Clinical Cases.

A CASE OF DOUBLE OPTIC NEURITIS, WITHOUT GROSS CEREBRAL LESION, WITH REMARKS UPON THE IMMEDIATE CAUSATION OF OPTIC NEURITIS. (*Illustrated.*)

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THE following case is of such interest that I hope I shall be pardoned for the detail with which I have narrated it. The patient had evidently been for a long time on the border-land of insanity, and the acute maniacal delirium which terminated the case may be taken to indicate that she had passed beyond the border. As in so many cases in which the mental faculties are at fault, no coarse anatomical changes were found in the brain beyond wasting of the organ. Microscopic examination, however, showed changes which fully accounted for the symptoms presented. The long period over which the mental symptoms extended would point to the brain being primarily at fault. Atrophy of the brain such as she had is sometimes found in arterio-capillary fibrosis. No such condition was, however, present in her case. Her body was well nourished, the kidneys healthy, the left ventricle not hypertrophied. In cases of atrophied brain and cord in connection with arterio-capillary fibrosis, similar exudative changes have been found by Sir William Gull and Dr. Sutton, and it is noteworthy that the end of such cases is frequently ushered in with acute symptoms referable to the brain and spinal cord. Indeed this seems to be in accordance with a broad pathological law that tissues in a state of decay or of impaired nutritive activity are prone to become inflamed.

The symptoms presented at one time resembled hysteria, and the attacks were thought by some to be hysterical. In this respect the case recalls the valuable one recorded by Dr. A. Hughes Bennett, in the first number of 'BRAIN.' In his case there was a tumour; in the present there were marked,

though minute, structural changes in the brain. In many of the attacks the patient had spasm of the pharyngeal and laryngeal muscles, reminding one of hydrophobia. The nurses of the ward, who have seen three or four cases of hydrophobia in the hospital, were struck by the resemblance. The case is as follows:

Marie Schwabe, a Frenchwoman, a native of Paris, aged 27. When I saw her, in September 1878, she was a woman of somewhat short stature, inclined to be stout; dark hair, almost black, and with long hairs growing from upper lip and chin; teeth good. Her expression was that of a woman who had suffered a great deal—an expression of anxiety and despair. Patient's father and mother living; the former had been afflicted with hemiplegia for some years. She has two sisters and one brother, living and healthy; no history of phthisis, epilepsy, or insanity in family. Her previous history was obtained chiefly from her husband, an intelligent clerk.

She had good health and manifested no mental peculiarity, as far as is known, up to the time of her marriage, at 18. During the whole of her married life, however, she used constantly to express to her husband a dread that she would go mad; and "she used to dream almost every night of mountains, serpents, lions, and other animals, with the heads of people she knew." She used also to complain of a sense of suffocation if she was in a room with the door shut. Her memory was impaired for ordinary affairs, but was unnaturally acute in recalling anything bad of those she knew. Was not pregnant until three years after her marriage. When five months advanced in pregnancy she had a very bad miscarriage, without assignable cause. She had been complaining of obscure pains over the region of the uterus for some fourteen days previously. She was attended by a doctor, but lost a good deal of blood. The following day, on her husband coming home in the afternoon, she complained that the nurse whom he had engaged to look after her had been drinking brandy, and was drunk. (This was quite imaginary.) She then, in a wild manner, said several times, "You give her coffee." She became extremely excited, sprang up in bed, tore down her clothes, which were hanging on a nail by her bedside, and said, "I want to go away—I want to go away." She then had a kind of fit, similar to those she had subsequently in the hospital—repeatedly opening and shutting her mouth, stoppage of respiration, turning pale, violent struggling, and "became insensible." The attack lasted about three or four minutes: she did not bite her tongue. She gradually recovered her senses, and remembered nothing of what had occurred. A few hours after this she had a laughing fit, and repeatedly said to her husband, "You

laugh with me!" Next day she had "convulsions" from time to time, and a peculiar twitching of the muscles of her face. This twitching lasted continuously for one week, and since that time to her death she had this twitching, if excited. She improved a good deal for three weeks after this, when she again became nervous; and one day, on her husband opening the door, said she believed that the members of the club to which he belonged would poison her, and expressed a great dread of everybody she knew.

