Visual perception of direction when voluntary saccades occur. I. Relation of visual direction of a fixation target extinguished before a saccade to a flash presented during the saccade[1]

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In experiments designed to clarify the mechanisms underlying the normal stability of visual direction for stationary objects when voluntary saccades occur, Ss reported on the horizontal visual direction of a brief test flash presented when the eye was at a specific point in the saccade (the trigger point) relative to a fixation target viewed and extinguished prior to the saccade. From these reports, PSEs (points of subjective equality) were calculated for the fixation target as measured by the test flashes. The distance of the trigger point from the previous fixation position was systematically varied in each experiment. Different experiments required saccades of different lengths and directions. With the exception of the presentation of the test flash the saccades were carried out in complete darkness so that the possible utilization of an extraretinal signal regarding the eye movement (change in eye position, the intention to turn the eye, or a change of attention related to the eye movement) in the determination of visual direction could be observed uncomplicated by a continuing visual context. According to classical theories, an extraretinal signal proportional to the change in eye position acts to maintain direction constancy by compensating for the shift of the retinal image resulting from the movement of the eye. In general, direction constancy was not preserved in the present experiments, and thus the data would not be predicted by classical theories. However, the PSE varied with distance of the trigger point from the fixation target. Since this displacement of PSE from the trigger point was in the correct direction for compensation, the presence of an extraretinal signal was confirmed. However, the growth of this signal appears to be time-locked to the saccade rather than locked to eye position; it is suggested that this growth takes place over a time period which is longer than the duration of the saccade itself.

Under normal illumination stationary objects do not appear to change location when we turn our eyes from one position to another, although the image of each of these objects stimulates different retinal regions at these different eye positions. Since a shift of the retinal locus stimulated is normally a sufficient condition for a perceived change of object location during steady fixation, it is clear that there are important differences between the conditions that determine the perception of object location during steady fixation and when voluntary eye movements occur. The retinal image of a single object moving in the environment involves a changed relation of this image to unshifted images of other objects, while during a voluntary saccade relative retinal loci of images do not change. This might appear to provide a simple basis for the difference between the appearances during steady fixation and when a voluntary saccade occurs. Two observations, however, suggest that such an explanation would be incomplete at best: (a) When an entire visual field (restricted to a frontal plane) is moved as a unit, no essential change in relative retinal loci occurs for images of objects in the field, yet movement may be perceived. (b) When the eye is moved by an external object, again, no change in relative retinal loci occurs for images of objects in the field, yet again movement is perceived in a direction appropriate to the direction of retinal image shift. To account for the apparent stability of the visual field when the eyes are turned voluntarily it has been suggested that an extraretinal signal regarding the eye movement is "taken into account" and acts to "null" the retinal image shift in perception. This suggestion has been proposed in several forms: (a) Helmholtz (1866) suggested that we take into account the "effort of will" exerted in making a voluntary movement. This has been recently labelled "outflow theory" (Whitteridge, 1964) to indicate that the theoretical extraretinal signal is presumed to arise centrally and flow outward to some other neural structure at which it and the retinal signal are coordinated prior to perception. (b) Sherrington (1918) suggested that we take into account signals from muscle spindles in the extraocular muscles. This has been recently labelled "inflow theory" (Whitteridge, 1964) to indicate that the theoretical extraretinal signal is presumed to arise in a peripheral structure and flow inward toward a central neural structure at which it is coordinated with the retinal signal. (c) Hering (1942) has suggested that "shifts of attention" are adequate to explain the appearance of stability when we turn from one fixation target to another and that no signal regarding eye position is necessary. While Helmholtz's "effort of will" is an operation leading to efferent motor activity, Hering's "attention" appears to be an operation leading to some kind of selective processing of afferent stimulation. Although such a distinction appears clear enough in principle, separating them experimentally is another matter. In any case, since a "shift of attention" would be a process originating centrally it would be appropriate to classify it as an "outflow theory." However, in this report we shall have nothing further to say regarding Hering's theory and in the discussion which follows we shall restrict the term "outflow theory" to the Helmholtzian view.

While the observations described above suggest the need for an extraretinal signal[3] to account for our perception of direction when voluntary saccades occur, they do not establish its necessity. The specific temporal and spatial characteristics of the retinal image displacement when a saccade occurs cannot be ruled out a priori as being solely responsible for the appearance of stability. Since neither a push of the eyeball by an external object nor a physical movement of the entire visual field as normally produced is likely to reproduce the temporal and spatial characteristics of the retinal image shift during a saccade, the appearance of movement under these two conditions is not critical to the question of the need for an extraretinal signal. More critical in this regard is the observation that when the eye is immobilized and voluntary eye movements attempted, motion of the visual field in the direction of the attempted ocular rotation is perceived although no motion of the retinal image has occurred. This observation, since corroborated (Kornmuller, 1931) is quoted by Helmholtz (1866) who considered it of central importance for his outflow theory. In addition, it has recently been shown that involuntary movements of the eye in the dark which occur while a S attempts to maintain a prior fixation position do not systematically influence the report of visual direction for a subsequent test flash except insofar as the eye movements affect the retinal location stimulated by the flash (Matin, Pearce, Matin, & Kibler, 1966). Analysis of these reports of visual direction indicate that the eye's position is not systematically taken into account. However, if signals from the extraocular muscles were relevant to the reports of direction they should be as useful when the movements are involuntary as when they are voluntary. The fact that they are not raises serious objections to inflow theory. Several other serious objections to inflow theory arise from the findings in the present series of experiments and will be discussed later. Aside

from the existence of muscle spindles in extraocular muscle (Whitteridge, 1964) there does not appear to be any evidence which might support the theoretical possibility of "inflowing feedback."

Despite the potential significance of an extraretinal signal for the visual perception of direction, however, none of the three theories have been developed beyond the roughest first order level and very little work has been done in subjecting them to a critical examination. Indeed there is a *prima facie* reasonableness about them which seems to have inhibited the study of some very easily observed phenomena which should lead us to question the generality of use of an extraretinal signal in the visual perception of direction: (a) Although the present authors have never seen it reported, it is possible to observe movement of the visual field as a consequence of a voluntary shift of fixation with the normal eye. This is most easily observed when viewing a dimly lit field. For example, looking at a small moonlit window covered by nearly-closed venetian blinds from inside a darkened room, one can see the blinds move as fixation is shifted from one side of the blind to the other; dimly perceived contours elsewhere in the room can also be seen to shift in a direction opposite to the eye movement. (b) If one shifts fixation from one point to another while viewing a visual field that contains a small intermittent light source (for example, a neon indicator light powered by 60 cps ac), under some conditions momentary changes in position of the pulsed light will be perceived consequent to the shifts of fixation. A similar result will appear if one shifts fixation across a narrow slit through which a portion of a moving picture is viewed–here distortions of the picture seen through the slit are apparent; a rapid single scan of a television picture also produces a distorted appearance.

The latter observations are not incompatible with some predictions that can be made from a more detailed theory of visual direction which includes an extraretinal signal. However, no studies have been reported in which the observations themselves are sufficiently extensive or quantitatively developed to provide convincing support for any theoretical viewpoint. Consider, in this regard, the observed motion resulting from attempts to move a paralyzed eye. The classical interpretation in terms of outflow theory maintains that this phenomenon involves an extraretinal process which is sufficient to explain the appearance of stability during voluntary saccades. One consequence of this interpretation is that the magnitude of apparent motion under paralysis must be equal to the magnitude of the attempted but unsuccessful movement of the eye. Based upon the essentially qualitative information which is available, we do not know whether this is so. Yet all of the above observations are important in that they point up the need for further investigation and direct our attention toward the problem with hypotheses in hand.

With these considerations in mind we performed several experiments (Matin & Pearce, 1965) in a situation designed to obtain quantitative information on the utilization of both retinal and extraretinal signals for stimulus flashes presented during voluntary saccades. In these experiments Ss reported the direction in which these flashes appeared relative to a target viewed and extinguished prior to the saccade; since the room was completely dark when the saccade began and ended, the presence of a stable visual context could not assist in maintaining stability of visual direction, and any extraretinal signal would have to do its work unaided. Under these circumstances, if there were no "compensation" due to an extraretinal process, it would be expected, for example, that a foveally located flash during the saccade would be reported to lie in the same direction as a fixation target viewed prior to the saccade. In contrast, if "compensation" occurred, a flash stimulating the fovea would appear displaced from the fixation target in the direction of the saccade by an amount equal to the extent of the saccade at the time the flash occurred. The latter result would support the existence of a mechanism that produces a "shift" in local sign[4] exactly equal and opposite to the motion of the image across the retina during a saccade, and could be responsible for the stability of visual direction in a normally illuminated environment. However, the data in the Matin and Pearce study showed two main characteristics which led to the conclusion that the utilization of an extraretinal signal in the visual perception of direction is not as general as has been heretofore thought: (a) Considerably more variability than might be expected from a mechanism in which there is a reliable coupling of retinal and extraretinal signals was present in the direction reports for a test flash triggered at a given target location when the eye was at a specific point in the saccade. (b) Under one condition the PSE (point of subjective equality) for the location of a target viewed prior to a saccade as measured with flashes delivered during a saccade was very close to the POE (point of objective equality). Under this condition, then, the shift of the eye appeared to be taken into account, suggesting the possibility that a mechanism is indeed available by which an extraretinal signal could effect a precisely-timed and continuous shift of local signs for the retinal signal. But for another condition the PSE and POE were separated by about 1 deg; for this condition both PSE and POE stimulated very nearly the same retinal locus. In our previous paper (Matin & Pearce, 1965) we pointed out that the mechanism suggested by the former result would have to be capable of providing a temporally synchronized treatment of both the retinal and extraretinal signals at some single neural locus, a particularly formidable feat since the latency of retinal response to a brief visual stimulus is a function of both the flash luminance and the state of adaptation of the eye while the neural delay for an extraretinal signal (regardless of its source) is not likely to be similarly affected. However, it is well-known that precise synchronization occurs for signals to the two ears as used in binaural localization, and the possibility of a synchronizing mechanism relating retinal and extraretinal signals seems worth considering; such a mechanism does, in fact, appear to be implied in previous statements of either inflow or outflow theory.

To elucidate these questions, we have studied the problem in more detail in a series of parametric experiments to be described in the present report and two succeeding reports (Matin, Matin, & Pola, 1969a, b). From an examination of the course of the shift before the saccade begins through the period following completion of the saccade we have found that the magnitude of the shift is time-locked to the occurrence of the saccade and is also related to the eye position. These two relations have been separated, and it is reasonably clear that the time-lock of shift magnitude is the more significant effect. In fact, the interpretation of the variation of shift magnitude to eye position is not yet clear; while it can be interpreted as due to an extraretinal signal that is nonlinearly related to eye position, an alternative and probably correct interpretation is possible in which the relation to eye position is a spurious one. Since the local sign shift is present for flashes delivered several hundred milliseconds before the saccade, however, the actual occurrence of the saccade itself is probably irrelevant to the occurrence of the local sign shift. The present paper describes four experiments in which a test flash presented during a saccade is used to measure the PSE for a fixation target viewed prior to the saccade. Two succeeding papers deal with experiments in which the test flash is presented before the beginning of the saccade (Matin, Matin, & Pola, 1969a) and after its completion (Matin, Matin, & Pola, 1969b). A fourth paper reports some experiments in which some of the temporal properties of a visual context are manipulated in the above situation (Matin, Matin, & Pola, 1969c).

APPARATUS AND PROCEDURE

Each of four experiments (Experiments 1, 2, 3, and 4) was performed with three Ss (EM, LM, and DP). A shorter version of Experiment 1 was also performed with a fourth S (JP). All of the experiments were conducted in a completely dark room with the S seated at a table with head position determined by a fitted mouthbite. Viewing was with the left eye; the right eye was covered by an eye patch.

