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Pneumatization of Temporal Bone

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MERRY CHRISTMAS HAPPY NEW YEAR

BULLETIN OF THE
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I. PNEUMATIZATION OF THE TEMPORAL BONE

Lawrence R. Boies, M.D.
L. Ian Younger, M.D.

Introduction

Acute infections involving the middle ear and mastoid in a normally pneumatized temporal bone can now be almost completely controlled if adequate chemotherapeutic and antibiotic measures are used at or near the onset of the infection, and administered in adequate dosage, combined with drainage where indicated.

However, chronic suppurations of the middle ear and mastoid present a different problem. In the past one of the common beginnings for chronic suppurative otitis media was the severe necrotizing type of infection seen most often with scarlet fever and measles, in which extensive necrotic change affected the middle ear, making repair improbable. These cases could practically be classed as chronic otitis media from the start.

That picture has almost vanished in our experience due to a better control of the complications of scarlet fever and measles or the severe streptococcal and pneumococcal infections. Today chronic suppurations are still a problem, however, and though we may eventually "run out" of chronic ears that originated in the pre-sulfonamide and pre-antibiotic era, there may still remain the frequent case whose etiology is associated with abnormal pneumatization of the temporal bone.

At the present time abnormal pneumatization also seems to play a role in the chronicity, and possibly in the etiology, of some cases of secretory effusion into the middle ear.

Normal Development of the Temporal Bone

At birth the temporal bone consists of three parts. The petrous portion, wedged in the base of the skull between the sphenoid and occipital bones, contains the inner ear. The flat thin squamous portion lies above the petrous bone and gives origin to the zygomatic process anteriorly. The tympanic portion is a thin incomplete ring of bone enclosing the tympanic membrane and forming a segment of the external auditory canal.

These bones are of membranous origin and do not unite as the intact temporal bone until the end of the first year of life. In the new-born infant there is no mastoid eminence, but during the first few years of life it gradually develops from a combination of the down growth of the petrous and squamous bones and traction of the sternomastoid muscle which is attached to it.

The middle ear space (tympanum) originates from an invagination of the nasopharyngeal wall, the first pharyngeal pouch, which pushes laterally toward the ingrowing external ear canal. The ear drum develops from the tissue separating these two cavities where they approximate. The inner cavity is modified to form the eustachian tube internally and the precursor of the middle ear laterally. This middle ear space is at first only a dorsal slit embedded in embryonic connective tissue. However, with growth of the foetus this connective tissue becomes progressively looser and less cellular, and by the twentieth week the tympanum begins to expand and push back this mucoid acellular mesenchyme.

At a still later stage of foetal life the antrum of the mastoid begins to form as a posterior extension of the middle ear space. It gradually tunnels backwards through the tissue lateral to the otic capsule and comes to occupy a position above and behind the tympanic membrane. This branch cavity of the middle ear is also lined with mucous membrane and remains in continuity with the middle ear through the aditus ad antrum. By full term the antral space has not only attained relatively large size, but has shown evidence of its potentiality to pneumatize the future mastoid process. Many small mucosal outpouchings dot the antral walls, and these will develop into

the air cells of the mastoid.

Beginning early in infancy and continuing most rapidly from the age of one until the fifth to the tenth year, the dome shaped mastoid process evolves. If there is no interference, as the bony mass accumulates, it is continuously hollowed out from within by a pneumatic system of mucosa lined, interconnected cells originating from the antrum and middle ear space.. Mastoid growth and pneumatization are in great part complete by the end of the tenth year or earlier but may continue slowly until puberty. In the fully pneumatized temporal bone the air cells may extend throughout its entire body from the tip of the mastoid process below to the squamosa of the skull above, from the zygomatic process anteriorly to the sigmoid sinus posteriorly, and medially may penetrate the petrous pyramid to its apex in close proximity to the sixth nerve and the cavernous sinus.

There is considerable variation in the degree of pneumatization. Tremble describes four types of mastoid bone based upon their cellular pattern: (1) the pneumatic, (2) the diploic, (3) the mixed (pneumatic and diploic) and (4) the sclerotic.

He states that in a pneumatic mastoid the bone is hollowed out by large intercommunicating cells lined by a delicate membrane of non-ciliated squamous epithelium which is closely adherent to the periosteum. This is often referred to as mucoperiosteum.

The diploic mastoid is formed of dense, compact bone which contains small marrow-filled cells without a mucosal lining.

The mixed type is composed of pneumatic and diploic cells in variable combination. In some, pneumatic cells preponderate while in others of this type the cortex is thick and solid with isolated groups of small cells.

