Bulletin of the

University of Minnesota Hospitals
and
Minnesota Medical Foundation

Head Pain

Volume XXII | Friday, December 15, 1950 | Number 10

BULLETIN OF THE
UNIVERSITY OF MINNESOTA HOSPITALS
and
MINNESOTA MEDICAL FOUNDATION

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Published weekly during the school year, October to June, inclusive.

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I. HEAD PAIN

A. HEAD PAIN

Lawrence R. Boies

Introduction

The clinical experience of the average victim of head pain which is a chronic problem is changing. This is the result of many contributions recording observations which have demonstrated that the basic mechanism of the production of head pain in many instances is a stretching of pain sensitive nervous structures along blood vessels. Also, there seems to be an adequate basis for the belief that autonomic dysfunction may play an important role in the etiology of some instances of head pain, and that localized tissue damage on the basis of a physical or intrinsic allergy causing a localized production of histamine may produce the pain from the vasodilating effect of this substance.

The term head pain is an inclusive reference which encompasses both headache and neuralgia. Stedman's medical dictionary defines headache as "a diffuse pain in various parts of the head not confined to the distribution of any nerve", and neuralgia as "a pain of a severe throbbing or stabbing character in the course of the distribution of a nerve".

On the basis of our knowledge to date, it would seem that head pain is produced in one of three possible ways.

1. By a change in cerebral blood volume. The greater arterial blood volume which occurs in the febrile state accompanying an infectious disease would be an example of this.

2. The localized vasodilating effect of histamine released through localized tissue damage on the basis of a physical or intrinsic allergy.

3. Direct irritation of a sensory nerve or nerves. The pain associated with dental disease or the headache produced by pressure from the swelling in acute maxillary sinusitis are examples of this mechanism.

A Classification of Headache

On the basis of modern ideas a relatively simple classification of headache is suggested. It follows the grouping of one suggested by Auerbach in 1913 who divided the types of headaches into three groups as follows: headaches due to general or systemic disorders, those seemingly independent of any bodily disorders, and those that have their origin from some disorder localized within the head.

Headache from Systemic or General Disorders

1. Infectious diseases or localized inflammations causing systemic disorder.
2. Circulatory disturbances
3. Abnormal blood states
4. Gastro-intestinal disturbances
5. Emotional disorders, anxiety, or nervous states
6. Miscellaneous: in certain relaxation states, from caffeine withdrawal, etc.

Independent Forms of Headache, from disorders in

1. Muscles and fibrous structures about the head (Williams)
2. Vascular structures about the head (Williams)

Headaches from Disorders within the Head

1. Intracranial disease
2. Disorders of the eye
3. Nasal space disease

Headaches originating from systemic or general disorders have long been recog nized and are adequately covered in the numerous writings on this subject. A recent small practical volume on the subject of headache is that written by Moench, an internist.

The headaches which seem to be inde-

pendent of systemic disorders or a disorder within the head are grouped according to Williams as disorders in

1. Muscles and fibrous structures about the head

    a. Muscle tension
    b. Fibrositis
    c. Myalgia of the head

and

2. Vascular structures about the head

    a. Migraine
    b. Histamine cephalgia
    c. "Sphenopalatine ganglion neuralgia"

Headache From Muscles And Fibrous Structures About The Head

One has only to recall the anatomy of the muscular and fibrous structure about the head to realize that there are numerous possibilities for factors of tension, fatigue and inflammatory disturbance to operate at these sites.

Muscle tension is a common cause of headache. It may result from the maintenance of the head in certain positions sometimes occupational, tension due to anxiety, chronic fatigue, etc.

Fibrositis has been described as of two types, a primary and a secondary. The latter is very common. It is often associated with arthritic changes in the cervical spine. The headache is localized at the back of the head, particularly at the nuchal line. Chilling as occurs in cold damp weather often causes exacerbation. Mild exercise, physical therapy, and the salicylates improve the symptoms.

Primary fibrositis is said to be present when localized sensitiveness occurs in and around the scalp and head in which the pain tends to be of a bright burning character, with sensitiveness of the scalp to light manipulation.