On each trial of each experiment the S viewed a fixation target presented for 4 sec (see Figs. 1 and 2); 300 msec following its

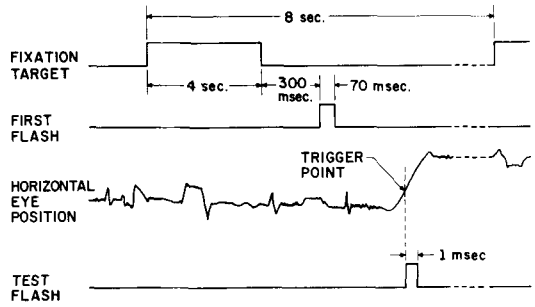

Fig. 1. Temporal sequence of events on a single trial in each experiment. The figure is schematic and not drawn to scale.

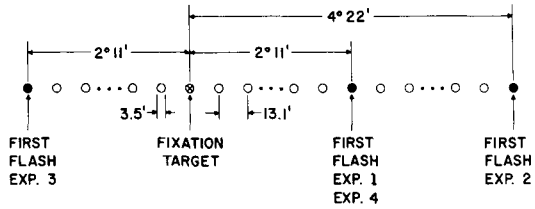

Fig. 2. Spatial locations of targets. Any target in the array could be used as a test flash; see text.

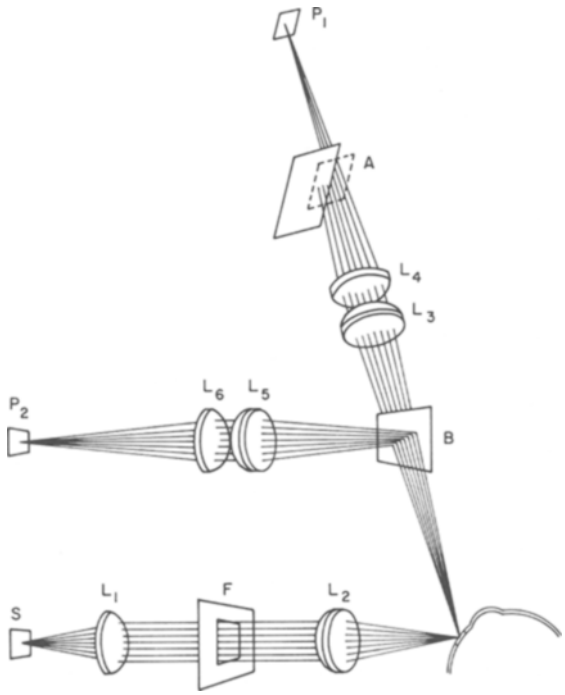

Fig. 3. Schematic diagram of optical apparatus for measuring horizontal eye movements. S is a source of infrared illumination; L_1 is a collimating lens; an image of S is formed by L_2 at the mirror on the contact lens and also at the photocells P_1 and P_2 by lenses L_3-L_4 and L_5-L_6 respectively. The square aperture in the field stop F is imaged in the plane of A which also contains an opaque surface with a vertical edge. As the eye rotates horizontally more or less of the beam reflected from the contact lens mirror reaches P_1. The photocell P_2 provides a reference through which spurious variations in the signal are monitored; corrections are made in the electronics before final readout. A filter for passing only wave-lengths in the infrared is placed next to F. B is a beamsplitter designed to transmit approximately 50% of the infrared illumination and reflect the remainder.

termination a 70 msec flash ("first flash") was presented at a predesignated location horizontally displaced from the location of the fixation target. (Different locations were used for the first flash in different experiments. However, in a given experiment this location was fixed.) Until the S saw the first flash he attempted to keep his eye in the original fixation position. As soon as he saw the first flash he turned his eye to it; since his reaction time for the saccade as measured from the onset of the first flash was always longer than the duration of the first flash, the first flash was over before the saccade was begun. When the eye reached a predetermined point in the saccade ("trigger point") a 1-msec flash ("test flash") was triggered by an electrical signal taken from the system monitoring eye position. The main independent variable of each experiment was systematic variation of the trigger point. At each trigger point the location of the test flash was randomly varied from trial to trial through a set of locations in a horizontal array that had been determined to lie in the neighborhood of the PSE during preliminary testing. On each trial the S reported whether the test flash appeared to lie to the left, to the right, or at the same location as the previously-viewed fixation target; if he reported "same" a second (forced choice) report of either "left" or "right" was given. The report was made by manual selection of a position on a three-position switch.

The stimulus array used for presenting the fixation target and test flashes consisted of a horizontal series of circular targets 3.5 min of arc in diameter viewed at a distance of 128 in. (see Fig. 2). Adjacent targets were separated by 13.1 min of arc and the array was distributed along a horizontal line that was perpendicular to the fixation axis when the S viewed the member of the array used as the fixation target. Each target was constructed of an electronically-controlled neon glow discharge lamp, Dialite No. 38H, in front of which was a diffusing disk and a mask that provided a homogeneous circular disk of orange-red light. The luminance of the fixation target was 4.5 ft-L; all flashes appeared at a brightness equal to that of the fixation target. Ultraviolet radiation invisible to the Ss shone on the lamps continuously to provide stable flashing behavior (Matin, 1964b).

Horizontal eye movements were continuously monitored during each experimental session by a contact-lens technique described in detail elsewhere (Matin, 1964a; Matin & Pearce, 1964). The measuring system used an invisible (infrared) beam incident upon a plane circular mirror embedded in the temporal margin of a scleral contact lens which was worn on the S's left eye (see Fig. 3). As the eye rotated horizontally, more or less of the energy in the beam reflected from the contact-lens mirror passed a vertical straight edge and reached a photocell; the change in energy was linearly proportional to the angle of rotation. The output of this photocell was corrected for spurious variations through the use of a reference beam and photocell which monitored the total energy reflected from the contact-lens mirror. The system measured horizontal eye movements independently of vertical and torsional movements although in this study only horizontal movements were recorded. Since the field stop in the incident beam and its image at the straight edge were at optical infinity with respect to the contact-lens mirror the system was insensitive to translations of the eye.

The current output of the photocells was linearly proportional to the radiant energy incident upon them. The current from photocell P_1 (see Figs. 3 and 4) was corrected for spurious variations as monitored by P_2, converted to a voltage and amplified. This final voltage was then proportional to the horizontal angular position of the eye. When a given trigger point was employed, this voltage was adjusted prior to experimentation to be at zero with the eye fixating a steadily illuminated target at this trigger point. Ocular positions to the left and right of the trigger point then resulted in negative and positive voltages, respectively, with magnitudes which increased linearly with ocular deviation from the given trigger point. The precise adjustment of a voltage zero at the trigger point was accomplished with the aid of monitoring of the voltage with an electronic integrator over a duration of from 15 sec to 1 min. This zero was checked frequently during an experimental session and adjusted when necessary. Setting errors of more than 2 min of arc were rarely

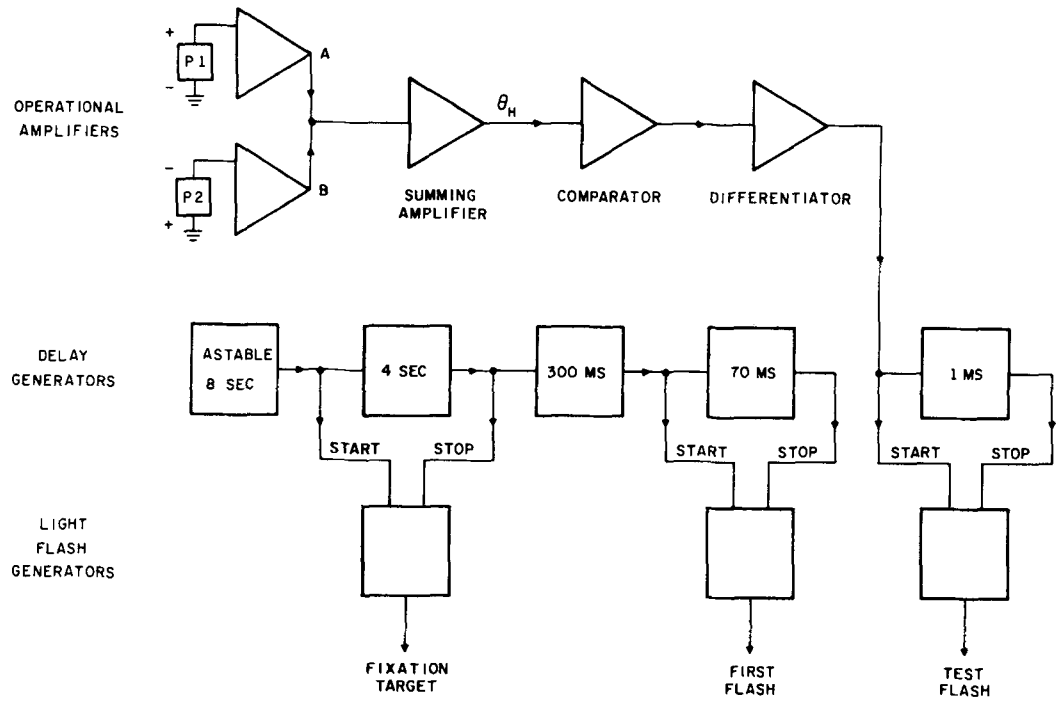

Fig. 4. Schematic of essential portions of electronic circuitry. See text for description.

observed and errors of more than 3 min of arc were never observed.

The voltage measuring eye position was fed to a comparator whose output was a fixed positive voltage when the eye was on the fixation target side of the trigger point and a fixed negative voltage when the eye was on the other side of the trigger point. As the eye traversed the trigger point during a saccade, the comparator's output underwent a negative voltage step which was inverted and differentiated into a sharp positive pulse. This positive pulse started a delay generator set for 1 msec and simultaneously turned on the test flash; the delay generator turned off the test flash 1 msec later. The delay between the eye's crossing of the trigger point and the turning on of the test flash was less than 3 μsec.[5]

The transduction of photocell current to a voltage, amplification, comparison, integration, differentiation and pulse shaping were performed by chopper-stabilized operational amplifiers (Philbrick SP656 or SK2-V). All stimulus timing was performed by phantastron delay generators (reliability better than .5%) that switched bistable multivibrators; the latter drove current generators providing the current to the glow discharge lamps. Calibration of durations was performed with the aid of a Beckman counter-timer 6144. Continuous records of eye movements, stimulus sequence and the S's psychophysical response were obtained on a Honeywell Visicorder 1508.

Each panel of Fig. 5 contains a record of a saccade and a 1 msec light flash triggered by it. Successive panels show the flash triggered at different points in the saccade.

Experiment 1

The first flash was centered 2 deg 11 min to the right of the fixation target. The test flash was triggered when the eye crossed one of five possible points in the saccade. The trigger points used

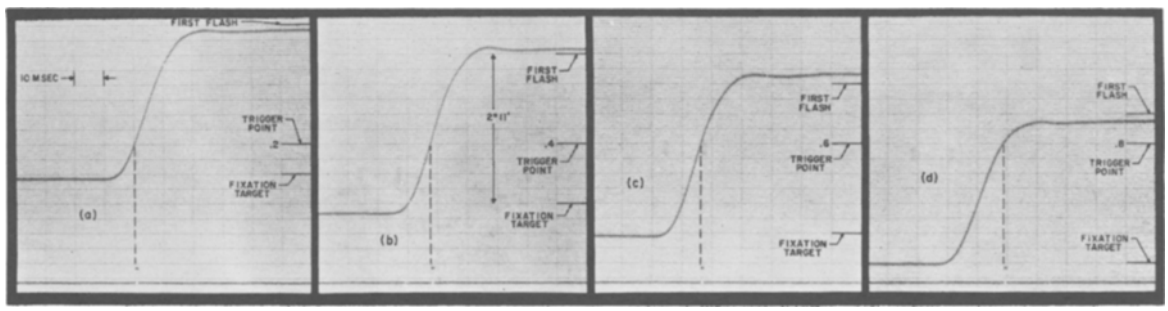

Fig. 5. Test flash triggered at four different trigger point locations during saccade. In each panel the upper trace is a recording of a saccade from the fixation target to a target located 2 deg 11 min to the right of the fixation target (upward in the figure refers to a rightward eye movement). The lower trace in each panel is a monitor from the flash generator controlling the test flash; a 1 msec test flash is indicated by the rectangular pulse directly under the dashed vertical line. FIXATION TARGET and FIRST FLASH respectively in each panel indicate average positions on ordinate when eye was continuously fixating each of the two targets steadily illuminated (measure obtained before recording). TRIGGER POINT (at the same height in each panel) indicates that eye position at which the summing amplifier (Fig. 4) output voltage is zero. To arrange for the trigger point to be at different proportions of the required saccade distance, the summing amplifier was biased differently, thus translating the relation of eye position to voltage output (and so translating the relation of eye position to height on the figure). Panels (a), (b), (c), and (d) show the flash triggered at .2, .4, .6, and .8 respectively of the distance between the fixation target and first flash location. For purposes of obtaining recordings for this figure fixation target and target at location of first flash were both illuminated continuously. S was EM. Time lines are separated by 10 msec intervals.

