Congenital Esotropia

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- Strabismus is one of the most prevalent health problems among children in the Western hemisphere, affecting 5 in every 100 U.S. citizens, or some 12 million persons in a population of 245 million.“
- Infantile strabismus (i.e., strabismus starting in the first year of life) will affect about 1% of full-term, healthy newborns" " and a much higher percentage of newborns who suffer perinatal difficulties due to prematurity or hypoxic/ischemic encephalopathy.

Fig. 1 Child with infantile estropia.

- Infantile esotropia is a form of ocular motility disorder where there is an inward turning of one or both eyes, commonly referred to as crossed eyes.

- It occurs during the first 6 months of life in an otherwise neurologically normal child. Congenital esotropia has been used synonymously but the condition is rarely present at birth.It is also accompanied by dissociated vertical deviation(DVD) 50%-90%, inferior oblique muscle overaction 70%, latent nystagmus 40%, and optokinetic asymmetry.

- Transient misalignment of the eyes is common up to the age of 3 months and it shouldn't be confused with infantile esotropia where the angle of deviation is constant and large(>30 PD).

- Of all subtypes of human strabismus, infantile strabismus may be the most important but least understood. Early-onset strabismus, first and foremost.
- stigmatizes children into their adult years by depriving them of the many benefits bestowed by normal binocular vision." It is important to clinicians because it is a leading cause of visual loss due to amblyopia
- and often requires multiple surgical procedures to restore proper eye alignment.
- Yet, despite the restoration of good to excellent ocular alignment, bifoveal fusion is seldom acquired
- It is important to vision scientists because, in addition to eye misalignment, it is accompanied by improper development of stereopsis, motion processing, ocular fixation, and eye tracking, defects not found commonly in children whose strabismus begins after infancy
- More than 90% of infants who become strabismic develop an esotropic (convergent) misalignment of the visual axes, as opposed to exotropic misalignment.

Etiology:

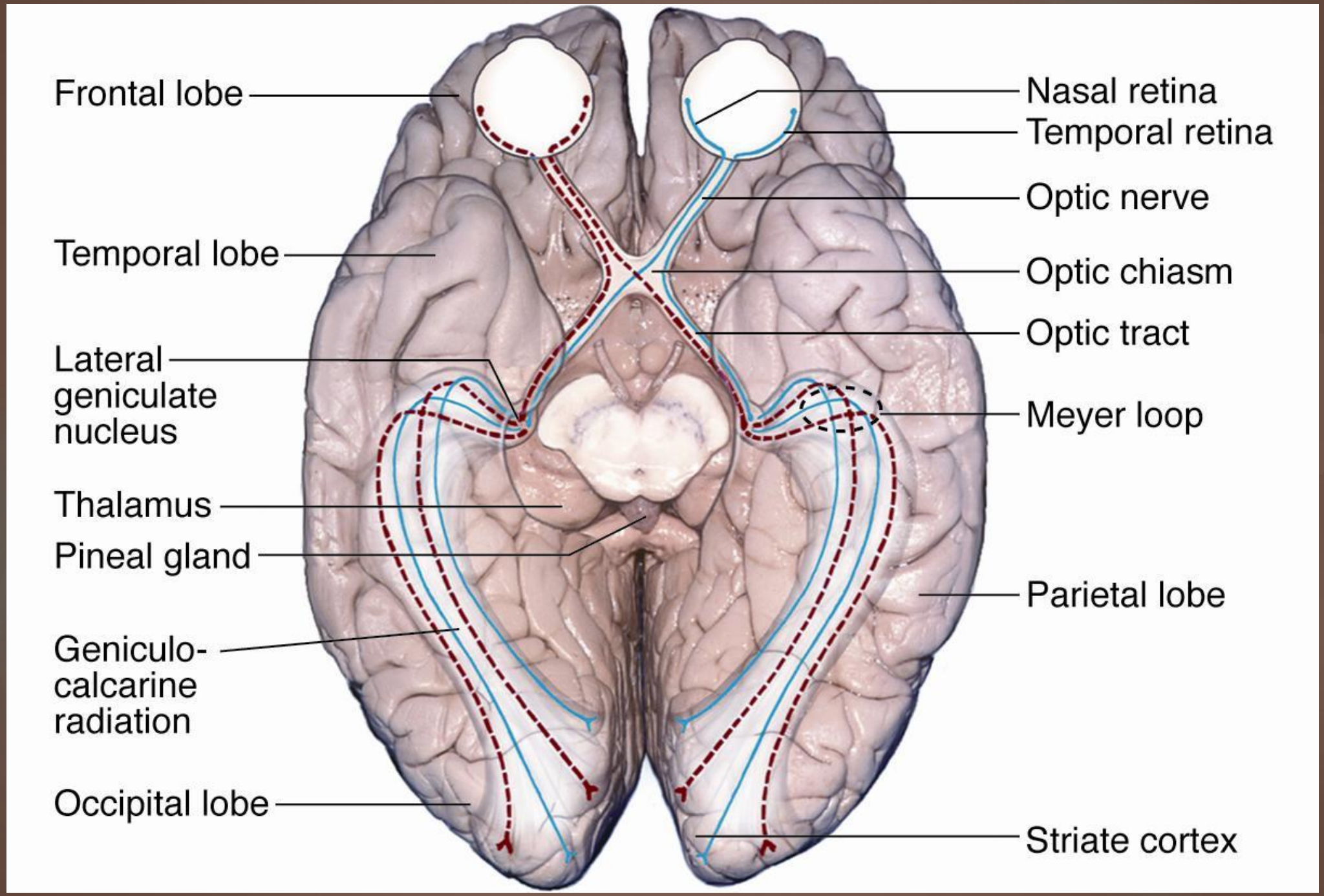
- The etiology of infantile esotropia remains unknown. Many theories have been postulated regarding the pathogenesis of the disease. Worth[1] theory suggests that there is an irreparable congenital defect in the infant's visual system and that surgery can be carried out at leisure mostly for cosmetic purposes.
- On the other hand, Chavasse[2] suggested a primary motor dysfunction, where the associated poor fusion and lack of high-grade stereopsis is probably a sensory adaptation to abnormal visual stimulation during early binocular development caused by the motor misalignment.
- Thus, surgical correction should be performed early during infancy. This view was largely accepted afterwards by Costenbader and Parks. There is a hereditary component with infantile occuring much more common in the children of families with monofixation syndrome.

Visual Pathways Versus Motor Pathways:

- Opposing viewpoints on the origins of infantile esotropia are also arrayed along a visual-motor axis. Worth" and Crone," who postulated a congenital deficit in binocularity, may be placed at the visual cortex end of this axis. A majority of other hypotheses fall into a vague middle ground between the visual cortex and extraocular muscles. Snellen," Scobee," Mindel," 7" and Porter* * define the muscle end of this axis.

CURRENT NOTIONS OF VISUAL CEREBRAL MECHANISMS:

- The afferent visual system operates in the first months of life not as a binocular system but as two parallel, overlapping, monocular visual channe At birth each channel displays, at the level of the primary visual cortex, a directional bias favoring nasally directed motion
- The available electrophysiologic evidence suggests that each eye actually drives visual cortical neurons that will respond to either nasally directed or temporally directed target motion but that in the first months of life only the nasally directed pool responds robustly and connects to eye tracking (pursuit and optokinetic) motoneurons


Frontal lobe
Temporal lobe
Lateral geniculate nucleus
Thalamus
Pineal gland
Geniculo-calcarine radiation
Occipital lobe
Nasal retina
Temporal retina
Optic nerve
Optic chiasm
Optic tract
Meyer loop
Parietal lobe
Striate cortex

