is absurd, even then the figure only reaches 63 per cent. I believe that Römer's estimate, 25 per cent., is the correct one. It is interesting to note that in Palestine iritis is almost unknown, although pyorrhoea is very prevalent. I only remember seeing two or three cases among several thousand seen at the British Ophthalmic Hospital.

A FEW POINTS ON SOME COMMON AFFECTIONS OF THE EYES.*

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I PROPOSE to touch upon some affections of the eyes which every busy general practitioner is liable to meet fairly frequently, and will endeavour to give a few practical points that may be of some help.

We all know the difference between the injection of an eye in iritis and conjunctivitis. In iritis the injection is immediately around the cornea and is of a pinkish hue, whereas in conjunctivitis the hyperaemia is more marked towards the periphery of the bulbar conjunctiva. But it is not very uncommon to come across cases of conjunctivitis where there is, from the appearance of the eye, some doubt as to iritis being also present, and I am sure you will all have seen cases like the following:—

A patient with one or both eyes markedly red, with no diminution of the injection towards the margin of the cornea. We often find that the eye has been treated at home by keeping it tied up, or by poulticing it—perhaps with tea leaves. Such treatment, besides increasing the vascularity of the conjunctiva, may cause partial maceration of the corneal epithelium, and this, if one is not on one's guard, may be mistaken for turbidity of the aqueous, which is another sign of iritis. To proceed with the diagnosis, we next test the reaction of the pupil to light, and, if this is not satisfactory, inquire as to pain; stabbing pain in the eye, waking the patient about 2 or 3 a.m., is very characteristic of most cases of iritis. If still in doubt whether there is or is not some iritis present one next puts in a drop of $\frac{1}{2}$ per cent. atropin (provided glaucoma can be excluded), and if the pupil dilates well in half an hour, with no iris pigment or exudate on the anterior capsule of the lens, we may then conclude that there is no iritis. Giving such a patient atropin to use regularly at home, in addition to the correct treatment for the conjunctivitis, may in many retard the cure, and is only permissible when, after a thorough examination, there is still some doubt as to threatened iritis; in such a case it is better to order weak atropin drops ($\frac{1}{2}$ to 1 grain in the ounce) and see the patient in a day or two, when the eye may be found free from any suspicion of iritis, and the drops can then be discontinued.

The mistake usually made in treating conjunctivitis is in using strong astringents or strong antiseptics at the commencement; the condition is thus usually aggravated.

Boracic acid lotion, to wash out the conjunctival sac thoroughly, or even only sterilized water with some sodium chloride in it, will in many cases bring about the cure. In other cases astringents may later on be necessary, and as there are many of these to choose from, it is better to avoid the *lead lotions*, because, if there be an ulcer on the cornea, a white and disfiguring incrustation of lead forms at the site of the ulcer. This could only be removed afterwards with great difficulty. I have seen this occur on more than one occasion after a patient has had a lead and opium compress on the eye for a short time to relieve pain. These ulcers proved most obstinate to get to heal, required scraping several times, and were very painful. Attention to the patient's health and surroundings and getting him into the open air are extremely important, and may effect a cure without any applications to the eyes.

Unless advised not to do so, patients think they should stay indoors when they have what they call "cold in the eyes," and that they should even keep in a darkened room. There is no advantage in keeping a patient suffering from conjunctivitis in a darkened room. Dr. Berry points out that though the actinic rays

are known to cause irritation of the conjunctiva, which may indeed, as in snow blindness and electric light ophthalmia, be so great as to lead to violent inflammation, on the other hand they have a powerful bactericidal effect. The light of an ordinary room when the sun's rays are not directly passing into it has no had effect on conjunctivitis.

There is one form of inflammation of the lids, *angular conjunctivitis* (*Morax diplobacillus*), in which zinc salts are the most effective treatment; here the inflammation is of a subacute type, with the redness most marked at the angles of the lids.

Poulticing, or what comes to the same thing, tying an eye up (as the tears and discharge from the eye keep the bandage moist), is usually harmful; it increases the vascularity, and favours the growth of germs which are always present in the conjunctival sac. Nature by the act of blinking does much in the way of cleaning the sac.

If one eye alone be affected by conjunctivitis, be on your guard against the *tear sac* being the source of the trouble. In chronic blenorrhoea of the sac without marked distension the condition may easily be overlooked. Here it is the contents of the tear sac regurgitating through the puncta that keep up the irritation of the conjunctiva. Firm pressure over the sac with one finger will force some purulent matter through the canaliculi, and show the cause of the conjunctivitis.

