

Anomalies of Convergence and Divergence

In this chapter, the discussion of anomalies of convergence and divergence is based on the premise that both mechanisms are active processes. Although there has never been an argument against convergence being an active force, this does not hold true for the divergence mechanism. Scobee and Green⁴⁹ postulated, for instance, that divergence is a passive form of eye movement elicited by relaxation of convergence, a divergent position of the orbital axes, and the elasticity of orbital tissues. Costenbader,¹⁶ who reviewed the evidence for and against an active divergence mechanism, concluded that divergence is passive and that its anomalies are variances of a divergent position of rest associated with anomalies of convergence. In our view, electromyographic findings and the presence of fusional divergence prove unequivocally that an active divergence mechanism does exist.

It was established from the beginning of the study of fusional eye movements that fusion can be maintained in spite of the presentation before the eyes of prisms base-in of increasing power or image separation by a haploscopic arrangement. Under these conditions the eyes will diverge beyond parallelism of the visual axes. Moreover, patients with esophoria keep their eyes aligned and recover fusion by fusional divergence after temporary dissociation of the eyes. Clearly, under these circumstances, nothing but an active process

could produce divergent eye movements capable of maintaining or recovering fusion.

Electromyographic findings further attest to the active nature of the divergence mechanism. Early support for this concept was supplied by the work of Adler¹ and of Breinin.⁷ Breinin recorded increased activity of the lateral rectus muscle at the breakpoint of fusion in intermittent exotropia. This increase of activity occurred before the inhibition of the medial rectus muscle, just before the eye diverged, and was maintained as long as the eye remained divergent. Tamler and Jampolsky⁵³ argued that to prove unequivocally the existence of active divergence, recordings must be made simultaneously from both lateral rectus muscles during recovery from a fusion-free position or while maintaining fusion during presentation of prisms base-in before the eyes. Using this method, these investigators could demonstrate an increase of electrical activity in both lateral rectus muscles as a patient maintained fusion when prisms base-in were placed before the eyes (Fig. 22-1) and as patients with intermittent esotropia recovered fusion from a fusion-free position (Fig. 22-2). Thus the existence of active fusional divergence has been proved beyond a doubt.

The fact that the location of a divergence center has not been identified should not be interpreted as an argument against active divergence. First, divergence has been produced experimentally in

