

Two cases of simultaneous paralysis of both third nerves : with remarks upon ophthalmoplegia / by W. Allen Sturge.

Contributors

Sturge, William Allen, 1850-1919.
Tweedy, John, 1849-1924
Doran, Alban H. G. 1849-1927
Royal College of Surgeons of England

Publication/Creation

[London] : Printed by J.E. Adlard, [1881]

Persistent URL

<https://wellcomecollection.org/works/uttpmnqt>

Provider

Royal College of Surgeons

License and attribution

This material has been provided by This material has been provided by The Royal College of Surgeons of England. The original may be consulted at The Royal College of Surgeons of England. where the originals may be consulted. This work has been identified as being free of known restrictions under copyright law, including all related and neighbouring rights and is being made available under the Creative Commons, Public Domain Mark.

You can copy, modify, distribute and perform the work, even for commercial purposes, without asking permission.

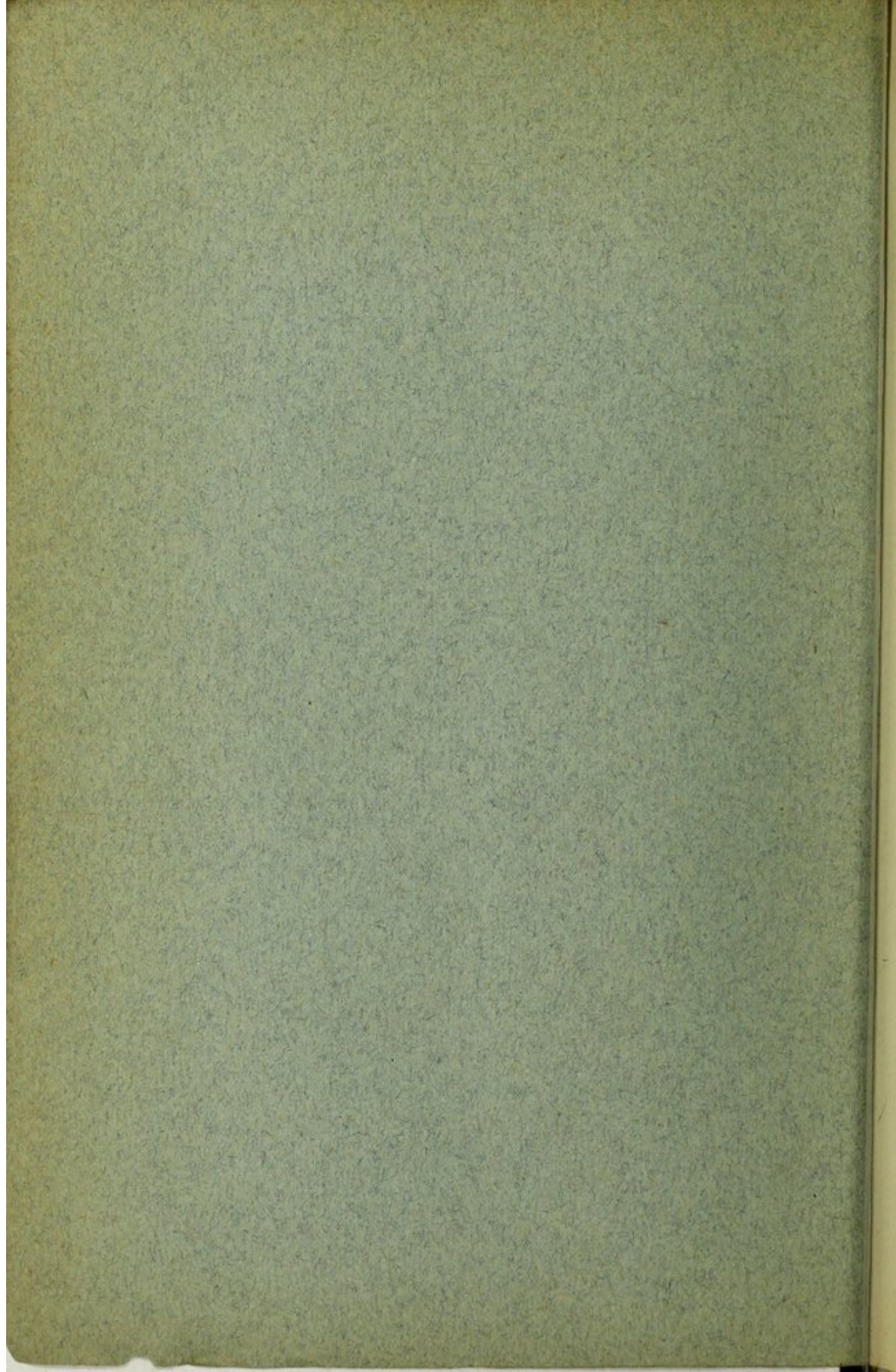


Wellcome Collection
183 Euston Road
London NW1 2BE UK
T +44 (0)20 7611 8722
E library@wellcomecollection.org
<https://wellcomecollection.org>

OPHTHALMOPLÉGIA.

