

OCULAR PALSIES.

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An acquired ocular palsy must always be regarded as a highly significant physical sign, and usually as an index of gross intracranial disease. By reason of its complex and extensive anatomical disposition, the oculomotor apparatus is particularly vulnerable to a large number of pathological processes. All the resources of diagnostic skill may, therefore, be required to reveal the causation of a particular case of ocular palsy.

It is convenient to consider the oculo-motor apparatus as consisting of an upper and a lower motor neurone; the upper comprises the supranuclear pathways from the cortex to the nucleus, the route being probably broken up into at least two constituents. The lower neurone comprises the nucleus of the ocular nerve and the peripheral portion of the nerve, between the emergence from the brainstem and its termination in the ocular muscles. The lower neurone also includes, of course, the "stem" or that short part of the nerve running through the substance of the midbrain (or pons) from the nucleus to its emergence from the brainstem.

Bearing in mind these anatomical points, we may divide ocular palsies into: (1) *upper neurone palsies*; (2) *lower neurone palsies*. Each of these is capable of sub-division. Thus the upper neurone palsies are usually divided into (a) *conjugate palsies* (where movements of both eyes in a particular direction are impossible, whatever the type of stimulus) and (b) *supranuclear palsies* (where conjugate movements are affected or not, depending upon the type of stimulus). The lower neurone palsies are divisible into (a) *nuclear palsies*; and (b) *peripheral palsies*. Theoretically, of course, it is also possible to recognise (c) *stem palsies*, where the intrapontine or intramesencephalic portions of the nerve are affected.

The chief clinical distinction between the upper and lower neurone palsies depends upon the fact that the former entails a paralysis of eye *movements*, whereas the latter consists in a paralysis of eye *muscles*.

Thus, in the case of a conjugate paralysis, the patient is unable to look in a particular direction, whether upwards, downwards or to the side. The defect is noticeable in *both* eyes.

With a supranuclear palsy there is again a disorder of conjugate movement of both eyes in a particular direction. But, by altering the type of stimulus, full movement is apparently effected. Thus, while the patient may be unable to look upwards on command, he may perhaps be able to follow with his eyes an object which is moved in an upward direction. Or, if the patient fixes his gaze steadfastly upon an object held before the eyes, and the head is then passively flexed upon the chest, the eyes may now appear to move upwards. This phenomenon is sometimes spoken of as the "sleeping doll" sign.

Nuclear palsies may be unilateral but are more usually bilateral. They are frequently associated with other cranial nerve palsies; thus a bilateral abducens nuclear palsy will typically be accompanied by a facial diplegia.

Peripheral nerve palsies may be isolated or multiple, unilateral or bilateral. Whereas in nuclear affections the ocular palsy is always complete, partial paralyses may be found in the case of peripheral lesions. Thus a nuclear lesion of the third nerve will give the complete syndrome of ptosis, a dilated and fixed pupil,

and an inability to look up, in or down on the affected side. Part only of this syndrome may occur with a peripheral oculomotor lesion.

Many of the morbid conditions under which ocular palsies occur are diffuse in nature, so that palsies of either upper or lower type may occur, or both may exist simultaneously. From the clinical standpoint, therefore, it is more convenient to consider ocular palsies as a whole and to examine the circumstances under which they most commonly occur.

The physician, attempting to investigate a given case of ocular palsy, relies on three chief clinical guides:—

1. An exact determination of the ocular palsy in anatomical terms.
2. The presence or absence of pupillary abnormalities (internal ophthalmoplegia).
3. The nature of other neurological abnormalities which may be present.

In other words, the physician must satisfy himself: (1) whether the ocular lesion is expressible in terms of supranuclear, conjugate, nuclear or peripheral paralysis; and (2) by very careful testing, whether the pupils are equal in size and whether they react with equal promptness and range to light and on accommodation. A full clinical examination of the nervous system will clear up the third point.

In an adult, the two commonest causes of ocular palsies are neurosyphilis, and cerebro-vascular disease. We are purposely restricting the category of an ocular palsy to those cases where some defect of movement is present in the eyeballs, and are excluding cases where the major abnormality is in the eyelids or the pupils (*e.g.*, Graves' disease; oculosympathetic syndromes; post-encephalitic states).

Neurosyphilis.

In syphilis of the nervous system, the oculomotor apparatus is frequently involved in numerous situations. A basal leptomeningitis may implicate the peripheral portions of the 3rd, 4th or 6th cranial nerves. Syphilitic periostitis of the orbit may give rise to a unilateral "syndrome of the sphenoidal fissure" with paralysis of the ophthalmic division of the 5th nerve as well as of the 3rd, 4th and 6th nerves. The result is a homolateral complete ptosis; a dilated and fixed pupil; absolute immobility of the eyeball; and impaired sensation over the supraorbital region as well as the upper eyelid, the cornea and conjunctiva. Gummatous thickening of the nerve-trunks may be responsible for an isolated ocular nerve-paralysis. Syphilitic vascular disease may cause lesions within the nuclei themselves, or in the "stem" of the ocular nerves, or in the supranuclear pathways. Ocular palsies can also occur from syphilitic hydrocephalus; and from direct cellular degeneration within the nuclei themselves.

The syphilitic nature is usually betrayed: (1) by the presence of pupillary disorders; (2) by other signs of neurosyphilis; (3) by positive serological reactions; and (4) at times, by the presence of other manifestations of syphilis, of which leukoplakia, aortitis and periostitis of the shins are the commonest.

