Bulletin of the

University of Minnesota Hospitals
and
Minnesota Medical Foundation

The Nature of Bell's Palsy

Volume XXI | Friday, October 28, 1949 | Number 5

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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. THE NATURE OF BELL'S PALSY

Jerome A. Hilger

When an apparently normal and healthy individual awakens in the morning and finds as he glances in his shaving mirror that he has lost the power of movement of one side of his face, he is said to be suffering from Bell's palsy. This is not, however, the condition originally described by Mr. Charles Bell, F.R.S., before the Royal Society in London in 1892[1]. At this time his concern was with the proper allocating of motor and sensory nerve distribution to the facial region, because of the accepted practice of dividing the peripheral trunk of the seventh cranial nerve for the relief of tic douloureux. Through various case histories and experiments he brought forth conclusive evidence that the "portio dura" of the seventh nerve had purely motor function and had no place in the genesis of facial pain. His case included peripheral facial paralysis due to wound from a pistol ball; due to injury from the horn of an ox; and due to operation for extirpation of a tumor before the ear. His careful research, thoughtful clinical observation, and compelling presentation established the concept of the motor seventh and sensory fifth which is today accepted as a matter of course. In the intervening century peripheral facial paralysis has been shown to result from a variety of causes including trauma, infection, and neoplastic compression. Still there exists a formidable number of cases not ascribable to any of these agents. It is this residual group which today, for want of an etiologic description, retains the title of Bell's palsy.

Many facts are known about this residual group. The paralysis is rather sudden in onset - usually a matter of hours. Though it has been described as painless, actually pain about the ear is a common symptom in the first forty-eight hours. Sex has no influence. The adult age group is most commonly afflicted. There is a definite familial incidence. Predisposition as evidenced by recurrences has some importance[2,3]. There is no apparent seasonal incidence[4]. Abnormal cooling of the auricular region has been cited as a cause in as high as seventy per cent of cases[5]. In the absence of this physical exposure, severe emotional upset or shock has been shown to be a precipitating factor[5].

Repeated thorough general examination has failed to show common systemic organic causation. There are no constant local findings in the peripheral course of the facial nerve, though it is surprising how commonly the vessels of the posterior aspect of the deep portion of the external auditory canal and of the tympanic membrane show notable injection in the first days of involvement. The nerve may be involved in a segment to include the stapedial and the chorda tympani branches or involvement may be below that level. Paralysis may be partial or complete. It may be permanent. Faradic response may disappear in ten days or two weeks or recovery may occur without the loss of faradism. There is no established prognostic sign other than the completeness of paralysis or the loss of faradism. There is no unanimity as to etiology or treatment - medical or surgical.

Until recent years the actual changes in the nerve trunk which destroyed its conductive function were largely a matter of conjecture. Few reports of the microscopic changes in the nerve were available and few gross observations of the diseased nerve had been made. Following the basic work of Ballance and Duell[6] and encouraged by their dictum to operate and decompress the nerve once paralysis is established, many cases of Bell's palsy have come under surgical care and there has been opportunity to grossly observe the disordered trunk in various periods after the onset of paralysis[3,7,8]. There is uniform agreement that the usual finding upon surgical exposure is edema of the nerve with the trunk most tightly constricted by the nerve sheath at its point of issuance from the stylomastoid foramen. Edema is not a universal finding, however. The nerve trunk may have a normal

macroscopic appearance. Kettel[3] has observed further that aseptic necrosis of adjoining portions of the temporal bone presumably due to ischemia is not uncommonly present. This has not been confirmed by others with significant experience.

Compression due to edematous swelling suggests an obvious source of paralysis. Actually failure of nerve conduction due to change in a localized segment of nerve trunk does not result from compression unless the latter is sufficiently severe to disrupt the axis cylinder. This degree of compression does not occur clinically. Denny-Brown and Brenner[9,10] have shown that conduction failure is a result of ischemia as a primary cause, not compression. Hence the paralysis of the facial nerve is a result of local vascular change which by ischemia has interrupted conduction and by attendant changes in the local circulation may or may not produce edema. If edema does form, the resulting compression may make the problem of relieving ischemia more difficult.

