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Spontaneous Thrombosis of the Carotid Arteries

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I. SPONTANEOUS THROMBOSIS OF THE CAROTID ARTERIES

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The impetus to the diagnosis of spontaneous thrombosis of the carotid arteries was provided by the introduction of angiography by Egas Moniz[1] in 1927 and the subsequent report of Moniz and his associates[2] in 1937 on four cases of nontraumatic thrombosis of the internal carotid artery among 537 patients submitted to angiography. From that time numerous cases have been reported from Germany, Scandinavia and Portugal and more recently the United States and Canada. A number of case reports and reviews of this subject are provided in the literature. The reader is referred to papers by Andrell[3], Sorgo[4], Gladston et al[5], Wolfe[6], Ameli and Ashby[7], Gurdjian and Webster[8], Webster et al[9], Fisher[10], Johnson and Walker[11], and Elvidge and Werner[12]. Johnson and Walker[11] were able to record 101 cases from the literature diagnosed by angiogram or open exposure, and to these they added six cases of their own. Antedating the introduction of angiography, the papers of Chiari[13] and Hunt[14] focused attention on this problem. Chiari[13] in 1905 found that emboli can break away from thrombus material lying in the carotid sinus and cause apoplexy. He observed thrombus deposited on atherosclerotic plaques in the carotid sinus in seven cases in a series of 400 consecutive autopsies. The carotid artery was completely blocked in only one case. Hunt[14] examined the carotid pulsations in 20 cases of hemiplegia and found it absent on the appropriate side in four instances. In addition the monograph by Hultquist[15] in 1942 described the results of the pathological study of the entire carotid system in a series of 1300 autopsies.

It is the purpose of this presentation, based upon the recorded cases in the literature and our own experience, to outline the clinical features of unilateral spontaneous thrombosis of the carotid arteries. Our series of 17 cases of spontaneous thrombosis of the carotid arteries will be reported in detail and comment made comparing the features demonstrated by these cases with those recorded in the literature.

SIGNS AND SYMPTOMS

1. Motor Symptoms. Some degree of muscular weakness is the most common symptom encountered. This may occur first in the hand and arm and later spread to involve the leg; or the upper and lower extremities may be involved simultaneously. Associated with the hemiplegia, there may be impairment of consciousness, drowsiness, urinary incontinence or nocturnal restlessness.

Comment: All the patients in the present series had muscular weakness. In nine of the patients, the initial symptom was muscular weakness and in the remaining eight, paresis developed at some time in the illness.

2. Headache. This is a frequent symptom encountered. There is nothing specific about the type of headache in this clinical condition. The headache may be localized to the side of the thrombosis but is frequently generalized. In many instances, it has the characteristic of a migraine headache.

Comment: In only one of our series was headache noted. (case III)

3. Sensory Disturbances. The clinical picture may be ushered in by paresthesias involving the affected side, most frequently in the upper or lower extremities. In association with the hemiplegia, there may be objective evidence of sensory disturbance of the cortical type and in more severe cases involvement of all sensations.

Comment: In one of the present series (case XIII) sensory disturbance was the initial symptom. Six others developed sensory disturbances at some time in their illness.

4. Aphasia. Both receptive and expressive aphasias frequently are encount-

ered. These symptoms may antedate all other symptoms or be present in association with a hemiplegia and hemianesthesia. Rarely aphasia may complicate in right handed patients on occlusion of the right internal carotid artery.

Comment: Nine patients in this series demonstrated aphasia in association with the motor involvement. In no instance, was aphasia present as an only symptom. Of particular interest is case I in which aphasia persisted in a right handed individual in whom the right internal carotid artery was occluded. Andrell[3] (case V) reported the persistence of an aphasia in a right handed individual in which occlusion of the right internal carotid artery was demonstrated.

5. Psychiatric Distrubances. The patient may present as a psychiatric problem. In some instances, the picture of a psychoneurosis characterized by neurasthenia and anxiety may be present. In others, the picture is one of a depression associated with marked deterioration. Some patients are euphoric.

Comment: In case V, the symptomatology was so vague that initially a diagnosis of a functional disorder was made. The initial symptom noted in case XII was that of memory loss. In the course of his illness, case XIV developed a change in personality. Slow cerebration and memory difficulty were noted in case XV.

6. Convulsions. Convulsions of either a focal or a diffuse type may herald the onset of this condition. Frequently, after the onset of hemiparesis, focal or generalized convulsions may become a part of the clinical picture.

Comment: Convulsions were not encountered in any of the patients in the present series.

7. Ophthalmological Features. The patients may complain of episodes of transient blindness early in the course of their illness. At the time of the "stroke", a hemonomous hemianopsia may be demonstrated. A primary optic atrophy with visual loss on the side of the involved arteries has been present in a small number of the reported cases. More unusual visual symptoms such as diplopia, ptosis, pupillary changes with the ipsilateral pupil being fixed, smaller or larger, the presence of papilledema, and paralysis of vertical ocular movements have been reported.

Comment: In four patients (cases II, III, XIV, XVII), visual disturbances ushered in the clinical picture. In two additional patients, visual complaints developed during the course of the illness. Case XVI and case XVII deserve special mention in that a primary optic strophy was visible on the side of the occluded artery. In two instances (cases II, III), the onset of visual complaints was followed within twelve hours by muscular weakness. In case XVII, the onset of visual complaints was coincident with the development of a hemiparesis.

8. Palpation of the Carotid and Temporal Arteries. This sign has received scant attention in the literature. Its importance has been pointed out previously by Hunt[14], Webster, et al[9], Fisher[10] and Johnson and Walker[11]. It should be part of the routine examination. Specific mention of this procedure is omitted in most of the case reports and further attention to this detail will be necessary before the value of this procedure can be established.

Comment: In five instances there was a diminution of absence of carotid pulsations on palpation. In one instance, in association with the absent carotid pulsation there was a decreased pulsation of the temporal and facial arteries on the same side.

9. Head Noises. Various noises in the head, such as throbbing in the mastoid area, sensation of a watch ticking, a soft roaring in the head, whistle sound in the ear, and a sensation of excaping steam in the ear are complained of occasionally by patients.

Comment: In one patient, case XI, whistling in the ear was the first symptom noted. Another, case XV, complained

of a sensation of escaping steam in the left ear.

10. Miscellaneous. Dizziness is frequently complained of in the reported cases but not described in sufficient detail to enable one to ascertain what the patient meant by the term. Andrell[3] had a patient with attacks of vertigo so severe she fell down. One of the reported cases suffered from fits of sneezing.

Comment: Only one of our patients (case IV) demonstrated dizziness and this symptom was the initial complaint of the patient.

CLINICAL COURSE

Any of the signs and symptoms outlined above may herald the onset of this illness and at a later date, the clinical picture may be complicated by the occurrence of others of the signs and symptoms. The course of this illness follows one of three different patterns.

