Physical and Refractive Characteristics of the Eye at Birth and During Infancy

front of the equator. The width of the rectus muscle insertions is between 6.8 and 7.6 mm at birth.

The depth of the anterior chamber in term newborns averages 2.6 mm and ranges from 2.4 to 2.9 mm as shown by ultrasound. Due to postmortem movement of the lens, in vivo ultrasonographic studies of anterior chamber depth are more reliable.

The iris has a different appearance in preterm than in older infants. The crypts appear less developed in preterm neonates and develop a more detailed architecture of crypts and hillocks with maturation. The color of the uveal tract appears paler in newborns than in older children. At birth, the iris in many white infants appears gray or blue, largely due to the pigment of the iris pigment epithelium, while other infants have a darker, even brown, iris from partial development of stromal mesodermal pigment. With time, the iris darkens as the stromal mesodermal cells mature and progressively develop more pigment. The iris blood vessels may contribute to the observed color of the iris. Due to immaturity of the dilator muscle of the pupil in the newborn, pupils dilate less than might otherwise be expected.

At birth, the lens volume is 90 cu mm, and the weight is 65 mg. The lens thickness in newborns varies from 3.4 to 4.0 mm, and the equatorial diameter is between 6.0 and 6.5 mm. Lens thickness does not correlate linearly with body weight. The curvature of the lens is nearly spherical at birth, with a radius of curvature of 5 mm for the anterior surface and 4 mm for the posterior surface. The curvature increases until 2 years of age. The volume and weight of the lens nearly double by 1 year of age. The greater spherical power in the newborn eye, when compared with the adult eye, contributes to the greater refractive power needed in the shorter eye of neonates, as will be discussed later. While the lens as a whole continues to grow throughout life, it becomes less spherical. The lens diameter decreases 0.3 mm in the first 18 months of life and another 0.2 mm between 2 and 5 years of age.

The lenticulovitreal adhesions (Weiger's hyaloid-capsular ligament) are very strong in infants. Attempts to weaken these with enzymes as a-chymotrypsin have been unsuccessful. Thus, intracapsular cataract surgery in infants can easily cause vitreous loss and is generally contraindicated.

The intraocular pressure in newborns is important since the clinician must occasionally measure it to rule out glaucoma. With various methods, the intraocular pressure in normal neonates has been reported as low as 8 and as high as 28 mm Hg. There are a number of variables that can affect these data, including whether the measurements are taken under anesthesia and, if not, whether the infant is crying, squeezing his eyelids, or straining at the time of measurement. A recent study found the mean intraocular pressure in unanesthetized noncrying premature infants to be 18 2 mm Hg.

Posterior Segment
At birth, the vitreous cavity measures 16.0 mm in sagittal diameter. By ultrasonography, the vitreous diameter has been shown to be decreased in certain developmental anomalies as fetal alcohol syndrome and holoprosencephaly. Remnants of the hyaloid vascular system can still be found in the eyes of premature infants. Jones found posterior remnants in the eyes of 95% of premature newborns but in only 3% of term babies. All the remnants resolved with time. Renz and Vygantas examined the vitreous cavity of 226 infants. They reported all the premature neonates with a gestational age less than 34 weeks (or 4 lb) to exhibit the complete hyaloid vessel reaching from the optic nerve head to the posterior capsule of the lens. At about 34 weeks, the center of the vessel appeared to resolve, which left a Bergmeisters papilla attached to the optic nerve head and a strand attached to the posterior lens capsule. The frequency of the remnants diminished until they were not observed at all after 41 weeks' gestational age (or greater than 9 lb). After resolution of the hyaloid vasculature,
quet's canal, consisting of the multilayered fenestrated sheaths of the vessel, persists in the eye. This canal follows the original course of the hyaloid vasculature and runs from the optic nerve to the posterior lens capsule. In infants, the vitreous is a gel with no liquid vitreous present. With time, the sodium hyaluronate content of the vitreous increases as the collagen fibers, which do not increase in number, become less concentrated.

Of all ocular structures at the time of birth, the retina is the least developed functionally. The retinal vessels first appear in the fourth month of gestational age from spindle cells at Bergmeister's papilla. These mesenchymal vessels advance peripherally toward the ora serrata. By the eighth month of gestation, the vessels have extended almost to the nasal ora serrata but have reached the equator on only the temporal side. Even in term infants, the retinal vessels on the temporal side have often not fully reached the ora serrata but do so within a few weeks. Microscopic maturity of the blood vessels does not necessarily correlate with the extent of vascularization of the anterior retina.27

Other than the retinal vessels and the general mild pallor of the retina (as well as the optic nerve) at birth, the retina appears similar to the adult retina. However, there are many differences on a histological level. At birth, the retinal capillaries have a relative endothelial hyperdensity. During the first year of life, the capillary basement membranes thicken, and the intramural pericytes develop. The macula is the last retinal area to reach maturity. Even after birth, the macular cones elongate and undergo other histological changes. In addition, the ganglion and bipolar cells continue to migrate peripherally. The examiner can appreciate some of the macular changes in preterm infants through the ophthalmoscope.28 Further details of macular development are presented later in this chapter. It is possible that the histologically immature newborn macula accounts for the poor fixation of infants in the first 3 months of life. However, the general inattentive nature of the newborn also contributes to their poor fixation.

