Central disruption of fusional amplitude

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The stability of binocular vision depends on good fusional amplitudes. These amplitudes are the result of a reflex arc, the sensory stimulus being caused by the images of the object of regard falling off one fovea. If this image falls outside Panum's area, diplopia will result. The motor response involves a movement of one or both eyes to maintain fusion and avoid diplopia. The afferent sensory stimulus from each eye must reach the cortex for conscious fusion to take place (Moses, 1970). The efferent motor limb of the reflex must produce a co-ordinated response from the eye muscles to keep each eye correctly aligned with its fellow. An occipito-mesencephalic nervous pathway conveys these fibres to the mid-brain area for supranuclear control of the eye movements.

The horizontal amplitudes of fusion are dependent on convergence and divergence. Convergence or divergence paralysis may be seen as a clinical entity (Walsh and Hoyt, 1969). A combination of convergence and divergence paralysis would result in a complete disruption of horizontal fusional motor response in the presence of normal versions. Such a lesion would leave the patient with the ability to fuse the images from the two eyes if they were superimposed but the inability to correct diplopia with any fusional vergence movements. From the practical point of view, therefore, a patient such as this would have constant and incurable diplopia.

Fusional amplitudes are also present on a vertical plane and supranuclear disruption may occur. It would seem likely that some cases of skew deviation, where a concomitant vertical separation of the two eyes takes place, may be due to manifestations of a disruption of the vertical fusional amplitude. This seems particularly likely in those cases where the vertical separation is under 10 prism diopters.

It is postulated that a motor association area, controlling fusional amplitudes, exists possibly in the mid-brain, and that if it is damaged, a loss of fusional amplitude occurs as the main sign, with consequent constant diplopia as the main symptom. The following case reports lend credence to this hypothesis.

Case reports

(1) CONGENITAL LACK OF FUSIONAL AMPLITUDES

Occasionally one sees patients whose presenting symptom is diplopia which they have had all their lives. It is due to poor fusional amplitude in the absence of other abnormal findings.

Case 1, a man aged 41 years, complained of diplopia, both horizontal and vertical, which he had frequently noticed at all distances. He had had prescriptions for prisms and had carried out numerous orthoptic exercises in an attempt to improve his fusional amplitudes. He had gained no relief from any method of treatment.

Sensory analysis

The visual acuity was 20/15 unaided in each eye, normal near reading without correction, stereo acuity 40 seconds of arc at near (Wirt test). Foveal fusion with poor amplitudes was confirmed on the troposcope.

Motor analysis

Orthophoria at 20ft in all positions of gaze, esophoria of 4 prism diopters at 25 cm., normal near point of convergence, normal ductions and versions. Fusional amplitudes, in free space, revealed 6 prism diopters of convergence amplitude and 4 prism diopters of divergence amplitude. The patient appreciated no relief from his symptoms, in the distance or at near, with the use of prisms or orthoptic exercises. It was concluded that he had a weakness of fusional amplitudes due, possibly, to a congenital lesion in the fusional amplitude association area.

(2) ACQUIRED DISRUPTION OF FUSIONAL AMPLITUDE

This is most commonly associated with trauma but may also occur with space-occupying or vascular lesions.

Case 2, a girl aged 11 years, first visited an ophthalmologist at the age of 6 when normal unaided vision was recorded. An exophoria was noted with excellent fusion and, at no time, was a tropia detected. At the age of 10 years the child was walking in the park when she suddenly became dizzy, fell to the ground, and was completely unconscious for 5 minutes. A complete neurological investigation did not reveal any abnormality other than the fact that she had constant double vision at all times.

Sensory analysis

The visual acuity unaided, was 20/20 in each eye. No fusion could be recorded with prisms in free space but momentary fusion at her angle could be recorded with the troposcope.

Motor analysis

Variable alternating exotropia of 0–25 prism diopters with a small left hypertropia of up to 2 prism diopters was recorded. Versions and ductions were normal. The patient could converge the eyes from the divergent resting position until the eyes were slightly convergent. No abnormality was seen in the fundus.

Conclusion

Since it was known that the child's sensory and fusional state was perfectly normal before this episode, it was concluded that she had an acquired, probably vascular, accident in the fusional amplitude area. No treatment could be offered.

Case 3, a youth aged 17 years, had noticed a sudden onset of double vision 10 days before being seen and had recently suffered from some headaches.

