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Divergence paralysis with raised intracranial pressure

An electro-oculographic study

T. H. KIRKHAM, A. C. BIRD, AND M. D. SANDERS

The National Hospital, Queen Square, London, W.C.1

Divergence paralysis is characterized by acquired horizontal homonymous diplopia when viewing distant objects but without limitation of ocular movements (Bielschowsky, 1935). Although raised intracranial pressure is a significant aetiological factor in the production of divergence paralysis (Bender and Savitsky, 1940; Chamlin and Davidoff, 1950, 1951), there has been much diversity of opinion as to the underlying mechanism.

The present report describes three patients with divergence paralysis in whom electro-oculography was used to measure peak angular velocities during horizontal ocular saccades.

The apparatus used for electro-oculography has been previously described (Hallpike, Hood, and Trinder, 1960; Hood, 1968).

Case reports

Case 1, a 9-year-old girl, was admitted to the National Hospital under the care of Prof. John Marshall, with the complaint of horizontal diplopia of 10 days' duration when viewing distant objects. She had suffered occasional nausea in the morning over the previous year but without headaches. A few hours before the onset of diplopia she had fallen at school and struck her head.

Examination

She was a normally developed child who held her head turned to the right with the chin depressed on the chest. The tendon reflexes were brisk and the left plantar response was extensor.

The visual acuity was 6/6 with each eye, perimetric examination showed enlarged blind spots, and the pupillary responses were normal. Fundus examination showed bilateral chronic papilloedema.

Ocular movements

She had a right convergent strabismus but the range of ocular movements was full. Homonymous diplopia was present beyond 9 cm. and increasing distance fixation produced wider separation of the images although fusion was possible for distant objects on extreme lateral gaze. Prism cover test showed 14$^{\triangle}$ esophoria at 9 cm. and 40$^{\triangle}$ esotropia at 6 m. The deviation was decreased on depression of the chin and increased on elevation of the chin. There was no nystagmus and the optokinetic responses were normal. The Hess chart showed a convergent deviation (Figure, opposite).

Skull

X-rays and the brain scan were normal and, in view of the history of head trauma, biparietal burr holes were made to exclude the presence of subdural haematoma; the brain was found to be tense and the cerebrospinal fluid in the lateral ventricles was under moderately elevated pressure.

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Address for reprints: Dr. T. H. Kirkham, The National Hospital, Queen Square, London, W.C.1

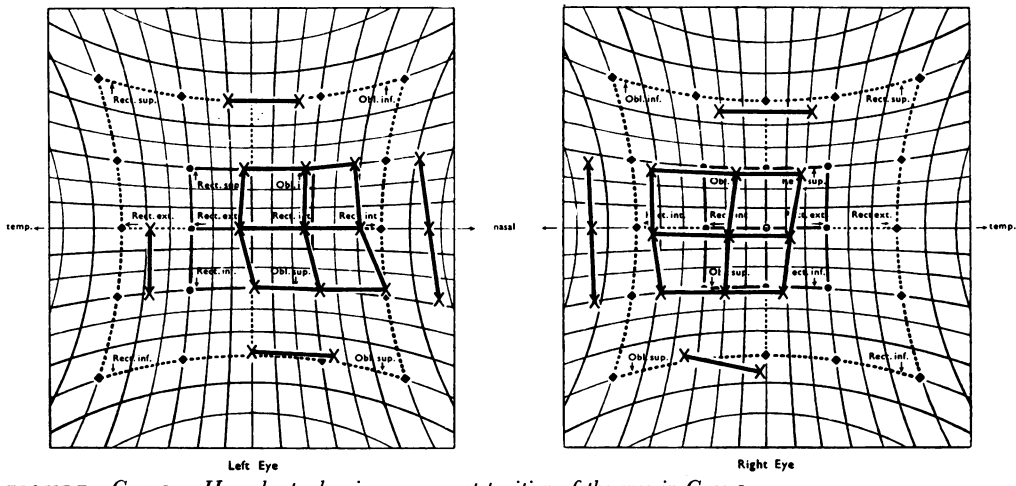

FIGURE *Case* 1. *Hess chart, showing convergent position of the eyes in Case* 1

Pneumo-ventriculography was performed and showed normal sized ventricles with no shift of the midline structures.