Menstruation had been irregular since her miscarriage; she had excessive flow every two or three weeks. After her miscarriage she walked with difficulty, and vomited after her food. She consulted a doctor, who placed a pessary in position, with temporary relief. She went into the country for a few weeks, but at the end of that time became nervous again, "lost the use of her limbs," and suffered constantly from severe headache. She says sometimes the pain was so severe she could not think; she used to say stupid things which she afterwards remembered. About nine months before her death she began to take a great dislike to people whom she saw for the first time, avoided persons her husband brought home to supper, and complained to him that he forced their society upon her. She took to eating the tops of lucifer matches (a favourite continental method of committing suicide), and her husband had to remove them forcibly from her. The least noise, or any one speaking to her, made her very nervous. She says that now she is not able to control her feelings, and burst out crying if refused anything she asks for, and this distresses her a good deal. For three months before admission took narcotics at bedtime, but even then has not had proper sleep, always being restless, and having disturbing dreams, seeing strange figures, &c., which have caused her to wake suddenly, and become very agitated. In one of her dreams she thought she was eating her own brain. For three days previous to admission she had suffered so much from headache that she could not be left alone, and she vomited after everything she took. The headache had been far greater than previously the six weeks preceding admission. Throughout her illness she was troubled a good deal by constipation.

The patient had had an operation performed on her right eye (for strabismus?) in childhood. For six months preceding admission she had been complaining of weakness of her eyes, especially the right, but ever since her husband has known her, she has had to put her hand over her right eye on reading or writing. There is no evidence to show that her left eye had been failing previous to admission, beyond the fact that on the day of admission she had been previously to see some

friends, and on entering the room had complained that she could not see any one.

A week after her miscarriage a Hodge's pessary was introduced, and she wore it for some months until it slipped out during defæcation. She then had it replaced, and wore it until the end of last March, when she was told the womb was in place. A few weeks later, when straining at stool, she felt the womb fall. The pessary was again introduced, and she wore it until a few days before admission, when she took it out herself.

She applied at the obstetric department of the London Hospital, on account of her uterine symptoms and of increasing pain in the head, and was admitted under the care of Dr. Herman, August 28th, 1878.

She then complained of great pain in left ovarian region, and down left leg and side, pain in the head, and dizziness. On vaginal examination, the uterus was found to be retroflexed, movable, and the fundus large and exceedingly tender. There was an erosion on each lip of the cervix, with a copious glairy discharge from cervical canal; the cervix was blue and congested. The cervix was scarified two or three times, and a mixture prescribed consisting of sulphate of magnesia and bromide of potassium. The uterine congestion having subsided, a Hodge's pessary was fixed, and the womb found to be well-retained in position.

Her other symptoms, however, increased; the headache was very severe, and she frequently vomited—sometimes clear fluid, sometimes green fluid. I was therefore asked to see her on Sept. 22nd.

I found her exceedingly nervous, starting whenever any one approached her, and looking extremely frightened. She complained very much of her head, and implored relief. Physical examination of her chest and abdomen revealed no important changes. Her tongue was coated with dark brown fur; bowels much constipated. There was no œdema of feet.

Ophthalmoscopic examination of left eye showed swelling of the optic nerve, the margin of the disc being wholly lost to sight. Owing to patient's restlessness, no detailed examination could be made. The right eye could not be examined with the ophthalmoscope, owing to old iritic adhesions.

Her vision was not tested by types, but the following facts indicate its condition. On her admission (about a month before I saw her, and before the neuritis was discovered) she wrote to her husband: this letter was a long one, and written in a small ordinary hand. Her letters gradually became shorter, and the type larger and larger. In three weeks she could not see to write at all, and got the patient in the next

bed to write for her; from time to time she asked her husband to write to her in larger type, and complained even then. She had to hold his letters quite close to her eyes. She could see quite well previous to admission: she could use her right eye for near objects (to read), but not for distant objects.

Her temperature since the beginning of September has averaged nearly 100° Fahr., the maximum being 101°, the minimum 98·4°: the evening temperature being usually higher than the morning. Urine, s. g. 1026. Urea 3·3 per cent. A very faint trace of albumen, a little epithelium only in deposit.

She was transferred to my care.

