Bulletin of the

University of Minnesota Hospitals
and
Minnesota Medical Foundation

Sympathetic Ophthalmia

Volume XXII | Friday, May 2, 1952 | Number 25

BULLETIN OF THE
UNIVERSITY OF MINNESOTA HOSPITALS
and
MINNESOTA MEDICAL FOUNDATION

Volume XXIII | Friday, May 2, 1952 | Number 25

INDEX

		PAGE
I.	SYMPATHETIC OPHTHALMIA	477 - 486
	ERNEST S. PALMERTON, M. D., Medical Fellow, Division of Ophthalmology, University of Minnesota Medical School	
II.	MEDICAL SCHOOL NEWS	487
III.	WEEKLY CALENDAR OF EVENTS	488 - 493

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

The Bulletin is sent to members of the Minnesota Medical Foundation. Annual membership fee - $10.00.

Address communications to: Staff Bulletin, 3330 Powell Hall, University of Minnesota, Minneapolis 14, Minn.

I. SYMPATHETIC OPHTHALMIA

Ernest S. Palmerton, M.D.

Introduction

In the course of conversations with members of the intern and resident staff of such services as pediatrics, surgery, and medicine, there have been frequent questions raised about sympathetic ophthalmia. Questions like: What is the mechanism of extension from one eye to the other in this disease? How long can one safely wait before removing an injured eye? How frequent is sympathetic ophthalmia? Why do the people in ophthalmology worry more about some injuries to eyes, than about others? Is recovery with useful vision possible in sympathetic inflammation, or is the process always disastrous?

Because of questions like these, also in view of recent changes in the treatment, namely the use of antibiotics and even more lately, of cortisone and ACTH, with considerably improved outlook, our department felt that it was fitting that a general discussion of this problem be given today.

Definition

First I want to give a rather all-embracing statement defining sympathetic ophthalmia or ophthalmitis. This is from Duke-Elder[1] and a thorough consideration of this definition affords considerable knowledge about the condition. Sympathetic ophthalmia is a specific, bilateral inflammation of the entire uveal tract whose etiology is unknown - characterized clinically by insidious onset and a progressive course with exacerbations and usually a disastrous termination, and pathologically by a nodular or diffuse infiltration of the uveal tract with lymphocytes and epithelioid cells, which condition almost invariably follows a perforating wound involving uveal tissue.

Historical

As early as 1000 A.D. Constantius Cephalis quoted from Agathias: "The right eye when diseased often gives its suffering to the left."[2] In the beginning of the 19th century it was common to call spontaneous ocular inflammations beginning in one eye "idiopathic", and "sympathetic", when they occurred in the other eye from that in which the disease or injury first took place. Without question many cases were called sympathetic ophthalmia that actually were not, and to a lesser extent that is doubtless true today - as a pathologic diagnosis is the only way to be positive of this disease. Mackenzie in 1840 published a Treatise on Diseases of the Eye, with a chapter on sympathetic ophthalmia. In 1851 Pritchard[3] first proposed enucleation of the exciting eye to save the sympathizing eye. In 1863 Critchett advised early enucleation of a badly wounded eye as a prophylactic measure in saving the other eye, and stressed that after sympathetic inflammation was begun it was often useless to remove the exciting eye. It was in 1905 that Fuchs[4] made a comprehensive pathologic study of this condition, using a series of 200 cases, and his criteria, to be given later in this paper, are still accepted. There have been numerous studies in the past three decades, among which may be mentioned those of Woods[2], Theobald[5], and Trowbridge[6].

Incidence

This inflammation is rather rare[2]. Reports vary from 0.1% to 0.15% of all eye diseases. It is better to consider the likelihood of occurrence in cases with penetrating wounds of the eye. Under these circumstances the incidence is reported to vary from 0.54% to 5.0%. The disease appears to be getting less frequent as better care is being taken of injured eyes, and as enucleation of potentially dangerous eyes is more generally practiced. It is more common in males, probably because there are more male patients who receive eye injuries. From the standpoint of age there is no definite predilection. The ages from childhood to young adult life show more of these cases than the older age brackets again because of the injury factor.

For some reason sympathetic ophthalmia is more common in the winter months.

Predisposing Causes

Perforating injuries precede about 65% of cases[1, 2, 7]. It has been shown repeatedly that wounds involving the iris and ciliary body so that there is incarceration of these structures, are particularly to be feared. However such involvement is not necessary for sympathetic uveitis to follow. Wounds with foreign bodies retained in the eye, and those that heal poorly, are considered especially dangerous.

While the foregoing refers to accidental injuries, operative wounds, particularly cataract extractions, precede about 25% of cases. Cataract operations account for about half of this 25%. Besides cataract extractions, there are reports of cases after iridectomy, sclerectomy, iris inclusions, needling operations, corneal tattooing, and evisceration of an eye. Intraocular surgery complicated by iris prolapse or incarceration of lens capsule has led repeatedly to this complication.

