Clinicians are faced with the challenge of differentiating the etiologies of asthenopia, blur, diplopia, and headaches associated with the use of the eyes by their patients. Oculomotor deficiencies can be one of several possible causes of such symptoms and are the result of defects in the central nervous system, afferent or efferent nerve pathways, or local conditions of a nature so as to impede appropriate oculomotor function. Oculomotor function will be extensively analyzed during phorometry (see Chapter 21) in terms of binocularity and muscle balance after the subjective refraction has been completed. This chapter focuses on clinical procedures that are typically used to analyze oculomotor function before the subjective refraction is performed, though in some cases the practitioner may decide to use a few of these tests after the refraction is determined.

By using primarily handheld devices or instruments, the normality or abnormality of the functions of the ocular muscles are estimated: the iris sphincter and dilator muscles, the eyelid muscles involved in maintenance of the palpebral aperture, the extraocular muscles, and the ciliary muscles. Hence, oculomotor motility is assessed by observation of (1) the pupils and pupillary reflexes, (2) the palpebral apertures and eyelid movements, (3) monocular and binocular eye movements, (4) monocular and binocular eye alignment, and (5) accommodative amplitudes and facility.

THE PUPILS AND PUPILLARY REFLEXES

The pupil controls retinal illumination and determines retinal image quality. The entrance pupil of the eye is formed by refraction of the real pupil by the cornea and is just over 3 mm behind the anterior corneal surface. Retinal illumination is proportional to the square of the pupillary diameter. Depths of field and focus for clear vision are inversely proportional to pupil diameter. The lower limit of pupil size for optimal visual acuity is approximately 2 mm, below which the effects of reduced retinal illuminance and diffraction outweigh the beneficial aspects of an increase in depth of field and reduction of ocular spherical aberration. The entrance pupil also controls blur circle size at the retina for object rays not originating from the far point plane of the eye.

The entrance pupil averages 3.5 mm in diameter in adults under normal illumination but can range from 1.3 mm to 10 mm. It is usually centered on the optic axis of the eye but is displaced temporally away from the visual axis or line of sight an average of 5 degrees. The entrance pupil is decentered approximately 0.15 mm nasally and 0.1 mm inferior to the geometric center of the cornea. This amount of decentration is not distinguished in casual observation or by the clinician's normal examination of the pupils. In general, the diameter of the pupil gradually becomes smaller after about the age of 12 to 18 years. This appears to be a linear relationship in which pupil size for light-adapted and dark-adapted eyes at age 20 (means nearly 5 mm and 8 mm, respectively) both diminish to about 2 mm and 2.5 mm, respectively, at age 80 (see Figure 25-4). The progressive change in size is known as senile miosis and is shown in Table 10-1. It is not totally explained by increased iris rigidity or loss of muscle fibers in the iris, but apparently also includes a progressive delayed latency of response time, indicating some neurological involvement. Aging is presumed to cause a reduction in sympathetic tone. Because it is only the entrance pupil that can be examined noninvasively by the practitioner, in clinical parlance the word "entrance" is dropped from the term and the single word "pupil" is meant to apply to that which is observed, which is the entrance pupil.

Pupil size is always changing in the normal eye because of convergence/accommodation (near or triad response), pupillary responses to light and small, regular oscillations from fluctuations in the sympathetic and parasympathetic nervous systems. Pupil size can be influenced by drugs and medications (see Chapter 12) and is slightly larger in persons with light irises.
TABLE 10-1 Approximate Pupil Diameters According to Age

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Photopic Diameter (mm)</th>
<th>Scotopic Diameter (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>5.0</td>
<td>8.0</td>
</tr>
<tr>
<td>40</td>
<td>4.0</td>
<td>6.0</td>
</tr>
<tr>
<td>50</td>
<td>3.5</td>
<td>5.5</td>
</tr>
<tr>
<td>60</td>
<td>3+</td>
<td>4.25</td>
</tr>
<tr>
<td>70</td>
<td>2.5</td>
<td>3.0</td>
</tr>
<tr>
<td>80</td>
<td>2+</td>
<td>2.5</td>
</tr>
</tbody>
</table>

Pupil size is controlled by smooth muscles innervated by the autonomic nervous system. An effective competition between the radial dilator muscle, which is sympathetically innervated and acts to dilate the pupil, and the annular sphincter muscle, which is parasymptetically innervated and acts to constrict the pupil, determines the pupil size. The parasympathetic innervation has more control over the pupil size, because the sphincter muscle is the stronger of the two. The dilator muscle acts in opposition such that mydriasis occurs when the sphincter tone is released. As a result, constriction of the pupil to light (myosis) is slightly faster than dilation (mydriasis) when the light is extinguished. The unstable equilibrium reached between the sphincter and dilator muscles creates small continuous variations in the pupil size, which are normal and called pupillary unrest or hippus. The normal function of the pupil and its innervation are extensively covered in Chapter 4, and pharmacological manipulation of the pupil is covered in Chapter 12. The reflexes controlling pupil size are important in the diagnosis of neuro-ophthalmic conditions.

The principal sensor for the pupillary light response is the photopic system (the cones); therefore, illumination of the fovea is the primary determinant of the pupillary light reflex. The retinal nerve fibers that relay information for pupillary control travel through the optic nerve to the optic chiasm. Here, the fibers undergo semidissuasion. Each of the pretectal nuclei sends crossed and uncrossed intercalated neurons through the posterior commissure to the Edinger–Westphal nucleus. The origin of the efferent (motor) fibers that control pupil size. Therefore, the photopic information given to one eye is normally transmitted to both pupils, creating direct and consensual (indirect) responses. Both of these responses, the direct pupillary light reflex and the consensual pupillary light reflex, should be evaluated when the pupillary responses are examined.

The parasympathetic innervation to the iris, starting in the Edinger–Westphal nucleus, travels with the third cranial nerve, emerging from the brain ventrally between the cerebral peduncles. The nerve then follows a course along the posterior communicating arteries to pierce the wall of the cavernous sinus. Here it is close to the first and second divisions of the fifth nerve. The nerve enters the orbit via the superior orbital fissure. The preganglionic parasympathetic fibers deviate from the third cranial nerve and synapse at the ciliary ganglion. Postganglionic fibers reach the iris sphincter muscle via the short ciliary nerves. The overwhelming majority of compared with dark irises. Pupils become mydriatic in response to large sensory, emotional, or psychological stimuli. Hyperthyroidism and ingestion of lead can produce mydriasis. Various medications, including antihistamines, over-the-counter decongestants, anticholinergics, phenothiazines, amphetamines, cocaine, and antianxiety agents, may also enlarge the pupils. The pupils become miotic in response to pain or irritation within the globe as a result of the oculopupillary reflex during keratitis, iritis, or trauma. However, pain elsewhere in the body tends to cause pupil dilation. Long-standing diabetes, sleep, and intraocular inflammation produce miosis. The pupils of infants and the elderly are small. Medications, including cholinergic agents used to treat glaucoma, chlorpromazine, cholinesterase inhibitors (found in insecticides and toxic nerve gases), and morphine derivatives, constrict the pupil. Under ordinary circumstances, the pupils of females may be larger than those of males, and the pupils of myopes larger than those of hyperopes. However, the pupil sizes of females and males are no different, and of myopes and hyperopes no different, when specialized conditions ensure that accommodative differences have been accounted for.

The pupils should appear round, roughly centered within the iris, and of equal size in the normal patient. The irises should be of the same coloration. The clinician should be aware of deviations from this norm, which could appear in the form of a unilateral or bilateral irregular pupil (a nonround pupil), ectopic pupil (significantly decentered pupil), polyopia (more than one pupil in an eye), anisocoria (pupils of different sizes in the two eyes), or heterochromia (irises of different color or lightness/darkness in the two eyes). In ocular albinism and oculocutaneous albinism, the irises of the two eyes appear to contain little or no color and scattered light enters the eye through the iris, such that the pupil is not allowed to perform its optical functions. In aniridia, the iris is absent or only partially present; therefore, the pupil does not exist or is considered to be expanded to the ciliary body.
these parasympathetic fibers innervate the ciliary muscle for control of accommodation, whereas only 3% of the fibers innervate the iris sphincter muscle.\(^1\)

The iris dilator muscle receives innervation from the sympathetic system, which begins in the posterior hypothalamus. Efferent fibers travel to the brain stem and synapse in the intermediolateral gray matter of the spinal cord (ciliospinal center of Budge) at the T\(_2\) level. Preganglionic efferent fibers or second-order neurons exit at this level into the thorax and then travel to synapse in the superior cervical ganglia, located at the level of the angle of the jaw. The postganglionic efferent fibers or third-order neurons follow the internal carotid arteries and reach the orbits by way of the superior orbital fissures, eventually joining the ophthalmic division of the fifth cranial nerve to innervate the iris dilator by way of the long ciliary nerve in each eye. At the carotid bifurcation, fibers that supply the sweat glands split from those that supply the iris dilator and muscle of Müller in the upper eyelid.\(^2\)

With near fixation, the pupils of both eyes constrict as part of triad response of accommodation, convergence, and miosis. The pupillary near reflex does not depend on retinal illumination and is, therefore, present in a blind eye.\(^2,12\) How the accommodative input reaches the third nerve nucleus is not completely understood. However, midbrain lesions affecting fibers just approaching or leaving the pretectal synapse often affect the light reflex but spare the accommodative reflex. It is believed the midbrain center for the accommodative reflex may be located slightly ventral to the center for the light reflex (pretectal nucleus).\(^13\) Hence, the near reflex is nearly always present when the direct light reflex is intact.

**Clinical Evaluation**

The pupils should be observed for their size and shape, direct and consensual responses to light, and accommodative miosis. They should be observed individually and in comparison with each other. The direct reflex is noted when a light beam is directed into one eye, and its pupil constricts. The consensual (indirect) reflex occurs when the light is shone into one eye and the pupil in the opposite eye contracts. The swinging flashlight test detects afferent defects due to anomalies in the retina or optic nerve pathway anterior to the lateral geniculate nucleus. The pupil cycle time is determined by the rapidity with which the neural and muscular components act to constrict and dilate the pupil.

**Analysis of Pupil Size**

Measurement of the physiological pupil diameters requires the patient to be adapted to the level of illumination in the immediate environment, typically under normal room illumination. After the patient history is completed, but before changes in room illumination are made, is an ideal time to make the assessment. The room should be illuminated well enough to clearly see the pupils, but light should not be beamed directly at the eyes. The patient is asked to fixate a distance target, and the diameter of each pupil is measured with a millimeter rule held against the cheekbones and covering the lower half of the patient's pupils. The zero mark is aligned with one edge of the pupil and the point that intersects the other edge of the pupil, at its largest diameter, is the pupil size. The pupil size can also be estimated by size comparisons with a series of filled circles, having a progression of diameters from small to large, as is often performed with a pupil gauge (Figure 10-1). An alternative pupil gauge has a series of filled hemispheres, progressing in size from small to large, for which one vertical or lateral pupil margin is aligned with the edge of a filled half circle (Figure 10-2). The scale is moved to the appropriate hemisphere, at which the opposing edge of a half circle is simultaneously coincident with the corresponding edge of the pupillary margin.\(^8,14\) The entoptic pupillometer, a less common subjective instrument for determining pupil size using the Scheiner disc principle, is covered in the appendix of Chapter 20.

If a millimeter rule is used, the pupil size should be recorded to the nearest 0.5 mm with the right eye recorded first (i.e., 3/3). Even if graded in 1.0-mm increments, the pupil size can be interpolated to the nearest 0.5 mm when using pupil gauges of the filled circle, hemispherical, or Scheiner principle designs. Pupillary unrest is generally of a magnitude below that of 0.5 mm and should not significantly influence the results.

**Figure 10-1**

Pupil sizes under bright and dim conditions may also be assessed using a direct ophthalmoscope. The examiner should be positioned directly in front of the patient in a darkened room, the only light present being a distance fixation target or chart. With the ophthalmoscope set to +1 D and turned up to its fullest intensity through the largest aperture, the light beam is directed onto the patient's face from a 1-m distance. This simulates the "bright" conditions binocularly. The practitioner views the red pupillary reflexes through the aperture of the scope and compares the sizes of the entrance pupils. The red reflexes enhance the ability to detect small differences in pupil sizes. The eye with the largest pupil should be noted and the size difference estimated or measured with a rule or gauge. To assess the pupils under "dim" conditions, the intensity is reduced until the red reflexes are barely visible.\(^14\) Again, differences in pupil size are noted and estimated or measured.

The findings for bright and dim conditions should be recorded separately. If no difference in the amount of anisocoria occurred between light and dim conditions, the examiner should record that the anisocoria was equal under bright and dim conditions. When anisocoria is present in equal amounts under both bright and dim conditions and is not accompanied by other clinical signs, it is known as simple, physiological, or essential anisocoria. Twenty percent of the population may manifest this discrepancy in the amount of 0.4 mm or more,\(^16\) which is of little or no adverse consequence. The anisocoria can often be observed in old photographs of the patient, especially if the red reflexes are present. Small amounts of anisocoria are often easier to detect in dim illumination.\(^13,17\) However, anisocoria that varies significantly with the amount of room illumination can be pathological in origin, and further investigation of such patients is necessary.

Pupil size, and its measurement, has become increasingly important in the outcomes of corneal and refractive lens surgery. Disturbance of vision and optical glare phenomena are produced when the pupil diameter increases, most commonly at night. A pupillometer should be used to assess the pupil size under scotopic conditions before surgery to choose the appropriate ablation zone or the phakic IOL optic size.\(^16\)

Direct and Consensual (Indirect) Light Reflexes

Upon completion of the pupil size analysis, each pupil should be observed for a direct response to light. The room illumination should be semi-darkened yet sufficient for the clinician to easily view the pupils from a distance of 30 cm or less. The patient is asked to fixate a distant target, perhaps a projected 20/400 (6/120) Snellen letter, as the examiner sits or stands slightly off to one side so as not to be in the patient's direct line of sight. The beam from a handheld light source, usually a penlight or transilluminator, is directed toward the patient's right eye for 2 to 4 seconds and is then removed. The clinician notes the change in the pupil size due to the direct light reflex, in terms of the initial constriction when the penlight beam is directed into the eye, and the dilation when the penlight beam is removed. The magnitude (quantity) of change and the rapidity (quality) of the change in pupil size should be noted. The constriction will be slightly faster than the dilation, as noted previously. The direct response of the right eye should be elicited two or three times to confirm the result. The penlight is then directed toward the patient's left eye. The quantity and quality of the left
pupil’s direct responses are noted two or three times and compared with the responses of the right eye. The direct responses of the two eyes should be the same in terms of magnitude and rapidity.

Repeating the process involved in assessment of the direct reflexes, the clinician next assesses the consensual (indirect) reflexes. When the penlight beam is directed into the right eye, the examiner observes the pupillary response of the left eye; similarly, the right eye is observed when the beam is directed into the left eye. Not only should the consensual responses of the two eyes be the same, but the consensual responses should be equal to the direct responses. Constriction will again be slightly faster than dilation. In routine cases, the examiner can simultaneously assess the direct and consensual responses in the same two or three exposures of each eye to the penlight beam.

In the presence of an afferent (sensory) defect, the direct and consensual responses will be weakened or absent when a light beam is directed into the affected eye. The severity of the defect can be graded on a clinical scale from 0 to 4, with 0 corresponding to no defect and 4 corresponding to an absence of the appropriate pupillary reflexes. In the presence of an efferent (motor) defect, the direct response of the affected eye will be weakened or absent, and the consensual response will be attenuated or absent when light is directed at the unaffected eye. Hence, significant anisocoria will exist only with lesions of the efferent system except in rare instances.

Swinging Flashlight Test
The swinging flashlight test compares the strength of the direct pupillary response with that of the consensual (indirect) response and is used to assess afferent (sensory) pupillary defects. The technique is the same as that described for the direct and indirect responses, with the exception that the light beam is alternated from one eye to the other, back and forth, while the practitioner observes the pupil sizes in the two eyes. The penlight is used to illuminate the right eye for 2 to 4 seconds before being quickly moved to illuminate the left eye for another 2 to 4 seconds and is swung back and forth between the eyes in this manner four or five times. The exposures of the eyes must be the same in terms of illumination and duration to avoid false-positive results, and movement of the light beam between the eyes is at a speed that prohibits pupil dilation during the instant that the light beam is not shining into the right or left eyes.

In the normal case, the pupils should remain equally constricted during the swinging flashlight test as the light beam is alternated between the eyes. This is because (1) the light beam is moved quickly back and forth between the eyes so as to prohibit dilation as a result of the interval during which the light beam is between the eyes, (2) the direct responses should be equal for the two eyes, (3) the consensual responses should be equal for the two eyes, and (4) the direct and consensual responses should be equal within each eye.

If an afferent pupillary defect (APD) is present, the pupils of both eyes will dilate slightly when the affected eye is illuminated. This is known as pupillary escape. The binocular dilation should be observed when the light is alternated to the affected eye, and constriction will again occur when the penlight beam is directed to the unaffected eye. The magnitude of the defect is usually correlated to the rapidity of the escape, and the severity of the defect can be graded on a typical clinical scale from 0 to 4. Neutral density filters can also be used to grade the severity of the defect. A filter is placed in front of the unaffected eye and increased in density until the pupil responses are equal. The density of the filter that produces an equal response between the eyes is taken to be the measure of the defect’s relative severity. In unilateral optic neuropathies, the magnitude of the APD correlates with the estimated percentage of retinal ganglion cell loss.

Near Response
If the direct response in each eye is brisk and the constriction is equal relative to the other eye, the near response need not be tested. As noted previously, it is rare for the near reflex to be abnormal when the direct response is intact. However, in the presence of an abnormal direct response, near testing can be of diagnostic importance and should be performed.

