Abnormal saccadic substitution during small-amplitude pursuit tracking in amblyopic eyes

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

Small-amplitude, low-velocity, predictable triangular tracking was tested in patients having amblyopia without strabismus, intermittent strabismus, or constant strabismus amblyopia by means of a photoelectric eye-movement recording technique. In the majority of amblyopic patients, abnormal saccadic substitution was found; that is, abnormally large saccades rather than small-amplitude smooth movements were used by the amblyopic eye to follow a spot stimulus that moved horizontally with low to high frequencies. Pursuit for the same range of stimuli was normal for binocular tracking and for monocular tracking with the dominant eye, pointing to a sensory rather than motor basis for the defect. This abnormal saccadic substitution response appeared to be related to the presence of amblyopia rather than strabismus. Several possible mechanisms responsible for causing this unusual response are discussed, including impairment of direction sense over small central regions of the amblyopic eye.

Key words: amblyopia, pursuit movements, saccadic movements, abnormal direction sense, suppression, strabismus, eye movements

During tracking of a smoothly moving target, the pursuit system attempts to match eye velocity with target velocity and thereby stabilize the target image on the retina. Accumulated position errors between the target and central retina are corrected by the saccadic system, which places the image of the target within the sensitive central foveal region.[1, 2] Thus the end result of accurate tracking is a relatively stabilized image of the moving target on the fovea.

Pursuit performance has been investigated in patients having amblyopia and strabismus by means of objective eye-movement recording techniques. Von Noorden and Mackensen[3] found tracking of large-amplitude, sinusoidally moving stimuli to be impaired at target frequencies beyond approximately 0.5 and 0.3 Hz in the normal and amblyopic eyes, respectively, of strabismic amblyopes. Fukai et al.[4] found pursuit abnormalities for tracking of large-amplitude, sinusoidally moving stimuli in the amblyopic and dominant eyes in many patients having amblyopia (with or without strabismus). Marked directional impairment of pursuit for targets (6 degrees peak-to-peak amplitude) moving into the nasal retina of amblyopic eyes in patients having constant strabismus amblyopia has recently been reported.[5]

We expanded on previous investigations by using small-amplitude stimuli, extending the range of stimulus frequencies used, and including patients from three clinically important diagnostic groups. In our study, pursuit movements in patients having amblyopia without strabismus, intermittent strabismus, or constant strabismus amblyopia were tested. Binocular tracking, as well as monocular tracking with the amblyopic or the dominant eye, were evaluated. Our new finding was abnormal saccadic substitution during small-amplitude pursuit tracking in amblyopic eyes; that is, abnormally large saccades rather than small-amplitude smooth movements were used to track the target.

Methods

A photoelectric method was used to record horizontal eye position.[6] The bandwidth of the entire recording system was 75 Hz (−3 dB). A headrest and chinrest, as well as a bite bar in most cases, were used to stabilize the head. Eye movements were analyzed directly from strip chart records.

A minicomputer (PDP-8/I) was used to generate a constant velocity (triangular) stimulus in the horizontal meridian having amplitudes of 1, 2, 4, and 8 degrees and velocities of 0.95, 1.75, 2.25, 3.75, and 6.75 degrees/sec (corresponding to frequencies of 0.48, 0.89, 1.12, 1.88, and 3.38 Hz, respectively), in any possible amplitude-velocity combination. All combinations were not tested on each patient. The small target (6 min arc) consisted of a bright spot of light viewed by the subject on a display screen placed either 57 or 91 cm away on the midline. Target luminance was always maintained at least 1 log unit above background screen luminance. Mean smooth-pursuit gain was determined by averaging values of smooth-pursuit gain (eye velocity divided by target velocity) for each individual segment of (smooth) tracking for 5 to 15 cycles of target motion.

Twelve patients were obtained from either the general refraction clinic or the orthoptics clinic at the optometry school. Ages ranged from 15 to 33 years, with a mean age of 25.5. All patients had a thorough vision examination and were free of ocular or neurological disease. Only functional amblyopes were used in the study. Patients from three diagnostic groups were included: (1) amblyopia without strabismus, (2) intermittent strabismus

(with or without mild amblyopia), and (3) constant strabismus amblyopia. We classified patients according to the presence of amblyopia and/or strabismus. This classification has proved successful for us in terms of relating eye movement abnormalities in our patients to either amblyopia or strabismus.[7-13] We are aware of two other classifications of amblyopia,[14, 15] both based on the clinical condition (i.e., strabismus or anisometropia) believed responsible for producing the amblyopia. We believe that all three classifications provide important, but different, ways of approaching and understanding underlying mechanisms and abnormalities related to amblyopia and strabismus. Spectacle or contact lens prescription was worn during all testing. See Table I for complete clinical data.

During monocular tracking, the eye not being tested either was tightly covered by a full, black patch and black tape or was occluded by a large, black partition blocking the target and most of the room from that eye's view. Method of occlusion did not have any effect on eye movements.