BY

W. ALLEN STURGE, M.D., M.R.C.P.





TWO CASES OF SIMULTANEOUS PARALYSIS
OF BOTH THIRD NERVES, WITH REMARKS
UPON OPHTHALMOPLÉGIA.

By W. ALLEN STURGE, M.D.

George C—, æt. 72, was brought to the Royal Free Hospital by a policeman on Oct. 26th, 1880, having been found lying on the pavement in a neighbouring street.

The patient stated in answer to subsequent inquiry that he had generally enjoyed good health. He had suffered from venereal disease in his youth, but he could not recollect of what description. To the best of his recollection he had never suffered from any constitutional symptoms from this cause. Fifty years ago he was kicked in the nose by a horse. What he terms "a cancer" subsequently formed at the seat of injury, which remained unhealed for five years, at the end of which time it was removed by the late Mr. Skey, and a new nose formed by a plastic operation. He has worked as a farm labourer in this country and in America, and has suffered repeatedly from rheumatism, the result of exposure to cold and wet. For several years past he has been subject to winter cough. He has always been temperate. For some months previous to his present attack his sight has been

failing, chiefly in the left eye. There is nothing noteworthy in the family history.

History of present attack.—For a few weeks before the attack in the street the patient had been liable to occasional attacks of giddiness, but in other respects he was in his usual health. He had been working in the country, but on Nov. 10th he came to London to look for a brother whom he had not seen for some years. Failing to find his brother, he was walking the streets in a depressed state of mind, when he began to get giddy; the giddiness rapidly increased, and at last, to use his own words, he “pitched headlong down” and became unconscious. In this state he was found by the policeman, and was brought to the hospital, having in great measure recovered consciousness before reaching that institution.

Condition of the patient on admission.—The patient is a tall man, who wears his years well. His face is disfigured by the plastic operation already spoken of. He is quite conscious but complains of headache. He is unable to smell, having lost that sense ever since the injury to his nose. The sight of the left eye is almost gone, that of the right eye is fairly good. Ophthalmoscopic examination shows old choroido-retinitis in the left eye, the right fundus being healthy.

There is complete paralysis of all the extensor muscles of the right eyeball supplied by the third nerve, including the levator palpebræ. The pupil is contracted and insensitive to light and accommodation, but the use of atropine shows that there are old iritic adhesions, which are sufficient to prevent dilatation of the pupil. There is complete paralysis of the left superior rectus, internal rectus, and inferior rectus, and the left pupil is widely dilated and insensitive to light or movement of accommodation. The levator palpebræ on that side is, however, only slightly affected. The condition of the fourth nerve not satisfactorily ascertained, but the sixth nerves are unaffected.

There is very marked numbness on the right side of

the face, involving all parts supplied by the fifth nerve. In closing the jaw forcibly the right masseter remains flaccid, nor can he move the jaw towards the left side, showing that the right external pterygoid acts weakly. There is complete paralysis of the right side of the face. He is unable to close the right eye completely, notwithstanding the coexistence of ptosis. On trying to whistle or show his teeth only the left side of the face acts; the palate moves less fully on the right than on the left side.

There is very marked deafness in the right ear, he cannot hear the ticking of a watch at a greater distance than half an inch from that ear, nor does he hear better through the bones of the skull on that side. With the left ear he can hear the watch at a distance of three feet. There is no affection of swallowing; the tongue is protruded distinctly towards the right side.

There is complete paralysis of the *left* arm and leg both of motion and sensation. No affection of the bladder.

The patient rapidly improved after his admission. He soon began to recover power in the limbs, the recovery commencing in the arm sooner than in the leg, and in the hand and forearm sooner than in the upper part of the arm, so that he had free movement in his fingers before he was able to stand alone, and free use of the hand, wrist, and elbow, before he was able to move the arm far away from the side. He in great measure recovered from the right facial paralysis; the numbness of the face disappeared; that of the body became much less marked; the right levator palpebræ almost entirely recovered its power, but all the other muscles supplied by the third nerves remained permanently and completely paralysed, except the left levator palpebræ which had been unaffected from the first.

The leading features in his case may therefore be summed up as follows:

I. Immediately after his attack.

1. Complete paralysis of all the external muscles of the eyeball supplied by the right third nerve, together with paralysis of the right levator palpebræ.
2. Complete paralysis of all the recti supplied by the left third nerve, the left levator palpebræ being unaffected.