Many early cases of inflammation of the tear sac, especially in young infants, can be cured by instructing the patients or the parents to empty the sac by firm pressure applied to it by the finger as frequently, it may be, as every half-hour. If the secretions are allowed to remain in the sac they act as an irritant to the mucous membrane, which becomes still more swollen, and so any stricture at the junction of the sac with the nasal duct is increased. Inflammation of the tear sac besides causing troublesome epiphora is a constant source of danger owing to the risk of infecting any corneal wound; a slight scratch of the cornea may in this way lead to a hypopyon ulcer, and perhaps to the ultimate loss of the eye. Similar danger to the cornea must be guarded against in ophthalmia neonatorum and in gonorrhoeal ophthalmia of adults; rough handling in examining such an eye may easily cause an abrasion of the cornea, and so allow its inoculation by the conjunctival discharge, which may result in a perforating ulcer and total loss of the eye. It is advisable, therefore, before examining such eyes to first instil a drop of cocaine inside the lids.

Crédé's method is an excellent prophylactic. I find the number of cases of ophthalmia neonatorum seen at the Eye Infirmary within the last few years has markedly decreased.

You may be called to see a patient complaining of pain in one eye, and on looking at it you find no conjunctivitis to speak of, but distinct circumcorneal injection. Assuming that the tension of the eye is not raised, and that glaucoma can therefore be excluded, you have to deal with either an affection of the iris (or deeper part of the eye) or of the cornea. It is not necessarily the iris that is affected; it may be a corneal ulcer, only to be discovered on minute examination. Many, I am afraid, at once jump to the conclusion that it must be iritis, and the patient thereby may be placed at a great disadvantage. A small corneal ulcer without any infiltrated edge may have to be searched for very carefully to be detected; the same remark applies to herpes of the cornea, or to the presence of a minute foreign body on the cornea, or to a slight abrasion of the cornea.

Here I wish to draw attention to the great advantage of using fluorescein (resorcin-phthalein anhydride) as a means of diagnosis in corneal affections. It may be used as drops (fluorescein 2 and sodium bicarbonate 3 in 100); one placed in the conjunctival sac, and then the excess washed out with, say, boracic lotion or sterilized water, will leave a brilliant green stain on any part of the cornea that has been denuded of epithelium, thus mapping out beautifully any corneal ulcer. A minute foreign body on the cornea would be more readily detected after using fluorescein, owing to the green ring which would appear round it, and it would therefore be much more easily removed. Fluorescein can be obtained in small tablets to place inside the lids, a more convenient form for carrying about.

* Read before the Newcastle-on-Tyne Division of the British Medical Association.

Another aid to diagnosis is oblique illumination combined with the use of a magnifying lens or corneal loup. By this method it is often possible to recognize signs of iritis, which otherwise might be difficult, for fine deposits on the anterior capsule of the lens can thus be detected. This method is extremely useful to see the punctate deposits on the posterior surface of the cornea, which are sometimes the only signs of cyclitis, and which may be so faint that it may be impossible to see them with the unaided eye. These deposits are due to inflammatory exudates from the ciliary processes, and, owing to gravitation, are usually situated on the lower part of the cornea.

Yellow oxide of mercury, or Pagenstecher's ointment, is extremely valuable in suitable cases, but if indiscriminately used it can do much harm. I am afraid that in some quarters it is almost looked upon as a panacea for diseases of the eye. The suitable cases for its use are phlyctenular conjunctivitis, strumous or superficial keratitis, blepharitis, and to aid in the clearing up of corneal opacities, when the eye is free from inflammation. There is a tendency to think that, if any ointment is to be used to the eyes, it must be the yellow oxide, and that if any drops are necessary they must be atropin drops.

In conclusion, I wish to insist on the importance of being able to tell whether the tension of an eye is raised. Take every opportunity of testing the tension; if in doubt as to what normal tension is, try your own eyes. Unless you are able to appreciate difference in tension you will fail to diagnose many cases of glaucoma. When house-surgeon, I saw admitted into an infirmary two spinster sisters almost completely blind through chronic glaucoma. Their medical attendant had diagnosed cataract, and had not sent them in till he thought the cataracts must be ready for extraction, as they came from a considerable distance. Nothing could be done at this late stage to avert complete blindness.

THE MECHANICAL ETIOLOGY OF OPTIC NEURITIS.

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OPTIC neuritis, or papilloedema,¹ to give it its more recent appellation, is the most interesting and at the same time the most puzzling symptom of intracranial disease.

The views held with regard to its causation are numerous, and we could do no better than quote F. W. Mott's summary of them:²

1. That it is due to inflammation or irritation of the optic nerve sheath, produced either by irritation arising from substances in the cerebro-spinal fluid of the sheath of the optic nerve or by direct extension of meningitis.

