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ACTH and Cortisone  
in Eye Disease

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UNIVERSITY OF MINNESOTA HOSPITALS  
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# I. THE EVALUATION OF ACTH AND CORTISONE IN EYE DISEASE

Burton G. Olson

Since 1949, when Hench, Kendall, Slocumb, and Polley<sup>1</sup> reported that Compound E (Cortisone) and ACTH had a dramatic, though temporary, therapeutic effect on rheumatoid arthritis, there have been reports of the therapeutic value of these products from every branch of medicine on a wide variety of disease conditions. In most cases the therapeutic value ceases with, or shortly after, the cessation of treatment; but these reports have shown that we are entering into a new, very large field of medicine. The implications and potentialities of these and related substances are exceedingly significant. Much remains to be learned about their action and results.

For many years, ophthalmologists have been confronted with acute and recurrent inflammations of the uveal tract and episcleral tissue that have been associated with rheumatoid arthritis. The results in the treatment of arthritis, and the known effects of these products on the acute inflammatory diseases of structures composed of collagenous tissue and of mesenchymal tissue<sup>2</sup> led ophthalmologists to investigate the use of ACTH and cortisone in the treatment of eye inflammation, since the cornea and sclera are high in collagen content. From the iritis associated with arthritis, we have branched out and have treated with favorable results other eye diseases that heretofore have been quite difficult to control and which have frequently been productive of blindness. It is the purpose of this paper to present a series of cases which were treated at the University Hospitals and at the Veterans Hospital, and to combine our results with those that have been presented in the ophthalmic literature, and to see if our conclusions will in any way clarify the picture in the use of these drugs in eye diseases.

In order to attempt a fuller understanding of these observations, a review of the pertinent physiological effects of

ACTH and cortisone is indicated. These physiological changes are thought to be mainly or entirely mediated through stimulation of the adrenal cortex by the adrenocorticotrophic hormone of the anterior pituitary. The parenteral administration of ACTH stimulates the adrenal secretion of cortisone and other cortical hormones, provided functioning adrenal cortex is present<sup>3</sup>. The administration of cortisone parenterally depresses adrenal cortical secretion, and is supplantive therapy for an already deficient amount of cortisone secreted. ACTH causes a hypertrophy of the adrenal cortex, and cortisone causes an atrophy. These changes are reversible when the administration of the drugs is stopped.

The adrenal cortex upon stimulation elaborates three groups of hormones: (1) the group of hormones such as cortisone and Compound F that regulate carbohydrate, protein and fat metabolism, and the control of lymphoid tissue and circulating eosinophils; (2) the group, such as desoxycorticosterone, that affect electrolyte balance; and (3) the group that have an androgenic effect similar to that of testicular androgens.

It is now quite evident that the physiological responses that are manifested by the administration of ACTH and cortisone simulate in most aspects those encountered in the alarm reaction, recognized by Selye in 1936. The alarm reaction is produced by a stress to the patient, and this stress also invariably causes some degree of damage. The reaction of the adrenal glands to various types of stresses, be these trauma, infection, emotion, or other types of internal or external environmental stresses, determines the health of the patient.

Ophthalmologists have long used intravenous foreign protein therapy in the management of diseases of the uveal tract. Milk and typhoid vaccine have been used extensively, and the response of the disease to the foreign protein has often been dramatic. In 1946, Selye<sup>6</sup> introduced his concept of the general adaptation syndrome. Prior to that, it was thought that the severe general reaction,

which was obtained with protein therapy, and which was accompanied by leukocytosis, general and local vasodilatation, increased antibody titer, etc., stimulated the mechanism of immunity of the entire body to excessive response. Since Selye's work, it is evident that foreign protein is an "alarming stimulus", and that such a stimulus indirectly activates the adrenal cortex. There is an increased resistance which occurs in a "counter-shock phase" and which is largely dependent on the increased output of 11,17 oxysteroids from the adrenal cortex.

Arendshorst and Falls<sup>7</sup> have shown that the response of the adrenal cortex in cases treated with ACTH and with typhoid H antigen are similar. By means of eosinophil counts, they found that the same eosinopenia occurred when typhoid vaccine was given as with ACTH. However, the response was not as rapid; the maximal eosinopenia occurred about 18 hours after injection. These authors followed the response of the adrenal cortex and coordinated the effects of the treatment in 21 patients with a variety of eye diseases. In 6 patients, little or no eosinophil drop occurred, and these patients also exhibited unfavorable therapeutic response to their disease. The other 15 patients showed the typical drop in circulating eosinophils, down to practically complete disappearance; their condition also improved.

Potts, Johnson and Goodman<sup>8</sup> have also called attention to the similarity of the response in cases of uveitis treated with typhoid vaccine. In sixteen intravenous administrations of typhoid they found without exception the same response as that which followed ACTH therapy.

In this respect, Sayers and Sayers<sup>13</sup> have shown in the experimental animal that both heat and injected killed typhoid organisms produce changes in the adrenal cortex which are characteristic of the response to stress.

From the series of cases which I will present and from those of other workers<sup>2,4,9</sup>, it seems definite that cortisone or ACTH causes a more rapid resolu-

tion of certain inflammatory diseases of the eye than does the presently employed foreign protein therapy. However, there are advantages and disadvantages to both. The most obvious advantage of foreign protein therapy is its low cost and availability, while the major disadvantage is the rapid development of tolerance to its effect. In patients with hypertension, congestive heart failure, or advanced degenerative renal disease, the use of fever therapy or ACTH may be limited. Cortisone may be the safest of the three in these cases. There are patients who apparently do not have a normally functioning adrenal cortex in which cortisone may give the desired response. There are patients who show improvement on ACTH when cortisone has failed. The reason for this is undoubtedly due to a release of some adrenal cortex substance other than Compound E. Little is known regarding modes of actions and variable responses from patient to patient. In addition, topical application of cortisone is proving to be very effective in anterior segment diseases, some of which were hopeless prior to the use of the steroids of the adrenal cortex.