The pupillary condition could not be satisfactorily ascertained in either eye.

3. Paralysis of the *right* fifth nerve in both its motor and sensory divisions.
4. A complete (or peripheral) paralysis of the *right* facial nerve—the form of facial palsy occurring in “alternate paralyses.”
5. Paralysis of the *right* auditory nerve.
6. Left hemiplegia, motor and sensory.

II. His permanent condition was :

1. Complete paralysis of all the recti supplied by the third nerves in both eyes.

No affection of the levator palpebræ in either eye.

2. *Slight* affection of the right fifth and facial, and marked paralysis of the right auditory nerve.
3. Very slight left motor and sensory hemiplegia.

The “alternate” fifth, facial, and auditory paralyses all point to a lesion in the pons Varolii. The wide extension of the symptoms at the time of and shortly after the attack, compared with the rapid and almost complete disappearance of most of them in the course of a few weeks, point also to the presence of a *small* lesion in an important part of the brain where a great number of nerve fibres are collected in a small space, the larger proportion of which were only affected by shock, comparatively few being really damaged.

The chief permanent mischief would seem to have been to the nervous apparatus governing certain recti muscles, and of this I will speak more at length presently.

A further point of interest was the inverse method of recovery, the arm before the leg, and the hand before

the shoulder. This method of recovery is seen in connection with lesions in the lower or posterior portion of the motor tract in the brain, the opposite being true of the higher or more anterior portions of the motor tract.

The nature of the lesion in this case was probably a small hæmorrhage.

CASE 2.—George A—, æt. 16, employed in a beer bottling establishment, was admitted into the Royal Free Hospital between 5 and 6 o'clock on Saturday afternoon, Feb. 5th, 1881, in a state of unconsciousness.

The following history was obtained from his step-mother:—His father was living and healthy, his mother had died of smallpox. He had one brother living and healthy; one sister died at 19, who was subject to fits, which began when she was about 16 years old. Several children died during infancy. A paternal aunt had died of consumption æt. 17.

The patient had, as a rule, been strong and healthy. He was subject to occasional attacks of headache occurring about once in three months—rather more frequently during the last twelve months. They were, as a rule, not very severe, but twice during the last five years he had had a severe "bilious attack," in which his head ached all over, he was very sick and was obliged to keep his bed for a day or two. Two years ago he had an attack of pain in the back of his head and neck, with stiffness, so that he could not bend his head forward freely; he seemed very unwell and was kept at home for a week.

It was subsequently ascertained from himself that for four or five years he had been subject to attacks of giddiness. These attacks were not brought on by stooping, but would come on when he was standing upright. He would have an attack every day for several days, and then would be free for a week or two. The giddiness was usually accompanied by frontal headache shooting across from one temple to the other. For some time also he

had been liable to attacks of vomiting, sometimes every day, at other times not so often; these attacks would come on without any apparent cause and at any time during the day, though they were more frequent in the morning.

On one or two occasions he has fallen down unconscious, but he seems to have recovered quickly from these attacks. For some years he has been employed in bottling beer in a hot cellar. He has on several occasions been the worse for liquor, but this was some time ago, and latterly he has been very steady.

He left home quite well on the morning of the day on which he was admitted into hospital, and he remained quite well until between 3 and 4 o'clock, when he complained of pain in his head, and said that he felt so giddy and unwell that he thought he must go home. A few minutes after he exclaimed "Oh, my head is bad!" His companions noticed that his eyes looked "ghastly"; his fingers began to twitch, and he fell to the ground unconscious. He did not struggle further or foam at the mouth.

On admission he was quite unconscious. His breath smelt strongly of beer, and soon after his admission he vomited, the vomit also smelling of beer. His eyes were nearly closed, but on account of his loss of consciousness it was difficult to ascertain the state of the ocular muscles. The right pupil was larger than the left, neither being markedly dilated, and both reacting a little to light. There was no paralysis of the face, nor, so far as could be ascertained, of the limbs. There was no retention of urine. The urine was free from albumen or sugar. He remained unconscious the whole of the following day, vomiting in the morning and again in the evening. His head was shaved and an ice bag applied, and he was ordered five grains of calomel with fifteen grains of compound jalap powder. A blister was applied to the back of the neck. The bowels acted freely, the urine and motions being passed unconsciously.

On the following day, Feb. 7th, he had partially re-

covered consciousness, and it was then discovered that there was complete paralysis of all the extrinsic muscles supplied by both third nerves. There was complete double ptosis and complete paralysis of the superior rectus, the inferior rectus, and the internal rectus in both eyes, the external rectus being unaffected in either eye. There was consequently a divergent strabismus. The pupils remained much the same as already mentioned, the right being slightly larger than the left, neither pupil being markedly dilated, and both reacting a little to light; their action to accommodation could not be tested. There was no affection of other cranial nerves nor any paralysis of the limbs. There was no optic neuritis. The patient complained a good deal of headache. The temperature varied between 100° and 101° ; the pulse was slow, varying between 60 and 72, slightly irregular; breathing regular.