Myalgia of the head is characterized by a headache. According to Williams it originates in muscle on the basis of a physical or intrinsic allergy. It is characterized by isolated, firm, tender areas in the body of certain muscles of the head and neck. These areas tend to recur in the same locations, and when the pain is present the areas are increasingly tender and the pain is referred in a myotomic distribution. The following muscles have been described as the site of myalgia; the upper border of the trapezius, the splenius capitis, the upper one third of the sterno cleidomastoid, the temporal, the stylohyoid, the anterior belly of the digastric, the cricoarytenoideus posticus, the mylohyoid, the insertion of the glossopalatinus into the tongue, and the superior constrictor of the pharynx.

The sites of reference of pain are usually unilateral.

The sufferer from myalgia is usually extremely sensitive to drafts and atmospheric changes.

Williams suggests that the causation of myalgia is a spasmodic contraction of the arterial limb of capillaries resulting in a localized release of histamine with its vasodilating effect. The use of vasodilators such as nicotinic acid offers relief by their action in opening up the spasmodically contracted arterial limbs.

Headache From Vascular Structures About The Head

Since the basic mechanism of headache is often the stretching of pain sensitive structures, most head pain has something to do with blood vessels. There are three types of headache which according to modern knowledge are clinical entities and more or less set apart from the rest. These are: migraine, histaminic cephalgia and one that we have designated in the past as "sphenopalatine ganglion" neuralgia. The latter, according to modern concepts is probably a vasodilating head pain involving the internal maxillary artery.

Migraine. There seems to be an adequate basis for the hypothesis that migraine is due to uncompensated fluctuations of the effective arterial blood volume. An inherited familial defect allows the development of vascular spasm which is followed by an over dilatation of the extra cranial and intracranial arteries. The most effective treatment consists of the subcutaneous injection of ergotamine tartrate.

Histamine cephalgia is an uncommon type of headache occurring in the third decade or beyond, usually unilateral, periodic, of sudden onset and short duration. The mechanism of this form of headache has been explained on the basis of localized capillary constriction with release of histamine into the adventitia of the wall of the affected artery. Vasomotor rhinitis on the affected side, vasodilation in the skin, injection of the conjunctiva on the involved side and tearing are associated phenomena of autonomic overactivity. The treatment consists of giving vasodilators such as histamine or nicotinic acid although the administration at the height of the attack will temporarily increase the pain.

"Sphenopalatine ganglion neuralgia". Williams and Hilger both suggest that an explanation of this picture can be satisfactorily made by considering it to be a vasodilating pain involving certain branches of the external carotid artery, principally the internal maxillary. I think most of you will agree that at least all previous explanations of this picture of headache were unsatisfactory. Fenton and Larsell, and Higbee, have pointed out that the actual structure of the sphenopalatine ganglion has no sensory nerve fibers although fibers from the maxillary division of the trigeminal nerve pass through it, and that transmission of pain by fibers of the autonomic nervous system can not be confirmed by any investigators. Dysart has suggested that the relief of pain when the sphenopalatine ganglion is anaesthetized might be explained on the basis that there is an interruption of a reflex arc in the sympathetic fibers in the ganglion. Hilger has recently stated that "cocainization of the sphenopalatine ganglion produces relief only if the spastic arteriolar bed is under control of the sympathetic fibers under blockage, or if sufficient intranasal vasoconstrictor is administered to give systemic absorption and direct constrictive influence on the dilated arterial trunk". Williams has reported relief of this so-called "sphenopalatine" pain in mild cases by the use of intramuscular injections of nicotinic acid and in the more severe cases by using the drug intravenously.

Headache from Disorders Within the Head

The third major category in the classification of headache that is suggested is concerned with the headaches that arise from intracranial disease, disorders of the eye, and nasal space disease.

In the early stages of an intracranial disorder, the symptom of headache may compare in its characteristics to the symptoms originating from other disorders within the head such as those caused by the eye or within the nasal space. However, the alert and informed physician in the field of ophthalmolory or of otolaryngology should have no great difficulty in establishing whether or not a problem head pain is caused by a disorder within his field. Probably the most common experience in the rhinologists' activities in dealing with a head pain due to intracranial disorder but suspected of being "a sinus condition" is with the cerebral arteriosclerotic who believes that his sense of fullness, pressure, head congestion or actual pain is caused by "blocked sinuses" and that he feels "clearer" after the nasal mucosa has been treated with a vasoconstrictor.

