with probable increased rates of cellular cation exchange.

The curious phenomenon of the latent period between exposure and clinical manifestation of injury remains to be explained. The mobilization of epithelial cellular glycogen and increase in cellular hydration are also rather slow following UV exposure. This may indicate that the effects of the radiation on the pathways responsible for energy transformation are secondary to cellular structural and enzymatic protein changes. Possibly, as in other cells, damage to structural membranes occurs through peroxide formation, with subsequent lipid peroxidation and cellular permeability changes.[8, 9] The delay in onset could also be explained by an effect of UV on RNA and DNA, with delayed transcription and translation to protein synthesis or frank inactivation by nucleotide dimerization or crosslinkage.[10, 11]

Regardless of the molecular mechanism involved, the effects noted in experimental UV keratitis can be accounted for by changes observed in the epithelial layer. These differences in the time course of the response to UV injury and to other forms of epithelial stress[3] imply that epithelial response to insult may differ depending on the form of stress applied.

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Key words: corneal epithelium, ultraviolet radiation, photokeratitis

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Increased saccadic latencies in amblyopic eyes.

Kenneth J. Ciuffreda,* Robert V. Kenyon, and Lawrence Stark.

Increased saccadic latencies were measured in the amblyopic eyes of subjects having amblyopia without strabismus, constant strabismus amblyopia, and intermittent strabismus. The subjects tracked a small, bright spot of light moving with random, horizontal step displacements of 0.25 to 8.5 degrees over the central retina. Normal saccadic latencies were generally found during monocular tracking with the nonamblyopic eye as well as during binocular tracking. Studies of eye-hand reaction time in amblyopic eyes have shown delays to occur over the central retina; our new finding establishes this for saccadic initiation. Normal trajectories found for all tracking saccades, normal saccadic latencies measured when the nonamblyopic eye was utilized for tracking, and synchronous movement of the eyes under all test conditions point to a sensory rather than motor basis underlying these delays. Our results are interpreted in terms of a processing delay in the sensory pathways leading from the central region of the amblyopic eye to those centers involved in saccadic initiation.

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Table I. Clinical data and saccadic latencies of subjects

Subject	*Age*	*Prescription*	*Visual acuity*	*Vergence abnormality*
Constant strabismus amblyopia:				
1	25	LE + 2.00 = −0.25 × 130	20/25	1-2$^{\Delta}$ left ET
		RE + 2.25	20/15	
2	23	LE + 3.75 = −0.50 × 165	20/30	18$^{\Delta}$ left ET
		RE + 0.50	20/15	
3	26	LE − 10.25 = −1.50 × 150	20/110	18$^{\Delta}$ left XT
		RE − 4.50 = −1.25 × 25	20/20	
4	15	LE − 1.50	20/122	10$^{\Delta}$ left ET
		RE − 1.75	20/20	1$^{\Delta}$ left HT
5	32	LE + 4.00	20/277	4$^{\Delta}$ left ET
		RE plano	20/20	
6	33	LE + 0.75 = −0.50 × 40	20/630	5-6$^{\Delta}$ left ET
		RE + 0.25 = −0.50 × 180	20/10	2$^{\Delta}$ left HT
Amblyopia without strabismus:				
1	24	LE + 0.75 = −2.00 × 90	20/38	None
		RE pl = −0.50 × 19	20/20	
2	25	LE − 2.50 = −1.25 × 72	20/25	None
		RE − 5.00 = −0.75 × 5	20/40	
3	19	LE + 5.00	20/110	None
		RE + 3.00	20/15	
Intermittent strabismus with and without amblyopia:				
1	22	LE − 0.75 = −0.25 × 148	20/20	18Δ alternating XT
		RE − 3.00 = −0.25 × 100	20/20	12$^{\Delta}$ HT
2	31	LE − 5.00	20/20	15$^{\Delta}$ left XT
		RE − 4.50 = −0.75 × 20	20/20	
3	32	LE + 0.75 = −1.75 × 180	20/20	40-50$^{\Delta}$ right
		RE + 1.25 = −3.50 × 5	20/23	XT
4	25	LE plano	20/32	10$^{\Delta}$ left XT
		RE − 1.00	20/16	20$^{\Delta}$ left HT

*Mean ± 1 S.D.; numbers in parentheses indicate sample size.
†Indicates a statistically significant difference (t test; $p < 0.05$) between the normal eye and the amblyopic or nondominant eye.

The function of the saccadic eye movement system is foveation of peripheral retinal stimuli. One parameter of the saccadic system, saccadic latency, has been extensively studied in normal human subjects. Measures of saccadic latency form a distribution function having a mean of 200 msec with a standard deviation of 30 msec.[1-2]

Few investigations of saccadic latency have been conducted in subjects having amblyopia and strabismus. Mackensen,[3] using peripheral stimuli (~15 degrees), found only a slight increase (250 vs. 225 msec) in saccadic latencies of amblyopic eyes; however, notable latency differences in several subjects were minimized by combining the data from all subjects. He theorized that the peripheral retina was little, if at all, affected in amblyopia and turned to eye-hand reaction time measures to study the central retina; he now found dramatic differences in reaction time between the amblyopic and normal eye (325 vs. 225 msec), as one might expect from the central vision loss in amblyopia. Mackensen's eye-hand reaction time study was confimed and expanded by von Noorden.[4] Schor[5] found normal saccadic latencies when predictable step stimuli were presented to the central retina of the amblyopic eye in subjects having constant-strabismus amblyopia.