Table 1
PSEs and JNDs for all Ss in all Experiments

Exp.	First Flash Location	Trigger Point	Subjects EM PSE	EM JND	DP PSE	DP JND	LM PSE	LM JND	JP PSE	JP JND
		26.2′R	10.8′R	17.9	16.4′L	16.5′	4.4′L	10.2′		
		39.3′R							34.0′L	17.6′
1	131′R	52.4′R	37.8′R	10.8	8.9′R	20.7′	1.4′R	12.6′		
		65.5′R	51.1′R	12.6	17.4′R	23.5′	20.7′R	46.9′		
		78.6′R	61.9′R	12.4	30.5′R	17.2′	7.7′R	19.4′	2.0′L	12.0′
		104.8′R	81.3′R	13.0	53.2′R	21.0′	12.7′R	27.3′		
		26.2′R	11.6′R	13.4′			12.0′L	10.0′		
		52.4′R	34.0′R	24.4′	4.3′R	32.7′	7.4′L	16.5′		
2	262′R	104.8′R	95.8′R	17.5′	62.5′R	37.2′	16.0′R	25.2′		
		157.2′R	134.0′R	13.8′	122.2′R	49.0′	19.5′R	22.5′		
		209.6′R	175.2′R	18.9′			51.2′R	31.1′		
		26.2′L	4.0′L	40.4′			10.1′L	16.6′		
		39.3′L			30.5′L	22.6′				
		52.4′L	19.5′L	17.7′			29.3′L	12.7′		
3	131′L	65.5′L	41.1′L	23.9′	59.8′L	14.9′	37.5′L	25.0′		
		78.6′L	45.4′L	62.0′			47.3′L	17.4′		
		91.7′L			88.7′L	17.7′				
		104.8′L	81.2′L	49.4′			36.8′L	35.5′		
4	131′R	26.2′R	7.5′R	18.7′	10.8′L	17.9′	18.8′L	13.6′		
		52.4′R	34.1′R	12.9′	14.9′R	18.6′	9.6′L	17.5′		

were located at .2, .4, .5, .6 and .8 of the distance between the fixation target and first flash locations (26.2, 52.4, 65.5, 78.6 and 104.8 min to the right of the fixation target respectively). The entire experiment was run in five sessions. Three different trigger points were used in each session. Each session consisted of 90 trials, first 30 at one trigger point followed by 30 at a second trigger point, then 30 at a third trigger point. Eight sec following the beginning of one trial a second trial began with the onset of the fixation target. The 30 trials at each trigger point involved the presentation of three differently-located test flashes in a series of 10 randomized blocks. The five trigger points were ordered among sessions in an incompletely balanced design with the restrictions that (a) each trigger point appeared at least once in the same session with every other trigger point and (b) each trigger point appeared once each in the first, second and third position in a session; this ordering was different for each S. The three test stimuli used at each trigger point were chosen from preliminary work to lie within the uncertainty range. Thus, the total data at each trigger point for the entire experiment consisted of responses on 30 trials to each of three differently-located test flashes.

JP was tested at only two trigger points located at .3 and .6 of the distance between fixation target and first flash (39.3 min and 78.6 min to the right of the fixation target respectively). These measurements were made as part of an experiment in which the other conditions involved flash presentation following the saccade; the latter conditions are detailed in a subsequent report (Matin, Matin, & Pola, 1969c).

Experiment 2

The procedure in this experiment was identical to the first experiment with the exception that the first flash was located at 4 deg 22 min to the right of the fixation target. Five trigger points were used for EM and LM. These points were at .1, .2, .4, .6 and .8 of the distance between the locations of the fixation target and the first flash (26.2, 52.4, 104.8, 157.2, and 209.6 min to the right of the fixation target respectively). DP was tested at trigger points .2, .4, and .6 of the distance between fixation and first flash locations.

Experiment 3

The procedure was identical to the one employed in the first experiment with the exception that the first flash was located 2 deg 11 min to the left of the fixation target. In this case, for Ss EM and LM a test flash was triggered during a saccade when the eye was at either .2, .4, .5, .6, or .8 of the distance between fixation and flash target locations; DP was tested at trigger points placed either .3, .5, or .7 of the distance between fixation and first flash locations.

Experiment 4

In the fourth experiment the first flash was located at 2 deg 11 min to the right of the fixation target as in Experiment 1. Here, however, only two trigger points were used. These were located at .2 and .4 of the distance between fixation and first flash target locations. In the three previous experiments during each session an entire block of 30 trials was obtained at one trigger point before going on to another trigger point. In this experiment trigger points and test flash locations were jointly randomized in blocks so that each of the test flash-trigger point combinations was presented once before a given one was repeated again. For Ss EM and DP three test flash locations were employed at each of the two trigger points; for LM four test flash locations were employed at each trigger point. The specific stimuli used are shown on the abscissa in Fig. 7a.

RESULTS

A. The Main Trends

The proportion of reports that the test flash during the saccade appeared to lie to the right of the previously-viewed fixation target was calculated for each of the test flashes used in conjunction with a given trigger point (on trials for which a "same" response was given, the second, forced-choice response was used). This was done separately for each S at each trigger point in each of the experiments. These proportions were used to calculate PSEs and JNDs (just noticeable differences)—the 50% point of the normal ogive fitted according to a maximum likelihood criterion[6] and the standard deviation of the underlying density, respectively. The PSEs and JNDs are shown for all four Ss and all four experiments in Table 1.

The main regularities in the data are present in the pattern of PSE values. These are also plotted in Fig. 6 (ordinate) against the trigger point (abscissa) for each S in each experiment. Zero on the ordinate corresponds to the location of the fixation target itself (compensation locus); values above and below zero correspond to

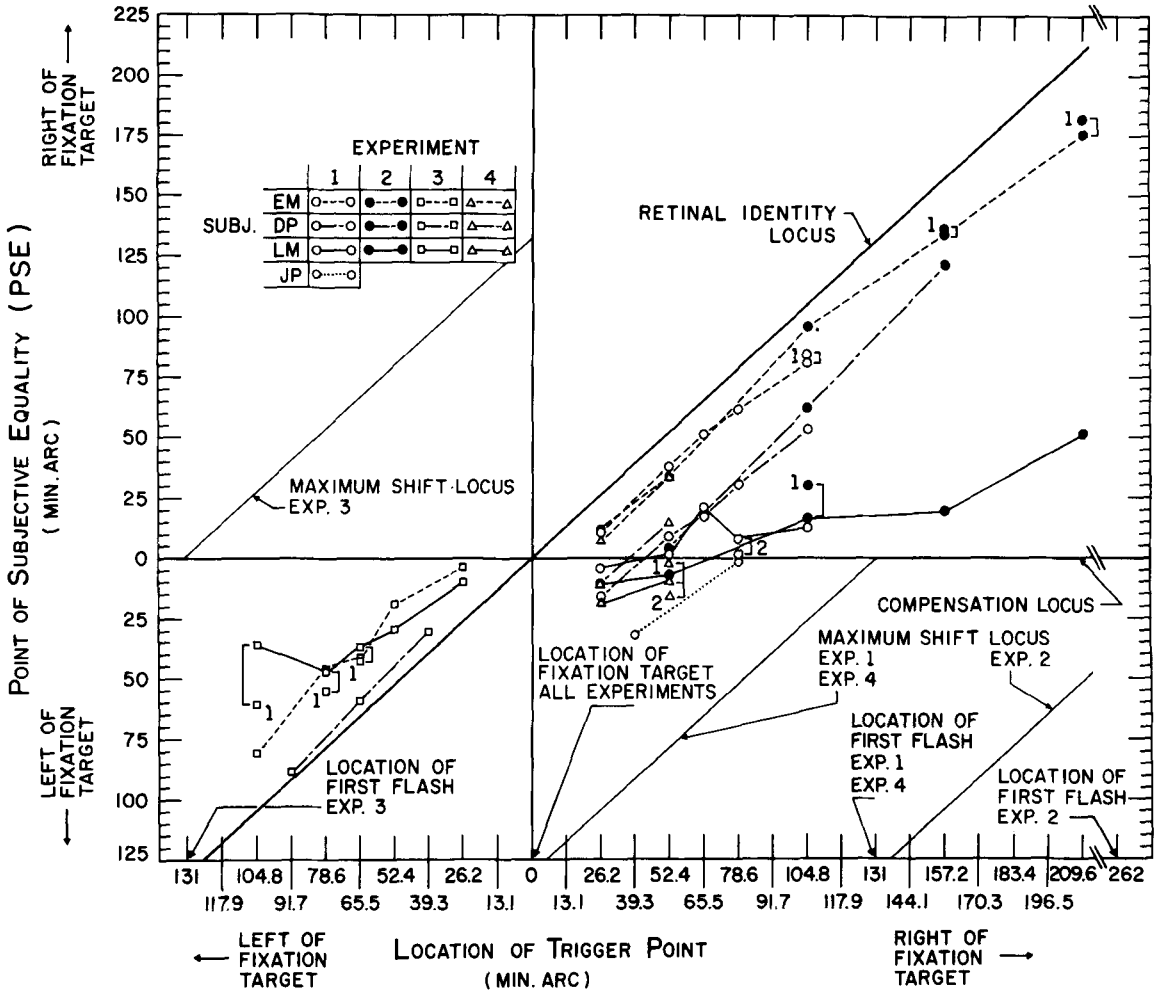

Fig. 6. PSE as a function of location of trigger point for each S in each experiment. When a PSE calculated for "one saccade" data alone or "two saccade" data alone is shown the number 1 or 2 is written next to the data point and a bracket is drawn connecting it with the corresponding PSE for the total data. It should be noted that total data PSEs for LM in Experiment 2 at trigger points of 157.2 min and 209.6 min, and in Experiment 1 at a trigger point of 104.8 min are based mainly on "two saccade" data (see Table 2). The compensation locus is the theoretical curve for which the PSE would be at the fixation target at all trigger points. The retinal identity locus is the theoretical curve for which the PSE would be at the central fovea (same retinal locus at which fixation target was imaged) at all trigger points. The maximum shift locus for each experiment is the theoretical curve for which the PSE would be displaced from the trigger point (in a "compensatory" direction) by an amount equal to the distance between fixation target and first flash.

PSEs to the right and left of the fixation target respectively. The main diagonal drawn through the origin (retinal identity locus) is the locus of points for which the PSE would be at the trigger point (for a PSE to fall at a point on the diagonal means that for the trigger point represented by the abscissa value, the test flash reported as being to the right of the fixation target 50% of the time struck the central fovea); values above and below this diagonal refer respectively to PSEs to the right and left of where the fovea was pointing at the moment the test flash was presented. The other diagonals (maximum shift loci) correspond to the loci for which the PSE would be displaced on the retina (on the side of the fixation target locus opposite to the first flash locus) by an amount equal to the distance between fixation target and first flash.