The remaining ten per cent of cases follow non-perforating wounds, injuries with subconjunctival rupture, contusions, perforating corneal ulcers and perforating malignant tumors of the eye. Many injuries, considered at first to be non-perforating, that were followed by sympathetic ophthalmia, have been later found on careful pathological examination to show some minute break in the globe.[1]

An eye which undergoes frank suppuration after an injury is rarely followed by sympathetic disease. For this reason, in the pre-Listerian days steps were sometimes taken to insure suppurative reaction in a blind, injured eye. It is the eye which smolders, staying red and irritated, flaring up so as to be worse at intervals, and usually very soft to palpation, which makes the ophthalmologist apprehensive. And it is not necessarily a blind eye.

Interval between injury and sympathetic involvement

The least time to be reported between initial injury and the onset of sympathetic disease is nine days[2]. The usual working minimum is given as fourteen days. Cases of sympathetic inflammation have followed the initial insult after a delay as long as 20 years, and there is one case on record of an interval of 42 years (Schirmer). These long delayed cases have usually occurred when the patient has kept a useless, shrunken eye which has remained fairly quiet until caused to flare up by some such event as a slight second injury.

Sixty-five per cent of cases begin within two months after injury to the exciting eye; ninety per cent occur before one year. As the time interval lengthens it is of course more and more difficult to be sure whether the inflammation is related to the original event, or to some episode of more recent date.

Etiology

The literature on this aspect of sympathetic ophthalmia is very large.

Mackenzie in 1840 had a theory that unhealthy nervous impulses affected the sympathizing eye and in time caused damage.

Although an organism has not yet been isolated, most ophthalmologists[1, 5, 8] are convinced that infection plays an essential role in the picture of sympathetic uveitis. A variety of organisms have been suspected, particularly the tubercle bacillus, a virus, or a rickettsia-like organism.

The pathology of this disease resembles in some ways that of tuberculosis. Because of this resemblance, Guillery[9] attempted to reproduce the disease in rabbits by implanting into the eyes semi-permeable capsules containing living tubercle bacilli. After an interval uveal irritation followed, first in the eyes containing the capsules, later in the unimplanted eyes. Guillery concluded that the inflammation of sympa-

thetic ophthalmia was very likely tuberculotoxic in origin. Kolen[10] repeated this work with similar results, then varied the experiments by using capsules containing Staphylococci, and did not get uveal irritation.

In 1924 von Szily[11] inoculated the eyes of rabbits with material taken from herpetic lesions on human eyes. A herpetic keratitis followed. At this stage the superficial layers of the corneas were removed and injected into other rabbit eyes near the ciliary bodies. Uveitis followed, and in 10% of these animals uveitis later developed in the fellow eyes that were untouched. Histologic examination moreover showed the nerve heads and the optic nerves of these inflamed eyes to have infiltrations of lymphocytes and epithelioid cells. This work was repeated by others, among them Gifford and Lucic[11]. In their sections these workers were able clearly to demonstrate inflammatory changes (Infiltrations of lymphocytes and epithelioid cells) suggesting extension of the process along the optic nerve of the eye first infected, then the chiasm and the second optic nerve to the fellow eye.

However no one was able to take material from the eyes of human cases of sympathetic ophthalmia and inoculate rabbit eyes, and get uveitis in the rabbits.

An interesting clinical report was given by von Bahr[12] who described the cases of three children who suffered eye trauma and concurrently had chickenpox, and who developed sympathetic ophthalmia.

The matter of hypersensitivity has been discussed since 1909, when Elschnig [13] raised the question of allergy in sympathetic disease. Elschnig believed that an injury to the exciting eye caused absorption of uveal tissue into the general circulation, with hypersensitivity developing particularly in the fellow eye. Continued absorption caused an allergic intoxication, and in the sensitized uveal tissue of the second eye this became sympathetic ophthalmia.

In the United States Dr. Alan C. Woods of the Wilmer Eye Institute has been working along lines indicated by this theory since the days of World War I.[14]. Woods succeeded in sensitizing dogs intraocularly with canine uveal pigment. Then, on producing an intoxication by intraperitoneal injection of pigment, he got, in a small percentage of his animals, not only uveal irritation in the sensitized eye, but also in the fellow eye. But this disturbance histologically did not closely resemble sympathetic inflammation.

Woods further tested the blood serum of patients having had penetrating wounds of the eyes. He tested for complement fixation with an antigen of uveal pigment. Those patients whose eyes healed normally had a positive complement fixation reaction while those with prolonged iridocyclitis or sympathetic ophthalmia did not show this reaction. This seemed to indicate a definite immune reaction in some patients - which was lacking in those patients whose eyes did not do well.