The purpose of the present investigation was to measure saccadic latencies, under monocular and binocular tracking conditions in response to random horizontal step displacements, in subjects having amblyopia without strabismus, constant-strabismus amblyopia, and intermittent strabismus with or without mild amblyopia, to determine whether saccadic latencies are increased over the central retina.

Methods. The photoelectric method was utilized to record horizontal eye position.[2] The bandwidth of the entire recording system was 75 Hz (−3 db). A chinrest and headrest, usually in

Eccentric fixation	*Saccadic latency** *(msec)*
½$^{\Delta}$ nasal LE	202 ± 27 (24) 208 ± 35 (15)
1$^{\Delta}$ nasal LE	212 ± 41 (30) 198 ± 34 (26)
1$^{\Delta}$ nasal and superior LE	254 ± 67 (25) 277 ± 66 (14)
2.5$^{\Delta}$ nasal and 2$^{\Delta}$ superior LE	483 ± 119† (6) 250 ± 56 (14)
16$^{\Delta}$ nasal and 4$^{\Delta}$ superior LE	427 ± 56† (11); 359 ± 70† (16) 223 ± 27 (17); 202 ± 39 (19)
2.5-3.5$^{\Delta}$ nasal and 3-4$^{\Delta}$ superior LE	290 ± 59† (8); 315 ± 71 (11) 213 ± 33 (8)
2$^{\Delta}$ nasal and 2$^{\Delta}$ inferior LE	212 ± 36† (21) 189 ± 26 (16)
2$^{\Delta}$ nasal and 2$^{\Delta}$ inferior RE	233 ± 29 (15) 229 ± 26 (39)
2$^{\Delta}$ temporal LE	283 ± 61† (35) 210 ± 27 (21)
Slight unsteady, central LE, RE	246 ± 46 (16) 224 ± 32 (14)
Central, steady LE, RE	296 ± 98 (20) 351 ± 97 (8)
1$^{\Delta}$ nasal and superior RE	259 ± 36 (10) 342 ± 67† (18); 260 ± 44 (23)
½$^{\Delta}$ nasal LE	254 ± 38 (17) 231 ± 32 (19)

conjunction with a bite bar covered with dental impression material, was used to stabilize the head.

A minicomputer (PDP-8/I) was used to generate horizontal random step displacements (0.25 to 8.5 degrees) of a small (4 to 8 min arc) spot of light on a display monitor placed either 57 or 91 cm away on the subject's midline. Target luminance was always maintained greater than 1 log unit above screen luminance.

Saccadic latencies, for both monocular and binocular tracking, were measured directly from the eye position traces on the strip chart paper. Paper speed ranged from 25 to 100 mm/sec; however, for the majority of measurements, it was 50 mm/sec, thereby assuring a sensitivity of about ±5 msec.

In the graphical displays of saccadic latency vs. percent response, bin widths of 50 msec were selected to assure an adequate number of samples per bin; this was especially important when samples were small.

The thirteen subjects were obtained from the general refraction clinic and/or orthoptics clinic at the School of Optometry. All had a thorough vision examination and were free of ocular disease. Ages ranged from 15 to 33 years, with a mean age of 25.5. Subjects from three diagnostic groups were tested: (1) constant strabismus amblyopia, (2) amblyopia without strabismus, and (3) intermittent strabismus with or without mild amblyopia. The spectacle or contact lens prescription was worn during all testing. See Table I for clinical data of the subjects.

Results. Increased saccadic latencies were observed in amblyopic eyes in six of 11 subjects having amblyopia with or without strabismus. Significant differences in saccadic latencies between dominant and nondominant eyes in our two subjects having intermittent strabismus without amblyopia were not observed (see Table I). The occurrence of increased saccadic latencies did not appear to be related to target amplitude or direction or to the initial starting point in the visual field. In general, when the primary saccade executed by the amblyopic eye exhibited an increased latency, the secondary corrective saccade(s) had normal latency. At times, the initial saccade was either slightly hypometric or hypermetric, although on occasion marked hypermetria was observed. Inaccurate saccades were most frequently observed in highly amblyopic eyes and will be the subject of a future communication (Ciuffreda, Kenyon, and Stark, in preparation).

Distribution curves of saccadic latency for monocular and binocular tracking are shown in Fig. 1 (top) for a subject having amblyopia without strabismus. A large, significant shift in the saccadic latency distribution is demonstrated in the amblyopic eye. Of interest is the dominance of the normal eye in determining saccadic latency during binocular tracking. Two of three subjects having amblyopia without strabismus exhibited significant increases in mean saccadic latency in the amblyopic eye; one did not. The triangles in Fig. 2 represent these three subjects.

Distribution curves of saccadic latency for monocular and binocular tracking are shown in Fig. 1 (middle) for a subject having constant strabismus amblyopia. As was true for amblyopic subjects without strabismus, there was a shift in the amblyopic eye distribution, indicating a preponderence of increased saccadic latencies as well as a similarity in the latency distributions between monocular tracking with the normal eye and binocular tracking. Three of six subjects in this diagnostic group, having the highest degrees of amblyopia, exhibited increased saccadic latencies