The mode of action of ACTH and cortisone is unknown. It seems probable that they act in some way at the local tissue level on the mesenchymal tissues. The beneficial effects of these hormones appear primarily to control the inflammatory and exudative phases of the ocular diseases. There are many diseases in the eye that present allergic backgrounds for the inflammatory reaction. Nongranulomatous iritis is currently believed to be a phenomenon due to bacterial hypersensitivity.<sup>10</sup> In tuberculous disease, the inflammatory phase is dependent upon a hypersensitivity of the tissues to tuberculo-protein. In sympathetic ophthalmia there is sound reason to believe that hypersensitivity to uveal pigment is a part of the symptom complex.<sup>10</sup>

Experimental studies by Woods<sup>4</sup> show that ACTH administered systemically, and cortisone systemically or topically, block, in the eye, the inflammatory and

exudative phases of the anaphylactic protein reaction. He sensitized six rabbits to horse serum. Two were used as controls, two were given ACTH and two cortisone for four days before an intoxicating injection of horse serum in the anterior chamber of the eye and for four days after the injection. The controls showed a prompt and violent ophthalmic reaction. The treated rabbits showed a complete blocking of the anaphylactic ocular reaction. The blocking action was found to be temporary; it had completely disappeared one month later when all the rabbits were again given anterior chamber injections of horse serum. All the rabbits now reacted with an equally severe ophthalmic reaction.

Woods<sup>4</sup> also showed that 10 mg. of cortisone per day gave a complete blocking; smaller doses of 5.0 mg. and 2.5 mg. gave proportional partial blocking actions.

Similar experiments with rabbits sensitized to beta streptococci showed a blocking of the bacterial hypersensitivity with cortisone. Focal reactions to tuberculin, in rabbits with experimentally produced tuberculous eyes, were also blocked. Although the number and size of the tubercles in the iris continued to increase, as long as cortisone was continued, the eyes had the appearance of a progressive tuberculosis totally<sup>4</sup> devoid of any inflammatory reaction.

Good<sup>28</sup> has produced the Shwartzman phenomenon<sup>29</sup> in the eyes of rabbits, and is now experimenting on the effects of topical cortisone in inhibiting or altering this reaction. His experiments as yet are not complete. It will be exceedingly interesting to learn the results in this phase of the blocking action of cortisone.

I am presenting a series of 43 cases of various eye diseases which we have treated with ACTH and cortisone. Thirty two of the cases were patients at the University Hospitals and 11 were at the Veterans Hospital. The patients treated were in an age range of 3 to 87

years. Unless otherwise stated, the dosage of ACTH was 20 mg every 6 hours parenterally, of cortisone 100 mg parenterally once a day, and of topical cortisone a saline suspension of 8 mg. per cc, one drop instilled into the conjunctival sac every hour. Doses were gradually reduced during the three to five days prior to stopping the treatment. The maintenance dose of topical cortisone was one drop of the above suspension three or four times daily. The routine measures to guard against side effects were the daily weighing of the patient, giving a low sodium diet with 2 - 4 Gms of K Cl daily, routine blood and urine examinations. The total circulating eosinophil count was used as an index of response in those receiving the hormones parenterally.

The side effects that occurred with the administration of ACTH were few and minimal with the exception of Case No. 42 who developed an acute psychotic episode, with confusion, delirium and paranoid trends. The side effects, including the latter, were reversible when the drug was stopped. There were no side effects noted with the parenteral or topical administration of cortisone.

Adjunctive therapy, such as mydriatics, hot packs, and antibiotics, was given when it was indicated.

Before beginning the summary of the cases, it may be worthwhile to differentiate between the two main types of iritis. Granulomatous iritis is a chronic, progressive condition associated with syphilis, tuberculosis, sarcoidosis, brucellosis, and some virus infections. The inflammatory reaction is not usually severe; there are large, greasy K.P.'s found with a faint aqueous ray and few cells. Nodules and granulomas are common, and there is a tendency toward the early formation of synchia. Sympathetic ophthalmia falls in this group.

Nongranulomatous iritis is an acute and recurrent inflammation which is associated with rheumatoid arthritis or bacterial sensitivities from focal infections, such as tonsillar and dental abscesses, urethritis and cervicitis. The aqueous ray is very dense with many cells;

the K.P.'s are pin point in size and synchia occur only after the first attack; there are no nodules found.

Table I shows that 74% of the cases treated were improved. Good improvement indicates a complete resolution of the primary disease with objective improvement in visual acuity. Those classified as fair improvement showed objective and subjective signs of improvement, but little or no improvement in visual acuity. Those classified as unimproved showed none of the above.

Table I

Number in the series	43	100%
Good improvement	26	60%
Fair improvement	6	14%
No improvement	11	26%

The incidence of recurrence of the primary disease as shown in Table II is similar to the findings in rheumatoid arthritis. Recurrences are very common. It is clear that maintenance doses must be given to control the symptoms. In the 15 cases that have been followed from three to nine months with no recurrences, we cannot deduce that they still may not recur. Many of the ocular diseases which are included in this series are prone to recur. However, we now have a more effective means of preventing the damage that occurs with each successive recurrence. As Sprague<sup>11</sup> has said, "It is now apparent that diseases which are favorably modified by cortisone and ACTH are not cured by them." If the inflammatory, exudative, and destructive phase of the disease can be blocked long enough for the normal defense mechanism of the body to take over, or for the other appropriate therapeutic measures, such as antibiotics, desensitization, etc., to accomplish their effect, then these agents have accomplished an important task in the treatment of eye diseases.<sup>4</sup>

Table II

Number showing improvement	32	100%
Number showing recurrence of disease following cessation of treatment, and necessitating a second course or maintenance doses	17	53%
Number showing no recurrence	15	47%

In Table III, the obvious discrepancy between the total number of diseases and the total number of cases is explained by the occurrence of more than one diagnosis in several patients. It can be seen from this table that the most favorable therapeutic results were obtained in the treatment of nongranulomatous diseases. It is also apparent that by far the greatest benefit was found in diseases of the anterior segment of the eye.

