The disease named above seems in the reality to be a new disease in so far as it has not been described in ophthalmic literature, although I described it myself in Geneeskundig Tijdschrift voor Nederlandsch-Indië in the year 1914. This is one of the reasons why I have waited so long a time before publishing it in ophthalmic literature. I wanted to make sure that it had not been mentioned there previously. If I am mistaken, I shall be thankful for any information.

In the first place I wish to emphasise, that this disease is quite different from the well known and well described keratitis ramificata or dendritica, as will be seen from the description in the following pages.

It seems nearly incredible, that I should have discovered a disease of the cornea unknown to other ophthalmologists and in a period where the cornea has been examined so thoroughly by means of slit-lamp and corneal-microscope, especially when I have to add, that the disease is very common in the tropics and in the summer time also in the temperate climates as for instance England and Denmark where I have also had the opportunity of
treatment. However, the greatest number of cases I have treated in Java.

The reason why it has formerly not been discovered is in the reality the perfection of the undulation of the cornea with secretion of the lacrimal glands. As known these glands are not effusing their content through a single big canal but through 6-12 fine apertures on the posterior aspect of the upper eyelid. On account of this arrangement and of the perfect way in which the secretion is controlled we never see any flow of tears in the normal eye, although all surfaces are kept humid and shining.

This is evidently the effect of capillary action and diffusion. In no part of the eye is this action more important than to the cornea which only in this way can be kept shining and pellucid. The living normal cornea is therefore always covered with a layer of lacrimal fluid. It is so constant and so necessary, that we, can say, physiologically speaking, that the cornea consists of six layers, viz., endothelium, membrana Descemeti, parenchyma, membrana Bowmani, epithelium and lacrimal layer.

This lacrimal layer has optically to fulfil two functions: (1) to keep the surface pellucid and (2) to give the surface a perfectly regular globular form. (1) needs no explanation, but how is (2) attained? In short we can say: through the combined laws of cohesion and adhesion. But I shall explain it more distinctly.

I beg to use an instance. When after an operation we pour the water or spirit out of a porcelain instrument tray, we see, that the remainder of the fluid does not form a regular thin cover on the bottom of the tray, but very quickly it forms separate drops.

The reason is, that the cohesion is stronger than the adhesion. The cohesion will cause water or spirit to form a globular body with perfectly regular, reflecting surface as we see it in the falling drop. In the instrument tray the drops are more flattened as a result of the gravity and adhesion, but yet they have a height of about half a millimetre. If the bottom under the drop should happen to have a small groove under one of the drops, 1/10 or 1/5 of a millimetre deep, this will not have any influence upon the surface of the drop which remains even and regular. In the same way the lacrimal layer on the cornea forms a regular, globular, reflecting surface, even if there should happen to be any very shallow depressions as in absence of some layers of epithelial cells or even of the whole epithelium.

This is the reason why the disease which I am going to describe in the following pages has been overlooked for so long a time.

In order to give a picture of the disease I shall describe a single case.

Mr. J. v. B., aged 50 years, pensioned official, Bandoeng, Java. Cons. January 28, 1936. For a long time much troubled in the
eyes. He cannot stand glare. The last two months blepharospasm of the left eye. Sometimes headache.

O.d. V. 5/36, with +2.0D. sph., 5/5, O.s. V. 5/36, with +2.0D. sph. 5/5.

B. has reading glasses d. and s. +3.5 sph.

Inspection of the eyes by good daylight straight inside a big window. With two fingers the right eye is kept widely open, and the patient is told to look along my right ear.

Very soon a pericorneal vascularisation takes place, and it increases quickly. The cornea is from the beginning perfectly clear and the surface all over bright and smooth as a mirror.

But suddenly a small groove is formed in the upper part. At the same moment the eye is getting irritated, and the cornea would escape upwards under the upper eyelid, if I did not ask the patient to keep it still. The groove is extending and a new one is formed; but now the patient cannot stand it any longer, and the cornea disappears under the upper eyelid.

The left eye behaves in the same way, but I succeed in keeping it in position long enough to determine that the cornea is deprived of its epithelium in the upper part as shown in Fig. 1.

Now cocaine is instilled in both eyes, 1 per cent., 2 per cent. and 4 per cent., and the patient is again examined in the same way. Then after the eyes have been kept open 1 to 2 minutes, we find that the corneas are deprived of the epithelium in an extent as shown in the Figs. 1 and 2.