The sclerotic type consists of hard ivory-like bone with little air space other than the antrum. This type is rare.

Most adult temporal bones fall into the pneumatic group, but percentages of diploic, mixed and sclerotic bones of as high as 20 to 30 per cent have been quoted.

The Etiology of Abnormal Pneumatization

According to Almour, the early mastoid process appears histologically as spongy bone containing fatty bone marrow or embryonal connective tissue. At birth the middle ear and antral region is more or less filled with mesenchyme containing pockets and extensions of epithelium. As the mastoid matures and gradually pneumatizes, the gelatinous connective tissue thins and atrophies between the epithelial spaces and the bony surfaces, allowing the air spaces to expand into tho bone. It appears to be more than a passive process for the sub-epithelial tissue actually invades the soft bone, causing indentations and and erosions through osteoclastic activity, thereby creating more spaces into which tho air containing epithelial sacs can press. Bone marrow undergoes a regressive metaplasia into embryonal connective tissue wherever it lies in the path of an extending air cell.

There has been much speculation as to the exact mechanism of this pneumatization. Wittmaach in 1918 was the first to expound a theory which substantially explained the variations seen in the adult temporal bone. He claimed that the invasive process was a function of the epithelial lining of the air spaces and that the eventual degree of pneumatization depended upon the inherent or inherited activity of the mucosa, plus the presence or absence of middle ear inflammation during the early months of infancy. It was hisstrong contention that infantile otitis media, by permanently altering the characteristics of the middle ear mucosa, caused deficient pneumatization. In the absence of any such inflammation the temporal bone should go on to complete or nearly

complete pneumatization, and it is such middle ear disease which primarily causes deficient air cell formation.

Albrecht agreed that healthy mucosa was essential to maximum pneumatization but added that the inherited characteristics of the mastoid bone as a whole were an important factor. He felt that a diploic or mixed type of mastoid might be the result of the constitutional growth pattern of the bone itself, as much as the result of neo-natal ear inflammation. In other words, the degree of pneumatization is in great part inherited, according to him.

Schwarz, in a study of identical and fraternal twins and of triplets, came to the same conclusion that hereditary factors played a great part in determining the ultimate air cell pattern.

Ruedi in 1939 stated that pneumatization depended primarily upon the preformation of spaces within the bone substance by osteoclastic activity and secondarily upon the ability of the mucosa to follow into these spaces after atrophy of the contained connective tissue.

These, then, are the principal theories advanced to explain pneumatization of the temporal bone. But the question remains of how to account for total lack of pneumatization as seen in the sclerotic mastoid. Can inflammation of bone account for complete ablation of all air cells? Or can early pathologic influences entirely inhibit the process of pneumatization and prevent any air cells from ever forming? The answer would seem to lie somewhere between the two extremes.

Histologic studies show that air cell formation is already under way at birth, before bacterial inflammation can prevent the formation of some cells. It is also known that the degree of inflammation within a mastoid varies in its different portions, and in a well pneumatized bone it is unlikely that all traces of the air cell system could be abliterated by the disease. Such remaining portions of epithelium, even though cut off from the antrum, resist the formation of new bone and remain as cystic or "pseudopneumatic" formations.

Thus, a markedly sclerotic mastoid may be the product of chronic or recurrent inflammation in a bone which was poorly pneumatized from the beginning. Partial sclerosis may occur in any portion of a mastoid subjected to chronic inflammation.

The conditions that interfere with the start and progress of normal pneumatization are, for the most part, definitely established.

Histologic studies of the temporal bones of full term fetuses have shown instances of residue of the solid constituents of the amniotic fluid in the middle ear, particularly in cases in which there has been a premature rupture of the foetal membrane. Meconium has also been encountered in the middle ear space. Following premature rupture of the amniotic sac, the middle ear of the new-born infant as early as eight hours following delivery may contain masses of pus cells and bacteria.

Otitis media complicating upper respiratory infections in infants is potentially more detrimental to the development of a normal middle ear than it is in children two or three years of age.

Certain aspects of the nursing acts of infants might also explain some cases of eustachian tube irritation which might develop middle ear disturbances. For instance, the too free flow of milk from a nursing bottle may cause coughing and sputtering to push some of this milk into the eustachian tube and thus, produce irritation and congestion of the lumen of the tube, which is relatively wider, shorter, and straighter than in the adult.

<u>The Role of Abnormal Pneumatization in Present Day Middle Ear Disease</u>

A. Chronic Suppurative Otitis Media

We believe that abnormal pneumatization of the temporal bone is a prominent factor in the development of chronic suppurative disease of the middle ear and mastoid.