Sept. 30th, 12.30.—She screamed out, was purple in the face, and tried to tear her pocket-handkerchief. When seen by the medical officer, she was quite quiet, but not unconscious. When spoken to, answered questions quite rationally. She asked to have her husband sent for. At times she cried out; at others made a peculiar yelping noise. When I came to her bedside in the afternoon, she was lying in bed quite quietly, with a very anxious expression of countenance. She said she felt comfortable, but was very anxious that something should be done to relieve her head. Whilst talking to her she was seized with spasm of the laryngeal and pharyngeal muscles; her mouth would be suddenly opened two or three times in succession; she made a gasping noise, and occasionally a sudden inspiration, accompanied by spasm of the larynx, which produced a yelping or barking noise. The spasms appeared occasionally like exaggerated hiccough. Some of the noises she made occurred during expiration. She said "Give me some water," and when a glass of water was placed in her hands, she could drink, swallowing without any great difficulty, but her agitation and tremor made it difficult for her to hold the glass to her lips. She seemed very anxious about herself, and said inquiringly, "I am not dying?" She frequently asked for water, and each time drank some. The spasms continued for about a quarter of an hour, as long as these notes were being made. She complained of a burning sensation, "like a fire," in her chest. She occasionally put her handkerchief into her mouth and bit it.

The following mixture was ordered to be taken every 3 hours:

℞ Potassii Bromid
Chloralis Hydratis ʒ gr. x.
Acid. Hydrocyanic. dil. ℥. iij.
Aquam ad ʒj.

8.30 P.M.—She complains during intervals of quiet that the bowels have not moved; she still has burning pain over

epigastrium, and severe headache. Her teeth are chattering (temp. 99.7°). She keeps twisting her pocket-handkerchief between her fingers. She has had a little quiet sleep since taking her medicine, but on waking up the spasm of the pharyngeal and laryngeal muscles returned. She can talk quite sensibly during the intervals. On giving her some water she drank it readily, but spasm followed immediately. She caught at the bedclothes, and made a peculiar rasping or barking noise, and then threw herself back in bed. She says the cause of her attack was seeing a big woman with a shawl over her head, walking up and down in front of her bed last evening, and that the woman struck her head with something black. She has not vomited to-day. Pulse 96, small.

2 A.M.—House Physician called to her bedside. He found her yelping, and on giving her something to drink, spasm immediately followed. After an interval of quiet, she began laughing, and continued to do so for about five minutes. She said, "You don't laugh; you see I do!" This was followed by a fit of crying.

Oct. 1st.—She has had about four hours of quiet sleep in all, but each time on waking up immediately began to make a yelping and barking noise. During intervals of quiet she protrudes her tongue when asked to do so. She says the pain in her head is still very bad, and she adds, "but I have more reason this morning." She complains of something which she says is grasping her throat with two hands, and also of severe burning pain over epigastrium.

9 P.M.—After $\frac{1}{4}$ grain of morphia hypodermically, patient was sleeping quietly, and afterwards passed a good night.

Oct. 2nd.—On coming to her bed this morning she was quiet, but immediately after being spoken to began yelping. This continued for about five minutes, and was followed by a fit of laughing, during which she threw herself about in bed. She appears afraid lest she should be going to die, and continually asks if she shall get better. She has not vomited since yesterday; bowels were open last night after an enema. Passes her urine freely. The mixture was stopped until this morning. She still complains of severe headache. Is able to drink without any spasm being produced, but a short time afterwards made the same noises as previously.

Oct. 3rd.—Slept until one o'clock this morning, when on waking, began laughing and crying. On her husband persuading her to try and sleep, she became composed, and passed a good night. Has had no more vomiting. Drank a pint of milk, with only a little spasm. A hypodermic injection of morphia, gr. $\frac{1}{4}$, was given at 11 A.M. This morning, on coming

to her bedside, she immediately had a fit of laughing, during which she beat the bed with both hands. This was followed by shrieking out at the top of her voice, and then she said, "Boo, boo." Still complains of headache.

Oct. 4th.—Passed a quiet night; has not vomited, and says headache is better. She was lying quite quiet, when, on another patient in the ward laughing, she immediately broke out laughing, screaming at the top of her voice, beating the bed with both hands. Has taken some milk. Passed her urine this morning.

When I saw her in the afternoon, I dictated, "patient was always pale, but now her face is white; there are dark halos under her orbits. The left eyelids are swollen and red. Altogether she looks extremely haggard, and as though she had had but little sleep for some time. She is still very much excited, tosses herself about in bed, and frequently strikes the bed with both hands a number of times in succession, and with great force. Her tongue is coated; there is slight sordes on her lips. The bowels have not acted for three or four days. Left pupil contracted, and an ophthalmoscopic examination impracticable."

