

*Questions about the cause, the effect, the diagnosis, the infectivity, the clinical pattern, the prevention, and the treatment of trachoma are answered by an ophthalmologist from his experience and from the literature.*

## The Trachoma Story

By ARTHUR A. SINISCAL, M.D.

THE IMPORTANCE of trachoma "as a source of human suffering, as a cause of blindness, and as a national economic loss over large tracts of the world's surface is second to none among the diseases of the eye, or indeed, among diseases of all kinds."

Thus Sir Stewart Duke-Elder, a distinguished British ophthalmologist, assessed trachoma in a textbook published in 1934 (1). As late as 1950, it was estimated that 15 to 20 percent of the world population suffered from this disease (2).

Throughout the world, trachomatologists, ophthalmologists, sociologists, public health workers, and others are striving earnestly to improve the health and socioeconomic conditions of those afflicted with trachoma. But they wave no magic wand; what they accomplish, they accomplish through steady, organized effort, which depends in turn upon public understanding and support. The following pages tell of medicine's planned attack against this ancient infection.

According to Moutinho (3), trachoma was

---

*Dr. Siniscal since 1944 has been medical director of the Missouri Trachoma Hospital in Rolla, a service of the division of health, Missouri Department of Public Health and Welfare.*

---

well known in the civilizations of the four great river valleys—the Hwang Ho and the Yangtze Kiang, the Indus and Ganges, the Euphrates and Tigris, and the Nile—many centuries before Christ. It was recognized and treated in ancient Egypt, Greece, and Rome, as well as in countries of Biblical fame. The Moslem conquests probably led to its spread to Europe as early as the eighth century (4), and, undoubtedly, Napoleon's campaign to Egypt in 1798–1802 was responsible in large measure for its dispersion among the Europeans (5).

Believed to have been introduced into the United States during colonial times by European immigrants (6), the disease was spread throughout a central belt reaching from the Allegheny Mountains to Kansas and Oklahoma, among the Indians as well as others. According to Cosgrove (7), the trachoma found in Missouri and Arkansas originated from persons who came into these States from Tennessee and Kentucky. The trachoma in New Mexico and Arizona and probably California is believed to have come from Mexico (8).

Today, trachoma is found on every continent (4). It is common in most of Asia, where it affects from 30 to 60 percent of the population. It is somewhat common in Europe, where it affects approximately 5 percent of the population. More prevalent in the Balkan States and in northern European countries than elsewhere on

that continent, it is present also in France, the Netherlands, Germany, Austria, Hungary, Italy, Ireland, Great Britain, Norway, Sweden, and Denmark.

Once a serious public health problem in the United States, particularly in the central portion where it is considered endemic, trachoma is now relatively rare. It is still present, however, in Missouri and Arkansas and, to a lesser extent, in southern Illinois. In Missouri, active cases are found in the southern half of the State, particularly in the cotton-growing lowlands and in remote regions of the Ozark hill country. Accounting for 1 out of 4 pensioners on the State's blind pension roll 25 years ago, trachoma now is the disease of only 1 out of every 10 blind pensioners.

Questions asked most frequently about trachoma are given here with the answers available from the experience of many workers. Although much is known about the cause, the diagnosis, the clinical manifestations, and the prevention and treatment of this disease, not every question can yet be answered finally or completely.

### *What Is Trachoma?*

Trachoma is an infectious disease of the external lining membranes (or conjunctiva) of the eyeball and eyelids. It causes an inflammation of chronic duration, characterized by burning, itching, excessive lacrimation, and photophobia. Generally insidious in onset, it develops usually at a slow pace and is somewhat resistant to treatment. The disease is localized strictly to the conjunctiva of the eyelids and eyeball and to the tarsus and the cornea. The pathological changes in the tarsus and the cornea are deep seated and usually irreversible when once established. Persistent duration of inflammatory symptoms may lead to blindness, which results from corneal opacification and disintegration and, further, from cicatrized deformities of the eyelids and eyeball.

### *What Is the Cause of Trachoma?*

The etiological agent is believed to be a large virus or inclusion body of the group which includes the psittacosis and lymphogranuloma venereum viruses. The inclusion body of

trachoma is indistinguishable morphologically from that of the virus causing inclusion blennorrhoea of infants and swimming pool conjunctivitis of adults (also called paratrachoma). Some workers have regarded the agents of these infections as belonging to the *Rickettsia* group, but Bengtson's (9) research has shown that the trachoma bodies are rather distinct from the rickettsiae. Generally speaking, the view is taken that the trachoma body is a virus, but the question has not yet been finally settled. The dividing line between bacteria and rickettsiae or between rickettsiae and viruses is not sharp.