1. Transient Attacks. This is frequently encountered and there are transient attacks characterized by a sudden onset of symptoms which last from a few minutes to an hour and then clearing either suddenly or gradually. Some residuals of the signs and symptoms may persist until the next attack which may occur days, weeks or months later. Not infrequently such a series of transient attacks are terminated by a "stroke like" picture with hemiplegia, aphasia and sensory disturbances. The term "stuttering" has been used to describe this type of clinical course.

Comment: In nine of the patients in our series, the clinical course was characterized by transient attacks. In two instances (case II and case III) the initial symptom was followed within 24 hours by the development of a hemiplegia and in these two instances, they might be classified under an apoplectic type of course.

2. Sudden apoplectic onset. In this group of patients without prior warning, there is a sudden onset of a hemiplegia which may be associated with severe headache, disturbance of consciousness, urinary incontinence, aphasia and sensory disturbances.

Comment: Five of the patients in this series had this type of onset.

3. Slowly progressive course. This picture closely resembles that encountered in brain tumors in which the clinical course is one of slow gradual progression of symptomatology.

Comment: Two of the patients in this series demonstrated this clinical course. In both, the diagnosis of brain tumor was seriously entertained and ventriculography was performed prior to the establishment of the correct diagnosis.

ANGIOGRAPHIC FINDINGS

Angiography in occlusions of the internal carotid artery may reveal:

1. A conical narrowing of the dye before it stops resulting in a stump or short segment of the internal carotid artery being visualized.

2. Retrograde flow of the dye in the common carotid artery and the flow of dye into the vertebral artery resulting in a vertebral angiogram.

3. Irregularities in the diameter of the vessel and narrowing of the vessel.

4. Failure on repeated attempts to produce any filling of the internal carotid artery.

5. Failure to fill the internal carotid artery beyond the carotid siphon.

The first three findings outlined above are strong evidence for occlusion of the carotid artery. Where the vessels are narrowed and irregular, a canalization of a thrombus or a partial thrombosis is suggested. Failure to fill the internal carotid artery at its origin after repeated attempts at angiography is suspicious but not diagnostic of in-

ternal carotid artery occlusion as it may well be due to technical difficulties. Failure of the carotid artery to fill beyond the carotid siphon likewise must be interpreted with reservation and such a factor as spasm of the artery must be considered.

Comment: Eleven patients in this series received angiographic studies and all examples of the five types of findings outlined above were represented in this group of patients. Case XI deserves special comment in that following an angiogram performed on the uninvolved side and a pneumoencephalogram performed simultaneously under anesthesia, the patient upon recovery had developed an aphasia. Johnson and Walker[11] have pointed out the danger in performing carotid angiography on the side opposite an occluded vessel and our experience with this one case tends to confirm their observation. A production of a vertebral angiogram, as in case VIII, in the course of attempting to produce filling of the internal carotid artery is strong suggestion of occlusion of the internal carotid artery. King[16] reported a similar finding in which reflux filling of the vertebral artery took place in a case of occlusion of the internal carotid artery.

ETIOLOGY

In the literature the commonest etiology reported is arteriosclerosis. In this regard atherosclerosis has been described in involving the carotid sinus in the teens and early twenties by Chiari[13] and Keele[17]. Thromboangiitis obliterans has been incriminated as the second most common cause of this condition. Fisher[10] feels that the evidence for thromboangiitis obliterans as recorded in the literature is inconclusive and states "further proof is necessary before thromboangiitis obliterans of the carotid and cerebral arteries can be accepted". Other etiologies recorded in the literature include retrograde thrombosis from an intracranial aneurysm and syphilis. Ameli and Ashby mentioned increased viscosity of the blood and polycythemia, temporal arteritis and polyarteritis nodosa but document no cases of these etiologies. Acute infections have been mentioned and King and Langworthy[18] described a case of a seven year old boy in whom thrombosis of the right internal carotid artery developed three weeks after pneumonia.

Comment: In fourteen of the cases in this series, no etiology was discovered for the thrombosis. Five of these patients had sections of the occluded arteries studied pathologically and no conclusion was made as to the etiology. This group of cases would probably be classified in the literature as due to arteriosclerosis. In case IX during convalescence from pneumonia, a thrombosis of the carotid artery developed. Noran, Baker and Larson[19] present evidence to indicate that in pneumonia there may be intravascular clotting and such a mechanism could explain the symptomatology in our patient as well as in the patient reported by King and Langworthy[18]. In case XII, the etiology was polycythemia vera and this cause has not been documented in the publications regarding spontaneous occlusions of the internal carotid artery. The current publications have very little information regarding studies for the demonstration of blood dyscrasias which might produce an increased tendency to clotting. Further information in this regard is desirable. In case XVII, periarteritis was found in other organs of the body and although not found in the artery studied may have still been an etiological factor in the production of the occlusion of the vessel. In three of the patients in this series hypertension was present. Another patient had a history of hypertension but during our period of observation, the pressure was within normal range.

INCIDENCE

There is a definite preponderance of males affected by this condition. There is a predilection for the 30 to 60 age group although cases from age 13 to age 71 have been reported. There is a definite tendency for the thrombosis to in-

volve the left side more frequently than the right.

Comment: In this series, 14 of the patients were males and three of the patients, females. Two patients were age 35 and 36 respectively. Five patients were between 40 and 50 years of age. Six patients were between 50 and 60 years of age and four patients were between 60 and 70 years of age. In 12 of the patients the thrombosis occurred on the left side and in the remaining five, the thrombosis occurred on the right side. In 14 of the patients, the internal carotid artery alone was involved. In case XII, the site of thrombosis was the common carotid artery. In case XIII in addition to the involvement of the internal carotid artery, there was thrombosis of the common carotid and external carotid arteries. In case XVII, obliteration of the ophthalmic artery in addition to thrombosis of the internal carotid artery was present on the involved side.

TREATMENT

In many instances, the patients received no specific treatment. Three forms of therapy have been tried: 1. Arteriectomy, 2. Cervical sympathectomy, and 3. Anti-coagulant therapy. This last type of therapy has been used in only a small number of reported cases. The rationale for excision of a segment of the thrombosed vessel is the belief, by those who used this procedure, that the presence of the thrombosed vessel might result in a reflex spasm of the smaller cerebral vessels and thus account for the transient nature of the symptoms. Against the use of this type of therapy have been the reports in which there is microscopic evidence of recanalization of an old thrombus.

Comment: In six of this series (cases VIII, IX, X, XIII, XV and XVI), a segment of the thrombosed vessel was removed without apparent effect on the symptomatology. In case XI, dicoumaral therapy was used for short periods without any apparent improvement and in this same case, repeated stellate blocks following the acute development of aphasia failed to produce any relief of this symptom. In case VIII, while receiving dicoumaral, the patient made a marked improvement. The use of neurological rehabilitation is indicated as non-specific therapy.