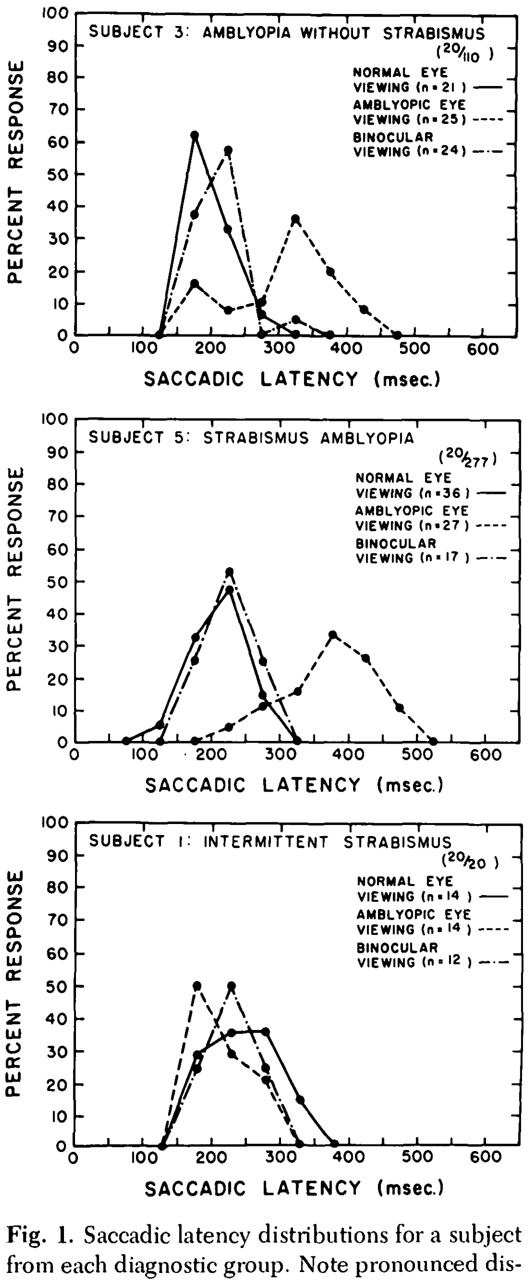

Fig. 1. Saccadic latency distributions for a subject from each diagnostic group. Note pronounced distribution shift toward longer saccadic latencies in amblyopic eyes. Latency distributions are similar for all conditions in subject having intermittent strabismus without amblyopia; in this subject, "amblyopic eye" refers to the nondominant eye.

in the amblyopic eye; the remaining three subjects from this group, having the lowest degrees of amblyopia, did not exhibit this phenomenon. It is interesting that Subject 3 (Table I), having high amblyopia (20/110), did not exhibit increased saccadic latencies with the amblyopic eye. Perhaps the effects of amblyopia in exotropia are different from those in esotropia, at least in terms of producing visual information processing delays. Further saccadic testing of constant exotropes will be required before this question can be resolved. The circles in Fig. 2 represent these six subjects.

Distribution curves of saccadic latency for monocular and binocular tracking are shown in Fig. 1 (bottom) for a subject having intermittent strabismus without amblyopia. The latency distributions for all three tracking conditions were similar. Significant differences in saccadic latencies between the dominant and nondominant eyes in subjects having intermittent strabismus only were not found; however, in our two subjects having intermittent strabismus with mild amblyopia, there was a tendency for increased saccadic latencies to be found in the amblyopic eye (see Table I). In Fig. 2, the open squares represent two subjects having intermittent strabismus only, and the filled squares represent two subjects having intermittent strabismus with mild amblyopia.

Similar trends were found for the combined data in each diagnostic group. For subjects having constant strabismus amblyopia, there was a significant ($p < 0.01$) difference between the amblyopic eye (280 ± 99 msec) and the normal eye (223 ± 49 msec). A significant difference ($p < 0.01$) was also found between the amblyopic eye (250 ± 64 msec) and the normal eye (210 ± 32 msec) for subjects having amblyopia without strabismus. However, for subjects having intermittent strabismus, the difference between the mildly amblyopic or nondominant eye (276 ± 73 msec) and the normal or dominant eye (259 ± 64 msec) was not significant ($p > 0.10$).

Discussion. The results of this study demonstrated that a necessary condition for the observance of increased saccadic latencies was the presence of amblyopia; the occurrence of strabismus was not essential. Subjects having amblyopia, with or without strabismus, exhibited increased latencies in the amblyopic eye; it was most frequently and clearly observed in subjects with higher degrees of amblyopia (Fig. 2), although a direct relationship between degree of amblyopia and increase in saccadic latency was not found.

Our new finding of increased saccadic latencies in amblyopic eyes suggests a slowing in the sensory pathways of processing of visual information, over the central retina, subsequently utilized by the oculomotor system in the generation of saccadic movements. It is interesting that the increase in visual evoked response latency averaged only 10 to 25 msec in a high amblyope (20/200)[6]; thus the larger increase in mean saccadic latency observed in the highly amblyopic eyes of our sub-

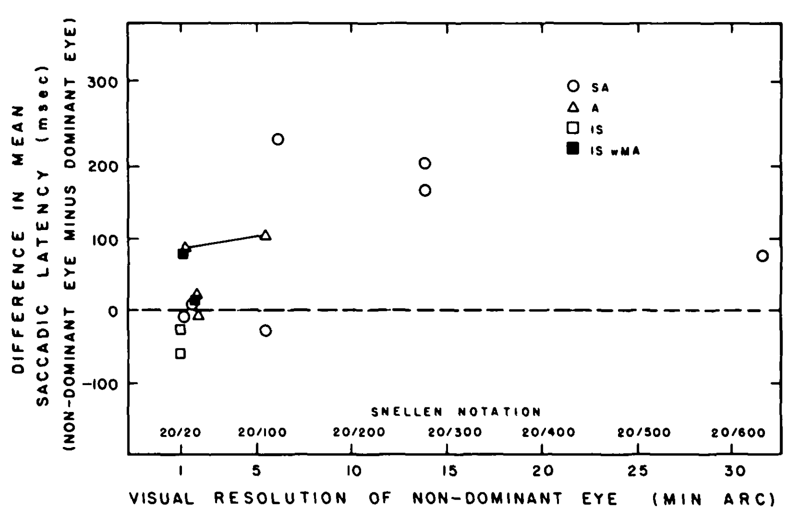

Fig. 2. Difference in mean saccadic latency between nondominant and dominant eye of each subject as a function of visual resolution in the nondominant eye. Most values for subjects having amblyopia lie above dotted zero line, indicating increased saccadic latencies in amglyopic eyes. That increased saccadic latencies persisted in the amblyopic eye following treatment which improved visual acuity is indicated by line connecting triangles representing Subject 3 having amblyopia without strabismus. *SA*, Constant strabismus amblyopia; *A*, amblyopia without strabismus; *IS*, intermittent strabismus without amblyopia; *ISwMA*, intermittent strabismus with mild amblyopia.