The hatched parts of the drawings here and in the following pages show the extent of the disease.

The reason why these epithelial defects did not appear from the beginning has been explained above.

The irrigation of cornea is so effective, that in many cases it will be necessary to stop or diminish the stream by means of cocaine in order to have a look at the uncovered cornea. In fact it will usually be necessary, if we want a full picture of the disease to drop 1 per cent. cocaine, 1 per cent. pantocaine and 4 per cent.
cocaine before the examination. If the upper eyelid be everted and kept well back from the cornea, the picture of the disease will often develop more quickly.

In fresh cases we find pictures as shown in Fig. 3 and Fig. 4; but usually the disease is of older standing, and the branches are then becoming confluent into flats with irregularly curved limits; but often we find at the same time isolated branches spread over the surface of the cornea.

Where the epithelium is missing the surface is slightly greyish and dull, in sharp contrast with the remaining shining surface of the cornea; but we never find any real dimness; as soon as the cornea is covered with its usual layer of tears, it is again perfectly clear, and the surface has regained its regular, globular form. Probably it is often only some single layers of epithelium that are missing, for sometimes I have found one part of the affected surface one layer deeper than the other.

The extension of the disease is very different as seen from the pictures in the following. In some cases I have found it spread over nearly the whole cornea.

Distribution

The disease is rather common, especially in the tropics where I have been working the last 30 years, at first 7 years in charge of a much frequented eye clinic in Semarang, Java, later on 15 years in the same city in charge of an eye hospital with 150 beds, and finally as a private practitioner.

Since I discovered the disease in 1908 I have treated a great number of cases here in Java.

Table II will show the number I have treated in the run of 15 years with a total of 3,335 and a yearly average of 222.

For the 11 years from 1921 till 1931 I have no statistics; but if we reckon the same average, we get a number of 2,442 for these 11 years. That would give a grand total of 5,777; thus if we reckon a grand total of 5,000, we may be on the safe side.
Keratitis Ramificata Superficialis  

Also in northern countries we find this disease, especially in the warm season. During my stay in Europe in 1915 and 1922 I treated several cases in London and in different places in Denmark.

Aetiology

As to the aetiology the disease seems mainly to be due to climatic influences such as heat, glare, wind and dust. This will also explain why in the temperate climates it is mainly met with in the warm seasons. According to my experience it is not contagious. The racemose way in which it spreads, like growing, over the surface of the cornea, a circumstance which has caused me to call it "ramificata," seems to indicate some living agent; but there has not been any opportunity for bacteriological examination.

Symptomatology

The symptoms of these patients are very different. The patients will complain about pain or itching in the eyes. They cannot stand the glare. They cannot stand work with the eyes, or at any rate only for a short time. This asthenopia is a very common symptom. Sometimes we find blepharospasmus nictitans, rather often cephalalgia, sometimes epiphora and photophobia. Vertigo is less frequent.

In Table I are given the statistics of four years showing the frequency of these different sufferings, and in the following pages they will be mentioned each singularly.

Asthenopia

In fact most cases of asthenopia are caused by keratitis ramificata superficialis, and when we make a habit of examining the cornea in the way I have described, we will find that the great number of cases of asthenopia nervosa will get reduced considerably.

That here we not have to do with an accidental coincidence, is proved by the fact that the asthenopia will disappear within one or two weeks, when the keratitis is well treated.

The asthenopia is felt and described by the patients in different ways. They will often say, that when they begin to work, they can see well, but after a period of half an hour, or longer, the vision is diminishing, and it is getting misty or blurred. The vision is sometimes diminishing so much, that the patients after a certain time cannot see any longer and have to stop work. Sometimes the letters are moving, when the patients try to read. Vision and refraction have of course always been examined and the last, when necessary, corrected.
Characteristic of these cases is the great contrast between the perfectly normal vision and the severe eye troubles of the patients. Table I shows the frequency of this symptom during four years. As seen, it is rather high, 38-45 per cent.

In this connection I shall mention another symptom which more intelligent patients will sometimes describe, viz., monocular diplopia of a special description seeing that the pictures are partly covering each other and that the one is more distinct than the other as shown in Fig. 5. I have myself been able to make this observation, when I was suffering from the disease. It comes into existence when the limit of the epithelial defect of the cornea is situated in front of the pupil, and the cornea then is partly getting dry.

**Blepharospasmus Nictitans**

Blepharospasmus nictitans is here not mentioned as a separate disease but as a symptom of the keratitis, because according to my 25 years' experience we always will find in these cases the keratitis, and when this has been cured, or already while it is improving, the blepharospasmus will disappear.