The outstanding features in Fig. 6 may be summarized in five points: (a) All of the PSEs for all four Ss in Experiments 1, 2, and 4 fell below the main diagonal; all PSEs for Experiment 3 fell above the main diagonal. In addition, most of the PSEs fell between the pair of scissors formed by the main diagonal and the compensation locus. (b) The PSEs for EM and DP in each of the experiments follows a clear and similar pattern. For each of these two Ss in each experiment the PSE varies in approximately linear fashion with variation in the trigger point. The best-fitting straight line through the data for each experiment on EM and DP does not differ significantly in slope from the slope of the main diagonal (for a discussion of possible departures of these data from linearity, see below). (c) For either EM or DP the data from Experiments 1, 2, and 4 could reasonably be considered as arising from the same linear function. Thus, the PSE for a trigger point that was at a fixed distance from the fixation target did not, in general, seem to be systematically affected by the length of the saccade (compare, for example, JNDs for the trigger points at 26.2 min R in Experiments 1 and 2). (d) The trend in the data of EM and DP is such that PSEs for trigger points early in the saccade are relatively close to the location of the fixation target while PSEs later in the saccade are considerably further from the fixation target; to a much less marked degree this is also true of the data for LM. (e) While EM's data in Experiments 1, 2, and 4 (saccade to the right) falls closer to the main diagonal than do DP's data, the reverse is true in Experiment 3 (saccade to the left).

LM's PSEs deviate from those of EM and DP in several ways: (a) Marked departures from linearity are apparent in Experiments 1, 2, and 3; LM's data are much more irregular. (b) The linear trend that is present in all four experiments on LM departs markedly

Table 2

Frequencies of left (L) and right (R) responses for LM in each experiment. Data are shown separately for trials in which the test flash was triggered by the first saccade (1 sac.) and trials in which the test flash was triggered on the second saccade (2 sac.). At each trigger point in Experiments 1 and 2 N = 30; in Experiment 3, N = 26; and in Experiment 4, N = 68. Most of the values in this table are slightly less than these numbers, however, since (a) the trials in which the flash was triggered on the third saccade are not shown, and (b) some trials were simply missed.

Trigger Point	Test Flash Location	1 sac. L	1 sac. R	2 sac. L	2 sac. R
		Experiment 1			
	13.1′L	25	4	0	1
26.2′R	0	8	19	0	2
	13.1′R	2	26	0	1
	13.1′L	22	0	4	4
52.4′R	0	16	3	1	8
	13.1′R	4	17	1	8
	13.1′	20	0	2	4
65.5′R	0	20	1	0	5
	13.1′R	17	3	0	9
	13.1′L	8	0	16	4
78.6′R	0	8	0	9	9
	13.1′R	5	2	6	14
	13.1′L	1	0	17	3
104.8′R	0	4	0	14	9
	13.1′R	4	0	6	10
		Experiment 2			
	13.1′L	18	8	0	0
26.2′R	0	8	19	0	0
	13.1′R	4	21	0	0
	13.1′L	18	8	0	1
52.4′R	0	8	19	0	2
	13.1′R	4	21	0	3
	13.1′R	16	3	0	11
104.8′R	26.2′R	10	9	0	9
	39.3′R	6	12	0	10
	13.1′R	3	0	13	8
157.2′R	26.2′R	1	1	12	14
	39.3′R	0	0	5	24
	26.2′R	2	0	19	4
209.6′R	52.4′R	0	0	12	14
	78.6′R	0	2	4	16
		Experiment 3			
	26.2′L	19	4	0	0
26.2′L	13.1′L	14	10	0	0
	0	6	18	0	0
	13.1′R	2	22	0	0
	0	0	24	0	0
52.4′L	13.1′L	1	21	1	1
	26.2′L	9	12	3	0
	39.3′L	13	6	3	0
	13.1′L	0	20	5	1
65.5′L	26.2′L	6	14	3	3
	39.3′L	9	14	2	0
	52.4′L	15	6	6	0
	39.3′L	3	14	6	1
78.6′L	52.4′L	3	12	9	0
	65.5′L	18	2	4	0
	78.6′L	15	1	7	0

Table 2 (cont.)

Trigger Point	Test Flash Location	1 sac. L	1 sac. R	2 sac. L	2 sac. R
	26.2′L	0	6	8	10
104.8′L	39.3′L	1	4	13	6
	52.4′L	1	2	16	5
	65.5′L	4	3	14	3
		Experiment 4			
	26.2′L	45	17	0	2
26.2′R	13.1′L	22	43	0	2
	0	4	55	1	3
	13.1′R	1	65	0	1
	26.2′L	23	0	26	9
52.4′R	13.1′L	26	9	14	19
	0	23	20	3	19
	13.1′R	2	38	1	24

from the slope of the main diagonal in Fig. 6. (c) This departure brings much of LM's data for trigger points toward the end of the saccade closer to the horizontal line through the zero ordinate than the data of either EM or DP.

The pattern of JNDs in Table 1 is notable for its paucity of systematic trends. No systematic relation is apparent between trigger point location and magnitude of JND either overall or in any single experiment; only LM's JNDs show signs of being smallest at the first two trigger points in each experiment and larger for later trigger points. While DP's JNDs in Experiment 2 are uniformly larger than those in Experiment 1, a similar general trend for EM and LM is only barely visible at best; thus the JND for a trigger point that was at a fixed distance from the fixation target did not, in general, seem to be systematically affected by the length of the saccade (compare, for example, JNDs for the trigger point at 26.2 min R in Experiments 1 and 2). EM's JNDs in Experiments 1 and 2 are, in general, smaller than those of DP and LM; this is not so in Experiment 3, however, where the saccade was to the left.

B. One-Saccade and Two-Saccade Trials

The differences between EM's and DP's PSEs on the one hand and LM's on the other hand can be related to strikingly different patterns of saccadic behavior. EM's saccades typically (but not invariably) overshot the position of the first flash frequently by as much as .5 deg to 1 deg; DP's saccades were fairly accurate (as were JP's), sometimes undershooting and sometimes overshooting by small amounts. LM's responses were peculiar in two ways which had significant consequences: (a) The initial saccade typically undershot the position of the first flash by a wide margin, ending about halfway between the fixation target and first flash locations. As a result there were numerous trials on which no test flash was presented; most of these trials were repeated although a few were missed. These mistrials occurred most frequently for trigger points furthest from the fixation target. (b) On many trials in which the initial saccade was short of the trigger point a second saccade followed during which the eye crossed the trigger point and resulted in the presentation of a test flash. (On rare occasions a third saccade would cross the trigger point.)[7] These successive saccades frequently followed each other at intervals of less than 100 msec. LM was unaware that the eye movement ever consisted of any more than a single smooth saccade. Occasionally EM and DP also triggered flashes on the second saccade.

Although the experiments were not designed to gather enough data to obtain systematic information separately for the "one-saccade" and "two-saccade" trials, the difference between their respective psychophysical response distributions is so marked that even with the limited data at hand some separate treatment is possible. In Table 2 the psychophysical responses for LM are

Table 3

Frequencies of actual left (L) and right (R) responses summed over all trigger points for "two-saccade" trials (2 sac.). The values in the 1 sac. WTD columns were obtained as follows: the frequency of "left" and "right" responses on "one-saccade" trials at each trigger-point-test-flash combination was weighted by the frequency of "two-saccade" trials at that combination; the resulting "left" and "right" weighted frequencies were summed over all combinations; the resultant values were scaled so that the total left (L) and right (R) values in the 1 sac. WTD column equalled the sum of the L + R responses on two-saccade trials.

Ss	Exp.	1 Sac. WTD		2 Sac.	
		L	R	L	R
EM	1	8.5	13.5	3	19
	2	17.7	37.3	3	52
	3	1.1	3.9	4	1
	4	0.8	1.2	0	2
DP	1	2.5	5.9	0	8
LM	1	135.0	32.0	76	91
	2	86.4	84.6	65	116
	3	50.4	79.6	100	30
	4	69.0	55.0	45	79

shown separately for "one-saccade" trials and "two-saccade" trials for each trigger point in each of the four experiments. For virtually every test flash location at each trigger point in each of Experiments 1, 2, and 4 at which there is some data for both types of trial, the proportion of "left" responses is greater on "one-saccade" trials than on "two-saccade" trials; in Experiment 3 where the saccade is to the left, the proportion of "right" responses is greater on "one-saccade" trials. In general, then, LM tends to report a test flash as lying toward the first-flash side of the fixation target more frequently on "two-saccade" trials.

Neither EM or DP had enough "two-saccade" trials to make a display similar to Table 2 feasible. However, Table 3 shows the response distribution for EM's "two-saccade" trials in each experiment summed over all trigger points and test flash locations. For comparison a response distribution for "one-saccade" trials is shown; this was obtained in each experiment by weighting the distributions at different trigger-point-test-flash-location combinations in terms of the frequency of "two-saccade" trials at those combinations and scaling the result to equal the same total number as the total number of "two-saccade" trials in that experiment. A similar comparison is shown in Table 3 for DP in Experiment 1; DP had only one "two-saccade" trial each in Experiments 2 and 4 (the response on both trials was "right") and none in Experiment 3. It is clear from Table 3 that the difference in response distributions for "one-saccade" and "two-saccade" trials shown for LM in Table 2 also holds for EM and DP.

In several cases there was enough data available to calculate separate PSEs and JNDs for either "one-saccade" trials or "two-saccade" trials. These PSEs are shown in Fig. 6; they are marked with a "1" or a "2" respectively and bracketed with the corresponding PSE for the total data. The "one-saccade" PSEs all lie closer to the main diagonal and the "two-saccade" PSEs all lie further from the main diagonal than do the PSEs for the total data. In Experiment 3 on LM the "one-saccade" PSEs at trigger points 65.5 min L, 78.6 min L and 104.8 min L in conjunction with the PSEs for the total data at the 26.2 min L and 52.4 min L trigger points form a set which are not unreasonably fitted by a straight line parallel to the main diagonal. (Note the paucity of "one-saccade" data for trigger at 104.8 min L.) It should be noted (see Table 2) that most of the data at the more distant trigger points for LM in Experiments 1 and 2 are the result of "two-saccade" trials. Hence "one-saccade" PSEs cannot be reasonably calculated from them and "two-saccade" PSEs do not then differ much from PSEs for the total data.

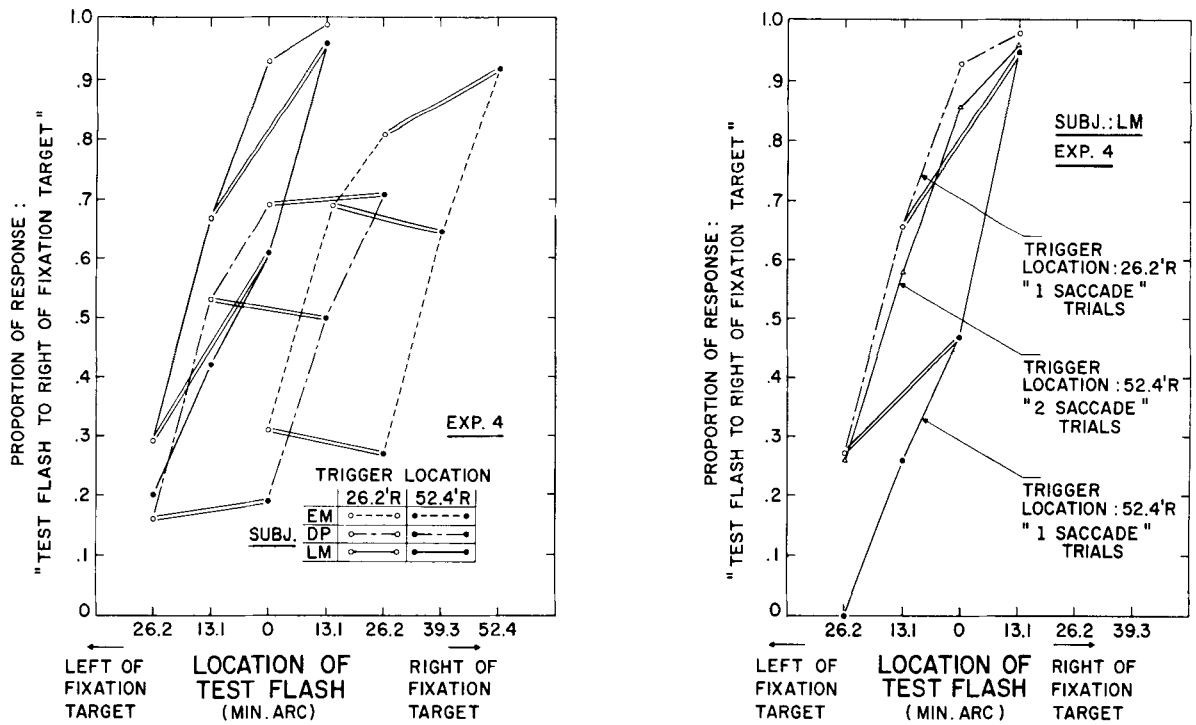

Fig. 7(a). Data of Experiment 4 plotted as the proportion of reports that the test flash lay to the right of the fixation target as a function of test flash location. The data points for a given S at a single trigger point are connected by a single line (coded by S as solid, broken, or dashed). Each pair of data points connected by double lines were for test flashes presented at the same retinal locus; the two members of such a pair were from differently located test targets presented when the eye was at two different trigger points. (b) Data of LM from Experiment 4 separated into "one-saccade" and "two-saccade" trials. Lines connecting data points have same meaning as in (a).