PROGNOSIS

The prognosis for improvement following the establishment of the diagnosis is not particularly hopeful. Twenty-five percent of the cases collected by Johnson and Walker[11] showed some improvement with or without treatment. There seems to be no correlation between the degree of involvement and the type of onset. Thirteen of the patients reviewed by Johnson and Walker succumbed within several months.

Comment: Two of the patients (case XII and case XVII) of the present series have died.

VENTRIBULOGRAPHY AND PNEUMOENCEPHALOGRAPHY

The characteristic picture following air studies is that of dilatation of the ventricles particularly on the involved side. A brain tumor may be present in addition to thrombosis of the carotid arteries and some suggest air studies in addition to angiography.

Comment: In cases V and XV, ventriculography revealed no abnormalities. In case XI a pneumoencephalogram was normal.

CASE REPORTS

CASE I. , a 46 year old white male was in good health until 8-23-52. At that time while fishing, he was approached by a game warden and suddenly became unconscious. He remained totally unresponsive for the next week. On 8-30-52 examination revealed the following findings: The patient was unresponsive except to painful stimuli, which produced movement of the right side of the body but not of the left arm or leg. There

was a left lower facial paralysis present. The deep reflexes on the left could not be elicited and there was a questionable Babinski on the left. The patient was unresponsive to painful stimulation on the left side of the body. On fundoscopic examination the vessels in the right fundus appeared very narrow with what appeared to be decreased blood in them. The discs were normal. On palpation of the carotid arteries in the neck, that on the right felt firm with a minimal pulsation. On 9-11-52, a percutaneous angiogram revealed a complete occlusion of the right internal carotid artery at its origin.

The patient showed gradual improvement. Over a period of several weeks, he became more alert and carried out commands. He was unable to talk and he could only eat with difficulty. The patient had always been right handed. The paralysis of the left arm and leg remained complete and the deep tendon reflexes on the left became more active than those on the right. The Babinski on the left was positive. There was no appreciation of sensation demonstrable in the paralyzed extremities.

Comment: This patient demonstrated the sudden apoplectic onset of his clinical symptomatology. The diagnosis of internal carotid artery occlusion was suspected by the palpation of the carotid arteries which revealed on the right a firm artery with minimal pulsation. It was proved by the angiogram which revealed complete occlusion of the right internal carotid artery at its origin. Of interest, is the persistence of speech disturbance in this right handed man.

CASE II. , a 41 year old white male, was admitted on 8-20-52 for investigation of his complaints of blurring of vision and previous history of right sided weakness. The patient had been in perfect health until June 25, 1952, when in the morning, he developed a transient blurring of vision. That evening, he experienced difficulty manipulating change in his pocket, dropped some and when bending over to pick the change up, he fell. He was unable to move his right upper and lower extremity and became unable to speak intelligibly. There was no loss of consciousness. The following morning he was able to move his extremities and speak more clearly and rapidly improved over the next week. In another week, he stated that the strength in his right extremities returned to normal. He returned to work, but on 8-6-52 had another episode of blurring of vision. The next morning he experienced a fainting sensation. Examination revealed the following positive findings: The general physical examination was negative. The systolic blood pressure was 134 and the diastolic 86. The deep tendon reflexes in the right upper extremity were greater than those in the left. The Oppenheim and Gordon signs on the right were questionable. The left ankle jerk was more active than that on the right. No evidence of paresis could be demonstrated.

A left carotid angiogram done under local anesthesia showed occlusion of the left internal carotid artery 1 cm. from its origin.

Comment: This patient had one transient attack of visual disturbance followed within a matter of hours by an apoplectic onset of weakness. He made a rapid recovery from this initial episode but subsequently has had one more bout of visual disturbance. The diagnosis of internal carotid artery occlusion was proved by angiogram.

CASE III. U.H. - , a 35 year old white male was admitted to hospital on 8-25-52. Thirteen days prior to his admission, he developed transient blindness in the right eye of five minutes duration. At 5:30 a.m. on the next day he awakened to find a right sided paralysis and complained of a severe headache. Examination at that time revealed a right hemiplegia, aphasia and a right homonomous hemianopsia. The headache lasted about four days and has not reappeared since. The right hemiparesis and aphasia gradually improved.

At the time of his admission to hospital, the examination revealed the following positive findings: There was a

mild right hemiparesis and a mild right lower facial. The deep tendon reflexes were hyperactive on the right side. An expressive aphasia was present. The previously present right hemianopsia had disappeared and there was only a mild field defect on the right side.

Routine blood and urine studies were normal. The serology was negative. Radioactive isotope encephalometry was normal. The blood pressure was 115/70. On 8-26-52 a left carotid angiogram was reported as follows: "Several attempts were made at a left carotid angiogram. The films show very incompletely filled somewhat ragged appearing internal carotid artery, especially in the region of the carotid siphon. The appearance was compatible with a partial thrombosis of the carotid artery. There was a faint filling of some of the branches of the posterior cerebral and on the latter films, the internal cerebral vein and the basal vein and the vein of Galen are filled with some contrast media as is also the straight sinus. There is no visualization on any of the films of the anterior and middle cerebral arteries."

Comment: The clinical picture and the findings on the angiograms suggest the diagnosis of probable thrombosis of the internal carotid artery. Interpretation of failure of the carotid artery to fill beyond the carotid siphon must be made with caution. The radiological picture presented suggested a partial thrombosis of the carotid artery as some of the contrast media passed into the posterior cerebral artery and subsequently into the veins. Radioactive isotope encephalometry revealed no abnormality.

CASE IV. ., a white female about 50 years old, was admitted to the hospital on 5-11-50. On the day of admission she had developed an acute onset of dizziness. The routine blood and urine studies were negative. The serology was negative. Examination revealed the following positive findings: The systolic blood pressure was 165 and the diastolic was 95. The neurological examination was negative. On palpation of the carotid arteries, it was found that there was no pulsation in the left internal carotid artery which was cord like and could be rolled beneath the examiner's fingers.

The patient's dizziness cleared in six months and she was asymptomatic until April of 1950 when weakness developed in the right upper extremity. Coincident with this, she complained of headaches and lack of pep. In June of 1950 there was a recurrence of the dizziness and this had been present for seven days at which time she was re-examined. At this time the blood pressure was unchanged. A mild right hemiparesis was present, most marked in the right upper extremity and accompanied by a mild right lower facial paralysis. A mild right sided hyper-reflexia was demonstrable. The occluded artery previously palpated had not changed.

Comment: The diagnosis of occlusion of the left internal carotid artery is strongly suggested by the palpation of this vessel which revealed absence of any pulsation and a cord like character of the vessel. This case is best classified under those in which transient attacks are characteristic.

