that learning and retention may be improved in the context of home environmental cues, since in each of many experiments, Rudy and Cheatle have simultaneously exposed all rats from a litter to the odor. Hence, all rats were experiencing the onset of illness not only in the presence of the paired odor, but also in the presence of an important home environmental cue, conspecifics. Experiment 3 compared the effects of conditioning pups in isolation and with conspecifics.

Albino rats ($N = 36$) were trained 2 days and tested 9 days after birth. Each pup was randomly assigned to one of four treatment groups (nine per group) arranged in a 2 by 2 factorial design [presence of conspecifics during training versus trained in isolation by illness (injection of 2 percent of body weight of a $0.15M$ solution of LiCl) versus no illness (saline)].

The conditioning procedure was the same as that of Rudy and Cheatle (*4*) except that each pup trained in isolation was exposed to the experimental treatments alone, in isolation from littermates. Otherwise the groups were identically treated. Each pup was tested individually over a 5-minute period for its preference between lemon or garlic odors (*21*) to determine the percentage of time spent over the lemon odor.

Pups given the lemon odor-illness pairing in the presence of conspecifics spent less time over the lemon odor than control pups given the lemon odor with saline (Fig. 2). This replicates the Rudy-Cheatle finding that 2-day-old pups can learn an association of odor with illness and retain it over a surprisingly long interval. However, of primary interest is the influence of isolating the pup during learning (Fig. 2). Pups given the odor-illness pairings in isolation spent as much time over the lemon odor as the controls did. Apparently, training the pup in isolation from littermates markedly disrupted learning or the subsequent retention of Pavlovian conditioning. Analysis of variance indicated a reliable interaction ($P < .05$) and main effect of training environment ($P < .01$). Although pups given the odor-illness pairing in isolation did not differ from their saline controls or saline controls experiencing the odor with conspecifics ($F < 1$), all these groups did differ reliably from the group given the odor-illness pairings in the presence of conspecifics ($P < .01$).

These experiments suggest that separation of the rat pup from familiar home environmental cues disrupts learning or retention. The early learning and retention reported by Kenny and Blass (*2*) may not necessarily reflect a general disposition of the organism to learn an appetitive response sooner than a shock-motivated response, but rather the importance of learning about the response contingencies in the presence of home environmental cues (in their case, the anesthetized adult female).

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Astigmatism in Infants

Abstract. *Compared with children of school age, infants show ten times the incidence and considerably greater amounts of clinically significant astigmatism. The amount begins to decrease in the second semester of life, and the incidence declines during the third year. These unanticipated results bear on both the etiology and the neural sequelae of astigmatism.*

In the course of refracting the eyes of 276 full-term infants between birth and 50 weeks of age by means of near-retinoscopy (*1*), we discovered that clinically significant astigmatism (*2, 3*) occurred in 45 percent of the sample (*4, 5*). This incidence is almost ten times that reported in children (*6*) and five times that reported in adults (*7*), which implies that much of the early astigmatism must be either reduced or eliminated in the course of development.

The unexpectedly high incidence is interesting in light of the inference that astigmatism has deleterious effects upon the development of the visual nervous system. The meridional amblyopia found in adult astigmats represents an optically uncorrectable loss of acuity, attributed to irreversible changes in the visual nervous system (*8*). These changes are, in turn, attributed to prolonged exposure to the blurred edges of images of contours of all orientations except the focused meridians. Furthermore, these central abnormalities in human observers have been said to parallel the changes in the incidence of orientationally selective neurons in the visual cortices of cats reared either with edges of only one orientation visible (*9*) or with strong astigmatic lenses (*10*). Finally, one wonders how the high incidence of astigmatism during human infancy bears on the timing of a sensitive period during which astigmatism may cause irreversible deficits in vision.

In this report we report the results of the initial refractions of the right eyes of 276 infants (*11*). In addition, we tracked the refractions of 28 of these infants who had shown 2 or more diopters of astigmatism at some time between 3 and 6 months of age. All of the infants were healthy and full term with birth weights ranging from 2381 to 4904 g for 141 males (median = 3487 g), and from 2410 to 5075 g for 135 females (median = 3289 g). The majority of the infants were solicited through letters mentioning tests of

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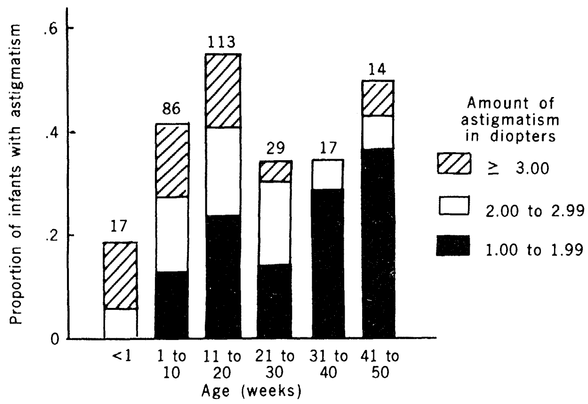

Fig. 1. Incidence of clinically significant astigmatism on initial refractions during the first year of life. The total number of infants refracted within an age group is given above each bar.

vision and sent to parents in the Boston area. Responses to the letters averaged 15 percent. Ninety-five percent of the infants were white, the remainder black and oriental.