Oct. 5th.—Had an enema last night, after which her bowels acted twice. Had a quiet night until 4 A.M. The pessary she was wearing was removed last night, since then she says she has been easier. On coming to her bed this morning she immediately began to shriek out, to beat the bed with her hands, and then to throw herself back in bed. It is very difficult to keep her in bed, and she struggles violently when any attempts are made to restrain her. Has passed water. She keeps crying out about "the naughty woman," and says "she was very naughty to make me so ill." This was followed by a fit of laughing. Has not vomited.

Evening: was quiet on coming to her bedside, but immediately began laughing immoderately.

Oct. 6th.—Passed a quiet night after an injection of morphia, but is in much the same condition this morning; first laughing, and then crying. She requests every one who comes near her to laugh, and then does so herself; she afterwards bursts out screaming as loud as she can, becomes very violent, and at times it is a difficult matter to keep her in bed. No vomiting. It became necessary to place her in a separate ward, as her incessant noise disturbed the other patients. She passed a large quantity of urine yesterday afternoon; has taken a fair amount of nourishment.

Oct. 7th.—Slept quietly until 2 A.M., when she became very noisy, laughing and crying. She is very irritable. Passed her urine twice yesterday, and her bowels have been freely opened.

Oct. 8th.—Passed a quiet night: no pain in head this morning. Has passed her urine; bowels open in the night. She has been much quieter this morning, but called her husband “a fish.” She has not screamed so much this morning. Says she is losing her reason. No vomiting; takes nourishment very well.

Oct. 9th.—Passed a quiet night. Is more tranquil this morning. Has a haggard, worn look. Has not vomited. Has taken a basin of arrowroot this morning. Both upper eyelids are now swollen. Drinks without any spasm being produced. Tongue coated. Passes urine very freely, but in varying positions.

Oct. 11th.—Passed a quiet night. No albumen in urine: no vomiting. Says she has no pain in the head. Tongue thickly coated. Passes urine freely. Her expression is still more haggard.

Oct. 14th.—Passed a very restless night, in spite of morphia and bromide of potassium. Bowels confined: passes urine freely. Eyelids less cedematous. Complains less of pain in head. Says she is better. She does not scream this morning, but makes a groaning noise. Will not protrude her tongue when asked to do so. Takes nourishment.

Oct. 16th.—Passed a restless night. Her husband (who is almost constantly with her) says she is able to swallow, but after having done so makes a gurgling noise, and then anything she has taken returns. She has a worn-out look. Her pulse is small, 96. She does not appear to have strength to scream. Has passed her urine and motion in the bed. No paralysis of limbs. Does not complain of pain in the head, but still complains of pain over epigastrium. Recognises her husband and the nurse.

Oct. 17th.—Passed a quiet night. Has taken a fair amount of nourishment. When asked if she is better, says “Yes.” She looks exceedingly ill; her pulse is small; feet and hands cold. She is very restless. Passes both urine and faeces in bed. Has been screaming more this morning.

Oct. 18th.—Passed a restless night. Has been screaming all night. Bowels moved freely yesterday. Has taken a fair amount of nourishment.

Oct. 19th.—Passed a quiet night, except at intervals. She is restless in bed this morning. On being asked if she has any pain, she does not speak, but shakes her head. Passes her urine in the bed. Takes nourishment.

Oct. 21st.—The patient passed a fairly quiet night. Nurse reports that several times, without any cause, her breathing stopped, and she turned purple in the face. After a few seconds there was a gurgling in the throat, and breathing

recommenced by her taking a long inspiration. This was observed several times (Cheyne-Stokes respiration). She does not answer when spoken to. Passes her urine in bed: bowels not open for several days. No paralysis of any of her limbs. Pulse 84, small and feeble. She cannot be roused. Swallows with difficulty. Feet and hands and surface of body cold. She is getting rapidly worse.

2 P.M.—Patient evidently sinking.

Died early in evening.

Her temperature averaged about 100° Fahr., the maximum being 101·6°, from Sept. 22 to Oct. 6. After this date she was too restless to admit of it being taken. On the two occasions her urine was examined, its specific gravity was 1026 and 1025 respectively, and contained 3·3 and 3·6 per cent. of urea. The quantity of urine secreted could not be measured. On the first occasion a very faint trace of albumen was present, on the second none.