Table III

<u>Disease treated</u>	<u>Im- proved</u>	<u>Unim- proved</u>
Conjunctivitis, vernal	1	0
Keratitis, parenchymatous	7	0
Keratitis, sclerosing	1	0
Keratitis, interstitial, luetic	1	1
Kerato-conjunctivitis, phlyctenular	1	0
Kerato-conjunctivitis, non-specific	4	0
Episcleritis	1	0
Ring abscess of the cornea	1	0
Iridocyclitis - nongranulomatous	14	2
Iridocyclitis - granulomatous	4	4
Sympathetic ophthalmia	1	1
Ocular complications of erythema multiforme exudativum	0	1
Glaucoma, secondary	6	4
Vitreous hemorrhage, recurrent	0	1
Endophthalmitis, postoperative	1	1
Choroiditis, juxta papillary	0	1

Table IV merely shows which hormones were used and how they were administered. It is significant that the diseases that we considered fulminating enough to require the most concerted treatment were the ones in which we used ACTH. Example: involvement of the iris with secondary glaucoma. Those diseases which were not as severe, such as parenchymatous keratitis, responded to topical cortisone equally as well as with ACTH. We found that the quickest response occurred with the systemic administration of ACTH. Some of these patients required maintenance installations of cortisone drops. The deeper and more serious inflammations did not respond well

to cortisone topically when it was the only therapy.

Table IV

<u>Drug Used</u>	<u>No. of Cases</u>
ACTH only	15
Cortisone only	3
Topical cortisone only	16
ACTH and topical cortisone	8
ACTH, parenteral and topical cortisone	1

The summary of these cases is found in Tables V, VI, and VII in the Bulletin. Four cases which are representative of the group receiving favorable results in the treatment with ACTH and cortisone are presented in detail.

Table V

Case No.	Diagnosis	Good Improvement	
		Drug	Results
1.	Keratitis, parenchymatous, R.E. Iridocyclitis, secondary, R.E.	ACTH 12 days Topical cortisone 2 months	Pain, photophobia subsided in 1 day. Opacity gradually cleared. V.A. improved from 20/40 to 20/20. Recurrence when topical cortisone stopped. Again controlled by drops.
2.	Kerato-conjunctivitis, chronic bilateral	topical cortisone 3 wks.	Cleared up conjunctival infection and superficial punctate staining, and enabled squint surgery to be done on left eye. No recurrence.
3.	Keratitis, disciformis, recurrent, R.E.	topical cortisone 3 mo.	Clearing of corneal infiltrate. Symptom free on drug t.i.d. Recurrence when drug stopped.
4.	Kerato-conjunctivitis, phlyctenular, bilateral, prob. on tbc hypersensitivity	topical cortisone 2½ mo.	Severe pain, photophobia disappeared in 2 days. Infiltrate cleared and grossly visible corneal vessels disappeared in 1 wk. Vision improved from L & M to 20/60. Recurrence when drug stopped.
5.	Keratitis, sclerosing, L.E. prob. on tbc hypersensitivity	topical cortisone 3 mo.	Disappearance of scleral nodule and redness in 3 weeks. Shrinking of corneal infiltrate in 6 wks. V.A. improved from 20/35 to 20/20. Recurrence when drug stopped.

Table V (Cont.)

Case No.	Diagnosis	Good Improvement	
		Drug	Results
6.	Keratitis, R.E. from corneal anesthesia	topical cortisone 2 wks.	Disappearance of stromal infiltration and superficial staining. Coincident return of sensation. No recurrence.
7.	Keratitis, disciformis, L.E. Secondary iridocyclitis, L.E.	ACTH 15 days	Disappearance of pain and epiphora; aqueous ray, cells and K.P.'s disappeared. Marked decrease in corneal infiltrate. No recurrence.
8.	Keratitis, disciformis, R.E. prob. on tbc hypersensitivity	topical cortisone 2 mo.	Marked clearing of infiltrate V.A. improved from 20/200 to 20/25. Recurred when drug stopped.
9.	Keratitis, parenchymatous, L.E. Secondary iritis and glaucoma, L.E.	topical cortisone 2 mo.	Tension fell to normal; iritis subsided; keratitis unchanged V.A. improved from 20/100 to 20/50. Required maintenance drops.
10.	Episcleritis, bilateral secondary glaucoma, bilateral	ACTH for 14 days. Recurrence 2 wks. later. ACTH 10 days Maintained on small dose t.i.d.	Eyes became white in 24 hours. Tension fell to normal in 4 days. Details in case report.
11.	Iridocyclitis, bilateral, non-gran. Secondary glaucoma, L. E.	ACTH - one course for 16 days; one for 6 days. Topical cortisone t.i.d.	Tension fell from 61 mm. Hg. to normal; aqueous ray and cells disappeared. V.A. improved in rt. eye from 20/30 to 20/20 and in left from 20/200 to 20/100. Recurrence required maintenance drops.
12.	Iridocyclitis, R.E., recurrent non-gran.	Topical cortisone 3 wks.	Previously treated twice with foreign protein. K.P.'s, aqueous ray and cells disappeared. V.A. improved from 20/40 to 20/20.
13.	Iridocyclitis, R.E., recurrent non-gran.	Cortisone 18 days for one course; 3 wks. with tapering off for second course.	Disappearance of iritis within one week; drug stopped abruptly gave recurrence. Tapering off of drug in second course gave no recurrence.

Table V (Cont.)

