LEPROSY OF THE EYE

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The eyes are subject to a great variety of infectious diseases. Of these the disturbances caused by the *Mycobacterium leprae* were, until recently, responsible for more ocular and adnexal damage and more blindness than the microorganisms and toxins of any other infectious disease. This led Boshoff to urge, "Always examine the eyes in leprosy."

The spread to the eye is most likely by the hematogenous route. However, an ascending infection from the nose by way of the nasolacrimal duct or a direct extension from macules and nodules of the face has been suggested. *Mycobacterium leprae* also enter the fine cutaneous twigs of the peripheral nerve of the face and forehead. From there they spread upwards into larger branches affecting both the motor and sensory axones. Involvement of the eyes and the extent of ultimate visual impairment are influenced by geographic and racial factors, the type and duration of the infection, the patients' general health, the adequacy of therapy, and other variables.

In Japan, Korea, Okinawa, Formosa, and Hong Kong, I found ocular involvement in approximately 10% of the patients. In India, I noted an even lower percentage of eye involvement. From Israel, Landau and Gabboy reported involvement of the eyes in over 90% of the cases. In Havana, Lopes found "some lesion of the eye or of its appendages in every single case of leprosy."

In Prendergast's series, at the United States Public Health Hospital in Carville, Louisiana, Mexicans developed fewer eye lesions than patients of other races. According to Cochrane, King, and Clemmy, dark-skinned natives of Asia and Africa also exhibit fewer ocular manifestations than lighter-skinned patients, such as Anglo-Indians.

In a recent survey amongst the multi-racial population of Hawaii, Chung-Hoon and Hedgcock found a smaller incidence of the disease among lighter-skinned, "diluted" part-Hawaiians than among other ethnic groups, such as the darker-skinned pure Hawaiians and Filipinos.

Defective nutritional status, vitamin, iron, other mineral and protein deficiencies, notably those of lysin and methionine, are capable of rendering susceptible individuals more prone to ocular complications and retard the healing of existing eye lesions.

General debility, diabetes, chronic liver or kidney disease, hypertension, amyloidosis, venereal disease, tuberculosis, chronic dysentery, malaria and parasitic infections may per se be responsible for ocular complications. When these conditions co-exist with leprosy they often predispose to the ocular manifestations of this disease. Foci of infection such as carious teeth, infected sinuses, infected or draining ulcers, areas of infected bone necrosis may aggravate lesions of the cornea and uveal tract or precipitate and prolong endogenous uveitis, episcleritis and scleritis.

Eye injuries among patients with leprosy are potentially more serious than those among a corresponding group of healthy population. It is therefore essential that patients' eyes be protected with goggles, case-hardened lenses, or at least with regular glasses or sunglasses when they are exposed to hazardous occupations. It is also highly desirable that patients wear glasses or goggles when they are out of doors, exposed to flying particles of dust and dirt. Trivial injuries in patients with anaesthetic cornea are capable of producing corneal abrasions and ulcers that pass unnoticed but may go on to perforation, uveal prolapse, and possible purulent endophthalmitis.

Acute or chronic alcoholism and drug addiction are also of considerable importance in leprosy. They render patients less aware of foreign particles and injuries to their eyes and predispose them to potentially grave and permanent ocular damage.

Duration of Infection

Kirwan called attention to the relative scarcity of eye complications during the first five years of the disease. Chance stated, "The incidence of ocular symptoms bears no relation to the duration of the general disease, but may come on at any time in its course; although it is not usually found till several years have passed." Morrow and Lee, on the other hand, in commenting on the frequency with which the eye is involved, said that such involvement occasionally occurs quite early in the disease. Elliott reported perineural beading along the course of corneal nerves together with early degenerative changes of the iris in a child four years old! He drew up an "ocular manifestation time scale," and postulated that the duration of infection bears a distinct relationship to the appearance of various ocular lesions.