The treatment consisted of large doses of potassium bromide, with and without chloral, of subcutaneous injections of morphia, of aperients as required, and the frequent administration of easily digested food.

The *necropsy* was made on the 22nd, at 3.30 P.M., by Dr. Sutton, in my presence. The following is Dr. Sutton's report, extracted from the Post-mortem Book:—

"Atrophied brain. Fatty deposition on right ventricle of heart. Much subcutaneous fat.

"Largely developed body, with large regular features. Limbs seemingly somewhat above the average bulk. No œdema. The subcutaneous fat over thorax and abdomen was about one inch thick.

"Skull and dura mater normal. Pia mater milky-looking and semi-opaque. The brain convolutions were much atrophied over anterior two-thirds of the hemispheres. The sulci were much widened. The grey matter of brain on section looks normal, merely venously congested, and the brain substance on section looked normal throughout. The vessels at the base were seemingly normal.

"The heart weighed 8½ oz.: it looked smaller than normal. There was much fat on the right ventricle near the apex: it had grown into the wall of the right ventricle, leaving scarcely any visible muscle: muscle more natural above. The left side of heart was natural, excepting that the wall looked thin.

"Lungs œdematous, and hypostatic pneumonia at back part of one.

"Liver and spleen normal. Kidneys normal. The mucous membrane of stomach was studded with a number of yellowish

masses, the size of pins' heads; they looked like stomach tubes distended with fat."

Dr. Sutton appends the following note to the case:—

"There was no evidence to show that the atrophy in this case was part of a general atrophic process. The post-mortem examination would leave it uncertain as to whether the fatty accumulation and degeneration occurred antecedently, rendering the right ventricle unable to empty itself properly—leading to venous distension of the pia mater and venules of the convolutions, and by pressure of these distended veins, or otherwise, producing the atrophy of convolutions, more or less dementia, and the subsequent obesity."

Microscopic Examination of optic nerves and retina, and of brain.—The right optic nerve was removed for the purpose of examination. This eye had not been seen during life owing to iritic adhesions. It shows increase of nuclei of trabeculæ and nerve fibres, and considerable collection of lymphoid cells in trabeculæ. This cell infiltration extends throughout the whole length of nerve examined (a considerable length), but the cells are most aggregated below the lamina cribrosa. There is very considerable infiltration of the outer part of the internal sheath of optic nerve with round cells (lymphoid cells or leucocytes), and the fenestrated membrane has great increase of nuclei owing to this germination. Nerve fibres of optic disc swollen, and lower layers infiltrated with cells continuous with those in trabeculæ of optic nerve. Slight infiltration of ganglionic layer with lymphoid cells. Both granule layers disturbed in regularity and arrangement. Retinal blood-vessels distended with blood corpuscles. Slight infiltration of choroid at optic entrance, at the part immediately contiguous to the cell infiltration of sheath. Arteria centralis has its adventitia very slightly thickened.

Pia mater slightly infiltrated, showing some chronic change.

Brain cortex infiltrated with lymphoid cells (leucocytes), especially in second layer (small pyramidal cell layers). The lymphoid cells can be seen following the tracks of the vessels, and have obviously exuded from them: they are especially in connection with venules, slightly with capillaries. Pyramidal cells so altered as to retain no longer the shape from which they derive their name: they are represented by a collection of three, four, or more small nuclei, and some ill-defined protoplasm. In the first and second layers of cortex are some ill-defined spherical masses, staining purple with hæmatoxylin: they are apparently masses of exudation (plasma or lymph). Nuclei of blood-vessels in places germinating. Several parts of the cerebral cortex were examined and the same changes

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Section through deeper part of Frontal Convolution showing longitudinal sections of small vessels and branches, filled with coloured and colourless corpuscles, with emigrant colourless corpuscles (leucocytes) collected around the vessels. The exuded corpuscles are represented dark as stained by logwood.

found at each. Similar changes in corpus striatum. Throughout brain-blood-vessels much gorged.