CASE V. U.H. - , a 51 year old white male, was admitted to hospital on 7-16-52. The patient had first been seen six days prior to his admission to hospital. There was a three month history of unusual involvement of the legs and arms. This condition consisted of some incoordination and a sensation as if the affected extremity were asleep. The left lower extremity was initially involved and subsequently the right lower extremity as well as both upper extremities became involved. These peculiar attacks lasted from a few minutes to one-half an hour. During the last episode which involved the legs, the patient stated he was so unsteady he was unable to walk. The neurological examination was completely negative and a diagnosis of a functional disorder was established.

Two days later, the patient became drowsy, confused and experienced diffi-

culty with speech and gait. At the time of admission he was unable to give any history because of a lack of response. At this time the neurological findings were minimal although there appeared to be some weakness of the right side with slight right hyporeflexia. At times, the patient appeared to be definitely aphasic but this was difficult to assess because of the general mental status. A spinal tap revealed a pressure of 280 mm. of water. The possibility of a brain tumor was seriously considered and a ventriculogram was performed. They were entirely negative.

Two attempts to perform a carotid angiogram failed to produce filling of the internal carotid system on the left. On August 16, 1952, an open angiogram was performed. The internal carotid artery on the left did not fill and there was a retrograde flow of dye in the common carotid artery. Routine blood and urine studies were negative. Radioactive isotope encephalometry was negative. The serology was negative. A biopsy of the cortex at the time of the ventriculogram was negative.

Comment: The bizarre nature of the presenting complaints with involvement of all extremities and the absence of any neurological findings initially suggested the diagnosis of a functional condition. The subsequent development of organic symptomatology and findings plus the presence of increased spinal fluid pressure made the entertainment of a diagnosis of a brain tumor a serious consideration. The demonstration of a complete occlusion of the left internal carotid artery by repeated angiography finally established the diagnosis. This case is probably best classified amongst those which have a slowly progressive course simulating brain tumors. The radioactive isotope encephalometry studies were negative. A biopsy of the cortex revealed no abnormalities.

CASE VI. U.H. - , a 60 year old white male, was admitted to the hospital on 2-12-52. About two years prior to hospital admission, this patient had a sudden onset of weakness of the right arm and leg without loss of consciousness. This weakness cleared in two days. One year prior to admission to the hospital, he again developed weakness of the right leg which cleared in several days. On 2-5-52 a loss of speech suddenly developed. He could understand everything that was spoken to him and could read but was unable to write. This began to clear on the fifth day and continued to improve up to the time of the patient's admission to hospital.

The general physical examination revealed a systolic blood pressure of 164 and a diastolic of 100. On palpation of the carotid arteries, the right carotid pulsated well. On the left, the internal carotid artery pulsated less strongly and felt smaller in caliber than the corresponding member on the right. The examiner estimated that it was about half the size of the artery on the right. The patient complained of some subjective difficulty with speech but this could not be demonstrated objectively. The patient fell to the right on tandem walking. The remainder of the neurological examination was negative.

Routine blood and urine studies were negative. The serology was negative. The bleeding time and clotting time were normal. The chest and skull were normal.

Comment: The clinical picture and the decrease in pulsation of the left internal carotid artery suggest the diagnosis of partial occlusion of the left internal carotid artery. This case falls into the group of cases characterized by transient attacks.

CASE VII. U.H. - , a 60 year old female Indian, was admitted to the hospital on 2-22-52. This patient had a history of hypertension for four to five years. In 1948, three left sided strokes occurred without loss of consciousness. Following the first two strokes, there were no residuals and recovery was complete. Following the third episode, there had been residual paresis and spasticity on the left. In January of 1952, there was sudden onset of slurring of speech and further weakness of the left arm and leg. Since that time she has had marked weakness of the left arm

and to a lesser extent the left leg.

On admission the blood pressure was 134/88. Palpation of the carotids in the neck were normal. The remainder of the general physical examination was negative. The positive findings on the neurological examination were as follows: There was a left hemiparesis with spasticity. There was a subjective hemihypesthesia on the left and the position sense was mildly reduced in the left upper and lower extremities. Hyper-reflexia, positive toe signs and a sustained ankle clonus were present on the left.

Visual fields were within normal limits. Routine urine and blood studies were normal. The Wassermann was negative. The bleeding, clotting and prothrombin times were normal. The electroencephalogram showed an abnormal electroencephalogram with a diffuse dysrhythmia but no focalization. The radioactive isotope encephalometry showed a focus in the right posterior fossa.

On 2-28-52 a right carotid angiogram was performed and only the external carotid was visualized. On 3-4-52 the angiogram was repeated and the conclusion was the common carotid and the external carotid on the right are filled and most likely normal. The internal carotid ended bluntly 1 cm. from the bifurcation and a diagnosis of occlusion of the internal carotid artery was made.

Comment: This patient's clinical course was characterized by transient attacks of paralysis, finally resulting in some permanent paralysis. The angiogram was diagnostic and established the diagnosis of occlusion of the internal carotid artery. A radioactive uptake study showed a focus in the right posterior fossa.

CASE VIII. U.H. - , a 51 year old farmer, was admitted to the hospital on 7-29-50. The patient was in good health until November of 1949 when he developed transient bouts of flaccid paralysis of the left leg. He began to note similar attacks involving the left arm about January 1950. The leg and arm attacks might occur separately or together and varied in their duration from ten minutes to two hours. These attacks usually occurred every several days but on one occasion, a two month period elapsed without any symptoms being present. During the attacks in which the arm was involved, the patient was unable to move the arm unless he yawned. When in the process of yawning, he was able to raise the left arm. Three weeks prior to admission to hospital, some numbness and tingling of the left lower face and some difficulty with speech was noted. At the time of admission the blood pressure was 160/90. There was a left sided hemiparesis. Astereognosis, decrease in vibration sense, and a slight decrease in position sense were demonstrated in the left upper extremity. There was a mild left hyperreflexia. There was an unsustained left ankle clonus. X-rays of the skull were negative. The routine blood and urine studies were negative. The blood Wassermann was negative.

On 8-2-50 bilateral angiograms were performed. On the left, there was good filling of the internal carotid artery, the anterior and middle cerebral arteries and their branches. When the left angiogram was performed, there was also some filling of the right anterior cerebral artery. On the right, there was no filling of the right internal carotid artery. There was considerable filling of the right external carotid artery and its main branches. Excellent filling of the vertebral artery on the right was obtained. On 8-3-50, the angiogram on the right side was repeated and exactly the same results found as at the time of the previous right carotid angiogram. On 8-7-50, the right internal carotid artery was explored. The common carotid artery was large and there was ecchymosis about it and it was very adherent to the surrounding tissues. This condition of the artery extended up to the bifurcation and through the bulb and it was the opinion of the surgeon that this reaction was due to the previous angiograms. There was good pulsation in the common carotid and external carotid artery. The internal carotid

artery was exposed for a distance of about two cm. above the bifurcation and it did not pulsate. Aspiration of the internal carotid artery failed to reveal the presence of any liquid blood. A one cm. segment of the internal carotid artery was excised and examined pathologically.

