OCULAR PALSIES.*

BY

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OCULAR palsies constitute a symptom of the greatest interest and importance—of interest because they offer a very wide field for diagnostic skill as well as an opportunity for a study of many different diseases, and of importance because they are not infrequently one of those early signs of disease of the nervous system which call for exact diagnosis and appropriate treatment. The eye is indeed an outpost, not only of the nervous system, but often of the cardiovascular and other systems of the body also.

Under the term "ocular palsies" I include any form of paralysis or weakness of any of the ocular muscles, both external and internal. It is not my intention to discuss separately the question of diplopia, for this is, of course, a regular symptom of a palsy of any of the external ocular muscles, unless such be of very long standing.

A real appreciation of this subject must include a brief anatomical and physiological introduction. The internal muscles of the eye are three: (1) the sphincter pupillae, whose action is to contract the pupil; (2) the dilator pupillae; and (3) the ciliary muscle, whose chief action is to relax the suspensory ligament of the lens and assist in the process of accommodation. The sphincter pupillae and the ciliary muscles are supplied by the third cranial or oculomotor nerve, via the ciliary ganglion and the short ciliary nerves. The dilator pupillae is innervated by the cervical sympathetic, which reaches it by a roundabout route, but eventually also by the ciliary ganglion.

The external ocular muscles, seven in number, are the levator palpebrae superioris, the four recti muscles, and the inferior oblique muscles. All but two are supplied by the third nerve, the exceptions being the external rectus, supplied by the sixth nerve, and the superior oblique, by the fourth nerve.

The third or oculomotor nerves have their cells of origin or nucleus in a longitudinally arranged column of cells lying on each side of the middle line in the grey matter, just below the aqueduct of Sylvius in the dorsal or tegmental part of the midbrain. This nucleus is placed just below the anterior corpora quadrigemina, and from it the fibres pass directly ventralwards through the substance of the midbrain to emerge on the inner side of the cerebral peduncles, close to the upper border of the pons. Two main sets of fibres arise from this centre: (1) those innervating the external muscles of the eye, and (2) fibres to the intrinsic muscles of the eye, the sphincter of the pupil and the ciliary muscle. This latter is an indirect connexion via the ciliary ganglion, and the whole system of fibres belongs to the so-called autonomic system.

Much dispute has raged over the exact arrangement of the cells of origin for the different muscular movements in the oculomotor nucleus, but we can distinguish three principal groups of cells: (1) a small-celled lateral nucleus, that of Edinger and Westphal, connected with the innervation of the sphincter pupillae; (2) a small-celled mesial nucleus connected with the process of accommodation; and (3) a large-celled lateral nucleus, whose fibres are destined for the supply of the external ocular muscles (Bing¹). Made up in this way the third nerve passes forwards, pierces the dura mater at the side of the posterior clinoid process, runs along the outer wall of the cavernous sinus, and enters the orbit through the sphenoidal fissure.

The fourth cranial nerve is of little clinical importance, so that I will not detail its anatomy.

Much farther down in the brain stem, in the lower part of the pons, is the nucleus of the sixth or abducens nerve. It is placed just below the grey matter in the floor of the fourth ventricle, and is in close relations to the emerging fibres of the seventh nerve. Leaving the lower border of the pons it passes forwards along the base of the skull,

occupies the inner wall of the cavernous sinus, and enters the orbit through the sphenoidal fissure. Its long intracranial course renders it of special clinical importance.

For the proper and co-ordinated movements of the eyes it is necessary that all these nuclei be in intimate relations with one another. This is brought about largely by the posterior or dorsal longitudinal bundle, and so important is this tract for the purpose that a lesion of it may produce diplopia or even actual ocular palsy, for which the special name of "internuclear" paralysis has been suggested. On this bundle depends the well known associated action of the sixth nerve of one side and the third nerve of the other, bringing about the conjugate lateral movements of the eyes. It is usually held that the sixth nerve nucleus or some centre in its close proximity is the lower or sub-cortical centre for this movement.

