

Ulceration of the cornea / by Angus Macmillan.

Contributors

Macnab, Angus, approximately 1876-1914.
University College, London. Library Services

Publication/Creation

London : Balliere, Tindall and Cox, 1907.

Persistent URL

<https://wellcomecollection.org/works/xzpz2mj>

Provider

University College London

License and attribution

This material has been provided by This material has been provided by UCL Library Services. The original may be consulted at UCL (University College London) where the originals may be consulted.

Conditions of use: it is possible this item is protected by copyright and/or related rights. You are free to use this item in any way that is permitted by the copyright and related rights legislation that applies to your use. For other uses you need to obtain permission from the rights-holder(s).



Wellcome Collection
183 Euston Road
London NW1 2BE UK
T +44 (0)20 7611 8722
E library@wellcomecollection.org
<https://wellcomecollection.org>

ULCERATION
OF THE CORNEA

ANGUS MACNAB

No. 4806 | 14

180



THE INSTITUTE
OF
OPHTHALMOLOGY
LONDON

EX LIBRIS

2809394871

OPHTHALMOLOGY HC467 MACNAB



Digitized by the Internet Archive
in 2014

<https://archive.org/details/b2128703x>

ULCERATION OF THE CORNEA

ULCERATION OF THE CORNEA

BY

ANGUS MACNAB, B.A., B.Sc., M.B., CH.B., F.R.C.S.

CHIEF CLINICAL ASSISTANT, ROYAL LONDON OPHTHALMIC HOSPITAL



LONDON

BAILLIERE, TINDALL AND COX

8 HENRIETTA STREET, COVENT GARDEN

1907

REPORT
OF THE GOVERNMENT

OF THE GOVERNMENT

OF THE GOVERNMENT



AN

MEINEN HOCHVEREHRTEN LEHRER

PROFESSOR TH. AXENFELD

DIREKTOR DER UNIVERSITÄTS-AUGENKLINIK

FREIBURG-IM-BREISGAU

P R E F A C E

THE author has attempted in the following pages to state the present position of our knowledge regarding corneal ulcers.

His thanks are due to Mr. Lister, Prof. Wintersteiner, Dr. Bronz, and Prof. Axenfeld for the use of sections. To Geheimrat Saemisch for reading and approving of the translation on p. 30 from *Das Ulcus Serpens*. To Dr. Zur Nedden for permission to use Fig. 16, and to Prof. Axenfeld for the drawings from which Figs. 13, 14, and 19 were made. To Messrs. Weiss for the use of blocks 2, 3, and 7.

A. M.

31 NEW CAVENDISH STREET,
LONDON, W.

INTRODUCTION

THE importance of the integrity of the cornea requires no emphasis. The fall in the economic value of the individual, due to any loss of transparency in that small area, is so out of proportion to the actual damage done, that it makes the cornea one of the most important structures in the whole body.

Although our knowledge of corneal ulceration is far from complete, the importance of the subject appears sufficient to justify an attempt to collect together the facts which have been ascertained, and to classify them on some scientific basis. It will readily be apparent how scanty our knowledge is, and how much work remains to be done. While so many observers appear willing and able to collect information on this subject, as evinced by the papers appearing in the medical press, it is to be regretted that no small proportion of the energy thus available is expended on unnecessary repetition of observations which require no further confirmation. Were it possible to direct this energy towards the solution of those problems which still remain obscure, substantial progress might result.

Our knowledge of corneal ulcers being very patchy,

the natural result follows that this work will be fairly complete concerning some subjects, and extremely vague and indefinite about others. An attempt has, however, been made to indicate the full range of the subject, and to render our ignorance as obvious as our knowledge.

The cornea is fortunately situated so that any change in its condition can be readily seen and accurately studied. The accumulation of facts is thus rendered easy; but until a rational basis is discovered on which such clinical phenomena can be classified, investigations appear to increase rather than decrease the complexity of the subject.

In recent years many excellent researches have been carried out to determine the pathology of the destructive lesions of the cornea. The results of these have been to compel us to give up the old classification of ulcers from their most obvious features, such as the presence of a hypopyon, the tendency to perforate, or the association of a conjunctivitis, and to group them according to the special organisms found to be present. This bacteriological classification has the great advantage over any other in that it gives us an excellent test of identity, and that it brings together those conditions which result from the action of the same cause.

Any practical method of treatment must depend on a diagnosis which can readily be made. A complete and thorough bacteriological examination of every corneal ulcer is not always practicable; the clinical

features, however, of the various bacteriological classes are sufficiently constant to be used as a means of determining the classification in most instances, and in doubtful cases the question can almost always be settled by staining and examining a film—a proceeding well within the power of all. Thus we shall have a bacteriological classification of corneal ulcers in general, but the diagnosis of any particular case will be made from its clinical aspects, and only confirmed by a thorough bacterial examination in a proportion of doubtful cases, which will decrease as our experience with this new classification extends.

Whether the practical utility of adopting a new classification be great or small, it certainly tends to a more accurate and scientific knowledge in a department of ophthalmology which must be admitted to be in a very unsatisfactory condition.

There are several forms of ulceration of the cornea which cannot be classified bacteriologically, in which the exact species of organism present seems immaterial: these have been arranged under separate headings.

I shall not give the exact references to all the articles, either clinical, pathological, or bacteriological, which have been consulted. It is impossible for me, however, to omit definitely recognising the admirable work of Saemisch, Fuchs, and Axenfeld with regard to diseases of the cornea. Our knowledge would indeed be scanty but for their labours.

The arrangement which will be adopted in this work is as follows :—

- CHAPTER I. EXAMINATION.
- CHAPTER II. *A.* TRAUMATIC ULCERATION.
- CHAPTER III. *B.* PRIMARY CORNEAL INFECTIONS.
- I. PNEUMOCOCCAL ULCER.
 (Typical Hypopyon Keratitis.)
- CHAPTER IV. II. ATYPICAL HYPOPYON KERATITIS.
1. Streptococcal Ulcer.
2. Pyocyaneus Ulcer.
3. Ulceration due to Friedländer's
 Bacillus.
4. Hypopyon Keratitis due to
 Bacterium Coli Commune,
 Staphylococcus Pyogenes,
 Rosa Hefa, Hay Bacillus, etc.
5. Keratomycosis Aspergillina.
- CHAPTER V. III. ULCUS CORNEAE RODENS
 (MOOREN'S).
- CHAPTER VI. *C.* SECONDARY CORNEAL INFECTIONS.
- I. DIPLOBACILLARY CORNEAL ULCER.
- II. INFECTIOUS MARGINAL ULCER
 (ZUR NEDDEN'S).
- CHAPTER VII. III. ULCERS OCCURRING IN ACUTE
 CONJUNCTIVITIS.
1. Koch-Weeks, Pneumococcal,
 Streptococcal, and Staphylo-
 cocal Conjunctivitis.
2. Diphtheria of the Conjunctiva.
3. Gonorrhœa of the Conjunctiva.
- CHAPTER VIII. IV. ULCERATION IN KERATITIS ECZE-
 MATOSA.

INTRODUCTION

xiii

CHAPTER IX. *D.* ULCERATION DUE TO TROPHIC DISTURBANCES, DESSICATION, OR DEGENERATIVE PROCESSES.

I. KERATITIS NEUROPARALYTICA.

II. KERATITIS E LAGOPHTHALMO.

III. KERATOMALACIA.

IV. HERPES CORNEAE, DENDRITIC
ULCER.

APPENDIX—CHAPTER X. BACTERIOLOGY.

CHAPTER XI. OPERATIONS.

Excision of Sac.

Saemisch Section.

Corneal Plastic Operation.

Removal of Material from an Ulcer.

ULCERATION OF THE CORNEA

CHAPTER I

METHODS OF EXAMINATION

THE method of examining the cornea which gives the most information is by inspection; in fact it is the only one which yields results worthy of consideration. As, however, the diameters of the cornea are only 11 mm. in the vertical, and 11.5 in the horizontal direction, our inspection must be of almost microscopical minuteness, and to this end we utilise several aids to observation without which diagnosis would be, in many cases, difficult, if not impossible. I propose, therefore, to describe first these aids to accurate observation. Patients are generally examined in a bright diffuse daylight, when of course the illumination of the cornea is not fully under our control; the improvement obtained by regulating the direction and intensity of the illumination, when examining any transparent body, is obvious at once to any one who has ever looked through a microscope. The advantage of *focal illumination* is obtained by placing the patient in a dim light, so that

the eye is only illuminated from one side, and then concentrating the rays of light on to the cornea by means of a convex lens of from 12-14 dioptries.

A small area is thus intensely illuminated, at the same time that the region immediately around is darkened (Fig. 1); any change in transparency in the

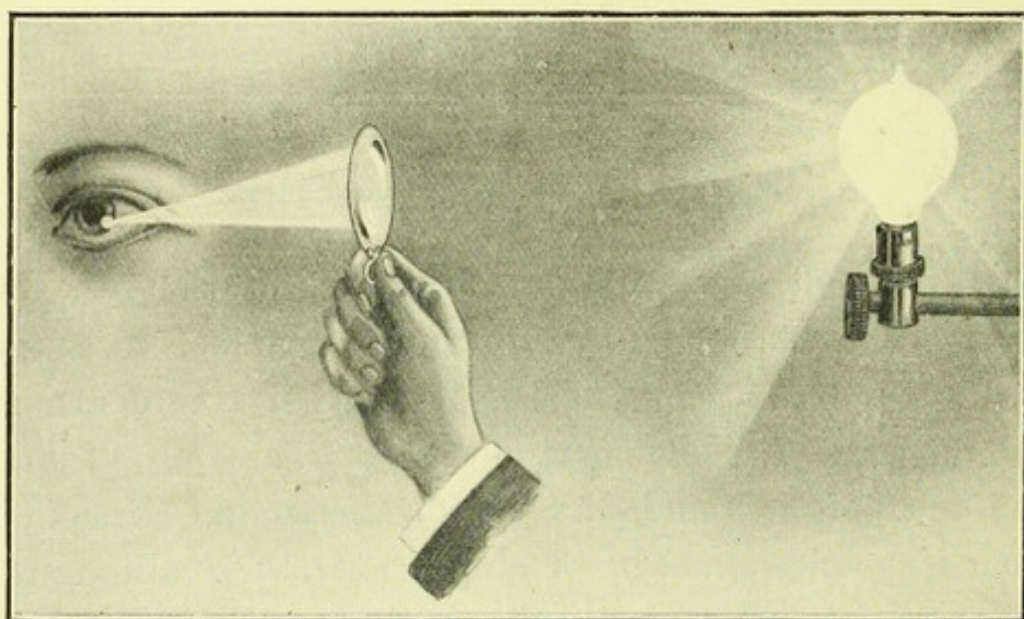


Fig. 1.

SKETCH OF ARRANGEMENT OF FOCAL LIGHT.

part thus lighted up is readily seen, and by moving the condensing lens to or from the eye various depths can be illuminated, and the position at which the changes occur estimated. In Priestly Smith's candle-lamp we have this arrangement in a very portable form; a candle is enclosed in a metal tube, at the bottom of which is a pad on the end of a spiral spring: this so compensates for the burning of the candle that the flame is always kept at the same place in the tube. On

one side of the light a lens is let into the cylinder, and opposite this is a reflector; thus a cone of light is produced (Fig. 2). When in use the lamp is brought nearer to, or removed further from the cornea, and thus the different levels can be illuminated. By holding the lamp at the side of the patient's head every part of the cornea can be successively brought into the brilliantly illuminated apex of the light cone, and details otherwise invisible become surprisingly obvious. This method has the further advantage of allowing us to use the *corneal loupe* along with a lateral focal illumination.

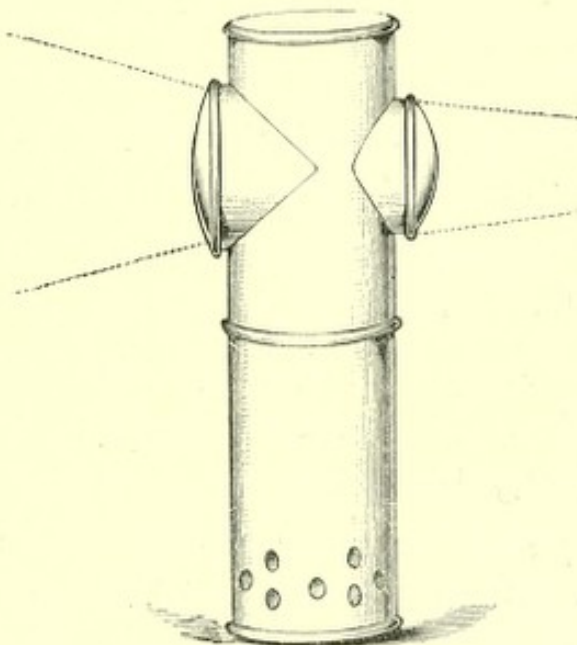


Fig. 2.

PRIESTLY SMITH LAMP.

There are many forms of corneal loupes, varying from the simple biconvex lens to the beautiful binocular, electrically

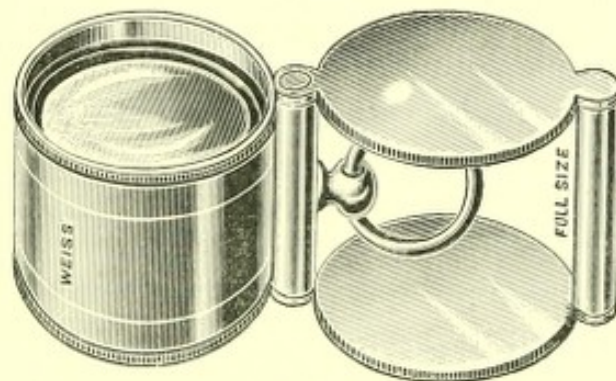


Fig. 3.

HARTNACK LOUPE.

lighted, corneal microscope. For ordinary work the most useful are the monocular Hartnack loupe (focal

length, .25 cm.; magnification, x10) Fig. 3, and the binocular. The former is made by all reputable opticians, is of small size, gives a sufficiently large magnification, and is very convenient in actual use. The binocular instruments are larger, and slightly more difficult to manipulate, but have the great advantage of showing everything in relief. They are excellent for deciding the depth of infiltrates or of new vessels in the cornea.

Fluoresceine is largely used in the diagnosis of corneal ulcers. One drop of a solution of Fluoresceine, grs. 10; Sod. Carb., grs. 16; Aq. ad ℥i., is applied to the cornea, and after the eye has remained gently closed for a minute, the brownish yellow fluid is washed out of the conjunctival sac with any sterile lotion; all those parts of the cornea not covered by epithelium take on a bright green stain, so that it is very easy to see the exact limits of any epithelial defect. The fluoresceine should not be washed out with a cocaine solution, as is often done, under the impression that by this means the colour is intensified; by such a proceeding the epithelium is damaged, and no further tests of the corneal or conjunctival sensibility can be made. The fluoresceine test should not be used for the purpose of avoiding a thorough examination of the cornea, but only as a means of confirming or amplifying our observations with regard to the condition of the corneal surface.

As the cornea is a highly transparent structure,

pathological processes in it become obvious as soon as they affect its surface, its transparency, or its form. When rays of light fall on the cornea they are reflected, refracted, or diffused; normally a large proportion of the light is refracted into the interior of the eye, to form a real image of the source of light on the retina; a part, which varies with the angle of incidence of the rays, is reflected to form an apparent image of the same object lying behind the cornea; the remaining inappreciable amount is diffused. When the cornea is attacked by a plastic or a destructive process its power of refraction, reflection, and diffusion of light changes greatly; in general more light is diffused, and less is refracted and reflected than is the case in the normal eye, the result of such a change being a diminution in the visual acuity, and alterations in the appearance of the cornea. Changes in visual acuity can readily be determined and accurately measured; unfortunately, however, they are of very little value for either diagnosis or prognosis, as no constant ratio is found between the severity of the various lesions and their interference with vision. On the other hand, it is of the greatest importance to carefully examine the alterations in appearance of the cornea, as the diagnosis of the various ulcers depends almost entirely upon visible changes.

The radius of curvature of the normal cornea is about 7.5 mm., and its surface is perfectly bright; when diffused light from a window falls upon this convex

surface, a small upright reflected image of the window can be seen, apparently a short distance behind the reflecting surface. As the central area of the cornea is practically spherical, and the peripheral only slightly flattened, this image will not alter appreciably in shape or size as the patient moves the eye, and different parts of the corneal surface successively act as the reflector ;

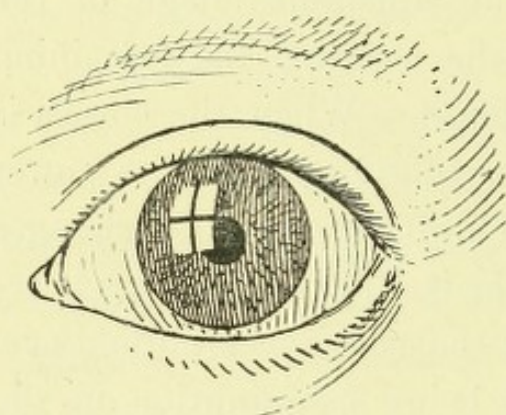


Fig. 4.

LIGHT-REFLEX ON NORMAL CORNEA.

in particular the reflected images of the crossbars of the window, seen as fine lines, will always appear sharp and clear, their direction straight or slightly curved in regular arcs, and this appearance will not change with movements of the patient's eye. Any-

thing which interferes with the brightness or evenness of the corneal surface causes a change in this 'light-reflex,' and as the condition of the surface of any transparent body is most readily determined by the nature of the reflected image which it forms of surrounding objects, we have in this 'light-reflex' a method of determining the surface condition of the cornea with great accuracy.

We will first consider the variations in the reflected image, and then endeavour to explain how these changes can be accounted for by different conditions of the corneal epithelium. The simplest change which can occur is that the image becomes cloudy or steamy,

without any alteration in the shape or proportions of its various parts. In our illustration the window will still be the same shape, but the lines will appear dull and show a fine waver. A further change in the same direction will produce a more obscured image, in which all fine details are lost and only the general outline is preserved. These changes are entirely due to alterations in the epithelium: any cause which will produce a fine roughening of its smooth surface, whether by the swelling of the superficial cells or by loosening their cohesion so that they are cast off, results in this dull, matt, steamy or breathed-on appearance of the cornea; and if the groups of cells so affected be larger, and the surface irregularity thus produced be so coarse as to be visible, then the granular appearance results. The dullest surface is seen when the epithelium is completely cast off, and the bare corneal lamellae are exposed, as in an abrasion or ulcer.

The corneal reflex can show a distorted, though clear and bright image. The fine lines, which on some parts of the cornea are perfectly straight, at other parts are sharply curved, or form irregularly concentric rings. In other cases they show quite well-marked angles. Such appearances occur when the corneal surface, while remaining smooth and polished, has gross irregularities on it, such as facets, linear scars, or protrusions.

The combination of these two variations in the surface reflex results in many appearances which are very important in the diagnosis of corneal ulcers. The

matt surface, whose distorted reflex shows it to be bulged forward, indicates the œdematous epithelium overlying a fresh infiltration; the line at one place straight, but close by sharply curved and ending abruptly at an area where no reflection can be got, tells of the pouting lip of an ulcer.

It is of great importance to accurately determine the extent and distribution of the loss of epithelium in every corneal ulcer. A rough chart of the cornea can easily be made which will show this clearly; as the case progresses these rough charts will be found of the greatest assistance in indicating the value of our treatment.

Having thoroughly examined the condition of its surface, the next step is to consider any changes in the substance of the cornea. Variations from the normal may occur in colour or transparency. Changes in colour are best observed by daylight, as they are often masked by the intensity and colour of the artificial light. The method of determining changes in transparency, however, is by means of lateral focal illumination. By the aid of the loupe opaque areas can be examined to determine their exact extent, depth, and relation to any superficial changes previously observed; also their constitution, whether resolvable on moderate magnification ($\times 10$) into fine or coarse-grained markings, or still remaining homogeneous. At the same time the occurrence of vessels in the cornea will be noted, with their depth, mode of origin, and

distribution. The posterior surface can also be examined for deposits precipitated upon it, or changes in the endothelium lining it.

The plan of sketching each case is nowhere more useful than when dealing with corneal conditions. The

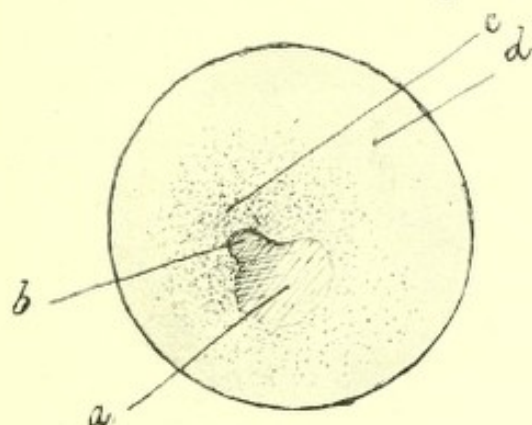


Fig. 5.

SURFACE APPEARANCE.

a Shallow loss of substance, becoming deeper at *b*; *c* Surface dulness, becoming brighter towards *d*.

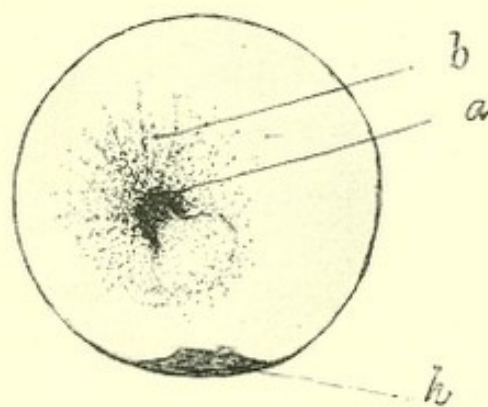


Fig. 6.

CHANGES IN SUBSTANCE.

a Dense opaque area; *b* Slightly infiltrated area.

most rudimentary knowledge of drawing is all that is necessary, as the different appearances can readily be represented by lines and dots. I represent loss of epithelium by shading (*a*, Fig. 5), and by cross-hatching when there appears to be loss of parenchyma also (*b*, Fig. 5); surface dulness by a dotted surface (*c*, Fig. 5). For changes in the substance of the cornea I make another sketch (Fig. 6), and represent the infiltration by dots (*a*) (*b*), which can be made denser by increasing their number and size. In this manner the infiltrated edge of an ulcer can be represented (*a*, Fig. 6).

The sensibility of the cornea should be tested. This

varies very considerably in different normal individuals, and the threshold of sensation is not so low as might be supposed from the sensitiveness of the eye in general; the conjunctiva, lid margins, and cilia are much more sensitive than the cornea itself, so that we must carefully avoid touching them. A wisp of cotton wool can be used to lightly brush over the cornea, but a pointed piece of blotting paper is rather better, as it is firmer and its touch is always appreciable by the normal cornea. The paper should be held quite close to the surface, so that contact is made by a very slight movement, otherwise the patient, seeing a body approach the eye, instinctively winks, when the lids will be touched. The whole cornea should be tested, as regional variations often occur, and are of importance. The presence of a thin leucoma with loss of sensibility has been held by some observers to be diagnostic of a Moorens ulcer, and in recurrent corneal erosion a localised lowering of sensibility is often easily determined, and of considerable importance.

The cornea may now be left for a moment while some other information is obtained. Most important in many conditions is the state of the lacrymal apparatus. The position of the puncta lacrymalia can be seen at a glance; the lids should be slightly everted by the finger and thumb pressing lightly upon them till the puncta stand out clearly in view, then with the blunt end of an instrument handle or a glass rod the lacrymal sac should be compressed in

its fossa; if any fluid exude from the puncta, the lacrymal passages are not clear, and some of the fluid should be removed with a sterile platinum loop for bacteriological examination. If there be no regurgitation, the lacrymal passages must not be considered as normal; but since their further examination may be painful and necessitate the use of cocaine, we pass on in our systematic examination to the conjunctiva. The condition of its surface should be noted as to moistness or dryness, smoothness or roughness, the presence of granulations or localised swellings; if there be any redness, whether it be due to the congestion of superficial or deep vessels, and how it is distributed with relation to the corneal changes. The discharge, if any, must be examined; in cases of acute conjunctivitis it may be necessary to stain a film. The sensibility of the conjunctiva should be tested at several points around the cornea. The lids must be thoroughly everted for the complete examination of the conjunctiva fornix, and to make certain that no foreign body is lodged under them.

In many cases the further examination of the eye will necessitate the use of cocaine, but it is hardly necessary to do more than point out that the size of the pupil and its reactions should first be taken, and all proving of sensibility made, before it is used. I would not like to say how often an anæsthesia of the cornea is missed by rashly dashing in cocaine to remove a foreign body, or by that still less defensible practice of commenc-

ing the diagnosis of a corneal ulcer by introducing fluoresceine into the conjunctival sac, and washing it out, after a minute or so, with a cocaine solution. When the patient is cocainised (and this drug should only be used when the following examination is considered necessary, as it has a distinctly deleterious effect on the epithelium) the edges of any ulcer should be carefully examined with a fine probe to see if the margin is overhung. And then with a platinum spud (Fig. 7) a small quantity of the débris of the ulcer should be removed for microscopical examination (for method see p. 184).

The lacrymal passages should still further be tested by means of the syringe as to their patency or the presence of a stricture. The punctum should be dilated and the patient directed to lean well forward, when about 30 minims of saline solution is injected into the lacrymal sac: if the nasal duct be patent some of the fluid will drop from the nose.

In every case, of course, as complete an examination as possible should be made of the patient's eye and its appendages. I will not now go more fully into the systematic examination of an ophthalmic case, but will mention the further points as they occur in considering the various lesions which are included under the term ulceration of the cornea.

CHAPTER II

SIMPLE LESIONS OF THE CORNEA

THE simplest destructive lesion of the cornea is a mechanical injury. There are many forms of injury which do not come within the province of ulcers or ulceration, such as incised or perforating wounds, but some others must be considered under this heading. These are :—

- (a) A corneal abrasion.
- (b) A contusion of the cornea.
- (c) A foreign body in the cornea.

A CORNEAL ABRASION

The term abrasion is used to define the result of an injury, which has removed and destroyed the surface of the cornea for a variable extent, with little or no damage to the parenchyma. Such abrasions are caused by the scratch of a finger-nail, a twig, or a branch, a foreign body under the lid, such as a husk of corn, or other similar accident. The symptoms are usually very severe, the pain, photophobia, and lacrymation are out of all proportion to the severity of the lesion and the

obvious physical signs. When the eye is examined the conjunctiva is found to be congested, and there is some ciliary injection, but beyond this there is nothing obvious until the cornea is examined carefully, when changes are found affecting its surface. Over a variable extent no light-reflex is obtained, and this dull area is abruptly bordered by normally reflecting cornea. It is difficult or impossible to determine any difference in level between the dull area and the surrounding bright surface, for the abrasion which has removed the epithelium has allowed fluid to have access to the cells under the surface, which swell sufficiently to counterbalance the defect due to loss of substance; in fact, a slight elevation of the affected area is occasionally observed. The transparency of the cornea beneath is very little affected; by direct inspection in a good light it may be impossible to detect any change, but by using focal illumination and a loupe a faint smoky appearance of the surface layers may be observed. The examination is completed by the use of fluoresceine, when the abraded area shows up beautifully as a bright green patch in an otherwise unchanged cornea.

Pathology.—The depth of such lesions varies greatly: they may only remove the epithelium, they may go through Bowman's membrane, or they may penetrate into the more superficial layers of the parenchyma. The most superficial are the most painful, for the sensory nerve endings in the cornea lie amongst the epithelial cells, and it is their exposure and irritation

which causes the pain and photophobia. If they be destroyed by the lesion the subjective phenomena are naturally much less severe, as is actually observed in the deeper abrasions.

The tears can enter the abraded surface, fill up the lymphatic spaces, and by imbibition swell the epithelial cells or corneal lamellae, so that sometimes there appears to be an increase of substance at the site of the lesion. The epithelium at the edge of the denuded area grows rapidly, and spreads over the abrasion, so that the surface is very soon restored.¹ If the abrasion has only affected the epithelium, and the wound is quite aseptic, the restoration is very rapid and complete, so that absolutely no trace may remain of the former loss of substance. When Bowman's membrane and the parenchyma of the cornea have been involved, a facet may result after the epithelium is restored; this will gradually fill up by a thickening of the surface layer and a deposit under it of connective tissue, forming a nebula or leucoma. When there is loss of substance to restore, and especially if the ulcer lies near the margin, new vessels form in the cornea, passing from the conjunctiva under the epithelium to the site of the injury; healing then takes place more rapidly as the nutrition of the regenerating tissues is improved, and cellular material is brought to fill up the loss.

The *Prognosis* depends entirely on the possibility of

¹ If anything should interfere with this regeneration, the condition known as *Keratalgia Traumatica Recidiva*, or recurrent corneal erosion, may result (see p. 19).

keeping the wound free from infection, especially pneumococcal; if this evil be avoided healing takes place rapidly, and there only remains a faint opacity, which gradually becomes thinner. Two peculiar sequelae sometimes follow an abrasion; these are *recurrent erosion* and *filamentary keratitis* (see p. 19).

The *treatment* must be directed towards, firstly, the prevention of infection, and secondly, the promotion of sound healing. The condition of the lacrymal passage should be examined; if there is a mucocœle the sac should at once be excised (see p. 176); if there be any stenosis, or stricture of the passage into the nose, the lacrymal sac and nasal duct should be frequently washed out with a strong solution of protargol (five per cent.), which is in turn removed by boracic lotion or normal saline.

If the nasal duct be impervious, that is to say, if fluid does not pass at all into the nose when the sac is distended, the sac should be excised. In all cases where there is any suspicion of a chronic conjunctivitis, or any epiphora, a smear preparation should be made, and cultures taken to determine, if possible, the presence or absence of pneumococci in the conjunctival sac.¹

¹ The above proceeding in cases of corneal abrasion will be considered by many as quite unnecessary, and much too severe. In London, where fortunately the pneumococcal ulcer is comparatively rare, the risk may be thought too slight to necessitate even an operation so simple and satisfactory as the extirpation of the sac, but in other localities where the pneumococcal ulcer is much more common, *e.g.* in Bonn, where at one time two per cent. of Saemisch's out-patients came suffering from 'ulcus serpens,' these preventative measures will commend themselves. The question is further discussed in connection with pneumococcal ulcer of the cornea (*q.v.*).

The local treatment consists in the use of an occlusive bandage and weak atropine if the pain be very severe ; with the atropine dionin can also be prescribed, R/ Atrop. Sulph. gr. ii., Dionin, gr. iv., Aq. ℥ i. Cocaine should be absolutely avoided ; it is directly detrimental to the epithelium, and prevents rapid and sound repair ; recurrent erosions are much more liable to take place in those cases where cocaine has been used.

When the epithelial surface has been completely restored there may be a facet remaining which, if it be in the pupillary area, interferes greatly with vision. To promote the filling up of such a defect massage of the cornea with Ung. Hydrarg. Flav. is very useful. This substance should be used at first in the strength of grs. iv. ad ℥ i., twice daily ; the strength and frequency can be increased up to grs. xvi. and four times daily as the eye becomes amenable to the irritant. The ordinary yellow ointment should not be used, but it should be specially made up absolutely free from grit.¹

In no case is the occlusive bandage to be too hurriedly laid aside ; it should be kept on for a few days after the eroded surface is re-covered with epithelium. For another week at least a simple boric vaseline, or weak sublimate ointment, must be used at bed-time to prevent the lids sticking, and so favouring recurrence of the erosion (*q.v.* p. 19).

¹ For the best method of preparing Ung. Hydrarg. Flav. see Martindale in the *Ophthalmoscope*, 1906.

CONTUSION OF THE CORNEA

An injury to the cornea, which differs from an abrasion only by its difference of appearance, is a *contusion* of the cornea. This results when a small body is driven forcibly against the eye, and in addition to causing a surface wound bruises the deeper layers of the cornea. The body does not penetrate the epithelium but rebounds from its surface, leaving a small tear or rupture in it. The shock of the blow is transmitted to the deeper layers, and causes an extravasation of fluid between them; the condition resembles a bruise, the fluid effused is lymph, the injury taking place in an avascular tissue. Such an appearance can result from a small stone driven forcibly against the eye by the wind, or a shot at long range not having sufficient momentum to perforate the cornea.

The superficial loss of substance is readily seen by means of the corneal light-reflex, and during the first hour or two after the injury, in the substance of the parenchyma, directly behind the defect, there is a dense white infiltration, round or oval in shape, and sometimes surrounded by a faintly marked line like a halo. Instead of this disc-like infiltrate we sometimes see a greyish diffused opacity in which run a network of clear lines.

Both of these opacities lie deeply, and are separated from the abrasion by layers of clear cornea. They are probably due to the shock of the injury separating some

of the corneal lamellae from each other, and an effusion of lymph taking place into the space thus formed. The appearance changes completely in the course of a few hours to that of an ordinary corneal abrasion.