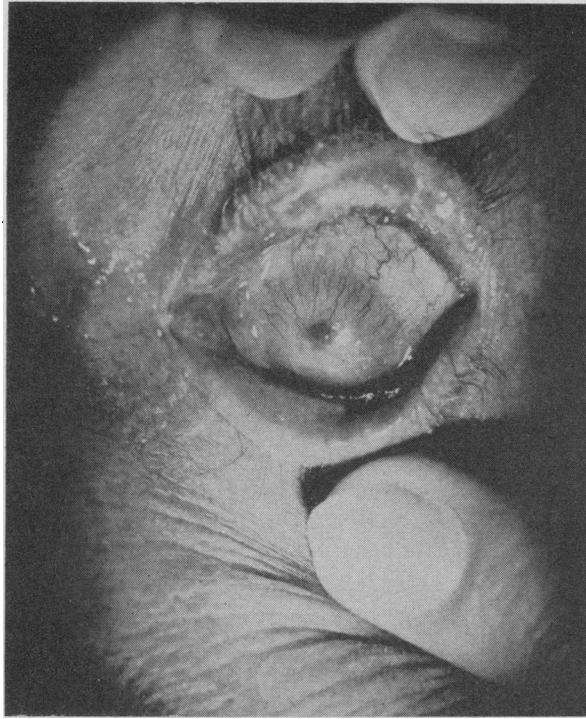
A great majority of scientists now believe that trachoma is due to the organism discovered by Prowazek and Halberstaedter. In 1907, they described cytoplasmic inclusions, the presence of which in the epithelial cells of the conjunctiva and the cornea manifests the first detectable sign of the disease in the laboratory. Thygeson (10) has pointed out that these intracellular parasites are relatively large particles and that their life cycle is very similar to that of the psittacosis and lymphogranuloma venereum viruses.

There is need for more research on the causative agent or agents of trachoma. The exact nature of the organism will remain obscure until it is definitely isolated and successfully cultivated.

### *How Are the Eyes and Eyesight Affected?*

If the infection is allowed to progress without treatment for several months or longer, a lymphoid infiltration in the conjunctiva and in the subepithelial tissue will result in hypertrophy and thickening of the conjunctiva and eventually in connective tissue formation and scarring. Even the tarsal plates are involved and may become buckled or deformed as a result of cicatrization. Follicles may or may not develop, but the absence of these should not rule out the presence of the disease (11, 12).

The most serious pathological complication of trachoma is considered to be the formation of pannus, which is a vascular invasion under the epithelium of the cornea. When pannus is allowed to progress centrally, the pupil becomes obscured by this veil-like opacity. Trichiasis,



**Florid pannus covering complete upper half of cornea, as seen in advanced stage of trachoma. Pannus is pathognomonic of the disease.**

a condition of ingrowing eyelashes, results from cicatrization of the lid structures; it causes the eyelashes to rub against the cornea, thereby destroying the epithelium and predisposing to ulceration, perforation, and dense corneal scarring. Blindness may be due to diffuse corneal scars in general, or to pannus formation over the pupillary area, or occasionally to secondary uveitis following perforation of the chamber and prolapse of its contents, resulting eventually in phthisis bulbi.

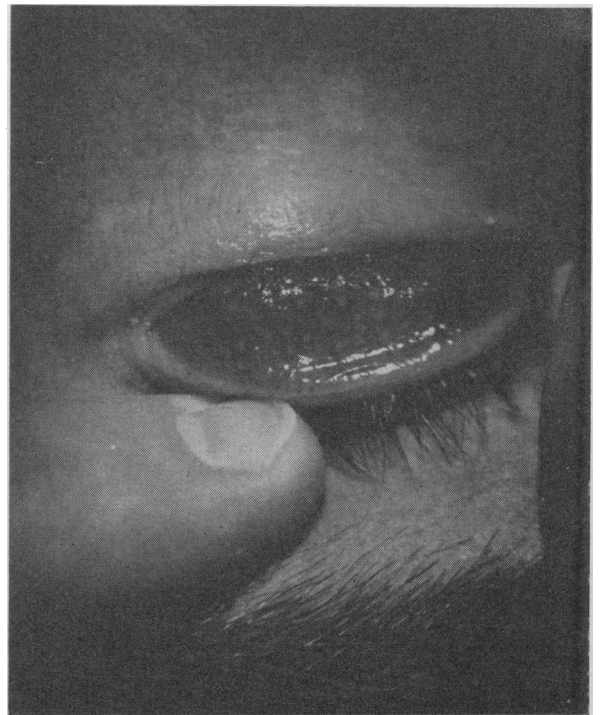
### *Is Pannus Essential to the Diagnosis?*

Although the presence of typical trachomatous pannus is a solid basis for making a diagnosis of trachoma, its absence does not absolutely rule out the diagnosis. Some observers (13-16) believe that any one or more of the following signs should be sufficient to establish a diagnosis of trachoma: (a) presence of typical follicles; (b) presence of pannus; (c) presence of cicatrization; (d) punctate depressions at the periphery of the cornea resulting from

cicatrization of corneal follicles; (e) finding of the inclusion bodies.

A study by Rice and Smith (17) of the records for 1,154 cases of trachoma found that 88 percent showed the presence of pannus involvement of the cornea. According to Cosgrove (7), a definite diagnosis of trachoma cannot be made without pannus unless a proved case has been seen in the same family. Recently, however, Julianelle and Smith (18) observed that approximately 37 percent of early trachoma cases do not show any pannus, and Mitsui (19) states that even a microscopic pannus can be absent in the earliest stage in some cases of trachoma. Mitsui observes that in Japan, microscopic pannus can be seen in about 50 percent of trachoma cases during the acute stage, and that marginal punctate infiltration of the cornea can be seen in a smaller percentage of cases.