The other movements of the eyeballs are seldom the result of the simple action of one muscle alone, with the exception of the levator palpebrae superioris, which elevates the upper lid. Thus the superior rectus and the inferior oblique are associated in the elevation of the eyeball, and the inferior rectus and the superior oblique with the movement of depression.

Nearly all the movements of the eyes, therefore, are associated movements, and there are other important association centres besides that in the sixth nerve nucleus. There is a very important supranuclear centre in the cerebral hemispheres which is not infrequently of clinical importance. Experimental evidence has shown that in all animals with binocular vision there is a cortical centre in the second frontal convolution, stimulation of which causes conjugate movement of the eyes to the opposite side. There is, further, clinical evidence that such a centre exists in man, governing the conjugate lateral movements of the eyes. We have no evidence at present that conjugate vertical movements of the eyes have any representation in the cerebral cortex, though we may surmise that such exist. "Undoubtedly, from clinical and *post-mortem* studies, we are justified in saying that in the upper end of the mid-brain in the neighbourhood of the anterior corpora quadrigemina are association centres which control the conjugate vertical movements of the eyes" (Holmes²).

It is well also to bear in mind, especially when the symptom of diplopia is in question, how close and how intricate are the connexions between the innervation of the eye muscles on the one hand, and the auditory and vestibular and cerebellar mechanism on the other. Finally, as Sherrington³ has pointed out, the existence of afferent fibres in the oculomotor nerves must be remembered, the integrity of which is probably essential for the proper functioning of the ocular movements, and the correlated postures and movements of the head and trunk.

One other point of both physiological and pathological interest may be mentioned—that is, the pathway of the pupillary light reflex. This reflex arc is composed of four sets of fibres, from the retina to the superior corpus quadrigeminum, thence through the Edinger-Westphal nucleus to the ciliary ganglion, and from that to the sphincter pupillae. The connexion between the superior corpus quadrigeminum and the Edinger-Westphal nucleus is both homolateral and crossed; hence the possibility of the consensual light reflex. It is fair to add that authorities differ as to the exact details of this reflex path. I have here followed Bing.¹

We are now in a position to consider the various sites where a lesion may interrupt the neuromuscular chain and bring about an ocular palsy. Following Paton⁴ we may classify these as follows:

1. Paralysis of central or cerebral origin.
 - (a) Supranuclear lesions—for example, paralysis of conjugate movements from a lesion of the second frontal convolution.
 - (b) Internuclear lesions—for example, of the posterior longitudinal bundle.
 - (c) Nuclear lesions—for example, degenerative lesions of the nuclei in *tabes dorsalis*.
2. Paralysis of extracerebral origin—that is, of nerve trunks.
 - (a) Intracranial—for example, from a basilar meningitis.
 - (b) Intraorbital—for example, new growths.
3. Paralysis of muscular origin—for example, *myasthenia gravis*.

* Abridged from a paper read before the Southampton Medical Society on November 7th, 1923.

To turn now to the pathological aspect of the question. What are the diseases which may cause an ocular palsy in the various parts of the neuromuscular chain? Here again I borrow Paton's⁴ classification, which is a very useful one on clinical grounds.

1. Acute and subacute ophthalmoplegias.
 - (a) Poisons: alcohol, lead, botulism.
 - (b) Infective agents: diphtheria, influenza, encephalitis lethargica, syphilis, tubercle. To this I would add infections of the accessory nasal sinuses.
 - (c) Trauma.
 - (d) No known etiological factor.
2. Chronic ophthalmoplegias.
 - (a) As an isolated lesion.
 - (b) In tabes and general paralysis of the insane.
 - (c) Disseminated sclerosis, syringomyelia, and paralysis agitans.
 - (d) Bulbar palsies, progressive muscular atrophy, myasthenia gravis.
 - (e) Graves's disease.
 - (f) Diabetes.
3. Congenital ophthalmoplegias of nuclear origin.
4. Unilateral ophthalmoplegia.
5. Recurrent and alternating ophthalmoplegia of nuclear origin.

