

Interactions Between Natural and Electrically Evoked Saccades.

III. Is the Nonstationarity the Result of an Integrator Not Instantaneously Reset?

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Schlag, John, Alexandre Pouget, Safa Sadeghpour, and Madeleine Schlag-Rey. Interactions between natural and electrically evoked saccades. III. Is the nonstationarity the result of an integrator not instantaneously reset? *J Neurophysiol.* 79: 903–910, 1998. In the monkey, fixed-vector saccades evoked by superior colliculus (SC) stimulation when the animal fixates can be dramatically modified if the stimulation is applied during or immediately after an initial natural saccade. The vector is then deviated in the direction opposite to the displacement just accomplished as if it were compensating for part of the preceding trajectory. Recently, it was suggested that the amplitude of the compensatory deviation is related to the amplitude of the initial saccade linearly, and that the ratio between the two decreases exponentially as stimulation is applied later. These two findings (spatial linearity and temporal nonstationarity) were invoked as evidence for the noninstantaneous resetting of a feedback integrator. Such an integrator is included in most models of saccade generation for the specific purpose of terminating a saccade when it has reached its intended goal. However, the hypothesis of a feedback integrator in the process of being reset implies that the exponential decay of the compensatory deviation is temporally linked to the end of the initial saccade. We analyzed the time course of this decay in stimulation experiments performed at 24 SC sites in two monkeys. The results show that if the start of the exponential decay of compensation is assumed to be linked to the end of the initial saccade, then the relation between the amount of compensatory deviation and the amplitude of the initial saccade is not linear. On the other hand, it is possible to show a linear relation if the measurements of compensatory deviation are made in terms of delay of stimulation from the saccade beginning. We conclude that stimulating the SC just after a visually guided saccade does not seem to test the properties of a feedback integrator. Whether such an integrator is or is not resettable is not likely to be decided by this approach. Conversely, as the nonstationarity of compensation is linked to the beginning of the saccade, the nonstationarity seems to represent a property of an event occurring at saccade onset. We suggest that this event, close to the input of the oculomotor apparatus, is the summation of the visual signal with a damped signal of eye position or displacement.

INTRODUCTION

Once a saccade is launched, its trajectory cannot easily be deviated and, immediately afterwards, there is a recovery period, lasting ~100 ms, during which new saccades rarely arise. Nevertheless, by electrical stimulation it is still possible to evoke an eye movement either during or shortly after a saccade. But in structures where stimulation, at rest, elicits stereotyped fixed-vector movements (i.e., their amplitude and direction depend only on the site stimulated), the vector

now can be systematically modified. It is deviated in the direction opposite to the movement just accomplished, thus appearing to correct (or compensate for) at least part of the preceding displacement. For instance, if the initial saccade is directed upward and rightward, the electrically evoked saccade will be less upward and less rightward than the control elicited from a steady position (see Fig. 2). Stimulating the superior colliculus (SC), Robinson (1972) first noticed this deviation and ascribed it to refractoriness, without implying any specific physiological mechanism. Initially, we called the observed change of trajectory “compensatory” (Schlag-Rey et al. 1989), following Sparks and Mays (1983) who studied the effect of collicular stimulation on natural saccades (i.e., the same paradigm in reverse order) and reported practically the same type of change. The term compensation immediately evokes the direction in which the course correction is made (i.e., opposite to the initial movement). Compensation is reliable and, eventually, can result in an inversion of the fixed-vector. This effect has been proposed as the basis of a test to probe mechanisms of saccade elicitation in various brain structures (Schlag and Schlag-Rey 1990).

Such a test was applied to the study of different structures including, in particular, the superficial and intermediate layers of SC with trains of electrical stimuli systematically delivered at varying delays during natural saccades, thereby provoking what is called a saccade collision (Schlag-Rey et al. 1989). This term refers to the fact that a saccade electrically evoked when another one is in progress no longer has its expected trajectory. Its course is deviated as described above. In our initial study, we estimated that the amount of deviation (measured linearly along the path of the initial saccade) depended on the distance traveled by the eye after the onset of stimulation, and we thought that the simplest way to interpret this observation was to assume that the electrical stimulation artificially creates a retinocentric goal (something like a phosphene) toward which a targeting movement is then programmed by the oculomotor apparatus (Schlag et al. 1989). However, because of the efferent delay (i.e., the saccade latency), the eye movement toward the goal cannot start immediately. As the initial saccade continues its course during this latency, the trajectory of the electrically evoked saccade, programmed to reach a goal, has to be adjusted to correct for the detour. We reported this effect for stimulating sites in superficial and intermediate SC layers, whereas stimulation deeper in SC elicited no saccade deviation (Schlag-Rey et al. 1989).

Because we only stimulated during the initial saccade in our first experiments with superficial and intermediate SC stimulation, we did not notice that evoked saccades can still be deviated if they are induced shortly after the end of the initial saccade. This important observation was made regarding the same structures of the SC by Nichols and Sparks (1995) and Kustov and Robinson (1995); both groups recently reported that the saccade deviation decays exponentially with a time constant close to 50 ms, similar to what was found with the stimulation of the frontal eye field (Dassonville et al. 1992a). In addition, Nichols and Sparks (1995) presented data suggesting that the amplitude of deviation is linearly related to the total amplitude of initial saccades. They argued that the two findings combined (nonstationarity and linear relation of deviation with initial saccade amplitude) are predicted if the resetting of the feedback displacement integrator is not instantaneous. Here they were referring to the integrator postulated by practically all oculomotor models (e.g., Jürgens et al. 1981) that stops the saccade generator precisely when its goal is attained. The displacement integrator is supposed to be maximally charged when the generation of any saccade is completed and, if it is not instantaneously reset, any residual charge should be automatically subtracted from the next desired displacement command. They reasoned that this is why the electrically evoked saccade is deviated.