Case No.	Diagnosis	Good Improvement	
		Drug	Results
14.	Iridocyclitis, R.E., non-gran.	ACTH for 13 days. Topical cortisone t.i.d.	Disappearance of aqueous rays and cells. Improvement of vision from 20/40 to 20/20. Required maintenance drops.
15.	Iridocyclitis, R.E., non-gran. Secondary glaucoma, R.E.	Cortisone 2 wks.	Tension fell to normal. Aqueous ray and cells disappeared. Improvement of vision from L & M to 20/50. No recurrence in 9 mo.
16.	Iridocyclitis, R.E. non-gran.	ACTH 8 days	Disappearance of aqueous ray and cells in 5 days. Improvement of vision from inability to keep eye open because of photophobia to 20/20.
17.	Iridocyclitis, recurrent L.E., non-gran.	Topical cortisone 6 wks.	Nine previous attacks treated with foreign protein. This attack subsided on topical cortisone, and hospitalization was avoided.
18.	Iridocyclitis, L.E., non-gran.	ACTH 2 wks.	Immediate relief of pain and photophobia. Subsidence of attack in 1 wk.
19.	Iridocyclitis, L.E., non-gran.	ACTH 11 days	Disappearance of aqueous ray and cells in 1 wk. V.A. improved from 20/200 to 20/70.
20.	Sympathetic ophthalmia	ACTH in IV drip 4 days Topical cortisone 2 mo.	See case report for details.
21.	Iridocyclitis, post-operative, L.E.	ACTH 11 days	Cataract extraction. 3 wks. previously. Inflammation subsided in 4 days. Improvement in vision from 20/200 to 20/40.
22.	Iridocyclitis, post-operative, L.E.	ACTH 2 wks. Topical cortisone 2 mos.	Cataract extraction 10 wks. previously. Iritis subsided in 5 days. Recurrence required maintenance dose.
23.	Postoperative hyphema with secondary glaucoma, L.E.	Topical cortisone 3 wks.	Successful iridencleisis 3 wks. previously. Hyphema absorbed in 4 days; tension fell to normal and has remained normal for 3 mo.

Table V (Cont.)

Case No.	Diagnosis	Good Improvement	
		Drug	Results
24.	Postoperative ring abscess of the cornea, L.E. Secondary iridocyclitis, L.E.	ACTH 12 days	Clearing of corneal infiltration within 48 hours. Vision improved from L & M to 20/40. Details in case report.
25.	Postoperative hyphema with secondary glaucoma, L.E.	Topical cortisone 6 wks.	Cataract extraction 3 wks. previously. Spontaneous hyphema from sneezing. Tension fell from 55 mm Hg to normal in 1 wk. Vision improved from light perception to 20/30.
26.	Iridocyclitis, post-operative chronic, L.E.	ACTH 27 days	Enabled uneventful cataract extraction, R.E., which otherwise may have resulted in iritis, as in L.E. Details in case report.

Table VI

Case No.	Diagnosis	Fair Improvement	
		Drug	Results
27.	Keratitis, parenchymatous, R.E.	Topical cortisone 6 wks.	Disappearance of foreign body sensation. Opacity about the same. Vascularization of cornea is shrinking. Improvement of vision from L & M to 20/200. Still being treated.
28.	Keratitis, striate	Topical cortisone 4 wks.	Immediate relief to pain and epiphora. Slow clearing of infiltrate. Still being treated.
29.	Iridocyclitis, bilateral, granulom. Keratitis	Cortisone 11 days. ACTH 6 days Topical cortisone 4 wks.	Iritis disappeared in both eyes. No change in keratitis.
30.	Conjunctivitis, vernal, bilateral corneal ulceration and vascularization, bilateral.	Topical cortisone 5 mo.	Pt. had IM cortisone elsewhere for 10 days with no improvement. Had surgery here to remove granulation tissue of lids and Beta irradiation for vascularization. Topical cortisone has enabled pt. to hold eyes open, and has held disease stationary.

Table VI (Cont.)

Case No.	Diagnosis	Fair Improvement	
		Drug	Results
31.	Iridocyclitis, gran., L.E. prob. due to sarcoid	ACTH 20 days Topical cortisone	No improvement on ACTH. Subsiding of inflammation on topical cortisone which may have been coincidental. Improvement of vision from 20/50 to 20/20.
32.	Iridocyclitis, acute, R.E., non-gran.	ACTH 8 days Topical cortisone 2 wks.	Improved for 4 days; then arrest in improvement. Patient failed appointment; hence unknown results.

Table VII

Case No.	Diagnosis	Unimproved	
		Drug	Results
33.	Keratitis, interstitial, bilateral. Congenital syphilis.	ACTH 24 days	No change in vision or appearance of lesions.
34.	Ocular complications of erythema multiforme exudativum.	Topical cortisone 1 mo. ACTH 1 wk.	No improvement in vision. No change in infiltration or vascularization.
35.	Iridocyclitis, bilateral, chronic non-gran. Oc- clusio pupillae, bilateral	Cortisone 2 mo. Cortisone maintenance 6 mo.	Pt. had 6 surgical procedures in attempt to make a new pupil, L.E. Vision unchanged.
36.	Iridocyclitis, post- operative, L.E.	Topical cortisone 3 wks.	No change in ray or cells. No change in vision.
37.	Iridocyclitis, L.E. Secondary glaucoma, L.E.	Topical cortisone	Tension remained high. No improvement in iritis. Eye enucleated 3 mo. later.
38.	Iridocyclitis, R.E., gran. Secondary glaucoma, R.E.	ACTH 1 mo.	Tension remained high. Surgery to lower it unsuccessful. No improvement in vision.
39.	Iridocyclitis, postoper- ative, chronic, R.E. Secondary glaucoma, R.E.	ACTH 12 days	Cataract extraction 2 years previously. No change in tension, vision or iritis.
40.	Sympathetic ophthalmia, L.E. Phthisis, L.E. bulbi	ACTH 3 days	Blind. Given short course only for satisfaction of patient. No improvement.

Table VII (Cont.)

Case No.	Diagnosis	Unimproved	
		Drug	Results
41.	Recurrent vitreous hemorrhage (Eale's Disease)	ACTH 1 wk.	No change in vision or hemorrhage that time itself would not give.
42.	Endophthalmitis, postoperative L.E.	ACTH 5 days	Pt. had atypical myelogenous leukemia and diabetes mellitus. Cataract extraction 2 days previously. No change in appearance of eye. Pt. developed an acute psychotic episode which disappeared in 24 hours after ACTH stopped.
43.	Choroiditis, juxta-papillary bilateral	ACTH 2 wks.	No improvement in vision. Size of scotoma increased while ACTH was given.

Case No. 10, , a 49-year old female, was first seen in the University Hospitals Eye Clinic on December 12, 1950. During the preceding year she had had recurrent bouts of joint pain with swelling and heat. These attacks appeared suddenly and disappeared in a few days. The knees were most frequently involved and the toes were also affected.