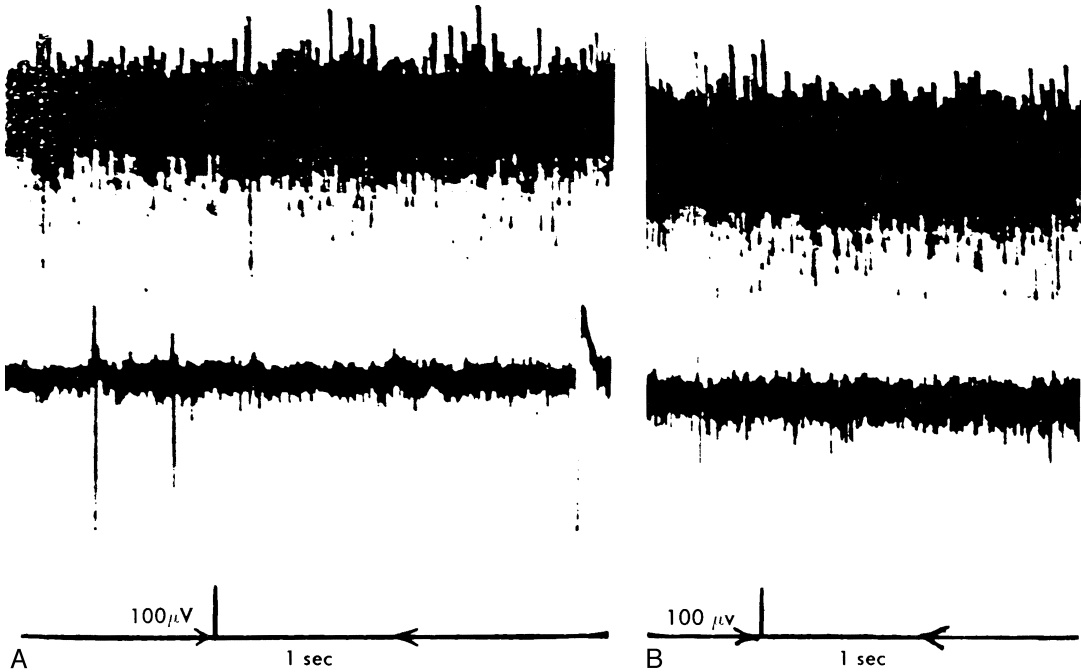


FIGURE 22-1. Active divergence elicited by prisms base-in. *A*, Electromyogram of right lateral rectus muscle (*upper tracing*) and left lateral rectus muscle (*lower tracing*) before introduction of the prism. *B*, Increased activity of both lateral rectus muscles after 18 Δ base-in was added while fusion was maintained. (From Tamler E, Jampolsky A: Is divergence active? An electromyographic study. *Am J Ophthalmol* 63:452, 1967.)

monkeys by means of electrical stimulation of the oculomotor area in the frontal lobe,^{17, 44} which suggests the existence of a supranuclear control mechanism for divergence. Second, the existence of active convergence is universally accepted, even though Warwick⁵⁷ effectively shattered the

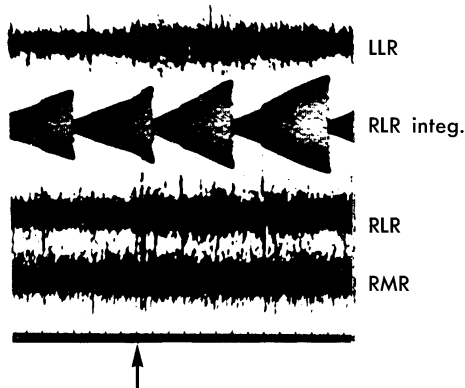


FIGURE 22-2. Electromyogram during fusional divergence movement. As the left eye of a patient with 17D intermittent esotropia is uncovered (*arrow*), there is increased activity of both left and right lateral rectus muscles (LLR, RLR) as the eye makes a fusional divergence eye movement. (From Tamler E, Jampolsky A: Is divergence active? An electromyographic study. *Am J Ophthalmol* 63:452, 1967.)

concept of Perlia's nucleus acting as a convergence center. Thus the lack of more precise anatomical information regarding the site of a divergence center does not dispose of the physiologic and clinical data that support an active divergence mechanism.

Jampolsky,³⁰ although accepting the fact that divergence is an active mechanism, denied the existence of nonretinal tonic vergence innervations. He rejected the concept of divergence insufficiency and paralysis and accepted only the fusional form of active divergence, that is, that which is elicited by temporal disparity of retinal images. It would exceed the purpose of this chapter to analyze this hypothesis, which is discussed further in Chapter 5, except to say that, in our opinion, a tonic vergence mechanism has not been disproved by the evidence presented. Moreover, one may argue that convergence and divergence paralyzes, as well as convergence nystagmus, are well-recognized entities that cannot be explained solely on the basis of deficient fusional divergence,¹⁴ that accommodation is not necessarily defective in convergence insufficiency, and that experimentally administered ethanol^{6, 15, 45} and barbiturates⁵⁸ have been shown to affect the tonic

vergence mechanism. Furthermore, Scott⁵⁰ reported electromyographic findings in a patient with V exotropia that could not have been caused by anything but tonic divergence.

Anomalies of Convergence

Convergence Insufficiency

ETIOLOGY AND CLINICAL FINDINGS. Convergence insufficiency is one of the most common causes of ocular discomfort and, in fact, is the most common cause of muscular asthenopia; therefore it is of considerable clinical significance.

There is frequently an etiologic connection with associated accommodative difficulties. For instance, a convergence insufficiency as a result of disuse of the accommodative convergence mechanism may have been caused by uncorrected high hypermetropia or myopia. Patients with hypermetropia exceeding, say, 5D or 6D, make little or no effort to accommodate, and in those with myopia there is no need to accommodate to obtain clear vision at near fixation. Likewise, a patient with beginning presbyopia may develop convergence insufficiency after wearing a bifocal prescription for the first time. The relief of sustained accommodative effort afforded by the new prescription causes a decrease of accommodative convergence; thus an exophoria that had been controlled by accommodative convergence may become manifest. In other patients without refractive problems the condition may arise without any obvious cause.