Type of Infection

In lepromatous leprosy, ocular lesions are due to destruction and replacement of the normal cellular structure by an overgrowth of granulomatous tissue. This process is accompanied by inflammatory and ultimately degenerative changes within the eye. Bacilli in this type of leprosy are usually present in large numbers with a tendency to aggregate to form "globi" and typical foamy "lepra cells."

The eye lesions are for the most part confined to the anterior segment. However, they do invade the posterior segment as well, though less frequently.

In tuberculoid leprosy, epithelioid cells predominate. The *Mycobacterium leprae* are rarely found in smears. Invasion occurs in the sheath of the nerves, producing a chronic interstitial neuritis with ultimate destruction of nerve fibres and thickening of fibrous tissue. The ocular lesions and mask-like expression which are often associated with tuberculoid leprosy are secondary to an extension of the inflammation along the source of the fifth and seventh cranial nerves.

In the indeterminate forms of leprosy, skin lesions show few bacilli and do not have a tuberculoid histologic structure. The severity and nature of ocular involvement run a parallel course with the systemic manifestations.

Acute Leprous Reaction and Erythema Nodosum

According to Muir, they are one of the most distressing conditions of leprosy, as they may bring on "irreparable damage to the eyes in a few days." From an ophthalmologic standpoint, acute lepra reactions may be accompanied by severe pain, lachrymation, photophobia, circumcorneal injection, cells in the anterior chamber, K.P.s, pigment deposits on the anterior lens capsule, posterior synechiae, and occasionally exudation into the vitreous body. In addition to severe uveal inflammation, episcleritis and nodular scleritis may also occur during these reactions. Erythema nodosum leprosum causes similar ocular complications.

Treatment of the eyes during these reactions depends upon the extent and severity of the involvement. If signs of iridocyclitis or other ocular complications supervene, they should be treated in accordance with standard accepted methods of therapy.

The advent of steroids such as cortisone, ACTH, prednisone, prednisolone, and others have replaced many of the older forms of therapy. These hormones are especially valuable, as they permit

continuation of sulfone therapy without interruption. In Wade's opinion, "cortisone does not cure iritis but changes the process from one of acute, increasing inflammation to a low-grade, easily controlled, subsiding one." It is very likely that as the physiologic and biochemical action of pituitary and cortical hormones becomes better understood, our concepts regarding them will undergo further modifications in the future. For the present, from the clinical standpoint, we are fortunate to have available substances that dramatically suppress the symptoms of inflammation. Long-term, individualized steroid therapy as advocated by Steffensen often produces great improvement or even clinical remission in chronic cases that heretofore have been relatively unresponsive to shorter and less intensive treatment.

If cortisone is used, Jopling and Cochrane recommend five-day courses, beginning with 100 mg. on the first day, 75 mg. the second day, 50 mg. the third day, 25 mg. the fourth day, and 12.5 mg. the fifth, as long as the reaction is being controlled. They feel that these courses may be repeated as the need for them arises.

If ACTH is used, Jopling and Cochrane recommend five-day courses, beginning with 40 mg. of long-acting ACTH gel on the first day, reducing it to 30 mg., 20 mg., 10 mg., and 5 mg. over a five-day period. We have used higher beginning dosages than these—40 mg. long-acting ACTH gel twice a day—for the first two to three days and then gradually reducing the dosage. We also use ACTH gel at the beginning of therapy and follow it with meticortone, 5 mg. three times a day.

Improvement of chronic cases can be maintained by the administration of prednisone or prednisolone. These drugs have been used continuously for ten months or longer. The dosage schedule for relief of patients varies from an initial dose of 10 to 30 milligrams daily for the first three or four days followed by a maintenance dose of 2.5 to 5.0 milligrams daily and later on alternate days.

When long-term steroid therapy is used, the intake of sodium should be limited, and 1 to 3 grams of potassium chloride by mouth daily should be prescribed. Antacids have been recommended to prevent gastrointestinal disturbances.

The long-term systemic administration of any of the steroids calls for thorough and repeated careful physical examinations and laboratory tests.

Therapy

The introduction of sulfone drugs has resulted in greatly improved prognosis for both the systemic and the ocular manifestation of leprosy.