In testing the near reflex, the patient is asked to view a distant target in normal room illumination and then to fixate a near target held 25 to 30 cm from the eyes. The illumination of the near target should be the same or similar to the illumination of the distant target such that the light reflex is not confused with the near reflex. Several letters on a near-point card work well as a target. Normally, both pupils will constrict when focusing on the near target, but this may be difficult to observe in those patients having small pupils. The near reflex in these cases may be easier to confirm in a semidarkened room by observing the red reflex produced with an ophthalmoscope or retinoscope. Indeed, the retinoscopist can assess the stability of accommodation by noting pupil size changes due to accommodative fluctuations and knows if a patient alternates focus from distance to near during the procedure (see Chapter 18). However, care should be taken in the retinoscopic analysis of the near response, that the patient focuses on a near target illuminated only by room light, rather than fixating on the bright light of the retinoscope or ophthalmoscope. If the near response is present in the absence of a direct reflex, it is called a light-near dissociation. The near response can be present even in blind eyes.
Pupil Cycle Time
Periodic oscillations of the pupil can be observed by use of the slit lamp biomicroscope, after introduction of a focused horizontal slit of light approximately 1-mm wide on the iris at the inferior pupillary border. When the light beam is placed such that a small portion of its thickness is focused inside the pupil, the pupil should constrict such that the stationary light beam is excluded from the pupil. With the light excluded, the pupil should then dilate such that a small portion of the beam’s thickness is again allowed into the pupil. Hence, the size of the pupil cycles as constriction and dilation repeatedly occur. The pupil cycle time is averaged over 30 cycles, measured with a stopwatch, and is indicative of a pupil anomaly if greater than 954 msec/cycle (just about 1 sec per cycle) for a single pupil or if the difference between the two pupils is greater than 70 msec/cycle. Most pupillary reflex anomalies will significantly increase the pupil cycle time.

Recording
If all pupil reflexes are normal, the acronym PERRLA is recorded. PERRLA stands for “pupils equal, round, responsive to light and accommodation.” If the near reflex was not tested, the acronym can be shortened to PERRL. One could also record APD-, indicating that no afferent pupillary defect was present. Because APD defects are also called Marcus-Gunn pupils (see later), MG- is often recorded as a substitute for APD-.

If a defect in the pupillary responses was noted, the type of defect, degree of defect, and the eye should be recorded. For instance, an afferent defect in the left eye having a severity of grade 2 would be recorded: APD 2 OS. The mean pupil cycle times, if performed, should be recorded in milliseconds per cycle for each eye.

Pupil Anomalies
Pupil size and reflex anomalies may be secondary to lesions in the afferent (i.e., retina, optic nerve) or efferent (i.e., sympathetic or third cranial nerve) pathways. Many unilateral efferent anomalies of the pupillary reflexes will generate anisocoria, which should be found in the assessment of pupil size at the beginning of the pupillary examination. The more common pupillary reflex anomalies are listed in this section, below, such that the eye care clinician can be looking for them in practice. A flow chart concerning the diagnosis of pupillary anomalies is shown in Figure 10-3.

Anisocoria Greater in Bright Conditions
Adie’s Tonic Pupil. A relatively common occurrence called Adie’s tonic pupil is noted primarily in females in their third and fourth decades of life, yet it has been seen in all age groups and both genders. The incidence is approximately 4.7 per 100,000 in the population per year, and the prevalence is approximately 2 per 1000 in the population. The mean age of onset is 32 years, and the ratio of affected females to males is 2.5:1. These patients appear otherwise healthy but present with a unilateral semi-dilated pupil that responds minimally and slowly (sluggishly) to light. In 10% to 20% of the cases, the fellow eye also becomes involved. The unilateral defect is present directly when light is shone into the affected eye and consensually when light is shone into the unaffected eye. The near reflex will also be sluggish for the affected eye, and dilation is slower in the affected eye when switching fixation from near to far. The anisocoria is more pronounced in bright conditions than in dim conditions. These findings suggest the existence of a lesion in the efferent parasympathetic pathway on the side of the semi-dilated pupil, resulting in poor constriction of the corresponding iris sphincter muscle. The denervation often appears secondary to a mild viral infection that adversely impacts the postganglionic fibers at or distal to the ciliary ganglion on the affected side. The affected eye’s accommodative motor control is likely to be diminished simultaneously. Because the assumed lesion is distal to the deviation of the parasympathetic fibers from the third cranial nerve, there is no involvement of the extraocular muscles.

Although Adie’s pupil is not associated with any ocular or nervous system disease that requires treatment, other orbital and ocular conditions should be ruled out in the differential diagnosis. Proptosis or engorged conjunctival vessels, for instance, could indicate a tumor or mass behind the globe that reduces the function of the parasympathetic innervation. Patients are often concerned about the cosmetic appearance of the anisocoria or blurred near vision from the simultaneous involvement of the accommodative fibers. Accommodation often returns within 2 years as efferent fibers regenerate, but the anisocoria persists. This is due to the much larger number of fibers in the ciliary ganglion that are destined for the ciliary muscle (97%) than for the iris (3%). The few pupillary fibers become lost among the many accommodative fibers and end up becoming misdirected. Indeed, the pupil often becomes controlled by regenerated accommodative fibers. The practitioner should be concerned with a proper accommodative balance (equalization) at distance and at near during the subjective refraction (see Chapter 20) for patients having Adie’s pupil. Unequal bifocal add powers may be indicated for presbyopes, a subject further covered in Chapter 20. The refractive error of the affected eye may also become slightly more hyperopic or less myopic.

Many patients with Adie’s pupil show absent or reduced deep tendon reflexes, especially in the lower extremities. The iris sphincter muscle resulting in an Adie’s pupil can become hypersensitive to cholinergic
Figure 10-3
A flow chart for diagnosis of anisocoria.

stimulation over time. A drop of only 0.125% pilocarpine, not concentrated enough to influence the normal pupil, will cause 80% of Adie's pupils to constrict. Hence, a drop of 0.125% pilocarpine is thought to be a diagnostic test indicative of Adie's tonic pupil. In the classic literature, 2.5% methacholine hydrochloride (Mecholyl) was used in an identical fashion for the diagnosis of Adie's pupil, but pilocarpine is now generally available, whereas Mecholyl is not. Greater variability in the sensitivity of Adie's patients to methacholine also limited the usefulness of the drug in making an accurate diagnosis.27

Palsy of the Third Cranial Nerve. Anisocoria greater in bright conditions is usually associated with involvement of the extraocular muscles (EOMs) during third cranial nerve palsies. Tumors, aneurysms, bone fragments, and herniated tissue can all compress the oculomotor nerve. Because the pupillary fibers travel superficially with the third nerve, they tend to be involved early in the compressive process, resulting in a fixed and dilated pupil.13 Thus, early diagnosis of the pupillary defect can be important in the evaluation and management of an acute third-nerve palsy.17 With an ischemic lesion, such as in diabetes or arteriosclerosis, the pupil is often spared while the extraocular muscles are adversely affected.13 Progressive involvement of functions related to the third cranial nerve may result in accommodative insufficiency, ptosis on the affected side secondary to interruption of innervation to the levator, and exotropia secondary to paralysis of the rectus muscles (excluding the lateral rectus) and the inferior oblique. Hence, the clinician can gain insight relative to the disease progression by evaluation of the pupils, accommodation, eyelids, and any incomitancies indicating the particular EOMs that are paretic or paralyzed. The corneal blink reflex should be tested to assess the
function of the fifth nerve, whose first and second branches pass alongside the third nerve in the cavernous sinus and through the superior orbital fissure. Orbital auscultation should be done to listen for bruises suggestive of a posterior communicating artery aneurysm.

Anomalous eyelid or eye movements with an abnormal pupil response may occur secondary to aberrant regeneration of oculomotor fibers following a compressive lesion due to trauma, tumor, or aneurysm. The pupil will most often constrict in downgaze and adduction when aberrant EOM fibers have regenerated. Abnormal accommodation may also be a result.

**Pharmacological Pupil.** It is simple for an atropinic substance to get on the hands and be rubbed into the eye, and some patients instill eye drops intended for other persons or purposes. A fixed and dilated pupil in an otherwise healthy and unremarkable patient should alert the practitioner to the possibility of pharmacologically induced pupil dilation. Health care workers should be questioned regarding their exposure to possible causative medications, and patients should be asked about use of topical ocular medications, especially those that were originally prescribed for another family member. Gardeners and those with outdoor interests may come into contact with plants leaving atropinic-like residues on the hands. Neurologically enlarged pupils will constrict to an application of 1% pilocarpine, but pharmacologically blocked pupils will not. Hence, the clinician has available a test to differentiate the two basic causes of pupil dilation. However, this test should not be used if an acute neurological lesion is strongly suspected, because the induced miosis leaves the pupil untestable in a potentially rapidly evolving neurological condition.7

**Anisocoria Greater in Dim Conditions**

**Horner’s Syndrome.** Horner’s syndrome is the name given to the condition wherein sympathetic innervation to the eye is interrupted, resulting in a miotic pupil with incomplete dilation in darkness. Because the sympathetic system also controls Müller’s muscles of the eyelids and the facial sweat glands, slight ptosis and decrease in facial sweating (facial anhydrosis) may occur on the same side as the miotic pupil. Hence, the three hallmark signs of Horner’s syndrome are “miosis, ptosis, and anhydrosis” on the affected side. The condition can be easily missed, because the anisocoria can be small, with less than 1-mm difference between the pupils, a result of the paretic iris dilator muscle being weaker than the sphincter. The ptosis is generally mild, because Müller’s muscle is weak and controls only the tonic retraction of the upper eyelid. Faint ptosis of the lower lid may also be present, in which the lower lid rises slightly, but this is difficult or impossible to document because it is smaller in magnitude than even ptosis of the upper lid.

The efferent lesion causing a Horner syndrome can be anywhere in the long sympathetic pathway to the pupil. First-order or central lesions may be due to stroke, multiple sclerosis, cerebral spinal cord trauma, syringomyelia, or neoplastic disease of the brain stem or spinal cord. Preganglionic lesions may be located in the thoracic apex or in the neck proximal to the superior cervical ganglion, such as carcinoma of the lung apex (Pancoast’s tumor) and neck lesions including those resulting from trauma and thyroid enlargement. Breast carcinoma, lymphadenopathy, and thoracic aneurysms may also result in preganglionic Horner’s. Postganglionic lesions may be extracranial with similar etiologies listed for preganglionic neck lesions and abnormalities of the internal carotid artery. They may also be intracranial from a cavernous sinus or middle cranial fossa lesion or cluster headaches. The lack of sympathetic innervation in congenital cases of Horner’s syndrome may cause heterochromia since the growth of pigmented melanocytic cells is modulated by the sympathetic system. Hypopigmentation in the affected eye may occur if the onset of oculosympathetic paresis is before age 2 years. Congenital Horner’s syndrome is generally benign. Acquired Horner’s syndrome can also be benign, as in trauma to the head or neck, or indicate a serious problem, as in a tumor along the sympathetic pupillary pathway or aneurysm of the carotid or subclavian arteries. Trauma is the leading cause of Horner’s in patients under 20 years. In patients aged 21 to 50, however, tumors are the cause in almost half the cases. Neoplasms are an important etiology in the over 50-year age group as well.

As a general rule, the postganglionic lesions are benign and the preganglionic lesions are indicative of a serious problem. Hence, it is important to be able to differentiate between the two. Postganglionic lesions do not generally cause facial anhydrosis, because the sympathetic fibers supplying the sweat glands split from those innervating the iris and Müller’s muscle at the carotid bifurcation. A history of endarterectomy, head trauma, or thyroidectomy suggests a postganglionic problem. Cluster headaches are also associated with postganglionic lesions. Auscultation of the neck and testing of corneal and facial sensitivity should take place, because the sympathetic path closely follows the carotid artery and first division of the 5th cranial (trigeminal) nerve.

Some experts advocate the topical use of 4% to 10% cocaine in the diagnosis of Horner’s syndrome. Cocaine prevents the reuptake of norepinephrine at the sympathetic neuromuscular junctions. Thus, the normal pupil will dilate in response to cocaine because its sympathetic innervation is capable of maintaining endogenous levels of norepinephrine at the neuromuscular junctions. Blockage of reuptake increases the concentration of norepinephrine at the neuromuscular junc-
Amaurotic Pupil. An amaurotic pupil occurs in an eye with no light perception. The direct reflex will be absent in the affected eye but its pupil contracts because of consensual reflex when the unaffected eye is illuminated. The unaffected eye will demonstrate a direct reflex but no consensual response when the affected eye is illuminated. Both eyes will constrict to a near target. Anisocoria will not be present under normal circumstances when the two eyes are equally illuminated. The amaurotic pupil is essentially a severe afferent pupillary defect.

Light-near Dissociation

Pupils that fail to constrict to light but demonstrate a near reflex are said to have a light-near dissociation. Previously, this was frequently seen in neurosyphilis, where it was associated with the bilateral Argyll Robertson pupil, discussed separately later. A true light-near dissociation can be seen in afferent pupillary defects and amaurotic pupils, which adversely influence the light reflex but leave the efferent pathways intact. Certain lesions of the midbrain including Parinaud's syndrome affect the initial motor control of the light reflex but allow slightly more distal input for the near response through otherwise intact efferent paths. The Argyll Robertson pupil is likely a type of a midbrain lesion resulting in a light-near dissociation. It is important to note that the near response in a light-near dissociation is present bilaterally, even if the pupillary defect in the light response is unilateral or bilateral.

It is difficult to understand how a light-near dissociation, having a bilateral near reflex, could appear in cases of efferent pupillary defects. One would expect the near response to be unilateral: diminished or absent on the affected side. Sometimes, however, a false light-near dissociation will present on the affected side when the "near response" is accomplished through aberrant regenerated nerve fibers to the iris musculature. This often occurs in lesions of the third nerve, when fibers originally destined for the medial rectus aberrantly innervate the iris sphincter, and in lesions of the ciliary ganglion (Adie's pupil), when fibers originally destined for the ciliary muscle aberrantly innervate the iris sphincter. Light-near dissociations have been reported with several types of motor neuropathies and are most likely related to aberrant regenerations.

Midbrain Lesions

If both pupils show little or no response to light, bilateral Adie's pupils should be considered. In these instances, the patient's vision would likely be decreased at near and the pupil cycle time large. A myopathy or neuropathy in the midbrain that affects the pretectal synapses can also present this bilateral condition, but visual acuity will be unaffected. Usually the near reflex is present because the light reflex fibers in the tegmen-
The eyelids protect the eyes and distribute the tear film to moisten the ocular surface and maintain the cornea's optical surface quality. Abnormal innervation of the musculature of the eyelids can cause visual impairment by an inability to provide excellent surface optics over the pupillary area, inability to keep all of the interpalpebral space adequately wet or moistened, inability to retract the upper eyelids such that all or part of the pupil is uncovered, or inability to blink at the appropriate times so as not to interfere with vision. Many of the aspects of an evaluation of the tear film and eyelids are concerned with the physiology of the ocular surface and logically fall into the external examination or biomicroscopy (see Chapter 13) or into the province of contact lens practice. Here, abnormal aperture size and eyelid movements resulting from neuromuscular eyelid disorders will be discussed, such that the examiner can recognize and manage these neuromuscular problems.

The position and movement of the eyelids are controlled by three separate neuromuscular systems actuating the levator palpebrae superioris, the muscle of Müller, and the orbicularis oculi. The levator palpebrae superioris is a muscle in the superior orbit that extends into the upper eyelid. The levator's tendon inserts into a large area of the skin of the upper eyelid, and some of its fibers insert into the anterior surface of the tarsal plate, as shown in Figure 10-4. The levator is the primary muscle responsible for retraction of the upper lid following the blink and in upgaze. The fold near the top of the upper lid marks the superior boundary of the insertion of the levator into the skin covering the eyelid. This fold is not present in oriental eyelids, because the levator's tendon does not insert as completely into the overlying skin.

The facial sheath of the levator is common to that of the superior rectus muscle (see Figure 10-4), and its neural input is coordinated with that of the superior rectus, such that upgaze produces simultaneous elevation of the upper lid. The frontalis muscles of the brow help the levators to elevate the upper lids in extreme upgaze. When a person forcibly closes the eyelids, as in blepharospasm, the eyes rotate bilaterally upwards. "Bell's phenomenon" is present in 90% of persons and believed to be a protective mechanism that brings the cornea underneath the upper lid and away from potential sources of injury. Bell's phenomenon can be observed by holding the lids open while the subject attempts to forcibly close the eyes.

The levator is supplied by the superior branch of the third cranial (oculomotor) nerve, which also innervates the iris sphincter, ciliary muscle, and four of the six EOMs on the same side. Both oculomotor nerves are supplied by fibers destined for the levator from a single nucleus located on the dorsal aspect of the oculomotor nuclear group in the mesencephalon. Hence, the two levator muscles, one in each upper eyelid, are activated together to achieve simultaneous retractions after the blink and in upgaze.

The muscles of Müller receive innervation from branches of the sympathetic nervous system, whose course was described earlier in this chapter for the pupillary dilator muscle and facial sweat glands. There are actually four Müller's muscles: one smooth muscle for each eyelid. The muscle of Müller in a superior eyelid is anchored into the inferior facial surface of the levator and inserts into the upper edge of the superior tarsal plate. Contraction of these muscle fibers retracts the superior tarsus and, therefore, moves the superior eyelid upward. In an inferior eyelid, the muscle of Müller is anchored into the upper facial surface of the inferior rectus and inserts into two places: the lower edge of the inferior tarsal plate and the conjunctival fornix. Contraction of these muscle fibers retracts the inferior eyelid downward. Being of smooth muscle and of relatively...
consistent activity, the muscles of Müller supply a tonus to the open-eye retractions of the upper and lower eyelids.

The orbicularis oculi is the primary muscle that closes the eyelids. There are two major divisions of the orbicularis: the palpebral portion and the orbital portion. The palpebral portion of the orbicularis covers the tarsal plates, yet lies below the outer surfaces of the upper and lower eyelids, and extends from the eyelid margins to the orbital rim. The physiology of the palpebral orbicularis is suited for rapid movement and acceleration. It is responsible for involuntary eyelid closure during the blink and voluntary eyelid closure as in winking. A small specialized portion of the palpebral orbicularis is called the muscle of Riolan, which is located in the margins of the eyelids and thought to help keep the margins in apposition with the ocular surface. During forced eyelid closure, as occurs in winking or blepharospasm, the orbital portion of the orbicularis and eyebrow muscles come progressively into action, depending on the force of closure involved. Another small specialized portion of the orbicularis ensnares the nasal lacrimal sac. Upon eye closure, as in blinking, this sprig of the orbicularis squeezes the sac, emptying its contents into the nasal passage. Upon eye opening, this unnamed portion of the orbicularis releases, allowing the lacrimal sac to take in tear fluid via the canalicular/punctal drainage system.

The orbicularis is innervated by fibers from the seventh cranial (facial) nerve, which originates in the pons. The nerve enters the internal auditory canal and then passes through the facial canal in the petrous portion of the temporal bone, emerging through the stylomastoid foramen, which is inferior to and slightly posterior to the external auditory opening. The two facial nerves also innervate the muscles of the corresponding sides of the face. The routes and branches of the facial nerves are highly variable among persons, such that certain people can “wiggle” one or both ears, or raise one or both eyebrows individually, whereas others cannot. Hence, some patients can voluntarily wink or close each eye by itself (monocularly), others can wink or close only one of the eyes by itself, yet a few are unable to voluntarily close or wink either eye by itself. In all cases, however, the normal patient should be able to voluntarily blink or close both eyes at the same time.