Results

The results of small-amplitude tracking with the amblyopic eye clearly demonstrated that patients from each diagnostic group primarily used saccades rather than smooth movements to follow the target (Fig. 1, *a* to *c*). Moreover, the amplitudes of these tracking saccades were generally two to five times larger than the target amplitude. Thus substitution of small-amplitude smooth movements by abnormally large saccades occurred during small-amplitude, constant-velocity tracking with the amblyopic eye in response to both nasalward and templeward target motions on the retina. This abnormal saccadic substitution was not found during binocular tracking or monocular tracking with the dominant eye (Fig. 2) in any patient. Mean pursuit gain in the dominant eye generally ranged from 0.7 to 0.95. These values for predictable triangular tracking with the dominant eye compare well to those values found under the same test conditions in our laboratory for normal (nonamblyopic) control subjects. Moreover, these values compare well to those reported by Puckett and Steinman[16] (0.6 to 0.9) for tracking 3-degree randomized

Table I. Clinical data of patients and presence of abnormal saccadic substitution in amblyopic (or nondominant) eye

Patient	Age	Prescription	Visual acuity	Vergence abnormality (prism diopters)	Eccentric fixation (prism diopters)
Constant strabismus amblyopia:					
1	25	LE + 2.00 = −0.25 × 130	20/25	1-2 ET LE	1/2 nasal LE
		RE + 2.25	20/15		
2	23	LE + 3.75 = −0.50 × 165	20/30	18 ET LE	1 nasal LE
		RE + 0.50	20/15		
3	15	LE − 1.50	20/122	10 ET LE and	2.5 nasal and 2 su-
		RE − 1.75	20/20	1 HT LE	perior LE
4	32	LE + 4.00	20/277	4 ET LE	16 nasal and 4 su-
		RE plano	20/20		perior LE
5	33	LE + 0.75 = −0.50 × 40	20/630	5-6 ET LE and	2.5-3.5 nasal and
		RE + 0.25 =0.50 × 180	20/10	2 HT LE	3-4 superior LE
Amblyopia without strabismus:					
6	24	LE + 0.75 = −2.00 × 90	20/38	None	2 nasal and 2 in-
		RE pl = −0.50 × 19	20/20		ferior LE
7	25	LE − 2.50 = −1.25 × 172	20/25	None	2 nasal and 2 in-
		RE − 5.00 = −0.75 × 5	20/40		ferior RE
8	19	LE + 5.00	20/110	None	2 temporal LE
		RE + 3.00	20/15		
Intermittent strabismus with or without mild amblyopia:					
9	22	LE − 0.75 = −0.25 × 148	20/20	18 ALT. XT and	Slight unsteady
		RE − 3.00 = −0.25 × 100	20/20	12 HT LE	central LE, RE
10	31	LE − 5.00	20/20	15 XT LE	Central steady
		RE − 4.50 = −0.75 × 20	20/20		LE, RE
11	32	LE + 0.75 = −1.75 × 180	20/20	40-50 XT RE	1 nasal and
		RE + 1.25 = −3.50 × 5	20/23		superior RE
12	25	LE plano	20/32	10 XT LE and	1/2 nasal LE
		RE − 1.00	20/16	20 HT LE	

single ramps having velocities of 1 and 2 degrees/sec. Although abnormal saccadic substitution rarely occurred for tracking of larger target amplitudes (4 and 8 degrees) in these patients (Fig. 3), other pursuit abnormalities found included saccadic intrusions[7, 11] (see Discussion for description), marked variability of pursuit gain,[7] and infrequent marked directional smooth-pursuit gain asymmetry[7] (high gain (>0.7) smooth pursuit in one direction and primarily saccadic tracking in the opposite direction). Abnormal saccadic substitution, if found in a patient, was almost always (~95% of the time) present for 1-degree target amplitudes, generally present (~85% of the time) for 2-degree target amplitudes, and infrequently present (<5% of the time) for 4- or 8-degree target amplitudes. It was present for the full range of low, moderate, and high target frequencies used (see Methods). Mean pursuit

gain for tracking of 4- and 8-degree amplitudes with the amblyopic eye generally ranged from 0.4 to 0.7. No direct relationship between depth of amblyopia and degree of tracking anomaly was evident, although abnormal saccadic substitution was always found in moderate and deep amblyopes.

Abnormal saccadic substitution was found during monocular tracking with the amblyopic eye in one of three patients having amblyopia without strabismus (see Table I). A representative record is presented in Fig. 1, *a*. Following the initial brief period when the patient appeared not to be trying to track the target, large saccades having amplitudes up to 2.5 degrees (middle of trace) and small saccades having amplitudes of approximately 1.0 degree were used to track the target. Little evidence of smooth movements was present in the tracking records.