Formerly I considered this disease as a rather obstinate one; but after I found its origin, it was easily cured. I am here only speaking about the real, pronounced cases, and, as seen in Table I, these are not very common; but some spasm of the eyelid, coming into appearance by the examination, is often present.

**Table I**

<table>
<thead>
<tr>
<th>YEAR</th>
<th>Kerat. ram. superf.</th>
<th>Asthenopia</th>
<th>Bleph. spasm. nictit.</th>
<th>Cephalalgia</th>
<th>Epiphora</th>
<th>Phosphobia</th>
<th>Vertigo</th>
</tr>
</thead>
<tbody>
<tr>
<td>1932</td>
<td>283</td>
<td>128</td>
<td>2</td>
<td>24</td>
<td>4</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>1933</td>
<td>400</td>
<td>163</td>
<td>6</td>
<td>58</td>
<td>14</td>
<td>9</td>
<td>5</td>
</tr>
<tr>
<td>1934</td>
<td>381</td>
<td>149</td>
<td>8</td>
<td>64</td>
<td>11</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>1935</td>
<td>235</td>
<td>90</td>
<td>10</td>
<td>26</td>
<td>16</td>
<td>17</td>
<td>4</td>
</tr>
</tbody>
</table>

**Cephalalgia**

Cephalalgia is a rather common symptom as seen at the Table I, and it has not seldom been this symptom that has caused the patient to seek ophthalmic treatment, because it was combined
with some eye troubles. Usually it was evidently caused by the keratitis and disappeared with treatment.

It was present in 85-193 per cent. of the cases in Table I, and it was usually localised to the forehead.

**Epiphora**

Real epiphora was not often present, but very often the eyes of these patients watered more easily than normal eyes.

**Photophobia**

Real, pronounced photophobia was also rather rare; but usually the eyes were much more sensitive to light than normal eyes. This is of course a great hindrance in the examination and often makes the use of cocaine and pantocain necessary.

In some cases the photophobia has been the main symptom. I remember especially one case. The patient was a landowner from the mountains in Preanger in West Java. His photophobia was so strong, that it was perfectly impossible for him to control his grounds and his workers. He had been under the treatment of several colleagues for a long time, but without any improvement. He was just on the point of giving up his position, when he, as a last effort, came to me in Semarang in middle Java. It appeared to be a severe case of keratitis ramificata superficialis, and he consented to stay in Semarang about 5 weeks for treatment.

After the expiration of this period he was perfectly cured and could return home and take up his work.

**Vertigo**

As seen in Table I vertigo was not often present, and it was never in a heavy degree. That it was due to the keratitis appeared from the fact that it disappeared together with this disease.

**Treatment**

In the first place the eyes must be protected against glare, wind and dust. For this purpose the patients must wear dark spectacles, when they are outdoors. The glasses need not be too dark Crookes' B. or middle dark smoke are suitable. At the same time they must keep as much as possible indoors and for a time give up sport, especially swimming.

When suffering from asthenopia, they must give the eyes the necessary rest; thus when reading or writing they must stop work for some minutes or as long as necessary and then begin again. In very heavy cases only will it be necessary to stop work for some days, or longer. Smoke must be avoided. The patients
may well smoke tobacco outdoors, but may not stay in rooms with tobacco smoke.

For treatment at home fluorescin is the best remedy in the following solution: fluorescini milligram 400, carb. cal. 200, s. in sol. sublimati 1-10,000, grm. 20, d.s. eyedrops.

Fluorescin may not be applied seeing that it is somewhat irritating and has not the same therapeutic effect as fluorescin.

Seeing, that this remedy has a very strong staining effect, the patients must get special instructions about the use of it. A humid tampon must be ready on the table, the patient must turn the head well backwards, the eye is kept widely open with two fingers, and only one drop is put just on the middle of the eye. Then the tampon is put on top of the closed eye without rubbing. In this way the superfluous liquid will get absorbed by the wadding; otherwise it would stain the face. For social reasons this precaution is very important.

This instillation must take place 5 times a day. It is perfectly painless. When this treatment is continued for a long time a great improvement will usually take place; the most troublesome symptoms will diminish or disappear, and the patient will often be satisfied.

However the disease will not get fully cured in this way. For this purpose a stronger, local treatment will be necessary. Formerly I used sol. nitr. arg. 1 or 2 per cent. for brushing of the cornea or instillation of one drop only directly on the cornea after cocaine instillation.

