BRAIN AND EYE—OPTIC NEURITIS.

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IT is now six years since the classical discussion on Optic Neuritis in Intracranial Disease took place during the first session of the Ophthalmological Society. Much work, clinical, microscopic and experimental, has since then been done in elucidation of the subject; but it cannot be regarded as much less of a *questio vexata* now than it was six years ago. Another more purely ophthalmological question has also been under discussion during these years, viz. Sympathetic Ophthalmitis. Notwithstanding important differences, the two questions have much in common, and it seems probable that when the one has been solved, the other will present but little difficulty. While in both cases the ultimate decision must rest on clinical observation, in both cases experimental observation can do much in clearing the ground, and in suggesting at least a *vera causa*. To Deutschmann, of Göttingen, we are indebted for experimental work on both subjects, and we purpose giving some account of his recent monograph on "Optic Neuritis in connection with Brain Disease,"¹ with some reference to the work of others, confirmatory and opposing.

Observers are now, and indeed they were six years ago, practically unanimous in regarding optic neuritis as a single process, not distinguishable into "choked disc" and "descending neuritis," as was formerly made out. It is clear on reading various descriptions of what were considered the distinguishing features of "choked disc," that there was no general agreement on the point. The term implied a theory, but expressed no clinical fact, and it has simplified matters that it is now practically obsolete. At the same time it would be very far from right to conclude that there are not types of neuritis. The neuritis of tubercular meningitis, for example, is as a rule of quite a different type from that of cerebellar tumour. The low, pinkish-white swelling of the first is very

¹ *Jena*, Gustav Fischer, 1887, p. 68. For full Abstract v. 'Ophthal. Review,' April, 1887.

distinct from the high, blood-coloured and generally blood-stained swelling of the other. Both however, are almost certainly but different intensities of a similar process, and probably the main element in the difference is the "choking," which, as shown by Gowers in the above-mentioned discussion, does actually take place, not at, but in front of, the lamina cribrosa.

In his paper, opening the discussion on Optic Neuritis, Dr. Hughlings-Jackson referred to three hypotheses regarding the cause of the inflammation: Von Graefe's, Schmidt's, and the reflex vaso-motor theory of Benedict, Brown-Séquard and others. The last hypothesis he considered, and the writer believes he still considers, the most plausible explanation, although he in no way commits himself to it. There are many difficulties in the way of accepting it. Reflex vaso-motor irritation, will, we presume, be readily admitted by all pathologists; but very few, we imagine, would admit that this irritation can proceed to actual inflammation, exudation of lymph, and hæmorrhage, such as occurs in tumour neuritis. The only instance at all parallel with it is that of Sympathetic Ophthalmitis, and the work of Leber, Deutschmann and others has made it highly probable that there is in every such case an infective process by way of the chiasma. The fact mentioned by Dr. Jackson, that tumours may induce discharges in neighbouring grey matter, goes only to show that tumours may cause an instability of grey matter, either by immediate pressure, or by interference with vascular supply, whether directly or reflexly. Von Graefe's theory attributes the neuritis to compression of the cavernous sinus by the tumour, the consequences of this compression appearing specially at the lamina cribrosa from its rigidity. The theory is negated by the fact, that the ophthalmic and the angular veins communicate so freely that even obliteration of the cavernous sinus will cause no venous stasis in the eyeball or orbit.

Schmidt's theory attributes the neuritis to the pressure of fluid effused in the subdural and subarachnoid spaces, and forced into the vaginal spaces of the optic nerve; and Schultén has recently shown, that the injection of fluids into the subdural and subarachnoid spaces will actually cause narrowing of the retinal arteries and distension of the retinal veins, the changes however, being temporary. Deutschmann has subjected this theory to further experimentation in order to settle the question, what degree of hydrops of the optic nerve in animals is required to produce changes in the disc similar to those of the choked disc in man. With antiseptic precautions he laid bare the optic nerve, divided it, injected the distal part of it with agar-agar solution, and ligatured it,

taking care not to injure the central vessels. With moderate distension, greater however than that produced by cerebral tumour, there was only the temporary vascular change observed by Schultén, while in order to produce swelling and œdema of the papilla, the nerve sheath required to be so forcibly distended as to arrest the circulation in the retina, and even then the nerve showed no true neuritis. Such distension and arrest of circulation is never present in the human subject. Again, repeated injections of agar-agar solution into the cranial cavity of an animal, although they caused considerable intracranial pressure and distension of the optic sheath, in no instance produced inflammation of the papilla, the nerve or its sheaths. These experiments seem certainly to decide against Schmidt's theory.