The palpebral aperture, or fissure, is 27 to 30 mm in length (horizontally) and 8- to 11-mm wide (vertically) at its widest point in adults, which is usually nasal of center by 1 to 4 mm, creating an “almond” shape. In Asians, the aperture may not be quite as wide, although
the characteristic shape is retained. In children the aperture is not as long and is relatively wider, compared with its length, whereas in infants the aperture can be nearly circular. The normal width of the palpebral aperture is the result of a competitive equilibrium of muscle tonus between the orbicularis, acting to lessen the fissure, and the combined tonus of the levator and muscles of Müller, acting to widen the fissure. The adult palpebral aperture can be made approximately 15 mm wide by voluntary lid retraction of the levator and maximally 17 or 18 mm wide by simultaneous action of the frontalis muscle of the eyebrow. \(^{36}\)

The upper eyelid normally covers the superior cornea from approximately the 10 o'clock to the 2 o'clock positions. It covers a mean of 2.1 mm of the superior cornea in Caucasians (±0.9 mm), perhaps more in Asians representing 7% of the corneal surface area. \(^{41}\) In most persons the lower eyelid margin will reside at or below the lower limbus by 1 or 2 mm. In a few persons, the upper eyelid margin will normally reside at the superior limbus or 1 to 2 mm above, the inferior eyelid margin will cover a small portion (1-2 mm) of the inferior cornea, or both. It is important that the clinician assess the geometry of the patient’s palpebral apertures in order to recognize the abnormal from the normal, so that underlying neuromuscular deficits can be detected.

**Clinical Evaluation**

In routine examinations, the width of the palpebral aperture is not actually measured, although it is assessed qualitatively by the clinician. The aperture sizes of the two eyes are compared with each other and the anatomical locations of the upper and lower lid margins are noted relative to the corneal limbus of each eye. Should the widths of the palpebral apertures require documentation, the patient is asked to fixate a distant target under normal room illumination, and a millimeter rule is positioned vertically at the aperture’s widest extent. The distance between the upper and lower lid margins is measured for the right and left eyes. Similarly, the positions of the eyelid margins with respect to the upper and lower extents of the corneal limbus can be measured.

Perfect symmetry of the right and left palpebral apertures exists only for a few patients. Typically, one eye will have a slightly wider aperture than the other, and the eyelid margins will intersect the upper and lower limbus at positions that are slightly different for the two eyes. A difference of 2 or more millimeters between the widths of the palpebral apertures is suggestive of unilateral ptosis, which results from inferior positioning of one upper lid relative to the other, superior positioning of one lower lid relative to the other, or both. However, ptosis can also be bilateral, in which both upper lids or both lower lids are insufficiently retracted. The eyelids may also appear to be overly retracted or widened, as is common in thyroid disease.

When one or both palpebral apertures appear malformed, or a difference in lid positions is noted between the eyes, it is important to question the patient about the asymmetry without suggesting or implying initially that the appearance of the eyes is abnormal. The onset, progression over time, and variation of the asymmetry with certain actions (e.g., upgaze, blinking, eye closure) are of particular interest. Ptosis can often be observed in old photographs of the patient. The patient may be instructed to follow, with the eyes, the clinician’s finger into upgaze and downgaze while the clinician observes the intersection of the eyelid margins with the corneas. In this manner the clinician may assess whether the asymmetry becomes greater or lesser in upgaze or downgaze compared with primary gaze. The clinician notes the completeness of the blink in each eye and of voluntary eyelid closure. Blinks and voluntary closure should be assessed bilaterally because, as noted earlier, it is not possible for some persons to voluntarily wink or close an eye monocularly.

**Recording**

There is no standardized system for the recording of palpebral aperture widths, eyelid margin locations, or palpebral abnormalities. Indeed, these are usually recorded only if the practitioner notices an abnormality of the palpebral aperture. Documentation of the palpebral aperture width is merely a recording of the widths for the right and left eyes. The positions of the eyelid margins can be recorded relative to the corneal limbus, with positive numbers indicating coverage of the cornea and zero indicating the margin at the limbus. For instance, assume that the palpebral aperture widths are 12 mm in the right eye and 9 mm in the left eye; the upper lid margins are overlapping onto the cornea by 1 mm in the right eye and 3 mm in the left eye; and the lower lid margins are 1 mm below the lower limbus in the right eye and at the lower limbus in the left eye. A recording could be: R 12/+1/-1 mm, L 9/+3/0 mm. The clinician might also note if, for instance, ptosis exists in the left eye, proptosis in the right eye, or that the eyes are normally asymmetric to this degree, whichever is determined.

**Neuromuscular Palpebral Anomalies**

**Ptosis**

The most common neuromuscular abnormality of the palpebral aperture is ptosis, which generally manifests as an abnormal location of the superior eyelid. Ptosis of the superior lids can be documented by the extent of upper lid overhang onto the cornea in both eyes, as noted earlier, and graded using a simple 0 to 4 clinical scale of severity. Gravity usually works in favor of ptosis.
of the upper eyelids. In the extreme, the low position of an upper lid can occlude all (grade 4) or part of (grade 3) the pupil, and the lid may not be retractable. This could be caused by a lesion of the levator's innervation or interruption of its function, as commonly occurs with a marked upper eyelid inflammation. The ptotic upper eyelid may not occlude any of the pupil (grade 2), and mild ptosis (grade 1) can be difficult to discern from normal asymmetry. This could be the result of a lesion that partially blocks the sympathetic innervation to the superior muscles of Müller or a small upper eyelid inflammation.

A very common cause of ptosis is an eyelid inflammation of microbial, allergenic, or traumatic nature. An inverse ptosis, also called an upside-down ptosis, is an elevation of the lower lid as a result of lower lid inflammation or interruption of innervation to the inferior muscles of Müller. Inverse ptosis is usually mild and is generally difficult to recognize, because gravity works in favor of retraction of the inferior eyelid. The positions of the lower lids can be documented relative to the lower corneal limbus as noted earlier. A mild (grade 1 or 2) to moderate (grade 3) bilateral ptosis often occurs in the aged as a result of disinsertion of the levator or reduction of retrobulbar orbital fat. The enophthalmos may bring about increased coverage of the globe by the superior and inferior eyelids. Most cases of ptosis will be more evident when the patient is sleepy or fatigued, and this can help in the diagnosis of the milder forms (grades 1 and 2). Unilateral or bilateral ptosis should be evaluated with consideration given to the pupillary examination, the function of the EOMs, and other clinical neurological signs.

**Dysfunction of the Levator Palpebrae Superioris.** Dysfunction of the levator can be caused by a lesion of the oculomotor nerve or by restriction of the levator's function. Head trauma, tumors, aneurysms, and thrombosis of the cavernous sinus can result in lesions of the oculomotor nerve, which are often accompanied by involvement of the EOMs supplied by the oculomotor nerve (exotropia) and an ipsilateral dilated pupil with accommodative involvement (efferent pupillary defect). Ptosis of recent onset is usually caused by an oculomotor nerve lesion and is accompanied by diplopia if the lid does not completely occlude the pupil. Mechanical restriction can result from excess pressure on the lid from tumors or inflammation, or scar tissue can interfere with lid retraction. Trauma of the eyelid may break some or many of the tendinous fibers inserting into the eyelid, as might occur resulting in postoperative ptosis (usually unilateral). The insertion of the levator may become less effective with aging, resulting in senile ptosis (usually bilateral). Myogenic defects are caused by impaired function of the muscle or the myoneural junction as occurs in congenital ptosis, myotonic dystrophy, and myasthenia gravis.

Ptosis involving the motor route to the levator will generally be unilateral. The eyelid does not retract completely after a blink or in upgaze. Hence, the ptosis will appear to be of greater magnitude in upgaze. The motor nuclei of the superior divisions of the 3rd cranial nerves are located dorsally in the midbrain, and damage there results in bilateral ptosis. An unaffected pupil with ptosis may occur in diabetic neuropathy, accompanied by a history of diabetes, and myasthenia gravis, accompanied by complaints of unusual fatigue. The ptosis can be increased with fatigue in myasthenia gravis, and fatigue of the levator may be elicited by having the patient hold the eyes in upgaze for several minutes. The superior muscle of Müller cannot compensate for a paretic levator because the smooth muscle is anchored on the underside of the levator, which is not able to supply much support in its paretic state.

**Dysfunction of Müller’s Muscle.** Horner's syndrome was described earlier, in which the sympathetic pathway to the ipsilateral pupil, muscles of Müller, and facial sweat glands was interrupted. Ptosis in this syndrome is primarily the result of the paresis or paralysis of the superior muscle of Müller. Much lid retraction after the blink and in upgaze is intact, because the levator is unaffected. The ptosis does not increase in upgaze. Ptosis in Horner's syndrome is generally not as pronounced as that occurring with paralysis of the levator. In addition, the EOMs are unaffected, the ipsilateral pupil is miotic, and anhydrosis may be present on the affected side of the face.

**Eyelid Retraction**

When the eye is in primary gaze, the visibility of sclera between the limbus and upper lid margin may indicate the presence of an eyelid retraction. It is common for 1 to 2 mm of sclera to show between the lower eyelid margin and the limbus. As will be noted later, a slight eyelid retraction may be apparent with a 7th cranial nerve palsy. However, eyelid retraction is most commonly due to thyroid disease or midbrain lesions. It can also be the result of surgical overcorrection of ptosis, scarring from eyelid trauma, or tumors. Aberrant regeneration of nerve fibers to the levator could be one cause of the Marcus–Gunn phenomenon (jaw-winking), for which the upper eyelid unilaterally retracts when the mouth is opened, and the pseudo-Graefe phenomenon, for which the upper lid retracts upon downgaze. Globe displacement or enlargement (as seen in axial myopia or congenital glaucoma), the use of topical sympathomimetics, or high doses of systemic steroids can produce eyelid retraction. In addition to identifying the etiology of retraction, the clinician should monitor for a resulting exposure keratitis, which may require treatment with topical lubricants or surgical correction.

**Thyroid Disease.** Thyroid disease is the most common cause of lid retraction and may be present in
hyper- or hypothyroidism. Euthyroid, wherein the ocular signs are apparent in the presence of normal thyroid function tests, is also a frequent cause. Lid lag in downgaze (Graefe’s sign), a staring appearance (Dalrymple’s sign), and infrequent and incomplete blinking (Stellwag’s sign) often occur with retraction in Grave’s disease. Although ocular effects from thyroid dysfunction are generally observed bilaterally, it is not uncommon for the signs to present asymmetrically and appear as a unilateral lid retraction. In hyperthyroidism the lids often return to normal after medical treatment of the thyroid condition, but usually the lid retraction in euthyroid persists if it has been present for a year or more.44 Ocular lubricants are often necessary for the treatment of the resulting dry eye, but any surgical correction of the lids should wait until the thyroid condition is stable.

**Midbrain Disease.** Lesions of the posterior third ventricle (Parinaud’s ophthalmoplegia) may manifest Collier’s sign, a staring appearance caused by bilateral lid retraction. Conjugate, upward movement of the eyes is restricted, and a convergence-retraction nystagmus is elicited on attempted upgaze. Abnormal pupils (light-near dissociation) are also present in Parinaud’s ophthalmoplegia. Etiologies range from hydrocephalus and pinealoma in infants and teens to arteriovenous malformations, tumors, and basilar artery disease in adults.

**Dysfunction of the Orbicularis Oculi.** Paresis of the orbicularis may cause ineffective or incomplete lid closure, whereas outright paralysis results in no blink whatsoever. Served by the 7th cranial (facial) nerve, some or all of the facial muscles of the cheek and mouth likely will be affected by paresis or paralysis, causing the patient to lose ipsilateral facial expression. The simultaneous influence on the muscle of Riolan leaves eyelid apposition to the globe chronically affected by gravity. The upper lid may remain loosely apposed to the ocular surface and perhaps slightly retracted because of the loss of orbicularis tone and the unopposed tonus of the levator and superior muscle of Müller. The lower lid may manifest ectropion. In the aged, a loss of tonus in the orbicularis is common and may contribute to incomplete blinking, lagophthalmos, and marked ectropion of the lower lid with epiphora.

A 7th cranial (facial) nerve palsy is usually unilateral, because the lesion occurs in the peripheral nerve instead of encompassing both nuclei in the pons. Bell’s phenomenon, noted earlier in this chapter, will be intact in the preponderance of these cases but will be absent if the lesion damages a 7th nerve nucleus. A common paresis of the 7th cranial nerve is Bell’s palsy, which is actually of unknown etiology, but inflammation around the 7th nerve inside the facial canal and trauma at the opening of the stylomastoid foramen are two suspected causes.40

**Idiosyncratic Eyelid Motions**

When assessing asymmetrical aperture fissures, it is important to consider that an apparent unilateral ptosis may actually be a contralateral proptosis, lid retraction, or slack lower lid caused by a weak orbicularis muscle. Ptosis can also induce a pseudo-lid retraction in the fellow eye as the patient uses the brows to raise the upper eyelid on the affected side. Because there are equal innervations to the upper eyelids from a single nucleus, a forcible attempt to raise the affected upper lid may cause the other upper lid to rise excessively. Raised eyebrows or furrowing of the forehead indicate this maneuver.

### MONOCULAR AND BINOCULAR EYE MOVEMENTS

The purpose of eye movements, actually rotations of the eye, is to initiate and maintain foveal fixation. Vertical gaze and lateral (horizontal) gaze direct the lines of sight in object space along the Y and X axes, respectively, such that combinations of vertical and lateral conjugate eye movements (or rotations) result in direction of the eyes toward targets within any of the four quadrants. Eye movements direct the lines of sight up, down, right, or left away from the primary gaze position (straight ahead). Conjugate eye movements (versions) are those in which both eyes rotate simultaneously in the same direction by equal amounts. Vergence (disconjugate) eye movements, covered in the next section of this chapter, rotate the eyes in opposite directions so as to align the eyes along the anteroposterior Z axis. Hence, the eyes can be directed toward objects located in three-dimensional space in front of the eyes by a combination of conjugate and vergence eye movements. All reflexive and voluntary eye movements are hierarchically controlled by a cortical network that involves the frontal, parietal, and occipital areas that send diverse premotor signals to the nuclei of the third, fourth, and sixth cranial nerves. Generally, reflexive eye movements originate in the posterior parts of the brain and voluntary movements from frontal areas. Structures involved in horizontal gaze generation occupy the lower pons and upper medulla, and those structures important for vertical gaze reside in the rostral midbrain.

Torsion (torsional eye movement) twists the eyes clockwise or counterclockwise as viewed by the clinician from the front: encyclorotation (intorsion) is the term applied when the top of the eye rotates toward the nose, and encyclorotation (extorsion) is the term applied when the top of the eye rotates away from the nose. Conjugate torsion twists the eyes in the same direction, clockwise or counterclockwise in both eyes, when the head is tilted to the right or left. Vergence or disconju-
Gate torsions occur in opposing directions, intorsion or extorsion in both eyes. Both types of torsional movements are necessary to maintain alignment of the meridians of the two eyes for single binocular vision.

Conjugate eye movements can be tested to determine if the neuromuscular systems controlling the movements are intact and functioning properly. The signal for ocular movements originates in the cerebral hemispheres and is transmitted to the gaze centers in the midbrain and motor nuclei in the pons. From there, the information travels through the 3rd (oculomotor), 4th (trochlear), and 6th (abducens) cranial nerves to supply the EOMs. Supranuclear neuronal pathways conduct impulses to the gaze centers, and internuclear pathways coordinate the gaze centers with the motor nuclei. The infranuclear pathways lie in the individual cranial nerves.

Abnormal conjugate eye movements can be used to help discern whether a lesion involves one or more of the three cranial nerves on each side (the infranuclear paths) or is located at the motor nuclei in the midbrain and pons, the gaze centers in the upper midbrain, or the cerebral centers where the eye movements are initiated. The internuclear or supranuclear pathways may also become dysfunctional. As in many other areas of the general eye examination, the degree of complexity becomes ever greater as more specificity is required and as more specialty topics are covered. Ocular motility can become a specialty by itself when taken to the "nth degree," wherein the border between the roles of a neurologist and an eye care practitioner is indistinct. However, we will limit this section of our chapter to a screening for conjugate eye movement defects in the initial phases of the routine eye examination.

### Monocular Eye Movements

Each eye is suspended within the bony orbit by six EOMs and a complicated system of connective tissue extending from the orbital apex, posteriorly, to the orbital rim, anteriorly. The connective tissue consists of ligaments, septa, and sheaths of the EOMs. The rectus muscles and their intermuscular septa form a “muscle cone,” in which the space is filled with the optic nerve, ophthalmic artery, blood vessels to the EOMs, nerves to the EOMs, and the ciliary ganglion. The remainder of the space is filled with orbital fat. The connective tissues, fat, and the extracocular muscles actually form a larger structure that surrounds the globe, dampening movement of the eye and acting as a “fluid brake” for smooth, quick completion of eye rotations. The widest part of the orbit is located 15 mm behind the orbital rim, corresponding roughly to the position of the widest diameter of the muscle cone situated within (Figure 10-5). Hence, the orbital space has been described as being in the shape of a pear.57

The EOMs are arranged in three planes of action, each containing a cooperative pair of muscles that act together to control rotations of the globe within the respective planes. With the exception of the superior oblique (see later), the planes contain the midpoints of the origins and scleral insertions of the respective pair

---

![Diagrammatic sagittal cross section through the center of the globe and orbit. Note the pear-like shape of the bony orbit, the fascial connection between the levator and superior rectus, and the fascial connection of the inferior rectus and inferior oblique.](image-url)
of muscles and the longitudinal axes of the muscle fibers. The lateral and medial recti are located in the horizontal plane. When the line of sight of the eye is in the horizontal plane, the actions of the lateral and medial rectus muscles are to direct the line of sight to the left or right within the horizontal plane. The superior and inferior recti are located in a vertical plane that intersects the line of sight in primary gaze (straight ahead) at an angle of 23 to 25 degrees (Figure 10-6). When the line of sight of the eye is 23 to 25 degrees temporal to that of the primary gaze position, the actions of the superior and inferior rectus muscles are to direct the line of sight up or down, respectively, within the plane of the muscles. The superior and inferior oblique muscles act in a vertical plane that intersects the primary line of sight at an angle of 51 to 53 degrees (Figure 10-7). When the line of sight of the eye is 51 to 53 degrees nasal to that of the primary gaze position, the actions of the superior and inferior oblique muscles are to direct the line of sight down or up, respectively, within the plane of action.