A skilled ophthalmologist should experience no great difficulty in determining whether or not a headache is due to a refractive error, muscle imbalance, increased intraocular tension, or an inflammatory condition in the eye.

Headache originating in the nasal

space has two possibilities as to its causation. It may be caused by:

1. Contacts within the nasal fossae
2. Disease within a sinus causing pressure or tension.

The areas in which contacts may result in the production of pain are:

1. The nasofrontal area
2. The middle meatus
3. Between the septum and the lateral nasal wall.

The pain produced by these contacts is usually acute and rather temporary except that because of abnormal changes in the tissue, repetition of the pain may become frequent. In my experience, I have never encountered any difficulty in definitely establishing the fact of whether or not a given pain was due to a congestive contact. This can be established by the simple process of cocainization and in some instances by simple vasoconstriction.

McAuliffe, Goodell and Wolff have demonstrated the pain sensitive areas in the nasal space and the regions to which pain originating from various portions of the nasal space is referred. When they stimulated the anterio-inferior portion of the nasal septum, the lateral wall of the maxillary sinus, the middle and inferior turbinates, and the ostium of the maxillary sinus, the pain was usually referred to the malar and zygomatic regions of the homolateral side. Stimulation of the superior portion of the nasal fossa showed reference of pain to the eye, the infraorbital region and the junction of the nasal bridge and frontal bone. When the mucosa of the sphenoid was stimulated, a slight degree of pain was referred to the vertex of the skull.

In our experience, headache from tension or pressure within a sinus is common but disappears as the sinus drains or the swelling subsides. Headache which is a chronic problem is rarely encountered due to tension within a sinus, and when it does occur it is usually a matter of something unusual such as a mucocele or osteoma.

THE NEURALGIAS

Outside of the writings of Sluder who popularized "Sphenopalatine ganglion neuralgia" and Vail who contended that this sphenopalatine neuralgia was probably due to irritation of the Vidian nerve from sphenoid sinus disease, most of the contributions on neuralgia have come from neurologists or neurosurgeons. Their detailed classifications indicated how little was actually understood about some types of head pain.

Today, with the newer knowledge regarding the mechanism of the causation of head pain it would seem possible to reduce the classification of neuralgic head pain into a few simple categories as follows:

1. Trigeminal neuralgia, primary and secondary
2. Glossopharyngeal neuralgia, primary and secondary
3. From irritation of certain branches of
   a. the facial nerve (glossopalatine)
   b. The vagus nerve (auricular, superior laryngeal)
   c. the second and third cervical nerves (occipital nerves, greater auricular N.)

The diagnosis of primary forms of trigeminal neuralgia and of glossopharyngeal neuralgia can practically be made from a description of the symptoms since both are such clear cut clinical entities. There should be little difficulty in diagnosing the "Tic Doloreaux" of these nerves.

Secondary forms of trigeminal neuralgia are encountered practically every day in the practice of otolaryngology. The many causes include dental origins, inflammations, vascular lesions, tumors, trauma, from such diseases as multiple sclerosis, syphilis, etc.

The secondary form of glossopharyngeal neuralgia is most frequently encountered as the aftermath of tonsillectomy in the immediate postoperative period.

Neuralgic disturbances from irritation of certain branches of the facial nerve are uncommon and apparently are limited to inflammations involving the geniculate ganglion. The pain is projected in an area limited to a portion of the tragus, concha, external auditory canal and tympanic membrane. Neuralgic disturbances in the form of earache which occurs through the superior laryngeal branch and auricular branch of the vagus and its sensory branches related to the pharyngeal plexus are not uncommon from malignant lesions on the rim of the larynx, involving the epiglottis or the pharynx.

Neuralgic disturbances involving branches of the second and third cervical nerves are invariably the result of local inflammation, trauma, etc.

Summary

Head pain which is a chronic problem is rarely the result of nasal space disease.