After this treatment the patients would get rather severe pain against which I used dionine, seeing that cocaine is very hurtful in this disease. This treatment was repeated three times a week. The effect was rather good, but it was too troublesome for the patients. It was therefore a real relief for these patients as well as for myself, when I found, that antigonococcus serum also in these cases had the same effect as sol. nitr. arg. without causing any pain worth mentioning. The patients first get two instillations of 1 per cent. cocaine and then two instillations of the serum. This procedure is repeated three times a week for a period of 1-2 months.

Sometimes a local anaphylaxis will develop after 2-3 weeks. The eyes will then get red and sore after the instillation and keep so some few days. The therapeutic effect is so much stronger, but still the serum treatment must be stopped for about a week and replaced by electrargol instillations. I have used this treatment 8-9 years.

The antigonococcus serum which I have used through all these years I have got from Parke, Davis and Co., Detroit, Michigan, U.S.A.
KERATITIS RAMIFICATA SUPERFICIALIS

The firm informs me, that the serum is obtained from the blood of horses that have been treated with gradually increasing doses of live cultures of gonococcus. The activity of the serum is controlled by agglutination tests, so that a uniformly potent product is assured. Each lot is subjected to rigid bacteriological and physiological tests, both while in bulk and also after enclosure in the final container. The serum is served out in glass bulbs, closed by a rubber cap, each containing two c.c. The bulb must be opened aseptically, and the pipette that has to be used must also be sterilised.

A handy way to keep the bulb is to wrap it up in a slip of wadding, and put it into an ointment pot of 15 c.c. The pipette must have a point 33 mm. long, or the point must be bent so much that it can get to the last drop of serum, when the bulb is inclined. After the bulb has been opened, the serum will keep sound for one or two days in the tropics, and longer in a cooler climate.

Last year the same firm produced a new serum, called gonococcus antitoxin, containing a stronger antitoxin and less albumen. Dr. T. Anwijl-Davis has tried this new remedy in 157 different cases of gonorrhoeal infections with very promising results.

He describes these cases in the British Medical Journal, February 13, 1937, under the title of "Treatment of gonorrhoea with a specific antitoxin." According to the wish of Parke, Davis and Co., Detroit, I have tried it in the cases of eye diseases which I have hitherto treated with the old serum. It seems to have a similar effect; but it must be diluted with half its volume of distilled, sterile water, otherwise it is painful. The time has been too short to form a definite opinion, but my confidence is increasing.

Besides the serum treatment the patients use the fluorescin at home and wear a dark spectacle outdoors.

Usually the patients can continue their daily work. Only exceptionally they must have a short holiday of some few days or a week, very seldom longer.

In the final examination we may not use cocaine, seeing that it is hurtful for the corneal epithelium. I keep the eye widely open while counting slowly to 25. If the eye stands the glare well, and no epithelial defect appears, the patient is dismissed from the treatment in the eye clinic; but he must continue the treatment at home.

One month he must instil fluorescin four times a day and the second month twice a day. During these two months he must regularly wear the goggles outdoors but after this period only if he has to walk or drive a longer distance against glare and wind.
Case Histories

In the following I am going to describe some instances of patients suffering from this disease. In the accompanying drawings the hatched part will show, in how great extent the cornea was affected with the disease.
THE BRITISH JOURNAL OF OPHTHALMOLOGY

CASE 1.—Mr. Th. J. B., aged 30 years. July 11, 1927. Asthenopia, solar and for reading. Keratitis ramificata superficialis, both sides rather severe. Astigmatism. For about 10 years he has difficulties with his eyes. He has very straining eye work. He can only with difficulty perform his work and gets headache. He sleeps badly. He has suitable glasses, correcting his astigmatism. Vision with corr. o.d. 6/6, o.s. 6/6. Figs. 6 and 7 show the state of the corneae. August 10 he entered the eye-clinic and commenced treatment. Rp. fluorescin 5, 'Crookes' glasses B, 3 times a week brushing of the cornea with 2 per cent. sol. nitr. arg.

September 7, 1927. Formerly the patient was much troubled with headache, but the last two weeks it has perfectly disappeared. Sleep is more quiet than it used to be. He resumes his work, wearing dark spectacles outdoors and dropping into the eyes fluorescin 3 to 4 times a day.

November 4, 1927. Sometimes a little headache, when he has been working harder than usual, otherwise all right.