Some ophthalmologists consider patients with convergence insufficiency to be neurotic and believe that their problem is something to be dealt with by a psychiatrist. We emphatically reject this generalization.¹¹ The often dramatic subjective improvement after appropriate therapy, noted concurrently with the objective clinical finding of improved near point of convergence and fusional convergence amplitudes, clearly puts this condition on an innervational basis.

In rare instances acquired convergence insufficiency may occur on an organic basis, such as secondary to a subdural hematoma.⁵²

An admirably concise description of the symptoms arising from convergence insufficiency was given by von Graefe, who pointed out as early as 1855^{26, 27} that such patients complain about eye-strain and a sensation of tension in and about the

globes. After brief periods of reading, the letters will blur and run together; crossed diplopia occasionally occurs during near work. Characteristically, one eye will be closed or covered while reading to obtain relief from visual fatigue. Little can be added to this classic description except that ocular headaches are another frequent complaint. Von Graefe thought that convergence insufficiency was myogenic, that is, caused by a congenital weakness of the medial rectus muscles secondary to overaction of the lateral rectus muscle. He recommended prisms base-out to exercise the action of the medial rectus muscles in some patients, prisms base-in to eliminate symptoms in others, or a weakening procedure of the lateral rectus muscles. Except for prisms base-out, which are still used by some, his views on the etiology of convergence insufficiency and therapeutic suggestions have not withstood the test of time.

DIAGNOSIS. The diagnosis of convergence insufficiency is based on the finding of a remote near point of convergence and decreased fusional convergence at near fixation. The reader is referred to Chapter 12 for a discussion of diagnostic methods and the clinically important differentiation between the subjective and objective near point of convergence. Most patients with convergence insufficiency exhibit varying degrees of exophoria at near fixation; however, this disorder also occurs in patients with orthophoria and occasionally even in those with esophoria. The near point of accommodation is normal and corresponds to the age of the patient. However, to identify patients who suffer from a combined insufficiency of convergence and accommodation (see p. 503) and who require a different form of therapy, the near point of accommodation should be determined in each case.⁴¹

Convergence insufficiency seldom becomes a clinical problem until a patient reaches the teenage years. Increased schoolwork and prolonged periods of reading may then exacerbate the characteristic symptoms. The type of patient most often encountered is a high-school, college, or university student who is especially prone to develop symptoms before examinations when special demands are made on the near vision complex during extended periods of studying. Needless to say, symptoms are aggravated by lack of sleep, reduction of general well-being, and anxiety.

THERAPY. Therapy for convergence insufficiency is in the realm of orthoptics. Indeed its treatment

is the most successful application of orthoptics and in most instances provides long-lasting relief from symptoms (see Chapter 24). On occasion, however, especially when convergence insufficiency is associated with a large exophoria at near vision, orthoptic treatment fails and surgery may be indicated. The ophthalmologist must remember that, as a rule, convergence insufficiency is a reversible disorder and that the decision to perform surgery should be made with extreme reluctance and not until all other therapeutic possibilities, including prisms base-in, have been exhausted. If surgery is imperative, we have advocated resection of both medial rectus muscles.⁴⁰ Frequently, a temporary overcorrection follows this procedure, and the patient must be warned to expect double vision for several weeks or even months postoperatively.⁴⁰ Of interest is the fact that the consecutive esotropia usually is greater at distance than at near fixation. If this occurs Fresnel prisms are prescribed as upper segment bifocals to neutralize diplopia. Fortunately, the consecutive esotropia has a tendency to disappear spontaneously. Nemet and Stolovitch³⁹ suggested resecting the upper border and recessing the lower border of the medial rectus muscles to make the operation more effective at near than at distance fixation.

From our experience with patients operated on by a resection of both medial rectus muscles and followed for several years, we observed that exophoria at near fixation tends to recur. In several patients, the deviation gradually increased to the preoperative angle. This recurrence notwithstanding, for unknown reasons patients who have been operated on in this manner usually remain asymptomatic. The efficacy of surgical treatment of intractable convergence insufficiency for relief of asthenopic symptoms was confirmed by Hermann.²⁸

Convergence Insufficiency Associated with Accommodative Insufficiency

First mentioned by Duane,²² systemic convergence insufficiency, associated with subnormal accommodation, was later described by Brown⁸ following diphtheria, mononucleosis, encephalitis, and streptococcal throat infections. Brown pointed out that this type of convergence insufficiency differs radically from the functional type, inasmuch as symptoms are more severe and remissions are few.