Abnormal saccadic substitution was found

Presence of abnormal saccadic substitution	Amblyopia therapy or corrective surgery
No	Surgery, age 16
Yes	Surgery, age 6
Yes	No
Yes	No
Yes	Surgery, age 6
No	No
No	No
Yes	Amblyopia therapy before & during study
No	No
No	Surgery, age 16
Yes	Therapy initiated 1 month before study
No	No

during monocular tracking with the amblyopic eye in four of five patients having constant strabismus amblyopia (see Table I). A representative record for Patient 4 is presented in Fig. 1, b. Evident was a marked absence of smooth movements. Tracking occurred primarily by large saccades (up to 5 degrees in amplitude). These large tracking saccades were initiated approximately 350 to 900 msec following a change in target direction. Similar results were found for the other three patients.

Abnormal saccadic substitution was found during monocular tracking with the amblyopic eye in one of two patients having intermittent strabismus with mild amblyopia. Neither of two patients having intermittent strabismus without amblyopia exhibited this phenomenon (see Table I). A representative record for Patient 11 is shown in Fig. 1, c. Tracking with the amblyopic eye was per-

formed primarily by abnormally large saccades (up to 5 degrees in amplitude). However, at target turnaround points, there was some evidence of smooth tracking (with smooth pursuit gain as high as 0.7 for brief 300 msec periods).

Discussion

Abnormal saccadic substitution was found in the majority of amblyopic eyes during small-amplitude, constant-velocity tracking for a variety of low (~0.5 Hz), moderate (~1.0 Hz), and high (~2.0 Hz) target frequencies. It is interesting that saccadic substitution commonly occurs in normal subjects during high-frequency tracking of smoothly moving targets; however, saccadic amplitudes are typically less than or equal to target amplitude.[6] That predictive mechanisms[6] exerted little influence during tracking was suggested by two general findings. (1) The large tracking saccades generally occurred at least 200 msec following a change in target direction; the presence of tracking saccades initiated up to 900 msec following a change in target direction is consistent with recent reports of increased saccadic latencies in amblyopic eyes.[9, 10] (2) The eye lagged behind the target when smooth movements were present.

Does abnormal saccadic substitution appear to be related primarily to the presence of amblyopia or strabismus? Two lines of evidence suggest it is the former. (1) Patient 8 having amblyopia without strabismus exhibited abnormal saccadic substitution. (2) Our two patients having intermittent strabismus without amblyopia did not display this anomalous tracking response. These findings suggest that amblyopia and not strabismus is the primary common factor. Only Patient 11 represents a possible exception to our conclusion. However, this patient appears to us to represent a borderline case. With Patient 11 having eccentric fixation of 1 prism diopter, visual acuity of 20/23, and increased saccadic latencies as frequently as found in more typical amblyopic patients,[9, 10] we believe that placement into an "amblyopic" classification is justified, even though
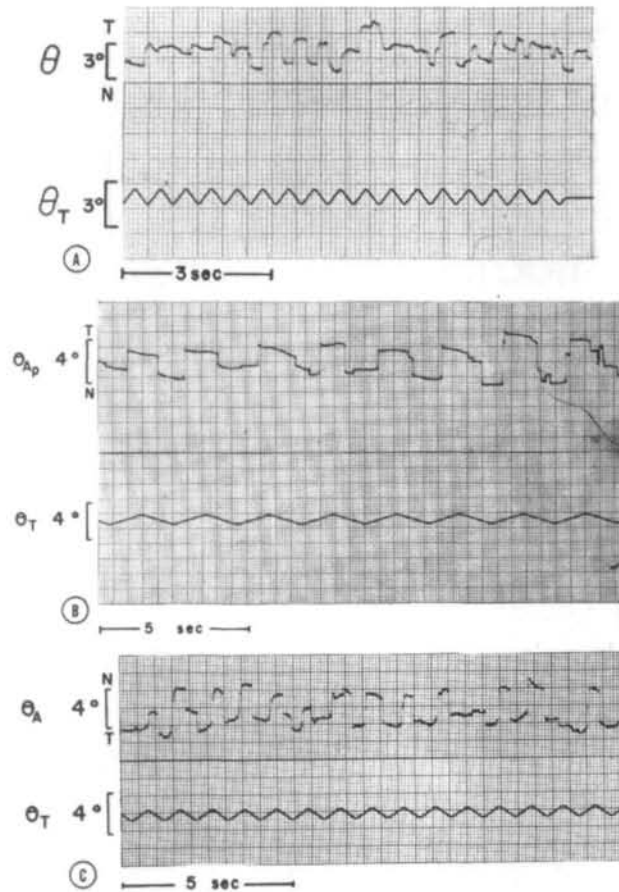
Fig. 1. Monocular tracking with the amblyopic eye. Top traces are eye positions, and bottom traces are stimuli. T and N near calibration bars indicate templeward and nasalward movements of eye, respectively. A, Patient 8: amblyopic eye 20/110; amblyopia without strabismus. Stimulus is 1.0 degree amplitude, 3.75 degrees/sec velocity, 1.88 Hz. Abnormally large saccades (up to 2.5 degrees in amplitude) and some small saccades (<1 degree in amplitude) used to track target. Little evidence of smooth tracking found. B, Patient 4: amblyopic eye 20/277; constant strabismus amblyopia. Stimulus is 1.0 degree amplitude, 0.95 degrees/sec velocity, 0.48 Hz. Little evidence of smooth following movements found; instead, target tracked by abnormally large saccades (up to 5.0 degrees in amplitude). C, Patient 11: mildly amblyopic eye 20/23; intermittent strabismus. Stimulus is 1.0 degree amplitude, 2.25 degrees/sec velocity, 1.12 Hz. Abnormally large saccades (up to 5 degrees in amplitude), rather than smooth movements, generally used to track target. Some short periods of smooth tracking found at turnaround points. Results of these patients clearly shows similarity of abnormal tracking response across the three diagnostic groups, as well as lack of dependence between target frequency (with constant amplitude) and abnormal tracking response.