An important concept in ocular motility is that a paretic or paralyzed EOM will always have its greatest adverse effect when the line of sight is directed into the muscle's primary action within its plane of action. Hence, the rotation of an eye will lag farthest behind that wanted or required to fixate a target when the line of sight of the eye is made to lie in the paretic muscle's plane of action, and the patient is asked to then direct the eye into the muscle's primary action. For instance, a paretic lateral rectus in the right eye will be the most obvious when the eye is directed along the horizontal to the patient's right. A paretic superior rectus in the left eye will be most obvious when the gaze is shifted 23 to 25 degrees to the patient's left and then in upgaze. When the line of sight is outside of a muscle's plane of action, the actions of the EOM become more complicated, as will be explained.

Rectus Muscles
The lateral, medial, superior, and inferior rectus muscles are anchored at the apex of the orbit in a thickened annular portion of the periosteum called the circle of Zinn and insert into the sclera anterior to the equator of the globe and posterior to the limbus. The insertion of the medial rectus is 5.5 mm from the limbus. The inferior rectus inserts 6.5 mm from the limbus, the lateral rectus 6.9 mm from the limbus, and the superior

---

Figure 10-6
Diagram of the top of the eye, from above, showing the origin, insertion, and longitudinal axis of the superior rectus muscle, which lie in the same vertical plane as those of the inferior rectus muscle (not shown).
rectus 7.7 mm from the limbus (Figure 10-8). An imaginary spiral formed around the corneal limbus by connecting the insertions of the medial, inferior, lateral, and superior rectus muscles is called the spiral of Tilleaux. A lateral check ligament is connected anteriorly to the muscle sheath of the lateral rectus and is anchored to the zygomatic bone. A medial check ligament is connected similarly to the medial rectus and is anchored to the nasal bone. The lateral and medial check ligaments limit the nasal and temporal rotations of the eye, respectively, in extreme positions of horizontal gaze. Along with the insertion of the four rectus muscles and the superior oblique at the orbital apex, the check ligaments prohibit the globe from moving forward outside of the orbit. These ligaments have no effect on normal rotations of the eye except for the limitation in extreme lateral gaze. Simultaneous contraction of all of the rectus muscles can result in retraction of the globe and apparent enophthalmos.

The lateral rectus muscle lies in the horizontal plane and is aligned with the middle of the globe as viewed from the temporal side. The lateral rectus is innervated by the 6th cranial (abducens) nerve; contraction of the lateral rectus results in temporal rotation of the globe (abduction). The medial rectus also lies in the horizontal plane and aligns with the middle of the globe. The medial rectus is innervated by the inferior division of the 3rd cranial (oculomotor) nerve; contraction of the medial rectus results in nasal rotation of the globe (adduction). The innervations of the two muscles are coordinated, such that one is inhibited while the other is active, thus directing component rotations of the eye in the horizontal plane. It is important to note that, under ordinary circumstances in primary gaze, actions of the medial and lateral rectus muscles do not result in torsion of the globe or in vertical eye rotation. However, when the eye is directed upward, contractions of the medial and lateral recti help slightly to elevate the eye; in downgaze, these rectus muscles help to slightly depress the eyes. This is because, as noted earlier, the insertions of the EOMs are anterior to the equator of the globe. The torsional movements of the eyes caused by the lateral and medial recti in upgaze and downgaze appear to be subclinical.

The superior rectus muscle is in a vertical plane having an angle of approximately 23 to 25 degrees with the line of sight in primary gaze (see Figure 10-6). Its anchorage is medial to the center of rotation of the eye,
and its insertion into the sclera is anterior to the center of rotation, superior to the corneal limbus. The muscle sheath of the superior rectus is continuous with that of the levator palpebrae superioris, as noted previously, and it is similarly innervated by the superior division of the 3rd cranial (oculomotor) nerve. Upon contraction in primary gaze, the major function of the superior rectus is to rotate the globe upward (elevation). However, because of its insertion anterior to the equator, contraction of the superior rectus in primary gaze results secondarily in a small nasal rotation of the eye (adduction) and slight encyclotorsion (intorsion). The action of the superior rectus varies significantly, depending on the horizontal rotation of the eye. When the line of sight is directed 23 to 25 degrees temporal to that of the primary gaze position, the superior rectus produces only ocular elevation. When the line of sight is 65 to 67 degrees nasal to that of primary gaze, the superior rectus produces only intorsion and adduction.

The innervations of the superior and inferior rectus muscles are coordinated, such that one is inhibited while the other is active, thus directing component rotations of the eye in the vertical plane. Adduction produced by the combined actions of the superior and inferior recti is countered by the lateral rectus, and torsions are countered by action of the superior or inferior oblique muscles (see the next section).

Oblique Muscles

The distal portion of the superior oblique muscle and the entire inferior oblique muscle are located in a vertical plane that intersects the primary line of sight by an angle of 51 to 53 degrees (see Figure 10-7). The superior oblique muscle is anchored in the lesser wing of the sphenoid bone at the orbital apex above the circle of Zinn. It runs outside the rectus muscle cone, superonasally, to the trochlear fossa in the frontal bone near the superonasal orbital rim. At this point, its route is redirected by slippage through a cartilaginous "pulley," or trochlea, into the plane of action. The superior oblique then runs back under the muscle cone and inserts into the sclera of the globe behind the insertion of the superior rectus and posterior to the equator (see Figure 10-7). Unlike a rectus muscle, the superior oblique pulls its insertion forward instead of backward.
The superior oblique is innervated by the 4th cranial (trochlear) nerve; contraction of the superior oblique in primary gaze acts primarily to encyclorotate (intort) the globe. However, because of the insertion posterior to the equator, contraction of the superior oblique in primary gaze results secondarily in a slight depression and temporal rotation of the eye. When the line of sight is directed 51 to 53 degrees nasal to that of the primary gaze position, the superior oblique produces only ocular depression. When the line of sight is 37 to 39 degrees temporal to that of primary gaze, the superior oblique produces only intorsion and slight abduction.

The inferior oblique muscle also pulls the globe forward instead of backward in the same plane of action as that of the superior oblique. Although the other EOMs originate at the orbital apex, the inferior oblique is anchored in a shallow depression at the front of the anteronasal floor of the orbit near the lacrimal fossa. The inferior oblique runs back within the rectus muscle cone and above the inferior rectus, where the muscle sheaths of the two muscles become attached. The inferior oblique inserts into the sclera behind the insertion of the inferior rectus and posterior to the equator, an area that is close to the macula, ciliary vessels, and ciliary nerves. The inferior oblique is a part of the "suspensory ligament of Lockwood," which consists of (1) the inferior oblique and its muscle sheath, (2) the anterior portion of the inferior rectus and its muscle sheath, (2) intermuscular septa connecting the anterior muscle sheaths of the lateral and medial recti to that of the inferior rectus, and (3) the lateral and medial check ligaments (see Figure 10-5). It is believed that Lockwood's ligament helps support the globe from underneath so as to maintain its vertical position within the orbit.

The inferior oblique is innervated by the inferior division of the 3rd cranial (oculomotor) nerve; its contraction in primary gaze acts primarily to excyclorotate (extort) the globe. However, because of the insertion posterior to the equator, contraction of the inferior oblique in primary gaze results secondarily in a small elevation and temporal rotation (abduction) of the eye. When the line of sight is directed 51 to 53 degrees nasal to that of the primary gaze position, the inferior oblique produces only ocular elevation. When the line of sight is 37 to 39 degrees temporal to that of primary gaze, the inferior oblique produces only extorsion and slight abduction.

The innervations of the superior and inferior oblique muscles are coordinated, such that one is inhibited while the other is active, thus establishing the torsional position of the eye. Vertical rotations or abduction produced by the combined actions of the superior and inferior oblique muscles are countered by action of the other two EOM pairs. The tendinous fibers that insert the oblique muscles into the sclera are spread out in a fan shape (see Figure 10-7), unlike the relatively straight insertion fibers of the rectus fibers (see Figure 10-6). Hence, the medial fibers in the fan are shortened by adduction and the temporal fibers are elongated; the opposite occurs during abduction. This tends to allow the contractile force of the oblique muscles to remain concentrated in the same plane of action for various horizontal positions of gaze. As a result, the primary action of an oblique muscle is to intort (superior oblique) and extort (inferior oblique) the globe through most of the lateral excursion of the line of sight. This leaves the superior and inferior rectus muscles as the primary muscles controlling vertical eye rotations throughout most of the lateral range of eye excursion. The secondary actions of the oblique muscles, noted earlier, are less powerful in primary gaze than are the secondary actions of the superior and inferior rectus muscles.

Cranial Nerves III, IV, and VI
It is important to review the neuroanatomy of the motor controls for the EOMs, because lesions of the nerves or at the central origins of the nerves will have consequences directly linked to the resulting lack of innervation.

Emerging ventrally from the midbrain (mesencephalon) between the cerebral peduncles, near the midline, the two 3rd cranial (oculomotor) nerves pass between the ipsilateral superior cerebellar and posterior cerebral arteries. Each 3rd nerve then follows a course forward and downward along the ipsilateral posterior communicating artery, and pierces the wall of the cavernous sinus on that side. Here, the 3rd nerve is close to the 4th and 6th cranial nerves, as well as the ophthalmic division of the 5th cranial (trigeminal) nerve. The 3rd cranial nerve divides and enters the orbit as the superior oblique, and lateral rectus. The preganglionic parasympathetic fibers exit the inferior branch of the 3rd nerve and synapse within the ciliary ganglion, which is normally attached to the outer surface of the inferior branch. As was noted earlier, postganglionic fibers from the ciliary ganglion innervate the ipsilateral pupillary sphincter and the ciliary muscle. The superior branch of the 3rd cranial (oculomotor) nerve is of smaller caliber than the inferior branch, because it serves only the ipsilateral superior rectus and levator palpebrae superioris. The inferior branch, of larger caliber, serves all of the remaining ocular muscles except the iris dilator, superior oblique, and lateral rectus.

Fibers in the 3rd cranial nerve are supplied by the oculomotor complex, which is located near the central gray matter of the midbrain at the level of the superior colliculi. The oculomotor complex consists of several coordinated nuclei and motor cell column pairs (the dorsal cell columns, intermediate cell columns, ventral...
cell columns, and dorsal median cell columns). Most of the fibers in the 3rd cranial nerve are uncrossed, but some are crossed. The dorsal cell column supplies uncrossed fibers destined for the inferior rectus. Similarly, the intermediate cell column and ventral cell column supply uncrossed fibers for the inferior oblique and the medial rectus, respectively. The dorsal median column provides crossed fibers to the superior rectus. The paired Edinger-Westphal nuclei and anterior median nuclei supply uncrossed preganglionic parasympathetic fibers for the ciliary ganglia. As a result, the columns on the right side of the midbrain send fibers destined for the right inferior rectus, right inferior oblique, right medial rectus, left superior rectus, right pupillary sphincter, and right ciliary muscle. The single caudal central nucleus gives rise to fibers destined for the two levator palpebrae superioris muscles that are equally crossed and uncrossed. The existence of a central nucleus of Perlia, which has been said to control convergence and divergence, has been postulated but has been difficult to substantiate. Smaller accessory nuclei exist, which are thought to be involved in torsional eye movements and reflex movements of the head and neck.

The pair of trochlear nuclei lie in the midbrain (mesencephalon) at the level of the inferior colliculi, in the peri-aqueductal gray matter, caudal (below) and adjacent to the oculomotor complex. Each trochlear nucleus supplies originally uncrossed fibers to its respective 4th cranial (trochlear) nerve. However, the two slender nerves emerge behind the midbrain (dorsally), in a downward direction, and decussate completely behind the brain stem in what is called the superior medullary velum. Each 4th cranial nerve then curves around the brain stem to attain a ventral direction, then inward and directly forward, to pass between the superior cerebellar and posterior cerebral arteries. Here, the 4th nerve is significantly inferior and lateral to the 3rd cranial nerve as the nerves follow the posterior communicating artery. Their vertical separation reduces as the 3rd nerve drops to nearly meet the 4th nerve prior to entering the cavernous sinus. The 4th nerve slips above the 3rd nerve in the cavernous sinus and escapes the circle of Zinn to innervate the superior oblique muscle.

The 4th cranial nerves have the longest intracranial course of any of the cranial nerves (75 mm) and are the only completely crossed cranial nerves. They are also the only nerves to emerge dorsally from the central nervous system and are the thinnest of the cranial nerves. As a result, the somewhat fragile 4th nerve supplies innervation to the superior oblique muscle on the contralateral side of its nucleus and is more likely to be injured as it runs most of its long course on the side ipsilateral to the superior oblique.

The pair of abducens nuclei lie in the very dorsal (back) portion of the pons next to the floor of the 4th ventricle, well below (caudal to) the trochlear nuclei and oculomotor complex. Each abducens nucleus is partially encircled by the root of a 7th cranial (facial) nerve as the complicated root loops behind and around the nucleus. The abducens nucleus supplies uncrossed fibers to the root of its respective 6th cranial (abducens) nerve. The 6th nerve root emanates ventrally from the nucleus and travels across nearly the entire width of the pons before emerging ventrally in the fissure between the pons and medulla, immediately next to the midline, as a slender 6th cranial nerve. The thin nerve runs a long course steeply up and over the petrous tip of the temporal bone, to which it is bound, then a less inclined route up to the cavernous sinus where it is adjacent to the other cranial nerves destined for the orbit. The 6th cranial nerve enters the orbit via the superior orbital fissure with the other ocular cranial nerves, and goes through the circle of Zinn to innervate the lateral rectus muscle. Because of its fragility and long course through the cranium, over the apex of the temporal bone, the 6th cranial nerve is vulnerable to injury and increased intracranial pressure.

**Binocular (Conjugate) Eye Movements**

The actions of the EOMs are coordinated between the two eyes, with bifoveal fixation as the goal. This is achieved by gaze centers in the midbrain and pons, which are responsible for the appropriate excitatory and inhibitory innervations to the individual ocular muscles in order to achieve the amount and direction of conjugate eye movement required. The vertical gaze center is a single nucleus in the posterior commissure of the midbrain above the level of the superior colliculi, which disseminates input to the nuclei of the oculomotor complex and the trochlear nuclei, such that the proper signals are sent along the cranial nerves to both eyes. The horizontal gaze center is also known as the paramedian pontine reticular formation (PPRF). The PPRF is a pair of sites in the lower pons, ventral to the nuclei of the 6th cranial nerves, which are connected to each other and the motor nuclei of both eyes by the medial longitudinal fasciculus (MLF). Therefore, a lesion in the upper midbrain may reduce the ability to rotate the eyes vertically, whereas a defect in the lower pons may reduce the ability to rotate the eyes horizontally.

**Hering's Law**

Under normal binocular circumstances, the direction, speed, and magnitude of rotation will be equal between the two eyes during conjugate movements. The EOMs of the two eyes are yoked together, with identical excitatory or inhibitory innervation supplied to corresponding yoked muscles (Table 10-2). This is the basis of Hering's law, which concludes that equal and simultaneous innervation is sent to the corresponding EOMs of the two eyes for all voluntary conjugate eye movements.
Hering's law applies whether the eyes are fixating binocularly or monocularly. For instance, the covered left eye will normally follow the right eye when the right eye fixates in different positions of gaze. However, Hering's law does not imply that the corresponding muscles actually receive the innervation that is intended, or that the muscles will equally react to the innervation that reaches them. This is because syndromes or lesions of the neural routes may reduce the actual innervations that arrive at the EOMs, or muscular defects and local physical abnormalities may reduce the ability of the muscles to carry out their functions. In these cases, the paretic muscle will induce two phenomena that can be recognized by the clinician when the line of sight is in or near the paretic muscle's plane of action. First, when fixating with the nonparetic eye, the line of sight of the eye with the paretic muscle will lag behind that of the nonparetic eye when the patient is asked to fixate into the direction of action of the paretic muscle. This is called the primary deviation, or undershooting. Second, when fixating with the paretic eye, the nonparetic eye will overshoot into the paretic muscle's direction of action. This is called the secondary deviation, or overshooting. Overshooting is more pronounced and noticeable to the clinician than is undershooting. Hence, it is often easier to recognize that an EOM paresis exists and to identify the paretic muscles using the secondary deviation in comparison with using the primary deviation.

### Types of Conjugate Eye Movements

There are three primary types of conjugate eye movements: saccades, pursuits, and vestibular eye movements. Saccades and pursuits are each generated in different cerebral areas and may be mediated through different supranuclear pathways. Vestibular movements are reflex actions initiated by the ear canal and mediated by the cerebellum and brain stem. However, they use the same gaze centers, motor nuclei, and motor nerves, which together constitute the "final common pathway" to the EOMs. All of the movements result from the coordinated action of the 12 EOMs (six EOMs per eye).

Saccades are rapid, voluntary or reflex fixational conjugate eye movements stimulated by alternation of the object of regard in the X, Y object plane. They can be elicited by asking the patient to look around the examination room at different distant targets. The fixations and refixations depend on the integrity of the fovea and cooperation of the patient. The cerebral origin of saccades appears to be in the two areas 8 of the frontal lobes (the frontal eye fields) and the posterior parietal cortex. Supranuclear fibers course from these areas to the midbrain (superior colliculus) and cross to the other side. The saccadic gaze center is most likely in the PPRF in the lower pons, which receives the supranuclear inputs from the frontal eye fields (FEF) and the superior colliculus. The gaze center is, in turn, responsible for the appropriate excitatory and inhibitory influences given to the motor nuclei of the 3rd, 4th, and 6th cranial nerves, such that a saccade is made to the proper approximate X, Y position in object space. Undershoots and overshoots are then corrected by subsequent additional saccadic movements.

Each FEF directs saccadic eye movements to the contralateral side. Thus, stimulation of the right FEF results in conjugate eye movements to the left side. The contralateral eye movements can be strictly lateral, or they may be also up or down to various degrees, depending on the location of the stimulus within area 8. Strictly vertical saccades are elicited by simultaneous and equal stimulation of both sides of the FEF. Saccadic dysfunction can be a result of cortical disease in the frontal lobes.

Pursuits are slow, smooth tracking conjugate eye movements stimulated by target motion, which maintain fixation at the foveas. They can be elicited by asking the patient to follow a slow moving target. In the absence of target motion, patients who attempt to move the eyes smoothly will produce a series of small saccades. Should a target be moving too fast or slow for a pursuit to keep the line of sight on target, a saccadic movement is made to regain fixation before the system again pursues the target.

The cerebral origin of pursuits appears to be in the striate visual cortex at the parieto-temporo-occipital junction. From the visual cortex the signal is relayed to the FEF, which projects to horizontal gaze center (PPRF) in the lower pons by supranuclear fibers that cross in the midbrain. The visual cortex directs pursuit movements to the ipsilateral side. Stimulation of the right area striate visual cortex results in conjugate eye movements to the right side. Vertical pursuit movements and component movements are elicited by simultaneous stimulation of both sides.