I have collected the notes of the last 45 cases of ocular palsies which I have had under my own observation, and propose to discuss these briefly, and chiefly from the etiological point of view. This series of cases does not include examples of affections of the internal eye muscles only. All the cases included showed some external ocular palsy, though naturally many were also associated with some disturbance of the internal musculature as well. Classifying these cases from the etiological point of view I find that they fall into the following groups:

1. Syphilis (including tabes; no case of general paralysis of the insane has occurred), 15 cases, or 33.3 per cent.
2. Cerebral tumours, 5 cases.
3. Encephalitis lethargica, 6 cases.
4. Sinus infections, 5 cases.
5. Ear disease, 2 cases.
6. Trauma, 1 case.
7. Vascular lesions, 3 cases.
8. Disseminated sclerosis, 2 cases.
9. Migraine, 1 case.
10. Post-diphtheritic neuritis, 1 case.
11. Congenital ophthalmoplegia, 1 case.
12. Of doubtful etiology, 3 cases.

Syphilis in some shape or form is generally held to be responsible for about 40 per cent. of all cases, and it is certainly a very important etiological factor when we remember the variety of ways in which it may produce an ocular palsy. Thus gummata may occur in the orbit, or at the base of the brain, or even in the cerebral substance itself. Meningitis, whether of the convexity or of the base, is common enough; syphilitic disease of the blood vessels with subsequent thrombosis may cause cerebral softening; and finally, degenerative lesions of the nerves and nuclei are frequently found in tabes and general paralysis of the insane.

Of my own cases, 9 could probably be classified as meningo-vascular syphilis, while the remaining 6 were probably due to tabetic degeneration and were associated with definite evidence of tabes—such as pains, loss of the deep reflexes, and alterations in sensation. One of these was an example of juvenile tabes in a girl of 14, who, besides dilated and fixed pupils, showed a ptosis on one side. Out of these 15 cases only 3 showed normal pupils with normal pupillary reflexes, a point emphasizing the diagnostic value of changes in the pupils in syphilis of the nervous system. Typical Argyll Robertson pupils were only found in 4 cases. But inequality of the pupils, irregularity of the pupils, and complete loss of pupillary reactions to light and accommodation were frequently encountered. I shall refer later to the diagnostic value of the Argyll Robertson pupil and its interpretation. The so-called tabetic myosis, or very small contracted pupils, did not occur in this series of 15 cases of ocular palsies; 6 cases showed a paralysis of the sixth nerve, whether alone or in combination with some affection of the third nerve; 11 cases showed paralysis of one or more muscles supplied by the third nerve, excluding the sphincter of the pupil. As far as prognosis is concerned, it may be

said that the tabetic cases showed little or no tendency to improve with treatment, whilst the meningo-vascular cases did.

Of the cases of cerebral tumour, 5 in all, 3 are of special interest, for in all of these eye symptoms were the first to draw attention to what eventually proved a fatal disease. A girl of 8 came under observation for diplopia of a week's duration. A failure of the power of convergence with weakness of both external recti muscles and nystagmus were the first signs in the eyes. Weakness of the conjugate movements of elevation and depression of both eyes appeared later, with sluggish pupil reactions. Still later double optic neuritis, a right hemiplegia with gross ataxia, and complete paralysis of the upward and downward movements of both eyes followed. A diagnosis of tumour of the midbrain was indicated, but the patient took her discharge from hospital and no autopsy was obtained.

A youth, aged 19, had suffered from diplopia for six months before coming under observation. He then showed nystagmus, a paralysis of the left sixth nerve, and a paralysis of the right third nerve. Later there appeared signs pointing, like the first case, to a tumour of the midbrain, with nearly complete ophthalmoplegia. He died within six months.

A third and not dissimilar case was of special interest as at an early stage the phenomenon of double Argyll Robertson pupils was found. This was a boy, aged 8, who was seen in consultation because "he had headache and his eyes had looked funny for fourteen days." He showed slightly unequal and somewhat dilated pupils, which gave the Argyll Robertson reaction, a paralysis of the upward movements of both eyeballs, a slight weakness of the left external rectus, and commencing optic neuritis. The further course of the case gave little room for doubt that he had a tumour of the midbrain, as he developed quite distinct hydrocephalus, with nearly complete double ophthalmoplegia, gross optic neuritis, followed by atrophy and a hemiplegia with a double extensor plantar response.