In a patient in apparent good health the question may be asked, "why and by what mechanism is ischemia and edema produced in so localized a region?" It is apparent that it is not dependent on change in the blood elements, or in blood chemistry, or in the permeability of endothelial tissues generally. To be thus localized it must result from dynamic local vascular change. The probable mechanism has been described and given experimental basis by Abell and Schenck[11]. The essential features are arteriolar constriction; followed by capillary dilatation due to ischemic damage or reflux from venous tonus; a widening of space between endothelial cells[12]; and resultant transudation. To fully understand this sequence the basic work of Krogh[13] on capillaries and Hudack and McMasters[14,15] on lymph capillaries must be appreciated. The pressure of fluid transudate is rapidly transmitted to the walls of the lymph capillaries and they may be closed by compression. Additional fluid then accumulates and compression capillaries and venules creates further zonal ischemia and a blanched wheal results. This dynamic mechanism of ischemia and edema formation has been shown to result from the precipitant influence of physical exposure or emotional stimuli in certain individuals who have an inherent tendency for this eccentric behaviour in their arterioles[16]. The impetus to arteriolar constriction in such instance is derived from autonomic nervous impulsion to the involved vessel. It is probable that the tendency is actually inherited through the inheritance of one's autonomic system and its potential imbalances. These imbalances in the head segments are expressed in large part through vagaries in the function of the carotid arterial tree. A high percentage of patients with Bell's palsy give history of other manifestation of carotid vasomotor disturbance: chronic vasomotor rhinitis; vasodilating pain; cervical myalgia; or vasomotor labyrinthitis.

The vessels of most vital physiologic import in any arterial trunk system are the arterioles. Because of the anatomy of their autonomic innervation in the peripheral vascular areas arterioles tend to behave in segmental arterial branch fashion rather than in diffuse, haphazard manner. When the arterioles of a small segmental branch of the carotid tree constrict simulataneously the resultant ischemia produces interesting clinical conditions in the tissues supplied by that branch. The conditions may vary widely according to the cranial tissue involved. In the case of end arteries without collaterals a peripheral cataclysm may occur as, for example, in the inner ear[17]. In a confined anatomic situation where associated edema is able by compression to prevent collateral arterial supply an analagous condition is created. Edema within the firm fibrous sheath of the facial nerve in the region of the stylomastoid foramen is an example.

There is a collateral arterial supply which can modify the ischemia of the facial nerve produced by widespread arteriolar constriction of the stylomastoid arterial bed. There are two principal anastomoses. The first is that of the terminal branch of the stylomastoid artery

with the superior petrosal branch of the middle meningeal through the hiatus for the great superficial petrosal nerve. The second is that of the posterior tympanic branch of the stylomastoid artery which leaves the facial canal with the chorda tympani and in the posterior aspect of the tympanum and tympanic membrane anastomoses with other branch arteries to the tympanum from the internal maxillary, the ascending pharyngeal, the middle meningeal, and the carotid arteries.

Hyperacusis resulting from stapedial nerve paralysis and the loss of taste sensation due to chorda tympani involvement are common in the early period of a Bell's palsy. Restoration of the normal function of these two branches commonly occurs in spite of continued facial paralysis. It probably results from early relief of ischemia at the knee of the facial nerve due to these two anastomoses. There is little or no collateral supply to relieve the ischemia below this level.

Spasm in arteriolar terminals can produce dilatation in their arterial trunk. This dilatation in the fine arterial segments of the posterior tympanic branch of the stylomastoid artery accounts for the injection in the region of the posterior annulus frequently seen in the first days of ischemia and palsy. Segmental peripheral spasm may be sufficiently widespread to cause retrograde arterial dilatation in larger branches and in the stylomastoid artery itself. The vasodilating pain produced is that not uncommonly seen with the onset of Bell's palsy. It usually denotes ultimate severe ischemic involvement.

Motor conduction through the ischemic nerve trunk is interrupted when ischemia results in destruction of myelin sheath[9,10]. This appears first at the nodes of Ranvier. The reaction of degeneration, on the other hand, will not be present until ischemic necrosis of the axon occurs. This clinically occurs most frequently after ten to fourteen days. By this time the most advanced degree of ischemic functional damage is present and a prolonged course is assured. Recovery from this phase commonly results in associated facial movements because it is possible for regenerating axons to cross the necrotic area and progress into wrong myelin tubules.

Since this paralysis is an ischemic neuritis, its relief may be expected to hinge upon the restoration of circulation in the involved stylomastoid arterial segments. One is derelict if he regards the onset of a Bell's palsy as other than a medical emergency. Immediate non-surgical measures must be directed toward relief of arteriolar spasm. These may embrance vasodilating drugs with peripheral effect such as nicotinic acid, intravenous histamine, or papaverine, or with sympathetic paralyzing effect as ergotamine tartrate or tetraethylammonium chloride; or cervical sympathetic block. The ischemia of the vertical segment of the nerve trunk must be countered through the constricted arteriolar bed since collateral supply is not present.