Cuenod and Nataf (20), who stress the value of slit-lamp biomicroscopy in their monograph on corneal studies (21), state that pannus is one of the most constant pathological signs of trachoma, but that they believe it possible for true trachoma to occur without pannus, especially



**Trachoma granules in the conjunctiva, as seen in the florid stage of the disease.**

the early stage. Nataf (22) states further that, in his opinion, it is possible for certain forms of the infection to occur without pannus but that diagnosis in such cases should be made with reservations. Nataf believes that trachoma may possibly occur in certain regions without pannus, but that in order to prove diagnosis in such cases, it would be necessary to detect, after regression, the typical scarring pathognomonic of trachoma.

### *Why Is Early Diagnosis Important?*

As in other infectious diseases, early diagnosis and treatment of trachoma prevent its spread. Early discovery also makes it possible to prevent the advanced stages with their irreversible complications.

A clinical diagnosis of trachoma in its early stage is always difficult, since the classical landmarks of pannus, corneal infiltration, mature follicles, or cicatrization may not be present. The lack of agreement as to a definite differential diagnosis of early trachoma makes necessary a laboratory confirmation of the diagnosis whenever possible. However, because the virus is difficult to isolate or cultivate, a negative laboratory finding is unreliable.

Although the presence of inclusion bodies may clinch a positive diagnosis, their absence does not necessarily verify a negative diagnosis. The reason for this is that microbiotic or cellular findings typical of trachoma infection are not present throughout the length of the disease; it may be possible to find positive material from conjunctival scrapings early in the disease, but not always. In this connection a serologic test would be helpful. There is a clear-cut need for research on the serology and immunology of this disease.

The presence of several or all of the following signs plus a history of contact should make one strongly suspect that the diagnosis is early trachoma: excessive lacrimation with photophobia; papillary hypertrophy of the palpebral conjunctiva; a peculiar haziness of the lower cul-de-sac indicating diffuse infiltration therein; swelling and decreased rigidity of the upper tarsus; tiny filamentous symblephara in the upper fornices; large, discrete follicles; incipient pannus; and upper corneal infiltration. These signs may

occur in other conjunctival diseases, but in an endemic area of trachoma, one would be remiss in his duties if he neglected to take cognizance of them.

Lindner (5) states that in areas where trachoma is prevalent, every case of slight, ordinary conjunctivitis may be beginning trachoma. He considers the most important sign of the first stage to be the peculiar haziness of the conjunctiva of the lower cul-de-sac. Kuo (23) also stresses the importance of general haziness of the lower cul-de-sac, together with incipient pannus, in making an early diagnosis. Tabone (24) discovered that in a series of cases of eye infection in which diagnosis was difficult, approximately one-half of those tentatively labeled conjunctivitis later proved to be real trachoma.

### *Is Trachoma Highly Contagious?*

Trachoma is not highly contagious; otherwise, it would be more widespread than it is. Because trachoma is more likely to be transferred from one member of a family to another member than to contacts outside the home, it is sometimes referred to as a family disease, but it is not hereditary.

Important factors in the epidemiology of trachoma are general levels of living, particularly as they relate to sanitation and housing conditions, and geography and climate. Migration of infected persons into areas with high standards of domestic hygiene is not attended with danger, but such migration into areas with low standards of hygiene, where geographic and climatic conditions favor the disease, would tend to facilitate its dissemination.

Like other infectious diseases, trachoma is more likely to spread where unsanitary and overcrowded living conditions prevail. Although it is sometimes seen in persons of exemplary habits of personal hygiene, it is more likely to take root in persons who are careless about such matters. The use of a common towel to wipe the face, for example, is an almost certain invitation to the disease. Improvement in economic status, with its concomitant improvement in sanitation and housing, will materially aid in the elimination of the disease.

In general, a high incidence of trachoma is

observed in areas with a high mean temperature and little rainfall and in flood districts near the estuaries of large rivers. In all of these, sun, wind, and dust act as conjunctival irritants and are factors predisposing to the infection. In Missouri, trachoma is prevalent not only in the mountainous areas, where the climate is hot and the air dusty, but also in the cotton-growing lowlands of the southeastern delta, where the climate is humid and windy and the winds are dust laden.

### *Does Diet Have Any Effect on Trachoma?*

According to Rice and his co-workers (25), there is no evidence that a balanced diet supplemented with cod liver oil affected the course of trachoma in 18 untreated patients kept under observation for varying periods of time. However, it is the opinion of this writer, as well as that of other observers, notably Stucky (26) who made a great contribution to the prevention of trachoma in Kentucky, that inadequate diet and avitaminosis are contributory factors. At present, however, this concept must be regarded as unproved.

