syndrome as convulsions, coma, relaxed sphincters, and fever to 104° F. (my first case), nor unconsciousness, hemiplegia, aphasia, irregularity of pupils, and relaxed sphincters (Cornil), may imply death. The gravest condition appears to be complete coma, general relaxation of the limbs, rising temperature, and Cheyne-Stokes breathing. The presence of ear mischief, old or recent, of organic disease of lungs, heart, or kidneys, of arterio-sclerosis, or advanced age, should be regarded as bad features; while, on the other hand, youth, a good constitution, and sound organs may afford a basis for hope, although cases have died which had all these points in their favour. The duration of the cerebral symptoms in the fatal cases has varied from two to fourteen days; but the last was exceptional, and death has generally occurred in less than a week from the onset of the cerebral symptoms. On the other hand, the cases that have recovered have usually shown definite signs of improvement in three or four days, but convalescence has been slow.

TREATMENT.

We are to a large extent powerless in the presence of the toxin of infectious processes, and can as a rule only wait and trust that the natural powers of resistance will prevail. Our hope lies in the future. The preparation of antitoxins is in its infancy, and who shall venture to predict the future? But if we can do little directly to oppose the effects of toxins, our means are even less available when such an organ as the brain becomes infected by a living pathogenic organism which causes death or grave impairment of the most essential functions by the destructive nature of the changes it produces in the delicate tissues of the very centre of life or of those powers which make existence worth possessing. When the lung or the heart is attacked, much mischief may ensue and permanent structural alteration result without causing death or even leaving behind serious impairment of function; but when the brain suffers, the consequences are so terrible that recovery may be often worse than death. If we possessed an effectual vaccine by which we could cut short the inflammatory process and induce healing, what fearful results might we see in impaired nerves or intellect, or partial paralysis of the eyes, the face, the muscles of the tongue, palate, and pharynx, which would make the patient or his friends regard the triumph of medicine as a sorry victory.

But meanwhile what can we do? If the patient can take medicine by the mouth, we should try small doses of quinine, which the late Sir William Broadbent believed to be a real prophylactic of influenza; and, if a prophylactic, it should possess some therapeutic power, too. We can feed the patient with milk, or egg beaten up, or with café au lait. If there is inability to swallow, we should try rectal feeding by small enemata of egg, milk, and a little salt, introduced slowly into the bowel through a soft rubber tube passed three or four inches into the bowel; or if, as too often happens, the sphincter is relaxed, we can inject half a pint to a pint of normal saline solution under the skin and stimulate the heart by similar injections of caffeine (5 grains) or camphorated oil. The last is much praised in France as a stimulant to the heart in collapse; the strength used is 1 in 10, and the dose 10 to 15 minims.

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At a meeting of the Royal Sanitary Institute, to be held at the Parkes Museum on Saturday, June 13th, at 11 a.m., Professor H. R. Kenwood will introduce a discussion on the pasteurization of milk. At a meeting of the institute at Coventry on the following Saturday, June 20th, at the same hour, a discussion on some aspects of the housing problem and town planning will be opened by Dr. E. H. Snell, M.O.H., and Mr. G. E. Swindlehurst, M.Inst.C.E., City Engineer, Coventry.

A Clinical Lecture

ON

HEMIPLEGIA WITH UNILATERAL OPTIC ATROPHY.

DELIVERED AT THE MANCHESTER ROYAL INFIRMARY.

By R. T. WILLIAMSON, M.D.LOND., F.R.C.P.,
ASSISTANT PHYSICIAN TO THE INFIRMARY.

GENTLEMEN,—I wish to describe to you four cases in which hemiplegia was associated with optic atrophy on the side opposite to the paralysis, whilst on the paralysed (hemiplegic) side the optic disc was normal. Such cases are rare, but they are of considerable interest with respect to the localization of the lesion.

The first case, presenting this association of symptoms, which came under my observation was that of a man, aged 46, who had suddenly become hemiplegic. There had been loss of consciousness at the onset, and from the symptoms it was not quite clear whether the case was one of cerebral thrombosis or haemorrhage, but the evidence was a little in favour of the former diagnosis. When I first examined him, about a month after the onset of the hemiplegia, there was optic atrophy on the side opposite to the paralysis, that is, there was left sided hemiplegia with right sided optic atrophy. At that time I was unable to explain the association of these symptoms.