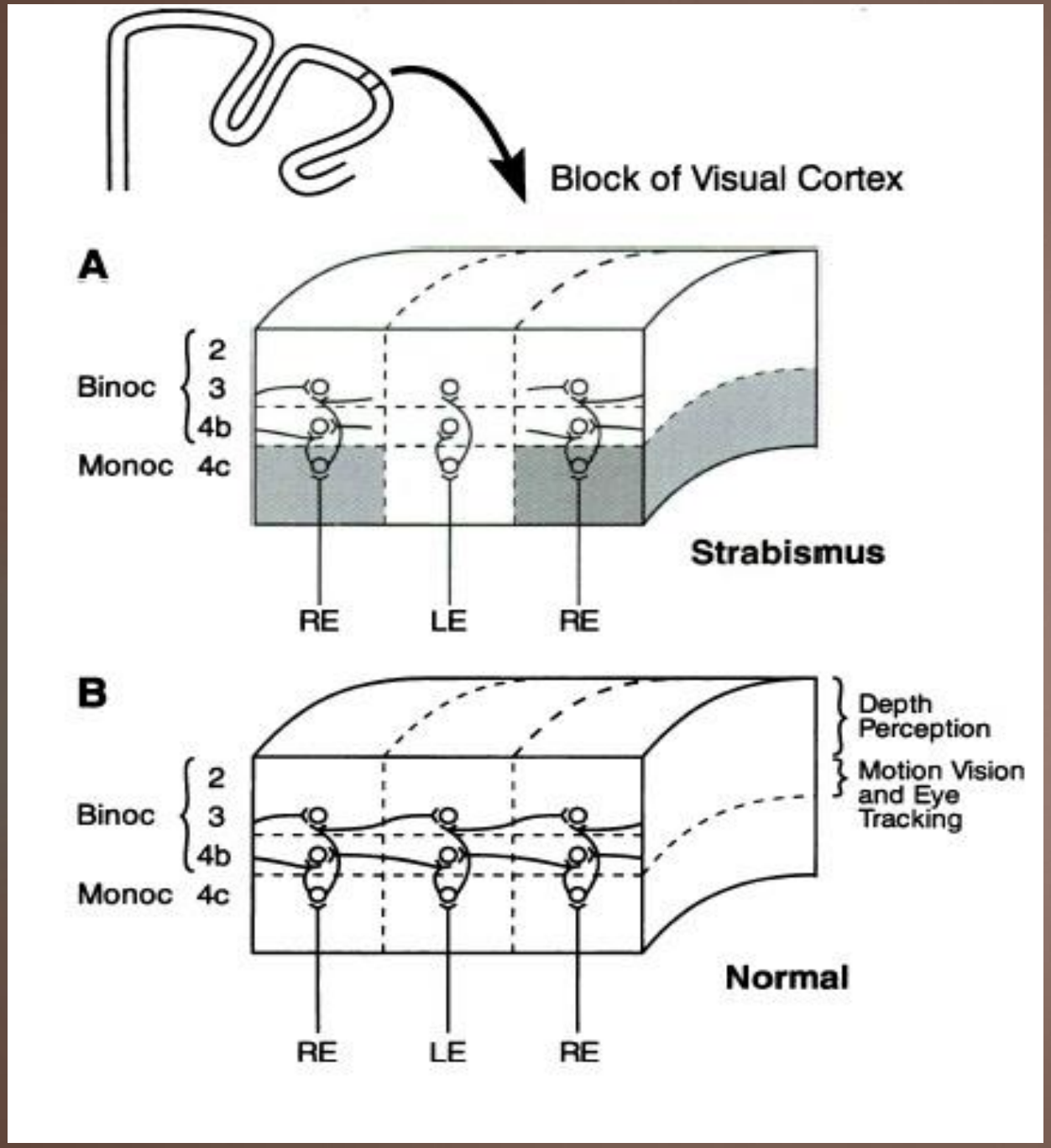
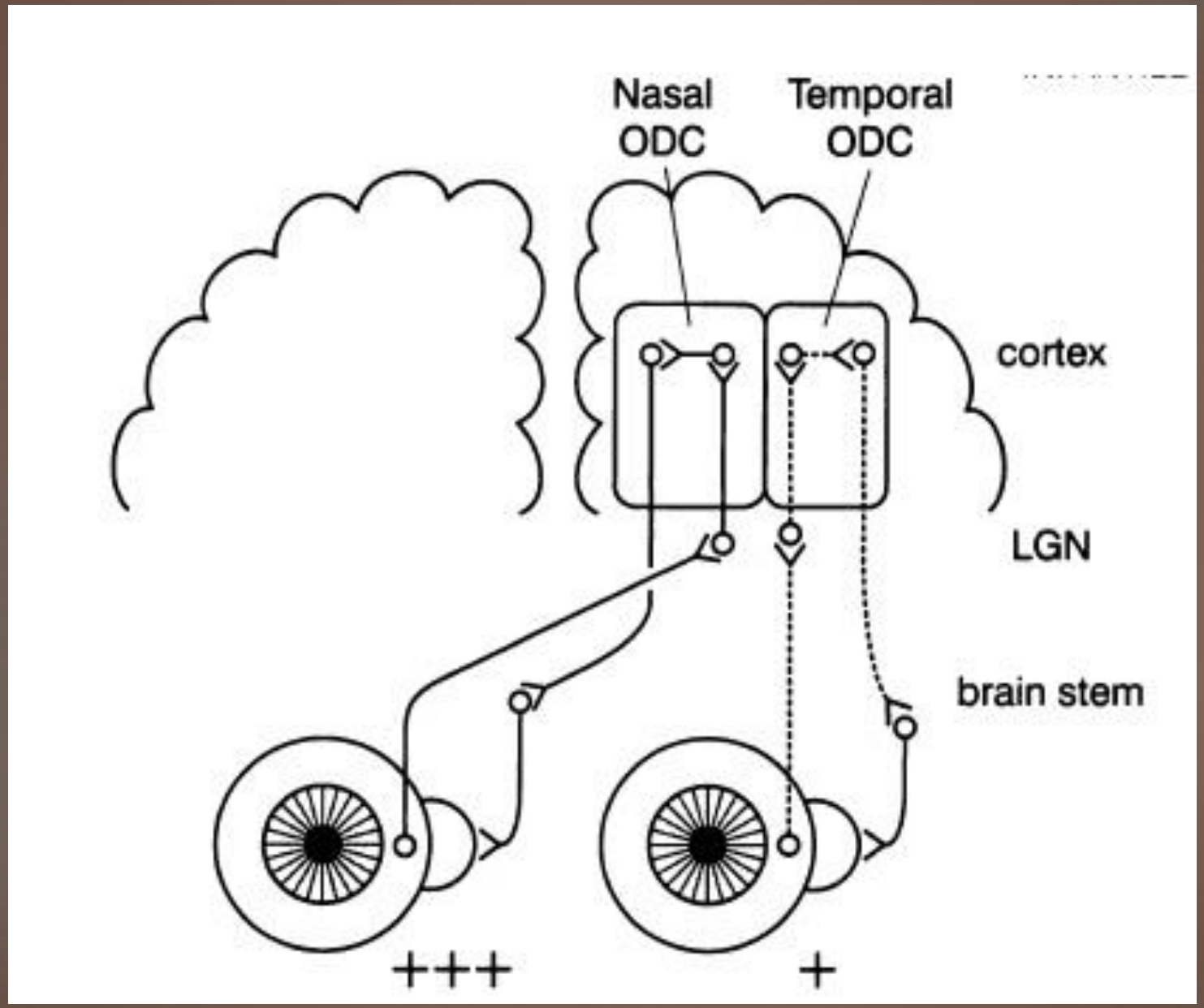


Table 8–2. Functional-Structural Correlation in Infantile Esotropia

Functional Deficit	Structural Deficit
Lack of fusion and binocular visual-evoked potential	Lack binocular connection layers 2 & 3
Asymmetric pursuit and motion visual-evoked potential	Lack binocular connection layer 4b
Alternating suppression	Inhibition metabolic activity layer 4c


Nasal
ODC
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cortex
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brain stem
+++
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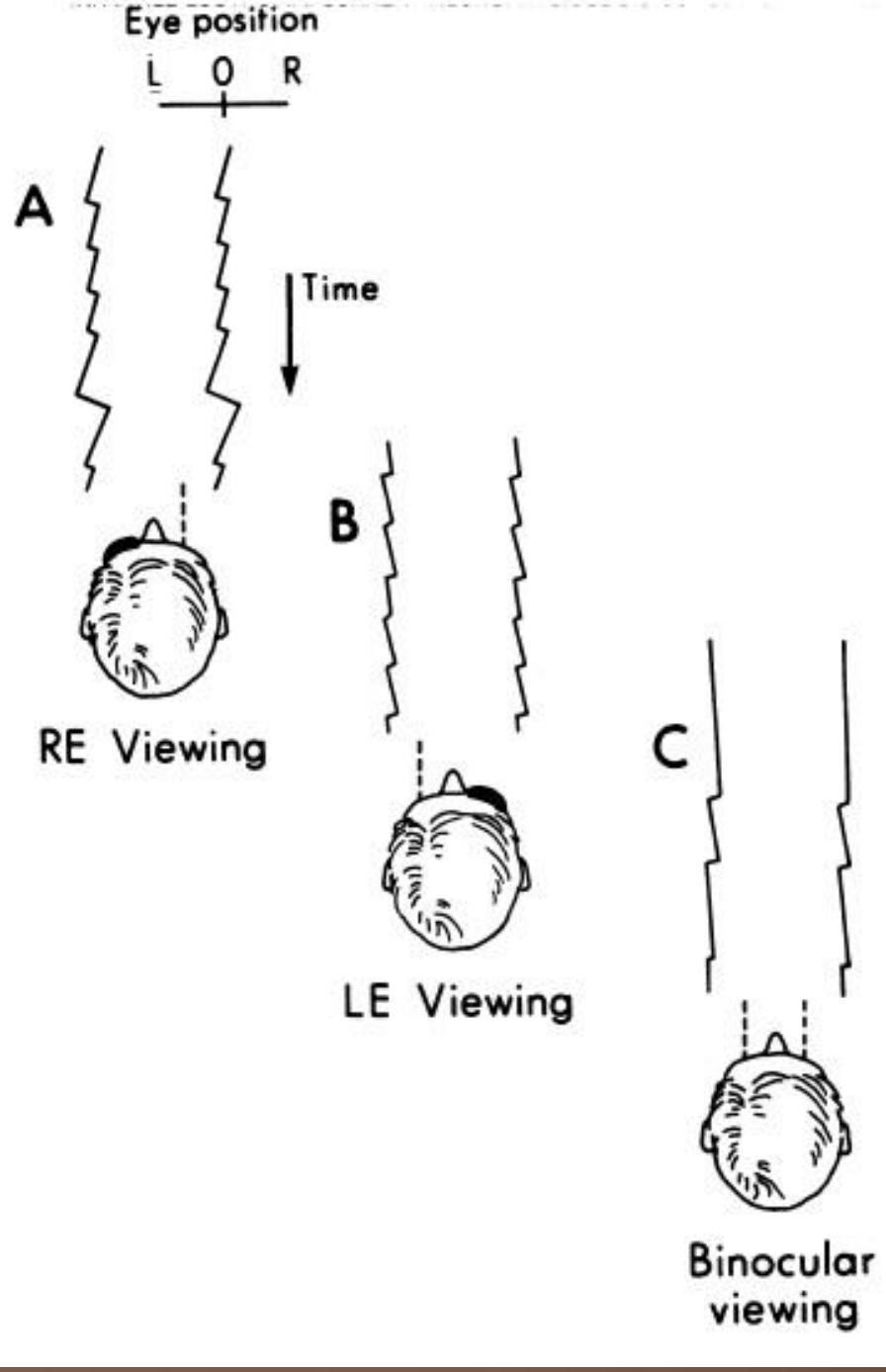
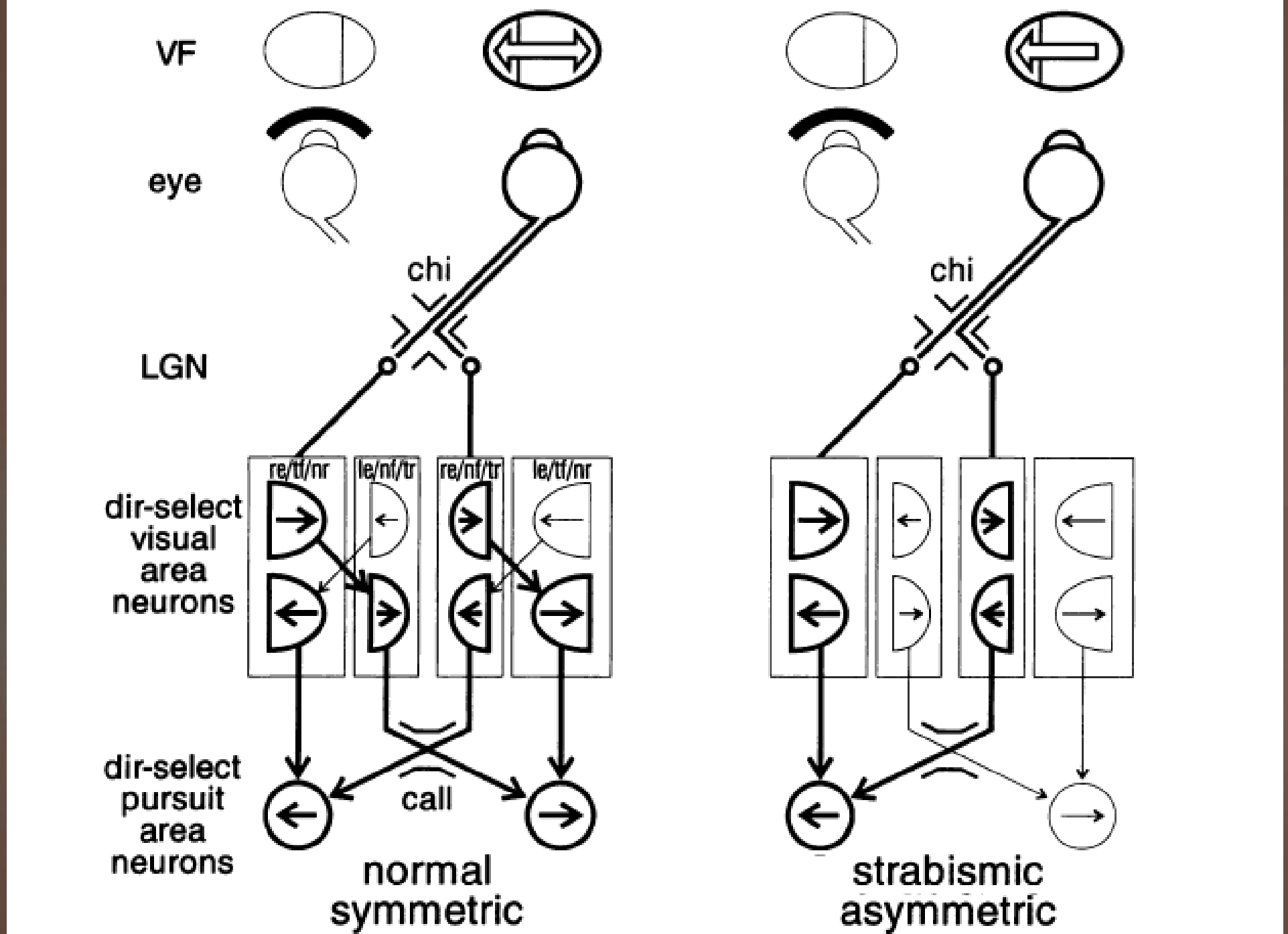