In Fig. 7a the data for Experiment 4 are plotted as the proportion of reports that the test flash was to the right of the fixation target as a function of test flash location. For each S the points generated at each of the two trigger points are separately connected, yielding two ogives for each S and six in all. For each S double lines connect the two points, one generated at each trigger point for which the two differently-located test flashes struck the *same retinal locus.* Differences in ordinate values between these latter pairs of points were tested separately for each S by the χ^2 test for correlated proportions (this test was possible since the experiment was run in a randomized block design). No significant differences were obtained for Ss EM or DP ($p > .20$), nor were the differences for either of these two Ss significant when χ^2s were added over the three contingency tables ($p > .30$); the differences were significant at the .01 level of confidence for each of the two analogous possible comparisons for LM.

The difference between proportions at the two trigger points for EM using the test flash at 26.2 min R, for DP using the test flash at 0 min, and for LM using the test flashes at 13.1 min L and at 0 min are each significant at the .01 level of confidence; the differences between proportions at the two trigger points for LM using the test flashes at 26.2 min L and 13.1 min R were not significant ($p > .10$); adding χ^2s over the four contingency tables for LM yielded a difference significant at the .01 level of confidence.[8]

In Fig. 7b the data for LM in Experiment 4 are plotted for "one-saccade" and "two-saccade" trials separately; not enough data were available to do this for "two-saccade" trials when the trigger point was at 26.2 min R, however (see Table 2). The vertical magnitude of the two double lines indicate the magnitude of the differences in proportions between the two pairs of different test flash locations whose flashes strike the same retinal locus at the two different trigger points on "one-saccade" trials; these differences are significant at the .01 level.

DISCUSSION

A. Direction of PSE Shift from the Retinal Identity Locus

Although the analysis of the data into "one-saccade" and "two-saccade" trials has brought the results of LM more closely into line with those of EM and DP, the differences remain sufficiently great so as to require some separate discussion. However, in spite of these differences one characteristic of the data is common to the four Ss: *Every one of the 49 PSEs in Table 1 as well as every PSE that could be calculated for "one-saccade" trials or "two-saccade" trials fell on the same side of the trigger point as did the fixation target.* Four alternatives will be separately considered as a basis for this result. Two of these alternatives derive from the possibility of contact lens slippage; these can be ruled out with almost complete certainty. A third alternative, that some dioptric change occurs inside the eye, seems extremely unlikely. The fourth alternative, that a genuine shift of local signs has occurred as measured by flashed stimuli presented during the saccade, appears to provide the only reasonable interpretation for the direction in which the PSEs deviate from the trigger point.

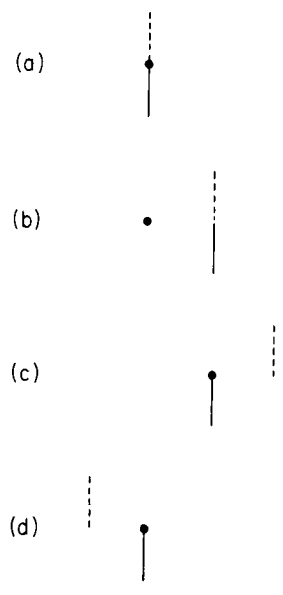

Fig. 8. Unbroken line is the direction of view in the frontoparallel plane of a normally presented target; broken line is the direction of view in the frontoparallel plane of a "retinally stabilized" target. Direction of fixation is indicated by •. (a) Original view before wedge prism or lens push is introduced. (b) View after base-out wedge prism is introduced but *before* S has regained fixation at top of nonstabilized target. (c) View after base-out wedge prism is introduced *after* S has regained fixation. (d) View (without base-out wedge prism) produced by lens push when it is assumed that push results in contact lens rotation but no prismatic deviation.

1. If the contact lens did not follow the eye accurately during the saccade, the test flashes would be triggered at points whose location we have measured inaccurately. Such a lack of fidelity would have led to incorrect labelling of the values on the abscissa in Fig. 6. In order for this alone to account for the direction of the PSE shift from the main diagonal in Fig. 6, however, it would be necessary that the contact lens *lead* the eye in the direction of the saccade. Further, for Ss EM and DP it would be necessary that the contact lens lead the eye by about the same amount at each point in the saccade, and also that this magnitude of lead be about the same for saccades to the right that are about 2 deg 11 min and 4 deg 22 min in extent. Since ocular velocity varies systematically during a saccade over a large range, and is larger when the eye is at a given distance from the starting point during a larger saccade than during a smaller saccade, this would also require that such a contact lens lead not be a function of ocular velocity. While we can offer no direct evidence that the contact lens does not lead the eye during a saccade, it is not easy to think of a means by which this could occur at all; the additional requirements on the way in which the contact lens would have to lead the eye in order to account for the data make this explanation even more unlikely. If contact lens slippage did occur during the saccade, one would expect it to be such that the contact lens lagged in following the eye; if a lag producing inaccurate measurements of eye location were to be the sole mechanism responsible for shifts of PSE from the trigger point, the measured PSEs should have fallen on the other side of the main diagonal from the fixation target (Fig. 6)—this, however, *never* occurred.

2. Contact lens slippage during the saccade could also introduce dioptric changes. These changes might be due to either a change in the shape of the liquid lens between contact lens and eye or a change in the orientation of lens on the eye. Such a distortion might account for the direction of the PSE shift from the main diagonal in Fig. 6 if it resulted in the introduction of a prismatic deviation during the saccade equivalent to a base-out wedge prism when the (left) eye moved from left to right (or a base-in prism as the left eye moved from right to left). That such an effect could not be significant in the production of the PSE shift is shown by the following:

(a) In Fig. 8(a) is shown a situation in which the S wearing his contact lens viewed a normally-presented target (unbroken line) and a target retinally stabilized in the horizontal dimension (broken line); fixation was at the top of the normally-presented target. The • in each portion of Fig. 8 represents the direction in physical space toward which the fovea is pointed; in Fig. 8(b) is shown the view when a base-out wedge prism is introduced; the apparent direction of both stabilized and normal views are displaced to the right as a consequence of the change of direction of both beams by the prism; the eye in Fig. 8(b) has not yet turned to regain fixation and this is shown by the fact that the • is not displaced. With the introduction of the prism, the upper target is now, however, stabilized on a new part of the retina and so when the eye regains fixation at the top of the nonstabilized target by turning to the right [Fig. 8(c)] the apparent location of the stabilized target is displaced to the right.

Figure 8(d) shows the situation if the contact lens were somehow rotated to the left while the eye remained pointed in the same direction as in (a); it is assumed that no prism is introduced as a consequence of the lens rotation in this figure; thus the fovea still sees the top of the normally presented target. The result of such a lens rotation is to displace the relation of the stabilized image to the normal target on the retina by the angular magnitude of the lens rotation and the apparent relation of the stabilized to normal target would be as shown in Fig. 8(d).[9]

We have concluded earlier that a contact lens leading the eye during the saccade is an extremely unlikely event but that it was possible for the lens to lag the eye. The question at issue here then is whether such a lens lag could, by introducing prismatic distortion, produce the shift of the PSEs from the main diagonal in Fig. 6 (we have already pointed out that a lagging lens could not produce the PSE shifts alone by triggering test flashes during the saccade at inaccurately known locations). To test this question the S viewed the combination of stabilized and normal target as in Fig. 8(a); a lens lag for a saccade to the right is simulated by simply pushing the contact lens to the left on the eye.[10] (By pushing sufficiently hard it is possible to produce a change of the relation of the contact lens to the eye that can be seen by the experimenter viewing the S's eye directly.) If the effect of the lens push is simply to rotate the contact lens (and so the mirror on the contact lens) without introducing prismatic distortion, then the stabilized image should appear displaced to the left of the normally viewed target [as in Fig. 8(d)]. If the lens push also introduces a prismatic deviation in the direction that is appropriate to account for the PSE shifts for saccades to the right and the deviation magnitude is sufficient to account for the PSE shifts, then the stabilized image should appear displaced to the right of the normally viewed target [as in Fig. 8(c)]. The results were unequivocal: regardless of the magnitude of the lens push, the appearance was as in Fig. 8(d). This held over a range from where the apparent horizontal displacement to the left of the stabilized view was as small as could be introduced by pushing on the lens to apparent displacements of about 2 deg–the largest that could comfortably be introduced. [For sufficiently large lens pushes the entire field appeared to move to the right but the stabilized view always appeared to the left of the normal view as long as pressure was maintained on the lens (see also footnote 10).] *This result appears to rule out with fair certainty the possibility that a lens that lags during the saccade could be responsible for the direction of PSE displacement from the main diagonal in Fig. 6.*

(b) Although it is conceivable that the lens push did not really simulate any possible lens slippage during a saccade and so failed to produce a prismatic deviation, it is not very likely; the suction holding the lens to the eye as well as its tight fit on the limbus limits the number of degrees of freedom for possible slippage very markedly. However, it is also possible to perform a calculation which adds further support to the view that lens slippage was not involved in the direction of PSE shift from the main diagonal in Fig. 6: Using Prentice's Rule,[11] we calculate that about 4 mm of decentration would be needed to produce a prismatic deviation of 20 min through the combination of contact lens and liquid lens of largest power that was employed in the present experiments; decentrations of about 1 mm are as large as can be produced by a hard external push on the lens without discomfort–it is not possible that such decentrations occurred under the conditions of use in the present experiment (when a push on the lens introduces a 1 mm displacement visible to the experimenter as a change in the relation of the junction of iris and sclera to a mark on the contact lens, it requires a second or two for the lens to reseat itself following removal of the finger from the lens; this reseating involves a change visible to the experimenter. No such change is visible following voluntary saccades by the S.) While the calculation from Prentice's Rule assumes that the lens system translates in a plane perpendicular to the lens axis and also that it does not change shape, assumptions not necessarily valid if the contact lens does slip during a saccade, it does serve to indicate further that the order of magnitude of dioptric change required to account for the direction of PSE shift is incompatible with the magnitude of any change that might reasonably be expected to have occurred.

(c) While the above calculations and observations appear to rule out lens slippage or any of its consequences as playing a role in the direction in which measured PSEs deviate from the main diagonal in Fig. 6, even more convincing is the finding that the PSE shifts occur for flashes presented *before* the saccade begins (Matin, Matin, & Pola, 1969a).

3. Another locus of possible dioptric change during the saccade is the eyeball itself. The asymmetrical change of muscular tension exerted on the globe or a small displacement of the lens in the eye (not the contact lens) during the saccade could conceivably introduce a deviation of the rays from the test flash on their way through the eye (for example, using Prentice's Rule a lateral displacement of the lens inside the eye of 200 μ would produce a deviation of about 20 min at the retina). No direct evidence for such a change exists, however, and would indeed be very difficult to measure. Further, the finding (mentioned above) of PSE shifts prior to the beginning of the saccadic movement lessen considerably the likelihood of dioptric change being of primary relevance.

