 5

- 7:45 - 8:50 Orthopedics Conference; Wallace H. Cole and Staff; Station 21, U. H.
- 9:00 - 9:50 Surgery-Roentgenology Conference; O. H. Wangensteen, L. G. Rigler, and Staff; Todd Amphitheater, U. H.
- 9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; M-515 U. H.
- 10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U. H.
- 11:30 - Anatomy Seminar; Effects of Androgen upon Reproductive Organs of Normal and Castrated Fetuses with a Note on Adrenalectomy; L. J. Wells; and Experimental Evidence for Secretion of Urine by the Fetal Kidney; L. J. Wells and G. T. Evans.

Monday, October 7

- 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; Interns Quarters, U. H.
- 12:15 - 1:15 Obstetrics and Gynecology Journal Club; M-435 U. H.
- 12:30 - 1:20 Pathology Seminar; Amebic Abscess of the Liver; Dr. W. B. Martin, 104 I. A.
- 12:30 - 1:20 Physiology Seminar; 214 M. H.

Tuesday, October 8

- 9:00 - 9:50 Roentgenology-Pediatrics Conference; L. G. Rigler, I. McQuarrie and Staff; Eustis Amphitheater, U. H.
- 12:30 - 1:20 Pathology Conference; Autopsies; Pathology Staff; 102 I. A.
- 2:00 - 3:00 Dermatology and Syphilology; H. E. Michelson and Staff; Veterans' Hospital Bldg. III.
- 3:15 - 4:15 Gynecology Chart Conference; J. L. McKelvey and Staff; Station 54, U. H.
- 3:45 - 5:00 Pediatric Staff Rounds; I. McQuarrie and Staff; W-205 U. H.
- 4:00 - 4:50 Surgery-Physiology Conference; Drs. David State and Richard Varco; Eustis Amphitheater.
- 5:00 - 5:50 Roentgenology Diagnosis Conference; Dr. Solvig Berg, et al; M-515, U. H.

Wednesday, October 9

- 8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-515 U. H.
- 9:00 - 10:50 Neuropsychiatry Seminar; Staff; Station 60 Lounge; U. H.
- 11:00 - 11:50 Pathology-Medicine-Surgery Conference; Cerebral Hemorrhage; E. T. Bell, C. J. Watson, O. H. Wangensteen and Staff; Todd Amphitheater, U. H.
- 12:30 - 1:20 Physiological Chemistry Journal Club; Staff; 116 M. H.
- 4:00 - 6:00 Medicine and Pediatrics Infectious Disease Rounds; W-205 U. H.

Thursday, October 10

- 9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; Todd Amphitheater; U. H.
- 12:15 - 1:15 Pediatrics Seminar; Irvine McQuarrie and Staff; Todd Amphitheater, U. H.
- 12:30 - 1:20 Physiological Chemistry; Karl Sollner; 129 M. H.
- 4:30 - 5:20 Ophthalmology Ward Rounds; Erling Hansen and Staff; E-534, U. H.
- 4:30 - 5:20 Bacteriology Seminar; 214 M. H.
- 5:00 - 5:50 Roentgenology Seminar; Drs. Chauncey Borman and L. A. Nash; M-515, U. H.

Friday, October 11

- 9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U.H.
- 9:00 - 9:50 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater.
- 10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.
- 10:30 - 12:20 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Otolaryngology Department; U. H.
- 11:50 - 1:15 University of Minnesota Hospitals General Staff Meeting; Annual Report; R. M. Amberg; New Powell Hall Amphitheater.
- 1:00 - 2:00 Dermatology and Syphilology; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U. H.
- 1:30 - 2:20 Roentgenology-Neurosurgery Conference; H. O. Peterson, W. T. Peyton, and Staff; Todd Amphitheater, U. H.

II. ATTENDING STAFF -- 1946-1947

ADMINISTRATION

Diehl, Harold S.,
Dean of Medical Sciences
Weaver, Myron,
Assistant Dean of Medical Sciences
Amberg, Ray M., Superintendent and Professor of Hospital Administration
Gilman, Gertrude, Assistant Superintendent and Director of Hospital Admissions
Klein, William K., Assistant Superintendent and Director of Hospital Service and Supply
Harrington, Ethel, Director of Volunteer Services
Carlson, Alice, Medical Records Librarian
Corliss, Ione, Out-Patient Nursing Supervisor
Halverson, Lucile, Acting Superintendent of Nurses
Hamilton, Hiram F., General Mechanic Foreman
Kurtzman, Mrs. Dorothy, Assistant Professor of Nursing and Superintendent of Nursing Projects
McHugh, Margaret, Hospital Housekeeper
Money, Frances M., Director of Social Service and Associate Professor
Nesbitt, Zula, P., Accountant
Thomas, Gertrude, Director of Hospital Nutrition Service and Assistant Professor

ANESTHESIA

Knight, Ralph T., Clinical Professor and Director of Anesthesia
Baird, Joseph, Clinical Associate Professor
Friend, A. William, Clinical Assistant Professor

Fellows

Adams, Marvin E.
Bailey, Jesse E.
Cohen, Ellis N.
Conner, Edward D.
Gordon, Ian E.
Johnson, Ward R.
Jones, Charles F.
Peterson, Carl W.
McNeil, John J.
Stevenson, Richard H.
Wilsey, David B.

Anderson, William H. (V.A.)
Lomas, Woodrow E. "
Van Bergen, Frederick H. "
Wesolowski, Stanley P. "
Wiggins, James K. "

DENTISTRY

Waldron, C. W., Professor Oral Surgery
Worman, Harold G., Associate Professor Dentistry

Staff Associates

Brussell, Irvin
Christensen, R. O.
Cohen, J. T.
Dostal, Donald W.
Freeman, Joseph
Peterson, R. G.
Scholtis, A. J.
Small, E. W.
Woldum, L. H.

X-RAY AND RADIATION THERAPY

Rigler, Leo G., Professor and Head of Radiology and Physical Therapy
Stenstrom, Wilhelm K., Professor of Biophysics
Peterson, Harold O., Associate Clinical Professor of Radiology
Stauffer, Herbert, Assistant Professor and Roentgenologist

Fellows

Ahern, Eugene
Bergh, Solveig
Blank, Samuel
Macklin, W. E.
McGraw, J. P.
Mixer, Harry
Summers, J. S.