Lowe believed that the serious eye problems in leprosy were preventable by early diagnosis and thorough general treatment. Cochrane felt that blindness may be prevented if treatment is commenced at an early stage. However, he called attention to the danger of complacency of assuming "all is well" because these drugs are being administered. He felt that active lepromatous eye lesions may become aggravated and blindness may be hastened if the drugs are administered by untrained personnel and the eyes are not carefully watched to prevent damage from iridocyclitic complicating acute lepra reactions. Choyce observed that sulfones prevent, mitigate, and delay ocular complication. In his series, as a result of adequate therapy, the ocular signs became arrested in several patients, while in others regression took place. Yet, he noted that no patient had a "complete cure" of his eye lesion.

Once a lepromatous eye lesion develops, sulfone therapy alone as a rule will not arrest its progress. Nor will sulfone drugs restore the vision of patients whose eyes have undergone irreversible damage. However, if chemotherapy is administered early in the course of the disease, at regular intervals, over a sufficiently long period of time and in adequate doses, it appears to have a beneficial effect on eye lesions in many, though by no means in all, patients. Unfortunately, even under the most ideal conditions, eye complications continue to occur both among patients admitted previous to and since the discovery of sulfone drugs.

Elective, intraocular surgery should not be undertaken unless the patient's eyes have been in a quiescent stage for at least three to six months. If it is decided upon, preliminary slit lamp examination of the eyes should be done. If this reveals evidence of active uveitis, the operation should be postponed. The nasolacrimal passages should be tested for their patency and should be free from discharge. The patient should be able to recognize light and be able to tell the direction from which it comes. For four to seven days preceding surgery, as a prophylactic measure, he should be given eye drops containing a sulfa drug (e.g. 30% sulfacetamide) or a topical antibiotic (e.g. 1.5% chloromycetin solution) for instillation into both eyes several times a day. His general health should be checked over. Regarding surgical treatment itself, only those operations should be chosen which combine the greatest safety with the best results; the operative technique

applied should be best suited to the type of surgery that is being planned; a great deal of experience should be acquired in the method of one's own personal preference; meticulous, aseptic surgical technique should be observed. The postoperative use of steroids, enzymes, antibiotics, analgesics, and hypnotics should be prescribed as need for them arises. Adequate electrolyte balance should be maintained. With these precautions, some degree of useful vision may often be restored to patients who were previously considered hopelessly blind.

Bony Orbit

Deep-seated orbital pain may be encountered. When it is associated with a blind eye, retrobulbar injection of 1 to 2% novocain followed by 1 to 2 c.c. 75% ethyl alcohol usually effects relief for several months or permanently. Enucleation is an alternative form of treatment. Supraorbital, infraorbital, and frontal neuritis, like neuritis elsewhere in the body, may also give rise to severe subjective discomfort. It may be treated with analgesics, salicylates, the administration of vitamins B and K, discontinuation of sulfone drugs and diathermy over the affected area. Intraneural injection of equal parts of 1 to 2 c.c. of 2% novocain and a suspension of 25 mg. per c.c. of hydrocortisone has been recommended to relieve the edema responsible for the pain. If these measures are ineffective, injection of 1 to 2% novocain followed by 75% ethyl alcohol or 25% magnesium sulphate may effect symptomatic relief. As a last resort, splitting the nerve sheath or avulsion of the nerve must be considered.

Lacrimal Apparatus

Fuchs noted that Japanese investigators found lepra bacilli in the tears of 66 per cent of patients whose eyeballs were normal.

Leprotic dacryoadenitis has been described by King, Cochrane, and Sloan. Amendola, prior to the discovery of sulfone drugs, recommended surgical excision of the lacrimal gland as the treatment of choice for acute eye complications. In his experience, "this procedure never failed. It completely relieved pain and stopped other acute ocular manifestations."

Leprotic dacryocystitis is often secondary to advanced nasal disease. It may be treated by intubation of the nasolacrimal duct, dacryocystectomy, or if the condition of the nose permits, with dacryocystorhinostomy.