CASE 2.—Miss J. A. M., aged 34 years. October 19, 1927. Asthenopia. Ker. ram. superf. both eyes, rather severe. Is quickly tired by reading, and then she gets a sensation above the eyes, sometimes already at 1 o'clock in the afternoon, but usually not until the evening. Then she gets a strong feeling of sleepiness and must sometimes blink. The patient cannot stand strong light. R. emm. 6/6, L. emm. 6/6. When covering left eye she sees the letters double, the one picture partly covering the other and less distinct than this (Fig. 5). Fig. 8 will explain this phenomenon. As seen a branch of the disease is so near the centre of the cornea, that the edge of it will sometimes be in front of the pupil and will then break the rays of light in an irregular way. The usual treatment. March 2, 1928. The weariness of the eyes has disappeared and there is no blinking any more.

CASE 3.—Mr. P. M., aged 44 years. April 23, 1928. During 2 months headache in the parietal region especially in the evening and night. The pain increases with reading, and when the patient has been reading a while, he gets sleepy. He cannot stand glare and is forced to wear dark spectacles.

R. emm. 6/6 |
L. emm. 6/6 |

With R. + 1.0 sph. | 0-45 in 45 ctm. distance.
L. + 1.0 sph. | 0·35 in 30 ctm. distance.

Patient gets a pair of spectacles with this correction. Figs. 9 and 10 show the state of the corneae. The usual treatment. June 11, 1928. No headache any more. The eyes stand the glare quite well.

CASE 4.—Mr. I. J. E., aged 27 years. June 29, 1928. Asthenopia, Hyperm. Astigm. Ker. ram. both sides severe. When working in the evening he gets a pricking, burning pain in the eyes and then the vision sometimes gets blurred. He has a correct pair of spectacles and normal vision. The state of the corneae is shown in Figs. 11 and 12. He gets the usual treatment.

July 30, 1928. Patient has much less difficulty with his eyes. October 10, 1928. The eyes feel quite normal.

CASE 5.—Mrs. Gr., aged 63 years. April 6, 1933. Photophobia. Ker. ram. superf. both sides severe. Has much trouble from the glare. Extension and form of the disease is shown on the two Figs. 13 and 14. Rp. fluorescin x 5, dark spectacles, instillation of antimonococci-serum, Parke, Davis & Co., Detroit, every second day. May 4, 1933. Can stand the glare much better.

May 22, 1933. Has been away one week on account of disease.


CASE 6.—Mr. Ch. O. van der P., aged 43 years. June 19, 1934. Asthenopia, Cephalalgia. Ker. ram. s. severe in right eye, very severe in left. When the patient is getting tired, he cannot see well. He is suffering much from headache. The eyes are red. Figs. 15 and 16 show the state of the cornea, R. emm. 8/5, L. emm. 8/5, 0·35 in 30 ctm. He gets the usual treatment, but can only visit the clinic from time to time. Seeing that he is driving much in an open motor-car, he gets a pair of dark spectacles with side covering for this purpose.
July 16, 1934. Patient can see well. No trouble from the eyes any more, not even when on tour. There is still some keratitis.

October 5, 1934. No headache or trouble from the eyes which are perfectly cured.

Case 7.—Raden N., aged 8 years, boy. May 29, 1935. Asthenopia severe, Ker. ram. s., both sides severe. The patient is nervous. At school, when writing the letters are at first good, but very soon they get very bad. His marks are bad for writing. Figs. 17 and 18 show the state of the corneæ. R. 5/5, L. 5/5, 0·35 in 30 ctm.

July 6, 1935. Patient is cured. The writing at school much better.

Case 8.—Mrs. E. K., aged 35 years. October 23, 1935. Cephalalgia, Asthenopia Epiphora. Ker. ram. s. severe. Burning feeling in the eyes, they are watering. Often headache. When she is reading, the eyes are quickly tired. R. H. 0·5, 5/5, L. emm. 5/5, 0·35 in 30 ctm. The state of the corneæ is shown in Figs. 19 and 20. She gets the usual treatment and is cured November 18, 1935.

Case 9.—Mr. A. A. L. G., aged 57 years. November 29, 1935. Vertigo, Keratitis ram. s., severe both sides. Pteryg. incip. on each side. During 8 months the patient is suffering from giddiness and has a feeling of weariness in the eyes. Some twitching in the eyelids. Urine clear without albumen or sugar. The state of the corneæ is shown in Figs. 21 and 22. Patient got the usual treatment.