This condition must be carefully distinguished from the first stage of a pneumococcal ulcer, in which we have a surface defect and a deep infiltrate. The history alone is quite sufficient for a differential diagnosis: the contusion can only be seen during the first few hours after the injury, while the pneumococcal infiltrate only shows itself after several days.

The prognosis and treatment are exactly the same as for a corneal abrasion.

Recurrent Corneal Erosion.

Keratalgia Traumatica Recidiva.

An abrasion of the cornea usually heals readily and causes no further trouble; there are, however, several injuries which are specially liable to give rise to recurrent affections at, or near, the site of the original abrasion. The type of such an injury is the scratch of a finger-nail on the cornea: this indeed is the commonest cause of that condition, which was first described in 1872 by Hensen under the title 'Nagel-Keratitis' (finger-nail keratitis). The affection is met with in two forms: the mild keratalgia which Hensen observed; and the more severe erosion to which Arlt called attention in 1874.

The patients complain of sudden sharp pain in the eye, as though a foreign body were in the cornea, occurring in the morning, but not so as to waken them from sleep, but rather as the result of their attempting to open their eyes. They may experience considerable difficulty in actually opening their lids, which are stiff and heavy, though not actually sticky. The pain lasts usually for a few seconds, exceptionally for an hour or two. The history of previous attacks, and of the original abrasion, is usually obtained. The pain may occur every morning for several days; and then a remission for weeks or months,—the attacks appear more common in warm weather. On examining the eye only a slight conjunctival injection is seen, the cornea may appear bright and clear; on very careful examination with focal light and a loupe, a faint greyish haze can be discerned on the surface, and prolonged staining with fluoresceine may impart a faint greenish hue to the part; some slight anæsthesia may be found over the affected area, from which the epithelium is very easily peeled off. This is the condition known as *keratalgia*.

With exactly similar symptoms examination of the cornea may show the loss of some epithelium, resulting in a small patch staining very readily with fluoresceine; at the margins of the erosion thus formed there may be small vesicles. If the eye be examined before the epithelium is actually cast off, a small bulla, or a crop of vesicles, may be

present. In these cases there is usually some opacity of the cornea surrounding the base of the elevations, the actual floor of the ulcer being rather clearer than the part around. These are the genuine *recurrent erosions*: in them the history is longer, often several attacks of the milder condition having preceded the one under observation. The vision may be seriously affected, when, as is often the case, the pupillary area is involved.

It is difficult to explain the *pathology* of these conditions. The general opinion seems to favour a chemical irritant, introduced at the time of the original lesion, causing a want of cohesion between the epithelial cells which grow so rapidly over the denuded area; during sleep the lacrymal and conjunctival secretions are diminished, and the corneal and tarsal epithelia tend to adhere, so that on waking a strain is put upon the deeper, less adherent cells, and thus an irritation of the nerve terminals results. Should this tearing strain be sufficiently strong, fluid accumulates in the intercellular spaces, and a vesicle or bulla results, the lesion described being caused by its rupture.

In considering the *treatment* of this condition, we must begin by appreciating the necessity of prevention by attention to the original lesion. All scratches of the cornea, where considerable surface is abraded, require the application of an occlusive bandage until the wound is firmly healed; in the case of obviously septic wounds,

such as a finger-nail scratch, the surface should be touched with carbolic or washed with diluted Liq. Chlori., and the eye bandaged for several days,—a weak sublimate ointment is also valuable. When we have to do with a keratalgia, a small quantity of Boric Lano-line, or Ung. Cetaceae softened with a drop or two of Ol. Olivae, should be introduced into the eye every night, and care used in opening the eyes in the morning. When an erosion is present, a bandage must be applied and maintained for several days (8-10). Should this fail to cause firm healing, the eye should be cocainised and the affected area scraped with a sharp spoon: the epithelium will readily peel off from a considerable area, leaving Bowman's membrane bare; the denuded surface can be lightly touched with carbolic or Liq. Chlori., some ointment inserted into the conjunctival sac, and the abrasion allowed to heal under a bandage.

These patients will require careful supervision afterwards, the use of an ointment at night should be insisted upon for some time after healing has occurred. Any slight conjunctivitis causing a stickiness of the lid margins may bring on a recurrence.

Other Sequelae of Simple Corneal Lesions.

Progressive ulceration of the cornea, or purulent keratitis, often results from such corneal abrasions as we have described: these are treated in other chapters.

In quite a number of cases a foreign body in the

cornea appears to determine the onset of a keratitis parenchymatosa. In patients who suffer from hereditary syphilis, it is quite recognised that any traumatism, even the rubbing of a misdirected eyelash on the cornea, may result in the development of a keratitis. In patients, however, whose personal and family history appears quite free from any suspicion of syphilis, it is possible for a foreign body to set up a parenchymatous keratitis, which is indistinguishable from the avascular type of that disease occurring in hereditary syphilitic patients, and which may result in severe deep-seated changes.¹

Several forms of superficial keratitis may follow an abrasion ; their connection with the original injury may sometimes be obscure, as in cases of recurrent corneal erosion. The conditions known as bullous keratitis, recurrent corneal bullae, filamentary (*Fädchen*) keratitis, are often distinctly related to injuries.

A condition known as *Filamentary Keratitis* (*Fädchen Keratitis*) can occur after an abrasion. A soft gelatinous thread hangs by one end from the cornea, the other being swollen into a knob, and lying free.

This thread can easily be seen if the patient recline with a speculum in the conjunctival sac, and the pocket formed when the speculum is raised from

¹ A case under my charge was one of a loco-fireman, who, fourteen days before seeking my advice, had a spark from the engine driven into his right cornea. He presented a perfectly typical avascular keratitis parenchymatosa. A hypopyon developed, then flattening of the cornea, low tension, and vision remained only at hand movements.

the eye filled with warm saline : the filament will then float freely in the little pool of fluid thus formed.

The filament is formed of epithelial cells, and is covered with a layer of mucus ; probably the causes of its formation are similar to those producing the bullae in a recurrent corneal erosion. The filaments may arise from the septum between two bullae (Collins).

The symptoms are very like those of a recurrent erosion (p. 19), and the treatment must be radical. The filament should be scraped off with a sharp spoon and the site cauterised with pure carbolic acid.

A FOREIGN BODY IN THE CORNEA

This is the commonest lesion which we are called on to treat : it is especially frequent amongst mechanics and labourers. As the results of such an accident may be very grave, even resulting in the loss of an eye, it is necessary to devote some attention to their treatment, as unfortunate results can be almost entirely prevented if the case be handled in a proper manner from the time of the accident.

Although a foreign body may injure any part of the cornea, there is a tendency for the lesion to occur in the central or lower parts of its surface ; that part covered by the upper lid is rarely affected ; most commonly the body will be found embedded in a band-shaped area of four or five millimeters' breadth across the cornea just below its centre, this being the part

usually exposed, and the last to be covered when the lids are closed.

In all trades which involve hammering at an anvil there is a danger of chips of metal lodging on the corneal surface. Special areas of the cornea are more liable to injury than others, the lower part mostly, and in right-handed subjects the left cornea; thus in blacksmiths and stonemasons the lower and outer quadrant of the left cornea is the common site, on account of the position of such men at work, with the left foot and shoulder thrown forward and the right arm free to strike; next in order of frequency is the inner and lower quadrant of the right cornea. In locomotive drivers or firemen the foreign body is almost always just below the centre of the cornea, from their habit of peering out through half-closed lids when exposed to this danger.

The sensation of 'some grit in the eye' is too well known to need description, but it must be noted that the sensibility of the cornea is not very acute, and certainly is quite dull when compared to that of the conjunctiva and lid borders. When a foreign body is embedded in the cornea the sensation of its presence is due to the irritation of the conjunctiva of the lids when they move over its sharp edges. This is strikingly exhibited in those cases where the foreign body lies exactly opposite the slit between the closed lids, that is, about the junction of the middle and lower thirds of the cornea; in such a case there may be absolutely no gritty or uncomfortable feeling in the eye at all. The

relief experienced when an occlusive bandage is applied to an eye with a foreign body in the cornea, is caused by bringing the lids to rest, preventing their movements over the gritty particle. Therefore, although the patient's positive history of something getting into the eye is of great importance in the diagnosis of a foreign body, the absence of any such must not be taken to exclude the possibility of the lesion.

The *symptoms* complained of are usually pain, photophobia, and lacrymation, occasionally merely diminution of sight.

In a few hours the superficial conjunctival vessels become injected and congested around the corneal margin, so as to stand out sharply against the sclerotic behind; later on the deeper structures also become congested, and then the bright red superficial vessels do not show up in such marked contrast against the pinkish background. On examining the surface of the cornea, the reflex will be found to be broken or deficient at the injured point, and if the injury has occurred a day or more before the examination there may be some dulness around. By focal illumination and the loupe the foreign body can easily be seen firmly embedded in the cornea or lying loose in an infiltrated area. It is of the utmost importance in such cases to examine the lacrymal apparatus, as, if the wound become infected from the nasal duct, a purulent ulcer may result. This question is discussed on page 46.

The *treatment* is removal. One or two instillations

of a one per cent. cocaine solution will produce sufficient anæsthesia,—stronger should not be used, as the drug damages the epithelium. With a sharp lancet-pointed needle the body is picked out of the cornea with as little

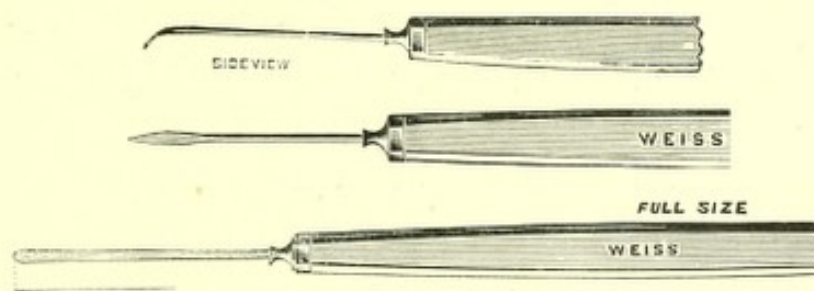


Fig. 7.
NEEDLE AND SPUD SKETCHES.

damage to the epithelium as possible. If the body be lying loose in a small infiltrated depression it can often be removed more readily with a spud. When the foreign body is iron we often see around it a brown ring of stained corneal tissue: it is good practice to scrape away all the brown deposit when removing the body itself; and no case can be considered as efficiently treated unless there is absolutely no trace of foreign substance to be seen in the wound even with a loupe.

The eye should be kept tied up until the epithelium has grown across the abraded surface; it is bad practice to uncover the eye too soon. When the foreign body has been removed immediately after the accident, healing of the epithelium will be completed in about thirty-six hours; but if it has been left for a day or two and there is considerable reaction in the cornea around,

in the interests of sound healing it is better to keep on the pad and bandage for three or four days. In these latter cases there is some difference of opinion as to whether atropin should be used or not: in a perfectly fresh case with no inflammatory reaction there is no indication at all for its use, but where the cornea around is dull and the body lies in an infiltrated area there is sufficient chance of an iritis occurring to justify the use of atropin, especially as the eye is to be tied up for several days, and its use will therefore cause very little inconvenience; one or two drops of a one per cent. solution are instilled into the eye after the removal of the foreign body, and if no signs of iritis develop it need not be repeated.

CHAPTER III

THE PNEUMOCOCCAL ULCER

ULCUS SERPENS (SAEMISCH)

TYPICAL HYPOPYON ULCER

SERPIGINOUS ULCER OF THE CORNEA

A PNEUMOCOCCAL ulcer results from the infection of the cornea with the *pneumococcus lanceolatus*. By necrosis of the superficial lamellae, and casting off of the epithelium, a shallow depression is formed in the cornea, bordered at some part of its circumference by a densely infiltrated, undermined margin, which progresses over the cornea. The ulcer has no marked tendency to perforate. It is accompanied by a hypopyon.

Although this is the most definite form of hypopyon keratitis, a want of precision with regard to its clinical features seems to exist, which is difficult to account for when we consider how thoroughly the subject has been studied. Being the most important form of purulent keratitis, its course and pathology will be described in considerable detail, so that other forms may be compared to it and repetition avoided. Amongst the many forms of treatment which are in

use, those which appear to be based on a thorough knowledge of the conditions present will be fully stated, and the others merely indicated.

The first description of the condition was given by Saemisch in 1870. He observed the association of certain well-marked characters in a large number of corneal ulcers, and, by comparing all cases of purulent keratitis, was able to collect together certain characteristic symptoms, and build up a type which he called *ulcus corneae serpens*. The original description is so interesting as a record of accurate clinical observation that I venture to translate in full a passage from his work, *Das Ulcus Serpens*.

‘*Ulcus corneae serpens* develops at or near the centre of the cornea. In the early stages inflammatory symptoms are well marked only in exceptional cases; it is more often the interference with vision rather than pain for which the patients seek advice. In the cornea we see a round greyish opacity, with superficial loss of substance. In rare cases the patient is seen early enough for us to observe a superficially situated infiltration of the cornea, which precedes and is transformed into this ulcer. Apparently the layer of corneal substance over the infiltrate is cast off so rapidly that the marked characteristics of the ulcer are very early observable. This condition very rarely develops from deeply situated infiltrates or abscesses.

‘The margin is characteristic from the first. At some part of its circumference, in extent usually less

than one half, it is swollen, greyish-white in colour, and crescentic in shape,—or it may consist of several crescents, separated from each other,—and shows points of infiltration which soon coalesce. The floor is greyish, rarely showing white spots, uneven, and covered with the remains of necrotic tissue. It always slopes down towards the greyish-white part of the margin. The transparency of the cornea around is only rarely markedly affected in the early stages—in fact, it is usually maintained with but slight diminution. The slight changes consist essentially in the formation of fine grey striae radiating from the infiltrated margin of the ulcer.

‘These can be followed through the cornea to its posterior surface. The ulcer has a tendency to spread superficially—to creep forward, as it were—for which reason it is called *ulcus serpens*. It pushes itself slowly into the healthy tissues, not spreading out in all directions, but only in that one in which the changed condition of the margin is most marked. The ulcer also progresses slightly in depth, and becomes white and opaque in cases where suppuration is profuse. There is a continual casting off from the floor, not however so unevenly as to cause crater-like or funnel-shaped depressions in it. The ulcer spreads out and covers a large area of cornea, and is followed by perforation, staphyloma, or phthisis anterior; in favourable cases by a dense leucoma.’

In 1894 Uhtoff and Axenfeld stated that the *ulcus*

serpens of Saemisch, in a large proportion of cases, contained the diplococcus lanceolatus (pneumococcus), and that the ulcer was caused by this organism infecting the cornea through a breach in its surface. They agree with other investigators about the characteristics of the type, that the individual cases are caused by this infection, and that, if purulent ulcers are carefully examined, a large number of them will be found to conform to this type. These ulcers are invariably associated with the presence of the pneumococcus, and the term pneumococcal ulcer should replace that of the *ulcus serpens*. The question of causation was finally settled by Römer, who, by improved methods, demonstrated the presence of the organism in cases where a previous examination had failed to detect them.

The pneumococcal ulcer is commonly found in adults, especially in the class of masons, navvies, and agricultural labourers. Very often the first inflammatory symptoms occur after a slight corneal abrasion, but in other cases no history of injury can be obtained. For a day or two there is an uncomfortable feeling of something in the eye, the sight is not so good, friends may notice a white spot on the eye, the eye weeps, and bright light is avoided. These symptoms may not cause alarm; and it is strange how often men will continue their work with a corneal ulcer until a sleepless night, severe pain, and great loss of sight drive them to seek advice. The attention of the surgeon turns at once to the cornea, where a grey area a few

millimeters in diameter is seen; the dull epithelium bulges forward over a round, sharply defined infiltrate situated in the superficial layers of the cornea (Fig. 8, A).

The conjunctiva is congested, and there is some ciliary injection. At a very early stage the bulging epithelium gives way, so that a definite defect occurs in the corneal reflex. When an infiltrate has reached the diameter of 3-4 mm. it will have generally separated, along the greater part of its circumference, from the surrounding cornea; but at one place it remains adherent, and there a gradual transition occurs from the dense white infiltrate to the slightly clouded cornea, without any demarcation line. A small slough lies in a cup-shaped depression, adherent on one side where the opaque necrotic mass passes into the infiltrated but still living tissue under cover of the epithelium. Near the ulcer the surface reflex of the cornea is dulled, and the transparency of its substance impaired; with the loupe grey lines can be seen in the deeper layers radiating outwards from the ulcer. There is some turbidity of the aqueous and iris in its lower half. The conjunctiva shows a varying degree of inflammatory reaction. With a stream of warm sterile lotion a whitish plug of necrotic tissue can be removed, after incising any epithelium remaining over the infiltrate, with, or sometimes without, the assistance of a sharp spud. If at this stage the condition be untreated, by necrosis of the epithelium and superficial corneal lamellae which overlie the

infiltrate, this pneumococcal infiltrate changes into the completely developed pneumococcal ulcer (Fig. 8, **B**). When this has occurred we find in the cornea a superficial defect of variable size, some parts of which are not at all well defined from the surrounding cornea, as the epithelium appears to pass unbroken into the shallow depression, whose exact extent is only appreciable on carefully studying the corneal light-reflex; at other parts we find the ulcer sharply contrasting with the surround-

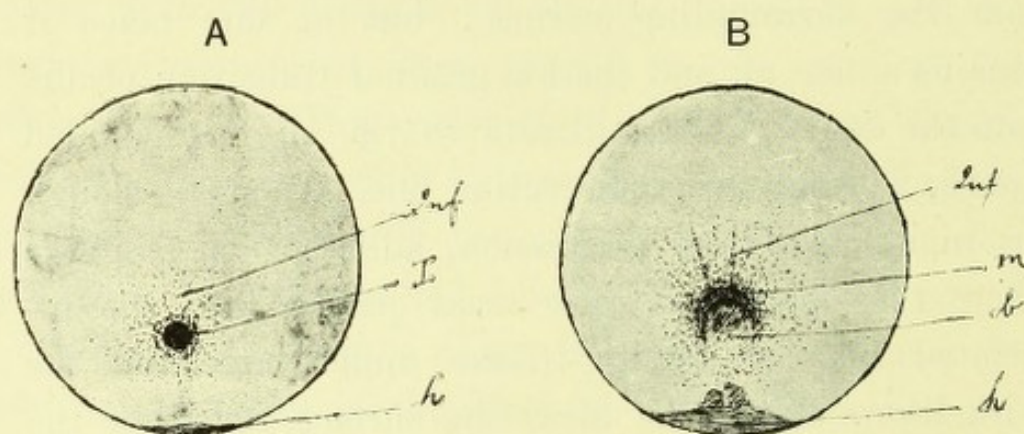


Fig. 8.

TRANSITION OF AN INFILTRATE **A** INTO AN ULCER **B**.

I Dense infiltrate.
Inf Dull cornea around.

m Advancing margin.
b Base of ulcer.
h Hypopyon.

ing cornea. A densely saturated white border (Fig. 8, *m*), which has the form of a bay projecting from the main outline, here marks a distinct difference in level between the floor of the ulcer and the surface of the cornea. This is due partly to the raising up of the epithelium by swelling and infiltration of the corneal lamellae, and partly to the fact that the greatest destruction of cornea is immediately inside this margin. An abrupt

and overhanging ledge is thus formed, which is made still more obvious by its yellowish white colour; under this ledge a probe can be passed, lifting up the epithelium to the extent of about a millimeter. The limit of the visible margin is then seen to be some little distance further into the cornea than the line of the broken epithelial edge. Thus there is the appearance of a double contour, the outer line of which is yellowish white and under the surface, the inner gelatinous, greyish, and quite superficial. At the other parts of the circumference the edge is much less distinct, and is rounded off, the epithelium apparently passing over it on to the floor, which has the appearance of rough ground glass.

Such an ulcer rarely heals spontaneously; it usually spreads over the surface of the cornea by the advance of the densely infiltrated border into the sound tissue; the dense white subepithelial infiltrate always preceding the actual process of necrosis. The floor of the ulcer behind the advancing line is covered with flaky *débris* of the dead lamellae, over which the epithelium from the opposite edge begins to grow. When the ulcer has reached considerable size the floor shows signs of thinning, flakes of white material are cast off, and the surface is covered with pus or *débris*. The intraocular pressure causes bulging, and perforation with iris-prolapse may occur. A hypopyon begins to form very early, and after a few days there is a well-marked iritis.

The early stages of the ulcer are not associated with much pain, shown by the fact that the patients often continue their work with an ulcer 4-5 mm. in diameter. There is some photophobia and lacrymation. In some cases, after a few days, intense pain develops, often referred to the top and back of the head and shooting through to the eye. This does not appear to be due to an iritis, as it is often very distressing when the iris reacts well, but most probably can be referred to oedematous pressure on the nerve endings in the cornea, —such pain may cause complete loss of sleep. The general symptoms are slight; the temperature is not affected, but patients complain of weakness and loss of appetite. The disease has no tendency to self-limitation, a point of considerable interest in connection with its etiology. There is generally considerable interference with vision, depending chiefly on the site of the ulcer, and the direction taken by its advancing edge, the amount of iritis, and the extent of the hypopyon. But even when the ulcer is situated peripherally with the pupillary area free from hypopyon or exudate, and the centre of the cornea unaffected by ulceration, oedema and infiltration of the retina may cause an otherwise unaccountable diminution in sight. It is worth noting that such a defect may remain after the ulcer has healed, and such patients can be usually told that improvement in their sight will continue for some time after this has occurred, and the morbid process has apparently completely resolved.

The amount of hypopyon is very variable, and in any one case grows larger or smaller as the ulcer spreads or heals. It is preceded by a turbidity of the aqueous and a cloudiness on the back of the cornea ; a thin yellow crescent is seen in the lower angle of the chamber ; this broadens till the whole cavity may be filled with pus. If the base of the ulcer is above the upper level of the hypopyon, threads of pus appear to run down from behind the ulcer into the collection below. This gave rise to the erroneous view that the hypopyon was due to a suppurative process in the cornea, discharging into the cavity of the anterior chamber. The real reason is something quite different, as will be explained later.

The ulcer is not, as a rule, associated with a conjunctivitis ; but in quite forty per cent. of the cases there is a blenorrhœa of the lacrymal sac (mucocœle), and in a considerable number of the remaining cases there is a stricture of the nasal duct, ozæna or some form of chronic rhinitis.

Pathology.—The structural changes can be clearly seen by examining sagittal sections through the cornea ; finer details of cell structure are best studied in tangential sections ; and the relation of the organism to the changed condition of the tissues is rendered evident by staining these sections by Gram's method. The mode of origin and development of the ulcer can be readily understood from a consideration of (*a*) sections through the infiltrate just as it is beginning to break

down ; and (b) sections through the fully developed ulcer.

(a) In the early stage we find a thickening of the cornea corresponding to the site of the infiltrate. Changes have taken place in every layer of which the cornea is composed. At a little distance from the swelling *the epithelium* is quite normal in appearance, but approaching it we find inflammatory changes becoming more and more marked ; the cell nuclei become lobular and slightly enlarged, they lose their normal staining reactions, and finally show only as a granular chromatic débris. The protoplasm swells, becomes granular and vacuolated, the cell borders are fibrillated, and separate from each other, till finally there is merely a necrotic hyaline mass adhering to Bowman's membrane, which loses its normal structureless appearance, develops a fibrous, and finally a loose laminated appearance.

Throughout *the parenchyma* we find the cellular elements increased to such an extent at parts, that the lamellae are obscured, and only a dense mass of nuclei is seen without any cell protoplasm between them. This corresponds to the thickest part of the cornea and the most advanced epithelial changes.

These new cells are derived by proliferation from the fixed connective tissue cells of the part ; and by invasion from the leucocytes of the surrounding blood-vessels. Both varieties show considerable variation from the normal at the focus of irritation, where,

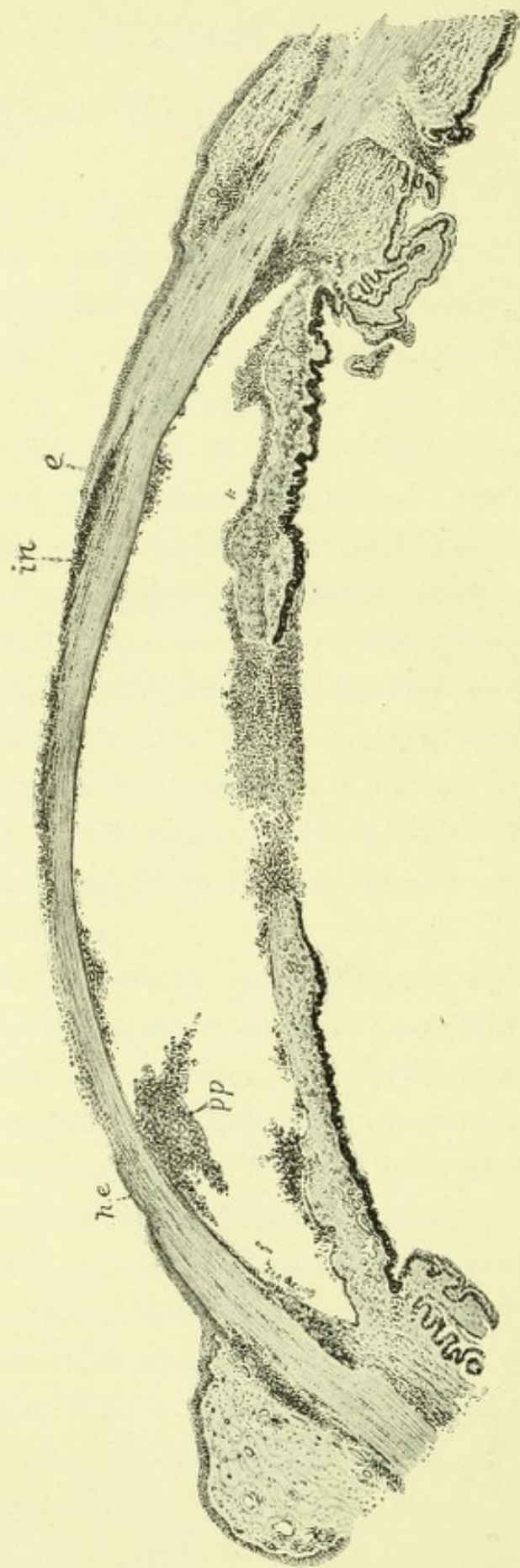


Fig. 9.

SECTION THROUGH CORNEA AND ANTERIOR CHAMBER IN A CASE OF PNEUMOCOCCAL ULCERATION (PROVED BACTERIOLOGICALLY).
Section by Dr. Bronz.

To face page 39.

indeed, they cannot be differentiated; but at a little distance we can see the normal corneal corpuscles swelling, losing their well-marked processes, and developing vacuoles in their interior; their nuclei show changes in the chromatic network and some loss of pigmentation. In the leucocytes, as they pass to the centre, changes in the staining reaction of the nucleus appear, and the cell protoplasm diminishes in amount. In the centre of the infiltrate we see only a mass of closely set nuclei whose origin cannot be recognised from their appearance. The corneal lamellae, which in the periphery are quite normal, towards the centre swell, fray out, and stain irregularly, some not taking up haematoxylin at all, others taking it up very deeply.

The shape and manner of limitation of the infiltrated zone is peculiar. It is a flat disc, with well-defined anterior and posterior surfaces, and an indefinite peripheral margin fading gradually away into the healthy tissues around. The zone of infiltration thus appears to be confined to a limited number of lamellae, and to affect them for a variable extent, but does not pass across to adjoining ones in front or behind. Thus

DESCRIPTION OF FIG. 9.

he is the healing edge of the ulcer, the corneal epithelium is here growing over the floor of the ulcer; at *e* the infiltrate *in* is pushing wedge-like under the epithelium; *pp* is a mass of leucocytes adhering to the back of the cornea. Descemet's membrane is not perforated. The pupil is blocked with an exudation.

In this ulcer pneumococci in pure culture were obtained from under the overhanging edge *e*.

the anterior surface of the infiltrate is bounded by two or three corneal lamellae, which are not much changed, and which separate it from Bowman's membrane; the posterior surface is similarly sharply marked off from the main mass of the cornea. Deep in the parenchyma and immediately in front of Descemet's membrane is a second cellular infiltration, similarly shaped to the principal one, like it having an indefinite margin, and a sharp anterior and posterior wall. Sometimes the infiltrating cells fill up and form a natural injection of the lymph spaces: thus the deep striae seen in the living cornea, sometimes called Recklinghausen's lines, are explained.

Descemet's membrane is sometimes quite normal, at other times it is split up into several layers, between which are round cells similar to those of the deep corneal infiltrate. These cells are probably leucocytes derived from the limbus of the cornea, but there is an opinion, strongly held, that they are derived from the mass of cells lying in the anterior chamber. The endothelium lining this chamber shows a condition of cloudy swelling, the cells are heaped up at places on the back of Descemet's membrane, which is laid bare at parts. This denudation is said to allow the phagocytes to pass through the back of the cornea into its substance, and so cause the deep infiltration zone already described (*früh perforation*).

(b) To study the structure of the fully developed ulcer sections are cut so that they pass through the

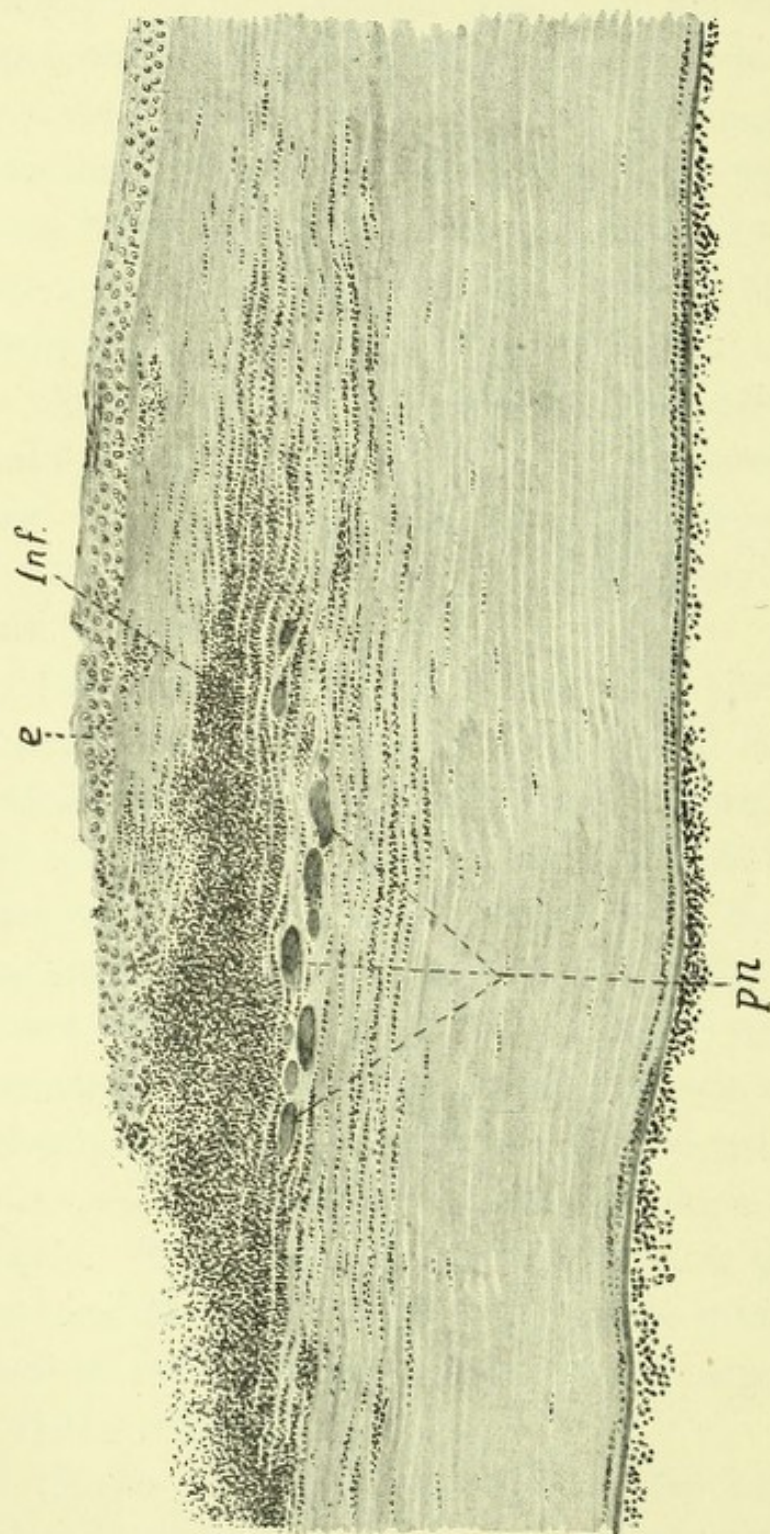


Fig. 10.

SECTION THROUGH ADVANCING EDGE OF PNEUMOCOCCAL ULCER ENLARGED FROM FIG. 9.

Section by Dr. BRONZ.