The pathogenesis of chronic suppurative otitis media may be as follows:

1. As the sequela of a severe infection causing necrotic change in some portion of the tympanum. This was not uncommon in severe middle ear infections such as occur in scarlet fever, measles, diphtheria, etc. The necrotic change may be marked enough so that the ear is destined to chronicity from the occurrence of this necrotic change. This change may involve the mucosa, the ossicles, and bony walls, in addition to the invariable destruction in the drum membrane. Granulations and polypi are common. A marginal performation involving the annular rim of bone allows a ready pathway for an ingrowth of squamous epithelium. This epithelial ingrowth is an attempt on the part of nature to heal the infection; it may invade the space rapidly, proliferate, desquamate and form a cholesteatoma.

2. In an acute otitis media in an ear in which the mucosa has remained hyperplastic. When there has been an interference with the normal development of the tympanic mucosa so that it has remained hyperplastic through the effect of an otitis media neonatorum or a middle ear infection in early infancy, this mucosa is considered to be poorly resistant to infection. An acute otitis media superimposed on this hyperplastic mucosa is thought to be destined to chronicity. There is, of course, no mastoid pneumatization or very limited pneumatization in this situation inasmuch as the pneumatizing process has never gotten started or has been checked early in its development. Granulations and polypi are common to this type of pathology. Unless there is a marginal perforation, the formation of cholesteatoma is not common.

3. As a result of the formation of a cholesteatoma from an ingrowth of epithelium from Shrapnell's membrane without preexisting perforation or otitis media. This ingrowth of epithelium results from a retraction of Shrapnell's membrane from a negative pressure in the attic. Two factors may produce this negative pressure. It may result from the closing off of the attic by the presence of a persistent hyperplastic subepithelial connective tissue in the epitympanic recess. Or it may result from a prolonged occlusion of the eustachian tube due to nasopharyngeal pathology. When Shrapnell's membrane is drawn in, a blind pouch is formed by the invagination. The neck of the pouch is too constricted to allow escape of the desquamating squamous epithelium. Thus, a cholesteatoma forms. Its presence becomes known when it becomes large enough to extend out of the attic, or when saprophytic infection of the epithelial debris causes discharge through the small perforation.

4. As a complication of ordinary acute otitis media in an ear with normal pneumatization. Ordinary acute otitis media in a normally pneumatized mastoid rarely becomes chronic. Even before the use of the sulfonamides and antibiotics chronicity developing in this situation was rare. The ear healed by natural methods or surgical therapy produced a cure or the patient died of complications such as meningitis, the sequelae of blood stream infection, etc. Today, it is probable that a case of ordinary otitis media in a normally pneumatized mastoid treated skillfully with the antibiotics practically never becomes chronic.

We stated in the introduction that the disease such as scarlet fever and measles, in the past such a potent cause of severe necrotic middle ear changes, are now less common in their incidence and their complications are better controlled by modern medical therapy. Therefore, chronic suppurations developing in the present day have their origin in some previously abnormal condition in

the middle ear. These abnormalities are referred to under the second and third causes of chronic middle ear suppuration. It is probable that in minor degrees of persistent embyronic middle ear mucosa, and a limited degree of subnormal mastoid development, that the tendency toward chronicity is less marked should an acute otitis media develop in this situation. However, when there is a practically complete lack of pneumatization, a chronic state of suppuration is very likely to occur.

B. Secretory Effusion into the Middle Ear

This is really an ancient disorder. Politzer devoted many pages to it in his textbook published in 1902. In the past decade or two one finds little mention of it in contemporary textbooks. This was emphasized by Hoople in a modern consideration of the disorder. Recent writings on this subject suggest that it is becoming more common.

Secretory effusion is referred to under several titles such as secretory otitis media, tubo-tympanic catarrh, catarrhal otitis media, etc. The suffix "itis" designates an inflammatory condition, but the fluid in the middle ear is actually sterile, usually serous, although sometimes mucoid. Undoubtedly, the number one factor in its production is occlusion of the eustachian tube producing a negative pressure in the middle ear, which subsequently results in an effusion of serum to fill the middle ear space, the mastoid antrum and often the mastoid cells.

Probably, a limited mastoid development might work toward a more ready occurrence of negative middle ear pressure, but this probably is not important in the total incidence of the disease. However, it seems probable on the basis of clinical observations that limited pneumatization of the temporal bone may be important as a factor in the chronicity of secretory effusion into the middle ear and mastoid.