On the following day, Feb. 9th, the patient was quite conscious and was restless, tossing about in bed, but without evident pain. On this day the pupils were widely dilated and insensible to light. The morning temperature was 99.2° that of the evening 100° ; pulse 60.

He was in much the same state on Feb. 10th, but the following night he was exceedingly restless, complaining of great pain in the lower part of the back. The pupils were a little smaller again, and the left slightly larger than the right; they again reacted a little, although slowly and imperfectly to light.

A slight weakness of the left external rectus was now noticed for the first time. There was marked flushing of the cheeks and conjunctivæ, and there was also a well-marked serpiginous flushing over both arms. The temperature was 99° both morning and evening, and pulse 72.

A mustard plaster was ordered to the lower part of the back, and three grains of iodide of potassium three times a day were prescribed.

The following day, Feb. 12th, the patient was still complaining of great pain in the back, which made him toss about in bed and frequently call out. The weakness in

the left external rectus was more decided. Temperature and pulse remained about the same.

On Feb. 13th the pain in the back continued; the condition of the eyes was unaltered. The patient complained that his fingers felt numb.

On the 14th the pain in the back began to lessen, but there was still considerable pain in the head.

There was at this date complete paralysis of the left external rectus, the right external rectus remaining unaffected. There was for the first time evidence of a slight return of power in the right levator palpebræ, but all the other muscles remained completely paralysed. The right pupil was again slightly larger than the left, neither being markedly dilated. He was unable to read the largest print with either eye; this is evidence in favour of paralysis of accommodation. He said that he did not see double. Up to this time he passed his urine and fæces involuntarily; to-day for the first time he had retention of urine, necessitating the use of a catheter. There was still no optic neuritis and no paralysis of limbs.

On February 6th (two days later) it was noted that there was considerable improvement in the action of the right levator palpebræ, and the power was beginning to return in the left levator palpebræ. The pain in the back was gone, and he was passing his urine and fæces voluntarily.

On the 17th he had recovered almost complete power over the right levator palpebræ, and there was commencing return of power in the superior, inferior, and internal recti of that eye. There was slightly increased power also in the left levator palpebræ, and he was beginning to recover power also in the left superior and inferior recti, and in the left external rectus.

On the 21st the following note was made:

Right eye.—The action of the levator palpebræ is normal; there is still considerable deficiency of power in the superior and inferior recti. He can bring the internal rectus into action, but when told to look to the

left the right eye quickly wanders back to the middle line, giving rise to a kind of nystagmus, evidently due to the weakness of the muscle.

Left eye.—Levator palpebræ acts very much better than when the last note was made, but its action is slower than normal.

Superior rectus.—Action improved, but still much less than that of the right side.

Inferior rectus.—Action equally good with that of the right side.

Internal rectus.—Very slight action.

External rectus.—Acts much better than it did, but the eye reverts quickly to the middle line when the muscle is kept on the strain.

On the 19th and 21st of February there was a trace of albumen in the urine.

From this time onward the patient steadily improved, and when discharged on March 28th, except that there was a slight external strabismus, there was nothing abnormal in the action of the eye muscles. The right pupil still remained slightly larger than the left, but the pupils acted well both to light and accommodation. The sight was quite good. All headache and pain in the back had disappeared.

This case comes as a fitting pendant to my first case. In that case also the symptoms apparently dated their origin from an apoplectic attack from which the patient had suffered a few minutes before he was admitted to hospital. Some little doubt was, I believe, entertained in connection with my first case as to whether the double third paralysis was really due to the same lesion as the other paralysees; whether in fact the ocular paralysees had not existed previously to the apoplectic attack. Against this possibility were the facts that the patient was a fairly intelligent man and denied having noticed anything whatever to be the matter with him before the attack, and that in some respects the ocular symptoms improved at the same time that some of the other paralysees also improved.

The boy's case I have just related proves, I think, beyond question that double third paralysis may follow an apoplectic attack, and, therefore, removes from my former case any element of improbability which it may have possessed. I may mention that Mr. Hutchinson has recorded a case of double third paralysis associated with other paralytic symptoms, apparently dating from an apoplectic attack.

As with my first case, so now two questions arise :

1. What is the seat of the lesion ?
2. What is the nature of the lesion ?

Three facts pointed to the conclusion that the lesion was central, involving the nerves or their nuclei in the brain substance, rather than peripheral catching the nerves in their course from the brain to the orbits. These were—

1. The suddenness and severity of the onset of the attack.
2. The extensive implication of both third nerves contrasted with the freedom from attack of all other nerves until the lapse of several days, when one sixth became involved.
3. The association of sudden and complete paralysis of all the extrinsic muscles of the eyeballs supplied by the third nerves, with a partial and varying affection only of the pupils.