2. That it is due to obstruction to the outflow of venous blood from the ophthalmic vein by increased intracranial pressure on the cavernous sinus. But this theory of von Graefe has been disproved by the fact that a free anastomosis occurs between the ophthalmic and facial veins.

3. That the intracranial pressure interferes with the return of lymph from the sheath of the optic nerve.

Sir W. Gowers,³ for instance, favours the first view by attributing optic neuritis to the possible presence of toxic substances in the cerebro-spinal fluid; and Osler⁴ and Elschnig⁵ regard the presence of inflammation as essential to its production.

It is obvious that the above views are more or less conjectural, and are of little or no help in trying to elucidate the condition which causes this change in the optic nerve by so many morphologically, pathologically, and regionally different lesions.

We should be in a position to account for the presence of papilloedema, for instance, in connexion with minute growths, especially of the cerebellum; its conspicuous

rarity in cases of certain cerebral neoplasms of large size, notably of the corpus callosum; its comparative frequency in otitis media; and its inconstant character in cases of meningitis, abscess, and other inflammatory lesions.

Clinical evidence has gone far to prove that the toxic or inflammation theories are untenable. The fact that papilloedema is rare in post-basal meningitis, even in cases in which the inflammatory exudate has been found, *post mortem*, to have extended as far as the optic commissure,¹⁰ and its predominance in non-inflammatory conditions, such as tumour, chlorotic and other non-infective sinus-thrombosis, and cerebral aneurysm, constitute an uncontested negation to such hypotheses.

Recent clinical and experimental work, especially the researches of Sir Victor Horsley¹ have greatly helped to convert us to the belief that increased intracranial pressure is the principal mechanical agent in the production of papilloedema. In spite of the unanimity of most observers as to this point we cannot but agree with the opinion of Horsley and Swanzy¹ that the chief factor which produces this increased intracranial pressure has not been definitely ascertained as yet, and, furthermore, it cannot even be said that any successful theoretical attempt has been made to correlate, or to suggest an interpretation applicable to all cases of increased intracranial pressure with optic neuritis, from all causes, irrespective of their character, site, or dimension.

There can be no doubt that most clinicians have viewed this question from a purely pathological standpoint, and have looked for its answer solely amongst morbid causes. A survey of the work of Hill,¹² Cushing and Bordley,¹³ Mott,² and others, shows, however, that a correct solution of the problem must be based upon physiological findings, which may now be said to be sufficiently advanced to enable us to locate, so to speak, any derangement in the physiological mechanism which induces increased intracranial pressure, and to which we now propose to refer.

It has been conclusively established that the cerebro-spinal fluid is secreted by the choroid plexuses of the ventricles, and that the channels of its resorption are the superior cerebral veins which join the superior longitudinal sinus.¹⁴ The ventricles, then, may be looked upon as reservoirs for the constantly secreted cerebro-spinal fluid, while the subarachnoid space constitutes the main absorptive area. Each vein is surrounded by a sheath, which is an "intracerebral prolongation of the subarachnoid space" (Mott¹²), and the capillary interval between this perivascular lymphatic sheath and the vein wall is normally surrounded, to use Mott's words, by a cuff of cerebro-spinal fluid, and thus these veins are chiefly concerned with the withdrawal of fluid from the subarachnoid space.¹⁵

Our observations lead us to advance the contention that arrest of, or marked interference with, the cerebral venous return (with consequent subnormal cerebro-spinal fluid absorption) is the predominant factor in producing increased intracranial pressure, and that papilloedema is due to the resulting rise of fluid pressure in the optic nerve sheath. Given a condition of inhibition of the process of absorption, the pressure in the subarachnoid space will increase, either by continued secretion of cerebro-spinal fluid or by the diminution of the cerebro-cranial space.

We hold, therefore, that in every case of optic neuritis, from whatever cause, the patency of the veins is obliterated or the flow of their contents obstructed by blockage somewhere in the "superior longitudinal jugular line" (commonly the right), or they are rendered impervious by disease of the lymphatic sheaths. Thus, our view is diametrically opposed to that of Thomson Henderson,⁸ whose deductions are based upon the assumption that "the intracranial pressure represents the venous pressure" (Hill),¹² in all pathological conditions, and who asserts that whenever there is increased intracranial there is increased venous pressure. But in cases of increased intracranial pressure with choked disc, there being no corresponding rise in the intraocular pressure, he ascribes the oedema and tumefaction of the nerve head to the intravenous pressure of the retinal veins reaching a higher level than the intra-ocular pressure. To refute Mr. Henderson's argument, suffice it to quote a few lines from the review of his book in the BRITISH MEDICAL JOURNAL⁹: "Chronic irido-cyclitis often ends in a soft eye, it may be one like a pudding bag with no tension." In such a globe the tension is