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PALSIES OF THE CONJUGATE OCULAR MOVEMENTS*

BY

GORDON HOLMES, C.M.G., M.D.,

LONDON.

IN attempting to determine the site and cause of a lesion which affects a conjugate movement of the eyeballs in any direction, we must keep before us the complex physiological apparatus which regulates the normal movements of the eyes. For these occur not only in response to voluntary impulses, but also as reflex movements excited by peripheral stimuli arising in the retinae, in the ocular muscles themselves, in the labyrinths, in the cervical muscles, and even in response to auditory and cutaneous stimuli. Further, while some of these reflex movements, such as the adjustments of our eyes to changes in the position of our heads and bodies, depend on the activity of sub-cortical centres only, others require the intervention of the forebrain cortex.

Our most natural starting point is the cerebral cortex, which is the organ directly concerned in the voluntary acts. From the earliest days of experimental cerebral physiology the existence of certain areas has been recognized in the forebrain, from the excitation of which associated ocular movements result. The best

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known of these cortical centres is that in the second frontal gyrus, which, when stimulated, causes, in animals with binocular vision, conjugate movements of the eyeballs towards the opposite side. This area is generally regarded as a motor centre for the ocular movements; but Sherrington and Leyton have found that the reactions elicited from it differ from those obtained from the precentral motor area, the chief differences being the stronger stimulus required to elicit the movement, the less regular response, the deliberate development of the movement, and the fact that the eyes remain for a time in the position into which they move, and come back slowly. Sherrington and Leyton obtained no purely vertical movements from this frontal area, but others have elicited them either directly, or, as Dr. Risien Russell and Bechterew, after section of the lateral rotators of the eyes, or destruction of the abducens nuclei.

There is unequivocal evidence of the existence of this frontal centre in man too. The most demonstrative evidence is afforded by those cases in which an irritant lesion of this region excites clonic spasms or convulsions; in such cases clonic conjugate movements of the eyes to the opposite side are often the most prominent feature. For instance, one man under my own observation, in whom a meningeal gumma compressed the second frontal convolution, was subject to an enormous number of seizures, all of which commenced with, and some only consisted of, conjugate movements of the eyes towards the opposite side. Weakness of the conjugate deviation of the eyes to one side is not uncommonly due to a lesion of this portion of the brain, but it is usually regarded as a temporary symptom only. During the late war I had the opportunity of observing a very large number of men in whom gunshot wounds had damaged the cortex of the second and third frontal gyri, and my experience was that in every case of recent injury of this region, the patient was either unable to move his eyes fully towards the opposite side, or the movement was less prompt and needed more effort, or that the eyes swung back quickly to their primary central position as though the innervation was not strong enough to maintain their lateral deviation. These disturbances seemed to disappear within a few weeks of the infliction of the wound, when the patient was merely tested by asking him to follow a moving object with his eyes; but other tests showed they were more permanent. When he was asked to turn his eyes promptly to the one side on receiving the order "eyes right" or "eyes left," or when the observer, holding up his hands each at an equal distance to the right and left of his visual axes, asked him to look at once at the fingers which moved on either side, it could be seen that though he turned his eyes promptly and without the slightest effort towards the side of the injury, their movements towards the opposite

side were slower, less complete and accurate, and obviously demanded more effort. Further, if the tests were continued for some time deviation in this direction tired quickly. This paresis of the purely voluntary movements of the eyes, or of movement in response to a stimulus that attracted attention, may be a more or less permanent effect of frontal lesions. I have also found it a valuable test in the localization of tumours in this region of the brain.

The frontal oculomotor centre seems to be concerned only with movements of the eyes which are initiated by volition. All the reflex movements, as those excited from the labyrinth and by purely retinal impressions, as well as those higher reflexes in which conscious visual perceptions are concerned, remain intact when it is destroyed.

I have seen no clinical evidence that the vertical movements of the eyeballs are represented in this frontal area. Its destruction has never, in my experience, influenced the upward and downward movements of the eyes, nor has its irritation produced clonic involuntary movements in these directions, though oblique lateral and upward or downward deviation is not uncommon in epilepsy.