The grey matter in the neighbourhood of the aqueduct of Sylvius appears to be largely devoted to the oculomotor functions. Anatomical investigation has shown that the third nerves arise chiefly from two nuclei in the floor of the front part of the aqueduct, known as their nuclei of origin. But it is important to bear in mind that a minor portion of each nerve can be traced to other parts, some fibres going to the valve of Vieussens, some to the corpora quadrigemina, whilst the most anterior have been traced to a nucleus in the floor of the third ventricle, that I shall mention presently. The fourth nerves can be traced to two pairs of nuclei, one pair being near the nuclei of origin of the third nerves, and

the other in the floor of the upper part of the fourth ventricle. The nuclei of the sixth nerves are not far off, being also in the upper part of the floor of the fourth ventricle.

Physiological investigation proves, however, that the central connections of the oculo-motor nerves are far from being so simple as would here appear. Thus, Hensen and Voelcker have shown that in the hinder part of the floor of the third ventricle is a centre governing the ciliary muscle, and thus controlling the function of accommodation; that just behind this is a centre governing the circular fibres of the iris; and that still further back is a centre whose function is to control the radiating fibres of the iris. Again, Adamuk found that irritation of a certain spot in the corpora quadrigemina will produce an upward movement in both eyes, irritation applied a little behind this will cause movement of both eyes to the right or both eyes to the left, according as the stimulation is applied to the right or to the left of the median line, whilst if the irritation be applied still further behind, both eyes move downwards and converge.

Now, these are bilaterally co-ordinated movements, and the experiments tend to prove that bilateral movements of the eyeballs may be dependent upon single centres. Such centres would appear to differ from the nuclei of origin of the nerves in this, that while the latter include the *chief bulk* of the fibres going to form a nerve on *one* side, the former include a *few* fibres from *both* sides, these fibres passing up probably from the nuclei of origin. In the centres for bilateral movement, a more complex arrangement would seem to have taken place than in the nuclei of origin, with a view to the production of more complex co-ordinations. The nerve trunks bear much the same relation to the nuclei of origin that the anterior roots of the spinal nerve bear to the anterior horns of grey matter of the spinal cord; but from these comparatively simple and unilateral nuclei the fibres pass on to the higher and more complex centres, much as the

motor nerves of the cord pass up (if I may be allowed to invert the real relationship) to the great ganglia at the base of the brain. The grouping of fibres and cells in these higher centres will have ceased, however, to correspond with the grouping of the fibres in the nerves at their exit as third nerves, fourth nerves, and sixth nerves, and will correspond rather with the exigencies of ocular co-ordination; so that instead of a third nerve and its nucleus, and a fourth nerve and its nucleus, and so on, there will be the nucleus concerned in looking up with its attendant bundles of fibres; the nucleus concerned in looking to the right with its fibres; the nucleus concerned with accommodation and its fibres, and so on.

On *à priori* grounds, then, arguing from our anatomical and physiological data, we might expect to find several types of paralytic affection of the ocular muscles.

1. Paralysis due to peripheral affection of nerves, in which case the muscles would be paralysed in groups strictly corresponding to their nerve supply.

2. Paralysis due to disease in the so-called nuclei of origin of the nerves. In this case, in consequence of the fact that the great bulk of a nerve comes through the nucleus of origin, the paralysis would correspond more or less closely to the nerve supply. At the same time we might expect that these cases would present features differing, perhaps strikingly, from the strictly peripheral paralyses. In the first place, *all* the fibres of the nerve do not come through the nucleus of origin, for instance, the most anterior have been traced straight to the nucleus for accommodation in the floor of the third ventricle, which would point to the probability that the nucleus of origin has little or nothing to do with accommodation; and in the next place there are facts which tend to show that some small degree of simple co-ordination may take place even in the nuclei of origin. Thus Duval states that fibres pass up from the nucleus of the sixth nerve, cross in the body of the pons to the opposite side, ascend to the nucleus of the third nerve, and, without communicating

with its cells, join the outgoing bundles of the third nerve, and assist in the supply of the internal rectus. The nucleus of the sixth nerve of one side supplies, therefore, the external rectus of its own side, and helps in supplying the internal rectus of the opposite side. Obersteiner confirms this statement, which is probably only one of several similar facts that might be made out by careful research.