The patient was started on dicoumaral therapy and remained on it until 5-7-51. At this time the patient's condition was reassessed. Following discharge from the hospital, there had been gradual improvement in motor weakness so that at the time of readmission, the chief weakness remained only in the finger movements of the left hand. Curiously enough in spite of dicoumaral therapy and maintenance of the prothrombin time around 50 seconds, the patient developed a coronary thrombosis which had necessitated hospitalization for two weeks.

Examination following readmission revealed a blood pressure of 190/110. The patient was alert and cooperative. There was some edema of the left hand with paresis of the muscles of the left hand and of the left wrist. The rest of the musculature was intact. The deep reflexes were all hyperactive and more so in the left upper extremity. Slight involvement of deep sensation remained in the left upper extremity but this also showed marked improvement. The laboratory studies were all within normal limits. Because of the patient's remarkable improvement, it was felt that no further dicoumaral therapy was indicated and it was discontinued.

Comment: This patient's clinical course was characterized by transient attacks of weakness involving the left arm and leg. There was an associated disturbance of deep sensation in the upper extremity. The diagnosis of thrombosis of the internal carotid artery was proved at surgery. Following the institution of dicoumaral therapy, the patient made a remarkable recovery. While on dicoumaral therapy, a coronary thrombosis developed.

CASE IX. U.H. - , this forty-eight year old white female was hospitalized on 2-1-52 for investigation of left sided weakness. In July 1951 this patient developed bilateral pneumonia for which she was hospitalized. During convalescence, on approximately the 10th day of illness, a sudden left hemiparesis developed associated with mental confusion. The patient had a gap in memory for approximately the next two weeks but there was no definite history of coma. The pain in the joints of the left extremities developed soon after the onset of the paresis. The paralysis and left sided pain had been present since their onset.

Examination revealed an obese dishevelled female. The patient was alert but uncooperative and evidenced excessive suspicion. The carotid arteries were palpable bilaterally. The abnormal neurological findings were limited to the left side and included hyperactive deep reflexes, absent abdominal reflexes, unsustained knee clonus, sustained ankle clonus, positive toe signs, rigid extremities with spasticity and possible ankylosis, hemihypesthesia, decreased stereognosis and decreased position sense. The blood pressure was 130/80.

Routine blood and urine studies were negative. The blood Wassermann was negative. X-rays of the skull and chest were negative. Radioactive isotope encephalometry showed a diffuse increase on the right with varying percentage differences but no focus.

On 2-6-52, a right carotid angiogram failed to visualize the internal carotid artery on this side. On 3-3-52, the right common carotid artery was explored. The bifurcation was found and no pulsation could be seen or felt in the internal carotid artery. The external carotid appeared to be normal and there was a good pulse. The internal carotid was ligated doubly about 2 cm. above the bifurcation and a section of the artery was taken and examined pathologically.

Comment: This patient had an apoplectic type of onset of symptoms and has as residuals a left sided hemiparesis and left sided sensory findings. This case

is of particular interest since the onset of the symptoms took place during convalescence from pneumonia. The diagnosis was established by the absence of filling of the internal carotid artery on angiogram and the subsequent surgical exposure and excision of a segment of the thrombosed artery. Radioactive isotope encephalometry showed a diffuse increase on the involved side with varying percentage differences but no focus.

CASE X. U.H. - , this fifty-two year old white male was admitted to the hospital on 2-4-52. On about 3 p.m. on the day prior to admission, this patient fell and struck the left side of his head. Following this injury, blood was noted on the head but the source was not determined.

At the time of admission, the patient was semi-comatose and it was noted that he was unable to move the right side of his body. The respirations were irregular. The pulse and blood pressure were within normal limits. Examination of the fundi was negative. There was a right hyperreflexia and positive toe signs on the right. As the patient's state of consciousness improved, a right hemihypesthesia and a right homonomous hemianopsia were suspected. A spinal fluid examination was done with negative results. Because of the disturbance of consciousness and the focal neurological findings, a dural hematoma was suspected and trephines were performed with negative results. The state of consciousness improved steadily and by 2-8-52, the patient was alert and followed simple directions but had some difficulty understanding and could not speak coherently. Three attempts at angiography were made on the left. On the first two attempts, the external carotid artery alone showed filling. On the third attempt, in addition to the filling of the external carotid and its branches, the internal carotid artery filled only in its initial few cms. An exploration of the internal carotid artery in the neck was performed and a thrombosed vessel was found. A section of this vessel was removed for pathological examination. The patient continued to gradually improve and was ambulatory at the time of his discharge on 3-18-52. A mixed aphasia persisted. The routine blood and urine studies were negative. The hematocrit on three occasions was 50.5, 51 and 47. The hemoglobin was reported as 17.5, 18.4 and 16.6. The routine serology was negative.

Comment: This patient demonstrated an apoplectic type of onset of symptomatology. Trephines were performed with negative results. The diagnosis of occlusion of the internal carotid artery was established by angiography and subsequent operative intervention.

CASE XI. U.H. - , this 48 year old white male was admitted to the hospital on 1-23-50. The onset of the patient's complaints dated eight weeks prior to admission to the hospital. At that time he began to hear a whistle in the ear synchronous with his heart beat. Two weeks later, intermittent numbness in the right arm and leg were noted, and at about the same time he noted difficulty in using the right arm and leg. On 1-10-52, he noted stiffness in the neck and at the same time the numbness in the right side became constant. The positive findings on examination were incoordination of the right arm and to a lesser extent, the right leg. There was a mild hyperreflexia involving the deep reflexes on the right.

Repeated attempts at closed angiography on the left failed to produce visualization of the internal carotid artery. Finally an open angiogram was done and the needle was directed downward into the common carotid artery below the bifurcation and again no filling of the internal carotid artery was produced. The diagnosis of thrombosis of the internal carotid artery on the left was made. Dicoumaral was administered until 2-11-50 without progression of symptoms and the buzzing in the ears disappeared. Before discharge, it was decided to do a right sided angiogram and a pneumoencephalogram to rule out the possibility of a tumor. Prior to these procedures, the dicoumaral was discontinued. Both procedures were performed at the same time under general anesthesia. When the patient awoke from the anesthesia, an

aphasia had developed. He was again put on dicoumaral and showed slight improvement in that he became able to say a few words. The aphasia was mostly motor and there was apraxia causing agraphia. Repeated stellate blocks were performed after the patient developed aphasia but this did not materially influence his condition. The routine skull rays were negative. The routine blood and urine studies were negative. The Wassermann was negative. The clotting time was within normal limits. The protein in the spinal fluid was normal. The right cerebral angiogram and pneumoencephalogram performed on 2-11-50 were normal.