Dr. Woods then made an extract of uveal pigment and used a suspension of it for an intradermal test. Such tests were negative in normal people but frequently were positive - indicating hypersensitivity - in a series of patients with sympathetic ophthalmia. Friedenwald[15] studied a number of biopsies of the sites of these intradermal tests; he reported that in those with positive skin tests there was a pathologic picture hard to differentiate from the pathology found in sympathetic ophthalmia itself.

Going a step farther, Woods treated patients with sympathetic ophthalmia by giving desensitizing doses of uveal pigment, with spectacular improvement in many, but not all, of his cases. His conclusions about the role of hypersensitivity to uveal pigment, as it relates to this disease, have always been expressed with much reserve and his final opinion is that another factor, probably infective, is present and essential for

the process to lead to sympathetic uveitis.

If infective, how does it get to the other eye? Blood stream invasion with localization in the sympathizing eye has been suspected, never proved. A stronger possibility is the extension of the infectious agent along the optic nerve, across the chiasm and back along the other optic nerve to the fellow eye. There are four cases on record[1, 16] in which the optic nerves and chiasms of patients who had sympathetic ophthalmia and who died of intercurrent disease, have been obtained and examined. Duke-Elder and Schreck both cite the case reported by Deutschmann, in which round cell infiltration of both optic nerves and of the chiasm were found - the brain and meninges being spared. The case reported by Schreck showed a continuous migration of the inflammatory process from one eye to the other via the chiasm.

I want to bring this part of the discussion to a close by telling more of the work of Dr. Eugen Schreck, very recently published and not yet confirmed by other workers.[16, 17, 18]

Schreck describes the migration of the sympathetic process as follows: in some cases the exciting eye shows irritation of the lens, iris and ciliary body, and in these the inflammation extends to the other eye by way of the retina and optic nerve as a migrating perivasculitis. In the sympathizing eye this develops into a perivascultitis of the retina and an anterior uveitis. In other cases both the exciting and sympathizing eyes show anterior and posterior uveitis, connected by a migrating perivasculitis along the posterior ciliary vessels and along the vessels in the optic nerves.

Furthermore this worker reports successful transmission of the agent of sympathetic ophthalmia from the eyes of humans suffering with the disease, to the eyes of chickens. He has repeatedly been able to inoculate one eye of a chicken with such material, and to have uveitis develop in this eye within a few weeks and then be followed by sympathetic uveitis in the other, untouched, eye. Schreck stressed that it was very important not only to use sterile technique in handling the material for inoculation, but also to keep this material at body temperature at all times. Not only did he succeed in the inoculations of the human material into the eyes of chickens, but he has been able to pass the disease along through as many as five generations of chickens - using material from either inoculated eyes or from sympathizing eyes. It was observed that with successive passages the infective agent appeared to become more and more virulent, since his chickens died more and more frequently with succeeding passages.

Schreck also succeeded in cultivating the agent in the chorio-allantoic membrane of chick embryos, and then in passing it back to the eyes of chickens, again to produce sympathetic uveitis. He found that rabbits, pigeons and guinea pigs were not susceptible. In his sections were found the histological characteristics of sympathetic ophthalmia, and what appeared to be rickettsia-like organisms.

From these and other findings Schreck has made rather sweeping conclusions: that the organisms responsible for sympathetic disease are in the conjunctival sac, harmless and saprophytic under ordinary conditions. They are also in the epithelial cells of the conjunctive, and in the adventitial cells of the conjunctival and scleral vessels and in the vessels of the limbal arcades. They are found in Tenon's fascia. (This would explain the occasional occurrence of sympathetic infection in those injuries of eyes with subconjunctival rupture.)

One must repeat that this work is unconfirmed but if it can be established much of the mystery about sympathetic ophthalmia will have been removed.

Clinical Course

If there is such a thing as a typical

case the story will be on this order: One eye receives a penetrating wound involving the ciliary region, probably with iris prolapse. After repair and closure the eye remains irritable and there are exacerbations with redness and photophobia. Examination shows an iritis. After a variable period the iris begins to look thickened and muddy, with nodules on the surface and on the pupillary margin. The lens capsule begins to look cloudy. In time, with more flareups, the eye becomes soft and sightless and eventually arrives at the condition known as phthisis. The picture of uveal involvement is not always as marked as this, and the injured eye by no means always degenerates into a blind organ.

After an interval the fellow eye begins to show signs of irritation. There is sensitivity to light and inspection shows circumcorneal injection. The patient may have accommodative failure and complain of not being able to focus his vision for near. The eye may not be very painful but will be tender on pressure.