A second cortical centre has been described by Sir David Ferrier in the angular gyrus. On stimulation of its anterior portion in monkeys he obtained upward movement of the eyes, downward movement from its posterior part, and lateral movement from the intermediate area. Bernheimer also obtained movements of the eyes in all directions from this region, but many other experimenters, as Lewandowsky, could elicit only conjugate lateral movement to the opposite side.

As far as I am aware, there is no definite evidence of an oculomotor centre in this position in man; I never saw a gunshot wound of this region of the brain disturb the ocular movements, and my civil experiences point to the same conclusion. I may cite two cases in which special observations were made. In one man, from whom the late Sir Victor Horsley removed a portion of the right angular gyrus, there was absolutely no limitation of the ocular movements after the operation. In a second case, the removal of a large tumour from the left angular gyrus, producing a destruction so deep that a right homonymous hemianopia resulted, did not influence the ocular movements in any way. We may therefore conclude against the existence of a motor centre for the ocular movements in the parietal lobe. I have, however, repeatedly observed a special disturbance of the ocular movements by gunshot wounds in this region, which must, however, be regarded as a manifestation of apraxia and not as a palsy of movement. In some of the most striking cases, as one which I published, there was apraxia of the limbs too. The chief feature of oculomotor apraxia is failure to obey an order to look in any direction, or to turn the

eyes promptly to any point, although the patient is fully aware of its position in space, and though there is no trace of weakness of movement. This condition of ocular apraxia, which we cannot discuss more fully here, is sometimes associated with disturbances of spatial orientation, but I observed it as an isolated symptom in men with lesions in the region of the left angular gyrus.

Schäfer first, and a large number of physiologists after him, obtained conjugate eye movements from the visual area in the occipital lobe, but it is improbable that this area contains a true motor centre of the same nature as that in the frontal lobe. According to Munk, the movements of the eyes elicited from it depend on the production of perceptions in the visual area by the stimulation, the eyes being then reflexly directed towards the point in the outer world to which the perception is projected.

Paretic deviation of the eyes is occasionally associated with lesions of the occipital lobes that produce hemianopia; the patients then look away from their hemianopic side, but this is not constant, and, in my experience, it rarely persists for long. In gunshot wounds of the visual cortex I never found more than a very transient deviation of the eyes towards the seeing side, and it was as a rule noteworthy only when the patients were in a dull or stuporose condition.

There has been considerable discussion on the nature of this deviation of the eyes associated with hemianopia. According to Bard, Roux, and others, it is secondary to the hemianopia, the loss of retinal afferent impressions producing a relative functional depression of the oculomotor centres in the affected hemisphere. But Déjerine and Roussy, who dispute this explanation, have observed conjugate deviation of the eyes associated with a recent occipital lesion in a woman who was blind from birth, and in whom, therefore, visual impressions can never have exerted any influence on their ocular movements. That the occipital lobes contain centres from which ocular movements may be effected is also made probable by the facts that fibres can be traced from them to the midbrain, and that movements of the eyes can be elicited from the calcarine region after removal of the frontal centres. It seems probable that this efferent occipital system is not concerned in willed and purposive movements of the eyes, but that through it, such movements may be excited by visual impressions that reach the cortical level, as those which bring objects perceived by the peripheral portions of the retinae into central vision, and those by which the eyes follow a moving object. The visual cortex is consequently a higher ocular reflex centre, as well as a perceptive area; and the deviation of the eyes towards the seeing side in hemianopia is due to loss of the visual impressions that under normal conditions excite its reflex activity.

It is, however, disease of the brain-stem that produces the most complete and permanent, and consequently the most obvious, disturbances of the conjugate movements of the eyes. The most common form is limitation or loss of the lateral movements to one side, and as it is easier to discuss this, especially with reference to the anatomical site of the causal lesion, than loss of the vertical movements, we will deal with it first.