During self-motion or motion in the environment, retinal images are stabilized by a reflex system consisting of the vestibulo-ocular and the optokinetic reflexes. The optokinetic response is simulated by...
retinal image slippage and adapts eye velocity to the velocity of the retinal image.\textsuperscript{3} It complements the vestibulo-ocular reflex to generate compensating gaze-stabilizing eye movements.

Vestibular eye movements are reflex, smooth, pursuit-like conjugate eye rotations that counteract head movements during locomotion. They are initiated by angular acceleration of the head sensed by the three semicircular canals or by head tilt sensed by the utriculus and sacculus. The former are often associated with the term vestibular nystagmus and the latter with the term doll's eye movements. These sensory organs are located adjacently in the vestibular apparatus of the inner ear.

Lateral eye movement is driven by the ampulla of a horizontal semicircular canal, whose fibers connect to vestibular nuclei in the pons and are relayed to the contralateral PPRF. Stimulation of a horizontal ampulla results in a pursuit-like conjugate eye movement to the contralateral side, within the plane of the canal (horizontally). Similarly, stimulation of the ampulla of an anterior or posterior vertical semicircular canal results in a pursuit-like eye movement in the respective plane of the stimulated canal. Hence, pursuit-like eye movements in the plane of the angular acceleration will occur because of the component eye movements produced by the three pairs of semicircular canals. These movements compose the "slow phase" of vestibular nystagmus. The "fast phase" of vestibular nystagmus is a corrective saccadic movement driven by the frontal eye fields. Dysfunction of the semicircular canals or of their afferent fibers can result in abnormal vestibular nystagmus that occurs without angular acceleration of the head.\textsuperscript{52}

Doll's eye movements, also called counter-rolling, are reflex pursuit-like compensatory eye rotations that help to maintain fixation when the head is tilted forward or backward or turned to the left or right. The eyes rotate up reflexly, the upper lids are raised as the head is tilted forward, and the eyes rotate down as the head is tilted back. Torsional eye movements are made in response to head tilt left or right, and are the basis for the Bielschowsky head tilt test, to be covered later in this chapter.

These eye movements are the result of the oculocephalic reflex. The utriculus and sacculus in each inner ear contain hair cells that sense the weight of small crystals of calcium carbonate (otoliths). The hair cells in the utriculus are situated parallel to the horizontal plane and the hair cells in the sacculus are parallel to a vertical plane. Hence, the tilt of the head forward or backward and to the left or right is coded and sent to the vestibular nuclei in the pons. The codes for head position are applied to vertical and torsional eye movements. Similar horizontal compensatory eye movements, which help maintain fixation during head rotation to the right or left (not head tilt), are doll's eye movements originating at the semicircular canals. Doll's eye movements become more evident when the patient's other conjugate eye movements have been incapacitated at the cerebral or supranuclear levels—for instance, after a stroke. Being reflexly driven at a lower level, counter-rolling is produced when the patient's head is tilted or turned by the examiner or another person.

Optokinetic nystagmus (OKN) is a phenomenon that is unrelated to vestibular nystagmus, except that the same final common pathways are likely. It probably results from intercortical connections between the frontal and occipital eye fields,\textsuperscript{53} activates the same network as saccades and pursuits,\textsuperscript{45} and is generated in response to sustained rotations. It is often elicited clinically by use of a vertically striped drum that is rotated before the patient's eyes. A particular stripe is fixated and followed by conjugate pursuit as the stripe travels in one direction across the field of vision. Once the stripe becomes no longer visible, a saccade is made in the opposite direction, such that another moving stripe is fixated and followed. The process repeats itself as long as the patient directs his or her attention to the moving stripes, resulting in an alternation of slow, smooth pursuit movements consistent with the direction and speed of the drum rotation and fast saccadic movements in the opposite direction. OKN is the basis for the well-known "railroad car nystagmus."

OKN is a strong involuntary reflex in the horizontal plane but is relatively weak vertically. The reflex is involuntary and can be induced in all persons with a normal visual system, if sufficient visual acuity is present to recognize the stripes. It can be used to document the function of both the saccadic and pursuit systems. OKN cannot be suppressed for long periods. As a result, malingerers and hysterical patients, who are expressing visual acuity much lower than the capability of their visual systems, can often be identified by use of OKN.\textsuperscript{53}

**Clinical Evaluation**

During the initial phases of the eye examination, the clinician observes the patient's ability to fixate and change fixation from one target to another (saccades). The patient is asked to maintain fixation while following a moving target into different gaze positions (pursuits). While the patient is fixating a target, the head can be tilted forward or back and to the right or left by the examiner and maintenance of fixation observed (doll's eye movements). If fixation is poor, the clinician should rule out poor vision, poor attention, and poor motivation as causes, before concluding that an abnormality is present. The clinician should be alert for abnormal fixation, saccades, or pursuits: nystagmus, head movements substituting for eye movements, deviations (differences) between the rotations of the two eyes, under-
shooting, and overshooting. Abnormal "cogwheel" eye movements appear as jerky, erratic pursuits with frequent refixation attempts.\textsuperscript{54}

In the routine case, much of the necessary observation for saccades and vestibular eye movements can be performed during the case history, as the patient looks over the examination room, or in concert with an examination of the external ocular structures. Although the broad H test, noted immediately following, is used to screen the pursuit system, it is rare when a lesion involves the supranuclear connections or the occipital eye fields. Hence, in nearly all examinations, the broad H test allows the clinician to assess specifically the final common pathway to the EOMs. If a neuromuscular abnormality is identified, the red lens test or the Parks three-step procedure may be used to inspect and perhaps identify the EOMs that are not operating appropriately. With this information, the clinician may diagnose the probable or potential neuromuscular deficiency and apply this knowledge to the management of the patient.

Versions and Ductions: Six Cardinal or Secondary Positions of Gaze

When the neuromuscular systems controlling eye movements are operating properly, each eye is able to fixate a distant target in all positions of gaze. The angle of deviation that exists between the lines of sight of the two eyes is zero in all gaze directions for those patients achieving binocularity or is constant in all gaze directions for patients with a nonparetic strabismus. These deviations are called comitant (also known as concomitant). When one or more EOMs are paretic or paralyzed, however, the angle of deviation between the eyes varies in different positions of gaze. These deviations are called incomitant. According to the anatomy of the EOMs described earlier, it is apparent that the lines of sight will be at their greatest misalignments from that required for bifoveal fixation when the patient is gazing in one or more of the following six positions, called the six cardinal or secondary positions of gaze (Figure 10-9):

1. To the patient's immediate left, in the direction and plane of primary action of the medial rectus of the right eye and the lateral rectus of the left eye. These EOMs are primarily responsible for rotation of the respective eye directly to the left.
2. To the patient's immediate right, in the direction and plane of primary action of the medial rectus of the left eye and the lateral rectus of the right eye. These EOMs are primarily responsible for rotation of the respective eye directly to the right.
3. To the patient's left and then up, in the general direction and approximate plane of action of the inferior oblique of the right eye and the superior rectus of the left eye. These EOMs are primarily responsible for rotation of the respective eye up when gazing to the left.
4. To the patient's right and then up, in the general direction and approximate plane of action of the inferior oblique of the left eye and the superior rectus of the right eye. These EOMs are primarily responsible for rotation of the respective eye up when gazing to the right.
5. To the patient's left and then down, in the general direction and approximate plane of action of the superior oblique of the right eye and the inferior rectus of the left eye. These EOMs are primarily responsible for rotation of the respective eye down when gazing to the left.
6. To the patient's right and then down, in the general direction and approximate plane of action of the superior oblique of the left eye and the inferior rectus of the right eye. These EOMs are primarily responsible for rotation of the respective eye down when gazing to the right.

The broad H test is used to perform screenings of pursuit eye movements and the final common pathways in the six cardinal positions of gaze. The "H" refers to the path that the lines of sight follow in the X, Y plane.

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure10-9.png}
\caption{Diagram of the six cardinal positions of gaze and the extraocular muscles of each eye that are involved primarily with maintenance of gaze in these positions. R, Right; L, left; SR, superior rectus; IR, inferior rectus; MR, medial rectus; LR, lateral rectus; SO, superior oblique; IO, inferior oblique.}
\end{figure}
in object space, because the patient is asked to fixate a small target in the examiner's hand that traces out an imaginary "H" pattern in front of the patient. The two positions at which the horizontal bar intersect the legs of the "H" are meant to correspond to cardinal gaze positions 1 and 2, noted earlier. The upper ends of the two legs are indicative of cardinal positions 3 and 4, whereas the lower ends indicate positions 5 and 6. By directing the two eyes to these six gaze positions, the clinician may observe deviations in eye movements, manifested as underactions or overactions, caused by neuromuscular EOM deficiencies.

The patient should remove his or her spectacles so the examiner can easily view the eyes and their alignment. The examiner stands or sits facing the patient at a distance of approximately 1 m or less in a fully illuminated room. The patient is instructed to fixate a small target, often a penlight or the examiner's finger, held approximately 40 cm in front of the midpoint between the patient's eyes. For children, a small finger puppet or other colorful target may be used. The patient is asked to follow the movement of the target with the eyes, without moving the head, as an "H" pattern is traced out in front of the patient (Figure 10-10). The ends of the legs of the "H" correspond to extreme gaze positions at the four corners of the possible field of fixation. Lid retraction may be necessary in downgaze to allow the examiner to note the extent of eye rotation. It is normal to see a slight nystagmus, called end-point nystagmus, when the eyes are in an extreme gaze position. However, end-point nystagmus may be accentuated by paresis of one or more of the EOMs.

Versions (binocular pursuits) are assessed first, because both eyes are observed at the same time for similarity in movements. If a penlight is used as the fixation target, the corneal reflections can be used to note changes in position of the two eyes relative to each other. If any overactions or underactions occur in either eye, the testing is repeated monocularly for each eye. These are ductions, and are conducted in the same manner as versions with the exception that the contralateral eye is occluded while the ipsilateral eye undergoes the broad H test.

In the routine eye examination, versions are screened first, because it is usually easier to identify a relative discrepancy between the sighting of the eyes than it is to observe the same discrepancy monocularly. The patient may report diplopia when gazing into the direction of action of the paretic muscle, yet single binocular vision may result when gazing in the opposite direction. If the patient can follow the target around the H pattern in the routine manner, with both eyes fixating the target, the broad H test result is negative. If an incomitant strabismus is encountered such that either or both eyes are unable to properly follow the target into one or more of the cardinal positions of gaze, the test result is positive, and the clinician will need to follow-up this finding with additional testing.

It is possible that the patient may appear to have comitant strabismus that is manifested in all of the six cardinal positions. Incomitancies are more apparent immediately after a lesion occurs, in the acute stage, because the visual system compensates for them over time. The incomitancies that are paretic in origin become minimized and are often difficult or impossible to diagnose in the chronic stages. Inspection of comitancy is somewhat inexact with the H test because any incomitancy would be necessarily large if it could be viewed by the practitioner in this manner. When a strabismus appears comitant, the clinician should perform a more exacting procedure to rule out lesser incomitancies, such as the red lens test or the cover test (in nine positions of gaze), noted later.

It is often difficult to tell an overaction of one eye from an underaction of the other. Having suspected or found an EOM deficiency, then, one observes ductions of each eye critically to identify which eye has the paretic EOMs. A paretic EOM will cause the eye to lag behind or undershoot when the patient is asked to gaze monocularly into the direction of action of the EOM. However, the eye rotation may appear normal when the patient is asked to gaze in the opposite direction. Ductions are usually not tested when the versions are unremarkable. The results of version and duction testing in cases of abnormal EOM function will be consistent with those discussed later under the neuromuscular EOM anomalies.

**Figure 10-10**
Testing of versions with the broad "H" pattern. Here, the patient is looking at the top of one of the legs of the "H" to his upper left.

**Inspection of Incomitancy**
The red lens test is a *subjective* determination of the binocular ocular deviations in nine positions of gaze,
and it is performed using the red accessory lens from the trial lens set. The nine positions include the six cardinal positions, the primary gaze position (straight ahead), upgaze, and downgaze. The test is used to identify and categorize incomitancies in cases of strabismus. The red lens is placed in front of the patient's right eye initially, and a penlight is directed toward the midpoint between the patient's eyes from a distance of 40 cm to 1 m. The strabismic patient should be able to see two lights: one is white as viewed by the left eye, and the other is red as viewed by the right eye. Diplopia is induced by reduced clues to fusion in the presence of a binocular system that is unable to fuse because of strabismus or that is on the edge of binocularity because of EOM paresis. The test is of little use for patients who are fully binocular such that the diplopia does not occur. Likewise, the test is inadequate for patients having abnormal retinal correspondence or who strongly suppress one eye as a result of longstanding strabismus. An alternative for these latter patients is to neutralize the deviations with an alternating cover test using loose prisms (a procedure described later) in the nine positions of gaze, which can serve as an objective measurement of the deviations for any strabismic patient.

The penlight is moved into the nine positions of gaze, and the patient is required to fixate the light without moving the head. At each position, the patient is asked whether one or two lights are seen. If two lights are seen, the clinician questions the patient about the relative position of the lights to each other and their degree of separation. The relative positions and degree of separation of the two images are recorded at each of the nine positions, as shown in Figure 10-11. The most peripheral image of the two is that viewed by the undershooting or lagging eye.

Should the red and white images appear aligned side by side, a horizontal deviation is present. Similarly, a vertical deviation is present when the red and white images appear to be aligned vertically. Most of the time, however, the total deviation will be the result of addition of its horizontal and vertical components. If the deviation is comitant, the separation between the red and white images will be equal in all positions of gaze. If the deviation is incomitant, the separation between the lights will be greatest in the field of gaze of the affected muscle or muscles. The separation will be the least or zero in the field of gaze opposite to that of the affected muscle or muscles. The separation will increase as the gaze position changes over the field of fixation from minimum to maximum.

The patient will generally fixate with the eye that is not covered by the red lens, because the lens produces a darker visual field. The condition of the affected eye can be substantiated by repeating the test after switching the red lens to the left eye. Because overactions are generally larger than underactions, greater deviations

![Figure 10-11](image)

Results of the red lens test for a classic paresis of the right superior rectus: (above) with the red lens over the right eye so that the left eye is fixating (primary deviation), and (below) with the red lens over the left eye so that the right eye is fixating (secondary deviation). The filled red circles represent the perceived position of the red spot relative to the unfilled white circles representing the perceived location of the white spot. This could occur, for instance, with an isolated lesion of the superior branch of the 3rd cranial nerve. The symbol at the lower left of each diagram is intended to indicate bifoveal fixation or superimposition of the two images.
should be noted when fixating with the paretic eye (see Figure 10-11). The results of red lens tests in cases of abnormal EOM function will be consistent with those discussed later under neuromuscular EOM anomalies. The Hess–Lancaster screen is a similar, more elaborate method for the analysis of incomitancies, which requires special instrumentation and is reserved for specialty practice.

The Parks three-step procedure is a specific series of three tests designed to isolate the paretic muscle in cases of vertical deviations (Figure 10-12). The three-step procedure was described by Hagedoorn, popularized by Parks, and included as its third step the Bielschowsky head tilt test. When a vertical strabismus is identified or suspected, the clinician may observe the hypertropia or, more accurately, measure objectively the hypertropia by use of the alternating cover test and loose prisms in the following three steps (Table 10-3):

1. The clinician determines whether the hypertropia is present in the right eye or the left eye in primary gaze, which limits the determination of the paretic muscle to a possible four EOMs. In right hypertropia (left hypotropia), the paretic muscle could be the right inferior rectus, right superior oblique, left superior rectus, or left inferior oblique. In left hypertropia (right hypotropia), the paretic muscle could be the left inferior rectus, left superior oblique, right superior rectus, or right inferior oblique.

2. The clinician determines whether the hypertropia increases in gaze directly to the patient's right or left. Right gaze is produced by a head turn to the left as the patient maintains fixation on a distant object straight ahead. Similarly, left gaze is produced by a head turn to the right as the patient fixates the same object. This step eliminates two of the four possible muscles remaining from step 1. If right hypertropia increases in right gaze (left head turn), the potentially paretic EOMs remaining are the right inferior rectus and the left inferior oblique; if the increase is in left gaze (right head turn), the remaining EOMs are the right superior oblique and the left superior rectus. If left hypertropia increases in left gaze (right head turn), the potentially paretic EOMs remaining are the left inferior rectus and the right inferior oblique; if the

---

**Figure 10-12**

Flow chart for the Parks three-step procedure. R, Right; L, left; SR, superior rectus; IR, inferior rectus; MR, medial rectus; LR, lateral rectus; SO, superior oblique; IO, inferior oblique.
TABLE 10-3 Diagnosis of a Paretic Muscle for a Vertical Deviation

<table>
<thead>
<tr>
<th>Vertical Deviation</th>
<th>Head Turn</th>
<th>Head Tilt</th>
<th>Paretic Muscle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right hyper</td>
<td>to Right</td>
<td>to Right</td>
<td>LIO</td>
</tr>
<tr>
<td></td>
<td>to Right</td>
<td>to Left</td>
<td>RIR</td>
</tr>
<tr>
<td></td>
<td>to Left</td>
<td>to Right</td>
<td>RSO</td>
</tr>
<tr>
<td></td>
<td>to Left</td>
<td>to Left</td>
<td>LSR</td>
</tr>
<tr>
<td>Left hyper</td>
<td>to Right</td>
<td>to Right</td>
<td>RSR</td>
</tr>
<tr>
<td></td>
<td>to Right</td>
<td>to Left</td>
<td>LSO</td>
</tr>
<tr>
<td></td>
<td>to Left</td>
<td>to Right</td>
<td>LIR</td>
</tr>
<tr>
<td></td>
<td>to Left</td>
<td>to Left</td>
<td>RIO</td>
</tr>
</tbody>
</table>

increase is in the right gaze (left head turn), the remaining EOMs are the left superior oblique and the right superior rectus.

3. The clinician determines whether the hypertropia is greater when the patient's head is tilted to the right or to the left. This step eliminates one of the two possible muscles remaining from step 2 and thus isolates the paretic muscle. The head tilts induce reflex conjugate torsions as a result of the vestibular apparatus, which attempt to keep the horizontal meridians of the eyes aligned with the horizon. Head tilt to the patient's right shoulder creates a clockwise rotation of both eyes as viewed by the clinician (intorsion of the right eye and extorsion of the left eye), and head tilt to the left shoulder creates a counterclockwise rotation of both eyes (extorsion of the right eye and intorsion of the left eye). Intorsion is the primary action of the superior oblique and extorsion of the inferior oblique. Vertical eye rotations are secondary for these muscles. Therefore, the inducement of torsion by head tilt also creates input for vertical eye movement, which is countered by the superior and inferior rectus muscles.