4. The three points above have been concerned with the fact that the PSEs never lie on the main diagonal or deviate from it to the first-flash side of the fixation target. The main diagonal provides the locus of points for the null hypothesis against which any shift in local signs must be tested. However, one could conceivably ask whether lens slippage and/or dioptric changes produced deviations of PSE from some other locus such as the "compensation" locus. Without detailing the argument further we can say that no reasonable concatenation of circumstances could provide support for such artifacts as lying behind the deviations from either the "compensation" or "maximum shift" loci.

In Fig. 6 the main diagonal has been labelled "retinal identity" locus to indicate that PSEs falling on it would be such that the retinal locus corresponding to the PSE as measured by test flashes would be identical to the mean retinal locus at which the fixation target was imaged; this identification of the main diagonal assumes that no significant contact lens slippage has contaminated our measurements. If we were to allow for the possibility of contact lens lag during the saccade a retinal identity locus would need to be above the one shown for Experiments 1, 2, and 4 (in upper right quadrant) and below the one shown for Experiment 3 (lower left quadrant). That such a lens lag probably did not contribute in any significant way to the present data is, however, indicated by the fact that in Experiments 1, 2, and 4 for Ss DP and LM at the trigger point 26.2 min to the right of the fixation target and in Experiments 2 and 4 for LM at the trigger point 52.4 min to the right of the fixation target, the PSEs were to the *left* of the fixation target; we can think of no concatenation of circumstances (either including lens lag or not) by which lens slippage could account for such a result; considerations regarding lens slippage here lead to the impossible prediction that no test flash could be triggered.

We have also labelled the zero ordinate as the "compensation" locus to indicate that a PSE on this locus is at the fixation point and that compensation for the displacement of flash from the mean retinal locus of the fixation target must have occurred; such an identification does not depend on any assumptions regarding contact lens slippage.

The discussion that has preceded has indicated that neither errors in our recording system nor dioptric changes in the eye or in the relation of the contact lens system to the eye provide a tenable basis for the direction in which the measured PSEs deviate from the "retinal identity" locus. We are thus left with only the possibility that this result involves some shift of local signs as measured by flashes presented during the saccade (i.e., a change in the relation between retinal locus and visual direction). The remainder of the discussion will be concerned with further considerations regarding the shift.

B. Relation to Classical Theories

For all Ss, the results of the present experiment are clearly not those predicted from the hypothesis that constancy of visual direction is maintained during voluntary saccades by an extraretinal signal regarding the position of the eye (constancy of visual direction would be represented by the "compensation locus" in Fig. 6). Rather, under reduced conditions of stimulation, where such a signal would be operating without the aid of a continuously present visual context, very gross deviations from constancy occur. But the results, in the light of the discussion in the previous section, have also led to the conclusion that the visual direction for a flash presented during a voluntary saccade does not bear the same relation to retinal locus of stimulation as does a fixation target viewed prior to the saccade; instead a shift of local signs occurs in the direction of compensation for the eye movement.[12] In the remainder of this discussion we shall consider some possible interpretations of this shift and the relation of our findings to some earlier theoretical viewpoints.

Although the classical "inflow" and "outflow" theories (see above) certainly seem reasonable on a first-order level, some important difficulties become apparent when we examine their implications in more detail. While these theories as originally presented provided suggestions regarding the source from which extraretinal signals guiding the local sign shift could arise, the nature of the mechanism by which retinal and extraretinal signals could be coordinated was not presented in sufficient detail to permit quantitative prediction of results in specific experiments. The theories seem to imply, however, that direction constancy would be maintained throughout the saccade. One development of these theories from which such a prediction would be made was indicated in a previous report (Matin & Pearce, 1965) and above in

the Introduction. While this development is our inference it appears to be clearly implied in descriptions of these theories.[1,3] This development says that a signal generated by the change in eye position ("inflow") or programmed by the intention to turn the eye ("outflow") is linear with ocular displacement and appropriately synchronized with the signal arising from successive portions of the retina as the image changes retinal locus during the saccade. Such a synchronization between signals from the two sources of information would need to occur prior to the entrance to the "final common path" within which the perception of direction is "finally stated." The consequence of such synchronization would be to shift local signs along a time course so that direction constancy would be maintained for retinal stimulation during the saccade; in the present experiments the PSE should then remain at the POE (point of objective equality—in this case the fixation target) regardless of trigger point; in Fig. 6, then, all of the data should fall on the compensation locus. This is clearly not the case. This failure of classical theories to account for the present data, however, should not be interpreted as a failure to find evidence supporting the operation of an extraretinal signal in the determination of local signs. Quite the contrary: The finding that all PSEs are on the fixation target side of the retinal identity locus (Fig. 6) is convincing support for the operation of such a signal. The failure in classical theories lies in the requirement of a special form of precise synchronization between retinal and extraretinal signals that yields direction constancy always. In addition to the failure of classical theories to account for the location of the PSEs the magnitude of the JNDs indicates considerable lack of precision in synchronization.

Quite apart from the data of the present experiments, it is worth reiterating that any theory which requires precise synchronization between the shift in a signal arising from stimulation of successive loci on the retina with a signal regarding change in eye position or the intention to turn the eye would have to become somewhat complex in order to deal with the fact that latency of a retinal response varies with both adaptation level and stimulus intensity. That is, unless we make the unlikely assumption that a signal regarding eye position or the intention to turn the eye varies with adaptation level and stimulus intensity, direction constancy would only be expected to hold for, at most, one particular set of adaptation and intensity conditions. Although we have no data available at present, it will be possible to study this question directly by varying adaptation level and intensity in experiments of the present type. In the absence of any

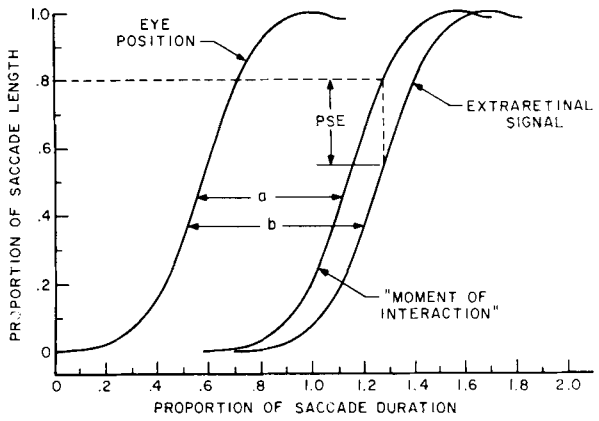

Fig. 9(a). Part of nomogram for calculation of values of PSE for first model extending classical theory ("latency mismatch" model). Time units on abscissa are in terms of proportions of saccade duration. (The actual values of time for the "moment of interaction" curve are a plus the values on the abscissa; for the extraretinal signal curve the values are b plus the values on the abscissa. Lateral separations between the curves were chosen arbitrarily and do not imply an estimate of the latencies a and b.) Units on ordinate refer to proportion of saccade length for all three functions; however, ordinate values for the extraretinal signal function are the negative of the values shown on the scale. The ordinate value of a point on the eye position function represents the distance of the fovea from the starting point of the saccade. An example of calculation of a PSE is indicated: The trigger point is assumed to be at .8 of the total saccade distance from the saccade's starting point. The interaction of retinal and extraretinal signals then occurs at the moment given by the abscissa value at the point of intersection between the horizontal line drawn at a height of .8 and the "moment of interaction" function. In addition, this ordinate value on the latter function can be taken to represent the position of the fovea when the flash is presented; points above and below the .8 value at this moment then can be taken to represent values of retinal signal magnitude for a test flash. For the example being calculated it is assumed that the extraretinal signal lags the retinal signal by .12 of the saccade's total duration [(b-a) = .12]. Since the extraretinal signal is drawn in the nomogram as a mirror image (reflected at the abscissa) of the theoretical extraretinal signal function, the PSE at the stimulus board (distance from the fixation target) is the vertical distance (drawn) between retinal and extraretinal signal functions at the moment of interaction. Since the magnitude of the extraretinal signal function at this time is smaller than the magnitude of the retinal signal function, the PSE is .26 saccade length units to the right of the fixation target for a rightgoing saccade or .26 saccade length units to the left of the fixation target for a left going saccade.

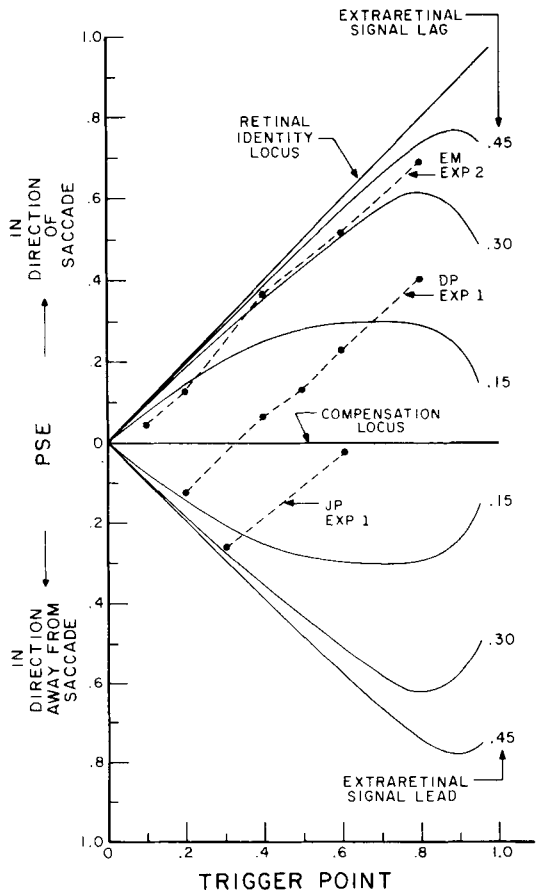

Fig. 9(b). Theoretical values of PSE predicted from the first model ("latency mismatch model"). Predictions were made from a nomogram as shown in Fig. 9(a). PSE is plotted as a function of the trigger point's distance from the fixation target for the several values of extraretinal signal lag that are indicated (theoretical curves are plotted as solid lines above the abscissa), and for several values of extraretinal signal lead (theoretical curves are plotted as solid lines below the abscissa). The values of extraretinal signal lag or lead are expressed in units of "proportion of saccade duration." Both abscissa (trigger point location) and ordinate (PSE) are expressed in units of "proportion of saccade length." Also shown are three sets of data; the set for EM is among those that receive the most reasonable fits; the data for DP and JP are among those that are not fitted well at all.

particular information on this question, however, two interpretations of the relation between the incomplete constancy found under the present conditions of stimulation and the normal stability of the visual world during voluntary saccades suggest themselves:

1. It is possible that imprecise synchronization between retinal and extraretinal signals is common, but in a normally illuminated environment such a failure of synchronization might not be capable of providing consequences that are generally detectable to the S as a loss of direction constancy for stationary objects[14] (it is worth noting again that under sufficiently dim illumination, failures of direction constancy may be observed; see above).

2. On the other hand, it is possible that precise synchronization between retinal and extraretinal signals occurs under normal conditions of stimulation and that the deviations from the compensation locus as well as the variability found in our data arise because the time between retinal stimulation and time of arrival of the signal from the retina at the point of interaction (retinal signal latency) for a brief flash is different from the retinal signal latency for the response from the same retinal locus when the stimulus is continuously present during the saccade, or that for some other reason, perhaps related to the intensity and adaptation level, a latency mismatch does occur.

With these ideas in mind, two models are developed in Section C below which extend the classical notion of compensation through an addition of retinal and extraretinal signals. In the first model both signals are assumed to be linear with eye position as in classical theories, but a latency mismatch between retinal and extraretinal signals is assumed for the present conditions of stimulation; this model does not fit the data well. In the second model we add an additional degree of freedom by deriving the form of the extraretinal signal function from the data. This proves to be nonlinear with eye position during the saccade, and thus raises some further problems that are discussed in Section D.