jects suggests two possibilities: (1) that a major portion of the visual information processing delay occurred after the site of generation of the visual evoked response or (2) that a pathway circumventing the primary visual cortex, which was even more sensitive than the visual cortex to the effects of amblyopia in terms of latency, was involved. Several arguments can be developed against motor involvement in these subjects. First, synchronous movement of the eyes was found under all conditions. Second, since saccadic latencies during binocular tracking and monocular tracking with the nonamblyopic eye were within normal limits, the motor controller (central nervous system processes above the lower motoneuron level) and plant (globe and eye muscle dynamics) appeared to be unaffected. Third, normal saccadic trajectories were found during all three tracking conditions. The similarity between the saccadic latency distributions for monocular tracking with the nonamblyopic eye and binocular tracking suggests that during binocular tracking, there was no interaction between the normal and amblyopic eye; dominance of the normal eye prohibited this from occurring. Sensory information from the dominant eye alone appeared to be utilized for the initiation of saccadic eye movements to targets of interest within the central visual field during binocular viewing.

Mackensen[3] found only a small increase in mean saccadic latency in amblyopic eyes, whereas Schor[5] found normal mean saccadic latencies. Can we account for the differences in results between these studies and our own? First, Mackensen used peripheral retinal stimuli (~15 degrees); step displacements were never greater than 8.5 degrees in the present study. Second, Mackensen grouped his data according to percent response in the normal and amblyopic eye of his subjects manifesting a wide range of amblyopias. By grouping the data, he masked the effects of subjects with high amblyopia and increased latencies by absence of effects in those subjects having low amblyopia and normal latencies. In Mackensen's table showing the range of saccadic latencies for individual subjects, a few subjects in several acuity groupings exhibited a wide range of latencies, indicating the presence of some increased saccadic latencies in these subjects. Schor's lack of finding may be accounted for by the stimulus conditions; step displacements with constant frequency and amplitude were employed, i.e., predictable inputs. Subjects could have predicted stimulus changes, which would reduce mean saccadic latency value.

It is interesting to speculate on a possible neurophysiological mechanism responsible for producing increased saccadic latencies in amblyopic eyes. The proposition has recently been advanced that the superior colliculus may be intimately involved in coding information regarding the location of objects in space relative to the fovea, the "foveation hypothesis,"[7] and in the initiation of saccadic eye movements. Cells have been found in the superior colliculus which appear to be related to readiness to make an eye movement[8]; it was suggested that these cells may be involved in early information processing for saccadic initiation. Increased saccadic latencies, approximately 150 to 300 msec greater than normal, have been measured in monkeys following focal lesions in the superior colliculus; accuracy of movement was not affected, and speed of movement was minimally affected.[9] In kittens reared with unilateral strabismus (and presumably amblyopia), the ability of the strabismic eye to drive collicular cells was decreased.[10] From these findings it seems reasonable to suggest that in man, as a result of abnormal visual experience (constant suppression and/or form deprivation producing amblyopia), deprivation of certain cells in the sensory pathway leading from the amblyopic eye directly to the superior colliculus or via the visual cortex, or within the superior colliculus itself, may be adversely affected, producing delays in information processing which result in abnormally long saccadic latencies.

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Key words: amblyopia, strabismus, saccadic latency, eye movements, temporal processing

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Television pupillometry via digital time processing. J. JAMES SALADIN.

A new modification of the dynamic, infrared recording pupillometer of the closed-circuit television type has been developed, utilizing recent developments in high speed electronic counters. The pupillometer is simple and reliable in operation, has excellent response characteristics, and can be used over a broad range of research and clinical applications.

Closed-circuit television systems have been used for over 20 years in the construction of devices to continuously monitor changes in the pupillary diameter. Lowenstein and Loewenfeld[1] reported an instrument design in which the pupil-iris region was scanned with a slit of infrared light which moved horizontally across the eye and worked its way downward on each successive sweep. A phototube measured the light level reflected from the eye, recording little light when the pupillary area was being scanned and more light when another area was being scanned. This created square "white-black" impulses from the phototube which could be converted into a triangular pulse with the base (time) of the triangle proportional to the height (voltage) of the original square impulse. The height of the triangular pulse was then proportional to the base and could be monitored with a diode peak detector which yielded a variable D. C. potential proportional to the largest pupillary diameter on each downward excursion of the slit. Asano et al.[2] developed an electronic scanner as an alternative to the mechano-optical scanner used by Lowenstein and Loewenfeld and used video clippers to detect the pupillary edge. Green and Maaseidvaag[3] described a pupillometer which also detected the edge of the pupil from the signal voltage of the