This involvement of the sympathizing eye may begin as a plastic iritis principally, with large greasy looking keratic precipitates and a felting over of the lens from the pupillary borders of the iris. Examination with the slit lamp in early cases, well before the keratic deposits and the felting, will regularly show cells in the aqueous and in the retrolental space.

In other cases the sympathizing eye first shows the trouble in the posterior segment with retinal edema and injection of the disc of the optic nerve. The fundus will show whitish spots peripherally, and before long the vitreous will become so cloudy that fundus detail cannot be made out.

An occasional case will run a mild, short course, with relatively little damage to the sympathizing eye. However most cases get permanent, often exceedingly severe damage, the eye first becoming hard with secondary glaucoma, then at last, as the mechanism for secreting aqueous fluid breaks down, soft and phthisical. There are usually periods of relative quiet, interspersed with flareups. The average duration of a sympathetic process is nine to twelve months but many times a case will stretch out for years, due to the repeated flareups.

Consideration of this grim chain of events leads one to understand why, if the exciting eye has any useful vision and the sympathetic inflammation is well underway in its fellow - it is felt best not to enucleate the exciting eye, as it may in the end have more vision than its mate.

Sympathetic ophthalmia and sympathetic irritation are to be carefully differentiated. In the latter there are photophobia and lacrimation and conjunctival redness but the pupil reacts well and there are no cells and no keratic precipitates.

Pathology[8, 19, 20]

The histologic picture of sympathetic ophthalmia will be the same in the two eyes; however there may be added changes in the exciting eye because of concomitant infection of some other type being present in the eye at the same time.

Fuchs[4] furnished the classical description in 1905 and the essential findings are as follows:

1. A tendency to general uniform infiltration of the entire uveal tract and involvement of the posterior layers of the iris.

2. Early infiltration along the walls of veins.

3. Early destruction of pigment epithelium and formation of Dalen-Fuchs nodules.

4. Absence of tissue destruction.

The inflammatory picture is always

most intense in the choroid, or uveal layer, of the globe. There is massive round cell infiltration. At first these cells are mostly lymphocytes, then epithelioid cells will appear and later giant cells form. Nodules appear, resembling the nodules of tuberculosis except for the absence of caseation. In the choroid however the nodule formation is overshadowed by the marked generalized thickening. Lymphocytes will be seen packed around the veins of the choroid. In the iris the infiltration tends to begin in the posterior layers. Nodular masses form which will break through the posterior epithelium of the iris and spread over the anterior capsule of the lens. In the case of the ciliary body the posterior part becomes involved first, later the entire structure is infiltrated. But the choroid proper will repeatedly get the examiner's attention because of its marked change. It may become three or four times its normal thickness. The choriocapillaris is relatively free of infiltration until late and the retina likewise escapes early infiltration, with the exception of the pigment epithelium.

A distinctive feature (which is not however pathognomonic of sympathetic disease) is the reaction of the pigment epithelium on the inner surfaces of the iris, ciliary body and choroid. Here and there (especially on the ciliary body) isolated groups of cells will be seen becoming spindleshaped, eventually undergo autolysis, and the freed pigment is phagocytosed by the lymphocytes and epithelioid cells that collect there. This is the Dalen-Fuchs nodule. All through the choroid phagocytosis of pigment by the epithelioid cells and giant cells will be seen.

In some eyes there will be infiltration behind the lamina cribrosa. Nodular formations have been seen along both optic nerves and the chiasm. There may even be the same type of infiltration subconjunctivally and into the extraocular muscles, the process having extended into these areas along the emissary veins of the eye.

Diagnosis

Clinically, the history of trauma is of course of great importance. This may be accidental or surgical trauma. The findings in early cases depend in large measure on what can be seen with the slit lamp and ophthalmoscope. The posterior form will show subretinal edema at an early stage, followed by small peripheral exudates. But usually it is the anterior form which is seen, with cells floating in the aqueous of the anterior chamber, in the retrolental space, and suspended in the vitreous. Keratic precipitates will soon be showing up on the back of the cornea, first granular and small, soon larger and waxy in appearance. Small patches of exudate will be seen at the pupillary margins of the iris. The eye will usually be soft, but not always, as in some secondary glaucoma comes on early.

There is not much systemic reaction. The differential blood count will usually show an increase in monocytic cells. The complement fixation test developed by Woods is not generally available due to the difficulty in keeping a stable antigen. The intra-dermal testing of the patient for uveal pigment sensitivity is likewise not in general use. Some authorities feel it is not dependable[21] and the pigment suspension is hard to prepare and to keep in stable form.

The pathologic diagnosis rests on the points given by Fuchs, and these have been given. With regard to differential diagnosis of this condition the following may be mentioned:

1. Chronic traumatic uveitis. The differentail points are that there will not be a sympathizing eye, and the thickening of the uveal tract will not be as marked. There will be less tendency to form a membrane over the anterior lens capsule. Theobald[5] stresses the lack of eipthelioid cells in this condition.