During refraction, the infant was held on the parent's lap in a completely darkened room with the retinoscope 50 cm from the eyes. The low-intensity beam of light from the retinoscope was shone in the infant's eye. While the infant gazed at the light source, the vergence of the retinal reflection was neutralized through the use of a graded set of lenses mounted in a bar for quick manipulation. Attention to the light was enhanced by testing the infant during feeding. With older infants, sounds produced by the examiner helped maintain stable gaze for the duration necessary for measurements (*12*).

Most of the infants were first refracted between 0 and 20 weeks of age (*13*). The incidence of astigmatism is low in the youngest infants and reaches a peak in the 11- to 20-week group (Fig. 1). Astigmatism in the latter group is evenly divided between direct and inverse (*14*) (40 percent each) with 20 percent of the infants showing cylindrical power at an oblique axis. After 20 weeks of age, the overall incidence is lower and, in particular, the higher amounts of astigmatism ($\geq$ 2.0 diopters) are reduced so that in the 41- to 50-week group, most infants are nonastigmatic or have small amounts of astigmatism (< 2.0 diopters). The infants in the youngest age groups (0 to 20 weeks) tend to be either simple or compound myopic astigmats, those between 21 and 30 weeks tend to be mixed astigmats, and those in the older two age groups (31 to 50 weeks) tend to be either simple or compound hyperopic astigmats. Nonastigmatic infants show a similar trend toward hyperopia with increasing age (*15*).

Since the incidence of large amounts of astigmatism ($\geq$ 2.0 diopters) is so high in the first semester of life and then declines in the second semester, we tracked the refractions of the right eyes of 28 infants who had shown two or more diopters between 3 and 6 months of age. All of these infants had five or more refractions at periodic intervals within the first year of life. By 50 weeks of age, half had no astigmatism, one-fourth showed a reduction of one or more diopters, and one-fourth maintained the amount present at 3 to 6 months. No increases in the amount of astigmatism were seen. Refractive data from three infants, representing three typical patterns of development of astigmatism, are shown in Fig. 2.

The infant who maintained the astigmatism had between 2.5 and 4.0 diopters throughout the first year of life. The amount was reduced to 2.0 diopters in the second year, and by 2½ years, the astigmatism was no longer present. The astigmatism measured in the infant who showed a reduction at 1 year has remained stable to 21 months. Of the ten infants (more than one-third of our sample) who had two or more diopters at 3 to 6 months and who still had astigmatism at 1 year, only three lost their astigmatism in the second year of life. We are now tracking their refraction into the third year of life. The infant who lost her astigmatism at 1 year of age has remained free of it into her second and third years of life. Of 13 infants who showed no astigmatism at 1 year, only one showed its reappearance during the second year of life. It therefore seems that once the astigmatism disappears, it rarely reappears.

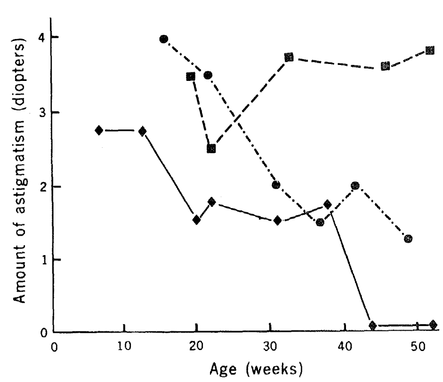

Fig. 2. Three typical cases of change in astigmatism during the first year of life. One infant maintained the astigmatism (■), in a second it was reduced (●), and the third lost the astigmatism (◆).

The factors responsible for the high incidence and lability of astigmatism in infancy must be relevant to its etiology, although that remains obscure at present (*3, 16*). A sample of the astigmatic infants tested psychophysically by a visual preference procedure revealed the effects of astigmatism by reduction of acuity in the blurred meridians (*4, 17*). However, optical correction for the cylindrical error brought acuity back to the age norm for this procedure (*18*) and hence revealed no evidence for meridional amblyopia up to the age of 1 year. Consequently, within the limitations of the sample, it appears that infantile astigmatism does not contribute to meridional amblyopia. We are now tracking the refraction and the meridional variations in acuity of some of our infant astigmats into their second and third years of life. Preliminary results indicate that the incidence of astigmatism does not decline to adult levels until after the age of 2 years. Moreover, the earliest that we have detected meridional amblyopia is just prior to the end of the third year of life, which suggests that the sensitive period for the development of meridional amblyopia may begin later than that for the development of amblyopia resulting from binocular anomalies (*19*). Further data from astigmatic children should specify the relation between early astigmatism and the development of meridional amblyopia.

