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.

BY

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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

manifestly lower than the intravenous tension. If the Henderson theory were correct, we should have optic neuritis in all "soft" eyes!

Needless to say, the anatomy of the venous sinuses of the brain has an important bearing on this question. We have already seen that the superior longitudinal receives the superior cerebral veins, and is the principal carrier of cerebro-spinal fluid. The superior usually becomes the right lateral,¹⁴ which thus acquires greater importance than the left. The same remarks apply to the right and left jugulars. The other sinuses take part in the production of papilloedema in so far as they may bring about closure of what we may term the primary sinuses by consecutive thrombosis.

We may now consider the pathological conditions which give rise to optic neuritis, to see whether they present any features which are not compatible with our views, which we may summarize, for the sake of clearness, as follows.

Papilloedema results from the lessened or arrested absorption of fluid from the subarachnoid space, in consequence of:

- (a) Obliteration of the patency of the superior cerebral veins.
- (b) Disorganization of the perivascular lymphatic sheaths.
- (c) Occlusion of one or more of the venous sinuses.

Optic neuritis is found in association with (a) vascular, (b) organismal, and (c) neoplastic lesions.

(a) Vascular.

The explanation of the papilloedema from thrombosis of the superior longitudinal sinus, as in chlorosis, typhoid, marasmus, pneumonia, etc., is self-evident, being due to the fact that the main channel for the discharge of surplus cerebro-spinal fluid is occluded. The observation that the subsidence of the neuritis in chlorosis is due to canalization of the clot¹⁰ lends strong support to our theory. Blockage of the venous stream at any point, more so on the right side, may produce the same results, and the papilloedema of otitic origin is due to lateral sinus thrombosis, especially if the clot extends into the superior longitudinal. We venture to assert that even ligation of the right jugular in the neck may be capable of producing papilloedema if the emissary veins, especially the mastoid vein, are insufficient to establish compensatory venous return or are occluded by thrombosis or other cause.

(b) Organismal.

1. *Syphilis*.—The histology of cerebral syphilis constitutes one of the most important links in the chain of our arguments. The lymphatic sheaths have mainly been studied in this disease as they become thickened and conspicuous.³ This endolymphangitis is doubtless responsible for the impairment of the absorptive function of the superior cerebral veins.

2. *Meningitis*.—We have already stated that in some cases of extensive basic meningitis there is no optic neuritis. In all probability in these cases the lymphatic sheaths are not involved. Papilloedema, however, is bound to result whenever the inflammatory process seals the openings of these "intracerebral prolongations of the subarachnoid space." For obvious reasons "abscesses" will be considered with tumours.

(c) Neoplastic.

Tumours in the cortex or substance of the cerebrum act by compressing the brain tissue adjacent to them, and ultimately the veins. Obviously, a tumour of the cerebrum must attain a considerable size before it can press upon the veins. A point of some importance is the well-known fact that optic neuritis is not so common a sequel of abscess in the temporo-sphenoidal lobe as it is of abscess in the cerebellar region, for the simple reason that a much larger accumulation of pus or more extensive accompanying meningitis is needed for its production in the cerebral than in the cerebellar area. On the toxic or inflammatory theory, however, optic neuritis should be equally common in both varieties.

Tumours of the Cerebellum and Corpora Quadrigemina.

We may first direct attention to the following facts:

1. Optic neuritis is present in 100 per cent. of quadrigeminal, and 89 per cent. of cerebellar, tumours.⁷
2. The neuritis is generally equal and bilateral.*¹⁰

* Sir Victor Horsley holds that in its early stages the neuritis is always more pronounced on the side of the lesion.

3. Subtentorial abscesses are more frequently accompanied by optic neuritis than abscesses in the cerebrum.

4. A condition of internal hydrocephalus is present in connexion with cerebellar and quadrigeminal tumours with optic neuritis.