Von Noorden and coworkers⁴¹ reported nine

adolescent and young adult patients with a combined insufficiency of accommodation and convergence. These patients did not respond to conventional orthoptic training. Except for one patient in whom symptoms developed suddenly after an automobile accident, all others had a gradual onset over many years, and the complaints were no different from those commonly associated with functional convergence insufficiency. Unlike in simple convergence insufficiency, the near point of accommodation was found to be drastically reduced (Fig. 22–3), and the accommodative convergence/accommodation (AC/A) ratio was extremely low or absent. In five patients, convergence response could not be elicited at all by stimulation of accommodation with minus lenses. With the exception of one case of trauma, the histories of all other patients were unremarkable except for severe febrile illnesses during childhood in two instances. Von Noorden and coworkers⁴¹ assumed that trauma or subclinical viral encephalopathies may be factors in the pathogenesis of this condition. Trimble⁵⁵ reported sudden loss of accommodation and convergence in a 12-year-old boy. A cause was not established.

Such patients are treated by us with plus lenses for reading and prisms base-in. Only the minimal power necessary to achieve comfortable vision should be prescribed. Fresnel membrane prisms and lenses are of value since frequent adjustment of lenses and prisms may be necessary before the optimal correction can be achieved. Miotics are totally ineffective since they increase the exodeviation at near vision, thus adding to the patient's discomfort. Resection of both medial rectus muscles followed by prescription of bifocals may be helpful.⁴⁰

To recognize this syndrome prior to orthoptic therapy, which has been futile in our hands but reported to be effective by others,³⁷ we recommend measuring the near point of accommodation in all patients with convergence insufficiency.

Convergence Paralysis

Parinaud⁴³ was the first to describe convergence paralysis as a condition distinct from convergence insufficiency, whereby diplopia exists only at near fixation, adduction is normal, and the patient is unable to converge. Accommodation may be normal, reduced, or absent, and the pupil may or may not be involved. In some patients the pupillary reflex may be abolished for convergence and re-

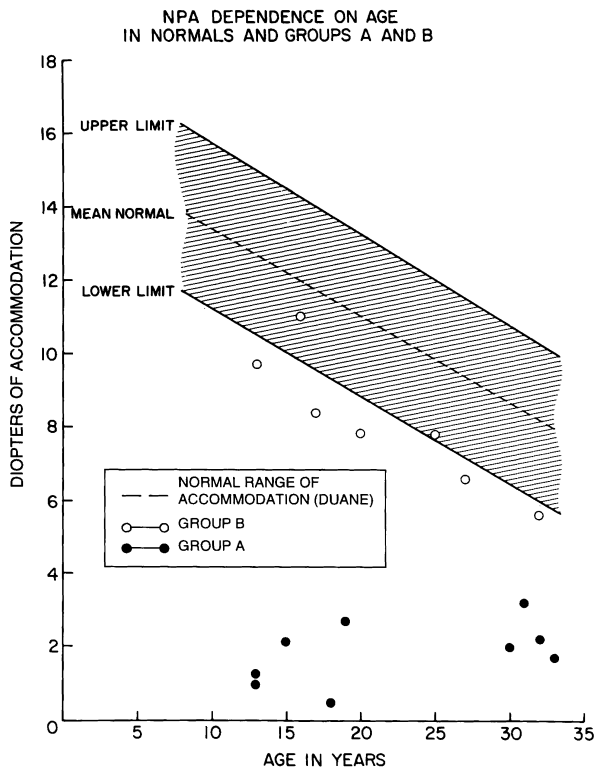


FIGURE 22-3. Near point of accommodation (NPA) in patients with ordinary convergence insufficiency (GROUP A) and combined convergence-accommodation deficiency (GROUP B). The shaded area represents the normal accommodative range established by Duane. (From Noorden GK von, Brown DJ, Parks M: Associated convergence and accommodation insufficiency. *Doc Ophthalmol* 34:393, 1973.)