Epiphora occurs occasionally. It may be attributed to lagophthalmos or to eversion of the lacrimal punctum. Both conditions are amenable to surgical repair.

Diminished secretion of tears—dry eyes—associated with facial palsy and inability to close the eyes in tuberculoid leprosy, is more frequently encountered than excessive tearing. For this condition, the local instillation of 1% methyl cellulose is an especially valuable artificial tear substitute, as it does not support bacterial growth.

Eyeball

Shrunk, deformed, phthisical blind eyes are still encountered among patients in the older age groups. Large, bulging, unsightly staphyloma, and corneal leproma are not infrequent accompaniments of the disease. At times the globe is so large that it protrudes between the lids; the upper lid is drawn up and the lower lid sags down in lagophthalmos. However, as long as light perception remains and the eye is free from pain, it is best to leave it alone. If light perception is lost or if the eye becomes red and painful, its removal for both therapeutic and cosmetic reasons is indicated. Contracted sockets may need surgical reconstruction to permit the wearing of an artificial eye. In this regard, it should be kept in mind that patients who have lost several fingers find it difficult or impossible to insert and remove artificial eyes. In these cases, dark glasses or merely an eye patch worn over the affected eye may be sufficient to conceal the cosmetic deformity and is preferable to a major surgical procedure such as reconstruction of the conjunctival cul de sac.

Brows and Lids

The disease affects the eyebrows and lids with great frequency. Simple hypertrophy of the lids and brows is common. Lepromas appear on the brows and upper lids, but do not invade the lower lids. These lesions often resolve in time on systemic treatment with sulfone drugs. Blepharochalasis of the upper lids is seen in late cases. It is due to stretching of the skin and relaxation of the tissues, by previous lepromatous nodules. If it impairs vision in the upper isopters of the peripheral visual field, it can be corrected by operative interference.

Anaesthetic areas on the face, upper and lower lids occur in tuberculoid leprosy. The blinking reflex is frequently absent. In this type of the disease abnormalities of the lids and lashes are primarily responsible for the ocular damage and ultimate visual impairment. Paralysis of the orbicularis with facial paralysis is noted in about ten per cent of the cases. Atrophy of the involved muscles usually follows. The lid margins are almost invariably involved. Entropion of the upper and lower lids causes subjective

discomfort and objective cosmetic deformity. When the lashes are present, they may be misdirected, causing trichiasis, conjunctival or corneal irritation, corneal ulcers and scars. Widening of the palpebral fissures with sagging of the lower lids is frequently observed. In early cases, this causes only slight cosmetic disfigurement. As the condition progresses, paralytic ectropion and lagophthalmos result. Loss of sensation, loss of protection by the lids, and loss of diminution of tearing may result of chronic conjunctivitis, exposure keratitis, corneal ulceration and perforation of the globe.

Entropion of the upper lid can be corrected by using Gleyze's technique or other similar operations.

Entropion of the lower lid may be corrected by resection of the tarsus and orbicularis, or by a Hughes type of tarso-conjunctival graft.

Trichiasis may be very annoying and may cause corneal damage. It is treated by periodic epilation or electrolysis of the misdirected lashes. If severe, the condition may be surgically repaired with a tarso-conjunctival graft from the opposite lid sutured into an incision at the muco-cutaneous junction.

Ectropion of the lower lid may be paralytic or cicatricial. If it is paralytic, it is usually associated with lagophthalmos. A transparent, plastic cup worn over the affected eye will protect the eyes, especially during sleep. The instillation of 1% methyl cellulose with or without 0.5% cortisone or mild antiseptic or antibiotic ointments also affords some degree of protection to partially exposed globes. However, the ideal treatment for ectropion and lagophthalmos is surgical repair. Lateral tarsorrhaphy, Minsky's figure "8" suture, the Kuhnt-Szymanowski operation, or repair by a fascia lata sling are all suitable and may be used, depending upon the severity of the defect and the discretion of the surgeon.

In paralytic lagophthalmos involving the upper lid, recession of the levator as recommended by Goldstein is a valuable approach.