visual acuity was not 20/30 or worse, and the difference in acuity between the eyes was not two lines or greater, two common clinical criteria for specification of amblyopia.[17]

Does abnormal saccadic substitution represent a true tracking response, or is it simply a threshold phenomenon in which "ambly-opic fixation tremor" occurs until target motion is large enough to elicit smooth pursuit responses? Our evidence suggests the former is true. Fixational and tracking eye movements were recorded and studied in our patients and reported in detail elsewhere.[7-13] Several interesting and important abnor-
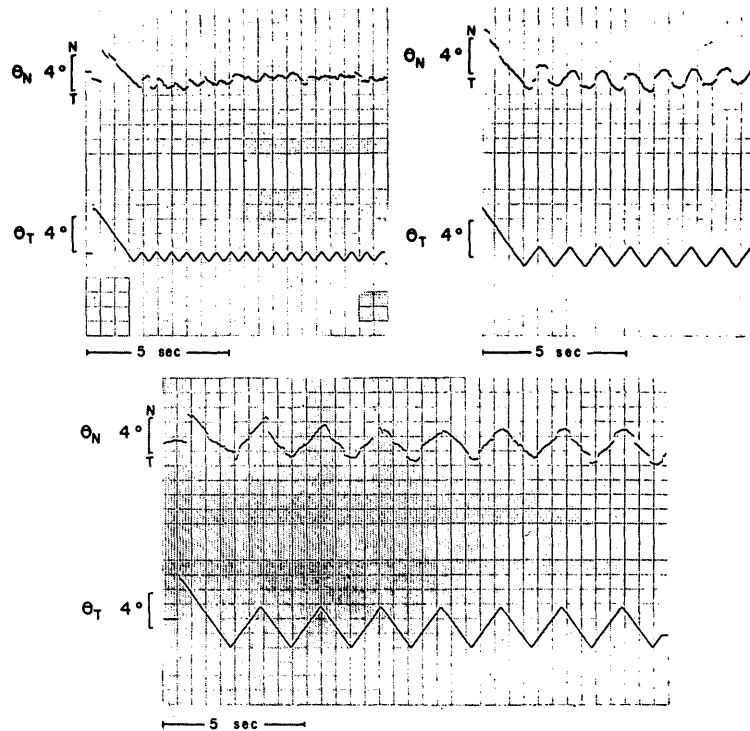
Fig. 2. Eye position as a function of time for dominant eye (20/15) of Patient 8 having amblyopia without strabismus. In each pair of records, upper trace is eye position, and lower trace is stimulus. Target amplitudes are 1 degree (upper left), 2 degrees (upper right), and 4 degrees (lower trace). T and N near calibration bars indicate templeward and nasalward movement of the eye, respectively. No evidence of abnormal saccadic substitution present, and pursuit gain within normal limits. Compare upper left trace of this figure to trace in Fig. 1, A, for Patient 8 tracking same target with amblyopic eye.

malities were noted, and some are presented in Figs. 4 and 5. The first abnormality is saccadic intrusions (a pair of saccades of approximately equal amplitude (average ~1.0 degree) but of opposite direction); they occur *irregularly* during fixation but average approximately 1/sec and have intersaccadic intervals ranging from 150 to 500 msec) (Fig. 4, upper left). These saccadic intrusions during fixation are quite different from the abnormal saccadic substitution response found in our amblyopes (Fig. 4, upper right). During abnormal saccadic substitution, *regular* movements (generally 2 to 5 degrees in amplitude) occur ~95% of the time in the same direction as target motion, and these are only found when patients *attempt to track* the small-amplitude stimulus. Thus abnormal saccadic substitution clearly represents a definite and