The hypothesis of an integrator in the process of being reset implies that the exponential decay of deviation observed is temporally linked to the end of the initial saccade (although not necessarily simultaneous with it). Nichols and Sparks (1995) provided some evidence in this respect by plotting the exponential decay of deviation as a function of the intersaccadic interval. This is a measure linked to the end of the initial saccade. However, Kustov and Robinson (1995) showed a similar exponential decay as a function of the delay of stimulation measured from the saccade beginning. As shown in Fig. 1, these two measures are not equivalent because the duration of the initial saccade varies with its amplitude, whereas the latency is fixed. Thus to maintain a constant intersaccadic interval, the delay of stimulation must increase as the amplitude of the initial saccade increases. Intersaccadic interval and delay of stimulation are two measures that have different implications, if the exponential decay is a function of the delay of stimulation from the start of the saccade (which, hereafter, we shall abridge to delay of stimulation), it must be a property of an event that occurs at the onset, and not at the offset, of a saccade.

Our first objective, in the present study, was to determine the time course of the deviation decay, particularly to determine whether its starting time is linked to the beginning or the end of the initial saccade. Then we sought to specify the conditions under which the amount of observed deviation is a linear function of the amplitude of the initial saccade. Some of these observations were included in a preliminary short report (Sadeghpour et al. 1996).

METHODS

Twenty-four SC sites of stimulation were tested in two adult rhesus monkeys (*Macaca mulatta*). Procedures have been described in detail previously (Schlag-Rey et al. 1989). During each

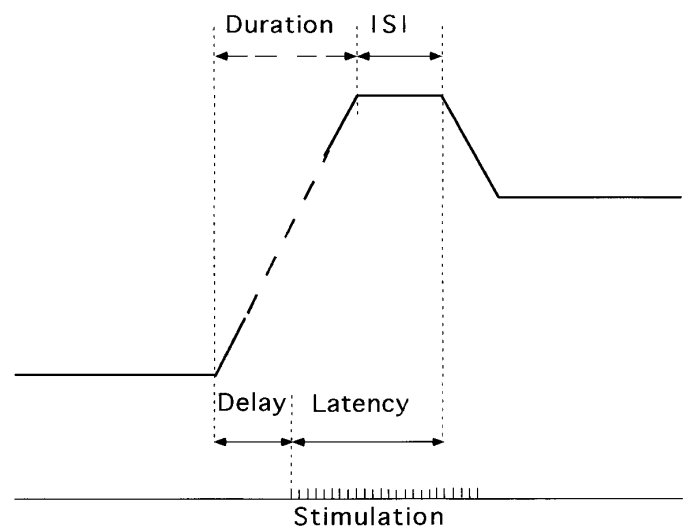


FIG. 1. Schematic representation of the compensatory effect of stimulation and definition of time parameters. An initial saccade is followed by an electrically evoked saccade. Stimulation, train of electrical pulses delivered in superior colliculus (SC); latency, latency of electrically evoked saccade from stimulation onset; delay, delay of stimulation from the onset of the initial saccade; duration, duration of initial saccade; ISI, intersaccadic interval. This schema illustrates the relation between ISI and Delay

$$\text{Delay} = \text{ISI} - \text{Latency} + \text{Duration}$$

Assuming the latency to remain relatively constant for a given site of stimulation, this relation implies that, for maintaining a constant ISI, the delay of stimulation has to increase with the duration of the initial saccade.

session the animal sat in a very dim red ambient illumination and looked at target points of light, rear-projected on a tangent screen from a cathode-ray oscilloscope. A tungsten microelectrode was lowered to intermediate layers of the SC. In each case preliminary recordings were made of the types of unit responses to luminous targets and these recordings were repeated after electrical stimulation showing, among other things, that cells had survived these stimulations. At the sites selected in SC, cells were either visual and presaccadic (burst-type) or presaccadic alone. Routinely, stimulation trains comprised 25 negative pulses of 0.2-ms duration at 400 or 500 Hz. Current level ($20\text{--}50\ \mu\text{A}$) was kept low but clearly above threshold: it was adjusted so that no change in saccade dimensions or latency were brought by further increasing the current. In two cases, the current was deliberately set at $50\ \mu\text{A}$ because this is the value used by Nichols and Sparks (1995). The results were the same as in our other experiments. Eye movements were recorded with a magnetic search coil and measurements were made by computer on stored records digitized at 1 kHz.

The monkeys were trained to make a saccade that they had to hold for 400–500 ms from a point of fixation to a target flashed for 50 ms. In each block of trials, the target was kept at the same place whereas the position of the point of fixation was varied, thereby changing the dimensions of the initial saccade. Controls were obtained by placing the point of fixation at the same position as the target in the experimental trials. Thus experimental and control evoked saccades that were compared started all from the same place. In control trials, stimulation was applied 100 ms after the extinction of the point of fixation. For each SC site studied there were 12–25 control trials intermixed with experimental trials; in about one-third of the experimental trials, no electrical stimulation was applied to make the occurrence of stimulation less predictable. Usually, the target was placed at the center of the screen (corresponding to the central eye position). However, when it was desirable for initial saccades to be larger than 25° vertically, the