In all operations involving flaccid lid tissue, it should be kept in mind that the underlying muscles lack tonus and are usually atrophic. To obtain an adequate functional and cosmetic result, it is advisable to correct these conditions fully or even slightly overcorrect them. Transplantation of the temporalis muscle according to Ferris Smith's technique has also been recommended to improve the appearance of patients and to lend tonus to paralytic lids.

Uni- or bilateral loss of hair follicles from the eyebrows with loss of lashes may appear early in the course of the disease. The

loss of brows commonly begins on the temporal side and may involve the entire brow. In countries where eyebrow pencils are available, female patients are often content to use them as cosmetic beauty aids. Intradermal artificial pigmentation—tattooing—to the area of the brows has been successfully performed at the United States Public Health Hospital in Carville, Louisiana. Transplantation of individual hair follicles to create eyebrows was first suggested by Fujita. At the present it is commonly and successfully practiced in Japan. We prefer hair-bearing grafts from the scalp or from the opposite brow if they are available.

Ocular Muscles

Leprosy seldom causes oculomotor disturbances. Mitsuda reported that he has not seen paralytic squints arising from the disease nor was he able to find the bacillus in the oculomotor nerve. However, Viallefont and Fuentes and King did find paralytic strabismus due to involvement of the third nerve. Divergent strabismus secondary to amblyopia of one eye is not uncommon. Paralysis of the intrinsic muscles of the eye involving paralytic mydriasis or accommodative palsy occur periodically. They occur more frequently in patients with lepromatous leprosy who have undergone erythema nodosum type of reactions. Prescription of eye glasses with suitable presbyopic reading addition is usually sufficient to enable patients with such defects to read, sew, and do close work.

Conjunctiva

Leprosy bacilli as a rule do not invade the conjunctiva. However, the bacillus has been recovered from the conjunctival secretions in large numbers, even in eyes showing no leprous stigmata.

Acute superimposed infectious conjunctivitis usually responds to topical applications of 30% sulfacetamide or ophthalmic aureomycin drops. If these drugs fail, a smear, culture and antibiotic sensitivity test from the conjunctival sac frequently help determine the antibiotic to which the bacilli are most sensitive and which is most likely to control the infection. Coexisting trachoma occurs frequently, especially in the tropics. According to Professor Ida Mann, the sulfones used in the treatment of leprosy "can entirely kill the trachoma virus." Professor Mann confirmed this observation by successfully treating with DDS a group of children who had trachoma, but did not have leprosy.

Chronic conjunctivitis is common. It is believed to be due to exposure and secondary bacterial infection rather than leprous

infiltration. The treatment of chronic conjunctivitis depends upon the bacterial flora of the conjunctival sac. It usually responds to antiseptic or antibiotic collyria or to the local application of 0.15 to 0.25% zinc sulfate drops, 1% methyl cellulose, etc. We have no experience with the topical instillation of sulfone drugs in the treatment of leprosy eye lesions. Due to the granulomatous nature and chronicity of the disease as well as the slow action of sulfone drugs, we feel that this method of administration is of doubtful value. However, Tsukahara, Ishihara, and Tajira advocated topical applications of 1% to 5% promin ointment as well as subconjunctival injections of 5% promin.

Episclera and Sclera

The episclera, according to Fuchs, is the earliest site of ocular involvement. Valle believed that the rich anastomosis between the anterior ciliary arteries and posterior conjunctival vessels is responsible for the preferred episcleral location. Yellowish, gelatinous, leprosy nodules containing bacilli usually abound in the episclera near the limbus. These nodules are often symmetrical and are more commonly situated on the temporal halves of the bulb. They tend to spread around the limbus and infiltrate the cornea. They may even invade the angle of the anterior chamber. They temporarily respond to topical or in severe cases to systemic administration of cortisone, hydrocortisone, or some of the other steroids. However, they frequently recur.