2. Infective endophthalmitis - in this condition the reaction will be

less insidious, and the interior structures of the eye - that is - retina and vitreous rather than the choroid, are involved.

3. Tuberculosis of the eye - the reaction tends to be less uniform, more nodular. The anterior rather than the posterior layers of the iris will be affected, and the anterior chamber is often invaded. Pathologically the retina is invaded early, and caseation in the nodules will be seen. The macula however is often spared. Phagocytosis of pigment is not so regularly seen as in sympathetic ophthalmia. The tendency for the lymphocytes to infiltrate in the region of the emissary veins, as seen in sympathetic inflammation, is not a feature of tuberculosis of the eye.

4. Syphilis - the "great mimic" will cause various types of lesions in the choroid. The retina is always involved. Often large patches of chorioretinitis will be seen due to endovasculitis of the larger choroidal vessels. In other cases small black pigmented spots are seen, these give rise to the term pepper and salt fundus - said to be characteristic of congenital syphilis. In these cases the choriocapillaris is damaged early.

5. Sarcoid. Fifty per cent of ocular sarcoidosis consists of uveal lesions. Often these are cases of anterior uveitis. The ciliary congestion is low grade, and the nodules seen in the iris look superficial and have a pinkish color and are rather vascular. In the choroid itself sarcoidosis results in small, deeply placed yellowish nodules, without a surrounding zone of inflammation. The course of the disease is rather slow and indolent. Diagnosis will be aided if enlarged lymph nodes can be found for biopsy. Pathologically sarcoid consists of nodular collections of epithelioid cells with relatively few lymphocytes. Giant cells are frequent and some may be found containing Schaumann bodies. Caseation is absent.

Prognosis

Sympathetic ophthalmia has been spoken of as a progressive disease usually with disastrous outcome. It is then a surprize to find the literature of even many years ago as optimistic as it is. In general, a fairly good outcome with retention of useful vision in 50% of cases has been reported, and this includes advanced and neglected cases. In series where only early cases are considered, reasonably good outcome in as high as 66% has been reported. With the advent of the antibiotics, especially the newer ones, and cortisone and ACTH, no series of any size has been reported and one can only make a guess as to what the proportion of good results would be.

Prophylaxis and Treatment

High in importance among preventive measures the eye man puts prompt enucleation, not evisceration, of badly injured eyes. This may be modified by such circumstances as:

1. Uneventful recovery without undue inflammation of the eye during the period of grace, which time is usually given as ten to fourteen days. During this time the fellow eye is closely watched, and Parsons stresses the importance of keratic deposits appearing in the fellow eye. An even earlier sign is the appearance of cells floating in the anterior chamber and retrolental space. If such danger signs do appear the ophthalmologist must quickly decide whether or not enucleation of the exciting eye should be done.

2. Another modifying circumstance would be a case wherein the fellow eye is already of little or no value because of some different disorder.

Further prophylaxis lies in more vigilant diagnosis of intra-ocular foreign bodies and prompt removal of same if possible.

Among preventive measures should be mentioned refraining from surgery on blind eyes, especially glaucomatous ones - simply because the patient wants to

avoid their removal.[22] It is truly a double tragedy when sympathetic uveitis follows in the fellow eye.

The use of antibiotics. If, as Schreck believes, the organism responsible in this disease is a rickettsia-like one, the use of terramycin or chloramphenicol is suggested, preference being given for chloramphenicol since this agent, when given systemically, has been found to penetrate the globe better than other antibiotics[23, 24, 25].

Prophylactic foreign protein therapy, usually in the form of injections of triple typhoid vaccine, or of typhoid H antigen, by vein, has long been considered by ophthalmologists to be of great value. It is now known that foreign protein therapy works, at least in part, by altering the function of the adrenal gland in a manner similar to ACTH,[26] and the result in the eye is a blocking of the inflammatory and exudative phases of certain eye conditions.

Many ophthalmologists are reluctant however to abandon the use of foreign protein therapy injections in favor of cortisone or ACTH, as they feel that other, as yet unknown protective effects are possibly elicited, that ACTH and cortisone may not give.

Treatment of sympathetic ophthalmia in early cases consists first of all in removal of the exciting eye - particularly if it be sightless. If the case is one with considerable findings already in the sympathizing eye, and if the exciting eye retains any vision, the exciting eye is usually not removed. Damage to the sympathizing eye is hard to control, but it can be reduced to some extent by the full use of cycloplegics to keep the pupil fully dilated, thus getting the lens and the iris separated and preventing posterior synechiae. If secondary glaucoma occurs repeated paracenteses may be done but they are usually fruitless.