A follow-up report received from a friend of the patient in May of 1952, revealed that there had been a progression in this patient's aphasia and that he had become unable to speak. His handwriting had become unreadable.

Comment: The symptomatology of this man's condition was ushered in by auditory manifestations. The course of illness has been a slowly progressive course and a brain tumor was considered a definite possibility so that an angiogram on the normal side and a pneumoencephalogram were performed. Following these procedures which were done simultaneously under anesthesia, the patient developed an aphasia. The patient received dicoumaral therapy with slight initial improvement. The diagnosis of occlusion of the left internal carotid artery was established by repeated angiography.

CASE XII. This thirty-six year old white male developed a moderately severe headache on 8-10-48. This continued for several days. On August 15th, he noted nausea and vomiting and became lethargic. He was hospitalized the following day when he became unable to talk or use the right arm. Past history revealed that he had had mild but definite memory loss for several months and on several occasions, had complained of numbness in his hands. Two years previously he had experienced an episode of ankle edema and was told he had "kidney disease". Examination revealed an expressive and receptive aphasia, weakness of the right arm and lower face, and hyperactive reflexes on the right. Physical examination showed enlargement of the spleen. The hemoglobin was 18 grams; red blood count, 6,360,000 per cubic mm.; hematocrit, 56.5%. The clinical diagnosis of polycythemia vera was made and treatment in the form of venesection was instituted.

A few days after admission, the patient became more alert and began to understand simple statements. He gradually improved and was beginning to talk when eight days after admission the aphasia became complete and weakness of the right leg developed. At this time no pulsation of the left carotid artery could be detected. The patient again began to improve but two weeks later developed a pulmonary infarct. Three months later death occurred following a thrombosis of the superior mesenteric artery.

Comment: This patient demonstrated a slowly progressive course. This case is of particular interest since the cause of the thrombosis of the common carotid artery was polycythemia vera. This condition has not been implicated in recent publications concerning the etiologies of spontaneous occlusion of the carotid arteries. Post-mortem examination showed complete occlusion of the left common carotid artery.

CASE XIII. This sixty-five year old white male was admitted to the hospital on 8-13-51. He complained of periodic episodes of numbness in the right arm and leg for four months duration. These episodes would come on when raising the head as when drinking water. Two months prior to admission he had an episode of expressive aphasia and subsequent to this had several similar episodes. One month prior to admission he sustained a weakness of the right leg which lasted about one hour. Subsequently, two similar episodes of weakness occurred. The general physical examination revealed an absence of carotid pulsations on the left. Pressure over the left carotid sinus caused this patient to complain of the numbness which he had

experienced in the attacks. The neurological examination failed to reveal any abnormalities. The systolic blood pressure was 120 and the diastolic, 80.

X-rays of the skull and chest were negative. A barium enema was negative. An esophagogram was negative. Cervical x-rays showed a blocked vertebra with congenital fusion of the 6th and 7th cervical vertebrae. The electrocardiogram was normal. The routine blood count was normal. Urinalysis showed 30 to 35 white blood cells. On 8-21-51, the left common carotid artery was explored. The left common carotid was found to be completely occluded by a thrombus which extended to involve the internal and external carotid arteries. The common carotid artery was opened and there was no bleeding. A segment of the common carotid in continuity with the proximal parts of the external and internal carotid arteries was excised.

Comment: This patient's condition was characterized by transient attacks of numbness, weakness, and aphasia. Of particular interest was the finding that palpation of the carotid sinus on the involved side reproduced his sensory complaints. Clinically there was an absence of carotid pulsations on the left and the diagnosis of thrombosis of the common, internal and external carotid arteries on the left was proved at surgery.

CASE XIV. U.H. - , this fifty-one year old white male was admitted to the hospital on 9-11-52. In the summer of 1952, the patient began to have attacks of visual disturbance which were described as attacks of "blackness". At first these attacks would last thirty minutes and come on about once a day. Coincident with the onset of the visual complaints, the patient developed a change in personality, manifested by forgetfulness and inability to initiate activity or direct his business. The visual symptoms increased in severity and duration. Upon awakening on 9-1-52, the patient was found to have developed an aphasia, right hemiplegia and right homonomous hemianopsia. He spent the next five weeks in bed and there was a gradual improvement in the motor weakness and the aphasia. The positive findings on admission were a mixed aphasia and on the right side, a homonomous hemianopsia, hemiparesis, hyperreflexia and positive toe signs. The carotid arteries were palpable in the neck. The systolic blood pressure was 135 and the diastolic 85. Radioactive uptake studies revealed an abnormal record with a high uptake in the posterior areas, more so on the left. Routine blood and urine studies were negative. The Wassermann was negative.

On two occasions, a left carotid angiogram was performed. In both instances, the external carotid branches were well visualized. The internal carotid artery was definitely narrowed and was not visualized past the region of the sella turcica.

Comment: This man's clinical picture was characterized at its onset by personality changes and transient attacks of visual disturbance. He subsequently developed a hemiplegia with an associated aphasia and homonomous hemianopsia. The angiogram suggested an occlusion of the internal carotid artery on the left. The internal carotid artery was definitely narrowed and was not visualized past the region of the sella turcica. The diagnosis in this case is probable thrombosis of the internal carotid artery. Radioactive encephalometry revealed abnormalities.

CASE XV. U.H. - ., this fifty-six year old white male was first seen in January 1951. It was difficult to obtain an accurate history from the patient. As far as could be ascertained, he injured his right knee in 1940 and for awhile had difficulty walking. Since 1947, the patient had complained of clumsiness in the right lower extremity and this had progressed to involve the left lower extremity. During the four year period prior to examination, he had also complained of various pains throughout the body, particularly in the chest, and vague sensory disturbances involving the left lower extremity and the right abdominal wall. During this same period,

auditory complaints consisted of a constant rushing escaping steam sound in the left ear. In December of 1950 the patient developed difficulty writing and talking. He found it a little hard to use the right words.

At the time of the initial examination, the neurological findings were normal except for slow cerebration, a mild right hyperreflexia and a positive Babinski on the right. The patient was admitted to the hospital on 2-28-52. At this time the difficulty with memory and mentation generally had clearly progressed. The neurological findings remained unchanged. Routine blood and urine studies were negative. The systolic blood pressure was 140 and the diastolic 80. The serology was negative. A ventriculogram was normal.

Two angiograms on the left side failed to fill the internal carotid artery. On 3-10-51, an open angiogram was done and a thrombosis of the internal carotid artery was discovered. A segment of the internal carotid artery was excised just above the bifurcation and examined pathologically.

