Temporary Divergence Paralysis in Viral Meningitis

Stef L. M. Bakker, MD, PhD and Ivan M. Gan, MD

Abstract: A 43-year-old woman who reported diplopia and headache was found to have comitant esotropia at distance fixation and normal alignment at reading distance (divergence paralysis). Eye movement, including abduction, was normal as was the rest of the neurologic examination. Brain MRI was normal. Lumbar puncture showed an elevated opening pressure and a cerebrospinal fluid formula consistent with viral meningitis. The patient was treated with intravenous fluids and analgesics and with a temporary prism to alleviate diplopia. Within 3 weeks, she had fully recovered. This is the first report of divergence palsy in viral meningitis.

(*J Neuro-Ophthalmol* 2008;28:111–113)

Divergence paralysis in the absence of other brainstem signs is a rare clinical sign characterized by esotropia at distance fixation with normal abduction and no ocular misalignment at near fixation. Acute onset of diplopia for increasing distances is the only complaint. Diplopia due to paralysis of divergence has been described in several neurologic diseases but not in meningitis (1–14). We describe a patient with impairment of divergence during a bout of viral meningitis who had a full recovery.

CASE REPORT

A 43-year-old woman was admitted because of severe headache and vomiting for 5 days and diplopia at distance for 4 days. She had a history of migraine but used no medication. There was no history of recent head trauma.

Blood pressure was 150/85 mm Hg, pulse was 60 per minute, and temperature was 35.8°C. Neurologic examination was normal. There were no meningeal signs. Consciousness was not impaired and attention, orientation, and concentration were normal. Uncorrected visual acuity was 20/20 in each eye. There was no refractive error and no history of ocular surgery. Pupils were equal in size with normal constriction to direct light. There was orthophoria at 14 cm; fixation at 30 cm showed a comitant esotropia of 30 prism diopters, which increased to 37 prism diopters at 6 m; ocular movements were normal with full abduction of either eye (Fig. 1). There was no nystagmus. Ophthalmoscopy showed no papilledema or retinal abnormalities.

Brain MRI showed no abnormalities. Lumbar puncture showed an opening pressure of 290 mm H_2O, 1,185 white blood cells/mL (98% monomorphonuclear leukocytes and 2% polymorphonuclear leukocytes), protein of 1.86 g/L and glucose of 2.3 mmol/L (normal = 2.2 –4.4 mmol/L). Gram stain results were negative. Cytologic examination of the cerebrospinal fluid showed no signs of malignant cells. The peripheral white cell count was 10,500 with normal differential. C-reactive protein level was 1 mg/L, and sedimentation rate was 7 mm/hr.

The patient received a diagnosis of nonspecific viral meningitis and was treated with intravenous fluids and analgesics. During the hospital admission, her headache slowly decreased. Diplopia at distance was palliated with a Fresnel prism of 30 prism diopters for 3 weeks. When the prism glasses were not in place, the patient reported diplopia with unvarying image separation for 2 weeks. At a 3-week follow-up visit, there were no signs of esotropia at near or distance fixation (Fig. 2).

DISCUSSION

Our patient showed all the clinical features of divergence paralysis, a rare clinical sign first described by Parinaud in 1883 (15). Criteria for this diagnosis include sudden onset of uncrossed horizontal diplopia at distance fixation with fusion at near, comitance of strabismus in all fields of gaze, normal ocular ductions and versions, and absence or marked reduced fusional divergence (16).

Divergence paralysis has been described in patients with encephalitis (1), spinocerebellar ataxia (2), neurobrucellosis (3), use of diazepam (4), brainstem ischemia (5), brainstem hematoma (6), Fisher syndrome (7), syphilis (8), multiple sclerosis (9), pontomedullary glioma (10), raised intracranial pressure (11,12), head trauma (13),

Departments of Neurology (SLMB) and Ophthalmology (IMG), St. Franciscus General Hospital, Rotterdam, The Netherlands; and Department of Ophthalmology (IMG), The Rotterdam Eye Hospital, Rotterdam, The Netherlands.

