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OBSERVATIONS

ON

DEFECTS OF SIGHT IN BRAIN DISEASE,

AND

OPHTHALMOSCOPIC EXAMINATION DURING SLEEP,

BY

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OBSERVATIONS ON DEFECTS OF SIGHT IN BRAIN DISEASE.

I WISH to say, first of all, that in writing this paper, I write as a physician, and not as an ophthalmologist. I have studied ophthalmic medicine merely as a help to the study of diseases of the Nervous System. I look at the fundus of the eye, in cerebral cases, when there is even slight failure of sight, in order to ascertain the calibre of the retinal vessels, and the supply of blood to the optic discs, as evidenced by their greater or less coloration. For instance, after a fit of epilepsy, there is often severe headache attended by giddiness, singing in the ears, and other symptoms referrible to disorder of the circulation, and, generally, trifling defect of sight. I trust that by a careful study of the circulation, both venous and arterial, in the eye, in cases like this, I may learn something as to the condition of the circulation of the brain itself in cerebral disease.

The above is put forward as some excuse for my venturing to address ophthalmologists on subjects on which they must necessarily have had more special experience than I have.

I intend chiefly to give a few particulars as to the causation of amaurosis in cases of tumours of the brain. The cases have already appeared in the Medical Times and Gazette for August 30, 1862. I then reported, from various hospitals, cases of diseases of the cerebellum, in many of which there was blindness.

Blindness not uncommonly occurs in cases of tumour of the brain. Sometimes the optic nerves are compressed, and here the blindness is easily accounted for. Sometimes also one optic nerve only, on the cerebral side of the commissure, is

affected, and then we get half-blindness of each eye. In many cases, however, it is somewhat difficult to account for the production of the amaurosis. In the Medical Times and Gazette for January 25, 1862, is recorded a case of echinococcus in one hemisphere of the brain. The patient, a girl, thirteen years of age, was in St. Thomas's Hospital, under the care of Dr. Risdon Bennett. In the report of the case, it is stated that she could neither walk nor stand, but that "when lying in bed, she could move the legs freely." There was no loss of sensation anywhere. There was complete blindness, and by the ophthalmoscope, the optic discs were seen to be white and atrophied. There was pain also, mostly at the vertex; but, at the first, the pain was at the occiput, where she had had a blow. Tumour of the brain was diagnosed, and "from the peculiar impairment of locomotive power, it was thought probable that the tumour was in the cerebellum." She had also epileptiform seizures. This case, during life, was certainly very like many cases of disease of the cerebellum, and yet at the autopsy, the cerebellum was quite healthy, and the optic tracts were, to the naked eye, quite unaltered.

The blindness in cases of tumour of the brain, not directly affecting the optic tracts, may be produced by the general pressure, or by reflex irritation, or because, in some way, the tumour interferes mechanically with the blood-vessels, so that parts at a distance do not receive their due supply of blood. It may be that the nutrition of the optic tract is interfered with because the nerves supplying its blood-vessels arise or pass in the parts injured.

In the case of echinococcus, just related, and also in a case (under the care of Dr. Gull, in Guy's Hospital), of cancerous tumour of the cerebellum pressing on the corpora quadrigemina, the optic discs were found, on ophthalmoscopic examination to be quite white (white atrophy), the rest of the fundus being healthy. This condition of the optic discs is what is found in nearly all cases of amaurosis in cerebral disease. In some, however, there appears to be not so much a defect of circulation, as an

obstruction, the optic papillæ being swollen, ill-defined, and blood being diffused near them, or in other parts of the fundus, as if the vessels had burst from over-distension. A well-marked case of this kind was shown to me by Mr. Bader. He considered that there was probably a tumour of the brain, and that, by pressure, it mechanically obstructed the circulation.