The advances in our knowledge of the etiology of head pain involve three considerations:

1. The basic mechanism of the pro duction of head pain is in many instances a stretching of the pain sensitive nervous structures along the blood vessels.

2. Autonomic dysfunction is a major factor in the production of some forms of head pain.

3. The vasodilating action of histamine released locally in certain forms of tissue change as may occur from physical stimuli and autonomic dysfunction may explain the mechanism by which vasodilation occurs.

On the basis of this newer knowledge, a simplified classification of headache and of neuralgic head pain is suggested.

B. SOME OBSERVATIONS ON THE PATHOLOGICAL PHYSIOLOGY OF VASCULAR HEAD PAIN

Jerome A. Hilger

Let us marshall the observed clinical facts relative to vascular head pain - the true chronic and recurrent problem. As an example vessel we must select an artery which can be kept under tactile observation because it is recognized that the bulk of clinical information gathered about the subject of vascular head pain is a purely subjective, patient description. The anterior temporal branch of the superficial temporal artery is not an impressive vessel in size but it is representative of the average stem from which vascular pain arises. It is superficially located beneath the skin of the temporal region and its peregrinations can be followed objectively and correlated with the subjective expression of the vasodilating phenomenon.

The pain period is preceded by a prodrome of variable duration; the patient knows he is soon to have pain by a definite peculiar sensory change in the area. This may be referred to as the ischemic period. There is usually little surface expression of it; collateral supply in the region is good. Soon pain along the course of the artery begins and mounts in intensity, oftentimes rapidly. The artery becomes distinctly palpable. As the full throbbing intensity reaches its apex the pressure of touch along the course of the artery is ill-tolerated. The vessel is noted to be not only enormously dilated and pulsating but in addition the character of the vessel wall is changing. It is becoming thickened, edematous. It is becoming, through increase in thickness and, presumably, length, tortuous. It is now visible as a tortuous, pulsating snake-like contour. The pain is excruciative, unbearable; it is ill-relieved by opiates. The patient knows gentle, external supportive pressure is as kindly treatment as any. There may be nausea, and other reflex neuro-motor reactions, sweating, flushing, pallor, etc.

As the artery subsides the pain subsides. For long hours afterward, however, the doughy edematous vessel wall remains palpable and severely sensitive to slightest touch. The brushing of the overlying hair, for example, evokes a miserable soreness.

In organizing medical observation and fact into a systematic collection for transmission to others we exhibit a most unfortunate human tendency to "pigeonhole". There comes a point at which the average human brain runs out of slots. The folly of expounding a clinical syndrome expressing the pain of every individual branch of the carotid arterial tree must be apparent. This is particularly true in that all combinations of segmental vascular functional misbehaviors occur, and in the same individual over many years the pattern tends to be a shifting one. Hence one ends with more atypical syndromes than typical. The subject must be taught and dealt with as a neurovascular imbalance with its expression a vasodilating pain and its locale limited only by the anatomic location of the peripheral branches of the carotid artery. The fog of typical and atypical migraines, histaminic cephalgias, pseudo-sinus headache, and atypical neuralgias is thus swept aside and the simple mind can go forward to meet disturbed vascular physiology in whatever locale it may be found.

Whence emanates this disturbed vascular physiology? From irregularities in the autonomic mechanisms which control it. Autonomic balance is a state of homeostatic bliss in which the individual is right with his environment. When he is not right with his environment the resultant autonomic imbalance becomes a clinical problem. The autonomic centers of the hypothalamus comprise the human thermostat. Inherently some are of poor stuff and hence the importance of inheritance and family history of autonomic imbalance in clinical investigation. Too, equally effective central mechanisms subjected to very different degrees of environmental stress break down to a state of imbalance and peripheral expression in proportion to the load they bear.

What comprises environmental stress? Principally it is the physical and emotional climate in which we live. However, inhalant and ingestant entities are truly environment also.

In the area of our disturbed subject artery, what autonomic disturbance produces edema and dilatation of its wall? The answer, incongruously enough, is vasospasm - arteriolar spasm. The functional level of the vascular bed is the arteriolar (capillary) venular unit. The vascular anatomy we teach students is purely a system of highways, meaningless without these vascular units in the periphery. It would be better if we taught them the function of these nameless units and forewent the pleasure of lurid freshmen ditties to systematize the main roads in their mind.