To face page 41.

clear, transparent, reflecting margin on one side, and on the other, the white, overhanging, advancing border. As could be judged from the clinical examination, the smooth clear margin is formed by young, rapidly proliferating epithelium growing over the broken edge of Bowman's membrane on to the floor of the ulcer; so that here we have only a sunken area due to the loss by necrosis of those structures which lay superficial to the infiltrate. At the opposite side, in the position of the advancing border, the epithelium is very like that which covered the infiltrate in the early stage; all degrees of inflammatory change being shown up to necrosis in the ragged tissue which forms the actual overhanging lip of the ulcer.

In the parenchyma, too, is a condition analogous to that of the early stage. An infiltrated area lies under the epithelium, separated from it by one or two layers of lamellae; it is wedge-shaped in the section, the base being towards the ulcer, and irregular, corresponding to its broken edge, the apex pushed outwards a short distance into the cornea under the surface, thus forming the limit of the infiltrated margin, that dense white outer contour lying beyond the breach of the epithelium. There is some swelling and loosening of the lamellae at

DESCRIPTION OF FIG. 10.

On the right side is the full thickness of the cornea, to the left the base of the ulcer. *Inf* is the cellular infiltration passing like a wedge into the cornea; *e* the epithelium covering the advancing border, and becoming necrotic towards the left. *Pn* are clusters of pneumococci amongst the lamellae, and mark the site of spread of the disease.

this point, which results in the edge of the ulcer being thicker here than elsewhere.

The floor of the ulcer is formed by a thin layer of necrotic tissue,—fibrillated and granular lamellae, with cellular débris staining badly,—beneath which is a narrow reaction-zone, showing well-stained nuclei of leucocytes, and the more faintly staining, spindle-shaped nuclei of corneal corpuscles, each surrounded by a scanty protoplasm passing out in processes between the lamellae. The deep infiltration of the cornea, noticed in the early stage, has greatly increased, and the splitting up of Descemet's membrane, when this does occur, is now quite marked ; sometimes, indeed, this structure is completely broken through, and we have the so-called posterior corneal abscess, communicating with and opening into the anterior chamber.

The endothelium on the back of the cornea is wanting over an area roughly corresponding to the extent of the ulcer. The anterior and posterior chambers contain a mass of fibrin whose meshes entangle many leucocytes ; this forms the hypopyon which is of such a variable extent. The iris and ciliary body show leucocytic infiltration, as also does the retina and choroid to a variable extent. Iritic pigment can be seen in the leucocytes of the hypopyon, and also in the clumps of cells which sometimes form large masses adherent to the back of the cornea (precipitates).

The question of whether perforation of Descemet's membrane ever takes place before the whole cornea is

perforated has been very keenly discussed. It is a fact that the membrane can be destroyed while the central portion of the cornea remains intact, and this is of considerable practical importance; for were it not so we should have to consider every anterior synechia following on an ulcer as proof of complete perforation of the cornea, and therefore of septic infection of the interior of the eye. This certainly is not the case. Also, when a paracentesis is done to remove a very dense hypopyon, it is sometimes found that the fibrinous plug is so firmly adherent to the back of the cornea that it can hardly be withdrawn from the posterior perforation. It is a matter of much less practical worth, whether this perforation takes place from the posterior infiltrate backwards, or from the hypopyon forwards. The important thing is that it sometimes does occur.

Bacteriology.—The pneumococcal ulcer is caused by an infection of the cornea by the pneumococcus. The organism enters the cornea through a breach in its epithelium, the intact surface being proof against infection, and settles down in the parenchyma; from here its toxins spread out, as the organisms increase in number, till they cause a localised necrosis. Sloughing results, and the ulcer is formed. Every pneumococcal ulcer is caused by the pneumococcus, but this micro-organism can cause lesions which do not conform to the type described; every pneumococcal infection is therefore not necessarily a 'pneumococcal ulcer.' Cases of 'ulcus serpens,' or 'typical ulcus serpens,' have been

described due to staphalococcus, rosa hefa, streptococcus, bacillus duplex, hay bacillus, bac. pyocyaneus, and others ; but it is doubtful if their clinical features did not allow of a diagnosis excluding pneumococcal ulcer, and certainly they must have failed in the crucial bacteriological test. Some ulcers which clinically have presented every characteristic of the pneumococcal ulcer have failed to show the organism. In my own experience this has never been the case. Such must be explained by the difficulty in cultivating the pneumococcus. As our methods improve so does the number of these cases diminish, and the nomenclature adopted in this work is more fully justified.

The pneumococcus is an organism which shows considerable variation in its morphology and biology. The actual shape and size of the organism differ within comparatively wide limits, but it is fortunate, for diagnostic purposes, that the capsule, which is one of its most characteristic features, is well developed when the organism is rapidly growing in the human cornea. The agglutination test is undoubtedly the most accurate means at our disposal of establishing the identity of any organism, and as serum from an acute pneumonia causes clumping of a culture of the pneumococcus from a corneal ulcer, the organisms in croupous pneumonia and in these ulcers must be considered the same. As in the lung, so here in the cornea there appear to be several, perhaps four or five, varieties of pneumococci. As yet no morphological differences have been estab-

lished between these, and their importance only relates to the method of serum treatment, which is still unsatisfactory. Aside from such differences there is a great variability in the virulence of the organisms obtained from different ulcers; this obviously is related to the variations in the virulence of the ulcers themselves. To attain an accurate idea of the virulence of an organism we must first consider its adaptation. When a microbe is introduced into a new species, *e.g.* rabbit, after growing for some time in another, *e.g.* man, it may show a very low pathogenic power, which, however, rapidly rises when it is passed through the bodies of a few animals of the new species. This is sometimes called an 'exhaltation of virulence'; it is, however, merely a development of 'adaptation,' which rapidly occurs, and allows the true virulence to become apparent. A true exhaltation of virulence is only very slowly acquired in a considerable period of time. Occasional observations that apparently virulent ulcers contain mild or a-virulent pneumococci when tested on rabbits are thus to be explained: the organisms are adapted to man, but not to the particular experimental animal used.

Pneumococci in the lung produce an antiserum which rapidly passes into the blood. This causes the general symptoms, and, as soon as the quantity of antibody is sufficiently large, arrests the progress of the disease in a characteristic manner. In the cornea, however, the blood supply is poor, very little or no antiserum is

produced, and there is no marked natural tendency to a cure. In this respect, therefore, there is a great difference between the corneal and the lung infection.

The pneumococcus can readily be obtained in a pure condition from the actual advancing edge of the ulcer, even after it has developed for several days, and in spite of the presence of other organisms in the conjunctival sac (see Appendix, p. 184). Involution forms are obtained in the débris from the floor, where the organisms are naturally older and degenerate; as a preliminary to making any examination, this débris should always be washed away.

The sources of infection of a corneal abrasion are :—

- (a) Direct inoculation from a mucocoele or infected conjunctiva. The organisms in the conjunctival secretion are in direct contact with the abraded surface, and readily enter through it.
- (b) The sputum of the patient, or some other person, in misguided attempts to remove a foreign body.
- (c) A handkerchief or finger soiled with nasal secretion being used to wipe the eye after an injury.
- (d) The actual foreign body may carry in the infection, *e.g.* particles of street dust, or dirty chips of stone.

Rarely an ulcer may develop during an acute pneumonia, due to an infection from the patient's own sputum; such ulcers are not typical, being modified by

the presence of antiserum developed in connection with the lung condition.

Unlike some other organisms, neither the pneumococcus itself nor its toxins can affect the intact corneal epithelium. Cases have occurred where, during an acute pneumococcal conjunctivitis, especially if it take on the pseudo-membraneous type, superficial infiltrates have occurred in the cornea; but evidence is still wanting that these are directly due to a pneumococcal corneal infection. Many patients with a pneumococcal ulcer attribute their condition directly to an injury, others will admit the injury when questioned, and as people of the class with which we have to do are very liable to slight abrasions, even if such cannot be elicited in the history, they must be presumed to have occurred. It is doubtful whether there is a true age incidence; the predisposing and exciting causes are more apt to be present in working men and women, therefore the acknowledged frequency of the condition in adults.

The actual position of the pneumococci in the diseased structure is of great interest, as it determines the extent to which radical measures should be used in treatment. Contrasted with other forms of hypopyon keratitis, where the organisms are found in the floor of the ulcer, the infiltrated edge is here the seat of the infection, while the floor is comparatively free from actively growing organisms. Under the infiltrated margin, in advance of its overhanging edge; at the

apex of the wedge where living tissue is being destroyed, amongst the lamellae which are still alive, we find the pneumococci greatest in number, largest in size, and most regular in shape. A few are found amongst the débris on the floor, but they are degenerate involution forms without a sign of active growth. No organisms are found in the epithelium, nor in any other part of the cornea, unless the ulcer be so advanced that it has lost its typical form, then cocci may be found

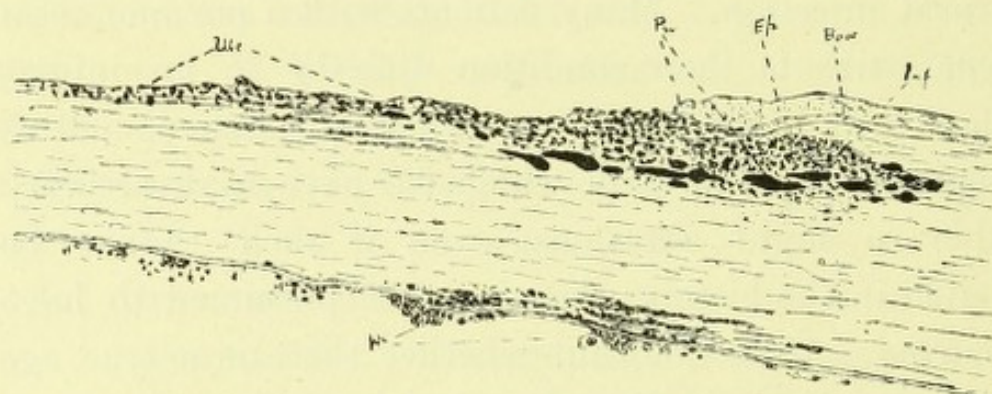


Fig. 11.

SITE OF PNEUMOCOCCI IN CORNEA.

Ulc Floor of ulcer; *Ep* Epithelium.
Bow Bowman's membrane.

Inf Infiltrated edge of ulcer.
Pn Clusters of pneumococci.

deeply amongst the lamellae. The hypopyon is quite sterile so long as the cornea is not perforated.

In the early stages the pneumococci are found in the infiltrate, and form the focus of irritation which determines the collection of cells at that point. The lateral spread of the organism to form a serpiginous ulcer has been explained by the fact that the lymph spaces which pass out between the lamellae do not offer the same resistance to their advance as do the lamellae them-

selves. This arrangement of the lymph channels may be one factor, but it certainly cannot be the only one, as streptococci can quite readily penetrate through the cornea, and it is not clear why one organism should follow the line of least mechanical resistance and another that of the greatest. At first sight it appears anomalous that the spread occurs where the cellular infiltration is densest, just where nature has reared a protective barrier against the attack, and where the Metchnikoff theory would lead us to suppose the greatest resistance would be offered. Römer supplies the explanation of this paradox. When a tissue is attacked by an organism its reaction consists of two distinct processes—histolysis and bacteriolysis. The leucocytes form a histolytic ferment which liquefies the invaded tissue, and causes the separation of the slough. A specific body is also formed called an amboceptor, which has the power of allowing a complemental substance in the blood to attach itself to the bacterium present, paralysing its activity so that the leucocyte can destroy it. This bacteriolysis can only occur when the tissues supply the necessary protective bodies in large amount. In pneumonia such is the case, but when the infection is a small focus in the cornea the development of an anti-serum is very slight; the leucocytes cannot therefore destroy the bacteria present, although their ferment is freely poured out, and solution of the tissue goes on rapidly. The result of the leucocytosis under these circumstances is to cause a more rapid spread of the

ulcer where the cells are thickest, that is where the densely infiltrated margin exists.

The rationale of serum treatment therefore is :—

(a) To supply a direct bacteriocidal substance to the ulcer.

(b) To supply the antibody necessary for bacteriolysis.

The hypopyon consists of threads of fibrin, amongst which are many leucocytes. These are attracted by chemiotaxis from the iris and ciliary body, and in making their way towards the focus of irritation they fall to the bottom of the anterior chamber. The attraction in some cases is so great that they climb up the back of the cornea and collect in a mass behind the ulcer, thus giving the appearance of pus flowing down from behind the ulcer. In some cases they appear to be capable of penetrating through Descemet's membrane and causing a posterior perforation. As they pass through the iris they pick up pigment granules from it; these can then be deposited on the back of the cornea, forming a precipitate on Descemet's membrane, that appearance so badly termed pigmented keratitis punctata.

Diagnosis.—When the ulcer is of moderate size the diagnosis can be made, in many instances, from the mere inspection of the cornea. When there is only an infiltrate it is more difficult; and when the cornea is almost wholly destroyed, it may be impossible to say, from the appearance, to what form of ulceration its destruction was due. The margin is the feature of

greatest value: should this show an infiltrated border, as already described, there is little doubt as to the nature of the ulcer. The condition of the floor is very important, as densely infiltrated areas, and considerable variations in its depth, are against the diagnosis of pneumococcal ulcer. The history of the case is the next point of importance, a 'watery eye' being strongly in favour of pneumococcal infection. A mucocœle or a nasal duct stricture is often the cause of the epiphora, but the possibility of a corneal ulcer not being pneumococcal, although associated with lacrymal obstruction, and a pneumococcal ulcer occurring with a normal sac, must be always borne in mind.

The crucial test in all cases, of course, is the microscopical examination of material removed from the ulcer. Where the smear or cover-glass preparation is not satisfactory, a culture in fluid blood-serum after the manner of Römer may be relied upon with certainty.

Every pneumococcal infection of the cornea does not appear as the typical ulcer described above. In eyes the subjects of glaucoma, trachoma, parenchymatous or eczematous keratitis the clinical picture may vary considerably from the type. So also in cases where a comparatively deep wound in the cornea has been infected. Such variations will not cause any difficulty if the practice of examining all ulcers bacteriologically be adopted.

The *prognosis* is always grave. Untreated, many eyes are lost from panophthalmitis, phthisis anterior,

staphyloma cornea, or acute glaucoma. If prompt and radical treatment be early adopted the majority of such eyes can be saved with some useful vision ; but in spite of every effort in a few cases the whole cornea will be destroyed and enucleation rendered necessary. These are the cases where the ulcerative process spreads with great rapidity, in badly nourished unhealthy subjects, and in eyes previously blind, and suffering from degenerative changes.

Treatment.—It is very unfortunate that so many different methods of treatment have been advocated for pneumococcal ulcer. The absence of a clear knowledge of the exact conditions present, and the life history of the ulcer, rendered it impossible to distinguish the essentials from the details in treatment ; empirical methods were therefore multiplied, and the virtues of special antiseptics extolled, the consideration of how their application was going to attain the desired end being completely ignored. From the account given of the pathology of the condition it is obvious that discussion over the relative merits of the actual cautery or pure carbolic as means of disinfection is quite out of place, until the question of the best means of preventing reinfection is settled. To render a 'septic' area 'aseptic' is surgically not of greater importance than to maintain it in that condition. It is necessary, therefore, not only to destroy the organisms lying in the cornea, but also to prevent the access of fresh infecting material to the wound ; any powerful and penetrating

antiseptic will effect the former, when its use is guided by accurate knowledge, the latter can only be achieved with certainty by cutting off entirely the source of infection.

The indications for treatment are :—

- (1) To remove any irritant from the cornea; to render it aseptic, and to maintain it in that condition.
- (2) To treat symptoms as they arise.
- (3) To prevent and remedy complications, and reduce the severity of sequelae.
- (4) To increase, especially locally, the resistance of the patient to the poison.

Any treatment will be efficient in so far as it fulfils these indications. The first is, however, the criterion by which the value of any procedure must be estimated.

There is a course of what may be termed, 'First aid to a purulent corneal ulcer,' which should always be applied in these cases, whatever may be the special means relied upon to cure the condition. As in other first-aid procedures, the aim is not to cure, but to prevent as far as possible extension, or increase in severity, of the lesion.

By means of a dilator the puncta lacrymalia are enlarged: a fine probe is passed along the canaliculi and across the lacrymal sac until it impinges against the lacrymal bone, and then withdrawn. On pressing over the lacrymal fossa the contents of the sac will pass

out into the conjunctiva. With an Anel's syringe the sac is distended with fluid and again emptied two or three times. The floor of the ulcer is then washed out with a considerable quantity of sterile or mild antiseptic fluid.

A solution of ten or fifteen per cent. protargol should then be syringed into the lacrymal sac, which is left full of this antiseptic. The following ointment can be applied to the ulcer by introducing it freely into the conjunctival sac :—

R/ Hydrarg. Perchlor.	gr. ss.
Iodoform.	grs. xl.
Atropin.	grs. ii. to viii.
Vaselin. pur.	ad $\frac{3}{4}$ i.

Hot fomentations and compresses will relieve the pain, but if this be not very severe, it is best to leave the eye uncovered. The general treatment consists in administering a smart purge and keeping the patient in bed lying on his back, the atropine should be pushed until the pupil dilates, when only enough should be given to maintain that condition; if this be difficult and the congestion of the conjunctiva be intense, adrenalin $\frac{1}{2000}$ added to the atropine will greatly assist its action. The pain is often very severe, but usually rapidly subsides after local treatment; should it persist, leeching the temple and hot fomentations are about the best means of giving relief. Aspirin and pyramidon are often used as analgesics, given by the mouth.

As soon as possible, after an exact diagnosis has been made and confirmed bacteriologically, radical measures should be taken to carry out the first indication for treatment.

The examination of the lacrymal apparatus will have determined the presence, or absence, of blenorrhœa of the sac, mucocœle, or stricture of the nasal duct. If there is any purulent condition, or a complete stricture of these passages, the lacrymal sac should be excised forthwith (Appendix). In the case of incomplete stricture, the duct should be dilated and washed out thoroughly with ten per cent. protargol.

Having thus achieved a condition where there is some hope of maintaining our antisepsis, the actual site of the pneumococci should be attacked. There are two methods of doing this: (1) by the actual cautery—electric, button, or Paquelin; or (2) by strong escharotics, pure carbolic, nitric acid, etc. Each of these methods will give good or bad results, according as they are used with an exact knowledge of the site of the organisms to be destroyed, or not.

The cornea is thoroughly cocainised. The floor of the ulcer, after thorough irrigation, is dried out with pieces of blotting paper, and gently mopped with a dry swab: this will show up very clearly the infiltrated margin, and the overhanging epithelial edge; the clear, almost transparent floor, and the healing parts of the circumference are, by contrast, very distinct. It is only in the densely white parts that the pneumococci

lie, and these are the only parts to be destroyed. The clear part of the floor must be just as carefully preserved as the intact cornea. Fluoresceine used in the manner previously described (p. 4) will assist in defining the exact edge of the epithelium.

With a small sharp spoon the infiltrates are scraped away, especially those under the overhanging epithelial edge; this projecting margin should be clipped away with a pair of fine scissors, and then the whole carefully

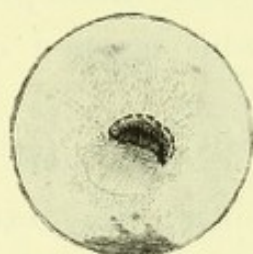


Fig. 12.

THE AREA REQUIRING THE
CAUTERY IS SURROUNDED
BY A DOTTED LINE.

inspected to determine the extent of the infiltrated area, and so the position of the pneumococci. The electric cautery at a dull red heat is then used to thoroughly destroy all remains of infiltrate and to sterilise the area scraped; it is well to encroach a little into the sound cornea at that part where

the advancing border was situated. Fig. 12 represents a pneumococcal ulcer with the dense margin directed upwards; in the sketch a dotted line has been drawn to surround the area requiring cauterisation. Another method of obtaining the same result—the destruction of the pneumococci in the cornea—is by applying pure carbolic acid to the infiltrates. A small sharpened piece of wood is prepared by whittling down a large safety match, the point is dipped in the acid and then applied in the same manner as the cautery: the point must be pushed in under the overhanging

margin and carried to and fro so as to thoroughly carbolicise the whole pneumococcal area,—to assist in this the margin may be cut away, when the acid can be more efficiently applied. After disinfection some of the atropine and corrosive vaseline is smeared on the ulcer. The rationale of cautery and carbolic is the same, and any powerful antiseptic would have the same result if properly applied. The following substances have been used: xeroform, airol, tinctura iodi, sublimate vaseline, formaline, iodoform, aqua chlori, and others.

Sub-conjunctival injections of corrosive sublimate, $\frac{1}{5000}$; saline, one to five per cent., and other solutions of various strengths have been made apparently with the idea of promoting the absorption of the hypopyon. The patient is directed to look upwards, and the needle is pushed through the inferior fornix as far away from the cornea as possible, and from 10-20 minims of the solution are injected. There seems, in some cases, to be a distinct improvement in the ulcer; but no satisfactory explanation has been given of the therapeutic action, if there indeed is any; and this treatment is being abandoned. Oxycyanide of mercury, $\frac{1}{5000}$, has actually been injected into the anterior chamber with a view to curing an ulcer; probably the most glaring example of aimless and misdirected therapeutics, even in this playground of ophthalmology.

Having treated the ulcer, the patient should be kept in bed; no bandage should be applied unless the floor

of the ulcer is very thin and tending to burst. The conjunctival sac should be irrigated every two hours with a large quantity of warm saline or weak corrosive sublimate, 1 : 10,000 solution.

In 1901 Römer brought up the question of the use of an antimicrobial serum, to destroy the pneumococci in the cornea; and followed it up by introducing a serum which was a very powerful preventative against pneumococcal infection. Unfortunately observers differ greatly with regard to the efficiency of Römer's, or any of the other sera in the market, in the treatment of an already established pneumococcal ulcer. In the hands of the introducer the results were most satisfactory, but some later observations have shown that many pneumococcal ulcers pursue their course, quite unaffected by large doses of the serum. This is almost certainly due to the specificity of the antitoxin, and the fact that there are at least some four or five varieties of pneumococci, any of which can cause the ulcer. The method of using the serum is as follows:—5 cc.'s are injected under the conjunctiva away from the cornea, the ulcer is dried out, and the liquid serum is dropped into it every two hours. A subcutaneous injection of 10 cc. serum can also be given into the back. The sub-conjunctival injection is repeated every twelve hours. In early cases of pneumococcal infiltrate, and in some actual ulcers, the process immediately ceased, the 'advancing' edge remained stationary, became very sharply defined from the rest of the cornea, and then

slowly the ulcer cleaned and healed up, with the great advantage of a very thin and almost transparent nebula corneae.

This is the method of passive immunisation now abandoned by its introducer in favour of an active method. At a varying interval after a dead culture of pneumococci is injected into the blood or lymph stream, there is a rise in the antimicrobial power (the opsonic index) of the blood towards that organism. Römer therefore injected dead pneumococci, and found that after a varying period of time there was a presumptive¹ rise in the antipneumococcal power of the blood-serum. In patients with pneumococcal ulcers an improvement in the local condition occurred in about twenty-four or thirty-six hours after such an injection, the time at which the rise in the opsonic index occurs.

As some twenty-four hours would under this treatment be lost, Römer suggests a combined method of active and passive immunisation, and has prepared vaccine and serum for it. 1 cc. of dead bouillon culture of pneumococci is injected intramuscularly, and three or four hours later 5 cc. of antipneumococcal serum subcutaneously. These serum methods of treatment are still in the experimental stage.

When Saemisch first described this ulcer he also introduced a method of treatment — the Saemisch

¹ Presumptive because Römer observed a rise in the agglutinating power of the serum, and therefore considered the antimicrobial power also to have increased.

section—which is still largely practised ; and in some hands seems to give very good results. A narrow-bladed Graefe knife is introduced, with its edge forwards, into the anterior chamber through the sound cornea, a counter puncture is made so that the ulcer lies in front of the knife with its advancing edge at right angles to the blade ; the floor of the ulcer is divided from behind forwards, the hypopyon is evacuated, and the wound is kept open by separating the lips every day with a spatula so that the chamber contents escape (see Appendix).

In some cases where no other form of treatment has succeeded, this little operation appears to have completely checked the ulceration ; it is also claimed that the resulting leucoma is less dense than those after cauterisation.

To open the sterile anterior chamber—a lymph space—through a septic wound, purposefully laying the cut through the most septic part of the cornea, appears utterly opposed to all antiseptic or aseptic principles ; however, a primary infection of such bulbi is rare ; late infection through an adherent iris is a much more frequent source of their loss. Still, until its advocates can supply some explanation of its working, this method of treatment must be reserved for those cases with very large hypopyon, and where the cautery has failed to arrest the progress of the ulcer, where there is almost the certainty of an acute glaucoma, or complete staphyloma of cornea, follow-

ing less radical measures. When the hypopyon is small and has only been present for a day or two, the section should not be made, as such cases will generally improve under the treatment advocated above.

The progress of the case when treated on the lines indicated will depend on the extent of the ulcer, and the rapidity with which it is spreading. Usually there is a very great and rapid improvement, the patient has a good sleep and the pain is much diminished. The surgeon should carefully examine the eye twice a day, especially noting any increase or decrease in the hypopyon, or in the saturation of the advancing edge; if the case be going favourably the hypopyon will lessen and the infiltration around the ulcer diminish. As it is impossible to sterilise the conjunctiva, and although we may have removed the chief source of infection in the nasal duct, reinfection of the ulcer is not uncommon; after a few days of improvement there is an increase in the symptoms, the hypopyon is larger, and at one or more points in the margin of the ulcer a saturated advancing edge begins to form. In such cases the cautery must again be used as in the original condition; and if there be any nasal duct trouble, and the sac has not been excised, such a reinfection of the ulcer should be considered as an imperative indication for that operation. Several complications can occur which may require special treatment. Acute iritis is very common, and seems to be the almost invariable accompaniment of this ulcer in young people.

In old adults it may not occur, but when it does, usually means the loss of the eye. The tension generally rises considerably with the attack, but in spite of that the use of atropine must be vigorously continued, even if necessary to the introduction of one or two dry crystals into the conjunctival sac. Absorption of the atropine can be prevented by pressure on the inner canthus to prevent the drug passing down the canaliculi.

Should the tension remain up when the pupil dilates, it must be brought down by a paracentesis through sound cornea; thus the aqueous, which is very albuminous from the presence of exudate, is drawn off, and the newly secreted fluid being more watery, may be able to filter away. The paracentesis should be repeated if necessary; but if the tension continues rising, as soon as the ulcer is clear and the conjunctiva aseptic, an iridectomy should be done, and the coloboma so arranged for optical as well as anti-glaucomatous effects.

The complications of perforation, adherent iris, vitreous abscess, absolute glaucoma, fistula corneae will be treated of after the other forms of purulent ulcer have been discussed.

Prophylaxis.—The close relationship between blenorrhœa of the sac and *ulcus serpens* is a warning of the danger of leaving such conditions alone, especially in the class of people who are subject to corneal abrasions. The existence of a mucocœle or a 'watery eye' is of grave import, if the presence of pneumococci in the discharge can be shown. For then a husk of grain, a chip

of stone, or a piece of grit striking the cornea, can readily induce a process which may result in complete loss of the affected eye, or a serious diminution in its visual acuity. Every case of mucocœle in such people should have a radical excision of the lacrymal sac; an operation of very slight gravity, though considerable difficulty, and yielding excellent results (see Appendix).

How often do we see a labourer, with a pneumococcal ulcer, whose other eye has been destroyed by the same process, and whose apparently insignificant double mucocœle or nasal stricture had existed untreated for years before the slight injuries lost him first one eye and then the other.

As, in the use of sera for therapeutic purposes, the benefits derived depend greatly on the time when the treatment is begun, in all cases where the development of an ulcer is to be expected, patients with a foreign body in the cornea and a lacrymal obstruction with pneumococci should have their sacs well washed out, and a intramuscular injection of a pneumococcal culture (Römer). The dangers of their condition should be indicated to them, and the necessity of an operation strongly urged. In the labouring class the time necessary for the treatment of a mucocœle by syringing and probing is rarely available. In considering, therefore, the argument for excision of the sac, the alternative of syringing and probing need hardly be considered, as the patients practically never have sufficient perseverance to carry out the treatment, and at best very few can be

cured by that method. The operation of excision of the sac is very satisfactory, the epiphora remaining is almost always much less than before, and is usually inappreciable; while the danger of self-infection of a corneal abrasion is almost entirely removed.

In those cases where a general anæsthetic is considered as somewhat risky, the operation can be very readily done under local anæsthesia.

CHAPTER IV

ATYPICAL HYPOPYON KERATITIS

UNDER this heading it is convenient to group several quite distinct lesions, whose clinical features are not well known, on account of the infrequency or inadequacy of detailed examinations in those cases which have been reported. They bear some resemblance to the pneumococcal ulcer, and are generally shelved as though unworthy of further consideration, being labelled *ulcus serpens* with unusual bacteria present, or some similar designation equally inaccurate. From this chaos it is possible to rescue the skeletons of what appear to be several distinct clinical entities, but our present knowledge provides but little flesh to clothe them with. In the hope that accurate descriptions may, in the future, be made of these conditions, our small knowledge and our great ignorance of their clinical features, pathology, and methods of treatment will be indicated. Here the bacteriological basis of classification is of great value, as it gives a definite ground on which to subdivide this collection ; and it will only require careful and accurate description of all ulcers in their bacterial classes to render a complete account of each one available.

Leber was the first to apply bacteriology to determine the cause of an unusual ulceration, and was able to add to the literature a very full account of that rare disease keratomycosis aspergillina. As we have seen, Uhtoff and Axenfeld treated the pneumococcal ulcer in the same way. Less successful efforts have been made to isolate *Streptococcus*, *Pyocyaneus*, and Friedländer's bacillus forms of ulceration as distinct clinical entities; and with regard to some other organisms we can only record the fact that they have been cultivated from the ulcerated cornea.

The classification of this group is naturally very unsatisfactory, but the following method appears to assist the study of these various ulcers:—

- | | |
|----------------------------------|------------------------------|
| 1. Ulceration due to | <i>Streptococcus</i> . |
| 2. " " | <i>Pyocyaneus</i> . |
| 3. " " | { <i>Friedländer's Pneu-</i> |
| | { <i>mobacillus</i> . |
| | { <i>Bacterium Coli</i> |
| | { <i>Commune</i> . |
| 4. " associated with | { <i>Staphylococcus</i> |
| | { <i>Pyogenes</i> . |
| | { <i>Rosa Hefa</i> . |
| | { <i>Hay Bacillus</i> , and |
| | { others. |
| 5. Keratomycosis Aspergillina. | |

It must be clearly understood, that while the typical pneumococcal ulcer is always due to infection with the

pneumococcus, this organism can cause destructive changes in the cornea, which do not present the appearances already described. Infection by the pneumococcus of a previously vascularised cornea, or of a deep wound of the cornea, or in a marasmic subject, may give rise to a modified ulcer.

Such cases, however, when carefully studied, do not present any serious difficulties, and do not require a separate description; but as they may be confused with members of this group, their occurrence must be borne in mind.

THE STREPTOCOCCAL ULCER

The streptococcus pyogenes can cause an ulceration of the cornea of a severe type, associated with considerable hypopyon. The number of cases which have been fully described clinically, and also studied bacteriologically, is too small to allow of any very particular statement of their features. Hertel, however, has supplied very full descriptions of two such ulcerated corneae, which he examined microscopically, and which presented many features in common; while Fuchs describes this organism as being the infective agent in some cases of ring abscess of the cornea, a form of infection which appears to be produced by any very virulent organism.¹

The streptococcus is well known as the cause of acute conjunctivitis of a very virulent type: it is also present in acute dacryocystitis. Neither of these conditions,

¹ See p. 72, pyocyaneus infection.

however, directly lead to the formation of a streptococcal ulcer, and there is no evidence to indicate how the organism actually does gain access to the cornea; from analogy with the pneumococcal ulcer, a chronic infection of the conjunctiva may be presupposed, a corneal abrasion then allowing the organisms to penetrate the epithelium. The early stages, the method of development and spread of such ulcers, have not yet been observed; the fully developed condition, however, is fairly well determined and sufficiently distinctive to justify its description.

The appearance of the ulcer and the rapidity of its spread depend upon the virulence of the organism which is present. Compared to the pneumococcal ulcer, this form is more acute, and destruction is usually more rapid. The conjunctival reaction is intense, the œdema and congestion being very marked. In the cornea there is a considerable loss of substance, tending to be circular and centrally placed: around this the surface of the cornea is dull, the margin of the ulcer being swollen and having a ragged edge all round. The base of the ulcer is densely opaque, white or yellowish in colour, irregular in depth, and appears to consist of a mass of necrotic corneal tissue, which is being cast off. There is usually a perforation at some part of the base, the aperture being plugged by a mass of lymph which may be blood-stained; the iris may be prolapsed through the aperture. If perforation has not occurred, Descemet's membrane may be bare and

bulging forward into the ulcer. Round the ulcer the cornea is densely infiltrated for a short distance from the actual broken edge: this opaque zone is sharply limited from the remaining cornea, and is about a millimeter broad; it may be possible to determine the fact that it extends further into the sound cornea in the deeper layers than at the surface. The actual margin of the ulcer consists of loosened lamellae; it is not overhung, and during the advance of the process healing is not especially observed at one part while destruction spreads at another. The ulcer discharges a thick yellowish pus with necrotic débris in it.