In the ordinary white atrophy, the atrophy may be due to some eccentric irritation contracting the blood-vessels, and thus diminishing the supply of blood; so that wasting of the nerve follows, as wasting of the muscles does in infantile paralysis, which, also, is generally supposed to be due to some eccentric irritation contracting the vessels. The fact that sometimes, though in few cases, and then only at the beginning of the disease, the sight varies remarkably, tends to favour this idea; but at the same time it is well known that in *organic* disease of the brain, intermission of symptoms is not very uncommon. It may be that it is an instance of atrophy of the nerve following loss of its function, just as it atrophies when the eyeball is lost. In the case of amaurosis above-mentioned, in which there was supposed to be mechanical obstruction to the vessels, the optic discs became after a time quite white, although their edges were ill-defined, and there were still remains of the apoplectic effusions.

That amaurosis does often occur in connexion with disease of the cerebellum, and also with disease of the spinal cord, has long been noticed. To explain how it occurs is not easy. It seems certain that the cerebellum is not the origin of the optic nerve fibres, as in Combette's case of congenital absence of the cerebellum (quoted by Mr. Solly in his work on the Brain), all the senses were perfect. "The child could see, hear, and taste in a perfect manner."

Dr. Brown-Séquard thinks that the defect of sight "is a result of an irritation of certain parts of the cerebellum acting upon the nutrition of some parts of the nervous apparatus of vision." He says also:—"It is not usually on account of a pressure upon the corpora quadrigemina that amaurosis exists

in cases of disease of the cerebellum, as we find that loss of sight is sometimes observed in one eye only, and that is the eye on the side where exists the alteration in the cerebellum; while, if it were owing to a pressure on the tubercula quadrigemina, the loss of sight would be on the opposite side." In those cases of disease of the cerebellum to which I have alluded, in which there was defect of sight, both eyes were affected. In the case of cancer of the cerebellum, no doubt there was, towards the end at all events, some interference with the corpora quadrigemina. The tumour was large, and when the venæ Galeni were pressed on, causing the effusion of serum and the "dropsy" of the brain, the corpora quadrigemina must also have been interfered with. In other cases, as in the case related by Andral (p. 18), and also in cases of amaurosis with paraplegia, pressure could have nothing to do with it.

A remarkable feature, however, in one case was, that the sight varied remarkably. Just as the man had had epilepsy and loss of function of the brain, so we might say he had temporary epilepsy of the retinæ (or optic nerves). In epilepsy the loss of consciousness is believed by Brown-Séquard to be coetaneous with contraction of the blood-vessels of the brain. It is attended by paleness of the face, and probably also, if I may use such expressions, paleness of the retinæ and paleness of the brain. It would be very desirable to examine the retina during a paroxysm of epilepsy. We are not, however, often present when a patient has a fit, or we arrive, as generally happens, just too late, or, when in time, from the struggling of the patient, the examination is impracticable.

In one case, however, a case of "epileptiform convulsions," I had the opportunity of examining the fundus of the eye, if not during a genuine fit, at least during a condition in which the consciousness was lost, and in which the pupils, ordinarily small, were dilated as if under the influence of atropine. The optic discs were extremely pale. Once, the vessels disappeared altogether for an appreciable time.

After a while, however, they reappeared, and were found to vary with the respiration. When the patient *in*-spired the vessels disappeared, returning again on expiration, like lines of red ink on white paper. This examination was too hurried, and the results too indefinite, to make it an observation of any great value. The case, too, was not one of genuine epilepsy, but one in which there was cancer of the sphenoid and secondary cancer of the glands in the neck. The fits occurred either as a result of obstructed circulation, or from pressure on the nerves of the neck. It is mentioned here merely to hint that it may be possible to study the cerebral circulation, by examining the vessels in the eye. I intend to examine the fundus of the eye in an epileptic guinea-pig, as a fit can be induced when we are ready for the examination.

In a few cases of epilepsy there is complete blindness for an appreciable time before the paroxysm—the patient is conscious, and yet in total darkness. And I have noticed that, in many cases of convulsions, in which one side of the body, only, is involved, there is temporary failure of sight before the full epileptic paroxysm. Cases in which there is failure of sight from temporary loss of accommodation are common enough, especially when the eyes are hypermetropic. In these cases the patients say that for a time they "cannot see;" but it is easily ascertainable that they can see, but that they see nothing distinctly—nothing in definition.

