DIVERGENCE PARALYSIS WITH INCREASED INTRACRANIAL PRESSURE

MAX CHAMPLIN, M.D., AND LEO M. DAVIDOFF, M.D.

Neurosurgical Service, Beth Israel Hospital, New York City

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Divergence paralysis was first described by Parinaud in 1888. 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 laevoversions 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 laevos- 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
had not found divergence paralysis in any cases of increased intracranial pressure without tumor and concluded that divergence paralysis is due to an involvement of the divergence center in the brain stem in some way.

AUTHORS' CASES

We are reporting upon 3 cases, each of which showed evidence of increased intracranial pressure, and typical divergence paralysis. All 3 patients were operated on, with disappearance of the increased intracranial pressure and complete disappearance of the divergence paralysis.

Case 1. B. N., a 16-year-old girl, complained of dizziness for 3 months and double vision for 3 weeks. There was typical divergence paralysis with a separation of images starting at 13 in., where the patient reported a 3-in. separation took place. At 7½ ft., the images were about 15 in. apart. Individual duction and version actions of all the extraocular muscles were normal. The fundi showed congestion of the nasal margins of the discs, and the blind spots were enlarged to a little beyond the limits of normal (with 2/2000 white, OD measured 8½° by 10°, and OS, 8½° by 9½°).

A ventriculogram revealed internal hydrocephalus with evidence of a tumor in the posterior fossa. A craniotomy was performed and a right cerebellar hemangio-blastoma removed.

One week postoperatively, with recession of the papilledema, the divergence paralysis had cleared considerably and, in 10 days, it was all gone. At the time of this report, 8 months later, there was no recurrence of any symptoms.

Case 2. S. B., a 56-year-old man with a 6-month history of headache, complained of seeing double for the past 2 weeks. Examination revealed bilateral frank papilledema and typical divergence paralysis. The patient reported a separation of images starting at 6 in. and increasing with the distance from him until, at 20 ft., he reported about 36 in. of separation. Individual muscle action of the internal and external recti was normal, so that both left and right conjugate gaze were unimpaired.

Operation revealed subdural hematomas bilaterally. These were evacuated and the patient was relieved of headache.

Five days postoperatively, the separation of images at 20 ft. was reduced from 36 in. to 10 in. On discharge 15 days after operation, the diplopia was entirely gone and the papilledema had receded. At the time of this report, 5 months postoperatively, there has been no recurrence of symptoms.

Case 3. P. T., a 35-year-old man, presented himself with a history of headache for 10 days. The disc margins were blurred nasally and the blind spots were definitely enlarged. With 2/2000 white, the right blind spot measured 8° by 13° and the left, 10° by 12°.

Ventriculograms were made and all the ventricles filled well. There were no clear-cut deformities or obstruction to warrant any surgical procedure at that time and he was sent home.

He returned 19 days later with a history of increasing headache as well as the onset of diplopia soon after having left the hospital. Examination revealed frank
papilledema bilaterally, as well as divergence paralysis. At 4 ft., he reported a separation of 10 in. of images and, at 15 ft., about 4½ ft. of separation. On the other hand, abduction and adduction, as well as left and right conjugate gaze, were normal, so that none of the individual recti muscles was paretic.

Following a subtemporal decompression, the papilledema and diplopia subsided. The final diagnosis was serous meningitis. Nine days postoperatively, the diplopia had diminished so that, at 15 ft., the separation was only 2 ft. Three weeks after operation the papilledema was almost all gone and no diplopia was present.

COMMENT

In all 3 of our cases of divergence paralysis, the common accompanying factor was increased intracranial pressure. Only in Case 1 was there a local space-occupying lesion in the posterior fossa. In Case 2 the increased intracranial pressure was due to bilateral supratentorial subdural hematomas. In Case 3 it was associated with serous meningitis.

In the 7 cases of proven brain tumor reported by other authors, all but 1 showed papilledema, and that one showed dilated ventricles at necropsy, thus indicating obstructive hydrocephalus and therefore probably increased intracranial pressure. In addition, in all these 7 cases there was a local space-occupying lesion in the posterior fossa.

The questions that arise are whether posterior fossa lesions near the region of the so-called divergence center plus increased intracranial pressure are necessary to produce divergence paralysis, or whether increased intracranial pressure alone can produce it. Our Cases 2 and 3 certainly showed no evidence of local lesions in the posterior fossa and the divergence paralysis cleared with the subsidence of papilledema.

While in some of the previously reported 7 cases diplopia had been present for even 2 years prior to operation or necropsy, in some instances of spontaneous remissions it would be difficult for us to prove that there was not a low-grade increased intracranial pressure during that time. Certainly, the absence of visible papilledema does not rule out increased intracranial pressure.

In view of these facts, and the occurrence of divergence paralysis in 2 of our patients with increased intracranial pressure but no evidence of a local space-occupying lesion in the posterior fossa, it seems reasonable to assume that increased intracranial pressure alone may cause divergence paralysis. The occurrence of 6th nerve palsy with increased intracranial pressure is well known. Whether the mode of action is similar in the two conditions remains to be seen. As a matter of fact, Bielschowsky observed cases of typical divergence paralysis that went on to true abducens palsy. He considered this as evidence of an organic lesion near the intact abducens nucleus and later extending to and finally involving the nucleus itself. He considered this as good evidence that true divergence innervation exists.

From a clinical point of view, we think that divergence paralysis is seen as an infrequent accompaniment of increased intracranial pressure, but must not be regarded as pathognomonic of a lesion in the posterior fossa. In other
words, increased intracranial pressure alone seems to be capable of producing divergence paralysis. The additional presence of a lesion in the region of the so-called divergence center does not seem to be necessary for its production.

CONCLUSIONS

1. Divergence paralysis, while infrequently encountered, must be thought of and looked for in cases of increased intracranial pressure, and should certainly be looked for in all cases of diplopia with no apparent limitation of individual ocular muscle action.

2. Divergence paralysis is found in cases of increased intracranial pressure, and, while frequently associated with a space-occupying lesion in the region of the so-called divergence center, has been demonstrated in 2 cases without any clinical evidence of any such posterior fossa lesion.

3. Divergence paralysis must not be considered pathognomonic of a local lesion in the posterior fossa.

REFERENCES