C. Two Models Employing an Extraretinal Signal

In order to predict the locus of PSEs with varying trigger point it is necessary to specify several theoretical functions: (1) It may be expected that the latency of the retinal response to a test flash will vary with retinal eccentricity. Such variation would produce a variation of retinal signal latency (time between the moment of stimulation and the moment when the retinal signal interacts with the extraretinal signal) with retinal eccentricity of the flash. However, for both models we assume as a first approximation that retinal signal latency is independent of retinal eccentricity. Although in more complete developments it will be necessary to incorporate a more accurate statement of the retinal signal latency-eccentricity function, this approximation will not influence the conclusions to be inferred below. In Figs. 9(a) and 10, then, we draw a function parallel to the function relating change of eye position (where the fovea is pointing) to time during the saccade. These two functions are displaced from each other by a duration a, equal to the retinal signal latency; each point on this second function then graphs the moment at which the retinal signal arising from a test flash interacts with the extraretinal signal (abscissa) for different values of trigger point (ordinate). (2) Implicit in assumption (1) was the assumption that the extraretinal signal operates on the retinal signal arising from the test flash to effect a shift of local signs, and that this operation occurs as a "momentary" interaction. It should be noted at this point that the two models being presented are our attempts at extending what we understand to be classical theory. A new interpretation will be presented later. (3) For the first model we assume an extraretinal signal whose magnitude grows with time parallel to the change in eye position but displaced from it by a fixed duration b, the extraretinal signal latency. For the second model we allow b to vary with trigger point. (4) The absolute values of retinal and extraretinal signal latencies are not significant for either the classical theories or their extensions in the models being presented. However, the difference between the two latencies is crucial. (5) The value of retinal signal y is the distance from the fovea measured on the retina between loci of the images of flash and fixation targets. Assuming then that the PSE at the stimulus board (Z_p) is equal to Y_p (the retinal PSE) plus the value of the extraretinal signal X at the moment of interaction between retinal and extraretinal signals implies that the local sign shift is equal to the magnitude of the hypothesized extraretinal signal (i.e., $X = Z_p - Y_p$).

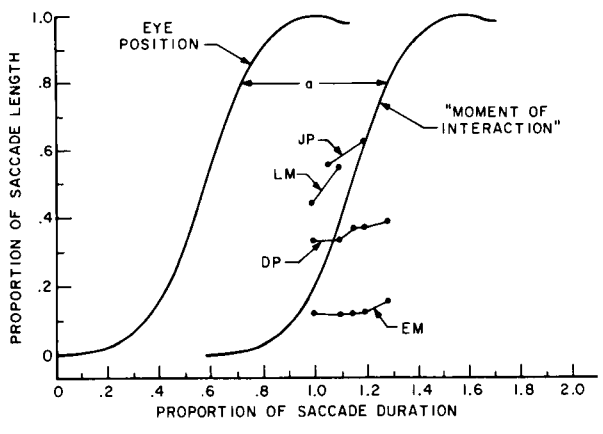

Fig. 10. Similar to Fig. 9(a) for second model extending classical theory. The set of points for each S are theoretical values of extraretinal signal as a function of trigger point calculated from the "one saccade" data of Experiment 1. (Only two points are shown for LM since reliable "one saccade" PSEs could not be obtained from the available data for the other three trigger points; see Table 2.)

In the first model, as indicated above, it is assumed as in classical theories, that retinal and extraretinal signals are both linear with eye position. But while $Y = f(t-a)$, $X = f(t-b)$; when $a \neq b$, $(a-b)$ represents the latency mismatch. When $a = b$, this model is identical to our understanding of classical theories. The test of the first model is shown in Fig. 9. Figure 9a is part of a nomogram for calculation of PSE as a function of trigger point for several values of latency mismatch; the calculation procedure is outlined in the legend. Figure 9b contains the calculated PSEs along with three sets of data from Fig. 6; the set of the data for EM, Experiment 2 is one of the few that is reasonably fitted by the model; the sets of data for DP, Experiment 1, and JP, Experiment 1, are among the majority that are poorly fitted by the model. The main features of the calculated PSE loci may be summarized as follows: (1) When the difference in latency between the extraretinal signal and retinal signal is zero, the predicted PSE locus is identical with the compensation locus (classical theory). (2) When the extraretinal signal lags the retinal signal by more than the saccade duration, the predicted PSE locus for flashes presented during the saccade is identical with the retinal identity locus. (3) When the extraretinal signal lags the retinal signal by less than the saccade duration, the predicted PSE locus begins at the origin, rises to a maximum and declines. Predicted functions fanning out from the origin for lags of .15, .30, and .45 of the saccade duration are shown by the upper three curves in Fig. 9b. (4) When the extraretinal signal leads the retinal signal, the predicted PSE locus is the reflection below the ordinate zero of the function with the identical extraretinal signal lag drawn above the abscissa (ordinate zero).

It is immediately apparent that the loci in (1), (2), and (4) are wholly incompatible with the results of the present experiments. The assumption in (3) that the extraretinal signal lags the retinal signal is somewhat more successful: The PSE locus derived from assuming a .30 lag fits some of the data for EM in Experiments 1, 2, and 4 within the limits of experimental variability; the deviations of theory from data are, however, systematic rather than random. In addition, DP's data in Experiment 3 is fitted rather well by the assumption of a .45 lag. *However, none of the other data can be fitted by any possible prediction from theory.* In particular, the model of Fig. 9 for any value of lag predicts a PSE locus radiating from the origin on only one side of the

compensation locus; the PSE loci of LM and DP for Experiments 1, 2, and 4 and for JP in Experiment 1 cross the compensation locus.[15] Other striking systematic deviations from theory are also clear.[16]

Any model purporting to account for the quantitative features of the present data must thus contain a degree of freedom in addition to the latency mismatch of retinal and extraretinal signals. Within the present framework three possible degrees are available: (1) The shape of the retinal signal vs time function; (2) the shape of the extraretinal signal vs time function; (3) the interaction of retinal and extraretinal signals.

It will be shown elsewhere (Matin, Matin, & Pola, 1969b) that a more reasonable assumption about the relation of retinal signal latency to eccentricity than the invariance already assumed cannot significantly alter the predictions derived from the model described above. Thus, as candidates for the additional degree of freedom we consider only (2) and (3). The present data do not afford any basis for deciding whether the extraretinal signal is nonlinear with eye position or whether an extraretinal signal that is linear with eye position (although displaced in time) adds nonlinearly with the retinal signal in determining the PSE. In fact, any prediction that can be made by employing a nonlinear extraretinal signal can be duplicated with a linear extraretinal signal if the addition of retinal and extraretinal signals is assumed to be a nonlinear process. However, since the addition of linear and nonlinear signals can be most simply conceptualized as a synaptic operation, and since substantial support exists for the linearity of synaptic interactions (Ratliff et al, 1963; Granit, 1966) it would be more reasonable to assume a nonlinear modification of the extraretinal signal prior to its interaction with the retinal signal.

Since we retain the assumption of linear addition of retinal and extraretinal signals, as before, then at the moment of interaction the magnitude of the extraretinal signal must be equal to the local sign shift. We may then employ the empirically determined local sign shifts to provide the best guide as to what the time course of the extraretinal signal must be. Theoretical extraretinal signal functions obtained in this fashion are plotted in Fig. 10. The most striking common aspect of these functions is their extreme flatness relative to the rate of rise of the retinal signal function. While the majority of the functions show some tendency to rise, this tendency is neither universal nor very large. In view of the limited range of magnitudes over which each of these functions varies, it is not feasible to attempt to specify its form within the experimental range any more closely. However, the limited range of variation for each of the curves leads to some significant predictions about its nature outside of the range of the present experiments. Since the local sign shift must approximate zero at the moment of extinction of the fixation target, the extraretinal signal must approximate zero then also. The sizeable magnitude of extraretinal signal early in the saccade then suggests that some of its growth has taken place before the "moment of interaction" corresponding to the beginning of the saccade. This raises the possibility that local sign shifts will be found for flashes presented prior to the beginning of the saccade. In addition, since the local sign shift late in the saccade is not yet of a magnitude to compensate completely for the eye movement, it is reasonable to expect that further shifts will be found for flashes presented after the completion of the saccade. Both of these predictions have been borne out in experiments to be described later in this series (Matin, Matin, & Pola, 1969b, c). The suggestion of asymptotes for the extraretinal signal magnitude of zero prior to the saccade and a value commensurate with full compensation after the saccade along with the flatness of the functions shown in Fig. 10 suggests that the extraretinal signal function will be sigmoidal in shape (as the saccade itself) but that its growth will last over a very much more extended time course than the saccade, and that it will lead the retinal signal at first and lag later. This theoretical extraretinal signal then must be nonlinear with eye position. Such a consequence raises further problems which we shall begin to explore in the remainder of this discussion.

D. Time-Lock or Position-Lock

Throughout the previous discussion it has been assumed that the extraretinal signal responsible for the local sign shift is either a consequence of the change in eye position or of the intention to turn the eye. It should be noted that by tieing the growth of extraretinal signal magnitude to change in eye position in the models developed in the previous section we have not at all committed the models to a view in which the extraretinal signal is a *consequence* of the change in eye position. Such a tie could as easily be thought of as a consequence of the intention to turn the eye. The nonlinear relation between eye position and extraretinal signal required by the second model, in fact, speaks well for the latter contingency. In fact, in the main data of Experiments 1-4 eye position at the moment of flash presentation is essentially confounded with the time from the beginning of the saccade, and the possibility that the local sign shift follows a time course independently of (or in conjunction with) any shift due to variation in eye position will have to be considered. This section will provide a discussion of this possibility in terms of the data of the present experiments. A more complete discussion will be given in a succeeding report (Matin, Matin, & Pola, 1969b). First, however, we will look more closely at the possible link between the growth of extraretinal signal and the change of eye position.

Having found sufficient evidence to reject a model in which the extraretinal signal parallels the change in eye position, we are left with the model which contains an extraretinal signal that changes very little if at all over the range of times we have considered. This is particularly clear for Ss DP and EM and is a direct consequence of the fact that very little systematic variation in the shift of local signs is found for them with variation of trigger point. However, the suggestion of a variation of local sign shift with trigger point is stronger for LM. This difference between Ss appeared clearly enough in Experiments 1-3 and was confirmed in Experiment 4 which was specifically designed to eliminate some possible sources of response bias in the earlier experiments.[17] A suggestion of response bias in LM's data for Experiments 1-3 lay in the fact that his saccade length was shorter on the average for trigger points closer to the fixation target (this was not true for DP, EM, or JP). However, in Experiment 4 also, LM's data showed an effect of trigger point location on the local sign shift; here no effect was obtained for either EM or DP. While this effect for LM was attenuated by removing the "two-saccade" trials it was not eliminated (Fig. 7b). This result for LM then suggests that eye position at the moment of flash presentation could play a role in determining the magnitude of the local sign shift. Again, however, LM's saccade length was related to trigger point location; thus, on "one-saccade" trials average saccade length was 26% greater for the trials on which the more distant trigger point was employed than for trials in which the trigger point nearer to the fixation target was employed.[18] The increased local sign shift for the more distant trigger point could thus have been related to saccade length rather than eye position at the moment of flash presentation per se, with the magnitude of both determined by the process initiating the saccade; thus when a longer saccade is programmed, so is a larger local sign shift. Although the present data do not readily offer a test of this hypothesis, it is supported by data from other experiments in which test flashes are presented after the saccade is over (Matin, Matin, & Pola, 1968b).[19]

In view of the fact that only LM showed a systematic influence of trigger point location on the magnitude of the local sign shift and that in his case it is possible that the effect is mediated by variation in saccade length, a direct effect of eye position at the moment of presentation of the test flash remains questionable and will require further study.