The condition of the anterior chamber will be difficult to examine, on account of the opacity in the cornea; before perforation occurs there is always a large hypopyon, after perforation this disappears, the aperture being plugged by fibrin and iris. The depth of the chamber varies: at the perforation the iris is adherent to the back of the cornea, and at other parts the aqueous collects and re-forms the space.

Pathology.—Hertel supplies a very full description of the condition of the cornea in two cases of streptococcal ulcer, one of which had perforated, and the other had not. The floor of the ulcer consists of a mass of necrotic corneal tissue infiltrated with round cells: this extends down to Descemet's membrane, or if any corneal lamellae still remain they are densely infiltrated with leucocytes and other round cells. Extending outwards from the actual ulcerated area is a dense cellular infiltration of the

cornea, which extends wider in the deeper layers than the superficial, showing throughout the type of an infiltration ring, such as is seen in ring abscess and is absent in the pneumococcal ulcer. This infiltrated zone is sharply marked off from the surrounding, almost normal, corneal tissue; and passing towards the ulcer the evidence of necrosis in the staining reactions of the tissues increases gradually till we reach the completely necrotic débris on the floor. Descemet's membrane may remain intact, but is much more likely to be split into layers between which are leucocytes gradually destroying the continuity of the membrane so as to cause a perforation. The posterior surface of the cornea is in places denuded of epithelium, at other places the cells are swollen, clustered together, and show a cloudy swelling of their protoplasm. When perforation occurs the iris covered with fibrin will be found plugging up the aperture. There are remains of the hypopyon. The iris is swollen, infiltrated with cells and coagulated lymph, and on its surface is a thick layer of fibrinous exudation.

Bacteriology.—The organism found is the streptococcus pyogenes: it occurs most plentifully in the necrotic débris on the floor of the ulcer, and can be found in the purulent discharge. It presents the usual features of a gram positive coccus occurring in chains, and grows on agar or blood-serum in the form of small clear colonies. The organisms are pathogenic for the ordinary laboratory animals, killing mice in a

few hours, when the streptococci can be grown from the blood of the animal.

Treatment.—The information which we have concerning the pathology of these ulcers is probably derived entirely from the very virulent cases, and it would be rash to express an opinion as to the efficacy of treatment were the ulcer diagnosed in its earliest stages. As, however, the whole thickness of the cornea can be destroyed in a few days, we certainly are called upon to treat conditions approaching those of the fully developed ulcer. The same treatment of the conjunctiva and lacrymal passages is called for as are recommended in the case of the pneumococcal ulcer (p. 54). The disinfection of the ulcer itself must be performed in quite a different manner: the necrotic material forming the floor of the ulcer contains many organisms, and must be scraped away with a sharp spoon, and the whole surface cauterised either with pure carbolic acid or the actual cautery.

This is the type of ulcer which is most suitable for Saemisch's section operation, and if improvement does not follow immediately on cleaning the ulcer, this operation should be performed. The general treatment for pneumococcal ulcers already given should be carried out.

An adherent leucoma is almost certain to occur, even in the most favourable cases, and necessitate an iridectomy.

ULCERATION DUE TO PYOCYANEUS

This organism is only rarely found in connection with corneal ulceration, and the early stages of the conditions present in such infections have not been observed, so that our knowledge concerning the mode of onset and development of this type of ulcer is very meagre.

Infection with this organism has been recorded under two different clinical pictures: by Dr. Hanke in the form of ring abscess, and by the present writer as a necrotic ulcer of great virulence. It is not possible to say whether or not these necrotic ulcers were derived from some form of deep infiltration which might be considered as corresponding to a ring abscess. One case was examined by Professor Sattler three days after an injury, and even then there was a large necrotic ulcer already present; the transition, if such occurs, must therefore take place very early. The infection usually follows an abrasion or other wound of the cornea, but has been recorded without any known injury. The early stages have not been observed, as they rapidly pass into a fully developed ulcer. The patient suffers from severe pain and sleeplessness; at an early stage in the disease an oedema of the lids appears; on separating them the conjunctiva is found to be swollen and chemotic, there is a greenish yellow purulent discharge containing necrotic flakes. A large area of the cornea is occupied by a round ulcer, and

what remains unbroken is dull and densely infiltrated. The margin of the ulcer is thickened, the epithelial edge ragged, and the edges of the broken lamellae loose and swollen; the floor is covered by a greenish yellow pus containing débris of necrotic tissue. When this is removed the floor is seen to be irregular and absolutely devoid of any light-reflex; the lamellae forming it are in a state of necrosis, densely opaque, yellowish white in colour, and flaking off on being touched with a probe. The ulcer progresses rapidly to involve the whole cornea except for a narrow margin at the extreme periphery, and strikes deeply till Descemet's membrane is reached. From this focus of infection the toxins spread rapidly into the deeper parts of the eye, and give rise to the clinical appearances of panophthalmitis. In a few cases an actual abscess has formed in the vitreous. The general symptoms increase, and protrusion of the bulb occurs, with increase of the œdema of the lids and conjunctiva.

Professor Fuchs explains the ring abscess of the cornea as follows: 'Bacteria enter the eye by a perforating wound, and cause septic infection of the deeper parts. From the circumcorneal vessels leucocytes wander in to combat the toxins, which are spreading outwards from the deep focus, thus we have the infiltration ring formed; as the destructive processes are more vigorous than those of repair, necrosis results, and the ring abscess, with a central mass of necrotic cornea, is produced.' It is obvious when we have to

deal with an organism such as pyocyaneus, which produces a large quantity of very virulent toxine, that the centre of positive chemiotaxis will be more deeply placed than would be the case were the infection confined to the superficial layers of the cornea; in some cases of pyocyaneus infection, circumscribed abscesses have been found in the vitreous, demonstrating this tendency to penetration. It is not surprising, therefore, that this infection can present the picture of a ring abscess of the cornea. It was in connection with several such cases that Dr. Hanke described a bacillus, since shown to be the Bac. Pyocyaneus.

Bacteriology.—The bacillus pyocyaneus can be very readily obtained in the discharge from the eye, or in the débris from the floor of the ulcer. It is also obtained in the hypopyon taken from the anterior chamber, even when there is no obvious breach in the cornea. Pure cultures have also been obtained from abscesses in the vitreous. We are quite unaware of the source of the organism, for, although it has been found in dacryocystitis, there is no evidence to show that the cases of pyocyaneus ulcer previously had such an infection. The probable means of infection is through a superficial abrasion. Cultures taken from the hypopyon have shown that the organism is present in the anterior chamber. It is certainly a difficult operation to obtain this pus without conjunctival contamination, but the observations of Professor Sattler, who found localised abscesses in the vitreous, appear to be quite conclusive.

The signs of panophthalmitis are due to the rapid diffusion of toxines, which even pass through the sclera and cause an acute tenonitis. The organism varies considerably in size and shape, is motile, gram negative, and on agar (neutral in reaction) produces a greenish fluorescence; from the medium a sky-blue pigment pyocyanin can be extracted, which is diagnostic (Kölle und Wasserman).

The *treatment* is unsatisfactory. The best results obtained have been the retention of the globe with a vision corresponding to hand-movements. Considering the great danger of deep infection of the eye, if that be not already present on commencing treatment, the aim of the surgeon should be to avoid excision. Saemisch's section appears to be the best procedure; we probably have to deal with a septic cavity—the anterior chamber—which cannot be disinfected. Applying the principles of surgery to such a case, drainage is clearly indicated. A few hours are quite sufficient to make a bacteriological diagnosis, after which the operation should follow immediately. The wound should be opened daily, and the contents of the anterior chamber evacuated.

Hot fomentations, repeated washing out of the conjunctival sac and floor of the ulcer with cyanide of mercury lotion, are of assistance; atropine should be pushed vigorously.

Secondary glaucoma is very likely to occur, and when the ulcer has healed an iridectomy for optical or preventative reasons may be necessary.

ULCERATION DUE TO FRIEDLÄNDER'S BACILLUS
(THE PNEUMOBACILLUS)

This organism has been found in a few cases of ulceration of the cornea, and there is considerable reason to suppose that it can be the cause of ulceration under certain circumstances. Certain facts about these ulcers have been observed which tend to show that they have characteristics in common; whether these are sufficient to allow even of a probable diagnosis of the definite organism is open to question. They certainly do differentiate these ulcers from those due to the pneumococcus.

We have records of the pneumobacillus in cases of dacryocystitis, in conjunctivitis, especially ophthalmia neonatorum, and in chalazion; but none of these assist us in explaining how the organism gets into the cornea. Inoculation of the cornea of animals with this organism results in ulceration, and it appears quite reasonable to suppose that this organism, where it gains access to the corneal tissues, can cause ulceration, and that in the recorded cases the destruction of the cornea was in fact due to its action. It is, as yet, quite impossible to draw any definite clinical picture of this form of ulceration, but in the histories of recorded cases there are one or two points worthy of note. There is severe pain radiating through the head and resembling that of an iritis. The loss of substance is round, spreading outwards as well as deeply, so that a

shallow funnel-shaped depression results: the destructive process seems to progress equally in all directions; there is therefore no advancing edge. The floor of the ulcer is covered with gelatinous débris; there are usually no signs of healing at any part of the lesion.

The amount of hypopyon is very great, and the evidence of severe plastic iritis well marked. Unless seen early there is a great difficulty in dilating the pupil; and when the corneal condition clears, there is usually a mass of organised lymph lying on the lens (*seclusio pupillae*), and an iridectomy will be necessary to establish circulation. Along with the ulcer of the cornea is usually an acute conjunctivitis.

How the pneumobacillus gains access to the cornea is merely a matter of speculation; very probably it comes from the nasal passages through the lacrymal sac or by external contamination, as does the pneumococcus. It is not clear whether a corneal abrasion is necessary or not; Gourfein obtained ulceration in rabbits as the result of inoculation, where no actual breach of the surface could be found, after the cornea had been bruised through the closed lids. It is very doubtful indeed if his precautions were sufficient to exclude the possibility that, in fact, a breach of surface was present, but undetected.

Bacteriology.—This organism belongs to a group of gram negative bacilli growing on solidified blood-serum without causing liquefaction, and causing septicæmia in animals.

In some cases other organisms have been present along with the pneumobacilli, but on separate cultivation and virulence testing they have been shown to be a-virulent. The organism grows readily on the ordinary media, and is with difficulty differentiated from the *ozæna bacillus* and Zur Nedden's bacillus (see Appendix).

The *treatment* of these ulcers is satisfactory, considering their apparent severity. The nasal passages should be douched with an antiseptic lotion, and the lacrymal sac and nasal duct syringed through very frequently with the object of preventing continuous infection of the eye. The whole of the conjunctiva should be thoroughly painted with protargol 15-20 per cent. The ulcer should be washed out, the débris scraped away, and the base and margins swabbed out with protargol 30 per cent., silver nitrate 20 grs. ad $\frac{3}{4}$ i., or pure carbolic acid. Xeroform or airol can be powdered into the base of the ulcer, and some atropine and sublimate ointment introduced into the conjunctival sac. Leeching and hot fomentations are very useful. The latter must be renewed constantly, and the discharge washed out of the conjunctival sac with some hot sterile fluid.

The progress of the ulcer is usually favourable, the hypopyon diminishes, and the symptoms decrease in severity. Considering the extent of ulceration, the resulting nebula is not at all dense, its edges especially being thin; it will clear considerably and allow of fair vision with or without an iridectomy. This

operation will probably be often found necessary, as the pupil may be blocked by exudate, and an iridectomy will relieve the tendency to secondary glaucoma as well as improve the vision.

OTHER FORMS OF ATYPICAL KERATITIS

From corneal ulcers, which appear to have been primary, other organisms have been cultivated, amongst which have been: *bacterium coli commune*, *staphylococcus pyogenes aureus*, *albus*, and *citreus*, *rosa hefa*, *hay bacillus*, and other organisms not sufficiently identified with any known bacteria. Some of these have proved virulent, but in many cases no virulence tests were made.

The present state of our knowledge merely allows us to say that these organisms have been found. There is no information on which to base any clinical picture, or rational special therapy. The infections with *staphylococcus* are usually mild in nature and yield readily to simple antiseptic treatment.

Bacterium coli commune will be again referred to under the heading of Keratomalacia.

KERATOMYCOSIS ASPERGILLINA

This affection of the cornea is usually classified as an atypical hypopyon keratitis from its resemblance to the pneumococcal ulcer. Although one of the rarest infections of the cornea, it was the first to be classified on an etiological basis. For our early knowledge about it

we are indebted to the genius of Professor Leber, who, recognising in a case some differences from the 'ulcus serpens' previously described by Professor Saemisch, removed some of the necrotic material from the cornea and found the infective agent to be a mould. Since Leber's article in 1879 several cases have been added to the literature, but our knowledge of the disease practically remains where he left it.

This infection of the cornea is rare, only some twenty cases having been recorded; there is very good reason, however, to suppose that many cases pass unrecognised. It is worthy of note that four of the recorded cases have been under the charge of Professor Axenfeld and three under Dr. Gentilini, so that were the features of this disease better known, and the cornea more rigorously examined, especially bacteriologically, more cases would be found.

Mycotic keratitis results from the infection of the cornea by a mould *Aspergillus Fumigatus*, which is generally introduced on a foreign body of vegetable origin. The mould grows amongst the corneal lamellae, forming a felted mass of interlacing *hyphae*, and causing necrosis of the invaded tissues. The reaction of the cornea to this irritant, and the appearance of the sequestrum formed, give rise to the characteristic features of the disease.

In the majority of the cases recorded there has been a history of some foreign body of plant life in the cornea, *e.g.* a husk of corn, a splinter of wood, a

grass seed, the dust from a sack which was shaken, the dust in a mill, a spike of a chestnut, a blow from a pear. The foreign body must be driven against the cornea, or rubbed well into a breach on its surface, before infection results. If a splinter of an infected substance be embedded in the cornea infection is almost certain to follow, and in several of the recorded cases the infecting *corpus alienum* has been removed by the surgeon, and found to have the fungus growing on it (Fig. 14).

The incubation period varies from a few days to several weeks, and the onset of the disease is marked by severe pain in the eye and loss of vision. The cases can be divided into two classes, the typical, severe attacks as described by Leber, and the milder 'fascicular' form recorded by Axenfeld. In the former an intense keratitis with hypopyon develops and the eye is always seriously damaged. In the latter there is a limited necrosis without hypopyon, and the final result is favourable.

In the severe cases (Leber, Fuchs, Schirmer) there is considerable conjunctival congestion and some papillary hypertrophy. The whole of the corneal surface is dull, and in the centre there is an area of from three to eight mm. in diameter, where the reflex is entirely lost. This area seems sunken from the general level of the cornea, and presents a peculiar dry earthy surface, which nowhere shows any light-reflex. It may vary in colour from yellow to grey. It is surrounded by a ridge

where the epithelium though intact appears very œdematous, and the rest of the epithelial surface shows a dulness diminishing towards the margin of the cornea. The ulcerated area is densely opaque, forming a yellowish white infiltrate which comes quite to the surface where the epithelium is deficient, and is overlapped at its margin by the ridge seen on the surface. The general appearance of the infiltrate is as though it were made of dirty wash leather covered by a dry scum; the margin is not undermined. Around the opacity there is usually an infiltration ring well marked, and the rest of the cornea is smoky; in some cases, however, it is so infiltrated as to be opaque and milky (Basso).

The hypopyon varies in amount. The condition of the iris cannot usually be examined on account of the corneal opacity. The general appearance is that of a sequestrum lying in the inflamed cornea.

If such a case be treated expectantly by means of hot fomentations, atropine, leeching, etc., the necrosis spreads into the previously sound tissue, the swelling of the conjunctiva increases, and even affects the lids, finally the whole cornea will be destroyed.

In the mild 'fascicular' cases the appearances are different. The epithelium is raised up over a small area and shows a small breach on its surface; the elevated area is limited by a furrow, and around this a dull reflex gradually fades away into one of normal brightness. In the substance of the cornea lies a round

yellowish white button of infiltrated tissue, very sharply marked off all round: its surface has the same dry earthy appearance where the epithelium is absent, and under the surface furrow is a distinct demarcation line and infiltration ring. To this nodule a leash of deep and superficial vessels pass from the adjacent edge of the cornea, giving the picture a certain resemblance to

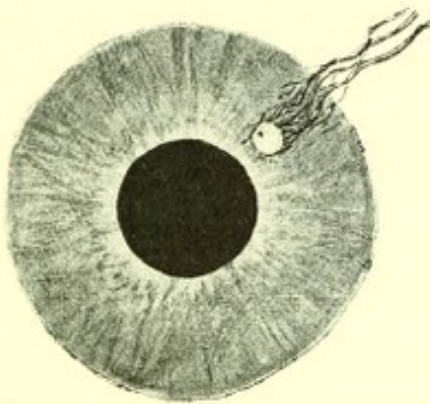


Fig. 13.



Fig. 14.

KERATOMYCOSIS ASPERGILLINA.
A mild case.

Fig. 13. Appearance of Cornea.

Fig. 14. Sequestrum attached to a small splinter of wood.

that form of keratitis eczematosa called the snail-track ulcer or wandering phlyctenule. There is no hypopyon, and the iris and pupil may be normal (Fig. 13). The further course of such cases untreated has not been observed, but it is probable that they either pass into the severer form or else the reaction is sufficient to set free the sequestrum, which is cast off, leaving a facet. Healing then takes place and a small scar remains.

The *diagnosis* is completed by removing the whole of the sequestrum if it be small; or a part of it if

large; and examining the material microscopically. In it we see a mass of necrotic corneal tissue, thick set throughout by clear, refractile, branching threads, which stain badly or not at all with the usual dyes, but take up Weigert's stain intensely (Fig. 14).

Pathology.—Several corneae have been examined, and the conditions found have been fairly constant. There is an ulcer on the surface with destruction of the epithelium and Bowman's membrane: the floor of the ulcer is formed by a felted layer of the fungus, under which the remains of corneal lamellae, necrotic and unstained, can be seen with many mycelial hyphae amongst them. The necrosed tissue separates readily from the underlying lamellae, but at its margins it is firmly attached to the cornea around.

Fructification organs are never found in the cornea. The temperature of the anterior chamber is about 31°C ., which is much below the optimum for the growth of this fungus, and decidedly lower than that necessary for its sexual reproduction.

The fungus can be readily grown on blood-serum at 36°C ., when the club-shaped conidia covered with spores are easily distinguished.

The *treatment* consists in the removal of the sequestrum. In the mild cases this can be done readily enough with a broad needle, when the whole of the infiltrated mass comes away. Any spots where adherence is rather firm can then be cauterised either with the galvano cautery or pure carbolic acid. It is much more

difficult to treat the severe cases ; but in some such the eye has been saved by cutting the sequestrum away with a knife and scissors—this can be done without gouging through the whole thickness of the cornea—when successful healing takes place, leaving a dense white leucoma. It is obvious that a Saemisch section is unsuitable for these cases. Hot fomentations must be avoided, as must anything in the nature of a poultice, as that by raising the temperature favours the growth of the fungus and the spread of the lesion.

CHAPTER V

MOOREN'S ULCER

RODENT ULCER

ULCUS RODENS

L'ULCÈRE RONGEANT

CHRONIC SERPIGINOUS ULCER

THE condition, which is known by these various names, is a rare form of chronic non-purulent keratitis, belonging to the marginal group. The superficial layers of the cornea are destroyed and replaced by connective tissue: a leucomatous scar is thus produced. The process begins at the margin, and slowly spreads over the cornea; a scarred area is produced which enlarges so as finally to replace the whole cornea, being bordered as it increases by an infiltrated line. It is a primary corneal condition of unknown cause.

The first description of this condition was published by Bowman in 1849. Mooren recorded cases in 1867, and emphasised its chronic and intractable nature. In England his name is generally associated with the disease. In recent years a considerable number of cases have been recorded: these were collected by Nettleship

in 1902; and as several eyes have been pathologically examined, our knowledge of the symptoms, course, and pathology of the disease is considerable, although its cause is still a mystery, and its treatment unsatisfactory.

Owing to the duration of the process, accurate and exact observations can readily be made of its course and mode of spreading. In several cases, while the patient has been under careful observation, the condition has commenced in the second eye; the mode of onset has, therefore, been determined. The phenomena observable in such cases were quite similar to those which can be seen when, after apparent healing, a recurrence takes place in the originally affected cornea. With our ignorance of the cause, there must be associated some doubt about what are the diagnostic symptoms, and what are merely associated signs; chronicity is generally taken as the deciding feature in doubtful cases, and hence, in the early stages, a definite diagnosis of Mooren's ulcer cannot always be made, as naturally the lesion is then quite recent. It is useful to have some standard to apply to such cases, and it is generally accepted that ulceration of the cornea which persists, and advances for three months, and which presents certain features to be described, is undoubtedly of this kind. If, however, a termination is reached in a shorter period, it may be doubted whether the condition really was a Mooren's ulcer, although of course treatment may cut short almost at once cases which really were of this class. Mooren's ulcer commences by a slight

change in the superficial parts of the cornea; the epithelium becomes hazy and loses its sharp light-reflex, so that at one part the surface is dull and matt; in the substance of the cornea there is a loss of transparency, in the form of a greyish hazy clouding of its superficial layers. This change occurs only in a small part of the whole corneal surface, and near its margin, the site being marked by an injection of the corresponding episcleral tissues. The patient complains of considerable pain in the eye, with photophobia and lacrymation. In some cases a loss of sensibility of the cornea has preceded the appearance of this cloudiness; but in others, throughout the whole course of the disease, normal corneal sensibility could be determined in all parts not actually destroyed by the ulcerative process. The sensibility of the conjunctiva is not impaired; if there be any change, it is in the opposite direction. The cloudiness of the cornea is seen to consist, when under the magnification of a loupe, in fine grey flecks and spots, not unlike those in an interstitial keratitis, which can be recognised as separate points of infiltration; their margins are irregular and ill-defined, so that they are not absolutely discrete, yet there is no difficulty in seeing that the infiltration is not an even diffusion but a collection of separate points. When the infiltration has reached some considerable extent, the epithelium over it gives way, and is cast off; thus a shallow clear superficial ulcer is formed.

When the ulcer is formed there is an abatement in

the pain and photophobia. The symptoms soon recommence with the formation of fresh infiltrations, just outside the edge of the ulcer: these break down, and so increase the size of the lesion. It is in this stage that patients are usually seen, and as a conservative treatment is at first generally pursued, the slow and intermittent advance of the margin of the ulcer can be observed. The cornea remains clear, except where the ulceration has actually occurred: here we find the smooth surface replaced by a wavy, irregular, but for the most part reflecting area in which vessels can be observed. At some places this surface may pass evenly over into the normal, showing a continuous intact epithelium, but at others there is a very distinct break in the reflex, caused by a well-defined line limiting the ulceration, and forming its advancing front. 'The advancing edge of the ulcer forms a narrow sinuous whitish band, level with the cornea beyond, and of which the two ends are in advance of the centre, so that the diseased surface at this stage is roughly crescentic, and sometimes semilunar' (Nettleship). Behind this advancing line the epithelium, from the opposite side of the ulcer, grows forward to cover up those layers of the cornea which remain undestroyed. The rapidity of destruction and regeneration of epithelium varies very greatly at different times and in different cases, so that the extent of the actually ulcerated and uncovered area also varies greatly; but whatever its size or shape, that edge which advances into the sound cornea has certain character-

istics which differentiate this form of ulceration from those which otherwise somewhat resemble it. The amount of infiltration is much less than is seen in a pneumococcal ulcer. The colour is a dull greyish white, not the intense yellowish white of the acute condition, whose advancing edge resembles it in being undermined, although to a much less extent. In Mooren's ulcer the epithelium forms a flap of from one to three millimeters extent, under which a probe can readily be passed into a cleft in which lies some granular débris; but even this undermined part of the cornea is fairly transparent, and the presence and extent of the undermining is not at all obvious until carefully searched for. Behind this edge there is a belt of cornea over which there is no epithelial covering, and which therefore stains with fluoresceine; on the other side of this area is the superficially scarred cornea with an epithelial covering, which does not take up the fluoresceine stain. During the periods of intermission, which may be brought about by treatment, the epithelium grows over this bare area, and then we find a patch of cornea, irregular in thickness, covered with a fine, slightly vascular leucoma, showing marked diminution or complete absence of sensibility: the extent of this leucoma depends on the amount of progress the disease has made; it is usually situated peripherally, and is pathognomonic of the condition. Intermissions may last for months; but untreated, and sometimes in spite of everything that can be done, recurrences take place,

and destruction proceeds until the whole surface of the cornea is replaced by scar tissue. In most cases the disease then dies out, but in exceptional instances recurrence has taken place in the scar; in such there is a tendency for the ulceration to penetrate deeply into the cornea and expose Descemet's membrane, or even penetrate through it.

The final condition of a cornea whose surface has been completely eroded by this disease is by no means so bad as would be expected; the leucoma clears up to a marvellous extent, and after a considerable lapse of time a visual acuity of $\frac{6}{36}$ or even $\frac{6}{24}$ is not uncommon, provided that no complications have occurred which would affect the vision.

Associated with the corneal ulceration is a varying degree of iritis, which may be insignificant and clear away completely, or may be a severe plastic form which closes the pupil. In some cases there is a slight hypopyon. The pain is sometimes very severe, and, as the disease may continue for many months, interferes with the general state of health and nutrition of the patient; considerable relief is experienced, in the worst cases, when the ulceration, having destroyed the whole corneal surface, has spent itself. Such patients rapidly regain their strength and weight.

Pathology.—The material available for examination consists of several eyes, which have been excised to relieve the suffering of the patient, or have been obtained post-mortem. The complete accordance of the various

with small cells. At the base of this flap the infiltration is more intense, and passes out a short distance into the sound tissue. When the ulcer has healed, the cornea may be thicker than normal; its epithelium is irregular, sending down processes into a layer of fibrous scar tissue lying below it. The deeper parts of the cornea are normal, and between the lamellae and the epithelium is a new-formed layer of slightly vascular fibrous tissue, derived from the granulation tissue which covered the floor of the ulcer when healing was in progress. Bowman's membrane is absent over the whole area. In those places where the cornea has been healed for a long time the scar thins so that the whole thickness is much less than the normal cornea. The great tendency for the epithelium to spread over the ulcerated area is shown by the occasional finding of epithelial processes even under the advancing edge (Fig. 15).

The etiology of the disease is quite unknown. From analogy a microbic origin can be presumed, but, although many ulcers have been examined bacteriologically, in only one instance has the claim been made that a specific organism has been found. Andrade discovered a motile organism in a case he examined, and suggested that it was the cause. Subsequent investigators have failed to discover his or any other organism which could produce the lesion.

Some cases have followed an injury, and some have occurred during periods of lowered vitality; but a glance at the records of the published cases shows that

such factors can only have a very slight influence. The evidence appears to be in favour of a microbic cause, and repeated re-infection.

The *diagnosis* depends on the clinical history and the physical signs. The important points are (*a*) the peculiar undermined advancing border; (*b*) the anæsthesia of the scar remaining; (*c*) the absence of purulent infiltration; (*d*) the absence of any specific organism; (*e*) its chronicity.

The *prognosis* is bad, but depends on the result of treatment. If the ulceration can be arrested the final vision may be normal; if it continues the final result will probably be about $\frac{6}{60}$, and that after the painful and distressing condition has existed for a year or more.

Treatment.—The rarity of this disease, the ignorance of its cause, and the variability of its response to treatment, result in our having almost as many therapeutic methods as there have been investigators in this field. The morbid process goes on under the overhanging edge, so that in all cases this flap of epithelium should be removed to allow the disinfecting or caustic substance to reach that point where its application is necessary. With a fine probe the amount and site of the undermining is determined, and the edge is clipped away with a curved iris scissors, so that the floor of the ulcer is everywhere completely exposed. With a small sharp spoon the thin layer of granulation tissue is scraped away, so that bare corneal lamellae are exposed.

By staining with fluoresceine we can ensure that no islands or outlying bays of the ulcer have escaped this scraping. The whole surface is then lightly touched with the cautery, or pure carbolic acid. The site of the advancing edge is thoroughly cauterised till sound cornea is reached. In a few days, when the slough from the cauterised surface is completely cast off, conjunctival flap should be grafted into the cornea in the manner described in the Appendix (p. 183); while the slough is separating, repeated bathings with hot perchloride of mercury solution $\frac{1}{8}$ gr. per $\frac{3}{4}$ i. should be employed to hasten the process. Atropine and iodoform in the form of an ointment should be introduced into the conjunctival sac. In a certain number of cases the disease will be arrested either permanently or for a considerable time; but in the majority of cases the ulceration will recur in the line where the advancing border was destroyed: in such cases the proceeding already described should be repeated. Although the ulceration is usually limited to the cornea, it is possible for the conjunctiva to be undermined, and thus the true ulcerating edge may escape notice. If, however, the precaution be taken to search the whole margin of the ulcer with a fine probe before attempting any curative measures, this peculiarity will be discovered. The treatment is exactly the same as for undermined corneal tissue: removal and cauterisation of the surface. The possibility of a secondary plastic operation being required to replace the conjunctiva should always be

borne in mind when the necessity arises for excising any part of it.

These measures will utterly fail to arrest the progress of the disease in some cases ; after each cauterisation only a short interval of rest is obtained, and then there is another advance into the remaining cornea. The question will then arise, are we to continue these repeated cauterisations or not ? The nebulae remaining after the use of the cautery are always thicker and more opaque than those due merely to the disease itself. So soon, therefore, as it is quite obvious that no means available will stop the spread of the disease all over the cornea, then cauterisation should be given up, so that the scar in the central area of the cornea is only due to destruction from the disease, and not due to the cautery. Many other antiseptics and escharotics have been recommended as applications to the advancing border, and amongst them tincture of iodine seems to have given the best results. This substance is applied to the ulcer after the overhanging edge has been thoroughly dissected away. It is quite worth a trial even if the more powerful escharotics have failed. When ulceration recurs in the scar it often penetrates deeply, and a descemetocœle may result. The best treatment for such a complication is paracentesis through the bulging floor, and cauterisation of the edge of the ulcer ; and, as soon as the surface is quite clean, conjunctival grafting to fill up the defect.

When the whole corneal surface has been destroyed

the vision is very low, finger counting at a few feet being the rule. The amount of improvement which takes place in the course of time, especially if the proper treatment for promoting the absorption of nebulae corneae be employed, is marvellous. A visual acuity of $\frac{6}{24}$ being not at all unlikely, provided that the pupillary area of the cornea has not been cauterised, or that there is a thin part of the scar so near the centre that an iridectomy can bring the pupil behind it.

The operation of iridectomy may be necessary for two different purposes: (*a*) optical, to take advantage of the clearest part of the cornea; (*b*) to open a pupil closed up by iritic adhesions.

CHAPTER VI

BACILLARY ULCERATION

THERE are two distinct infections of the cornea, giving rise to ulceration, which bear sufficient resemblance to each other to allow of their being classified together. These are

- (a) *Diplobacillary infection* and
- (b) *Infection by Zur Nedden's bacillus*.

The former is in London one of the common corneal infections with which we have to deal, the latter is rare, a few cases only occurring in the summer. In the Rhine Valley it is much more common.

Each infection gives rise to various clinical appearances denoting lesions of varying severity, which, without the aid of our bacteriological standard of classification, could not possibly have been associated together.

INFECTION OF THE CORNEA BY THE BACILLUS DUPLEX

When this micro-organism is introduced into the conjunctival sac it gives rise to a form of conjunctivitis, which, although it may vary within considerable limits,

has such marked features that it is now recognised as a definite clinical entity, under various names, as Diplobacillary Conjunctivitis, Marginal Conjunctivitis, Angular Conjunctivitis or Blepharo-conjunctivitis. The vehicles of infection are towels, handkerchiefs, the fingers, or dust; the eye may be infected from some other individual, or, much more commonly, directly from the nose which has previously been contaminated: any error of refraction will conduce to this by causing patients to rub their lids with infected fingers. The ease with which the bacillus may be transferred to the eye by the fingers, when the nose is irritated by its presence, and the eye by an error of refraction, is sufficiently obvious. In a district where diplobacillary conjunctivitis is prevalent (Rostock) the bacilli have been found present in the nasal secretions of forty-five per cent. of the population (Erdman). Although the organism has slight powers of resistance, it is peculiar in that it retains its vitality for a long time when dried, especially if kept in the secretions from the mucous membrane on which it grows; as, for instance, in dried nasal discharges on linen.