A person, whose eyes are hypermetropic, cannot see clearly, in the distance even, unless the ciliary muscle is *acting*. Failing power of this muscle will therefore cause confusion of sight. The patient cannot see well because the rays are not brought to a focus on the retina—the retina itself being sound. Hence defect of sight in hypermetropia is often significant of debility only. It is due, then, in many cases, to the debility so often attending the brain disease, and not directly to a fault of the nervous system. Whilst the patient is in health he can accommodate, in spite of the flatness of his eyeball; but when he gets out of health he is unable to do it. In many cases these distinctions are of

consequence in diagnosis, as, of course, it makes a great difference as to the value of the symptom whether the defect of sight be due to disorder of the dioptric apparatus, or to the disease of the retina or optic nerve.

The total blindness preceding the epileptic paroxysm is a different thing; and just as epilepsy is supposed to depend on contraction of the vessels of the brain, so the temporary amaurosis, in these cases, probably depends on contraction of the blood-vessels of the retinæ—an epilepsy of the retinæ.

I believe that the following is an instance of epilepsy of the retinæ. It seems clear that it was not merely failure of accommodation. One morning, Julia W., a middle-aged woman, came to me saying, that for five whole minutes she had been "blind." She was at the time seated peeling potatoes. The blindness came on suddenly and left suddenly. It was not total darkness, but "dark," which was the word she used herself in describing it. It was not from failure of accommodation. I asked her to look through a very strong convex glass. It was not like that, she said. It was not spots, nor specks, nor clouds, nor colours. When I saw her a minute afterwards, she could read well with each eye, and the fundus of each, as seen by the ophthalmoscope, was normal. She had headache across the forehead, which continued the next day. She said it felt "tight" across the forehead. She had no giddiness. She was regular, but subject to dyspepsia.

In cases in which loss of sight is followed by the epileptic paroxysm, may we not say that the contraction of the blood-vessels has begun in an outpost of the cerebral circulation (the retina being supplied by branches of the same vessels as the brain, these vessels being supplied by the same vaso-motor nerves), and that, on extension to the other branches of the carotid, the "brain's blindness," loss of consciousness supervenes? In but one of the cases of disease of the cerebellum, which I have reported, was there any note of temporary intermissions of sight. As a rule, the progress is gradual, and the loss of sight permanent.

To pursue the subject of the cause of reflex amaurosis in these cases, I may mention that it is not unusual, Dr. Brown-Séquard tells me, for guinea-pigs rendered epileptic by section of one lateral half of the spinal cord, to become amaurotic. Whether they are first subject to occasional and temporary loss of sight, it is, of course, impossible to tell. In a case reported in one of the Indian Journals, by Dr. H. V. Carter, in which it was clear, by the symptoms, that a man had, from an accident, suffered an injury similar to that artificially made in the guinea-pigs, there was a transient epileptiform seizure and transient affection of the sight of one eye. Dr. Carter writes:—"One night the patient had a sudden attack of dizziness and confusion, and the head fell towards the left side, and the sight of the eye temporarily failed." But transitory confusion of sight is due now and then to spasmodic affections of the external muscles of the eye. The patient, of course, does not know to what the confusion of sight is due; and when we do not see him during an attack, we can only judge by circumstantial evidence, more or less trustworthy. In a case of syphilitic epilepsy under my care, the patient was convulsed on the right side of the body. He had convulsions of the face, arm, and leg, and confusion of sight *before* he became insensible. After one of these attacks, the side on which he had been convulsed, became paralysed. The external rectus, as well as the face, arm, and leg, was paralysed. To the convulsive action of this muscle was due, no doubt, the defect of sight preceding the epileptic paroxysms.

Dr. Brown-Séquard has frequently drawn my attention to cases of paraplegia in which amaurosis has also existed, without any other symptoms to suggest disease within the cranium. These cases he refers to the same category as amaurosis from disease of the cerebellum. The blindness in both, he believes, is the result of eccentric irritation. Dr. Wilks also has observed several such cases.

