Patterns of eye movements and their clinical evaluation.

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CONJUGATED EYEMOTOR DISTURBANCES REFLECTING
BRAIN STEM LESIONS.

Neurotology includes analysis of not only vestibular eyemovements but also voluntary, concomitant as well as pursuit and optokinetic eyemovements. By these kind of tests the information is not restricted to the peripheral labyrinths, but is extended also to central oculomotor pathways. The analysis of the complex eyemovements and varying combinations of irregularities is difficult to analyse and the interpretation is sometimes even impossible. A main obstacle for a meaningful analysis of eyemovements is a lack of proper understanding or how eyemotor impulses are treated in the brain stem. Therefore will be made on the basis of clinical observations of eyemovements an effort to outline a model of the functional organization in the brain stem for conjugated eyemovements.

Impulses initiating eyemovements are fed into the mesencephalic and pontine retine reticular formation from frontal and occipital cortex as well as from cerebellum and vestibular receptors in the labyrinths.

Before reaching nuclei in the brain stem a complex processing, different functional purposes, seems to take place. We will start by demonstrating the anticipated organization of this processing by presenting by block diagramme (fig. 1.) based on seven different patterns of eyemotor disturbances which are to be accounted for in this paper.

The disturbances will be related to one, some or all of the following functional concepts: (fig. 1.)

- I. Structures located in the paramedian portions of the brain stem are relaying impulses for all fast eyemovements; for voluntary saccades, for fast phases of vestibular and optic nystagmus and also for with active head turnings concomitant eyemovements. This centre might be identical with the pontine gaze centre.
- II. Presumably cerebellar structures engaged in reciprocal inhibition and facilitation of fast and slow phases in pursuit eyemovements.
- III. Presumably pontine structures involved in reciprocal inhibition and facilitation of fast and slow phases of vestibular and optic nystagmus.
- IV. Structures controlling optic slow phases (pursuit eyemovements).
- V. Structures controlling vestibular slow phases.
- VI. Vestibular impulses reaching the vestibular nuclei from the vestibular nerve.
- VII. Central vestibular spinal pathways.

RECORDINGS:

1. Eyespeed recording of horizontal eyemovement.
2. Conventional DC-recording of horizontal eyemovements.
3. Conventional DC-recording of vertical eyemovements.
4. Vestibulo-spinal recordings.
5. Recordings of the position of the head at head turnings.

STIMULATION OF EYEMOVEMENTS: (fig. 2.)

1. Voluntary to and fro movements of 80°.
2. Concomitant eyemovements at voluntary turning of the head (45°) in the horizontal plane.
3. Pursuit eyemovements following a figure (1,2,3,4) travelling at 20°/sec. on a circular screen one metre in front of the patient.

4. Caloric tests by using water of 30° and 44° C during 30 seconds (eyes open in darkness).
5. Angular acceleration of 60°/sec.² for 2 seconds (eyes open in darkness).
- (6). Exposure to an optokinetic drum one metre in front of the subject covering the total visual field.

RESULTS:

PARALYSIS OF FAST GAZE CENTRE

Disturbance of the centre for fast eyemovements (paramedian pontine gaze centre).

Case 1. Male, 54 years of age. Supra nuclear palsy in a case with Steele-Richardson-Olszewski Syndrome.

Already the calibration reveals pathological findings. The patient is unable to produce rapid eyemovements and performs instead slow, almost sinusoidal eyemovements (fig. 3). The normal concomitant eyemovements at head turnings are replaced by deviation of the eyes opposite to the turning of the head. The pursuit eyemovements are relatively well preserved. The vestibular tests (caloric and rotatory) demonstrate deviation in direction of slow component almost without any fast phases. This case thus shows: Abolishment of voluntary fast eyemovements, of normal rapid concomitant eyemovements and of the fast phases of vestibular as well as of optokinetic nystagmus, while slow eyemovements of both optic and vestibular origin seem to be preserved. In conclusion, all fast eyemovements, saccades and fast nystagmus phases seem at least at one level to use identical pathways, while slow eyemovements may use others.

CEREBELLAR SACCADDES

Disturbance of slow and fast phase relation in pursuit eyemovements (cerebellar lesions?).

Fig. 1

Fig. 2

Case 2. Male, 51 years of age. Multiple sclerosis.

A case with a pronounced inability for pursuit eyemovements with regular saccades is demonstrated. The rhythmic vestibular nystagmus indicates a normal inhibiting mechanism between vestibular slow and fast phase (fig. 4). The difference between the optic and vestibular patterns may be explained by lesions in the cerebellum affecting the optic pathways or resulting in a defect cerebellar control of interrelation between fast and slow optic centres in the brain stem. The normal vestibular nystagmus patterns in this case indicates at least a non pontine location of the disorder.

Fig. 3 PARALYSIS OF FAST GAZE CENTRE

Fig. 4 CEREBELLAR SACCADDES

PONTINE SACCADING :

Dysrhythmic vestibular nystagmus in combination with irregular pursuit eyemovements.

Case 3. Male, 71 years of age. Ischemia of the brain stem.

The dysrhythmic vestibular nystagmus pattern presented by this patient is frequently found in combination with irregular saccades in pursuit eyemovements (fig. 5). This may be interpreted as due to a brain stem lesion affecting the interrelation between saccadic centre and of slow phase centres of both optic and vestibular systems.