Glikson (27) states that his observations of over 25 years in Israel indicate that general nutrition is a decisive factor in an individual's immunity to this disease, and he adds that proper diet, including fresh milk and fresh, green vegetables, will shorten the duration of the disease or actually heal it. Murray (28) states that in South Africa adequate feeding supplemented by vitamin concentrates generally brings about a remarkable improvement within a few weeks even if no specific medication other than ablutions with saline is used. According to Bietti (29), if there is any importance in nutritional factors, lack of animal proteins and of calories is more likely to play a role than avitaminosis.

### *Are All Races Equally Susceptible?*

Generally speaking, all races are susceptible to trachoma in only slightly varying degrees. American Negroes seem not to acquire the disease, but miscegenates are not necessarily immune. Murray (28) reports that in South Africa, Negro tribes become afflicted with trachoma to a rather large extent. Extensive sur-

veys indicate that the disease affects about 29 percent of the natives in that area.

In Missouri, the disease predominates among persons of northern European ancestry, notably Irish, Scottish, English, German, and Scandinavian. Their forefathers formed the forward ranks of the pioneers who moved westward from the coastal States, bringing the infection with them. The disease became established in these families and still flourishes in some of their settlement areas, notably in the southern half of the State. Negroes in the State do not acquire the disease.

It is thought by some observers that the dark-pigmented races, especially Negroes, are more immune to trachoma than the light-pigmented ones. Seemingly in support of this contention is the fact that when trachoma in an Egyptian passes over to a European, the disease manifests itself much more rapidly and has a much more devastating effect. Wilson (30) states that trachoma tends toward spontaneous cure more frequently among Egyptians than among Europeans. He observes further that some of the Egyptians suffer little from this disease and that the lesions are mild in comparison with the lesions among Europeans.

Studies by Thygeson (31) indicate that the trachoma bodies show a predilection for the superficial cells of the conjunctiva without involvement of the basal cells and that they tend to spare the pigmented cells. This possibly could explain the resistance in certain dark-pigmented races to trachoma and the more favorable development of the disease among them.

Bietti (32) states that there are racial differences in the sensitivity to the disease and that the Negro particularly is more resistant. He does not maintain that degree of pigmentation is important, but rather that some innate, racial characteristic itself is responsible. In support of this theory, he mentions that among various African native tribes of equal pigmentation, some tribes develop the disease more severely and in greater numbers than others.

Rice (33) believes that the severity of trachoma varies considerably in different parts of the world and even within the borders of our own country. He states that trachoma probably does less damage among the Chinese, for

instance, than it does among the white population of Missouri and Arkansas. Factors of immunity and adaptability may even be more important than factors of race.

To sum up, although trachoma is very widespread throughout the Near East and the Orient, the disease appears to be less distressing in natives of these regions than in Europeans and their descendants, among whom the prevalence is relatively low.

### *Why Does Trachoma Still Exist?*

Trachoma still exists because it was so widespread in the ancient civilized world and because, too, it did not receive the advantages of a modern medical campaign until the past 3 or 4 decades. Before the advent of sulfonamides and antibiotics, treatment was largely empirical rather than specific.

Until recently, there was little organized attack against this disease abroad and practically none in this country. The idea of international collaboration in trachoma control has been in existence for almost 100 years, but only with the founding of the Anti-Trachoma League and the International Organization Against Trachoma in 1923 did the idea progress to action. Their quarterly publication, *Revue internationale du trachome*, provides an authoritative and dynamic organ for the consolidation and dissemination of knowledge and facts about the disease.

At present, mass treatment of trachoma has been started by the governments of many foreign countries, notably Formosa, French Morocco, and Tunisia, with the assistance of the World Health Organization, and initial favorable results are reported. Projects are being planned in Yugoslavia and Egypt, and surveys in Western Australia (34) and in South Africa (28) have emphasized the need for action in these areas.

In the United States, a program of attack against the disease was begun in the Appalachian area in 1913 by the Public Health Service (35). During the next 11 years, one or more temporary diagnostic and treatment centers were set up in Kentucky, Virginia, West Virginia, Tennessee, North Dakota, Arkansas, Georgia, and Missouri, and in 1923 a permanent

**Table 1. Operations performed at the Missouri Trachoma Hospital, 1932-53**

Year	Number of patients admitted	Number of operations			
		Total	Gratage	Entropion	Other <sup>1</sup>
1932-33----	635	598	257	64	277
1934-35----	609	452	228	77	147
1936-37----	562	297	139	56	102
1938-39 <sup>2</sup> ----	468	137	65	53	19
1940-41----	750	143	25	69	49
1942-43----	934	181	60	36	85
1944-45----	868	111	25	21	65
1946-47----	893	114	25	33	56
1948-49----	1,076	40	8	5	27
1950-51----	1,179	61	8	11	42
1952-53----	1,072	30	18	2	10

<sup>1</sup> Includes canthoplasties, electrolytic epilation, pterygia, dacryocystectomies, tarsiectomies, enucleations, and miscellaneous procedures. <sup>2</sup> No surgery done for 6 months in 1939 during changeover to new hospital.

hospital and clinic center was established at Rolla, Mo., by the Public Health Service in cooperation with the State. In more recent years, the States of the trachoma belt have carried on the battle against the disease.