In cases of paraplegia of the lower limbs, there could, of course, be no pressure on the corpora quadrigemina or the

optic tracts, and there does not appear to be greater difficulty in assuming a relation to exist betwixt the cerebellum or the spinal cord, and the retina, than betwixt the dorsal region of the spinal cord, and a certain tract on one side of the face in epilepsy artificially produced in the guinea-pig. That there is, in this latter case, a relation, is proved, not only by the fact that irritation of this part excites the fits, but also by the fact that the nutrition of the same part suffers; as on this side lice are often found, and not on the other. To use a common expression, the tract on the face and a certain part of the spinal cord are correlated, and it is, I think, possible that there is a similar correlation between the cerebellum or (posterior column of?) the spinal cord and the retina.

But there is, I think, another explanation. It is well known that there may be disease of the brain, and yet no appreciable alteration to the eye unaided by the microscope. There may be enough disease in the tubercular quadrigemina to cause blindness, and yet little to be seen on ordinary examination. In the production of epilepsy in guinea-pigs, by section of one lateral half of the cord, the condition in which the fits can be excited by irritating certain parts of the face is not established for several weeks. It is supposed that the epilepsy results, not because there is an injury to the spinal cord, but because disease spreads upwards from the part injured to the medulla oblongata. Changes at a still greater distance even are induced. The nutrition of a certain tract of the face is altered. Possibly in the same way in diseases of the cerebellum, changes of nutrition may creep along (the processus e cerebello ad testes?) to the corpora quadrigemina and optic tract, and thus give rise to blindness.

If this be considered plausible we should seek supporting evidence from other cases of cerebral disease. In a very valuable communication, by Dr. J. W. Ogle, in the Transactions of the Medico-Chirurgical Society, is recorded a case of hemiplegia caused by an aneurism pressing on the crus cerebelli of *the same side*. As it is known that injury of the crus cerebelli

does not produce paralysis on the same side of the body, some other explanation must be sought. It is supposed by Dr. Brown-Séquard that the paralysis is reflex, and is the result of irritation of the tumour; and as it cannot be due to the damage it inflicts locally, I venture to suggest that changes of nutrition may have passed along the portio dura and portio mollis (which were compressed and paralysed by the aneurism) to their origin in the opposite side, and that there they produced enough disease to cause paralysis on the then opposite side, *i.e.*, the side on which was the aneurism.

Andral, in his work on Clinical Medicine, narrates cases of disease of the cerebellum in which there was amaurosis, and also cases in which there was not. The following quotation is interesting.

"With respect to the fourth case published by Dr. Michelet in his Thesis, it is deserving of all our attention.

"This was the case of a girl, eighteen years of age, who ten years before her death had had an attack of apoplexy, the result of which was *amaurosis without any other paralysis*, and habitual headache. An apoplectic cavity of an old standing was found in the right lobe of the cerebellum."

Speaking of another case in which there was hemiplegia as well as blindness, and in which the disease found was softening of one lobe of the cerebellum, Andral writes: —"With respect to blindness, it seems at first that it has nothing to do with disease of the cerebellum, and yet this case is not the only one in which different affections of the cerebellum have been accompanied by a loss of vision. May this fact be explained by the anatomical relations established between the cerebellum and the tubercula quadrigemina by means of the prolongations known by the name of *processus e cerebello ad testes?*"

OPHTHALMOSCOPIC EXAMINATION DURING SLEEP.

It is scarcely necessary to say that my reason for examining the eye during sleep, was to help to form some idea as to the condition of the circulation in the brain itself in this physiological condition; the retina and the brain being supplied by branches of the same trunk, the carotid, and these by the same vaso-motor nerves. We may consider the retina as part of the brain extruded through an opening in the skull.