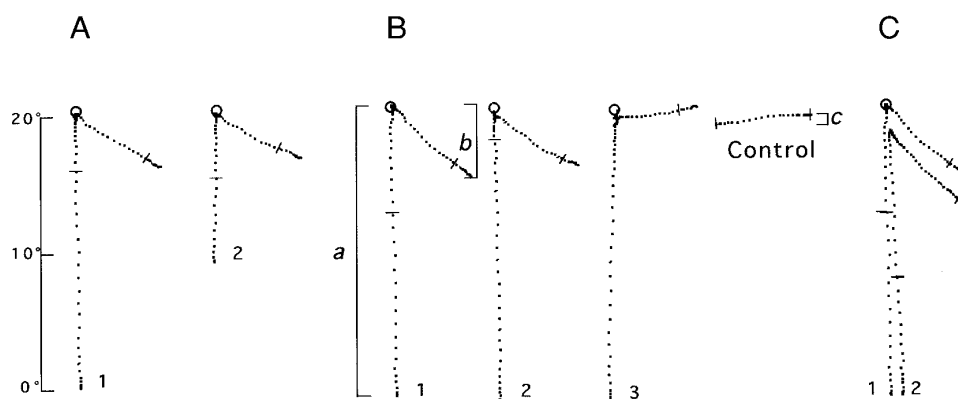


FIG. 2. Illustration of amplitude and time series using actual data from 1 SC stimulation site. All initial saccades were upward and followed by rightward electrically evoked saccades. Fixed-vector saccade labeled control in *B* is saccade evoked when the eyes were immobile. Ticks indicate onsets and offsets of trains of stimulation. \circ , locations where the targets appeared. *A*: amplitude series run with 10 ms intersaccadic intervals; initial saccade was 20° in *record 1*, and 10° in *record 2*. *B*: time series; intersaccadic intervals were 5, 22, and 116 ms, respectively, in *records 1–3*; initial saccades were 20° . Vectors *a*, *b*, and *c* are measurements of the vertical components of the initial saccade (*a*), deviated saccade (*b*), and control saccade (*c*). See text for further details. *C*: *record 2* is an initial saccade aborted by the stimulus 2.6° before reaching the goal, compared with *record 1*, which is not aborted.

target was displayed 10° above or below the horizon for the whole series of measurements. Of course, appropriate controls were then run from these eccentric target positions.

At all selected sites, electrical stimulation produced saccade deviation by collision. As Nichols and Sparks (1995) did in their experiments, we ran two series of trials at the beginning. In one of them, it was the amplitude of the initial saccade that pseudo-randomly varied (see sample of raw data in Fig. 2*A*) and, in the other, it was the timing of stimulation (Fig. 2*B*). The initial saccades in Fig. 2*A* had different amplitudes: 20° in *record 1* and 10° in *record 2*. Both were aimed at the target represented by a circle, and stimulation (onset and offset indicated by tick marks) was applied at a fixed intersaccadic interval. For this purpose stimulation was triggered after an adjustable delay by the eye signal entering an electronic window centered on the target site. Thus stimulation started here before the end of the initial movement to minimize the intersaccadic interval (to 10 ms in these cases). The electrically evoked saccades in *records 1* and *2* were directed to the right and deviated downward (vertically inverted) with respect to the control displayed at right in Fig. 2*B*. Although the stimulation usually did not outlast the evoked saccade (as in Fig. 2), this saccade was not artificially cut short because the amplitude of its horizontal component remained constant despite the amount of deviation.

In contrast, in the time series experiments the amplitude of the initial saccade was kept constant at one or two fixed values chosen between 5 and 32° . For instance, in Fig. 2*B* the amplitude of the upward initial saccades was 20° . Intersaccadic intervals shown were, respectively 4, 22, and 116 ms in *records 1–3*. To obtain such intervals, the stimulation was applied with prearranged delays from the beginning of the initial saccade (detected on-line).

There was an important constraint on the choice of the timing of stimulation. If the latency period in Fig. 1 terminated before the end of the initial saccade, this saccade was interrupted and replaced by the evoked saccade. This situation is illustrated in Fig. 2*C* where *record 1* is similar to *record 1* in Fig. 2*B*, whereas the stimulation was applied 7 ms earlier in *record 2*. As a consequence, the initial saccade was cut short 2.6° from the target and the intersaccadic interval was null. Such trials were not included in the reported series of measurements because the stimulation had possibly interfered with the completion of the initial saccade. An initial saccade

was considered aborted if it terminated more than 2° short of the target and the intersaccadic interval was zero.

At the beginning of this study, experiments were run with initial saccades in various directions including horizontal, or oblique, and perpendicular to the control fixed-vector. In some cases the deviation was limited because it was not possible to obtain left-right reversals by collicular stimulation (in contrast to cortical stimulation, of frontal eye field for instance, that produces horizontal reversals easily) (Dassonville et al. 1992a). Because our objective was to quantify deviation and since up-down reversals readily occurred with SC stimulation, this report only includes observations made with vertical initial saccades. Actually, most data published by Nichols and Sparks (1995) were also obtained with vertical initial saccades. We did not study SC sites at the depth where previously we failed to find compensation (Schlag-Rey et al. 1989) because such sites were apparently not explored either by Nichols and Sparks (1995) or by Kustov and Robinson (1995).

The purpose of Fig. 2 is to explain how measurements were made. The vertical component of the evoked saccade (vector *b* in Fig. 2*B*) minus the amplitude of the vertical component of the control fixed-vector (vector *c* in Fig. 2*B*), provides a measure of the vertical difference that we call deviation. In the time series, the result of subtracting *c* from *b* was normalized by dividing it by the amplitude of the vertical component of the initial saccade (vector *a* in Fig. 2*B*) and multiplying by 100. We call the result percentage of deviation (as seen plotted on the ordinate in Figs. 3 and 4). Expressing results as percentages of deviation is justified if the amplitude of deviation is linearly related to the amplitude of the initial saccade. Then, this measure allows a precise comparison between data obtained with different sizes of initial saccades. Moreover, the significance of the findings becomes immediately clear: 0% means that the evoked saccade is the same as the control fixed-vector (i.e., no deviation), whereas 100% means that the total trajectory of the initial saccade has been compensated for (as is predicted by the hypothesis of a resetting integrator if it is fully charged at the time of stimulation).