purposeful tracking response on the part of the patient to follow a moving stimulus (all patients *did* perceive the target as moving from side to side) and is not simply the occurrence of large, irregularly timed fixational saccades (saccadic intrusions). Furthermore, although the saccades of the abnormal saccadic substitution pattern occurred to either side of a previously established baseline position, in many instances saccadic intrusions during fixation were directed nasally in these same patients.[11] The second abnormality, increased drift amplitude[7] (up to 3.5 degrees peak to peak) and velocity[12] (up to 3.3 degrees/sec) (Fig. 4, lower left), resulted in relatively smooth, "saccade-free" traces, and this clearly represents a *fixation* response quite different from the abnormal saccadic substitution *tracking* response. The third abnormality is
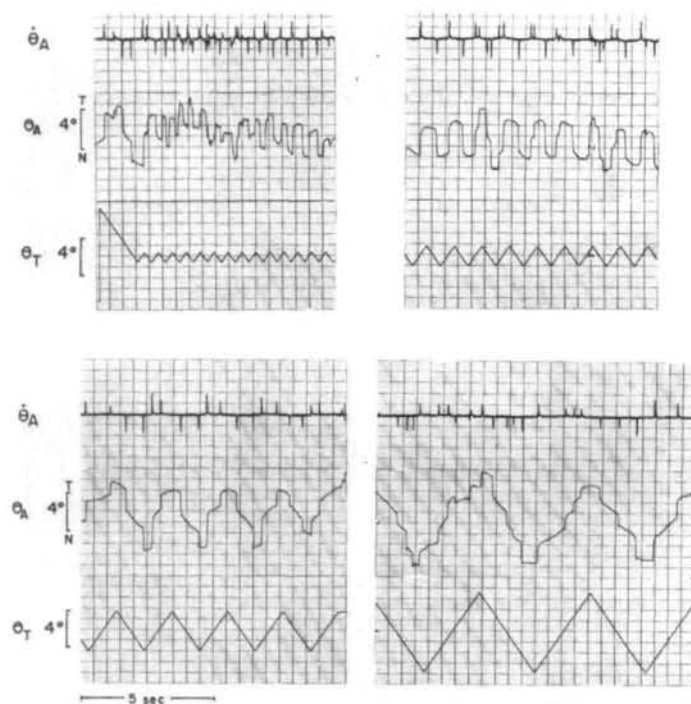
Fig. 3. Eye position as a function of time for amblyopic eye of Patient 8 having amblyopia without strabismus. Each set of traces show eye velocity, eye position, and stimulus, respectively, from top to bottom. Target amplitudes are 1 degree (upper left), 2 degrees (upper right), 4 degrees (lower left), and 8 degrees (lower right). T and N near calibration bars indicate templeward and nasalward movement of the eye, respectively. Abnormal saccadic substitution clearly seen for 1- and 2-degree stimuli, as well as some smooth movements at turnaround points; for 4-degree stimulus, saccadic pursuit evident with over-all tracking response amplitude being larger than stimulus amplitude; for 8-degree stimulus, saccadic pursuit evident, but over-all tracking response amplitude approximately equal to stimulus amplitude. Thus, as target amplitude increases (but with constant velocity maintained), pursuit becomes more normal-looking.

hypermetria. This was evidenced by single large saccades (Fig. 4, lower right) and static overshooting[18] (Fig. 5) during small-amplitude step tracking. These two findings suggest an increase in saccadic gain in the amblyopic eye for tracking of small target displacements over the central retina. The gain increase also provides a possible explanation, at least in part, for the large saccades found in our patients during predictable small-amplitude, constant-velocity tracking, i.e., the abnormal saccadic substitution response. That these abnormally large saccades frequently occurred 500 msec or more following a (predictable) target step change can be explained by the increased saccadic la-

tencies found in the amblyopic eyes of these two patients as is true for most amblyopic eyes.[9, 10]