Edema which appears as a result of arteriolar spasm may prevent the restoration of normal circulation in spite of the most aggressive medical measures. Surgical decompression of the nerve trunk in its course in the vertical bony canal can obviate this factor. In the very nature of surgical trauma to the local area of arteriolar constriction, vasodilatation may be reflexly stimulated. In addition release of the constricting sheath relaxes pressure on lymph and blood capillaries and arterioles and venules and mechanically facilitates subsequent vasodilating therapy. However, the majority of cases get eventual full return of function without this major surgical procedure. There is no established method for differentiating the case of simple ischemic demyelinization from that which is progressing to axon necrosis. And yet the means to differentiate are possibly at hand and want only for lack of application. One can hardly gainsay the lack of good sense in deeming all Bell palsies surgical problems. Yet one cannot fail to see the tragedy of permitting ischemic damage to progress to axon necrosis. Full recovery from the latter stage may be regarded as a happy accident. The permanent deformi-

ty of associated facial movements or persistence of partial or total paralysis is too likely.

Assume an ischemic facial paralysis is properly recognized by the first examining doctor as a real medical emergency. It is likely that the immediate application of intravenous vasodilating measures and the inhibition of sympathetic overactivity can reverse the circle of ischemia, edema, and further ischemia to the limit of our present medical means. If paralysis persists axon necrosis may be developing. Faradic stimulation of the nerve trunks has been commonly used to determine the onset of axon necrosis. It is a crude mass stimulation and when once it shows the reaction of degeneration to be present the damage to the axon has been done. The opportunity for maximal recovery through the addition of surgical decompression to medical circulatory restoration has passed. A more refined test of beginning axon necrosis is available through the techniques of electromyography. As the axon necroses, fibrillation of the individual motor unit appears. With such evidence at hand despite the best of non-surgical measures, decompression should not be further postponed, else one bears the responsibility of incomplete therapy when permanent deformity results.

In our social order the deformity of residual facial paralysis constitutes a grave disability.

Summary

Bell's palsy is an ischemic neuritis.

It results from segmental arteriolar spasm.

The latter may also produce edema as a secondary phenomenon.

Therapy should be directed toward the relief of vasospasm.

In some cases it may be necessary to facilitate vasodilating therapy by surgical decompression of the edematous nerve trunk.

Surgical decompression should be instituted with the first appearance of axon necrosis.

Axon necrosis is detectable early while a minimum of motor units are involved through the techniques of electromyography.

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II. MEDICAL PROGRESS THROUGH RESEARCH

Owen H. Wangensteen

Research is the life-line of progress in every undertaking, which has as its objective betterment of the condition of man. Scientific research has opened up new vistas of life and has recreated our entire manner of living. Today, the pauper enjoys luxuries denied kings of half a century ago, because of the contributions of science and research to the conveniences of life, which most of us are quick to regard as necessities. However much all these gains have improved the condition of man, there is little evidence that they have made man happier. A capacity for happiness is something which man must cultivate within himself.

Contemplation of the great advances, which have come in medicine through the agency of research during the one hundred years which have elapsed since Minnesota became a State, suggests that hope and confidence have replaced despair in many diseases. Were it not that an inscrutable aura of ignorance surrounds so many problems in medicine, we might well share a feeling of complacent satisfaction for what has been accomplished. With reference to many illnesses which affect man, there is good reason for rejoicing over what research has accomplished; no one has ever sensed real pleasure who has not, in its enjoyment, experienced a feeling of gratitude. Let us be grateful for what has been done; at the same time our faces must be turned with determination toward the large tasks of bringing light into those areas now pervaded by darkness.

Research in medicine is a field of endeavor which touches the life of every living person many times during his journey from the cradle to the grave. Yet, there are some amongst us who affect no interest in medical research; in fact, a few even oppose animal experimentation. The day of hypotheses, postulations and theories unverified by experimentation in medicine is by. And the only effective manner in which the shackles of speculative rationalization can be severed in the pursuit of biologic knowledge is through the agency of the testing of ideas by experimentation upon animals. Medical research has saved more lives than have been lost in all the wars since the beginning of time. When man no longer slays animals for food or clothing or holds them subservient to his will, the significant evidence learned in experiments upon animals will fully justify their performance for the protection and prolongation of human life.