The sclera itself usually does not harbour bacilli. A yellowish discolouration of the sclera has been reported by several authors. Anterior, intercalary, or scleral staphyloma follow repeated acute attacks or chronic forms of episcleritis or scleritis. Tissue therapy consisting of intramuscular or subconjunctival injections of placenta extracts has been recommended by Pennec for these unsightly lesions. In a few clinics in India, staphyloma are resected. We feel that if the lesions are sufficiently large to cause cosmetic deformity or pain, the eye should be removed.

Cornea

The cornea is the most vulnerable of all ocular structures affected by the *Mycobacterium leprae*. Bacilli may be found in corneal scrapings.

Infiltration of the corneal nerves may be demonstrated by slit lamp examination. This process is essentially similar to the infiltration that takes place in the peripheral nerves elsewhere in the body. Beading of the corneal nerves in the superior lateral quadrants of both eyes was observed by Pillat. Thickening of the nerves

in the stroma with minute granulomatous infiltrations has been described by Boshoff.

Partial or total loss of corneal sensitivity is an important sign, as either may be the forerunner of neuroparalytic keratitis with consequent visual impairment. Thomas stated that sensory nerves are believed to exert some controlling effect upon the metabolism of the corneal cells, chiefly the epithelial cells. When this proper, regulatory effect is lacking, there is an accumulation of cellular metabolites causing an edema and tissue destruction. The cellular edema and disturbed nutrition with its accumulated extracellular and intracellular deposits leads to a breakdown and exfoliation of the epithelium so that minor trauma, bacteria, and foreign bodies can readily damage this structure.

Exposure keratitis or keratitis lagophthalmos is a serious complication of tuberculoid leprosy. It is the aftermath of paralysis of the orbicularis muscle. The involvement is usually in the lower, exposed portions of the cornea. As the cornea derives some of its nutrition from the tears as well as from the limbal vessels and the aqueous, abrasions, ulcers, and scars that accompany this type of keratitis are partly due to evaporation of tears.

The prophylactic treatment of both neuroparalytic and exposure keratitis includes protection of the cornea with goggles. If goggles are equipped with side shields they create a moist chamber and afford added protection and comfort. Especially constructed plastic cups which can serve as moist chambers are commercially available. If such are not available, at least eyeglasses or sunglasses should be provided for added protection. Patients should also be instructed to apply 1% methyl cellulose to their eyes before retiring and to keep their eyes patched at night. Methyl cellulose is preferable to bland ointments and oils such as U.S.P. lanolin or U.S.P. petrolatum, as the latter tend to produce mechanical irritation, delay or inhibit wound healing despite lubrication. If patients do not fully appreciate the potential hazards of neuroparalytic or exposure keratitis, their eyes should be permanently protected by a lateral tarsorrhaphy or a lateral and a medial tarsorrhaphy. Two small adhesions between the upper and lower lids afford considerable protection to the cornea, and still permit patients to see through a central unobstructed narrow slit.

The active treatment of corneal abrasions and ulcers consists of the application of local, subconjunctival or systemic antibiotics, the use of mydratics and patching the eye. Grossly infected corneal ulcers may require curettement and thermal or chemical cautery.

The systemic administration of vitamins A, C and D, riboflavin, or multivitamin preparations has been recommended to assist the healing or corneal ulcers.

Superficial punctate keratitis is considered pathognomonic of the disease. Shionuma found it in 21.7 per cent cases of lepromatous leprosy. It usually begins at the superior limbus as a light milky haze in the substantia propria, dotted by tiny, white, irregular spots resembling dust or grains of chalk. As it spreads downwards, its lower margin is delineated by a wavy line. Lepra cells and lepra bacilli may be seen in the scrapings from such lesions.

Leptotic pannus may be seen in all stages of vascularization and granulomatous infiltration. Clinically it resembles the pannus of phlyctenular kerato-conjunctivitis. It differs from that of trachoma by the absence of involvement of the tarsal plates. The lepromatous pannus encroaches and often destroys Bowman's membrane as it advances into the parenchyma. In doing so, it causes a partial or complete hyperplastic keratitis and ultimately brings on severe visual loss. Its progress may be checked by peridectomy and recession of the vascularized tissue four to five millimeters back of the limbus. Large limbal or corneal vessels may be destroyed with the electrocautery. These operations should be followed with local applications of cortisone drops or ointment for weeks and months. Beta radiation has also been recommended as an effective means to control extensive vascularization of the cornea.