Paralysis of the Conjugate Lateral Movements of the Eyes

It is well known that inability to move the eyes to one side is frequently associated with disease in the lower and dorsal portion of the pons Varolii, especially when it extends to the neighbourhood of the abducent nucleus of the same side. A lesion, for instance, in the region of the left abducens nucleus may make the patient unable to move either eye to the left of the middle line, that is to contract the left external rectus and the right internal rectus muscles. But these two muscles are innervated from two different nuclei about 3 cms. apart, and the root fibres proceeding from them nowhere come together in the brain-stem. It is therefore necessary to assume that these two nuclei are connected by association fibres which distribute the exciting impulses to the cells of both. We have such association fibres in the dorsal longitudinal bundles; these, it is well known, contain ascending and descending fibres, many of which are intimately connected with the motor nuclei of the ocular nerves. Some of these fibres come from Deiter's nuclei, others ascend from the ventral columns of the spinal cord, but a large proportion of them spring from the nuclei and scattered cells of the tegmentum of the brain-stem, or take origin in the tectum of the midbrain, chiefly in the anterior quadrigeminal bodies. The latter receive retinal fibres that are probably concerned in the reflex movements and adjustments of the eyes. The dorsal longitudinal bundles may be consequently regarded as associational and reflex optic pathways. The next question which arises is the origin of those fibres that convey to the abducens and oculomotor nuclei, the impulses that excite conjugate lateral movements. According to one hypothesis, which is generally accepted, these fibres spring from the cells of the abducens nucleus and ascend in the dorsal longitudinal bundle of the same or of the opposite side to the midbrain. There are, however, certain facts which controvert this view; in the first place the external rectus may be completely paralysed by a lesion that involves its nucleus though the opposite internal rectus is merely weak, and in the second place cases have been recorded of primary or secondary degeneration of all the cells of the abducens nucleus in which the opposite internal rectus acts normally in lateral movement of the eyes. Gerver, too, has found that disappearance

of all the abducens cells, as a result of the tearing out of one sixth nerve in animals, does not affect the functions of the opposite internal rectus. The second hypothesis that assumes the existence of a supranuclear centre in the neighbourhood of the nucleus of the sixth nerve is consequently more probably correct. There is now considerable evidence that this supranuclear centre lies oral and slightly ventral to the abducens nucleus. Into it come all the impulses that can excite conjugate movements of the eyes to the same side; voluntary impulses from the opposite frontal centre, impulses from the occipital and temporal lobes which effect reflex adjustments of the eyes to visual and auditory stimuli, others from the lower visual and auditory reflex systems in the roof of the midbrain, impressions of vestibular origin from Deiter's nucleus, and probably proprioceptive afferent impressions from the neck muscles. All impulses that can excite conjugate lateral movements of the eyes reach this centre, in it they are probably correlated and coordinated to produce an adequate response, and by efferent fibres from it the resultant is transmitted to the motor cells by which the corresponding internal and external recti muscles are innervated. It is probably through it too that the antagonists of the contracting muscles are reciprocally inhibited.

Destruction of this supranuclear mechanism consequently abolishes all voluntary and reflex lateral movements of the eyes to the same side. But these muscles are paralysed for conjugate lateral movement only; we can at least say this of the internal rectus, which still acts normally in convergence of the eyes, though it may fail to contract when lateral movement is attempted. It is less certain in the case of the external rectus, since we have a very limited power of conjugate divergence of the eyes, and since a lesion which destroys the supranuclear centre is liable to injure at the same time its adjacent nucleus. Therefore this supranuclear mechanism has the physiological property common to all motor mechanisms above the level of the final common paths in that it is concerned in the production of movements and not in exciting the contraction of individual muscles.

The various fibres of different function which converge in this supranuclear centre may be injured separately. Lesions of the lateral portion of the pons, for instance, may abolish the reflex turning of the eyes to labyrinthine impressions though the eyes may move normally to volitional and other impulses. But the most striking form of dissociation of lateral conjugate movement is that in which the voluntary deviation of the eyes is lost, but their reflex movements persist. This is presumably due to interruption of the cortico-bulbar fibres above the supranuclear centre, but the exact course that these take is not yet known. In this condition the patient may be unable to move his eyes to one side on command,

though he can follow a slowly moving object in this direction, or, as in one of my cases, his eyes may remain on a stationary object which he fixes when his head is passively turned in the other direction ; or his eyes may suddenly swing round in response to a sudden retinal or auditory stimulus. This isolated disturbance of voluntary lateral movement may be produced by cortical or subcortical lesions that destroy the cerebral ocular centres or their efferent fibres, but it is usually then incomplete and transient ; here, as is the case with the other cranial motor nerves, the paralysis is greater the nearer the injury of the upper motor neurons is to their nuclei.