Growth or abscesses in the cerebellum or tumours of the corpora quadrigemina bring about compressional closure of the iter, which may also be closed by intraventricular tumours. The foramina of Majendie and Luschka may become occluded by lymph or scar stenosis (meningitis). The secretion of cerebro-spinal fluid being uninterrupted, the shutting-off of its natural outlet results in the production of internal hydrocephalus. The dilatation of the ventricles under considerable internal hydraulic pressure cannot but produce collapse of the delicate veins in the brain substance, and therefore cessation of cerebro-spinal fluid absorption. It may be argued that, there being no increase of fluid in the subarachnoid space owing to the non-patency of the iter or the foramen of Majendie, there should be no rise of pressure in the subarachnoid space, and therefore no neuritis. It must be borne in mind, however, that by the dilatation of the ventricles the size of the brain generally undergoes increase, and the cerebro-cranial space becoming diminished, the intervening fluid, however small in quantity, is bound to be subjected to pressure, and driven into the sheath of the optic nerve, and at the same time plays the rôle of an additional force in compressing the veins.

Mott describes a most instructive case² of a tumour of the choroid plexus which gave rise, intermittently, to all the symptoms pathognomonic of cerebral tumour, and which, according to Mott, seems to have acted like a ball valve in periodically blocking the iter. He attributes the intermittency to a condition of cortical *anaemia* resulting from the temporary internal hydrocephalus. The production of anaemia or ischaemia by internal hydrocephalus is taught, then, by so great an authority as Mott. We venture to differ from him, however, in so far as we believe that it is unlikely that the arteries are interfered with, and that the thin-walled minute veins with their low blood pressure are the vessels that undergo collapse, a view which also affords us an explanation for the drowsiness, vomiting, and other symptoms of intracranial disease, and has an important bearing upon operative measures.

In conclusion we may refer to a valuable paper by J. Stoddart Barr and John Rowan of Glasgow.⁴ We have already stated that papilloedema in connexion with ear disease is, in our opinion, evidence of sinus thrombosis, and we are supported by Grüning⁴ and Friedenber.⁴ Barr and Rowan found 6.8 per cent. of optic neuritis in cases of middle-ear disease that came under their observation, and they attribute it to the possible existence of "serous meningitis"—a vague statement, which does not harmonize with their declared belief in the mechanical origin of papilloedema. We have little difficulty in persuading ourselves that the cases they reported as otitic optic neuritis consequent upon serous meningitis were cases of lateral sinus thrombosis.

The following case, which presented many remarkable features, is a characteristic example of sinus thrombosis with optic neuritis.

H. W., aged 28, was admitted to the Ashton District Infirmary (sent in by Dr. Spencer of Ashton) on October 5th, 1910, suffering from acute mastoiditis. He gave a history of six years' discharge from the right ear. On admission his temperature was 99°, pulse 120. He had a well-marked retro-auricular abscess. He was operated on immediately. The complete mastoid operation was performed, but as the disease had spread into the cranial cavity, more bone was removed so as to get a thorough exposure of the lateral sinus. The latter was bathed in sanious pus, with a conspicuous erosion on its wall which bled profusely on entering the skull. The bleeding was rapidly arrested with gauze plugging. The patient's condition gave rise to anxiety, and we did not deem it advisable to carry out our original intention of tying the jugular vein. Accordingly the wound was plugged, and he was returned to bed. He progressed satisfactorily for the next three days. His temperature rose, however, on October 8th, and he had a rigor on October 11th. The wound looked perfectly clean. On October 12th there was some swelling and erythema on the right side of the neck. He was operated on again this day. The right jugular vein was exposed and found full of clot as far as the omo-hyoid. Its cardiac end was ligatured, and the cephalic segment dissected up to its junction with the common facial vein. The latter was ligatured, and the jugular itself, being emptied of its clot, was sutured to the upper angle of the wound,