November 28, 1935. The corneæ are cured. The "heads" of the pterygia have withered. The giddiness has diminished very much and disappeared after a short time by the use of br. amm.

Case 10.—Mr. J. J., aged 45 years. January 23, 1936. Asthenopia for reading. Keratitis ram. s. For 8 years he has difficulties with his eyes. When he has been reading 5 minutes, he cannot see anything any more. He has a pair of spectacles; but they do not help him. In the evening it is still worse. R. 6/8, L. 6/8. There is some little astigmatism, but the patient does not want any correction. He has suitable reading glasses. Figs. 23 and 24 show the condition of the corneæ. He gets the usual treatment. The condition of the eyes is improving rapidly, and March 18, 1935, he is dismissed as cured.

Case 11.—Mr. J. B., aged 55 years. January 28, 1936. Hypermetr. Presbyopia, Blepharospasm nictitans left eye. Asthenopia, Ker. ram. s. severe both sides. The patient has much trouble from his eyes and cannot stand the glare. The last two months he has suffered from blepharospasmus nictitans of the left eye. He often has headache. He cannot stand his office work. R. 5/36, with + 2·0 D. sph. 5/5, L. 5/36, with + 2·0 D. sph. 5/5. He gets this correction with Crookes' B. For reading he has + 3·5 D. sph. for both eyes. V. 0·35 in 30 ctm. The patient must stop his office work. He gets the usual treatment. Figs. 25 and 26 show the state of the corneæ.

February 8, 1936. The blepharospasm has diminished very much.
February 29, 1936. Much improved. There is still a little keratitis.
March 4, 1936. To-day he has done his office work without any difficulty. No blepharospasm any more.
March 19, 1936. The keratitis is cured. He feels quite well.

Case 12.—Miss W. J. S., aged 20 years. November 20, 1936. Cephalalgia, photophobia, Ker. ram. sup. both sides, severe, scotoma scintillans. When working (typing) patient gets headache, and she cannot stand the glare. Rather often scotoma scintillans. The state of the corneæ is shown in the Figs. 27 and 28. The usual treatment.

November 30, 1936. No headache any more.
December 11, 1936. The patient states spontaneously that she has no trouble with her eyes any more.

Case 13.—Mr. L., aged 35 years. November 30, 1936. Photophobia. Cephalalgia. Ker. ram. sup. severe. Cannot at all stand the glare and wears dark spectacles. Is suffering from headache in the forehead. A feeling of pressure in the eyes. When looking at the test types on the wall with left eye only, he sees a double picture as a shadow, partly covering the original. When looking at Fig. 29, we see, that the border of the epithelial defect is just passing

Pterygium

The number of cases of this disease we will find in the Tables II and III, but the last gives only the cases that have been operated upon.

The reason why it is mentioned here is, that there seems to be a strong connection between the two diseases. After my attention had been fixed upon this matter in the year 1918, I have examined the cornea in the way described above in all cases of pterygium. That means in the years 1919, 1920, 1932, 1933, 1934 and 1935, 172 cases. From 1921 and 1922 I have no statistics and for the years 1923-1930 both inclusive, I have only the operated cases, altogether 73. This makes a total number of 245.

In all these cases without one single exception I have found on the cornea of the affected eye keratitis ramificata superficialis in a heavy degree.

And in nearly all cases with some very few exceptions only the pterygium was in touch with the affected part of the cornea.

**Table II**

<table>
<thead>
<tr>
<th>Year</th>
<th>Kerat. ramif. superf.</th>
<th>Pterygium</th>
</tr>
</thead>
<tbody>
<tr>
<td>1909</td>
<td>207</td>
<td>22</td>
</tr>
<tr>
<td>1910</td>
<td>250</td>
<td>22</td>
</tr>
<tr>
<td>1911</td>
<td>164</td>
<td>29</td>
</tr>
<tr>
<td>1912</td>
<td>44</td>
<td>25</td>
</tr>
<tr>
<td>1913</td>
<td>154</td>
<td>33</td>
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<tr>
<td>1915</td>
<td>166</td>
<td>13</td>
</tr>
<tr>
<td>1916</td>
<td>241</td>
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<tr>
<td>1917</td>
<td>208</td>
<td>29</td>
</tr>
<tr>
<td>1918</td>
<td>225</td>
<td>38</td>
</tr>
<tr>
<td>1919</td>
<td>179</td>
<td>51</td>
</tr>
<tr>
<td>1920</td>
<td>197</td>
<td>71</td>
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<td>1932</td>
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<td>8</td>
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<td>1933</td>
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<td>12</td>
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<td>1934</td>
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<td>15</td>
</tr>
<tr>
<td>1935</td>
<td>235</td>
<td>15</td>
</tr>
<tr>
<td>Total</td>
<td>3,334</td>
<td>405</td>
</tr>
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</table>
When the keratitis was treated in the way described above I found that after a time the "head" of the pterygium was withering away, and it stopped growing.