Still I have drawn no conclusions as to the condition of the circulation of the brain in sleep, as the subject requires to be studied on a very extensive basis, of which these observations can form but one part. Indeed I study the physiology of sleep, in order to learn somewhat as to the circulation in the brain in certain allied pathological conditions. For instance, I am anxious to know the condition of the veins and arteries of the brain in the profound sleep, or, perhaps more correctly, in the stupor which follows a paroxysm of epilepsy. I have examined the retina in this condition and several times in cases of severe congestive headache after a fit. The results of these and of many other examinations in cerebral cases I hope to have the pleasure of giving in some future number.

A girl, aged 11, was admitted into the Hospital for the Epileptic and Paralysed, under the care of my colleague, Dr. Brown-Séquard, for hemiplegia, which had existed several years. Of this, at the time when the following observations were made, there was little or nothing left, and the child was in fair general health.

I give the following extracts from my diary as the simplest way of recording several observations:—

Sept. 3rd.—I tried, first, to examine the eye without using atropine, but the pupil was so small, as is usual in sleep, that I could not illuminate the fundus. I therefore dilitated *one* pupil by atropine, and then examined the fundus of both eyes when the child was awake. I found the optic discs normal. They were equally well coloured, but not abnormally so. I had examined her sight carefully before dropping in the atropine, and found it perfect. When in deep sleep, one pupil was contracted; that dilated by atropine remained enlarged. By the aid of a very intelligent nurse, who held up the upper lid, I was enabled to examine the optic entrance, to which, for the present, I confine my observations. I found that the optic disc was whiter, the arteries a little smaller, and the veins larger than in waking. The veins were thick, and almost plum-coloured. The neighbouring part of the retina also was more anæmic.

Sept. 6th.—The pupil was now rather small. I saw the optic disc steadily, and could confirm my first statement. The arteries were certainly smaller, and the veins larger than in waking. The other parts of the optic disc were whiter, as was also the neighbouring part of the fundus. She had been well tired by a long romp with the nurse.

Oct. 3rd.—The pupils had regained their normal size. I again put atropine in the right eye, and examined with the opthalmoscope. I carefully noted each vessel, especially the smaller ones, and learnt by heart the position and size of both veins and arteries, and also the condition of the optic disc as to colour. At night I examined the eye during sleep. The pupil was smaller than when the child was awake, but I luckily saw well for a long time. The optic disc was not so red, the arteries were certainly smaller, and on this occasion, I think, the veins were no larger, and about the same as when the child was awake. I then roused her, and examined under similar conditions of light, position, &c. She was awake, but sleepy. I found that the arteries were larger; but, on looking again, I found them smaller, as in sleep. They alternated several times. I could not long dwell on

the disc; and my opinion is, that the alternation was gradual.

Oct. 16th.—A girl, aged 11, a patient under the care of my colleague, Dr. Ramskill. I dilated the right pupil with atropine. In sleep it remained dilated; the other was contracted. I saw well in this case. The child was deeply asleep, and I had the optic disc under view for a long time. All I can say is, that the disc itself was rather paler in sleep. I roused the child till she was fairly awake, and the only difference then was, that the disc was a little redder.

The pupil, under the influence of atropine, dilated to the fullest extent when awakened; twice the size it was when the child was asleep. The contraction was not due to the light only. It was the contraction of sleep.

Oct. 21st.—I put atropine in the right eye, and dilated the pupil to the fullest extent. The child is a somnambulist, and I found her in the ward at 10 p.m., in the arms of the nurse. The left pupil (the one without atropine) was not so small as usual in sleep. The other was as large as it was when she was awake. She was apparently asleep, however. I examined the eye, and then fairly awakened her, by pinching and making her speak, and getting her to look in certain directions. I again examined her eyes when she had gone to sleep in bed. I feel convinced that the arteries were a little smaller, and the veins larger. I saw well, and for some time.

Oct. 24th.—Atropine as usual. I saw well; the disc was whiter, and the arteries smaller.

I ought to observe, that in all these examinations the difference in the size of the arteries and in the coloration and the optic disc during sleeping and waking was but slight.

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