Treatment

A provisional diagnosis of benign intracranial hypertension was made and the patient was treated with dexamethazone 1 mg. 6 hrly. Lumbar puncture 2 days later showed an opening pressure of 155 m. of CSF, and dexamethazone was reduced to 1 mg. daily.

Course

After 3 days the diplopia suddenly resolved; ophthalmic examination showed 6△ of esophoria to be present for distance fixation.

Electro-oculography was performed whilst the patient had diplopia and peak angular velocities of eye movements were measured during 30° saccades on either side of the midline target and two points. Average peak angular velocities in degrees per second were:

		Right eye	*Left eye*
SACCADES TO RIGHT	Left to midline	310	495
	Midline to right	295	460
SACCADES TO LEFT	Right to midline	466	300
	Midline to left	480	285

These results show that abduction velocity of both eyes was abnormally slow. Recordings were repeated immediately after recovery of single vision and the peak velocities were:

		Right eye	*Left eye*
SACCADES TO RIGHT	Left to midline	405	530
	Midline to right	410	520
SACCADES TO LEFT	Right to midline	530	470
	Midline to left	485	455

These results show that abduction of the right eye was still slow but that abduction of the left eye had recovered. Electro-oculography was repeated after an interval of 2 months when the papill-oedema had completely resolved; the findings were:

		Right eye	*Left eye*
SACCADES TO RIGHT	Left to midline	476	490
	Midline to right	457	450
SACCADES TO RIGHT	Right to midline	440	443
	Midline to left	438	430

Thus the peak velocity of abduction of both eyes was now fully recovered.

Comment

A 9-year-old girl with benign intracranial hypertension presented with divergence paralysis. Electro-oculography showed that peak velocity of eye movements during abduction was abnormally slow. After recovery of binocular single vision there was at first recovery of abduction of one eye, and 2 months later abduction velocity of each eye was normal.

Case 2, a 20-year-old woman, was admitted to the National Hospital under the care of Prof. Valentine Logue, with the complaint of horizontal diplopia for 24 hrs. She had experienced a severe throbbing biparietal headache 3 weeks previously accompanied by nausea and vomiting which lasted for 3 days. On the day before admission she again suffered a severe headache with vomiting, which was accompanied by the sudden onset of horizontal diplopia on distance fixation; she could read without difficulty. She denied any head injury or antecedent illness and was not receiving any medication.

Examination

She was a cheerful obese girl weighing 80 kg. Neurological examination was normal. Corrected visual acuity was 6/6 in each eye, perimetry showed enlarged blind spots, and there was marked bilateral chronic papilloedema with numerous haemorrhages and nerve fibre infarcts.

Ocular movements

She had homonymous diplopia in the primary position at all distances beyond 40 cm. which increased on distance fixation. The diplopia diminished on extreme lateral gaze, increased with chin elevation, and decreased with chin depression. Prism cover test showed $8^{\triangle}$ esophoria at $\frac{1}{3}$ m. and $23^{\triangle}$ esotropia at 6 m. The ocular movements were full; there was no nystagmus and the optokinetic responses were normal.

Investigations

Skull *x* rays were normal and a brain scan showed an increased uptake on the left side suggestive of a subdural haematoma, the presence of which was confirmed by carotid angiography.

Treatment

The haematoma was evacuated, and the patient made a good recovery with gradual resolution of the papilloedema.

Course

21 days postoperatively the diplopia suddenly resolved. The ocular movements were normal and cover test showed a slight exophoria for both near and distance fixation.

Electro-oculography performed immediately after recovery of single vision during saccades between points 30° to either side of the midline and a point straight ahead gave the following results:

		Right eye	*Left eye*
SACCADES TO RIGHT	Left to midline	490	550
	Midline to right	460	540

SACCADES TO LEFT	Right to midline	520	515
	Midline to left	505	420

These results show that peak velocities of the abducting eye were slower than those of the adducting eye particularly during abduction beyond the midline.

The recordings were repeated 6 weeks later and showed that peak abduction velocity in each eye had now fully recovered.