### *Can We Rid This Country of Trachoma?*

During the past four decades, the prevalence of trachoma has decreased markedly (36). The program against the disease has been changing gradually from treatment of active, florid cases to the practice of preventive medicine and treatment of early and late and terminal cases. As shown by the data in tables 1 and 2, the number of operations performed at the Missouri Trachoma Hospital for trichiasis, entropion, gratage, and lid deformities and the number of cases of trachoma found among field clinic patients are declining steadily.

In the other States of the trachoma belt, the disease is no longer considered a significant problem. Arkansas has discontinued its extensive case-finding program, but within the next few years it will survey former high-incidence areas to see whether the disease has actually been eradicated. In southern Illinois, once an area of high incidence, a marked decline in the number of cases was observed between 1949 and 1953. No active cases have been re-

ported in the State since 1949. Kentucky discontinued its trachoma control program more than 4 years ago, and Oklahoma reports that the disease is now a negligible problem in areas where the incidence was once high. In Arizona and New Mexico, where an estimated 20 percent of the Indian population before 1938 was afflicted with the disease, a 1952 survey of the Indian reservation residents in all areas except Albuquerque found only 2.6 cases per 1,000 population.

With continued organized attack against this disease, it should be entirely wiped out in the

United States in time. Cooperation of independent ophthalmologists in trachoma areas and constant effort to teach the affected people good health habits will aid in reaching this goal. An improved standard of living and a rise in the general socioeconomic status are factors of importance.

### *What Treatment Is Given for Trachoma?*

At the Missouri Trachoma Hospital and generally in this country, the infection is treated with sulfonamide medications. Sulfonamide solutions are used as drops in the eyes several times daily, and sulfonamide tablets administered orally and sulfonamide ointments used nightly are adjunctive therapeutic measures. If the infection is in the early stage, treatment may be completed in approximately 3 weeks; if it is of long duration, treatment may last as long as 2 or 3 months, or even longer if the disease has become established.

Sulfonamides have been used to treat trachoma since about 1938, and most authorities agree they are active against the trachoma virus. More recently, the antibiotics have been used with favorable results in some instances. In the experience of this writer, the sulfonamides have proved more effective than any other therapeutic agents, the antibiotics appearing to be of secondary help (37).

Of the antibiotics, terramycin and possibly aureomycin seem to be most active against the trachoma virus (38-45). Chloramphenicol is considered much less effective, and penicillin, bacitracin, and streptomycin seem to be of little or no value in uncomplicated trachoma.

Mitsui (43, 44) reports that in Japan both acute and chronic trachoma respond well to terramycin treatment; but he observes further (45) that while in his opinion sulfonamides may not be the best agents against trachoma in these days of antibiotics, they certainly constitute a means of treatment in those cases that do not respond to antibiotics alone. Loe, who first used sulfonamides successfully against trachoma in 1937 among the American Indians in the southwest (46), has stated that in his experience the antibiotics have not proved successful in the treatment of this disease in Arizona and New Mexico.

**Table 2. Trachoma cases among patients of the Missouri Trachoma Hospital field clinics**

Year	Total number patients	New and old trachoma		New trachoma	
		Number cases	Per-cent of patients	Number cases	Per-cent of patients
St. Louis County					
1941	83	17	20.4	11	14.2
1942	0	0	0	0	0
1943	229	35	15.2	27	11.7
1944	368	95	25.8	42	11.4
1945	217	65	29.9	25	11.5
1946	382	85	22.2	21	5.4
1947	411	41	10.0	11	2.6
1948	0	0	0	0	0
1949	112	15	13.3	0	0
Potosi, Washington County					
1945	135	62	45.9	35	25.9
1946	154	70	45.4	36	23.3
1947	90	64	71.1	30	33.3
1948	213	91	42.7	47	22.0
1949	244	140	57.3	58	23.7
1950	456	191	41.8	98	21.4
1951	780	241	30.8	108	13.8
1952	428	89	20.7	48	11.2
1953	159	54	33.9	8	5.0
Eminence, Shannon County					
1945	268	59	22.0	25	9.3
1946	264	42	15.9	14	5.3
1947	130	11	8.4	3	2.3
1948	289	107	37.0	50	17.3
1949	106	51	48.1	16	15.0
1950	125	58	46.4	20	16.0
1951	442	74	16.0	36	8.1
1952	464	42	9.0	25	5.3
1953	395	21	5.3	6	1.5

Cosgrove (47), director of the Arkansas State health program for trachoma control, believes that sulfonamides are the most effective agents against trachoma, and that the antibiotics, including the broad-spectrum group, have no effect against this disease.

### *Does Trachoma Tend to Recur?*

Trachoma is a disease of recurrences and relapses. The infection may have become dormant or latent so that not infrequently an arrested case may later show signs of activity. A recurrence may be due to the patient's having returned to an environment of smoke, dust, or wind, or to a mode of living not conducive to eye health.

Recurrences sometimes result from insufficient or inadequate treatment and occasionally from trauma, scarring, or possibly allergy. Some recurrences, of course, are due to reinfection from a member of the family who has not had treatment. All family members should be examined when trachoma is found to be present in one of them.

