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Processing Delays in Amblyopic Eyes: Evidence from Saccadic Latencies

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Abstract
Saccadic latencies were measured in amblyopes with constant strabismus, amblyopes without strabismus, and intermittent strabismics with or without amblyopia. Subjects tracked a small spot of light, either monocularly or binocularly, which moved with random horizontal step displacements of 0.25–8.5 deg over the central field. Increased saccadic latencies were observed in the amblyopic eyes of 6 of 11 subjects, with or without strabismus; saccadic latencies were similar in each eye of 2 subjects having intermittent strabismus without amblyopia. Amblyopia was a necessary condition for increased saccadic latencies and not strabismus. Evidence for normal motor control of eye movements in amblyopic subjects is as follows: normal saccadic durations in the amblyopic eyes, normal saccadic-latency distribution curves for binocular tracking and monocular tracking with the nonamblyopic eyes, and synchronous movements of the 2 eyes. Our results are interpreted in terms of a processing delay in the sensory pathways leading from the central region of the amblyopic eye to the centers involved in saccadic initiation.

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

The function of the saccadic eye-movement system in normal humans is to foveate, by a fast, staccato movement, peripheral objects of interest in the visual field. One parameter of saccadic movements, saccadic latency, has been studied extensively both in normal subjects and in patients with neurological disease. Since Dodge's early report on average saccadic latency in normals,[1] other investigators have confirmed his results and have shown saccadic latencies to form a distribution function,[2-9] averaging approximately 200 msec with a standard deviation of 30 msec.[5, 7, 8] A variety of factors, including prediction,[2] practice,[9, 10]

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TABLE 1. Clinical data and experimentally measured saccadic latencies (msec) are tabulated for the right and left eyes of 13 subjects who participated in the present study. For 4 subjects saccadic latencies were measured at more than 1 experimental session; therefore multiple means and standard deviations are tabulated. An asterisk indicates that the mean saccadic latency for an amblyopic (or nondominant) eye was significantly longer than for the normal (or dominant) eye—t-test, $p < 0.05$.

Subject	Ametropia	Acuity	Squint Angle, Δ		Eccen. Fix., Δ		Corresp./ Age Surgery	Saccadic Latency (msec)	
								Monocular	Binocular
No./(Age)	Sph/Cyl/Ax	20/( )	Horiz	Vert	Horiz	Vert		Mean/SD/N	Mean/SD/N
Constant-strabismus amblyopia									
1	+2.25	15					Anom.	208/35/15	186/17/16
(25)	+2.00/−0.25/130	25	1.5 eso		0.5 nas		16	202/27/24	
2	+0.50	15					—	198/34/26	180/26/19
(23)	+3.75/−0.50/165	30	18 eso		1 nas		6	212/41/30	
3	−4.50/−1.25/25	20					Norm.	277/66/14	210/30/30
(26)	−10.25/−1.50/150	110	18 exo		1 nas	1 sup.	—	254/67/25	
4	−1.75	20					Anom.	250/56/14	235/46/14
(15)	−1.50	122	10 eso	1 hyper	2.5 nas	1 sup.	—	483/119/6*	
5	Plano	20					—	223/27/17	230/31/17
(32)	+4.00	277	4 eso		16 nas	4 sup.	—	427/156/11*	
								202/39/19	
								359/70/16*	
6	+0.25/−0.50/40	10					—	213/33/8	215/36/28
(33)	+0.25/−0.50/180	630	5.5 eso	2 hyper	3 nas	3.5 sup.	8	290/59/8*	
								—	
								315/71/11	
Amblyopia without strabismus									
1	Plano/−0.50/19	20	—				Norm.	189/26/16	171/22/18
(24)	+0.75/−2.00/90	38			2 nas	2 inf.	—	212/36/21*	
2	−5.00/−0.75/5	40	—		2 nas	2 inf.	Norm.	229/26/39	220/40/40
(25)	−2.50/−1.25/72	25					—	233/29/15	
3	+3.00	15	—				Norm.	210/27/21	221/27/24
(19)	+5.00	110			2 temp		—	314/75/25	
		15						—	
		110			2 temp			283/61/35	
		15						—	