When an arteriole goes into spasm through autonomic motor impulsion, whether the stimulus be from central impulsion or local axon reflex or antigen-antibody interplay at the smooth muscle sphincter, an interesting sequence of events is initiated: the dependent capillary dilates due to ischemic damage to the endothelium and the presence of uncontested reflux venous tonus; fluid transudate leaks between separated endothelial cells into the tissue spaces. This then is the motor unit of wheal formation, the dynamic origin of edema. The biomicroscope has demonstrated it sufficiently frequently so doubt as to its correctness no longer exists.

Where are the arterioles in subject artery whose spasm effects the dilatation of the main trunk? They can be of two kinds. First are those of the artery's peripheral bed whose simultaneous constriction erects a formidable peripheral dam and calls for a proximal circulatory shunt. In the absence of shunt, pulsating arterial force butting a peripheral dam exerts a dilating pressure against the banks of the main stream. Simultaneous with this development one may expect ischemic aura from the tissues of the periphery if collateral arterial supply is not adequate. Peripheral autonomic innervation provides an ana-

tomic basis for segmental arteriolar behaviour. The commonplace blanched single phalanx is a case in point. The second, and probably more important, arteriolar group involved in the vaso-dilating phenomenon is that of the vaso-vasorum of the subject artery wall. The integrity of vascular intima, media, and adventitia is solely dependent upon the vaso-vasoral arteriolar (capillary) venular complexes. The initial contraction of arterial smooth muscle produced by vaso-vasoral spastic ischemia cannot endure for long. The inevitable subsequent arterial muscular flaccidity in the presence of enduring ischemia when impinged by the shunt complexities detailed above lays the mechanical ground work for dilatation of the arterial trunk. The edema of the subject artery wall resulting from this autonomic crisis of the vaso-vasoral arteriolar (capillary) venular periphery is that which is felt as a doughy thickening of the arterial wall beneath the temporal skin. It is safe to project that the final obliterative change of temporal arteritis is the end product of exceptionally severe and intractably persistent vaso-vasoral ischemia.

In this same manner it is apparent that many of the clinical phenomena we see and relegate to the "itis" or "idiopathic" categories have their source in the functional behaviour of the arteriolar units. Segmental behaviour can produce syndromes ranging from tic to epilepsy. It is a fascinating horizon and need not be limited to the pain phenomena of the dilated arterial wall.

THERAPY

Therapy which has stood up best through the empiric years is that which produces autonomic blockade at central, gnaglion, or myoneural levels or that which influences smooth muscle sphincteric action directly or that which repairs the ischemically damaged endothelium. The drugs are legion. Recent years have brought a flood of new and useful agents. More are in process. Grouping for clarity of usage according to level of action they include: at the central level, sedation; at the ganglion level, local ganglion block with procaine or dolamine, excision, tetraethylammonium chloride, etc.; at the myoneural junction, the antihistamine group, artane, bantine, ergotamine derivatives, dibenamine, intravenous procaine, etc.; at the smooth muscle directly, nicotinic acid, papaverine, the nitritoid group, histamine, procaine amide, etc.; on endothelium, the steroids, thyroid, and ascorbic acid.

Drugs have secondary purpose in treatment. Making the individual right with his environment is fundamental. The whole of the psychosomatic approach is basic.

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II. MEDICAL SCHOOL NEWS

Coming Events

January 4 - 6 Continuation Course in Geriatrics for Physicians
January 22 - 26 Continuation Course in Ophthalmology for Specialists
Jan. 29 - Feb. 10 Continuation Course in Clinical Neurology for General Physicians, Internists, and Pediatricians
February 15 - 17 Continuation Course in Cardiovascular Diseases for General Physicians

* * *

The Importance of Mitral Insufficiency in Surgery on the Mitral Valve

Surgical attack upon the stenosed mitral valve of the "fish-mouth" variety has been revived recently with encouraging results. How long the beneficial effects of commissurotomy will last in a given patient is not yet known. Sufficient experience has been gained, however, to point out that a limited number of persons with mitral valvular disease are suitable candidates for this procedure.