Fourteen weeks previous to her first visit she had noted the onset of a painful, red right eye, with itching. The left eye had been similarly involved for six weeks. Her vision was blurred for six weeks. Her past history was negative.

Examination of her eyes revealed that the conjunctiva and episclera were bilaterally injected and edematous. No discharge was seen in the conjunctival cul de sacs. Her visual acuity with correcting lenses was 20/20 in each eye. The cornea in each eye was edematous; slit lamp exam showed no K.P.'s, no aqueous ray nor cells, no iris nor scleral nodules. The fundi were normal. The intraocular tension was elevated to 49 mm. Hg in the right eye and 43 mm Hg. in the left eye as measured with a Schiottz Gradle tonometer. Her peripheral visual fields were normal; however,

the central fields showed a definite depression. There was a baring of the blind spot with a 1 mm. test object in the left eye, and with a 3 mm. test object in the right eye.

Her general physical condition was essentially negative. She was admitted to the hospital on December 15, 1950.

Laboratory work was reported as follows: normal CBC and urine; sedimentation rate 73 mm. in 60 minutes; blood uric acid was 3.4, total proteins 6.0 Gm. with a reversal of the albumin - globulin ratio. Blood serum added to non-lupus bone marrow showed no L.E. cells. X-rays of her hands showed no gouty changes.

The diagnosis of bilateral episcleritis with secondary glaucoma was made, and after unsuccessful treatment with miotics, ACTH was begun intramuscularly. She was given 50 mg. at the first dose and then 20 mg. q 6 h. The patient stated she had complete relief of her discomfort four hours after the first injection. Her eyes were completely white and the intraocular tension was normal after 72 hours. The ACTH was given for eight days. When administration was reduced to once a day, she had

a mild flare-up of her symptoms. She was again given 20 mg. q 6 h for 3 days and then placed on topical cortisone. The eyes remained white and she was discharged on January 6, 1951, on topical cortisone q 1 h. She returned on January 19, 1951, with a recurrence, and was again admitted for further ACTH therapy. She was again given 20 mg q 6 h for three days, and after 24 hours she was symptom-free. It was found that a maintenance dose of 10 mg t.i.d. controlled her symptoms. We are now in the process of attempting to control her with topical cortisone alone.

She showed a good response to ACTH; her admission eosinophil count was 81; 48 hours later it was zero. Her visual acuity has remained at 20/20 in each eye. The central fields are now normal. She has had fairly heavy doses of ACTH for three months with no side effects.

This case demonstrates the typical dramatic response to the hormone, and also the fairly typical pattern of recurrences when treatment is stopped.

Case No. 24: , 87 year old male, was first seen in the Eye Clinic on September 21, 1950, with a history of gradual, painless loss of vision in each eye for six years previously. His corrected vision in the right eye was 20/100 and in the left eye was limited to light and motion. Examination of the eyes revealed bilateral cataracts. His general physical condition was excellent for his age. On September 26, 1950, an extra-capsular cataract extraction was performed on his left eye. On the fifth postoperative day, the left eye showed a very cloudy cornea with a hypopyon present. The aqueous was so purulent that the iris could barely be seen. A diagnosis of postoperative ring abscess of the cornea with a secondary uveitis was made, and the patient was started on intramuscular ACTH 20 mg q 6 h and penicillin. Twenty four hours later, the signs of the purulent uveitis had almost disappeared and after 12 days of ACTH therapy the cornea and media were clear. An aqueous ray has persisted to the present time. His vision has been corrected to 20/40 in his left eye.

This case demonstrates the amazing ability of ACTH to clear inflammation and exudation. It can conservatively be said that had it not been for ACTH the vision in this eye would have been lost. Too often similar cases have gone on to a panophthalmitis and subsequent evisceration or enucleation in spite of heavy doses of antibiotics.

Case No. 26, ., 71 year old male, had an intracapsular cataract extraction on his left eye in July, 1948. Six weeks later he developed a low grade uveitis that has persisted to the present time. His vision in that eye went from 20/50 to light and motion in spite of all treatment. On August 22, 1950, because the patient was extremely handicapped visually due to a now advanced immature cataract in his right eye, he was admitted for a course of ACTH prior to surgery on his right eye. He was given ACTH for 16 days preoperatively in a dose of 20 mg. q 6 h. The uveal infection quieted down, but did not disappear. The media cleared enough so that we could now see that he had a hole in the macula of the left eye. On September 5, 1950, an intracapsular cataract extraction was done on his right eye. The ACTH was continued for 11 days postoperatively. The only side effect which was noted on such a long course of ACTH was that the corneo-scleral wound was slow in healing as evidenced by a shallow anterior chamber that occurred eleven days postoperatively and persisted for two weeks. His vision with correcting lenses in the recently operated eye is 20/20. The vision in his left eye remains at light and motion perception.

This case is an example of the change in surgical principles in ophthalmology that has occurred since the discovery of ACTH and cortisone. Previously, we could not operate except in emergencies on an eye or its fellow if an active inflammation were present. Even after the inflammation had subsided, it was advisable to wait three to six months before any surgical procedure was attempted. In the above case we had waited over two years for the inflammation to subside.

Case No. 20, W.J., a 69 year old male,

a private patient of one of our staff members, was admitted to the University Hospitals on November 8, 1950. Visual acuity in his right eye was limited to light and motion, and in his left eye was 20/70. Examination revealed bilateral immature cataracts. On November 9, 1950, an intracapsular cataract extraction was done on his right eye. Postoperatively, he had an eccentric pupil, and on November 19, 1950, when he was discharged, a slight iris prolapse was seen. During the next ten days the iris prolapse increased and on November 30, 1950, he was readmitted to the Hospital, and repair of the iris prolapse was done on that day. His second post-operative course was uneventful and he was discharged on December 7, 1950. On December 18 his corrected vision on the right eye was 20/20-2. On December 26, 1950, his right eye was congested and there was a definite iritis present. He was given a subconjunctival injection of 0.2 cc of 1:3 cortisone suspension in the right eye. The following day, the right eye was improved; however, the left eye now showed a uveal inflammation. The diagnosis of sympathetic ophthalmia was made; he was given a subconjunctival injection of cortisone in the left eye, and readmitted to the hospital. The treatment at this time was cortisone topically q 1 h, atropine and neosynephrine and hot packs to both eyes, plus a continuous IV drip of 16 mg of ACTH in 1000 cc of distilled water. The latter was given daily for four days. Both eyes quieted down within three days, and he was discharged on January 2, 1951, still receiving topical cortisone q 1 h to both eyes. During the next two months, the topical cortisone was gradually tapered off and discontinued. Both eyes remained quiet -- no ray was seen in either eye 2 months after the treatment was started. His vision in the right eye, the exciting eye, is now 20/50 x 3, and there has been no change in the sympathizing eye. He has a secondary membrane over the pupil of his right eye which may require a discission at a later date.