RESULTS

Fixed-vector saccades evoked from the 24 collicular sites studied were all contraversive; they varied in amplitude from

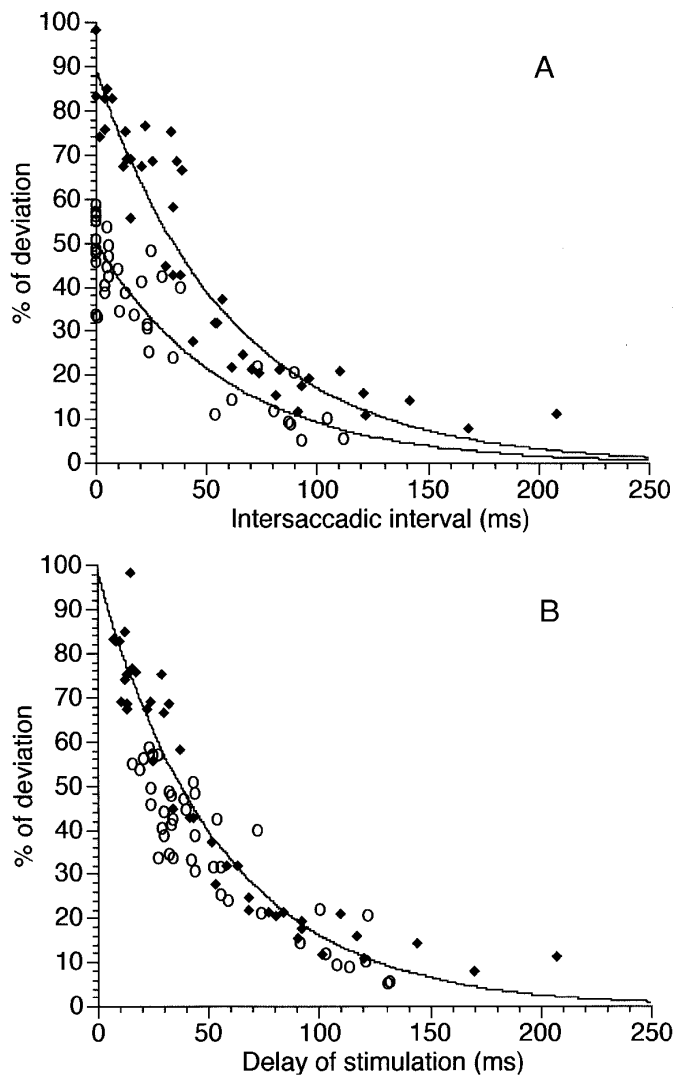


FIG. 3. Time series data from 1 stimulation site with 2 sizes of initial saccades: 9.43° (\blacklozenge) and mean 22.88° (\circ). A: both sets of data are plotted as a function of intersaccadic interval (duration of the interval between the end of the initial saccade and the start of the electrically evoked saccade). Each set is fitted by an exponential curve. Time constants: 61.3 ms for 9.43° curve ($R^2 = 89.02$), and 59.5 ms for 22.88° curve ($R^2 = 80.20$). Time constant for global exponential curve (not shown) fitting both data clusters: 60.2 ms ($R^2 = 58.25$). Earlier stimulations were not included in the plot because they abridged the initial saccade. B: same data plotted as a function of the delay of stimulation (from the onset of the initial saccade to the onset of stimulation). Time constant: 55.6 ms ($R^2 = 86.40$).

4.8 to 26.9° . Thirteen control saccades were oblique upward, nine oblique downward, and two horizontal. Amplitude and time series were run at seven sites, only time series at seven sites, and only amplitude series at seven sites. Quantitative results on three sites are not included because initial saccades were not vertical (see METHODS).

Figure 3A presents data of a time series, collected by stimulation at a single collicular site under two conditions: 1) the initial saccade was either 9.43° (mean $\pm 0.76^\circ$ SD; filled diamonds) or 2) 22.88° (mean $\pm 1.51^\circ$; open circles). The latency of the evoked saccade was 26.3 ms (mean ± 1.3 ms). Therefore *left* data points in Fig. 3A, at intersaccadic intervals shorter than 26.3 ms, correspond to stimulations

starting during the initial saccade. Although stimulation was applied intentionally early to minimize the intersaccadic interval, we verified that in none of these cases was the initial saccade aborted (see METHODS and Fig. 2C).

The data points in Fig. 3A were fitted by exponential curves with a time constant of 61.3 ms (filled diamonds) and 59.5 ms (open circles). Confirming what Kustov and Robinson (1995) already noted, the data points obtained by stimulation during the initial saccade (i.e., at intervals < 26.3 ms) seem to form a continuum with those obtained by stimulating after the saccade. But, although clearly a 9.43° initial saccade was almost entirely compensated for at the shortest intersaccadic intervals (i.e., close to 100%), 60% of deviation was the maximum that could be achieved under any circumstances for an initial saccade of 22.88° mean amplitude. Applying earlier stimulations resulted only in aborting the initial saccade which could no longer reach 22.88° .