Address correspondence to S. L. M. Bakker, MD, PhD, Department of Neurology, St. Franciscus General Hospital Rotterdam, Kleiweg 500, 3045 PM Rottterdam, The Netherlands; E-mail: s.bakker@sfg.nl

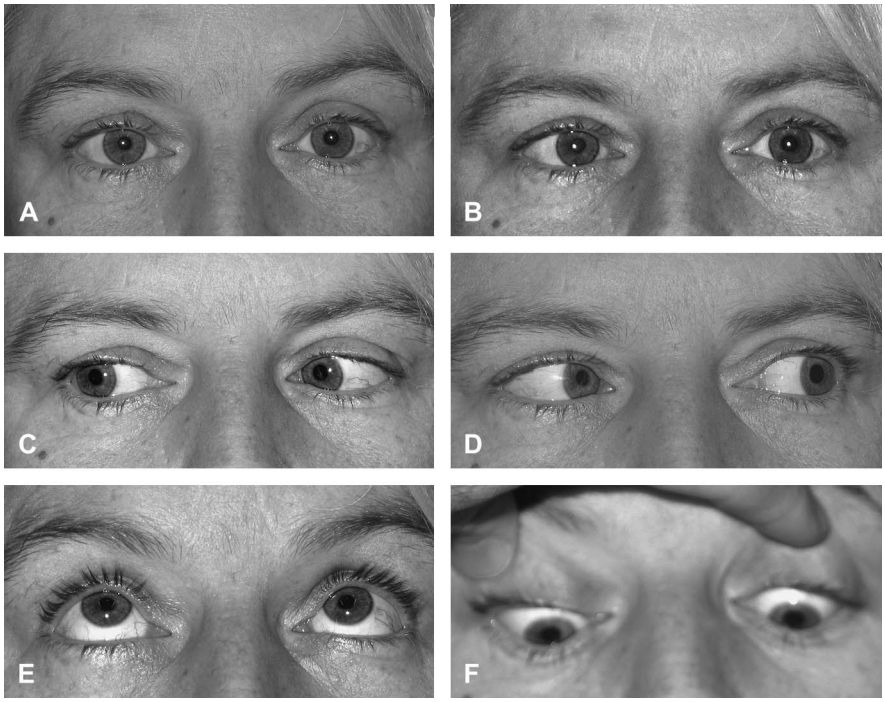

FIG. 1. Ocular alignment at presentation. There is orthophoria at near (14 cm) fixation (**A**) and 37 prism diopters of esotropia at distance (6 m) fixation (**B**). Ocular horizontal and vertical pursuit versions are normal (**C**-**F**).

progressive supranuclear palsy, and seizure disorder (14). Viral meningitis has not previously been described as a cause of divergence paralysis.

The duration of the divergence palsy in the reversible causes ranges from hours after withdrawing diazepam to days after treatment of raised intracranial pressure, 8 weeks in Fisher syndrome, and several months after presumed brainstem ischemia. A specific anatomic site for a divergence center remains speculative; most documented clinical cases relate to brainstem lesions (5,6,9,10). Neuroradiologic support for the supposed anatomic localization of the divergence center in the brainstem has been acquired in a patient with a small hematoma in the tegmentum of the upper pons (6) and in a pontine glioma that extended to the upper medulla (10), both in the absence of hydrocephalus. Localization of a specific divergence site in the brainstem has received neurophysiologic support from Mays (17), who demonstrated discharge from divergence neurons, found to be intermixed with convergence neurons, in the mesencephalic reticular formation just dorsal or dorsolateral to the oculomotor nucleus in monkeys during divergence movements. This observation supports the idea that divergence is a positive act in contrast to just relaxation of convergence tone. However, it remains difficult to understand the selectivity of the syndrome of divergence palsy without other apparent brainstem dysfunction. Raised intracranial pressure is thought to be an important common factor in divergence palsy, both in isolation as in the presence of space-occupying lesions in the posterior fossa and cerebrum (12).

Divergence palsy must be differentiated from other conditions that cause a comitant esotropia at distance. These include convergence spasm, bilateral sixth nerve palsies, decompensated esophoria, and early acquired restrictive extraocular myopathies such as thyroid eye disease. In spasm of the near reflex, the esotropia is generally variable, and there is an associated pupillary miosis and accommodative spasm. Incomitant strabismus characterizes sixth nerve palsy. Decompensated esophoria