The other two cases were as follows. One was a girl, aged 10, who had a glioma of the left lateral lobe of the cerebellum, verified by operation. She showed a paralysis of the left external rectus. The other was an elderly woman suffering from a large carcinoma of the breast, in whom double optic neuritis with a paralysis of the right external rectus appeared. This last sign was probably an example of the so-called "false localizing signs of intracranial tumour," for later a complete right hemiplegia appeared, and it seemed certain that the lesion lay in the left cerebral hemisphere. A left subtemporal decompression was performed, with temporary relief. Later a paralysis of the conjugate movement of both eyes to the right appeared. This was considered to be due to the lesion involving the left second frontal gyrus. *Post-mortem* examination showed that this was the case, but that the damage to the cortex had followed the operation. No lesion was found in the pons. The early paresis of the right sixth nerve remained unexplained, unless it was due to the general increase in intracranial tension. In any event, the case is of value as showing the little localizing value to be attached to a weakness of one sixth nerve.

The cases of ocular palsies due to infection of the accessory nasal sinuses form a particularly interesting group. They are of importance, too, because their occurrence seems not generally recognized.

Case 1.—A man of 56 had suffered from diplopia for two months. On examination paresis of the left external rectus was found. Two months later there was slight ptosis of the left eye, with some oedema of the upper lid and a clear nasal discharge. Transillumination showed the left antrum to be slightly opaque. Operation was refused.

Case 2.—A man of 65 had diplopia of ten days' duration, pain behind the left eye, and a paresis of the left external rectus. There had been occasional discharge of clots of blood and matter from the nose. Resection of part of the nasal septum and operation on the ethmoidal sinus completely cured the ocular palsy.

Case 3.—A man of 42 had pains in the eyes and headaches for three months, with occasional nasal discharge. There was ptosis in the right eye. Extensive ethmoidal and sphenoidal sinus disease was found, treatment of which resulted in cure of the ocular palsy.

Case 4.—A girl of 14, with left ptosis of eight weeks' duration. Occasional discharge of "thick stuff" from the nose. After operation on the nasal septum and adequate nasal treatment, the ptosis gradually disappeared.

Case 5.—A man aged 40. The left eye had been inflamed at times for the last three years; for one year there had been discharge of blood-stained material from the left nostril; for six weeks pain in the left side of the neck. There was an incomplete palsy of the left third nerve, with ptosis and intense photophobia. Proptosis of the eye followed. After operation on the sphenoidal cells great improvement followed.

In the investigation and treatment of this group of cases I had the great advantage of the collaboration of my late and much lamented colleague, George Nixon Biggs, who operated on all of them. He also cured for me the two cases of ocular palsy associated with disease of the middle ear. In each of these the external rectus only was affected. Both were completely cured by a radical mastoid operation.

The cases of ocular palsies following encephalitis lethargica are an interesting group. Of all focal symptoms referable to the central nervous system in this disease, I suppose that affections of the ocular muscles are perhaps the most common, certainly during the acute stage of the disease. They are, however, much less common as a final result or sequela. The common sequela in this respect is some lesion of the internal muscles of the eye, inequality of the pupils often, and especially common is a loss or a defect in the power of accommodation or convergence of the eyes, or both. All the cases here quoted, however, were with one exception seen within at least one month of the supposed onset of the disease.

Case 6.—Examined fourteen days after the onset of symptoms; showed a paralysis of the left external rectus with inequality of the pupils. Re-examination at the end of eight months revealed no abnormality.

Case 7.—Seen twelve days after the onset; showed double ptosis.

Case 8.—Seen fourteen days after the onset; showed unequal pupils, with slight left ptosis and nystagmus. In this case the slight ptosis and the pupil inequality persisted for nine months, after which time the patient was lost sight of.

Case 9.—Was not seen until five months after the original illness, the diagnosis of which, therefore, had to be made in retrospect. There was present paralysis of the left external rectus, slight paresis and slight atrophy of the left side of the tongue, and a paresis of the left side of the palate. One year later the paralysis of the left external rectus had completely disappeared, but the weakness and wasting of the left side of the tongue persisted.