Comment: This patient falls into the group characterized by progressive symptomatology. During the early stages of illness, there were auditory complaints. In addition vague complaints of clumsiness involving the lower extremities, various pains throughout the body and vague sensory disturbances were also present. After repeated angiography failed to produce filling of the internal carotid artery, the neck was explored and diagnosis of thrombosis of the internal carotid artery was discovered and a segment of this artery excised. A ventriculogram done prior to angiography was normal.

CASE XVI. U.H. - _ _., this forty-three year old white male was admitted to the hospital on 1-22-51. Four months prior to hospital admission, the patient had a sudden onset of aphasia and a right hemiplegia.

At the time of admission to the hospital, the systolic blood pressure was 90 and the diastolic 60. There was no pulsation palpable in the common or internal carotid arteries on the left. There was absence of pulsation of the left temporal artery while the corresponding artery on the right demonstrated excellent pulsation. The facial artery on the left pulsated weakly in comparison with that on the right. The neurological examination revealed a primary optic atrophy on the left with very tenuous arterioles in the left retina, right hemiparesis, hyperreflexia and positive toe signs, and a mixed aphasia. The patient was totally blind in the left eye.

Routine blood and urine studies were negative. Electrocardiograms, esophagograms and skull x-rays were normal. The right angiogram was normal. The serology was negative.

On 2-15-51 the left common carotid, external and internal carotid arteries were exposed at operation. There were seen to be no pulsations in the common carotid artery, in the carotid bulb or in the internal carotid artery at all. The external carotid artery was pulsating and the branches of ascending pharyngeal and the superior thyroid arteries were all pulsating well, probably from collateral circulation in the neck through the branches. The internal carotid artery was then sectioned above the bifurcation and it was found to be completely thrombosed. A large segment of the internal carotid artery in its wall was then taken out to effect the periarterial sympathectomy and for biopsy.

Comment: This patient had an apoplectic onset of symptomatology. Absence of pulsation of the carotid artery, decreased pulsation of the temporal and facial arteries on the same side were present. The diagnosis of occlusion of the common carotid and internal carotid arteries was proved at operation and a section of the internal carotid artery was removed. Of particular interest was the presence of blindness and a primary optic atrophy on the side of the occluded arteries.

CASE XVII. U.H. - _ _., this

sixty-eight year old white male was admitted to the hospital on 8-2-51. In 1943, this patient had the sudden onset of left sided weakness associated with marked diminution of vision in the right eye. The weakness gradually improved and cleared in one month. Diminution of vision persisted. At the time of the last admission to hospital, the patient was markedly cachectic and expressed the desire to die.

The routine blood and urine studies were negative. The blood serology was negative. The systolic blood pressure was 110/80. The neurological examination was negative except for a primary optic atrophy in the right eye and with this eye, the patient could distinguish only fingers.

Suddenly on 9-22-52, the patient became semi-comatose and rapidly lapsed into coma. At this time examination revealed conjugate deviation of the eyes to the left, right primary optic atrophy, and increased left ankle jerk. A tentative diagnosis of right internal carotid artery occlusion was made. The patient's condition rapidly deteriorated and he succumbed on 8-25-51.

Post-mortem examination revealed periarteritis nodosa with involvement chiefly of the kidneys, muscle, adrenal, spleen and bladder. No evidence of periarteritis nodosa was discovered in the internal carotid artery. The right ophthalmic artery is represented only as a thin fibrous band.

Comment: This patient had an apoplectic type of onset of illness. Of interest is the persistence of the visual difficulty and the demonstration of a primary optic atrophy in the involved eye. Eight years after the initial episode, the patient suddenly lapsed into coma and succumbed several days later. Of interest, is the demonstration of periarteritis nodosa in the organs of the body other than the carotid arteries and nervous system. The thrombosis of the right internal carotid artery and obliteration of the right ophthalmic artery were present at post-mortem. The encephalomalacia was in the distribution of the right middle cerebral artery.

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II. MEDICAL SCHOOL NEWS

Coming Events

November 21 J. B. Johnston Lectureship in Neurology; "Hypophysectomy in Man," Prof. Herbert Olivecrona, Professor of Neurosurgery, Stockholm, Sweden; Museum of Natural History Auditorium; 8:00 p.m.

November 21-22 Continuation Course: Conference on Pemphigus and the Bullous Dermatoses for Dermatologists

December 4-6 Continuation Course in Endocrinology for General Physicians

December 5 Journal-Lancet Lecture; "Some Studies on Experimental Diabetes," Dr. Dwight J. Ingle, Senior Research Scientist, Research Division, The Upjohn Company, Kalamazoo, Michigan; Owre Amphitheater; 8:00 p.m.

* * *

Continuation Course

The University of Minnesota will present a continuation course in Endocrinology at the Center for Continuation Study on December 4 to 6. This course is intended primarily for physicians engaged in general practice and will include discussions of recent advances in the clinical use of ACTH and cortisone and the management of diabetes mellitus and other common endocrine disorders. Dr. Dwight J. Ingle, Senior Research Scientist, Research Division, The Upjohn Company, Kalamazoo, Michigan, will be the guest faculty member for the course. Dr. Ingle will also present the annual Journal-Lancet Lecture on December 5. The course will be presented under the direction of Dr. C. J. Watson, Professor and Director, Department of Medicine, and the remainder of the faculty will include members of the full-time and clinical staff of the University of Minnesota Medical School and the Mayo Foundation.

* * *

Faculty News

At the recent meeting of the Central Society for Clinical Investigation which was held on November 7 and 8 in Chicago the Department of Medicine was represented by Doctors C. J. Watson, Frederick W. Hoffbauer, Wesley W. Spink, Samuel Schwartz, Paul Lowry, Richard Frey, Paul Frick, Rudi Schmid, and Robert Wise. Dr. Lowry presented a paper entitled, "The Conversion of N^{15}-Labeled Mesobilirubinogen to Stercobilinogen by Fecal Bacteria," and Dr. Frick gave a paper on "Hemophilia-like Disease Following Pregnancy with Transplacental Transfer of an Acquired Circulating Anticoagulant."

At the same meeting Dr. Robert A. Good, Assistant Professor, Department of Pediatrics, discussed "Prevention of Shwartzman Reaction by Heparin," and a paper entitled, "Production of Fibrinoid Necrosis in the Arteries of the Heart by the Shwartzman Reaction in Streptoccus-Infected Rabbits" was presented by Doctors Lewis Thomas, Floyd Denny, and Miss Joan Floyd.

Important Notice

The personnel of the Record Room are being very severely inconvenienced and a fair number of patients are having their studies in the Out-patient Clinic and in the hospital very greatly delayed by the fact that certain members of the staff are in the habit of concealing hospital charts in their offices during the course of case studies, or of taking them home for study. When this is done and a patient comes to the Clinic, someone in the Record Room inevitably wastes a great deal of time in trying to find the chart, and patients have been stymied for as long as two or three days while futile attempts were being made to find such charts.