REMARKS.—The existence of double optic neuritis without coarse local cerebral disease is a point not only of interest but of considerable clinical importance. Optic neuritis is so commonly symptomatic of some adventitious intracranial product, or of some gross intracranial lesion, that a diagnosis often turns on this one point. That it is not always indicative of coarse intracranial disease cases reported by other observers show. Dr. Hughlings-Jackson, in particular, has drawn attention to this point. In reporting "A Case of Double Optic Neuritis without Cerebral Tumour,"¹ he quotes from a Lecture on Optic Neuritis, delivered in 1871.²

"I have been wrong several times in the diagnosis of an adventitious product within the skull, in cases where there had been found double optic neuritis; *but I have far oftener been wrong by neglecting the inferences above stated to be deducible from the presence, or absence, of optic neuritis*—wrong in saying there *was* an adventitious product when the discs were normal, and wrong in saying there *was not* when there was double optic neuritis. I feel, therefore, justified in saying that double optic neuritis does point *very strongly indeed* to coarse disease inside the head [*italics in original*].

In the case I have narrated I had the benefit of Dr. Hughlings-Jackson's opinion. He kindly took charge of the patient during a short absence, and wrote to tell me on my return that he did not expect "local coarse disease" would be found in the brain after death.

But why should this patient get optic neuritis, the commonly ascribed causes being absent? This raises the question, Why does optic neuritis occur in connection with its usually acknowledged causes—tumours, abscess, &c.?

I think that in spite of the eminence and ability of its advocates, from V. Graefe downwards, the pressure theory of swelling of the optic nerves, whether called choked disc or neuritis, cannot be maintained. We see large encephalic tumours, sometimes of rapid growth, without any neuritis; and we see small tumours in distant parts of the cranial cavity—as in the cerebellum—with power to provoke the neuritis. Neither is there any ante- or post-mortem evidence, other than that of the optic nerves themselves, to bear out the theory of a Stauung's papilla. The inflammation of the optic nerves, which we see with the mirror during life and with the microscope after death, appears to me to be excited in all cases by either an extension process from the membranes of the brain

¹ The 'Royal Ophth. Hosp. Rep.' Vol. VIII, part iii. p. 445.

² 'Mod. Times and Gaz.,' Sept. 16, 1871, p. 341.

to the optic sheath, and thence to the nerve and retina, from the brain to the optic tract or nerves directly, or by an independent affection of brain and optic nerve by a common cause acting by selection upon histologically allied structures. Of course the inflammatory changes in the optic nerves and discs are modified by the anatomical peculiarities of the part. Vascular dilatation, and serous and corpuscular exudation, though constituent factors in the process of inflammation, do not necessarily advance *pari passu*. In some cases the dilatation of vessels predominates (influenced no doubt by the condition of the vascular system of the patient at the time of the disease inducing the neuritis), whilst in others exudative changes are in excess of vascular dilatation. Hence arise differences in the appearances of the optic nerves, to which many observers attach importance. It is acknowledged, however, by most that these differences have no practical significance as indicating the nature, the position, or the duration of the intracranial lesion on which they depend, and we may therefore conveniently speak of the swelling and exudative changes of the optic nerves under the title of neuritis. To repeat, the neuritis associated with intra-cranial disease is, in my opinion, an inflammation communicated from the membranes, or from the brain itself to the optic nerves, except in the rare cases where they are independent, but due to a common cause. In some cases the implication of the membranes, especially basal, affords a ready explanation of the occurrence of the neuritis. Where the meningitis (tubercular, zymotic, traumatic, or an extension process from abscess, &c.) appears the sole encephalic lesion, this explanation may suffice without bringing in another; but at the same time it must be remembered the *symptoms* of meningitis are due to the changes wrought in the brain, and which are never absent; and so it must be borne in mind that a possibility exists that the neuritis accompanying meningitis may really be an extension process from the implicated brain. Excluding cases of primary meningitis, a secondary meningeal inflammation cannot be brought in to explain the occurrence of neuritis from tumours, as the evidence of such meningitis is frequently wholly wanting. We are therefore thrown back upon some change communicated from the brain tissue to the optic nerves, to explain the neuritis due to coarse cerebral disease, and my impression is that the inflammatory changes found in the optic nerves are in all cases (excepting cases of meningitis) either an extension process from the brain, that is to say, "a descending neuritis," or a common process shared in by the brain and optic nerves. I say my *impression*, because I am not prepared to speak dogmatically on the point without further evidence. I am engaged just now in investi-