3. The third type of oculomotor paralyses would be those due to disease in the higher, bilaterally co-ordinating centres. In this case we should expect that the grouping of the paralysed muscles would cease to have any tendency to correspond to nerve supply, and would be governed by the association of muscles which produces co-ordinated movements. Of this type we should expect to find several varieties, thus :

a. Paralysis of the muscles involved in a single co-ordinated action, as of both levatores palpebrarum alone, of both superior recti alone, &c.

b. The converse of this condition, *i.e.* paralysis of all the muscles of the eyeball, with the exception of those on both sides involved in one or two co-ordinated actions.

c. Paralysis of accommodation alone, by disease of Hensen and Voelcker's centre of the function.

d. Paralysis of the radiating fibres of the pupil alone, by disease of Hensen and Voelcker's centre for dilating the pupil.

e. Paralysis of the circular fibres of the pupil alone, by disease of the centre, ascertained by these observers to govern the contraction of the pupil.

f. Far more commonly a mixture of the above varieties. In so complicated a mechanism included in so small a space we might expect that only in exceptional cases should we have any one function picked out cleanly without involvement of any other ; but where certain muscles are involved and others left out, the group of paralysed muscles in this type of disease is more likely to consist of some of the muscles supplied by the third nerves in conjunction with those supplied by the fourth and sixth nerves, than to

consist of all the muscles supplied by the third nerve alone, to the exclusion of those supplied by the fourth and sixth nerves—in fact, the association in the paralysis of all the muscles supplied by the third nerve, the superior oblique and external rectus being healthy, would point to the conclusion that we were dealing with the first and second types of ocular paralysis rather than with the more complex third type.

I will now pass on to consider to what extent these *à priori* paralyses are met with in practice, and I will begin with the third type of paralyses.

1. As an example of paralysis of muscles involved in a single co-ordinated movement, I will quote the case of a girl shown at one of the early meetings of this Society by Dr. Gowers, in whom both superior recti were paralysed, no other muscle being implicated.

2. For example of the second group of cases of this type, viz. those in which nearly all the muscles of the eyeball are paralysed, except one or two muscles in each eye associated in a co-ordinated action, I must refer to Mr. Hutchinson's paper on "Ophthalmoplegia Externa," published in the 'Medico-Chirurgical Transactions' for 1879, in which the following cases occur:—

a (Case 4).—All the muscles supplied by both third nerves were paralysed in addition to muscles supplied by other nerves, except the levatores palpebrum in which there was no "positive paralysis," and the two inferior recti.

b (Case 5).—All the recti were paralysed on both sides, the levatores palpebrarum being unaffected.

c (Case 6).—Like the last, all the recti were paralysed on both sides without ptosis.

d (Case 8).—There was moderate ptosis, and more or less paralysis of all the recti except the internal recti, the two muscles in fact so constantly associated together in converging the eyeballs to look at near objects.

e (Case 9).—Partial ptosis and paralysis of all the ocular muscles except the two inferior recti and one internal rectus.

f (Case 15).—All the muscles of both eyeballs, except the internal recti. No mention is made of ptosis.

g. In the first case of the series the levatores palpebrarum were only partially paralysed, but all the other muscles supplied by both third nerves, and some of the muscles supplied by other nerves, were completely paralysed.

Mr. Hutchinson included in his paper a brief account of seventeen cases of various forms of ophthalmoplegia externa. Out of the seventeen, we thus see that at least seven presented features bearing upon the point to which I am endeavouring to draw attention. Of the others, in several cases no muscles were omitted from the paralysis, and in several other cases the details published in the 'Transactions' of the Society are insufficient to enable us to judge of the exact condition present. Leaving out these two sets of cases it is sufficiently remarkable to find seven cases out of so small a number which may be quoted in this connection.

I will now pass on to the next groups of paralyses of this type, the occurrence of which might be suspected on *à priori* grounds, viz. paralysis of the radiating fibres of the iris; paralysis of the circular fibres of the iris; paralysis of the ciliary muscle. The functions of the three sets of muscular fibres are so intimately associated that it will be better to treat of the three forms of paralysis together.

In proof of the real occurrence of different varieties of paralysis of this description, I shall refer to Mr. Hutchinson's paper on "Ophthalmoplegia Interna," in the 'Medico-Chirurgical Transactions' for 1878. In that paper Mr. Hutchinson described seven cases in which papillary and ciliary symptoms occurred, and which he attributed to disease of the ciliary ganglion. It will be in the recollection of many of the members present, that at a recent meeting of the Society Mr. Hulke contended that these cases were due rather to disease of small ciliary ganglia. In the course of the discussion that followed, Dr. Gowers argued

that in all probability the disease in these cases is situated neither in the intraocular ganglia nor in the ciliary ganglion, but in the centre in the floor of the third ventricle, which have been shown by Hensen and Voelcken to govern their movements. I cannot help thinking that a careful analysis of Mr. Hutchinson's cases will prove that Dr. Gowers is right in his conjecture.