Diplobacillary conjunctivitis amounts in London to about two per cent. of the total outpatients at our hospitals. Amongst these cases the occurrence of corneal complications will probably be observed in ten per cent., which proportion could be considerably reduced were the clinical appearances of the disease earlier recognised, and its specific treatment persistently carried out.

The corneal complications which may occur in this infection are various:—*Infiltrates* of varying density and extent, usually marginal in position, but possibly occurring in any part of the cornea. *Shallow ulcers*, with slight or no infiltration, often situated one or two millimeters from the corneal margin, oval and placed concentrically to the limbus. *Deep infiltrated ulcers*, lying centrally, round, passing deeply into the cornea, sometimes even down to Descemet's membrane, with infiltrated base and margin, and associated sometimes with iritis and hypopyon. There is also a form of *superficial marginal keratitis*, which advances from the margin towards the centre of the cornea by means of a very shallow ulcerating edge, followed by vessels coming from the conjunctiva. It is in fact a form of phlyctenular keratitis.

The relation of these varying lesions to the type of diplobacillary conjunctivitis present is worthy of notice. Generally speaking, corneal lesions are not found until after the conjunctival condition has been present for some time, or has recurred several times. They may occur when the conjunctival symptoms are very slight: in such cases we usually have to do with the shallow marginal ulcer, or the superficial keratitis form. When the conjunctivitis takes on the acute blepharitis form with predominant moist eczema and excoriation of the lid margins and skin adjoining, infiltrates are common, often breaking down and having ragged ulceration over them. The deep in-

filtrated ulcer appears to be associated with an injury, and it occurs in the exposed area of the cornea, often in cases where the conjunctival symptoms are of the very mild angular type.

The formation of a corneal infiltration is accompanied by considerable photophobia and lacrymation, as would be expected in a superficial condition: the symptoms resemble those of eczematous keratitis, for which the condition is very often mistaken. When the infiltrate breaks down to form an ulcer, the subjective symptoms diminish, and may entirely disappear. The epithelium grows over the loss of substance, leaving a facet which slowly fills up, and all that remains is a faint nebula; the process of healing is very slow if the condition be untreated, and relapses or recurrences are almost invariably the rule. The ulcer formed in this way is usually oval, shallow, clear or slightly infiltrated on the side away from the limbus, and opposite to it is an area of conjunctival congestion, from which superficial vessels may sometimes pass over into the ulcer itself. Should the ulcer be in the pupillary area, there is very considerable interference with vision; this remains after the ulcer has healed until the facet is filled up, when, in spite of the nebula, there is marked improvement.

The small deep infiltrated ulcer differs in appearance from the shallow marginal one from the first. It very commonly follows an injury; when seen, usually about six to eight days after, there is a round, centrally

placed loss of substance in an infiltrated area of the cornea. The ulcer is comparatively deep, its floor is thin and irregularly infiltrated; sometimes only Desce-met's membrane remains, bulging forwards to produce a descemetocoele; occasionally the floor is thick and covered with necrotic, purulent, yellow material, rich in bacteria. The margin is usually infiltrated all round. There is sometimes an appearance very like the advancing margin of a pneumococcal ulcer, but the progress is rarely so rapid, nor is the edge so much overhung, or so densely infiltrated. In the rest of the cornea there is a diffused loss of transparency, fading as we pass away from the ulcer, near which secondary infiltrates may occur. Around the ulcer an infiltration ring is sometimes seen, and the radial striations from the margin of the ulcer towards the limbus, described in the pneumococcal ulcer, are often found in this condition also. Conjunctival and ciliary injection are well marked. There is an iritis with exudation, and a hypopyon usually of one or two millimeters in height, and with a horizontal upper border.

This form of ulceration resembles that caused by the pneumococci; in fact, they are usually put into the class of *ulcus serpens*. It is, however, very important that they should be kept quite distinct, as the prognosis and treatment of the two differ very widely, and the differential diagnosis can be exactly made. In the great majority of cases this is possible from a consideration of the history and appearances of the cornea. In

all cases it is possible by a bacteriological examination of the ulcer itself. The points of comparison are shown in tabular form :—

INFILTRATED DIPLOBACILLARY ULCER.	PNEUMOCOCCAL ULCER.
History of chronic or recurrent angular conjunctivitis, or marginal blepharitis.	History of 'watery eye,' and often the presence of a mucocoele or nasal duct stricture.
Generally no mucocoele, or nasal duct affection.	
Traumatism frequent.	Traumatism generally.
Small and deep.	Large and shallow.
Outline circular.	Outline serpiginous.
Border not well defined, not undermined, less infiltrated than base.	Densely infiltrated, undermined border advancing in one direction. Base clear, regular.
Floor irregularly infiltrated.	
Iritis of mild type.	Iritis varies greatly in intensity.
Hypopyon small, flat upper border.	Hypopyon usually large, convex upper border.
Other grey infiltrates in cornea, often marginal in position.	Rarely other infiltrates. When present, dense white, round, and rapidly breaking down.
Infiltration ring round ulcer sometimes.	No infiltration ring. Densely infiltrated advancing margin.
Pain sometimes absent, usually slight.	Pain usually intense—insomnia.
Progress usually slow; rarely more than 3-4 mm. in extent after a week's duration.	Progress rapid—whole cornea may be destroyed in a few days.
Diplobacilli in ulcer.	Pneumococci in ulcer.

The most important of these points of difference, and the one of the greatest diagnostic worth, is the con-

dition of the margin of the ulcer. This may sometimes, in the diplobacillary ulcer, resemble that described as typical of pneumococcal ulceration in what might be considered its attenuated form; the resemblance, however, is usually temporary, and in a day or two the deceptive features will have probably disappeared. The progress of such infiltrated borders in the two conditions is very different. The tendency of the bacillary ulcer to spread deeply generally contrasts with superficial spread of the coccal. The mere presence of a mucocœle must not be taken as diagnostic, for the lacrymal sac may contain no pneumococci even when there is a blenorrhœa.

The existence of a marginal blepharitis or angular conjunctivitis is also not sufficient ground for the diagnosis of diplobacillary ulcer, as, of course, in such a cornea a pneumococcal ulcer can develop; such cases probably contain a mixture of the two organisms in the ulcer, and should be diagnosed and treated as pneumococcal. The final proof in all cases rests upon the bacteriological findings.

The group of ulcers called Atypical Hypopyon Keratitis should perhaps be made to include these diplobacillary ulcers. Some writers refer to them as 'Atypical Ulcus Serpens,' which is an exceedingly bad and misleading title. I have placed them in their proper etiological place, and would use the term Atypical Hypopyon Keratitis as a temporary expedient to cover those forms of ulceration with hypopyon

whose bacteriology and clinical appearances have not yet been sufficiently elaborated to allow of their separate description. With this view of the classification it is a distinct advance to remove the diplobacillary hypopyon ulcer from the indefinite group into the well-defined class now under discussion.

In cases where the vitality of the cornea has been seriously interfered with—by lagophthalmos, anæsthesia, keratitis, or in glaucoma—the appearances presented by a diplobacillary keratitis will vary considerably from those described as typical. I have seen a case where the lower half of the cornea was occupied by a terraced perforated ulcer with iris prolapse, hæmorrhages into its base; and an extremely large hypopyon had developed. Here the severity was due to a lagophthalmos, but the bacteriological examination indicated the diagnosis and treatment, which was quite satisfactory.

The *pathology* of these diplobacillary ulcers is uncertain. The shallow clear ulcer seems to begin in what looks like a marginal phlyctenule of the cornea, probably by an infiltrate under Bowman's membrane. This breaks down, the epithelium covering it is cast off, and so the small shallow clear ulcer is formed.

The deep central ulcer seems to follow a definite injury in many cases; and in those where no such history is obtainable, an abrasion of the corneal surface may be presumed. They occur always in those parts

of the cornea—the central and lower regions—where slight injuries are common, especially amongst the labouring classes, who are mostly affected by these ulcers. There is no adequate explanation of their tendency to pass deeply into the cornea. The scanty pathological material available for investigation consists of neglected cases or glaucomatous eyes, and their study gives us practically no more information than can be gathered from the clinical appearances.

Bacteriology.—The cause of these lesions is an infection of the cornea with the *bacillus duplex* of Morax and Axenfeld. The type described by Petit and myself appears to cause identical lesions and is a very slightly modified organism of the same group, to be identified only by slight differences in its culture peculiarities. The organism can be obtained from the ulcers in the manner described later (p. 184), and grows very readily on blood-serum or ascites agar.

The method of infection of the cornea is quite unknown. The deep ulcer with hypopyon probably arises from an abrasion infected from the conjunctiva, but the other lesions seem to occur independently of any traumatism; they are very commonly found quite at the upper margin of the cornea under complete protection of the upper lid. It seems probable that the presence of the bacilli in the conjunctival sac so lowers the vitality of the cornea that infiltrates form under the epithelium, which breaks down,

and the exposed surface becomes infected with the organisms.

Treatment.—The corneal condition being secondary to the blepharo-conjunctivitis, the treatment must be directed primarily against the latter; and the drug which, above all, must be persistently employed is sulphate of zinc. This has a specific action against the bacillus duplex, and the conjunctival condition rapidly yields to its use. Guttae Zinci Sulphatis grs. ii. ad $\bar{3}$ i. instilled into the conjunctival sac every three or four hours is sufficient to cause rapid improvement in all except the deep infiltrated ulcers, in which it is much better to syringe out the floor of the ulcer with the same solution from an Anels syringe. If the general infection take the form of an ulcerative marginal blepharitis, the margins of the lids must be thoroughly cleaned with a warm sodium bicarbonate solution, and then a strong protargol (10 to 15 per cent.) rubbed into the roots of the cilia with a cotton-wool swab. An ointment consisting of Ichthyol Ammon. grs. iv. to x., Zinci Oxidi and Pulv. Amyl $\bar{a}\bar{a}$ gr. xx., made up with Vaseline. $\bar{3}$ i., is very useful for the lids or where the sulphate of zinc is not well borne. A small portion is introduced under the lid and rubbed well into the cornea. The edges of the lids are also anointed two or three times a day.

In cases where there is much papillary hypertrophy we have to deal with a mixed infection, and these cases are benefited by the application of silver nitrate

grs. v. ad ʒi. to the everted and dried conjunctiva, care being taken that the silver salt does not come in contact with the ulcers. Unless there are signs of iritis the use of atropine is not indicated, except in the deep infiltrated ulcers, where, on account of the great tendency to hypopyon and iritis, its use should be routine. The pain is best relieved by hot bathing with plain boiled water as hot as can be borne. Dark glasses give great relief in all cases where there is photophobia and lacrymation.

In a great many cases the nose will be found to be infected with the same organism. There are crusts around the anterior nares, a thin acrid milky discharge irritates the skin around, and there is much thickening of the nasal mucous membrane. A simple alkaline nasal douche should be ordered, to be followed by the introduction into the nose of Ung. Hydrarg. Nit. dil.

When the corneal condition has sufficiently subsided the condition of the refraction should be examined, and, if there is any astigmatism, it should be corrected, as in these patients it is very difficult to remove the nasal infection entirely, and their astigmatism induces them to rub the lids with fingers which are usually already infected from the nose, and so to continually reinfect their eyes.

Under this treatment the condition rapidly improves. The gummy irritating discharge ceases, and the ulcers slowly heal, leaving faint, rounded, sharply

defined superficial scars which fade in the course of time.

Even in the deep infiltrated ulcers with hypopyon which are due to the bacillus duplex, caustics either in the form of strong antiseptics or the actual cautery are distinctly contra-indicated. By their means tissue is destroyed which could otherwise have been saved, and the resulting leucoma is always much denser than that obtained by the treatment above indicated. The objections to the cautery are that it does not cause that marked improvement which follows its use in pneumococcal ulcers; the hypopyon especially does not diminish after its use. There is usually no special part of the ulcer whose appearance indicates that it should be cauterised; to be thorough, therefore, the whole margin and base must be touched, thus causing a great increase in the size and density of the resulting leucoma. Equally good results are got by the zinc therapy as regards the certainty of an actual cure, while the resulting vision is usually better, and the deformity less. These ulcers are very slow in their progress when untreated, but usually, as soon as treatment is begun, they cease progressing, slowly clean, and begin to fill up. In the healing stage, after washing out the ulcer as directed, a little xeroform or airol can be dusted into its floor. As soon as the ulcer is quite clean the sulphate of zinc can be left off or diminished in strength until there is a sound epithelial covering over the defect, when, if the con-

conjunctival condition shows any signs of recurrence, as it usually will, the drops should be continued.

If the ulcer should perforate and an iris prolapse occur, the portion of iris should be excised and the wound freed. A conjunctival bridge graft should be planted into the wound, which will then rapidly consolidate.

A descemetocœle is not uncommon. The best means of treating such a complication is to perforate the bulging floor with the actual cautery and apply a pressure bandage to bring the lips of the wound together. In this manner a flat scar is obtained with the least danger of a persistent fistula resulting.

THE INFECTIOUS MARGIN ULCER OF ZUR NEDDEN

(Das Infektiöse Randgeschwür)

This special form of marginal ulceration of the cornea was described by Zur Nedden, who observed it frequently in the patients attending the Bonn Clinic. In England it appears to be uncommon: there have been no cases yet recorded, although I have had the opportunity of observing three cases during the summer of 1905 in Moorfield's Hospital. The ulcers are undoubtedly due to a special organism whose morphology and biology have been described by the discoverer. They are probably primary in the sense of not being directly due to a conjunctivitis; in

fact they often occur when the conjunctiva is unaffected, only showing an injection of the superficial vessels in the neighbourhood of the ulcer. It might have been more strictly correct to place this form of ulceration in the class with pneumococcal and other primary infections; but as it may still be considered an open question as to whether they are primary or not, and as the diplobacillary ulcer also occurs in conditions which appear not to be due to a conjunctivitis, these two forms may be taken together, as they resemble each other in many respects.

When a conjunctivitis is present, it is of the usual mucopurulent form, with velvety thickening of the conjunctiva in the fornix, which fades away over the tarsus. Phlyctenules are not uncommon, and appear to be more an integral part of the special infective process than does the conjunctivitis. The condition is usually one-sided. The whole corneal surface is dull, about one millimeter from the limbus there is usually a crescentic breach of surface parallel to the margin, about one to two millimeters broad and varying in length. Near this ulcer, especially at its ends, there may be small round ulcerations of about a pin's head size, and throughout the rest of the peripheral portion of the cornea are minute points where the epithelium is raised up sharply. The substance of the cornea is clear in the pupillary area, but throughout the remainder a grey infiltrate can be seen arranged in clusters of varying size, the smallest being immedi-

ately under the small surface elevations, and the larger forming the floors of the ulcers. These ulcers are peculiar in appearance and differ very much from others which may occur at the corneal margin; their edges are sharp, slightly irregular, and at places rather prominent,—they are not undermined nor deeply infiltrated; their bases are dull grey, on the level of the general corneal surface, sodden, and appear soft and velvety; if large, the centre may be slightly deeper and clearer than the rest of the base. The infiltration

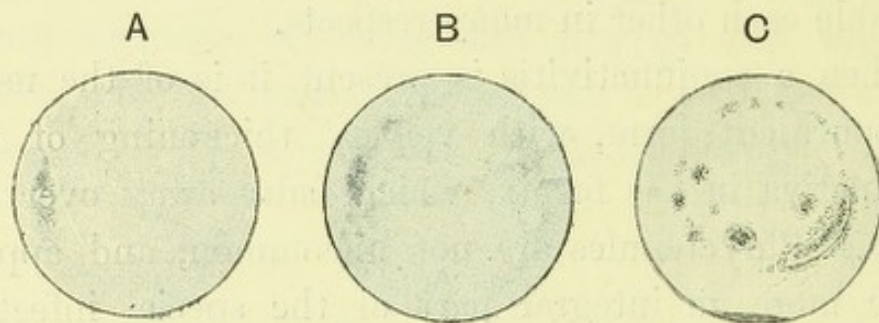


Fig. 16.

ZUR NEDDEN'S ULCER OF THE CORNEA.

A B From Zur Nedden's cases.

C Author's case.

is not confined to the broken area, but extends diffusely into the cornea, especially at the ends of the crescent, where it may be continuous with that of an adjoining ulcer. There is no deep infiltration. The anterior chamber is usually slightly clouded, and occasionally a hypopyon is present. A varying degree of iritis accompanies the attack.

The patients complain of some pain not usually very severe, as they may not consult a surgeon for several days, together with difficulty in seeing. They are

generally adults, and show no signs of scrofula, or keratitis eczematosa (*q.v.*).

The *diagnosis* usually is distinctly suggested by the appearance, the features of importance being the marginal position of the ulcers, their shape, the other infiltrates, the presence of a one-sided conjunctivitis, and the absence of any of the signs of the strumous condition associated with keratitis eczematosa. A bacteriological examination of material from the base of the ulcer confirms the diagnosis; a stained slide (Gram's method) is often sufficient; the organism may be mistaken for the bacillus duplex, from which it is distinguished by its growth on agar, and without liquefaction of blood-serum; the differential diagnosis from Friedländer's pneumobacillus is best determined by inoculation, the Zur Nedden organism not causing septicæmia in the mouse.

The organism is a straight or slightly curved bacillus of about $\cdot 6$ m. in thickness, and $\cdot 9$ to $1\cdot 2$ μ . in length; single individuals are smaller, and others may grow, especially in cultures, into long threads. It is Gram negative, decolorising very readily, and taking up the aniline dyes faintly. Individuals often show lighter parts at their ends, and vacuoles sometimes occur in their substance. There is no spore formation. Capsules are absent; pairs may occasionally be seen end on to each other like the bacillus duplex. It is an obligate aerobe. Heat at 36° for twenty-four hours kills the dry organism.

Growth in the form of a thick slime occurs on agar, gelatine, or blood-serum. The bacillus can also be cultivated in milk or bouillon.

Injected into animals it does not cause septicæmia.

The *treatment* is quite satisfactory. The conjunctiva should be lightly pencilled with two per cent. silver nitrate, and the ulcers syringed out with sulphate of zinc grs. ii. ad \bar{z} i. Zinc drops of the same strength should be used every two or three hours. The pupil is to be kept dilated by atropine. Under this treatment the ulcer will clear in a few days and the infiltrates in the cornea disappear. The conjunctivitis may remain for several days and require repeated pencilling. The cautery is distinctly contra-indicated, Zur Nedden's results show that sound healing is to be obtained without its use, a much fainter nebula remaining. Recurrences are common, and should be treated in the same way as an original attack. Zur Nedden recommends $\frac{1}{1500}$ oxycyanate of mercury lotion as a good antiseptic in this condition.

CHAPTER VII

ULCERATION OF THE CORNEA OCCURRING IN ACUTE CONJUNCTIVITIS

IN a previous chapter the corneal complications occurring in diplobacillary conjunctivitis have been discussed, also the infection by Zur Nedden's bacillus which may be associated with a conjunctivitis. In any acute conjunctivitis, whatever be the specific causal organism, secondary ulceration of the cornea may occur. In diphtheria or gonorrhœa of the conjunctiva, in pneumococcal, Koch-Weeks, streptococcal, or staphylococcal conjunctivitis,¹ this secondary infection not uncommonly is the most serious feature of the case, and dominates prognosis and treatment to such an extent that it deserves considerable attention.

Although there are many points of resemblance in all these infections, still they may be for convenience divided into three groups:—

GROUP I.—Koch-Weeks, pneumococcal, streptococcal, and staphylococcal infections.

GROUP II.—Diphtheria.

GROUP III.—Gonorrhœa.

¹ Influenzal corneal infection has been observed by Zur Nedden.

This division is useful from the point of view of treatment. In the first group the different infections vary considerably in their clinical history, especially with regard to onset, duration, and result; it is therefore important that the actual organism should be determined. Serum treatment does not give sufficiently reliable results to justify its adoption, and we have to rely on antiseptic irrigation, which is the same whatever be the organism. In Group II. the best results follow serum treatment, so that this condition forms a special class. Gonorrhœa reacts in a definite specific manner to the silver salts, and so must also be separately considered.

GROUP I.—ULCERATION OF THE CORNEA OCCURRING IN
CONJUNCTIVITIS DUE TO THE KOCH-WEEKS BACILLUS,
THE PNEUMOCOCCUS, STREPTOCOCCUS, OR STAPHY-
LOCOCCUS.

It is quite outside the scope of this work to discuss the clinical appearances of these various forms of conjunctivitis; however, as the bacteriological classification of conjunctivitis is not yet generally adopted, a few words of explanation may be useful.

The great majority of conjunctivitis cases result from an infection by some definite organism. The inflammation resulting is named after the organism, thus we have Koch-Weeks, pneumococcal conjunctivitis, etc. Different persons react very differently to the same organism. Professor Axenfeld has introduced into his

own conjunctiva pneumococci from an acute conjunctivitis without producing any result, and patients are very often observed to have a mucocoele infected by pneumococci without suffering from a definite conjunctivitis. The incidence of a conjunctivitis depends on whether or no the person is susceptible to the particular organism which has been introduced into the conjunctival sac; the severity of the conjunctivitis depends on the degree of the susceptibility to the particular organism. Considering the well-known variation both in the virulence of the pneumococcus and the susceptibility of different persons to that organism, it is only to be expected that pneumococcal conjunctivitis will present all grades clinically, from a simple, very mild catarrh to the most acute membranous form with sloughing of the conjunctiva. In the same way the other forms of conjunctivitis vary in intensity, so that the terms simple catarrhal, mucopurulent, purulent, or pseudomembraneous convey very little idea of the infective agent at work. To those, therefore, who desire to simply label a conjunctivitis with some name, that it may be treated by some rule of thumb, the bacteriological classification presents few attractions.

The symptomatic grouping is very useful in determining the likelihood of corneal complications arising in any given case. A series of 90 cases of pneumococcal conjunctivitis is published by Schmidt: in 20 which were simple chronic or subacute catarrhal, and 7 which were mucopurulent or mild purulent, there were no

corneal complications; in 56 severe purulent 4, or seven per cent.; and in 7 pseudomembranous 2, or twenty-eight per cent., had corneal complications. The same is true of Koch-Weeks, staphylococcal, and streptococcal conjunctivitis. The Koch-Weeks bacillus is more easily killed out, and few persons are very susceptible to staphylococcus, especially the common staphylococcus albus or citreus; as a matter of experience, therefore, such infections rarely last long enough, or are sufficiently acute, to affect the cornea. Streptococcal conjunctivitis is uncommon, but a large proportion of the cases are very severe, many being membranous; we have corneal complications, therefore, in quite a large number of such cases.

An ulcer may form in any cornea from an abrasion which becomes infected from the conjunctival secretion. The great majority of the infiltrates and ulcers which we are now considering, however, are due to some lowering of the vitality of the cornea from interference with its blood supply, diffusion of toxins into it, or maceration by discharges accumulating on its surface. The destructive changes usually occur near the corneal margin, often under cover of the conjunctiva, when it is swollen and protrudes over the cornea, allowing the secretions to collect and remain in contact with the epithelium. In the case of infected abrasions, of course, the site is central or in the lower part of the cornea.

A change is first noticed in the epithelium, whose surface becomes dull, then a grey infiltrate is seen in

the superficial layers of the cornea, at first faint, but rapidly increasing in density, so that it becomes yellowish,—the epithelium is cast off and a shallow infiltrated ulcer is formed. In severe cases the ulcer may penetrate, perforate, and cause panophthalmitis; several ulcers may form around the margin, and so cut off the nutrition that the cornea may slough; or in rare cases a form of interstitial keratitis may result, the whole cornea becoming densely white like porcelain without actual destruction occurring; later on vessels pass into its substance and some clearing occurs, although never sufficiently for useful vision to be restored.

The cornea must be carefully watched in all cases of purulent or pseudomembranous conjunctivitis, irrespective of the particular infective agent which is present. The only satisfactory method of either diagnosing or excluding an ulcer is to carefully examine the cornea in a good light, preferably by focal illumination, and in any case of doubt, especially in children, to do so under a general anæsthetic; for whatever be the prognosis of the conjunctivitis, any implication of the cornea renders the case much more serious, and necessitates greatly increased care from the surgeon. The commencement of an infiltrate is usually associated with increase of pain, photophobia, and a general feeling of discomfort. The inflammation in the conjunctiva usually spreads to the iris, so that we have the symptoms of iritis added; this is most often the case in streptococcal infections.

Treatment.—Anything in the nature of an occlusive bandage must be absolutely prohibited, and the freest exit allowed to the pus and discharge. The attendant should separate the lids every quarter of an hour when there is pyorrhœa and wipe away the secretion, and three or four times a day the surgeon should thoroughly irrigate the conjunctival sac, passing the douche nozzle well into the fornix, and remove all discharge. The whole surface can then be painted with five or ten per cent. protargol, and sufficient atropine instilled to keep the pupil widely dilated. It is important that the douche should be very carefully used,—a head of two feet of water is the maximum allowable; the fluid may be any mild non-irritating antiseptic, potassium permanganate diluted to a light claret colour is very useful; it should be at a temperature of about 95° F. The nozzle of the douche should be flattened and curved to pass under the lid, and the greatest care should be taken not to abrade the cornea.

If the conjunctiva be very œdematous, it should be incised, especially if the limbus be so swollen as to project over the cornea. In those cases where the swelling is so great that the lids cannot be opened, and it is impossible to wash out the fornix, the pressure on the cornea is very injurious and must be removed by splitting the external canthus. One blade of a strong scissors is placed in the conjunctival sac and the other on the skin, with one cut the canthus is divided and the pressure relieved. No treatment directed at the

ulcer itself is of much avail : when large it may be dried out after the irrigation and dusted with powdered xeroform, airol, or chinosol; if perforation be impending, paracentesis through the floor often checks the progress and leaves much less destruction of tissue than when spontaneous rupture is allowed to occur.

The treatment of ulcers in this group is not very satisfactory. The field appears to be one where serum treatment might be used with advantage, and in isolated cases of pneumococcal and streptococcal infections brilliant results have been recorded. It is doubtful, however, how much the favourable course of these cases is due to the serum used, as pneumococcal conjunctivitis very often ends by a well-marked crisis, which may have coincided with the serum injection.

The variation in streptococci and the monovalency of the sera on the market leaves it an open question whether the serum available actually is toxic to the particular 'brand' of organism present. No serum has yet been prepared for Koch-Weeks infections, and as staphylococci rarely cause ulceration, it is obvious, therefore, that serum treatment can only be relied upon in very exceptional circumstances.

GROUP II.—KERATITIS SECONDARY TO INFECTION OF
THE CONJUNCTIVA BY KLEBS—LOEFFLER (DIPH-
THERIA) BACILLUS.

Superficial infiltrations occasionally occur in the cornea, secondary to a true diphtheria of the conjunctiva.

Coppez has studied the action of diphtheria toxine on the cornea, and has established that the toxine can of itself produce an infiltration of the superficial lamellae. When dropped into the conjunctival sac the corneal epithelium becomes loosened, much in the same manner as by the action of cocaine, a slight superficial infiltration of the parenchyma then occurs, and after a few days again disappears, even though the local application of the toxine be continued. By a single continuous contact of the toxine with the intact cornea for one and a half hours, an inflammatory reaction commenced after an 'incubation' of from twenty-four to forty-eight hours; the cornea slowly became dull and then cleared again. If the epithelium be rubbed or abraded, the interval was shorter and the reaction more pronounced and of longer duration. A purulent infiltration, however, rarely occurred unless there was a secondary infection with some other organism.

Coppez established, by pathological examination, that these changes were not due to an interference with the nutrient vessels in the conjunctiva or sclera, but to the direct action of the toxine on the corneal tissues. As his experiments were entirely conducted on the lower animals, it is quite possible that they cannot be unreservedly applied to man; the conditions present in a diphtheria of the conjunctiva are somewhat different from those of the artificial toxine poisoning which he observed.

The number of cases of pure diphtheritic conjunctivitis

associated with corneal lesions is so small, that we may consider all ulcerative cases to be mixed infections. In man certainly diphtheritic conjunctivitis interferes with the nutrition of the cornea, on account of the coagulation necrosis which occurs around it; there is also the direct action of the toxine on the epithelium and the presence of a mixed infection, very often streptococcal; it is not remarkable, therefore, that we sometimes have the cornea gravely affected, even to the extent of total necrosis.

The best method of preventing corneal complications in diphtheritic conjunctivitis is by subcutaneous injection of the antitoxine, in exactly the same manner as for a laryngeal diphtheria. The conjunctival condition rapidly improves and the cornea becomes more or less immune to the bacillus or its toxine. Should, however, corneal complications already be present, the antitoxic treatment will not cause their rapid disappearance; by improving the nutrition of the part it greatly aids the treatment, but it is not directly curative. The treatment in such cases must follow the lines indicated for streptococcal infection (see p. 120).

GROUP III.—GONORRHOËAL CONJUNCTIVITIS

From the point of view from which we are regarding these acute inflammations, the most dangerous must be considered to be that due to the gonococcus. Conjunctival gonorrhœa occurs at three different periods—

at birth ; during childhood ; and in adult life. This subdivision of the disease into three groups is of some practical importance, as corneal complications are graver, both from their proportion and severity, in adults than in the newly born, while children occupy an intermediate position in both respects. The corneal complications usually develop late in the infiltration stage, or early in the pyorrhœa stage of the conjunctivitis, the actual time of onset being determined by the acuteness of the infection. The conditions of the conjunctiva which specially tend to corneal affections are excessive chemosis or a diphtheritic infiltration of the membrane : the former by raising up an overhanging edge around the cornea under which secretion can collect and macerate the epithelium, and the latter by interfering with the corneal nutrition.

The signs of corneal infection may appear about the fourth day of the disease—that is, in the newly born on or after the seventh day of life,—or any time up to about the fourteenth day. There is a superficial infiltration which is either marginal and related to the overhanging chemotic limbus, or central, when there is often a defect of the epithelium.

The infiltrate is grey and around it is a zone of inflammatory œdema. In a short time, often as little as twenty-four hours, the infiltrate becomes yellow, and actual ulceration occurs.

The further progress varies very much according to the intensity of the infection and the efficiency of treat-

ment. When centrally placed these ulcers usually strike deeply, while their spread laterally is restricted, so that perforation is frequent. The marginally situated lesions, however, tend to spread round the cornea and form a deep furrow, which may perforate, but as this type of ulceration is much rarer than the central its perforation is very infrequent.

In unhealthy children, and in adults with a very virulent membranous infection, the whole cornea may become opaque, dense white, and then simply flake away like wet blotting-paper till it perforates, and healing occurs with a large flat or bulging scar, consisting of the iris in a fibrous mass of new tissues.

It is often very difficult to get a good view of the ulcer, and in children it is advisable to make the first examination under an anæsthetic if there be much difficulty in opening the lids, for the ulcer may be on the point of perforation, and the effort to open the lids may cause rupture, and even protrusion of the lens, so that the eye is irretrievably damaged. There are no prominent characteristics of the ulcer itself: it is deeper in the middle than the edges, which shelve down and are not undermined, the floor is covered with a yellowish purulent débris, and the surrounding cornea is dull and cloudy; there is usually considerable hypopyon.

The toxine of the gonococcus seems to have the power of attacking the cornea, especially when assisted by the macerating influence of the secretions,—the marginal ulcers are due to this action. More commonly

the infection of the cornea occurs through an abrasion, and such is the case in the central ulcers.

The *diagnosis* is readily made in adults from the history and the appearance of the conjunctivitis. It is quite a mistake to consider all cases of ophthalmia neonatorum as gonorrhœal; many are due to pneumococcus, bacterium coli commune, streptococcus, and other organisms, but the true conjunctival gonorrhœa is most dangerous to the cornea. In all cases, therefore, the diagnosis must be confirmed by the microscope.

The *treatment* consists in (a) the removal of the secretions without damaging the cornea; and (b) the direct attack on the diseased conjunctiva by means of antiseptics: to which of course is added the general treatment of a corneal ulcer by atropine, hot douching, and an antiseptic ointment. In the early stages the chemosis of the conjunctiva may prevent the lids being opened and cause pressure on the cornea; in such case the outer lid canthus should be split by introducing into the conjunctival sac one blade of a scissors and cutting freely through the angle,—not only does this relieve the tension directly, but by depleting the conjunctiva it removes the chemosis which causes it. The greatest care should be taken to remove all the secretion as quickly as possible,—the best lotion is a $\frac{1}{5000}$ potassium permanganate solution lukewarm, and delivered from a douche can with a head of about one and a half to two feet. At least three times a day the surgeon should thoroughly wash out the conjunctiva,

using the greatest care not to injure the cornea, but still to cleanse all the crevices and folds thoroughly ; in the interval the nurse should gently separate the lids every ten to fifteen minutes and wash away the secretion with the solution.