Sclerosing keratitis originates as a white, milky band in the episclera or sclera. It gradually advances to the cornea, often giving rise to sclero-corneal leproma. These lepromas are generally bilateral and may attain very large proportions. Mitsuda reports that 94 per cent of his patients with lepromatous leprosy developed leproma of the cornea. This figure is out of proportion with our statistics (less than 10 per cent) and with my personal observations in Korea, Formosa, and India.

Another type of leproma occurs in the centre of the cornea surrounded by relatively transparent tissue (Ruato's corneal leproma). Large, isolated corneal leproma, according to De Souza, may be extirpated surgically or treated with galvanocautery or with carbon dioxide snow. However, when light perception is lost and the eye becomes red or painful, little can be gained by temporizing procedures, and enucleation is the treatment of choice.

Interstitial, nodular or discoid keratitis is also seen in the lepromatous form of the disease.

Iris and Ciliary Body

Lepromatous iritis and cyclitis may be caused by actual invasion of the uveal tissue by the bacillus itself—granulomatous uveitis—or it may be due to hypersensitivity to anaphylaxis resulting from protein sensitization—non-granulomatous uveitis.

Granulomatous uveitis of leprosy is a chronic, nonpurulent inflammation of the uveal tract which results from actual infection. It runs a prolonged course and causes tissue necrosis. In this type of inflammation, bacilli are present in enormous numbers. Fuchs demonstrated large nests of lepra bacilli in the iris and in the ciliary body on histologic examination. The essential pathologic change is characterized by exudation, mobilization, and proliferation of inflammatory cells. These changes may be observed under the slit lamp. They consist of an aqueous flare with mutton fat type of keratic precipitates and fibrinous exudate in the anterior chamber. The clinical course is characterized by slow, often insidious onset and gradually decreasing visual acuity. Pericorneal injection is usually slight. If the disease progresses, multiple—according to Mendonca de Barros, myriads of—miliary, glistening lepromatous nodules considered pathognomonic of the disease, can be seen near the pupillary margin or in the iris stroma. There is marked tendency to form heavy posterior synechiae. The latter are usually permanent and are difficult to break. Whole sectors of iris may become atrophic, depigmented, and lead to heterochromic iridocyclitis. Seclusio and oclusio pupillae and secondary cataract frequently supervene.

Each exacerbation produces increased damage to the eyes. In severe cases, the eye may progress to phthisis.

Non-granulomatous iridocyclitis has been described by Woods as a sterile reaction. He states: "It is the result of acute and later chronic recurrent insult to the tissues. The latter could be due to bacterial hypersensitivity, or to an anaphylaxis caused by protein sensitization. The absorption of the soluble bacterial protein from a focus of infection may readily explain the inflammatory reaction in these eyes. The clinical course in this type of involvement is characterized by sudden onset of considerable pain which reaches its maximum intensity in two or three days. Intense pericorneal congestion, hyperemia, muddy iris, contracted pupil, photophobia, and lacrimation are present. On slit lamp examination, fibrinous exudation into the anterior chamber with many cells and intense aqueous ray are noted. There is only slight tendency to the formation of posterior synechiae. The attacks are short-lived, and run a self-limited course in one or several weeks.

“ After repeated attacks, however, annular posterior synechiae may be found. The iris becomes thinned and atrophic. There may be clouding of the lens with secondary cataract. Organized fibrinous exudates on the iris may simulate the picture of severe granulomatous disease.”

Ashton called attention to the frequent co-existence of uveitis with infective, usually streptococcal, foci in the tonsils, teeth, etc., and the improvement which sometimes follows the removal of such foci. He felt that these findings offered at least persuasive support for regarding focal sepsis as a factor of importance in the etiology of non-granulomatous uveitis. For this reason, in non-granulomatous types of uveitis of leprosy a thorough medical search should be carried out and infected foci should be eradicated.