Fink, Daniel, Fellow - (V.A.)
Leighton, William, Fellow - (V.A.)

Friedman, Jack, Fellow - Ancker Hosp.

Peterson, Donald, Fellow - Minneapolis General Hospital

DERMATOLOGY

Michaelson, Henry, Professor and Director
 Lynch, Francis, Associate Professor of
 Dermatology
 Epstein, Stephan, Clinical Associate
 Professor
 Laymon, Carl, Clinical Associate Professor
 Cummings, Harry, Assistant Professor
 Freeman, Charles D., Clinical Assistant
 Professor
 Madden, John, Clinical Assistant Professor
 Rusten, Elmer, Clinical Assistant
 Professor
 Ceder, Elmer, Clinical Instructor
 Kendall, Rodney F., Clinical Instructor
 Freeman, Charles D. Jr., Medical Fellow
 (General Hospital)
 Haserick, John, Medical Fellow
 Hill, Elmer, Medical Fellow (General Hosp.)
 Steves, Richard J., Medical Fellow

Fellows

Barthell, John
 Fisher, Isodore
 Hauser, George W.
 Hurst, Harold
 Murphy, Robert
 Ravits, Harold
 Sevenants, John

INTERNSMEDICINE

Breitenbucher, Robert
 Hubler, Willis
 Peterson, Ralph
 Quist, Henry
 Rames, Eugene
 Ross, Neff
 Shaffer, James
 Tarail, Robert

SURGERY

Aldrich, Alvin
 Campbell, Gilbert
 Ide, Arthur
 Maluf, Noble
 Marshall, Clark
 Maxwell, Walter
 Moore, George
 Sako, Yoshio

PEDIATRICS

Aldrich, Robert*
 Erickson, Lawrence*
 Galligan, John
 Greig, Jack
 Lienke, Roger
 Perkins, Georgia
 Siegel, Sheldon

OBSTETRICS AND GYNECOLOGY

Barno, Alex
 Hakanson, Erick

OTOLARYNGOLOGY

Wittels, Theodore*

OPHTHALMOLOGY

Peterson, John*

DENTISTRY

Baronofsky, Harold

LABORATORY

Evans, Gerald T., Director Hospital
 Laboratories and School of Medical
 Technology
 Larson, Evrel, A., Instructor Medicine
 and Associate Scientist
 Marwin, Richard M., Bacteriologist
 Sundberg, Dorothy, Instructor of Anatomy

Technologists

Daughenbaugh, Elaine
 Ertl, Evelyn, Mrs.
 Harvey, Mrs. Delores
 Hovde, Ruth
 Lamberg, Harriet
 Rausch, Verna
 Rietz, Jane

PATHOLOGY

Bell, E. T., Head and Professor
 McCartney, James, Assoc. Professor
 Hebbel, Robert, Assistant Professor

*Fellows serving as interns.

MEDICINE

Watson, Cecil J., Head and Professor
 Spink, Wesley W., Professor
 Aagaard, George, Asst. Professor
 Hoffbauer, Frederick, Asst. Professor
 Lowry, Thomas, Clinical Asst. Professor
 Hagen, Paul, Clinical Instructor
 Hall, Wendell, Clinical Instructor
 Horns, Howard, Clinical Instructor

FellowsIn-Patient

Cullen, Richard C.
 Greenberg, Albert
 Thomes, A. Boyd

Out-Patient

Jacobson, Wyman C.
 Johnson, John
 Koff, Sheldon
 LaBree, John W.
 Sborov, Victor
 Berris, Barnet

Laboratory

Myre, James G.

Research

Hayes, Elmer R.
 Wikoff, Howard W.

Pathology

Green, Robert

Neurology

Schulze, William

Chas. T. Miller Hospital

Minckler, John E.

Northwestern Hospital

Keil, Marcus A.

St. Barnabas Hospital

Chalmers, Hugh J.

Veterans Hospital

Blumberg, Henry B.
 Borden, Craig
 Constable, V. Pepper, Jr.
 Crosbie, Stanley B.
 Falk, Abraham
 Forsgren, Arthur
 Gullord, Edward G.
 Haas, William R.
 Hill, Earl
 Kallsen, Robert A.

Veterans Hospital (Cont.)

Masler, Sherman
 Mooney, Robert D.
 Poppe, Frederick P.
 Skouge, Oren T.
 Soucheray, Phillip H.
 Wilson, Russell H.
 Wishart, John H.
 Zieve, Leslie

NEUROLOGY & PSYCHIATRY

Hastings, Donald W., Professor of Neuro-
 psychiatry and Head
 Baker, A. B., Associate Professor of
 Neurology and Director, Division of
 Neurology
 Schiele, Burtrum C., Associate Pro-
 fessor of Neuropsychiatry

Hathaway, Starke R., Chief Clinical
 Psychologist
 Meller, Robert, Clinical Asst. Professor
 Dumas, Alexander, Clinical Asst. Pro-
 fessor
 Challman, S. Allan, Clinical Asst.
 Professor
 Henry, Charlotte, Chief Social Worker

Fellows

Cranston, Robert W.
 Havik, John E. T.
 Kiesler, Frank, Jr.
 Kuiper, Klaire Van Zanten
 Levin, Jules
 Miller, Zondal R.
 Resch, Joseph
 Schumacher, John W.
 Selvig, Howard S.
 Wesley, Louis W., Teaching Assistant

Caplan, Leslie (V.A.)
 Carpentieri, Joseph "
 Cowan, George M. "
 Geib, Marvin J. "
 Joslin, Blocker H. "
 Parker, Benjamin F. "
 Raths, Otto N., Jr. "
 Rowe, Clarence J., Jr. "
 Rowland, Robert T. "

CHILD PSYCHIATRY

Jensen, Reynold, Associate Professor
of Pediatrics and Psychiatry
Stenswick, Ellsworth, Speech Clinician
Arkola, Audrey, Clinical Psychologist
and Instructor
Kramer, Jo Ann, Psychiatric Social
Worker and Instructor

OPHTHALMOLOGY & OTOLARYNGOLOGYOPHTHALMOLOGY

Hansen, Erling, Clinical Professor
and Director of Ophthalmology
Hoffman, Walter L. Hoffman, Clinical
Professor
Stanford, Charles E., Clinical Pro-
fessor
Tracht, Robert, Clinical Instructor
and Refractionist

Fellows

Dolmage, George
Bushard, Wilfred
Peterson, John H.