Several small pterygia were cured in this way without operation.

At the same time I observed that an effective treatment of the keratitis was the best way to prevent relapse of the pterygium.

These different facts give the impression, that the pterygium is caused by the keratitis, and that we here in reality have to do with a sort of natural healing in that the conjunctiva bulbi is trying to cover the denuded parts of the cornea, but—alas—not always with the best result.

The cure consisted in the usual operation combined with the treatment of the keratitis. The very small pterygia only were cured without operation.

In the following I am going to describe some cases of pterygium. The figures show the distribution of the keratitis in the same way as in the other diagrams.

**Case 1.**—O. K. H. April 4, 1923. Pterygium. Ker. ram. super., severe. Figs. 30 and 31 show the state of the cornea. He gets instillation of sol. nitrat. arg. 1 per cent., fluorescin, and protargol.


June 9, 1923. Op. pterygii. Right. After a treatment of about three months he was perfectly cured.

**Case 2.**—Mr. A. J., aged 28 years. October 26, 1923. Small recurrent pteryg. on right side, medium sized on left. Ker. ram. superf. severe on right and worse on left side. The state of the left cornea is shown in Fig. 32. He was treated with instill. of sol. nitr. arg. 1 per cent., fluor. and prot. 20 per cent.

November 6, 1923. Oper. pteryg. Left eye. When he went home after about one month of treatment, he was nearly cured.
CASE 3.—S. H. P., Javanese, aged 25 years. October 15, 1925. Ker. ram. s. severe both sides, double pterygium. The state of the corneae is shown in Figs. 33 and 34. Rp. fluor. x 4; dark spectacles, coc. instill. sol. nitr. arg. 1 per cent. protargol 20 per cent., dionine. October 8, 1925. Oper. pteryg. Right eye. The left eye was not operated upon. The small pterygium withered away during the medical treatment of the cornea. October 21, 1925. Cured, going home.

CASE 4.—F. S. H., aged 49 years. August 9, 1926. Asthenopia. Pterygium both eyes. Keratitis ramificaata superf. very severe both sides. R. Emm. 5/6, L. Emm. 5/6. The state of the right cornea is shown in Fig. 35. Rp. coc. 2 per cent. x 2, instill. nitr. arg. 1 per cent., protargol 20 per cent. and later on coc. 1 per cent. x 2, instill. of antimonococcic serum. Dark spectacles. September 2, 1926. Oper. pteryg. R. eye. September 9, 1926. Oper. pteryg. L. eye. October 5, 1926. Cured. Going home. Cont. fluor. and dark spectacles 2 months.

CASE 5.—Mr. F. B. W., aged 43 years. October 3, 1927. Asthenopia severe. Pterygium R. side. Ker. ram. severe both sides. Malaria chron. The state of the right cornea is shown in Fig. 36. When the patient in the evening is working, the eyes get hot, and begin to water. It has been like that for about a year. He has R. & L. 1-0 D. sph. He cannot stand the glare, especially on the lime-covered roads. The spleen reaches the curvature. Four years ago he had a fit of tertian malaria. Rp. decoctum foliarum Blumeae balsamiferae No. 7 ad remanentiam colatursae 750 c.c. The whole lot must be taken in the run of a day and a night. Fluor. x 4, dark spectacles, instillation of serum antigon. October 10, 1927. The spleen is normal in size (12-5 ctm. shorter than October 3, 1927. Cont. decoct. fol. Blum. bals., 6 leaves. Patient feels much better. October 17, 1927. Patient stands the glare much better. October 31, 1927. Formerly he would get headache if he had to work in the evening, but at present none. His appetite has increased very much. The eyes stand examination much better. November 15, 1927. Fully cured. The pterygium has atrophied.