Our Legislative Body in Minnesota recognized the intimate dependence of medical progress upon medical research in the recent enactment of a law making it possible for educational institutions prosecuting medical research to obtain animals from pounds for purposes of experimentation, where such animals previously were destroyed.

The rise of this Medical School from mediocrity to recognition as a true center of learning is owing largely to a growing interest in research. Teaching, which fails to distill into the minds of young learners the hopeful prospects of research, leads to intellectual starvation. It is the function of Universities, not alone to teach what is known but to strive to give accelerated momentum to the enlargement of knowledge. Only since the members of the faculty of this Medical School looked upon this function as an important duty and a privilege, only since then has this Medical School grown to full stature, reawakened by the animated and refreshing spirit of research which breathed new and invigorated life into its being.

The fundamental studies on factors underlying growth processes initiated in the Department of Anatomy in this Medical School more than 30 years ago helped to earn it the reputation of being one of the outstanding faculties of anatomy in the world. The teaching was of a high order, made so in part by the captivating spirit of enthusiasm of teachers intent upon contributing to the patrimony of knowledge. The contagion of that influence broadened the vision and the horizon of the students as well as colleagues in other departments. Today, active research is in progress in every

major teaching division of the Medical School. Transmitting passively the lifeless body of knowledge of the past to students of succeeding generations is not enough. The leaders in medicine of the future must receive the nurture of stimulating instruction at the hands of teachers whose thinking is being sharpened by active conflict with medical problems, crying out for solution. Students who have had first hand training in this type of discipline learn early to ask: what is the evidence? They are not likely to confound dogmatism and fact or empiricism and proof.

Studies prosecuted in various Departments of this Medical School upon: the nature of fatigue, the heart and the circulation in health as well as in disease, disturbances of respiration, cancer biology, nutritional disorders, metabolism in its broad aspects, endocrine disorders, diseases of the nervous system, fevers and infectious diseases, affections of the kidney, liver and bile passages as well as those of the alimentary tract, such studies have played an integral part in shedding additional light upon many important problems. In many areas still, there is great need for more luminous light which will permit clearer vision and a more intelligible comprehension.

Understanding the nature of a difficult problem comes rarely, if ever, all at once. The luminescent flash of a firefly, the dimness of candlelight, and the incandescence of the electric light reflect relative differences in the luminosity of our present understanding of many pressing problems. Persistent fact-gathering through research is the only instrument which can dispel the veil of darkness which obscures our vision and defies our understanding of perplexing medical enigmas. Institutions which count amongst its faculty ardent seekers after light -- such institutions will be bringers of the light. In Universities, where the lamp of learning burns brightly, and the fervor of enthusiastic interest at the forge of its workers does not go out -- such institutions irradiate an influence which brightens the lives of all who live within its shadow. Into the lives of those, who become completely absorbed in its work and life, a leaven is projected, which like the after-glow of sunset in the western sky brings brief light again to a dying day. And long after the feverish activity of such participation is past, the memory is easily stirred to recollection of pleasant labors, which can bring comfort and joy into distant cheerless days.

Research exerts a catalytic effect upon the growth of knowledge; in no small measure, that result is owing in part to the side chain reactions of an augmented student interest and the enlarged perspective of an institution, sensitive and alert to its responsibilities. The people of Minnesota have an important stake in its institutions. Let us, who enjoy the opportunity which their struggles and wise planning have provided, strive to have our institutions merit the great pride with which the democratic people of Minnesota regard them.

The faculty of the Medical School, mindful of its great trust, is endeavoring to justify the faith of the people of Minnesota in their assurance that medical research brings better medicine to them and added luster to their Medical School and University.

Ten years ago, on the occasion of the celebration of the fiftieth anniversary of the founding of the Medical School, the Minnesota Medical Foundation was organized by members of the Medical Alumni Association in the firm belief that the future of the School was linked directly with the character of the support given medical research, within and without the walls of the University. With the years, that belief has crystallized into conviction. As long as research is regarded by the faculty as one of its important functions, the future of the Medical School of the University of Minnesota is secure. In the final analysis, adequate support of research becomes the responsibility of those who are the recipients of its benefactions. Medical research has returned usurious dividends to society. We can well afford to support it generously.