Plotke, Harry L. (V.A.)
Watson, Richard "

Keppen, Ford F. (Ancker)

Hammerstad, Lynn M. (Mpls. General)
Lindblom, Alton E.

Lehman, S. John (Chas. T. Miller)
Cochrane, Byron B. "
Sternor, Donald "

Christensen, Llewellyn E., (Jr. V.A.)
Cundy, Donald T. "
Shaw, Howard A. "

OTOLARYNGOLOGY

Boies, L. R., Professor and Head
Bryant, Frank L., Clinical Assistant
Professor
Hilger, Jerome A., Clinical Assistant
Professor
Priest, Robert E., Clinical Assistant
Professor
Hochfilzer, John H., Clinical Assist-
and Professor
Tangen, George M., Clinical Instructor

Fellows

Carris, James V. (V.A.)
Sjoding, J. Donald, "
Eyjolfsson, Gudmundur (U. H.)
Smith, Graham G. "
Wittels, Theodore S. "
Ulvestad, Harold S. (Ancker)
Goltz, Neill F., (Minneapolis General)

ORTHOPEDICS

Cole, Wallace H., Professor of Ortho-
pedics and Chief
Hall, Harry B., Asst. Professor
Nydahl, Malvin J., Instructor

Fellows

Beer, John J.
Burnham, Wesley (V.A.)
Elliott, Robert B. (Mayo Clinic)
Foreman, Forrest H. (Gillette Hospital)
Lannin, Donald R.
Lindsay, Douglas T. (Shriners Hospital)

PHYSICAL MEDICINE

Knapp, Miland E., Chief
Erickson, Don J.
Kottke, Frederic J.
Maschmeyer, Joseph E.

PEDIATRICS

McQuarrie, Irvine, Professor and Head
Adams, John, Professor
Jensen, Reynold, Professor
Stoesser, Albert, Clinical Professor
of Allergy
Ziegler, Mildred, Assistant Professor
Bosma, James, Instructor -
University and General

Fellows

Grulee, Clifford
Jammond, Margaret
Mayon-White, Richard
Panos, Theodore
Nelson, Edward, Teaching Assistant
(Hematology)

PEDIATRICS (Cont.)Fellows (Cont.)

Crump, Edwin, National Research Council
 Bergan, Robert
 Fridin, Frank
 Hanson, Carl
 Mulholland, William

Hall, Thomas (Childrens Hospital)

Moore, I. H. (St. Barnabas Hospital)

Augusstson, Hreidar (Northwestern Hosp.)

Anderson, Warren (General Hospital)

Lueck, Wallace "

Rogers, Charles "

Scherling, Sidney "

Schroeder, "

Walsh "

Smith, Carol

Smith, Theodore

Hesdorffer, Meredith B.
 Ingalls, Edgar G.
 Kugler, A. A.
 Leick, Richard M.
 Lindberg, Vernon L.
 Lynch, Francis W.
 Lytle, Francis T.
 Manson, Arnold J.
 McCloud, Charles N., Jr.
 McKenzie, Charles H.
 McKinlay, C. A.
 Morris, Stanley W.
 Myers, J. Arthur
 Richardson, Robert J.
 Sevenants, John J.
 Shimonek, Stewart W.
 Sinykin, Melvin B.
 Smith, Theodore S.
 Stebbins, Theodore L.
 Tangen, George M.
 Teisberg, John E.
 Wendland, John P.
 Winther, Nora
 Wipperman, Frederic F.
 Zeigler, Charles M.

STUDENTS HEALTH SERVICESTAFF ASSOCIATES

Boynton, Ruth E., Director
 Bates, Murray B., Associate
 Cottrell, Lillian "
 Cowan, Donald W. "
 Hinckley, Robert G. "
 Kernan, Phillip D. "
 Todd, Ramona L. "
 Weaver, Myron M. "

PART-TIME PHYSICIANS

Ausman, Duane R.
 Boreen, C. A.
 Bulinski, Theodore J.
 Bushard, W. J.
 Christensen, Llewellyn E.
 Christensen, Mary P.
 Cundy, Donald T.
 Christianson, Harry W.
 Fink, Lillian
 Fowler, L. Haynes
 Fuller, Alice E.
 Gallagher, Robert P.
 Garten, Joseph L.
 Hagen, Paul S.
 Hagen, Wayne S.
 Hanson, William A.
 Hauser, George W.

SURGERYGENERAL SURGERY

Wangensteen, Owen H., Professor and
 Chief
 Paine, John R., Professor
 Dennis, Clarence, Associate Professor
 Varco, Richard L., " "
 Merendino, K. Alvin, Instructor
 State, David, "
 Kolouch, Fred, "

Medical Fellows

Baronofsky, Ivan D.
 Brown, Schuyler P.
 Culmer, Charles U.
 Eddy, Frank D.
 Felder, Davitt
 Friesen, Stanley
 Gavisser, David
 Helferty, John
 Kremen, Arnold J.
 Lewis, F. John
 Lillehei, C. Walton
 Mears, Frederick B.
 Sanchez, Enrique
 Tongen, Jyle
 Toon, Robert
 Utendorf, Robert

GENERAL SURGERY (Cont.)

Medical Fellow (Cont.)

Sinclair, William (Northwestern Hosp.)
Strickler, Jacob (St. Barnabas Hosp.)
Stewart, Donald, (Miller Hospital)

Wild, John, Research Assistant

NEUROSURGERY

Peyton, William T., Professor and
Director

Buchstein, Harold F., Instructor
Titrud, Leonard, Instructor

Medical Fellows

French, Lyle A.
Simmons, Donald B.

UROLOGY

Creevy, C. D., Professor and Direc-
tor
Webb, Edgar A., Clinical Instructor

Medical Fellows

Eaves, George B.
McGroarty, Brian J.

Graduate Students

Minneapolis General Hospital

McCarthy, Austin
McCormick, Donald P.
Papermaster, Ralph
Schwyzer, Arnold

Fellows

Veterans Hospital

Boosalis, Michalas
Burnham, Wesley
Butter, John
Carlisle, Joseph
Chesler, Merrill
Davis, Donald D.
Farsht, Irving
Feuling, John
Giebink, Robert
Hagen, Clifford
Harper, Harry

Fellows
(Veterans Hospital) (Cont.)