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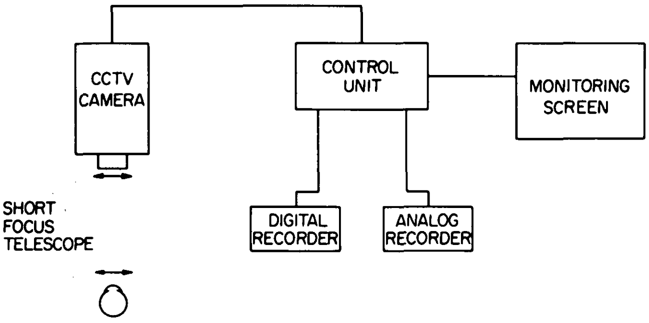

Fig. 1. Pupil is viewed through a short-focus telescope by the CCTV Model HV-16S; Hitachi, Ltd., Tokyo, Japan). The electronic signal is sent to the control unit (designed on special order by G. A. Galbreath and Associates, Columbus, Ohio). The control unit contains the clipping level mechanism, devices to control the height, width, and placement of the measurement rectangle, and a calibration circuit to provide the proper signals to the recording devices. The calibration circuit has a three digit LED readout good to 0.01 mm. The digital recorder (Model 5055A; Hewelett-Packard Co., Palo Alto, Calif.) prints out the pupillary diameter either 5 or 10 times/sec upon selection, with a reading to the nearest 0.01 mm. The analog recorder (Brush Model 260; Gould, Inc., Control & Systems Div., Cleveland, Ohio) simultaneously records the output. The monitoring screen (RLC 14; Conrac Corp., Covina, Calif.) enables the operator to set the placement of the measurement rectangle and to visually monitor the proper setting of the clipping level. The stimulus presentation is recorded on a separate channel of the analog recorder and by a characteristic mark placed on the digital recording. The pupil is illuminated with a microscope stage lamp (Model 359; American Optical Corp., Buffalo, N. Y.) covered with a deep red filter (No. 7-69; Corning Glass Works, Corning, N. Y.).

videcon tube and counted the number of scan lines crossing the pupil. This technique yielded the vertical pupillary diameter as seen by the television camera. O'Neill and Stark[4] used a similar design in the pupillometer included in their triple-function ocular monitor. The pupillometer reported in the present paper also detects the edge of the pupil from the video signal but makes a horizontal measurement of the pupil by measuring the time of the scans across the pupil at the different levels, storing the largest absolute value and then computing from this maximum value the diameter of the pupil.

Instrumentation

As can be seen from Fig. 1, the pupillometer uses a short-focus telescope to bring infrared light reflected from the subject's iris to the closed-circuit TV (CCTV) camera. The light pattern is converted by the CCTV camera into an electronic signal which is sent to the control unit and thence to the monitoring screen where the pupil is displayed. The control unit has adjustments which enable the operator to move a measurement rectangle about on the face of the monitoring screen. It is within the measurement rectangle that the pupil diameter is measured (Fig. 2, *A*).

The electronics of the control unit enhance the video output to full-black or full-white within the measurement rectangle according to the clipping level set by the operator. The clipping level is the point on the spatial-contrast curve at which the division between black and white is made. The operator sets the clipping level by looking at the monitor and adjusting a control knob so that the two edges of the black area are at the edges of the pupil (Fig. 2, *B*). If the clipping level is set correctly, the electronics will then "lock-onto" the edges of selected contrast (the edges of the pupil) within the measurement rectangle. The measurement rectangle (Fig. 2, *A*) can be adjusted for size and position. Theoretically, it is desirable to make the measurement rectangle as small as possible and still contain the pupillary diameter because this reduces the probability of the electronics locking onto a spurious edge having a spatial-contrast similar to that of the pupillary edge. Practically speaking, however, this did not prove to be a problem, and the measurement rectangle was always made large enough to cover almost the entire pupil. This had the advantage that the pupillary diameter remained in the measurement rectangle when a small eye movement occurred.

The electronic action of the pupillometer can be understood in the following step-by-step manner.

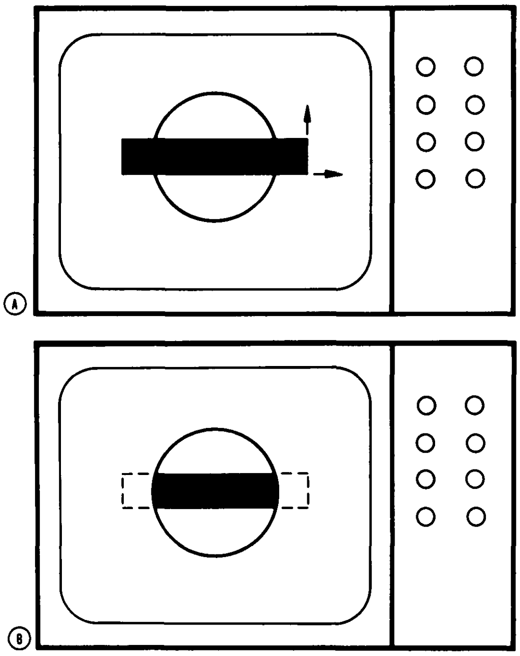

Fig. 2. A, Television monitor is shown with a properly magnified pupil and appropriately positioned measurement rectangle but without the appropriate clipping level adjustment made to obtain a lock onto the edges of the pupil. **B,** Measurement rectangle is now properly positioned, and the clipping level adjustment has been appropriately made. The operator can visually monitor the success of the lock onto the pupillary edge by noting that the edges of the dark area are on and moving with the edges of the pupil. The limits of the measurement rectangle are still faintly visible. The correct diameter will be read no matter what the eye movement as long as the diameter is within the measurement rectangle.