The full use of salicylates has a time honored place in treatment of these cases. Here again it may very well be that the benefit comes from the ACTH-like effect of these drugs.

Antibiotics are indicated for the same reasons they are useful in prophylaxis of the disease.

In the past eighteen months cortisone and ACTH have been used in treatment of sympathetic ophthalmia with encouraging results[27, 28, 29]. It has been stated in this paper that the disease is a progressive condition. However Woods regards it as a self-limited process which runs its course and burns out - in which case the prevention of damage to the eyes while the disease runs its course, is clinically equivalent to a cure.

It is well to recall the good results that were obtained prior to cortisone and ACTH and to withhold judgment until more time has passed. There are times when the use of cortisone or ACTH is most valuable: when the clinical course of the disease indicates that prolonged treatment will be necessary, and when patients rapidly become refractory to foreign protein injections, or are in such poor physical condition that the rigors of intravenous typhoid injections might be hazardous to life. The ophthalmologist must bear in mind that full treatment with either one of these hormones has its dangers also.

Since this is bilateral eye disease with posterior uveal tract involvement, systemic dosage rather than the local administration of cortisone drops, is necessary. The use of the cortisone suspension locally, in a strength of five to eight milligrams per cc, and given hourly, is of added use in preventing iris and lens damage, and is of further value in maintenance therapy after the patient leaves the hospital.

Summary

1. A historical background, and the clinical and pathological features of sympathetic ophthalmia, one of the granulomatous diseases to affect the eyes, are given.

2. A discussion is given of the recent work of Schreck, who has been able for the first time to transmit sympathetic uveitis from human cases to experimental animals, also to culture the infective agent in chick embryos and then reproduce the disease in other experimental subjects.

3. Prevention and treatment of sympathetic ophthalmia are reviewed with special mention of the use of cortisone, ACTH, and antibiotics.

References.

1. Duke-Elder, Sir S.
Textbook of Ophthalmology.
Vol. III, P. 2324. Mosby, 1940.

2. Woods, A. C.
Sympathetic Ophthalmia (I)
Am. J. Ophth. 19: 9, 1936.

3. Pritchard cited by Alvaro, M. E. in berens
The Eye and Its Diseases
Saunders, p. 597, 1950.

4. Fuchs, E.
Über sympathisierende Entzündung (nebst Bemerkungen über seröse traumatische Iritis)
Arch f. Ophth. 61: 365, 1905.

5. Theobald, G. D.
Frequency of Sympathetic Ophthalmia
Am. J. Ophth. 13: 597, 1930.

6. Trowbridge, D. H.
Sympathetic Ophthalmia. A Study of Some Clinical and Pathological Factors in 33 Cases.
Am. J. Ophth. 20: 135, 1937.

7. Parsons, Sir J. H. and Duke-Elder, Sir S.
Diseases of the Eye
11th Ed. p. 464. The MacMillan Co. 1948.

8. Wolff, E.
A Pathology of the Eye.
2nd Ed. Blakiston. 1948.

9. Guillery, H.
Die Sympathische Ophthalmie eine Tuberculotoxische Erkrankung.
Munchener Med. Wchnschr. 72: 298, 1925.

10. Kolen cited by Woods, A. C. and Little M. F.
Uveal Pigment - Hypersensitivity and Therapeusis.
A.M.A. Arch, Ophth. 9: 200, 1933.

11. von Szily cited by Gifford, S. R. and Lucic, L. H.
Sympathetic Uveitis Caused by the Virus of Herpes Simplex.
J.A.M.A. 88: 465, 1927.

12. von Bahr, G.
Is There a Connection Between Herpes Zoster, Varicella and Sympathetic Ophthalmia?
Acta Pediatrica 25: 1, 1939.

13. Elschnig, A.
Arch. f. Ophth. 75: 459, 1910.
Arch. f. Ophth. 76: 509, 1910.

14. Woods, A. C.
Sympathetic Ophthalmia; The Use of Uveal Pigment in Diagnosis and Treatment.
Tr. Ophth. Soc. U. Kingdom 45: 208, 1925.

15. Friedenwald, J. S.
Notes on the Allergy Theory of Sympathetic Ophthalmia.
Am. J. Ophth. 17: 1008, 1934.

16. Schreck, E.
Weitere Beitrage zur Frage der Klinik, Microbiologie und Pathologischen Anatomie der Sympathischen Ophthalmie.
Arch f. Ophth. 149: 656, 1949.

17. Schreck, E.
Uber Wesen and Migrations weg der Sympathischen Ophthalmie.
Arch. f. Ophth. 148: 361, 1948

18. Schreck, E.
The Microorganisms Causing Sympathetic Ophthalmia.
A.M.A. Arch. Ophth. 46: 489, 1951.

19. Woods, A. C.
Sympathetic Ophthalmia (II)
Am. J. Ophth. 19: 100, 1936.

20. DeCoursey, E. and Ash, J. E.
Atlas of Ophthalmic Pathology.
American Academy of Ophthalmology and Otolaryngology. 1942.