There is no uniformly beneficial treatment for acute iridocyclitis. In both granulomatous and non-granulomatous forms of the disease, the local instillation of mydriatics (1-2% atropine, 0.2% scopolamine, 10% neosynephrine) and 1 to 2% dionin are used. Cortisone and other steroids are of great value in the treatment of acute attacks of iridocyclitis, choroiditis, and occasionally optic neuritis. Treatment with these preparations was discussed in conjunction with acute lepra reactions.

External heat in the form of hot, moist compress, heating pads, or short-wave diathermy is gratefully received by most patients. The parenteral administration of certain enzymes, such as trypsin, occasionally provides relief in the management of recalcitrant cases of uveitis. The intravenous administration of calcium gluconate or lactate is also occasionally used in arresting refractory cases of iritis.

Lesions of the Posterior Segment

Lesions of posterior segment are rare. Valle advised that all conditions capable of producing changes in the retina and choroid, such as syphilis, tuberculosis, and others, should be ruled out before the diagnosis of chorioretinitis due to leprosy is made. Changes in the retina are believed to be secondary to those in the uveal tract. Neither Kirwan nor Prendergast was able to demonstrate the bacillus leprae in the retina or optic nerve. However, Mancione and Inatomi reported acid fast organisms in both the choroid and retina. Prendergast observed involvement of the fundus in 42 out of 241 patients. Trantas and Pupert reported uni- or bilateral isolated punctate lesions in the periphery of the choroid with some pigmentary proliferation. Stallard noted clumps of lepra bacilli in types of changes in the fundus, during acute leprosy reactions, in 43 the subrachoroidal lymph spaces. Verne described four different

out of 120, or 35 per cent, of his patients. They consisted of: congestion of the disc with blending of the shades of the nasal and temporal halves of the disc; papilloedema, which varied from blurring to complete disappearance of the disc margins; infiltration of the posterior pole seen as an increase of the retinal glimmer; a brilliant perimacular circle with or without vascular changes; persistent venous dilatation accompanied by vascular undulations; and a decrease, sometimes amounting to collapse, of the central artery. In addition to the foregoing, at times, Verne also observed perivascular infiltration of the afferent and efferent central vessels and a grayish, edematous appearance of the macula.

Kennedy reported nine cases of leprous choroiditis. Elliott reported six cases of retinal pearls visible through the ophthalmoscope. They appeared as small, waxy and creamy white pedunculated nodules projecting into the vitreous. Somerset and Sen described round, yellow homogenous nodules situated superficially on the retina.

Van Poole described 49 cases of optic neuritis among 206 patients. He believed that these were transitory and were caused by bacterial allergy. Takahashi, in lepromatous cases, was able to demonstrate bacilli in the optic nerve. He ascribed the involvement to an extension of the lepromatous infiltration.

In conclusion, I should like to quote my former colleague, Dr. Paul W. Brand, distinguished orthopedic surgeon of the Christian Medical College, Vellore, South India. Dr. Brand, in his Hunterian lecture before the Royal College of Surgeons in 1952, called on "orthopedic and plastic surgeons to come forward and open the door that leads the leprosy patient from isolation back to his family and job." Dr. Brand's timely challenge is equally applicable to the ophthalmic profession. With adequate sulfone therapy, timely and appropriate prophylaxis, we can prevent much suffering and eliminate needless blindness. With proper medical and surgical management, we can conserve eyesight and restore useful vision to patients who were previously considered beyond help.

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Trichiasis, lower lid.

Corneal leproma.

Left Corneal abscess.
Purulent endophthalmitis.

Corneal Leproma, right.
Anterior staphyloma, left.

Right exposure keratitis.
Left lepromatous pannus.

Partial madarosis both brows.
Corneal staphyloma right eye.

Perforation of eyeball.
Lepromatous pannus.

Corneal leproma.

Plastic cup to protect the eye during sleep.