Figure 8–11. *Latent fixation nystagmus in human who had interruption of binocular development during the critical period. A, Viewing a stationary target with the right eye; the left eye is occluded. The eyes drift conjugately to the left, in a nasal direction with respect to the fixating eye. Corrective fast-phase jerks to the right are made. B, Viewing with the left eye, the direction of slow-phase drift reverses so that it is again nasally directed with respect to the fixing eye. Note that the velocity of the drift is different when viewing with the left versus the right eye. C, When viewing binocularly, the velocity of the drift is slow and the nystagmus is less conspicuous ("manifest" latent nystagmus). (For an explanation of the neural mechanism, see Figure 8–2.)*


VF
eye
chi
LGN
re/tf/nr
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re/nf/tr
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dir-select visual area neurons
dir-select pursuit area neurons
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normal symmetric
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strabismic asymmetric

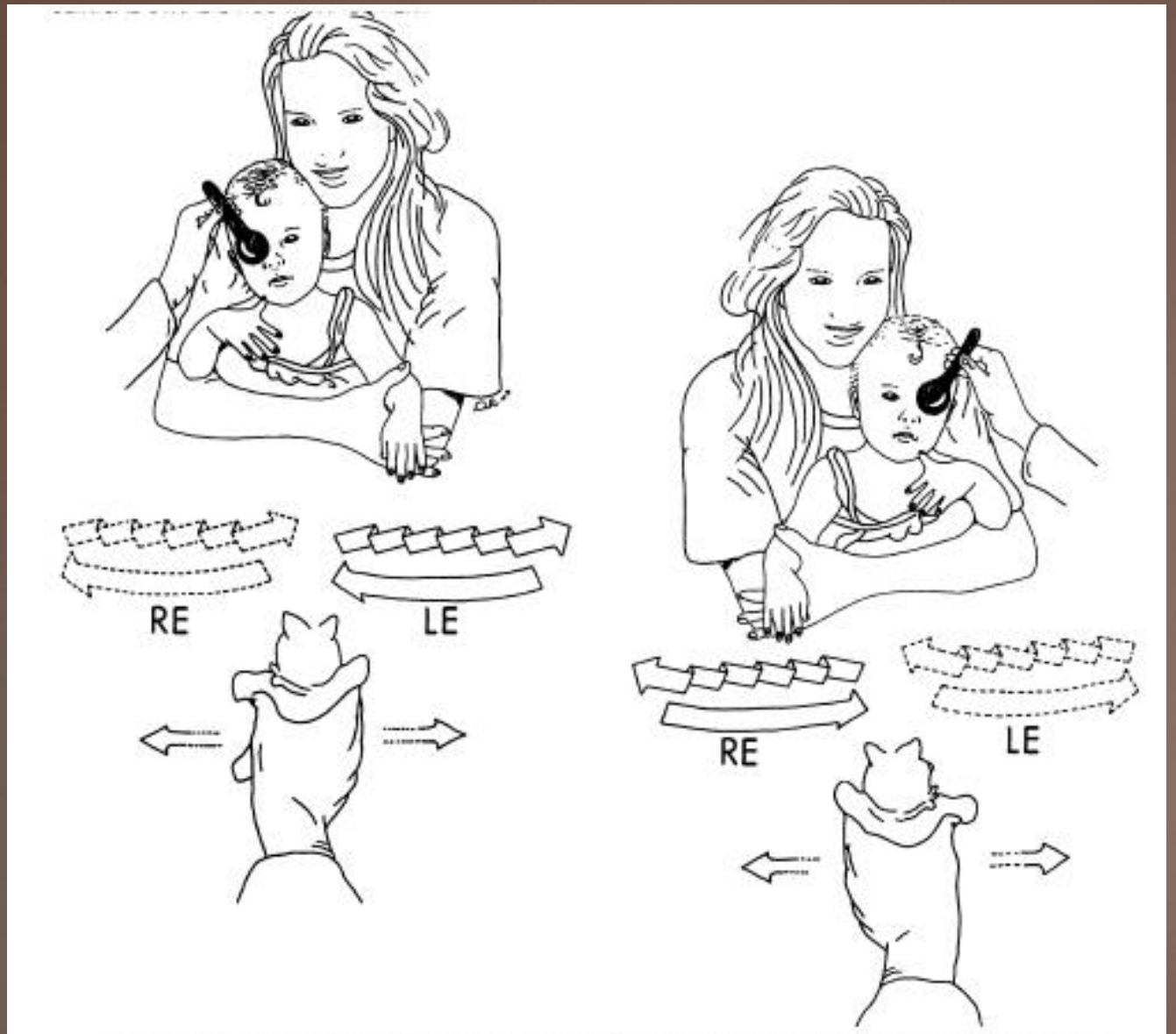


Figure 8–10. *Asymmetry of horizontal smooth pursuit evident during monocular viewing. When a hand-held toy is moved from temporal to nasal before the fixating eye, pursuit is smooth. Pursuit is absent or cogwheel when the target moves nasal to temporal. The movements of the two eyes are conjugate, and the direction of the asymmetry reverses instantaneously with a change of fixating eye, so that the direction of robust pursuit is always for nasally directed targets in the visual field. The asymmetry is seen best by moving the target at a brisk pace. The asymmetry indicates immaturity of binocular connections for pursuit in visual cortex (see Fig. 8–2). Dashed lines indicate conjugate movements of the eye under the cover.*