The above diagnosis is necessarily a presumptive diagnosis. The only positive diagnosis of sympathetic ophthalmia

is by microscopic examination. The findings of a bilateral uveitis in a case which had had trauma to the iris surgically are presumed to be sympathetic ophthalmia. If our presumption is correct, and if the final outcome in this case is as good as it is now, then we can say that we now have a very potent tool to wield in the treatment of this serious disease. Before ACTH, complete blindness was very common, and the loss of useful vision was the rule. We certainly cannot infer that we now can prevent sympathetic ophthalmia, but we can say that we have a treatment for it if it does occur. Prior to this, we were without any means of successful treatment.

Quite a few of our cases were given a course of ACTH or were tried on cortisone drops for exploratory reasons. Following an article by Jones and Meyer<sup>12</sup>, who experimentally produced vascularization of the cornea of rabbits by intra-corneal injection of alkali and found that the subconjunctival injection of 1.25 mg of cortisone markedly inhibited the vascularization, we tried these agents on patients with vascularization of the cornea. Case No. 34, with extensive vascularization present for 4 years, showed no improvement on either ACTH or topical cortisone. Case No. 4 with an active phlyctenular kerato-conjunctivitis with recent vascularization and with vessels visible grossly, showed a complete disappearance of the vessels by slitlamp examination in less than one week on topical cortisone. Case No. 27 with a parenchymatous keratitis of 10 years duration showed a shrinking in the size of the vessels with topical cortisone treatment.

Our experience has taught us that to obtain the best results, the hormones should be used early in the course of the disease. As in rheumatoid arthritis, where proliferative and reparative histological changes not influenced by ACTH or cortisone are produced in joints, so in eye diseases, treatment must be begun before synechiae, scarring, complicated cataracts, and vascularization have occurred.

In summary, a composite table (Table VIII) of the cases reported in the literature and our own cases has been prepared. This shows that by far the best results have been obtained in the treatment of nongranulomatous diseases and in diseases of the anterior portion of the eye. It also is quite evident that many of the

diseases which have shown a favorable response fall into the groups that are prone to recurrences and remissions and are also susceptible to treatment with intravenous foreign protein. We do know, however, that ACTH and cortisone give a more rapid resolution of the lesions.

Table VIII

Composite Table of Cases in Literature and Our Cases  
Treated with ACTH and Cortisone

Disease Treated	Improved	Unimproved
Exophthalmos, associated with Graves Disease	0	3
Blepharitis, allergic	3	0
Conjunctivitis, vernal	4	1
Ocular complications of Erythema multiforme exudativum	0	2
Keratitis, parenchymatous	7	1
Keratitis, nonspecific	14	2
Keratitis, interstitial luetic	1	3
Ring abscess of the cornea	1	0
Corneal dystrophy	0	1
Episcleritis	7	0
Iridocyclitis, nongranulomatous	51	4
Iridocyclitis, granulomatous	6	9
Sympathetic ophthalmia	6	1
Glaucoma, primary	0	3
Glaucoma, secondary	13	4
Vitreous hemorrhage, recurrent	0	1
Chorio-retinitis	6	3
Endophthalmitis	0	1
Retinitis pigmentosa	0	1
Retrolental fibroplasia	0	2

With the increasing number of reports on the serious sequelae of ACTH therapy, notably perforated peptic ulcers, we are turning more and more to the topical use of cortisone. Judging from the clinical and experimental data available, and from the fact that 22 of the 32 cases that improved were given topical cortisone either alone or with ACTH, it would appear that topical cortisone may have an immense field of usefulness in controlling anterior segment inflammations. Topical cortisone is easily administered, inexpensive, and has given no side effects or undesirable extension effects. It is possible that subconjunctival injection of cortisone may supersede instillation of drops for some deeper in-

flammations. It is evident that more information must be obtained on the mode of action, proper dosage and administration, and the ultimate results and side effects of these hormones.

However spectacular the action of ACTH and cortisone may seem to be, they definitely are not a panacea for every eye disease. Local heat, atropine, antibiotics are not to be replaced by these agents but should be used either alone or concurrently as indicated.

With the use of ACTH and cortisone, we now have in our hands a means to control, not to cure, eye diseases that in the past have resulted in the loss of vision.

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## II. MEDICAL SCHOOL NEWS

### Coming Events

- April 9 - 11 Continuation Course in Gynecology for General Physicians
- April 16 - 18 Diseases of the Blood in Infancy and Childhood
- April 16 - 21 Continuation Course in Proctology for General Physicians
- April 26 - 28 Continuation Course in Atomic Medicine for General Physicians
- May 7 - 11 Continuation Course in Electrocardiography for General Physicians
- May 8 George E. Fahr Lecture; "Certain Mechanical Peculiarities of the Heart", George E. Burch, Tulane University School of Medicine

### Continuation Courses

Dr. H. C. Hesseltine, Professor of Obstetrics and Gynecology at the University of Chicago, will come to our campus April 9 - 11 to participate in a continuation course in Gynecology. The course, which is given for general physicians, is under the direction of Dr. John L. McKelvey, Professor and Head of the Department of Obstetrics and Gynecology. Dr. Hesseltine will present two formal papers and will participate in round table discussions which make up the closing sessions of each of the three days of the course.