In the stage of pyorrhœa, which is usually fully developed when the ulcers occur, the long-established silver nitrate pencilling gives the best results : after thorough cleansing away all secretion the everted conjunctiva is dried and pencilled over with a two per cent. argenti nitras solution, special attention being paid to the folds of the fornix, and then washed with a saline solution to neutralise the excess. The pencilling is best done by means of a small wooden rod round the end of which is wrapped a wisp of cotton wool,—after being used these can be burnt, they form a very inexpensive and convenient means of applying the silver salt. Immediately after the pencilling a thin bluish white membrane forms on the surface, and there is some increase in the inflammatory symptoms, but after a few hours this membrane disappears and the discharges again are profuse and purulent. The pencilling should at first be repeated twice daily, the strength of the silver nitrate not exceeding two per cent. The second application of silver nitrate should never be made until the slough, or membrane, from the previous one has completely cleared away. In some cases, therefore, once a day will be quite frequent enough. In a few days the thickening of the conjunctiva will subside and

the discharge lessen, the cornea begin to clear and the ulcer clean; then the silver applications should be made less frequently, and when the ulcer has cleaned may be replaced by protargol or argyrol.

The best treatment for the ulcer is always an energetic attack on the conjunctiva. In spite of every effort the ulcer may progress and perforation be inevitable,—it is better to open the anterior chamber through the floor than to allow Descemet's membrane to rupture spontaneously: the wound should be opened daily to allow the aqueous to escape and relieve all tension on the cornea,—the ulcer will sometimes begin to clean at once and the resulting anterior synechia will be less extensive than would otherwise be the case were spontaneous rupture to occur.

CHAPTER VIII

ECZEMATOUS ULCER

THE chief point of difference between Keratitis Parenchymatosa (Interstitial Keratitis) and Keratitis Eczematosa is that the former affects the deeper layers of the corneal parenchyma, and the latter the epithelium and superficial layers. In the former disease ulceration is extremely rare; but in the latter it is quite common. We find names in plenty to designate this disease—Strumous, Perforating, or Scrofulous ulcer, Fascicular Keratitis, Snail's-track ulcer, Phlyctenular ulcer, and others, with the inevitable confusion which results. The most widely used term for the disease is Keratitis Eczematosa, and it is preferable to use this term also for the ulcers which occur in the course of the disease. The associated conjunctivitis is called conjunctivitis eczematosa, and thus in the names themselves an indication is given that we have to deal with a single affection with many varied manifestations. Professor Fuchs has adopted this classification, and no author is worthier of being followed in anything pertaining to corneal diseases.

Etiology.—The disease especially affects children, and is common up to the age of puberty, after which time it decreases very much in frequency, and is rarely

seen except as a recurrence in a cornea previously affected. The children are of the strumous or scrofulous types, which Erichsen has so graphically described: the thick-skinned, heavy-featured type, with coarse hair, pasty complexion, and dull intellect suffer more than the pale, clear-skinned, fair-haired and generally precocious, 'delicate' children of the second variety. They are usually badly housed and nourished, the dwellers in slums. Enlarged tonsils and adenoids, an arched palate, chronic rhinitis, enlarged cervical glands are very common amongst them. They are mouth-breathers; they suffer from eczema of the scalp, ears, and face. In a large proportion of cases tubercular lesions are found in some part of the body, and it has been suggested that the eye condition is in some way due to the toxins of tuberculosis circulating in the bloodstream. There is a close relationship between tuberculosis and keratitis eczematosa, but our knowledge is not yet sufficient to allow us to define this as cause and effect.

The exciting cause of an attack of keratitis and the formation of an ulcer is often an acute fever, especially measles or scarlet fever, less often whooping-cough and influenza.

Corneal infiltrates, which break down and form ulcers, resembling eczematous ulcers very closely, occur in cases of subacute or chronic conjunctivitis affecting strumous individuals: some of these are infections of the cornea with the special organism of the conjunctivitis; others, however, are cases of keratitis eczematosa.

An attack begins with photophobia, some lacrymation, a sharp pricking pain in the eyes, and conjunctival injection. Some blepharospasm is usually set up leading to a slight œdema of the lids, and later to cracks or fissures at the outer canthus. The patients avoid the light, mope in dark rooms often with their eyes bandaged, and apply poultices of cold water or tea, all of which are more injurious than beneficial, especially the bandage, which retains the irritating secretions and favours the ulcerative process.

The examination of such cases is often very difficult, but it must always be thorough; if necessary, under an anæsthetic. When, after repeated instillations of cocaine, the child cannot be persuaded to open the eyes in a darkened room, it is best to have the patient on a table with the head held firmly by an assistant, and then, with great care, to separate the lids with some form of elevator; usually a good view can be obtained if the globe be rotated downwards by means of a fixation forceps. Such an examination allows us not only to make an exact determination of the condition present, but to apply treatment in an efficient manner to the conjunctiva, the cornea, and the lids as required.

The lids are seen to be slightly œdematous, and forcibly closed, so as to offer great resistance to any examination. The conjunctiva bulbi is deeply injected, but not œdematous. The individual vessels are very distinct, and pass quite up to the cornea; hæmorrhages are very rare. Round the cornea there is often

thickening of the subepithelial tissue in the form of small nodules or phlyctenules, opposite which the conjunctival hyperæmia is greatest. The nodules may break down and form ulcers, varying in size from the smallest points visible to the naked eye up to shallow circular depressions of one to two millimeters in diameter. In the fornix enlarged follicles are usually seen, and there is some velvety congested conjunctiva over the

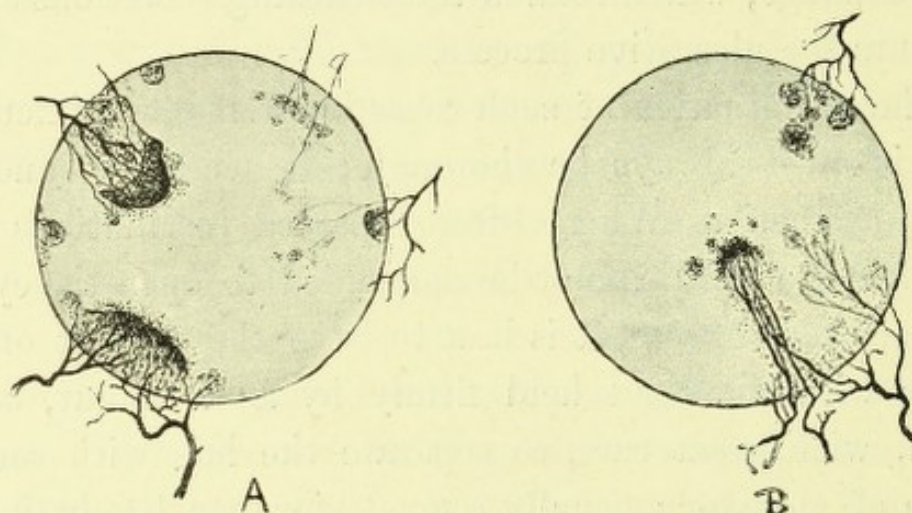


Fig. 17.

ECZEMATOUS ULCERATION.

A Marginal crescentic ulcers with some old nebulæ.

B Snail's track ulcer with old and some fresh infiltrations.

tarsal plate, especially in the upper lid. There is a watery discharge, containing a few flakes of mucus, which causes some excoriation around the lid margins.

The cornea always shows changes on its surface and in its substance ; and an exact diagnosis depends upon the nature and distribution of these changes. At some parts the cornea shows a normal bright reflex, but irregular areas have a dull, granular, or even broken

surface; these are arranged in clusters, sometimes around the margin, sometimes towards the centre, and separated from each other by more or less normally bright strips. Commonly two or three complete breaches may be seen, crescentic in shape, convex towards the centre, and the sector-shaped areas between these ulcers and the margin of the cornea are irregular, ridged, but still reflecting. Round or oval patches may be seen slightly raised up with a dull surface of increasing irregularity towards their centres, where the epithelium may be completely gone. Later on a punched-out ulcer takes the place where formerly the dull surface was visible.

When the substance of the cornea is examined infiltrations will be found in it corresponding to these surface changes. Even with the naked eye the prominent feature of the disease will be obvious—the clustered arrangement of the infiltrations (*herdförmige*) in contradistinction to the diffuse uniform infiltration with ill-defined margin seen in keratitis parenchymatosa. This is still more evident on examination with the loupe, when the opacity is seen to be made up of minute specks forming clusters separated by comparatively clear spaces. The infiltrate is generally light grey and superficial, that is, under the epithelium and in the superficial layers of the parenchyma; but it may be dense yellowish and comparatively deep, when perforation of the ulcer which results is usual (Fuchs). Vessels are usually seen in the cornea; they may be old or recent; they are found in the areas whose surface is disturbed, and may form

dense leashes (*Gefässbändchen*) passing out to a crescentic ulcer, and causing a ribbed appearance of the surface (Fig. 17, B), or may branch and spread to the clusters of infiltration, to form the well-known pannus eczematosa (Fig. 17, A). The vessels are superficial; they spring from conjunctival vessels, which can be seen to pass over the limbus into the cornea; they branch dichotomously, and are usually quite different from the brushlike deep vessels found in parenchymatous keratitis. When the infiltrates are deep a few deep vessels may form. Such, however, are extremely rare.

The ulcers which occur conform to several types:—

(a) Small shallow clear ulcers with a very slight infiltration around, formed by the breaking down of quite superficial infiltrates; these rapidly heal, and if Bowman's membrane is intact leave no opacity behind.

(b) The crescentic grey superficial ulcer which begins at the margin of the cornea, and creeps towards the centre, drawing after it a band of superficial vessels. This ulcer has a sharp infiltrated convex edge towards the centre, and an irregular concave edge towards the limbus of the cornea. It has no tendency to perforate, and heals, leaving a permanent 'snail's-track' (*Gefässbändchen*) across the cornea. It is distinguished from a simple vascularised ulcer by its creeping tendency and the denseness of the vascular band; it destroys Bowman's membrane, and leaves therefore a permanent trail.

(c) The round perforating ulcer situated either marginally or centrally. This probably begins in the

deep-seated yellowish infiltrate (Fuchs). It has no band of vessels associated with it, but has a distinct tendency to perforate, making a round clear-cut hole in the cornea, which often heals with prolapse of the iris. The ulcer has a yellowish grey opaque floor, and is surrounded by a region of diffused cloudiness. They are most commonly situated at the margin of the cornea, and are multiple. A similar ulcer may occur in the sclera just at the limbus, and perforate into the anterior chamber; the iris prolapse, after healing, then appears as a round black or brown subconjunctival nodule in the sclera.

(*d*) Multiple, small, marginal ulcers deeper than (*a*), beginning as infiltrates which break down, become vascular, clean, and heal up, leaving only small facets or irregularities on the surface of the cornea.

The deeper parts of the eye are usually unaffected; the iris may be slightly hyperæmic, the pupil is small; only in the deep ulcers is there a hypopyon.

The diagnosis is not difficult in the great majority of cases; the most important differentiation is between Eczematous and Parenchymatous Keratitis; the points can thus be indicated in tabular form:—

KERATITIS ECZEMATOSA.	KERATITIS PARENCHYMATOSA.
Strumous type. Glands in neck. Tubercular lesions.	Hereditary specific type. Saddle nose, flat malar bone. Hutchinson teeth.
Lacrymation, photophobia, blepharospasm.	Ditto.
Phlyctenules.	No phlyctenules.
Surface reflex uneven, and broken.	Surface reflex dull, but even and unbroken.

KERATITIS ECZEMATOSA.	KERATITIS PARENCHYMATOSA.
<i>Infiltrate clustered, superficial.</i>	Infiltrate diffuse, deep.
<i>Vessels superficial, derived from conjunctiva, open branching.</i>	Vessels deep, not passing over from conjunctiva, parallel, 'broomlike.'
No precipitate on back of cornea.	Precipitate on back of cornea.
No affection of iris.	Usually (? always) iris affected.
Rarely hypopyon.	Often hypopyon.
<i>Ulceration usually.</i>	Ulceration rare.

It must be noted that parenchymatous keratitis can occur from other causes than hereditary syphilis, in which, of course, some of the above signs are wanting. The important signs in the table are in italics.

In both conditions the conjunctiva is sterile or contains only indifferent organisms on its surface.

The diagnosis from pannus trachomatosa with superficial ulceration is settled by the condition of the lids and the distribution of the pannus.

A condition which much resembles eczematous ulceration occurs in diplobacillary infection of the cornea. Indeed, it might be quite reasonably urged that such cases were infected eczematous conditions. On account of treatment it is well to separate them, and this is done by the bacteriological examination.

Pathology.—The first change to occur is a deposit of lymphoid cells under Bowman's membrane in the form of small nodules around the ends of the sensory nerves. These are grouped in clusters, and can be seen as grey spots over which the corneal epithelium is slightly oedematous and appears dull. Vessels constituting the

pannus eczematosa make their way to these infiltrates. They are derived from the superficial conjunctival vessels. Bowman's membrane is eroded and destroyed as these nodules enlarge. The cells also invade the superficial layers of the parenchyma; the epithelium covering the eczematous patch is lost, and an ulcer forms. The deposition of lymphoid cells and destruction of the intervening lamellae may continue at the base of the ulcer till Descemet's membrane is exposed; or even, by destruction of this, till a perforation of the anterior chamber results with prolapse of the iris. In the 'snail's-track' ulcer or fascicular keratitis, the infiltration does not pass below the most superficial layers of the parenchyma. Bowman's membrane is destroyed along the line, and the epithelium grows over a layer of vascular connective tissue which is densest at the head or centripetal end.

Bacteriology.—Keratitis eczematosa appears to be the same condition as that called conjunctivitis eczematosa, or phlyctenulosa, or scrofulosa, the lesion affecting the denser avascular connective tissue of the cornea instead of the loose vascular conjunctiva. For obvious reasons no attempts have been made to scrape out infiltrates of this nature from the cornea, but phlyctenules have been excised and examined for organisms without any definite results. In the later stages various organisms can be found in the conjunctiva and cornea, and so staphylococcus albus, aureus, or citreus, the bacillus duplex, streptococcus,

Koch-Weeks bacillus, and others have been described as present. The introduction of these organisms into the conjunctiva or cornea is, however, never followed by a typical kerato-conjunctivitis eczematosa. The infection must be endogenous, and it is most probably some toxic substance circulating in the blood-stream which is carried to the cornea by lymph channels; which, possibly aided by the presence of micro-organisms in the conjunctival sac, excites the inflammatory reaction which is the immediate cause of the infiltrates. Axenfeld has thoroughly disposed of the contention that phlyctenules are true tubercular lesions, and Müller, in a series of transplantation experiments in which fresh phlyctenules were introduced into the anterior chamber of rabbits, failed in every case to produce a typical tubercular lesion. The experiments of Leber with dead tubercle cultures, and the observations of Wright with tuberculine (R), indicate that the endogenous infective agent may be a tubercular toxine circulating in the blood.

Treatment.—We have to deal with a general condition which, if it be not tubercular, is closely associated with a lowered resistance against the bacillus tuberculosis. Every case should undergo the open-air treatment in some form. In severe and obstinate cases it should be rigorously insisted upon, even to the extent of sending the patient to a sanatorium. The condition of the mouth and nasopharynx should be examined: carious teeth, hypertrophied tonsils, and adenoids

should be dealt with at once. Many of the children are mouth-breathers, and when their nasopharyngeal condition has been remedied this habit must be broken. Eczema of the scalp, ears, nose, and face must not be neglected, but vigorously treated. Whatever the original source of corneal infection may be, we have to deal with a septic conjunctiva, and it is not only necessary to clean it, but to prevent the repeated fouling which occurs from the nasal discharge on the fingers and handkerchief. In all cases where there is a discharge from the nose or any excoriation at the nostrils, an alkaline nasal douche with some antiseptic added should be used several times daily, the anterior nares dried, and then a little Ung. Hydrarg. Nit. dil. introduced into each nostril.

Attention to these matters will rapidly cause an improvement in the general health—the cervical glands will diminish, and the corneal ulcers clean and commence healing.

Treatment at a watering-place with sea-bathing is highly beneficial.

From our ignorance of the cause of the disease we can only treat the symptoms. The local treatment, therefore, varies according to the condition of the cornea. Photophobia and blepharospasm are very troublesome in the early stages, and are best relieved by atropine, hot fomentations, leeches to the temple, dark glasses, and occasional cold douches to the face. On no account should the child be allowed to wear a

bandage over its eyes, or be kept in a dark room. A deep shade combined with smoked glasses may be used, as they encourage the child to open its eyes; but a bandage keeps them closed, retains the tears, and promotes eczema of the lids and blepharitis. Each day an attempt should be made to induce the patient to relax the spasm by thoroughly cocainising the conjunctiva and cornea; in a darkened room the patient may then be coaxed into opening his eyes for a few moments.

The intense photophobia, blepharospasm, and lacrymation result from the irritation to the nerve endings by fresh deposits of lymphoid cells, and during this stage it is best merely to keep the pupils well dilated with atropine, and bathe the eyes at short intervals with a lotion as hot as can be borne. The blepharospasm is often kept up by the irritation of small cracks or fissures at the outer angle of the lids, which bleed when the eyes are opened. Such a condition improves rapidly, if the fissures be touched with the mitigated silver nitrate stick. The profuse lacrymation is quite sufficient to neutralise any excess and prevent it affecting the cornea. As soon as the eyes are freely opened irritants must be used, commencing with the milder ones, and, as the fresh infiltrates disappear, increasing their strength. The mildest irritant in use is the white precipitate ointment. Finely powdered calomel dusted on to the cornea is more stimulating, and the Ung. Hydrarg. dil. and Ung. Hydrarg. Ox.

Flav. fort. are still more stimulating, and very useful as the condition improves. The occurrence of fresh infiltrations is the only contra-indication to the application of these irritants.

A small glass rod is dipped into the ointment, the lids are separated, and the cornea is smeared by drawing the rod lightly across it; or the upper lid is drawn away from the globe, the rod inserted under it, the eye allowed to close, and the rod withdrawn, leaving the ointment in the conjunctival sac. A small square of lint is then laid on the closed lids and the cornea massaged through the lids for several minutes, so that the ointment is well rubbed in. This should be done at least once a day, and, as tolerance is developed, two or three times. It is a useful practice to combine atropine with the ointment used in the strength of grs. ii. ad ζ i.

In some cases the Ung. Hydrarg. Flav. is not well borne; the patient complains of great pain after its use. This may be due to the preparation used. To obtain the best results the drug should be specially prepared from the freshly precipitated oxide, made by the interaction of solutions of caustic soda and corrosive sublimate. The oxide should not be dried but directly incorporated in a basis of paraffinum molle BP. The order Ung. Hg. Flav. (Martindale) 1.25 %, 2.5 %, 4 %, 5 %, 10 %, will always give a satisfactory preparation.

When we have to deal with a single ulcer which is penetrating deeply and is situated near the margin of

the cornea, the whole process may sometimes be cut short by lightly cauterising the ulcer with carbolic acid or the electric cautery, and then, as soon as the slough separates and the wound is clean, filling it in with a conjunctival flap. A perforation may be often avoided by this means.

When the ulcer has penetrated down to Descemet's membrane, and we have a descemetocœle at the bottom of a small pit, the bulging membrane should be pierced with the cautery if it be in the pupillary area, and when the aqueous has escaped a firm bandage should be applied.

Should the ulcer be situated so far from the centre that it is obvious an iris prolapse would follow perforation, it is better practice to treat the condition by means of a conjunctival plastic operation ; for with the pupil under atropine the conditions are unfavourable for dealing with the prolapsed iris, and an iridectomy would probably be necessary to free the scar.

Mr. Tweedy's lotion of Sulphate of Quinine grs. iv. ad \bar{z} i., with a drop or two of Acid Sulph. dil. to allow of solution, or the Hydrochlorate of Quinine iv. grs. $1\frac{1}{2}$ —iv. ad \bar{z} i., is often found of great benefit in eczematous ulcers, and is probably the best lotion which can be ordered.

CHAPTER IX

NEUROPARALYTIC KERATITIS, OR TROPHIC ULCER OF THE CORNEA

CONSIDERABLE confusion has arisen with regard to the destructive lesions, which may occur in the cornea as the result of interference with its nerve supply. There is a tendency to designate as neuroparalytic keratitis any corneal condition which may occur subsequent to a lesion of the nervous system. An anæsthetic cornea and a keratitis seem to necessitate this diagnosis, while a facial paralysis and a keratitis seem also to imply the combination neuroparalytic in their description. On the other hand some would try and explain away the disease altogether, and account for the appearances which are present by dryness, exposure, or anæsthesia of the cornea. It is abundantly proved that there is a distinct form of keratitis, sometimes passing on to ulceration, which immediately follows a destructive lesion involving the trophic centre, or trophic nerve fibres, of the cornea; to this condition, entirely trophic in origin, the term neuroparalytic keratitis will be restricted, and the following remarks apply only to it.

A few days after some traumatic or inflammatory head lesion, fine grey infiltrated spots appear in the superficial layers of the cornea : these coalesce, become dense white or yellow, the epithelium is cast off over them, and an ulcer is formed. This is the Trophic Ulcer of the Cornea, or Neuroparalytic Keratitis.

Etiology.—Considerable confusion has arisen in the study of this affection, from the fact that the results obtained from experimental lesions in the lower animals have been applied to the human subject, without the requisite modifications necessitated by the difference in the anatomical relations present in the latter. Extirpation of the Gasserian ganglion in rabbits or dogs is followed by extensive ulceration of the cornea on the side of the lesion ; but in man, where the same operation has been performed for the relief of trigeminal neuralgia, this result only occurs in exceptional cases ; usually there is merely an anæsthesia with the resulting tendency for foreign bodies to remain in the conjunctival sac, together with a slight diminution in the lacrymal secretion,—the obvious explanation of the difference being the fact that in man the trophic nerves of the cornea do not pass through the ganglion, while in many animals they do. On considering the lesions which have caused a neuroparalytic ulcer, we can conclude that these fibres in man leave the Pons in the root of the fifth nerve, and, before reaching the ganglion, pass to the carotid plexus of the sympathetic, in which they traverse the carotid canal ; coming again into

relation with the first division of trigeminal in the cavernous sinus, they leave the plexus in the communications which it makes with lacrymal and nasal branches, and by them, especially through the subtrochlear nerve, are carried to the cornea. Occasionally in man they seem to take the direct course through the Gasserian ganglion to the first division of the nerve, as they do in some of the lower animals. The nerve centre from which these fibres arise appears to be in the Pons.

A lesion may destroy the centre or the nerve fibres at any part of their course, and it has been suggested that the lesions should be divided into nuclear and fascicular, and the latter again into intra-dural and extra-dural: such a refinement is of very little practical utility. From the intimate anatomical relations which these trophic nerves have to other cranial nerves, it is very unlikely that any lesion which interrupts the trophic nerves can avoid interference with the oculomotor, trigeminal, facial, or sympathetic nerves.

Any traumatism causing a fracture, or effusion so that the nerve is lacerated, contused, or compressed in any part of its course, will cause rapid ulceration; an inflammatory exudate compressing or involving the nucleus, or the nerve itself, will also cause ulceration, but not so rapidly; in the case of tubercular or gummatous deposits the keratitis may come on very slowly. In the course of acute fevers such as influenza nuclear lesions have occurred, as also in tabes; meningitis

and otitis media may cause fascicular lesions, while any malignant growth will have the same result if it infiltrates the nerve or its centre.

The *symptoms* which are observed, in a case of neuroparalytic keratitis, can be grouped into two classes: those *essentially* the result of the trophic nerve lesion, and those due to *concomitant* lesions of other nerve fibres supplying the eye or its appendages. It is obvious, from consideration of the track of the nerve fibres, that, while a nuclear lesion may cause a pure trophic paralysis, it is most unlikely that any fascicular lesion could affect the trophic nerve fibres, without, at the same time, seriously interfering with the sympathetic, sensory, or motor nerves, which lie in such intimate relation with them. The tendency, therefore, is for nuclear lesions to present a pure picture of trophic ulceration, while in fascicular lesions this is complicated by sensory and motor phenomena.

The changes commence in the cornea without any obvious cause, and unattended by the usual inflammatory symptoms which accompany a keratitis; of course in the traumatic cases there will be the history of some accident to the head, but in the inflammatory cases, especially the chronic, there is nothing to suggest the actual cause. The centre of the cornea usually becomes dull and cloudy. The light-reflex is obscured and the surface granular or even pitted, so that one has the impression that the epithelial cells are being destroyed and cast off. In the superficial layers of the

cornea small grey spots appear which coalesce, so that the centre of the cornea is pervaded with a greyish white dense opacity, gradually thinning out towards the periphery, where the infiltrate can be resolved with the loupe into flecks and lines. The central area becomes yellow and opaque, the epithelium is cast off over it, and a large ulcer results, which however does not usually quite reach the margin, a narrow band of cornea about 2 mm. broad remaining intact. There is not necessarily any diminution of sensibility of the corneal epithelium before the ulceration begins, nor any abnormal dryness, or exposure of its surface; the whole process can take place while the cornea is kept moist by a normal lacrymal secretion, and will continue in spite of careful protection by a bandage; in fact, the whole cornea has been destroyed in an eye with complete levator paralysis where the lid formed a thorough protection from any external source of injury.

The rapidity and extent of the ulceration varies with the lesion, so that we have acute cases, and cases which are slow, healing and relapsing, where the reparative power of the tissue seems sometimes to overtake the destructive, at other times to be unable to prevent a spread of the ulceration. In favourable cases where the lesion can be treated, or resolves naturally, only a part of the cornea is affected; it may be noted that there is no tendency for that part lying opposite the fissure or below it, that is the most exposed area of the cornea, to be affected, as is the case in keratitis e lagophthalamo.

The ulcer formed has no absolutely characteristic appearance. However large it may be, a narrow margin of unaffected cornea usually surrounds it; this may be considered as a peculiar and distinguishing feature, but in quite a number of cases the ulceration only affects a part of the cornea, and under these circumstances a diagnosis cannot be made without carefully considering the collateral evidence.

The sympathetic plexus is very often affected by the lesion which interrupts the trophic nerves; the evidence of sympathetic paralysis is a slight ptosis, some enophthalmos, a normal tension, and a contracted pupil which will not dilate with cocaine. Anæsthesia of the cornea is not essential in a case of neuroparalytic keratitis; indeed, there is a case recorded in which a patient had corneal anæsthesia in one eye, and a trophic ulcer in the other, where the sensibility was normal. However, as a matter of practical experience, in almost every case there is some sensory defect to be found in the area of distribution of the fifth nerve; as the caruncle and part of the cornea are both supplied by the infratrochlear nerve, an anæsthesia of the caruncle is the most probable sensory disturbance.

Motor paralyses may also occur, but are less frequent than sensory,—the muscles of mastication may be affected, or a ptosis or diplopia may result from interference with the oculomotor. It is conceivable that a facial paralysis may occur along with a trophic ulcer, but of course the lagophthalmos would then not be the

cause of the keratitis, but merely an accidental phenomenon occurring with it. In the same way the secretory nerves to the lacrymal gland may be involved, and thus the cornea and conjunctiva may be dryer than normal,—in all cases the absence of that lacrymation which usually accompanies a keratitis will be noticeable; this is due to interference with the reflex arc, whose afferent limb lies in the sensory nerves of the cornea, and efferent in the secretory nerves to the lacrymal gland.

When the lesion is syphilitic or tubercular, evidence of such a general infection will usually be found in other parts of the body.

Diagnosis.—It will readily be appreciated, from this account of the signs and symptoms, that the diagnosis of the affection may be very easy, or very difficult, according as the lesion affects one or more groups of nerve fibres. Where we have only a trophic lesion, and no other changes in the cornea, the diagnosis is quite easy. The occurrence of a keratitis, passing on to necrosis, after any of the head lesions already mentioned, can hardly be attributed to anything but its true cause. On the other hand, an anæsthesia may gradually occur in the cornea and conjunctiva, some facial paralysis, with inability to close the lids completely, may ensue, and then a necrotic destruction of the cornea; in such a case the diagnosis may be very difficult indeed, if not actually impossible, until the reaction to treatment decides the question.

It is not often that a true case of neuroparalytic keratitis is mistaken for any other affection; the acute cases which so rapidly lead to destruction of the cornea bear some resemblance to keratomalacia, but the general conditions of the patient are so different that this error should not be made. The purulent ulcers are at once differentiated by the prominence of their inflammatory symptoms. It is quite a common mistake, however, to diagnose other lesions as neuroparalytic keratitis; in fact this diagnosis is often made on the very slender evidence of the presence of an ulcer, and of some interference with the trigeminal nerve. An ulcer in a cornea, anæsthetic from some previous fifth nerve lesion, should be correctly diagnosed according to the causative organism, for the appearances only vary in degree from those which occur when the same ulcer is observed in a normally sensitive cornea. Keratitis e lagophthalmo, or keratitis xerotica, as it is also called, can generally be correctly diagnosed from the paralytic or cicatricial lesion which has caused it: in such the cornea is only affected in its lower or middle portion, where the exposure has had the greatest effect on the epithelium, while a neuroparalytic ulcer may occur anywhere in the cornea. I have had two cases of a diplobacillary ulcer, one in a cornea exposed by cicatricial ectropion, the other in one anæsthetic from a trigeminal lesion: these presented quite unusual features, and a diagnosis was only made by means of a bacteriological examination. In both cases perforation had occurred, but a good result

was obtained by an occlusive bandage combined with zinc sulphate applications. In both cases the bacteriological examination and the course under treatment were quite sufficient to establish the diagnosis.

Keratomalacia in children and marasmic ulcer in adults are sometimes diagnosed as neuroparalytic lesions, the destruction of the cornea being referred to some weakness in the nerve supply. The general condition of the patient, as well as the local appearance of the eye, are ready means of distinguishing these conditions from each other (see p. 158).

A rational *treatment* of neuroparalytic keratitis is directed in the first place against the lesion which is affecting the trophic nerve fibres. In traumatic cases rest and the use of apparatus to immobilise the part are the indications to be followed. In acute inflammatory lesions the keratitis is probably only an incident in a very severe cerebral lesion, which will be treated on the lines of general surgery. Chronic inflammatory cases are either tubercular or syphilitic, and are amenable to the treatment of these conditions. Cases due to the pressure of tumours on nerves are for the general surgeon.

In all cases where there is any hope of success, especially when the progress of the corneal condition is arrested, some benefit may be obtained from the injection of strychnine subcutaneously, and the application of the constant current—one pole over the eye, and the other behind the ear.

Local irritation of the cornea in any form causes spreading of the ulceration ; the lids, therefore, should be kept closed by means of a light bandage, and the conjunctival sac kept clean. It is not necessary for this purpose to use antiseptics ; indeed, the reaction of the cornea in these cases varies so much that it is better to avoid their use entirely, and to employ a sterile normal saline solution (Aq. Dist. $\frac{3}{4}$ i. Sod. Chlor. grs. $3\frac{1}{2}$ -4).

The *prognosis* depends on the cause : syphilis, hæmorrhage, or simple exudation, are most favourable ; fracture or other traumatism fair ; tumour growth bad. Subject to consideration of the cause, those ulcers which progress rapidly have a bad prognosis ; and with regard to those which are small, stationary, or only intermittently progressive, we may hope, if they are at all amenable to treatment, for a favourable result.

KERATITIS E LAGOPHTHALMO (KERATITIS XEROTICA)

This is a form of keratitis which results from an inability of the patient to close the lids. When such is the case the cornea becomes infiltrated and ulcerates, the amount of destruction varying greatly in different cases. The condition has been variously named, but as Professor Fuchs points out in his *Lehrbuch*, the cause being the exposure of the cornea when uncovered by the lids, the term Keratitis e Lagophthalmo is much better than Keratitis Xerotica. It is not a true xerosis, and this name leads to confusion, seeing that a true xerosis of the conjunctiva is found in keratomalacia,

trachoma, and other diseases, which have no relation to keratitis e lagophthalgo.

Etiology.—Exposure of the cornea may be brought about in three different ways:—(a) by *destruction of the lids themselves*, or scarring around them, so that their effective parts are shortened.

Cuts or tears passing through the whole thickness of the lid, if not accurately sutured, are apt to heal leaving a notch, which is not only very unsightly but exposes a small area of cornea, in which a keratitis e lagophthalgo may develop.

Wounds and burns of the skin of the face may, when scarring, so drag on the lids that they produce ectropion and lagophthalmos. The most common of these scars results from periostitis of the orbital margin, which has led to suppuration and formed a fistula; when healing occurs, adhesions result,—these contract and cause a variable degree of ectropion by dragging on the lids.

(b) *Facial paralysis*, affecting the orbicularis palpebrarum muscle, results in the patient being unable to close the palpebral fissure. To some extent this is compensated for by an upward and inward rotation of the globe, so that when the attempt is made to close the eye the cornea lies under the upper lid; a contraction of the unopposed levator palpebrae superioris sets in after some time, and increases the extent of the cornea exposed. The protection of the lid is only partial, and a variable amount of the lower and outer portion of the cornea may soon show a superficial keratitis.

(c) The gravest cases are due to *protrusion of the eyeball* to such an extent that the lids can no longer cover the cornea. If the movements of the globe are not much affected, as in the case of exophthalmic goitre, the upward rotation of the eye may protect the centre of the cornea; but when the proptosis is due to a new formation at the back of the orbit, and the mobility of the eye is much restricted, or even entirely abolished, a band across the centre of the cornea is permanently exposed. In such cases rapid ulceration takes place, and may lead very soon to panophthalmitis.