As the electron beam begins its sweep across and down the display screen, it eventually begins to cross the area of the measurement rectangle. It is only within this area that the electronic circuits monitor the video signal. When the electronically created black-level edge (as previously set by the clipping level adjustment) is detected, the information is sent to the measurement system that a distance is to be measured. That distance is measured by a counter which records the time required for the cathode ray beam to move across the electronically "black" pupil area. This time measurement is then stored for comparison to the time measurement made on the next sweep of the electron beam. A simple logic system selects the measurement with the longest time which must indicate the largest chord or the diameter. The counters are of a sufficient speed to allow 0.01 mm changes in pupil diameter to be detected. Because the sweep speed of the electron gun is known, the distance can then be computed from the time-distance-rate relationship. The beam makes one trip down the raster each one-sixtieth of a second, permitting the diameter of the pupil to be measured 60 times/sec. The essential part of the electronics is the counter; it is the recent development of integrated-circuit high-speed electronic counters and oscillators which has made possible the development of this pupillometer.

Small horizontal eye movements have little effect on the measurement while the diameter of the pupil remains in the measurement rectangle. The same comment can be made for vertical eye movements. Large (> ~ 15 degrees) horizontal eye movements affect the measurement of pupil diameter because of the foreshortening of the pupil as seen by the CCTV camera.

The response characteristics of the pupillometer are quite adequate for the measurement of changes in pupillary diameter.[5, 6] The pupillometer–analog recorder system will follow a sinusoidally moving border up to a frequency of 10 Hz with only a 3% decrease in amplitude of the sinusoidal output. Internal noise (drift) to a test target after a 2 min warm-up is less than 0.01 mm over a period of minutes. The chief limitation on the precision of the pupillary measurement seems to be the steadiness of the subject's head and eye. For that reason, a head and chin rest are used.

The pupillometer has proved remarkably reliable and easy to use for measuring the pupillary responses to variable accommodative and photic stimuli. The measurements are routinely made through the subject's spectacle lenses. Spectacle corrections of 6.00 D. hyperopia and 8.00 D myopia have caused only a problem in calibration due to the magnification effects of the lenses. No more than 30 sec are required to find and lock onto a human pupil, no matter what the iris coloration. Blinks are easily recognized on the record, and the electronics regain the lock onto the pupillary margins in the next sweep of the electron beam after the blink.

The pupillometer has been in regular use for the past year and has proved to be very reliable in its operation. The versatility of the pupillometer was shown in an experiment in which guinea pigs were used as subjects.[7] It is used as a regular member of a diagnostic battery for problems of the near triad in a manner similar to that described by Stark et al.[8] We have experienced no problems in data attainment attributable to the pupillometer.

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Key words: pupil, pupillometry, pupillography

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Accommodation and chromatic aberration in young children.

J. G. Sivak and C. W. Bobier.

Retinoscopy through colored filters (chromoretinoscopy) was used to determine the portion of the chromatic aberration interval in focus when young children (2 to 6 years of age) fixate at far and near. The results indicate that the children may be divided into three distinct groups. In the youngest group there is haphazard focusing within the chromatic aberration interval at far and near. The middle group shows selective focusing of the red end of the chromatic interval at both far and near. Children in the oldest group focus the red end when fixating at far and the green end when fixating at near, thereby sparing accommodation. These results suggest that the eye's use of the chromatic aberration interval to spare accommodation, as found in previous studies carried out on adults, is learned by about the fourth year of a child's life.

Investigators have measured axial chromatic aberration in the adult eye when it fixates at varying distances. They reported that the wavelength in focus on the retina varied from the long to the short end of the spectrum when fixation was changed progressively from far to near.[1-3] Millodot and Bobier[4] monitored accommodation while a near fixation target was illuminated by different wavelengths and found that accommodation tended to remain fixed throughout. These results indicate that when the eye takes up fixation, it

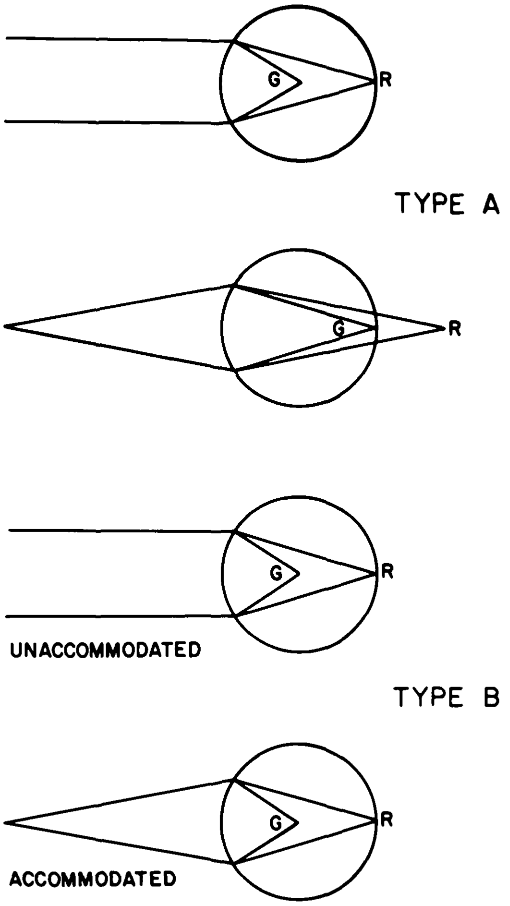

Fig. 1. Schematic drawings indicating the focal positions of red *(R)* and green *(G)* wavelengths when subject is fixating at far and near. *Type A,* Adults and children over 4 years of age; *Type B,* children 3 to 4 years of age.