VISUAL DEVELOPMENT (OTHER THAN ACUITY)

The development of vision as a whole is covered in Chapter 4. However, there are new data indicating the development of some parameters of vision other than acuity.

The visual field in infants can be used to indicate the development of the peripheral retina as well as the visual cortex. Mohn and Van Hot-van Duin found the visual field in newborns to extend 28 degrees to either side horizontally, 11 degrees superiorly, and 16 degrees inferiorly.29 The visual field expanded slowly immediately after birth but increased rapidly after 2 months of age. By 1 year of age, the upper visual field had reached adult size, while the horizontal and lower fields were still smaller than those in the adult. They attributed these changes to maturation of cortical and subcortical pathways.

Newborn infants can accommodate very shortly following birth. In a series of studies, Banks reported that accommodation was present as early as 1 month of age, which enables the newborns to intermittently fixate on near targets as determined by retinoscopy.30 Because the depth of focus of newborns is greater than that of adults for a number of reasons including a smaller pupil and poorer central visual acuity, their accommodation does not have to be as exact.

Color vision develops earlier than previously thought. Adams and colleagues tested 240 infants within the first week of life for discrimination between checkerboards by preferential forced looking techniques.31 They showed discrimination between gray and green, yellow, and red stimuli. However, they did not show discrimination between gray and blue stimuli. Contrast and illumination were controlled during these investigations. Thus, newborns have a limited ability to discriminate chromatic from achromatic stimuli.

DEVELOPMENT OF REFRACTION

When considering the development of re-
fraction, one must examine the contribution of each refractive surface. Then, the net effect of these components on overall refraction can be evaluated. The steep nature of the newborn cornea and subsequent flattening was discussed before, as was the high refractive power of the lens at birth. In the first 3 months of life, the lens power decreases approximately 4 D for term infants and 7.5 D for preterm infants. The sagittal (or axial) length of the eye increases dramatically in the first 2 years of life and less thereafter. Larsen reported the axial length to increase from about 16.5 mm at birth to about 20.8 mm at 2 years of age. In the next 11 years of development, he found the axial length to increase only another 2 mm.

Refractive Error

Most early studies of refraction in newborns were not done with the use of cycloplegic eyedrops and are subject to the criticism that the powerful accommodation present in infants introduced error. By administering atropine to infants, Cook and Glassock studied the refractive error of 1,000 newborn infants. They found simple hyperopia in 43.9%, simple myopia in 16.7%, hyperopic astigmatism in 29.1% and myopic astigmatism in 6.4% of the infants. Goldschmidt performed retinoscopy on 356 term newborns less than 10 days old who had received atropine eyedrops. He found the mean refractive error to be 0.62 D of hyperopia (± 2.24 with a 0.1 SEM). Myopia was found in 24.2% of his study population.

Gordon and Donzis reported that ten term newborns had a mean axial length of 16.8 mm, corneal curvature of 51.2 D, and lens power of 34.4 D which yielded a mean refractive error of 0.4 D of hyperopia. They confirmed the rapid elongation of the globe and corneal flattening in infants as previously reported. In addition, they calculated that lens power rapidly decreased from 43.5 D in premature neonates to about 24 D by 2 years of age. The mean net refractive error remained between 0.3 and 1 D of hyperopia until 10 years of age.

Astigmatism

Astigmatism in neonates is more frequent and bears a different axis than that later in life. Utilizing a near-retinoscopy procedure without cycloplegia, Gwiazda and colleagues examined 521 children under 1 year of age. This method can be questioned because a number of assumptions were made in regard to the accommodative status of the subjects. Nonetheless, they found about half the children to have an astigmatism of 1 D or greater. The axis was as likely to be against the rule as with the rule. They longitudinally followed 29 children with significant astigmatic errors from 6 months to 6 years of age. The amount of astigmatism significantly decreased over that period. Of 16 children with against-the-rule astigmatism, only one had greater than 1 D remaining at 6 years of age. Of 8 with with-the-rule astigmatism, the astigmatism decreased or vanished in all cases.

Fulton and coworkers, utilizing retinoscopy following instillation of 1% cyclopentolate, examined 75 infants under 1 year of age. They reported 19% to have an astigmatism of greater than 1 D. Between 7 and 12 months of age, the astigmatism rate increased to about 25%. Of their astigmatic infants, 82% had an against-the-rule axis, and 18% had a with-the-rule axis. Only 5.7% of their subjects had an astigmatism exceeding 2 D. The use of cycloplegic agents may have made their data more accurate than previous noncycloplegic studies. The prevalence of against-the-rule astigmatism may only be true for white infants. Thorn and colleagues compared a group of white infants with Chinese infants. They confirmed the high incidence of against-the-rule astigmatism in whites but found the majority of Chinese infants to have a with-the-rule astigmatism. They felt that the difference could not be attributed to the difference in eyelid shape. In adults, the axis of astigmatism appears to be similar in the two groups.