What accounts for this difference between the two sets of data plotted in Fig. 3A? Note that large initial saccades last longer than short initial ones (~ 50 ms for a 23° amplitude compared with 30 ms for a 9.5° amplitude). Hence, data points plotted at equal intersaccadic intervals in the time series Fig. 3A were, in fact, collected at very different delays from the onset of the initial saccades: ~ 20 ms later for the open circles than for the filled diamonds. When we replotted the data points of Fig. 3A as a function of the delay of stimulation [i.e., the time reference that we previously used (Schlag et al. 1989) as did Kustov and Robinson (1995)], we obtained the plot presented in Fig. 3B. We should expect to find less dispersed data points if the amplitude of deviation is linearly related to the amplitude of the initial saccade and if the amount of deviation is expressed as a percentage (see METHODS). The fact that the data points in Fig. 3A are better fitted by the separate curves shown ($R^2 = 89.02$ for 9.43° and $R^2 = 80.20$ for 22.88°) than by a single one (not shown, $R^2 = 58.25$) suggests that the intersaccadic interval may not be the most appropriate time reference for measuring the amount of deviation. Conversely, the fact that the goodness of fit ($R^2 = 86.40$) for the curve in Fig. 3B is better than the goodness of fit ($R^2 = 58.25$) for the global curve in Fig. 3A (calculated but not shown) suggests that the delay of stimulation is a more appropriate time reference.

Figure 4 illustrates in a different way the effect of changing the time reference from the end of the initial saccade (as does the intersaccadic interval) to the beginning of that saccade (as does the delay of stimulation). The data in this figure pertain to two other experiments (A and B) in which initial saccade amplitudes were varied widely, but the intersaccadic intervals were kept relatively constant. If the intersaccadic interval was a relevant parameter, the percentage of deviation should have remained constant too. The data points should be clustered at single percentage values in A_1 and B_1 , but instead they were widely dispersed. In A_2 and B_2 , we plotted the same percentage values as a function of the delay of stimulation. The curves in A_2 and B_2 suggest that the dispersion seen in A_1 and B_1 is actually due to varying delay times (i.e., intentionally varied to keep the intersaccadic interval relatively constant). We compared plots of deviation percentages, made as a function of the

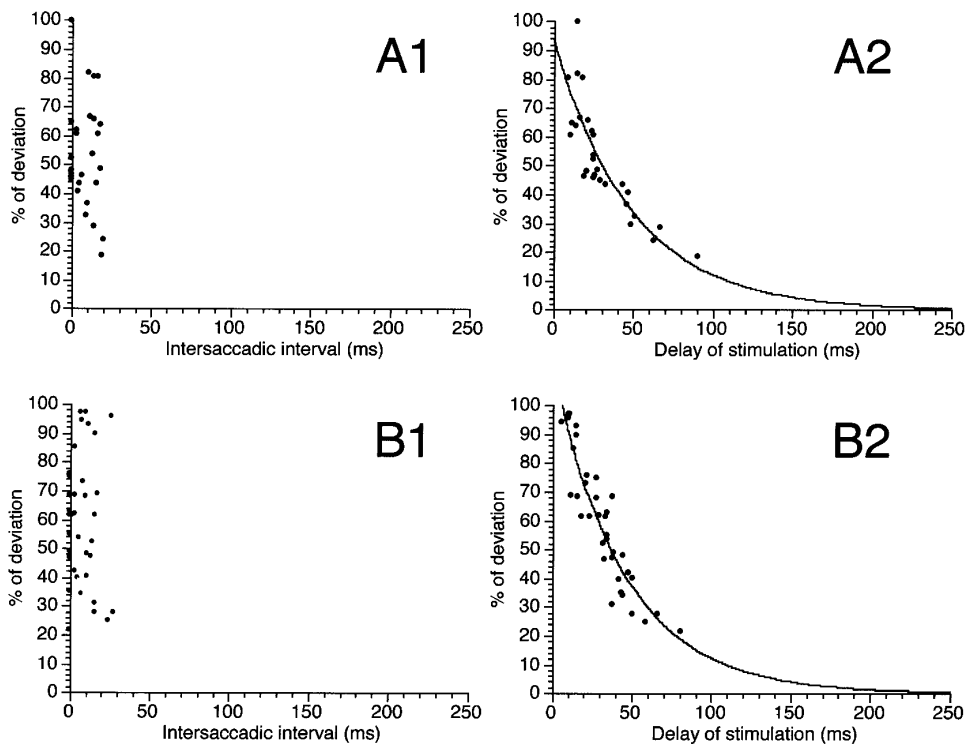


FIG. 4. Comparison of same time series data plotted in terms of intersaccadic interval in *A1*, vs. delay of stimulation in *A2* in one experiment. *B1* and *B2*: same plots in another experiment. Amplitudes of initial saccades varied from 6.5 to 31.3° in *A1*, and from 5.3 to 30.8° in *B1*; time constants are 49 ms in *A2* and 45.5 ms in *B2*; goodness of fit for exponential curves was $R^2 = 85.02$ for *A2* and 84.98 for *B2*.

intersaccadic interval, for 14 time series experiments, with deviation percentages made as a function of delay of stimulation. The best fits for the measurements in terms of delay of stimulation always accounted for a larger percentage of the variance than did the best fits for the measurements made in terms of intersaccadic interval. In seven of these experiments that included initial saccades of different amplitudes, trying to fit an exponential function to data collected as a function of intersaccadic intervals appeared meaningless, as shown in Fig. 4. On the basis of the curves of Fig. 4, one can predict that the results of amplitude series should be very sensitive to the choice of the time reference at which amplitude measurements are made.