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III. MEDICAL SCHOOL NEWS

Coming Events

October 31-November 5 - Continuation Course in Pediatric Roentgenology for Pediatricians and Roentgenologists.

Monday, October 31, - 4:00 p.m. - Dr. Lawson Wilkins, Johns Hopkins University Medical School - "Abnormalities and Variations in Sexual Development and the Methods Used in Diagnosis," Medical Science Amphitheater.

Wednesday, November 2 - 8:15 p.m. - Rigler Lecture in Roentgenology - Dr. John Caffey, Columbia University Medical Center - "Some Normal Variations in the Growing Skeleton: Their clinical Significance" - Museum of Natural History Auditorium.

November 10-12 - Continuation Course in Pediatric and Traumatic Surgery for General Physicians.

Wednesday, November 16 - 3:00 p.m. - Dr. Frank H. Lahey, Boston, Massachusetts - "A Surgical Clinic" - Medical Science Amphitheater.

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Faculty News

Nursing Instructors who have recently resigned from our faculty to accept positions elsewhere:

Myrtle I. Brown, formerly an Instructor in pediatric nursing, has joined the World Health Organization as nursing consultant in maternal and child care in India.

Miss Myrtle Kitchell, former Instructor in nursing education, has recently accepted the position as Dean of the School of Nursing in the State University of Iowa.

Miss Ruth Weise recently resigned as Instructor in advanced clinical operating room nursing to enroll in the University of Minnesota Graduate School for work in educational psychology.

Radiologists Visit Campus

Distinguished radiologists who will participate as members of the faculty in the continuation course in Pediatric-Roentgenology, October 21 through November 5, will include Dr. John Caffey, Babies Hospital, Columbia University Medical Center; Dr. Edward B. D. Neuhauser, Children's Hospital, Boston; Dr. Edith Potter, University of Chicago; and Dr. Frederic N. Silverman, Children's Hospital, Cincinnati.

Dr. Caffey will also give the annual Leo G. Rigler Lecture on Wednesday, November 2, at 8:00 p.m. in the auditorium of the Museum of Natural History.

Dr. Caffey has chosen "Some Normal Variations in the Growing Skeleton: Their Clinical Significance", as the subject for the Rigler Lecture.

Registrants for the course are invited to attend the meeting of the Minnesota Radiological Society on the evening of Friday, November 4, at the Minneapolis Athletic Club. Both Dr. John Caffey and Dr. Edward Neuhauser will speak at this meeting.

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We are pleased to publish in this issue of the Bulletin a short paper, "Medical Progress Through Research", by Dr. Owen H. Wangensteen, president of the Minnesota Medical Foundation. Portions of Dr. Wangensteen's paper recently were published in the Minnesota-Voice of the Alumni, monthly journal of the Minnesota Alumni Association. We are pleased to publish it in the Bulletin in its entirety because we feel that it contains an inspiring message for the entire medical profession.

III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
CALENDAR OF EVENTS

October 30 - November 5, 1949

No. 263

Sunday, October 30

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

10:30 - 11:00 Subject to be announced; Rm. M-109, U. H.

Monday, October 31

8:00 - Fracture Rounds; A. A. Zierold and Staff; Ward A, Minneapolis General Hospital.

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 - 11:50 Physical Medicine Seminar; E-101, U. H.

11:00 - 11:50 Roentgenology-Medicine Conference; Veterans Hospital.

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

12:00 - 1:00 Physiology Seminar; Experimental Endocarditis; Drs. C. W. Lillehei and J. R. R. Bobb; 214 M. H.

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

12:30 - 1:20 Pathology Seminar; 104 I. A.

12:30 - 1:30 Surgery Problem Case Conference; A. A. Zierold, C. Dennis and Staff; Small Classroom, Minneapolis General Hospital.

1:30 - 2:30 Surgery Grand Rounds; A. A. Zierold, C. Dennis and Staff; Minneapolis General Hospital.

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

4:00 - Public Health Seminar; Subject to be announced; 113 Medical Sciences.

*4:00 - Special Lecture - Abnormalities and Variations in Sexual Development and the Methods Used in Diagnosis - Lawson Wilkins; Med, Sc. Amph.

4:00 - Medical-Surgical Conference; Vagotomy; Dr. Owens; Bldg. I, Main Conference Room.

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

5:00 - 6:00 Urology-Roentgenology Conference; D. Creevy, O. J. Baggenstoss and Staffs; M-109, U. H.