Most studies have shown a decrease in the against-the-rule astigmatism so commonly found in infants with increasing age, especially...
after the age of 4. Without proof, this decrease is usually attributed to the effect of the pressure of the upper eyelid against the superior aspect of the cornea. This would induce a tendency toward with-the-rule astigmatism. Indirect support of this hypothesis comes from Robb who showed that hemangiomas of the eyelids can exert pressure on the cornea and cause an astigmatism with the positive axis pointing in the direction of the hemangioma. Bogan and associates have demonstrated that astigmatism may also be caused by dacryoceles and dermoid tumors as well.

**Refraction in Premature Infants**

Premature neonates have a different refractive status than do term infants. Shapiro and associates performed cycloplegic refractions on 236 children with a birth weight of 2,000 gm or less. In 22 eyes under 1 year of age, the mean refractive error was 1.5 D of hyperopia (± 0.8). The mean birth weight of 1,707 gm (range, 1,300 to 1,960) indicates that this population was not very premature. This may explain the lack of a significant difference from a population of term infants.

On the other hand, Dobson and colleagues reported the cycloplegic refractions of 146 infants at a mean postconception age of 34.2 weeks ± 2.9. These infants had normal retinal examination results. The mean refractive error was 0.55 D of myopia (± 2.8). The mean amount of astigmatic correction was about 1.6 D. Full-term infants were not found to be myopic (P < .001). Infants who were born more prematurely tended to have more myopia and greater astigmatism than infants who were born less prematurely. Of the 101 infants with greater than 1 D of astigmatism, 83% had against-the-rule, 10% had with-the-rule, and 7% had oblique axes. Nissenkorn and coworkers examined 155 premature infants weighing between 600 and 2,000 gm. They reported that the 113 infants without retinopathy of prematurity had an average of 1.50 D of myopia. Prematurity appears to be a more important cause of myopia than does low birth weight since it has been shown that full-term infants with intraterine growth retardation have refractive errors and visual acuities resembling those of normal birth weight infants.

**Myopia in Infants**

It must also be recognized that there are special circumstances, besides prematurity, that can cause myopia in neonates. Hoyt and colleagues reported eight children who developed unilateral axial myopia associated with neonatal eyelid closure. The eyelid closure resulted from various causes such as congenital third cranial nerve palsy, blepharoptosis from hemangioma or neuroma of the eyelid, and periorbital edema from obstetric trauma. The myopia ranged from 4.25 to 7.00 D and resulted primarily from an increase in the sagittal diameter of the posterior ocular segment as shown by ultrasonography.

Retinopathy of prematurity has been shown to be associated with myopia. In 42 premature infants with retinopathy of prematurity, Nissenkorn and colleagues reported an average of 4 D of myopia, with half of the children demonstrating between 0.25 and 15.5 D of myopia. The greater the birth weight of the infants with retinopathy of prematurity, the smaller the amount of myopia. The cause of the myopia appears to be multifactorial. Some cases seem to result from increased axial length, while others arise from increased lenticular power or a combination of both these factors. Increased corneal curvature may also contribute to the myopia.

**MARKERS OF GESTATIONAL AGE**

Although almost all aspects of the neonatal eye change with maturation, only two have been carefully correlated to gestational age in living neonates. This relationship has been exact enough for these parameters to be considered indicative of a neonate's postconceptual age by virtue of their degree of maturation.
TABLE 3-4.
Assessing Gestational Age by Dissolution of the Anterior Vascular Capsule of the Lens*

<table>
<thead>
<tr>
<th>Grade</th>
<th>Age, wk</th>
<th>Finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>27–28</td>
<td>Lens surface entirely covered by vessels</td>
</tr>
<tr>
<td>3</td>
<td>29–30</td>
<td>Central vessels begin to atrophy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Peripheral vessels thin</td>
</tr>
<tr>
<td>2</td>
<td>31–32</td>
<td>Central lens area more visible</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Peripheral vessels thin more</td>
</tr>
<tr>
<td>1</td>
<td>33–34</td>
<td>Only a few vessels remain at lens periphery</td>
</tr>
</tbody>
</table>


Tunica Vasculosa Lentis

At the 17-mm stage of embryogenesis, small buds from the annular vessel form vascular loops carrying mesodermal tissue onto the anterior surface of the lens even before development of the anterior chamber. By the 22-mm stage, the lamina iridopupillaris is a richly vascularized layer of mesodermal cells closely opposed to the anterior surface of the lens. The peripheral part thickens to form the embryonic iris. The central portion is much thinner, composed almost entirely of blood vessels. It is this plexus of blood vessels that constitutes the tunica vasculosa lentis (also known as the anterior vascular capsule of the lens). With time, this plexus further thins, only to eventually disappear.

In 1977, Hittner and colleagues reported that the degree of dissolution of the tunica vasculosa lentis could be closely correlated to gestational age. They examined the anterior vascular capsule of the lens on the first day of life with a direct ophthalmoscope after pupillary dilation with tropicamide, 0.5%, and phenylephrine, 2.5%. Their grading system can be found in Table 3-4 and Figure 3-8.

Subsequently, these investigators found