Hartwick, Roger
Haskins, James
Howell, Carter
Johnson, Frank
Jones, Richard H.
Kusz, Clarence
Lenz, Gilbert
Mandell, Edward
Marturano, Frank P.
McCleery, Robert
Merner, Thomas
Olson, Eldon
Peterson, Lowell
Peterson, Wendell
Rosendahl, Frederick
Shanoski, Stanley
Sherman, Lloyd F.
Webber, Richard
Wiekman, Fred

OBSTETRICS & GYNECOLOGY

McKelvey, John L., Professor and Head
Lund, Curtis J., Associate Professor
Rogers, George E. B., Instructor

Fellows

Belleville, Titus
Freeman, Donald
Sargent, Edward
Seashore, Rosel
Stowe, Lyman

Anderson, James (Miller Hospital)
Baker, Milton (Minneapolis General Hosp.)

- - - - -

(To be concluded next week.)

III. RELATIONSHIPS BETWEEN ACUTE RHEUMATIC ENDOCARDITIS AND SUB-ACUTE BACTERIAL ENDOCARDITIS

B. J. Clawson

It is impossible to study one of the two types of endocarditis without considering the other. This is especially true of the bacterial type since it so commonly has a background of rheumatic endocarditis, acute, recurrent, healed or nearly healed valve deformities. The relationships, in many cases, are similar but dissimilar ones are encountered. An effort is made to evaluate the significance of these dissimilar relationships. Experiments in rats are used to further this study.

The chief relationships observed may be grouped as clinical, bacteriological, immunological and anatomical.

Clinical Relationships

Age. - Age is an outstanding clinical dissimilar relationship. Rheumatic endocarditis is the type found primarily on the pediatric wards while, on the other hand, the internist generally sees the subacute bacterial type. The separating line is close to the fifteenth year.

Cohn¹, in his analysis of a large number of cases of rheumatic endocarditis, found that the first manifestations of acute rheumatic endocarditis occurred under 13 years of age in 86 per cent of his cases. Gelfman², in an analysis of 578 cases of rheumatic heart disease, found subacute bacterial endocarditis as a cause of death to be particularly low under the age of 10. In an analysis of the ages in which death occurred in 98 cases of acute rheumatic endocarditis

TABLE I

Ages at which death occurred in acute rheumatic endocarditis (96 cases) and subacute bacterial endocarditis (417 cases)

Decades	Kinds			
	Acute rheumatic		Subacute bacterial	
	No.	Per cent	No.	per cent
1	40	41.6	8	1.9
2	40	41.6	38	9.1
3	9	9.3	91	21.8
4	5	5.2	98	23.5
5	1	1.0	79	18.9
6	0	0.0	56	13.4
7	0	0.0	36	8.6
8	1	1.0	8	1.9
9	0	0.0	3	0.7
10	0	0.0	0	0.0
Below 20	80	83.3	46	11.0
Above 20	16	16.6	371	88.9

and 417 cases of subacute bacterial endocarditis, I found that 83.3 per cent of the cases of acute rheumatic endocarditis which died in the first attack were under 20 years of age. Only 11 per cent of the 417 cases of subacute bacterial endocarditis were under 20 years of age at the

time of death (Table 1). The reason for this age difference in the two types of endocarditis has not been determined.

Transition of acute rheumatic into the subacute bacterial. - A gradual passing of an acute rheumatic endocarditis into

the bacterial type is commonly observed by the clinicians and the pathologists. The bacterial type is more severe clinically and nearly always ends fatally. An original diagnosis of acute rheumatic endocarditis is often changed to subacute bacterial endocarditis if the case progresses toward a fatal termination. The transition from the acute rheumatic to the subacute bacterial type may be so gradual that it is impossible to say where the one ends and the other begins. The following history illustrates such a case:

Case Report

A white girl, age 20, was admitted to the hospital June 21, 1935. At this time, she stated that she had been well up to about one month before admission when she had an acute upper respiratory infection. Following this, she became weak, tired and feverish and also had anorexia. Two weeks before admission she had noticed pain in the ankles, knees and wrists. The joints were swollen, red and especially painful on motion. This, however, receded before admission. On June 17, she had some pain over the precordium and began to have edema of the ankles which disappeared on rest. She then became dyspneic. Past history revealed two previous attacks of rheumatic fever which were characterized by swollen, red and painful joints. The first one occurred at 9 years of age when she was in the hospital for one month and in bed at home for one month; she had another attack similar to the first one at the age of 12. In addition, at this time, she had bronchopneumonia. After the second attack of rheumatic fever, she was told that she had a murmur in the heart.

Physical examination showed the patient to be well developed and well nourished. She was in no apparent discomfort. The lungs were normal. The blood pressure was 120/45. There were systolic and diastolic murmurs throughout the precordium. These were most marked over the aortic area and were transmitted toward the vessels of the neck. The heart was enlarged to the nipple line. The pulse was 85. There was no edema of the ankles.

The hemoglobin varied from 44 to 59 per cent and the red count from 3,050,000 to

3,920,000. The white count varied from 6,120 to 8,550. The sedimentation rate was 117 mm. in one hour. The Wassermann was negative. The blood culture on three occasions was negative. Repeated urine examinations showed from negative to a trace of albumin and a few to many leukocytes. There were occasional hyaline and granular casts but no erythrocytes. An electrocardiogram showed an inverted T wave in lead III. A cardiac study showed the heart equal to 53 per cent with an increased prominence of the left ventricular area.

The temperature varied daily from 99 to elevations of 103 and on several occasions to 104. Following admission, the cardiac condition did not improve and on July 20 a left sided hemiplegia developed. On July 22 petechiae developed on the conjunctivae. The respirations were increased to 42 and the patient began to have emesis. There were rales in both lung bases. She died at 6:20 A.M. on August 22, 1935.

The autopsy revealed a typical anatomical case of subacute bacterial endocarditis on the aortic valve with infarcts in the spleen, kidneys and brain, embolic glomerulonephritis and fibrinous pericarditis. Many streptococci were found in the thrombus on the valve when sectioned and stained by the Gram Weigert's method.