When the head is tilted toward the side of a paretic superior oblique, the compensatory upward action of the superior rectus on that side is unopposed, and the vertical deviation becomes greater than if the head is tilted away from the affected eye. Had the paretic muscle been the contralateral superior rectus, the head tilt toward that affected side would create intorsion of that eye's superior oblique, the secondary downward input unopposed by the paretic superior rectus. Hence, the head tilt produces greater hypertropia when it is toward the paretic superior oblique of one eye or the paretic superior rectus of the other eye, and the effect of either is isolated.

Similarly, head tilt away from the side of a paretic inferior oblique results in unopposed downward action of the inferior rectus on that side, and the vertical deviation becomes greater than if the head were tilted toward the affected eye. Had the paretic muscle been the contralateral inferior rectus, the head tilt away from that affected side would create extorsion of that eye's inferior oblique, the secondary upward input unopposed by the paretic inferior rectus. Hence, the head tilt produces greater hypertropia when it is away from the paretic inferior oblique of one eye or the paretic inferior rectus of the other eye, and the effect of either is isolated.

The three-step procedure assumes that only a single paretic muscle is present and so is of particular use in the diagnosis of palsies of the 4th cranial (trochlear) nerve or the superior division of the 3rd cranial (oculomotor) nerve. It is of little help when the entire 3rd cranial nerve is affected or in the diagnosis of 6th cranial (abducens) nerve palsies because paresis of the lateral rectus produces a horizontal instead of vertical deviation.

Neuromuscular Extraocular Muscle Anomalies

Symptoms of oculomotor problems include decreased ability to fixate an object, eye fatigue, and headaches associated with the need for critical vision or near work. Paresis of the oblique muscles is often accompanied by head tilts to the left or right, and paresis of the vertical rectus muscles by chin elevation or depression. Paretic horizontal rectus muscles are often similarly accompanied by head turns to the left or right. These compensatory head postures allow the gaze position of minimum deviation to occupy the straight-ahead position. Many patients are able to achieve single binocular vision by tilting or turning the head toward the primary actions of the involved extraocular muscles, which permits the eyes to be rotated away from the primary actions. An acute episode of strabismus may result in complaints of diplopia, especially in the gaze position where an affected muscle has its greatest action, which may recede as the visual system compensates by suppressing the vision of the deviating eye.

Sixth Cranial (Abducens) Nerve Palsy and the Lateral Rectus

The long external course of the slender 6th nerve through the cranium, particularly over the apex of the temporal bone, makes it especially susceptible to injury and increased intracranial pressure. Lesions of the nerve, its root, and its nucleus will cause ipsilateral paresis of the lateral rectus, convergent strabismus increasing in temporal gaze, and lateral diplopia (Figure 10-13). Nuclear lesions will most likely be accompanied by ipsilateral paresis or paralysis of the facial muscles, includ-
ing the orbicularis, due to simultaneous involvement of the root of the 7th cranial (facial) nerve, which encircles the 6th nerve nucleus. Sixth nerve palsies are also associated with involvement of the 5th and 8th cranial nerves. Unlike the other rectus muscles, which are supplied by two anterior ciliary arteries originating from muscular branches of the ophthalmic artery, the lateral rectus is supplied by only a single anterior ciliary artery (see Figure 10-8). As a result, it is possible that the lateral rectus is more frequently and adversely affected by ischemia than are the other EOMs.

In Duane’s retraction syndrome, the globe of the affected eye is retracted and the palpebral fissure narrowed when adduction is attempted. The affected eye is also unable to abduct in most cases. The traditional explanation has been that the lateral rectus is fibrotic, such that it cannot contract or stretch properly. Hence, the lateral rectus is unable to abduct the eye properly and is not elastic enough to allow much adduction by the medial rectus. An alternative explanation is a miswiring of the innervation to the lateral rectus, resulting in cocontraction of the lateral and medial rectus muscles. It has been shown that the abducens nucleus may be hypoplastic in cases of Duane’s syndrome, possibly because of disuse, and that innervation of the lateral rectus is supplied by 3rd nerve fibers similar to those innervating the medial rectus. Despite the inability to abduct the eye, patients with Duane’s syndrome do not appear esotropic in the primary gaze position. Approximately 90% of the cases are unilateral and 10% bilateral.

Fourth Cranial (Trochlear) Nerve Palsy and the Superior Oblique

The thinness and long course of the 4th nerve, crossing in back of the brain stem and partially encircling the midbrain, makes it also especially susceptible to injury. Lesions of the nerve distal to its decussation will cause an ipsilateral paresis of the superior oblique and hypertropia with vertical diplopia increasing in inferonasal gaze (Figure 10-14). There will be significant torsional diplopia existing in other gaze positions, especially temporally. The Parks three-step procedure will reveal increased ipsilateral hypertropia with lateral gaze in the contralateral direction and with head tilt toward the side.
of the lesion. Nuclear lesions will be contralateral, as will lesions of the nerve root prior to decussation, whereas a lesion at the site of decussation can result in bilateral oblique paresis.

Palsy of the 4th cranial nerve often occurs simultaneously with paresis of the 3rd cranial nerve because of their close proximity along the posterior communicating arteries, within the cavernous sinus, and through the superior orbital fissure. The presence or absence of a 4th nerve palsy in conjunction with a palsy of the 3rd nerve is a special diagnostic problem, because the eye cannot be directed into the abducted gaze position from which vertical eye movements can be assessed. If the clinician can have the patient direct the eye to the temporal side (with 3rd nerve palsies, the affected eye is usually exotropic) and attempt to move the eye up and down, torsion of the eye as a result of a functioning superior oblique can be positively identified. This can be most easily observed by watching a landmark, such as a conjunctival or limbal blood vessel.

Third Cranial (Oculomotor) Nerve Palsy and the Other Extraocular Muscles

The parasympathetic effects of 3rd nerve paresis on the pupillary sphincter muscle and the ciliary muscle were described earlier in this chapter, as was the impact of 3rd nerve paresis on the levator palpebrae superioris. The 3rd nerve is considerably thicker than the 4th and 6th cranial nerves, and though significant lesions can interfere with the function of the entire nerve, compressive and traumatic lesions of lesser impact may disrupt only a proportion of fibers in the nerve—those on the side of the nerve affected by the lesion. Function of the pupil, for instance, may be spared in some lesions and not in others. In addition, the 3rd nerve splits into two branches before the superior orbital fissure. Hence, diagnosis of 3rd nerve lesions can be more difficult as one attempts to ascertain the location of the lesion along the 3rd nerve.

A total block of the 3rd nerve before it divides will, of course, lead to ipsilateral paralysis of the medial rectus, inferior rectus, inferior oblique, superior rectus, levator, pupillary sphincter, and ciliary muscle. Third nerve lesions can leave the eye mydriatic, with dysfunctional accommodation (cycloplegia), ptosis, and divergent strabismus. The patient is unable to move the eye down, up, or in. Incomplete paralysis (palsy or paresis) of the 3rd nerve can result in combinations of these signs and symptoms, as can lesions distal to the branching of the nerve. The ipsilateral superior rectus and levator, for instance, may be paretic because of a lesion of the superior branch (see Figure 10-11) of the 3rd nerve, yet the inferior oblique, inferior rectus, medial rectus, and the motor root to the ciliary ganglion can be compromised by a lesion to the inferior branch. Lesions in the oculomotor complex of the 3rd nerve can adversely influence some motor nuclei and leave others intact, resulting in paresis or paralysis of the ipsilateral medial rectus, inferior rectus, inferior oblique, and the contralateral superior rectus, of variable collective involvement and severity.

Internuclear, Gaze Center, Supranuclear, and Cortex Lesions

Having studied the control of the ocular musculature presented earlier in this chapter, one must realize that lesions in various areas of the cortex and supranuclear connections can bring about specific conjugate dysfunctions in saccades, pursuits, or vestibular eye movements. Lower down, in the midbrain, interference with the gaze centers and internuclear connections can result in inability to produce conjugate eye movements of any particular type, because they each require the same or similar gaze centers to coordinate the movements of all of the EOMs for both eyes. It is only at the level of the motor nuclei to or the individual pathways of the cranial nerves that the ill effects of nervous damage is present in only one eye or the other, with adverse function of a specific EOM or small set of EOMs.

**MONOCULAR AND BINOCULAR EYE ALIGNMENT**

Vergences are binocular eye movements that are not conjugate. Indeed, they are often called disconjugate eye movements, because the lines of sight are rotated toward or away from each other—not in the same direction as occurs for conjugate eye movements. The function of lateral (horizontal) vergences is to maintain bifoveal fixation of targets at various distances, and their control was extensively discussed in Chapter 5. Therefore, lateral phorias and vergences are evaluated during fixation of a distant target and a near target during the typical eye examination. There are vertical vergences, in which one eye rotates up or down in the direction opposite to that of the other eye, and torsional vergences, in which an eye cyclorotates relative to the other eye in order to achieve corresponding meridians. All three vergence motions are necessary for attainment and maintenance of bifoveal fixation. Because vertical and torsional vergences do not normally depend on target distance, they are usually tested only at a single distance, whichever is easier to accomplish or gives the most information with the particular method being used.

The signal for vergences begins in area 19 of the occipital cortex and is relayed to the oculomotor complex by supranuclear fibers. Apparently, the exact center for distribution of fibers to the nuclei of the 3rd and 6th cranial nerves, thought necessary for coordination of the ocular muscles when vergences are desired,
has not been found. It is known that the vergence system does not use the horizontal gaze center in the PPRF as the beginning of its final pathway to the EOMs. Therefore, a "nucleus of Perlia" in the oculomotor complex has been postulated but not substantiated. Nearly all pareses and paralyses of vergence eye movements can be explained on the basis of lesions involving the other known ocular neurological sites within the cortex and midbrain.

In the initial phases of the ocular examination, the ability to fixate and the alignments of the lines of sight are assessed monocularly (angles lambda) and binocularly (the Hirschberg test) in primary gaze by observation of the corneal reflex with respect to the center of the pupil. These assessments enable the clinician to estimate the angle of deviation for a large strabismus or tropia, which is evaluated for comitancy or incomitancy as noted in the previous section. Even if a phoria is present in primary gaze, a tropia may manifest at one or more of the cardinal directions, should an EOM be slightly paretic. The interpupillary distance (IPD) is measured so that the eyes can be later aligned with the optical centers of lenses in the phoropter or trial frame. These tests can be performed without the patient's spectacle correction in place, so that the examiner can obtain an excellent view of the eyes. Contact lenses may be worn when they do not interfere with the examiner's view. These tests enable the clinician to roughly gauge the fixational ability of the patient before more refined testing is conducted.

The angle of deviation (phoria or tropia) in primary gaze at distance and at near can be critically assessed via loose prisms with the cover test, an objective evaluation that is one of the more underrated of all diagnostic eye procedures. Lateral and vertical ranges of vergence ability, respectively, can be tested using a series of horizontal or vertical prisms arranged in a prism bar. Hence, the name bar vergences. The Maddox rod is an efficient and more accurate method for measurement of vertical deviations and vergences using loose prisms or a rotary prism. If necessary, two Maddox rods can be used to measure cyclo deviations and their associated ranges of cyclovergence. The closest distance from the spectacle plane to which the eyes can converge and maintain single binocular vision is measured by testing the near of point convergence (NPC).

The results of these tests through the habitual optical correction allow the practitioner to modify the procedures of the objective and subjective refractions (see Chapters 18 and 20) in order to achieve the most accurate refractive analysis possible. During phorometry (see Chapter 21), the data from these tests can be refined even further to the point that the final optical correction including refractive and prismatic components can be prescribed. The cover test and, perhaps, bar vergences are sometimes repeated through the predicted new optical prescription toward the end of the examination, so as to confirm the potential correction's expected effects on the deviation and associated vergences at near or distance.

### Observation of the Corneal Reflections

#### Angles Lambda (or Kappa)

Clinicians must objectively verify that the patient's eyes are looking in the direction that they are supposed to. One might conclude falsely that this is an easy assignment, given that the eye should be pointed directly toward the object of regard. Upon more critical inspection, however, the globe generally appears to be viewing at an angle temporal to the object of regard. This is because the optical components of the eye are not aligned with the line of sight, but along an optical axis at an angle temporal to it. The pupil, in particular, may not be centered exactly on the optical axis of the eye. Because the center of the pupil is an easy landmark to locate by visual inspection, a special "axis" has been assigned to it: The imaginary line normal to the cornea and containing the center of the pupil is called the pupillary axis. To identify the position of the line of sight, which has no anatomical landmark that is available to the clinician, its angular position is noted relative to the pupillary axis. The angle between the pupillary axis and the line of sight normally averages +5 degrees, ranging from +3 to +7 degrees, and is known as angle lambda. The angle is plus (+) when the line of sight is nasal to the pupillary axis (the usual situation) and negative (−) when temporal.

The route of the line of sight through the pupil can be located by observing the corneal reflex of the object of regard, which is in approximately the same plane as the entrance pupil. With the left eye occluded, typically using a handheld occluder, the patient is asked to fixate a penlight held in front of one of the clinician's eyes. The penlight is directed at the midpoint between the patient's two eyes from a distance of approximately 40 to 50 cm (Figure 10-15). While sighting just over the penlight, the clinician notes the position of the corneal reflection with respect to the center of the entrance pupil of the patient's right eye. The corneal reflex usually appears approximately 0.4 mm nasal to the center of the entrance pupil, because it has been shown that 1.0 mm of displacement corresponds to an eye rotation of 22°, or 12.5 degrees. Switching the occlusion to the right eye, the position of the corneal reflection is noted with respect to the center of the entrance pupil of the left eye. Hence, the angle lambda is assessed for each eye according to the lateral (almost always nasal) displacement of the corneal reflex relative to the pupillary center.

Although the average position of the corneal reflex will be 0.4 mm nasal of center (+), there is some individual variability accounting for normal displacement.
Figure 10-15
Assessment of angle lambda of a young patient’s right eye, with the left eye occluded.

of the reflex from +0.25 mm to +0.6 mm, corresponding to angle lambdas of +3 to +7 degrees. Seldom will the angles be negative or zero (at the pupillary center) or equal to +1.0 mm or greater in eyes that are not fixating eccentrically. Angles lambda of the two normal eyes are rarely significantly different, such that the monocular reflex positions of the two eyes should be identical. If the location of the monocular reflex in one eye is significantly different than in the other, the clinician should suspect strabismus (see Chapter 31), which can be accompanied by reduced monocular visual acuity in the deviating eye. Angle kappa (between the visual and pupillary axes) was confused with angle lambda in the early literature, and this test is even today sometimes called a test for angle kappa. This distinction is clinically inconsequential.

The Hirschberg Test and Krimsky Method
With both eyes unoccluded and the patient still fixating the penlight, the clinician may note the positions of the corneal reflexes in both eyes under binocular conditions, and compare these with the corresponding positions noted under monocular conditions. This method of assessing the presence or absence of strabismus is called the Hirschberg test. When the eyes are unoccluded, the corneal reflexes should remain in their monocular positions unless a strabismus is present. In a strabismus one reflex will move away from its monocular position in evidence of a strabismic deviation. The magnitude of the deviation can be estimated by the amount of movement of the reflex in millimeters (1.0 mm = 22'), or loose prisms of increasing power can be placed in front of the fixating eye until the reflex of the deviating eye has matched its monocular position relative to the pupillary center. The latter is the Krimsky method for measurement of the strabismic angle of deviation.

The Hirschberg test and the Krimsky method are relatively inaccurate in comparison with the cover test, noted later, but in certain situations they are the best methods available for identification and measurement of the strabismic angle of deviation. The tests are particularly suitable for infants and young children, or for those adults who cannot or will not respond or cooperate appropriately. Corneal reflexes are recorded on film during certain photorefractive screening techniques, and a binocular photograph is shown in Chapter 18 (see Figure 18-46). In the examination of a cooperating adult, the Krimsky method is of less value, because the cover test will ultimately measure the deviation within ±2", but the Hirschberg test can serve to forewarn the clinician of a strabismic deviation prior to the cover test, and takes only a little time and effort in conjunction with the assessment of angles lambda.

Interpupillary Distance (IPD)
The entrance pupils determine the size and location of the bundles of light that enter the eyes and stimulate the retinas. The horizontal distance between the centers of the entrance pupils is called the interpupillary distance (IPD), or merely pupillary distance (PD). The IPD can be measured for distance fixation and also for near fixation. The “distance IPD” is useful for the horizontal placement of the optical centers of spectacle lenses before the entrance pupils of the eyes in primary gaze, such that the appropriate amounts of lateral prism are located before the eyes (see Chapters 23 and 24). Similarly, the distance IPD is used to separate the optical centers of the interchangeable lenses in the refractor during the subjective distance refraction (see Chapter 20). It is, thus, important for the clinician to accurately measure the IPD to later assess the visual system using other tests and to properly prescribe the optical correction. The distance IPD is related to the amount of binocular convergence required for bifoveal fixation of a target by the equation

\[ \text{Convergence}(\)\(^{\circ}\) = \frac{\text{IPD (in cm)}}{d'' (in cm)} \]

where IPD is the interpupillary distance in centimeters (cm) for bifoveal fixation of a distant target, and \(d''\) is the distance in meters (m) of the target plane from the midpoint between the centers of rotation of the eyes (Figure 10-16). The distance (\(d''\)) is, therefore, 0.013 m (13 mm) longer than the distance from the anterior corneal surface to the target plane (\(d'\)). The clinician should know that patients with extremely large distance IPDs have increased demands for convergence to a near target and that patients with small IPDs have reduced convergence demands at near. The mean distance IPD
for adults is 64 mm, and for children the IPD ranges from 50 to 60 mm.