Although there is no clear mechanism to explain the phenomenon of abnormal saccadic substitution, we would like to discuss three possibilities. The first is abnormal direction sense in amblyopic eyes. As a result of amblyopia, a depression of the directional-sensitivity gradient over the central retina may occur. This could result in impaired and/or marked variability of direction sense, including errors or biases in estimation of angular distance of targets relative to the preferred fixation locus. In the present situation, overestimation of small-angle target move-
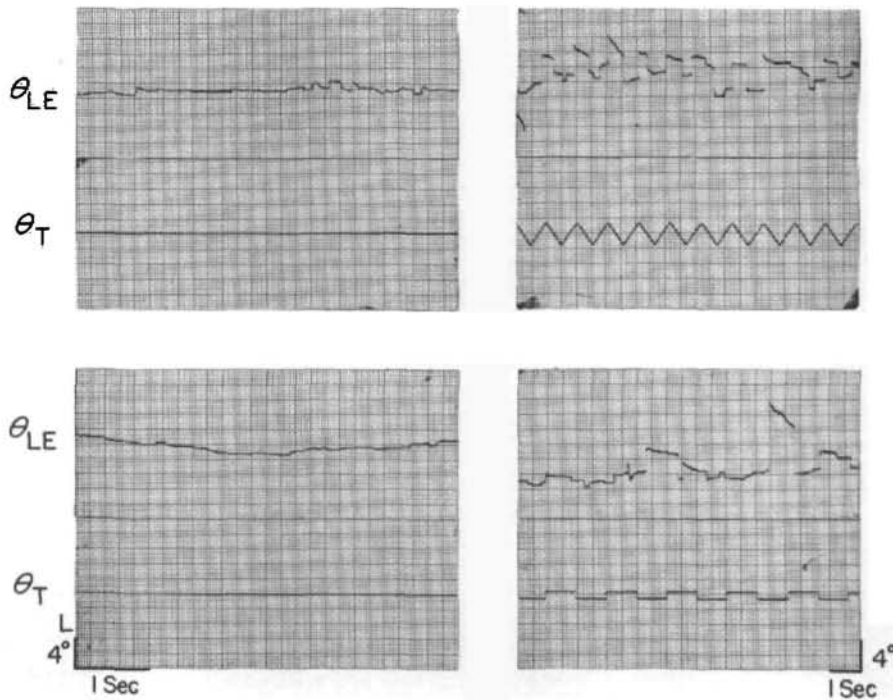
Fig. 4. Fixation and tracking responses for amblyopic eye of Patient 4 having constant strabismus amblyopia (20/277). In each set of records, upper trace is amblyopic eye position, and lower trace is stimulus. **Upper left,** Midline fixation showing intermittent steadiness of eye as well as some saccadic intrusions. Note irregularity and relatively small amplitude of intrusions. **Upper right,** Abnormal saccadic substitution in response to small-amplitude (2 degrees), constant-velocity (0.95 degrees/sec, 1.12 Hz) stimulus. Note regularity and large amplitudes of saccadic movements during abnormal saccadic substitution in contrast to irregularity and small amplitudes of saccadic intrusions during fixation (**Upper left**). Also note similarity between abnormal saccadic substitution shown here and response of same patient tracking stimulus having different amplitude (1 degree) and frequency (0.48 Hz) but same velocity (0.95 degrees/sec) (Fig. 1, **B**), suggesting generalized nature of response. **Lower left,** Fixation trace showing period of increased amblyopic drift[7] and only a few irregularly occurring small saccades. Thus both this trace and **Upper left** trace show *fixation* characteristics that are clearly much different from abnormal saccadic substitution *tracking* response (**Upper right**). **Lower right,** Predictable (0.5 Hz), small-amplitude (0.6 degrees) step tracking. Note some large saccades (up to 9.0 degrees in amplitude) in response to periodic target changes, suggesting fluctuations of saccadic gain, especially greater than 1.0. Increased saccadic gains noted for both abnormal saccadic substitution (**Upper right**) and step tracking suggest similar underlying mechanism operating in response to small-amplitude changes in retinal stimulation in amblyopic eye.

ments on the retina would result in the inappropriately large eye movements exhibited by our patients. If our abnormal-direction-sense hypothesis is correct and is generalized for all types of tracking, one would predict that hypermetria (static overshooting[18] and single large saccades) would be commonly found during small-amplitude step tracking;

although hypermetria was found in amblyopes (Figs. 4 and 5), it was not as consistent a finding as was abnormal saccadic substitution. However, it appears reasonable to assume that a constantly moving target providing repeated information regarding target direction by stimulating retinal regions in an orderly sequence would elicit this response in a more

Fig. 5. Predictable, small-amplitude step tracking response in former amblyopic eye of Patient 8 (similar to that found in same patient when visual acuity was 20/110[12]). Upper trace is eye position, and lower trace is stimulus (0.5 Hz, 0.6 degree amplitude). Note static overshoots present in majority of responses, suggesting increased gain of saccadic eye movement system for small changes in target position. This saccadic gain increase appears to be related to increased saccadic amplitudes observed in abnormal saccadic substitution responses (Fig. 4, **Upper right**) and the large saccades sometimes observed in Patient 4 during small-amplitude step tracking (Fig. 4, **Lower right**).