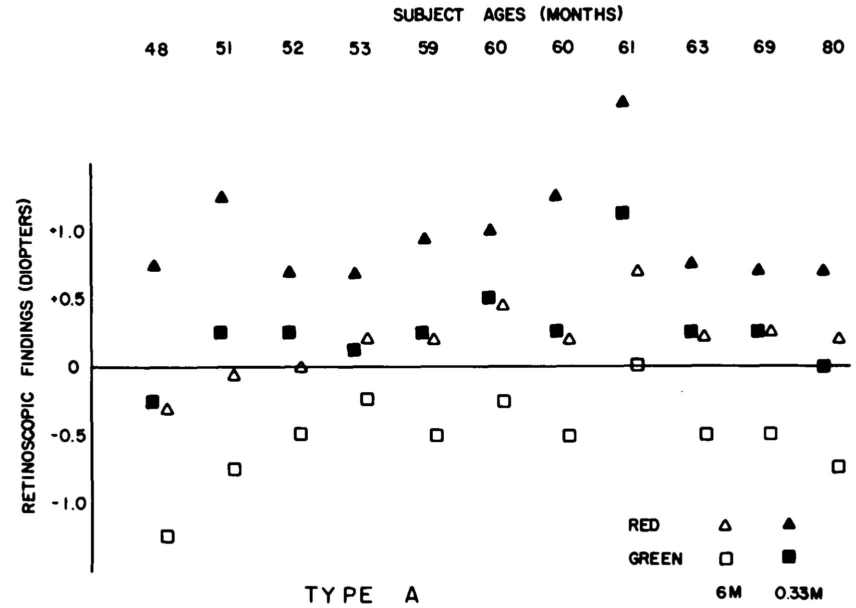

Fig. 2. Retinoscopic measurements through red and green filters for 0.33 and 6 m target distances for subjects from 48 to 80 months of age. In these subjects (Type A) red is in focus on the retina for distance fixation and green for near.

Table I. Response types resulting from the performance of chromoretinoscopy in young children fixating at far and near

Type	No.	Age range (mo.)	Sample findings (diopters)						
			Fixation at 6 m			Fixation at 0.33 m			
			No filter	Red*	Green*	No filter	Red	Green	
A	11	48-80	+0.25	+0.25	−0.75	+0.75	+0.75	0.00	(Subj. B. K.)
B	9	40-61	0.00	0.00	−0.75	0.00	0.00	−0.50	(Subj. E. V.)
C	6	31-45	+0.25	+0.50	−0.50	+0.12	+0.62	−0.50	(Subj. A. S.)

*The red and green filters used have dominant wavelengths of 617 and 530 nm, respectively.

tends to use the retinal stimulus without adjustments in its focusing as long as some cross-section of the chromatic aberration interval lies in the plane of the retina. The efficient use of the chromatic interval in focusing in this manner has in the past been interpreted as a failure of accommodation (lag of accommodation) to fully compensate for changes in target distance.[3-5] This indiscriminate use of wavelength adds flexibility to the system and spares accommodation.

Bobier and Sivak[5] demonstrated that it is possible to measure the eye's chromatic aberration and determine the cross-section of the chromatic aberration interval in focus for a given fixation distance by performing retinoscopy through colored filters (chromoretinoscopy). Their results agreed with the above studies, i.e., that the adult eye uses its chromatic aberration in such a way as to spare accommodation. It has been suggested that this sparing of accommodation is learned.[3]

In the study which follows, chromoretinoscopy was carried out in young children at far and near to determine whether the young eye uses its chromatic aberration to spare accommodation.

Methods. Static and dynamic retinoscopy were carried out on 26 children between the ages of 2 and 6 years. Static measurements were made with trial lenses while each child fixated a Snellen chart at 6 m. The chart consisted of black symbols on a white background pointing in various directions.

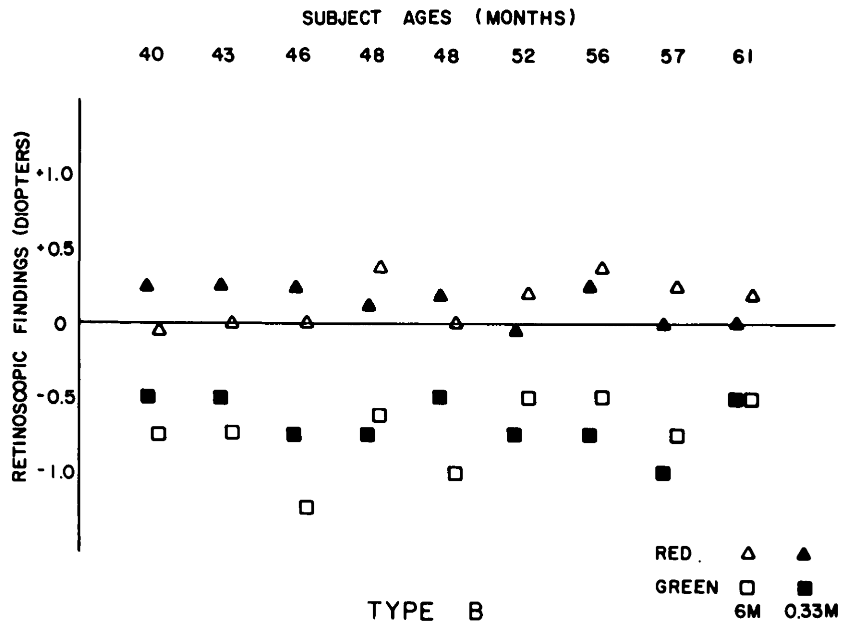

Fig. 3. Retinoscopic measurements through red and green filters for 0.33 and 6 m target distances for subjects from 40 to 61 months of age. In these subjects (Type B) red is in focus for both distances.