		Right eye	*Left eye*
SACCADES TO RIGHT	Left to midline	480	500
	Midline to right	480	510
SACCADES TO LEFT	Right to midline	505	490
	Midline to left	500	480

Comment

A 20-year-old woman had divergence paralysis associated with raised intracranial pressure due to a subdural haematoma. Peak velocity movements during horizontal saccades showed that immediately after recovery of single vision abduction was slower than adduction particularly after the eyes had crossed the midline. Full recovery was demonstrated 6 weeks later.

Case 3, a 16-year-old boy, was admitted to the National Hospital under the care of Prof. Valentine Logue. He had been involved in a motorcycle accident 3 weeks previously and had struck the right side of his head. He had amnesia for the incident and for 5 days afterwards. 1 week after the accident on waking he noticed constant diplopia for distant objects. He did not suffer from headaches.

Examination

Abnormal neurological findings were a mild left hemiparesis and bilateral palmo-mental reflexes.

The visual acuity was 6/5 with each eye. Perimetry showed the presence of enlarged blind spots and the pupillary reactions were normal. Bilateral papilloedema was present.

Ocular movements

A right convergent squint of $14^{\triangle}$ was present on distance fixation and single vision was obtained at 50 cm. Homonymous diplopia was present beyond this point which did not increase on lateral gaze. The diplopia was more marked on elevation of the chin and single vision was obtained on depression of the chin. The range of ocular movements was full and abduction of each eye exceeded adduction when measured perimetrically. A Hess chart showed a convergent position of the eyes, but no muscle overaction or underaction.

Investigations

Skull *x*-rays showed a long linear fracture on the right side of the vault extending into the floor of the anterior fossa, which traversed the middle meningeal vascular groove. Left carotid angiography showed that the anterior cerebral artery was shifted 12 mm. to the left and the internal cerebral vein 3 mm. to the left. Right carotid angiography showed there to be a large extracerebral accumulation in the region of the right frontal lobe.

Treatment

A right frontal craniotomy was performed and a large extradural haematoma was evacuated.

Course

The diplopia suddenly resolved 10 days after the operation.

Electro-oculography was performed 2 days after surgery during horizontal saccades between two points 30° to each side of the midline and a point straight ahead. The average peak velocities were:

		Right eye	*Left eye*
SACCADES TO RIGHT	Left to midline	456	592
	Midline to right	356	540
SACCADES TO LEFT	Right to midline	590	475
	Midline to left	560	350

These results show reduction of the peak angular velocities of the abducting eye particularly during abduction beyond the midline.

Comment

A 16-year-old boy had divergence paralysis as the presenting sign of raised intracranial pressure due to an extradural haematoma. Electro-oculography showed reduction of the peak velocity of the abducting eye particularly during abduction beyond the midline.

Discussion

The features of divergence paralysis were summarized by Bielschowsky (1935). It is characterized by a convergent squint with diplopia when viewing a distant object but without limitation of ocular movement. The diplopia is homonymous and increasing distance fixation produces wider separation of the images. Single vision is usually obtained at about 20 to 30 cm. although heteronymous diplopia may occur at distances nearer than 30 cm. on account of an associated convergence deficiency. Single vision may be obtained on extreme lateral gaze. The separation of the images increases on looking downwards and decreases on looking upwards; the patient therefore adopts an abnormal head posture with the chin depressed in order to utilize the physiological divergence on elevation to minimize the diplopia. Divergence paralysis may be difficult to differentiate from spasm of the near reflex, bilateral sixth nerve palsies, and esophoria.

There has been considerable speculation regarding the site of a specific divergence centre, a lesion of which might be responsible for the feature of divergence paralysis. Leber (1903) thought that divergence resulted simply from a relaxation of convergence tone and Berry (1901) suggested that cases reported with divergence paralysis were suffering from spasm of the near reflex. Duane (1905), Bielschowsky (1935), and Bruce (1935) thought it inconceivable that convergence would be a neural mechanism without an antagonistic system and independently proposed a brain-stem centre for divergence near the abducens nuclei. Duane (1905) thought that a lesion of this brain-stem centre was responsible for divergence paralysis. That divergence is a positive act has been supported by electromyographic studies (Adler, 1953; Breinin and Moldaver, 1955). Divergence was produced by cortical stimulation in monkeys by Bender and Savitsky (1940), but this observation was not confirmed by Jampel (1959), and Warwick (1955) did not think it profitable to attempt to separate discrete cortical centres for convergence or divergence.