The second case was that of a boy aged 7. He had suffered from pneumonia at the age of 4. Soon after the lung affection he became unconscious suddenly, and remained unconscious for a few days. There were no convulsions. On recovering consciousness it was found that the left side was paralysed. This left-sided hemiplegia gradually diminished, the leg recovering more rapidly than the arm. The movements of the left arm, however, remained very feeble, and though in course of time he was able to walk, he dragged the left leg. At the age of 7—a few weeks before I first saw the patient—he noticed one day, on closing the left eye, that he could not see with the right. How long the right eye had been blind could not be determined. Probably the affection of the right eye commenced at the onset of the hemiplegia. When I first saw him there was partial hemiplegia on the left side, with marked right-sided optic atrophy, whilst the left optic disc was normal. All the movements of the left arm were very feeble, especially those of the hand. He was able to walk without assistance, but he dragged the left leg a little. There was slight weakness of the lower facial muscles on the left side, seen when the patient retracted the angle of the mouth in showing the teeth. There was slight athetosis in the fingers and toes on the left side. The knee-jerks were present, but there was no ankle clonus. There was no anaesthesia, and no loss of the stereognostic sense in the left hand. There was no perception of light with the right eye. The right optic disc presented the well-marked changes indicating advanced optic atrophy, evidently of very long duration, but there were no retinal changes. The left optic disc and retina were normal. The vision of the left eye was normal. The heart and lungs were normal, and the general health appeared quite good.

The hemiplegia in this case could be explained by thrombosis of the right middle cerebral artery. If the thrombus extended downwards into the right internal carotid, to the point at which the ophthalmic artery is given off, it is conceivable that thrombosis might have occurred in the central artery of the retina, and that optic atrophy may have been caused thereby; or if the thrombosis had extended from the middle cerebral into the internal carotid, and a very small portion of the clot had become detached and passed into the ophthalmic artery, embolism of the central artery of the retina might have been produced on the right side. Thrombosis of the right middle cerebral artery, with thrombosis or embolism affecting the central artery of the retina would explain both the left-sided hemiplegia and the right-sided optic atrophy.

The diagram shows the internal carotid artery (1) passing from the base of the skull to the brain; the branch marked (2) is the ophthalmic artery; (3) is the central artery of the retina; (4) is the anterior cerebral,

(5) the posterior communicating, and (6) the middle cerebral artery. Obstruction of the internal carotid at the point (x), followed by obstruction in the central artery of the retina (3), and by obstruction extending into the

middle cerebral (6), would account for cerebral softening, followed by hemiplegia on the opposite side, and blindness and optic atrophy on the side of the lesion.

The pathological examination in the third and fourth cases gives some support to this view.

The third case was that of a woman, aged 37, who had suffered from syphilis. There was paralysis of all the ocular muscles of the right side (ophthalmoplegia and ptosis), with anaesthesia in the distribution of the first branch of the right fifth cranial nerve, and blindness of the right eye due to optic atrophy. The left optic disc was normal. On the left side there was hemiplegia which had developed suddenly. The *post-mortem* examination revealed a small gumma at the base of the skull under the dura mater and just behind the sphenoidal fissure. This had caused the paralysis of the ocular muscles and anaesthesia in the distribution of the first division of the fifth nerve on the right side, by pressure on the third, fourth, sixth cranial nerves, and on the first division of the fifth just behind the sphenoidal fissure. There was also thrombosis in the right middle cerebral artery, and this had produced the hemiplegia on the left side. Probably the blindness of the right eye had been caused by obstruction (thrombosis or embolism) of the central artery of the retina, but unfortunately the extent of the thrombus in the internal carotid was not definitely determined at the *post-mortem* examination.