Of Mr. Hutchinson's seven cases there was one only in which all three functions were completely paralysed in both eyes, and in which therefore it would be impossible to say where the lesion was situated.

In one other case (No. 6 of the series) there was complete iridoplegia with weakened accommodation in both eyes, the power of accommodation varying at different times. In another case (No. 2 of the series) there was complete iridoplegia of one eye, the accommodation being weakened only. In a fourth case (No. 4 of the series) there was complete paralysis of all three functions in one eye, whilst in the other there was paralysis of the pupil (whether of both sets of fibres is not stated) combined with healthy accommodation. In a fifth case (No. 7 of the series) there was complete paralysis of both sets of fibres in the iris, combined at first with partial paralysis of accommodation. But the important point is this, the patient got nearly well from the paralysis of accommodation, whilst the iridoplegia was permanent. Another case is still more instructive. The patient (No. 1 of the series) had, in the left eye, at first paralysis of the *radiating* muscles of the iris alone, associated with partial paralysis of accommodation. Later on the circular fibres of the iris also became paralysed. In the right eye he had complete paralysis of both sets of fibres of the iris without any paralysis of accommodation.

If we are to attribute these different varieties of ophthalmoplegia interna to disease of the ciliary ganglion, we must perforce come to the conclusion that the ciliary ganglion is mapped out geographically in the most sharply defined manner; that there is the region in the ganglion

that governs dilatation of the iris, the region which governs contraction of the iris, the region governing accommodation. But there is nothing whatever to point to the probability of the mapping out of functions in a ganglion like the ciliary. All analogy would, on the contrary, point in the other direction, and lead us to conjecture that ganglia removed from the nerve centres and belonging rather to the sympathetic than to the cerebro-spinal nervous system, have more to do with the intimate association of closely allied automatic functions than with their differentiation from one another. On the other hand, experiment has clearly proved that in the cerebral centres this differentiation of correlated functions is carried to a high degree. When, therefore, we meet with lesions like those described by Mr. Hutchinson, which out of three intimately connected functions pick out one or two that they abolish completely, and leave one or two nearly or entirely healthy, is it not more reasonable to locate the lesion in the part where we know these functions are differentiated than in a part where we have every reason to suspect that no such accurate geographical differentiation is present.

But one of Mr. Hutchinson's own cases comes in to help us. In this patient (No. 5 of the series to which I last alluded) there was complete iridoplegia and complete paralysis of accommodation, but there was in addition slight weakness of the superior, inferior, and internal recti. Mr. Hutchinson attributed the paralysis of the iris and ciliary muscle, as in his other cases, to disease of the ciliary ganglion, and suggested that the paralysis of the external muscles was due to ascending degeneration passing up from the ciliary ganglion to the other parts of the third nerve. Two objections to this hypothesis at once present themselves. These are: (1) that degenerations do not tend in motor nerves to *ascend* from a periphery to the centre, but to *descend* from the centre to the periphery; (2) even supposing that an ascending degeneration might possibly or probably take place in a motor nerve, the

degeneration would be likely to confine itself to the fibres in the nerve proceeding upwards from the diseased part, and not to extend transversely across the nerve, so as to involve nerve fibres which have nothing whatever to do with the diseased part. Is it not far more probable that the whole disease was in this case central, involving chiefly the nuclei for the intraocular muscles, but extending in a modified degree to the nuclei for some of the extraocular muscles? And does not this case form an evident link between the group of cases constituting Mr. Hutchinson's "ophthalmoplegia interna" and the group constituting his "ophthalmoplegia externa?"

Again, the probability of the truth of this hypothesis is strengthened by reference to the condition of the pupils in Mr. Hutchinson's cases of ophthalmoplegia externa. Out of his seventeen cases, five were blind, and these cases consequently are of little or no value in this particular respect. In three others there is no mention of the condition of the pupils or accommodation. Of the remaining nine cases, in four the pupils and accommodation were normal; in one case both pupils were paralysed (apparently in both sets of muscles), the accommodation being healthy. In one case there was paralysis of the pupil and of accommodation in one eye, but no paralysis of either in the other eye—the external muscles of both eyes being affected; and in two other cases the pupil and accommodation were paralysed in both eyes.

We thus see that the cases in which the pupils and accommodation were affected, closely resemble Mr. Hutchinson's cases of ophthalmoplegia interna, so far as the varieties of affection of the pupils and accommodation are concerned, the only difference being that in the one set of cases the external muscles are also involved, and in the other that they are not. We thus get a chain of cases.