21. Gifford, S. R. and Lucic, L. H.
The Etiology of Sympathetic Ophthalmia.
A.M.A. Arch. Ophth. 1: 468, 1929.

22. Simmonds, N. T.
Sympathetic Ophthalmia.
Am. J. Ophth. 34: 1601, 1951.

23. Leopold, I. H., Nichols, A. C., and Vogel, A. W.
Penetration of Chloramphenicol U.S.P. (Chloromycetin(R)) into the Eye.
A.M.A. Arch. Ophth. 44: 22, 1950.

24. Dunphy, E. B.
Ocular Therapeutic Principles and Practical Applications.
A.M.A. Arch. Ophth. 44: 797, 1950.

25. Benkwith, K. B.
Sympathetic Ophthalmia (Correspondence) A.M.A. Arch Ophth. 47: 268, 1952.

26. Arendshorst, W. and Falls, H. F.
Role of the Adrenal Cortex in Treatment of Ocular Diseases with Pyrogenic Substances.
A.M.A. Arch. Ophth. 44: 635, 1950.

27. Leopold, I. H., Purnell, J. E., Cannon, J. E., Steinmetz, C. G., and McDonald, P. R.
Cortisone in Ocular Disease.
Am. J. Ophth. 34: 361, 1951.

28. Woods, A. C.
Present Status of ACTH and Cortisone
Am. J. Opth. 34: 945, 1951.

29. Harvey, A. M. and Kattus, A. A.
Effect of Adrenocorticotropic Hormone (ACTH) and Cortisone on Certain Diseases of the Eye in which Hypersensitivity Plays a Role.
Bull. Johns Hopkins Hosp. 87: 461, 1950.

II. MEDICAL SCHOOL NEWS

Coming Events

May 12-14	Continuation Course in Eye, Ear, Nose, and Throat for General Physicians
May 15-17	Continuation Course in Allergy and Hematology for General Physicians
May 20	Minnesota Pathological Society Meeting; "Crime and the Doctor," Dr. C. Keith Simpson, Reader in Forensic Medicine, Supervisor of Medico-Legal Post-Mortems, and Home Office Pathologist, Guy's Hospital, University of London, London, England; Owre Amphitheater; 8:00 p.m.
June 23-28	Continuation Course in Otolaryngology for Specialists

* * *

Continuation Course in EENT

The University of Minnesota will present a continuation course in Eye, Ear, Nose, and Throat for General Physicians at the Center for Continuation Study on May 12-14, 1952. This course will be of interest primarily to physicians engaged in general practice. Common disorders of the eye, ear, nose, and throat will be stressed, and specific therapeutic measures will be discussed in detail. Dr. Erling W. Hansen, Clinical Professor and Director, Division of Ophthalmology, and Dr. George M. Tangen, Clinical Assistant Professor, Division of Otolaryngology, have joined in planning the program. An outstanding feature will be the presence on the program of Dean Diehl who will discuss the common cold.

* * *

Faculty News

On Saturday, April 19, several members of the Departments of Surgery and Pathology participated in a panel discussion of cancer which was presented on a local television station. Dr. George E. Moore, Assistant Professor of Surgery, acted as moderator, whole the panel included Doctors Claude R. Hitchcock, Robert A. Huseby, T. Brannon Hubbard, and Norman H. Jacob. They discussed the questions which are most commonly asked by people about cancer.

Dr. George E. Moore and Dr. Joseph T. King, Associate Professor of Physiology, attended the M. D. Anderson Symposium on Cancer Research in Houston, Texas, on April 25 and 26. Dr. King presented a paper entitled, "Diets and Incidence of Tumors in Mice." Prior to this meeting, Dr. Moore discussed, "The Cancer Research Program at the University of Minnesota," at Louisiana State University on April 23.

Dr. Irvine McQuarrie, Professor and Head, Department of Pediatrics, delivered the Annual Renziehausen Foundation Lecture at the University of Pittsburgh on April 15. His subject was, "Hypoglycemia -- Clinical and Experimental."

The Third Annual ACTH Conference was held at the Drake Hotel in Chicago on April 25 and 26. The University of Minnesota was represented by Dr. C. Walton Lillehei and Dr. Bernard Zimmermann, of the Department of Surgery. Dr. Lillehei discussed experimental studies on the use of ACTH for protection against liver anoxia, the use of ACTH in the prevention of glomerulonephritis in dogs, and the use of pituitary growth hormore in the treatment of peptic ulcer. Dr. Zimmermann discussed the role of ACTH in the preparation of patients for surgery and its use in treatment of shock.