		45			—			313/85/25	
		15						225/19/13	
		25			—			313/56/25*	
		15						—	
		20			0			313/60/31	
		15						—	
		20			0			314/76/31	
Intermittent strabismus with and without amblyopia									
1	−3.00/−0.25/100	20	18 exo	12 hyper	Unstead.		Norm.	224/32/14	226/36/12
(22)	−0.75/−0.25/148	20					—	246/46/14	
2	−4.50/0.75/20	20					Anom.	351/97/8	364/161/11
(31)	−5.00	20	15 exo		0		16	296/98/20	
3	+1.25/−3.50/5	23	45 exo		1 nas	1 sup.	Anom.	342/67/18*	—
(32)	+0.75/−1.75/180	20					—	259/36/10	
								260/44/23	
								—	
4	−1.00	16					Anom.	231/32/19	223/29/19
(25)	Plano	32	10 exo	20 hyper	0.5 nas		—	254/38/17	

fatigue and sleepiness,[10] target eccentricity,[7, 11-13] and target luminance,[14] may influence saccadic latency; it may[6, 15, 16] or may not[17-21] be increased in some neurological diseases.

In contrast to the numerous studies of saccadic latency in normal subjects and in patients with neurological disease, few investigations of saccadic latency or eye-hand reaction time have been conducted in patients with strabismus and/or amblyopia. Mackensen[22] studied saccadic latency and eye-hand reaction time in patients having constant-strabismus amblyopia. For peripheral retinal stimuli (~15 deg), saccadic latencies averaged 225 msec in the normal eye and 250 msec in the amblyopic eye, thus showing only a small increase in the amblyopic eye. To study the central retina, he then turned to eye-hand reaction time; subjects tapped a telegraph key with their finger when a small light viewed monocularly went on or off. Eye-hand reaction times averaged 225 msec in the normal eye and 325 msec in the amblyopic eye, these results showing abnormally long reaction times for central stimuli in the amblyopic eye. Mackensen attributed this 100-msec increase to a defect in the retina or sensory cortex of the afferent pathway of the amblyopic eye. Von Noorden[23] tested eye-hand reaction times in 2 patients having constant-strabismus amblyopia and 1 patient having amblyopia without strabismus. In all 3 subjects, increased reaction times were found in the amblyopic eyes, and the increase was especially pronounced for subjects having strabismus. There appeared to be a good correlation between visual acuity and reaction time in the amblyopic eyes. Von Noorden concluded, like Mackensen, that this effect was a manifestation of sensory disturbance in the amblyopic eye. Schor,[24, 25] using a 0.25-Hz square-wave stimulus having an amplitude of 6 deg, found mean saccadic latencies within normal limits, but exhibiting marked variability, in the amblyopic eyes of 5 patients having constant-strabismus amblyopia. There was a low correlation between visual acuity and mean saccadic latency in the amblyopic eyes.

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

SUBJECTS AND METHOD

Thirteen subjects were obtained from the clinics at the University of California, Berkeley, School of Optometry. All had a thorough vision examination (visual acuity was assessed by a psychophysical method[26]) and were free of ocular or neurological disease. Ages ranged from 15-33 yr, with a mean of 25.5 yr. Subjects included those having constant-strabismus amblyopia, amblyopia without strabismus, or intermittent strabismus with or without mild amblyopia; saccadic-latency results in subjects having latent or manifest nystagmus, with constant-strabismus amblyopia or intermittent strabismus, have been reported elsewhere.[27] The spectacle or contact lens prescription was worn during all testing. See Table 1 for clinical data on subjects.

A photoelectric method was used to record horizontal eye position.[2, 28] This method consisted of monitoring the amount of infrared light reflected from the horizontal limbal regions, which was directly related to changes in direction of gaze for the range of movements recorded. The bandwidth of the entire recording system was 75 Hz (−3 db). A chinrest and headrest, usually in conjunction with a bite bar covered with dental impression material, were used to stabilize the head.

A minicomputer generated random horizontal step displacements (0.25–8.5 deg) of a small spot of light (4–8 min arc) on a display monitor placed either 57 or 91 cm away on the subject's midline. Target luminance was always maintained at least 1 log unit above screen luminance.

Overall room illumination was maintained over a narrow range of moderate photopic levels; large variations in room illumination did not influence saccadic latency or other aspects of oculomotor control.