If the opposite internal rectus receives its innervation for lateral conjugate movements through fibres which ascend the brain-stem from the region of the sixth nucleus, interruption of these connecting fibres should produce an isolated palsy of the internal rectus in lateral movement, though this muscle may still contract on convergence. I have seen such a paralysis of the internal rectus in conjugate lateral deviation only in two cases ; in both it contracted normally in convergence, and in both the symptoms made it probable that the dorsal longitudinal bundles were interrupted in front of the sixth nucleus, but the sites of the lesions could not be confirmed by autopsy.

Paralysis of the Vertical Conjugate Movements of the Eyes

Disturbances of the conjugate vertical movements of the eyes may be treated more briefly, as the physiological mechanisms which control them must be very similar to those of the lateral movements. It is not uncommon for neurologists at least to see loss or limitation, equal in degree in the two eyes, of either upward or downward rotation, or of both, with the lateral movements intact. Upward movements are most frequently affected, and next in order come paralysis of downward movement and of convergence, but all vertical movements of the eyes, with or without disturbance of convergence, are commonly abolished.

Such disturbances of the conjugate vertical movements have been explained by local lesions of the oculomotor nuclei, in which it is usually assumed that the separate ocular muscles are represented by separate groups of cells. Though this may be true, the divergent conclusions arrived at by the numerous authors who have attempted to identify muscular representation in these nuclei, as Hensen and Völker, Perlia, Bernheimer, Bach, Tsuchida and more recently Brouwer, show that we have no definite facts on which to rely. And it is scarcely conceivable that, as happens in the conjugate palsies of the vertical movements, a nuclear lesion could affect only the elevators or the depressors of the eyes, paralyse these to exactly the same degree on the two sides, and not affect the functions of the other muscles innervated by these nuclei. This explanation

becomes even more improbable when we remember that upward movement requires not only the coordinated cooperation of the two elevators of each eye, the two superior recti and the two inferior obliques, but also a parallel elevation of the lids by the levatores palpebrarum; and that the depressors, the inferior recti and the superior obliques, are innervated by two distinct nuclei separated by a relatively considerable space.

We are consequently, in my opinion, forced to the conclusion that, as in the case of the lateral conjugate rotators of the eyes, there are supranuclear mechanisms for upward and downward movements and for convergence, mechanisms which would in fact correspond to the associational cells intercalated between the spinal afferent reflex fibres and the descending spinal tracts on the one hand, and the motor cells of the ventral horns of the cord on the other.

But we are ignorant of the anatomical position of these supranuclear centres. According to one hypothesis they lie in the anterior quadrigeminal bodies; this is based partly on the statements of Adamük, Ferrier and others who elicited conjugate vertical movements by stimulation of this region, or observed loss of them after its destruction, and on the unquestionable fact that the vertical movements of the eyes are frequently disturbed by lesions of the tectum of the mid-brain. But in the great majority of the cases recorded the quadrigeminal lesions were tumours, which may affect indirectly by pressure either the third nuclei or centres in the tegmentum. It is in the tegmentum, or in the central grey matter around the aqueduct of Sylvius, that others would place the supranuclear centre. Freund, for instance, attributed loss of upward movement of the eyes to destruction of Darkschewitsch's nucleus, and the consequent degeneration of the lateral fibres of the dorsal longitudinal bundle, which was present in his case. Even after collating my own experiences with the literature on the subject, I am unable to come to a definite conclusion as to the position of these supranuclear mechanisms for the vertical movements, though I incline to the opinion that they are situated in the anterior quadrigeminal bodies. This opinion is based, in the first place, on the frequency of disturbance of the 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.