gating this subject from the histological stand-point. I may, meanwhile, draw attention to some facts which appear to favour this view. In the first place a tumour in the brain as such cannot give rise to symptoms, except by exciting changes in the nervous tissues surrounding it. Where we have permanent paralysis, there must be destruction of nerve tissue; where there is spasm, pain, vomiting, &c., there must be disturbed nutritive activity associated with tissue changes. Whether the tissue changes around tumours are or are not to be called inflammatory is a question of terminology. There are found vascular dilatation, corpuscular infiltration, thickening of neuroglia, &c., the changes which we should regard as interstitial inflammation in any other organ than the brain. So I take it the changes found around tumours, &c., in the brain are of an inflammatory character, tending to spread in all directions or in certain directions, and in so doing frequently implicate the optic nerves, which really are modified portions of the brain outside the general cranial cavity. Of course I must not be understood to deny that a large and rapidly growing tumour will produce pressure symptoms. My object is to show that pressure cannot be the usual explanation of neuritis from local coarse intra-cranial disease.

The direction the cerebritis takes and the rate of its progression are matters of great moment which require further investigation. Whilst it would at first sight seem probable that inflammatory changes would spread in all directions around the nucleus formed by a tumour, yet it is more probable that the cerebritis spreads most in certain directions—directions of least resistance, of more direct anatomical continuity, &c. We see this in rapid changes as hæmorrhage, and in slow changes as sclerosis. A strong point in favour of the cerebritis which I assume excites optic neuritis, having a tendency to travel in special directions, is the fact that in two cases of unocular neuritis from cerebral tumour recorded by Dr. Hughlings-Jackson,¹ the neuritis was on the side opposite to the tumour. On any pressure or vaso-motor theory, the neuritis should have been on the same side as the tumour, and the same may be said of the neuritis being set up by intermediation of meningitis. But the inflammation travelling down the optic tracts would explain the occurrence. Such cases of unocular neuritis are rare, and it would be very valuable if those who have had the opportunity of seeing any completed by a post-mortem examination would place them on record.

If the cerebritis spread in various directions, we should expect the implication of other cranial nerves. It is the expe-

¹ 'A Physician's Notes on Ophthalmology,' Dr. J. Hughlings-Jackson, 'Reprints from Ophth. Hosp. Reports,' &c.

rience of most observers that, whereas amaurosis is common from coarse cerebral disease, deafness is rare, whilst defects of smell and taste, being much less important, are passed over with little notice. As regards nervous deafness, Dr. Hughlings-Jackson has especially remarked upon its scarcely ever occurring in connection with coarse cerebral disease. But it must be remembered that he has (as he says, he fears, in season and out of season) repeatedly called attention to the existence of double optic neuritis without defect of sight; and so it may be that, less open to our examination, inflammatory changes occur in the auditory and other cranial nerves without being detected. It is significant that of the only two cases Dr. Hughlings-Jackson has recorded (so far as I know), in one case he expressly states that he could not account for there being deafness of both sides, seeing there was only disease (tumour) on one side; in the other case he only states (in reprint) that the patient was blind and deaf, and that at the autopsy a tumour was found in one sphenoidal fossa, reaching as far back as the seventh pair of nerves, producing changes in the hemisphere which had reached the auditory nerve. The other cranial nerves, as the third, fourth, sixth, &c., are sometimes paralysed without any explanation of the causation, such as meningitis, &c. It may be in such cases the inflammatory process has reached them from the brain, setting up neuritis.

We know little concerning the circumstances which influence *the rate* at which the cerebritis spreads from the vicinity of tumours. The difficulties underlying this point are very great. We only know of the existence of intracranial tumours at all from the changes excited in the brain-substance around them. We know that optic neuritis may be one of the earliest signs of a tumour, and at the same time we know a tumour may exist for a long time without exciting neuritis. I have watched a patient of Dr. Hughlings-Jackson's, in whom a cerebral tumour was subsequently found, who had symptoms (unilateral convulsions beginning in one great toe) indicating its existence and precise locality several years before neuritis occurred. Nearness to the optic tracts is probably one circumstance which influences the period of occurrence of optic neuritis. Cases of cerebral tumours without neuritis are probably cases in which the cerebritis has not reached the optic nerves.