Case 10.—Was seen eighteen days after the onset; there was partial double ptosis, with involuntary movements. Later a divergent squint in the left eye appeared.

The next case was remarkable in every way, and but for the very clear history of the original illness would almost certainly have been diagnosed as myasthenia gravis, for varying ocular palsies appeared and disappeared in a striking manner over a period of at least two years.

Case 11.—A woman, aged 29, was seen on October 7th, 1920. She gave a history of the acute onset of diplopia with fever and severe headache one month before. Careful examination excluded any general nervous disease and syphilis. On the first examination there was found a divergent squint in both eyes with a paresis of both internal recti. In November this persisted, but a right ptosis had appeared. In December of the same year (1920) this ptosis was extreme, but fourteen days later the ptosis had disappeared and defective downward movements of the right eye had appeared. In January, 1921, ptosis of the left eye was found; there was still a paresis of both internal recti and in addition a defect in the up and down movements of the left eye. In March, 1921, there was no ptosis in either eye, but a marked weakness of the left internal rectus. In May, 1921, right ptosis again appeared. In June the right ptosis was replaced by a left ptosis. In July the left ptosis was still present combined with a paresis of both internal recti. By August, 1921, the left ptosis had gone. In September of the same year no ptosis was found, but an alternating divergent squint in each eye, with marked weakness of the left internal rectus and defects in the vertical movements of each eye. A failure of convergence was also present. In January, 1922, no defect was found except a divergent squint, but in November, 1922, the ptosis had appeared again on the right side. A slight divergent squint in the right eye and a failure of convergence was also found.

This case is also especially interesting as illustrating the fact that the virus of this disease probably persists in some cases for a very long time, and is still capable of producing new effects after the acute phase of the disease is long past.

The only case in the present series due to trauma was that of a man who was knocked down by a motor car, and who was unconscious for three days. Persistent diplopia with paralysis of the left external rectus ensued, presumably caused by a fracture of the base of the skull.

The three cases diagnosed as due to vascular lesions all occurred in patients the subjects of high arterial tension,

in whom syphilis was excluded. Curiously enough, they were all women.

One was a woman of 68 in whom a sudden onset of diplopia with intense giddiness occurred. She showed a paralysis of the right internal rectus muscle and probably of the right inferior oblique also. A further point of great interest was the presence of an Argyll Robertson pupil in each eye. She was the subject of advanced arterio-sclerosis, with a blood pressure of 210 mm. mercury systolic and 110 diastolic. The Wassermann reaction was negative in the blood and the cerebro-spinal fluid was quite normal. No further symptoms developed and the ocular palsies gradually improved, including the Argyll Robertson reactions, which gradually disappeared, leaving, however, pupils with a distinctly sluggish reaction to light.

The second case was a woman of 53 with a paralysis of the right external rectus of quite sudden onset associated with headache. The blood pressure was 185 systolic and 120 diastolic. Slow but almost complete recovery took place.

The third case was a woman of 54 with a partial paralysis of the left third nerve, again of sudden onset. The blood pressure was 170 systolic and 110 diastolic.

Only two cases of undoubted disseminated sclerosis occurred in this series. One was a woman of 25, who suffered at intervals from diplopia and giddiness. She showed nystagmus and a weakness of the left external rectus. The other was a woman of 34, who in 1914 had a paralysis of the left external rectus which recovered spontaneously. In 1922 this reappeared and was associated with a weakness of the upward movements of both eyes, as well as other definite signs of the disease.

The single case of migraine here included was a youth of 19, who for many years had suffered from typical attacks of severe headache, nearly always on the left side of the head. For three weeks before coming under observation he had had diplopia. On examination a paresis of the left internal rectus was found.

One example of persistent post-diphtherial ocular palsy has occurred, although the evidence as to the etiology is incomplete, bacteriological proof being lacking. A man was examined on account of a right ptosis and unequal pupils. The only cause found was a very definite sore throat some weeks before, when the throat had been bad for two weeks and had been "covered with a whitish film." Tests for syphilis were all negative.