A continuation course in Diseases of the Blood in Infancy and Childhood will be presented for pediatricians April 16 - 18 under the direction of Doctors Irvine McQuarrie and Charles D. May. The course will bring to our campus three medical scientists distinguished for their contributions to the field of hematology. Dr. William Dameshek, Tufts University, Boston, will speak on "The Role of the Spleen in Anemia" and "Purpura." Dr. Clement A. Finch, University of Washington, Seattle, will present "The Metabolism of Iron" and "The Clinical Aspects of Iron Deficiency Anemia and the Anemia of Infection." Dr. Robert W. Heinle, Western Reserve University, Cleveland, will discuss "Recent Studies on the Nature and Role of Intrinsic Factor" and "Leukemia: Investigative and Clinical Aspects of Therapy."

Dr. Robert A. Scarborough, Associate Professor of Surgery, Stanford University Medical School, will participate in a continuation course in Proctology to be presented April 16 - 21. Dr. Scarborough will present the subjects, "Polyps of the Rectum and Colon," "Diverticulitis - Diverticulosis," and "Cancer of the Colon." The course is given under the direction of Doctors Owen H. Wangensteen and Walter A. Fansler.

### Faculty News

Dr. David Glick and Mr. B. G. Malmstrom of the Department of Physiological Chemistry and Dr. Lane Williams of the Department of Anatomy attended the second annual meeting of the Histochemical Society in Detroit on March 19 and 20, 1951. Dr. Glick was elected President of the Society.

Doctors E. A. Boyden, Berry Campbell, Arthur Kirschbaum, L. J. Wells, Lane Williams and Mr. Sam Cornwell presented papers at the sixty-fourth annual meeting of the American Association of Anatomists, Detroit, Michigan, March 21 - 23, 1951.

Dr. Spencer F. Brown, Clinical Instructor, Department of Pediatrics, attended the third annual Florida State Conference on the Handicapped in Tallahassee, Florida, March 19 - 20. Dr. Brown presented a paper on "The Physician's Part in Rehabilitation of the Cleft Palate and Cerebral Palsy Patient."

III.

UNIVERSITY OF MINNESOTA MEDICAL SCHOOL  
WEEKLY CALENDAR OF EVENTS

Visitors Welcome

April 9 - 14, 1951

Monday, April 9

Medical School and University Hospitals

- 9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.
- 9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; M-109, U. H.
- 10:00 - 12:00 Neurology Rounds; A. B. Baker and Staff; Station 50, U. H.
- 11:00 - 11:50 Physical Medicine Seminar; Pre-Vocational Occupational Therapy; Ardietta Johnson; E-101, U. H.
- 11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Eustis Amphitheater, U. H.
- 12:00 - 12:50 Physiology Seminar; Application of Newer Hydrolytic Procedures to Steroid Excretion Studies; Saul Cohen; 214 Millard Hall.
- 12:15 - 1:20 Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.
- 1:30 - 2:30 Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; U. H.
- 4:00 - Public Health Seminar; 113 Medical Sciences.
- 4:00 - Pediatric Seminar; Mid-Century Conference on Children and Youth; R. A. Jensen; Powell Hall Amphitheater.
- 4:30 - 5:30 Dermatological Seminar; M-436, U. H.
- 5:00 - 5:50 Clinical Medical Pathologic Conference; Todd Amphitheater, U. H.
- 5:00 - 6:00 Urology-Roentgenology Conference; C. D. Creevy, O. J. Baggenstoss, and Staffs; Powell Hall Amphitheater.

Minneapolis General Hospital

- 8:30 - 10:00 Pediatric Rounds; Dr. Lowry; 7th Floor Annex.
- 11:00 - Pediatric Rounds; Franklin Top; 7th Floor Annex.
- 1:00 - 2:00 Staff Meeting; Classroom, 4th Floor.
- 1:30 - Pediatric Rounds; Dr. Ulstrom; 5th Floor Annex.

Monday, April 9 (Cont.)Veterans Administration Hospital

- 9:00 - G. I. Rounds; R. V. Ebert, J. A. Wilson, Norman Shriffter; Bldg. I.  
 11:30 - X-ray Conferences; Conference Room; Bldg. I.  
 1:00 - Metabolic Disease Rounds; N. E. Jacobson and G. V. Loomis; Bldg. I.  
 4:00 - Research Conference; Dr. Hall; Conference Room, Bldg. I.

Tuesday, April 10Medical School and University Hospitals

- 9:00 - 9:50 Roentgenology-Pediatric Conference; L. G. Rigler, I. McQuarrie and Staffs; Eustis Amphitheater, U. H.  
 9:00 - 12:00 Cardiovascular Rounds; Station 30, U. H.  
 12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 102 I. A.  
 1:00 - 2:00 Physiology Seminar on Cardiac Metabolism; 129 Millard Hall.  
 3:15 - 4:20 Gynecology Chart Conference; J. L. McKelvey and Staff; Station 54, U. H.  
 4:00 - 5:00 Pediatric Rounds on Wards; I. McQuarrie and Staff; U. H.  
 5:00 - 6:00 X-ray Conference; Presentation of Cases by Veteran's Hospital Staff; Drs. Fink, O'Loughlin, et al; Eustis Amphitheater, U. H.  
 8:00 p.m. Journal Club; E-101, U. H.

Ancker Hospital

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

Veterans Administration Hospital

- 8:45 - Surgery Journal Club; Conference Room, Bldg. I.  
 8:30 - 10:20 Surgery Conference; Seminar Conference Room, Bldg. I.  
 9:30 - Surgery-Pathology Conference; Conference Room, Bldg. I.  
 10:30 - 11:50 Surgical-Pathological Conference; Lyle Hay and E. T. Bell.  
 10:30 - Surgery Tumor Conference; Conference Room, Bldg. I.  
 1:00 - Chest Surgery Conference; J. Kinsella and Wm. Tucker; Conference Room, Bldg. I.  
 1:30 - Liver Rounds; Samuel Nesbitt.

Tuesday, April 10 (Cont.)Veterans Administration Hospital (Cont.)

- 2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III.
- 3:30 - 4:20 Clinical Pathological Conference; Conference Room, Bldg. I.