consistent manner than would a target which moves in relatively infrequent, discrete, random steps and only stimulates small, isolated retinal regions. Our idea of abnormal direction sense in amblyopic eyes is in agreement with that of previous investigators. Mackensen[19] believed that the physiological superiority of the fovea and the graded sensory function of the macula region had been transformed, due to amblyopia, into a region of equivalent sensory characteristics; Mackensen[20] also believed that eccentric fixation represented a transposition of the principal visual direction; von Noorden[21] believed that amblyopes possessed a vagueness of directional sense; and Griffin[17] believed direction sense in an amblyopic eye with eccentric fixation is impaired and results in past-pointing. These factors could contribute to the anomalous tracking response found in our patients. It is interesting to speculate that our notion of abnormal direction sense in amblyopic eyes, based upon *dynamic* test results, may be related to eccentric fixation, which is based upon *static* test results. Eccentric fixation is found in the majority of amblyopic eyes,[22, 23] and its presence suggests a shift in

the zero sensorimotor directional reference point under "steady-state" monocular test conditions. A second mechanism, possibly related to the first, is suppression. It is a well-established fact that suppression is commonly found in amblyopic eyes under binocular test conditions.[24, 25] Suppression may result in an over-all reduction in sensitivity over central retinal regions of the amblyopic eye. This sensorially depressed central zone would correspond to all or part of the area undergoing suppression in the amblyopic eye during binocular viewing. If this is true, a strategy of placing the target just outside this depressed region might be adopted during monocular tracking with the amblyopic eye. Impaired or vague direction sense would prevent the phenomenon of eccentric viewing from being reported. Thus the average size of these large tracking saccades (~2.5 degrees) may represent the horizontal angular dimension of this abnormal region. Direction, amplitude, and velocity of target movement could be processed for a longer period of time as target amplitude increased (and target movement on the retina increased). This could provide a possible ex-

planation for the abnormal tracking response (large tracking saccades and absence of smooth movements) found for small-target excursions and the more normal tracking response (lack of large tracking saccades and presence of smooth movements) for larger-target excursions. This would be consistent with the notion of increased information-processing delays in amblyopic eyes.[9, 10, 26-30] A third explanation might simply be that abnormal saccadic substitution represents a normal phenomenon for small-amplitude pursuit tracking with the peripheral retina. However, the persistence of this abnormal response in one subject whose fixation centralized during orthoptics therapy does not lend support to this idea.[12] Moreover, recent findings by Fukai and Tsutsui[31] suggest that nonfoveal tracking would result in asymmetric pursuit rather than the symmetric abnormal saccadic substitution found in our patients. That tracking small-amplitude stimuli binocularly or monocularly with the dominant eye never resulted in abnormal saccadic substitution further points to a sensory rather than motor basis for this pursuit defect.

The fact that all amblyopic patients within a diagnostic group, as well as across groups having similar visual acuities, did not exhibit abnormal saccadic substitution may be attributed to such factors as differences in age of onset or constancy of the anomaly, since these factors are related to the period of human vision development.[32-40] Moreover, effects due primarily to suppression, commonly found in strabismus with approximately equal refractive error, may differ from effects due primarily to mild form deprivation, as found in anisometropia. Further testing of sensory and motor function over the central region of amblyopic eyes will be required to elucidate possible interrelationships and common underlying mechanisms related to this pursuit defect.

REFERENCES

1. Rashbass, C.: The relationship between saccadic and smooth tracking eye movements, J. Physiol. 159: 326, 1961.
2. Young, L.R., and Stark, L.: Variable feedback experiments testing a sampled data model for eye tracking movements, IEEE Trans. Hum. Factors Electronics, HFE-4:38, 1963.
3. von Noorden, G.K., and Mackensen, G.: Pursuit movements of normal and amblyopic eyes: an electro-ophthalmographic study. II. Pursuit movements in amblyopic eyes, Am. J. Ophthalmol. 53:325, 1962.
4. Fukai, S., Tsutsui, J., and Nakamura, Y.: Abnormal pursuit movements of the fellow eye in amblyopia with strabismus. In Moore, S., Mein, J., and Stockbridge, L., editors: Orthoptics: Past, Present, Future, New York, 1976, Stratton Intercontinental Medical Book Corp.
5. Schor, C.M.: A directional impairment of eye movement control in strabismus amblyopia, INVEST. OPHTHALMOL. 14:692, 1975.
6. Stark, L., Vossius, G., and Young, L.R.: Predictive control of eye tracking movements, IRE Trans. Hum. Factors Electronics, HFE-3:52, 1962.
7. Ciuffreda, K.J.: Eye movements in amblyopia and strabismus, Ph.D. dissertation, School of Optometry, University of California, Berkeley, 1977.
8. Kenyon, R. V.: Vergence eye movements in amblyopia and strabismus, Ph.D. dissertation, School of Optometry, University of California, Berkeley, 1978.
9. Ciuffreda, K.J., Kenyon, R.V., and Stark, L.: Increased saccadic latencies in amblyopic eyes, INVEST. OPHTHALMOL. VISUAL SCI. 17:697, 1978.
10. Ciuffreda, K.J., Kenyon, R.V., and Stark, L.: Processing delays in amblyopic eyes: evidence from saccadic latencies, Am. J. Optom. Physiol. Opt. 55:187, 1978.
11. Ciuffreda, K.J., Kenyon, R.V., and Stark, L.: Saccadic intrusions in strabismus, Arch. Ophthalmol. (in press)
12. Ciuffreda, K.J., Kenyon, R.V., and Stark, L.: Different rates of functional recovery of eye movements during orthoptics treatment in an adult amblyope, INVEST. OPHTHALMOL. VISUAL SCI. 18:213, 1979.
13. Ciuffreda, K.J., Kenyon, R.V., and Stark, L.: Suppression of fixational saccades in strabismic and anisometropic amblyopia, Ophthalmic Res. (in press).
14. von Noorden, G.K.: Classification of amblyopia, Am. J. Ophthalmol. 63:238, 1967.
15. Amos, J.F.: Refractive amblyopia: its classification, etiology, and epidemiology, J. Am. Optom. Assoc. 48:489, 1977.
16. Puckett, J.W., and Steinman, R.M.: Tracking eye movements with and without saccadic correction, Vision Res. 9:695, 1969.
17. Griffin, J.R.: Binocular Anomalies: Procedures for Vision Therapy, Chicago, 1976, Professional Press Inc., pp. 74-75, 95-96.
18. Bahill, A.T., Clark, M.R., and Stark, L.: Dynamic overshoot in saccadic movements is caused by neurological control signal reversals, Exp. Neurol. 48:107, 1975.
19. Mackensen, G.: Blickbewegungen amblyopischer Augen, Graefes Archiv. Ophthalmol. 159:212, 1957.