Sensory analysis

The visual acuity with myopic correction was 20/15 in each eye. Fusion was obtained at the angle of strabismus both on the troposcope and using prisms in free space, but no amplitude was obtainable.

Motor analysis

A small esotropia of 4 prism diopters was noted in all positions of gaze at 20 ft, together with a left hypertropia of 8 prism diopters which was constant in all positions of gaze. Versions illustrated a slight limitation of elevation of both eyes. No fusional amplitudes were demonstrable with prisms in free space or on the troposcope.

Diagnosis

The diagnosis of an acquired lesion in the mid-brain affecting the fusional amplitude mechanism was made, and the patient was referred to a neurologist. Subsequent investigation proved that he was suffering from a tumour of the mid-brain from which he died 9 months later. The diagnosis of glioblastoma multiformae in the brain stem was confirmed *post mortem*. Widespread infiltration was noted in the ventricular system, thalamae, mid-brain, pons, and cerebellum with spread down the spinal cord in the subarachnoid space.

Case 4, a man aged 32 years, had suffered from constant diplopia ever since he was involved in an accident involving a head injury with loss of consciousness at the age of 11 years. He complained of constant horizontal diplopia in the normal position of gaze and when he looked up a vertical component gave rise to some vertical as well as horizontal separation.

Sensory analysis

The visual acuity, unaided, was 20/20 in each eye. The patient was able to fuse at his angle but had no fusional amplitude that one could record.

Motor analysis

A constant exotropia of 20 prism diopters with a left hypertropia of 3–4 prism diopters was noted in the primary position at 20 ft. This decreased to 7 prism diopters when gazing 25° below the horizontal meridian at 20 ft, whereas it remained constant at 20 prism diopters on upward gaze. At near, the exotropia increased to 35 prism diopters with the same amount of hypertropia. Ductions were normal and versions illustrated that a slight V pattern present. The Hess chart showed the slight V pattern exotropia, but nothing diagnostic from a paretic point of view. No fusional amplitudes could be recorded on the troposcope or by the use of prisms in free space.

Diagnosis

Traumatic disruption of fusional amplitude. No treatment could be offered.

Case 5, a woman aged 26 years, had enjoyed good sight without symptoms before an accident in which she had been involved 2 weeks before examination. The accident involved a whiplash injury without loss of consciousness, leaving her with headache and constant double vision.

Sensory analysis

The visual acuity with 0.50 D sph. was 20/20 in both eyes. The patient could fuse on the troposcope at -5 but had no fusional amplitude. Since she had a complete paralysis of convergence one would not expect her to have any convergence amplitude, but she also had no divergence amplitude although she had no difficulty fusing if the targets were superimposed. The amplitude of accommodation was 0.50 diopters in each eye.

Motor analysis

A small but constant exotropia of 2 prism diopters was noted at 20 ft. A 20 prism diopter exotropia was present at 25 cm. associated with a complete paralysis of convergence and accommodation. Versions and ductions were full and normal. No divergence amplitudes were present on the troposcope or in free space using prisms. The visual fields were normal.

Diagnosis

A thorough investigation by a neurologist showed no other neurological lesion. The findings were unchanged a year after the accident and the diagnosis was made of a traumatic lesion, probably involving the mid-brain, disrupting fusional amplitude, convergence, and accommodation.

Discussion

These cases are presented as evidence that fusional amplitudes can be permanently disrupted although sensory fusion is still present. Convergence paralysis alone is sufficient to produce this catastrophe if the patient had previously had an exophoria.

If this phenomenon of central disruption of the fusional amplitudes is not clearly recognized, serious error may occur in advising surgical correction of an infranuclear muscle imbalance which has occurred concurrently. Since reasonable fusional amplitudes are necessary for the preservation of single binocular vision in different positions of gaze, if disruption of the fusional amplitude mechanism has taken place the patient is untreatable with surgery or with prisms.

Summary

The possibility is discussed that a lesion which produces a central disruption of the fusional amplitude may result in incurable diplopia. Five cases are presented to illustrate congenital, traumatic, possibly vascular, and neoplastic lesions affecting the fusional amplitude mechanism. It is postulated that the mid-brain is the most likely site for such a lesion. It is emphasized that failure to recognize this lesion as an entity may result in poor advice regarding corrective surgery for a nuclear or infranuclear paresis when this is combined with this lesion.

References

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