The present state of our knowledge, then, I hold is not inconsistent with the neuritis occurring in connection with coarse cerebral disease being due to an extension of the inflammation to the optic nerves from the inflamed brain around the encephalic lesion causing the neuritis by direct continuity of tissue, though I am aware how deficient is the

direct evidence of such causation. What is wanted is careful microscopical examination of continuity of cerebral tissue from the tumour to the optic discs in many cases with and without neuritis. I am collecting material for such examination. I would, however, refer the reader to Dr. Clifford Allbutt's work, 'The Ophthalmoscope in Diseases of the Nervous System,' where the subject is discussed with such completeness and ability. Dr. Allbutt, however, draws a wide distinction, both as regards the appearances and causation, between the choked disc and descending neuritis, and believes the latter only to occur by propagation from the membranes or cerebral tissue.

But how are we to explain the cases where optic neuritis occurs without its usual exciting causes? I think the microscopical examination of the brain in my case affords some solution. It cannot have failed to have attracted attention of observers that, apart from neuritis, the changes which occur in the optic nerve and retina are alike in kind with those co-existing in the brain. We see this in Bright's disease, in diabetes, in leuchæmia, in idiopathic anæmia, in ague, in purpura, &c.

In the above case the changes I have described and depicted in the brain are of a kind which in any other organ would be regarded as inflammatory. Fashion is largely responsible for our terminology. Similar exudative changes in the liver or kidney, in the skin, the stomach, or the optic nerves, would undoubtedly receive the name of inflammatory, and we should speak of interstitial hepatitis or nephritis, or of dermatitis, interstitial gastritis, optic neuritis. So, I take it, the changes found in the brain in the above case show a condition of cerebritis or encephalitis. Though to the naked eye there appeared only atrophy of the brain, the microscope showed a corpuscular infiltration of an inflammatory character. But it may be objected, How could the change be inflammatory when the brain was atrophied, seeing that inflammation causes swelling (tumour)? I need only instance interstitial nephritis and hepatitis as examples in which inflammation and shrinking go hand in hand together. The changes need not be exactly contemporaneous throughout the brain; whilst exudation is taking place at some parts, other parts previously affected may be undergoing contraction. The microscopical changes in the above case in the brain and optic nerve are precisely of the same character. We saw with the ophthalmoscope in the eye during life what we could only see in the brain with the microscope after death. As to whether the changes in the optic nerves were sequential to, or contemporaneous with, the changes in the brain, there is no precise evidence to show, though the longer duration of

cerebral symptoms and the shrinking of the brain would incline me to believe that the optic neuritis was secondary—an extension-process from the brain. In Dr. Hughlings-Jackson's patient, to which I have referred, there was no wasting of brain or gross-naked eye change. Dr. Sutton examined the brain microscopically, and reported that sections of the convolutions of anterior lobe showed a large number of nuclei, about the size of white blood-cells, some appeared to be dividing. In places they were collected into groups of from ten to twenty, but they were not especially numerous around the capillaries. Dr. Hughlings-Jackson has since had another case of neuritis without coarse cerebral lesion, which he hopes shortly to place on record.

I think the view I have been advancing—the similarity of the changes in the optic nerves and brain—is of much practical importance, for we can see the optic nerves during life and, I submit, infer similar changes to what we find there in the brain. If this correspondence holds good, the ophthalmoscope may help us to correctly interpret many cases. Thus, in some cases of lead-poisoning we find optic neuritis. When such patients die, all the naked eye can discover is either atrophy of brain or no marked alterations. The test-tube tells us that the brain is saturated with lead. But neither the naked eye nor chemistry tell us what changes have gone on in the brain producing the headache, the delirium, the convulsions, and the coma, which have preceded death. Moreover, we know that under appropriate treatment the optic neuritis and nervous symptoms may coincidentally disappear, as in a case which I hope shortly to report in 'BRAIN.' I believe that the neuritis in such cases throws a light upon the character of the cerebral lesion, that the neuritis we see indicates a simultaneous and like affection of the brain, the neuritis being an extension process from the cerebritis, or the two like nervous structures being similarly affected by a common cause—the lead circulating in the blood. I believe that cerebritis will be revealed in such cases by microscopical examination. If these speculations are correct, it shows the extreme importance of the routine use of the ophthalmoscope, apart from any defect of sight, or even sign of nervous disorder. The eye has been happily called the window of the brain. By looking through it with the ophthalmoscope we may often see exactly what is taking place within the cranium, when without this window we should remain in ignorance.