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* Indicates special meeting. All other meetings occur regularly each week at the same time on the same day. Meeting place may vary from week to week for some conferences.

Tuesday, November 1

8:15 - 9:00 RoentgenologypSurgical-Pathology Conference; Craig Freeman and L. G. Rigler; M-109, U. H.

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

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

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

12:30 - Pediatric-Surgery Rounds; Sta. I, Minneapolis General Hospital; Drs. Stoesser, Wyatt, Chisholm, McNelson and Dennis.

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

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

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

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

3:30 - 4:20 Clinical Pathological Conference; Staff; Veterans Hospital.

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

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

5:00 - 6:00 X-ray Conference; Presentation of Cases by University Hospitals Staff; Todd Amphitheater, U. H.

Wednesday, November 2

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-515, U. H.

8:30 - 9:30 Clinico-Pathological Conference; Auditorium, Ancker Hospital.

8:30 - 10:00 Orthopedic-Roentgenologic Conference; Edward T. Evans, Room 1AW, Veterans Hospital.

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

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

11:00 - 12:00 Electrocardiography Lecture; Rhythm and Arrhythmias; Dr. Berman; Main Conference Room, Veterans Hospital.

12:00 - 1:00 Radio-Isotope Seminar; 113 Medical Science Bldg.

3:30 - 4:30 Journal Club; Surgery Office, Ancker Hospital.

4:00 - 5:00 Infectious Disease Rounds; General Hospital, Basement Amphitheater.

5:00 - 5:50 Urology-Pathological Conference; C. D. Creevy & Staff; E-101, U. H.

Thursday, November 3

8:30 - 10:20 Surgery Grand Rounds; Lyle Hay and Staff; Veterans 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.

10:30 - 11:50 Surgery-Radiology Conference; Daniel Fink and Lyle Hay; Veterans Hospital.

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

11:30 - 12:30 Clinical Pathology Conference; Steven Barron, C. Dennis, George Fahr, A. V. Stoesser and Staffs; Large Classroom, Minneapolis General Hospital.

12:00 - 1:00 Physiological Chemistry Seminar; Anti-ACTC; Clay E. Pardo, Jr.; 214 M. H.

1:00 - 1:50 Fracture Conference; A. A. Zierold and Staff; Minneapolis General Hospital.

2:00 - 3:00 Errors Conference; A. A. Zierold, C. Dennis and Staff; Large Classroom, Minneapolis General Hospital.

4:15 - 5:00 Bacteriology and Immunology Seminar; Factors Influencing Natural Resistance to Influenza Virus Infection; 214 M. H.

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

Friday, November 4

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.

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

10:30 - 11:20 Medicine Grand Rounds; Veterans Hospital.

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

11:00 - 12:00 Surgery-Pediatric Conference; C. Dennis, O. S. Wyatt, A. V. Stoesser and Staffs; Minneapolis General Hospital.

11:45 - 12:50 University of Minnesota Hospitals General Staff Meeting; The Significance, Incidence, and Treatment of Adenomas of the Rectum and Recto-Sigmoid Colon; Harry Christianson and Robert Tenner; Powell Hall Amphitheater.

12:00 - 1:00 Surgery Clinical Pathological Conference; Clarence Dennis and Staff; Large Classroom, Minneapolis General Hospital.

1:00 - 1:50 Dermatology and Syphilology; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U. H.

1:00 - 3:00 Pathology-Surgery Conference; Auditorium, Ancker Hospital.

Friday, November 4 (Cont.)

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

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

4:00 - 5:00 Electrocardiographic Conference; George N. Aagaard; 106 Temp. Bldg., Hospital Court, U. H.

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

5:00 - 6:00 Otolaryngology Seminar; Review of Current Literature; Dr. Frey; Todd Memorial Room, U. H.

Saturday, November 5

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

8:00 - 9:00 Pediatric Psychiatric Rounds; Reynold Jensen; 6th Floor, West Wing; U. H.

8:00 - 9:00 Surgery Literature Conference; Clarence Dennis and Staff; Small Classroom, Minneapolis General Hospital.

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

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 Amph., U. H.

9:00 - 11:30 Surgery-Roentgenology Conference; Todd Amphitheater, U. H.

9:00 - 11:30 Psychiatry Conference; Insulin Therapy; Dr. Simon; Veterans Hospital.

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.

11:00 - 12:00 Anatomy Seminar; The Value of Aspiration of Bone Marrow; R. Dorothy Sundberg; 226 I. A.