At the beginning of this section we noted that eye position and time from the beginning of the saccade are confounded in the data discussed so far. This confounding is a consequence of the remarkable uniformity from saccade to saccade of the time course of a saccade of a given length.[20] Some access to the separation of time and position is available, however, as a result of the separation of "one-saccade" from "two-saccade" trials. For a given

trigger point the eye is at the same position (i.e., at the trigger point) on both types of trials at the moment the flash is presented. However, a longer time to flash presentation from the beginning of the first saccade has intervened on "two-saccade" trials than on "one-saccade" trials. Nearly all of this increased duration was taken between the termination of the first saccade and the beginning of the second saccade on "two-saccade" trials. We do not have detailed quantitative information on the magnitude of this interval. However, the local sign shift is invariably greater on "two-saccade" than on "one-saccade" trials [i.e., for a given trigger point, a test flash at a given target locus was reported as lying to the first-flash-side of the fixation target more frequently on "two-saccade" trials than on "one-saccade" trials (Tables 2 and 3); thus, the PSE for the "two-saccade" trials lies further from the "retinal identity" locus than the PSE for the "one-saccade" trials (Fig. 6)]. This result implies the possibility that *the local sign shift follows a given time course aside from any influence of the change in eye position during the saccade.*

The latter implication is not sharp, however, since it depends on the assumption that the local sign shift follows the same time course from the beginning of the first saccade on both "one-saccade" and "two-saccade" trials.[21] (It should be noted that if we were to assume that the time course of the local sign shift was time-locked to the beginning of the saccade during which the flash occurred, we would predict that the "one-saccade" and "two-saccade" PSEs would be reversed from the relation observed in the data.) Such an assumption does not appear reasonable on a *prima facie* basis: One might more reasonably argue that if the shift in local signs is programmed by the S to follow a specific time course such programming would occur before the saccade and at least in some sense, however distorted, mirror the action of the S which is also preprogrammed.[22] Since the actions are different on "one-saccade" and "two-saccade" trials, it would be reasonable to assume that the intentions guiding both the actions and the local-sign shifts would be different also, and so one would also expect some difference in time course for the local sign shift on the two types of trials. One way in which to hunt for such a difference would be to compare data at a given trigger point on "one-saccade" trials in which a second saccade occurred after the trigger point was passed with data on "one-saccade" trials in which no second saccade occurred. If the time course of the local sign shift were fundamentally different on trials in which two saccades occurred a difference in PSE should be found. The data of LM in Experiment 4 was processed in this way and no difference in PSE was found (there was not enough of this type of data in any other experiment to test the question elsewhere). The possibility that the PSE follows a time course locked to the beginning of the first saccade thus gains some indirect support and appears worthy of further considerations.

The direction of the difference in psychophysical response between "one-saccade" and "two-saccade" trials is identical for all Ss, and so the above time-locking interpretation accounts for all Ss' data on this point. It also predicts an increase in the local sign shift with distance of the trigger point from the fixation target. Such an increase is equivocal however. While some increase does appear to be present, it is very difficult to be certain about its nature as the previous discussion has been at pains to point out. Nevertheless, the difficulty on this point may be due to the slowness with which the local sign shift changes relative to the rapidity of the saccade itself.

A final word regarding the interpretation of the models is in order. The two models discussed in the previous sections were essentially extensions of our understanding of classical theories. It seems clear that this kind of model requires not only a latency mismatch between retinal and extraretinal signals, but an extraretinal signal that is nonlinear with change in eye position to the extent that its growth is very much slower than the change in eye position. In deriving the predictions of PSE locus from the models, classical theory was followed insofar as it was assumed that the extraretinal signal operates on (or mixes with) the retinal signal arising from the test flash to produce a shift of local signs; presumably this interpretation would require that it is the local sign for the test flash that has been shifted. A completely parallel interpretation of the quantitative features of the models can be given in which it is assumed that the extraretinal signal operates on the *memory of the visual direction of the fixation target* instead of the retinal signal arising from the flash. *The local sign shift is then a shift of remembered direction of the fixation target;* the test flash then provides the standard direction against which this memory is compared, and the comparison occurs at the moment the test flash is seen.

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NOTES

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3. In a previous report (Matin & Pearce, 1965) we employed the term proprioception to refer to any signals regarding eye position or change in eye position or intention to move the eye not derived from the retina. Since proprioception is a term which has a number of connotations that have arisen in different historical contexts, we shall avoid its use and substitute the noncommital term extraretinal signal instead.

4. By "local signs" we refer here to the mapping on the retina of a psychophysical measure of visual direction. In the present report we shall be discussing the relation between visual directions for a pair of targets viewed at different times. The PSE will be that target presented later which appears in the same visual direction as a standard target presented earlier. If the retinal locus struck by the PSE is different from the one struck by the standard we shall say that the local sign map has been shifted between the two presentations. See Matin, Matin, & Pola (1969b) for a further clarification of this usage.

5. The comparator used in the previously-reported experiments (Matin & Pearce, 1965) was set so that the pulse out of the differentiator would be too small to turn on the flash when either the saccade was begun too close to the trigger point (less than about 30 min of arc) or the eye drifted across the trigger point. This variation in pulse height is proportional to the rate of change of comparator output voltage in the neighborhood of the zero crossing; the latter is in turn controlled by the saccade velocity at the trigger point. In the present experiments the system was set to turn on the test flash when the typical saccade started from as little as 5 min of arc on the fixation-target-side of the trigger point; drifts across the trigger point failed to trigger a test flash.

6. The fits were obtained by Dr. Joyce Kerr on the 7094 computer using the probit technique (Finney, 1942).

7. This did not happen in any of the trials in the previously reported experiments (Matin & Pearce, 1965) where the system would not yield a sufficiently large pulse to trigger a flash for velocities at which the trigger point would be crossed during a second saccade (see footnote 5).

8. Each of the differences referred to is for a test flash generated at a given locus in the physical array but differing by 26.2 min in retinal locus when presented from the two different trigger points.

9. The apparatus employed was described earlier (Matin & MacKinnon, 1964). With it a beam was reflected from the contact-lens-mirror and sent through a telescope of .5 angular magnification before returning it to the pupil. The system presented an image of a field stop at optical infinity to the S.

10. When such a push is provided with a S viewing a normal (non-stabilized) target alone the target appears to move to the right. However, such a push undoubtedly moves the eye to the left as well as the contact lens, and such an apparent movement is seen when an eye without a contact lens is pushed (see above); it is thus not possible to tell whether or not the movement seen here by the S is due to optical distortion in the relation of contact lens to eye or to passive eye movement itself. Hence the need for stabilized viewing.

11. For a lens of X diopters power, a deviation of X prism-diopters will be produced for every centimeter of decentration.

12. The magnitude of the local sign shift is given in Fig. 6 at a given trigger point by the vertical difference between the data point and the point above it on the main diagonal.

13. We do not intend to use either Helmholtz or Sherrington as a "straw man." However, with the exception of the von Holst model (1954) which appears to be essentially a modern explication of the Helmholtzian view, we are not aware of any other attempts to develop either outflow or inflow theory in sufficient detail to permit the kind of quantitative prediction that is necessary to indicate what should be expected in the present kind of experiment. Thus, the implications we draw from the "inflow" and "outflow" theories are those that we think most workers who discuss these theories would infer; however, Helmholtz and Sherrington might have inferred otherwise. Although von Holst does not specifically state that direction constancy should be preserved for visual stimulation present throughout the saccade, it seems clear that this is intended. He does indicate that the "re-afferent pattern" (i.e., the change in retinal stimulation during the saccade). . ."exactly nullifies the efference copy" (where the "efference copy" is an "image" of the intended eye movement) during a normal saccade and so explains the stability of the visual perception of direction. Further, his use of + and − for efference and re-afference respectively and his addition of these quantities could hardly be understood otherwise.

14. One mechanism which could produce such an effect is the following: At a particular moment during a saccade, a specific retinal locus receives the image of one stationary environmental object. But both before and after this moment, images of differently located objects strike the same retinal locus and it is possible that these stimuli mask each other. The consequence of such masking might well be "suppression" of vision for stimulation during a saccade in a normally illuminated environment. This mechanism of suppression would be an alternative to the frequently suggested mechanisms of suppression through central inhibition (Volkmann, 1962); the alternative do not exclude each other. Although we have not studied this problem in detail, we have found evidence for masking effects between stimuli which strike the same retinal locus at different times in the saccade. Questions of localization for these stimulations during saccades thus may not normally arise.

15. It is worth reiterating that PSEs to the left of the fixation target for rightgoing saccades could not be due to artifacts resulting from lens slippage (see above).

16. The predictions from the above model assumes a fixed saccade length in a given experiment. In fact, as has already been pointed out, considerable variability in saccade length did occur particularly for LM. When a saccade is shorter than that prescribed by the first flash location, the distance of a given trigger point from the fixation target will occupy a larger proportion of the saccade than it will for a longer saccade. For a given latency difference, then, a larger local sign shift will be predicted for the shorter saccade than for the longer saccade (over most of the range of saccade lengths and trigger points). Increased variability in saccade length should thus lead to larger JNDs at a given trigger point. There is no real indication of such a general trend in the data although EM's JNDs are larger in Experiment 3 where saccade length was more variable than in Experiments 1, 2, and 4. However, LM's JNDs are not generally larger than those of the other Ss although his saccade lengths were more variable.

17. Each of the trigger points in each of the first three experiments was employed in a separate block of trials. Although the different trigger points were counterbalanced among blocks both within and across experimental sessions thus reducing the possibility of within and between session effects, this does not completely eliminate the possibility that some sort of response bias played a role in the relation of local sign shift to trigger point. The randomization of trigger point-test flash combinations in Experiment 4 was intended to eliminate this possibility.

18. Increased saccade length at the more distant trigger point for LM is a consequence of the following: Considerable variation occurred in the length of LM's saccades; some were long enough to reach the trigger point farthest from the fixation position, others were so short that they failed to trigger a flash even when the trigger point closest to the fixation target was used. Thus, on some trials a flash was triggered at the nearer trigger point by a saccade that was not long enough to have triggered a flash at the farther trigger point. We are not yet able to reconcile the observation that saccade length and local sign shift are correlated in the data of LM for Experiment 4 with the finding of no consistent difference in local sign shift magnitude between Experiments 1 and 2.

A trial on which the saccade was too short to cross the trigger employed on that trial was immediately repeated with the same trigger point and test flash. Since LM had considerable previous experience as a S in this type of experiment, he was aware of the fact that such missed trials were more likely at trigger points more distant from the fixation target and his report could have thus been biased; it is also possible that for such repeated trials a different response distribution resulted for some other reason. In fact, however, the psychophysical response distribution on these repeated trials was not different from the distribution on other trials and removing these repeated trials from the data did not change the effect at all from that shown in Fig. 7b.

19. An alternative hypothesis to account for the correlation of local sign shift and saccade length in the present experiments is that the S "sensed" trigger point location by means of saccade length and used the acquired information to bias his reports. While we could not test this possibility in the present experiments it could not account for the results in the experiments in which a test flash is presented after the saccade is over.

20. In the present experiments the eye is in the dark between the extinction of the fixation target and the moment the saccade begins (with the exception of the presentation of the brief first flash). Drifts of as much as 20 min can take place in this interval with the consequence that the beginning of the saccade on one trial may be as much as 20 min closer to a given trigger point than on another trial. In addition, under the conditions of the present experiments, some variability in the length of a saccade occurs with the consequence of slowing or speeding the time course of displacement during the saccade. However, both of these factors together introduce variations in the moment from saccade beginning when the eye crosses a given trigger point of at most ± 2 msec. It would not be feasible in the data of the present experiments to attempt to partial out eye position from time in the saccade on these bases.

21. This assumption can be weakened somewhat, but no essential qualification of the following discussion results.

22. We have no direct evidence of such preprogramming of saccadic behavior, however. Certainly Ss were not able to report from casual observation that they were doing anything different on "one-saccade" than on "two-saccade" trials, although further investigation may conceivably show that such a discrimination is possible. The brief time (as small as 50 msec) between the two saccades on "two-saccade" trials suggests that the second saccade could not have always been programmed during or after the first saccade. However, the possibility that information is available to the S on the length of the first saccade is suggested by casual observation of the recordings which indicates that a second saccade is more likely to occur on trials in which the first saccade is short than on trials in which it is longer (this is not yet rigorously established); this information may, however, be available before the beginning of the first saccade.

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