□ *Symptoms.*—When the cornea is exposed from the action of any of the causes already mentioned, the first changes take place in its superficial layers: the moist, smooth, even epithelial surface becomes dry, rough, and coated with fine crusts of inspissated mucus and epithelial débris; if this be moistened and removed a fine smoky opacity of the superficial part of the cornea is readily seen. The extent of these changes corresponds to that of the exposed portion of the cornea, and the line of separation of the normal and the altered cornea corresponds to the outline of the lid margins. The part of the cornea protected by the lids is comparatively normal. The conjunctiva, of course, is also exposed to some extent, and here, too, there is a dryness of the surface with a formation of crusts. The whole of such area is intensely injected, and all round the exposed part of the cornea the conjunctiva is very deeply congested, even in parts covered by the lids. The opacity

in the cornea becomes denser, the surface epithelial cells die and are cast off, until the parenchyma of the cornea is laid bare, or is merely covered by the crust which may have formed. Then we have an ulcer of the cornea, and associated with it is a variable degree of iritis, sometimes with hypopyon. In severe cases the affected part of the cornea becomes necrotic, and perforation occurs, which may lead to panophthalmitis; this grave result is most often observed in those cases which are due to a high degree of proptosis.

In practically all cases the lower part of the cornea is affected. In the normal closing of the lids the upper descends to a greater extent than the lower ascends; at the same time the eyeball rotates upwards, so that the palpebral fissure in the closed eye corresponds almost to the lower border of the cornea. When the lagophthalmos is due to some rather peculiar condition which would obviously cause the upper part of the cornea to be exposed—for example, proptosis combined with marked downward displacement of the globe—the corresponding keratitis will, of course, affect the exposed portion of the cornea.

The subjective phenomena are not severe. When there is iritis, we have the usual symptoms of that condition, but they are not so severe as the objective findings would lead us to suppose.

Pathology.—The direct cause of the keratitis is the entrance of cocci through the dessicated and unresistant epithelium,—the organisms are probably of no great

virulence, and although the bacteriology of the condition is not satisfactorily known, the organisms generally found are various staphylococci.

In the epithelium necrotic changes occur, the cells are then cast off and the parenchyma laid bare, a small-celled infiltration of the superficial lamellae occurs at the denuded area, and sometimes a slough is separated off so as to leave a shallow depression. Rarely a deep infiltration of the cornea also occurs, forming what has been called an onyx. When the exposure is constant the ulceration advances by successive layers of the cornea becoming affected until finally Descemet's membrane gives way and panophthalmitis results. In the healing stages the depression is covered with epithelium, and under which vessels and connective tissue are carried in from the episcleral tissues.

There is not necessarily an anæsthesia of the cornea; of course, where the surface epithelium has been destroyed, the sensibility will be lowered; but this is not very marked, and is only in proportion to the actual drying and dessication which has occurred.

The *diagnosis* presents no difficulty. Complicated cases may occur, in which a mixed keratitis e lagophthalmo and keratitis neuroparalytica occur, when perhaps the latter factor may escape notice, until the failure of treatment calls attention to the more complicated nature of the case.¹

¹ It is hardly necessary to emphasise the fact that keratitis due to paralysis of the seventh nerve is not 'neuroparalytic keratitis' as the term is used in this work.

The *prognosis* depends entirely on the cause and the possibility either of removing it or protecting the cornea. It is worst in cases of orbital tumour, less bad in cicatricial cases, and perhaps most favourable in simple paralytic cases.

The *course* and *treatment*.—In mild cases due to paralysis of the orbicularis, bandaging the eye so as to keep the lids closed, and frequent washing out of the conjunctival sac, will induce a cure; but on cessation of treatment the keratitis begins anew. In some of these cases, where only the lower margin of the cornea is exposed, a natural cure may result from the formation of a pseudopterygium; the conjunctiva grows over the exposed part, and the result is a vascular epithelial covered surface which can resist the drying action of the air and causes no discomfort: such a state of affairs can readily be produced artificially by scraping the affected area and grafting on to it a flap of conjunctiva. In many paralytic cases an operation is necessary to reduce the size of the palpebral fissure to such an extent that the upward rolling of the eye causes the cornea to be completely covered. There are several excellent methods of performing tarsorrhaphy to be found in any textbook. That peculiar form of lagophthalmos which occurs in exophthalmic goitre, and may give rise to a keratitis, must be treated by this operative method. When the cause is a cicatricial ectropion or a defect of the lid—as in cases where the lower lid has been removed

for malignant growths—plastic operations to restore the lids must be undertaken.

In those cases where orbital growths are causing the protrusion of the eyeball, operative proceeding to remove such growths must be undertaken; the Krönlein operation is indicated in the majority of such cases.

KERATOMALACIA OR MARASMIC ULCER OF THE CORNEA

This is a condition allied to the preceding forms of ulceration; it is not due to any special infecting agency, but to the lowered resistance of the tissues attacked. It would be incorrect, therefore, to classify such conditions according to the organisms found in the cornea: the best method seems to be to put them in a group along with the other two forms of ulceration which have just been discussed. We have to do with a necrosis spreading from a single focus in a badly nourished cornea, and causing its destruction over the greater part of its extent down to Descemet's membrane. It occurs in two conditions. In adults passing into a comatose state before death from some exhausting fever, ulceration may occur. The secretions of the whole body diminish, the conjunctiva and cornea become dry; sensibility is dulled, and the partly opened eyes do not respond to any irritation of the cornea. The motionless lids press heavily on the dry and dessicated epithelium, which begins to be cast off. The resisting powers of all the tissues are very low.

Without any inflammatory reaction a white opaque spot appears in the dull and cloudy cornea at the edge of the lid; it extends transversely; the surface over the infiltration gives way, and an ulcer results which rapidly destroys the whole cornea.

The second form occurs in very young children of low vitality, bad nutrition, and feeble resisting power. Their strength has usually been reduced by some form of gastro-intestinal catarrh, till they are in a condition not very far removed from that of the adults already mentioned. The corneal sensibility is lowered, the lids remain partly opened, so that the epithelium becomes dry, and the dirt, so plentiful in their surroundings, infects the cornea. A dense white infiltration rapidly develops in its substance, the epithelium is cast off, the lamellae separate like layers of wet paper, finally leaving Descemet's membrane bare and bulging forward. Destruction is usually complete in two or three days, but sometimes the process is slower and does not affect the whole cornea.

There are two conditions which may produce a keratomalacia :—

- (1) A resisting power almost absent, a very slight traumatism, and a mild or doubtful infection.
- (2) A lowered vitality, and an infection of moderate severity.

Looking at these from the point of view of the resistance of the patient, they can both be classified as Marasmic Ulcer of the Cornea. That occurring in

the comatose adults needs no further consideration, it most nearly resembles the acute bedsores found in the same patients. The marasmic ulcer in children occurs during the first few weeks of life, very rarely after the sixth month, and may destroy both corneae in bad cases. Many of these children do not survive long, the corneal condition being merely an index of their general health. The actual infecting agent found in the cornea may be the pneumococcus, gonococcus, bacterium coli commune, staphylococcus, streptococcus, or any mixture of these organisms. The worst case that I have ever examined was due to a mixed infection of coli communis and pneumococcus in an ill-nourished baby of six weeks old; thirty-six hours after the cornea became infected only Descemet's membrane remained, covered with shreds of adherent necrotic tissue.

The process is an acute necrosis; the corneal tissue is filled with lymph which coagulates, and death *en masse* takes place. There is only a very slight inflammatory reaction, no trace of œdema of the lids, though there is nearly always a slight chemosis of the conjunctiva. It seems certain that hereditary syphilis has a considerable bearing on the causation of keratomalacia. Peltessoehn has recorded two cases of children under one year old who presented this condition, and in whom an inunction cure was attended with excellent results. Knaebel's statistics of eighteen cases of keratomalacia infantum are instructive; the youngest child affected was three weeks and the oldest six months.

Half the cases were bilateral. Fifteen were artificially delivered, the remaining three being premature. Only three of the children were breast-fed. Nine children died, and the remaining nine had thirteen eyes affected, eleven of which were finally completely blind.

The *treatment* of keratomalacia must be directed towards remedying the general condition of the patient, and locally to combat the infection and necrosis which it causes. The children are almost always hand-fed, their diet must be very carefully attended to,—the modern patent food must be looked upon with great suspicion. Carelessness and ignorance with regard to their feeding have often caused an intestinal catarrh leading to wasting and lowered vitality. A change to fresh cow's milk humanised by the addition of barley-water and sugar often causes a remarkable general improvement as well as a local one. If there is any reason to suspect a congenital syphilitic condition an inunction cure should be vigorously carried out.

Locally the conjunctival sac and ulcer should be washed out very often with large quantities of sterile saline lotion or boric acid, antiseptics are not necessary for the corneal condition; if there be any conjunctivitis present it should be treated according to general principles. Hot fomentations and atropine tend to restrain the spread of the necrosis as well as assist the separation of the slough. If the tension keeps high the thinned cornea will bulge, and a staphyloma or ectasia

corneae result; this complication is best met by a Saemisch's section. The cautery should not be used, as the tissue reaction of the cornea is so low that it would only cause an extension of the necrosis.

Besides keeping the cornea clean it must be protected against further irritation; a light bandage, only sufficient to keep the eye closed, should be applied so as not to press on the globe. As soon as any improvement in the general health begins the cornea will also begin to clear. The destruction of tissue has, however, been so great that a very dense leucoma will remain, and it is rare that useful vision is conserved.

DENDRITIC ULCER OF THE CORNEA

This condition was first described by Hansen Grut, and later in 1885 by Emmert, who gave it the name of *Keratitis Dendritica*. Grut describes the condition as 'a Keratitis which is essentially superficial and of little density, with a great tendency to spread by nodules and branches, the edges of which are much denser than the central portion. Its course is rather chronic; injection, photophobia and pain are very slightly marked: the nature of the disease is probably mycotic.' Emmert describes the same condition: 'A greyish subepithelial opacity appears in some part of the cornea; it begins as a small nodule, from which branches are sent out; or as a small linear opacity, which becomes elongated and sprouts to either side. The epithelium is cast off and

thus small channels are formed ; the main branch remaining narrow.'

Following Hansen Grut's description and expressed opinion of the nature of this condition, there has been a tendency to look upon dendritic ulcers as directly infective ; we must, however, admit that there is no evidence in support of such a contention ; repeated bacteriological examination of such ulcers has failed to establish even the consistent presence of an organism which might be considered as causal.

The true origin of the ulcer is probably a *Herpes Corneae Febrilis* (Horner). In most cases a history will be obtained of some chill, slight rigor, or acute coryza a few days before the onset of the corneal condition ; many attacks follow influenza. There is a slight superficial conjunctival injection with photophobia and lacrymation ; careful examination may show some slight superficial disturbance of the cornea ; patches of anæsthesia are very often found, and patients often complain of the sensation that a foreign body is in the cornea. After a day or two a few minute vesicles appear on the corneal surface and soon burst, leaving the fine branching channels which are typical of the disease ; until this stage there is only an extremely faint opacity to be seen in the epithelium, but after rupture of the vesicles the margins of the fine channels become grey, so that we have a double contour on the lines thus formed. In this stage the exact limits and distribution of the epithelial loss are beautifully shown by fluores-

cine staining. The subjective phenomena lessen, and often are quite insignificant when the case is advanced, a disturbance of vision being usually the most prominent complaint. The further course of the disease is very chronic; the epithelium shows little tendency to grow over the ulcer, and several weeks elapse before healing is complete, relapses and fresh outbreaks in the form of new branches from the original one are very common; and even after sound healing has taken place recurrences are not uncommon. A faint branching

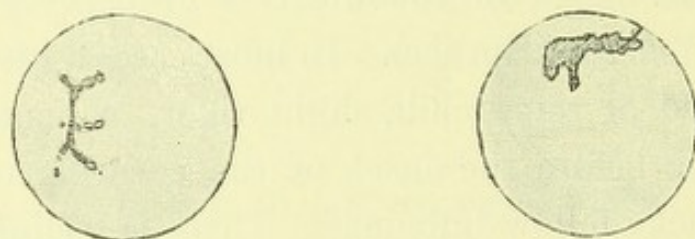


Fig. 18.

DENDRITIC ULCER.

nebula remains for a very long time at the site of the ulceration.

The *pathology* of this condition is not understood; the essential lesion is probably connected with the nerve terminals in the epithelium, and so the disease may be looked upon as trophic: the long course and tardy healing would thus be accounted for. Probably there is a collection of fluid under the epithelium, and rapid necrosis of the cells overlying the vesicles, the increasing opacity being caused by a small-celled infiltration at the site of the vesicles. In many ways the disease resembles the herpes labialis so often associated

with acute coryza. As regards the bacteriology, no special organisms have been put forward as the cause; the supporters of the mycotic theory apparently do not restrict themselves even to any group of bacteria. The claim that the disease is infective cannot at present be taken seriously.

Treatment.—The object naturally is to cut short the attack and to promote rapid healing, and for this purpose it is best to use rather vigorous means at the outset. The cornea should be cocainised, stained with fluoresceine and thoroughly dried with absorbent paper; the rills, which show up very well, should then be carefully scraped out with a fine spud, and then the abraded surface thoroughly painted with pure carbolic acid, absolute alcohol, or tincture of iodine, so that no small branches, however minute, escape. Pure carbolic appears to me to be best, as the pain which follows is much less, and although the whitening of the surrounding epithelium at the time of application appears rather serious, it clears away and leaves a very faint nebula. The carbolic can be applied on the end of a pointed wooden match, and the excess dried up with blotting paper. Atropine and a bandage should be used until the surface is completely healed.

It is of course necessary to make a careful examination of the lacrymal sac and nasal duct, as in the case of a simple corneal abrasion (see p. 11).

CHAPTER X

COMMON ORGANISMS FOUND IN CORNEAL ULCERS

COCCL.

Gram positive.

Pneumococcus.

Streptococcus.

Staphylococcus albus, aureus, and citreus.

Gram negative.

Gonococcus (Neisser).

Pseudogonococcus (Kayser).

BACILLI.

Gram positive.

Bacillus Xerosis.

Bacillus Diphtheriae (Klebs-Loeffler).

Bacillus Pseudodiphtheriticus (Hoffmann).

Bacillus Subtilis.

Gram negative.

Bacillus Duplex. *a.* Morax-Axenfeld.

β. Petit.

Bacillus Ulceris Corneae (Zur Nedden).

Pneumobacillus (Friedländer).

Bacillus Koch-Weeks.

Bacillus Influenzae.

Bacillus Müller (?).

Bacillus Pyocyaneus.

VARIOUS.

Bacterium Coli Commune.

Streptothriciae, Aspergillus Fumigatus.

These organisms can usually be very readily distinguished by their mode of growth and their reaction to Gram's stain. They are readily divisible into four groups :—

- I. Cocci which are Gram positive.
- II. Cocci which are Gram negative.
- III. Bacilli which are Gram positive.
- IV. Bacilli which are Gram negative.

In conducting a differential diagnosis we consider first the morphology of the organism in smear preparations, attention being paid to its shape, size, and variability; the presence or absence of capsules, and the arrangement of the organisms in groups, whether irregular, in clusters, or in chains. Then its culture peculiarities must be considered, for which purpose agar, agar with ascites fluid, and solidified blood-serum media are usually sufficient. In a few cases it may be necessary to test the virulence, for which purpose a bouillon culture twenty-four hours old is generally used, and the result of injecting 1 cc. into a guinea-pig is noted.

Proceeding in this way, we find that the Gram positive cocci which form Group I. may occur in chains, and grow sparsely on blood-serum or agar when we are dealing with the pneumococcus or streptococcus; or in

clusters with vigorous growth on the same media in the case of the various staphylococci.

The cocci of Group II. are differentiated by cultures ; the pseudogonococcus grows rapidly on agar, and the gonococcus on human blood-serum and not on agar. In Group III. the septate bacilli are readily distinguished by their striated appearance and variable shape : these are the *Bacillus Xerosis*, *B. Diphtheriae*, and *B. Pseudodiphtheriae* ; they grow on serum and agar. The non-septate *B. Subtilis* is larger, longer, and quite different in appearance and growth. Group IV. contains many organisms, and their diagnosis practically is more difficult ; an experienced observer may recognise many of them in a simple smear preparation, but the exact diagnosis of some of them is a matter of great difficulty. Their growth on blood-serum divides them into a class which liquefy this medium, and one which either fails to grow or does not cause liquefaction. The former class is very important in ophthalmology and contains the bacillus duplex, a comparatively large organism growing in chains of paired elements, staining the media brown ; and the fluorescent bacilli of which *B. Pyocyaneus* is the most important, small motile organisms variable in size causing a blue or green colour in the media. Amongst those which do not liquefy blood-serum we have the hæmophiles, which only grow on media containing hæmoglobin, the Koch-Weeks, Müller's, and the influenza bacillus, and two organisms which grow on agar and in shape and size resemble closely the

B. Duplex : these are Friedländer's *Pneumobacillus* and Zur Nedden's *Bacillus* ; the former causes septicæmia in mice, the latter does not. Besides these organisms members of the coli group are sometimes met with, Gram negative bacilli of varying size and shape, growing without liquefaction on blood-serum or agar, and forming gas when sugar is present in the media.

This classification, which has no pretence to be biological, can be tabulated for convenience.

Cocci.	I. Gram positive.	Growth in chains : on blood-serum easily, on agar sparsely.	<i>Pneumococcus.</i> <i>Streptococcus pyogenes.</i>
		Growth in clusters : on blood-serum and agar readily.	<i>Staphylococcus pyogenes.</i> Albus, Citreus, or Aureus.
	II. Gram negative.	Growth on agar.	<i>Pseudogonococcus</i> (Kayser).
		Growth on human blood-serum.	<i>Gonococcus</i> (Neisser).
Bacilli.	III. Gram positive.	Septate. Growth on blood-serum and agar. Indistinguishable in appearance.	<i>B. Xerosis.</i> <i>B. Diphtheriae.</i> <i>B. Pseudodiphtheriae.</i>
		Non-septate. Growth on blood-serum and agar. Different from above.	<i>B. Subtilis.</i>
	IV. Gram negative.	Liquefying blood-serum.	Large, brown colour on acites agar. <i>B. Duplex.</i>
			Growth on agar. <i>Petit type.</i> None on agar. <i>Axenfeld type.</i>
		Do not liquefy blood-serum.	Small, motile fluorescent. <i>B. Pyocyaneus.</i> Forming Pyocyanin pigment.
			Hæmophiles grow only on media containing Hæmoglobin. <i>B. Koch-Weeks.</i> <i>B. Müller.</i> <i>B. Influenzae.</i>
		Grow on agar.	<i>B. Zur Nedden.</i> <i>B. Friedländer.</i> Pneumobacillus (causes septicæmia).

Bacteria. Variable in appearance. Gram negative bacilli and cocci. Motile, gas formation in sugared media. } Organisms of the coli group especially. *B. coli commune.*

There are only three groups which cause any difficulty in diagnosis:—

GROUP I.	{ Pneumococcus. Streptococcus.	} Gram positive cocci.
GROUP III.	{ B. Xerosis. B. Diphtheriae. B. Pseudodiphtheriae.	} Gram negative bacilli septate.
GROUP IV.	{ B. Koch-Weeks. B. Müller. B. Influenzae.	} Gram negative hæmo- philes.

It is a simple matter to place any one of the above organisms in its proper group, but it may require difficult and complicated bacteriological methods to distinguish the individuals. Those in Group IV. are readily distinguished from the others, but it is outside the powers of any but a skilled bacteriologist to decide which one is present in a given case; as the differentiation is of very slight practical utility, with regard to corneal ulcers, it will not be discussed.

In Group III. we are confronted with a similar difficulty, but in this case the differential diagnosis is of great importance with regard especially to membranous conjunctivitis. It is to some extent outside the province of corneal ulceration.

172 ULCERATION OF THE CORNEA

PATHOGENIC FORM.

Klebs-Loeffler Bacillus.

NON-PATHOGENIC.

Pseudodiphtheria Bacillus.

Bacillus Xerosis.

Hoffmann's Bacillus.

Growth on agar or bouillon produces an acid reaction in the medium.	On agar or bouillon produces an alkaline reaction.
---	--

Staining with a weak aniline dye shows many Babes-Ernst bodies at the ends of the bacilli; these take on the stain, while the general protoplasm remains clear or very faintly stained.	Babes-Ernst bodies very rarely seen, therefore no polar staining.
---	---

Inoculation produces local and general reaction; usually death in susceptible animals.	Non-pathogenic for animals.
--	-----------------------------

There is also a slight difference in the size, shape, and arrangement of the organism, when grown in parallel cultures, which, however, is only reliable in the case of a very experienced observer.

Neisser's stain for the Babes-Ernst bodies:—

With Sol. A. Methylene blue,	. gr.	1
Alcohol 96 per cent.,	ccm.	20
Acetic acid,	. ccm.	50
Water,	. ccm.	950

Stain for one to three seconds, and then wash with water.

With Sol. B. Bismarck brown,	. grs.	2
Water,	. ccm.	1000

Stain for three to five seconds.

The smear should be made from a growth at 34°-36° C. on blood-serum or agar nine to twenty-four hours old.

To test the pathogenic power inject a series of guinea-pigs of 250-350 grams weight, with .1, .3, .5 ccm. of a twenty-four hours old bouillon culture, subcutaneously in the chest wall; in twenty-four hours there is œdema, and about two to three days, death. At the site of injection is an œdematous blood-stained swelling; the axillary glands are enlarged. In the pleural cavity is an effusion of fluid containing the bacilli; the suprarenals are enlarged and dark red in colour, and sometimes white nodules occur in the omentum.

The most important problem which the ophthalmic bacteriologist has to settle is the diagnosis of pneumococcus. Given an organism which comes into Group I., the question of its nature and virulence must be settled before treatment can be exactly indicated. The important question to decide is whether the organism present is the pneumococcus or not.

The pneumococcus varies very considerably in its appearance, growth, and pathogenicity; when growing rapidly in an acute conjunctivitis, or the advancing edge of a pneumococcal ulcer, it approximates a type having a definite form, mode of growth, and virulency for susceptible animals.

Growing merely as a saprophyte on the conjunctiva, or in old cultures on unfavourable media, it shows *atypical* and *degenerate forms* which vary from the

type in all these particulars, but which can be readily transformed into it by suitable means of culture.

The type is a diplococcus, each member of which is slightly elongated, often lancet or flame shaped, with the pointed end more often pointing away from than towards the other. The pair are enveloped in a fine capsule. Division is by transverse, and never by longitudinal cleavage.

Atypical forms are more or less round, and may form chains of three or four members of varying shape; they may or may not have capsules. When degenerate the organisms may be larger or smaller than the type, are often rod-shaped with clubbed ends, they may stain badly and partially decolorise with Gram's method.

The pneumococcus grows best on a mixture of agar and human ascitic fluid: surface colonies after twenty-four hours at 36° C. appear as small transparent drops like condensed water; by transmitted light they are transparent and may show a slight bluish opalescence. In the condensed water there is a slight turbidity or precipitate. The medium should be neutral or faintly alkaline. Rapid growth occurs in fluid rabbit's serum from young animals (Römer), and on a mixture of human blood-serum and agar (Weichselbaum).

The atypical and degenerate forms may fail to grow on ordinary media, but on any of those given above they can generally be cultivated.

The pneumococcus is pathogenic, especially for mice and young rabbits, but it is susceptible to adaptation in a high degree. A standard culture at first equally virulent for rabbits and mice, after passage through mice may kill a mouse in twenty-four hours with a dose of $\frac{1}{100,000}$ ccm., and yet take .2 ccm. for a rabbit. The adaptation is rapidly broken down, and the apparent virulence of an adapted stem may increase three hundred-fold by passage through a single animal (Römer).

The virulence of the particular organism must therefore not be used for the diagnosis of a Gram positive coccus without great care. But if the organism be grown in fluid serum from a young rabbit, and then the fatal dose for rabbits determined by experiment, the virulence thus ascertained can be relied upon.

From the heart's-blood of the experimental animal a smear preparation is made and stained by Gram's method; the presence of the typical encapsulated diplococcus allows a positive diagnosis of pneumococcus to be made.

CHAPTER XI

OPERATION FOR EXCISION OF THE LACRYMAL SAC (AXENFELD)

THE methods of excising the lacrymal sac are very various; on the Continent the Kuhnt-Völckers method is very popular, and those of Graefe, von Hoffman, and Czermak, in the hands of able exponents, give very good results. In England, Lang's method is used to some extent. Professor Axenfeld has elaborated the operation by attention to detail, so that it can take a place amongst the classical surgical procedures; his method is based on an accurate knowledge of the anatomy of the sac and a clever device for checking hæmorrhage, the great cause of difficulty in all lid operations, and especially in this one. The landmark to which all topography is referred is the crest of the lacrymal bone; a mark readily found by putting the internal palpebral ligament on the stretch, and following it in to the lacrymal bone. This crest marks the inner boundary of the sac, which lies immediately behind the tarsal ligament and projects above it to the extent of 3-4 mm. The capsule of the sac is closely adherent to

the periosteum of the lacrymal bone, and between these two layers lies the vascular plexus which is the cause of the troublesome bleeding so often occurring during the operation. The periosteum of the lacrymal bone is only loosely adherent to it, and when separated the vitality of the bone is unimpaired.

The plan which Axenfeld adopted was to make a large incision and thus get a free view; to incise the periosteum in front of the crista lacrymalis, and then separate it with the contents of the fossa lacrymalis from the bone, thus working in an avascular plane, and having a bony wall as a guide, any chance of cutting into the sac before its limits were clearly defined were avoided. The size of the incision increased the amount of hæmorrhage from the superficial structures; this was obviated by an extremely ingenious compression retractor. To dry out the wound, wooden rods of 20 cm. long, on the ends of which a tuft of cotton wool is wound, serve admirably, and keep the operating area clear of assistants' fingers, besides allowing of efficient compression of the deeper parts of the wound.

A curved incision is made about 2 or 3 mm. in front of the crista lacrymalis, commencing 5 mm. above the internal tarsal ligament and passing downwards and outwards in a curve corresponding to the orbital boundary, and in a sulcus normally present at the edge of the orbicularis palpebrarum. This incision is carried down to the bone with one cut, and the periosteum

laid bare throughout its extent; a Müller's speculum, with extra long and curved hooks, is then placed lengthwise in the wound and opened about 1 cm. The Axenfeld *Sperrer* is then adjusted with the longer hooks downwards and placed transversely in the wound, so that they catch up all the soft parts down to the periosteum, and then expanded by pressing on the ends of the transverse bar till the bleeding from the lips of the wound ceases. On drying out the wound, we find an oblong area of bone laid bare, and in it can readily be seen the crista lacrymalis, with the original incision in front of it, and the lacrymal fossa and sac behind it, the sac being crossed by the tendo oculi. The periosteum is completely divided along the line of the original incision, or if this be not visible, about 2 mm. in front of the crest, and then elevated outwards to the fossa; crossing the crest the elevator is turned at right angles to the surface, and can be pushed down into the fossa between the sac and periosteum on the one side, and the lacrymal bone on the other. The inner wall of the sac is thus freed up and down throughout the whole extent, and can be also separated from the posterior wall of the fossa (see Fig. 19). Seizing the sac with a dissecting forceps, its dome-shaped upper end can be freed by a few snips with a curved scissors, the upper and lower canaliculi come into view, the sac is drawn over towards the nose, and they are cut through from above and behind. The sac is now free except below where it passes into the nasal duct, and in front where

the remains of the tendo oculi attach it to the lids, and a firm fascial band attaches it to the anterior lip of the upper opening of the nasal duct. By drawing the sac upwards and inwards, and rotating it outwards, these can be divided close to it so as to leave as much tissue as possible behind. A curved sharp-pointed scissors is pushed down the duct behind the sac, which is drawn upwards, and the duct divided as low as possible. The cut mucous membrane can be curetted with a sharp spoon and touched with pure carbolic on a dressed probe.

The wound is closed by two sutures taking up the whole depth of the divided tissues; one is placed opposite the internal canthus and brings the remains of the tendo oculi into position, and the other about 8 mm. lower; the inferior part of the wound is best left open for drainage, as the lips here fall together without suturing.

A graduated compress is adjusted so as to obliterate the cavity, and a firm bandage applied. Healing by first intention is the rule, and the stitches can be removed in five or six days. The resulting scar is so slight as not to be noticeable unless particularly looked for, the disfigurement being much less than that caused by merely slitting up the canaliculi.

The epiphora, after the operation, is always much less than it was before, provided the whole of the sac be removed; this at first appears anomalous, but the explanation is very simple. Tears do not flow down a

healthy nasal duct under normal circumstances ; sufficient tears are secreted merely to supply evaporation from the cornea and moisten the conjunctiva and mucous membrane of the nasal duct. The epiphora in a nasal

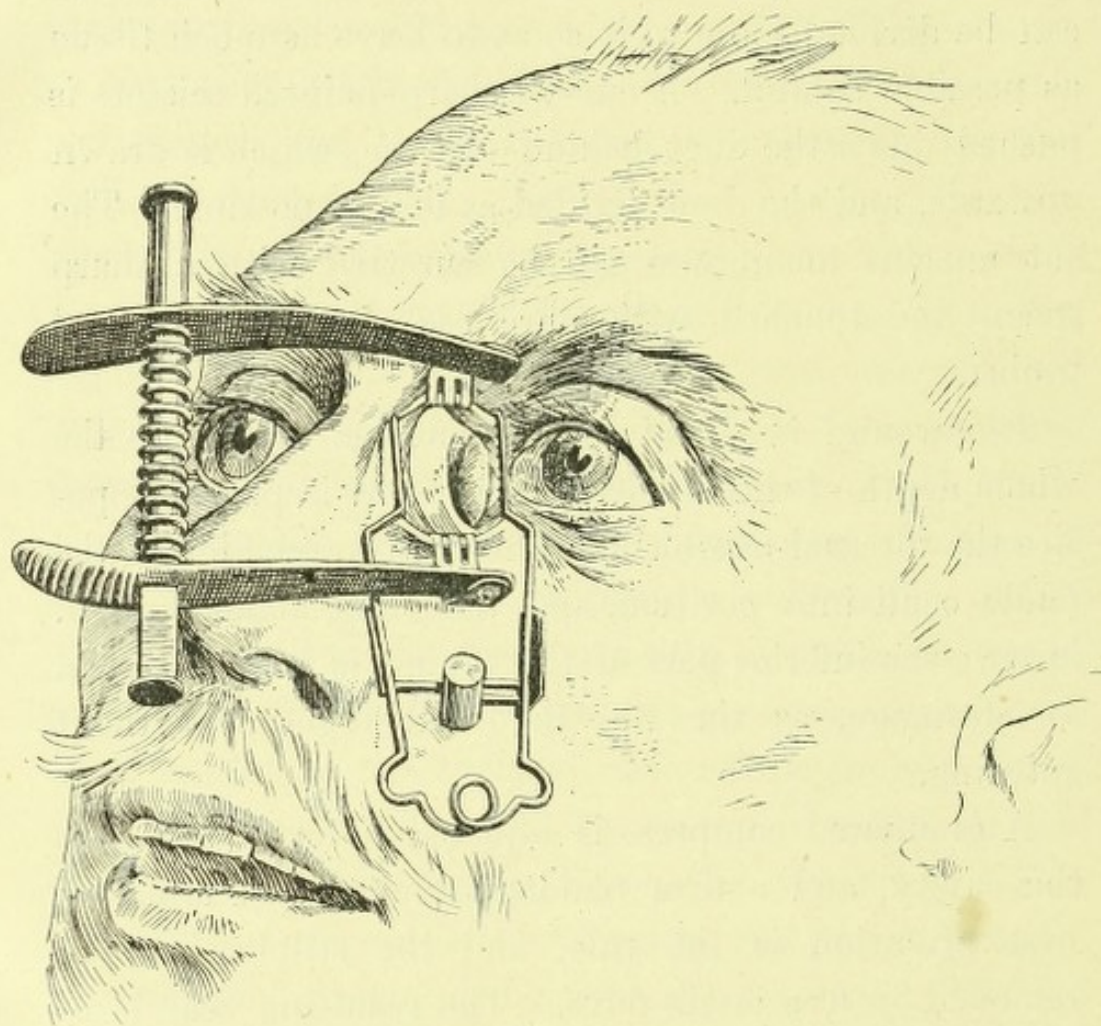


Fig. 19.

EXCISION OF SAC. RETRACTOR IN POSITION. INNER WALL OF SAC FREED.

duct stricture is not due to the fact that the tears cannot pass into nose. A stagnant pool is formed in the sac filled with albuminous fluid, débris from its walls, and organisms, usually pneumococci ; this induces a chronic inflammatory condition in the mucous mem-

brane, exactly as does a stricture in the urethra. Irritation of the cornea, conjunctiva, or nasal mucous membrane causes a reflex increase in the lacrymal secretion, which, not being able to evaporate or escape into the nose, shows itself as an epiphora. When the sac is removed this reflex stimulation of the lacrymal gland usually ceases at once, although it may persist as a habit for a few months, and the flow of tears, under normal conditions, is only sufficient to suitably moisten the cornea. Under abnormal conditions, such as a cold wind, or emotion, the epiphora is more than that of a normal individual, inasmuch as the winking movement of the lids would diminish the overflow if the nasal duct were patent.