Background of the subacute bacterial type. - A third clinically observed relationship is that in at least 90 per cent of the cases of subacute bacterial endocarditis there is a background of a rheumatic valvular lesion either acute or partially or completely healed (Christian³, Gelfman²). Christian observed that the determinative background of subacute bacterial endocarditis is rheumatic heart disease in 89.33 per cent of 150 consecutive adult cases of subacute bacterial endocarditis caused by *Str. viridans* and in 90.24 per cent of 174 consecutive adult cases of subacute bacterial endocarditis of all causes studied at the Peter Brnet Brigham Hospital. Gelfman from a study of 115 cases of bacterial endocarditis in the same Hospital and in the Infants' and Children's Hospital found the incidence of bacterial endocar-

ditis in rheumatic heart disease to be 26.2 per cent in adults and 15.2 per cent in the children. He found the frequency of bacterial endocarditis was not the same in all decades. It was particularly low under the age of 10 and was highest between the ages of 20 and 29.

This age difference in the frequency of bacterial endocarditis in the first and third decades has been found to stand out conspicuously in 1104 cases of rheumatic heart disease in the Department of Pathology at the University of Minnesota. In the first and second decades recurrent rheumatic endocarditis is more likely to follow a previous attack while bacterial endocarditis appears in the late second decade and the third decade.

Subacute bacterial endocarditis a cause of death in rheumatic heart disease. - A fourth clinical relationship is that subacute bacterial endocarditis is the primary cause of death in from 25 to 30 per cent of all cases of rheumatic valve deformities (Gelfman², Clawson⁴). Table II shows how death occurred in 1407 cases of rheumatic heart disease. On the basis of there being a rheumatic background in 90 per cent of the cases of subacute bacterial endocarditis it is noted that 26.7 per cent of all cases of rheumatic heart disease died with subacute bacterial endocarditis. When the acute and recurrent rheumatic cases are omitted the per cent is increased to about 30.

TABLE II

How death occurs with
rheumatic heart disease (1407 cases)

Kinds	No.	Per Cent
Acute rheumatic	114	8.1
Recurrent rheumatic	97	6.8
Valve deformities	784	55.7
Adherent pericardium	37	2.6
Subacute bacterial Endocarditis	375*	26.7
Total	1407	99.9

*90 per cent of subacute
bacterial endocarditis.

Bacterial Relationships

The organisms found in the blood stream in both types are usually *Str. viridans*, a few per cc. of blood in the acute rheumatic and many in the bacterial. While the general belief is that acute rheumatic fever is in some way closely related to hemolytic streptococcal sore throat yet as stated by Jones⁵ and according to the observations of others the organisms found in the blood stream are generally *Str. viridans*. There are different opinions concerning the significance of these organisms in the blood.

Immunological Relationships

Humoral antibodies. - Both types have high titers of streptococcal humoral antibodies, as agglutinins, precipitins, complement fixing antibodies and generally in the acute rheumatic type antistreptolysins. These antistreptolysins tend to disappear since they are only present in about 30 per cent of the cases of healed valve deformities which obviously are preceded by attacks of acute rheumatic endocarditis (Winblad⁶).

Hypersensitiveness. - Skin tests to streptococci or streptococcal products are positive in 75 to 90 per cent of the cases of acute rheumatic endocarditis, 50 per cent in healed valve deformities and negative in subacute bacterial endocarditis (Birkhaug⁷, Gibson, Thomson and Stewart⁸, and Favour, Janeway and Gibson⁹).

The bacterial type of allergy in rheumatic fever has been studied by Swift¹⁰, Birkhaug⁷, Gibson, Thomson and Stewart⁸, and others. It has been shown by intradermal skin tests that a high percentage of patients with acute rheumatic fever are allergic and that patients with bacterial endocarditis are not allergic. It has also been observed that patients with bacterial endocarditis who have been cured by penicillin therapy again become hypersensitive. This suggests that patients having bacterial endocarditis had previously been allergic but had been desensitized by heavy blood stream infection.

A point to be considered in thinking of the role of delayed hypersensitiveness in rheumatic endocarditis is whether the rheumatic child was allergic before contracting rheumatic fever or whether allergy exists as a result of rheumatic infection or of intercurrent infections with increasing age. Birkhaug's⁷ observations showed that 20 per cent of normal children and 72 per cent of rheumatic children were allergic to streptococci. Gibson, Thomson and Stewart⁸ by skin tests with extracts of *Str. hemolyticus* found that the percentage of allergy rose from the age of 5 years to that of 15 years at which it ceased to increase in both rheumatic and control children. At 15 years of age there was practically no difference in the frequency and the degree of allergy to *Str. hemolyticus* extracts (68 per cent rheumatic, 66 per cent controls). They concluded that their work did not add much support to the allergic theory of acute rheumatic fever. The

same conclusion was reached by Jones⁵. Clawson and Wetherby¹¹ found that 50 per cent of normal people gave a positive reaction to *Str. viridans* intracutaneous tests. Hypersensitiveness to streptococcal products is rare in young children when acute rheumatic fever generally begins but is common in adults. It is especially high in both children and adults who have or have recently had rheumatic fever. In adults rheumatic fever seldom occurs for the first time, but bacterial endocarditis is rare. It may be suspected that allergy may be a factor in the age difference in the two types of endocarditis.

Anatomical Relationships

Valves involved. - The valvular involvement in acute rheumatic endocarditis and bacterial endocarditis is noted in Table III. In either group, the

TABLE III

Valve involvement in acute rheumatic and subacute bacterial endocarditis

Kinds	Valves														Total
	A	M	T	P	AM	AT	AP	AMT	AMP	AMTP	MT	MP	MTP	TP	
Acute rheumatic	2	43	0	1	24	0	0	10	0	9	9	0	0	0	98
Subacute bacterial	65	137	7	7	132	4	1	6	1	0	3	0	0	0	363
Total	67	180	7	8	156	4	1	16	1	9	12	0	0	0	461

aortic and mitral are the only valves involved to any extent. The tricuspid alone and the pulmonary alone are seldom involved in the acute rheumatic type but are more frequently affected in the subacute bacterial.

The relation of sex to the three kinds of valvular involvement (aortic alone, mitral alone, and aortic and mitral combined) is of interest in showing a low aortic involvement and a high mitral involvement in the females (Table IV). This is most pronounced in the rheumatic group, but it is definitely significant in the

subacute bacterial group. This anatomical relationship shows a marked similarity in the two groups.