The three cases labelled as of doubtful etiology were all of considerable interest.

One was a man of 52, with persistent diplopia and probably a paresis of the left inferior oblique muscle. Repeated examination has failed to disclose a cause.

The second case was that of a man with slight ptosis and double Argyll Robertson pupils. He stated that the eyes had become affected during an attack of typhoid fever a year before. No evidence of any active disease has been found and I certainly think that his own account of the etiology of the condition must be accepted.

The third case was that of a woman with a recurrent palsy of the left external rectus in whom all the available means of exact diagnosis have been exhausted.

The case of congenital ophthalmoplegia was that of a child aged 1 year and 8 months at date of examination, in whom a defect in the movements of the eyes had been noticed at the age of 3 months. There was a complete loss of the movements of the eyeballs outwards, with presumably complete loss of action of external rectus.

Except in so far as other paralytic symptoms have been noted as occurring in certain of the cases of cerebral tumour quoted, I have not specifically described any of those forms of ocular palsies associated with different varieties of hemiplegia. These conditions form a very definite and interesting albeit a rare group of syndromes, which generally go by the name of the writer who first described them. They are commonly due to vascular lesions, haemorrhage or softening, affecting the crura, midbrain, or pons. Four in particular may be mentioned, as they are usually recognized as constituting definite syndromes. They are:

1. *Weber's syndrome.* A paralysis of one third nerve, with hemiplegia of the opposite side. The lesion is in the posterior or caudal end of the crus cerebri.

2. *Benedikt's syndrome.* Paralysis of one third nerve with tremor of the opposite limbs. The lesion lies in the more dorsal part of the midbrain, involving the red nucleus or its connexions.

3. *Foville's syndrome.* The sixth nerve nucleus is affected, with paralysis of the external rectus, loss of the conjugate

deviation of the eyes to the side of the lesion, paralysis of the seventh nerve on the same side, and a hemiplegia of the opposite limbs. The lesion is in the pons.

4. The *Millard-Gubler syndrome* is the same, but without the paralysis of the conjugate deviation of the eyes. The lesion is lower down in the pons, the nucleus of the sixth nerve escaping damage.

Frequent reference has been made in the course of my remarks to the Argyll Robertson pupil. Although I have been dealing more especially with affections of the external ocular muscles I feel obliged to make more than a passing reference to this symptom, whose importance certainly deserves a paper to itself. The Argyll Robertson pupil consists in an absence or a gross diminution of the reaction of the pupil to light, with the preservation of the reaction on convergence-accommodation. It was originally described in association with cases of tabes, and it is in this disease that it is still best recognized and most widely known. The characteristic pupillary reactions were described by Argyll Robertson in cases of spinal disease with myosis or contracted pupils. It is, however, now a matter of common agreement *not* to restrict the term to cases with contracted pupils. There has been a tendency at times to regard this reaction as specific evidence of syphilis of the nervous system. This view cannot now be upheld, although it may be granted at once that syphilis is by far the most common cause. Those who are interested in the subject will do well to consult Wilson's⁵ paper in the *Journal of Neurology and Psychopathology*, where an admirable review of the whole question is set out. This author mentions the following different conditions where the Argyll Robertson pupil has been recorded:

1. Epidemic encephalitis.
2. Disseminated sclerosis.
3. Cerebral tumours in the vicinity of the third ventricle, aqueduct, or anterior corpora quadrigemina.
4. Syringomyelia or syringobulbia.
5. Chronic alcoholism.
6. Traumatic cases.

In cases from my own personal experience, which I have already quoted, there have been three examples of the Argyll Robertson pupil, in all of whom syphilis was excluded. These were as follows: (1) a tumour of the midbrain; (2) a vascular lesion; and (3) a case in which the condition appeared to have originated during an attack of typhoid fever. Wilson and de Rudolf⁶ have lately published a case of a mesencephalic tumour with Argyll Robertson pupils, and Natrass⁷ one of bullet wound of the midbrain. De Monchy⁸ also records a highly interesting case of tumour of the pineal gland with ocular disturbances, including Argyll Robertson pupils.