1. Accommodation alone affected.
2. Accommodation + pupil.
3. Accommodation + pupil + external muscles of eyeball.

4. Pupil alone.
5. Pupil + external muscles of eyeball.
6. External muscles of eyeball alone.

It is needless to add that there would be many sub-varieties in this chain according as the radiating or circular fibres of the iris, or both, are affected, and according to the grouping of the paralysis of the external muscles.

I may mention in further proof of the marked differentiation of the pupillary and accommodation centres from the centres for the eye movements in the neighbourhood of the aqueduct of Sylvius, that the cases of ophthalmoplegia externa mentioned by Mr. Hutchinson, where the pupil and accommodation were unaffected, were some of the worst, so far as the external muscles are concerned, that he has recorded; and, further, that in the patient with ophthalmoplegia externa, who had paralysis of the pupil and of accommodation in one eye and not in the other eye, the paralysis of the external muscles was much more extensive and complete in the eye with healthy pupil and accommodation than in the eye in which the pupil and accommodation were paralysed.

I will now pass on to speak very briefly of the second type of ocular paralyses, viz. those due to lesion of the *nuclei of origin* of the nerves. From the fact that in both the cases of double ocular paralysis after apoplexy that I am here recording, the paralysis was grouped much more according to the geographical distribution of the third nerves than to the association of muscles for the production of co-ordinated movements. I believe that both cases belong to this secondary category, and we may, I think, by analysing the cases, learn some of the facts relating to this type of paralysis.

In my first case the patient, an old man, presented complete double third paralysis, with the exception of the left levator palpebræ, the fourth and sixth nerves being healthy. For reasons which I gave when speaking on the case, it was difficult to ascertain the condition of the pupils. Subsequently, the right levator palpebræ re-

covered power, but the three recti supplied by the third nerve on each side remained permanently and completely paralysed. This fact that it was only the recti supplied by the third nerves that were paralysed, the muscles supplied by the fourth and sixth nerves being healthy, would lead us, as I before said, to locate the lesion in or near the nuclei of origin of the nerves; whilst, on the other hand, the fact that the levatores palpebrarum were unaffected or only temporarily affected, would lead us to believe that already in the nuclei of origin a certain degree of differentiation of function has taken place, and that the fibres supplying the levatores palpebrarum have their nuclei of origin independently of the general nuclei of origin of the third nerves.

I have already mentioned that anatomical investigation has pointed to the probability of this early differentiation of function in the nuclei of origin, and this case comes in further support of the probability.

In the second case there was, after an apoplectic attack, complete paralysis of all the muscles supplied by both third nerves, at first without implication of muscles supplied by other nerves. Subsequently a complete paralysis of one sixth nerve came on slowly. It is doubtful whether at any time the pupils were completely paralysed. For the greater part of the time, at any rate, they were only partially affected, and it is worthy of note that this partial affection applied equally to the radiating and circular fibres of the iris. The remarkably well-defined geographical distribution of the paralysis points to lesion of the nuclei of origin of the third nerves, including the nuclei for the levatores palpebrarum. The partial affection only of the pupil, tends to prove, what was already suggested by anatomical investigation, that the pupil centres are early differentiated from the generating third nerve centre, and that the nucleus of origin of the third nerve has little to do directly with the pupils. As regards the subsequent and gradual onset of sixth paralysis, three possible explanations arise.

1. That in the inflammatory condition which was set up as the result of the lesion, the sixth nerve became compressed in its course beneath the lens. A supposition rendered possible by our knowledge of the great ease with which the sixth nerve becomes paralysed by slight pressure.

2. That as the inflammation became established round the lesion, the effects of the lesion extended to neighbouring parts and implicated the nucleus for the movement of both eyeballs to the left; or,

3. Still more probably, perhaps, that the fibres passing from the nucleus of origin of the sixth nerve upwards to this co-ordinating nucleus became compressed as the tension round the seat of lesion became greater.

We may, I think, shut out the possibility of the after effects of the lesion extending to the nucleus of origin of the sixth nerve, because of its distance from that of the third nerve, and the impossibility that the effects of the lesion should have extended to it in a degree sufficient to completely paralyse it, without affecting other centres in the neighbourhood.

As to the nature of the lesion; it was probably a small hæmorrhage due to rupture of a minute artery or capillary, sufficient to cause pressure upon the part but insufficient to destroy the nerve structures.

I must apologise for having trespassed so much upon the time of the Society, but the subject is a complex one, and has grown under my hand to a greater length than I had anticipated. It will grow much more under the hands of those who are more competent than myself to deal with it. All I have endeavoured to do, has been to analyse such material as we have already, with a view to establishing some working hypotheses that may help us in investigating future cases. I believe that a careful study of clinical facts, in their manifold variety, will enable us, at no distant date, to explain fully the complex mechanisms connected with the movements of the eyes.

(May 12th, 1881).