Saccadic latencies, for both monocular and binocular tracking, were measured directly from the eye-position traces on strip-chart paper. Paper speed ranged from 25–100 mm/sec; however, for the majority of measurements, it was 50 mm/sec, ensuring a sensitivity of about ±5 msec. In the graphic displays of saccadic latency versus percentage of response, bin widths of 50 msec were selected to ensure an adequate number of samples per bin.

RESULTS

The principal finding of this investigation was increased saccadic latencies in amblyopic eyes for stimuli presented over the central retina; a necessary condition for the occurrence of increased saccadic latencies was amblyopia, and not strabismus. Results within each diagnostic group are of particular interest.

Constant-Strabismus Amblyopia

The increases in saccadic latency were most striking in subjects 4–6 (Fig. 1 and Table 1). Mean saccadic latencies and variability were similar for monocular tracking with the normal eye and binocular tracking; mean saccadic latencies ranged from 202–250 msec with standard deviations ranging from 27–56 msec, and were in good agreement with previous investigations in normals.[1-9] For monocular tracking with the amblyopic eye, in contrast, mean saccadic latencies ranged from 290–483 msec with standard deviations ranging from 56–119 msec. These 3 subjects also had the lowest acuity. Increased latencies for the amblyopic eyes were also evident from the distribution curves; there was a pronounced increase in modal values for these subjects. The data for subjects 1–3 did not show significant trends.

Amblyopia without Strabismus

Similar results were obtained for 2 of 3 subjects in this diagnostic group (Fig. 2 and Table 1). Increased saccadic latencies were most striking for subject 3, who also exhibited the lowest acuity. Mean saccadic latency for this subject was about 100 msec *greater* for monocular tracking with the amblyopic eye than for either monocular