Exertional dyspnea, the common disability in mitral disease, depends largely upon the severity and duration of hypertension in the pulmonary circuit. Mitral insufficiency, either alone or in combination with varying degrees of mitral stenosis, is known to produce pulmonary hypertension as often as pure mitral stenosis.

Direct pressures recorded simultaneously from the left atrium and pulmonary artery at the time of surgery permit an accurate prediction as to outcome.[1] Left atrial pressure curves containing "W"-shaped contours during ventricular systole are indicative of mitral insufficiency. Increasing the regurgitation by commissurotomy usually ends fatally. Even thoracotomy without commissurotomy is poorly tolerated. In pure mitral stenosis, on the other hand, left atrial pressure curves are relatively flat and show little evidence of regurgitation following successful commissurotomy. Improvement in symptoms is well correlated with the lowering of pressure in both the left atrium and the pulmonary artery and the establishment of a more normal pressure gradient across the pulmonary circuit.

This knowledge restricts the procedure to instances of pure advanced mitral stenosis and points up the frequency with which insufficiency is associated with stenosis in the majority of patients with mitral valvular disease. Because the diastolic murmur of mitral stenosis is diagnostic, an associated systolic murmur may not be evaluated. In selecting patients for surgery, not only is careful auscultation indicated, but mechanical aids such as phonocardiography and elecktrokymography must be enlisted in an attempt to detect and assess the degree of mitral insufficiency.

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-- Craig Borden, M.D.

* * *

Faculty News

Dr. Reynold A. Jensen, Associate Professor of Psychiatry and Pediatrics, attended the Mid-Century White House Conference on children and youth in Washington, D. C. Dr. Jensen, who is chairman of the American Psychiatric Association Committee on Child Psychiatry, attended the conference as a representative of that professional organization.

* * *

Correction

In the Bulletin of Friday, December 8, 1950, page 138, the sentence beginning in the last lines of column one should read, "He admits, however, that under conditions of warfare when patients must be evacuated and where the skill of the surgeons in rear installations is not known that colostomy and drainage of the perirectal spaces is advisable."

III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
CALENDAR OF EVENTS

Visitors Welcome

December 17 - December 23, 1950

Sunday, December 17

University Hospitals

9:00 - 10:00 Surgery Grand Rounds; Station 22.

10:30 - Surgical Conference; Todd Amphitheater.

Monday, December 18

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.

4:00 - 5:00 Pediatric Seminar; Adrenal Gland; Paul Adams; 6th Floor West, U. H.

4:00 - Public Health Seminar; 113 Medical Sciences.

4:30 - 5:30 Dermatological Seminar; M-436, U. H.

5:00 - 5:50 Clinical Medical Pathologic Conference; Todd Amphitheater, U. H.

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

Minneapolis General Hospital

9:00 - 10:00 Pediatric Rounds; Dr. Lowry; 5th Floor.

1:00 - 2:00 Staff Meeting; Classroom, 4th Floor.

2:00 - 3:00 Journal Club; Classroom, Station I.

Veterans Administration Hospital

9:00 - G. I. Rounds; R. V. Ebert, J. A. Wilson, Norman Shrifter; Bldg. I.

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

Monday, December 18 (Cont.)

Veterans Administration Hospital (Cont.)

1:00 - Metabolic Disease Rounds; N. E. Jacobson and G. V. Loomis; Bldg. I.

4:00 - Research Conference; Conference Room, Bldg. I.

Tuesday, December 19

Medical School and University Hospitals

9:00 - 9:50 Roentgenology Pediatric Conference; L. G. Rigler, I. McQuarrie and Staffs; Eustis Amphitheater, U. H.

9:00 - 12:00 Cardiovascular Rounds; Station 30, U. H.

12:30 - 1:20 Pathology Conference; Autopsies; J. R Dawson and Staff; 102 I. A.

3:15 - 4:20 Gynecology Chart Conference; J. L. McKelvey and Staff; Station 54, U. H.

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

*8:00 p.m. Minnesota Pathological Society Meeting; The Experimental Production of Heart Disease; The Surgical Management of Certain Acquired and Congenital Forms; C. W. Lillehei, I. D. Baronofsky, R. L. Varco; Medical Science Amphitheater.