Some general remarks on the diagnosis of the site of the lesion may be attempted. Ocular palsies due to lesions in the orbit do not come under the notice of the physician or neurologist so much as those due to lesions in the skull. But some characteristics of orbital lesions may be mentioned: (1) They are apt to be unilateral. (2) A single muscle may be affected. (3) The internal muscles are not as a rule affected so much as the external. (4) Proptosis is common. (5) A complete paralysis of all three ocular nerves is perhaps in favour of an orbital lesion.

Palsies due to lesions at the base of the brain tend to show the following characters:

1. Other cranial nerves are often affected.
2. A paralysis affecting all branches of the third nerve, and especially if bilateral, is usually due to a basal lesion.
3. In cases of trauma other signs, such as bleeding at the nose or escape of cerebro-spinal fluid, will of course speak for a basal lesion.
4. Signs of meningitis, such as severe headache with head retraction and fever, naturally will suggest a lesion at the base of the brain.

Pontine lesions are suggested by a paralysis of the conjugate lateral movements of the eye, especially if associated with a paralysis of the fifth and seventh nerves as well and with a crossed hemiplegia.

Palsies due to lesions in the midbrain—that is, affecting the oculomotor nuclei in the region of the anterior corpora quadrigemina—may be distinguished by some of the following characters: (1) Other signs and symptoms of intracranial

tumour, especially optic neuritis. (2) Various affections of the movements of the eyes governed by the third nerves. In the earlier stages these affections tend not to be complete, but to pick out some of the movements, while others are spared. Thus, ptosis may be present alone, or a defect in the upward movements only. Holmes,² speaking of the existence of supranuclear centres for the vertical movements of the eyes, mentions "the frequency of disturbance of conjugate vertical movements of the eyes by tumours growing from the pineal body or from the splenium of the corpus callosum, which readily compress the anterior colliculi, and by the observation during the war of three cases of injury of the tectum alone by small penetrating missiles in which the vertical movements of the eyes were affected."

In conclusion I must record my best thanks to my colleagues on the staff of the Western Ophthalmic Hospital, for their kindness in giving me the opportunity of seeing many of their cases, a number of which are included in the personal series which I have quoted.

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Three Lectures

ON

CONTRACTED PELVIS.

DELIVERED IN THE UNIVERSITY OF LONDON,
FOR ADVANCED STUDENTS,

BY

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II.

DIAGNOSIS AND CLINICAL EXAMINATION IN CONTRACTED PELVIS.

Diagnosis.

THE diagnosis of contracted pelvis is easy and can be made at any time before or during pregnancy or in labour. In the nulliparous woman it is made from physical characters and facts; in the parous it is often suggested by the history of previous labours.

Ante-natal clinics establish the fact of contraction, and by so doing serve a useful purpose by preventing a number of cases from entering or continuing in useless labour before the condition is recognized, but owing to the absence of any method of accurately classifying such cases it is impossible to define the treatment in more than a very few.

Contracted pelvis should always be suspected in women of small stature, quite irrespective of any maldevelopment. The majority are under 5 ft. in height; this or the previous history, in women free from maldevelopment, is all that is likely to suggest contraction before the thirty-sixth week; the actual contraction will be established by measurements.

External Measurements.—There are three principal measurements to be taken. The external conjugate, which is normally 20 cm.; and I think it may be accepted that reduction below 19 cm. is chiefly at the expense of the cavity, while when the external conjugate is reduced to 18 cm. the true conjugate is below the average biparietal diameter. There are two transverse measurements—the interspinous and the intercrystal; of these the intercrystal is the truer indicator of the transverse diameter of the brim; it is normally 29 cm., and reduction to 27 cm. means diminution of the cavity. The interspinous is normally 3 cm. less than the intercrystal, but as it is subject to outward splaying in cases of rachitic origin, it is often misleading as to the internal dimensions. From these measurements some idea of the type and degree of contraction is obtained, but little else.

Internal Pelvimetry.—The internal measurements of the pelvis can be taken with special pelvimeters—the best