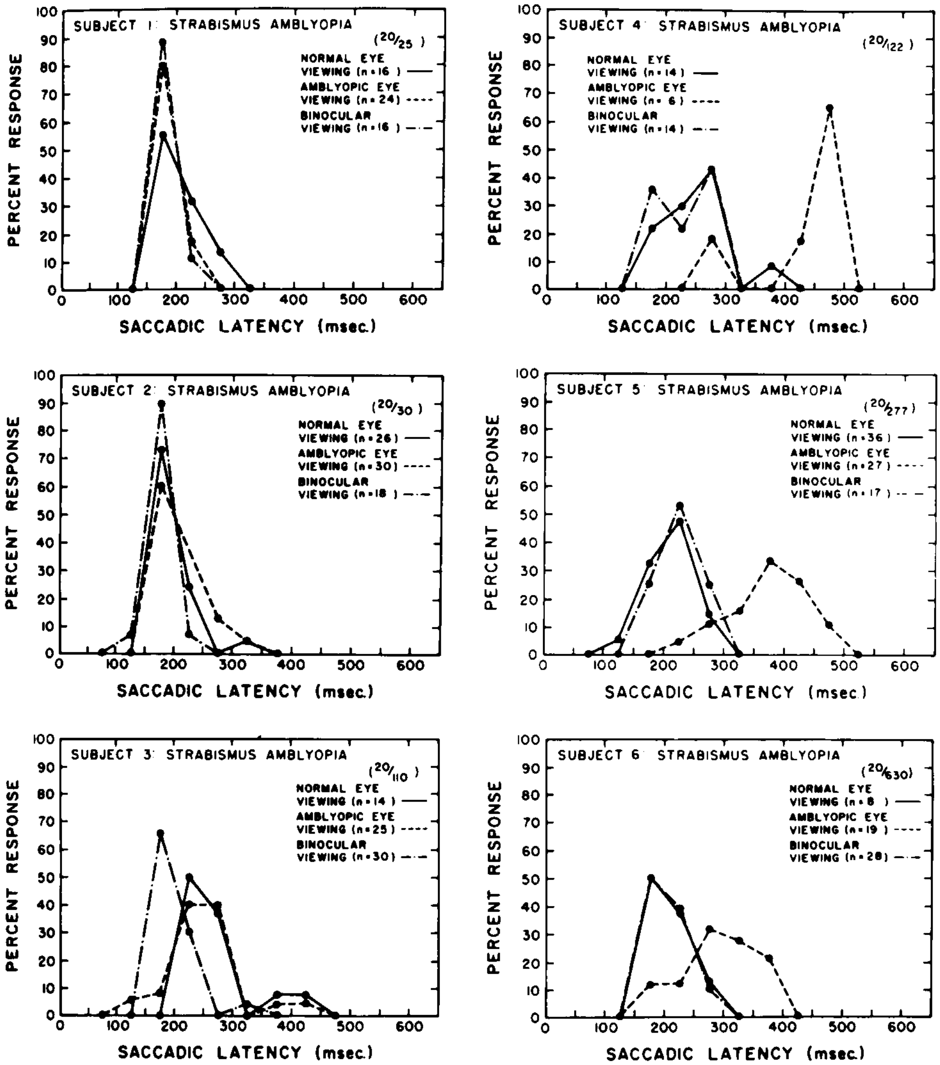

FIG. 1. Frequency distribution of saccadic latencies (msec) for 6 strabismic amblyopes. Subjects 4–6 had distinctly longer saccadic latencies when viewing with the amblyopic eye than when viewing with the normal eye or binocularly.

tracking with the normal eye or binocular tracking; a significant increase ($p < 0.05$). Pooled data for this subject, as well as single-session data, clearly demonstrated increased saccadic latencies in the amblyopic eye; the nearly exact overlap of saccadic-latency distributions for monocular tracking with the normal eye and for binocular tracking suggested a similarity in these 2 test conditions, and was in marked contrast to the amblyopic-eye distribution curves, where the modal values were increased, and a greater percentage of increased saccadic latencies was found. Most interestingly, as visual acuity (and fixation) tended to normalize in this subject, saccadic latencies remained abnormally high (Table 1). A significant increase ($p < 0.05$) in saccadic latency for the amblyopic eye of subject 1 was also found. No significant trends were evident for subject 2.

Intermittent Strabismus with or without Mild Amblyopia

The finding of increased saccadic latencies was also suggested from the data of 2