Wednesday, April 11Medical School and University Hospitals

- 8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-109, U. H.
- 8:00 - 9:00 Roentgenology-Surgical-Pathological Conference; Allen Judd and L. G. Rigler; Todd Amphitheater, U. H.
- 11:00 - 12:00 Pathology-Medicine-Surgery Conference; Medicine Case; O. H. Wangensteen, C. J. Watson and Staffs; Todd Amphitheater, U. H.
- 4:00 - 6:00 Ophthalmology Seminar; Todd Room, 5th Floor, U. H.
- 5:00 - 5:50 Urology-Pathological Conference; C. D. Creevy and Staff; Eustis Amphitheater.
- 5:00 - 7:00 Dermatology Clinical Seminar; Dining Room, U. H.
- 7:00 - 8:00 Dermatology Journal Club; Dining Room, U. H.
- 8:00 - 10:00 Dermatological Pathology Conference; Review of Histopathology Section; Robert Goltz; Todd Amphitheater, U. H.

Ancker Hospital

- 8:30 - 9:30 Clinico-Pathological Conference; Auditorium.
- 3:30 - 4:30 Journal Club; Surgery Office.

Minneapolis General Hospital

- 8:30 - Pediatric Rounds; Dr. Lowry; 7th Floor Annex.
- 9:00 - Pediatric Allergy Rounds; Dr. Nelson; 4th Floor Annex.
- 11:00 - 12:00 Pediatric Rounds; Franklin Top; 7th Floor Annex.
- 12:15 - Staff Meeting; 4th Floor Annex.
- 1:30 - Pediatric Rounds; Dr. Huenekens and Dr. Ulstrom; 5th Floor Annex.

Veterans Administration Hospital

- 8:30 - 10:00 Orthopedic-Roentgenologic Conference; Edward T. Evans and Bernard O'Loughlin; Conference Room, Bldg. I.

Wednesday, April 11 (Cont.)Veterans Administration Hospital (Cont.)

- 8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker.  
 7:00 p.m. Lectures in Basic Science of Orthopedics; Conference Room, Bldg. I.

Thursday, April 12Medical School and University Hospitals

- 9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; M-109 U. H.  
 10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.  
 11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Todd Amphitheater, U. H.  
 12:00 - Physiological Chemistry Seminar; Mechanism of Action of Vitamin E; Oleg Jardetsky; 214 Millard Hall.  
 4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hansen and Staff; E-534, U. H.  
 5:00 - 6:00 Radiology Seminar; Vascular Lesions in the Lungs; L. G. Rigler; Eustis Amphitheater, U. H.  
 7:30 - 9:30 Pediatrics Cardiology Conference and Journal Club; Review of Current Literature 1st hour and Review of Patients 2nd hour; 206 Temporary West Hospital.

Minneapolis General Hospital

- 8:30 - Neurology Rounds; Dr. Heilig; 4th Floor Annex.  
 11:30 - Pathology Conference; Main Classroom.  
 1:00 - 2:00 EKG and X-ray Conference; Classroom, 4th Floor Annex.  
 2:00 - Psychiatry Rounds; Dr. Benton; 4th Floor Annex.  
 2:00 - 4:00 Infectious Disease Rounds; 8th Floor.  
 4:00 - 5:00 Infectious Disease Conference; Classroom, 8th Floor.

Veterans Administration Hospital

- 8:00 - Surgery Ward Rounds; Lyle Hay and Staff.  
 9:15 - Surgery Grand Rounds; Conference Room, Bldg. I.  
 11:00 - Surgery-Roentgen Conference; Conference Room, Bldg. I.  
 1:00 - Chest Rounds; William Stead.

Friday, April 13

Medical School and University Hospitals

- 8:30 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.
- 9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.
- 10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.
- 10:30 - 11:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.
- 11:45 - 12:50 University of Minnesota Hospitals Staff Meeting; The Treatment of Lymphosarcoma; Charles Nice; Powell Hall Amphitheater.
- 1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.
- 2:00 - 3:00 Dermatology and Syphilology Conference; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U. H.
- 3:00 - 4:00 Neuropathological Conference; F. Tichy; Todd Amphitheater, U. H.
- 4:00 - 5:00 Clinical Pathological Conference; A. B. Baker; Todd Amphitheater, U.H.
- 4:00 - 5:00 Dermatology Seminar; W-312, U. H.
- 4:15 - 5:15 Electrocardiographic Conference; 106 Temporary Bldg., Hospital Court, U. H.
- 5:00 - Urology Seminar; Osterstitis Pubis; Robert Wood; Eustis Amphitheater, U. H.

Ancker Hospital

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

Minneapolis General Hospital

- 8:30 - Pediatric Rounds; Dr. Lowry; 7th Floor Annex.
- 10:00 - Pediatric Rounds; Franklin Top; 7th Floor Annex.
- 1:30 - Pediatric Rounds; Dr. Ulstrom; 5th Floor Annex.

Veterans Administration Hospital

- 10:30 - 11:20 Medicine Grand Rounds; Conference Room, Bldg. I.
- 1:00 - Microscopic-Pathology Conference; E. T. Bell; Conference Room, Bldg. I.
- 1:30 - Chest Conference; Wm. Tucker and J. A. Myers; Ward 62, Day Room.

Friday, April 13 (Cont.)Veterans Administration Hospital (Cont.)

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

Saturday, April 14Medical School and University Hospitals

- 7:45 - 8:50 Orthopedic X-ray Conference; Wallace H. Cole and Staff; M-109, U. H.
- 9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; E-221, U. H.
- 9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater, U. H.
- 9:15 - 10:00 Surgery-Roentgenology Conference; J. Friedman, O. H. Wangensteen and Staff; Todd Amphitheater, U. H.
- 10:00 - 11:30 Surgery Conference; O. H. Wangensteen and Staff; Todd Amphitheater, U. H.
- 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; Vital Staining of Fibrous Tissue; W. Lane Williams; Effect of Peripheral Innervation Area on Nerves; Marge Cavanaugh; 226 Institute of Anatomy.

Ancker Hospital

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

Minneapolis General Hospital

11:00 - 12:00 Pediatric Clinic; Dr. Thomas and Dr. Good; Classroom, 7th Floor Annex.

Veterans Administration Hospital

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

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