The results of three typical amplitude series are illustrated in Fig. 5 where the vertical component of deviation (ordinate) is plotted against the vertical amplitude of the initial movement (abscissa). The black dots represent amplitude measurements made at equal intersaccadic intervals. Twelve amplitude series experiments yielded sufficient results and the relation between the compensatory and the initial saccades was linear in all of them, as Nichols and Sparks (1995) have shown, but only within a limited range of initial saccade amplitudes (6 – 12° up or down). With larger initial saccades, the amount of deviation wore off considerably.

To establish this point, in these 12 amplitude series, the data points were divided into 3 blocks according to the dimension of the initial saccade. Straight lines were fitted through the points within each of these blocks. We found that the slopes within the central range of $+10^\circ$ to -10° initial saccades had a mean of -0.61 (± 0.19). The smallest value for a slope within that central range was much larger than the largest value (-0.29) for slopes outside that range ($> +10^\circ$ and $< -10^\circ$), thus demonstrating the nonlinearity of the overall amplitude relation beyond any doubt. In the

three examples offered in Fig. 5, the data represented by black dots were fitted by sigmoid curves. Nichols and Sparks (1995) had already noticed that the linear relation does not hold well in the reversal region (where an upward saccade is deviated downward, or the converse). Actually, we found that the linear relation breaks down not only in the reversal region but similarly at the other end of the curves (i.e., in the overshoot region).

Could the amplitude relation become linear if amplitude measurements were made instead at equal delays of stimulation? To verify this point we analyzed the data from seven sites of stimulation for which complete amplitude and time series was obtained. The purpose was to convert the data from one frame of reference (constant intersaccadic interval) into another frame (constant delay of stimulation) by computation. This computation is straight forward, it involves the following steps: 1) measure the delay of stimulation for each trial, 2) find the percentage of deviation a on the exponential time curve corresponding to this particular value of the delay of stimulation, 3) find the percentage of deviation b on the exponential time curve corresponding to a given delay of stimulation (e.g., 20 ms), 4) multiply the vertical component of deviation by the ratio b/a , and 5) plot the result (i.e., the vertical component of deviation calculated for a 20 -ms delay of stimulation) against the vertical amplitude of the initial saccade. The results obtained in this manner are shown as open circles for three cases in Fig. 5 and fitted by straight lines. The chosen value of the calculated delay of stimulation was 20 ms for Fig. 5, *A* and *B*, and 0 ms for Fig. 5*C* (which is expected to introduce more noise in the data). In all seven cases analyzed, all best linear fits for measurements made in terms of delays of stimulation (R^2 between 94.27 and 97.37) were better than the best linear fits for

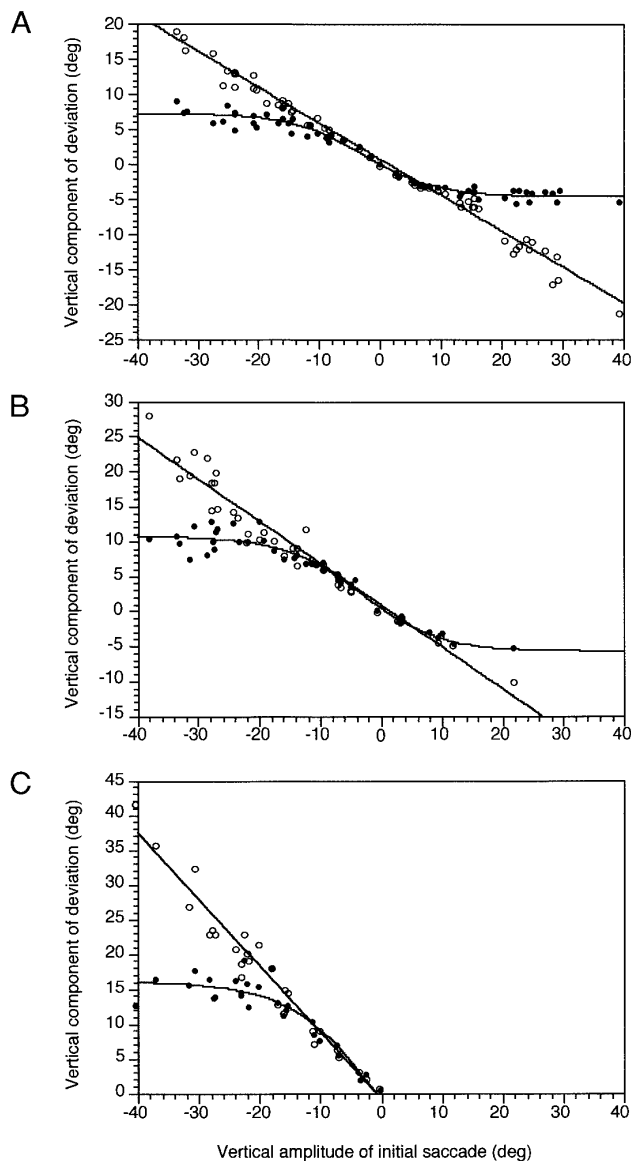


FIG. 5. Amplitude series. Relation between vertical component of deviation and vertical amplitude of initial saccade for 3 sites of stimulation. Black dots are experimental data collected at 20 ms intersaccadic intervals. Best fits (R^2 values) for sigmoid curves shown are 97.65 in A, 87.63 in B, and 95.28 in C; open circles, calculated data for 20-ms delays of stimulation. Best linear fits are 97.33 in A, 95.45 in B, and 95.00 in C. The corresponding best linear fits for black dot data (lines not shown) are 90.68, 65.35, and 85.78 in A–C, respectively.

the corresponding measurements made in terms of intersaccadic intervals (R^2 between 65.35 and 93.20).