The "near IPD" is the distance between the pupillary axes where they pierce the spectacle plane as the patient fixates a near target bifoveally. The near IPD is important in determining the nasal decentration of multifocal segments and the near zones of progressive-addition lenses (PALs) into their proper positions inferiorly before the eyes (see Chapter 23). Although the near IPD can be measured, there is a simple trigonometric relationship among the distance IPD, near IPD, vertex distance (vd), and distance (d) of the target plane from the spectacle plane (see Figure 10-16). By similar triangles, the following equation can be derived:

$$\text{near IPD} = \frac{d}{d''} \times \text{distance IPD}$$

where $d''$ is the distance of the target plane from the midpoint between the centers of rotation of the eyes (see Figure 10-16), which is longer than the distance from the spectacle plane to the target plane (d) by an amount equal to 13 mm plus the vertex distance. These above equations treat the pupillary axes as if they were lines of sight, but the relevance of the discrepancy is subclinical. The near IPDs for the average male and female patient are approximately 3.7 mm less than the corresponding distance IPDs, noted earlier. The difference between near and far IPDs does not reach 4.5 mm until the distance IPD is greater than 75 mm, and the difference does not fall to 3.0 mm until the distance IPD is 50 mm or less. Hence, multifocal segments are usually decentered nasally by a standard 2.0 mm in each eye. The interpatient variation of the differences between near and far IPDs is generally subclinical and ignored.

Clinical Evaluation

The centers of the entrance pupils are thought to be difficult to precisely locate in the clinical situation, which is the primary reason why the Krimsky method is felt to be inaccurate. Hence, the identical distance between two anatomical landmarks on the eyes, usually the temporal pupillary margin in one eye and the nasal pupillary margin in the other, is most often used to assess the IPD. The distance between these landmarks is measured with a millimeter rule and should be equal to the distance between the pupillary centers if the pupils are symmetric. Alternatively, the temporal limbus of one eye and the nasal limbus of the other might be used, for instance, when anisocoria is present. The IPD can be determined for fixation at distance and at near.

To measure the distance IPD, the examiner should be standing or sitting directly in front of the patient at a distance approximating 40 cm. The examiner's eyes should be in the same horizontal plane as those of the patient. Room illumination should be sufficient for the examiner to identify the landmarks to be used for the measurement (i.e., the pupillary margins or limbi) and to read the inscriptions on a millimeter rule. The ruler, often called a "PD stick" in clinical parlance, is held horizontally in the patient's spectacle plane by one hand of the examiner, slightly below the pupils. The examiner closes the right eye and, with the other hand, points to his or her open left eye. The patient is instructed to fixate

![Figure 10-16](attachment://image.png)

**Figure 10-16**

Relationship of the interpupillary distance (IPD) to convergence demand for a near target, and of the distance IPD to the near IPD.
bifoveally on the examiner's open left eye, while the examiner's left eye sights along the line of sight of the patient's right eye and aligns the zero mark of the ruler with the temporal pupillary margin or limbus of the patient's right eye (Figure 10-17). The examiner opens the right eye and closes the left, switching the pointing hand to delineate his or her right eye. The patient is asked to fixate bifoveally the examiner's open right eye. The examiner's right eye sights along the line of sight of the patient's left eye, and the distance IPD is specified on the ruler where it intersects the corresponding nasal pupillary margin or limbus of the patient's left eye.

The patient does not actually fixate a distant target, but the difference between the examiner's IPD and the patient's IPD is not enough to deviate the patient's eyes from viewing straight ahead by an amount that would alter the measurement significantly. The IPDs of patients who are strabismic can be measured by occluding the patient's left eye while the PD stick is being aligned with respect to the patient's right eye, and then by occlusion of the patient's right eye when the reading is determined on the left eye. In this way, both eyes are viewing straight ahead at the appropriate times during the measurement procedure.

The near IPD can be measured immediately at the end of the distance IPD procedure by keeping the ruler in the same position. The patient is instructed to refixate on the examiner's reopened left eye as the examiner closes the right eye and points to the left eye. The examiner confirms that the zero position on the ruler still marks the temporal pupillary margin or limbus of the patient's right eye. With his or her left eye, the examiner then sights across to the patient's left eye and notes the distance to the corresponding nasal landmark of the patient's left eye. This is the patient's near IPD, which can be measured at any working distance used by the patient so long as the examiner's eyes are placed at that working distance. The IPDs are recorded in millimeters with the distance IPD listed first, followed by a slash, and then the near IPD (i.e., 62/58).

It is sometimes assumed that half of the distance IPD (the "split IPD") should be the horizontal distance from the center of the bridge of the nose to the center of the pupil on either side. However, the eyes are seldom located symmetrically relative to the nose, such that the true distance of the left eye from the center of the bridge is usually different than for the right eye of the same patient. This distance is better called monocular PD and should be specified for each eye instead of the distance IPD or split IPD when the clinical situation warrants a more accurate description of the positions of the pupillary centers. Precise monocular PDs are necessary for the prescription of highly powered spectacle prescriptions (see Chapter 33) and especially for PALs (see Chapter 24). Use of the split IPD persists because, as an approximation, it is often "close enough to get the job done" without having to measure the monocular PDs separately.

Monocular PDs can be measured using a PD rule to the center of each of the patient's pupils, in a manner similar to that described earlier. The patient fixates on the examiner's left eye, the examiner's right eye being closed, and the zero mark of the PD rule is aligned with the center of the patient's right pupil. The distance to the center of the patient's bridge is noted and becomes the right monocular PD. The patient then fixates the examiner's opened right eye, the examiner's left eye being closed, and the zero mark of the rule is moved to the center of the patient's bridge. The distance to the center of the patient's left pupil is noted and becomes the left monocular PD. When precise monocular PDs are required, this method may not be as accurate as necessary, because the exact positions of the pupillary centers and the center of the bridge of the nose are difficult to isolate. The monocular PDs are recorded in millimeters with the right PD listed first, followed by a slash, and then the left PD (i.e., 33/31).

Some practitioners prefer to use the corneal reflexes as landmarks for measurement in these procedures. Instead of using the eyes of the examiner as fixation targets for the patient, the examiner's pointing hand is used to hold a penlight as a fixation target immediately below the examiner's left and right eyes during the appropriate times during the measurements. Use of the corneal reflex eliminates the need to estimate the position of the center of the pupil. However, the corneal reflexes mark the positions of the lines of sight and, as has been noted, average 0.4 mm nasal to the center of the entrance pupil. Therefore, measurement by corneal reflection will usually underestimate the monocular PD by 0.2 to 0.6 mm in each eye or the IPDs by a total of

![Figure 10-17](image-url)

The millimeter ruler is zeroed on the temporal limbus of a young patient's right eye during the measurement of the distance IPD. The patient is fixating the examiner's left eye.
0.4 to 1.2 mm, depending on the individual patient's angles lambda.

A special instrument called a pupillometer is recommended when monocular PDs are a necessity. Several pupillometers are available that measure to either the centers of the pupils or the corneal reflexes, depending on the instrument, as reviewed by Brown. These are optical devices that each have a spectacle-like padded bridge that rests on the bridge and upper sides of the nose during measurement. Thus, the center of the bridge of the nose is precisely located with respect to the two optical pathways of the instrument. The devices can be battery-operated and present a fixation light directly in front of each eye of the patient along the sighting paths of the examiner. A reticle projected at the plane of the patient's pupil allows measurement of the monocular PD by alignment of the reticle with the center of the corneal reflex or the center of the pupil. The distance IPD is a simple addition of the two monocular PDs. Some pupillometers allow adjustment of the two optical pathways to ascertain the near IPD. A pupillometer is shown in Chapter 24 (see Figure 24-40).

**The Cover Test**

The "cover test" is an objective method of evaluating and measuring the deviation of the lines of sight from those directions necessary for bifoveal fixation of a target. It is a test that is unnecessary in the monocular patient but of much importance in the binocular patient. The test is accomplished in three segments: (1) observation of fixation, (2) the unilateral cover test, and (3) the alternating cover test, with measurement of the deviation using loose (Figure 10-18) or bar prisms. The other equipment necessary for the cover test are an occluder, or "cover paddle" (Figure 10-19), and targets for distance and near viewing. The clinician is seated beside the patient, and in front of the patient by a short distance of perhaps 25 to 40 cm (Figure 10-20). The clinician must be close enough to be able to critically note movements made by the patient's eyes yet not block the vision of the patient. The room must be well lighted to promote visual inspection of the eyes.

The cover test is performed with the patient wearing his or her habitual optical correction (spectacles or contact lenses) in the early phases of the eye examination (see Figure 10-20). The clinician may wish occasionally to do a cover test without the correction, or with an updated correction in the trial frame nearer the

**Figure 10-18**

A set of loose prisms used in the cover test. Note the red lens at the left, which can be used in the red lens test for incomitancy and the Maddox rod test for vertical phoria.

**Figure 10-19**

A typical occluder or "cover paddle," above in this photograph, which is often accompanied by a Maddox rod at the other (lower) end.

**Figure 10-20**

Performance of the unilateral cover test with the patient fixating a distant target. Note that the spectacle correction is being worn and that the clinician is in a position to closely monitor the movements of the eyes.
Observation of Fixation
The ability to fixate should have already been assessed by the clinician earlier in the eye examination (see discussions of angles lambda and the Hirschberg test). Nearly all patients except for the very young, anxious, hyperactive, or inattentive should be able to sustain steady fixation for 10 seconds or more, monocularly and binocularly, which is sufficient to conduct the cover test. At the beginning of the cover test, the clinician should watch the right eye to see if it can maintain steady fixation at distance while the left eye is covered with an occluder for several seconds. Upon removal of the occluder, the right eye should maintain fixation. Switching occlusion to the right eye, the left eye should attain fixation at distance and maintain it when the contralateral occlusion is removed.

If latent nystagmus or reduced central vision keeps monocular fixation from being steady, it becomes more difficult or impossible (depending on the severity) to assess the necessary eye movements in the upcoming segments of the cover test. Unsteady monocular fixation can also be an indication of eccentric fixation. Eye movement in the fixating eyes should not occur immediately after removal of the contralateral occlusion in patients capable of bifoveal fixation. The formerly occluded eyes, showing heterophoria (or merely "phoria") will move to take up fixation after the occlusion is ended unless the eyes have zero phoria (orthophoria). If movement of either or both of the fixating eyes is detected immediately after removal of the contralateral occlusion, strabismus may be present. The strabismus, also called heterotropia (or merely "tropia") will be categorized and measured during the rest of the cover test. Hence, the initial segment of the cover test determines if fixation is sufficient to conduct the rest of the segments and tentatively establishes whether phoria or tropia exists.

Unilateral Cover Test
The unilateral cover test confirms the presence of a phoria or tropia and defines its component directions (eso, exo, hyper, or hypo). The knowledgeable practitioner can also obtain a general idea about the deviation's magnitude. In the case of a strabismus, the unilateral cover test further classifies the tropia as alternating or unilateral and, for the latter, in which eye the deviation is manifested. The strabismus may also be characterized as constant or intermittent. The cover test can be performed in different directions of gaze for assessment of incomitancy, but such testing is not a part of the routine cover test. Testing for incomitancy should be performed in all cases of strabismus, as noted earlier in this chapter.

The unilateral cover test is accomplished by observing the movement of the fixating eye when the other eye is first covered with the occluder for 2 or 3 seconds and then by observing the movement of both eyes immediately after the contralateral occluder is quickly removed (see Figure 10-20). The eye movements are assessed while the right eye fixates the distant target, covering and uncovering the left eye, and then while the left eye fixates, covering and uncovering the right eye. It is important to allow the eyes to fuse the target, if possible, before switching the cover paddle from one eye to the other. In practice, it is not possible to pay attention to both eyes at the same time when uncovering an eye. Hence, the clinician covers and uncovers an eye through several cycles, first watching the fixating eye for a few cycles, then the other for a few cycles, in order to evaluate the movements of both.

When fusion is broken by the occluder, the occluded eye will assume its normal tonic vergence position while the unoccluded eye will either: (1) remain fixating the target or (2) take up fixation of the target. The movement of the occluded eye will not be visible to the clinician. When the contralateral occlusion is removed, the fixating eye then either (3) remains fixating the target or (4) gives up fixation to the formerly occluded eye. Simultaneously, the formerly occluded eye (5) will take up fixation of the target, (6) will remain deviated, or (7) may not move if orthophoric. On the basis of these eye movements, or the lack thereof, the clinician reaches a diagnosis of the type of phoria or tropia present and deduces the direction in which the occluded eye's line of sight was pointed during the occlusion.

In phorias, only the eye that is covered moves. It is obvious that, in orthophoria, neither eye will move after the contralateral eye is occluded or unoccluded. In esophoria, the occluded eye will adduct to its tonic vergence position, and once occlusion is removed, it will be seen to abduct (move temporally) as it takes up fixation along with the other eye. In exophoria the occluded eye will abduct to its tonic vergence position and will be seen to adduct (move nasally) when the occlusion is removed. The same response will be generated regardless of the eye that is occluded. Hence, a phoria is in the direction opposite to the movement seen upon removal of the eye's occlusion. The experienced practitioner will be able to assess the relative magnitude of the phoria by the amount of eye movement that is observed. However, definitive measurement of the phoria's magnitude will be performed later during the alternating cover test.

The eye with hyperphoria will move down as it is uncovered, and the other eye will appear to have
This is usually not malingering, but a mechanism that is accomplished by the alternating strabismic when the clinician is toward a deviating eye. The clinician would perceive the eyes to be both moving as the cover paddle is brought forward. The alternating cover test will falsely appear to indicate orthophoria, unless the clinician perceives that an eye is deviating from the temporal side. One way of minimizing this problem is to center the cover paddle over the forehead of the patient and to cover the appropriate eye from above by moving the paddle down and out. Or, the clinician could center the cover paddle over the nose and suddenly cover one eye or the other by moving the paddle up and out. In these manners, the patient may not predict which eye will be occluded. If the clinician ever reaches the alternating cover test (see later), believing an orthophoria to exist, and then measures an unexpectedly large deviation, the chances are that an alternating tropia was missed earlier. The clinician will need to go back and more critically perform the unilateral segment of the cover test.

A constant tropia appears always as a tropia when tested at the same distance. If the deviation appears to be tropia in some instances during the cover test and appears to be phoria on other instances or occasions, the tropia is intermittent.

The expert clinician should be aware of a few conditions, other than phorias and tropias, that can cause eye movements during the unilateral cover test. A small flick of an eye may occur, prior to taking up fixation, when a large phoria is broken by occlusion of the other eye. In these cases the phoria may actually be an intermittent tropia or a small-angle strabismus called a microstrabismus or microtropia. Eccentric fixation and unsteady fixation can also cause movement, but these should have been detected previously. Uncorrected or residual anisometropia can create the illusion of esophoria or esotropia because of greater accommodative convergence initiated by the more hyperopic eye.

Alternating Cover Test

The alternating cover test confirms the direction and measures the magnitude of a phoria or tropia. The alternating cover test begins immediately after the unilateral cover test has ended, using the same occluder, target, and patient/clinician relationship. Fusion is broken by covering the right eye for 2 to 3 seconds and allowing the left eye to take up fixation, if necessary. The paddle is then quickly moved, without pausing between the eyes, to cover the left eye for 2 or 3 seconds while the right eye takes up fixation. Binocular fixation is not permitted as in the unilateral cover test. The paddle is repeatedly alternated from one eye to the other, pausing 2 or 3 seconds over each eye to allow for alternation of fixation. The clinician observes the direction and relative magnitudes of the eye movements as each eye is alternately uncovered and takes up fixation of the target.

Regardless of whether a phoria or tropia exists, the eyes will alternately each move temporally to take up fixation as occlusion is alternately removed if a deviation exists in the “eso” direction, and the eyes will move nasally in the case of an “exo” deviation. An eye will
move downward for a "hyper" deviation, in which case the other eye will move upward, indicating a corresponding "hypo" deviation. These results should confirm the directions of the deviations that were diagnosed as a result of the unilateral cover test. The clinician should have an estimate of the magnitude of the deviation on the basis of the amount of eye movement seen during the first portions of the cover test. The clinician then proceeds to the measurement of the deviations using loose prisms or a prism bar (see Figure 10-21).

The occluder is left in place before one of the eyes and a loose prism (or a bar prism as shown in Figure 21-11) of the estimated magnitude and direction is placed before the occluded eye (under the cover paddle). The flat posterior surface of the prism should be placed in the spectacle plane of the eye or immediately in front of a spectacle lens worn by the patient. The magnitude of the deviation is measured by neutralizing of the eye movements during the alternating cover test with prisms of increasing power until the residual deviation is zero. This amount of prism is noted, then the prism is increased until the deviation is reversed and bracketed (Figure 10-21). An "eso" deviation is neutralized with base out (BO) prism, an "exo" deviation with base in (BI) prism, a "hyper" deviation with base down (BD) prism, and "hypo" deviation with base up (BU) prism. Prismatic power is always increased incrementally underneath the cover of the occluder, which is kept before one eye as loose prisms are exchanged. The reason that unilateral occlusion is maintained is to discourage any prior latency from redeveloping should bifoveal fixation be allowed.

The accuracy of this technique is influenced by the fact that the minimum deviation recognizable by the clinician is about 2°. The manifest deviation is that neutralized when eye movement is first eliminated. Between the last minimum detectable eye movement in one direction and the first recognition of reversal, there is usually a range of 2 to 4° over which no eye movement is observed. The amount of the latent deviation is taken to be the midpoint of the bracketed range. If orthophoria is apparent, reversal should be achieved in both lateral directions with BI and BO prism.

Some patients have both lateral and vertical component deviations. To assess both magnitudes, BO or BI loose prisms are used to first neutralize the eso or exo deviation. Loose vertical prisms can then be held over the horizontal prism (or the horizontal prism can be held by the patient over the other eye) for neutralization of the remaining vertical deviation. Although the precision of prismatic neutralization is adequate for lateral deviations (±2°), it is not at this point generally acceptable for vertical deviations. Hence, some methods of enhancing the precision of the alternating cover test are to be described.

Small eye movements during the alternating cover test are often difficult to discern. A low power (approximately 5°) loose prism can be used to find and measure small angle (3° or less) deviations and is particularly useful when orthophoria is suspected or when a small vertical deviation requires more accurate measurement. The prism is inserted BI over one eye during the alternating cover test, and the magnitude of the residual esophoric deviation is observed. The prism is then inserted BO over the same eye, and the magnitude of the residual exophoric deviation is observed. If the eye movements appear to be of the same magnitude in the two prismatic orientations, but opposite in direction, lateral orthophoria truly exists. Exophoria exists if the eye movements appear lesser when the prism is oriented BI, and esophoria exists if the movements are lesser when the prism is BO. The experienced practitioner can estimate the magnitude of the small lateral deviation from the relative amounts of eye movements when the prism is placed BI versus BO. Similarly, the prism can be oriented BU and BD to rule out vertical orthophoria or to measure a small vertical deviation.

It is also possible in such a situation that the patient's manifest phoria could be from 3 to 7° or be latent to an even larger extent. The practitioner might not see eye movement when the 5° prism is placed before the eye in a manner that will correct the deviation. This could occur, for instance, if the patient is 3 to 7° esophoric and the 5° prism is placed over the right eye BI and then BO to observe the direction and magnitude of eye movement. The clinician would likely see a large amount of eye movement through BI prism and no movement with BO prism. The large amount of movement in one direction and the lack of movement in the other direction...
should tip the clinician that the deviation is approximately 5° and that a prism of larger magnitude will be necessary to achieve reversal in the latter.