Gross vegetations. - In the endocarditis immediately associated with acute rheumatic fever (rheumatic endocarditis, simple verrucous endocarditis) the heart valves on gross examination show small smooth globular nodules at points of contact of the leaflets a short distance back from the free margin on the atrial surfaces of the mitral and tricuspid and on the ventricular surfaces of the aortic and pulmonary valves.

TABLE IV

Combinations of valvular involvement (aortic alone, mitral alone, and aortic and mitral combined) per thousand autopsies in rheumatic heart disease and bacterial endocarditis.

	Males					Females				
	No.	A	M	AM	Total	No.	A	M	AM	Total
Rheumatic	478	8.1	8.1	9.9	26.2	300	2.2	18.7	9.8	30.8
Subacute bacterial	217	3.0	3.8	5.0	11.9	111	1.4	6.0	3.9	11.4

In subacute bacterial endocarditis the vegetations are, in the main, large, soft and villous. The consistency and size of the subacute bacterial vegetations make it easily possible for particles to break loose into the blood stream and produce the embolic processes so commonly found in subacute bacterial endocarditis.

Vegetations characteristic of the rheumatic type may generally be found (80 per cent Clawson¹², 100 per cent Von Glahn and Pappenheimer¹³) on the same cusp with the vegetations of the subacute type. Acute rheumatic vegetations may be present on the cusp of one valve while on another there may be subacute bacterial lesions. Recent observations have illustrated how the presence of the two types of lesions can be demonstrated on the same valve. Typical cases of subacute bacterial endocarditis cured by penicillin therapy have come to autopsy with death from other causes. The large villous vegetations were absent and organisms could not be cultured from the blood or found in the valve by the Gram Weigert stain. Within the valve the inflammatory reaction was typically that of acute or recurrent rheumatic endocarditis.

Microscopic vegetations. - Microscopically the acute rheumatic lesions show the inflammation within and throughout the substance of the leaflet. The elevated nodules form only at the line of contact of the leaflets. An important histological feature is the presence of hyaline fibrinoid material within the valves. A small platelet thrombus is present in which bacteria are not found.

The cellular inflammatory reaction occurs with the fibrinoid changes and is chiefly a proliferative inflammation. The cells are spindle-shaped or rounded, generally with an abundant cytoplasm. They are usually mononuclear, but multinucleated forms are common. They are derived from fibrocytes, histiocytes, blood lymphocytes and from a special histiocyte, the Anitschkow cell. This cell is peculiar in being found only in the heart. Definite Aschoff nodules are frequently seen in the acutely inflamed valve.

In subacute bacterial endocarditis all the structures of the acute rheumatic valve are present and in addition there is a large platelet thrombus in which many bacteria, generally in colonies, are usually found. The infected thrombus is the distinguishing characteristic lesion of bacterial endocarditis.

Embolic phenomena. - Evidences of embolic phenomena are common with subacute bacterial endocarditis. In 364 cases, embolic phenomena were present as follows: spleen 72 (19.7 per cent), kidneys 22 (6.0 per cent), brain 14 (3.8 per cent), spleen and kidneys 88 (24.1 per cent), spleen and brain 11 (3.0 per cent), spleen, kidney and brain (11.5 per cent), kidneys and brain 7 (1.9 per cent), or a total of 256 (70.3 per cent). Embolic Phenomena are not present with acute rheumatic endocarditis. Evidence of embolic processes is a dissimilar relationship.

Mural endocardial involvement. - Vegetations of the subacute type were present in 364 cases of subacute bacterial endocarditis in the following places: left ventricle 13.5 per cent, right ventricle 1.5 per cent, left atrium 13.5 per cent, right atrium 1.5 per cent, left atrium and right ventricle 2.5 per cent, right ventricle and left ventricle 0.5 per cent, left atrium and right atrium 0.5 per cent, or in a total 35.5 per cent. The percentage of mural involvement is about the same in acute rheumatic cases. The involvement is confined almost entirely to the atrium and the vegetations are like the vegetations on the acute rheumatic valve.

The myocardium. - The various types of inflammation are seen in Table V. The Aschoff nodule is less frequent in subacute bacterial endocarditis, 45 per cent, while in the acute rheumatic endocarditis it was present in 67.2 per cent of the cases. Von Glahn and Pappenheimer found the Aschoff nodule to be present in 46 per cent of the cases of subacute bacterial endocarditis which they studied. Abscesses are more common in the subacute bacterial type. Evidences of inflammation, acute or healed, occurred in 79.1 per cent of the acute rheumatic cases and in 88.5 per cent of the subacute bacterial cases.

TABLE V

Myocarditis in acute rheumatic and subacute bacterial endocarditis

	Aschoff nodules		Diffuse inflammation		Abscesses		Peri-arterial scars		One or more	
	No.	%	No.	%	No.	%	No.	%	No.	%
Acute Rheumatic (295)	47	67.2	12	17.9	2	2.9	21	31.3	53	79.1
Subacute bacterial (60)	27	45.0	36	60.0	13	21.5	38	63.5	53	88.5

Pericarditis. - The presence or absence of pericarditis has at times been used to differentiate acute rheumatic endocarditis from subacute bacterial endocarditis. Libman¹⁴ states that pericarditis is not observed in subacute bacterial endocarditis except when there is a diffuse glomerulonephritis with marked retention of nitrogen. Table VI shows the frequency of pericarditis in our cases

TABLE VI

Frequency of pericarditis in rheumatic endocarditis and subacute bacterial endocarditis

Kind	No. with pericarditis	Per cent
Acute and recurrent rheumatic endocarditis (183)	86	41.5
Healed valve deformities (671)	125	18.5
Total rheumatic (854)	211	24.7
Subacute bacterial endocarditis (364)	78	21.4

of acute and recurrent rheumatic endocarditis, in healed rheumatic valve deformities and in subacute bacterial endocarditis. Pericarditis is more frequent in the acute type of rheumatic endocarditis (41.5 per cent). This incidence does not represent the clinical incidence for the acute pericarditis evidently contributes largely as a cause of death.