CASE 6.—Mr. T. I. D., aged 27 years. June 25, 1928. Pterygium both eyes. Ker. ram. sup. severe both sides. The state of the corneae is shown in Figs. 37 and 38. R. V 5/5, L. V 5/5. Patient is suffering from chronic malaria and is treated with quinine. The spleen is a little enlarged. Rp. instillation of antigon. serum, dark spectacles, fluor. x 3.


Conjunctivitis Aestivalis

The reason why this disease is mentioned in this connection is that it is often found together with ker. ram. sup.

Table IV will show how often this combination has taken place in the years 1932-1935. But still there is a much greater amount of keratitis without conj. aest., and on the other side there is a certain number of cases of conj. aestiv. without keratitis. Under these circumstances it seems to be most rational to consider the two diseases as being independent of each other. The frequent coincidence can be explained by the partly common aetiology, viz., glare, heat and dust. On one point however there is a decided difference in the aetiology seeing that the conjunctivitis will only develop when there is a deficiency in the calcium metabolism and the production of parathyroidine.
KERATITIS RAMIFICATA SUPERFICIALIS

Consequently these two substances have a great curative effect in this disease, the first especially in the form of calcium phosphate.

**Table IV**

<table>
<thead>
<tr>
<th>Year</th>
<th>Kerat. ram. s. only</th>
<th>Conjunct. aestival. and ker. ram. s.</th>
<th>Conjunct. aestival. only</th>
</tr>
</thead>
<tbody>
<tr>
<td>1932</td>
<td>185</td>
<td>98</td>
<td>8</td>
</tr>
<tr>
<td>1933</td>
<td>292</td>
<td>108</td>
<td>8</td>
</tr>
<tr>
<td>1934</td>
<td>273</td>
<td>108</td>
<td>9</td>
</tr>
<tr>
<td>1935</td>
<td>182</td>
<td>53</td>
<td>10</td>
</tr>
</tbody>
</table>

**Summary**

The author has formerly only once published a description of the eye disease here described in a medical periodical in the Dutch East Indies in the year 1914.

In ophthalmological literature it has never been described either by the author, or by anyone else, as far as is known.

It is exclusively a disease of the epithelium. In fresh cases we find a branch formed figure on the surface of the cornea, as if it had been carved out with a small hollow chisel. By and by the figures join up and in this way arise more or less extensive epithelial defects.

The reason why the disease has so long avoided discovery is, that the living cornea is always covered with a thin layer of lacrymal fluid which layer according to the laws of coherence and adherence will always have a globular form and a regularly reflecting surface just like a drop of water. For examination it will consequently be necessary to keep the eye of the patient widely open until this lacrymal layer has evaporated. For this purpose it will often be necessary to instil cocaine, or still better pantocain repeatedly previously to the examination.

The description of the treatment must be read in extenso.

Special attention is given to the connection between the described eye disease and pterygium. Seeing, that the author since he 20 years ago began this examination has found a high degree of keratitis in all cases of pterygium, and seeing, that the head of the pterygium will always wither away, when the keratitis is well treated, he comes to the conclusion, that the pterygium is caused by the keratitis.
The connection between the keratitis and conjunctivitis aestivalis is also considered. The conclusion of the author is, that they are two separate diseases.

The fact that they often are found together, is explained by the fact, that they have a partly common aetiology.

**INTRA-OCULAR TUMOURS**

**by**

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In practice, intra-ocular tumours are vaguely classified as gliomata if they originate in the retina, or as melanomata if they have originated in the choroid and contain brown pigment that does not give the iron reaction. Those attempting greater precision place most of the gliomata in the more restricted group of retino-blastomata. The problem, however, is not so simple, for on closer examination a more detailed grouping becomes necessary.

Tumours occurring in other parts of the body have been classified with varying degrees of success, according to the development of the tissue of the part. Similarly, the classification of intra-ocular tumours can be based on retinal and choroidal development. In the present paper a series of 60 intra-ocular tumours is reviewed and a classification is attempted on a developmental basis.

**Material**

In the earlier stages the eyes were fixed in 10 per cent. formol saline, frozen with carbon dioxide snow, cut antero-posteriorly through the optic nerve, and then prepared according to the paraffin method. Later Helly's fluid was employed as the fixative, and the eyes were cut after fixation as for the previous method. By this latter procedure the inner portions of the eye did not collapse since the vitreous had coagulated during fixation. The sections were stained with haemalum and eosin, and these stains proved adequate in most cases.

According to some workers all neurological material must be prepared by complicated stains and impregnation methods. However, the haemalum and eosin appearance of the various cells in neural development have been described repeatedly, and although