Although divergence paralysis has been described in patients with tumours of the posterior fossa (Bender and Savitsky, 1940; Robbins, 1941; Lippmann, 1944; Savitsky and Madonick, 1946; Chamlin and Davidoff, 1950; Møller, 1970), it has also been described with supratentorial subdural haematomas (Chamlin and Davidoff, 1950), cerebral tumours (Møller, 1970; Anderson and Lubow, 1970), and benign intracranial hypertension (Chamlin and Davidoff, 1950). Local involvement of a specific divergence mechanism by the primary lesion cannot therefore account for divergence paralysis, and it was recognized by Bender and Savitsky (1940) and Chamlin and Davidoff (1950, 1951) that raised intracranial pressure was the most important common factor in patients with divergence paralysis.

The association between lateral rectus palsy and raised intracranial pressure is well documented and the clinical correlation has been clearly demonstrated by Lundberg (1960) and Van Allen (1967). The mechanism by which raised intracranial pressure produces lateral rectus palsy is not well understood and it has generally been assumed that the peripheral portion of the sixth nerve is compressed against adjacent structures or stretched as a result of tentorial herniation.

Our patients with divergence paralysis had raised intracranial pressure and reduction of the pressure was followed by restoration of single vision. Electro-oculography in cases in which divergence paralysis was present showed that peak abduction velocities during horizontal saccades were markedly reduced. Soon after recovery of single vision, the abduction velocities were still slow, particularly during the second half of the saccade, and that recovery of abduction velocity was asymmetrical. These findings suggest that impaired abduction is present in cases of divergence paralysis despite the absence of other signs of lateral rectus palsy. All our patients had a full range of abduction, and abduction was greater than adduction when measured on the perimeter—fulfilling the criteria of Bedrossian (1958). There was lessening of diplopia on lateral gaze and no overaction of the medial recti was shown on the Hess chart. This abduction paresis may be due to a supranuclear lesion in the region of the sixth nerve nuclei or to a nuclear or infranuclear lesion of the sixth nerves. Abnormalities of ocular movement may be manifest not only by a limited range of movement but also by a reduction in the speed of the movement; such a situation has been demonstrated in patients with internuclear ophthalmoplegia (Smith and David, 1964; Bird and Sanders, 1970) and lateral rectus palsies (Metz, Scott, O'Meara, and Stewart, 1970).

We believe that there may be involvement of the sixth cranial nerve, probably nuclear or infranuclear, in patients with divergence paralysis and that the asymmetry of recovery perhaps strengthens this impression. The speed of onset of divergence paralysis and its sudden recovery is interesting and may best be explained on the basis of vascular insufficiency. The sixth nerve nuclei lie in an area of the brain stem which is supplied by terminal branches of the circumflex and paramedian perforating branches of the basilar artery. With increased intracranial pressure it is conceivable that a critical perfusion level is reached such that divergence paralysis suddenly appears, and that on reduction of intracranial pressure single vision is restored when perfusion of the nuclei exceeds this critical level.

Some support for this idea may be adduced from the work of Johnson and Yates (1956) with regard to the development of midbrain signs in patients with supratentorial subdural haematoma. However, divergence paralysis has not been reported in patients with vertebral-basilar insufficiency syndromes (Williams and Wilson, 1962; Bradshaw and McQuaid, 1963) and the selectivity of the syndrome without other apparent brain-stem dysfunction perhaps argues against the concept of a critical perfusion level in the region of the sixth nerve nuclei. The similarity of the ocular findings in all reported cases may suggest some simpler mechanism such as compression of the peripheral portions of the sixth nerves against adjacent structures with consequent impaired perfusion as a non-specific consequence of raised intracranial pressure.

Conclusion

Three patients with raised intracranial pressure developed the classical features of divergence paralysis. Peak eye movement velocities during horizontal saccades measured by electro-oculography showed reduced abduction velocities. It is suggesetd that minimal

interference of sixth nerve function by raised intracranial pressure may produce the features of divergence paralysis without other evidence of sixth nerve palsy and without the need to invoke a specific lesion of a divergence mechanism. Relative ischaemia of the sixth nerves at either nuclear or infranuclear level may be the explanation for the sudden onset of divergence paralysis and for its sudden disappearance when the intracranial pressure is returned to normal.

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