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References and Notes

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12. Repeated refractive measures taken on the same day on 13 infants rarely deviated by more than ± 0.5 diopters.
13. Letters soliciting infants for testing were sent to new parents, who usually responded when the infants were between 2 and 5 months of age. Consequently, our sample of first refractions is largest in this age range.
14. In direct (with-the-rule) astigmatism, the meridian of greatest refractive power of the eye is within 30° of the vertical. In inverse (against-the-rule) astigmatism, the meridian of greatest refractive power of the eye is within 30° of the horizontal.
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Infant Astigmatism Measured by Photorefraction

Abstract. *Photorefraction of a sample of 93 infants of ages 1 day to 12 months showed that 63 percent of the subjects had astigmatism of 0.75 diopter or greater, and 12 percent greater than 2 diopters. Seventy percent of these astigmatisms were in the horizontal-vertical meridians. By comparison, only 8 percent of a sample of 26 adults tested by the same method showed astigmatism (all 0.75 to 1 diopter). The high incidence of infant astigmatism has implications for critical periods in human visual development and for infant acuity.*

Photorefraction (*1*) is a photographic method for estimating the instantaneous refractive error of a subject's eyes, relative to the distance of the camera used. It requires no subjective report from subjects, nor their cooperation except in the brief fixation of a target, and so may be used with freely accommodating infants of all ages.

Figure 1 shows an example of the photographs on which measurements are made. The stars centered on each eye are produced by the retinal images of a point flash source centered in the camera lens and imaged on the film by a set of four cylindrical lens segments around the source. From the lengths of the star arms the defocus of each image in two orthogonal directions may be derived (*2*). The technique is therefore well suited for measuring astigmatic errors.

We now report a high incidence of astigmatism in an unselected sample of infants examined as part of an extensive photorefractive study of accommodative and refractive errors of infants from birth to 1 year of age (*3*). This finding is of interest because the first year of life is thought to be at least part of the critical period for human visual development (*4*), during which time the visual system may be vulnerable to deficient input; differences in image quality in different meridians have been suggested to have a developmental effect (*5*). If so, either infant astigmatism must result in meridional variations in acuity in later life, or neural plasticity must outlast the period of infant astigmatism (which we find to be at least 1 year).

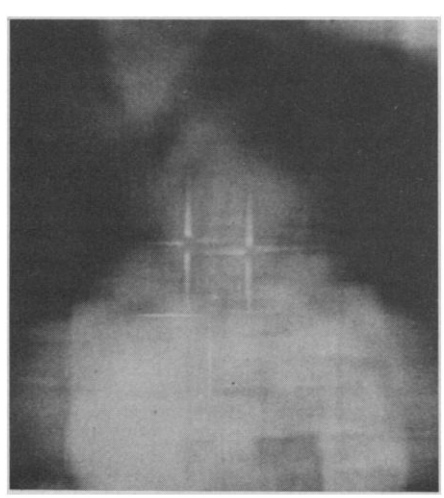

Fig. 1. Photorefractive picture of infant with a 2.0-diopter astigmatism. The horizontal meridians of both eyes are relatively well focused while the vertical meridians are severely defocused.

The method used differed from the original method of photorefraction (*1*) in three ways. (i) An electronic flash rather than a tungsten light source was used. (ii) The photographs were taken on color transparency film (High Speed Ektachrome) rather than black-and-white film; this helped interpretation of the images in that the retinal reflexes were more readily distinguished from the background of the infant's face, and the white light from the retinal image of the source could be separated from the redder, more broadly distributed scattered light. (iii) The camera-to-subject distance was varied.

Infants in five age groups were studied: newborns (0 to 9 days), 1 month (4 to 7 weeks), 2 to 3 months, 6 to 8 months, and 9 to 12 months. Newborn infants from the postnatal wards of the (Cambridge, England) Maternity Hospital were tested when alert, usually shortly before or after feeding. The older groups were volunteered by their parents in response to recruiting leaflets distributed in well baby clinics in the Cambridge area. No infant in any group was more than 14 days premature.

Each infant was photorefracted while seated on the mother's or experimenter's knee at 150 and 75 cm from the camera. The estimates of astigmatism are principally based on photographs taken with the infant fixating at the camera distance. Between four and six photographs were taken at each of the two camera distances for each infant. This was achieved by using the photographer as the visual target; he or she attracted the infant's attention by calling, shaking brightly colored rattles, peekaboos, and other attention-seeking activity. The camera was operated when the infant was judged to be fixating the operator's face close to the camera. In this way the camera was always within 5° of the optical axis of the eyes at the moment of exposure, so off-axis errors of refraction (*6*) were avoided. Photographs were also taken with

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