THE OPERATION OF SAEMISCH SECTION

The object of this operation is to thoroughly open the anterior chamber, so that its contents escape and all tension is removed from the cornea. It is indicated in purulent corneal ulcers, which in spite of treatment are spreading deeply and threatening to perforate. It is not usually necessary in the pneumococcal ulcer, unless the case has been neglected until the greater part of the cornea is destroyed. The cornea is incised so that the floor of the ulcer is divided, and the incision is at right angles to the infiltrated edge of the ulcer. The two ends of the incision should lie in the intact cornea outside the ulcer.

The point of a Graefe knife is introduced into the

anterior chamber on one side of the ulcer, and about 2 mm. from its margin, the blade is held so that its edge is forwards and its back towards the iris and pupil, the knife is then pushed across the chamber behind the ulcer, and a counter puncture made in the sound cornea on the opposite side of the ulcer; with a slight sawing movement the base of the ulcer is completely divided from behind forwards, the aqueous and its contents escape, and the chamber empties com-

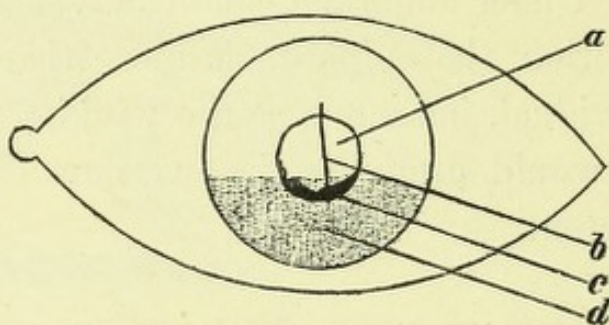


Fig. 20.

SAEMISCH SECTION AFTER SCHMITZ.

a Ulcer; *b* Section; *c* Advancing edge; *d* Hypopyon.

pletely; at the moment of completing the section the pain is very severe, and the operator should be prepared for a movement on the part of the patient. The wound should be opened with a spatula and the chamber emptied every day until the ulcer has cleaned, and is healing (Fig. 20).

In a certain number of cases this operation results in the ulcer at once commencing to heal, the infiltration of the cornea decreases, and vessels grow into the ulcer. Unfortunately, prolapse of the iris into the wound is a common mishap, although in the Bonn clinic this unfor-

tunate result does not seem to occur so frequently as to be considered a serious objection to the operation.¹

While we must admit that a proceeding which consists in opening an aseptic space through the floor of a septic ulcer does not fall in line with the principle of aseptic surgery, this operation has the support of many surgeons of great clinical experience, and certain pathological facts can be brought forward to support it. If a culture of pneumococci be introduced into a pocket made in the cornea a purulent ulcer will result, while if the same culture be introduced into the anterior chamber the results are not nearly so severe, indicating that the aqueous has some power of counteracting the virulence of the organisms. Those forms of ulceration in which the greatest benefit is to be derived from this operation are those which tend to a perforation and infection of the anterior chamber, and, when this space is septic, the objection to opening it is in a great measure removed.

THE CORNEAL PLASTIC OPERATION

When an ulcer has penetrated deeply into the cornea, so that the membrane of Descemet is exposed, and more especially when the ulcer has perforated into the anterior chamber, it is advisable that the loss of substance thus occasioned should be made good as soon as possible, and for this purpose a plastic operation can often be employed.

¹ Through the kindness of Prof. Saemisch the author has had the opportunity of seeing cases operated on in this manner in Bonn, in which the results were excellent.

An incision is made around the cornea and concentric with its margin so that the line joining its two ends passes over the central edge of the ulcer, a second incision is made parallel to the first, and separated from it by a space equal to the breadth of the ulcer, and the conjunctival bridge between them raised up from the sclera, any débris in the floor of the ulcer is scraped away, and if the floor be perforated the iris is freed from entanglement. The conjunctival bridge is then laid across the cornea so as to cover the ulcer, and steadied in position by a stitch at each end by taking up the excess. The edges of the conjunctival gap from which the flap has been removed are brought together by sutures and the lids are gently closed, leaving the flap in position over the ulcer, and a firm bandage applied. The flap will heal into the ulcer, and the bridge will serve to bring plastic tissue for its rapid repair; the ends of the bridge atrophy and disappear, leaving a firm scar in the situation of the ulcer.

THE REMOVAL OF MATERIAL FROM AN ULCER FOR EXAMINATION

In scraping out an ulcer for diagnostic purposes it is necessary to remove material likely to contain the infecting organism with as little damage to the sound tissues around as possible. The exact method of procedure will therefore vary with the special clinical type of ulcer under consideration, but there is a certain routine to be observed in all cases.

The eye should be well cocainised, and the conjunctival sac and the floor of the ulcer thoroughly washed out with some sterile solution, such as normal saline, not an antiseptic; this is best done by means of a small douche can, and a fine nozzle, so that a stream can be directed into the ulcer. Having removed the débris and exposed that part of the cornea where active changes are occurring, we must remove some of the tissue where the infiltration is densest. For this purpose the small platinum spoon and the spud designed by Zur Nedden are very useful; with such a sterile instrument some of the infiltrate is scraped out of the floor, or from underneath the edge of the ulcer, and with the material thus obtained suitable tubes are inoculated and cover-glass preparations made.

The difficulty in obtaining a satisfactory preparation will vary very much in the different forms of ulceration: from a severe purulent ulcer discharging freely from its base, it is very easy to obtain a positive result; but in the case of a superficial infiltrate commencing to break down, such as has been described in the diplobacillary or Zur Nedden's ulceration, the difficulty of getting sufficient material for a satisfactory examination is very much greater. In these cases it is advisable to make a culture, both on account of the paucity of material available, and the difficulty in giving a diagnosis merely from the microscopical appearance of the organisms.

In the case of an ulcer of the serpiginous type, it is,

of course, essential that the advancing edge of the ulcer be examined; the overhanging edge should be clipped away and some of the densely infiltrated underlying material removed with the scoop. As, of course, such a proceeding will open up fresh tracks for the infection to pass further into the cornea, it is advisable that the cautery should be used immediately the bacteriological examination is completed. In all cases where the cautery is not indicated some antiseptic such as protargol, ten to twenty per cent., should be applied to the freshly scraped surface to prevent further infection.

A light bandage should be applied to the eye for several hours to prevent the dessication of the epithelium which occurs after the use of cocaine.

In staining smear preparations it must be borne in mind that only a small amount of material is available, and that all possible information must be got from usually not more than two preparations. It is advisable to stain the smear by Gram's method. Amongst the many variations of this stain the following will be found satisfactory:—

Solution (a)	Methyl violet five per cent. in water,	88 parts.	}
	Absolute alcohol,	12 parts.	
	Anilin oil,	2 parts.	
Solution (b)	Iodine,	1 part.	}
	Pot. Iodide,	2 parts.	
	Aqua,	200 parts.	

Stain the preparation for 45 seconds in solution (a), wash in water, then treat with solution (b) for 25 seconds,

dry with blotting-paper and decolorise with absolute alcohol for 1 minute, wash with water and counterstain with five per cent. safranin for 20 seconds.

Or the following :—

Solution (a)	Gentian violet five per cent., .	88 parts.	}
	Anilin oil,	2 parts.	
	Absolute alcohol,	100 parts.	
Solution (b)	Iodine,	1 part.	}
	Pot. Iodide,	2 parts.	
	Water,	300 parts.	

Stain for 25 seconds with (a), wash with water, then 15 seconds with (b), decolorise with alcohol till no more colour washes out, wash with water, and counterstain for a few seconds with a watery solution of safranin, 5 %.

The secret of success with Gram's method lies in having the staining solutions freshly made, either of the above will give good results for a few days after mixing; but when a few days old the Gentian violet is partly precipitated, and the results are very unsatisfactory. My own practice is to keep the Gentian violet separate from the rest of the Gram solution, and by means of graduated pipettes make up a small quantity of the mixture every morning. By having everything ready the making of a Gram preparation takes only about 60 seconds more than a simple methylene blue one, and this time is more than made up by the greater ease and rapidity of diagnosis.

INDEX

ABRASIONS, Corneal—

- Symptoms, 13-14 ; pathology, 14-15 ; prognosis, 15-16 ; treatment, 16-17 ; sources of infection, 46.
- Abscess, posterior corneal, 42.
- Adenoids, 130.
- Adrenalin, 54.
- Agar culture, 70, 75, 106, 114, 167-74.
- Agglutination test, 44.
- Airol, 57, 78, 109, 121.
- Alcohol, absolute, 165.
- Amboceptor, action of an, 49.
- Anæsthesia, corneal, 105, 148, 163.
- Anæsthetics, employment of, 64, 119, 125, 131.
- Andrade, Prof., 94.
- Anel's syringe, 54, 107.
- Angular conjunctivitis, 99, 101, 104, 107.
- Antiserum, production, 45-49.
- Antitoxine injections, 123.
- Aqua chlori, 57.
- Argyrol, 128.
- Arlt, Prof., 'Nagel-Keratitis,' 19-20.
- Ascitic agar, 167, 174.
- Aspergillus fumigatus, 80.
- Aspirin, 54.
- Astigmatism, 108.
- Atropine, 17, 27, 28, 54, 57, 62, 75, 78, 83, 95, 108, 114, 120, 126, 129, 140, 161, 165.
- Atypical hypopyon keratitis, 65-85, 104-105.
- Avascular keratitis, 23.
- Axenfeld, Prof.—
 - Observations, 31, 66, 80, 81, 106, 116-17, 138 ; method of excising lacrymal sac, 176-81.
- BABES-ERNST bodies, 172.
- Bacilli found in corneal ulcers, 166, 170.
- Bacteria tabulated, 170.
- Bacteriolysis, process, 49.
- Bandages, employment of, 17, 21, 22, 25, 110, 120, 139, 151, 157, 162.
- Basso, Prof., 82.
- Blepharitis, acute, 100 ; marginal, 104, 107.
- Blepharo-conjunctivitis, *see* angular conjunctivitis.
- Blepharospasm, 131, 139, 140.
- Blood-serum cultures, 51, 70, 84, 106, 114, 167-70, 173.
- Bonn clinic, the, 16*n*, 110, 182.
- Boracic lotion, 16.
- Boric acid, 161.
- lanoline, 22.
- Bouillon culture, 59-60, 167, 172, 173.
- Bowman, Prof., 86.
- Bowman's membrane, 14, 15, 22.
 - Condition in pneumococcal ulcer, 38 ; in keratomycosis aspergillina, 84 ; in Mooren's ulcer, 92, 93 ; in diplobacillary ulcer, 105 ; in keratitis eczematosa, 134, 136, 137.
- Bullae, corneal, 20, 21, 23, 24.
- Bullous keratitis, 23.
- CALOMEL, 140.
- Canaliculi, the, 53, 62, 178.
- Candle-lamp, Priestly Smith's (Fig. 2), 2-3.

190 ULCERATION OF THE CORNEA

- Canthus, the inner, 62 ; the outer, 120, 126.
- Carbolic acid, 21, 22, 24, 52, 55, 57, 71, 78, 85, 95, 141, 165, 179.
- Carotid canal, the, 144.
- plexus of the sympathetic, 144, 145.
- Caruncle, the, 148.
- Caustics, 109.
- Cauterisation, 55-57 (Fig. 12).
- Cautery, use of the, 55, 56, 71, 85, 95, 96, 109, 141, 162, 185.
- Cavernous sinus, the, 145.
- Cervical glands, 130, 139.
- Chalazion, 76.
- Chemiotaxis, 50.
- Chemosis, 124, 160.
- Children—
- Marasmic ulcer in, 159-61 ; feeding of, 161.
- Chinosol, 121.
- Choroid, the, 42.
- Cilia, the, 9, 33, 42.
- Cocaine, use of, 4, 10, 11, 17, 22, 26, 55, 122, 131, 148, 165.
- Cocci found in corneal ulcers, 166, 170.
- Coli commune, bacterium, 66, 79, 126, 160, 167, 170.
- Collins, 24.
- Compresses, 54.
- Conjunctiva, the—
- Sensibility, 9 ; examination of, 10-11 ; condition in abrasions, 14 ; in pneumococcal ulcer, 33 ; in pyocyaneus ulcer, 72 ; in mycotic keratitis, 81 ; in Mooren's ulcer, 88 ; grafting of, 95, 97, 110, 141, 157 ; diphtheria of, 115, 121-23 ; condition in keratitis eczematosa, 136 ; in keratitis e lagophthalmo, 154 ; in keratomalacia, 158.
- Conjunctival sac, irrigation, 58, 120.
- Conjunctivitis—
- Acute, 10-11, 67-68, 77, 115-28.
- Chronic, 16.
- Duplex bacillus, from the, 98-99.
- Conjunctivitis—*continued.*
- Eczematosa, 129, 137.
- Gonorrhoeal, *see* Gonorrhœa of the Conjunctiva.
- Pneumococcal, 37, 47.
- Zur Nedden's marginal ulcer, 111-14.
- Coppez, Prof., 122.
- Cornea, the—
- Examination, 1-12 ; surface condition determined by light-reflex, 4-8 ; changes in substance, 8-9 (Fig. 6) ; sensibility, 9-10 ; condition in abrasions, 14 ; contusions, 18-19 ; foreign bodies in the, 24-26 ; condition in pneumococcal ulcer, 38, 42 ; in the pyocyaneus ulcer, 72-73 ; in keratomycosis aspergillina, 81-83 ; in Mooren's ulcer, 86-93 ; complications in bacillary ulceration, 100-132 ; condition in Zur Nedden's marginal ulcer, 111-12 ; in acute conjunctivitis, 119 ; in keratitis eczematosa, 132-34 ; in neuro-paralytic keratitis, 146-49 ; in keratitis e lagophthalmo, 150, 154, 156 ; in keratomalacia, 158.
- Cornea, staphyloma, 52.
- Corneal plastic operation, the, 183-84.
- Corrosive sublimate, 57, 58.
- Coryza, acute, 163, 164.
- Crista lacrymalis, 177, 178.
- Czermak, Prof., excision of the lacrymal sac, 176.
- DACRYOCYSTITIS, 67-68, 74, 76.
- Dendritic ulcer—
- Illustration, 164 ; origin, etc., 162-64 ; pathology, 164-65 ; treatment, 165.
- Descemetocoele, a, 97, 102, 110, 141.
- Descemet's membrane—
- Condition in pneumococcal ulcer, 40, 42, 43 ; in the streptococcal ulcer, 68-70 ; in pyocyaneus ulcer, 73 ; in

Descemet's Membrane—*continued*.

Mooren's ulcer, 91, 92 ; in deep infiltrated ulcer, 100, 102 ; in keratitis eczematosa, 137 ; in eczematous ulcer, 141 ; in keratomalacia, 158, 159.

Dionin, 17.

Diphtheria of the conjunctiva, 115, 121-23.

— Laryngeal, 123.

Diphtheriae, bacillus, 166, 168, 170, 171.

Diplobacillary conjunctivitis, 99.

— Ulcer—

Manner of infection, 98-103 ; comparison with pneumococcal ulcer, 103-104 ; pathology, 105-106 ; bacteriology, 106-107 ; treatment, 107-10 ; diagnosis, 150-51.

Diplococcus lanceolatus, the, 32, 174.

Diplopia, 148.

Douches, 120, 126, 139.

Duplex, the bacillus, 44, 98-107, 109, 137, 166-70.

ECTASIA CORNEAE, 161.

Ectropion, a cicatricial, 150, 153, 157.

Eczema, 100, 130.

Eczematosa keratitis, 51, 83, 101, 113.

Etiology, 129-36 ; pathology, 136-37 ; bacteriology, 137-38 ; treatment, 138-42.

Eczematosa, pannus, 133, 136.

Emmert, Prof., 162.

Endothelium, the, 8, 40, 42.

Enophthalmos, 148.

Enucleation, 52.

Epiphora after excision of the sac, 179-80.

Epithelium, the—

Condition determined by light-reflex, 4-8 ; loss of, 4-9, 20 ; effect of cocaine, 11, 17 ; condition in abrasions, 14, 15 ; in pneumococcal ulcer, 33, 38, 41, 42 ; in streptococcal

Epithelium, the—*continued*.

ulcer, 70 ; in keratomycosis aspergillina, 81-84 ; in Mooren's ulcer, 88-93 ; in acute conjunctivitis, 118-19 ; in keratitis eczematosa, 136 ; in neuroparalytic keratitis, 146-47 ; in marasmic ulcer, 158, 159 ; in dendritic ulcer, 162-64.

Erdman, Prof., 99.

Erichsen, 130.

Erosion, recurrent corneal, 10, 15*n*, 16, 17, 19, 24.

Symptoms and pathology, 20-21 ; treatment, 21-23.

Escharotics, 55, 96, 97.

Eyeball, protrusion, 154, 158.

FACETS, 7, 15, 17, 83, 101.

Facial paralysis, 143, 145, 148, 149, 153.

Fascicular keratitis, 129, 137.

— lesions, 145-46.

Filamentary keratitis, 16 ; description and treatment, 23-24.

Fluoresceine, 4, 11, 14, 20, 56, 90, 95, 163-65.

Focal illumination, use, 1-2, 8, 14, 26, 119.

Fomentations, hot, 54, 75, 78, 83, 85, 139, 161.

Forceps, fixation, 131 ; dissecting, 178.

Foreign bodies in the cornea—

Manner of occurrence, 24-26 ; symptoms, 26 ; treatment, 26-28.

Formaline, 57.

Fornix, the, 57, 120, 127, 132.

Fossa lacrymalis, 177, 178.

Friedländer's bacillus, 66, 166, 169, 170—

Ulceration due to, symptoms, 76-77 ; bacteriology, 77-78 ; treatment, 78-79.

Fuchs, Prof., observations of, 67, 73, 81, 129, 133, 134, 152.

GANGLION, the Gasserian, 144, 145.

Gelatine culture, 114.

192 ULCERATION OF THE CORNEA

- Gentian violet, 186, 187.
 Gentilini, Dr., 80.
 Glaucoma, 51, 105; acute, 52; secondary, 75, 79.
 Goitre, exophthalmic, 154; treatment, 157.
 Gonococcus, the, 123, 160, 166, 168, 170.
 Gonorrhœal conjunctivitis—
 Symptoms, etc., 115, 116, 123-26; diagnosis, 126; treatment, 126-28.
 Gourfein, Prof., 77.
 Graefe, Prof.—
 Excision of the lacrymal sac, 176; the Graefe knife, 60, 181-82.
 Gram's method of staining, 37, 70, 113, 167-75, 186-87.
 Grut, Prof. Hansen, 162.
 HÆMOGLOBIN* media, 168, 170.
 Hæmophiles, 170, 171.
 Hæmorrhage, 131.
 Hanke, Dr., 72, 74.
 Hartnack loupe, the, 3-4; illustration, fig. 3.
 Hay bacillus, 44, 66, 79.
 Hensen, Prof., 'Nagel-Keratitis,' 19-20.
 Herpes Corneae Febrilis, 163.
 — labialis, 164.
 Hertel, Prof., 67, 69.
 Histolysis, process, 49.
 Hoffmann, Prof. von—
 Bacillus of, 166, 172; excision of lacrymal sac, 176.
 Horner, 163.
 Hypertrophy, papillary, 81, 107-108.
 Hyphae, mycelial, 84.
 Hypopyon—
 Amount in pneumococcal ulcer, 35-37, 39, 42, 60-62; in streptococcal ulcer, 69; in Friedländer's ulcer, 77, 78; in the mycotic keratitis, 81-83; in Mooren's ulcer, 91; in the small, deep, infiltrated ulcer, 102; in bacillary ulceration, 109; in Zur Nedden's ulcer, 112; in gonorrhœa of Hypopyon—*continued.*
 the conjunctiva, 125; in keratitis eczematosa, 135; in keratitis e lagophthalmo, 155; removal of a, 43.
 — Keratitis, 29.
 IMMUNISATION, Römer's method, 59-60.
 Infiltrates, corneal, 59, 100-101; illustration, 33.
 Influenza, 145, 163.
 Influenzae, bacillus, 115*n*, 166, 168, 170, 171.
 Interstitial keratitis, *see* parenchymatous keratitis.
 Iodine, tincture of, 57, 96, 165.
 Iodoform, 57, 95.
 Iridectomy, the operation of, 62, 71, 75, 77-79, 97.
 Iris, the—
 Condition in pneumococcal ulcer, 33, 42; in streptococcal ulcer, 68-70; in keratomycosis aspergillina, 83; prolapse of, 35, 110, 134, 135, 137, 182.
 Iritis, 28, 35, 36, 62, 76, 77, 91, 102, 108, 112, 119, 155.
 KAYSER, cocci of, 166, 170.
 Keratalgia traumatica recidiva, 15*n*, 19-20.
 Keratomalacia—
 Diagnosis, 150-52; course, 158-61; treatment, 161-62.
 Keratomycosis aspergillina, 66.
 Symptoms, 79-83; illustrations, 81, 82; diagnosis, 84; pathology, 84; treatment, 84-85.
 Klebs-Loeffler, bacillus of, 166, 172.
 Knaebel, statistics, 160.
 Koch-Weeks bacillus, 137, 166, 168, 170, 171.
 — — conjunctivitis—
 Symptoms, etc., 115-19; treatment, 120-21.
 Kölle and Wasserman, 75.
 Krönlein operation, the, 158.
 Kuhnt-Völckers, excision of the lacrymal sac, 176.

- LACRYMAL apparatus, examination, 26, 55.
 — fossa, the, 53, 176-78.
 — sac —
 Blenorrhoea of the, 37, 55, 62-63, 104.
 Distension, 53-54.
 Excision, 16, 55, 63-65; Axenfeld, 176-81.
 Syringing, 78.
 Lacrymation, 13, 26, 36, 88, 101, 108, 131, 140, 149, 163.
 Lagophthalamo, keratitis e—
 Diagnosis, 150-53, 156; etiology, 153-54; symptoms, 154-55; pathology, 155-56; prognosis, 157; course and treatment, 157-58.
 Lagophthalmos, 105, 148, 153.
 Lang, excision of lacrymal sac, 176.
 Leber, Prof., 66, 80, 81, 138.
 Leeching, employment of, 78, 83, 139.
 Leucocytes, 40, 42.
 Leucocytosis, 49.
 Leucoma, 15, 60, 90, 91, 162.
 Lid margins, sensibility, 9; excoriation, 100, 132.
 Lids, oedema of the, 72, 73, 131, 160.
 Light-reflex—
 Variations in, determine condition of cornea, 4-8, 14, 26; appearance in the pneumococcal ulcer, 33, 34; in pyocyaneus ulcer, 73; in keratomycosis aspergillina, 81-83; in Mooren's ulcer, 88, 89; in keratitis eczematosa, 132; in neuroparalytic keratitis, 146.
 London, corneal affections in, 98.
 Lotions, Mr. Tweedy's, 142.
 Loupe, the corneal, 3-4, 8, 26, 27, 33, 88, 133, 147; the Hartnack, 3-4.
 Lungs, effect of pneumococci on the, 45-46.
 MARASMIC ulcer, *see* keratomalacia.
 Marginal conjunctivitis, 99.
 Marginal keratitis, superficial, 100.
 Martindale's Ung. Hydrarg Flav., 17, 14.
 Mastication, muscles of, 148.
 Matt surface reflex, the, 7.
 Meningitis, 145.
 Mercury, oxycyanide of, 57-8, 75, 114.
 — perchloride of, 95.
 Metchnikoff theory, the, 49.
 Mooren, Prof., 86.
 Mooren's ulcer—
 Symptoms, 86-91; pathology, 91-94; prognosis, 94; diagnosis, 94; treatment, 94-97.
 Moorfield's Hospital, 110.
 Morax-Axenfeld bacillus, 106, 166, 170.
 Motor paralysis, 148.
 Mouth-breathing, 130, 138.
 Mucocoele, 16, 37, 46, 51, 55, 63, 104.
 Müller, Prof.—
 Experiments, 138; bacillus of, 166, 168, 170, 171; speculum, use, 178.
 Mycotic keratitis, 80-81.
 'NAGEL-KERATITIS,' 19-20.
 Nasal duct—
 Strictures, 16, 37, 51, 55, 63, 180; condition in pneumococcal ulcer, 61-62; syringing of the, 78.
 Nebula, formation of a, 15.
 Needle, the lancet-pointed, 27; illustration, fig. 7.
 Neisser, Prof.—
 Cocci of, 166, 170; stain, 172-73.
 Nerves, the trophic, etc., 144-66, 148; the secretory, 149.
 Nettleship, 86, 89.
 Neuralgia, trigeminal, 144, 145.
 Neuroparalytic keratitis—
 Symptoms, 143-44, 146-49; etiology, 144-46; diagnosis, 149-51; treatment, 151-52.
 Nitric acid, 55.
 Nuclear lesions, 145, 146.

- OCULOMOTOR nerve, the, 145, 148.
 Ointments, 54, 107, 140, 141.
 Ophthalmia Neanatorum, 76, 126.
 Opsonic index, the, 59.
 Orbicularis, paralysis of the, 157.
 — Palpebrarum, 177.
 Otitis media, the, 146.
 Ozæna bacillus, the, 78.
- PALATE, arched, 130.
 Palpebral fissure, the, 153, 155, 157.
 — ligament, the, 176.
 Panophthalmitis, 51, 75, 119, 154, 155.
 Paquelin cautery, the, 55.
 Paracentesis, employment of, 43, 62, 97, 121.
 Paralysis, levator, 147.
 Parenchyma, the—
 Loss of, 9 (Fig. 6); condition in abrasions, 14, 15; in pneumococcal ulcer, 38, 39, 41; in Mooren's ulcer, 93.
 Parenchymatous keratitis, 22-23, 51, 88, 119, 129, 133, 135-36.
 Peltsohn, Prof., 160.
 Perforating ulcer, 129.
 Perforations, 31, 40, 43.
 Periostitis of the orbital margin, 153.
 Petit, Prof.—
 Observations, 106; bacillus, 166, 170.
 Phagocytes, 40.
 Phlyctenular keratitis, 100.
 Phlyctenule of the cornea, 83, 105, 111, 129, 137, 138.
 Photophobia, 13, 15, 26, 36, 88, 101, 108, 131, 139, 140, 162, 163.
 Phthisis, anterior, 31, 51.
 Plexus, the sympathetic, 148; the vascular, 177.
 Pneumobacillus, the, *see* Friedländer's bacillus.
 Pneumococcal conjunctivitis—
 Symptoms, etc., 115-19; treatment, 120-21.
 Pneumococcal ulcer—
 Prevalence of, 16; symptoms, 19, 29-37; pathology, 37-43; illustrations, 34, 39, 41; bacteriology, 43-50; diagnosis, 50-51; prognosis, 51-52; treatment, 52-62; prophylaxis, 62-64; comparison with diplobacillary ulcer, 103-104.
 Pneumococci, site of, in the cornea, 50 (Fig. 11).
 Pneumococcus, the, 43-46, 66-67, 126, 160, 166, 167, 170-75.
 Pneumococcus lanceolatus, the, 29, 32.
 Pneumonia, croupous, serum from, 44-45; acute, infection from, 57; formation of antiserum, 49.
 Pons, the, 144, 145.
 Potassium permanganate, 120, 126.
 Precipitate ointment, white, 140.
 Precipitates, 43.
 Proptosis, 154, 155.
 Protargol, use of, 16, 54, 55, 78, 107, 120, 128, 185.
 Pseudodiphtheriae bacillus, 166, 168, 170-172.
 Pseudogonococcus, 166, 168, 170.
 Pseudopterygium, formation of a, 157.
 Ptosis, 148.
 Puncta, lacrymalia, 10, 53.
 Punctata, pigmented keratitis, 50.
 Pupil, the—
 Examination, 11-12; condition in keratomycosis aspergillina, 83; in neuromyolytic keratitis, 148.
 Purulent keratitis, 22, 29, 30, 32.
 Pus, 35.
 Pyocyanus, bacillus, 44, 166, 168, 170.
 — ulceration due to—
 Symptoms, 72-74; bacteriology, 74-75; treatment, 75.
 Pyocyanin, 75.
 Pyorrhœa, 120.
 Pyramidon, use of, 54.
- RECKLINGHAUSEN'S lines, 40.
 Refraction, 108.
 Retina, the, 42.
 Rhine Valley, corneal affections in, 98.
 Rhinitis, chronic, 130.
 Rodent ulcer, *see* Mooren's ulcer.
 Römer, Prof., 32, 49, 51, 58-60, 63, 174, 175.

- Rosa Hefa, 44, 66, 79.
 Rostock, statistics in, 99.
- SAEMISCH, Prof., 80.—
Das Ulcus Serpens, 30-31; employment of Saemisch section, 60, 71, 75, 85, 162; method of operation, 181-83.
- Saline solutions, use, 12, 16, 57, 58, 152, 161.
- Sattler, Prof., 72, 74.
- Schirmer, Prof., 81.
- Schmidt, Prof., 117-18.
- Scissors, curved iris, 95.
- Sclera, the, 75, 135.
- Scrofula, 113.
- Scrofulosa, conjunctivitis, 137.
- Scrofulous ulcer, 129.
- Sensory paralyses, 148.
- Septicæmia, 77, 114.
- Serpens, Ulcus, 30, 31, 63.
- Serpiginous ulcer, chronic, *see* Mooren's ulcer.
- Serum—
 from acute pneumonia, 44-45; treatment, 49, 115-16, 121; Römer's use of, 58-60; from rabbits, 174, 175.
- Silver nitrate, use, 78, 108, 114, 127-128, 140.
- Smith, Priestly, candle-lamp, 2-3 (Fig. 2).
- Snail-track ulcer, 83, 129, 137.
- Sodium bicarbonate, 107.
- Spatula, use of a, 60, 182.
- Sperrer, the Axenfeld, 178.
- Spud—
 Illustration, 27 (Fig. 7); use, 11, 34, 165; Zur Nedden's, 184.
- Sputum, infection from, 46, 47.
- Staphylococcal conjunctivitis—
 Symptoms, 115-19; treatment, 120-21.
- Staphylococcus, the, 44, 79, 137, 156, 160, 166, 170.
 — ulcer, 66.
- Staphyloma, 31, 161.
- Stenosis, 16.
- Streptococcal conjunctivitis—
 Symptoms, 115-19; treatment, 120-21.
- Streptococcal ulcer—
 Cause, 67-69; pathology, 69-70; bacteriology, 70-71; treatment, 71.
- Streptococcus, the, 44, 48, 70, 71, 126, 137, 166, 167, 170, 171.
- Streptothriciae, *aspergillus fumigatus*, 167.
- Strumous ulcer, 129.
- Strychnine, injections of, 151.
- Sublimate ointment, 78.
- Subtilis, bacillus, 166, 168, 170.
- Subtrochlear nerve, the, 145.
- Syphilis, hereditary, 22-23, 136, 149, 151, 160, 161.
- Syringe, Anel's, 54, 107.
- TABES, 145.
- Tarsal ligament, the, 176, 177.
- Tarsal plate, 132.
- Tarsorrhaphy, method of performing, 157.
- Tendo oculi, 178, 179.
- Tenonitis, acute, 75.
- Tonsils, enlarged, 130.
- Toxines, action of, 73-75, 122.
- Trachoma, 51, 153.
- Trachomatosa, pannus, 136.
- Trigeminal lesion, 150.
- Trophic ulcer, *see* neuroparalytic keratitis.
- Tuberculosis, 130, 149, 151.
- Tweedy, Mr., 142.
- UHTOFF, Prof., 31, 66.
- Ulcère Rongéant, L', *see* Mooren's ulcer.
- Ulcus corneae, bacillus, 166, 170.
- Ulcus corneae serpens, 30-32, 44, 48.
 „ Rodens, *see* Mooren's ulcer.
- Ung. Hydrarg. Flav., 17, 141.
- Urethra, stricture in the, 180.
- VASELINE, boric, 17; sublimate, 57.
- Virulence, 'exaltation of,' 45.
- Vitreous, abscesses in the, 73, 74.
- 'WATERY' eye, 51, 63.
- Weichselbaum, 174.
- Weigert's stain, 84.

196 ULCERATION OF THE CORNEA

Wintersteiner, Prof., 156.

Wright, Dr., 138.

XEROFORM, use, 57, 78, 109, 121.

Xerosis of the conjunctiva, 152-153.

— the bacillus, 166, 168, 170.

Xerotica, keratitis, *see* lagophthalamo,
keratitis c.

ZINC, sulphate of, 107, 109, 114, 151.

Zur Nedden margin ulcer—

Symptoms, 110-13; illustration,
112; diagnosis, 113-14;
treatment, 114.

Zur Nedden, 110, 115*n*;—spud, 184;

—bacillus of, 78, 98, 166, 169,
170.