The retinoscopic working distance (0.33 m) was controlled by using a string of appropriate length, one end of which was attached to the retinoscope while the retinoscopist held the other end against the child's forehead. One experimenter remained beside the fixation chart and asked the child to describe the direction in which each symbol pointed. Measurements were made only when the retinoscopist was certain the child's attention was directed to the chart. A subtraction of three diopters was made from the lens power needed for neutrality, to compensate for the working distance of 0.33 m.

Measurements were confined to the horizontal meridian of the right eye. Retinoscopy was performed without filters and through Kodak Wratten Filters No. 25 and 55 with dominant wavelengths of 615 and 530 nm, respectively. Lenses were added in front of the eye being measured until movement of the light reflex was neutralized. The retinoscopist directed his attention to the center of the pupil reflex. Pupil size was not controlled, since an earlier study indicated that this factor had little or no effect on chromoretinoscopy.[5]

Dynamic retinoscopy was performed with a fixation and working distance of 0.33 m. The target consisted of a near-point Snellen chart designed for children by the American Optical Co. (cat. no. 11087). It consists of black symbols of varying sizes on a white background. The child was asked to identify the smaller figures. Measurements were made through the two filters specified above and with no filter. Care was taken to ensure that the child's attention was directed toward the target when measurements were made. The fact that the amounts of chromatic aberration measured in all subjects fall within normal limits and that it was approximately the same for both distances (Figs. 2 to 4) supports the reliability of the findings and the accuracy of fixation during measurements.

In 14 of the 26 children tested, measurements were carried out by one of the experimenters. The remaining 12 were examined independently by two experienced retinoscopists who were not aware of the purpose of the study.

Results and discussion. The results indicate that accommodative behavior in many children differs from that of adults. Although some overlap exists, the findings may be broken down into three general types on the basis of age: type A, 48 to 80 months; type B, 40 to 61 months; and type C, 31 to 45 months (Table I). The findings for type A (11 children) were essentially the same as those reported in an earlier study carried out with adults[5]

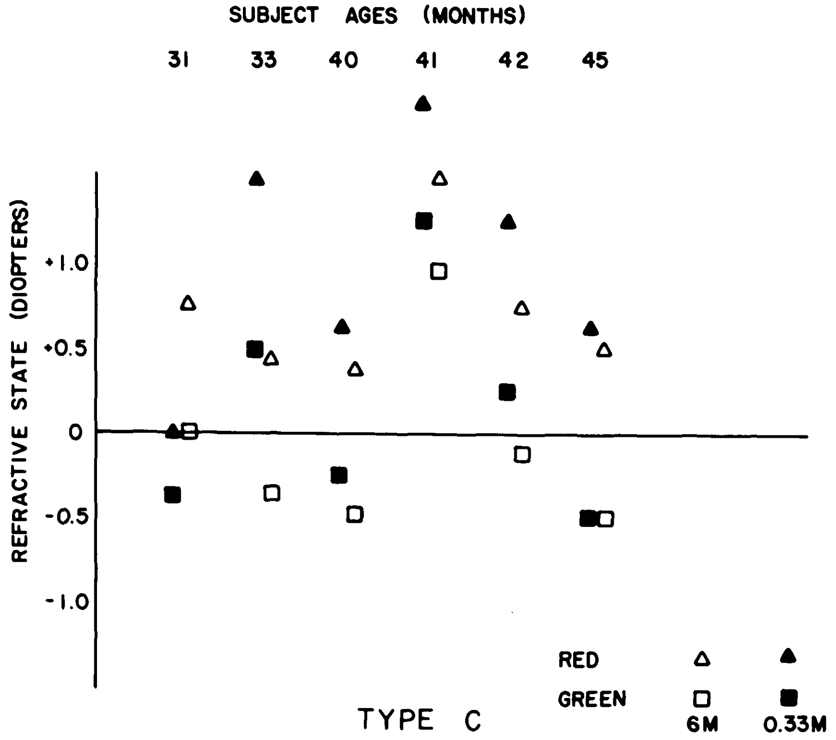

Fig. 4. Retinoscopic measurements through red and green filters for 0.33 and 6 m target distances for subjects from 31 to 45 months of age. In these subjects (Type C), unlike types A and B, no consistent pattern of focusing is apparent.

(Figs. 1 and 2). In these subjects distance retinoscopy findings showed the red wavelengths in focus on the retina while the green were focused about 1.00 diopter in front of the retina. While fixating at near, these subjects showed green to be in focus, but to bring the red end of the chromatic aberration interval into focus would require an addition of about 1.00 diopter. This is consistent with the premise that the eye uses its chromatic aberration to spare accommodation.

A second group consisting of nine children (type B) showed that the red wavelengths were focused in the retinal plane while the children were fixating both distance and near targets (Fig. 1). These subjects did not, therefore, use the chromatic aberration interval to spare accommodation. Instead they indicated a preference for focusing red light in the retinal plane at both distances (Fig. 3).

The six remaining subjects (type C) showed a mixed response (Fig. 4), some focusing neither the red nor the green end of the chromatic aberration interval in the retinal plane, but selecting instead some intermediate portion of the interval (yellow perhaps) for both distances.

Should these data prove to be representative of children in these age groups, it suggests that the focusing mechanism is not fully developed until about the fourth year of life. There appear to be three stages to this development: the first being one in which there is haphazard focusing within the chromatic aberration interval, the second showing selective focusing of the red end of the interval but without sparing accommodation, and the third showing selective focusing with sparing of accommodation.

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