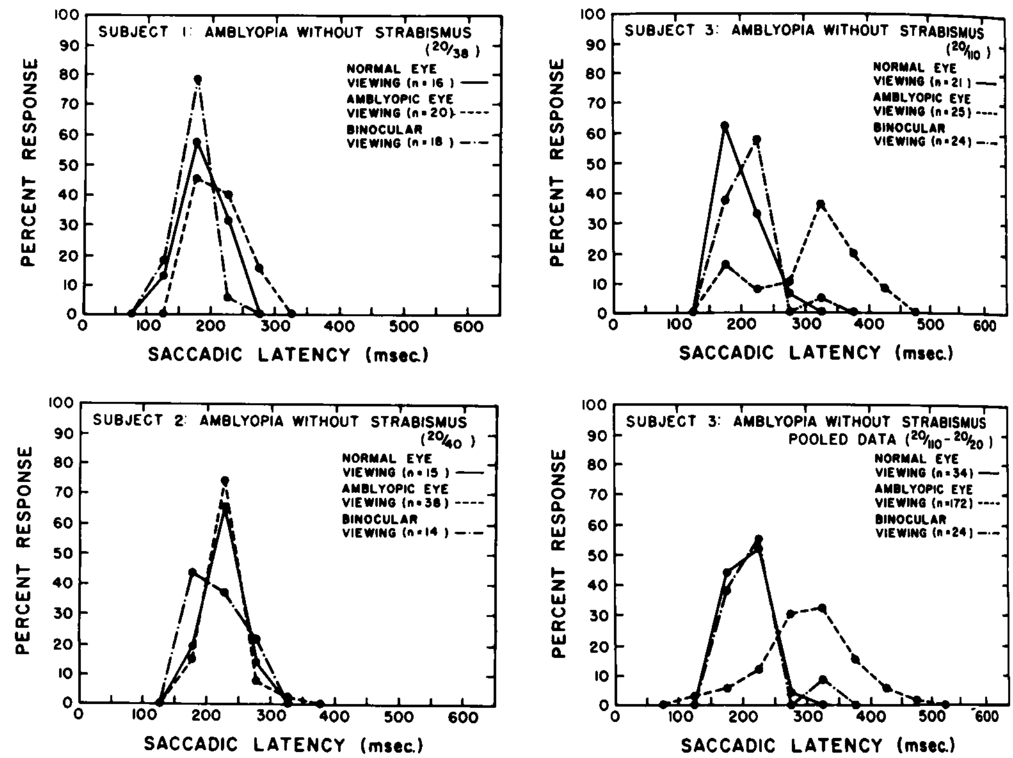

FIG. 2. Frequency distributions of saccadic latencies (msec) for 3 amblyopes without strabismus. Subject 3, who had fairly deep amblyopia (20/110), had distinctly longer saccadic latencies when viewing with the amblyopic eye than with the normal eye or binocularly. The lower-right plot shows the pooled data for the period during which the acuity was improved by treatment from 20/110 to 20/20.

subjects having intermittent strabismus and mild amblyopia (Fig. 3 and Table 1). Data for subject 3 showed a significant increase ($p < 0.01$) in saccadic latency, as well as increased variability in this measure, for the amblyopic eye (at 1 test session). Data for subject 4 showed a similar trend ($p < 0.10$). For subjects 1 and 2, both without amblyopia, saccadic latencies for monocular tracking with the 2 eyes were not significantly different; however, subject 2 exhibited high mean saccadic latencies and high variability under all test conditions.

Group Data

Similar trends were evident in the combined data for the 3 diagnostic groups (Fig. 4). For subjects having constant-strabismus amblyopia, there was a significant difference ($p < 0.01$) between the amblyopic eye (280 ± 99 msec) and the normal eye (223 ± 49 msec). For subjects having amblyopia without strabismus, 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 intermittent strabismus, however, 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

Our finding of increased saccadic latencies in amblyopic eyes suggests a slowing in the sensory pathways that process visual information subsequently used by the oculomotor system in generating saccadic eye movements. Three findings provide evidence for normal motor control of eye movements in amblyopic subjects. First, that the saccades were generated by normal pulse-step motoneuronal controller signals is supported by the data in Fig. 5, where

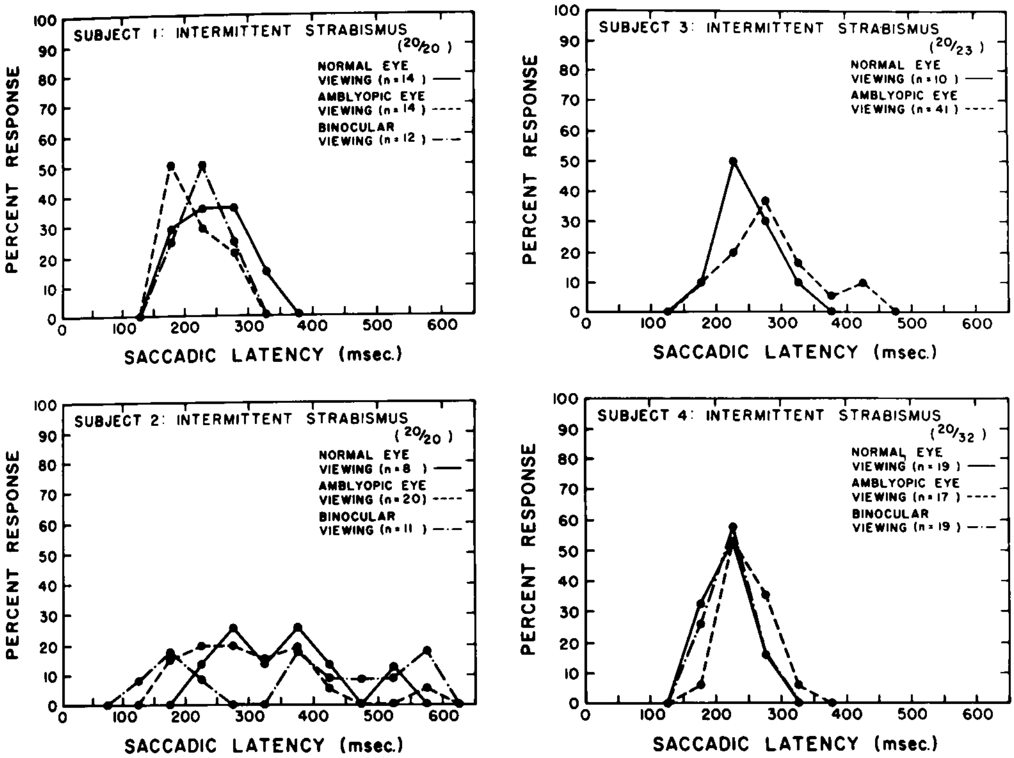

FIG. 3. Frequency distributions of saccadic latencies (msec) for 4 intermittent strabismics, 2 without amblyopia (subjects 1 and 2) and 2 with mild amblyopia. Subjects 3 and 4 had somewhat longer saccadic latencies with the amblyopic eye viewing than with the normal eye or binocularly.