### *Is There a Definite Criterion of Cure?*

As yet there is no definite criterion of cure in trachoma. However, a combination of all the negative signs—quiescence of inflammation, deturgescence of the pannus, resolution of the follicles, a smooth, pink conjunctiva with or without cicatrices, absence of inclusion bodies—indicates that the disease is arrested. Without any one of these signs, the patient cannot be considered cured, even if no active lesion is present.

Thygeson (48) points out that, when confronted with the problem of distinguishing healed trachoma from trachoma of low activity, microscopic examination is helpful only in a limited way. The finding of inclusion bodies indicates trachomatous activity, but inability to find them, even on repeated examination, does not necessarily mean a lack of a trachomatous activity.

### *Is There a Worldwide Clinical Pattern?*

Although the cardinal signs of trachoma (follicle formation, pannus, cicatrization, and

the presence of inclusion bodies) are universally accepted, the disease varies considerably in different parts of the world and would therefore seem to be polymorphic. According to Lyons (49), trachoma may show variations in any of the following factors: (a) mode of transmission, (b) average age of onset, (c) clinical course and the resultant disability, (d) conditioning effect of secondary infection, (e) incidence of inclusion bodies at various stages of the disease, and (f) response to treatment with sulfonamides and antibiotics. Reports from workers in different countries illustrate some of these variations.

Lyons (50) states that in Egypt trachoma in its active stages causes surprisingly little disability; that gross thickening of lids is rare and the pannus seldom encroaches on the pupillary zone; and that photophobia and lacrimation are usually minimal and often absent. He adds that it usually is only the corneal lesions caused by neglected trichiasis in the late stage of the disease which lead to any serious reduction in vision.

Mitsui (45) of Japan declares trachoma to be an acute disease in its initial stage without exception. MacCallan (51) states that in Egypt and in eastern Europe, trachoma begins always with an inflammatory stage that continues for some time, but that in England the onset of trachoma in an acute or subacute form rarely occurs. He states further (13) that unmixed trachoma is always a chronic disease; that when it commences with acute manifestations or exhibits acute exacerbations, these are the result of superadded infections. Postic (52) of Yugoslavia observes that Mitsui's statement that all trachoma is acute in the initial stage must be taken to mean that such is the case in Japan, but not necessarily everywhere.

Gradle (6) states that trachoma in the United States varies somewhat with the characteristics of the population and the degree of sanitation present, but that entirely lacking is the onset of acute trachoma associated with ophthalmia, as seen so universally in Egypt. Lavery (53) states that in Ireland the disease usually starts insidiously, and that he has never seen an attack of acute trachoma. He reports, however, that



he has observed attacks of acute conjunctivitis superimposed upon trachoma.

In Missouri, trachoma is usually a chronic disease, progressing slowly over a long period of time; acute trachoma may occur, but only occasionally. In my experience, the disease takes from one to several months to show early clinical signs after contact with a known infection has taken place.

Birch-Hirschfeld (11) believes that the first stage of trachoma with its inflammatory infiltration passes early and imperceptibly into the stage of scarring and that the follicles are not of essential importance for the progress of the disease; that they are found both in early and late trachoma and are usually invaded and broken up by strands of fibroblasts.

The first appearance of the follicles on the conjunctiva may vary as to location. MacCallan (13) notes that in Egyptians the granulations appear first on the tarsus, but in Europeans, in the cul-de-sac (retrotarsal fold of the conjunctiva). Fuchs (12) states that papillary hypertrophy is a predominant feature of the tarsal conjunctiva, and that the retrotarsal fold is the seat of formation for trachoma granules. Among the white population of Missouri, it is in the retrotarsal fold that follicle formation is most pronounced and where the follicles first manifest themselves. When trachoma is seen in the florid form, however, a generalized follicular distribution throughout the conjunctiva of both tarsus and fornix in both the upper and lower lids is found.

In many, but not all, parts of the world, trachoma is frequently complicated with secondary infections, commonly the Koch-Weeks bacillus and the gonococcus (in epidemic and endemic form), and less frequently the Morax-Axenfeld diplobacillus and the pneumococcus. The associated infections greatly affect the clinical picture as well as the prognosis and the infectivity of the disease. Nataf (54) of Tunisia includes trachoma with secondary infections always; he believes that pure trachoma exists very rarely and that numerous infections are very often associated with it, the trachoma itself constituting the basic disease, the endemic and epidemic characteristics of which are conditioned by related disorders.

### *Do Associated Infections Affect Treatment?*

Most authorities agree that the associated infections of trachoma have a conditioning effect on treatment. Lyons (50) states that antibiotics usually produce rapid and striking improvement in secondarily infected cases and have little or no immediate effect in uncomplicated trachoma. Bietti (39) points out that sulfonamide and antibiotic treatments may give different results according to the country in which they are administered. In Egypt, for instance, where associated infections are the rule, the response to treatment by various sulfonamides and antibiotics may well depend on the conditioning effect of these infections, as well as perhaps on other factors. Pages' (55) survey of experiments has shown that the trachoma-bacteria complex is characterized by a kind of mutual stimulation on the part of the associated micro-organisms, or, in other words, a synergic attack. In the United States, where the complicated trachoma common in Egypt is seldom seen, sulfonamides have generally produced more favorable results than the antibiotics (37).