DISCUSSION

Nichols and Sparks (1995) designed a paradigm, relying on the effects of stimulating the superior colliculus just after a visually guided saccade, to decide whether the feedback integrator of the saccade generator is instantaneously reset. They argued that the hypothesis of a progressively resetting integrator predicts two features: first, the relation between amplitude of deviation and amplitude of the initial saccade should be linear and second, the ratio of this relation should

decay with time (implicitly, as soon as the saccade generation is completed). On this basis, the hypothesis of a progressively reset integrator was accepted as demonstrated and it served as a premise in two subsequent studies (Nichols and Sparks 1996a,b). Yet, when we tested the two predictions specified above we found that their verification depends very much on the choice of the time reference used to measure the amount of deviation. Our results indicate that the two predictions cannot be simultaneously fulfilled. If one assumes that the decay of deviation is temporally tied to the end of saccade generation, then the first prediction is not satisfied: the amplitude relation is not linear (black dots in Fig. 5). Conversely, if one assumes that the amplitude relation has to be linear, then the second prediction is not satisfied: the decay starts in relation with the beginning, not the end, of saccade generation (Figs. 3 and 4).

It appears that the stimulation experiments do not, as proposed by Nichols and Sparks (1995), test the properties of a resettable integrator but do address a different issue. If our concern was to learn how fast the resettable integrator resets, another experimental approach ought to be found. For instance, one can stimulate the omnipause neuron region of the brain stem to interrupt saccades in midflight. But Keller et al. (1996) using this method found no evidence that the neural integrator starts resetting during the saccade interruption.

Because in our experiments the time decay of deviation appears linked to the start of the initial saccade, we think that this decay must be related to an event (or state) simultaneous with, or preceding rather than following, a saccade. The number of options is limited in this respect. Two signals are assumed to exist prior to a saccade generated by a visual stimulus; one indicates the target retinocentric position and the other is an internal representation of eye position or eye displacement. We suggest that the effect of SC stimulation is to elicit a target retinocentric position signal to which, ulteriorly, an eye position signal is added (or an eye displacement signal subtracted) to correct the aiming disturbed by the displacement of the eyes. We postulate that this signal is damped. At the time of a saccade it grows more slowly than the saccade, with a time constant ~ 50 ms.

The hypothesis invoking a damped eye position, or eye displacement, signal was originally advanced to interpret the results of stimulation in the monkey frontal eye field (Dassonville et al. 1992a). Our first interpretation of the results of SC stimulation was different (Schlag et al. 1989) and, certainly, it must be reevaluated on the evidence of nonstationarity found by Nichols and Sparks (1995) and Kustov and Robinson (1995). Now we think that the results of stimulation in the monkey frontal eye field and those of stimulation in superficial and intermediate SC layers can be accounted for by a similar, perhaps common, mechanism. The purpose of Fig. 6 is to illustrate two hypotheses using simplified functional models (Fig. 6, A and B) of the visuo-oculomotor apparatus. In the hypothesis of a progressively resettable integrator, formulated by Nichols and Sparks (1995) and Kustov and Robinson (1995), SC stimulation would generate a signal at the site marked by the arrow A, before the second summing junction S_2 . This signal would then enter the saccade generator stage in Fig. 6A, which is the same here as the one these authors considered in their

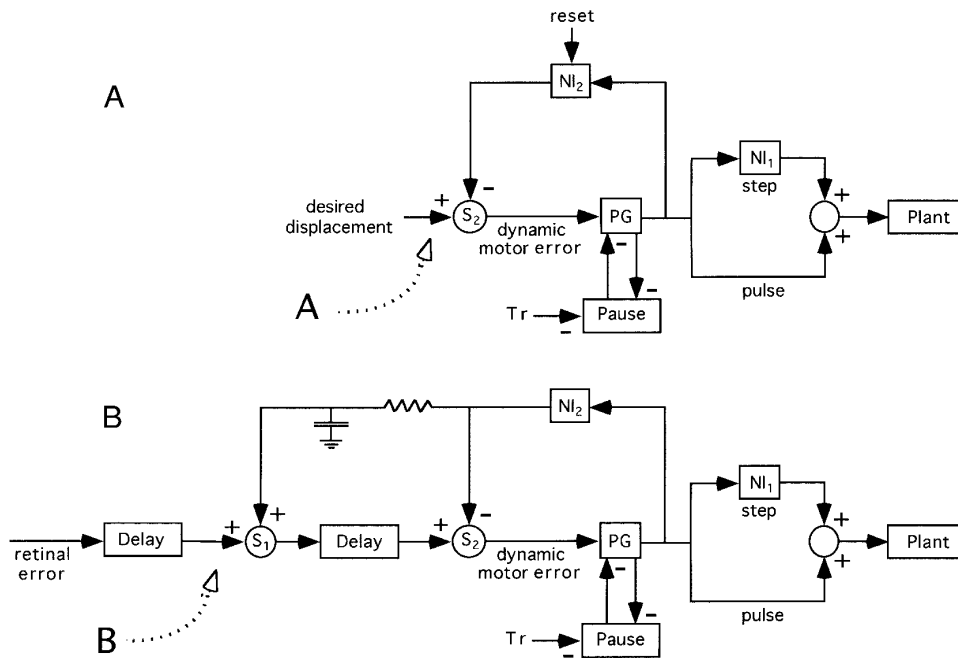


FIG. 6. Models of oculomotor apparatus. NI_1 and NI_2 are 2 separate integrators as suggested by Jürgens et al. (1981). *A*: saccade deviation is explained by progressive resetting of the displacement integrator NI_2 (Nichols and Sparks 1995). In this case, stimulation in intermediate layer of SC is supposed to act at site marked by arrow *A*. The front end of the model is not shown because it is essentially irrelevant for this hypothesis. *B*: saccade deviation is attributed to a temporal mismatch of signals reaching the summing junction S_1 ; the same stimulation in intermediate layer of SC is supposed to act at site marked by arrow *B*. Based on the assumption that integrator NI_2 is not resettable; modifications would be required if this integrator were resettable. PG, the pulse generator; Tr, trigger (go-signal); S_1 and S_2 , summing junctions; and Pause, the omnipause neurons.