It is important that the movements of the eyes be reversed when the BO (or BU) prism is used in comparison with the BI (or BD) prism. If reversal does not occur, the angle of deviation is likely to be latent and larger than the correcting prism. The clinician should proceed by increasing the amount of prism in the orientation of nonreversal until a reversal has occurred. The latent phoria in some cases may be significantly larger than the manifest phoria, especially for lateral deviations. A common error in the measurement of a phoria deviation, seen so often by one of the authors (WJB), is to omit the reversal of the deviation. A lateral orthophoria is not proven to be orthophoria until reversed in both horizontal directions.

Deviations can also be more accurately detected subjectively by asking the patient to identify the perceived movement of the target as the cover paddle is moved from one eye to the other. Immediately after the cover paddle is moved from the right to the left eye, and before a fixational eye movement has been made, the target previously seen by the fovea in the left eye is now imaged at a point on the retina of the right eye. If the eye is deviated in an eso direction, the image is located on the nasal retina and is projected into the patient's temporal visual field. If deviated in an exo direction, the image is located on the temporal retina and is projected into the nasal visual field. Hence, the patient will perceive that the target moves "with" the cover paddle when there is esophoria and "against" the paddle when there is esophoria. The clinician can neutralize the perceived movement of the target by addition of the appropriate prisms before the eyes in a manner identical to that described earlier. Vertical phorias may be identified and neutralized subjectively in this manner as well, allowing for the enhanced precision of measurement necessary for small vertical phorias (±1°). Alternatively, even more precise vertical measurements may be achieved using the Maddox rod (±0.5°), a technique especially recommended by the authors and explained later in this chapter.

After the entire cover test has been performed at distance, the procedure is repeated at near. The patient should fixate a single letter, one line above the threshold of the poorer eye, or an equivalent target at his or her habitual working distance. The common working distance (40 cm in front of the spectacle plane) is used in many instances. For children, a picture may be used as long as it contains enough detail to attract the attention of the patient and stimulate accommodation. It is beneficial to have the patient hold the target at the appropriate working distance so that both of the clinician's hands are free to manipulate the cover paddle and prisms (see Figure 10-21). A better stimulus to accommodation and fusion will also be achieved when the near target is held by the patient. An overhead lamp should be directed to increase the illumination of the near target. The patient should be asked to follow the target with the eyes if it appears to move and be reminded during the procedure to keep the near target clear, such that accommodation is kept relatively stable. Otherwise, the procedure at near is the same as the procedure at distance.

Recording

Results of the cover test should be reported for distance and near. The magnitude in prism diopters, direction (eso, exo, hyper, hypo), and type of deviation (phoria or tropia) should be expressed. If a vertical deviation or unilateral tropia, the laterality of the deviation must be signified. The word "alternating" must be inserted if the condition is an alternating tropia. The frequency of a tropia (i.e., "constant" or "intermittent") and the comitancy of a tropia should be indicated (i.e., "comitant" or "incomitant"). Some examples: 4° exophoria (4° XP); 2° right hyperphoria (2° RHyperP); 3° eso + 1° left hyperphoria (3° EP + 1° LHyperP); 8° constant comitant alternating esotropia (constant comitant 8° Alt ET); 12° right intermittent incomitant exotropia (intermittent incomitant 12° RXT); 6° left constant comitant alternating hyper-tropia (constant comitant 6° Alt LHyperT); 8° exo + 4° right constant incomitant hypotropia (constant incomitant 8° XT + 4° RHypoT).

Cyclophorias and Cyclotropias

The reader may note that the methods of measurement of cyclodeviations have not been mentioned previously in this chapter. It is generally believed that cyclophorias seldom alone contribute to muscle balance problems at distance or at near. It is apparent that a significant cyclophoria must accompany a simultaneous vertical deviation and is almost always associated with a paretic superior oblique. Unilateral paresis of a superior oblique may result in a cyclodeviation of 3 or 4 degrees, whereas bilateral paralysis can triple or quadruple that amount. Therefore, a critical screening for a vertical deviation using the cover test or the Maddox rod (see later) also serves to screen for a cyclodeviation. No optical devices can be reasonably prescribed for the alleviation of a cyclophoria. Hence, the reader is referred to Chapter 20 for a discussion of how fusional torsions may affect the astigmatic axis during the monocular and binocular refractions at distance and at near, and to Chapter 21, in which a detailed procedure for cyclophoria measurement with dual Maddox rods is presented.

Bar Vergences

Bar vergences are performed to measure the fusional (disparity) vergence reserves that the patient has available to obtain and maintain bifoveal fixation in the presence of a binocular deviation. The technique is
covered in Chapter 21, and a photograph is included as Figure 21-11. The basic relevance of the blur, break, and recovery findings was discussed in Chapter 5. The expected clinical findings, specific evaluations, and overall patient assessments are covered in detail in Chapters 21 and 22. Blur, break, and recovery findings are determined in the horizontal plane at distance and at near. Break and recovery findings may be determined in the vertical (sagittal) plane, but vertical vergences are usually only pursued at a single distance.

Bar vergences are performed “outside of” a phoropter and are analogous to vergences performed with rotary prisms with the phoropter, which are also described in Chapter 21. The effects of proximal convergence on the horizontal vergence amplitudes are reduced for bar vergences in comparison with rotary prism vergences and, as a result, bar vergences are thought by many practitioners to better represent the natural viewing situation. On the other hand, bar vergences require stepped increments of prism to be placed before a single eye, whereas rotary prisms split continuous increases between the two eyes and are less likely to disrupt vision during the prismatic alterations.

The Maddox Rod

The Maddox rod can be used to evaluate lateral phorias and cyclophorias as described in Chapter 21. However, it is especially well suited for the measurement of vertical deviations at this stage of the eye examination. A Maddox rod is usually present on the opposite end of a cover paddle (see Figure 10-19) and is placed before one eye with its baffles oriented vertically (Figure 10-22, A) as the patient fixates on a penlight at 40 cm in a darkened room. The Maddox rod is usually placed over the right eye. A red lens may or may not be placed before the left eye to equalize the color and luminance of its view. The test can be performed at distance by shining a bright spot on the wall in a dark room, but it is usually only performed at near because this is more convenient and because vertical phorias should not significantly alter from distance to near.

The Maddox rod will break fusion, and the nonsuppressing patient should perceive a red horizontal streak (Figure 10-22, B) in the right eye covered by the Maddox rod and a bright white spot of light with the other (left) eye. The spot of light will appear red if a red lens was placed before the left eye. If the eyes are vertically orthophoric, the patient will perceive the bright spot of light to be directly in the middle of the red streak. If the streak runs above (right hypo = left hyper) or below (right hyper = left hypo) the bright spot, loose prisms or a rotary prism (Figure 10-23) can be used to neutralize the deviation by bracketing of residual orthophoria. When the streak apparently touches the bright spot the magnitude of the vertical phoria is approximately 1.0° and when overlapping but not centered the phoria is approximately 0.5°. A detailed procedure is discussed in Chapter 21.

The precision of this technique (±0.5°) is such that misadjustment of the typical spectacle correction interferes with accurate measurement of the vertical phoria. It is best to measure the phoria without the habitual spectacle correction in place (contact lenses are okay) if the true vertical phoria is the end point desired. Residual vertical phorias induced by poor frame fit or optical center placement can be ruled out or verified by comparing the vertical phorias measured with and without wearing of the suspect spectacles. If verified, the clinician can then go about eliminating the problem by adjustment or re-order of the optical correction.

Figure 10-22

The baffles of a red Maddox rod present on one end of a cover paddle (A). When placed over the patient’s eye with the baffles vertical, the penlight is seen as a horizontal streak perpendicular to the baffles (B).
Near Point of Convergence

The near point of convergence (NPC) is the point of intersection of the lines of sight when maximum fusional (disparity) convergence is used. The distance to this point from the spectacle plane, typically taken to coincide with the middle of the forehead, is the "NPC finding." The NPC is related to the patient's ability to converge the eyes while simultaneously maintaining bifoveal fixation. Binocular vision problems, eyestrain (asthenopia), discomfort in performing near work, and reading difficulties may occur in persons with inadequate NPC findings. As a result, the NPC finding is used as a screening test for obvious convergence insufficiencies.

The NPC should be found with a nondescript target, such as a penlight or another simple target on a tongue depressor (Figure 10-24), to help isolate the fusional (disparity) vergence response from accommodative convergence. The accommodative and vergence subsystems are tightly cross-coupled\(^{62}\) with an accommodative response normally accompanied by vergence eye movement. Using an accommodative target stimulates accommodative demand and accommodative convergence, which will affect (lower) the expected values for the NPC and recovery.\(^{63}\)

The examination room should be fully illuminated, and the patient should be wearing the habitual optical correction. The examiner should be positioned slightly off to one side of the patient but in a manner that allows observation of the patient's eye movements. The target is held approximately 50 cm away at the patient's eye level and is brought slowly along the midline toward the midpoint between the patient's eyes. The patient is instructed to follow the target inward with the eyes, as closely as possible, and to report if the target doubles or "breaks into two." The NPC is that point reached when the patient reports diplopia or when the examiner first observes loss of bifoveal fixation by the outward turning of one eye (see Figure 10-24). The target is then backed away from the eyes and the clinician notes the distance at which the deviating eye regains fixation. This is called the NPC recovery finding.

The patient may suppress the deviated eye when the NPC is passed. Fusion is lost and, in these instances, diplopia will not be reported. As the target is brought closer, it assumes a position in the nasal visual field of the fixating eye. To continue fixation, that eye turns in and the suppressing eye concomitantly turns out as the vergence becomes a version. Hence, the clinician must be vigilant in monitoring the eyes for movement as the target is brought closer and closer to the top of the nose. The anteroposterior distance from the middle of the forehead to the plane of the target is measured with a ruler and recorded in centimeters to within 0.5 cm. If the patient is able to converge to the point that the target
comes in contact with the patient's nose, the NPC finding is often recorded as "nose."

The mean NPC is 3 cm (±4 cm) from the spectacle plane (midforehead), and the recovery finding is 2 or 3 cm larger. NPC findings greater than 7 cm and recovery findings greater than 10 cm are generally regarded as inadequate and could be signs of a convergence insufficiency. Further testing and analysis would be warranted at near in the area of muscle balance and accommodation (see Chapters 21 and 22). The test may be repeated several times to check for fatigue, which is indicated by an increase of 3 cm or more between the first and last repetitive NPC findings.

### ACCOMMODATIVE AMPLITUDES AND FACILITY

#### Accommodative Amplitudes

Accommodative insufficiencies (see Chapter 4) can occur with normal and abnormal nervous innervation. Autonomic parasympathetic fibers from the Edinger-Westphal nucleus innervate the ciliary muscle which controls accommodation, but cortical processes preceding and monitoring the ciliary motor command signals are poorly understood. Screening tests for accommodative insufficiencies are typically addressed after the distance refractive correction has been determined, and their procedures are covered in detail in Chapter 21. However, some tests can be performed during the assessment of ocular motility in the early stages of the eye examination, if the refractive correction is not expected to alter considerably from that of the habitual correction. The accommodative amplitude, in particular, is measured with a technique that closely parallels the method used to assess the NPC.

The measurement of monocular and binocular accommodative amplitudes is discussed in Chapter 21, and the reader is referred there for the detailed procedures. The accommodative test uses a more finely detailed target to better stimulate accommodation in the finding of the near point of accommodation, and the end point is first sustained blur instead of diplopia (see Chapter 21). The reciprocal of the distance from the midforehead (approximately the spectacle plane) to the near point of accommodation, in meters, is the amplitude of accommodation, in diopters.

Monocular and binocular results should be recorded in the order and number of times they are measured (i.e., OD 7,7,6; OS 8,7,8; OU 7,5). The monocular amplitudes are measured along the line of sight in primary gaze, with the contralateral eye occluded, and the binocular amplitude is measured along the midline. This ensures that the binocular amplitude will normally be artifactually greater than the monocular amplitudes, because the distance to the target will be shorter in the monocular measurement for a given dioptric amplitude value (see Figure 10-16). Accounting for this artifact of measurement, however, the binocular amplitude is only slightly greater than the monocular amplitudes by a fraction of a diopter. This minimal effect is thought to be the result of the lack of convergence accommodation in the monocular situation. The difference between monocular and binocular amplitudes of accommodation is subclinical unless an accommodative abnormality exists. Accommodation is consensual and equal in the two eyes, so a difference of 1 D or more between the eyes may indicate a unilateral insufficiency. Further accommodative testing would be indicated (see Chapter 21).

Hofstetter derived formulas for the expected maximum, mean, and minimum accommodative amplitudes in the population from the normative data of Duane and Donders, a topic covered in great detail by Borish. The formulas were based on age. Accommodative insufficiency should be suspected in patients with amplitudes less the values calculated from Hofstetter's formula for the minimum accommodative amplitude (Table 10-4):

- Expected minimum amplitude = 15.0 D - [0.25 D × (age in years)]
- Expected mean amplitude = 18.5 D - [0.30 D × (age in years)]
- Expected maximum amplitude = 25.0 D - [0.40 × (age in years)]

The clinical measurement of accommodative amplitudes may be affected by visual acuity, target size and detail, depth of focus, patient effort, blur interpretation, ability to converge, refractive state, spectacle lens effects, and examiner technique. Although full room lighting is desired, excessive light should be avoided because of pupil constriction with resulting increased depth of

<table>
<thead>
<tr>
<th>Age</th>
<th>Minimum</th>
<th>Mean</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>12.5</td>
<td>15.0</td>
<td>21.0</td>
</tr>
<tr>
<td>20</td>
<td>10.0</td>
<td>12.5</td>
<td>17.0</td>
</tr>
<tr>
<td>30</td>
<td>7.5</td>
<td>9.5</td>
<td>13.0</td>
</tr>
<tr>
<td>40</td>
<td>5.0</td>
<td>6.5</td>
<td>9.0</td>
</tr>
<tr>
<td>50</td>
<td>2.5</td>
<td>3.5</td>
<td>5.0</td>
</tr>
<tr>
<td>60</td>
<td>0</td>
<td>0.5</td>
<td>1.0</td>
</tr>
</tbody>
</table>
focus, which can increase the measured amplitude. Uncorrected refractive errors will alter the location of the near point of accommodation: Uncorrected hyperopes will have erroneously low amplitudes, and uncorrected myopes will appear to have greater amplitudes, than would be the case with the proper refractive correction.

In addition to the effect of age, amplitudes may also be reduced by disease, drug reactions, or functional problems. Illnesses such as mumps, measles, influenza, anemia, and encephalitis may reduce amplitudes. Multiple sclerosis and myotonic dystrophy can have a similar effect. Transient accommodative paresis may occur in diabetics. Atrophy of the ciliary body in some glaucomas may produce accommodative problems. A lesion in the Edinger-Westphal nucleus or pineal tumors can cause reduced accommodation. Iridocyclitis, sinus problems, focal infections, dental caries, or injections may be suspected in unilateral deficiencies. Trauma to the craniocervical region, often seen in whiplash, may also be responsible for bilateral problems, whereas trauma, in the form of a tear in the iris sphincter or the zonules of Zinn, might reduce a monocular measurement. Systemic drugs such as alcohol, central nervous system stimulants and tranquilizers, antihistamines, tricyclic antidepressants, and phenothiazines may lead to bilateral accommodative insufficiencies. Topical agents such as cycloplegics may have unilateral or bilateral effects, depending on their administration. If a unilateral decrease in accommodation is noted in conjunction with a dilated pupil, Adie’s tonic pupil and 3rd cranial nerve problems need to be ruled out.

**Accommodative Facility**

The ability to alter accommodation rapidly and accurately is called accommodative facility. The evaluation of accommodative facility is discussed in Chapter 21, and the reader is referred there for discussion of the detailed procedure. Briefly, the clinician asks the patient to repeatedly alternate vision between a distant and a near (40 cm) target, both being slightly above the acuity threshold of the patient (approximately 20/30 or 6/9), making each target clear before the ocular focus is immediately changed to the next target. With both targets well illuminated, the patient verbally indicates when a target becomes clear after alternating focus from one target to the other, then reverses focus to the original target and reports when it again becomes clear. Alternatively, flipper bars of lenses +2.00 OU and −2.00 OU can be used to alternate the accommodative demands required to clear binocularly a near target.

The clinician notes the number of full cycles per minute (cpm) completed in 60 seconds binocularly and monocularly in each eye and identifies with which target (distance or near) the patient may be having a problem. Is there an accommodative facility insufficiency and, if so, is the problem with relaxing accommodation or with increasing accommodation? The minimum value for adults is 12 cpm binocularly. Monocular findings should be approximately 2 or 3 cpm faster (higher) than the binocular findings. The actual number of cycles completed by the patient should be recorded in cycles per minute (i.e., facility: 6 cpm OU, 5 cpm OD, 6 cpm OS).

Facility problems may result in difficulty focusing from distance to near, or vice versa. Students may complain about not being able to see the blackboard after near-point activities. Older patients report blur of distance objects and extensive time needed to clear near targets. Asthenopia, eye rubbing, and blinking are common with infacility. Aging of the crystalline lens will decrease facility, as will diabetes, Grave’s disease, measles, and chronic alcoholism. Patient’s with Adie’s tonic pupil may show unilateral facility problems. Systemic medications with cycloplegic side effects can negatively influence facility results.

**SUMMARY**

The eye examination includes an assessment of ocular motility, usually performed in the early stages of the examination with handheld instruments or devices, and before the accommodative system and pupils are topically paralyzed by a mydriatic. Most clinicians develop routines for the series of ocular motility tests, such that they can be done in an efficient manner. During their performance, the astute practitioner may simultaneously conduct a gross external examination of the eyes, overall physical examination, and/or a friendly and engaging verbal case history. Confrontation visual fields (Chapter 15) can be skillfully performed immediately after observation of versions with the “H pattern” since the position of the clinician, visual target, and general format of these two tests are similar.

The ocular motility tests are seemingly uncomplicated, yet they must be accomplished in an exacting manner, observed with a critical eye, and interpreted with caution. The practitioner must have a detailed approach to distinguish normal from abnormal and to follow up abnormal findings with more complicated motility testing. The ocular motility findings have a direct bearing on the way subsequent portions of the eye examination are conducted. Their apparent simplicity belies a tremendous diagnostic value that is supported by a great deal of clinically relevant underlying knowledge.

**References**