### **Summary and Conclusions**

Trachoma is a disease that may vary considerably in different parts of the world. Its clinical phase may be conditioned by climate, environment, associated infections, race, and general health and living conditions. From a worldwide point of view, therefore, both sulfonamides and antibiotics are useful in therapy, although sulfonamides have been found more effective in the United States.

There is definite need for research on the serology and immunology of this disease and on its causative agent or agents. In this connection, a more simple and more direct diagnostic laboratory test would be of infinite value in making an early diagnosis. A more clearly defined clinical picture of trachoma as it occurs in various regions of the world would make it possible to correlate better the results of chemo-antibiotic treatment. A universally accepted classification of trachoma would help clarify many questions of medical, administrative, investigational, and international character.

Although trachoma has been virtually eliminated from many areas of the world, the job of complete and universal eradication is not yet accomplished. The combined efforts of research, mass therapy, and preventive measures, however, are producing increasingly favorable results. New facts and new ideas in trachomatology are being brought to light and are being set forth in the literature. These reports help to define the disease, to recognize its variations, to make possible early diagnosis, and to determine the therapeutic methods most suitable for coping with it regionally.

The outlook today for a worldwide attack on trachoma is more encouraging than ever before, particularly because of better understanding between ophthalmologists and trachomatologists here and abroad; and because, too, of greater recognition by governmental authorities of the importance of this disease in relation to the health, economy, and welfare of the affected people.

#### REFERENCES

- (1) Duke-Elder, S.: Text book of ophthalmology. Ed. 1. St. Louis, C. V. Mosby Co., 1934, vol. 2, p. 1593.
- (2) Sorsby, A.: The task of ophthalmological research. *Brit. M. Bull.* 1: 102-104 (1943).
- (3) Moutinho, H.: Legislation internationale contre le trachome, projet d'unification mondiale. *Rev. internat. du trachome* 26: 3-65 (1949).
- (4) Siebeck, R.: Trachoma in Europe and the Near East. *In*: World-atlas of epidemic diseases. Hamburg, Falk-Verlag, 1952, pp. 79-80.
- (5) Lindner, K.: Trachoma. *In* The eye and its diseases, edited by C. Berens. Philadelphia, W. B. Saunders and Company, 1936, p. 423.
- (6) Gradle, H.: Trachoma in the United States of America. *Rev. internat. du trachome* 16: 143-145 (1939).
- (7) Cosgrove, K. W.: Trachoma problems in Arkansas. *South. M. J.* 34: 1037-1040 (1941).
- (8) Forster, W. G., and McGibony, J. R.: Trachoma. *Am. J. Ophth.* 27: 1107-1117 (1944).
- (9) Bengtson, I. A.: Question of the rickettsial nature of trachoma. *Am. J. Ophth.* 23: 770-779 (1940).
- (10) Thygeson, P.: The nature of the elementary and initial bodies of trachoma. *Arch. Ophth.* 12: 307-318 (1934).
- (11) Birch-Hirschfeld: Pathology of trachoma. *Rev. internat. du trachome* 16: 110-112 (1939).
- (12) Fuchs, E.: Text-book of ophthalmology. Duane Ed. 8. Philadelphia, Lippincott, 1924, pp. 454-456.
- (13) MacCallan, A. F.: Trachoma and its complications in Egypt. Cambridge, Cambridge University Press, 1913, p. 55.
- (14) Romero, E.: Mi experiencia en mas de cinco mil casos de trachoma. *Arch. Soc. oftal. hispano-am.* 9: 1107-1116 (1949).
- (15) Taborisky, J.: Trachoma in northern Russia and in southern Palestine. *Rev. internat. du trachome* 16: 201-208 (1939).
- (16) Ching, R.: A new trachoma concept. *A. M. A. Arch. Ophth.* 51: 750-761 (1954).
- (17) Rice, C. E., and Smith, J. E.: Trachoma in Missouri. *J. Missouri M. A.* 29: 13-17 (1932).
- (18) Julianelle, L. A., and Smith, J. E.: A statistical analysis of clinical trachoma. *Am. J. Ophth.* 26: 158-166 (1943).
- (19) Mitsui, Y.: Acute trachoma. *Rev. internat. du trachome* 29: 323-325 (1952).
- (20) Cuenod, A., and Nataf, R.: Le trachome. Paris, Masson et Cie., 1930.
- (21) Cuenod, A., and Nataf, R.: Biomicroscopie du pannus trachomateux au debut. *Arch. d'ophth.* 48: 737-742 (1931).
- (22) Nataf, R.: Le trachome. Paris, Masson et Cie., 1952.
- (23) Kuo, P. K.: Incipient pannus in early diagnosis of trachoma. *Am. J. Ophth.* 29: 645-653 (1946).
- (24) Tabone, V.: Anti-trachoma campaign in Gozo (Malta). *Rev. internat. du trachome* 29: 56-57 (1952).
- (25) Rice, C. E., Sory, R., Smith, J. E., Faed, P. E., and Drake, A. A.: Effect of diet and vitamins on trachoma. *Am. J. Ophth.* 17: 735-741 (1934).
- (26) Stucky, J. A.: Observations and experiences with trachoma in eastern Kentucky. *Tr. Am. Acad. Ophth.* 35: 217-224 (1930); *Eye, Ear, Nose & Throat Monthly* 9: 455-457 (1930).
- (27) Glickson, J.: Observations on the trachoma problem. *Am. J. Ophth.* 33: 616-617 (1950).
- (28) Murray, N. L.: Trachoma in South Africa. *South African J. Clin. Sc.* 4: 119-203 (1953).
- (29) Bietti, G. B.: Il tracoma. Rome, Sanite Publica, 1947.
- (30) Wilson, R. P.: Trachoma: A selection of personal observations and experiences. Giza Memorial Ophthalmic Laboratory, Report No. 14. Cairo, Egypt, The Laboratory, 1939-1944, pp. 24-25.
- (31) Bietti, G. B.: Le trachome; vue d'ensemble des travaux de la période de guerre et d'après guerre (Trachoma; summary of the literature of the period of the war and after the war). *Bibl. ophth. Basel* No. 58, pp. 232-333.
- (32) Bietti, G. B., and Cavara, V.: Le manifestazioni oculari delle malattie da virus e da rickettsie. Bologna, L. Cappelli, 1952, p. 184.