The personnel of the Record Room, of the Hospital Administration, and of the Record Committee will very much appreciate it if members of the staff will refrain from taking charts out of the building. If they take them to their offices in the building, someone in the office should be informed as to the whereabouts of the chart.

III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

November 24 - 29, 1952

Monday, November 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; W-612, U. H.

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

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

11:30 - 12:30 Physical Medicine Seminar; Fundamentals of the Nervous Motor System; W. G. Kubicek; Heart Hospital Auditorium.

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

12:30 - 1:30 Physiology Seminar; Vencmotor Mechanisms; F. J. Haddy; 214 Millard Hall.

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

4:00 - 5:30 Seminar on Fluid and Electrolyte Balance; Gerald T. Evans; Todd Amphitheater, U. H.

4:00 - 5:00 Pediatric Seminar; Choledochal Cyst; Dr. Novick; Sixth Floor West, U.H.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

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

4:30 - 6:00 Physiology 114A and Cancer Biology 140 -- Research Conference on Cancer, Nutrition, and Endocrinology; Drs. Visscher, Bittner, and King; Immune Reactions; Howard Shear; 129 Millard Hall.

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

Minneapolis General Hospital

9:30 - Pediatric Rounds; Eldon Berglund; Newborn Nursery, Station C.

10:30 - 12:00 Tuberculosis and Contagion Rounds; Thomas Lowry; Station M.

11:00 - Pediatric Rounds; Erling Platou; Station K.

Monday, November 24 (Cont.)

Minneapolis General Hospital (Cont.)

12:30 - Surgery Grand Rounds; Dr. Zierold; Sta. A.

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

2:00 - Pediatric Rounds; Robert A. Ulstrom; Stations I and J.

Ancker Hospital

8:30 - 10:00 Chest Disease Conference.

1:00 - 2:00 Medical Grand Rounds.

Veterans Administration Hospital

8:00 - 9:00 Neuroradiology Conference; J. Jorgens, R. C. Gray; 2nd Floor Annex.

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

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

2:00 - Psychosomatic Rounds; Bldg. 5.

3:30 - Psychosomatic Rounds; C. K. Aldrich; Bldg. I.

Tuesday, November 25

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Pediatric Conference; L. G. Rigler, I. McQuarrie and Staff; 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.

12"30 - 1:30 Physiology 114D -- Current Literature Seminar; 129 Millard Hall.

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

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

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

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

Ancker Hospital

8:00 - 9:00 Fracture Conference; Auditorium.

8:30 - 9:30 Medical-Roentgenology Conference; Auditorium.

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

Tuesday, November 25 (Cont.)

Minneapolis General Hospital

10:00 - Pediatric Rounds; Spencer F. Brown; Stations I and J.

10:00 - Cardiac Rounds; Paul F. Dwan; Station I, Classroom.

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

12:30 - Grand Rounds; Fractures; Sta. A; Willard White, et al.

12:30 - Neuroroentgenology Conference; O. Lipschultz, J. C. Michael and Staff.

12:30 - EKG Conference; Boyd Thomes and Staff; 302 Harrington Hall.

1:00 - Tumor Clinic; Drs. Eder, Cal, and Lipschultz.

1:00 - Neurology Grand Rounds; J. C. Michael and Staff.

Veterans Administration Hospital

7:30 - Anesthesiology Conference; Conference Room, Bldg. I.

8:30 - Infectious Disease Rounds; Dr. Hall.

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

9:00 - Liver Rounds; Drs. Nesbitt and MacDonald.

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

10:30 - Surgery Tumor Conference; L. J. Hay, J. Jorgens; Conference Room, Bldg. I.

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

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

3:30 - 4:20 Autopsy Conference; E. T. Bell and Donald Gleason; Conference Room, Bldg. I.

Wednesday, November 26

Medical School and University Hospitals

8:00 - 9:00 Roentgenology-Surgical-Pathological Conference; Paul Lober 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 Staff; Todd Amphitheater, U. H.

12:30 - 1:20 Radioisotope seminar; Physical Characteristics and Biological Applications of Some Important Radioisotopes; Sol Sandhaus; 110 Botany Bldg.

Wednesday, November 26 (Cont.)

Medical School and University Hospitals (Cont.)

1:30 - 3:00 Physiology 114B -- Circulatory and Renal System Problems Seminar; Dr. M. B. Visscher, et al; 214 Millard Hall.

4:00 - 5:30 Physiology 114C -- Permeability and Metabolism Seminar; Nathan Lifson; 214 Millard Hall.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

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

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

Ancker Hospital

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

2:00 - 4:00 Medical Ward Rounds;

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

Minneapolis General Hospital

9:30 - Pediatric Rounds; Max Seham; Stations I and J.

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

11:00 - Pediatric Seminar; Arnold Anderson; Classroom, Station I.

11:00 - Pediatric Rounds; Erling S. Platou; Station K.

12:30 - Pediatric Conference; Influenzal Meningitis; J. B. Balkan; Classroom, Station I.

1:30 - Visiting Staff Case Presentation; Station I, Classroom.

Veterans Administration Hospital

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

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

2:00 - 4:00 Infectious Disease Rounds; Main Conference Room, Bldg. I.

4:00 - 5:00 Infectious Disease Conference; W. Spink; Conference Room, Bldg. I.

4:00 - Combined Medical-Surgical Conference; Conference Room, Bldg. I.

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

Thursday, November 27 (HOLIDAY)

Friday, November 28

Medical School and University Hospitals

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

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

10:30 - 11:50 Medicine Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

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; Football Pictures; Powell Hall Amphitheater.

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

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

4:00 - 5:00 Physiology 124 -- Seminar in Neurophysiology; Ernst Gelhorn; 113 Owre Hall.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

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

9:30 - Pediatric Rounds; Wallace Lueck; Station J.

10:30 - Surgery Conference; Oswald Wyatt; Tague Chisholm; Station I., Classroom.

12:00 - Surgery-Pathology Conference; Dr. Zierold, Dr. Coe; Classroom.

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

1:15 - X-ray Conference; Oscar Lipschultz; Classroom, Main Building.

2:00 - Pediatric Rounds; Robert Ulstrom; Stations I and J.

Veterans Administration Hospital

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

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

Saturday, November 29

Medical School and University Hospitals

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

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

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

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

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

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

Ancker Hospital

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

Minneapolis General Hospital

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

11:00 - Pediatric Clinic; C. D. May and Floyd Denny; Classroom, 4th Floor.

Veterans Administration Hospital

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

8:30 - 11:15 Hematology Rounds; Drs. Hagen, Goldish, and Aufderheide
11:15 - 12:00 Morphology . . . . . Dr. Aufderheide