which was closed without drainage. The wound healed per primam, and the vein, after discharging for three days, became completely obliterated five days after the operation. From the date of the jugular operation the patient had neither circulatory nor thermic disturbance, and seemed quite comfortable. On October 15th he complained of his sight being dim and of being unable to read his papers. His eyes were examined for the first time by us and by Mr. Wilfrid Lawson, ophthalmic surgeon to the hospital. He had intense double optic neuritis. We decided to relieve his pressure by lumbar puncture. At the first tapping his cerebro-spinal fluid pressure was 13 in. by Eve's instrument; 3iv were removed, and the pressure reduced to 1 in. He was thus tapped successively on November 2nd, 5th, and 10th, removing on each occasion 3vi, 3iv, and 3viii respectively, and leaving the pressure at 1 in. every time. The improvement after each aspiration was marked but transient, and so we decided to submit him to a subtemporal decompressive operation. On removing the disc of bone, the dura bulged out of the opening, but no pulsations were discernible. The dura was slit open by a crucial incision. A large amount of cerebro-spinal fluid escaped. On inspection the following were the striking features: There was an interval of about half an inch or more between the brain and the skull. The pial veins were prominent and distended. Even towards the close of the operation there was no tendency on the part of the brain to herniate or even to approach the skull. The tips of the four dural flaps were brought together, leaving four minute slits for drainage. The wound was closed in the usual way. Convalescence was uneventful. The patient's sight rapidly improved, and the papilloedema had entirely subsided on December 12th.

Peculiarly enough, we had the opportunity of watching another case presenting similar features, but involving the left mastoid, and, though the antrum, sinus, and jugular procedures were carried out, there was no optic neuritis.

With regard to the advisability of performing a decompressive operation in these cases Barr and Rowan disagree with Sir Victor Horsley. They state that, in their experience in these otitic cases, the optic neuritis clears up without operative interference. Their attitude is right in a sense, as compensatory venous return does get established in most instances by canalization of the clot or otherwise; but in severe types of papilloedema delay may cause irremediable damage to vision, while a decompressive operation—a minute opening is sufficient in these cases—is a precautionary measure the delay of which entails greater danger than its performance. In this connexion the words of a renowned surgeon (!), Oliver Cromwell,¹¹ are worth bearing in mind:

But as in quinsy or pleurisy where the humour fixeth in one part, give it scope; all diseases will gather to that place to the hazarding of the whole, and it is natural to do so till it destroy life in that person on whomsoever this befalls. So will these diseases take accidental causes of aggravation of their distemper, if a timely remedy were not applied.

We are indebted to Sir Victor Horsley for his kindness in reading our contribution, for the encouragement he has given us, and for drawing our attention to Schieck's work.

REFERENCES.

- ¹ Sir Victor Horsley, Optic Neuritis, BRITISH MEDICAL JOURNAL, March 5th, 1910. ² F. W. Mott, On the Cerebro-spinal Fluid (Oliver-Sharpey Lecture), LANCET, July 9th, 1910. ³ F. W. Mott, On the Cerebro-spinal Fluid, *ibid.*, July 16th, 1910. ⁴ Barr and Rowan, Optic Neuritis and Suppurative Otitis, BRITISH MEDICAL JOURNAL, March 26th, 1910. ⁵ F. W. Mott, Cerebral Tumours, Gibson's Textbook of Medicine. ⁶ Sir W. Gowers, Optic Neuritis, *ibid.* ⁷ Osler, Textbook of Medicine. ⁸ T. Henderson, Glaucoma. ⁹ Review of Glaucoma, BRITISH MEDICAL JOURNAL, January 7th, 1911. ¹⁰ Clifford Allbutt, System of Medicine. ¹¹ Oliver Cromwell, Letters and Speeches (Carlyle), Speech IV. ¹² Hill, Physiology and Pathology of the Cerebral Circulation. ¹³ Cushing and Bordley, Johns Hopkins Hospital Bulletin, 1909. ¹⁴ Cunningham, Practical Anatomy, 1907. ¹⁵ Schieck, Archiv für Ophthalmologie, Bd. 78, 1911.

THE CURATIVE EFFECT OF SALVARSAN SERUM IN A CASE OF SYPHILIS.

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HAVING read the interesting article of Dr. Henry Alston in the issue of the BRITISH MEDICAL JOURNAL for February 18th, we applied this treatment to a patient suffering from syphilis in the Lock Hospital, Harrow Road.