tained for light (reversed Argyll Robertson pupil). Numerous case reports have followed Parinaud's original description, and the reader is referred to the current texts on neuro-ophthalmology for reviews of the pertinent literature. Bielschowsky³ further analyzed convergence paralysis and its differential diagnosis. He recognized the difficulties involved in differentiating organic from functional disturbances of convergence, that is, distinguishing between the patient's *inability* to converge and his or her *unwillingness* to do so. Convergence paralysis may be easily simulated—intentionally in uncooperative patients and unintentionally in neurotic patients and those with debilitating diseases. To establish whether the patient is unable or unwilling to converge, one must test the fusional convergence with a rotary prism by gradually introducing prisms base-out at a fixation distance of 1 to 2 m. A patient with convergence paralysis will immediately recognize diplopia when prisms are used. The condition is functional when fusional convergence can be elicited with prisms. Bielschowsky³ established the following prerequisites for the diagnosis of convergence paralysis: (1) evidence of intracranial disease, (2) history of sudden onset of crossed diplopia at near fixation, (3) reproducible findings on subsequent examina-

tions, and (4) preservation of accommodation and pupillary reaction on attempts to converge. If internal ophthalmoplegia is associated with convergence paralysis, the presence of an organic lesion of nuclear or supranuclear location is almost certain.

Autopsy findings have shown that convergence paralysis occurs most frequently when lesions are present in the corpora quadrigemina or the third cranial nerve nucleus. The frequent association of convergence paralysis with vertical gaze paralysis (Parinaud's syndrome) further emphasizes that convergence paralysis may be caused by lesions in this location.

Walsh and Hoyt^{56, p.237} listed encephalitis, disseminated sclerosis, and tabes as etiologic factors in convergence paralysis but stated that convergence paralysis occurs most commonly as a result of head injury. Convergence paralysis following mushroom poisoning has also been reported.²⁵ If accommodation is also defective (internal ophthalmoplegia), therapy consists of prisms base-in for near vision in combination with bifocals.

Convergence Spasm

In our experience, convergence spasm occurs almost exclusively in "hysterical" or otherwise neu-

rotic persons. Such patients will exhibit intermittent episodes of sustained maximal convergence associated with accommodative spasm (induced myopia) and miosis. The functional nature of this condition is emphasized by the finding that such patients may have tubular field defects. In some instances, the spasm can be triggered by asking the patient to fixate an object held closely before the eyes. After the fixation object has been removed, the eyes will remain in a convergent position, as demonstrated by Case 22–1.

CASE 22–1

A 28-year-old female school teacher complained of intermittent double vision and blurring. Her visual acuity was 6/6 OU without correction. Refraction revealed emmetropia. Examination of ocular motility showed orthophoria at distance. During the cover and the cover-uncover test at near fixation, the patient's eyes suddenly converged, she complained of diplopia, and her pupils had become miotic. She maintained convergence after removal of the fixation target. Refraction was repeated, and myopia of 6D was now present in each eye. After 12 minutes the eyes returned spontaneously to a normal position. On further questioning the patient complained about a multitude of bizarre symptoms. She was referred to a psychiatrist who diagnosed "hysteria."

Souders⁵¹ reported convergence spasm in two neurotic persons. The clinician should be aware, however, that convergence spasm may have an organic basis, and it has been reported to occur after encephalitis, tabes, labyrinthine fistulas,⁵ trauma,^{20; 56, p.239} Arnold-Chiari malformation, pituitary adenoma, and posterior fossa neurofibroma.¹⁹ Thus patients with spasm of the near reflex should undergo neurologic evaluation to exclude these organic causes.¹⁹ Bielschowsky³ reported convergence spasm with traumatic unilateral depressor paralysis in a patient in whom a spasm of convergence and accommodation occurred after an attempt to overcome the vertical diplopia.

Unless an organic cause can be found, therapy consists of prolonged atropinization of the eyes and plus lenses prescribed as lower segment bifocals. Monocular occlusion may also be considered. This treatment may have to be applied for many months, sometimes for as long as a year, before the convergence spasm subsides and the patient regains visual comfort.

Anomalies of Divergence

Divergence Insufficiency

Divergence insufficiency is regarded as a separate and benign clinical entity to be differentiated from divergence paralysis and bilateral cranial nerve VI paresis. It is characterized by intermittent or constant esotropia at distance fixation with symptomatic uncrossed diplopia in patients who maintain fusion at near. Characteristically, the angle of strabismus is the same in primary position and lateral gaze. Fusional divergence is reduced markedly both at distance and near fixation. Unlike in the case of divergence paralysis, the neurologic findings are normal.