Ancker Hospital

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

Minneapolis General Hospital

8:00 - 9:00 Pediatric Rounds; Dr. Adams; 4th Floor.

8:30 - Pediatric Allergy Rounds; Dr. Nelson; 4th Floor.

9:00 - 10:00 Pediatric Rounds; F. H. Top; 7th Floor.

Veterans Administration Hospital

8:45 - Surgery Journal Club; Conference Room; Bldg. I.

8:30 - 10:20 Surgery Conference; Seminar Conference Room, Bldg. I.

9:00 - Infectious Disease Rounds; W. Hall.

9:30 - Surgery-Pathology Conference; Conference Room, Bldg. I.

10:30 - 11:50 Surgical Pathological Conference; Lyle Hay and E. T. Bell.

10:30 - Surgery Tumor Conference; Conference Room, Bldg. I.

1:00 - Chest Surgery Conference; J. Kinsella and Wm. Tucker; Conference Room, Bldg. I.

Tuesday, December 19 (Cont.)

Veterans Administration Hospital (Cont.)

1:30 - Liver Rounds; Samuel Nesbitt.

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

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

Wednesday, December 20

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; Medicine Case; O. H. Wangensteen, C. J. Watson and Staffs; Todd Amphitheater, U. H.

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

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

8:00 p.m. Dermatological Pathology Conference; 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

9:00 - 10:00 Pediatric Rounds; Dr. Lowry; 5th Floor.

12:15 - Staff Meeting; Classroom, 4th Floor.

3:00 - 4:00 Pediatric Rounds; E. J. Huenekens; 4th Floor.

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

Veterans Administration Hospital

8:30 - 10:00 Orthopedic-Roentgenologic Conference; Edward T. Evans and Bernard O'Loughlin; 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 21

Medical School and University Hospitals

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.

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

7:30 - 9:30 Pediatrics 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; Forrest Adams; 4th Floor.

9:00 - 10:00 Pediatric Rounds; F. H. Top; 7th Floor.

10:00 - Pediatric Rounds; Adult Contagion

11:00 - 12:00 Clinical Pathology Conference; Large Classroom.

11:30 - Pediatric Conference; Main Classroom.

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

2:00 - EKG and X-ray Conference; Classroom, Station I.

Veterans Administration Hospital

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

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

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

1:00 - Chest Rounds; William Stead.

Friday, December 22

Medical School and University Hospitals

8:30 - 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.

11:00 - 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.

11:45 - 12:50 University of Minnesota Hospitals Staff Meeting; Surgical Therapy of Congenital Megacolon; David State; Powell Hall Amphitheater.

Friday, December 22 (Cont.)

Medical School and University Hospitals (Cont.)

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

2:00 - 3:00 Dermatology and Syphilology Conference; Presentation of Selected

2:00 - 4:00 Physiology Conference; 214 Millard Hall.

3:00 - 5:00 Neuropathology Conference; F. Tichy; Todd Amphitheater, U. H.

4:00 - 5:00 Clinical Pathological Conference; A. B. Baker; Todd Amphitheater, U. H.

4:15 - 5:15 Electrocardiographic Conference; 106 Temp. Bldg., Hospital Court, U. H.

Ancker Hospital

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

Minneapolis General Hospital

9:00 - 10:00 Pediatric Rounds; Dr. Lowry; 5th Floor.

9:30 - Surgery-Pediatric Conference; O. S. Wyatt & T. C. Chisholm; 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. Myers; Ward 62, Day Room.

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

Saturday, December 23

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; O. H. Wangensteen and Staff; Todd Amphitheater, U. H.

Saturday, December 23 (Cont.)

Medical School and University Hospitals

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.

Ancker Hospital

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

Minneapolis General Hospital

8:00 - Pediatric Rounds; Forrest Adams; 4th Floor.

9:00 - 10:00 Pediatric Rounds; F. H. Top; 7th Floor.

11:00 - 12:00 Pediatric Clinic; Charles May; Classroom, 4th Floor.

Veterans Administration Hospital

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

8:30 - Hematology Rounds; P. Hagen and E. F. Englund.

* 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.