It will be remembered that Dr. Alston produced a blister on patients who were suffering from framboesia and were under the influence of salvarsan. He then injected the serum obtained into other patients suffering from framboesia, with excellent results.

5

The history of the case in which we adopted the same method is as follows:

X. Y., aged 19 years, was admitted into hospital on February 8th, 1911, suffering from syphilis.

1. *Genitalia*.—The vulva and anal regions were covered with large vegetating condylomata and mucous tubercles discharging a watery fluid.

2. *Skin*.—Universal areas of raised papular, somewhat scaly, syphilide, the face being also involved; syphilitic alopecia was present.

3. *Glands* of groin and neck discrete, soft, moderately large.

4. *Mouth and Throat*.—Fauces normal; general stomatitis; tongue normal.

The patient was also suffering from scabies.

Date of infection about July, 1910.

Weight 6 st. 2½ lb.

Wassermann + +.

February 14th. The patient's general condition being very feeble she was ordered sulphur baths and inunction of mercury. Three baths and one inunction only were administered.

February 19th. The patient weighed 5st. 8 lb., having lost 8½ lb. The scabies was better but the syphilitic signs were worse. The patient was injected with 10 c.cm. of a serum obtained from a cantharides blister on the abdomen of another syphilitic patient who four days previously had had an intravenous injection of 0.4 gram of salvarsan.

February 24th. Genitalia better, condylomata and mucous tubercles drier. Rash clearing up. Mouth practically well.

February 25th. 15 c.cm. of similar serum were given. After the third bath a good deal of sulphur irritation was noticed; baths were therefore discontinued. Mucous tubercles were now present on the tongue. Weight 5 st. 11½ lb.

March 3rd. 18 c.cm. of a similar serum were administered. Genitalia much better, condylomata flatter and drier. The patient was feeling much better in health.

March 8th. 25 c.cm. of a similar injection from a patient seven days after an injection of salvarsan. Weight 5st. 13 lb.

March 12th. 10 c.cm. of a similar injection.

March 13th. Genitalia much drier, condylomata much flatter, mucous tubercles gone. Rash fading fast, being flat and showing no hyperaemia. Mouth, fauces, and tongue quite well. Glands as before. Weight 6 st. 1½ lb.

Wassermann + ± March 7th.

REMARKS.

The noteworthy features of this case are:

1. The marked improvement in general health and increase in weight as soon as the serum treatment was begun. Previously the patient had lost 8½ lb. during the eleven days she was in hospital in spite of being carefully nursed and tended.

2. The effect on the Wassermann reaction is most striking; a deviation of 15 per cent. (+ +) of complement is reduced to a partial deviation of 15 per cent. of complement (+ ±) in sixteen days; this compares very favourably with any known treatment.

3. In our experience of some 30 or 40 cases of intravenous injection of salvarsan the results of the serum treatment are found as good as those of the drug, while the treatment produces no signs of illness.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

POST-ANAESTHETIC ACID INTOXICATION.

WITH reference to the two cases of post-anaesthetic acid intoxication reported by Dr. Gilbert Brown, I should like to point out that the administration of sodium bicarbonate in large doses, although ideal theoretically, practically never appears to have the slightest effect, or, if any, in the wrong direction.

There is, however, a remedy, tincture of iodine, which appears to quiet the stomach, and, as I have thought, to lessen also the smell of acetone in the breath. I believe that, if it had not been for its use, I should have lost a little patient who had been operated upon for tuberculous glands, and who vomited for four days, gradually getting worse, but who rapidly improved after the administration of single drop doses of tincture of iodine in a tablespoonful of water. I can well remember the smell of acetone in his breath.

Whilst resident in different hospitals I saw several mild cases, but owing to the early administration of this simple remedy I never saw any severe symptoms follow the prolonged administration of anaesthetics.

Not very long ago a rather stout virgin, from whom I removed some caruncles, suffered from severe vomiting, acid breath, etc., on the third day. The first administration