saccadic amplitude is plotted against saccadic duration for the normal and the amblyopic eyes; the data are in agreement with saccadic durations for normal subjects.[29] Second, the precise synchronous movements[30] of the 2 eyes under all test conditions further supported the idea of normal neurological control of saccadic eye movements, guided by 2 basic laws of oculomotor physiology—Descartes's law of reciprocal innervation[31] and Hering's law of equal innervation[32]—in these amblyopic subjects. Third, saccadic latencies for binocular tracking, as well as for monocular tracking with the nonamblyopic eye, were within normal limits.

The results of this study are consistent with other investigations involving timed events utilizing the amblyopic eye, including eye-hand reaction times,[22, 23] perceptual blanking,[33] visual cognitive processing times,[34] and eye-hand coordination activities;[35] however, they are not in agreement with previous studies of saccadic latency in amblyopic eyes. Mackensen[22] found only a small (25 msec) increase in mean saccadic latency in amblyopic eyes, and Schor[24, 25] found normal mean saccadic latencies but unusually high variability in these measures. Can we account for the differences in results between these studies and our own? First, Mackensen used peripheral retinal stimuli (~15 deg); in the present study, step displacements were never larger than 8.5 deg. Second, Mackensen grouped his data according to percentage of response in the normal and amblyopic eyes of his subjects, who manifested a wide range of acuities (Mackensen, Figs. 3 and 4); in the present study, the data were analyzed in 2 ways for each diagnostic category: (1) individual subject data (Figs. 1-3) and (2) group data (Fig. 4). Similarities between saccadic-latency-distribution curves in Mackensen's

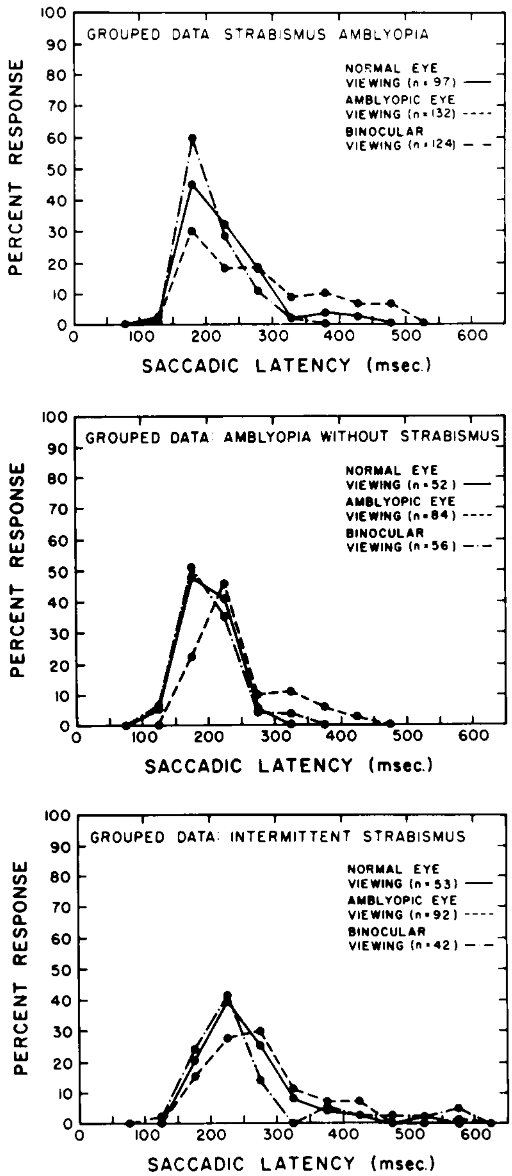

FIG. 4. Frequency distributions of saccadic latencies (grouped data) for 3 groups of subjects. The percentage of frequency (response) was calculated by dividing the number of responses in a bin by the total number of responses obtained for 1 viewing condition. For all 3 groups, the amblyopic eyes had a higher percentage of long saccadic latencies.