The frequency of pericarditis with the entire group of rheumatic hearts closely represents the clinical incidence of pericarditis with acute rheumatic endocarditis. It is obvious that in the cases of rheumatic valvular deformities there has been an acute rheumatic endocarditis. In the cases of healed valve deformities in which there is no acute pericarditis or adherent pericardium, evidently in most cases there never had been an acute pericarditis, for the type of pericarditis (fibrinous) which is associated with acute rheumatic fever undergoes organization early in its course and permanent fibrous adhesions result. The incidence of pericarditis with the group of rheumatic hearts is 24.7 per cent, only slightly higher than the incidence with subacute bacterial endocarditis (21.4 per cent). Since the incidence of pericardial involvement in the total number of rheumatic heart disease cases represents closely the actual clinical incidence of pericarditis with acute rheumatic endocarditis and since this incidence is nearly the same as the incidence of pericarditis in cases of subacute bacterial endocarditis it appears obvious that the presence or absence of pericarditis can not be used clinically to differentiate the two types.

Interpretations of the Relationships of Acute Rheumatic Endocarditis and Subacute Bacterial Endocarditis

The relationships are most similar with the following exceptions in the subacute bacterial type: The older age at which the endocarditis appears; the more severe clinical course; the greater ease with which organisms are recovered from the blood stream; the absence of streptococcal allergy in the active cases; the larger and infected thrombus on the valve and the embolic phenomena.

The first of these dissimilar relationships may possibly be explained by a difference of hypersensitiveness in the two age groups. More investigation is needed to interpret the age difference of the two types.

The remaining five differences may be accounted for by the effect of the large infected thrombus, the characteristic valvular lesion in subacute bacterial endocarditis.

From a study of the various relationships of the two types, two different theories have evolved:

1. The two types of endocarditis are different diseases.
 - a. A healed rheumatic valve becomes reinfected.
 - b. An acute rheumatic vegetation becomes infected with another infective agent (Von Glahn and Papperheimer¹³).

Which or whether either of these two theories is correct has not been answered in human cases.

Experimental Endocarditis

Since the relationship of acute rheumatic and bacterial endocarditis and the possible substances or products of immune processes which cause the inflammation in the valves in the two types of endocarditis has not been determined definitely in human cases, the question arises whether it is possible to obtain additional information from experiments on animals.¹⁵ Can either or both types of valvulitis be produced by forced methods from a single inoculation or a short course of inoculations with a specific substance in an animal in which endocarditis seldom or never occurs spontaneously? In the following experiments an attempt is made to answer this question and also to study some of the foregoing considerations concerning the relationships and the genesis of the two types of endocarditis.

Results. - In Table VII it is noted

TABLE VII

Incidence of acute rheumatic-like endocarditis and bacterial endocarditis in rats injected with the following substances:

Substances injected	No.	Acute rheumatic-like		Bacterial		Both	
S. viridans (intracardiac)	94	38	41.4%	5	5.3%	5	5.3%
S. hemolyticus (")	75	25	30.6%	11	14.6%	2	2.6%
S. viridans (intraperitoneal)	10	0	0	0	0	0	0
S. hemolyticus (")	15	0	0	0	0	0	0
Dick toxin (intracardiac)	10	0	0	0	0	0	0
Rabbit serum (")	10	0	0	0	0	0	0
Horse serum (")	10	0	0	0	0	0	0
Egg white (intraperitoneal)	5	0	0	0	0	0	0
S. hemolyticus (subcutaneous in agar)	13	0	0	0	0	0	0
Control animals	37	0	0	0	0	0	0

that endocarditis, acute rheumatic-like or bacterial endocardial lesions, developed only when the whole organisms were put into the blood stream. The animals which had been inoculated so as to produce an anaphylactic shock, as with serum, egg albumin or Dick toxin, had no valvular injuries. The animals given intraperitoneal injections and those given subcutaneous injections of streptococci in agar, which was to hold the organisms locally and permit the toxin to diffuse into the blood stream, likewise had no valvular injuries.

The rheumatic-like lesions found on the valves (the mitral, the aortic and tricuspid in order of frequency) had a definite anatomical resemblance to the verrucous lesions on the human heart valves in acute rheumatic fever. No claim is made that these verrucous vegetations are etiologically similar to the vegetations in acute rheumatic endocarditis. Microscopically, as in the human lesions, the inflammation was present throughout each involved valve. The hyaline fibrinoid material, emphasized by Klinge¹⁶ and others as characteristic, was within the valve. The cellular inflammatory reaction occurring with the fibrinoid change was chiefly a proliferative type. When there was desquamation of the endothelium a small platelet thrombus was formed. No bacteria were found in the valves or the thrombus. Def-

inite healing was seen to occur in a few cases.

The bacterial type of endocarditis showed a cellular and fibrinoid reaction similar to that in the rheumatic-like vegetations, but the platelet thrombus, which was usually more extensive, was infected and had colonies of streptococci lying within the thrombus.

Comment. - Some additional suggestions concerning the relation between acute rheumatic and bacterial endocarditis are obtained from the experiments. It is evident that both types of experimental endocarditis are the result of infection with the same agent, *Str. viridans* or *Str. hemolyticus*. The possibility of the verrucous rheumatic-like vegetation being due to a previous cause can hardly appear likely.

Another fact to be noted is that bacteria are not found in the rheumatic-like lesions, the production of which was evidently started by the streptococci only a relatively short time before the lesions were examined. This should be considered when examining human rheumatic endocarditis.

The experimental lesions differ only in degree of injury and reaction in the cusps and in the larger vegetations con-

taining colonies of streptococci in the typical bacterial endocarditis. The conception of Von Glahn and Papperheimer that the platelet thrombus in the rheumatic type becomes infected and produces the bacterial type appears to represent the probable course of events in these experimental lesions. The infection is evidently the same and the degree of valvular involvement in the two types can not be widely separated. Evidences of healing in some of the rheumatic-like lesions are observed suggesting the probable process in the development of healed rheumatic valvular deformities.

Some additional knowledge is obtained from the experiments concerning what enters into the valve to start the inflammatory process.

There is no evidence favoring the theory that streptococcic toxin or anti-toxin acts directly or by an allergic reaction on the valve, for toxin producing hemolytic streptococci were grown in the animal in the subcutaneous agar nodules and in the peritoneal cavity and the Dick streptococcic toxin was injected into the cavity of the heart without development of valvular lesions.

Precipitins probably were present in the animals receiving the toxin, serums and egg albumin but no lesions were observed in these animals. Bacterial allergy was probably present in the animals inoculated subcutaneously with streptococci in agar. The animals in which endocarditis developed in all probability did not have allergy of the bacterial type. The anapylactic type of hypersensitivity resulting from inoculations with rabbit and horse serums, Dick toxin, streptococcic toxin and egg albumin did not stimulate any inflammatory change in the valves. In some of the animals, especially those given injections of the Dick toxin, definite and severe symptoms of anaphylaxis were observed.