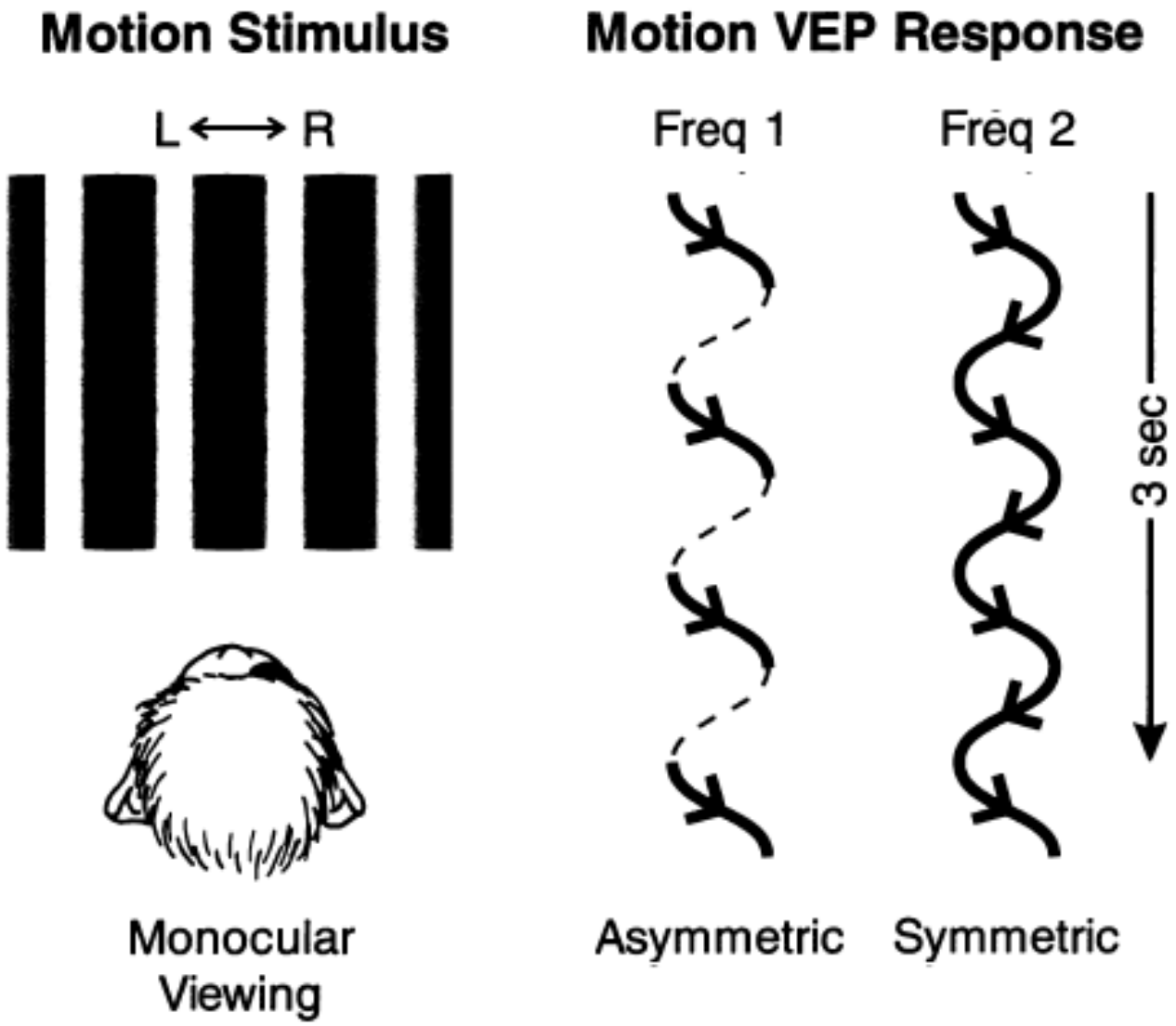
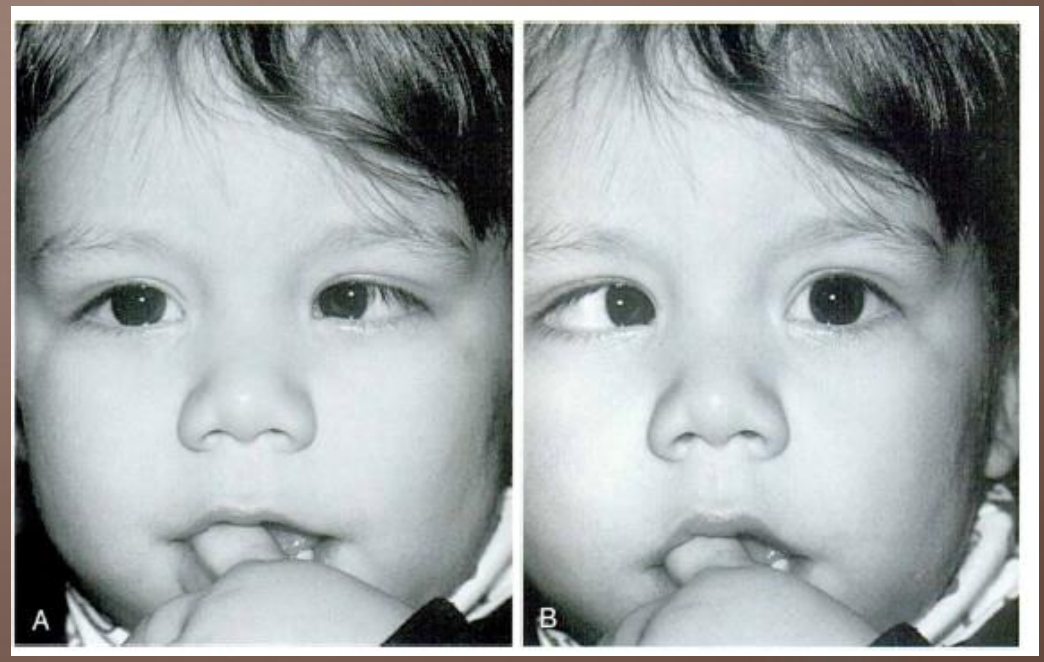


Figure 8–13. *Motion visual evoked potential stimulus and response. The stimulus was a vertically oriented sinusoidal-contrast grating that oscillated left and right several times per second on a video screen. F1 (asymmetric) signals are evoked from cortical motion neurons in young normal infant human and monkey (shown) before development of binocular vision. A strong F1 response persists in uncorrected infantile strabismus. F2 (symmetric) signals represent the response of children and adults who have normal binocular vision. Time in graph on right progresses from top to bottom.*

EPIDEMIOLOGY AND RISK FACTORS:

- Both genetic and environmental factors appear to play a role in the causation of esotropia
- As an example of genetic factors, in the study reported by Tychsen and Lisberger" in 1986, the strabismic subject who had the most severe pursuit/motion processing asymmetry had two siblings with infantile strabismus.
- Nonstrabismic kindred in pedigrees of infantile strabismus have been found who manifest nasally directed biases of pursuit not present in the normal population." Large-scale studies have documented that 20% to 30% of children born to a strabismic parent will themselves develop strabism.

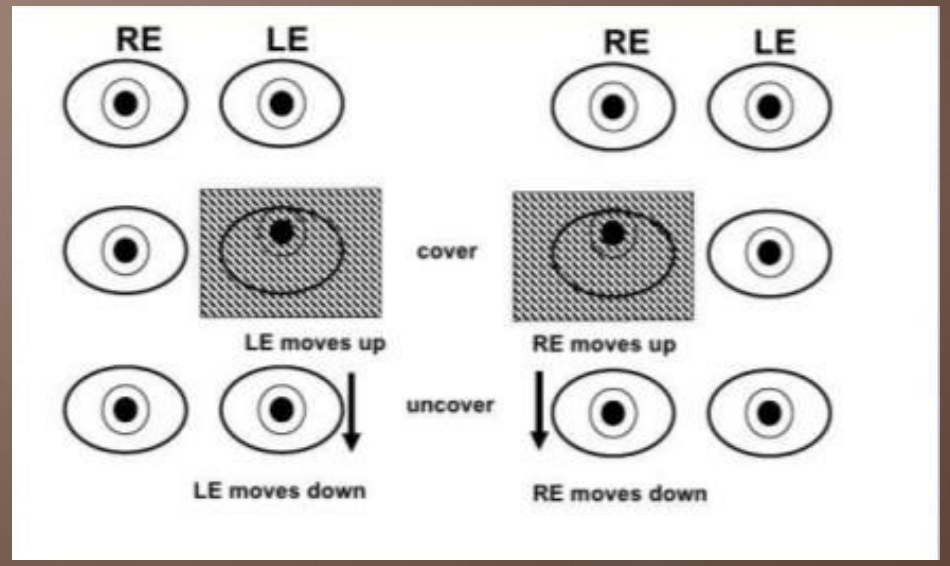
- As for environmental factors, the prevalence of strabismus and amblyopia is substantially higher in low-birth-weight, premature infants". " " " " or those who suffer perinatal hypoxia
- The increased risk of strabismus in these infants is probably due to the maldevelopment of binocular connections in the visual cortex and the downstream effects of this damage on cerebral ocular motor-related neurons.
- effects of this damage on cerebral ocular motor-related neurons. The occipital lobes in newborns are especially vulnerable to damage from hypoxia." "The striate cortex is susceptible to hypoxic injury because it has the highest neuron-to-glia ratio in the entire cerebrum" " and the highest regional cerebral glucose consumption.



- DISSOCIATED VERTICAL OR HORIZONTAL DEVIATION:

- Dissociated vertical deviation (DVD) is characterized by an upward-directed slow movement of the nonfixing eye.
- The hallmark of the deviation is that it violates Hering's law of equal innervation. The fixing eye does not move, or moves minimally, whereas the eye with the DVD is moving up under cover and down when uncovered as much as 10 degreees.
- DVD has been subdivided into several variants:
- dissociated hypertropia,
- dissociated hyperphoria, and
- dissociated horizontal (exo)deviation.