Fig.5 PONTINE SACCADDES

Fig.6 SLOW PHASE PARALYSIS

SLOW PHASE PARALYSIS

Loss of pursuit eyemovements as well as of vestibular slow.

Case 4. Female, 74 years of age. Mesencephalopathy, brain stem ischemia.

In this case pursuit eyemovements and vestibular nystagmus could not be produced, while the vestibulo-spinal reflexes were normal and the saccadic centre seem to be only partially damaged (fig. 6). Concomitant eyemovements were present, but not to a normal degree.

TOTAL BILATERAL OPHTHALMOPLEGIA WITH PRESERVATION OF VESTIBULOSPINAL RESPONSES.

Case 5. Male, 46 years of age. Multiple sclerosis.

In this case no eyemovements could be recorded (fig. 7). The presence of normal vestibulo-spinal reflexes indicated a location of the lesions restricted to structures above the vestibular nuclei.

TOTAL OPHTHALMOPLEGIA IN COMBINATION WITH A LOSS OF VESTIBULOSPINAL RES-

PONSES.

Case 6. Female, 48 years of age. Wernicke's disease.

In this case a complete loss of all eyemovements in combination with a loss of vestibulo-spinal reflexes indicate a larger topographical involvement than caused by the disorder in the previous case. (fig 8). Other clinical signs and symptoms also indicated in this last case a more serious condition.

VESTIBULAR AREFLEXIA

Case 7. Female, 46 years of age. A vestibular areflexia due to ototoxic lesions.

In this case is demonstrated a complete loss of vestibular reflexes. Both nystagmus responses as well as vestibulospinal responses were absent due to bilateral peripheral labyrinthine lesions. This case demonstrates normal calibration eyemovements, normal pursuit eyemovements and normal concomitant eyemovements in spite of the complete labyrinthine areflexia.

Discussion :

The selective abolishment of all types of fast phases with preservation of slow eyemovements found in the first case described in this paper indicates that pathways for all fast eyemovements seem to converge in one location which is separate from pathways for slow eyemovements. Such patterns are described as Dell's head phenomenon referring to the turning of the doll's eye opposite to that of the turning of the doll's head. The pattern of eyemovements in this group of patients shows striking similarities to doll's eyemovements. This phenomenon is in itself a support of the conception of different pathways for slow and fast eyemovements first advocated by Fox and Holmes (1926).

As the lesion causing this abolishment did not produce a disconjugate pattern of eyemovements, the lesion must be located above the level of eyemotor nuclei and also above the coordinating pathways of the medial longitudinal fasciculus. It may be assumed that this center of fast eyemovements corresponds to the paramedian pontine gaze centre in the rhesus monkey extensively studied among others also by Cohen (1968).

As a further proof of such separate location serves the finding in our case number n:r 4 four, in

which the slow phases of vestibular and of optic nystagmus were severely impaired with a relative preservation of fast eyemovements.

The observation of totally different effect on slow and fast phases of nystagmus by tobacco smoking is another evidence for separated slow and fast centres.

The question whether slow phases of vestibular and optic nystagmus is initiated in the same structures cannot yet be regarded as settled. The same centre for the two types of eyemovements might be expected as the slow phases of optic as well as of vestibular nystagmus frequently serve the same purpose of having the eyes keep a visual target fixed on the retina. Optic as well as vestibular centres are at work for instance at optic fixation during head turning. Further, the vestibular as well as the optic slow phase centre must during their working periods constantly inhibit the fast phases.

The pursuit eyemovements in case number 2 showed regular saccades of cerebellar type while, the vestibular nystagmus quieted normal and quite rhythmic. As this pattern is frequently met with in cerebellar disorders the pattern has been interpreted as possibly due to a disturbance of cerebellar remote control of pontine reciprocal inhibition of fast and slow optic phases. The arrangement according to fig. 1 with a cerebellar control of optic slow and fast phases but not of the corresponding vestibular parameters would be in conformance with this idea.

The disturbance in case number three of the nystagmus rhythm of the optic as well as of the vestibular nystagmus has been interpreted as due to essentially pontine lesions. The disturbances are assumed to affect the reciprocal inhibition between the fast phase and the slow phase centre(s) of optic as well as of vestibular nystagmus. Ischemia of the brain stem without symptoms and signs from cerebellum may be responsible for these patterns.

Case number 5 shows a total bilateral ophthalmoplegia with remaining vestibulo-spinal responses. And case number 6 shows the same kind of bilateral ophthalmoplegia but without vestibulo-spinal responses. This loss of vestibulo-spinal responses indicates a wider brain stem engagement and this was also in accordance with the more serious clinical picture in that last patient. The lesions causing complete ophthalmoplegia have tributed to defects in eyemotor nuclei, although paralysis of fast and slow gaze centres above the level of eyemotor nuclei could be held responsible for this clinical picture.

In the last case is demonstrated the pattern of eyemovements in a patient who has vestibular areflexia in which neither vestibulo-spinal nor vestibulo-ocular responses were produced, while the voluntary and the pursuit eyemovements were quite normal. This may not be interpreted as speaking in favour of two slow phase centres. A common centre for both vestibular and optic eyemovements could still be considered as the same centre could well be brought in action from two different sources.

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