DIVERGENCE PARALYSIS WITH INCREASED
INTRACRANIAL PRESSURE

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Divergence paralysis was first described by Parinaud in 1883. Since then, it has been observed by other investigators and there seems to be some good evidence that a center for divergence exists in the midline of the brain stem, probably between the nuclei of the 6th nerves. The latter view was advanced by Bruce in 1935.

However, divergence paralysis is still rather infrequently described in the neuro-ophthalmological literature. While this is undoubtedly due to a low incidence of occurrence, it is also possible that it is occasionally not recognized clinically, but passed off as atypical diplopia with no individual ocular muscles being found at fault. However, despite this apparent infrequency, we encountered 3 cases of divergence paralysis on our service over a period of 3 months, although we are not aware of having seen divergence paralysis for at least 4 years prior to this period.

The syndrome of divergence paralysis is described by Duke-Elder as "the appearance of a convergent strabismus with homonymous diplopia of the concomitant type when the eyes view a distant object, together with the absence of any limitation of movement of either eye in ductions or in versions in any part of the field." Normally, when one changes his gaze from a near to a more distant object, one or both globes may turn out so that the apex of the angle formed by the visual axes of the two globes is transferred from the near object to the more distant one. This action is called divergence, and, to a great extent, invokes the action of the external recti muscles. In divergence paralysis, the eyes can converge normally, and, by relaxing this convergence, can view an object singly up to a certain near point which is usually 10 or 20 inches in front of the patient. Beyond this near point, divergence power is necessary for binocular single vision, and, since divergence is absent, the patient begins to see double. The further away from this near point the object is held, the greater the distance between the two images. At the same time, while the disconjugate movement of divergence is gone, the individual ocular muscles and conjugate movements show no palsies. In other words, the abduction and adduction, as well as dextro- and laevo-versions are normal.

At any one distance from the patient, the distance between the two images is, with some minor variations, constant, in all the cardinal directions of gaze. This is in contrast to the picture in an individual muscle palsy, where the separation of images is greatly increased in the field of action of the paretic muscle.

In making a differential diagnosis, two conditions may cause confusion—
convergence spasm and bilateral 6th nerve palsy. The latter can be ruled out by the lack of external rotation of each eye on attempted laevo- and dextroversion. In convergence spasm, as fixation approaches the near point the diplopia increases, while, in divergence paralysis, it decreases.

While divergence paralysis was described as possibly due to a functional disturbance by von Hippel and Clark, Savitsky and Madonick pointed out that among over 2000 cases of psychoneuroses observed in private practice, no case of divergence paralysis was encountered that could be considered entirely functional.

In organic disease, divergence paralysis has been described in the following conditions:

1. Inflammatory or toxic diseases of the cerebrum. Duke-Elder cited a number of references where divergence paralysis occurred in syphilis and tabes, encephalitis, multiple sclerosis, diphtheria, poliomyelitis, influenza, chorea and lead poisoning.

2. Cerebral hemorrhage. Alger and Wheeler (cited by Duke-Elder, p. 4176) reported 1 case each in which they assumed the divergence paralysis was due to hemorrhage into the divergence center, because of the rather sudden onset of symptoms.

3. Head trauma. Divergence paralysis was reported by Bielschowsky, Weed, and Savitsky and Madonick, the latter expressing the opinion that in such cases there was trauma to the hypothetic center for divergence in the midbrain.

4. Brain tumor. There are 7 cases in the literature in which the nature and site of the lesion were confirmed either by necropsy or surgery, as well as 3 cases in which the same picture seemed to be present, but was unverified. The latter 3 cases were reported by Straub, Holden, and Howard.

The 7 verified cases were reported from 4 different sources. Bender and Savitsky reported a case of a small vascular tumor in the pons verified by necropsy. Divergence paralysis was the chief complaint during life and papilledema was present.

Lippmann described a case of cerebellar tumor verified by necropsy. Divergence paralysis had been present for 2 years prior to death and papilledema developed before death.

Robbins reported a case of divergence paralysis in which the patient died after 6 months. Necropsy revealed a cerebellar cyst. While papilledema was not observed during life, the ventricles were found to be dilated.

Savitsky and Madonick reported 4 cases of posterior fossa tumor with divergence paralysis, all verified by surgery. Two were acoustic neuromas and the other 2, cerebellar tumors. All patients showed papilledema preoperatively. In 3 cases, the tumor was removed and the divergence paralysis cleared completely as the papilledema diminished. In the 4th case, due to the vascularity of the tumor, only a biopsy was taken, and the divergence paralysis and papilledema were still present at the time of their report. They