In 1881 Leber put forward the view expressed in the Transactions of the Medical Congress of that year:¹ "The optic nerve is the path of communication between the affection of the brain and that of the eye. An essential part in this transmission is taken by the effusion of a serous fluid into the sheath of the nerve extruded from the cranium by the increased intracranial pressure. This fluid does not act by simple mechanical pressure, since its quantity is sometimes small; probably it possesses phlogogenic properties." To test this theory, Deutschmann repeated his injection experiments, using an infecting material in place of the agar-agar solution. A solution of common salt containing traces of staphylococcus was injected, in the manner above described, within the optic sheath. During the first day the papilla became red and swollen, the veins distended, the arteries perhaps diminished in size. Next day the papilla was greatly swollen, and the retina turbid; and the microscope showed well-marked neuritis and peri-neuritis. Injections of the same solutions into the skull caused acute inflammatory changes, resulting in death without the eyes being affected, and it was necessary, therefore, to select an infective material of slower action. A few drops of tubercular pus were introduced, with antiseptic precautions, into the subdural space. The animal remained apparently healthy for three weeks, when the papilla became red, and the veins tortuous. True papillitis followed, which either gradually subsided, or went on to post-neuritic atrophy. The microscope showed tubercular meningitis, the intracranial portion of the nerve normal, the orbital portion more or less altered, the sheath being distended more or less with exudation in the early stages, and occupied with tubercle deposits in the later; the nerve and its sheath being both inflamed. Deutschmann considers that this proves

¹ 'Abstracts,' p. 474.

Leber's theory as to the cause of neuritis, and he suggests that the phlogogenic elements contained in the fluid secreted in the subdural and subarachnoid spaces are micro-organisms already present in the system, but congregated round the tumour as a place of least resistance. Deutschmann considers that a true descending neuritis is rarely demonstrable, and when a continuous neuritis can be traced, he considers that it is generally an ascending, rather than a descending, neuritis; that the bulbar end of the nerve is affected first, and from this point it travels backward. Where only one eye is affected in intracranial tumour, Deutschmann suggests that there is in the one nerve-sheath some unusual obstruction, either recent, or of older date, preventing the passage of the infective material.

It will at once be seen that the evidence afforded by the injections of tubercular matter into the subdural space is not absolutely cogent, and Edmunds and Lawford point out that,¹ in order to substantiate his point, Deutschmann would require to show that if the animals be killed in the first few hours or days after inoculation, no optic neuritis is present; that at a later stage inflammation confined to the bulbar end of the nerve is found, and that later still the whole length of the nerve is involved. Edmunds and Lawford¹ have recorded the results of their examination of the optic nerves in cases of intracranial disease, and an analysis of cases of intracranial tumour with respect to the existence of optic neuritis. Having examined some forty cases, they found that in every case in which papillitis was seen during life there were inflammatory changes in the optic nerves in their entirety. In a few instances the inflammation was confined to the proximal part of the nerve, and in these the papilla, both to the ophthalmoscope and microscope, showed no changes, death having occurred before the inflammation had time to travel down the nerves as far as the papillæ. They conclude therefore that the optic neuritis which occurs in intracranial disease is due to the presence of a secondary meningitis, and they find themselves strengthened in this opinion by the result of their analysis of cases. Of ninety-six cases of fatal cerebral tumour, optic neuritis was present in eighty-six per cent. of the cases in which the new growth was situated in the basal ganglia or in the cerebellum, whereas in only forty-six per cent. of those cases where the tumour was at the convexity of the brain did neuritis occur. In the discussion on optic neuritis, Gowers stated that it was probable that mere distension of the sheath

¹ 'Ophth. Rev.' May, 1887.

¹ 'Ophth. Soc. Trans.' vol. iii. p. 138, and vol. v. p. 172.

might cause papillitis, but he showed by specimens that descending inflammation was almost invariably present, even where there was no naked eye evidence of it. One of his specimens is interesting in relation to Leber's theory. It showed, as usual, inflammation along the length of the optic nerve, but much more intense at the chiasma and behind the globe. From it he argued that a very slight degree of descending inflammation may be the connection between a neuritis of the cerebral, and one of the ocular extremity of the nerve. The optic papilla, as he remarked, is a structure in which inflammation readily occurs, whether from intracranial or from general disease. And it is to be remembered that the papilla may be repeatedly and severely inflamed. The writer has recorded a case of relapsing neuritis¹ where a patient recovered with perfect vision from two separate and severe attacks of neuritis. Since the case was recorded the patient has had a third attack similar to the others, and has again recovered with perfect vision. Such a case is somewhat difficult of explanation on Leber's theory alone; but Leber's theory does not actually contradict the theory of a descending neuritis, or peri-neuritis, as advocated by Edmunds and Lawford. It is possible, perhaps it is probable, that both views are correct. each cause playing a part, more or less, in different cases.

¹ 'Ophth. Rev.' May, 1886.