- Studies of pursuit and motion perception in individuals with DVD have revealed vertical asymmetries analogous to those that are horizontal.
- Patients who have DVD are more sensitive to upward-directed motion, measured as better pursuit of upward-directed moving targets and misperception of downward target velocities.“
- The reported prevalence of DVD in infantile esotropia ranges from 76% to 88%. " It is nearly always 132 e CLINICAL STRABISMUS MANAGEMENT bilateral, but of differing magnitude in the two eyes. It is rarely detected in infants.
- Typically DVD appears in pre school-age and school-age children who have had horizontal muscle surgery to correct esotropia earlier in life.



NEUROANATOMIC FINDINGS IN INFANTILE ESOTROPIA:

- Hubel and Wiesel," in 1977, reported a series of experiments in mon keys describing the functional architecture of the normal primary visual cortex (striate cortex or area V1)
- The primary cortex can be divided into layers containing neurons with different response properties, organized in columns such that alternating columns receive input from only the right or the left eye. When signals were recorded from these neurons, the majority responded to both eyes, implying the presence of binocular connections between ocular dominance columns (ODCs), although the binocular connections themselves could not be visualized using available anatomic methods.

Table 8–1. Functional Deficits in Infantile Esotropia

Fusion Deficits
Absence of disparity vergence (motor fusion)
Lack of two-dimensional fusion and stereopsis
Alternating monocular suppression
Subnormal binocular visual-evoked potential response
Motion/Pursuit Deficits
Asymmetric monocular tracking
Asymmetric monocular motion visual-evoked potential
Asymmetric motion perception

Clinical Characteristics:

- CLASSIC PRESENTATION:
- The paradigmatic infant who develops strabismus begins to manifest a chronic esodeviation of the visual axes at 2 to 4 months of age.
- Transient episodes of misalignment may precede this by several weeks and may account for the history often given by the parents that the eyes crossed “at birth.” Chronic esotropia in the neonatal period is rare.‘
- If it is well documented by good serial photographs or ophthalmologic examination, the major concern is Duane syndrome or neonatal sixth nerve palsy, not classic infantile esotropia
- Classic infantile esotropia is constant and cosmetically conspicuous, typically exceeding 20 PD on corneal light reflex measurement.

SPECTRUM OF CLINICAL PRESENTATIONS:

- A substantial proportion of infants who develop strabismus becomes esotropic beyond age 2 to 4 months. As many as 10% may not display a constant esotropia until as late as 9 to 12 months of age. These children usually do exhibit an intermittent esodeviation earlier in infancy.
- The magnitude of the strabismus may increase in the first few weeks or months of observation, and the angle can vary depending on the level of attention.
- Incomitance may also be observed; the most common type is a V pattern, in which esotropia is greater in downgaze and less in upgaze.
- V-pattern infantile esotropia is commonly, but not invariably, associated with overaction of the inferior oblique muscles.
- Other variants the clinician may encounter include either a combination of refractive (hypermetropic) and “baseline” infantile esotropia or high accommodative convergence/ac commodation (“high AC/A”) esotropia with infantile esotropia.

Diagnosis:

- Infants who develop strabismus begin to exhibit a constellation of ocular motor signs:
- 1) esotropia, with or without strabismic amblyopia;
- (2) pursuit asymmetry;
- (3) latent fixation nystagmus;
- (4) motion visual-evoked potential (VEP)asymmetry and motion perception abnormalities;
- (5) a face turn and abduction deficit; and
- (6) vertical deviation.

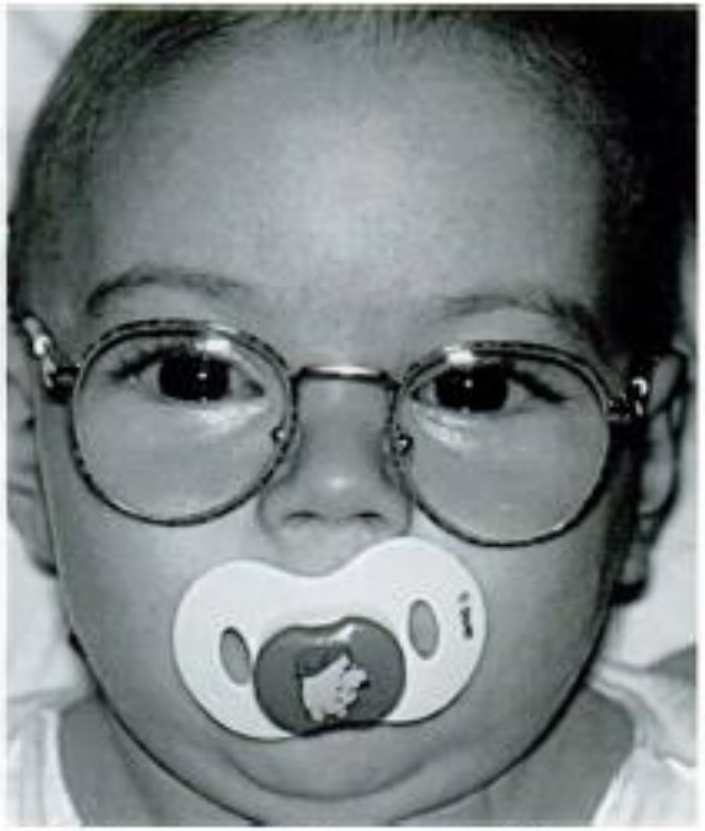
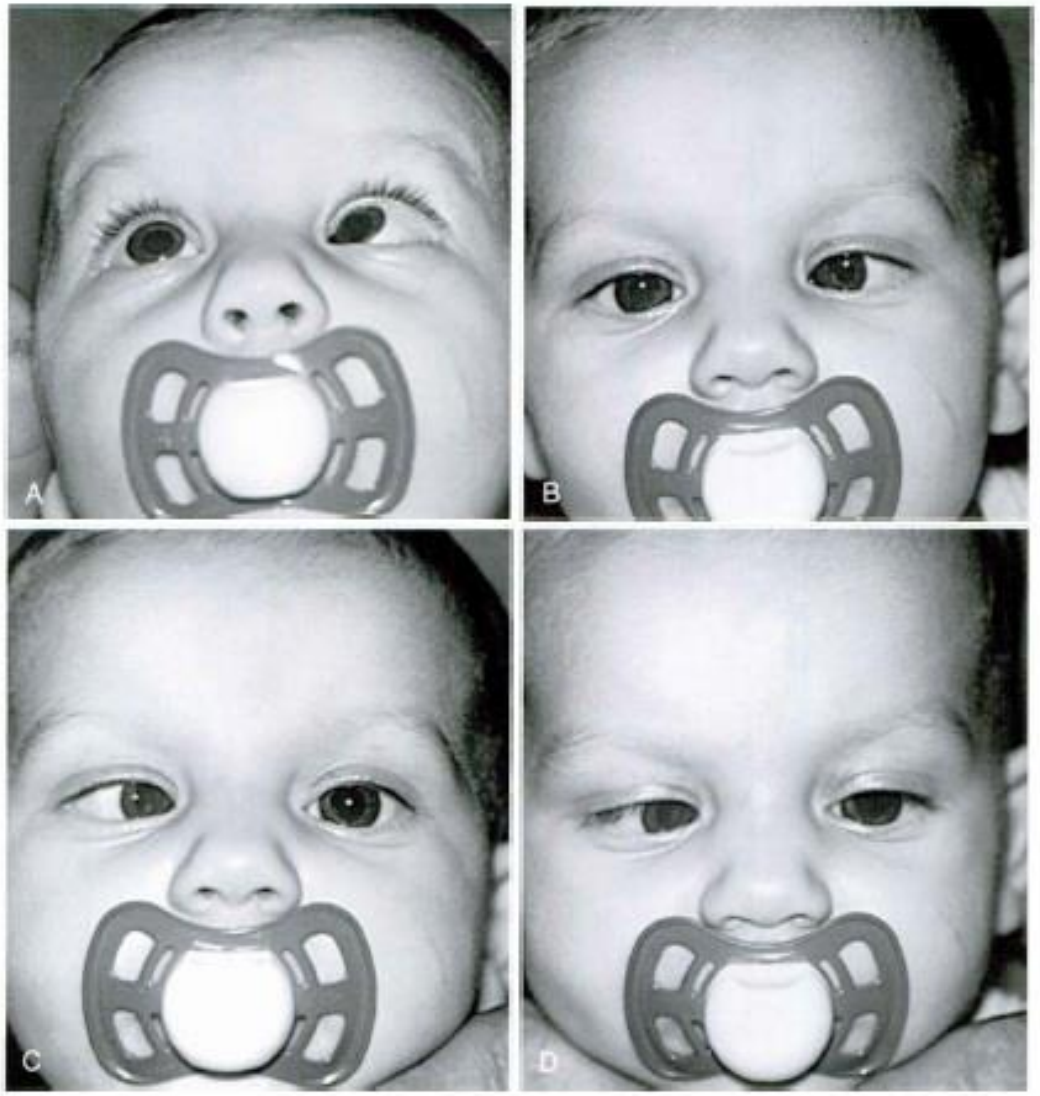


Figure 8–8. *Nine-month-old infant who had infantile esotropia and superimposed refractive esotropia (refractive error = +4.50 D in both eyes). Spectacles for the full amount of the refractive error were prescribed, which reduced the total magnitude of the strabismus by about half—from 50 PD to 25 PD. Surgery was performed for the nonrefractive component of the esotropia (i.e., 25 PD).*



PURSUIT ASYMMETRY:

- Infants in whom normal binocularity fails to develop exhibit asymmetric horizontal pursuit.
- When one eye is occluded and a hand-held toy is moved from temporal to nasal before the fixing eye, pursuit is smooth .Pursuit is absent or jerky (cogwheel or low gain) when the target moves nasal to temporal.
- The movements of the two eyes are conjugate, and the direction of the asymmetry reverses instantaneously with a change in the fixing eye, so that the direction of normal pursuit is always nasally directed with respect to the fixing eye. Infants with the maldevelopment may appear to ignore temporally directed targets.