Figs. 3 and 4 and in Fig. 4 of our study are striking. Comparing Fig. 4 of our study with Figs. 1–3 of our study shows that grouping the data tends to mask the effects of subjects having low acuity and increased saccadic latencies by subjects having high acuity and normal saccadic latencies. Third, upon study of Mackensen's individual subject data displaying the range of saccadic latencies (his Fig. 2), it is clear that a few subjects in each acuity grouping showed a wide range of latency values, indicating the presence of at least some increased saccadic latencies. Schor's results may be accounted for by the stimulus conditions; step displacements with constant frequency and amplitude were employed. It is possible that, at times, subjects predicted stimulus changes after only a few repetitions, thus producing some abnormally short or even negative saccadic latencies; definite signs of prediction for saccadic tracking of periodic stimuli in naive clinical patients and normal control subjects have been observed after only a few cycles of movement (personal observation). Moreover, Hackman[9] has clearly shown increased variability in saccadic latency when subjects had knowledge of future target location. Hence, caution must be exercised in interpreting results obtained with repetitive stimuli.

Several important facts were uncovered by comparing the clinical data with the saccadic-latency data (Table 1). In constant-strabismus amblyopia, the 3 subjects with the most pronounced increases in saccadic latency had lower acuity than the others, were small-angle esotropes, and had the largest magnitudes of eccentric fixation; type of refractive error or correspondence, presence of vertical deviation, or past history of strabismus surgery did not appear to be related. In amblyopia without strabismus, 2 of 3 subjects had significantly increased latencies in the amblyopic eye. The subject with the worst acuity showed markedly increased saccadic latencies; most interestingly, saccadic latency remained abnormally high even after 20/20 acuity was attained and fixation became central. Type of refractive error or correspondence, magnitude of eccentric fixation, or past history of extraocular-muscle surgery did not appear to be related to increased saccadic latencies. In intermittent strabismus, mild amblyopia and small magnitudes of eccentric fixation were found in those subjects having increased saccadic latencies. We therefore conclude that the principal common feature in all subjects exhibiting in-

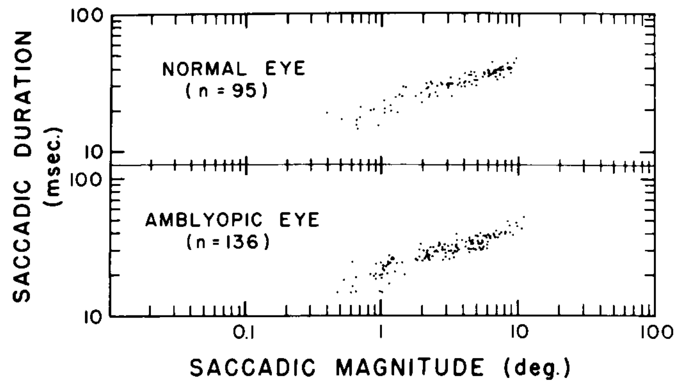

FIG. 5. Saccadic duration (msec) was found to be related to saccadic magnitude (deg) in a similar way for 95 measures of 13 normal eyes (upper) and 136 measures of 13 amblyopic (or nondominant) eyes (lower).

creased saccadic latencies was the presence of amblyopia.

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PENNSYLVANIA COLLEGE CELEBRATES OPENING OF NEW EYE INSTITUTE BUILDING

Opening ceremonies for the new building of The Eye Institute of the Pennsylvania College of Optometry were held on April 7, 1978. Dr. Leonard Bachman, Secretary of Health, Commonwealth of Pennsylvania, was among the special guests gathered to celebrate this milestone in the history of the 59-year-old institution. The entire community was invited to an open house at the Institute the following Sunday.

The Eye Institute offers total eye care to the public and provides professional training for future optometrists. It serves as a major resource for the general public and for referrals from health care practitioners of all disciplines in and beyond the Delaware Valley. The new $5.3 million building was partially financed with a $3.7 million grant from the U.S. Department of Health, Education, and Welfare.