But, whatever may be the anatomical situation of these supranuclear mechanisms, we have considerable evidence of their relative positions, since tumours and other lesions extending from before backwards into the midbrain affect first upward movements of the eyes, then downward movements and finally convergence.

Consequently, we can assume that the centre for elevation lies the most anteriorly, and that for depression the furthest posteriorly. It is interesting in this connection that loss of upward deviation is usually associated with disturbances of the pupillary reactions to light, and that of downward movement frequently with loss of convergence and accommodation. There may be, however, an isolated loss of convergence and accommodation, as in two cases I have seen, and in those recorded by Eales, Parinaud, Stölting and Bruns.

The palsies of the vertical movements may be also dissociated; the most common form is loss of the voluntary movements with reflex movement unaffected. For instance, the patient may be unable to raise or lower his eyes from the horizontal level on command, or by a deliberate effort, but he may do so when a light is suddenly flashed above or below his visual axes, or his eyes may deviate upwards when he closes his lids, or these movements may be elicited by either asking him to follow a slowly moving object, or by bending his head backwards or forwards while his gaze is directed on a stationary object. These involuntary ocular movements evoked by passive movement of the head may represent labyrinthine reactions, but as Schüster found in one of his cases that they occurred only when the head was moved on the vertebral column and not when the head and body were displaced together, they may obviously be reflex adjustments excited by the proprioceptive afferents from the cervical muscles, similar to those which have been investigated by de Kleijn and Magnus. In these dissociated palsies we must assume the interruption in the neighbourhood of the supranuclear centre, of the cortico-bulbar fibres that terminate within it.

We are therefore led to the conclusion that there exists in the upper end of the midbrain, at a higher physiological level than the nuclei of the ocular nerves, a more complex associational or coordinating mechanism which receives all the impulses that can effect vertical and convergent conjugate movements of the eyes, combines and correlates them into an appropriate resultant, and distributes this to the nuclei of those muscles that should contract or relax.

Considerable attention has been directed to disturbances of the ocular movements in cerebellar disease, but they are relatively unimportant, even as clinical signs.

For some days after an acute unilateral injury of the cerebellum, the eyes, while at rest, are generally deviated towards the opposite side, especially if the patient happens to be unconscious, and it is often difficult to make him move them conjugately towards the injured side.

When he attempts this the range of movement is occasionally

incomplete, but usually a more striking feature is its slowness and the effort necessary to execute it. This conjugate paresis may be associated with erroneous projection towards the homolateral side. It diminishes gradually, but the difficulty in movement to this side, in comparison with deviation in the opposite direction, frequently persists for weeks. The vertical movements of the eyes and convergence are never similarly affected.

Occasionally the position known as "skew-deviation" is observed in patients with acute cerebellar lesions, that is the homolateral eye is directed downwards and inwards and the other upwards and outwards. This lack of parallelism in the optic axes disappears on fixation, when this can be obtained, and consequently diplopia does not result. It is produced only by extensive destruction of the cerebellum, and has not, in my opinion, any localizing value.

OCULAR PALSIES*

BY

LESLIE PATON

LONDON.

THE subject of ocular palsies is one of great complexity, and in its consideration the relationship of the oculomotor mechanism to many different parts of the central nervous system has to be taken into account. If we take only one disease, syphilis, and consider the different ways in which it can produce an ocular palsy, it will enable us to realize dimly the complexity of the subject. Syphilis may affect directly any part of the oculomotor apparatus, cortical, supra-nuclear, nuclear, nerve roots, nerve trunks in their intra-cranial course, in the cavernous sinus, or in the orbit, or it may affect the muscles themselves. A gumma may develop at any point. A syphilitic meningitis may affect the nerves. Syphilis sets up disease of the vessels and we may get haemorrhages affecting the nuclei or nerve roots, or a syphilitic arteritis in the internal carotid may press on nerve trunks in the wall of the cavernous sinus, or an endarteritis may affect the vessels supplying the crura, the mid brain or the pons, and give rise to areas of softening; or syphilis may give rise to tabes or general paralysis, and to all the variety of ocular disturbances arising in these diseases. Yet it is doubtful if more than 40 per cent. of cases of ophthalmoplegia are due to syphilis, directly or indirectly.

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