LATENT FIXATION NYSTAGMUS:

- Infants in whom normal binocularity fails to develop display a fixation nystagmus. When attempting to fixate a small stationary target, the eyes drift nasally with respect to the fixing eye (the velocity of the slow drift and the number of corrective fast-phase jerks are accentuated by covering the nonfixing eye, hence the term latent).
- As is true with pursuit asymmetry, the direction of the nystagmus reverses instantaneously with a change in the fixing eye: the direction of the slow drift is always nasally directed with respect to the fixing eye, and the movements of the two eyes are conjugate
- The nystagmus persists into adulthood despite surgical correction of strabismus and thus serves (as does the pursuit asymmetry) as a permanent marker of abnormal binocular motion neuron development

MOTION VISUAL-EVOKED POTENTIAL ASYMMETRY:

- Motion VEPs provide additional evidence that the directional asymmetry of pursuit and latent nystagmus is due to cortical maldevelopment." "Esotropic infants have asymmetries in their VEP response to horizontally oscillating stimuli (Fig. 8–13), responding robustly to only one direction of horizontal motion when viewing monocularly.
- The responses are directionally inverted by 180 degrees in the two eyes, analogous to the nasotemporal asymmetry of eye movements and motion perception. The motion VEP asymmetry tends to resolve in esotropic infants who have early surgical realignment of the eyes but persists in children and adults with uncorrected esotropia.
- "The motion VEP asymmetry is not present in children who develop strabismus after infancy. It serves as an additional diagnostic marker for maldevelopment of binocular vision and an indicator documenting repair of the maldevelopment after early strabismus surgery.

FACE TURN AND ABDUCTION DEFICIT:

- Infants with latent fixation nystagmus and the pursuit asymmetry prefer to view targets by placing the eye at a nasal position in the orbit. This is achieved by turning the face toward the fixing eye.
- Eye movement recordings indicate that, with the fixing eye in the nasal orbit, the velocity of nystagmus decreases an average of 25%." "The reduced nystagmus velocity improves visual acuity.
- A consistent face turn in one direction often indicates amblyopia in the eye that is in a more temporal position in the orbit (the left eye in an infant with a right face turn).
- Infants who have esotropia may appear as though they have limited abduction

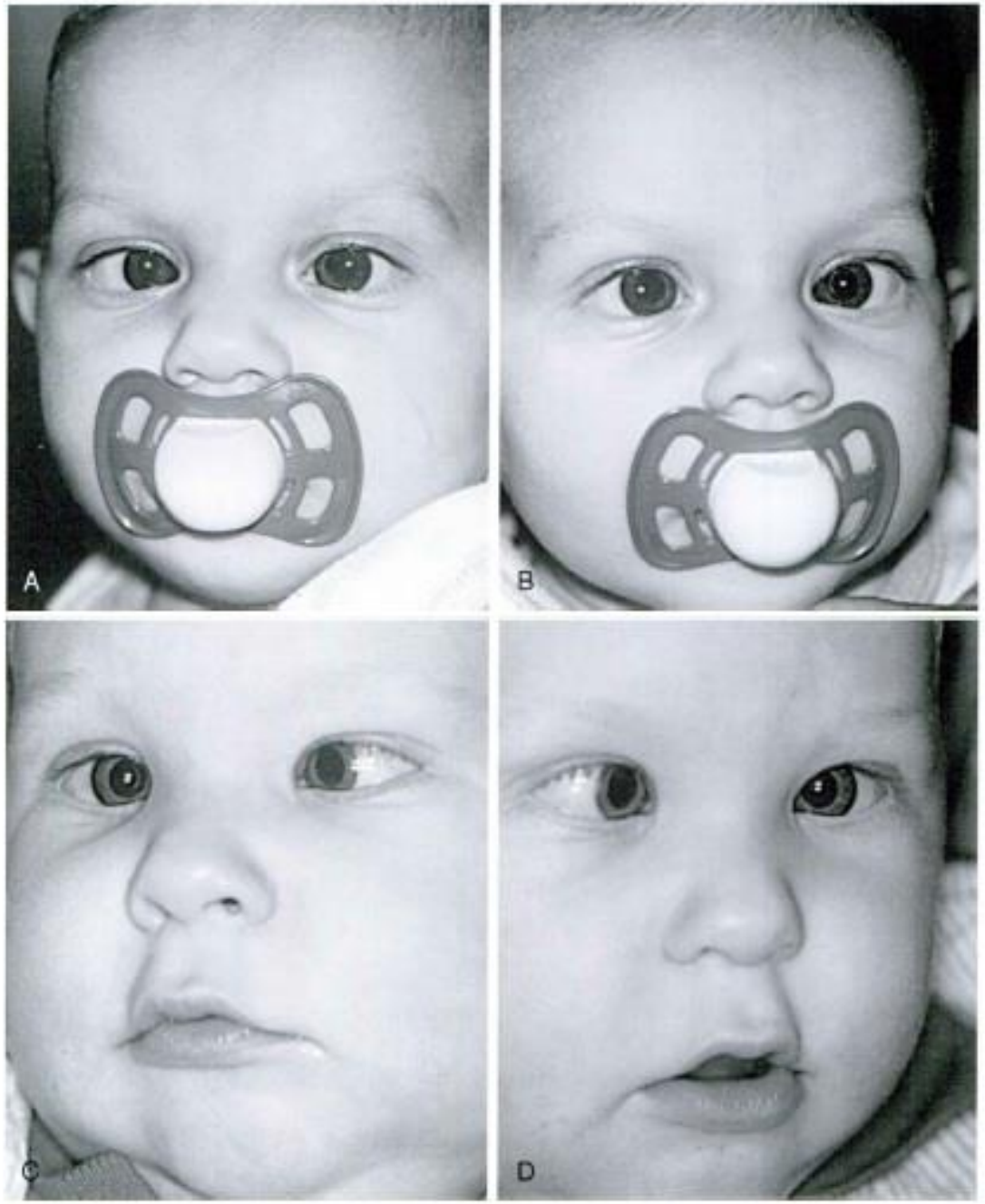


Figure 8–14. *Infants who have latent nystagmus and the pursuit asymmetry prefer to view targets by placing the eye at a nasal position in the orbit. This is achieved by turning the face toward the fixing eye, which reduces nystagmus velocity. A, A 6-month-old infant with esotropia who turns the face to the right when viewing with the right eye and to left (B) when viewing with the left eye. A 7-month-old infant with esotropia viewing with (C) right eye and (D) left eye. Alternating face turn implies near equal acuity in the two eyes. A consistent face turn in one direction often indicates amblyopia in the eye that is opposite the direction of the face turn (the left eye in an infant with a right face turn).*

CONSTANT INFANTILE EXOTROPIA: A NEURO-OPHTHALMIC DISORDER:

- The ophthalmologist must be particularly diligent in ruling out neuro-ophthalmic abnormalities in any infant presenting with constant exotropia, as opposed to esotropia, in the first 12 months of life.
- This dictum does not apply to infants who display early-onset intermittent exotropia, nor does it apply to normal infants younger than 3 to 5 months of age who display a transient physiologic exodeviation in early infancy.
- The ratio of infantile esotropia to constant infantile exotro pia at our institution is greater than 10:1.
- Unlike the majority of infants with esotropia, more than 90% of those with constant exotropia have significant eye or brain abnormalities such as optic nerve hypoplasia, morning glory anomaly of the optic disc, retinoblastoma, microcephaly, infantile spasm, encephalomalacia, or static encephalopathy

- Thus, constant exotropia in infancy should be considered unusual enough to warrant careful neuro-ophthalmic examination for a relative afferent pupillary defect, a visual field defect (which may be tested using the evoked-saccade method"), ptosis or other evidence of third nerve palsy,anomalous optic discs, nerve fiber layer loss, a history of seizures, or failure to thrive

Figure 8–15. *Four-month-old infant who presented with constant, large-magnitude (50 PD) exotropia. Infant proved to have no structural lesion of the eyes or abnormality of the brain on neuroimaging, but follow-up revealed delayed somatic motor development and hypotonia. Strabismus surgery was performed at age 6 months.*

Treatment:

- NONSURGICAL MANAGEMENT:

Glasses:

A good refraction with full cycloplegia (e.g., using 2.5 phenylephrine [Neo-Synephrine] and 1% cyclopentolate) should be performed on all esotropic infants.

- Spectacles are generally prescribed when the degree of hyperopia exceeds +2.50 D and/or when anisometropia exceeds + 1.50 D.
- Any cylinder of +0.50 D or more should also be given.
- Spectacles should be prescribed for myopia exceeding -4.00 D.