The literature contains relatively few reports dealing with divergence insufficiency.^{12, 29, 38, 42, 46} A distinction between divergence insufficiency and paralysis is by no means an easy task in every case, nor is it always possible. On clinical grounds, however, whenever possible we find it useful to differentiate between a patient with esotropia of sudden onset at distance fixation in the absence of neurologic disease (divergence insufficiency) and one in whom this event is associated with a history of head trauma, hypertensive vascular disease, or other neurologic problems (divergence paralysis). In divergence insufficiency without neurologic signs we first use prisms base-out in the amount required to give the patient comfortable single vision at distance fixation. This prescription usually does not interfere with single binocular vision at near fixation but if it does, upper segment Fresnel prisms are prescribed.

Since divergence insufficiency is a self-limiting condition, the prisms usually can be reduced in power after several weeks or months and eventually be dispensed with. However, if the patient does not respond to prisms, resection of both lateral rectus muscles should be considered^{11, 36} and adjustable sutures are advisable.³⁵

Divergence Paralysis

Divergence paralysis was described first as a clinical entity by Parinaud,⁴³ who observed patients with a sudden onset of homonymous diplopia at distance fixation in whom ductions and versions were normal. Many subsequent cases have been reported.^{10, 21, 24, 32} Among the underlying diseases most frequently cited to be etiologically significant are tabes, encephalitis, disseminating sclerosis,

pseudotumor cerebri,³⁰ poliomyelitis,^{23; 56, p.238} influenza,² vascular lesions involving the vertebro-basilar arterial system,¹⁸ neoplasms,^{9, 34, 36} increased intracranial pressure,^{13, 31, 54} and head trauma,³³ in which divergence paralysis may be associated with papilledema.⁴⁸ Even though the existence of divergence paralysis has been disputed for a long time, many current authors have accepted it.²⁹ Bielschowsky⁴ discussed the difficulties encountered in making the diagnosis of divergence paralysis and pointed out how frequently this condition may be simulated by paresis of one or both lateral rectus muscles. He observed several cases of lateral rectus paresis in which comitance developed rapidly, and within a matter of a few weeks the motility defect became indistinguishable from divergence paralysis. Jampolsky³⁰ expressed the view that divergence paralysis does not exist and that all such patients actually have a bilateral cranial nerve VI paresis. Kirkham and coworkers³¹ reported three patients with increased intracranial pressure and divergence paralysis. The deviation remained unchanged in lateroversion, but the electro-oculographically determined saccadic velocities were significantly reduced in abduction, which attests to weakness of lateral rectus muscle action. After the patients recovered, symptoms of divergence paralysis subsided and saccadic velocities in abduction returned to normal. These authors concluded that cranial nerve VI pareses, caused by increased intracranial pressure, may produce symptoms of divergence paralysis without other evidence of cranial nerve VI palsy and without the need to evoke a specific lesion involving the divergence mechanism. Hence a clear distinction between divergence palsy and lateral rectus muscle paresis may be difficult at times, or even impossible, since one condition may merge with the other.

On the other hand, the evidence from carefully conducted studies of individual cases is sufficiently convincing to consider divergence paralysis as a clinical entity separate from a bilateral cranial nerve VI paresis. Several findings clearly inculcate the divergence mechanism and cannot be explained on the basis of bilateral abducens nerve paresis: (1) fusional divergence is markedly reduced or absent if measured from the fusion-free position at near and distance fixation, (2) the esotropia not only remains unchanged but may even decrease in lateroversion,^{14, 21, 43} and (3) saccadic velocities are only mildly reduced, as a

recent study of 12 patients with the clinical findings of divergence paralysis has shown.³⁵

According to Bielschowsky,⁴ the diagnosis of divergence insufficiency or paralysis is based on the following criteria: (1) there is a sudden onset of uncrossed diplopia at distance fixation, (2) the angle of strabismus remains unchanged or may decrease on lateroversion, (3) when an object is brought nearer to the patient, the two images approach each other and are finally fused when the object is at a distance of 25 to 40 cm from the patient, and (4) the field of fixation is unrestricted. Bielschowsky⁴ also pointed out that divergence paralysis occasionally may be confused with convergence spasm. In both instances the patient will complain of uncrossed diplopia at distance fixation; however, in convergence spasm, fusional divergence is unimpaired and visual acuity at distance is decreased.

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