- (33) Rice, C. E.: Trachoma in the native white population of the United States. *Sight-Saving Rev.* 3: 105-114, (1933).
- (34) Mann, I.: Ophthalmic survey of the Kimberley Division of Western Australia. Perth, W. H. Wyatt, 1954.
- (35) Williams, R. C.: The United States Public Health Service, 1798-1950. Washington, D. C., Commissioned Officers Association of the United States Public Health Service, 1951, pp. 286-295.
- (36) Siniscal, A. A.: Trachoma in Missouri. *Arch. Ophth.* 42: 422-437 (1949).
- (37) Siniscal, A. A.: The sulfonamides and antibiotics in trachoma. *Am. J. Ophth.* 35: 671-683 (1952).
- (38) Antibiotics in trachoma—Bibliography. WHO/Trachoma/8. Geneva, World Health Organization, 1951.
- (39) Bietti, G. B.: Treatment of trachoma by various antibiotics and chemotherapy. WHO/Trachoma/17. Geneva, World Health Organization, 1951.
- (40) Fernandez, P.: Aureomycin in the therapy of trachoma. *Arch. Soc. oftal. hispano-am.* 13: 79-99 (1953).
- (41) Freyche, M. J.: Chemotherapy of trachoma. *Bull. World Health Org.* 2: 523-544 (1950).
- (42) Naccache, R.: Terramycin in trachoma. *Brit. J. Ophth.* 37: 106-108 (1953).
- (43) Mitsui, Y., and Tanaka, C.: Terramycin, aureomycin and chloramphenicol in the treatment of trachoma. WHO/Trachoma/2. Geneva, World Health Organization, 1951.
- (44) Mitsui, Y., and others: Terramycin in the treatment of trachoma. WHO/Trachoma/9. Geneva, World Health Organization, 1951.
- (45) Mitsui, Y.: Use of the new antibiotics in the treatment of trachoma. WHO/Trachoma/16. Geneva, World Health Organization, 1951.
- (46) Loe, F.: Sulfanilamide treatment of trachoma. Preliminary report. *J. A. M. A.* 111: 1371-1372 (1938).
- (47) Cosgrove, K. W.: Local use of sulfanilamide in trachoma. *Am. J. Ophth.* 23: 911-913 (1940).
- (48) Thygeson, P.: Criteria of cure in trachoma. *Rev. internat. du trachome* 30: 450-462 (1953).
- (49) Lyons, F. M.: Observations on the international coordination of research in trachoma. WHO/Trachoma/4. Geneva, World Health Organization, 1951.
- (50) Lyons, F. M.: Dual problem of acute ophthalmia and trachoma in Egypt. WHO/Trachoma/20. Geneva, World Health Organization, 1951.
- (51) MacCallan, A. F.: The initial signs of trachoma. *Rev. internat. du trachome* 27: 112-117 (1950).
- (52) Postic, S.: Comments on document WHO/Trachoma/16; "Use of the new antibiotics in the treatment of trachoma" by Dr. Y. Mitsui. WHO/Trachoma/27. Geneva, World Health Organization, 1951.
- (53) Lavery, F. J.: Incidence and clinical type of trachoma in Europe. *Rev. internat. du trachome* 16: 137-143 (1939).
- (54) Nataf, R.: Organization of control of trachoma and associated infections in underdeveloped countries. WHO/Trachoma/19. Geneva, World Health Organization, 1951.
- (55) Pages, R.: Role of seasonal acute conjunctivitis in the development of trachoma. WHO/Trachoma/7. Geneva, World Health Organization, 1951.