The fourth case was that of a man aged 44, who came under treatment on account of loss of the stereognostic sense in the left hand. He had suffered from pain on the right side of the head for three months previously. On examination paresis of the left side of the face was detected. The optic disc and retina were normal on the right side. Vision was normal on the right side. The vision of the left eye was greatly impaired owing to an old opacity of the lens and choroiditis, caused by an injury in childhood. There was no paresis of the left arm or leg. The stereognostic sense was lost in the left hand, and there was also loss of the sense of pressure and position in the left hand. (I have recorded in detail the symptoms of the case in the *BRITISH MEDICAL JOURNAL*, December 9th, 1899, p. 1600.) Twelve days after the patient came under my care he suddenly became blind in the right eye, late in the evening. Next morning, on ophthalmoscopic examination, I found the appearances characteristic of obstruction of the central artery of the retina—pale optic disc with a large white patch in the centre of the retina, in the middle of which was a cherry-red spot. In a few days the large white patch with its cherry-red spot had disappeared, but the disc remained pale and passed into the condition of optic atrophy. The vision was lost permanently in the right eye. Soon afterwards hemiplegia developed on the left side, and death occurred in a few months. In this case there was finally hemiplegia on the left side, with optic atrophy on the right side.

During the last few weeks of life the patient was in the Manchester Workhouse Infirmary, under the care of Dr. E. S. Reynolds, who very kindly allowed me the opportunity of making a pathological examination of the brain. The *post-mortem* examination was made by Dr. Sizsons. I was unable to be present, but the brain was preserved for me in spirit. On examination of the

brain two days later I found cortical softening in the right cerebral hemisphere, chiefly in the region of the fissure of Sylvius—in the convolutions above and below this fissure. The basal ganglia did not appear to be affected. The exact limit of the softening could not be accurately defined, as there had been slight *post-mortem* decomposition. There was no softening in the left cerebral hemisphere.

The point of chief interest was the presence of a thrombus occluding the right internal carotid at its point of division into the middle and anterior cerebral arteries. The part of the internal carotid removed with the brain—just before its division—was filled with clot, also the anterior and middle cerebral arteries and the posterior communicating artery were occluded by thrombus at the circle of Willis. From the presence of the thrombus in the internal carotid, at a point close to the origin of the ophthalmic artery, there can be little doubt that either the thrombus had extended from the internal carotid along the ophthalmic artery to the central artery of the retina or that an embolus—that is, a fragment detached from the clot in the internal carotid—had passed along into the central artery of the retina. This was no doubt the cause of the ophthalmoscopic signs—white patch in the centre of the retina, with the cherry-red spot at the macula—and the optic atrophy which were observed during life. As already mentioned, the large white patch in the centre of the retina and the cherry-red spot at the macula soon disappeared, and only the signs of atrophy of the optic disc remained.

In this case the pathological examination could leave little doubt that the optic atrophy on the right side, with hemiplegia on the left side, were due to thrombosis of the right internal carotid extending to the middle cerebral, and also causing occlusion of the central artery of the retina on the right side (either by thrombosis which had extended along the ophthalmic artery to the central artery of the retina, or by an embolus detached from a clot in the internal carotid which had passed along the ophthalmic artery to the central artery of the retina).

These cases are examples of a peculiar combination of symptoms—optic atrophy on one side with hemiplegia (or hemiparesis) on the opposite side. Such symptoms could be produced by an obstruction (thrombosis) of the internal carotid and middle cerebral, with occlusion of the central artery of the retina by thrombosis or embolism. In the fourth case the pathological examination, and the ophthalmoscopic appearances just after the onset of the blindness in the right eye are strongly in favour of this explanation.

These cases are of interest with respect to the diagnosis and localization of the lesion. Probably a number of similar cases have been recorded in medical literature, but I believe that this combination of symptoms and its cause are not well known to medical practitioners.

Oliver-Sharpey Lectures

ON THE

PRESENT CONDITION OF OUR KNOWLEDGE REGARDING THE FUNCTIONS OF THE SUPRARENAL CAPSULES.

DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS
OF LONDON ON APRIL 7TH AND 9TH, 1908.

By E. A. SCHÄFER, LL.D., Sc.D., F.R.S.,
PROFESSOR OF PHYSIOLOGY IN THE UNIVERSITY OF EDINBURGH.

LECTURE II.

WE may now return to the results of the investigations made by Dr. Oliver and myself during the winter of 1893-4. The first point established was the *punctum saliens* of suprarenal activity—namely, that intravenous injection of the merest trace of suprarenal extract is capable of producing a striking rise of blood pressure; while with larger doses the rise may be so great as to be beyond the registering power of a mercurial manometer of ordinary length, the mercury being driven out from the open end of the manometer tube. We found this rise of blood pressure to be due to two causes—namely, (1) contraction of