The influence of immune reactions on the genesis of endocarditis has not been interpreted conclusively. It is suggested that a high humoral antibody content favors the development of valvular lesions by localizing the organisms in the valves and that the bacterial allergic state may

favor the development of the bacterial type of endocarditis, for it has been found that with equal doses of streptococci a greater degree of tissue reaction occurs in animals having the delayed type of hypersensitiveness than in non-allergic animals. It may be possible that the absence of, or the low incidence of, the degree of streptococcic hypersensitiveness in children and its higher incidence in adults may be a prominent factor in the difference in the age incidence of acute rheumatic and bacterial endocarditis. Further observations and experiments are necessary before definite statements may be made.

The only substance in the experiments which obviously stimulated the inflammatory reaction in the valves was the bacterial protein itself, both that of *Str. viridans* and *Str. hemolyticus*.

Conclusions

1. Many similarities are found in acute rheumatic and subacute bacterial endocarditis.
2. The dissimilar relationships may largely be accounted for by the larger, soft and infected thrombus in the subacute bacterial type.
3. It is suggested that the age difference in the 2 types of endocarditis may be due to a difference in streptococcic hypersensitiveness at different age periods.
4. Anatomical rheumatic-like vegetations and the subacute bacterial type of vegetations can be produced in the same animals by a single or a short course of inoculations.
5. The 2 types of lesions produced by the same cause offer some support to the theory that the 2 types of endocarditis are etiologically similar with different manifestations.

References

1. Cohn, A. E. and Lingg, C.
J.A.M.A. 121, '43.

2. Gelfman, R.
Ann.Int.Med., 19:253, '43.
3. Christian, H. A.
Am.J.M.Sc.201:34, '41.
4. Clawson, B. J.
Unpublished data.
5. Jones, J. D.
J. Pediat. 15:772, '39.
6. Winblad, Sten.
Acta path. et microbiol.
Schandinav.Supplimentum. 44:199, '41.
7. Birkhaug, K. E.
J. Infect.Dis. 43:280, '28.
8. Gibson, H. J.
Thomson, W.A.R., and Stewart, D.
Arch.Dis.Childhood, 8:57, '33.
9. Favour, C. B., Janeway, C. A., Gibson,
J. G. and Levine, S. A.
New Eng. J.M. 234: 71, '46.
10. Swift, H. F.
J.A.M.A. 90:906, '28.
11. Clawson, B. J., and Wetherby, M.
Ann. Int. Med. 5:1447, '32.
12. Clawson, B. J.
Arch. Int.Med. 37:66, '26.
13. Von Glahn, W. C., and Pappenheimer,
A.M.
Arch.Int.Med., 55:173, '35.
14. Libman, E.
Arch.Int.Med.33: 701, '24.
15. Clawson, B. J.
Arch.Path.40:153, '45.
16. Klinge, F.
Ergebn.d.allg. Path.u.path. Anat.
27:1, '33.

IV. GOSSIP

The first meeting in the present series of University of Minnesota Hospitals General Staff Meetings was held November 14, 1929 under the chairmanship of Paul B. Fesler who was Superintendent at that time. This is our eighteenth year of meetings under this plan and we anticipate as in previous years that we will have a successful program. Every department and division of the medical school which performs a clinical service will take part in the program. Some of the larger departments will appear three times, while most of the divisions will appear only once. A complimentary lunch is served before the meeting, starting at 11:45. We urge all those who can do so to come early to make way for those who must come later. Another way in which you could help would be to occupy the front seats first, especially if you must leave the meeting before its conclusion. (There are convenient exits in all parts of the room, front or back). The meetings will start at 12:15 and end at 1:15. As a general rule the last 15 minutes is reserved for discussion, questions and answers. A copy of the Staff Meeting Bulletin will be given to each member as he leaves the room. In addition to this service, we also send the Bulletin to any physician or institution for \$2.00 a year to cover part of the cost of postage and mailing. The main cost of the Bulletin is borne by the Citizens' Aid Society, through a special grant for this and other purposes. During the current year, because of help shortage in the Mimeograph Department, all copy for the Bulletin must be in our hands on or before Saturday noon of the week previous to the scheduled meeting....The medical school is still on the "no vacation" schedule and a new freshman class will enter January 1, 1947; a senior class will graduate and the freshman class which enters in September 1947 will be the first to have the traditional summer vacation (1948). Contrary to expectations, the incoming class this winter has a large number of applicants (350 for 100 places). In addition to the undergraduate and

graduate (resident) classes, we have a group of 90 veteran students enrolled in special departmental courses in the Basic Sciences and Their Clinical Application, at the Center for Continuation Study. They are taking a nine-months' course which will be completed at the end of the spring quarter, 1947, following which most of them hope to obtain a residency, have already done so, or plan to go back to one which they discontinued in order to take the basic sciences. In connection with this course, a number of distinguished men and women have been invited to serve on our faculty. The list for October is the following: October 3-4, George E. Burch, Tulane; October 7-8, Norman C. Wetzel, Western Reserve; October 8-9, Alexander W. Winkler, Yale; October 9-10, C. A. Smith, Harvard; October 10-11, William Dock, Long Island; October 14-15, Joseph Warkany, Cincinnati; October 16-17, Arild E. Hansen, Texas; October 18-19, Allan M. Butler, Harvard; October 21-22, Lawson Wilkins, Johns Hopkins; October 24-25, Paul C. Bucy, Illinois; October 28-29, Frederic Gibbs, Illinois; October 29-30, Hobart A. Reimann, Jefferson; November 1-2, Arnold Gesell, Yale; November 1-2, Leo Kanner, Johns Hopkins; and others to be announced. You are welcome to attend any meeting at the Center. The special series of Pediatric lectures during October will be supplemented by seminars by our own staff....We welcome the publication of the Surgical Seminars at the United States Veterans Hospital as a friendly rival. The new "University of Minnesota Hospital Service" at the Veterans Hospital plans to make the "parent" organization step this year to keep up with it. With this challenge and with a knowledge of our outstanding performance in the past, we ask your cooperation:

- (1) Attend meetings,
- (2) Take part in the discussion,
- (3) Take your turn for a program.

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