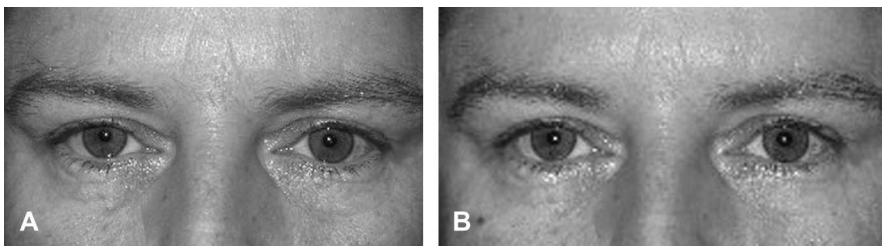

FIG. 2. Ocular alignment 3 weeks after presentation. There is orthophoria at near (**A**) and distance (**B**) fixation.

usually causes an esotropia that is of the same degree at distance and near fixation. Restrictive ocular diseases may be differentiated by forced duction testing.

Early sixth nerve palsies are often considered to be responsible for an apparent divergence palsy when they are associated with increased intracranial pressure (11). This "pseudo-divergence paralysis" should resolve when intracranial pressure returns to normal. At time of presentation, our patient had increased intracranial pressure, and this might have played a role in causing temporary divergence paralysis in our patient. The acute onset and the absence of papilledema and end-point nystagmus, however, probably negate the idea that sixth nerve palsies due to increased intracranial pressure caused divergence palsy in our patient.

There are no prospective data about prognosis of isolated divergence paralysis, but in the cases described no sequelae have been seen. Diplopia is normally relieved with prism therapy, and only rarely is extraocular muscle surgery required. Bilateral lateral rectus resections of 5–6 mm have been reported to be satisfactory (14). Our patient completely recovered from the divergence paralysis as the meningitis resolved.

REFERENCES

1. Dunnington JH. Paralysis of divergence with report of three cases due to epidemic encephalitis. *Arch Ophthalmol* 1923;52:39–4.
2. Ohyagi Y, Yamada T, Okayama A, et al. Vergence disorders in patients with spinocerebellar ataxia 3/Machado-Joseph disease: a synoptophore study. *J Neurol Sci* 2000;173:120–3.
3. Tekeli O, Tomaç S, Gürsel E, et al. Divergence paralysis and intracranial hypertension due to neurobrucellosis: a case report. *Binocul Vis Strabismus Q* 1999;14:117–8.
4. Arai M, Fujii S. Divergence paralysis associated with the ingestion of diazepam. *J Neurol* 1990;237:45–6.
5. Cunningham RD. Divergence paralysis. *Am J Ophthalmol* 1972;74: 630–3.
6. Lee SA, Sunwoo IN, Kim KW. Divergence paralysis due to a small hematoma in the tegmentum of the brainstem. *Yonsei Med J* 1987;28: 326–8.
7. Pinchoff BS, Slavin ML, Rosenstein D, et al. Divergence paralysis as the initial sign in the Miller Fisher syndrome. *Am J Ophthalmol* 1986; 101:741–2.
8. Alger EM. Paralysis of divergence. *Trans Am Ophthalmol Soc* 1916; 14:665–77.
9. Dunphy EB. Paralysis of divergence. *Am J Ophthalmol* 1928;11: 298–9.
10. Schanzer B, Bordaberry M, Jeffery AR, et al. The child with divergence paresis. *Surv Ophthalmol* 1998;42:571–6.
11. Kirkham TH, Bird AC, Sanders MD. Divergence paralysis with raised intracranial pressure: an electro-oculographic study. *Br J Ophthalmol* 1972;56:776–82.
12. Chamlin M, Davidoff LM. Divergence paralysis with increased intracranial pressure. *J Neurosurg* 1950;7:539–43.
13. Rutkowski PC, Burian HM. Divergence paralysis following head trauma. *Am J Ophthalmol* 1972;73:660–2.
14. Krohel GB, Tobin DR, Hartnett ME, et al. Divergence paralysis. *Am J Ophthalmol* 1982;94:506–10.
15. Parinaud MH. Paralysie des mouvements associés des yeux. *Arch de Neurol* 1883;5:145–72.
16. Von Noorden GK. Anomalies of convergence and divergence. In: Von Noorden GK, ed. *Binocular Vision and Ocular Motility. Theory and Management of Strabismus*. 5th ed. St. Louis, Mosby; 1996: 468–476.
17. Mays LE. Neural control of vergence eye movements: convergence and divergence neurons in midbrain. *J Neurophysiol* 1984;51: 1091–108.