Is the therapy now used in acute otitis media adequate? The incidence of acute suppurative otitis media is obviously less common today for the reason already referred to; namely, the early control of the diseases of which acute otitis media is a complication. When acute otitis media does develop, the common treatment on the part of the family doctor or pediatrician is the administration of one of the antibiotics. The usual result is a prompt abatement and apparent resolution of the infection. However, there seems to be clinical evidence that some of these cases heal slowly because of retained secretion, and that there follows a fibrosis in the middle ear and mastoid mucosa, and in some instances a limited bony sclerosis. These changes are more likely to occur when there has been previously abnormal pneumatization. This change makes these tissues more vulnerable to subsequent infections, and the patient more likely to acquire some degree of a more or less permanent conductive hearing loss. The importance of adequate drainage and follow-up hearing tests is over-looked.

Summary

1. Abnormal pneumatization of the temporal bone is common. An important cause is found in neonatal otitis apparently associated with retention of the solid constituents of amniotic fluid or the presence of meconium in the middle ear. Equally important, no doubt, are the infections reaching the middle ear in early infancy and childhood.

2. Abnormal pneumatization of the temporal bone is an important factor in the development of chronic suppurative otitis media in the present era of antibiotic therapy. It is also apparently one factor in the incidence of chronic secretory effusion into the middle ear.

3. There seems to be evidence that the therapy now commonly used in acute suppurative otitis media without re-

gard for drainage results in some instances of permanent changes in the middle ear and mastoid mucosa and some bony sclerosis. This may produce a degree of permanent hearing loss and subsequent vulnerability to infection. This sequence of events is more common in instances of previously abnormal pneumatization.

From these facts it is obvious that the accurate interpretation of the signs on the ear drum, of the information to be obtained from x-ray studies, and from modern hearing tests are extremely important.

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II. MEDICAL SCHOOL NEWS

Coming Events

Jan. 3-5 Continuation Course in Gynecology for General Physicians
Jan. 7-9 Continuation Course in Pediatrics for General Physicians
Jan. 15 George Chase Christian Lecture; "Current Thoughts on Viruses and Cancer," Prof. C. H. Andrewes, University of Leeds, England; 15 Owre Amphitheater; 8:00 p.m.
Jan. 21-25 Continuation Course in Electrocardiography for General Physicians
January 22 Minnesota Pathological Society Meeting; "The Problem of Intra-Cellular Parasitism in Brucellosis," Dr. Wesley W. Spink, Owre Amphitheater, 8:00 p.m.

* * *

Continuation Course in Pediatrics

A continuation course in Pediatrics will be presented for general physicians January 7-9, 1952. The course, which will be presented at the Center for Continuation Study, will emphasize the management of trauma and accidental poisonings in pediatric practice. Dr. Julian D. Boyd, Professor of Pediatrics, University of Iowa Medical School, will be the guest faculty member for the course. He will present the following subjects: "The Growth and Development of Children" and "Practical Problems in Infant Feeding." Dr. Boyd will also participate in the Pediatrics X-ray Conference along with Doctors Irvine McQuarrie, Leo G. Rigler, and other members of the staff.

* * *

Faculty News

Dr. Robert B. Howard, Instructor, Department of Medicine, has been appointed Director of Continuation Medical Education to succeed Dr. George N. Aagaard, who has accepted the Deanship at Southwestern Medical School of the University of Texas. Dr. Howard is a graduate of the University of Minnesota Medical School. He served his internship at the University of Minnesota Hospitals 1944-45 and except for a period of 22 months as a medical officer of the United States Army Medical Corps, Dr. Howard has spent the years since the completion of his internship in graduate study in internal medicine at the University of Minnesota Medical School. He has served as Fellow and Instructor under Dr. Cecil J. Watson and is at present completing his thesis for his Ph.D. in medicine. Dr. Howard's appointment is hailed by his many friends and associates who appreciate his outstanding qualities as a scholar and organizer, his devotion to duty, and his sparkling sense of humor. Students, faculty, and friends of the Medical School wish to join in congratulating Dr. Howard on his new appointment and extending best wishes for a stimulating and successful time in his new post.

Dr. John S. Gillam, formerly a member of the Department of Obstetrics and Gynecology and now associated with the Fargo Clinic, Fargo, North Dakota, will be a guest faculty member for the continuation course in Gynecology to be presented January 3-5, 1952. Dr. Gillam will present the subject, "Experiences with Marshall-Marchetti Type of Surgical Repair of Urinary Incontinence."

* * *

SEASONS GREETINGS AND FAREWELL

In July, 1941, it was my good fortune to obtain an appointment as fellow in the Department of Medicine of the University of Minnesota Hospitals. Since that time it has been my privilege to work in a variety of posts with colleagues to whom I owe much. It is impossible to do more than acknowledge my great indebtedness and express my deepest and most sincere thanks. Finally, I wish to extend my best wishes for the future which will most certainly bring additional growth and honor to this institution.