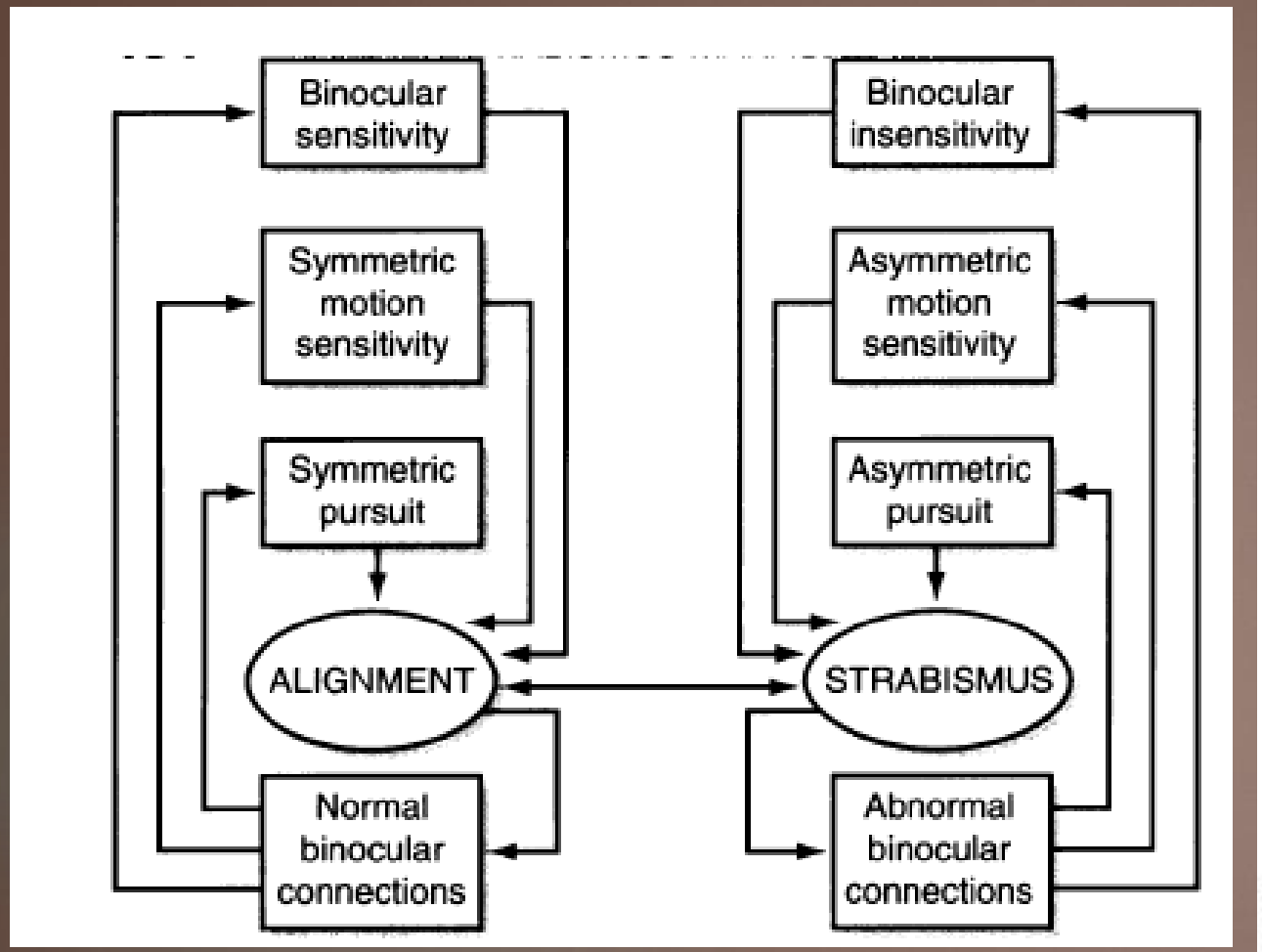
- Occlusion Therapy for Amblyopia:
- If amblyopia is detected, occlusion therapy is institute after the first office visit (Fig. 8–16). If a strong fixation preference for one eye is detected, high-percentage occlusion (e.g., 90% of waking hours) is prescribed using opaque skin patche (Opticlude or Coverlet).

- SURGICAL MANAGEMENT :

Rationale for Early Surgical Correction

Careful psychophysical experiments found that a substantial propor tion (41%) of infants whose eyes were aligned to within 8 PD in the first 16 months of life had the restoration of random-dot stereopsis on follow-up years later and that those whose eyes were aligned at 12 months who achieved stereopsis (49%) tended to achieve a finer grade.

In addition to stereopsis, it appears that the defects in the motion pathway can also be repaired in a substantial number of strabismic infants." " " " The bar graph shows data from two groups of human infants who were operated on before age 18 months. Testing 3 to 6 months postoperatively showed that infants whose eyes were aligned within 10 PD of orthotropia tended to show a return of symmetric motion sensitivity, a finding not as apparent in infants whose eyes were poorly aligned.



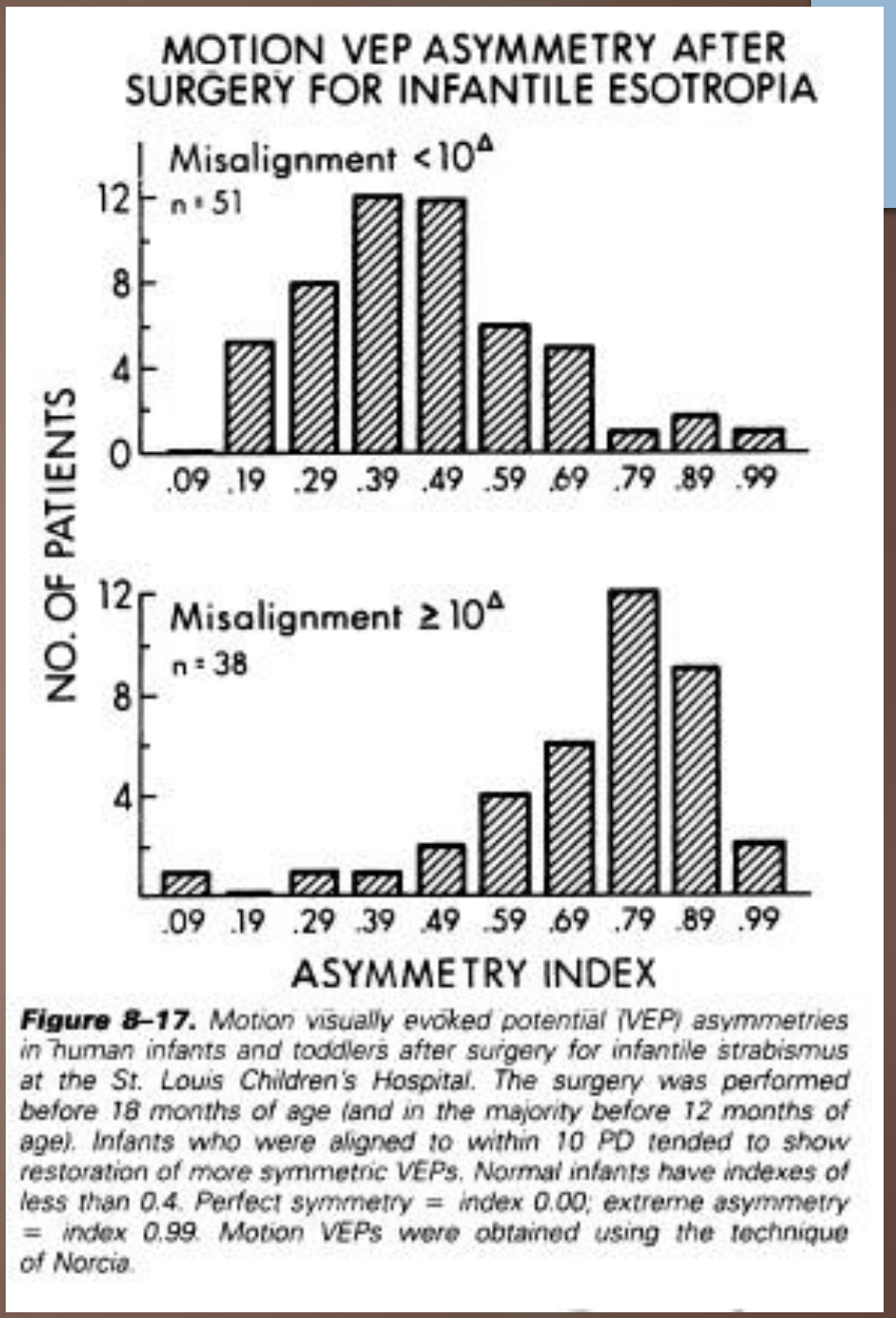


Figure 8–17. *Motion visually evoked potential (VEP) asymmetries in human infants and toddlers after surgery for infantile strabismus at the St. Louis Children's Hospital. The surgery was performed before 18 months of age (and in the majority before 12 months of age). Infants who were aligned to within 10 PD tended to show restoration of more symmetric VEPs. Normal infants have indexes of less than 0.4. Perfect symmetry = index 0.00; extreme asymmetry = index 0.99. Motion VEPs were obtained using the technique of Norcia.*

Surgical Timing and Preoperative Measurements:

- When the surgeon has documented that the infant has a constant esotropia exceeding 12 PD, surgical realignment should be carried out as soon as is practical for the surgeon and family (assuming there are no major cardiopulmonary problems that would pose a high risk for general anesthesia).
- Ideally, the angle of the strabismus is measured using the alternate prism cover test to gauge the full magnitude of any combined esotropia and esophoria. Prism cover testing is done carefully for distance and near fixation in primary position wearing any prescribe spectacle correction.

Preoperative Counseling:

- A neurophysiologic basis for correcting esotropia was addressed in the preceding discussion. The surgeon should also provide a physiologic rationale to the family: the opportunity for brain repair with some recovery of three-dimensional vision, motion vision, accurate eye tracking, reduction of nystagmus, and elimination of conflicting images that promote amblyopia.
- The parents should be told up front of the possibility that reoperation may be needed in the months and years ahead. Infants with esotropia have required, on average, 1.9 to 2.6 operations to achieve stable alignment with some motor fusion.