papers. In contrast, the hypothesis we propose assumes that stimulation in SC superficial and intermediate layers acts upstream before the first summing junction S_1 in Fig. 6*B*, at the site indicated by the arrow *B*, and therefore engages another feedback loop. The resistor-capacitor circuit on that loop represents the damping. The loop does not function continuously; eye position is supposed to be sampled and stimulus location updated only when a new signal appears at the input of S_1 . In Fig. 6*B* we have to include a saccade generator stage because its nature determines the type of signal that has to be fed back into the first loop. However, our hypothesis does not make assumptions regarding the saccade generator stage and, in particular, about integrator NI_2 ; with some modifications the model can be accommodated to the possibility that this integrator is resettable (see Dominey et al. 1997).

Note that, in both hypotheses distinguished by the site of stimulation (Fig. 6, *A* or *B*), the signal mimicked by that stimulation is supposed to represent a visual goal. If so, theoretically, natural (e.g., visual) stimuli should also be able to elicit the same temporal decay as that produced by electrical stimulation. Indeed, the only difference assumed between natural and electrical stimuli is that the second generates the same signal as the first but after a much shorter delay. There are two experimental situations with visual stimuli in which the two hypotheses under discussion make opposite predictions. These two situations are depicted in Fig. 7.

The first case, shown in Fig. 7*A*, relies on the rather infrequent although natural occurrence of short intersaccadic intervals (e.g., <50 ms) in humans, e.g., in response to successive flashes (stimuli S_1 and S_2). If the resetting rate of the integrator in the saccade generator plays a determinant role, then the second saccade should be inaccurate. The error should be the amount left in this integrator when the second saccade starts. But as Goossens and Van Opstal (1997) have

recently shown, no such error occurs even with very short intersaccadic intervals.

The other case, shown in Fig. 7*B*, concerns the presentation of flashed targets near the time of a saccade. Because of the work of Hallett and Lightstone (1976), it has been widely recognized that the localization of a target flashed before a saccade in total darkness depends on an internal representation of that saccade. Yet an inspection of Fig. 6 reveals that accuracy of targeting crucially depends on the temporal match of signals arriving at the first summing junction S_1 . Retinal processes are responsible for long delays

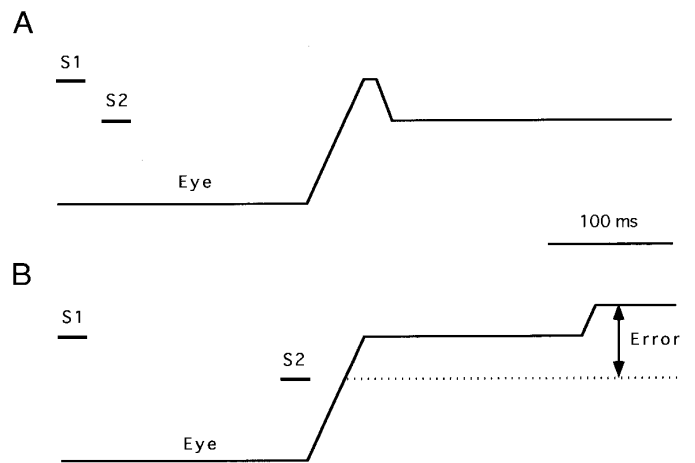


FIG. 7. Schematic representation of 2 situations where saccade deviations could be expected to occur with natural visual stimuli as they do with SC electrical stimulation. One trace of eye movement (—) is shown as a function of time, with the location of two brief stimuli S_1 and S_2 . *A*: 2 saccades occur in rapid succession and 2nd terminates on target (see Goossens and Van Opstal 1997). *B*: the 2nd stimulus is flashed just before a saccade, a condition that consistently produces an error of localization. The time course of this error can be explained by a damping of the eye position signal, which decays exponentially.

(probably >40 ms) on the visual input and it does not seem that such delays are compensated by equal delays on the feedback loop to S_1 . Whereas target localization is usually accurate, it progressively deteriorates as the time interval narrows between target presentation (flash) and the initial saccade (Dassonville et al. 1992b; Honda 1990). Then the localization error by the oculomotor system can become as large as 70% of the initial saccade amplitude (Schlag and Schlag-Rey 1995). Because errors of stimulus localization lawfully depend on the imminence of a saccade, they have been attributed to the combination of a retinocentric vector with a temporally unmatched and distorted eye position signal. The time course of this signal can be recovered from mislocalization data. It is simply the instantaneous difference between the mislocalization error and the actual eye position. When the calculation is done, the results yield an internal eye position (or eye displacement) signal that grows exponentially with a time constant between 51 and 172 ms (for humans and monkeys) (Dassonville et al. 1992b, 1993). The situation where a stimulus is flashed just before a saccade is schematized in Fig. 7B, which also indicates the error of targeting observed in such cases. Note that the second saccade is considerably deviated from its goal even though the intersaccadic interval is very long. Experiments with real stimuli support our hypothesis because it quantitatively accounts for both the target mislocalization of a target flashed near the time of a saccade in the double-step task and the deviation of electrically evoked saccades during and after a natural saccade. A single model covering both situations has been recently offered (Dominey et al. 1997).

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