George N. Aagaard, M.D.

III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

December 24 - 29, 1951

Monday, December 24

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; M-109, U. H.

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

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

12:15 - 1:20 Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.

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

4:30 - 5:30 Dermatological Seminar; M-346, U. H.

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

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

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

Minneapolis General Hospital

7:30 a.m. Fracture Grand Rounds; Dr. Zierold, Station A.

11:00 - Pediatric Rounds; Dr. Top; 7th Floor.

12:30 p.m. Surgery Grand Rounds; Dr. Zierold; Station E.

1:00 - 2:00 X-ray Conference; Classroom, 4th Floor.

1:30 - Pediatric Rounds; Dr. Ulstrom; 4th Floor.

Veterans Administration Hospital

9:00 - G. I. Rounds; Drs. Ebert, Wilson and Breidenbach; Bldg. I.

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

2:00 - Psychosomatic Rounds; Building 5, Dr. Aldrich.

3:30 - Psychosomatic Rounds; Building 1, Dr. Aldrich.

Tuesday, December 25 (H O L I D A Y)

Wednesday, December 26

Medical School and University Hospitals

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

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

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

1:30 - Conference on Circulatory and Renal Systems Problems; M. B. Visscher; 116 Millard Hall.

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

5:00 - 6:00 Vascular Conference; Todd Amphitheater, U. H.

5:00 - 7:00 Dermatology Clinical Seminar; Dining Room, U. H.

7:00 - 8:00 Dermatology Journal Club; Dining Room, U. H.

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

Ancker Hospital

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

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

Minneapolis General Hospital

9:30 - Pediatric Rounds; Dr. Platou; 7th Floor Annex.

11:00 - Pediatric Rounds; Dr. Top, 7th Floor.

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

12:15 - Pediatric Conference; 4th Floor Annex.

1:30 - Pediatric Rounds; Dr. Huenekens and Dr. Ulstrom; 4th Floor Annex.

Veterans Administration Hospital

8:30 - 10:00 Orthopedic X-ray Conference; Conference Room, Bldg. I.

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

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

Thursday, December 27

Medical School and University Hospitals

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

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; M-109, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

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

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

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

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

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

Minneapolis General Hospital

8:00 - Pediatric Rounds; Dr. Gibbs; 5th Floor.

8:30 - Neurology Rounds; Dr. Heilig, 4th Floor Annex.

9:00 - Neurology Grand Rounds; J. C. Michael and Staff; Station A.

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

11:30 - Pathology Conference; Main Classroom.

1:00 - 2:00 Fracture - X-ray Conference; Dr. Zierold; Classroom, 4th Floor Annex.

2:00 - Psychiatry Rounds; Dr. Benton, 4th Floor Annex.

Veterans Administration Hospital

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

9:15 - Surgery Grand Rounds; Conference Room, Bldg. I.

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

1:00 - Metabolic Disease Rounds; Drs. Heller, Jacobson, and Johnson; Bldg. I.

Friday, December 28

Medical School and University Hospitals

8:30 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

Friday, December 28 (Cont.)

Medical School and University Hospitals (Cont.)

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

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

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

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

2:00 - 3:00 Dermatology and Syphilology Conference; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U. H.

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

4:00 - 5:00 Dermatology Seminar; W-312, U. H.

4:00 - Neurophysiology Seminar; 113 Owre Hall.

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

Ancker Hospital

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

Minneapolis General Hospital

8:00 - Pediatric Allergy Rounds; Dr. Nelson; 4th Floor.

11:00 - Pediatric Rounds; Dr. Top; 7th Floor.

11:00 - Pediatric-Surgery Conference; Drs. Wyatt and F. Adams; Classroom, Sta. I.

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

1:30 - Pediatric Rounds; Dr. Ulstrom, 4th Floor.

Veterans Administration Hospital

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

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

1:30 - Chest Conference; Wm. Tucker and J. A. Meyers; Ward 62, Day Room.

3:00 - Renal Pathology; E. T. Bell; Conference Room, Bldg. I.

Saturday, December 29

Medical School and University Hospitals

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

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; E-221, U. H.

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

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

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

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

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

Minneapolis General Hospital

8:00 - Pediatric Rounds; Dr. Gibbs; 5th Floor.

11:00 - 12:00 Pediatric Clinic; Dr. Thomas and Dr. May Classroom, 4th Floor Annex.

Veterans Administration Hospital

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

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