Treatment affects the syphilitic ocular palsies in a varied way; meningeal, gummatous and cerebrovascular lesions will tend to respond much more satisfactorily than parenchymatous affections of the nuclei.

Cerebrovascular disease.

Considered in its widest sense, disease of the intracranial bloodvessels is probably the second commonest cause of an ocular palsy in the adult. Here again, all parts of the oculomotor system are liable to be attacked.

In cases of cerebral softening or hæmorrhage conjugate paralyzes are commonly present, in association with hemiplegias. More rarely, supranuclear palsies, including the interesting cases of ocular apraxia may be noted—more especially with diffuse or multiple vascular lesions.

Small foci of softening within the brain stem may affect the ocular nuclei and—more especially—the “stem” portion of the nerves. There may result an interesting series of crossed paralyzes, of which the following are the best known:—

- (a) Weber's syndrome: homolateral third nerve paralysis and a contralateral hemiplegia. Here the lesion is in the ventral part of the mid-brain, affecting the pyramids and the stem portion of the oculo-motor nerve.
- (b) Benedikt's syndrome: homolateral third nerve paralysis and a contralateral tremor and rigidity of extrapyramidal type. The lesion lies in the middle zone of the mid-brain, and affects the oculomotor nucleus and the red nucleus.
- (c) Millard-Gubler syndrome: homolateral paralysis of the sixth and seventh cranial nerves, and a contralateral hemiplegia. A lesion in the dorsal part of the upper pons is responsible.
- (d) Foville's syndrome: this differs from that described by Millard and Gubler by the addition of the conjugate deviation of head and eyes to the side of the lesion. The focus of disease here lies a little higher.

Isolated ocular palsies may also occur as the result of direct pressure upon the peripheral nerves by tortuous, atheromatous or aneurysmal arteries. Two interesting anatomical relationships are of importance here: the third nerve, on emerging from the mid-brain passes between the superior cerebellar artery and the posterior cerebral artery, and hooks abruptly forward over the latter. Any abnormality in shape or calibre of this artery is liable to pull upon the third nerve and interfere with its function.

The sixth nerve also bears an intimate—though variable—relationship with the anterior inferior cerebellar and internal auditory arteries, and disease of either of these vessels may cause traction upon the nerve.

In spontaneous subarachnoid hæmorrhage the cranial nerves may become entangled in the basal meshwork of fibrin and blood clot, with resultant ocular palsies.

The diagnosis of vascular disease as the cause of ocular palsies is suggested by the abruptness of onset of symptoms, evidences of peripheral arteriosclerosis, of hypertension and of cardiac hypertrophy. Most suggestive are the signs of retinal arteriosclerosis, viz.: excessive brilliancy of the arteries, irregularities in the calibre, and compression and distortion at the arteriovenous crossings (Marcus Gunn's sign). The absence of clinical or serological evidence of syphilis is most important. Normal pupillary reflexes are the rule though there is an exception in a rare but well defined vascular syndrome comprising lid retraction (or ptosis), paralysis of conjugate upward deviation of the eyes, and absence of pupillary contraction to light. The lesion is probably near the posterior commissure and upper quadrigeminal region.

A great number of less common causes of ocular palsies may be enumerated. They comprise:—

Myasthenia gravis. Here variability of paralysis, and integrity of the pupils are important. Characteristic signs in other parts of the nervous system are usually evident.

Intracranial tumour. Here one must distinguish cases where ocular palsies are of localizing significance (as in the case of pineal, mesencephalic, pontine and auditory tumours), from the palsies which constitute "false localizing signs". Amongst the latter, an isolated abducens paralysis is a well known feature of internal hydrocephalus irrespective of the site of the growth.

Meningitis.

Cranial injury, especially when the base of the skull has been fractured.

Acute and chronic forms of encephalitis.

Polyneuritis cranialis, associated with an acute peripheral neuritis.

In the case of a *child*, cerebrovascular disease obviously does not appear as a common cause of ocular palsy. Syphilis, too, is infrequent owing to the comparative rarity of inherited tabes and general paralysis of the insane. The more usual causes of ocular palsy will, therefore, be found in meningitis and in cerebral tumour, although in the very young the possibility of a congenital defect should be borne in mind.

CORRESPONDENCE.

26 Great Ormond Street,
W.C.1.
27th July, 1934.

The Editor,
"The Post-Graduate Medical Journal".

DEAR SIR,

There appears to have been a little misunderstanding, in some quarters, over the issue by us of our advertisement of HALMAGON which appears on your front cover this month.

It has been pointed out to us that we issued this advertisement some days before your magazine was published. If this has caused you any inconvenience we are extremely sorry and can assure you that it was ignorance of your publishing date, that caused us to anticipate your Journal's publishing day.

We would point out, however, in mitigation of our offence, if we have offended, that it is a general practice after permission has been obtained to circulate, for various reasons, extracts from periodicals in advance of the periodicals' publication. This refers to both reprinted articles and advertisements.

That this reprint was not submitted for your inspection before we circulated it, was, perhaps, a lack of courtesy on our part, which we also regret; but as we were under the impression that we actually had obtained permission from your advertising manager to issue the copy, and as we desired to get this advertisement out as soon as possible, this formality was overlooked.

We trust this will clear up any misunderstanding over the matter. We assure you that throughout we acted in perfect good faith and if we have caused you the slightest inconvenience we are extremely sorry and ask you to accept our apologies.

Yours faithfully,

For TONICITY LABORATORIES LIMITED,

CHARLES VIVIAN,

Director.

NOTE.—The Editor gladly accepts the explanations contained in the above letter and fully appreciates the sentiments therein expressed.