20. Mackensen, G.: Diagnosis and phenomenology of eccentric fixation, Int. Ophthalmol. Clin. 6:397, 1966.
21. von Noorden, G. K.: Pathogenesis of eccentric fixation, Doc. Ophthalmol. 23:263, 1967.
22. Brock, F. W., and Givner, I.: Fixation anomalies in amblyopia, Arch. Ophthalmol. 47:775, 1952.
23. Girard, L. J., Fletcher, M. C., Tomlinson, E., and Smith, B.: Results of pleoptic treatment of suppression amblyopia, Am. Orthoptic J. 12:12, 1962.
24. Travers, T. B.: Suppression of vision in squint and its association with retinal correspondence and amblyopia, Br. J. Ophthalmol. 22:577, 1938.
25. Nawratzki, I., and Jampolsky, A.: Regional hemiretinal difference in amblyopia, Am. J. Ophthalmol. 46:339, 1958.
26. Mackensen, G.: Reaktionszeitmessungen bei Amblyopie, Graefes Archiv. Ophthalmol. 159:636, 1958.
27. von Noorden, G. K., and Burian, H. M.: Perceptual blanking in normal and amblyopic eyes, Arch. Ophthalmol. 64:817, 1960.
28. von Noorden, G. K.: Reaction time in normal and amblyopic eyes, Arch. Ophthalmol. 66:694, 1961.
29. Burian, H. M., Benton, A. L., and Lipsius, R. C.: Visual cognitive functions in patients with strabismus amblyopia, Arch. Ophthalmol. 68:785, 1962.
30. Goldstein, J. H., and Greenstein, F.: Eye-hand coordination in strabismus. In Fells, P., editor: The First Congress of the International Strabismological Association, St. Louis, 1971, The C. V. Mosby Co.
31. Fukai, S., and Tsutsui, J.: Asymmetric version in pursuit eye movement under extrafoveal fixation, Jpn. J. Ophthalmol. 17:30, 1973.
32. Hickey, T. L.: Postnatal development of the human lateral geniculate nucleus: relationship to a critical period for the visual system, Science 198:836, 1977.
33. Harris, L., Atkinson, J., and Braddick, O.: Visual contrast sensitivity of a 6-month-old infant measured by the evoked potential, Nature 264:570, 1976.
34. Banks, M. S., and Salapatek, P.: Acuity and contrast sensitivity in 1-, 2-, and 3-month-old human infants, INVEST. OPHTHALMOL. VISUAL SCI. 17:361, 1978.
35. Hohmann, A., and Creutzfeldt, O. D.: Squint and the development of binocularity in humans, Nature 254:613, 1975.
36. Banks, M. S., Aslin, R. N., and Letson, R. D.: Sensitive period for the development of human binocular vision, Science 190:675, 1975.
37. Sokol, S., and Dobson, V.: Pattern reversal visually evoked potentials in infants, INVEST. OPHTHALMOL. 15:58, 1976.
38. Marg, E., Freeman, D. N., Peltzman, P., and Goldstein, P. J.: Visual acuity development in human infants: evoked potential measurements, INVEST. OPHTHALMOL. 15:150, 1976.
39. Dobson, V., Teller, D. Y., and Belgum, J.: Visual acuity in human infants assessed with stationary stripes and phase-alternated checkerboards, Vision Res. 18:1233, 1978.
40. Sokol, S.: Measurement of infant visual acuity from pattern reversal evoked potentials, Vision Res. 18:33, 1978.