III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

May 5 - 10, 1952

Monday, May 5

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 - Physical Medicine Seminar; Abnormalities of the Temporomandibular Joint; N. O. Holte; Eustis Amphitheater, U. H.

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

12:30 - Physiology Seminar; Discussion of Aging; M. B. Visscher, and A. J. Carlson, Department of Physiology, University of Chicago; 214 Millard Hall.

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

4:00 - Seminar on Fluid and Electrolyte Balance; Todd Amphitheater, U. H.

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

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

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

Minneapolis General Hospital

7:30 - Fracture Grand Rounds; Dr. Zierold; Sta. A.

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

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

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

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

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

Ancker Hospital

8:30 - 10:00 Chest Disease Conference

1:00 - 2:00 Medical Grand Rounds.

Monday, May 5 (Cont.)

Veterans Administration Hospital

8:00 - 9:00 Neuroradiology Conference; B. J. O'Loughlin, 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; B. J. O'Loughlin; Conference Room, Bldg. I.

2:00 - Psychosomatic Rounds; Bldg. 5.

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

Tuesday, May 6

Medical School and University Hospitals

8:30 - Conference on Diet Endocrines and Cancer; M. B. Visscher; 116 Millard Hall.

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.

12:00 - 1:30 Selected Topics, Permeability and Metabolism; Nathan Lifson; 129 Millard Hall.

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

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.

5:00 - 6:00 X-ray Conference; Presentation of Cases by General Hospital Staff; Drs. Lipschultz and Von Drashek; Eustis Amphitheater; U. H.

Ancker Hospital

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

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

Minneapolis General Hospital

8:00 - Pediatric Rounds; Spencer F. Brown; 5th Floor.

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

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

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

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

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.

Tuesday, May 6 (Cont.)

Veterans Administration Hospital (Cont.)

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, B. J. O'Loughlin; Conference Room, Bldg. I.

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

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, May 7

Medical School and University Hospitals

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

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

12:30 - 1:30 Permeability and Metabolism Seminar; Nathan Lifson; 129 Millard Hall.

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

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

5:00 - 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; R. Goltz; Todd Amphitheater, U. H.

Anoker 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

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

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

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

Wednesday, May 7 (Cont.)

Minneapolis General Hospital (Cont.)

12:30 - Pediatric Staff Meeting; The Well Baby Clinic Program; Sidney Scherling; 4th Floor Annex.

1:30 - Pediatric Rounds; E. J. Huenekens and Robert Ulstrom; 4th Floor.

2:00 - 4:00 Infectious Disease Rounds; 8th Floor.

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

Veterans Administration Hospital

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

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

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

Thursday, May 8

Medical School and University Hospitals

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

9:00 - 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.

12:30 - Physiological Chemistry Seminar; Metabolic Defects in Diabetes; William D. Cohen; 214 Millard Hall.

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

3:30 - Medicine-Pediatric Infectious Disease Conference; Heart Hospital Auditorium.

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

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

5:00 - 6:00 Radiology Seminar; Aortography in Urology; Drs. C. D. Creevy and Ronald Krumbach; Eustis Amphitheater, U. H.

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

Ancker Hospital

4:00 - Medical Pathological Conference; Auditorium.

Minneapolis General Hospital

8:00 - Pediatric Rounds; Spencer F. Brown; 5th Floor.

8:30 - Neurology Rounds; William Heilig; 4th Floor.

10:00 - Psychiatry Grand Rounds; J. C. Michael and Staff; Sta. H.

Thursday, May 8 (Cont.)

Minneapolis General Hospital (Cont.)

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

1:00 - Fracture-X-ray Conference; Dr. Zierold; Classroom.

Veterans Administration Hospital

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

8:00 - Surgery Grand Rounds; Conference Room, Bldg. I.

11:00 - Surgery Roentgen Conference; B. J. O'Loughlin; Conference Room, Bldg.I.

Friday, May 9

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.

10:30 - 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; Carcinoma of the Vagina; Irwin H. Kaiser; 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 Dermatology Seminar; W-321, U. H.

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

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

11:00 - Pediatric-Surgery Conference; Dr. Wyatt, Forrest Adams; Classroom, Sta. I.

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

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

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

Veterans Administration Hospital

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

Friday, May 9 (Cont.)

Veterans Administration Hospital (Cont.)

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

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

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

Saturday, May 10

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.

11:30 - Anatomy Seminar; Spinal Integration of Locomotion in the Elasmobranchs, Berry Campbell; Nerve Fiber Growth, Jennifer Sullivan; 226 Institute of Anatomy.

Ancker Hospital

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

Minneapolis General Hospital

8:00 - Pediatric Rounds; George Lund; 5th Floor.

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 - Hematology Rounds; P. Hagen and E. F. Englund.