Anesthetic and Operative Considerations:

- Infants who were markedly uncooperative during office examinations may benefit from a brief examination under anesthesia at the beginning of surgery.
- After antiseptic preparation and draping, forced duction testing is done to rule out restrictive myopathy.
- The surgical strategy most frequently employed is recession of both medial rectus muscles. For esotropia greater than 60 to 70 PD, botulinum toxin may be injected under direct visualization into one of the maximally recessed muscles to augment the effect of the recession."
- If the infant displays an A or V pattern of 15 PD or more, the medial rectus muscle tendons can be displaced vertically relative to their normal insertions or, if the pattern is accompanied by substantial oblique muscle overaction, oblique muscle weakening is carried out in lieu of transposition

Follow-Up Regimen:

- At the first postoperative visit, typically 3 to 10 days after the procedure, the surgeon checks the visual acuity, rules out an afferent pupillary defect, and ensures that a good red reflex is visible from both fundi.
- In infants, alignment is assessed using the Krimsky or Hirschberg method, and versions are examined to verify the absence of gross underaction and a "slipped muscle.
- A second postoperative appointment is scheduled for 3 to 4 months hence. If amblyopia is present, occlusion therapy can be reinstituted. If marked overcorrection or undercorrection is noted, the child may be seen sooner, but reoperation seldom will be seriously considered until 3 to 4 months have elapsed.
- Reoperation is performed when a constant or poorly controlled intermittent esotropia or exotropia exceeding 12 PD is detected. Reoperation also is indicated for conspicuous oblique muscle overaction, DVD, or dissociated horizontal deviation.

Differential diagnosis:

Pseudoesotropia

Congenital sixth nerve palsy

Nystagmus blockage syndrome

Type I Duane's syndrome

Ciancia syndrome

Congenital fibrosis syndrome

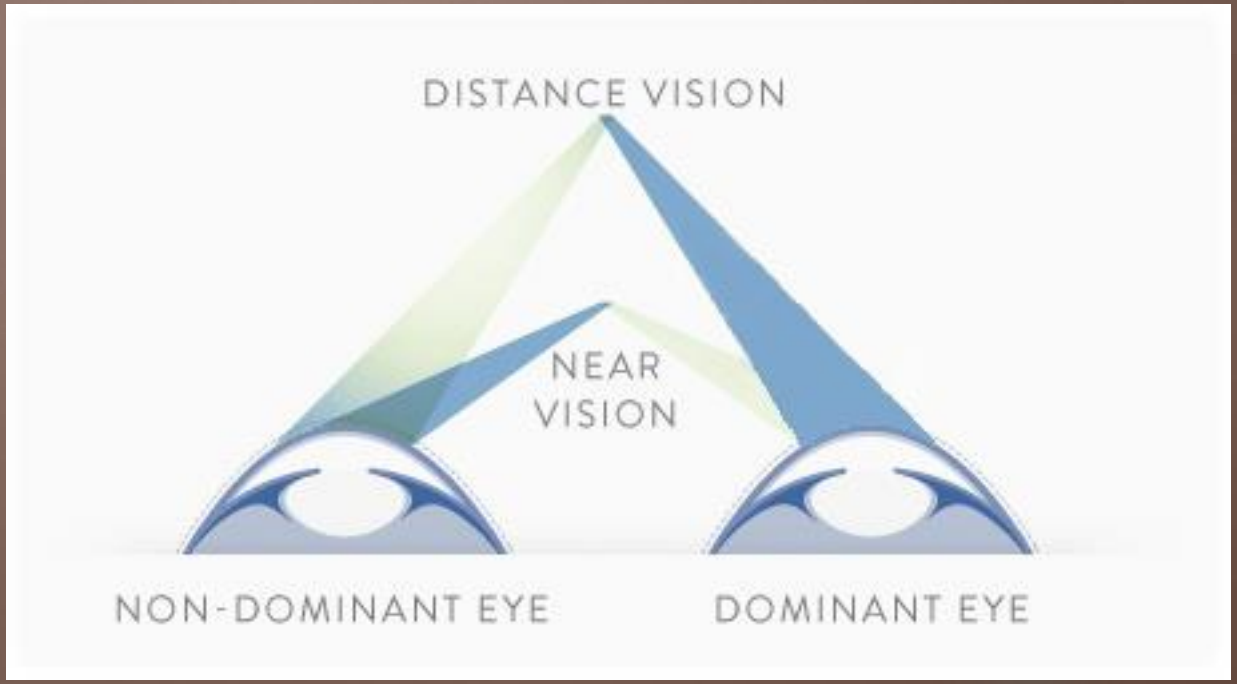
Mobius syndrome

Infantile Myasthenia Gravis

Associated with neurologic diseases e.g. cerebral palsy, periventricular encephlomalasia

Divergence paralysis:

- Divergence insufficiency is a rare ophthalmologic disorder manifesting itself among older adults. Primary and secondary forms exist, with the latter more urgently addressed due to neurologic comorbidities. Ultimately, the diagnosis of DI, particularly in the primary form, tends to be elusive. This article will review the typical presentation, diagnosis and treatment options, and report a case of primary DI, along with the often complex consideration leading to this diagnosis.



Background:

- Divergence insufficiency can vary in severity, from minor deficits to complete divergence paralysis. Similarly, the theories on mechanism of divergence itself have varied

1. Drs. Bielchovsky and Duane favored the presence of a dedicated active divergence center
2. while Drs. Bergman, Pugh and Duke-Elder favored the view of divergence as a passive result of relaxation of convergence.

- Upon review of the literature, Alexander Duane can be credited with the first comprehensive description of this entity

- Dr. Duane suggested this diagnosis required 2 to 8 degrees of esophoria at distance and only slight esophoria or even exophoria at near
- In a 1971 study, authors reported on the magnitude of misalignment, ranging from 8 to 30 prism diopters at distance, but only 4 to 18 prism diopters at near
- The importance of measuring the magnitude of alignment defect in different directions of gaze was underscored in a 1947 study,5 allowing clinicians to distinguish between external rectus paresis/paralysis and DI due to other causes

- Among patients with high myopia the presence of a long axis has been associated with development of DI,2 likely due to altered angles at which the extraocular muscles insert and exert their force on the globe.
- Another historically reported feature of DI is significantly decreased negative fusional vergence (fusional divergence), along with the deficit's direct relationship to distance of gaze. This is a feature useful in differential diagnosis of DI particularly from other, more ominous conditions like divergence paralysis.

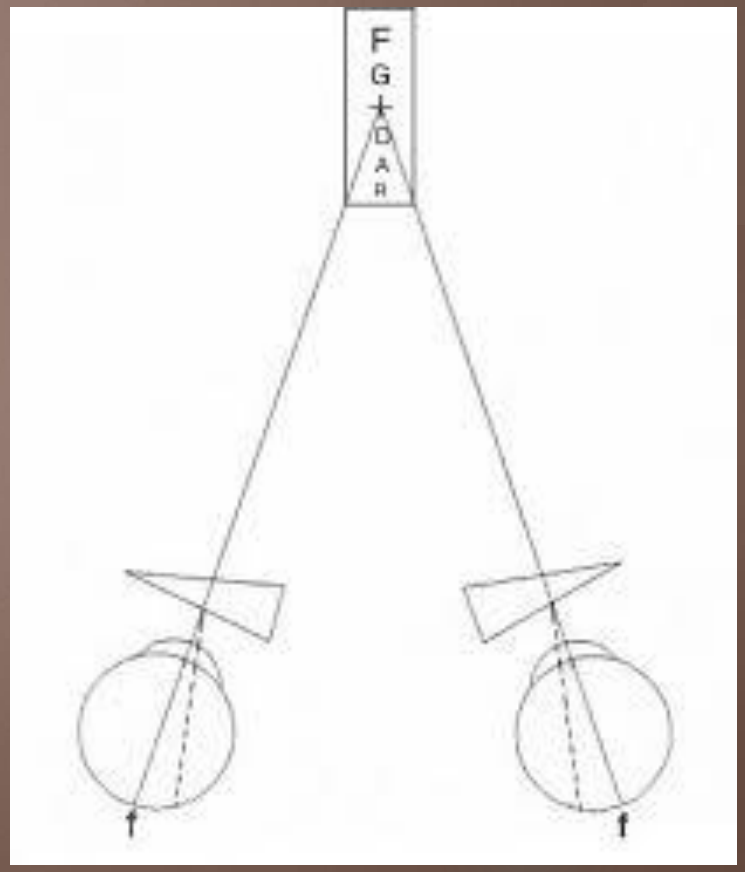


Table 1. Evolution of Diagnostic Criteria for Divergence Insufficiency

Author	Diagnostic Criteria	Comments
Duane, A (1937)	ET 2-8 Δ at distance Slight ET or XT at near Significantly decreased fusional divergence	Magnitude of deviation depends on distance of gaze
Moore, S (1971)	ET 8-30 Δ at distance ET 4-18 Δ at near	
Scheiman, M (1988)	Fusional divergence	Good for differentiating DI from other conditions
Berscheid C (2005)	Fusional divergence	Not helpful in differentiating primary from secondary DI

Typical Presentation:

- Patients with DI typically complain of gradual onset, variable frequency, homonymous diplopia, which is worse at distance. Sometimes it is exacerbated by fatigue and improves with rest.
- Other associated symptoms can include asthenopia of panoramic type,5 motion sickness, headaches or sensitivity to light.
- Early presbyopia is also a frequent comorbidity.
- Nausea and headaches are reported to be uncommon among patients with the primary form of DI, as are any history of head trauma or intracranial pathology.
- The pattern of gradual onset is an important distinction from usually more sudden sixth-nerve palsy
- a common item in the differential of DI, as is the absence of papilledema or endpoint nystagmus

- Evidence of sudden onset or rapid progression should also point the clinician towards secondary DI and re-doubled efforts to find the underlying neurologic abnormality
- The association with refractive errors (and high myopia in particular) has been a point of contention for some time,6 with most recent experiments attributing this association to specific anatomic differences among high myopes with DI and high myopes without diplopia.2 Specifically, the former group had the superior rectus shifted nasally, lateral rectus-inferiorly, in the setting of normal orbital lengths

Treatment Options:

- Treatment options include correction with base-out prisms for distance,5,8 and orthoptic exercises, but surgical options (e.g., medial rectus recession) have also been put forth
- Naturally, all of the above rely on a manifest refraction, particularly to identify high myopes, as well as a meticulously performed measurement of lateral vergence at distance and at near to serve as a starting point.